Osteomyelitis: Pathology and MRI findings – with Attention to the Foot

One night when I was on call as a radiology resident, a young mother traveling from Antigua brought in her child. Her son had taken ill with fever in Antigua and had progressively worsened with a mysterious ailment. She bundled him up that day, boarded a United States bound plane, and was told by the flight attendant to try Boston. This being prior to 9/11, she arrived at Logan Airport and hailed a cab. The cab driver told her to go to Children's Hospital Boston, where mother and child waited for radiographs near a Red Sox player. The child’s chest film showed septic emboli; his femur, metaphyseal osteomyelitis.

Osteomyelitis can affect all age groups. It can mimic other diagnoses, presenting with symptoms either protean or prosaic. This quarter’s MRI Mentor reviews the pathology and MRI presentation of osteomyelitis.

1. What are the pathologic findings in osteomyelitis?

a) Osteomyelitis denotes bone and marrow inflammation, most commonly used to imply infection. Any type of organism can be the offender: bacteria, viruses, parasites, and fungi, with the most common being pyogenic bacteria and mycobacteria. Pyogenic osteomyelitis may originate from hematogenous spread, contiguous spread, or direct implantation, and Staphylococcus aureus causes 80-90 percent. Even trivial incidents such as vigorous chewing or a small skin wound can cause the bacteremia. (1)

b) The location of osteomyelitis within the bone varies with age due to vascular circulation differences. In the neonate, metaphyseal blood vessels penetrate the growth plate, and infection commonly involves the metaphysis, epiphysis, or both. In children, metaphyseal osteomyelitis predominates. After growth plate closure, bacteria can seed the epiphysis and subchondral region. (1)

c) The morphology of pyogenic osteomyelitis depends on the location and stage: acute, subacute, and chronic.

   i) Bacteria proliferate in bone, causing inflammation and cell death.

   ii) Entrapped bone becomes necrotic within 48 hours; the process spreads in the shaft and may reach the periosteum.

   iii) Since periosteum is loosely attached in children, large subperiosteal abscesses may form and spread.

   iv) Lifted periosteum impedes blood supply, and resultant dead bone is called sequestrum.

   v) Ruptured periosteum can lead to soft tissue abscess and a draining sinus.

   vi) In infants, epiphyseal infection can spread into the joint through the articular surface or along the capsule, tendons, or ligaments.
vii) With time, the host response can lead to a reactive sleeve of bone deposition; when formed around dead bone, it is called an involucrum.

viii) A small intraosseous abscess that involves cortex, surrounded by reactive bone, is called a Brodie abscess. (1)

d) The morphology of tuberculous osteomyelitis:

i) Typically results from hematogenous spread of active visceral disease.

ii) Can result from direct extension or lymphatic spread.

iii) May fester for years prior to discovery.

iv) Occurs most commonly in the thoracic and lumbar spine, followed by the knees and hips. (1)

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Above: History of diabetes with chronic ulcer plantar foot and osteomyelitis distal 3rd metatarsal and proximal 3rd phalanx. Periosteal inflammation extends along the shaft of the 3rd metatarsal. Ulcer plantar to the 3rd metatarsal with ulcer tract that leads to the osteomyelitis in the 3rd metatarsal head.

a. Sagittal T1 weighted image shows effacement of T1 signal in the 3rd proximal phalanx.

b. Sagittal T1 weighted image shows effacement of T1 signal in the 3rd distal metatarsal.

c. Coronal STIR shows periosteal inflammation and medullary edema extending along the shaft of the 3rd metatarsal.

d. Axial post contrast T1 fat suppressed image shows skin ulcer and sinus tract coursing to the osteomyelitis.
2. How do the pathologic changes translate as MRI findings?

a) Infection changes the ratio of free water to bound water, prolonging T1 and T2 relaxation times. This leads to low signal on T1 and increased signal on T2 weighted images. The location, morphology, and severity of signal change play roles to increase diagnostic specificity. (2)

3. How is osteomyelitis diagnosed by MRI in the ProScan experience?

a) A search pattern that concentrates on knowledge of anatomic sites at risk, characteristic MRI signal changes, and appearance of ancillary findings is important. Our practice pattern at ProScan is to perform noncontrast MRI in most patients with a clinical question of osteomyelitis. Our interpretation search pattern pays particular attention to the signal characteristics of medullary bone on water weighted fat suppressed sequences to locate abnormal osseous edema; then we use standard nonfat suppressed T1 images to characterize it.

