Diagnostic Imaging of Diabetic Foot Disorders

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KEYWORDS

• Imaging • Radiography • MRI • CT

KEY POINTS

- In cellulitis, the presence of an abscess or osteomyelitis has to be searched.
- MRI is the gold standard diagnostic tool for osteomyelitis.
- It can be challenging to discriminate between osteomyelitis and bone marrow edema.

INTRODUCTION

Early and accurate diagnosis of diabetic foot pathologies, such as infection, neuroarthropathy, and ischemia, is key in the successful treatment of patients with diabetic feet. Correct usage of various imaging modalities and knowledge of the different imaging findings are essential. Plain radiography, if possible, with full weight-bearing, is the preferred first-line imaging in the diabetic patient. Depending on the clinical suspicion, additional diagnostic imaging, such as computed tomography (CT), magnetic resonance imaging (MRI), and scintigraphy, is indicated. CT provides similar information as plain radiography, but with 3D reconstruction capabilities and with the possibility of contrast enhancement (CE). An iodinated contrast agent is used to enhance the visibility of internal structures as well as pathologies such as an abscess or angiopathy. Allergic reactions to the contrast agent are rare accounting for 0.6% of cases with only 0.04% considered severe.[1] The renal function has to be controlled before application of iodinated contrast agent as—particularly diabetic—patients with oftentimes impaired renal function are at risk for developing contrast-induced nephropathy.

Using MRI, the following 4 pulse sequences are the most often used: T1-weighted (T1w), T2-weighted (T2w), fluid sensitive sequences, for example, short-tau inversion recovery (STIR), and T1w with fat saturation (FS) and CE.

T2w imaging displays fluid as bright and fat as intermediate intense. In the STIR sequence, fluid is very bright; accordingly, it is useful to detect inflammation; however,

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the details are less well depicted on this sequence. T1w imaging displays anatomic features in detail. Fat (including bone marrow) is bright, liquid is dark. T1w imaging with fat suppression has a low signal in fatty and fluid areas and therefore is ideal for postcontrast imaging. Intravenous application of the contrast agent gadolinium will lead to hyperintensity in the area of hyperemia and inflammation. The nephrotoxicity of gadolinium is less than the contrast agent in CT scans, which is an advantage in diabetic patients, which often suffer from chronic renal failure.

Nuclear medicine techniques such as scintigraphy and, more recently, fluorine 18 fluorodeoxyglucose PET have demonstrated their ability to aid in bone marrow evaluation and provide functional information about the presence of osteomyelitis, when used alone or in combination with CT.[2,3] According to a recent meta-analysis, bone scanning has an 81% sensitivity in the detection of osteomyelitis in the diabetic foot but only a 28% specificity.[4] PET/MRI has been reported recently as a viable method for evaluating osteomyelitis in diabetic patients, and preliminary results are promising with a sensitivity of 100%.[5]

SOFT TISSUE PATHOLOGIES: CALLUS, ULCERATION, CELLULITIS, TISSUE ABSCESS

A diagnosis of callus and ulcer is mainly clinical. Plain radiography often does not display direct visualization of the ulcer, but sometimes a defect or swelling of the soft tissue. Callus and most ulcers evolve over an area with high pressure due to a bony prominence. Weight-bearing plain radiography of the foot is useful to locate these areas and find the underlying pathology leading to abnormal pressure distribution. Calcification of vessels may suggest underlying diabetic angiopathy. According to this, radiological imaging is not necessary for the diagnosis of callus or ulceration; however, it can be useful to find the underlying mechanic pathology for soft tissue pathologies and thereby the suitable operative or nonoperative therapy.

Swelling of the foot is a common finding in diabetic patients. There are many possible etiologies, including vascular insufficiency, peripheral neuropathy, and infection. Although cellulitis can be diagnosed clinically, further imaging is indicated if an underlying deep infection such as an abscess or osteomyelitis is suspected. The distinction between cellulitis, abscess, osteomyelitis, and Charcot arthropathy is clinically relevant, as in case of any deep infection, surgical treatment has to be evaluated. Standard radiography is not helpful in the diagnosis of an abscess.

Sonography is an ideal first-line imaging modality for the evaluation of cellulitis and soft tissue abscess. Healthy subcutaneous tissue is hypoechoic with few hyperechoic strands representing connective tissue. Increased thickness, increased echogenicity, and haziness of the subcutaneous tissue are signs of cellulitis. Progressive accumulation of edema in the connective tissue leads to striation and a “cobble-stone” appearance.[6] In ultrasound, fluid collections such as an abscess or a joint effusion can be demonstrated as anechoic or hypoechoic spherical findings with increased through-transmission. In case of an abscess, an echogenic capsule, as well as septa, may be seen. Furthermore, ultrasound can be used for the detection of foreign bodies, with a sensitivity higher than in MRI, especially if wooden.[7]

In the special case of necrotizing fasciitis, ultrasound can also be used to support the diagnosis. The findings are similar to cellulitis but more severe and with a thickened fascial layer and fluid tracking along the deep fascia. In addition, the finding of subcutaneous gas is pathognomonic for necrotizing fasciitis. The reliability of ultrasound in necrotizing fasciitis is high with sensitivity and specificity reaching 88% and 93%, respectively.[8] However, the final diagnosis can only be made intraoperatively and no diagnostic procedure should delay the surgery.

