Foot & ankle

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The acute Charcot foot in diabetics: diagnosis and management

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- Acute Charcot foot is a diagnostic challenge.
- The exact pathophysiology is not fully understood.
- Acute Charcot foot is often present with a history of trauma or cellulitis which does not respond to antibiotics.
- The condition is best managed within a multidisciplinary team.
- The mainstay of the treatment is mechanical off-loading and total contact casting.
- Surgery is reserved for select cases.

Keywords: acute Charcot foot; diagnosis; management

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Introduction

Acute Charcot foot is a rare, disabling condition which can cause widespread destruction of bone and joint architecture with loss of function. Diabetes mellitus is currently the commonest cause typically affecting the foot due to loss of its protective sensations. Early recognition of acute Charcot foot in diabetics is a diagnostic challenge as the clinical suspicion even in high risk patients is often low, and the consequences of a missed diagnosis can be devastating. This article provides an overview of the clinical presentation, investigation and treatment of acute Charcot foot.

Charcot neuroarthropathy (CN) is a rare, but serious, disabling condition which can cause widespread destruction of bone and joint architecture with loss of function.[1] The pathogenesis of CN was classically described by French physician Jean Martin Charcot[2] in 1883 but a complete knowledge of this challenging condition continues to evolve to date. It is, however, certain that any condition resulting in loss of protective sensory innervation or autonomic neuropathy can lead to CN. Common causes include diabetes mellitus, alcoholism, spinal injuries, syringomyelia, syphilis, and congenital insensitivity to pain,[3] and its prevalence among diabetics is about 1%.[4]

The chronic stages of CN are easily recognizable with the appearance of an irreversible 'rocker bottom' deformity due to midfoot collapse or joint destruction (Fig. 1). However, the earlier stage of CN, which is often described as acute Charcot foot, remains a diagnostic challenge (Fig. 2). It usually presents as a red, hot, swollen foot, and may be indistinguishable from other aetiologies of swollen foot such as cellulitis, sprains or deep vein thrombosis.[5]

Pathophysiology

The understanding of pathophysiology of acute Charcot foot is rapidly evolving, but a multifactorial pathogenesis seems certain.[6] Historically there have been two main theories of how this process develops. The neurovascular theory suggests that nerve damage results in increased local vascularity. This precipitates osteoclastic activation with secondary osteopenia, fractures and deformity.[7] The neurotraumatic theory suggests that microtrauma in insensate joints causes progressive bony destruction with repeated partial healing. There is also associated activation of pro-inflammatory cytokines with resulting increased vascularity and activation of the receptor activator kappa beta (RANK)–RANK ligand (RANKL) axis. This results in osteoclastic activation and bone loss. Ultimately there may be a cycle of fractures followed by progressive joint deformity.[8]

Hyperglycaemia in diabetic patients has been shown to increase levels of advanced glycosylation end products (AGEs). This may partially explain the association between poor diabetic control and the development of Charcot as

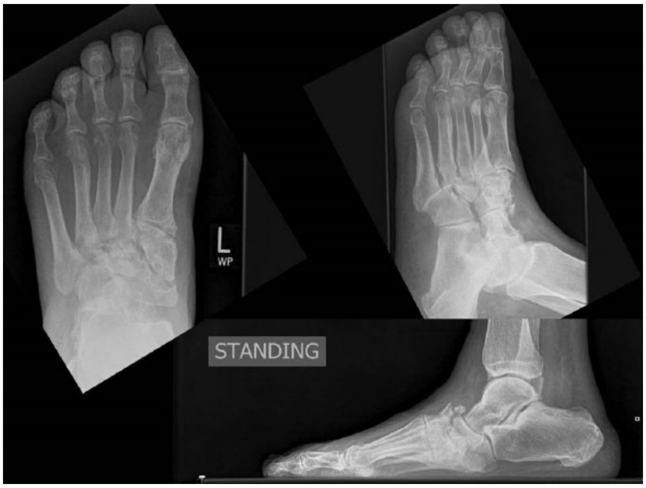

Fig. 1 Radiographs of the foot demonstrating the classic midfoot collapse associated with acute Charcot foot.

the AGEs are able to upregulate the RANK–RANKL pathway by interacting with their receptor, the RANK receptor, especially after a fracture or repetitive trauma.[9]

