**TEAM 3**

**DERMATOLOGY:SKIN, NAIL,HAIR**

**Acne**: A condition that occurs when oil and dead skin cells clog the pores, leading to pimples and blackheads.

* **Atopic Dermatitis (Eczema)**: A chronic skin condition that causes dry, itchy, and inflamed skin.
* **Cold Sores**: Caused by the herpes simplex virus, these are painful blisters that appear around the mouth.
* **Psoriasis**: A skin disease that causes red, scaly skin that may feel painful, swollen, or hot.
* **Rosacea**: A long-term disease that causes reddened skin and pimples, usually on the face.
* **Vitiligo**: A disorder that causes patches of skin to become white.
* **Hidradenitis Suppurativa**: An inflammatory skin disorder that causes bumps or boils that look like pimples.
* **Lichen Planus**: A condition that causes shiny, firm, purplish bumps on the skin.
* **Cellulitis**: A bacterial skin infection that causes redness, swelling, and pain.
* **Melanoma**: A type of skin cancer that can spread to other parts of the body without early detection and treatment.
* **Ringworm**: A fungal infection that causes a ring-shaped rash.
* **Shingles**: A viral infection that causes a painful rash.
* **Hives**: A skin condition characterized by red, itchy, and swollen areas on the skin.
* **Fungal Nail Infections**: Infections that cause thickened, discolored, and sometimes painful nails.
* **Actinic prurigo (AP)**, itchy rash in response to sun exposure.
* **Argyria**, changes in skin color due to silver buildup in your body.
* **Chromhidrosis**, colored sweat.
* **Epidermolysis bullosa**, a connective tissue disorder that causes fragile skin that blisters and tears easily.
* **Harlequin ichthyosis**, thick, hard patches or plates on the skin that are present at birth.
* **Lamellar ichthyosis**, waxy skin layer that sheds in the first few weeks of life, revealing scaly, red skin.
* **Necrobiosis lipoidica**, rash on the lower legs that can develop into ulcers (sores).
* scleroderma
* pemphigus
* raynaud’s phenomenon

# **Nails**

Common Diseases Associated with Nails

Nail psoriasis

Brittle splitting nails

Onychogryphosis

onchogryptosis

onychmycosis

Paronychia

Leuconychia

Furrows and ridges

Splinter hemorrhage

**Hair Diseases**

Alopecia Areata

Tinea Capitis (Scalp Ringworm)

Telogen Effluvium

Trichorrhexis Invaginata

Lichen Planopilaris

Folliculitis

Pili Torti

Trichotillomania

Hirsutism

Monilethrix

Head Lice

Androgenetic alopecia

anagen effluvium

scarring alopecia

uncombable hair syndrome

trichorrhexis nodosa

cicatricial alopecia

trichothiodystrophy

**ACNE**  
 **Alternative names**

pimples, blackhead, zit,spot,blotch,papule,hickey,boil,polyp  
**Definition**

Acne is a skin disorder that leads to an outbreak of lesions called pimples or "zits." The most common form of the disease is called acne vulgaris—the rash that affects many adolescents. Acne vulgaris is triggered by the hormonal changes that occur in **puberty.**

**Description**

Acne is a condition in which pimples appear on the face, chest, and back. In teenagers, acne usually appears on the forehead, nose, and chin. It is caused by the overproduction of sebum. Sebum is an oily substance that forms in glands just under the surface of the skin called sebaceous glands. Sebum normally flows out hair follicles onto the skin to act as a natural skin moisturizer. The glands are connected to hair follicles that allow the sebum, or oil, to empty onto the skin through a pore.

If hair follicles become blocked by sebum, dead skin cells, and bacteria, acne is the result. The sebaceous gland units are most found on the face, neck, and back.

During puberty, there are increased levels of the male hormone androgen. High levels of androgen cause excess sebum to form. Sometimes the sebum combines with dead, sticky skin cells and bacteria called *Propioni-bacterium acnes (P. acnes)* that normally

live on the skin. The mixture of oil and cells allows the bacteria to grow in the plugged follicles. When this happens, a hard plug called a comedo can form. A comedo is an enlarged hair follicle. It can take the following forms:

* a blackhead, which is a comedo that reaches the skin's surface and looks black
* a whitehead, which is a comedo that is sealed by keratin, the fibrous protein produced by the skin cells and looks like a white bump.

In addition, pimples can form on the skin. Types of pimples include:

* papules, which are small, red bumps that may be tender to the touch
* pustules, which are pus-filled lesions that are often red at the base
* nodules, which are large, painful lesions deep in the skin
* cysts, which are painful pus-filled lesions deep in the skin that can cause scarring

Pimples form when the follicle is invaded by the *P. acnes* bacteria. The damaged follicle weakens and bursts open, releasing sebum, bacteria, skin cells, and white blood cells into surrounding tissues. Scarring happens when new skin cells are created to replace the damaged cells. The most severe type of acne includes both nodules and cysts.

Acne is a skin condition that occurs when your hair follicles become plugged with oil and dead skin cells. It causes whiteheads, blackheads or pimples. Acne is most common among teenagers, though it affects people of all ages.

Effective acne treatments are available, but acne can be persistent. The pimples and bumps heal slowly, and when one begins to go away, others seem to crop up.

Depending on its severity, acne can cause emotional distress and scar the skin. The earlier you start treatment, the lower your risk of such problems.

## Demographics

## Acne affects as many as 17 million people in the United States, making it the most common skin disease. Acne usually begins at puberty and worsens during **adolescence.** Nearly 85 percent of people develop acne at some point between ages 12 to 25. As many as 20 million teens have the condition. Acne may appear as early as age 10 and even may be found in some newborns. Some people may continue to be affected by acne after age 30.

## **Symptoms**

## Acne signs vary depending on the severity of your condition;

* Whiteheads (closed clogged pores)
* Blackheads (open plugged pores)
* Small red, tender bumps (papules)
* Pimples (pustules), which are papules with pus at their tips
* Large, solid, painful lumps under the skin (nodules)
* Painful, pus-filled lumps under the skin (cystic lesions)

Acne usually appears on the face, forehead, chest, upper back and shoulders.

### **When to see a doctor**

If self-care remedies don't clear your acne, see your primary care doctor. He or she can prescribe stronger medications. If acne persists or is severe, you may want to seek medical treatment from a doctor who specializes in the skin (dermatologist or pediatric dermatologist).

For many women, acne can persist for decades, with flares common a week before menstruation. This type of acne tends to clear up without treatment in women who use contraceptives.

In older adults, a sudden onset of severe acne may signal an underlying disease requiring medical attention.

The Food and Drug Administration (FDA) warns that some popular non-prescription acne lotions, cleansers and other skin products can cause a serious reaction. This type of reaction is quite rare, so don't confuse it with any redness, irritation or itchiness that occurs in areas where you've applied medications or products.

**Seek emergency medical help** if after using a skin product you experience:

* Faintness
* Difficulty breathing
* Swelling of the eyes, face, lips or tongue
* Tightness of the throat

**Causes**

Four main factors cause acne:

* Excess oil (sebum) production
* Hair follicles clogged by oil and dead skin cells
* Bacteria
* Inflammation

Acne typically appears on your face, forehead, chest, upper back and shoulders because these areas of skin have the most oil (sebaceous) glands. Hair follicles are connected to oil glands.

The follicle wall may bulge and produce a whitehead. Or the plug may be open to the surface and darken, causing a blackhead. A blackhead may look like dirt stuck in pores. But the pore is congested with bacteria and oil, which turns brown when it's exposed to the air.

Pimples are raised red spots with a white center that develop when blocked hair follicles become inflamed or infected with bacteria. Blockages and inflammation deep inside hair follicles produce cyst like lumps beneath the surface of your skin. Other pores in your skin, which are the openings of the sweat glands, aren't usually involved in acne.

Certain things may trigger or worsen acne:

* **Hormonal changes.** Androgens are hormones that increase in boys and girls during puberty and cause the sebaceous glands to enlarge and make more sebum. Hormone changes during midlife, particularly in women, can lead to breakouts too.
* **Certain medications.** Examples include drugs containing corticosteroids, testosterone or lithium.
* **Diet.** Studies indicate that consuming certain foods — including carbohydrate-rich foods, such as bread, bagels and chips — may worsen acne. Further study is needed to examine whether people with acne would benefit from following specific dietary restrictions.
* **Stress.** Stress doesn't cause acne, but if you have acne already, stress may make it worse.

### **Acne myths**

These factors have little effect on acne:

* **Chocolate and greasy foods.** Eating chocolate or greasy food has little to no effect on acne.
* **Hygiene.** Acne isn't caused by dirty skin. In fact, scrubbing the skin too hard or cleansing with harsh soaps or chemicals irritates the skin and can make acne worse.
* **Cosmetics.** Cosmetics don't necessarily worsen acne, especially if you use oil-free makeup that doesn't clog pores (non comedogenic) and remove makeup regularly. Nonoily cosmetics don't interfere with the effectiveness of acne drugs.

**Complications**

People with darker skin types are more likely than are people with lighter skin to experience these acne complications:

* **Scars.** Pitted skin (acne scars) and thick scars (keloids) can remain long-term after acne has healed.
* **Skin changes.** After acne has cleared, the affected skin may be darker (hyperpigmented) or lighter (hypopigmented) than before the condition occurred.

**Risk factors**

Risk factors for acne include:

* **Age.** People of all ages can get acne, but it's most common in teenagers.
* **Hormonal changes.** Such changes are common during puberty or pregnancy.
* **Family history.** Genetics plays a role in acne. If both of your parents had acne, you're likely to develop it too.
* **Greasy or oily substances.** You may develop acne where your skin comes into contact with oil or oily lotions and creams.
* **Friction or pressure on your skin.** This can be caused by items such as telephones, cellphones, helmets, tight collars and backpacks.

## **Treatment**

If you've tried over –the- counter (nonprescription) acne products for several weeks and they haven't helped, ask your doctor about prescription-strength medications. A dermatologist can help you:

* Control your acne
* Avoid scarring or other damage to your skin
* Make scars less noticeable

Acne medications work by reducing oil production and swelling or by treating bacterial infection. With most prescription acne drugs, you may not see results for four to eight weeks. It can take many months or years for your acne to clear up completely.

The treatment regimen your doctor recommends depends on your age, the type and severity of your acne, and what you are willing to commit to. For example, you may need to wash and apply medications to the affected skin twice a day for several weeks. Topical medications and drugs you take by mouth (oral medication) are often used in combination. Treatment options for pregnant women are limited due to the risk of side effects.

Talk with your doctor about the risks and benefits of medications and other treatments you are considering. And make follow-up appointments with your doctor every three to six months until your skin improves.

### **Topical medications**

The most common topical prescription medications for acne are:

* **Retinoids and retinoid-like drugs.** Drugs that contain retinoic acids or tretinoin are often useful for moderate acne. These come as creams, gels and lotions. Examples include tretinoin (Avita, Retin-A, others), adapalene (Differin) and tazarotene (Tazorac, Avage, others). You apply this medication in the evening, beginning with three times a week, then daily as your skin becomes used to it. It prevents plugging of hair follicles. Do not apply tretinoin at the same time as benzoyl peroxide.

Topical retinoids increase your skin's sun sensitivity. They can also cause dry skin and redness, especially in people with brown or Black skin. Adapalene may be tolerated best.

* **Antibiotics.** These work by killing excess skin bacteria and reducing redness and inflammation. For the first few months of treatment, you may use both a retinoid and an antibiotic, with the antibiotic applied in the morning and the retinoid in the evening. The antibiotics are often combined with benzoyl peroxide to reduce the likelihood of developing antibiotic resistance. Examples include clindamycin with benzoyl peroxide (Benzaclin, Duac, others) and erythromycin with benzoyl peroxide (Benzamycin). Topical antibiotics alone aren't recommended.
* **Azelaic acid and salicylic acid.** Azelaic acid is a naturally occurring acid produced by a yeast. It has antibacterial properties. A 20% azelaic acid cream or gel seems to be as effective as many conventional acne treatments when used twice a day. Prescription azelaic acid (Azelex, Finacea) is an option during pregnancy and while breast-feeding. It can also be used to manage discoloration that occurs with some types of acne. Side effects include skin redness and minor skin irritation.

Salicylic acid may help prevent clogged hair follicles and is available as both wash-off and leave-on products. Studies showing its effectiveness are limited. Side effects include skin discoloration and minor skin irritation.

* **Dapsone.** Dapsone (Aczone) 5% gel twice daily is recommended for inflammatory acne, especially in women with acne. Side effects include redness and dryness.

Evidence is not strong in support of using zinc, sulfur, nicotinamide, resorcinol, sulfacetamide sodium or aluminum chloride in topical treatments for acne.

### **Oral medications**

* **Antibiotics.** For moderate to severe acne, you may need oral antibiotics to reduce bacteria. Usually, the first choice for treating acne is a tetracycline (minocycline, doxycycline) or a macrolide (erythromycin, azithromycin). A macrolide might be an option for people who can't take tetracyclines, including pregnant women and children under 8 years old.

Oral antibiotics should be used for the shortest time possible to prevent antibiotic resistance. And they should be combined with other drugs, such as benzoyl peroxide, to reduce the risk of developing antibiotic resistance.

Severe side effects from the use of antibiotics to treat acne are uncommon. These drugs do increase your skin's sun sensitivity.

* **Combined oral contraceptives.** Four combined oral contraceptives are approved by the FDA for acne therapy in women who also wish to use them for contraception. They are products that combine progestin and estrogen (Ortho Tri-Cyclen 21, Yaz, others). You may not see the benefit of this treatment for a few months, so using other acne medications with it for the first few weeks may help.

Common side effects of combined oral contraceptives are weight gain, breast tenderness and nausea. These drugs are also associated with increased risk of cardiovascular problems, breast cancer and cervical cancer.

* **Anti-androgen agents.** The drug spironolactone (Aldactone) may be considered for women and adolescent girls if oral antibiotics aren't helping. It works by blocking the effect of androgen hormones on the oil-producing glands. Possible side effects include breast tenderness and painful periods.
* **Isotretinoin.** Isotretinoin (Amnesteem, Claravis, others) is a derivative of vitamin A. It may be prescribed for people whose moderate or severe acne hasn't responded to other treatments.

Potential side effects of oral isotretinoin include inflammatory bowel disease, depression and severe birth defects. All people receiving isotretinoin must participate in an FDA-approved risk management program. And they'll need to see their doctors regularly to monitor for side effects.

### **Therapies**

For some people, the following therapies might be helpful, either alone or in combination with medications.

* **Light therapy.** A variety of light-based therapies have been tried with some success. Most will require multiple visits to your doctor's office. Further study is needed to determine the ideal method, light source and dose.
* **Chemical peel.** This procedure uses repeated applications of a chemical solution, such as salicylic acid, glycolic acid or retinoic acid. This treatment is for mild acne. It might improve the appearance of the skin, though the change is not long lasting and repeat treatments are usually needed.
* **Drainage and extraction.** Your doctor may use special tools to gently remove whiteheads and blackheads (comedos) or cysts that haven't cleared up with topical medications. This technique temporarily improves the appearance of your skin, but it might also cause scarring.
* **Steroid injection.** Nodular and cystic lesions can be treated by injecting a steroid drug into them. This therapy has resulted in rapid improvement and decreased pain. Side effects may include skin thinning and discoloration in the treated area.

### **Treating children**

Most studies of acne drugs have involved people 12 years of age or older. Increasingly, younger children are getting acne as well. The FDA has expanded the number of topical products approved for use in children. And guidelines from the American Academy of Dermatology indicate that topical benzoyl peroxide, adapalene and tretinoin in preadolescent children are effective and don't cause increased risk of side effects.

If your child has acne, consider consulting a pediatric dermatologist. Ask about drugs to avoid in children, appropriate doses, drug interactions, side effects, and how treatment may affect a child's growth and development.

**Alternative medicine**

Some alternative and integrative medicine approaches might be helpful in reducing acne:

* **Tea tree oil.** Gels containing at least 5% tea tree oil may be as effective as lotions containing 5% benzoyl peroxide, although tea tree oil might work more slowly. Possible side effects include minor itching, burning, redness and dryness, which make it a poor choice for people with rosacea.
* **Brewer's yeast.** A strain of brewer's yeast called Hansen CBS seems to help decrease acne when taken orally. It may cause gas (flatulence).

More research is needed to establish the potential effectiveness and long-term safety of these and other integrative approaches, such as biofeedback and ayurvedic compounds. Talk with your doctor about the pros and cons of specific treatments before you try them.

**Lifestyle and home remedies**

You can try to avoid or control mild or moderate acne with nonprescription products, good basic skin care and other self-care techniques:

* **Wash problem areas with a gentle cleanser.** Twice a day, use your hands to wash your face with mild soap or a gentle cleanser (Cetaphil, Vanicream, others) and warm water. And be gentle if you're shaving affected skin.

Avoid certain products, such as facial scrubs, astringents and masks. They tend to irritate the skin, which can worsen acne. Too much washing and scrubbing also can irritate the skin.

* **Try over-the-counter acne products to dry excess oil and promote peeling.** Look for products containing benzoyl peroxide and adapalene as the active ingredients. You might also try products containing salicylic acid, glycolic acid or alpha hydroxy acids. It may take a few weeks of using a product before you see any improvement.

Creams are less irritating than gels or ointments. Nonprescription acne medications may cause initial side effects — such as redness, dryness and scaling — that often improve after the first month of using them.

* **Avoid irritants.** Oily or greasy cosmetics, sunscreens, hair styling products or acne concealers can worsen acne. Instead, use products labeled water-based or non comedogenic, which means they are less likely to cause acne.
* **Protect your skin from the sun.** For some people, the sun worsens the discoloration that sometimes lingers after the acne has cleared. And some acne medications make you more susceptible to sunburn. Check with your doctor to see if your medication is one of these. If it is, stay out of the sun as much as possible. Regularly use a non oily (non comedogenic) moisturizer that includes a sunscreen.
* **Avoid friction or pressure on your skin.** Protect your acne-prone skin from contact with items such as phones, helmets, tight collars or straps, and backpacks.
* **Avoid touching or picking acne-prone areas.** Doing so can trigger more acne or lead to infection or scarring.
* **Shower after strenuous activities.** Oil and sweat on your skin can lead to breakouts.

**Preparing for your appointment**

If you have acne that's not responding to self-care and over-the-counter treatments, make an appointment with your doctor. Early, effective treatment of acne reduces the risk of scarring and of lasting damage to your self-esteem. After an initial examination, your doctor may refer you to a specialist in the diagnosis and treatment of skin conditions (dermatologist).

Here's some information to help you get ready for your appointment.

### **What you can do**

* **List your key medical information,** such as other conditions you're dealing with and any prescription or over-the-counter products you're using, including vitamins and supplements.
* **List key personal information,** including any major stresses or recent life changes.
* **List questions to ask** your doctor. Creating your list of questions in advance can help you make the most of your time with your doctor.

Below are some basic questions to ask your doctor about acne. If any additional questions occur to you during your visit, don't hesitate to ask.

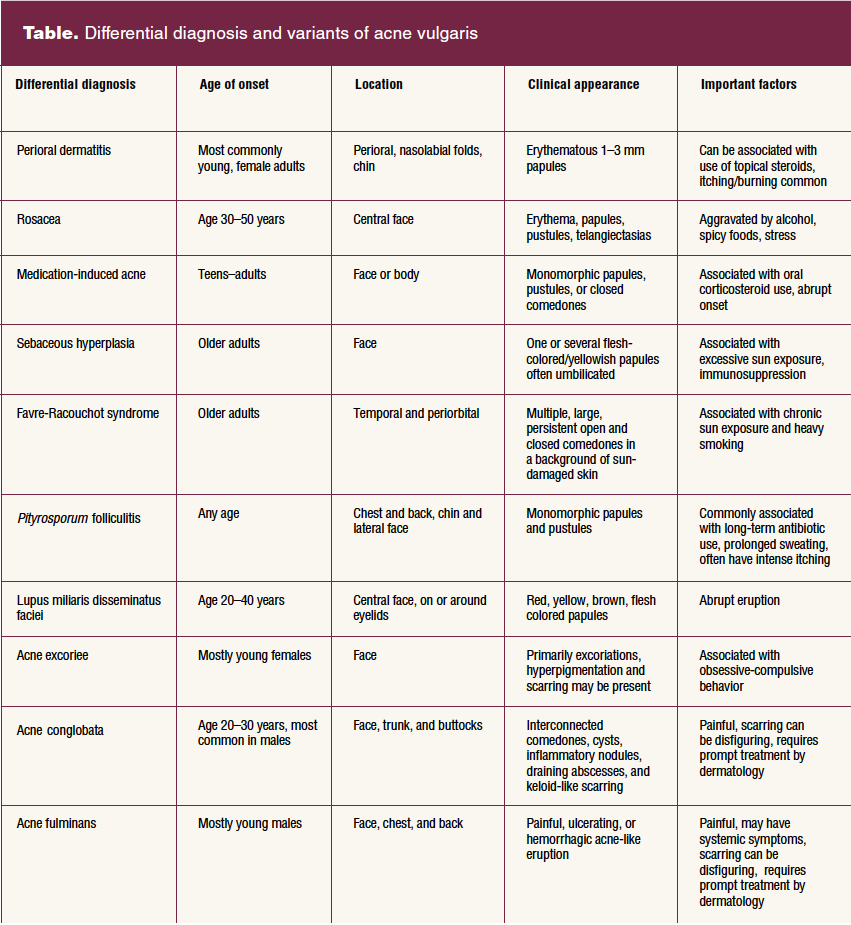
* What treatment approach do you recommend for me?
* If the first treatment doesn't work, what will you recommend next?
* What are the possible side effects of the medications you're prescribing?
* How long can I safely use the medications you're prescribing?
* How soon after beginning treatment might my symptoms start to improve?
* When will you see me again to evaluate whether my treatment is working?
* Is it safe to stop my medications if they don't seem to be working?
* What self-care steps might improve my symptoms?
* Do you recommend any changes to my diet?
* Do you recommend any changes to the over-the-counter products I'm using on my skin, including soaps, lotions, sunscreens and cosmetics?

### **What to expect from your doctor**

Your doctor is likely to ask you a few questions. Being ready to answer them may reserve time to go over any points you want to talk about in-depth. Your doctor may ask:

* When did you first develop this problem?
* Does anything seem to trigger an acne flare, such as stress or — in girls and women — your menstrual cycle?
* What medications are you taking, including over the counter and prescription drugs as well as vitamins and supplements?
* In girls and women: Do you use oral contraceptives?
* In girls and women: Do you have regular menstrual periods?
* In girls and women: Are you pregnant, or do you plan to become pregnant soon?
* What types of soaps, lotions, sunscreens, hair products or cosmetics do you use?
* How is acne affecting your self-esteem and your confidence in social situations?
* Do you have a family history of acne?
* What treatments and self-care steps have you tried so far? Have any been effective?
* Have other family members had isotretinoin treatment or hormone therapy to treat their acne? Has it been effective?

**Differential diagnosis (how it’s distinguished from other illnesses)**

  
  
**Recent guidelines or updates**

Strong recommendations were made for the use of the following:

* **Topical benzoyl peroxide** to reduce the number of acne-causing bacteria on the skin.
* **Topical retinoids** (e.g. adapalene, tretinoin, tazarotene, trifarotene) to help unclog pores and reduce inflammation.
* **Oral antibiotics,** such as **oral doxycycline,** and **topical antibiotics** to reduce the number of acne-causing bacteria on the skin and lessen inflammation.
* **Combinations of topical benzoyl peroxide, retinoids, or the above antibiotics.**

Employing best practices in guideline development, the multidisciplinary workgroup also issued 5 good practice statements.

* When managing acne with topical medications, the guidelines recommend combining multiple different treatment types, as this can lead to better results.
* The guidelines recommend limiting use of oral antibiotics when possible, to reduce the development of antibiotic resistance and other antibiotic-associated complications.
* It is recommended that topical and oral antibiotics are used simultaneously with benzoyl peroxide to prevent the development of antibiotic resistance.
* For patients with larger acne bumps, the guidelines recommend injectable corticosteroids as a potential option for more rapid relief of inflammation and pain.
* For patients with severe acne or for patients who have failed standard treatment with oral or topical therapy, the guidelines recommend isotretinoin.

Additionally, the guidelines provide conditional recommendations. Conditional recommendations apply to most patients, but the most appropriate action may differ depending on individual patient factors.

* **Topical clascoterone**, which addresses hormonal causes of acne.
* **Topical salicylic acid** to unclog pores and exfoliate the skin.
* **Topical azelaic acid** to unclog pores, kill bacteria, and fade dark spots that may continue when an acne spot clears.
* **Oral minocycline** or **sarecycline** to reduce the number of acne-causing bacteria on the skin and lessen inflammation.
* **Hormonal therapies** such as **combined oral contraceptives** or **spironolactone** to address hormonal causes of acne.

Available evidence was insufficient to develop recommendations for procedures such as chemical peels, laser and light-based devices, microneedling, as well as for dietary changes, or alternative therapies such as vitamins or plant-based products. Additionally, a conditional recommendation against adding broadband light, intense pulsed light, to adapalene 0.3% gel.

Since there are a variety of treatment options for acne and treatment plans are not one-size-fits-all, it is important to partner with a board-certified dermatologist to decide which options work best for you.

**statistics or epidemiology**

Acne is estimated to affect 9.4% of the global population, making it the eighth most prevalent disease worldwide. Epidemiological studies have demonstrated that acne is most common in post pubescent teens, with boys most frequently affected, particularly with more severe forms of the disease. This paper aims to provide an update on the epidemiology of acne worldwide. Recent general and institutional studies from around the world have shown that the prevalence of acne is broadly consistent globally (with the exception of specific populations, which are discussed). However, this review highlights that there is a wide range of disparate outcome measures being applied in epidemiology studies, and we emphasize the need to develop a widely accepted, credible, standard assessment scale to address this in the future. In addition, we discuss special populations, such as those devoid of acne, as well as the impact of potential determinants of acne on disease epidemiology.

REFERENCE

<https://pubmed.ncbi.nlm.nih.gov/25597339/>

<http://www.healthofchildren.com/A/Acne.html>

<https://www.npwomenshealthcare.com/differential-diagnosis-and-variants-of-acne-vulgaris/>

<https://www.mayoclinic.org/diseases-conditions/acne/diagnosis-treatment/drc-20368048>

<https://www.mayoclinic.org/diseases-conditions/acne/symptoms-causes/syc-20368047>

**Atopic dermatitis (eczema)**   
**Definition and description**

Atopic dermatitis (eczema) is a condition that causes dry, itchy and inflamed skin. It's common in young children but can occur at any age. Atopic dermatitis is long lasting (chronic) and tends to flare sometimes. It can be irritating but it's not contagious.

People with atopic dermatitis are at risk of developing food allergies, hay fever and asthma.

Moisturizing regularly and following other skin care habits can relieve itching and prevent new outbreaks (flares). Treatment may also include medicated ointments or creams.

**Causes**

In some people, atopic dermatitis is related to a gene variation that affects the skin's ability to provide protection. With a weak barrier function, the skin is less able to retain moisture and protect against bacteria, irritants, allergens and environmental factors — such as tobacco smoke.

In other people, atopic dermatitis is caused by too much of the bacteria Staphylococcus aureus on the skin. This displaces helpful bacteria and disrupts the skin's barrier function.

A weak skin barrier function might also trigger an immune system response that causes the inflamed skin and other symptoms.

Atopic dermatitis (eczema) is one of several types of dermatitis. Other common types are contact dermatitis and seborrheic dermatitis (dandruff). Dermatitis isn't contagious.

**Risk factors**

The main risk factor for atopic dermatitis is having had eczema, allergies, hay fever or asthma in the past. Having family members with these conditions also increases your risk.

**Signs and symptoms**

Atopic dermatitis (eczema) symptoms can appear anywhere on the body and vary widely from person to person. They may include:

* Dry, cracked skin
* Itchiness (pruritus)
* Rash on swollen skin that varies in color depending on your skin color
* Small, raised bumps, on brown or Black skin
* Oozing and crusting
* Thickened skin
* Darkening of the skin around the eyes
* Raw, sensitive skin from scratching

Atopic dermatitis often begins before age 5 and may continue into the teen and adult years. For some people, it flares and then clears up for a time, even for several years.

## **Diagnosis**

To diagnose atopic dermatitis, your health care provider will likely talk with you about your symptoms, examine your skin and review your medical history. You may need tests to identify allergies and rule out other skin diseases.

If you think a certain food caused your child's rash, ask your healthcare provider about potential food allergies.

### **Patch testing**

Your doctor may recommend patch testing on your skin. In this test, small amounts of different substances are applied to your skin and then covered. During visits over the next few days, the doctor looks at your skin for signs of a reaction. Patch testing can help diagnose specific types of allergies causing your dermatitis.

**Treatment**

Treatment of atopic dermatitis may start with regular moisturizing and other self-care habits. If these don't help, your health care provider might suggest medicated creams that control itching and help repair skin. These are sometimes combined with other treatments.

Atopic dermatitis can be persistent. You may need to try various treatments over months or years to control it. And even if treatment is successful, symptoms may return (flare).

### **Medications**

* **Medicated products applied to the skin.** Many options are available to help control itching and repair the skin. Products are available in various strengths and as creams, gels and ointments. Talk with your health care provider about the options and your preferences. Whatever you use, apply it as directed (often twice a day), before you moisturize. Overuse of a corticosteroid product applied to the skin may cause side effects, such as thinning skin.

Creams or ointments with a calcineurin inhibitor might be a good option for those over age 2. Examples include tacrolimus (Protopic) and pimecrolimus (Elidel). Apply it as directed, before you moisturize. Avoid strong sunlight when using these products.

The Food and Drug Administration requires that these products have a black box warning about the risk of lymphoma. This warning is based on rare cases of lymphoma among people using topical calcineurin inhibitors. After 10 years of study, no causal relationship between these products and lymphoma and no increased risk of cancer have been found.

* **Drugs to fight infection.** Your health care provider may prescribe antibiotic pills to treat an infection.
* **Pills that control inflammation.** For more-severe eczema, your health care provider may prescribe pills to help control your symptoms. Options might include cyclosporine, methotrexate, prednisone, mycophenolate and azathioprine. These pills are effective but can't be used long term because of potential serious side effects.
* **Other options for severe eczema.** The injectable biologics (monoclonal antibodies) dupilumab (Dupixent) and tralokinumab (Adbry) might be options for people with moderate to severe disease who don't respond well to other treatments. Studies show that it's safe and effective in easing the symptoms of atopic dermatitis. Dupilumab is for people over age 6. Tralokinumab is for adults.

### **Therapies**

* **Wet dressings.** An effective, intensive treatment for severe eczema involves applying a corticosteroid ointment and sealing in the medication with a wrap of wet gauze topped with a layer of dry gauze. Sometimes this is done in a hospital for people with widespread lesions because it's labor intensive and requires nursing expertise. Or ask your health care provider about learning how to use this technique at home safely.
* **Light therapy.** This treatment is used for people who either don't get better with topical treatments or rapidly flare again after treatment. The simplest form of light therapy (phototherapy) involves exposing the affected area to controlled amounts of natural sunlight. Other forms use artificial ultraviolet A (UVA) and narrow band ultraviolet B (UVB) alone or with drugs.

Though effective, long-term light therapy has harmful effects, including premature skin aging, changes in skin color (hyperpigmentation) and an increased risk of skin cancer. For these reasons, phototherapy is less commonly used in young children and is not given to infants. Talk with your health care provider about the pros and cons of light therapy.

* **Counseling.** If you're embarrassed or frustrated by your skin condition, it can help to talk with a therapist or other counselor.
* **Relaxation, behavior modification and biofeedback.** These approaches may help people who scratch out of habit.

### **Baby eczema**

Treatment for eczema in babies (infantile eczema) includes:

* Identifying and avoiding skin irritants
* Avoiding extreme temperatures
* Giving your baby a short bath in warm water and applying a cream or ointment while the skin is still damp

See your baby's health care provider if these steps don't improve the rash or it looks infected. Your baby might need a prescription medication to control the rash or treat an infection. Your health care provider might also recommend an oral antihistamine to help lessen the itch and cause drowsiness, which may be helpful for nighttime itching and discomfort. The type of antihistamine that causes drowsiness may negatively affect the school performance of some children.

**Lifestyle and home remedies**

Taking care of sensitive skin is the first step in treating atopic dermatitis and preventing flares. To help reduce itching and soothe inflamed skin, try these self-care measures:

* **Moisturize your skin at least twice a day.** Find a product or combination of products that works for you. You might try bath oils, creams, lotions, shea butter, ointments or sprays. For a child, the twice-a-day regimen might be an ointment before bedtime and a cream before school. Ointments are greasier and may sting less when applied. Choose products that are free of dyes, alcohols, fragrances and other ingredients that might irritate the skin. Allow the moisturizer to absorb into the skin before getting dressed.
* **Apply an anti-itch cream to the affected area.** A nonprescription cream containing at least 1% hydrocortisone can temporarily relieve the itch. Apply it no more than twice a day to the affected area before moisturizing. Once your reaction has improved, you may use this type of cream less often to prevent flares.
* **Take an oral allergy or anti-itch medication.** Options include non prescription allergy medicines (antihistamines) — such as cetirizine (Zyrtec Allergy) or fexofenadine (Allegra Allergy). Also, diphenhydramine (Benadryl, others) may be helpful if itching is severe. But it causes drowsiness, so it's better for bedtime. The type of antihistamine that causes drowsiness may negatively affect the school performance of some children.
* **Don't scratch.** Rather than scratching when you itch, try pressing on or patting the skin. Cover the itchy area if you can't keep from scratching it. Keep your nails trimmed. For children, it might help to trim their nails and have them wear socks or gloves at night.
* **Take a daily bath or shower.** Use warm, rather than hot, water. If you're taking a bath, sprinkle the water with colloidal oatmeal, which is finely ground oatmeal made for bathing (Aveeno, others). Soak for less than 10 minutes, then pat dry. Apply moisturizer while the skin is still damp (within three minutes).
* **Use a gentle, non soap cleanser.** Choose one without dyes, alcohols or fragrances. Harsh soaps can wash away your skin's natural oils. Be sure to rinse off the cleanser completely.
* **Take a bleach bath.** The American Academy of Dermatology recommends a bleach bath for relief from severe or frequent flares. Talk with your health care provider about whether this is a good option for you.

A diluted-bleach bath decreases bacteria on the skin and related infections. Add 1/2 cup (118 milliliters) of household bleach, not concentrated bleach, to a 40-gallon (151-liter) bathtub filled with warm water. Measurements are for a U.S.-standard-sized tub filled to the overflow drainage holes.

Soak from the neck down or just the affected areas for 5 to10 minutes. Don't put the head under water. Rinse off the bleach water with tap water. Take a bleach bath 2 to 3 times a week.

* **Use a humidifier.** Hot, dry indoor air can parch sensitive skin and worsen itching and flaking. A portable home humidifier or a humidifier attached to your furnace adds moisture to the air inside your home.
* **Wear cool, smooth-textured clothing.** Avoiding clothing that's rough, tight or scratchy. Also, in hot weather or while exercising, choose lightweight clothing that lets your skin breathe. When washing your clothing, avoid harsh detergents and fabric softeners added during the drying cycle.
* **Treat stress and anxiety.** Stress and other emotional disorders can worsen atopic dermatitis. Being aware of stress and anxiety and taking steps to improve your emotional health may help your skin too.

**Alternative medicine**

Many people with atopic dermatitis have tried alternative (integrative) medicine approaches to easing their symptoms. Some approaches are supported by clinical studies.

* **Cannabinoids.** When applied to skin, creams containing cannabinoids have been shown to ease itching and skin thickening. Several studies over more than 10 years showed some benefit.
* **Natural oils.** When added to bathwater, natural oils might help improve dry skin. Examples of such oils are soybean oil and mineral oil. Use caution with oils in a bathtub as they can make the tub slippery.
* **Manuka honey.** When applied to the skin, manuka honey has been shown to calm reactions on the skin. It has been used for centuries as an antimicrobial. Don't use it on children under 1 year of age, as it carries the risk of infantile botulism.
* **Acupuncture and acupressure.** Several studies show that acupuncture and acupressure can reduce the itchiness of atopic dermatitis.

If you're considering alternative therapies, talk with your health care provider about their pros and cons.

**Outlook / Prognosis**

### **What can I expect if I have eczema?**

Eczema and other types of dermatitis aren’t harmful to the rest of your body. The condition isn’t deadly. Nearly half of children with eczema outgrow the condition or experience improvement by the time they reach puberty. Others will continue to have some form of the condition throughout their life. For adults with eczema, the condition can be well managed with a good skin care routine.

### **How long does eczema last?**

Eczema can be a lifelong condition. It can start in infancy and continue through adulthood. You can manage your symptoms with at-home remedies, over-the-counter medications and prescription medications.

### **Is there a cure for eczema?**

No, there isn’t a cure for eczema. There are treatments available, but no treatment can eliminate your symptoms 100% of the time. Eczema is a chronic condition, which means it can go away and come back unexpectedly. Treatments are very effective in reducing the symptoms of itchy, dry skin.

**How do I take care of myself?**

Many people live with eczema, and it can be challenging. But there may be times when your eczema disappears. This is known as a “remission” period. Other times, you may have a “flare-up,” which is when your symptoms show up or get worse. The goal of treatment is to prevent flare-ups and your symptoms from getting worse. Be sure to avoid triggers, moisturize, take your medicine and follow your healthcare provider’s instructions.

**Complications**

Complications of atopic dermatitis (eczema) may include:

* **Asthma and hay fever.** Many people with atopic dermatitis develop asthma and hay fever. This can happen before or after developing atopic dermatitis.
* **Food allergies.** People with atopic dermatitis often develop food allergies. One of the main symptoms of this condition is hives (urticaria).
* **Chronic itchy, scaly skin.** A skin condition called neurodermatitis (lichen simplex chronicus) starts with a patch of itchy skin. You scratch the area, which provides only temporary relief. Scratching actually makes the skin itchier because it activates the nerve fibers in your skin. Over time, you may scratch out of habit. This condition can cause the affected skin to become discolored, thick and leathery.
* **Patches of skin that's darker or lighter than the surrounding area.** This complication after the rash has healed is called post-inflammatory hyperpigmentation or hypopigmentation. It's more common in people with brown or Black skin. It might take several months for the discoloration to fade.
* **Skin infections.** Repeated scratching that breaks the skin can cause open sores and cracks. These increase the risk of infection from bacteria and viruses. These skin infections can spread and become life-threatening.
* **Irritant hand dermatitis.** This especially affects people whose hands are often wet and exposed to harsh soaps, detergents and disinfectant at work.
* **Allergic contact dermatitis.** This condition is common in people with atopic dermatitis. Allergic contact dermatitis is an itchy rash caused by touching substances you're allergic to. The color of the rash varies depending on your skin color.
* **Sleep problems.** The itchiness of atopic dermatitis can interfere with sleep.
* **Mental health conditions.** Atopic dermatitis is associated with depression and anxiety. This may be related to the constant itching and sleep problems common among people with atopic dermatitis.

**Prevention**

Developing a basic skin care routine may help prevent eczema flares. The following tips may help reduce the drying effects of bathing:

* **Moisturize your skin at least twice a day.** Creams, ointments, shea butter and lotions seal in moisture. Choose a product or products that work well for you. Ideally, the best one for you will be safe, effective, affordable and unscented.

Using petroleum jelly on your baby's skin may help prevent development of atopic dermatitis.

* **Take a daily bath or shower.** Use warm, rather than hot, water and limit your bath or shower to about 10 minutes.
* **Use a gentle, non soap cleanser.** Choose a cleanser that's free of dyes, alcohols and fragrance. For young children, you usually need only warm water to get them clean — no soap or bubble bath needed. Soap can be especially irritating to the skin of young children. For people of any age, deodorant soaps and antibacterial soaps can remove too much of the skin's natural oils and dry the skin. Don't scrub the skin with a washcloth or loofah.
* **Pat dry.** After bathing, gently pat the skin with a soft towel. Apply moisturizer while your skin is still damp (within three minutes).

The triggers for atopic dermatitis vary widely from person to person. Try to identify and avoid irritants that trigger your eczema. In general, avoid anything that causes an itch because scratching often triggers a flare.

Common triggers for atopic dermatitis include:

* Rough wool fabric
* Dry skin
* Skin infection
* Heat and sweat
* Stress
* Cleaning products
* Dust mites and pet dander
* Mold
* Pollen
* Smoke from tobacco
* Cold and dry air
* Fragrances
* Other irritating chemicals

Infants and children may have flares triggered by eating certain foods, such as eggs and cow's milk. Talk with your child's health care provider about identifying potential food allergies.

Once you understand what triggers your eczema, talk with your health care provider about how to manage your symptoms and prevent flares.

**When to see a doctor**

Talk with a health care provider if you or your child:

* Has symptoms of atopic dermatitis
* Is so uncomfortable that the condition is affecting sleep and daily activities
* Has a skin infection — look for new streaks, pus, yellow scabs
* Has symptoms even after trying self-care steps

**Seek immediate medical attention** if you or your child has a fever and the rash looks infected.

For atopic dermatitis, some basic questions you might ask your healthcare provider include:

* What might be causing my symptoms?
* Are tests needed to confirm the diagnosis?
* What treatment do you recommend, if any?
* Is this condition temporary or chronic?
* Can I wait to see if the condition goes away on its own?
* What are the alternatives to the approach you're suggesting?
* What skin care routines do you recommend to improve my symptoms?

### **What to expect from your doctor**

Your health care provider is likely to ask you a few questions. Being ready to answer them may free up time to go over any points you want to spend more time on. Your health care provider might ask:

* What are your symptoms and when did they start?
* Does anything seem to trigger your symptoms?
* Do you or any family members have allergies or asthma?
* Are you exposed to any possible irritants from your job or hobbies?
* Have you felt depressed or been under any unusual stress lately?
* Do you come in direct contact with pets or other animals?
* What products do you use on your skin, including soaps, lotions and cosmetics?
* What household cleaning products do you use?
* How much do your symptoms affect your quality of life, including your ability to sleep?
* What treatments have you tried so far? Has anything helped?
* How often do you shower or bathe?

**Differential diagnosis for atopic dermatitis**

The list of differential diagnoses for atopic dermatitis is long. A short list of common and important diagnoses to consider in children include:

* Seborrhoeic dermatitis and psoriasis
* Genetic disorders with scaly skin, including inherited forms of ichthyosis, primary immunodeficiency diseases, and inherited metabolic disorders
* Contact dermatitis.

**What are the types of eczema?**

There are several types of eczema. Each type has unique triggers that can affect your skin’s barrier function, including:

* Atopic dermatitis.
* Contact dermatitis.
* Dyshidrotic eczema.
* Neurodermatitis.
* Nummular eczema.
* Seborrheic dermatitis.

It’s possible to have more than one type of eczema at the same time.

**Recent guidelines or updates**

Bleach Baths

There has been controversy over whether dilute bleach baths may help AD. The linked systematic review and meta-analysis synthesizing 10 RCTs16 revealed that the probability to improve AD severity by 50% with adjunctive dilute bleach baths was 32% vs 22% in the control group (moderate certainty).

There was little to no difference in adverse events, with mild events consisting of dry skin and irritation noted. Changes in other patient-important outcomes (e.g., itch, patient-reported disease severity, sleep quality, AD-related quality of life, and risk of AD flares) were uncertain. Given this relatively minor improvement, the panel suggests that dilute bleach baths may be beneficial in patients with moderate and severe AD. Written instructions will be needed to ensure that patients use the correct type and concentration of bleach. Some patients may not have access to a bathtub and may find bleach baths too much effort.

In patients with mild disease, the limited magnitude of improvement was not felt to justify the burden. Elimination Diets Patients with AD have a higher risk for food allergies than those without AD. Food allergy testing and elimination diets are often considered to try to inform how to improve AD control. Recent evidence, however, suggests that tolerance to food allergens is promoted through frequent, and perhaps high-dose, oral exposure. Avoidance of food allergens is therefore strongly associated with promoting the development of IgE-mediated food allergy.

The linked systematic review and meta-analysis identified 10 RCTs (599 participants) addressing benefits and harms of dietary elimination for AD.17 Compared with no dietary elimination, low-certainty evidence revealed that dietary elimination may slightly improve AD severity (50% with vs 41% without dietary elimination improved by a minimally important difference, risk difference [RD] of 9% [95% CI, 0-17]), pruritus (daytime itch score [range, 0-3] mean difference [MD], 0.21 [95% CI, 0.57 to 0.15]), and sleeplessness (sleeplessness score [range, 0-3] MD, 0.47 [95% CI, 0.80 to 0.13]). Bayesian sensitivity analyses revealed that most individuals pursuing a diet elimination strategy would most likely experience little to no benefit. The JTF panel suggests against the use of elimination diets compared with an unrestricted diet. Between both the uncertain benefits and uncertain harms,17 including the potential risk of promoting food allergy, the panel inferred that most well-informed patients would place a higher value on avoiding potentially large harms. This was particularly the case in infants and children whom the risk for developing food allergy is thought to be greater. All ages, however, were thought to be at risk of malnutrition and burdensome to patients and their caregivers with following a strict dietary elimination strategy. Allergen Immunotherapy The previous practice parameter noted that AIT could be effective for AD. This guideline’s linked systematic review of 23 RCTs (11 subcutaneous immunotherapy [SCIT] and 12 sublingual immunotherapy [SLIT]) included 1957 adult and pediatric patients (median of study mean ages, 19 years; range of means, 4-34 years).

Most studies desensitized patients to house dust mites (HDMs; Dermatophagoides pteronyssinus and/or D farinae), whereas 4 included other inhaled allergens (e.g., pollens). Patients were mostly on standard topical therapy including TCSs and moisturizers with AIT added on. Furthermore, most studies included polysensitized patients in addition to HDM sensitization. Based on a combination of clinician-reported AD severity (e.g., SCORing Atopic Dermatitis [SCORAD]), AIT likely improved AD severity by 50% or more from baseline compared with no AIT (40% vs 26%), with similar estimates of effect for SCIT and SLIT. The main adverse effects were like AIT for allergic rhinitis and asthma, that is, local injection site reaction for SCIT (66% of individuals) and oropharyngeal itching for SLIT (13% of individuals). Systemic reactions or those severe enough to cause discontinuation occurred in approximately 10% of those receiving SCIT and were rare with SLIT (0.14% systemic reaction; 1.2% discontinue).

The panel inferred that most well-informed patients would value the moderate certainty for net benefit with AIT for moderate and severe AD especially if the patient had other allergic diseases that would respond to AIT. The panel noted that there would be variability in patient values and preferences regarding the burden associated with SCIT (multiple clinician visits for administration; often starting as weekly) and SLIT (daily self-administered medication) and time to effect. Systemic Treatments Including Ultraviolet Phototherapy (Light Therapy) There are multiple approved options for systemic treatment of AD refractory to, at least, topical therapy. Such patients will often have moderate-severe disease. These therapies include biologics, small molecules (mostly immunosuppressants), and UV light therapy (phototherapy).

The currently approved biologics target IL-4 and IL-13 cytokine signaling pathways, or IL-13 signaling alone. Dupilumab binds a common receptor IL-4Ra and inhibits IL-4R signaling induced by both IL-4 and IL-13. Tralokinumab binds to the IL-13 cytokine in an epitope that overlaps with the binding site of the IL-13Ra receptors, preventing IL13 from binding to the receptor. The linked systematic review and network meta-analysis (NMA) revealed that, compared with continued standard topical treatment alone, adding dupilumab or tralokinumab led to improvements in multiple patient-important outcomes. The improved outcomes included AD signs and symptoms, judged either by patients or clinicians, itch, and sleep disturbance. There was no clear increase in serious adverse events or adverse events leading to discontinuation. Conjunctivitis, however, was higher with dupilumab or tralokinumab in comparison to placebo.

The linked systematic review of patient values and preferences for treatment of AD,20 along with direct patient and caregiver input, revealed that patients with AD value stepping-up therapy based on severity, safe medications, relief and normalization of daily activities, and a strong patient provider relationship, despite the need for injections and potential fear of needles. Compared with dupilumab, tralokinumab was one category lower in efficacy across multiple patient-important outcomes. Tralokinumab is approved for AD in ages 12 years and older. Dupilumab is approved for children/adults aged 6 months and older for AD and asthma (ages 6 years and older), eosinophilic esophagitis (ages 12 years and older), and, for adults, chronic rhinosinusitis with nasal polyposis and prurigo nodularis. Patients and caregivers may also value having one systemic therapy treat multiple conditions. There are multiple oral JAK inhibitors currently available and additional ones in development.

The linked systematic review and NMA revealed that the benefits and harms of JAK inhibitors (in alphabetical order), abrocitinib, baricitinib, and upadacitinib, varied by drug and increased with dose of each medication. Although mild and common harms (e.g. acne, minor infection) increased with the dose of each medication, data addressing less common serious harms were hampered by the short duration of studies (16 weeks typically).For example, although serious infections such as herpetic were consistently increased in patients with AD using all 3 studied oral JAK inhibitors, there were no clear increase in deaths, cancer, or thrombosis detected in the short studies done.

The FDA placed a Boxed Warning label on the oral JAK inhibitors due to a recent study in rheumatoid arthritis using tofacitinib. The risk-benefit profile of JAK inhibitors should be considered when selecting JAK inhibitors in clinical practice. Risk considerations should include both observed safety data for the individual drugs from clinical trials of patients with AD and class-wide theoretical safety concerns and boxed warnings for JAK inhibitors from the US FDA. Oral JAK inhibitors are contraindicated in pregnancy and breastfeeding.

Risk factors for adverse outcomes, including age or other strong risk factors for cancer, serious infection, venous thrombosis, or cardiovascular disease, favor against JAK inhibitor use in these populations. JAK inhibitors are immunosuppressants and therefore screening for conditions before use (e.g., age-appropriate cancer screening, active or latent tuberculosis or viral hepatitis, vaccination including herpes zoster, cytopenias, diverticular disease or bowel perforation, renal and liver function, pregnancy) and subsequent clinician and patient monitoring for adverse effects are required. These can range in severity from acne, abdominal pain, easy bruising, tiredness, and blood abnormalities (lipids and other biochemistries, cell counts) to the serious harms described previously. There are thus multiple implementation considerations, including drug-drug interactions, laboratory and clinical monitoring, FDA approved doses, and practical considerations.  
  
**statistics or epidemiology data**

The prevalence of AD was 8.5%, which is much higher than the prevalence of AD reported in various parts of Nigeria 15 years ago. AD occurred before the age of 10 years in 523 (51.3%) patients, whilst 250 (24.5%) had onset after 21 years. The earliest age of onset in infants was in the first 6 weeks of life, and this was found in 129 patients (12.7%). Education and occupation of household heads were the most significant (P < 0.001) factors associated with seeking proper health care for the child's AD. Four hundred and forty-one (43.3%) patients presented with subacute atopic eczema and 326 (32%) patients with severe impetiginized eczema. Four hundred and twenty-five patients (41.7%) had at least one first-degree family member with AD (16.7%), allergic rhinitis (10.3%), asthma (14.6%) and allergic conjunctivitis (2.1%), while 55 (13.3%) of controls had a positive family history (P < 0.01) of allergy. A personal history of AD only, without concomitant respiratory allergies, was seen in 486 (47.7%) patients. The face was affected in 431 (42.3%) patients. Inverse distribution of a flexural rash was observed over the extensor aspect of the joints: the elbow in 502 patients (49.3%), the wrist joint in 183 patients (17.9%) and the knee joints in 354 patients (34.7). The commonly observed minor features included xerosis in 719 patients (71%), papular lichenoid lesions in 547 patients (54.1%), infraorbital folds in 498 patients (49.2%), palmar hyper linearity in 524 patients (51.8%) and raised peripheral blood eosinophils in 519 patients (51%), particularly for those with severe AD. Fissured heels, forehead lichenification, orbital darkening, nail pitting, sand paper-like skin lesions on the elbows/knees/lateral malleolli, knuckle dermatitis of the hands, palmar erythema and pitted keratolysis occurred more uncommonly as minor features. Infective complications were very common and included bacterial infections (folliculitis, impetiginized dermatitis and pyodermas) in 425 (41.7%) patients, fungal infections in 377 (37%) patients, parasitic infections (scabies) in 90 (8.8%) patients and viral infection (herpes simplex and molluscum contagiosum) in 29 (2.9%) patients. Thirteen of these atopics were also HIV positive. Aggravating factors most reported included heat intolerance, excessive sweating, humidity, grass intolerance, thick woolen clothing and drug reactions. Only three patients had food intolerance. Three hundred and ten patients (30.4%) recalled their AD being worse in the hot humid periods and 383 (37.6%) could not recall any periods of relief or remission.

The prevalence of AD amongst south-eastern Nigerian Blacks is on the increase, as in other areas, although it is still lower here than in other parts of the world. Many conventional minor features were found, but some occurred less frequently than in other countries, which may be attributed to ethnicity. Further studies will be required to confirm the ethnic differences in these features of AD amongst Nigerians and other Africans, to clarify the features of AD that are peculiar to Africans.

REFERENCES

<https://pubmed.ncbi.nlm.nih.gov/15485531/>

<https://www.aaaai.org/Aaaai/media/Media-Library-PDFs/Allergist%20Resources/Statements%20and%20Practice%20Parameters/JTF-Atopic-Dermatiti>

<https://www.mayoclinic.org/diseases-conditions/atopic-dermatitis-eczema/diagnosis-treatment/drc-20353279>

**Cold sores, or fever blisters**  
Other names for a cold sore are **fever** blister, oral herpes, labial herpes, herpes labialis, and herpes febrilis.

**Definition and description**

Cold sores, or fever blisters, are a common viral infection. They are tiny, fluid-filled blisters on and around the lips. These blisters are often grouped together in patches. After the blisters break, a scab forms that can last several days. Cold sores usually heal in 2 to 3 weeks without leaving a scar.

Cold sores spread from person to person by close contact, such as kissing. They're usually caused by herpes simplex virus type 1 (HSV-1), and less commonly herpes simplex virus type 2 (HSV-2). Both viruses can affect the mouth or genitals and can be spread by oral sex. The virus can spread even if you don't see the sores.

There's no cure for cold sores, but treatment can help manage outbreaks. Prescription antiviral medicine or creams can help sores heal more quickly. And they may make future outbreaks happen less often and be shorter and less serious.

**Causes**

Cold sores are caused by certain strains of the herpes simplex virus (HSV). HSV-1 usually causes cold sores. HSV-2 is often the cause of genital herpes. But either type can spread to the face or genitals through close contact, such as kissing or oral sex. Shared eating utensils, razors and towels can also spread HSV-1.

Cold sores are most likely to spread when you have oozing blisters. But you can spread the virus even if you don't have blisters. Many people who are infected with the virus that causes cold sores never develop symptoms.

Once you've had a herpes infection, the virus can hide in nerve cells in the skin and may cause another cold sore at the same place as before. A return of cold sores may be triggered by:

* Viral infection or fever.
* Hormonal changes, such as those related to a menstrual period.
* Stress.
* Fatigue.
* Being in the sun or wind.
* Changes in the immune system.
* Injury to the skin.

**Risk factors**

Almost everyone is at risk of cold sores. Most adults carry the virus that causes cold sores, even if they've never had symptoms.

You're most at risk of complications from the virus if you have a weak immune system from conditions and treatments such as:

* HIV/AIDS.
* Atopic dermatitis (eczema).
* Cancer chemotherapy.
* Anti-rejection medicine for organ transplants.

**Symptoms**

A cold sore usually passes through several stages:

* **Tingling and itching.** Many people feel itching, burning or tingling around the lips for a day or so before a small, hard, painful spot appears and blisters form.
* **Blisters.** Small fluid-filled blisters often form along the border of the lips. Sometimes they appear around the nose or cheeks or inside the mouth.
* **Oozing and crusting.** The small blisters may merge and then burst. This can leave shallow open sores that ooze and crust over.

Symptoms vary, depending on whether this is your first outbreak or a recurrence. The first time you have a cold sore; symptoms may not start for up to 20 days after you were first exposed to the virus. The sores can last several days. And the blisters can take 2 to 3 weeks to heal completely. If blisters return, they'll often appear at the same spot each time and tend to be less severe than the first outbreak.

In a first-time outbreak, you also might experience:

* Fever.
* Painful gums.
* Sore throat.
* Headache.
* Muscle aches.
* Swollen lymph nodes.

Children under 5 years old may have cold sores inside their mouths. These sores are often mistaken for canker sores. Canker sores involve only the mucous membrane and aren't caused by the herpes simplex virus.

**Diagnosis**

Your health care provider can usually diagnose cold sores just by looking at them. To confirm the diagnosis, your health care provider might take a sample from the blister for testing in a laboratory.

**Treatment**

Cold sores often clear up without treatment in 2 to 4 weeks. Your doctor might prescribe antiviral medicine that may speed the healing process. Examples include:

* Acyclovir (Zovirax).
* Valacyclovir (Valtrex).
* Famciclovir.
* Penciclovir (Denavir).

Some of these products are pills. Others are creams you put on the sores several times a day. In general, the pills work better than the creams. For very severe infections, some antiviral medicines can be injected.

**Lifestyle and home remedies**

The cold sore ointment docosanol (Abreva) may shorten the healing time of a cold sore. At the first sign of symptoms, apply it to the affected skin as directed on the package. Use a cotton-tipped swab to put medicine on a cold sore. This helps prevent the spread of the sores to other parts of the body.

To ease the discomfort of a cold sore:

* **Try other cold sore remedies.** Some nonprescription products contain a drying agent, such as alcohol, that may speed healing.
* **Use lip balms and cream.** Protect your lips from the sun with a zinc oxide cream or lip balm with sunblock. If your lips become dry, apply a moisturizing cream.
* **Apply a compress.** A cold, damp cloth may ease symptoms and help remove crusting. Or try using a warm cloth on the blisters to ease pain.
* **Rest and try pain relievers.** Take non-prescription pain medicine if you have a fever or the cold sore is painful. Creams with lidocaine or benzocaine may offer some pain relief.

**Alternative medicine**

Studies about whether alternative medicine helps with cold sores have had mixed results. Some approaches that people use for cold sores include:

* **Lysine.** An amino acid, lysine is available as an oral supplement and as a cream.
* **Rhubarb and sage.** A cream combining rhubarb and sage may be about as effective as acyclovir (Zovirax) cream.
* **Stress reduction.** If your cold sores are brought on by stress, you might want to try relaxation techniques. Examples include deep-breathing exercises and meditation.
* **Propolis.** This is available as a 3% ointment. When applied early and often, it might shorten the duration of the breakout. This product is also called synthetic beeswax.

**Preparing for your appointment**

Cold sores often clear up without treatment in 2 to 4 weeks. Make an appointment with your primary care provider if your cold sores:

* Are lasting or severe.
* Return often.
* Are accompanied by eye pain or gritty eyes.

### **What you can do**

Before your appointment, you may want to list answers to the following questions:

* Have you ever had these symptoms before?
* Do you have a history of skin problems?
* What medicines and supplements do you take regularly?

Below are some basic questions to ask your health care provider about cold sores.

* Do I have a cold sore?
* What treatment do you recommend, if any?
* What self-care steps might ease my symptoms?
* Can I spread this condition to others? For how long?
* How do I reduce the risk of spreading this condition to others?
* How soon do you expect my symptoms will improve?
* Am I at risk of complications from this condition?
* Can I do anything to help prevent another blister?

### **What to expect from your doctor**

Your health care provider is likely to ask you a few questions. Being ready to answer them may reserve time to go over any points you want to talk about in-depth. Your health care provider may ask:

* Could you sense a cold sore coming before you could see it?
* Do your symptoms include eye pain or gritty eyes?
* Does anything seem to bring on your symptoms?
* Have you been treated for cold sores in the past? If so, what treatment helped the most?
* Have you been under stress lately or had major life changes?
* Are you pregnant?
* Does your work or home life bring you into contact with infants or with people who have major illness?

**Complications**

In some people, the virus that causes cold sores can cause problems in other areas of the body, including:

* **Fingertips.** Both HSV-1 and HSV-2 can be spread to the fingers. This type of infection is often referred to as herpes whitlow. Children who suck their thumbs may transfer the infection from their mouths to their thumbs.
* **Eyes.** The virus can sometimes cause eye infection. Repeated infections can cause scarring and injury, which may lead to vision problems or loss of vision.
* **Widespread areas of skin.** People who have a skin condition called atopic dermatitis (eczema) are at higher risk of cold sores spreading across their bodies. This can become a medical emergency.

**Prevention**

Your health care provider may prescribe an antiviral medicine for you to take on a regular basis if you develop cold sores more than nine times a year or if you're at high risk of serious complications. If sunlight seems to trigger your condition, apply sunblock to the spot where the cold sore tends to form. Or talk with your health care provider about using an oral antiviral medicine before you do an activity that tends to cause a cold sore to return.

Take these steps to help avoid spreading cold sores to other people:

* **Avoid kissing and skin contact with people while blisters are present.** The virus spreads most easily when the blisters leak fluid.
* **Avoid sharing items.** Utensils, towels, lip balm and other personal items can spread the virus when blisters are present.
* **Keep your hands clean.** When you have a cold sore, wash your hands carefully before touching yourself and other people, especially babies.

### **What are the cold sore stages?**

Cold sores develop and go away over the course of one to two weeks. Here’s what you can expect for a typical cold sore outbreak:

* **Day 1.** You notice tingling, itching, pain or numbness on your lip or nearby skin. This is the area where cold sores will form. Healthcare providers call this the prodromal (pronounced “proh-DROH-mul”) stage. It means HSV has reactivated in your nerve cells and started making copies of itself (replicating). The prodromal stage is a warning sign that cold sores are about to form.
* **Days 1 to 2.** Within 24 hours of the start of the prodromal stage, bumps form on or around your lips (most often, along the outer edge). On average, three to five bumps form, but you could have more or fewer. Within hours, the bumps fill up with fluid and become blister-like. The area becomes red/discolored, swollen and painful.
* **Days 2 to 3.** The blisters break open (rupture) and ooze a clear or slightly yellow fluid. This is sometimes called the “weeping phase.”
* **Days 3 to 4.** The blisters stop oozing and a crust forms. The crust often looks like a golden-brown scab. It covers the sore as it heals but may sometimes crack open or bleed.
* **By day 14**. The scab usually falls off within six to 14 days of the start of the outbreak. The skin underneath may be a little more pink or red than usual for a few days before fully healing.

#### **How long does a cold sore last?**

Cold sores usually last one to two weeks. Symptoms may be more severe, and sores may take longer to heal if you’re immunocompromised.

**Prognosis**

Cold sores develop in response to the herpes simplex virus type 1 (HSV-1). Once a person contracts the virus, it lays dormant and can flare up, resulting in a cold sore. Most people recover completely from a cold sore outbreak without treatment.

People who experience frequent outbreaks may wish to look into antiviral therapy to help reduce the number of outbreaks and the risk of spreading the HSV to others.

Anyone who gets cold sores should be careful to avoid spreading them, especially to babies, children, and those with weaker immune systems.

**Differential diagnosis of cold sores:**

Aphthous ulcers - are not unilateral and are more likely to be on non-keratinized mucosa.

Chickenpox.

Impetigo.

Lip cancer.

Primary oral chancre of syphilis.

**Signs of possible oral cancer include**:

Ulceration of the oral mucosa persisting for more than three weeks.

Oral swellings persisting for more than three weeks.

All red or red and white patches of the oral mucosa.

The level of suspicion is further increased if the person is a heavy smoker, heavy alcohol drinker, aged over 45 years or male.

**Epidemiology**

Herpes labialis is common throughout the world. A large survey of young adults on six continents reported that 33% of males and 28% of females had herpes labialis on two or more occasions during the year before the study. The lifetime prevalence in the United States of America is estimated at 20–45% of the adult population. Lifetime prevalence in France was reported by one study as 32% in males and 42% in females. In Germany, the prevalence was reported at 32% in people aged between 35 and 44 years, and 20% in those aged 65–74. In Jordan, another study reported a lifetime prevalence of 26%.

**REFERENCES**

<https://www.mayoclinic.org/diseases-conditions/cold-sore/symptoms-causes/syc-20371017>

<https://www.mayoclinic.org/diseases-conditions/cold-sore/diagnosis-treatment/drc-20371023>

[Cold Sores (Causes, Symptoms, and Treatment)](https://patient.info/childrens-health/viral-skin-infections-leaflet/cold-sores)

**Psoriasis**

**Definition and description**

Psoriasis is a skin disease that causes a rash with itchy, scaly patches, most commonly on the knees, elbows, trunk and scalp.

Psoriasis is a common, long-term (chronic) disease with no cure. It can be painful, interfere with sleep and make it hard to concentrate. The condition tends to go through cycles, flaring for a few weeks or months, then subsiding for a while. Common triggers in people with a genetic predisposition to psoriasis include infections, cuts or burns, and certain medications.

Treatments are available to help you manage symptoms. And you can try lifestyle habits and coping strategies to help you live better with psoriasis.

**Causes**

Psoriasis is thought to be an immune system problem that causes skin cells to grow faster than usual. In the most common type of psoriasis, known as plaque psoriasis, this rapid turnover of cells results in dry, scaly patches.

The cause of psoriasis isn't fully understood. It's thought to be an immune system problem where infection-fighting cells attack healthy skin cells by mistake. Researchers believe that both genetics and environmental factors play a role. The condition is not contagious.

### **Psoriasis triggers**

Many people who are predisposed to psoriasis may be free of symptoms for years until the disease is triggered by some environmental factor. Common psoriasis triggers include:

* Infections, such as strep throat or skin infections
* Weather, especially cold, dry conditions
* Injury to the skin, such as a cut or scrape, a bug bite, or a severe sunburn
* Smoking and exposure to secondhand smoke
* Heavy alcohol consumption
* Certain medications — including lithium, high blood pressure drugs and antimalarial drugs
* Rapid withdrawal of oral or injected corticosteroids.

**Risk factors**

Anyone can develop psoriasis. About a third of instances begin in childhood. These factors can increase the risk of developing the disease:

* **Family history.** The condition runs in families. Having one parent with psoriasis increases your risk of getting the disease. And having two parents with psoriasis increases your risk even more.
* **Smoking.** Smoking tobacco not only increases the risk of psoriasis but also may increase the severity of the disease.

**Signs and symptoms**

Common signs and symptoms of psoriasis include:

* A patchy rash that varies widely in how it looks from person to person, ranging from spots of dandruff-like scaling to major eruptions over much of the body
* Rashes that vary in color, tending to be shades of purple with gray scale on brown or Black skin and pink or red with silver scale on white skin
* Small scaling spots (commonly seen in children)
* Dry, cracked skin that may bleed
* Itching, burning or soreness
* Cyclic rashes that flare for a few weeks or months and then subside

There are several types of psoriasis, each of which varies in its signs and symptoms:

* **Plaque psoriasis.** The most common type of psoriasis, plaque psoriasis causes dry, itchy, raised skin patches (plaques) covered with scales. There may be few or many. They usually appear on the elbows, knees, lower back and scalp. The patches vary in color, depending on skin color. The affected skin might heal with temporary changes in color (post inflammatory hyperpigmentation), particularly on brown or Black skin.
* **Nail psoriasis.** Psoriasis can affect fingernails and toenails, causing pitting, abnormal nail growth and discoloration. Psoriatic nails might loosen and separate from the nail bed (onycholysis). Severe disease may cause the nail to crumble.
* **Guttate psoriasis.** Guttate psoriasis primarily affects young adults and children. It's usually triggered by a bacterial infection such as strep throat. It's marked by small, drop-shaped, scaling spots on the trunk, arms or legs.
* **Inverse psoriasis.** Inverse psoriasis mainly affects the skin folds of the groin, buttocks and breasts. It causes smooth patches of inflamed skin that worsen with friction and sweating. Fungal infections may trigger this type of psoriasis.
* **Pustular psoriasis.** Pustular psoriasis, a rare type, causes clearly defined pus-filled blisters. It can occur in widespread patches or on small areas of the palms or soles.
* **Erythrodermic psoriasis.** The least common type of psoriasis, erythrodermic psoriasis can cover the entire body with a peeling rash that can itch or burn intensely. It can be short-lived (acute) or long-term (chronic).

**Diagnosis**

Your health care provider will ask questions about your health and examine your skin, scalp and nails. Your health care provider then might take a small sample of skin (biopsy) for examination under a microscope. This helps determine the type of psoriasis and rule out other disorders.

**Treatment**

Psoriasis treatments aim to stop skin cells from growing so quickly and to remove scales. Options include creams and ointments (topical therapy), light therapy (phototherapy), and oral or injected medications.

Which treatments you use depends on how severe the psoriasis is and how responsive it has been to previous treatment and self-care measures. You might need to try different drugs or a combination of treatments before you find an approach that works. Even with successful treatment, usually the disease returns.

### **Topical therapy**

* **Corticosteroids.** These drugs are the most frequently prescribed medications for treating mild to moderate psoriasis. They are available as oils, ointments, creams, lotions, gels, foams, sprays and shampoos. Mild corticosteroid ointments (hydrocortisone) are usually recommended for sensitive areas, such as the face or skin folds, and for treating widespread patches. Topical corticosteroids might be applied once a day during flares, and on alternate days or weekends during remission.

Your health care provider may prescribe a stronger corticosteroid cream or ointment — triamcinolone (Trianex) or clobetasol (Cormax, Temovate, others) — for smaller, less-sensitive or tougher-to-treat areas.

Long-term use or overuse of strong corticosteroids can thin the skin. Over time, topical corticosteroids may stop working.

* **Vitamin D analogues.** Synthetic forms of vitamin D — such as calcipotriene (Dovonex, Sorilux) and calcitriol (Vectical) — slow skin cell growth. This type of drug may be used alone or with topical corticosteroids. Calcitriol may cause less irritation in sensitive areas. Calcipotriene and calcitriol are usually more expensive than topical corticosteroids.
* **Retinoids.** Tazarotene (Tazorac, Avage, others) is available as a gel or cream. It's applied once or twice daily. The most common side effects are skin irritation and increased sensitivity to light.

Tazarotene isn't recommended when you're pregnant or breastfeeding or if you intend to become pregnant.

* **Calcineurin inhibitors.** Calcineurin inhibitors — such as tacrolimus (Protopic) and pimecrolimus (Elidel) — calm the rash and reduce scaly buildup. They can be especially helpful in areas of thin skin, such as around the eyes, where steroid creams or retinoids are irritating or harmful.

Calcineurin inhibitors aren't recommended when you're pregnant or breastfeeding or if you intend to become pregnant. This drug is also not intended for long-term use because of a potential increased risk of skin cancer and lymphoma.

* **Salicylic acid.** Salicylic acid shampoos and scalp solutions reduce the scaling of scalp psoriasis. They are available in nonprescription or prescription strengths. This type of product may be used alone or with other topical therapy, as it prepares the scalp to absorb the medication more easily.
* **Coal tar.** Coal tar reduces scaling, itching and inflammation. It's available in nonprescription and prescription strengths. It comes in various forms, such as shampoo, cream and oil. These products can irritate the skin. They're also messy, stain clothing and bedding, and can have a strong odor.

Coal tar treatment isn't recommended when you're pregnant or breastfeeding.

* **Anthralin.** Anthralin is a tar cream that slows skin cell growth. It can also remove scales and make skin smoother. It's not intended for use on the face or genitals. Anthralin can irritate skin, and it stains almost anything it touches. It's usually applied for a short time and then washed off.

### **Light therapy**

Light therapy is a first line treatment for moderate to severe psoriasis, either alone or in combination with medications. It involves exposing the skin to controlled amounts of natural or artificial light. Repeated treatments are necessary. Talk with your health care provider about whether home phototherapy is an option for you.

* **Sunlight.** Brief, daily exposures to sunlight (heliotherapy) might improve psoriasis. Before beginning a sunlight regimen, ask your healthcare provider about the safest way to use natural light for psoriasis treatment.
* **UVB broadband.** Controlled doses of UVB broadband light from an artificial light source can treat single psoriasis patches, widespread psoriasis and psoriasis that doesn't improve with topical treatments. Short-term side effects might include inflamed, itchy, dry skin.
* **UVB narrowband.** UVB narrowband light therapy might be more effective than UVB broadband treatment. In many places it has replaced broadband therapy. It's usually administered two or three times a week until the skin improves and then less frequently for maintenance therapy. But narrowband UVB phototherapy may cause more-severe side effects than UVB broadband.
* **Psoralen plus ultraviolet A (PUVA).** This treatment involves taking a light-sensitizing medication (psoralen) before exposing the affected skin to UVA light. UVA light penetrates deeper into the skin than does UVB light, and psoralen makes the skin more responsive to UVA exposure.

This more aggressive treatment consistently improves skin and is often used for more-severe psoriasis. Short-term side effects might include nausea, headache, burning and itching. Possible long-term side effects include dry and wrinkled skin, freckles, increased sun sensitivity, and increased risk of skin cancer, including melanoma.

* **Excimer laser.** With this form of light therapy, a strong UVB light targets only the affected skin. Excimer laser therapy requires fewer sessions than does traditional phototherapy because more-powerful UVB light is used. Side effects might include inflammation and blistering.

### **Oral or injected medications**

If you have moderate to severe psoriasis, or if other treatments haven't worked, your health care provider may prescribe oral or injected (systemic) drugs. Some of these drugs are used for only brief periods and might be alternated with other treatments because they have potential for severe side effects.

* **Steroids.** If you have a few small, persistent psoriasis patches, your health care provider might suggest an injection of triamcinolone right into them.
* **Retinoids.** Acitretin and other retinoids are pills used to reduce the production of skin cells. Side effects might include dry skin and muscle soreness. These drugs are not recommended when you're pregnant or breastfeeding or if you intend to become pregnant.
* **Biologics.** These drugs, usually administered by shot, alter the immune system in a way that disrupts the disease cycle and improves symptoms and signs of disease within weeks. Several of these drugs are approved for the treatment of moderate to severe psoriasis in people who haven't responded to first line therapies. Options include etanercept (Enbrel), infliximab (Remicade), adalimumab (Humira), ustekinumab (Stelara), risankizumab-rzaa (Skyrizi), ixekizumab (Taltz), guselkumab (Tremfya), apremilast (Otezla), bimekizumab-bkzx (Bimzelx) and secukinumab (Cosentyx). Four of them — etanercept, ixekizumab, secukinumab and ustekinumab — are approved for children. These types of drugs are expensive and may or may not be covered by health insurance plans.

Biologics must be used with caution because they carry the risk of suppressing the immune system in ways that increase the risk of serious infections. People taking these treatments must be screened for tuberculosis.

* **Methotrexate.** Usually administered weekly as a single oral dose, methotrexate (Trexall) decreases the production of skin cells and suppresses inflammation. It's less effective than adalimumab and infliximab. It might cause an upset stomach, loss of appetite and fatigue. People taking methotrexate long-term need ongoing testing to monitor their blood counts and liver function.

People need to stop taking methotrexate at least three months before attempting to conceive. This drug is not recommended for those who are breastfeeding.

* **Cyclosporine.** Taken orally for severe psoriasis, cyclosporine (Gengraf, Neoral, Sandimmune) suppresses the immune system. It's similar to methotrexate in effectiveness but cannot be used continuously for more than a year. Like other immunosuppressant drugs, cyclosporine increases the risk of infection and other health problems, including cancer. People taking cyclosporine long-term need ongoing testing to monitor their blood pressure and kidney function.

These drugs aren't recommended when you're pregnant or breastfeeding or if you intend to become pregnant.

* **Other medications.** Thioguanine (Tabloid) and hydroxyurea (Droxia, Hydrea) are medications that can be used when you can't take other drugs. Talk with your health care provider about possible side effects of these drugs.

### **Treatment considerations**

You and your health care provider will choose a treatment approach based on your needs and the type and severity of your psoriasis. You'll likely start with the mildest treatments — topical creams and ultraviolet light therapy (phototherapy). Then, if your condition doesn't improve, you might move on to stronger treatments.

People with pustular or erythrodermic psoriasis usually need to start with stronger (systemic) medications.

In any situation, the goal is to find the most effective way to slow cell turnover with the fewest possible side effects.

### **Alternative medicine**

Some studies claim that alternative therapies (integrative medicine) — products and practices not part of conventional medical care or that developed outside of traditional Western practice — ease the symptoms of psoriasis.

Examples of alternative therapies used by people with psoriasis include special diets, vitamins, acupuncture and herbal products applied to the skin. None of these approaches is backed by strong evidence, but they are generally safe and might help reduce itching and scaling in people with mild to moderate psoriasis.

* **Aloe extract cream.** Taken from the leaves of the aloe vera plant, aloe extract cream may reduce scaling, itching and inflammation. You might need to use the cream several times a day for a month or more to see any improvement in your skin.
* **Fish oil supplements.** Oral fish oil therapy used in combination with UVB therapy might reduce the extent of the rash. Applying fish oil to the affected skin and covering it with a dressing for six hours a day for four weeks might improve scaling.
* **Oregon grape.** Oregon grape — also known as barberry — is applied to the skin and may reduce the severity of psoriasis.

If you're considering alternative medicine to ease the signs and symptoms of psoriasis, talk with your health care provider about the pros and cons of these approaches.

**Lifestyle and home remedies**

Try these self-care measures to better manage your psoriasis:

* **Take daily baths.** Wash gently rather than scrubbing your skin in the shower or bath. Use lukewarm water and mild soaps that have added oils or fats. It might help to add bath oil, Epsom salts or oatmeal to bathwater and soak for at least 15 minutes.
* **Keep your skin moist.** Apply moisturizer daily. If you're moisturizing after bathing, gently pat dry and apply your preferred product while your skin is still moist. For very dry skin, oils or heavy ointment-based moisturizers may be preferable — they stay on the skin longer than creams or lotions do. If moisturizing seems to improve your skin, apply the product more than once a day.

If the air where you live is very dry, use a humidifier to add moisture to the air.

* **Cover the affected areas overnight.** Before going to bed, apply an ointment-based moisturizer to the affected skin and wrap with plastic wrap. When you wake, remove the plastic and wash away the scales.
* **Expose your skin to small amounts of sunlight.** Ask your healthcare provider about the best way to use natural sunlight to treat your skin. A controlled amount of sunlight can improve psoriasis, but too much sun can trigger or worsen outbreaks and increase the risk of skin cancer. Log your time in the sun and protect skin that isn't affected by psoriasis with a hat, clothing or sunscreen with a sun protection factor (SPF) of at least 30.
* **Avoid scratching.** It might help to apply a non-prescription anti-itch cream or ointment containing hydrocortisone or salicylic acid. If you have scalp psoriasis, try a medicated shampoo that contains coal tar. Keep your nails trimmed so that they won't hurt your skin if you do scratch. Wear soft fabrics that don't contribute to itchiness.
* **Avoid psoriasis triggers.** Notice what triggers your psoriasis and take steps to prevent or avoid it. Infections, injuries to your skin, smoking and intense sun exposure can all worsen psoriasis.
* **Stay cool.** Being too hot can make your skin feel itchy. Wear light clothing if you're outside on hot days. If you have air conditioning, use it on hot days to keep cool. Keep cold packs in your freezer and apply them to itchy spots for a few minutes of relief. You might try storing your moisturizing lotion in the refrigerator to add a cooling effect when you apply it
* **Strive to maintain a healthy lifestyle.** Try practicing other healthy-living habits to help manage psoriasis. These include being active, eating well, limiting or avoiding alcohol consumption, and maintaining a healthy weight.

**Prevention**

There isn’t a way to entirely prevent psoriasis. You can reduce your risk by following your healthcare provider’s treatment, living a healthy lifestyle, taking good care of your skin and avoiding triggers that can cause an outbreak of symptoms.

**Outlook / Prognosis**

If you have psoriasis, it’s common to see symptoms show up during early adulthood, but the timeline of when symptoms begin is unique to every person. You may notice certain triggers in your environment that can cause a flare up of symptoms. Avoiding these triggers can lead to fewer outbreaks in the future.

Psoriasis can make you uncomfortable, itchy and self-conscious. If these symptoms are causing you physical or emotional distress, contact your healthcare provider for treatment.

### **Is there a cure for psoriasis?**

There isn’t a cure for psoriasis. Psoriasis is a chronic condition, which means that symptoms may come and go throughout your life. Treatment can relieve symptoms so you can look and feel your best.

**Living With**

To feel your best with psoriasis:

* Take medications as instructed.
* Use moisturizer regularly, especially after bathing.
* Avoid harsh soaps.
* Use medicated shampoo for scales on your scalp.

Other steps you should take to stay as healthy as possible:

* Talk to your healthcare provider about lowering your risk for related conditions, such as heart disease, depression and diabetes.
* Lower your stress with meditation, exercise or seeing a mental health professional.

**Complications**

If you have psoriasis, you're at greater risk of developing other conditions, including:

* Psoriatic arthritis, which causes pain, stiffness, and swelling in and around the joints
* Temporary skin color changes (post-inflammatory hypopigmentation or hyperpigmentation) where plaques have healed
* Eye conditions, such as conjunctivitis, blepharitis and uveitis
* Obesity
* Type 2 diabetes
* High blood pressure
* Cardiovascular disease
* Other autoimmune diseases, such as celiac disease, sclerosis and the inflammatory bowel disease called Crohn's disease
* Mental health conditions, such as low self-esteem and depression

### **When to see a doctor**

If you suspect that you may have psoriasis, see your healthcare provider. Also seek medical care if your condition:

* Becomes severe or widespread
* Causes you discomfort and pain
* Causes you concern about the appearance of your skin
* Doesn't improve with treatment

## **Differential Diagnoses**

**Adult Blepharitis**  
A chronic inflammation of the eyelid margins causing redness, swelling, itching, crusting, and irritation. It can involve the front (anterior) eyelid affecting eyelashes or the back (posterior) eyelid affecting oil glands. Symptoms include gritty, burning eyes, crusty eyelashes, and eyelid sticking, often worse in the morning. It is associated with bacterial colonization, skin conditions like rosacea, seborrheic dermatitis, and dry eye syndrome

**Allergic Contact Dermatitis**  
An inflammatory skin reaction caused by exposure to allergens or irritants, leading to redness, itching, swelling, and sometimes blistering. It occurs when the immune system reacts to substances touching the skin.

**Atopic Dermatitis in Emergency Medicine**  
A chronic, pruritic inflammatory skin disease (eczema) characterized by red, scaly, itchy rashes often on flexural areas like elbows and knees. It may be complicated by infections or severe flares requiring urgent care. It is linked to other atopic conditions like asthma and allergic rhinitis

**Atopic Keratoconjunctivitis (AKC)**  
A chronic inflammatory eye disease affecting patients with atopic dermatitis, presenting with bilateral conjunctival inflammation, itching, burning, tearing, and mucoid discharge. It can cause serious ocular complications such as keratitis, cataracts, and corneal scarring. It involves both IgE-mediated and delayed hypersensitivity reactions

**Cutaneous Squamous Cell Carcinoma**  
A common type of skin cancer arising from squamous cells in the epidermis, often caused by UV exposure. It presents as scaly, red patches, open sores, or elevated growths that may crust or bleed.

**Diaper Dermatitis (Diaper Rash)**  
An inflammatory skin condition in infants caused by prolonged exposure to moisture, friction, and irritants in diapers. It presents as red, irritated skin in the diaper area.

**Dry Eye Disease (Keratoconjunctivitis Sicca)**  
A disorder of the tear film causing dryness, irritation, burning, and redness of the eyes. It can be associated with blepharitis and other eyelid conditions

**Gout and Pseudogout**  
Gout is a metabolic disorder characterized by deposition of urate crystals in joints causing acute inflammation and pain. Pseudogout involves calcium pyrophosphate crystal deposition causing similar symptoms.

**Lichen Planus**  
A chronic inflammatory condition affecting skin and mucous membranes, characterized by purplish, itchy, flat-topped papules.

**Lichen Simplex Chronicus**  
A skin disorder caused by chronic scratching or rubbing leading to thickened, scaly, itchy patches.

**Mycosis Fungoides**  
A type of cutaneous T-cell lymphoma presenting with patches, plaques, or tumors on the skin.

**Nummular Dermatitis (Nummular Eczema)**  
A chronic eczema variant characterized by round or oval itchy, scaly plaques on the skin.

**Onychomycosis**  
Fungal infection of the nails causing thickening, discoloration, and brittleness.

**Pityriasis Alba**  
A common skin condition in children causing pale, scaly patches on the face.

**Pityriasis Rosea**  
An acute, self-limiting skin rash starting with a single herald patch followed by a widespread scaly rash.

**Pustular Eruptions**  
Skin conditions characterized by the presence of pustules, which are pus-filled lesions.

**Reactive Arthritis**  
An inflammatory arthritis triggered by infection elsewhere in the body, often causing joint pain, conjunctivitis, and urethritis.

**Seborrheic Dermatitis**  
A chronic inflammatory skin condition causing flaky, greasy scales and redness, commonly on the scalp, face, and eyelids. It is associated with blepharitis and dandruff

**Sicca Keratoconjunctivitis**  
Another term for dry eye disease, involving dryness and inflammation of the conjunctiva and cornea.

**Subcorneal Pustulosis**  
A rare, chronic pustular skin disorder characterized by superficial pustules just beneath the stratum corneum.

**Syphilis**  
A sexually transmitted infection caused by Treponema pallidum, presenting in stages with varied symptoms including skin rashes, mucous membrane lesions, and systemic involvement.

Tinea in Emergency Medicine

**Epidemiology data**

41 164 records were identified, and 168 studies met the inclusion criteria. In adults, the incidence of psoriasis varied from 30.3 per 100 000 person years (95% confidence interval 26.6 to 34.1) in Taiwan to 321.0 per 100 000 person years in Italy. The prevalence of psoriasis varied from 0.14% (95% uncertainty interval 0.05% to 0.40%) in east Asia to 1.99% (0.64% to 6.60%) in Australasia. The prevalence of psoriasis was also high in western Europe (1.92%, 1.07% to 3.46%), central Europe (1.83%, 0.62% to 5.32%), North America (1.50%, 0.63% to 3.60%), and high income southern Latin America (1.10%, 0.36% to 2.96%).

