Soft-Tissue Infections and Their Imaging Mimics: From Cellulitis to Necrotizing Fasciitis[1]

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Abbreviations: AZT = azidothymidine, HIV = human immunodeficiency virus, NF = necrotizing fasciitis, STIR = short τ inversion-recovery

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Understand the spectrum of soft-tissue infections, organized by an anatomic approach.
- Describe the imaging appearances of soft-tissue infections, particularly at cross-sectional imaging.
- Discuss soft-tissue processes that mimic infections and their imaging features.

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Infection of the musculoskeletal system can be associated with high mortality and morbidity if not promptly and accurately diagnosed. These infections are generally diagnosed and managed clinically; however, clinical and laboratory findings sometimes lack sensitivity and specificity, and a definite diagnosis may not be possible. In uncertain situations, imaging is frequently performed to confirm the diagnosis, evaluate the extent of the disease, and aid in treatment planning. In particular, cross-sectional imaging, including computed tomography and magnetic resonance imaging, provides detailed anatomic information in the evaluation of soft tissues due to their inherent high spatial and contrast resolution. Imaging findings of soft-tissue infections can be nonspecific and can have different appearances depending on the depth and anatomic extent of tissue involvement. Although many imaging features of infectious disease can overlap with noninfectious processes, imaging can help establish the diagnosis when combined with the clinical history and laboratory findings. Radiologists should be familiar with the spectrum of imaging findings of soft-tissue infections to better aid the referring physician in managing these patients. The aim of this article is to review the spectrum of soft-tissue infections using a systematic anatomic compartment approach. We discuss the clinical features of soft-tissue infections, their imaging findings with emphasis on cross-sectional imaging, their potential mimics, and clinical management.

Introduction

Soft-tissue infections are common in clinical practice, encompassing a wide spectrum of pathologic conditions that range from involvement of the skin to the deep soft tissues and bone. Prompt diagnosis is key in the management algorithm to avoid further progression and complications. Although soft-tissue infection is mainly a clinical diagnosis, even an expert physical examination is not sufficient to define the extent and nature of the infection. In such circumstances, imaging becomes pivotal in differentiating various patterns of soft-tissue infections, depicting the extent of the disease, and identifying collections potentially amenable to intervention (1). In this article, we systemically review the spectrum of musculoskeletal soft-tissue infections organized by anatomic compartments from superficial to deep, excluding the bones, while providing relevant case examples and differential diagnoses.

TEACHING POINTS

- The microbiology of NF is either polymicrobial (type 1), with a mixture of aerobic and anaerobic organisms, or monobacterial (type 2), for which the main organisms are Gram-positive cocci. Type 1 infections are more common and usually involve the trunk and perineum, whereas type 2 infections are more frequently found in the extremities.
- The characteristics of involvement of the deep fasciae significantly associated with necrotizing infectious fasciitis include *(a)* extensive involvement of the deep intermuscular fascia, not just the area contiguous to the deep peripheral fascia, *(b)* thickening of fascia measuring 3 mm or more at STIR or fat-suppressed T2-weighted imaging, *(c)* involvement of three or more compartments, and *(d)* low signal intensity with fat-suppressed T2-weighted imaging in the deep fascia with corresponding nonenhancement on postcontrast images.
- Healthy muscles are generally resistant to infection, but if they are affected, a predisposing factor such as diabetes, drug use, malnutrition, human immunodeficiency virus (HIV) infection, immunodeficiency, malignancy, or trauma usually coexists.
- An intramuscular abscess is the hallmark of pyomyositis. In contrast, viral myositis does not progress to abscess formation.
- MR imaging findings in early subacute muscle denervation include diffuse T2 hyperintense signal and normal T1 intensity signal of the muscles and absence of subcutaneous edema.

Imaging Techniques

Radiography generally constitutes the initial examination for patients with soft-tissue infections. Findings pointing to possible inflammatory changes on radiographs include soft-tissue swelling, effacement of fat planes, and skin discontinuity in the setting of deep ulcers. However, these findings are nonspecific and can be seen in other settings, such as trauma, systemic causes of subcutaneous edema, venous insufficiency, and deep venous thrombosis. The presence of gas in the soft tissues or the identification of foreign bodies generally raises concern for underlying infectious processes. Moreover, radiography can help exclude other causes of soft-tissue swelling, such as underlying fractures (2). Ultrasonography (US) can aid physicians in excluding noninflammatory causes of soft-tissue swelling, such as deep venous thrombosis and soft-tissue tumors. It also provides localization of superficial nonradiopaque foreign bodies and real-time guidance for percutaneous interventions and tissue sampling (3). Computed tomography (CT) plays an important role in the assessment of soft-tissue infection in the emergency department due to its wide availability, scanning speed, high spatial resolution, and multiplanar reformatting capabilities (1). Overall, CT provides higher sensitivity for detection of soft-tissue gas and foreign bodies.

Magnetic resonance (MR) imaging has become the mainstay imaging tool for the diagnosis of soft-tissue infections owing to its inherent high spatial and contrast resolution, providing exquisite anatomic and pathophysiologic information about the extent and degree of involvement of both the soft tissues and the underlying bone (4). Optimum protocols include T1-weighted images (for anatomic details) and short τ inversion-recovery (STIR) sequences or T2-weighted images that are fat suppressed (for detection of soft-tissue edema). Intravenous contrast medium can be used to define extent of disease, outline abscess collections or sinus tracts, and detect nonenhancing devitalized soft tissue. Contrast medium administration should be avoided if the renal function is moderately (ie, glomerular filtration rate [GFR] of 30–44 mL/min) to severely (GFR <30 mL/min) impaired to avoid risk of developing nephrogenic systemic fibrosis (5). Complementary sequences such as diffusion-weighted imaging with apparent diffusion coefficient maps may be helpful by identifying abscesses (6).

Anatomic Considerations and Terminology

Anatomically, musculoskeletal infections can be classified into superficial and deep infections. Superficial infections are considered to include infections involving the skin and hypodermis, whereas deep infections involve the soft tissues at and below the level of the fascia (Fig 1). This classification is oversimplified and arbitrary, as infection spreads not only in horizontal planes but also vertically, invading the boundaries between the superficial and deep layers. This is important in larger areas of the body, such as the trunk and extremities, where the natural lack of fibrous boundaries between subcutaneous tissues and fascia enables widespread infection (7).

The skin is the most superficial layer of the soft tissues and consists of the epidermis and dermis. These two layers of skin cannot be resolved with imaging. Therefore, diagnosis of epidermal infections (such as impetigo or furunculosis) and dermal infections (such as erysipelas or folliculitis) is almost always clinical, with a limited role for imaging in the diagnosis and treatment of skin infections. Below the dermis is the subcutaneous tissue or hypodermis, which is a fatty layer of variable thickness. The fascia is the next anatomic layer, with the most confusing and ambiguous terminology in the literature and between surgeons and anatomists. Anatomists classify the fascia into superficial and deep layers. Some designate the whole hypodermis as superficial fascia, whereas others consider superficial fascia to be a distinct fibroareolar layer of connective and vascular tissue with various description in its location, including immediately deep to the dermis, deep to the hypodermis, or

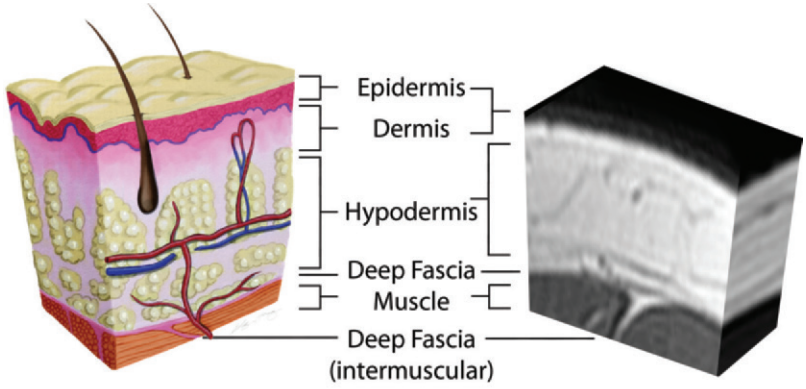


Figure 1. Schematic diagram shows the composition of soft-tissue layers on the left and corresponding layers at MR imaging on the right. Note that distinction between the epidermis and dermis is below the resolving power of current cross-sectional imaging using clinical sequences.

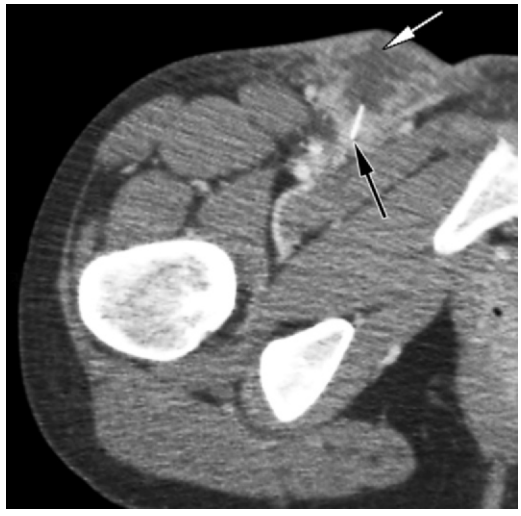
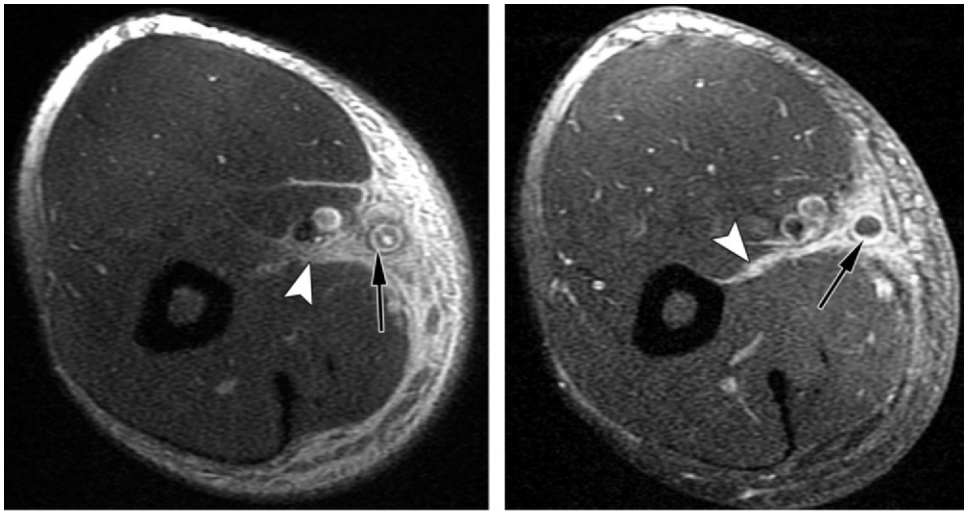


Figure 2. Subcutaneous abscess in a 25-year-old man with groin pain and fever after injection of drugs into his groin. Axial contrast-enhanced CT image of the right groin shows a fluid collection with a thick rim of enhancement (white arrow), consistent with an abscess. Subcutaneous and superficial soft-tissue swelling and enhancement surrounding the collection are consistent with cellulitis. Note the relatively intact subcutaneous fat in the lateral and posterior thigh for comparison. A broken needle fragment (black arrow) is seen deep within the collection.

in close proximity but separate from the deep fascia (also known as deep superficial fascia or membranous layer of the superficial fascia) (8,9). The deep fascia is a more tightly packed layer of collagen fibers that consists of an outer investing layer and a deep intermuscular component. The peripheral portion of the deep fascia is continuous with the epimysium, which is the connective tissue surrounding the muscles. The peripheral portion sends deep extensions to divide the muscles into compartments. These septa are known as intermuscular septa or intermuscular portions of the deep fascia (8). To most surgeons, however, the term *fascia* simply refers to the deep fascia. To avoid ambiguity, given that the delineation of the superficial fascia is below the resolving power of current imaging modalities and that infectious involvement of the hypodermis and superficial fascia often coexist and are managed similarly, we use the term *cellulitis* to describe infectious involvement of subcutaneous tissue and superficial fascia. *Deep peripheral fascia* and *deep intermuscular fascia* are terms reserved to describe the peripheral and deep layers of the deep fascia, respectively (8). Nevertheless, one should consider that the fascia is a continuum of tissue layers extending from the skin to the muscle and that infection can spread from the surface to muscle and bones and vice versa (10). The muscles are the next anatomic layer and are enveloped by a fibrous sheath and epimysium and divided by the deep fascia into different compartments. Finally, the bones are deep to and often surrounded by muscles.

Cellulitis

Cellulitis is a nonnecrotizing infection limited to the subcutaneous tissue, hypodermis, and superficial fascia without muscular or deep fascial involvement. *Staphylococcus aureus* and *Streptococcus pyogenes* are the most common offending agents gaining access to the skin through a penetrating skin defect (4). Less commonly, it can be secondary to hematogenous spread or extension from an underlying deep infection in immunocompromised patients. Common risk factors include vascular insufficiency, soft-tissue ulceration in the setting of diabetes or immunosuppression, and retained foreign bodies. Patients typically present with local erythema, swelling, warmth, and tenderness along with systemic manifestations including fever, malaise, and chills. Cellulitis is usually a clinical diagnosis; however, imaging can be performed in the setting of rapidly progressing disease or severe systemic manifestations to detect underlying deep tissue extension and possible localized collections (Fig 2).



