

CONSEQUENCES OF MISDIAGNOSIS OF DIABETIC CHARCOT ARTHROPATHY OF THE ANKLE

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Abstract

Permanent deformity and disability can occur in diabetic Charcot arthropathy (neuropathic arthropathy) if not diagnosed and treated promptly. We report two patients with uncontrolled diabetes mellitus in whom the diagnosis of ankle neuro-arthropathy was delayed by up to six months, with misdiagnoses including ankle arthritis, osteomyelitis and cellulitis. The clinical scenario and appearances of the ankle and foot were typical of Charcot arthropathy. Unfortunately, both of them sustained ankle fracture-dislocation without a history of significant trauma. Both the patients were treated by ankle arthrodesis (fusion of joint). Prevention and early diagnosis of diabetic foot is the key to avoid the development of complications. In diabetic patients, a higher index of suspicion for the possibility of Charcot's disease is needed.

Ibrahim Med. Coll. J. 2010; 4(2): 83-86

Key Words: Diabetes mellitus; Charcot arthropathy; ankle fracture-dislocation; arthrodesis.

Introduction

Diabetic Charcot arthropathy, also known as neuropathic arthropathy, is a part of diabetic foot disease. Diabetic foot is usually associated with neuropathy which may lead to ulceration and neuroarthropathy. Diabetic neuropathic arthropathy is a destructive process of the bony components of a denervated joint. Diabetes mellitus is now the most common cause of neuro-arthropathy, which often manifests itself as a 'Charcot foot'. Patients usually have established diabetes with a sensory neuropathy, and present with painless or painful swelling and warmth in the region of the ankle and/or mid foot.^{1,2}

Charcot arthropathy is treated in early stages with immobilisation by a variety of casting procedures, and bisphosphonates have also been shown to accelerate resolution. Surgical procedures are often needed in case of fracture-dislocation and deformed foot. Accurate and early diagnosis is important, as without these treatments significant and permanent deformity may result.^{3,4} Two cases are presented here, in which a variety of misdiagnoses were made, resulting in deformed foot with fracture-dislocation of the ankle.

Case 1:

A 80-year-old man with type 2 diabetes of 15 years duration was referred to BIRDEM hospital OPD with a 7 days history of dull pain and swelling in his right ankle and foot following a minor trauma. He was unable to walk and his ankle was grossly deformed and unstable. There were no signs of inflammation, and sensation in the foot including pain and touch was



Fig. 1. Pre-operative X-ray: Fracture and dislocation in a right ankle Charcot joint

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Fig. 2. Post-operative X-ray: Ankle arthrodesis with plate and screws

reduced. He was known to have neuropathy and peripheral vascular disease with poor control of blood sugar. Intermittent pain and swelling in the foot had started six months previously. He was treated by doctors with diagnosis of ankle arthritis and osteomyelitis on different occasions. Several courses of antibiotics and analgesics were prescribed. Two separate ankle and foot X-rays were done in BIRDEM, which revealed gross osteopenia, soft tissue swelling and fracture-dislocation of the right ankle with bony fragmentation of the mid foot (Fig.1). His inflammatory markers of the blood were unremarkable.

History of trivial injury to the foot, clinical picture and investigations were considered compatible with Charcot arthropathy of the ankle and foot. As his right ankle was grossly unstable, he was treated with ankle arthrodesis (Fig.2). A 'T' shaped plate and five screws were used for arthrodesis of the ankle and immobilised



Fig. 3. Pre-operative X-ray: Fracture and dislocation in a left ankle Charcot joint



Fig. 4. Post-operative X-ray: Ankle arthrodesis with plate and screws

with a short leg plaster cast. His ankle fused over the next eight months. As a result, he could walk pain free but with some difficulty.

Case 2:

A 65-year-old man with type 2 diabetes of eight years duration was referred to BIRDEM hospital OPD with unexplained pain and swelling in his left foot and ankle. He was known to have retinopathy, neuropathy and peripheral vascular disease. His glycaemic control was poor. Pain and swelling in the ankle and foot had started six months previously. He could walk with the help of a crutch. His ankle was deformed and unstable, and sensation in the foot including pain and touch was reduced. He had been investigated by his general practitioner. As there was no history of trauma, his physician did not get a x-rays done. Alkaline phosphatase, C-reactive protein, urate level were slightly elevated and rheumatoid factor was normal. Possible diagnosis was considered as gout and cellulitis and several courses of antibiotics were prescribed.

X-rays of the ankle and foot were done in BIRDEM and revealed gross osteopenia, soft tissue swelling and fracture-dislocation of the left ankle with bony fragmentation and callus formation at the fracture site (Fig.3). His inflammatory markers of the blood were unremarkable. The clinical picture and investigations were considered compatible with a Charcot arthropathy of left ankle. Arthrodesis of the ankle was done with a 'T' shaped plate and five screws and immobilised with a short leg plaster cast. His ankle fused over the next twelve months.

