

Divergent Lisfranc's Dislocation and Fracture in the Charcot Foot: A case report

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A case report discusses the presentation, diagnosis and treatment of a 45 year old diabetic man with a divergent, Lisfranc's dislocation of the first metatarsal in a Charcot foot. The patient also presents with associated laterally subluxed lesser metatarsals and multiple fractures. Conservative treatments such as TTC or total contact casting, prefabricated pneumatic walking brace (PPWB), patellar-tendon brace and CROW custom orthosis are discussed.

Key words: Charcot foot, Lisfranc's dislocation, fracture

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Charcot joint in the foot typically refers to painless fracture and dislocation of the foot in patients without normal sensation or feeling in their foot. Loss of sensation in the foot for any reason can be responsible for developing a Charcot fracture, although this is most commonly seen with neuropathy. Neuropathy of the nerves that affect the foot is most commonly seen with diabetes, but is associated with other diseases as well. Treatment depends on the severity of the condition and the amount of deformity that is present.

We present a 45 year old diabetic man with Lisfranc's dislocations along with fracture of 2,3,4,5 metatarsals. These patients frequently present complaining of a deep, aching, non-descript pain in the ankle joint that worsens with activity.

Case Report

A 45 year old man came to our out patient department with complaints of swelling in the left foot for 20 days duration. There was no history of trauma, fever or constitutional symptoms. The patient is a non-insulin dependent diabetic on oral hypoglycemic drugs. The swelling was diffuse, red, warm, non tender on palpation. (Fig. 1)

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Figure 1 Diffuse swelling is noted to the left foot in a typical, Charcot presentation. The swelling is diffuse and non-painful.

There was no sinus or active discharge. Radiographs of the foot showed fractures at the neck of 2,3,4, and shaft of 5 metatarsal along with divergent type of Lisfranc's dislocation, bony destruction, fragmentation, joint subluxation and bony remodeling. (Figs. 2,3) Random blood sugar was 201mg/dl. C-reactive protein was negative; Erythrocyte sedimentation rate was 12mm/hr. Other blood parameters were normal. The patient was treated with a total contact cast.

Casts were replaced approximately every 2 weeks. The foot was inspected, and cutaneous temperature measurements were done. Serial plain radiographs were taken approximately every month. Casting lasted for 3 months. We used a patellar tendon bearing brace in addition to custom-molded footwear after the cast. The brace was eliminated from the regimen after six months. Thereafter, continued use of custom footwear to protect and support the foot was given.



Figure 2 Oblique views reveal a divergent, Lisfranc dislocation of the first metatarsal with associated lesser metatarsal fractures.



Figure 3 Dorsoplantar view reveals complete dislocation of the first metatarsal at the medial cuneiform articulation. Typical TMT joint fracture, fragmentation, joint subluxation and bone remodeling is seen.

Discussion

Charcot neuropathy is a progressive deterioration of weight-bearing joints, usually in the foot or ankle. It is a condition of acute or gradual onset and, in its most severe form, causes significant disruption of the bony architecture of the foot. It often results in foot deformities and causes abnormal pressure distribution on the plantar surface, foot ulcers and, in some cases, requires amputation. The exact pathogenesis is unknown, but underlying sensory neuropathy is nearly universal. Arteriovenous shunting due to autonomic neuropathy is also thought to play a role. Repeated unrecognized microtrauma or an identifiable injury may be the inciting factors of Charcot foot. Approximately 50 percent of patients with Charcot foot will remember a precipitating event such as a slip or a trip, or they may have had unrelated surgery on the foot as an antecedent event. In approximately 25 percent of patients, a similar problem ultimately develops on the other foot.^{1,2}

The process is characterized by pathologic fractures with an exuberant repair mechanism and is associated with mixed peripheral neuropathies. The common denominator in these various conditions is that motor function is not as severely affected as are sensory modalities in the patient.^{3,4,5} The Charcot foot in the diabetic patient is a progressive condition that is not confined to bones but affects all of the tissues in the lower extremity. It is often confused with osteomyelitis and massive infection of the foot necessitating early identification and management to prevent amputation of the lower extremity. With the advent of advanced surgical techniques and a better understanding, the physician may be optimistic with the treatment of this condition. By thoroughly understanding the etiologic factors and deforming forces, treatment can be planned for each specific patient.

The etiology of Charcot joints has been argued by many authors. Two theories (neurotraumatic and neurovascular) explain the pathogenesis of Charcot foot.⁴

The neurotraumatic theory attributes bony destruction to the loss of pain sensation and proprioception combined with repetitive and mechanical trauma to the foot. The neurovascular theory suggests that joint destruction is secondary to an autonomically stimulated vascular reflex that causes hyperemia and periarticular osteopenia with contributory trauma. Intrinsic muscle imbalance with increased heel and plantar forces can produce eccentric loading of the foot, propagating microfractures, ligament laxity and progression to bony destruction.⁶ Neuropathic arthropathy is prevalent in 0.8 to 7.5 percent of diabetic patients with neuropathy; 9 to 35 percent of these affected patients have bilateral involvement.^{7,8} The higher prevalence is seen in referral-based practices. Most patients with neuropathic arthropathy have had poorly controlled diabetes mellitus for 15 to 20 years. Clinical findings in patients with an acute Charcot process include warmth, erythema and swelling.^{13,14,15} Pain and tenderness are usually absent because of sensory neuropathy, which is universal and is probably a component of the basic pathogenesis of the Charcot foot. Cellulitis should be considered in any patient with diabetes. Missing the diagnosis of Charcot foot can be disastrous since failure to initiate proper treatment of the Charcot foot exacerbates the problem. We strongly recommend that the diagnosis of acute Charcot foot be considered in any patient with diabetes and unilateral swelling of the lower extremity and/or foot. The existence of little or no pain can often mislead the patient and the physician.

