Charcot osteoarthropathy of the foot

Byron M Perrin
Marcus J Gardner
Anwar Suhaimi
David Murphy

Background
One of the most devastating complications of diabetes is Charcot osteoarthropathy. It can lead to gross structural deformities of the foot and ankle, and subsequent skin ulceration and lower limb amputation from soft tissue or bony infection. However, it is often unrecognised, with deleterious consequences.

Objective
This article describes the case of a man with type 1 diabetes who presented with Charcot osteoarthropathy of both feet, with a 3 month delay in diagnosis between the two presentations.

Discussion
Treatment, patient comorbidities and risk management were similar for both feet, with a marked difference in outcome, demonstrating the importance of the timely diagnosis of Charcot osteoarthropathy.

Keywords: diabetes mellitus; peripheral nervous system diseases; arthropathy, neurogenic; Charcot's joint

Charcot osteoarthropathy, a destructive joint disorder, was first described in detail around 130 years ago by the celebrated French physician, Jean Martin Charcot, based on reports of patients afflicted with tabes dorsalis.[1] Today diabetes mellitus is the most common aetiology associated with Charcot osteoarthropathy, with the joints of the foot and ankle being most commonly affected.[2] With the prevalence of diabetes at epidemic proportions, it is likely that the medical practitioner will be exposed to an increasing number of patients with this condition.

Charcot osteoarthropathy is a condition associated with peripheral neuropathy, and is characterised in its early stages by acute inflammation that leads to bone and joint fracture, dislocation, instability and gross deformities.[3] The prevalence is estimated to range from 0.08–13% of people with diabetes;[4] however the actual prevalence is probably much greater due to misdiagnosis or delay in diagnosis.[5] Appropriate diagnosis has been reported to be delayed by as much as 29 weeks in some cases, and the condition is often misdiagnosed as gout, deep vein thrombosis, soft tissue injury, osteo/rheumatoid arthritis or infection (more often osteomyelitis).[3,5,6] The high frequency of misdiagnosis is largely due to a lack of awareness rather than the difficulty of diagnosing the condition (where the diagnosis is made on examining the risk profile, clinical examination and imaging.)

In most cases a delay in diagnosis can lead to severe and debilitating structural deformity of the foot.[6] This subsequent deformity in the presence of peripheral neuropathy greatly increases the risk of skin ulceration and lower limb amputation.[7,8] In fact, Charcot osteoarthropathy has been described as a medical emergency,[9] in that an early diagnosis followed by appropriate treatment (such as aggressive offloading) can effectively impede the condition's destructive process.[6]

In patients with diabetes, Charcot osteoarthropathy is associated with a longstanding duration of diabetes and peripheral neuropathy.[4] In the early stages of Charcot osteoarthropathy, the patient presents with a warm, erythematous and oedematous foot with or without associated pain or reported previous injury.[10] Traditionally, the clinical staging of the condition has relied upon a radiographic system which outlines three distinct stages of the process:

- fragmentation (demonstrated by visible bony fragmentation and joint disruption with osseous debris around the joint)
- coalescence, and

- reconstruction of the damaged joints and bone.[3]

However, more recently there have been descriptions of an earlier stage (stage 0) whereby clinical signs of inflammation are present but no visible bone or joint pathology is seen on plain X-ray.[11] It is during this 'stage 0' that timely diagnosis and treatment can prevent severe bone and joint destruction.[6] To aid in diagnosis during stage 0, imaging such as such as magnetic resonance imaging (MRI) or a three phase technetium[99] bone scan can be undertaken.[10] In the absence of an open wound, these imaging techniques can help confirm increased bone activity to suggest a diagnosis of Charcot osteoarthropathy.

In the *Case study*, timely diagnosis and appropriate treatment of a 'stage 0' Charcot osteoarthropathy prevented subsequent bone and joint deformity. This example is particularly instructive in the context of the contralateral foot of the same patient being affected by Charcot osteoarthropathy at the same time; however for this foot the diagnosis was delayed by up to 3 months.

