CHAPTER **30**

NEUROTROPHIC CHARCOT JOINTS HEAL FASTER THAN NORMAL JOINTS: Fact or Ficton?

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INTRODUCTION

Charcot neuroarthropathy was first described by Jean-Martin Charcot in 1868. He detailed a condition characterized by significant and progressive joint and bone breakdown, thought to be associated with tabes dorsalis. Despite the decline in tertiary syphilis, the incidence of Charcot joint disease did not decrease. Conditions that cause Charcot joint include diabetes mellitus, leprosy, syringomyelia, and poliomyelitis. Diabetes mellitus is the most common cause.

ETIOLOGY

The cause of destructive neuroarthropathy is unclear, but several theories have been proposed. Volkman and Virchow proposed that the cause is from an insensate extremity subjected to trauma, both acute trauma and microtrauma. The trauma leads to fractures secondary to abnormal biomechanical stresses. In fact, Fishco reported that trauma can precipitate the development of Charcot, and this includes surgical trauma. Subsequent investigators noted that osteopenia was often associated with this condition. Theory exists that sympathetic denervation associated with autonomic neuropathy results in hyperemia with subsequent osteopenia and bone weakening. Increased blood flow is also thought to increase osteoclastic activity. Gough et al measured pyridinilone cross linked carboxy terminal peptide domain of Type I collagen, a marker of bone resorption. It was increased in patients with acute Charcot, compared with non-Charcot control subjects. Selby et al. measured urinary deoxypyridilone and alkaline phosphatase. Again, both are markers indicating increased osteoclastic activity. It is likely that the real etiology is a combination of all of these theories.

CLINICAL PRESENTATION

Charcot neuropathic arthropathy is clinically characterized as a painless, progressive degeneration of peripheral joints. In the acute phase, the extremity is erythematous, warm, and moderately to severely edematous. The patient may or may not remember a history of trauma.

EICHENHOLTZ CLASSIFICATION

The most widely accepted staging classification is the Eichenholtz Classification. Although this is a radiographic staging, clinical parameters have been devised. (Table 1). The big disadvantage to the Eichenholtz classification is that early staging is absent. Schon, et al and Yu, et al describe a Stage 0, where radiographic changes are evident but subtle. Mild fracture and/or joint space widening may be present with the clinical identifiers of erythema, edema and calor. Clinically, it is difficult to differentiate from a Stage I. With such subtle radiographic changes, a diagnosis of Stage 0 may only be determined by ruling out other differentials (i.e., cellulitis, osteomyelitis, DVT).

Eichenholtz Classification

Stage	*I - Acute/Developmental*	*II - Coalescence*	*III - Remodeling*
Radiographic	Debris formation, bone fragmentation, subluxation	Debris absorption, coalescence of fragments, sclerosis	Bony ankylosis, reformation of architecture
Clinical	Significant erythema, edema, calor	Regression of erythema, edema, calor	Resolved

Table 1. The Eichenholtz Classification.

DIAGNOSIS

The diagnosis of Charcot is made with the above described clinical and radiographic findings, and can be confirmed with other studies. Technetium 99 bone scans can be employed, which will reveal increased uptake in all 3 phases. A synovial tap will reveal multiple shards of bone and cartilage embedded in layers of the synovium. This study will also help to exclude osteomyelitis from the differential. Dermal thermometry can also be used simply to confirm any temperature change in the extremity. Murff et al studied reliability of manual detection of temperature changes. They found that physicians could correctly detect a temperature gradient using their hands 1 out of 10 times, thus suggesting that it may be difficult to detect subtle calor associated with Charcot. Dermal thermometry may be crucial in stage 0.

CHARCOT PERIOPERATIVE CONSIDERATIONS

Several factors must be considered when deciding to operate on a Charcot foot, including age, lifestyle, health, and physician and patient expectations. One must consider not only the physiologic status of the patient and the involved extremity, but peripherals about the patient. For example, does the patient have personal assistance and home setting requirements that provide for a maximum healing environment postoperatively?

The following concerns must be considered pre-operatively for any patient undergoing Charcot reconstruction.

Many diabetics have co-morbidities that need to be addressed. Many are renally and visually impaired, obesity is a concern and their nutritional status must be assessed. The patient should be under the best metabolic and nutritional control.

