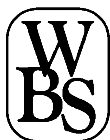

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Soft tissue disease

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In the assessment of patients with soft tissue complaints, it is important to consider infectious etiologies in the differential diagnosis, especially in immunocompromised hosts. The exact categorization of some bacterial infections of the soft tissues may be difficult. The structures potentially involved include the skin, subcutaneous tissue, fascia, and skeletal muscle. Classification is usually based upon the anatomic structure involved, the infecting organism, and the clinical picture. The categorization is complicated by the fact that some infections may involve several soft tissue components and multiple bacterial species (Fig. 1). In this review, we will cover cutaneous and subcutaneous tissue infections, fasciitis, septic bursitis, tendonitis, and pyomyositis.

Cutaneous infections

Impetigo

Impetigo is an initially vesicular, later crusted superficial infection of the skin. Most cases occur in children. Causative organisms include group A strep (20%–30%), and *Staphylococcus aureus* [1–3]. The group A streptococci responsible usually belong to different M types than those found in the strains producing pharyngitis.

Pathogenesis

Impetigo is most common during hot humid summer weather. Epidemiologic studies demonstrate colonization of normal skin with group A streptococcus about 10 days before the development of skin lesions [4]. Minor trauma sets off infected lesions. Impetigo is highly communicable. Spread in families is facilitated by crowding and poor hygiene.

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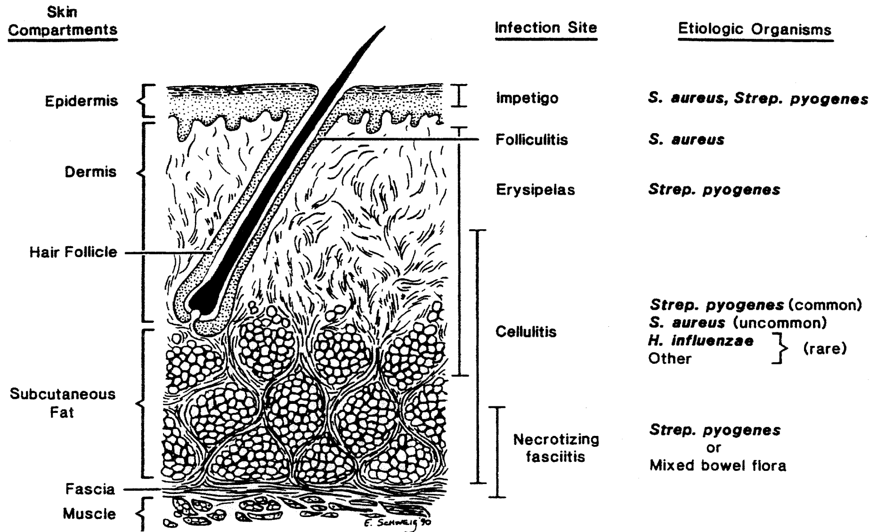

Fig. 1. Cutaneous anatomy, sites of infection, and infecting organisms. (*From* Gorbach infectious diseases. 3rd edition. Philadelphia: Lippincott Williams & Wilkins; with permission.)

Clinical findings

The infection begins as small vesicles, at times with narrow inflammatory halos that rapidly pustulate and rupture. A thick, golden yellow crust forms from the purulent discharge. The lesions are painless; they remain superficial and do not ulcerate. Pruritus is common and leads to the spread of infection. Constitutional symptoms are minimal, and mild regional lymphadenopathy is common.

Differential diagnosis

Other vesicular lesions must be included in the differential diagnosis, such as viruses (Herpes simplex, varicella), acute palmoplantar pustulosis, pustular psoriasis, and primary cutaneous listeriosis. [5]

Treatment

Therapy includes systemic antibiotics. Penicillin has been the drug of choice in the past for ordinary impetigo because of the predominant role of group A streptococcus and the possible occurrence of acute glomerulonephritis as a sequela. Mixed staphylococcus- streptococcus impetigo may require treatment with penicillinase-resistant penicillin.