Eighty-one percent of the countries of the world lack information on the epidemiology of psoriasis. The disease occurs more frequently in adults than in children. Psoriasis is unequally distributed across geographical regions; it is more frequent in high income countries and in regions with older populations. The estimates provided can help guide countries and the international community when making public health decisions on the appropriate management of psoriasis and assessing its natural history over time.

For psoriasis, some basic questions you might ask include:

* What might be causing my signs and symptoms?
* Do I need diagnostic tests?
* What treatments are available, and which do you recommend for me?
* What types of side effects can I expect?
* Will the treatment you recommended cause a remission in my symptoms?
* How quickly can I expect results?
* What are the alternatives to the primary approach you're suggesting?
* I have other medical conditions. How can I manage these conditions together?
* What skin care routines and products do you recommend to improve my symptoms?

**What part of my body will psoriasis affect?**

A psoriasis rash can show up anywhere on your skin. Psoriasis is common on your:

* Elbows and knees.
* Face and inside of your mouth.
* Scalp
* Fingernails and toenails.
* Genitals.
* Lower back.
* Palms and feet.

In most people, psoriasis covers a small area of their skin. In severe cases, the plaques connect and cover a large area of your body.

**What is psoriatic arthritis?**

Psoriatic arthritis is a type of arthritis that causes joint pain and swelling. Like psoriasis, psoriatic arthritis is an autoimmune condition that causes your immune system to function abnormally and cause symptoms. About 1 in 3 people diagnosed with psoriasis will also develop arthritis due to inflammation. Early treatment of psoriatic arthritis can reduce damage to your joints.

### **Is psoriasis the same as eczema?**

Psoriasis and eczema are two different skin conditions. Both conditions cause similar symptoms like discolored skin, a rash and itching. Psoriasis plaques cause areas of thick skin covered in scales. Eczema causes a rash of dry and bumpy skin. Eczema also typically causes more intense itching than psoriasis.

**RECENT GUIDELINE**

This guideline addresses multiple phototherapy treatment options ranging from widely used ultraviolet modalities to the combined use of photosensitizing agents to newer and less prevalent choices, which have demonstrated promise. The recommended dosing regimen, efficacy, and adverse effects of the various phototherapy modalities used as monotherapy or in combination with other psoriasis therapies to treat moderate-to-severe psoriasis in adults was assessed for each of the following phototherapy treatments:

* Narrowband UVB
* Broadband UVB
* Targeted UVB
  + Excimer laser
  + Excimer light
  + Targeted narrowband UVB light
* UVA with psoralens (PUVA)
  + Topical
  + Oral
  + Bath
* Photodynamic therapy
* Grenz ray
* Climatotherapy
* Visible light
* Goeckerman therapy (not a form of phototherapy)
* Pulsed dye laser (PDL)

A prior guideline was last published in 2009. This 2019 update provides significant additional scope including:

* Evidence-based use of phototherapy in different types of psoriasis
* Evidence-based use of phototherapy in combination with other treatment modalities
* New modalities and specific applications identified within the past decade
* Safety data including:
  + Adverse events
  + Contraindications
  + Pregnancy and lactation
  + Risk of malignancy
* Role of the dermatologist: Identifying those patients in whom phototherapy may be a viable or preferred treatment option, either as monotherapy or an adjunct, and working with the patient to outline risks and benefits and to make a joint decision on the best modality and dosing schedule.
* Role of patient preferences addresses the need of openly discussing safety and efficacy factors that may have an impact on patient decision to start certain treatments.
* Patient education referring to the importance of educating psoriasis patients regarding etiology, comorbidities and treatment options associated with psoriasis were discussed.

REFERENCES

<https://www.mayoclinic.org/diseases-conditions/psoriasis/diagnosis-treatment/drc-20355845>

[Psoriasis Differential Diagnoses](https://emedicine.medscape.com/article/1943419-differential?form=fpf)

[Psoriasis clinical guideline](https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

<https://www.mayoclinic.org/diseases-conditions/psoriasis/symptoms-causes/syc-20355840>

<https://my.clevelandclinic.org/health/diseases/6866-psoriasis>

**Rosacea**   
**Definition and description**

Rosacea (roe-ZAY-she-uh) is a common skin condition that causes flushing or long-term redness on your face. It also may cause enlarged blood vessels and small, pus-filled bumps. Some symptoms may flare for weeks to months and then go away for a while.

Rosacea can be mistaken for acne, dermatitis or other skin problems.

There's no cure for rosacea. But you may be able to control it with medicine, gentle skin care and avoiding things that cause flare-ups.

#### **What are the types of rosacea?**

The four different types of rosacea include:

* **Erythematotelangiectatic**: Rosacea is persistent and causes facial redness with enlarged and visible blood vessels (vascular). This type is characterized by flares, where symptoms come and go unexpectedly.
* **Papulopustular**: Pus- or fluid-filled pimples form on your skin. Your skin could swell, and symptoms are like acne.
* **Phymatous**: Symptoms cause your skin to swell and thicken. Your skin could be bumpy, and it most often affects your nose. Symptoms could make your nose appear bulbous (rhinophyma).
* **Ocular**: Rosacea can affect your eyes, causing them to feel irritated, bloodshot or watery. Your eyes may be sensitive to light, and painful bumps may form on your eyelids (styes).

### **How common is rosacea and who does rosacea affect?**

Rosacea affects more than 14 million people in the U.S. It can affect anyone, but it most often affects women and those with fair skin. Symptoms usually arise after age 30. The condition can affect children and adolescents, but it’s very rare. You’re more likely to have rosacea if someone in your family has it.

Studies suggest that men with rosacea may have more severe symptoms as a result of delaying treatment until the condition becomes advanced.  
  
**Causes**

The cause of rosacea is not known. It could be due to genetics, an overactive immune system or things in your daily life. Rosacea is not caused by poor hygiene, and you can't catch it from other people.

Flare-ups might be brought on by:

* Sun or wind.
* Hot drinks.
* Spicy foods.
* Alcohol.
* Very hot and cold temperatures.
* Emotional stress.
* Exercise.
* Drugs that dilate blood vessels, including some blood pressure medicines.
* Some cosmetic, skin and hair care products.

**Risk factors**

Anyone can develop rosacea. But you may be more likely to develop it if you:

* Have skin that burns easily in the sun.
* Are between the ages of 30 to 50 years.
* Have a history of smoking.
* Have a family member with rosacea.

**Signs and symptoms**

Symptoms of rosacea include:

* **Facial redness and flushing.** Rosacea can make your face flush more easily. Over time, you may notice that your face stays red. Depending on skin color, redness may be subtle or look more pink or purple.
* **Visible veins.** Small blood vessels of the nose and cheeks break and become larger. These are also called spider veins. They may be subtle and hard to see, depending on skin color.
* **Swollen bumps.** Many people with rosacea develop pimples on the face that look like acne. These bumps sometimes contain pus. They also may appear on the chest and back.
* **Burning sensation.** The skin of the affected area may feel hot and tender.
* **Eye problems.** Many people with rosacea also have dry, irritated, swollen eyes and eyelids. This is known as ocular rosacea. Eye symptoms may show up before, after or at the same time as skin symptoms.
* **Enlarged nose.** Over time, rosacea can thicken the skin on the nose, causing the nose to look bigger. This condition also is called rhinophyma. It occurs more often in men than in women.

## **Diagnosis**

To determine whether you have rosacea, a doctor or other healthcare professional examines your skin and asks about your symptoms. You may have tests to rule out other conditions, such as psoriasis or lupus. Some symptoms of rosacea may be harder to see on brown and Black skin. These include spider veins and flushing. So, it's important to pay attention to other symptoms, such as swelling, bumps, facial stinging and dry-looking skin.

If your symptoms involve your eyes, you may see an eye doctor, also called an ophthalmologist, for other tests.

**Treatment**

If your symptoms don't improve with the self-care tips below, talk with a member of your healthcare team about a prescription gel or cream. This kind of medicine may help ease symptoms. For more serious rosacea, you might need prescription pills. Laser treatment may be used to reduce flushing and enlarged blood vessels in the face.

How long you need treatment depends on the type of rosacea you have and how serious your symptoms are. Even if your skin calms with treatment, the symptoms often return.

### **Medicines**

Several medicines are used to help control rosacea symptoms. The type of medicine you are prescribed depends on your symptoms. For example, some medicines or treatments work better for flushing, and some medicines work better for pimples and bumps. You may need to try one or more medicines to find a treatment that works for you.

Medicines for rosacea include:

* **Gels or other products applied to the skin.** For the flushing of mild to moderate rosacea, you may try a medicated cream or gel that you apply to the affected skin. Examples are brimonidine (Mirvaso) and oxymetazoline (Rhofade), which reduce flushing by constricting blood vessels. You may see results within 12 hours after use. The effect on the blood vessels is temporary. Overuse might lead to worse flushing. So rather than using it every day, you might use it only before important events.

Brimonidine and oxymetazoline often aren't covered by insurance.

Other prescription topical products help control the pimples of mild rosacea. Examples are azelaic acid (Azelex, Finacea), metronidazole (Metrogel, Noritate, others) and ivermectin (Soolantra). With azelaic acid and metronidazole, you may not see results for 2 to 6 weeks. Ivermectin may take even longer to improve skin. But the results tend to last longer than they do for metronidazole. Sometimes, using two or more of these products leads to the best results.

* **Antibiotic medicine taken by mouth.** For more serious rosacea with bumps and pimples, you may be prescribed an oral antibiotic pill such as doxycycline (ORACEA, others).
* **Acne medicine taken by mouth.** For severe rosacea that doesn't respond to other medicine, you may be prescribed isotretinoin (Amnesteem, Claravis, others). It's a powerful oral acne medicine that also helps clear up the bumps of rosacea. This medicine is not to be taken during pregnancy as it can cause birth defects.

### **Laser treatment**

Laser treatment can help improve the look of enlarged blood vessels. It also can help the long-term redness of rosacea. And it often works better than a cream or a pill for this symptom. Because the laser targets visible veining, this method is most effective on skin that isn't tanned, brown or Black.

Talk with a member of your healthcare team about the risks and benefits of laser treatment. Common side effects include redness, bruising and mild swelling for a few days following the treatment. Rare side effects include blistering and scarring. Icing and gentle skin care help while you heal. On brown or Black skin, laser treatment might cause long-term or permanent changes to the color of the treated skin.

The full effect of the treatment might not be seen for weeks. Repeat treatments may be needed to keep the improved look of your skin.

Laser treatment for rosacea is sometimes considered a cosmetic procedure. Such procedures often aren't covered by insurance. However, nowadays some insurances do cover the procedure. Check with your insurance company directly to see if they cover laser treatment for rosacea.

**Lifestyle and home remedies**

These self-care tips may help you calm your skin and prevent flare-ups:

* **Identify and avoid things that make your symptoms worse.** Notice what tends to cause flare-ups for you and avoid those things.
* **Protect your face.** Use a broad-spectrum sunscreen or moisturizer containing sunscreen with an SPF of at least 30, even on cloudy days. Apply sunscreen generously. Reapply every two hours, or more often if you're swimming or sweating.

People with rosacea might benefit from selecting sunscreens that contain titanium dioxide, zinc or both. These are called mineral-based sunscreens or physical sunscreens. Examples include Eucerin Sensitive Mineral Zinc Oxide Protection, La Roche-Posay Tinted Mineral, and others. Or look for products with silicone, such as dimethicone, or cyclomethicone.

Apply sunscreen after you put on any medicine for your face and before applying makeup, if you use it.

Take other steps to protect your face, such as wearing a hat and avoiding midday sun. In cold, windy weather, wear a scarf or ski mask.

* **Treat your skin gently.** Don't rub or touch your face too much. Use a Non soap cleanser two times a day and moisturize. Some face creams may help reduce redness. Products made for the face that contain azelaic acid, dicarboxylic or niacinamide may help with rosacea symptoms. These products are available without a prescription.

Choose fragrance-free products, and avoid those that contain skin irritants such as alcohol, camphor, urea and menthol.

* **Reduce facial symptoms with makeup.** Some makeup products may help reduce the facial flushing common with rosacea. For example, green-tinted makeup can help cover up the red color. Avoid alcohol-based gels and thin lotions.

**Alternative medicine**

Gently massaging your face daily may help ease symptoms of rosacea. Using your fingers, make little circles starting on the center of the face and working to the ears. Do this for a few minutes.

If stress seems to make your symptoms worse, try stress management methods. Examples are deep breathing and meditating.

**Prevention**

Since the cause of rosacea is unknown, you can’t prevent it. However, you can reduce your risk of having a rosacea flare by identifying and avoiding things in your environment that trigger your symptoms.

Always use caution when going outdoors and exposing your skin to the sun’s UV rays. Sunlight can trigger symptoms of rosacea and sun damage can make it difficult for your skin to heal after a flare. Wear sunscreen daily and reapply sunscreen often throughout the day. You can also wear UV-protective clothing and accessories to cover your skin from the sunlight.

**Outlook / Prognosis**

Rosacea is a chronic condition, and symptoms can come and go unexpectedly. If you know certain things in your environment trigger your symptoms, avoid those triggers to prevent a flare.

Rosacea is a harmless condition and only causes skin discomfort and appearance changes. Severe and untreated cases can lead to skin disfiguration that most often affects your nose. Surgery treats severe cases of rosacea, but many people find relief from mild symptoms with prescribed medicines or over-the-counter (OTC) creams, gels or lotions.

#### **Does rosacea ever go away?**

There’s no cure for rosacea, but treatment options are available to reduce symptoms and flares. Talk to your provider about your symptoms and they’ll help you manage your condition and keep symptoms in remission (keep them from returning).

**Living With**

### **Can I cover rosacea with makeup?**

Over-the-counter makeup products can help cover rosacea. This could include:

* Green-tinted base moisturizers. These can minimize redness if you have a pink-to-red tint to your skin. The color green balances the red tone.
* Concealers or foundations that are oil-free and one shade lighter than your natural skin tone.
* Mineral powders to reduce redness using fewer ingredients that could irritate your skin.
* Fragrance-free or sensitive-skin products to avoid skin irritants.
* Topical antibacterial creams to treat skin redness and small acne-like pimples.
* Sunscreen to protect your skin from the sun’s harmful UV rays, which can trigger symptoms of rosacea. Many makeup products are multi-functioning and include sunscreen in addition to other ingredients.

**complications of rosacea**

Complications of rosacea include:

Phymatous rosacea

Inflammatory eye complications, e.g., blepharokeratoconjunctivitis, sclerokeratitis

Physical discomfort, e.g., from ocular symptoms

Negative psychosocial effects such as increased anxiety, depression, low self-esteem, and social isolation

Trigger avoidance leading to lifestyle limitations.

**Epidemiology**

As the diagnosis of rosacea is mainly based on clinical judgment, many patients, especially those with mild disease, may remain undiagnosed. It is estimated that the worldwide incidence of rosacea is higher than 5% of the population. It favors adults between 30 and 50 years of age, affects females more than males, and is more commonly diagnosed in individuals with fair skin, affecting more than 10% of White

Caucasians with fair sun-sensitive skin appear to have the greatest risk for rosacea. It is unknown whether factors such as masking of facial redness by abundant skin pigment, protective effects of melanin against ultraviolet radiation (an exacerbating factor for rosacea), or genetic differences in susceptibility to rosacea contribute to the lower rate of diagnosis in people with darker skin. Estimates of the prevalence of rosacea in fair-skinned populations range from 2 to 22 percent. A recent prospective study from Germany reported an overall rosacea prevalence of 12 percent, with erythematotelangiectatic and papulopustular subtypes making up 9 and 3 percent, respectively. Prevalence rates for ocular involvement in rosacea patients range from less than 10 percent to more than 50 percent. Cutaneous rosacea exhibits a strong female predominance, with the exception of phymatous rosacea, and is usually diagnosed after the age of 30 years.

**Differential diagnosis for rosacea**

Other conditions that could present with similar cutaneous features include:

Acne vulgaris

Carcinoid

Demodicosis (demodex folliculitis)

Dermatomyositis

Drug reaction

Eczema

Idiopathic facial aseptic granuloma

Periorificial dermatitis or periocular dermatitis

Photo-damaged skin

Pyoderma faciale

Seborrhoeic dermatitis

Steroid-induced acne

Steroid-induced rosacea

Systemic lupus erythematosus.

**Acne Vulgaris**  
A common chronic inflammatory skin disorder characterized by the formation of comedones (whiteheads and blackheads), papules, pustules, nodules, and cysts due to obstruction and inflammation of pilosebaceous units (hair follicles and sebaceous glands)

**Carcinoid**  
A slow-growing type of neuroendocrine tumor that can arise in various organs, often secreting hormones causing symptoms like flushing, diarrhea, and wheezing.

**Demodicosis (Demodex Folliculitis)**  
A skin condition caused by overpopulation of Demodex mites in hair follicles, leading to follicular inflammation, redness, itching, and pustules, often on the face.

**Dermatomyositis**  
An inflammatory disease marked by muscle weakness and a characteristic skin rash, including heliotrope rash around the eyes and Gottron’s papules on knuckles.

**Drug Reaction**  
Adverse skin reactions to medications, which can range from mild rashes to severe life-threatening conditions like Stevens-Johnson syndrome.

**Eczema**  
A group of inflammatory skin disorders characterized by itchy, red, dry, and scaly skin; atopic dermatitis is the most common form.

**Idiopathic Facial Aseptic Granuloma**  
A rare, benign, chronic inflammatory facial nodule in children, presenting as painless, red or purple bumps without infection.

**Periorificial Dermatitis or Periocular Dermatitis**  
A facial rash around the mouth, nose, or eyes characterized by small papules and pustules, often linked to topical steroid use or irritants.

**Photo-damaged Skin**  
Skin changes due to chronic sun exposure, including wrinkles, pigmentation changes, rough texture, and increased risk of skin cancers.

**Pyoderma Faciale**  
A severe form of rosacea presenting with sudden onset of painful, red, inflamed nodules and pustules on the face, primarily in adult women.

**Seborrheic Dermatitis**  
A chronic inflammatory skin condition causing flaky, greasy scales and redness, commonly affecting the scalp, face, and eyelids; linked to Malassezia yeast and associated with blepharitis.

**Steroid-induced Acne**  
Acneiform eruptions triggered by systemic or topical corticosteroid use, presenting with monomorphic papules and pustules without comedones.

**Steroid-induced Rosacea**  
Rosacea-like facial redness and inflammation caused by prolonged topical steroid use, characterized by flushing, erythema, and pustules.

**Systemic Lupus Erythematosus (SLE)**  
A systemic autoimmune disease with diverse manifestations including a characteristic malar rash on the face, photosensitivity, joint pain, and organ involvement.

**RECENT GUIDELINE**

| **ROSACEA PRESENTATION** | **MANAGEMENT OPTIONS** | **QUALITY OF EVIDENCE OF MANAGEMENT OPTIONS (A, B, C)** | **EVIDENCE COMMENTS** |
| --- | --- | --- | --- |
| Persistent central facial erythema without papulopustular (PP) lesions | •Topical alpha-agonist (brimonidine, oxymetazoline)  •Intense pulsed light (IPL), potassium titanyl phosphate (KTP) crystal laser, or pulsed-dye laser | B: Systematic review/meta-analysis of lower-quality clinical trials or studies with limitations and inconsistent findings; lower-quality clinical trial | •More data are needed on optimal use of specific device therapies and topical alpha-agonist therapy in combination |
| Diffuse central facial erythema with PP lesions | •Topical metronidazole  •Topical azelaic acid  •Topical ivermectin  •Oral tetracyclines  •Topical alpha-agonists  •Oral isotretinoin | B: Systematic review/meta-analysis of lower-quality clinical trials or studies with limitations and inconsistent findings; lower-quality clinical trial | •Combination of an oral and topical agent that reduce PP lesions and perilesional erythema based on severity; topical alpha-agonist used for persistent background erythema caused by fixed dilated vasculature  •Sub antibiotic dose doxycycline is the preferred initial oral therapy option due to absence of bacterial selection pressure  •Oral azithromycin is an alternative option if an oral tetracycline is not effective or poorly tolerated (caution in some patients due to potential cardiac risks)  •Oral isotretinoin for refractory disease (transition to intermittent therapy after initial control)  •Other alternative topical agents include sulfacetamide-sulfur, calcineurin inhibitors, retinoids, and permethrin (limited data available on these agents)  •While the data on the use of IPL, KTP or pulsed-dye laser are limited for PP lesions, these options are useful to treat erythema |
| Flushing of rosacea (acute-subacute intermittent vasodilation) | •Flushing is better prevented than treated via avoidance of known triggers, such as sun exposure and photoprotection  •Use of low-dose oral drugs with vasoconstrictive properties, including mirtazapine, propranolol, or carvedilol  •The use of intradermal botulinum toxin achieved good results in a small group of patients, but there remain limited data | B: Systematic review/meta-analysis of lower-quality clinical trials or studies with limitations and inconsistent findings; lower-quality clinical trial | •Data are limited on the management of flushing of rosacea  •Limited data exist on topical therapies  •Some botanicals and natural ingredients might improve facial redness and flushing (niacinamide, parthenolide-free extract of feverfew (*Tanacetum parthenium)*, licorice derivatives, chamomile, green tea) based on preliminary small studies  •An anti-inflammatory cleanser night mask combination was found to markedly reduce facial redness (limited data) |
| Ocular rosacea | •Lid hygiene, sunglasses, eye lubrication formulations.  •Cyclosporin ophthalmic emulsion (3-month, randomized, controlled trial [n=37])  •Topical metronidazole or ivermectin (blepharitis; applied to external eyelid skin)  •Oral doxycycline, erythromycin, or azithromycin | B: Systematic review/meta-analysis of lower-quality clinical trials or studies with limitations and inconsistent findings; lower-quality clinical trial | •Data are based on clinical experience, case reports, and small studies  •Topical corticosteroids for short-term therapy but avoid chronic use  •Oral omega-3 fatty acids may reduce inflammation and dry eye symptoms  •Sub antibiotic dose doxycycline suggested for long-term therapy |
| Granulomatous rosacea | •Oral tetracyclines  •Topical pimecrolimus (case reports)  •Oral isotretinoin (0.7mg/kg/day for 6 months)  •Oral dapsone  •Intense pulsed-dye laser (case)  •Photodynamic therapy (case)  •Topical brimonidine | C: Consensus guidelines; usual practice, expert opinion, case series—limited trial data | •No current standard of treatment; limited data based mostly on case reports  •Oral isotretinoin may produce improvement without recurrence |
| Phymatous rosacea | •Surgical therapy for fully developed phymatous changed (carbon dioxide laser, erbium-doped yttrium aluminum garnet (YAG) laser, electrosurgery, dermabrasion) | C: Consensus guidelines; usual practice, expert opinion, case series—limited trial data | •Treatment selection dependent on stage of development (early or fibrotic) and extent of inflammation (active or burnt out)  •Oral isotretinoin might improve early soft phymatous changes due to sebaceous hyperplasia |

REFERENCES  
[Update on the Management of Rosacea from the American Acne & Rosacea Society (AARS) - PMC](https://pmc.ncbi.nlm.nih.gov/articles/PMC7710291/#xd_co_f=ZjY5NDc4Y2ItMjkzYS00OTU1LTk0MGYtODhhN2NkMjkwMzkx~)

[Rosacea - Symptoms and causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/rosacea/symptoms-causes/syc-20353815)

[Rosacea - Diagnosis and treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/rosacea/diagnosis-treatment/drc-20353820)

[Rosacea: Epidemiology, pathogenesis, and treatment - PMC](https://pmc.ncbi.nlm.nih.gov/articles/PMC5821167/)

**Vitiligo or leucoderma**  
**Definition and description**

Vitiligo (vit-ih-LIE-go) is a disease that causes loss of skin color in patches. The discolored areas usually get bigger with time. The condition can affect the skin on any part of the body. It can also affect hair and the inside of the mouth.

Normally, the color of hair and skin is determined by melanin. Vitiligo occurs when cells that produce melanin die or stop functioning. Vitiligo affects people of all skin types, but it may be more noticeable in people with brown or Black skin. The condition is not life-threatening or contagious. It can be stressful or make you feel bad about yourself.

Treatment for vitiligo may restore color to the affected skin. But it doesn't prevent continued loss of skin color or a recurrence.

Vitiligo is an acquired, chronic, depigmenting disorder of the skin, in which pigment-producing cells (melanocytes

) that determine the color of skin, hair, and eyes are progressively lost. It appears as milky-white patches of skin (leukoderma) and can be cosmetically very disabling, particularly in people with dark skin.

It is currently widely accepted that vitiligo is the result of autoimmune destruction of melanocytes.

### **Who does vitiligo affect?**

Vitiligo affects all races and sexes equally. It’s more visible in people with darker skin tones. Although vitiligo can develop in anyone at any age, macules or patches usually become apparent before age 30.

You might be at a higher risk of developing vitiligo if you have certain autoimmune conditions like:

* Addison’s disease.
* Anemia.
* Diabetes (Type 1).
* Lupus.
* Psoriasis.
* Rheumatoid arthritis.
* Thyroid disease.

Vitiligo occurs in over 1% of the population throughout the world.

Vitiligo usually starts with a few small white macules or patches that may gradually spread over your body. Vitiligo typically begins on your hands, forearms, feet and face, but can develop on any part of your body, including your mucous membranes (the moist lining of your mouth, nose, genital and rectal areas), your eyes and inner ears.

Sometimes, larger patches continue to widen and spread, but they usually stay in the same place for years. The location of smaller macules shifts and changes over time, as certain areas of skin lose and regain their pigment.

The amount of affected skin varies for each person diagnosed with vitiligo. Some people experience a few depigmented areas, while others have a widespread loss of skin color.

### **What are the types of vitiligo?**

Types of vitiligo include:

* **Generalized**: This is the most common type of vitiligo that causes macules to appear in various places on your body.
* **Segmental**: This type only affects one side of your body or one area, such as your hands or face.
* **Mucosal**: Mucosal vitiligo affects mucous membranes of your mouth and/or genitals.
* **Focal**: Focal vitiligo is a rare type where the macules develop in a small area and don’t spread in a certain pattern within one to two years.
* **Trichome**: This type causes a bullseye with a white or colorless center, then an area of lighter pigmentation, and an area of your natural skin tone.
* **Universal**: This rare type of vitiligo causes more than 80% of your skin to not have pigment.

**Causes**

Vitiligo occurs when pigment-producing cells (melanocytes) die or stop producing melanin — the pigment that gives your skin, hair and eyes color. The involved patches of skin become lighter or white. It's unclear exactly what causes these pigment cells to fail or die. It may be related to:

* A disorder of the immune system (autoimmune condition)
* Family history (heredity)
* A trigger event, such as stress, severe sunburn or skin trauma, such as contact with a chemical

### **Skin layers and melanin**

Melanin is a natural pigment that gives your skin its color. It's produced in cells called melanocytes.

## **Risk factors**

Anyone can develop vitiligo. But you may be more likely to develop it if you:

* Have a family history of the condition.
* Have frequent direct contact with phenol-containing chemicals, such as those found in some detergents.

## **Symptoms**

## Vitiligo signs include:

* Patchy loss of skin color, which usually first appears on the hands, face, and areas around body openings and the genitals
* Premature whitening or graying of the hair on your scalp, eyelashes, eyebrows or beard
* Loss of color in the tissues that line the inside of the mouth and nose (mucous membranes)

Vitiligo can start at any age but usually appears before age 30.

Depending on the type of vitiligo you have, it may affect:

* **Nearly all skin surfaces.** With this type, called universal vitiligo, the discoloration affects nearly all skin surfaces.
* **Many parts of the body.** With this most common type, called generalized vitiligo, the discolored patches often progress similarly on corresponding body parts (symmetrically).
* **Only one side or part of the body.** This type, called segmental vitiligo, tends to occur at a younger age, progress for a year or two, then stop.
* **One or only a few areas of the body.** This type is called localized (focal) vitiligo.
* **The face and hands.** With this type, called acrofacial vitiligo, the affected skin is on the face and hands, and around body openings, such as the eyes, nose and ears.

It's difficult to predict how this disease will progress. Sometimes the patches stop forming without treatment. In most cases, pigment loss spreads and eventually involves most of the skin. Occasionally, the skin gets its color back.

**Diagnosis**

Your health care provider will ask about your medical history and examine your skin, possibly with a special lamp. The evaluation might also include a skin biopsy and blood tests.

## **Treatment**

The choice of treatment depends on your age, how much skin is involved and where, how quickly the disease is progressing, and how it's affecting your life.

Medications and light-based therapies are available to help restore skin color or even out skin tone, though results vary and are unpredictable. And some treatments have serious side effects. So, your health care provider might suggest that you first try changing the appearance of your skin by applying a self-tanning product or makeup.

If you and your health care provider decide to treat your condition with a drug, surgery or therapy, the process may take many months to judge its effectiveness. And you may have to try more than one approach or a combination of approaches before you find the treatment that works best for you.

Even if treatment is successful for a while, the results may not last or new patches may appear. Your health care provider might recommend a medication applied to the skin as maintenance therapy to help prevent relapse.

### **Medications**

No drug can stop the process of vitiligo — the loss of pigment cells (melanocytes). But some drugs, used alone, in combination or with light therapy, can help restore some color.

* **Drugs that control inflammation.** Applying a corticosteroid cream to affected skin might return color. This is most effective when vitiligo is still in its early stages. This type of cream is effective and easy to use, but you might not see changes in your skin's color for several months. Possible side effects include skin thinning or the appearance of streaks or lines on your skin.

Milder forms of the drug may be prescribed for children and for people who have large areas of discolored skin.

Corticosteroid pills or injections might be an option for people whose condition is progressing rapidly.

* **Medications that affect the immune system.** Calcineurin inhibitor ointments, such as tacrolimus (Protopic) or pimecrolimus (Elidel) might be effective for people with small areas of depigmentation, especially on the face and neck. The U.S. Food and Drug Administration (FDA) has warned about a possible link between these drugs and lymphoma and skin cancer.

### **Therapies**

* **Light therapy.** Phototherapy with narrow band ultraviolet B (UVB) has been shown to stop or slow the progression of active vitiligo. It might be more effective when used with corticosteroids or calcineurin inhibitors. You'll need therapy two to three times a week. It could take 1 to 3 months before you notice any change, and it could take 6 months or longer to get the full effect.

Given the Food and Drug Administration (FDA) warning regarding possible risk of skin cancer with use of calcineurin inhibitors, talk with your health care provider about the risks and benefits of using these drugs with phototherapy.

For people who can't go to a clinic for treatment, smaller portable or handheld devices for narrow band ultraviolet B therapy are available for home use. Talk with your health care provider about this option as well if needed.

Possible side effects of narrow band ultraviolet B therapy include redness, itching and burning. These side effects usually clear up within a few hours after treatment.

* **Combining psoralen and light therapy.** This treatment combines a plant-derived substance called psoralen with light therapy (photochemotherapy) to return color to the light patches. After you take psoralen by mouth or apply it to the affected skin, you're exposed to ultraviolet A (UVA) light. This approach, while effective, is more difficult to administer and has been replaced in many practices by narrow band ultraviolet B (UVB) therapy.
* **Removing the remaining color (depigmentation).** This therapy may be an option if your vitiligo is widespread and other treatments haven't worked. A depigmenting agent is applied to unaffected areas of skin. This gradually lightens the skin so that it blends with the discolored areas. The therapy is done once or twice a day for nine months or longer.

Side effects can include redness, swelling, itching and very dry skin. Depigmentation is permanent.

### **Surgery**

If light therapy and medications haven't worked, some people with stable disease may be candidates for surgery. The following techniques are intended to even out skin tone by restoring color:

* **Skin grafting.** In this procedure, your doctor transfers very small sections of your healthy, pigmented skin to areas that have lost pigment. This procedure is sometimes used if you have small patches of vitiligo.

Possible risks include infection, scarring, a cobblestone appearance, spotty color and failure of the area to recolor.

* **Blister grafting.** In this procedure, your doctor creates blisters on your pigmented skin, usually with suction, and then transplants the tops of the blisters to discolored skin.

Possible risks include scarring, a cobblestone appearance and failure of the area to recolor. And the skin damage caused by suctioning may trigger another patch of vitiligo.

* **Cellular suspension transplant.** In this procedure, your doctor takes some tissue on your pigmented skin, puts the cells into a solution and then transplants them onto the prepared affected area. The results of this repigmentation procedure start showing up within four weeks.

Possible risks include scarring, infection and uneven skin tone.

### **Potential future treatments**

Treatments being studied include:

* **A drug to stimulate color-producing cells (melanocytes).** Called afamelanotide, this potential treatment is implanted under the skin to promote the growth of melanocytes.
* **A drug that helps control melanocytes.** Prostaglandin E2 is being tested as a way to restore skin color in people with vitiligo that isn't widespread or spreading. It's applied to the skin as a gel.

## **Self-care**

If you have vitiligo, the following self-care tactics may help you care for your skin and improve its appearance:

* **Protect your skin from the sun and artificial sources of UV light.** Use a broad-spectrum, water-resistant sunscreen with an SPF of at least 30. Apply sunscreen generously and reapply every two hours — or more often if you're swimming or sweating.

You can also seek shade and wear clothing that shields your skin from the sun. Don't use tanning beds and sunlamps.

Protecting your skin from the sun helps prevent sunburn of the discolored skin. Sunscreen also minimizes tanning, which accentuates the vitiligo patches.

* **Conceal affected skin.** Makeup and self-tanning products can help minimize the differences in skin color. You may need to try several brands of makeup or self-tanners to find one that blends well with your normal skin tone. The coloring of self-tanning products doesn't wash off, but it gradually fades over several days. If you use a self-tanner, select one that contains dihydroxyacetone, as it is approved by the U.S. Food and Drug Administration.
* **Don't get a tattoo.** Damage to your skin, such as that caused by a tattoo, may cause a new patch of vitiligo to appear within two weeks.

## **Alternative medicine**

Limited studies show that the herb Ginkgo biloba may return skin color in people with vitiligo. Other small studies show that alpha-lipoic acid, folic acid, vitamin C and vitamin B-12 plus phototherapy may restore skin color for some people.

As with any nonprescription treatment, check with your health care provider before trying alternative medicine therapies to be sure they won't interfere with other treatments you're using.

**Prevention**

As there could be several causes of vitiligo, there’s no known way to prevent it. You can reduce your risk of developing vitiligo by:

* Practicing safe sun exposure habits.
* Taking care of your skin by using a moisturizer daily.
* Avoiding stress or injury to your body.
* Managing any underlying autoimmune conditions.

## **Outlook / Prognosis**

Vitiligo affects your appearance and can affect how you feel about your skin in social situations. Many people find comfort in speaking with a mental health professional to help them feel more confident and build their self-esteem.

There’s no cure for vitiligo but if you’d like to get treatment, your healthcare provider will help you choose the treatment that’s right for you and your skin.

### **Will my natural skin color return with vitiligo?**

About 10% to 20% of people who have vitiligo fully regain their skin color. This is most common among people who:

* Receive an early diagnosis before age 20.
* Experience the peak of the condition spreading within six months or less.
* Have symptoms mainly in their facial area.

It’s less likely that you’ll regain your pigment if you:

* Develop vitiligo symptoms after age 20.
* Have symptoms on your lips, limbs or hands.

### **How do I hide vitiligo?**

If you’re uncomfortable with how vitiligo looks on your skin, you can hide macules or patches at home by:

* Using sunscreen with an SPF of 30 or higher. The use of sunscreens minimizes tanning and limits the contrast between affected and normal skin.
* Wearing makeup to camouflage depigmented areas.
* Dying your hair with hair dye to help it blend in with unaffected hair on your head.
* Getting micropigmentation, which is a tattoo over your vitiligo spots. It acts as permanent makeup to hide symptoms of the condition.

### **Is vitiligo contagious?**

No. Vitiligo isn’t contagious. It doesn’t spread from person to person through physical contact.

**Living With**

### **When should I see my healthcare provider?**

Contact your healthcare provider if:

* Your skin loses pigmentation or color rapidly.
* Depigmentation spreads to a large area of your body.
* The changes to your skin affect your mental health and well-being.

### **What questions should I ask my doctor?**

* What type of vitiligo do I have?
* Will my skin get its pigment back?
* How do I protect myself from the sun?
* What treatment options are best for my skin?
* Will my future children inherit this condition?

## **Additional Common Questions**

### **What’s the difference between tinea versicolor and vitiligo?**

Tinea versicolor and vitiligo are different conditions that affect the pigment of your skin. Tinea versicolor is a fungal infection that causes your skin to develop white, yellow, red, pink or brown spots. Vitiligo is an autoimmune condition where you lose pigment. It causes your skin to turn lighter than your natural skin tone or white.

### **Is piebaldism the same as vitiligo?**

No. Both conditions cause white or light patches of skin or hair. Piebaldism occurs when a portion of your skin doesn’t have melanocytes, which are cells that produce pigment (melanin). You’re born with piebaldism. Vitiligo occurs when your body has melanocytes, but they’re destroyed. You develop vitiligo during your lifetime.  
  
  
**Possible complications**

Although vitiligo is mainly a cosmetic condition, vitiligo may cause:

* **Sensitive skin**: Macules and patches lack melanocytes, so your skin can be more sensitive to sunlight than the rest of your skin. This can cause your skin to quickly burn instead of tanning.
* **Eye abnormalities**: People with vitiligo may have some abnormalities in their retinas (the inner layer of your eye that contains light-sensitive cells) and some variation of color in their irises (the colored part of your eye). In some cases, there’s inflammation of the retina or iris, but vision usually isn’t affected.
* **Predisposition to autoimmune conditions**: People with vitiligo may be more likely to get other autoimmune conditions that affect how their body’s immune system functions. Common autoimmune conditions include hypothyroidism, diabetes and anemia.
* **Emotional challenges**: People with vitiligo may feel embarrassed about the way their skin looks. Some people diagnosed with vitiligo develop low self-esteem. This could cause anxiety or depression and make someone want to isolate themselves or avoid social situations. If this happens, you should talk to your healthcare provider, a mental health professional.

**Differential Diagnosis**

* **Nevus depigmentosus:** They are circumscribed, segmental depigmented, or hypopigmented areas present at birth.
* **Pityriasis alba:** It is commonly considered a spectrum of atopic dermatitis and usually affects children. It presents as white hypopigmented, scaly macules and patches on the face and other photo-exposed areas.
* **Idiopathic guttate hypomelanosis:** It is characterized by small, asymptomatic pearly white macules on photo-exposed areas. Most of these lesions appear in older age groups.
* **Tinea (pityriasis) Versicolor:** It is a superficial fungal infection that causes loss of pigment. It appears as pale scaly macules present on the back and chest. They give yellow fluorescence under Wood's lamp
* **Halo nevus**: It is a type of melanocytic nevus surrounded by an oval halo of depigmentation
* **Progressive macular hypomelanosis:** It is clinically present as asymptomatic hypopigmented patches on the trunk of young adults
* **Drug-induced leukoderma:** Potent topical or intralesional corticosteroids can induce hypopigmentation at the site of application. Depigmentation is also noticed in patients treated with the tyrosine kinase and epidermal growth factor receptor inhibitor.
* **Hypopigmented mycosis fungoides:** It is a variant of early-stage MF commonly seen in children and dark-skinned people. It appears as widespread hypopigmented patches with atrophy and scaling and atrophy.

**EPIDEMIOLOGY**

Vitiligo is commonly described with an estimated prevalence of 0.5%-1%, and recent epidemiological studies have suggested an increase in prevalence. However, no studies have systematically evaluated the epidemiology of vitiligo. Therefore, we examined the epidemiology of vitiligo. Studies reporting on the prevalence or incidence of vitiligo in the general population were included and each study was categorised in subgroups. Pooled proportions were calculated with the DerSimonian-Laird method for random-effects models. We included 171 studies, comprising 572,334,973 participants. The overall incidence was 1.59 events per 10,000 person-years (95% confidence interval [CI]: 0.70 to 2.83). The overall prevalence was 0.40% (95% CI: 0.37-0.44); there was no difference between females (0.50%, 95% CI: 0.36-0.66) and males (0.49%, 95% CI: 0.35-0.65). Children and adolescents showed a lower prevalence (0.27%, 95% CI: 0.24-0.31) than adults (0.70%, 95% CI: 0.59-0.81).

Questionnaire-based studies showed a higher prevalence (0.73%, 95% CI: 0.52%- 0.98%) than examination-based studies (0.59%, 95% CI: 0.46%-0.73%) and register-based studies (0.13%, 95% CI: 0.10%-0.17%). The prevalence increased in examination-based studies from 0.40% (95% CI: 0.17%-0.73%) to 0.89% (95% CI: 0.68%-1.13%) in studies conducted between 1943-1979 and between 2020-2023. The reported prevalence estimates show the global impact of vitiligo and how subgroup analyses influence the prevalence. The overall prevalence is lower than previously assumed; females and males are equally affected by vitiligo, and it is more common in adults than in children.

**GUIDELINE**

While vitiligo is an incurable condition, it is manageable through various treatment options. These include FDA-approved and off-label medications, light therapy, microsurgery, and adjunctive therapies. The effectiveness of these treatments varies based on factors such as the patient's age, time since disease onset, skin phototype, genetic background, and other considerations.

Leading the drug treatments are advanced JAK inhibitors like Opzelura® from biotech firm Incyte, and Litfulo® from pharmaceutical giant Pfizer, in the last phase of clinical trials. These drugs act as immune system regulators, targeting specific communication pathways to control irregular immune responses.

Opzelura® (1.5% ruxolitinib), notably the first FDA-approved drug for vitiligo, is a topical treatment suitable for individuals aged 12 and above. Recommended usage involves twice-daily application to affected areas, especially on the face, with no fixed limit on duration of use, provided there are visible improvements. Results vary; some may see benefits within 24 weeks, while others might need up to a year. Dermatologists often prioritize its use on the face and sensitive areas like the genitals, areola, or ventral breast. Research suggests that low-level light exposure may enhance the drug's effectiveness, although the drug alone can maintain repigmentation.

The drug development pipeline for vitiligo treatments is promising. Currently, a dozen pharmaceutical and biotech companies are actively investing in vitiligo research and development programs. This ongoing research holds the potential for new and more effective treatments in the future.

**REFERENCE.**

[Vitiligo - Symptoms & causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/vitiligo/symptoms-causes/syc-20355912)

[Vitiligo - Diagnosis & treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/vitiligo/diagnosis-treatment/drc-20355916)

[Vitiligo - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK559149/)

[Vitiligo Research Foundation | Treatment guideline](https://vrfoundation.org/treatment_guidelines/5)

**Hidradenitis suppurativa**

**Definition and description**

Hidradenitis suppurativa (hi-drad-uh-NIE-tis sup-yoo-ruh-TIE-vuh), also known as **acne inversa,** is a condition that causes small, painful lumps to form under the skin. The lumps usually develop in areas where your skin rubs together, such as the armpits, groin, buttocks and breasts. The lumps heal slowly, recur, and can lead to tunnels under the skin and scarring.

Hidradenitis suppurativa tends to start after puberty, usually before age 40. It can persist for many years and worsen over time. It can affect your daily life and emotional well-being. Combined medical and surgical therapy can help manage the disease and prevent complications.

Women are three times more likely to develop hidradenitis suppurativa, though this ratio can differ by location around the world. Also, Black people are more likely to develop this disease than people of other races. This could be attributed to genetic factors.

**Causes**

Hidradenitis suppurativa develops when hair follicles become blocked, but why this blockage occurs isn't known. Experts think it could be connected to hormones, genetic predisposition, cigarette smoking or excess weight.

An infection or being unclean does not cause hidradenitis suppurativa, and it can't be spread to other people.

**Risk factors**

Factors that increase your chance of developing hidradenitis suppurativa include:

* **Age.** The risk of hidradenitis suppurativa is higher for people in their teens and 20s.
* **Sex.** Females are more likely to develop hidradenitis suppurativa than males.
* **Race.** Ethnicity or race might affect risk level. The condition occurs most in Black people, possibly due to genetic factors.
* **Family history.** A tendency to develop hidradenitis suppurativa can be inherited.
* **Certain conditions.** Hidradenitis suppurativa is more common and severe in people who are overweight. It also has an association with severe acne, arthritis, diabetes, metabolic syndrome and inflammatory bowel disease.
* **Smoking.** Smoking tobacco has been linked to hidradenitis suppurativa.

**Symptoms**

Hidradenitis suppurativa can affect one or several areas of the body. Signs and symptoms of the condition include:

* **Blackheads.** Blackheads appear in small, pitted areas of skin, often appearing in pairs.
* **Painful pea-sized lumps.** The condition usually starts with a single, painful lump under the skin that persists for weeks or months. More bumps may form later, usually in areas where you have more sweat and oil glands or where the skin rubs together, such as the armpits, groin, buttocks and breasts.
* **Leaking bumps or sores.** Some bumps or sores get bigger, break open and drain pus with an odor.
* **Tunnels.** Over time, tunnels might form under the skin, connecting the lumps. These wounds heal slowly, if at all, and drain blood and pus.

Some people with this condition experience only mild symptoms. The course of the disease is highly variable. Excess weight and being a smoker are associated with worse symptoms, but people who are thin and don't smoke can experience severe disease.

## **Diagnosis**

Hidradenitis suppurativa can be mistaken for pimples or acne. For many people, it takes years to receive a correct diagnosis.

Your health care provider will base a diagnosis on your signs and symptoms, skin appearance, and medical history. You might be referred to a health care provider who specializes in skin conditions, also known as a dermatologist. Hidradenitis suppurativa can be difficult to diagnose and requires specialized care.

No laboratory test is available to diagnose hidradenitis suppurativa. But if pus or drainage is present, your health care provider might take a sample for lab testing.

**Treatment**

Treatment with medicines, surgery or both can help control symptoms and prevent complications of hidradenitis suppurativa. Talk with your health care provider about the risks and benefits of the treatment options and how to develop an approach that's right for you.

Expect to have regular follow-up visits with your dermatologist. Some people might need the comprehensive care provided by a health care team with members from multiple medical specialties.

### **Medications**

Your health care provider might prescribe one or more of these types of medicines:

* **Antibiotics.** An antibiotic applied to the skin in liquid or gel form may be used to manage mild symptoms. These types of medicines are called topical antibiotics. For more widespread disease, your health care provider might prescribe antibiotic pills, such as doxycycline, or Monodox; clindamycin, or Cleocin; rifampin, or Rimactane; or more than one of these medicines. Rifampin also is known as rifampicin. People with severe disease might need to take antibiotics for months.
* **Steroid injections.** Triamcinolone acetonide, or Kenalog-10, injected into the sores might reduce swelling and inflammation.
* **Hormonal therapy.** Hormone pills, such as estrogen-containing combined oral contraceptives such as estradiol and estradiol/norgestimate might be effective for people with mild hidradenitis suppurativa. Spironolactone often is used to reduce the need for antibiotics, and isotretinoin, which is a medicine that is used mostly to treat acne. Isotretinoin is sometimes used to treat hidradenitis suppurativa.
* **Biologics.** These medicines, usually administered by injection, alter the immune system in a way that disrupts the disease cycle and improves symptoms and signs of disease within weeks. Several of these medicines are approved to treat moderate to severe hidradenitis suppurativa. Two are the tumor necrosis factor inhibitors adalimumab, or Humira, and infliximab, or Remicade. These medicines work by dampening part of the immune system called tumor necrosis factor. Many other biologics are in clinical trials for hidradenitis suppurativa.
* **Retinoids.** Oral retinoids might be an option for some people with acne-like disease. These medicines are not recommended when you're pregnant, breastfeeding or if you intend to become pregnant.
* **Pain medicine.** If pain relievers available without a prescription don't help, your health care provider might prescribe a stronger pain medicine or refer you to a pain clinic.

### **Surgery or other procedures**

Combined medical and surgical approaches help manage hidradenitis suppurativa. Surgery is an important part of disease management when a tunnel, and bump, or abscess, are present. Which surgical approach is right for you depends on the extent and severity of your condition. Talk with your health care provider about the risks and benefits of the options, including:

* **Uncovering the tunnels.** This procedure involves removing tissue to expose the tunnels under the skin. This also is known as unroofing. It's used for people with moderate or severe hidradenitis suppurativa. This solution usually doesn't have to be repeated.
* **Punch debridement.** This procedure, also called limited unroofing, involves removing a single inflamed bump.
* **Laser therapy.** A carbon dioxide laser can be used to make hidradenitis suppurativa sores go away. After this treatment, the sores are unlikely to return. Laser hair removal can help hidradenitis suppurativa in its early stages.
* **Surgical removal.** This approach might be an option for people with persistent or severe symptoms. It involves removing all the affected skin. A skin graft might be needed to close the wound. Even after surgery, sores might still occur in other areas.
* **Incision and drainage.** Surgical drainage is no longer considered an effective option for treating hidradenitis suppurativa. The method might be considered to provide short-term pain relief, but sores usually flare again afterward.

**Lifestyle and home remedies**

Mild hidradenitis suppurativa can sometimes be effectively controlled with self-care measures. Self-care is also an important complement to medical treatment.

These suggestions might relieve discomfort, speed healing or prevent flare-ups:

* **Follow a daily skin care routine.** Gently wash your body with a cleanser that is not soap. It can sometimes be helpful to use an antiseptic wash such as chlorhexidine 4% or benzoyl peroxide wash when showering. Try it once a week at first and then increase use to once daily if your skin tolerates it well. Pat dry. When washing, avoid using washcloths, loofahs or other such items on affected areas, as they can irritate skin. Don't squeeze pimples and sores. And avoid shaving or using hair-removing, or depilatory, creams.
* **Manage your pain.** Gently applying a warm compress can reduce swelling and ease pain. Ask your health care provider about an appropriate pain reliever and how to care for your wounds at home.
* **Try to keep or achieve a healthy weight and stay active.** Not being at a healthy weight can worsen the symptoms of hidradenitis suppurativa. Talk with your health care team to develop a plan. Try to find activities that don't irritate your skin.
* **Consider altering your diet.** Diets that include dairy, red meat and foods with a high glycemic index might worsen hidradenitis suppurativa symptoms. If your diet includes these foods, talk with a dietitian about the benefits of eliminating them.
* **Quit smoking.** If you smoke, try to quit. Stopping smoking can ease the symptoms of hidradenitis suppurativa.

**Prevention**

Some risk factors, like family history, are out of your control. But there are steps you can take to lower the risk of HS flare-ups and complications.

Prevention may include:

* Limit sweating by staying indoors when it’s hot outside.
* Lose weight if you have obesity (a BMI, or body mass index, greater than 30).
* Don’t use scented deodorants or skin products.
* Quit smoking if you use tobacco. (A healthcare provider will have resources to help you.)
* Wear loose-fitting clothing.

## **Outlook / Prognosis**

The outlook is good. There are several treatments currently available that can help people with hidradenitis suppurativa. Experts are also testing new potential treatments.

**Living With**

There’s no cure for hidradenitis suppurativa. It’s an ongoing skin condition with symptoms that may come and go for years. Treatments can manage symptoms and clear up boils. But there’s still a chance they could come back.

### **What’s it like living with hidradenitis suppurativa?**

Recurrent draining abscesses make many people feel self-conscious. The stress of taking care of a chronic, painful condition can also impact mental health. Anxiety or depression are common in people with HS. If you’re struggling with these issues, talk to your healthcare provider.

**What are common HS complications?**

Longstanding untreated HS increases your risk of:

* Anemia.
* Cellulitis.
* Damage to your body’s lymphatic system.
* Fistula, an abnormal connection between your skin and bowel or bladder.
* Scars.
* Sepsis.
* Skin cancer (rare).

### **When to see a doctor**

Early diagnosis of hidradenitis suppurativa is key to effective treatment. See your dermatologist if your condition:

* Is painful.
* Makes it difficult to move.
* Doesn't improve in a few weeks.
* Returns within weeks of treatment.
* Appears in several locations.
* Flares often.

Your dermatologist can create a treatment plan for you.

Hidradenitis suppurativa is not just a boil, and many people with this condition also have related conditions. People with hidradenitis suppurativa benefit from a healthcare team with medical and surgical dermatologists at the core. Other specialists are involved as needed.

## **Differential Diagnoses**

* Blastomycosis
* Cat Scratch Disease (Cat Scratch Fever)
* Dermatologic Aspects of Actinomycosis
* Dermatologic Manifestations of Lymphogranuloma Venereum
* Dermatologic Manifestations of Nocardiosis
* Erysipelas
* Granuloma Inguinale (Donovanosis)
* Syphilis

**Blastomycosis**  
A systemic fungal infection caused by *Blastomyces dermatitidis*, primarily affecting the lungs but often disseminating to the skin and other organs.

**Cat Scratch Disease (Cat Scratch Fever)**  
An infectious disease caused by *Bartonella henselae*, transmitted by scratches or bites from cats. It typically presents with regional lymphadenopathy near the site of inoculation, fever, and sometimes skin papules or pustules at the scratch site.

**Dermatologic Aspects of Actinomycosis**  
A chronic bacterial infection caused by *Actinomyces* species, characterized by the formation of abscesses, draining sinus tracts, and firm nodules, often on the face and neck.

**Dermatologic Manifestations of Lymphogranuloma Venereum**  
A sexually transmitted infection caused by *Chlamydia trachomatis* serovars L1-L3, presenting initially with a painless genital ulcer followed by painful inguinal lymphadenopathy (bubo formation).

**Dermatologic Manifestations of Nocardiosis**  
An infection caused by *Nocardia* species, presenting with cutaneous abscesses, nodules, ulcers, or cellulitis, often following traumatic inoculation. It can disseminate to lungs and brain, especially in immunocompromised patients.

**Erysipelas**  
An acute superficial bacterial skin infection caused mainly by *Streptococcus pyogenes*, characterized by sharply demarcated, raised, red, swollen, and tender plaques, usually on the face or lower limbs, often accompanied by fever and systemic symptoms.

**Granuloma Inguinale (Donovanosis)**  
A chronic bacterial sexually transmitted infection caused by *Klebsiella (Calymmatobacterium) granulomatis*, characterized by painless, beefy-red ulcerative genital lesions that bleed easily and progressively destroy tissue if untreated.

**Syphilis**  
A sexually transmitted infection caused by *Treponema pallidum*, presenting in stages: primary (painless chancre), secondary (widespread rash including palms and soles), latent, and tertiary (gummas, cardiovascular and neurological involvement).

**EPIDEMIOLOGY**

The prevalence of HS in the sample was 2.2% (18/802; 95% CI: 1.4-3.5%) with no gender predominance. The mean age in the HS group was 34 years (IQR 28-42) and the median body mass index (BMI) of the HS patients was 27.0 (IQR 21.4-28.6). There was no significant difference in BMI between the HS and control group. The screening questionnaire had a sensitivity of 1 (18/18), specificity of 0.8 (20/25), positive predictive value of 0.8 (18/23), and a negative predictive value of 1 (20/20). The axilla was the predominant site of affection (66.7%), and all HS patients were classified as mild disease (Hurley score 1).

The prevalence of HS in Lagos, Nigeria, was 2.2% and, in this population, BMI did not appear to be a risk factor. The axilla was the most affected site, and all patients had a mild disease severity

**GUIDELINE**

Recommendations for systemic antibiotics

| Tetracyclines are recommended in mild-to-moderate HS for a 12-week course or as long-term maintenance when appropriate. |
| --- |
| Clindamycin and rifampin in combination is effective as a second-line treatment for mild-to-moderate disease or as a first-line or adjunct treatment in severe disease. |
| Moxifloxacin, metronidazole, and rifampin in combination are recommended as second- or third-line treatment in moderate-to-severe disease. |
| Dapsone may be effective for a minority of patients with Hurley stage I or II disease as long-term maintenance therapy. |
| IV ertapenem is recommended for severe disease as a 1-time rescue therapy or as a bridge to surgery or other long-term maintenance. |
| Determining the duration and frequency of antibiotic use should balance the benefit received by each patient with the risk of antibiotic resistance. Recurrence following cessation is frequent. |

Recommendations for hormonal agents

| Hormonal agents, including estrogen-containing combined oral contraceptives, spironolactone, cyproterone acetate, metformin, and finasteride, should be considered in appropriate female patients, either as monotherapy for mild-to-moderate HS or in combination with other agents for more severe disease. |
| --- |
| Anecdotal data suggest that progestogen-only contraceptives may worsen HS and should potentially be avoided. |
| Small sample sizes, variable outcome measures and methods, and reporting bias are major limitations in all described evidence of hormonal therapies. |

Recommendations for retinoids

| Results from isotretinoin studies have been mixed. Its use should be considered only as a second- or third-line treatment or in patients with severe concomitant acne. |
| --- |
| Acitretin may be superior to isotretinoin for the treatment of HS, but robust comparative studies are lacking. It should be considered a second- or third-line treatment. |
| Alitretinoin is supported by a single study in women. It is available in Canada and many other countries but not in the United States. |

Recommendations for immunosuppressants

| The available limited evidence does not support the use of methotrexate or azathioprine in the treatment of HS. |
| --- |
| Weak evidence supports the use of colchicine in combination with minocycline in refractory mild-to-moderate disease, but not colchicine monotherapy. |
| Cyclosporine can be considered in patients with recalcitrant moderate-to-severe HS who have failed or are not candidates for standard therapy. |
| Short-term pulse steroid therapy can be considered for acute flares or to bridge patients to other treatments. |
| Long-term systemic corticosteroids tapered to the lowest possible dose can be considered in cases of severe HS, as an adjunct therapy in patients with suboptimal response to standard therapy. |

Recommendations for pediatric and pregnant patients

| Perform laboratory evaluation for precocious puberty in pediatric patients with HS who are age 11 or younger when other suspicious physical examination findings are present. |
| --- |
| Avoid tetracyclines in children younger than 9 years and acitretin in female patients during the childbearing years. |
| Avoid retinoids, hormonal agents, most systemic antibiotics, and most immunosuppressive medications in pregnant patients. |
| Use topical treatments, procedures, and safe systemic agents in pregnant patients. |

REFERENCE

https://pmc.ncbi.nlm.nih.gov/articles/PMC9131892/

https://pubmed.ncbi.nlm.nih.gov/37343527/

[Hidradenitis suppurativa - Diagnosis and treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/hidradenitis-suppurativa/diagnosis-treatment/drc-20352311)

[Hidradenitis Suppurativa (Acne Inversa): Symptoms & Treatments](https://my.clevelandclinic.org/health/diseases/17716-hidradenitis-suppurativa)

### **Lichen planus**

Other names include lichen planus subtropicus, lichen planus tropicus, lichenoid melanodermatitis, and summertime actinic lichenoid eruptions.

Lichen planus (like-en play-nes) is a skin condition that causes a rash to develop on one area of your body or several parts of your body at the same time. It can affect your:

* Skin.
* Inside of your mouth (oral mucosa).
* Scalp (skin on your head usually covered with hair).
* Nails.
* Genitals (penis, vagina and vulva).

Lichen planus is a lichenoid dermatoses. Healthcare providers use the term used to characterize skin disorders according to the presence of firm, raised, discolored bumps on your skin.

### **Is lichen planus an autoimmune condition?**

Lichen planus isn’t an autoimmune condition, but it can cause a similar response. It’s an idiopathic (meaning the cause is unknown) condition that causes inflammation in your skin. In lichen planus, your body’s immune system attacks parts of your body instead of protecting your body from foreign invaders like bacteria or viruses.

In a healthy immune system, special cells in it called T cells help protect your body from infection. In people with lichen planus, their immune system’s T cells attack a protein in their skin and mucus membranes.

No one knows why the T cells attack the protein.

### **What is the difference between lichen planus and lichen sclerosus?**

Lichen planus and lichen sclerosus are both idiopathic conditions that affect your skin, especially mucus membranes.

Lichen sclerosus is a long-term skin condition that causes your skin to gradually become thinner, itch, develop sores and eventually scar. It can occur anywhere on your skin, but it mainly affects the skin around your genitals and anus (anogenital region). You're more likely to have it if you've gone through menopause.

The difference between lichen planus and lichen sclerosus is that lichen sclerosus rarely affects the mucous membranes in your mouth.

### **What’s the difference between lichen planus and psoriasis?**

Lichen planus and psoriasis are both skin conditions that can cause discolored rashes. The main difference between lichen planus and psoriasis is that psoriasis is scaly and typically won’t affect the inside of your mouth.

### **Who does lichen planus affect?**

Anyone can get lichen planus. However, you’re more likely to develop it if you’re a female between 30 and 60 years old.

Some studies suggest that females get lichen planus twice as often as males, and they’re more likely to develop it in their 60s. Males are more likely to develop it in their 40s.

If you have lichen planus on your skin, you’re 50% to 75% more likely to have it in your mouth.

### **How common is lichen planus?**

Lichen planus affects approximately 0.22% to 1% of the adult population. Oral lichen planus affects 1% to 4% of the world population.

### **How does lichen planus affect my body?**

Lichen planus commonly affects the skin around your wrists and elbows (flexor surfaces), the back of your hands (dorsal surfaces) and the fronts of your lower legs.

About half of all people who have lichen planus develop oral lichen planus, which affects the skin inside of your mouth and your tongue.

The affected spots (lesions) on your body usually start as tiny, raised dots (papules) that are about the size of the tip of a pin (0.4 millimeters [mm]). They may grow up to the width of a pencil (1 centimeter [cm]).

Oral lichen planus typically starts as tiny white dots on the skin inside of your cheeks or your tongue. In severe cases, the surrounding skin may become inflamed, and sores may develop.

## **Causes**

The cause of lichen planus is likely related to the immune system attacking cells of the skin or mucous membranes. It's not clear why this irregular immune response happens. The condition isn't contagious.

Lichen planus may be activated by:

* Hepatitis C infection.
* Pain relievers and other medicines.
* An allergic reaction to the metal in dental fillings.

**Risk factors**

Anyone can develop lichen planus. It most often affects middle-aged adults. Lichen planus in the mouth is more likely to affect women than men.

**Complications**

Lichen planus can be difficult to treat on the vulva and in the vagina. It can cause scarring and severe pain. Sores on the genitals can make sex painful.

The affected skin and nails might stay slightly darker even after healing.

Oral sores may affect your ability to eat. Oral lichen planus increases the risk of oral cancer. Rarely, lichen planus affects the ear canal. Left untreated, it may lead to hearing loss.

**Symptoms**

Symptoms of lichen planus vary depending on the part of the body affected. Nail disease usually affects several nails. Symptoms include:

* Purple, shiny, flat bumps, often on the inner forearms, wrists or ankles.
* Lines of rash where the skin has been scratched.
* Lacy white patches on the tongue or inside of the cheeks.
* Itchiness.
* Painful sores in the mouth or genitals.
* Rarely, hair loss.
* Nail scarring or loss.
* Dark lines from the tip of the nail to the base.

### **When to see a doctor**

See your healthcare provider if tiny bumps or a rash appears on your skin for no known reason, such as contact with poison ivy. Also see your health care provider if you have any symptoms related to lichen planus of the mouth, genitals, scalp or nails.

It's best to get a prompt and correct diagnosis because a number of skin and mucous membrane conditions can cause sores and pain.

## **Diagnosis**

To find the cause of your illness, your health care provider will likely talk with you about your symptoms and medical history and do a physical exam. You may also need some tests. These might include:

* **Biopsy.** Your health care provider removes a small piece of affected tissue for examination in a laboratory. The tissue is examined to see if it has the cell patterns typical of lichen planus.
* **Blood tests.** You may have your blood drawn to test for health problems related to lichen planus. For example, hepatitis C.

**Treatment**

If you have no pain or discomfort, you may not need any treatment. Lichen planus on the skin often clears up on its own in months to years.

Medicines and other treatments might help relieve itching, ease pain and speed healing. Talk with your health care provider to weigh the pros and cons of treatment options. You may need more than one approach to control your symptoms.

If the disease affects your mucous membranes and nails, it tends to be harder to treat. Even if treatment works, the symptoms may return. You'll likely need to visit your health care provider for follow-up care at least once a year.

### **Corticosteroids**

Often, the first choice for treatment of lichen planus of the skin is a prescription corticosteroid cream or ointment. This may help ease pain, swelling and inflammation.

If a topical corticosteroid doesn't help and your condition is severe or widespread, your health care provider might suggest corticosteroid pills or injections.

Side effects vary, depending on the method of use. Corticosteroids are safe when used as directed.

### **Oral anti-infections drugs**

Other oral medicines used for lichen planus are the antimalarial hydroxychloroquine (Plaquenil) and the antibiotic metronidazole (Flagyl, others).

### **Immune response medicines**

For more-severe symptoms, you may need prescription medicine that changes your body's immune response. The following drugs have been used with some success, but further study is needed:

* cyclosporine (Sandimmune).
* Azathioprine (Azasan).
* methotrexate (Trexall).
* mycophenolate (Cellcept).
* sulfasalazine.
* thalidomide (Thalomid).

### **Antihistamines**

An antihistamine medicine taken by mouth might ease the itchy skin caused by lichen planus.

### **Light therapy**

Light therapy may help clear up lichen planus affecting the skin. This approach is also called phototherapy. One method involves exposing the affected skin to ultraviolet Blight 2 to 3 times a week for several weeks.

One possible side effect is lasting changes in skin color (post inflammatory hyperpigmentation) even after the skin heals.

### **Retinoids**

Your health care provider might prescribe a retinoid medicine taken by mouth or applied to the skin. One example is acitretin.

Retinoids can cause birth defects, so this type of medicine isn't for people who are pregnant or may become pregnant. If you're pregnant or nursing, your health care provider may suggest that you delay treatment or choose a different treatment.

### **Dealing with triggers**

If your health care provider thinks that your lichen planus is related to an infection, allergies, a medicine you take or some other trigger, you might need other treatment or tests to address that. For example, you may need to switch medicine, or your health care provider may suggest additional testing for allergens.

**Lifestyle and home remedies**

Self-care steps can help reduce itching and pain caused by lichen planus. These include:

* Taking a bath in lukewarm water. Sprinkle in an oatmeal-based bath product (Aveeno, others). Rinse well, pat dry and apply a moisturizer.
* Applying a cool, damp cloth.
* Using the type of hydrocortisone cream or ointment that you can get at a store without a prescription. Get a product that has at least 1% hydrocortisone. Do this only if you aren't using a prescription corticosteroid product on your skin.
* Avoiding scratching your skin and injuring your nails.
* Brushing your teeth twice a day and flossing daily, if you have oral lichen planus.

**Alternative medicine**

A few small clinical trials have suggested the benefit of aloe vera gel for treating lichen planus of the vulva and aloe vera mouthwash for disease of the mouth.

Look into alternative medicine approaches that help reduce stress, as stress can worsen the symptoms of lichen planus.

Talk with your health care provider before trying an alternative treatment for lichen planus. Some alternative medicines or supplements have unwanted side effects.

### **Are there any home remedies for symptoms of lichen planus?**

There are several over-the-counter products or home remedies that can help stop your rash from itching.

While home remedies are safe for most people, it’s a good idea to check with your healthcare provider before trying some of the following options. You may be at risk of developing an allergic reaction.

* **Aloe vera**. Aloe vera is a wound care gel that can moisturize, heal and treat sores. Some research suggests that it can relieve lichen planus symptoms in your mouth or on your vulva.
* **Antihistamines**. Antihistamines are a class of drugs commonly used to treat allergy symptoms, including itchy skin.
* **Hydrocortisone creams or ointments**. Over-the-counter (OTC) hydrocortisone is a corticosteroid combined with an anesthetic pain reliever.
* **Oatmeal**: The best way to use oatmeal on your lichen planus skin rash is to grind it into a fine powder (colloidal oatmeal) in a blender or food processor. Mix the oatmeal powder with warm water until it becomes a thick, sticky paste. Apply enough of the oatmeal paste to cover your rash completely. After at least 10 minutes, wipe off the paste with a clean towel.

Stress can also make skin disease worse. There are mental/emotional signs of stress and physical signs of stress. Stress management techniques can help you prevent or ease your stress-induced lichen planus symptoms.

#### **What signs of stress can make my symptoms worse?**

Some psychological signs of stress that can make your lichen planus symptoms worse include:

* Depression.
* Difficulty relaxing.
* Use of alcohol, tobacco or drugs to relax.
* A negative opinion of yourself (low self-esteem).
* Anxiety (constant worry).
* Feeling overwhelmed.
* Difficulty concentrating.
* Irritability, mood swings or a short temper.

Some physical signs of stress that can make your lichen planus symptoms worse include:

* Nausea and dizziness.
* Not wanting to have sex.
* Sleeping too much (hypersomnia).
* Sleeping too little (insomnia).
* Diarrhea.
* Constipation.
* Muscle tension.
* Aches and pains.

### **What foods and drinks should I avoid if I have lichen planus?**

If you have oral lichen planus, it’s a good idea to avoid spicy or acidic foods or drinks that may cause further irritation, including:

* Hot peppers.
* Salsas.
* Citrus fruits.
* Tomatoes.
* Alcohol.

In addition to avoiding spicy and acidic foods and drinks, you should avoid smoking.

**Prevention**

There isn’t any way to prevent lichen planus.

**Outlook / Prognosis**

Lichen planus can be managed without treatment. But most cases will require treatment. If you have lichen planus on your skin, it may take a few months or a few years to go away. If you have oral lichen planus, it may take up to five years to go away. Therapy and home remedies can provide relief if you experience any symptoms.

If you have lichen planus on your penis, vagina or vulva, you may experience discomfort or pain during sex. You can’t give your partner lichen planus through unprotected sex.

**Living With**

**When should I see my healthcare provider?**

Call your healthcare provider if:

* You develop new symptoms.
* Your symptoms don’t improve after treatment.
* Your rash lasts longer than expected.
* Your rash looks infected (red, purple, gray or white skin; irritation and swelling).

### **What questions should I ask my healthcare provider?**

* How can you tell that I have lichen planus?
* If I don’t have lichen planus, what other skin condition might I have?
* How long will it take my body to recover?
* What medications do you recommend?
* Do the medications have any side effects?
* What at-home treatments do you recommend?
* Is there a prescription cream or ointment that you can prescribe?
* Should I see a dermatologist or another specialist?

## **Epidemiology**

## The prevalence of cutaneous lichen planus is approximately 0.2% to 1% among adults worldwide. Oral lichen planus is more common and reported in 1% to 4% of the population. Overall, women are more frequently affected compared to men at a ratio of 1.5:1, and most cases develop between the ages of 30 and 60. The condition is rare in children, representing less than 5% of all affected patients. Although lichen planus is not generally considered to have an ethnic predilection, recent studies suggest a higher incidence of the disease in African Americans and individuals of Indian and Arabian descent. There appears to be a familial component, as up to 10% of first-degree relatives of patients may also develop the disease.

## **Differential Diagnosis**

## There are many disorders to be considered in the differential diagnosis of lichen planus (Table [**1**](https://onlinelibrary.wiley.com/doi/full/10.1111/jdv.16464?msockid=2cdb3456348e693e1f0921f335a968dc#jdv16464-tbl-0001)):

## Lichen nitidus, lichen sclerosus, lichen spinulosus, graft-versus-host disease, lichen striatus, linear epidermal naevus, naevus unius lateralis

## Eczema, lichen simplex chronicus, prurigo nodularis

## Pityriasis rosea, guttate psoriasis, psoriasis vulgaris, eczematid-like purpura

## Drug eruption, syphilis, tinea corporis, papular acrodermatitis of childhood

## Granuloma annulare, lichen amyloidosus, pityriasis lichenoides, Kaposi sarcoma

## **Table 1.** Differential diagnosis of site-specific LP

| Nail | Psoriasis, onychomycosis, alopecia areata, atopic dermatitis |
| --- | --- |
| Genital | Lichen sclerosus, mucous membrane pemphigoid, vulvar intraepithelial neoplasia, graft-vs-host disease, psoriasis, seborrhoeic dermatitis, intertrigo |
| Palms and soles | Secondary syphilis, psoriasis vulgaris, warts, calluses, porokeratosis, hyperkeratotic eczema, pityriasis rubra pilaris, tinea, drug reaction, gloves-and-socks disease |
| Lichen planopilaris | Cicatricial alopecia, lupus erythematosus, inflammatory folliculitis, alopecia areata, mucous membrane pemphigoid, frontal fibrosing alopecia |
| Mucosal | Paraneoplastic pemphigus, candidiasis, lupus erythematosus, secondary syphilis, leucokeratosis, traumatic patches, cicatricial pemphigoid |

The following interventions can be considered for the treatment of cutaneous lichen planus

| Topical calcipotriol ointment |
| --- |
| Metronidazole (250 mg every 8 h for 12 weeks) |
| Trimethoprim–sulphomethoxazole |
| Hydroxychloroquine sulphate (200–400 mg/day) |
| Itraconazole, terbinafine, griseofulvin (why antifungal therapy is sometimes effective in LP remains to be elucidated) |
| Tetracycline, doxycycline |
| Mycophenolate mofetil (0.5 g twice daily for four weeks, and then 1 g twice daily for at least 20 weeks) |
| Azathioprine (50 mg twice daily orally or 1–2 mg/kg/day, for a period varying from 3 to 7 months) |
| Methotrexate (15–20 mg/week for 4–15 weeks) |
| Cyclophosphamide (50–100 mg/day for 3–6 months) |
| Thalidomide |
| Adalimumab |
| Interferon a2b. Interesting approach especially if lichen planus is associated with hepatitis C. |
| Alitretinoin |
| Low molecular weight heparin (enoxaparin 3 mg/week) |
| Photodynamic therapy |
| Extracorporeal photochemotherapy |
| Nd-YAG laser, low-dose 308 nm excimer laser[**81**](https://onlinelibrary.wiley.com/doi/full/10.1111/jdv.16464?msockid=2cdb3456348e693e1f0921f335a968dc#jdv16464-bib-0081) |
| Apremilast |
| Ustekinumab |

## REFERENCES

## <https://www.ncbi.nlm.nih.gov/books/NBK526126/>

## <https://my.clevelandclinic.org/health/diseases/17723-lichen-planus>

## <https://www.mayoclinic.org/diseases-conditions/lichen-planus/diagnosis-treatment/drc-20351383>

## <https://www.mayoclinic.org/diseases-conditions/lichen-planus/symptoms-causes/syc-20351378>

**Cellulitis**   
**Definition and description**

Cellulitis (sel-u-LIE-tis) is a spreading skin infection, most commonly of the lower leg. It's caused by bacteria entering through a break in the skin.

The affected skin is swollen, painful and warm to the touch. The infection can cause a fever and become very serious, involving deeper tissues.

The condition often clears up with antibiotic medicine.

**Causes**

Cellulitis happens when bacteria enter the body through a crack or break in the skin. Bacteria are most likely to enter broken, dry, flaky or swollen skin. Examples of entry points for bacteria are a recent surgical site, a cut, a puncture wound, a sore, and skin affected by athlete's foot or dermatitis. Cellulitis usually isn't spread from person to person.

The most common bacteria with this condition are streptococcus and staphylococcus. The incidence of a more serious staphylococcus infection called methicillin-resistant Staphylococcus aureus (MRSA) is increasing.

## **Risk factors**

Several factors put you at increased risk of cellulitis:

* **Injury.** Any cut, fracture, burn or scrape gives bacteria an entry point.
* **Weakened immune system.** Conditions that weaken the immune system increase the risk of infection. Examples are diabetes, leukemia and HIV/AIDS. Certain medicines also can weaken the immune system.
* **Skin conditions.** Conditions such as atopic dermatitis — also called eczema — athlete's foot and shingles can cause breaks in the skin and give bacteria an entry point.
* **Long-term swelling of the arms or legs.** This condition is called lymphedema. It sometimes happens after surgery.
* **History of cellulitis.** Having had cellulitis before increases the risk of getting it again.
* **Being overweight.** Excess weight increases the risk of developing cellulitis.

**Symptoms**

Cellulitis is a common condition that can occur anywhere on the body, but it often involves the lower leg and usually just one side of the body.

Cellulitis symptoms include:

* Swelling.
* Warmth.
* Pain.
* Fever.
* Chills.
* Spots on the skin.
* Blisters.
* Skin dimpling.

**Diagnosis**

Your healthcare professional will likely be able to diagnose cellulitis by looking at your skin. You might need to have blood tests or other tests to help rule out other conditions.

## **Treatment**

Cellulitis treatment usually includes a prescription antibiotic medicine taken by mouth. You take it for as long as your healthcare professional directs, usually 5 to 10 days, even after you feel better. Symptoms typically disappear a few days after you start treatment.

You may need to be hospitalized and receive medicine through your veins if:

* Symptoms don't respond to the medicine taken by mouth.
* Symptoms are extensive.
* You have a high fever.

**Home care**

You can relieve your cellulitis symptoms with some self-care practices at home alongside your medication. These can include:

## Elevating the affected part of your body to reduce swelling

## Regularly moving the joint near the affected area, such as your ankle, to prevent stiffness

## Drinking plenty of fluids

## Avoiding compression stockings

## The best way to prevent cellulitis is through good hygiene and wound care practices. These include:

## Keeping skin clean

## Moisturizing skin to prevent cracks

## Wearing proper footwear

## Cleaning wounds and cuts

## Wearing gloves while working outside

#### **Alternative therapies**

## You should not treat cellulitis with alternative therapies alone. Cellulitis can be very serious and potentially life-threatening, so you will need antibiotics.

## Your doctor may recommend alternative therapies alongside antibiotics to strengthen a weak immune system, or if you have antibiotic-resistant bacteria. These may include herbs or supplements such as:

## Vitamin C

## Vitamin E

## Zinc

## Probiotics

## Thyme oil

## Yarrow

## Calendula flower

## Tea tree oil

## You should not only use herbal supplements or plant oils to treat cellulitis. Although they can help kill bacteria on the skin and be useful if your cellulitis is resistant to treatment, they may also interact negatively with your medications. You should speak to your doctor about using alternative therapies.

## **Self-care**

Try these steps to help ease any pain and swelling:

* Place a cool, damp cloth on the affected area as often as needed for your comfort.
* Ask your healthcare professional to suggest a nonprescription pain medicine.
* Elevate the affected part of the body.
* Ask your healthcare professional whether it might help to wear compression wraps or stockings.

**Prevention**

If you tend to have repeated episodes of cellulitis, your healthcare professional may recommend taking antibiotic medicine to prevent it from coming back.

To help prevent cellulitis and other infections, take these precautions when you have a skin wound:

* Wash the wound daily with soap and water. Do this gently as part of your regular bathing.
* Ask your healthcare professional whether it would help to apply cream or ointment. For most minor wounds, the products you can buy without a prescription provide good protection. One example is petroleum jelly (Vaseline).
* Cover the wound with a bandage. Change bandages at least daily.
* Watch for signs of infection. Irritation, pain and pus all signal possible infection and the need for medical care.

People with diabetes or poor circulation need to take extra care to prevent skin injury. Good skin care includes the following:

* Inspect your feet daily. Check your feet for signs of injury so that you can catch infections early.
* Moisturize your skin at least once a day. This helps prevent the skin from cracking and peeling. Don't apply moisturizer to open sores.
* Trim your fingernails and toenails with care. Try not to cut the surrounding skin.
* Protect your hands and feet. Wear footwear and gloves suitable to your activities.
* Treat athlete's foot and toenail fungal infections as soon as they occur.

**Outlook / Prognosis**

### **What can I expect if I have cellulitis?**

With early diagnosis and treatment, the outlook for people with cellulitis is good. Most people feel better after seven to 10 days.

It’s very important to take cellulitis seriously and get treatment right away. Cellulitis can quickly progress and lead to more severe conditions. The bacteria could spread to your bloodstream (bacteremia) or heart (endocarditis), which may be fatal.

**Living With**

### **How do I take care of myself?**

* **Follow your healthcare provider’s instructions**. It’s important to finish your full course of antibiotics, even if you start to feel better. If you don’t finish your full course of medicine, your cellulitis may come back and be more challenging to treat.
* **Refrain from touching the affected area**. If you must touch the affected area, wash your hands before and after touching it. Don’t squeeze or puncture the area. Squeezing and puncturing won’t drain any swelling or provide relief.
* **Rest**. It’s a good idea to avoid any activities that may aggravate your affected areas, including walking, running or other exercises.

### **What questions should I ask my healthcare provider?**

* How do you know that I have cellulitis?
* If I don’t have cellulitis, what other condition might I have?
* How did I get cellulitis?
* What bacteria caused my cellulitis?
* For how long and at what times of the day should I take my medication?
* How should I store my medication?
* When will I start to feel better?
* Do I need to schedule a follow-up visit?
* What activities should I avoid?
* Which OTC pain relievers do you recommend?

## **Additional Common Questions**

### **Does cellulitis itch?**

No, cellulitis doesn’t itch. However, your affected area may itch once your skin starts to heal.

**Why do I keep getting cellulitis?**

Many people who get cellulitis again usually have skin conditions that don’t go away without treatment, such as athlete’s foot or impetigo. Poorly controlled diabetes may also contribute to repeat instances of cellulitis.

Approximately 33% of all people who have cellulitis get it again.

**Complications**

Untreated cellulitis might lead to serious conditions such as bacteremia, endocarditis, osteomyelitis, toxic shock syndrome or sepsis. Rarely, the infection can spread to the deep layer of tissue called the fascial lining. Necrotizing fasciitis is an example of a deep-layer infection. It's an extreme emergency.

Repeated episodes of cellulitis may cause the lymph nodes to enlarge. This can cause long-term swelling of the affected limb.

**When to see a doctor**

It's important to find and treat cellulitis early because the condition can spread rapidly throughout the body.

**Seek emergency care if:**

* You have a swollen rash or a rash that's changing rapidly.
* You have a fever.

See a healthcare professional within 24 hours if you have a swollen rash or a rash that's growing but you don't have a fever.

## **Differential Diagnoses**

* Burn Wound Infections
* Dermatologic Manifestations of Nocardiosis
* Emergent Treatment of Gas Gangrene
* [Erysipeloid](https://emedicine.medscape.com/article/1054170-overview)
* Erythema Multiforme
* Impetigo
* Insect Bites
* Leukemia Cutis
* Lymphoma, Cutaneous T-Cell
* Mycosis Fungoides
* Myiasis
* Osteomyelitis
* Pyoderma Gangrenosum
* Stevens-Johnson Syndrome
* Wells Syndrome (Eosinophilic Cellulitis)

**Burn Wound Infections**  
Infections occurring in burn wounds are common due to loss of the skin barrier and moist, nutrient-rich environment favoring microbial growth. Wound care involves gentle cleaning, antibiotic ointments, and dressings. **Dermatologic Manifestations of Nocardiosis**  
Cutaneous nocardiosis presents with abscesses, nodules, ulcers, or cellulitis, often after traumatic inoculation.

**Gangrene**  
Gas gangrene is a rapidly progressive, life-threatening infection caused by *Clostridium* species producing gas in tissues.

**Erysipeloid**  
A localized skin infection caused by *Erysipelothrix rhusiopathiae*, typically after handling fish or meat.

**Erythema Multiforme**  
An acute, immune-mediated condition characterized by target-shaped lesions on the skin, often triggered by infections (especially herpes simplex virus) or drugs.

**Impetigo**  
A contagious superficial bacterial skin infection mainly caused by *Staphylococcus aureus* or *Streptococcus pyogenes*, presenting with honey-colored crusted erosions, primarily on the face and extremities.

**Insect Bites**  
Skin reactions to insect saliva or venom causing localized itching, redness, swelling, and sometimes blistering or secondary infection.

**Leukemia Cutis**  
Cutaneous infiltration by leukemic cells, resulting in various skin lesions such as papules, nodules, or plaques.

**Lymphoma, Cutaneous T-Cell**  
A group of non-Hodgkin lymphomas presenting primarily in the skin with patches, plaques, or tumors.

**Mycosis Fungoides**  
The most common cutaneous T-cell lymphoma presents with slowly progressive patches, plaques, and tumors on the skin, often mimicking eczema or psoriasis.

**Myiasis**  
Infestation of skin by fly larvae (maggots), causing painful nodules or ulcers. Treatment involves mechanical removal of larvae and wound care.

**Osteomyelitis**  
Infection of bone, which may arise from contiguous spread of skin infections or hematogenous seeding.

**Pyoderma Gangrenosum**  
A rare, inflammatory ulcerative skin condition characterized by painful, rapidly enlarging ulcers with undermined borders, often associated with systemic diseases like inflammatory bowel disease.

**Stevens-Johnson Syndrome (SJS)**  
A severe mucocutaneous reaction usually triggered by drugs or infections, characterized by widespread skin necrosis, blistering, and mucous membrane involvement.

**Wells Syndrome (Eosinophilic Cellulitis)**  
A rare inflammatory skin disorder presenting with recurrent, itchy, red, and edematous plaques resembling cellulitis but without infection.

**statistics or epidemiology data**

Of the 562 cases of acute erysipelas of the leg recruited in the eight countries, 339 were women, with a mean age of 43.7±16.9 years (15 and 88 years); 104 patients consulted 3 days after the onset of erysipelas and 93 patients consult after 10 days. A point of entry was found in 485 patients. A neglected wound was found in 324 (66. 80%) patients (traumatic wound, grazed dermatitis, vascular ulcer) and intertrigo of intertoe found in 161 (33.19%) patients. The use of bleaching products was detected in 97 (17.3%) patients, lymphedema in 130 (23.1%) patients, and varicose veins of the lower limb in 19 (3.4%) patients. Concerning general factors involved with the onset of acute erysipelas of the leg, obesity was found in 230 (40. 9%) patients, diabetes in 27 (4. 8%) patients and HIV infection in 16 (2.8%) patients. The use of non-steroid anti-inflammatory drugs (NSAID) and cataplasm before consultation were detected respectively in 207 and 104 patients (Table 1). Infection of one leg was found in 535 patients, and especially the right leg in 335 patients; both right and left legs were infected in 27 cases. A lymph node was present in 336 patients, and fever (> 37. 8°C) in 424 patients. Of the 562 recruited patients with erysipelas of the leg, 95 were associated with bullous lesions, and 167 were associated with purpuric lesions. Complications were observed, mainly necrotizing fasciitis in 31 patients (6.1%) and abscesses in 63 patients (11.2%). They were due mainly to the delay of antibiotics treatment, and the use of non steroid anti-inflammatory drugs and cataplasms

**GUIDELINE**

MANAGEMENT AT HOME

It is essential that the patient’s response to treatment is monitored, and the patient should seek further medical attention by the GP, at a walk-in Centre or out of hours service if not responding within 48 hours. To establish a baseline to monitor progress, record: • extent and severity of rash – if possible, mark and date the edge of the erythema (may be difficult in lymphoedema as the rash is often blotchy)

• level of systemic upset

• CRP/ESR/white cell count – these may be helpful in diagnosis and monitoring of treatment

• microbiology of any cuts or breaks in the skin – this should be considered before antibiotics are started Oral flucloxacillin 500mg – 1g 6-hourly is recommended as the treatment of choice. Although the likely causative organisms of cellulitis in lymphoedema are beta hemolytic streptococci, microbiologists suggest the use of single agent flucloxacillin for all cellulitis, as this covers both streptococcal and staphylococcal infections.

