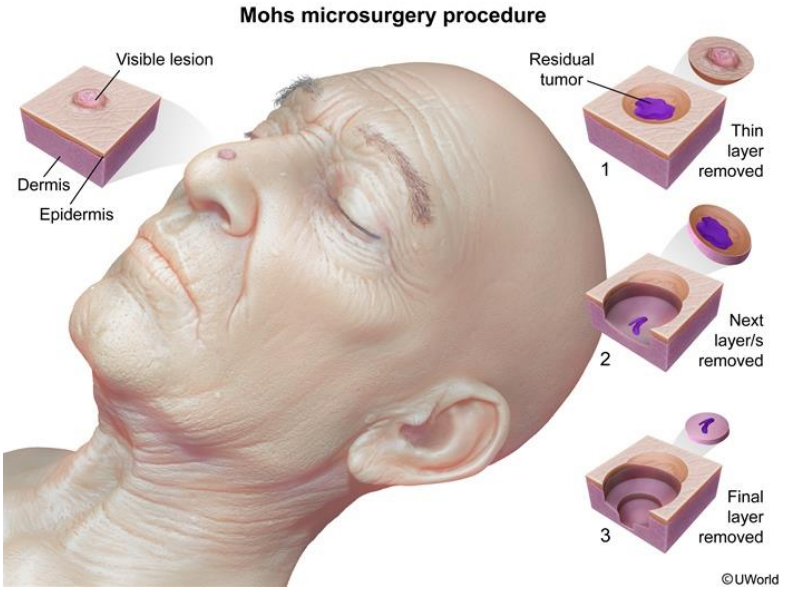


DERMATOLOGY

UWorld Step 2 Tables and Images (Subject)

Table of Contents

Dermatology

Disorders of epidermal appendages

Hair loss

Female & male pattern hair loss

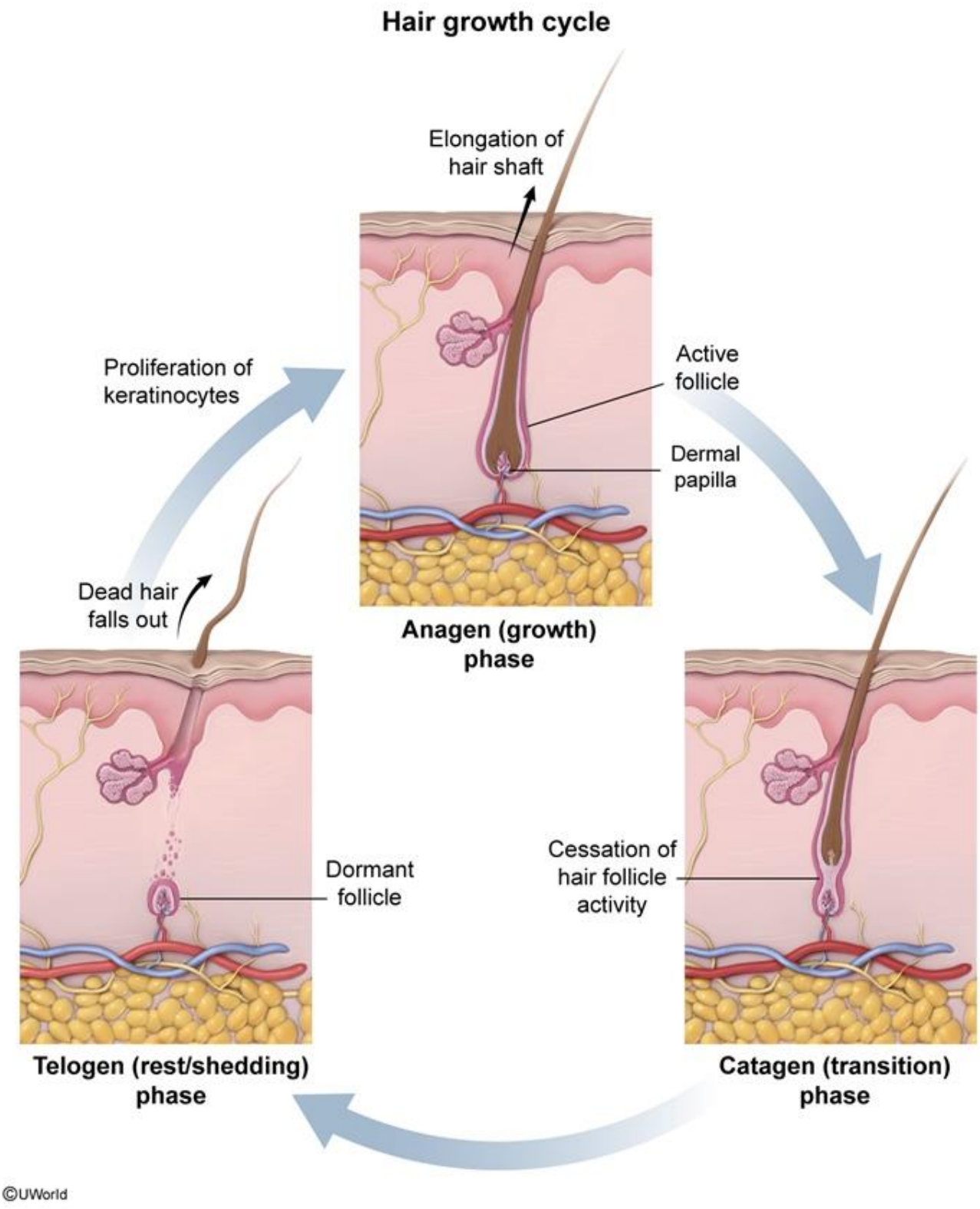
Etiology	• Polygenetic inheritance • Hormonal factors (dihydrotestosterone)
Clinical features	• Chronic, progressive thinning of hair • **Men:** vertex, frontal hairline, temporal areas • **Women:** vertex, center of scalp (sparing of hairline)
Management	• **Men:** minoxidil, finasteride • **Women:** minoxidil

Alopecia areata

Pathogenesis	• Autoimmune attack on hair bulb cells • Genetic predisposition
Clinical presentation	• Painless, patchy, nonscarring hair loss • Narrowing of hair shafts close to skin surface (exclamation point hairs) • Positive hair pull test (>5-6 hairs extracted)
Management	• **Mild/moderate hair loss:** topical or intralesional corticosteroids • **Extensive hair loss:** topical immunotherapy (eg, diphenylcyclopropenone), oral corticosteroids

Telogen effluvium

Clinical findings	• Acute, diffuse, noninflammatory hair loss • Scalp & hair fibers appear normal • Hair shafts easily pulled out (hair pull test)
Triggers	• Severe illness, fever, surgery • Pregnancy, childbirth • Emotional distress • Endocrine & nutritional disorders
Management	• Address underlying cause • Reassurance (self-limited disorder)



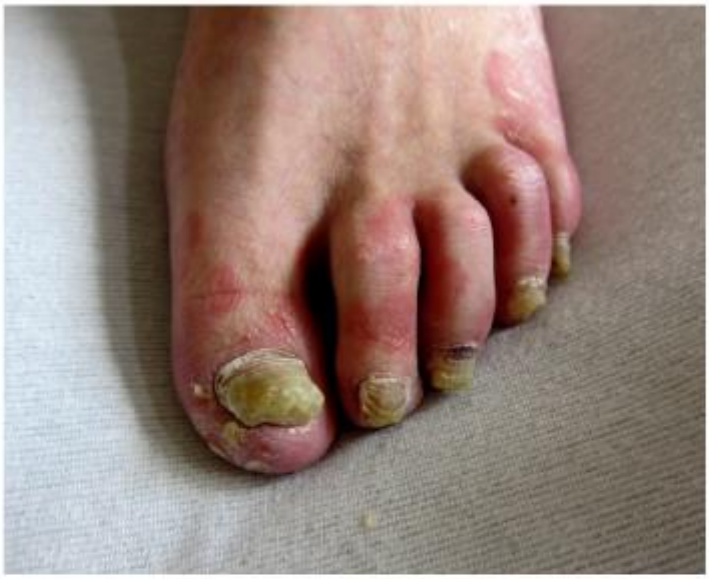
Hair growth cycle

Ichthyosis vulgaris

Pathophysiology	• Loss of function in filaggrin gene – Impaired epidermal barrier – Reduced skin moisturization
Clinical features	• Onset in infancy/early childhood • Diffuse, scaly skin with mild pruritus • Worse on extensor extremities, spares intertriginous areas
Associated conditions	• Keratosis pilaris • Palmar hyperlinearity • Atopic disease
Diagnosis	• Clinical findings • Biopsy if uncertain: reduced/absent granular layer in epidermis
Therapy	• Long baths to remove scales • Moisturization • Keratolytics (eg, urea, alpha-hydroxy acid, salicylic acid)

Benign neonatal rashes

Diagnosis	Onset	Clinical features	Management/resolution
Erythema toxicum neonatorum	• Birth to age 3 days	• Pustules with erythematous base on trunk & proximal extremities	• Observation • Resolves within a week
Milia	• Birth	• Firm, white papules on face	• Observation • Resolves within a month
Miliaria rubra	• Any age, but not present at birth	• Erythematous, papular rash on occluded & intertriginous areas	• Avoid overheating (eg, cool environment, thin/cotton clothing) • If severe, topical corticosteroid
Neonatal pustular melanosis	• Birth	• Nonerythematous pustules → evolve into hyperpigmented macules with collarette of scale • Diffuse, may involve palms & soles	• Observation • Pustules resolve within days • Hyperpigmentation may last months
Neonatal cephalic pustulosis	• Around age 3 weeks	• Erythematous papules & pustules on face & scalp only	• Observation • Resolves in weeks to months • If severe, topical corticosteroid or ketoconazole

Onychomycosis	
	
Risk factors	• Advanced age • Tinea pedis • Diabetes • Peripheral vascular disease
Examination findings	Thick, brittle, discolored nails
Diagnosis	KOH, periodic acid–Schiff stain, culture
Treatment	• **First line:** terbinafine, itraconazole • **Second line:** griseofulvin, fluconazole, ciclopirox

KOH = potassium hydroxide.

