

Skin and Soft Tissue Infections

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Primary skin infections (ie, pyodermas) typically are initiated by some breach in the epidermis, resulting in infection by organisms, such as *Streptococcus pyogenes* and *Staphylococcus aureus*, that colonize the skin. Host-associated factors, such as immunosuppression, vasculopathy, neuropathy, or decreased lymphatic drainage, may predispose to skin infection. The clinical syndromes associated with skin infections are often characteristic and are defined most simplistically by anatomic distribution (Fig. 1). Although often mild and self-limited, skin infections can be more aggressive and involve deeper structures, including fascia and muscle. This article discusses skin and soft tissue infections, including impetigo, hair follicle-associated infections (ie, folliculitis, furuncles, and carbuncles), erysipelas, cellulitis, necrotizing fasciitis, pyomyositis, septic bursitis, and tenosynovitis.

Impetigo

Impetigo is a superficial skin infection that typically presents with multiple vesicular lesions on an erythematous base that eventually crust over. The microbiology of these infections consists primarily of *S aureus* and *S pyogenes*, either alone or in combination. Recent trends have shown an increasing incidence of *S aureus*-associated impetigo. Although usually seen in children aged 2 to 5 years, these infections can occur in individuals of any age. Impetigo is more common in humid and warm climates, but also can occur in cooler climates during the fall and summer months. It usually occurs on exposed parts of the body, including the face, extremities, and scalp. Predisposing factors include skin abrasions; minor trauma; burns; poor

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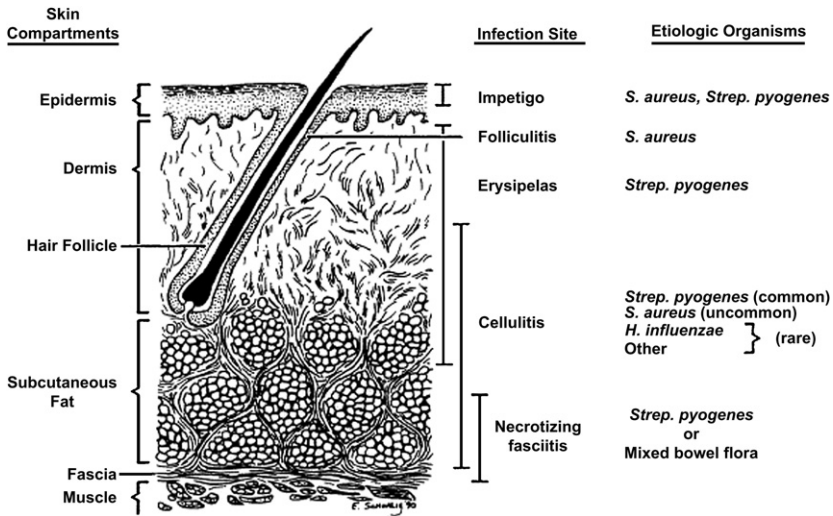


Fig. 1. Cutaneous anatomy, sites of infection, and infecting organisms. (From Feingold DS, Hirschmann JV. Approach to the patient with skin or soft tissue infection. Gorbach SL, Bartlett JG, Blacklow NR, editors. Infectious diseases. 3rd edition. Philadelphia: Lippincott Williams & Wilkins; with permission.)

hygiene; insect bites; diabetes mellitus; primary varicella infection; preexisting skin disease, such as eczema, atopic dermatitis, and cheilitis; hypogammaglobulinemia; and HIV infection [1]. Impetigo also can spread among people who are in close contact with each other (ie, children in day care centers and family members of the same household).

Two types of impetigo are described. The nonbullous form is most common and is more likely to be attributable to a mixed staphylococcal and streptococcal infection. Multiple ultimately vesiculopustular lesions cluster together before skin breakdown occurs with the development of a classic thick honey-colored crust. Regional lymphadenopathy without systemic signs often is present [2]. Pruritus and pain are not common. Bullous impetigo is less common than the nonbullous form and is seen primarily in newborns and infants. The microbiology typically consists of phage type II *S aureus* strains that secrete exfoliative toxin A. Initially vesicular, these lesions ultimately evolve into large yellow fluid-filled flaccid bullae. After rupture, thin light-brown crusts develop. Localized lymphadenopathy is less common than in the nonbullous form, and systemic signs usually are absent.