However, from clinical experience, amoxicillin (500mg 8-hourly) can be an effective alternative, e.g. in those who develop side effects with flucloxacillin.

NB Unusual circumstances, e.g. animal bite or lick preceding an attack should be discussed with a local microbiologist.

Patients who are allergic to penicillin should be prescribed clarithromycin 500 mg 12-hours.

(NB Erythromycin (500mg 6-hourly) is preferred if a macrolide is needed in pregnancy, for example, if there is true penicillin allergy and the benefits of antibiotic treatment outweigh the harms. For those allergic to penicillin and unable to take macrolides e.g. because they are taking statins, doxycycline 100mg 12-hourly is recommended. If there is no response or a poor response (unresolving systemic symptoms or worsening inflammation) to oral flucloxacillin (or amoxicillin / clarithromycin) after 48 hours, then clindamycin 300mg 6-hourly should be substituted as second line oral treatment. If signs or symptoms deteriorate despite oral flucloxacillin (at any time) consider hospital admission/ IV antibiotics.

Ano-genital cellulitis: For those with cellulitis associated with lymphoedema of the Ano-genital region, flucloxacillin or amoxicillin should be used as first line treatment as the causative organism may be streptococcal. If penicillin is allergic, clarithromycin should be used. If not responding to this, then the causative organism may not be streptococcal and co-amoxiclav 625mg 8hrly is recommended.

For those allergic to penicillin, co-trimoxazole 960mg 12 hourly and metronidazole 400mg 8 hourly in combination should be used. If these are unsuccessful, advice from microbiology/local lymphoedema service should be sought.

Clostridium difficile infection is a rare but serious complication of treatment with a variety of antibiotics. If this occurs while on antibiotic treatment, then those antibiotics should be stopped immediately, and alternatives considered. Antibiotics should be given for 14 days.

Experience in lymphoedema clinics suggests a significant rate of early recurrence of cellulitis with shorter courses, implying incomplete resolution of the infection.

Local community / hospital or NICE guidance may recommend 5 – 7 days of treatment but these may not be specifically aimed at treating cellulitis in lymphoedema. If recurrence/deterioration occurs soon after completion of a 14-day course, advice should be sought from local microbiology/ specialist lymphoedema service. Longer courses are occasionally needed.

Skin changes e.g. discoloration/ staining may persist for months or longer following severe cellulitis and do not require ongoing antibiotics. Patients report that rest and elevation are important to help resolve the symptoms of cellulitis. If wearing the usual compression garment causes pain, then it should be removed but replaced as soon as the affected area is comfortable enough to tolerate it. This should reduce the risk of worsening of the swelling if the garment is left off for a prolonged period e.g. one week. The fit of the compression garment may need to be checked as the area may become more swollen after an episode of cellulitis.

The recommended analgesia is paracetamol. Ibuprofen is an alternative (NB It has been suggested previously that non-steroidal anti-inflammatory drugs (NSAIDs) taken at the time of cellulitis may increase the risk of necrotizing fasciitis, but a causative link has not been proven. One small RCT (n=48) has demonstrated no benefit of the addition of ibuprofen to IV antibiotics in accelerating the resolution of cellulitis, but no patients developed necrotizing fasciitis.

When the patient is feeling better, a return to normal levels of activity is encouraged.

PATIENTS ALLERGIC TO PENICILLIN

Because penicillin antibiotics are valuable in the treatment of acute cellulitis, and phenoxymethyl penicillin is known to be effective and safe in prophylaxis against recurrent cellulitis, it is important to check the nature of any “penicillin allergy” to confirm it is a true allergy e.g. anaphylaxis/ widespread rash. If penicillin allergy testing and desensitisation is available locally then this should be considered. Patients who have experienced an anaphylactic reaction to penicillin should not be given antibiotics from the cephalosporin family e.g. cefuroxime, cefotaxime, ceftazidime and cefalexin.

MANAGEMENT IN HOSPITAL Choice of antibiotics in hospital is usually made according to local guidelines. Hospital guidelines commonly recommend single agent IV flucloxacillin 2g 6hly, as this is felt to cover both Staph. and Strep. infections.

Local hospital guidelines will also recommend alternative IV antibiotics for patients allergic to penicillin. It is recommended that patients who have had an attack of cellulitis should carry a two-week supply of antibiotics with them particularly when away from home for any length of time, e.g. on holiday.

Flucloxacillin 500mg – 1g 6-hourly is recommended or, for those allergic to penicillin, clarithromycin 500mg 12-hourly or doxycycline 100mg 12-hourly if taking statins.

Antibiotics should be started immediately when familiar symptoms of cellulitis develop but a medical opinion should be sought as soon as possible, to confirm the diagnosis and response to treatment.

PREVENTING OR REDUCING THE FREQUENCY OF EPISODES OF CELLULITIS

This is an uncommon occurrence but, in this group, it is suggested that a therapeutic course of antibiotics is considered for the duration of the intensive treatment. Other risk factors for recurrent cellulitis including cracked and / or macerated interdigital skin, dermatitis, open wounds including leg ulcers, and weeping lymphangiectasia (leaking lymph blisters on the skin surface) should be treated. Skin care including the use of emollients as part of routine maintenance DLT is recommended to optimize the skin’s natural barrier function.

Obesity is known to reduce lymph drainage. We, therefore, recommend weight management in addition to the treatment of oedema in those who are obese.

PROPHYLACTIC ANTIBIOTICS

In addition, antibiotic prophylaxis should be considered in patients who have had two or more attacks of cellulitis per year. The following should be taken into account in this decision:

• Were the episodes all bacterial cellulitis?

• Could they have been due to conditions such as acute venous hypertension/ lipodermatosclerosis, which are not bacterial and should be managed with compression etc.

• Were the episodes bacterial cellulitis which were incompletely treated e.g. by multiple short (5-7 days) courses of antibiotics? In this situation the symptoms of cellulitis may resolve in a few days but recur after 2-3 weeks. This may reflect an incompletely treated single episode of cellulitis which should be treated with a longer course of antibiotics (at least 2 weeks) and counted as one episode

• Was there a clear, easily reversible cause e.g. athlete's foot/other skin problem? If so, treating this may reduce the risk of further cellulitis and remove the need for antibiotic prophylaxis

For those allergic to penicillin, clarithromycin 250mg daily is recommended

For those with penicillin allergy and taking statins, doxycycline 100mg daily is recommended. It is recommended that patients requiring antibiotic prophylaxis for Ano-genital cellulitis should receive phenoxymethylpenicillin (or alternative as above if penicillin allergic) but if this is not effective, trimethoprim 100mg daily taken at night should be used instead. Following one year of successful prophylaxis, discontinuation should be considered, particularly if the risk factors have been successfully addressed. However, if there are ongoing significant risk factors, continuing prophylaxis for a further year should be considered. If there have been no further episodes of cellulitis during this period, antibiotic prophylaxis should be stopped. Prophylaxis may need to be life-long if relapse occurs after prophylactic antibiotics have been discontinued and there are persistent risk factors.

However, ongoing regular review (at least annually, ideally by local specialist lymphoedema services) is still recommended for those on long term prophylaxis. Discontinuation again should be considered if risk factors have improved at any stage.

It may not be possible to fully prevent further episodes of cellulitis even with prophylactic antibiotics. However, there Summary Type of operation Minor skin surgery Previous history No cellulitis One attack of cellulitis may be a reduction in the frequency of cellulitis and/or the severity of episodes. If the response to first line prophylactic antibiotics is inadequate, then alternative strategies including trials of other prophylactic antibiotics e.g. cefalexin 125mg daily or clindamycin 150mg daily may need to be considered. In these circumstances, review by local specialist lymphoedema services and advice from microbiologists is recommended.

There is a need to balance the use of certain antibiotics (e.g. clindamycin, cefalexin) as prophylaxis against the risks of predisposing to Clostridium difficile infections and promoting antibiotic resistance. If at any stage with prophylactic antibiotics Clostridium difficile occurs, then those antibiotics should be stopped immediately.

It is usual practice to discontinue antibiotic prophylaxis while antibiotics are taken to treat acute cellulitis. Patients undergoing surgical procedures such as knee replacement or carpal tunnel surgery in the lymphoedematous region should receive a therapeutic course of antibiotics commenced before surgery (oral or IV as appropriate) or as indicated by the procedure. This would also include surgery to treat lymphoedema, such as lymphaticovenular anastomosis or lymphoedema liposuction. The antibiotics should begin just before surgery and are usually continued for five to seven days after surgery. The risk of cellulitis after minor skin surgery e.g. mole removal is believed to be small. For minor skin procedures in people who have previously had cellulitis a single prophylactic dose of antibiotics may be considered by the operating surgeon.

REFERENCE  
[Cellulitis - Symptoms & causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/cellulitis/symptoms-causes/syc-20370762)

[2023 BLS Management of cellulitis doc-66331665737684 (2).pdf](https://lnni.org/sites/default/files/documents/2023%20BLS%20Management%20of%20cellulitis%20doc-66331665737684%20%282%29.pdf)

[Cellulitis - Diagnosis & treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/cellulitis/diagnosis-treatment/drc-20370766)

[Cellulitis: Symptoms, Causes, Treatment & Recovery](https://my.clevelandclinic.org/health/diseases/15071-cellulitis)

[Cellulitis Differential Diagnoses](https://emedicine.medscape.com/article/214222-differential?form=fpf)

**Melanoma**

Alternative Names Skin cancer - melanoma; Malignant melanoma; Lentigo maligna melanoma; Melanoma in situ; Superficial spreading melanoma; Nodular melanoma; Acral lentiginous melanoma

**DEFINITION AND DESCRIPTION**

Melanoma is a type of skin cancer that develops when melanocytes (the cells that give the skin its tan or brown color) start to grow out of control.

Melanoma is much less common than some other types of skin cancers. But melanoma is more dangerous because it’s much more likely to spread to other parts of the body if not found and treated early.

Melanoma, which means "black tumor," is the most dangerous type of skin cancer. It grows quickly and has the ability to spread to any organ.

Melanoma comes from skin cells called melanocytes. These cells produce melanin, the dark pigment that gives skin its color. Most melanomas are black or brown in color, but some are pink, red, purple or skin-colored.

About 30% of melanomas begin in existing moles, but the rest start in normal skin. This makes it especially important to pay attention to changes in your skin because the majority of melanomas don't start as moles. However, how many moles you have may help predict your skin’s risk for developing melanoma. It’s important to know if you’re in a high-risk group for developing melanoma skin cancer. Because of the fast growth rate of melanomas, a treatment delay sometimes may mean the difference between life and death. Knowing your risk can help you be extra vigilant in watching changes in your skin and seeking skin examinations since melanomas have a 99% cure rate if caught in the earliest stages. Early detection is important because treatment success is directly related to the depth of the cancerous growth.

## **Where do skin cancers start?**

Most skin cancers start in the top layer of skin, called the *epidermis*. There are 3 main types of cells in this layer:

* **Squamous cells:** These are flat cells in the upper (outer) part of the epidermis, which are constantly shed as new ones form.
* **Basal cells:** These cells are in the lower part of the epidermis, called the *basal cell layer*. These cells constantly divide to form new cells to replace the squamous cells that wear off the skin’s surface. As these cells move up in the epidermis, they get flatter, eventually becoming squamous cells.
* **Melanocytes:** These are the cells that can become melanoma. They normally make a brown pigment called *melanin*, which gives the skin its tan or brown color. Melanin protects the deeper layers of the skin from some of the harmful effects of the sun.

The epidermis is separated from the deeper layers of skin by the **basement membrane**. When a skin cancer becomes more advanced, it generally grows through this barrier and into the deeper layers.

## **Melanoma skin cancers**

Melanoma is a cancer that begins in melanocytes.

Most melanomas start in the skin. Another name for these cancers is **cutaneous melanoma**.

Melanomas can start anywhere on the skin, but in people with lighter skin color they are more likely to start on the trunk (chest and back) in men and on the legs in women. The neck and face are other common sites.

People with darkly pigmented skin have a lower risk of melanoma at these more common sites.

### **Types of melanoma skin cancer**

There are different types of skin melanoma. The most common types are:

* **Superficial spreading melanoma:** This type makes up about 7 in 10 melanomas of the skin. These tumors tend to grow outward on the surface of the skin (at least at first), so they might be noticed as a dark spot on the skin that is changing shape and/or getting bigger. Some of these melanomas start in existing moles (see below), but others do not.
* **Nodular melanoma:** This type accounts for about 2 in 10 skin melanomas. These tumors often appear as a distinct, raised bump (nodule) on the skin that is often dark brown or black, but it can also be pink or red. This can make them hard to find early. Nodular melanomas tend to grow down into deeper layers of the skin fairly early, so they’re often at a more advanced stage than superficial spreading melanomas by the time they are found.
* **Lentigo maligna melanoma:** This type of melanoma tends to occur in older people. It often first appears as an abnormally shaped tan or brown spot in an area that gets a lot of sun (such as the face, ears, or arms), and it tends to grow slowly (or change in other ways) over time.
* **Acral lentiginous melanoma (acral melanoma):** This type of melanoma starts in areas that don’t get a lot of sun exposure, such as the palms of the hands, soles of the feet, or under the nails. Acral melanomas make up a large portion of melanomas in people with darker skin tones.

### **Melanomas in other parts of the body**

Melanomas can also form in other parts of the body, such as:

* Inside the eye (known as **ocular melanomas**). Most of these start in the uvea (the middle layer of the eyeball) and are known as **uveal melanomas**.
* Inside the nose, mouth, throat, genital, or anal area (known as **mucosal melanomas**)

These are much less common than melanoma of the skin.

## **Other types of skin cancer**

There are many other types of skin cancer. Skin cancers that are not melanomas are sometimes grouped as **non-melanoma skin cancers** because they develop from skin cells other than melanocytes. They tend to behave very differently from melanomas and are often treated with different methods.

### **Basal cell and squamous cell skin cancers**

Basal cell cancer (BCC) and squamous cell cancer (SCC) are by far the most common types of skin cancer. In fact, they are more common than any other form of cancer in the United States. These cancers (especially BCCs) are much less likely to spread (metastasize) to other parts of the body than are melanomas, so they are usually less concerning and are treated differently. These cancers are discussed in Basal and Squamous Cell Skin Cancer.

### Less common skin cancers

Other types of non-melanoma skin cancer are much less common than basal and squamous cell cancers and are treated differently. They include:

* Merkel cell carcinoma
* Kaposi sarcoma
* Cutaneous (skin) lymphoma
* Skin adnexal tumors (tumors that start in hair follicles or skin glands)
* Various types of sarcomas

Together, these types account for less than 1% of all skin cancers.

## Benign skin tumors

Many types of benign (non-cancerous) tumors can develop from different types of skin cells.

#### **Benign tumors that start in melanocytes**

A **mole** (nevus) is a benign skin tumor that develops from melanocytes. Almost everyone has some moles. Nearly all moles (nevi) are harmless, but having some types can raise your risk of melanoma.

A **Spitz nevus** is a kind of mole that sometimes looks like melanoma. It’s more common in children and teens, but it can also be seen in adults. These tumors are typically benign and don’t spread. But sometimes doctors have trouble telling Spitz nevi from true melanomas, even when looking at them under a microscope. Therefore, they are often removed, just to be safe.

#### **Benign tumors that develop from other types of skin cells**

* **Seborrheic keratoses:** tan, brown, or black raised spots with a “waxy” texture and a “stuck on” appearance
* **Hemangiomas:** benign blood vessel growths, often called *strawberry spots*
* **Lipomas:** soft growths made up of fat cells
* **Warts:** rough-surfaced growths caused by some types of human papillomavirus (HPV)

Most of these tumors rarely, if ever, turn into cancers. There are many other kinds of benign skin tumors, but most are not very common.

**RISK FACTOR**

Several risk factors can make a person more likely to develop melanoma.

## **Ultraviolet (UV) light exposure**

Exposure to ultraviolet (UV) rays is a major risk factor for most melanomas. Sunlight is the main source of UV rays. Tanning beds and sun lamps are also sources of UV rays.

While UV rays make up a very small portion of the sun’s rays, they are the main cause of the damaging effects of the sun on the skin. UV rays damage the DNA (genes) inside skin cells. Skin cancers can begin when this damage affects the genes that control skin cell growth.

The pattern and timing of the UV exposure may play a role in melanoma development. For example, melanoma on the trunk (chest and back) and legs has been linked to frequent sunburns (especially in childhood). This might also have something to do with the fact that these areas aren’t constantly exposed to UV light. Some research suggests that melanomas that start in these areas are different from those that start on the face, neck, and arms, where the sun exposure is more constant.

And different from either of these are melanomas on the palms of the hands, soles of the feet, or under the nails (known as acral lentiginous melanomas), or on internal surfaces such as the mouth and vagina (mucosal melanomas), where there has been little or no sun exposure.

## Moles

A mole (also known as a *nevus*) is a benign (non-cancerous) pigmented tumor. Babies are not usually born with moles; they often begin to appear in children and young adults.

Having many moles: Most moles will never cause any problems, but someone who has many moles is more likely to develop melanoma.

Atypical moles (dysplastic nevi): These moles look a little like normal moles but have some features of melanoma. They are often larger than other moles and have an abnormal shape or color. They can appear on skin that is exposed to the sun, as well as skin that is usually covered, such as on the buttocks or scalp.

Dysplastic nevi often run in families. A small percentage of dysplastic nevi may develop into melanomas. But most dysplastic nevi never become cancer, and many melanomas seem to arise without a pre-existing dysplastic nevus.

**Dysplastic nevus syndrome (atypical mole syndrome):** People with this inherited condition have many dysplastic nevi. If at least one close relative has had melanoma, this condition is referred to as **familial atypical multiple mole and melanoma (FAMMM) syndrome**.

People with this condition have a very high lifetime risk of melanoma, so they need to have very thorough, regular skin exams by a dermatologist (a doctor who specializes in skin problems). Sometimes full-body photos are taken to help the doctor recognize if moles are changing and growing. Many doctors recommend that these patients be taught to do monthly skin self-exams as well.

**Congenital melanocytic nevi:** Moles present at birth are called *congenital melanocytic nevi*. The lifetime risk of melanoma developing in congenital melanocytic nevi is estimated to be between 0 and 5%, depending on the size of the nevus. People with very large congenital nevi have a higher risk, while the risk is lower for those with small nevi. For example, the risk for melanoma is very low in congenital nevi that are smaller than the palm of the hand, while those that cover large portions of the back and buttocks (*bathing trunk nevi*) have significantly higher risks.

Congenital nevi are sometimes removed by surgery so that they don’t have a chance to become cancer. Whether doctors advise removing a congenital nevus depends on several factors, including its size, location, and color. Many doctors recommend that congenital nevi that are not removed should be examined regularly by a dermatologist and that the person should be taught how to do monthly skin self-exams.

Again, the chance of any single mole turning into cancer is very low. However, anyone with lots of irregular or large moles has an increased risk for melanoma.

## **Lighter skin, hair, and eye color**

The risk of melanoma is much higher for people with lighter skin color than for people with darker skin.

Among people with lighter skin, those with red or blond hair, blue or green eyes, or skin that freckles or burns easily are at increased risk.

## **Family history of melanoma**

Your risk of melanoma is higher if one or more of your first-degree relatives (parents, brothers, sisters, or children) has had melanoma. Around 1 in 10 people with melanoma have a family history of the disease.

The increased risk might be because of a shared family lifestyle of frequent sun exposure, a family tendency to have lighter skin tone, certain gene changes (mutations) that run in a family, or a combination of these factors.

For some people with a strong family history of melanoma, doctors might advise genetic counseling and testing to see if they have gene mutations that increase their risk.

## **Personal history of melanoma or other skin cancers**

A person who has already had melanoma has a higher risk of getting melanoma again. In people who’ve had several melanomas or who’ve had melanoma at an early age, doctors might advise genetic counseling and testing to see if they have gene mutations that increase their risk.

People who have had basal or squamous cell skin cancers are also at increased risk of getting melanoma.

## **Having a weakened immune system**

A person’s immune system helps the body fight off cancers of the skin and other organs. People with weakened immune systems (from certain diseases or medical treatments) are more likely to develop many types of skin cancer, including melanoma.

For example, people who get organ transplants are usually given medicines that weaken their immune system to help prevent them from rejecting the new organ. This increases their risk of melanoma.

People infected with HIV, the virus that causes AIDS, often have weakened immune systems and are also at increased risk for melanoma.

## **Being older**

The risk of melanoma increases as people age, but melanoma can also develop in younger people. In fact, melanoma is one of the most common cancers in people younger than 30 (especially younger women).

Melanoma that runs in families may occur at a younger age.

## **Being male**

In the United States, men are more likely than women to get melanoma, although this varies by age. Before age 50, the risk is higher for women; after age 50, the risk is higher in men.

## **Xeroderma pigmentosum**

Xeroderma pigmentosum (XP) is a rare, inherited condition that lowers skin cells’ ability to repair damage to their DNA. People with XP have a high risk of developing melanoma and other skin cancers when they are young, especially on sun-exposed areas of their skin.

**CAUSES**

## **Gene changes that might lead to melanoma**

DNA is the chemical in each of our cells that makes up our **genes**, which control how our cells function. We usually look like our parents because they are the source of our DNA. But our genes affect more than just how we look.

Some genes control when our cells grow and divide into new cells, repair mistakes in DNA, or cause cells to die when they’re supposed to. If these genes aren’t working properly, it can lead to cells growing out of control. For example:

* Changes in genes that normally help cells grow, divide, or stay alive can lead to these genes being more active than they should be, causing them to become **oncogenes**. These genes can result in cells growing out of control.
* Genes that normally help keep cell division under control or cause cells to die at the right time are known as **tumor suppressor genes**. Changes that turn off these genes can result in cells growing out of control.
* Some genes normally help repair mistakes in a cell’s DNA. Changes that turn off these **DNA repair genes** can result in the buildup of DNA changes within a cell, which might lead to them growing out of control.

Mutations or other changes in any of these types of genes might lead to cells growing out of control. Changes in several different genes are usually needed for a cell to become a cancer cell.

## **Acquired gene mutations**

Most often, gene changes related to melanoma are acquired during a person’s lifetime and are not passed on to a person’s children (inherited). Sometimes these acquired mutations seem to happen randomly within a cell, without having a clear cause. At other times, they likely happen as the result of exposure to an outside cause.

For example, ultraviolet (UV) rays are a major cause of melanoma. Most UV rays come from sunlight, but some can come from man-made sources such as tanning beds. UV rays can damage the DNA in skin cells. Sometimes this affects certain genes that control how the cells grow and divide. If these genes no longer work properly, the affected cells may become cancer cells.

In many cases a melanoma might not appear until many years after the DNA damage from UV rays has been done. Children and young adults often get a lot of intense sun exposure that might not result in cancer until many years or even decades later.

The most common change in melanoma cells is a mutation in the *BRAF* oncogene, which is found in about half of all melanomas. Other genes that can be affected in melanoma include *NRAS, CDKN2A,* and *NF1*. (Usually only one of these genes is affected.)

Melanomas that start on the palms of the hands, soles of the feet, or under the nails (known as acral lentiginous melanomas), or on internal surfaces such as the mouth and vagina (mucosal melanomas), often have different gene changes than those in melanomas that develop in sun-exposed areas, such as changes in the *C-KIT* (or just *KIT*) gene.

## Inherited gene mutations

Less often, people inherit gene changes from a parent that clearly raise their risk of melanoma.

Familial (inherited) melanomas most often have changes in tumor suppressor genes, such as *CDKN2A* (also known as *p16*), *CDK4,* or *BAP1,* that prevent these genes from doing their normal job of controlling cell growth. This could eventually lead to cancer.

For some people who have a strong family history of melanoma or who have had several melanomas (or melanomas that started at an early age), doctors might advise [genetic counseling and testing](https://www.cancer.org/cancer/types/melanoma-skin-cancer/causes-risks-prevention/genetic-counseling-and-testing-for-people-at-high-risk-of-melanoma.html) to see if they have a mutation in one of these genes (or possible other genes) that increases their risk.

Some people, such as those with **xeroderma pigmentosum (XP),** inherit a change in one of the *XP* (*ERCC*) genes, which normally help to repair damaged DNA inside the cell. Changes in one of these genes can lead to skin cells that have trouble repairing DNA damaged by UV rays, so these people are more likely to develop melanoma, especially on sun-exposed parts of the body.

## **Gene mutations can sometimes affect treatment**

Some of the gene changes found in melanoma cells have proven to be good targets for drugs to help treat this disease. For example, drugs that specifically target cells with changes in the *BRAF* gene or the *KIT* gene are now used to treat advanced melanomas with these changes

## **Signs and symptoms of melanoma**

* The most important warning sign of melanoma is **a new spot on the skin or a spot that is changing in size, shape, or color**.
* Another important sign is **a spot that looks different from all of the other spots on your skin.** (This is sometimes known as "the ugly duckling sign.")

If you have one of these warning signs, have your skin checked by a doctor.

## **What should a normal mole look like?**

Most people have moles, and almost all moles are harmless. A normal mole is:

* Usually an evenly colored brown, tan, or black spot on the skin
* Either flat or raised
* Round or oval
* Generally smaller than 6 millimeters (about ¼ inch) across (about the width of a pencil eraser)

Some moles can be present at birth, but most appear during childhood or young adulthood. New moles that appear later in life should be checked by a doctor.

Once a mole has developed, it will usually stay the same size, shape, and color for many years. Some moles may eventually fade away.

It’s important to recognize changes in a mole's size, shape, color, or texture. These changes could suggest a melanoma is developing.

## **The ABCDE rule for signs of melanoma**

The **ABCDE rule** is another guide to the usual signs of melanoma. Be on the lookout and tell your doctor about spots that have any of the following features:

* **A is for Asymmetry:** One half of a mole or birthmark does not match the other.
* **B is for Border:** The edges are irregular, ragged, notched, or blurred.
* **C is for Color:** The color is not the same all over and may include different shades of brown or black, or sometimes with patches of pink, red, white, or blue.
* **D is for Diameter:** The spot is larger than 6 millimeters across (about ¼ inch – the size of a pencil eraser), although melanomas can sometimes be smaller than this.
* **E is for Evolving:** The mole is changing in size, shape, or color.

Some melanomas don’t fit these rules. It’s important to tell your doctor about any changes or new spots on your skin, or growths that look different from the rest of your moles.

## **Other signs of melanoma on the skin**

Other warning signs are:

* A sore that doesn’t heal
* Spread of pigment from the border of a spot into surrounding skin
* Redness or a new swelling beyond the border of the mole
* Change in sensation, such as itchiness, tenderness, or pain
* Change in the surface of a mole – scaliness, oozing, bleeding, or the appearance of a lump or bump

Be sure to show your doctor any areas that concern you. It’s sometimes hard to tell the difference between melanoma and an ordinary mole, even for doctors, so it’s important to show your doctor any mole that you are unsure of.

## **Signs of hidden melanoma**

While most melanomas start on sun-exposed skin, a small portion of melanomas start in places that aren't exposed to the sun. These might look different from melanomas on the skin. For example:

* **Under a fingernail or toenail (acral melanoma):** May appear as a dark line or streak in the nail.
* **On the palms or soles (acral melanoma):** May appear as dark, irregular areas.
* **In the eye (uveal melanoma):** May appear as a dark spot in the colored part of the eye (iris).
* **In the mouth, nose, and genitals (mucosal melanoma):** May develop as dark spots or irregular areas in these tissues.

It’s important to show a doctor anything that concerns you in these areas as well.

### **DIAGNOSIS**

## **Skin self-exam**

**knowing your own skin** is important to finding skin cancer early. You should know the pattern of moles, blemishes, freckles, and other marks on your skin so that you’ll notice any new growths or changes in existing moles or other spots.

Many doctors recommend checking your own skin, preferably once a month. **Skin self-exams** are best done in a well-lit room in front of a full-length mirror. Use a hand-held mirror to help look at areas that are hard to see, such as the backs of your thighs. Examine all parts of your body, including the palms of your hands and soles of your feet, as well as your scalp, ears, nails, and back (in men, the back is a common place for melanomas to start). A spouse, partner, or close friend or family member can also help you with these exams, especially for those hard-to-see areas, such as your scalp and back.

## **Exam by a healthcare professional**

Some doctors and other health care professionals do skin exams as part of routine health checkups.

If your primary doctor finds any unusual moles or other suspicious areas, they may refer you to a **dermatologist**, a doctor who specializes in skin problems.

Regular skin exams are especially important for people at higher risk of melanoma, such as people with dysplastic nevus syndrome, people with a strong family history of melanoma, and people who have had melanoma before. If you have many moles, your doctor might advise taking full-body photos so your moles can be tracked over time and new ones can be seen more readily. (This is sometimes called *total body photography* or *mole mapping*.) Talk to your doctor about how often you should have your skin examined.

If you have an abnormal area on your skin that might be cancer, your doctor will examine it and might do tests to find out if it is melanoma, another type of skin cancer, or some other skin condition.

If you are being seen by your primary doctor and melanoma is suspected, you may be referred to a **dermatologist**, a doctor who specializes in skin diseases, who will look at the area more closely.

If melanoma is found, other tests might then be done to learn more about it, such as if it has spread to other areas of the body.

## **Special techniques to look at the skin**

Dermatologists sometimes use special tools when trying to determine if an abnormal area might be a melanoma, and therefore if a skin biopsy (see below) is needed.

### **Dermoscopy**

Dermatologists often use dermoscopy, also known as *dermatoscopy, epiluminescence microscopy [ELM], or surface microscopy*, to get a closer look at abnormal spots on the skin. In this technique, the doctor uses a dermatoscope, which is a special magnifying lens and light source held near the skin. Sometimes a thin layer of alcohol or oil is put on the skin before using this instrument.

Dermoscopy allows doctors to look at a suspicious area more closely, even giving them the ability to see some structures below the surface of the skin that can’t be seen with the naked eye.

Digital images of the area can also be taken during dermoscopy. These can be used to see if an area changes over time. In some systems, the images can be analyzed by a computer, which can help the doctor determine if the area might be a melanoma.

### **Reflectance confocal microscopy (RCM)**

RCM is another technique that lets the doctor look at an abnormal area of skin to a certain depth without having to cut into the skin. In this technique, a low-powered laser is aimed at the suspicious area. The light from the laser enters the upper layers of the skin and reflects off the structures there. A special microscope detects the light as it bounces back, which is used to create a detailed, three-dimensional image of the area. This can help the doctor determine if the area needs to be biopsied.

RCM may be especially useful for people with many unusual moles, as it can cut down on the number of skin biopsies these people might need. RCM might also be helpful in determining the edges of a melanoma, which could help during surgery.

### **Other methods that don’t require cutting into the skin**

Other approaches to help doctors get a better idea if an abnormal area is a melanoma are also being developed. For example:

* Some handheld **spectroscopic devices** detect reflections of different wavelengths of light or other forms of energy to help determine if an area is likely to be a melanoma.
* In another approach, known as **adhesive patch testing**, a sticky patch is placed over the area. When it’s removed, some skin cells from the area come with it, which can then be tested for certain gene changes that are often linked with melanoma.

## **Skin biopsy**

If the doctor thinks a spot might be a melanoma, the suspicious area will be removed and sent to a lab to be looked at under a microscope. This is called a *skin biopsy*.

There are many ways to do a skin biopsy. The doctor will choose which one is best based on the size of the affected area, where it is on your body, and other factors. No matter which type of biopsy is done, it should remove as much of the suspected area as possible so that an accurate diagnosis can be made.

Any biopsy is likely to leave at least a small scar. Different methods can result in different types of scars, so ask your doctor about scarring before the biopsy.

Skin biopsies are done using a local anesthetic (numbing medicine), which is injected into the area with a very small needle. You will likely feel a small prick and a little stinging as the medicine is injected, but you should not feel any pain during the biopsy.

### **Skin Biopsy and Treatment Procedures**

### **Deep shave (tangential) biopsy**

For this type of biopsy, also known as **saucerization**, the doctor shaves off the top layers of the skin with a small surgical blade. Bleeding from the biopsy site is stopped by applying an ointment, a chemical that stops bleeding, or a small electrical current to cauterize the wound.

A shave biopsy is useful in diagnosing many types of skin diseases and in sampling moles when the risk of melanoma is very low. If this type of biopsy is used for a suspected melanoma, it’s important that the biopsy blade will go deep enough to get below the suspicious area. Otherwise, if it is a melanoma, the biopsy sample may not be thick enough to measure how deeply the cancer has invaded the skin.

### **Punch biopsy**

For a punch biopsy, the doctor uses a tool that looks like a tiny, round cookie cutter to remove a deeper sample of skin. The doctor rotates the punch biopsy tool on the skin until it cuts through all the layers of the skin. The sample is then removed, and the edges of the biopsy site are often stitched together.

### **Excisional and incisional biopsies**

To examine a tumor that might have grown into deeper layers of the skin, the doctor may use an excisional (or less often, an incisional) biopsy.

* An **excisional biopsy** removes the entire tumor (along with a small margin of normal skin around it). This is usually the preferred method of biopsy for suspected melanomas if it can be done, although this isn’t always possible.
* An **incisional biopsy** removes only a portion of the tumor.

For these types of biopsies, a surgical knife is used to make an elliptical or circular cut through the full thickness of skin. The skin is then removed for examination, and the edges of the cut are usually stitched together.

## **Biopsies of melanoma that may have spread**

Biopsies of areas other than the skin may be needed in some cases. For example, if melanoma has already been diagnosed on the skin, nearby lymph nodes may be biopsied to see if the cancer has spread to them.

Rarely, biopsies may be needed to figure out what type of cancer someone has. For example, some melanomas can spread so quickly that they reach the lymph nodes, lungs, brain, or other areas while the original skin melanoma is still very small. Sometimes these tumors are found with imaging tests (such as CT scans) or other exams even before the melanoma on the skin is discovered. In other cases, they may be found long after a skin melanoma has been removed, so it’s not clear if it’s the same cancer.

In still other cases, melanoma may be found somewhere in the body without ever finding a spot on the skin. This may be because some skin lesions go away on their own (without any treatment) after some of their cells have spread to other parts of the body. Melanoma can also start in internal organs, but this is very rare, and if melanoma has spread widely throughout the body, it may not be possible to tell exactly where it started.

When melanoma has spread to other organs, it can sometimes be confused with a cancer starting in that organ. For example, melanoma that has spread to the lung might be confused with a primary lung cancer (cancer that starts in the lung).

Special lab tests can be done on the biopsy samples that can tell whether it is a melanoma or some other kind of cancer. This is important because different types of cancer are treated differently.

Biopsies of suspicious areas inside the body often are more involved than those used to sample the skin.

### **Fine needle aspiration (FNA) biopsy**

Fine needle aspiration (FNA) isn’t used to biopsy suspicious moles. But it may be used to biopsy large lymph nodes near a melanoma to find out if the melanoma has spread to them.

For this type of biopsy, the doctor uses a syringe with a thin, hollow needle to remove very small pieces of a lymph node or tumor. The needle is smaller than the needle used for a blood test. A local anesthetic is sometimes used to numb the area first. This test rarely causes much discomfort and does not leave a scar.

If the lymph node is just under the skin, the doctor can often feel it well enough to guide the needle into it. For a suspicious lymph node deeper in the body or a tumor in an organ such as the lung or liver, an imaging test such as ultrasound or a CT scan is often used to help guide the needle into place.

An FNA is not as invasive as some other types of biopsies, but it may not always collect enough of a sample to tell if a suspicious area is melanoma. In these cases, a more invasive type of biopsy may be needed.

### **Surgical (excisional) lymph node biopsy**

This procedure can be used to remove an enlarged lymph node through a small incision (cut) in the skin. A local anesthetic (numbing medicine) is generally used if the lymph node is just under the skin, but the person may need to be sedated or even asleep (using general anesthesia) if the lymph node is deeper in the body.

This type of biopsy is often done if a lymph node’s size suggests the melanoma has spread there but an FNA of the node wasn’t done or didn’t find any melanoma cells.

### **Sentinel lymph node biopsy**

If melanoma has been diagnosed and has any concerning features (such as being at least a certain thickness), a sentinel lymph node biopsy (SLNB) is often done to see if the cancer has spread to nearby lymph nodes, which in turn might affect treatment options. This test can be used to find the lymph nodes that are likely to be the first place the melanoma would go if it has spread. These lymph nodes are called *sentinel nodes* (they stand sentinel, or watch, over the tumor, so to speak).

To find the sentinel lymph node (or nodes), a doctor injects a small amount of a radioactive substance into the area of the melanoma. After giving the substance time to travel to the lymph node areas near the tumor, a special camera is used to see if it collects in one or more sentinel lymph nodes. Once the radioactive area has been marked, the patient is taken for surgery, and usually a blue dye is injected in the same place the radioactive substance was injected. A small incision is then made in the marked area, and the lymph nodes are then checked to find which one(s) became radioactive and/or turned blue. These sentinel nodes are removed and looked at under a microscope.

If there are no melanoma cells in the sentinel nodes, no more lymph node surgery is needed because it is very unlikely the melanoma would have spread beyond this point. If melanoma cells are found in the sentinel node, the remaining lymph nodes in this area are typically removed and looked at as well. This is known as a *lymph node dissection.*

If a lymph node near a melanoma is abnormally large, a sentinel node biopsy probably won’t be needed. The enlarged node is simply biopsied.

## **Lab tests of biopsy samples**

Samples from any biopsies will be sent to a lab, where a doctor called a **pathologist** will look at them under a microscope for melanoma cells. Often, skin samples are sent to a **dermatopathologist**, a doctor who has special training in looking at skin samples.

If the doctor can’t tell for sure if melanoma cells are in the sample just by looking at it, special lab tests will be done on the cells to try to confirm the diagnosis. These might include:

* Immunohistochemistry (IHC)
* Fluorescence in situ hybridization (FISH)
* Comparative genomic hybridization (CGH)
* Gene expression profiling (GEP)
* Next-generation sequencing (NGS)

If melanoma is found in the samples, the pathologist will look at certain important features such as the tumor thickness and mitotic rate (the portion of cells that are actively dividing).

### **Molecular testing for certain gene changes**

For some people with melanoma, biopsy samples (or blood samples) may be tested to see if the cancer cells have mutations (changes) in certain genes. This type of testing, sometimes referred to as biomarker testing, might affect a person’s treatment options, especially if the melanoma has spread.

For example, about half of melanomas have mutations in the *BRAF* gene. Some drugs used to treat advanced melanomas are only likely to work if the cells have *BRAF* gene mutations, so this test is important in helping to determine treatment options.

Tests to look for changes in other genes that could affect treatment options might be done as well. These might include tests for changes in genes such as *C-KIT*, *NRAS*, *ALK*, *ROS1*, and the *NTRK* genes. These gene changes aren’t common in melanomas, but some targeted drugs might be a treatment option if one of these changes is found.

## **Imaging tests**

Imaging tests use x-rays, magnetic fields, or radioactive substances to create pictures of the inside of the body. They are used mainly to look for the possible spread of melanoma to lymph nodes or other organs. **These tests are not needed for most people with very early-stage melanoma, which is very unlikely to have spread.**

Imaging tests can also be done to help determine how well treatment is working or to look for possible signs of cancer coming back (recurring) after treatment.

### **Chest x-ray**

This test might be done to help determine if melanoma has spread to the lungs, although a CT scan of the chest (see below) is often done instead.

### **Ultrasound**

Ultrasound uses sound waves and their echoes to create images of the inside of your body on a computer screen. This test might be used to look at the lymph nodes near the tumor, especially if it’s not clear if they’re enlarged based on a physical exam. Ultrasound is typically quick and easy to do, and it doesn’t expose you to radiation.

**Ultrasound-guided needle biopsy:** Ultrasound can also be used to help guide a biopsy needle into a suspicious lymph node.

### **Computed tomography (CT) scan**

A CT scan uses x-rays to make detailed, cross-sectional images of your body. Unlike a regular x-ray, CT scans can show the detail in soft tissues (such as internal organs). This test can show if any lymph nodes are enlarged or if organs such as the lungs or liver have suspicious spots, which might be from the spread of melanoma.

**CT-guided needle biopsy:** CT scans can also be used to help guide a biopsy needle into a suspicious area within the body.

### **Magnetic resonance imaging (MRI)**

MRIs use radio waves and strong magnets instead of x-rays to create detailed images of parts of your body. MRIs can be very helpful in looking at the brain and spinal cord.

### **Positron emission tomography (PET) scan**

A PET scan can help show if the cancer has spread to lymph nodes or other parts of the body. It is most useful in people with more advanced stages of melanoma.

For this test, you are injected with a slightly radioactive form of sugar, which collects mainly in cancer cells. A special camera is then used to create a picture of areas of radioactivity in the body.

**PET/CT scan:** Many centers have special machines that do both a PET and CT scan at the same time (PET/CT scan). This lets the doctor compare areas of higher radioactivity on the PET scan with the more detailed appearance of that area on the CT scan.

## **Blood tests**

Blood tests aren’t used to diagnose melanoma, but some tests may be done before or during treatment, especially for more advanced melanomas.

Doctors often test a person’s blood for levels of a substance called **lactate dehydrogenase (LDH)** before treatment. If the melanoma has spread to distant parts of the body, a high LDH level is a sign that the cancer may be harder to treat. This can affect the stage of the cancer.

Other tests of **blood cell count,** and **blood chemistry levels** may be done in a person who has advanced melanoma to see how well the bone marrow (where new blood cells are made), liver, and kidneys are working before and during treatment.

## **Treating stage 0 melanoma**

Stage 0 melanoma (melanoma in situ) has not grown deeper than the top layer of the skin (the epidermis). It is usually treated by surgery (wide excision) to remove the melanoma and a small margin of normal skin around it. The removed sample is then sent to a lab to be looked at with a microscope. If cancer cells are seen at the edges of the sample, a second, wider excision of the area may be done.

Some doctors may consider the use of imiquimod cream (Zyclara) or radiation therapy after surgery if not all the cancer cells can be removed for some reason, although not all doctors agree with this.

For melanomas in sensitive areas on the face, some doctors may use Mohs surgery or even imiquimod cream if surgery might be disfiguring, although not all doctors agree with these uses.

## **Treating stage I melanoma**

Stage I melanomas have grown into deeper layers of the skin, but they haven’t grown beyond the area where they started.

These cancers are typically treated by wide excision (surgery to remove the tumor as well as a margin of normal skin around it). The width of the margin depends on the thickness and location of the melanoma. Most often, no other treatment is needed.

Some doctors may recommend a sentinel lymph node biopsy (SLNB) to look for cancer in nearby lymph nodes, especially if the melanoma is stage IB or has other traits that make it more likely to have spread. You and your doctor should discuss this option.

**If the SLNB does not find cancer cells in the lymph nodes**, then no further treatment is needed, although close follow-up is still important.

**If cancer cells are found on the SLNB** (which changes the cancer stage to stage III – see below), a lymph node dissection (removal of all lymph nodes near the cancer) might be recommended. Another option might be to watch the lymph nodes closely by getting an imaging test such as ultrasound of the nodes every few months.

If the SLNB found cancer, adjuvant (additional) treatment with immune checkpoint inhibitors or targeted therapy drugs (if the melanoma has a *BRAF* gene mutation) might be recommended to try to lower the chance the melanoma will come back. Other drugs or perhaps vaccines might also be options as part of a clinical trial.

## **Treating stage II melanoma**

Stage II melanomas have grown deeper into the skin than stage I melanomas, but they still haven’t grown beyond the area in the skin where they started.

Wide excision (surgery to remove the melanoma and a margin of normal skin around it) is the standard treatment for these cancers. The width of the margin depends on the thickness and location of the melanoma.

Because the melanoma may have spread to nearby lymph nodes, many doctors recommend a sentinel lymph node biopsy (SLNB) as well. This is an option that you and your doctor should discuss.

**If a SLNB is done and does not find cancer cells in the lymph nodes**, then sometimes no further treatment is needed, but close follow-up is still important.

For certain stage II melanomas, the immune checkpoint inhibitor pembrolizumab (Keytruda) might be given after surgery to help reduce the risk of the cancer returning. Radiation therapy to the area might be another option, especially if the melanoma has features that make it more likely to come back.

**If the SLNB finds that the sentinel node contains cancer cells** (which changes the cancer stage to stage III – see below), then a lymph node dissection (where all the lymph nodes in that area are surgically removed) might be recommended. Another option might be to watch the lymph nodes closely with an imaging test such as ultrasound of the nodes every few months.

Whether or not the lymph nodes are removed, adjuvant (additional) treatment with immune checkpoint inhibitors or targeted therapy drugs (if the melanoma has a *BRAF* gene mutation) might be recommended to try to lower the chance the melanoma will come back. Other drugs or perhaps vaccines might also be options as well as part of a clinical trial.

Your doctor will discuss the best options with you depending on the details of your situation.

## **Treating stage III melanoma**

These cancers have spread to nearby areas in the skin or lymph vessels, or they have reached the nearby lymph nodes.

Surgical treatment for stage III melanoma usually requires wide excision of the primary tumor as in earlier stages, along with a lymph node dissection (where all the nearby lymph nodes are surgically removed).

After surgery, (additional) adjuvant treatment with immune checkpoint inhibitors or with targeted therapy drugs (for cancers with *BRAF* gene changes) may help lower the risk of the melanoma coming back. Other drugs or perhaps vaccines may also be recommended as part of a clinical trial to try to reduce the chance the melanoma will come back. Another option is to give radiation therapy to the areas where the lymph nodes were removed, especially if many of the nodes contain cancer.

If melanoma tumors are found in nearby lymph vessels in or just under the skin (known as **in-transit tumors**), they are removed, if possible. Other options might include injections of the T-VEC vaccine (Imlygic), interleukin-2 (IL-2), or Bacille Calmette-Guerin (BCG) vaccine directly into the melanoma; radiation therapy; or applying imiquimod cream. For melanomas on an arm or leg, another option might be isolated limb perfusion or isolated limb infusion (infusing just the limb with chemotherapy). Other possible treatments might include targeted therapy drugs (for melanomas with a *BRAF* or *C-KIT* gene change), immunotherapy, or chemotherapy.

Some stage III melanomas might be hard to cure with current treatments, so taking part in a clinical trial of newer treatments might be a good option.

## **Treating stage IV melanoma**

Stage IV melanomas have already spread (metastasized) to other parts of the body, such as distant lymph nodes, areas of skin, or other organs.

Skin tumors or enlarged lymph nodes causing symptoms can often be removed by surgery or treated with radiation therapy.

**If there are only a few metastases**, surgery to remove them might sometimes be an option, depending on where they are and how likely they are to cause symptoms. Metastases that can’t be removed may be treated with radiation or with injections of the T-VEC vaccine (Imlygic) directly into the tumors. In either case, this is often followed by adjuvant treatment with medicines such as immunotherapy or targeted therapy drugs.

The **treatment of widespread melanomas** has changed in recent years as newer forms of immunotherapy and targeted drugs have been shown to be more effective than chemotherapy.

Immunotherapy drugs called **checkpoint inhibitors** are often the first treatment. These drugs can shrink tumors for long periods of time in some people. Options might include:

* Pembrolizumab (Keytruda) or nivolumab (Opdivo) alone
* Nivolumab combined with relatlimab (Opdualag)
* Nivolumab or pembrolizumab, plus ipilimumab (Yervoy)

Combinations of checkpoint inhibitors seem to be more effective, although they’re also more likely to result in serious side effects, especially if they contain ipilimumab.

People who get any of these drugs need to be watched closely for serious side effects.

In about half of all melanomas, the cancer cells have ***BRAF* gene changes**. These melanomas often respond to treatment with targeted therapy drugs – typically a combination of a *BRAF* inhibitor and a *MEK* inhibitor. However, the immune checkpoint inhibitors mentioned above are often tried first, as this seems to be more likely to help for longer periods of time. Another option might be a combination of targeted drugs plus the immune checkpoint inhibitor atezolizumab (Tecentriq).

While immunotherapy is often used before targeted therapy, there might be situations where it makes sense to use targeted therapy first. For example, the targeted drugs are more likely to shrink tumors quickly, so they might be preferred in cases where this is important. In either case, if one type of treatment isn’t working, the other can be tried.

A small portion of melanomas have changes in the *C-KIT* gene. These melanomas might be helped by targeted drugs such as imatinib (Gleevec) and nilotinib (Tasigna), although these drugs often stop working eventually.

Rarely, melanomas might have changes in other genes such as *NRAS, ROS1, ALK*, or the *NTRK* genes, which can be treated with targeted drugs.

Immunotherapy using other medicines might be an option if immune checkpoint inhibitors or other treatments aren’t working. Options might include:

* Interleukin-2 (IL-2) (also known as aldesleukin)
* Lifileucel (Amtagvi), a type of tumor-infiltrating lymphocyte (TIL) therapy

These treatments can cause serious side effects in some people, so they are usually given in the hospital.

Chemotherapy (chemo) can help some people with stage IV melanoma, but other treatments are usually tried first. Dacarbazine (DTIC) and temozolomide (Temodar) are the chemo drugs used most often, either by themselves or combined with other drugs. Even when chemo shrinks these cancers, the cancer usually starts growing again over time.

It’s important to carefully consider the possible benefits and side effects of any recommended treatment before starting it.

Because stage IV melanoma is often hard to cure with current treatments, people may want to think about taking part in a clinical trial. Many studies are now looking at new targeted drugs, immunotherapies, and combinations of different types of treatments.

## **Treating recurrent melanoma**

Treatment of melanoma that comes back after initial treatment depends on where in the body the melanoma is, what treatments a person has already had, the person’s overall health and preferences, and other factors.

### **Local recurrence**

Melanoma might come back in the skin near the site of the original tumor, sometimes even in the scar from the surgery. In general, these local (skin) recurrences are treated with surgery similar to what would be recommended for a primary melanoma. This might include a sentinel lymph node biopsy (SLNB). Depending on the results of the SLNB, other treatments might be recommended as well.

### **In-transit recurrence**

If melanoma recurs in nearby lymph vessels in or just under the skin (known as **in-transit recurrence**), it should be removed with surgery, if possible. Other options might include injections of the T-VEC vaccine (Imlygic), interleukin-2 (IL-2), or Bacille Calmette-Guerin (BCG) vaccine directly into the melanoma; radiation therapy; or applying imiquimod cream. For melanomas on an arm or leg, another option might be isolated limb perfusion or isolated limb infusion (infusing just the limb with chemotherapy). Other treatments might include targeted therapy (for melanomas with a *BRAF* or *C-KIT* gene change), immunotherapy, or chemotherapy.

### **Recurrence in nearby lymph nodes**

If the nearby lymph nodes weren’t removed during the initial treatment, the melanoma might come back in these lymph nodes. Lymph node recurrence is typically treated by lymph node dissection if it can be done, sometimes followed by adjuvant (additional) treatments such as radiation therapy and/or immunotherapy or targeted therapy (for cancers with *BRAF* gene changes). If surgery is not an option, radiation therapy or systemic treatment (immunotherapy, targeted therapy, or chemo) can be used.

### **Recurrence in other parts of the body**

Melanoma might also come back in distant parts of the body. Almost any organ can be affected. Most often, the melanoma comes back in the lungs, bones, liver, or brain. Treatment for these recurrences is generally the same as for stage IV melanoma (see above). Melanomas that recur on an arm or leg may be treated with isolated limb perfusion/infusion chemotherapy.

Melanoma that comes back in the brain can be hard to treat. Single tumors can sometimes be removed by [surgery](https://www.cancer.org/cancer/types/melanoma-skin-cancer/treating/surgery.html). Radiation therapy to the brain (stereotactic radiosurgery or whole-brain radiation therapy) may help as well. Systemic treatments (immunotherapy, targeted therapy, or chemo) might also be options.

As with other stages of melanoma, people with recurrent melanoma may want to think about taking part in a clinical trial of newer treatments.

**PREVENTION**

## **Limit your exposure to ultraviolet (UV) rays**

The most important way to lower your risk of melanoma is to protect yourself from exposure to UV rays. Practice sun safety when you are outdoors.

### **Seek shade**

Simply staying in the shade is one of the best ways to limit your UV exposure.

### **Slip! Slop! Slap! ® and Wrap!**

If you are going to be in the sun, this catchphrase can help you remember some of the key steps you can take to protect yourself from UV rays:

* Slip on a shirt.
* Slop on sunscreen.
* Slap on a hat.
* Wrap on sunglasses to protect the eyes and sensitive skin around them.

### **Avoid using tanning beds and sunlamps**

Many people believe the UV rays of tanning beds are harmless. This is not true. Tanning lamps give off UV rays, which can cause long-term skin damage and can contribute to skin cancer. Tanning bed use has been linked with an increased risk of melanoma, especially if it is started before a person is 30 years old. Most dermatologists (skin doctors) and health organizations recommend not using tanning beds and sun lamps.

### **Protect children from the sun**

Children need special attention, since they tend to spend more time outdoors and can burn more easily. Parents and other caregivers should protect children from excess sun exposure by using the steps above. Children need to be taught about the dangers of too much sun exposure as they become more independent.

### **Watch for new, changing, or abnormal moles**

Checking your skin regularly may help you spot any new or abnormal moles or other growths and show them to your doctor before they even have a chance to turn into skin cancer.

## **Avoid weakening your immune system (when possible)**

Having a weakened immune system increases your risk of getting melanoma and other types of skin cancer.

Infection with HIV, the virus that causes AIDS, can weaken the immune system. Avoiding known risk factors for HIV infection, such as intravenous (IV) drug use and having unprotected sex with many partners, might lower your risk of skin cancer, as well as many other types of cancer.

Some people need to take medicines to suppress their immune system. This includes people who have had organ transplants and some people with autoimmune diseases. People with cancer also sometimes need to take medicines such as chemotherapy, which can lower their immune function. For these people, the benefit from taking these medicines will likely far outweigh the small increased risk of getting skin cancer.

## **Outlook / Prognosis**

Most skin cancers can be cured if they’re treated before they have a chance to spread. However, more advanced cases of melanoma can be fatal. The earlier skin cancer is found and removed, the better your chances for a full recovery.

**Living With**

### **When should I call my doctor?**

You should have a skin examination by a doctor if you have any of the following:

* A personal history of skin cancer or atypical moles (nevi).
* A family history of skin cancer.
* A history of intense sun exposure as a young person and painful or blistering sunburns.
* New or numerous large moles.
* A mole that changes in size, color or shape.
* Any mole that itches, bleeds or is tender.

## **Complications**

## Management of malignant melanoma involves a combination of surgery, radiation therapy in very few cases, adjuvant immunotherapy, or targeted therapies. These interventions, which are proven to improve survival and long-term outcomes associated with melanoma, are associated with some potential complications.

## Surgery can be associated with complications, including bleeding, infection, damage to nerves, and scarring. Cosmetic outcomes from surgery might also have psychological implications, including depression and anxiety. In patients undergoing lymph node dissection, there is an increased incidence of lymphedema. In a few patients who receive radiation to the lymph node basin for extensive lymph node involvement, there could be potential radiotherapy-related complications, including skin irritation, fatigue, risk of secondary malignancies, and lymphedema.

## Adjuvant therapies, including immunotherapies and targeted therapies, have various potential adverse effects that could complicate the treatment course. These include immunotherapy-related adverse events (irAEs) and targeted therapy-related drug-induced fevers, risk of secondary malignancies, fatigue, risk of infections, and immunosuppression.

## All these potential complications can affect the quality of life of a patient, along with the inability to complete the treatment course, which could result in an increased risk of recurrence and poorer survival outcomes. Therefore, vigilance is essential due to the potential complications of various management strategies. Additionally, selecting the appropriate therapy and monitoring patients is critical.

## **Diagnostic Considerations**

## Differentials to consider in the diagnosis of malignant melanoma include the following conditions:

* Benign melanocytic lesions
* Dysplastic nevus
* Squamous cell carcinoma
* Metastatic tumors to the skin
* Blue nevus
* Epithelioid (Spitz) tumor
* Pigmented spindle cell tumor
* Halo nevus
* Atypical fibroxanthoma
* Pigmented actinic keratosis
* Sebaceous carcinoma
* Histiocytoid hemangioma

Also see the following:

* Lentigo Maligna Melanoma
* Oral Malignant Melanoma
* Head and Neck Mucosal Melanomas

## Differential Diagnoses

* Basal Cell Carcinoma
* Lentigo Maligna Melanoma
* Mycosis Fungoides

**Benign Melanocytic Lesions**  
Benign melanocytic lesions are noncancerous proliferations of melanocytes presenting as well-circumscribed, symmetric, pigmented macules, papules, or nodules.

**Dysplastic Nevus**  
Atypical moles with clinical and histologic features between benign nevi and melanoma. They tend to be larger, asymmetric, with irregular borders and varied pigmentation.

**Squamous Cell Carcinoma (SCC)**  
A common skin cancer arising from keratinocytes, presenting as scaly, erythematous plaques, nodules, or ulcers. It may develop on sun-exposed skin and can metastasize if untreated.

**Metastatic Tumors to the Skin**  
Secondary skin involvement by internal malignancies, presenting as nodules or plaques, often firm and rapidly growing. Common primaries include breast, lung, and melanoma.

**Blue Nevus**  
A benign melanocytic lesion characterized by a blue to blue-black color due to melanin deep in the dermis. It is typically a small, well-defined papule or nodule.

**Epithelioid (Spitz) Tumor**  
A benign melanocytic tumor often seen in children and young adults, presenting as a pink to reddish dome-shaped papule or nodule.

**Pigmented Spindle Cell Tumor**  
A rare melanocytic lesion composed predominantly of spindle-shaped melanocytes, which can be benign or malignant and requires histologic evaluation.

**Halo Nevus**  
A benign melanocytic nevus surrounded by a depigmented (halo) area due to immune-mediated melanocyte destruction.

**Atypical Fibroxanthoma**  
A superficial skin tumor usually occurs on sun-damaged skin of elderly individuals. It presents as a rapidly growing nodule and is considered a low-grade sarcoma.

**Pigmented Actinic Keratosis**  
A precancerous lesion caused by sun damage, presenting as a scaly, pigmented patch on sun-exposed skin.

**Sebaceous Carcinoma**  
A rare aggressive skin cancer arising from sebaceous glands, often presenting as a painless nodule on the eyelids or head and neck region.

**Histiocytoid Hemangioma**  
A benign vascular tumor characterized by red to purple papules or nodules, often on the head and neck.

**Lentigo Maligna Melanoma**  
A melanoma subtype arising from lentigo maligna, typically on chronically sun-exposed skin of elderly individuals.

**Oral Malignant Melanoma**  
A rare but aggressive melanoma arising on mucosal surfaces of the oral cavity, presenting as pigmented or nonpigmented lesions with ulceration.

**Head and Neck Mucosal Melanomas**  
Aggressive melanomas arising on mucosal surfaces of the head and neck region, often diagnosed late with poor prognosis.

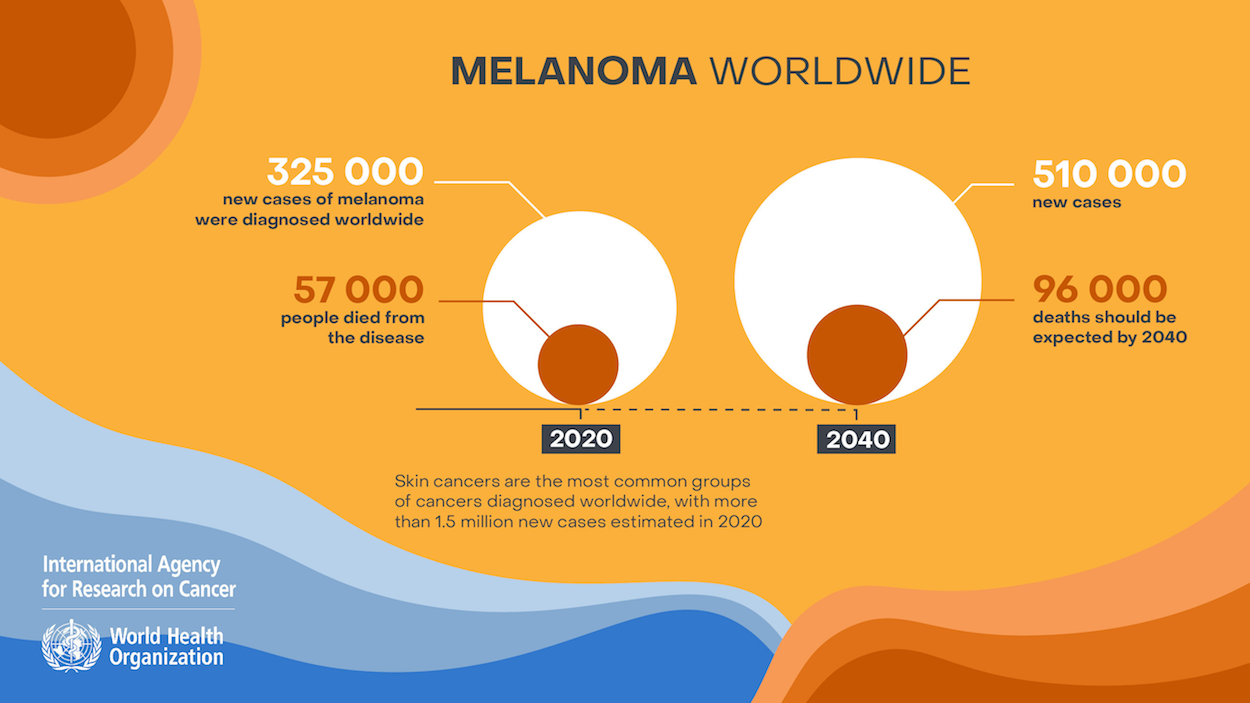
**Basal Cell Carcinoma (BCC)**  
The most common skin cancer, arising from basal keratinocytes, presenting as pearly, translucent nodules with telangiectasia, often on sun-exposed areas. It grows slowly and rarely metastasizes.

**Mycosis Fungoides**  
The most common cutaneous T-cell lymphoma, presenting with patches, plaques, and tumors on the skin, often mimicking eczema or psoriasis.

**EPIDEMIOLOGY**

A new study by scientists from the International Agency for Research on Cancer (IARC) and partners predicts that the number of new cases of cutaneous melanoma per year will increase by more than 50% from 2020 to 2040.

In 2020, an estimated 325 000 new cases of melanoma were diagnosed worldwide and 57 000 people died from the disease. There are large geographical variations in incidence rates across countries and world regions.



REFERENCE

[Treatment of Melanoma by Stage | American Cancer Society](https://www.cancer.org/cancer/types/melanoma-skin-cancer/treating/by-stage.html)

[Melanoma: Symptoms, Staging & Treatment](https://my.clevelandclinic.org/health/diseases/14391-melanoma)

[Global burden of cutaneous melanoma in 2020 and projections to 2040 – IARC](https://www.iarc.who.int/infographics/global-burden-of-cutaneous-melanoma-in-2020-and-projections-to-2040/)

**Ringworm**   
 tinea, athlete-s-foot, circular herpes, dermatophytosis, serpigo, roundworm.

**Definition and description**  
Ringworm of the body (tinea corporis) is a rash caused by a fungal infection. It's usually an itchy, circular rash with clearer skin in the middle. Ringworm gets its name because of its appearance. No worm is involved.

Mild ringworm often responds to antifungal medications applied to the skin. For more-severe infections, you may need to take antifungal pills for several weeks.

### Fungi thrive in warm and humid areas such as locker rooms and public showers. This common and contagious skin infection gets its name from the red, itchy, ring-shaped skin plaque (a type of scaly rash). It spreads easily and through close contact.

You get ringworm from contact with an infected person, animal or object. Ringworm goes by different names depending on which body part it affects. Ringworm on your body is called tinea corporis. This type of ringworm affects your arms, legs, torso and face. Ringworm is treated with antifungal medication available either over the counter or as a prescription.

#### **Types of ringworm**

Ringworm has different names based on where it appears on your body — and it can appear just about anywhere. Ringworm infections include:

* **Athlete’s foot**: Also called tinea pedis, this fungal infection causes an itchy, burning skin rash between your toes and on the soles of your feet. Your skin may become scaly and cracked or develop blisters. Sometimes, your feet smell bad.
* **Jock itch**: Tinea cruris, or jock itch, causes a red, itchy rash in your groin, upper thighs or rectum. Some people get blisters.
* **Scalp ringworm** (**tinea capitis)**: This causes scaly, red, itchy bald spots on your scalp. If left untreated, the bald spots can grow bigger and become permanent.
* **Hands (tinea manuum)**: Signs of ringworm on your hands include dry, cracked palms and ring-like patches.
* **Beard (tinea barbae)**: Ringworm appears on your neck, chin and cheeks. The patches might become crusted over or filled with pus.
* **Toenails or fingernails (tinea unguium or onychomycosis)**: Nails become thick, discolored and deformed.

### **What does ringworm look like?**

Ringworm typically begins as a flat, discolored patch, which may appear red in lighter complexions and brown in darker complexions. The patch has a ring-like or circular shape with a raised, scaly border.

Ringworm affects people of all ages. You’re more at risk for ringworm if you:

* Have a weakened immune system or an autoimmune disease like lupus.
* Participate in high-contact sports, such as wrestling (this ringworm is called tinea gladiatorum).
* Sweat excessively (hyperhidrosis).
* Use public locker rooms or public showers.
* Work closely with animals that might have ringworm.

Ringworm is contagious and extremely common. It can affect 20% to 25% of the world’s population at any given time.

**Causes**

Ringworm is a contagious fungal infection caused by common mold-like parasites that live on the cells in the outer layer of your skin. It can be spread in the following ways:

* **Human to human.** Ringworm often spreads by direct, skin-to-skin contact with an infected person.
* **Animal to human.** You can contract ringworm by touching an animal with a ringworm. Ringworm can spread while petting or grooming dogs or cats. It's also fairly common in cows.
* **Object to humans.** It's possible for ringworm to spread by contact with objects or surfaces that an infected person or animal has recently touched or rubbed against, such as clothing, towels, bedding and linens, combs, and brushes.
* **Soil to humans.** In rare cases, ringworm can be spread to humans by contact with infected soil. Infection would most likely occur only from prolonged contact with highly infected soil.

## **Risk factors**

You're at higher risk of ringworm of the body if you:

* Live in a warm climate
* Have close contact with an infected person or animal
* Share clothing, bedding or towels with someone who has a fungal infection
* Participate in sports that feature skin-to-skin contact, such as wrestling
* Wear tight or restrictive clothing
* Have a weak immune system

**Symptoms**

Signs and symptoms of ringworm may include:

* A scaly ring-shaped area, typically on the buttocks, trunk, arms and legs
* Itchiness
* A clear or scaly area inside the ring, perhaps with a scattering of bumps whose color ranges from red on white skin to reddish, purplish, brown or gray on black and brown skin
* Slightly raised, expanding rings
* A round, flat patch of itchy skin
* Overlapping rings

**Diagnosis**

Your doctor might be able to diagnose ringworm simply by looking at it. Your doctor may take skin scrapings from the affected area so that they can be examined under a microscope.

## **Treatment**

If over-the-counter treatments don't work, you may need prescription-strength antifungal medications — such as a lotion, cream or ointment that you apply to the affected skin. If your infection is particularly severe or extensive, your doctor might prescribe antifungal pills.

## **Self-care**

For a mild case of ringworm, try these self-care tips.

* Keep the affected area clean and dry.
* Apply an over-the-counter antifungal lotion, cream or ointment such as clotrimazole (Lotrimin AF) or terbinafine (Lamisil AT) as directed on the packaging.

## **Home remedies**

### Soapy water

## To prevent ringworm from spreading or infecting other areas of the body, it is advisable to keep the skin as clean as possible. To do this, a person can rinse the infection with soap and warm water once or twice daily. Be sure to dry the skin fully, as fungus thrives in moist areas.

## Always clean the skin in this way before using any of the other home remedies listed below. Before using any of the following substances on the ringworm patches, a person should apply a small amount to a healthy area of skin to ensure they do not have a sensitivity or allergy to the treatment.

### 2. Garlic

## Garlic is often a natural option that people may use to treat infections.

## Although there are no studies specifically examining the effects of garlic on ringworm, it may possess antifungal properties. For example, a 2023 study notes that garlic can be effective against Candida albicans, which is the fungus responsible for yeast infections.

## To use garlic as a treatment, a person can make a paste of crushed garlic cloves by blending the garlic with some olive or coconut oil. Apply a thin layer of paste to the affected skin and cover with gauze. Leave in place for up to 2 hours before rinsing. Repeat twice daily until symptoms resolve.

## If the garlic paste causes stinging, swelling, or redness, rinse off immediately and do not reapply.

### 3. Apple cider vinegar

## Apple cider vinegar may possess some antifungal properties, which could help it to treat ringworm when a person applies it topically to the skin.

## To treat ringworm with apple cider vinegar, a person may consider soaking a cotton wool pad in the undiluted vinegar and wipe it on the affected area. Repeat up to 3 times daily.

### 4. Aloe vera

## Aloe vera contains antiseptic agents that may exhibit antifungal, antibacterial, and antiviral activities. A 2024 study notes that applying a gel extract containing aloe vera was effective against C. albicans. As such, the antifungal properties of aloe vera may also apply to ringworm.

## A person can apply the gel from an aloe vera plant onto the ringworm patch three or four times daily. The gel also has cooling properties, so it may soothe itchy and swollen skin.

### 5. Coconut oil

## Certain fatty acids found in coconut oil may possess antifungal properties. Some research suggests that coconut oil may be able to inhibit fungi comparably to antifungal medication. However, more research is still necessary.

## People may be able to use it to treat ringworm by applying liquid coconut oil to the skin three times per day. Also, people can use coconut oil as a moisturizing lotion, which may be an effective way to prevent future ringworm infections.

### 6. Grapefruit seed extract

## Some evidence suggests that grapefruit seed extract may also possess antifungal activity. To help treat ringworm, people can mix 1 drop of grapefruit seed extract with a tablespoon of water and apply it to the skin twice daily.

### 7. Turmeric

## Turmeric is a popular spice that may possess many possible health benefits. Health experts believe that a part of turmeric known as curcumin is likely responsible for these benefits. A 2019 study suggests that turmeric may possess potent antifungal action.

## A person can consume turmeric as a tea or add it to meals to reap its benefits. For topical applications, they can mix it with a small amount of water or coconut oil until it forms a paste and apply this to the skin. Leave it to dry before wiping off. However, be aware that turmeric may stain lighter skin a yellow color, but this will fade within a few days.

### 8. Tea tree oil

## Tea tree oil is a popular natural remedy that people may apply to their skin for many different reasons. Evidence notes that tea tree oil may possess antimicrobial properties. A 2021 study suggests that an antifungal gel containing tea tree oil may be an effective option for treating fungal skin infections.

## People can dilute tea tree oil by mixing 12 drops of the essential oil with 1 ounce of a cold-pressed carrier oil, such as coconut oil. Then can then apply this to the skin three times daily.

### 9. Oregano oil

## Oregano oil derives from wild oregano (Origanum vulgare) and contains compounds that possess antifungal properties.

## As oregano oil is an essential oil, it is advisable to dilute oregano oil with a carrier oil before use. A person can then apply the oil to the affected area up to three times daily.

### 10. Lemongrass oil

## Lemongrass essential oil may also possess some fungicidal properties. To use lemongrass oil for ringworm, a person can try mixing it with a carrier oil, and applying it to the skin twice daily with a cotton ball.

## **Preparing for your appointment**

Your family doctor or a skin specialist (dermatologist) can diagnose ringworm of the body. Here are some tips to help you get ready for your appointment.

### **What you can do**

Your time with your doctor is limited, so preparing a list of questions helps you make the most of your appointment. List your questions from most important to least important in case time runs out. For ringworm, some basic questions to ask your doctor include:

* What might be causing the signs and symptoms?
* Are tests needed to confirm the diagnosis?
* What is the best treatment?
* Is this condition temporary or chronic?
* Is there a generic alternative to the medicine you're prescribing?
* Can I wait to see if the condition goes away on its own?
* What can I do to prevent the infection from spreading?
* What skin care routines do you recommend while the condition heals?

### **What to expect from your doctor**

Your doctor is likely to ask you a number of questions, such as:

* When did you first notice your symptoms?
* What did the rash look like when it first started?
* Have you had this type of rash in the past?
* Does a pet or family member already have a ringworm?
* Is the rash painful or itchy?
* Have you used any medications on it already? If so, what?

**Complications**

A fungal infection rarely spreads below the surface of the skin to cause serious illness. But people with weak immune systems, such as those with human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), may find it difficult to get rid of the infection.

## **Prevention**

Ringworm is difficult to prevent. The fungus that causes it is common, and the condition is contagious even before symptoms appear. Take these steps to reduce your risk of ringworm:

* **Educate yourself and others.** Be aware of the risk of ringworm from infected people or pets. Tell your children about ringworm, what to watch for and how to avoid infection.
* **Keep clean.** Wash your hands often. Keep shared areas clean, especially in schools, childcare centers, gyms and locker rooms. If you participate in contact sports, shower right after practice or a match and keep your uniform and gear clean.
* **Stay cool and dry.** Don't wear thick clothing for long periods of time in warm, humid weather. Avoid excessive sweating.
* **Avoid infected animals.** The infection often looks like a patch of skin where fur is missing. If you have pets or other animals, ask your veterinarian to check them for ringworm.
* **Don't share personal items.** Don't let others use your clothing, towels, hairbrushes, sports gear or other personal items. And don't borrow such things.

**Outlook / Prognosis**

### **Can ringworm come back?**

Yes, ringworms can come back. Ringworm will go away if you treat it appropriately. Follow your healthcare provider’s treatment plan until the infection clears completely. If you stop treatment or treatment ends too soon, the infection can come back.

**Living With**

### **When should I call the doctor?**

Call your healthcare provider if the ringworm infection:

* Appears on your scalp.
* Looks infected (redness and swelling).
* Occurs during pregnancy.
* Spreads to other areas of your body.
* Doesn’t improve after using over-the-counter antifungal medication as directed.
* Should I look out for signs of complications?

**Differential diagnosis**

The differential diagnosis can include:

Discoid eczema

Psoriasis

Pityriasis rosea herald patch

Seborrhoeic dermatitis.

**Discoid Eczema (Nummular Eczema)**  
A chronic inflammatory skin condition characterized by well-defined, coin-shaped (discoid) patches of eczema, typically 1–10 cm in diameter. **Psoriasis**  
A chronic autoimmune skin disease characterized by well-demarcated, erythematous plaques with silvery-white scales.

**Pityriasis Rosea (Herald Patch)**  
An acute, self-limiting skin eruption that begins with a single, large, round or oval pink patch called the herald patch, usually on the trunk. Within days to weeks, a widespread secondary rash of smaller oval patches appears, often following skin cleavage lines in a “Christmas tree” pattern. Lesions have fine scaling and mild itch. The cause is thought to be viral, possibly human herpesvirus 6 or 7. The condition resolves spontaneously within 6–8 weeks.

**Seborrhoeic Dermatitis**  
A chronic inflammatory skin disorder affecting sebaceous gland-rich areas such as the scalp, face (especially nasolabial folds and eyebrows), and upper chest. It presents with erythema, greasy yellowish scales, and flaking. It is associated with Malassezia yeast proliferation and may worsen with stress or cold weather. Symptoms include itching and dandruff. Treatment involves antifungal agents, topical corticosteroids, and keratolytics.

**Recent guidelines or updates**   
Healthcare providers are familiar with common ringworm ("tinea" and "dermatophytosis"), typically mild rashes treatable with antifungal medication.

Over the past decade, increasing reports of severe atypical cases of ringworm have been reported worldwide. One new strain is primarily sexually transmitted. Some emerging strains cause antimicrobial-resistant infections.

Three emerging strains that have been reported in the United States are:

* *Trichophyton indotineae (T. indotineae)*
* *Trichophyton mentagrophytes* genotype type VII (TMVII)
* Terbinafine-resistant *Trichophyton rubrum (T. rubrum)*

## **Antimicrobial resistance**

## Not all emerging types of ringworm are antimicrobial-resistant. However, it is important to monitor patient response to treatment. Consider antimicrobial susceptibility testing when symptoms do not improve with treatment. Two antimicrobial-resistant strains of public health concern in the United States are *T. indotineae* and terbinafine-resistant *T. Rubrum.*

## The true number of antimicrobial-resistant ringworm cases is difficult to estimate. Antifungal susceptibility testing for dermatophytes is not widely available and the United States does not require reporting.

## Several factors may be contributing to the emergence and spread of antimicrobial-resistant ringworm:

* Use of topical antifungal-corticosteroid combination products.
* Inappropriate prescription of antifungal drugs.
* Misuse of over-the-counter topical antifungal drugs.
* Inadequate adherence to prescribed courses of antifungal medication.

**Emerging strains**

### *Trichophyton indotineae (T. indotineae)*

*T. indotineae*, previously considered a subtype of *Trichophyton mentagrophytes* is now considered its own species. *T. indotineae* often have genetic mutations that make it resistant to antifungal drugs, including terbinafine, a first-line treatment.

Ringworm caused by *T. indotineae*  is often severe (covering large regions of the body) and difficult to treat. Lesions affecting the genitals are uncommon but have been reported.

In South Asia, cases of antimicrobial-resistant *T. indotineae*  infections became widespread. Although less common, cases are increasingly being reported outside of the Indian subcontinent, including in Europe, North America, South America, and Africa.

Diagnosing *T. indotineae*infection requires advanced molecular techniques such as genomic sequencing. Most clinical laboratories cannot distinguish *T. indotineae* from *T. mentagrophytes* or *T. interdigitale*, other types of dermatophytes that cause ringworm.

### ***Trichophyton mentagrophytes* genotype VII (TMVII)**

TMVII is an emerging dermatophyte fungus that may be spread during sex. It can cause inflamed, painful, itchy, and persistent skin lesions, located on the genitals, buttocks, or face.

TMVII has been circulating in Europe for several years, mostly reported among men who have sex with men. Some cases have also occurred among people who traveled to Southeast Asia for sex tourism. TMVII can also spread through clothing, towels, and bedding that have not been disinfected after use by someone with TMVII.

In June 2024, TMVII infection was documented in a man in New York City following travel to several European countries and California. TMVII cases do not appear to be widespread in the United States. Public health officials have identified several additional in New York City who have been affected.

Diagnosing TMVII infection requires advanced molecular techniques such as genomic sequencing. Most clinical laboratories cannot distinguish TMVII from *T. mentagrophytes* or*T. interdigitale*, two other types of dermatophytes.

Current evidence suggests that oral terbinafine is effective for TMVII infections, but some patients may require itraconazole. Patients may require weeks to months of antifungal therapy.

### **Terbinafine-resistant *Trichophyton rubrum (T. rubrum)***

*Trichophyton rubrum (T. rubrum)* is the most common cause of fungal nail infections (onychomycosis) and ringworm worldwide. Cases of *T. rubrum* that are resistant to the terbinafine, the first-line treatment, are increasingly being reported.

## **Epidemiology**

## Tinea corporis is the most common dermatophytosis. While tinea corporis occurs worldwide, it is most observed in tropical regions. The lifetime risk of acquiring tinea corporis is estimated to be 10–20%. Tinea corporis occurs most frequently in post-pubertal children and young adults. Rare cases have been reported in the newborn period. There is no sex predominance. Humans may become infected through close contact with an infected individual, an infected animal (in particular, domestic dog or cat), contaminated fomites, or contaminated soil. Infection may be acquired as a result of spread from another site of dermatophyte infection (e.g. tinea capitis, tinea pedis, onychomycosis). Transmission among household family members is by far the most common route; children often become infected by spores shed by an infected household family member. Autoinfection by dermatophytes elsewhere in the body may also occur. Transmission of the fungus is facilitated by a moist, warm environment, sharing of towels and clothing, and wearing of occlusive clothing. Predisposing factors include personal history of dermatophytosis (e.g. tinea capitis, tinea pedis, tinea cruris, and tinea unguium), concurrent affected family members, pets in the home, crowding in home, recreational exposure (e.g. wrestling and martial arts), hyperhidrosis, low β-defensin 4 levels, immunodeficiency, diabetes mellitus, genetic predisposition (in particular, tinea imbricata), xerosis, and ichthyosis.

**References**

[Tinea corporis: an updated review - PMC](https://pmc.ncbi.nlm.nih.gov/articles/PMC7375854/#sec8)

[Ringworm (body) - Symptoms & causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/ringworm-body/symptoms-causes/syc-20353780)[Ringworm (body) - Diagnosis & treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/ringworm-body/diagnosis-treatment/drc-20353786)

[Ringworm (Tinea Corporis): What It Looks Like, Causes & Treatment](https://my.clevelandclinic.org/health/diseases/4560-ringworm)

[Tinea corporis (Body Ringworm) — DermNet](https://dermnetnz.org/topics/tinea-corporis)

[Information for Healthcare Providers: Emerging Ringworm | Ringworm | CDC](https://www.cdc.gov/ringworm/hcp/clinician-brief-resistant-infections/index.html)

[Home remedies for ringworm: 11 natural treatments](https://www.medicalnewstoday.com/articles/320911#natural-home-remedies)

**Shingles**

Shingles, also called herpes zoster or zona

**DEFINITION AND DESCRIPTION**

Shingles is a viral infection that causes a painful rash. Shingles can occur anywhere on your body. It typically looks like a single stripe of blisters that wraps around the left side or the right side of your torso.

Shingles is caused by the varicella-zoster virus — the same virus that causes chickenpox. After you've had chickenpox, the virus stays in your body for the rest of your life. Years later, the virus may reactivate as shingles.

Shingles aren't life-threatening. But it can be very painful. Vaccines can help lower the risk of shingles. Early treatment may shorten a shingles infection and lessen the chance of complications. The most common complication is postherpetic neuralgia. This is a painful condition that causes shingles pain for a long time after your blisters have cleared.

## **Causes**

Shingles is caused by the varicella-zoster virus — the same virus that causes chickenpox. Anyone who's had chickenpox may develop shingles. After you recover from chickenpox, the virus enters your nervous system and stays inactive for years.

Sometimes the virus reactivates and travels along nerve pathways to your skin — producing shingles. But not everyone who's had chickenpox will develop shingles.

The reason for shingles is unclear. It may be due to lowered immunity to infections as people get older. Shingles is more common in older adults and in people who have weakened immune systems.

Varicella-zoster is part of a group of viruses called herpes viruses. This is the same group that includes the viruses that cause cold sores and genital herpes. As a result, shingles is also known as herpes zoster. But the virus that causes chickenpox and shingles isn't the same virus that causes cold sores or genital herpes, which is a sexually transmitted infection.

### **Shingles affects the nerves**

The shingles rash is associated with an inflammation of nerves beneath the skin.

### **Are you contagious?**

A person with shingles can pass the varicella-zoster virus to anyone who isn't immune to chickenpox. This usually occurs through direct contact with the open sores of the shingles rash. Once infected, though, the person will develop chickenpox rather than shingles.

Chickenpox can be dangerous for some people. Until your shingles blisters scab over, you are contagious. Avoid physical contact with anyone who hasn't yet had chickenpox or the chickenpox vaccine. That includes people with weakened immune systems, pregnant women and newborns.

## **Risk factors**

Anyone who has ever had chickenpox can develop shingles. Most adults in the United States had chickenpox when they were children. That was before the availability of the routine childhood vaccination that now protects against chickenpox.

Factors that may increase your risk of developing shingles include:

* **Age.** The risk of developing shingles increases with age. Shingles typically occurs in people older than 50. And people over the age of 60 are more likely to experience more-severe complications.
* **Some diseases.** Diseases that weaken your immune system, such as HIV/AIDS and cancer, can increase your risk of shingles.
* **Cancer treatments.** Radiation or chemotherapy can lower your resistance to diseases and may trigger shingles.
* **Some medications.** Drugs that prevent rejection of transplanted organs can increase your risk of shingles. Long-term use of steroids, such as prednisone, may also increase your risk of developing shingles.

## **Diagnosis**

Health care providers usually diagnose shingles based on the history of pain on one side of your body, along with the telltale rash and blisters. Your health care provider may also take a tissue sample or culture of the blisters to send to the lab.

## **Treatment**

There's no cure for shingles. Early treatment with prescription antiviral drugs may speed healing and lower your risk of complications. These drugs include:

* Acyclovir (Zovirax)
* Famciclovir
* Valacyclovir (Valtrex)

Shingles can cause severe pain, so your health care provider also may prescribe:

* Capsaicin topical patch (Qutenza)
* Anticonvulsants, such as gabapentin (Neurontin, Gralise, Horizant)
* Tricyclic antidepressants, such as amitriptyline
* Numbing agents, such as lidocaine, in the form of a cream, gel, spray or skin patch
* An injection including corticosteroids and local anesthetics

Talk with your health care provider or pharmacist about benefits and potential side effects of any drugs you're prescribed.

Shingles generally last between 2 and 6 weeks. Most people get shingles only once. But it's possible to get it two or more times.

## **Alternative treatment**

## **Cold compresses**

Holding a cool cloth or compress against the rash site may assist in relieving itchiness and reducing inflammation.

People can lightly soak a natural cotton cloth or towel with cool water and wring it out before placing it on sore, itchy areas. They can then repeat this as necessary.

It is also best not to expose the skin to extreme temperatures, so people should avoid taking ice baths or using very hot water. Hot water will increase blood flow and potentially slow down the healing of sores, whereas ice will increase skin sensitivity.

Some essential oils contain properties that may help with skin irritation and healing. These oils include:

* Chamomile oil: This has anti-inflammatory and antimicrobial
* Eucalyptus oil: This has anti-inflammatory properties and can speed up the healing of sores (for example, in people with cancer). Learn more about eucalyptus.
* Tea tree oil: This has anti-inflammatory and antimicrobial properties and can promote wound healing. Learn more about tea tree oil.