Onychomycosis

PSORIASIS

Plaque psoriasis

Skin lesions	• Well-defined, erythematous plaques with silvery scale • Extensor surfaces (knees, elbows), hands, scalp, back, nail plates
Extradermal manifestations	• Nail pitting • Conjunctivitis, uveitis • Psoriatic arthritis
Treatment	• **Topical:** high-potency glucocorticoids, vitamin D analogs, tar, retinoids, calcineurin inhibitors, tazarotene • Ultraviolet light/phototherapy • **Systemic:** methotrexate, calcineurin inhibitors, retinoids, apremilast, biologic agents

Inflammatory dermatoses and bullous diseases

ATOPIC DERMATITIS

Atopic dermatitis (eczema)

Risk factors	• Family history of atopy (eczema, asthma, allergic rhinitis) • Mutation in filaggrin gene
Clinical features	• Acute: pruritic, erythematous patches & papules – Infant: extensor surfaces, trunk & face – Child/adult: flexural creases • Chronic: lichenified plaques
Treatment	• Topical emollients • First line: topical corticosteroids • Second line: topical calcineurin inhibitors (eg, pimecrolimus)
Complications	• Secondary infection – Impetigo (eg, *Staphylococcus aureus*) – Eczema herpeticum (ie, herpes simplex virus)
Prognosis	• Chronic with intermittent flares in early childhood • Usually resolves by adulthood

BULLOUS PEMPHIGOID

Bullous pemphigoid

Clinical features	• Age >60 • Pruritic, tense bullae • Rare mucosal involvement • Prodrome of eczematous/urticaria-like rash
Associated disorders	• Dementia • Parkinson disease • Depression, bipolar disorder
Diagnosis	• Histology: subepidermal cleavage • Immunofluorescence: linear IgG/C3 deposition along basement membrane • Serology: autoantibodies to bullous pemphigoid antigens (hemidesmosomes)
Treatment	• Topical therapies: high-potency corticosteroids • Systemic therapies: corticosteroid, doxycycline

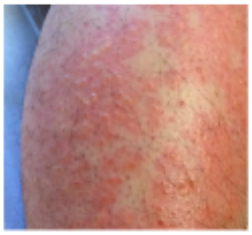
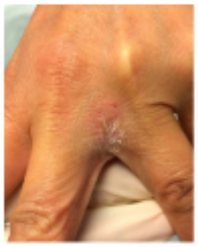
CONTACT DERMATITIS

Hypersensitivity reactions

	Immunology	**Examples**
Type I (immediate)	IgE-mediated	• Anaphylaxis • Urticaria
Type II (cytotoxic)	IgG & IgM autoantibody-mediated	• Autoimmune hemolytic anemia • Goodpasture syndrome
Type III (immune complex)	Antibody-antigen complex deposition	• Serum sickness • Poststreptococcal glomerulonephritis • Lupus nephritis
Type IV (delayed type)	T cell- & macrophage-mediated	• Contact dermatitis • Tuberculin skin test

Allergic contact dermatitis

Pathophysiology	• Type IV hypersensitivity reaction • Common triggers: poison ivy/oak, nickel, dyes, topical medications, skin care products
Clinical presentation	• Acute: pruritic red, indurated plaques with vesicles/bullae • Chronic: lichenification, fissuring
Diagnosis	• Clinical findings • Patch testing for persistent cases
Treatment	• Avoidance of suspected allergen • Topical or systemic corticosteroid

Contact dermatitis		
	Allergic 	**Irritant** 
Pathophysiology	Type IV hypersensitivity	Physical or chemical irritation
Triggers	• Poison oak/ivy/sumac • Nickel • Rubber/latex • Leather dyes • Medications	• Soaps/detergents • Chemicals • Acid/alkali
Appearance	• Primarily on exposed skin, well demarcated • Erythema • Papules/vesicles • Chronic lichenification	• Commonly on hands • Erythema • Fissures

Contact dermatitis

DYSHIDROTIC ECZEMA

Acute palmoplantar eczema (dyshidrotic eczema)

Clinical features	• Recurrent, acute episodes • Deep-seated, pruritic vesicles & bullae at hands & feet • Complications: desquamation, chronic dermatitis, secondary infection
Diagnosis	• Clinical features usually adequate for diagnosis • Biopsy: intraepidermal spongiosis, lymphocytic infiltrate
Treatment	• Topical emollients • High/super high–potency topical corticosteroids

ERYTHEMA NODOSUM

Erythema nodosum

Clinical features	• Tender, indurated, erythematous nodules • Most common on anterior legs
Etiology	• Infections (eg, *Streptococcus*) • Inflammatory bowel disease • Sarcoidosis • Medications (eg, antibiotics, oral contraceptives)
Pathologic findings	• Septal panniculitis without vasculitis
Natural history	• Spontaneous resolution (weeks) • Residual hyperpigmentation

HSV INFECTION

Infectious complications of atopic dermatitis

Diagnosis	Pathogen	Presentation
Impetigo	*Staphylococcus aureus* *Streptococcus pyogenes*	• Papules & pustules with honey-crusted adherent coating • ± Pain or pruritus
Eczema herpeticum	Herpes simplex type 1	• Painful vesicular rash • "Punched-out" erosions & hemorrhagic crusting
Molluscum contagiosum	Poxvirus	• Skin-colored papules with central umbilication
Tinea corporis	*Trichophyton rubrum*	• Pruritic circular patch with central clearing • Raised, scaly border

Hidradenitis suppurativa

Pathogenesis	• Disordered folliculopilosebaceous units: – Ductal keratinocyte proliferation → follicular occlusion → follicular rupture → inflammation • Risk factors: smoking, metabolic syndrome (obesity, diabetes mellitus)
Clinical presentation	• Chronic & recurrent lesions in intertriginous areas • Mild: painful nodules, draining abscesses • Moderate: sinus tracts & scarring • Severe: extensive sinus tracts, widespread disease
Treatment	• Mild: topical clindamycin • Moderate: oral tetracycline • Severe: tumor necrosis factor-α inhibitors (eg, adalimumab), surgical excision
Complications	• Depression & suicide • Squamous cell carcinoma of skin

Henoch-Schönlein purpura (IgA vasculitis)

Pathogenesis	• Perivenular leukocytoclastic (neutrophils & monocytes) vasculitis • Deposition of IgA, C3 & fibrin in small vessels
Clinical manifestations	• Classic findings*: – Palpable purpura/petechiae on lower extremities – Arthritis/arthralgia – Abdominal pain, intussusception – Renal disease (similar to IgA nephropathy) • Other findings: scrotal pain & swelling
Laboratory findings	• Normal platelet count & coagulation studies • Normal to ↑ creatinine • Hematuria ± RBC casts &/or proteinuria
Management	• Supportive care (hydration & NSAIDs) for most patients • Hospitalization & systemic glucocorticoids for severe symptoms

*Clinical diagnosis requires purpuric rash plus ≥2 additional classic findings.

NSAIDs = nonsteroidal anti-inflammatory drugs; **RBC** = red blood cell.

