

Hand Infections

Orrin I. Franko, MD^a, Reid A. Abrams, MD^{b,*}

KEYWORDS

• Hand infections • Paronychia • Tenosynovitis • Animal bites

KEY POINTS

- Acute hand infections are most commonly caused by *Staphylococcus* spp and *Streptococcus* spp, with an increasing prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA).
- Empiric antibiotic coverage should be withheld until cultures have been obtained. Then it should begin with broad-spectrum treatment directed by the injury environment and mechanism, including strong consideration for coverage of MRSA.
- The cornerstone of surgical treatment includes incision, drainage, debridement, and irrigation followed by daily dressing changes.
- Herpetic whitlow is a viral infection that may mimic acute bacterial infections and typically resolves spontaneously without treatment.
- Flexor tenosynovitis can be diagnosed by the classic signs of fusiform swelling, flexed resting posture, flexor sheath tenderness, and pain with passive extension.
- Septic arthritis of the wrist is typically diagnosed by a joint aspirate white blood cell count greater than 50,000 with 75% polymorphonuclear lymphocytes. It should be treated as soon as possible.
- Bite injuries should always be explored for involvement of an underlying joint or tendon, especially human bite clenched fist injuries over the metacarpal head.
- Necrotizing fasciitis is a life-threatening condition with a mortality rate of 33% that requires emergent surgical debridement.

INTRODUCTION

Infections of the hand are present in all communities, but prevalence is dependent on patient factors such as immunodeficiency (HIV, diabetes, malnutrition) and exposure (occupation, intravenous drug use). The epidemiology of cultured organisms has demonstrated a trend toward increasing rates of methicillin-resistant *Staphylococcus aureus* (MRSA) species, although many infections are polymicrobial or culture-negative.¹ Rapid treatment of these infections with appropriate surgical decompression, debridement, and antibiotics, followed by wound care and hand therapy is required to minimize or prevent lasting sequelae.

MICROBIOLOGY

The most common hand pathogens are *S aureus*, *Streptococcus* spp, and gram-negative species. *Staphylococcus* is the primary organism in 50% to 80% of infections.² MRSA species are becoming more prevalent both in community and hospital settings with current rates as high as 78%.³⁻⁷ Industrial and home-acquired infections routinely involve a single gram-positive organism, whereas infections from intravenous drug use, bites, mutilating farm injuries, and those associated with diabetes mellitus are generally polymicrobial with gram-positive, gram-negative, and anaerobic species.^{2,8-12} Some of the most

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^a University of California, San Diego School of Medicine, San Diego, CA, USA; ^b Division of Hand and Microvascular Surgery, Department of Orthopedic Surgery, University of California, San Diego School of Medicine, 200 West Arbor Street 8894, San Diego, CA 92103, USA

* Corresponding author.

E-mail address: raabrams@ucsd.edu

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common risk factors for various bacteria include the following:

- MRSA infections are more common in patients with diabetes mellitus, immunocompromised patients, intravenous drug abusers, prisoners, and homeless individuals⁵⁻⁷
- Alpha-hemolytic streptococcus and *S aureus* are the most common pathogens in human bite infections^{2,9,10}
- *Eikenella corrodens* is isolated in approximately one-third of human bite wounds^{2,9}
- *Pasteurella multocida* commonly infects animal bite and scratch wounds¹³
- Necrotizing fasciitis can be caused by group A streptococcus alone or infections can be polymicrobial, involving alpha-hemolytic and beta-hemolytic streptococci, *Staphylococcus* spp, and anaerobes¹⁴
- Fungi and atypical mycobacterium cause chronic indolent infections.

ANTIBIOTICS

Empiric antibiotics should be administered after performing cultures and Gram stain. Initial antibiotics should be aimed only at suspected organisms because overly broad coverage can select for resistant organisms, is costly, and needlessly exposes patients to side effects. However, increasing rates of MRSA suggest that empiric treatment to cover methicillin-resistant organisms is routinely advised, particularly in urban regions.¹

Box 1 serves as a guide for the selection of appropriate antibiotic treatment.