Clinical features and diagnosis

Patients are most commonly affected in their fifth and sixth decades and both sexes are affected almost equally, although there is a slight male predominance. Most patients have been diabetic for more than five years before the onset of the acute Charcot process. Whilst patients may recall a specific traumatic event which started their symptoms, very often no causative event will be identified.[10] About 25% of patients will ultimately develop similar changes in the other foot.[3]

Diagnosis of this condition remains predominantly clinical and is based on the presence of swelling, erythema, increased skin temperature and joint effusion in an insensate joint. The presentation may be indistinguishable from that of cellulitis, gout or deep vein thrombosis. As such, most patients in fact present to emergency medical services at two to three months after the onset of symptoms. Peripheral sensory neuropathy associated with reduced sensation is the predisposing condition that permits the development of arthropathy. Pain, however, may sometimes still be a feature.[11]

The Charcot process, as described by Eichenholtz, passes from this acute phase of development through a stage of coalescence, in which the bone fragments are reabsorbed, the oedema lessens, and the foot heals, to the stage of consolidation, in which the final repair and remodelling of bone occurs.[12] The predictable pattern of untreated disease leads to collapse of the longitudinal and transverse arches resulting in a 'rocker-bottom' foot or a collapsed and destroyed ankle joint.[13]

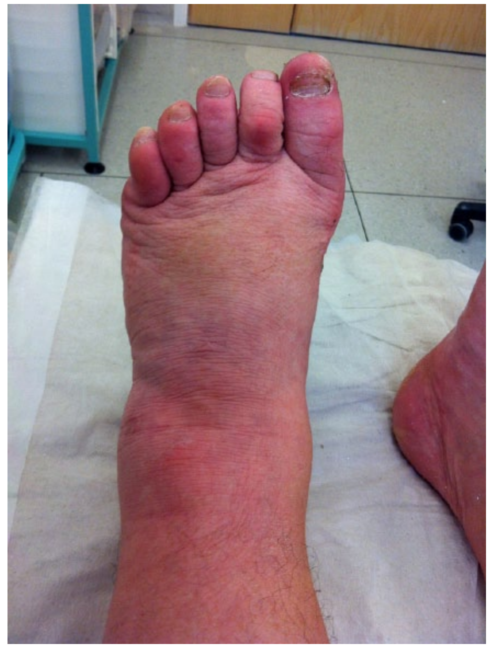

Fig. 2 Early appearance of acute Charcot foot. Charcot should be suspected in any diabetic with a swollen red foot.

Diagnosis

Pattern recognition is one of the key features in early diagnosis of acute Charcot foot. This must be suspected in any diabetic patient who presents with swelling, redness, and, sometimes, pain in the foot and ankle that is of short duration (within four to six weeks) as shown in Figure 2. Acute Charcot foot after fracture is also a commonly missed diagnosis and a high index of suspicion with vigilant monitoring is required to prevent complications (Fig. 3).[14] An acute process should be suspected if the temperature of the affected foot is 2°C or more than the contralateral unaffected foot. This is usually measured using an infrared thermometer.[15]

Biochemical markers

Laboratory investigations usually are not very helpful and there are no accepted criteria for diagnosis. Common blood markers such as erythrocyte sedimentation rate and C-reactive protein values can be normal in the presence of acute Charcot. However, these markers should be used to differentiate from infection.[16]

Imaging

For many years there has been a lot of interest in developing an imaging technique which could be both sensitive and specific for detection of acute Charcot foot.[17–22] Plain radiographs have traditionally been the first line of investigation for patients with suspected acute Charcot foot and have stood the test of time. Radiographs provide valuable information about bony anatomy and alignment. In the

