Learning objectives

1. Describe the role and potential limitations of imaging in the evaluation of diabetic foot,

2. Recognize the various imaging manifestations of diabetic foot on MRI and their clinical significance.

3. Identify the imaging features which help to differentiate osteomyelitis from neuropathic arthropathy.
Background

Diabetic foot remains a challenge for the clinicians due to confusing clinical picture and associated complications. It may present as a cellulitis, ulceration, sinus tracts, myositis, abscesses, osteomyelitis, septic arthritis, ischemic devitalized bone or neuropathic osteoarthropathy. Radiographs, although often used for initial work up, are neither sensitive nor specific. Triple phase bone scintigraphy is sensitive however lacks anatomic details and specificity. MRI with its excellent contrast resolution and anatomic detail is an essential tool to help differentiate osteomyelitis from neuropathic arthropathy, identify conditions requiring surgical interventions and map the extent of infection. However, distinction between osteomyelitis and neuropathic arthropathy may sometimes be difficult based on MRI alone. Labelled leukocyte scintigraphy is complementary to MRI in making this diagnosis and is a useful tool for follow up of the patients with osteomyelitis. In this exhibit, we describe the spectrum of imaging findings in a diabetic foot and discuss the role and limitations of the various imaging modalities in evaluation of diabetic foot.
Findings and procedure details

Plain radiography is the preferred first line imaging investigation, 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 low, due to inability to adequately demonstrate the soft tissues. In addition, Charcot's foot and osteomyelitis may show overlapping features of advanced Charcot's foot and osteomyelitis.

MRI scanning is the gold standard at present for differentiating the various pathologies in a diabetic foot. It is superb for evaluating for synovial effusions, cellulitis, muscle edema, bone marrow edema, tendon and ligament injuries, cysts or abscesses, damage to cartilage and osteonecrosis. MRI provides excellent contrast resolution and anatomical detail, allowing effective identification of conditions requiring surgical intervention from those that can be managed conservatively. 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. MRI can detect early bone and soft tissue abnormalities with resultant early therapeutic intervention, and better symptomatic relief and functional outcome. It is a useful tool for preoperative mapping of the extent of infection and thus helps to limit the area of resection.

**MRI FINDINGS:**

**OSSEOUS COMPLICATIONS**

1. **NEUROARTHROPATHY (CHARCOT'S FOOT)**
   - Pathophysiogenesis-combination of loss of deep sensation, proprioception and peripheral vascular disease, exacerbated by chronic, repetitive trauma to the foot, results in poor healing, joint instability, malalignment, erosion of chondral surfaces and eventual fracture, disorganization and increased new bone formation.
   - Develops in < 1% of persons with diabetes.
   - Site- mid/hind foot (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).
   - Early changes (figure 1) - erythema, edema and warmth of the foot. MRI may show soft tissue and marrow edema of the mid foot, effusions and fluid collections in patients with normal radiographs at this stage. The bone
marrow edema typically is not restricted to one or two bones, but is seen in the entire midfoot. In addition, periarticular soft tissue and mild bone marrow enhancement can also be seen on contrast enhanced MRI.

- Late changes (figure 2, 3) - bone erosion/resorption, bone fragmentation, increased density, debris, joint disorganization, 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. These features are easily seen on radiographs however a co-existent osteomyelitis cannot be excluded. MRI may not show any substantial soft tissue or marrow edema at this stage helping to differentiate it from active infection.