The differential diagnosis primarily includes herpes simplex virus infections and contact dermatitis. The diagnosis of impetigo usually is based on clinical findings. The identification of the organism from skin lesions or bullae-associated fluid by Gram staining and culture is pursued when the diagnosis is uncertain. Lesions should be de-crusted aseptically before the exudative material beneath is sampled for evaluation. Serologic

antibody studies, such as anti-DNase B or antihyaluronidase B, are not considered routinely in this setting except when the diagnosis of acute poststreptococcal glomerulonephritis is entertained [3].

Topical therapy with mupirocin can be used to treat impetigo when there are a limited number of lesions [4]. When disease is more extensive and severe, oral penicillinase-resistant penicillins (ie, dicloxacillin) and first generation cephalosporins (cephalexin) for a total duration of 10 days are recommended because of their antistaphylococcal activity. As in many other skin-associated infections, the practitioner needs to be aware of the increased prevalence of methicillin-resistant *S aureus* (MRSA) causing infection in the community [5].

Folliculitis

Folliculitis refers to a circumscribed superficial pustular infection of the hair follicle. These pruritic lesions, which are up to 5 mm in diameter, often present as small red papules with a central area of purulence that may rupture and drain. Systemic signs and symptoms are rare. Lesions typically are located on the head, back, buttocks, and extremities. The most common organism isolated is *S aureus*. A condition known as hot tub folliculitis is attributable to hot tub or whirlpool water contaminated with *Pseudomonas aeruginosa*; the ear canal, the breast area, and other skin locations covered by a swim suit are commonly involved. A condition known as eosinophilic pustular folliculitis should be considered in the differential diagnosis of chronic excoriated-appearing follicular infections in HIV-infected patients who have a peripheral eosinophilia and CD4 count of less than 250/mm³ [6,7]. In normal hosts, folliculitis usually resolves spontaneously without systemic treatment, and the use of warm compresses for symptomatic relief and topical antibiotic therapy (ie, mupirocin, clindamycin, erythromycin) may be sufficient. Good hygiene is the best measure to prevent this type of infection.

Furuncles and carbuncles

Furuncles (ie, boils) are single hair follicle-associated inflammatory nodules extending into the dermis and the subcutaneous tissue, usually affecting moist, hairy, friction-prone areas of the body, such as the face, axillae, neck, and buttocks. Firm and tender, these nodular erythematous lesions may spontaneously drain purulent material. Fever and other constitutional symptoms rarely are present. The most common causative microorganism is *S aureus*, but the microbiology of furuncles depends on the location of the lesions. Risk factors for developing this condition include obesity, diabetes mellitus, atopic dermatitis, parenteral drug use, chronic kidney disease, impaired neutrophil function, use of corticosteroids, close exposure to

others who have furuncles (ie, family members, contact sports), and malnutrition [8–11]. When the subcutaneous infection extends to involve multiple furuncles, the lesions are called carbuncles. Carbuncles are often located in the back of the neck, posterior trunk, or thigh. This multiseptate coalescence of multiple abscesses can be painful, and constitutional signs and symptoms, including fever and malaise, often are present. Purulent material may be expressed from multiple draining sinuses.

Severe complications of furuncular infections, such as bacterial endocarditis, have been reported [12]. If systemic involvement of any type is suspected, evaluation should include a complete blood count, blood cultures, and Gram stain and culture of purulent material. Small-sized furuncles usually can be managed by applying moist heat to encourage drainage. Incision and drainage is needed for carbuncles and larger furuncles [4]. Systemic antibiotics usually are reserved for individuals who have systemic signs of infection or associated cellulitis. Surgical consultation often is recommended for patients who have carbuncles.

