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Seminars in
ROENTGENOLOGY

Musculoskeletal Faces of Death: A Diagnostic Imaging Review

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Introduction

Cellular death of the extremities encompasses a broad variety of pathologies. From avascular necrosis to necrotizing fasciitis, diseases of the musculoskeletal system are a common cause of morbidity and mortality.[1-3] Delayed diagnosis of any process that leads to tissue death can lead to poor outcomes.[3] In this article, we review the cellular death of bone, muscle, fascia, subcutaneous adipose tissue, and skin. Avascular necrosis, bone infarction, sequestrum in chronic osteomyelitis, myonecrosis, necrotizing fasciitis, and gangrene are discussed. The conventional radiographic, computed tomographic, and magnetic resonance imaging findings of each of the pathologies are outlined. Proper understanding of how these devastating diseases appear on different imaging modalities is imperative to practicing radiologists.

Avascular Necrosis

Pathophysiology

By convention, avascular necrosis (AVN) is a term that has historically been used to describe death of bone tissue, or osteonecrosis, specifically involving the subchondral surface of bone. Although a large proportion of AVN is due to ischemia and interruption of vascular supply, other multifactorial pathophysiologic mechanisms may result in the same disease process, such as direct toxicity by means of radiation or chemotherapy.[4,5] Therefore, this term has fallen out of favor in the recent years. For the purpose of distinguishing this process from infarct elsewhere in the bone, discussed subsequently, the term AVN will be used to describe subchondral osteonecrosis, ischemic or otherwise.

As interruption of vascular supply constitutes the mechanism of a large portion of AVN cases, any process that limits blood supply to the subchondral surface may thus predispose a patient to avascular necrosis. Representative etiologies include sickle-cell disease, hematopoietic disorders that can lead to excessive clotting, and trauma, in addition to other factors such as corticosteroid use, radiation, and alcohol abuse. It is often an indolent process and if left untreated, can lead to devastating consequences for the patient with high morbidity, including long-term joint failure. For this reason, it is critical that avascular necrosis is identified and managed expediently.[4,5]

Clinical Features

The most well-known manifestation of avascular necrosis is osteonecrosis of the femoral head. This can occur at almost any age, however in children and teenagers, this disease process is termed Legg-Calvé-Perthes disease.[4] If not incidentally discovered on radiologic evaluation during the earliest subclinical stages, the most common initial clinical presentation is worsening hip pain, unilateral or bilateral, along with limited hip motion.[5] Though less common, this process may occur at other sites, such as the humeral head.[4]

Treatment of avascular necrosis may begin conservatively with rest and anti-inflammatory medications, however it may require core decompression or eventually joint replacement for end-stage disease. Core decompression is predominantly used in the precollapse stages, in hopes that it may delay or negate the necessity for subsequent total hip arthroplasty. The osteonecrotic lesion is cored using a single large or multiple smaller drillings. Adjunctive measures have been trialed including bone grafting and the use of mesenchymal

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stem cells, however the overall goal remains the same. When used in the precollapse stages of AVN, core decompression is effective at preventing conversion to total hip arthroplasty, however is much less effective in this regard when employed after subchondral collapse has occurred.[6]

Imaging Features

Conventional Radiograph

Although the gold standard for diagnosing avascular necrosis is magnetic resonance imaging (MRI), the first study typically ordered by clinicians is a conventional radiograph. Radiographs may demonstrate areas of sclerosis at the articular surface, and later, the classic "crescent sign," denoted by a subchondral lucency, which is an ominous sign for impending articular collapse (Figs. 1 and 2). In osteonecrosis of the femoral head, flattening of the femoral head is common, however it may be difficult to detect on initial radiographs. As the condition progresses, degenerative changes of the hip joint become increasingly obvious.[4,7] Although multiple classification systems have been developed to incorporate other modalities and aspects of this disease process, the most well-known classification system on conventional radiograph is the Ficat and Arlet staging system (Table 1).[4]

Although the femoral head is the prototypic location where AVN may occur, it can, and often does, occur in other bones in which the blood supply becomes increasingly diminutive. It is important to note that although conventional radiograph is a good first diagnostic test for suspected AVN, early AVN often has an occult or "normal" appearance, and it can take up to 3 months to become evident on conventional radiograph.[7] Therefore, in the setting of a high index of suspicion for AVN despite a normal radiograph, a computed tomography (CT) scan or MRI study should be ordered for further evaluation.[4,7]

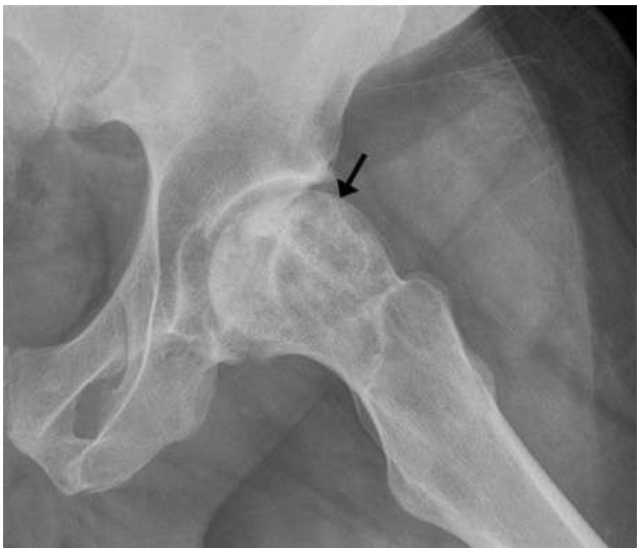


Figure 1 A 54-year-old man with avascular necrosis of the left femoral head. A frog-leg lateral radiograph of the left hip demonstrates subarticular lucency in the left femoral head with femoral head collapse (*arrow*).

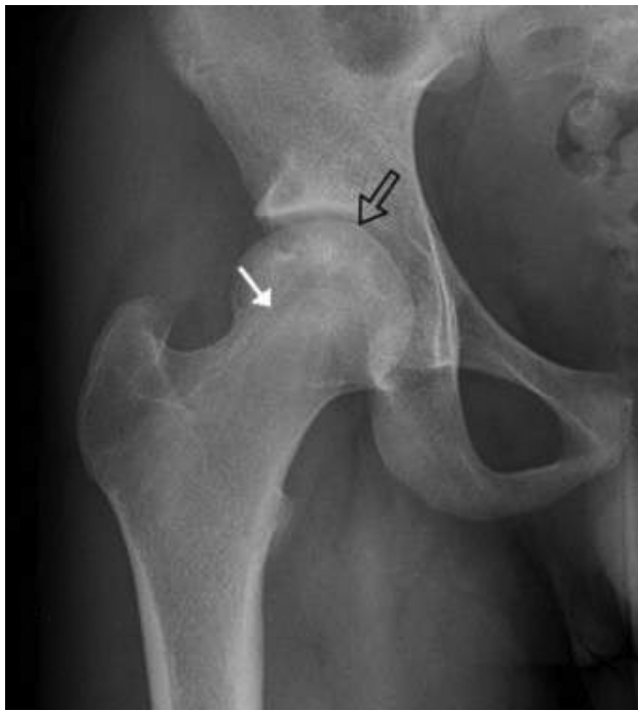


Figure 2 A 21-year-old man with avascular necrosis and prior core decompression of the right femoral head. An AP radiograph of the right hip demonstrates subarticular sclerosis in the right femoral head (*hollow black arrow*) and prior core decompression (*white arrow*).

