

A Pictorial Review of Diabetic foot Manifestations

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SUMMARY

The diabetic foot with its many associated complications and presentations can provide a challenge in diagnosis and subsequent treatment. MRI, being increasingly available commonly, is now the main investigative modality. In particular, it is helpful in differentiating between neuroarthropathy and osteomyelitis and in cases where the latter is superimposed on the former. By being well versed in the interpretation of the images, the radiologist can make crucial contribution to the care and management of these patients.

INTRODUCTION

Diabetes is a burgeoning condition worldwide, in part due to increasing sedentary lifestyles and changing dietary trends. The worldwide prevalence of DM is currently estimated at 5.1% and is predicted to reach 7.7% by 2030¹. It has a constellation of presentations, with foot complications being amongst the forefront cause of increased morbidity and occasionally mortality².

Unsurprisingly, the diabetic foot can present in a myriad manner of ways, from structural abnormalities, to a wide range of infective manifestations (superficial or deep seated). These can indeed provide a significant diagnostic dilemma in the management of such patients. Imaging thus plays a crucial role in the differentiation of various pathologies. Radiographs are the preferred initial form of imaging/screening, being readily available and relatively inexpensive. These also provide excellent resolution and visualization of osseous structures, joint spaces, fractures, loose bodies, osteophytes and enthesophytes. However, especially in the scenario of early infection or neuroarthropathy, the detection rate and accuracy is at best only 50-60%³, due to inability to adequately demonstrate the soft tissues. In addition, overlapping features of advanced Charcot's foot and osteomyelitis prevent confident exclusion of one condition from the other on radiographs. Ultrasound is an alternative widely available and noninvasive imaging modality, although its role in the evaluation of diabetes-related foot complications (in particular of the osseous structures) is limited⁴. It is mostly used in the detection of soft tissue pathologies, such as infectious/inflammatory soft tissue changes and localization of foreign bodies as well as identification of tenosynovitis. Radionuclide bone studies, if utilized, are able to demonstrate increased uptake in inflammatory, neuropathic and degenerative diseases, but finely differentiating between these conditions is difficult.

The current gold standard for differentiating the various possible conditions afflicting the diabetic foot is thus MRI.

MRI can not only effectively identify conditions requiring surgical intervention from those that can be conservatively managed; by providing superb anatomical detail, it is also useful for preoperative mapping of the extent of infection and thus helping to limit the area of resection^{5,6}. It is superb in looking at effusions, muscle edema, bone marrow edema, tendon and ligament injuries, cysts or abscesses, damage to cartilage and osteonecrosis. In general, as foot infections in persons with diabetes become more severe and take longer to cure than do equivalent infections in persons without diabetes, early introduction of therapy is paramount.

The aim of our pictorial review is to demonstrate the varying MRI characteristics of a diabetic foot, its common complications, and unique associated afflictions.

MRI TECHNIQUES

The diabetic foot should be adequately imaged in the axial, sagittal, and coronal planes, whilst avoiding large fields of view, so that adequate detail and visualization is obtained. Each plane confers individual benefits. For example, sagittal views are useful for evaluation of midfoot involvement, the plantar surface and the posterior calcaneus; axial or coronal planes are best suited for depicting medial or lateral ulcerations in the hindfoot; and a view perpendicular to the toes is adequate for imaging the metatarsophalangeal and interphalangeal joints, any ulcer and its relationship with the adjacent osseous structures.

Every study should be tailored accordingly to each patient; any pre-existing bandages should, if possible, be unwrapped and the foot examined prior to imaging. Markers are placed over shallow ulcers that may be difficult to appreciate on images as well as suspected sinus tracts. Since osteomyelitis occurs next to ulcers, these areas should be clearly identified and imaged. In cases of alignment deformities, the technologist should orient the slices to optimize imaging of the area of concern.

T1 weighted sequences are utilised as these are anatomically detailed with high resolution, and are sensitive for bone marrow changes; it has the highest specificity in the detection of osteomyelitis. These are also important in identifying blood products, as well as confirmation of the presence of gas locules or foreign bodies.

T2 weighted sequences are excellent for depicting bony and soft tissue edema as well as inflammatory changes or collections. However, when fast spin echo sequences are employed, due the bright signal of edema blending with the bright signal of the fatty bone marrow, diagnostic errors may

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occur. T2 with fat saturation avoids this problem, though maximum care must thus be made for placement of the foot in the centre of the magnet, to obtain homogeneous fat suppression.

Short inversion time inversion recovery (STIR) is very sensitive to bone marrow edema changes, and uniform/better fat saturation is obtained, due to the curvature of the foot.

Intravenous gadolinium is generally not needed on a routine basis. It however improves sensitivity for devitalized regions, abscesses, sinus tracts and joint or tendon involvement, greatly aiding preoperative planning when surgery is required, as it allows differentiation of viable from non-viable bone or soft tissue

OSSEOUS COMPLICATIONS

1. NEUROARTHROPATHY

It has been estimated that less than 1% of persons with diabetes will develop Charcot neuroarthropathic joint disease⁷. Typically, due to a combination of loss of deep sensation, proprioception and peripheral vascular disease, exacerbated by chronic, repetitive trauma to the joints and supporting ligaments of the foot, results in poor healing, joint instability, malalignment, erosion of chondral surfaces and eventual fracture, disorganization and increased new bone formation. This usually involves the mid/hindfoot; example the metatarso-phalangeal joints, the Lisfranc articulation, the talonavicular joints and of the medial column (formed by navicular, cuneiform and 1st, 2nd and 3rd metatarsal bones). Articular disease around and dissolution of the talus as well as insufficiency fractures and avulsion of the calcaneal tuberosity are also demonstrated⁸.

Early changes are characterized by erythema, edema and warmth of the foot, but no structural changes are demonstrated on radiographs. Many as a result are misdiagnosed as having a foot infection, gout, arthritis, fracture or even venous insufficiency. MRI at this stage could actually show soft tissue and marrow edema of the mid foot, effusions and fluid collections⁹. The bone marrow edema typically is not restricted to one or two bones, but is seen in the entire midfoot (Figure 1). In addition, periarticular soft tissue and mild bone marrow enhancement can also be seen on contrast enhanced MRI¹⁰. Early intervention at this point could prevent permanent deformity and resultant morbidity. This involves casting/immobilization, stress reduction and later removable braces or a Charcot restraint orthotic walker¹¹.