In patients with cellulitis, CT can identify skin edema and subcutaneous fat stranding. Small collections may be found inside the diffusely affected soft tissue layers. An abscess typically shows a necrotic center and a well-defined fibrous capsule, which contains dilated blood vessels, and its postcontrast rim enhancement is pathognomonic.

The gold standard diagnostic modality for soft tissue pathologies is MRI. Callus appears as a focal prominence in the subcutaneous fat with low signal intensity in T1w and low to intermediate signal intensity on T2w sequences. Sometimes a bursa is formed below the callus, seen as fluid on MRI with low signal intensity in T1w and high intensity in T2w imaging. However, the bursa has no adjacent soft tissue reaction and no contrast agent enhancement and can thereby be distinguished from an abscess.[9] Ulcers appear as a soft tissue defect, bright on T2w sequences and with peripheral CE. A potential sinus tract to the bone has to be searched. After contrast administration, "tramtrack" enhancement of the sinus tract can be seen.

On MRI, soft tissue edema and cellulitis are seen as fat reticulation with intermediate signal intensity in T1w and high intensity in T2w images. In contrast to soft tissue edema, cellulitis is enhanced postcontrast. In a phlegmon, the subcutaneous fat shows ill-defined areas with low T1w and intermediate to high T2w and STIR signal intensity and vague post-CE. An abscess presents as a fluid collection area, often with rim enhancement after application of contrast agent. However, other findings such as a hematoma or a tumor can show a similar finding (**Table 1**).[10]

Areas of ischemia such as gangrene are seen in contrast-enhanced MRI as nonenhanced areas.

ADULT OSTEOMYELITIS

Plain radiographs are recommended as first-line imaging when osteomyelitis is suspected. Initial findings include soft tissue swelling and stranding of adjutant fat. The classic radiological triad including osteolysis, periosteal reaction, and bone destruction is generally not evident before a later stage at least 10 to 20 days after onset of symptoms.[11] Osteolysis is seen as small, ill-defined lucencies in the medullary bone and cortex (**Fig. 1**A). Cortical destruction may be evident. In a later stage, sequestration of dead, sclerotic bone can be found. In patients with implanted metal, sometimes a poorly marginated lucency can be found around the implant indicating hardware loosening. The shape of the lucency may help to discriminate between aseptic implant loosening and hardware-associated infection. While in case of infection, the bone lysis is round; in aseptic loosening, the lucency is conically shaped and starts at the point of most movement.[11] Sensitivity and specificity of plain radiography for osteomyelitis are 60% and 80%, respectively.[9] In serial radiographs, progressive change can support the diagnosis in uncertain cases. Gas might be seen in soft tissues, especially in cases with a sinus tract and adjacent osseous destruction (see **Fig 1**B).

If the clinical examination in combination with plain radiographs does not lead to a conclusion, MRI is the gold standard in patients with suspected osteomyelitis. Osteomyelitis is bright on STIR and T2w and confluent hypointense in T1w images. In contrast, bone marrow edema is also bright on T2w images but the T1w image has an intermediate to decreased reticulated hazy intensity (**Figs. 2** and **3**). Bone marrow enhancement after the administration of contrast agent is in favor of osteomyelitis.[12] Osteomyelitis in patients with diabetic foot syndrome is often in proximity to the entry point, such as an ulcer. However, bone marrow edema without osteomyelitis can also be found as a reaction to a soft tissue infection or an ulcer. Other signs of osteomyelitis include cortical interruption and enhancement at the margins of the periosteum

Table 1
Key points of diagnostic imaging of diabetic foot disorders

Pathology	Radiography	CT	MRI	Sonography	Overall
Cellulitis	Soft tissue swelling, adjacent fat stranding, rarely soft tissue gas	Soft tissue swelling, adjacent fat obliteration and stranding, fluid collection, rarely soft tissue gas	T1w: low to intermediate SI T2w: high SI T1w with CE: thick rim enhancement	Increased echogenicity and haziness of subcutaneous tissue, possible fluid collection	Search for an abscess and adjacent osteomyelitis. Exclude tumor.
Acute osteomyelitis	Early: cortex indistinctness Advanced: osseous destruction, dense periosteal reaction Late: sequester and Brodie abscess	Osteolytic destruction, reactive bone formation, +/− soft tissue abscess Late: sequester and Brodie abscess	T1w: confluent low SI T2w + STIR: high SI CE: within bone and around abscess. +/− soft tissue abscess or gas	Not useful	Difficult differentiating between osteomyelitis and Charcot foot. Bone destruction faster in osteomyelitis than in tumor.
Bone marrow edema	Bone density is normal, unless decreased by diabetic or elderly patients.	Not useful	T1w: hazy low SI T2w + STIR: high SI CE: +/− thin rim in Charcot foot.	Not useful	Charcot foot might have fluid collections with enhancing rim in absence of osteomyelitis.
Septic arthritis	Early: normal Advanced: joint effusion and narrowing, periarticular osteoporosis, marginal erosions, sclerotic reaction.	Not modality of choice. Soft tissue swelling, joint effusion, articular narrowing, bone and cartilage erosions.	T1w: low SI T2w + STIR: high SI CE: subchondral adjacent to joint, thickened synovium, joint effusion, articular narrowing.	Highly sensitive for joint effusion, but not specific for septic effusion. Can guide aspiration.	With clinical suspicion, aspiration required. MRI examination must include postcontrast sequences.