Lichen planus

Clinical findings	• 5 "Ps": pruritic, purple/pink, polygonal papules & plaques • Lacy, white network of lines (Wickham striae)
Disease associations	• Hepatitis C • Medications: ACE inhibitors, thiazide diuretics
Natural history	• Chronic symptoms • Formation of lesions at sites of trauma (Köbner reaction) • Spontaneous resolution within 2 years
Treatment	• Topical high-potency glucocorticoids (eg, betamethasone) • Widespread lesions: systemic glucocorticoids, phototherapy

Lichen planus

Clinical findings	• 5 "Ps": pruritic, purple/pink, polygonal, papules & plaques • Lacy, white network of lines (Wickham striae) • Locations: – Skin (eg, ankles, wrists) – Oral mucosa (white papules & plaques ± erythema, mucosal atrophy, ulcers) – Genitalia
Disease associations	• Hepatitis C • Medications: ACE inhibitors, thiazide diuretics
Natural history	• Chronic symptoms • Formation of lesions at sites of trauma (Köbner phenomenon) • Spontaneous resolution within 2 years
Treatment	• Topical high-potency glucocorticoids (eg, betamethasone) • Widespread lesions: systemic glucocorticoids, phototherapy

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PEMPHIGUS VULGARIS

Pemphigus vulgaris vs bullous pemphigoid

	Pemphigus vulgaris	**Bullous pemphigoid**
Age of onset	• 40-60	• >60
Clinical features	• Painful • Flaccid bullae → erosions • Mucosal involvement common	• Pruritic • Tense bullae • Mucosal involvement rare
Histology	• Intraepidermal cleavage	• Subepidermal cleavage
Immunofluorescence	• Net-like intercellular IgG against desmosomes	• Linear IgG against hemidesmosomes along basement membrane

PITYRIASIS ROSEA

Pityriasis rosea

Clinical features	• ± Viral prodrome • Annular, pink herald patch on trunk • Oval lesions in "Christmas tree" pattern • Pruritus
Management	• Reassurance (spontaneous resolution) • Treatment of pruritus (eg, antihistamines)

Skin conditions & associated diseases

Skin conditions	Associated conditions
• Acanthosis nigricans	• Insulin resistance • Gastrointestinal malignancy
• Multiple skin tags	• Insulin resistance • Pregnancy • Crohn disease (perianal)
• Porphyria cutanea tarda • Cutaneous leukocytoclastic vasculitis (palpable pupura) secondary to cryoglobulinemia	• Hepatitis C
• Dermatitis herpetiformis	• Celiac disease
• Sudden-onset, severe psoriasis • Recurrent herpes zoster • Disseminated molluscum contagiosum	• HIV infection
• Severe seborrheic dermatitis	• HIV infection • Parkinson disease
• Explosive onset multiple, itchy seborrheic keratoses	• Gastrointestinal malignancy
• Pyoderma gangrenosum	• Inflammatory bowel disease

Pyoderma gangrenosum

Clinical features	• Begins with small papule or pustule • Rapidly progressive, painful ulcer with purulent base & violaceous border • Precipitation of ulceration at site of injury (pathergy)
Epidemiology	• Peak onset age 40-60 • Women > men • Association with inflammatory bowel disease, inflammatory (eg, rheumatoid) arthritis, malignancy
Diagnosis	• Exclusion of other causes of ulceration (eg, infection) • Skin biopsy: mixed inflammation (neutrophil predominant)
Treatment	• Local or systemic glucocorticoids

Treatment of rosacea

General measures	• Avoidance of sun exposure, hot/spicy foods, alcohol • Gentle cleansers & emollients
Erythematotelangiectatic rosacea (flushing, erythema, telangiectasia)	• Topical brimonidine • Laser/intense pulsed light therapy
Papulopustular rosacea (small papules & pustules)	• **First line:** topical metronidazole, azelaic acid, ivermectin • **Second line:** oral tetracyclines
Phymatous rosacea (irregular thickening of skin)	• Oral isotretinoin • Laser therapy/surgery
Ocular rosacea (burning/foreign body sensations, blepharitis, keratitis, conjunctivitis, corneal ulcers)	• Lid scrubs & ocular lubricants • Topical or systemic antibiotics (eg, metronidazole, macrolides)

Rosacea

Erythemato-telangiectatic	• Persistent facial erythema/flushing • Telangiectasias
Papulopustular	• Papules & pustules on central face
Ocular	• Conjunctival hyperemia • Lid margin telangiectasias
Treatment	• Avoidance of triggers (eg, alcohol, spicy foods) • Sun protection • Gentle cleansers & emollients • Topical metronidazole for papulopustular type • Laser or topical brimonidine (vasoconstrictive α-2 agonist) for erythematotelangiectatic type

SEBORRHEIC DERMATITIS

Seborrheic dermatitis in adults

Clinical features	• Erythematous, pruritic plaques with greasy scales • Scalp, central face, ears, chest
Risk factors	• CNS disease (eg, Parkinson disease) • HIV
Treatment	• Topical antifungals (eg, selenium sulfide, ketoconazole) • Topical glucocorticoids • Topical calcineurin inhibitors (eg, pimecrolimus)

TINEA

Tinea capitis

Epidemiology	• Most common in African American children • Transmission via direct contact or from fomite (eg, shared combs)
Clinical features	• Scaly, erythematous patch with hair loss on scalp • ± Black dots in affected area • ± Tender lymphadenopathy
Management	• Oral griseofulvin or terbinafine

Stevens-Johnson syndrome & toxic epidermal necrolysis

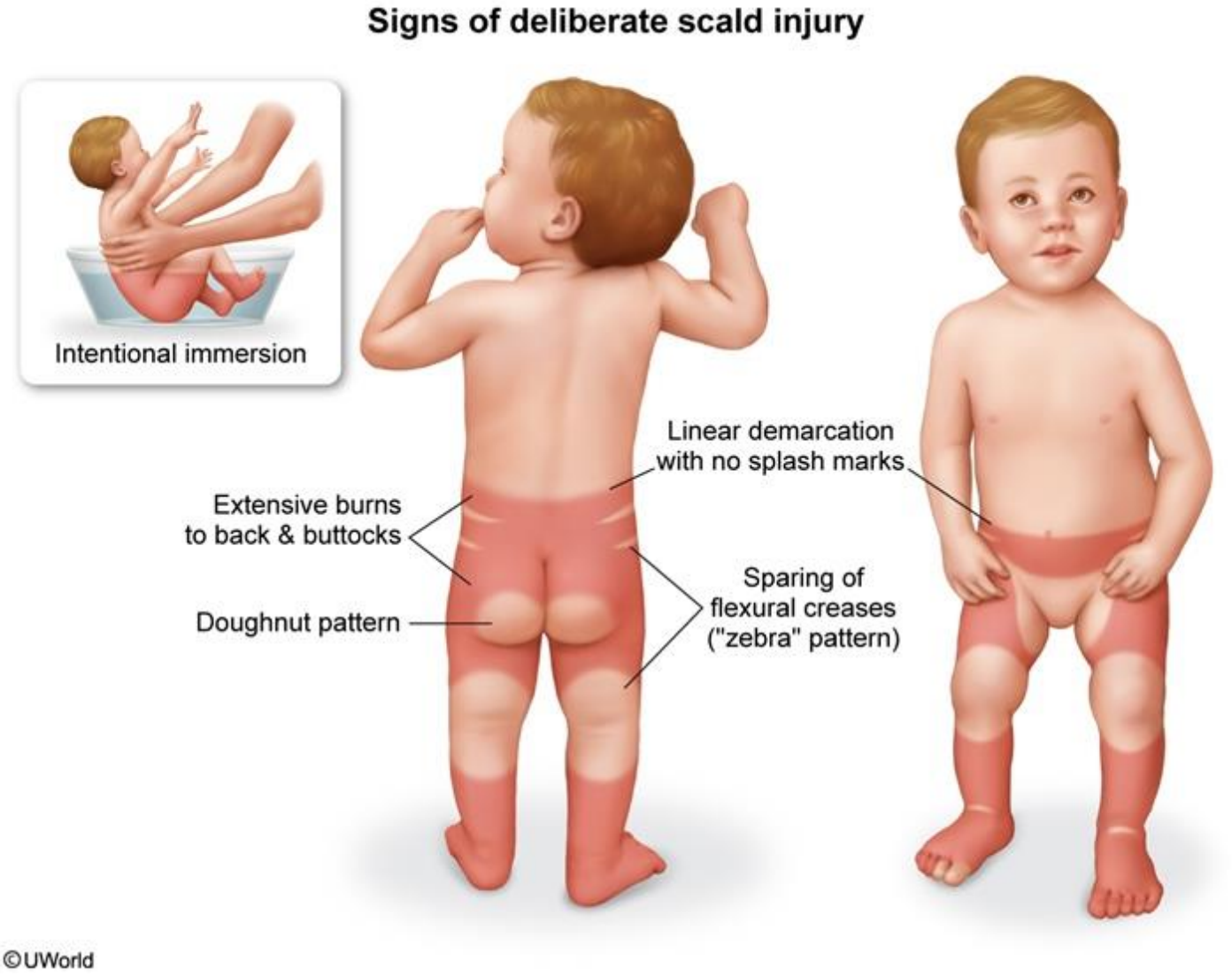
Nomenclature	<10% of BSA: SJS 10%-30%: SJS/TEN overlap >30%: TEN
Clinical features	• 4-28 days after exposure to trigger (2 days after repeat exposure) • Acute influenza-like **prodrome** • Rapid-onset erythematous macules, vesicles, bullae • Necrosis & **sloughing of epidermis** • **Mucosal involvement**
Common triggers	**Drugs** • Allopurinol • Antibiotics (eg, sulfonamides) • Anticonvulsants (eg, carbamazepine, lamotrigine, phenytoin) • NSAIDs (eg, piroxicam) • Sulfasalazine **Other** • *Mycoplasma pneumoniae* • Vaccination • Graft-vs-host disease

BSA = body surface area; **NSAIDs** = nonsteroidal anti-inflammatory drugs**; SJS** = Stevens-Johnson syndrome; **TEN** = toxic epidermal necrolysis.