Case study

In May 2008, a male, 48 years of age, with type 1 diabetes of over 30 years duration presented to the clinic with a 3 month history of a painless, swollen and warm right foot. He had not had any prior definitive diagnosis or treatment. He lived alone and had continued to work during this time (on his feet) for 12 hours a day, 6 days a week. At this first presentation he also reported that in the previous 5 days he had noticed that his left foot had become red, hot and swollen. On examination peripheral neuropathy was confirmed. Both the patient's feet were red, hot and swollen (*Figure 1*). There was no break in the skin of either foot. The right foot was obviously structurally deformed; the left foot, however, appeared to have an intact structure. There were no clinical signs of infection and the C-reactive protein level was normal. Plain X-rays showed gross destruction of the bones and joints of the right foot and no pathology in the left foot (*Figure 2*). As expected, MRI showed gross marrow oedema in the right foot. More importantly, MRI also showed marrow oedema in the midfoot of the left foot (*Figure 3*). In both feet, the MRI changes were consistent with Charcot osteoarthropathy. The diagnosis of bilateral Charcot osteoarthropathy was made – the right foot in the 'fragmentation' stage, with the left foot, notably, in stage 0.

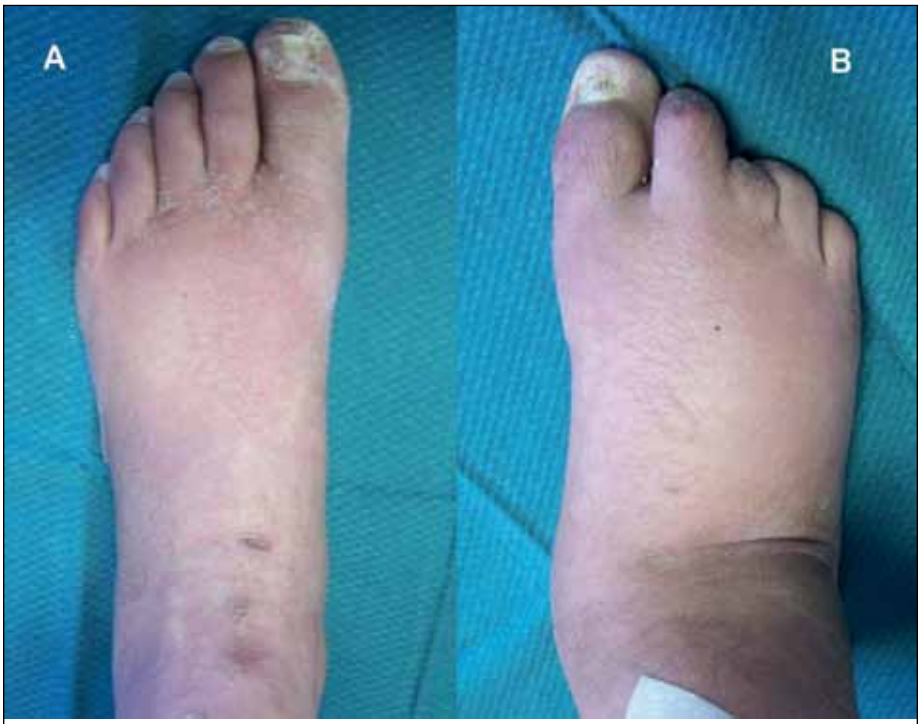

Figure 1. A) the left foot was red, hot and swollen, with no structural deformity; B) the right foot was red, hot and swollen, with obvious structural deformity