Subacute/late changes involve bone erosion/resorption, bone fragmentation, joint dislocation and subsequent permanent deformity. Eventually, there is collapse of the longitudinal arch and increased load bearing on the cuboid, resulting in a "rocker-bottom" deformity. There is sclerosis of the bone surrounding the affected joints, followed by remodeling, and possible considerable morbidity of the patient. No substantial soft tissue or marrow edema may be noted at this stage¹². (Figure 2).

2. OSTEOMYELITIS

Acute vs Chronic Osteomyelitis

Osteomyelitis has a continuum of presentations, from acute to chronic stages. Diabetics generally have a higher incidence of chronic osteomyelitis, often secondary to a recurrent or chronically poor wound. In these patients, the ongoing infection is often more than 6 weeks, with low grade fever and possibly chronically discharging sinus tracts. Recognizing and differentiating between these stages may be important in terms of treatment aspects, for example, identification of a sequestrum (which may be seen in chronic osteomyelitis) often leads to surgical debridement.

Radiology, in particular MRI, can help in the diagnosis. On radiographs, findings may be normal for the first 2-3 weeks after the onset of infection because 30% to 50% of bone density must be lost before a lucency can be appreciated¹³. Detection of chronic osteomyelitis is also difficult because a sequestrum is visible in only 9% of cases. Progressive changes on serial radiographs have a sensitivity of 14% and specificity of 70%¹³.

In contrast, detection of marrow abnormality on MRI is a far more sensitive indicator of osteomyelitis than are the lytic changes seen on plain radiography. Osteomyelitis is characterized by low signal of the marrow on T1 weighted images, high signal on T2 weighted/STIR images, with enhancement of the marrow after administration of intravenous contrast. The STIR pulse sequence is considered highly sensitive for abnormalities with a negative predictive value approaching 100% for acute osteomyelitis (Figure 3).

In acute infection, there tends to be a wide zone of transition and poor definition between normal and diseased marrow, as compared to a relatively sharp interface between normal and diseased marrow¹³. Also in chronic osteomyelitis, marked sclerosis and periosteal reaction is often demonstrated (Figure 4), and additional features may also include sequestra, cloacae, abscesses, and subperiosteal fluid collection.

Osteomyelitis vs Neuroarthropathic Joint

Uniquely in diabetic patients, osteomyelitis and neuropathic osteoarthropathy of the foot may additionally (and not uncommonly) be superimposed onto one another, making diagnosis and thus management difficult. Both entities may demonstrate bone marrow oedema and enhancement, joint effusion and surrounding soft tissue oedema¹⁴. Early differentiation between these conditions is crucial, as initiation of appropriate treatment for osteomyelitis can curtail future morbidity and even mortality¹³.

Several features are useful in differentiating both these conditions on MRI.

A) MARROW CHARACTERISTICS

As mentioned, osteomyelitis is visible as a low signal intensity on T1-weighted images and high signal intensity on T2weighted, STIR, or fat-saturated images. In those instances where the marrow is hyperintense on T2 weighted images, but no significant signal loss is seen on the corresponding T1 weighted images, periosteitis (reactive related or a non



Fig. 1: Known diabetic patient on oral hypoglycaemic agents initially presented with erythema and swelling of the foot, with no recent history of any trauma to the region. There was also no other systemic signs or symptoms present. MRI performed after the initial screening radiographs demonstrates increased osseous edema at the region of the midfoot (as illustrated), suspicious for early neuroarthropathic changes. The patient was however eventually lost to follow up.
 (a) Oblique radiographic projections of the foot demonstrates diffuse periosteal reaction along the shaft of the 4th metatarsal, likely secondary to previous fracture/trauma. No other significant structural changes are however noted.
 (b) T2-weighted fat suppressed axial MRI image demonstrates marrow edema at all the metatarsophalangeal joints (arrows). No evidence of fracture or bony erosions to account for such findings. Findings were thus attributed to possible early neuroarthropathic change.

