

Charcot Ankle Neuroarthropathy Pathology, Diagnosis and Management: A Review of Literature

Keywords: Ankle charcot Arthropathy; Ankle arthrodesis; Ankle external fixation arthrodesis; Total contact cast; Ankle neuroarthropathy

Key Points: Charcot ankle neuroarthropathy is a common hard to treat condition, early diagnosis and proper management is the key point of treatment. Non- surgical options are feasible in certain stages, but surgical intervention is required in advanced disease.

Introduction

Charcot neuroarthropathy can be described as a non-infective, destructive process activated by an isolated or accumulative neuro-traumatic stimulus that manifests as dislocation, peri-articular fracture or both in patients rendered insensate by peripheral neuropathy [1].

The ankle has swelling, warmth, and erythema, and the syndrome may initially be difficult to distinguish from infection. The bones and joints develop fractures, ligamentous laxity, dislocations, cartilage damage, bone erosions, and hypertrophic repair [2]. The resulting bone and joint deformities may be associated with instability and may compromise the fitting of shoes or braces. Furthermore, ulceration may result from instability or bony prominence and may cause chronic or recurrent soft tissue infection and osteomyelitis. Amputation may be required for management of infection or instability [2].

Almost all affected individuals have a dense sensory peripheral neuropathy. The neuropathy is most commonly associated with diabetes, but may also be associated with leprosy, alcoholism, tabes dorsalis (Syphilis), syringomyelia, peripheral nerve injuries, or congenital absence of pain sensation [2]. Usually the larger joints of the lower extremity are involved in syphilis, and the larger joints of the upper extremity are involved in syringomyelia [2]. In contrast, diabetes related Charcot arthropathy primarily affects the foot and ankle [3].

Risk Factors

The presence of sensory peripheral neuropathy is essential for Charcot arthropathy to develop [4] (Table 1).

The amount of bone and joint damage in the Charcot ankle is determined by severity of sensory loss, mechanical stress on joints, and physical activity of the patient [2].

Vibration sensation and cardiovascular autonomic function are similar in patients with Charcot neuroarthropathy and diabetic patients with ulcers [5]. Obesity is present in at least two-thirds of patients with Charcot neuroarthropathy [6].

Trauma or surgery may initiate Charcot arthropathy [7-11] The trauma may be apparently minor, such as ligament sprain, twisting injury, or stress fracture [7,12,13]. A history of trauma

Review Article

Volume 6 Issue 2 - 2016

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Received: October 01, 2016 | **Published:** November 03, 2016

is present in 22% to 53% of patients with Charcot Arthropathy [6,14,15].

Classification

Modified eichenholtz stages

Shibata T et al. [16] modified the Eichenholtz system to include an earlier stage prior to "development." Several authors have proposed that this early inflammatory phase following injury be called "Charcot in situ," "pre-stage 1," or "stage 0 Charcot" [17-20]. This classification is currently being applied by the majority of foot-and-ankle physicians to Charcot arthropathy patients in the staging of the disease (Table 2).

Brodsky (anatomical) classification

The most widely used anatomic classification by orthopaedic foot and ankle surgeons is that based on the four most common anatomic regions affected [21] (Table3).

The ankle comprises 9% of Charcot joints of the foot and ankle (Type 3A) (Figure 1). [3] Although less common than midfoot or hindfoot Charcot joints, Charcot ankles may be complicated by ulceration at the malleoli and severe uncontrollable deformity and hypermobility that may preclude fitting of a brace.

The diagnosis of Charcot arthropathy is primarily reliant on clinical presentation, but a physician's high index of suspicion should not be underappreciated [7]. A thorough patient history is essential to any assessment; however, a neuropathic patient's history can be unintentionally misleading. It is therefore up to the clinician to know what questions to ask and what information is important when making an assessment.

Table 1: Risk factors for the development of Charcot Arthropathy [5].

Neuropathy (sensory, autonomic, motor)	Age
Obesity	Physical activity
Trauma	Peripheral vascular disease
Ulceration	Plantar pressure
Instability	Achilles tendon / gastro soleus contracture

Table 2: Modified Eichenholtz Stages [19,20].