Computed Tomography

Though not as sensitive as MRI for the detection of avascular necrosis, CT is the most sensitive modality for the detection of a subchondral fracture. CT may also demonstrate the classic appearance of serpiginous sclerosis with central lucency (Fig. 3). Evaluation of the overall morphology of the involved joint and articular surfaces is an additional strength of this modality.[6]

MRI

MRI is 99% sensitive and specific for the detection of avascular necrosis.[4] There is a spectrum of appearances on MRI depending on the stage of AVN. Often, the first finding on MRI is the "reactive interface line," typically a low signal serpiginous line on T1-weighted imaging that discriminates necrotic from viable bone. MRI may also reveal the classic

Table 1 Radiographic Classification of Osteonecrosis[4]

Ficat and Arlet Staging	
I	Normal
II	Osteosclerotic or cystic changes with normal sphericity
	A—No crescent sign
	B—Crescent sign (subchondral collapse)
III	Flattening of the femoral head
IV	Osteoarthrosis with joint space narrowing and articular collapse

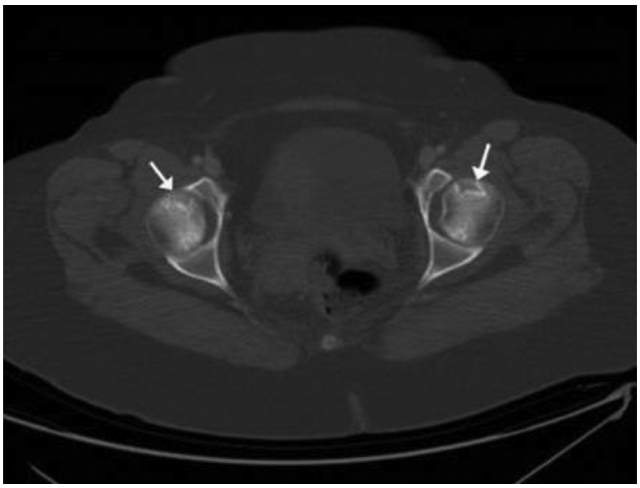


Figure 3 A 49-year-old woman with avascular necrosis of the bilateral femoral heads. An axial CT image through the level of the femoroacetabular joints demonstrates serpiginous sclerosis with central lucency (*arrows*), denoting the osteonecrotic regions of the subchondral femoral heads.

"double line sign," which is denoted by a low intensity line outlining the infarct, indicative of sclerosis, with a relatively hyperintense line more centrally within the affected bone tissue, indicative of granulation tissue (Fig. 4).[4,6] Unlike many other disease processes, edema is visualized later in the course of AVN and is not an early manifestation. Edema is important to note, however, as it is a good indicator of the final stage in which core decompression may be of utility. After the more immediate hemorrhage and subsequent edema, MR characteristics do not change until the healing process commences and fibrosis begins to develop, a process that may take months. At this stage, signal will be dark on both T1 and T2 weighted sequences.[6]

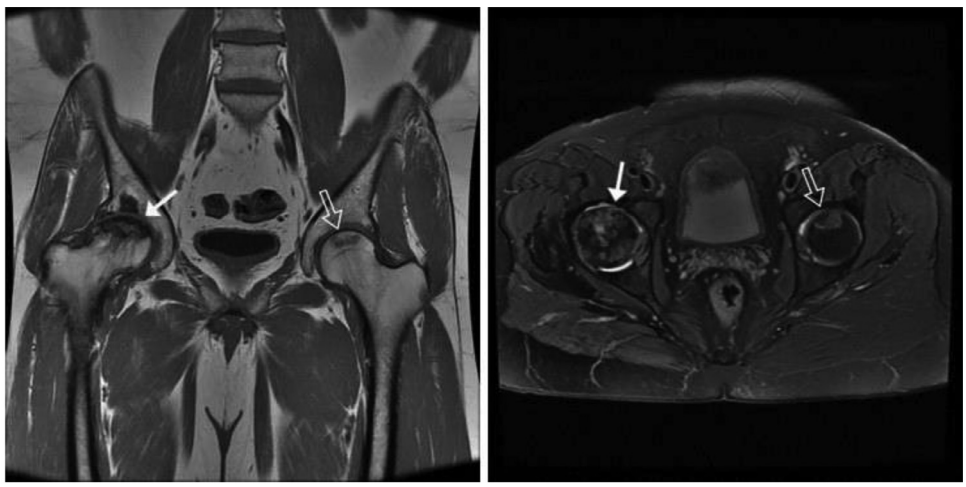
Bone Infarct

Pathophysiology

In contrast to avascular necrosis, which traditionally refers to a subchondral osteonecrotic process, bone infarct characteristically occurs within the medullary diaphysis or metaphysis of a long bone, typically through a similar process of interrupted vascular blood supply, ultimately leading to bone destruction. Causes are varied, and often idiopathic, however several well-known predisposing factors and conditions include hemoglobinopathies, such as sickle-cell disease and the thalassemias, corticosteroid use, alcoholism, Gaucher disease, and Caisson disease, among others. The mechanism of interrupted vascular supply depends on the specific etiology of a patient's bone infarct, and may include increased marrow pressure, embolic phenomena, or decreased vessel caliber.[8,9]

Clinical Features

As with avascular necrosis, the initial presenting clinical feature of bone infarct is pain at the site of infarction. Subsequent treatment is aimed at the underlying cause. In the common setting of idiopathic bone infarct, there is often no treatment required, and the infarct is typically self-limited with good prognosis. Cessation of corticosteroids, when possible, or alcohol, may help prevent future occurrences in those patients with these exogenous predisposing factors. Though rare, the patient's provider should also remain vigilant to the possibility of malignant transformation. In the setting of new or worsening symptoms, such as pain, at a site of prior infarct, CT should be considered as an initial investigative tool for evaluation of possible malignant transformation.[10]



(A) (B)

Figure 4 A 47-year-old man with avascular necrosis of the bilateral femoral heads. Coronal T1 (A), and axial T2 fat-suppressed (B) MR images of the pelvis demonstrate flattening and collapse of the right femoral head (*solid arrows*). Also demonstrated is a region of avascular necrosis in the left femoral head without collapse (*hollow arrows*).

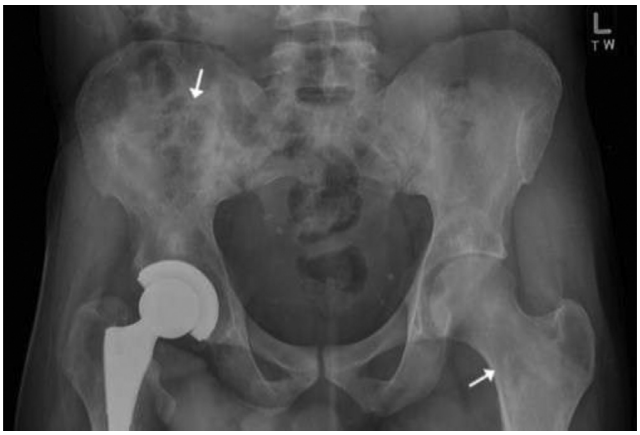


Figure 5 A 36-year-old man with a history of sickle cell disease and multiple bone infarcts. An AP radiograph of the pelvis reveals patchy sclerosis (*arrows*) throughout the pelvis, left femoral neck and intertrochanteric femur compatible with bone infarcts.