Box 1

Antibiotic selection guide

- Cefazolin and penicillin G: intravenous antibiotic coverage for aerobic and anaerobic pathogens in serious infections requiring incision and drainage and hospitalization¹⁵
- Gentamicin (gram-negative coverage): if intravenous drug abuse is involved or the patient is diabetic^{11,15}
- Vancomycin: the drug of choice for cases of methicillin resistant MRSA. Occasionally MRSA is sensitive to Septra, quinolones, tetracyclines, or rifampin; however, rifampin should never be used as a single agent
- Penicillin G (parenteral) or first-generation cephalosporin: empiric coverage for human bites; however, some investigators also recommend aminoglycoside coverage^{2,10}

- Piperacillin-tazobactam or ampicillin-sulbactam: good initial coverage for human and animal bites
- High doses penicillin G and aminoglycoside: empiric coverage for necrotizing fasciitis¹⁴
- Clindamycin: used for both anaerobic coverage and, in the case of hemolytic streptococcal infection, to stop toxin production

PATIENT FACTORS

Immunosuppressive drugs, acquired AIDS, and diabetes mellitus predispose patients to hand infections. In diabetic hand infection, treatment is often delayed, resolution slowed, repeated debridement is often necessary, and amputations are frequent (20%–63%) to control infection or because of poor function.^{11,16} Hand infections in AIDS patients are typically from routine pathogens with atypical presentations and unusually virulent courses. Herpetic lesions are unusually dire and often require antiviral treatment.¹⁷ Diabetes, alcohol, and intravenous drug abuse are risk factors for necrotizing fasciitis.¹⁴

INCISION AND WOUND MANAGEMENT

Successful management of hand infections pre-dates antibiotics. Incision and drainage remains the basis of treatment. Straight incisions are preferred to avoid flap necrosis. Placement over tendons or neurovascular structures is discouraged. Highly contaminated wounds should be left open and dressed with moist gauze and a splint, immobilizing digits in the intrinsic-plus posture and maintaining the breadth of the first web space. Sometimes repeated debridement is necessary until the wound is clean enough for delayed closure or healing by secondary intention. Wound closure over irrigation or suction drainage is sometimes feasible. Vacuum-assisted closure therapy (also known as negative pressure wound therapy) has transformed the management of large open wounds arising from extensive debridement, such as in necrotizing fasciitis.^{18,19} Wounds should be reexamined at 24 to 48 hours. Dressings are changed daily thereafter. Incorporating whirlpools or soaks can help encourage continued drainage. Hand therapy is initiated after inflammation and swelling have begun to resolve, usually before the wound has healed.

MANAGEMENT OF INFECTIONS UNIQUE TO THE HAND

Paronychia

An acute paronychia is an abscess beneath the eponychial fold.²⁰ It can remain superficial to the

nail plate, localized to the radial or ulnar side, or the infection can spread transversely around the entire nail fold. The abscess can track proximally around and deep to the nail plate between the nail and matrix.²⁰ Manicures, artificial nails, nail biting, or hangnails are common causes. Eponychial swelling, tenderness, erythema, and drainage are characteristic. *S aureus* is the most common pathogen, followed by *Streptococcus pyogenes*, *Pseudomonas pyocyanea*, and *Proteus vulgaris*.²¹

Although early infections can be treated with oral antibiotics alone, the safest approach is drainage whenever an abscess is present under a digital block. When possible, the nail should be elevated and retained; however, if necessary, it should be removed fully or partially to achieve adequate decompression. An incision across the eponychial fold should be avoided to prevent late nail fold deformity. Soaks can be started immediately and antibiotics should be administered for at least 5 to 7 days, or until resolution of the infection.²²

If an abscess is not present, nonoperative treatment with antibiotics is performed in conjunction with warm soaks 3 to 4 times per day.^{21,23,24} One study examined the use of topical antibiotics in a retrospective fashion and concluded that gentamicin antibiotic alone was superior to a combined steroid-antibiotic ointment.²⁴ When oral antibiotics are selected, options include amoxicillin with clavulanic acid (Augmentin); however, in areas with high rates of MRSA infections, clindamycin or trimethoprim-sulfamethoxazole (Bactrim, Septra) may be first-line agents.²⁵⁻²⁷

