

Cellulitis and skin abscess: Epidemiology, microbiology, clinical manifestations, and diagnosis

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INTRODUCTION

Cellulitis and abscess are among the most common skin and soft tissue infections [1-3]. Cellulitis (which includes erysipelas) manifests as an area of skin erythema, edema, and warmth; it develops as a result of bacterial entry via breaches in the skin barrier [4]. A skin abscess is a collection of pus within the dermis or subcutaneous space. Misdiagnosis of these entities is common [5], and alternative diagnoses should be considered ([figure 1](#)). (See '[Differential diagnosis](#)' below.)

The epidemiology, microbiology, clinical manifestations, and diagnosis of cellulitis and skin abscess are reviewed here. Issues related to treatment of cellulitis and skin abscess are discussed separately. (See "[Acute cellulitis and erysipelas in adults: Treatment](#)".)

Specific epidemiologic factors (such as diabetes mellitus, animal bites, and water exposure) associated with skin and soft tissue infections are discussed separately. (See "[Clinical manifestations, diagnosis, and management of diabetic infections of the lower extremities](#)" and "[Animal bites \(dogs, cats, and other mammals\): Evaluation and management](#)" and "[Soft tissue infections following water exposure](#)".)

Infection involving the gluteal area and perineum are discussed separately. (See "[Pilonidal disease](#)" and "[Perianal and perirectal abscess](#)".)

EPIDEMIOLOGY

Cellulitis is observed most frequently among middle-aged and older adults. Erysipelas occurs in young children and older adults [6,7]. The incidence of cellulitis is about 200 cases per 100,000 patient-years [8] and, in nontropical regions, has a seasonal predilection for warmer months [8-12].

Skin abscess may occur in healthy individuals with no predisposing conditions. The burden of skin abscess has varied. During the 1990s, the number of cases in the United States increased; this was attributed to the increasing prevalence of community-acquired methicillin-resistant *Staphylococcus aureus* strains (USA300 clone). In the 2000s, however, the incidence of skin abscess has plateaued [13-15].

Predisposing factors associated with risk of cellulitis and/or skin abscess include [16-25]:

- Skin barrier disruption due to trauma (such as abrasion, penetrating wound, pressure ulcer, venous leg ulcer, insect bite, injection drug use)
- Skin inflammation (such as eczema, radiation therapy, psoriasis)
- Edema due to impaired lymphatic drainage
- Edema due to venous insufficiency
- Obesity
- Immunosuppression (such as diabetes or HIV infection)
- Skin breaks between the toes ("toe web intertrigo"); these may be clinically inapparent
- Pre-existing skin infection (such as tinea pedis, impetigo, varicella)
- Prior saphenous vein harvesting for coronary artery bypass graft surgery

Lymphatic compromise may occur following surgical procedures (such as saphenous venectomy or lymph node dissection) or in the setting of congenital abnormalities. In a retrospective analysis of more than 165,000 hospital admissions for lymphedema in the United States between 2012 and 2017, most cases were associated with cellulitis (92 percent) [26]. (See "[Early noncardiac complications of coronary artery bypass graft surgery](#)", section on '[Post-venectomy cellulitis](#)' and "[Cellulitis following pelvic lymph node dissection](#)".)

An additional risk factor associated with purulent skin and soft tissue infections is close contact with others with methicillin-resistant *S. aureus* infection or carriage. (See "[Methicillin-resistant Staphylococcus aureus \(MRSA\) in adults: Epidemiology](#)" and "[Methicillin-resistant Staphylococcus aureus infections in children: Epidemiology and clinical spectrum](#)", section on '[Epidemiology and risk factors](#)'.)

MICROBIOLOGY

Cellulitis and erysipelas — The most common causes of cellulitis are beta-hemolytic streptococci (groups A, B, C, G, and F) with group A *Streptococcus* or *Streptococcus pyogenes* most commonly identified in some series; *S. aureus* (including methicillin-resistant strains) is a notable but less common cause [4,21,27-33]. Gram-negative aerobic bacilli are identified in a minority of cases.

Most erysipelas cases are caused by beta-hemolytic streptococci [7,27,34,35]. One study of nonpurulent cellulitis including 179 patients found that beta-hemolytic streptococci accounted for 73 percent of cases (diagnosed by positive blood culture results or serologic testing for anti-streptolysin-O and anti-DNase-B antibodies) [32]. No etiology was identified in 27 percent of cases, but the overall clinical response rate to beta-lactam therapy was 96 percent.

Less common causes of cellulitis include *Haemophilus influenzae* type b (buccal cellulitis), clostridia and non-spore-forming anaerobes (crepitant cellulitis), *Streptococcus pneumoniae*, and *Neisseria meningitidis* [36-42]. In immunocompromised patients, the spectrum of potential pathogens is much broader, and infectious disease consultation is warranted.

Pathogens implicated in special clinical circumstances discussed in detail separately include (table 1):

- *Pasteurella multocida* and *Capnocytophaga canimorsus* (see "Animal bites (dogs, cats, and other mammals): Evaluation and management")
- *Aeromonas hydrophila* and *Vibrio vulnificus* (see "Soft tissue infections following water exposure")
- *Pseudomonas aeruginosa* (see "Fever and rash in immunocompromised patients without HIV infection" and "Clinical manifestations, diagnosis, and management of diabetic infections of the lower extremities" and "Pseudomonas aeruginosa skin and soft tissue infections")
- Group B *Streptococcus* (see "Group B streptococcal infection in neonates and young infants", section on 'Other focal infection' and "Cellulitis following pelvic lymph node dissection", section on 'Timing of onset')
- *Clostridium* species (see "Clostridial myonecrosis")
- *Erysipelothrix rhusiopathiae* (see "Erysipelothrix infection")
- *S. pneumoniae* (see "Orbital cellulitis")
- *Cryptococcus neoformans* (see "Fever and rash in immunocompromised patients without HIV infection")

- *Streptococcus iniae* (see "Fever and rash in the immunocompetent patient")
- *Helicobacter cinaedi* (see "Fever and rash in patients with HIV")
- *Mycobacterium abscessus* (see "Rapidly growing mycobacterial infections: Mycobacteria abscessus, chelonae, and fortuitum")

Skin abscess — The most common cause of skin abscess is *S. aureus* (either methicillin-susceptible or methicillin-resistant *S. aureus* [MRSA]), which occurs in up to 75 percent of cases. Risk factors are summarized in the table ([table 2](#)); many patients with MRSA infection have no known risk factors [[3,43-48](#)].