It must be determined whether the patient can tolerate general, spinal, or intravenous sedation anesthesia. Will lower extremity tourniquets affect cardiopulmonary status? A decision must also be made by the surgeon as to whether the entire procedure can and should be performed wet.

Assessment of vascular status must be completed. Of particular importance are the ABI, Doppler waveform, and transcutaneous oxygen pressure.

One must consider an existing ulceration and the ability to excise the lesion. Baravarian et al suggested a 25% infection rate involving surgical intervention with an existing ulceration.

Osteomyelitis must be ruled out. If osteomyelitis is present, surgical debridement and complete resolution prior to any reconstruction is recommended. Large defects may remain post surgical debridement. These defects will need to be addressed before final reconstruction. Intravenous antibiotics, possible antibiotic beads, and bone grafting may need to be used.

The region of collapse should be noted. Specifically, one should consider the level of laxity, joint stability, and the number of joints involved because this may help to determine the amount of surgery required.

The quality of bone should be assessed. As discussed earlier, many patients may present with a significant amount of osteopenia, and this can have a dramatic effect on surgical outcome, particularly if the surgery involves osteotomies and/or arthrodesis. The surgeon must be prepared to use internal fixation, external fixation, or combination methods to best address each component of the deformity.

Most patients with Charcot joint breakdown demonstrate a significant pes valgo planus deformity as a result of LisFranc, midtarsal and/or subtalar joint collapse. An acquired triceps shortage usually ensues further wedging of the midfoot and rearfoot worsening the deformity. Tendoachilles lengthening may be considered when total repair of this deformity is performed.

Another issue that may be considered, is the appropriate Eichenholtz stage for reconstructive intervention. Simon et al published a comprehensive arthrodesis study in 2000. The study involved 14 patients who underwent reconstruction involving a fusion procedure at some level. All patients were a Stage I Charcot, and all had complete healing with no evidence of delay. Wang et al followed the study in 2002 involving 28 patients, all Stage I Charcot. All underwent an arthrodesis with the application of an external fixator and bone stimulator. All patients went on to complete healing with no further breakdown. The significance of both of these studies is that acute Charcot may no longer be a contraindication to surgical intervention.

SURGICAL CONSIDERATIONS

The question often posed in neuropathic Charcot joint reconstruction is, does a Charcot joint pose any challenges (internal physiologic, neural, or circulatory, etc.) that makes surgery in these patients more dangerous? Is there some reason that makes these bones more difficult to heal? And does this phenomenon increase morbidity in these patients? There is very little information published on this subject, and these questions have never really been answered directly. But there are some factors that must be considered, which may partially explain, and in fact, support some concerns of healing in Charcot foot surgery. We will demonstrate some clinical examples for the reader to consider regarding this question later. Despite the idea that staging may be of little consequence when performing a reconstructive procedure, many of these cases go on to delayed union, nonunion or malunion. The remainder of this discussion will explore variables that may interfere will postoperative healing.

As mentioned previously, strict glucose control is of extreme importance perioperatively. Loder and associates reported that an overall union rate for fracture was 163% longer in diabetic patients compared with healthy controls. Edelson recommended that blood glucose be maintained at 200 mg/dL or lower perioperatively. Uncontrolled diabetes mellitus causes polymorphonuclear leukocyte (PMN) imbalance, including but not limited to decreased chemotaxis, phagocytosis, and diapedesis. All of these abnormalities combined, directly results in impaired wound healing, impaired collagen formation and decreased ability to fight infection. Edelson reported that diabetics have a 40% increased risk of developing a wound infection following surgical intervention.

In addition to PMN function, diabetic patients have an increased incidence of microvascular disease. Chronic hyperglycemia has a toxic effect on vascular and endothelial cells, as it promotes glycosylation of blood vessel walls resulting in capillary basement membrane thickening. Edelson speculated that microvascular disease of the skin and subcutaneous tissues may impair wound healing through decreased delivery of glucose, oxygen, and other nutrients to the site of injury. While the macrovascular system often remains normal or increased (hyperemia secondary to autonomic denervation), Pham and associates reported decreased vasodilation within the microvasculature system of patients with diabetic neuropathy and Charcot neuroarthropathy.