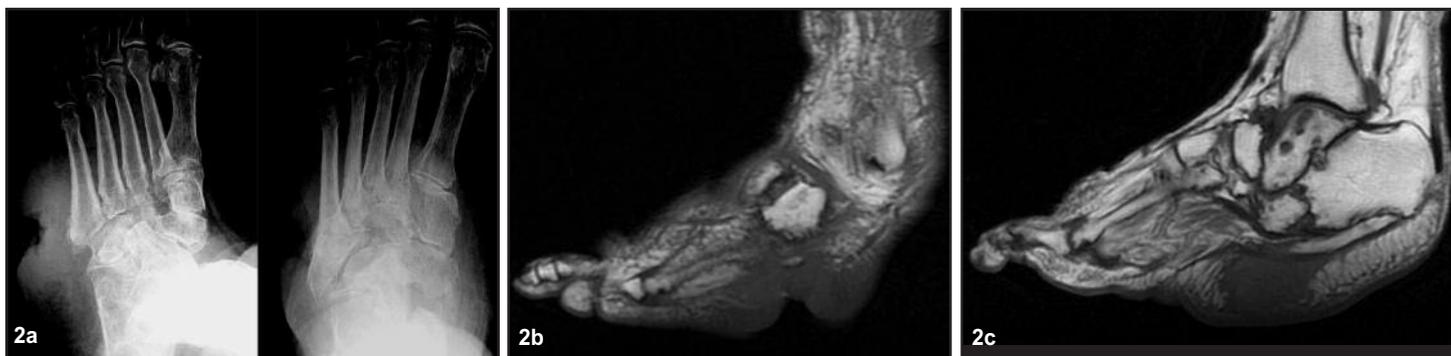


Fig. 2: Patient has poorly controlled diabetes, with Charcot foot and long standing ulcer over the lateral aspect of the left foot; was admitted with sepsis from recurrent ulcer infection. Exploration of the wound post imaging revealed deep extension till the muscle layers, but no osseous involvement.
 (a) Frontal and oblique radiographic projections of the foot demonstrate Lisfranc injury, as well as Chopart's fracture-dislocation with severe secondary degenerative changes. There is a fracture dislocation at the talonavicular joint. There is widening of the tarsal-metatarsal joint between the second and third metatarsals (better demonstrated on the MRI), and deformities of the cuneiforms and cuboid. Large subcutaneous defect in keeping with chronic ulceration is demonstrated at the lateral aspect of the foot. No adjacent radiographic features of osteomyelitis at the base of the 5th metatarsal.
 (b), (c) T1-weighted sagittal MRI images of the same patient demonstrate loss of normal longitudinal arch of the patient, with resultant "rocker-bottom" deformity, and excessive weight distribution on the cuboid bone (b). This has resulted in underlying pressure ulcer formation. Background of disorganised mid foot osseous structures with smaller fragments is also noted.

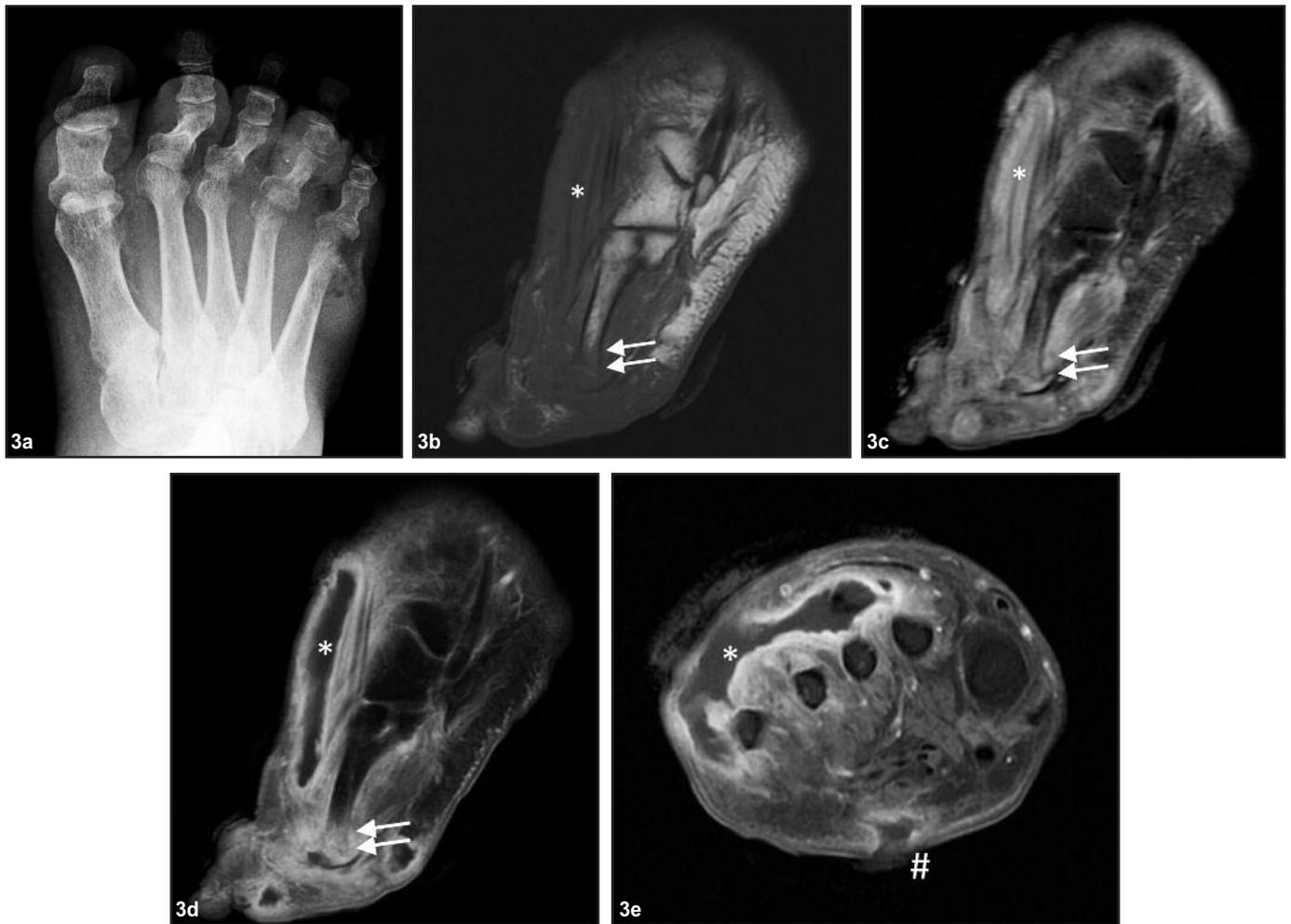


Fig. 3: Poorly compliant diabetic. Noticed foot swelling with discharge from plantar ulcers x 2 weeks, but initially self medicated and refused to seek medical attention. On examination, the patient was noted to have foot drop (attributed to motor neuropathy secondary to his diabetes), with resultant pedal ulcers over the pressure points at the metatarsal heads, plantar surface of the midfoot, as well as dorsal aspect of the mid foot (likely from chronic abrasion due to poor fitting feet wear). There was erythema and fluctuance of the foot, with several sinuses at the sole draining serous fluid. Post imaging, patient eventually underwent below knee amputation due to wound complications and poor vascular supply.

(a) Radiographic projection of the foot demonstrates diffuse soft tissue swelling over the right forefoot. Subtle bony erosions and ill defined lucencies seen over the 4th and 5th metatarsal heads, suspicious for osteomyelitis. There are flexion deformities at the metatarsophalangeal joints, possibly related to underlying neuropathy.

(b) T1-weighted sagittal (c) STIR sagittal (d) gadolinium enhanced T1-weighted fat suppressed MRI images demonstrate marrow edema, with low signal intensity on T1-weighted image, high signal on STIR image and consequently enhancement of the marrow post contrast administration of the 4th and 5th (not demonstrated) metatarsals. Features are in keeping with osteomyelitis of the 4th and 5th metatarsal heads, with likely septic arthritis of corresponding metatarsophalangeal joints.