Recently, a prospective study from 11 university-affiliated emergency departments reported an overall prevalence of MRSA in 59% of 422 patients who presented acutely with purulent skin and soft tissue infections [13]. This finding dictates a consideration of empiric coverage of MRSA in these infections in addition to incision and drainage when antibiotics are indicated [13]. Typically these *S aureus* isolates contain the *PVL* (Panton-Valentine Leukocidin) gene, exhibit a single pulsed-field electrophoretic pattern, and possess a type IV SCCmec element that confers methicillin resistance [14,15]. Patients who have recurrent furuncles may require eradication of *S aureus* from the nares with agents such as mupirocin ointment or oral clindamycin to decrease risk for infection [16].

Erysipelas

An infection of the upper dermis, erysipelas classically presents abruptly as a painful superficial cellulitis with associated fever, regional lymphadenopathy, and lymphangitis. A prodrome of a flu-like illness may precede the appearance of rash by several hours to 2 days. Distinct elevated borders surround brightly erythematous plaques that have no central clearing. Usually involving the lower extremities in more than 75% to 90% of cases, the face also is reported to be involved in 2.5% to 10% of cases [17,18]. Risk factors include tinea pedis, venous insufficiency, obesity, puncture sites, pressure ulcers, and lymphedema [19]. The microbiology associated with this infection is primarily *S pyogenes*. Other organisms reported to be associated with erysipelas include other beta-hemolytic streptococci (ie, Group B, C, or G), *S aureus*, enterococci, and, rarely, Gram-negative bacilli. The diagnosis remains clinically based; about 5% of patients are bacteremic and cultures from the skin are positive in less than 50% of cases [17]. Most patients can be treated as outpatients with oral antibiotics, such as

penicillin, dicloxacillin, or cephalexin. Hospitalization can be considered for more severe cases, including patients who manifest systemic signs and symptoms, such as confusion and hypotension, or who are immunocompromised. Intravenous penicillin, cefazolin, nafcillin, or oxacillin can be considered for treatment of these patients. Rest, immobilization, moist heat, and elevation of the affected area are often recommended as supportive measures. Necrosis of the skin, abscess formation, bursitis, venous thrombophlebitis, osteitis, and septic arthritis are uncommon complications [20]. Recurrences occur in up to 29% of patients and appear more likely in the setting of leg ulcers, skin trauma, and tinea pedis [19,21].

Cellulitis

Cellulitis is a diffuse skin infection that involves the deep dermis and the subcutaneous fat tissues. Unlike erysipelas, which demonstrates superficial skin involvement with distinct margins, cellulitis is diffuse and spreading with no distinct demarcations. Although the inciting event typically is not discernable, predisposing factors include traumatic skin lesions, including puncture wounds, lacerations, surgical wounds, and burns; prior history of cellulitis; dermatophyte infections, such as tinea pedis; pressure ulcers; eczema; dermatitis; vascular insufficiency; lymphedema; presence of a foreign body; malnutrition; and immunosuppressive conditions, such as diabetes mellitus and cirrhosis [22,23]. Any cutaneous surface may be involved, but the most common site of infection is the lower extremity [24,25]. In immunocompetent hosts, the microorganisms most commonly implicated include Gram-positive cocci such as beta-hemolytic streptococci (particularly *S pyogenes*) and *S aureus*. In immunocompromised hosts, other microorganisms need to be included as potential pathogens. For example, Group B streptococcal-associated skin and soft tissue infections should be considered in adults who are pregnant, elderly, diabetic, cirrhotic, or have cancer [26,27]. A necrotic skin lesion with a hemorrhagic halo in a febrile neutropenic patient should include *P aeruginosa* in the microbiologic differential diagnosis. Likewise, *Vibrio vulnificus* infection should be considered in cirrhotic patients who present with hemorrhagic bullous lesions and septic physiology, particularly if there is a recent history of warm brackish water exposure or consumption of undercooked shellfish, such as oysters, that derive from warmer waters [28].