Miscellaneous

Common drugs associated with photosensitivity reactions

Antibiotics	Tetracyclines (eg, doxycycline)
Antipsychotics	Chlorpromazine, prochlorperazine
Diuretics	Furosemide, hydrochlorothiazide
Others	Amiodarone, promethazine, piroxicam



Signs of deliberate scald injury

DIAPER DERMATITIS

Diaper dermatitis

Diagnosis	Irritant contact dermatitis	*Candida* dermatitis
Pathogenesis	• Skin breakdown from exposure to stool/urine • Most common diaper rash	• Yeast superinfection of irritant contact dermatitis • Second most common diaper rash
Examination	• Erythematous papules, plaques • Spares skinfolds	• Beefy-red confluent plaques • Involves skinfolds • Satellite lesions
Treatment	• Topical barrier (eg, petrolatum, zinc oxide)	• Topical antifungal (eg, nystatin)

Clinical features of chronic hepatitis C

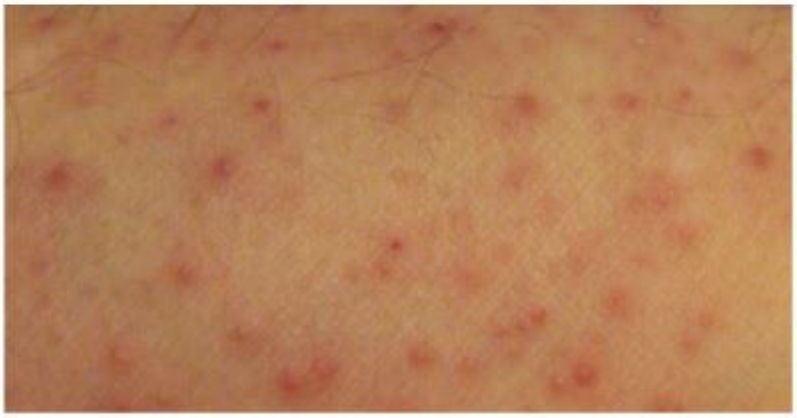
Clinical manifestations	• Asymptomatic or nonspecific symptoms (eg, fatigue, anorexia, arthralgia/myalgia) • Hepatic manifestations: cirrhosis, hepatocellular carcinoma • Extrahepatic manifestations – Hematologic: mixed cryoglobulinemia, ITP, autoimmune hemolytic anemia – Renal: membranoproliferative glomerulonephritis – Dermatologic: porphyria cutanea tarda, lichen planus
Laboratory findings	• Anti-HCV IgG antibodies (screening): current or past infection • HCV RNA PCR (confirmatory/diagnostic): active infection • Elevated AST, ALT • Thrombocytopenia (cirrhosis or ITP) • False-positive antinuclear antibodies & rheumatoid factor • Liver biopsy: portal triad lymphocytic inflammation, lymphoid follicles, fibrosis & regenerative nodules (cirrhosis)

ALT = alanine aminotransferase; **AST** = aspartate aminotransferase; **HCV** = hepatitis C virus; **ITP** = immune thrombocytopenia.

Common insect bites/infestation	
Insect	**Clinical features***
Bed bug 	Pruritic, small puncta & maculopapules in linear groups ("breakfast, lunch, dinner" pattern) on unclothed skin
Pediculosis 	Widespread itching of hair, body, or genitalia with visible louse
Scabies 	Pruritic burrows or hemorrhagic crusts in intertriginous areas
Spider 	Solitary papule, pustule, or wheal +/- pruritus
Tick 	Painless red papule +/- pruritus during the spring & summer

*Some patients have no symptoms

Common insect bites/infestation

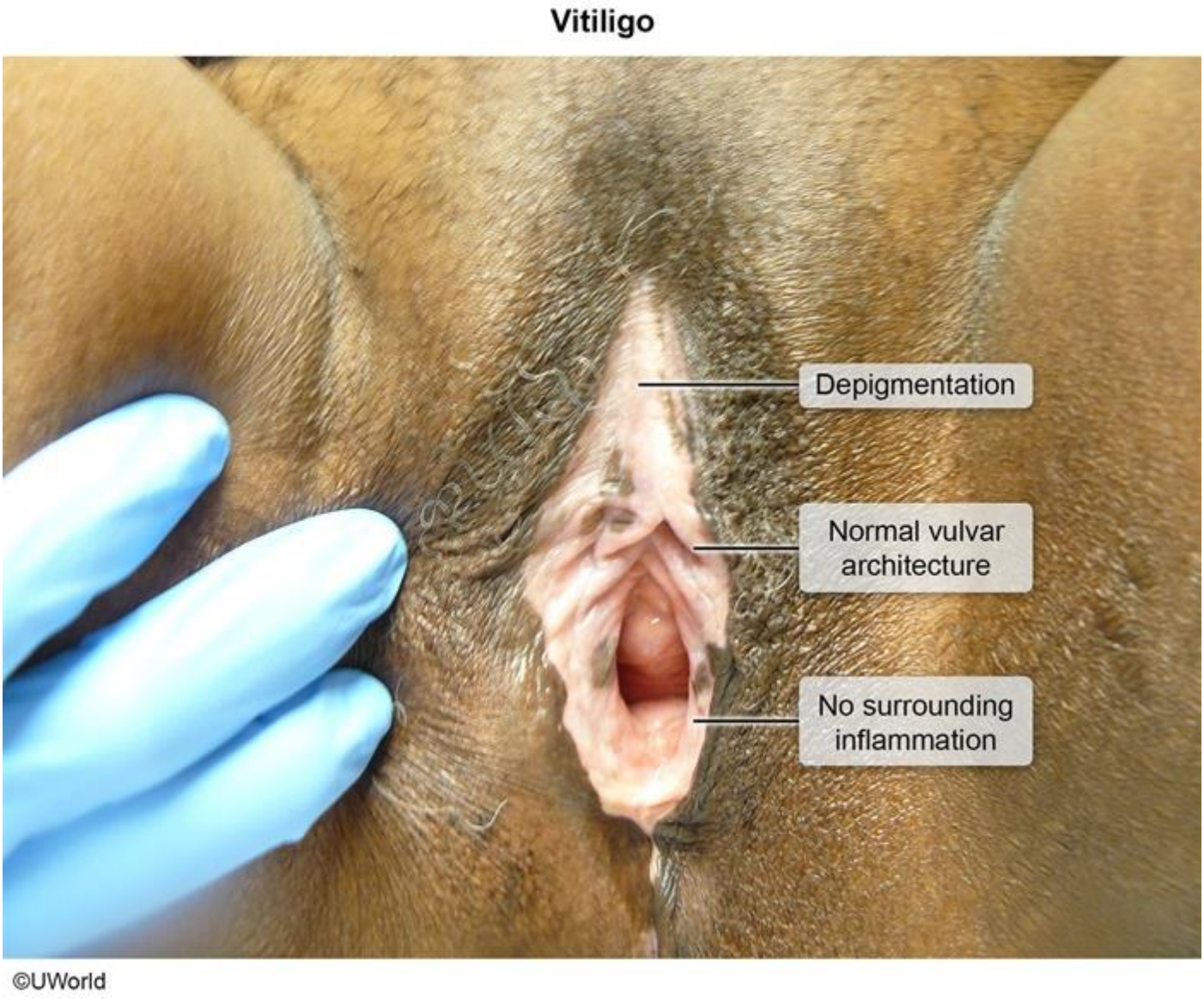
Drug-induced acne	
	
Common triggers	• **Glucocorticoids**, androgens • Immunomodulators (eg, azathioprine, EGFR inhibitors) • Anticonvulsants (eg, phenytoin), antipsychotics • Antituberculous drugs (eg, isoniazid)
Presentation	• **Monomorphic papules** or pustules • Lack of comedones, cysts & nodules • Location & age of onset may be atypical for acne
Management	• Discontinue offending medication • Standard acne therapy unlikely to be effective

EGFR = epidermal growth factor receptor.

