Chapter 12

EXTREMITY CONDITIONS

Robert J. Tubbs
Daniel L. Savitt
Selim Suner

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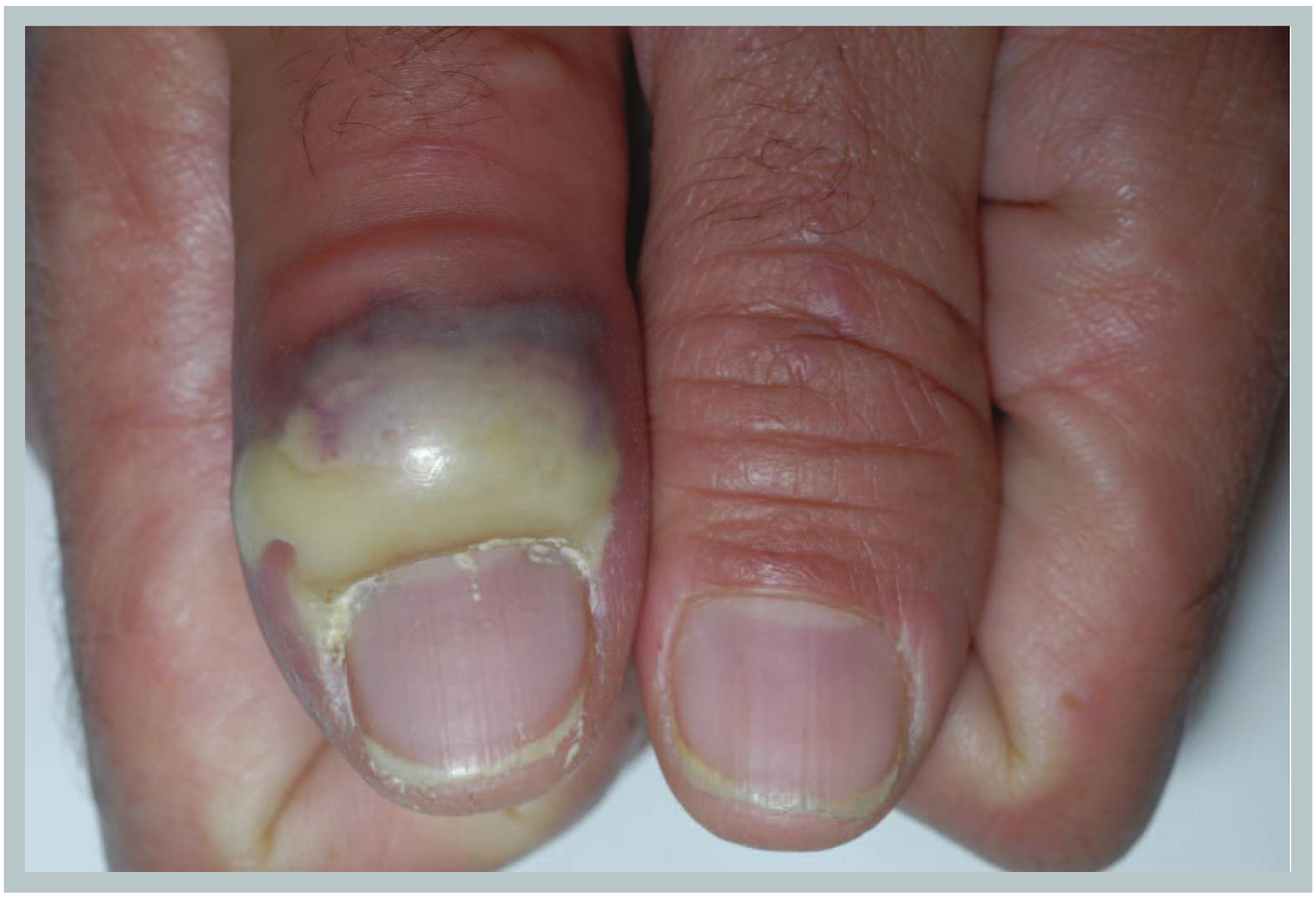

Paronychia. (Photo contributor: Lawrence B. Stack, MD.)

CELLULITIS

Clinical Summary

Cellulitis is a common infection of the skin or subcutaneous tissues with characteristic findings: erythema with poorly defined borders, edema, warmth, pain, and limitation of movement. Fever and constitutional symptoms may be present and are commonly associated with bacteremia. Predisposing factors include trauma, lymphatic or venous stasis, immunodeficiency (including diabetes mellitus), and foreign bodies. Common etiologic organisms include group A β-hemolytic Streptococcus and Staphylococcus aureus in nonintertriginous skin, and gram-negative organisms or mixed flora in intertriginous skin and ulcerations. In immunocompromised hosts, Escherichia coli, Klebsiella species, Enterobacter species, and Pseudomonas aeruginosa are common. In recent years, there has been a dramatic increase in the incidence of community-acquired methicillin-resistant S aureus (CA-MRSA), particularly in cellulitis associated with a cutaneous abscess.

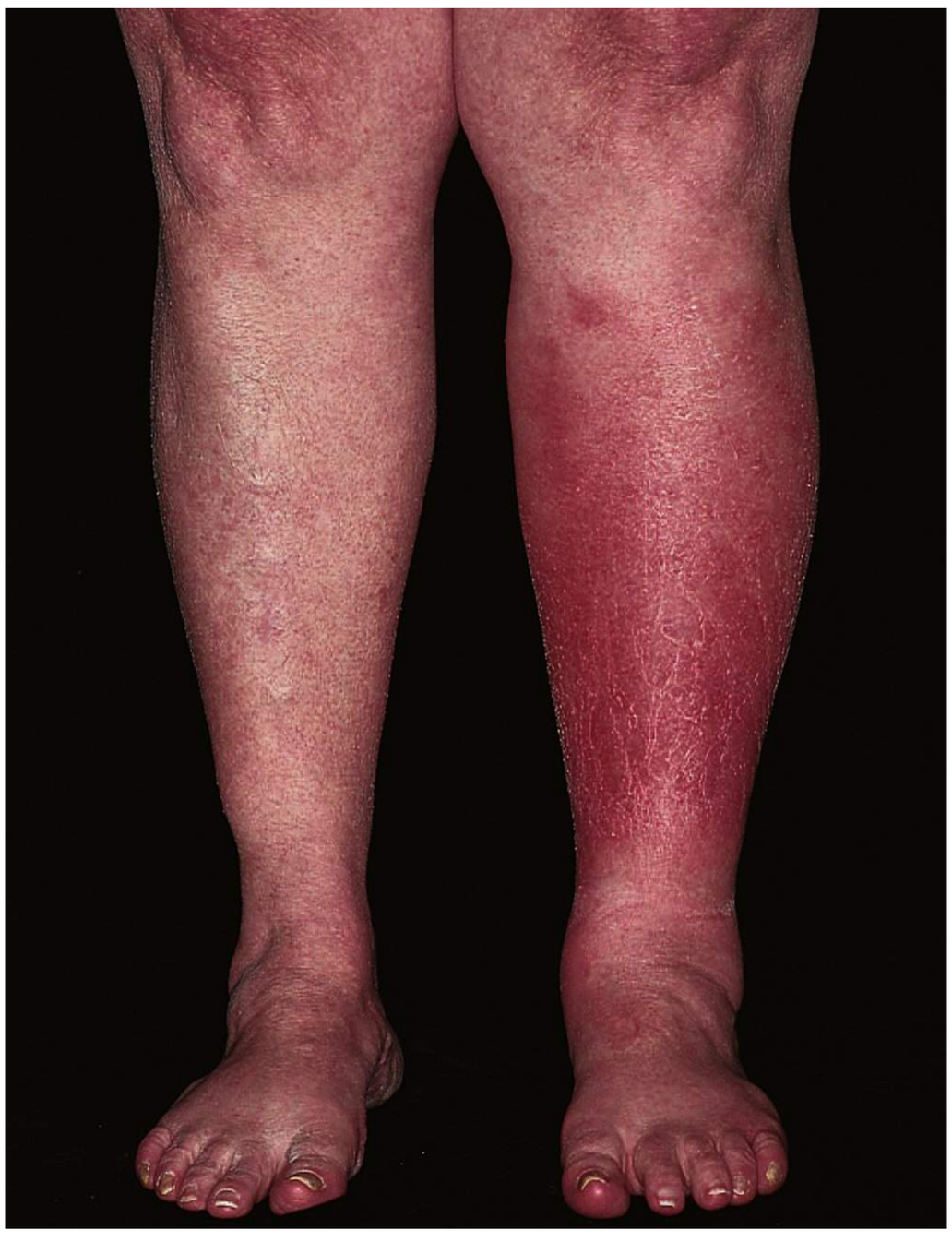

FIGURE 12.1 ■ **Cellulitis.** Cellulitis of the left leg characterized by erythema and mild swelling. (Photo contributor: Frank Birinyi, MD.)

The differential diagnosis includes deep venous thrombosis (DVT), venous stasis, erythema nodosum, septic or inflammatory arthritis/bursitis, and allergic reactions.

Management and Disposition

Treatment of minor cases commonly consists of immobilization, elevation, analgesia, and oral β-lactam antibiotics with reevaluation in 48 hours. The increase in the incidence of CA-MRSA has prompted some, especially in highly endemic areas, to advocate coverage with trimethoprim/sulfamethoxazole or other agents (refer to current recommendations) in addition to conventional β-lactam antibiotics. Admission and parenteral administration of antibiotics may be necessary for immunocompromised or toxic-appearing patients, or those who do not respond to outpatient therapy.

Pearls

1. Rapidly progressive cellulitis or one that progresses despite treatment with β-lactam antibiotics should raise suspicion for CA-MRSA or deeper infections such as fasciitis.
2. Known risk factors for CA-MRSA include military personnel, prison inmates, and competitive sports players.
3. Routine blood or leading-edge cultures in nontoxic patients are generally low yield.

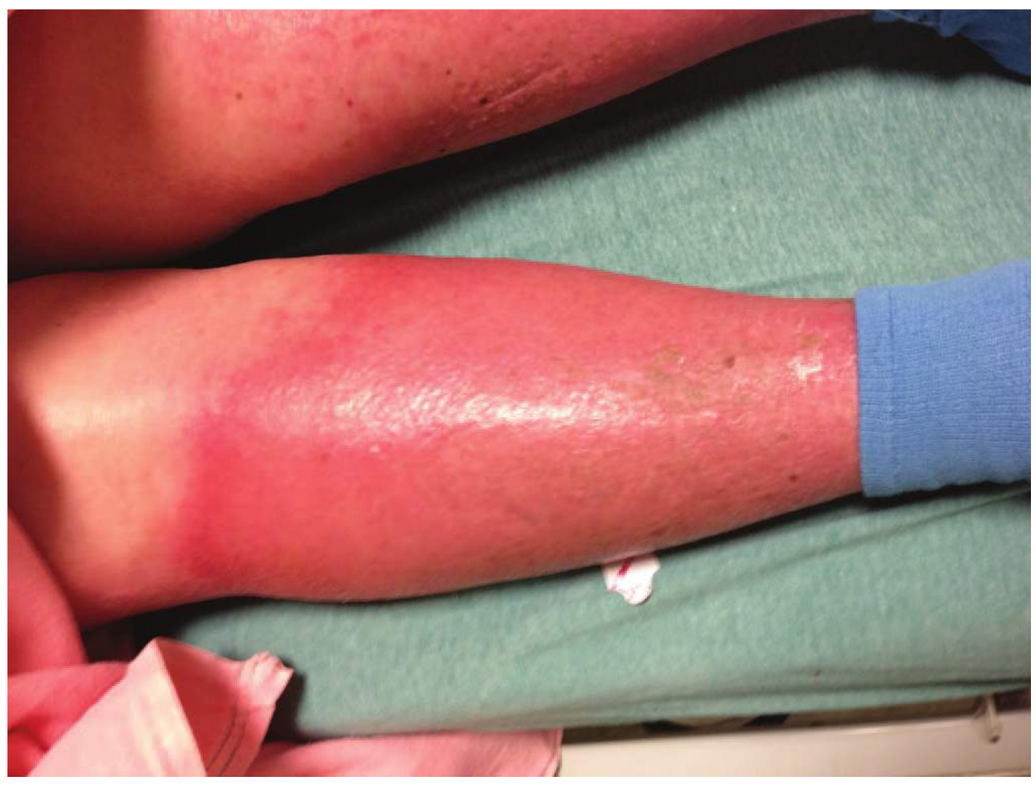

FIGURE 12.2 ■ **Cellulitis.** Cellulitis of the right lower extremity characterized by sharply demarcated erythema and edema. (Photo contributor: Robert Tubbs, MD.)

Clinical Summary

A felon is a pyogenic infection of the distal digital pulp space, with pus collecting in the spaces formed by the vertical septa anchoring the pad to the distal phalanx. This condition is characterized by severe pain, exquisite tenderness, and tense swelling of the distal digit with erythema. There may be a visible collection of pus or palpable fluctuance. Complications include deep ischemic necrosis, osteomyelitis, septic arthritis, and suppurative tenosynovitis. The differential diagnosis includes paronychia, herpetic whitlow, and hematoma following traumatic injury.

Management and Disposition

Incision and drainage is generally necessary to treat a felon. To ensure complete drainage of the abscess cavity, all affected compartments should be entered. The packing of the abscess space is made with a small, loose-fitting wick to facilitate drainage. Oral antibiotics directed against gram-positive organisms should be used for 10 days and the packing removed or replaced after 24 to 48 hours. Consider treatment for CA-MRSA in addition to standard coverage in highly endemic areas.

Pearls

1. Do not extend the incision proximal to the distal flexion crease.
2. Incisions should be made dorsal to the neurovascular bundle; the pincer surfaces (radial aspects of the index and long fingers and ulnar aspect of the thumb and small finger) should be avoided when possible.
3. "Hockey stick" and "fish mouth" incisions are associated with increased occurrence of unnecessary sequelae and are not recommended.
4. If there is radiographic evidence of osteomyelitis, or concern for tenosynovitis, consultation with a hand surgeon is required.

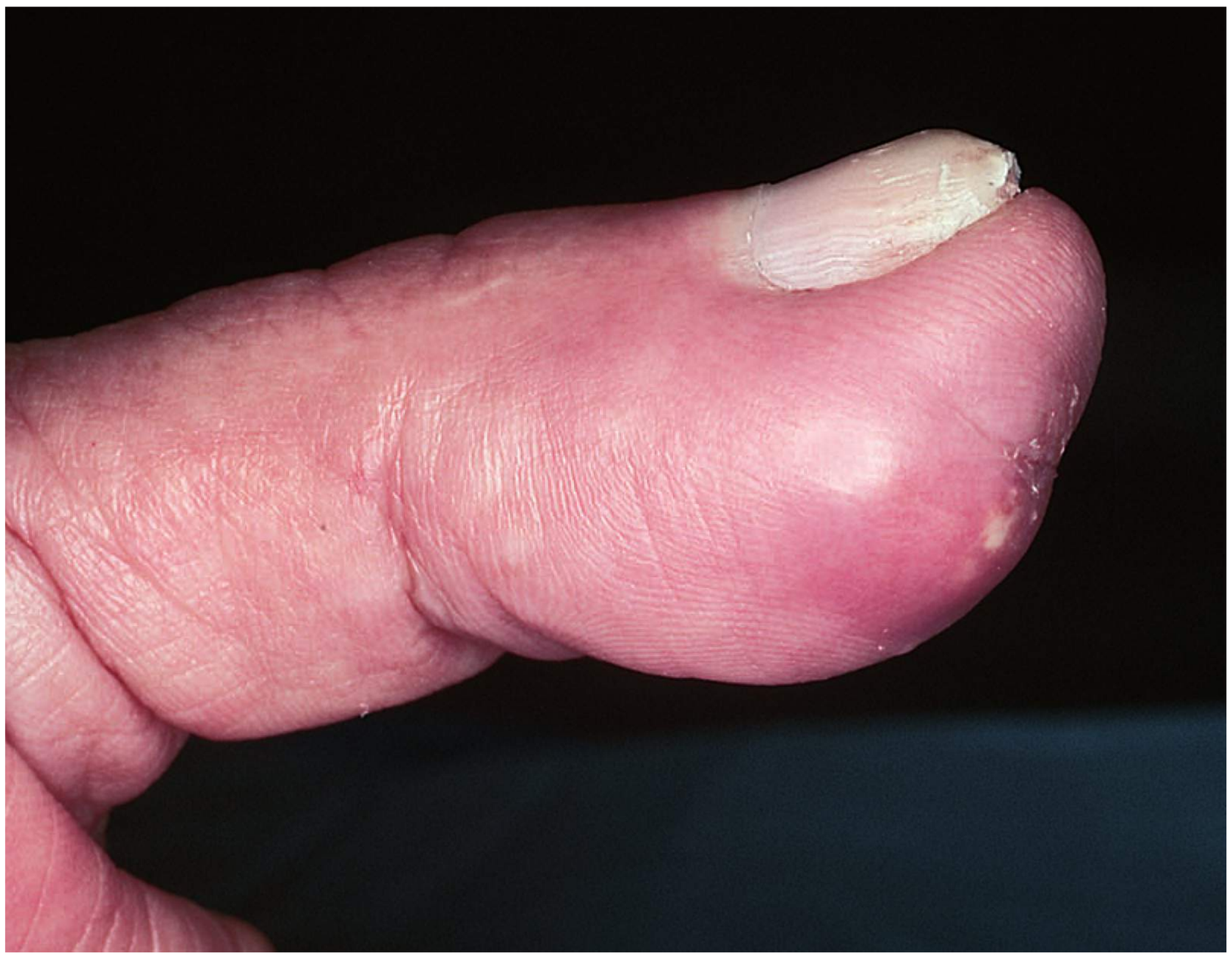

FIGURE 12.3 ■ **Felon.** Purulence, swelling, and erythema at the center of the palmar pad. (Photo contributor: Daniel L. Savitt, MD.)

GANGRENE

Clinical Summary

Gangrene denotes tissue with lost blood supply and undergoing necrosis. The term dry gangrene is used for tissues undergoing sterile ischemic coagulative necrosis. Patients with atherosclerotic disease and diabetes are at risk for the development of dry gangrene, usually as a result of embolization to the forefoot or toe. Wet gangrene is associated with bacterial proteolytic decomposition, and is characterized by its moist appearance, frequently with blistering.

Management and Disposition

Hospitalization is usually required. The treatment consists of amputation, debridement, and antibiotic therapy as needed. Wet gangrene requires emergent surgical consultation. Underlying vascular pathology must be evaluated by arteriography and corrected surgically or endovascularly. Patients with systemic toxicity should be resuscitated aggressively.

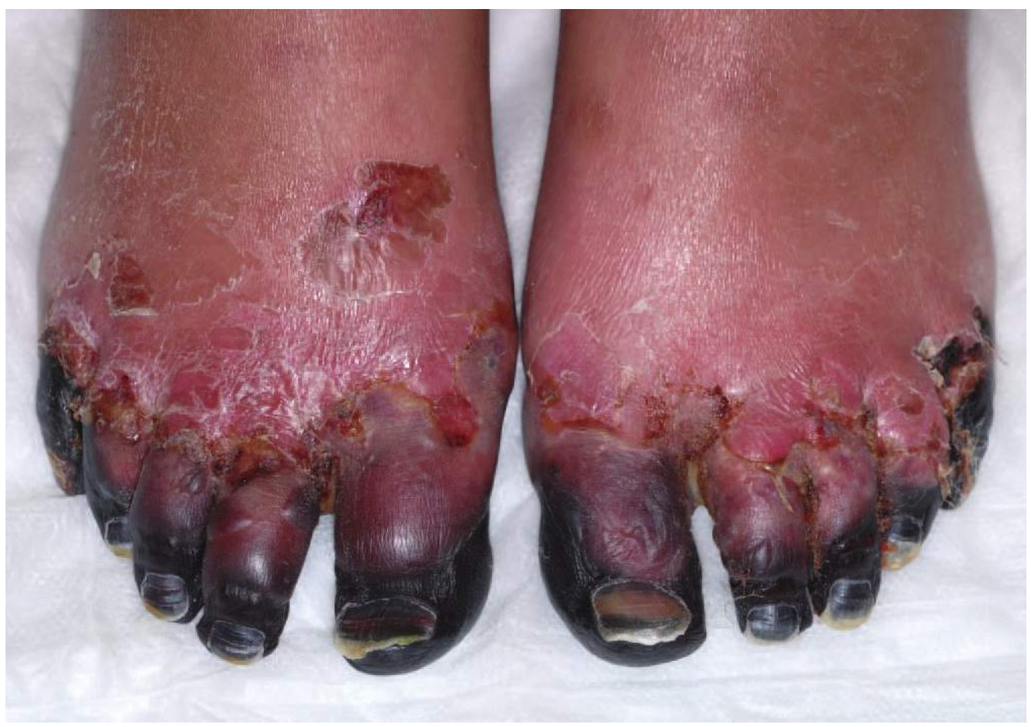

FIGURE 12.4 ■ **Dry Gangrene.** Dry gangrene of the toes showing the areas of total tissue death, appearing as black and lighter shades of discoloration of the skin demarcating areas of impending gangrene. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Obtain radiographs to help rule out clostridial myonecrosis (gas gangrene, see related item) and osteomyelitis.
2. Aggressive surgical debridement is necessary for cure in most cases of wet gangrene.

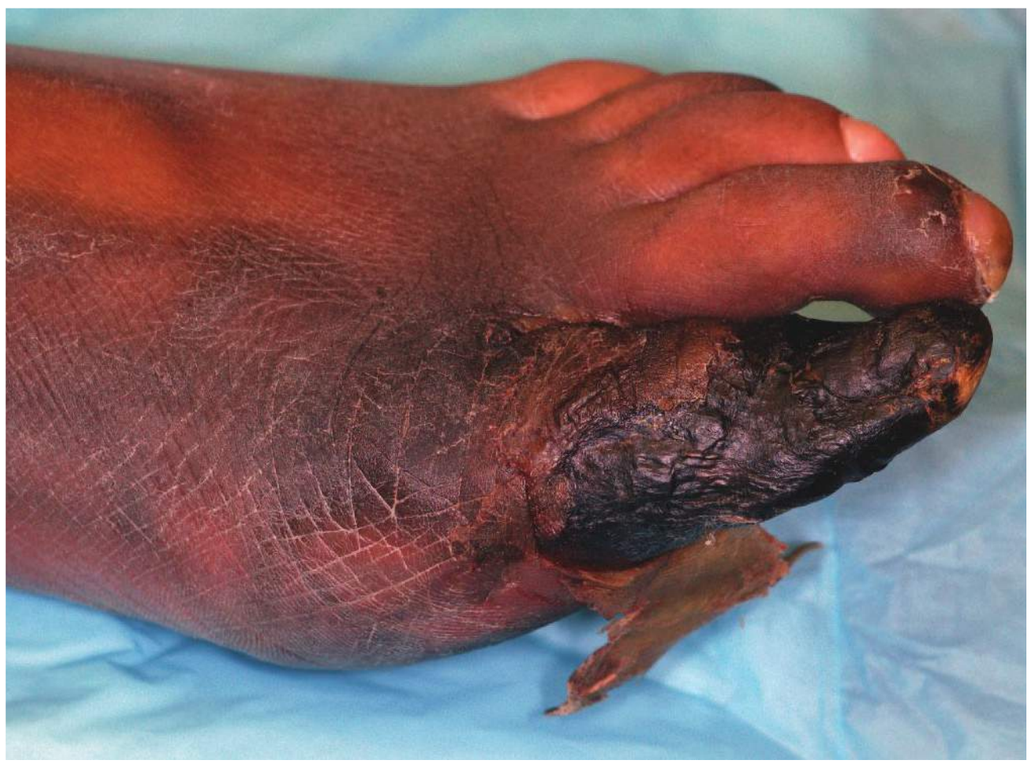

FIGURE 12.5 ■ **Dry Gangrene.** Complete tissue death characterized by black, desiccated tissue, and lighter areas demarcating areas of impending gangrene. (Photo contributor: David Effron, MD.)

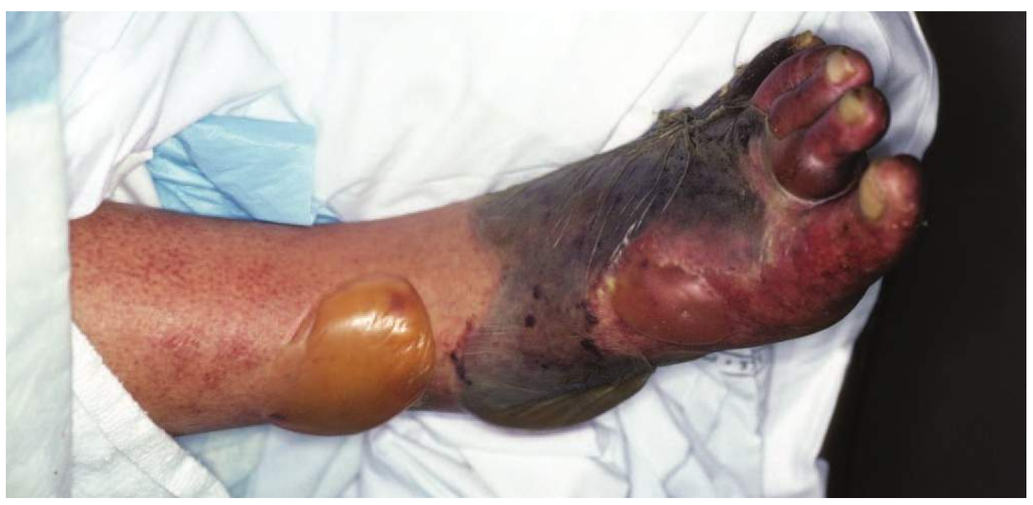

FIGURE 12.6 ■ **Wet Gangrene.** Note the moist appearance and blistering due to bacterial proteolytic decomposition of gangrenous tissue. (Photo contributor: Robert Tubbs, MD.)

Clinical Summary

This infection causes rapid necrosis and liquefaction of fascia, muscle, and tendon. Most involve Clostridium perfringens; Streptococcus pyogenes accounts for the majority of remaining cases. Myonecrosis is classically associated with trauma (including surgery) and diabetes. There is edematous bronze or purple discoloration, flaccid bullae with watery brown nonpurulent fluid, and a foul odor. The classic clinical presentation is pain out of proportion to physical findings. Low-grade fever and other systemic signs are also typically present, and may develop rapidly into shock.