2. OSTEOMYELITIS

- Initial radiographs are neither sensitive nor very specific for diagnosing osteomyelitis. Radiographs may be normal in the first 2-3 weeks after the onset of infection. They are not very sensitive to detect sequestrum, an important feature of chronic osteomyelitis which often needs surgical debridement. MRI can detect marrow changes due to osteomyelitis much earlier than development of the lytic changes seen on plain radiography.
- Higher incidence of chronic than acute osteomyelitis in diabetes, often due to a recurrent or chronically poor wound.
- Infection is often present for several weeks, with low grade fever and possibly chronically discharging sinus tracts. On MRI, tracking of the ulcer or sinus tract down to bone at MR imaging and evaluate the signal intensity of marrow can determine if osteomyelitis is present (figure 4). Noticeably low signal intensity on T1-weighted images is a primary sign of osteomyelitis. STIR pulse sequence is considered highly sensitive for abnormalities with a negative predictive value approaching 100% for acute osteomyelitis. Periosteal reaction is a useful secondary sign of osteomyelitis, seen as a low-signal-intensity line separated from underlying bone by a high-signal-intensity layer of fluid or pus.
- Acute osteomyelitis - low marrow signal on T1 weighted images, high signal on T2 weighted/STIR images, with marrow enhancement on gadolinium enhanced images. Acute osteomyelitis shows tends a relatively wide zone of transition and poor definition between normal and diseased marrow.
- Chronic osteomyelitis - relatively sharp interface between normal and diseased marrow sclerosis. Additional features include periosteal reaction, sequestra, cloacae, abscesses, discharging sinuses and subperiosteal fluid collection.
- Imaging is often used in the post operative evaluation of diabetic foot (figure 5). Most patients who undergo amputation have little postoperative bone marrow edema. The criteria for diagnosing osteomyelitis at the site of amputation are the same as those for patients who have not undergone
amputation. However, postoperative bone edema may occur at the surgical site in patients who undergo débridement instead of amputation and should not be mistaken for osteomyelitis.

- Uniquely in diabetic patients, osteomyelitis and neuropathic osteoarthropathy of the foot may be superimposed and show overlapping features making diagnosis and thus management difficult. Both entities may demonstrate bone marrow oedema and enhancement, joint effusion and surrounding soft tissue oedema. An interesting observation on MRI is "ghost sign". The ghost sign is indicative of neuro-osteoarthropathy with superimposed osteomyelitis. It refers to poor definition of the margins of a bone on T1-weighted images, which become clear after contrast administration.

- Early differentiation between osteomyelitis and neuropathic osteoarthropathy is crucial, as initiation of appropriate treatment for osteomyelitis may reduce future morbidity and even mortality. MRI may help resolve this dilemma since several features are useful in differentiating both these conditions on MRI as described in the table below:

<table>
<thead>
<tr>
<th>MRI features</th>
<th>Osteomyelitis</th>
<th>Neuroarthropathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marrow signal</td>
<td>T1 hypointense and T2/STIR hyperintense</td>
<td>Acute - may mimic osteomyelitis (and may require other secondary signs for differentiation); Chronic - typically either normal or low signal on both T1 and T2 weighted imaging.</td>
</tr>
<tr>
<td>Distribution</td>
<td>Epicentred on bone.</td>
<td>Epicentred on bone. Bone marrow edema tends to be more periarticular and subchondral in origin as well as involves multiple midfoot bones.</td>
</tr>
<tr>
<td>Soft tissues</td>
<td>Soft tissues are generally not involved.</td>
<td>skin callus and ulcer formation, replacement of normal subcutaneous fat, abscesses, cellulitis,</td>
</tr>
</tbody>
</table>

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Ancillary findings | presence of subchondral cysts and intraarticular bodies with absence of the secondary signs for osteomyelitis, support neuroarthropathy without infection | periosteal reaction, sequestra, cloacae, abscesses, discharging sinuses and subperiosteal fluid collection.

3. SEPTIC ARTHRITIS

- Septic arthritis in diabetic foot is often secondary to neighboring ulcer/sinus tract formation with associated inoculation.
- Features include synovial enhancement, joint effusion and perisynovial soft tissue edema (figure 6). 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. CELLULITIS

- Non necrotizing inflammation of the skin and subcutaneous tissues that does not involve the deep fascia or muscles.
- Common amongst patients with advanced diabetic foot
- MR imaging demonstrates skin thickening along with prominent reticulation of fat and contrast enhancement, the latter helps to distinguish it from of the former edematous change of acute neuropathy (figure 7).

2. ULCER/CALLUS (figure 8)

- Callus and ulcer formation occur as a result of repetitive microtrauma applied on the pressure points in the setting of neuropathy and ischemia 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 joints. Patients with significant neuroarthropathy and resultant collapse of the longitudinal arch of the foot also tend to develop calluses/ulcers over the cuboid bone, which becomes weight-bearing.

• Non ambulatory patients develop these manifestations in the calcaneus and lateral malleolus, because of chronic pressure on the externally rotated foot. 90 % cases of pedal osteomyelitis in a diabetic patient are underlined by these ulcers.

• Calluses, on MR imaging, 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.

• 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 osteomyelitis. Ulcers may also be associated with sinus tracts (which display a tram track pattern of enhancement) or abscesses.