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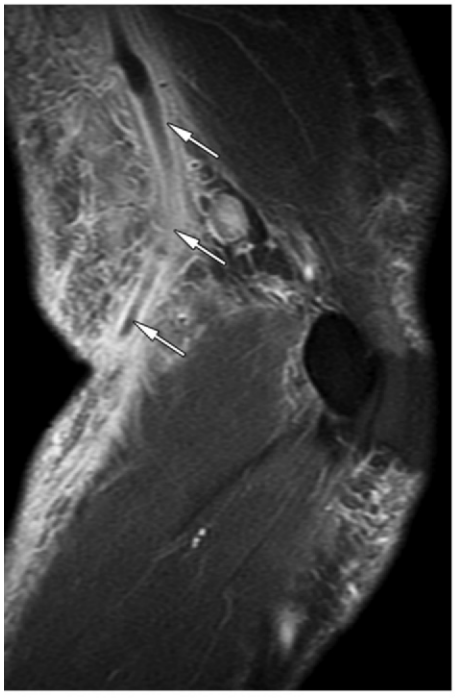


c.

Figure 3. Septic thrombophlebitis in a 22-year-old man with a history of intravenous drug use and elbow pain. **(a)** Axial fat-saturated proton density–weighted MR image shows subcutaneous soft-tissue edema at the anterior, medial, and posterior aspects of the distal arm. There is also edema centered about the basilic vein (arrow) with edema tracking along the deep fascia (arrowhead). **(b, c)** Axial **(b)** and sagittal oblique **(c)** T1-weighted fat-saturated postcontrast images show a long-segment filling defect in the basilic vein (arrows) with perivenous soft-tissue enhancement also extending along the deep fascia (arrowhead). Subsequent US (not shown) of the upper extremity demonstrated occlusive basilic vein thrombosis. These findings are consistent with septic thrombophlebitis (ie, superficial venous thrombosis and accompanying adjacent soft-tissue infection).

Radiographs demonstrate nonspecific diffuse soft-tissue swelling and may detect radiopaque foreign bodies. US gray-scale imaging findings are also nonspecific, demonstrating subcutaneous tissue edema with hypoechoic stranding insinuated between the echogenic fat lobules, giving a characteristic cobblestone pattern. Increased vascular flow at color or power Doppler US is more specific for the presence of an underlying inflammatory process (11). US is also the first line to differentiate cellulitis from venous thrombosis, although in some cases they may coexist (Fig 3). MR imaging of cellulitis demonstrates diffuse linear or ill-defined soft-tissue thickening with hyperintensity at T2-weighted imaging and/or STIR MR imaging and hypointensity at T1-weighted imaging and postcontrast enhancement (eg, Figs 3, 4). CT of cellulitis shows infiltration of the subcutaneous fat and can demonstrate underlying abscesses (Fig 2). Cellulitis is usually managed medically by antibiotics and controlling underlying metabolic abnormalities (5).

Infectious Tenosynovitis

Tenosynovitis refers to inflammation of the synovial membrane surrounding a tendon. It can result from infection, systemic inflammatory arthropathy, crystal deposition, or overuse. Infectious tenosynovitis most commonly results from direct inoculation from a puncture wound or contiguous spread from adjacent infections. Less commonly, it may result from hematogenous spread (12). Besides pyogenic causes of tenosynovitis, fungi, *Mycobacterium tuberculosis*, and nontuberculous mycobacterial (NTM) infections are among the less common pathogens, particularly affecting immunocompromised individuals (13). The most common site of involvement in the musculoskeletal system is the hand and wrist. Patients can present with tenderness, swelling, erythema, and painful range of motion of the involved tendon. NTM pathogens are insidious

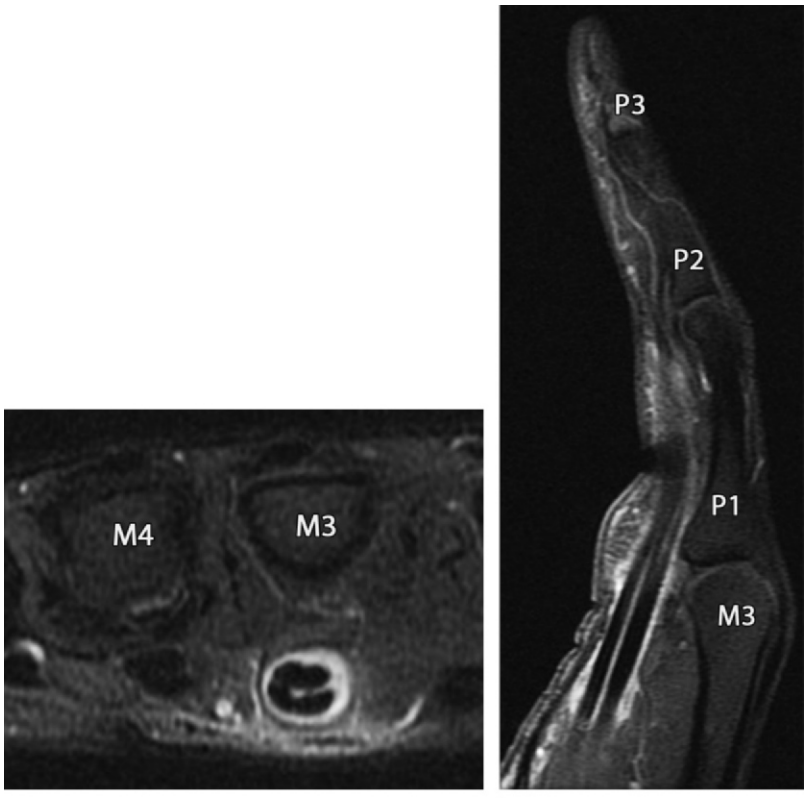


Figure 4. Atypical mycobacterial tenosynovitis and cellulitis in a 35-year-old man with swelling of the middle finger after scraping barnacles off the bottom of a boat several months prior. **(a)** Axial T2-weighted fat-suppressed MR image of the distal aspects of the middle *(M3)* and ring *(M4)* metacarpal bones shows fluid surrounding the third flexor tendon sheath with overlying subcutaneous soft-tissue swelling. The tendon itself is intact. **(b)** Sagittal T1-weighted fat-suppressed postcontrast MR image of the middle finger shows enhancement of the synovium of the flexor tendon from the level of the third distal metacarpal shaft *(M3)* to the midshaft of the proximal phalanx *(P1)*, without involvement of the middle *(P2)* or distal *(P3)* phalanges. These findings are consistent with tenosynovitis. Also note the mild overlying subcutaneous enhancement of the volar aspect of the middle finger, reflecting cellulitis. The patient underwent surgery with irrigation and débridement of the third flexor tendon. Tissue culture showed *Mycobacterium marinum*, the organism in an atypical infection seen in patients working around seashores.

and less symptomatic than their pyogenic counterparts and may manifest as soft-tissue swelling without disabling pain, signs of infection, or significant functional impairment (14).

MR imaging is the preferred modality for evaluation of tenosynovitis, although it is insensitive in differentiating various causes of tenosynovitis. The presence of gas or complex tenosynovial fluid favors an infectious cause, whereas multiple joint involvement (eg, radiocarpal, midcarpal, or carpometacarpal joints) with associated tenosynovitis suggests an inflammatory or crystal-induced arthropathy (15). MR imaging shows fluid distending the tendon sheath that is associated with thickening of the synovial sheath and intense enhancement on postcontrast images (12,16) (Fig 4). The fluid may be complex and can have varying signal intensity characteristics depending on the presence of pus, blood, or gas (Fig 5) (17). Tendons lose their normal low signal intensity and may appear thickened and show intermediate signal intensity with varying degrees of enhancement. There is usually edema in the surrounding soft tissues. Imaging findings of NTM overlap with those of pyogenic infections showing tenosynovitis with intact tendons and rarely involve underlying bones or muscles (Fig 4) (18). Rice bodies can be seen with tuberculosis, atypical mycobacterial infections, rheumatoid arthritis, and seronegative spondyloarthropathies and can be confused with synovial chondromatosis. US can also show hyperemia of a thickened synovium and tendon sheath as well as distention of the tendon sheath with accompanying fluid (Fig 6) (3). Radiography has a limited role in evaluation of infectious tenosynovitis and is most useful in detection of radiopaque foreign bodies and soft-tissue gas (19). As with MR imaging, CT shows fluid distention of the tendon sheath with variable enhancement of the synovium.

Infectious tenosynovitis is considered a surgical emergency, particularly in cases of acute bacterial flexor tenosynovitis of the hand. If left untreated, it may be complicated by osteomyelitis, tendon necrosis, or stenosing tenosynovitis (12). Aspiration of synovial fluid or a synovial biopsy is needed for definitive diagnosis.

Septic Bursitis

Bursitis usually refers to sterile inflammation of the bursal cavities. The pathologic processes leading to bursitis may include direct trauma, overuse, crystal-induced arthropathies such as gout, inflammatory arthropathies such as rheumatoid arthritis, or infection in case of septic bursitis (19,20). Septic bursitis is caused by bacterial inoculation of the synovial bursa; *S aureus* is the most common pathogen found (12). Superficial bursae such as the prepatellar or olecranon bursae are most commonly involved, often due to direct transcutaneous

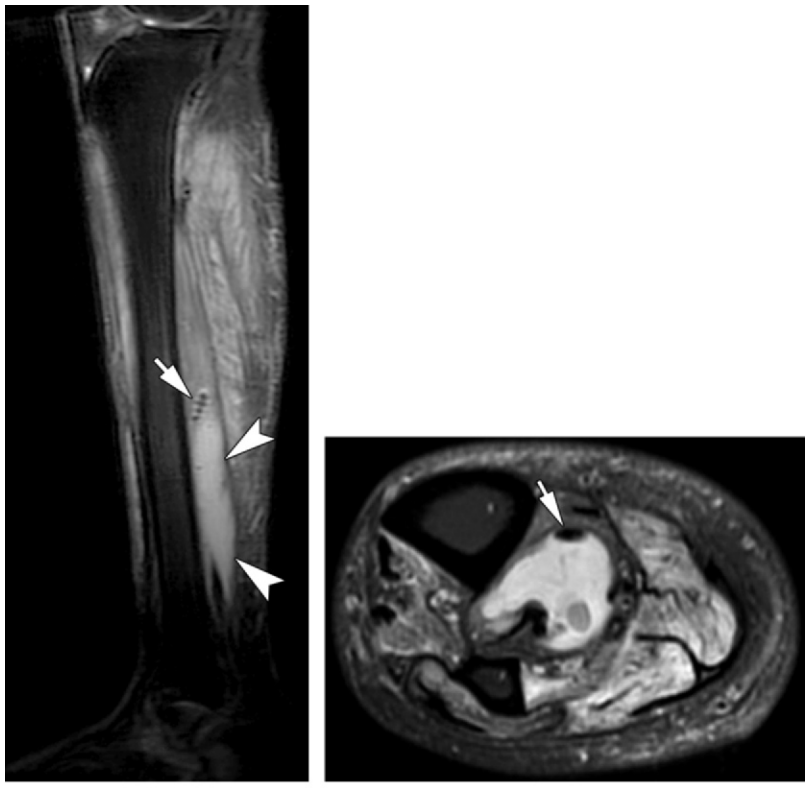


Figure 5. Tenosynovitis with intrasheath gas in a 63-year-old man with a diabetic ulcer at the plantar aspect of the foot and extensive septic arthritis and osteomyelitis of the ankle and foot. Sagittal **(a)** and axial **(b)** T2-weighted fat-suppressed MR images of the calf show a large tenosynovial fluid collection (arrowheads) containing debris and nondependent locules (arrow) of gas extending from the tibialis posterior myotendinous junction and dissecting through the muscle, consistent with infectious tenosynovitis with myositis. Although gas in the tendon sheath can be seen in the setting of gas-producing organisms, in this case it is probably the result of communication of the tendon sheath with the plantar sinus tract at the foot (not shown). Diffuse edema involving the calf muscles is also present, which may indicate concomitant pyomyositis, diabetic myonecrosis, muscle denervation changes from underlying diabetic polyneuropathy, or a combination of these.

