CASE REPORT

Charcot foot associated with chronic alcohol abuse

Christina Arapostathi,¹ Nicholas Tentolouris,² Edward B Jude¹

¹Department of Diabetes, Tameside Hospital NHS Foundation Trust, Ashton under Lyne, UK
²Department of Internal Medicine and Diabetes Centre, Laiko General Hospital of Athens, Athens, Greece

Correspondence to
 Dr Edward B Jude,
 edward.jude@tgh.nhs.uk

SUMMARY

Two patients without a history of diabetes mellitus but with a history of chronic alcohol abuse were referred to our foot clinic due to pain and deformity of the midfoot. On examination both of the feet of the first patient and the left foot of the second patient were swollen and warm but all the inflammatory markers were negative. Subsequent imaging revealed Charcot deformity and the patients were treated with casting and special shoes. The temperature and the swelling of the feet after the offloading improved. x-Rays which were performed 1 and 2 years after the diagnosis did not show any progression of the Charcot deformity.

symmetrical sensory motor neuropathy of axonal type. He had an MRI scan of his feet which showed extensive bony reactive changes and oedema in the tarsal and metatarsal region in both the feet and especially in the right foot. He had an isotope bone scan which showed symmetrical isotope uptake, involving the metatarsophalangeal and the proximal interphalangeal joints and the tarsal bones on the feet (figure 1). These findings were consistent with CN, so he was referred to our foot clinic. When we examined the patient, he had absent vibration sense on both his hallux up to the ankle joints and was insensitive to the monofilament and to the pin prick.

BACKGROUND

Charcot neuroarthropathy (CN) is a musculoskeletal condition that leads to a chronic progressive and destructive arthropathy. The most frequently involved joints are the tarsus and tarsometatarsal joints followed by the metatarsophalangeal joints and the ankle. Early offloading is recommended as it can prevent the further destruction of the joint. Peripheral neuropathy due to diabetes is the most common cause of Charcot foot. However, alcohol-induced neuropathy causing Charcot deformity is not a commonly seen condition. Our aim, with these two case reports is to raise the awareness that Charcot foot must be considered in any patient with alcohol abuse who presents with warm, swollen, erythematous foot, particularly in the context of peripheral neuropathy.

CASE PRESENTATION (TABLE 1)**First case**

A 67-year-old man with hypertension, hyperlipidaemia and a history of gastrointestinal bleeding and chronic alcohol abuse (28–32 units of alcohol per week) was referred to the orthopaedics department by his general practitioner due to spontaneous midfoot collapse. He had an x-ray that showed Lisfranc dislocations on both his feet. His problems with his feet started 12 months ago, when he was having difficulty in walking and when he walked his feet were turning inwards. He did not have any pain in his feet or any breaks in the skin, redness or swelling in the past. When the orthopaedic surgeon examined him there were deformities of both his feet and a definite prominence on the dorsum of the feet with loss of plantar arch. The patient noticed that he had a reduction in the plantar arch 1 month before seeing the surgeon. An oral glucose tolerance test (OGTT) was performed to rule out diabetes, and vitamin B₁₂ levels were normal. Nerve conduction studies showed generalised

Second case

A 64-year-old man, former smoker with hypertension, peripheral vascular disease, cervical spondylitis, osteoarthritis of both his knees (total left knee replacement) and alcohol abuse (8 pints of beer per day) was referred by the orthopaedics department to our foot clinic due to pain and deformity of his left midfoot without a history of preceding trauma. On examination his left foot was swollen and warm. The temperature difference between the left and the right foot was 2°. He had pins and needles and burning sensation on both his feet and was insensitive to the 10 g monofilament, to pin prick and to vibration 128 Hz tuning fork, suggesting severe bilateral peripheral neuropathy. The OGTT, B₁₂ and folate levels were normal. He had an x-ray, which showed early changes of CN affecting the tarsal bones, especially in the medial tarsal joints. The isotope bone scan showed an increase uptake in the left tarsal bones in the forefoot and in the tarsometatarsal joint at the navicular, and bone oedema related to the mid-tarsal joints especially in the medial tarsal joints (figure 2). These findings were consistent with Charcot neuroarthropathy.

Table 1 Investigations

	First patient	Second patient
WBC ($\times 10^9/l$)	8.8	7.4
ESR (mm/h)	6	16
CRP (mg/l)	29	23
Creatine ($\mu\text{mol/l}$)	74	118
e-GFR (ml/min)	>90	56.7
HbA1c (%)	5.4	5.2
Fasting glucose (mmol/l)	5.3	5.1
Vitamin B ₁₂ (ng/l)	226	232

e-GFR, estimated-glomerular filtration rate; HbA1c glycated (glycosylated) haemoglobin; WBC, white blood count; ESR, erythrocyte sedimentation rate; CRP, C reactive protein

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FIRST PATIENT

Figure 1 Foot x-ray and bone scan of first patient.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis included cellulitis, osteomyelitis and deep vein thrombosis (DVT). All the inflammatory markers were normal, the patient did not have any fever or chills and the Doppler scanning of the legs did not show any signs of thrombosis. From the subsequent imaging the diagnosis of Charcot arthropathy was made.

TREATMENT

The first patient had been given medial arch support, moulded full cast insoles, special shoes for both of his feet for approximately 1 year.