Crepitance and appearance of gross pockets of air in the tissue are appreciated, but may not be present early. The incubation period for clostridia ranges between 1 and 4 days, but it can be as early as 6 hours. Decreased tissue oxygen tension along with wound contamination is required for the infection to progress. Crepitant cellulitis, synergistic necrotizing cellulitis, acute streptococcal hemolytic gangrene, and streptococcal myositis are some conditions that may be mistaken for clostridial myositis. Often, surgical exploration of the fascia and muscle is required to make the correct diagnosis.

Management and Disposition

Aggressive resuscitation should be initiated, and consideration given to packed red blood cell transfusion. Broad-spectrum antibiotics, especially clindamycin, in conjunction with penicillin G, are given urgently. Tetanus prophylaxis must not be overlooked. Surgical debridement or amputation is the mainstay of therapy. Hyperbaric oxygen may have a synergistic effect in preventing the progression of infection and toxin production.

Pearls

1. Shock and multiorgan failure may be rapidly progressive. Mortality is 80% to 90% if untreated, 10% to 25% when treated appropriately.
2. Clindamycin may improve survival by inhibiting toxin production.
3. Gram stain of gram-positive bacilli with a relative lack of leukocytes may rapidly confirm suspected clostridial myonecrosis.

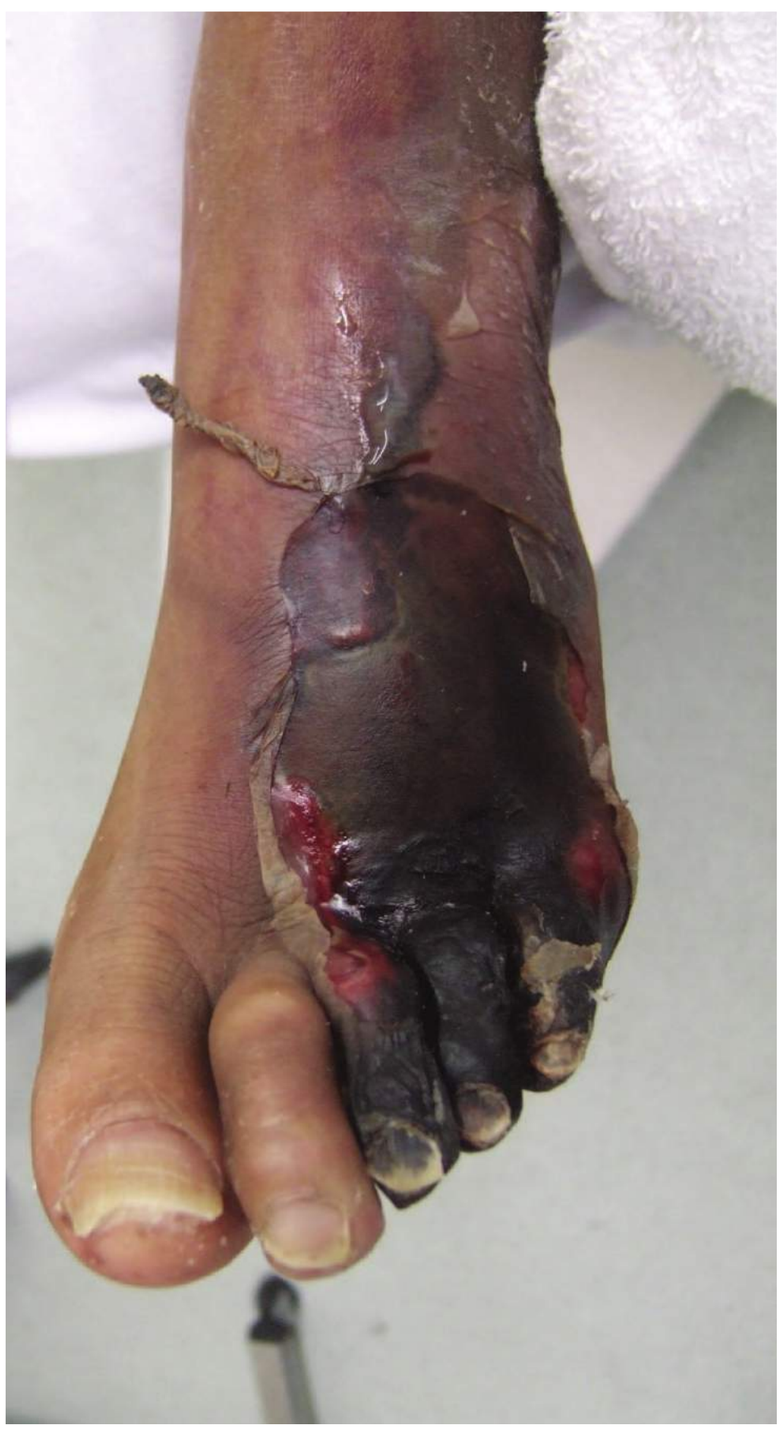

FIGURE 12.7 ■ **Gas Gangrene.** A foot with significant areas of gangrene and sloughing skin. (Photo contributor: David Kaplan, MD.)

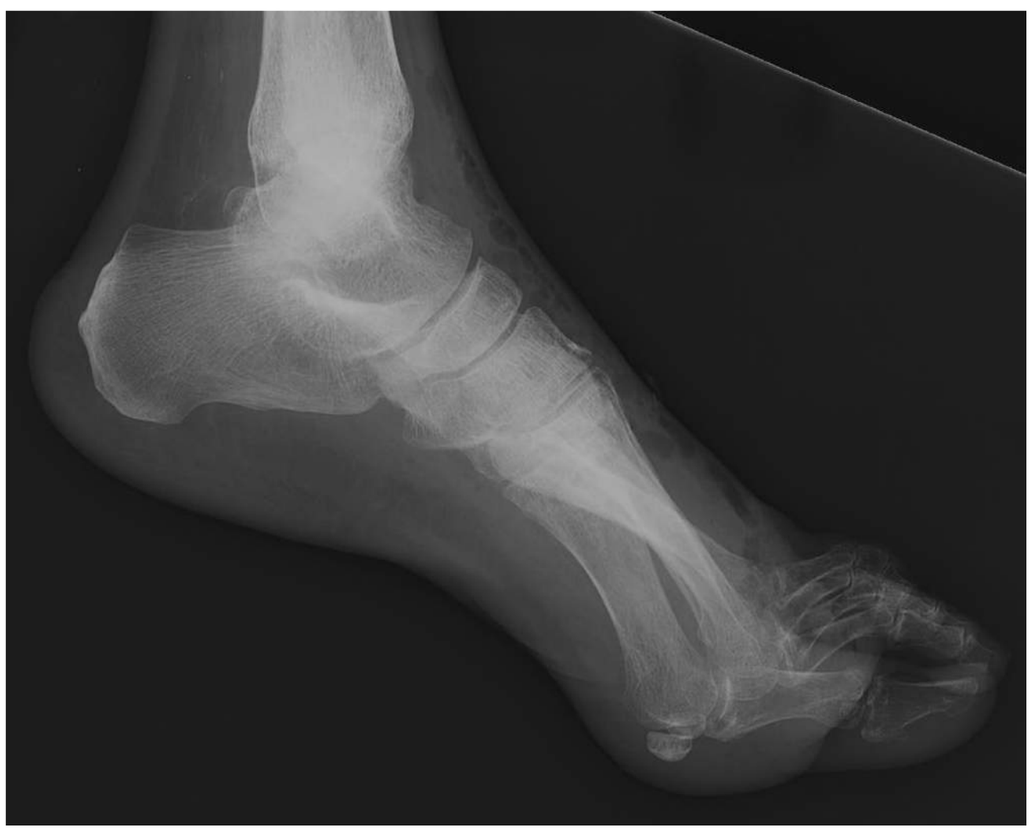

FIGURE 12.8 ■ **Gas Gangrene.** Radiograph of the foot in Fig. 12.7 exhibiting pockets of soft tissue gas tracking up the dorsal surface. (Photo contributor: Douglas Nilson, MD.)

NECROTIZING FASCIITIS

Clinical Summary

This uncommon, severe infection involves the subcutaneous soft tissues, including the superficial and deep fascial layers, with early skin sparing and late muscle involvement. It is most commonly seen in the lower extremities, abdominal wall, and perianal and groin area, as well as in postoperative wounds. It is commonly spread from a trauma site, surgical wound, abscess, or decubitus ulcer. Alcoholism, parenteral drug abuse, and diabetes are predisposing factors. Pain, tenderness, erythema, swelling, warmth, shiny skin, lymphangitis, and lymphadenitis are early findings. Later, there is rapid progression of bullae with clear pink or purple fluid and cutaneous necrosis. The skin becomes anesthetic, and subcutaneous gas may be present. Systemic toxicity may be manifest by fever, dehydration, leukocytosis, and frequently positive blood cultures. Type I includes anaerobic species (Bacteroides and Peptostreptococcus) and type II includes either group A streptococci alone or group A streptococci with S aureus.

Management and Disposition

Prompt diagnosis is critical; if made within 4 days from symptom onset, the mortality rate is reduced from ~50% to 12%. Initial treatment involves resuscitation with volume expansion, operative debridement, and prompt initiation of broad-spectrum antibiotics.

Pearls

1. Intravenous calcium may be necessary to reverse hypocalcemia from subcutaneous fat necrosis.
2. Radiographs may detect nonpalpable subcutaneous gas.
3. Hemolysis and disseminated intravascular coagulation (DIC) may be present.
4. Community-acquired MRSA has been implicated as a causative organism.

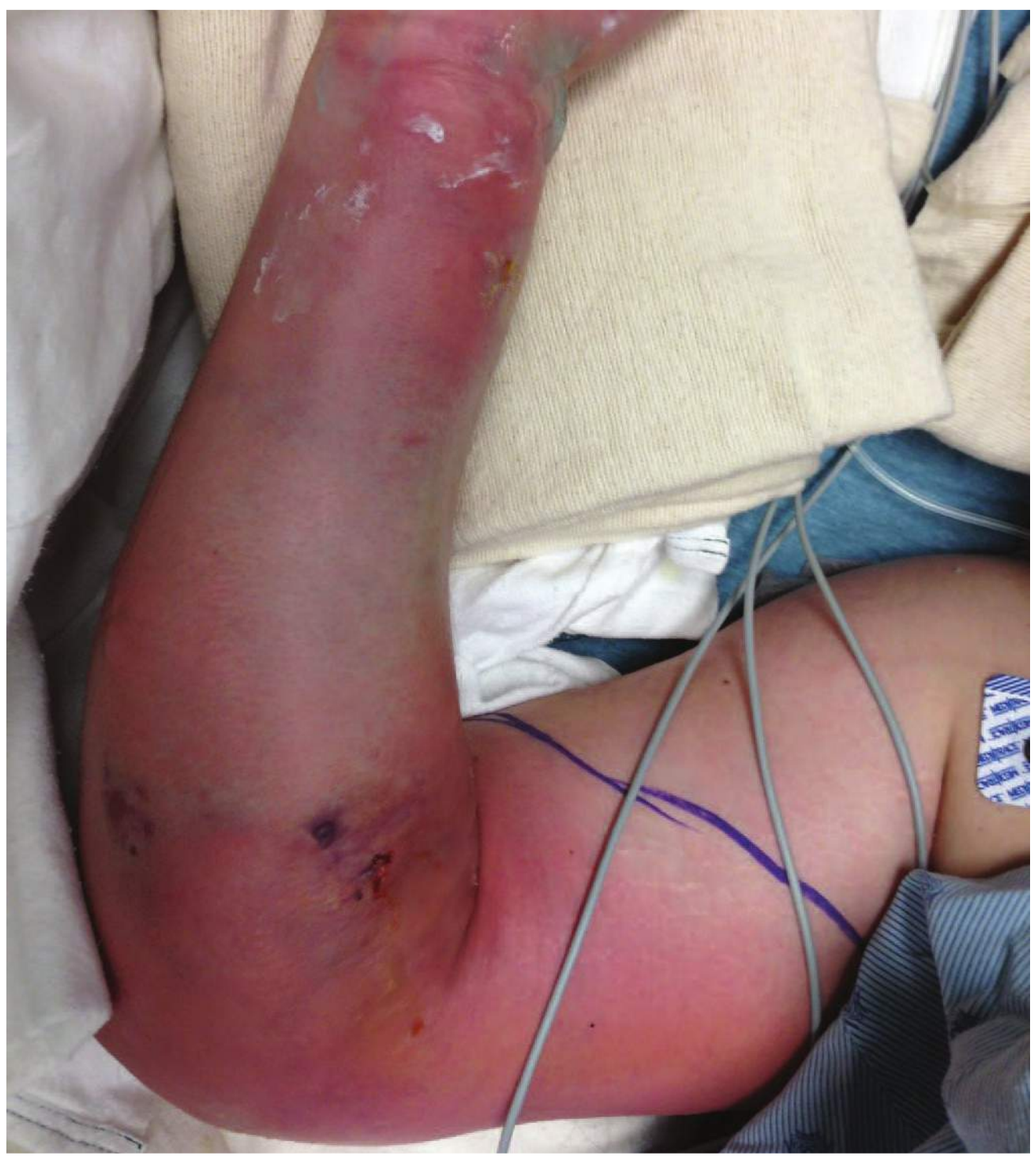

FIGURE 12.9 ■ **Necrotizing Fasciitis.** Markedly swollen, dusky, erythematous arm with necrotizing fasciitis in an IV drug user. (Photo contributor: Alexis Lawrence, MD.)

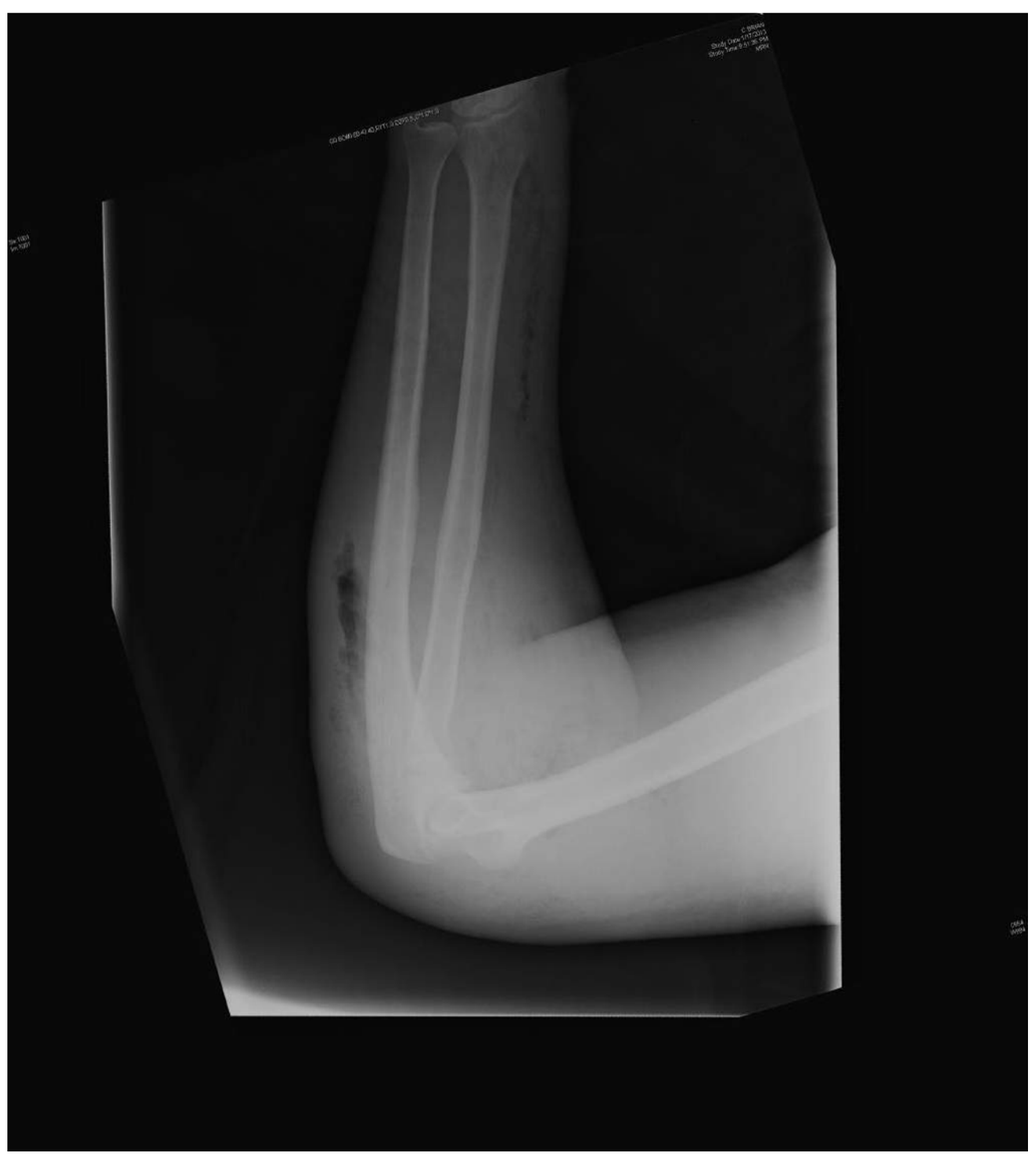

FIGURE 12.10 ■ **Necrotizing Fasciitis.** Radiograph of the forearm in Fig. 12.9 showing extensive subcutaneous gas. (Photo contributor: Alexis Lawrence, MD.)

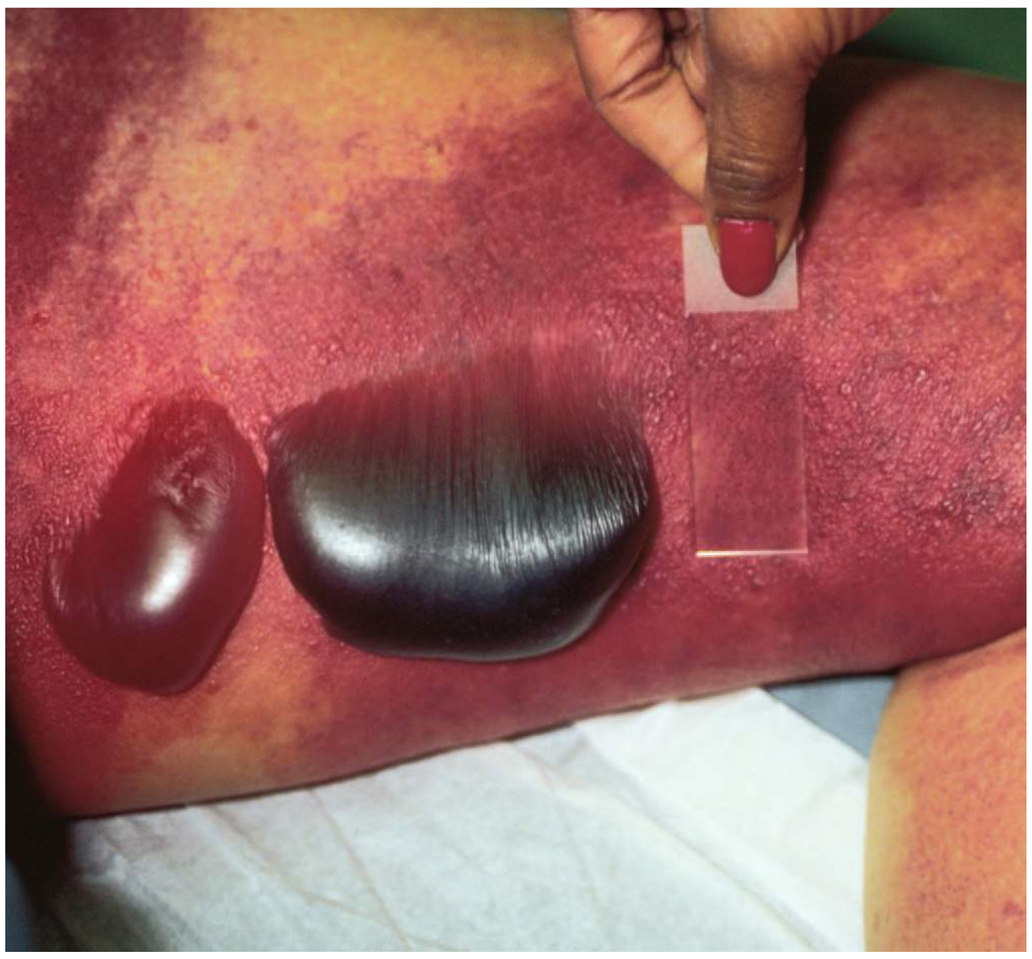

FIGURE 12.11 ■ **Necrotizing Fasciitis.** Large cutaneous bullae on the leg of this patient with necrotizing fasciitis. Note the dark purple fluid in the bullae. (Photo contributor: Lawrence B. Stack, MD.)

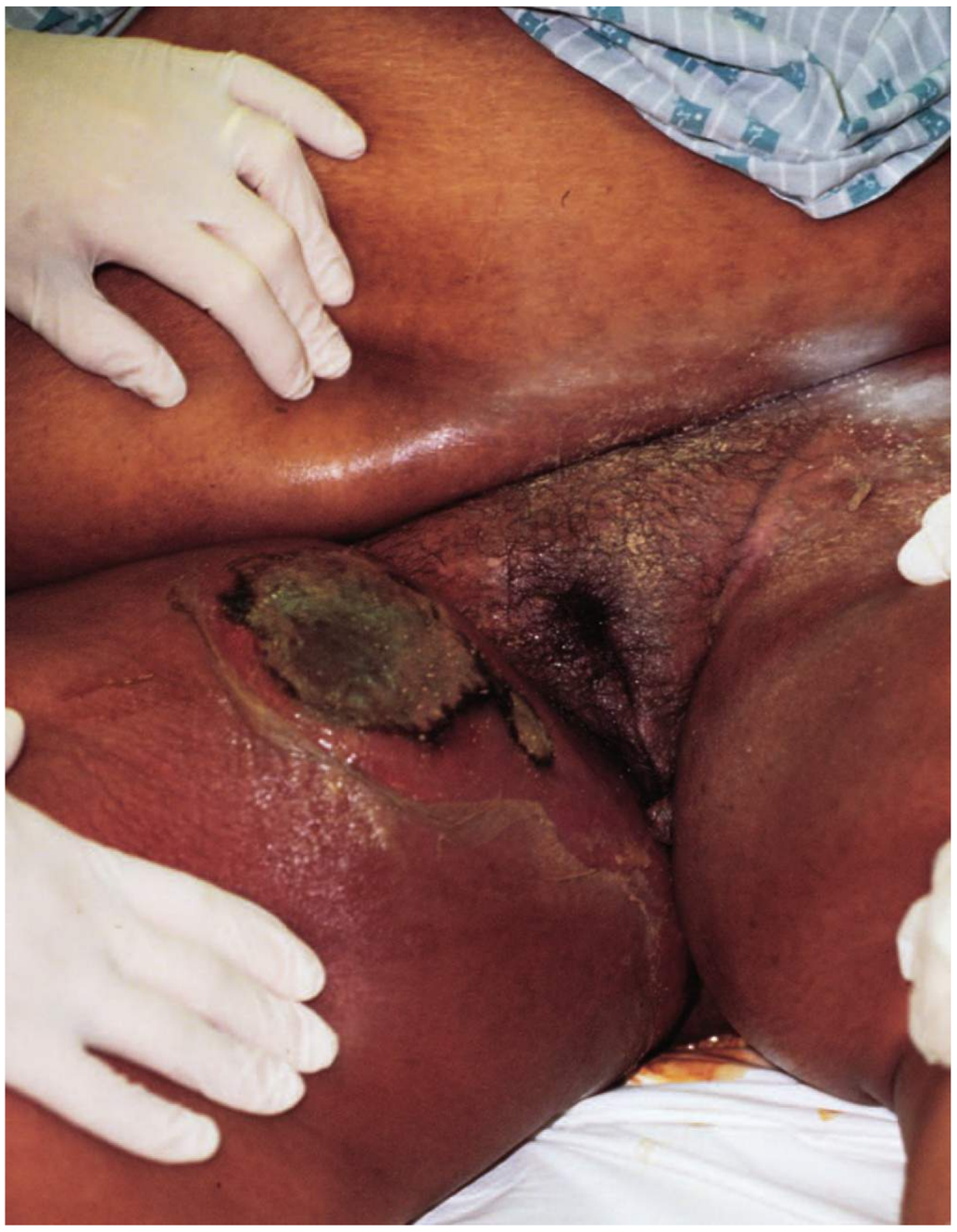

FIGURE 12.12 ■ **Necrotizing Fasciitis.** Necrotizing fasciitis with cutaneous necrosis in the inner thigh. (Photo contributor: Lawrence B. Stack, MD.)

CRYSTAL-INDUCED SYNOVITIS (GOUT AND PSEUDOGOUT)

Clinical Summary

Gout is an inflammatory disease characterized by deposition of sodium urate monohydrate crystals in cartilage, subchondral bone, and periarticular structures. An acute attack is characterized by sudden onset of monoarticular arthritis (commonly in the metatarsophalangeal joint of the great toe) with swelling, erythema, and tenderness. The deposits of crystals about the joint produce a chronic inflammatory response termed a tophus.

In pseudogout, calcium pyrophosphate dihydrate (rod- or rhombus-shaped, weakly birefringent) crystals are deposited. Although any joint may be involved, knees and wrists are most common. Presenting signs and symptoms are identical with gout, but formation of tophi is not seen. Low-grade fever, pain, and erythema are common to both entities.

Cellulitis and septic arthritis must be excluded. In the synovial fluid cell count, white blood cell count of 2000 to 50,000 with PMN predominance is expected (Table 1), although it may be significantly higher. The diagnosis is confirmed with urate or calcium pyrophosphate dihydrate crystals on polarized microscopy (see Microscopic chapter) coupled with negative Gram stain and cultures.

Management and Disposition

Nonsteroidal anti-inflammatories are used with excellent results acutely, along with joint immobilization and rest. Oral or intravenous (IV) colchicine is an alternative, but can cause nausea, vomiting, and diarrhea, as well as serious toxicities, including bone marrow suppression, neuropathy, myopathy, and death (particularly when given IV). Systemic and intra-articular corticosteroids and adrenocorticotropic hormone (ACTH) are alternatives. Allopurinol or probenecid is used in the chronic management of gout and plays no role in acute treatment.

Pearls

1. Serum urate may be normal in acute gout.
2. Acute attacks may be triggered by minor trauma, diuretic or salicylate use, alcohol, or diet.
3. Punched-out subchondral bone lesions may be seen on radiography in chronic tophaceous gout. Chondrocalcinosis may be seen in pseudogout.
4. Synovial fluid cell counts alone cannot be used to differentiate crystalline arthropathies from infectious etiologies.