In some cases, pure essential oils can cause allergic reactions. People should always do a patch test before trying them.

It is best to dilute essential oils with a carrier oil, such as almond or jojoba, or visit a pharmacy to purchase them pre-mixed as a safe topical ointment.

## **Gentiana scabra**

Gentiana scabra, a blue or purple flower occurring throughout North America, has a positive effect on pain relief in shingles and decreases the likelihood of postherpetic neuralgia, a potential complication of shingles.

By reducing inflammation in the skin, Gentiana scabra minimizes pain and promotes healing. A reputable Chinese medicine practitioner can prepare the herbal formula by boiling the plant in water. People can then take the remedy orally.

## **Diet**

A healthy, balanced diet is important for preventing and fighting illness.

The Dietary Guidelines for Americans 2020–2025

recommend eating a varied diet, including:

* vegetables
* fruits
* whole grains
* legumes
* nuts
* lean meats

People should aim to include orange, red, and green foods that contain

the carotenoids lycopene, lutein, zeaxanthin, and provitamin A in their diet. Carotenoids are very important for immune function, and occur in the following foods:

| **Type of food** | **Examples** |
| --- | --- |
| orange foods | carrots  pumpkin  apricot |
| red foods | watermelon  red pepper  grapefruit  cherries |
| green foods | kale  parsley  spinach  melon  lettuce  endive |

Limiting trans fats and saturated fats and avoiding added sugar and salt where possible can also reduce inflammation and improve immune function.

## **Vitamin supplements**

There is a linK between vitamin D and immune function. Many older people are at risk of low vitamin D levels, so it is important they get sufficient sun exposure or take supplements to protect their immunity.

Taking vitamin C, zinc, and selenium supplements can also improve immunity in older adults.

However, taking high doses of vitamins and minerals can have harmful effects. Multivitamins, which contain lower and safer levels of many vitamins and minerals, can be a suitable option. Individuals should discuss supplements with their healthcare professional before using them.

## **Avoid smoking**

Smoking lowers immunity

against infection, especially in older adults. It can also delay recovery and healing. Plus, smoking causes various other negative effects on the body and can increase a person’s risk of harmful conditions and cancer.

If necessary, quitting or avoiding smoking can help a person manage shingles symptoms and recover more easily.

## **Reduce stress**

Stress can lower a person’s immune system. This can make them more susceptible to conditions such as shingles.

Managing stress levels can help boost immunity and protect against various other negative effects that stress can have on the body. A few stress-reducing strategies include:

* getting an adequate amount of sleep
* getting regular exercise or physical activity
* maintaining a good support network

## **Self-care**

Taking a cool bath or using cool, wet compresses on your blisters may help relieve the itching and pain. And, if possible, try to lower the amount of stress in your life.

## **Preparing for your appointment**

You may start by seeing your primary care health care provider.

Here's some information to help you get ready for your appointment.

### **What you can do**

When you make the appointment, ask if there's anything you need to do in advance, such as fasting before having a specific test. Make a list of:

* **Your symptoms,** including any that seem unrelated to the reason for your appointment
* **Key personal information,** including major stresses, recent life changes and family medical history
* **All medications, vitamins or supplements** you take, including the doses
* **Questions to ask** your doctor

Take a family member or friend along, if possible, to help you remember the information you're given.

For shingles, some basic questions to ask your doctor include:

* What's likely causing my symptoms?
* Other than the most likely cause, what are other possible causes for my symptoms?
* What tests do I need?
* Is my condition likely temporary or chronic?
* What's the best course of action?
* What are the alternatives to the primary approach you're suggesting?
* I have these other health conditions. How can I best manage them together?
* Are there restrictions I need to follow?
* Should I see a specialist?
* Are there brochures or other printed material that I can have? What websites do you recommend?

Don't hesitate to ask other questions.

### **What to expect from your doctor**

Your health care provider is likely to ask you several questions, such as:

* When did your symptoms begin?
* Have your symptoms been continuous or occasional?
* How severe are your symptoms?
* What, if anything, seems to improve your symptoms?
* What, if anything, appears to worsen your symptoms?
* Do you know if you've ever had chickenpox?

### **What you can do in the meantime**

Avoid doing anything that seems to worsen your symptoms.

## **Symptoms**

Shingles symptoms usually affect only a small section on one side of your body. These symptoms may include:

* Pain, burning or tingling
* Sensitivity to touch
* A red rash that begins a few days after the pain
* Fluid-filled blisters that break open and crust over
* Itching

Some people also experience:

* Fever
* Headache
* Sensitivity to light
* Fatigue

Pain is usually the first symptom of shingles. For some people, the pain can be intense. Depending on the location of the pain, it can sometimes be mistaken for problems with the heart, lungs or kidneys. Some people experience shingles pain without ever developing the rash.

Most commonly, the shingles rash develops as a stripe of blisters that wraps around either the left or right side of the torso. Sometimes the shingles rash occurs around one eye or on one side of the neck or face.

## **When to see a doctor**

Contact your health care provider as soon as possible if you suspect shingles, especially in the following situations:

* The pain and rash occur near the eye. If left untreated, this infection may lead to permanent eye damage.
* You're 50 or older. Age increases your risk of complications.
* You or someone in your family has a weakened immune system. This may be due to cancer, medications or chronic illness.
* The rash is widespread and painful.

## **Complications**

Complications from shingles can include:

* **Postherpetic neuralgia.** For some people, shingles pain continues long after the blisters have cleared. This condition is known as postherpetic neuralgia. It occurs when damaged nerve fibers send confused and exaggerated messages of pain from your skin to your brain.
* **Vision loss.** Shingles in or around an eye (ophthalmic shingles) can cause painful eye infections that may result in vision loss.
* **Neurological problems.** Shingles may cause inflammation of the brain (encephalitis), facial paralysis, or problems with hearing or balance.
* **Skin infections.** If shingles blisters aren't properly treated, bacterial skin infections may develop.

## **Prevention**

A shingles vaccine may help prevent shingles. People who are eligible should get the Shingrix vaccine, which has been available in the United States since its approval by the Food and Drug Administration in 2017. The Zostavax vaccine is no longer available in the U.S., but other countries may still use it.

Shingrix is approved and recommended for people age 50 and older, whether they've had shingles or not. People who've had the Zostavax vaccine in the past or don't know whether they've had chickenpox may also receive the Shingrix vaccine.

Shingrix is also recommended for people who are 19 years of age and older who have weakened immune systems due to disease or medication.

Shingrix is a nonliving vaccine made of a virus component. It's given in two doses, with 2 to 6 months between doses. The most common side effects of the shingles vaccine are redness, pain and swelling at the injection site. Some people also experience fatigue, headache and other side effects.

The shingles vaccine doesn't guarantee that you won't get shingles. But this vaccine will likely reduce the course and severity of the disease. And it will likely lower your risk of postherpetic neuralgia. Studies suggest that Shingrix offers protection against shingles for more than five years.

Talk to your health care provider about your vaccination options if you:

* Have had an allergic reaction to any component of the shingles vaccine
* Have a weakened immune system due to a condition or medication
* Have had a stem cell transplant
* Are pregnant or trying to become pregnant

The shingles vaccine is used only to prevent shingles. It's not intended to treat people who currently have the disease.

## **Outlook / Prognosis**

It can take three to five weeks from the time your first symptoms start until the rash totally disappears. Your other symptoms might start getting better in seven to 10 days.

## **Differential diagnoses**

The **differential diagnoses** for shingles include:

* Contact dermatitis
* Herpes simplex
* Impetigo
* Dermatitis herpetiformis
* Autoimmune blistering disease
* Candidiasis
* Insect bites
* Primary HIV infection

As pain typically precedes rash, shingles may initially **mimic** other disorders depending on the affected dermatome. For example, pain within the right T7 and T10 dermatomes may mimic cholecystitis or appendicitis, respectively.

**Contact Dermatitis**  
An itchy, red rash caused by direct skin contact with an irritant or allergen. There are two main types:

* *Irritant contact dermatitis* results from damage to the skin by substances like soaps, detergents, bleach, or frequent hand washing. Symptoms appear quickly and include dry, scaly, itchy, or burning skin, sometimes with blisters.
* *Allergic contact dermatitis* is an immune-mediated reaction occurring after sensitization to allergens such as nickel, latex, fragrances, cosmetics, or poison ivy. Symptoms usually develop 1–3 days after exposure and may include redness, swelling, blistering, and itching.  
  Treatment involves identifying and avoiding triggers, using moisturizers, topical corticosteroids, and antihistamines for itching. Severe cases may require prescription steroids. Prevention includes protective clothing and barrier creams.

**Herpes Simplex**  
A viral infection caused by herpes simplex virus (HSV), commonly HSV-1 or HSV-2. It presents with grouped vesicles on an erythematous base, often around the mouth (cold sores) or genital area. Lesions are painful and may be preceded by tingling or burning. The infection can recur and is contagious. Treatment includes antiviral medications.

**Impetigo**  
A contagious superficial bacterial skin infection mainly caused by *Staphylococcus aureus* or *Streptococcus pyogenes*. It presents as honey-colored crusted erosions or pustules, typically on the face and extremities. It is common in children and spreads by direct contact. Treatment includes topical or systemic antibiotics.

**Dermatitis Herpetiformis**  
A chronic, intensely itchy, blistering skin condition associated with gluten sensitivity (celiac disease). It presents with grouped vesicles and papules, mainly on extensor surfaces like elbows, knees, and buttocks.

**Autoimmune Blistering Disease**  
A group of disorders characterized by autoantibodies against skin components leading to blister formation. Examples include pemphigus vulgaris and bullous pemphigoid. They present with tense or flaccid blisters and erosions on skin and mucous membranes. Diagnosis requires immunopathology, and treatment involves systemic immunosuppression.

**Candidiasis**  
A fungal infection caused by *Candida* species, commonly *Candida albicans*. It affects moist skin areas, mucous membranes, and nails, presenting as red, itchy, sometimes white-coated patches. In skin folds, it causes maceration and satellite pustules. Treatment includes topical or systemic antifungals.

**Insect Bites**  
Skin reactions to saliva or venom from insects causing localized erythema, swelling, itching, and sometimes blistering. Reactions range from mild irritation to severe allergic responses. Secondary infection may occur from scratching. Treatment includes antihistamines, corticosteroids, and wound care.

**Primary HIV Infection**  
The initial phase of HIV infection often presents with a nonspecific viral illness including fever, rash, lymphadenopathy, sore throat, and mucocutaneous ulcers. The rash is typically maculopapular and generalized. Early diagnosis and antiretroviral therapy are crucial.

**EPIDEMIOLOGY**

About 1 in 3 people in the United States will develop shingles in their life. Your risk of having shingles increases as you get older or if you have a weakened immune system.

Most people who have shingles only have it one time. However, you can have shingles more than once.

The most common complication of shingles is severe pain in the area where the shingles rash occurred. This is known as postherpetic neuralgia, or PHN. Approximately 10% to 18% of people with herpes zoster will get PHN. The risk of PHN also increases with age.

Approximately 1 to 4% of people with shingles go to the hospital for complications. Older adults and people with weakened or suppressed immune systems are more likely to need to go to the hospital.

About 30% of people in the hospital for shingles have a weakened or suppressed immune system.

### Deaths

Fewer than 100 people die from shingles each year. Almost all shingles deaths are in older adults or people with compromised immune systems.

**RECENT GUIDELINE**

PREVENTION AND VACCINATION

Preventing herpes zoster is the best way to avoid post-herpetic neuralgia and other complications. There are two zoster vaccines available in Australia; Zostavax and Shingrix.

WHO SHOULD BE VACCINATED WITH THE ZOSTER VACCINE?

• Zoster vaccines are registered for use in people aged 50 years and over. Shingrix (NIP listed as of 1 November 2023) is more efficacious than Zostavax (no longer listed on the NIP as of 1 November 2023 - see below) particularly in the elderly, and will likely offer longer-lasting protection against herpes zoster than Zostavax

• People aged ≥18 years who are immunocompromised or shortly expected to be immunocompromised are recommended to receive a 2-dose schedule of Shingrix.

• Household contacts (50 years of age and older) of a person who is, or who is expected to become immunocompromised

• People who have previously received Zostavax can receive Shingrix to increase their protection against herpes zoster since protection using Zostavax wanes significantly from around 5 years after vaccination

• People who have had a previous episode of herpes zoster can receive zoster vaccine at the recommended age The Shingles Prevention Study (SPS) was conducted among 38,546 adults aged ≥60 years and showed that compared to placebo, vaccination with Zostavax reduced:

• Herpes zoster (HZ) by 51.3%

• Post-herpetic neuralgia by 66.5% Burden of illness associated with HZ by 61.1% over a median of more than three years follow-up

WHO SHOULD NOT RECEIVE THE LIVE ZOSTER VACCINE?

• While pregnant

• Previous anaphylaxis to the vaccine (either Zostavax or varicella vaccine) or its components

• People who are severely immunocompromised: Primary or acquired immunodeficiency– Haematologic neoplasms: leukaemias, lymphomas myelodysplastic syndromes – Post-transplant: solid organ (on immunosuppressive therapy), haematopoietic stem cell transplant (within 24 months)– Immunocompromised due to primary or acquired (HIV/AIDS) immunodeficiency– Other significantly immunocompromising conditions Immunosuppressive therapy: current or recent– Chemotherapy, radiotherapy– High-dose corticosteroids >= 20mg prednisolone per day, or equivalent for 14 days– All biologics and most disease- modifying anti- rheumatic drugs DMARDs

BEFORE VACCINATING PEOPLE WITH ZOSTAVAX

Obtain medical history prior to vaccination with Zostavax, check contraindications of Zostavax in immunocompromised individuals. Please note: Zostavax is no longer NIP listed but is still available on a private script. In persons who are or have recently been immunocompromised, the safety of administering Zostavax should always be considered on a case-by case basis. If there is uncertainty around the level of immunocompromise and when vaccine administration may be safe, vaccination should be withheld and expert advice sought from the treating physician and/or an immunization specialist. Zostavax is not recommended for people who have already received a zoster vaccine. Vaccination of a person with Zostavax if they have previously received Shingrix should be assessed on a case-by case basis.

UPDATE: Denosumab has been removed from the list of immunosuppressive medications contraindicated with Zostavax as there is currently not enough evidence to suggest it is a contraindication to Zostavax.

SHINGRIX

From 1 November 2023, the shingles vaccine Shingrix® replaced Zostavax® on the National Immunizationrogram (NIP) schedule for the prevention of shingles and post-herpetic neuralgia. It is available for eligible people most at risk of complications from shingles. A 2-dose course of Shingrix® will be available free for:

• People aged 65 years and older

• First Nations peoples aged 50 years and older

• Immunocompromised people aged 18 years and older with medical conditions including: haemopoietic stem cell transplant, solid organ transplant, hematological malignancy, advanced or untreated HIV.

Unlike Zostavax®, Shingrix® does not contain any live virus so it can be given to people aged 18 years and over who are immunocompromised.

WHO SHOULD NOT RECEIVE SHINGRIX?

Previous anaphylaxis to the vaccine. There is currently no data on the use of Shingrix during pregnancy (Category B2). Zostavax contains live attenuated varicella-zoster virus. It is safe and well tolerated. Some people may experience a headache, fatigue or soreness around the site where the shot was given. The reaction is typically mild and resolves within a few days.

VACCINE SAFETY Shingrix causes moderately high rates of local and systemic infections. Common reactions include:

• Injection-site pain (up to 79%)

• Redness (up to 39%) and swelling (up to 26%)

• Systemic symptoms such as: – Fatigue – Myalgia (up to 46%)

• Headache (up to 39%)

• Shivering (up to 28%)

• Fever (up to 22%)

• Gastrointestinal symptoms (up to 18%

REFERENCES

[Shingles Facts and Stats | Shingles (Herpes Zoster) | CDC](https://www.cdc.gov/shingles/data-research/index.html)

<https://www.mayoclinic.org/diseases-conditions/shingles/symptoms-causes/syc-20353054>

[Shingles (Herpes Zoster): Causes, Symptoms & Treatment](https://my.clevelandclinic.org/health/diseases/11036-shingles#outlook-prognosis)

[Shingles-Guide-2024.pdf](https://www.immunisationcoalition.org.au/wp-content/uploads/2017/11/Shingles-Guide-2024.pdf)

[Natural treatments and home remedies for shingles](https://www.medicalnewstoday.com/articles/322131#avoid-smoking)

### **Hives**

**DEFINITION AND DESCRIPTION**

Urticaria, also known as hives

Hives are raised red bumps (welts) or splotches on the skin. They’re a type of swelling on the surface of your skin and happen when your body has an allergic reaction. Allergic reactions happen when your immune system comes in contact with an allergen. Allergens are proteins that are harmless to many people but cause an allergic reaction in sensitive people.

Hives are often very itchy, but you might also feel burning or stinging. They can be as small as a fingertip or as big as a dinner plate. The medical name for hives is urticaria.

Sometimes, the welts from hives join together to form larger areas called plaques. Hives tend to fade within 24 hours, although they may be noticeable for several days or longer.

#### **Types of hives**

Acute urticaria refers to hives that don’t last very long (less than six weeks). Chronic urticaria refers to hives that happen at least twice a week for more than six weeks.

Chronic, spontaneous urticaria is the name for chronic hives that don’t have an obvious cause. An older name for this condition is chronic idiopathic urticaria.

There’s also a condition called physical urticaria, or inducible urticaria. These hives might pop up when you’re in the cold, heat or sun. Some people react to vibrations or pressure, exercising or sweating. Physical hives usually appear within an hour after exposure. This type of hive can also be chronic.

### **What’s the difference between hives and a rash?**

A rash is a skin condition that involves something out of the ordinary, like spots, swelling, itchiness or redness. Hives are an example of a rash, but not all rashes are hives.

### **Who is affected by hives?**

Anyone can get hives. If you’re someone who reacts to many types of allergens, you may get hives frequently. Other people who don’t react to allergens may get hives once or a few times in their lives.

There seems to be a relationship between acute hives and conditions like asthma, allergic rhinitis and atopic dermatitis, especially in children. You might also be affected by hives during periods of extreme stress.

#### **How common are hives?**

Around 20% of the population will get hives at least one time. About 1% to 3% of the population has chronic hives.

## **Symptoms**

### **What are the symptoms of acute hives?**

Hives look different depending on the person and the situation. They can show up anywhere on your body. Signs of acute hives include:

* Raise welts or bumps on your skin. The bumps may look reddish on lighter-colored skin.
* Hives blanch (the center of the hive becomes pale when pressed).
* Itchy skin.
* Swelling under your skin causes puffiness (angioedema).
* Also appearing with painful swelling of your lips, eyes and inside your throat.

### **What are the symptoms of chronic hives?**

In many respects, chronic hives and acute hives may look alike: they can be itchy, swollen raised welts that turn lighter in the center and with pressure. However, chronic hives can:

* Shift sizes and shapes.
* Appear, disappear and then reappear at least every few days for long periods of time, even months or years.
* It Happens along with heat, exercise or stress.

### **What causes hives?**

#### **Causes of acute hives**

Acute hives are often an allergic reaction to something you put into your body, like food, drink or medication, or something that you touch. The skin has immune cells called mast cells. When these cells go into action, they release chemicals, including one called histamine. [Histamine](https://my.clevelandclinic.org/health/articles/24854-histamine) is the reason that hives form.

You can also get hives for a variety of other reasons. Some of these include having an infection, stress or physical pressure on your skin. It’s not uncommon for healthcare providers to be unable to determine exactly what caused your hives.

#### **Causes of chronic hives**

Unlike acute hives, chronic hives aren’t usually caused by allergies. They may be caused by infections from bacteria or viruses, or because of other medical conditions like lupus. Your provider may not discover an exact cause. In these cases, chronic hives are said to be idiopathic or spontaneous.

Chronic hives do last for long periods of time but usually aren’t permanent. They can be uncomfortable, but they aren’t life-threatening.

### **Are hives contagious?**

Unlike some other skin conditions, hives aren’t contagious. But if you develop hives because your skin is exposed to secretions from a plant like poison ivy, you can spread the allergenic plant product to others until you wash it off your skin.

## **Diagnosis and Tests**

Your healthcare provider can diagnose hives and angioedema by looking at your skin. Allergy tests can help identify what’s triggering a reaction, but this is true primarily for acute hives. Knowing the cause can help you avoid allergens and the hives that come with them. Allergy tests to diagnose hives include:

* **Skin tests:** During this test, healthcare providers test different allergens on your skin. If your skin turns red or swells, it means you’re allergic to that substance. This type of allergy test is also called a skin prick or scratch test. Skin testing usually isn’t done for chronic hives.
* **Blood tests:** A blood test checks for specific antibodies in your blood. Your body makes antibodies to fight off allergies. If your body makes too many antibodies, you can develop hives and swelling.

## **Management and Treatment**

### **How are hives treated or managed?**

Most of the time, hives go away without treatment. Your healthcare provider might recommend medications and at-home care to help you feel better and lower your chances of having hives again. Treatments include:

* **Allergy medications:** Medicines called antihistamines block histamine’s effects. They can be taken orally (swallow a pill) or topically (put on the affected skin). Antihistamines relieve itching from hives and make allergic reactions go away or become less severe. Some antihistamines react quickly, such as diphenhydramine (Benadryl®). Depending on how severe the hives are, your healthcare provider may recommend daily allergy medications, like loratadine (Claritin®), fexofenadine (Allegra®), cetirizine (Zyrtec®) or levocetirizine (Xyzal®).

**Allergy shots:** For hard-to-treat chronic hives, your healthcare provider may discuss monthly injections of drugs that block allergic reactions. People with severe allergies make too much IgE. These injections block your immune system from making IgE.

* **At-home treatments:** To relieve hives, you can take a cool bath or shower, wear loose-fitting clothing and apply cold compresses. An over-the-counter (OTC) hydrocortisone or antihistamine cream can relieve itching and swelling.
* **Oral steroids:** Corticosteroids, such as prednisone, can relieve hive symptoms that don’t respond to antihistamines or topical steroids.
* **Epinephrine:** Severe acute allergic reactions can lead to a life-threatening condition called anaphylaxis. Symptoms include hives, swelling of your face, mouth or throat, shortness of breath, [wheezing](https://my.clevelandclinic.org/health/symptoms/15203-wheezing), vomiting and low blood pressure. Anaphylaxis is life-threatening and anyone having this kind of reaction needs an immediate epinephrine injection (EpiPen®) to open a swollen airway.

#### **Complications of hives**

Anyone who has a severe acute allergic reaction could have life-threatening swelling of the airways — your throat and lungs. This condition is known as anaphylaxis. It can potentially close off the airways, resulting in death.

Anaphylaxis is often triggered by a severe allergic reaction to a certain food, like peanuts and tree nuts, or to a bee sting. If you have anaphylaxis, you need an immediate shot of epinephrine, such as injectable epinephrine (EpiPen® or AUVI-Q®).

Epinephrine opens airways, raises blood pressure and reduces hives and swelling. If you take epinephrine outside of a medical setting, you should go to the emergency room to be monitored. Symptoms of anaphylaxis can return as the epinephrine wears off.

## **Prevention**

#### **Acute hives**

Your healthcare provider can use the results of allergy tests to help you figure out which substances bring on acute hives. Once you know your triggers, you can avoid them. You may want to:

* Cut certain food products out of your diet.
* Reduce exposure to airborne allergens.
* Switch to detergents and soaps without scents or dyes.
* Avoid extreme changes in temperature.
* Relax and take a break when you’re stressed or overworked.
* Wear loose-fitting, lightweight clothing.

Some of these tips can also help with chronic hives.

#### **Chronic hives**

It may not be possible to prevent chronic hives. Your provider may not be able to find exactly what causes them. They may also be a part of a bigger medical condition that affects your immune system.

## **Outlook / Prognosis**

For most people, hives don’t cause serious problems. Children often outgrow allergies that cause hives.

For some people, allergic reactions like angioedema can cause anaphylaxis — severe swelling of the airways and lungs. If you have this life-threatening condition, you should carry and know when and how to use injectable epinephrine (EpiPen®).

**Living With**

### **When should I call my healthcare provider about hives?**

Hives can get better without treatment. Call your healthcare provider if you have:

* Hives or swelling that lasts more than a week.
* Infected-looking bumps (red, swollen or pus-filled).
* Recurring hives (they come back every few months).
* Severe itching that might even keep you from sleeping.
* Signs of anaphylaxis, including wheezing, shortness of breath or vomiting.
* Swollen lips or face.

### **What questions should I ask my healthcare provider?**

If you develop hives, you might want to ask your healthcare provider these questions:

* Why did I get hives?
* When should the hives go away?
* Should I get an allergy test?
* What steps can I take to prevent getting hives in the future?
* What’s the best treatment to reduce itching?
* What’s the best way to get rid of hives?
* Should I look out for signs of complications?

### **Hives Triggers**

* Some food (especially peanuts, eggs, nuts and shellfish)
* Medications, such as antibiotics (especially penicillin and sulfa), aspirin and ibuprofen
* Insect stings or bites
* Physical stimuli, such as pressure, cold, heat, exercise or sun exposure
* Latex
* Blood transfusions
* Bacterial infections, including urinary tract infections and strep throat
* Viral infections, including the common cold, infectious mononucleosis and hepatitis
* Pet dander
* Pollen
* Some plants

## **Differential Diagnoses**

* Urticarial Vasculitis
* Hereditary Angioedema
* Mastocytosis
* Erythema Multiforme
* Atopic Dermatitis
* Allergic Contact Dermatitis
* Irritant Contact Dermatitis
* Scabies
* Drug Eruptions

**Urticarial Vasculitis**  
A form of small vessel vasculitis presenting with urticaria-like (hive-like) skin lesions that last more than 24 hours, often painful or burning rather than itchy. Lesions are red patches or plaques sometimes with a white center and petechiae, which may heal with bruising or hyperpigmentation.

**Hereditary Angioedema**  
A rare genetic disorder caused by deficiency or dysfunction of C1 esterase inhibitor, leading to recurrent episodes of non-itchy, non-pitting swelling (angioedema) of the skin, mucous membranes, gastrointestinal tract, and airway. Attacks can be life-threatening if they involve the airway. It is not mediated by histamine, so antihistamines and corticosteroids are ineffective. Treatment includes C1 inhibitor replacement, bradykinin receptor antagonists, and prophylactic therapies.

**Mastocytosis**  
A disorder characterized by accumulation and activation of mast cells in the skin and/or other organs. Cutaneous mastocytosis presents with brownish macules or papules (urticaria pigmentosa) that may urticate (Darier’s sign) on rubbing. Systemic mastocytosis can cause flushing, anaphylaxis, gastrointestinal symptoms, and bone pain due to mast cell mediator release. Treatment includes antihistamines, mast cell stabilizers, and avoidance of triggers.

**Erythema Multiforme**  
An acute, immune-mediated condition characterized by target-shaped (iris) lesions, often triggered by infections (commonly herpes simplex virus) or medications. Lesions are symmetrically distributed on the extremities and may involve mucous membranes. It ranges from mild (erythema multiforme minor) to severe forms overlapping with Stevens-Johnson syndrome.

**Atopic Dermatitis**  
A chronic, relapsing inflammatory skin disease marked by intense itching and eczematous lesions. It commonly affects flexural areas and is associated with a personal or family history of atopy (asthma, allergic rhinitis). The skin is dry, inflamed, and prone to secondary infection. Management includes emollients, topical corticosteroids, and addressing triggers.

**Allergic Contact Dermatiti**s  
A delayed hypersensitivity reaction resulting from skin contact with allergens such as nickel, fragrances, or poison ivy. It presents with erythema, edema, vesicles, and intense itching confined to the area of contact. Diagnosis is clinical and confirmed by patch testing. Treatment involves allergen avoidance and topical corticosteroids.

**Irritant Contact Dermatitis**  
A non-immunologic inflammatory reaction caused by direct damage to the skin barrier by irritants like detergents, solvents, or frequent hand washing. It presents with dry, red, cracked, and sometimes painful skin, often on hands. Management includes avoiding irritants, skin barrier repair with emollients, and topical steroids if needed.

**Scabies**  
A contagious parasitic infestation caused by *Sarcoptes scabiei* mites burrowing into the skin. It presents with intense nocturnal itching and a characteristic rash of papules, vesicles, and burrows, especially in web spaces of fingers, wrists, and genital areas. Diagnosis is clinical and by identifying mites or eggs. Treatment includes topical scabicides (permethrin) and treating close contacts.

**Drug Eruptions**  
Adverse cutaneous reactions to medications ranging from mild maculopapular rashes to severe life-threatening conditions like Stevens-Johnson syndrome or toxic epidermal necrolysis. Presentation varies widely but often includes widespread erythematous rash, itching, and sometimes systemic symptoms. Management involves withdrawal of the offending drug and supportive care.

**EPIDEMIOLOGY**

The lifetime prevalence for all types of urticaria is usually described below 10% per different reports, while chronic urticaria (CU) only develops in approximately one-fourth of these individuals.

Point prevalence of CU, based on coding reports in health systems from different countries, ranges from 0.1 to less than 1% globally. Currently the point prevalence is the best method to compare the frequency of CU between different populations but the development of a standardized and practical tool for this purpose remains an unmet need.

From the total of CU patients, one-third suffer from both hives and angioedema, 30%–40% present isolated hives, and around 10% show isolated angioedema.

The natural history of the disease has a very wide range. Around half of patients will follow a three-month self-limited evolution and within a year it will resolve in almost 80% of them. However, in more than 10% of patients a duration of 5 years or longer is expected. Factors conditioning time to remission will be discussed later.

Females are affected at least twice as often as males, and most patients are over 20 years of age. In children, the prevalence varies from less than 1% to almost 5%, depending largely upon the methodology. Ethnic differences, although described as statistically significant in a wide American population sample, do not seem to deserve in-depth attention in the real world. Additionally, a proportion of patients experience exacerbations when taking non-steroidal anti-inflammatory drugs (NSAIDs).

The observation that chronic inducible urticaria (CIU) is much more frequent among first-degree relatives of affected individuals than in the general population suggests the existence of a genetic background for the disease and provides clinical support to the reported association between CIU and human leukocyte antigen DR4.

The economic burden of the pathology is not negligible. The CU related cost has been reported to be as high as $2050 per year per patient in the United States, having a huge personal and familiar impact, particularly in low to middle income countries. Economic burden analysis using purchasing power parity dollars (PPP$) demonstrated a higher therapy and inpatients costs in France with almost 3000 $, compared to less than 1000 $ in Italy; nonetheless, loss of work productivity was greater in Germany than in France with over PPP$ 1.000 and over PPP$ 500.

REFERENCE

[The challenges of chronic urticaria part 1: Epidemiology, immunopathogenesis, comorbidities, quality of life, and management - PMC](https://pmc.ncbi.nlm.nih.gov/articles/PMC8233382/)

[Hives: Causes, Symptoms, Diagnosis, Treatment & Prevention](https://my.clevelandclinic.org/health/diseases/8630-hives)

**NAIL FUNGUS**

Onychomycosis” traditionally referred to a non dermatophyte infection of the nail but is now used as a general term to denote any fungal nail infection (tinea unguium specifically describes a dermatophytic invasion of the nail plate)

**DEFINITION AND DESCRIPTION**

Nail fungus is a common infection of the nail. It begins as a white or yellow-brown spot under the tip of your fingernail or toenail. As the fungal infection goes deeper, the nail may discolor, thicken and crumble at the edge. Nail fungus can affect several nails.

If your condition is mild and not bothering you, you may not need treatment. If your nail fungus is painful and has caused thickened nails, self-care steps and medications may help. But even if treatment is successful, nail fungus often comes back.

Nail fungus is also called onychomycosis (on-ih-koh-my-KOH-sis). When fungus infects the areas between your toes and the skin of your feet, it's called athlete's foot (tinea pedis).

**Symptoms**

Symptoms of nail fungus include a nail or nails that are:

* Thickened
* Discolored
* Brittle, crumbly or ragged
* Misshapen
* Separated from the nail bed
* Smelly

Nail fungus can affect fingernails, but it's more common in toenails.

### **When to see a doctor**

You may want to see a health care provider if self-care steps haven't helped and the nail becomes increasingly discolored, thickened or misshapen. Also talk with your health care provider if you have:

* Diabetes and think you're developing nail fungus
* Bleeding around the nails
* Swelling or pain around the nails
* Difficulty walking

Nail fungus is caused by various fungal organisms (fungi). The most common is a type called dermatophyte. Yeast, bacteria and molds also can cause nail infections. The discoloration from a bacterial infection tends to be green or black.

Fungal infection of the foot (athlete's foot) can spread to the nail, and a fungal infection of the nail can spread to the foot. You can also get the infection from contact with spaces where fungi can thrive, such as the floor tile in a gym shower or inside dark, sweaty, moist shoes.

**Risk factors**

Factors that can increase your risk of developing nail fungus include:

* Older age
* Wearing shoes that make your feet sweat heavily
* Having had athlete's foot in the past
* Walking barefoot in damp public areas, such as swimming pools, gyms and shower rooms
* Having a minor skin or nail injury
* Having a skin condition that affects the nails, such as psoriasis
* Having diabetes, blood flow problems or a weakened immune system

**Complications**

A severe case of nail fungus can be painful and may cause permanent damage to your nails. And it may lead to other serious infections that spread beyond your feet if you have a suppressed immune system due to medication, diabetes or other conditions.

**Prevention**

The following habits can help prevent nail fungus or reinfections and athlete's foot, which can lead to nail fungus:

* Keep your nails clean and dry. Wash your hands and feet regularly. Wash your hands after touching an infected nail. Dry well, apply an antifungal foot powder and moisturize your nails. Consider applying a nail hardener, which might help strengthen nails and cuticles.
* Keep your nails trimmed. Cut nails straight across, smooth the edges with a file and file down thickened areas. Disinfect your nail clippers after each use. Letting your nails grow long creates more places for the fungus to grow.
* Wear absorbent socks or change your socks throughout the day.
* Choose shoes made of materials that breathe.
* Discard old shoes or treat them with disinfectants or antifungal powders.
* Wear footwear in pool areas and locker rooms.
* Choose a nail salon that uses sterilized manicure tools for each customer. Or disinfect tools you use for home pedicures.
* Give up nail polish and artificial nails.
* If you have athlete's foot, treat it with an antifungal product.

## **Diagnosis**

Your health care provider will examine your nails and perhaps take some nail clippings or scrape debris from under your nail. These samples are sent to a lab to identify the cause of your symptoms.

Other conditions, such as psoriasis, can mimic a fungal infection of the nail. Microorganisms such as yeast and bacteria also can infect nails. Knowing the cause of your infection helps determine the best treatment.

**Treatment**

Treatment for toenail fungus isn't always needed. And sometimes self-care and nonprescription products clear up the infection. Talk with your health care provider if your condition doesn't improve. Treatment depends on the severity of your condition and the type of fungus causing it. It can take months to see results. And even if your nail condition improves, repeat infections are common.

### **Medications**

Your health care provider may prescribe antifungal drugs that you take by mouth (orally) or apply to the nail.

* **Oral antifungal drugs.** These drugs are often the first choice. One option is itraconazole (Sporanox). These drugs help a new nail grow free of infection, slowly replacing the infected part.

You typically take this type of drug daily for 6 to 12 weeks. But you won't see the result of treatment until the nail grows back completely. It may take four months or longer to eliminate an infection. Treatment success rates with these drugs appear to be lower in adults over age 65.

Oral antifungal drugs may cause side effects such as rash and liver damage. Or they may interfere with other prescription drugs. You may need occasional blood tests to check on how you're doing with these types of drugs. Health care providers may not recommend oral antifungal drugs for people with liver disease or congestive heart failure or those taking certain medications.

* **Medicated nail polish.** Your health care provider may prescribe an antifungal nail polish called ciclopirox (Penlac). You paint it on your infected nails and surrounding skin once a day. After seven days, you wipe the piled-on layers clean with alcohol and begin fresh applications. You may need to use this type of nail polish daily for almost a year.
* **Medicated nail cream.** Your health care provider may prescribe an antifungal cream, such as efinaconazole (Jublia) and tavaborole (Kerydin). You rub this product into your infected nails after soaking. These creams may work better if you first thin the nails. This helps the medication get through the hard nail surface to the underlying fungus.

To thin nails, you apply a nonprescription lotion containing urea. Or your health care provider may thin the surface of the nail (debride) with a file or other tool.

Antifungal nail creams may cause side effects such as rash.

### **Surgery**

Your health care provider might suggest temporary removal of the nail so that the antifungal drug can be applied directly to the infection under the nail.

The most effective but least used option is surgery to permanently remove the nail and its root.

**Lifestyle and home remedies**

Often, you can take care of a fungal nail infection at home:

* **Try nonprescription antifungal nail creams and ointments.** Several products are available, such as terbinafine (Lamisil). If you notice white markings on the surfaces of the nails, file them off, soak your nails in water, dry them, and apply the medicated cream or lotion. Even if this clears up your symptoms, it's common for the infection to come back.
* **Trim and thin the nails.** This helps reduce pain by reducing pressure on the nails. Also, if you do this before applying an antifungal, the drug can reach deeper layers of the nail.

Before trimming or using a nail file to thin, thick nails, soften them with urea-containing creams. See a health care provider for foot care if you have a condition that causes poor blood flow to your feet.

**Alternative medicine**

Some research suggests that the nutritional supplement biotin might help strengthen weak or brittle fingernails. Talk with your health care provider if you have any questions or concerns about whether this is right for you.

Also, some research shows that tea tree oil has antifungal effects. It is often used to treat nail fungus.

**Preparing for your appointment**

You're likely to start by seeing your primary care provider. In some cases when you call to set up an appointment, you may be referred immediately to either a doctor who specializes in skin conditions (dermatologist) or one who specializes in foot conditions (podiatrist).

Here are some steps you can take to prepare for your appointment:

* **List your symptoms,** including any that may seem unrelated to nail fungus.
* **List key personal information,** including any major stresses or recent life changes.
* **List all the medications,** vitamins and supplements you're taking.
* **List questions to ask** your healthcare provider.

For nail fungus, your questions might include:

* What is likely causing my symptoms or condition?
* What are other possible causes for my symptoms or condition?
* What tests do I need?
* What is the best course of action?
* What are the alternatives to the primary approach you're suggesting?
* I have other health conditions. How can I best manage them together?
* Is a generic alternative available for the medicine you're prescribing?
* Do you have any brochures or other printed material that I can take home? Do you recommend any websites on nail fungus?

**PROGNOSIS**

With treatment, many people can get rid of nail fungus. Even when the fungus clears, your nail(s) may look unhealthy until the infected nail grows out. A fingernail grows out in 4 to 6 months and a toenail in 12 to 18 months.

To clear the fungus, it’s important to:

* Use the treatment exactly as prescribed
* Apply (or take) the medicine for as long as prescribed
* Keep all follow-up appointments with your dermatologist

Nail fungus can be stubborn. If you have a severe infection, it’s possible to clear the infection. A healthy-looking nail, however, may be unrealistic, but you can expect the nail to look better and feel more comfortable.

**Differential diagnosis of onychomycosis**

Many other nail diseases may mimic the clinical signs of onychomycosis, hence the importance of diagnostic confirmation to ensure malignant

conditions are not missed.

Benign conditions include bacterial infection such as :

* pseudomonas argeuniosa (CAP)
* psoriasis
* lichen planus
* subungual and periungual verruca
* paronychia,
* subungual exostosis,
* onychomatricoma,
* yellow nail syndrome,
* and idiopathic/traumatic onycholysis.

Malignant conditions include subungual squamous cell carcinoma and subungual melanoma.

## 

* **Bacterial Infection** (e.g., *Pseudomonas aeruginosa* causing Chronic Paronychia - CAP)  
  *Pseudomonas aeruginosa* can cause greenish discoloration and infection of the nail folds, particularly in chronic paronychia, leading to inflammation and nail changes.
* **Psoriasis**  
  Nail psoriasis manifests with pitting, onycholysis (nail lifting), subungual hyperkeratosis, and discoloration. It is a chronic inflammatory condition often associated with skin and joint disease.
* **Lichen Planus**  
  Nail involvement includes longitudinal ridging, thinning, fissuring, and sometimes permanent nail loss due to scarring.
* **Subungual and Periungual Verruca**  
  Warts caused by human papillomavirus (HPV) presenting as rough, hyperkeratotic lesions under or around the nail.
* **Paronychia**  
  Inflammation and infection of the nail folds, often bacterial or fungal, causing redness, swelling, and tenderness.
* **Subungual Exostosis**  
  A benign bony outgrowth beneath the nail bed causing nail deformity and pain.
* **Onychomatricoma**  
  A rare benign tumor of the nail matrix presenting as thickened, yellowed nails with splinter hemorrhages and longitudinal over curvature.
* **Yellow Nail Syndrome**  
  Characterized by thickened, slow-growing yellow nails, often associated with lymphedema and respiratory disease.
* **Idiopathic/Traumatic Onycholysis**  
  Detachment of the nail plate from the nail bed without infection, often due to trauma or unknown causes.

Malignant Conditions

* **Subungual Squamous Cell Carcinoma (SCC)**  
  A rare but important malignancy arising beneath the nail. It often mimics benign conditions such as infections or trauma, leading to delayed diagnosis. Clinical features include nail discoloration, deformity, ulceration, and sometimes pain. Bone invasion occurs in about 20% of cases. Diagnosis requires biopsy and imaging to assess extent. Treatment primarily involves surgical excision, ranging from wide local excision of the nail unit to amputation if bone is involved. Mohs micrographic surgery can be used but has higher recurrence rates compared to wide excision. Radiation therapy is an option for non-surgical candidates. Early diagnosis improves prognosis, as metastases are rare but possible. Long-term follow-up is essential due to recurrence risk.
* **Subungual Melanoma**  
  A malignant melanocytic tumor under the nail, presenting as a dark pigmented streak, nail dystrophy, or ulceration. It carries a poor prognosis if diagnosed late and requires prompt biopsy and surgical management.

**EPIDEMIOLOGY**

Clinical features of onychomycosis were found in 119 (68.4%) participants. Distal subungual onychomycosis (68-57.1%) was the most common clinical type, followed by total dystrophic onychomycosis (49-41.2%), candida onychomycosis (34-28.6%), proximal subungual onychomycosis (14-11.8%) and superficial white onychomycosis (9-7.6%). One hundred and one (84.9%) respondents with clinically described onychomycosis had positive results in mycology studies. The non-dermatophyte molds (Aspergillus and Penicillium spp.) were found in 130 samples (78.8%); dermatophytes in 31 (18.8%) and yeast in 7 (4.2%).

Non-dermatophyte molds, traditionally thought to be contaminants of nail cultures, were the main causative agents of primary fungal nail infections. Garri processors will benefit from public health intervention geared towards automation of some of these processes to minimize contact with soil and water, and health education on the use of protective materials.

identified a total of 44 well-documented studies in 13 (24.1%) of the 54 African countries amounting to a total of 6773 cases of nail fungal infections: 4609 (68.0%) from North Africa, 1338 (19.6%) from West Africa, 524 (7.7%) from East Africa, 243 (3.6%) from Central Africa, and 59 (0.9%) from Southern Africa, with a pooled prevalence of 19.6% (6773/34604). Identification of fungal pathogens was mainly by conventional methods: microscopy (*n* = 43, 97.7%), culture (*n* = 42, 95.5%), and periodic acid-Schiff staining (*n* = 1, 2.3%). Advanced diagnostics with improved sensitivity were also deployed in some studies: PCR (*n* = 2, 4.5%), sequencing (*n* = 2, 4.5%), and matrix-assisted laser desorption/ionization time of flight mass spectrometry (*n* = 1, 2.3%). The most frequent fungal pathogens identified in North and Central Africa were *Trichophyton rubrum* (80.4% and 49.3%), followed by *Candida albicans* (12.2% and 27.4%), respectively, while in the West and East African regions, it was *Candida albicans* (63% and 47.3%) followed by *Trichophyton rubrum* (19.1% and 18.9%), respectively. Antifungal susceptibility testing was performed in only five studies and showed varied outcomes with high resistance of dermatophytes to ketoconazole, itraconazole, and fluconazole and non-dermatophyte moulds to caspofungin and 5-flucytosine, respectively.

REFERENCES

[Nail fungus - Diagnosis and treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/nail-fungus/diagnosis-treatment/drc-20353300)

[Nail fungus - Symptoms and causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/nail-fungus/symptoms-causes/syc-20353294)

[Nail fungus: Diagnosis and treatment](https://www.aad.org/public/diseases/a-z/nail-fungus-treatment)

[Fungal Nail Infections amongst Cassava Farmers and Processors in Southwest Nigeria - PubMed](https://pubmed.ncbi.nlm.nih.gov/36453258/)

**Actinic prurigo (AP)**

**DEFINITION AND DESCRIPTION**

Actinic prurigo (AP) is a rare form of idiopathic photo dermatosis that primarily affects sun-exposed areas of the skin. The affected regions of the skin typically include the face, neck, and dorsal surface of the upper extremities. Sun-protected areas of the skin, such as the buttocks, have also been described. Actinic prurigo typically manifests in the spring as symmetric intensely pruritic papulonodular dermatitis and can persist into the winter months. In severe cases, excoriations, cheilitis, conjunctival disease, and scarring may develop. Actinic prurigo is typically described in prepubescent females but can occur at any age or gender. The disease has a strong genetic component and is more commonly seen in American Indians of North, Central, and South America. The diagnosis is mainly clinical. Disease management begins with sun protection and sunlight avoidance. Treatment involves topical antihistamines, corticosteroids, photochemotherapy (PUVA), as well as systemic therapies for severe cases. Without treatment, this disease course remains chronic and can persist into adulthood.

## **Causes of Actinic Prurigo**

The condition is often seen in those living in high-altitude regions or with indigenous ancestry. Other factors like environmental factors, such as pollen or dust, can also exacerbate symptoms. Sunscreen and protective clothing are recommended to prevent flare-ups.

Prolonged exposure to sunlight, particularly UV-A and UV-B rays, triggering an immune response in the skin.

Genetic predisposition, as individuals with a family history of actinic prurigo are at a higher risk of developing the condition.

Environmental factors such as high altitude or proximity to the equator, which can increase the intensity of UV radiation exposure.

Certain medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs) or antibiotics, may trigger or exacerbate actinic prurigo in some individuals.

Immune system dysfunction, where abnormalities in the immune response to UV radiation can lead to the development of actinic prurigo.

## **Types Of Actinic Prurigo**

Acute actinic prurigo typically occurs within hours of sun exposure and causes intense itching and redness. Chronic actinic prurigo is more persistent, with symptoms lasting for an extended period and potentially leading to skin thickening and scarring. Both types can cause discomfort and require proper management to alleviate symptoms.

Actinic prurigo polymorphous light eruption (AP-PLE) is a common type of actinic prurigo that usually presents as itchy, red bumps or patches on sun-exposed areas of the skin, such as the face, neck, and hands.

Actinic prurigo actinico (APA) is a severe form of the condition characterized by intense itching, burning sensations, and the development of blisters or ulcers on the skin after sun exposure.

Actinic prurigo Cheilitis is a subtype of actinic prurigo that primarily affects the lips, causing redness, swelling, and painful sores, often triggered by exposure to sunlight.

## **Risk Factors**

Actinic prurigo risk factors include genetics, with a higher prevalence among Native American and Hispanic populations. Sun exposure is a key factor, with ultraviolet radiation triggering the condition. Individuals with certain HLA gene types are more susceptible. Other factors include a history of [atopic](https://www.medicoverhospitals.in/diseases/atopic-dermatitis/) dermatitis and family history of actinic prurigo. Adequate sun protection is crucial in prevention.

Fair skin with light-colored eyes and hair increases the risk of developing actinic prurigo.

Prolonged or intense exposure to sunlight, especially in high altitudes or regions closer to the equator, is a significant risk factor for actinic prurigo.

Family history of actinic prurigo or other photosensitive conditions can predispose individuals to the condition.

Certain genetic factors, such as variations in immune system function, may contribute to an increased susceptibility to actinic prurigo.

Individuals with a history of chronic sunburns or frequent sun exposure without proper protection are at a higher risk of developing actinic prurigo.

**SYMPTOMS**

Symptoms may include burning sensation, swelling, and crusty sores. It can be triggered by sunlight and worsen during spring and summer months. In severe cases, it can lead to scarring and changes in skin pigmentation. Using sun protection and avoiding sun exposure can help manage symptoms.

Itchy bumps or rash on sun-exposed skin, such as the face, neck, and arms, are common symptoms of actinic prurigo.

Swelling, redness, and tenderness of the affected skin areas may occur in individuals with actinic prurigo.

Patients with actinic prurigo may experience a burning sensation or pain in the skin when exposed to sunlight.

Thickened or scaly patches on the skin, especially on the lips and around the eyes, can be indicative of actinic prurigo.

Some individuals with actinic prurigo may develop blisters or sores that can be itchy and uncomfortable, worsening with sun exposure.

## **Diagnosis of Actinic Prurigo**

To diagnose actinic prurigo, your doctor will examine your skin and ask about your symptoms and sun exposure history. A skin biopsy may be needed to confirm the diagnosis. Blood tests may be done to rule out other conditions. Keeping a record of your symptoms and sun exposure can help with diagnosis. It's important to see a dermatologist for proper evaluation and treatment.

Physical examination: A healthcare provider may visually inspect the affected skin areas to look for characteristic signs of actinic prurigo, such as redness, blisters, and crusts.

Skin biopsy: A small sample of skin tissue may be taken and examined under a microscope to confirm the diagnosis of actinic prurigo and rule out other skin conditions.

Phototesting: This diagnostic test involves exposing the skin to controlled amounts of ultraviolet (UV) light to identify specific wavelengths that trigger symptoms of actinic prurigo.

Blood tests: Blood tests can help assess levels of specific antibodies or immune cells that may be elevated in individuals with actinic prurigo, aiding in the diagnostic process.

## **Treatment for Actinic Prurigo**

Treatment options for actinic prurigo may include topical corticosteroids, antihistamines, and phototherapy. Over-the-counter creams and avoidance of sun exposure are also recommended. In severe cases, prescription medications or immunosuppressants may be prescribed. It is important to consult a dermatologist for a personalized treatment plan tailored to your specific condition. Regular follow-ups are crucial for managing symptoms and preventing flare-ups.

Topical corticosteroids can help reduce inflammation and itching associated with actinic prurigo by suppressing the immune response in the affected skin areas.

Oral antihistamines such as loratadine or cetirizine may be prescribed to alleviate itching and discomfort caused by actinic prurigo, providing relief for patients.

Phototherapy, specifically narrowband UVB therapy, is a common treatment option for actinic prurigo as it helps desensitize the skin to UV light, reducing the severity of symptoms over time.

Immunosuppressive medications like hydroxychloroquine or azathioprine may be recommended for severe cases of actinic prurigo to modulate

**Differential Diagnosis**

Originally, actinic prurigo was thought to be a hereditary type of polymorphous light eruption (PMLE), more common idiopathic photodermatoses. Genetic testing and the unique clinical presentation of actinic prurigo now support two separate disease entities. The HLA-DR4 allele, specifically the DRB1\*0407 subtype is strongly associated with actinic prurigo and not PMLE. Actinic prurigo also has an earlier age of onset and frequently presents with cheilitis and sometimes conjunctivitis, which is never seen in patients with PMLE. When suspecting actinic prurigo, laboratory tests are also necessary to rule out systemic diseases such as lupus erythematosus or porphyria.

Polymorphous light eruption

Systemic lupus erythematosus

Porphyria

**Prognosis**

Actinic prurigo is a chronic disease process and can reoccur with repeated sun exposure. Treatment with immunosuppressive agents like thalidomide and cyclosporine A has been shown to be effective at long-term suppression of symptoms. Some adolescents may have spontaneous resolution of symptoms and may not demonstrate disease progression into adulthood.

**Complications**

* Complications of actinic prurigo include:
* Secondary bacterial infections
* Contact dermatitis
* Impetigo

**Prevention**

Actinic prurigo has no known cure. The basic objective is to avoid sun exposure as much as possible. Patients must understand that their illness will deteriorate during the hottest months of the year, and they must use sun protection techniques to minimize or limit breakouts

## Protect your skin with clothing. Ensure that you wear a hat that protects your face, neck, and ears, and a pair of UV-protective sunglasses.

## Make use of shade between 11am and 3pm when it’s sunny.

## No sunscreen can offer you 100% protection. They should be used to provide additional protection from the sun, not as an alternative to clothing and shade.

## It may be worth taking vitamin D supplement tablets (available from health food stores) as strictly avoiding sunlight can reduce your vitamin D levels.

## Increasing intake of food rich in vitamin D such as oily fish, eggs, meat, fortified margarine, and cereals.

## **EPIDEMIOLOGY**

## Actinic prurigo is a rare photo dermatosis in the United States. However, actinic prurigo is more common in the American Indian populations of North, Central, and South America. The disease can affect both genders but is more commonly described in female patients. Actinic prurigo can manifest at any age, although the disease typically presents in prepubescent individuals with the mean onset before age 10. Some studies suggest an increased prevalence of actinic prurigo in certain geographic areas with dry, warm climates at an altitude of at least 1000 meters above sea level.

## REFERENCES

## [Actinic Prurigo - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK499957/)

[Actinic Prurigo: Signs, Causes, And Treatment](https://www.medicoverhospitals.in/diseases/actinic-prurigo/)

### **ARGYRIA**

**DEFINITION AND DESCRIPTION**

Argyria is a condition that causes your skin and mucous membranes (the lining of parts of your body) to turn blue to gray. This is the result of silver building up in your body. You can get this condition if you have frequent exposure over time to microscopic silver compounds that absorb into your body.

Silver is an element that’s found in the Earth’s crust. This element has many functions and can be formed into jewelry, tableware, mirrors and batteries. Microscopic metal particles surround us every day. They’re in the air, the food you eat, the water you drink, in dental fillings and some medicines and supplements. You have small amounts of silver in your body at all times. When there’s too much silver in your body, you’ll experience symptoms of argyria.

Argyria isn’t common because the use of silver in manufacturing and medicine significantly declined in the 21st century. People who work with silver or use colloidal silver as a dietary supplement are most at risk of developing this condition.

There are three types of argyria:

* **Generalized**: Symptoms of argyria affect your entire body or large areas of your skin.
* **Localized**: Symptoms only affect a small part of your body or one area of your skin.
* **Argyrosis**: Symptoms of argyria affect your eye or eyes only.

## **Symptoms**

Symptoms of argyria include:

* Your skin turning blue-gray or gray.
* Your skin being darker in sun-exposed areas (hyperpigmentation).
* Your fingernails are darker than normal.
* Small bumps (macules) forming on a patch of your skin that’s blue to gray.

Early signs of the condition can begin in your mouth, where sections of your gums could appear brownish gray before the condition spreads to your skin.

Your symptoms could vary based on:

* How much silver is in your body.
* How the silver entered your body.
* The length of time silver has been in your body.

Additional symptoms caused by a buildup of silver in your body could include:

* Abdominal pain.
* Fatigue.
* Headaches.
* Kidney damage.
* Resistance to certain medications.
* Skin irritation.
* Seizures.

### **causes**

Silver toxicity causes argyria. Silver toxicity occurs when too much silver is in your body.

Your body has very small amounts of silver and other metals in it. Toxicity usually happens when you’re exposed to silver particles for a long period of time. Silver can enter your body by:

* **Absorption**: Absorbing into your skin.
* **Inhalation**: Entering your lungs when you breathe through your nose and mouth.
* **Ingestion**: Entering your stomach through the foods and beverages you eat or drink or through the medications or supplements you take.

#### **How do I get silver exposure?**

Silver exposure occurs when microscopic silver deposits absorb into your body. This normally happens over a long period of time. You can’t get silver exposure by eating with a silver fork or wearing jewelry. You can get silver exposure by:

* Working with elemental silver on the job (occupational exposure). This affects people who mine silver or work in a manufacturing facility that adds silver to products.
* Taking medication or dietary supplements that include silver salts, colloidal silver or silver acetate. Long-term use of certain medications or supplements that contain small amounts of silver can cause this condition.
* Receiving a surgical procedure that used silver sutures to stitch a wound or receiving dental fillings made with amalgam.

## **Diagnosis and Tests**

Your provider will diagnose argyria after a complete medical history and a physical exam to learn more about your symptoms. Your provider might ask questions to see if you recently had exposure to metals or if you used any products that contain silver compounds.

Tests could confirm your diagnosis and rule out other conditions. A skin biopsy is the most effective test where your provider will remove a small sample of your skin tissue that’s gray or blue and examine it for silver deposits under a microscope.

## **Management and Treatment**

Treatment for argyria is challenging for healthcare providers because it’s difficult to reverse the changes to your skin after they appear.

If you receive an argyria diagnosis, your provider will recommend you stop using any products that contain silver, including medicines, eye drops or supplements. If you work in an environment where silver is present, your provider will recommend you wear personal protective equipment while at work, which could include gloves, eye protection and/or a mask.

Treatment with chelating agents, which are medicines that remove metals from your body, don’t work well with argyria.

Some cases of argyria skin discoloration improve with laser therapy. Laser therapy is a technique where your provider will use a powerful light on your skin where symptoms appear to destroy existing skin tissue. This helps your body produce new, healthy skin tissue.

#### **Are there side effects of the treatment?**

All treatment options for argyria vary based on how much silver is in your body. The results from treatment may range from somewhat effective to not effective at all since skin discoloration symptoms can be irreversible. Research is ongoing to learn more about treatment options for argyria.

### **How do I manage my symptoms?**

Your symptoms of skin discoloration could increase with ultraviolet (UV) exposure from the sun. To avoid further skin discoloration, wear protective clothing or accessories when outdoors and wear sunscreen daily. If you’re active outside, reapply sunscreen throughout the day when you start to sweat or if your skin gets wet.

You can use makeup to temporarily cover up areas of your skin where you have symptoms of argyria. Make sure you wash off the makeup at the end of the day to prevent clogging your pores.

## **Prevention**

You can reduce your risk of developing argyria by:

* Not using products that contain silver.
* Wearing personal protective equipment if you work with elemental silver.
* Wearing sunscreen to prevent your skin from getting darker from sun exposure (hyperpigmentation).
* Talking to your provider if you take a medicine that contains silver.
* Replacing amalgam dental fillings that contain silver.

## **Outlook / Prognosis**

### **What can I expect if I have argyria?**

Argyria can be difficult to treat, and symptoms may be irreversible. You can wear makeup to temporarily cover up the blue-gray tone of your skin if treatment is ineffective.

Talk to your provider before using any products or supplements that contain trace amounts of silver, especially colloidal silver. If you work with silver at your job, talk to your employer about steps you can take to protect yourself from silver compounds in your workplace by wearing personal protective equipment.

## **Living With**

### **When should I see my healthcare provider?**

Visit your healthcare provider if you have symptoms of argyria. Early detection and avoidance of silver prevent symptoms from spreading throughout your body.

### **What questions should I ask my doctor?**

* Should I get my amalgam fillings replaced?
* How can I protect myself from silver at work?
* Is laser therapy right for me?
* Are there side effects of the treatment?

## **Differential Diagnosis**

* Cyanosis
* Hemochromatosis
* Methemoglobinemia
* Methylene blue poisoning
* Melanoma
* Ochronosis
* Chrysiasis
* Amiodarone, minocycline, or phenothiazines use

## **Cyanosis**

A clinical sign characterized by bluish or purplish discoloration of the skin, mucous membranes, and nail beds due to increased levels of deoxygenated hemoglobin (>3-5 g/dL) in the blood. It indicates inadequate oxygenation of blood or impaired oxygen delivery to tissues. Cyanosis is classified as:

* Central cyanosis: Involves the core, lips, tongue, and trunk, usually due to systemic hypoxemia from heart or lung diseases such as congenital heart defects, pneumonia, pulmonary embolism, or chronic obstructive pulmonary disease.
* Peripheral cyanosis: Affects extremities and is often caused by vasoconstriction, poor circulation, or local hypoxia (e.g., Raynaud’s phenomenon, shock).

Treatment targets the underlying cause and may include oxygen therapy, warming of affected areas, intravenous fluids, or surgery in congenital heart disease. Cyanosis can also result from exposure to cold, medication overdose, or poisoning.

**Hemochromatosis**

A genetic disorder causing excessive iron accumulation in tissues, leading to skin hyperpigmentation (bronze or slate-gray discoloration), diabetes, liver disease, and other systemic effects. The pigmentation results from iron deposition and increased melanin production.

**Methemoglobinemia**

A condition where hemoglobin is oxidized to methemoglobin, which cannot bind oxygen effectively, causing functional hypoxia and cyanosis unresponsive to oxygen therapy. It may be congenital or acquired from exposure to oxidizing agents or certain drugs.

**Methylene Blue Poisoning**

An adverse reaction to methylene blue, a treatment for methemoglobinemia, which in overdose can paradoxically cause methemoglobinemia or hemolysis, leading to cyanosis and other systemic symptoms.

**Melanoma**

A malignant tumor of melanocytes presenting as pigmented skin lesions with asymmetry, border irregularity, color variation, diameter enlargement, and evolution. Acral and mucosal melanomas can cause pigmentation changes and require prompt diagnosis and treatment.

**Ochronosis**

A rare disorder characterized by bluish-black pigmentation of connective tissues and skin due to accumulation of homogentisic acid in alkaptonuria or from exogenous causes (e.g., topical hydroquinone use). It causes dark discoloration of cartilage, sclera, and skin.

**Chrysiasis**

A condition of blue-gray skin discoloration resulting from prolonged gold salt therapy, historically used in rheumatoid arthritis treatment. The pigmentation is permanent and results from gold deposition in the dermis.

**Drug-induced Pigmentation (Amiodarone, Minocycline, Phenothiazines)**

Certain medications can cause skin discoloration:

* **Amiodarone**: Causes blue-gray pigmentation, especially on sun-exposed areas, due to drug and metabolite deposition.
* **Minocycline**: Leads to blue-gray or slate pigmentation on skin, nails, and mucosa after prolonged use.
* **Phenothiazines**: Can cause blue or gray pigmentation due to drug accumulation in the skin.

**Epidemiology**

## Argyria is much less common in the 21st century because of the decline in heavy exposure to silver and its use in medicine; however, it does still exist. Generalized argyria cases in modern society are typically seen in individuals who consume colloidal silver as a form of alternative medicine. Argyria affects individuals of all races, genders, and age groups without any specific predilection.

## REFERENCE

## [Argyria - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK563123/)

## [Argyria: Symptoms, Causes & Treatment](https://my.clevelandclinic.org/health/diseases/25163-argyria)

**CHROMHIDROSIS**

**DEFINITION AND DESCRIPTION**

Chromhidrosis is a chronic condition that causes a person to have colorful sweat.

There are three types

* **Apocrine chromhidrosis:** This affects areas that contain apocrine sweat glands, such as the torso, eyelids, scalp, ears, and areola — the darker area of skin around the nipple.
* **Eccrine chromhidrosis:** Because eccrine sweat glands are widely distributed, this can affect sweat in almost every area of the body.
* **Pseudochromhidrosis:** This results when dyes, chemicals, or pigment-producing bacteria mix with colorless eccrine sweat to form colored sweat.

Chromhidrosis can occur at nearly any age, but it usually becomes noticeable after puberty, when the apocrine glands begin secreting fluid.  
  
The International Hyperhidrosis Society (IHS) notes that information about this condition is scarce because it is so rare. There seems to be no association between chromhidrosis and sex, geographic location, season, or weather, though it may be more likely to develop in people of African descent.

Although the condition is chronic, the discoloration of sweat may decrease over time as the body produces less lipofuscin, a pigment that is likely responsible for the color changes.

People with chromhidrosis may have more lipofuscin, or lipofuscin that is more oxidized, than others.

Chromhidrosis is a harmless condition. However, stress or embarrassment about the coloration can lead to depression and anxiety.

## **Symptoms**

## The defining symptom of chromhidrosis is the production of colored sweat. The color may only affect sweat in certain areas or sweat all over the body. The color and the vividness of the shade can vary from person to person.

Some people experience a warm or prickly feeling caused by stress or physical activity before colored sweat appears.

Chromhidrosis can cause sweat to turn:

* black
* green
* blue
* yellow
* brown

Anyone who has chromhidrosis should speak with a healthcare provider if they start to experience symptoms of emotional distress, depression, or anxiety. These symptoms can include:

* a general feeling of hopelessness, worthlessness, helplessness, guilt or pessimism
* a persistent anxious, sad, or “empty” mood
* a decrease in energy or increase in fatigue
* weight loss
* a lack of interest in once enjoyable activities
* physical symptoms, such as a headache, that does not respond to treatment
* insomnia
* trouble concentrating, making decisions, or remembering
* a lack of appetite or overeating
* irritability
* thoughts of suicide or death

## **Causes**

There are a few possible causes of chromhidrosis, depending on the type.

In a person with apocrine chromhidrosis, lipofuscin causes discoloration as part of the natural process of creating sweat.  
  
Certain situations stimulate the apocrine glands and make this discoloration more likely to occur:

* friction against the skin
* hot showers or baths
* stimuli such as anxiety, sexual arousal, or pain

In people with eccrine chromhidrosis, the discoloration usually happens because the person has ingested;

* water-soluble dyes
* heavy metals, such as copper
* certain food colorings or flavorings
* certain medications, such as bisacodyl, a laxative, when it is coated in tartrazine, a yellow dye

Pseudochromhidrosis is more common, and it occurs when the skin comes into contact with:

* chemicals
* dyes
* pigment-producing bacteria

The [IHS](https://www.sweathelp.org/where-do-you-sweat/other-sweating/chromhidrosis.html) also point to other health issues that can cause sweat to change color:

* infection
* blood in the sweat
* extra bilirubin from the liver
* poisoning

It is a good idea to see a doctor about colored sweat. They can rule out more serious causes and recommend any necessary treatment.

## **Diagnosis**

* Chromhidrosis is typically diagnosed through a physical exam and medical history
* Additional tests, such as sweat and blood tests, may be ordered to confirm the diagnosis and rule out other conditions

Many clinical conditions may have similar signs and symptoms. Your healthcare provider may perform additional tests to rule out other clinical conditions to arrive at a definitive diagnosis.

## **Complications of Chromhidrosis**

Complications of Chromhidrosis can include embarrassment, anxiety, and social isolation due to the unusual appearance of sweat.

## **Management and Treatment of Chromhidrosis**

Managing chromhidrosis involves addressing the underlying causes and mitigating symptoms. Treatment options vary based on the type and severity of the condition.

### **Topical Treatments**

Topical treatments are often the first line of defense in managing chromhidrosis. These may include:

Topical Aluminum Chloride: Commonly used for hyperhidrosis, aluminum chloride can help reduce sweat production and mitigate symptoms.

Capsaicin Cream: Capsaicin cream can help desensitize the sweat glands and reduce the production of colored sweat.

### **Oral Medications**

In some cases, oral medications may be prescribed to manage chromhidrosis. These can include:

Anticholinergics: Medications that reduce sweat production by inhibiting the activity of sweat glands.

Beta-Blockers: Beta-blockers can help manage stress-induced sweating, which may exacerbate chromhidrosis.

### **Lifestyle Modifications**

Lifestyle modifications can also play a significant role in managing chromhidrosis. These may include:

Avoiding Trigger Substances: Identifying and avoiding substances that contribute to colored sweat can help manage symptoms.

Stress Management: Stress can exacerbate sweating, so stress management techniques such as meditation and yoga can be beneficial.

### **Medical Procedures**

In severe cases, medical procedures may be considered to manage chromhidrosis. These can include:

Botulinum Toxin Injections: Botulinum toxin injections can help reduce sweat production by temporarily paralyzing the sweat glands.

Laser Therapy: Laser therapy can target and reduce the activity of the affected sweat glands.

## **Prevention**

Currently, it may not be possible to prevent Chromhidrosis, but avoiding triggers or substances that may exacerbate the condition can help reduce the frequency and severity of symptoms.

## **Prognosis of Chromhidrosis**

* The prognosis for Chromhidrosis varies depending on the severity of the condition and the underlying cause
* With proper treatment and management, many people are able to manage their condition and maintain a good quality of life

**DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of chromhidrosis includes bleeding diathesis (hematidrosis), hyperbilirubinemia, Addison’s disease, hemochromatosis, poisoning, and alkaptonuria. Hematidrosis, or bloody sweat, is a rare condition where blood seeps from the skin and mucosa. It may occur anywhere on the body and can be associated with pain. Alkaptonuria is a rare genetic condition caused by a deficiency in functional homogentisic oxidase. Patients may present in early adulthood with darkened skin and urine, and arthritis

**EPIDEMIOLOGY**

Apocrine chromhidrosis may appear at any age but usually appears after puberty, when the apocrine secretory function begins. The disease is considered chronic, however, may regress with age as apocrine secretion diminishes. Apocrine chromhidrosis displays no occupational or geographical predisposition and is not influenced by climatic or seasonal variation. There is no gender predilection, but chromhidrosis has been reported in the literature more commonly in blacks, barring facial chromhidrosis, which has been reported more commonly in whites. However, there are too few patients reported to draw meaningful conclusions.

REFERENCE

[Chromhidrosis - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK554395/)

[Chromhidrosis - DoveMed](https://www.dovemed.com/diseases-conditions/chromhidrosis)

[Chromhidrosis: Types, Causes, Symptoms, and Treatment](https://www.medicoverhospitals.in/diseases/chromhidrosis/)

[Chromhidrosis: Definition, causes, and treatment](https://www.medicalnewstoday.com/articles/chromhidrosis#causes)

**EPIDERMOLYSIS BULLOSA**

**DEFINITION AND DESCRIPTION**

Epidermolysis bullosa (ep-ih-dur-MOL-uh-sis buhl-LOE-sah) is a rare condition that causes fragile, blistering skin. The blisters may appear in response to minor injury, even from heat, rubbing or scratching. In severe cases, the blisters may occur inside the body, such as the lining of the mouth or stomach.

Epidermolysis bullosa is inherited, and it usually shows up in infants or young children. Some people don't develop symptoms until they're teens or young adults.

Epidermolysis bullosa has no cure, but mild forms may improve with age. Treatment focuses on caring for blisters and preventing new ones.

**Symptoms**

Epidermolysis bullosa symptoms include:

* Fragile skin that blisters easily, especially on the palms and feet
* Nails that are thick or unformed
* Blisters inside the mouth and throat
* Scalp blistering and hair loss (scarring alopecia)
* Skin that looks thin
* Tiny pimple-like bumps (milia)
* Dental problems, such as tooth decay
* Difficulty swallowing
* Itchy, painful skin

Usually, epidermolysis bullosa blisters are noticed during infancy. But it's not uncommon for them to appear when a toddler first begins to walk or when an older child begins new activities that cause more friction on the soles of the feet.

### **When to see a doctor**

Contact your health care provider if you or your child develops blisters for an unknown reason. For infants, severe blistering can be life-threatening.

**Seek immediate medical care** if you or your child:

* Has problems swallowing
* Has problems breathing
* Shows signs of infection, such as warm, painful or swollen skin, pus, or an odor from a sore, and fever or chills

**Causes**

Epidermolysis bullosa is caused by an inherited gene. You may inherit the disease gene from one parent who has the disease (autosomal dominant inheritance) or from both parents (autosomal recessive inheritance).

The skin is made up of an outer layer (epidermis) and an underlying layer (dermis). The area where the layers meet is called the basement membrane. The types of epidermolysis bullosa are mainly defined by which layers separate and form blisters. The skin injury might be brought on by a minor injury, bump or nothing at all.

The main types of epidermolysis bullosa are:

* **Epidermolysis bullosa simplex.** This is the most common type. It's brought on by heat and friction and develops in the outer layer of skin. It mainly affects the palms and feet. The blisters heal without scarring.
* **Junctional epidermolysis bullosa.** This type may be severe, with blisters beginning in infancy. A baby with this condition may develop a hoarse-sounding cry from continual blistering and scarring of the vocal cords.
* **Dystrophic epidermolysis bullosa.** This type is related to a flaw in the gene that helps produce a protein that glues the skin layers together. If this protein is missing or doesn't function, the layers of the skin won't join properly. It can cause skin that looks thin. Diseased mucous membranes can cause constipation and make it hard to eat.
* **Kindler syndrome.** This type tends to cause blisters in multiple layers and so can look very different from person to person. The blisters tend to show up in infancy or early childhood. It increases sun sensitivity and causes skin to look thin, mottled and wrinkly.

Epidermolysis bullosa acquisita is distinct from these conditions, as it isn't inherited and it's rare in children.

**Risk factors**

The major risk factor for developing epidermolysis bullosa is having a family history of the disorder.

**Complications**

Epidermolysis bullosa can worsen even with treatment, so it's important to spot signs of complications early. Complications may include:

* **Infection.** Blistering skin can become infected by bacteria.
* **Bloodstream infection.** Sepsis occurs when bacteria from an infection enter the bloodstream and spread throughout the body. Sepsis can spread rapidly and lead to shock and organ failure.
* **Fusion of fingers and changes in the joints.** Severe forms of epidermolysis bullosa can bind together fingers or toes and cause unusual bending of the joints (contractures). This can affect the function of the fingers, knees and elbows.
* **Problems with nutrition.** Blisters in the mouth can make eating difficult and lead to malnutrition and anemia, such as low iron levels in the blood. Problems with nutrition can also cause delayed wound healing and slowed growth in children.
* **Constipation.** Difficulty passing stool may be due to painful blisters in the anal area. It can also be caused by not ingesting enough liquids or high-fiber foods, such as fruits and vegetables.
* **Dental problems.** Tooth decay and problems with tissues inside the mouth are common with some types of epidermolysis bullosa.
* **Skin cancer.** Teenagers and adults with certain types of epidermolysis bullosa are at increased risk of a type of skin cancer called squamous cell carcinoma.
* **Death.** Infants with severe junctional epidermolysis bullosa are at high risk of infections and loss of body fluids from widespread blistering. Blisters in the mouth and throat also make it harder to eat and breathe. Many of these infants don't survive.

**Prevention**

It's not possible to prevent epidermolysis bullosa. But these steps may help prevent blisters and infection.

* **Handle your child gently.** Your infant or child needs cuddling, but be very gentle. To pick up a child with epidermolysis bullosa, place the child on soft material and give support under the buttocks and behind the neck. Don't lift the child from under the arms.
* **Take special care with the diaper area.** If your child wears diapers, remove the elastic bands and avoid cleansing wipes. Line the diaper with a non stick dressing or spread it with a thick layer of zinc oxide paste.
* **Keep the home environment cool.** Try to keep your home cool and the temperature steady.
* **Keep the skin moist.** Gently apply moisturizer as needed throughout the day.
* **Dress your child in soft clothes.** Use soft clothing that's simple to get on and off. It may help to remove labels and put on clothing seam-side out to reduce scratching. Try sewing foam pads into the lining of clothing by elbows, knees and other pressure points. Use soft special shoes, if possible.
* **Prevent scratching.** Trim your child's fingernails regularly.
* **Encourage your child to be active.** As your child grows, encourage activities that reduce the risk of skin injury. Swimming is a good option. For children with mild forms of epidermolysis bullosa, they can protect the skin by wearing long pants and sleeves for outdoor activities.
* **Cover hard surfaces.** Consider padding a car seat or bathing tub with sheepskin, foam or a thick towel. Soft cotton or silk can be used as a top layer over the padding.

## **Diagnosis**

Your health care provider may identify epidermolysis bullosa from the skin's appearance. You or your child may need tests to confirm the diagnosis. The tests may include:

* **Biopsy for immunofluorescence mapping.** With this technique, a small sample of affected skin or mucous membrane is removed and examined with a special microscope. It uses reflected light to identify the layers of skin involved. This test also identifies whether the proteins needed for skin growth are present and healthy.
* **Genetic testing.** With this test, your health care provider takes a small sample of blood and sends it to a lab for DNA analysis.
* **Prenatal testing.** Families with a history of epidermolysis bullosa may want to consider prenatal testing and genetic counseling.

**Treatment**

Treatment for epidermolysis bullosa may first include lifestyle changes and home care. If these don't control symptoms, your health care provider might suggest one or more of the following treatments:

### **Medications**

Medications can help control pain and itching. Your health care provider may also prescribe pills to fight infection (oral antibiotics) if there are signs of widespread infection, such as fever and weakness.

### **Surgery**

Surgical treatment may be needed. Options sometimes used for this condition include:

* **Widening the esophagus.** Blistering and scarring of the long, hollow tube that runs from the throat to the stomach (esophagus) may lead to narrowing of the tube. This makes it hard to eat. Making the tube wider with surgery can make it easier for food to travel to the stomach.
* **Placing a feeding tube.** To improve nutrition and help with weight gain, a feeding tube (gastrostomy tube) may be needed to deliver food directly to the stomach.
* **Grafting skin.** If scarring has affected the function of a hand, the surgeon may suggest a skin graft.
* **Restoring movement.** Repeated blistering and scarring can cause fusing of the fingers or toes or unusual bends in the joints (contractures). A surgeon might recommend surgery to correct these conditions if they restrict movement.

### **Rehabilitation therapy**

Working with a rehabilitation specialist can help in learning to live with epidermolysis bullosa. Depending on your goals and how movement is limited, you might work with a physical therapist or an occupational therapist.

### **Potential future treatments**

Researchers are studying better ways to treat and relieve the symptoms of epidermolysis bullosa, including:

* Gene therapy, including a gel applied to wounds of people with dystrophic epidermolysis bullosa
* Bone marrow (stem cell) transplantation
* Protein replacement therapies
* Other cell-based therapies

**Lifestyle and home remedies**

You can take steps at home to care for blisters and prevent new ones from forming. Talk with your health care provider about how to care for wounds and provide good nutrition.

### **Caring for blisters**

Your health care provider can show you how to care for blisters properly and advise you on ways to prevent them. Ask about safe ways to drain blisters before they get too large. Ask about recommended products for keeping the affected areas moist. This helps with healing and preventing infection.

In general, take these steps:

* **Wash your hands.** Wash your hands before touching blisters or changing dressings.
* **Control pain.** About 30 minutes before a dressing change or other painful procedure, older children and adults may take a prescription-strength pain medication. For people who don't respond to pain relievers, other options include antiseizure drugs such as gabapentin.
* **Cleanse skin daily.** To cleanse a wound, soak it for 5 to 10 minutes in a mild solution of salt and water. Other options are mild solutions of diluted vinegar or bleach. Soaking loosens stuck bandages and helps reduce the pain of changing bandages. Rinse with lukewarm water.
* **Puncture new blisters.** This prevents them from spreading. Use a sterile needle to puncture each new blister in two spots. But leave the roof of the blister intact to allow for drainage while protecting the underlying skin.
* **Apply treated dressings.** Spread petroleum jelly or other thick moisturizer on a nonstick bandage (Mepilex, Telfa, Vaseline gauze). Then gently place the bandage on the wound. Secure the bandage with rolled gauze if needed.
* **Wrap blistered hands and feet daily.** With some severe forms of this condition, daily wraps help prevent contractures and fusion of the fingers and toes. Special wraps and gauze dressings are useful for this treatment.
* **Watch for signs of infection.** If you notice heat, pus or lines leading from the blister, talk with your health care provider about prescription antibiotics.
* **Keep it cool.** Blistering is often worsened by heat and warm conditions.

### **Providing good nutrition**

A varied, nutritious diet promotes growth and development in children and helps wounds heal. If blisters in the mouth or throat make it difficult to eat, here are some suggestions:

* For babies with mild epidermolysis bullosa, breastfeeding is fine. Otherwise, minimize injury from feeding by using bottle nipples designed for premature infants, a syringe or a rubber-tipped medicine dropper. Try softening bottle nipples in warm boiled water.
* For older children, serve nutritious, soft foods that are easy to swallow, such as vegetable soup and fruit smoothies. Puree solid foods with broth or milk.
* Serve food and beverages lukewarm, at room temperature or cold.

Talk with your health care provider about how you or your child can get all the needed nutrients and vitamins.

**Preparing for your appointment**

You may be referred to a doctor who specializes in the diagnosis and treatment of skin conditions (dermatologist).

### **What you can do**

* **List your or your child's signs and symptoms** and how long they've been present.
* **Note any new sources of friction around the blistering areas,** if any. For example, tell your health care provider if your toddler has recently started walking or your older child has begun physical activities that put new pressure on the affected areas.
* **List key medical information,** including other medical problems you or your child has received a diagnosis for. Also list the names of all nonprescription and prescription medications you or your child is taking. Also list any vitamins and supplements.
* **Ask a trusted family member or friend to join you for the appointment.** If your health care provider tells you that your child has epidermolysis bullosa, you may have difficulty focusing on anything else that's said. Take someone along who can offer emotional support and help you recall all the information discussed at your appointment.
* **List the questions** to ask your healthcare provider.

### **Questions to ask your doctor**

* What's the most likely cause of the signs and symptoms?
* What are other possible causes for these signs and symptoms?
* What kinds of tests are needed?
* What treatments are available, and what types of side effects might they cause?
* What can be done to relieve pain or discomfort?
* How do I take care of my child's needs, such as feeding, bathing and clothing?
* What are the possible complications of this condition?
* What signs or symptoms related to this condition should prompt me to call you?
* What signs or symptoms should prompt me to call 911 or my local emergency number?
* What restrictions do we need to follow?
* Do you think my child's symptoms will improve with age?
* If I plan to have more children, are they at increased risk of this condition?
* How can I find other people who are coping with epidermolysis bullosa?
* Where can I find additional information and resources?

**EPIDEMIOLOGY**

Approximately 70% of epidermolysis bullosa is epidermolysis bullosa simplex. Dystrophic epidermolysis bullosa accounts for around 25% of all cases, and junctional epidermolysis bullosa the remaining 5%. Kindler epidermolysis bullosa is notably the rarest of the 4 epidermolysis bullosa types, with only around 400 cases reported worldwide.

Despite the challenges in formally diagnosing epidermolysis bullosa in resource-poor settings, robust epidemiological studies agree that epidermolysis bullosa prevalence is around 10 per one million population, with incidence at around 20 per one million live births. While epidermolysis bullosa appears not to be sex-preferential, trials conducted in Scottish and Middle Eastern populations suggest a geographically biased distribution; this is thought to be attributable to fewer germ lines within small geographical confines, and sociocultural influences where consanguineous marriage is more common

**DIFFERENTIAL DIAGNOSIS**

It is essential to rule out possible differential diagnoses, particularly in neonatal populations:

**Pompholyx Eczema** presents with acral pruritic blistering. A history of atopy may be present. Patch testing may be indicated to identify delayed hypersensitivity reactions to contact allergens.

**Porphyria** presents with scarring, milia, and photosensitivity, with possible hypertrichosis. Skin biopsies and porphyrin testing may be indicated.

**Bullous Pemphigoid** presents with large, tense bullae and itch. Clinical examination and skin biopsy can confirm bullous pemphigoid.

**Bullous Systemic Lupus Erythematosus** presents with tense vesicles, bullae, and erosions, often in sun-exposed areas. Histopathology and immunofluorescence confirm this differential.

**Bullous Tinea Pedis** presents with multilocular pedal blistering, with possible skin maceration between digits. Tinea pedis is diagnosed through mycological cultures from skin scrapings.

**Palmoplantar Keratoderma** causes acral skin thickening, often with nail dystrophy. Genetic testing may be indicated, though acquired palmoplantar keratoderma may be associated with internal malignancy, thyroid abnormalities, inflammatory dermatoses such as lichen planus, and certain medications.

**Epidermolysis Bullosa Acquisita** is an autoimmune disorder that may present similarly to dystrophic epidermolysis bullosa but later in life. Immunofluorescence studies on skin biopsies are indicated.

**Ehlers-Danlos Syndrome** features joint hypermobility, hyperextensible skin, and easy bruising with skin fragility. Ehlers Danlos is diagnosed by skin biopsy and genetic testing.

**Incontinentia Pigmenti** features Blaschkoid vesicles forming hyperpigmented verrucous whorls.

REFERENCE

[Epidermolysis bullosa - Diagnosis and treatment - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/epidermolysis-bullosa/diagnosis-treatment/drc-20361146)

[Epidermolysis bullosa - Symptoms and causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/epidermolysis-bullosa/symptoms-causes/syc-20361062)

[Epidermolysis Bullosa - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/sites/books/NBK599531/)

### **Harlequin ichthyosis**

**DEFINITION AND DESCRIPTION**

Harlequin ichthyosis is a rare genetic skin disease that affects newborns. When an infant is born with the condition, their body is covered with plates of hard, thick skin that crack and split apart. These diamond-shaped plates can pull at and distort your baby’s facial features, affecting the shape of their ears, eyelids, nose and mouth. The disease can also limit the movements of your baby’s arms and legs and restrict their eating and breathing.

Your skin ordinarily forms a protective wall or barrier between your body and its environment. When your baby has harlequin ichthyosis, their skin abnormalities disrupt this barrier. This can make it hard for your baby to:

* Control water loss.
* Regulate their body temperature.
* Fight infections.

Your baby may experience dehydration and develop life-threatening infections in their first few weeks of life. After the newborn period, the plates shed, and your baby’s skin develops widespread redness and scales.

Harlequin ichthyosis is the most severe kind of ichthyosis, a skin disease with more than 20 types, including lamellar ichthyosis and ichthyosis vulgaris. In the past, it was rare for babies with harlequin ichthyosis to survive the newborn period. But now, with improved treatment options and intensive medical care, babies have a better chance of living into childhood and reaching adulthood.

**Other names for harlequin ichthyosis include:**

* Autosomal recessive congenital ichthyosis — harlequin type ichthyosis.
* Harlequin baby syndrome.
* Harlequin fetus.
* Ichthyosis congenita.
* Ichthyosis fetalis.

#### **How common is harlequin ichthyosis?**

Harlequin ichthyosis affects about 1 in every 300,000 to 500,000 babies born in the United States.

## **Symptoms**

Babies with harlequin ichthyosis are typically born prematurely. When they’re born, their bodies are covered in thick, platelike scales of skin. Skin tightness causes the scales to form deep cracks (fissures). The tightness also pulls the skin around your baby’s eyes and mouth, causing their eyelids and lips to turn inside out. It also pulls on the skin of your baby’s chest and abdomen, making it difficult to breathe and eat. Other symptoms may include:

* Flat nose.
* Ears fused to their head.
* Small, swollen hands and feet.
* Abnormal hearing.
* Frequent respiratory infections.
* Decreased joint mobility.
* Low body temperature.