Discussion

Neuroarthropathy, or “Charcot arthropathy”, is a diagnosis that predates the modern era of long-term survival with diabetes, having been first described in patients with tertiary syphilis.⁵ Charcot arthropathy is a severe destructive arthropathy which can occur in any patient with a sensory deficit. It was originally described in tertiary syphilis. Nowadays most cases occur in diabetics, but about 10% of Charcot patients have other causes, such as spina bifida, hereditary motor/sensory neuroopathy, post-traumatic sensory deficits, alcoholic peripheral neuropathy and sensory neuropathy of unknown origin.^{6,7}

Charcot arthropathy probably begins with a trivial trauma – about 30-50% of patients have a recognised injury, others probably have subclinical or no history of injuries. There appears to be many theories regarding pathogenesis of Charcot arthropathy, which includes osteoclast overactivity, bone vascular shunting and bone breakdown.^{8,9} As a result, demineralisation and joint destruction occur at the involved site. After a few weeks healing begins and after a few months there is usually a bony union with joint incongruity and deformity, which may lead to skin pressure and ulceration. Many patients with Charcot arthropathy, but not all, may feel pain and others have no pain or lessened pain because of loss of sensation.¹⁰

The progression of Charcot neuroarthropathy most often follows a predictable clinical and radiographic pattern, and is classified by the widely recognized Eichenholtz classification, which consist of 3 stages of fragmentation, coalescence and reconstruction.¹¹ Charcot arthropathy can be sudden and dramatic. It is one of the most difficult and intractable sources of excess mechanical pressure in the diabetic foot and can create large osseous prominences in various locations. Ulceration and rapid progression to osteomyelitis can follow. A large prospective study of risk factors for ulcerations in a population of male diabetic patients showed that the presence of Charcot arthropathy carried the highest relative risk of all of the factors examined, eclipsing even the absence of protective sensation.¹²

Clinical and radiographic signs of Charcot neuroarthropathy and osteomyelitis may overlap significantly, especially in the setting of an adjacent open wound. One challenge in managing Charcot neuroarthropathy is determining whether there is

superimposed osteomyelitis. The presence or absence of systemic signs of infection, such as fever, leukocytosis, elevated inflammatory markers, and increased blood glucose or insulin requirement, may not always be a reliable indicator. An underlying ulcer, particularly one that probes to bone, will provide an important clue because deep infection without evidence of skin compromise is rare. In doubtful cases sequential bone scans, computed tomography, or magnetic resonance imaging (MRI) may be more helpful than plain film radio-graphs. Duplex ultrasound may be used to rule out deep vein thrombosis.^{7,13}

Management is based on a variety of factors, including location, phase of the disease process, presence of infection, deformity, and comorbidities. Treatment should be guided by specific and realistic goals, depending on the severity of the disease and the patient’s functional capacity. This can vary from basic shoe modifications to major limb amputations. It is important to prevent the development of Charcot arthropathy by controlling blood sugar and early diagnosis of diabetic foot. Marked osteopenia has been noted in patients with Charcot neuroarthropathy and Bisphosphonates have shown promising short-term results in preventing bone resorption.¹⁴

The goal of treatment is to ensure a fully plantigrade foot. Immobilization is the mainstay of treatment in the unstable phases of Charcot neuroarthropathy, and the total-contact cast is the most widely used and accessible modality for maintaining stability and decreasing swelling.^{15,16} The decision for surgical intervention is multifactorial and is typically influenced by patient comorbidities and compliance, deformity location and severity, and the presence of infection, pain, or instability.¹⁵ Surgery may be required in severe instability in the early stages of the Charcot process, with progressive deformity, ulceration or infection, and persistent or recurrent ulceration in a foot with fixed deformity, despite optimal orthopedic management. Surgical options include soft tissue procedures (e.g. tendon-lengthening), exostectomy and arthrodesis. Unfortunately, amputation is occasionally required.

Arthrodesis (fusion of joint) may be the only option in severely unstable and deformed joint. Effective internal fixation techniques in arthrodesis include screws, pin, and plate fixation. Simon *et al.* showed promising results with fusion during the fragmentation stage,

with no major complications, and a return to regular shoe wear in a mean of 27 weeks.¹⁷ Correction of deformity may be a good option by midfoot osteotomy-fusion, triple fusion or tibio-talo-calcaneal fusion, depending on the level of deformity.^{6,18}

The cases described here had clinically typical Charcot arthropathy, yet a variety of misdiagnoses had been made. They had peripheral sensory neuropathy and there was history of recurrent swelling, pain and deformity of the foot and ankle. Due to diagnostic delay, their bone became osteopenic and fracture-dislocation of the ankle occurred following trivial trauma.

Conclusion

There is clearly a worrying lack of awareness of the possibility of Charcot arthropathy in diabetic patients presenting with acute foot and ankle swelling. A high index of diagnostic suspicion is required. Diabetic Charcot feet are often thought to be relatively rare, but this is not the impression received by our department. Indeed, more patients with the condition appear to be presenting. Strict metabolic control, prevention or minimisation of deformity by total contact casting or use of a diabetic walker boot and avoidance of weight bearing may prevent or delay the development of complication of diabetic arthropathy. A safe clinical policy would be to assume that diabetic patients with recent onset of foot or ankle swelling have neuroarthropathy until proved otherwise.

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