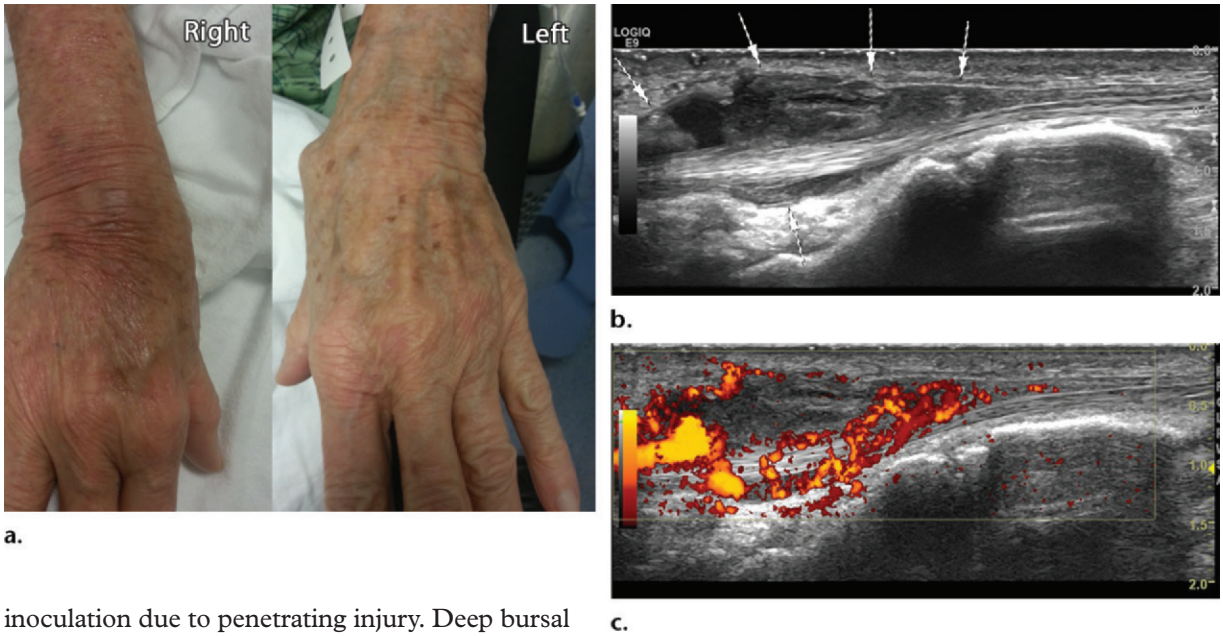
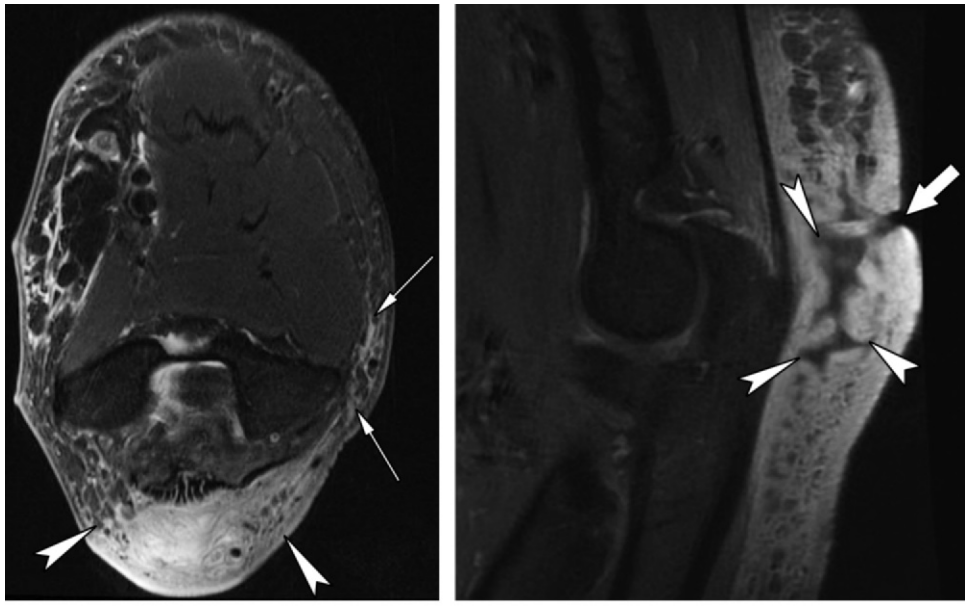


Figure 6. Septic tenosynovitis and cellulitis in a 76-year-old woman with dorsal right hand swelling following a cat bite. **(a)** Photograph of the right hand demonstrates swelling and erythema of the dorsum of the hand and wrist with obscuration of dorsal extensor tendon impressions in that region. Note the normal left hand for comparison. **(b, c)** Longitudinal gray-scale **(b)** and power Doppler **(c)** US images of the wrist at the fourth dorsal extensor compartment demonstrate a large amount of heterogeneous fluid and synovial proliferation (arrows) within the tendon sheath with corresponding hyperemia on the power Doppler image. These findings are consistent with septic tenosynovitis and cellulitis.

inoculation due to penetrating injury. Deep bursal infection is less common and typically occur hematogenously (21). Infection may also arise from the adjacent joint and may result in septic arthritis (5). Clinical manifestations include point tenderness over the inflamed bursa, soft-tissue swelling and/or erythema, fever, and local lymphadenopathy. Deep bursal infection is rarely associated with visible swelling or erythema.

Radiography typically shows nonspecific soft-tissue fullness if the superficial bursae are involved.



a. b.

Figure 7. Olecranon bursitis and cellulitis in a 52-year-old man with posterior right elbow pain and swelling with fluid draining from an open skin ulcer. **(a)** Axial fat-saturated T2-weighted MR image of the elbow shows a localized olecranon bursal fluid collection (arrowheads) with tracking subcutaneous soft-tissue edema (arrows) about the collection. **(b)** Sagittal T1-weighted fat-suppressed postcontrast MR image shows thick irregular peripheral rim enhancement (arrowheads) around the bursa, consistent with bursitis. There is a sinus tract (arrow) extending from the bursa to the skin surface, with fluid drainage seen clinically. The presence of drainage and communication to the skin surface suggests that this is an infected bursa. Enhancement and edema within the subcutaneous tissues about the elbow are consistent with regional cellulitis.

At US, the involved bursa shows wall thickening with hyperemia and is distended by mixed echogenic fluid with internal debris. Sometimes it demonstrates echogenic shadowing foci, indicating gas bubbles (5). As with US, CT can demonstrate a thickened bursal wall, bursal distention with infected fluid, and inflammatory edema within the adjacent soft tissues (22). At MR imaging, the involved bursa is distended by complex fluid that contains internal debris and septa and is surrounded by minimal peribursal edema. Gas bubbles in the bursa can be seen as punctate foci of signal voids. Postcontrast images show a thick enhancing rim surrounding the low-signal-intensity bursal fluid collection (Fig 7). If there is enhancement of the overlying skin, then accompanying cellulitis is invariably present (Fig 7). Unfortunately, there is a considerable overlap between the imaging findings of septic and nonseptic bursitis. Except for gas, which can be seen with gas-producing organisms, there is no other reliable sign to differentiate septic from nonseptic bursitis (5,12,23). As with tendon sheaths and joint spaces, bursal rice bodies can be seen with mycobacterial infections as well as other inflammatory and noninflammatory processes (5).

Necrotizing Fasciitis

Necrotizing fasciitis (NF) is a rapidly progressive infection of the deep soft tissue with a high mortality rate, reported in one study to be 29% even when treated (24). The disease can be classified on the basis of the affected anatomic part (eg, Fournier gangrene for the perineum or Ludwig angina for the submandibular region), microbial cause, or depth of infection. These different classifications have led to different terminologies such as *necrotizing adipositis*, *necrotizing fasciitis*, or even *myonecrosis* to describe infections that share a common pathophysiology and clinical management strategy (25). As such, the term *necrotizing soft-tissue infections* has been proposed to encompass all soft-tissue infections deep to the hypodermis (26). However, for the purpose of this article, we use the more-recognized term *necrotizing fasciitis* to describe infection involving the deep fascia and reserve the term *necrotizing soft-tissue infection* to describe a continuum of involvement of the remaining soft tissues, excluding the fascia, with emphysematous osteomyelitis on the extreme side of the spectrum. Nevertheless, radiologists should be familiar with the different terminologies when communicating with referring physicians, to avoid confusion.

There are no true risk factors for NF, but there are associations between its development and conditions such as injectable drug abuse, chronic debilitating comorbidities (eg, diabetes mellitus, immunosuppression, obesity), and peripheral

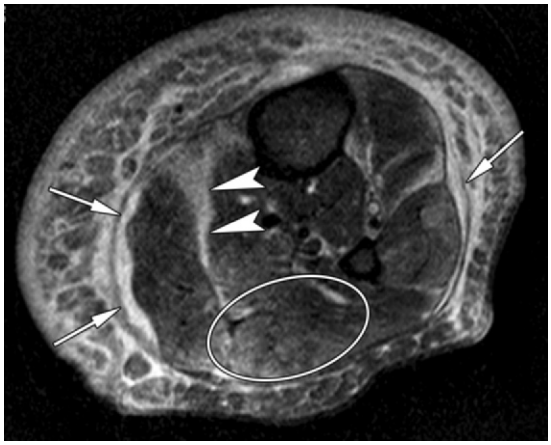


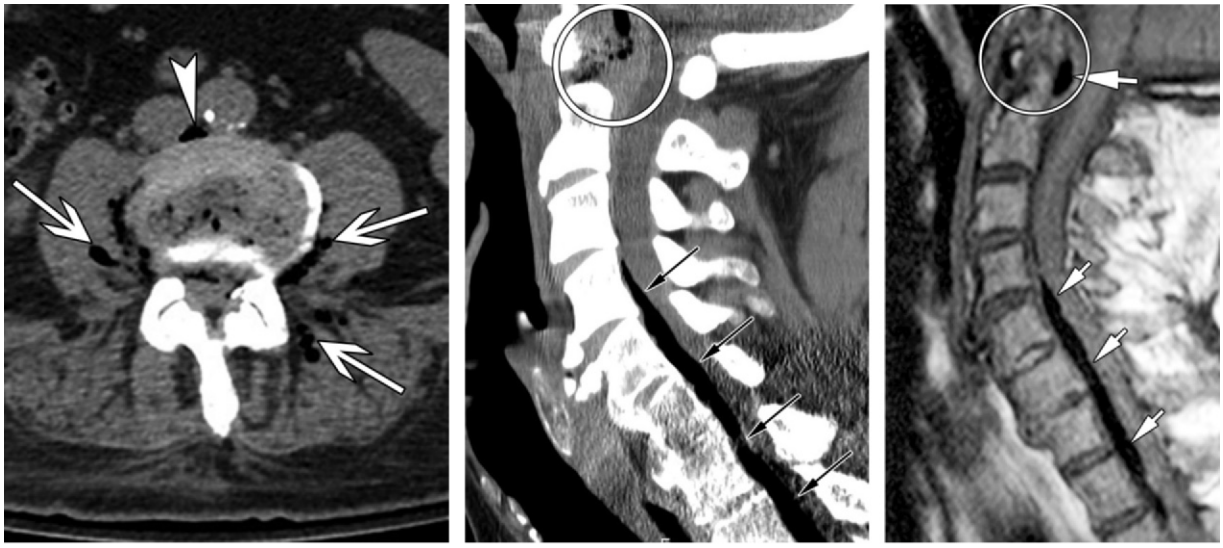
Figure 8. NF in a 48-year-old man with leg pain and fever. Axial STIR image of the midcalf shows fluid signal intensity tracking along the peripheral (arrows) and intermuscular (arrowheads) layers of the deep fascia with thickening of the fascia. Note that the thickened intermuscular fascia is continuous with the peripheral layer of the deep fascia, a finding suggestive of NF. The subcutaneous edema with skin thickening reflects cellulitis and superficial fasciitis. Patchy areas of muscle edema, predominantly involving the muscles of the posterior compartment (circle), are presumably reactive to inflammation of the adjacent fascia but may represent early pyomyositis or myonecrosis.

vascular disease (27–29). The most common sites of infections are the extremities, accounting for approximately half of the cases, followed by the perineum, trunk, and the head and neck (30). The microbiology of NF is either polymicrobial (type 1), with a mixture of aerobic and anaerobic organisms, or monobacterial (type 2), for which the main organisms are Gram-positive cocci (31,32). Type 1 infections are more common and usually involve the trunk and perineum, whereas type 2 infections are more frequently found in the extremities (31,32).

NF usually starts with inoculation of bacteria in the deep soft tissues following a skin surface breach due to penetrating injury, injection, or surgery (33). Bacteria rapidly multiply and the infection and tissue destruction rapidly progress, particularly in areas such as the limbs or trunk where there are no fibrous attachments between subcutaneous tissues and fascia to limit infectious extension (33). Gas-producing organisms (eg, *Clostridium* species) lead to subcutaneous gas formation, whereas toxin-producing organisms (eg, Gram-positive cocci) cause toxic shock–like symptoms.

Establishing the diagnosis is challenging and requires a high index of clinical suspicion. Patients may present with signs and symptoms of sepsis, including high fever, hypotension, and multiorgan failure; however, the skin manifestations may be minimal relative to systemic findings. Skin manifestations of NF, if present, include pain, edema, and erythema. Pain is generally out of proportion to the degree of skin involvement, which is a helpful clinical clue to differentiate NF from less serious conditions such as cellulitis or erysipelas (10). As the disease progresses, a number of skin changes, including discoloration, brown discharge, vesicles, bullae, necrosis, and crepitus, can subsequently develop (7). The main diagnostic dilemma is distinguishing superficial from deep soft-tissue involvement, the latter being more serious and necessitating immediate surgical débridement. In principle, differentiation between the two is clinical. In practice, however, this may be challenging and patients with NF may be treated for cellulitis and superficial infections before they rapidly deteriorate (34).