Imaging Features

Conventional Radiograph

Conventional radiograph is often not immediately helpful in detecting acute bone infarction due to a delay from initial infarction to radiographic manifestations. The radiographic appearance of early bone infarction is nonspecific and may demonstrate a wide range of appearances including mild reactive sclerosis (Fig. 5), periostitis, bone rarefaction, or may even appear completely normal.[11] Radiographic manifestations of more advanced and chronic bone infarction include intramedullary dystrophic calcifications with a central radiolucent area and a serpentine border.[11,12]

Computed Tomography

CT does not provide as definitive of an evaluation as MRI in the detection of bone infarction, however may demonstrate findings similar to conventional radiography (Fig. 6), with the added benefit of higher sensitivity in the detection of associated calcifications. Specific etiologies may exhibit classic findings. For example, a classic CT manifestation of sickle-cell disease related bone infarction is an H-shaped vertebral body deformity that results from infarction of the central growth plate. [13]

MRI

MRI allows for the discernment of bone infarction from alternative entities. For instance, osteomyelitis may be similar-appearing. Intravenous contrast is required in order to make this distinction, however. In osteomyelitis, irregular peripheral enhancement is observed on postgadolinium images, with patchy central enhancement, a feature distinct from bone infarct. Bone infarct will fail to enhance centrally secondary to the devitalized nature of the affected bone.[11]

Cystic degeneration, an uncommon result of bone infarction, may appear as thin-walled cystic expansion of the involved bone, which is visible on conventional radiograph and CT. When evaluated on MRI, the lesion will demonstrate decreased T1 signal and heterogeneously bright T2 signal (Fig. 7). Contrast-enhanced images will reveal a well-defined enhancing rim surrounding fluid of low signal intensity.[12]

Sarcomatous degeneration, an even more unusual consequence of bone infarction, may manifest as an aggressive-appearing lytic lesion with cortical invasion and an associated soft tissue mass, most commonly with transformation to malignant fibrous histiocytoma. MRI will again demonstrate heterogeneous T2 hyperintensity and contrast-enhanced images will reveal patchy enhancement with areas of necrosis.[14,15]

Sequestrum in Chronic Osteomyelitis

Pathophysiology

Osteomyelitis is an often devastating disease process characterized by inflammation of bone, typically secondary to bacterial infection. Usually, infection reaches the bone by hematogenous spread; however trauma or ulceration may result in direct inoculation of bone. Once infected, the bone progresses through multiple stages of an acute inflammatory response, typically over the course of days to weeks, which may further progress to chronic osteomyelitis if left untreated or if refractory to treatment. Development of a sequestrum is the touchstone of chronic osteomyelitis and is an often-used feature to establish the progression from acute to chronic osteomyelitis. Prior to the advent of antibiotics, cases of osteomyelitis were often deadly,

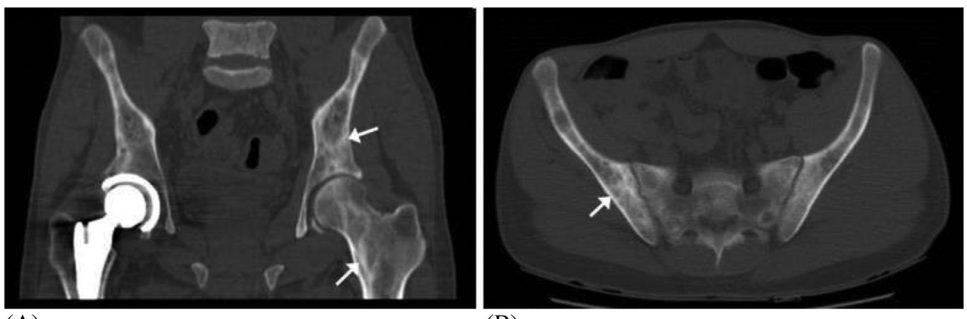


Figure 6 A 36-year-old man with a history of sickle cell disease and multiple bone infarcts. Coronal (A) and axial (B) CT images reveal patchy sclerosis (*arrows*) throughout the pelvis, left femoral neck, and intertrochanteric femur compatible with bone infarcts.

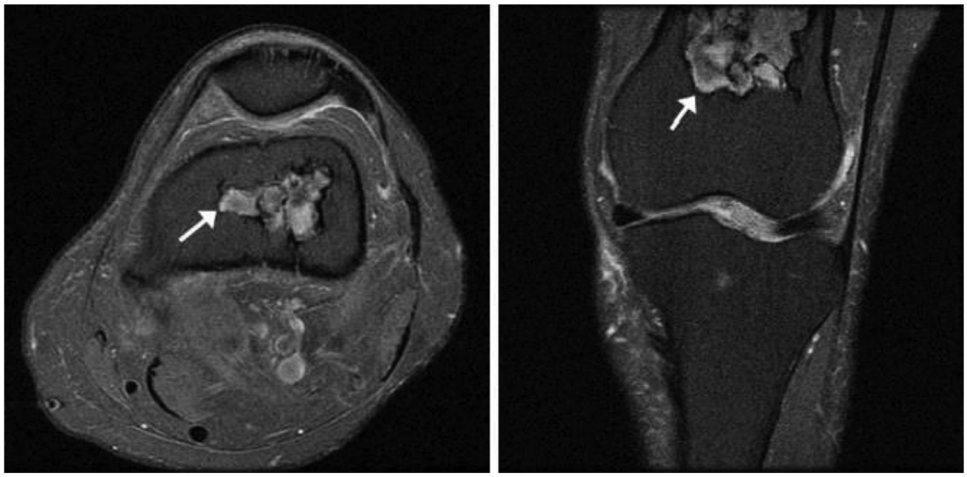


Figure 7 A 65-year-old woman with a bone infarct in the left distal femoral metadiaphysis. Axial proton density fat-suppressed (A) and sagittal T2 fat-suppressed (B) MR images of the left lower extremity demonstrate a serpiginous mixed signal abnormality in the distal femoral metadiaphysis (*arrows*) compatible with a bone infarct.

even in the acute setting, and therefore chronic osteomyelitis was relatively uncommon.[16]

A sequestrum is a portion of devascularized bone that has separated from adjacent living bone by means of necrosis and resorption of neighboring bone. The pathophysiology of this process is thought to involve increasing medullary pressure secondary to accumulating exudate within the marrow, leading to compression of the vascular channels and subsequent interruption of the bone's vascular supply. Once the sequestrum forms, it may act as a nidus for continued infection, as the lack of blood supply prevents effective treatment with antibiotics. It should be noted that sequestra may also be seen in the context of intracortical abscesses, osteoid osteoma, osteoblastoma, lymphoma, and other diseases of the bone.[16] As the disease process progresses, the sequestrum may form a thick new periosteum, known as an involucrum. This may be further complicated by an opening in the involucrum, known as a cloaca, which allows for the drainage of purulent material from the dead bone. Chronic osteomyelitis occurs most often in patients with vascular insufficiency, classically diabetics, with an amputation rate of up to 66%.[16,17]