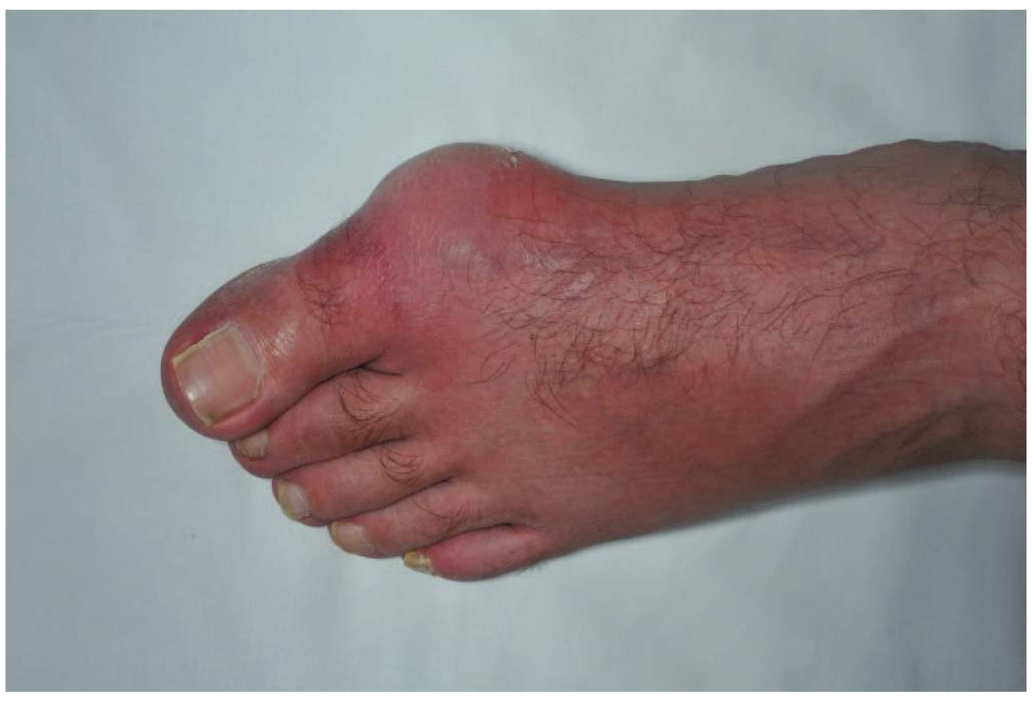

FIGURE 12.13 ■ **Podagra.** Foot with erythematous base of great toe classic for gout ("podagra"). (Photo contributor: Lawrence B. Stack, MD.)

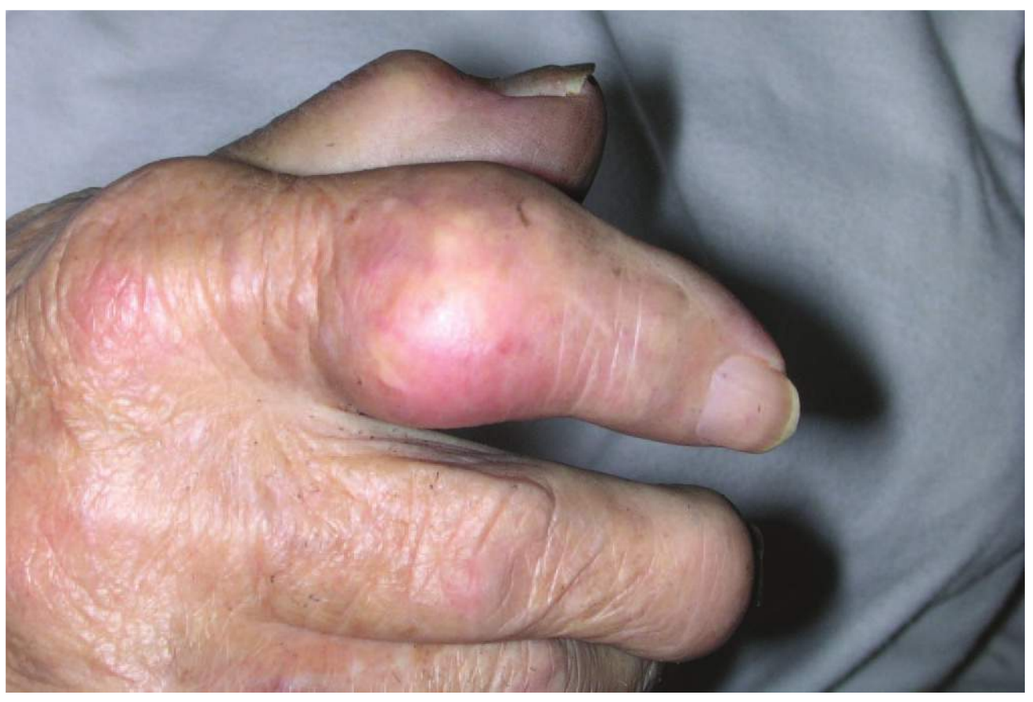

FIGURE 12.14 ■ **Gout.** Markedly enlarged index finger proximal interphalangeal joint showing classic tophaceous gout. (Photo contributor: Robert Tubbs, MD.)

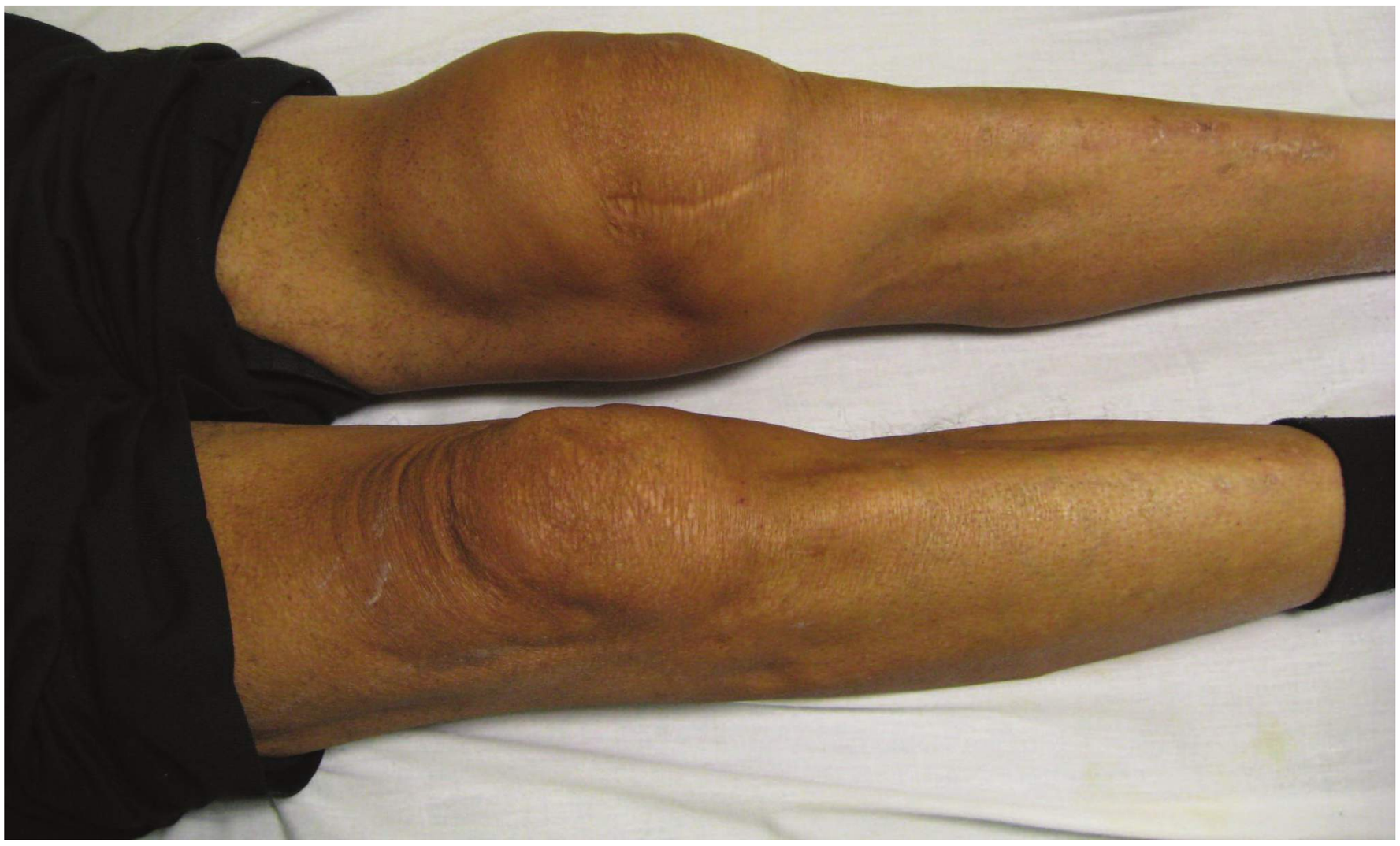

FIGURE 12.15 ■ **Pseudogout.** Markedly swollen left knee in a patient with an acute exacerbation of pseudogout. (Photo contributor: Robert Tubbs, MD.)

TABLE 12.1 ■ SYNOVIAL FLUID ANALYSIS

	Normal	Septic Arthritis	Gout	Pseudogout
Appearance	Clear	Purulent, turbid	Turbid	Turbid
WBC	<200	Usually >50,000	2000-50,000	2000-50,000
PMN	<25%	>75%	>50%	>50%
Special Tests/ Crystals	Normal "string sign"	+ Gram stain, culture positive	Negative birefringence, needle crystals	Positive birefringence, rhomboid crystals

INGROWN TOENAIL (ONYCHOCRYPTOSIS)

Clinical Summary

This condition is the result of impingement and puncture of the medial or lateral nail fold epithelium by the nail plate, allowing growth into the dermis. Tenderness and swelling of the nail fold is followed by granulation tissue causing sharp pain, erythema, and further swelling. If not promptly treated, the granulation tissue becomes epithelialized, preventing elevation of the nail above the medial or lateral nail groove. Often, there is secondary bacterial or fungal infection. Risk factors include cutting the nail too short, tightly fitting shoes, and trauma. The differential diagnosis includes paronychia, felon, and tumor.

Management and Disposition

Early treatment: Elevation of the nail out of fold and placement of gauze under it to prevent contact, in conjunction with warm soaks, is the initial therapy.

Late treatment: Surgical management involves removal of part of the nail and the inflamed tissue, and sometimes destruction of the involved nail matrix. The lateral nail is removed followed by packing of the paronychial fold with petroleum gauze or other nonadherent dressing. Follow-up by a podiatrist until growth of the nail plate is complete should be considered. The destruction of the nail matrix is required only in patients with recurrent infected ingrown toenails and is not part of routine emergency care.

Pearls

1. Ingrown toenail is most common in the great toe.
2. Use of antibiotics is not a substitute for surgical excision and will result in only transient improvement of symptoms.

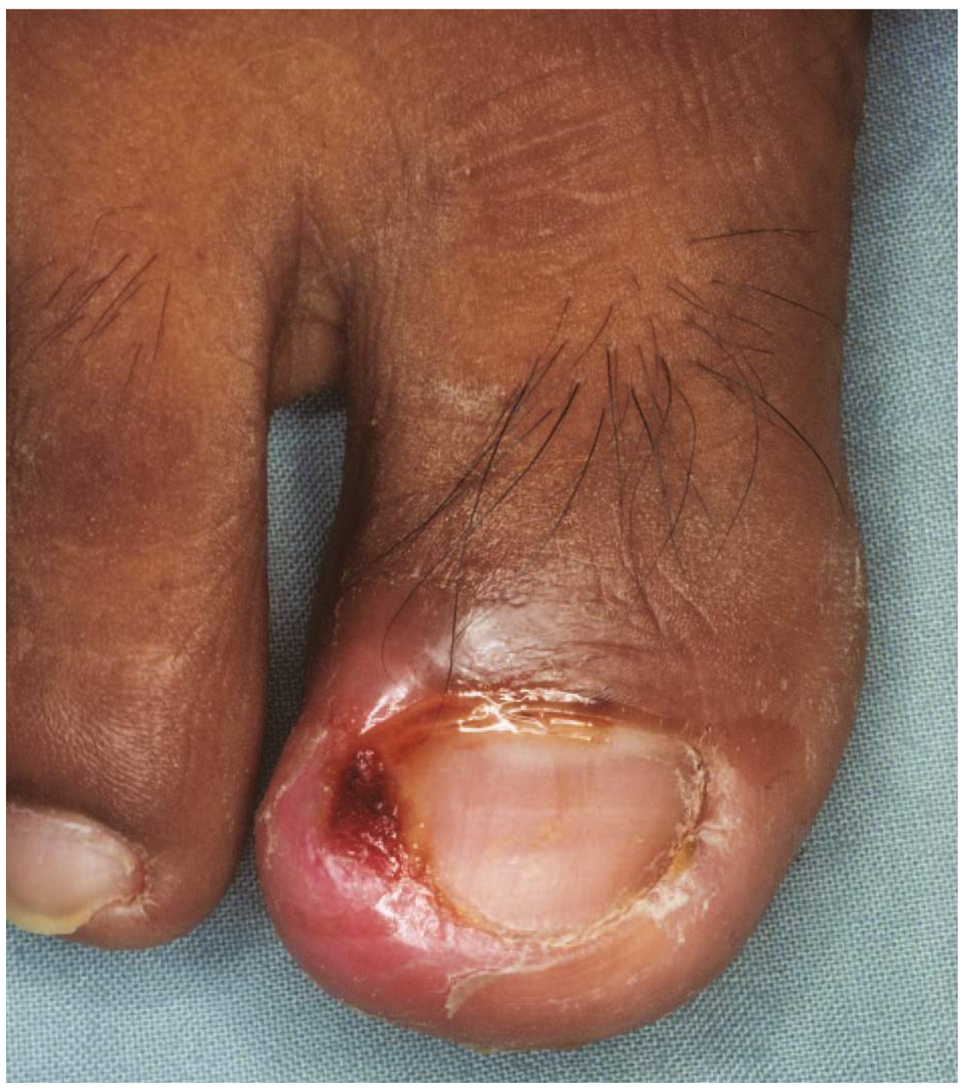

FIGURE 12.16 ■ **Ingrown Toenail.** Ingrown toenail on the lateral aspect of the great toe with granulation tissue and erythema. (Photo contributor: Selim Suner, MD, MS.)

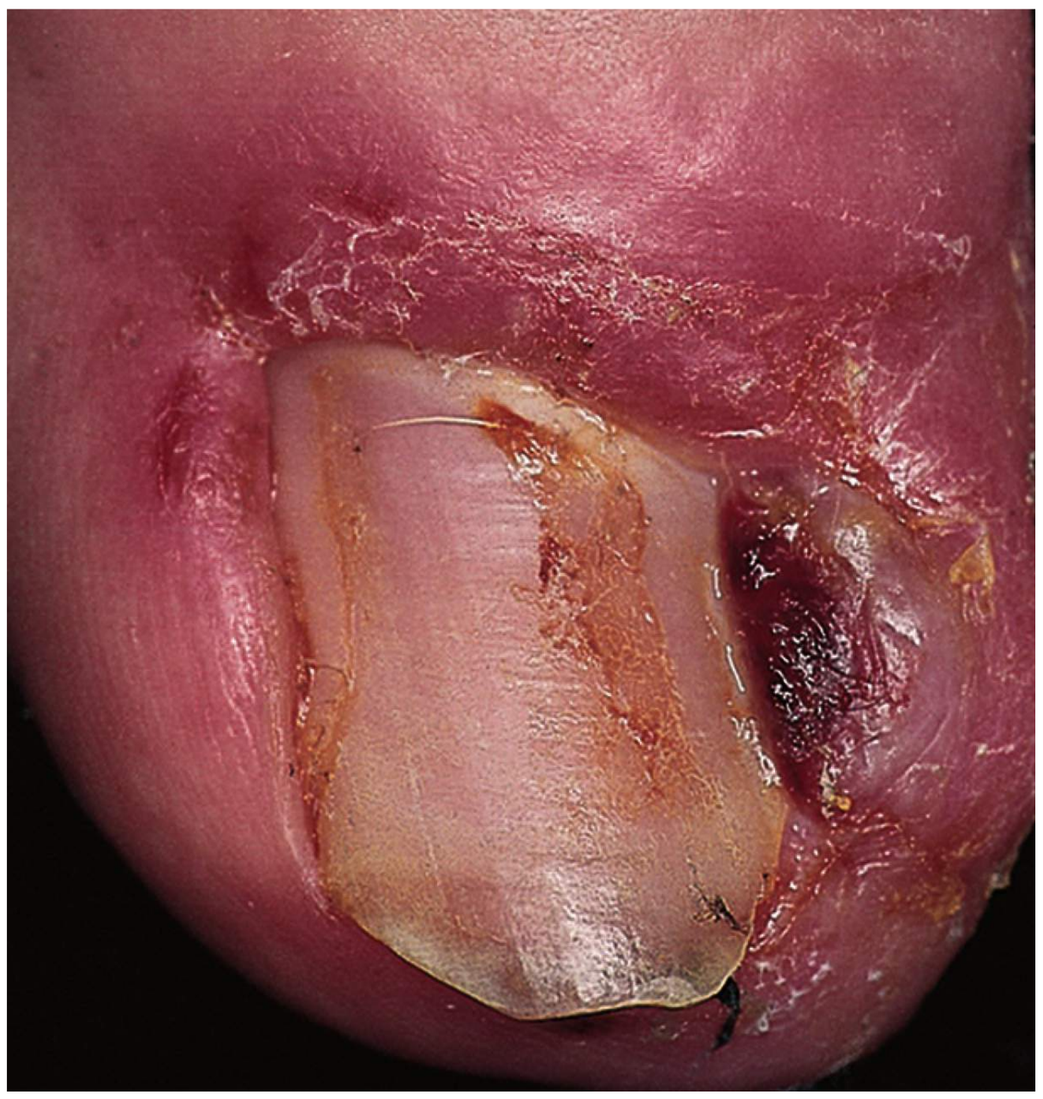

FIGURE 12.17 ■ **Ingrown Toenail.** An ingrown toenail on the medial aspect of the left great toe. (Photo contributor: Frank Birinyi, MD.)

Clinical Summary

Inflammation of lymphatic channels in the subcutaneous tissues is commonly caused by spread of local bacterial infection; Group A β-hemolytic streptococcal species are the most frequently implicated. Lymphangitis is characterized by red linear streaks extending, within 24 to 48 hours, from a primary site of infection (eg, abscess, cellulitis) to regional lymph nodes (eg, axilla, groin). The lymph nodes are often enlarged and tender. The differential diagnosis includes cellulitis, trauma, and superficial thrombophlebitis.

Management and Disposition

Rest, elevation, immobilization, and antibiotics are the initial treatment. Coverage for Streptococcus and Staphylococcus is appropriate. Toxic-appearing patients require admission for parenteral antibiotics. Any patient sent home with oral antibiotics should be followed up in 24 to 48 hours. Patients who subsequently do not show improvement require admission for parenteral antibiotic therapy. Consider treatment for CA-MRSA in addition to standard skin coverage in highly endemic areas.

Pearls

1. Consider Pasteurella multocida with cat and dog bites, Spirillum minus with rat bites, and Mycobacterium marinum in association with swimming pools and aquaria.
2. Chronic lymphangitis may be associated with mycotic, mycobacterial, and filarial infection.
3. Aspiration of the leading edge is generally not helpful for acute management.

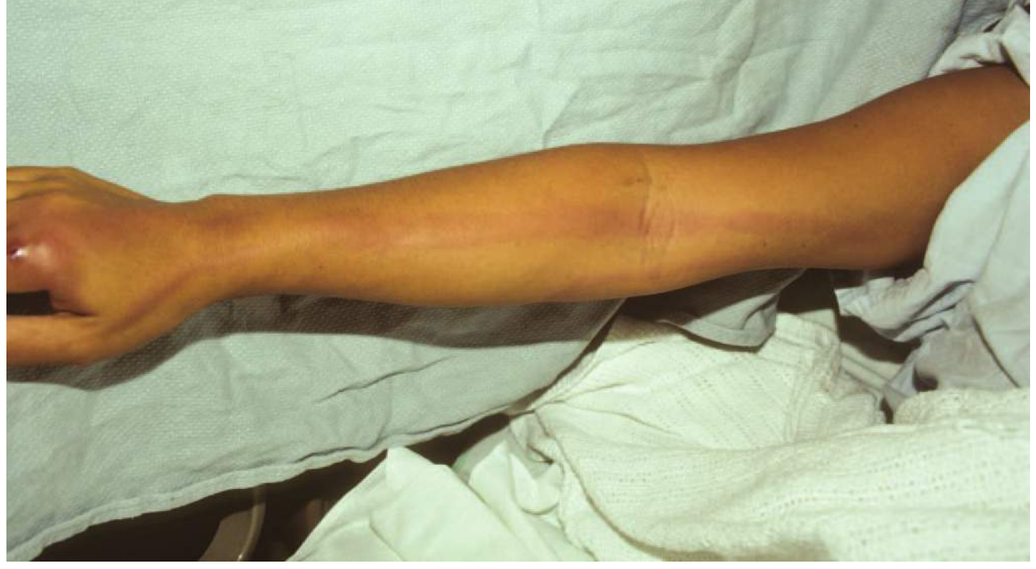

FIGURE 12.18 ■ **Lymphangitis.** Severe lymphangitis extending from metacarpophalangeal wound up the arm from a "fight bite." The red streak extends from the hand to the axilla, extending along lymphatic channels. (Photo contributor: Selim Suner, MD, MS.)

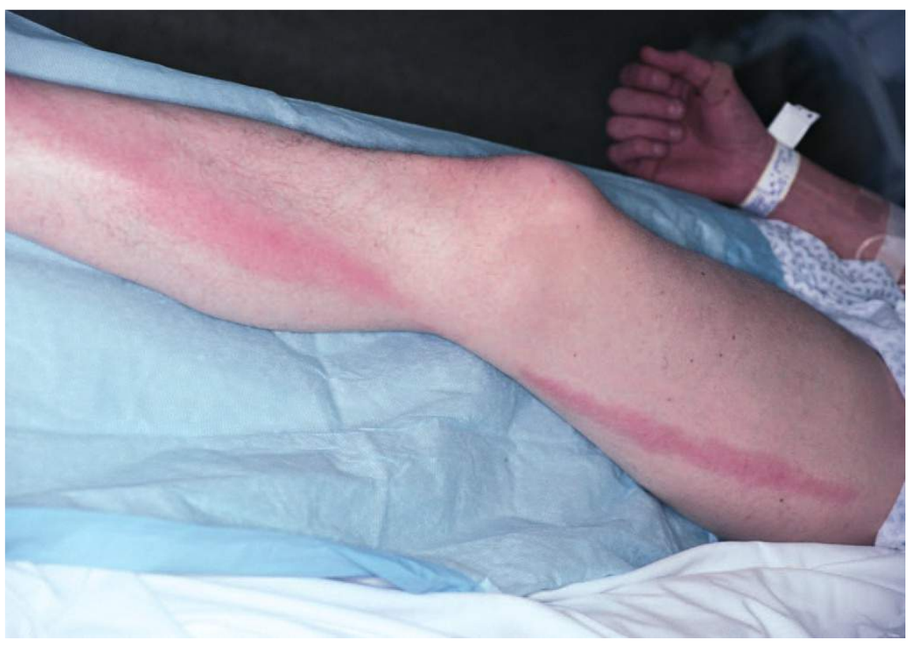

FIGURE 12.19 ■ **Lymphangitis.** The red streak extends from ankle to groin along lymphatic channels. The site of infection was the great toe. (Photo contributor: Liudvikas Jagminas, MD.)

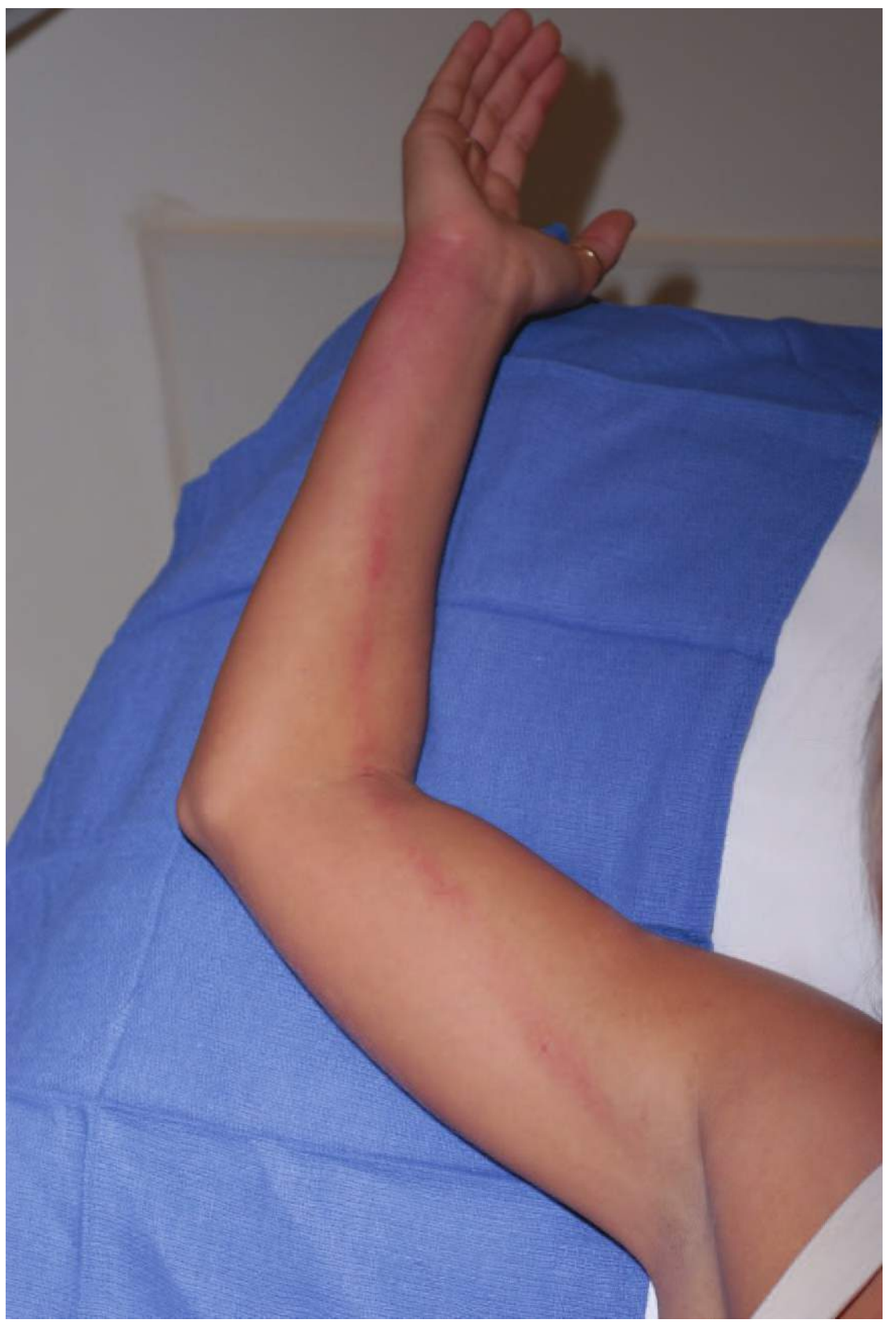

FIGURE 12.20 ■ **Lymphangitis.** Lymphangitis in the upper extremity, in this case from wrist to upper arm, commonly arises from nail biting. (Photo contributor: R. Jason Thurman, MD.)