At MR imaging, the key finding is high T2 signal intensity along the deep fascia, especially the deep intermuscular fascia, such that absence of T2 signal intensity changes in the deep fascia essentially excludes NF (ie, high negative predictive value) (Fig 8) (8,35). The characteristics of involvement of the deep fasciae significantly associated with necrotizing infectious fasciitis include *(a)* extensive involvement of the deep intermuscular fascia, not just the area contiguous to the deep peripheral fascia, *(b)* thickening of fascia measuring 3 mm or more at STIR or fat-suppressed T2-weighted imaging, *(c)* involvement of three or more compartments, and *(d)* low signal intensity with fat-suppressed T2-weighted imaging in the deep fascia with corresponding nonenhancement on postcontrast images (35). In contradistinction, signal intensity changes that are limited to superficial fascia or peripheral portions of the deep fascia or involve only small portions of contiguous intermuscular septa are more likely to be due to superficial soft-tissue infection and cellulitis than NF. It should be noted that high signal intensity in the deep fasciae at T2-weighted imaging, although sensitive for NF, is not specific and can be seen in other entities such as nonnecrotizing inflammatory fasciitis, cellulitis, postradiation changes, ruptured popliteal cysts, inflammatory myositis, trauma, lymphedema, and vasculitis (36). Use of contrast material in routine assessment of NF is limited, as renal failure is common in these patients and its routine use may not be warranted (37). Fascial enhancement patterns following contrast material administration are variable, with enhancement, nonenhancement, and a



a. b. c.

Figure 9. Necrotizing soft-tissue infection and emphysematous osteomyelitis in a 57-year-old man with septicemia. CT of the abdomen was performed to search for the source of infection. **(a)** Axial nonenhanced CT scan of the abdomen shows perivertebral soft-tissue gas (arrows) in the adjacent psoas and erector spinae muscles and a focus of gas (arrowhead) in the retroperitoneum insinuating between the abdominal aorta and inferior vena cava. Also note the intramedullary gas within the vertebral body, as well as gas extending into the epidural space. **(b, c)** Sagittal CT **(b)** and T1-weighted MR **(c)** images of the cervical spine show that the source of infection is emphysematous osteomyelitis of the C1-C2 articulation with soft-tissue gas and swelling in and about the joint (circle). Extension of the epidural gas (arrows), characterized as low-signal-intensity foci, along the anterior cervical and thoracic spine is seen. These findings are consistent with necrotizing soft-tissue infection and emphysematous osteomyelitis.

mixed pattern being reported in the literature, with the latter being the most common (38,39). Enhancement of the involved fascia has been attributed to extravasation of contrast material due to increased capillary permeability. On the other hand, nonenhancement of the fascia is thought to be due to necrosis, thus distinguishing nonnecrotizing infectious fasciitis from NF (38,39). The presence of soft-tissue gas, although a rare finding, has high specificity for diagnosis of NF in the appropriate clinical setting (40). Soft-tissue gas, if present, is characterized by low-signal-intensity foci with all pulse sequences (Fig 9c) and blooming artifact with gradient-echo sequences (35). Muscle signal intensity changes, either peripheral band–like or diffuse hyperintense T2 signal with postcontrast enhancement, may be seen in NF and are thought to be reactive to adjacent fascial inflammation and necrosis (35). However, it is important to note that absence of soft-tissue gas does not exclude NF. The most common CT finding is focal subcutaneous fat infiltration and asymmetric fascial thickening with or without soft-tissue gas (Fig 9a, 9b) (41). On the other hand, in conditions that result in fluid retention, such as renal failure, low-protein states, or congestive heart failure, subcutaneous infiltration is symmetric and diffuse (41). Rarely, NF can spread deeper to involve bone, resulting in emphysematous osteomyelitis, characterized by intramedullary gas (Fig 9a) (42).

Infectious Myositis (Pyomyositis)

Skeletal muscle infection, or pyomyositis, is usually caused by hematogenous spread and transient bacteremia rather than direct extension from an adjacent soft-tissue infection (43,44). In the majority of cases of pyomyositis, the causative agent is a Gram-positive organism, most frequently *S aureus*. However, Gram-negative bacteria, anaerobes, mycobacteria, microsporidia, viruses, parasites, and fungi may be causative pathogens (43,44). Healthy muscles are generally resistant to infection, but if they are affected, a predisposing factor such as diabetes, drug use, malnutrition, human immunodeficiency virus (HIV) infection, immunodeficiency, malignancy, or trauma usually coexists (44). In the majority of patients, pyomyositis usually involves a single muscle, but multiple-site involvement is present in up to 40% of cases (45). The muscles of the lower extremity are involved more frequently, with the quadriceps muscles being the most common site, followed by the gluteal and iliopsoas muscles (45). Historically, an iliopsoas abscess implied an underlying tuberculous infection of the spine (Fig 10) (46,47). Today, the most common source for iliopsoas pyomyositis is gastrointestinal or urinary tract infections (46). Three stages of pyomyositis have been described (47). The first stage, the invasive stage, is characterized by muscle edema and pain due to bacterial seeding. The second stage of pyomyositis, the

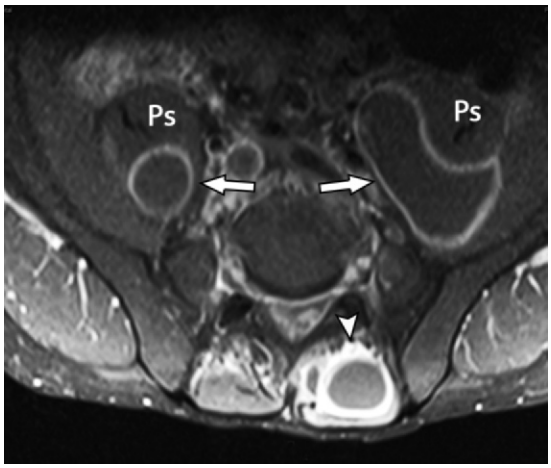
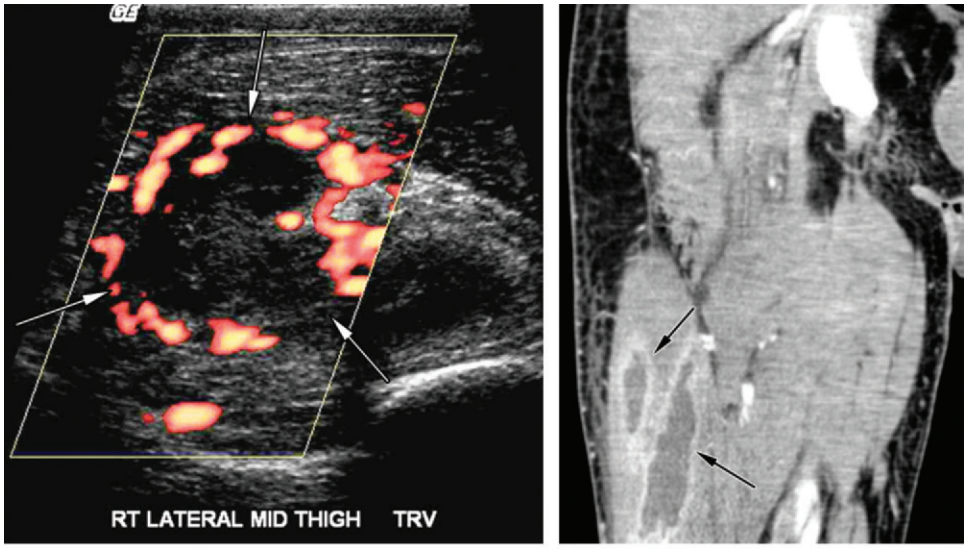


Figure 10. Psoas abscess from *M tuberculosis* infection in a 27-year-old man with back pain. Axial postcontrast T1-weighted fat-suppressed MR image of the pelvis shows rim-enhancing fluid collections (arrows) in both psoas *(Ps)* and left paraspinal (arrowhead) muscles, consistent with abscesses. The source of infection is due to contiguous spread of *M tuberculosis* diskitis and osteomyelitis in the lower lumbar spine (not shown).

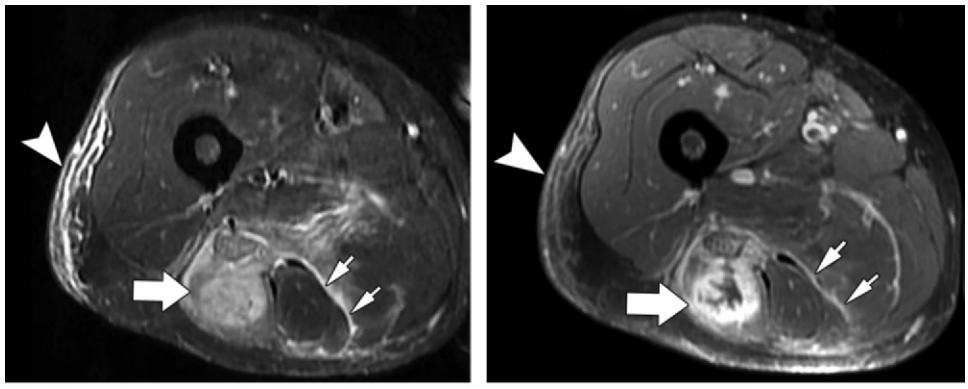


a. b.

Figure 11. Pyomyositis in a 25-year-old man with anterior thigh pain. **(a)** Transverse power Doppler US image of the thigh demonstrates peripheral flow around a heterogeneous hypoechoic collection in the vastus intermedius muscle (arrows). **(b)** Corresponding coronal postcontrast CT image demonstrates two rim-enhancing intramuscular fluid collections (arrows), consistent with pyomyositis.

suppurative phase, appears 10–21 days later; 90% of cases are diagnosed at this stage. This stage is characterized by intramuscular abscess formation and fever. The third stage, or late stage, is characterized by septicemia and multiorgan failure and carries a high mortality rate (47).

At CT, the affected muscles enlarge and demonstrate decreased attenuation, with effacement of the fat planes between the involved muscles and surrounding soft tissues. As the infection evolves, an intramuscular fluid collection develops with rim enhancement following contrast material administration (Fig 11). Intravenous contrast material may also be helpful in differentiating viable musculature from necrotic tissue, with the latter demonstrating lack of enhancement (1). At MR imaging, muscle edema can be the only abnormality in the early stages. However, muscle edema is a nonspecific finding and can be seen in a variety of conditions, which will be discussed later. In the second stage of pyomyositis, intramuscular abscesses can be identified (Fig 12). In fact, an intramuscular abscess is the hallmark of pyomyositis. In contrast, viral myositis does not progress to abscess formation (Fig 13) (48). Surrounding inflammatory changes are usually disproportionate and more extensive compared with the size of the abscess itself. This is in contrast to soft-tissue tumors, which usually produce less inflammatory change in the surrounding soft tissues. The signal intensity characteristics of an abscess vary and depend on its age and contents. It is usually low to intermediate at T1-weighted imaging and high at T2-weighted imaging, with a characteristic pattern of peripheral enhancement with lack of central enhancement on fat-suppressed



a. b.

Figure 12. Pyomyositis and cellulitis in a 28-year-old man with fever and thigh pain. **(a)** Axial T2-weighted fat-suppressed image of the proximal thigh demonstrates diffuse edema (large arrow) involving the biceps femoris and to a lesser extent the adductor magnus muscle, with fluid tracking (small arrows) along the deep fascia between the adductor and hamstring muscles. Note the subcutaneous edema (arrowhead) of the lateral thigh. **(b)** Axial postcontrast T1-weighted fat-suppressed MR image demonstrates intense enhancement (large arrow) of the biceps femoris muscle with a central area of nonenhancement, consistent with pyomyositis. Enhancement (small arrows) of the nonthickened fascia between the adductor magnus and hamstring muscles and subtle enhancement (arrowhead) of the subcutaneous tissue laterally are consistent with cellulitis.

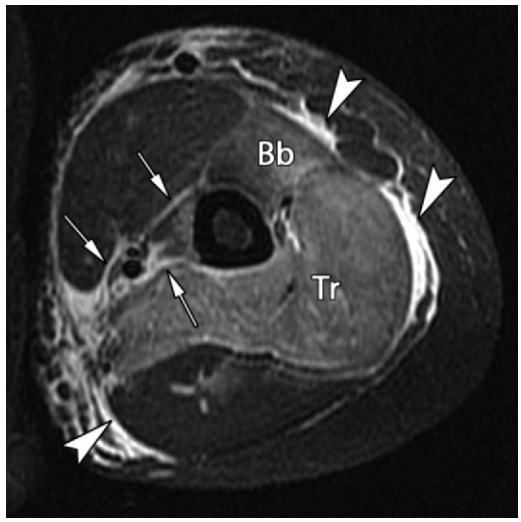


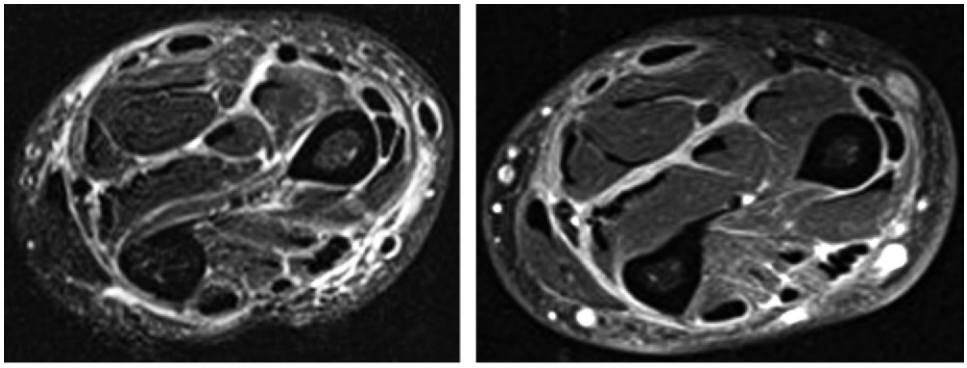
Figure 13. Viral myositis and rhabdomyolysis in a 27-year-old woman with myalgia and elevated muscle enzymes following an upper respiratory tract infection. Axial STIR image of the arm shows diffuse muscle edema of the arm involving portions of the triceps *(Tr)* and lateral portions of the biceps *(Bb)* muscles. There is near-complete circumferential edema along the nonthickened superficial layer of the deep fascia (arrowheads) and intermuscular layer of the deep fascia (arrows), indicating fascial involvement. Along with the history, the findings are consistent with myositis and rhabdomyolysis.