Clinical Features

Presenting features of osteomyelitis will depend on the acuity of the process and the patient's comorbid conditions. Initially, there may be nonspecific systemic signs of infection such as fever, chills, and fatigue. Alternatively, local symptoms may present first. In diabetic patients, growing soft tissue ulcerations secondary to neuropathic disease may prompt initial investigation. In patients with hematogenous infection of bone, pain may be the initial presenting symptom.[17,18]

The mainstay of treatment for osteomyelitis is antibiotic therapy. In patients such as diabetics and those with peripheral vascular disease, compromised vasculature may preclude effective antibiotic treatment, and other more invasive surgical measures such as amputation may need to be undertaken in order to prevent systemic infection and ultimately, death. Additionally, though unusual, providers should also be vigilant of the possibility of malignant degeneration within areas of chronic inflammation, such as in those patients with recalcitrant chronic osteomyelitis. This often presents as an ulcerating squamous cell carcinoma, known as a Marjolin ulcer.[18]

Imaging Features

Conventional Radiograph

Conventional radiographs are often normal-appearing in the initial stages of osteomyelitis. An early finding may include soft tissue swelling without a definite bony abnormality. Lytic lesions, diminished trabecular architecture, endosteal scalloping, periosteal thickening, sequestra, and new bone growth may also appear on conventional radiography as the condition worsens (Fig. 8). It may take up to 2-3 weeks for initial signs of osteomyelitis to appear on conventional radiograph, thus in order to diagnose this disease process in the earlier, more acute stages, the clinician must employ more sensitive imaging modalities, such as CT and MRI. Even in the early stages of chronic osteomyelitis, necrotic bone may be difficult to visualize on conventional radiograph. Additionally, sonography may better aid in the detection of complications such as sinus tracts, which is also often difficult to visualize on conventional radiography.[9,18]

Computed Tomography

CT and MRI have complementary roles in the diagnosis of osteomyelitis. CT is more sensitive than conventional radiography, however as would be expected, is worse at delineating soft tissue pathology when compared to MRI. CT excels in the detection of many of the well-known findings that typify chronic osteomyelitis, such as cortical thickening, osteosclerotic changes, and periosteal new bone formation. It is particularly useful and outcompetes MRI in the evaluation of sequestra, involucra, and cloacas (Fig. 9).[18]

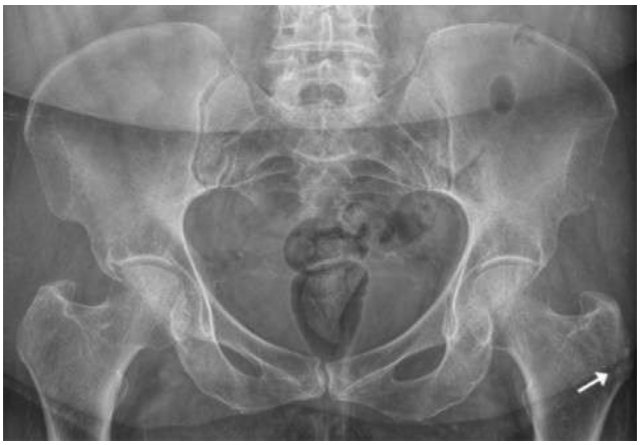


Figure 8 A 71-year-old woman with chronic osteomyelitis. AP radiograph of the pelvis and hips demonstrates osteolysis in the left lateral greater trochanter with a small sequestrum (*arrow*).

MRI

Regarding early detection of osteomyelitis, MRI is the gold standard and may reveal pathology as early as 1-2 days into the disease process. Marrow and soft tissue edema will manifest with low signal intensity on T1-weighted imaging and high signal intensity on fluid-sensitive sequences with diffuse enhancement. Discrete abscesses will have a similar appearance, however enhancement will be peripheral (Fig. 10). Sequestra may display fat signal from contained marrow, however will fail to enhance secondary to devascularization. Sinus tracts will appear as a thin tunnel of enhancing soft tissue extending from the diseased bone to the skin.[9,17,18] On occasion, scintigraphic studies such as bone scans and tagged white blood cell scans may become part of the overall evaluation, however these tests are often less sensitive and specific for the detection of osteomyelitis.[18]

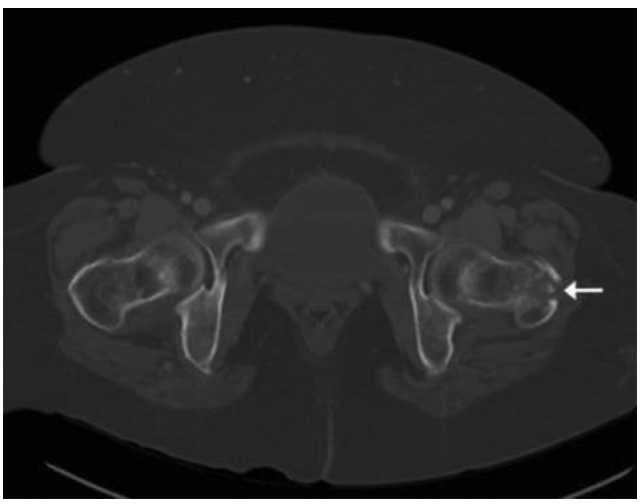


Figure 9 A 71-year-old woman with chronic osteomyelitis. An axial CT image through the level of the femoroacetabular joints demonstrates osteolysis in the left lateral greater trochanter with a small sequestrum (*arrow*).

Myonecrosis

Pathophysiology

Myonecrosis is a broad term used to describe a destructive process of muscle ischemia, ultimately leading to muscle infarction. It encompasses a wide range of pathophysiologies, and may include disruption of vascular supply to skeletal muscle by way of atherosclerotic disease, hypoxia-reperfusion injury, or vasculitis with thrombus. Nonspecific imaging characteristics make myonecrosis vulnerable to misinterpretation for other disease entities such as hematoma, abscess, intramuscular mass, deep venous thrombosis, fasciitis, myositis, or malignancy.[19] Predisposing factors and conditions include trauma, poorly controlled diabetes, sickle-cell anemia, compartment syndrome, rhabdomyolysis, and intraarterial chemotherapy.