Patients who have cellulitis typically present with erythematous, edematous tender skin lesions that are warm to palpation. Regional lymphadenopathy and lymphangitis may be present. Systemic signs usually are more prominent in patients who have associated bacteremia. The differential diagnosis for cellulitis is broad and includes deep and superficial venous thromboses; gout; herpes zoster; angioedema; contact dermatitis; relapsing polychondritis; local reactions to chemicals, foreign bodies, and insect venom; and lymphedema, among others [29].

The diagnosis typically is determined clinically particularly when the cellulitis is considered uncomplicated (ie, lack of systemic signs, absence of immunocompromising conditions, and minimal skin surface area involved). A recent review of the available reported literature concluded that blood cultures do not assist diagnostics or therapeutics in immunocompetent adults who present with acute cellulitis [30]. Based on one analysis of 553 patients with community-acquired cellulitis, blood cultures are recommended when there is acute onset of infection in the geriatric patient associated with significant leukocytosis and high-grade fever or immunocompromised hosts [31]. Other possible diagnostic modalities for cellulitis include needle aspiration and skin biopsy, especially for patients who have strange exposures, refractory infections, or immunocompromising comorbidities. Unfortunately, cultures yield an organism in less than one third of samples obtained by needle aspiration or biopsy of infected tissue [32–37]. Swabbing ipsilateral tinea pedis-affected areas for bacterial culture may be helpful in identifying the microbiologic cause of cellulitis [38,39]. In general, imaging studies are not indicated in cellulitis unless a complicating factor (ie, foreign body, osteomyelitis, abscess, necrotizing fasciitis) or alternative diagnosis (ie, deep venous thrombosis) is being considered.

Treatment in most cases is directed against streptococci and *S aureus*. In the afebrile, immunocompetent patient who has a small area of involvement, oral antibiotic therapy consisting of dicloxacillin, cephalexin, clindamycin, or erythromycin can be considered if the community antibiotic-resistance profiles are favorable [4]. In immunocompetent patients who are more severely ill, initial parenteral antibiotics that can be used include nafcillin, oxacillin, or cefazolin [4]. The emergence of methicillin resistance in *S aureus* isolates from the community needs to be considered because these organisms are resistant to beta-lactam antibiotics, such as penicillins and cephalosporins. Agents such as trimethoprim-sulfamethoxazole, doxycycline or minocycline, clindamycin, linezolid, vancomycin, and daptomycin often are used to manage community-acquired MRSA skin infections. Immobilization and elevation of involved areas and treatment of underlying predisposing conditions is recommended. Surgical evaluation should be considered when the development of necrosis and gas is suspected or when there is no response to appropriate antibiotics.

Necrotizing fasciitis

Although uncommon, necrotizing fasciitis (NF) is an infectious diseases emergency. Predisposing conditions include diabetes mellitus, surgery, trauma, peripheral vascular disease, and injection drug use. Classification of this life-threatening infection has been proposed based on the microbiology of infection [40]. Type 1 necrotizing fasciitis refers to a mixed aerobic and anaerobic infection (ie, diabetic foot infection and Fournier's gangrene, for example). Facultative streptococci, staphylococci, enterococci, aerobic

Gram-negative bacilli, such as *Escherichia coli*, *Klebsiella*, *Pseudomonas*, *Enterobacter*, and *Proteus*, and anaerobes, such as *Peptostreptococcus*, *Bacteroides*, and *Clostridium* spp are the characteristic pathogens associated with these infections. Type II infections are monomicrobial, classically associated with the “flesh-eating” bacteria *S pyogenes*. Other bacteria capable of producing monomicrobial infection include *Streptococcus agalactiae*, *V vulnificus*, *Clostridium* spp, and *S aureus* [28,41]. Of note, *Aeromonas* spp– and *Streptococcus pneumoniae*–associated NF have been reported in patients who have underlying collagen vascular diseases [42–44].