Diabetes mellitus is also associated with insulin resistance. Chronic insulin resistance leads to altered lipid and protein metabolism. Insulinemia has an anabolic effect on protein, which results in impaired neovascularization, fibroblastic activity and poor PMN bactericidal capacity. Insulinemia also prevents the production of free fatty acids, which are necessary for cell membrane synthesis needed for wound healing.

Another risk factor for poor surgical healing is suboptimal patient compliance. This may include poor weight-bearing compliance, poor blood glucose control, noncompliance with physical therapy and external fixation adjustments, poor hygiene with pin sites, and impaired antibiotic and/or medication regimen. Edelson reported that patient noncompliance is a direct contraindication for surgery. The patient truly is the rate limiting factor, and if a patient is noncompliant with diabetes management or ulcer management, then that behavior will most likely continue through postoperative recovery.

It is important that the length of time required for immobilization is not underestimated. Baravarian and associates suggested that diabetics be immobilized twice as long as the nondiabetic patient. Surgical planning must be complete. Hamilton and associates remind the surgeon that aggressive bone resection of a plantar and/or midfoot prominence can create an unstable structure, which can further progress to a rocker bottom deformity and ulcer formation. Compression and stability are also important, particularly with arthrodesis procedures. Baker et al advocate external fixation to allow for early weightbearing and retardation of disuse osteoporosis, muscle atrophy and joint stiffening. They suggest internal beaming of the medial column with short, large-diameter screws to provide support in conjunction with a frame. Recent advances in locking plate mechanics and design have improved bone-plate interface such that an internal construct is created with rigidity similar to an external fixator.

Although we use cutting-edge technology, instrumentation and surgical techniques, often less surgery is the better surgical choice, particularly for high risk Charcot patients. The goal of surgery is to create a stable and functional foot, which may be achieved with a "less is more" approach.

CASE PRESENTATIONS

The first case is a 73-year-old neuropathic, type II diabetic female who presented to the office with a Jones compression dressing after being treated at Eastside Medical Center for a right ankle fracture experienced while she was descending stairs. Clinical evaluation revealed moderate edema of the right ankle, lateral displacement of the foot on the ankle, and very mild discomfort inferior to the medial malleolus. Neurological examination revealed absent protective response to Semmes-Weinstein monofilament testing. Radiographic examination revealed a PER IV ankle fracture with shortening of the fibula and increased medial clear space (Figure 1). The patient was taken for ORIF and repair consisting of fibular interfragmental screw and neutralization plate fixation and a transfixion syndesmotic screw to protect the interosseous membrane (Figure 2). The patient was kept non-weight-bearing for 6 weeks. This was followed by use of a fracture boot and physical therapy. Thirteen-week postoperative radiographs revealed loss of correction as a result of transyndesmotic screw loosening. The fibular fracture had not yet consolidated and the medial clear space had increased (Figure 3). It was felt that because the patient was a neuropathic diabetic, the absence of the protective proprioceptive and kinesthetic responses were absent and partly responsible for reduction failure. The patient was returned to surgery for exchange of the distal syndesmotic screw and addition of another proximal transfixion screw to provide a more rigid construct (Figure 4). As recommended by Baravarian et al the patient was kept nonweightbearing for an additional 12 weeks. One year postoperatively, the ankle was clinically stable with radiographs revealing complete maintenance of reduction and osseous healing (Figure 5).

The second case is a 78-year-old IDDM neuropathic female who underwent midfoot and rearfoot Charcot reconstruction of the right foot (Figure 6). Postoperatively, against instructions, the patient walked out of bed, applying weight to the operated foot. The patient did not ask for assistance and while trying to sit back on the bed, slipped on the floor, falling and traumatizing the left contralateral ankle. Ankle radiographs revealed

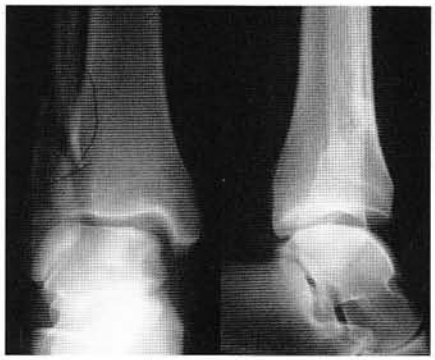

Figure 1. Preoperative radiographic view reveals PER IV ankle fracture.