Since the overall prevalence of trauma and diabetes is high, these are 2 of the most frequently encountered etiologies for myonecrosis. Calcific myonecrosis, for example, is a specific subtype of myonecrosis most commonly caused by limb trauma, which leads to post-traumatic ischemia and subsequent cystic muscular degeneration.[20,21] Diabetic myonecrosis is a rare complication of advanced diabetes, and usually occurs after more than a decade of the disease with poor glycemic control. The pathophysiology is believed to involve thrombosis of medium and small arterioles.[19]

Clinical Features

Trauma-related calcific myonecrosis, usually manifests as an enlarging and painful dystrophic calcified mass in the lower extremities.[20,21] Diabetic myonecrosis often presents with normal white blood cell count and tender swelling at the site of the injured muscle in these patients, often prompting imaging for further investigation.[19] Rhabdomyolysis is a clinical term used to describe the syndrome that may result from myonecrosis. Muscle death leads to liberation of the intracellular components within the myocytes, which contributes to a destructive biochemical cascade. Characteristic features of rhabdomyolysis include elevated serum creatine kinase, myoglobinuria, and frequently acute renal injury. In severe cases, related electrolyte disturbances can be life-threatening.[22]

Imaging Features

Conventional Radiograph

Given that myonecrosis is inherently a soft tissue process, conventional radiography often has limited diagnostic value in the diagnosis of myonecrosis. It may demonstrate generalized soft tissue swelling at the region of interest.[23] In calcific myonecrosis, conventional radiographs may reveal a fusiform soft tissue mass, with linear or irregular calcifications (Fig. 11).[20] As an aside, since patients often present with a swollen, painful extremity, ultrasound is frequently employed early to evaluate for the possibility of deep venous thrombosis.

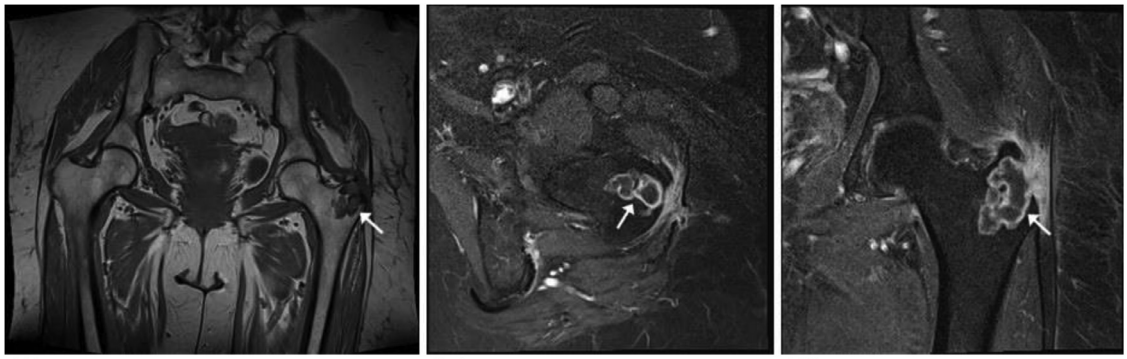


Figure 10 A 71-year-old woman with chronic osteomyelitis and associated sequestrum of the left femoral greater trochanter. Coronal T1 (A), axial contrast-enhanced T1 fat-suppressed (B), and coronal contrast-enhanced T1 fat-suppressed (C) MR images reveal a peripherally enhancing, lobulated signal abnormality in the left femoral greater trochanter (*arrows*) related to chronic osteomyelitis.

CT

Findings on both CT and MRI will vary based on the specific etiology of the patient's myonecrosis. In the case of calcific myonecrosis, CT will demonstrate an expansile calcific soft tissue mass (Fig. 11). Regardless of etiology, however, there is almost always diffuse enlargement of the affected muscles (Figs. 12 and 13). Diabetes and sickle-cell related injury may exhibit random muscle involvement, and classically at least partially involves the anterior compartment of the thigh in approximately 80% of cases, with the calf accounting for the majority of the remaining 20%. The process may be bilateral in more than one-third of cases and may involve multiple compartments. Muscle architecture is often preserved early-on, with increasingly apparent distortion as the process advances.[24]

MRI

MRI will demonstrate diffuse enlargement of the involved muscle, along with subfascial edema and obliteration of the intermuscular fatty septa. T2 weighted images will reveal diffuse increased intramuscular and intermuscular signal intensity, with corresponding diffuse enhancement on contrast enhanced images. Necrotic foci will be demarcated by rim enhancement, likely secondary to surrounding hyperemia (Figs. 12 and 13). The appearance of myonecrosis is similar in the case of compartment syndrome, however will involve the entire compartment of interest. In delineating the precise etiology of a patient's myonecrosis, providers must incorporate the history and clinical presentation along with these imaging findings.[24]

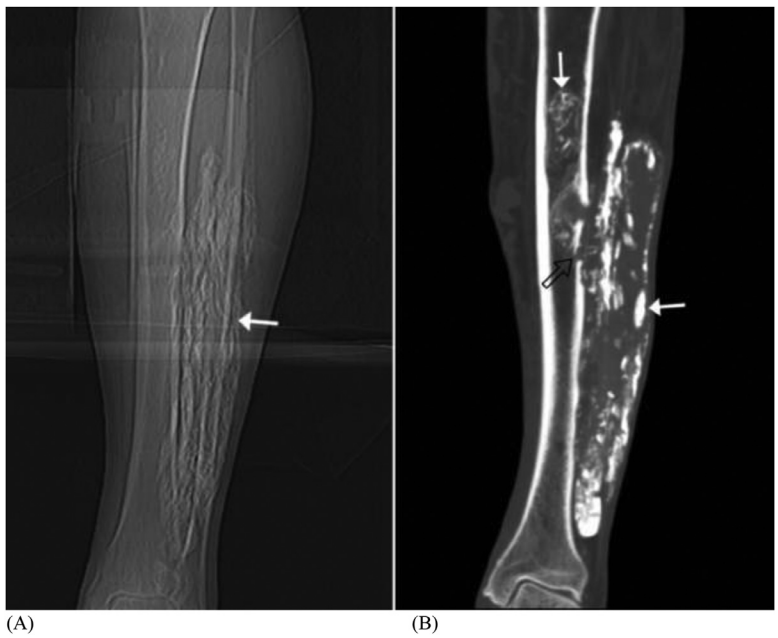


Figure 11 A 81-year-old man with calcific myonecrosis of the left lower extremity. Scout radiograph (A) and coronal CT (B) images demonstrate a large, calcified, fusiform mass with peripheral plaque-like calcifications (*white arrows*) and erosion through the lateral tibial diaphysis with extension into the tibial medullary cavity (*hollow black arrow*) related to calcific myonecrosis.

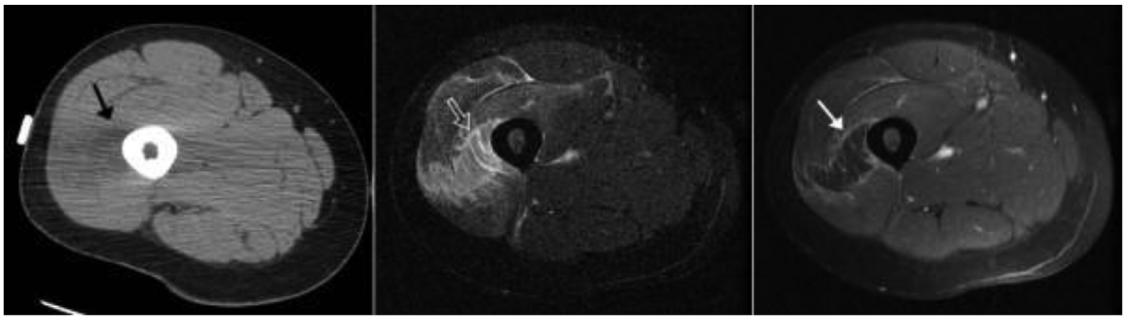


Figure 12 A 27-year-old man with myonecrosis of the right lower extremity. An axial CT image (A) of the right lower extremity demonstrates a poorly-defined area of low attenuation within the proximal thigh musculature (*black arrow*), corresponding to the site of reported pain. Axial T2 fat-suppressed MR image (B) demonstrates edema within the vastus intermedius and lateralis musculature (*hollow arrow*). Axial contrast-enhanced T1 fat-suppressed (C) MR image demonstrates rim enhancement and central nonenhancement (*solid white arrow*).