Abbreviations: CE, contrast enhancement; CT, computed tomography; SI, signal intensity; T1w, T1 weighted; T2w, T2 weighted.

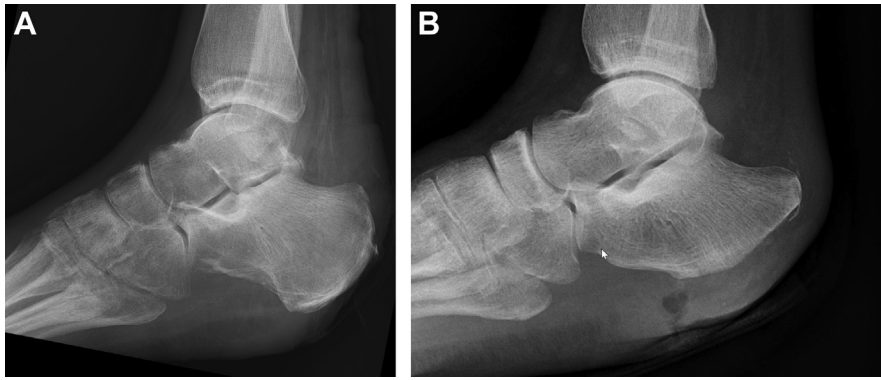


Fig. 1. (*A*) Lateral radiograph of the midfoot and hindfoot of a diabetic patient showing osteopenic changes. (*B*) Lateral radiograph of the midfoot and hindfoot (different patient than *A*) after partial resection of the calcaneus shows soft tissue swelling and gas inclusion in the soft tissue suggestive of pressure ulcer in the diabetic foot. The soft tissue defect dorsal of the calcaneus is suggestive of pressure ulcer. Dorsal of the ankle and around the plantar fascia, cortical lytic lesions due to osteomyelitis, and soft tissue swelling due to inflammation can be seen.

indicating a periostitis. The sensitivity of MRI for osteomyelitis ranges between 77% and 100%, and specificity between 79% and 100%.[13,14] The application of gadolinium increases the accuracy of osteomyelitis from 78% to 89%.[15]

CT is not recommended as first-line modality to evaluate osteomyelitis; however, it can be helpful to evaluate other pathologies leading to soft tissue swelling in the foot, such as neuroarthropathy or an abscess. In late-stage osteomyelitis, periostitis, rarefication of bone or bone destruction can evolve and be seen on the CT scan. In chronic osteomyelitis, bony sequestrum can form, representing a central area with necrotic

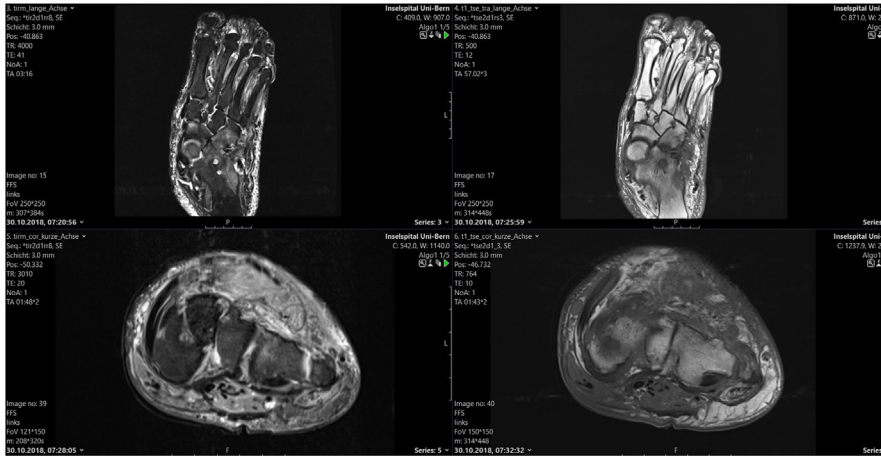


Fig. 2. MRI of a Charcot foot. (*A*) Upper left: long axis turbo inversion recovery magnitude (TIRM); (*B*) upper right T1 TSE long axis; (*C*) lower left: TIRM coronal short axis; and (*D*) lower right: T1 TSE short axis. Hyperintensities in TIRM and not confluent, stranding hypointensities in T1w in the midfoot are more suggestive of osseous reaction to Charcot joints than to an infection.