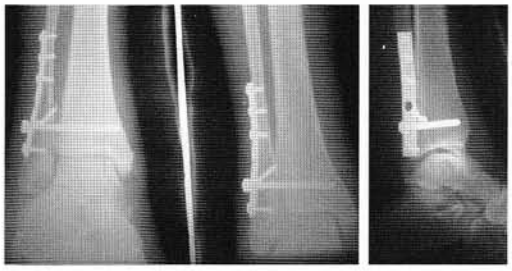

Figure 2. Postoperative ankle radiographs demonstrating reduction of clear space, neutralization plate fixation of lateral malleolus and transfixion screw protecting the interosseous membrane.

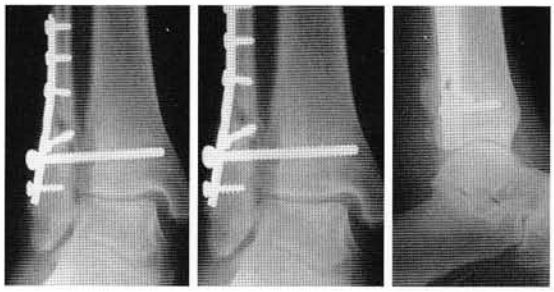

Figure 3. Radiographs 3.5 months postoperatively. Note widening of medial clear space as a result of transfixion screw loosening.

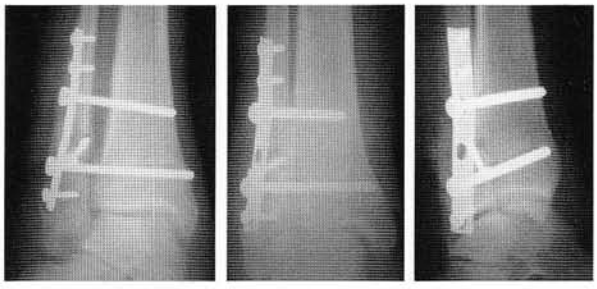

Figure 4. Improvement of medial clear space and restabilization of ankle after exchange of hardware and addition of second proximal transfixion screw.

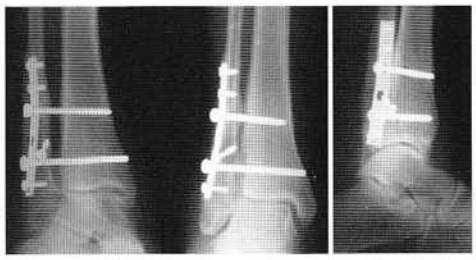

Figure 5. One year following hardware exchange and restabilization, complete healing of all osseous segments has occurred. The ankle has remained completely stable.

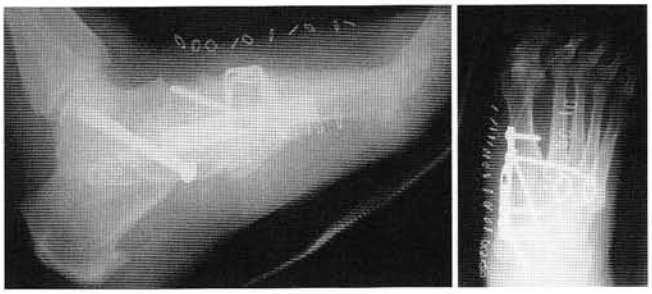

Figure 6. Postoperative radiographs of right foot following reconstruction of Charcot foot.

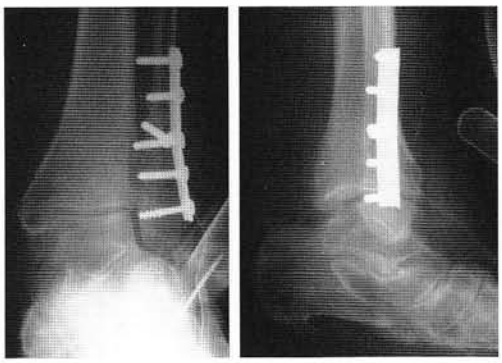

Figure 7. Postoperative reduction of fibular fracture.

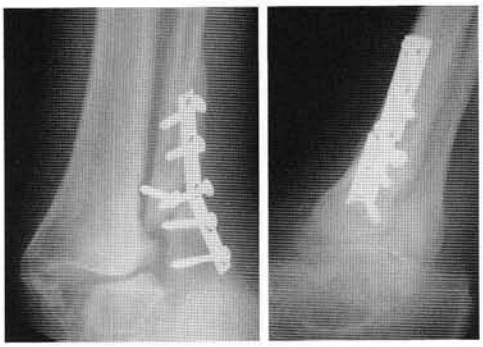

Figure 8. Ankle instability as a result of hardware loosening, non-union of fibular fracture and broken 1/3 semitubular plate.