Necrotizing Fasciitis

Pathophysiology

Necrotizing fasciitis is a rare, rapidly progressive, and often fatal soft-tissue infection of the fascia deep to the skin but superficial to muscle, causing necrosis by way of microvascular occlusion, and with reported mortality rates ranging from 15% to 80%. The disease is most commonly polymicrobial, with infectious agents including both aerobic and anaerobic microorganisms such as *Streptococci, Clostridium, Proteus, Escherichia coli, Bacteroides*, and *Enterobacteriaceae*, among others.[25-28] The infection originates in the superficial fascial planes and rapidly progresses to the deep fascia.[25,26]

Predisposing conditions include diabetes mellitus, peripheral vascular disease, alcoholism, malignancy such as leukemia or lymphoma, immunosuppression such as in HIV, postsurgical status, history of blunt trauma, penetrating injuries, varicella zoster, IV drug abuse, childbirth, burns, and nonsteroidal anti-inflammatory drugs.[25,28]

Death from necrotizing fasciitis is usually related to sepsis, or by way of respiratory, renal, or multisystem organ failure. While necrotizing fasciitis is considered a clinical diagnosis, imaging is frequently performed during the diagnostic evaluation.[25]

Clinical Features

In early disease, the overlying skin is warm, erythematous, and indurated, producing a "wooden skin" appearance. The affected areas are extremely painful, with pain out of proportion to the degree of swelling and erythema.[25,27] Blisters and bullae form, eventually becoming hemorrhagic, with crepitus rarely noted on physical examination. As skin necrosis develops in late disease, the affected areas become painless.[25,28] The external skin lesions are often deceptively less severe than the underlying tissue damage and necrosis.[26]

Diagnostic criteria include the following[26]:

- Histopathologic evidence.
- Intraoperative findings of grayish fasciae, loss of resistance of the skin, which detaches easily, and easy tearing of the deep soft tissue by blunt probe.
- Failure of antibiotic therapy to improve the clinical picture.

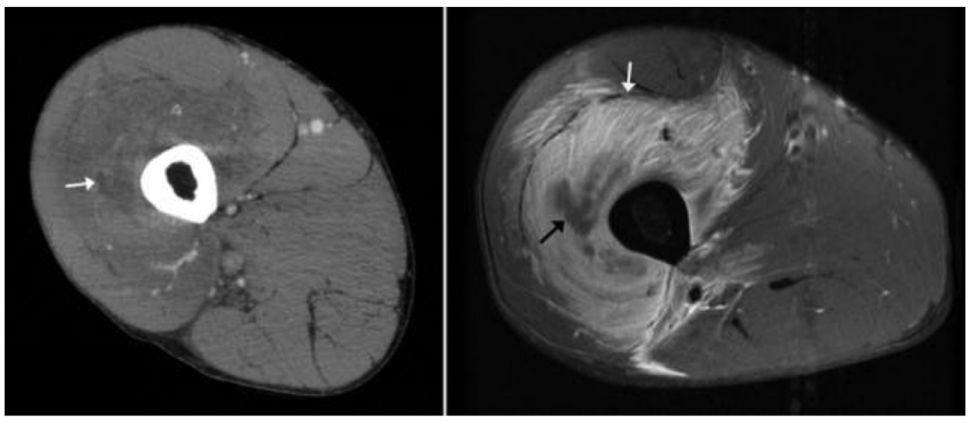


Figure 13 A 57-year-old man with myonecrosis of the right lower extremity. Axial contrast-enhanced CT image (A) of the right lower extremity demonstrates enlargement and a focal region of low attenuation (*arrow*) within the vastus intermedius muscle. Axial contrast-enhanced T1 fat-suppressed (B) MR image of the right lower extremity demonstrates edema of the vastus intermedius muscle (*white arrow*) with a central area of nonenhancement (*black arrow*).

It is critical to note that treatment should not be delayed for the performance of imaging. The diagnosis is clinical, with imaging useful in those who are not toxic, in an effort to map disease extent and exclude alternative etiologies.[25,26,28] If treatment is delayed, patients will invariably progress to sepsis.[28]

Tissue necrosis and blood vessel thrombosis provide barriers to the penetration of antibiotics, reducing their efficacy. As such, surgical treatment is mandatory, to include fasciotomy and debridement of the necrotic tissue.[26,28]

Imaging Features

Conventional Radiograph

Radiographs may be normal in early stages and noncontributory. When features are present, they include soft tissue opacification and thickening, with possible emphysema along the fascial planes (Fig. 14).[25-28]

CT

CT features of necrotizing fasciitis include dermal thickening, increased attenuation of the soft tissue, inflammatory stranding within the fat, with fluid and/or air in the superficial and deep thickened subfascial planes (Fig. 15).[25,27,28] Intravenous contrast assists with evaluation of the deep fascia and for the detection of soft tissue abscess, however is contraindicated in acute renal failure.[26,28] It is important to note that while extremely specific, the absence of soft tissue gas does not exclude necrotizing fasciitis, and is present in only about 55% of cases.[25-28]

MRI

MRI is an excellent modality for evaluating soft tissue infection; however in the context of necrotizing fasciitis, its lengthy acquisition time is prohibitive for routine use.[25]

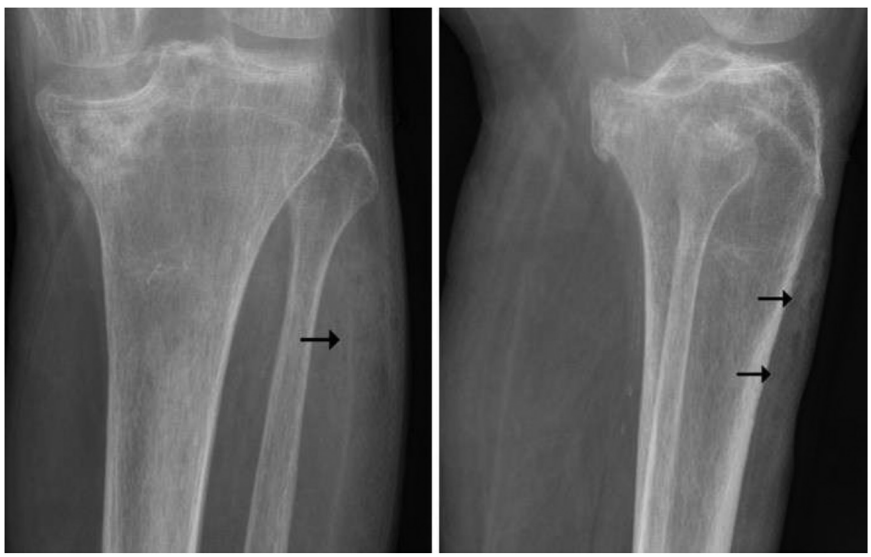
Imaging features include dermal and soft tissue thickening with variable signal on T1 weighted imaging, and increased signal on fat suppressed fluid sensitive sequences (Figs. 16 and 17). Fusiform, T2 bright deep fascial thickening, typically beginning along the superficial fascial planes, is considered a hallmark feature of the disease, but is notably not specific.[25,27,28] Gas is very specific, but less conspicuous on MRI when compared to CT, seen as T1 and T2 hypointense foci.[25,28] Gradient echo sequences are best at detecting gas on MRI, with resultant blooming artifact.[26,27] Intravenous gadolinium may assist with the detection of necrotic tissue and soft tissue abscess, but the presence of renal failure may prevent its application.[25,26,28]

