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Charcot Arthropathy: A Case Study

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Abstract

Charcot Arthropathy is a serious complication of Diabetic neuropathy, usually affecting foot or ankle. It causes widespread destruction of affected joints and bones around them leading to severe deformities that may require major amputation. Here, we report the case of a 62 year-old male who presented with severe left foot deformities and swelling on the left big toe that developped over a short period of time. His primary presentation raised the suspicion for a bone tumor or cellulitis which then revealed by X-Ray that it's a Charcot foot necessitating foot fixation, strict glycemic control and treatment with Biphosphonates. This report will therefore serve as a reminder for clinicians to keep in mind Charot Arthropathy in diabetic patients with peripheral neuropathy even if it doesn't present in a typical manner.

Keywords: Charcot, arthropathy, diabetes, trauma, destruction, joints, deformity, sensation, neuropathy, inflammation, chronic, Fixation, Osteodegenerative, ambulation, uncontrolled.

Introduction

Charcot Arthropathy is a serious but rare progressive condition that can affect 0.15 – 2.5% of diabetic patients especially those who developed peripheral neuropathy. Despite the voluminous work reported in literature on CA, very few studies mention the prevalence of this disabling disease. The actual incidence of CA may be greater than what is reported, as in many cases, the clinicians fail to diagnose or are late to diagnose this serious complication. Charcot affects the bones, joints, and soft tissues of the foot or ankle. Whilst the exact pathophysiology remains debated, a multifactorial pathogenesis seems likely predominating. This involves repetitive micro-trauma in a foot with impaired sensation and neurovascular changes caused by pathological innervation of the blood vessels leading to bones deformities. Clinical features include signs of inflammation, profound unilateral swelling, an increase in local skin temperature and bone resorption in an insensate foot may be present.

In advanced cases with considerable mid-foot destruction, a typical "rocker bottom" appearance is seen. In most cases, progression of deformity takes a more chronic form and is seen over a period of months, to years.

This case report describes a case of a chronic destructive Charcot foot in a patient with uncontrolled diabetes, which was initially managed as a case of bone tumor.

Case Report

A 62 year old male with a background of Type 2 Diabetes Mellitus, peripheral neuropathy and obesity presented with complain of numbness and decreased sensation of forefoots and a mass in the arch of left foot. Eight days prior, he sustained a fall whilst walking but denied any injury, swelling or pain at that time. He said his left big toe was totally straight but now has deformed (into Morton) and his 2nd toe looks bigger due to big toe deformation and often remains bend in shoes which he doesn't feel, but when removing the shoes he often finds it bent. The patient denies any pain in the deformed toe but a little

feeling of warmth in the pathological area {figure.1}.

Figure 1

Examination revealed a warm, swollen left foot with a 2 mm healing ulceration on the plantar surface and a lumpy sensation on the left big toe. Blood tests showed normal white blood cells count, elevated C-reactive Protein (CRP-67) and a thrombocytosis. His fasting blood glucose level was 221 mg/dl with features of uncontrolled diabetes.

Plain film X-Ray revealed complete fracture dislocations of the 1st , second and fifth tarsometatarsal joints with large bone deformity on the left arch of the foot as well as another deformity on the right arch and lower calcaneus {Figure.2}. This was correlated with bone CT (computed tomography) and MRI (Magnetic Resonance) imaging, with interval changes noted to be of chronic onset.

Figure 2

Discussion

Charcot foot is a well-documented complication of diabetic neuropathy. The mechanism of this is not fully understood but two theories exist – namely, the neurovascular and neurotraumatic theories. In the neurovascular theory, an underlying autonomic neuropathy leads to hypervascularity, osteolytic changes and demineralization. However, the neurotraumatic hypothesis indicates that unperceived trauma to an insensate extremity leads to continued bony destruction on ambulation, which worsens and progresses. These theories imply that a sufficient time frame is required for established Charcot foot destruction to develop.

Typical progression of Charcot foot occurs over months to years, although rare acute onsets have been reported. However, the progression seen in our patient, from normal X-Ray appearances to established, acute Charcot foot with considerable mid-foot destruction over a period of just 10 days is a rarely described progression of Charcot arthropathy. The combination of local skin warmth with decreased sensation in the left foot and feeling of a bony lump on the left arch raised the suspicion of a bone tumor, but X-Rays as well as CT Scan of the foot beside the uncontrolled diabetes in this patient made Charcot foot a more possible diagnosis.

Early recognition of Charcot Arthropathy is a mandatory to ensure improved outcomes. It is important to differentiate between acute Charcot foot, infection and bone tumors through clinical investigations as well as patient medical history. The presence of ulcers should raise the suspicion of infection or osteomyelitis, and therefore, the importance of early imaging cannot be overstated. Modestly raised inflammatory markers (CRP-68) in this diabetic patient along with left foot deformities suggest an acute Charcot foot than an infectious process. The latter frequently presents with high laboratory values.

The initial management of the patient was fixation with a total contact leg cast, followed by open reduction and internal fixation (ORIF) with locking plates, combined with lengthening of Achilles tendon to permit full dorsiflexion and subsequent re-casting.

Due to osteodegenerative nature of CN, all attempts at pharmacological treatment have focused on anti-osteoporotic drugs so we started the patient on an Alindronate beside a more strict control of his diabetes. His post-operative progress was satisfactory and he is currently non-weight bearing for 6 months post-ORIF.

Conclusion

The case highlights an important clinical scenario that can be misdiagnosed in a diabetic patient. A clinical presentation of unilateral foot swelling, erythema, warmth and decreased sensation should raise suspicion for Charcot Arthropathy even in the absence of trauma history. I hope this will be a helpful reminder to clinicians when dealing with similar presentations.

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