LYMPHEDEMA

Clinical Summary

Lymphedema occurs from obstruction of lymphatic channels and is associated with malignancy, radiation, trauma, surgery, inflammation, infection, parasitic invasion, DVT, paralysis, renal insufficiency, heart failure, cirrhosis, and malnutrition. Lymphedema is characterized by painless pitting edema, fatigue, increase in limb size (particularly during the day), and presence of lymph vesicles. The skin becomes thickened and brown in the late stages.

Management and Disposition

Elevation, pneumatic compression boots and firm elastic stockings, maintenance of healthy skin, and avoidance of cellulitis and lymphangitis are the mainstays of symptomatic treatment. Treatment of the underlying disease may be curative.

Pearls

1. The dorsum of the toes and feet are always involved in lymphedema, unlike other causes of edema.
2. Careful examination for heart failure and screening for renal insufficiency should be completed for all patients.
3. Patients with chronic lymphedema are at risk for development of a rare secondary malignancy, lymphangiosarcoma, characterized by red-blue or purple macular or papular lesions.

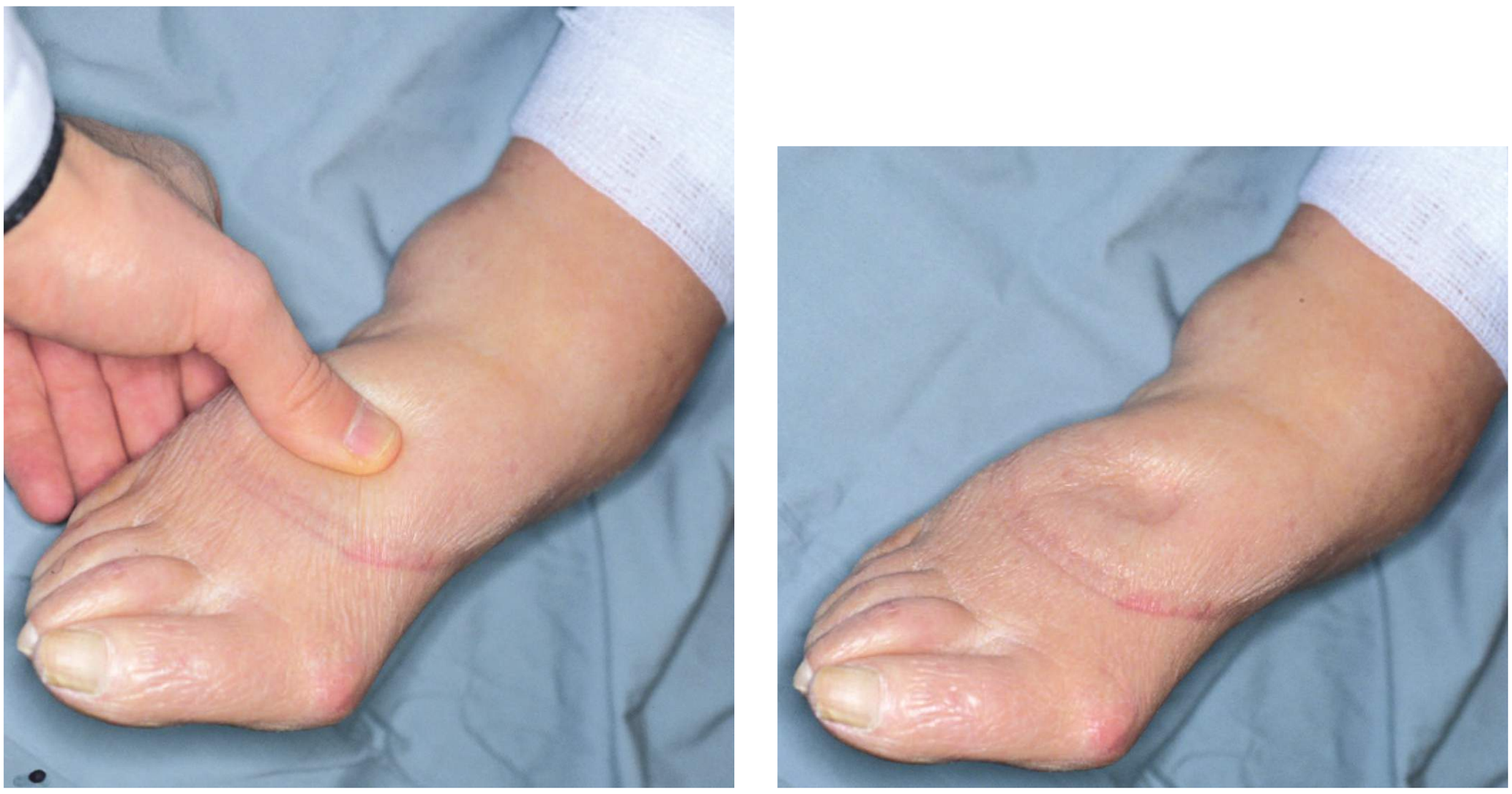

FIGURE 12.21 ■ **Lymphedema.** Pitting edema is seen in a woman with lymphedema of the lower extremities. Note how the impression of the thumb remains on the foot in this patient with lymphedema. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

Bursitis is an inflammatory reaction in a fluid-filled synovial sac, commonly over the subacromial, prepatellar, olecranon, or trochanteric bursa. It is associated with repetitive motion, trauma, or infection. The fluid collection may be bacterial (septic bursitis), gouty, or, most commonly, inflammatory. It produces pain, tenderness, swelling, warmth, and limited range of motion. It is critical to differentiate septic from benign inflammation.

Since bursitis does not involve the intra-articular space, signs and symptoms should be isolated to the bursal area. Typically, intra-articular involvement is associated with pain on minor range of motion, while the discomfort of bursitis occurs with skin and synovial sac stretching at extreme ranges of joint movement. When this differentiation is difficult, fluid aspiration and analysis for cell count, Gram stain, protein, glucose, and polarized microscopy (see Crystal-Induced Synovitis) may be helpful. Fluid with >50,000 cells per cubic millimeter, polymorphonuclear neutrophil predominance, increased protein, reduced glucose, and a positive Gram stain are associated with bacterial infection.

Management and Disposition

Rest, bulky compression dressings, and nonsteroidal anti-inflammatory drugs (NSAIDs) are initially used. Bursal injection of local anesthetics mixed with corticosteroids can be considered if septic bursitis has been ruled out, usually in patients who have failed treatment with NSAIDs. Reducing the effusion volume by aspiration may provide temporary relief, although it often recurs. Septic bursitis requires aspiration, gram-positive antibiotic coverage, and consideration of open incision and drainage by orthopedic surgery. Most patients can be treated as outpatients with follow-up.

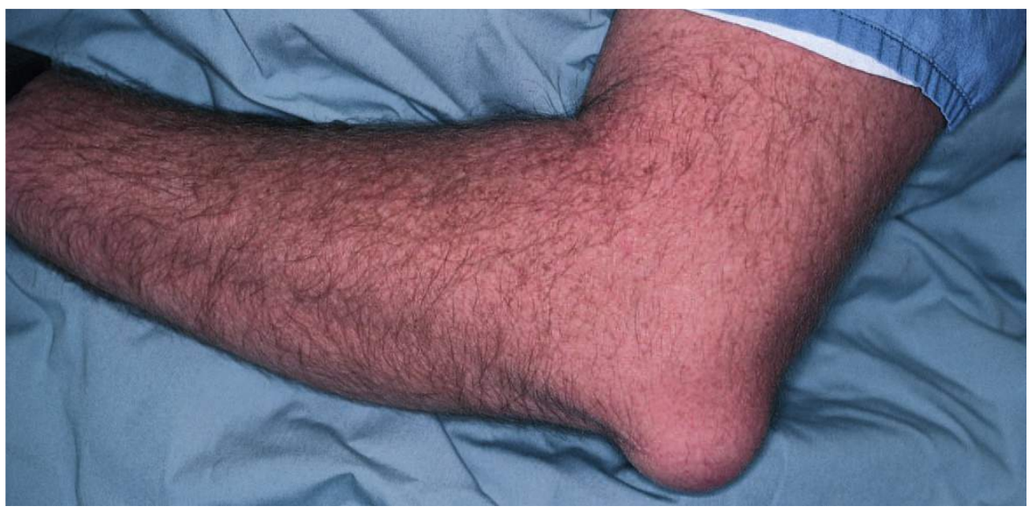

FIGURE 12.22 ■ **Olecranon Bursitis.** Enlarged olecranon bursa in this patient with bursitis. (Photo contributor: Selim Suner, MD, MS.)

Pearls

1. Septic joint infections in patients with cancer, who are taking corticosteroids, or who are IV drug users may have lower synovial fluid leukocyte counts (<30,000/mm^3) than usual (>50,000/mm^3).
2. Bursal fluid from a septic bursitis typically has a lower nucleated cell count than septic joint fluid; lower limits of 2000/mm^3 have been proposed.
3. S aureus is the most common etiologic agent.

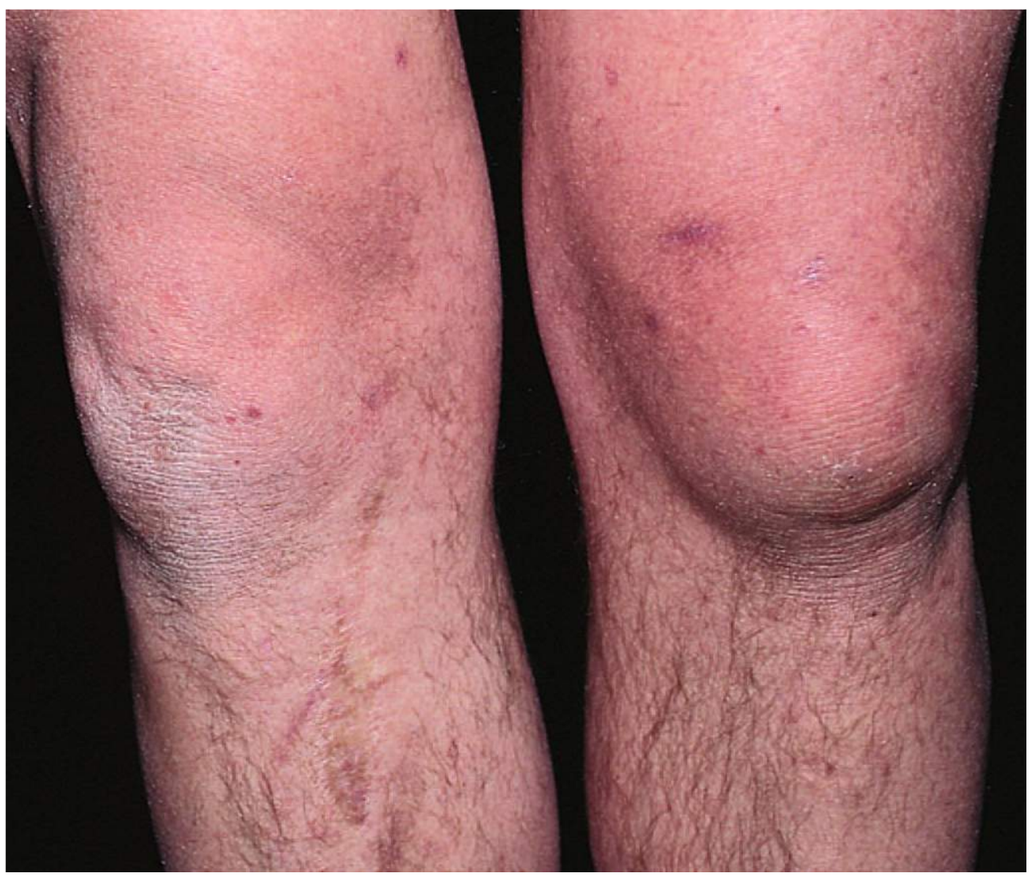

FIGURE 12.23 ■ **Prepatellar Bursitis.** Local bursal swelling is evident over the left knee. (Photo contributor: Kevin J. Knoop, MD, MS.)

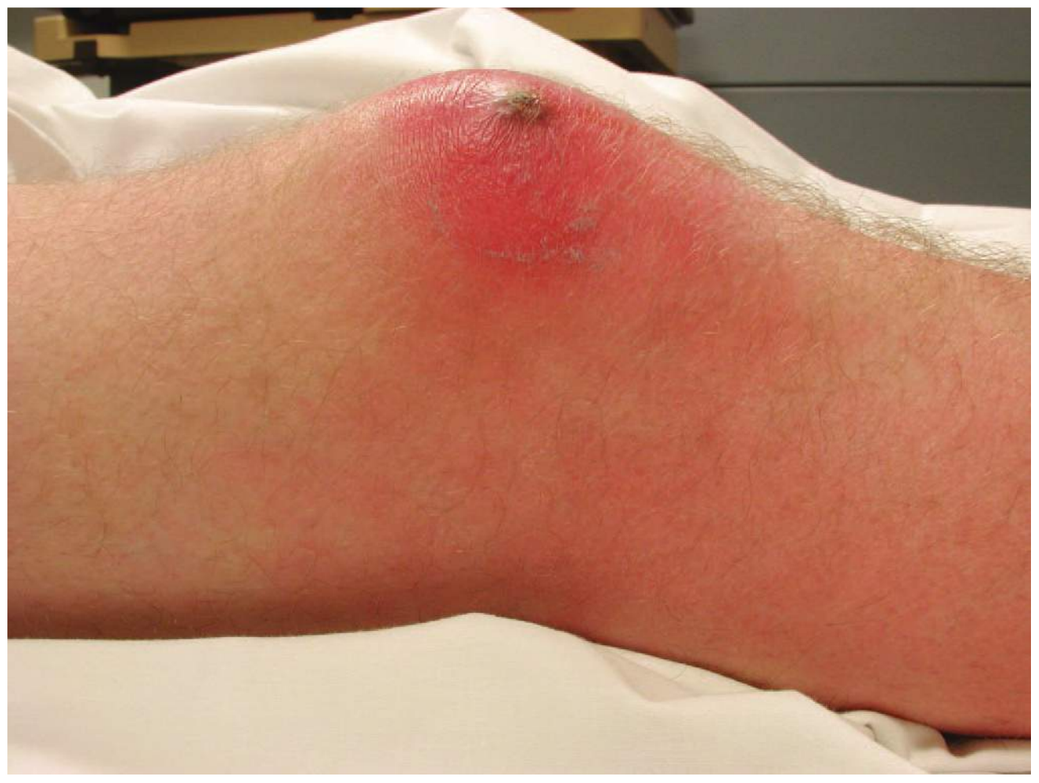

FIGURE 12.24 ■ **Septic Prepatellar Bursitis.** Erythematous, enlarged bursa in this patient with septic prepatellar bursitis. (Photo contributor: R. Jason Thurman, MD.)

PALMAR SPACE INFECTION

Clinical Summary

Palmar space infections occur within the deep fascial spaces of the hand. They commonly arise from puncture wounds, flexor tenosynovitis of the digits, or by hematogenous spread. The palm loses its concavity; tenderness, erythema, warmth, and fluctuance are evident. A thenar space infection is characterized by swelling over the thenar eminence and pain with thumb movement. With a midpalmar space infection, motion is limited and painful for the middle and ring fingers. Infection in the web spaces may involve both the palmar and dorsal sides, forming the classic hourglass-shaped (collar-button) abscess. Cellulitis, local traumatic injury, fractures, and soft tissue mass are included in the differential.

Management and Disposition

All deep space infections of the hand should be managed by a hand surgeon. Prompt incision and drainage in the operating room is necessary for the best outcome. Parenteral antibiotics against S aureus (including CA-MRSA) as well as anaerobes should be started urgently.

Pearls

1. Palmar space infections may cause swelling on the dorsal hand due to the dorsal location of hand lymphatics.
2. There may be minimal signs of swelling over the palmar surface.
3. Infections may be associated with compartment syndrome of the hand.

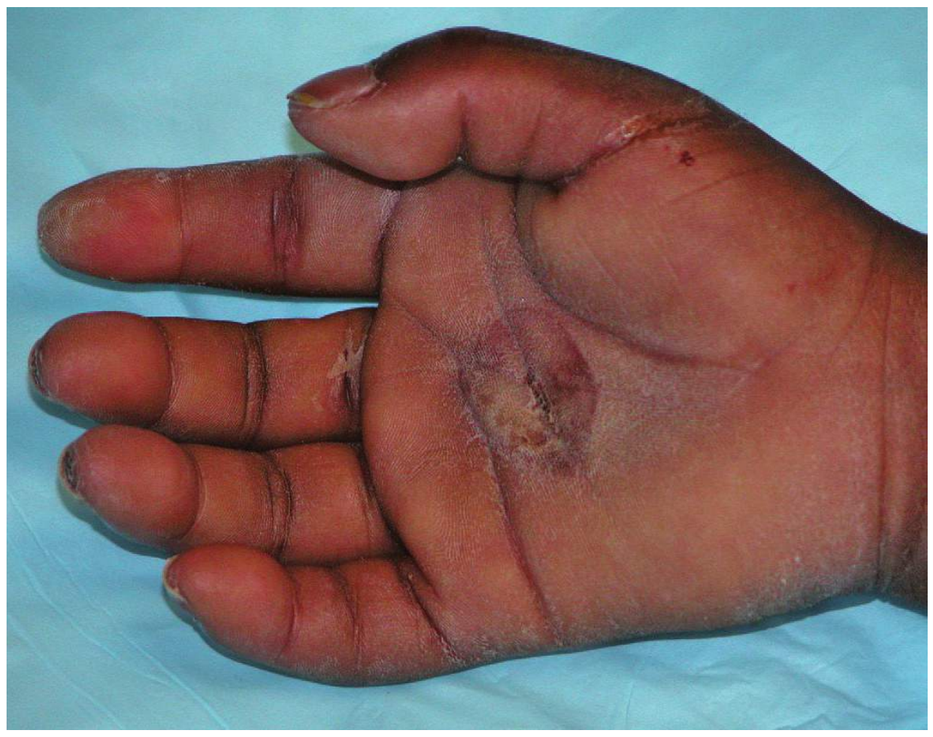

FIGURE 12.26 ■ **Palmar Space Infection.** Diffuse palmar and hand swelling after trauma. (Photo contributor: David Effron, MD.)

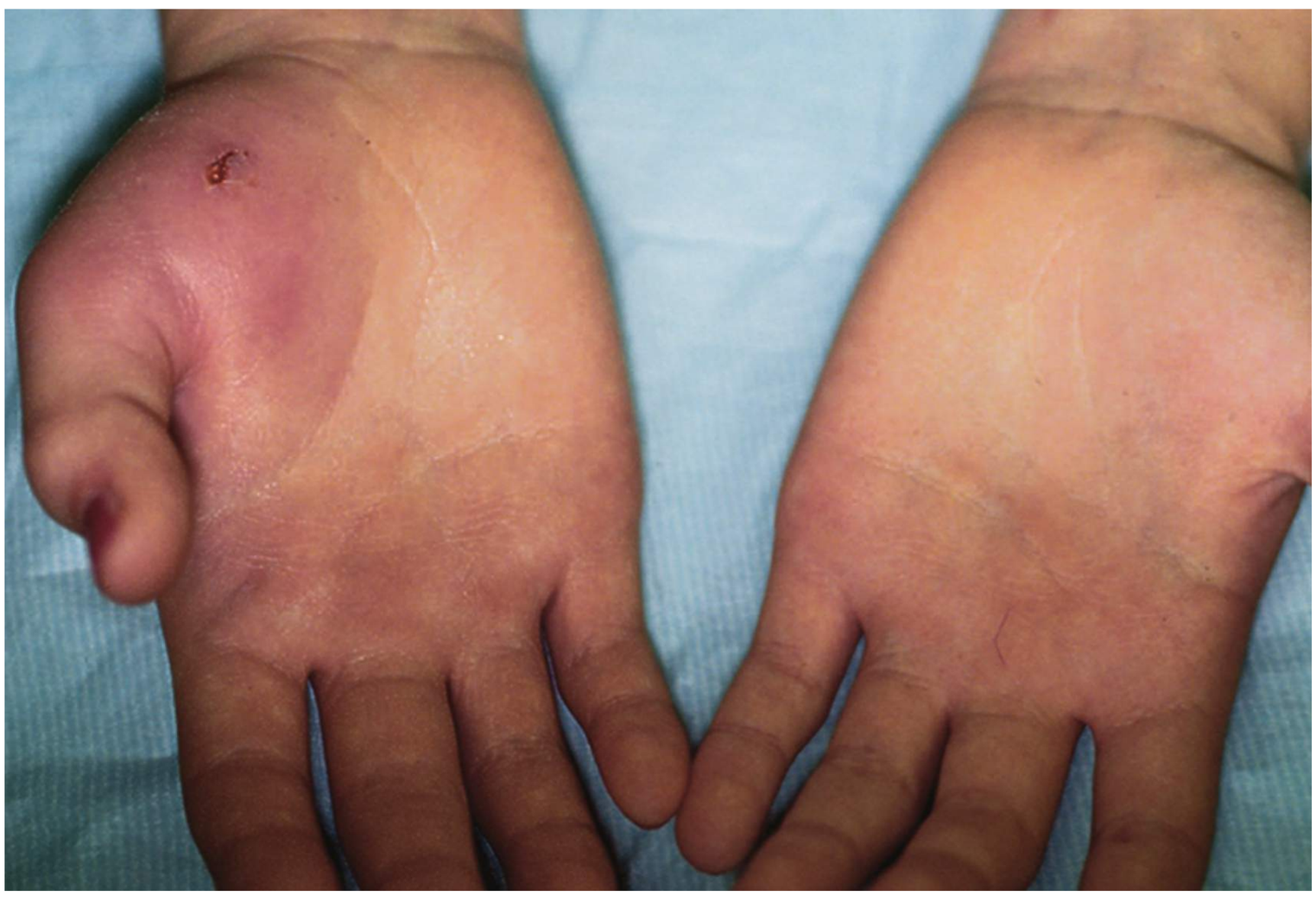

FIGURE 12.25 ■ **Thenar Space Infection.** Thenar space infection following injury to the thumb. In this palmar view, erythema and swelling in the right thenar area is evident. (Photo contributor: Richard Zienowicz, MD.)

Clinical Summary

Tenosynovitis, an inflammation of the tendon and surrounding synovial sheath, is characterized by pain and tenderness. Pyogenic flexor tenosynovitis is a serious tendon sheath infection resulting from puncture wounds, local extension, or hematogenous spread; it is characterized by the four cardinal Kanavel signs: finger held in mild flexion; fusiform digit swelling; tenderness along the entire tendon sheath, especially at the palmar surface of the metacarpophalangeal (MCP) joint; and severe pain with passive extension. Tenosynovitis may be complicated by fibrosis and adhesions leading to stiffness, loss of function, and tendon necrosis.

Inflammatory flexor tenosynovitis is typically due to rheumatoid arthritis, overuse, diabetes mellitus, or connective tissue disorders. The time course is typically more indolent than pyogenic flexor tenosynovitis, although the sequelae can be similar.

Intersection syndrome is tenosynovitis of the radial wrist extensors. It causes pain, swelling, and crepitus of these muscle bellies in the distal dorsoradial forearm.

Management and Disposition

It is difficult to distinguish infectious and noninfectious causes early (24-48 hours). Management of noninfectious includes immobilization and NSAIDs. Broad-spectrum parenteral antibiotics and emergent consultation with a hand surgeon for incision and drainage are mandated with pyogenic flexor tenosynovitis.

Pearls

1. S aureus is the most common organism, but Streptococcus as well as gram-negative and anaerobic organisms may occur.
2. The most specific sign of tenosynovitis is pain with passive digit extension.
3. Patients with immunocompromised states or recently administered antibiotics may not exhibit the classic tetrad of Kanavel signs.

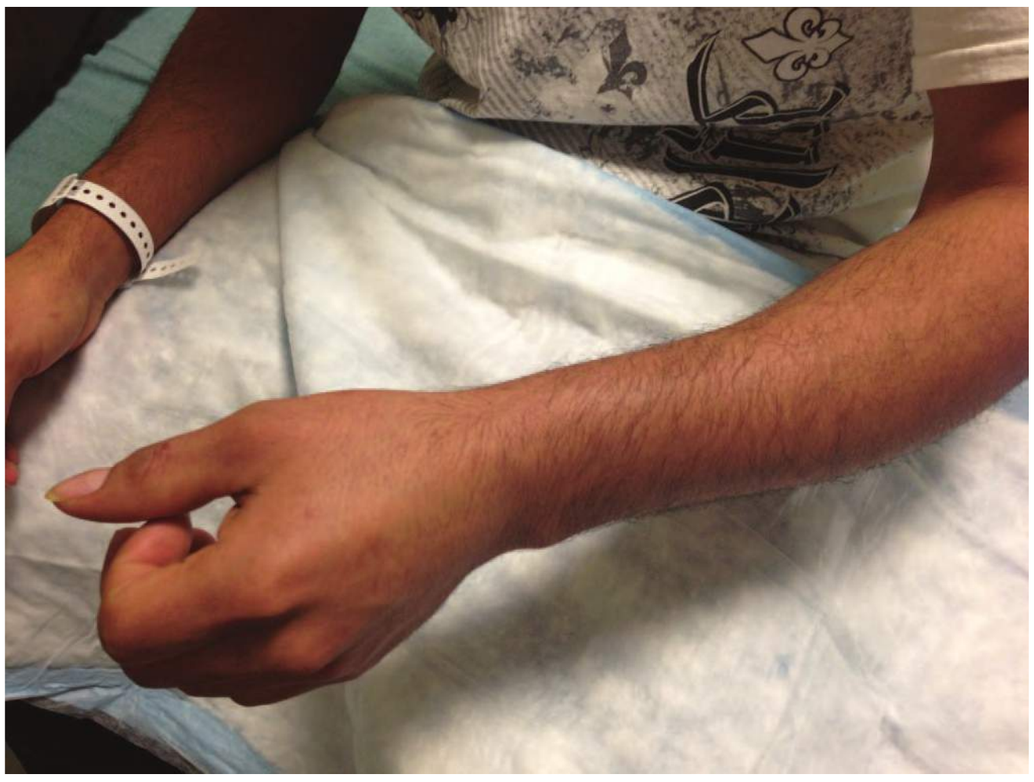

FIGURE 12.28 ■ **de Quervain Tenosynovitis.** Swollen and markedly tender area over radial styloid in de Quervain tenosynovitis. (Photo contributor: Robert Tubbs, MD.)