Chronic paronychias can result from excessive exposure to moisture; they are characterized by intermittent periods of inflammation around the eponychium. Eventually there is separation of the nail fold from the underlying nail plate. A cheese-like drainage may exude from beneath the eponychium. *Candida albicans*, atypical mycobacteria, and gram-negative bacteria have all been implicated.^{2,20} Treatment is challenging. The most successful interventions are marsupialization or total nail removal.²⁰ Adjunctive topical steroid-antifungal ointment (3% Vioform in Mycolog) or oral antifungal medications have been recommended (itraconazole, fluconazole).²⁰

Felon

Felons are painful abscesses in the digital pulp, typically following a puncture wound. Tense distension can compromise soft tissue and distal phalanx vascularity. The most common organism

is *S aureus*.²⁰ Expeditious drainage is necessary to prevent digital pad necrosis, distal phalanx osteomyelitis, or flexor tenosynovitis. Fish-mouth incisions should be avoided because they can compromise pulp vascularity.²⁰ The point of maximal tenderness guides incision placement with high lateral (made just below the fingernail) and midvolar configurations preferred.²⁰ Proximal probing can inoculate the flexor sheath and should be avoided. The wound should be dressed with loose gauze packing, which is removed in 24 to 48 hours, followed by warm daily soaks and gauze dressing changes until healing by secondary intention.

Herpetic Whitlow

Herpetic whitlow is a viral infection of the digital tip caused by herpes simplex virus (HSV), resulting from exposure to genital (HSV-2) or oral (HSV-1) lesions. A painful cytolytic infection occurs 2 to 14 days after contact, usually maturing in 14 days. Viral shedding, and the risk of infecting others, persists until lesion epithelialization is complete.²⁸ As the infection subsides, the virus becomes latent, retreating to the sensory ganglia, avoiding immune clearance.²⁸ The natural history of the infection results in complete resolution without treatment, usually in 3 weeks.²⁹ The diagnosis of herpetic whitlow is made based on history and clinical examination. Patients may present with flu-like prodromal symptoms before the lesion appears. The lesion is often preceded with throbbing pain, tingling, or numbness at the lesion site. A single vesicle and/or additional vesicles may coalesce as a single larger bulla with clear vesicular fluids that can be often mistaken for a pustule. Unlike a felon, herpetic whitlow vesicles do not appear on the pulp of the digit. Nonoperative treatment is the best option because, typically, the vesicles unroof, form ulcers, crust, and flake off over 2 to 3 weeks.

The disease demonstrates a bimodal age distribution that usually effects children less than 10 years of age (HSV-1) or young adults between 20 and 30 years (HSV-1 or 2).³⁰ Although diagnosis is typically by history and physical examination, it can be confirmed with cultures of the vesicular fluid, Tzanck smear, direct fluorescent antibody testing, or a rise in serum antibody titers. Superinfections can follow unnecessary incision and drainage and, for this reason, it is important not to mistake the clear vesicular fluid for pus. Acyclovir, famciclovir, or valacyclovir may benefit patients troubled by frequent recurrences, to abort recurrent infections in patients who have a prodrome, decrease the clinical course in

particularly protracted cases, and they are usually necessary to induce remission in patients with AIDS.^{28,31}

Flexor Tenosynovitis

Pyogenic flexor tenosynovitis results when a puncture wound inoculates the flexor sheath. The sheath is a mesothelium-lined closed space, extending from the proximal end of the A1 pulley to the distal interphalangeal joint. The thumb sheath is contiguous with the radial bursa and the small finger sheath is contiguous with the ulnar bursa. The radial and ulna bursae extend proximal to the carpal tunnel. In 50% to 80% of individuals, the radial and ulnar bursae communicate (Fig. 1).² *S aureus*, *Streptococcus* spp, and gram-negative organisms are the most common pathogens. Less often, chronic indolent infections, characterized by abundant tenosynovitis, can be caused by fungi and atypical mycobacterium.³² The classic clinical symptoms of a septic or pyogenic flexor tenosynovitis was described by

Kanavel³³ and includes fusiform swelling, sheath tenderness, flexed resting posture, and pain on passive extension of the involved digit. Typically, patients with a pyogenic flexor tenosynovitis will have most, but not necessarily all, of these signs (Box 2).

Box 2 **Kanavel signs**

- Flexed resting posture of the involved digit
- Flexor sheath tenderness
- Digital fusiform swelling
- Pain on passive digital extension²

Treatment should be instituted expeditiously because a delay can result in tendon vascular compromise and necrosis, adhesions, or extension into adjoining deep spaces (Box 3).

