Charcot foot in a person with impaired glucose tolerance

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Article points

1. Diabetes is thought to be the most common cause of Charcot neuroarthropathy, but Charcot neuroarthropathy is an uncommon complication of diabetic peripheral neuropathy and associated with significant morbidity.
2. The authors present a case of Charcot neuroarthropathy in a man with impaired glucose tolerance and a previous unilateral below-knee amputation.
3. To the authors' knowledge this is only the second time Charcot neuroarthropathy has been reported in association with impaired glucose tolerance.

Keywords

- Amputation
- Charcot neuroarthropathy
- Impaired glucose tolerance

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The authors report the case of a 72-year-old man with impaired glucose tolerance and a hypoplastic right-leg deformity of unknown aetiology, who presented with acute Charcot neuroarthropathy in the residual foot. Charcot now rarely occurs in the absence of established diabetes in the developed world.

Charcot neuroarthropathy is an uncommon complication of peripheral neuropathy associated with significant morbidity. Jean-Marie Charcot (1868) first described the condition and noted its association with syphilis. Diabetes is the cause of almost all modern cases of Charcot neuroarthropathy in the developed world. It has a reported incidence of between 0.1% and 0.4% of people with diabetes (Rajbhandari et al, 2002). Outside of people with diabetes, the condition is rare.

Case report

A 72-year-old white man – Mr G – presented with a 4-week history of painless swelling and erythema in his left foot. His GP treated him unsuccessfully for presumed cellulitis in the weeks prior to this. He was febrile at 39°C and his left foot was swollen, erythematous and warm to the touch. He was admitted for inpatient care.

Investigations revealed a normal white blood cell count but elevated C-reactive protein (172 mg/L). The working diagnosis was partially treated cellulitis and Mr G was treated with intravenous flucloxacillin.

Mr G had recently been diagnosed with impaired glucose tolerance by his GP. This diagnosis was confirmed on admission with a fasting blood glucose level of 6.3 mmol/L.

Mr G had a hypoplastic deformity of the right leg. Aetiology of this deformity was unknown and corrective surgery in his 40s had lead to chronic infection and a below-knee amputation. Mr G had a prosthetic limb that he had worn since the amputation.

The foot did not improve and an magnetic resonance imaging scan was performed. This showed abnormal enhancement of the bone marrow in the base of all five metatarsal bones and all the midtarsal bones to the level of the subtalar and talonavicular joint, with soft tissue enhancement around the forefoot and also within the intraarticular region. This was thought to indicate an infection of the soft tissues and midfoot osteomyelitis. Debridement and bone biopsy were performed to isolate the infecting organism. Cultures from the biopsy failed to

identify an organism, with histology showing degenerative changes, with no signs of acute or chronic inflammation.

Mr G completed a 6-week course of intravenous antibiotics and was discharged home with oral antibiotics. Six weeks after discharge Mr G was seen in clinic for review when it was noted that he had developed a painless foot deformity with loss of the arch. There had been no associated trauma. The wounds from the bone biopsy site had healed well, however the foot was erythematous and warm to touch (*Figure 1*). Peripheral neuropathy was confirmed by nerve conduction studies, which were consistent with distal axonal neuropathy. There was no evidence of peripheral vascular disease with all pedal pulses being palpable.

Further investigations were performed to elucidate the cause of the peripheral neuropathy. He had no history of malignancy or use of chemotherapeutic agents. He drank alcohol very occasionally, with no history of alcohol abuse. He had no family history of neuropathy. Syphilis serology was negative. Protein electrophoresis did not show evidence of a paraproteinaemia and there was no Bence Jones protein detected in his urine. His renal function was normal, with a normal creatine kinase. An autoantibody screen was negative and his erythrocyte sedimentation rate was 12 mm/hour. His thyroid function tests and vitamin B12 level were normal.

A diagnosis of an acute midfoot Charcot process with associated deformity was made. The foot was immobilised in a removable total-contact walker. Due to previous right below-knee amputation, Mr G was wheelchair bound to achieve maximum offloading. Encouragingly, at 6-month follow-up Mr G was able to progress from the walker to a custom insole.

Discussion

The term Charcot's disease was not coined until the late 19th century when Jean-Marie Charcot described the pathology of joint degeneration in chronic neuropathy. It is now used in relation to any neuropathic arthropathy independent of aetiology. The condition was first linked to diabetes by William Jordan in 1936.