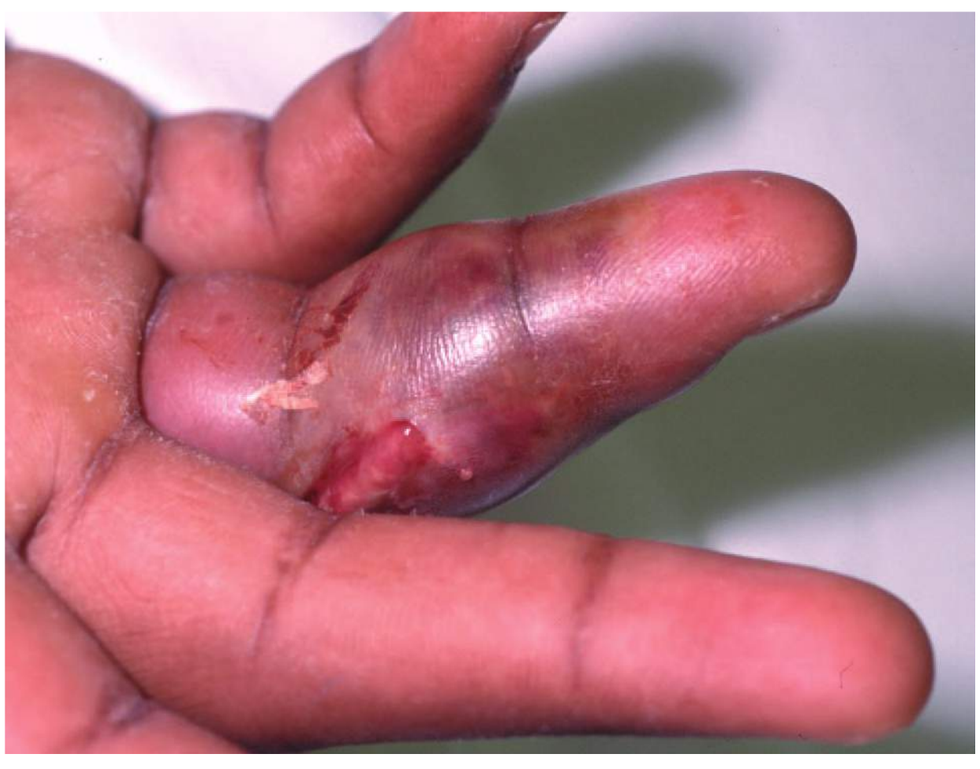

FIGURE 12.27 ■ **Flexor Tenosynovitis.** Pyogenic flexor tenosynovitis of the fourth finger with fusiform swelling, tenderness along the flexor tendon sheath, and pain on flexion. (Photo contributor: Lawrence B. Stack, MD.)

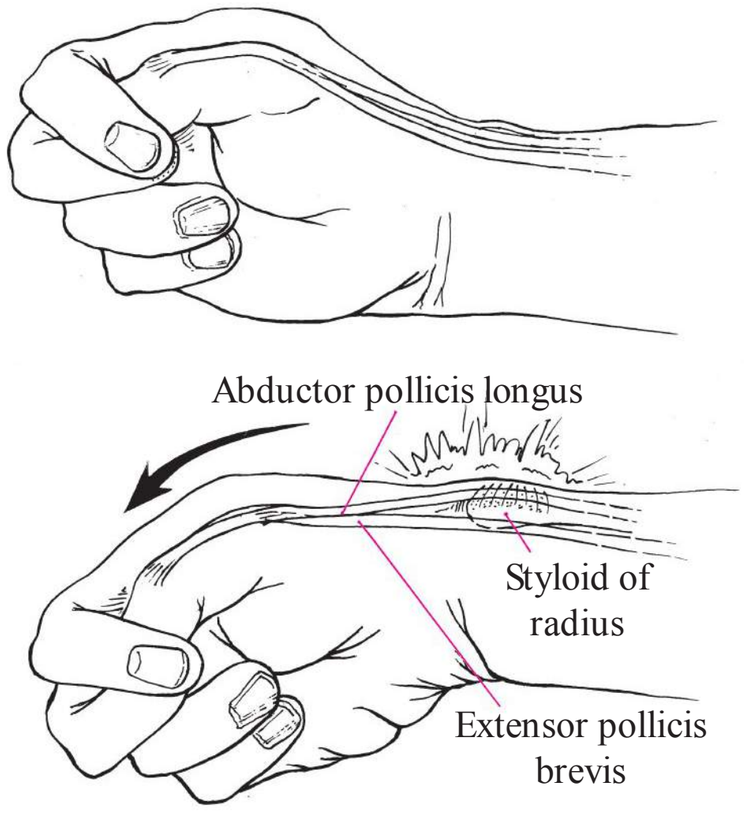

FIGURE 12.29 ■ **Finkelstein Test for de Quervain Tenosynovitis.** Pain over the radial styloid is elicited with ulnar deviation of the wrist as shown.

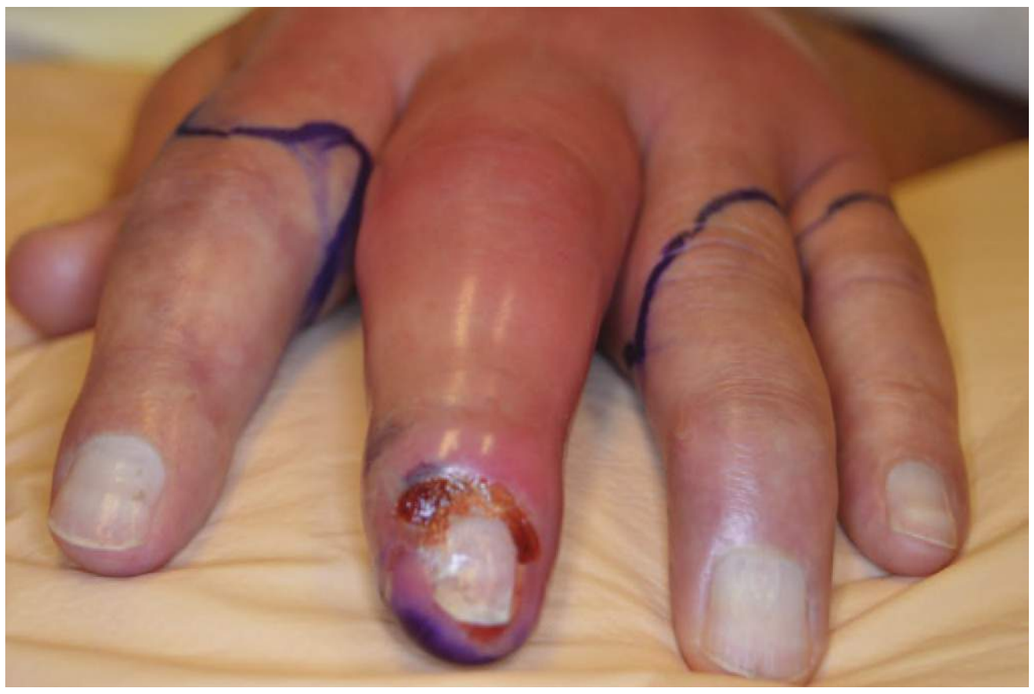

FIGURE 12.30 ■ **Tenosynovitis.** Pyogenic tenosynovitis of the middle finger with fusiform swelling, tenderness along the tendon sheath, and pain on movement. (Photo contributor: Edmond A. Hooker, MD, DrPH.)

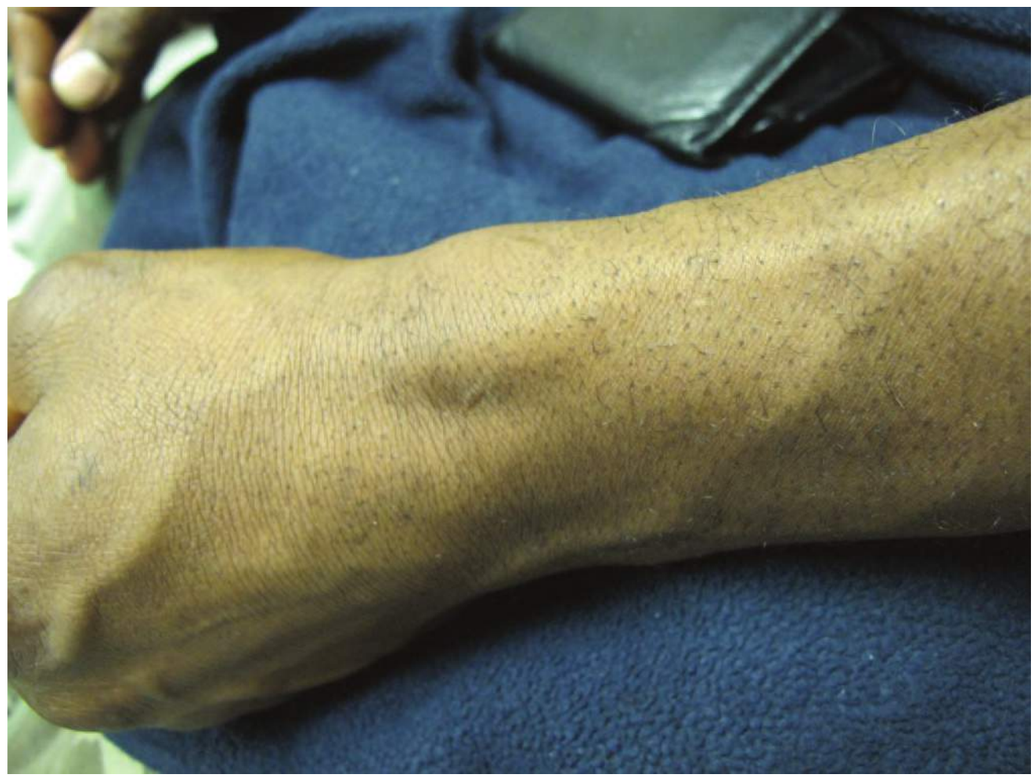

FIGURE 12.31 ■ **Intersection Syndrome.** Discrete swelling at the intersection of muscles that connect to the thumb and underlying wrist tendons. (Photo contributor: Larry Mellick, MD, MS.)

Clinical Summary

Thrombophlebitis is superficial thrombosis and inflammation of veins or varicosities characterized by redness, tenderness, and palpable, indurated, cordlike venous segments. Common associations are IV insertion, irritant IV solutions, trauma, pregnancy, and recent postpartum states. There is little risk of embolism when associated with varicose veins or superficial veins distal to the popliteal fossa. However, pulmonary embolism can occur secondary to thrombus propagation to more proximal veins of the deep venous system, particularly with greater saphenous vein involvement. Lymphangitis, DVT, and cellulitis are in the differential. Septic superficial thrombophlebitis should be considered with a history of IV drug use. The patient should be evaluated for the possibility of DVT if no underlying cause of superficial thrombosis is elucidated.

Management and Disposition

Elevation with warm compresses, rest, and analgesia is sufficient treatment for uncomplicated superficial thrombophlebitis. Superficial thrombophlebitis of the saphenofemoral or iliofemoral system requires treatment as a DVT. Admission is warranted if there is extensive involvement, septic signs, progression of symptoms despite treatment, or severe inflammatory reactions.

Pearls

1. Thrombophlebitis of the greater saphenous vein may be confused with lymphangitis, since the lymphatic drainage from the leg runs along the vein.
2. The superficial femoral vein, despite its name, is considered a deep vein, and thrombosis involving this requires standard DVT treatment.
3. Anticoagulation may be considered for lower extremity thrombophlebitis involving the greater saphenous vein close to the femoral junction.
4. Thrombophlebitis of the upper extremity is unlikely to progress to DVT.
5. Suppurative thrombophlebitis should be suspected with high fevers, signs of extensive erythema or purulent drainage, or IV drug use.

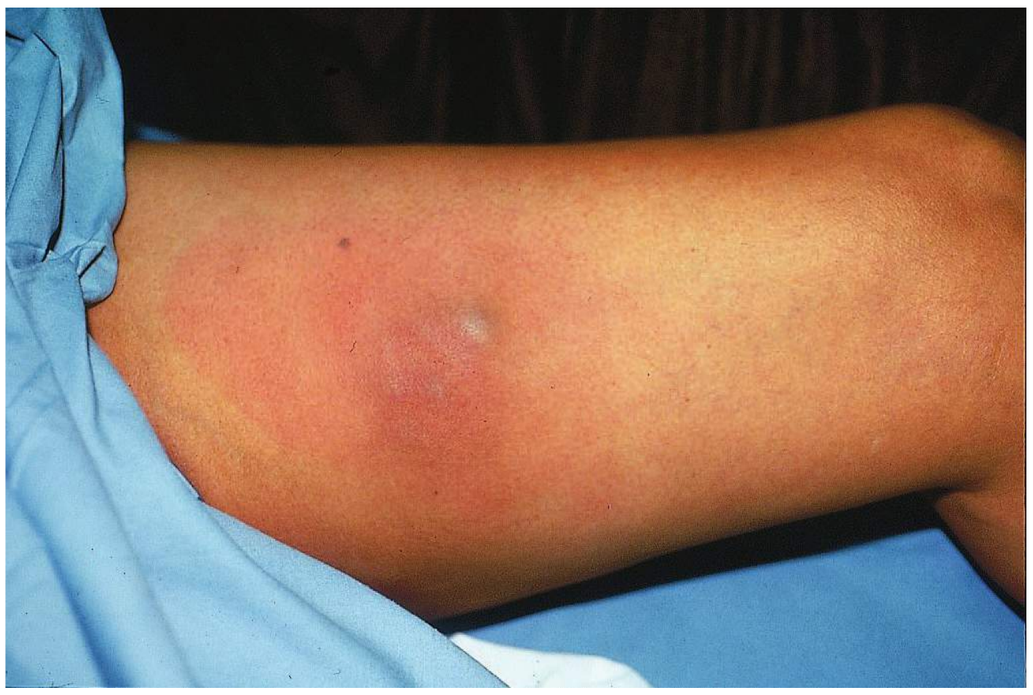

FIGURE 12.33 ■ **Septic Thrombophlebitis.** Thrombophlebitis may be complicated by bacterial infection. Note the purulence associated with the erythematous thrombosed veins. (Photo contributor: Lawrence B. Stack, MD.)

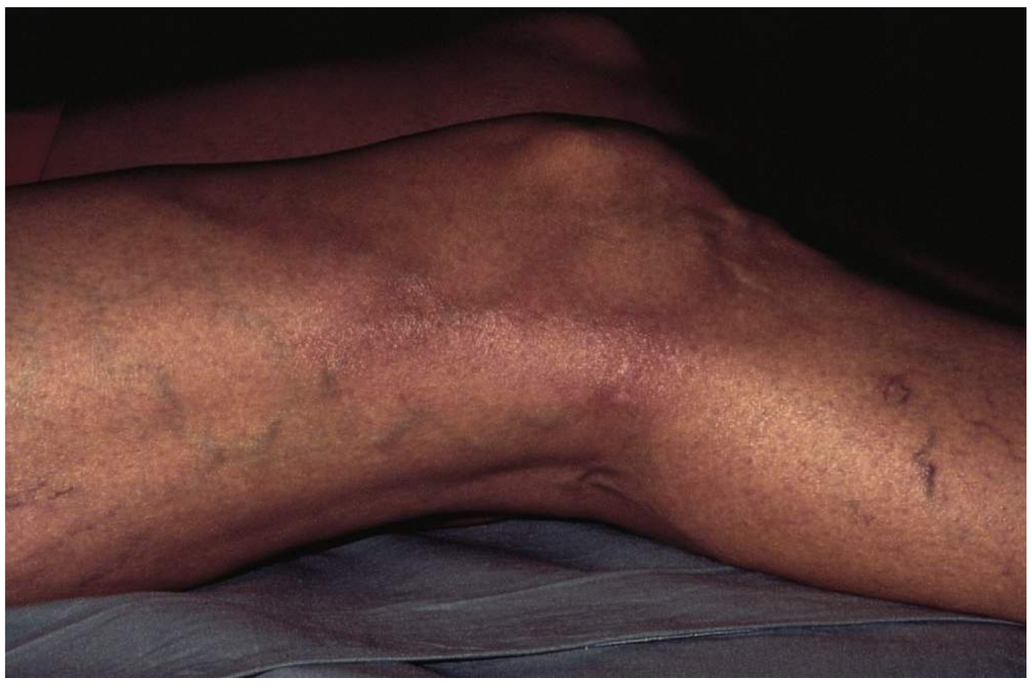

FIGURE 12.32 ■ **Thrombophlebitis.** Thrombophlebitis of the superficial leg veins. The thrombosed veins are erythematous, close to the surface, and palpable. (Photo contributor: Lawrence B. Stack, MD.)

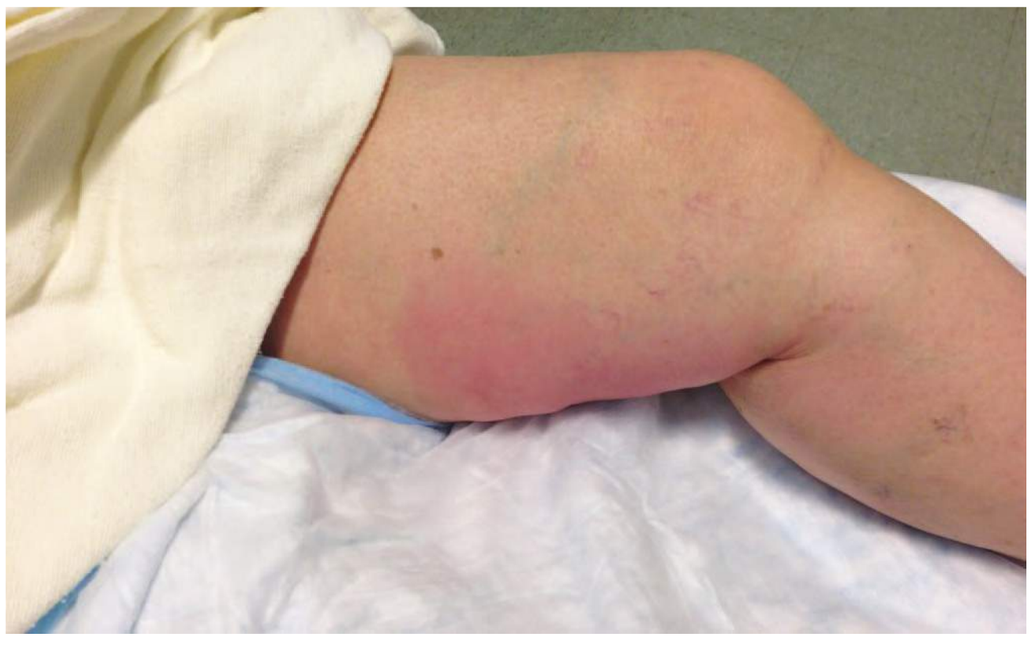

FIGURE 12.34 ■ **Thrombophlebitis.** Erythema and tenderness in the medial thigh of this patient with saphenous thrombophlebitis. This may be difficult to differentiate from a deep venous thrombosis by physical exam alone. (Photo contributor: Robert Tubbs, MD.)

PARONYCHIA

Clinical Summary

Paronychia is the most common hand infection and is characterized by inflammation and pus accumulation along a lateral nail fold. It may spread to involve the eponychium at the base of the nail and the opposite nail fold if untreated. S aureus is most frequently isolated, although the infection is generally mixed flora. Felon, dactylitis, herpetic whitlow, hydrofluoric acid burn, and traumatic injury should be considered in the differential.

Management and Disposition

If paronychia is recognized early, prior to frank abscess formation, warm soaks with or without oral antibiotics may be sufficient. After 2 to 3 days, there may be sufficient pus accumulation along the eponychial fold to warrant incision and drainage. After digital block, a #11 blade or 18-gauge needle is advanced parallel to the nail and under the eponychium at the site of maximal fluctuance. If pus has collected under the nail (subungual abscess), then a portion must be removed to provide drainage. Incisions should be packed open with gauze (removed in 24-48 hours). Oral antibiotics should be prescribed, and the patient reevaluated in 2 to 3 days.

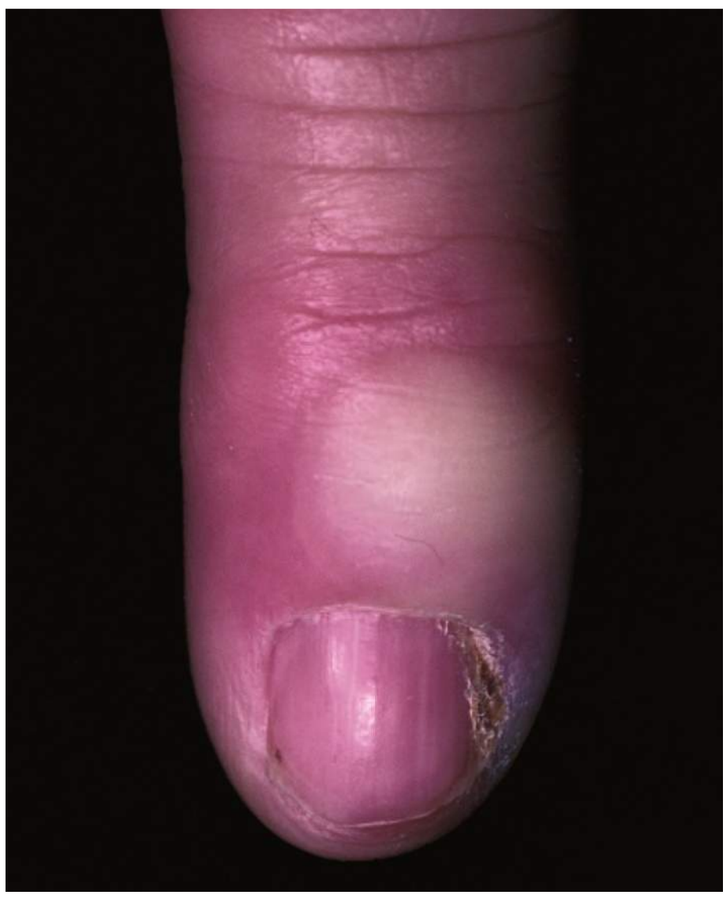

FIGURE 12.35 ■ **Paronychia.** A paronychia involving one lateral fold and the eponychium. There is swelling, erythema, and tenderness on the dorsum of the distal phalanx. (Photo contributor: Frank Birinyi, MD.)

Pearls

1. Paronychia is associated with nail biting, manicure trauma, and foreign bodies.
2. Germinal matrix damage during nail plate excision may result in nail deformity.
3. It is important to distinguish a paronychia from herpetic whitlow, where incision and drainage is contraindicated.
4. Paronychia may occur in the toes as well as the fingers.

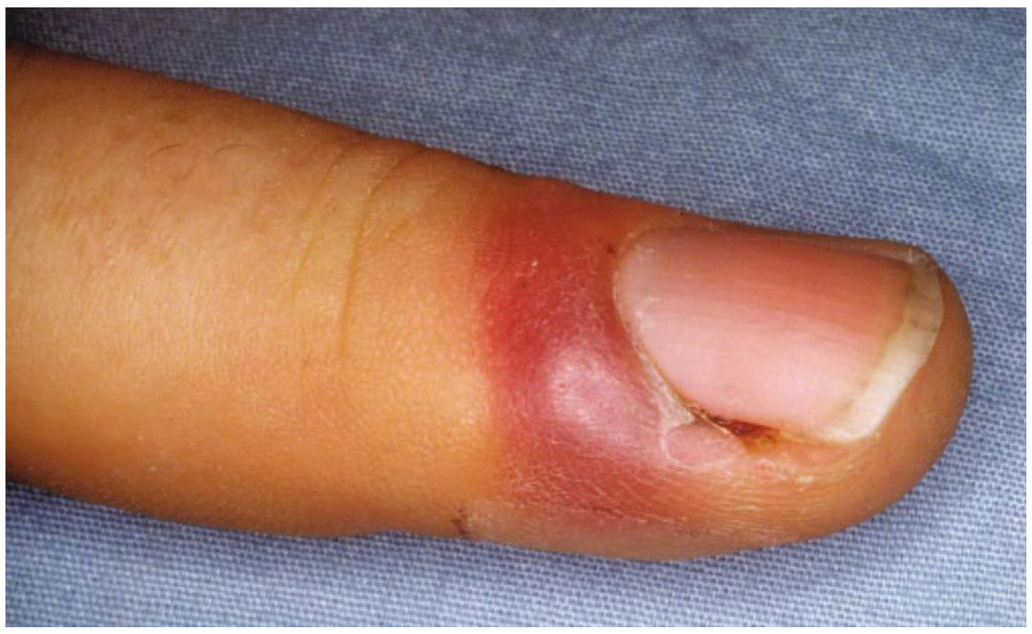

FIGURE 12.36 ■ **Paronychia.** A paronychia involving the medial fold and eponychium. (Photo contributor: Selim Suner, MD, MS.)