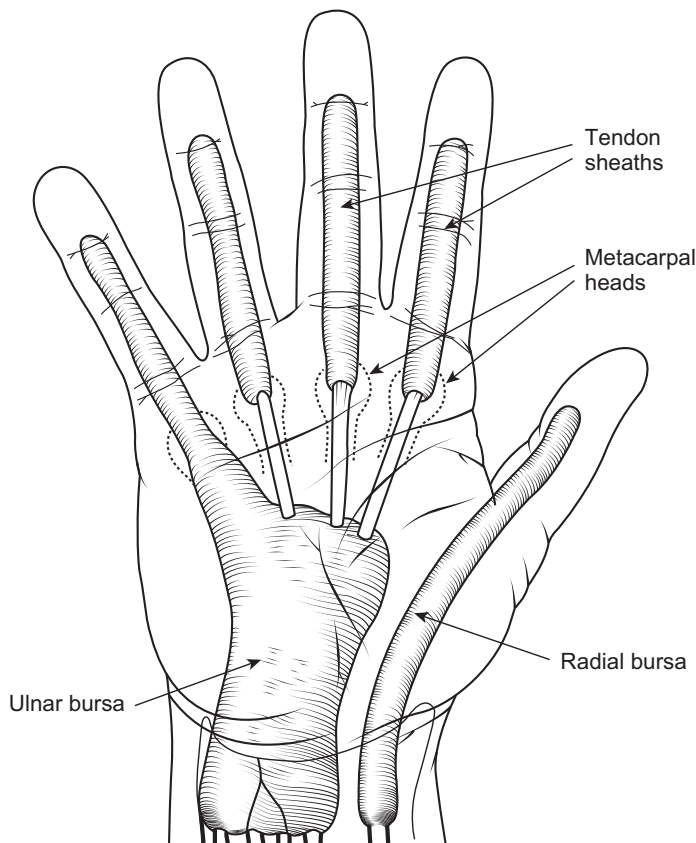


Fig. 1. Anatomy of the digital flexor sheaths, radial, and ulnar bursae. The small finger sheath is contiguous with the ulnar bursa and the thumb flexor sheath is contiguous with the radial bursa. The proximal extents of the radial and ulnar bursae are the carpal tunnel and Parona space and, in 5% to 80% of persons, the two bursae communicate.

Box 3**Deep space infection sources**

- Horseshoe abscess: infection of the thumb flexor sheath can spread through communication of the radial and ulnar bursae extending into the small finger sheath
- Parona space: extensive proximal spread from any digit can lead to involvement of Parona space
- Thenar space: proximal spread from the index finger flexor sheath can cause infection of the thenar space
- Midpalmar space: proximal extension from the long or ring finger sheaths

Early infections (within 24 hours) can be treated with observation, elevation, splinting, and intravenous antibiotics.^{2,34} Surgical drainage is prudent if there is no improvement within 24 hours or if initial presentation is delayed 24 hours or more beyond the onset of symptoms. A popular treatment method is limited incisions at the proximal and distal ends of the flexor sheath, catheter insertion into one end, and either continuous or intermittent through-and-through irrigation. Extravasation is common with this technique probably due to sheath erosions or inaccurate catheter placement resulting in digital swelling and stiffness. This can be avoided by adding a midlateral incision, which facilitates catheter placement and better drainage, especially when the infection consists of viscous purulent material. In digits compromised by infection, zigzag Bruner incisions should be avoided to prevent flap tip necrosis and poor tendon coverage.

Recommended treatment includes a proximal incision made just proximal to the A1 pulley, near the distal palmar crease. For the midlateral approach, an incision is made from the metacarpophalangeal joint flexion crease to the distal

interphalangeal joint flexion crease. An angio-catheter or No. 5 pediatric feeding tube is placed in the sheath for irrigation. After thorough irrigation, the catheter can be removed, or left for continuous irrigation. After 48 hours, the wet dressing and catheter are removed and digital motion is started. Wounds are left open and they typically heal rapidly by secondary intention.