b) The site of anatomic risk varies by patient age and medical history. In particular settings, such as the diabetic foot, some characteristic sites occur, as will be described later. Osteomyelitis characteristically shows abnormal osseous edema in the medullary bone so coalescent that its T1 signal matches that of cortical bone, resulting in it effacing or erasing the bone signal. The T1 signal appearing this decreased therefore provides the most important clue to the diagnosis. Ancillary tip-offs such as skin ulceration, sinus tracts, and soft tissue edema or collections are helpful.

c) In patients with foot neuroarthropathy, the ProScan practice pattern is to give contrast as needed to help assess for a sinus tract that may course from a skin ulcer to the bone or to help differentiate bone abscesses from pseudocysts. Contrast will also be given in other patients, as needed, to help assess abnormal soft tissue edema for drainable collections or tracts and bone lesions for the rim enhancement of an abscess.

Left: History of diabetes with osteomyelitis of the 4th metatarsal and proximal phalanx, with septic 4th MTP joint. Severe diabetic amyotrophy.

a. Sagittal T1 weighted image shows effacement of the medullary bone signal in the 4th metatarsal.

b and c: T1 weighted axial images show effacement of the medullary bone signal in the 4th metatarsal. Diabetic amyotrophy is present.

d. Coronal T1 shows effacement of the medullary bone signal in the 4th metatarsal and proximal toe phalanx.
4. What are the diagnostic criteria for diagnosing osteomyelitis by MRI according to the literature?

a) Low T1 signal medullary bone in a geographic confluent pattern, concordant with fat-suppressed T2 and T1 weighted postcontrast abnormal signal, was reportedly present in 100 percent of surgically proven cases of foot osteomyelitis. If the T1 decreased signal was in a subcortical distribution or showed a hazy, reticulated pattern, regardless of the fat suppressed T2 or postcontrast T1 imaging appearance, none of the patients had surgically proven osteomyelitis. (3)

b) Decreased T1 medullary marrow signal in a confluent pattern correlates highly with foot osteomyelitis. (4)

c) Differentiating between hematogenous osteomyelitis and bone tumor can be difficult. The “penumbra sign,” a thin layer of granulation with higher signal lining the abscess cavity on T1 weighted images along with high C-reactive protein, was reported as supportive of osteomyelitis rather than tumor. (5)

d) The “penumbra sign” on T1 weighted images can show a high specificity for musculoskeletal (MSK) infection. (6)

e) Although rare, an extra-osseous fat fluid level, especially when accompanied by spongy bone destruction, is considered specific for osteomyelitis. (7)

5. What role does MRI play in the diabetic foot at risk for osteomyelitis? How is diabetic foot neuropathy distinguished from osteomyelitis?

a) MRI is regarded as the imaging method of choice for pedal osteomyelitis. It shows the extent of bone and soft tissue infection and may help limit extent of resection. To evaluate for the presence of osteomyelitis, follow the ulcer or sinus tract’s path to the bone and assess the bone marrow appearance. Findings seen in osteomyelitis consist of low T1 and increased T2 marrow signal and marrow enhancement. Additional signs include periosteal reaction, subtending skin ulcer, sinus tract, cellulitis, abscess, and foreign body. An important distinguishing feature of osteomyelitis compared to neuroarthropathy is the location of marrow abnormality. Osteomyelitis more often occurs distal to the tarsometatarsal joint, in the calcaneus, and malleoli. Neuroarthropathy most commonly involves the tarsometatarsal and metatarsal phalangeal joints. Differentiating midfoot osteomyelitis from neuroarthropathy is helped by assessing for secondary signs of infection. (8)
b) Diabetic related foot osteomyelitis occurs almost exclusively due to contiguous spread of infection from skin ulceration, compared to neuroarthropathy, which occurs in a primarily articular distribution. The MRI specificity for osteomyelitis may be limited when neuroarthropathy is present. However, when a patient has neuropathy and an ulcer that extends to the bone, there is a greater likelihood of osteomyelitis than in patients without neuroarthropathy, and MRI helps delineate the extent of disease. According to Donovan et al, bones that “disappear” on T1-weighted images and “reappear” (become morphologically more distinct) on T2-weighted images or contrast (the ghost sign) probably have osteomyelitis. (8)