Usually associated with antecedent skin infection or trauma, NF is rapid in onset with the sequential development of erythema, extensive edema, and severe, unremitting pain, often despite antibiotic therapy. Hemorrhagic bullous lesions, skin necrosis, and crepitus associated with gas in soft tissues also may develop. Systemic toxicity manifest by fever, hypotension, tachycardia, delirium, and multiple organ dysfunction is characteristic of severe cases. Anesthesia localizes at the infection site because of associated nerve necrosis. The legs and other extremities are the most common locations for NF, although any site can be involved, including the perineum, trunk, head and neck, and buttocks [45,46].

Early recognition of NF is essential. Plain films and CT scans can be helpful in demonstrating gas in the soft tissues when infection is attributable to anaerobic bacteria or the Enterobacteriaceae. Magnetic resonance imaging is reported to be more sensitive in identifying necrotizing skin and soft tissue infections and the extent of involvement [47–49]. No laboratory test is specific for the diagnosis of NF. A diagnostic scoring system for distinguishing these infections based on routine laboratory tests has been proposed. Titled the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score, it uses factors that have been reported to be independently predictive of NF (total white cell count, hemoglobin, sodium, glucose, serum creatinine, and C-reactive protein) to create a score to assist physicians in the evaluation of NF [50]. The negative predictive value and positive predictive value of a cut-off score of 6 is reported to be 96% and 92%, respectively. The authors conclude that patients who have an LRINEC score greater than or equal to 6 should “be carefully evaluated for the presence of necrotizing fasciitis” [50]. Tissue oxygen saturation monitoring also has been reported to be helpful in identifying NF of the lower extremities in patients who do not have chronic venous stasis, peripheral vascular diseases, shock, or systemic hypoxia. A cut-off value of less than 70% for the involved tissue oxygen saturation demonstrated a sensitivity of 100% and a specificity of 97% [51].

When NF is suspected, surgical evaluation is needed. Definitive diagnosis of NF is made by visualization of affected fascia. The passage of a probe or finger without resistance across soft tissue that typically is adherent to deep fascial planes is characteristic [52]. Needle aspiration of involved soft tissue or blood cultures may yield the putative pathogen but is not specific for

diagnosing necrotizing skin infections [53,54]. Frozen section full-thickness biopsy performed early in the course of infection may provide histopathologic confirmation of NF and prompt definitive surgical debridement [55]. Culture of intraoperatively-obtained tissue is most helpful in determining the microbial cause.

Surgery is not only diagnostic but also therapeutic. It is intended to preserve viable skin while removing necrotic tissue and ensuring hemostasis. Repeat intervention is recommended and multiple debridements may be required until evolving necrosis is no longer appreciated. Plastic surgical reconstruction with split-thickness skin grafting often is needed. In addition to surgery, polymicrobial-associated NF should be treated with a broad-spectrum antibiotic regimen consisting of ampicillin-sulbactam or piperacillin-tazobactam and ciprofloxacin and clindamycin [4]. Group A streptococcus-associated NF is best treated with penicillin plus clindamycin. The potential presence of *S aureus* may dictate the need for vancomycin because of the emergence of methicillin resistance in this organism. Overall mortality rates range from 17% to 40% and are increased when there is a delay in surgery or when toxic shock syndrome with NF secondary to *S pyogenes* is present [45,46,54,56].

Pyomyositis

Pyomyositis refers to purulent infection deep within a striated muscle group, often manifesting as an abscess. Infection does not result from a contiguous site but rather secondary to hematogenous seeding of injured muscle. Although usually involving one muscle group, multiple sites may be infected simultaneously in approximately 15% of cases [57]. Most commonly targeting the thigh, pyomyositis also can occur in the calf, upper extremity, gluteal, chest wall, and psoas muscles [57]. The incidence of this rare infection seems to be increasing in areas other than the tropics where it was originally described and it seems to be associated with immunocompromising conditions, such as HIV infection, malnutrition, concomitant parasitic or viral infection, diabetes mellitus, intravenous drug use, immunosuppressive drugs, rheumatologic diseases, cirrhosis, and malignancy [58,59]. Patients initially present with fever, swelling, and crampy pain at the involved muscle site before developing edema, tenderness, and “woody” induration. Diagnosis depends on a high index of clinical suspicion, and if not considered and addressed the infection can progress to sepsis and shock.

