Acute Charcot’s Arthropathy of the Foot and Ankle

Charcot’s joint (neuropathic osteoarthropathy) is a progressive condition affecting the musculoskeletal system and is characterized by joint dislocation, pathologic fractures, and often debilitating deformities (Figs. 1 and 2). The condition most commonly occurs in patients with diabetes mellitus who have severe peripheral neuropathies. The prevalence of Charcot’s joint is variable, ranging from 0.16% of all patients with diabetes to as high as 13% of patients receiving care at a high-risk diabetic foot clinic. The frequency of diagnosis of this condition appears to be increasing as a result of increased awareness of its signs and symptoms.

The Etiology of Neuropathic Osteoarthropathy (Charcot’s Joint)

Neuropathic osteoarthropathy was first reported by Musgrave in 1703. He described it as an arthralgia secondary to venereal disease. In 1868, the noted French neurologist Jean-Martin Charcot became the first investigator to concisely describe the neuropathic component of the disease. Charcot linked the degenerative condition to syphilis, which was then a common malady. Syphilis was the disease most commonly associated with this type of arthropathy until 1936, when Jordan linked it to diabetes mellitus. Since these first descriptions, numerous theories on its etiology have been promoted.

Charcot believed that neuropathic osteoarthropathy was secondary to deficiencies in trophic centers in the spine. It was for this concept, along with his brilliant description of the malady, that neuropathic osteoarthropathy was subsequently renamed “Charcot’s arthropathy.” This spinal-centric view of Charcot’s arthropathy, however, was not shared by all of Charcot’s contemporaries. Volkman, Virchow, and other members of the “German school” vehemently opposed Charcot’s theory, which they believed was based solely on observation and assumption. They believed that the etiology of Charcot’s arthropathy was neurotraumatic in nature. That is, an insensate foot subjected to trauma would fracture and heal with exuberant bone formation. In testing this theory, Eloesser, in 1917, sectioned the posterior nerve roots to the forelimbs in 38 cats. Following a period of activity, Eloesser noted neuropathic

Key Words: Arthropathy, neurogenic; Joint diseases; Lower extremity, ankle and foot.


David G Armstrong

Lawrence A Lavery
With the possible exception of bony changes in 71% of the animals. Six decades later, Finsterbush and Friedman repeated Eloesser’s experiments using a rabbit model. After sectioning the posterior nerve roots, the rabbits’ hind limbs were casted. A difference was noted in the response to immobilization between normal and denervated groups. Finsterbush and Friedman concluded that trauma was important but not the primary factor leading to the deterioration of insensate joints.

Finsterbush and Friedman’s work opened the way for further conjecture about the nature of Charcot’s arthropathy. Subsequent investigators hypothesized that trauma alone could not explain the sometimes striking osteopenia seen in patients with Charcot’s arthropathy. This hypothesis led to the notion that increased blood flow was at least partially responsible for the arthropathy, causing a resorption of bone and a subsequent weakening of the supporting structure. Thus, fractures could be caused by even trivial stress. In an attempt to lend more credence to this concept, Edmonds and coworkers, using scintigraphy, demonstrated that blood flow within bone was greater when neuropathy was present. This neurovascular theory has gained a great deal of favor among many clinicians treating patients with this condition.

The actual etiology of Charcot’s arthropathy may lie somewhere between these neurovascular and neurotraumatic theories. The current theory suggests that, following the development of autonomic neuropathy, there is an increased blood flow to the extremity, resulting in osteopenia. Subsequently, motor neuropathies result in muscle imbalance, which places abnormal stress on the affected extremity, while sensory neuropathy renders the patient unaware of the often profound osseous destruction taking place with each step during ambulation.

Diagnosis of Acute Charcot’s Arthropathy

The initial diagnosis of acute Charcot’s arthropathy is often based on profound unilateral swelling, locally increased skin temperature, erythema, joint effusion, and bone resorption in an insensate foot. These characteristics, in the presence of intact skin and loss of protective sensation, are often pathognomonic of acute Charcot’s arthropathy. Armstrong et al have also noted that there is some degree of pain in an otherwise insensate extremity in over 75% of patients with acute Charcot’s arthropathy. Diagnosis is complicated by the fact that 40% of patients with acute Charcot’s arthropathy have a concomitant ulceration, thus raising the issue of whether there is contiguous osteomyelitis.

When faced with a warm, edematous, erythematous, insensate foot with a concomitant wound, clinicians may find it difficult to differentiate between acute Charcot’s arthropathy and osteomyelitis solely on the basis of plain radiographs. Additional laboratory studies may prove to be useful in arriving at a correct diagnosis. The white blood cell count will often be elevated, with a left shift, on differential analysis in patients with acute osteomyelitis.

DG Armstrong, DPM, is Assistant Professor, Department of Orthopaedics, University of Texas Health Science Center, 7703 Floyd Curl Dr, San Antonio, TX 78284-7776 (USA) (armstrong@usa.net). Address all correspondence to Dr Armstrong.