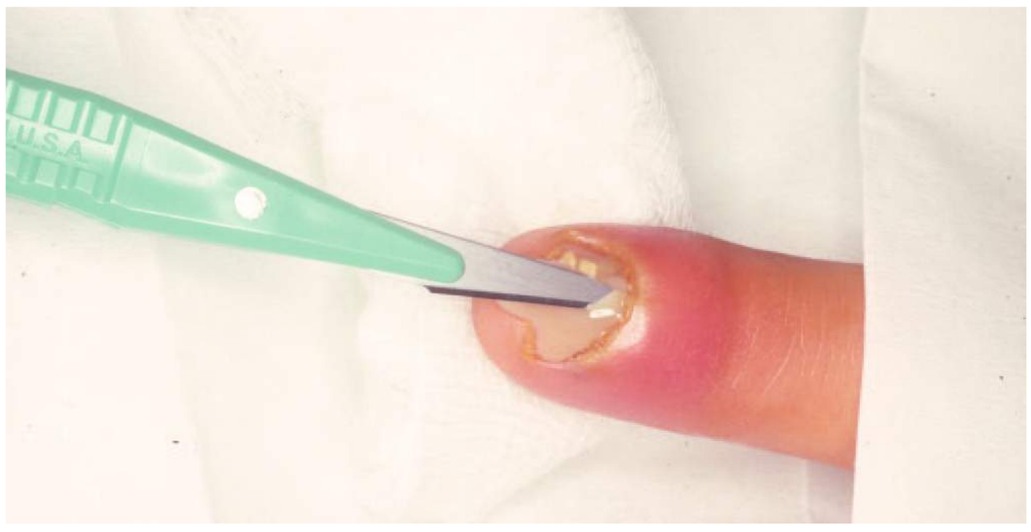

FIGURE 12.37 ■ **Paronychia.** Incision and drainage of a paronychia with considerable purulent drainage. (Photo contributor: Selim Suner, MD, MS.)

SUBCLAVIAN VEIN THROMBOSIS

Clinical Summary

Thrombosis of the subclavian vein (Paget-von Schrötter syndrome) is an uncommon condition, typically occurring in young patients following exercise and compression injury to the subclavian or axillary vein from a narrow thoracic outlet (effort thrombosis). Pain, tightness, and arm swelling are manifest within a day. Pitting edema develops in the fingers, hand, and forearm. There is no arterial insufficiency, and the pulses are palpable. This syndrome is separate from iatrogenic upper extremity thrombosis, generally as a result of vascular access catheters. There is a 15% risk of developing pulmonary embolism from thrombosis of upper extremity veins; however, large or fatal emboli are very rare. Ultrasound has become the screening test of choice. Superior vena cava syndrome, upper extremity trauma, heart failure, angioedema, and lymphatic obstruction are in the differential.

Management and Disposition

Treatment consists of hospital admission, elevation, local heat, analgesia, and anticoagulation with heparin for patients presenting with long-term thrombosis. In cases of acute thrombosis (within 5 days of symptom onset), thrombolysis with direct catheter infusion of thrombolytic agents or mechanical clot retrieval may be considered. Surgical thrombectomy has also been employed. Operative correction of anatomic abnormalities should be accomplished to prevent long-term morbidity.

Pearls

1. Swelling of the neck and face may signify thrombosis or compression of the superior vena cava.
2. The superficial veins in the upper extremity are often distended and do not collapse when the arm is elevated.
3. There is a greater incidence of subclavian vein thrombosis in men and in the right arm.
4. Ultrasound may be limited in visualizing nonocclusive mural thrombi, or those located in the proximal subclavian or innominate veins.

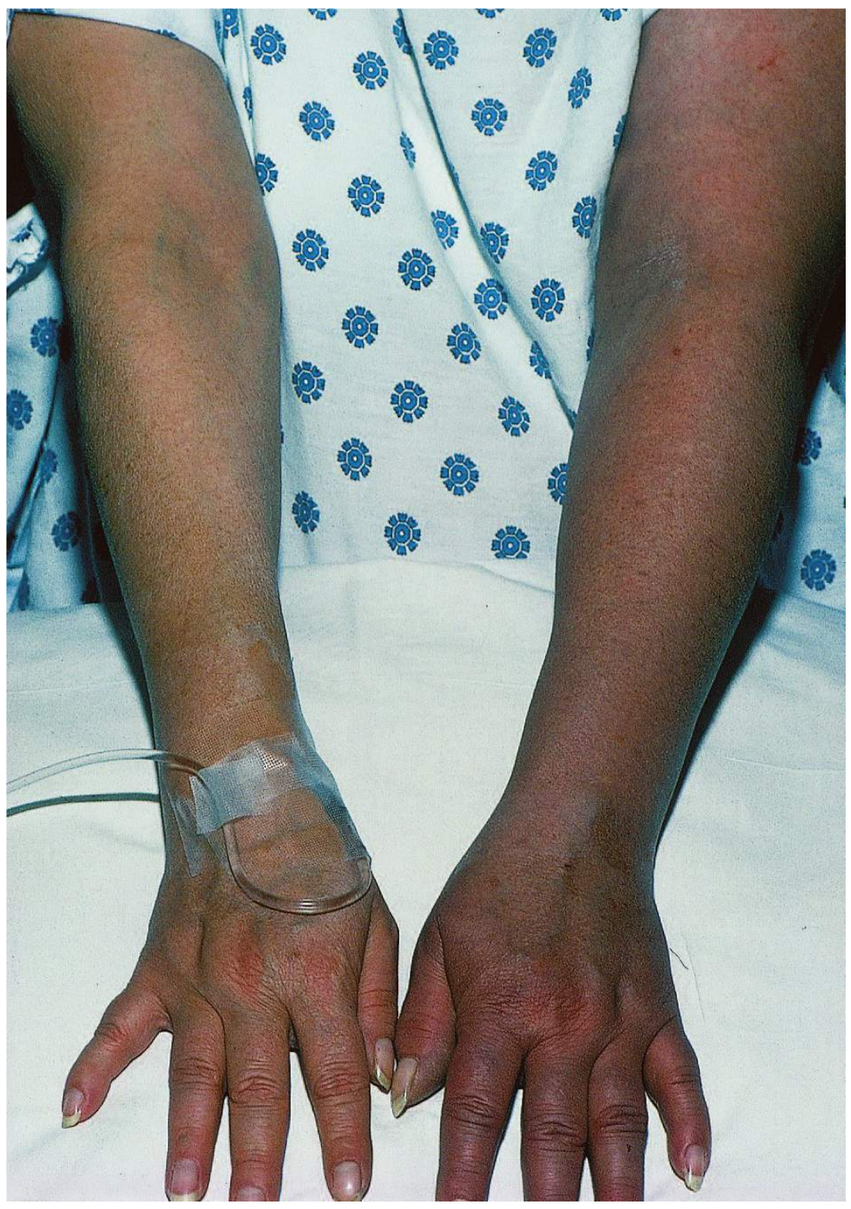

FIGURE 12.38 ■ **Subclavian Vein Thrombosis.** Left subclavian vein thrombosis manifested by swelling of the upper extremity. (Photo contributor: Frank Birinyi, MD.)

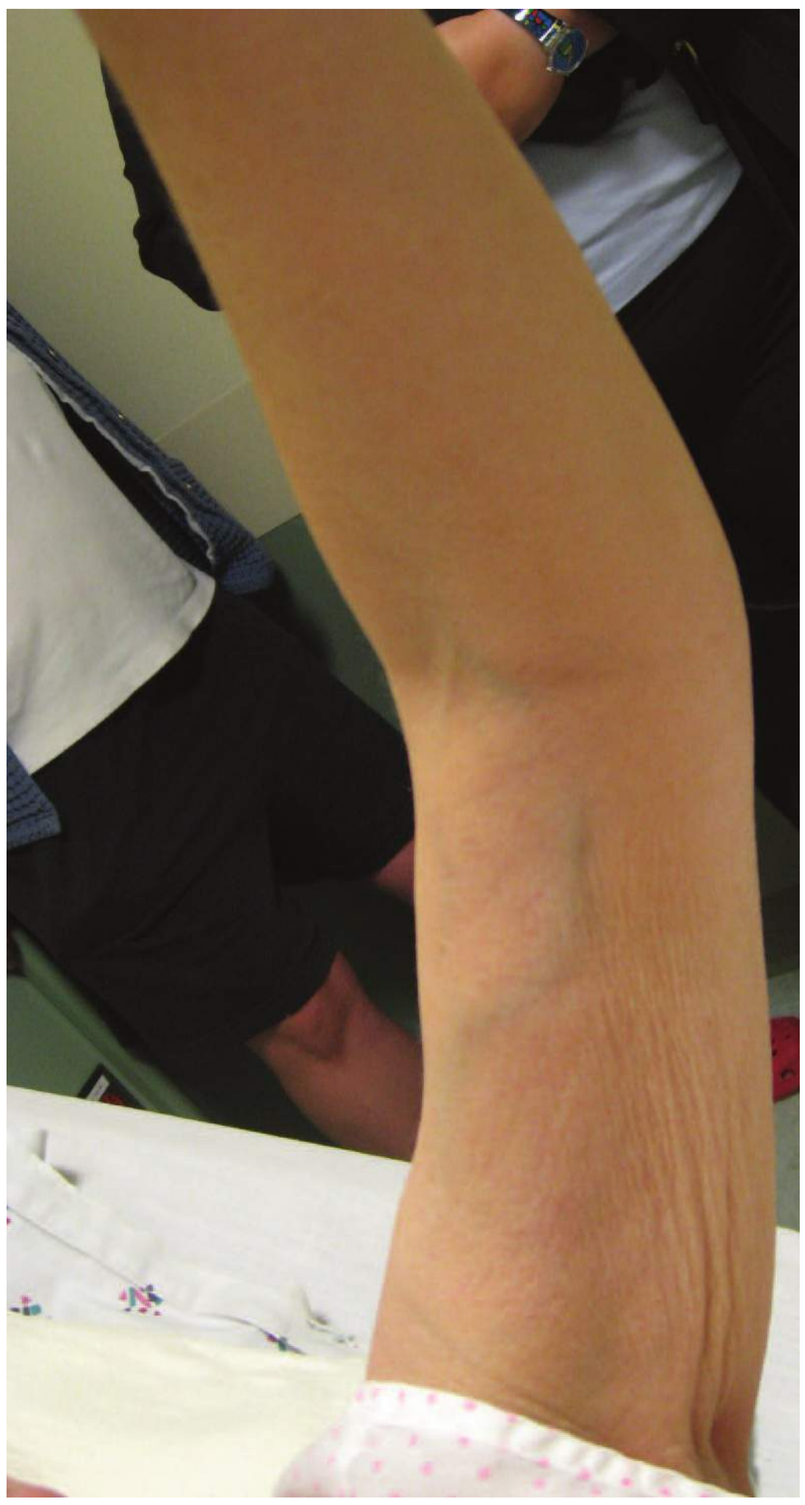

FIGURE 12.39 ■ **Subclavian Vein Thrombosis.** Diffuse arm swelling and dilated superficial veins. Note that the veins remain dilated even when the arm is elevated. (Photo contributor: Robert Tubbs, MD.)

CERVICAL RADICULOPATHY

Clinical Summary

Cervical radiculopathy is caused by compression of a nerve root by a laterally bulging or herniated intervertebral disk, osteoarthritis, or degenerative spondylosis. Pain results from injury to the nerve roots and nerves innervating the dura, ligaments, facet joints, and bone. Common clinical features include pain, paresthesia, and root signs (sensory loss, lower motor neuron muscle weakness, impaired reflexes, and trophic changes). The pain is sharp, stabbing, and worse with cough; it often radiates over the shoulder and down the arm. Frequently, there is numbness and tingling following a dermatomal distribution. Root signs may be found corresponding to anatomic distribution. MRI is the test of choice to distinguish cervical radiculopathy from disc and bone disease. Electromyelography studies may also be helpful in ruling out other disease processes. Trauma, myelopathy, plexopathy, neurofibromatosis, metastatic tumor infiltration of nerve roots, neoplasm, shingles, and central cord syndrome should be considered in the differential.

Management and Disposition

Emergency treatment includes pain control and referral to an orthopedic surgeon or neurosurgeon. Although management often requires narcotic analgesics, appropriate doses of NSAIDs should also be initiated in patients without contraindications. Since prolonged nerve root compression can lead to permanent deficits, immediate referral is necessary for progressive neurologic signs. Patients with intractable pain, progressive weakness, and myelopathy should be admitted.

Pearls

1. Most radiculopathies resulting from cervical disk disease are seen in the age group 30 to 60 years and in the C5 to C7 region.
2. Patients with acute cervical radiculopathy may present with their upper extremity supported by their head to counteract the cervical root distraction caused by the weight of their dependent extremity.
3. CT myelography may be the next most appropriate study in patients with a contraindication to MRI.

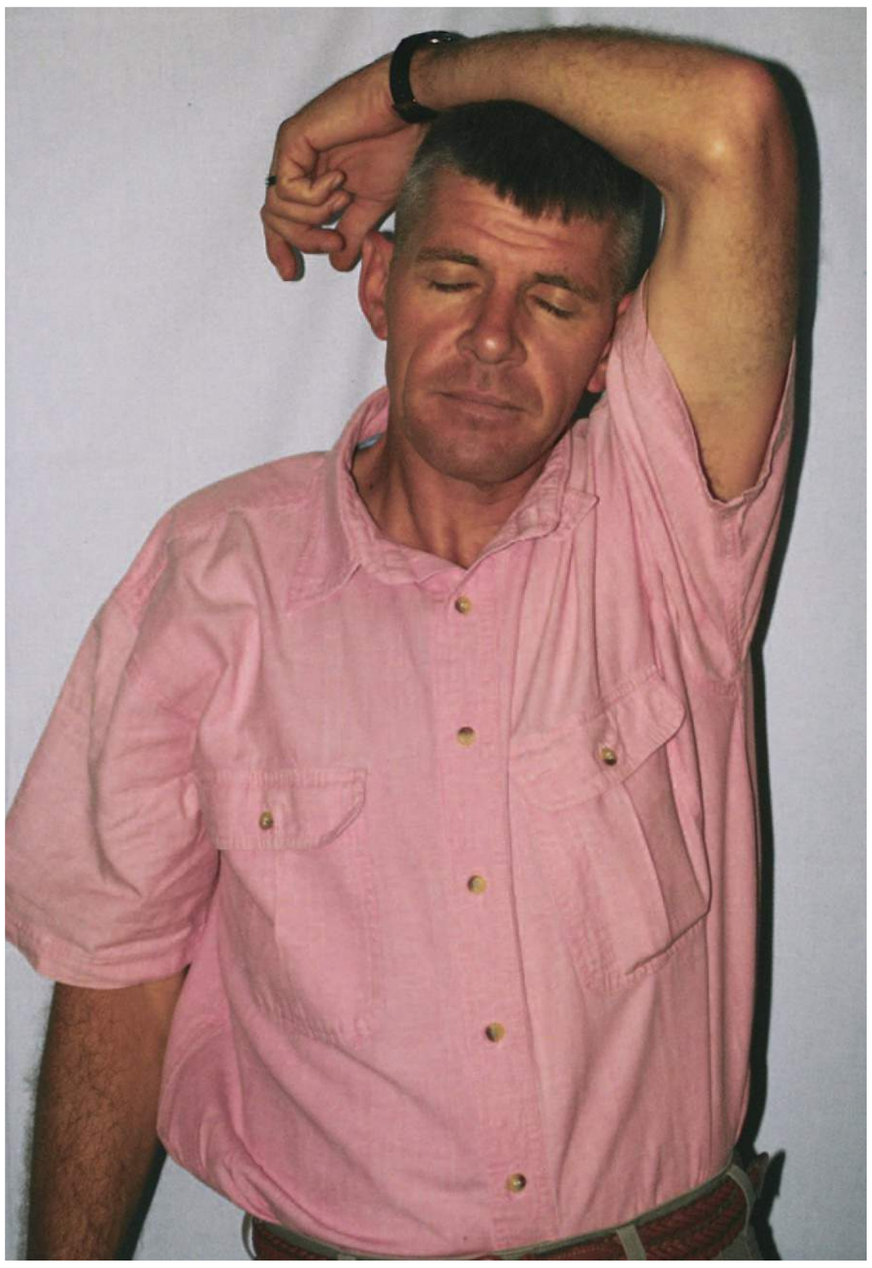

FIGURE 12.40 ■ **Cervical Radiculopathy.** This is the classic position of relief for cervical radicular pain. This patient presented with severe neck pain with radiation to the extremity. The only way the patient was able to get relief was by holding his arm over his head in the position shown. This patient has a C5-C6 herniated nucleus pulposus. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Digital clubbing is characterized by bulbous fusiform enlargement of the distal portion of a digit with loss of the angle between the proximal nail fold and the nail plate (Lovibond angle). The mechanism underlying clubbing is not known. It may be hereditary, idiopathic, or acquired, and is associated with multiple medical conditions including carcinoma, intrathoracic sepsis, bacterial endocarditis, cyanotic congenital heart disease, esophageal disorders, cirrhosis, inflammatory bowel disease, pulmonary disorders, atrial myxoma, multiple pregnancies, and pachydermoperiostosis. The incidence of clubbing with each of these conditions is variable. It may be reversible in certain disease processes.

Management and Disposition

Treatment of the underlying condition is indicated.

Pearls

1. Patients rarely recognize clubbing in their own fingers even if the condition is marked.
2. Pseudoclubbing is an overcurvature of the nails in both longitudinal and transverse axes, with preservation of the angle between the proximal nail fold and nail plate.

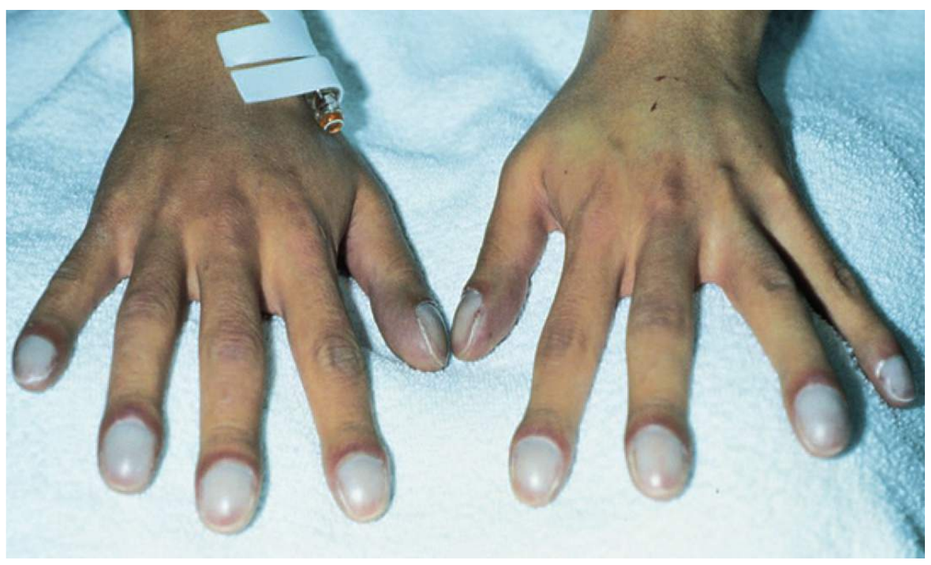

FIGURE 12.41 ■ **Clubbing.** Marked digital clubbing can be seen in this patient. Note the hyperemia in the skin folds around the nail. (Photo contributor: Alan B. Storrow, MD.)

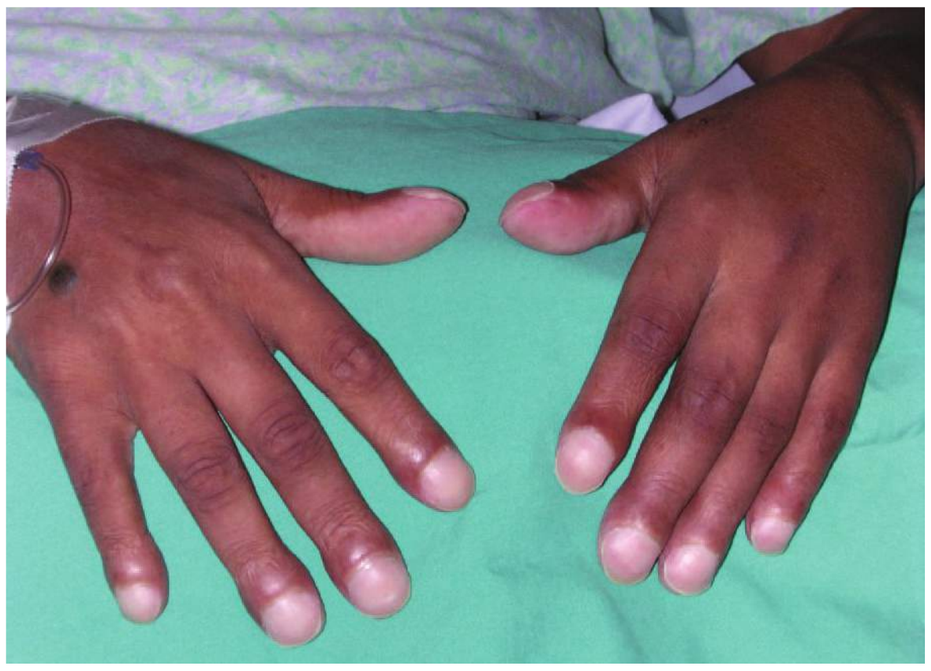

FIGURE 12.42 ■ **Clubbing.** Pronounced digital clubbing. Note the loss of angle between the proximal nail fold and the nail plate (Lovibond angle). (Photo contributor: Robert Tubbs, MD.)

PHLEGMASIA DOLENS

Clinical Summary

Phlegmasia alba dolens (painful white leg, or milk leg) is caused by massive thrombosis of the iliofemoral veins and characterized by pitting edema of the entire lower extremity, inguinal area tenderness, and a pale extremity secondary to arterial occlusion. Phlegmasia cerulea dolens (painful blue leg) arises from massive thrombosis of the lower extremity veins, including the perforators and collaterals, resulting in venous ischemia with a cool, painful, swollen, tense, and cyanotic-appearing lower extremity. Occasionally, there is bullae formation; compartment syndrome and gangrene may follow.

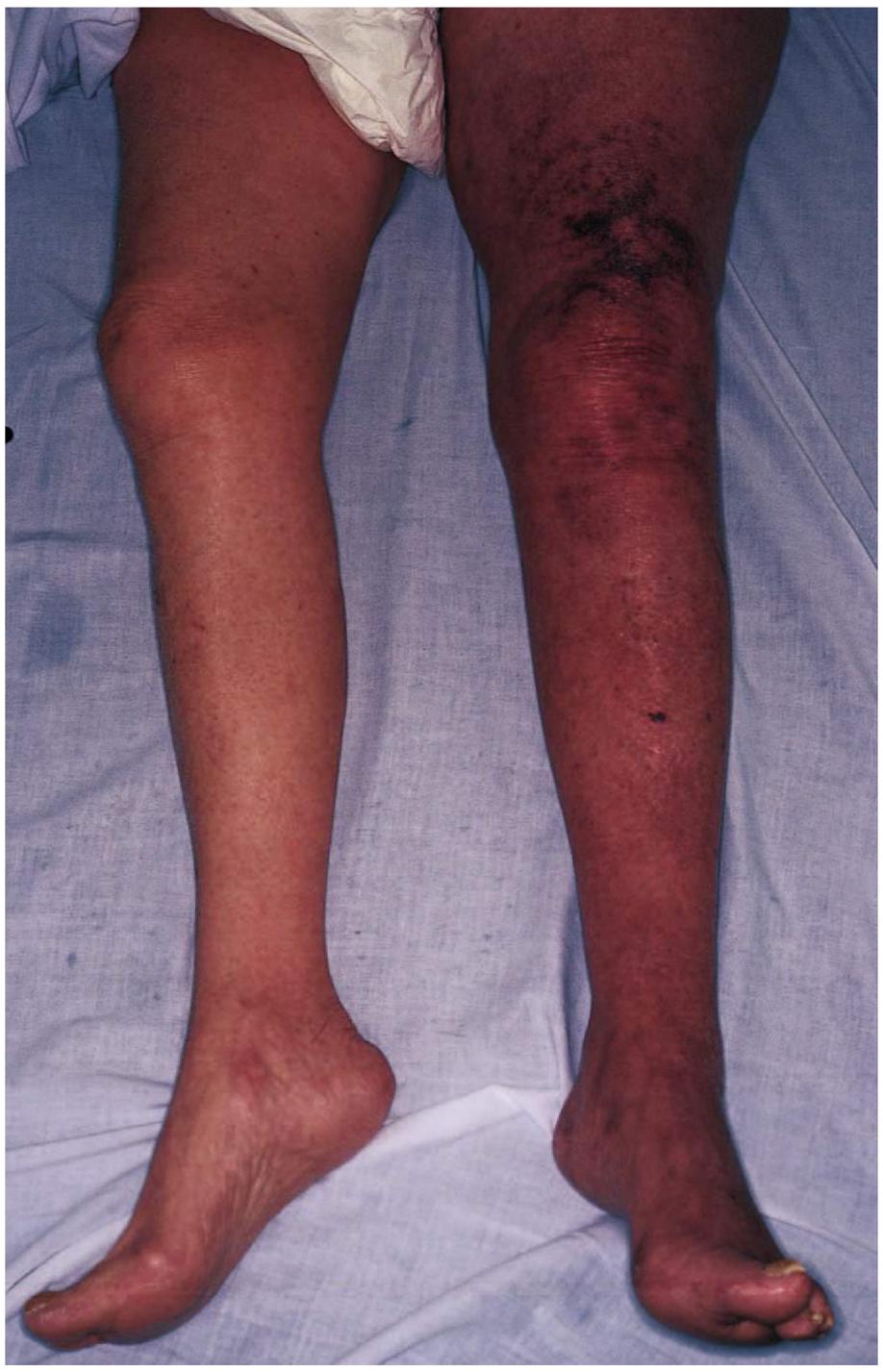

FIGURE 12.43 ■ **Phlegmasia Dolens.** Phlegmasia cerulea dolens of the left lower extremity. Note the bluish discoloration and swelling. (Photo contributor: Daniel L. Savitt, MD.)

The differential includes arterial insufficiency or thrombosis, aortic dissection, abdominal aortic aneurysm, cellulitis, and lymphedema. Doppler ultrasound, impedance plethysmography, and CT venography (most accurate for determining extent) are used in the diagnosis.

Management and Disposition

Systemic anticoagulation with heparin should be initiated immediately; consultation with vascular surgery or interventional radiology should be obtained emergently. There is a significant rate of pulmonary embolization in this condition. In addition, there is a high incidence of postphlebitic syndrome due to venous valvular incompetence. New endovascular techniques for pharmacomechanical clot dissolution are showing promising results for treating these patients.

