



Charcot Joints: A Case Study

KEYWORDS

Charcot joint, Neuropathy, TCC, CROW

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ABSTRACT

Charcot joint is an uncommon degenerative osteoarthopathy associated with decreased sensory innervations of the involved joint. It is characterised by the "6 D's" i) Distended joints ii) Density increases iii) Debris production iv) Dislocation v)Disorganisation vi) Destruction. Early identification and treatment of charcot process can minimize potential foot deformity, ulceration and loss of function and prevents amputation or death. Here we present a case of charcot joint responding to pressure relieving methods.

Introduction:

Neuropathic joints often called charcot joints are caused by loss of sensation in the joint so that it is severely damaged and disrupted. The damage and disruption of the joint occurs which indicates the diagnosis in a patient with decreased sensory innervations. It is degenerative arthropathy. Historically, neuropathy of knee joints was most frequently caused by syphilis and neuropathy of shoulder joints was most often caused by syringomyelia. Nowadays charcot foot occurs usually in a patients of diabetic neuropathy. Other predisposing conditions are alcoholic neuropathy, sensory loss caused by cerebral palsy or leprosy and congenital insensitivity to pain. In 1968, Jean-Martin Charcot described neuropathic joint disease in patients with tabes dorsalis; hence, the condition named after him.

Charcot joint is prevalent in 0.8 to 7.5% of diabetic patients with neuropathy; 9 to 35% of these affected patients have bilateral involvement^{1,2}. Most of these patients have had poorly controlled diabetes mellitus for 15 to 20 years.

Case Report:

In March 2008, a 56 years man presented with history of progressive loss of sensations and slapping of feet while walking since 15 years. History of ulcers on both feet was present since 7 - 8 years. In 1993, he was misdiagnosed as Hansen's disease and received treatment for six months. There was no consanguinity and the parents and siblings were normal. On CNS examination, higher mental function, cranial nerves were normal. Motor system examination revealed wasting of distal muscles in both upper & lower extremities. Tendon reflexes at knee and ankle joints were diminished and rest were normal. Sensory system examination revealed loss of superficial sensations in glove and stocking distribution. Vibration and joint position sensations were preserved. Peripheral nerves (ulnar and peroneal) were thickened.

Investigations revealed normal haemogram and urine routine examination. Blood sugar examination, renal function tests and hepatic function tests were normal. Varicose vein was present on the left ankle. No evidence of venous / arterial thrombosis. Nerve conduction velocity study showed severe generalised sensory motor axonal polyneuropathy. Investigations for collagen vascular disease were negative. Dermatologist did not favour Hansen's disease. Diagnostic consideration was in favour of autosomal recessive sensory motor polyneuropathy.

In July 2010 patient presented with abscess on right foot for which debridement was done. In October 2010 patient had right foot plantar ulcer. X-ray right foot showed no evidence of osteomyelitis.(Fig 1) Plantar casting was done for non weight bearing.

Figure 1: Stage 0 : X Rays Showing Prefragmentation Stage



In July 2011 patient presented with swelling on lateral aspect of right foot which went on increasing gradually and not responded to antibiotics. In November 2011 X-ray right foot was done and diagnosed as charcot joint (Fig 2) and advised charcot restraint orthotic walker (CROW).After using CROW, patient is improving clinically. Swelling subsided, trophic ulcers healed.X ray of August 2012 shows coalescence stage of charcot joints.(Fig 3)

Figure 2: Stage 1 : X Rays Showing Fragmentation Stage



Figure 3: Stage 2 : X Rays Showing Coalescence Stage

**Discussion:**

Charcot joint is a progressive degeneration of weight bearing joint, a process marked by bony destruction, bone resorption and eventual deformity. The tarsometatarsal (Lisfranc's) joint is the most common site for charcot joint (medial joints affected more than lateral).The distribution of charcot joint is 70% at the midfoot and 15% at the forefoot and rear foot; it is usually contained in one area. Nearly 50% of the patients with neuropathy had an associated plantar ulcer².

Two theories explain the pathogenesis of charcot joint³. 1) Neurotraumatic theory: A joint without proper innervations is subject to repeated trauma and it continues to damage the joint overtime. 2)Neurovascular theory: Loss of sympathetic vascular tone leads to increased blood flow to the joint, causing imbalance in metabolism. Over time the joint becomes osteopenic and mechanical stress leads to bony destruction. In reality both these mechanisms probably play a role in the development of charcot joint.

Radiologically charcot joint is of two types 1)Atrophic –“Licked candy stick” appearance commonly seen at the distal aspect of the metatarsals. There is bone resorption and osteolysis. It is usually localized to the forefoot. 2)Hyper-trophic- According to Yochum and Rowe, “6 D's” of hypertrophy are i) Distended joints ii)Density increases iii) Debris production iv) Dislocation v)Disorganisation vi) Destruction⁴. It usually occurs at the midfoot ,rarefoot or ankle joint and is traditionally defined according to the Eichenholtz classification system^{5,6}. The first stage is developmental or fragmentation stage (acute charcot) and characterised by periarticular fracture and joint dislocation leading to unstable, deformed

foot. Patient in the coalescence stage (subacute charcot) presents with resorption of bone debris. The consolidation or reparative stage (chronic charcot) is associated with restabilization of foot with fusion of involved fragments. This leads to the return of a stable, although deformed foot. An updated version of this classification system⁷ identifies a prefragmentation (or acute inflammatory) stage zero. This is the stage when early diagnosis and intervention are critical to prevent further long range sequelae⁸⁻¹⁰. This updated classification system also more closely relates clinical findings to treatment options.

Because trauma is not prerequisite for charcot foot, a patient with diabetes and neuropathy, erythema, edema, increased temperature of the foot and normal radiograph most likely has an acute charcot process. These patients are afebrile, have stable insulin requirement and normal white blood cell counts and often have no break in skin integrity. These are all conditions that make infection unlikely.

In charcot foot plantar ulcers may be present. It is often difficult to differentiate osteomyelitis from charcot joint, as they may have similar tagged WBC scan and MRI features (joint destruction, dislocation, edema). Synovial and bone biopsies might be necessary for definitive diagnosis^{5,11}.

Treatment of charcot joint is by pressure relieving methods, as follows.1) Total contact casting (TCC) - in active phase when swelling and erythema present. (upto four months)¹² 2) Charcot restraint orthotic walker (CROW) - After resolution of swelling and erythema and achieving radiographic stability, this device is used for six months to two years until a stable foot is obtained. Patients can then be fitted to extra depth shoes with custom insoles or orthotics to accommodate any residual deformity. Return to conventional foot gear may not be possible in all cases⁵.

Surgical intervention is required in the form of exostosectomy for residual exostosis and joint stabilisation for subluxation of joint in case of markedly unstable extremity⁵.

Physicians should always educate their patients about the proper care of the neuropathic foot and the use of orthotic devices or custom footwear. In these patients, any minor trauma requires careful observation because of the tendency to proceed to charcot process. Early identification and treatment of charcot process can minimize potential foot deformity, ulceration and loss of function and prevents amputation or death⁵.

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