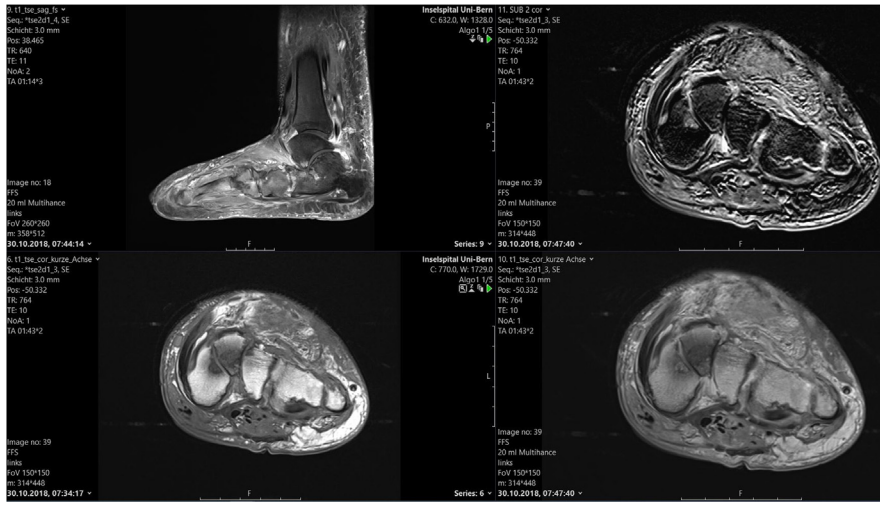


Fig. 3. The same patient in Fig. 3 with a Charcot foot. (*A*) Upper left: T1w TSE fat suppression with CE; (*B*) upper right: CE subtraction short axis; (*C*) lower left: T1w TSE without CE coronal short axis; and (*D*) lower right: T1w TSE with CE coronal short axis. The diffuse hypointensities in the subcutis in T1w with CE in subtraction series and the slight post-CE of the midfoot and metatarsals are suggestive of inflammation and cellulitis around Charcot joints.

bone and granulation tissue around it. On CT, sequestra are seen as a dense bone spicule in the medullary cavity surrounded by soft tissue density.[16] If MRI is available, ultrasound is not recommended as a diagnostic tool for osteomyelitis. However, sometimes a periosteal abscess can be seen. With the progress of MRI techniques, the role of scintigraphy in the diagnosis of osteomyelitis is limited. Technetium (99mTc) 3-phase bone scan can differentiate between cellulitis and osteomyelitis.[11] In cellulitis, tracer activity increases in the early images but is normal in delayed images, 2 to 4 hours after application. Noninflammatory bone conditions such as ischemic necrosis demonstrate normal activity in early images and increased activity in delayed images. Osteomyelitis results in high activity in both, early and delayed images. It has to be kept in mind that the diabetic foot can have vascular problems; decreased blood flow will lead to false-negative results and, on the other hand, pathologies with hyperemia will lead to false-positive results.

SEPTIC ARTHRITIS

One-third of patients with pedal osteomyelitis have adjacent septic arthritis.

On plain radiography as well as on a CT scan, septic arthritis may be seen as joint effusion, articular narrowing, and, in a chronic stage, joint destruction. As discussed earlier, the presence of an additional abscess can be evaluated using CT.

On MRI, the findings are similar with also joint effusion and articular narrowing due to cartilage destruction. Furthermore, the synovia is usually thickened with intense CE. Sometimes a sinus tract can be found. Periarticular osteopenia due to hyperemia and direct communication of joint fluid with an adjacent sinus tract may be present. The adjacent soft tissues may show perisynovial edema, in addition to subchondral marrow with a thin rim of reactive marrow edema and marginal erosions. The