LA Lavery, DPM, is Associate Professor, Department of Orthopaedics, University of Texas Health Science Center.
Joint from osteomyelitis include bone scans utilizing technetium bone scans are expensive and nonspecific in assisting in the differentiation between osteomyelitis and acute Charcot's arthropathy. We do not believe that technetium scanning is very useful for the diagnosis of acute pedal sequelae to peripheral neuropathy. Indium scanning, although expensive, has been shown to be far more specific. Indium-111 scintigraphy may be used in two instances. First, indium-111 scintigraphy may be used to initially assist in differentiation between osteomyelitis and Charcot's joint in the presence of a pedal ulcer. Second, several weeks following debridement of osteomyelitic bone, indium-111 scanning may provide some benefit in determining the adequacy of bony resection. If an indium-111 scan is returned positive, a bone biopsy is indicated to confirm the diagnosis of osteomyelitis and rule out Charcot's joint. If a scan is returned negative, the presumptive diagnosis is Charcot's joint until proven otherwise. Additional studies currently used for assistance in differentiating Charcot's joint from osteomyelitis include bone scans utilizing white blood cells labeled with technetium hexamethyl propylenamine oxime and magnetic resonance imaging.

Although imaging studies may be useful at many centers, including our own, we prefer to use a sterile blunt probe. Probing to bone, combined with radiographic and clinical evaluation, may be the most practical and cost-effective means of diagnosing osteomyelitis prior to surgical debridement and definitive bone biopsy. If a wound is probed directly to bone, osteomyelitis is frequently assumed. This diagnosis may then be confirmed with a bone biopsy. A bone biopsy is currently the "gold standard" by which all other diagnostic modalities are measured. Bone biopsies have a very low complication profile and are less expensive than many advanced imaging techniques. A positive histologic diagnosis of Charcot's joint is less clinically important than a negative diagnosis of osteomyelitis because a prolonged course of parenteral antibiotics or surgical ablation may be obviated by a negative diagnosis. Nonetheless, a biopsy consisting of multiple shards of bone and soft tissue embedded in the deep layers of synovium is pathognomonic for neuropathic osteoarthropathy.

The Classification of Charcot's Arthropathy

The most common classification system used in the treatment of patients with Charcot's arthropathy was described by Eichenholz in 1966. This classification system is primarily radiographic in nature and is divided into developmental, coalescent, and reconstructive stages. The developmental stage is characterized by profound osseous destruction, with frequent dislocation. The coalescent stage is marked by evidence of repair of large fracture fragments. The reconstructive stage denotes bony ankylosis and often large amounts of hypertrophic proliferation. Although this system is very descriptive, it is not very clinically useful.

Sanders and Mrdjencovich introduced a classification system based on the location of arthropathy. Loosely based on Harris and Brand's classic study, this system is highly descriptive, and, because it denotes the location of the arthropathy, it is clinically useful. The reason that Sanders and Mrdjencovich's system is more clinically useful is that location is pivotal when considering potential complications and fracture healing. For instance, midfoot fractures, which frequently lead to a "rocker-bottom" foot type where the majority of the patient's weight is on the midfoot, are often the most debilitating and result in permanent deformity. Using Sanders and Mrdjencovich's system for location of arthropathy, we further classify Charcot's arthropathy, based on radiographic, dermal thermometric, and clinical signs, as consisting of two treatment-oriented phases: (1) an...
acute phase and (2) a postacute (quiescent) phase. The initial clinical diagnosis of acute Charcot’s arthropathy (as described earlier) is well documented in the literature. Following resolution of acute neuropathic osteoarthropathy, patients are converted to the postacute phase, during which uncasted weight bearing is introduced (Fig. 3).  

Management of Acute Charcot’s Arthropathy

Immobilization and reduction of stress are essential in the treatment of patients with acute Charcot’s arthropathy. Many investigators advocate no weight bearing, through the use of crutches or other assistive devices, during the acute phase of Charcot’s arthropathy. Although this is an accepted form of treatment, a three-point gait may increase pressure to the contralateral limb, thus predisposing it to repetitive stress, ulceration, and neuropathic fracture. Armstrong et al reported that, through the use of appropriately applied total contact casts (Fig. 4), most patients may ambulate during the entire period of treatment.

All patients at our center were initially treated with total contact casting. The total contact cast consists of an inner layer of plaster with thin felt applied to the malleoli and tibial crest and foam applied to the digits for protection. The outer splints and remaining layers are made of fiberglass, with an optional rubber cast plug secured to the plantar aspect of the cast to increase durability. Casts are routinely checked weekly and evaluated for proper fit. Casts of patients with concomitant ulceration (Fig. 5) are changed weekly for ulcer evaluation and debridement. Cast change intervals for patients without ulcers are dependent on cast comfort and integrity (3 weeks maximum). Casting is discontinued based on clinical, radiographic, and dermal thermometric signs of quiescence. Skin temperatures are monitored using a portable infrared thermometric probe. Use of dermal thermometry in the diabetic foot has been well described.

