Abstract

Osteomyelitis in the Diabetic Foot: Can you be confident??

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Target audience: – Practicing Radiologists

Learning Objectives:

- Discussion of the range of pedal problems in diabetics
- Recommended imaging modalities for assessing the pedal diabetes
- Discussion of Diabetic pedal cohorts by ANATOMIC LOCATION, Chronology/STAGE of neuropathic lesions, PATTERNS of plantar arch collapse
- Tips for improving confidence in interpreting images of the ulcerated diabetic foot
- New treatment strategies

Diabetes presents a public health crisis, affecting > 7% of the US population; the number of Americans with diabetes is expected to double by 2030.

Musculoskeletal complications of diabetes result from:

- (1) Abnormal collagen deposition in periarticular connective tissues, with greater frequency of carpal tunnel, Dupuytren's contracture, adhesive capsulitis, DISH
- (2) Macrovascular disease: muscle and bone infarcts
- (3) Microvascular disease resulting in neuropathy. Both macro-and microvascular disease and poor neutrophil chemotaxis potentiate infection, especially in feet.

Management of the infected diabetic foot has classically been amputation, with 3 year survival @ 50% and >50% contralateral foot amputation by 4 years. These poor outcomes challenge surgeons to revise their practice philosophies, with amputation increasingly a last resort. Radiologists are also challenged to provide accurate roadmaps of devitalized and infected tissue, Overlap of MRI findings with Charcot and infection limits certainty. Many surgeons are inured to radiologists' overcalls in diabetic feet.

ANATOMY: By location. Forefoot ulcers/infection are seen in vasculopaths. Mid/ hindfoot involvement is seen more in neuropaths.

Vasculopathy: Diabetics are 20 x more likely to have peripheral vascular disease, with a cold, pale foot, absent pulses. Small painful ulcers are seen mostly at the toes and 1st and 5th metatarsal heads. These cases are often managed by vascular surgeons. Imaging: Angiography/MRA. Treatment: revascularization or amputation.

Neuropathy—mid- and hindfoot. Insensate foot with poor proprioception In contradistinction to the vasculopathic foot, the acute neuropathic foot is warm, red and swollen, with bounding pulses, vasodilatation, and hyperemia Intact vascularity is required to develop pedal Charcot

STAGES: Neuro-arthropathy occurs in stages that can be evaluated on radiographs and MRI

- A. Clinical neuropath (pre-Charcot) Loss of sensation to filament testing, without fractures or arch collapse. On MRI there is muscle, subcutaneous fat and marrow edema due to hyperemia. B. Acute Charcot: clinically mimics cellulitis/osteomyelitis.
 - Progression to acute Charcot/neuroarthropathy is not inevitable.

- Seen more in advanced age, poor glycemic control. Arch collapse can be gradual, or precipitous
- MRI: extensive marrow edema and fractures in an articular distribution. Soft tissue & marrow hyperemia is often self-limiting

Management of an acute Charcot episode includes off-loading the foot with bracing, total contact casting.

C. Sub-acute to chronic Charcot: bony debris, fragmentation, joint derangement In stabilized Charcot the soft tissue and marrow edema eventually abates. We see callus, OA/subchondral cysts, pseudoarticulations--quiescent on MRI, but the developing fracture fusion mass can predispose to ulcer formation.

<u>PATTERNS</u> of collapse: 4 types, by location, with subsets by degree of severity (Lew Schon). Describes mid- and hind-foot Charcot changes, predicts outcome, useful for surgical planning.

A Lisfranc

B Naviculo-cuneiform

C Peri-navicular

D Transverse tarsal

Mixed pattern: Vasculopath s/p revascularization, developing Charcot.

ACR Appropriateness Guidelines for Suspected Pedal Osteomyelitis in DM. X-ray and MRI are indicated and complimentary. Other modalities including scintigraphic techniques, CT ultrasound, PET CT are less recommended.

Imaging: Standing lateral X-ray with weight bearing line. Survey for bony excrescence that extends plantar to this line.

MRI provides a surgical road map. Sensitivity 77-100 %. Specificity 79-100% T2/IR is sensitive but less clinically helpful. T1 most specific, but limited by neuroarthropathy, RA, prior intervention. Primary MRI signs for osteomyelitis are hypo/isointense T1SI = replaced marrow and cortical effacement

The use of Gadolinium to evaluate osteomyelitis is controversial. *Enhancement of marrow* does not raise specificity (use T1). Gadolinium helps with secondary supporting signs including ulcers, tracts, abscesses and helps visualize devitalization/soft tissue necrosis (false negatives>false positives). However, GFR considerations may militate against its use in many cases

Changing surgical approaches for Charcot

If non-infected, reshaping the rocker-bottom foot with arthrodesis, internal fixation. If ulcerated/infected, external fixation cages. Goal is salvage / stability, not motion.

SUMMARY:

Bone marrow edema in pedal diabetes is nonspecific. It can be seen with altered mechanics, ongoing collapse and super-infected Charcot: T1is more specific for osteomyelitis than T2. It should be stressed that: >90% of pedal infection is by transcutaneous route. Look for the ulcer and track to the exostosis under the ulcer. If no ulcer—almost always, no osteomyelitis, despite marrow edema.

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