(e) Axial gadolinium-enhanced T1-weighted fat-suppressed MR image shows a central plantar ulcer (#, with discontinuity of the skin) and a partially imaged lateral dorsal ulcer. The dorsally located ulcer is noted to be associated with a deep seated collection/abscess (*), with additional ramifications/extensions in keeping with sinus tract formation and adjacent cellulitis.

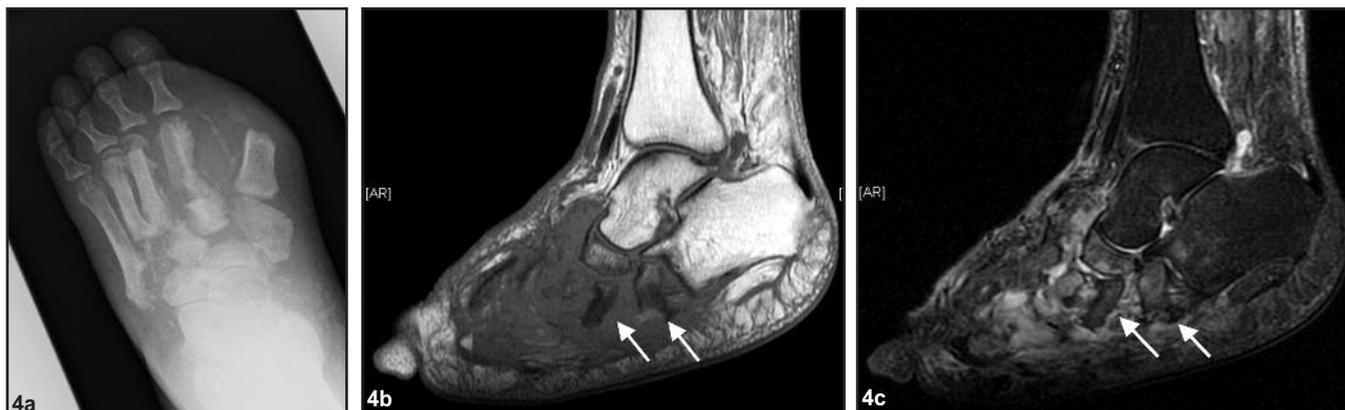


Fig. 4: Patient has a left Charcot's foot with multiple surgical debridements for a long standing infected plantar ulcer at the region of the mid foot. Radiological findings are in keeping with chronic osteomyelitis.
 (a) Frontal radiographic projections of the foot demonstrates severe disorganization/destruction of the mid foot in particular, with bony debris, osseous erosion and dislocated/subluxed joints. Previous Ray amputation of the big toe. There is increased sclerosis/periosteal reaction, in particular of the 2nd and 3rd metatarsals, as well as the middle and lateral cuneiform bones. Background extensive soft tissue swelling and vascular calcifications are noted.
 (b) T1-weighted and (c) STIR sagittal MRI images of the same patient demonstrate edema of the deformed cuneiform bones (arrows), with attendant significant soft tissue swelling/edema. No contrast administration due to the patient having end stage renal failure.



Fig. 5: Gadolinium enhanced T1-weighted fat suppressed MRI images demonstrate faint enhancement of the marrow post contrast administration of the base of the 5th metatarsal with attendant underlying plantar ulcer. There was however no significant signal loss seen on the corresponding T1 weighted images; periosteitis is thus more likely than osteomyelitis. Evidence of tiny bony fragments seen, in keeping with associated neuroarthropathic changes.

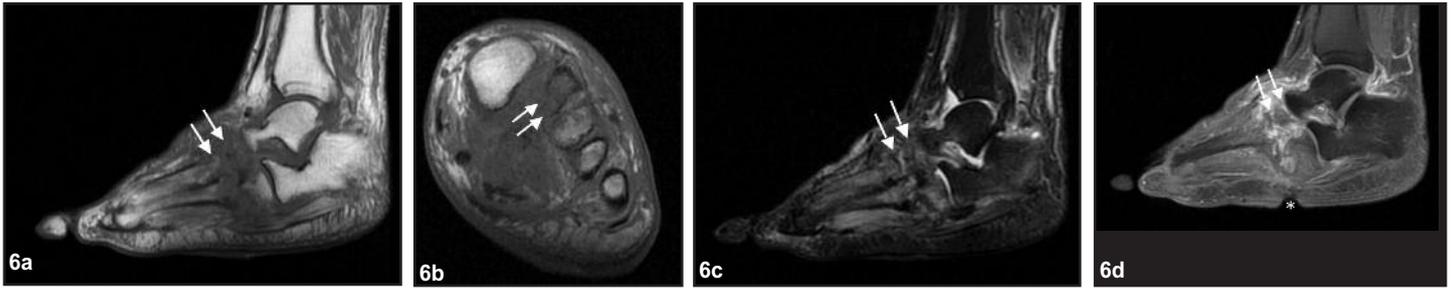


Fig. 6: Diabetic patient with increasing purulent discharge from long standing plantar ulcer. Features suspicious for septic arthritis was confirmed on surgery.

(a) Sagittal T1-weighted, (b) Axial T1-weighted, (c) Sagittal STIR, and (d) Sagittal gadolinium-enhanced T1-weighted fat-suppressed MR images demonstrate a plantar ulcer (* - d), adjacent cellulitis, small joint effusion, with a thick area of enhancement (arrows) and erosion in the 2nd and 3rd tarso-metatarsal joint, findings indicative of septic arthritis. Attendant marrow changes with enhancement of the 2nd and 3rd metatarsal bases in keeping with associated osteomyelitis.