A skin abscess can be caused by more than one pathogen [[44,49-51](#)]; isolation of multiple organisms (including *S. aureus* together with *S. pyogenes* and gram-negative bacilli with anaerobes) is more common in patients with skin abscess involving the perioral, perirectal, or vulvovaginal areas [[44](#)]. Organisms of oral origin, including anaerobes, are seen most frequently among intravenous drug users [[44](#)].

Unusual causes of skin abscess include nontuberculous mycobacteria, blastomycosis, nocardiosis, and cryptococcosis (see related topics).

Most abscesses are due to infection. However, sterile abscesses can occur in the setting of injected irritants. Examples include injected drugs (particularly oil-based ones) that may not be fully absorbed and so remain at the site of injection, causing local irritation. Sterile abscesses can turn into hard, solid lesions as they scar.

CLINICAL MANIFESTATIONS

Patients with skin and soft tissue infection may present with cellulitis, abscess, or both [[1-3,43](#)].

Cellulitis and erysipelas — Cellulitis and erysipelas manifest as areas of skin erythema, edema, and warmth; they develop as a result of bacterial entry via breaches in the skin barrier [[4](#)]. Petechiae and/or hemorrhage can be seen in erythematous skin, and superficial bullae can occur. Fever and other systemic manifestations of infection may also be present. Cellulitis and erysipelas are nearly always unilateral, and the lower extremities are the most common site of involvement; bilateral involvement should prompt consideration of alternative diagnoses ([figure 1](#) and [picture 1](#) and [picture 2](#) and [picture 3](#) and [picture 4](#)) [[4,6,52](#)]. (See 'Differential diagnosis' below.)

Cellulitis involves the deeper dermis and subcutaneous fat; erysipelas involves the upper dermis and superficial lymphatics ([figure 2](#)). Cellulitis may present with or without purulence;

erysipelas is nonpurulent [1-3]. Patients with cellulitis tend to have a more indolent course with development of localized symptoms over a few days.

Patients with erysipelas generally have acute onset of symptoms with systemic manifestations, including fever, chills, severe malaise, and headache; these can precede onset of local inflammatory signs and symptoms by minutes to hours. In erysipelas, there is clear demarcation between involved and uninvolvled tissue [53]. There may be a raised, advancing border or erythema with central clearing. Classic descriptions of erysipelas note "butterfly" involvement of the face ([picture 5](#)). Involvement of the ear (Milian's ear sign) is a distinguishing feature for erysipelas since this region does not contain deeper dermis tissue.

Additional manifestations of cellulitis and erysipelas include lymphangitis and enlargement of regional lymph nodes. Edema surrounding the hair follicles may lead to dimpling in the skin, creating an appearance reminiscent of an orange peel texture ("peau d'orange"). Vesicles, bullae, and ecchymoses or petechiae may be observed ([picture 6](#) and [figure 1](#)). Cutaneous hemorrhage can occur in the setting of significant inflammation in the skin. Crepitant and gangrenous cellulitis are unusual manifestations of cellulitis due to clostridia and other anaerobes. Severe manifestations with systemic toxicity should prompt investigation for additional underlying sources of infection. (See "[Invasive group A streptococcal infection and toxic shock syndrome: Epidemiology, clinical manifestations, and diagnosis](#)" and "[Staphylococcal toxic shock syndrome](#)".)

The interdigital toe spaces should be examined for fissuring or maceration; minimizing these conditions may reduce the likelihood of recurrent lower-extremity cellulitis.

Bilateral lower leg cellulitis is very uncommon. Such a presentation with bilateral erythema, swelling, and tenderness is more likely due to alternative diagnosis. (See '[Cellulitis and erysipelas](#)' below.)

Other forms of cellulitis include orbital cellulitis, abdominal wall cellulitis (in persons with obesity), buccal cellulitis (due to *S. pneumoniae* and, prior to the conjugate vaccine era, *H. influenzae* type b) and perianal cellulitis (due to group A beta-hemolytic *Streptococcus*) [54,55]. Rarely, infections involving the medial third of the face (ie, the areas around the eyes and nose) can be complicated by septic cavernous thrombosis, since the veins in this region are valveless ([figure 3](#)). (See "[Orbital cellulitis](#)" and "[Septic dural sinus thrombosis](#)".)

Laboratory findings are nonspecific and may include leukocytosis and elevated inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) [4,56].

Skin abscess — Skin abscess is a collection of pus within the dermis or subcutaneous space ([picture 7](#) and [figure 2](#)). It manifests as a painful, fluctuant, erythematous nodule, with or without surrounding cellulitis [44]. Spontaneous drainage of purulent material may occur. Regional adenopathy may be observed. Fever, chills, and systemic toxicity are unusual.

A skin abscess may develop via deep infection of a hair follicle (known as a furuncle or boil), which reflects extension of purulent material through the dermis into the subcutaneous tissue. Multiple furuncles can coalesce to form carbuncles ([picture 8](#)), which may be associated with systemic symptoms. Common areas of involvement include the back of the neck, face, axillae, and buttocks.

Complications — Complications of cellulitis and abscess include bacteremia, endocarditis, septic arthritis or osteomyelitis, metastatic infection, sepsis, and toxic shock syndrome [4].

DIAGNOSIS

The diagnosis of cellulitis, erysipelas, and skin abscess is usually based upon clinical manifestations. Cellulitis and erysipelas manifest as areas of skin erythema, edema, and warmth ([figure 1](#)). Erysipelas lesions are raised above the level of surrounding skin with clear demarcation between involved and unininvolved tissue. A skin abscess manifests as a painful, fluctuant, erythematous nodule, with or without surrounding cellulitis ([picture 7](#)).

Laboratory testing is not required for patients with uncomplicated infection in the absence of comorbidities or complications.

Patients with drainable abscess should undergo incision and drainage [57,58]. Routine culture of debrided material is not necessary in healthy patients who do not receive antibiotics.