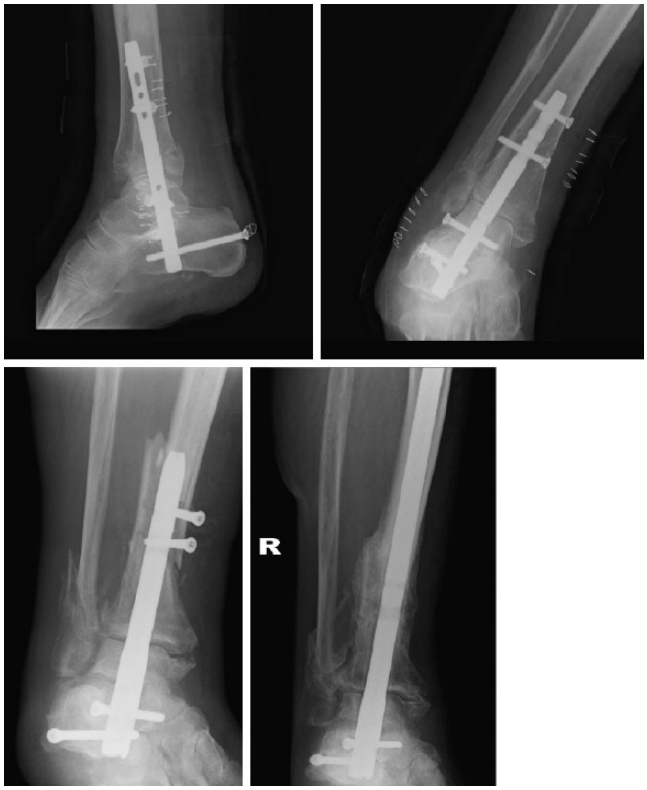

Fig. 3 Series of radiographs demonstrating acute Charcot after ankle fracture surgery. Fracture fully united after six months.

early stages plain radiographs are often normal but this should not exclude the disease process. In later stages, radiographic findings vary from a subtle fracture to increased bone mineralization or loss of alignment and destruction of the foot architecture.[17]

Magnetic resonance imaging (MRI) allows detection of subtle changes in an acute Charcot foot in the presence of a normal radiograph. MRI has a high sensitivity and specificity for osteomyelitis and is considered the test of choice for the evaluation of the foot complications in diabetic patients. However, MRI cannot reliably differentiate between an acute Charcot foot and osteomyelitis and has a sensitivity of between 77% and 100% and a specificity of between 80% and 100% in this differentiation. Findings need to be correlated with the clinical picture, along with other evidence of infection in the form of tissue biopsies, cultures and biochemical markers.[18–19]

Computed tomography (CT) is more sensitive than plain radiography for detecting osteomyelitis but there are several problems with its use; CT is unreliable in detecting osteomyelitis in the early stages of disease although it can be of significant value in detecting soft tissue collection or abscess. Both metformin therapy and diabetic nephropathy can preclude the use of IV contrast, limiting the use of contrast CT in this patient subgroup.

Several imaging techniques are available which use radioisotope tracers. Triple-phase bone scintigraphy is a very sensitive but not a specific technique for detecting active bony pathology, since when a foot is also affected by vascular insufficiency the study may be falsely reassuring. Indium-111-labelled leukocyte scanning results in the highest sensitivity and specificity for osteomyelitis, which is the commonest differential for acute Charcot foot, and may be of benefit in highly selected patients where a diagnostic dilemma persists despite other investigations.[20]

Positron emission tomography (PET) is gaining popularity, especially when combined with CT. The PET–CT hybrid allows improved localization and may prove a more useful tool than MRI in differentiating from osteomyelitis.[21] Weekly Doppler analysis has been successfully used as a tool to help stage the Charcot process. This may be of some benefit in deciding timing of weight-bearing status or surgery.[22]

In a patient with low clinical suspicion of osteomyelitis and no sign of acute Charcot foot on the radiographs, either a triple-phase bone scan or non-contrast MRI can be effective in excluding a bony process. In patients with ulceration where deep infection is likely, MRI is the best diagnostic modality, although ultimately there is no test which is both very sensitive and very specific. Therefore, the diagnosis of acute Charcot foot is a combination of clinical features, blood tests and imaging studies combined.[23]

Biopsy

Bone biopsy is potentially the only diagnostic technique for definitive discrimination between osteomyelitis and acute Charcot foot. This is not, however, always appropriate and can lead to a number of secondary complications including infection, excessive bleeding, fracture, or new onset of acute Charcot process. These factors may limit the use of this technique to cases where the diagnosis is in significant doubt or where there is a high index of suspicion for osteomyelitis.[24]