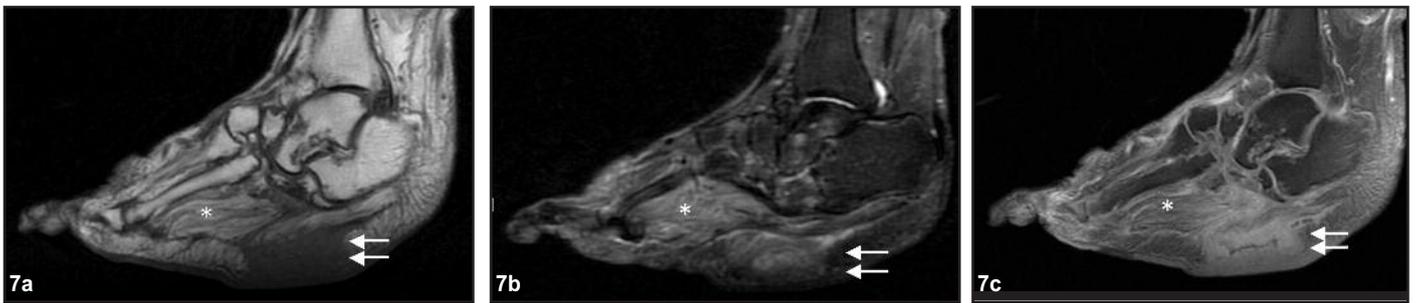


Fig. 7: Identical patient as illustrated in Fig. 2. Current images illustrate adventitial bursitis (arrows) secondary to prolonged pressure at mid foot due to Rocker bottom deformity. Adjacent ulcer formation is demonstrated in Fig. 2.

(a) Sagittal T1-weighted, (b) STIR and (c) gadolinium-enhanced T1-weighted with fat suppression MR images show an area of high signal fluid intensity at the plantar surface of the mid foot secondary to a rocker bottom deformity, with peripheral rimlike enhancement, findings indicative of a focal fluid collection/bursitis (arrows). There is preservation of the adjacent subcutaneous fat (thus excluding cellulitis). Atrophy is seen in the intrinsic foot musculature (*), with increased quantities of fat replacement and corresponding high signal intensity on T1-weighted image. There is also no significant contrast enhancement. Such findings are in keeping with diabetic related denervation of the small muscles.

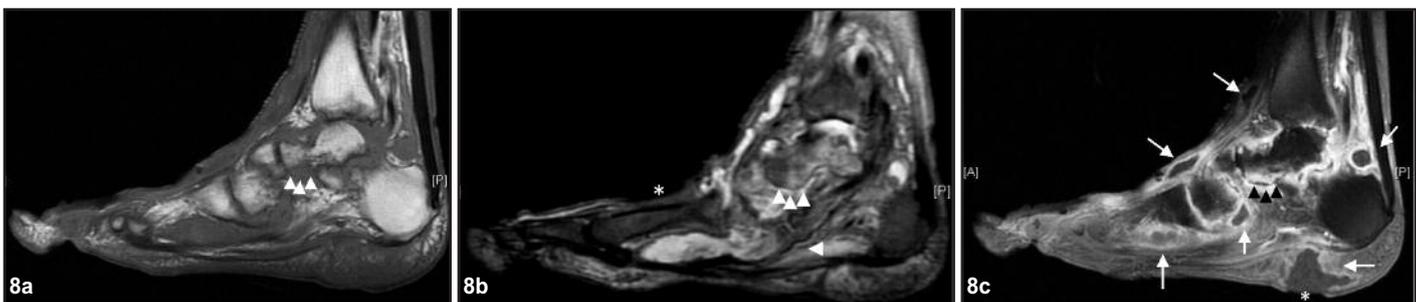


Fig. 8: Elderly patient with long standing diabetes, peripheral vascular disease and known chronic osteomyelitis. The patient has had a chronic heel ulcer with multiple infective exacerbations; repeated attempts at surgical debridement were largely unsuccessful. Latest MRI demonstrates widespread superficial and deep abscesses (as illustrated). Being a poor surgical candidate for amputation due to multiple co-morbidities, the patient was instead treated empirically with IV antibiotics and eventually discharged for outpatient follow up.

(a) Sagittal T1-weighted, (b) STIR and (c) gadolinium-enhanced T1-weighted fat-suppressed MR images shows plantar and dorsal ulcers (*), with associated subcutaneous collections (arrows) that are hypointense centrally on the T1-weighted images and hyperintense on the T2-weighted image, with a thick rimlike layer of enhancement in c. Of note are the smaller collections, which would not have been accurately detected/ diagnosed without the benefit of contrast administration. Background neuroarthropathic changes involving predominantly the mid foot is demonstrated. In addition, the osseous structures of the anterior talus appears more regular and better defined in b and c (arrowheads). This appearance, which is known as the ghost sign, is indicative of neuroarthropathy with superimposed osteomyelitis.

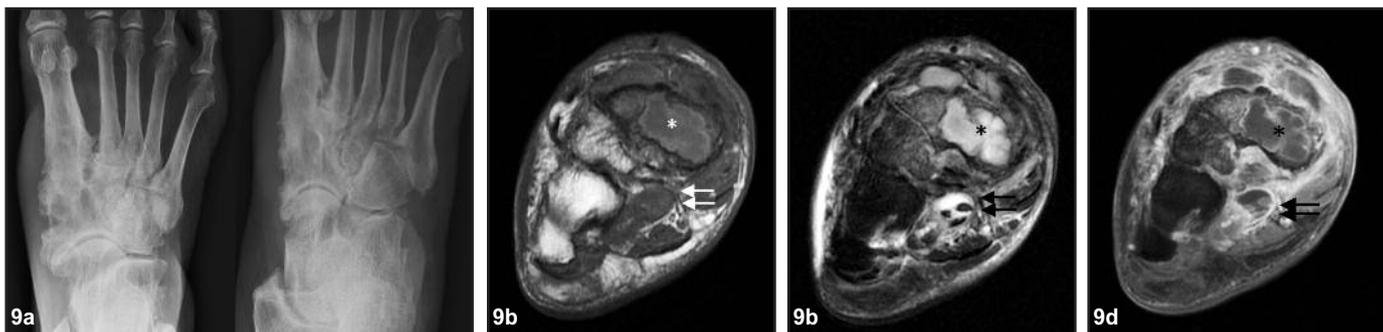


Fig. 9: Patient had poor social support, staying alone at home. She presented with right foot pain for 2 months, before seeking medical attention as she was no longer able to weight bear/ambulate on the affected foot. Radiographs, and subsequently MRI was performed, demonstrating the large intraosseous abscesses (the immense advantage of MRI over radiographs in demarcating the exact extent of the condition is well illustrated here). Drainage of the abscesses with debridement, curettage and application of negative pressure dressing was performed. Patient subsequently improved and was discharged well under antibiotic cover.