Deep Space Infections

The deep spaces (**Fig. 2**) of the hand include the thenar, midpalmar, Parona, and the interdigital web spaces.³⁵ Deep space infections arise from penetrating inoculation or contiguous spread and, rarely, hematogenous seeding. *S aureus*, streptococci and coliforms are the common pathogens.²

Thenar space infections can result from contiguous proximal spread from index finger flexor tenosynovitis. Infection can spread around the distal edge of the adductor pollicis and first dorsal interosseous muscles to involve the dorsal first web (or in the interval between the adductor pollicis and first dorsal interosseous) causing a pantaloon-shaped abscess.³⁶ The first web becomes markedly swollen and the thumb rests in palmar abduction because thenar space volume is largest in this thumb position. Dorsal, palmar, combined dorsal and palmar, and transcommissural³⁶ incisions have been described, with many surgeons preferring the two-incision approach.^{2,32} Incisions paralleling the first web commissure are discouraged to avoid web contracture.

Direct penetration or contiguous spread from the flexor sheaths of the middle two fingers may cause infection of the deep palmar space.³⁶ The palm becomes markedly tender and swelling obscures the normal concavity. Dorsal hand swelling may be so impressive that the palmar process may be mistaken for a dorsal infection.

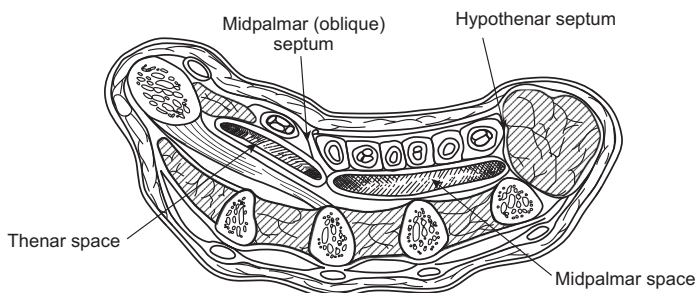


Fig. 2. Cross section of the midpalmar and thenar spaces through the midpalm.

Passive motion of the middle two fingers elicits pain. Transverse or oblique palmar incisions have been routinely used for drainage.

Parona space is bordered by the pronator quadratus, digital flexors, flexor pollicis longus, and flexor carpi ulnaris. Involvement of this space usually results from contiguous spread from the radial or ulnar bursae.³² Symptoms may include acute carpal tunnel syndrome and pain with finger flexor motion. Management is similar to the other deep space infections, emphasizing wide exposure and thorough drainage, avoiding placement of incisions directly over the flexor tendons and median nerve to avoid desiccation.

Old-fashioned collar buttons were dumb-bell shaped, securing the collar closure by passing each end through overlapping buttonholes. Collar button abscess describes a web space infection that spreads palmarly and dorsally through a narrow fascial hole in the middle. It usually results from contiguous spread of an infected palmar blister, skin fissure, or puncture. Due to adherence of the palmar skin and underlying fascia, instead of spreading peripherally, the infection is forced to expand dorsally through a perforation in the palmar fascia (distal to the bifurcation of the neurovascular bundle) to involve the subcutaneous tissue of the dorsal web.^{2,36} The involved web is swollen and the adjacent digits are abducted. A palmar incision can be used with excision of the palmar fascia in the interdigital space, which allows drainage of the palmar and dorsal extension of the infection, or palmar and dorsal incisions can be used.³⁶ Transverse incisions should be avoided to prevent a web contracture.

After adequate debridement of deep space abscesses, wounds can be loosely approximated over an irrigating catheter. If there is any doubt regarding the adequacy of the drainage or tissue viability, the wound should be left open for daily wet loose gauze packing dressing changes and possibly repeat trips to the operating room.

