Charcot foot: Imaging insights from the High Risk Diabetic Foot MDT

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Learning objectives

The purpose of our educational exhibit is to:

1. Present key features of Charcot neuropathic osteoarthropathy (CN) and lessons learned from the High Risk Diabetic Foot (HRDF) Multidisciplinary Team (MDT) collaboration at our institution.

2. Emphasise the central role of the radiologist in facilitating early diagnosis and expediting management of this potentially devastating disease.

Background

Charcot neuropathic osteoarthropathy, commonly known as Charcot foot, is a progressive disease affecting the bones, joints and soft tissue, especially of the foot and ankle, most commonly associated with diabetic peripheral neuropathy. Delay in the diagnosis of CN can lead to disruption of the bony architecture of the foot, deformity, recurrent foot ulceration, cellulitis, osteomyelitis and ultimately, amputation [1]. Furthermore, patients with diabetes complicated by Charcot foot have especially high morbidity, frequency of hospitalisation and therefore, significant utilization of expensive medical resources [2]. Mortality has also been reported to be higher in patients presenting with acute CN than diabetes alone [3].

Population-based studies report an incidence of CN in the range of 0.1% to 29% in the diabetic population [2]. However, it is likely the condition has been underreported because of the lack of clear clinical and radiological diagnostic criteria. Importantly, a significant number of patients experience a delayed or missed diagnosis [2]. In a recent study, the diagnosis of acute Charcot was missed in 19 of 20 patients prior to specialist referral [4].

Early diagnosis and intervention are essential for the prevention of debilitating structural deformities of the foot among CN patients. The diagnosis of CN is challenging, especially in the initial stages of the disease process. CN lacks precise diagnostic markers, and diagnosis is based on clinical presentation supported by various imaging modalities [5]. Therefore the radiologist plays a pivotal role in facilitating early diagnosis and expediting management of this potentially devastating disease. At our institution, the HRDF MDT comprises senior clinicians from a number of relevant disciplines and meets weekly to review HRDF patients and expedite management.
Findings and procedure details

Key learning point 1: The Charcot foot has well preserved arterial blood supply

The current consensus is that a number of factors and precipitating events leads to the development of CN. Once the disease is initiated, it is thought to be mediated through an inflammatory cascade.

- A triggering factor (such as a minor trauma, previous ulcer infection or foot surgery) may go unnoticed because of the sensory deficit [6, 7].
- This induces the release of proinflammatory cytokines that increase the expression of receptor activator of nuclear factor kappa-B ligand (RANKL) [6, 7].
- The overexpression of RANKL results in inflammation, osteoclast maturation and activation, osteolysis and osteopaenia [7]. Osteolysis is responsible for the progressive bone destruction that characterises the condition [6].
- In the presence of peripheral neuropathy, reduced pain perception allows the inflammatory process to continue resulting in the establishment of a vicious cycle [7].

In addition to an established (profound) peripheral neuropathy, relatively intact arterial supply is a feature of the affected limb:

- Patients with peripheral arterial disease are less likely to develop CN probably due to limitation of the inflammatory response as a result of relative ischaemia [8].

Key learning point 2: CN is not a fait accompli

- CN may present as an acute active condition (Fig. 1 on page 6) or as a chronic inactive disease (Fig. 2 on page 8).
- In both circumstances, accurate clinical and radiologic assessment is vital to initiate prompt and appropriate treatment.
- Diagnosis of early CN followed by expeditious treatment - early offloading and total contact casting (Fig. 3 on page 8) - can be critical in preventing progression to debilitating subsequent stages.

Key learning point 3: The classic radiographic "five D's" are late findings

- "The five D's" described by Rajbhandari and colleagues [9] are joint distension, dislocation, debris, disorganisation and increased density (Fig. 4 on page 9).
• They are late findings seen on plain radiographs and not useful for diagnosis of Charcot foot in the acute phase [10].

**Key learning point 4: CN is not necessarily confined to the midfoot**

CN can involve the:

• Forefoot
• Midfoot
• Subtalar and ankle joints (Fig. 5 on page 11)
• Knee (Fig. 6 on page 11), wrist, spine and shoulder [10].