(a) Frontal and oblique radiographic projection of the foot shows extensive bony erosions involving the base of the 1st and 2nd metatarsals, navicular as well as the medial, middle and lateral cuneiform bones. Findings are compatible with osteomyelitis. There is also likely bony fusion of the first metatarsal, medial cuneiform and navicular bones. Soft tissue swelling of the mid-foot is noted.

(b) Axial T1-weighted, (c) T2-weighted fat suppressed and (d) gadolinium-enhanced T1-weighted fat-suppressed MR images show intraosseous rim enhancing abscesses involving predominantly the medial and middle cuneiform bones (*). A discharging tract was also noted medially (not demonstrated on available images) with surrounding adjacent cellulitis, small dorsal abscesses and soft tissue edema noted. Osteomyelitis of the 1st metatarsal, base of the second metatarsal, medial and middle cuneiform and navicular bones were also demonstrated. There is also synovial thickening and fluid distension of the flexor hallucis longus tendon (arrows), in keeping with tenosynovitis.

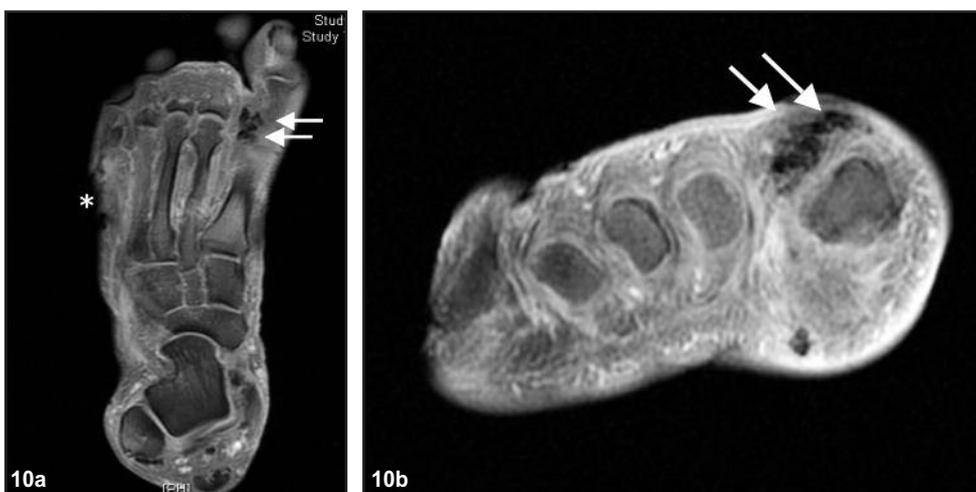


Fig. 10: Poorly controlled diabetic patient had initially presented with right foot pain for 2 weeks, of which pain was progressively worsening. On examination, patient was very septic, and there was swelling of the right fore foot involving mainly the first digit, associated with redness and warmth. Hemorrhagic bullae measuring 2X3cm was noted in between the first and second digit.

(a) Sagittal and (b) axial gadolinium-enhanced T1-weighted fat-suppressed MR images demonstrate non enhancing subcutaneous gas locules (which were too minute to be effectively seen on the prior radiographs), with an additional remote ulcer (*) over the lateral aspect of the foot. It is unlikely that gas entered from the ulcer. Findings are suggestive of wet gangrene and infection.

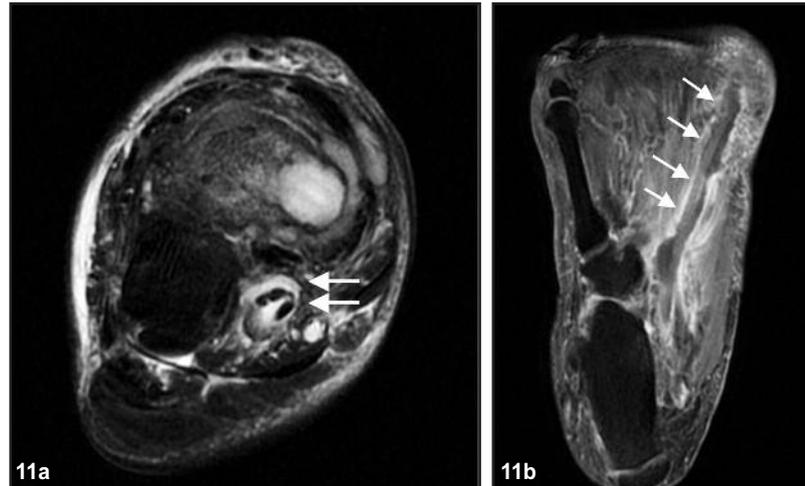


Fig. 11: Identical patient as in Fig. 9. Septic tenosynovitis was confirmed under surgical aseptic techniques, with exposure of the tendon and drainage performed.

(a) Axial STIR and (b) coronal gadolinium-enhanced T1-weighted fat-suppressed MR images shows tendinous thickening, fluid distension and peritendinous nodular enhancement of the anterior flexor hallucis longus tendon, in keeping with tenosynovitis. Adjacent intraosseous abscess noted.



Fig. 12: Sagittal gadolinium-enhanced T1-weighted fat-suppressed MR images demonstrate multiple rim enhancing abscesses, some of which are noted within the small plantar muscles of the foot (arrows)/pyomyositis. There is an additional heel ulcer demonstrated (*), likely the source of infective spread.

medullary infection) is more likely than osteomyelitis. Acute cases of neuroarthropathy may actually mimic osteomyelitis (and may require other secondary signs for differentiation); however, chronic neuroarthropathic joints are typified by either normal or low signal on both T1 and T2 weighted imaging.