Gangrene

Pathophysiology

Gangrene is a disease process describing the death of body tissues secondary to, ultimately, interrupted blood supply. It is similar in pathogenesis to avascular necrosis or myonecrosis in this regard, however is broader in scope and may simultaneously involve the cellular death of multiple tissue types. This disease process is often divided into "dry" and "wet" gangrene.

Dry gangrene refers to tissue death from interrupted vascular supply in the absence of infection. Often, this form of gangrene is seen in patients with peripheral vascular disease or in acutely ischemic extremities. The lack of oxygen leads to tissue death, and limits the proliferation of most bacteria. Wet gangrene, however, refers to tissue death associated with bacterial invasion, and has a poor prognosis secondary to its propensity to cause sepsis. The pathophysiology of wet gangrene involves interruption of predominantly venous



(A) (B)

Figure 14 A 63-year-old man with necrotizing soft tissue infection of the left lower extremity. Frontal (A) and lateral (B) radiographs of the left lower extremity demonstrate gas within the anterolateral soft tissues (*arrows*).

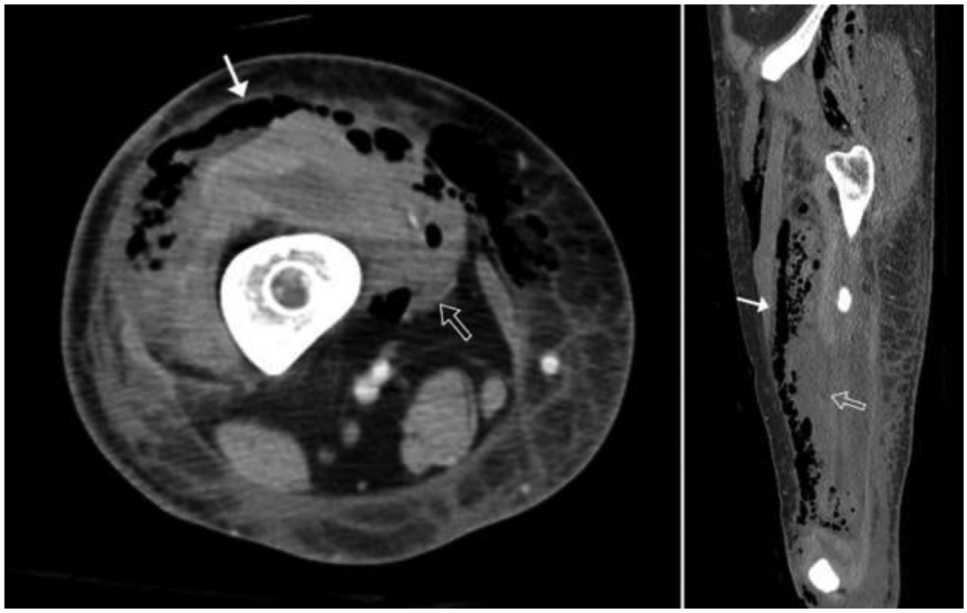


Figure 15 A 63-year-old woman with necrotizing fasciitis of the right lower extremity. Axial (A) and coronal (B) contrast-enhanced CT images demonstrate extensive intramuscular and perifascial emphysema (*white arrows*) with associated edema (*hollow arrows*) throughout the anterior compartment of the thigh.

vasculature, allowing for stagnant blood to potentiate the rapid proliferation of putrefying bacteria.[29,30]

Additionally, gas-forming organisms, most commonly Clostridial species such as *C. perfringens*, may infect the diseased tissue and infiltrate the neighboring healthy tissue with expanding gas, which facilitates the spread of bacterial toxins to previously healthy tissue. Gangrene may occur in many parts of the body, however the extremities are particularly susceptible as they are less perfused than the more central anatomic structures, and even less so in patients with pathologic conditions that further decrease perfusion, such as diabetes or peripheral vascular disease.[29-31]

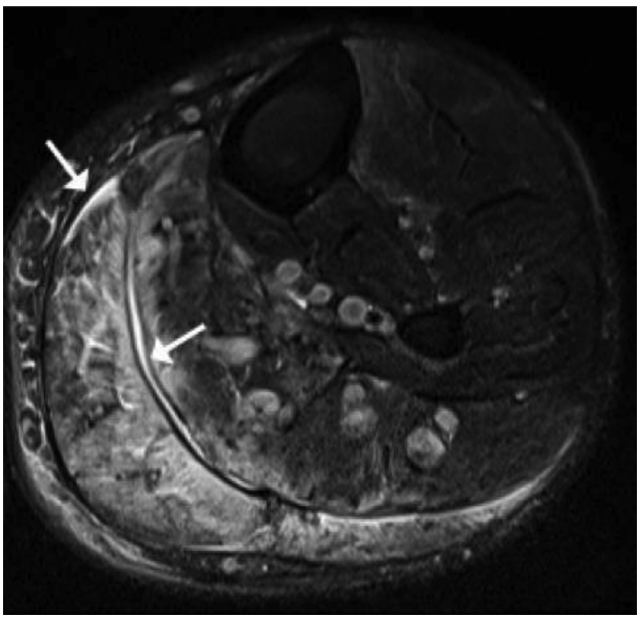


Figure 16 46-year-old man with necrotizing fasciitis of the left lower leg. Axial T2 fat-suppressed MR image demonstrates edema throughout the subcutaneous fat of the posteromedial extremity with concomitant intramuscular and, most notably, fascial edema (*arrows*).