Pearls

1. Pregnancy is one risk factor for phlegmasia alba dolens.
2. About 40% of patients with phlegmasia cerulea dolens have an underlying malignancy.
3. Hypotension may result from venous pooling of blood in the lower extremity and diminished venous return.

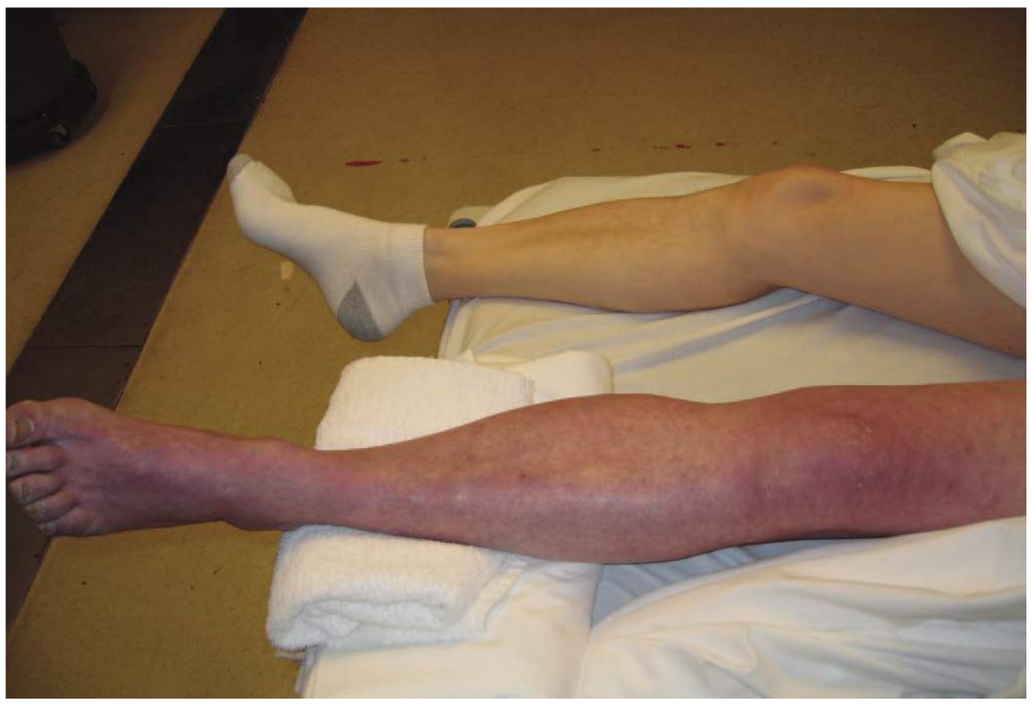

FIGURE 12.44 ■ **Phlegmasia Dolens.** Discoloration and edema secondary to phlegmasia cerulea dolens. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

Deep venous thrombosis (DVT) is often associated with intrinsic coagulopathy, impaired fibrinolysis, recent surgery, or trauma. Other associated conditions include immobilization, increased estrogen (pregnancy, oral contraceptives) with smoking, malignancy, prior DVT, inflammatory disease processes, or coronary artery disease. Intravenous catheters are also a major cause of DVT, particularly in the upper extremity. Unilateral swelling and tenderness, classically in the calf and thigh, are characteristic. Doppler ultrasonography is the screening test of choice in most institutions. In selected low-risk patients, a quantitative d-dimer study may be used to rule out DVT. Cellulitis, lymphedema, heart failure, compartment syndrome, myositis, arthritis, and superficial phlebitis should also be considered in the differential.

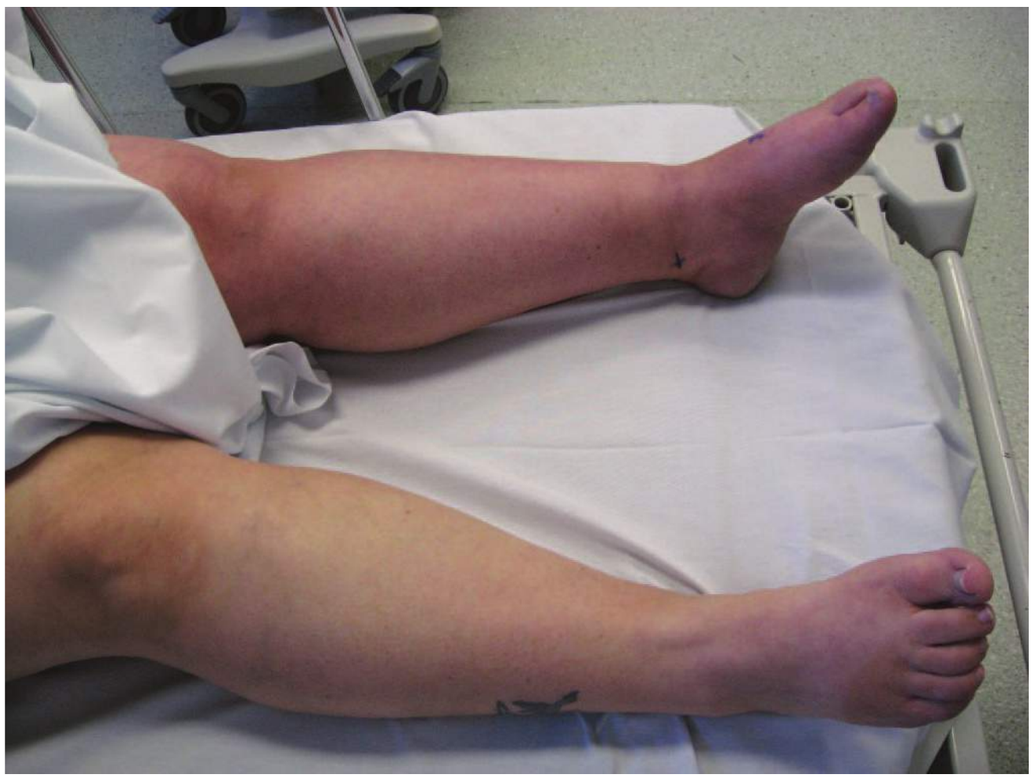

FIGURE 12.46 ■ **Deep Venous Thrombosis.** Erythema and swelling in the left lower extremity. (Photo contributor: Selim Suner, MD, MS.)

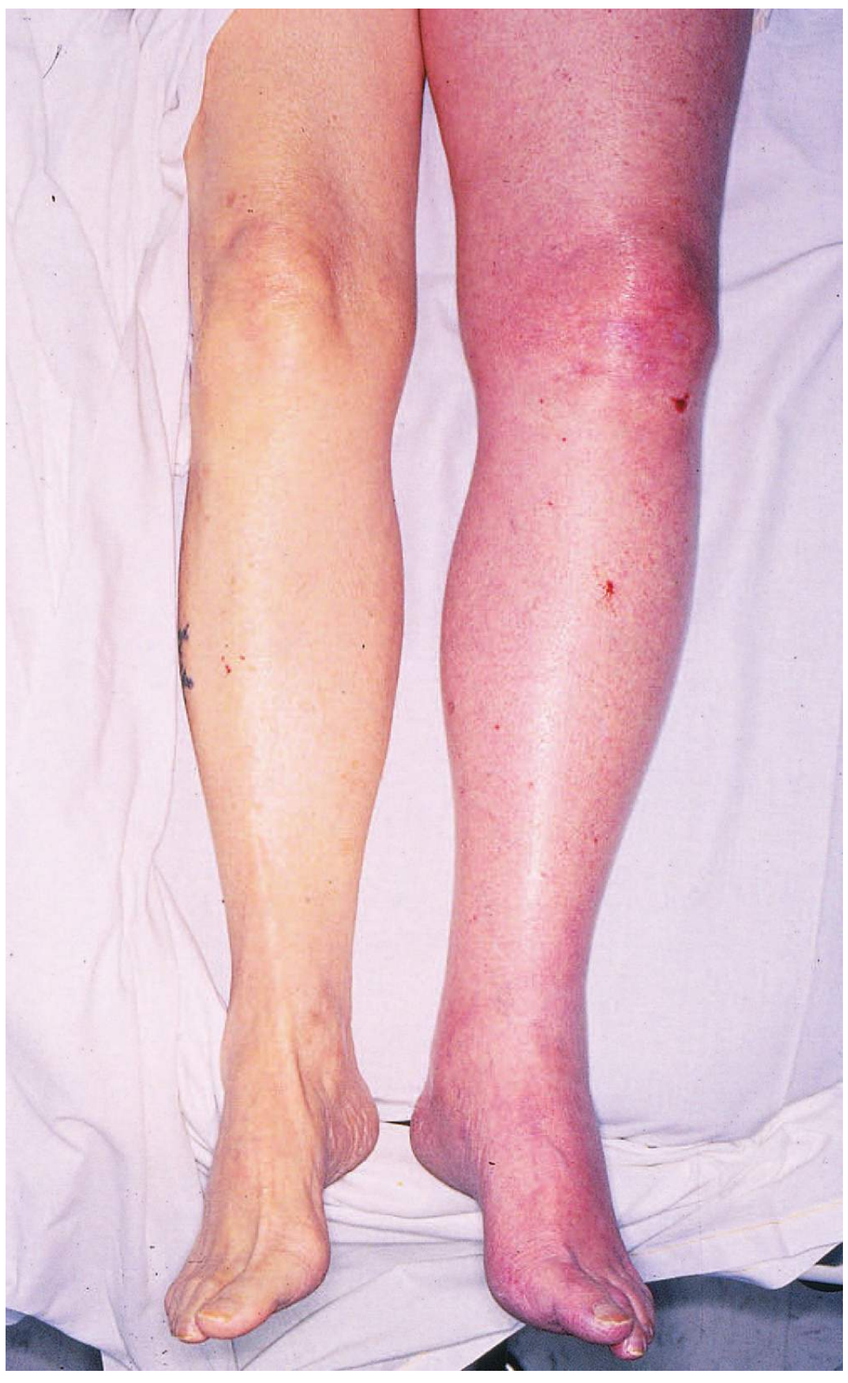

FIGURE 12.45 ■ **Deep Venous Thrombosis.** This patient has some classic findings of left lower extremity DVT: swelling, erythema, pain, and tenderness. (Photo contributor: Kevin J. Knoop, MD, MS.)

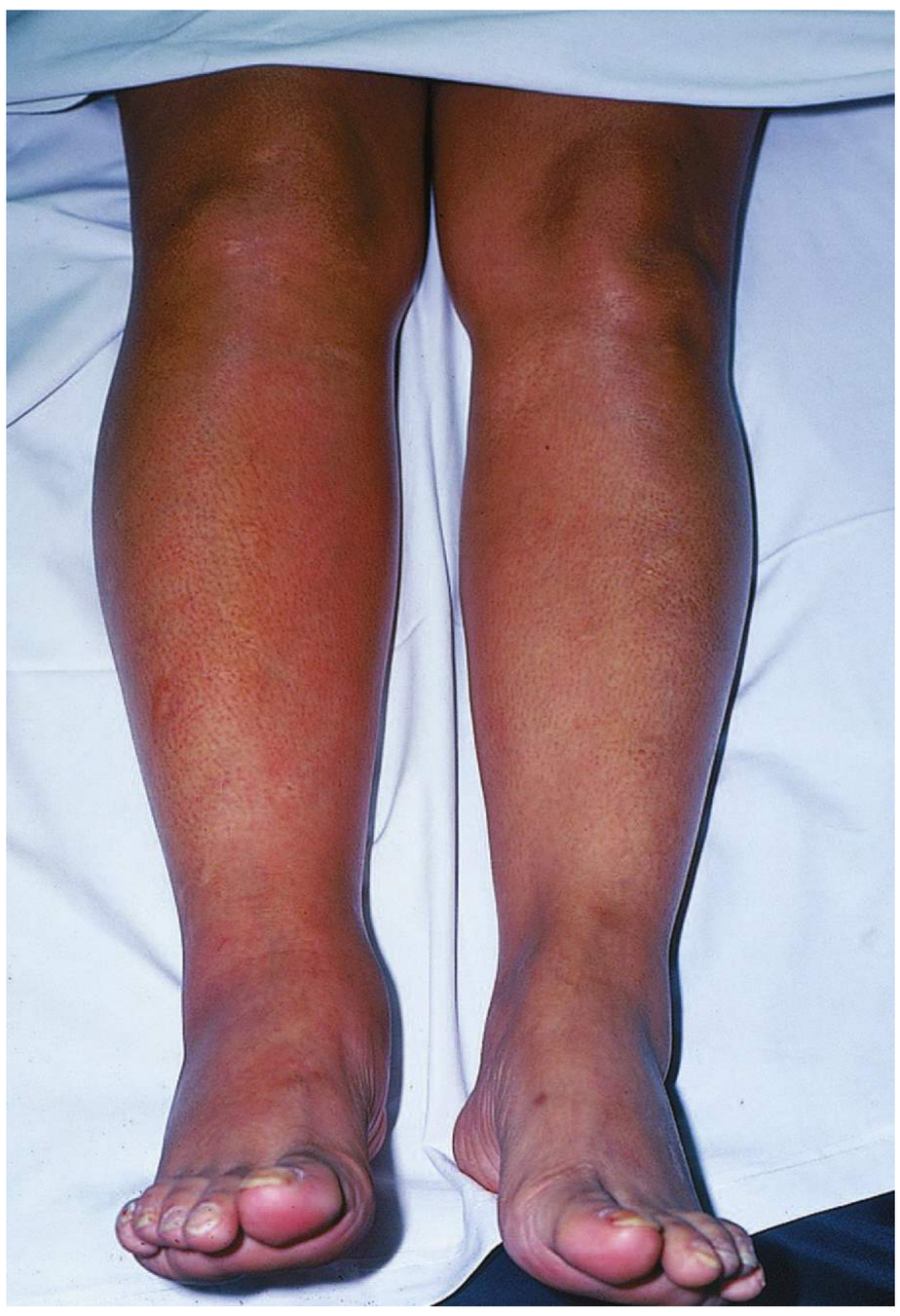

FIGURE 12.47 ■ **Ruptured Baker Cyst**. Comparison of this patient's ankles reveals circumferential swelling around the right side. MRI revealed a ruptured Baker cyst in the right popliteal fossa. Such a presentation may mimic acute lower extremity DVT. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Classic treatment is heparin anticoagulation and admission for warfarin loading. However, low-molecular weight heparin (LMWH) has been shown to be more efficacious for initial treatment. It has also allowed outpatient management in selected patients. In cases of large DVT, consultation with interventional radiology or vascular surgery may be indicated for endovascular thrombectomy. Although DVT in the calf and superficial veins of the lower extremity do not typically embolize, these thrombi can propagate into the deep venous system and may eventually lead to emboli. Serial diagnostic studies are performed to follow the course.

Pearls

1. A Baker cyst, herniation of the synovial membrane through the posterior knee capsule, may rupture, causing unilateral calf swelling similar to DVT.
2. Patients with unexplained DVT should be screened for occult malignancy.
3. Homan sign (calf pain during passive dorsiflexion) is unreliable in the diagnosis.
4. Patients with an unclear diagnosis of cellulitis should have an objective study to rule out DVT.
5. The superficial femoral vein is, despite the name, a part of the deep venous system; thrombosis there requires systemic anticoagulation.

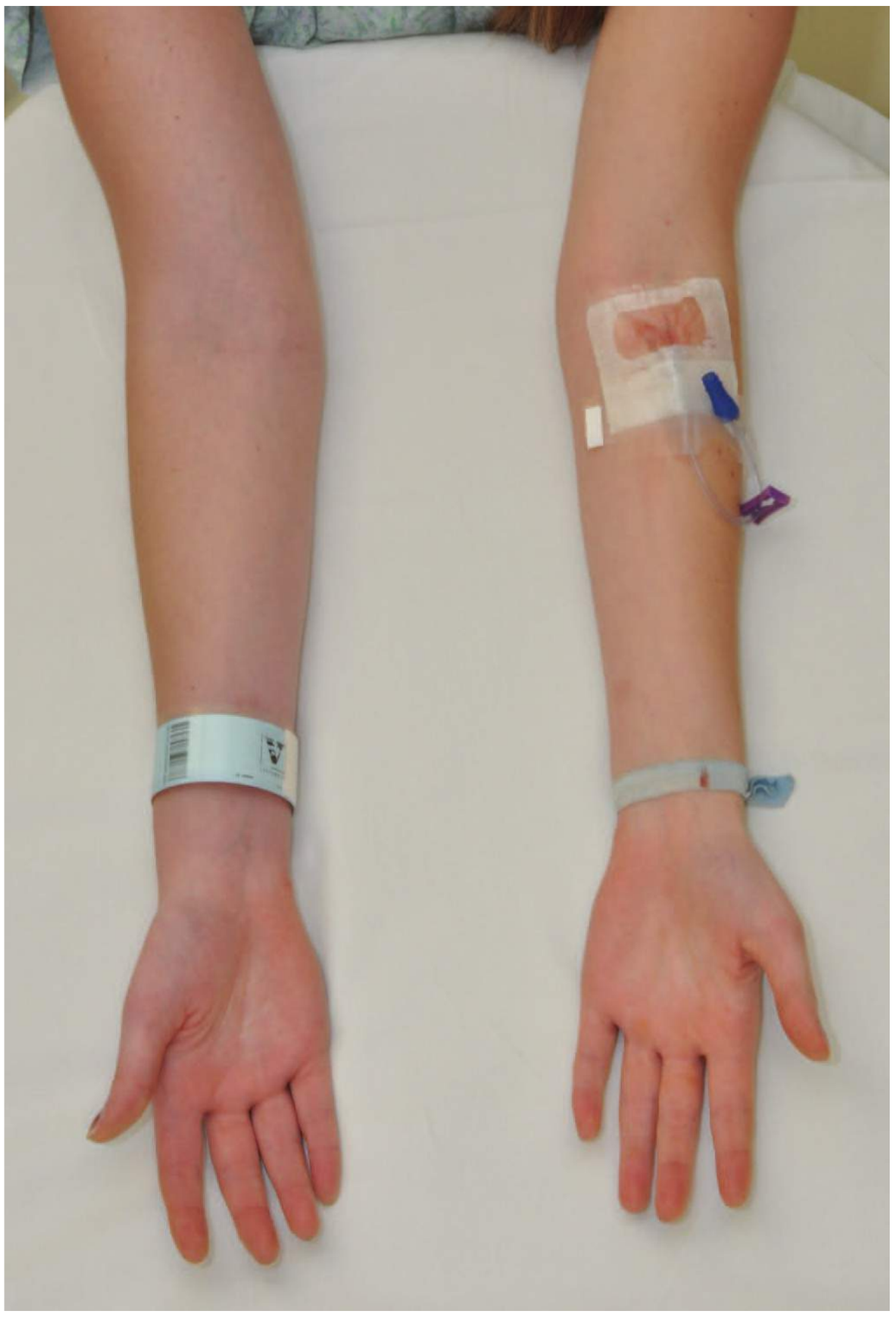

FIGURE 12.48 ■ **Deep Venous Thrombosis.** Swelling and erythema consistent with right upper extremity DVT. (Photo contributor: Hanyuan Shi.)

Clinical Summary

Dupuytren contracture results from shortening and fibrotic changes in the subcutaneous palm tissue and longitudinal bands of the palmar aponeurosis. It may begin as a nodule and then progress to contracture of a finger or fingers. Usually, this is noted at the MCP joint, but the proximal interphalangeal (PIP) or distal interphalangeal (DIP) joint may be involved.

Management and Disposition

The only effective treatment is surgery. Patient education and referral to a hand surgeon is recommended. Recurrence and development of a contracture in other areas may occur.

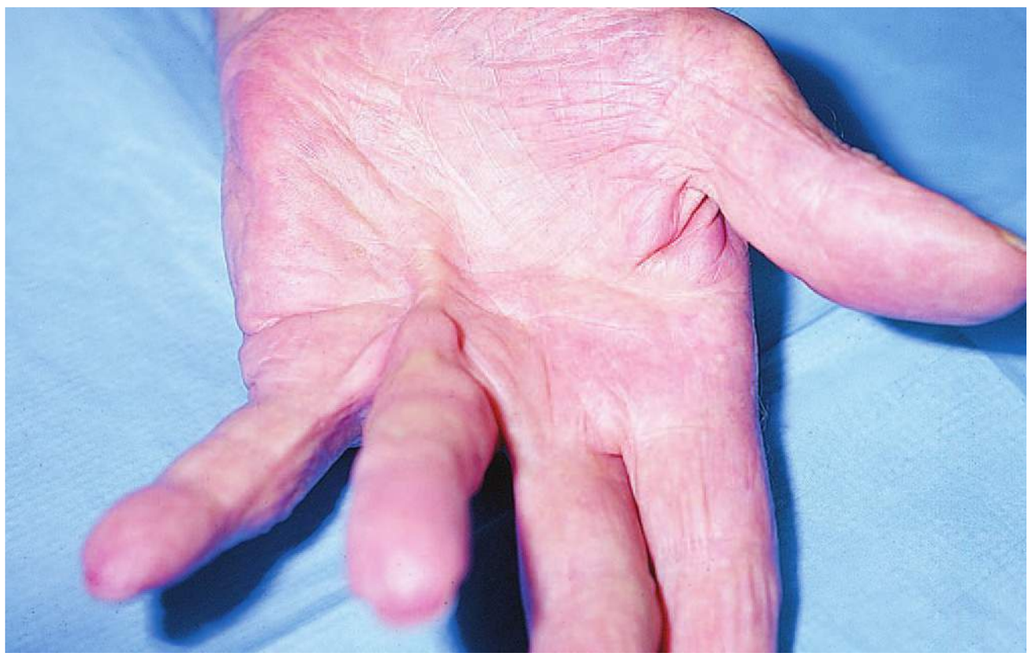

FIGURE 12.49 ■ **Dupuytren Contracture.** This chronic problem is seen at the most common site: the ring finger. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. The flexor tendons are not involved.
2. The ring and small fingers are the most commonly involved.
3. Alcoholic liver disease is associated with an increased risk of development.

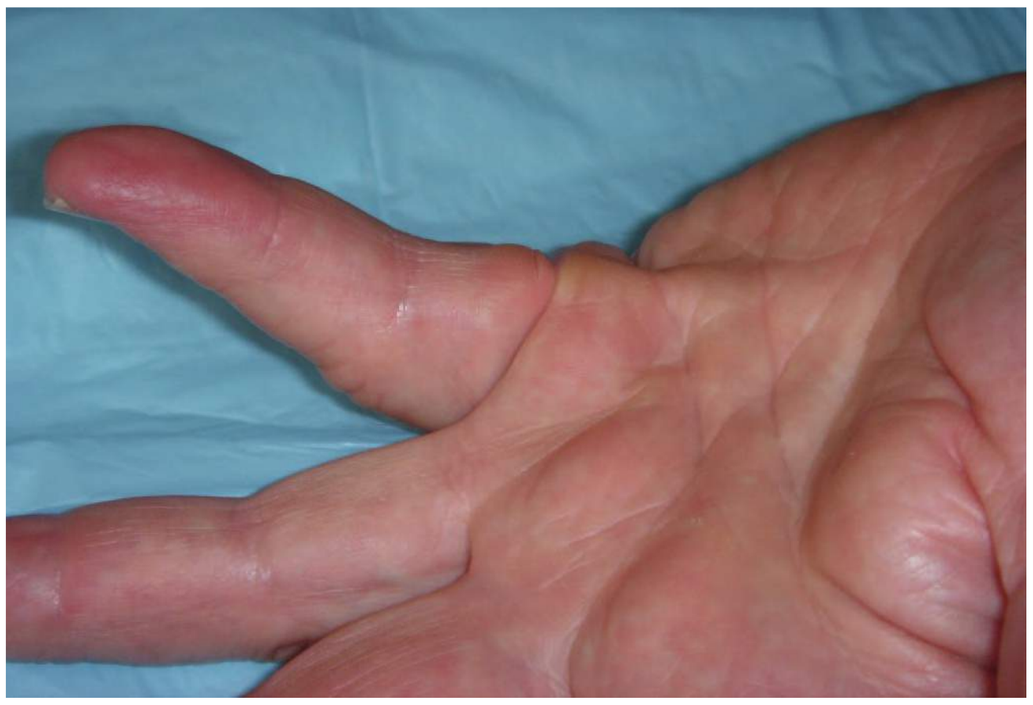

FIGURE 12.50 ■ **Dupuytren Contracture.** Note the contracture at the fifth metacarpophalangeal joint. (Photo contributor: Vineet Mehan, MD.)

ACHILLES TENDONITIS

Clinical Summary

Achilles tendonitis refers to a spectrum of disease ranging from nonpainful nodules to painful swelling of the tendon and paratendon sheath. It most typically develops from overuse, usually after sudden changes in activity or training level. It often occurs in older recreational athletes, who are generally more sedentary and deconditioned. Multiple factors generally contribute to the condition, including inappropriate footwear, training on poor surfaces, or prolonged running or jumping. High-risk factors include anatomic abnormalities, such as cavus feet, tibia vara, and heel or forefoot varus deformities. A tight Achilles tendon may develop in women who frequently wear high-heeled shoes or in patients who wear boots with high heels.

The most common area of pain is typically the area 2 to 6 cm proximal to the insertion site. This is due to a relative paucity of blood vessels in that region, making it more susceptible to inflammation and degeneration from repetitive microtrauma. Pain and tenderness increase with dorsiflexion of the foot. In some cases, a tendon friction rub may be palpable.