B) DISTRIBUTION

Bone marrow edema in osteomyelitis tend to involve a single bone, and are overlined by ulcers (which provide a portal of entry for infection) in the vast majority; whereas in neuroarthropathy, it is more periarticular and subchondral in origin as well as involving multiple midfoot bones.

C) MARROW ENHANCEMENT CHARECTERISTICS

As mentioned, abnormal enhancement of the bone marrow is helpful in identifying cases of acute osteomyelitis. This is particularly when the “ghost sign” is demonstrated, which aids in distinguishing osteomyelitis in a background of neuroarthropathy from changes due to acute neuropathy (Figure 8). It refers to poor definition of the margins of a bone on T1-weighted images, which become clear after contrast administration¹⁵.

D) ADDITONAL SECONDARY SIGNS

Several additional features seen on MRI tend to be associated with osteomyelitis. Their presence, in the appropriate clinical context, are very helpful in making the diagnosis. These

include overlying skin callus and ulcer formation, replacement of normal subcutaneous fat, abscesses, cellulitis, tenosynovitis and sinus tracts. Implanted foreign bodies may also occasionally be demonstrated. These are detailed subsequently. Similarly, an abnormal joint in a diabetic foot, presence of subchondral cysts and intraarticular bodies with absence of the aforementioned secondary signs for osteomyelitis, support neuroarthropathy without infection⁸.

3. SEPTIC ARTHRITIS

Similarly to osteomyelitis, cases of septic arthritis are often secondary to neighboring ulcer/sinus tract formation with associated inoculation. A combination of T1-weighted, T2-weighted, short-tau inversion recovery, and postcontrast T1-weighted fat-suppressed series are most helpful¹⁶.

Synovial enhancement and the presence of a joint effusion (at times complex) have been reported to have the highest correlation with the clinical diagnosis of a septic joint. Perisynovial soft tissue edema is also commonly seen (Figure 6). Characteristics of enhancement can also distinguish a periarticular abscess from surrounding myositis and to evaluate the degree of synovial inflammation. However, the absence of a joint effusion, especially in the small joints of the hands and feet, does not exclude infection of the joint.

On T2-weighted images, high signal intensity in the adjacent bone marrow helps in differentiating septic arthritis from synovitis. However, increased signal intensity does not necessarily indicate osteomyelitis, as previously discussed.

SOFT TISSUE COMPLICATIONS

1. ULCERS/CALLUSES

Pedal osteomyelitis in a diabetic patient is underlined by up to 90% by ulcers which develop as a result of repetitive microtrauma applied on pressure points³. In addition, approximately 20% of patients may have infection at two or more sites²⁰. Of all these diabetic ulcerations, approximately 45% to 60% are purely neuropathic, while up to 45% have neuropathic and ischemic components¹⁷. Peripheral sensory neuropathy in the face of unperceived trauma is the primary contributing factor leading to diabetic foot ulcerations. These patients do not demonstrate any overt clinical signs or symptoms other than for the ulcer itself. More rarely, diabetes can result in motor neuropathy which leads to anterior crural muscle atrophy or intrinsic muscle wasting, with resultant deformities such as foot drop, equinus, hammertoe, and prominent plantar metatarsal heads¹⁸. Autonomic neuropathy often results in dry skin with cracking and fissuring, creating a portal of entry for bacteria. Calluses, and ultimately ulcers, occur over common pressure points of the foot. In ambulatory patients, these are the metatarsal heads (in particular at the curve of the first and fifth digits), the tendo-achilles bursa, the soft tissue medial to the first metatarsal head, the malleoli, the distal toes and the tarsometatarsal joints¹⁹. Patients with significant neuroarthropathy also tend to develop calluses/ulcers over the cuboid bone, which becomes weight-bearing after collapse of the longitudinal arch of the foot (Figure 5). In non ambulatory patients, these occur in the calcaneus and lateral malleolus, because of chronic pressure on the externally rotated foot.

On MR imaging, calluses often appear as a low signal mass of the skin/infiltrating the subcutaneous fat on T1 weighted imaging, and a low to intermediate signal on T2 weighted imaging. These may enhance post contrast, but may be differentiated from a superficial infection due to lack of attendant soft tissue changes. Underlying adventitial bursa formation may also be demonstrated, as a thin fluid collection, again without attendant subcutaneous fat stranding (Figure 7). Skin ulceration, on MR imaging, is typified by focal interruption of the cutaneous line, with an elevated margin. Acute ulcers tend to demonstrate high signal intensity on T2 weighted imaging, with peripheral enhancement. Chronic ulceration however may be associated with fibrous healing and thus appears as intermediate to low signal. Ulcers greater than 2 cm in depth are particularly susceptible to osteomyelitis¹⁹. Ulcers may also be associated with sinus tracts (which display a tram track pattern of enhancement) or abscesses (Figure 3).

2. CELLULITIS

Cellulitis is commonly used to indicate an acute nonnecrotizing inflammation of the skin and subcutaneous tissues that does not involve the deep fascia or muscles. It is characterized by localized pain, swelling, tenderness, erythema, and warmth, and is rather common amongst patients with advanced diabetic foot. On clinical examination, it can be easily confused /masked by edematous change brought about by acute neuropathy²¹. Utilising MR imaging, both of these conditions can demonstrate skin thickening, though prominent reticulation of fat and contrast enhancement²² are more distinguishing of the former.

3. ABSCESES

Abscesses normally develop after a long period of non treatment, and are usually associated with loss of skin/ulcer formation. Eventually, due to spread along fascial planes, these may involve the deep fascia. Abscesses may communicate with sinus tracts and be associated with osteomyelitis (Figure 3). The reported incidence of soft tissue abscesses in patients with foot infection ranges from 10%²² to 50% on MRI. On MRI, an abscess is seen as a focal collection of fluid signal, with peripheral rim enhancement on post-contrast images²³. Routine administration of contrast is recommended as it not only helps in the identification of small, superficially located abscesses, it can also aid in the demarcation of its extent, such that surgically, it would be helpful during debridement (Figure 8). Abscesses can not only develop in the superficial tissues, these also occur in the deep muscles (pyomyositis) as well as the within the bones (Figure 9). Occasionally, attendant foreign bodies may be seen in diabetic feet secondary to sensory neuropathy. These may have low signal intensity on both T1 and T2 weighted sequences, with blooming artefact seen on gradient echo images. There may also be a rim of enhancement, which is due to granulomatous reaction⁸.

