

Charcot's finger: Not to be forgotten

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ABSTRACT

Charcot joint disease or neuropathic arthropathy is a destructive arthropathy associated with diseases of the central nervous system. Diabetic neuropathy is the most common cause and largely affects the tarsal or ankle joints. Upper limb involvement is rare and there is only one well-described report of diabetic neuropathy of the digit in the literature. We present a case of Charcot arthropathy of the proximal interphalangeal joint of the ring finger secondary to diabetes mellitus.

Keywords: Charcot joint, neuropathic joint, septic arthritis, spontaneous deformity

INTRODUCTION

The manifestations of diabetes mellitus (DM) in the hand such as limited joint mobility, Dupuytren's contractures and trigger fingers are well-described in the literature. ¹ These complications are associated with duration of DM, level of control and presence of micro-vascular disease. Charcot arthropathy, even though a well-known complication of DM, is extremely rare in the hand. There have been only two reported cases involving the wrist, which were attributed to repeated hand strain. ^{2, 3} Only one case report described the involvement of the distal interphalangeal joint of the small finger in a patient with DM and this too was associated with increase hand strain from using crutches. ⁴

Unlike DM, other causes of neuropathic arthropathy are more likely to involve the upper limbs. Glenohumeral joint neuropathic arthropathy is commonly due to syringomyelia. Guille *et al.* in 1992 reported a case of Charcot joint involving both shoulders in a patient with congenital insensitivity to pain with anhidrosis. ⁵ Two recent case series of neuropathic joint of the elbow included only one diabetic subject. ^{6, 7} Deirmengian *et al.* (2001) reported a series of four other patients with syringomyelia, end-stage renal failure and polyneuropathy of unknown origin as their underlying medical conditions. ⁶ Kwon and Morrey (2006) reported patients with syringomyelia, congenital neuropathy, spinal tumour and another of unknown cause. ⁷

Leo Leung *et al.* (2003) reported a case of Charcot arthropathy of the digit secondary to DM following the use of crutches

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after lower limb surgery.⁴ Two other cases of neuropathic arthropathy involving the digits have been reported; one following replantation and another due to congenital insensitivity to pain.⁴ None of these subjects had underlying DM. To the best of our knowledge, our subject is the only reported case of spontaneous onset Charcot arthropathy of the finger secondary to diabetic neuropathy.

CASE REPORT

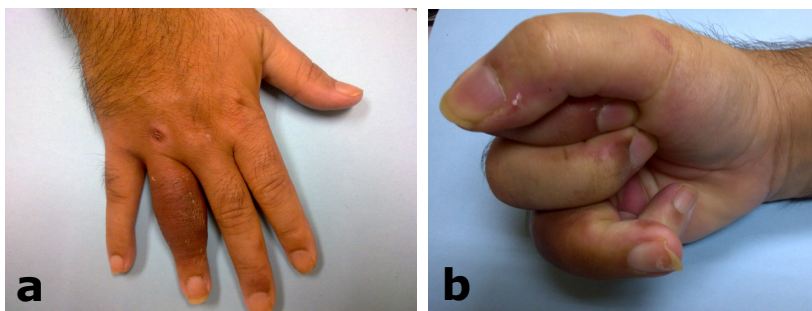
A 48-year-old left-hand-dominant man presented with progressive swelling of the right ring finger of two weeks duration. He was diagnosed with DM type II six years previously but had been non-compliant to treatment. There was no fever or constitutional symptoms and the subject could not recall any trauma prior to the onset of the swelling. He is a self-employed man who previously worked as a chef. His initial treatment included oral antibiotics from another hospital. He came to our hospital after one week to seek a second opinion.

On examination, the patient looked well. Inspection of the hand revealed a fusiform swelling of the ring finger at proximal interphalangeal (PIP) joint (Figure 1a) that was associated with erythema of the surrounding skin. The affected PIP joint was grossly unstable with crepitus detected on

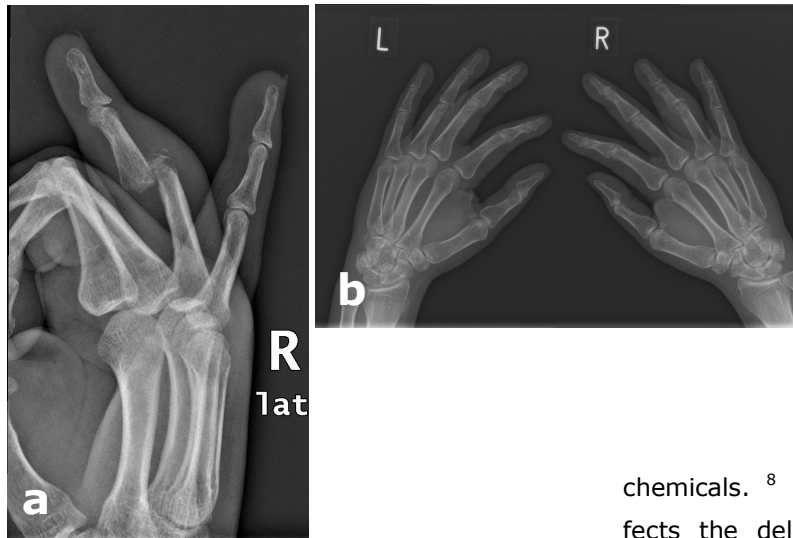
passive motion. Active flexion was limited to 90° and movement of the PIP joint was painless (Figure 1b). The range of motion of the metacarpophalangeal and distal interphalangeal joints were normal. Neurological examination of the fingers revealed reduced sensitivity to light touch and temperature stimuli with two-point discrimination being impaired to 8mm for all fingers. Reflexes as well as the vascular assessment were normal in both upper limbs. Further examination of the upper limb revealed no associated lymphadenopathy, swellings or deformity. Range of motion of the neck, shoulder and wrist were within normal limits. Examination of the lower limb revealed a left rocker bottom foot with tight achilles tendon, high medial arch and clawing of toes, consistent with Charcot foot arthropathy.