The tarsometatarsal (Lisfranc's) joint is the most common site for arthropathy, with initial involvement usually occurring on the medial column of the foot. The distribution of neuropathic arthropathy is 70 percent at the midfoot and 15 percent at the forefoot or rearfoot; it is usually contained in one area.

Nearly 50 percent of patients with neuropathy had an associated plantar ulcer.^{8,9}



Figure 4 Total Contact Cast is used for immobilization and protection of the Charcot foot. It is commonly used as initial conservative treatment in the acute Charcot episode.

Bony destruction, fragmentation, joint subluxation and bony remodeling are considered radiographic hallmarks of the disease. These radiographic changes take time to develop, however, and may be absent at the time of presentation. The initial radiographic findings can be normal, making the diagnosis difficult but, if a

Charcot foot is strongly suspected from the clinical presentation, treatment should be initiated and serial radiographs should be taken. Biopsy is the definitive test for the diagnosis of Charcot joints. The specimen will demonstrate the presence of multiple shards of bone and cartilage embedded within the deeper layers of the synovium. If osteomyelitis is of concern then a bone biopsy is essential for proper and accurate diagnosis.

The proper treatment for a hot, swollen foot in a patient with sensory neuropathy is immobilization. We believe that the best form of immobilization is a total contact cast, when available. Strict immobilization and protection of the foot (most often in a total contact cast) is the recommended approach to managing the acute Charcot process.^{11,12,13,14,15} We used the total contact cast for our patient which allowed some measure of ambulation for the patient and prevented the progression of deformity. (Fig. 4) Charcot fractures that are not treated progressively, typically lead to marked deformity and skin ulceration over the new bony prominence. Casts should be replaced approximately every one to two weeks. The foot should be inspected, and cutaneous temperature measurements should be made. Serial plain radiographs should be taken approximately every month during the acute phase. Casts should be kept on until the active phase of the Charcot process is complete, as evidenced by temperature normalization and radiographic stability. Casting usually lasts from three to six months. The initial post-cast phase usually includes the use of some sort of a brace to protect the foot.

We used a patellar tendon bearing brace in addition to custom-molded footwear. The brace can sometimes be eliminated from the regimen after six to 24 months. Thereafter, continued use of custom footwear to protect and support the foot is essential.

An alternative to TCC is a prefabricated pneumatic walking brace (PPWB), which has been found to decrease forefoot and midfoot plantar pressure in the treatment of neuropathic plantar ulceration.^{11,12,13,14,15} (Fig. 5)



Figure 5 The alternative to the total contact cast is the PPWB or prefabricated pneumatic walking brace. (Courtesy [Aircast Corp.](#)®)

Benefits include easier wound surveillance, ease of application and the ability to use several types of dressings. Use of the PPWB is limited in patients who have severe foot deformity or who are noncompliant. After swelling and erythema resolve and radiographic stability has been achieved, the TCC can be changed to a CROW, an ankle foot orthosis or a patellar tendon-bearing brace, depending on residual anterior edema. If anterior edema persists, the CROW full-enclosure system is used. (Fig. 6) This device is used for six months to two years, until a stable foot is obtained.



Figure 6 The CROW or Charcot Restraint Orthotic Walker (A) and the patellar tendon-bearing brace (B). The CROW is a custom molded device that when properly constructed can improve plantar off-loading up to 50 percent. It can be used for 6 months to 2 years until the foot is stabilized. The patellar tendon-bearing brace can reduce offloading pressures of up to 90 percent. [16]

Patients can then be fitted for extra-depth shoes with custom insoles or orthotics to accommodate any residual deformity. Return to conventional foot gear may not be possible in all cases.

Other treatments for the Charcot process have included electrical bone stimulation or low-intensity ultrasonography during the acute phase to enhance healing.^{11,12} Another study found that use of a bisphosphonate (pamidronate) resulted in decreased erythema, decreased temperature and decreased Charcot activity.^{12,13,14} Additional controlled studies are needed to further evaluate the effectiveness of these treatments.

While it is still unknown why some patients with diabetes develop a Charcot process and others do not and more interestingly why some patients only develop this condition in one of their feet, an introspective review is necessary.

The literature on Charcot foot is huge and refers, not specifically, to every joint and metatarsals. The fact that 2,3,4,5 metatarsal involvement has not been extensively described, does contribute a base for our observation.

In summary, the Charcot foot commonly goes unrecognized, particularly in the acute phase, until severe complications occur. Early recognition and diagnosis, immediate immobilization and a lifelong program of preventive care can minimize the morbidity associated with this potentially devastating complication of diabetic neuropathy. If unrecognized or improperly managed, the Charcot foot can have disastrous consequences, including amputation. A lifelong program of patient education, protective footwear and routine foot care is required to prevent complications such as foot ulceration.

With proper planning, timing and knowledge of all facets of diabetic neuropathy, many patients may retain their foot and benefit from its function.

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