S aureus is the causative organism in most cases (ie, 75%–90%) [60]. Other organisms reported to cause pyomyositis include *S pyogenes* and other beta-hemolytic streptococci, *S pneumoniae*, *Haemophilus influenzae*, Gram-negative bacilli, anaerobes, mycobacteria, and fungi [61,62]. Blood cultures are positive in 5% to 30% of cases and are more likely to yield an organism in temperate climates [57,62,63]. Aspiration, Gram-stain, and culture of abscess material also may yield the causative organism.

Laboratory values are nonspecific, often revealing a neutrophilia, an elevated erythrocyte sedimentation rate, and normal aldolase and creatinine kinase levels. In the tropics, an eosinophilic leukocytosis is characteristic. Radiographic imaging is the most helpful noninvasive diagnostic test. Ultrasound, CT, and MRI (with gadolinium administration) are helpful, although the latter is most helpful in defining the extent of infection [64].

Once abscesses have formed, treatment consists of adequate drainage and antibiotics. Empiric antimicrobial therapy initially is broad in spectrum, particularly when the patient is immunocompromised, and should include anti-*S aureus* coverage. Pathogen-directed therapy is administered once culture and susceptibility testing results are available. Drainage can be performed percutaneously under ultrasound or CT guidance, but surgical drainage may be needed when infection is extensive or necrotic [59]. The usual duration of antibiotics is 3 to 6 weeks with the exact duration dictated by the patient's clinical course and serial radiographic imaging [59,62]. The reported mortality rate is 1% to 6% and patients usually recover with minimal sequelae [57,62].

Septic bursitis

Bursae are small sac-like cavities that contain fluid and are lined by a synovial membrane. They are located subcutaneously between bony prominences and tendons or in deeper fascial tissue between bone and muscle, essentially serving to reduce friction between these structures. Inflammation of bursae (ie, bursitis) can be caused by infection, particularly when the superficial subcutaneously located bursae are involved. These infections are typically secondary to local trauma and rarely attributable to hematogenous seeding. In addition to prior history of trauma to superficial bursae, other predisposing factors may include prior rheumatoid arthritis or gouty involvement of the bursae, alcoholism, diabetes mellitus, renal insufficiency, intravenous drug use, and other forms of immunosuppression [65,66]. Trauma often is occupationally associated, favoring situations that place pressure on the olecranon, prepatellar, and superficial infrapatellar bursae, the most common sites of septic bursitis [67,68].

Peribursal cellulitis, warmth, and erythema are common. Close inspection of the skin also may reveal lacerations, abrasions, and ecchymoses [67]. Fever and pain with movement of the associated joint also is characteristic of superficial bursitis. Interestingly, Smith and colleagues [69] have reported that in septic olecranon bursitis, a temperature of greater than or equal to 2.2°C between the affected and unaffected olecranon processes was 100% sensitive in distinguishing septic from nonseptic bursitis.

Like many other inflammatory conditions, a peripheral leukocytosis, an elevated C-reactive protein, and an elevated erythrocyte sedimentation rate are often present. Although radiographic evaluation may reveal abnormal accumulations of fluid, the presence of a foreign body, and the extent of

infection, the diagnosis of septic bursitis requires sampling of the bursal fluid. The range of leukocytosis per mm^3 is broad and the absolute number usually is less than is present in septic arthritis. In one series of 32 patients with septic bursitis, the bursal fluid leukocyte count averaged $23,350/\text{mm}^3$ with a standard deviation of $22,065/\text{mm}^3$; more than 10% of patients had fewer than 2000 leukocytes/ mm^3 [65]. Gram stain is positive in 50% or less of all cases [70]. Sensitivity of culture may be increased by directly inoculating bursa-associated fluid into liquid media versus direct culture methods [71]. Blood cultures also may be helpful in identifying the microbiologic cause of infection in deeper and more severe forms of septic bursitis [65]. More than 75% of septic bursitis cases are attributable to *S aureus*; other causes include streptococci, *Staphylococcus epidermidis*, enterococci, diphtheroids, and, rarely, Gram-negative bacilli [72]. Subacute and chronic infectious bursitis can be attributable to other organisms such as, *Brucella*, mycobacteria, *Prototheca*, and *Aspergillus*.

