**Background**
Charcot neuro-osteoarthropathy (CNO) of the foot is a devastating neuropathic complication of diabetes. It is characterised by deformity of the foot architecture, which can be initiated by trauma to the neuropathic limb or occur spontaneously. The acute phase of the disease is often misdiagnosed and can rapidly lead to deformity and amputation. The aim of management is to halt further bone destruction through immobilisation of the affected limb.

**Objective**
To discuss the diagnosis and management of bilateral diabetic CNO of the foot, diagnosed early according to clinical presentation with normal radiograph findings (Eichenholtz stage 0).

**Discussion**
The importance of early detection of clinical signs and subsequent diagnosis of CNO of the foot is vital in order to allow for the institution of management, with the aim of preserving normal foot architecture.

**Keywords**
diabetes mellitus; arthropathy; neurogenic; diabetic foot

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**Case study**
Max, a school teacher aged 52 years, presented with 5 days of left midfoot swelling. It was initially painless, but over the past 2 days had become painful after walking. He reported no fevers or other joint swelling and had no recollection of recent trauma. He had been diagnosed 17 years previously with type 2 diabetes mellitus, which was now complicated by nephropathy and retinopathy.

On examination, the left foot showed swelling at the medial midfoot, which was warm and inflamed with bounding dorsalis pedis and posterior tibialis pulses. No ulceration or open wounds were identified. There was no deformity noted on the right foot. Bilaterally, the feet had a loss of protective sensation with reduced pinprick sensation, absent monofilament test using 10 g Semmes Weinstein monofilament, as well as loss of vibration and proprioception.

Blood tests showed no significant changes of acute infection. Max’s white blood cell count was 10.1 g/dL, haemoglobin 11.9 g/dL, C-reactive protein 0.6 and HbA1c 8.1%. His serum uric acid level was normal. An X-ray of the left foot showed no evidence of fracture or bone destruction.

Based on the clinical assessment and lack of investigation findings, a provisional diagnosis of acute Charcot neuro-osteoarthropathy (CNO) of the left foot was made. Management was immediate offloading of the left foot with total contact casting in consultation with a specialist foot team. (Total contact casting is a fibreglass shell that fits around the leg and foot with a bar on the bottom to keep weight off the foot.) Max was advised to rest, but allowed partial weightbearing ambulation on the left foot by using a walking frame. At review 2 weeks later, he reported 5 days of right midfoot swelling. Clinically, there was swelling at the medial side of the right midfoot, which was similar to the left foot. All blood parameters were normal, effectively excluding cellulitis and acute gouty arthritis. An X-ray of the right foot showed no significant bone changes.

Similar to his first presentation, a provisional diagnosis of acute CNO of the right foot was made. Bilateral total contact casting was immediately instituted with wheelchair mobility. Both casts were removed biweekly to accommodate reduction of oedema and to monitor foot and skin changes. Signs of inflammation were monitored (eg. skin erythema, oedema, local skin warmth). Contact casting was completely removed after 8 weeks, when there were no signs of inflammation of either foot. Max was then reviewed biweekly to ensure no recurrence of CNO after the offloading period. A pair of extra-depth custom-made shoes with bilateral total contact insoles was prescribed for better plantar pressure distribution.

Max returned to work after 3 months. At 1 year follow up he did not have any further episodes of foot swelling or ulceration.
The prevalence of diabetic Charcot neuro-osteoarthropathy (CNO) of the foot is difficult to determine due to the lack of clear clinical and radiological diagnostic criteria, as well as lack of awareness, which leads to many cases being misdiagnosed. The acute phase of diabetic CNO often goes unnoticed, resulting in delayed management and progression to the chronic phase and subsequent irreversible foot deformity.

In the early phase of acute CNO, patients may present with foot swelling, erythema and elevated foot temperature, but have normal radiological findings. This is classified as Eichenholtz stage 0. This is a pre-fragmentation stage, and it is critical to identify this early to prevent the long term sequelae that may lead to foot deformity and ulceration. General practitioners should have a high index of suspicion for CNO when soft tissue, bone or joint deformity is present in the foot of a diabetic patient, along with loss of protective sensation, absent deep tendon reflexes and diminished vibratory sense.

**Differential diagnosis**

A presentation of CNO at Eichenholtz stage 0 may mimic or be misdiagnosed as sprain, acute gouty arthritis, cellulitis or osteomyelitis. Most cases of infection will usually involve a direct source of inoculation through an opening in the skin with neuropathic ulcer. Clinical examination and investigations, erythrocyte sedimentation rate, c-reactive protein and white blood cell count can exclude infective causes. Indium-111 leucocyte scanning or magnetic resonance imaging (MRI) may be warranted, especially in evaluating patients with apparent soft tissue infection or plantar ulcer.

However, differentiating between acute infection and CNO remains difficult. MRI and indium-111 scanning may show bone marrow oedema and localisation of leucocytes to infected areas, respectively. These findings are highly sensitive to detect acute infection but may give false positive results in the presence of osteoarthropathy. Definitive diagnosis of acute CNO can be established through bone marrow biopsy. However, this procedure is invasive and involves risks to the patient.

The high index of suspicion for acute CNO in the case study was based on history, clinical examination and basic investigations, which did not support the diagnosis of infection or acute gouty arthritis. Several predictors that heightened the risk for acute CNO were diabetes mellitus for more than 10 years, macrovascular (nephropathy) and microvascular (retinopathy and neuropathy) diabetic complications, and poor glycemic control with HbA1c of 8.1%. Further investigations, such as an MRI or leucocyte scan, were not performed in this case due to availability and cost. Hence, further management with total contact casting was commenced to arrest the progression of acute CNO.

**Management**

The aim of management in the acute phase of CNO is to halt the inflammatory process, relieve pain and minimise potential foot deformity. Total contact casting of the affected limb is one of the most effective pressure offloading and immobilisation devices to bring about bone healing and reduce inflammation.

In acute CNO, especially with normal radiological findings, the period of casting and the decision to cease offloading is based on the disappearance of inflammation by clinical evidence such as a reduction in oedema, skin erythema and local skin temperature. In view of the difficulty in establishing the resolution of acute inflammation and the lack of sensitivity in objective imaging (particularly MRI), several authors have advocated a protocol for a period of casting for 3–6 months to ensure total healing. However, prolonged casting can lead to negative sequelae including restriction of mobility, which can lead to an increased risk of falling, reduced quality of life, reduced bone mineral density and increased body mass index. Therefore, the duration of casting needs to be closely monitored and decisions around ceasing casting and offloading must be balanced between the risks and benefits of offloading and reloading.

**Summary**

We have reported an unusual case of bilateral acute diabetic CNO of the foot, which presented with sequential involvement of each foot within a very short time interval. It was diagnosed early based on clinical presentation and with normal radiograph findings (Eichenholtz stage 0). The duration of casting was based primarily on the disappearance of oedema and skin erythema, as well as skin temperature. The patient’s feet were monitored closely with biweekly reviews and casts were removed as early possible (after 8 weeks). This meant the degree of deformity was minimised and the patient was then able to ambulate and return to work after 3 months with accommodative custom-made shoes and custom-moulded total contact insoles.

**Authors**

Aishah Ahmad Fauzi MBBS, MRehabMed, is clinical specialist and lecturer, Department of Rehabilitation Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia. aishah_fauzi@um.edu.my

Chung Tze Yang MBBS(Malaysia), MRehabMed, is clinical specialist and senior lecturer, Department of Rehabilitation Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia.

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correspondence afp@racgp.org.au