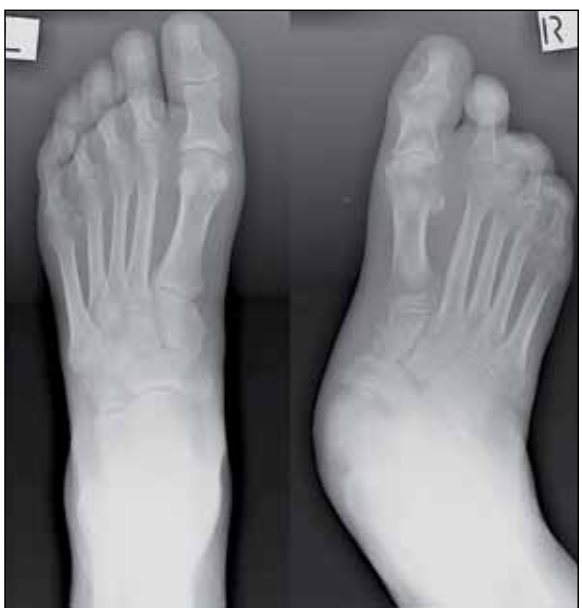

Figure 2. There was no plain film evidence of Charcot osteoarthropathy affecting the bones of the left foot. There were gross bone and joint changes in the mid and rear foot of the right foot, including severe subluxation of the talonavicular joint

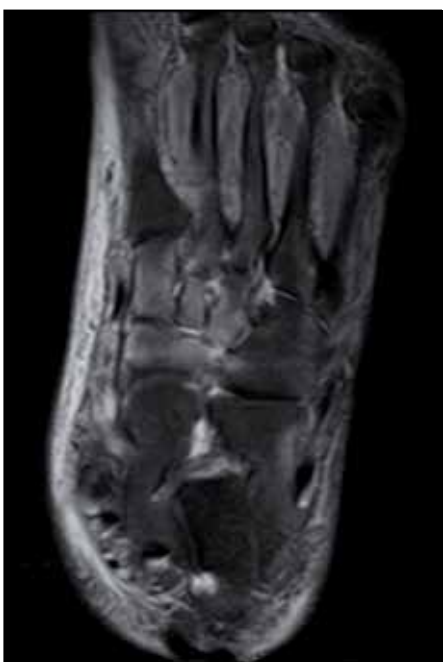

Figure 3. The cuneiforms and navicular of the left foot demonstrate abnormal T2 weighted signal intensity consistent with marrow oedema

Following this diagnosis, the patient was immediately admitted to an inpatient rehabilitation ward with goals to cease all weight bearing activities, stabilise his diabetes control and implement a long term regimen of bilateral total contact casting (the gold standard treatment for acute Charcot osteoarthropathy).[12] After 2 weeks he was discharged home with bilateral total contact casts, which were only weight bearing for transfers from the wheelchair to the bed or toilet. After subsequent MRI showed regression of marrow oedema bilaterally, the levels of weight bearing in the total contact casts increased to ambulation using a four wheel frame.

After 6 months of total contact casting he was able to walk without a gait aid, wearing a custom made Charcot Restraint Orthopaedic Walker on his right foot to accommodate the severe structural deformity of his right foot. Importantly, the left foot remained structurally intact and he was (and continues to be) able to wear a normal, flat, lace up walking shoe. Unfortunately, his right foot remains at an extremely high risk of skin ulceration due to the structural deformity of the foot. The left foot, however, remains structurally sound and is therefore at a much lower risk of future skin ulceration.

Discussion

The case study provides a unique opportunity to demonstrate how an early and appropriate diagnosis of Charcot osteoarthropathy can prevent the destruction of a foot. The patient had bilateral Charcot osteoarthropathy; in the left foot the diagnosis was made in a timely fashion, and in the right foot the diagnosis was delayed by 3 months. The management was the same for both feet. The results show that in the foot where the diagnosis was delayed there was almost complete destruction of the bony structure of the foot, and as a result this foot is at an extremely high risk of skin ulceration and lower limb amputation. It is unlikely that this damage can be remediated. In the foot where the diagnosis was not delayed the bone and joint integrity of the foot remain intact. It was the timely, deliberate management of this foot (predominately with the application of a total contact cast) while the Charcot process was in its early stages that prevented any further bone or joint damage.