Drug-induced acne

Acute urticaria

Etiologies	• Medications, insect stings • Infections, rheumatologic diseases • Many cases are idiopathic
Pathophysiology	• Mast cells release histamine → dermal edema
Clinical features	• Pruritic, erythematous plaques (ie, wheals) • Each wheal lasts <24 hr, urticaria duration <6 weeks • Diffuse or localized
Evaluation	• No workup for 1st episode • Allergy testing if specific trigger (eg, food) is suspected • Laboratory testing/biopsy if systemic symptoms are present
Treatment & prognosis	• 2nd-generation H_1 antihistamines • Add H_2 antihistamines or systemic corticosteroids if severe • Two-thirds of cases self-resolve



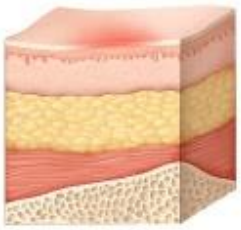
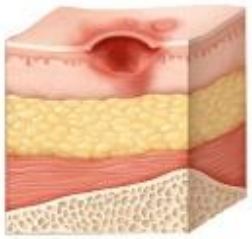
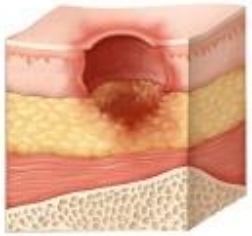
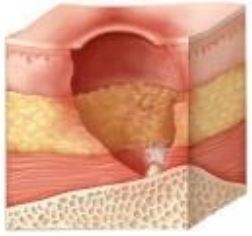
Vitiligo

Normal structure and function of skin

PORPHYRIA CUTANEA TARDA

Porphyria cutanea tarda

Clinical presentation	• Blisters, bullae, scarring, hypopigmentation/hyperpigmentation on sun exposed skin (eg, back of hands, forearms, face) • Scarring & calcification similar to scleroderma
Associated conditions	• Hepatitis C • HIV • Excessive alcohol consumption • Estrogen use • Smoking
Diagnostic testing	• Mildly elevated liver enzymes & iron overload • Elevated plasma or urine porphyrin levels

Stage	Clinical features	Illustration
1	• Intact skin • Non-blanchable with localized redness	
2	• Shallow, open ulcer • Red-pink wound with no sloughing • Possible intact or ruptured blister	
3	• Full-thickness skin loss with possible visible subcutaneous fat • No exposed bone, tendon, or muscles	
4	• Full-thickness skin loss • Exposed bone, tendon, or muscle	
Unstageable	• Full-thickness skin loss • Ulcer base covered by slough and/or eschar that needs removal to stage	

Pressure induced injury

SUNBURN

Sun-protective measures

Exposure	• Sun avoidance, especially age <6 months • Reduce exposure 10:00 AM–4:00 PM
Sunscreen	• SPF ≥30 • Apply 15-30 minutes prior to sun exposure • Reapply every 2 hours & after swimming
Clothing	• Long sleeves, broad-brim hats • Tight weave, dark color

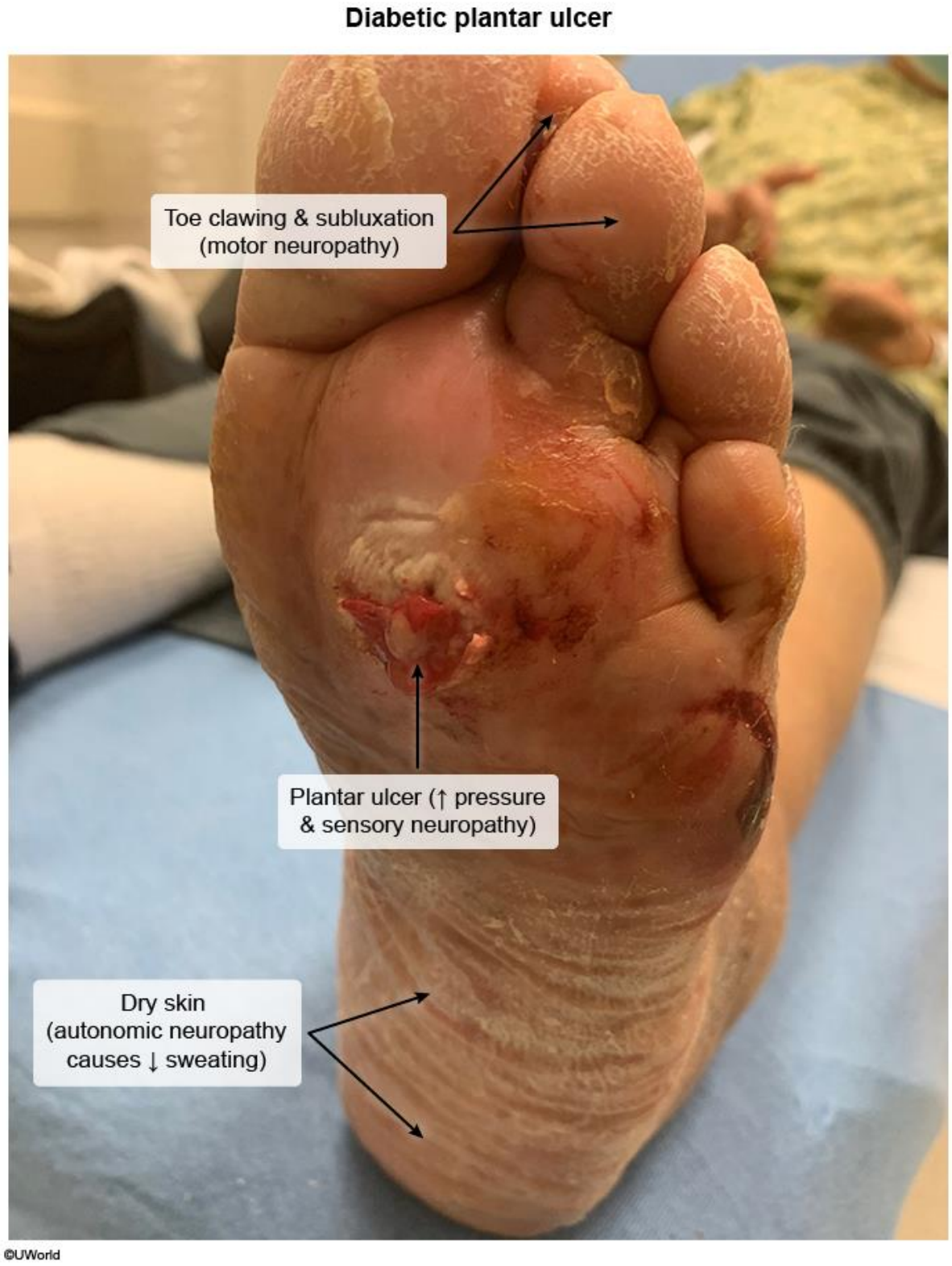
SPF = sun protection factor.

VITILIGO

Vitiligo

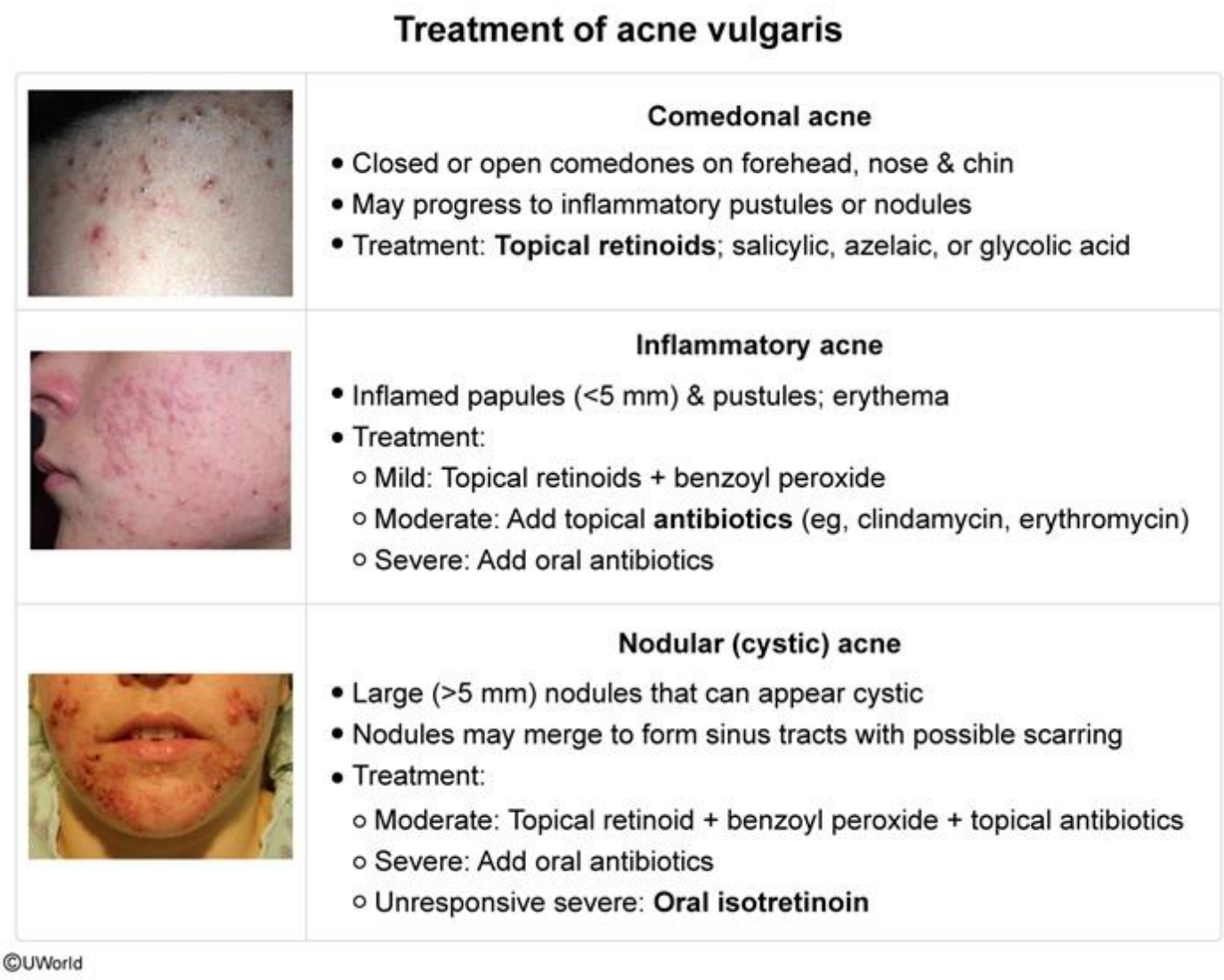
Clinical manifestations	• Depigmented macules on acral areas & extensor surfaces; face commonly affected • Lesions may be symmetrical, dermatomal, or unilateral
Clinical course	• Most cases progress gradually • Repigmentation is spontaneous in 10%-20% of cases • Increased incidence of other autoimmune disorders (eg, lupus, thyroid disease, pernicious anemia, Addison disease)
Treatment	• Limited disease: topical corticosteroids • Extensive/unresponsive disease: oral corticosteroids, topical calcineurin inhibitors, PUVA

PUVA = psoralen + ultraviolet A light.