Erysipelas

Erysipelas is a distinctive type of superficial cellulitis of the skin with prominent lymphatic involvement. The lesion is painful with a bright red, edematous, indurated appearance and an advancing raised border that is sharply

demarcated from normal skin. Fever is commonly associated [6]. The causative organism is usually group A strep, (rarely other streptococci or staphaureus) and the condition is more common in infants, young children, and older adults. Seventy to 80% of the lesions affect the lower extremities and 5% to 10% affect the face [7]. Predisposing factors include alcohol, diabetes mellitus, paraparesis, venous stasis, nephrotic syndrome, radical mastectomy, and areas of pre-existing lymphedema [7]. This infection can itself produce lymphatic obstruction and tends to recur in areas of earlier infection. The differential diagnosis includes giant urticaria, rash of familial Mediterranean fever, erythema chronica migrans, early Herpes zoster involving cranial nerves V and VII, and dermatitis. An erysipelas-like skin lesion has been reported in several patients with hypogammaglobulinemia and *Campylobacter jejuni* bacteremia [8]. Early cases can be treated with intramuscular penicillin. More extensive cases should be hospitalized for intravenous penicillin [6].

Staphylococcal scalded skin syndrome

Staphylococcal scalded skin syndrome is the most severe manifestation of infection with *S aureus* strains, producing an exfoliative exotoxin and is characterized by widespread bullae and exfoliation. This condition usually occurs in children but rarely occurs in adults. It begins abruptly with fever, skin tenderness, and a scarlatiniform rash. The Nikolsky sign may appear when large, flaccid, clear bullae form, rupture, and result in separation of sheets of skin [9–11]. Lesions resolve over 2 weeks with appropriate treatment and antimicrobial therapy.

Cellulitis

Cellulitis is an acute spreading infection of the skin that extends deeper than erysipelas and involves the subcutaneous tissues. *S aureus* and group A streptococcus are the most common etiologic agents. Organisms are usually introduced by local trauma or an underlying skin infection; it rarely results from hematogenous spread.

Clinical findings

The involved area is often extensive and the lesion is red, hot, and swollen. In contrast to erysipelas, the borders are not elevated or sharply demarcated. Small patches of skin may necrose and abscesses may develop. Fever, chills, malaise, and regional lymphadenopathy are common.

Aspiration of material from the advancing edge of cellulitis, skin biopsy, and blood cultures are successful in identifying the organism in only 25% of patients [12]. In light of the low yield of aspiration, this technique can be used primarily when unusual pathogens are suspected as in immunocompromised patients, when fluctuant areas develop, or when initial antibiotic therapy has been unsuccessful [13] (Box 1).

Box 1. Conditions that can mimic bacterial cellulitis

- Other infectious causes
  - Cutaneous tuberculosis
  - Cutaneous nocardiosis
  - Cutaneous cryptococcosis
  - Inflammatory tinea capitis/cruris
  - Septic arthritis
  - Septic bursitis
- Hematology/oncology
  - T-cell lymphoma/leukemia
  - Kaposis sarcoma
  - Sweets syndrome
  - Carcinoma erysipeloids
- Miscellaneous
  - Caffey-Silverman syndrome (infantile cortical hyperostosis)
  - Dermal mucinosis
  - Deep venous thrombosis
  - Rash of familial Mediterranean fever
  - Superficial thrombophlebitis
  - Insect bite

Treatments

Presumptive therapy in mild cases is penicillin G. If *S aureus* is suspected, treatment is with a penicillinase-resistant penicillin, or erythromycin in penicillin-allergic patients. Local measures include immobilization, elevation, and cool sterile saline dressings to remove purulent exudate from any associated ulcer or infected abrasion and to decrease local pain. Prevention of recurrent cellulitis in patients with edema includes support stockings and good skin hygiene. If there is a recurrence despite initial measures, there may be a role for prophylactic penicillin G or erythromycin [14].