**Key learning point 5: Subchondral osteopaenia is an early radiographic finding**

CN is most commonly staged using the Eichenholtz classification system, which encompasses the sequence of changes on plain radiographs [11, 12]:

• Eichenholtz Stage 1 (development) is characterised by bone resorption, bone fragmentation and joint dislocation. Clinically, there is warmth, oedema and erythema.
• The earliest finding on plain radiographs is focal bone demineralisation (Fig. 7 on page 12) [13].
• Eichenholtz Stage 2 (coalescence) demonstrates decreasing warmth, oedema, erythema and radiographic appearance of absorption of fine debris and fusion of large fragments to adjacent bones, and/or new periosteal bone formation.
• Eichenholtz Stage 3 (reconstruction) is characterised by remodelling and rounding of bone ends without the inflammatory signs of previous stages.
• Final consolidation of fractured bone indicates that the deformity (for example, subluxation, incongruity, and dislocation) is permanent.

**Key learning point 6: MRI is the most useful modality in assessing early CN and late complications**

MRI is most useful in:

1. Evaluating tendons and ligaments ('internal derangements') (Fig. 8 on page 14);
2. Assessing for deep infection complicating CN, such as soft tissue abscess, septic arthritis and osteomyelitis.
3. Establishing the diagnosis early in the disease:
• Shibata and colleagues proposed a Stage 0, also called 'Charcot in situ' or 'pre-stage 1', in addition to the three stages of the Eichenholtz classification [14].
• Stage 0 is characterised by erythema, oedema, and heat but minimal if any abnormality present on x-ray [11].
• Early diagnosis with MRI may prove to be critical in preventing progression to debilitating subsequent stages [12].
• In Stage 0, MRI typically reveals subchondral bone marrow oedema with or without microfracture [1] (Fig. 9 on page 14).
• Early offloading and immobilisation in patients with Charcot Stage 0 has proven to stop the disease activity and prevent major foot deformities [15].

Key learning point 7: MRI is accurate in demonstrating osteomyelitis on a background of CN

• Deformity and collapse of the midfoot, and particularly descent of the cuboid, in an ambulatory patient predisposes to pressure ulceration (Fig. 10 on page 15), with risk of infection, deep penetration (sinus tract) and osteomyelitis.
• The presence of the 'ghost sign' is particularly helpful in identifying superimposed infection [13].
• The 'ghost sign' (Fig. 11 on page 17) is characterised by the presence of profound low signal in T1-weighted images with loss of definition of margins of the affected bone, which becomes clear after contrast administration [13].

Key learning point 8: Computed tomography is indicated when fine detail of the bony midfoot architecture is required

• Computed tomography (CT) has superior sensitivity for early intra-articular fractures not readily visualised by plain radiography [11] (Fig. 12 on page 17).
• CT can also show periosteal new bone formation and small foci of gas within bone better than MRI.
• Multidectector CT with the addition of multiplanar and three-dimensional reformatted images is most useful in preoperative planning and follow-up of immobilisation therapy.

Key learning point 9: Stress fractures and osteonecrosis are less well recognised bone abnormalities seen in association with, or secondary to CN

• These bone abnormalities are often overlooked, and develop as a result of markedly altered biomechanics from Charcot foot in a weight-bearing patient and/or obesity (Fig. 13 on page 18).
Key learning point 10: A multidisciplinary team approach has a positive effect in CN