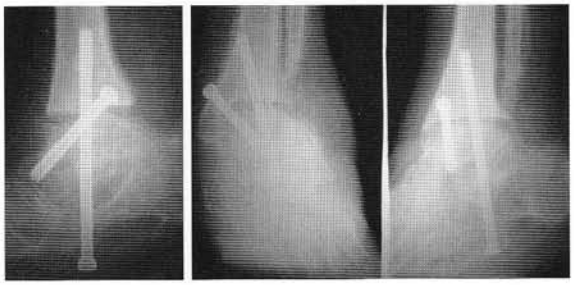

Figure 9. Four-month postoperative ankle arthrodesis radiographic views identifying loose cannulated screws.

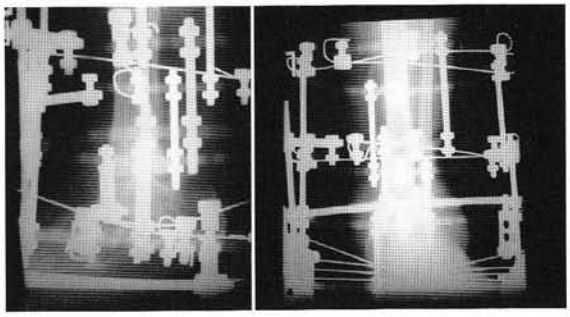

Figure 10. Refusion of ankle and stabilization with Ilizarov frame.

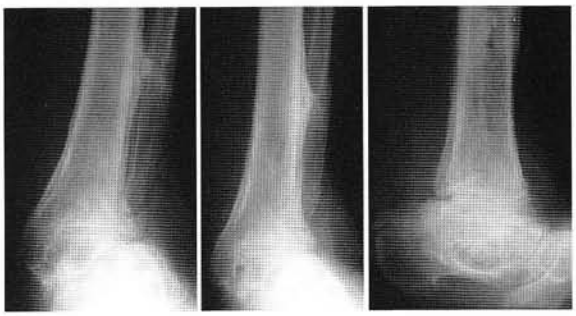

Figure 11. Complete fusion of ankle noted 2 years following Ilizarov frame external fixation of left ankle.

an SER IV fracture. The patient was taken to surgery the following day where repair was accomplished using an interfragmental screw and a one-third tubular neutralization plate (Figure 7). Unfortunately, the patient was very non-compliant with maintaining a nonweight-bearing status of the left ankle. This resulted in hardware failure, nonunion of the fibula and plate breakage (Figure 8). One year later, the patient was taken to surgery for hardware removal and ankle fusion using 2 cannulated screws. Again, the patient ambulated too soon on the operated ankle, causing loosening of the cannulated screws later resulting in nonunion of the ankle (Figure 9). The patient was taken to surgery for hardware removal and refusion of the ankle using an Ilizarov external fixation frame (Figure 10). The ankle was healed and stable after 6-and-a-half months and the external fixator was removed. The last office visit 2 years later revealed excellent consolidation and stability of the ankle (Figure 11).

SUMMARY

In summary, neuropathic patients with autonomic neuropathy, lack sympathetic control of their blood vessels and therefore, vasoconstriction cannot be accomplished. As a result of chronic arterial vasodilation, arthrodesed joints will consolidate earlier than in the sensate patient. Yet, because of lack of proprioception and lack of muscle and kinesthetic control, these patients are at a higher risk for loss of intraoperatively acquired alignment. Fractures and osseous procedures, such as arthrodeses, must be augmented with added forms of fixation and nonweightbearing continued 10 to 12 weeks longer than the sensate patient. Twenty years ago, surgical reconstruction of the diabetic neuropathic Charcot foot was rare. However, using advances in medical and surgical knowledge, and cutting edge instrumentation and techniques, Charcot foot reconstruction is now a daily event. Multiple morbidities can occur with treatment of the neuropathic Charcot patient. When a multidisciplinary approach is used, the podiatric surgeon can continue to achieve very acceptable outcomes in these patients.

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