Septic bursitis

Because of their superficial location and exposure to trauma, the subcutaneous olecranon, prepatellar, and superficial infrapatellar bursae are most commonly affected by bacterial infection. It is presumed that local cutaneous trauma leads to direct inoculation of the bursae with normal skin flora. Alternatively, septic bursitis may develop by spread of cellulitis to already traumatized subcutaneous bursae. The frequent finding of skin lesions such as abrasions, lacerations, and contusions near the infected bursa and the lack of bacteremia support the theory of direct transcutaneous bacterial seeding [15]. Bacteremia has been reported in up to 8% of cases with septic bursitis [16–18]. Trauma precedes septic bursitis in

50% to 70% of cases. The trauma is often associated with occupational or recreational activity, it may be acute or chronic trauma, isolated or repetititive, leading to inflammation and infection in the superficial bursae [19,20]. Examples of trauma related to the development of prepatellar bursitis include wrestling [21] and carpet laying [22]. For olecranon bursitis, sustained pressure on the elbows, such as that occurring [23,24] during dialysis or for aiding the respiratory effort in patients with chronic obstructive pulmonary disease, seems to be a predisposing factor [15]. Systemic illness such as diabetes mellitus and chronic alcohol abuse predispose one to septic bursitis [16]. Previous inflammation of the bursae by rheumatoid arthritis, gout, or trauma also predisposes one to the condition [18]. Immunocompromised patients do not appear to be at any greater risk of developing septic bursitis, but they may have a more dramatic presentation and a more prolonged course [15,25].

Diagnosis

Septic and nonseptic bursitis must be considered in the differential diagnosis of a painful, swollen joint. The history, physical, and laboratory are helpful in distinguishing between these possibilities.

In septic olecranon or prepatellar bursitis there is usually discrete bursal swelling, maximal tenderness in the center of the bursa, varying degrees of local inflammation, and preservation of joint motion [26]. Peribursal cellulitis is a frequent finding and may be severe enough to obscure the underlying bursitis [17].

Analysis of the bursal fluid aids in the diagnosis of the cause of bursal inflammation. Superficial bursae react less intensely than diarthrodial joints to pathogenic stimuli, thus in septic bursitis, white blood cell (WBC) counts in bursal fluid tend to be lower. In a review of 13 cases, 8 had WBC of 20,000 or less [27], thus a relatively low WBC count in bursal fluid does not rule out infection. Grossly purulent fluid suggests an infectious cause, but the fluid in septic bursitis is often only slightly turbid and may be hemorrhagic [28].

The diagnosis is confirmed by culture of freshly aspirated fluid. The reported sensitivity of the Gram stained smear of bursal fluid in culture-proven septic bursitis has ranged from 15% [29] to approximately 50% [27,30].

Gram-positive organisms account for the majority of cases of septic bursitis and *S aureus* is the most common (up to 80% to 100% of published cases). *Streptococcus* sp account for up to 5% to 30%, with group A β-hemolytic streptococci being the most common [31]. Unusual Gram-positive organisms such as enterococcus and diphtheroids have been described in foot infections [32]. Gram-negative infections are rare [33]. Appropriate treatment of septic bursitis includes both antibiotic therapy and bursal drainage. The choice of antibiotics should be guided by bursal fluid analysis as well as the age and comorbidities of the patient [34]. The indications for surgical intervention include failure of needle aspiration to drain the bursa adequately, inaccessibility of the bursal site for repeated needle aspirations, presence of a foreign body, and presence of necrotic tissue [31].

Resistance to treatment may result from the immunocompromised status of the host, pre-existing bursal pathology (ie, tophus or rheumatoid nodule), long duration of infection preceding treatment, infection extending beyond the bursa (ie, osteomyelitis) [34], or loculated pus in an adjacent tissue space [31].

Chronic infectious olecranon bursitis

This presents with firm swelling and little tenderness. Infection follows minor trauma, which inoculates low-virulence organisms including Prototheca, non-tuberculous mycobacteria, and agents of phaeohyphomycosis. A history of indolent chronic bursitis resistant to local therapy with negative cultures should raise the suspicion of fungal or mycobacterial bursitis.

Acute flexor tenosynovitis

Acute flexor tenosynovitis is a bacterial infection of the closed synovial sheaths of the flexor tendons. Infection is usually caused by penetrating trauma to the sheath but occasionally may result from hematogenous spread. *S aureus* is the most commonly isolated organism [35]. Disseminated *Neisseria gonorrhea* is another common cause of tenosynovitis and often involves multiple tendons simultaneously, especially around the dorsum of the wrists, fingers, ankles, and toes [36,37].

The involved digit is held in semiflexion. There is volar erythema, the digital crease tends to be erased, there is dorsal swelling in the digit, and passive extension elicits pain. Passive flexion, although limited by swelling, is relatively painless [38].