Diabetes is thought to be the most common cause of Charcot neuroarthropathy (Jeffcoate et al, 2000; Houston and Curran, 2001; Dutta et al, 2004; Jeffcoate, 2008; van der Ven et al, 2009), but any condition that causes sensory neuropathy may lead to a Charcot process. Thus, Charcot neuroarthropathy has been linked to diabetes, syphilis, chronic alcoholism, meningomyelocele, spinal cord injury, syringomyelia, renal dialysis, and congenital insensitivity to pain. Outside of the developed world, leprosy remains a major cause of Charcot neuroarthropathy.

In the present case, Mr G did not have diabetes according to the World Health Organization (2006) definition. Charcot neuroarthropathy associated with impaired glucose tolerance has only been described once in the literature in a patient who was later found to have POEMS syndrome (Maurer and Sommer, 1999).

The role of Mr G's previous below knee amputation must be considered as playing a role in the aetiology of the Charcot process reported here. It is known that the prosthetic limb does infer abnormal loading on the residual limb, and repetitive abnormal loading can lead to inflammatory autolysis of skin and, ultimately, to ulceration. However, Charcot neuroarthropathy is not a recognised

Page points

1. Six weeks after discharge Mr G was seen in clinic for review when it was noted that he had developed a painless foot deformity with loss of the arch.
2. Further investigations were performed to elucidate the cause of the peripheral neuropathy and no evidence of the traditional causes could be found.
3. The foot was immobilised in a removable total-contact walker. Due to previous right below-knee amputation, Mr G was wheelchair bound to achieve maximum offloading.
4. The role of Mr G's previous below knee amputation must be considered as playing a role in the aetiology of the Charcot process reported here.

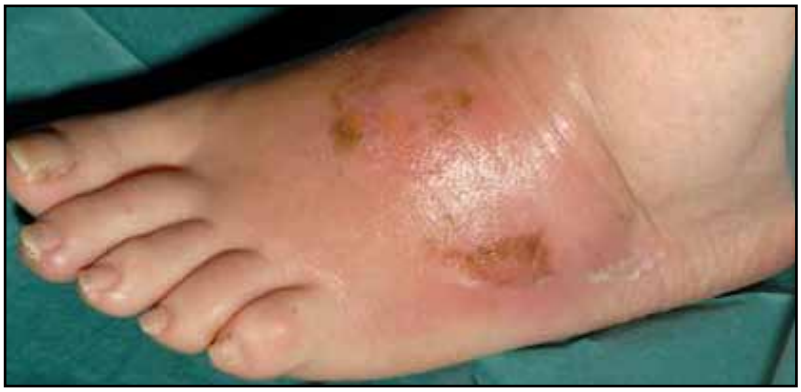

Figure 1. Six weeks after discharge Mr G was seen in clinic for review when it was noted that he had developed a painless foot deformity with loss of the arch. The foot was erythematous and warm to touch.

"It is possible that the Charcot process was present on initial presentation in this case ... it is also possible that the bone biopsy undertaken to assess for osteomyelitis may have triggered the acute process that followed."

complication in the residual limb of amputees. Hence, the combination of the amputation on the unaffected side and Mr G's impaired glucose tolerance are both likely to be relevant contributory factors in this case (Zhang and Roberts, 2000; Klute et al, 2001; Mak et al, 2001; Klute and Berge, 2004).

There are various theories on the causes of Charcot, the most commonly accepted are those involving neurovascular destruction and neurotraumatic injury. While there were no clinical features of micro- or macrovascular disease in the case described here, the presence of a unilateral below-knee amputation would have predisposed the remaining foot to abnormal loading and an increased risk of microtrauma. It has been suggested that microtraumas not remembered by the patient as significant events may be the precipitant in Charcot neuroarthropathy (Rajbhandari et al, 2002). Following an initial insult, autonomic dysfunction results in an increase in local blood flow that contributes to abnormal osteoclastic healing response.

It is possible that the Charcot process was present on initial presentation in this case, although fever and elevated inflammatory markers suggest a concurrent infection. It is also possible that the bone biopsy undertaken to assess for osteomyelitis may have triggered the acute process that followed.

Conclusion

The authors present a case of acute Charcot neuroarthropathy in a man with impaired glucose tolerance and suggest that a previous contralateral below-knee amputation was a contributing factor. To the authors' knowledge this is only the second time Charcot neuroarthropathy has been reported in association with impaired glucose tolerance. ■

Authors

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