The left foot of the second patient was put in total contact cast for 6 months.

OUTCOME AND FOLLOW-UP

The x-rays that the two patients had, the first 2 years later and the second 1 year after the diagnosis of CN, did not show any progression of the Charcot deformity.

The first patient is followed up until now in our foot clinic and his feet remain stable.

The second patient 1 year after the diagnosis, despite of the non-progressive Charcot in the x-ray, his left foot remained warm and swollen and it was recommended to be put again in a total cast. At that time he was scheduled for a right knee replacement due to his osteoarthritis but he did not attend, neither to the foot clinic appointment.

DISCUSSION

CN (Charcot foot) is characterised by gradual destruction of the involved joints, in patients with concomitant neurosensory loss, affecting mainly the foot but other joints are known to be affected.

The most common cause of Charcot foot is diabetic neuropathy.¹ However a number of other conditions such as neurosyphilis, multiple sclerosis,² syringomyelia, leprosy, peripheral

nerve injuries, meningocele, spinal cord lesions, folate deficiency, poliomyelitis, vitamin B₁₂ deficiency, neurotoxic medications, HIV infection³ and chronic alcohol abuse^{3, 4} can cause CN. Most publications, demonstrate the correlation of diabetes and Charcot foot, as the prevalence of diabetic neuropathy is greater than that of other types of neuropathy. However, the prevalence of peripheral neuropathy in alcoholic patients is actually similar to that for neuropathy in diabetic patients.

According to the National Institute on Alcohol Abuse and Alcoholism (NIAAA), consumption of more than 7 drinks/week (a standard drink contains 12 g of alcohol—ethanol and is equivalent to 360 ml of beer, 150 ml of wine or 45 ml of 80 proof distilled spirits)⁵ is associated with high risks for alcohol problems. Alcoholism refers to recurrent episodes of excessive drinking despite serious economic, social or medical consequences.

Alcohol dependence is also a chronic disease, associated with malnutrition, trauma and a wide variety of CNS disorders. Most of these disorders appear to be due to neurotoxicity by ethanol, except from Wernicke's encephalopathy, which is the best-known neurological complication of thiamine (vitamin B₁) deficiency. Other neurological-related disorders to chronic alcohol consumption are alcoholic cerebellar degeneration, dementia and myopathy.

The most common neurological complication in alcoholism is polyneuropathy. Polyneuropathy is thought to be caused by nutritional deficiencies as well as direct alcohol toxicity. Malabsorption of thiamine may damage the autonomic, motor and sensory elements of the peripheral nervous systems. In alcoholic polyneuropathy, paraesthesias, pain and weakness especially in the feet are common. Clinical examination reveals reduced pain and temperature sensation, distal muscle weakness and atrophy in the legs. Deep tendon reflexes are reduced and ankle reflexes are usually absent.

The pathogenesis of Charcot arthropathy remains uncertain but it is probably due to a combination of mechanical and vascular factors resulting from peripheral neuropathy. There are two theories, the French or neurovascular theory according to which blood flow to the affected limb is altered and this results to bone resorption and bone weakening and the German or neurotraumatic theory according to which repetitive microtrauma in the presence of neuropathy results in fractures and dislocations.

In the acute phase the leg is warm, erythematous and swollen with bounding pulses. It often resembles to cellulitis, foot infection, gout or DVT.

The radiographic findings in Charcot arthropathy show a rocker-bottom foot deformity. Radiographic changes such as osteopenia may be present due to increased blood flow associated with autonomic neuropathy. Increased blood flow results in increased radiopharmaceutical uptake.⁶ Bone destruction and joint subluxation or dislocation is common as well as medial arch collapse. The radiographic examinations may take some weeks after presentation to clarify the diagnosis of Charcot foot.⁷

Early diagnosis of Charcot is difficult, yet vitally important for the survival of the joint. No single clinical, radiological or laboratory finding can confirm the diagnosis. Diagnosis of Charcot foot is delayed in more than 25% of patients. Evaluation of inflammatory factors such as C reactive protein (CRP), erythrocyte sedimentation rate (ESR) and white blood count (WBC) are necessary for the diagnosis. Presence of local joint inflammation signs but without marked systems signs of infection must alert the practitioners for Charcot foot.

Therapy of Charcot deformity is offloading until the resolution of the oedema and erythema occurs with improvement of the radiological findings. The response to therapy depends on

SECOND PATIENT

Figure 2 Foot x-ray and bone scan of second patient.

the stage at which the condition is diagnosed. If the diagnosis is made before significant radiological damage has occurred then the successful treatment is more likely to prevent future joint destruction.⁸ Oral bisphosphonates⁹ may also be useful, as well as surgical correction¹⁰ in carefully selected cases.

Early diagnosis is essential to give the best clinical outcome and physicians should have a high index of suspicion for Charcot arthropathy when a patient with a history of chronic alcohol abuse presents with a swollen foot.

Learning points

- ▶ Peripheral polyneuropathy is the most common neurological manifestation in chronic alcoholism.
- ▶ Charcot neuroarthropathy (CN) can occur to patients with peripheral polyneuropathy.
- ▶ No single clinical, radiological or laboratory findings can confirm the diagnosis of CN.
- ▶ Early diagnosis and treatment of CN is crucial for prevention of future joint destruction.

Competing interests None.

Patient consent Obtained.

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