Management

Diabetic patients with acute Charcot foot are best managed within a multidisciplinary team (MDT). Care is directed at optimizing diabetic control and other risk factors such as smoking and nutritional status. In addition, its aim is to identify patients at risk for further fractures requiring off-loading and management of bone disease with medical or surgical treatment. This approach has helped to improve the quality of diabetic foot care as well as to reduce the rate of major amputations.[25,26]

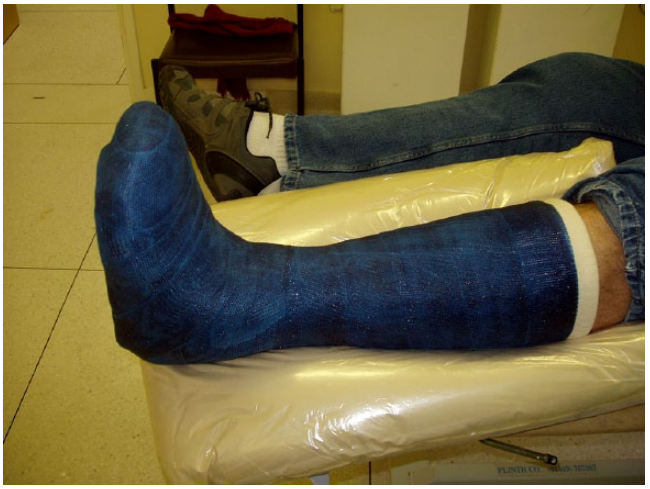

Fig. 4 Total contact casting in a patient with Charcot neuroarthropathy. The patient may bear weight in the cast but it is changed weekly.

Role of diabetic MDT or foot care teams

The National Institute for Health and Care Excellence (NICE) has issued guidance recommending hospitals having both a pathway for the admission of diabetic patients with acute foot conditions, and a regular multidisciplinary review of patients at risk. The multidisciplinary team should include a diabetes nurse specialist, podiatrist and tissue viability nurse. The team needs access to a diabetologist as well as both vascular and orthopaedic surgeons who have experience in treating diabetic foot problems.[27] The role of the orthopaedic surgeon is increasingly recoginized in the management of acute Charcot foot and is central in providing a biomechanical perspective to reduce the risk of complications.[28]

Medical treatment

Initial management consists of off-loading the foot to disrupt the cycle of inflammation and disease progression whilst maintaining bony architecture and preventing deformity. Antiresorptive drugs, in the form of oral bisphosphonates or intravenous pharmacological agents such as Pamidronate, have been used for the management of acute Charcot foot. However, there is no conclusive evidence to support the routine use of pharmacological adjuncts in the management of acute Charcot foot.[29]

Mechanical off-loading and the role of total contact casts

Prompt diagnosis is the key element to achieve optimum results in acute Charcot foot as early immobilization and off-loading can arrest the disease progression. Total contact casting (TCC) remains the preferred treatment modality although a range of orthotic, restricted weight bearing and rehabilitative options are available.[30] Figure 4 demonstrates a total contact cast.

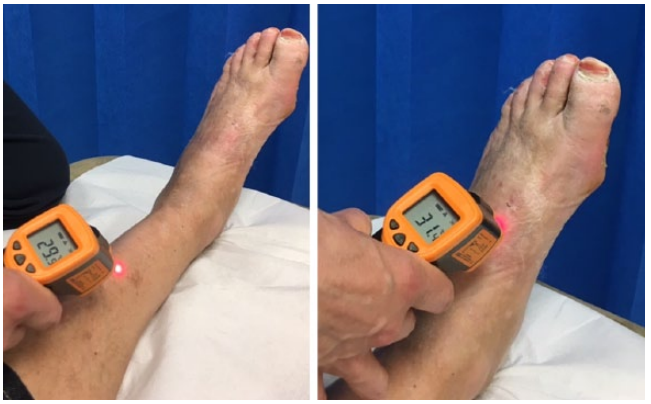

Fig. 5 Acute Charcot foot temperature monitoring showing difference of skin temperature during serial total contact casting.