Septic Arthritis

Hand joint infections are typically caused by penetrating injuries, extension from contiguous infections, or from hematogenous spread. Bacterial toxins, proteolytic enzymes, bacteriocidal enzymes of synovial and reticuloendothelial origin, and proteoglycanolytic enzymes of cartilaginous origin are released and mediate cartilage destruction.³⁷ Increased intraarticular pressure impedes synovial perfusion and can

result in cartilage damage, capsular and bony erosion, sinus formation, and osteomyelitis.³⁷

The most common bacteria for septic arthritis in the hand include *S aureus* and beta-hemolytic streptococcus.^{2,37} *Hemophilus influenza* should be considered in unvaccinated young children. *Neisseria gonorrhoeae* should be considered in sexually active patients with atraumatic septic arthritis.^{2,37} *S aureus*, gram negative rods, anaerobes, polymicrobial infections, positive blood cultures, and osteomyelitis portend a poor outcome.³⁷

Examination reveals a painful, swollen, and erythematous joint that assumes the posture that accommodates maximal joint volume. Motion, active or passive, and axial loading is painful. A puncture wound may be identified. Hematogenous spread is suspected in patients with systemic symptoms. Crystalline arthropathies may present similarly. It has been demonstrated that serum white blood cell count, erythrocyte sedimentation rate, and C-reactive protein are not particularly useful for diagnosing a septic joint.³⁸ If possible, the involved joint should be aspirated and sent for culture, Gram stain, cell count, and crystal analysis (Box 4).

Box 4

Characteristic fluid for joint sepsis

- A friable mucin clot
- A white blood cell count of more than 50,000 of which more than 75% are polymorphonuclear lymphocytes
- A glucose ⁴⁰40 mg less than the fasting blood glucose^{2,37}
- Gram stain may not show organisms³⁷

Of note, the threshold white blood cell count of 50,000 has been examined more closely in recent years with newer studies suggesting that sensitivity for detecting septic arthritis of the wrist is as low as 47% to 61% and that a value of 17,500 cells may increase sensitivity to 83% with a specificity of 67%.^{39,40} Treating joint sepsis with serial aspirations is unreliable in the hand.³⁷ Definitive incision and drainage evacuates the offending exudate, allows removal of pannus and necrotic debris, and diminishes intra-articular pressure. Studies have demonstrated that septic wrists drained more than 16 hours after presentation had worse results compared with those surgically treated within 10 hours (Box 5).⁴¹

Box 5**Incision locations for drainage**

- Wrist: incisions should be placed dorsally, usually entering between the third and fourth dorsal wrist compartments
- Carpometacarpal joints: incisions should be dorsal and adjacent to extensor tendons
- Metacarpophalangeal joints: incisions should be placed dorsally, entering the joint proximal to the sagittal bands preserves their function and prevents common extensor tendon subluxation
- Interphalangeal and thumb metacarpophalangeal joints: a midaxial incision is preferred, entering the joint between the collateral ligament and the volar plate,^{2,37} protecting the extensor mechanism and avoiding septic boutonniere and mallet deformities

Postoperatively, the wound can be loosely closed over an irrigation catheter or left open and closed later. The hand is splinted for 24 to 48 hours in a functional position, after which drains and splints are removed and motion is encouraged. Parenteral antibiotics are continued until resolution of local and systemic signs. Thereafter, parenteral or oral antibiotics are given for the duration of 2 to 4 weeks after the initial debridement.³⁷

Bites from Humans and Other Animals

A true human bite wound is rare compared with the more typical clenched fist injury, which usually presents with a wound over the metacarpal head. When the metacarpophalangeal joint is examined in extension, the wound may appear innocuous due to retraction of lacerations in the extensor mechanism and joint capsule proximal to the skin laceration. Radiographs may show a fracture, foreign body (eg, a tooth) or osteomyelitis.

Group A streptococcus, *S aureus*, and *E corrodens* are the most common strains isolated from human bite wounds.⁹ *E corrodens* is associated with 7% to 29% of human bite infections and is variably susceptible to cephalosporins and not susceptible to penicillinase-resistant penicillins.⁹ *Bacteroides* spp have been the most commonly isolated anaerobes, usually associated with mixed cultures.

Animal bites are most frequently inflicted by dogs, cats, and rodents, often involving the hand.⁴² Dog bites account for 80% to 90% of domestic animal bites. Only 2% require hospitalization and they rarely become infected, whereas up to 50% of cat bites may become infected with up to 37% requiring admission.⁴³⁻⁴⁷ *P multocida*,

Staphylococcus spp, *Streptococcus* spp, and some anaerobes are the usual pathogens.^{42,43} A hallmark of *P multocida* infection is rapid onset and intense cellulitis, with symptoms occurring sometimes within hours. Empiric antibiotics typically include amoxicillin-clavulanate or penicillin V. Patients who are allergic to penicillin can be treated with alternative antibiotics, including doxycycline, tetracycline, or ciprofloxacin.^{48,49}