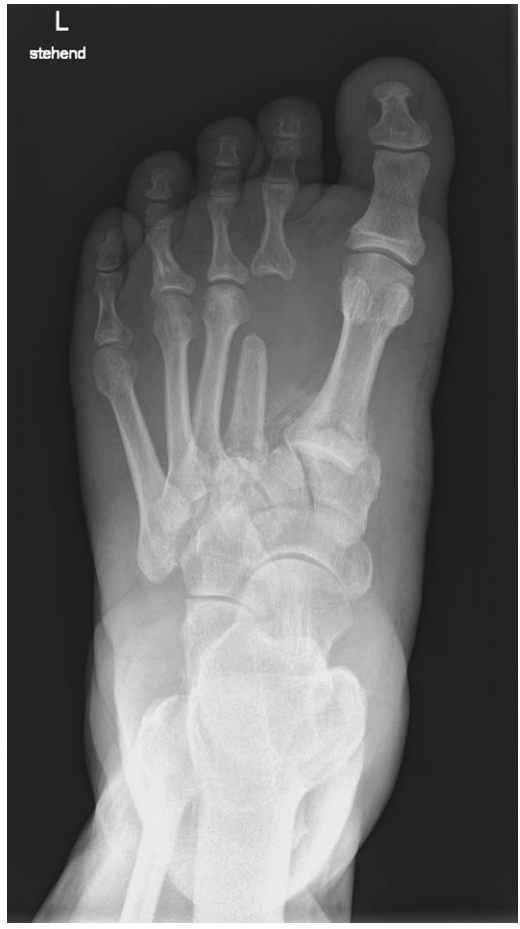


Fig. 4. AP radiograph in a diabetic Charcot foot with prior amputation of the distal metatarsal II. 5Ds findings on the midfoot: normal bone density, joint distension, bony debris, joint disorganization, and dislocation.

difference between reactive edema and osteomyelitis has to be evaluated carefully. As described earlier, both are bright in STIR and T2w images, but while osteomyelitis is confluent and intense T1w hypointense, bone marrow edema shows an intermediate, hazy and reticulated intensity in T1w images; however, there is significant overlap. Furthermore, a proximal extension beyond the subchondral bone also is a sign of osteomyelitis. Joint aspiration is required to confirm the diagnosis.

CHARCOT ARTHROPATHY

Charcot arthropathy refers to a progressive and destructive disorder affecting the joints, bones, and soft tissue in diabetic feet. The pathology is discussed in detail in the according article. In imaging, the diagnosis of Charcot arthropathy includes 5Ds: bone density, bony debris, joint distension, joint disorganization, and dislocation (**Figs. 4** and **5**). The prevalence of this arthropathy is not distributed equally in all joints but is found in the following order: Lisfranc joint > talonavicular joint > Chopart

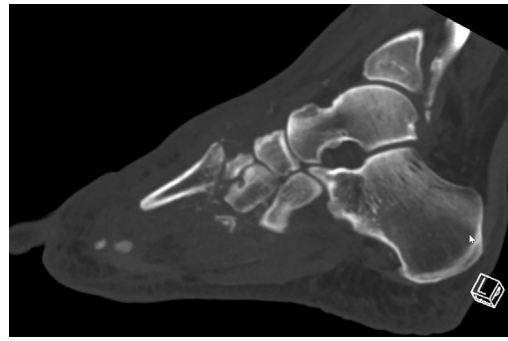


Fig. 5. Sagittal reconstruction of noncontrast enhancement CT in a diabetic Charcot foot with prior amputation of the distal metatarsal II, the same patient as Fig. 4. 5Ds findings on the midfoot: normal bone density, joint distension, bony debris, joint disorganization, and dislocation, there is diffuse swelling of soft tissue around midfoot.

joint > intercuneiform and naviculocuneiform joints. Conventional radiographs are important for staging and monitoring Charcot arthropathy. Eichenholtz classification[17,18] is a historical, widely used system based on radiological findings. In stage 1, the developmental stage, the following findings appear: focal bone demineralization and fragmentation of subchondral bone, leading to periarticular debris formation or fractures and finally joint subluxation and dislocation. In stage 2, the coalescence stage, the debris is absorbed, new periosteal bone forms, and large fragments fuse. Stage 3, the remodeling stage, displays the final stage with remodeled bone, new bone formation, and possibly gross residual deformity. Standard radiography is important for monitoring progression, but it cannot serve to rule out Charcot arthropathy. Shibata and colleagues[18] proposed a stage 0, the inflammation stage, before stage 1 according to Eichenholtz. In stage 0, clinical signs such as erythema and changes in MRI are present but there are no changes in standard radiography evident yet. Correct diagnosis and treatment in this stage are critical to prevent further progression and final foot deformity.[19] Stage 0 on MRI is seen as subchondral edema with or without microfracture, leading to intraarticular debris and subchondral cysts. Also, soft tissue edema, fluid collections, and effusion may be present. Bone marrow will show post-CE. The bone marrow edema appears as hypointensity to intermediate hazy intensity in T1w images and hyperintensity in T2w and STIR images. This mimics the changes seen in osteomyelitis, the most important differential diagnosis to Charcot arthropathy. Findings, which help to distinguish between the 2 diagnoses are the following: Charcot arthropathy favors the midfoot and is often periarticular in multiple joints involved, whereas osteomyelitis is characterized by focal involvement of weight-bearing surfaces including the toes, metatarsal heads, and calcaneus and is often in proximity to an ulcer. Charcot arthropathy leads to cysts and fragmentation; osteomyelitis is associated with cortical lesions. T1w images in osteomyelitis show confluent and prominent hypointensity. CT scan is not the first choice for the diagnosis of Charcot arthropathy, but it can be used to show bone changes including fragmentation, bone remodeling new bone formation in more detail than plain radiographs. Subsequently, it is useful for preoperative planning.

