MR Imaging of the Diabetic Foot

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KEYWORDS

• MR imaging • Diabetic foot • Osteomyelitis • Septic arthritis • Neuropathic osteoarthropathy • Ulcer • Abscess

KEY POINTS

- Osteomyelitis occurs from direct inoculation in most cases, and identification of a skin defect should be the first step in evaluation of all diabetic feet.
- T2 hyperintensity and T1 hypointensity are required for the diagnosis of osteomyelitis. T2 hyperintensity on its own likely represents osseous stress response.
- Osteomyelitis tends to occur distal to the tarsometatarsal joints and in the malleoli and calcaneus.
- Neuropathic osteoarthropathy tends to be centered at the Lisfranc, Chopart, or metatarsophalangeal joints.
- Imaging findings suggestive of superimposed infection in neuropathic osteoarthropathy are ghosting of bones (indistinct on T1, but present on T2 or T1 postcontrast studies), disappearance of subchondral cysts, and greater-than-expected fluid collections.

INTRODUCTION/CLINICAL PRESENTATION

Diabetic patients develop injury and progressive diseases of the foot from numerous sources, including disease of the peripheral nervous, vascular, and immune systems. There is frequently significant overlap between these issues, with one-third of all diabetic patients having a mixed neuropathic-ischemic foot ulcer.[1] Sensory, motor, and autonomic nervous system problems arise in the setting of chronic hyperglycemia. Sensory neuropathy results in the inability to adapt to mechanical stresses with resultant soft tissue ulceration and articular structural disruption. Autonomic neuropathy deregulates perspiration, skin temperature, and arteriovenous shunting resulting in excessive callus formation and skin cracking. Motor neuropathy results in intrinsic muscle dysfunction or, less commonly, a single nerve defect, most frequently involving the common peroneal nerve. Diabetic patients have both large and small vessel ischemia. This ischemia is worsened by coexisting vascular risk factors, including smoking, hypertension, and hyperlipidemia. It is often refractory to revascularization of the larger vessels because of the extent of microvessel disease. Diabetes also inhibits the activity of polymorphonuclear leukocytes, reducing cellular immune responses. Collagen and keratin formation is also impaired.[2] The primary role of imaging is to identify and delineate the sequelae of these systemic processes, including soft tissue infection, abscess formation, osteomyelitis, and the neuropathic joint. Prompt identification and accurate diagnosis are important for limb-sparing treatment planning.[3]

IMAGING THE DIABETIC FOOT

The first-line examination of the diabetic foot is conventional radiographs, which should be performed in at least 3 planes and optimally 4. Relevant radiographic findings that should be observed include

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soft tissue swelling, radiopaque foreign bodies, cortical disruption/destruction, periostitis, joint incongruity, arterial calcification, and prior amputation. Radiographs can also be a beneficial adjunct in the evaluation of complex midfoot disruption. However, radiographs are insensitive to early osteomyelitis and notoriously underestimate the extent of osseous infection.[4] Ultrasound may be used to evaluate soft tissue processes, such as abscess formation and tenosynovitis, and to locate radiolucent foreign bodies. However, this modality is limited in evaluating underlying bone and is also extremely user dependent. Triple phase bone scans that should be positive on all 3 phases (angiographic, blood pool, and delayed) in the setting of osteomyelitis are sensitive for osseous activity but not specific.[5,6] Scintigraphic studies may be positive in other processes with high bone turnover, such as injury and neuropathic osteoarthropathy, and even osseous stress response.[7] Labeled white blood cell scans have an increased sensitivity over bone scans; but the major limitation of nuclear medicine is the poor anatomic resolution, thus limiting the usefulness of these studies as a preoperative road map.[8] MR imaging has emerged as the dominant imaging modality in the assessment of the diabetic foot, particularly the infected diabetic foot. It has high sensitivity (90%) and specificity (83%) for the diagnosis of osteomyelitis.[9,10] Furthermore, it has the added benefit of providing good anatomic definition, allowing it to serve as an appropriate road map for surgical resection.

MR IMAGING SCAN PROTOCOLS

The MR imaging examination should be tailored to the site of suspected abnormality. The authors divide the diabetic foot examination into either the ankle, including the ankle and hindfoot, or the foot, including the midfoot and forefoot. This designation allows for focused, smaller field-of-view imaging for the precise area of concern. Late-model multichannel ankle/foot receiver coils can provide high-resolution imaging from the ankle through the forefoot with a single acquisition, but prescription of imaging planes becomes difficult in this scenario. Most commercial payers still accept foot and ankle MR imaging examinations as distinct procedures; there are distinct *Current Procedural Terminology* codes: 73,718 and 73,720. The field of view for either examination can easily be tailored to the location of clinical concern.

As a standard protocol, with the use of dedicated extremity receiver coils, 2 sets of acquisitions are obtained in each plane. T1-weighted non–fat-suppressed imaging is performed in at least 2 planes to evaluate the bone marrow and the subcutaneous soft tissues. For these sequences, traditional spin echo is ideal; but multiecho acquisitions with a short echo train are adequate. Fat suppressed, fast spin echo/turbo spin echo T2-weighted images are used to evaluate for edema and fluid signal. A short tau inversion recovery (STIR) sequence is recommended in at least one plane (generally sagittal) to mitigate potential near field homogeneity artifacts. Noncontrast examinations are almost always diagnostic; given the great frequency of renal disease in diabetic patients, contrast is rarely administered. When necessary and feasible, precontrast and postcontrast fat-suppressed, T1-weighted, fast gradient-echo sequences can be used to better delineate sinus tracts and abscess cavities and to identify devitalized/necrotic tissue.[11,12] Dynamic contrast runs can be helpful in some cases, as the rate of enhancement can be measured and compared between normal tissues and devitalized tissues. To date, 1.5 T is still considered the imaging standard. Imaging at 3 T offers theoretic advantages, with shorter imaging times and/or higher resolution; but it is also prone to more artifacts and signal homogeneity issues.

MR IMAGING FINDINGS AND DIAGNOSTIC CRITERIA IN THE DIABETIC FOOT

Soft Tissue Edema, Cellulitis

Skin thickening and edema (T1 hypointensity and T2 hyperintensity) are findings found in both soft tissue edema and cellulitis. Enhancement on postcontrast imaging is a characteristic feature of cellulitis. Furthermore, skin thickening and edema in the vicinity of soft tissue ulcer or abscess should raise suspicion of focal cellulitis rather than bland soft tissue edema.

Callus, Ulcer

Callus is a focal, masslike infiltration of the subcutaneous fat, seen as hypointense T1 and intermediate T2 signal.[13] Callus enhances on T1-weighted postcontrast imaging.[14] Typical locations for callus formation include beneath the first and fifth metatarsal heads and the distal phalanx of the hallux in the forefoot. In the midfoot, callus forms deep to the cuboid in patients with rocker bottom deformities and at the heel in the hind foot.[15,16] Chronic friction at the site of callus can lead to the formation of overlying adventitial bursitis, which appears as a thin, linear, T2 fluid collection.[13] Ulcers typically result from the breakdown of callus. Identifiable skin defects and heaped margins will allow differentiation of these two entities (**Fig. 1**). Unlike callus, ulcers are T2 hyperintense. This high T2 signal is secondary to granulation tissue at the base and