3. ABSCESSES

• Abscesses normally develop after a long period of non treatment, and are usually associated with loss of skin/ulcer formation. They develop in the superficial tissues, spread to deep fascia, muscles (pyomyositis) as well as bones.

• On MRI, an abscess is seen as a focal collection of fluid signal, with peripheral rim enhancement on postcontrast images. 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.

4. SINUS TRACT
• If a sinus tract is present, an ulcer may extend to the level of the adjacent osseous prominence. Familiarity with typical ulcer locations and sinus tract features is critical in the evaluation of osteomyelitis.
• A meandering sinus tract may appear round if viewed in cross section and may be mistaken for an abscess. On contrast-enhanced images, sinus tracts display a "tram-track" pattern of enhancement (figure 3). Sinus tracts should be evaluated in all imaging planes.

5. FOREIGN BODY

• Foreign bodies may be seen in diabetic patients with sensory neuropathy or after they undergo surgery.
• A careful search for a foreign body should be performed in patients with a soft-tissue infection and no adjacent ulcer. Foreign bodies usually are located under the metatarsal heads.
• A foreign body usually has low signal intensity on both T1- and T2-weighted MR images, and may show blooming artifact on gradient-echo images with surrounding rim of enhancement. The latter is indicative of a granulomatous reaction and should not be mistaken for an abscess. Distinction can be made on T2-weighted images: An abscess has the signal intensity of fluid, and a foreign body does not.

6. GANGRENE

• Gangrene in diabetic patients occurs due to end-organ ischemia. In most cases, gangrene is diagnosed clinically, and advanced imaging is unnecessary.
• Noninfected devitalized tissue is referred to as dry gangrene, whereas the term wet or gas gangrene refers to gangrenous tissue with superimposed infection. The latter spreads rapidly, often with systemic signs such as fever and can be fatal; hence needs to be treated urgently, via surgery.
• Contrast-enhanced MR imaging can delineate areas of soft-tissue devascularization which are depicted as a nonenhancing area of devitalized tissue that is sharply demarcated from surrounding viable tissue (figure 9). The periphery of the devitalized tissue may demonstrate reactive hyperemia and enhancement. Gas in a wet gangrene can be difficult to visualize at MR imaging (figure 10). Gas is seen as signal-void areas in all conventional sequences. Gradient echo sequences are sensitive for depicting small foci of blooming artefacts secondary to gas in these instances.
• Soft-tissue gas related to gangrene should be distinguished from that related to a skin ulcer through which gas can enter soft tissues. Gangrene usually
is associated with nonenhancing devitalized tissue and demonstrates more extensive soft-tissue gas than that seen surrounding an ulcer

7. TENOSYNOVITIS

- Due to contiguous spread of infection from an adjacent ulcer or sinus tract, most commonly in the peroneal tendons from a lateral malleolus ulcer and in the Achilles tendon from a calcaneal ulcer.
- MRI findings- 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. Tendons become thickened and indistinct with loss of normal low signal intensity. The synovial lining of the tendon sheath thickens and becomes indistinct with surrounding edema.

8. DENERVATED MUSCLE

- Peripheral neuropathies in diabetic patients eventually leading to muscle denervation.
- Stages of denervation: Acute, subacute, chronic
  - Acute stage-featureless on MRI.
  - Subacute stage- uniform edema throughout the involved muscle, with bright signal intensity on T2 weighted and inversion STIR images, usually evident on MR images approximately 2-4 weeks after denervation.
  - Chronic -Permanent atrophy with fatty infiltration indicating irreversible change develops if normal innervation is not restored.

9. MYOSITIS/PYOMYOSITIS

- Bacterial myositis may result from direct extension of infection in tissues adjacent to a muscle, such as osteomyelitis or a subcutaneous abscess.
- Muscle infection (myositis) without abscess or necrosis may produce edema as the sole abnormality on MR images (figure 7). Clinical history may suggest the presence of infection. Bacterial myositis frequently progresses to abscess formation (pyomyositis).