Diabetic plantar ulcer

Treatment of acne vulgaris



Treatment of acne vulgaris

DIAPER DERMATITIS

Perianal dermatoses

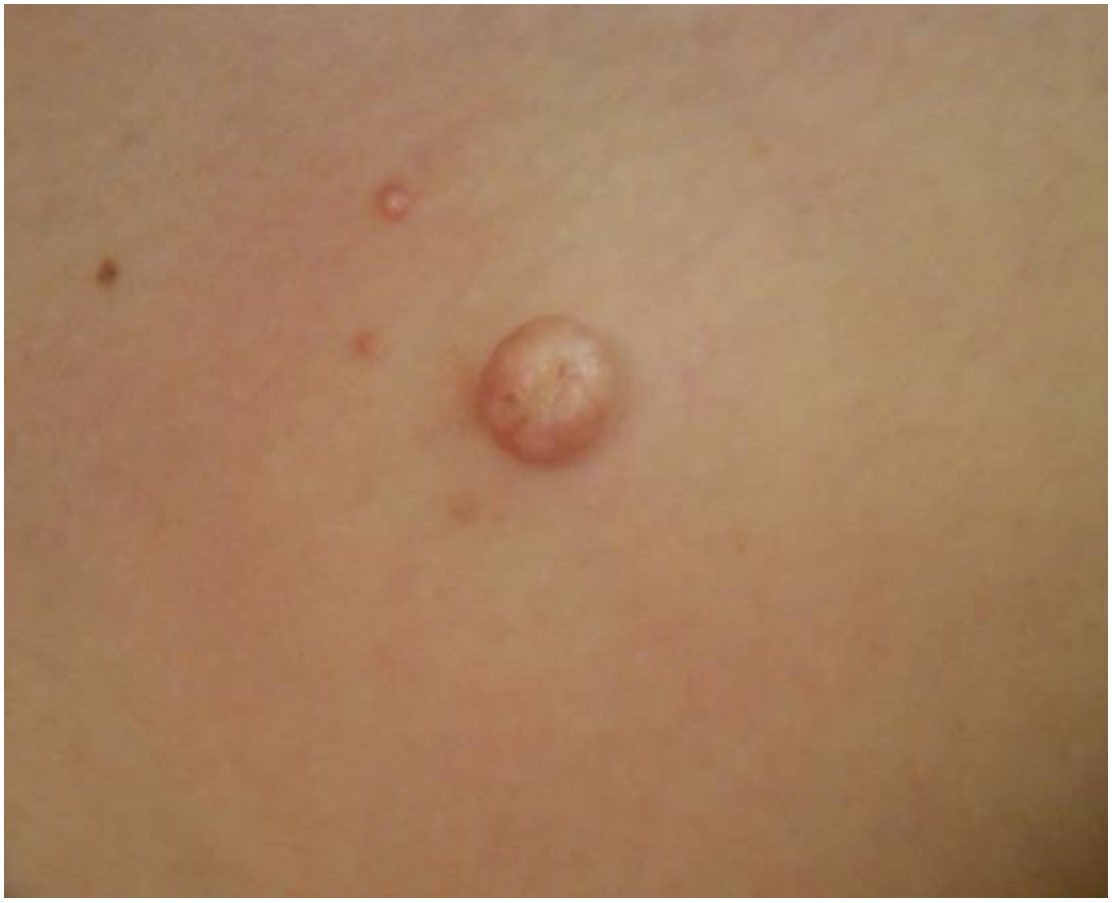
Diagnosis	Irritant contact diaper dermatitis	*Candida* diaper dermatitis	Perianal *Streptococcus*
Epidemiology	• Most common diaper rash in infants	• Second most common diaper rash in infants	• Infants through school-aged children
Examination	• Erythematous papules, plaques • Spares skinfolds	• Beefy-red, confluent plaques • Involves skinfolds • Satellite lesions	• Bright, sharply demarcated erythema of perianal/perineal area
Treatment	• Topical barrier (eg, petrolatum, zinc oxide)	• Topical antifungal (eg, nystatin)	• Oral antibiotics (eg, amoxicillin)

MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum

	Children	Adults
Typical location	• Trunk • Intertriginous areas (eg, axillae) • Face (including eyelids)	• Lower abdomen, genitals, upper thighs
Evaluation	• Clinical diagnosis (ie, firm, domed, papule with central umbilication) • No further evaluation	• Clinical diagnosis • Genital lesions: STI testing • Extensive lesions: HIV testing
Treatment	• Reassurance	• Cryotherapy • Curettage • Topical therapy (eg, cantharidin, podophyllotoxin)

STI = sexually transmitted infection.



Molluscum contagiosum

SCABIES

Scabies

Pathogenesis	• *Sarcoptes scabiei* mite infestation • Spread by direct person-to-person contact
Clinical features	• Extremely pruritic pathognomonic burrows & small, erythematous papules • Rash located on interdigital web spaces, flexor wrists, extensor elbows, axillae, umbilicus & genitalia
Treatment	• Topical 5% permethrin OR • Oral ivermectin

Impetigo

Type	Nonbullous	Bullous
Microbiology	• *Staphylococcus aureus* • Group A *Streptococcus* (*S pyogenes*)	• *S aureus*–producing exfoliative toxin A
Clinical features	• Papules & pustules with honey-crusted lesions	• Enlarging, flaccid bullae with yellow fluid • Ruptured lesions with a collarette of scale at periphery
Treatment	• Limited: topical antibiotics (eg, mupirocin) • Extensive: oral antibiotics (eg, cephalexin)	• Oral antibiotics (eg, cephalexin)

Common skin infections

Infection	Organism	Manifestations
Erysipelas	• *Streptococcus pyogenes*	• Superficial dermis & lymphatics • Raised, sharply demarcated edges • Rapid spread & onset • Fever early in course
Cellulitis (nonpurulent)	• *S pyogenes* • MSSA	• Deep dermis & subcutaneous fat • Flat edges with poor demarcation • Indolent (over days) • Localized (fever later in course)
Cellulitis (purulent)	• MSSA • MRSA	• Purulent drainage • Folliculitis: infected hair follicle • Furuncles: folliculitis → dermis → abscess • Carbuncle: multiple furuncles

MRSA = methicillin-resistant *Staphylococcus aureus;* **MSSA** = methicillin-sensitive *S aureus*.

Staphylococcal scalded skin syndrome

Pathogenesis	• *Staphylococcus aureus* exfoliative toxin
Clinical features	• Fever, irritability • Generalized erythema, blisters • Epidermal shedding (Nikolsky sign)
Management	• Antistaphylococcal antibiotic (eg, nafcillin, vancomycin) • Wound care

TINEA

Tinea pedis

Etiology	• *Trichophyton* species (most common)
Clinical features	• **Acute:** pruritus, burning pain, erythematous vesicles/bullae • **Chronic:** pruritus, erythema, interdigital scales/fissures/erosions with extension onto the sole, side, or dorsum of the foot
Treatment	• Topical antifungal therapy • Keep feet dry & dispose of old footwear

Tinea pedis

Etiology	• Dermatophyte fungi (*Trichophyton* species most common)
Risk factors	• Barefoot walking in public areas (eg, swimming pools, athletic facilities)
Clinical features	• **Interdigital type:** pruritus, erythema, erosions between toes • **Moccasin type:** scales/fissures; extension onto the sole, side, or dorsum of foot • **Vesiculobullous type:** painful bullae, erythema (lateral midfoot) • Complications: secondary infection, recurrence
Diagnosis	• Clinical presentation • **Potassium hydroxide** microscopy of skin scraping
Treatment	• **First line:** topical antifungals (eg, miconazole, tolnaftate) • **Second line:** oral antifungals (eg, fluconazole) • Keep feet dry & dispose of old footwear

Tinea corporis (ringworm)

Risk factors	• Athletes who have skin-to-skin contact • Humid environment • Contact with infected animals (eg, rodents)
Presentation	• Scaly, erythematous, pruritic patch with centrifugal spread • Subsequent central clearing with raised, annular border
Treatment	• First-line/localized: topical antifungals (eg, clotrimazole, terbinafine) • Second-line/extensive: oral antifungals (eg, terbinafine, griseofulvin)

Risk factors for dermatophyte infection

Environmental exposures	• Warm, humid environments • Direct contact with infected person, fomites, or public showers • Contact with animals (eg, kittens)
Patient factors	• Concurrent dermatophyte infection (autoinoculation) • Occlusive clothing • Obesity • Peripheral artery disease
Immune deficiency	• Diabetes mellitus • HIV infection • Systemic glucocorticoid therapy

TINEA VERSICOLOR

Tinea versicolor (pityriasis versicolor)

Pathogenesis	• *Malassezia globosa* skin flora grows in exposure to hot & humid weather
Clinical features	• Hypopigmented, hyperpigmented, or mildly erythematous lesions (face in children, trunk & upper extremities in adolescents & adults) • ± Fine scale • ± Pruritus
Diagnosis	• KOH preparation shows hyphae & yeast cells in a "spaghetti & meatballs" pattern
Treatment	• Topical ketoconazole, terbinafine, or selenium sulfide

KOH = potassium hydroxide.