- Our HRDF Unit is a multidisciplinary team comprising of consultants and trainees in Vascular Surgery, Radiology, Endocrinology, and Podiatrists.
- The HRDF MDT meeting (Fig. 14 on page 19) is conducted weekly at midday on a Wednesday. Preceding this, there is a combined ward round attended by representatives from all groups of the MDT, where all inpatients are reviewed. Digital clinical photographs are obtained (with patient consent) of problematic ulcers and sinuses as well as complicated postoperative wounds.
- Immediately following the MDT meeting, the HRDF Outpatient Clinic is conducted. All relevant information regarding outpatients is also presented at the MDT meeting.
- At the MDT meeting, the clinical details of each patient are presented, along with a clinical photograph if appropriate, and this is reviewed in conjunction with relevant radiology - usually plain radiographs, MRI, nuclear medicine bone scans and vascular imaging which may entail one or more of arterial duplex ultrasound, CT angiography, digital subtraction angiography or MR angiography.
- All members of the MDT are availed of all information provided. There is a fully informed discussion, and a treatment plan is formulated for inpatients, as well as outpatients; the latter group being reviewed in clinic immediately after the MDT meeting.
- Because of the way the entire day is structured, the fact that senior clinicians from key specialties are in attendance at ward round, MDT meeting and clinic, all information is presented concurrently and fully informed decisions can be made in collaboration, we have observed far superior outcomes without the undue delay incurred from "clinical unit to clinical unit" or "anonymous clinical unit to radiology" referrals, particularly when this occurs at a junior level.

Images for this section:
**Fig. 1:** Dorsal comparison view of the feet in a patient with acute Charcot neuroarthropathy.

**Fig. 2:** A Charcot foot with evident "rocker-bottom" deformity.
**Fig. 3:** Immobilisation with a total contact cast in a patient with early Charcot neuroarthropathy. Early offloading and immobilisation in patients with Charcot Stage 0 has proven to stop the disease activity and prevent major foot deformities.
**Fig. 4:** Plain radiograph demonstrating joint distension, dislocation, disorganisation, debris and increased density, especially of the tarsal navicular bone.

**Fig. 5:** Charcot neuroarthropathy involving the subtalar (a) and ankle (b) joints.
Fig. 6: 75 year-old female with poorly controlled type 2 diabetes mellitus, progressive knee pain and deformity following an acute inflammatory episode where no organism was isolated or cultured from knee joint aspirate. Presumed CN knee. Note background calcification of diabetic vasculopathy.
Fig. 7: Oblique radiograph of foot shows subchondral osteopaenia, the earliest finding of acute CN on plain radiograph.

Fig. 8: 63-year-old male with Charcot neuroarthropathy. Sagittal MRI shows inferior subluxation of cuboid (arrow) and impingement of peroneus longus tendon (asterisk), resulting in severe tendinosis, tendon tear and tenosynovitis.
Fig. 9: 40-year-old female with diabetes mellitus and peripheral neuropathy who presented with two weeks of foot pain, swelling and erythema. Sagittal fat-suppressed T2-weighted image shows marked soft tissue oedema and periarticular subchondral bone marrow oedema in the midfoot bones along with small amounts of joint fluid, compatible with CN stage 0.
Fig. 10: A neuropathic ulcer on the plantar midfoot, typical of the rocker-bottom deformity.

Fig. 11: 66-year-old male with infected Charcot neuropathic osteoarthropathy (a) Sagittal STIR sequence shows abnormal T2 signal. (b) Gadolinium-enhanced fat-suppressed T1-weighted image shows abnormal enhancement. Both findings in (a) and (b) are non-specific. (c) The ‘ghost sign’ is diagnostic of CN with superimposed infection and is characterised by the presence of profound low signal in T1-weighted images with loss of definition of margins of the affected bone.
Fig. 12: 53-year-old female with diabetes mellitus and peripheral neuropathy. (a) and (b): Axial CT of the foot demonstrates subacute midfoot CN with multifocal fractures in the tarsus, not easily demonstrated on plain radiography.
Fig. 13: Plain radiographs demonstrating healing metatarsal stress fractures (a) and collapse of the metatarsal heads indicative of osteonecrosis (b).
Fig. 14: Trainees and consultants in Vascular Surgery, Radiology, Endocrinology, and Podiatrists participate in the weekly High Risk Diabetic Foot Multidisciplinary Team meeting.
Conclusion

Imaging is important for the detection of early Charcot neuroarthropathy and is useful in monitoring progression and complications of the disease. The later stages of CN are potentially devastating for individuals and present an increasing socio-economic challenge for health systems. The astute radiologist, particularly in the context of a multidisciplinary team, plays a critical role in diagnosis of the primary disease and its complications as well as being able to offer valuable guidance on how best to image this clinical problem.

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References