This case demonstrates the need for all health professionals who have contact with people with diabetes to be aware of this condition and to have a high index of suspicion whenever a person with diabetes presents with a warm, swollen foot. Accurate diagnosis can lead to appropriate treatment and subsequent reduction in the risk of skin ulceration and lower limb amputation in an already high risk population.

Summary of important points

- Charcot osteoarthropathy is an extremely destructive condition that primarily affects the bones and joints of the foot and ankle. It can lead to reduced mobility, foot ulceration and lower limb amputation.
- If this condition is diagnosed early (before X-ray changes) and treated appropriately the risk of future morbidity is greatly reduced. However, it is often misdiagnosed by primary health care physicians.
- Charcot osteoarthropathy should be considered for any patient with established peripheral neuropathy who presents with a warm, red and swollen foot or ankle.
- If Charcot osteoarthropathy is suspected there should be immediate cessation of all weight bearing activities. Once formally diagnosed the most appropriate management is the use of total contact casts. High risk foot clinics are the most appropriate referral destination.

Authors

Byron M Perrin BPod, MHlthSc, is lecturer, Department of Podiatry and Musculoskeletal Research Centre, La Trobe University, Victoria. b.perrin@latrobe.edu.au

Marcus J Gardner BAppSc(Pod), is chief podiatrist, Bendigo Health, Victoria

Anwar Suhaimi MBBS, is a rehabilitation registrar, Bendigo Health, Victoria

David Murphy MBBS, FAFRM, is Clinical Director Rehabilitation, Bendigo Health, Victoria.

Conflict of interest: none declared.

References

1. Hoche G, Sanders LJ. On some arthropathies apparently related to a lesion of the brain or spinal cord, by Dr JM Charcot, January 1868. J Hist Neurosci 1992;1:75–87.
2. Frykberg RG, Kozak GP. Neuropathic arthropathy in the diabetic foot. Am Fam Physician 1978;17:105–13.
3. Nielson DL, Armstrong DG. The natural history of Charcot's neuroarthropathy. Clin Podiatr Med Surg 2008;25:53–62.
4. Frykberg RG, Belczyk R. Epidemiology of the Charcot foot. Clin Podiatr Med Surg 2008;25:17–28.
5. Pakarinen TK, Laine HJ, Honkonen SE, Peltonen J, Oksala H, Lahtela J. Charcot arthropathy of the diabetic foot. Current concepts and review of 36 cases. Scand J Surg 2002;91:195–201.
6. Chantelau E. The perils of procrastination: effects of early vs. delayed detection and treatment of incipient Charcot fracture. Diabet Med 2005;22:1707–12.
7. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation. Basis for prevention. Diabetes Care 1990;13:513–21.
8. Reiber GE, Vileikyte L, Boyko EJ, et al. Causal pathways for incident lower extremity ulcers in patients with diabetes from two settings. Diabetes Care 1999;22:157–62.
9. Tan AL, Greenstein A, Jarrett SJ, McGonagle D. Acute neuropathic joint disease: a medical emergency? Diabetes Care 2005;28:2962–4.
10. Rogers LC, Bevilacqua NJ. The diagnosis of Charcot foot. Clin Podiatr Med Surg. 2008;25:43–51.
11. Yu GV, Hudson JR. Evaluation and treatment of stage 0 Charcot's neuroarthropathy of the foot and ankle. J Am Podiatr Med Assoc 2002;92:210–20.
12. Sanders LJ, Frykberg RG. The Charcot foot (Pied de Charcot). In: Bowker JH, Pfeiffer MA, editors. Levin and O'Neal's The Diabetic Foot. 7th edn. Philadelphia: Mosby Elsevier, 2008.

correspondence **afp@racgp.org.au**