Stage	Clinical	Radiography	Differential Diagnosis
Stage 0: Charcot (Inflammatory) Arthropathy	Localized swelling Redness Warmth	Diagnosis of this stage with MRI or technetium bone scan	Frequently, stage 0 patients are misdiagnosed as cellulitis, gout, or deep vein thrombosis
Stage I: Development-Fragmentation	Erythema Marked swelling Increase warmth	Bone debris at joints Fragmentation of subchondral bone Subluxation Dislocation	
Stage II: Coalescence	Decrease erythema Decrease swelling Decrease warmth	Absorption of fine debris New bone formation Coalescence of large fragments Sclerosis of bone ends	
Stage III: Reconstruction-Consolidation	Resolution of edema Residual deformity	Remodling, rounding of bone Decrease sclerosis	

Table 3: Charcot arthropathy of the foot and ankle: Brodsky anatomical classification [21].

Type 1	Tarsometatarsal (Lisfranc's)
Type 2	Chopart's/ subtalar
Type 3A	Ankle
Type 3B	Calcaneus
Type 4	Multiple regions : Sequential Concurrent
Type 5	Forefoot

Special attention must be given to the patient especially if history of any trauma, history of neuropathy, recent swelling, redness in the limb. Counter intuitively, the history may include pain sensations in an insensate limb but no recollection of any sustained trauma. In a study of 55 patients with Charcot arthropathy, more than 75% complained of pain in the foot and ankle upon presentation even though all subjects had a clinical loss of protective sensation to the 10-g Semmes-Weinstein monofilament wire [22].

Repetitive trauma to the foot and ankle may be entirely absent from verbal history even though clinical symptoms prove otherwise. The study found that only 22% of patients were able to

recall some specific traumatic event prior to the onset of Charcot arthropathy. The loss of protective sensation may leave the patient unaware of any particular event or reoccurring minor events [23].

It is important to investigate any previous history of infection or ulcers to rule out a recurring acute or chronic infection. The usual presentation of Charcot arthropathy involves, a warm, swollen, erythematous foot or ankle in an insensate patient and, because of its similarity to an acute soft-tissue infection, heightened awareness is needed when dealing with the diabetic neuropathy patient population [22]. Most infection in the diabetic foot and ankle involve a direct source of inoculation through an opening in the skin, usually caused by neuropathic ulcers [24,25].

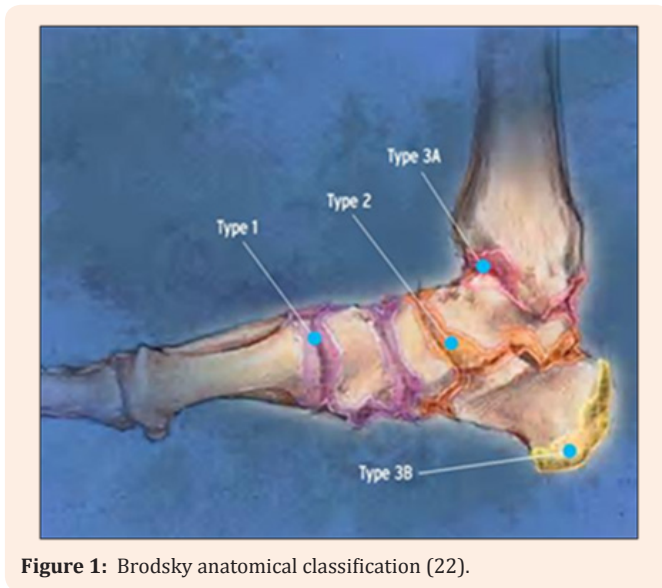


Figure 1: Brodsky anatomical classification (22).

When this initial stage is suspected but not proven, the patient should be prevented from incurring any additional injury to the suspected limb. With immobilization and offloading, the patient is protected while awaiting the results of further investigation.

Radiographs of the foot and ankle that are taken in the non-weight-bearing position can have obvious variability in image quality and may not show subtle instability compared to radiographs in the weight-bearing position. It is recommended that all foot-and-ankle radiographic examinations be obtained in a weight-bearing position if possible.

MRI examination are increasingly being used and recommended for diagnosing Charcot arthropathy, especially at the earliest stage [26-28].

In diagnosing Charcot arthropathy, one review described findings of abnormal values of bone-specific alkaline phosphatase, type 1 collagen carboxyterminal telopeptide, and urinary deoxypyridinoline cross-links, indicating increased osteoclastic/osteoblastic activities throughout Eichenholtz stages 1 and 2 [29].

Baumhauer et al. [30] found that Charcot arthropathy-reactive bone had osteoclasts disproportionately increased compared to osteoblasts, and the osteoclasts demonstrated immune reactivity for IL-1, IL-6 and TNF α . Although these laboratory findings may assist the clinicians' assessment, it is impractical to obtain histological samples from patients presenting with the initial symptoms. It is recommended that biopsies be collected only when there is a non healing wound or suspected bone infection [26,31].