T1-weighted images following contrast material administration. Differential diagnosis at this stage includes other lesions resembling soft-tissue masses, such as neoplasms, intramuscular hematomas, myonecrosis, myositis ossificans, sarcoidosis, and parasitic infections (48). Attention to signal intensity characteristics and findings from other imaging modalities may reveal clues to the final diagnosis. For example, fluid-fluid levels can be seen with necrosis, blood, or purulent material. High signal intensity at T1-weighted imaging suggests blood breakdown products and/or fat. If the central portion of the mass enhances, myonecrosis and abscess would be unlikely, and other possibilities, such as neoplasm, granulation tissue, and myositis ossificans, should be entertained. Intramuscular hematoma and myositis ossificans may show peripheral calcifications on radiographs (48,49). Additionally, clinical history and laboratory findings can be helpful in distinguishing abscess from other masslike lesions. For example, an immunocompromised patient with fever and a tender mass in the thigh associated with leukocytosis is more likely to have a soft-tissue abscess, with other diagnoses less likely. Nevertheless, differentiating abscess from other masslike lesions may be challenging, warranting further clinical and imaging follow-up and even biopsy.

Soft-Tissue Infection Mimics

Noninflammatory Causes of Subcutaneous Edema

Noninflammatory causes of soft-tissue edema, such as congestive heart failure, diabetic vascular insufficiency, lymphatic obstruction, and acute and subacute venous thrombosis, may manifest as soft-tissue swelling and pain, mimicking cellulitis. These entities are often diagnosed clinically, and imaging is used to provide support for



a. b.

Figure 14. Eosinophilic fasciitis in a 52-year-old woman with upper extremity pain. **(a)** Axial T2-weighted fat-suppressed MR image of the forearm shows thickening and hyperintense signal along the deep fascia of the forearm. **(b)** Axial postcontrast T1-weighted fat-suppressed image shows enhancement of the fascia. Note that the muscles themselves are relatively uninvolved. Peripheral eosinophilia, hypergammaglobunimia, increased ESR, and scleroderma-like skin changes in this patient favor a diagnosis of eosinophilic fasciitis. The patient rapidly responded to steroid therapy.

the diagnosis (eg, US in diagnosing deep venous thrombosis) or to exclude underlying infection. At imaging, soft-tissue edema in the setting of noninflammatory causes is usually more extensive and diffuse. Unlike cellulitis, hyperemia is not a feature of these entities; therefore, if intravenous contrast material is used, they do not show enhancement (50).

Noninfectious Fasciitis

This category encompasses a group of diseases characterized by soft-tissue inflammation and fascial thickening or enhancement, without evidence of necrosis at clinical evaluation or imaging studies. There are three separate diseases in this category: paraneoplastic fasciitis, eosinophilic fasciitis, and nodular and proliferative fasciitis (51). Definite diagnosis requires clinicopathologic correlation. Treatment usually requires high-dose steroids or immunosuppressive medications, and in cases of nodular fasciitis, surgical resection (51,52).

Paraneoplastic Fasciitis.—Paraneoplastic fasciitis falls under the spectrum of Sweet syndrome, also known as acute febrile neutrophilic dermatosis. Myofascial involvement in the form of myositis and fasciitis is a rare manifestation of the disease (53). As its name implies, it may occur concurrently with a malignant neoplasm, precede malignancy, or be a sign of tumor recurrence (54). Patients present with painful red or purple-red papules or nodules in the skin, an elevated erythrocyte sedimentation rate (ESR), and neutrophil-predominant peripheral leukocytosis (54). Imaging findings are nonspecific and include dermal thickening, soft-tissue edema, and often multiple sites of involvement. At MR imaging, fascial thickening and concomitant muscle edema, although rare, may be present (54).

Eosinophilic Fasciitis.—Eosinophilic fasciitis is a rare disease, manifested by scleroderma-like skin lesions, usually in the trunk and extremities. Patients have hypergammaglobulinemia, elevated ESR, and peripheral eosinophilia. Imaging findings resemble those of paraneoplastic fasciitis. At MR imaging, there is fascial thickening at T1-weighted imaging with corresponding hyperintense signal with fluid-sensitive sequences and a variable degree of enhancement on postcontrast images (Fig 14) (55).

Nodular and Proliferative Fasciitis.—Nodular fasciitis, also known as infiltrative fasciitis, pseudosarcomatous fasciitis, and pseudosarcomatous fibromatosis, is a disease of unknown cause, typically affecting patients younger than 50 years of age (52). Patients present with a rapidly growing nodule or nodules usually less than 2 cm in size in the extremities and trunk over a period of 1–2 weeks (52). The lesions can be found in subcutaneous, intrafascial, intramuscular, or rarely in intradermal and intravascular locations and are painful in approximately one-half of cases (56). It is thought to be the result of a benign reactive fibrous proliferation in response to a local stressor. Its imaging and histopathologic characterization can sometimes overlap with that of soft-tissue sarcomas and may warrant surveillance imaging to document posttreatment stability or resolution. Depending on the fibrous, cellular, or myxoid content of the lesions, different imaging characteristics can be seen at MR imaging. In predominantly

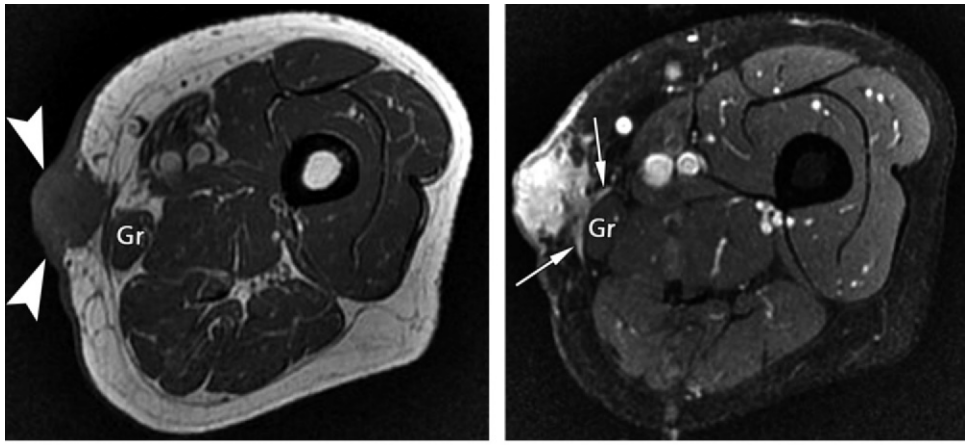


Figure 15. Nodular fasciitis in a 78-year-old woman with a rapidly growing mass in the medial thigh and a history of nodular fasciitis resected 5 years previously in the same area. **(a)** Axial T1-weighted MR image of the upper thigh shows an ovoid mass (arrowheads), isointense to muscle, located in the subcutaneous tissues immediately medial and superficial to the gracilis muscle *(Gr)*. Also note the thickening of the overlying skin. **(b)** Axial postcontrast T1-weighted fat-suppressed MR image shows avid enhancement of the mass and overlying skin as well as a curvilinear enhancing tail (arrows) extending over the deep fascia and epimysium of the gracilis muscle *(Gr)*. Biopsy of the mass demonstrated nodular fasciitis.

fibrous lesions, the signal intensity is low with all pulse sequences, whereas hypercellular lesions are isointense to muscle at T1-weighted imaging and hyperintense to muscle at T2-weighted imaging. Postcontrast images can show variable enhancement, but homogeneous enhancement is the most common pattern (Fig 15) (57).

Proliferative fasciitis is similar to nodular fasciitis in many respects; it also usually involves subcutaneous tissue or superficial fascia, grows rapidly, and is usually small at presentation. MR imaging of proliferative fasciitis has not been well-described in the literature (58).

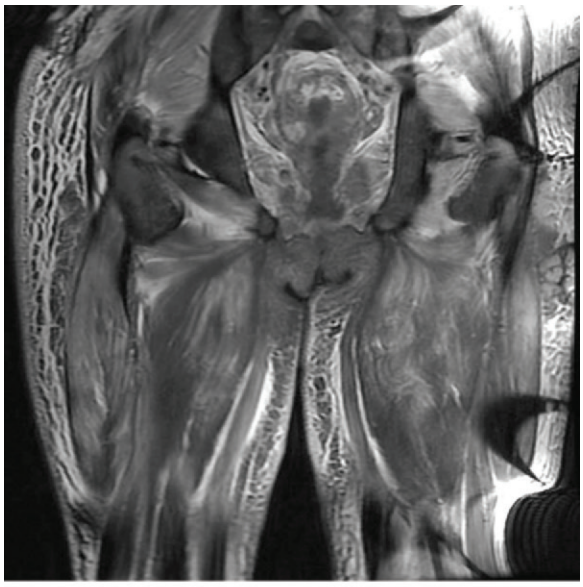
Inflammatory Myopathy

Inflammatory myopathies are a group of autoimmune disorders involving the skeletal muscles and often the adjacent fascia with elevated levels of muscle-derived proteins such as creatine kinase. The criteria and classification of autoimmune inflammatory myopathies are still evolving but historically include three separate diseases: dermatomyositis (DM), polymyositis (PM), and inclusion body myositis (IBM). More recently, a new class of autoimmune inflammatory myopathies was introduced, necrotizing autoimmune myopathy (59).

In DM, patients present with bilateral, symmetric muscle weakness, typically involving the musculature of the thighs and pelvis, progressing to involve the upper extremities. The affected muscles are not limited to specific compartmental or neural anatomy. Typical skin manifestations in DM are Gottron papules, which are scaly nontender lesions over the metacarpophalangeal and proximal interphalangeal joints and knees; heliotrope rash over the eyelids; a rash over sun-exposed areas in the upper chest, back, and neck in a "shawl" pattern; poikiloderma; and calcinosis cutis. The age of onset in DM is early adulthood, with a female predominance. The diagnosis is often established by laboratory parameters (eg, elevated muscle enzymes, antinuclear antibodies, rheumatoid factor, anti–tRNA synthetase), clinical findings, muscle biopsy, and electromyography (60,61).

At MR imaging, subcutaneous edema is prominent with fluid-sensitive sequences and may precede development of skin rash. Typical MR imaging findings include bilateral symmetric muscular and perimuscular edema of the proximal thigh and pelvic muscles with varying degrees of postcontrast enhancement (Fig 16). Relative sparing of the biceps femoris and rectus femoris muscles has been reported in DM (62).

PM also has a female predominance but usually affects older age groups than does DM, with the peak incidence between 40 and 60 years old. Its clinical manifestations overlap with DM but patients lack the skin findings. Muscle involvement is similar to what happens in DM, with symmetrical involvement of proximal musculature. Some muscles may be spared, and MR imaging can help target the best area for biopsy. Whole-body MR imaging is currently used for diagnosis and posttreatment follow-up (63). In chronic forms of DM and PM, progressive fatty replacement of muscle with atrophy occurs over months to years. Subcutaneous and intramuscular calcifications that are punctate and sheetlike,



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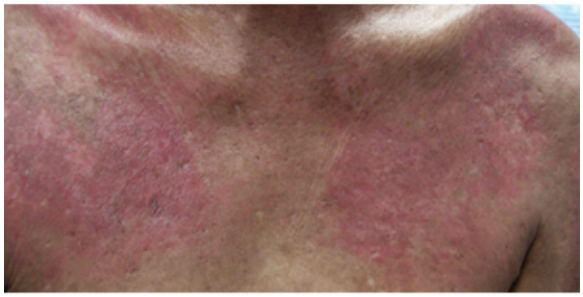
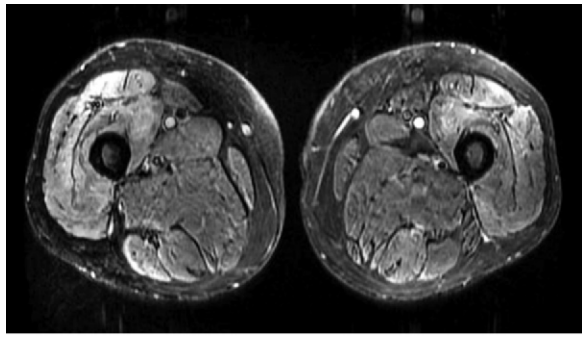


b.