Cultures of debrided material and blood cultures (prior to addition of antibiotic therapy) are warranted in the following circumstances [59,60]:

- Severe local infection (eg, extensive cellulitis)
- Systemic signs of infection (eg, fever)
- History of recurrent or multiple abscesses
- Failure of initial antibiotic therapy
- Extremes of age (young infants or older adults)
- Presence of underlying comorbidities (lymphedema, malignancy, neutropenia, immunodeficiency, splenectomy, diabetes)
- Special exposures (animal bite, water-associated injury)

- Presence of indication for prophylaxis against infective endocarditis
- Community patterns of *S. aureus* susceptibility are unknown or rapidly changing

Positive blood cultures can impact antibiotic selection or prompt evaluation for additional infections (eg, osteomyelitis, endocarditis). Overall, blood cultures are positive in less than 10 percent of cellulitis cases, but certain populations may have higher rates of culture positivity [61-63]. For example, one observational study found that positive blood cultures occurred in 25 percent of older (≥ 65 years) individuals who were hospitalized for cellulitis, erysipelas, or skin abscess [64]. A skin biopsy may be warranted if the diagnosis is uncertain; cultures of skin biopsy specimens yield a pathogen in 20 to 30 percent of cases [65-68]. Cultures of swabs from intact skin are not helpful and should not be performed [2,3].

Ultrasonographic examination can be useful to determine whether a skin abscess is present (see "[Techniques for skin abscess drainage](#)", section on '[Bedside ultrasonography](#)') and for distinguishing cellulitis from osteomyelitis (via magnetic resonance imaging) [69]. Radiographic evaluation may be warranted in patients with underlying immunosuppression, diabetes mellitus, venous insufficiency, or lymphedema and in patients with persistent systemic symptoms. Radiographic examination cannot reliably distinguish cellulitis from necrotizing fasciitis or gas gangrene; if there is clinical suspicion for these entities, radiographic imaging should not delay surgical consultation or intervention [70,71]. (See "[Necrotizing soft tissue infections](#)" and "[Clostridial myonecrosis](#)".)

In patients with recurrent cellulitis, serologic testing for beta-hemolytic streptococci may be a useful diagnostic tool. Assays include the anti-streptolysin-O (ASO) reaction, the anti-deoxyribonuclease B test (anti-DNAse B), the anti-hyaluronidase test (AHT), or the Streptozyme antibody assay [28]. Anti-DNAse B and AHT responses are more reliable than the ASO response following group A streptococcal skin infections. (See "[Acute cellulitis and erysipelas in adults: Treatment](#)", section on '[Recurrent infection](#)').

DIFFERENTIAL DIAGNOSIS

Cellulitis and erysipelas — Cellulitis is often confused with other infections or a variety of noninfectious illnesses ([table 3](#)) [72,73].

Rapidly progressive erythema with signs of systemic toxicity should prompt consideration of severe infection, including:

- Necrotizing fasciitis – Necrotizing fasciitis is a deep infection that results in progressive destruction of the muscle fascia. The affected area may be erythematous, swollen, warm,

and exquisitely tender. Pain out of proportion to exam findings may be observed. The diagnosis is established surgically with visualization of fascial planes. (See "[Necrotizing soft tissue infections](#)".)

- Toxic shock syndrome – Toxic shock syndrome typically presents with pain that precedes physical findings. Clinical signs of soft tissue infection consist of local swelling and erythema followed by ecchymoses and sloughing of skin. Fever is common. Patients may be normotensive on presentation but subsequently become hypotensive. (See "[Invasive group A streptococcal infection and toxic shock syndrome: Epidemiology, clinical manifestations, and diagnosis](#)".)
- Clostridial myonecrosis (gas gangrene) – Gas gangrene should be suspected in the setting of fever and severe pain in an extremity, particularly in the setting of recent surgery or trauma. The presence of tissue crepitus favors clostridial infection. Gas gangrene can also be detected radiographically. (See "[Clostridial myonecrosis](#)".)

Cellulitis must be distinguished from other infections including ([table 3](#)):

- Erythema migrans – Erythema migrans is an early manifestation of Lyme disease; it consists of a region of erythema at the site of a tick bite, often with central clearing and a necrotic center ([picture 9](#)). The diagnosis is established based on serologic testing, although sensitivity in early disease is low. A similar lesion may occur in patients with Southern tick-associated rash illness. (See "[Clinical manifestations of Lyme disease in adults](#)" and "[Southern tick-associated rash illness \(STARI\)](#)".)
- Erythrasma – This condition manifests as brown- or orange-hued erythema with distinct borders in an intertriginous area. (See "[Erythrasma](#)", section on 'Clinical presentation' and "[Erythrasma](#)", section on 'Diagnosis').
- Herpes zoster – The rash of herpes zoster begins as erythematous papules that evolve into grouped vesicles ([picture 10](#)). The rash is generally limited to one dermatome but can affect two or three neighboring dermatomes. The diagnosis is established by herpes zoster virus polymerase chain reaction on a swab of vesicular fluid. (See "[Epidemiology, clinical manifestations, and diagnosis of herpes zoster](#)".)
- Pyomyositis – Severe localized muscle pain with movement or palpation are cardinal features of pyomyositis. Overlying erythema and warmth are often present. Radiographic imaging can lead to a presumptive diagnosis, and cultures from drainage procedures confirm the diagnosis. (See "[Primary pyomyositis](#)", section on 'Clinical manifestations' and "[Primary pyomyositis](#)", section on 'Diagnosis').

- Septic arthritis – Erythema and swelling may overlie a septic joint. Clinical manifestations include joint pain, swelling, warmth, and limited range of motion. The diagnosis of septic arthritis is established based on synovial fluid examination. (See "[Septic arthritis in adults](#)".)
- Septic bursitis – Cellulitis may precede or accompany septic bursitis. Distinguishing cellulitis with and without bursitis depends on skilled palpation. Radiographic imaging is warranted if septic bursitis is suspected. (See "[Septic bursitis](#)".)
- Osteomyelitis – Osteomyelitis may underlie an area of cellulitis. It is prudent to pursue imaging for assessment of bone involvement in the setting of chronic soft tissue infection that fails to improve with appropriate antibiotic therapy. (See "[Nonvertebral osteomyelitis in adults: Clinical manifestations and diagnosis](#)".)
- Mycotic aneurysm – Mycotic aneurysm should be suspected in the setting of erythema, swelling, and tenderness at an intravenous drug injection site such as antecubital fossa [74]. The diagnosis is established via ultrasonography. (See "[Overview of infected \(mycotic\) arterial aneurysm](#)".)
- Medical device infection – Orthopedic device infection and vascular graft infection involving (usually) a lower extremity can present as cellulitis. Infectious diseases consultation should be obtained in these cases. (See "[Prosthetic joint infection: Epidemiology, microbiology, clinical manifestations, and diagnosis](#)" and "[Overview of the evaluation and management of surgical site infection](#)".)