Skin tumors and tumor-like lesions

Actinic keratoses

Actinic keratosis

Clinical features	• Erythematous, scaly papules; rough plaques • Sun-exposed areas
Diagnosis	• Clinical appearance • Biopsy if features of possible SCC (eg, size ≥1 cm, rapid growth, ulceration, tenderness)
Prognosis	• Chronic/persistent • Progression to SCC • Spontaneous resolution
Treatment	• Isolated lesions: cryotherapy • Diffuse lesions: topical fluorouracil, imiquimod, tirbanibulin

SCC = squamous cell carcinoma.

Hemangioma

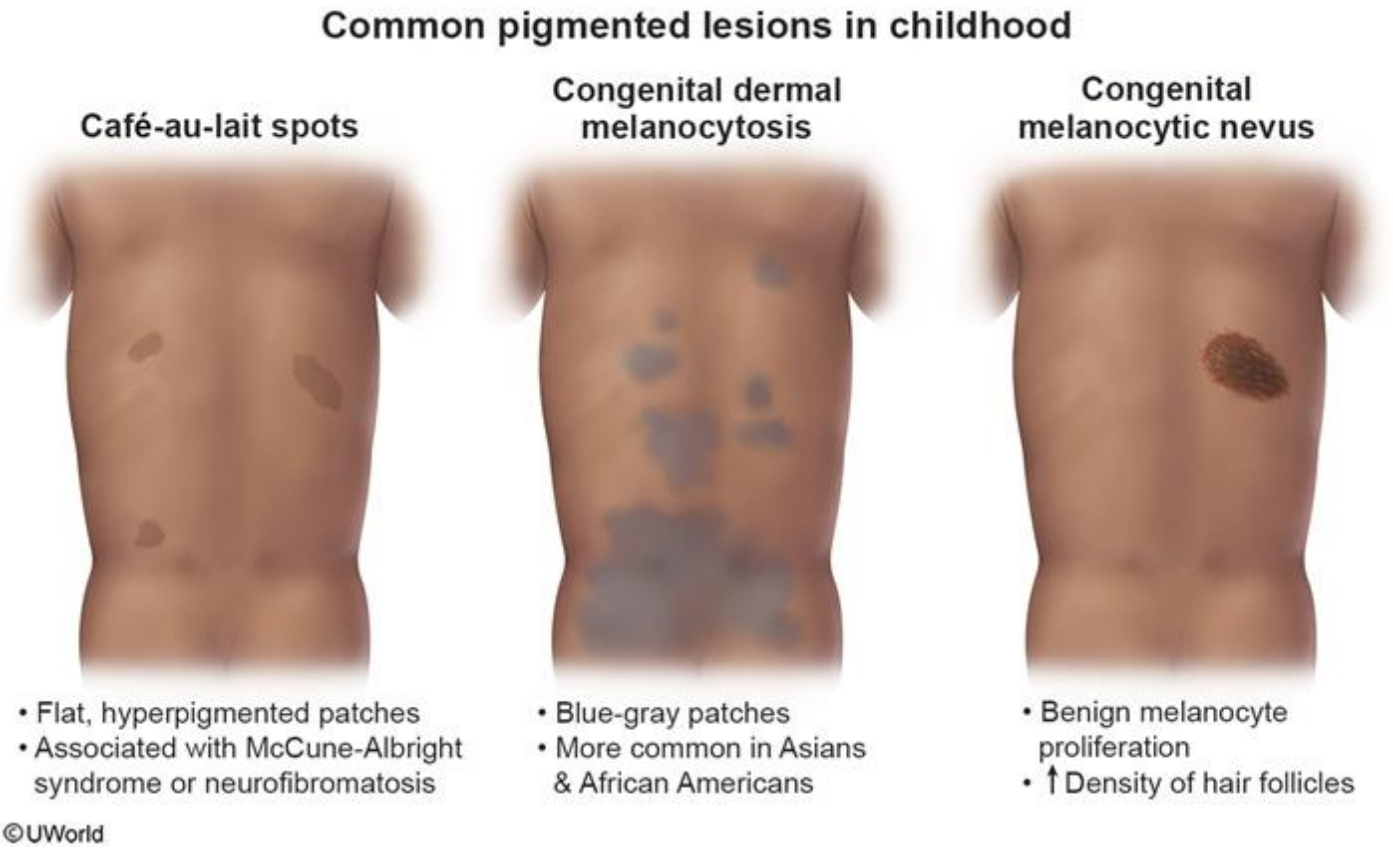
Infantile hemangioma

Natural history	• Appears days to weeks after birth • Proliferation (age 0-6 months): growth of bright red, soft, raised plaque • Involution (age >6 months): deep red/violet lesion that regresses in size
Evaluation	• Clinical diagnosis • Special considerations: – ≥5 cutaneous lesions → liver ultrasound – Facial/segmental → echocardiography & MRI of the head (ie, PHACE) – Cervicofacial (beard distribution) → laryngoscopy – Lumbosacral → spinal ultrasound
Management	• Observation for most lesions • Beta-blocker therapy (eg, propranolol) for high-risk features: – Large, facial, segmental, &/or rapidly growing (ulceration/scarring) – Periorbital (visual impairment) – Hepatic (high-output heart failure) – Subglottic (airway obstruction)

PHACE = posterior fossa anomalies, **h**emangioma, **a**rterial anomalies, **c**ardiac anomalies, **e**ye anomalies.

Keratoacanthoma

Clinical features	• Rapidly growing nodule with ulceration & keratin plug • Often shows spontaneous regression/resolution
Clinical significance	• May resemble or progress to squamous cell carcinoma
Management	• Excisional biopsy with complete removal of lesion



Common pigmented lesions in childhood

Clinical features of melanoma (ABCDE)

- **A**symmetry: when bisected, the 2 sides are not identical
- **B**order irregularities: uneven edges, pigment fading off
- **C**olor variegation: variable mixtures of brown, tan, black & red
- **D**iameter: ≥6 mm
- **E**volving: lesion changing in size, shape, or color; new lesion

Visual assessment of melanoma

ABCDE criteria (≥1-2 is suspicious)	• **A**symmetry • **B**order irregularity • **C**olor variation (within lesion or compared to other lesions) • **D**iameter ≥6 mm • **E**volving appearance over time
7-point checklist (≥1 major or ≥3 minor is suspicious)	• **Major criteria:** change in size, shape, or color • **Minor criteria:** size ≥7 mm, local inflammation, crusting/bleeding, sensory symptoms
Ugly duckling sign	• One lesion is significantly different from others on the patient