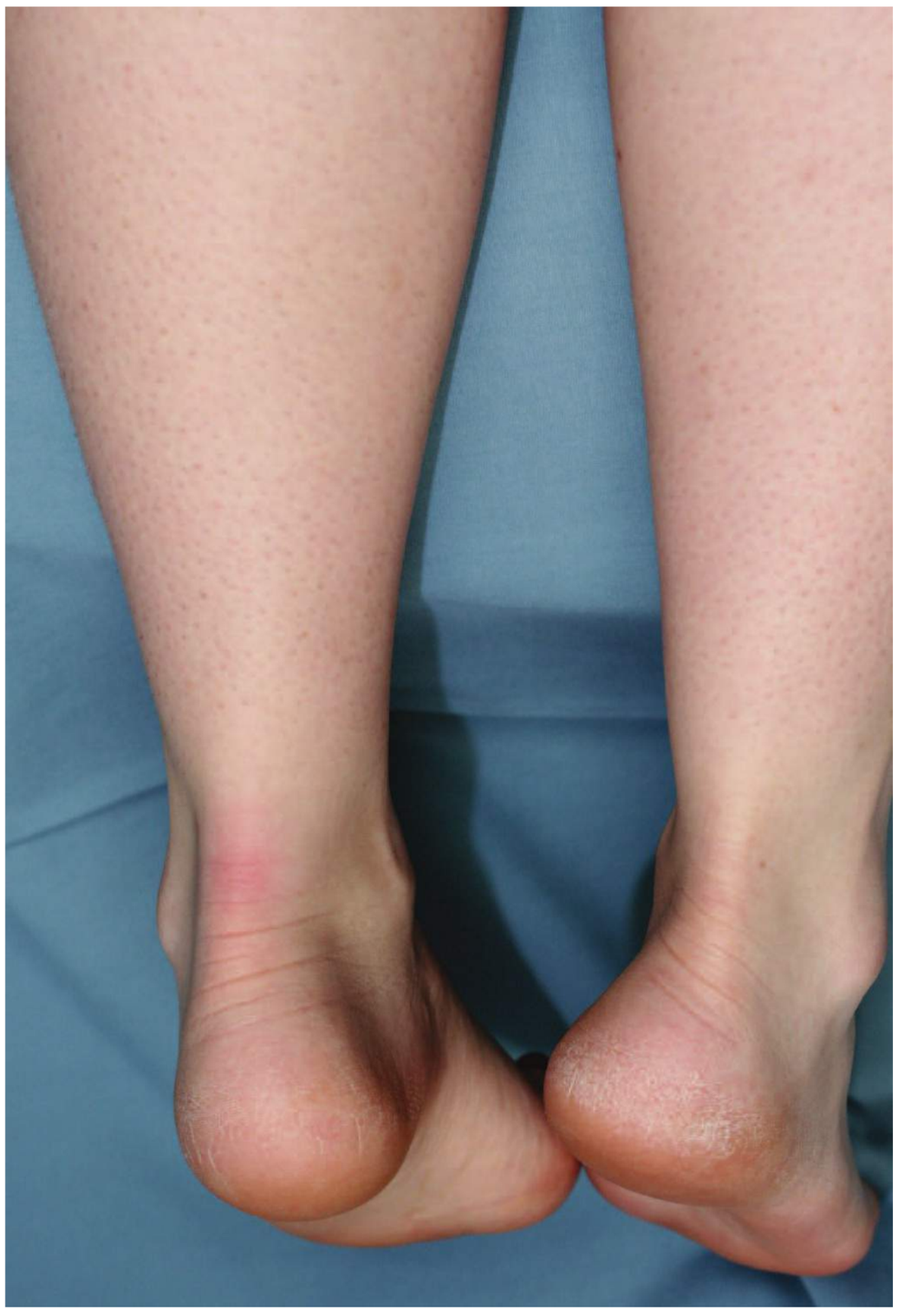

FIGURE 12.51 ■ **Achilles Tendonitis.** Note the significant erythema and thickening of the Achilles tendon on the patient's left compared to the unaffected right side. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Careful examination should be performed to distinguish between Achilles tendonitis and tendon rupture. The Thompson test and palpation of the tendon for gaps or discontinuity should be performed. Any patients with suspicion of partial or complete tendon rupture should be splinted and urgently referred to orthopedic surgery. In cases of tendonitis, gentle progressive stretching and lengthening exercises are helpful. In athletes, a reduction in activity is recommended. Use of gel heel inserts may be helpful in the short term, by cushioning and raising the heel, thereby decreasing tendon excursion. Use of ice and NSAIDs are useful in reducing pain and inflammation. Referral to an orthopedic surgeon for physical therapy is generally indicated in refractory cases.

Pearls

1. Fluoroquinolone use has been reported to increase the risk of Achilles tendonitis and possible tendon rupture. Patients presenting with pain, who are currently taking a fluoroquinolone, should have alternative antibiotic therapy considered.
2. Corticosteroid injection for Achilles tendonitis is very controversial, and should not be performed in the emergency department.
3. Bilateral tendon involvement, especially at the insertion site, suggests a systemic inflammatory condition such as ankylosing spondylitis, reactive arthritis (Reiter syndrome), or psoriatic arthritis.

GANGLION (SYNOVIAL) CYST

Clinical Summary

Ganglion (synovial) cysts are a cystic swelling overlying a joint or tendon sheath. They are the most common soft-tissue tumors of the hand and wrist, although they can arise over any joint. The etiology is currently debated; the most commonly accepted theory is the cyst forms secondary to mucoid degeneration of collagen and connective tissues. Other theories postulate a traumatic origin. It is unclear whether repetitive motion leads to causation, although it does appear to provoke symptoms and possibly lead to cyst enlargement. They may occur in any age, although the majority arise between the second and fourth decades of life.

Ganglion cysts are composed of collagen fiber walls with clear, highly viscous mucin content. They may be unilocular or multilocular. The most common presentations include swelling over or in close proximity to a joint, as well as pain, limitation of motion, weakness, and paresthesias. They are rarely greater than 2 cm in diameter. The most common location is dorsally over the scapholunate ligament of the wrist (60-70%), with the volar wrist the next most common site (20%).

Management and Disposition

Ganglion cysts may spontaneously regress; therefore, treatment is generally reserved for symptomatic lesions. Treatments include both nonsurgical and surgical options. The most common nonsurgical option is cyst aspiration, generally using a 16-gauge needle, followed by steroid injection. For recurrent lesions, referral to a hand surgeon for excision is warranted.

Pearls

1. Transillumination may aid in differentiating a ganglion cyst from a solid tumor; ganglia transilluminate, while solid lesions do not.
2. Patients with chronic dorsal wrist pain of unknown etiology should be screened for occult ganglion cysts.
3. MRI or ultrasound may be useful in detecting occult ganglion cysts.

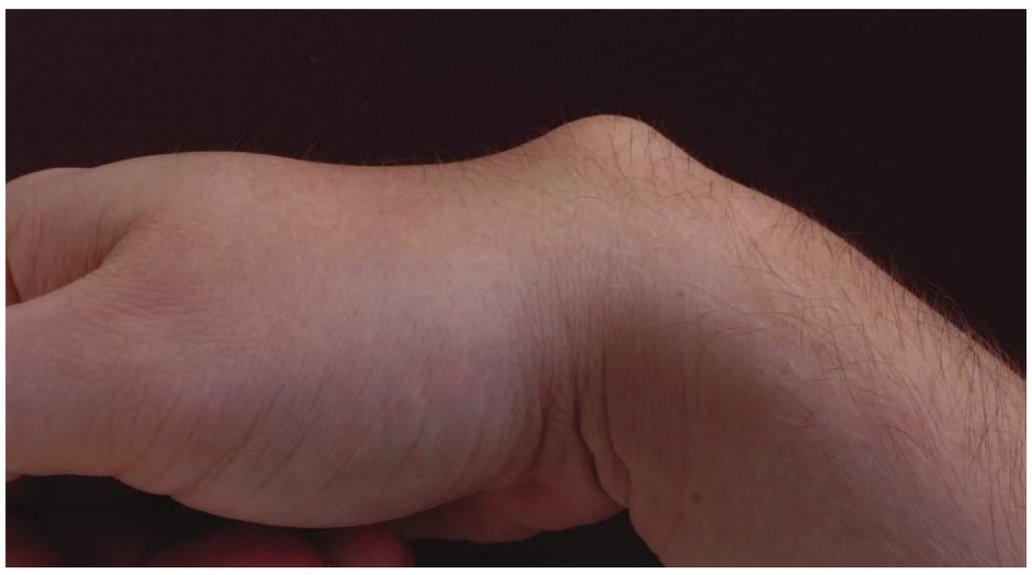

FIGURE 12.52 ■ **Ganglion Cyst.** This large ganglion cyst is located over the scapholunate ligament, the most common location for this entity. (Photo contributor: Lawrence B. Stack, MD.)

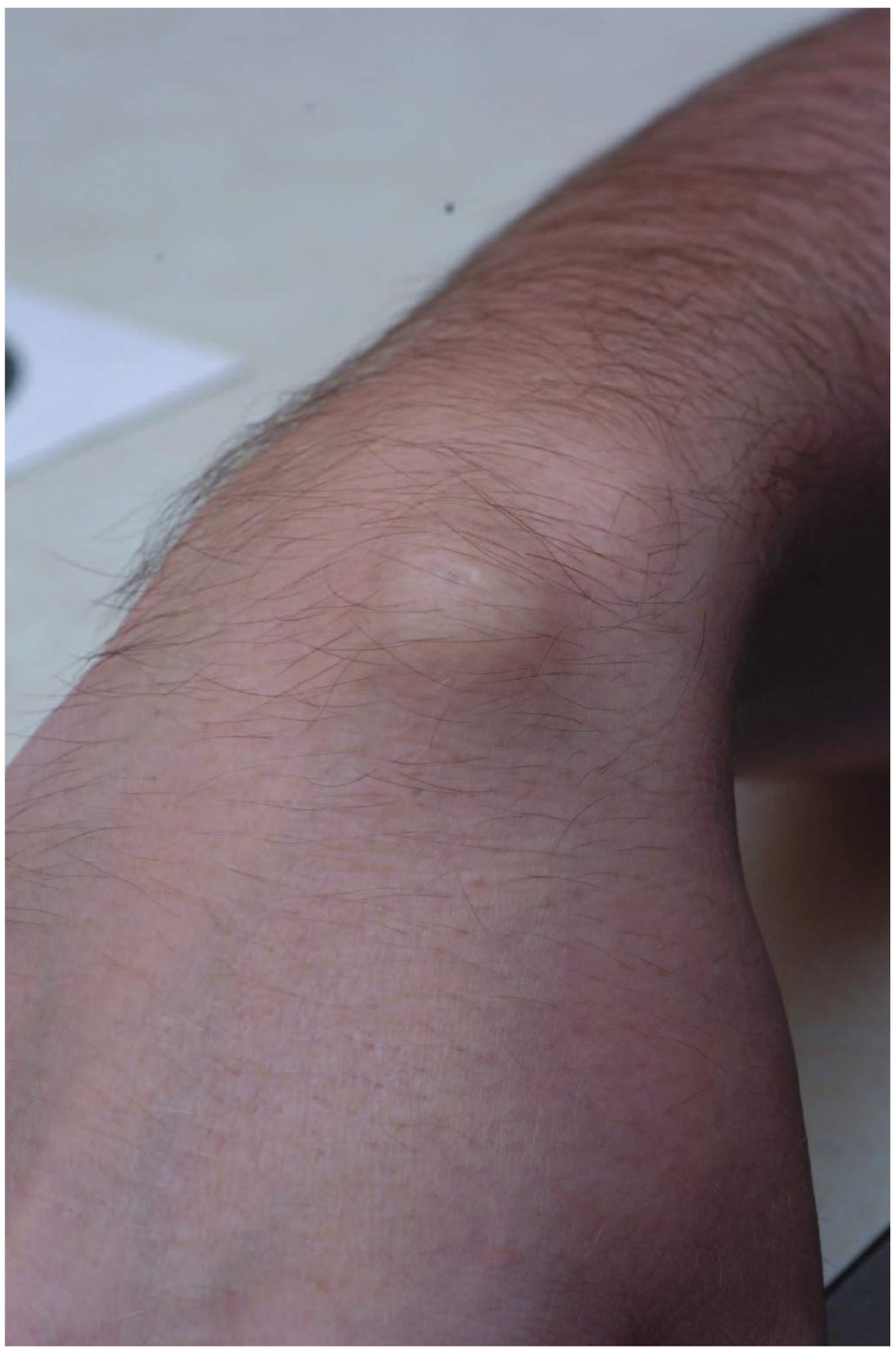

FIGURE 12.53 ■ **Ganglion Cyst.** Classic example of a ganglion cyst located over the scapholunate ligament of the wrist. (Photo contributor: Lawrence B. Stack, MD.)

RAYNAUD DISEASE

Clinical Summary

Raynaud disease refers to reversible ischemia of peripheral arterioles, usually in response to cold exposure or emotional stress. In general, it is more prevalent in women and most often affects the fingers, although the toes, face, ears, nose, and nipples may be involved.

A typical episode usually starts suddenly with the onset of cold digits associated with sharply demarcated, blue or white, color changes. With rewarming, erythema develops due to a reactive hyperemia. The vasospasm may last for several hours, but usually resolves with removal of the initial stimulus.

In general, it is called Raynaud disease, or primary Raynaud phenomenon, if symptoms occur without evidence of any other associated disease process. In contrast, secondary Raynaud phenomenon occurs in association with a related disease process, such as systemic lupus erythematosus or scleroderma.

Management and Disposition

Treatment involves removal of the inciting stimulus. As many cases are induced by going from a warm to a cold environment, active rewarming with warm water soaks or placing in the axilla are generally effective. Sympathetic stimulation may also trigger an episode, so calming patients who are anxious, or removing them from stressful situations, may be efficacious.

A thorough history and physical examination should be performed with careful attention paid to signs and symptoms of connective tissue disorders. Acrocyanosis (Crocq disease) is a circulatory disorder in which the hands, and less commonly the feet, are persistently cold and blue; some forms are related to Raynaud phenomenon. Counseling patients about methods for reducing the frequency and duration of attacks is helpful, including avoiding sudden cold exposure, minimizing stress, keeping the digits warm, avoiding cigarette smoking, and avoiding sympathomimetic drugs. Referral for primary care follow-up is recommended for all patients.

Pearls

1. A history of cool skin and sharply demarcated color changes is essential for diagnosis.
2. Routine ordering of blood tests, such as erythrocyte sedimentation rate (ESR) or antinuclear antibody (ANA), is not recommended unless other symptoms of connective tissue disorders are present. In these cases, close primary care or rheumatology follow-up is recommended.

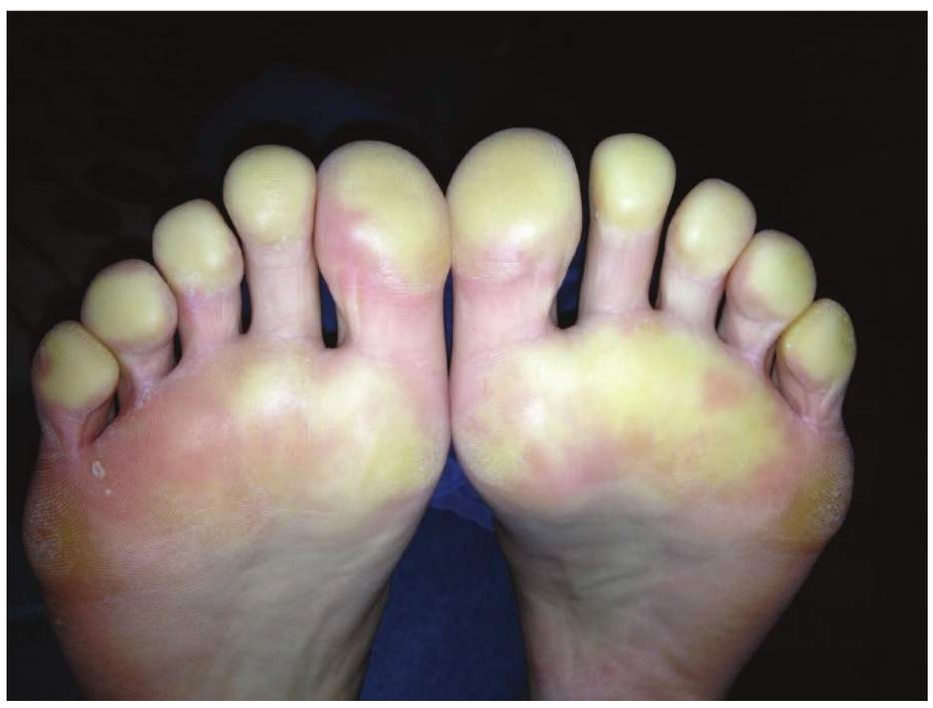

FIGURE 12.55 ■ **Raynaud Disease.** A patient with sharply demarcated color changes characteristic of an acute attack of Raynaud phenomenon. (Photo contributor: Katherine Farmer, MD.)

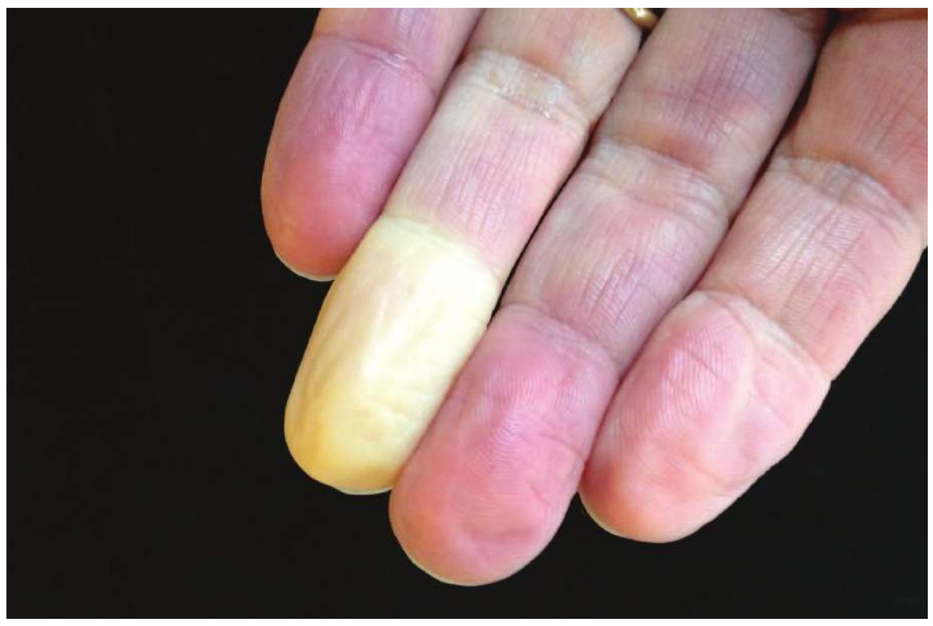

FIGURE 12.54 ■ **Raynaud Disease.** A patient with sharply demarcated color changes consistent with an acute attack of Raynaud phenomenon due to cold exposure. (Photo contributor: Kevin J. Knoop, MD, MS.)

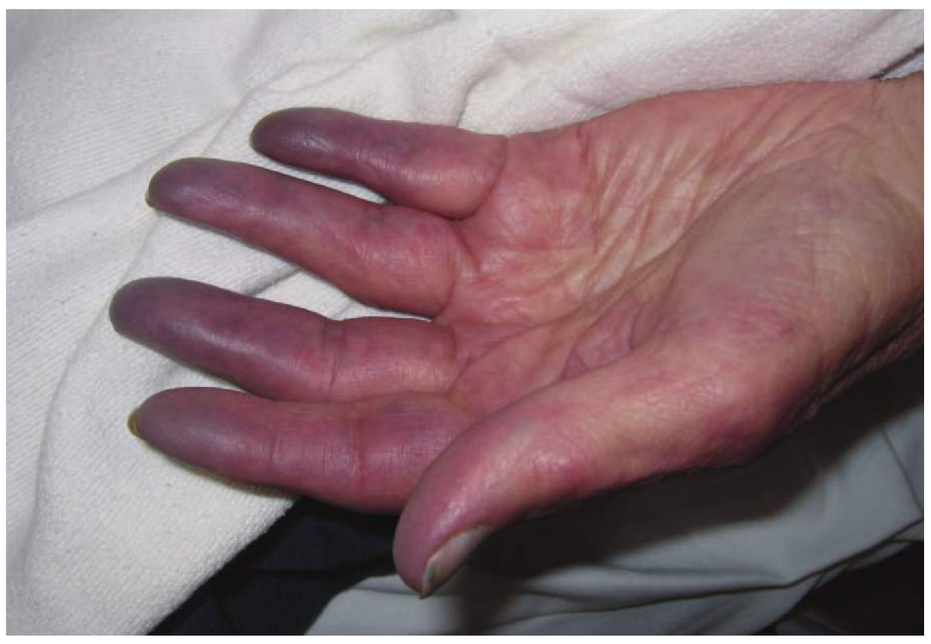

FIGURE 12.56 ■ **Acrocyanosis.** Persistently blue and cold fingers. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

An embolus refers to a foreign body in a blood vessel, carried by the blood to a site distant from its origin. Most emboli result from a detached piece of thrombus, often originating from left ventricular thrombus following myocardial infarction, or from atrial fibrillation. Other sources include atheroemboli from ruptured plaque, tumor, or foreign bodies such as venous or arterial catheters or guidewires.

Acute arterial embolization usually results in distal tissue infarction. Most embolic occlusion occurs at the branch points of arteries, due to the generally abrupt change in diameter at these bifurcation points. The most frequent site of arterial embolism is the bifurcation of the common femoral artery, accounting for 35% to 50%. Patients generally present with some or all of the "six Ps": pain, pallor, pulselessness, paresthesias, poikilothermia, and paralysis.

The general predictors of degree of ischemic insult include the amount of collateral circulation, as well as the size of the involved vessel and obstructing embolus. Generally, patients with longstanding peripheral vascular disease have a greater amount of collateral circulation, and are able to tolerate an acute occlusion better than a patient with normal arteries.

Management and Disposition

Acute arterial embolus is a surgical emergency. Immediate initiation of IV heparin is indicated to prevent further clot propagation. Prompt consultation with a vascular surgeon is imperative, as the rate of limb salvage drastically decreases after 4 to 6 hours.

In clear-cut cases of acute arterial embolism, the treatment is generally Fogarty catheter embolectomy without prior angiography. In these cases, a preoperative angiography only prolongs the limb's ischemic time and decreases the chance of salvage. Ambiguous cases, where it is difficult to distinguish between acute embolic occlusion and in situ thrombosis, may benefit from preoperative angiography. Emergent surgical intervention may aggravate thrombosis in the case of in situ thrombus formation.

Pearls

1. Acute arterial embolus generally presents with sudden onset of severe pain. In contrast, in situ thrombosis tends to have a more subacute presentation.
2. Aortic dissection may mimic acute arterial embolus. Involvement of multiple sites suggests dissection.
3. Intra-arterial thrombolytic therapy for acute arterial embolus remains controversial.

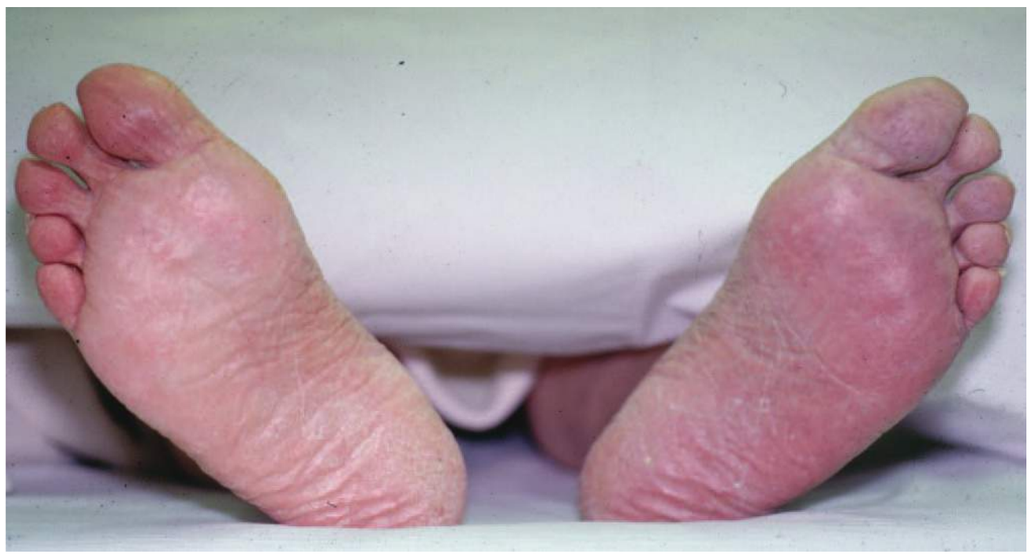

FIGURE 12.57 ■ **Arterial Embolus.** Note right foot pallor, consistent with an acute arterial embolus, in this case secondary to femoral artery occlusion. (Photo contributor: Lawrence B. Stack, MD.)

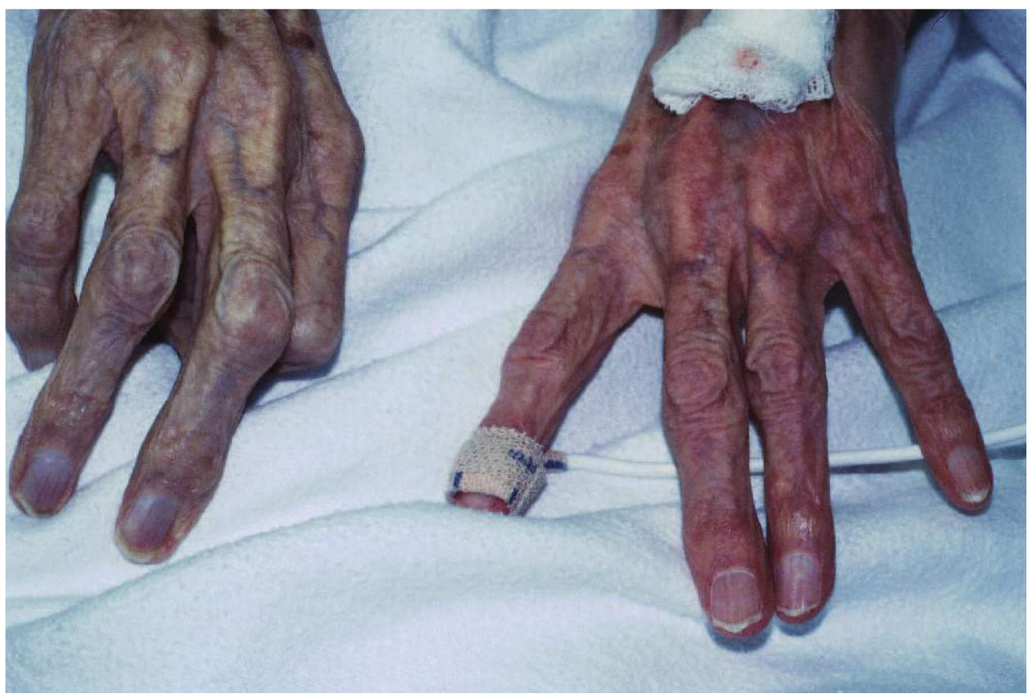

FIGURE 12.58 ■ **Arterial Embolus.** Pallor in the right hand secondary to arterial embolus, likely from atrial fibrillation. (Photo contributor: Lawrence B. Stack, MD.)