Noninfectious masqueraders of cellulitis (unilateral) include ([table 3](#)):

- Contact dermatitis – Contact dermatitis may be distinguished from cellulitis in that the contact dermatitis lesions are pruritic. Clinical features include erythema, edema, vesicles, bullae, and oozing. The reaction is generally limited to the site of contact and is associated with burning, stinging, or pain. (See "[Irritant contact dermatitis in adults](#)".)
- Acute gout – Acute gouty arthritis consists of severe pain, warmth, erythema, and swelling overlying a single joint. The diagnosis can be established by synovial fluid analysis, which should demonstrate the characteristic urate crystals of gout or the calcium pyrophosphate crystals of pseudogout. Additional clues suggestive of gout include involvement of the first metatarsophalangeal joint, prior self-limited attacks of arthritis, and presence of tophi. (See "[Clinical manifestations and diagnosis of gout](#)".)

- Drug reaction – A drug reaction presents with an erythematous maculopapular rash that involves the trunk and proximal extremities. It may be accompanied by pruritus, low-grade fever, and mild eosinophilia. The diagnosis is suspected in a patient receiving drug treatment who presents with a rash of recent onset. The clinical suspicion can be substantiated by histopathologic examination of a skin biopsy. (See "[Exanthematous \(maculopapular\) drug eruption](#)".)
- Vasculitis – The morphology of cutaneous lesions of vasculitis is variable. Macular and papular (including palpable purpura) lesions are characteristically nonblanchable due to the presence of extravasated erythrocytes in the dermis, which occurs as a result of damaged vessel walls. The diagnosis is established by skin biopsy. (See "[Evaluation of adults with cutaneous lesions of vasculitis](#)".)
- Hematoma – Hematomas may have an erythematous, orange, or violaceous hue and may be painful. They are often associated with local trauma. (See "[Soft tissue musculoskeletal injuries of the abdomen, flank, and lumbar region in adults and adolescents](#)", section on 'Muscle contusions' and "[Soft tissue musculoskeletal injuries of the abdomen, flank, and lumbar region in adults and adolescents](#)", section on 'Rectus abdominus sheath hematoma'.)
- Insect bite – An insect bite triggers an inflammatory reaction at the site of punctured skin, which appears within minutes and consists of pruritic local erythema and edema. In some cases, a local reaction is followed by a delayed skin reaction consisting of local swelling, itching, and erythema. (See "[Insect and other arthropod bites](#)".)
- Deep venous thrombosis – Findings suggestive of cellulitis involving the lower extremity should prompt consideration of deep venous thrombosis; the evaluation consists of ultrasound evaluation. (See "[Clinical presentation and diagnosis of the nonpregnant adult with suspected deep vein thrombosis of the lower extremity](#)".)
- Panniculitis – Panniculitis refers to inflammation of subcutaneous fat and may have many causes, both infectious and noninfectious ([table 4](#)). The diagnosis is confirmed via biopsy. (See "[Panniculitis: Recognition and diagnosis](#)".)
- Vaccination site reaction – A local reaction to vaccination manifests with erythema, swelling, and tenderness at the injection site; these are typically self-limited. (See "[Allergic reactions to vaccines](#)", section on 'Delayed vaccine reactions' and "[Allergic reactions to vaccines](#)", section on 'Reactions to COVID-19 vaccines'.)

- Calciphylaxis – This condition occurs in patients with chronic kidney disease, especially those on hemodialysis, and manifests as extremely painful indurated nodules or plaques that progress to ulceration. (See "[Calciphylaxis \(calcific uremic arteriolopathy\)](#)".)
- Malignancy – Some malignancies present with slowly progressive erythematous patches or plaques (eg, inflammatory breast cancer, extramammary Paget disease). (See "[Inflammatory breast cancer: Clinical features and treatment](#)" and "[Vulvar cancer: Epidemiology, diagnosis, histopathology, and treatment](#)", section on 'Paget disease of the vulva' and "[Carcinoma of the penis: Epidemiology, risk factors, and pathology](#)", section on 'Extramammary Paget disease').)
- Eosinophilic cellulitis (Well syndrome) – This condition is characterized by recurrent erythematous patches or plaques that are typically pruritic and nontender. (See "[Eosinophilic cellulitis \(Wells syndrome\)](#)".)
- Erythema ab igne - Erythema ab igne is an erythematous pigmented dermatosis resulting from repeated exposures to moderate heat or infrared radiation. The diagnosis is established clinically and may be confirmed by biopsy. (See "[Acquired hyperpigmentation disorders](#)", section on 'Erythema ab igne').)
- Radiation recall – Certain chemotherapeutic agents can trigger painful erythema at sites of prior radiation therapy. The condition may occur minutes to months after drug administration. (See "[Cutaneous adverse effects of conventional chemotherapy agents](#)", section on 'Radiation recall dermatitis and radiation enhancement').)

Noninfectious masqueraders of cellulitis (bilateral) include ([table 3](#)):

- Stasis dermatitis – Stasis dermatitis is an inflammatory dermatosis of the lower extremities that occurs in patients with chronic venous insufficiency. It is usually bilateral but can be unilateral in the setting of anatomic asymmetry. The diagnosis is usually established clinically ([figure 1](#)). (See "[Stasis dermatitis](#)" and "[Clinical manifestations of lower extremity chronic venous disease](#)".)
- Lipodermatosclerosis – Lipodermatosclerosis is a fibrosing panniculitis of the subcutaneous tissue that can develop in the setting of chronic venous insufficiency following severe cases of deep venous thrombosis or associated with lymphatic compromise. Typically the overlying skin is heavily pigmented and bound down to the subcutaneous tissues. (See "[Clinical manifestations of lower extremity chronic venous disease](#)", section on 'Lipodermatosclerosis (C4b)').)

- Lymphedema – Lymphedema is abnormal accumulation of interstitial fluid resulting from injury or anatomic abnormality of the lymphatic system. The diagnosis is usually established clinically. (See "[Clinical features and diagnosis of peripheral lymphedema](#)".)