MUSCLE DISORDERS IN DIABETIC FEET

Diabetic individuals can develop a variety of muscular problems, including diabetic muscle ischemia (DMI), viral and inflammatory myositis, and muscle denervation.

Although muscle edema is a frequent imaging finding in all of these disorders, the clinical symptoms, anatomic distribution, and imaging findings associated with each vary.

Diabetic Muscle Infarction

DMI, also known as diabetic myonecrosis or diabetic muscle infarction, is a type of end-organ complication that arises in people with long-term, poorly managed diabetes and is associated with nephropathy and neuropathy.[20] The clinical onset of DMI is abrupt, with significant thigh or calf pain and swelling that develops over days or weeks,[21] however, in absence of leukocytosis or fever. A palpable lump can also be present. The cause of DMI is unknown, but microangiopathy has been suggested as a possible cause.[22] Muscle fiber necrosis and edema are evident on pathologic examination, along with fibrinous blockage of arterioles and capillaries. The preferred modality for evaluating patients suspected of having DMI is MRI. In acute and subacute phases: in STIR and T2w hyperintensity with fascicular enlargement. Muscle enhancement is common, with hypoenhancement or nonenhancement in the core regions. In chronic phase: atrophic-appearing fascicles with intraepineurial fatty changes. Muscle infarction shows hyperintense muscle swelling on MR with adjacent soft tissue reaction. The thigh is the most common location for myopathy ($\geq$80%); the calf is the second most common site. The symptoms might be unilateral or bilateral, and they usually manifest themselves in noncontiguous muscles in the thighs and calves.[21]

Infectious and Inflammatory Myositis

Diabetic patients are susceptible to infectious pyomyositis, a disease caused by the hematogenous spread of bacteria to muscle, due to underlying immunologic failure.[23] When DMI is suspected in a patient, this entity is an important differential diagnostic consideration. Although the imaging appearances of the 2 entities may be similar, the presence of smooth-walled intramuscular abscesses with rim-like enhancement favors the diagnosis of pyomyositis over DMI. Areas of muscular ischemia or necrosis, on the other hand, tend to appear heterogeneous in DMI, with linear enhancement streaks crossing central nonenhancing areas surrounded by widespread regions of enhancing muscle.[24] Fever, leukocytosis with a left shift, increased inflammatory markers, and bacteremia are clinical characteristics that favor the diagnosis of infectious pyomyositis over DMI. It is necessary to distinguish between DMI and pyomyositis because the latter requires antibiotics and abscess drainage. Findings of bilateral symmetric edema in the proximal muscles, especially those in the pelvis and thighs, on MRI can assist diagnose inflammatory myopathy and determine its severity.[25] Muscle biopsy based solely on clinical markers has a 25% false-negative rate. By making it easier to select damaged muscles for sampling, MRI improves diagnostic yield. Inflammatory myositis is frequently accompanied by skin lesions. Clinical history, physical examination, muscle enzyme tests, and muscle biopsy with immunostaining are used to make the diagnosis.

Muscle Denervation

Muscle denervation has a variety of causes, one of the most frequent is due to diabetic peripheral neuropathy. T2w images show signal hyperintensity in the afflicted muscles and is a marker of subacute muscular denervation, although T1w images show normal signal intensity and architecture.[26] Muscles with chronic denervation exhibit decreased mass and fatty infiltration, which is best seen on T1w imaging. In diabetic patients, subacute or chronic denervation appears early and conspicuously, usually affecting the foot's intrinsic musculature. Denervation induced by diabetic peripheral

neuropathy can be distinguished from that caused by DMI by the presence of muscles within a peripheral nerve distribution, the absence of concomitant fascial edema, and the presence of peripheral neuropathy at physical examination.

CALCANEUS INSUFFICIENCY FRACTURE

A neuropathic avulsion fracture of the tuberosity in a patient with long-term diabetes mellitus is significant sequelae.[27] The fracture occurs in these patients without a history of major trauma or overuse activity. The primary fracture line runs parallel to the apophyseal scar, and the fracture usually affects the superior cortex, although not always. In addition, the fracture tends to spread posteriorly, with a horizontal component directly distal to the Achilles tendon insertion..[28] Distraction and fragmentation are common findings when imaging the fracture sequentially. Neuropathic fractures are important because they have a much higher rate of infection, nonunion, malunion, and fixation failure than nonneuropathic insufficiency fractures, and they take much longer to heal. The MRI shows low signal fracture line with displacement of posterior tuberosity fragment in T1w images and on fluid-sensitive sequences, for example, TIRM, a high signal fracture line with surrounding bone edema.