Patients with bilateral acute Charcot’s arthropathy present a unique dilemma. Because of increased inflammation (and subsequent increase in temperature) on both sides, dermal thermometry is less effective in providing clinically useful information. These patients’ lower limbs, therefore, remain in bilateral total contact casts until both feet and ankles normalize clinically and radiographically. Clinical signs of quiescence include reduction of edema and erythema and return of skin lines to the foot. Radiographic signs of quiescence are evidenced by trabecular bridging on serial radiographs. Although the prevalence of bilateral arthropathy has been reported to be as high as two thirds of cases, our recent report of a large series of patients showed a prevalence of only 9%. Perhaps more interesting than the prevalence of bilateral acute Charcot’s arthropathy is whether contralateral Charcot’s joint events occur during treatment. We have observed no contralateral events during treatment of patients with unilateral acute Charcot’s arthropathy. Patients whose lower limbs are placed in these casts are able to ambulate freely during the majority of treatment. We believe that the resultant reduced stride length and decreased cadence expose the contralateral extremity to less repetitive trauma than might occur if a patient walked with crutches. These factors, combined with frequent monitoring and appropriate prescription of footwear (eg, depth-inlay shoes versus custom-molded shoes), may reduce the risk of precipitating a bilateral episode of acute Charcot’s arthropathy.

Until recently, there were no reports concisely detailing treatment of patients with acute Charcot’s arthropathy through the postacute period. The mean time of immobilization (casting followed by removable cast walker) prior to return to permanent footwear was approximately 6 months in our study of 55 patients. Patients receiving arthrodeses and patients who had bilateral Charcot’s arthropathy were casted for longer periods of time (approximately 6 months versus 4 months) and took longer to return to permanent footwear (approximately 11 months versus 6 months). Myerson and
coworkers reported that, following open reduction and internal fixation of acute Charcot's fractures or dislocations in eight patients, the mean time of casting was 5 months. This report, however, did not discuss when patients returned to permanent prescription footwear and fully ambulatory functional status.

Following casting until quiescence, the patients move into the postacute phase of treatment. Patients may, as required, then progress from casting to removable cast walkers to accommodative footwear with ankle-foot orthoses. Removable cast walkers or braces may be used to ease the transition from total contact casting to full, unprotected weight bearing in prescription footwear. Certain models of removable cast walkers (eg, EasyStep Walker) have been shown to be as effective as total contact casts for reducing vertical plantar pressures. Several models of removable cast walkers, however, have poor off-loading characteristics. Therefore, care must be taken in using a removable cast walker that will not off-load effectively. The transition to a removable cast walker should be made when skin temperature gradients are within 1°C for 2 consecutive weeks at the affected site compared with the corresponding site on the contralateral extremity. The transition from a removable cast walker to prescription footwear is based on 1 month of skin temperature equilibrium (± 1°C) at the affected site compared with the corresponding site on the contralateral extremity. This period of protected weight bearing provides the pedorthic shoe specialist time in which to fabricate and appropriately fit prescription footwear.

Reconstructive surgery should be performed if a deformity places the foot at risk for ulceration and if the deformity cannot be safely accommodated in prescription footwear. If the arthropathy is identified in its early stages, surgery is often unnecessary. Only 25% of the patients in the recent study by Armstrong et al ultimately required any form of surgical intervention, with about two thirds of those patients requiring an exostectomy to remove a bony prominence and about one third of the patients needing an arthrodesis. The goal of any surgery for patients with Charcot's arthropathy is to create a stable, plantigrade foot that may be appropriately shod and that can support an ambulatory adult. Surgery is generally undertaken only after radiographic,
dermal thermometric, and clinical signs of Charcot's joint quiescence. After surgery, the patient's lower limb is immobilized until skin temperatures and postoperative edema normalize. Following immobilization, the patient is progressed to a removable cast walker, followed by prescription of permanent footwear.

**Limitations of This Review**

The focus of this update is to provide current information regarding the etiology, diagnosis, and treatment of neuropathic osteoarthropathy. Most of the literature on this topic has focused on surgical treatment for neuropathic osteoarthropathy, often neglecting the nonsurgical aspects of the treatment protocol. We believe (and we have reported1) that the majority of patients with Charcot's arthropathy do not require surgical intervention. For this reason, we have concentrated on outlining this important aspect of care. There have been brief discussions of pharmacologic augmentation to current treatments, using bisphosphonates to retard bone resorption,55,56 but this is in need of further investigation. Additionally, electrical bone stimulation may prove to be of some assistance as an adjunctive modality.57 We refer the reader to the references in this report to thoroughly investigate this complex neuropathic sequela of diabetes mellitus.

**Summary**

With the possible exception of osteomyelitis, Charcot's arthropathy is perhaps the most debilitating orthopedic sequela of diabetes mellitus. For this reason, early diagnosis and aggressive, noncompromising immobilization, pressure reduction, and consistent follow-through are paramount to effecting an acceptable result.

**References**