Skin abscess — Skin lesions that should be distinguished from skin abscess include:

- Epidermoid cyst – An epidermoid cyst is a skin-colored cutaneous nodule. The diagnosis is usually clinical, based on the clinical appearance of a discrete cyst or nodule, often with a central punctum, that is freely movable on palpation. Epidermoid cysts may become secondarily infected. (See "[Overview of benign lesions of the skin](#)", section on '[Epidermoid cyst](#)').
- Folliculitis – Folliculitis refers to inflammation of one or more hair follicles. The diagnosis is often established clinically; rarely, Gram stain and culture or skin biopsy may be warranted to differentiate folliculitis from other conditions. (See "[Infectious folliculitis](#)".)
- Hidradenitis suppurativa – Hidradenitis suppurativa is a chronic suppurative process involving the skin and subcutaneous tissue of intertriginous skin. The diagnosis is usually established clinically. (See "[Hidradenitis suppurativa: Pathogenesis, clinical features, and diagnosis](#)".)
- Nodular lymphangitis – Nodular lymphangitis presents as nodular subcutaneous swellings along the course of the lymphatic channels. The differential diagnosis is broad and is summarized separately. (See "[Lymphangitis](#)", section on '[Nodular lymphangitis](#)').
- Botryomycosis – Botryomycosis is a chronic, suppurative infection characterized by a granulomatous inflammatory response to *S. aureus* and other bacteria; it occurs most commonly in immunocompromised patients. The diagnosis is established via Gram stain, culture, or examination of pus for granules. (See "[Botryomycosis](#)".)
- Myiasis – Myiasis presents as an enlarging nodular mass associated with an insect bite; it is caused by penetration of fly larvae into subdermal tissue. The diagnosis is established via clinical manifestations in the setting of epidemiologic exposure to tropical and subtropical areas. (See "[Skin lesions in the returning traveler](#)", section on '[Myiasis](#)').

The differential diagnosis for skin and soft tissue infections in immunocompromised patients is summarized separately. (See "[Clinical manifestations, diagnosis, and grading of acute graft-versus-host disease](#)", section on '[Differential diagnosis](#)').

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Skin and soft tissue infections](#)".)

SUMMARY AND RECOMMENDATIONS

- **Definitions** – Cellulitis is an infection of the deeper dermis and subcutaneous fat. Erysipelas is a more superficial infection that involves the upper dermis and superficial lymphatics. A skin abscess is a collection of pus in the dermis or subcutaneous space ([figure 2](#)). (See '[Introduction](#)' above.)
- **Risk factors** – Healthy individuals of any age can develop cellulitis, but it occurs most frequently in middle-aged and older adults. Predisposing conditions include skin barrier disruption (eg, abrasion, ulcer, eczema, toe web intertrigo, tinea pedis), edema from venous insufficiency or impaired lymphatic drainage, obesity, and immunocompromise. (See '[Epidemiology](#)' above.)
- **Microbiology** – Beta-hemolytic streptococci, usually group A *Streptococcus* (ie, *S. pyogenes*), cause the vast majority of erysipelas cases and are the most common cause of cellulitis. *Staphylococcus aureus*, including methicillin-resistant *S. aureus* (MRSA), is the most common cause of skin abscess but is a less common cause of cellulitis. Gram-negative aerobic bacilli cause a minority of cases.

Certain exposures may be associated with specific pathogens ([table 1](#)). (See '[Microbiology](#)' above.)

- **Clinical manifestations** – (see '[Clinical manifestations](#)' above)
 - **Cellulitis** – Cellulitis manifests as an area of skin erythema, edema, warmth, and tenderness with indistinct borders. The lower extremities are the most common site of involvement, and the infection is nearly always unilateral.
Other potential findings include a shiny appearance, an orange-peel texture ("peau d'orange"), petechiae, ecchymoses, superficial bullae, purulent discharge, lymphangitis, and regional lymphadenopathy.
- Cellulitis tends to present acutely, with localized symptoms developing over several hours, although more indolent presentations can occur over a few days. Systemic manifestations, such as fever, tachycardia, or sepsis, may also occur but are not always present.

- **Erysipelas** – Compared with cellulitis, erysipelas often has a brighter red appearance and has distinct borders that are often raised. The presentation is often more acute than cellulitis.
- **Skin abscess** – A skin abscess manifests as a painful, fluctuant, erythematous nodule, with or without surrounding cellulitis.
- **Diagnosis** – The diagnosis of cellulitis or erysipelas is made clinically in a patient with new onset of skin erythema, edema, warmth, and tenderness. Diagnosis of skin abscess is usually made clinically in a patient with a new tender fluctuant skin nodule.

History should assess the pace of the presentation, underlying comorbidities, accompanying systemic symptoms, and potential exposures. Examination should evaluate potential portals of entry in addition to the affected area. (See '[Diagnosis](#)' above.)

- **Limited role for testing** – Laboratory testing is not required for uncomplicated skin and soft tissue infection.
 - **Cultures** – We obtain blood cultures in patients with extensive or rapidly progressive cellulitis, systemic signs of infection (eg, fever), older age, or neutropenia. Cultures of swabs of intact skin should not be performed because positive results are often not indicative of the underlying pathogen. Cultures of skin abscess drainage can identify the causative pathogen and should be performed.
 - **Imaging** – Ultrasound can help evaluate for a skin or deep abscess, if suspected. However, imaging studies cannot reliably distinguish cellulitis from necrotizing fasciitis or gas gangrene and should not delay surgical intervention if these are suspected. (See '[Diagnosis](#)' above.)

• **Differential diagnosis**

- **Cellulitis and erysipelas** – Misdiagnosis of skin findings as cellulitis or erysepilas is common, and alternative infectious and noninfectious diagnoses should be considered in all patients ([table 3](#)).

In particular, systemic toxicity with rapidly progressive erythema or pain out of proportion to exam findings should prompt consideration of severe infection, including necrotizing soft tissue infection (eg, necrotizing fasciitis), toxic shock syndrome, and myonecrosis.

Noninfectious processes that are commonly misdiagnosed as cellulitis include stasis or contact dermatitis (figure 1), lymphedema, and deep vein thrombosis. (See 'Cellulitis and erysipelas' above.)

- **Skin abscess** – Skin conditions that can be mistaken for skin abscess include epidermoid cyst, hidradenitis suppurativa, botryomycosis, and myiasis. (See 'Skin abscess' above.)