4. GANGRENE

Gangrene occurs when there is necrosis of the soft tissues because its blood supply is interrupted, which is not uncommon in the extremities of diabetics due to microvasculopathy. It can present as either dry or wet, with

the latter at times associated with gas. Wet/gas gangrene spreads rapidly, often with systemic signs such as fever and can be fatal; hence needs to be treated urgently, via surgery.

On contrast enhanced MRI, gangrene appears as a non enhancing area of devitalized tissue that is distinctly demarcated from normal surrounding tissue. The periphery of the devitalized tissue may demonstrate reactive hyperaemia and enhancement²⁴. Gradient echo sequences are also important, as these are most sensitive in these instances for depicting small foci of blooming artefacts secondary to gas, which also appear as signal-void areas in all conventional sequences (Figure 10).

TENDINOUS COMPLICATIONS

1. TENOSYNOVITIS

Septic tenosynovitis of the foot typically occurs as a result of contiguous spread of infection from an adjacent ulcer or sinus tract. MRI usually reveals T1-hypointense and T2-hyperintense fluid distending the tendon sheath; however, the signal characteristics of the fluid can vary depending on the presence of debris, gas, or blood. In addition, the tendons lose their normal low signal intensity and become thickened and indistinct; the synovial lining of the tendon sheath thickens and becomes indistinct; and there is surrounding edema. Although the presence of fluid in a tendon can be normal (due to mechanical causes or trauma), fluid seen in the anterior tendons is almost never normal²⁵. In the forefoot, nearly two-thirds of all tendon infections involve the flexor tendons and are a result of plantar forefoot ulceration (Figure 9, 11). Septic tenosynovitis most commonly occurs in the peroneal tendons from a lateral malleolus ulcer and in the Achilles tendon from a calcaneal ulcer²⁵.

MUSCLE COMPLICATIONS

1. DENERVATED MUSCLE

Peripheral neuropathies have been commonly described in diabetic patients, arising from a host of multifactorial causes, eventually leading to muscle denervation.

When acute, muscle denervation may be featureless on MR imaging. In the subacute stage, there is demonstrable uniform edema throughout the involved muscle²⁶, with bright signal intensity on T2weighted and inversion recovery imaging. This finding usually becomes evident on MR images until approximately 2–4 weeks after denervation has occurred²⁶. If normal innervation is not restored, chronic and permanent atrophy with fatty infiltration develops, indicating irreversible change²⁶. On MR images, the affected muscle bellies reveal increased quantities of fat with its characteristic signal intensity, usually with a decreased volume of muscle tissue. T2-weighted and inversion-recovery images show variable findings in chronic denervation²⁶ and thus are less reliable than T1-weighted images in revealing changes of chronic muscle denervation (Figure 7).

2. MYOSITIS/PYOMYOSITIS

Muscle infection (myositis) without abscess or necrosis may produce edema as the sole abnormality on MR images. Bacterial myositis may result from direct extension of infection in tissues adjacent to a muscle, such as osteomyelitis or a subcutaneous abscess²⁷. The MR images and clinical history may suggest the presence of such an

infection. Bacterial myositis frequently progresses to abscess formation (pyomyositis). As previously detailed, these can appear as focal lesions with an enhancing rim, a diffuse signal increase on T2-weighted images, and an associated irregular enhancement of the deep fascia on postcontrast T1-weighted images (Figure 12).

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Multiple Choice Questions

Stem A: Imaging Techniques

1. Radiographs are the preferred initial imaging modality when assessing the diabetic foot.
2. MR imaging of the foot should always be performed with as large a field of view as possible.
3. It is important, where possible, to place markers near visible ulcers during MR imaging.
4. T1w sequences are anatomically detailed with high resolution, is sensitive for bone marrow change, and have high specificity in the detection of osteomyelitis (OM).
5. when performing MR imaging of the foot, IV gadolinium must be administered for all patients with adequate renal function.

Stem B: Osseous Complications

1. The "ghost sign" is useful for identifying osteomyelitis in a background of neuropathic change.
2. T1W sequence is crucial in differentiating acute OM from chronic OM.
3. Acute osteomyelitis is characterized by increased sclerosis and periosteal reaction on radiographs.
4. On MRI, both acute osteomyelitis (OM) and neuropathic foot can demonstrate bone marrow edema, bone marrow enhancement, joint effusion and surrounding soft tissue edema.

Stem C: Septic arthritis/abscesses/tenosynovitis

1. To diagnose septic arthritis, there MUST be perisynovial soft tissue edema, synovial enhancement, increased signal within the adjacent bone marrow and a joint effusion.
2. On T2W images, high signal intensity of the bone marrow helps differentiate septic arthritis from reactive synovitis.
3. MRI is important in demarcating the extent of an abscess or sinus tract for the purpose of surgical planning.
4. On MRI, fluid seen in the anterior tendons of the foot are often attributed to reactive change and can be ignored.
5. Septic tenosynovitis of the peroneal tendons are commonly related to medial malleolar ulcers.

Stem D: Miscellaneous

1. In the acute phase of de-nervated muscle, MRI findings is similar that of acute myositis.
2. Pedal OM in a diabetic patient can be underlined by an ulcer in up to 90% of patients.
3. It is not uncommon for patients with advanced diabetic related neuroarthropathy to develop OM of the cuboid bone.
4. Adventitial bursitis is one of the secondary findings in a diabetic foot.
5. Calluses can be differentiated from superficial infections by their lack of contrast enhancement.
6. T1W images are most important when evaluating for chronic muscle de-nervation.
7. On contrast enhanced MRI, gangrenous tissue appears as a non enhancing area of devitalized tissue that is distinctly demarcated from the normal surrounding tissue.