NONMELANOMA SKIN CANCER

Basal cell carcinoma

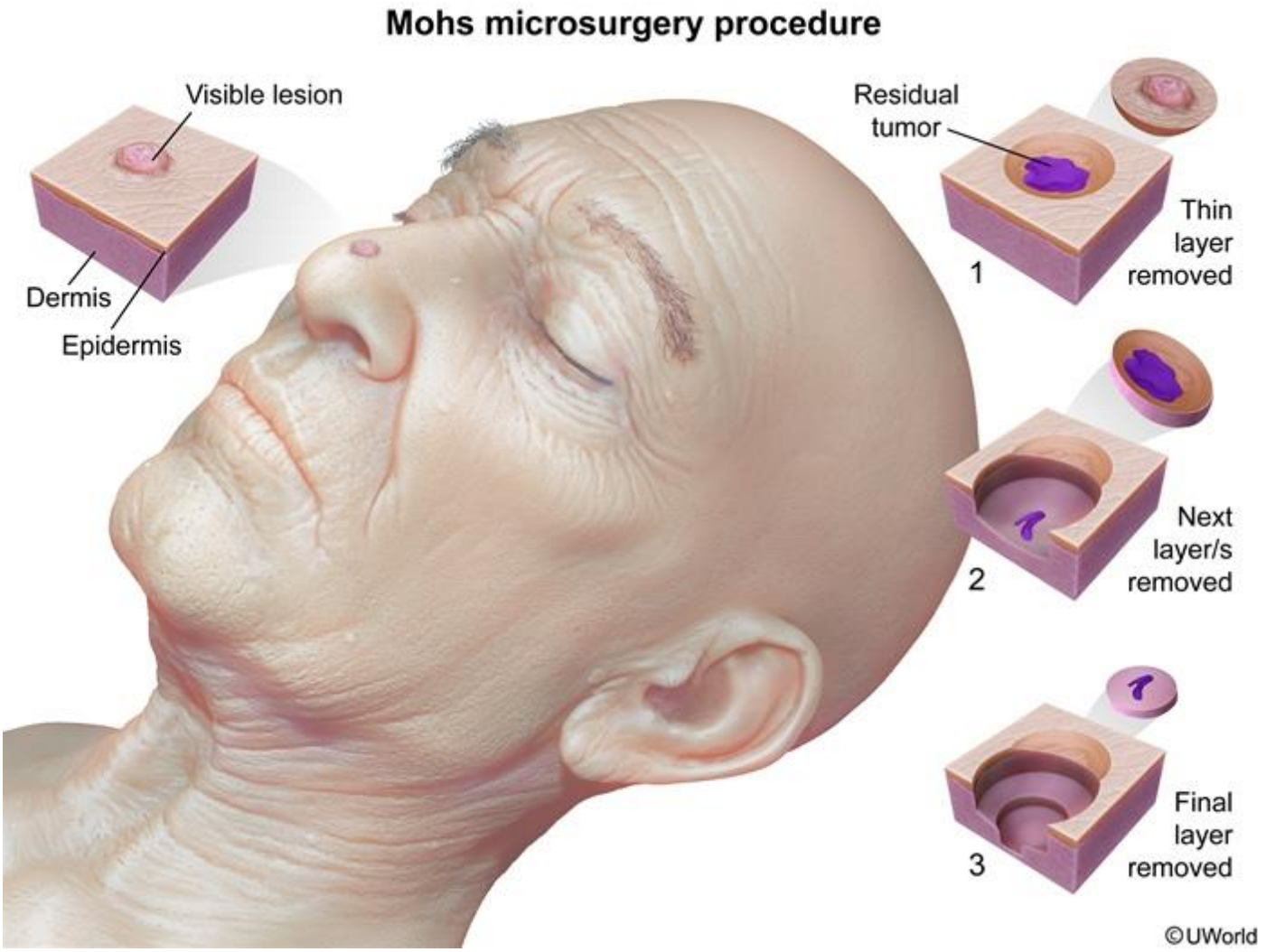
Risk factors	• Sun/ultraviolet light • Fair skin • Ionizing radiation
Clinical features	• Skin-colored, pearly nodule ± rolled borders • Telangiectatic vessels • ± Central ulceration, local invasion
Diagnosis	• Shave, punch, or excisional biopsy
Treatment	• First-line: – Surgical excision with 4-mm margins – Mohs micrographic surgery (face/high-risk tumors) • Second-line: – Topical fluorouracil, topical imiquimod, C&E (low-risk tumors only)

C&E = curettage & electrodessication.

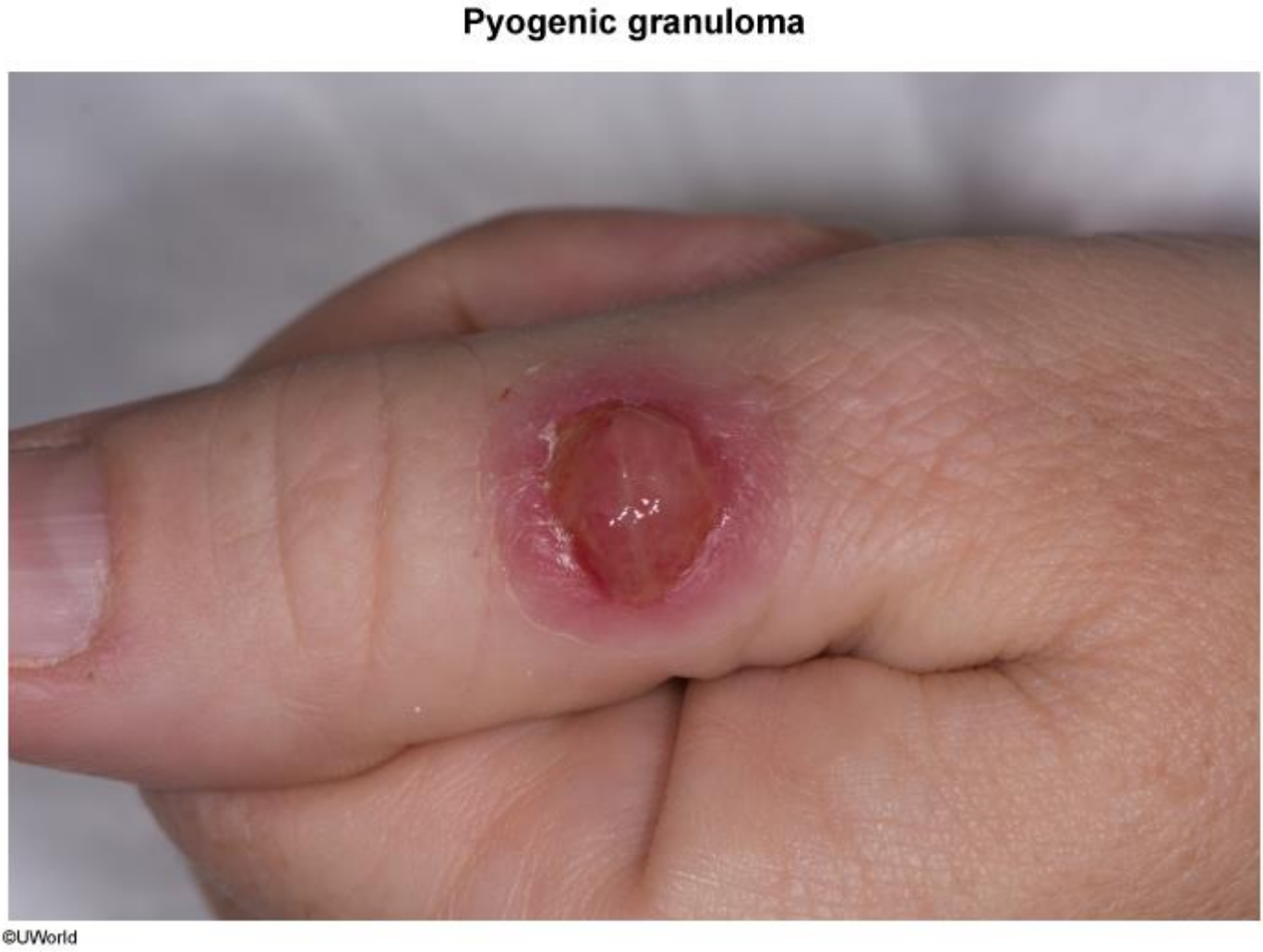
Squamous cell carcinoma of skin

Risk factors	• Sun/ultraviolet, ionizing radiation exposure • Chronic scars/wounds/burn injuries • Immunosuppression	
Clinical features	• Scaly plaques/nodules • ± Ulceration • SCC in situ: well-demarcated patches/plaques	
Diagnosis	• Punch, shave, or excisional biopsy • Dysplastic/anaplastic keratinocytes	
Treatment	Invasive SCC • Excision with 4- to 6-mm margins • Mohs micrographic surgery	SCC in situ • Excision with 4- to 6-mm margins • C&E • Cryotherapy • Topical 5-FU, imiquimod

5-FU = 5-fluorouracil; **C&E** = curettage & electrodesiccation; **SCC** = squamous cell carcinoma.



Mohs microsurgery procedure



Pyogenic granuloma

SEBORRHEIC DERMATITIS

Seborrheic dermatitis in infants

Clinical features	• Scalp (cradle cap), face (eg, eyebrows), retroauricular areas – Yellow, greasy scales • Intertriginous regions (eg, neck folds, axillae, diaper area), umbilicus – Glistening, confluent erythema • ± Mild pruritus; no systemic symptoms
Treatment	• First-line: emollients, nonmedicated shampoos • Second-line: topical antifungals or low-potency corticosteroids

Tuberous sclerosis complex

Pathophysiology	• Mutation (inherited or de novo) in *TSC1* or *TSC2* gene • Autosomal dominant
Clinical features	• Dermatologic – Ash-leaf spots – Angiofibromas of the malar region – Shagreen patches • Neurologic – CNS lesions (eg, subependymal tumors) – Epilepsy (eg, infantile spasms) – Intellectual disability – Autism & behavioral disorders (eg, hyperactivity) • Cardiovascular: rhabdomyomas • Renal: angiomyolipomas
Surveillance	• Tumor screening – Regular skin & eye examinations – Serial MRI of the brain & kidney – Baseline echocardiography & serial ECG • Baseline electroencephalography • Neuropsychiatric screening