Figure 16. DM in a 30-year-old man with muscle weakness. **(a)** Coronal STIR image shows diffuse symmetric muscle enlargement, edema, and inflammation of the thigh and pelvic musculature. There was heterogeneous enhancement of the involved muscles following intravenous contrast material administration (not shown). Note also the skin and subcutaneous signal intensity changes. **(b)** Photograph of the patient's chest shows associated skin changes from DM.

especially in DM, can also be seen and are best appreciated on radiographs. Treatment consists of high-dose steroids and immunosuppressants.

IBM is a form of focal myositis affecting patients older than 50 years. Inflammation, however, is not a prominent feature of the disease. IBM has a male predominance, unlike DM and PM. Skin changes are not a feature of the disease, and certain muscles including the finger flexors, quadriceps, and ankle dorsiflexors are more commonly affected, with relative sparing of the pelvic girdle muscles (64). IBM may progress to involve the pharyngeal muscles and cause dysphagia (64). MR imaging findings are not specific and can be limited to edema of the muscles in the acute phase and muscular atrophy in the chronic phase of the disease (Figs 17, 18). The disease is usually not responsive to corticosteroids and immunosuppressive drugs. Intravenous immunoglobulin shows short-lasting benefits in some cases (59).



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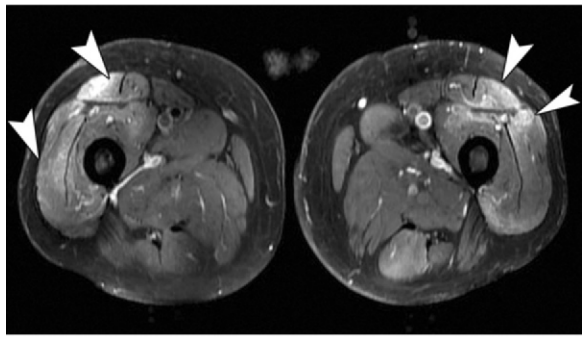


b.

Figure 17. IBM in a 73-year-old man with weakness of the quadriceps muscles. **(a)** Axial STIR image of the upper thighs demonstrates symmetric edema predominantly involving the quadriceps muscles with relative sparing of the hamstring and the adductor compartment, preservation of muscle architecture, and absence of adjacent subcutaneous and fascial fluid. **(b)** Axial postcontrast T1-weighted fat-suppressed MR image shows mild enhancement (arrowheads) of the muscles, most prominent in the vastus lateralis and rectus femoris muscles. These findings are most suggestive of IBM, particularly given the age of onset. PM and DM typically are associated with subcutaneous edema and occur in a younger age group. Drug-induced myositis may also be considered, but sparing of the buttock musculature (not shown) and relative sparing of the hamstring muscles make this diagnosis less likely. Ultimately, muscle biopsy is necessary for a specific diagnosis, which helps confirm the diagnosis of IBM. Biopsy can be targeted to the area of active muscle inflammation depicted at MR imaging.

Collagen Vascular Disease

Collagen vascular diseases, also known as connective tissue disorders, are a heterogeneous group of multisystem often autoimmune disorders with a predilection for connective tissue involvement. A few examples of the disorders in this group include systemic lupus erythematosus (SLE), mixed connective tissue disorder, scleroderma, polyarteritis nodosa, Sjögren syndrome, and rheumatoid arthritis (RA). Differentiation of these diseases is usually not difficult, as clinical and laboratory findings specific to each disease are almost always present.

True inflammatory myositis is a rare but well-recognized complication of connective tissue disorders, observed in less than 5% of patients (65,66). Myositis is much more common in mixed connective tissue disorder (ie, overlap

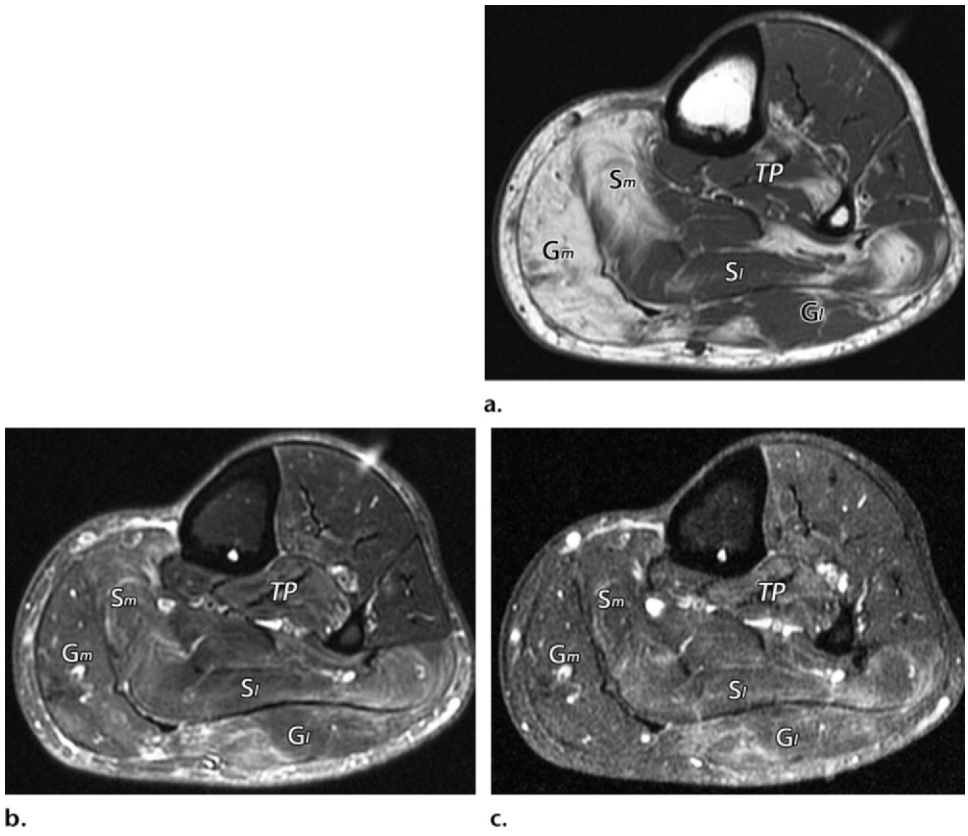


Figure 18. Chronic IBM in a 60-year-old man with a 5-year history of bilateral lower extremity weakness and elevated creatine phosphokinase levels. MR imaging was performed to guide biopsy. Axial T1-weighted **(a)**, STIR **(b)**, and postcontrast T1-weighted fat-suppressed **(c)** MR images of the proximal calf show complete fatty atrophy of the medial head of the gastrocnemius *(G_m)* and medial aspect of the soleus *(S_m)* muscles, indicating chronic involvement. There is moderate fatty atrophy, edema, and enhancement of the lateral head of the gastrocnemius *(G_l)*, and to a lesser degree, the lateral aspect of the soleus *(S_l)* and tibialis posterior *(TP)* muscles. In the distal half of the calf (not shown), there is mild fatty atrophy, edema, and enhancement of the anterior compartment muscles. Similar findings are seen in the contralateral leg (not shown). The anterior compartment muscles of the leg were targeted as an optimal site for biopsy with the highest potential diagnostic yield, given the presence of muscle edema, enhancement, and lack of significant muscle atrophy. The biopsy results helped confirm IBM.

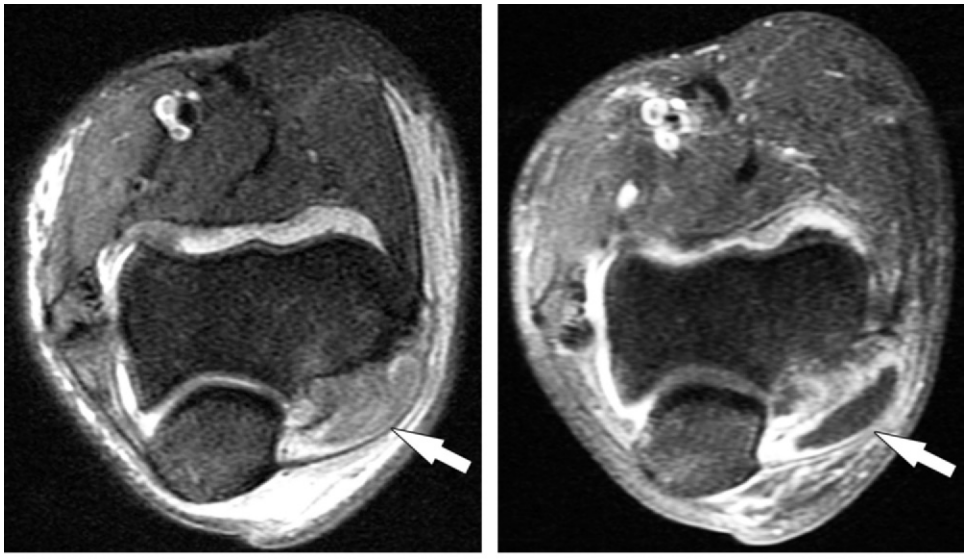
syndrome of SLE, scleroderma, and PM), with muscular involvement similar to that of PM and with similar imaging findings (67,68). Rheumatoid myositis is well documented in the literature but overall is an underdiagnosed and undertreated disease with a dearth of imaging findings in the radiologic literature (69). At MR imaging, focal or diffuse increased muscle signal intensity with fluid-sensitive sequences with varying degrees of enhancement are the most common findings (Fig 19).

Traumatic Soft-Tissue Injuries

Different patterns of muscular injury can mimic soft-tissue infection. The key in differentiating infection from trauma-related muscular injury at MR imaging is clinical history. Traumatic muscular injury can be in the form of laceration, contusion, strain, muscle hemorrhage and hematoma, compartment syndrome, or myositis ossificans or be exercise related.

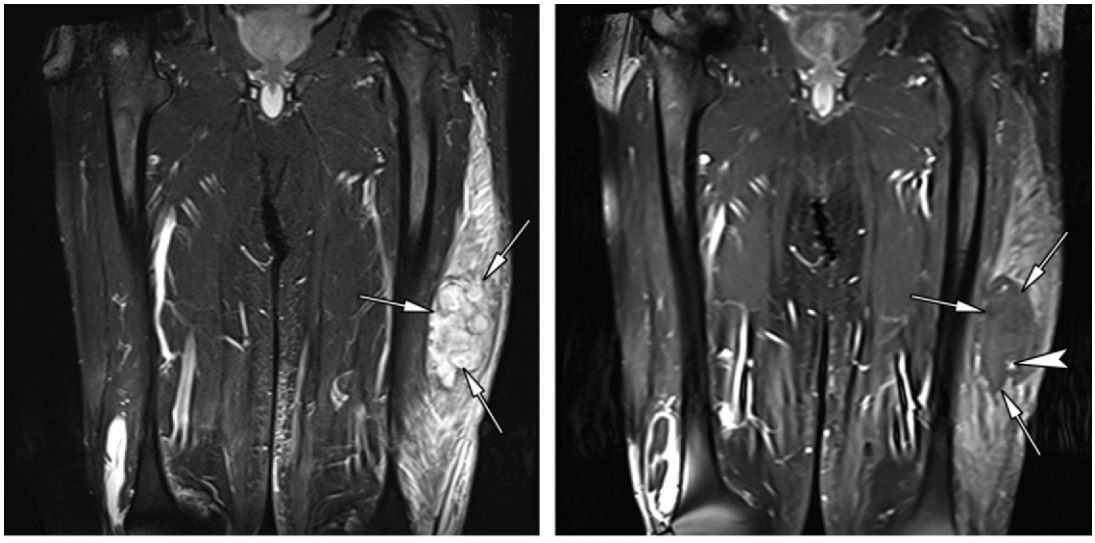
Muscular contusion is the result of a direct external blow. MR imaging findings include interstitial hemorrhage and edema at the site of injury and in the underlying muscle. If contusion is severe enough, a hematoma may develop that appears as a masslike lesion, in addition to the edema. MR signal intensity characteristics of blood depend on its age. Subacute blood shows high T1 signal intensity from methemoglobin. Chronic blood breakdown and hemosiderin deposition shows low-signal-intensity rim with all pulse sequences. Intramuscular hematomas may also occur in the absence of trauma, either spontaneously or in patients who are receiving anticoagulation therapy (Fig 20). The former, however, may be due to an underlying soft-tissue mass, and therefore a spontaneous hematoma should be followed up with serial imaging to document complete resolution and exclude underlying neoplasm (48).

Muscular strain affects the musculotendinous junction and frequently occurs in muscles that



a. b.

Figure 19. Myonecrosis in a 36-year-old woman with systemic lupus erythematosus presenting with elbow pain. **(a)** Axial T2-weighted fat-suppressed MR image shows edema (arrow) in the anconeus muscle with overlying subcutaneous tissue edema extending anteromedially. **(b)** Axial postcontrast T1-weighted fat-suppressed MR image demonstrates peripheral enhancement (arrow) of the anconeus muscle with a central area of nonenhancement, consistent with myonecrosis.



a. b.