### **causes**

A genetic variant (genetic mutation) in the *ABCA12* gene causes harlequin ichthyosis. The *ABCA12* gene gives your body instructions for making a protein that’s vital for the development of healthy skin cells. This protein has an important role in transporting fats (lipids) to the outermost layer of your skin (epidermis), producing a barrier.

If you have harlequin ichthyosis, you have abnormally small amounts of the ABCA12 protein or none. This disrupts the normal development of your epidermis, which leads to the severe symptoms that the condition produces.

You inherit harlequin ichthyosis in an autosomal recessive manner, which means you receive both copies of the affected gene — one from each parent. The parents are both carriers of the mutated gene but typically don’t show symptoms of the condition.

**complications**

Complications of harlequin ichthyosis may range in severity and include:

* Poor hair growth.
* Nail deformities.
* Itchiness.
* Heat and cold intolerance.
* Recurrent skin infections.
* Electrolyte imbalances.
* Hardening and tightening of muscles, tendons, skin and other tissues.
* Respiratory failure.
* Sudden and severe sepsis.

**Diagnosis and Tests**

Your baby’s healthcare provider can diagnose harlequin ichthyosis at birth based on their physical appearance. If you have a biological family history of the condition, your provider may recommend prenatal genetic testing to look for mutations in the *ABCA12* gene. In addition, providers can sometimes see the features of harlequin ichthyosis on ultrasound during your second and third trimester.

## **Management and Treatment**

Your baby will go to the neonatal intensive care unit (NICU) as soon as they’re born. There, your baby will stay in a high-humidity incubator to help regulate their body temperature. Nurses will care for your baby by:

* Feeding them frequently.
* Bathing them often to soften their skin and loosen skin scales.
* Rubbing their skin gently with a pumice stone, rough-textured sponge or loofah to remove scales.
* Applying moisturizers to their skin to reduce dryness and help the skin be more pliable and flexible.

If your baby has a severe case of harlequin ichthyosis, they may receive treatment with an oral retinoid called etretinate. This drug can help remove the thick, platelike scales covering their skin. It can also help reverse issues such as constricting fingers, compromised blood flow, tightened chest making it difficult to breathe and tightened face making it impossible to feed. Your baby’s healthcare provider will only use oral retinoids if your baby has severe symptoms because long-term use can cause serious adverse side effects.

Once the thick, platelike skin begins to split and peel off, your baby may be able to go home. But they’ll require continued medical care. Harlequin ichthyosis treatment involves a team of healthcare providers. This team may include:

* Neonatologists.
* Dermatologists.
* Plastic surgeons.
* Geneticists.
* Ophthalmologists.
* Otolaryngologists.
* Physical therapists.
* Orthopedists.
* Nutritionists.

This team of providers will work with your child throughout their life to provide the appropriate treatment for their condition.

**Prevention**

You can’t prevent harlequin ichthyosis because it’s a genetic condition. If you have a biological family history of the condition, you may want to talk to your healthcare provider about genetic testing or genetic counseling.

**Outlook / Prognosis**

Harlequin ichthyosis is a long-term (chronic) condition that’ll need lifetime care. It’s important to find a dermatologist who specializes in the condition so that your child can get the treatment they need throughout their life. You should also develop a daily routine to help your child control their skin issues (scales, cracking, itching), which will continue throughout their life.

In addition to skin issues, your child may develop stiff knuckles and thick fingers, which can make it hard for them to grip objects. They may also have stiff ankles and knees, making it difficult to walk. Your child may experience a delay in their physical growth and development. But their mental development should be typical.

### **What’s the outlook for this condition?**

Despite advances in the treatment of harlequin ichthyosis, many infants still die from the condition. In one study, 44% of babies born with the condition died. In the first three months of life, the most common causes of death were sepsis, respiratory failure or a combination of both.

But early introduction of oral retinoids may help increase the survival rate. In the same study, 83% of babies that were treated with oral retinoids survived.

## **Living With**

Some ways to help take care of your child include:

* **Learning about your child’s condition.** Become an expert on your child’s condition by reading reputable sources of information.
* **Keeping your child as healthy as possible.** Provide nutritious food, make sure they get enough exercise and keep up with their regular checkups.
* **Finding what works for your child.** Every child is unique, and what works for one person may not work for another. Find a daily skin care routine that fits your child’s needs.
* **Think about your child’s environment.** Forced air heat and/or cold and dry temperatures can make your child’s skin more brittle and can affect how they feel overall.
* **Have a personal skin care kit.** Put together and carry a bag containing all your child’s necessary skin care items, such as sunscreen, ointment or pain relievers.
* **Let your child take charge.** Eventually, your child will need to learn how to care for themselves. Give your child some responsibility over their skin care needs.
* **Infant care.** Skin-to-skin care with your baby and breastfeeding is encouraged.

### **What questions should I ask my child’s healthcare provider?**

If your child has harlequin ichthyosis, you probably have lots of questions. Some that you may want to ask your provider include:

* Is harlequin ichthyosis painful?
* What makes harlequin ichthyosis more severe than other kinds of ichthyosis?
* What treatments do you recommend for my child?
* How can I help my child live a normal life?
* What triggers should my child avoid,, that could make their condition worse?
* Should my child stay out of the sun?
* What complications or other health conditions should I watch out for?
* Where can I find a good support network?

**EPIDEMIOLOGY**

The incidence of harlequin ichthyosis has been reported to be 1 in 300,000 births. No evidence to support susceptibility in either males or females. No evidence of frequency seen in the disease has been identified in racial groups or sex distribution.

**DIFFERENTIAL DIAGNOSIS**

Ichthyoses represent a group of cutaneous disorders with a common finding of abnormal epidermal differentiation mostly inherited in an autosomal recessive manner. Collodion baby is a common presentation of the autosomal recessive congenital ichthyoses (ARCI), which includes congenital ichthyosiform erythroderma (CIE), lamellar ichthyosis (LI), and self-healing collodion baby (shed the initial membrane with no further skin pathology). ARCI phenotypes range from extremely fatal HI to less severe CIE and LI. Other disorders that present with "collodion baby" include Sjogren-Larsson Syndrome, trichothiodystrophy, and neutral lipid storage disease.

REFERENCE

[Ichthyosis Fetalis - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK560492/)

[Harlequin Ichthyosis (HI): Causes, Symptoms & Treatment](https://my.clevelandclinic.org/health/diseases/harlequin-ichthyosis)

**Necrobiosis lipoidica (NL)**

**Definition and Description**

Necrobiosis lipoidica (NL) is a rare inflammatory skin condition that can cause patches of skin that can sometimes develop into ulcers

NL is a rare inflammatory skin condition that typically affects the lower legs.

Although anyone can develop NL, it typically affects those with type 1 diabetes.

**Causes of Necrobiosis Lipoidica**

The exact etiology of necrobiosis lipoidica remains unknown, but several theories suggest a combination of vascular, metabolic, and immunological factors. The relationship between necrobiosis lipoidica and diabetes is particularly noteworthy, as approximately 60% of individuals with NL have diabetes.

**Vascular Factors**

One hypothesis posits that necrobiosis lipoidica results from vascular abnormalities, leading to reduced blood flow and subsequent tissue ischemia. This ischemia triggers an inflammatory response, resulting in the characteristic skin lesions.

**Metabolic Factors**

Metabolic disturbances, particularly those related to diabetes, are also considered potential contributors. Poor glycemic control can lead to microvascular complications, which may predispose individuals to NL. Additionally, hyperlipidemia and other metabolic conditions might exacerbate the disease.

**Immunological Factors**

Immunological dysregulation is another proposed mechanism. Autoimmune reactions and chronic inflammation are thought to play a role in the pathogenesis of necrobiosis lipoidica. This theory is supported by the presence of immune complexes and lymphocytic infiltration in affected tissues.

**Symptoms of Necrobiosis Lipoidica**

Recognizing the symptoms of necrobiosis lipoidica is essential for timely diagnosis and intervention. The condition typically presents with several distinct features:

**Skin Lesions**

* Appearance: Yellowish-brown, atrophic plaques with a shiny surface.
* Location: Commonly affects the shins but may also appear on the forearms, hands, and trunk.
* Progression: Lesions may enlarge over time and develop central atrophy.

**Pain and Tenderness**

* Discomfort: Lesions can be tender to touch and may cause pain, mainly if ulceration occurs.

**Ulceration**

* Complications: Ulceration is a common complication, particularly in individuals with diabetes. These ulcers can be challenging to heal and may become infected, necessitating prompt medical attention.

**Risk Factors for Necrobiosis Lipoidica**

Several risk factors increase the likelihood of developing necrobiosis lipoidica. Understanding these can help identify individuals at higher risk and implement preventive measures.

**Diabetes Mellitus**

* Prevalence: As previously mentioned, there is a significant correlation between diabetes and NL. Poor glycemic control exacerbates the risk.
* Management: Effective management of diabetes is crucial in preventing and controlling necrobiosis lipoidica.

**Gender and Age**

* Demographics: Women are more commonly affected than men, with a peak incidence in the third to fifth decades of life.

**Other Risk Factors**

* Metabolic Syndrome: Conditions such as hyperlipidemia and hypertension are also associated with an increased risk of NL.
* Trauma: Localized trauma to the skin can precipitate the development of lesions.

**Complications Associated with Necrobiosis Lipoidica**

Necrobiosis lipoidica can lead to several complications, mainly if left untreated. These complications underscore the importance of early diagnosis and effective management.

**Ulceration and Infection**

* Chronic Ulcers: Lesions may ulcerate and become chronic wounds, posing a risk of secondary infection.
* Treatment: Prompt treatment of ulcers is essential to prevent severe infections and other complications.

**Cosmetic Concerns**

* Scarring: The atrophic, shiny appearance of lesions can be disfiguring, leading to cosmetic concerns and potential psychological impact.

**Pain and Discomfort**

* Chronic Pain: Persistent pain and tenderness can significantly affect the quality of life, necessitating appropriate pain management strategies.

**Diagnosis of Necrobiosis Lipoidica**

Accurate diagnosis of necrobiosis lipoidica involves a combination of clinical evaluation and diagnostic tests. Dermatologists play a crucial role in identifying and confirming the condition.

**Clinical Examination**

* Visual Inspection: Dermatologists will examine the characteristic appearance and distribution of lesions.
* Patient History: A thorough history, including any underlying conditions such as diabetes, is essential.

**Biopsy**

* Histopathology: A skin biopsy may be performed to confirm the diagnosis. Histopathological examination reveals features such as granulomatous inflammation, necrobiosis, and lipid deposits.

**Additional Tests**

* Blood Tests: Assessing blood glucose levels and other metabolic parameters can provide insights into underlying conditions and help guide management.

**Treatment Options for Necrobiosis Lipoidica**

Treatment of necrobiosis lipoidica aims to control symptoms, prevent complications, and address underlying conditions. A multidisciplinary approach involving dermatologists, endocrinologists, and other specialists is often necessary.

**Topical Treatments**

* Corticosteroids: Topical corticosteroids can reduce inflammation and slow the progression of lesions.
* Calcineurin Inhibitors: These immunomodulatory agents may be used as an alternative to corticosteroids.

**Systemic Treatments**

* Corticosteroids: Systemic corticosteroids may be required for severe cases, although their long-term use is limited by potential side effects.
* Immunosuppressants: Drugs such as methotrexate and cyclosporine can be considered in refractory cases.

**Wound Care**

* Ulcer Management: Proper wound care, including debridement and infection control, is crucial for healing ulcers.
* Dressings: Specialized dressings may be used to promote healing and protect the affected area.

**Laser Therapy**

* Laser Treatment: Laser therapy can improve the appearance of lesions and promote healing, particularly for cosmetic concerns.

**Lifestyle Modifications**

* Diabetes Management: Effective glycemic control is paramount in managing necrobiosis lipoidica, reducing the risk of complications.
* Healthy Habits: Adopting a healthy lifestyle, including a balanced diet and regular exercise, can improve overall health and mitigate risk factors.

**EPIDEMIOLOGY**

Over half of patients with necrobiosis lipoidica have diabetes mellitus, although this association is not uniform. The incidence among people with diabetes mellitus is only 0.3% to 1.2%. Necrobiosis lipoidica precedes diabetes mellitus in up to 14%, appears simultaneously in up to 24% of cases, and occurs after diabetes mellitus is diagnosed in about 62% of cases. No definitive connection exists between the level of glycemic control and the likelihood of developing necrobiosis lipoidica.

Although necrobiosis lipoidica may present in healthy individuals with no known disease, other commonly associated conditions are thyroid disorders and inflammatory diseases, including Crohn disease, ulcerative colitis, rheumatoid arthritis, and sarcoidosis. A predominance of about 3:1 for females is noted and is especially prevalent in patients with diabetes mellitus.

The average age of onset for necrobiosis lipoidica is the third decade of life in patients with type 1 diabetes and the fourth decade of life in those with type 2 diabetes or without diabetes mellitus. Some studies demonstrate that ulceration, observed in about one-third of patients, is most prevalent in men and patients with diabetes mellitus

**DIFFERENTIAL DIAGNOSIS**

The clinical presentation of necrobiosis lipoidica is distinct, but many atypical appearances still exist, and early presentations can be hard to recognize. The following are some important considerations for differential diagnoses:

* Acute complications of sarcoidosis
* Granuloma annulare
* Hematologic malignancies
* Paraproteinemia
* Rheumatoid arthritis
* Sarcoidosis
* Xanthogranuloma
* Xanthomas

The primary differential diagnoses that require differentiation are granuloma annulare, necrobiotic xanthogranuloma, sarcoidosis, diabetic dermopathy, and lipodermatosclerosis. Granuloma annulare and sarcoidosis lesions generally do not exhibit the same degree of atrophy, telangiectasias, or yellow-brown color as necrobiosis lipoidica does. In addition, the appearance of lipids and decreased amount of mucin in necrobiosis lipoidica help differentiate this from granuloma annulare

**PROGNOSIS**

As the management of necrobiosis lipoidica is not very reassuring, its prognosis is also not very satisfactory. The disease is classically chronic, with variable progression. Squamous cell cancers can be found in more chronic lesions of necrobiosis lipoidica associated with previous trauma and ulceration.

From an aesthetic viewpoint, the prognosis of necrobiosis lipoidica is not reassuring. Treatment can help halt the expansion of lesions that tend to have a chronic course. Lesional ulcerations can result in significant morbidity, requiring comprehensive wound care. These ulcers can be painful, may become infected, or often heal with scarring.

A concerning complication is the increased incidence of squamous cell carcinoma. Necrobiosis lipoidica is often present for decades before this develops. However, it is unknown whether chronic inflammation or other factors cause this, but this should be closely monitored

**Recent Advances and Research**

Due to the rarity of necrobiosis lipoidica in the population, no uniform treatment guidelines have been established to date. Topical glucocorticosteroids (GCSs), which are the most used in the treatment of NL, had a positive effect in 40% (14/35) of uses in a multicenter study conducted by Erfurt-Berge et al. This positive effect was characterized by no increase in the number and surface area of lesions, no new ulcerations, and a reduction in active inflammation. Topical GCS can cause skin atrophy, which is why it is not advisable to apply these preparations on atrophic lesions. Other side effects of topical GCS include striae, rosacea, perioral dermatitis, acne, purpura, hirsutism, pigmentation alterations, delayed wound healing, and aggravation of cutaneous infections. Prolonged use of topical corticosteroids, particularly on a large surface area, can exacerbate hyperglycemia, which complicates glycemic control in diabetic patients. For this reason, the systemic use of GCS in patients with NL and diabetes is controversial. Calcineurin inhibitors (especially tacrolimus) are also frequently used in the topical treatment of NL. In a study by Erfurt-Berge et al., tacrolimus was found to be more effective than topical GCS, with a positive effect observed in 61.5% (8/13) of uses. Tacrolimus has an advantage over GCS as it does not cause skin atrophy and can be applied to areas with atrophic lesions and on the face. Additionally, the literature indicates that tacrolimus is particularly effective in treating NL ulcers. Other therapeutic options include phototherapy, fumaric acid esters, or dapsone. Antimalarials (chloroquine, hydroxychloroquine), cyclosporine, doxycycline, and pentoxifylline have also been used in the treatment of NL. Biological treatment is used when other therapeutic options are ineffective or there are contraindications to the use of other drugs. Attempts to use biologics have mainly involved TNF-α inhibitors (adalimumab, infliximab, and etanercept). The discovery of a key role for TNF- α in granuloma formation in mouse models provides a theoretical basis for explaining the efficacy of TNF-α inhibitors in granulomatous inflammatory diseases such as NL. Although medications in this class have had a beneficial effect on many patients, there were cases reported that did not respond to treatment or the treatment had to be discontinued due to the loss of efficacy or adverse effects. This has necessitated the search for new drugs effective in the treatment of necrobiosis lipoidica. In recent years, cases have been described of the successful use of biologics with a different molecular target—ustekinumab and secukinumab

New treatments and studies are changing how we understand and manage necrobiosis lipoidica. Doctors now have more options to help patients with this skin condition.

Biologic Agents

Biologic agents are showing promise for necrobiosis lipoidica. These drugs target specific parts of the immune system.

Tumor necrosis factor (TNF) blockers have helped some patients. They reduce inflammation in the skin.

Ustekinumab and secukinumab are two other biologics being tested. Early results look good, but more research is needed.

These drugs may work when other treatments fail. They can be expensive and have side effects.

Emerging Therapeutics

New drugs are being tested for necrobiosis lipoidica. Tapinarof is one example. It's a cream that may help reduce skin inflammation.

Researchers are also looking at light therapies and laser treatments. These may help heal skin lesions.

Combination therapies are another area of study. Using multiple treatments together might work better than one alone.

**REFERENCE**

[Necrobiosis Lipoidica - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK459318/)

[Necrobiosis Lipoidica: Causes, Symptoms, and Treatment](https://www.medicoverhospitals.in/diseases/necrobiosis-lipoidica/#:~:text=Necrobiosis%20lipoidica%20is%20characterized%20by%20yellowish-brown%20patches%20on,and%20may%20ulcerate%2C%20causing%20discomfort%20and%20potential%20complications.)

**Lamellar ichthyosis (LI)**

**Synonyms**

* congenital lamellar ichthyosis
* Collodion baby
* Collodion baby syndrome
* Ichthyoses, lamellar
* Ichthyosis, lamellar
* LI

**DEFINITION AND DESCRIPTION**

Lamellar ichthyosis (LI) is a rare genetic skin disorder that is present at birth. It is one of three genetic skin disorders called autosomal recessive congenital ichthyoses (ARCI). The other two are known as harlequin ichthyosis and congenital ichthyosiform erythroderma. All ARCI conditions are considered a clinical spectrum. There is overlap in symptoms between ARCI conditions. In LI, the body creates skin cells at a normal rate. However, they do not separate from each other at the surface of the skin the way they should. In addition, the body does not shed the skin fast enough, causing brown scales to form.

**CAUSES**

Mutations in one of many genes can cause lamellar ichthyosis. These genes provide instructions for making proteins that are found in the outermost layer of the skin (the epidermis). The skin abnormalities associated with lamellar ichthyosis disrupt the normal formation of the epidermis, resulting in impaired regulation of body temperature, water retention, and resistance to infections.

Mutations in the *TGM1* gene are responsible for approximately 90 percent of cases of lamellar ichthyosis. The *TGM1* gene provides instructions for making an enzyme called transglutaminase 1. This enzyme is involved in the formation of the cornified cell envelope, which is a structure that surrounds skin cells and helps form a protective barrier between the body and its environment. *TGM1* gene mutations lead to severely reduced or absent enzyme production, which prevents the formation of the cornified cell envelope.

Mutations in other genes associated with lamellar ichthyosis are each responsible for only a small percentage of cases. In some people with lamellar ichthyosis, the cause of the disorder is unknown. Researchers have identified multiple chromosome regions that contain genes that may be associated with lamellar ichthyosis, although the specific genes have not been identified.

## Symptoms

Many babies with LI are born with a clear, shiny, waxy layer of skin called a collodion membrane. For this reason, these babies are known as collodion babies. The membrane sheds within the first 2 weeks of life. The skin underneath the membrane is red and scaly resembling the surface of a fish.

With LI, the outer layer of skin called the epidermis cannot protect the body like the healthy epidermis can. As a result, a baby with LI may have the following health problems:

* Difficulty in feeding
* Loss of fluid (dehydration)
* Loss of balance of minerals in the body (electrolyte imbalance)
* Breathing problems
* Body temperature that is not stable
* Skin or body-wide infections

Older children and adults with LI may have these symptoms:

* Giant scales that cover most of the body
* Decreased ability to sweat, causing sensitivity to heat
* Hair loss
* Abnormal finger and toenails
* Skin of the palms and soles is thickened

**DIAGNOSIS**

Symptoms vary from person to person but there are several common signs that doctors will check to differentiate LI from other skin conditions.

For the first few days or weeks following birth, babies with LI have a “collodion membrane”, a clear but tight film which covers the baby’s skin. (This condition is also seen in other forms of ichthyosis).

The skin will have large, dark “scales” all over the body which have a “plate-like” or “fish-scale” appearance. Thickening of the palms and soles of the feet is common. There may be slight redness of the skin, but this is less prominent in LI compared to other types of ichthyoses.

Patients with LI may have nails which are curved or thickened and may resemble “sandpaper”. This is known as nail dystrophy. In LI, nails may grow either faster or slower than normal.

A small piece of skin (a skin biopsy) may be taken to check for certain proteins commonly associated with LI, such as transglutaminase 1 (TGM1). However, problems with these proteins can also be seen in other forms of ichthyosis. A skin biopsy is usually done by a dermatologist rather than a GP or pediatrician.

A blood test can also be taken (and sent to a national reference laboratory) to check for a fault in certain genes. Mutations in 11 genes are currently known to cause ARCI with TGM1 and ALOX12B being the most common. However, some people with ichthyosis will have faults in genes that are not yet known about, and this test may be negative.

**Why is Lamellar Ichthyosis sometimes misdiagnosed?**

Occasionally patients are not diagnosed for months or even years as scaly and red skin can be a symptom of many other skin conditions, such as severe eczema or immune deficiency disease. It is important that LI is considered in any persistently red baby to avoid misdiagnosis or incorrect treatment. The skin of newborns with LI can also closely resemble another form of ichthyosis called CIE, where redness of the skin tends to be more pronounced, making it difficult to distinguish between the two until later in life.

## **Treatment**

Collodion babies usually need to stay in the neonatal intensive care unit (NICU). They are placed in a high-humidity incubator. They will need extra feedings. Moisturizers need to be applied to the skin. After the collodion membrane is shed, babies can usually go home.

Lifelong care of the skin involves keeping the skin moist to minimize the thickness of the scales. Measures include:

* Moisturizers applied to the skin
* Medicines called retinoids that are taken by mouth in severe cases
* High-humidity environment
* Bathing to loosen scales

## **Possible Complications**

Babies are at risk for infection when they shed the collodion membrane.

Eye problems may occur later in life because the eyes cannot close completely.

## **Epidemiology**

Lamellar ichthyosis affects all populations, and the prevalence is less than 1 case per 300,000 individuals. Incidence in males and females is equal. The disease is present at birth and continues throughout life.

It should be noted that both autosomal recessive lamellar ichthyosis and X-linked recessive ichthyosis have been identified in a consanguineous family.

A rare phenotype of lamellar ichthyosis has been described in South Africa. The term bathing-suit ichthyosis describes the characteristic distribution of the lesions, which involve the trunk, the proximal parts of the upper limbs, the scalp, and the neck, with sparing of the central face and extremities. This form of lamellar ichthyosis is caused by temperature sensitive and non-temperature sensitive mutations in *TGM1*

## **Differential Diagnoses**

* Cornel Netherton syndrome
* Dermatologic Manifestations of Sjogren-Larsson Syndrome
* Epidermolytic Ichthyosis (Epidermolytic Hyperkeratosis or Bullous Congenital Ichthyosiform Erythroderma)
* Harlequin Ichthyosis
* Hereditary and Acquired Ichthyosis Vulgaris
* Rud syndrome
* Trichothiodystrophy
* X-Linked Ichthyosis

## **Cornelia de Lange Syndrome**

A rare genetic disorder characterized by distinctive facial features, growth delay, intellectual disability, and limb abnormalities. Skin manifestations may include hirsutism and ichthyosis-like scaling, but it is primarily a multisystem developmental disorder.

**Dermatologic Manifestations of Sjögren-Larsson Syndrome (SLS**)

SLS is a rare autosomal recessive neurocutaneous disorder caused by mutations in the *ALDH3A2* gene leading to deficiency of fatty aldehyde dehydrogenase. This enzyme defect disrupts lipid metabolism, resulting in the characteristic triad of:

* Congenital ichthyosis: Present at birth or early infancy as red, dry, scaly skin with fine scales that worsen over time, especially on the trunk, extremities, and flexural areas; the face is usually spared. Pruritus is prominent and distinguishes SLS from other ichthyoses.
* Neurological symptoms: Intellectual disability, spastic diplegia or tetraplegia, speech abnormalities, and sometimes seizures.
* Ophthalmologic signs: Photophobia and retinal changes.

Histologically, skin shows hyperkeratosis and psoriasiform epidermal hyperplasia. Management is multidisciplinary, focusing on skin hydration with emollients and keratolytics (urea, lactic acid), symptomatic neurological care, and supportive therapies. Oral retinoids may be used in severe ichthyosis.

Epi**dermolytic Ichthyosis (Epidermolytic Hyperkeratosis or Bullous Congenital Ichthyosiform Erythroderma)**

A rare inherited disorder caused by mutations in keratin genes leading to fragile skin and blistering at birth, followed by widespread hyperkeratosis and scaling. It presents with erythroderma and blistering in the neonatal period and thickened, verrucous skin later.

**Harlequin Ichthyosis**

A severe, often fatal autosomal recessive disorder caused by mutations in the *ABCA12* gene. Newborns present with thick, plate-like scales separated by deep fissures, ectropion, eclabium, and severe dehydration. Intensive neonatal care and retinoid therapy have improved survival.

**Hereditary and Acquired Ichthyosis Vulgaris**

The most common form of ichthyosis, usually inherited in an autosomal dominant pattern due to filaggrin gene mutations. It manifests as dry, scaly skin predominantly on extensor surfaces, sparing flexures and face. Acquired forms may be secondary to systemic diseases or medications.

**Rud Syndrome**

A rare neurocutaneous syndrome characterized by ichthyosis, mental retardation, hypogonadism, and sometimes epilepsy. It is poorly defined and overlaps with other ichthyosis syndromes.

**Trichothiodystrophy**

A rare autosomal recessive disorder characterized by brittle hair with low sulfur content, intellectual disability, photosensitivity, ichthyosis, and short stature. Skin findings include scaling and photosensitivity.

**X-Linked Ichthyosis**

Caused by deficiency of steroid sulfatase due to mutations or deletions on the X chromosome. It presents in male infants with generalized scaling, especially on the neck, trunk, and extensor surfaces, sparing the palms and soles. It may be associated with cryptorchidism and corneal opacities.

***RECOMMENDATION***

Topical and systemic retinoids have long been used in the treatment of ichthyoses and other disorders of cornification. Due to the need for long-term use of retinoids for these disorders, often beginning in childhood, numerous clinical concerns must be considered. Systemic retinoids have known side effects involving bone and eye. Additionally, potential psychiatric and cardiovascular effects need to be considered. Contraceptive concerns, as well as the additive cardiovascular and bone effects of systemic retinoid use with hormonal contraception must also be deliberated for patients of childbearing potential. The Pediatric Dermatology Research Alliance (PeDRA) Use of Retinoids in Ichthyosis Work Group was formed to address these issues and to establish best practices regarding the use of retinoids in ichthyoses based on available evidence and expert opinion.

**PROGNOSIS**

Many would wonder "what is the life expectancy of individuals with lamellar ichthyosis?" Do these children with this rare condition survive?

The fact is that lamellar ichthyosis is often stable over a lifetime with occasional periods of exacerbation.

The condition also causes impairment to growth due to a defective skin permeability barrier. But, lamellar ichthyosis is generally not life-threatening, and life expectancy is normal.

People with ichthyosis live relatively normal, productive lives. Before now, babies born with the most severe form of ichthyosis known as **Harlequin ichthyosis** survived just about the first few days of life.

But, with recent advancements in medicine and neonatal care, harlequin infants now survive and live quite productive and fulfilling lives. Harlequin ichthyosis life expectancy can now be said to be normal.

**REFERENCES**

<https://medlineplus.gov/genetics/condition/lamellar-ichthyosis/>

<https://www.ichthyosis.org.uk/faqs/lamellar-ichthyosis>

[Lamellar ichthyosis: Causes, symptoms, treatment, life expectancy](https://www.semichealth.com/diseases/lamellar-ichthyosis-the-skin-condition-that-causes-constant-skin-shedding)

<https://www.mountsinai.org/health-library/diseases-conditions/lamellar-ichthyosis>

**Pemphigus**

**Definition and Description**

Pemphigus is a disease that causes blistering of the skin and the inside of the mouth, nose, throat, eyes, and genitals.

Pemphigus is an autoimmune disease in which the immune system mistakenly attacks cells in the top layer of the skin (epidermis) and the mucous membranes. People with the disease produce antibodies against desmogleins, proteins that bind skin cells to one another, and less commonly other proteins in the skin. When these bonds are disrupted, skin becomes fragile, and fluid can collect between its layers, forming blisters.

There are several types of pemphigus, but the two main ones are:

Pemphigus vulgaris, which normally affects the mucous membranes, such as the inside of the mouth, and can also affect the skin.

Pemphigus foliaceus, which only affects the skin.

There is no cure for pemphigus, but in many cases, it is controllable with medications.

You are more likely to get pemphigus if you have certain risk factors. These include:

**Ethnic background.** While pemphigus occurs across ethnic and racial groups,

Some populations are at greater risk for certain types of the disease. People of Jewish (especially Ashkenazi), Indian, Southeast European, or Middle Eastern descent are more susceptible to pemphigus vulgaris. Certain populations in South America and Tunisia are more susceptible to pemphigus foliaceus.

**Geographic location.** Pemphigus vulgaris is the most common type worldwide, but as noted above, pemphigus foliaceus is more common in some places, such as certain rural regions of Brazil and Tunisia.

**Sex and age.** Women get pemphigus vulgaris slightly more frequently than men do, and the age of onset is usually between 50 and 60 years old. In some geographical areas, symptoms may begin in childhood.

**Genes.** Scientists believe that the higher frequency of the disease in certain populations is partly due to genetics. For example, evidence shows that certain variants in a family of immune system genes called HLA are linked to a higher risk of pemphigus vulgaris and pemphigus foliaceus. Other genes have also been linked to a higher risk of pemphigus. However, even in these higher risk populations, the incidence of pemphigus is still quite rare, so pemphigus is not considered an inherited disease where a parent can directly pass the disease to a child.

**Medications.** In rare cases, pemphigus has resulted from taking certain medicines, such as certain antibiotics and blood pressure medications. Medicines that contain a chemical group called a thiol have previously been linked to pemphigus.

**Cancer.** Rarely, the development of a tumor—in particular a growth in a lymph node, tonsil, or thymus gland—can trigger the disease.

## Types of Pemphigus

## There are two major forms of pemphigus, and they are categorized based on the layer of skin where the blisters form and where the blisters are found on the body. The type of antibody that attacks the skin cells also helps define the type of pemphigus.

## The two main forms of pemphigus are:

## **Pemphigus vulgaris** is the most common type in the United States. Blisters form in the mouth and other mucosal surfaces and can also involve the skin. They develop within a deep layer of the epidermis and are often painful. There is a subtype of the disease called pemphigus vegetans in which blisters form mainly in the groin and under the arms or on the scalp, where they can leave persistent sores.

## **Pemphigus foliaceus** is less common and only affects the skin. The blisters form in upper layers of the epidermis and may be itchy or painful.

## Other rare forms of pemphigus include:

## **Paraneoplastic pemphigus.** This type is characterized by sores in the mouth, particularly on the tongue and lips, but blisters or inflamed lesions usually also develop on the skin and other mucosal surfaces. Severe lung problems may occur with this type. People with this type of the disease usually have a tumor, and the disease may improve if the tumor is surgically removed.

## **IgA pemphigus.** A type of antibody called IgA causes this form. Blisters or pimple-like bumps often appear in groups or rings on the skin.

## **Drug-induced pemphigus.** Certain medicines, such as some antibiotics and blood pressure medications, as well as drugs that contain a chemical group called a thiol, may bring on pemphigus-like blisters or sores. The blisters and sores sometimes go away when you stop taking the medication.

## **Symptoms of Pemphigus**

## The main symptom of pemphigus is blistering of the skin and in some cases, the mucosal surfaces, such as the inside of the mouth, nose, throat, eyes, and genitals. The blisters are fragile and tend to burst, causing crusty sores. Blisters on skin may join, forming raw-looking areas that are prone to infection and that ooze large amounts of fluid. The symptoms can vary depending on the type of pemphigus.

## **Pemphigus vulgaris** blisters often start in the mouth but can develop on the skin later. The skin may become so fragile that it peels off by rubbing a finger on it. Mucosal surfaces such as those of the nose, throat, eyes, and genitals may also be affected. Blisters form within the deep layer of the epidermis, and they are often painful.

## **Pemphigus foliaceus** only affects the skin. Blisters often appear first on the face, scalp, chest, or upper back, but they may eventually spread to other areas of skin on the body. The affected areas of skin may become inflamed and peel off in layers or scales. The blisters form in the upper layers of the epidermis, and they may be itchy or painful.

**Pemphigus vulgaris**Enlarge image



## **Causes of Pemphigus**

## Pemphigus is an autoimmune disorder that happens when the immune system attacks healthy skin. Immune molecules called antibodies target proteins called desmoglein's, which help link neighboring skin cells to one another. When these connections are broken, skin becomes fragile and fluid can collect between layers of cells, forming blisters.

## Normally, the immune system protects the body from infection and disease. Research suggests that both genetic and environmental factors can contribute to disease onset. Something in the environment may trigger pemphigus in people who are at risk because of their genetic makeup. In rare cases, pemphigus may be caused by a tumor or by certain medications. Once the disease occurs, removing the potential triggers may or may not reverse the disease.

## **Risk factors**

## The risk of pemphigus increases if you're middle-aged or older. The condition also is more common in people of Jewish, Indian, southeast European or Middle Eastern ancestry.

## **Diagnosis of Pemphigus**

## Early diagnosis is important, so if you have blisters on the skin or in the mouth that do not go away, it is important to see a doctor as soon as you can. Your doctor may try to rule out other conditions first, since pemphigus is a rare disease. Your doctor may:

## **Take your medical history and give you a physical exam.** A dermatologist (a doctor who specializes in conditions of the skin, hair, and nails) may ask you about your medical history and look at the appearance and location of blisters. He or she may run a finger or cotton swab over the surface of your skin to see if it shears off easily.

## **Take a tissue sample.** Your doctor may take a sample from one of your blisters to:

## Examine it under the microscope to look for cell separation and to determine the layer of skin in which the cells are separated.

## Determine which antibodies attacked the skin.

## **Take a blood sample.** Blood tests can help determine the types of antibodies that are in the blood and their levels, which can help predict the severity of the disease. This blood test may also be used later on to see if treatment is working.

## **Treatment of Pemphigus**

## There is no cure for pemphigus, but treatment can control the disease in most people. The initial goal of treatment is to clear existing blisters and help prevent relapses. Treatment typically depends on the severity and stage of the disease.

## Symptoms of pemphigus may go away after many years of treatment, but most people need to continue taking medications to keep the disease under control. Treatment for pemphigus may involve the following medications:

## **Corticosteroids.** These anti-inflammatory medicines are a mainstay of treatment for pemphigus. They may be applied topically as a cream or ointment, or by mouth or injection (systemically). Most people will be prescribed systemic corticosteroids, at least initially, to bring the disease under control. Because they are potent drugs, your doctor will prescribe the lowest dose possible to achieve the desired benefit.

## **Biologic response modifiers (or biologics).** These target specific immune messages and interrupt the signal, helping to stop the immune system from attacking the skin. Rituximab is an approved biologic administered directly in the vein. It targets and depletes the immune cells that ultimately make the disease-causing antibodies.

## **Antibiotics, antivirals, and antifungal medications** to control or prevent infections.

## If the above treatments do not work or are not tolerated, other treatments may be considered. These treatments include:

## **Immunosuppressants.** Although less effective than rituximab, these are oral medications that help suppress or curb the overactive immune system and may help to lower the dose of daily steroids.

## **Plasmapheresis or immunoadsorption.** These are procedures that remove or dilute out damaging antibodies from the blood.

## **Intravenous immunoglobulin therapy.** This is an intravenous infusion of pooled antibodies from 1,000 or more healthy blood donors, which dilute out the bad antibodies and calm inflammation.

## Be sure to report any problems or side effects from medications to your doctor.

## In some cases, a person with pemphigus may need to be hospitalized to treat health problems that the disease or its treatment can cause. Widespread sores on the skin can result in dehydration or infection, and painful blisters in the mouth can make it difficult to eat. In the hospital, you may be given an IV to replace lost fluids, to get much-needed nutrition, and to treat infection.

## Who Treats Pemphigus?

## The following health care providers may diagnose and treat pemphigus:

## Dermatologists, who specialize in conditions of the skin, hair, and nails.

## Dentists, who can tell you how to take care of your gums and teeth if you have blisters in your mouth.

## Mental health professionals, who help people cope with difficulties in the home and workplace that may result from their medical conditions.

## Ophthalmologists, in cases where the eyes are affected. Ophthalmologists specialize in treating disorders and diseases of the eye.

## Otolaryngologists, if the larynx (voice box) or upper throat is affected, and visualization is necessary to ensure symptoms are due to blisters or some other factor.

## Primary care doctors, such as a family physician or internal medicine specialist, who coordinate care between the different health care providers and treat other problems as they arise.

## **When to see a doctor**

## See a healthcare professional if you have blisters that don't heal in the mouth or on the skin or genital mucous membranes.

## **Complications**

## Possible complications of pemphigus include:

* Infection of the skin.
* Infection that spreads to your bloodstream, also called sepsis. This type of infection can be life-threatening.
* Scarring and changes in skin color after the affected skin heals. This is called post inflammatory hyperpigmentation when the skin darkens and post inflammatory hypopigmentation when the skin loses color. People with brown or Black skin have a higher risk of long-term skin color changes.
* Malnutrition, because painful mouth sores make it difficult to eat.
* Side effects from the medicine used to treat pemphigus. Examples are high blood pressure and infection.
* Death, rarely, if certain types of pemphigus are left untreated.

## Living With Pemphigus

## Blisters in the mouth may make brushing and flossing your teeth painful, so talk to your dentist about ways to keep your teeth and gums healthy. Generally, gentle cleanings every 3 months are recommended. Avoid foods that irritate your mouth blisters, and do not brush the gums when disease is active as this can slough off the mucosa. Your dermatologist may recommend baths and wound dressings to help heal the skin sores and blisters.

## Pemphigus and its treatments can be debilitating and cause lost time at work, weight loss, sleep problems, and emotional distress. A mental health professional or a support group may help you cope with the disease.

## Remember to follow the recommendations of your health care providers.

## **Lifestyle and home remedies**

## Here are steps you can take to improve your skin and overall health:

* **Following wound care instructions.** Taking good care of your wounds can help prevent infection and scarring. Talk with your healthcare professional about how best to care for your wounds and control pain.
* **Wash your skin gently.** Use mild soap, rinse and apply lotion afterward.
* **Protecting your skin.** Avoid activities that may hurt the skin. And protect it from too much heat and sun, even on cool, cloudy or hazy days.
* **Avoiding certain foods.** Blisters in the mouth could be made worse by spicy, hot or crunchy foods.
* **Taking care of your teeth and gums.** Regular follow-up with a dentist is important for people with pemphigus. Talk with your dentist about how to best take care of your teeth and gums.

## **Differential Diagnoses**

* Bullous Pemphigoid
* Dermatitis Herpetiformis
* Drug-Induced Pemphigus
* Erythema Multiforme
* Familial Benign Pemphigus (Hailey-Hailey Disease)
* IgA Pemphigus
* Linear IgA Dermatosis
* Paraneoplastic Pemphigus
* Pemphigus Erythematosus
* Pemphigus Foliaceus
* Pemphigus Herpetiformis

## **Bullous Pemphigoid (BP)**

An autoimmune subepidermal blistering disease primarily affecting elderly patients, characterized by tense blisters on erythematous or normal skin, often with intense pruritus. Immunopathology shows linear deposition of IgG and C3 along the basement membrane zone. BP may rarely coexist or overlap with dermatitis herpetiformis (DH), showing mixed immunofluorescence patterns of IgG and IgA deposits. Treatment typically involves systemic corticosteroids and immunosuppressants.

**Dermatitis Herpetiformis (DH)**

A chronic autoimmune blistering disorder linked to gluten sensitivity and celiac disease. It presents with intensely pruritic, grouped vesicles and papules symmetrically distributed on extensor surfaces. Direct immunofluorescence reveals granular IgA deposits in the papillary dermis. DH may increase the risk of developing BP, and rare mixed forms with overlapping features exist. Treatment includes a gluten-free diet and dapsone.

**Drug-Induced Pemphigus**

A variant of pemphigus triggered by certain medications, presenting with flaccid blisters and erosions on skin and mucous membranes. Diagnosis is confirmed by histology and immunofluorescence showing intercellular IgG deposits. Management

## **Erythema Multiforme (EM)**

An acute, immune-mediated condition characterized by target lesions on the skin, often triggered by infections (notably herpes simplex virus) or drugs. It involves epidermal necrosis and inflammation but is distinct from autoimmune blistering diseases. Mucosal involvement may occur in EM major.

## **Familial Benign Pemphigus (Hailey-Hailey Disease)**

A genetic disorder causing recurrent blisters and erosions in intertriginous areas due to defective keratinocyte adhesion. It is not autoimmune but can mimic pemphigus clinically. Histology shows suprabasal acantholysis without antibodies.

## Ig**A Pemphigus**

A rare autoimmune blistering disease characterized by IgA autoantibodies against desmosomal proteins. It presents with vesiculopustular eruptions, often in flexural areas. Direct immunofluorescence shows intercellular IgA deposits in the epidermis.

**Linear IgA Dermatosis**

An autoimmune subepidermal blistering disease with linear IgA deposition along the basement membrane zone on immunofluorescence. It presents with tense blisters and annular or grouped vesicles, often in children (chronic bullous disease of childhood) or adults.

## **Paraneoplastic Pemphigus**

A severe autoimmune blistering disease associated with underlying neoplasia. It features painful mucosal erosions and polymorphous skin eruptions. Autoantibodies target multiple epidermal and basement membrane antigens. Prognosis is poor, and treatment focuses on managing the malignancy and immunosuppression.

## **Pemphigus Erythematosus**

A localized variant of pemphigus foliaceus with features overlapping lupus erythematosus. It is presented with crusted, scaly plaques on the face and upper trunk. Autoantibodies target desmoglein 1.

## **Pemphigus Foliaceus**

An autoimmune blistering disease with superficial blisters and erosions, primarily affecting the scalp, face, and trunk. Autoantibodies target desmoglein 1, leading to loss of cell adhesion in the upper epidermis.

## **Pemphigus Herpetiformis**

A rare variant of pemphigus combining clinical features of dermatitis herpetiformis (pruritic vesicles and plaques) with immunopathology of pemphigus (intercellular IgG deposits). It responds to corticosteroids and dapsone.

## **Epidemiology**

### United States and international statistics

Pemphigus vulgaris is uncommon in the United States, and the exact incidence and prevalence depend on the population studied.

Pemphigus vulgaris has been reported to occur worldwide. The incidence of this condition has been reported to be in the range of 0.5-3.2 cases per 100,000 population. It is higher in patients of Ashkenazi Jewish descent and those of Mediterranean origin. Few familial cases have been reported. As with endemic pemphigus, there is some evidence to suggest clustering near industrial sites.

### Age-, sex-, and race-related demographics

Although most cases of pemphigus vulgaris occur between the ages of 50 and 60 years, the range is broad, and disease onset in older individuals and in children has been described. Patients are younger at presentation in India than they are in Western countries.

The male-to-female ratio is approximately equal. In adolescence, girls are more likely to be affected than boys.

Pemphigus vulgaris affects persons of all races and ethnic groups. As noted, it is more prevalent in regions where the Jewish population is predominantand in Mediterranean regions. For example, in Jerusalem, the prevalence of pemphigus vulgaris has been estimated at 1.6 cases per 100,000 population, whereas in Connecticut, the prevalence has been reported as 0.42 cases per 100,000 population.

**Guideline**

In the updated guideline, the EADV Task Force stated that the responsibility for creating a treatment plan for patients with pemphigus should fall on an experienced dermatologist in a hospital setting or specialized center. Before developing the treatment plan, the dermatologist and all other relevant clinicians should review the medical history and conduct or review findings from a physical examination. A pemphigus diagnosis should then be made based on clinical presentation, histopathology, direct immunofluorescence examination of skin or mucosal biopsy, and serological detection of autoantibodies.

For therapeutic management of pemphigus, the primary objective is to control and heal the bullous skin and mucous lesions without substantially increasing the burden of treatment-related side effects. Dapsone, topical corticosteroids, systemic corticosteroid therapy with prednisone, or rituximab could be considered as initial treatment of mild pemphigus foliaceus (PF), despite that few of these therapies have robust data to support their use in mild PF. In addition, the guideline suggests second-line management could include rituximab or systemic corticosteroid therapy with prednisone.

The Task Force recommends tapering systemic corticosteroids during 4 to 6 months after the consolidation phase if treatment is associated with rituximab. The members of the Task Force were unable to reach a consensus on the best tapering regimen.

The updated guideline recommends clinicians inform their patients and their family members about the disease, including its clinical course and prognosis, relapse signs, and possible treatment-related adverse events. Clinicians should also recommend their patients seek out patient support groups for pemphigus.

**REFERENCES**

[Pemphigus Vulgaris Differential Diagnoses](https://emedicine.medscape.com/article/1064187-differential?form=fpf)

<https://www.dermatologyadvisor.com/news/eadv-updates-guidelines-on-management-of-pemphigus-vulgaris-and-foliaceus>

<https://www.niams.nih.gov/health-topics/pemphigus>

[Pemphigus - Symptoms and causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/pemphigus/symptoms-causes/syc-20350404)

**Raynaud’s phenomenon**

**Definition and description**

Raynaud’s phenomenon is a condition that causes the blood vessels in the extremities to narrow, restricting blood flow. The episodes or “attacks” usually affect the fingers and toes. In rare cases, attacks occur in other areas such as the ears, tongue, or nose. An attack usually happens from exposure to cold temperatures or emotional stress.

There are two types of Raynaud’s phenomenon—primary and secondary. The diagnosis between the two types is best made with a device called nailfold capillaroscopy, where the doctor looks at your skin at the bottom of your fingernails under a microscope. The primary form has no known cause, but the secondary form is related to another health issue, especially autoimmune diseases like lupus or scleroderma. The secondary form tends to be more serious and to need more aggressive treatment.

In most people, lifestyle changes such as staying warm keep symptoms under control, but in severe cases, repeated attacks lead to skin sores or gangrene (death and decay of tissue). The treatment depends on how serious the condition is and whether it is the primary or secondary form.

## Who Gets Raynaud’s Phenomenon?

## Anyone can get Raynaud’s phenomenon, but some people are more likely to have it than others. There are two types and the risk factors for each are different.

## The *primary* form of Raynaud’s phenomenon, which is of unknown cause, has been linked to:

## **Sex.** Women get it more often than men.

## **Age.** It usually occurs in people younger than age 30 and often starts in the teenage years.

## **Family history of Raynaud’s phenomenon.** People with a family member who has Raynaud's have a higher risk of getting it themselves, suggesting a genetic link.

## The *secondary* form of Raynaud’s phenomenon occurs in combination with another disease or an environmental exposure. Factors that have been linked to secondary Raynaud’s phenomenon include:

## **Diseases.** Among the most common ones are lupus, scleroderma, inflammatory myositis, rheumatoid arthritis, and Sjögren’s disease. Conditions such as certain thyroid disorders, clotting disorders, and carpal tunnel syndrome have also been linked to the secondary form.

## **Medications.** Medications used to treat high blood pressure, migraines, or attention-deficit/hyperactivity disorder may cause symptoms similar to Raynaud’s phenomenon or make underlying Raynaud’s phenomenon worse.

## **Work-related exposures.** Repeated use of vibrating machinery (such as a jackhammer), or exposure to cold temperatures or certain chemicals. A history of frostbite may result in nerve damage in the fingers and Raynaud’s phenomenon symptoms.

## Types of Raynaud’s Phenomenon

## There are two types of Raynaud’s phenomenon.

## **Primary Raynaud's phenomenon** has no known cause. It is the more common form of the condition.

## **Secondary Raynaud's phenomenon** is associated with another problem, such as a rheumatic disease like lupus or scleroderma. Factors such as exposure to cold temperatures or certain chemicals may also be linked to this form. The secondary form is less common but typically more serious than the primary form due to damage that occurs to the blood vessels.

## **Symptoms of Raynaud’s Phenomenon**

## Raynaud’s phenomenon happens when episodes or “attacks” affect certain parts of the body, especially the fingers and toes, causing them to become cold and numb, and change colors. Exposure to cold temperatures is the most common trigger, such as grabbing hold of a glass of ice water or taking something out of the freezer. Sudden changes in ambient temperature, such as when entering an air-conditioned supermarket on a warm day, can lead to an attack.

## Emotional stress, cigarette smoking, and vaping can also trigger symptoms. Parts of the body besides the fingers and toes, such as the ears or nose, may be affected as well.

## **Raynaud’s attacks.** A typical attack progresses as follows:

## The skin of the affected part of the body turns pale or white due to lack of blood flow.

## The area then turns blue and feels cold and numb, as the blood that is left in the tissue loses its oxygen.

## Finally, as you warm up and circulation returns, the area turns red and may swell, tingle, burn, or throb.

## Only one finger or toe may be affected at first; then, it may move to other fingers and toes. The thumbs are less likely to be affected than the other fingers. An attack may last a few minutes or a few hours, and the pain associated with each episode can vary.

## **Skin ulcers and gangrene.** People with severe Raynaud’s phenomenon can develop small, painful sores, especially at the tips of the fingers or toes. In rare cases, an extended episode (lasting days) of a lack of oxygen to tissues can lead to gangrene (cellular death and decay of body tissues).

## For many people, especially those with the primary form of Raynaud’s phenomenon, the symptoms are mild and not highly troublesome. People with the secondary form tend to have more severe symptoms.

## **Causes of Raynaud’s Phenomenon**

## Scientists do not know exactly why Raynaud’s phenomenon develops in some people, but they do understand how attacks happen. When a person is exposed to cold temperatures, the body tries to slow the loss of heat and maintain its temperature. To do so, blood vessels in the surface layer of the skin constrict (narrow), moving blood from vessels near the surface to those deeper in the body.

## In people with Raynaud’s phenomenon, blood vessels in the hands and feet react to cold or stress, narrowing quickly and staying constricted for a long period. This causes the skin to turn pale or white, then bluish as the blood left in the vessels becomes depleted of oxygen. Eventually, when you warm up and the vessels expand again, the skin flushes and may tingle or burn.

## Many factors, including nerve and hormonal signals, control blood flow in skin, and Raynaud’s phenomenon happens when this complex system gets disrupted. Emotional stress releases signaling molecules that cause blood vessels to narrow, which is why anxiety can trigger an attack.

## More women than men are affected by primary Raynaud’s phenomenon, suggesting that estrogen may play a role in this form. Genes may also be involved. The risk of the condition is higher in people with a relative who has it, but the specific genetic factors have not yet been definitively identified.

## In secondary Raynaud’s phenomenon, damage to the blood vessels from certain diseases, such as lupus or scleroderma, or work-related exposures are associated with the condition.

## **Diagnosis of Raynaud’s Phenomenon**

## There is no single test to diagnose Raynaud’s phenomenon. Doctors usually diagnose it based on symptoms, in particular, on a description of a typical attack upon exposure to cold. Your doctor will likely also take a medical history and perform a physical exam.

## Your doctor may perform additional tests to distinguish between the two forms of the condition. These include:

## **Nailfold capillaroscopy.** During this test your doctor uses a magnifier to look at the base of your fingernails for signs of changes in capillaries (tiny blood vessels), a sign of secondary Raynaud’s phenomenon.

## **Blood tests.** If your doctor suspects that you have the secondary form, they may order blood tests that may indicate you have a disease that has been linked to Raynaud’s phenomenon, such as lupus or scleroderma. One of the more common of these tests is the antinuclear antibody (ANA) test and a thyroid stimulating hormone (TSH) test.

## **Treatment of Raynaud’s Phenomenon**

## The goals of treatment for Raynaud’s phenomenon are to:

## Reduce how many attacks you have.

## Make attacks less severe.

## Prevent tissue damage.

## For most people with Raynaud’s phenomenon, avoiding getting cold prevents attacks and keeps symptoms under control. But if this is not enough, medications and, in some cases, surgical procedures can help.

## Secondary Raynaud’s phenomenon is more likely to be serious and to need more aggressive therapy, such as prescription medications.

## **Preventing Attacks**

## **Medications.** While there are no medications approved by the U.S. Food and Drug Administration for Raynaud’s phenomenon, medications that have been approved for other conditions are routinely used to treat it. Some medications, including stimulants, can make Raynaud’s phenomenon worse and may be stopped if they are contributing to this condition.

## **Surgery.** If you have severe Raynaud’s phenomenon, your doctor may recommend a procedure called a sympathectomy to destroy the nerves that trigger blood vessel narrowing in the affected areas. This is usually done by incision or injections. The procedure often relieves symptoms, but it may need to be repeated after a few years.

## **Treating Tissue Damage**

## In serious cases, repeated attacks can lead to skin sores or gangrene (death and decay of tissue). If this happens, the person may need to be admitted to the hospital for a few days for imaging studies and intravenous medications to rapidly improve blood flow and to treat infection. Wound care services may be needed. In rare cases, the person may need surgery to remove dead tissue and provide pain relief.

## **Who Treats Raynaud’s Phenomenon?**

## Raynaud’s phenomenon is primarily treated by:

## Rheumatologists, doctors who treat diseases of the joints, muscles, and bones. Rheumatologists are also specialists in autoimmune diseases. They treat Raynaud’s phenomenon because it sometimes occurs in association with rheumatic diseases, like lupus.

## Other specialists who may be involved in your care include:

## Cardiologists, who specialize in treating heart and blood vessel problems.

## Dermatologists, who specialize in conditions of the skin, hair, and nails.

## Mental health professionals, who can help people cope with difficulties in the home and workplace that may result from their medical conditions.

## Primary care doctors, such as family physicians or internal medicine specialists, who coordinate care between the different health care providers and treat other problems as they arise.

## Surgeons, including hand specialists, who may be orthopaedists, plastic surgeons, or vascular surgeons.

## **Living With Raynaud’s Phenomenon**

## In most people, Raynaud’s phenomenon can be controlled by making lifestyle changes. The following tips can decrease the number and severity of attacks you have.

## Keep warm. Keeping your hands and feet, as well as your entire body, warm is important. Often, it is not enough to keep your hands and feet warm; you need to keep your “core body” (chest, abdomen, and head) warm, too.

## If it is cold outside, try not to go out.

## If you go out when it is cold, dress warmly, wearing several layers of clothing. Be sure to use a hat or hood, because you lose a lot of body heat through your head. Consider heated gloves or hand warmers.

## Protect your hands with gloves when you handle cold or frozen items.

## Bring a sweater or jacket if you go to an indoor setting that may be air-conditioned.

## If you smoke, talk to your doctor about making a plan to quit. Nicotine in cigarettes and some vaping solutions can cause blood vessels to narrow, increasing the chance of an attack. Smoking also may cause permanent damage to blood vessels, which is particularly dangerous for people with Raynaud’s phenomenon.

## Some medications can bring on attacks, so talk to your doctor about those you take and before starting any new ones. Medications that can bring on attacks include:

## Decongestants that contain phenylephrine or pseudoephedrine.

## Appetite suppressants (diet pills) that contain pseudoephedrine.

## Beta blockers for high blood pressure.

## Migraine medications that contain ergotamine.

## Certain stimulant medications, such as methylphenidate, for attention deficit-hyperactivity disorder.

## Act quickly to end an attack. If an attack occurs, place your hands or feet in a warm place, such as under warm (not hot) water or under a heating pad. You can also warm your hands by whirling your arms in a windmill pattern or placing them under your armpits.

## Cope with stress. Because stress can bring on an attack, learning how to manage it is important. Meditation, deep breathing, or other relaxation techniques may help. Seek help from a mental health professional if these approaches do not work and you continue to experience high stress levels.

## Regular exercise and a healthy diet can help you deal with stress and improve blood flow.

## Pay attention to your skin. Keep your hands clean and dry, use a moisturizer regularly, and treat any cracks or sores promptly.

## **Prognosis**

## Primary Raynaud's is typically benign and does not progress or lead to tissue damage. Studies have found rates of remission (no attacks for two cold seasons, or 12 months without symptoms) between 3% and 33% after 7-14 years. The frequency and severity of symptoms fluctuate with changes in daily temperature and may lessen with increasing age.

## An underlying disorder develops in 13% of cases of the primary form. Primary Raynaud's disease may go into remission. Most patients have a stable course and over half improve over time. Secondary cases are more prone to be problematic - ulceration, scarring, or gangrene occur in 17% of people with Raynaud's disease secondary to systemic sclerosis

## **Complications**

## Severe cases may lead to digital infarction and gangrene with loss of the tissue of the finger pulp or distal phalanx. The skin may become chronically ischemic and ulcerate. The digit(s) may lose viability and require amputation in the very worst cases.

## **Epidemiology**

## The prevalence of Raynaud’s phenomenon (RP) in the general population lies between 3 and 5% in most studies. Primary RP, accounting for 80–90% of cases, is characterized by reversible vasospasm in peripheral arteries without underlying disease. In contrast, secondary RP develops in association with an underlying disorder and often involves structural vascular abnormalities and irreversible vascular occlusion. The prevalence of primary RP ranges from 2 to 20% in women and 1–12% in men depending on geographic location, the population studied, the definition of RP used, and the method of case ascertainment.

## Primary RP in women typically begins at an early age, with genetics, hormonal factors, and emotional stressors playing a potential aetiologic role. In men, RP is associated with increasing age, smoking, atherosclerotic peripheral vascular disease, and occupational factors including vibration. Cooler climates and low body weight are risk factors in both sexes.

## The prevalence of secondary RP is related to the underlying disease. Progression to secondary RP occurs in 14–37% of subjects with primary RP. Almost 99% of patients who progress to secondary RP develop an autoimmune disease, most commonly systemic sclerosis. Risk factors for progression include positive ANA, SSc-specific autoantibodies, and abnormal nailfold capillaroscopy.

## Primary RP follows a relatively benign course with minimal impact on function and quality of life. The greatest impact of secondary RP on morbidity and function arises from the complications of digital ulceration and ischemic necrosis, namely pain, infection, gangrene, and amputation, with resultant loss of hand function.

## **Differential Diagnosis**

## Anatomic syndromes that may be confused with Raynaud phenomenon include the following:

* Carpal tunnel syndrome
* Complex regional pain syndrome (reflex sympathetic dystrophy)
* Thoracic outlet syndrome

Miscellaneous circulatory syndromes that may be confused with Raynaud phenomenon include the following:

* Peripheral vascular disease
* Thromboangiitis obliterans
* Vasculitis
* Thromboembolic disease

Vasospastic syndromes that may be confused with Raynaud phenomenon include the following:

* Livedo reticularis
* Acrocyanosis
* Chilblains

Other problems to be considered in the differential diagnosis of Raynaud phenomenon include the following:

* Cryoglobulinemia, mixed or type 3, associated with hepatitis B and C
* Cryoglobulinemia, monoclonal or type I
* Dermatomyositis
* Fabry disease
* Leukemia
* Lymphoma
* *Mycoplasma* infection with cold agglutinins
* Myeloma
* Scleroderma, diffuse and localized (CREST syndrome)
* Systemic lupus erythematosus (SLE)
* Vibration injury
* Vinyl chloride exposure
* Waldenström macroglobulinemia

## **Carpal Tunnel Syndrome**

A compressive neuropathy of the median nerve at the wrist causing numbness, tingling, and pain in the thumb, index, middle, and radial half of the ring finger. It results from repetitive wrist movements, systemic diseases (e.g., diabetes), or trauma.

## **Complex Regional Pain Syndrome (Reflex Sympathetic Dystrophy)**

A chronic pain condition usually affecting a limb after injury, characterized by severe pain, swelling, skin color and temperature changes, and motor dysfunction due to abnormal sympathetic nervous system activity.

## **Thoracic Outlet Syndrome**

Compression of neurovascular structures (brachial plexus, subclavian vessels) at the thoracic outlet causing pain, numbness, weakness, and vascular symptoms in the upper limb.

## **Peripheral Vascular Disease**

Atherosclerotic or non-atherosclerotic diseases cause impaired blood flow to the limbs, leading to claudication, ischemic ulcers, and gangrene.

## **Thromboangiitis Obliterans (Buerger Disease)**

A non-atherosclerotic inflammatory occlusive disease of small and medium arteries and veins, strongly associated with tobacco use, causing ischemia, pain, and ulceration in distal extremities.

## **Vasculitis**

Inflammation of blood vessel walls causing vessel damage and tissue ischemia. It can affect vessels of various sizes with systemic and cutaneous manifestations such as palpable purpura, ulcers, and livedo reticularis.

Thromboembolic Disease

Formation of blood clots that obstruct vessels, leading to ischemia or infarction. It includes deep vein thrombosis and pulmonary embolism.

Livedo Reticularis

A mottled, net-like, reddish-blue discoloration of the skin caused by impaired blood flow or vasospasm in the cutaneous microcirculation. It may be idiopathic or associated with systemic diseases like vasculitis or antiphospholipid syndrome.

Acrocyanosis

Persistent, painless, symmetric cyanosis of the hands and feet due to vasospasm of small vessels, often aggravated by cold exposure.

Chilblains (Pernio)

Inflammatory skin lesions caused by cold exposure resulting in erythematous to violaceous papules or plaques on acral sites, often itchy or painful.

Cryoglobulinemia

A systemic vasculitis caused by the precipitation of cryoglobulins—immunoglobulins that aggregate at cold temperatures—leading to vascular occlusion and inflammation. It is classified into:

* Type I: Monoclonal cryoglobulins associated with hematologic malignancies (e.g., multiple myeloma). Symptoms include Raynaud phenomenon, digital ischemia, and necrosis.
* Types II and III (Mixed Cryoglobulinemia): Polyclonal immune complexes often linked to chronic infections (notably hepatitis C), autoimmune diseases (SLE, rheumatoid arthritis), or lymphoproliferative disorders. Clinical features include palpable purpura, livedo reticularis, arthralgia, neuropathy, renal involvement, and fatigue.

Dermatomyositis

An inflammatory myopathy with characteristic skin findings (heliotrope rash, Gottron’s papules) and muscle weakness. It is associated with systemic involvement and malignancy risk.

Fabry Disease

A rare X-linked lysosomal storage disorder causing angiokeratomas, neuropathic pain, and progressive renal and cardiac disease due to alpha-galactosidase A deficiency.

Leukemia and Lymphoma

Hematologic malignancies that can cause various cutaneous manifestations including infiltration (leukemia cutis), paraneoplastic syndromes, and vasculitis.

Mycoplasma Infection with Cold Agglutinins

Infections with *Mycoplasma pneumoniae* can induce cold agglutinin disease, causing hemolytic anemia and Raynaud-like symptoms due to antibody-mediated red cell agglutination at low temperatures.

Myeloma

A plasma cell malignancy associated with monoclonal gammopathy that can cause type I cryoglobulinemia and related vasculitis.

Scleroderma (Diffuse and Localized CREST Syndrome)

A connective tissue disease characterized by skin thickening, Raynaud phenomenon, and internal organ fibrosis. CREST stands for Calcinosis, Raynaud’s, Esophageal dysmotility, Sclerodactyly, and Telangiectasia.

Systemic Lupus Erythematosus (SLE)

A systemic autoimmune disease with diverse manifestations including malar rash, photosensitivity, vasculitis, and renal involvement.

Vibration Injury

Exposure to prolonged vibration causes vascular and neurological damage, leading to Raynaud’s phenomenon and sensory deficits.

Vinyl Chloride Exposure

Occupational exposure can cause scleroderma-like skin changes, Raynaud’s phenomenon, and increased risk of angiosarcoma.

Waldenström Macroglobulinemia

A lymphoplasmacytic lymphoma producing monoclonal IgM, which can cause hyperviscosity syndrome and type I cryoglobulinemia.

Reference

[Raynaud Phenomenon Differential Diagnoses](https://emedicine.medscape.com/article/331197-differential?form=fpf)

<https://www.niams.nih.gov/health-topics/raynauds-phenomenon/diagnosis-treatment-and-steps-to-take>

**SCLERODERMA**

**DEFINITION AND DESCRIPTION**

Scleroderma is an autoimmune disease that causes inflammation and fibrosis (thickening) in the skin and other areas of the body. When an immune response tricks tissues into thinking they are injured, it causes inflammation, and the body makes too much collagen, leading to scleroderma. Too much collagen in your skin and other tissues causes areas of tight, hard skin. Scleroderma may involve many systems in your body.

There are two major types of scleroderma:

Localized scleroderma only affects the skin and the structures directly under the skin.

Systemic scleroderma, also called systemic sclerosis, affects many systems in the body. This is the more serious type of scleroderma and can damage your blood vessels and internal organs, such as the heart, lungs, and kidneys. This subset is also divided into two additional categories called "limited" and "diffuse" which represents how much skin involvement there is in the body.

There is no cure for scleroderma. The goal of treatment is to relieve symptoms and stop the progression of the disease. Early diagnosis and ongoing monitoring are important.

### **What happens in scleroderma?**

The cause of scleroderma is unknown. However, researchers think that the immune system overreacts and causes inflammation and injury to the cells that line blood vessels. This triggers connective tissue cells, especially a cell type called fibroblasts, to make too much collagen and other proteins. The fibroblasts live longer than normal, causing a buildup of collagen in the skin and other organs, leading to some of the signs and symptoms of scleroderma. There can also be injuries to blood vessels.

## Who Gets Scleroderma?

## Anyone can get scleroderma; however, some groups have a higher risk of developing the disease. The following factors may affect your risk.

## **Sex.** Scleroderma is more common in women than in men.

## **Age.** The disease usually appears between the ages of 30 and 50 and is more common in adults than children.

## **Race.** Scleroderma can affect people of all races and ethnic groups, but the disease can affect African Americans more severely. For example:

## The disease is more common in African Americans than European Americans.

## African Americans with scleroderma develop the disease earlier when compared with other groups.

## African Americans are more likely to have more skin involvement and lung disease when compared with other groups.

## **Types of Scleroderma**

## Localized scleroderma affects the skin and underlying tissues. Localized scleroderma occurs more commonly in children but can also appear in adults. It generally appears in one or both of these patterns:

## Morphea, or patches of scleroderma that may be a half-inch or larger in diameter.

## Linear scleroderma, when the scleroderma thickening occurs in a line. This usually extends down an arm or leg, but sometimes runs down the forehead and face.

## Systemic scleroderma, sometimes called systemic sclerosis, affects your skin, tissues, blood vessels, and major organs. Doctors usually divide systemic scleroderma into two types based on the degree of skin involvement:

## Limited cutaneous scleroderma, which comes on gradually and affects the skin on your fingers, hands, face, lower arms, and legs below the knees.

## Diffuse cutaneous scleroderma, which comes on more rapidly and starts as being limited to the fingers and toes but then extends beyond the elbows and knees to the upper arms, trunk, or thighs. This type usually has more internal organ damage.

## **Symptoms of Scleroderma**

## The symptoms of scleroderma vary from person to person depending on the type of scleroderma you have.

## Localized scleroderma typically causes patches of thick, hard skin in one of two patterns.

## Morphea causes patches of skin to thicken into firm, oval-shaped areas. These areas may have a yellow, waxy appearance surrounded by a reddish or bruise-like edge. The patches may stay in one area or spread to other areas of skin. The disease usually becomes inactive after over time, but you may still have darkened patches of skin. Some people also develop fatigue (feeling tired).

## Linear scleroderma causes lines of thickened or different colored skin to run down your arm, leg, and, rarely, on the forehead.

## Systemic scleroderma, also known as systemic sclerosis, may come on quickly or gradually and may also cause problems with your internal organs in addition to the skin. Many people with this type of scleroderma have fatigue.

## Limited cutaneous scleroderma comes on gradually and usually affects skin on your fingers, hands, face, lower arms, and legs below the knees. It often causes problems with your blood vessels and esophagus. The limited form has less frequent major internal organ involvement, such as kidney disease or progressive lung disease, but it is generally milder than in the diffuse form.

## Diffuse cutaneous scleroderma comes on suddenly, usually with skin thickening on your fingers or toes. The skin thickening then spreads to the rest of your body above the elbows and/or knees. This type can damage your internal organs, such as:

## Anywhere along your digestive system.

## Your lungs.

## Your kidneys.

## Your heart.

## **Causes of Scleroderma**

## Researchers do not know the exact cause of scleroderma, but they suspect that several factors may contribute to the disease:

## **Genetic makeup.** Genes can increase the chance for certain people to develop scleroderma and play a role determining the type of scleroderma they have. You cannot inherit the disease, and it is not passed from parent to child like some genetic diseases. However, first-degree relatives of people with scleroderma are at higher risk of developing scleroderma than the general population.

## **Environment.** Researchers suspect that exposure to some environmental factors, such as some chemicals, may trigger scleroderma.

## **Immune system changes.** Abnormal immune or inflammatory activity in your body triggers cell changes that cause the production of too much collagen. In some cases, an immune reaction to developing cancer cells may trigger scleroderma.

## **Hormones.** Women develop most types of scleroderma more often than men. Researchers suspect that hormonal or immunological differences between women and men might play a part in the disease.

## **Diagnosis of Scleroderma**

## It can be difficult for doctors to diagnose scleroderma because the symptoms vary from person to person and are similar to other diseases. There is no single test to diagnose the disease; instead doctors use a combination of the following to help diagnose scleroderma. Your doctor may:

## Ask about your medical history.

## Ask about your current and past symptoms.

## Perform a physical exam.

## Your doctor may recommend additional testing such as:

## Ordering laboratory tests to check for certain antibodies that mistakenly target and react to your own tissues. Some of the antibodies may be common in people with scleroderma. However, antibodies may develop due to other factors, so a blood test alone does not diagnose scleroderma.

## Performing a skin biopsy.

## To look for problems with internal organs, such as the heart, lungs, or kidneys, your doctor may order additional testing. Early diagnosis of organ involvement helps doctors treat and manage the disease. Testing may include:

## Computerized tomography (CT), which uses a scanner to take images of the lungs and other organs.

## Echocardiogram, which uses sound waves to create moving pictures of your heart.

## Pulmonary function tests, which measure the function of the lungs.

## **Treatment of Scleroderma**

## Your treatment depends on the type of scleroderma you have, your symptoms, and which tissues and organs are affected. Treatment can help control the symptoms and limit damage.

## Your doctor may recommend medications, including:

## Anti-inflammatory medications to manage pain and reduce swelling.

## Topical creams to treat skin changes, including tightness and itching.

## Immunosuppressants, which may suppress the overactive immune system and can help control some aspects of the disease. Your doctor may prescribe oral, IV, or injectable immunosuppressants.

## Vasodilators help blood vessels dilate (widen), which may treat Raynaud’s phenomenon and some lung issues.

## In addition, your doctor may prescribe medications that are typically approved to treat other rheumatic diseases that have similar symptoms to scleroderma.

## Many people benefit from physical or occupational therapy to:

## Relieve pain.

## Improve muscle strength and mobility, including muscles in your arms, legs, and jaw.

## Teach you techniques to help with activities of daily living. For example, if hand pain and stiffness make it hard to brush your teeth, a therapist can recommend toothbrushes and devices to make flossing easier.

## Regular dental care is important because scleroderma can make your mouth dry and damage connective tissues in your mouth, speeding up tooth decay and causing your teeth to become loose. Tightening facial skin can also make your mouth opening smaller and narrower, which makes it harder to care for your teeth. Here are some ways to avoid tooth and gum problems:

## Brush and floss your teeth regularly.

## Have regular dental checkups. Contact your dentist immediately if you experience mouth sores, mouth pain, or loose teeth.

## Talk to your dentist and doctor about the best methods for you to use to keep your mouth moist.

## Use special mouthwashes or toothpastes for dry mouth. You can also talk to your doctor about medications that treat dry mouth.

## *Lung Damage*

Almost all people with systemic scleroderma have some loss of lung function. Some people develop severe lung disease, which comes in two forms:

Pulmonary fibrosis, a hardening or scarring of lung tissue because of excess collagen.

Pulmonary hypertension, high blood pressure in the artery that carries blood from the heart to the lungs.

Treatment differs for these two conditions:

Pulmonary fibrosis may be treated with medications that suppress the immune system, or medications which can help counter fibrosis.

Pulmonary hypertension may be treated with medications that dilate the blood vessels.

To help minimize lung complications, work closely with your doctor.

Watch for signs of lung disease, including fatigue, shortness of breath, dry cough, or difficulty breathing, and swollen feet. Report these symptoms to your doctor.

Follow up regularly with your doctor for evaluation of your lung function. This may include standard lung function tests, which measure your lung volumes to monitor the course of lung fibrosis. Checking for pulmonary hypertension early helps doctors manage and treat the condition, even before you may notice symptoms.

Get regular flu and pneumonia vaccines as recommended by your doctor, especially if you are taking immune-suppressing medications or have lung disease.

*Heart Problems*

Some people may develop complications that cause heart problems, including:

Cardiomyopathy, scarring and weakening of the heart.

Myocarditis, inflamed heart muscle.

Arrhythmia, abnormal heartbeat.

Treatments for heart complications can range from medications to surgery and vary depending on the nature of the condition.

*Kidney Problems*

Renal crisis is uncommon but can be serious for people with systemic scleroderma. Renal crisis happens when blood pressure levels rise suddenly to dangerous levels, which can quickly lead to kidney failure. Side effects of certain medications, such as corticosteroids, can also trigger renal crisis. It is important that you and your doctor work together to monitor your blood pressure, including:

Check your blood pressure regularly, and let your doctor know if you have any new or different symptoms such as a headache or shortness of breath. If your blood pressure is higher than usual, call your doctor right away.

If you have kidney problems, remember to take your medications as prescribed. In the past several decades, medications known as ACE (angiotensin-converting enzyme) inhibitors have made scleroderma-related kidney failure a less threatening problem than it used to be.

## **Who Treats Scleroderma?**

## Most people will see a rheumatologist for scleroderma treatment. A rheumatologist is a doctor who specializes in rheumatic diseases such as arthritis and other inflammatory or autoimmune disorders. Dermatologists, who specialize in conditions of the skin, hair, and nails, may also play an important role in treating the disease, particularly for people with localized scleroderma.

## Because scleroderma can affect many different organs and organ systems, you may have several different doctors providing your care. These health care providers may include:

## Cardiologists, who specialize in treating diseases of the heart and blood vessels.

## Dental providers, who can treat complications from the thickening of tissues of the mouth and face.

## Gastroenterologists, who treat digestive problems.

## Mental health professionals, who provide counseling and treat mental health disorders such as depression and anxiety.

## Nephrologists, who treat kidney disease.

## Occupational therapists, who teach how to safely perform activities of daily living.

## Orthopedists, who treat and perform surgery for bone and joint diseases or injuries.

## Primary care providers, including physicians, nurse practitioners, and physician assistants.

## Physical therapists, who teach ways to build muscle strength.

## Pulmonologists, who treat lung disease and problems.

## Speech-language pathologists, who specialize in the treatment of speech, communication, and swallowing disorders.

## **Complications**

## Scleroderma complications range from mild to serious and can affect the:

## **Fingertips.** In systemic sclerosis, Raynaud's phenomenon can become so severe that the restricted blood flow permanently damages the tissue at the fingertips, causing pits or skin sores. In some people, the tissue on the fingertips may die.

## **Lungs.** Scarring of lung tissue can impact the ability to breathe and tolerance for exercise. High blood pressure in the arteries to the lungs also may happen.

## **Kidneys.** A serious kidney complication, called scleroderma renal crisis, involves a sudden increase in blood pressure and rapid kidney failure. Prompt treatment of this condition is important to preserve kidney function.

## **Heart.** Scarring of heart tissue increases the risk of irregular heartbeats and heart failure. Scleroderma also can cause inflammation of the sac surrounding the heart.

## **Teeth.** Serious tightening of facial skin can cause the mouth to become smaller and narrower. This may make it hard to brush the teeth or to have them professionally cleaned or restored. People who have scleroderma often don't make typical amounts of saliva, so the risk of dental decay increases even more.

## **Digestive system.** Digestive complications of scleroderma can include heartburn and difficulty swallowing. Scleroderma also can cause bouts of cramps, bloating, constipation or diarrhea. Some people who have scleroderma also may have problems absorbing nutrients due to overgrowth of bacteria in the intestine.

## **Joints.** The skin over joints can become so tight that it restricts flexibility and movement, particularly in the hands.

## Living With Scleroderma

## Depending on the type of scleroderma you have and your symptoms, living with the disease may be hard. To help, try to take an active part in treating your scleroderma. The following tips and suggestions may help.

## Keep warm. Your body regulates its temperature through the skin. So, dress in layers, wear gloves and socks, and avoid cold rooms and weather when possible.

## Try to avoid cold or wet environments that may trigger Raynaud’s phenomenon symptoms.

## If you smoke, quit. Nicotine and smoking cause blood vessels to contract, which can make some symptoms worse and cause lung problems.

## Apply sunscreen before you go outdoors to protect against further damage from the sun’s rays.

## Use moisturizers on your skin to help lessen stiffness.

## Use humidifiers to moisten the air in your home in colder winter climates. Clean humidifiers often to stop bacteria from growing in the water.

## Avoid hot baths and showers, as hot water dries the skin.

## Avoid harsh soaps, household cleaners, and caustic chemicals. Wear rubber gloves if you use such products.

## Exercise regularly. Exercise, especially swimming, stimulates blood circulation to affected areas.

## Visit the dentist regularly for check-ups.

## Reach out to online and community support groups.

## Keep the lines of communication open. Talk to your family and friends to help them understand the disease.

## Talk to a mental health professional for help with coping with a chronic illness.

## Some types of scleroderma can affect parts of the digestive system. Doctors may prescribe heartburn, constipation, and motility medications to help manage these symptoms. Here are some tips to help if you have digestive symptoms:

## Eat small, frequent meals.

## After meals, stay upright for 3 hours. Try to avoid reclining or slouching after eating.

## Eat moist, soft foods, and chew them well. If you have difficulty swallowing or if your body doesn’t absorb nutrients properly, your doctor may prescribe a special diet.

## Drink less alcohol and caffeine.

## Stay hydrated.

## When it is time to sleep, raise the head of your bed with blocks or use a wedge pillow. Using several pillows is not as helpful as raising the head of the bed by using blocks or special wedges.

## **DIFFERENTIAL DIAGNOSIS**

## The following disorders may present clinical similarities with systemic sclerosis (“scleroderma mimics”) and need to be included in the differential diagnosis:

* Nephrogenic Systemic Fibrosis
* Eosinophilic Fasciitis
* [Eosinophilia-Myalgia Syndrome](https://emedicine.medscape.com/article/329614-overview)
* Graft Versus Host Disease
* Reflex Sympathetic Dystrophy
* Generalized morphea
* Diabetic cheiroarthropathy
* Porphyria cutanea tarda
* Morphea
* Linear scleroderma
* Radiation exposure
* Scleromyxedema (generalized lichen myxedematosus)
* Scleredema adultorum of Buschke
* Scleredema diabeticorum

## **Nephrogenic Systemic Fibrosis (NSF)**

A rare, progressive fibrosing disorder occurring almost exclusively in patients with advanced kidney disease, especially after exposure to certain gadolinium-based contrast agents (GBCAs) used in MRI. NSF is characterized by thickening, hardening, and darkening of the skin, primarily on the extremities and trunk, sparing the face. Early symptoms include swelling, redness, itching, and pain. Over time, skin becomes woody and may develop an orange-peel (peau d’orange) texture, leading to joint contractures and severe disability. Internal organs such as muscles, lungs, heart, and diaphragm can also be involved. There is no consistently effective cure; treatment focuses on improving renal function (dialysis or transplantation), physical therapy to maintain mobility, and symptomatic pain control. Experimental therapies like extracorporeal photopheresis, imatinib, pentoxifylline, sodium thiosulfate, and IV immunoglobulin have shown limited and variable success. Early recognition and prevention by avoiding high-risk GBCAs in renal impairment are critical.

**Eosinophilic Fasciitis**

A rare inflammatory disorder causing symmetrical swelling, thickening, and induration of the skin and fascia, often with peripheral eosinophilia. It presents with painful, woody induration of limbs, sparing the hands and feet, and may cause joint contractures. Treatment involves systemic corticosteroids and immunosuppressants.

**Eosinophilia-Myalgia Syndrom**e

A multisystem disorder associated with elevated eosinophils and severe muscle pain, often linked to ingestion of contaminated tryptophan supplements. Skin findings include edema, rash, and fibrosis.

**Graft Versus Host Disease (GVH**D)

A complication of allogeneic stem cell transplantation where donor immune cells attack host tissues, including skin. Cutaneous GVHD ranges from maculopapular rash to sclerodermoid changes resembling morphea or systemic sclerosis.

**Reflex Sympathetic Dystrophy (Complex Regional Pain Syndrome**)

A chronic pain syndrome following injury, characterized by pain, swelling, skin color and temperature changes, and limited mobility due to abnormal sympathetic nervous system activity.

**Generalized Morphea**

A localized scleroderma subtype with widespread skin thickening and fibrosis but without internal organ involvement. Presents with multiple indurated plaques or patches.

**Diabetic Cheiroarthropathy**

A diabetic complication causing thickened, tight skin and limited joint mobility, mainly affecting the hands.

**Porphyria Cutanea Tarda (PCT)**

A disorder of heme metabolism causing photosensitivity, blistering, and skin fragility on sun-exposed areas, often with hyperpigmentation and hypertrichosis.

**Morphea (Localized Scleroderma)**

A fibrosing skin disorder with circumscribed plaques of thickened, hardened skin. It may be limited or linear and can cause cosmetic and functional impairment.

**Linear Scleroderma**

A form of morphea presenting as linear bands of skin thickening and fibrosis, often on limbs or face, potentially causing growth defects in children.

**Radiation Exposure**

Can cause localized skin fibrosis, atrophy, and pigmentary changes as late effects of radiotherapy.

**Scleromyxedema (Generalized Lichen Myxedematosus)**

A rare systemic disorder characterized by widespread papular and sclerodermoid skin thickening due to mucin deposition, often associated with monoclonal gammopathy.

**Scleredema Adultorum of Buschke**

A skin condition with non-pitting woody induration and thickening of the upper back, neck, and face, often following infection or associated with diabetes.

Scleredema Diabeticorum

A variant of scleredema occurring in diabetic patients, presenting with thickened, indurated skin over the upper back and neck, causing stiffness.

**EPIDEMIOLOGY**

Global trends of scleroderma demonstrate greater prevalence of SSc in European, North, and South American patients compared with East Asian patients. However, the greatest prevalence (47 in 100 000), was found among the indigenous peoples in Canada. Phenotypical differences exist depending on the age of presentation with greater internal organ involvement and disease acceleration present in older patients. Sex differences include greater severity of disease expression, relative prevalence of diffuse cutaneous SSc, and organ involvement in males versus females. New studies conflict with previous data reporting a greater proportion of pulmonary arterial hypertension in females. Furthermore, the effect of low median household income is demonstrated as a factor increasing risk of death in SSc patients.

Understanding the epidemiological factors in SSc enables patient care through patient classification, prognostication, and monitoring. Future research may emphasize enrichment of SSc patients in randomized trials who are more likely to progress or be treatment responsive, focused screening, and personalized patient care through the creation and validation of new SSc criteria and subsets.

**REFERENCES**

https://www.niams.nih.gov/health-topics/scleroderma/diagnosis-treatment-and-steps-to-take

[Scleroderma - Symptoms and causes - Mayo Clinic](https://www.mayoclinic.org/diseases-conditions/scleroderma/symptoms-causes/syc-20351952)

# **Nails**

Common Diseases Associated with Nails

Nail psoriasis

Brittle splitting nails

Onychogryphosis

onchogryptosis

onychmycosis

Paronychia

Leuconychia

Furrows and ridges

Splinter hemorrhage

**Nail psoriasis**

Other names: psoriasis

## **Definition and description**

Nail psoriasis is an autoimmune disease that causes your skin cells to reproduce quickly. It’s a type of psoriasis that affects the nails on your fingers and toes. Nail psoriasis typically appears along with a psoriatic rash on other parts of your body. Nail psoriasis alters the way your toenails and fingernails look. They may get thick, develop pinprick holes, and change color or shape. They also can feel tender and hurt. Nail psoriasis isn’t contagious. You can’t spread nail psoriasis to another person through skin-to-skin contact.

### **Causes**

Nail psoriasis is an autoimmune disease. Your immune system overreacts, which leads to new skin cells growing too fast. Nail psoriasis is an immune system problem. Typically, new skin cells grow every 28 to 30 days. However, in people with psoriasis, new cells grow and move to the skin surface every three to four days, which creates a skin rash. In some people with psoriasis, it affects their nails in addition to their skin or other parts of their bodies.

### **Risk Factor**

People living with psoriasis may develop symptoms. It occurs when psoriasis affects the skin of the nail bed or near the nail beds.