The TCC when appropriately applied reduces mechanical forces, inflammation and oedema. It aids in the redistribution of plantar pressure, limits bone and joint destruction, and can help to consolidate the progression of deformity. The TCC is designed to cover all major bony prominences of the entire foot and ankle, with well-padded cotton-based bandages (Fig. 4). Frequent cast changes are critical to avoid pistoning due to loosening and ulceration within the cast, and patients should be closely monitored on a weekly basis until the active phase has ended. The patient can then be fitted with a Charcot Restraint Orthotic Walker (CROW) and later with a custom shoe or orthoses. A CROW is a total-contact ankle-foot orthosis, which resembles a TCC but is removable.[31]

The duration of off-loading should be guided by clinical assessment; this focuses on the presence of skin-colour and temperature changes. Casting should be continued until the swelling and redness have clinically resolved or the temperature of the affected foot is within 2°C of the contralateral foot or shin (Fig. 5), and there is radiological evidence of good bony union.[32–33] This period could be quite variable and can last from four to six months. The patient should be prescribed diabetic footwear with a custom-made orthosis to prevent recurrence, ulceration, or subsequent deformities after an acute or active episode has resolved.[30]

Surgical management of acute Charcot foot

Where the correct diagnosis is made and non-operative treatment is successful, surgery may be avoided and the risk of subsequent ulcerations and/or amputation may be decreased. Regardless of chosen technique, most surgical management can be challenging owing to the bone and neuropathic changes seen in diabetic Charcot patients. Only patients who are likely to comply with the postoperative regime should be considered for fusion or deformity correction. The patient must have sufficient vascularity

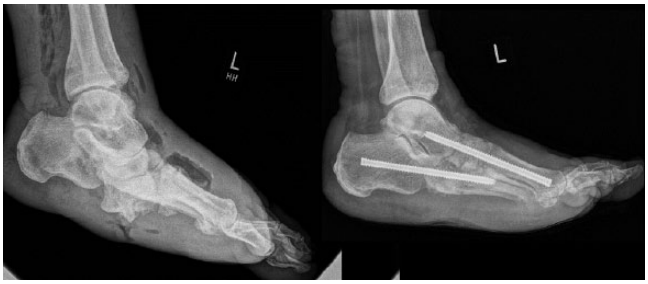

Fig. 6 Radiograph demonstrating midfoot collapse as a result of acute Charcot foot (left) and after reconstruction (right).

in the lower limbs, determined by the ankle brachial index (0.9 to 1.2) and transcutaneous partial oxygen pressure (> 50 mm Hg pressure), in order to permit adequate healing of the surgical wounds.[34]

The aim of surgery is to hold the foot in an anatomical position so as to avoid deformity development or progression. This can be achieved by internal or external fixation. Hence the need to take radiographs of the foot in contact cast early on. Typically, changes in Meary's angle would suggest fore- or midfoot deformity development or progression and the need to abandon casting for rigid means of holding the foot. Hence the need to take serial radiographs of the foot in TCC. The role of surgery is often confused with the chronic setting, which is nothing but deformity correction – albeit rather complex – and may be complicated by ulceration. It is therefore of paramount importance that foot deformity development should be avoided. Once the foot is plantigrade it can be accommodated in a custom-made ankle-foot orthosis.[35,36]

Surgery is often avoided during the active inflammatory stage because of the perceived risk of wound infection or mechanical failure of fixation. Early correction of the deformity combined with arthrodesis can, however, be performed in selected cases with adequate soft-tissue perfusion.[35] More recently, reconstruction techniques have gained popularity. These either employ osteotomy and bony fusion supplemented with rigid internal or external fixation to avoid excessive soft-tissue violation.[37]

Surgical reconstruction of the midfoot relies on realigning the foot with the constant first ray. A long medial column screw or bridge plate spanning multiple joints may be used to provide stability (Fig. 6).[38] In hindfoot disease, encouraging results have been demonstrated recently using a hindfoot nailing technique. Prolonged immobilization and rehabilitation after surgery is often required.[39]

Patient education

Patient education is of paramount importance to improve the outcome of management of acute Charcot foot. If the patient understands the nature of this limb-threatening condition and the rationale for estimated length of treatment, they are likely to be more motivated to follow the management plan.[40] Emphasis on the importance of optimizing glucose control and attending regular MDT clinics in the long term is likely to reduce the risk of complications related to acute Charcot foot.

Conclusion

All physicians treating diabetic patients should be vigilant for acute Charcot foot and once diagnosed it should be treated as a medical emergency. Prompt treatment can help prevent more extensive collapse and the need for more radical surgeries such as amputation.

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