Modalities of treatment

- A. Non-Operative Treatment including
 - I. Total Contact Cast (TCC) [26].
 - II. Prefabricated Pneumatic Walking Brace (PPWB)

- III. Charcot Restraint Orthotic Walker (CROW)
- IV. Antiresorptive Drugs
- V. Electrical Bone Stimulators
- VI. Ultrasound
- B. Operative Treatment including
 - I. Debridement of Ulcer.
 - II. Osteotomy.
 - III. Arthrodesis with Internal Fixation.
 - IV. Arthrodesis with External Fixation.
 - V. Amputation: with uncontrolled infection and
 - VI. Uncontrolled deformity.

Non operative therapies and medical management

The goals for every patients undergoing treatment for an acute or quiescent Charcot process should be to maintain or achieve structural stability of the foot and ankle, to prevent ulceration, and to preserve a plantigrade foot [15].

Total contact cast

Most cases of acute Charcot ankle especially stage 0 or stage 1 Eichenholtz Charcot neuroarthropathy can be treated non-surgically with pressure-relieving methods such as total contact casting (TCC), which is believed to be the gold standard of treatment [32].

TCC was developed in the 1950s. Most of the cast padding is eliminated for exact conformity to the lower extremity, with the goal of evenly distributing forces across the plantar surface of the foot. A tubular stockinette with low-density foam or one quarter inch felt is applied over the tibial crest and malleoli, and around the metatarsal heads with one layer of synthetic padding. A three layer inner plaster shell is followed by a fiberglass outer shell.

The total time of non-weight-bearing TCC and the immobilization period in weight-bearing TCC may last up to 4-6 months [33]. Once there is bony consolidation, custom inserts or extra-deep shoes can be worn, followed by proper physician visits to ensure no uneven distribution of plantar pressures. Although the TCC is an effective treatment for the ACA process, there have been complications associated with this treatment, usually related to weight-bearing allowance [34].

Prefabricated pneumatic walking brace

An alternative to TCC is a prefabricated pneumatic walking brace (PPWB). Use of the PPWB is limited in patients who have severe foot deformity or who are noncompliant.

Charcot restraint orthotic walker

After swelling and erythema resolve and radiographic stability has been achieved, the TCC is changed to either a CROW or an ankle foot orthosis or a patellar tendon-bearing brace, depending on residual anterior edema.

Antiresorptive drugs

Bisphosphonates are popular as antiresorptive drugs against osteoporosis, Paget's disease, and other diseases with increased bone turnover [35], and there have been reports on the possibility of pharmacologic therapy for Acute Charcot Arthropathy [36].

Electrical bone stimulators

Other adjunct therapies have also been offered to help manage ACA. Electric bone growth (EBG) stimulators [37] have been experimentally applied and clinically tested to promote healing of fractures in the acute phase.

Ultrasound

A series of case reports has described the use of adjunct low-intensity ultrasound for Charcot arthropathy treatment. There are no subsequent studies to validate this method [38].

Surgical treatment (goals and Indications)

Goals of surgery in the charcot ankle include

- I. Restoration of stability and alignment.
- II. Prevention of deformity, to facilitate functional ambulation.
- III. Ulcer prevention with prescription footwear and braces [39].

Surgery for Charcot deformity does not eliminate the necessity for use one of non-operative methods [39]. An unstable ankle may be more difficult to control with an orthosis and may be more susceptible to ulceration and secondary deep infection. Therefore, surgery in the Charcot ankle may prevent ulceration and amputation. Limb salvage may be important, in part, because the contralateral foot may be at risk for Charcot neuroarthropathy; if an amputation is done and the other side becomes involved, the patient might end up with bilateral amputation [39,40].

Indications for surgery in the Charcot ankle include

- i. Acute dislocation [12]
- ii. Recurrent ulceration,
- iii. Secondary to either instability or bony prominence, that cannot be managed successfully non-surgically [39].

Severe or uncontrolled deformity or instability that may cause ulceration or that may make it impossible to fit a custom brace or footwear [41].

Timing of surgery

The demineralization, soft bone, and swelling in stage I may increase technical difficulty and surgical complications such as infection and loss of fixation [20,39]. Surgery may be considered after decrease in warmth, erythema, and swelling [2,41].