## **Signs and Symptoms**

Symptoms of nail psoriasis include:

* **Discoloration**: The skin underneath your nails (nail bed) may change colors. These changes, called salmon patches or oil drop spots, may look yellow, red, pink or brown.
* **Pitting**: Your nails may develop dents or pits (cupuliform depressions). They can be as small as the tip of a pin (0.4 millimeters) or as large as the tip of a crayon (2 millimeters), and they can be shallow or deep. You may only have one or two pits on your nails, or you may have more than 10 per nail.
* **Changes in nail structure**: You may develop grooves that run horizontally across your nails (Beau’s lines). Your nails may grow so thin that they start to crumble. The thick layer of skin underneath the tip of your nail may also start to peel and slowly separate from the nail bed (onycholysis), which may lead to the development of nail fungus.
* Debris buildup. Chalky white material can gather under your nail, causing it to lift away from the skin. This can be painful.
* Thickening. About a third of people with nail psoriasis can also get a fungal infection that can cause your nails to get thick. They may also get brittle and break.

Some of these symptoms may result in discomfort, tenderness or pain that can affect your comfort or ability to stand, walk or use your hands.

## **Diagnosis methods**

Your healthcare provider can typically diagnose nail psoriasis after a physical exam. They’ll examine your affected areas to look for common signs of nail psoriasis. They’ll also ask about your symptoms and your family history.

Once your healthcare provider has diagnosed nail psoriasis, they may use the nail psoriasis severity index (NAPSI) to grade its severity. The NAPSI uses imaginary lines to divide your nail into four even sections (quadrants). Your nails receive a 0-4 score based on the presence of any nail psoriasis symptoms in each of the quadrants. Your healthcare provider will then add the scores together. A low number means your nail psoriasis is mild, and a high number means your nail psoriasis is more severe.  
 *People with nail psoriasis may develop a number of symptoms. Providers use NAPSI to determine the severity of the condition.*

If there’s any doubt about your symptoms, your healthcare provider may order a potassium hydroxide (KOH) preparation or fungal culture to rule out a fungus as the cause of your symptoms. If those test results aren’t clear, a fungus test known as a periodic acid-Schiff (PAS) stain can also determine the presence of a fungus

## **Treatment Option**

There isn’t a cure for nail psoriasis. It’s a long-lasting (chronic) condition, which means flare-ups can occur throughout your life. You may have flare-ups and times when the symptoms go away (remission). Treatments can provide relief for your symptoms.

Mild nail disease which isn't causing discomfort does not need any treatment. If the nail disease is severe and causing problems, then your doctor may refer you to see a skin specialist for advice and treatment. Unfortunately, treatment of psoriatic nail disease is difficult and not always successful.

Nail psoriasis is often resistant to some treatment options, so it can be challenging to manage without standard treatment. You and your healthcare provider may have to explore different treatment options, including:

* **Corticosteroids**: Your healthcare provider may prescribe a corticosteroid cream, ointment or nail polish. For corticosteroids to effectively treat your nail psoriasis, they must reach the nail bed and the area of your fingers where the nail starts to grow (nail matrix), which can be difficult. You may have to apply the medication up to twice a day, and you may not see noticeable improvement until at least four to six months.
* **Medicine injections**: In more serious cases of nail psoriasis, your healthcare provider may use a thin needle to inject medicine into the skin around your nails. These medicines may include etanercept, adalimumab or ustekinumab.
* **Oral medicines**: In more serious cases of nail psoriasis, your healthcare provider may prescribe liquid medicines or pills or tablets that you swallow with water. These medicines may include methotrexate, cyclosporine or apremilast. Methotrexate: **Drugs that target specific parts of your immune system.** You may hear your doctor call these "biologics." They are given as a shot under the skin, in a pill, or through an IV. Some examples are:
  + TNF-alpha inhibitors:
  + Adalimumab (Humira)

* **Phototherapy**: Phototherapy uses ultraviolet light from special lamps. Your healthcare provider may use a drug called psoralen combined with ultraviolet A (PUVA) or ultraviolet B (UVB). The ultraviolet light waves in phototherapy can help certain skin and nail disorders, including nail psoriasis.
* **Laser therapy**: Your healthcare provider may use a pulsed dye laser (PDL) to target the blood vessels under your nails, which may reduce the severity of nail psoriasis. Laser therapy treatments usually occur every six months.

There are several home remedies and tips that can help manage the symptoms of nail psoriasis.

While home remedies are safe for most people, it’s a good idea to check with your healthcare provider before trying some of the following options. You may be at risk of developing an allergic reaction.

* **Aloe vera**: Aloe vera gel has anti-inflammatory properties that may relieve the symptoms of nail psoriasis. Apply aloe vera gel to your nails and the surrounding skin up to several times a day, including before bed.
* **Capsaicin**: Capsaicin is a chemical compound naturally found in hot peppers that gives peppers their spicy taste. Capsaicin creams or ointments may relieve the symptoms of nail psoriasis. However, be careful not to touch or rub your eyes after applying it, because it can cause eye irritation.
* **Dead Sea salt**: Dead Sea salt comes from the Dead Sea in southwest Asia, and it can provide relief from nail psoriasis symptoms. Add Dead Sea salt to a bowl of warm water — around 95 degrees Fahrenheit (35 degrees Celsius) — and soak your nails for at least 10 minutes. You may have to soak your nails several times per week.
* **Turmeric**: Turmeric is a spice that contains a chemical called curcumin. Studies show that curcumin has anti-inflammatory and anti-fungal properties that may relieve the symptoms of nail psoriasis. You may add turmeric as a spice to foods in your diet, or you may take it as a tablet or pill in specific dosages.

It’s also a good idea to:

* Wash your hands and nails regularly to prevent an infection.
* Moisturize your nails and the skin around your nails.
* Keep your nails trimmed short.
* Apply a nail hardener polish.
* Cut off hangnails.
* Wear gloves when doing activities that may damage or dry out your nails or the skin around your nails, like washing the dishes, playing sports or working outside.

If your nail psoriasis makes you feel self-conscious or embarrassed, you may gently buff your nails and apply nail polish to hide any pits or discoloration and improve your nails’ appearance. However, avoid using fake nails. Fake nails can damage your actual nails.

## **Preventive Tips**

There isn’t any way to prevent nail psoriasis. If you have nail psoriasis, it may come and go throughout your life. Treatments can reduce symptoms, even in people with severe nail psoriasis.

Good nail care is the best way to treat nail psoriasis. Try these prevention tips:

* Keep your nails trimmed short.
* Use a nail file to keep nail edges smooth.
* Wear gloves to clean and do other work with your hands.
* Moisturize your nails and cuticles every day and after they've been in contact with water.
* Wear comfortable shoes with enough room for your toes.

## **Prognosis**

Nail psoriasis may flare up and go into remission throughout your life. However, it can generally be well managed with treatment.

Nail psoriasis can be difficult to treat and there is no cure. It doesn't usually grow out without treatment, so can continue to cause problems. The appearance of the affected nails can also sometimes cause distress.

The treatment of severe psoriatic nail disease is now improving with modern medicines.

Psoriatic nail disease can also be mild, not needing any treatment, and able to be hidden with nail varnish.

## **Possible Complications**

If you have nail psoriasis, you may be at a higher risk of:

* Diabetes.
* Heart attack.
* High cholesterol.
* Obesity.
* Stroke.
* **Depression**

## **Epidemiology**

Based on a review of the literature, the prevalence of nail psoriasis ranges between 4.2% (8) and 69% (9) of all patients suffering from psoriasis. Nail psoriasis may occur with the involvement of the skin, or it may occur alone, being the only symptom of psoriasis. Nail psoriasis is not only a problem of an aesthetic nature but can also restrict manual dexterity. The nail disease may be acute or chronic, with varied severity. There may be involvement of only a single nail or of all nails associated with severe nail destruction or loss

## **Reference**

<https://iris.who.int/bitstream/handle/10665/204417/9789241565189_eng.pdf.psoriasis?sequence=1>

<https://my.clevelandclinic.org/health/diseases/22841-nail-psoriasis#management-and-treatment>

<https://www.webmd.com/skin-problems-and-treatments/psoriasis/nail-psoriasis#1-4>

**Brittle nail syndrome**

Other names: Brittle splitting nail, onychorrhexis, onychoschizia,

## **Definition and description**

Brittle nail syndrome is described as a constellation of nail abnormalities including onychorrhexis and/or onychoschizia that collectively contribute to increased fragility of the nail plate.Onychorrhexis is classically characterized by longitudinal ridging of the nail.It has been cited as the ungual alteration most associated with patient perception of nail fragility.Onychoschizia refers to lamellar splitting of the distal free edge portion of the nail plate.It may also include breaks of the lateral edges causing transverse splitting.

Onychorrhexis is a condition that affects your nails, causing them to develop ridges and splitting. These ridges often look like thin grooves running vertically along the length of your nail. Your nails may also become brittle, break easily, and split at the edges.

Onychorrhexis involves the nail matrix, which is responsible for making your nails grow. Issues with making skin cells and a skin protein called keratin can change how your nails grow, leading to onychorrhexis.

## **Causes and Risk Factors**

Brittle nail syndrome classically develops gradually. Patients may disclose that their nail abnormalities were initially mild and worsened over time, causing additional pain or discomfort. The brittle nail findings may be associated with the onset of systemic disease, in certain cases.

Most of the time, onychorrhexis isn’t concerning and is just a cosmetic annoyance. But it can also be a sign of another health problem. Conditions that can cause onychorrhexis include:

**Hand washing.** Frequent hand washing can protect you from illness and help you stay healthy. But it does a number on your nails, which lose water faster than skin does. The constant washing and sanitizing can cause nails to dry out and become brittle.

**Aging.** As you get older, your nails can naturally become ridged, dry, brittle, or thick. This is a normal part of the aging process and one of the most common causes of onychorrhexis.

**Heart and circulatory diseases.** Your blood delivers nutrients and oxygen to tissues and cells. If you have problems with your heart, lungs, blood vessels, or oxygen circulation, you might not have enough nutrient delivery. This can affect your nails.‌

**Nutrient deficiencies.** If your diet doesn't include enough iron, protein, or folic acid, you can have nail growth problems. This may be caused by a diet that isn't well balanced or by malnutrition from bulimia or other eating disorders.

**Systemic diseases.** Hypothyroidism slows down your metabolism. This can cause dry, brittle, and ridged nails. Liver disease and chronic kidney disease can also cause onychorrhexis.

**Nail syndromes.** Witkop syndrome is a hereditary genetic disease that leads to missing teeth, vertical ridging in fingernails, and toenail koilonychia, which causes indented nails called spoon nails.

**Rheumatology diseases.** Diseases that affect the joints and cause deformities can cause problems with your nails. These diseases include gout, rheumatoid arthritis, osteoarthritis, and systemic sclerosis.

**Other diseases.** Systemic amyloidosis is a rare disease that causes amyloid protein to build up in your organs and tissues. This can affect your nails.

**Nail trauma.** Injury to your nails, picking at your nails, or strong chemicals can damage your nail matrix, affecting how your nails grow.

**Chemotherapy.** If you’re currently undergoing cancer treatment, brittle nails may be a side effect. Speak to your oncologist — they can give you advice for preserving your nails.

**Length and cosmetic use** you’re more likely to experience split, cracked and broken nails if you grow your nails longer and regularly use nail cosmetics than if you keep your nails short.

## **Signs and Symptoms**

The main symptom of onychorrhexis is vertical ridges in your nail. Rather than having a smooth nail, you might have nails with vertical grooves that feel bumpy. You may also have:

* Brittle nails that break easily
* Splitting nails
* Single or multiple ridges

## **Diagnosis Method**

### **Approach Considerations**

A thorough review of systems (ROS) and a review of medical history and medications should direct subsequent workup to assess for potential systemic causes.

Examination of all 20 nails is recommended. Close inspection of the nail plate, lunula, and proximal, distal, and lateral nail folds is essential. The clinician should make note of any periungual scale, erythema, or other cutaneous findings that might indicate an underlying primary dermatologic disorder. The presence of increased longitudinal or transverse nail curvature and onycholysis should be assessed. Nailfold capillaroscopy should be performed to look for irregularities in the capillaries of the proximal nail folds, which may indicate an autoimmune connective-tissue disorder. Signs of cyanosis or ischemia suggest that circulatory insufficiency may play a role in the nail abnormalities.

### **Physical Examination**

The diagnosis of brittle nail syndrome is established based on patient history and physical examination findings.

Classic physical examination findings for onychorrhexis include shallow, parallel longitudinal furrows of the nail, often leading to distal longitudinal breakage along a furrow. The furrows may even look as though they have been scratched by sandpaper.The physical examination for onychoschizia likely will reveal transverse lamellar splitting of the distal free edge portion of the nail plate.

### 

### **Laboratory Studies**

Depending on the clinical severity and pertinent positives on ROS, it may be important to collect bloodwork to investigate further. In a brittle nail workup, physicians may be inclined to order thyroid studies, an erythrocyte sedimentation rate (marker of acute inflammation), complete blood cell counts, a comprehensive metabolic panel (which includes glucose, electrolytes, and markers of renal and hepatic function), antinuclear antibody titers, and iron (iron deficiency), ferritin, and zinc levels. If onychomycosis is suspected, nail plate and subungual debris samples should be sent for fungal culture, periodic acid-Schiff staining, and/or molecular testing.

### **Histologic Findings**

The histopathology for onychorrhexis can be variable depending on the underlying cause. Nail plate thinning seen in onychorrhexis is caused by a shortening of the nail matrix length. However, a biopsy is not always performed, as onychorrhexis is more often a clinical diagnosis. When onychorrhexis is associated with a dermatologic disorder, biopsy of the nail matrix reveals typical histology for that disorder (e.g., lichen planus, sarcoidosis, amyloidosis). In one study,nail bed biopsy of age-related onychorrhexis revealed lichen planus–like changes including hyperkeratosis, hypergranulosis, and hydropic degeneration of the basal cell layer with necrotic keratinocytes. Epidermal thickening, papillomatous change, and bending of rete ridges was also noted.

## **Treatment Options**

### **Approach Considerations**

It is important to exclude the possibility of damage to the nail matrix from long-term arsenic exposure, disorders of the microcirculation (ie, arteriosclerosis, Raynaud disease), and disorders of oxygenation (ie, anemia, polycythemia vera, sarcoidosis).It is also vital to rule out systemic diseases, exogenous insults, and primary inflammatory dermatologic conditions impacting the nail matrix.

Treatment of Brittle Nail Syndrome (Medical summary)

Medical therapy should be strongly considered when brittle nail syndrome is suspected to have developed secondary to an underlying systemic or dermatologic disease. Otherwise, the level of medical therapy is guided by the desires of the patient and impact on quality of life.

### **Biotin**

Biotin is a water-soluble B vitamin commonly found in egg yolk, cereal, peanuts, walnuts, and milk and may be used to treat symptoms of brittle nail syndrome.The majority of studies investigating the impact of biotin on brittle nails have been small and patients were successfully treated with a dose of 2.5 mg/day.Doses ranging from 2.5-10 mg/day have been suggested to be effective. Additional studies with larger sample sizes are needed to elucidate the magnitude of its efficacy. Of importance, oral biotin supplementation has been implicated in interference with blood testing, including tests measuring levels of troponin, thyroid function, and prolactin.It may also impact pregnancy testing.Therefore, it is important that patients disclose to their healthcare providers whether they are on biotin supplementation in order to ensure the most optimal clinical care. Patients taking typical-dose biotin supplements should withhold their dose at least 8 hours prior to any laboratory tests.

#### **Biotin (Appearex, Coenzyme R,** Nail**-ex)**

Biotin supplementation may help to treat brittle nails. Biotin may promote nail growth by aiding in the metabolism of amino acids responsible for building proteins associated with nail growth. It is known to function as a coenzyme of metabolic processes.

### **Hydroxypropyl chitosan**

In patients with brittle nail syndrome, hydroxypropyl chitosan–based nail lacquer has been associated with a clinically relevant improvement of nail appearance. In one study, the use of oral biotin in addition to the nail lacquer prompted a synergistic effect associated with even further clinical improvement.

### ***Poly-ureaurethane (Nuvail)***

Poly-ureaurethane is a biocompatible, polymeric solution that forms a uniform film when applied to the nail; it adheres to the nail surface, preventing direct abrasion and friction on the nail surface; it also provides protection against moisture.

## **Preventive Tips**

To prevent brittle nails, it is helpful to follow a diet that is well-balanced in vitamins and minerals that are beneficial for the nails and skin. Direct trauma to the nails should be avoided. Patients should be advised to wear cotton gloves whenever involved in intensive manual work. It is recommended that patients take caution with the overuse of nail polish remover, as it can be drying to the nails. Other options for prevention include keeping nails short to reduce the surface area accessible for weakening and avoiding vigorous or frequent manicures and long-term, repetitive use of artificial nails.

Long-Term Monitoring

Nail disorders can be difficult to treat and require time to see promising results. Successful resolution of brittle nails may even require multiple treatment strategies.It is important that healthcare providers and patients collaborate to establish an optimal management plan.

Most of the causes of brittle nails are beyond your control — you need to keep washing your hands and there’s no way to turn back the clock. But don’t worry, we have plenty of solutions … on hand.

**Don’t bite your nails:** There are several recommendations for keeping your nails from splitting or cracking, but this pearl of wisdom can also keep you healthy: Keep your fingers away from your mouth. If you tend to bite your nails, then keep them short so you aren’t tempted to chew on dirty nails all day long.

**Coat and condition them:** At bedtime, apply heavy hand cream. During the day, moisturize in between exposure to water or hand sanitizer. You can also apply a nail conditioner with lanolin a few times a day. Avoid nail conditioners that contain alcohol, because that will further dry out your nail.

**Try nail slugging:** If you’re looking to do some extreme moisturizing, nail slugging — a trendy new technique that’s gone viral on social media — might be right up your alley. Nail slugging is the quirky younger sister of face slugging, a practice that’s been popular for several years now.

Slugging, as the name suggests, is a slimy process. After applying skin care products, you cover your hands in a layer of petroleum jelly (or a similarly occlusive product, like an emollient) before going to sleep. The goop traps all that product on your skin, allowing it to more fully seep in.

You could technically slug your nails — or your face for that matter — during the day, but most people would rather be greasy in the privacy of their own homes.

**Limit use of nail polish:** Gel manicures provide a durable coating. Unfortunately, the removal process is damaging to the nail. It’s smarter to skip the gel or, at a minimum, avoid gel manicures in the winter when nails are driest.Even removing regular nail polish can damage the nail, suggests choosing a clear polish if you can’t go without a coating. “Find a formaldehyde-free clear polish that you can apply and leave on for a week. If you choose a color and it chips, you’ll have to replace the polish, which dries out the nail.”

**Some vitamins may help:** Taking a daily biotin supplement (one with about 5,000 micrograms) can improve nail health. To see a difference, you’ll have to wait about six to eight weeks for the entire nail bed to grow out.”

Protein also helps keep nails healthy. Make sure you take in a minimum of 45 grams each day.

**Manicure must-dos:** The cuticle protects your new nail as it grows out. While manicurists routinely push back or trim the cuticles, it’s better to leave them alone. Moisturizing your hands will keep the cuticles from becoming ragged. If your nails are breaking, clip or file them, so they are temporarily shorter. By taking protective steps, including moisturizing, you’ll give your nails a chance to grow out less rigid.

**Beware of myths:** Drinking water is essential to good health, but you can’t drink your way to more supple nails. Consuming gelatin doesn’t improve nail health, either.

## **Prognosis**

The condition is not considered life-threatening. However, it can have a negative impact on quality of life and may impede with one’s daily activities and occupational responsibilities.Idiopathic onychorrhexis is typically milder than onychorrhexis associated with inflammatory nail matrix diseases, such as lichen planus.It can take a significant amount of time for patients to notice major clinical improvement, especially given the slow growth of the nail plate (range, 0.5-1.2 mm/wk.),and that many systemic conditions known to contribute to onychorrhexis are considered chronic.

## **Possible Complications**

This syndrome may cause significant pain and discomfort for patients, as well as increased risk of nail breakage owing to the nail plate fragility. It may also lead to functional impairment in severe cases.

## **Differential Diagnoses**

* Alopecia Areata
* Amyloidosis
* Atopic Dermatitis
* Keratosis Follicularis (Darier Disease)
* Lichen Planus
* Onychomycosis
* Pityriasis Rubra Pilaris
* Psoriasis
* Sarcoidosis

## **When you should see a doctor**

Usually, brittle nails don’t require a doctor visit, but these conditions warrant a call:

* Grooving or separation of your nail plate.
* Redness, swelling or soreness of the skin folds around your nail.

While less common, brittle nails can also be a symptom of several medical conditions, including:

* **Fungal infections.** If in addition to cracking, your nails are getting thicker or turning yellow, you may have a fungal infection.
* **Nutritional deficiencies.** There are several different nutrients we need to keep our nails strong. Fragile nails could be a sign that you aren’t getting enough protein, iron or vitamin B.
* **Psoriasis.** Psoriasis is an immune system disorder that causes dry, scaly patches to form on your skin — including under your nail beds. The condition can also change the color and texture of your nails.
* **Thyroid disorders.** According to a 2019 study, brittle nails can be an early indicator of thyroid conditions like hyperthyroidism, hypothyroidism and Graves’ disease
* **Raynaud’s syndrome.** In Raynaud’s syndrome, brittle nails are the result of diminished blood flow to your fingers.

If you’ve been diagnosed with any of these conditions, it’s worth visiting your doctor to learn how to manage your symptoms.

## **Epidemiology**

### **Frequency**

Brittle nail syndrome affects approximately 20% of the population.Onychoschizia is estimated to affect up to 35% of adult women specifically.The prevalence rate of onychorrhexis alone has not been well-described in literature.

### **Sex**

It is reported to have a notably increased prevalence in women.

### **A**ge

It has an increased prevalence in adults older than 60 years.Commonly referred to as a senile disorder, onychorrhexis has been associated with the age-related reduction in cholesterol sulfate within the nail over time.The

older age predilection may also be linked to the increased likelihood of impaired circulation and concurrent dermatologic or systemic disease with age.

**Race**

Racial predilection of secondary onychorrhexis, if at all, may relate to the racial prevalence of the underlying disease.

## **References**

<https://emedicine.medscape.com/article/2500113-overview#a5>

<https://www.webmd.com/skin-problems-and-treatments/what-to-know-about-onychorrhexis#1-2>

<https://health.clevelandclinic.org/brittle-nails-causes-treatment>

**Onychogryphosis**

Other names: Ram's horn nails

**Definition and description**

Onychogryphosis is a condition mostly affecting the big toenail, in which one set of toenails grows substantially more, and faster, than the other. The nails often become yellow, hardened, and take on a veined, curvy appearance that reminds many people of a ram’s horns — hence the name. It can develop at any stage of life, but older adults are especially susceptible to it.

Very rarely, severe congenital Onychogryphosis or Ram’s Horn Nails in all the twenty nail beds can occur. Congenital onychogryphosis of the fifth toe is quite common and is asymptomatic, so the patient rarely seeks treatment for it and is in fact not even aware of the problem.

### **Causes**

Nails are predominantly made up of a protein called keratin, which ordinarily accumulates one layer after the next in mostly symmetric order in both your hands and feet. When something in your body is out of sync, your finger and toenails often provide some early warning signs. For example:

* Brittle nails may be a sign of iron deficiency or thyroid disease.
* Cracks in your nails are common signs of psoriasis.
* Kidney disease often causes white lines to form in the nails.
* Dark lines in the nails can be a sign of melanoma skin cancer.

Such being the case, it’s important to keep a close eye on your nails and watch for any unusual changes or growths. Simply put, onychogryphosis doesn’t just develop from one day to the next. Many cases result from years of inadequate personal care.

### **Risk Factor**

Onychogryphosis is especially prominent in older people, particularly those who live with or have had:

* Poor circulation
* Type 2 Diabetes
* Bunions
* Gout
* Ichthyosis

It can also be caused by trauma to the feet, caused by either foot injury or wearing poorly fitting shoes. While onychogryphosis is most common in the big toe, it can affect any of the toes on either foot.

### **The Possible Role of Toenail Fungus in Onychogryphosis**

Although some are inclined to think of toenail fungus as purely an aesthetic problem, the truth is that toenail fungus has many serious complications if left untreated for too long. Onychogryphosis may well be one of them.

As many as 50% of those living with onychogryphosis reported having had toenail fungus for many years before their condition developed into onychogryphosis. That doesn’t mean toenail fungus was the cause, but there’s very likely a correlation between the two.

## **Signs & Symptoms**

* The nail gradually becomes thick, long and deformed as they increase in length.
* There can be yellowish discoloration to the nail.
* The nail plate also becomes thick and tender.
* Pain or discomfort in the affected nails upon pressure.
* Nails which are long and curved can affect the adjacent toes resulting in more pain and infection.
* If Onychogryphosis or Ram’s Horn Nails occurs due to fungal infection of the nails, then the nail becomes brittle, crumbly with yellowish discoloration.

## **Diagnosis Method**

Onychogryphosis is diagnosed clinically based on its characteristic appearance. In the early stages, it may be difficult to recognize, as the only feature is hypertrophy of the nail plate, and the classical features usually appear in the later stages.

On histology, the keratinocytes appear disorderly and there is associated hyperchromatism, parakeratosis, and numerous splits .

**The clinical features of onychogryphosis**

The Involvement of one or both great toenails, but any of the nails can be involved

* Opaque, yellow–brown thickening of the nail plate with elongation and increased curvature
* What is often described as a ‘ram’s horn’ or ‘oyster-like’ appearance
* The nail plate initially growing upwards and deviating in a lateral direction towards the other toes
* The nail bed exhibiting an irregular surface marked by striations that are most commonly transverse rather than longitudinal.

## **Treatment options**

Once it develops, onychogryphosis is only treatable through surgery. This can be a delicate procedure since many of those with onychogryphosis have vascular and circulatory problems in their legs and feet. Surgically removing the entire nail bed is the most common treatment, but there are promising innovations in treatment that may make this process easier in the future.

For example, a 2018 study reported a surgery for onychogryphosis in which the entire toenail was simply repaired, not removed. The doctors used a combination of fungal treatments and precise cutting to reshape the nails and eliminate the “hornlike” growth pattern. They achieved satisfactory results in 90% of cases in which the new method was used.

Treatment for onychogryphosis can be either conservative or operative, depending on its cause and symptoms.

Excessive pressure or microtrauma to the nail bed can be minimized by selecting properly fitted footwear.

Conservative treatment involves:

* Regular use of an electric drill, bur, or mechanical debridement

with a nail clipper to shorten the nail and remove subungual hyperkeratosis

* Cryotherapy prior to debridement will soften the nail plate so it is easier to cut
* Blunt dissection with a nail clipper after medical nail avulsion with either 40% urea or 50% potassium iodide.

If conservative treatment fails, nail avulsion may be considered followed by ablative or excisional matricectomy (surgically or chemically removing the proximal nail matrix at the base of the nail).

* Excisional techniques include scalpel excision, cutting electrosurgery, or laser in cutting mode.
* Ablative techniques include chemical cautery, electrosurgery, or laser in ablative mode.

The Zadik technique or a V–Y advancement flap can be used to completely remove the nail matrix. The Syme method, whereby half of the terminal phalanx is removed with the nail fold, is rarely used.

## **Preventive options**

* Everyone should try to take care of their feet as much as they can, but onychogryphosis particularly affects older adults who already have problems with their feet, legs, and circulation. The following methods for preventing nail problems like onychogryphosis will work for anyone, but people who already have conditions like fungus or diabetes ought to be especially diligent:
* **Keep a close eye on your feet.** Don’t ignore fungus on your toenails. Any cuts, spots, or unusual growths on your feet should be meticulously cared for. Consult a doctor if you’re not sure what to do.
* **Trim your nails after washing.** Washing has the two-fold benefit of keeping your feet clean and making rough toenails easier to cut. Just avoid cutting into the corner of the nail.
* **Don’t be afraid of activity.** Poor circulation in the feet can cause many complications, including onychogryphosis. Getting regular exercise, even just in the form of a daily walk, can greatly benefit your overall foot health.

## **Prognosis**

Onychogryphosis tends to recur after conservative treatment. For both clinical and cosmetic purposes, treatment can be repeated to keep the nail bed short and prevent secondary complications. The use of proper footwear to prevent excessive nail pressure on the nail bed is important.

## **References**

<https://dermnetnz.org/topics/onychogryphosis>

<https://www.epainassist.com/joint-pain/foot-pain/onychogryphosis-or-rams-horn-nails>

<https://www.webmd.com/skin-problems-and-treatments/what-is-onychogryphosis#1-7>

**onychocryptosis**

Other names: Ingrown toenails

## **Definition and description**

Ingrown toenails (unguis incarnatus), oronychocryptosis, are a common problem, and causes include poorly fitting (tight) footwear, infection, improperly trimmed toenails, trauma, and heredity. The great toe is the most commonly involved, with the lateral side being involved more commonly than the medial side. The ingrown nail is often diagnosed in schoolchildren, adolescents, young adults, and pregnant women.The underlying cause of this condition is a foreign body reaction. When the nail bed is compressed from the side, the edge of the nail penetrates the cuticle. A foreign body reaction is set up by the presence of the keratinaceous nail material in the flesh of the toe.

Ingrowth of the toenail is generally thought to be multifactorial, including the following:

* Nail length: Cutting the nail so short that it is not constrained by the distal portion of the cuticles, allowing side slippage and penetration of the lateral nail bed by the nail substance.
* External pressure: Wearing shoes that are so tight that they compress the ridges of the cuticles against the relatively stiff nail material, turning the nail into a cutting surface.

## **Causes and Risk Factors**

Many factors can lead to an ingrown toenail. In fact, more than one factor often causes onychocryptosis.

The following factors all increase the risk of having an ingrown toenail:

* **Improper nail trimming technique:** cutting the toenail in a curve instead of straight or cutting it too short may cause the nail to grow inward
* **Constricting footwear:** shoes that are too tight or do not fit correctly at the toes
* **Trauma to the toe:** injuries, such as stubbing the toes or someone accidentally stamping on them
* **Obesity:** increased soft tissue in the feet may mean shoes are too tight, while extra weight may apply more pressure on the nail folds
* **Underlying conditions:** diabetes, thyroid conditions, and renal disorders may swell the feet, increasing pressure on the toes
* **Abnormal nail or toe shape:** pincer or trumpet nails or bony abnormalities in the toes
* **Hyperhidrosis:** excessive sweating may make the nail plate and the surrounding tissue too soft
* **Fungal infection:** these infections may cause the nail to become thick or wide
* **Onychotillomania:** the urge to bite or chew the toenails, or pull and pick at them, can cause damage to the nails, encouraging growth into the skin and infection

The American Academy of Orthopedic Surgeons (AAOS) also states that sometimes the cause is congenital, meaning the person was born with a nail too large for the toe.

## **Signs and symptoms**

The symptoms of an ingrown toenail may include:

* the toe feeling hard or tender to touch
* swelling or redness surrounding the nail
* pus or discharge that signals an infection
* the toe being warm or hot
* experiencing pain when wearing shoes, walking, or putting any pressure on the foot or toe

Doctors may classify an ingrown toenail in the following categories depending on the symptoms:

* **Mild (stage 1):** The nail folds and the toe becomes swollen and red, with pain that worsens with pressure.
* **Moderate (stage 2):** The symptoms are the same as a mild ingrown toenail but with an infection. There may be bleeding or discharge and red, bumpy tissue around the site.
* **Severe (stage 3):** In addition to the symptoms of a moderate ingrown toenail, the toe has chronic inflammation with overgrown skin tissue forming at the site where the nail embeds into the skin.

## **Treatment for onychocryptosis**

A doctor may recommend one or several treatments, depending on the severity of the ingrown toenail, its cause, and whether there are any underlying conditions.

For mild ingrown toenails, a doctor may recommend:

* soaking the foot in warm water 3–4 times a day for 10–20 minutes
* keeping the foot as dry as possible when not soaking it
* wearing comfortable shoes or sandals to give the toes plenty of space
* taking over-the-counter pain relief, such as ibuprofen or acetaminophen
* using dental floss to gently separate the edge of the ingrown toenail from the skin and packing to hold it away from the skin or pulling the nail fold from the nail by using a taping technique

For more severe ingrown toenails with an accompanying infection, a doctor may recommend a different treatment or a minor surgical procedure to treat the condition, such as:

* using a nail brace to lift the nail clear of the skin
* trying a gutter splint as support under the nail to help it grow above the skin edge
* removing portions of the nail that have overgrown, or even parts of the nail bed, to prevent the nail from growing back
* using a chemical to remove the nail over several weeks
* shortening the distal phalanx, the bone at the tip of the toe

Surgeons perform surgical procedures using a local anesthetic. A doctor may apply antibiotic ointment to the wound after the surgery. A person may also require oral antibiotics.

Recovery from a surgical procedure takes a few weeks. However, it may take 2–4 months for the toenail to grow back. Some surgical procedures involving removing parts of the nail bed may prevent the toenail from growing back.

## **Preventive options**

The following tips may help reduce the risk of an ingrown toenail developing:

* **Cut toenails straight across when trimming:** Try not to cut them too short or round off the edges of the nail.
* **Wear comfortable shoes that do not constrict the toes:** Wide or open-toe shoes are best. If wearing heels, use cushioning to prevent the foot from sliding forward in the shoe and compressing the toes.
* **Keep the feet clean and dry:** Wash feet regularly and change socks if they are sweaty.
* **Ask for help with trimming:** A person can get help from a friend or relative or visit a podiatrist when having difficulty trimming nails.
* **Treat underlying conditions correctly:** Conditions such as diabetes increase the risk of onychocryptosis.

## **Reference**

<https://emedicine.medscape.com/article/828072-treatment?form=fpf>

<https://www.medicalnewstoday.com/articles/onychocryptosis>

<https://dermnetnz.org/topics/ingrown-toenail>

# **Onychomycosis**

Other names: Nail fungal infections, tinea unguium

**Definition and Description**

A fungal nail infection is a common condition that can leave you with brittle, discolored nails, usually on your toes.

Its formal name is onychomycosis, and it’s a lot like athlete's foot. But instead of affecting the skin on the bottom of your feet or between your toes, it invades your nails.

Fungi are tiny organisms that you can only see through a microscope. Many different types can cause a nail infection. Sometimes they live on your skin and don’t cause any trouble. But if you have a lot in one area, you might get infected.

Don’t be embarrassed if you have toenail or fingernail fungus. It’s way more common than you think.

**Types of Fungal Nail Infections**

There are 4 main kinds of fungal nail infection. Each looks slightly different:

* **Distal or lateral subungual onychomycosis.** This is the most common kind. It results from a fungus called a dermatophyte. You can get it in your fingernails or toenails. It starts in the nail bed, underneath the nail. You’ll see a yellowish colored area that spreads from the edges of the nail to the center, and places where it comes apart from the nail bed.
* **White superficial onychomycosis.** This is less common and only affects the nail surface, mainly on your toenails. It starts as white spots, which become powdery and cause the nail to crumble.
* **Proximal subungual onychomycosis.** This appears first as white spots in the center of the nail bed at the cuticle. They move outward as the finger or toenail grows. It’s rare and usually affects people who have immune system problems, like HIV infection.
* **Candidal onychomycosis.** Yeast causes this infection that usually affects your fingernails. The area around the nails is often swollen and inflamed, and the nails may come off entirely. It tends to happen to nails that have been damaged by an injury or another infection.

## **Causes and Risk Factor**

### **Causes**

You get an infection when a crack in your nail or the skin around it allows fungus to get inside and grow.

Since fungus thrives in dark, warm places, your toenails are more likely to be affected than your fingernails. Your toes also have less blood flow than your fingers, which makes it harder for your body to pick up on and prevent an infection.

### **Risk Factor**

* Are a man
* Are older, since nails become more brittle and likely to crack as you age
* Have a weak immune system or ongoing health problems like diabetes
* Wear shoes that make your feet hot and sweaty
* Walk barefoot through gym showers, swimming pools, and locker rooms –places where fungus spreads easily
* Live with someone who has a fungal infection
* Have athlete’s foot, as the fungus that causes it can spread to your nails
* Recently had an injury or surgery on your nail, or had a previous infection
* Wear plastic gloves or keep your hands wet for long periods

See your doctor if you think you have nail fungus. You may need a prescription to treat it, whether that means taking a medicine by mouth or using a special cream. In serious cases, your nail may need to be removed so a healthy new one can grow in its place. Doctors can also use lasers to treat nail fungus.

## **Signs and Symptoms**

Symptoms are different, depending on which type of fungal nail infection you have. They usually start mild and get more serious.

* At first, you may only see a white or yellow spot under your nail. Over time, this spreads and can turn your whole nail white, yellow, green, or black.
* The nail may thicken and could be hard to trim.
* It may start to curl up or down or loosen from the nail bed.
* Your nail could become brittle and crumble when you touch it.
* Your nail may become misshapen.
* You may notice a bad smell.

It’s easy to ignore fungal nail infections at first, since you may not have any pain. But if you don’t treat them, it can hurt to put any pressure on the area. If an infection gets bad enough, it could even become hard to walk.

## **Diagnosis Method**

Your health care provider will examine your nails and perhaps take some nail clippings or scrape debris from under your nail. These samples are sent to a lab to identify the cause of your symptoms.

Other conditions, such as psoriasis, can mimic a fungal infection of the nail. Microorganisms such as yeast and bacteria also can infect nails. Knowing the cause of your infection helps determine the best treatment.

## **Treatment options**

See your doctor if you think you have nail fungus. It can be tough to get rid of, and you’re more likely to have success with a prescription. Treatments include:

* **Oral antifungals.** The doctor may give you a pill to kill fungus in your whole body. This is usually the best way to get rid of a nail infection. Treatment may last 2 months for an infection in your fingernails, or 3 months if it’s in your toenails.
* **Topical antifungals.** You rub or brush these medicines onto your nails. They may work for a mild infection, but they can’t get deep enough into the nail to cure a more serious one. You might use a topical treatment in combination with a pill.
* **Surgery.** If other treatments don’t work, the doctor may need to remove your nail entirely and let a healthy one grow back in its place. The new nail could also get infected. The most effective but least used option is surgery to permanently remove the nail and its root.
* **Laser or photodynamic therapy.** Doctors are studying newer treatments that use special light to try to kill the fungus.

### **Medications**

Your health care provider may prescribe antifungal drugs that you take by mouth (orally) or apply to the nail.

* **Oral antifungal drugs.** These drugs are often the first choice. One option is itraconazole (Sporanox). These drugs help a new nail grow free of infection, slowly replacing the infected part.

You typically take this type of drug daily for 6 to 12 weeks. But you won't see the end result of treatment until the nail grows back completely. It may take four months or longer to eliminate an infection. Treatment success rates with these drugs appear to be lower in adults over age 65.

Oral antifungal drugs may cause side effects such as rash and liver damage. Or they may interfere with other prescription drugs. You may need occasional blood tests to check on how you're doing with these types of drugs. Health care providers may not recommend oral antifungal drugs for people with liver disease or congestive heart failure or those taking certain medications.

* **Medicated nail polish.** Your health care provider may prescribe an antifungal nail polish called ciclopirox (Penlac). You paint it on your infected nails and surrounding skin once a day. After seven days, you wipe the piled-on layers clean with alcohol and begin fresh applications. You may need to use this type of nail polish daily for almost a year.
* **Medicated nail cream.** Your health care provider may prescribe an antifungal cream, such as efinaconazole (Jublia) and tavaborole (Kerydin). You rub this product into your infected nails after soaking. These creams may work better if you first thin the nails. This helps the medication get through the hard nail surface to the underlying fungus.

To thin nails, you apply a nonprescription lotion containing urea. Or your health care provider may thin the surface of the nail (debride) with a file or other tool.

Antifungal nail creams may cause side effects such as rash.

### **Surgery**

Your health care provider might suggest temporary removal of the nail so that the antifungal drug can be applied directly to the infection under the nail.

## **Preventive options**

To prevent an infection, wash your hands and feet often. Use soap, and make sure you get between your fingers and toes. Also:

* Keep your fingernails and toenails short and trimmed straight across.
* Wear socks that wick away (absorb) moisture. If your feet sweat a lot, change your socks once or twice a day, or take off your shoes and let your feet cool when you have the chance.
* Use antifungal powder or spray on your feet as well as in your shoes. Throw away old pairs of closed-toe shoes since fungi might be living in them.
* If you get manicures at nail salons, visit only the ones that disinfect tools after each client. You can also bring your own file and clippers from home. Ask that your cuticles not be cut, since this can cause tiny breaks in the skin that let germs in.
* Don’t share towels if someone else in your family has nail fungus. This will pass around the infection.

## **Prognosis**

Recurrence may occur following treatment, with a 20-25% relapse rate within 2 years of successful treatment.Nail fungus can be painful and cause permanent damage to nails. It may lead to other serious infections if the immune system is suppressed due to medication, diabetes or other conditions. The risk is most serious for people with diabetes and with immune systems weakened by leukemia or AIDS, or medication after organ transplant. Diabetics have vascular and nerve impairment, and are at risk of cellulitis, a potentially serious bacterial infection; any relatively minor injury to feet, including a nail fungal infection, can lead to more serious complications.

## **Possible Complications**

It can take a year or more for your nails to look like they did before the infection. And you may continue to have problems:

* The fungus can come back.
* Your nails may be permanently discolored or misshapen.
* The infection may spread to other parts of your body.

It’s especially important to take care of a fungal nail infection if you have diabetes. You’re at greater risk of getting a serious skin infection.

## **Differential Diagnosis**

In many cases of suspected nail fungus there is actually no fungal infection, but only nail deformity.

To avoid misdiagnosis as nail psoriasis, lichen planus, contact dermatitis, nail bed tumors such as melanoma, trauma, or yellow nail syndrome, laboratory confirmation may be necessary.

Other conditions that may appear similar to onychomycosis include: psoriasis, normal aging, green nail syndrome, yellow nail syndrome, and chronic paronychia

## **When to see a doctor**

You may want to see a health care provider if self-care steps haven't helped and the nail becomes increasingly discolored, thickened or misshapen. Also talk with your health care provider if you have:

* Diabetes and think you're developing nail fungus
* Bleeding around the nails
* Swelling or pain around the nails
* Difficulty walking

## **Epidemiology**

Onychomycosis is estimated to affect around 14% of the general population and is especially common in older adults. People with diabetes are at increased risk for fungal nail infections.

## **Reference**

<https://www.mayoclinic.org/diseases-conditions/nail-fungus/symptoms-causes/syc-20353294>

<https://www.cdc.gov/ringworm/hcp/clinical-overview/index.html>

<https://www.webmd.com/skin-problems-and-treatments/fungal-nail-infe>ctions#1-6

## **Onycholysis**

## **Definition and description**

Onycholysis is characterized by a spontaneous separation of the nail plate starting at the distal free margin and progressing proximally. In onycholysis, the nail plate is separated from the underlying and/or lateral supporting structures. Less often, separation of the nail plate begins at the proximal nail and extends to the free edge, which is seen most often in psoriasis of the nails (termed onychomadesis). Rare cases of onycholysis are confined to the nail's lateral borders. onycholysis isn’t a fungal infection. However, fungal infections can sometimes cause onycholysis.

Depending on what causes your onycholysis, it may be contagious. Onycholysis caused by an injury, nail psoriasis or reaction to a medication or chemical isn’t contagious. However, onycholysis caused by a fungus may be contagious.

A person with an infection can spread the fungus to someone else through skin-to-skin contact. You can also get nail fungus by touching an infected surface (indirect contact), like walking barefoot around public pools or showers or by sharing items like towels, nail clippers and nail scissors.

## **Causes and Risk Factors**

The following are common causes of onycholysis:

* **Injuries (trauma)**: Injuries to a nail or the area where your nails start to grow (nail matrix) can cause onycholysis. You can injure these areas by bumping or hitting your nails, wearing shoes that are too small or even keeping your nails in water for too long. Regularly going to a nail salon to get manicures can also cause onycholysis. Manicurists use a lot of force to trim, buff and polish your nails. Even tapping your nails on a hard surface over a long period can cause onycholysis.
* **Reaction to chemicals**: Chemicals in nail polish, nail gloss, nail hardener, nail polish remover and fake nails can cause onycholysis.
* **Fungal infections**: Fungal infections occur when fungus gets between your nail and nail bed, usually through cracks or cuts in your finger or toe. Fungal infections cause your nails to become thick and yellow, and they may show white spots and streaks.
* **Medications**: Chemotherapy and some medications that cause light sensitivity can cause onycholysis. These medications include tetracycline, nonsteroidal anti-inflammatory drugs (NSAIDs), psoralens and oral retinoids (vitamin A derivatives).
* **Nail psoriasis**: Nail psoriasis causes changes to the structure of your nails, which may include onycholysis.

Onycholysis rarely affects all of your nails. The following may cause onycholysis in all of your nails:

* **Iron deficiency**: An iron deficiency is when you don’t have enough iron in your diet. An iron deficiency can also cause anemia.
* **Hyperthyroidism**: Hyperthyroidism causes your thyroid gland in your neck to create and release more hormones than your body needs. Hyperthyroidism can also cause a rapid heartbeat, unexplained weight loss, increased appetite and anxiety disorders.

## **Signs and Symptoms**

Symptoms of onycholysis include:

* Nails that peel away from their nail beds.
* Tough, thick nail beds.
* An abnormal border between the pink area of your nail and the white edge of your nail. The border may look wavy, and the white areas may be thicker in some areas and thinner in others.
* Discoloration in your nails and nail beds. They may look gray, green, purple, white or yellow.
* Dents or pits (cupuliform depressions) in your nails.
* Crumbling nails.
* Hemorrhages underneath your nails.

Onycholysis usually isn’t painful, but what causes your onycholysis may be painful or irritating. If you have onycholysis due to injuries or fungal infections, you may experience pain and irritation.

## **Diagnosis Method**

Onycholysis is easy to recognize, so you don’t necessarily need a healthcare professional to diagnose it, especially if you know its cause.

If you don’t know what’s caused your onycholysis, seeing your healthcare provider is a good idea. They’ll look closely at your affected nails to evaluate your symptoms.

Evaluation of patients with onycholysis requires a careful history of exposure to etiologic agents.

### Physical Examination

In onycholysis, nails are smooth, firm, and without inflammatory reaction.

Discoloration underneath the nail may occur as a result of secondary infection.

Spontaneous separation of the nail plate in onycholysis starts at the distal free margin and progresses proximally. Less often, nail plate separation may begin at the proximal nail and extend to the free edge. The nail plate is separated from underlying and/or lateral supporting structures.

### Biopsy

Your healthcare provider will use a razor or surgical knife with a thin blade (scalpel) to scrape away a small sample of cells from your nails. The cells go to a laboratory for testing, and researchers examine them under a microscope.

### Fungal test

Your healthcare provider may clip off some of your nails and order a potassium hydroxide (KOH) preparation or fungal culture to rule out a fungus as the cause of your onycholysis. If those test results aren’t clear, a fungus test known as a periodic acid-Schiff (PAS) stain can also determine the presence of a fungus.

### Blood test

During a blood test, your healthcare provider will use a thin (21 gauge, slightly smaller than the size of a standard earring) needle to withdraw a small amount of blood from a vein in your arm. The blood sample goes to a laboratory for testing, and researchers examine it to check for the presence of any systemic diseases that may cause onycholysis.

## **Treatment Options**

### **Can onycholysis be cured?**

There isn’t a cure for the section of your nail that’s detached from the nail bed — you can’t reattach it. But treatment can keep new nail growth attached to your nail bed.

### **What can I use to treat onycholysis?**

If you have onycholysis because of an injury, the detached part of your nail will eventually grow out. Use nail clippers or nail scissors to remove your affected nail as it grows out.

If you have onycholysis because of a fungus, your options may include:

* **Oral antifungal medication**: Your healthcare provider may prescribe liquid medicines or pills or tablets that you swallow with water. These medications may include terbinafine (Lamisil®), itraconazole (Sporanox®) and fluconazole (Diflucan®).
* **Topical antifungal medication**: Topical medications come in the form of creams, ointments or gels. You rub them directly onto your nails.

Nail fungi can be difficult to treat. It’s important to finish your full course of medicine. If you stop too soon, the fungus that caused your onycholysis may come back and be harder to treat.

Onycholysis related to psoriasis or eczema may respond to a midstrength topical corticosteroid. Psoralen plus ultraviolet A (PUVA) treatment has also been reported as an effective therapy for psoriatic onycholysis.

Laser treatments, often in conjunction with topical agents, have shown favorable results in the treatment of nail psoriasis. A systematic review of 19 studies of pulse dye laser (PDL), long-pulsed neodymium:yttrium aluminum garnet (Nd:YAG) laser, and fractional carbon dioxide laser (FCL) interventions reported all were effective in lowering Nail Psoriasis Severity Index (NAPSI) scores. In addition, PDL and Nd:YAG laser treatment were more effective at reducing nail bed features, while FCL was effective at reducing both nail bed and matrix features.

### **How do I take care of my nails?**

The following tips can help you take care of your nails if you have onycholysis:

* Take medications and apply treatments as instructed by your healthcare professional.
* Regularly trim your affected nails.
* Protect your nails from any additional damage.
* Wash your hands regularly to prevent an infection. Use a clean washcloth to help reach beneath your nails.
* Use antifungal or antimicrobial soaks to help prevent infection. Antimicrobial soaks may include lemon juice, vinegar, hydrogen peroxide and tea tree, orange or lemongrass essential oils.

While essential oils are safe for most people, it’s a good idea to check with your healthcare provider before trying them. You may be at risk of developing an allergic reaction.

## **Prevention**

The following tips can help you prevent onycholysis:

* Keep your nails trimmed short. Keeping your nails short will prevent them from catching on objects and pulling further off of your nail beds.
* Avoid biting your nails.
* Be careful when cleaning underneath your nails. Some tools that clean underneath your nails, like cuticle sticks or nail brushes, can break the skin underneath your nails and cause an infection.
* Avoid chemicals or products that can irritate your nails or the skin around your nails. These may include nail polish, nail gloss, nail hardener, nail polish remover and fake nails.
* Wear gloves when doing activities that may damage your nails, like washing the dishes, playing sports or working outside.
* Wear clean socks and comfortable, protective shoes. Be careful when putting down heavy objects near your feet.

## **Prognosis**

If you and your healthcare provider can determine the cause of your onycholysis, your nails will slowly but surely regrow. Your fingernails grow slowly, and your toenails grow even slower. It may take up to six to nine months for your fingernails to grow completely out, and it may take 12 to 18 months for your toenails to grow completely out.

## **Possible Complication**

Onychomycosis may serve as a reservoir for cutaneous fungal infections such as tinea pedis, tinea corporis, and tinea cruris. The fungus may also disseminate to other nails. There is an increased risk for bacterial infections such as cellulitis and paronychia, especially in immunocompromised individuals including diabetics. Severe onychomycosis may interfere with standing, walking, nail function, and daily activities. The condition, if left untreated, may cause discomfort, pain, paresthesia, nail deformities such as transverse over-curvature, difficulties in trimming thick nail plates, difficulties in fitting shoes, and low self-esteem. In addition, onychomycosis can be unsightly and socially embarrassing (especially for females) and may have an adverse effect on quality of life

## **Differential Diagnosis**

Differential diagnosis includes nail changes in psoriasis, lichen planus, alopecia areata, chronic dermatitis, onychogryphosis, chronic paronychia, pityriasis rubra pilaris, pachyonychia congenita, trachyonychia, onychogryphosis, median nail dystrophy, melanonychia striata, subungual melanoma, pemphigus vulgaris, pemphigoid, epidermolysis bullosa acquisita, bullous epidermolysis, subungual wart, subungual exostosis, subungual keratoacanthoma, rheumatoid arthritis, scleroderma, lupus erythematosus, scabies, tungiasis, twenty nail dystrophy, yellow nail syndrome, traumatic onychodystrophy, onychomatricoma, idiopathic onycholysis, porphyria, amyloidosis, myxoid cyst, fibroma, glomus tumor, Bowen disease, and squamous cell carcinoma

## **Epidemiology**

The overall worldwide prevalence of onychomycosis in the general population is approximately 5.5%, based on recently published epidemiological studies. A 2013 systematic review of 11 population-based and 21 hospital-based studies showed that the mean prevalence of onychomycosis in North America and Europe was 4.3% (95% confidence interval: 1.9 to 6.8) in the population-based studies and 8.9% (95% confidence interval: 4.3 to 13.6) in the hospital-based studies. There is evidence that the prevalence is rising, possibly because of longer life expectancy, use of occlusive modern footwear, increased prevalence of obesity, and increased urbanization. The condition is much more common in adults than in children and the prevalence increases with age. The prevalence in children in North America is approximately 0.4%, whereas the prevalence may be as high as 35% in the elderly (> 65 years of age). Toenail onychomycosis is more common in males whereas *Candida* fingernail onychomycosis is more common in females. Other predisposing factors include fungal infection elsewhere on the body (in particular, tinea pedis), chronic paronychia, previous onychomycosis, wearing of occlusive and tight shoes, hyperhidrosis, participation in sports or fitness activities, nail trauma, poor nail grooming, use of commercial swimming pools, communal bathing, living with family members with fungal infection, poor health, genetic factors, immunodeficiency (in particular, acquired immune deficiency syndrome and transplant patients), diabetes mellitus, obesity, Down syndrome, psoriasis, smoking, peripheral vascular disease, venous insufficiency, hallux valgus, and asymmetric gait nail unit syndrome.

## **Reference**

Leung AKC, Lam JM, Leong KF, Hon KL, Barankin B, Leung AAM, Wong AHC. Onychomycosis: An Updated Review. Recent Pat Inflamm Allergy Drug Discov. 2020;14(1):32-45. doi: 10.2174/1872213X13666191026090713. PMID: 31738146; PMCID: PMC7509699.

<https://pmc.ncbi.nlm.nih.gov/articles/PMC7509699/#sec3>

<https://dermnetnz.org/topics/onycholysis>

<https://my.clevelandclinic.org/health/diseases/22903-onycholysis#outlook-prognosis>

**Paronychia**

Other names: Infection of skin around the nail

**Definition and Description**

Paronychia is nail inflammation that may result from trauma, irritation or infection. It can affect fingernails or toenails.

Paronychia can develop when bacteria enter broken skin near the cuticle and nail fold, causing an infection. The cuticle is the skin at the base of the nail. The nail fold is where the skin and nail come together.

Healthcare providers treat paronychia with antibiotics to kill the infection. Providers may also drain pus (thick, infectious fluid that builds up around a wound). They may also culture the fluid to see what specific bacteria might be causing the infection.

Sometimes, the infection comes back or symptoms last for weeks (chronic paronychia). Chronic paronychia is more commonly caused by irritation from occupational or environmental exposures. Less often, it may be caused by a chronic bacterial or fungal infection.

### **What are the types of paronychia?**

There are two types of paronychia. Both types have similar signs and symptoms:

* **Acute paronychia:** Symptoms of acute paronychia appear over hours or a few days. The infection is only in the nail fold and doesn’t extend deeper inside the finger or toe. Symptoms go away with treatment and last less than six weeks.
* **Chronic paronychia:** Symptoms develop more slowly than acute paronychia, and they usually last six weeks or longer. Several fingers or toes can be infected at once. A nail fungus (usually from a type of fungus called candida) may occur along with the bacterial infection. Candida is one of several types of fungi that cause toenail fungal infections.

## **Causes and Risk Factor**

Most commonly, infectious paronychia results from a staph infection. *Staphylococcus aureus* bacteria cause staph infections. Other bacteria (such as *Streptococcus pyogenes*) can also cause the infection. Bacteria get into the skin through:

* Cuts, broken skin or hangnails.
* Ingrown nails (this happens most often with ingrown toenails).
* Irritation from water or chemicals.
* Trauma to the nailbed or cuticle area. Trauma can result from accidents, nail biting or frequent manicures or pedicures.
* Some medications can also cause paronychia. Some of these medications include retinoids, anti-cancer medications, HIV medications and some antibiotics.

## **Signs and Symptoms**

Symptoms of paronychia usually develop over several hours or days. Sometimes they take longer to develop. Symptoms appear where the nail meets the skin (the nail fold and cuticle). The sides of the nail can also be affected.

Paronychia symptoms include:

* Pain, swelling and tenderness around the nail.
* Skin that is red and warm to the touch.
* Pus that builds up under the skin. A white to yellow, pus-filled abscess may form. If an abscess forms, it may require antibiotics and/or drainage.

Untreated, the nail can start to grow abnormally and may have ridges or waves. It may look yellow or green, and it can be dry and brittle. The nail can detach from the nail bed and fall off.

## **Diagnosis Method**

To diagnose a paronychia, you will need to obtain a good history and physical, revealing a swollen and tender nail fold, as there is no laboratory testing or imaging that will lead to the diagnosis. The infection is usually straightforward; however, the presence of an abscess is not always evident, and the digital pressure test described above can be used to guide you.

## **Treatment option**

Paronychia are usually either treated with incision and drainage or antibiotics. If there is inflammation with no definite abscess, treatment can include warm soaks with water or antiseptic solutions (chlorhexidine, povidone-iodine) and antibiotics. Warm soaks should be for 10 to 15 minutes, multiple times a day. There is not strong evidence recommending topical vs. oral antibiotics, and this may be physician-dependent based on experience. Antibiotics used should have staph aureus coverage. Topical antibiotics used may be a triple antibiotic ointment, bacitracin, or mupirocin. In patients failing topical treatment or more severe cases, oral antibiotics are an option; dicloxacillin (250 mg four times a day) or cephalexin (500mg three to four times a day). Indications for antibiotics with anaerobic coverage include patients where there is a concern for oral inoculation; this would require the addition of clindamycin or amoxicillin-clavulanate. If the patient has risk factors for MRSA (including but not limited to: recent hospitalization, recent surgery, ESRD on hemodialysis, HIV/AIDS, IVDU, resident of long term care facility), choose an antibiotic with the appropriate coverage. Options include trimethoprim/sulfamethoxazole DS (1 to 2 tablets twice a day), clindamycin (300 to 450 mg four times a day) or doxycycline (100mg twice a day).

If an abscess is present, the infection will require drainage. Incision and drainage are usually with a #11 scalpel, and the blade is inserted under the eponychial fold (lateral nail fold) until pus begins to drain. Local or digital block anesthetic is generally helpful to allow comfort to ensure complete drainage. An abscess requires irrigation with normal saline, and if the abscess and incision site is large, the clinician can pack it with plain gauze for continued drainage. If the abscess extends to the nail bed or is associated with an ingrown nail, a partial nail plate removal may be needed. If an abscess is present and not drained, it can spread under the nail to the other side and result in a "run-around abscess." This scenario may require complete removal of the nail to allow adequate drainage and treatment. Warm soaks should be initiated after incision and drainage to encourage continued drainage by keeping the wound open and prevent secondary infection. The patient should follow up with a provider in the next 24 to 48 hours to ensure drainage and to look for signs of worsening infection. Usually, incision and drainage is the adequate treatment of acute paronychia; however, if there is a significant extension of cellulitis, oral antibiotics may be prescribed as above.

In chronic paronychia, the patient should be instructed to avoid trauma as to the hands as much as possible. Wearing gloves is advised for manual workers. Treatment in chronic paronychia should point toward fungal etiology. Topical and systemic antifungal agents such as itraconazole and terbinafine are options since the etiological factor in chronic type is mostly *Candida* species. Other inflammatory diseases of the digits like ingrown nails, psoriasis, etc. should have treatment as well. In difficult to treat chronic paronychia, other causes such as malignancy merit exploration.

## **Prevention**

To prevent a nail infection, you should:

* Avoid biting or chewing on your nails or hangnails. Don’t pick at your cuticles.
* Be careful not to cut your nails too short. When trimming cuticles, avoid cutting too close to the nail fold.
* Maintain good hygiene by washing your hands and keeping your nails clean. Use gentle soaps that are not irritating to your skin.
* Use lotion on your nail fold and cuticles if your skin is dry. Excessive dryness can cause the skin to crack.
* Wear waterproof gloves if you work with chemicals or your hands will be wet for a long period.

## **Prognosis**

Paronychia usually has a good prognosis. Acute paronychia usually resolves within a few days and will rarely recur in healthy individuals. Chronic paronychia may persist for several months or longer and may recur in predisposed patients

Paronychia usually clears up with treatment. Some people get more than one infection, or the infection comes back after treatment (chronic paronychia). Untreated, the infection can cause damage to the nail.

Rarely, untreated paronychia can go deeper into the finger or toe and lead to a serious infection. The infection may progress to involve the underlying bone. In severe cases, providers need to remove a finger or toe to make sure the infection doesn’t spread to the rest of the body. Severe, chronic paronychia most often affects people who have diabetes or conditions that cause problems with blood circulation.

## **Possible Complication**

Acute paronychia can cause a severe infection of the hand and may spread to involve underlying tendons, which is why appropriate treatment on initial presentation is essential. This status may require evaluation and treatment by a hand surgeon as it often involves debridement, washout, or amputation, based on the severity of the infection. The major complication of chronic paronychia is nail dystrophy. It is often associated with brittle, distorted nail plates. Nail discoloration is not an uncommon complication of chronic paronychia.

## **Differential Diagnosis**

Differential diagnosis of paronychia include:

1- Cellulitis - Cellulitis is a superficial infection and will present as erythema and swelling to the affected portion of the body with no area of fluctuance. Treatment is with oral antibiotics.

2 - Felon - A felon is a subcutaneous infection of the digital pulp space. The area becomes warm, red, tense, and very painful due to the confinement of the infection, creating pressure in the individual compartments created by the septa of the finger pad. These require excision and drainage, usually with a longitudinal incision and blunt dissection to ensure adequate drainage.

3 - Herpetic whitlow - This is a viral infection of the distal finger caused by HSV. Patients usually develop a burning, pruritic sensation before the infection erupts. A physical exam will show vesicles, vesicopustules, along with pain and erythema. It is important to not confuse this with a felon or a paronychia as incision and drainage of herpetic whitlow could result in a secondary bacterial infection and failure to heal.

4- Onychomycosis - This is a fungal infection of the nail that causes whitish-yellowish discoloration. Sometimes difficult to treat and requires oral antibiotics instead of topical.

5- Nail Psoriasis - psoriasis can also affect the fingernails and toenails. It may cause thickening of the nails with areas of pitting, ridges, irregular contour, and even raising of the nail from the nail bed.

6- Squamous cell carcinoma - Squamous cell carcinoma is mainly cancer of the skin but can also affect the nail bed. It is a rare malignant subungual tumor subject to misdiagnosis as chronic paronychia.

## **Epidemiology**

Paronychia is more common in women than in men, with a female-to-male ratio of 3 to 1. Usually, they affect manual labor workers or patients in occupations that require them to have their hands or feet submerged in water for prolonged periods (e.g., dishwashers). Middle-aged females are at the highest risk of infection.

## **Reference**

Dulski A, Edwards CW. Paronychia. 2023 Aug 7. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 31335027. <https://www.ncbi.nlm.nih.gov/books/NBK544307/#_article-26682_s4_>

<https://my.clevelandclinic.org/health/diseases/15327-nail-infection-paronychia#diagnosis-and-tests>

# **Leukonychia**

Other names: leukonychia, white nail

## **Definition and Description**

Leukonychia is a common condition that causes white spots or streaks on your fingernails or toenails. There are three types of leukonychia:

* **True leukonychia**: In true leukonychia, the white spots form in the area of your fingers or toes where the nail starts to grow (nail matrix), and appear in the hard part of your nail (nail plate).
* **Apparent leukonychia**: In apparent leukonychia, the white spots form in the skin on which your nails rest (nail bed).
* **Pseudoleukonychia**: In pseudoleukonychia, the white spots form on the surface of your nail. Outside organisms — like fungi — cause pseudoleukonychia.

In some people, leukonychia appears as one or two medium-sized spots or many tiny specks. In others, the spots may be very large. You may have spots on only one nail, or you may have spots on many nails.

White spots usually mean that your nails have experienced some sort of stress. The stress could be from an injury, like hitting your nail against a hard surface, an infection or an allergic reaction. White spots are sometimes the side effects of medications.

## **Causes and Risk Factor**

The following are common causes of leukonychia:

* **Allergies**: Your immune system may interpret products that you use on your nails as allergens and cause white spots to form. These products may include nail polish, nail gloss, nail hardener, nail polish remover and fake nails.
* **Injuries (trauma)**: Injuries to a nail plate or nail matrix are the most common cause of white spots on your nails. You can injure your nail plates or nail matrixes by bumping or hitting your nails, wearing shoes that are too small or biting your nails. Regularly going to a nail salon to get manicures can injure these areas, too. Manicurists may use a lot of force to trim, buff and polish your nails.
* **Fungal infections**: Fungal infections can make your nails look discolored (sometimes, they might look like they have white spots), thick or cracked.
* **Poisoning and medications**: White spots may sometimes appear on your nails due to exposure to toxic heavy metals, including arsenic and lead. Chemotherapy and some medications used for bacterial infections and urinary tract infections, including sulfa drugs (sulfonamides), may also cause white spots.
* **Systemic diseases**: A systemic disease is a disease that affects your entire body. White spots on your nails are sometimes a rare symptom of many systemic diseases, including diabetes, heart failure, HIV, liver cirrhosis and psoriasis.
* **Hereditary conditions**: Hereditary means inherited **—** a biological parent passes down something from their genes to their child. Genes determine a person’s physical traits, including hair color, eye color and height. Some hereditary conditions that affect your nails, including Bart-Pumphrey syndrome and Darier disease, may cause white spots to appear.

**What deficiency causes white spots on nails?**

Healthcare providers and medical researchers aren’t sure whether deficiencies cause white spots to appear on your nails. A deficiency is a shortage of a basic substance in your body that’s essential to your health, like certain vitamins or minerals. Some believe that a lack of minerals — including iron, calcium and zinc — may cause leukonychia. Others think it might be a vitamin deficiency. Still, others believe this isn’t true, or feel there isn’t enough research to make any accurate conclusions.

**Does anxiety cause white spots on nails?**

Anxiety itself doesn’t cause white spots to develop on your nails. But injury to your nail — like from picking or biting them — might. If you pick at or bite your nails as a result of your anxiety, speak with a healthcare provider.

## **Signs and Symptoms**

The primary symptom of onycholysis is the visible separation of the nail from the nail bed. Other symptoms may include:

* **Discoloration:** The affected nail may appear white, yellow, or greenish.
* **Thickening of the Nail:** The nail may become thicker or develop irregularities.
* **Pain or Discomfort:** Some individuals may experience pain, especially if the nail is injured.

## **Diagnosis Method**

Diagnosis of leukonychia is clinical with the need for an additional test in a minority of cases. The diagnostic algorithm, which is presented in, can be a useful tool in most patients. By combining both the anatomical and the morphological classification, the most likely causes can be identified or excluded. Dermoscopy is very useful to integrate the clinical examination, unmasking minor alterations not visible or unclear to the naked eye. This technique has become increasingly popular in recent years to facilitate the clinical diagnosis of nail disorders, opening up a valuable second front with a potential to avoid invasive diagnostic methods. Dermoscopy is also valuable in monitoring the evolution of a disease and response to treatment through stored images. When dealing with leukonychia, besides the morphological aspect that is clearly distinguishable by the naked eye, dermoscopy allows true leukonychia to be better distinguished from apparent leukonychia and pseudoleukonychia. The test of the whitish discoloration of the nail plate that disappears with pressure in cases of apparent leukonychia, but not in true leukonychia, can also be done with the lens of the dermoscope. Moreover, being a color abnormality, leukonychia is better appreciated with an interface solution (ultrasound gel) between the nail plate and the dermoscope lens

## **Treatment Option**

Treatment for leukonychia depends on its cause. If you have white spots on your nails because of injuries, they’ll slowly grow out until you can remove them with nail clippers or nail scissors. Fingernails grow slowly, and toenails grow even slower. It may take up to six to nine months for white spots on your fingernails to grow out, and it may take 12 to 18 months for white spots on your toenails to grow out.

If you have white spots on your nails but had no injury, a healthcare provider may recommend several tests to help make a diagnosis, including:

* **Biopsy**: Your healthcare provider uses a razor or surgical knife with a thin blade (scalpel) to scrape away a small sample of cells from your nails. The cells go to a laboratory for testing, and researchers examine them under a microscope.
* **Blood test**: During a blood test, your healthcare provider will use a thin (21 gauge, slightly smaller than the size of a standard earring) needle to withdraw a small amount of blood from a vein in your arm. The blood sample goes to a laboratory for testing, and researchers examine it to check for the presence of any systemic diseases.
* **Potassium hydroxide (KOH) preparation or fungal culture**: Your healthcare provider will clip off parts of your affected nails and send the clippings to a laboratory to check for the presence of fungi.

If you have white spots on your nails because of a fungus, your options may include:

* **Oral antifungal medication**: Your healthcare provider may prescribe liquid medicines or pills or tablets that you swallow with water. These medications may include terbinafine (Lamisil®), itraconazole (Sporanox®) and fluconazole (Diflucan®).
* **Topical antifungal medication**: Topical medications usually come in the form of creams, ointments or gels. You rub them directly onto your nails.

Nail fungi can be difficult to treat. It’s important to finish your full course of medicine. If you stop too soon, the fungus that causes white spots on your nails may come back and be harder to treat.

If you have white spots on your nails because of an allergic reaction, stop using the product you believe to be the cause.

## **Prevention**

The following tips can help prevent white spots from developing on your nails:

* Protect your nails. Use protective gloves when doing activities that may damage your fingernails, including playing sports, working outside or using tools like a hammer. Wear comfortable and protective shoes, and be careful when putting down heavy objects near your feet.
* Moisturize your nails.
* Keep your nails trimmed short.
* Avoid irritating chemicals or products.

## **Prognosis**

## **When to Call the Doctor**

Contact a healthcare provider if:

* You develop new symptoms on or around your nails, including nails that easily crack or break (brittle), changes in color, or dents or ridges (Beau’s lines).
* You have white spots on your nails alongside more serious symptoms, including weakness, feeling tired all the time (fatigue), blurred vision, confusion (disorientation) and shortness of breath.
* Your symptoms don’t improve after treatment.

## **Possible Complication**

Leukonychia are a cosmetic nuisance but may be a marker of an underlying systemic disease, but per se do not have any physical complications.

## **Differential Diagnosis**

* Onychomycosis (also known as pseudoleukonychia) — the disease of the nail plate is due to external factors
* Onycholysis
* Nail psoriasis
* Trachyonychia (twenty-nail dystrophy)
* Vitiligo of the nail.

## **Reference**

<https://my.clevelandclinic.org/health/symptoms/25243-white-spots-on-nails#care-and-treatment>

Iorizzo M, Starace M, Pasch MC. Leukonychia: What Can White Nails Tell Us? Am J Clin Dermatol. 2022 Mar;23(2):177-193. doi: 10.1007/s40257-022-00671-6. Epub 2022 Feb 2. PMID: 35112320; PMCID: PMC8809498.

<https://pmc.ncbi.nlm.nih.gov/articles/PMC8809498/#Sec9>

**Ridges in nails**

Other nails: Beau’s lines, Furrows

## **Definition and Description**

Ridges in your nails are visible lines or dents in your fingernails or toenails. The ridges may run vertically (up and down) or horizontally (across) your nail. Ridges in your nails can be a sign of a health condition or a previous injury to your nail. In some cases, they’re harmless

### **Types**

### **Vertical ridges in nails**

Vertical ridges are furrows that run from the tip of your fingernail down to the cuticle. They are sometimes called longitudinal striations or bands.

According to 2015 research, slight vertical ridges in fingernails often develop in older adults, possibly due to a slowing of cell turnover. This is when new skin cells produced below the surface of your skin rise up to take the place of discarding dead cells from the surface.

### **Horizontal ridges in nails**

Deep horizontal ridges, called Beau’s lines, are often a symptom of serious conditions. They may actually stop nail growth until the underlying condition is treated.

If Beau’s lines appear, acute kidney disease may also be present.

## **Causes and Risk Factors**

Certain health conditions and nutrient deficiencies can cause ridges, dents or lines in your nails. Common causes of vertical nail ridges, also called longitudinal ridges, include:

* **Normal aging:** Many people develop vertical ridges on their nails as they get older. These lines aren’t dangerous.
* **Skin conditions:** If you have very dry skin or eczema, you may have vertical lines on your nails.
* **Thyroid disease:** If you have hypothyroidism, you may have thick, brittle nails with vertical ridges. Your nails may also crumble or break easily. They may look more rounded and your fingertip may be puffy.

Horizontal ridges or dents in your nails are usually due to a condition called Beau’s lines. These dents may happen when something interrupts your nail growth. Possible causes include:

* Chemotherapy.
* Damage from using artificial or acrylic nails or getting gel manicures for long periods.
* Injury to your nail, like slamming your finger in a door or dropping something on your foot.
* Peripheral vascular disease (PAD).
* Severe illness with high fever, such as COVID-19, measles or pneumonia.

According to a 2023 research review medications — particularly chemotherapy drugs — were the most common cause of Beau’s lines. Other causes included:

* non-autoimmune system diseases, such as:
  + mumps
  + parathyroid disease
  + unmanaged diabetes
  + syphilis
  + respiratory conditions
  + illnesses that cause prolonged high fevers
  + zinc deficiency
* trauma
* infection

Trauma to your nails can cause red or brown spots to form underneath your nails.

If you notice dark brown, black, or red color changes under your nails and haven’t experienced nail trauma, it may be a symptom of a more serious condition, like endocarditis or melanoma.

### **Can vitamin or mineral deficiencies cause** ridges **in nails?**

Certain nutritional deficiencies can cause changes to your nails. Zinc deficiency can cause Beau’s lines and white spots on your nails. Iron deficiency can cause vertical nail ridges and koilonychia (spoon nails). Spoon nails have a depression in the middle, like the center of your nail was scooped out. You may be able to hold a drop of water on your nail.

### **Other causes and symptoms**

#### **Skin conditions**

Many people with psoriasis experience problems with their fingernails and nail bed.

Eczema may also cause ridges and discoloration in the nail, along with other symptoms on the skin.

#### **Digestive disorders**

Digestive disorders that affect the absorption of nutrients may also influence the nails.

Crohn’s disease, celiac disease, and ulcerative colitis are all examples of disorders that can make it difficult for the body to absorb nutrition from food.

If the body does not have the proper building blocks to make new cells, the skin and nails may suffer.

#### **Injury**

An accident such as dropping a book on the fingernail can cause bruising to form underneath the nail and may temporarily change its shape.

Bruising can cause reddish-brown spots to form underneath the nail, which will go away gradually as the cells heal and the nail grows out.

If discoloration shows up without an accident or injury, it may be a sign of something more serious. Dark brown, purple, or red spots under the nails may be signs of serious conditions, such as melanoma or endocarditis.

### **Vertical ridges sign and symptoms**

If you experience other symptoms like texture changes in your nails, it may be caused by a medical condition. In trachyonychia (twenty-nail dystrophy), the ridges may look shiny or brittle.

Iron deficiency anemia can also trigger vertical ridges and changes to your nails that make them concave or spoon shaped.

## **Diagnosis Method**

A doctor should examine changes in your nails. If you damaged your nail in an injury, you may wait to see how the nail and your finger heal for a few weeks before deciding whether to see a doctor.

However, you should see a doctor as soon as possible if the injury results in:

* a clean or ragged cut through your nail
* a crushed nail
* a nail that is torn off
* bleeding under your nail

If you experience nail changes outside an injury, you’ll still want to be evaluated. During your appointment, your doctor will examine your nails and ask about any other symptoms you’re experiencing.

Your doctor may order urine and blood tests if they suspect kidney disease, diabetes, or nutritional deficiencies.

If it seems like the ridges are the result of a skin condition, a dermatologist can start you on a treatment plan.

If the cause of your fingernail ridges is unclear, a dermatologist may take some fingernail clippings to have them analyzed in a lab for signs of infection.

## **Treatment Option**

First, see a healthcare provider to find out the underlying cause. Treating the cause can usually improve the look of your nails and allow healthy nails to grow in.

For instance, medication for thyroid disease often improves or resolves related nail problems. Taking iron or zinc supplements — with your provider’s guidance — may help if you have a deficiency. However, it takes several months for your nails to grow out and it takes time to see a change in your nails.

If you have Beau’s lines from a previous illness, the lines should gradually grow out. But if you keep getting new Beau’s lines, tell your provider. Repeated Beau’s lines could be a sign that you have a condition that keeps interrupting your nail growth.

**What can I do at home to treat ridges in nails?**

If your provider has ruled out any health conditions, you can treat nail ridges with home care. The ridges may not go away completely, but you can improve the appearance of your nails if you:

* Apply moisturizer to your nails and cuticles daily to relieve dry skin or eczema. At bedtime, apply a skin cream to your hands and put on thin cotton gloves for sleeping. You can also apply a cream to your feet and wear cotton socks. If you have diabetes or neuropathy, talk to your healthcare provider about foot care.
* Ask your provider about taking biotin supplements, which could help with nail growth.
* Don’t bite your fingernails or cut your cuticles.
* Take breaks from getting gel manicures or using artificial nails or acrylics. Using these treatments continually can weaken your nails or dry them out.
* Trim your fingernails with a curved shape and your toenails straight across.
* Use a nail buffer to gently smooth the surface of your nails.
* Wear gloves that protect your hands when working with chemicals like household cleaners.

## **Prevention**

There’s no guaranteed way to prevent nail ridges. But seeing your provider as recommended can help. Tell your provider about any health changes, including changes to your nails. If you develop nail ridges, your provider can work with you to treat the underlying cause.

## **Prognosis**

Most of the time, ridges in fingernails are a typical sign of aging. However, it’s important to pay attention to fingernail ridges and other nail changes. These might be the first signs of a serious medical problem.

## **When to Call a doctor**

Always tell your provider if you notice new ridges or lines in your nails. You may not need treatment, but you won’t know until you find out what’s causing the ridges. Other nail changes to mention to a healthcare provider include:

* Changes to the shape of your nails, including nail clubbing or spoon-shaped nails.
* Color changes in your nails, including white, green, yellow or dark marks.
* Dents (nail pitting).
* Redness and swelling around one or more of your nails.

## **Reference**

<https://my.clevelandclinic.org/health/symptoms/24459-ridges-in-nails#overview>

<https://www.medicalnewstoday.com/articles/319867>

<https://www.healthline.com/health/ridges-in-fingernails#takeaway>

**Splinter hemorrhage**

Other names: haemorrhages

## **Definition and Description**

Splinter hemorrhages are small areas of bleeding (hemorrhaging) under your nails. They can affect your fingernails or toenails. They look like thin, red or reddish-brown lines below your nails. They run vertically in the direction of

your nail growth. Splinter hemorrhages get their name because they look like wood splinters under your nail.

Splinter hemorrhages occur when small blood vessels called capillaries under the skin beneath your nail (nail bed) burst due to damage. The leaking blood becomes visible through your nail. Splinter hemorrhages most often occur after you injure your nail, but sometimes they are a sign of a health condition.

## **Causes and Risk Factors**

### **causes**

When blood vessels under your nail bed sustain damage, splinter hemorrhages can occur. They can happen after you injure yourself. Trauma to your nail is the most common cause of the condition. Stubbing your toe or getting acrylic nails put on can cause splinter hemorrhages. If you have a splinter hemorrhage due to trauma or an injury, it will go away on its own and not cause any concern.

In some cases, splinter hemorrhages may be a sign of a medical condition that can damage your blood vessels. Infections, diseases and conditions that can cause the condition may include:

#### **Skin conditions**

Splinter hemorrhages are commonly seen in conditions such as nail psoriasis and lichen planus. Nail psoriasis is an autoimmune disorder that causes skin cells to collect on your nails. Lichen planus is an inflammatory skin condition that often results in an itchy rash. Up to 35% of people with lichen planus reported having splinter hemorrhages.

#### **Vasculitis and systemic diseases**

Splinter hemorrhages may be due to inflammation of your blood vessels (vasculitis). Several diseases such as antiphospholipid syndrome and lupus can cause vasculitis.

#### **Infectious diseases**

Bacterial infections such as endocarditis may cause splinter hemorrhages. Endocarditis is an infection of your heart valves. Studies show that 15% to 33% of people with endocarditis have splinter hemorrhages. People with chronic kidney disease also report them. This may be due to hemodialysis or a kidney transplant.

#### **Drug reactions**

Splinter hemorrhages are found in 60% to 70% of people taking kinase inhibitors such as sunitinib and sorafenib. Kinase inhibitors are cancer medications that stop cancer cells from growing. Splinter hemorrhages are also more common in people taking blood thinners such as aspirin, warfarin, apixaban or rivaroxaban.

#### **Other causes**

There are various other causes of splinter hemorrhages. They can occur because of a rare benign (noncancerous) tumor of your nail called onychomatricoma. They can look similar to a type of skin cancer called subungual melanoma.

### **Risk Factor**

Splinter hemorrhages can occur at any age; however, they are more common in older people.

* In healthy individuals, splinter hemorrhages occur more frequently in men than women.
* Splinter hemorrhages are more frequent in dark-skinned people than in light-skinned people.
* The characteristics of patients who develop splinter hemorrhages relate to their underlying cause.

## **Signs and Symptoms**

### **What does a splinter hemorrhage look like?**

Splinter hemorrhages resemble wood splinters under your nail. They look like tiny lines or streaks under your nail plate that run in the direction of your nail growth. They’re usually 1 to 3 millimeters long. They normally appear closer to the end of your nails, but any part of your nail may be affected.

When they first form, splinter hemorrhages appear reddish or purple, but they’ll darken to brown or black within a few days. They’ll usually move with your nail as it grows.

Splinter hemorrhages occur more often in fingernails than toenails. They usually only affect one nail in otherwise healthy people. It may be a sign of an underlying condition if you have the condition in more than one nail.

## **Diagnosis Method**

Your healthcare provider will perform a physical examination. They’ll also ask you about your symptoms and medical history. They may ask you:

* When you first noticed the splinter hemorrhage.
* If you’ve injured yourself recently or had an acrylic nail application.
* If the condition is affecting more than one nail.
* If you have other symptoms.
* About any other health conditions, you have.
* If you’re taking any prescription or over-the-counter medications.

Your healthcare provider may request a test called a dermoscopy. Using a dermoscope, a dermatologist can clearly see the splinter hemorrhage in order to help diagnose it.

## **Treatment Options**

Most splinter hemorrhages don’t require any treatment. If an injury caused the condition, it should go away on its own as the nail grows out.

If you don’t remember injuring yourself or you have splinter hemorrhages affecting more than one nail, contact your healthcare provider. They will determine treatment based on the underlying cause of the condition.

Your healthcare provider may prescribe you a medication or recommend an over-the-counter option if you have a disease such as nail psoriasis.

## **Prevention**

You can reduce your risk of developing splinter hemorrhages due to injury or trauma by keeping your nails strong. Eat healthy foods rich in vitamin B and zinc to help support nail growth. In addition, drink plenty of water.

You can prevent splinter hemorrhages caused by certain drugs by stopping or reducing the medication. Be sure to talk to your healthcare provider before making any changes to your medication.

Other causes of splinter hemorrhages can’t always be prevented. They are typically a symptom of an underlying condition.

## **Prognosis**

Splinter hemorrhages are harmless if caused by an injury or other trauma. They may look unpleasant but they’ll go away within a few days or when your nail grows out.

If you haven’t sustained an injury, a splinter hemorrhage may be a sign of a more serious condition. Contact your healthcare provider to determine the underlying cause of the condition. Your outlook (prognosis) will depend on the cause of the condition and treatment.

## **Possible Complications**

There are no complications of splinter hemorrhages themselves; complications arise as a consequence of the underlying disease process.

## **When to See a Doctor/ Red Flag**

Many splinter hemorrhages are due to trauma or injury and will clear up on their own. However, there are certain reasons you should contact your healthcare provider about the condition. These reasons include:

* If you don’t remember injuring yourself or sustaining any trauma.
* If the splinter hemorrhage doesn’t go away within a few days or when your nail grows out.
* If you keep getting splinter hemorrhages.
* If the condition is affecting more than one nail.

Reference

<https://my.clevelandclinic.org/health/diseases/23341-splinter-hemorrhage#overview>

<https://dermnetnz.org/topics/splinter-haemorrhage>

**HAIR DISEASES**

**Alopecia Areata**

Alopecia is the general medical term for hair loss.

Alopecia areata totalis means you’ve lost all the hair on your head.

Alopecia areata universalis is the loss of hair over your entire body.

Diffuse alopecia areata is a sudden thinning of your hair rather than lost patches.

Alopecia barbae, is round, smooth patchy hair loss on your beard.

Ophiasis alopecia areata causes hair loss in a band shape around the sides and back of your head.

**Definition and description**

Alopecia areata is an autoimmune disorder that causes hair to fall out, often in clumps the size and shape of a quarter. Alopecia can affect just your scalp or your entire body, and it can be temporary or permanent. It can be the result of heredity, hormonal changes, medical conditions or a normal part of aging. The disease attacks the hair follicles.

**Causes and risk factors**

· Family history (heredity). The most common cause of hair loss is a hereditary condition that happens with aging.

· Hormonal changes and medical conditions. A variety of conditions can cause permanent or temporary hair loss, including hormonal changes due to pregnancy, childbirth, menopause and thyroid problems.

· Autoimmune diseases causing patchy hair loss, scalp infections such as ringworm, and a hair-pulling disorder called trichotillomania.

· Medications and supplements. Hair loss can be a side effect of certain drugs, such as those used for cancer, arthritis, depression, heart problems, gout and high blood pressure.

· Radiation therapy to the head. The hair may not grow back the same as it was before.

· A very stressful event. Many people experience a general thinning of hair several months after a physical or emotional shock. This type of hair loss is temporary.

· Hairstyles and treatments. Excessive hairstyling or hairstyles that pull your hair tight, such as pigtails or cornrows, can cause a type of hair loss called traction alopecia. Hot-oil hair treatments and permanents also can cause hair to fall out. If scarring occurs, hair loss could be permanent.

· Iron deficiency

· Weight loss

· Cancer treatment, chemotherapy

**Risk factors**

· Asthma

· Down syndrome

· Pernicious anemia

· Seasonal allergies

· Thyroid disease

· Vitiligo

· Balding of a family member

· Age

· Significant weight loss

· Certain medical conditions, such as diabetes and lupus

· Stress

· Poor nutrition

**Signs and symptoms**

· Gradual thinning on top of head. This is the most common type of hair loss, affecting people as they age. In men, hair often begins to recede at the hairline on the forehead.