RADIONUCLIDE IMAGING STUDIES:
• TRIPLE PHASE BONE SCINTIGRAPHY using 99mTc-MDP is sensitive for bone disease in diabetic foot however lacks anatomic details and specificity.
• Although, it has been commonly used for work up of patients with diabetic foot in the past however the isolated routine usage of bone scanning as a screening modality for osteopathy in diabetes is decreasing and has largely been taken over by MRI. However, in early stages of osteitis and Charcot neuro-osteoarthropathy, radionuclide imaging may be superior to MRI.
• 99mTc-MDP is taken up in areas of hyperaemia, which may be a result of cellulitis in soft tissue, healing fractures, or osteitis, and is therefore not specific for bone infection. The uptake of radio-isotopes is increased in the feet of patients with diabetic neuropathy as a result of increased peripheral blood flow even in the absence of radiological abnormalities, making this a very sensitive test. The specificity of 99mTc-MDP scans in the diagnosis of osteomyelitis is better if three-phase imaging is performed with the addition of a radionuclide angiogram and 'blood-pool' images.
• INFLAMMATION-TARGETED LABELLED LEUKOCYTE SCINTIGRAPHY such as using WBCs labelled with 111In are used to detect infection in soft tissue and bone (figure 5). In patients of diabetic foot with a positive bone scan, a WBC scan can help identify presence of osteomyelitis. Similarly, it may be used as a problem solving tool where distinction between osteomyelitis and neuropathic arthropathy is difficult on MRI (figure 3). Labelled leukocyte scintigraphy is also useful tool for follow up of the patients with osteomyelitis.
• Dual-isotope imaging using both 99mTc-MDP and labelled leukocytes can reliably localise supra-added osteomyelitis and determine its extent in diabetic foot with known neuroarthropathy and ulcers.

**VASCULAR IMAGING**

• Vascular status should be determined in patients with diabetic foot can be assessed with Doppler ultrasound, with following conventional angiography or MR angiography.
Fig. 1: Figure 1a: Early neuroarthropathy: Sagittal T2-weighted fat-suppressed (a), coronal T1-weighted (b) and T2-weighted fat-suppressed MR images show marrow edema of the mid foot without frank erosion, destruction or disorganization. There is mild adjoining soft tissue edema and an ankle joint effusion.

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**Fig. 2:** Figure 1b: Early neuroarthropathy: Sagittal T2-weighted fat-suppressed (a), coronal T1-weighted (b) and T2-weighted fat-suppressed (c) MR images show marrow edema of the mid foot without frank erosion, destruction or disorganization. There is mild adjoining soft tissue edema and an ankle joint effusion.

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Fig. 3: Figure 1c: Early neuroarthropathy: Sagittal T2-weighted fat-suppressed (a), coronal T1-weighted (b) and T2-weighted fat-suppressed (c) MR images show marrow edema of the mid foot without frank erosion, destruction or disorganization. There is mild adjoining soft tissue edema and an ankle joint effusion.

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Fig. 4: Figure 2a: Advanced neuroarthropathy (Charcot's foot) with Lisfranc's fracture dislocation: AP radiograph of the foot (a) demonstrates disorganization and lateral subluxation of the 1st to 5th tarsometatarsal joints with increased osseous density and some degree of fusion across the joints. Old fractures are also noted at the shafts of the 3rd to 5th metatarsals. Appearances are consistent with Charcot's foot. Axial T2-weighted fat-suppressed (b and c) confirm the findings of joint destruction, however demonstrate absence of bone marrow edema or joint effusion indicative of chronic neuroarthropathic changes and absence of features of osteomyelitis or septic arthritis. However, there is overlying soft tissue edema.

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**Fig. 5:** Figure 2b: Advanced neuroarthropathy (Charcot's foot) with Lisfranc's fracture dislocation: AP radiograph of the foot (a) demonstrates disorganization and lateral subluxation of the 1st to 5th tarsometatarsal joints with increased osseous density and some degree of fusion across the joints. Old fractures are also noted at the shafts of the 3rd to 5th metatarsals. Appearances are consistent with Charcot's foot. Axial T2-weighted fat-suppressed (b and c) confirm the findings of joint destruction, however demonstrate absence of bone marrow edema or joint effusion indicative of chronic neuroarthropathic changes and absence of features of osteomyelitis or septic arthritis. However, there is overlying soft tissue edema.

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Fig. 6: Figure 2c: Advanced neuroarthropathy (Charcot's foot) with Lisfranc's fracture dislocation: AP radiograph of the foot (a) demonstrates disorganization and lateral subluxation of the 1st to 5th tarsometatarsal joints with increased osseous density and some degree of fusion across the joints. Old fractures are also noted at the shafts of the 3rd to 5th metatarsals. Appearances are consistent with Charcot's foot. Axial T2-weighted fat-suppressed (b and c) confirm the findings of joint destruction, however demonstrate absence of bone marrow edema or joint effusion indicative of chronic neuroarthropathic changes and absence of features of osteomyelitis or septic arthritis. However, there is overlying soft tissue edema.