Figure 20. Nontraumatic intramuscular hematoma in a 58-year-old man with thigh pain and swelling for 7 days and no history of trauma or anticoagulation therapy. **(a)** Coronal STIR image shows a heterogeneous multiloculated soft-tissue collection (arrows) within the vastus lateralis muscle causing bulging of the overlying fascia. **(b)** Coronal postcontrast T1-weighted fat-suppressed MR image demonstrates nonenhancement of the collection (arrows) except for a single punctate region of enhancement inferiorly (arrowhead), which may represent an active bleeding vessel or less likely a small vascular malformation. Also note the extensive edema of the vastus lateralis muscle above and below the collection with corresponding muscle enhancement, which is probably reactive to the collection. Intramuscular abscess and diabetic myonecrosis are in the differential diagnosis, but the patient was not diabetic and infection was not clinically suspected. Open biopsy of the mass and evacuation were performed, which helped confirm the diagnosis of a spontaneous intramuscular hematoma, with no underlying malignancy or vascular malformation found at histopathologic analysis.

cross two joints, such as the hamstring, gastrocnemius, and biceps brachii muscles. MR imaging reveals edema centered at the myotendinous junction. In the severe form of muscular strain, hematoma may develop and appear as an intramuscular masslike lesion (48).

Delayed-onset muscle soreness (DOMS) is a form of overuse muscle injury. As its name implies, the symptoms are delayed by hours to days after the overuse injury. This clinical presentation is in contrast to a muscle contusion or strain, where pain occurs immediately (Fig 21).

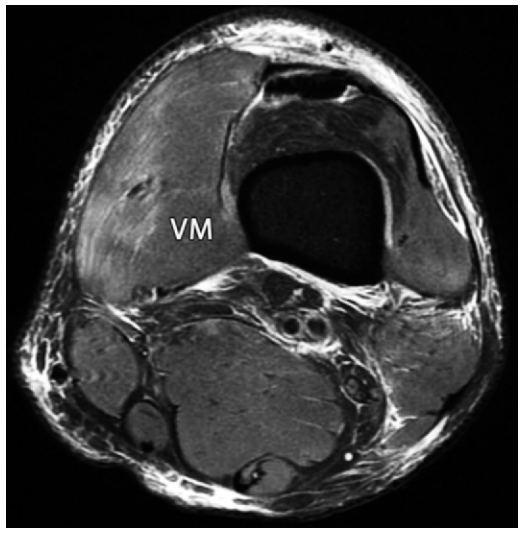


Figure 21. DOMS in a 33-year-old man with onset of knee pain 3 days after intense exercise in a gym. Axial proton density–weighted fat-suppressed MR image of the knee shows edema involving all muscles about the knee, most prominent in the vastus medialis muscle *(VM)*. These findings are consistent with a diagnosis of DOMS.

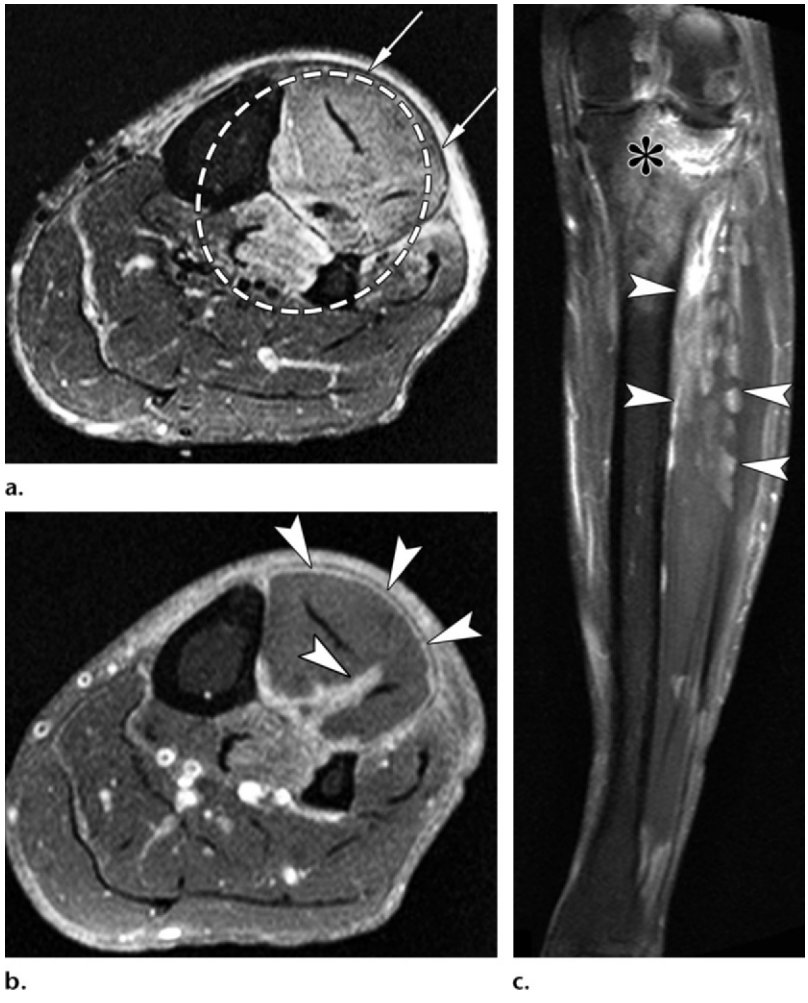


Figure 22. Compartment syndrome, rhabdomyolysis, and myonecrosis in a 33-year-old man with a medial tibial plateau fracture. **(a)** Axial STIR image of the left leg shows edema in anterior and anterolateral subcutaneous tissue also extending to the deep fascia. There is edema (outline) in anterior and deep posterior muscle compartments. There is bulging (arrows) of the superficial layer of the deep fascia, suggestive of compartment syndrome. **(b, c)** Axial **(b)** and coronal **(c)** postcontrast T1-weighted fat-suppressed MR images show nodular and irregular thick enhancement (arrowheads) of the superficial and deep fascia peripheral to the anterior compartment muscles with loss of muscle architecture, consistent with myonecrosis. Also note the medial tibial plateau fracture with accompanying marrow enhancement (*), reflecting hyperemia.

Rhabdomyolysis may accompany more severe cases of DOMS (48).

Rhabdomyolysis is caused by a rapid breakdown of muscle fibers with subsequent release of cellular protein contents into the systemic circulation. There are multiple causes for rhabdomyolysis, which can be classified into traumatic (eg, crush injury, excessive exercise) and nontraumatic (eg, extreme body temperature change, arterial occlusion, metabolic disturbances, toxins, inflammation, infection) causes (70). The most common site of involvement is the lower extremity and lower back. MR images show edema in the involved muscles, but the adjacent fasciae are rarely involved (Figs 13, 22).

Rhabdomyolysis may resolve or progress to myonecrosis, depending on the severity of injury. At MR imaging, the signal changes can be homogenously or heterogeneously hyperintense at T2-weighted imaging. Following contrast material administration, they enhance either homogenously or show a stippled pattern of enhancement (Fig 22) (71). The *stipple sign* on postcontrast MR images is attributed to residual viable muscle fibers or inflammatory vessels within infarcted muscles (71).

Compartment syndrome is the result of increased pressure within a confined space compromising capillary blood flow and resulting in tissue ischemia. There are multiple possible causes that lead to increase of compartment volume (eg, hematoma, edema) or restriction of compartment size (eg, constrictive dressings and casts, closure of fascial defects). In the acute stage, patients present with severe pain with or without neurologic symptoms. Management of acute compartment syndrome is immediate fasciotomy and decompression of all tissues within the affected compartment. Chronic compartment syndrome, also known as chronic exertional compartment syndrome, occurs gradually after repetitive motion or exercise and does not require emergency medical attention. The clinical presentation of chronic compartment syndrome is pain on exertion that is relieved after activity cessation.

There is a limited role for imaging in compartment syndrome. At MR imaging, there is enlargement and diffuse increased signal intensity in the muscles within boundaries of a compartment in the acute phase. Edema and hemorrhage may also be present between muscles in fascial planes (Fig 22). Postcontrast images show intense enhancement of the involved compartment due to increased cell membrane permeability (72). If areas of myonecrosis are present, loss of muscle architecture at T1-weighted imaging with lack of enhancement of the involved area with postcontrast sequences is seen. In the chronic phase, muscular fibrosis, calcification, cystic changes, or muscular atrophy may be seen (72).

Myositis ossificans is defined as heterotopic formation of ossification in soft tissue, usually as a result of trauma; however, it can occur spontaneously or in other conditions such as paralysis, burns, and intramuscular hematomas. Initially at MR imaging, the involved muscle demonstrates heterogeneous edema. Progression to a masslike lesion characterized by high T2 signal intensity makes it difficult to distinguish from soft-tissue sarcoma. As the lesion evolves over a period of 6–8 weeks, a characteristic peripheral calcification develops with corresponding low signal intensity with all MR pulse sequences (Fig 23) (73).

Muscle Denervation

Peripheral neuropathy should show signs at clinical examination, confirmed and localized to a specific anatomic region by electromyography. MR imaging can then be used to look for surgically correctable causes, such as localized nerve compression by a ganglion cyst or mass. MR imaging can be normal in acute muscle denervation, but may show intramuscular edema within 24 hours. Subacute muscle denervation occurs 2–4 weeks following an insult (74). In the early subacute form, uniform edema involving the muscles innervated by the injured nerve results in paradoxical muscle enlargement due to either pseudohypertrophy or true hypertrophy of the muscles (75). MR imaging findings in early subacute muscle denervation include diffuse T2 hyperintense signal and normal T1 intensity signal of the muscles and absence of subcutaneous edema (Fig 24). Mixed muscle edema and atrophy occur in the late subacute form, followed by muscle atrophy in the chronic form over months. It is important to recognize muscle denervation in its early stages for possible restoration of the nerve before fatty atrophy and irreversible muscle changes occurs (74).

Neoplasm and Post-therapy Soft-Tissue Changes

Neoplasms may cause hyperintense signal with fluid sequences in the surrounding soft tissues and muscles. The signal intensity alteration in the muscles surrounding a soft-tissue tumor may reflect tumor invasion, edema, or both (76). Distinguishing the two may be necessary, as accurate delineation of tumor margins is essential for surgical planning and radiation therapy. Unfortunately, there are no MR imaging findings to reliably distinguish between edema and tumor invasion. When the involved muscle is thickened and the border between the muscle and tumor is indistinct, tumor invasion is favored over reactive edema (76).

Chemotherapy can result in an apparent increase in tumor size initially from intratumoral hemorrhage, which should not be confused with tumor growth. The effectiveness of chemotherapy is assessed by the degree of tumor necrosis at contrast-enhanced MR imaging (77). Chemotherapy can also cause muscle edema and myonecrosis in normal muscles (78).

Radiation therapy is commonly used in treatment of high-grade soft-tissue sarcomas in the form of either adjuvant or neoadjuvant therapy. Soft-tissue changes related to radiation therapy include subcutaneous lattice-like edema with overlying skin thickening and radiation-induced muscle edema (77). Muscle edema is typically diffuse with sharp and straight margins delineating

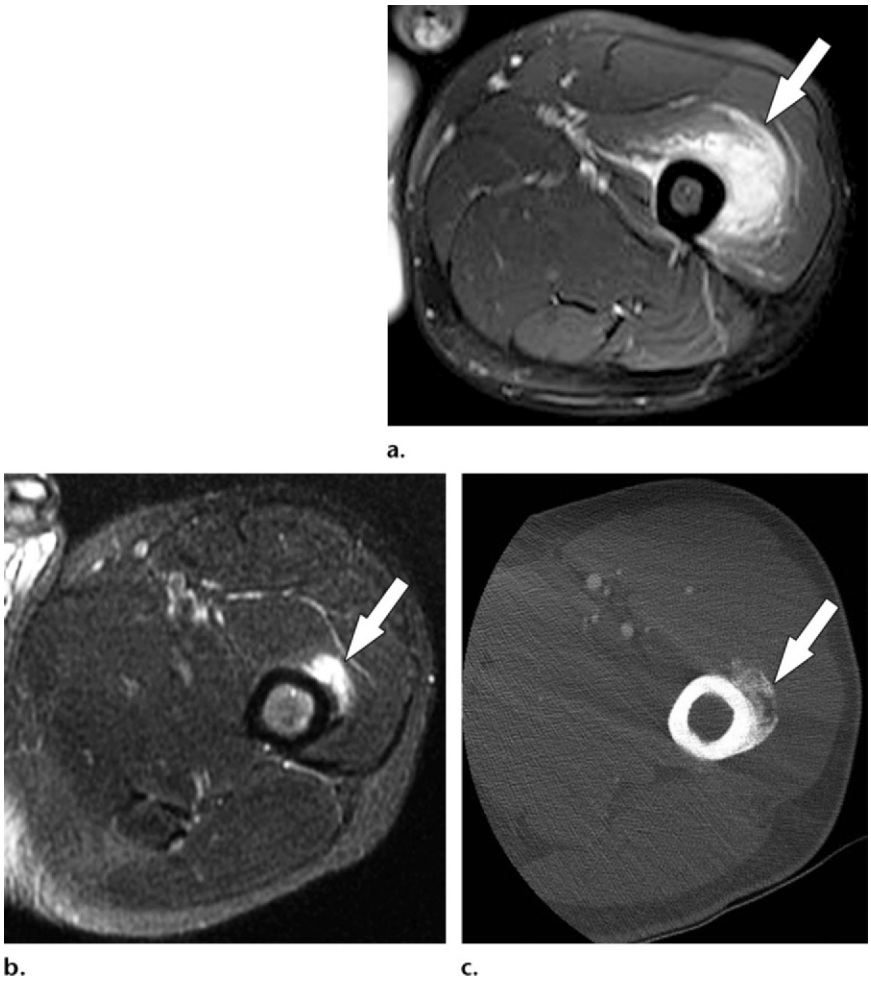


Figure 23. Myositis ossificans in a 21-year-old man with a thigh mass and a questionable history of trauma a few weeks prior. **(a)** Axial T2-weighted fat-suppressed MR image shows an inhomogeneous hyperintense masslike region with surrounding edema (arrow) on the surface of the anterolateral cortex of the proximal femoral diaphysis. No associated cortical erosion or marrow edema is seen. Fluorine 18 fluorodeoxyglucose (FDG) positron emission tomography (PET)/CT showed avid radiotracer uptake in the region (not shown). **(b)** Axial STIR image obtained 3 months later shows significant interval decrease in the size of the mass (arrow) and resolution of the surrounding edema. **(c)** Axial CT image obtained 1 month later shows maturing periosteal reaction (arrow) with central lucency and peripheral ossification, characteristic of myositis ossificans. This case illustrates how the early stages of myositis ossificans may resemble aggressive processes such as sarcoma. Even at pathologic analysis, differentiating between myositis ossificans and sarcoma can be challenging. A thorough clinical history and high level of clinical suspicion are necessary to avoid unnecessary and aggressive treatment.

the radiation field (Fig 25), with low-grade muscle enhancement following contrast material administration (48,77).