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Topic 110529 Version 34.0

GRAPHICS

Distinguishing stasis dermatitis from cellulitis of the lower extremities



Stasis Dermatitis vs. Cellulitis of the Lower Extremity

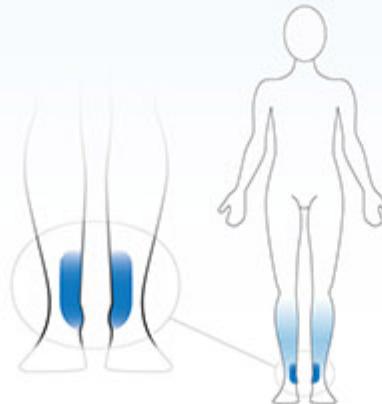
Stasis Dermatitis

Inflammatory skin condition caused by venous insufficiency.

DISTRIBUTION

Lower extremity involvement is usually **bilateral** in patients with bilateral edema.

Medial ankle most frequently involved.



COURSE

- Chronic with acute exacerbations

CLINICAL FEATURES

- Poorly-demarcated, chronic, eczematous, scaly patches or plaques
- Pink to red, violaceous, or hyperpigmented color change
- Chronic edema
- Usually pruritic



Skin color can influence the appearance of erythema
In lightly pigmented skin, stasis dermatitis and cellulitis usual

Cellulitis

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exhibit prominent pinkness or redness. As pigmentation darkens, a subtle pink or violaceous color may be the most prominent color change. In skin of color, chronic stasis dermatitis may also cause brown to black hyperpigmentation.

ADDITIONAL FEATURES

Systemic symptoms:

Absent

Associated local findings:



Eczematous plaques



Hemosiderin deposition



Varicose veins



Ulcers



Bullae

RISK FACTORS

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Chronic lower extremity edema.

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KEY CONCEPTS

Stasis dermatitis and cellulitis are common conditions that most often occur on inflammation and edema. Misdiagnosis of stasis dermatitis as cellulitis is common, so history and physical examination is essential for distinguishing these conditions.

Key features that suggest **cellulitis** over stasis dermatitis include:

- Acute onset
- Warmth
- Systemic symptoms (vomiting, fever)
- Unilateral involvement
- Tenderness

The recognition of chronic eczematous skin changes (*inflamed, scaling patches or plaques*) supports **stasis dermatitis**. Stasis dermatitis is usually (*though not always*) associated with red-brown pigmentation due to hemosiderin deposition.

Cellulitis can occur in patients with preexisting stasis dermatitis. The recognition of key features of cellulitis (acute onset, unilateral involvement, warmth, tenderness, systemic symptoms) may help identify patients who have coexisting conditions.

Stasis dermatitis, Clinical features images and Cellulitis, Clinical features, right image – Original photograph used with permission from Dr. P. Marazzi/Science Photo Library. Stasis dermatitis, Additional features, first three images in top row and Cellulitis, Additional features, first and third image in top row -- Reproduced with permission from Dr. P. Marazzi/Science Photo Library. Stasis dermatitis, Additional features, last image in top row – Reproduced with permission from Science Photo Library. Cellulitis, Additional features, center image in top row – Reproduced with permission from Biophoto Associates/Science Photo Library.

Microbiology of acute cellulitis

Exposure	Pathogen
Most common pathogens (regardless of exposure)	<ul style="list-style-type: none"> ▪ Group A <i>Streptococcus</i> (ie, <i>Streptococcus pyogenes</i>) ▪ Non-group A, beta-hemolytic streptococci (groups B, C, G, and F) ▪ <i>Staphylococcus aureus</i> (<i>S. aureus</i>)
Cirrhosis	<ul style="list-style-type: none"> ▪ Gram-negative bacilli: <ul style="list-style-type: none"> • <i>Klebsiella</i> spp • <i>Escherichia coli</i> • <i>Vibrio vulnificus</i> and <i>Vibrio parahaemolyticus</i>* • <i>Aeromonas</i> spp*
Splenic or humoral immune dysfunction	<ul style="list-style-type: none"> ▪ Encapsulated bacteria: <ul style="list-style-type: none"> • <i>Streptococcus pneumoniae</i> • <i>Haemophilus influenzae</i>
Neutropenia	<ul style="list-style-type: none"> ▪ <i>Pseudomonas aeruginosa</i> and other Gram-negative bacilli ▪ <i>Clostridium</i> spp ▪ Invasive fungal infections
Fresh water (lakes, rivers)	<ul style="list-style-type: none"> ▪ <i>Aeromonas hydrophila</i> ▪ <i>Plesiomonas shigelloides</i> ▪ <i>Edwardsiella tarda</i> ▪ <i>Pseudomonas aeruginosa</i> ▪ <i>Shewanella</i> spp
Salt water	<ul style="list-style-type: none"> ▪ <i>Vibrio vulnificus</i> and <i>Vibrio parahaemolyticus</i> ▪ <i>Erysipelothrix rhusiopathiae</i>
Shellfish ingestion, especially oysters	<ul style="list-style-type: none"> ▪ <i>Vibrio vulnificus</i> and <i>Vibrio parahaemolyticus</i>
Animal bite	<ul style="list-style-type: none"> ▪ <i>Pasteurella multocida</i> ▪ <i>Capnocytophaga canimorsus</i> ▪ Anaerobic bacteria
Human bite	<ul style="list-style-type: none"> ▪ <i>Eikenella corrodens</i> ▪ Anaerobic bacteria ▪ Viridans streptococci
Traumatic wound contaminated by soil	<ul style="list-style-type: none"> ▪ <i>Clostridium</i> spp ▪ <i>Pseudomonas aeruginosa</i> and other Gram-negative bacilli ▪ Fungi (eg, mucormycosis)

Nail puncture through sneakers	■ <i>Pseudomonas aeruginosa</i>
Recent travel	■ Depends on the location of travel

* In patients with cirrhosis, *Vibrio* spp are only isolated following salt-water or raw shellfish exposure; *Aeromonas* spp cause infections following fresh-water exposure.

Graphic 143607 Version 1.0

Risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) infection in adults

Health care exposures during the prior 12 months:

- Recent hospitalization
- Residence in a long-term care facility
- Recent surgery
- Hemodialysis

Patient-specific risk factors:

- Known MRSA colonization or past infection with MRSA
- Recent close contact with a person colonized or infected with MRSA
- HIV infection
- Injection drug use
- Homelessness
- Men who have sex with men
- Antibiotic use within prior 6 months

Environmental exposures associated with outbreaks of MRSA skin abscesses^{*}:

- Incarceration or working as prison guard
- Military service
- Attending schools or living in communities with high colonization rates
- Living in crowded conditions
- Attending or working in childcare centers
- Playing contact sports or sharing sporting equipment
- Sharing needles, razors, or other sharp objects

This list of risk factors is based on observational studies performed in different regions of the world. MRSA risk factors for individual countries may vary depending on local MRSA epidemiology.

