Charcot Arthropathy

OVERVIEW

Charcot arthropathy is the progressive destruction of the foot that can be stimulated by isolated or cumulative, repetitive trauma in patients with peripheral neuropathy. While diabetic neuropathy is the most frequent cause of Charcot deformity, other causes include syringomyelia, alcoholism, chemotherapy-induced neuropathy, syphilis, and multiple sclerosis. This results in an inflammatory response that can lead to bone resorption and potential deformity of the foot.

PATHOPHYSIOLOGY

Peripheral neuropathy, regardless of cause, is the underlying inciting event that leads to Charcot deformity. With respect to diabetes, neuropathy is the result of glycosylated hemoglobin affecting conductivity of peripheral nerve fibers. Patient can often recall a specific traumatic event and most events are typically low energy such as a misstep off a curb. However, it can also occur without any overt trauma. In others, Charcot deformity can occur due to a motor imbalance as the neuropathy preferentially affects smaller motor nerves and innervated muscles. This leads to a motor imbalance in which the stronger plantarflexors of the foot overpower the smaller dorsiflexors. In addition, contracture of the gastrocnemius-soleus creates both a static and dynamic equinus deformity. This can result in pathologic bending moments being created along the transverse tarsal and tarsometatarsal levels of the foot and mechanical overload predisposing to destructive changes of the bone often seen at these levels. Further autonomic microvascular changes can also occur leading to increased blood flow, stimulation of osteoclasts with resultant focal osteopenia that can lead to deformity with repetitive loading.

CLASSIFICATION

The Eichenholtz classification is the most commonly utilized to describe charcot changes within the foot. Stage I is the fragmentation phase and is characterized by destruction of the bone, fractures, joint subluxation and potential risk of instability. Stage II is a transitional

period of coalescence during which time osseous destruction can occur with resultant deformity leading to a risk of ulceration. Stage III is the period of consolidation in which the bone heals in a suboptimal position with deformity. This can lead to shoewear difficulty, increased risk of ulceration, and deep infection. Anatomic classifications have also been utilized to describe the location and severity of the deformity.

CLINICAL PRESENTATION

Loss of protective sensation is present in patients with Charcot arthropathy. In particular, screening with the Semmes-Weinstein 5.07 monofilament should be performed in any patient suspected of neuropathy. Patients with an active Charcot episode will often present with a warm, erythematous, and edematous foot. Often, simple elevation can help to distinguish a Charcot foot from an active infection. It is important to realize, however, that patients with a Charcot deformity can often present with osteomyelitis or abscess that can complicate the clinical picture. However, Charcot patients often lack clinical (ie, fever, abscess, etc) and laboratory evidence of infection.

IMAGING STUDIES

Imaging studies should include radiographs of the foot. Prior radiographis, if available, are helpful as well to identify structural changes within the foot or deformity progression. Recent studies have demonstrated that even in stable Charcot arthropathy, there can be subtle progression over time that includes loss of the medial longitudinal arch predisposing patients to ulcer development. Computed tomography scans can be obtained primarily for surgical planning and also to evaluation for osseous union following reconstruction. MRI studies can also be performed when one suspects an abscess or osteomyelitis. However, Charcot arthropathy is primarily a clinical diagnosis.

TREATMENT

Initial treatment is directed at total contact casting that is aimed at reducing swelling and limiting the osseous fragmentation that can occur leading to structural changes within the foot. Casting is frequently changed on a weekly basis until the swelling and warmth have subsided. Infection, if present, must be treated in a timely fashion via debridement and wound care. Once the initial episode has subsided, patients can be transitioned into

accommodative footwear that can include orthoses, custom shoes or a CROW (charcot restraint orthotic walker) device.

Surgical treatment is often employed for those patients who fail conservative management with refractory ulceration or deformity that is unable to be braced. There is no one particular treatment as each Charcot deformity is unique. Methods of surgical intervention include simple exostectomy, arthrodesis via internal fixation or external fixation. Each surgical plan is unique, tailored to correction of the deformity to create a stable, plantigrade foot that is braceable.

CONCLUSION

Charcot arthropathy is a challenging entity to treat and each treatment must be tailored to the individual needs of the patient. It is important to have a high-degree of suspicion when evaluating the Charcot patient and to initiate treatment in a timely fashion to reduce the risk of limb-threatening complications.

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