Drug and Toxin-induced Myositis

A wide range of drugs and toxins can have myopathic effects (79). One of the more extensively reported classes of medications with adverse muscle effects is statins. Statins act by inhibiting cholesterol synthesis. Statin-induced myopathy has a spectrum of clinical and laboratory findings ranging from myalgia to myositis to rhabdomyolysis (79). The incidence of statin-induced symptoms is dose-dependent and is seen in less than 1% of patients taking these medications. The most common sites of involvement are the posterior compartment muscle groups of the thigh and leg, and the distribution can be focal or diffuse and bilateral (80). At MR imaging, statin-induced myositis and rhabdomyolysis are characterized by focal or diffuse muscle edema (Fig 26). Drug discontinuation usually results in symptom relief and resolution of MR imaging signal intensity abnormalities.

Diabetic Myonecrosis

Diabetic myonecrosis is a rare manifestation of long-standing (mean duration, about 15 years) and poorly controlled diabetes mellitus and can be seen in both type I and II diabetes (81,82).

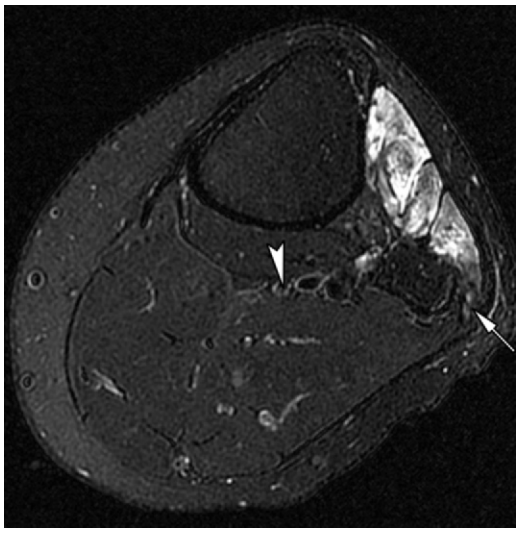


Figure 24. Subacute denervation in a 34-year-old woman with foot drop. Axial STIR image of the calf shows widespread edema of the muscles of the anterior and lateral compartments. There is slightly increased signal intensity in the deep and superficial branches of the common peroneal nerve (arrow), reflecting neuritis. Note the normal signal intensity of the tibial nerve (arrowhead) for comparison. These findings are consistent with subacute denervation of muscles innervated by the common peroneal nerve. Note the absence of subcutaneous edema, which makes infectious and inflammatory processes less likely.

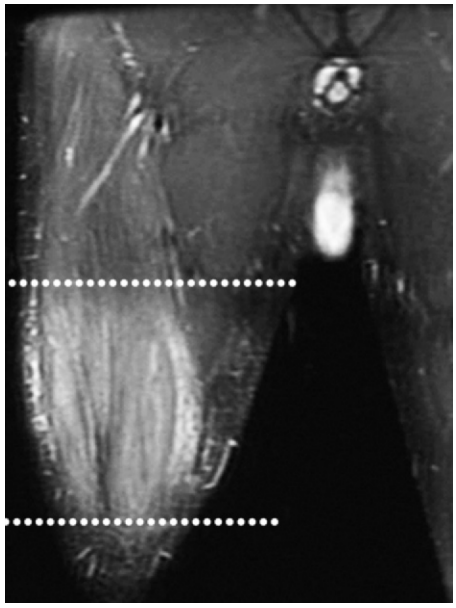


Figure 25. Postradiation myositis in a 40-year-old man with soft-tissue sarcoma of the ankle after local radiation therapy for a metastatic focus in the midshaft of the femur (the primary and the metastatic focus are not shown). Coronal STIR image of the thigh shows edema in the anterior compartment muscles. An abrupt bandlike appearance of intramuscular edema (dotted lines) corresponds to the prescribed radiation field.

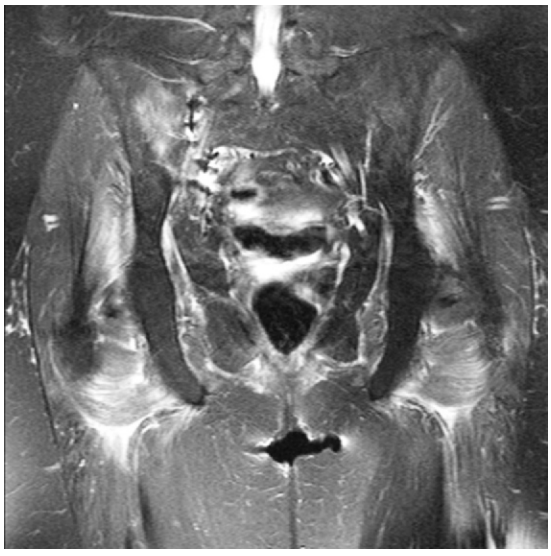


Figure 26. Myositis in a 57-year-old man on statin medication for hypercholesterolemia who presented with myalgias. Coronal STIR image of the pelvis shows diffuse and nearly symmetric muscle edema involving the gluteal, obturator, and quadratus femoris muscles. The adductor muscles were also involved bilaterally. Although nonspecific, these findings are consistent with statin-induced myositis in this patient, who had been on a high dose of statin medication.

The most accepted cause for myonecrosis is diffuse microangiopathy. Patients present with acute pain, swelling, and induration of the involved extremity for several weeks. Signs of systemic infection (eg, fever and leukocytosis) are usually absent. The anterior thigh muscle groups followed by the posterior calf muscles are the most common sites of involvement, and bilateral involvement is frequent (81,83). At MR imaging, varying degrees of subcutaneous and fascial edema and fluid are frequently demonstrated (81,84,85). The involved muscles show hyperintense signal at T2-weighted imaging, representing muscle ischemia (Fig 27), or regions of hyperintense signal at T1-weighted imaging, reflecting hemorrhagic muscle infarction (81). Two patterns of muscle enhancement may be seen: *(a)* diffuse enhancement with a similar distribution of altered signal intensity at T2-weighted imaging, representing muscle ischemia, or *(b)* intense rim enhancement around masslike regions of the muscle with linear streaks of enhancement crossing central nonenhancing regions, reflecting myonecrosis (81,85,86). Nevertheless, the pattern of enhancement in diabetic myopathy (diffuse vs rim) does not seem to affect the clinical outcome, and therefore intravenous

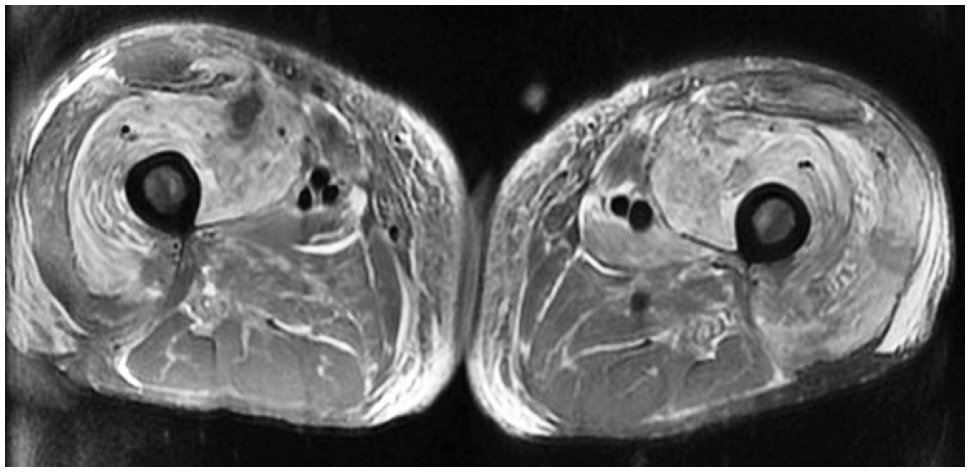


Figure 27. Myonecrosis in a 57-year-old man with uncontrolled type II diabetes who presented with thigh pain. Axial STIR image of the bilateral thighs demonstrates diffuse edema involving the left adductor longus and sartorius muscles and bilateral quadriceps musculature, with relative sparing of the vastus lateralis and rectus femoris muscles on the right. There is extensive subcutaneous soft-tissue swelling. Although these findings are nonspecific, the diagnosis of diabetic myonecrosis was established clinically, and the patient was managed conservatively, with eventual resolution of symptoms.

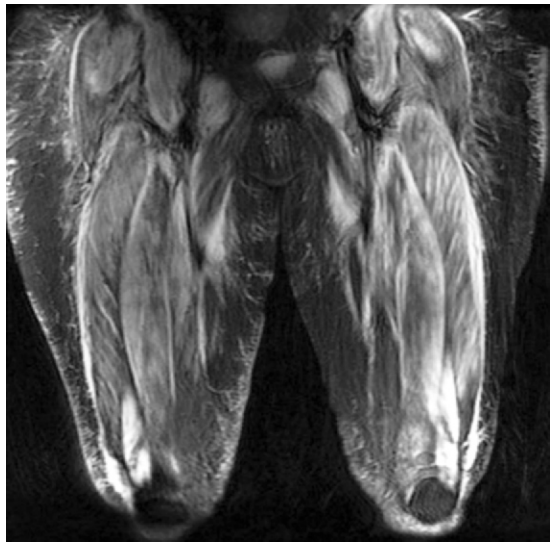


Figure 28. Polymyositis in a 38-year-old man with HIV infection being treated with AZT. Coronal STIR image of the proximal thighs shows bilateral symmetric muscle edema with accompanying subcutaneous edema. The clinical and imaging findings are suggestive of either HIV polymyositis or myositis secondary to antiretroviral therapy.

contrast material might not be necessary (81). In addition, in patients with diabetic nephropathy, gadolinium contrast agents should be used cautiously. In chronic forms, diabetic myonecrosis shows muscle atrophy with or without calcification (51). Differentiating soft-tissue infection (eg, NF, pyomyositis) from diabetic myopathy may be difficult at MR imaging, as the imaging findings may overlap. Clinical history, absence of signs of systemic infection, bilateral involvement, and multiple noncontiguous sites of involvement favor diabetic myopathy over infection (81). Additionally, in diabetic myonecrosis, the infarcted muscles manifest as regions of heterogeneous signal intensity without a well-defined intramuscular abscess, as seen in pyomyositis (84). The distinction between soft-tissue infection and diabetic myopathy is important, as conservative management is favored over surgical débridement in the latter presentation (87).

HIV Myositis

A wide spectrum of musculoskeletal disorders can be detected in HIV-positive patients, such as infection, inflammation, autoimmune disorders, HIV- and antiretroviral therapy–associated neuropathy, and tumors (88). Muscle involvement in HIV-positive patients could be due to host immunologic response to the virus (ie, HIV myositis), secondary to azidothymidine (AZT) therapy or infection (88). The most significant factor distinguishing infection from HIV myositis is symmetric and bilateral involvement in myositis (Fig 28). Infections are often unilateral or at least asymmetric. AZT myositis is dose related and improves on cessation of therapy.

Conclusion

Diagnosing superficial and deep musculoskeletal soft-tissue infections is challenging in clinical practice. Although superficial infections are almost always managed medically, involvement of deep soft tissue may require surgical attention. Imaging can be performed to confirm the

diagnosis and define the extent of the disease; however, it should never delay treatment. Knowledge of the imaging findings, patterns of soft-tissue involvement, and differential diagnoses enable radiologists to be effective team members in the management of these patients.

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