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Fig. 7: Figure 3a: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2-weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which showed several superficial foci of increased radiolabelled WBC accumulation in the plantar aspect of the left mid foot compatible with underlying soft tissue infection. However no uptake was noted in the bones, hence excluding osteomyelitis or septic arthritis.

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**Fig. 8:** Figure 3b: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2-weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which showed several superficial foci of increased radiolabelled WBC accumulation in the plantar aspect of the left mid foot compatible with underlying soft tissue infection. However no uptake was noted in the bones, hence excluding osteomyelitis or septic arthritis.

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Fig. 9: Figure 3c: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2-weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which showed several superficial foci of increased radiolabelled WBC accumulation in the plantar aspect of the left mid foot compatible with underlying soft tissue infection. However no uptake was noted in the bones, hence excluding osteomyelitis or septic arthritis.

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**Fig. 10:** Figure 3d: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2-weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which
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Fig. 11: Figure 3e: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2- weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which showed several superficial foci of increased radiolabelled WBC accumulation in the
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Fig. 12: Figure 3f: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2- weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which showed several superficial foci of increased radiolabelled WBC accumulation in the plantar aspect of the left mid foot compatible with underlying soft tissue infection. However no uptake was noted in the bones, hence excluding osteomyelitis or septic arthritis.
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Fig. 14: Figure 3h: Advanced neuroarthropathy with secondary flat foot deformity: Sagittal T1-weighted (a) and T2- weighted fat-suppressed (b) and gadolinium enhanced images in sagittal (c) and coronal planes (d) show disorganization of the mid-foot joints with cystic foci in the marrow, patchy marrow edema, cortical erosions and foci of enhancement in the tarsal bones. Enhancement is predominantly around the cystic changes and non-specific. Extensive myositis and cellulitis over the mid plantar foot is also noted. Enhancing and bulging inflammatory soft tissue is noted in the mid plantar region with a tortuous sinus tract within (arrows). Triple-phase bone scintigraphy (e,f) was positive on all phases in the tarsal bones. MRI features were compatible with neuroarthropathy however in view of marrow enhancement, relationship of the involved bone to the plantar sinus tract and non-specific bone scan findings, a superimposed osteomyelitis could not be confidently excluded. Hence, a labeled leukocyte scan (g,h) was performed which showed several superficial foci of increased radiolabelled WBC accumulation in the plantar aspect of the left mid foot compatible with underlying soft tissue infection. However no uptake was noted in the bones, hence excluding osteomyelitis or septic arthritis.

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**Fig. 15**: Figure 4a: Osteomyelitis in a patient with a calcaneal ulcer. Coronal T1-weighted (a), T2-weighted fat-suppressed (b) and gadolinium enhanced (c) MR images show a large soft-tissue ulcer (arrows) and an extensive region of altered calcaneal marrow signal deep to the ulcer appearing hypointense on T1-weighted, hyperintense on T2-weighted and showing enhancement on post contrast images.

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Fig. 16: Figure 4b: Osteomyelitis in a patient with a calcaneal ulcer. Coronal T1-weighted, T2-weighted fat-suppressed and gadolinium enhanced MR images shows a large soft-tissue ulcer (arrows) and an extensive region of altered calcaneal marrow signal deep to the ulcer appearing hypointense on T1-weighted, hyperintense on T2-weighted and showing enhancement on post contrast images.

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**Fig. 17:** Figure 4c: Osteomyelitis in a patient with a calcaneal ulcer. Coronal T1-weighted (a), T2- weighted fat-suppressed (b) and gadolinium enhanced MR images (c) shows a large soft-tissue ulcer (arrows) and an extensive region of altered calcaneal marrow signal deep to the ulcer appearing hypointense on T1-weighted, hyperintense on T2-weighted and showing enhancement on post contrast images.