· Circular or patchy bald spots. Some people lose hair in circular or patchy bald spots on the scalp, beard or eyebrows.

· Sudden loosening of hair. A physical or emotional shock can cause hair to loosen. Handfuls of hair may come out when combing or washing your hair or even after gentle tugging. This type of hair loss usually causes overall hair thinning but is temporary.

· Full-body hair loss. Some conditions and medical treatments, such as chemotherapy for cancer, can result in the loss of hair all over your body. The hair usually grows back.

· Patches of scaling that spread over the scalp. This is a sign of ringworm. It may be accompanied by broken hair, redness, swelling and, at times, oozing.

· Small bald patches on your scalp or other parts of your body.

· Patches may get larger and grow together into a bald spot.

· Hair grows back in one spot and falls out in another.

· Tingling, itching, or burning sensation on your skin right before the hair falls out.

· Lose a lot of hair over a short time.

· More hair loss in cold weather.

· Fingernails and toenails become red, brittle, and pitted

**Diagnosis methods (tests, lab work, imaging, etc.)**

Physician Examination

Take a close look at the areas where you have hair loss

Pull gently on the hairs at the edges of the bald patch to see if they come out easily

Check individual hairs and follicles to see if they’re abnormally shaped

Examine your nails

Biopsy

Rarely, you may have a biopsy, in which a small piece of skin is removed from your scalp and looked at under a microscope.

Tests

Many conditions can cause hair loss. So, your doctor may test your skin for a fungal infection

Blood tests

Physicians may order blood tests to check for thyroid, hormone, or immune system problems.

**Treatment options (medications, therapies, surgeries, etc.)**

Alopecia areata can’t be cured. But it can be treated and hair can grow back. If you have it, there are several things to try:

Corticosteroids. These are anti-inflammatory drugs that are prescribed for autoimmune diseases. They can be given as an injection into the scalp or other areas. They can also be

given as a pill or rubbed on the skin as an ointment, cream, or foam. The downside is that it may take a long time to work.

Topical immunotherapy. This is used when there’s a lot of hair loss or if it happens more than once. Chemicals are applied to the scalp to produce an allergic reaction. If it works, this reaction is actually what makes the hair grow back. It also causes an itchy rash and usually has to be repeated several times to maintain the new hair growth.

Minoxidil (Rogaine). This treatment, which is put on the scalp, is commonly used for pattern baldness. It usually takes about 12 weeks before you see growth, and some users find the results disappointing. Read more about which types of alopecia are most likely to respond to minoxidil.

JAK inhibitors are oral prescription medications used to treat alopecia areata:

Leqselvi (deuruxolitinib)

Litfulo (ritlecitinib)

Olumiant (baricitinib)

Other treatments for alopecia areata include medications that are sometimes used for other autoimmune disorders. These medicines have differing amounts of success in regrowing hair.

**Management**

Apart from medications, there are other things you can try if you have alopecia areata.

· Wear wigs, hats, or scarves. They cover your hair loss and will protect your head from the sun.

· Reduce stress. Personal troubles seem to trigger alopecia areata, but this has not been proven scientifically.

Other home remedies for alopecia areata include:

· Styling products. Gels, mousses, powders, and sprays can help hide hair loss and add volume.

· A scalp prosthesis. This is a custom-made wig that fits perfectly on your head.

· Shaving. Some people choose to shave their head or other areas of their body to hide hair loss.

· Artificial eyelashes and eyebrows. You'll apply these synthetic or human hair brows or lashes with glue.

Prevention tips

Most baldness is caused by genetics (male-pattern baldness and female-pattern baldness). This type of hair loss is not preventable.

These tips may help you avoid preventable types of hair loss:

· Be gentle with your hair. Use a detangler and avoid tugging when brushing and combing, especially when your hair is wet. A wide-toothed comb might help prevent pulling out hair. Avoid harsh treatments such as hot rollers, curling irons, hot-oil treatments and permanents. Limit the tension on hair from styles that use rubber bands, barrettes and braids.

· Ask your doctor about medications and supplements you take that might cause hair loss.

· Protect your hair from sunlight and other sources of ultraviolet light.

· Stop smoking. Some studies show an association between smoking and baldness in men.

· If you're being treated with chemotherapy, ask your doctor about a cooling cap. This cap can reduce your risk of losing hair during chemotherapy.

**Prognosis**

· Patients had less favorable prognosis when the duration of hair loss was longer, and when they had a longer period from disease onset to hospital visit.

· The presence or absence of atopic disease did not significantly affect prognosis, nor did family history or nail involvement.

· Complete hair regrowth was more frequent when topical agents were used. In particular, complete hair regrowth was less when diphenylcyclopropenone immunotherapy and systemic immunosuppressants were used; however, this was because of the difference in the severity of the condition in patients who participated in the treatment

**Possible complications**

Some potential complications of alopecia areata include the following:

· Depression and anxiety, permanent hair loss, recurrence, nail abnormalities, sunburn, and skin damage.

· Unpredictable pattern, texture, and rate of hair regrowth.

· Increased risk of concurrent dermatologic and systemic illnesses such as thyroid disease, vitiligo, psoriasis, lupus erythematosus, and atopic dermatitis.

· Skin atrophy, hypopigmentation, infections, malignancy, dermatitis, and thrombosis due to adverse effects of medication.

· A 3-fold higher risk of developing retinal diseases such as retinal detachment, retinal vascular occlusion, and retinopathy.

**When to see a doctor / red flag**

See your doctor if you are distressed by persistent hair loss in you or your child and want to pursue treatment. For women who are experiencing a receding hairline (frontal fibrosing alopecia), talk with your doctor about early treatment to avoid significant permanent baldness.

Also talk to your doctor if you notice sudden or patchy hair loss or more than usual hair loss when combing or washing your or your child's hair. Sudden hair loss can signal an underlying medical condition that requires treatment.

**Differential diagnosis**

Specific medical conditions that may be mistaken for alopecia areata include androgenetic alopecia, traction alopecia, trichotillomania, tinea capitis, secondary syphilis, aplasia cutis, and temporal triangular alopecia.

**Epidemiology data**

· Alopecia areata occurs more often among Asian, Black, and Hispanic patients. Alopecia areata affects children and adults, and the incidence rises steadily with age. The mean age at onset is 32 for males and 36 for females.

· Studies reveal that dermatologic and systemic conditions associated with alopecia areata include vitiligo, lupus erythematosus, psoriasis, atopic dermatitis, thyroid disease, and allergic rhinitis.

· Patients with Down syndrome (trisomy 21) and polyglandular autoimmune syndrome have an increased risk of developing alopecia areata.

**Tinea Capitis (Scalp Ringworm)**

Tinea capitis is related to athlete's foot (tinea pedis), jock itch (tinea cruris) and ringworm of the body (tinea corporis).

**Definition and description**

Ringworm of the scalp (tinea capitis) is a rash caused by a fungal infection. It usually causes itchy, scaly, bald patches on the head. Ringworm gets its name because of its circular appearance. No worm is involved. It is a contagious infection. It's most common in toddlers and school-age children.

**Causes and risk factors**

· Ringworm of the scalp is caused by a common fungus. The fungus attacks the outer layer of skin on the scalp and the hair. This causes those hairs to break. The condition can be spread in the following ways:

· Human to human. Ringworm often spreads through direct skin-to-skin contact with an infected person.

· Animal to human. You can contract ringworm by touching an animal with ringworm. Ringworm can spread while petting or grooming dogs or cats with ringworm. Ringworm is fairly common in kittens, puppies, cows, goats, pigs and horses.

· Object to humans. It's possible for ringworm to spread by contact with objects or surfaces that an infected person or animal has recently touched. This includes items such as clothing, towels, bedding, combs and brushes.

**Risk factors**

Age. Ringworm of the scalp is most common in toddlers and school-age children.

Exposure to other children. Outbreaks of ringworm are common in schools and child care centers where the infection easily spreads with close contact.

Exposure to pets. A pet, such as a cat or a dog, can have the infection without showing any signs. Children can get the infection by touching the animal.

**Signs and symptoms**

· One or more round, scaly or inflamed patches where the hair has broken off at or near the scalp

· Scaly, dry, swollen or itchy rash.

· Patches that slowly get bigger and have small, black dots where the hair has broken off

· Brittle or fragile hair that can be easily broken or pulled out

· Tender or painful areas on the scalp

**Diagnosis methods (tests, lab work, imaging, etc.)**

Your doctor will likely be able to diagnose ringworm of the scalp by looking at the affected skin and asking certain questions.

To confirm the diagnosis, your doctor may take a sample of hair or skin to be tested in a lab. Testing a sample of hair or skin can show if a fungus is present.

**Treatment options (medications, therapies, surgeries, etc.)**

· Scalp ringworm is often treated with an antifungal medicine, griseofulvin, which you’ll take by mouth. Treatment may last for 4 to 8 weeks. Your doctor may also prescribe other medicines for scalp ringworm that you’ll take by mouth because creams, lotions, or powders alone don’t work for scalp ringworm. These medicines include:

· Itraconazole. This is prescribed in pill form for 7 or 15 days. It’s not for use in children, the elderly, or those with severe liver disease. While taking it, you may experience nausea, vomiting, indigestion, diarrhea, or headache. See your doctor if you don’t see any improvement in your symptoms or infection after you’ve finished your treatment.

· Terbinafine. You take this once a day for 4 weeks. Side effects usually are mild and don’t last long. They might include nausea, diarrhea, indigestion, and rashes. You won’t get a prescription for this if you have liver disease or lupus.

· Fluconazole (Diflucan). The dosage and length of time you'll need to be on this prescription vary from person to person.

· Steroid therapy: Steroid therapy to reduce inflammation and lower your risk of having hair loss when you have kerions

· Antifungal shampoos and creams to prevent the scalp ringworm from spreading

Best medicine for ringworm

Ringworm is treated with an over-the-counter antifungal cream in mild cases, and the best medicine will depend on where it is on your body and how much it has spread.

For example, if ringworm is on your hands, you may need stronger prescription antifungal medicine like itraconazole or terbinafine.

Duration of antifungal medicine

Depending on your case, you may need to take antifungal medicine for 2 to 4 weeks, but others may need treatment for up to 12 weeks. See a doctor if the ringworm worsens or doesn’t go away after treating it with over-the-counter or prescription medicines.

**Natural Ringworm Treatment**

· Tea tree oil for ringworm

Tea tree oil is an essential oil made from the leaves of the Australian tea tree. Early studies show it does work as an antifungal against ringworm. Other studies show that it works against athlete's foot when applied as a cream. You should only use tea tree oil topically as it is toxic if ingested.

· Apple cider vinegar for ringworm

Some sources suggest treating ringworm with apple cider vinegar by rubbing some on the infected area and covering it with a bandage. Studies show this vinegar does have some antifungal properties. However, doctors warn that, due to its acidic nature, apple cider vinegar can cause open sores and scarring when used to treat ringworm.

**Managements of Tinea capitis**

Keep your hands clean. Wash your hands each time you touch your rash and before you touch anyone else.

Wash your clothes, bedding, and towels. Wash everything you’ve touched or worn in hot water and laundry detergent.

Shower after workouts. This is especially important if you play contact sports.

Wear shower shoes. Protect your feet at the gym or pool.

Have your dog checked for fungal infection. Dogs can spread infection to you.

Clean the affected area with soap, and dry with a different towel from the rest of your body.

Wear fresh clothes, especially undergarments, every day.

Wash your clothes regularly and keep them dry when not in use.

Throw out or disinfect shoes in the case of athlete's foot.

**Prevention tips**

Ringworm of the scalp is difficult to prevent. The fungus that causes it is common, and the condition is contagious even before symptoms appear. Take these steps to reduce the risk of ringworm:

· Educate yourself and others. Be aware of the risk of ringworm from infected people or pets. Tell children about ringworm, what to watch for and how to avoid the infection.

· Shampoo regularly. Be sure to wash your child's scalp regularly, especially after haircuts. Some scalp conditioning products, such as coconut oil and pomades with selenium, might help prevent ringworm of the scalp.

· Keep skin clean and dry. Be sure children wash their hands, including after playing with pets. Keep shared areas clean, especially in schools, child care centers, gyms and locker rooms.

· Avoid infected animals. The infection often looks like a patch of skin where fur is missing. If you have pets or other animals that commonly carry ringworm, ask your veterinarian to check them for the infection.

· Avoid sharing personal items. Teach children not to let others use their clothing, towels, hairbrushes, sports gear or other personal items.

**Prognosis**

Tinea capitis has a good prognosis with treatment. However, those who remain untreated are at risk for the development of an abscess, also known as a kerion. The fungi can shed spores for many months leading to spread. A common cause of treatment failure is a lack of medication compliance. Tinea capitis usually has a good prognosis when treated early and appropriately.

**Possible complications**

Some people with ringworm of the scalp may develop a severe inflammation called kerion. Kerion appears as soft, raised swellings that drain pus and cause thick, yellow crusting on the scalp. With kerion, the hair falls out or can be easily pulled out. The condition may be caused by an overly vigorous reaction to the fungus and can lead to scarring and permanent hair loss.

**When to see a doctor / red flag**

· If the ringworm is on your skin and you’ve used an over-the-counter (OTC) antifungal medication for 2 weeks with no improvement, see your doctor. They can give you a prescription for something stronger.

· If it’s on your scalp, OTC treatments won’t work. Make an appointment so your doctor can give you a prescription medication that you can take by mouth.

· See your child's doctor if your child has any hair loss, scaling or itchiness of the scalp, or other unusual appearance of the scalp. It's important to get an accurate diagnosis and prompt treatment with prescription medicine. Nonprescription creams, lotions and powders won't get rid of ringworm of the scalp.

· There are a number of conditions that look like scalp ringworm but aren’t. If you’ve got an itchy, scaly scalp and you’re losing hair, have your doctor check it out. They’ll find out what’s behind it and find the right treatment.

**Differential diagnosis**

Dissecting folliculitis (folliculitis decalvans)

cellulitis

Bacterial folliculitis

Secondary syphilis

Abscess

Infected eczema

Pyoderma

Pustular psoriasis

Syphilis

Seborrheic dermatitis

Systemic lupus erythematosus

Drug eruption reaction

**Recent guidelines or updates**

Screening

All household contacts should be screened for tinea capitis. Asymptomatic individuals should be treated; otherwise, the cycle of transmission will continue.

The use of antifungal or selenium shampoo

This is recommended for 2 to 4 weeks. Teachers should be educated on tinea capitis and place infected children away from other healthy children. The sharing of personal care products should be avoided.

Antifungal resistance

Over the past decade, dermatophyte infections resistant to treatment with topical and oral antifungal agents have emerged. While tinea capitis infections resistant to antifungal therapy have been rarely reported to date, antifungal resistance is rising among superficial fungal infections in general, and antifungal stewardship is necessary to ensure that resistance to treatment does not develop among dermatophytes that cause tinea capitis.

**Epidemiology data**

Tinea Capitis infections occur worldwide, they are more common in developing countries with tropical or subtropical climates. Furthermore, socioeconomic conditions, including overcrowding and living in close proximity to animals, may influence the prevalence of Tinea capitis infections in certain communities across the world.

Adult women may be more frequently affected by Tinea capitis than adult men. Women in the postmenopausal years may be particularly susceptible, possibly due to decreased sebum production caused by a reduction in estrogen levels.

**Telogen Effluvium**

**Definition and description**

Telogen effluvium is the excessive shedding of resting or telogen hair after some metabolic stress, hormonal changes, or medication. Significant stress pushes large numbers of hair follicles into a resting phase. Within a few months, affected hairs might fall out suddenly when simply combing or washing your hair.

**Causes and risk factors**

Drugs

Numerous drugs can cause telogen hair loss and it usually starts after 12 weeks of dosage . Changes in the dosage of drugs can also lead to excessive shedding. Drugs that can cause telogen effluvium include oral contraceptive pills, androgens, retinoids, beta-blockers, ACE (angiotensin-converting enzyme) inhibitors, anticonvulsants, antidepressants, and anticoagulants (heparin).

Physiological Stress

Increased physiological stress such as surgical trauma, high fever, chronic systemic illness, and hemorrhage can cause telogen effluvium. Childbirth can also cause excessive hair to enter the telogen phase. This hair loss, telogen gravidarum, occurs approximately three months after childbirth.

Emotional Stress

The relationship between emotional stress and hair loss is ambiguous since hair loss itself is a source of emotional stress to the patient .

Medical Conditions

Numerous medical disorders can lead to telogen effluvium. Both hyper- and hypothyroidism can cause telogen effluvium, and this is reversed once the euthyroid state is achieved . Chronic systemic disorders such as systemic amyloidosis, hepatic failure, chronic renal failure, inflammatory bowel disease, and lymphoproliferative disorders can also cause telogen effluvium. It is also reported in some autoimmune diseases including dermatomyositis, chronic infections such as HIV, and secondary syphilis. Inflammatory disorders such as psoriasis and seborrheic dermatitis can also lead to diffuse telogen hair loss.

Dietary Triggers

Severe protein, fatty acid and zinc deficiency, chronic starvation, and caloric restriction can lead to telogen effluvium. Essential fatty acid deficiency leads to telogen effluvium, and this usually occurs two to four months after insufficient intake. Decreased body iron stores can cause it. However, this relationship is very controversial. Vitamin D is vital for cell growth and, hence, its deficiency could also be a possible cause of it. Another cause can be biotin deficiency but is reportedly very rare.

Ultraviolet Light

Researchers found an increased frequency of telogen effluvium between July and October. They hypothesized that it could be actinic effluvium, a summer effect, induced by sunlight and ultraviolet (UV) light, manifesting in autumn. Electron microscopy of hair exposed to sunlight reveals alterations in the cellular components and damage to the hair cuticle and cortex. Both of these mechanisms can be attributed to increased shedding of hair in the telogen phase; however, it is not scientifically proven yet

**Signs and symptoms**

· Trichodynia

A major symptom of telogen effluvium is trichodynia. It presents with complaints such as tenderness, pain, burning, itching, stinging, and diffuse alopecia .

· Modified Wash Test and Hair Loss Count

The modified wash test is an office procedure that permits the identification of patients with telogen effluvium or androgenetic alopecia, and the severity of diseases. It is performed after five days of abstention from shampooing. The patients are asked to wash and rinse their hair in a sink covered by gauze, collect the hair, let them dry, and put them in an envelope. Afterward, the collected hair is counted along with the percentage of vellus hair. The results and possible diagnosis are as follows: Telogen effluvium: More than 100 shed hair, less than 10% vellus. Androgenetic alopecia: Less than 100 shed hair, more than 10% vellus.

**Diagnosis methods (tests, lab work, imaging, etc.)**

Typically, obtaining a detailed history and performing a thorough physical examination are sufficient for diagnosing telogen effluvium. If a biopsy is performed during the acute shedding phase (when the pull test is positive), it can confirm an increase in the percentage of telogen hairs. Testing for underlying hormonal conditions, such as hypothyroidism; chronic metabolic illnesses; or iron deficiency is recommended if there are concerns about these conditions.

Trichogram

Trichogram is a plucking of hair in a defined area (40-60 hair). Cases of telogen effluvium show a significant reduction of the anagen:telogen ratio. More than 25% of hair are found to be in the telogen phase in the case of telogen effluvium.

Phototrichogram and TrichoScan

This technique involves trimming the hair of a 2 sq. cm area of scalp, pictures of the same area taken on different days, and then compared in hair density, hair growth, and rate of shedding. Since only anagen hair would elongate it helps in the assessment of the ratio of anagen:telogen hair. A TrichoScan is a fully computerized phototrichogram [20]. A TrichoScan is a simpler, noninvasive, reproducible, and more sensitive than a classical trichogram and very useful in the diagnosis of hair loss

**Laboratory Testing**

Hypothyroidism test

Hypothyroidism can lead to chronic telogen effluvium.

· When symptoms of hypothyroidism, such as tiredness, constipation, weight gain, and cold sensitivity, are present, a thyrotropin test is warranted.

**Iron deficiency test**

· Iron deficiency should be evaluated with a complete blood count, serum iron, iron saturation, and ferritin.

· Given the priority of blood for survival over hair, the body prioritizes shedding hair before red blood cell indices become microcytic.

· Ferritin acts as an acute-phase reactant, and inflammation can result in normal ferritin levels in an iron-deficient individual.

· Low ferritin confirms iron deficiency; a normal ferritin level does not exclude iron deficiency.

· Iron saturation is the most sensitive indicator of iron deficiency.

**Syphilis test**

· When symptoms of syphilis, such as fatigue, patchy hair loss, sore throat, or swollen lymph nodes, are present and considered a cause, a rapid plasma reagin or venereal disease research laboratory (VDRL) test should be performed.

Biopsy

· Scalp biopsy is the most useful test to confirm the diagnosis, but it is seldom necessary if gentle hair pull produces numerous telogen hairs.

· A white bulb and no gelatinous hair sheath can identify telogen hair.

· If a patient is unwilling to allow a scalp biopsy, serial hair collections can be obtained.

· The patient should be instructed to collect all shedding hair in 24 hours, refraining from washing their hair during this time. This process should be repeated every week for three or four collections.

· Collecting 100 or more hairs in 24 hours suggests telogen effluvium. The number of hairs collected may decrease if the collections are performed over several weeks while the telogen effluvium improves.

**Treatment options (medications, therapies, surgeries, etc.)**

Acute telogen effluvium is a self-limited condition. If the causative event has been identified and appropriately treated, there is no need for further treatment. If a hormonal or dietary imbalance, such as low iron levels, zinc levels, vitamin D levels; or metabolic illness is present, hair growth returns after these factors are corrected.

If a medication is the cause of the shedding, hair growth restarts after the medication is withdrawn.

Although topical minoxidil has not been proven to promote hair recovery in telogen effluvium, it has theoretical benefits. Patients who wish to take an active role in their treatment may choose to use topical minoxidil. Recent trials have proven that oral minoxidil can be an effective and well-tolerated treatment alternative for healthy patients with difficulty with topical formulations.

Recent studies have also shown that both botulinum toxin A and multivitamin mesotherapy are effective in treating telogen effluvium. These treatments demonstrated improvement of the criteria terminal hair and multiple follicular units.

Most importantly, all the patients should be offered emotional support and reassurance about the benign course of the disease and the possibility of complete recovery of hair with time and nutritional support.

Topical Corticosteroids

Topical corticosteroids are employed by dermatologists in the treatment. If the patient reports decreasing trichodynia after the application of topical corticosteroids, it is a sign of the therapy being effective

**Prevention tips**

· Eat extra protein, especially if you’re vegetarian or vegan. You need 40 to 60 grams of protein a day. The Mediterranean diet includes fruits, vegetables and protein that may help minimize hair loss.

· Take vitamins. Certain vitamins and minerals, including vitamins A, B, C, D, E, zinc, biotin and iron, help maintain healthy hair, skin and muscle tissue. Ask your healthcare provider before adding any new supplements to your diet.

· Find ways to cope with stress. Stress is one of the leading causes of telogen effluvium.

· Get enough sleep. Most adults need between seven and nine hours of sleep per night. The benefits of a good night’s sleep include decreased stress.

· Avoid extreme or restrictive diets. Rapid weight loss can trigger telogen effluvium. Restrictive diets can lead to nutritional deficiencies that can cause telogen effluvium. If you must lose weight, it’s a good idea to get regular exercise and follow a Mediterranean-style diet that focuses on fresh fruits and vegetables, whole grains, lean protein and healthy fats.

**Prognosis**

Telogen effluvium can be stressful, and you may fear that you’ll lose all of your hair. However, if you have telogen effluvium, the outlook is good. It usually goes away three to six months after you start noticing your hair loss. Your healthcare provider can also help you take steps to promote new hair growth.

**Possible complications**

Telogen effluvium is a benign and spontaneously reversible condition with no associated complications. As it is a non-cicatricial alopecia, the scalp has no scarring, even during the active hair loss phase.

**Differential diagnosis**

The differential diagnoses of telogen effluvium include alopecia areata, anagen effluvium, androgenetic alopecia, scarring alopecia, syphilis, and trichotillomania.

**Epidemiology and Demographics.**

Telogen effluvium is a condition that can affect individuals of any age, gender, or racial background. The exact prevalence of telogen effluvium is unknown, but it is considered quite common. Many adults experience an episode of telogen effluvium at some point in their lifetime. Although telogen effluvium can manifest in both men and women, women tend to be more susceptible because of postpartum hormonal changes. In addition, women are more disturbed by hair shedding compared to men and are more likely to seek medical attention.

**Trichorrhexis Invaginata**

Trichorrhexis invaginata is also known as bamboo hair

**Definition and description**

Trichorrhexis invaginata (bamboo hair) is an abnormality of the hair in which the hair shaft telescopes into itself (invaginates) at several points along the shaft. Trichorrhexis invaginata is a hair disorder characterized by the folding of the hair shaft into itself, resulting in a ball and socket appearance, also known as "Bamboo hair." It is often associated with Netherton disease, a rare genetic skin disorder that combines ichthyosis, bamboo hair, and atopic dermatitis .

**Causes and risk factors**

## **Causes of Trichorrhexis Invaginata**

The primary cause of Trichorrhexis invaginata, also known as bamboo hair, is a genetic defect that affects the hair shaft's structure, leading to fragile and easily breakable hair strands.

* Genetic factors
* Nutritional deficiencies
* Hair care practices
* Medical conditions
* Trauma or injury to the hair

**Types of Trichorrhexis Invaginata**

Trichorrhexis invaginata can present in various forms, each characterized by distinct features and manifestations in the hair structure.

* Bamboo hair syndrome: Characterized by fragile hair shafts with nodes and internodes, resembling a bamboo stalk.
* Netherton syndrome: An inherited disorder causing fragile, easily breakable hair due to a defect in the protein structure of the hair shaft.
* Trichorrhexis nodosa: Commonly caused by hair damage from excessive heat, chemicals, or mechanical stress, leading to nodes along the hair shaft.
* Menkes syndrome: A rare genetic condition resulting in kinky hair with a flattened, twisted appearance due to a copper metabolism disorder.
* Pili torti: Hair shaft abnormality where the hair is twisted at irregular intervals, causing a fragile and brittle texture.

## **Risk Factors**

Trichorrhexis invaginata, a hair shaft disorder, can be caused by genetic factors or associated with conditions like Netherton syndrome or structural abnormalities of the hair shaft.

* Genetic factors
* Structural hair abnormalities
* Overuse of hair styling tools
* Chemical hair treatments
* Nutritional deficiencies
* Hormonal imbalances

**Signs and symptom**

Dry, dull, fragile, and short.

Hair strands that have a knotty appearance

Loss of eyelashes

Loss of eyebrows

Sparse hair growth or hair loss pattern

Dry hair

Hair that lacks luster

Spiky hair

Short hair due to consistent breakage

Hair on the eyebrows that resembles matchsticks

**Diagnosis methods (tests, lab work, imaging, etc.)**

High magnifications produced by a video dermoscope show invagination of the distal portion of the hair shaft into its proximal portion, forming a “ball-in-cup” appearance.

To diagnose bamboo hair, your doctor will pluck a hair from your scalp to observe it under a microscope.

To diagnose Netherton syndrome, your doctor may order a series of DNA tests or a skin biopsy to test for gene mutations. For a skin biopsy, your doctor will remove a small amount of skin tissue for testing in a lab. DNA tests are often used to test the SPINK5 gene for abnormalities

## **Treatment for Trichorrhexis Invaginata**

Trichorrhexis invaginata is typically managed through a combination of supportive measures and targeted interventions to improve hair health and prevent further damage.

* Topical Treatments: Application of emollients or keratolytic agents to soften and smoothen the affected hair shafts, promoting healthy hair growth and reducing breakage.
* Avoiding Harsh Hair Treatments: Minimizing the use of heat styling tools, chemical relaxers, and hair dyes to prevent further damage to the hair shafts.
* Gentle Hair Care Practices: Using a wide tooth comb, gentle detangling techniques, and avoiding tight hairstyles to prevent hair breakage and worsening of Trichorrhexis invaginata.
* Nutritional Supplements: Consuming a balanced diet rich in vitamins, minerals, and proteins to support overall hair health and strengthen the hair shafts.
* Regular Trims: Getting regular trims to remove split ends and prevent further damage to the hair, promoting healthier hair growth.

Since the condition is a direct result of a gene mutation, there’s no current, known way to prevent the condition. But there are many types of lotions and ointments you can use to treat bamboo hair. These include:

emollients and keratolytics (especially those with urea, lactic acid, and salicylic acid) to moisturize your skin

antibiotics for infections in the skin and elsewhere

antihistamines for itching of the skin

topical steroids, but these shouldn’t be used on infants

photochemotherapy (PUVA) and oral retinoids

**Prognosis**

Although the condition can’t be prevented or fully cured because it’s the result of a genetic mutation, there are ways to manage your symptoms by hydrating your hair and healing your skin.

Avoid chemicals that dry your hair and scalp. Use hair care products that hydrate your hair. Ointments and lotions can lessen symptoms, too.

The condition also improves with age, even if it’s left untreated.

## Diagnostic Considerations

Also consider the following:

* Pediculosis
* Peripilar casts
* Dermatophytosis
* Trichothiodystrophy
* Argininosuccinic aciduria
* Deposits of extraneous material
* Hypothyroidism
* Trichorrhexis Invaginata (Netherton Syndrome or Bamboo Hair)

Patients with syndromic diarrhea, also known as phenotypic diarrhea or tricho-hepato-enteric syndrome, have a congenital enteropathy with a distinct hair abnormality characterized by woolly hair that is easily removed and poorly pigmented. Hair-shaft microscopic analysis may show twisted hair (pili torti), anisotrichosis and poilkilotrichosis, trichorrhexis nodosa and longitudinal breaks, and trichothiodystrophy.

A feature of normal healthy Black African hair is an apparent increased fragility, with certain similarities to that reported for trichorrhexis nodosa (weathering secondary to physical damage).This excessive structural damage is probably due to physical trauma (resulting from grooming) rather than an inherent weakness due to any structural abnormality.

## **Differential Diagnoses**

* Alopecia Areata
* Anagen Effluvium
* Androgenetic Alopecia
* Dermatologic Manifestations of Menkes Kinky Hair Disease
* Monilethrix
* Piedra
* Seborrheic Dermatitis
* Trichomycosis Axillaris
* Trichomycosis Pubis
* Trichorrhexis Invaginata (Netherton Syndrome or Bamboo Hair)
* Trichotillomania

## **Epidemiology**

Trichorrhexis nodosa is an uncommon disorder. A retrospective review of 129 hair-mount samples from 119 patients over a 10-year span found 25 cases of loose anagen hair syndrome, 6 cases of uncombable hair syndrome, and trichorrhexis nodosa in 13 patients.

Acquired proximal trichorrhexis nodosa is common in Blacks and appears to occur in individuals who are genetically predisposed. Some consider it an ethnic hair disorder.Acquired distal trichorrhexis nodosa primarily occurs in Asian or White persons. Acquired proximal trichorrhexis nodosa is more common in females than in males.

Congenital trichorrhexis nodosa may be present at birth, or it may appear within the first couple months of life. It can present in patients with the late form of argininosuccinic aciduria at age 2 years or older.

[Trichorrhexis Nodosa Differential Diagnoses](https://emedicine.medscape.com/article/1073664-differential?form=fpf)

**Lichen Planopilaris**

**Definition and description**

Lichen planopilaris is an inflammatory, primary cicatricial alopecia, resulting in several hair loss patterns. Alopecia is considered a follicular variant of lichen planus. The condition is considered a follicular variant of lichen planus based on clinical and histopathological findings. The most widely accepted theory states that lichen planopilaris is a hair-specific autoimmune disorder in which activated T-lymphocytes target follicular antigens.

Lichen planopilaris results in patchy, progressive, permanent hair loss, mainly affecting the scalp while also extending to other hair-bearing regions such as the eyebrows and pubic area.

**There are three types of lichen planopilaris**:

**Classic lichen planopilaris**: Scarring causes bald patches on your scalp.

**Frontal fibrosing alopecia:** Bald patches and scarring appear at your hairline near your forehead. You may also lose hair in your eyelashes or eyebrows.

**Lassueur-Graham-Little-Piccardi syndrome:** You may have scarring and bald patches on your scalp, as well as thinning hair in your armpits and groin. You may also notice rough bumps around your hair follicles.

**Causes and risk factors**

· Hepatitis C infection.

· Pain relievers and other medicines.

· An allergic reaction to the metal in dental fillings.

· Autoimmune diseases

**Signs and symptoms**

· Bald patches on your scalp.

· Red, thick or scaly patches of skin on your scalp.

· Scalp pain, itching or burning.

· LPP is a form of lichen planus, a condition that causes an itchy rash on your arms and legs and in your mouth. Lichen planus can also cause ridges or splitting of your nails. Nearly half of people with LPP also develop symptoms of lichen planus.

**Diagnosis methods (tests, lab work, imaging, etc.)**

The diagnosis is based on the clinicopathologic correlation rather than solely on clinical signs and symptoms. A 4-mm-deep punch biopsy specimen should be submitted for horizontal sectioning and hematoxylin-eosin staining. The best biopsy site is an active, symptomatic hair-bearing area with perifollicular erythema and perifollicular scale located at the margin of a bare patch with a positive anagen pull test

**Treatment options (medications, therapies, surgeries, etc.)**

The main objectives of treatment are to reduce hair loss, control the symptoms, and stop the scarring process. Regrowth should not be expected as the complete elimination of inflammation is unlikely. As no consistent markers measure the disorder's progress, therapy is based on perceived severity and patient tolerance to treatment. The duration of treatment should be guided by clinical response and relapse rates. General measures include avoiding chemical or physical insults to the hair, such as coloring or perming. Contrary to many patients' beliefs, the frequency of shampooing does not impact overall hair loss. Potent corticosteroids and topical tacrolimus are commonly used in all forms of primary cicatricial alopecia and are frequently considered first-line treatments.

Antimalarial drugs are commonly used to treat lichen planopilaris. Hydroxychloroquine at a dose of 200 mg twice daily is generally used and is often considered first-line systemic therapy. Improvement is often observed within 6 months.

Minoxidil helps maximize the hair growth of the remaining follicles. Recently, JAK inhibitors such as baricitinib and tofacitinib have been used to treat lichen planopilaris.

A new scoring system, the lichen planopilaris activity index, has been introduced to monitor treatment response and document disease progression. This system assigns numerical values to subjective and objective markers of the disease: symptoms (pruritus, pain, burning) and signs (erythema, perifollicular erythema, perifollicular scale), a measure of the activity (anagen pull test), and spreading of the condition—statistical comparison of pretreatment and posttreatment responses.

**Prevention tips**

There is no known way to prevent lichen planopilaris (LPP). But if you notice skin changes or hair loss, don’t ignore it. Early treatment for LPP may help prevent future scarring and hair loss.

**Prognosis**

The prognosis of lichen planopilaris can vary widely among affected individuals, influenced by several factors such as the extent and severity of scalp involvement, the presence of associated signs and symptoms, the effectiveness of the treatment regimen, and individual patient characteristics. Overall, alopecia is considered a chronic and progressive condition that can lead to significant morbidity, particularly in cases of extensive and severe disease.

**Possible complications**

Permanent hair loss (cicatricial alopecia): Lichen planopilaris can result in irreversible destruction of hair follicles, leading to scarring and permanent hair loss in affected areas of the scalp.

Scarring and fibrosis: The chronic inflammation characteristic of lichen planopilaris can lead to significant scarring and fibrosis within the affected scalp tissue. This scarring may cause scalp tightness, pain, and discomfort and can also affect the ability of remaining hair follicles, decreasing their functionality.

Follicular hyperkeratosis: Lichen planopilaris can accumulate keratin around the hair follicles, leading to hyperkeratosis. This condition may manifest as rough, scaly patches on the scalp, contributing to itching, irritation, and further inflammation.

Secondary infections: Persistent inflammation and compromised skin barrier function may subject affected individuals to secondary bacterial or fungal infections of the scalp area. These infections can exacerbate existing symptoms such as itching, pain, and inflammation and may require additional treatment with antibiotics or antifungal medications.

Psychological impact: The visible changes associated with lichen planopilaris, including hair loss, scarring, and scalp abnormalities, can have a noteworthy psychological impact on affected individuals, leading to decreased self-esteem, anxiety, and depression. Therefore, managing the psychosocial aspects of lichen planopilaris is an essential component of comprehensive care for these patients.

Ocular involvement: In rare cases, lichen planopilaris can extend beyond the scalp and involve the eyebrows and eyelashes. Eyebrow and eyelash loss can occur, leading to cosmetic concerns and potential eye irritation or discomfort.

**Differential diagnosis (how it’s distinguished from other illnesses)**

The most important differential diagnosis may be seborrheic dermatitis, as many patients have a long history of scalp scaling, often diagnosed initially as seborrheic dermatitis. A sudden onset of patchy hair loss on the scalp may be diagnosed as alopecia areata. However, if there's only partial hair loss, perifollicular erythema, and scaling within the patch, lichen planopilaris is considered a diagnosis of high suspicion.

**Epidemiology data**

The incidence of any of the cicatricial alopecias is not precisely known. Lichen planopilaris has been reported as the most frequent primary scarring alopecia, accounting for 43% of cases in a series involving 72 patients. Frontal fibrosing alopecia and Graham-Little syndrome are considered variants of lichen planopilaris. The alopecia typically affects women between ages 40 and 60, more often compared to men. Up to 50% of patients may develop characteristic lichen planus lesions that affect the skin, mucous membranes, or nails

**Folliculitis**

**Definition and description**

Folliculitis is a common skin condition that happens when hair follicles become inflamed. It's often caused by an infection with bacteria. At first it may look like small pimples around the tiny pockets from where each hair grows (hair follicles).

The condition can be itchy, sore and embarrassing. The infection can spread and turn into crusty sores.

Mild folliculitis will likely heal without scarring in a few days with basic self-care. More-serious or repeat infections may need prescription medicine. Left untreated, severe infections can cause permanent hair loss and scarring.

Certain types of folliculitis are known as hot tub rash and barber's itch.

**Causes and risk factors**

Folliculitis is often caused when hair follicles are infected with bacteria, commonly Staphylococcus aureus (staph). It may also be caused by viruses, fungi, parasites, medications or physical injury. Sometimes the cause isn't known.

**Risk factors**

Regularly wearing clothing that traps heat and sweat, such as rubber gloves or high boots

Soaking in a hot tub, whirlpool or public pool that's not maintained well

Causing damage to hair follicles through shaving, waxing, wearing tight clothes or hair styling practices such as traction, wigs and oils

Using some medications, such as corticosteroid creams, prednisone, long-term antibiotic therapy for acne and certain chemotherapy drugs

Having dermatitis or excessive sweating (hyperhidrosis)

Having diabetes, HIV/AIDS or another condition that lowers your resistance to infections

**Signs and symptoms**

Clusters of small bumps or pimples around hair follicles

Pus-filled blisters that break open and crust over

Itchy, burning skin

Painful, tender skin

An inflamed bump

**Diagnosis methods (tests, lab work, imaging, etc.)**

Your health care provider will likely be able to tell whether you have folliculitis by looking at your skin and asking about your medical history.

If early treatments don't clear up your infection, your health care provider may run some tests.

**Tests**

Scraping of the skin to look for yeast under the microscope

Obtaining a swab for culture to determine the cause of infection

**Biopsy**

Rarely, doing a skin biopsy to rule out other conditions

A standard KOH preparation can be used to visualize hyphae and spores associated with folliculitis caused by Malassezia. KOH preparation could also be used to diagnose Demodex folliculitis; however, this is not common in clinical practice. Also, a skin biopsy is usually required to confirm the diagnosis of eosinophilic folliculitis.

**Treatment options (medications, therapies, surgeries, etc.)**

Treatments for folliculitis depend on the type and severity of your condition, what self-care measures you've already tried, and how you'd like to proceed.

If you've tried nonprescription products for a few weeks and they haven't helped, ask your health care provider about prescription-strength medications. A dermatologist can help you:

Control your folliculitis

Figure out whether a drug you take might be causing your symptoms and whether you can stop taking it

Avoid scarring or other damage to the skin

Make scars less noticeable

Even if treatment helps, the infection may come back. Talk with your health care provider about the risks of the treatments you're considering.

**Medications**

Lotions, gels or pills to control bacterial infection. For mild infection caused by bacteria, your health care provider may prescribe an antibiotic lotion or gel. Infection-fighting pills (oral antibiotics) aren't routinely used for folliculitis, but you may need them for a severe or repeat infection.

Creams, shampoos or pills to fight fungal infections. Antifungals are for infections caused by yeast rather than bacteria. Antibiotics aren't helpful in treating this type of folliculitis.

Creams or pills to calm inflammation. If you have mild eosinophilic folliculitis, your health care provider may suggest you try a steroid cream to ease the itching. If you have human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), you may see improvement in your eosinophilic folliculitis symptoms after antiretroviral therapy.

Other interventions

Minor surgery. If you have a large boil or carbuncle, your health care provider may make a small cut in it to drain the pus. This may relieve pain, speed recovery and lower the risk of scarring. Your health care provider may then cover the area with sterile gauze to absorb any leaking pus.

Laser hair removal. Your health care provider may suggest laser hair removal as an option for pseudofolliculitis barbae, especially when other treatments haven't improved your symptoms. This treatment often requires multiple visits to the health care provider's office.

Talk with your health care provider about possible side effects of laser treatment. They include scarring and skin that lightens (hypopigmentation) or darkens (hyperpigmentation).

**Prevention tips**

Wash your skin regularly. Use a clean washcloth and towel each time and don't share your towels or washcloths.

Do laundry regularly. Use hot, soapy water to wash towels, washcloths and any oil-soaked uniforms or other clothing.

Avoid friction or pressure on your skin. Protect skin that's prone to folliculitis from the friction caused by backpacks, helmets and tight clothes.

Dry out your rubber gloves between uses. If you wear rubber gloves regularly, after each use turn them inside out, wash with soap, rinse and dry well.

Avoid shaving, if possible. For people with facial folliculitis, growing a beard may be a good option if you don't need a clean-shaven face.

Using a washcloth or cleansing pad in a gentle circular motion to raise embedded hairs before shaving

Applying a good amount of shaving lotion before shaving

Shaving in the direction of hair growth

Avoiding shaving too close by using an electric razor or guarded blade and by not stretching the skin

Using a clean, sharp blade and rinsing it with warm water after each stroke

Avoiding shaving the same area more than twice

Applying moisturizing lotion after you shave

Avoiding the sharing of razors, towels and washcloths

Try hair-removing products (depilatories) or other methods of hair removal. Though they, too, may irritate the skin.

Treat related conditions. If you know that a condition other than folliculitis is triggering your symptoms, treat that condition. For example, excessive sweating (hyperhidrosis) can cause folliculitis. You can try to prevent this by changing out of sweaty clothing, bathing daily and using antiperspirant.

Use only clean hot tubs and heated pools.

Talk with your healthcare provider. If your folliculitis often returns, your health care provider may suggest controlling bacterial growth in your nose. You might need a five-day course of prescription antibacterial ointment. And you may need to use a body wash with chlorhexidine (Hibiclens, Hibistat, others).

**Prognosis**

As this condition is generally benign and often self-limiting, the outlook and prognosis are very good for a full recovery. With proper hygiene and management of any underlying conditions, recurrence rates can remain minimal.

**Possible complications**

Recurrent or spreading infection

Permanent scarring

Patches of skin that are darker (hyperpigmentation) or lighter (hypopigmentation) than before the condition occurred, usually temporary

Destruction of hair follicles and permanent hair loss

**When to see a doctor / red flag**

Make an appointment with your health care provider if your condition is widespread or the symptoms don't go away after a week or two of self-care measures. You may need a prescription-strength antibiotic or antifungal medication to help control the condition.

Seek immediate medical care if you experience signs of a spreading infection. These include a sudden increase in redness or pain, fever, chills, and a feeling of being unwell (malaise).

**Differential diagnosis**

* Acne vulgaris
* Papulopustular rosacea
* Drug-induced folliculitis
* Hidradenitis suppurativa
* Scabies
* Pseudofolliculitis barbae
* Keratosis pilaris
* Acne keloidalis nuchae

**Recent guidelines or updates**

The Centers for Disease Control also suggests that after getting out of the water you remove your swimsuit and shower with soap. Then wash your swimsuit too. If you own a hot tub or a heated pool, clean it regularly and add chlorine as recommended.

**Epidemiology data**

While the precise incidence of folliculitis is not currently known, we do know that patients who have a history of diabetes, obesity, prolonged use of oral antibiotics, are immunosuppressed/immunocompromised or who shave frequently are at risk for developing this condition. While gender does not correlate with an increased incidence of folliculitis, there may be a correlation between the type of folliculitis and gender. For example, Malassezia folliculitis is commonly seen in men more than women.

**Pili torti**

**Definition and description**

Pili torti is a rare condition characterized by the presence of the hair shaft, which is flattened at irregular intervals and twisted 180° along its long axis. It is a form of hair shaft disorder with increased fragility.

Pili torti, also known as “twisted hair”, was first described by Galewsky, and, independently, by Ronchese in 1932. It is characterized by the presence of the hair shaft, flattened at irregular intervals and twisted 180° along its long axis, with each twist being 0.4 to 0.9 mm wide and occurring in groups of 3 to 10

**Causes and risk factors**

Pili torti is caused by genetic mutations, also known as pathogenic variants. Genetic mutations can be hereditary, when parents pass them down to their children, or they may occur randomly when cells are dividing. Genetic mutations may also result from contracted viruses, environmental factors, such as UV radiation from sunlight exposure, or a combination of any of these

## **Risk Factors**

Pili torti is a rare hair condition characterized by fragile, twisted hair shafts. While the exact cause is often unknown, several risk factors may contribute to its development. These include genetic factors, certain medical conditions like various syndromes, nutritional deficiencies, autoimmune disorders, and exposure to environmental factors such as chemicals or radiation. Understanding these risk factors can help healthcare professionals in diagnosing and managing pili torti effectively.

* Genetic predisposition: Individuals with a family history of pili torti are at higher risk of developing the condition.
* Certain medical conditions: Underlying conditions such as Menkes disease or ectodermal dysplasia can increase the likelihood of pili torti.
* Environmental factors: Exposure to certain chemicals, toxins, or radiation may contribute to the development of pili torti.
* Nutritional deficiencies: Inadequate intake of essential nutrients like biotin or zinc can be a risk factor for pili torti.
* Hair care practices: Overuse of heat styling tools, harsh chemical treatments, or frequent braiding can increase the risk of pili torti.

**Signs and symptoms**

Clinically, patients with pili torti have fragile, brittle, dry, and coarse hair. Patchy alopecia may develop. The scalp hair, especially in the occipital and temporal areas, is most commonly affected.

The hairs become dry, lusterless, fragile and brittle. The condition may be associated with neurosensorial deafness1 and is probably caused by changes in the internal hair sheath.

**Diagnosis methods (tests, lab work, imaging, etc.)**

The diagnosis of pili torti is based on trichoscopic and microscopic examination.

Trichoscopy, hair and scalp dermoscopy, is a rapid technique that is useful in the diagnosis of scalp and hair diseases as well as genetic disorders, including ectodermal dysplasias. It can be performed with a manual dermoscope (10 magnification) or a video dermoscope (20–1000 magnification). This noninvasive method replaced light microscopy, which required pulling of multiple hairs for investigation. This is particularly burdensome in cases, where only a few hairs might be affected.

In pili torti, low magnification trichoscopy reveals the hair shafts bent at different angles and at irregular intervals. Regular twists of the hair shaft along the long axis are observed at high magnification

**Treatment options (medications, therapies, surgeries, etc.)**

There is no specific treatment of pili torti. The avoidance of trauma to the hair is recommended.

Other forms of management include:

Sleeping on a satin pillowcase,

Avoiding excessive grooming, braiding, heat treatments, and dying.

Gentle shampoos may be beneficial

Congenital pili torti may improve spontaneously after puberty. Drug-induced cases tend to resolve after the discontinuation of the offending agent.

In regard to acquired pili torti, the treatment of the underlying condition is most important.

Efficacy of pharmacological treatment in pili torti is limited.

Topical minoxidil has been suggested as a beneficial therapeutic option for patients with hair shaft abnormalities with increased fragility. However, it only has an impact on hair density and does not induce a causal treatment.

**Prognosis**

There is no specific treatment for pili torti. It may improve spontaneously after puberty. If pili torti is detected, further evaluation to investigate possible neurological disorders, ectodermal disturbances and hearing loss is mandatory.1 In the case of our patient, we did not identify any changes. The patient remains in outpatient follow-up and shows good psychomotor development.

**Trichotillomania**

**Definition and description**

Trichotillomania, also called **hair-pulling disorder**, is a mental health condition. It involves frequent, repeated and irresistible urges to pull out hair from your scalp, eyebrows or other areas of your body. You may try to resist the urges, but you can't stop. Trichotillomania is part of a group of conditions known as body-focused repetitive behaviors.

Pulling out hair from the scalp often leaves patchy bald spots. This can cause a lot of distress and can affect your work, school and social life. You may go to great lengths to hide the hair loss.

For some people, trichotillomania may be mild and can be managed. For others, the automatic or deliberate urge to pull out hair is too much to handle emotionally. Some treatment options may help reduce hair pulling or stop it entirely

**Causes and risk factors**

The cause of trichotillomania is not clear. But like many complex disorders, trichotillomania likely results from a combination of genetic and learned factors.

**Risk factors**

These factors tend to increase the risk of trichotillomania:

· Family history. Genetics may play a role in the development of trichotillomania. You may be more likely to have the condition if you have a close relative with trichotillomania.

· Health conditions. Some people may have hair or skin conditions that feel uncomfortable. This may focus their attention toward pulling hair or picking at their scalp.

· Age. Trichotillomania usually develops just before or during the early teens — most often between the ages of 10 and 13 years. It's often a lifelong problem. Babies may pull out their hair, but this is usually mild and goes away on its own without treatment.

Other mental health conditions. Other conditions, such as depression, anxiety or obsessive-compulsive disorder (OCD) may occur along with trichotillomania.

· Stress. Severely stressful situations or events may trigger trichotillomania in some people.

· Environment. Boredom, isolation and privacy often increase the likelihood of hair pulling.

Although far more women than men are treated for trichotillomania, this may be because women are more likely to seek medical advice. In early childhood, trichotillomania occurs just as often in boys and girls

**Signs and symptoms**

Symptoms of trichotillomania often include:

· Repeatedly pulling out your hair, whether it's automatic or on purpose, usually from your scalp, eyebrows or eyelashes, but sometimes from other body areas. The sites may vary over time.

· An increasing sense of tension before pulling out your hair, or when you try to resist pulling.

· A sense of pleasure or relief after the hair is pulled out.

· Hair loss that's easy to see, such as shortened hair or thinned or bald areas on the scalp or other areas of your body. This may include thin or missing eyelashes or eyebrows.

· Pulling out specific types of hair, taking the same steps in the same way each time hair is pulled out or pulling out hair in certain patterns.

· Biting, chewing or eating pulled-out hair.

· Playing with pulled-out hair or rubbing it across your lips or face.

· Repeatedly trying to stop pulling out your hair or trying to do it less often without success.

· Experiencing a great deal of distress or problems at work, school or in social situations related to pulling out your hair.

Often trichotillomania also includes picking your skin, biting your nails or chewing your lips. Sometimes pulling out hairs from pets or dolls or from materials, such as clothes or blankets, may be a sign. Pulling out hair is usually done in private. An episode can last from a few seconds to hours. You may try to hide your condition from others.

With trichotillomania, pulling out hair can be:

Automatic. You may pull out your hair without even realizing that you're doing it. This might happen, for example, when you're bored, reading or watching TV.

Focused. You may pull out your hair on purpose to relieve tension or distress. You may develop specific rituals for pulling out hair, such as finding just the right hair. You may play with, bite or eat pulled-out hairs.

You may do both automatic and focused hair pulling, depending on the situation and your mood. Certain positions or activities may trigger pulling out hair, such as resting your head on your hand or brushing your hair.

Trichotillomania can be related to emotions, including:

Negative feelings. Pulling out hair may be a way of dealing with negative or uncomfortable feelings, such as stress, anxiety, tension, boredom, loneliness, extreme tiredness or frustration.

Positive feelings. You may find that pulling out hair feels satisfying and provides some relief. As a result, you may continue to pull out your hair to keep these positive feelings.

Trichotillomania is a long-term disorder. If not treated, symptoms may come and go for weeks, months or years at a time. Also, symptoms can vary in severity over time. For example, hormone changes during the menstrual period can worsen symptoms in some females. Rarely, pulling out hair ends within a few years of starting.

**Diagnosis methods (tests, lab work, imaging, etc.)**

To diagnose trichotillomania, you'll likely start by having a physical exam. You may then be referred to a mental health professional with experience in treating trichotillomania. Diagnosing trichotillomania may include:

Examining your hair loss.

Checking for possible medical causes of your hair loss. This may include lab tests.

Talking with you about hair loss, including your behaviors and emotions related to pulling out your hair.

Identifying any physical or mental health conditions that may occur along with pulling out your hair.

**Treatment options (medications, therapies, surgeries, etc.)**

Some treatment options have helped many people reduce hair pulling or stop completely. These include therapy and sometimes medicine.

**Therapy**

Types of therapy that may be helpful for trichotillomania include:

· **Habit reversal training.** This behavior therapy is the main treatment for trichotillomania. You learn how to recognize situations where you're likely to pull out your hair and how to substitute other behaviors instead. For example, you might clench your fists to help stop the urge. One form of habit reversal training, called decoupling, involves quickly redirecting your hand from your hair to another location when you feel the urge to pull out your hair. Other therapies may be used along with habit reversal training.

· **Acceptance and commitment therapy.** This therapy can help you learn to accept your hair-pulling urges without acting on them.

· **Cognitive therapy.** This therapy can help you identify and examine beliefs you have about hair pulling that are not realistic. You can learn healthy ways to think about your condition.

Therapies that help with other mental health conditions that often occur along with trichotillomania, such as depression, anxiety, or problems with alcohol or drug use, can be an important part of treatment.

**Medicines**

Although no medicines are approved by the U.S. Food and Drug Administration specifically for the treatment of trichotillomania, some medicines may help control certain symptoms, such as anxiety and depression.

For example, your health care provider may recommend an antidepressant, such as clomipramine (Anafranil). Research suggests that N-acetylcysteine, an amino acid that affects mood, also may help. Another option that research suggests may have benefits is olanzapine (Zyprexa). This drug is used to treat certain serious mental health conditions that affect the mind.

Talk with your health care provider about any medicine recommended. The possible benefits of medicines should be balanced against possible side effects.

**Prevention tips**

There is no proven way to prevent trichotillomania, but getting treatment as soon as symptoms start can be a big help. Learning stress management is also a good idea because stress often triggers hair pulling behavior.

**Prognosis**

The overall outlook for this condition depends partly on the age of the person who has it. Infants and children with TTM often have the best outlook, with the condition commonly going away on its own. The prognosis is better when the disorder is diagnosed early, and treatment begins early. It is also associated with a better prognosis the younger the age of occurrence.

However, the older a person gets — especially from adolescence onward — the greater the odds that treating the condition becomes difficult. TTM on its own is rarely a life-threatening problem. But its impacts on a person’s life, especially their mental health, are often severe. Because of this, early diagnosis and treatment are very important.

**Possible complications**

· **Emotional distress**. You may feel frustrated, ashamed and embarrassed because of your condition and hair loss. You may feel that you don't have control over pulling out your hair. You may experience low self-esteem, depression, anxiety, and problems with alcohol or drugs.

· **Problems in your social life and with work**. Hair loss may lead you to avoid social activities and school and job opportunities. You may wear wigs, style your hair to disguise bald patches or wear false eyelashes. You may avoid intimacy to hide your condition.

· **Skin and hair damage.** Constantly pulling out hair can cause scarring, infections and other damage to the skin on your scalp or the area where hair is pulled out. This can permanently affect hair growth.

· **Hairballs**. Eating your hair may lead to a large, matted hairball that stays in your digestive tract. Over a period of years, the hairball can cause weight loss, vomiting, an intestinal block and even death.

**When to see a doctor / red flag**

If you can't stop pulling out your hair or you feel embarrassed or ashamed by your appearance as a result, talk to your health care provider. Trichotillomania is not just a bad habit, it's a mental health condition. It's not likely to get better without treatment.

**Differential diagnosis**

Neurodevelopmental disorders, such as Autism Spectrum Disorder and Stereotypic Movement Disorder, can also include repetitive hair pulling. However, in TTM, hair pulling may seem driven, but unlike stereotypies, it is not purposeless, and the movements are not always rhythmic. Also, neurodevelopmental disorders manifest during early childhood, whereas TTM often emerges later.

Some have noted that disorders involving affect regulation and self-injurious behaviors (e.g., Borderline Personality Disorder-BPD), can have features similar to TTM. Although there are surface similarities, in that self-injury and pulling may regulate emotion and lead to noticeable physical damage, there are important differences between the two conditions. First, individuals engaging in self-injurious behavior often do so to purposely experience pain whereas individuals with TTM do not generally experience this phenomenon. Second, self-injury tends to be more episodic, compared to pulling, which is more habitual in nature.

Other forms of hair loss must be placed on the differential diagnosis. Examples include traction alopecia, male pattern baldness, pressure alopecia, alopecia areata, tinea capitis, short-term habit, obsessive-compulsive disorder, and systemic diseases like cancer, lupus, hypothyroidism, and factitious disorder.[[](https://www.ncbi.nlm.nih.gov/books/NBK493186/)

**Recent guidelines or updates**

Trichotillomania falls under the overall category of obsessive-compulsive disorder, but it has some key differences from OCD itself.

Obsessions. OCD involves obsessions, which are thoughts or urges that a person can’t control and doesn’t want. TTM doesn’t involve obsessions.

Feeling of reward. When people with TTM pull out their hair, they often feel relief or other positive emotions. OCD doesn’t involve positive feelings in that way.

**Epidemiology data**

Beginning in adolescence, the lifetime prevalence of TTM is reported as high as 3.5%. The adolescent patients do not all meet the criteria for trichotillomania as described by the DSM-V criteria but they do experience some form of the symptoms. The disorder is reported more commonly in females, with the ratio shown to be about 9:1 toward females. It is thought that the stigma of the disorder creates underreporting in general.

**Hirsutism**

**Definition and description**

Hirsutism means the growth of excessive male-pattern hair in women after puberty. It affects facial and body areas dependent on androgens, namely mustache and beard, pubic hair, buttocks, and thighs. It is a frequent reason for dermatological consultation . Hirsutism is the most common endocrine disorder affecting nearly 10% of women in the United States.

Irrespective of the cause, hirsutism can cause significant emotional stress and mental anguish. The key is to find the cause and address the cosmetic issue.

**Causes and risk factors**

Hirsutism may be caused by:

· **Polycystic ovary syndrome (PCOS).** This condition, which often begins with puberty, causes an imbalance of sex hormones. Over years, PCOS may slowly result in excess hair growth, irregular periods, obesity, infertility and sometimes multiple cysts on the ovaries.

· **Cushing syndrome.** This occurs when your body is exposed to high levels of the hormone cortisol. It can develop from your adrenal glands making too much cortisol or from taking medications such as prednisone over a long period.

· **Congenital adrenal hyperplasia.** This inherited condition is characterized by abnormal production of steroid hormones, including cortisol and androgen, by your adrenal glands.

· **Tumors.** Rarely, an androgen-secreting tumor in the ovaries or adrenal glands can cause hirsutism.

· **Medications.** Some medications can cause hirsutism. These include minoxidil (Minoxidil, Rogaine); danazol, which is used to treat women with endometriosis; testosterone (Androgel, Testim); and dehydroepiandrosterone (DHEA). If your partner uses topical products containing androgens, you can be affected as well, through skin-to-skin contact.

Often hirsutism occurs with no identifiable cause.

## **Risk factors**

Several factors can influence your likelihood of developing hirsutism, including:

· **Family history.** Several conditions that cause hirsutism, including congenital adrenal hyperplasia and polycystic ovary syndrome, run in families.

· **Ancestry.** Women of Mediterranean, Middle Eastern and South Asian ancestry are more likely to have more body hair with no identifiable cause than are other women.

· **Obesity.** Being obese causes increased androgen production, which can worsen hirsutism.

**Signs and symptoms**

Hirsutism is stiff or dark body hair, appearing on the body where women don't commonly have hair — primarily the face, chest, lower abdomen, inner thighs and back. People have widely varying opinions on what's considered excessive.

When high androgen levels cause hirsutism, other signs might develop over time, a process called virilization. Signs of virilization might include:

· Deepening voice

· Balding

· Acne

· Decreased breast size

· Increased muscle mass

· Enlargement of the clitoris

**Diagnosis methods (tests, lab work, imaging, etc.)**

Tests that measure the amount of certain hormones in your blood, including testosterone or testosterone-like hormones, might help determine whether elevated androgen levels are causing your hirsutism.

Your doctor might also examine your abdomen and do a pelvic exam to look for masses that could indicate a tumor.

**Treatment options (medications, therapies, surgeries, etc.)**

Treatment of hirsutism with no sign of endocrine disorder is not necessary. For women who do need or seek treatment, it may involve treating any underlying disorder, developing a self-care routine for unwanted hair, and trying various therapies and medications.

### **Medications**

If cosmetic or self-care methods of hair removal haven't worked for you, talk with your doctor about drugs that treat hirsutism. With these medications it usually takes up to six months, the average life cycle of a hair follicle, before you see a significant difference in hair growth. Options include:

· **Oral contraceptives.** Birth control pills or other hormonal contraceptives, which contain estrogen and progestin, treat hirsutism caused by androgen production. Oral contraceptives are a common treatment for hirsutism in women who don't want to become pregnant. Possible side effects include nausea and headache.

· **Anti-androgens.** These types of drugs block androgens from attaching to their receptors in your body. They're sometimes prescribed after six months on oral contraceptives if the oral contraceptives aren't effective enough.

The most commonly used anti-androgen for treating hirsutism is spironolactone (Aldactone, CaroSpir). The results are modest and take at least six months to be noticeable. Possible side effects include menstrual irregularity. Because these drugs can cause birth defects, it's important to use contraception while taking them.

· **Topical cream.** Eflornithine (Vaniqa) is a prescription cream specifically for excessive facial hair in women. It's applied directly to the affected area of your face twice a day. It helps slow new hair growth but doesn't get rid of existing hair. It can be used with laser therapy to enhance the response.

### **Procedures**

Hair removal methods whose results may last longer than self-care methods — and which may be combined with medical therapy — include:

· **Laser therapy.** A beam of highly concentrated light (laser) is passed over your skin to damage hair follicles and prevent hair from growing (photoepilation). You might need multiple treatments. For people whose unwanted hair is black, brown or auburn, photoepilation is usually a better option than electrolysis.

Talk with your doctor about the risks and benefits of the various lasers used for this hair removal method. People with tanned or darkly pigmented skin are at increased risk of side effects from certain lasers, including a darkening or lightening of their usual skin tones, blistering, and inflammation.

· **Electrolysis.** This treatment involves inserting a tiny needle into each hair follicle. The needle emits a pulse of electric current to damage and eventually destroy the follicle. You might need multiple treatments. For people with naturally blond or white hair, electrolysis is a better option than laser therapy.

Electrolysis is effective but can be painful. A numbing cream spread on your skin before treatment might reduce discomfort.

## **Self care**

Self-care methods such as the following temporarily remove or reduce the visibility of unwanted facial and body hair. There is no evidence that self-removal of hair leads to heavier hair growth.

· **Plucking.** Plucking is a good method to remove a few stray hairs, but is not useful for removing a large area of hair. Plucked hair usually regrows. This hair removal method may be done with tweezers, thin threads (threading) or other devices designed for this purpose.

· **Shaving.** Shaving is quick and inexpensive, but it needs to be repeated regularly.

· **Waxing.** Waxing involves applying warm wax on your skin where the unwanted hair grows. Once the wax hardens, you pull it from your skin to remove hair. Waxing removes hair from a large area quickly, but it may sting temporarily and sometimes causes skin irritation and redness.

· **Depilation.** Chemical depilatories are applied to the affected skin, where they dissolve hair. These products are available in a variety of forms, such as gel, cream or lotion. They may irritate the skin and cause dermatitis. You'll need to repeat depilation regularly to maintain the effect.

· **Bleaching.** Bleaching lightens hair color, making it less noticeable on people with light skin. Hair-bleaching products, which usually contain hydrogen peroxide, may cause skin irritation. Test any product you use on a small area of skin first.

**Prevention tips**

Hirsutism generally isn't preventable. But losing weight if you're overweight might help reduce hirsutism, particularly if you have polycystic ovary syndrome.

**Prognosis**

Hirsutism has significant morbidity and some women with a malignant cause tend to have a very poor prognosis. Postmenopausal hirsutism has been associated with a high risk of osteoporosis and fractures.

**Possible complications**

Hirsutism can be emotionally distressing. Some women feel self-conscious about having unwanted hair. Some develop depression. Also, although hirsutism doesn't cause physical complications, the underlying cause of a hormonal imbalance can.

If you have hirsutism and irregular periods, you might have polycystic ovary syndrome, which can inhibit fertility. Women who take certain medications to treat hirsutism should avoid pregnancy because of the risk of birth defects.

**When to see a doctor / red flag**

If you think you have too much coarse hair on your face or body, talk with your doctor about treatment options.

Excess facial or body hair is often a symptom of an underlying medical problem. See your doctor for assessment if over a few months you experience severe or rapid hair growth on your face or body or signs of virilization. You may be referred to a doctor who specializes in hormone disorders (endocrinologist) or skin problems (dermatologist).

**Differential diagnosis**

Androgen-Secreting Adrenal Tumors

Androgen-Secreting Ovarian Tumors

Congenital Adrenal Hyperplasia

Exogenous Androgens

Iatrogenic Cushing Syndrome

Idiopathic Hirsutism

Polycystic Ovary Syndrome (PCOS) Imaging

**Epidemiology data**

The exact prevalence of hirsutism is not well known. It may be 10% or even higher than 50%. The psychological problem that this disease causes depends on ethnic and socio-cultural factors. Indeed, in some societies where the lack of hair is considered an important criterion of female beauty, minimal hirsutism could be considered a severe disorder, while much more pronounced hirsutism could be accepted in other societies.

Hirsutism appears to be most common in dark-skinned individuals. Hirsutism can occur in men but is difficult to recognize. In children, hirsutism is a sign of precocious puberty.

Hirsutism is also known to occur in women who discontinue the oral contraceptive pill and gain weight.

**Monilethrix**

**Definition and description**

Monilethrix is characterized by a regular, periodic thinning of the hair-shaft, leading to a characteristic beaded appearance of the hair. The term monilethrix comes from "monile" (Latin) and "thrix" (Greek), meaning "necklace" and "hair" respectively, which emphasizes the clinical resemblance of the hair to a string of beads or a necklace.

Monilethrix is a rare structural hair shaft disorder characterized by hair fragility and resulting in patchy dystrophic alopecia

**Causes and risk factors**

In most cases, monilethrix is inherited as an autosomal genetic trait.

Monilethrix is caused by mutations in one of several genes. Mutations in the KRT81 gene, the KRT83 gene, the KRT86 gene, or the DSG4 gene account for most cases of monilethrix. These genes provide instructions for making proteins that give structure and strength to strands of hair

**Signs and symptoms**

In most cases of monilethrix, the hair is normal at birth; it may then be slowly replaced by abnormal hair during the first few months to two years of life. In some rare cases, the hair may be abnormal at birth (congenital). The hair may be sparse, dry, lusterless, and/or brittle. In addition, the hair is unusually short and breaks off before growing longer than a few inches.

Scalp hair is most frequently affected by monilethrix. The entire scalp or small areas of the scalp may be involved. In some cases, the eyelashes, eyebrows, pubic hair, and/or other body hair may also be affected. In addition, the patchy loss of hair (alopecia) is a common characteristic of this disorder. Progressive hair loss may lead to scattered bald patches or baldness.

In most cases of monilethrix, a skin condition known as perifollicular hyperkeratosis may develop. The condition is characterized by firm dark lesions (papules) covered with gray-brown scales and crusts that appear on the skin, especially the scalp.

The severity and progression of symptoms may vary greatly from case to case. In some cases, individuals with monilethrix may experience remission of the disorder for no apparent reason (spontaneously), most often during puberty or pregnancy. In other cases, the condition may remain the same throughout life or the symptoms may become progressively worse.

**Diagnosis methods (tests, lab work, imaging, etc.)**

The diagnosis of monilethrix may be confirmed by a thorough clinical evaluation and microscopic examination of the hair. When viewed under a microscope, the hair resembles a string of evenly-spaced beads

**Treatment options (medications, therapies, surgeries, etc.)**

To date, there is no known successful cure for this hair condition.

Topical minoxidil, oral acitretin, griseofulvin, systemic corticosteroids, and peeling ointments have shown to be effective in some cases. When used in low doses, oral minoxidil seems to be a promising and well-tolerated treatment for monilethrix. Also, there have been reports of cases treated with N-acetyl cysteine, but symptoms recurred after an initial improvement.

Wigs may be a consideration for cosmetic purposes, but there have been suggestions that friction caused by wig adhesives may exacerbate hair loss.

The cornerstone of the management of monilethrix remains the avoidance of chemical and mechanical damage caused by excessive hair combing or washing and friction. Hence, the education of the patient and his/her parents relating to lifestyle modification is necessary.

**Prognosis**

The prognosis of monilethrix considerably varies among affected individuals. Some cases remit spontaneously in adulthood, whereas most cases are persistent throughout life.

There have been suggestions that hair shine improvement and regrowth of apparently normal hair may occur in summer, with pregnancy and at the time of puberty. Hormonal effects have also been suggested to play a role in recovery as there has been a report of clinical improvement in a patient after her first menstrual period.

**Possible complications**

Monilethrix can predispose affected patients to low self-esteem and negative body perception, hence the need to assess its impact on the quality of life. Other than psychological impact, complications related to the treatment modalities.

**Differential diagnosis**

Some other hair shaft anomalies merit consideration in the differential diagnosis of monilethrix, such as pseudo-monilethrix, congenital alopecia, trichorrhexis invaginata, and ectodermal dysplasia

**Epidemiology data**

The prevalence and incidence are not known. No racial nor sex predilection is known for monilethrix, and it is not related to any particular hair color.

**Head Lice** **(pediculosis capitis)**

**Definition and description**

Head lice are tiny insects that feed on blood from the human scalp. Head lice most often affect children. The insects usually spread through direct transfer from the hair of one person to the hair of another.

Having head lice isn't a sign of poor personal hygiene or an unclean living environment. Head lice don't carry bacterial or viral diseases.

Nonprescription and prescription medications can help treat head lice. Follow treatment instructions carefully to rid the scalp and hair of lice and their eggs.

People also use a number of home or natural remedies to get rid of head lice. But there is little to no clinical evidence that they're effective.

**Causes and risk factors**

A head louse is a tan or grayish insect about the size of a strawberry seed. It feeds on human blood from the scalp. The female louse produces a sticky substance that firmly attaches each egg to the base of a hair shaft less than 1/4 inch (5 millimeters) from the scalp.

The louse life cycle

A louse goes through three stages:

Eggs that hatch after 6 to 9 days.

Nymphs, immature forms of the louse that become mature adults after 9 to 12 days.

Adult lice, which can live for 3 to 4 weeks. The female louse lays 6 to 10 eggs a day.

**Transmission**

Head lice crawl, but they can't jump or fly. Head lice often spread from one person to another by direct head-to-head contact, often within a family or among children who have close contact at school or play.

It's less common for head lice to spread without direct contact. But the insects may spread from one person to another through personal items, such as:

Hats and scarves

Brushes and combs

Hair accessories

Headphones

Pillows, towels and upholstery

Head lice may also spread when items of clothing are stored together. For example, hats or scarves hung on the same hook or stored in the same school locker could serve as vehicles for spreading lice.

Household pets, such as dogs and cats, don't play a role in spreading head lice.

**Risk factors**

Head lice are spread primarily by direct head-to-head contact. So the risk of spreading head lice is greatest among children who play or go to school together. In the United States, cases of head lice most often occur in children in preschool through elementary school.

**Signs and symptoms**

Common signs and symptoms of head lice may include:

· Itching. The most common symptom of head lice is itching on the scalp, neck and ears. This is an allergic reaction to louse bites. When a person has head lice for the first time, itching may not occur for 4 to 6 weeks.

· Lice on scalp. You may be able to see the lice, but they're often hard to spot because they're small, avoid light and move quickly.

· Lice eggs (nits) on hair shafts. Nits stick to hair shafts and may be hard to see because they're very tiny. They're easiest to spot around the ears and the hairline of the neck. Empty nits may be easier to spot because they're lighter in color and further from the scalp. However, the presence of nits doesn't mean there are live lice.

· Sores on the scalp, neck and shoulders. Scratching can lead to small, red bumps that may sometimes get infected with bacteria.

**Diagnosis methods (tests, lab work, imaging, etc.)**

According to the American Academy of Pediatrics guidelines, the gold standard for diagnosing head lice is to identify a live nymph or adult louse.

The guidelines recommend examining wet hair lubricated with hair conditioner or another product. Your child's health care provider will carefully comb your child's hair with a fine-toothed comb (nit comb) from the scalp to the end of the hair. If no live louse is found, the provider will likely repeat the entire exam at a second appointment.

**Identifying nits**

Your health care provider will also look for nits in your child's hair. To find nits, your child's provider may use a specialized light called a Wood's light, which causes nits to appear bluish. But the identification of nits does not necessarily confirm the diagnosis of live lice.

A live nit needs to be near the scalp to survive. Nits found more than about 1/4 inch (6 millimeters) from the scalp are likely dead or empty. Suspect nits can be examined under a microscope to determine if they're living.

If the provider doesn't find any live nits, they're probably left from a previous case of head lice and don't need to be treated.

**Treatment options (medications, therapies, surgeries, etc.)**

Your health care provider will likely recommend a medication available without a prescription that kills lice and some of the nits. These medications may not kill recently laid eggs. Therefore, an appropriately timed second treatment is usually necessary to kill nymphs after they hatch but before they become adult lice.

Some studies suggest that re-treating 7 to 9 days after the first treatment is the ideal time for a second treatment, but other re-treatment schedules exist. Ask your health care provider for written instructions for a recommended treatment schedule.

**Nonprescription products**

Medications available without a prescription include:

**Permethrin (Nix).** Permethrin is a synthetic version of pyrethrin, which is a chemical compound extracted from the chrysanthemum flower. Permethrin is toxic to lice.

Before using permethrin, wash your child's hair with shampoo but not conditioner. Rinsing the hair with white vinegar before washing may help dissolve the glue that holds the nits to the hair shafts. Leave the medication in the hair for the amount of time indicated in the directions on the package. Then rinse your child's hair over a sink with warm water.

Permethrin doesn't kill nits, and treatment needs to be repeated 9 to 10 days after first application. Side effects may include redness and itching of the scalp.

**Ivermectin (Sklice).** Ivermectin is toxic to lice. The lotion is approved for use in adults and children age 6 months or older. It can be applied once to dry hair and then rinsed with water after 10 minutes.

**Prescription medications**

In some regions, lice have developed resistance to nonprescription medications. Nonprescription treatment also may fail because of incorrect use, such as not repeating the treatment at an appropriate time.

If the correct use of a nonprescription treatment has failed, your health care provider may recommend a prescription treatment. These include:

**Spinosad (Natroba).** Spinosad is approved for adults and children age 6 months and older. It can be applied to dry hair and rinsed with warm water after 10 minutes. It kills lice and nits and usually doesn't need repeated treatment.

**Malathion**. Malathion is approved for adults and children age 2 or older. The lotion is applied, left to dry naturally and rinsed out after 8 to 12 hours. The drug has a high alcohol content, so it can't be used with a hair dryer or near an open flame. Malathion can be reapplied 7 to 9 days after the first treatment if necessary.

**Ivermectin (Stromectol).** In addition to the nonprescription lotion, ivermectin is available by prescription as a tablet to be taken by mouth. It can be given to children weighing more than 33 pounds if other topical treatments don't rid the scalp of head lice.

**Home remedies**

If you prefer not to use a medication for treating head lice, you may consider a home treatment. However, there's little to no clinical evidence that home treatments are effective.

**Wet-combing**

Combing wet hair with a fine-toothed nit comb may remove lice and some nits. Studies show that wet-combing results vary.

Start by wetting the hair and lubricating it with hair conditioner or olive oil. Comb the entire head from the scalp to the end of the hair at least twice during a session. The process typically should be repeated every 3 to 4 days for several weeks — at least two weeks after no more lice are found.

**Essential oils**

Small clinical studies have suggested that some natural plant oils may kill lice by depriving them of air, but effectiveness is uncertain. These products include:

Tea tree oil

Anise oil

Ylang-ylang oil

Essential oils aren't required to meet safety, efficacy and manufacturing standards used for drugs approved by the Food and Drug Administration (FDA), and can sometimes cause allergic reactions.

**Smothering agents**

A number of household products are used to treat head lice. These products are thought to deprive the lice of air when generous amounts are applied to the hair, covered with a shower cap and left on overnight. Products used for this purpose include:

**Mayonnaise**

**Olive oil**

**Margarine or butter**

**Petroleum jelly**

However, it's unclear if these treatments are effective.

**Dehydration**

Another option is a machine that uses one application of hot air to kill head lice and their eggs through dehydration. The machine requires special training and is currently available only at professional lice treatment centers.

The machine uses air that is cooler than most hair dryers and at a much higher flow rate to kill the lice by drying them out. Don't use a regular hair dryer to accomplish this result as it's too hot and could burn the scalp.

**Dangerous products to avoid**

Flammable products, such as kerosene or gasoline, should never be used to kill lice or to remove nits.

**Household cleaning**

Lice usually don't live past one day without feeding from a human scalp. And eggs can't survive without the temperature near the scalp. Therefore, the chance of lice surviving on household items is small.

As a precaution, you may clean items that the affected person has used in the previous two days. Cleaning recommendations include the following:

Wash items in hot water. Wash bedding, stuffed animals and clothing in hot, soapy water — at least 130 degrees Fahrenheit (54.4 degrees Celsius) — and dry at high heat.

Clean hair care items. Clean combs, brushes and hair accessories by soaking them in hot, soapy water for 5 to 10 minutes.

Seal items in plastic bags. Seal items that can't be washed in plastic bags for two weeks.

Vacuum. Give the floor and upholstered furniture a good vacuuming.

**Prevention tips**

It's difficult to prevent the spread of head lice among children in child care facilities and schools because there is so much close contact.

The chance of indirect spread from personal items is slight. However, to help prevent head lice from spreading, you may tell your child to:

Hang clothes on a separate hook from other children's clothes

Avoid sharing combs, brushes, hats and scarves

Avoid lying on beds, couches or pillows that have been in contact with a person who has head lice

It's not necessary to avoid sharing protective headgear for sports and bicycling when sharing is required.

**Prognosis**

The prognosis of louse infestations is generally good. When used appropriately, the medications are very effective in eradicating nymphs and mature lice. Treatment failure can be the result of several causes, including lack of ovicidal activity, failure to remove live nits, non-compliance-especially with retreatment in 7-10 days, inadequate application of the pediculicide (ie, duration, amount), failure to treat close contacts, insufficient environmental eradication, and drug resistance to the pediculicide. Some patients with body lice may contract a louse-borne infection such as trench fever, typhus, or relapsing/recurrent fever, but these are rare.

**Possible complications**

If your child scratches an itchy scalp due to head lice, it's possible for the skin to break and develop an infection.

**When to see a doctor / red flag**

See a health care provider before you begin treatment if you suspect that you or your child has head lice. Your or your child's health care provider can confirm that head lice are present. Studies show that many children have been treated for head lice with nonprescription medications or home remedies when they didn't have lice.

Things often mistaken for nits include:

Dandruff

Residue from hair products

Beads of dead hair tissue on a hair shaft

Scabs, dirt or other debris

Other small insects found in the hair

**Differential diagnosis (how it’s distinguished from other illnesses)**

Differential diagnosis for pediculosis includes:

* Dandruff
* Seborrhea
* Superficial fungal infection
* Eczema
* Folliculitis
* Scabies

**Recent guidelines or updates**

The CDC does not have scientific evidence that suffocating head lice with mayonnaise, olive oil, margarine, butter, or similar substances is an effective form of treatment.

**Epidemiology data**

Head lice infestation is a main public health problem and a great threat for personal hygiene involving children aged between 5 years and 13 years. The disease is widespread around the world but is more common in developing countries, among school age children, and in crowded places with low socio-economic and poor hygiene conditions. Indicators concerning the infestation of head lice are used to assess the health, cultural, and economic situation of rural and urban communities.

The infestation rate was found to be associated with low educational level of parents, long hair, family size, mother's job (housewife), father's job (worker/unemployed), using a common comb, lack of bathrooms in the house, and a low frequency of bathing**.**

According to a 2022 review of studiesTrusted Source, global estimates suggest about 19% of school-aged children have head lice.

Head lice are found in every country and across cultures and socioeconomic classes. In short, lice are nothing to feel embarrassed about.

**References**

[https://www.nhs.uk](https://www.nhs.uk/)

mayoclinic.org

<https://www.webmd.com/>

<https://pmc.ncbi.nlm.nih.gov/articles>

<https://www.ncbi.nlm.nih.gov/>

<https://my.clevelandclinic.org/health/diseases/24486-telogen-effluvium>

Rakowska, A., Olszewska, M., Rudnicka, L. (2012). Trichorrhexis Invaginata and Netherton’s Syndrome. In: Rudnicka, L., Olszewska, M., Rakowska, A. (eds) Atlas of Trichoscopy. Springer, London.<https://doi.org/10.1007/978-1-4471-4486-1_9>

<https://taylorandfrancis.com/knowledge/Medicine_and_healthcare/Dermatology/Trichorrhexis_invaginata>

<https://www.healthline.com/health/trichorrhexis-nodosa>

Yang JJH, Cade KV, Rezende FC, Pereira JM, Pegas JRP. Clinical presentation of pili torti - Case report. An Bras Dermatol. 2015;90(3 Suppl 1):S29-31.

rarediseases.org/rare-diseases/monilethrix/#

<https://www.healthline.com/health/how-common-are-lice>

<https://www.cdc.gov/lice/treatment/index.html>

### 

### 

### **Hair loss in women**

**DEFINITION AND DESCRIPTION**

Hair loss in women is just that — when females experience unexpected, heavy loss of hair. Generally, humans shed between 50 and 100 single hairs per day. Hair shedding is part of a natural balance — some hairs fall out while others grow in. An interruption in this balance — when hair falls out and less hair grows in — causes hair loss. The medical term for hair loss is “alopecia.”

Hair grows on almost all of your skin surfaces — not the palms of your hands, soles of your feet, lips or eyelids. Light, fine, short hair is called vellus hair. Terminal hair is thicker, darker and longer.

#### **What are the cycles of hair growth?**

Hair goes through three cycles:

* The anagen phase (growing phase) can last from two years to eight years. This phase generally refers to about 85% to 90% of the hair on your head.
* The catagen phase (transition phase) is the time that hair follicles shrink and takes about two to three weeks.
* The telogen phase (resting phase) takes about two to four months. At the end of this phase, the hair falls out.

Your shorter hairs — like eyelashes, arm and leg hair, and eyebrows — have a short anagen phase (about one month). Your scalp hair can last up to six years or even longer.

#### **What are the types of hair loss in women?**

There are three types of hair loss in women:

* Androgenetic alopecia/female pattern alopecia/female pattern hair loss (FPHL)/baldness: This is the most common type. Hair thins over the top of your head and on the sides.
* Anagen effluvium: Medications cause this type because they compromise a growing hair follicle. An example is chemotherapy.
* Telogen effluvium: An increased number of hair follicles reaching the telogen phase causes this type. This is the stage where hair falls out.

#### **How common is hair loss in women?**

Many people think that hair loss only affects males. But studies show that more than 50% of females will experience noticeable hair loss. The most significant cause of hair loss in women is female-pattern hair loss (FPHL). This affects about 30 million people in the United States.

## **Symptoms**

### **What are the signs of hair loss in women?**

The signs of hair loss in women may include:

* Seeing more hair fall out daily.
* Having noticeable patches of thinner or missing hair, including a part on the top of your head that gets wider.
* Seeing scalp skin through your hair.
* Tying up smaller ponytails.
* Feeling hair break off.

### **What causes hair loss in women?**

There are several possible causes of hair loss in women, including:

* Damaged hair follicles.
* Changes to your eating habits (rapid weight loss).
* Stress.
* Chemical hair treatments.
* Treatments like chemotherapy or radiation therapy.
* An underlying health condition like an abnormal thyroid, anemia, vitamin deficiency, etc.
* Hormonal changes (pregnancy, menopause).
* Certain medications and supplements (blood pressure medicines, gout medicines and high doses of vitamin A).
* Genetic predisposition (it runs in your biological family history).

#### **What is the relationship between hair loss in women and menopause?**

Your body experiences changes during menopause. This can affect your hair and cause:

* Hair growing where it didn’t before.
* The hair you have thinning out.

These changes happen due to varying levels of hormones during menopause. In addition, your hair follicles shrink. This makes your hair grow finer (thinner).

#### **What are the risk factors for hair loss in women?**

Hair loss in women can affect any female at any age. However, it’s usually more common if it runs in your biological family history and/or after:

* Age 40 years old.
* Pregnancy.
* Chemotherapy or radiation treatment.
* Menopause.

## **Diagnosis and Tests**

### **How will a healthcare provider diagnose hair loss in women?**

A healthcare provider will do a thorough examination and take a detailed history to understand changes in your hair growth. Your provider will also ask about what medications or supplements you currently take. Tests may follow the exam.

#### **What tests diagnose hair loss in women?**

The tests to diagnose hair loss in women may include:

* Gently pull on your hair to see how many hairs come out.
* Scalp examination under a microscope.
* Blood tests. These check for vitamin and mineral levels (like vitamin D, vitamin B, zinc and iron) and hormone levels (like thyroid).
* Scalp biopsy to remove and examine a very small piece of scalp skin.