TARSAL/METATARSAL STRESS FRACTURE

Foot overuse injuries are an issue in diabetic patients with inadequate pain sensitivity due to diabetic polyneuropathy; accordingly, they may not notice the overuse. An early stress injury may proceed to a complete fracture if the mechanical load is maintained and there is no protective sensation. Early detection and treatment of stress fractures, such as unloading and immobilization, can reduce the risk of development to stage 1

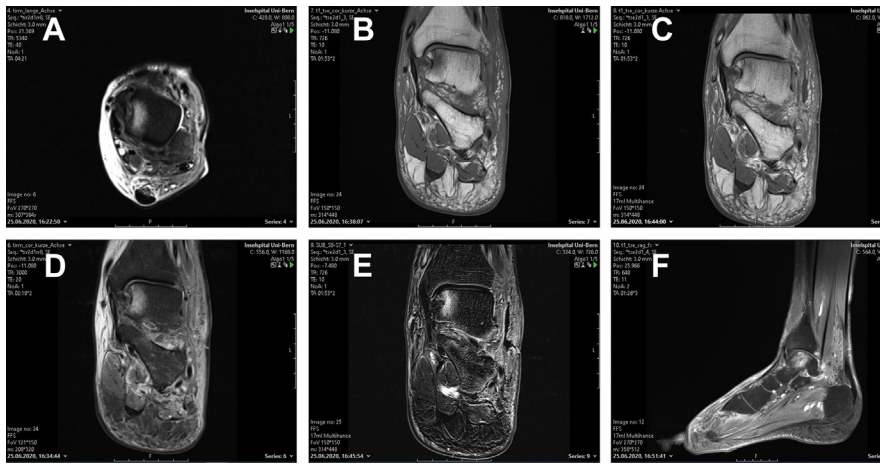
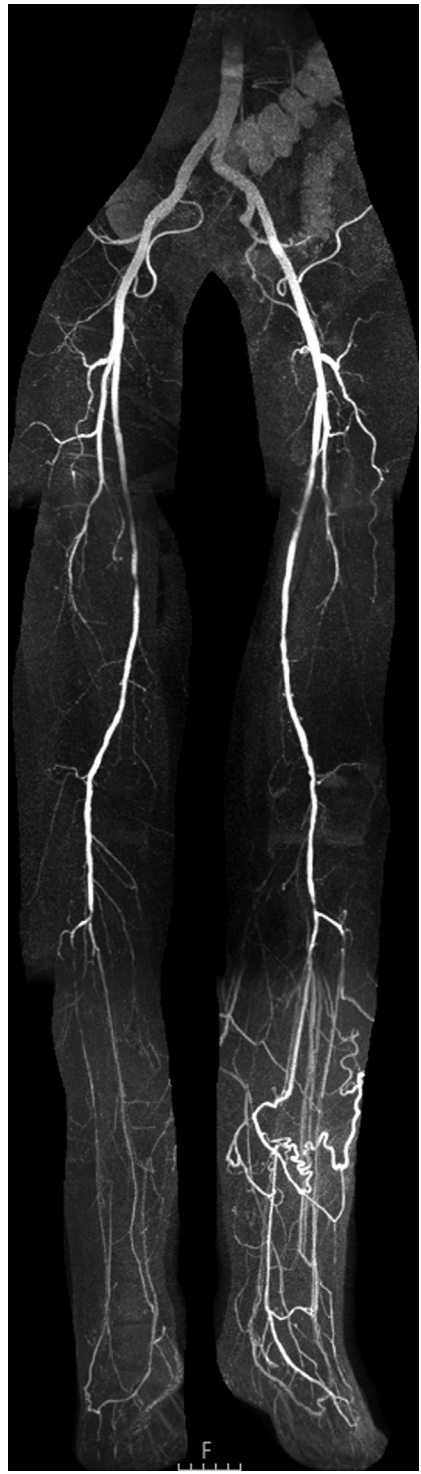


Fig. 6. MRI of diabetic foot with pressure ulcer. (*A*) Upper left: axial STIR; hyperintensity on medial talus indicates edema. (*B*) Upper middle: T1w TSE coronal without contrast-enhanced (CE); shows confluent T1w hypointensity, suggestive of osteomyelitis. (*C*) Upper right: T1w TSE coronal with CE; shows CE in medial of talus, in addition, there is soft tissue swelling with CE on lateral of ankle. (*D*) Lower left: coronal STIR; hyperintensity on medial talus indicates edema. (*E*) Lower middle: subtraction coronal with CE; shows CE in medial of talus, in addition, there is soft tissue swelling with CE on lateral of ankle including soft tissue defect on pressure ulcer. (*F*) Lower right: T1 TSE FS sagittal with CE. CE in medial of talus.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Charcot arthropathy , which causes bone breakdown or complete fracture, as well as irreversible foot deformities and amputation. The most common imaging modality for identifying stress fractures is radiography; however, early bone stress injuries may not be visible. The most sensitive method for identifying stress injuries is MRI. MRI is particularly sensitive in detecting bone bruising, bone marrow edema, and microfractures associated with chronic stress responses. MRI offers useful information about the surrounding soft tissues, too. Bone marrow edema is a nonspecific characteristic that can occur in osteomyelitis, tumors, and bone bruises and is an early marker of stress-related bone damage. In individuals with neuropathy, foot edema should be checked and stress fracture should be considered as differential diagnosis.