Treatment typically requires drainage and antibiotics. Initial antibiotics are dictated by Gram-stain evaluation of bursal fluid. If the Gram stain is negative, antistaphylococcal therapy is initiated with intravenous cefazolin, nafcillin or oxacillin, or vancomycin. Subsequent treatment then is dictated by culture and susceptibility results. Daily percutaneous drainage procedures are performed until sterility is attained and antibiotics usually are administered for at least 2 to 3 weeks. Indications for surgery include inadequate needle drainage of bursa, inability of needle to access bursa for drainage, necrosis, presence of foreign body, and refractory infection [67].

Infectious tenosynovitis

Infection of the synovial sheaths that surround a tendon is known as tenosynovitis. The flexor muscle-associated tendons and tendon sheaths of the hand are most commonly involved (flexor tenosynovitis). Penetrating trauma is the most common inciting event and infection then can travel rapidly from the digit to ulnar and radial bursae. Acute infection is most commonly attributable to *S aureus* and other skin-associated flora, such as streptococci, although the nature of the traumatic injury may impact the associated microbiology (ie, organisms associated with mammal bites, gardening, or fish handling) [59]. Nontraumatic causes of tenosynovitis are classically associated with disseminated gonococcal infection (DGI). In DGI, one or several tendons may be involved (extensor more often than flexor), usually targeting the ankles, toes, wrists, and fingers [73]. Chronic infectious flexor tenosynovitis is caused primarily by fungi and mycobacteria.

Patients who have acute flexor tenosynovitis present with erythematous fusiform swelling of an involved finger, which is held in a semiflexed position. Tenderness over the length of the tendon sheath and pain with extension of the finger also are typical [74]. Classically, patients who have DGI have fever, rash, tenosynovitis, and an asymmetric polyarthritis. The rash

initially is macular, papular, or petechial before progressing to a pustular stage that can become hemorrhagic and necrotic. The lesions characteristically are few in number (ie, <20–30) and are distributed periarticularly over distal extremities.

Patients in whom there is clinical suspicion of flexor tenosynovitis should be evaluated by a hand surgeon and receive empiric antibiotics. The nature of the exposure and the underlying status of the host dictate the antimicrobial selection. Infection in an immunocompetent host resulting from a simple puncture would require at least Gram-positive skin flora coverage against staphylococci and streptococci. Pet bite-associated infectious tenosynovitis dictates expanded coverage to include Gram-negative organisms, such as *Pasteurella* and anaerobes. Imaging of the involved area, particularly with MRI, may be helpful in identifying the presence of gas, bone infection, foreign body, or fluid, and the extent of infection, but a definitive diagnosis requires evaluation of fluid from within the tendon sheath. Although some patients who present within 48 hours of developing trauma-associated flexor tenosynovitis can be treated with intravenous antibiotics and splinting alone, effective surgical drainage coupled with pathogen-directed therapy established by microbiologic evaluation of drainage material typically is required. DGI-associated tenosynovitis can be diagnosed with blood cultures. In addition, the organism may be detected in mucosal sites, such as the rectum, oropharynx, cervix, and urethra, and in the synovial fluid (when arthritis is present). Initial therapy with ceftriaxone intramuscularly or intravenously and hospitalization are recommended for patients who have DGI. Unless the diagnosis is excluded, treatment of concomitant chlamydial infection also should be administered. Intravenous therapy can be switched to oral antibiotics, such as cefixime, 24 to 48 hours after improvement begins to complete at least one week of treatment [75].

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