Treatment includes systemic antibiotics and surgical drainage of the fingers [39–41].

Infection can lead to tendon necrosis and proximal spread if not treated early.

Chronic microbial tenosynovitis

Chronic tenosynovial infections are usually mycobacterial or fungal. Marked proliferative changes usually result in a relatively painless enlargement of the sheath. Causative organisms include *Mycobacterium tuberculosis* [42,43], *Mycobacterium marinum* [44], and *Mycobacterium terrae* [45].

M marinum inhabits both warm fresh- and saltwater environments. Infection is associated with scrapes or penetrating trauma from boats, piers, fishing equipment, and fish bites. The trauma may precede the development of clinical infection by up to 6 months and can be acquired from fish tanks or swimming pools. In addition to involving the tenosynovium, this organism can involve the bursae, bone, or joint. Verrucous skin lesions and subcutaneous granuloma also can be present [46–49]. Histologic specimens show typical caseating and

noncaseating granulomas [46] and diagnosis is dependent upon appropriate tissue biopsy and culture.

Fungal agents causing chronic tenosynovitis include *Exophiala mansonii*, *Sporothrix schenckii*, *Phialophora richardsia*, *Prototheca wickerhamii*, histoplasmosis, coccidiomycosis, *Candida*, and *Cryptococcus neoformans* [38].

Anaerobic cellulitis

Anaerobic cellulitis is a necrotizing infection of devitalized subcutaneous tissue which can be caused by clostridial species and a variety of non–spore-forming anaerobic bacteria such as *Bacteroides* spp, Peptostreptococcus, or Peptococcus, alone or as a mixed infection [50].

The infecting organisms are introduced into subcutaneous tissue through a dirty or inadequately debrided traumatic wound, surgical contamination, or from a pre-existing localized infection. The localized infection is often in the case of clostridial species, located in the perineum, abdominal wall, buttocks, and lower extremities, areas that can be contaminated by fecal flora. This can rarely develop in situations other than after primary skin injury, as in the setting of leukemia and granulocytopenia [51].

Clinical features

Local pain, tissue swelling, and systemic features may be mild, distinguishing anaerobic cellulitis from gas gangrene (clostridial myonecrosis). Thin, dark, foul-smelling drainage from the wound is characteristic. Crepitus is present and may extend very widely. Soft tissue X-rays show abundant gas. Surgical explorations distinguish cellulitis from gas gangrene, revealing normal muscle tissue in the former. The distinction is important to make because anaerobic cellulitis requires much less extensive surgical debridement. Initial antibiotic therapy is chosen to cover the possibility of a polymicrobial infection. Intravenous penicillin and clindamycin or metronidazole covers anaerobic organisms. If Gram stain reveals Gram-negative bacilli, an aminoglycoside, ciprofloxacin, or a third-generation cephalosporin may be added. Definitive antimicrobial selection is based on culture results and susceptibility tests.

Necrotizing fasciitis

Necrotizing fasciitis is an uncommon severe infection involving the subcutaneous soft tissues, particularly the superficial and often the deep fascia. There are two types, based on the causative organisms. In type I at least one anaerobic species (*Bacteroides* and *Peptostreptococcus* spp) is isolated in combination with one or more facultative anaerobic species such as streptococci (other than group A) and members of Enterobacteriaceae (eg, *E coli*, Enterobacter, Klebsiella, proteus). In type II, known as hemolytic streptococcal gangrene, group A streptococci are isolated alone or in combination with other species.

Necrotizing fasciitis is usually an acute process, but rarely follows a subacute progressive course. Any part of the body can be affected, but most commonly the extremities, particularly the legs, are involved. Other sites include the abdominal wall, perianal, groin, and postoperative wounds [52]. Potential portals of entry include trauma site, laparotomy in the setting of peritoneal soiling, any other surgical procedure, perirectal abscess, decubitus ulcers, or intestinal perforation [53,54]. Diabetes, alcoholism, cirrhosis, peripheral vascular disease, and intravenous drug abuse may predispose to necrotizing fasciitis [55,56].