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Fig. 18: Figure 5a: A patient who underwent trans-metatarsal amputation evaluated with MRI and 111In-labeled leukocyte scan. Sagittal T1-weighted (a), Sagittal and axial T2-weighted fat-suppressed (b,c), and axial gadolinium-enhanced T1-weighted fat-suppressed images (d) show an air-fluid level in the surgical defect and granulation tissue along the amputation margins. The cut margins of the amputated metatarsal (thick arrow) show mild T2 signal hyperintensity (thin arrow) suggestive of non-specific edema and could be related to either post-operative change or osteomyelitis. The marked inflammatory changes might be partly due to post-operative changes, however, underlying infection remained a concern. Labeled leukocyte scan (e) shows an area of intense radiotracer activity in the region of soft tissue between the 1st and 4th metatarsal bones of the right foot, indicative of soft tissue infection. However, no bone uptake is noted to suggest osteomyelitis.

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Fig. 19: Figure 5b: A patient who underwent trans-metatarsal amputation evaluated with MRI and 111In-labeled leukocyte scan. Sagittal T1-weighted (a), Sagittal and axial T2-weighted fat-suppressed (b,c), and axial gadolinium-enhanced T1-weighted fat-suppressed images (d) show an air-fluid level in the surgical defect and granulation tissue along the amputation margins. The cut margins of the amputated metatarsal (thick arrow) show mild T2 signal hyperintensity (thin arrow) suggestive of non-specific edema and could be related to either post-operative change or osteomyelitis. The marked inflammatory changes might be partly due to post-operative changes, however, underlying infection remained a concern. Labeled leukocyte scan (e) shows an area of intense radiotracer activity in the region of soft tissue between the 1st and 4th metatarsal bones of the right foot, indicative of soft tissue infection. However, no bone uptake is noted to suggest osteomyelitis.

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Fig. 20: Figure 5c: A patient who underwent trans-metatarsal amputation evaluated with MRI and 111In-labeled leukocyte scan. Sagittal T1-weighted (a), Sagittal and axial T2-weighted fat-suppressed (b,c), and axial gadolinium-enhanced T1-weighted fat-suppressed images (d) show an air-fluid level in the surgical defect and granulation tissue along the amputation margins. The cut margins of the amputated metatarsal (thick arrow) show mild T2 signal hyperintensity (thin arrow) suggestive of non-specific edema and could be related to either post-operative change or osteomyelitis. The marked inflammatory changes might be partly due to post-operative changes, however, underlying infection remained a concern. Labeled leukocyte scan (e) shows an area of intense radiotracer activity in the region of soft tissue between the 1st and 4th metatarsal bones of the right foot, indicative of soft tissue infection. However, no bone uptake is noted to suggest osteomyelitis.
Fig. 21: A patient who underwent trans-metatarsal amputation evaluated with MRI and 111In-labeled leukocyte scan. Sagittal T1-weighted (a), Sagittal and axial T2-weighted fat-suppressed (b,c), and axial gadolinium-enhanced T1-weighted fat-suppressed images (d) show an air-fluid level in the surgical defect and granulation tissue along the amputation margins. The cut margins of the amputated metatarsal (thick arrow) show mild T2 signal hyperintensity (thin arrow) suggestive of non-specific edema and could be related to either post-operative change or osteomyelitis. The marked inflammatory changes might be partly due to post-operative changes, however, underlying infection remained a concern. Labeled leukocyte scan (e) shows an area of intense radiotracer activity in the region of soft tissue between the 1st and 4th metatarsal.
Fig. 22: Figure 5e: A patient who underwent trans-metatarsal amputation evaluated with MRI and 111In -labeled leukocyte scan. Sagittal T1-weighted (a), Sagittal and axial T2-weighted fat-suppressed (b,c), and axial gadolinium-enhanced T1-weighted fat-suppressed images (d) show an air-fluid level in the surgical defect and granulation tissue along the amputation margins. The cut margins of the amputated metatarsal (thick arrow) show mild T2 signal hyperintensity (thin arrow) suggestive of non-specific edema and could be related to either post-operative change or osteomyelitis. The marked inflammatory changes might be partly due to post-operative changes, however, underlying infection remained a concern. Labeled leukocyte scan (e) shows an area of intense radiotracer activity in the region of soft tissue between the 1st and 4th metatarsal bones of the right foot, indicative of soft tissue infection. However, no bone uptake is noted to suggest osteomyelitis.
Fig. 23: Figure 6a: Septic arthritis (a,b,c): A deep ulcer is seen overlying the 2nd metatarsal head with articular erosions, synovial enhancement, joint effusion and perisynovial soft tissue edema at the metatarsophalangeal joint consistent with septic arthritis. Marrow edema and enhancement is noted within the 2nd metatarsal bone and proximal phalanx of the second toe.