MRSA: methicillin-resistant *Staphylococcus aureus*.

* These exposures are generally not associated with other types of MRSA infections, including cellulitis without abscess.

Graphic 53504 Version 14.0

Cellulitis with erythema and edema



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An extensive edematous and erythematous plaque on the arm.

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Graphic 128910 Version 1.0

Erysipelas of the leg



Erysipelas of the lower leg. The rash is intensely red, sharply demarcated, swollen, and indurated.

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Graphic 82542 Version 3.0

Cellulitis of the ankle



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Edema and erythema around the ankle and on the dorsal foot.

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Graphic 128906 Version 1.0

Resolving cellulitis of the leg



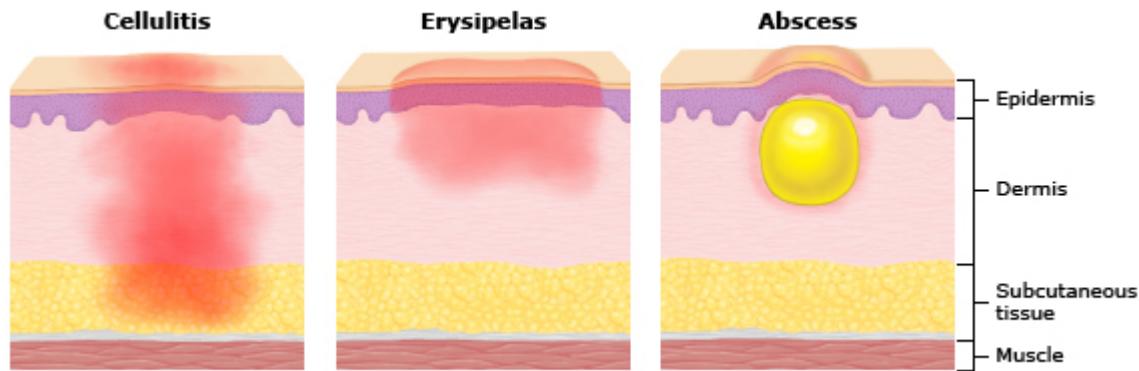
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Resolving cellulitis showing an extensive dusky, violaceous plaque with crusting on the leg.

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Graphic 128908 Version 1.0

Skin anatomy: Cellulitis, erysipelas, and skin abscess



Cellulitis and erysipelas manifest as areas of skin erythema, edema, and warmth; they develop as a result of bacterial entry via breaches in the skin barrier. Cellulitis involves the deeper dermis and subcutaneous fat; in contrast, erysipelas involves the upper dermis, and there is clear demarcation between involved and unininvolved tissue. A skin abscess is a collection of pus within the dermis or subcutaneous space.

Graphic 110605 Version 2.0

Erysipelas



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Erysipelas lesions are raised above the level of surrounding skin, and there is a clear line of demarcation between involved and uninvolves tissue.

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Graphic 67112 Version 6.0

Cellulitis with erosion



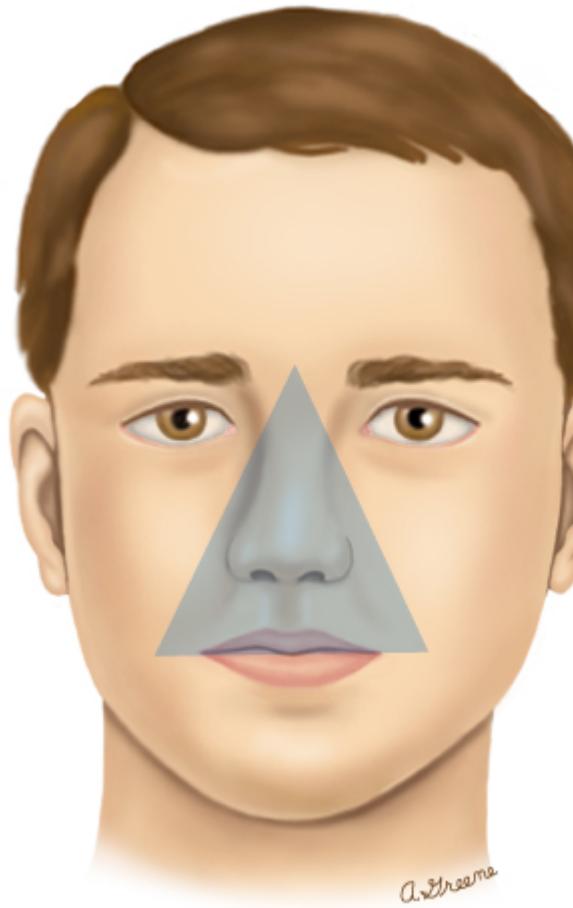
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An edematous plaque with overlying vesiculation and erosion on the leg.

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Graphic 128909 Version 1.0

Central triangle of the face



A. Stremma

Rarely, infections involving the medial third of the face (ie, the areas around the eyes and nose) can be complicated by septic cavernous thrombosis, since the veins in this region are valveless.

Graphic 55404 Version 4.0

Skin abscess



Courtesy of Larry M Baddour, MD.

Graphic 53261 Version 1.0

Carbuncle



Carbuncle, which is a series of abscesses in the subcutaneous tissue that drain via hair follicles.