Arthrodesis with internal fixation

Arthrodesis with plate and screws: Arthrodesis using internal fixation has been a viable option for many surgeons. Banks & McGlamry [42] treated the Charcot foot and ankle by stabilizing the foot and ankle with internal fixation arthrodesis. Stabilization

of the joints prevented the further progression of ulcerations and joint breakdown. These patients were immobilized with a cast postoperatively. Casting and immobilization can arrest the degenerative progression of Charcot disease but there is the potential for demineralization with cast disease. Because of the progressive nature of this pathology with Charcot hyperemia and bone demineralization caused by cast disease and non weight bearing, the internal fixation could fail. The importance of avoiding immobilization is that partial to full weight bearing helps to maintain bone stock if the foot and ankle are adequately stabilized [43].

Internal fixation used in arthrodesis for Charcot ankle neuroarthropathy may include 6.5-, 7.0-, or 7.3-mm cancellous bone screws, Locked plates, and staples. Screw fixation with smaller screws may be useful in midfoot arthrodesis, but multiple screws may be required because of the risk of hardware failure [44].

Arthrodesis with nail

Intramedullary fixation for arthrodesis of the ankle was first described by Adams in 1948 [45], but nowadays there are several specific ankle fusion nail:

- i. The TRIGEN™ Hindfoot Fusion Nail (HFN): Offers unique locking configurations allowing the surgeon to target the best bone possible within the hindfoot to maximize purchase and position.
- ii. The ZIMMER Hindfoot Fusion Nail.

In several published series, an intramedullary retrograde nail was used for advanced degenerative or post-traumatic arthrosis. Few studies have provided any indications for the use of this technique in Charcot neuroarthropathy [12].

The goal of treatment with intramedullary fixation is to obtain alignment of the ankle-foot system, reducing significantly the risk of ulceration. The percentages of success in the treatment of this complication are variable, with documentary evidence varying between 50% and 90 % [46,12]. The causes of failure are uncontrolled infection, hardware failure, and malunion. Recently, another study reported 90% limb salvage using intramedullary nail technique [47].

Arthrodesis with external fixation

External fixation is a viable alternative that allows micromotion to occur through fracture and joint dislocation areas. This can facilitate arthrodesis by compressing fragments or joint areas with osteoarthropathy through this protected micromotion [47]. External fixation allows rigid immobilization postoperatively. It has the ability to adjust compression and distraction at the breakdown level and allows for ambulation. Early weight-bearing provides biomechanical stress and compression to the bone to prevent osteopenic changes and cast disease.

This stresses the bone and will increase the chance for arthrodesis by compression and fracture healing. [48] Because of the hyperemic phase of the Charcot foot, this response will promote osseous bridging in fractures and dislocations.

External fixation should be strongly considered as the best choice of fixation when addressing the multiple degenerative areas of Charcot disease. Of the multiple uses of external fixation, Charcot reconstruction is ideal for helping the podiatric surgeon stop the progressive disease of joint breakdown and degeneration [49].

The goals of Charcot reconstruction with external fixator are three fold [50]:

- i. First, correct the patient's equinus and reestablish the calcaneal inclination angle;
- ii. Second, maintain the rear foot to leg relationship; and,
- iii. Finally, correct and stabilize the degenerative joints.

This can usually be performed percutaneously and without hemostasis. It is very important to allow patients to weight-bear within 1 week postoperatively. This is possible with a unique combination of innovative external fixation techniques. Though this strategy is foreign and contrary to traditional thoughts and approaches to surgical intervention, external fixation allows for safe and percutaneous Charcot reconstruction. The use of uniplane and multiplane fixators in a percutaneous manner is an important facet of addressing Charcot pathology [50].

Complications of external fixation

The most common complications with external fixation in the Charcot ankle including pin tract infections that has been found to range from 0.92% to 60% [31]. Erythema and drainage around pin sites is usually due to micromotion or unstable pins and can be dissipated by tightening or retensioning pins, or by meticulous pin care. At times, short doses of oral antibiotics can also help alleviate these symptoms. The progressive pin tract infection, with resultant loosening of the frame, occurred in a small number of patients. This complication is treated with a frame revision in the operating room with removal of the infected, unstable pins and replacing them with additional fixation. Half pin fixation usually is replaced with 2 to 3 additional wires to improve fixation and reduce the risk of fractures through the larger pin sites.

Contraindications to Arthrodesis Include

1. Stage 1 Charcot disease,
2. Poor nutrition or diabetic control,
3. Severe peripheral vascular disease,
4. Active soft tissue or bone infection,
5. Noncompliant patient, and
6. Poor bone stock for fixation.

Conclusion

The Charcot arthropathy process can take up to 2 years to run its course. Primary care physicians must consider the diagnosis of Charcot arthropathy in any neuropathic patient presented with erythema, edema and warmth regardless of local or systemic signs of infection.

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