#### **What questions might your healthcare provider ask to diagnose hair loss?**

To diagnose hair loss, your healthcare provider might ask questions about your hair habits, like:

* What kinds of hair products do you use?
* What kinds of hairstyles do you wear?
* Do you have a habit of pulling your hair out (trichotillomania)?

They might ask questions about your history, including:

* Has anyone in your immediate family experienced hair loss?
* Is there anything stressful going on in your life?
* What medications and supplements do you take every day?
* Has hair loss ever happened to you before?
* What foods are in your diet?

Also, they might ask questions about your observations, like:

* How long have you been losing hair?
* Have you been shedding more?
* Have you noticed hair loss in places other than your scalp, like your eyebrows, leg and arm hair?
* Does anything worsen your hair loss?
* Does anything improve your hair loss?
* Have you noticed hair loss occasionally or has it been going on continuously?
* Have you noticed if your hair growth has changed?
* Has your hair been breaking more often?

## **Management and Treatment**

### **How is hair loss in women treated?**

Treatment for hair loss depends on the cause. It may include:

* Reducing your stress, like talking with a mental health professional.
* Not using hair products (like chemical treatments) that damage your hair.
* Taking vitamins or supplements for a vitamin deficiency.
* Changing your hairstyling routine to avoid damaging your hair follicles.
* Taking medications.
* Managing any underlying health conditions.

In addition, a healthcare provider might recommend forms of light therapy like using the HairMax Lasercomb®. This low-light laser is approved by the U.S. FDA to treat FPHL. Another FDA-approved laser product is the Theradome LH80 PRO® helmet and low-light laser helmets and caps.

If you have hair loss due to stress or hormone changes like pregnancy, you may not need treatment. The hair loss will stop after a period of time.

Other forms of hair loss treatment may include:

* Microneedling of the scalp with and without the application of minoxidil.
* Injections of protein-rich plasma (PRP) to encourage hair growth.
* Hair transplant surgery.

It’s important to talk to your healthcare provider before starting any form of treatment for hair loss. Some types of treatment aren’t safe to use if you’re pregnant, planning on becoming pregnant or going through menopause.

#### **What medicines treat hair loss in women?**

A healthcare provider might recommend using minoxidil (Rogaine®). This is approved for treating FPHL. You can purchase the 2% or 5% solution over the counter (OTC). However, you have to follow directions exactly and use the product indefinitely. Don’t use this product if you’re pregnant, if you plan to get pregnant or if you’re breastfeeding.

Other medications that treat hair loss in women may include:

* Spironolactone and other anti-androgens.
* Finasteride and other alpha-reductase enzyme inhibitors.
* Estrogens.
* Prostaglandin analogs.
* Steroids.

#### **Are there side effects of minoxidil?**

Minoxidil may irritate your scalp and cause dryness, scaling, itching and/or redness. See your dermatologist if this happens.

With minoxidil, you might also see hair growing in places other than your scalp (cheeks and forehead, for example). Wash your face after you apply minoxidil and make sure you avoid other areas when you apply it.

#### **Who treats hair loss in women?**

A dermatologist usually treats hair loss in women.

## **Prevention**

### **Can hair loss in women be prevented?**

You can’t prevent all cases of hair loss in women. You can prevent hair loss caused by chemical hair treatments by not using them. You might be able to prevent some hair loss by eating nutritious foods that provide necessary nutrients (like vitamins, minerals and protein) or adding vitamins to your daily routine.

## **Outlook / Prognosis**

### **What is the prognosis for hair loss in women?**

The type and severity of hair loss you experience may determine the outcome (prognosis). Some types of hair loss are permanent, especially if you have damage to your hair follicles. But not all cases are. For example, anagen and telogen shedding may stop with time. Managing any underlying health conditions improves hair loss. And early treatment of alopecia may reduce the speed of thinning and promote regrowth. A healthcare provider can tell you more about what to expect in your situation.

## **Living With**

### **What are some tips for dealing with hair loss?**

There are some things you can do to manage your hair loss and feel more comfortable, including:

* Changing your hair color. Adding dye can increase strand volume, making your hair seem fuller.
* Massaging your head. When you wash your hair, use your fingers to massage your head to stimulate blood flow to your scalp and hair follicles.
* Changing your hairstyle. Cutting your hair shorter or changing your look with layers or new styling techniques can boost your confidence and potentially hide any hair loss.
* Using different types of shampoo and hair products. Look for a shampoo that adds volume without using sulfate detergents.

### **When should I see a healthcare provider?**

See a dermatologist as soon as possible when you notice hair loss. The sooner you get treatment, the more effective it’ll be.

## **Key Hair Loss Statistics**

* Hair loss is diagnosed if you lose more than 100 hairs per day. There is a fine difference between hair loss and hair shedding. It is considered acceptable to shed 50 to 100 hair pieces a day.
* Around 85% of men and 33% of women will at some time face hair loss. This problem is more common than you might think. Male pattern baldness or androgenetic alopecia makes up around 95% of male hair loss cases.
* More than 65% of American men will have hair loss of various degrees by age thirty-five. By the age of fifty, around 85% of men will have signs of thinning hair.
* Male pattern baldness may affect men of different ages. Around 25% of men having hair loss start the process before they are twenty-one.
* Genetics is the most significant cause of hair loss: Male pattern baldness affects around 95% of all men who suffer from hair loss. Other causes can be diet, stress, lifestyle, and illness.
* Research shows that, in the UK, Alopecia Areata affects two people out of every 1,000. Alopecia Areata is considered by some researchers an autoimmune condition that causes bald patches affecting any body part, scalp, chest, armpits, eyelashes, eyebrows, etc.
* The top three nations with the highest rates of male hair loss are all in the West: Spain (44.50% of the male population affected), Italy (44.37%), and France (44.25%).
* 40% of women suffering from alopecia have had problems in their marriage due to their hair loss. Around 63% claim to experience problems related to their career.
* More than half of women may experience hair loss in the postmenopausal period. A new study indicates that more than 50% of women experience hair thinning after they turn 50. The intensity of hair loss changes depending on various factors.
* Women leading stressful lives are 11 times more likely to suffer from hair loss. While it is unclear how exactly stress can prevent hair follicles from growing, researchers have noticed such a connection. Different studies are conducted to find out how stress can affect tissue regeneration.

DIFFERENTIAL DIAGNOSIS

Various subtypes of alopecia are confused with each other and require detailed examination through dermoscopy and biopsy. Some of the differentials include:

* **Androgenetic alopecia:** Alopecia areata, telogen effluvium, traction alopecia, trichotillomania, FFA, LPP, and central centrifugal cicatricial alopecia
* **Alopecia areata:** Tinea capitis, trichotillomania, and temporal triangular alopecia for patchy alopecia areata and female-pattern hair loss, telogen effluvium, and drug-induced alopecia for diffuse forms of alopecia areata
* **Telogen effluvium:** Androgenetic alopecia and diffuse forms of alopecia areata
* **FFA and LPP:** Chronic cutaneous lupus erythematosus, central centrifugal cicatricial alopecia, and folliculitis decalvan

REFERENCES

[Hair Loss in Women: Causes, Treatment & Prevention](https://my.clevelandclinic.org/health/diseases/16921-hair-loss-in-women)

[Hair Loss Statistics 2025 (Types, Treatments...)](https://medihair.com/en/hair-loss-statistics/)

[Alopecia - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK538178/#article-17384.s4)

**ANDROGENETIC ALOPECIA**

Androgenetic alopecia is a progressive type of hair loss, meaning the loss happens slowly over time. Although you may hear this type of alopecia referred to as **androgenic alopecia**, that’s a common misspelling. The correct term is androgenetic alopecia. This condition affects as many as half of women and men, so if you’re struggling with seeing more of your scalp in the mirror, there are millions of people who know exactly what you’re going through.

### **What is female pattern baldness?**

Female pattern baldness is a type of hair loss that affects women. It causes you to lose hair on the skin covering your head (scalp). Your hair doesn’t grow back without treatment.

You may hear your healthcare provider refer to female pattern baldness as female pattern hair loss or androgenic alopecia. The medical term for male pattern baldness is also androgenic alopecia. Male pattern baldness is similar to female pattern baldness but it affects men.

#### **What are the stages of female pattern baldness?**

There are five stages of female pattern baldness according to the Sinclair scale:

* Stage 1: Little or no hair loss.
* Stage 2: A slight gap appears in your center hair part.
* Stage 3: A wider gap is present in your center hair part, and there’s hair loss on either side of your part line.
* Stage 4: Bald spots appear toward the front of your hairline.
* Stage 5: Advanced hair loss.

#### **Can you reverse female pattern baldness?**

If you’ve lost hair from female pattern baldness, it won’t grow back without treatment. Proper treatment can stop hair loss and help regrow some hair.

#### **How common is female pattern baldness?**

Female pattern baldness is common. It’s the most significant cause of hair loss in women. Approximately 30 million women in the United States have female pattern baldness.

#### **How does female pattern baldness affect my body?**

Female pattern baldness causes the small, tube-like structures in your scalp that grow your hair (hair follicles) to shrink gradually. As your hair follicles shrink, your individual hairs get thinner and shorter. Over time, those hairs stop growing.

Female pattern baldness doesn’t affect your physical health. But it can affect you psychosocially (how society and social groups affect your thoughts and emotions). It can also affect you psychologically (how you think about yourself and your behavior). You may experience emotional stress, anxiety and depression.

## 

## **Symptoms and Causes**

### **What are the symptoms of female pattern baldness?**

Symptoms of female pattern baldness include:

* Hair thinning or hair loss around your center part.
* A widening of your center part and hair thinning or hair loss on either side of your part.
* Hair thinning or hair loss throughout the top of your head.

Hair loss usually starts to appear near your center part. In the middle and later stages of female pattern baldness, you’ll lose hair on either side of your part and toward the front of your scalp.

Female pattern baldness doesn’t hurt. Many start to notice the early stages of female pattern baldness after menopause.

#### **Will I notice female pattern baldness if I don’t have a center part?**

Female pattern baldness begins in the top-middle portion of your head. This falls around your center part, which is a natural line between the right and left sides of your hair. If you often style your hair to show a side part, you may not notice female pattern baldness immediately, but you’ll still see changes to the texture of your hair that indicate female pattern baldness near the center of your head.

#### **Does female pattern baldness worsen?**

Without treatment, female pattern baldness will get worse.

### **What causes female pattern baldness?**

Causes and contributing factors of female pattern baldness include:

* Age: The chances of getting female pattern baldness increase with age. Female pattern baldness affects about one-third of all women at some point during their lives. After menopause, about two-thirds of all women have thinning hair or total hair loss.
* Hormones: The hormone dihydrotestosterone (DHT) is a type of androgen. Androgens are a group of sex hormones that help people enter puberty and mature physically. Physical developments include hair growth on their face, scalp, chest, underarms and genitals. After menopause, your hormone levels drop, which may affect your DHT levels. Medical experts and researchers think that there might be a link between DHT and your hair follicles shrinking.
* Genetics: You’re more likely to have female pattern baldness if your first-degree relatives have hair loss.

#### **What are the risk factors for female pattern baldness?**

Female pattern baldness can affect all women. It can also happen at any age. Hair loss can affect you in your 20s and 30s. However, you’re more likely to have female pattern baldness if you have a family history of hair loss or after menopause.

## 

## **Diagnosis and Tests**

### **How is female pattern baldness diagnosed?**

Female pattern baldness is easy to recognize, so you don’t necessarily need a healthcare provider to diagnose it. However, a healthcare provider can confirm it during a physical exam of your scalp. They’ll ask you about your medical history, including when you started noticing hair loss and whether you have a family history of hair loss or baldness. Your provider will note the width of your center part and any areas of your scalp showing signs of thinning or balding.

#### **What tests diagnose female pattern baldness?**

Your healthcare provider may use a special tool called a densitometer to examine your scalp. A densitometer measures the thickness of your hair follicles.

If your healthcare provider suspects your hair loss isn’t related to female pattern baldness, they may:

* Examine your scalp for signs of infection.
* Take a hair sample and send it to a lab for analysis.
* Take a scalp biopsy to check for skin disease.
* Conduct blood tests.

## **Management and Treatment**

### **How is female pattern baldness treated?**

There are many hair loss treatments for female pattern baldness. Treatments may include:

* Medications: Over-the-counter medications you apply to your scalp, like minoxidil (Rogaine®), are usually the first course of treatment for female pattern baldness. A prescription oral medication, such as finasteride (Propecia®), can also treat female pattern baldness. Your healthcare provider may suggest using finasteride along with ketoconazole 2% shampoo.
* Hair transplant: A healthcare provider takes skin grafts from areas of your body that contain healthy hair follicles and moves them to bald or thinning areas of your scalp.
* Platelet-rich plasma: A healthcare provider removes blood from your body, processes it and then injects it into your scalp to stimulate hair growth.
* Red light therapy: Your healthcare provider treats your scalp with a low-wavelength red light to improve hair growth.
* Styling techniques: You may hide your female pattern baldness with certain hairstyles, wigs or hair weaves.

Treatment that works for one person may not work for another. Your healthcare provider will help you find a treatment option that’s right for you.

## **Prevention**

### **Can female pattern baldness be prevented?**

There’s no known way to prevent female pattern baldness. But there are ways to help keep your hair healthy that may promote hair growth, including:

* Eat extra protein, especially if you’re vegetarian or vegan. You need 40 to 60 grams of protein a day. The Mediterranean diet includes fruits, vegetables and protein that may help minimize hair loss.
* Take vitamins. Certain vitamins and minerals, including vitamins A, B, C, D, E, zinc and iron, help maintain healthy hair, skin and muscle tissue. But check with your healthcare provider first before starting any new supplements.
* Find ways to cope with stress. Stress may trigger female pattern baldness by increasing activity in your androgens.

## **Outlook / Prognosis**

### **What can I expect if I have female pattern baldness?**

Female pattern baldness is a treatable condition. Without proper treatment, female pattern baldness is permanent. Many people with female pattern baldness are comfortable with how they look and don’t seek treatment. However, your healthcare provider can help slow or replace your hair loss.

If female pattern baldness affects your emotional well-being, your healthcare provider may recommend you meet with a mental health professional.

## **Living With**

### **When should I see a healthcare provider?**

Many people choose not to see a healthcare provider if they have female pattern baldness. But if you’d like to maintain your hair, you should call a healthcare provider as soon as you notice hair loss on your scalp.

### **What questions should I ask my healthcare provider?**

* How can you tell that I have female pattern baldness?
* If I don’t have female pattern baldness, what’s causing my hair to fall out?
* What medications or treatments do you recommend?
* What’s the complete list of side effects of the medications and treatments?
* What else can I do to prevent further hair loss?
* Should I see a dermatologist or plastic surgeon?

**EPIDEMIOLOGY**

Caucasian individuals are most notably more affected, followed by Asians and African Americans, and subsequently by Native American and Inuit populations. The incidence aligns closely with age in Caucasian males, as approximately 50% are affected by 50 and approximately 80% by age 70. The disorder is quite common in females, with its incidence showing a notable rise after menopause.

**DIFFERENTIAL DIAGNOSIS**

Differential diagnosis of androgenetic alopecia encompass other hair loss disorders such as:

* Alopecia areata
* Anagen effluvium
* Syphilis
* Systemic diseases
* Telogen effluvium

REFERENCES

[Androgenetic Alopecia - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK430924/)

[Female Pattern Baldness: Symptoms, Stages, Causes & Treatment](https://my.clevelandclinic.org/health/diseases/24943-female-pattern-baldness)

## 

## 

## **Frontal Fibrosing Alopecia**

## Synonym(s)

Alopecia frontal fibrosis; FAPD; frontal fibrosing alopecia (e); Kossard's disease; Kossard syndrome; Postmenopausal frontal fibrosing alopecia; PPFA

## **Definition**

"Postmenopausal", a circumscribed, band-shaped, symmetrical, fibrosing form of scarring **alopecia** (irreversible) in the frontotemporal hairline in women with lymphoid-histiocytic infiltrates around the hair follicles. Often associated with rarefaction of the eyebrows. Men suffer less frequently from this clinical picture. This form of chronic creeping alopecia is often accompanied by keratosis pilaris, which is clinically conspicuous in adolescence and early adulthood.

The Frontal Fibrosing Alopecia - FFA Severity Index is used for the standardized assessment of the severity of the clinical picture. The score includes spread, involvement of body and facial hair, skin involvement, mucous membrane and nail involvement.

If >/= 4 points with typical criteria for frontal fibrosing alopecia, the diagnosis must be made. The frontal recession of the frontal hairline with loss of hair follicle ostia counts for 2 points, the positive biopsy in one of the affected areas: frontal or temporal scalp area or eyebrows also counts for 2 points, 1 point each for the loss of at least 50 % of the eyebrows and 1 point for follicular erythema on the frontal scalp, also 1 point for perifollicular scalp hyperkeratosis or dandruff on the frontal scalp.

## 

## **What Causes Frontal Fibrosing Alopecia?**

Hormones, genetics and autoimmune diseases are all mentioned as causes of Frontal Fibrosing Alopecia (FFA), however, the **cause is not yet fully understood**. In most cases 1 of these factors is diagnosed as the cause, however, a combination of the 3 is also possible for this type of **alopecia**. Studies have suggested that mainly post-menopausal women get FFA which has been the main indicator for it being hormonal based, although incidence is said to be increasing globally in all demographics. The lack of oestrogen or a hormonal shift has therefore been thought to be a cause of Frontal Fibrosing Alopecia. However, it’s frequently reported in both **men and women with autoimmune diseases**. Autoimmune diseases include rheumatoid arthritis, thyroid disease and lupus.

### Other causes of FFA

Furthermore, clinical studies blame an **inflammatory autoimmune reaction** for frontal and temporal hair loss. Additionally, there is substantial evidence that FFA existed way before Kossard’s discovery, since **artistic portraits from the 15th and 16th centuries** often show a fashionable receded frontal hairline. Although there are suggestions that this is from Traction Alopecia rather than FFA. In recent studies, leave on cosmetics such as sunscreen or makeup have been seen to have a potential link to the cause of FFA. This is because it can be seen to be in line with the rise in cases as these products have become more popularised in the last few decades.

### How is FFA related to Lichen Planopilaris (LPP)?

Frontal Fibrosing Alopecia is a form of Lichen Planopilaris. Other names for it include follicular lichen planus, Axel Munthe’s Syndrome and Kossard’s disease. According to the Genetic and Rare Diseases Information Center, there are 3 types of LPP; the classic Lichen Planopilaris, FFA, and Lassueur Graham-Little Piccardi Syndrome. The cause of LPP is unknown, however, it’s thought to be caused by an autoimmune disease. Like with LLP, the sebaceous gland which is in the same area as the stem cell region of the follicle is the target of inflammation. The main signs and symptoms of this medical condition are **tiny red bumps around your hair follicles that can cause itchiness, pain and a burning sensation**, later resulting in a permanent loss of hair. Follicular Lichen Planopilaris causes scarring, which leads to **permanent hair loss along the frontal hairline**. Thus, a lichen planopilaris scalp infection leads to Frontal Fibrosing Alopecia.

## 

## **What Are the Symptoms?**

The signs and symptoms of Frontal Fibrosing Alopecia **last for a few years,** whereby the loss of hair is slowly progressive, unlike other types of **alopecia areata** that are sudden and cyclical. Symptoms only present in the frontal and temporal hairline, which **gradually turns bald with scarring alopecia**. While the pattern is typically uniform, it can include a zigzag appearance or continuous receding all around the head. After a few years, the condition spontaneously stabilises, however, hair growth cannot naturally return.

### The symptoms include:

* Hair loss in a band-like pattern along the forehead and temples
* Lone hairs within the affected area
* Occasionally it spreads to other parts of the body, including loss of eyebrows and eyelashes
* Scarred skin after total hair loss in this area

As the condition continues over time, hair can be lost in extremity areas on the arms and legs. While this is often seen as a sign of natural ageing, with Frontal Fibrosing Alopecia the hair loss can be patchy and totally lacking in hair. The nearby hair follicles will have lost their redness and scaling typical of the area where the hair loss has occurred.

### How do you know if you have frontal fibrosing alopecia?

Peri follicular inflammation occurs along the hairline, which affects the growth of your hair. As the number of hair follicles decreases, they are replaced with fibrosis. You will know if you have FFA if you notice that your frontal hairline has **signs of scarring**. The skin in the area affected by FFA will show a lack of sun damage on your forehead that is typically seen. This can help to show the extent of the hairline recession. The diagnosis of Frontal Fibrosing Alopecia is established through a **skin biopsy,** which analyses whether Lichen Planopilaris is present. Areas with remaining hairs are studied to find evidence of scalp inflammation. The area that has a lack of hair will be pale and shiny with the lack of visible follicle openings. In men, loss of hair in the beard area can be possible. Early signs of FFA can be itching and pain in the area with discoloured skin on the forehead and temples.

## 

## **How Do You Treat Frontal Fibrosing Alopecia?**

Due to the lack of research about FFA, scientists haven’t discovered a cure for this type of hair loss. Many women look towards using the best hairstyles for frontal fibrosing alopecia like an asymmetrical bob or long bangs in order to cover the condition. However, the **British Association of Dermatologists** have identified several treatment options, but their success is variable, and sometimes no treatment is effective. Early diagnosis is important in order to prevent long term damage to the hair follicles. Managing the condition can be complex, as treatment can be different for each person. As it is often slowly progressive, it can be resolved on its own after several years in some cases.

### Treatments include:

* Topical corticosteroids (steroid cream)
* Intralesional steroids (steroid injections)
* Immunotherapy
* Finasteride
* Topical Tacrolimus
* Antibiotics

A **SDHI hair transplant is sometimes considered** for treating Frontal Fibrosing Alopecia, however, it is only possible **if your condition has stabilised**. The SDHI method works well for restoring hair loss in the hairline region, as it offers precision while also restoring density to the affected regions of hair loss. Whether hair can be restored with surgery depends on the cause as well. Therefore, you will need to consult a doctor/hair loss expert for an assessment.

**Prognosis**

Alopecia can affect half of the scalp, often called crown alopecia. The progression of the disease is variable among patients, ranging from 0.2 to 2 cm per year without treatment or, on average, 0.9 mm per month. The final degree of alopecia before stabilization is difficult to predict.

## **Complications**

## Complications of frontal fibrosing alopecia extend beyond cosmetic concerns, encompassing psychosocial distress due to visible alopecia and the potential for irreversible scarring leading to permanent hair loss. In addition, frontal fibrosing alopecia is associated with ocular and facial involvement, including eyebrow loss, periorbital erythema, and cicatricial ectropion. Beard hair loss has recently been reported in patients. The diagnosis often necessitates a multidisciplinary approach involving dermatologists, endocrinologists, and, occasionally, ophthalmologists to manage the condition comprehensively and address the diverse manifestations.

## 

## Differential diagnosis

* Clinical:
  + **Graham-Little-Lasseur syndrome**: variant of **lichen planus follicularis** with follicular, lacy keratotic lesions on the trunk, the typical clinical and histologic signs of lichen planus, and scarring alopecia. Nail dystrophies are possible. No ulerythema; no keratosis pilaris.
  + **Lichen planus follicularis capillitii**: Minus variant of lichen planus follicularis. Otherwise see before!
  + **Alopecia marginalis**: Reversible, mechanically caused hair loss due to chronic traction, e.g. with tight hairstyle. Traction alopecia with corresponding history and clinic. No follicular inflammatory signs.
  + **Alopecia androgenetica**: Absence of any follicular inflammatory phenomena as characteristic of fibrosing alopecia and lichen planus follicularis.
  + **Alopecia areata**: The type and duration of the "continuously receding hairline" is completely atypical of alopecia areata.
  + **Chron. discoid lupus erythematosus**: The morphologic pattern of fibrosing alopecia is atypical for CDLE. Usually evidence of other active or scarred lesions.
* Histologic:
  + **Lichen planus follicularis capillitii**: lichenoid infiltrate pattern perifollicular, on the surface epithelium signs of interface dermatitis. This is completely absent in frontal fibrosing alopecia.
  + **Chronic discoid lupus erythematosus**: Interface dermatitis, immunohistological differentiation with deposits of immunoglobulins at the dermo-epidermal junctional zone.

**EPIDEMIOLOGY**

The incidence of frontal fibrosing alopecia is increasing in Europe, the United States, and Japan. Frontal fibrosing alopecia mainly affects women after menopause, typically around the age of 60. However, instances have been documented in premenopausal women, with the youngest reported case aged 21, and rarely in men. Men are mainly affected when genetic factors are involved. Among African women, frontal fibrosing alopecia is often associated with traction alopecia that worsens the progression of the disease. Japanese women have less severe forms compared to European women

REFERENCE

[Frontal Fibrosing Alopecia - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK519001/)

<https://elithair.co.uk/blog/frontal-fibrosing-alopecia/>

## 

# **Trichorrhexis nodosa**

Trichorrhexis nodosa is a common hair problem in which thickened or weak points (nodes) along the hair shaft cause your hair to break off easily.

The essential abnormality of trichorrhexis nodosa is the formation of nodes along the hair shaft through which breakage readily occurs. In 1852, Samuel Wilks of Guy’s Hospital first described the condition, although the term trichorrhexis nodosa was not proposed until 1876 by M. Kaposi. Trichorrhexis nodosa is ultimately a response to physical or chemical trauma. Trichorrhexis nodosa may be acquired in patients with normal hair through exposure to a sufficient level of trauma. Trichorrhexis nodosa may also be congenital, occurring in defective, abnormally fragile hair following trivial injury. It is the most common congenital defect of the hair shaft.

It may affect the hair of the scalp, pubic area, beard, and moustache and may be particularly noticeable in persons of Afro-Caribbean descent because of hairstyling techniques. Hair care practices and hairstyles often used among women of African descent may contribute to trichorrhexis nodosa and central centrifugal cicatricial alopecia. The more common acquired form results from excessive or repeated trauma caused by frequent application of hair-permanent liquid, hair dyes, frequent brushing, scalp massage, and lengthy and repeated ultraviolet exposure, a reflection of the amount of trauma inflicted on the hair shafts rather than by some inherent or structural defect.

## **Causes**

Trichorrhexis nodosa can be an inherited condition.

The condition may be triggered by things such as blow-drying, ironing the hair, over-brushing, perming, or excessive chemical use.

In some cases, trichorrhexis nodosa is caused by an underlying disorder, including very rare ones, such as:

* Thyroid gland not making enough thyroid hormone (hypothyroidism)
* Buildup of ammonia in the body (argininosuccinic aciduria)
* Iron deficiency
* Menkes syndrome (Menkes kinky hair syndrome)
* Ectodermal dysplasia (a group of conditions in which there is abnormal development of the skin, hair, nails, teeth, or sweat glands
* Trichothiodystrophy (inherited disorder that causes brittle hair, skin problems, and intellectual disability)
* Biotin deficiency (inherited disorder in which the body is not able to use biotin, a substance needed for hair growth)

## **Symptoms**

Your hair may break easily or it may appear like it is not growing.

In African Americans, looking at the scalp area using a microscope shows that the hair breaks off at the scalp area before it grows long.

In other people, the problem often appears at the end of a hair shaft in the form of split ends, thinning hair, and hair tips that look white.

## **Exams and Tests**

Your health care provider will examine your hair and scalp. Some of your hairs will be checked under a microscope or with a special magnifier usually used by skin specialists.

Blood tests may be ordered to check for anemia, thyroid disease, and other conditions.

## **Treatment**

If you have a disorder that is causing trichorrhexis nodosa, it will be treated if possible.

Your provider may recommend measures to reduce damage to your hair such as:

* Gentle brushing with a soft brush instead of aggressive brushing or ratting
* Avoiding harsh chemicals such as those used in straightening compounds and perms
* Not using a very hot hair dryer for long periods and not ironing the hair
* Using a gentle shampoo and a hair conditioner

## **Outlook (Prognosis)**

Improving grooming techniques and avoiding products that damage hair will help correct the problem.

This condition is not dangerous, but may affect a person's self-esteem.

## **When to Contact a Medical Professional**

Call your provider if symptoms do not improve with changes in grooming and other home-care measures.

## **Alternative Names**

Hair shaft fracture; Brittle hair; Fragile hair; Hair breakage

## Differential Diagnoses

* Alopecia Areata
* Anagen Effluvium
* Androgenetic Alopecia
* Dermatologic Manifestations of Menkes Kinky Hair Disease
* Monilethrix
* Piedra
* Seborrheic Dermatitis
* Trichomycosis Axillaris
* Trichomycosis Pubis
* Trichorrhexis Invaginata (Netherton Syndrome or Bamboo Hair)
* Trichotillomania

**Epidemiology**

Trichorrhexis nodosa is a rare disorder. A retrospective review of 129 hair-mount samples from 119 patients over a 10-year span found 25 cases of loose anagen hair syndrome, 6 cases of uncombable hair syndrome, and trichorrhexis nodosa in 13 patients.

Acquired proximal trichorrhexis nodosa is common in Blacks and appears to occur in individuals who are genetically predisposed. Some consider it an ethnic hair disorder. Acquired distal trichorrhexis nodosa primarily occurs in Asian or White persons. Acquired proximal trichorrhexis nodosa is more common in females than in males.

Congenital trichorrhexis nodosa may be present at birth, or it may appear within the first couple months of life. It can present in patients with the late form of argininosuccinic aciduria at age 2 years or older.

REFERENCES

[Trichorrhexis Nodosa Differential Diagnoses](https://emedicine.medscape.com/article/1073664-differential?form=fpf)

[Trichorrhexis nodosa: MedlinePlus Medical Encyclopedia](https://medlineplus.gov/ency/article/001449.htm)

### 

### **Anagen Effluvium**

Anagen effluvium is a type of hair loss characterized by the sudden and widespread shedding of actively growing hairs. This condition typically occurs as a result of exposure to damaging agents such as chemotherapy or radiation therapy. These therapies target rapidly dividing cells, including hair follicles. Anagen effluvium leads to the abrupt cessation of hair growth and rapid hair loss, often within weeks of the trigger. Unlike some other forms of hair loss, anagen effluvium can result in almost complete hair loss on the scalp. Fortunately, once the causative treatment is completed, hair follicles can recover, and hair regrowth usually begins within a few months.

### **Causes of Anagen Effluvium**

Anagen effluvium occurs when hair follicles are damaged during the anagen (growth) phase of the hair cycle. Unlike telogen effluvium, which results in hair loss during the resting phase, anagen effluvium causes more immediate and widespread hair loss. There are several known causes, most of which involve external factors that disrupt the normal hair growth process.

#### **Chemotherapy and Radiation Treatment**

One of the most common causes of anagen effluvium is chemotherapy. Many cancer treatments involve powerful drugs that target rapidly dividing cells in the body. Since hair follicles are among the fastest-growing cells, chemotherapy drugs can damage them, leading to hair loss. Radiation therapy, particularly when directed at the scalp, can cause anagen effluvium by targeting hair follicles in the radiation zone. Both chemotherapy and radiation cause rapid shedding of hair, often within a few weeks of starting treatment. While this hair loss is typically temporary, the hair may not always regrow to its original thickness. It can take several months after the treatment ends before regrowth starts.

#### **Medications**

Certain medications, including those used to treat autoimmune conditions and infections, can cause anagen effluvium as a side effect. Drugs like high-dose vitamin A, certain antifungals, and immunosuppressive medications can interfere with the normal functioning of hair follicles. This leads to accelerated shedding during the anagen phase. In some cases, medications used to treat conditions like lupus or psoriasis can directly affect hair follicles. They can cause the hair follicles to stop growing and enter a shedding phase prematurely. If medication is identified as the cause, your healthcare provider may adjust the dosage to help manage hair loss.

#### **Toxins and Poisoning**

For example, exposure to arsenic, heavy metals (such as mercury or lead), or pesticides can disrupt the hair growth cycle. Toxins may damage the hair follicles directly, leading to hair shedding that occurs within days or weeks of exposure. The severity of hair loss depends on the amount and duration of exposure. Once the body is cleared of toxins, hair can begin to regrow although this process can take time.

#### **Autoimmune Disorders**

Autoimmune diseases can lead to anagen effluvium. Particularly in conditions where the immune system mistakenly targets healthy cells, including those in hair follicles. For example, autoimmune diseases like lupus or rheumatoid arthritis can cause hair loss by disrupting the normal growth cycle of hair follicles. In some cases, the immune system may attack hair follicles directly, leading to hair shedding during the anagen phase. The extent of hair loss varies depending on the severity of the autoimmune response. Treatment often involves addressing the underlying autoimmune disorder through immunosuppressive medications or other targeted therapies.

#### **Severe Infections**

Severe infections, particularly those that involve high fever or systemic illness, can lead to anagen effluvium. Infections such as viral illnesses, bacterial infections, or fungal infections that affect the body’s overall health can stress the body to the point where hair follicles enter a premature shedding phase. The intense physiological stress caused by the infection triggers hair follicles to stop growing, resulting in rapid hair loss. Once the infection is treated and the body recovers, hair regrowth usually follows. It may take several months for hair to return to its normal density.

#### **Hormonal Imbalances**

Hormonal imbalances, such as those that occur during pregnancy, menopause, or due to thyroid dysfunction, can contribute to anagen effluvium. Hormones like estrogen, progesterone, and thyroid hormones play a crucial role in regulating the hair growth cycle. Significant hormonal shifts, such as those that occur during childbirth, after stopping birth control, or in the case of thyroid disease, can disrupt the normal hair growth process and cause rapid hair loss. In cases where an imbalance is detected, treatment to restore hormonal levels can help prevent or reverse the hair loss. This balance restoration will allow the hair to regrow once the underlying issue is addressed.

#### **Physical Trauma**

Severe physical trauma or injury to the body can lead to anagen effluvium due to the body’s stress response. For example, a significant injury like a car accident or a major surgery can cause the body to focus its energy on recovery. This leads to the interruption of hair follicle growth. The physical stress associated with trauma can cause a large number of hair follicles to prematurely enter the shedding phase. Although the hair loss is often temporary, it may take several months for the hair to return to its normal growth cycle after the trauma has healed.

#### Nutritional Deficiencies

Severe nutritional deficiencies, particularly in essential vitamins and minerals such as iron, zinc, and protein, can contribute to anagen effluvium. Malnutrition or an insufficient diet deprives the hair follicles of the nutrients needed to sustain normal hair growth. Conditions like anorexia, bulimia, or crash dieting that result in nutrient deficiencies can also trigger anagen effluvium. Once nutritional imbalances are corrected through proper diet or supplementation, hair regrowth can begin.

### **Symptoms of Anagen Effluvium**

Anagen effluvium is a condition characterized by rapid and widespread hair loss that occurs during the anagen (growth) phase of the hair cycle. Unlike other forms of hair loss, such as telogen effluvium, which causes diffuse shedding, anagen effluvium results in more severe and sudden shedding.

#### **Rapid and Widespread Hair Loss**

The most noticeable symptom of anagen effluvium is the sudden and rapid shedding of hair. Unlike the slower hair loss seen in other forms of alopecia, hair in the anagen phase falls out within days to weeks after the triggering event. The shedding is often extensive and affects large portions of the scalp. In many cases, large amounts of hair may fall out when brushing or washing hair. Excess hair may be found on clothing, pillowcases, or in the drain. This type of hair loss can be alarming, especially when it occurs quickly, and is typically more dramatic than other forms of hair loss.

#### **Thinning of the Entire Scalp**

As the hair falls out rapidly during the anagen phase, the scalp becomes noticeably thinner. Unlike other types of hair loss that may present as patchy or localized, anagen effluvium leads to a uniform thinning across the entire scalp. The hair becomes sparse, and the scalp may be more visible, especially in areas where the hair is finer or shorter. This thinning can occur very quickly, and individuals may notice a significant reduction in their hair volume. The overall appearance of the hair may look much less dense than usual, and the thinning can be distressing.

#### **Breakage of Hair Strands**

Along with the shedding of hair, another symptom of anagen effluvium is the breakage of hair strands. Unlike other conditions where hair falls out from the root, anagen effluvium often results in the hair becoming brittle and prone to breakage. This can occur because the hair follicles are prematurely pushed into the shedding phase, causing the hair to weaken and snap off easily. As a result, individuals may notice that their hair appears frayed or broken, especially at the ends. This breakage can add to the appearance of hair thinning and contribute to the distress caused by the condition.

#### **Hair Loss Within Weeks of Triggering Event**

One of the key characteristics of anagen effluvium is the rapid onset of hair loss. Hair shedding typically begins within days or weeks of the triggering event, such as chemotherapy, radiation, or exposure to toxins. This fast progression can be a sign that the hair is in the anagen phase when the damage occurs. People experiencing anagen effluvium will often notice dramatic shedding during the first month after the triggering event, which can be overwhelming and noticeable to others. The speed at which hair falls out distinguishes anagen effluvium from other forms of hair loss, which tend to develop more gradually.

#### **No Bald Patches (Diffuse Hair Loss)**

Unlike some other forms of hair loss, such as alopecia areata, which causes bald patches, anagen effluvium typically results in diffuse hair loss. There are no distinct bald spots; instead, the hair thins uniformly across the scalp. As the hair follicles are all affected at once, there is no pattern of isolated bald areas. The thinning is often so widespread that it can be difficult to notice a specific spot where the hair is thinning most. However, the overall reduction in hair volume can make the scalp more visible and may lead to an overall feeling of hair loss.

#### **Eyebrow, Eyelash, and Body Hair Loss**

While anagen effluvium primarily affects the scalp, it can also cause hair loss in other areas of the body, including the eyebrows, eyelashes, and body hair. This is particularly common with chemotherapy or radiation treatments that target the entire body. In some cases, the shedding of body hair may not be as noticeable as scalp hair loss, but it can still contribute to a feeling of total hair loss. Eyebrows and eyelashes are often affected, which can be distressing because they play an important role in facial appearance. Regrowth typically begins once the underlying cause is addressed, but regrowth in these areas may take longer to occur compared to the scalp.

### **Treatment Options for Anagen Effluvium**

Anagen effluvium is primarily a consequence of exposure to damaging agents which disrupt the active growth phase. The most effective way to address anagen effluvium is to discontinue or complete the causative treatment, allowing hair follicles to recover and restart the hair growth cycle. While there is no specific treatment to reverse the damage caused by anagen effluvium, several approaches can help manage the condition and support the regrowth of hair.

#### Discontinuation of Causative Medications or Treatment

The first and most important step in treating anagen effluvium is to address the underlying cause. For instance, if chemotherapy or radiation therapy is causing the hair loss, the healthcare provider may adjust the treatment plan or provide alternative therapies. In some cases, switching to a different medication or adjusting dosages may help reduce the severity of hair loss. For non-cancer treatments, such as certain medications or toxins, discontinuing or changing the triggering agent is the most effective way to stop the shedding. Once the causative factor is removed, hair loss typically slows down, and regrowth can begin, though the process can take several months.

#### Supportive Care and Scalp Protection

#### Since anagen effluvium causes rapid hair loss, it’s important to take measures to protect the scalp and hair follicles during the shedding phase. Gentle hair care practices are crucial, as harsh treatments can exacerbate hair breakage. Using a mild, sulfate-free shampoo and avoiding excessive heat styling, harsh chemical treatments, or tight hairstyles can help reduce further damage. Scalp protection is especially important for individuals undergoing chemotherapy or radiation therapy, as their skin may be more sensitive. Wearing hats or scarves can provide protection from sun exposure and prevent irritation from external elements. Though this doesn’t directly speed up regrowth, it helps minimize further stress on the scalp and hair.

#### Nutritional Support and Supplements

In cases where anagen effluvium is linked to nutritional deficiencies, ensuring the body receives the proper nutrients is crucial. Deficiencies in essential vitamins and minerals, such as iron, vitamin D, zinc, and biotin, can contribute to hair loss, so supplementing with these nutrients may support hair health and regrowth. Iron supplements are particularly helpful for individuals with iron-deficiency anemia, which is often associated with hair loss. Consulting with a healthcare provider or a nutritionist to check for deficiencies and develop a plan for supplementation is a vital step. A well-balanced diet rich in protein, healthy fats, and vitamins can also promote overall hair health and help the body recover from the stress caused by the condition.

#### Platelet-Rich Plasma (PRP) Therapy

Platelet-rich plasma (PRP) therapy involves extracting a small amount of the patient’s blood, concentrating the platelets, and injecting the platelet-rich plasma into the scalp. The growth factors in PRP are believed to stimulate hair follicles, promote healing, and accelerate the regrowth of hair. Although PRP therapy is more commonly used for androgenetic alopecia (pattern baldness), some dermatologists suggest it as a treatment for anagen effluvium, especially when hair regrowth is delayed. While more research is needed to determine its efficacy for anagen effluvium, PRP may help improve scalp health and encourage regrowth in cases where other methods have not been as effective.

#### Minoxidil

Minoxidil, an over-the-counter topical solution, is commonly used to treat hair loss and may be recommended for anagen effluvium to stimulate hair regrowth. It works by improving blood flow to the hair follicles and promoting hair growth. While it is primarily used for androgenetic alopecia, it may be beneficial for individuals with anagen effluvium who are experiencing slow regrowth after the triggering event has been resolved. Minoxidil is generally safe for most people, but it can cause scalp irritation in some individuals. It’s important to note that it does not work immediately, and results can take several months to appear.

#### Corticosteroids (In Severe Cases)

For individuals experiencing severe hair loss due to an underlying autoimmune condition or if inflammation is contributing to the shedding, corticosteroids may be used. These medications help to suppress the immune response and reduce inflammation, potentially allowing hair follicles to recover and resume growth. Corticosteroids can be administered orally, topically, or through injections directly into the scalp. While corticosteroids may be effective for some individuals, they are generally reserved for severe cases and should be used under the supervision of a healthcare provider due to potential side effects from prolonged use.

#### Patience and Time

In many cases, the best treatment for anagen effluvium is simply time. Once the underlying cause is addressed and the hair follicles are no longer being disrupted, hair regrowth often resumes naturally. However, this process can take time—several months for the hair to fully regrow. During this period, individuals may feel frustrated with the slow pace of regrowth, but it’s important to understand that patience is essential. Regular follow-up with a healthcare provider can help track progress, manage any lingering concerns, and provide guidance on additional treatment options if necessary.

prognosis

Chemotherapy-induced alopecia is typically reversible due to its selective targeting of proliferating cells in the bulb, sparing the quiescent stem cells in the bulge responsible for restarting follicle growth. Once treatment is discontinued, the hair follicle promptly returns to its regular cycle and exhibits visible regrowth within 3 to 6 months. Approximately 65% of patients encounter graying, curling, or straightening effects in their newly regrown hair, which is plausibly attributed to chemotherapy's differential effects on hair follicle melanocytes and inner root sheath epithelia. Importantly, these adverse effects often decrease over time.

The occurrence of permanent alopecia following standard-dose chemotherapy for breast cancer is infrequent. However, there is now compelling evidence indicating the presence of permanent or prolonged alopecia following standard-dose chemotherapy, particularly with docetaxel. The dosage per infusion and the duration of exposure closely correlate with this phenomenon. Furthermore, a study linked a genetic predisposition, specifically in the ABCB1 gene, to the development of permanent docetaxel-related alopecia in individuals with breast cancer. A single case series has indicated instances of delayed recovery associated with paclitaxel, although such occurrences are infrequent given the current dose regimens.

complication

Alopecia is commonly recognized as a substantial hindrance to the well-being of cancer patients undergoing chemotherapy. The sudden loss of hair due to chemotherapy can severely impact mental health and quality of life, particularly in young women. Some patients may undergo considerable emotional distress, leading them to choose suboptimal treatment or refuse or delay treatment that could otherwise be beneficial.

In most cases, hair regrowth follows a typical pattern, but in certain instances, individuals with straight hair may develop curly hair upon regrowth. The color of the hair may also undergo alterations. Although chemotherapy-induced anagen effluvium typically leads to complete hair regrowth and is reversible, specific chemotherapy treatments can cause persistent alopecia, depending on the administered dosage. This alopecia has histological characteristics comparable to nonscarring alopecia, similar to androgenetic alopecia

**Epidemiology**

Anagen effluvium has no gender or regional preference; it is equally prevalent among men and women worldwide. Furthermore, there is no observed association between hair type, ethnicity, and race and changes in the severity of alopecia or the rate and pattern of hair regrowth.

**DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of anagen effluvium includes other nonscarring alopecias, such as telogen effluvium, trichotillomania, and androgenetic alopecia. These conditions can be differentiated through a detailed examination of the patient's history, the hair pull test, and trichoscopy. Hair loss may be a prominent sign of an underlying disorder. A thorough review of systems should be completed to exclude other causes of hair loss, such as nutritional deficiencies, metabolic or endocrine disorders, and infections.

[Anagen Effluvium - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK482293/)

### 

### 

### **Telogen effluvium**

Telogen effluvium is a type of temporary hair loss caused by a stressor or change to your body. Your hair has three stages of growth and loss (shedding):

* Anagen (growth): This is the most active stage of your hair growth cycle. The cells in the lower part of a hair follicle rapidly divide. Once new hair has formed, it pushes the hair out of your follicle and elongates your hair shafts. A hair follicle can grow hair in the anagen stage for up to four years. Around 80% to 90% of your hair follicles are in the anagen stage at any given time.
* Catagen (resting): This is a transitional stage of your hair growth cycle. The lower portion of your hair follicle regresses and the hair stops growing. Around 5% of all hairs are in the catagen stage at any given time.
* Telogen (shedding): This is the resting stage of your hair growth cycle. The hair follicle is inactive, and there’s a white bulb of keratin at the root (club hair). The bulb of keratin keeps the hair in the follicle until it sheds, which then starts the hair growth cycle over again. Around 5% of all hairs are in the telogen stage at any given time.

Telogen effluvium affects your hair when it’s in the telogen stage. After a stressor or change to your body, up to 70% of your hair in the anagen stage prematurely enters the telogen phase, which causes hair loss.

### **What’s the difference between acute telogen effluvium and chronic telogen effluvium?**

Acute telogen effluvium lasts fewer than six months, and your hair loss tends to happen two to three months after a stressor or change to your body. In 95% of cases, acute telogen effluvium goes away (resolves).

Chronic telogen effluvium lasts longer than six months. It affects your entire scalp and may not have a clear cause. You may lose your hair in handfuls during the early stages of chronic telogen effluvium, but it won’t cause total baldness.

### **Who does telogen effluvium affect?**

Anyone can develop telogen effluvium. However, your chances of developing chronic telogen effluvium without a detectable cause increase if you’re a woman and are between 30 and 60 years of age.

### **How common is telogen effluvium?**

Telogen effluvium is one of the most common causes of rapid hair loss. It’s also one of the most common causes of hair loss in women.

### **How does telogen effluvium affect my body?**

Telogen effluvium causes hairs to enter the resting stage from the growing stage prematurely. Most people who are healthy lose up to 100 strands of hair per day. If you have telogen effluvium, you may lose up to 300 strands of hair per day.

Telogen effluvium may affect the hair all over your scalp, but it most commonly appears on the top of your head rather than the back or sides of your head. It usually won’t affect your hairline or cause total baldness, but severe cases of telogen effluvium may affect other areas of your body, including your eyebrows and body hair. Occasionally, hair-thinning can mimic male- or female-pattern hair loss. Telogen effluvium has heavy shedding and rapid loss. Male- and female-pattern hair loss has slow thinning.

Telogen effluvium won’t affect your physical health, but it can affect you psychosocially (how society and social groups affect your thoughts and emotions) and psychologically (how you think about yourself and your behavior). You may experience emotional stress, anxiety and depression.

## **Symptoms**

Symptoms of telogen effluvium include:

* Increased hair loss, which you may notice in your hairbrush, in your shower drain or on your pillow.
* Thinning hair on your scalp.
* Dry hairs that fall out easily.

If you have telogen effluvium, your scalp should look healthy. You shouldn’t have any other symptoms, such as a rash, itching, burning, pain or flaking.

### **What triggers telogen effluvium?**

The following factors may trigger telogen effluvium:

* High fever.
* Childbirth.
* Severe infections.
* Psychological stress.
* Major surgery.
* Hyperthyroidism.
* Hypothyroidism.
* Discontinuing use of birth control pills.
* Fad diets that don’t have enough protein.
* Some medications, including retinoids, beta-blockers, calcium channel blockers, depression medicines and nonsteroidal anti-inflammatory drugs (NSAIDs).

### **Is telogen effluvium contagious?**

No, telogen effluvium isn’t contagious.

## **Diagnosis and Tests**

Telogen effluvium is easy to recognize. A healthcare provider can confirm it during a physical examination of your scalp. They may conduct a “pull test,” in which they gently pull a small clump of 40 to 60 hairs between their fingers. Under typical conditions, they may only pull two to three hairs from your scalp. If you have telogen effluvium, they may pull at least four to six hairs from your scalp with white bulbs at the roots.

Your healthcare provider will also ask you about your diet and recent medical history. They may identify a dietary cause or a stress or illness that occurred about three months before you noticed hair loss. In many cases, someone with telogen effluvium has fully recovered from a stressor or illness and doesn’t see a connection between it and their hair loss.

### **What tests will be done to diagnose telogen effluvium?**

In most cases, your healthcare provider can diagnose telogen effluvium without any testing. They may recommend further testing, including blood tests or a scalp biopsy, if they suspect a condition or illness has caused telogen effluvium.

## **Management and Treatment**

Because one of the main causes of telogen effluvium is a stressor on your body, it’s important to identify the cause. Once you address the cause, most cases of telogen effluvium will resolve without treatment within six to eight months.

### **What medications/treatments are used to treat telogen effluvium?**

Telogen effluvium should resolve on its own, so you may not need treatment. However, common telogen effluvium treatments may include:

* Medications: Over-the-counter (OTC) medications you apply to your scalp, such as minoxidil (Rogaine®), promote hair growth. Some side effects of minoxidil may include headache, scalp irritation and unusual hair growth. You shouldn’t use minoxidil if you’re pregnant or breastfeeding.
* Multivitamins or supplements: Multivitamins that contain iron or an iron supplement help promote hair growth. Biotin supplements also help promote strong, healthy hair.
* Styling techniques: You may be able to obscure or hide your hair loss with certain hairstyles, wigs or hair weaves.

### **How long does telogen effluvium last?**

Telogen effluvium usually lasts between three and six months.

### **Will my hair grow back after telogen effluvium?**

Yes, your hair will grow back after telogen effluvium. After the three- to six-month shedding period, you’ll notice new hair growth in your affected areas.

## **Prevention**

There are ways to help keep your hair healthy and promote hair growth, including:

* Eat extra protein, especially if you’re vegetarian or vegan. You need 40 to 60 grams of protein a day. The Mediterranean diet includes fruits, vegetables and protein that may help minimize hair loss.
* Take vitamins. Certain vitamins and minerals, including vitamins A, B, C, D, E, zinc, biotin and iron, help maintain healthy hair, skin and muscle tissue. Ask your healthcare provider before adding any new supplements to your diet.
* Find ways to cope with stress. Stress is one of the leading causes of telogen effluvium.
* Get enough sleep. Most adults need between seven and nine hours of sleep per night. The benefits of a good night’s sleep include decreased stress.
* Avoid extreme or restrictive diets. Rapid weight loss can trigger telogen effluvium. Restrictive diets can lead to nutritional deficiencies that can cause telogen effluvium. If you must lose weight, it’s a good idea to get regular exercise and follow a Mediterranean-style diet that focuses on fresh fruits and vegetables, whole grains, lean protein and healthy fats.

## **Outlook / Prognosis**

### **What can I expect if I have telogen effluvium?**

Telogen effluvium can be stressful, and you may fear that you’ll lose all of your hair. However, if you have telogen effluvium, the outlook is good. It usually goes away three to six months after you start noticing your hair loss. Your healthcare provider can also help you take steps to promote new hair growth.

## **Additional Common Questions**

### **What’s the difference between telogen effluvium and androgenic alopecia?**

Telogen effluvium is a type of hair loss that involves rapid shedding of hair over a short period. It typically happens a few months after your body goes through something physically or emotionally stressful. It can also result from sudden hormonal changes. Hair loss due to telogen effluvium is usually temporary, and your hair often grows back without treatment once you no longer have that stress.

Androgenic alopecia (male pattern baldness and female pattern baldness) is a type of hair loss that’s more gradual than telogen effluvium. It’s not known exactly what causes androgenic alopecia. Without medications or treatment, hair loss due to androgenic alopecia is permanent.

**DIFFERENTIAL DIAGNOSIS**

The differential diagnoses of telogen effluvium include alopecia areata, anagen effluvium, androgenetic alopecia, scarring alopecia, syphilis, and trichotillomania.

**EPIDEMIOLOGY**

Telogen effluvium is a condition that can affect individuals of any age, gender, or racial background. The exact prevalence of telogen effluvium is unknown, but it is considered quite common. Many adults experience an episode of telogen effluvium at some point in their lifetime. Although telogen effluvium can manifest in both men and women, women tend to be more susceptible because of postpartum hormonal changes. In addition, women are more disturbed by hair shedding compared to men and are more likely to seek medical attention

REFERENCES

<https://my.clevelandclinic.org/health/diseases/24486-telogen-effluvium>

[Telogen Effluvium - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/sites/books/NBK430848/)

### 

### 

### 

### 

### **scarring alopecia**

**DEFINITION**

Scarring alopecia, also called cicatricial alopecia, is a type of hair loss caused by the destruction of your hair follicles (shafts on the surface of your skin that hair grows through). It’s usually the result of infections, chemicals, burns or autoimmune disorders. Hair loss due to scarring alopecia can be permanent because your hair can’t grow back without healthy hair follicles.

Scarring alopecia can affect any part of your body that has hair, but commonly affects the scalp. Visible hair loss due to scarring alopecia can also have emotional and psychological effects.

### **DESCRIPTION**

Scarring alopecia usually appears as a bald patch where there’s typically hair. There might be one bald area or several. The skin where hair used to be tends to look smooth and shiny. Scarring alopecia can look different on different people. Some people also have redness, flaky skin or blisters.

### **What’s the difference between scarring and nonscarring alopecia?**

Scarring alopecia is permanent hair loss due to hair follicle destruction. In nonscarring alopecia, hair may fall out or get thinner, but your hair follicle isn’t destroyed. So nonscarring alopecia may be temporary, and your hair can sometimes grow back. Androgenetic alopecia, also called male or female pattern baldness, is the most common type of nonscarring alopecia.

### **Are there different types of scarring alopecia?**

Cicatricial alopecia can be one of two types:

* Primary scarring alopecia is due to an inflammatory or autoimmune disorder that directly targets and destroys your hair follicles.
* Secondary scarring alopecia is a side effect of injury or damage to your skin. Hair loss might result from burns, infections, radiation or tumors.

There are many types of primary scarring alopecia. Some include:

* Central centrifugal cicatricial alopecia (CCCA): CCCA is the most common type of hair loss in women, especially Black women. It usually occurs around age 30. CCCA often starts as a bald patch on the crown of your scalp and then spreads outward. It may be related to chemical hair products, such as relaxers, or hot hair tools, such as combs. CCCA can also result from too much tension on hair follicles from braids, weaves or extensions. But some research suggests that CCCA might be hereditary.
* Chronic cutaneous lupus erythematosus (CCLE): CCLE describes skin problems caused by lupus erythematosus, an autoimmune disease. In addition to hair loss, some people with CCLE also have skin rashes, discoloration and sensitivity to the sun. CCLE is more common in women.
* Folliculitis decalvans: Folliculitis decalvans may be the result of a bacterial infection. Some people with *Staphylococcus aureus* (*Staph A*), a staphylococcus infection, have ongoing inflammation that can affect their hair follicles.
* Lichen planopilaris: This is the most common primary scarring alopecia and mostly affects women over 50. It’s a type of lichen planus that can cause skin scaling, burning, itching, ulcers, discoloration and papules (inflamed bumps).

### **Is scarring alopecia contagious?**

Scarring alopecia isn’t contagious, so you can’t get it from person-to-person contact.

### **Who might be affected by scarring alopecia?**

Anyone can get scarring alopecia. It tends to affect adults more than children. People of a certain sex or race may be more likely to develop certain types of scarring alopecia. For example, central centrifugal cicatricial alopecia (CCCA) is the most common type of hair loss in Black women.

### **How common is scarring alopecia?**

Scarring alopecia accounts for about 7% of people who see healthcare providers about hair loss. So it’s much less common than nonscarring alopecia, which affects between 50% and 75% of adults over 50.

## **Causes**

In most types of scarring alopecia, there’s inflammation around the middle (bulge) of your hair follicle. This part of your follicle contains stem cells and oil glands, which are both needed for new hair growth. Inflammation destroys this part of the follicle and causes scar tissue to grow (fibrosis), so hair can’t regenerate.

The follicular ostia (openings in your skin where hair follicles reside) are usually closed in people with scarring alopecia.

In nonscarring alopecia, there’s usually damage to the base (bulb) of your hair follicle, not the bulge. That’s why hair can often regrow if you have nonscarring alopecia.

### Can cancer treatment cause scarring alopecia?

You may know that chemotherapy can cause hair loss. But chemotherapy doesn’t lead to scarring alopecia. Chemotherapy drugs can cause cells in your hair follicle to die, so your hair falls out. But the follicle itself isn’t destroyed, so hair can usually grow back after chemotherapy.

Radiation therapy, on the other hand, can lead to secondary scarring alopecia. Some people get skin burns from radiation therapy that permanently destroy hair follicles.

### **symptoms of scarring alopecia**

Hair loss is the main symptom of cicatricial alopecia. It usually causes focal hair loss, meaning you lose hair in patches. Hair loss might start slowly and happen gradually over many years. Or it can start suddenly and progress quickly.

Scarring alopecia tends to affect the scalp, but it can happen anywhere you have hair.

In addition to hair loss, some people experience skin problems, such as:

* Bleeding.
* Blistering.
* Burning.
* Crusting or scaling.
* Itching, tingling or tenderness.
* Pustules.
* Redness or other discoloration.

## **Diagnosis and Tests**

Your healthcare provider uses a few techniques to diagnose hair loss, including:

* Physical exam: Your provider looks at the location and pattern of your hair loss. They also check your skin where you’ve lost hair. This is to see if you still have hair follicle openings and visible signs of inflammation. Make sure to report when you first notice hair loss and any symptoms such as tingling, burning or itching.
* Medical history: Your provider does a careful review of your medical history. They evaluate your age, sex, hair care practices and overall health. Tell your provider about any medical conditions you have, such as lupus, anemia or thyroid disease. They might do blood tests to check for diseases that could cause hair loss.
* Skin biopsy: A skin biopsy can confirm a diagnosis of scarring alopecia. Your provider takes a small skin sample from your scalp or another area affected by hair loss. A pathologist (healthcare provider specializing in examining tissue samples) looks at the sample under a microscope. They check for inflammatory immune cells, which tell your provider what type of cicatricial alopecia you have.

### **Are there different stages of scarring alopecia?**

Sometimes a biopsy shows scar tissue but no inflammatory cells. Providers might refer to this as end-stage scarring alopecia (ESSA). ESSA means the condition is past the point of active inflammation, so treatment might be less effective.

### **What type of healthcare provider diagnoses scarring alopecia?**

A dermatologist (healthcare provider specializing in skin disorders) can diagnose scarring alopecia. But it can be a difficult condition to diagnose, so it’s important to see a dermatologist with a lot of experience in hair loss.

## **Management and Treatment**

There isn’t a cure for scarring alopecia. Treatment aims to manage symptoms and slow or stop more hair loss. Some cosmetic treatments can improve the appearance of bald patches.

### **How is scarring alopecia treated?**

Treatment depends on the type of cicatricial alopecia you have. Anti-inflammatory medication is the most common treatment for most forms of scarring alopecia. Anti-inflammatory medication fights the inflammatory cells causing hair follicle destruction. You might take a pill, such as hydroxychloroquine, or receive a corticosteroid injection, such as triamcinolone acetonide. Some anti-inflammatory medications come as creams or oils you apply to your skin.

Scarring alopecia caused by bacterial infections requires antibiotics, such as doxycycline or minocycline.

Most people take medication for six to 12 months until symptoms improve and hair loss slows or stops. You may need to start taking medication again if you start losing more hair.

### **Can I get a hair transplant for scarring alopecia?**

A hair transplant is surgery to move hair from one part of your scalp to an area with little or no hair. It’s usually not a good treatment for people with scarring alopecia. The scar tissue under the skin’s surface can prevent transferred hair from growing. But you may be a candidate for hair restoration if you haven’t lost new hair for a few years. Ask your healthcare provider if a hair transplant might be right for you.

### **Can I get platelet-rich plasma therapy for scarring alopecia?**

Platelet-rich plasma (PRP) uses cells from your own blood to reduce inflammation and help tissues heal. It can also help regrow your hair and treat hair loss caused by nonscarring alopecia. PRP therapy for scarring alopecia is less common, but some studies have shown positive results for certain types, such as lichen planopilaris.

### **Will my hair regrow if I have scarring alopecia?**

Scarring alopecia can cause permanent hair loss, so your hair will be difficult to regrow after the follicle is destroyed. But if you catch the condition very early, medication may be able to stop inflammation before it completely destroys the hair follicle.

## **Prevention**

### **How can I prevent scarring alopecia?**

It’s challenging to prevent scarring alopecia. Early treatment may help stop more hair loss.

## **Outlook / Prognosis**

### **What’s the outlook for someone with scarring alopecia?**

The outlook for scarring alopecia can vary depending on how soon you get treatment. Some people only experience small patches of hair loss. Others have widespread hair loss. Hair loss may return after treatment, so some people need to be on medication for a long time.

## **Living With**

### **What questions should I ask my healthcare provider about scarring alopecia?**

If you have scarring alopecia, there are a few questions you may want to ask your healthcare provider:

* Are there any natural or home remedies that can help scarring alopecia?
* Are there any side effects from being on medication for a long time?
* Are there community resources or support groups that can help me?
* Can changes to my diet or lifestyle help prevent further hair loss?
* Is there a chance I might go completely bald?

**Epidemiology**

Central centrifugal cicatricial alopecia is the most common type of cicatricial alopecia among middle-aged women of African descent; it is most common in those with a tightly curled or kinked hair configuration, with a reported prevalence varying between studies from 2% to 7%.. CCCA is uncommon in men and children, though it has been reported in some case studies. The mean age of onset of the disease in women is 36 years. These are the known characteristics of the disease process, and further population-level studies are needed as most studies are based on data in cities with small samples of patients. Currently, no well-documented published evidence about the involvement of CCCA in other populations is available.[[](https://www.ncbi.nlm.nih.gov/books/NBK559187/#)

**DIFFERENTIAL DIAGNOSIS**

Central centrifugal cicatricial alopecia clinically resembles:

* **Female pattern hair loss**: A form of nonscarring alopecia occurring after menopause or at puberty, associated with hyperandrogenism. The key feature to differentiate it from CCCA is the absence of scarring and the presence of visible follicular openings.
* **Lichen planopilaris**: This is a type of scarring alopecia that is also indistinguishable in some cases. In contrast to central centrifugal cicatricial alopecia, lichen planopilaris presents with perifollicular erythema and follicular keratosis. Frontal fibrosing alopecia is another form of lichen planopilaris. This is characterized by facial papules and slowly progressive scarring alopecia of the scalp. Alopecia also affects the eyelashes, eyebrows, and other body parts.
* **Tinea capitis:** This is a fungal infective condition of the scalp, differentiated by Wood lamp examination, which emits bright green fluorescence with microsporum and faint blue fluorescence with *Trichophyton schoenleinii* species. The condition presents with both scarring and non-scarring alopecia.
* **Discoid lupus erythematosus**: This is a form of scarring alopecia that usually affects the scalp; it appears as erythematous scaly plaques with follicular plugging along with pigmentary changes. The histological findings differentiate this condition from CCCA. Histopathology shows perivascular and periadnexal lymphohistiocytic infiltrate and interface dermatitis.The basal layer shows degenerative changes.
* **Pseudopelade of Brocq:** This condition usually affects middle-aged and older women and commonly presents as irregular patches of hair loss that usually begin at the vertex. The bald areas look like 'footprints in the snow.' Histopathology of the lesion shows a thin epidermis with sclerotic dermis and streamers of fibrosis that go up to the fat layer.

Lichen planopilaris presents with superficial perifollicular fibrosis, infundibular inflammation, and destruction, leading to free hair shafts in the dermis, similar to central centrifugal alopecia. Still, it is differentiated by vacuolar lichenoid dermatitis with epidermal cytoid bodies and peri-infundibular hypergranulosis. Dyskeratosis with perifollicular lymphocytic inflammation is a prevalent feature. A gradual progressive hair follicle loss with lymphohistiocytic infiltration and lamellar fibrosis around the isthmus and lower infundibulum is seen in frontal fibrosing alopecia.

REFERENCES

[Central Centrifugal Cicatricial Alopecia - StatPearls - NCBI Bookshelf](https://www.ncbi.nlm.nih.gov/books/NBK559187/)

[Scarring (Cicatricial) Alopecia: What It Looks Like & Treatment](https://my.clevelandclinic.org/health/diseases/24582-scarring-alopecia)

**Trichothiodystrophy**

**Alternative names**

* Amish brittle hair syndrome
* BIDS syndrome
* Brittle hair-intellectual impairment-decreased fertility-short stature syndrome
* IBIDS
* PIBIDS
* TTD

**Definition**

Trichothiodystrophy, commonly called TTD, is a rare inherited condition that affects many parts of the body. The hallmark of this condition is hair that is sparse and easily broken.

In people with trichothiodystrophy, tests show that the hair is lacking sulfur-containing proteins that normally gives hair its strength. A cross section of a cut hair shows alternating light and dark banding that has been described as a "tiger tail."

The signs and symptoms of trichothiodystrophy vary widely. Mild cases may involve only the hair. More severe cases also cause delayed development, significant intellectual disability, and recurrent infections; severely affected individuals may survive only into infancy or early childhood.

Mothers of children with trichothiodystrophy may experience problems during pregnancy including pregnancy-induced high blood pressure (preeclampsia) and a related condition called HELLP syndrome that can damage the liver. Babies with trichothiodystrophy are at increased risk of premature birth, low birth weight, and slow growth. Most children with trichothiodystrophy have short stature compared to others their age.

Intellectual disability and delayed development are common in people with trichothiodystrophy, although most affected individuals are highly social with an outgoing and engaging personality. Some people with trichothiodystrophy have brain abnormalities that can be seen with imaging tests. A common neurological feature of this disorder is impaired myelin production (dysmyelination). Myelin is a fatty substance that insulates nerve cells and promotes the rapid transmission of nerve impulses.

Trichothiodystrophy is also associated with recurrent infections, particularly respiratory infections, which can be life-threatening. People with trichothiodystrophy may have abnormal red blood cells, including red blood cells that are smaller than normal. They may also have elevated levels of a type of hemoglobin called A2, which is a protein found in red blood cells. Other features of trichothiodystrophy can include dry, scaly skin (ichthyosis); abnormalities of the fingernails and toenails; clouding of the lens in both eyes from birth (congenital cataracts); poor coordination; and skeletal abnormalities including degeneration of both hips at an early age.

About half of all people with trichothiodystrophy have a photosensitive form of the disorder, which causes them to be extremely sensitive to ultraviolet (UV) rays from sunlight. They develop a severe sunburn after spending just a few minutes in the sun. However, for reasons that are unclear, they do not develop other sun-related problems such as excessive freckling of the skin or an increased risk of skin cancer. Many people with trichothiodystrophy report that they do not sweat.

## **Who gets Trichothiodystrophy? (Age and Sex Distribution)**

* Trichothiodystrophy is a very rare inherited disorder, with only about 100 cases reported worldwide in the scientific literature
* It is a congenital disorder, and the onset of symptoms may occur at birth or in infancy
* Both genders may be affected by this disorder
* Individuals of all racial and ethnic groups may be affected

## **Risk Factors for Trichothiodystrophy**

In a vast majority of individuals, there are no identified risk factors for Trichothiodystrophy (TTD).

* A positive family history may be an important risk factor, since TTD can be inherited in some cases
* Children born to consanguineous parents may bear a higher risk of this disorder

It is important to note that having a risk factor does not mean that one will get the condition. A risk factor increases one’s chances of getting a condition compared to an individual without the risk factors. Some risk factors are more important than others.

Also, not having a risk factor does not mean that an individual will not get the condition. It is always important to discuss the effect of risk factors with your healthcare provider.

**causes**

Variants (also called mutations) in at least 10 genes have been found to cause trichothiodystrophy. Most cases of the photosensitive form of trichothiodystrophy result from variants in one of three genes: *ERCC2*, *ERCC3*, or *GTF2H5*. The proteins produced from these genes work together as part of a group of proteins called the general transcription factor 2 H (TFIIH) complex. This complex is involved in the repair of DNA damage, which can be caused by UV rays. The TFIIH complex also plays an important role in gene transcription, which is the first step in protein production.

Variants in the *ERCC2*, *ERCC3*, or *GTF2H5* genes reduce the amount of TFIIH complex within cells, which impairs both DNA repair and gene transcription. An inability to repair DNA damage probably underlies the sun sensitivity in affected individuals. Studies suggest that many of the other features of trichothiodystrophy may result from problems with the transcription of genes needed for normal development before and after birth.

Variants in at least seven genes have been reported to cause non-photosensitive forms of trichothiodystrophy. Variants in the *MPLKIP* gene account for fewer than 20 percent of all cases of non-photosensitive trichothiodystrophy. The protein produced from the *MPLKIP* gene does not appear to be involved in DNA repair. This protein interacts with another protein that is involved in processing and repairing RNA molecules, which are chemical cousins of DNA. Some forms of non-photosensitive trichothiodystrophy are caused by variants in genes that are also involved in RNA repair and protein production.

In some cases, the genetic cause of trichothiodystrophy is unknown.

### **Signs & Symptoms**

*Pregnancy and newborn*

The first sign an infant may have TTD can be before birth. Maternal complications during pregnancy such as pre-eclampsia, HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome, premature labor and placental abnormalities occur in approximately 80% of TTD pregnancies. TTD infants often have low birthweight (<2500 grams) and are born with a collodion membrane on the skin. The low birth weight and collodion membrane are serious complications, and many TTD infants are admitted to the neonatal intensive care unit (NICU). In addition to the skin abnormalities, TTD infants may have feeding difficulties and experience gastrointestinal reflux with aspiration and require nasogastric feeding tubes.

*Skin, hair, and nails*

When the collodion membrane peels off in the newborn period, the skin in many areas of the body may be dry and covered with fine scale called ichthyosis. The scale over time can thicken and darken especially at the waist and flanks; due to the scaly dry nature of their skin, TTD patients can have issues with chronic itching (pruritis). In addition, some children with TTD may not sweat very much and may have issues with hyperthermia in warmer climates. The skin on the palms and soles of the feet is often thickened and dry; painful fissures in flexural areas such as over the knuckles of the hands and instep of the foot can occur. Hyperlinear palms and soles can also be seen in many TTD children and adults. Eczema occurs in a small percentage of patients and increases the problems with the pruritus.

The scalp hair is brittle and can be sparse; it breaks easily and, in some patients, will fall out during fever (febrile) illnesses. The hair may be ‘fine’ and feel soft or it may feel more course and dry. It can be fragile and break off after a slight amount of ‘trauma’ such as combing, and for some affected people, hair breaks so easily that there are broken hairs on the pillow after sleeping. TTD patients may have more hair on the top of their heads and little to no hair on the sides or back. Testing of the hair in research laboratories shows low sulfur content and abnormal levels of the amino acid cystine.

The scalp skin can be dry, itchy, scaly rough and red (ichthyotic), and this can prevent hair growth.

The eyebrows are also sparse and brittle, and the hair of the eyebrows may break off as well. In many patients, the eyelashes may be nearly normal. TTD children often have a smaller receding chin, and larger appearing ears.

Onychodystrophy, or abnormal nails, is a common finding in TTD. The nails may be abnormally short, broad and ridged, and may be soft and split easily. They may also be thick, brittle and slow growing. The nails may demonstrate koilynichia, (spoon nails) a turning upward at the tips of the nails.

*Growth and feeding*

TTD children have short stature, many have poor weight gain, and will not grow along the standard growth curves. Most children and adults with TTD will be shorter than their peers.

People with TTD can have problems with chronic gastroesophageal reflux disease and may need medications and feeding modifications to manage the symptoms. Due to feeding problems and poor weight gain, some TTD children will have feeding tubes (either gastrostomy or nasogastric tubes) placed to augment oral feeding. As TTD children age, they may lose subcutaneous fat and have poor weight gain, resulting in a thinner prematurely aged-looking face.

*Neurologic*

Most children and adults with TTD have some form of developmental delay and/or intellectual disability. Small head size (microcephaly) is also a common finding. Often, these children do not walk or talk at the usual ages. Many children with TTD will have MRI scans of the brain for evaluation of the developmental delay. Most TTD children are found to have a reduced amount of myelin (white matter) in the brain and central nervous system. The myelin acts as an ‘insulator’ around nerve sheaths, helping to speed up nerve transmission. In addition, TTD children can have tremors and difficulty coordinating fine and gross muscle coordination. A few patients have developed seizures. Hearing loss can also be seen but it is generally due to recurrent ear infections and not nerve deafness. Despite the developmental delays, individuals with TTD are outgoing and highly social.

Minor infections may lead to prolonged illness with hospitalization, sometimes requiring ICU management. This often leads to regression with decline in functional abilities. When recovered from the infections, the children usually improve and regain most of their abilities. However, repeated infections can interfere with normal development over time. This is different from the progressive degeneration typical of patients with xeroderma pigmentosum (XP), a related disorder with mutations in some of the same genes as TTD (see below). In contrast to TTD, XP neurologic degeneration is slow, progressive, and occurs without repeated infections and hospitalizations. TTD patients without repeated infections and hospitalization may develop slowly but generally do not have the progressive loss of function that occurs with XP neurologic degeneration.

*Hematologic and immune*

TTD children may also have recurrent infections. The most common infections occur in the gastrointestinal and respiratory tracts. These infections can be life threatening. The children may have low levels of neutrophils, a type of white blood cell important in fighting infections. They may also have lower levels of immunoglobins especially immunoglobulin G (IgG) in the blood. This blood protein is also important in fighting infections.

*Eyes*

TTD patients have a wide variety of ocular abnormalities and ophthalmologic care is an important part of their health monitoring. In some patients, ocular abnormalities are present at birth; however other patients may not develop ocular problems until later in childhood. The most common findings in the newborn period are congenital cataracts and nystagmus (the eyes make repetitive, uncontrolled movements that can affect visual acuity and depth perception). Children with TTD can also develop cataracts at later ages, and identification and surgical removal of the cataracts as soon as they become visually significant is important to preserve eyesight. Other ocular abnormalities include small corneas (microcornea) and small eyeballs (microphthalmia) with decreased best corrected vision. As patients with TTD become older, they can develop dry eyes, leading to corneal surface abnormalities. Normally dry eye is a condition seen in older adults, and this TTD complication is often not identified until the child becomes symptomatic. A few TTD patients may develop macular/retinal degeneration as they age.

*Skeletal and dental*

Skeletal and dental abnormalities may also be present. TTD patients have been found to have unusual skeletal findings. They have thick dense bones (osteosclerosis) in the central skeleton including the skull, spine and pelvis. They also have thinner bone (osteopenia) in the peripheral bones of the lower arms, hands and feet. The bone symptoms can vary between people with the condition.

Some children with TTD develop debilitating hip degeneration leading to pain, inability to walk and avascular necrosis of the femoral head. The debilitating hip degeneration is seen most commonly in TTD children who also have the combination of osteosclerosis and osteopenia, and mutations in the *XPD/ERCC2* gene.

TTD patients often have poorly developed teeth. The tooth enamel is often thin and hypoplastic, leading to recurrent cavities (caries). TTD children may need extensive dental care including extractions and tooth caps.

### **Diagnosis**

An initial evaluation for TTD involves a diagnostic work-up, including obtaining a detailed history of the patient’s prenatal and neonatal history. A thorough physical exam is performed to assess clinical features such as hair abnormalities, short stature, small chin, ichthyosis, intellectual impairment or developmental delay, cataracts, cryptorchidism (in males) and bone and teeth anomalies. Evaluation by a developmental pediatrician or neurologist may determine whether there is any developmental delay or intellectual disability. MRI imaging of the brain to identify abnormal patterns of myelination is often performed. Laboratory testing for immune function, blood count, low red blood cell MCV, elevated hemoglobin A2 level and iron levels can also be performed. TTD is often diagnosed by polarized light microscopy of hair shafts, revealing a tiger-tail pattern. The classical tiger-tail pattern alone usually is enough to diagnose TTD. However, there are other conditions with similar hair shaft abnormalities and often genetic testing is needed to confirm the diagnosis. Some patients with features of TTD will not have mutations in the known genes. They may have mutations in yet to be identified TTD associated genes.

Many infants with TTD will be discharged from the NICU after several weeks, then require close medical monitoring at home for several months.

TTD patients generally have complex health care needs and benefit from a multidisciplinary approach to their medical management. No formal guidelines for the medical management of TTD exist and management is largely based on symptoms.

Patients who are sensitive to ultraviolent rays must be protected from exposure to the sun and other sources of ultraviolet radiation to prevent severe burns. Patients should avoid being outside for prolonged periods without protection. They should wear hats, sunglasses and clothing to cover their skin, such as long sleeves and long pants. When going outside during the day, they should have sunscreen applied to uncovered skin such as face, neck, ears and hands. UV can come from other artificial light sources such as halogen and fluorescent light bulbs and mercury vapor lamps. Photosensitive patients should avoid these types of light sources. Despite the burning on sun exposure, skin cancer has only very rarely been reported in patients with TTD, there are a few very rare patients who exhibit symptoms of both xeroderma pigmentosum (XP) and TTD (the XP/TTD syndrome) and may develop skin cancers after UV exposure. These patients require more stringent UV protection.

Management of the ichthyosis and dry skin varies with severity. For some patients it is a minor issue but for others management is a daily process of gently removing thickened scale during bathing and followed immediately by moisturizing the skin. Moisturizers or emollient creams and lotions are designed to make the external layers of the skin (epidermis) softer and more pliable. They also increase the skin’s hydration (water content) by reducing evaporation. The moisturizers ideally are fragrance and dye free to avoid allergic reactions. It can be helpful to include the nails when moisturizing the skin of the hands and palms. Applying oil to the scalp and then washing the hair with a gentle shampoo designed for scaling skin conditions can help loosen the scale.

Some children with TTD have received IgG infusions to help treat the recurrent infections. Anemia and low iron levels can be treated with dietary iron supplementation.

Monitoring is needed for developmental delay and special education services may be required in school. The children should be evaluated for rehabilitation needs. Ongoing physical therapy may be advised for joint stiffness, muscle tightening (contractures), and poor coordination.

Genetic counseling is recommended for families of children with trichothiodystrophy.

### **Disorders with Similar Symptoms**

Symptoms of the following disorders may resemble those of trichothiodystrophy. Comparisons may be useful for a differential diagnosis.

Ichthyoses or “disorders of cornification” are general terms describing a group of scaly skin disorders. (See “ichthyosis” in the Rare Disease Database for more information.)

Xeroderma pigmentosum is a disorder in which damage done by ultraviolet light (especially sunlight) is not repaired properly leading to dry skin with heavy freckling and development of skin cancer at a young age. Patients may also have progressive neurologic impairment. Xeroderma pigmentosum is also caused by alterations in genes involved in DNA repair

### **Affected populations**

TTD presents at birth. Males and females are affected in equal numbers. The estimated incidence is about 1 in 1,000,000 newborns in the United States and Europe. Over 100 patients have been reported worldwide. TTD has been reported in all ethnic groups.

REFERENCES

[Trichothiodystrophy - Symptoms, Causes, Treatment | NORD](https://rarediseases.org/rare-diseases/ichthyosis-trichothiodystrophy/#disease-overview-main)

[Trichothiodystrophy: MedlinePlus Genetics](https://medlineplus.gov/genetics/condition/trichothiodystrophy/)

[Trichothiodystrophy - DoveMed](https://www.dovemed.com/diseases-conditions/trichothiodystrophy)

### 

### 

### 

### 

### 

### **uncombable hair syndrome**

**OTHER NAMES**

* Cheveux incoiffables
* Pili trianguli et canaliculi
* Spun glass hair
* UHS
* Unmanageable hair syndrome

Uncombable hair syndrome is a genetic condition that causes your child’s hair to grow in a way that can’t be flattened down with a comb. This happens because your child’s hair grows in multiple directions instead of down. Hair may be a lighter tone, dry and frizzy. The hair on the rest of your child’s body grows as expected and the condition only affects the hair on your child’s scalp. The condition resolves itself over time.

### **Who does uncombable hair syndrome affect?**

Uncombable hair syndrome can affect anyone since a genetic mutation causes the condition. Several genes cause this condition and based on which one causes your symptoms, you can inherit it during conception either from both biological parents (autosomal recessive) or from only one parent (autosomal dominant).

### **How rare is uncombable hair syndrome?**

The exact rate of occurrence is unknown because the condition goes away as children grow into adults. There are over 100 cases of uncombable hair syndrome recorded in medical literature but more than 100 cases exist.

### **How does uncombable hair syndrome affect my body?**

Uncombable hair syndrome only affects the hair on your child’s scalp. It causes their hair to grow in all directions instead of down. It can be difficult to comb or brush your child’s hair, which is where the condition gets its name. This condition doesn’t affect any other parts of your child’s body other than the hair on their head.

## **Symptoms and Causes**

Symptoms of uncombable hair syndrome affect the hair on your child’s scalp and include hair that’s:

* Coarse or has a rough texture.
* Frizzy and can’t be combed or brushed smooth or flat.
* Light (hypopigmented) in color or appears silver, white or blonde to light brown.
* Shiny.
* Dry.
* Untamed and grows in all directions instead of only growing downward.

Not all children diagnosed with this condition will experience all symptoms. For example, the tone or color of a person’s hair could be naturally black or brown but still be uncombable.

#### **What age do symptoms of uncombable hair syndrome show up?**

Uncombable hair syndrome begins during infancy and symptoms are usually present around age 3. Symptoms vary for each person and can begin as a baby or appear later in childhood by age 12. Symptoms slowly go away during adolescence and early adulthood, when your child’s hair will grow down and lay flat.

### **What causes uncombable hair syndrome?**

A genetic mutation causes uncombable hair syndrome. One of the following genes causes this condition:

* *PADI3*.
* *TGM3*.
* *TCHH*.
* An additional gene not yet identified.

These genes provide instructions that tell your hair strands to grow in a cylindrical shape, which is the same shape as a tin can. The cylinder shape guides your hair to grow in one direction out of your hair follicle, similar to how a vase holds a flower to help it grow upright.

If you have a genetic mutation on the *PADI3*, *TGM3* or *TCHH* gene, it affects the shape and structure of your hair shaft. The shape of your hair shaft could be a triangle, octagon or heart instead of a cylinder. The angles or points in your hair shaft change the direction in which the strand will grow.

## **Diagnosis and Tests**

### **How is uncombable hair syndrome diagnosed?**

Your child’s provider will diagnose uncombable hair syndrome after a complete medical history and a physical exam, where your provider will learn more about your child’s symptoms. Symptoms of this condition are unique and usually lead to a diagnosis after a visual examination of the hair on your child’s scalp.

### **What tests diagnose uncombable hair syndrome?**

Your provider may offer tests to confirm an uncombable hair syndrome diagnosis, including:

* Hair shaft test: Your provider will remove a strand of hair to examine the shaft — the visible part of the hair that sticks out of your child’s skin — under a microscope. Your provider will look for an irregular shape, which will lead to an uncombable hair syndrome diagnosis.
* Genetic test: Your provider will remove a small sample of your child’s blood to look for any changes to your child’s genetic code. If your provider detects a genetic mutation, it’ll lead to an uncombable hair syndrome diagnosis.

## **Management and Treatment**

### **How is uncombable hair syndrome treated or managed?**

There’s no treatment available for uncombable hair syndrome. It may be challenging to manage hair that grows in every direction and can’t lay flat, but you can take steps to make your child’s hair care routine easier by:

* Not using hair treatments that involve a lot of harsh chemicals like perms or dyes, since they can be ineffective or worsen symptoms.
* Not over-brushing or over-combing your child’s hair.
* Limiting how often you use tools on your child’s hair, like a curling iron or a blow dryer.
* Regularly cutting or trimming your child’s hair.

#### **What hair products tame uncombable hair?**

Each person’s hair reacts differently to hair products and hair treatments. In general, hair products like detanglers, conditioners or hair masks don’t work well on hair affected by uncombable hair syndrome. Stronger chemicals to treat hair, like perms or hair relaxers, have trouble binding to hair strands and rarely offer benefits to your hair if you have uncombable hair syndrome.

### **How long does uncombable hair syndrome last?**

Uncombable hair syndrome starts to resolve itself or go away during adolescence, usually around the onset of puberty. The condition could last into early adulthood. During this time, your child’s hair will start to grow in one direction (downward) instead of in multiple different directions. It could take several years for all of their hair strands to start growing in one direction.

## **Prevention**

You can’t prevent uncombable hair syndrome since it’s caused by a genetic mutation. To learn more about your risk of having a child with a genetic condition, talk to your provider about genetic testing.

## **Outlook / Prognosis**

### **What can I expect if I have uncombable hair syndrome?**

Uncombable hair syndrome is a short-term condition that goes away as your child grows into an adult. While their hair may be difficult to manage, it can be easier to work with if you stay consistent with haircuts or choose to keep it at a shorter length. Perms, straightening chemicals or hair treatments may not work as expected, so it’s best to avoid products that could further damage your child’s hair. Their hair will grow at a normal rate or a slightly slower rate than expected.

Some children may have low self-esteem since their hair looks different from their peers. Your child may benefit from talking to a mental health professional throughout childhood to improve their self-image.

## **Living With**

### **When should I see my healthcare provider?**

Visit your healthcare provider if your child has trouble with their self-esteem and mental health as a result of their uncombable hair syndrome diagnosis. Symptoms of the condition don’t affect your child’s overall health and are only cosmetic.

### **What questions should I ask my doctor?**

* How do I manage my child’s uncombable hair?
* How often should I take my child to get their hair cut?
* Do you recommend any hair products to tame uncombable hair?

REFERENCES

[Uncombable Hair Syndrome: What It Is, Cause & Treatment](https://my.clevelandclinic.org/health/diseases/24688-uncombable-hair-syndrome)