Radiographs of the hand revealed a destruction of the PIP joint which had dislocated with bony fragmentation and soft tissue swelling (Figure 2a). White cell count was not elevated and the serum C-reactive protein (CRP) was slightly elevated at 3.1 mg/dL (normal <0.5). Needle aspiration produced minimal fluid and this failed to isolate any organisms on cultures.

He was initially treated with intravenous antibiotics for two days which was dis-



Figs. 1: a) Right hand at presentation (dorsal aspect) showing swollen digit in extension, and b) Restricted flexion of proximal interphalangeal joint of ring finger (volar aspect).



Figs. 2: a) Radiograph showing destruction of proximal interphalangeal joint of ring finger with fragmentation (lateral view) at presentation, and b) Radiograph showing less fragmentation of proximal interphalangeal joint of right ring finger (oblique view) after 6 weeks.

continued when the diagnosis of Charcot joint was entertained. In subsequent follow ups, the swelling had reduced and the warmth and erythema resolved. Monitoring of serum CRP showed no elevation throughout the follow up visits. A repeat radiograph at six weeks was similar to the previous imaging but with less fragmentation (Figure 2b). Based on the clinical and radiological evidence, a diagnosis of Charcot joint disease of the digit secondary to DM was made. No further intervention was required for the patient. He was advised to avoid putting too much stress on the joint and be compliant to his DM medications.

DISCUSSION

DM is the leading cause of Charcot arthropathy, due to the nature of the disease where peripheral neuropathy is a common complication. Several factors have been proposed as the cause of the widespread nerve damage. High glucose concentrations can affect enzymes and proteins affecting their functions by binding to them.⁸ They also increase intracellular osmolarity, drawing water into cells which can alter concentrations of key

chemicals.⁸ Microvascular damage also affects the delivery of oxygen to peripheral nerves.^{1, 8} Once peripheral nerve damage is established, repeated trauma can lead to progressive destruction of the insensate joint.

Two theories have been described to explain the progressive nature of joint destruction in Charcot arthropathy. The neurological theory proposed that the loss of proprioception and deep sensation predispose to repetitive unrecognised trauma resulting in progressive joint degeneration and destruction.^{2, 8} The neurovascular theory that has been widely accepted theorise that the underlying neurologic changes lead to hypervascularity of the subchondral bone with increased osteoclastic resorption causing bone weakening, microfractures resulting in structural collapse and joint destruction.^{2, 8} These two theories are believed to work hand in hand in producing Charcot arthropathy.

Bayne and Lu (1998), and Lambert and Close (2005) reported a case each of diabetic Charcot arthropathy of the wrist in patients who had used crutches.^{2, 3} The former occurred in the right wrist of a man who had used crutches due to amputation of the left

lower limb. The latter case involved a woman who worked as a rose grower where increased load-bearing from using crutches, in addition to repetitive hand strain from rose pruning were thought to be contributory factors. The use of crutches was also reported in the case of Charcot arthropathy of the digit by Leung *et al.*⁴ These cases support the neurological traumatic theory.

Our case differed in that there was no clear evidence of repetitive trauma to the affected limb. This suggests that a traumatic event is not necessary to cause Charcot joint, and this may be explained by the neurovascular theory. As our patient was non-compliant with his DM treatment for a significant amount of time, the persistent high glucose concentration may lead to the development of Charcot arthropathy.

The differential diagnoses in such patients include septic arthritis, osteomyelitis, inflammatory arthritis and malignancy. Generally, our patient did not have any history of headache or neck pain and examination revealed no evidence of cranial nerve dysfunction, scoliosis or spina bifida occulta to suggest syringomyelia. There was no joint pain to suggest an inflammatory cause. The finger was relatively painless, which was disproportionate to the degree of distension and bony destruction seen on radiograph. These are classical features of neuropathic arthropathy. Although initially treated as infection, the condition did not progress after stopping antibiotics and the inflammatory markers remained stable, which is not consistent with either septic arthritis or osteomyelitis. Repeat radiographs showed no deterioration of bony changes. In fact there was less fragmentation

seen at six weeks. Other investigations such as uric acid, Rheumatoid factor and anti-nuclear antibody were also negative.

Treatment revolves around halting the progression of the joint destruction. Lifestyle modification, immobilisation of and avoiding further stress to the affected joint are usually adequate.^{4, 8} More importantly, the role of glucose control cannot be overemphasised. Recently, bisphosphonates have been used in the treatment of Charcot arthropathy and clinical trials have been promising.⁹ Unfortunately, it only works in the acute destructive phase and no trial has been performed for Charcot joint involving the upper limbs. Surgical intervention is considered when the acute phase has settled. Arthrodesis is an option if further deformity and instability occurs, leading to deterioration of function.^{4, 8}

In conclusion, the diagnosis of Charcot joint disease of the fingers and hand should be considered in patients with DM once infection and malignancy has been excluded. Glucose control is important in the management.

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