c) In the diabetic foot, osteomyelitis must be distinguished from reactive marrow edema of neuropathy. Osteomyelitis and reactive marrow have increased T2 signal, but osteomyelitis is confirmed by T1 hypointensity while reactive change has isolated T2 hyperintensity. To differentiate osteomyelitis from neuropathy, if findings show a localized or contiguously spreading forefoot focus of abnormal bone marrow beyond the subchondral surface, adjacency to a skin ulcer, cellulitis, abscess, or sinus tract, these would indicate osteomyelitis. A diagnosis of neuroarthropathy is supported by a midfoot, subchondral, periarticular, or polyarticular distribution of findings that lack a contiguous focus of skin ulceration. Features that correlate with acute infection coexisting with neuroarthropathy are diffuse bone marrow abnormal signal, subarticular progressive enhancement, loss of subchondral cysts, and existence of MRI “ghost sign.” (9)

d) An MRI specific feature of foot osteomyelitis was reported as a confluent pattern of decreased T1 signal in the medullary bone. When the bone edema was subcortical or a hazy reticulated pattern, none of the cases were osteomyelitis. (10)

e) Neuropathic marrow changes are not hyperintense on T2* GRE. If marrow is hyperintense on T2* it has high association with osteomyelitis, as it indicates increased free water. (11)

Above: Pain since surgery. Osteomyelitis 5th metatarsal head with septic arthritis and abscess lateral to the MTP joint.

a. Coronal T1 weighted image shows erasure of the 5th metatarsal head and infection extending into the MTP joint.

b. Axial STIR showing marked 5th metatarsal medullary and periosteal, and soft tissue edema.

c. Axial T1 weighted fat suppressed post contrast image shows nonenhancing abscess collection in the soft tissues lateral to the infected 5th metatarsal.
f) A summary of imaging foot findings prevalent in 85 diabetic patients with radiographic and MRI feet exams found: 18.9 percent with vascular changes (infarcts or necrosis), 22.2 percent traumatic changes (edema, occult fracture), 8.9 percent had bone destruction, dislocation (12.2 percent), osteomyelitischondritis (26.7 percent), and osteomyelitis (11.1 percent). (12)

g) Midfoot and hindfoot MRI bone marrow edema proves common in the feet of people with diabetes and neuropathic ulceration. It has been reported as often transient and of unknown significance (13)

h) Collection of microbiologic data using deep swabs and transcutaneous bone biopsy are considered ideal to aid treatment of diabetic foot infection. (14)

i) A study of diabetics who underwent conservative management of neuropathic forefoot ulcers with underlying osteomyelitis found that use of MRI showed association with long courses of antibiotics, but particularly low relapse rates. (15)

j) Diagnosing diabetic foot osteomyelitis had association with elevated ESR > or = 65 mm/h together with wound size > or = 2 cm2. (16)

k) Pooled sensitivity and specificity data respectively for osteomyelitis imaging tests in patients with diabetic foot ulcers found: plain radiography 0.54 and 0.68, MRI 0.90 and 0.79, bone scan 0.81 and 0.28, and leukocyte 0.74 and 0.68. (17)

l) A review of English language articles from 1966 to March 2007 reported an 89 percent overall accuracy of MRI to diagnose osteomyelitis in the diabetic foot. (19) An ulcer area larger than 2 cm2 and a positive “probe-to-bone” test proved the best clinical findings. An ESR of greater than 70 mm/h increases the probability of osteomyelitis. Abnormal radiographs doubled the odds of osteomyelitis. (18)

m) In a study of 158 patients, abscesses, predominantly in the forefoot, were revealed in 18 percent of MRI studies of patients suspected of having foot osteomyelitis. (19)

**Conclusion:** Osteomyelitis typically appears on MRI as geographic low T1 medullary signal so coalescent that it matches cortical bone, essentially erasing bone signal. Ancillary tip-offs like skin ulceration, sinus tracts, and soft tissue edema or collections may be present. In patients with foot neuroarthropathy, contrast can assist in certain cases to show a sinus tract leading from a skin ulcer to bone or to differentiate bone abscess from a pseudocyst. Contrast may help interrogate soft tissue edema for drainable collections and bone lesions for abscess related rim enhancement.

**Sources**


This issue is dedicated in loving memory of Miss Gable Deception and Mr. Paul Pomeranz.