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Fig. 24: Figure 6b: Septic arthritis (a,b,c): A deep ulcer is seen overlying the 2nd metatarsal head with articular erosions, synovial enhancement, joint effusion and perisynovial soft tissue edema at the metatarsophalangeal joint consistent with septic arthritis.
arthritis. Marrow edema and enhancement is noted within the 2nd metatarsal bone and proximal phalanx of the second toe.

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Fig. 25: Figure 6c: Septic arthritis (a,b,c): A deep ulcer is seen overlying the 2nd metatarsal head with articular erosions, synovial enhancement, joint effusion and perisynovial soft tissue edema at the metatarsophalangeal joint consistent with septic arthritis. Marrow edema and enhancement is noted within the 2nd metatarsal bone and proximal phalanx of the second toe.

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**Fig. 26:** Figure 7: Cellulitis and myositis: Sagittal T2-weighted fat-suppressed MR image shows features of cellulitis and myositis on both plantar and dorsal aspects of foot.

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**Fig. 27:** Figure 8a: Callus and ulcer. Sagittal T1-weighted (a), T2-weighted fat-suppressed (b), and gadolinium-enhanced T1-weighted fat-suppressed images show Charcot’s foot with Rocker bottom foot deformity. Callus formation and ulceration (arrows) is noted at the pressure point.

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**Fig. 28:** Figure 8b: Callus and ulcer. Sagittal T1-weighted (a), T2-weighted fat-suppressed (b), and gadolinium-enhanced T1-weighted fat-suppressed images show Charcot's foot with Rocker bottom foot deformity. Callus formation and ulceration (arrows) is noted at the pressure point.

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**Fig. 29:** Figure 8c: Callus and ulcer. Sagittal T1-weighted (a), T2-weighted fat-suppressed (b), and gadolinium-enhanced T1-weighted fat-suppressed images show Charcot's foot with Rocker bottom foot deformity. Callus formation and ulceration (arrows) is noted at the pressure point.
**Fig. 30:** Figure 9a: Dry gangrene. Coronal T2-weighted fat-suppressed (a) and gadolinium-enhanced T1-weighted fat-suppressed (b) images show an area of soft tissue thickening and T2 high signal on the plantar aspect of 1st metatarsal with non enhancement on post contrast images suggestive of an area of devitalized tissue (arrow).
Fig. 31: Figure 9b: Dry gangrene. Coronal T2-weighted fat-suppressed (a) and gadolinium-enhanced T1-weighted fat-suppressed (b) images show an area of soft tissue thickening and T2 high signal on the plantar aspect of 1st metatarsal with non enhancement on post contrast images suggestive of an area of devitalized tissue (arrow).

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Fig. 32: Figure 10a: Wet gangrene. Sagittal T1-weighted (a), T2-weighted fat-suppressed (b), and gadolinium-enhanced T1-weighted fat-suppressed (c) images of the great toe show loss of the normal marrow signal in the distal phalanx of the great toe with gas within the partly eroded bone and in the overlying soft tissues along with inflammatory changes. These findings are consistent with gas gangrene in this region.

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Fig. 33: Figure 10b: Wet gangrene. Sagittal T1-weighted (a), T2-weighted fat-suppressed (b), and gadolinium-enhanced T1-weighted fat-suppressed (c) images of the great toe show loss of the normal marrow signal in the distal phalanx of the great toe with gas
within the partly eroded bone and in the overlying soft tissues along with inflammatory changes. These findings are consistent with gas gangrene in this region.

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**Fig. 34:** Figure 10c: Wet gangrene. Sagittal T1-weighted (a), T2-weighted fat-suppressed (b), and gadolinium-enhanced T1-weighted fat-suppressed (c) images of the great toe show loss of the normal marrow signal in the distal phalanx of the great toe with gas within the partly eroded bone and in the overlying soft tissues along with inflammatory changes. These findings are consistent with gas gangrene in this region.

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Conclusion

Awareness of the various imaging findings of a diabetic foot, their relevance to the therapeutic decisions and correct usage of various imaging modalities to answer pertinent clinical questions are very important to reduce complications and prevent amputation.
References