Acute uninfected bite wounds should be extended, explored and debrided. Of note, cat bites tend to be caused by longer, slender teeth causing puncture wounds that seal off and form abscesses quickly, thus necessitating open debridement. Dog bites, on the other hand, are more likely to cause lacerations that stay open by themselves.⁴⁴ If a joint is entered, splinting, elevation, and hospitalization with parenteral antibiotics have been recommended for 48 hours.¹⁰ In the setting of an uninfected wound in which no tendon or joint was injured, debridement and antibiotics did better than debridement alone with no significant difference in outcome if the antibiotics were given intravenously or orally.⁵⁰

Infected bites require hospitalization, debridement, and intravenous antibiotics. Often, repeat debridement at 48 hours is prudent. Warm soaks or whirlpools and wet gauze dressing changes are started 24 to 48 hours after debridement.¹⁰ Generally, tendon repairs are best delayed and wounds are left open to heal by secondary intention.³²

Necrotizing Fasciitis

Necrotizing fasciitis is a life-threatening and limb-threatening soft tissue infection involving the fascial layer. It occurs primarily, but not exclusively, in the indigent and in abusers of drugs and alcohol.¹⁴ The extremities are the most frequently involved areas of the body. In one study, 63% of cases arose out of intravenous drug abuse.¹⁴ In about half of cases, one organism was isolated and, most frequently, it was group A streptococcus.¹⁴ Otherwise, infections were polymicrobial, involving alpha-hemolytic and beta-hemolytic streptococci, *S aureus*, and anaerobes.^{14,51}

Necrotizing fasciitis presents with a spectrum of clinical findings that includes painful and rapidly advancing cellulitis with tensely swollen and shiny skin. Radiographs may demonstrate gas in the tissue.⁵² Bullae and ecchymoses appear within days. Systemic signs may initially be lacking, although a leukocytosis is consistently present.¹⁴ Azotemia in the absence of shock or hypotension is an early tipoff to streptococcal toxic shock syndrome, which frequently accompanies necrotizing fasciitis due to the hemolytic streptococci.

With disease progression, hemodynamic instability in the face of an otherwise trivial-appearing cellulitis should increase suspicion for necrotizing fasciitis. The definitive diagnosis is made at surgery when fibrinous necrotic fascia is found accompanied by liquefaction of the subcutaneous fat and a characteristic foul-smelling thin fluid referred to as dishwater pus (Fig. 3). There is usually thrombosis of subcutaneous vessels and, depending on the stage of the disease, the skin may or may not appear viable. The muscle is usually spared. Wide debridement of the involved fascia and necrotic skin is the definitive treatment. Due to the tendency for rapid advancement of the infection, the authors' strategy is to first identify the proximal extent of the involved fascia through exploratory incisions and to start debridement at this level and progress distally. Cultures and Gram stain are taken at the first opportunity and antibiotics are started emergently. Skilled supportive medical care is imperative, often requiring ICU management. Empiric antibiotic treatment should be started early with vancomycin and clindamycin. The addition of clindamycin is important for suppression of the toxins produced by strep and staph species.^{52,53}

Mortality has ranged from 8.7% to 33%. The single-most important factor influencing morbidity

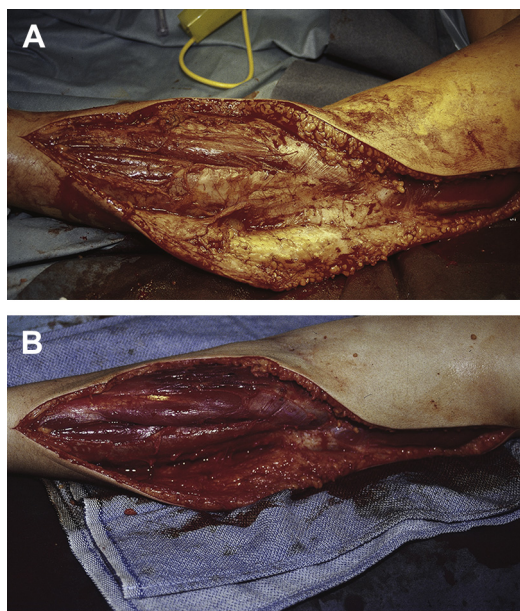


Fig. 3. Intraoperative photos of a patient with necrotizing fasciitis before (A) and after (B) the involved fascia was excised. The classic and telltale finding is the layer of purulent, thickened, and friable fascia ordinarily not present between the subcutaneous layer and muscle. Note the lack of muscle involvement.

and mortality is early and adequate debridement.¹⁴ Negative prognostic factors include age more than 50 years, underlying chronic illness, diabetes mellitus, and involvement of the trunk.