Clinical Features

Presenting features of gangrene affecting the extremities include changes in skin color, which may begin as red discoloration and progress to black as the skin necrosis advances. There may be pain associated with the extremity, however depending on the etiology of the gangrene as well as the extent of necrosis, the patient may also experience numbness in the affected extremity. Given the lack of adequate perfusion, the extremity is also often cool to touch. In the context of wet gangrene, the patient may present with stigmata of infection including high-grade fever and neutrophilia. Crepitus and swelling of the extremity may be noted on exam. As with necrotizing fasciitis, the diagnosis of gangrene is primarily clinical and the role of imaging is complementary, namely in the evaluation of disease extent for the purpose of guiding further management and surgical planning.[29,30]

Imaging Features

Conventional Radiograph

Findings on conventional radiograph may be nonspecific and include osteolysis and overlying soft tissue defects (Fig. 18). In the setting of gas gangrene, subcutaneous emphysema may also be visible, however is not required for this diagnosis. Any of these findings should prompt further investigation with multiplanar imaging to evaluate the full extent of disease.[29,30]

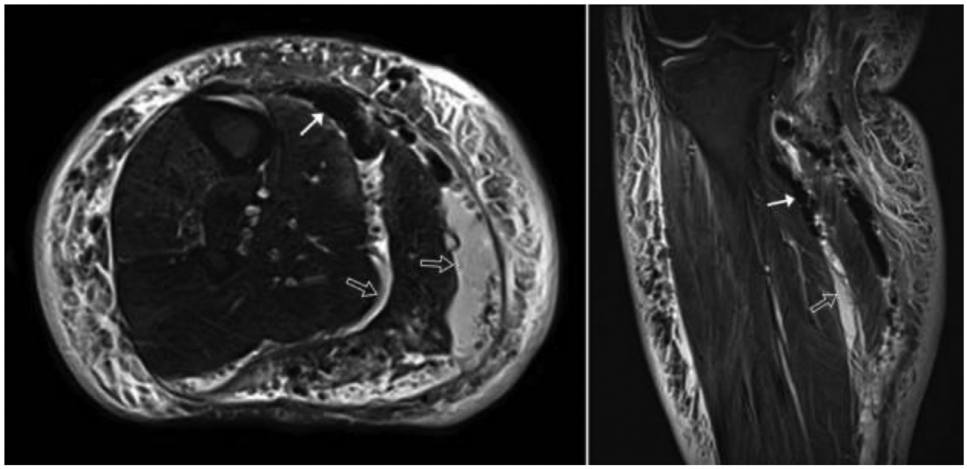


Figure 17 A 44-year-old man with necrotizing fasciitis of the right lower extremity. Axial STIR (A) and coronal STIR (B) MR images demonstrate gas (*solid arrows*) and fluid (*hollow arrows*) tracking superficial and deep to the medial head of the gastrocnemius muscle.

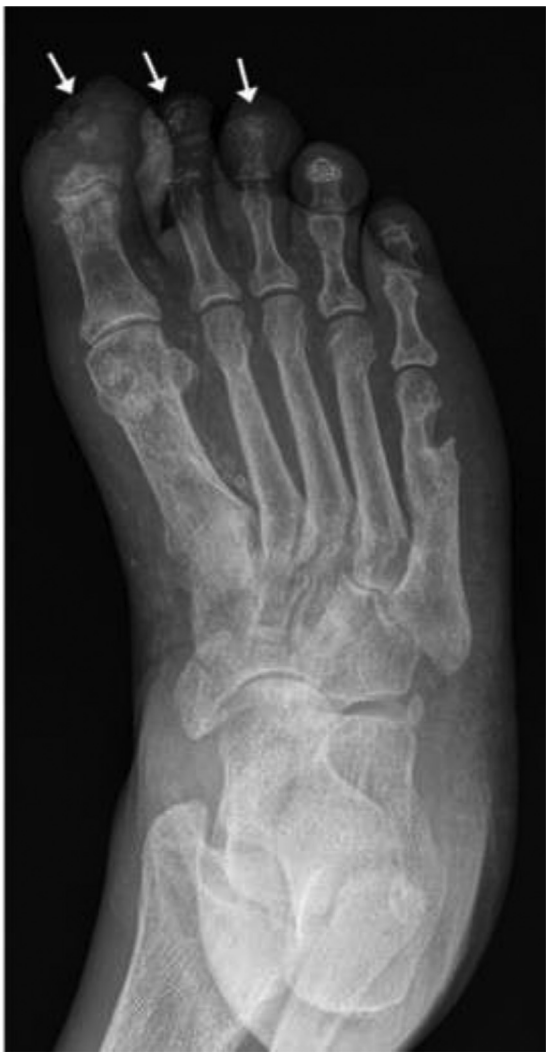


Figure 18 A 62-year-old woman with gangrene of the right toes. An oblique radiograph of the right foot demonstrates diffuse osteolysis of the first through third phalanges (*arrows*).

CT

Soft tissue stranding and thickening of the involved fascial planes will help delineate soft tissue extent on CT, although MRI is superior for this purpose in the setting of gangrene. CT excels in assessing osseous extent, and findings of associated osteonecrosis will be similar to the previously described findings regarding bone infarcts. Additionally, in the context of wet gangrene, gas may be more conspicuous on CT than MRI, however it should be noted that soft tissue gas is not pathognomonic for gas gangrene and alternatively, a patient may have gas gangrene without visible gas.[29,30]

MRI

MRI is useful in the delineation of soft tissue necrosis when evaluating a patient with suspected gangrene and is helpful in the surgical planning process. Multiple tissues will be involved, and often MRI will detect skin ulcerations with enhancing granulation tissue and surrounding cellulitis. Reticulation and enhancement of the subcutaneous adipose tissue is also often seen. As the condition progresses and the skin and underlying soft tissues devitalize, they will fail to enhance (Fig. 19). Inflamed muscle will demonstrate increased T2 signal and once necrosed will display imaging features of myonecrosis, similar to those previously discussed. If the necrotic tissue has liquefied, this will manifest as high signal on fluid-sensitive sequences. Gas is less conspicuous on MR than CT, however may be seen as blooming artifact on gradient-echo sequences.[29-31]

Conclusion

The radiological evaluation of conditions leading to cellular death of the various components of the musculoskeletal system is multipurposed. As discussed, at times the purpose is diagnostic, however may also be complementary to clinical diagnoses in guidance of further management and surgical

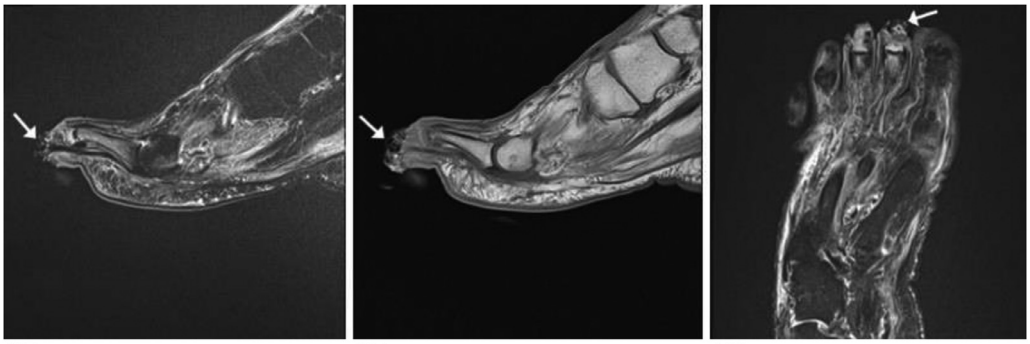


Figure 19 A 62-year-old woman with gangrene of the right second phalanx. Sagittal STIR (A), sagittal T1 (B), and coronal STIR (C) MR images of the right foot demonstrate exposed bone, osteolysis, and signal voids suggestive of gangrene at the distal second phalanx (*arrows*).

planning, where appropriate. In either case, prompt and accurate radiological evaluation may prevent further damage to the involved components of the musculoskeletal system, in hopes of ultimately improving consequent morbidity and mortality.

Acknowledgments

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