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Graphic 70089 Version 3.0

Differential diagnosis of acute cellulitis

Etiology	Distinguishing features
Infectious	
Erysipelas	<ul style="list-style-type: none"> ▪ Bright red appearance, most commonly on malar region of face ▪ Borders are raised and distinct
Necrotizing soft tissue infection (eg, necrotizing fasciitis)*	<ul style="list-style-type: none"> ▪ Dusky skin appearance or woody induration ▪ Pain out of proportion to exam; tenderness may extend beyond the area of visible skin inflammation ▪ Rapid progression with severe sepsis or shock ▪ Crepitus and/or bullae in some cases
Toxic shock syndrome*	<ul style="list-style-type: none"> ▪ Sustained hypotension ▪ Signs of organ failure (eg, acute kidney injury, thrombocytopenia)
Clostridial myonecrosis (gas gangrene)*	<ul style="list-style-type: none"> ▪ Rapidly progressive severe muscle pain ▪ Crepitus and/or bullae in some cases
Skin abscess	<ul style="list-style-type: none"> ▪ Tender, erythematous, fluctuant subcutaneous nodule
Pyomyositis*	<ul style="list-style-type: none"> ▪ Localized muscle pain
Vascular graft infection*	<ul style="list-style-type: none"> ▪ Cellulitis overlying a vascular graft ▪ Bloodstream infection
Mycotic aneurysm*	<ul style="list-style-type: none"> ▪ Painful pulsatile mass
Native or prosthetic joint infection*	<ul style="list-style-type: none"> ▪ Erythema overlying a joint ▪ Severe pain with joint manipulation, or inability to bear weight due to pain ▪ Prosthetic joint infection may have less severe pain
Acute osteomyelitis	<ul style="list-style-type: none"> ▪ Erythema, edema, and point tenderness overlying bone
Erythema migrans	<ul style="list-style-type: none"> ▪ Nontender, slowly progressive round or oval erythematous patch ▪ May have central clearing ▪ Patient may recall tick bite
Erythrasma	<ul style="list-style-type: none"> ▪ Brown- or orange-hued erythema ▪ Distinct borders ▪ Located in intertriginous areas
Herpes zoster	<ul style="list-style-type: none"> ▪ Vesicles or crusted lesions in dermatomal pattern

Vascular

Venous stasis dermatitis	<ul style="list-style-type: none">▪ Chronic scaly plaques with acute erythematous exacerbations▪ Usually bilateral▪ May be pruritic▪ Often involves the medial ankle
Lipodermatosclerosis (panniculitis from chronic venous stasis)	<ul style="list-style-type: none">▪ Firm, pigmented, painful induration▪ Often begins on medial ankle▪ Can be unilateral or bilateral
Lymphedema	<ul style="list-style-type: none">▪ Chronic extremity edema▪ May have pitting or firm induration▪ Can be unilateral or bilateral
Deep venous thrombosis*	<ul style="list-style-type: none">▪ Unilateral extremity edema▪ Erythema, warmth, tenderness, and rate of progression less impressive compared with cellulitis▪ May have low-grade fever
Hematoma	<ul style="list-style-type: none">▪ Erythematous, orange, or violaceous hue▪ Often associated with local trauma▪ May be painful

Allergic or inflammatory

Contact dermatitis	<ul style="list-style-type: none">▪ Pruritic eczematous erythema with edema, vesicles, and distinct border▪ Located around area of contact with allergen▪ Patient may recall exposure
Insect bite/sting	<ul style="list-style-type: none">▪ Painful or pruritic erythema expanding from site of bite▪ Examination may reveal bite marks▪ Some patients may not recall bite
Fixed drug reaction	<ul style="list-style-type: none">▪ Round or oval erythematous or violaceous patch or patches▪ Located on any part of body▪ Begins 30 minutes to 8 hours after drug administration
Reaction to foreign body implant (eg, metal, mesh, silicone)	<ul style="list-style-type: none">▪ Erythema overlying site of known foreign material
Injection site reaction	<ul style="list-style-type: none">▪ Erythema expanding from site of injection
Eosinophilic cellulitis (Well syndrome)	<ul style="list-style-type: none">▪ Recurrent erythematous patches or plaques▪ Pruritic and non-tender

Miscellaneous

Compartment syndrome*	<ul style="list-style-type: none">▪ Tense, firm, "wood-like" edema▪ Pain out of proportion to exam
Panniculitis	<ul style="list-style-type: none">▪ Palpable nodules or plaques on deep palpation
Malignancy*	<ul style="list-style-type: none">▪ Slowly progressive erythematous patches or plaques▪ Examples include Inflammatory breast cancer and extramammary Paget disease of the genitalia or perineum
Calciphylaxis	<ul style="list-style-type: none">▪ Extremely painful indurated nodules or plaques that progress to necrotic ulcers▪ Occurs in patients with chronic kidney disease, especially those on hemodialysis
Radiation recall	<ul style="list-style-type: none">▪ Painful erythema at site of prior radiation therapy▪ Triggered by certain chemotherapeutic agents▪ Occurs minutes to months after drug administration

The differential diagnosis of cellulitis is broad, and this table is not all-inclusive. In cases of presumed refractory cellulitis, consultation with an expert (eg, infectious diseases clinician, dermatologist) may be helpful.

* These conditions are associated with high morbidity or mortality if not promptly recognized. Surgical intervention is often required.

Graphic 143606 Version 1.0

Erythema migrans



Erythema migrans with central clearing and a necrotic center.

Courtesy of Dori F Zaleznik, MD.

Graphic 81270 Version 1.0

Herpes zoster



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Grouped vesicles and underlying erythema are present on the back.

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Graphic 58282 Version 6.0

Clinical classification of panniculitis based upon etiology*

Infection and infestation

Bacterial, mycobacterial, or fungal infections

Parasites

Arthropod bite

Trauma

Cold panniculitis (popsicle panniculitis, equestrian panniculitis, subcutaneous fat necrosis of the newborn, sclerema neonatorum)

Blunt trauma

Factitial panniculitis (iatrogenic, accidental, or intentional injections)

Post-irradiation panniculitis

Enzymatic destruction

Pancreatic panniculitis

Alpha-1 antitrypsin deficiency

Malignancy

Lymphoma

Cytophagic histiocytic panniculitis[¶]

Leukemia cutis/metastases

Deposition

Gouty panniculitis

Calciphylaxis

Hyperoxaluria

Inflammatory

Erythema nodosum

Lipodermatosclerosis (sclerosing panniculitis)

Erythema nodosum migrans/subacute nodular migratory panniculitis/chronic erythema nodosum^Δ

Erythema induratum/nodular vasculitis

Vasculitis (especially polyarteritis nodosa)

Superficial migratory thrombophlebitis

Post-steroid panniculitis

Lupus panniculitis
Panniculitis of dermatomyositis
Connective tissue disease panniculitis
Deep morphea
Necrobiosis lipoidica diabetorum
Rheumatoid nodule
Subcutaneous granuloma annulare
Subcutaneous sarcoidosis
Erythema nodosum leprosum

* The list is not intended to be exhaustive. Some conditions may be multifocal in etiology (eg, traumatic and inflammatory). In some conditions, the etiology is not definitively established.

¶ Cytophagic histiocytic panniculitis may not represent malignancy in all cases.

Δ There is not consensus about whether subacute nodular migratory panniculitis is a variant of chronic erythema nodosum or a separate entity.

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