The affected area is initially erythematous, swollen, hot, shiny, exquisitely tender, and painful. Over 3 to 5 days, the skin changes progress from red-purple to patches of blue-gray, then to bullae, frank cutaneous gangrene marked with swelling, and edema may cause a compartment syndrome with complicating myonecrosis requiring fasciotomy. Fever (102° to 105°F) is present and leukocytosis commonly occurs. Blood cultures are frequently positive. With extensive subcutaneous fat necrosis, hypocalcemia can occur. The development of anesthesia over the affected area results from destruction of superficial nerves due to thrombosis of small blood vessels, and may be a clue to differentiating necrotizing fasciitis from a simple cellulitis.

Infectious myositis

Infection of skeletal muscle is uncommon and can be caused by a wide range of organisms: bacteria, mycobacteria, fungi, viruses, and parasitic agents.

Pyomyositis is an acute bacterial infection of skeletal muscle, usually caused by *S aureus*. It usually occurs after a penetrating wound, prolonged vascular insufficiency in an extremity, or a contagious infection. Bacteremic spread of infection to skeletal muscle is extremely uncommon [58]. Pyomyositis is very rare in North America; most cases occur in the tropics. About 40% of cases in temperate climates lack any relevant underlying disease but the remaining cases have possible predisposing conditions such as diabetes mellitus, alcoholic liver disease, corticosteroid therapy, or immunosuppressive disease (ie, leukemia, HIV) [59] (Box 2).

Muscle abscesses are a frequent reason for hospitalization in the tropics [60,61]. The condition often affects overexerted muscles or follows minor blunt trauma, and develops without a penetrating injury or other known portal of entry. It is more common in men than in women, in young than in middle-aged or older individuals, and is caused by *S aureus* in more than 90% of cases. Other causative organisms include *Streptococcus pyogenes*, *E coli*, and Streptococcus pneumonia. Antecedent arbovirus or parasitic infection has been suspected but remains unproven. One or more muscles may be affected, most frequently the thigh and buttocks [60,61], but the paraspinal, deltoid, triceps, and other muscles may also be affected. Staphaureus, Gram-negative bacilli, or *M tuberculosis* most often causes psoas abscesses [62–64]. The muscles initially feel achy or crampy and examination reveals a wood deep induration in the muscle belly. Within

Box 2. Conditions that can mimic pyomyositis

- Hematology/oncology
  - Soft tissue sarcoma
  - Schwannoma
  - Ossifying muscle
  - Metastases (usually adenocarcinoma)
- Rheumatologic
  - Focal myositis
- Miscellaneous
  - Deep venous thrombosis
  - Diabetic Myonecrosis
  - Muscular infarct (sickle-cell, etc.)

2 weeks, if untreated, fluctuation, erythema, then boggy swelling develops. Leukocytosis and eosinophilia are common. The eosinophilia may reflect a concurrent parasitic infection such as trichinosis or cysticercosis. Blood culture is seldom positive; serum muscle enzymes maybe elevated but are frequently normal despite gross muscle destruction. Empyema, suppurative pericarditis, acute endocarditis, or infectious arthritis may develop [23,61].

Crepitant myositis is most often caused by clostridium perfringens; however, other clostridial species and nonclostridial species can cause a crepitant myositis. Peptostreptococcus is the most common etiology of anaerobic streptococcal gangrene, and group A strep can rarely cause a necrotizing myositis [65].

The clinical spectrum of nonpyogenic myositis can range from mild myalgias, more severe myalgias, to acute rhabdomyolysis. The most common causes are viruses (eg, Influenza, dengue, echovirus, and coxsackie); however, infective endocarditis, systemic bacteremias, rickettsioses, and toxoplasmosis must also be considered [65].

Sonography or computed tomography (CT) scanning can guide aspiration, which provides a specific diagnosis of pyomyositis. CT can reveal low-density areas with loss of muscle planes, a central fluid collection, and a surrounding rim of contrast enhancement characteristic of pyomyositis [66]. Magnetic resonance image scanning can demonstrate enlargement of involved muscles and slight increase signal intensity on T_1-weighted images in the involved area with a hypointense central area and surrounding gadolinium-enhanced rim, as well as a diffuse increase in signal intensity on T_2-weighted images with a central high signal intensity fluid collection surrounded by low intensity rim [66].

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