Mycobacterial Infections

Seventy-five percent of atypical mycobacterial infections involve the hand.^{2,54} *Mycobacteria marinum* is the common organism, usually caused by wounds associated with contaminated swimming pools, fish tanks, piers, boats, fish bites or injuries from fish fins or spines.⁵⁴ Infections can be cutaneous (verrucal), subcutaneous (granulomatous), or deep (involving tendon, joint or bursal synovium or bone).⁵⁴ Tenosynovial infection is the most frequent deep manifestation. Variation from a typical rheumatoid pattern should alert the clinician. Misdiagnosis often delays appropriate treatment.⁵⁴ Skin tests and smears for acid-fast organisms are unreliable.^{2,54} When smears are negative, fungal infections, though rare, should be ruled out. Typically, systemic symptoms are absent and white blood cell count and sedimentation rate are normal.⁵⁴ Granulomas are noted on histology. Cultures must be incubated in Lowenstein-Jensen media at 30°C for up to 6 to 8 weeks.⁵⁴ Superficial infections are usually self-limited unless the lesions are picked at or biopsied without antibiotic coverage, in which case subcutaneous lesions can ensue.⁵⁴ A subcutaneous lesion requires debridement and 2 to 6 months of antibiotics and deep lesions require tenosynovectomy, synovectomy, or debridement of the involved bone or joint, with antibiotic duration from 4 to 24 months.⁵⁴ In cases of digital flexor tenosynovitis, the tenosynovium is completely removed, preserving the annular pulleys. Wrist dorsal tenosynovial infection is excised through a longitudinal incision and retinacular flaps. An extensile carpal tunnel approach is used to excise involved the flexor tenosynovium at the wrist.³² Minocycline is the antibiotic of choice. Ethambutol and rifampin are alternatives in case of allergy or organism insensitivity.^{2,54}

Fungal Infections

Fungal infections of the upper extremities can be cutaneous, subcutaneous, deep, or systemic.⁵³ Cutaneous lesions often involve the skin or nails. Keratinophilic fungi colonize glabrous skin (tinea corporis), the interdigital areas and palms (tinea manuum), or nails (onychomycosis), causing a pruritic scaling of the skin or nail deformity. Diagnosis can be made with potassium hydroxide preparations and fungal cultures. Skin infections are treatable with topical agents such as tolnaftate

and miconazole. Onychomycosis typically occurs in hands exposed to constant moisture and begins as a minor paronychia infection, progressing to whole nail involvement characterized by thickening, softening, discoloration, and cracking. Psoriatic nail deformities can mimic onychomycosis. Onychomycosis is notoriously resistant to treatment. Nail removal and application of topical agents is less successful than systemic griseofulvin, ketoconazole,⁵³ fluconazole, or itraconazole. The cure rate ranges from 57% to 80%.⁵³

Rare subcutaneous and deep fungal infections are often overlooked in the differential diagnosis of hand infections. Sporotrichosis is a common subcutaneous fungal lesion occurring predominantly in the upper extremities.⁵³ Subcutaneous implantation is the typical mode of transmission usually by plant handling (rose thorns, cacti, sphagnum moss). Ulceration occurs at the initial puncture site with eventual nodules forming along lymphatics that in turn can ulcerate. Cultures secure the diagnosis because standard stains are rarely helpful. Oral potassium iodide is the treatment of choice.

Deep fungal infections of the hand involve tenosynovium, joints, or bone. They may be caused by pathogenic fungi (histoplasmosis, blastomycosis, coccidioidomycosis) or opportunists (mucormycosis, aspergillosis, *Candida*). Infections usually enter through the pulmonary route with musculoskeletal infections occurring via hematogenous spread. Treatment requires debridement and antifungals.

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