AMYLOID AND CRYSTAL DEPOSITION

In diabetic, end-stage renal disease patients, renal osteodystrophy secondary to diabetes mellitus is common. Renal osteodystrophy may show altered bone density, resorptive patterns on all bone, mainly in hands, cranium, and distal clavicles. They show nodular soft tissue densities. Periarticular amyloid, sodium urate, and hydroxyapatite depositions are common in patients on dialysis.

REVIEWING THE IMAGING MODALITIES

Radiography

Plain, full weight-bearing radiography is the preferred first-line imaging in the diabetic patient to assess the alignment of the foot, bony prominences as cause of ulcers, and potential fractures and joint dislocations. It is widely acknowledged that radiography is insensitive to the early stages of osteomyelitis.[1,2] Bone infection can occur up to 4 weeks before radiological changes, though most changes occur within a couple of weeks. Serial radiographs can be very convincing if they show progressive bone resorption, cortical destruction, and periosteal elevation. There have been no formal evaluations of the role of serial radiographs in the diagnosis of osteomyelitis that we are aware of. Furthermore, other clinical conditions common in diabetic patients, such as gout and Charcot osteoarthropathy, may complicate radiographic interpretation.

CT Scan

Although CT has a limited function in the imaging of diabetes-related foot problems, it does have some advantages over radiography, such as the ability to provide pictures with high tissue contrast. Furthermore, it is more sensitive and specific for detecting cortical erosions, tiny sequestra, soft tissue gas, calcifications, and foreign bodies when compared with radiography. The presence of ionizing radiation, as well as insufficient differentiation of healthy and sick tissues, are the fundamental limitations of CT.

MRI

MRI is currently recognized as an effective modality for assessing soft tissue and bone marrow changes associated with diabetic foot.[12,13] MRI has a high sensitivity

Fig. 7. MR angiography in a patient with chronic pressure ulcer along with calcaneal osteomyelitis due to diabetic foot syndrome with peripheral artery disease on the right lower leg and concomitant chronic venous insufficiency on the left lower leg.

and specificity (90%–100% and 40%–100%, respectively) for detecting bone marrow edema as an early finding of neuroarthropathy[14] (Fig 6). The main advantages of MRI for detecting and delineating the extent of an infection are its high soft tissue contrast and multiplanar imaging capabilities. MRI can also help to distinguish osteomyelitis from neuroarthropathy and reactive bone marrow edema, as well as sterile joint effusion from septic arthritis, all of which require entirely different treatment.

Angiography

Peripheral arterial disease is common in patients with diabetic foot syndrome with a prevalence of 50% in patients with foot ulcers. It is a known risk factor for inferior outcome.[29,30] Subsequently, the adequate diagnosis and therapy if necessary are important for optimal patient care. Angiography is indicated in patients with diabetic foot syndrome in the case of suspected or known peripheral arterial disease, with non-healing ulcers and preoperative for optimization of the postoperative wound healing.

Conventional peripheral angiography is performed by injection of an iodinated contrast agent over the femoral artery, followed by fluoroscopic assessment of the distribution of the contrast agent in the arteries. A stenosis can be diagnosed and treated accordingly. MR angiography is also helpful in diagnosis and follow-up of the patients (Fig 7).

Nuclear Medicine

In early phase of osteomyelitis, the sensitivity of a 3 (or 4) phase bisphosphonate-linked technetium bone scan is greater than that of radiography. However, specificity[1,6,7] (averaging 50%) is poor because almost any type of bone disorder (including neuroarthropathy and healing osteomyelitis) can cause increased isotope uptake on a bone scan. As a result, some authorities have concluded that positive technetium bone scans do not significantly increase the likelihood of disease, while negative ones do not significantly decrease it, and that this modality should be used sparingly.[8] Other radionuclide imaging agents, such as scans using white blood cells (labeled autologous leukocytes), labeled immunoglobulin, or other infection-specific radiopharmaceuticals, are more specific than technetium bone scans.[9,10] They can help differentiate osteomyelitis from soft tissue infection or Charcot-type changes but their sensitivity is limited.[11] They lack spatial resolution, are expensive, and technically demanding, and should be regarded as special-purpose problem-solving tools rather than first- or second-line modalities.

CLINICS CARE POINTS

- Start with weight-bearing radiography of the foot.
- In case of a soft tissue infection such as cellulitis, search for an abscess.
- If the clinical examination in combination with plain radiography does not lead to a conclusion regarding possible osteomyelitis, the next imaging modality recommended is MRI if available.
- To differentiate between osteomyelitis and aseptic bone marrow edema, assess T1w images on MRI: osteomyelitis will appear as confluent low signal intensity, bone marrow edema as reticulated, hazy low to intermediate signal intensity.

DISCLOSURE

The authors have nothing to disclose.

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