Treatment of Severe Ankle Diabetic Neuroarthropathy (Charcot Ankle) with Retrograde Intramedullary Nailing: Report of Two Cases

Ian Power MD† and Richard Miller MD†

†UNM Department of Orthopaedics and Rehabilitation

Introduction

Neuroarthropathy, commonly referred to as Charcot joint, results in fracture and progressive osseous destruction. The foot and ankle are the most frequently involved.[1] Patients in the acute process, progress through a variety of stages until consolidation or the reparative stage is reached.[2,3] Jean-Marie Charcot first described this condition in patients with tertiary syphilis in 1868. It was later attributed to patients with diabetes mellitus in 1936.[1] Today, in the United States, diabetes is the most common etiology of Charcot joint. This destruction of the normal foot and ankle anatomy leads to altered weight-bearing mechanics with decreased function and skin ulceration. Treatment of Charcot joint is usually non-operative with immobilization and limited weight-bearing until the healing phase is reached.[4] This may take months, or even up to a year.[5] It marks the beginning of lifetime treatment and surveillance to prevent future complications. These include ulceration, which often leads to infection including osteomyelitis, and eventually amputation.[4]

Though not fully understood, there have previously been two proposed mechanisms behind neuroarthropathy. Virchow and Volkman, postulated that changes leading to an insensate foot allowed repetitive trauma which in turn led to inflammatory and destructive phases. This accounts for the minimal or absent history of trauma in some diabetic patients who present with a Charcot joint.[6] The alternate theory proposed by the Frenchman Charcot stated that autonomic disregulation led to altered vasoregulation and vascular shunting followed by osteopenia and set the stage for bony destruction with microtrauma.[6] More recent research has pointed to inflammatory cytokines and molecular stimulation of osteoclast formation as being involved.[7]

The following cases demonstrate severe destruction of the ankle joint resulting in unbraceable deformity, instability, and inability to ambulate. Realignment and fusion with intramedullary nailing was performed in both cases.

Case Report

Two patients with a Charcot ankle were evaluated for consideration of alternative treatment to amputation.

The first patient is a 41-year-old female with diabetes and end stage renal disease being considered for renal transplant. She presented two years after sustaining a trauma to her left foot and ankle. At the time of presentation she has a severe deformity consisting of varus angulation of the ankle and apex plantar deformity of the sole. (Figure 1) She was barely able to ambulate in a cast boot with the deformity. Given the lack of passive correction this was not a braceable deformity. Though the skin was intact, the prominence of the distal fibula with overlying callus was felt to be at high risk of impending ulceration.

She underwent left ankle fusion with a hindfoot intramedullary nail, and was kept non-weight-bearing for two months. (Figure 2) This was followed by progression to partial, then full weight-bearing in a cast boot. At six months she was doing well with a healed fusion and improved ambulation. A slight plantar prominence remained, which was treated with custom plastizote insoles. At 8 months she was pain free, walking in shoes, and had returned to work.

The second patient is a 36 year-old male with no initial diagnosis of diabetes. He had a two year history of right ankle instability symptoms with minimal pain. He had no history of significant trauma, but did feel a "pop" while rolling his ankle several years before. He was initially able to ambulate in an air cast boot, though this became difficult given his worsening varus instability and progressive deformity. (Figure 3) He eventually had prominence of the distal fibula with abrasions but no frank skin breakdown. Due to concern for neuroarthropathy in this scenario, labs were performed which revealed a glucose of 354 and HgA1c of 12.1. The patient was referred for treatment of his newly diagnosed diabetes.

After controlling his diabetes, he underwent right ankle arthrodesis with a hindfoot nail. His post-operative course was similar to our first patient, with good pain relief and a functional foot at his 6 month follow-up. (Figure 4)

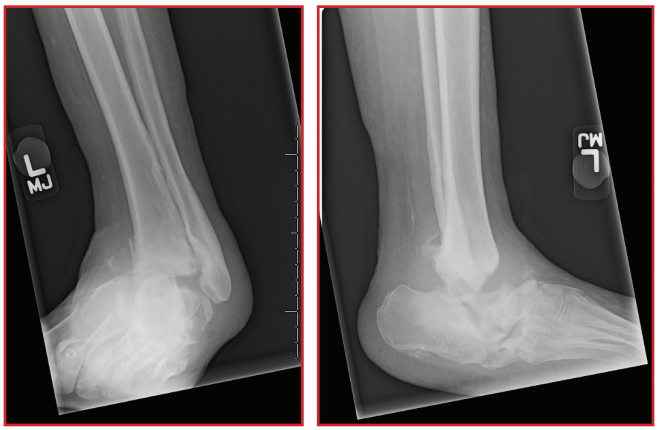

Figure 1: Preoperative AP and lateral x-rays demonstrating varus instability with weight-bearing, prominence of the fibula, and bone loss from talus.

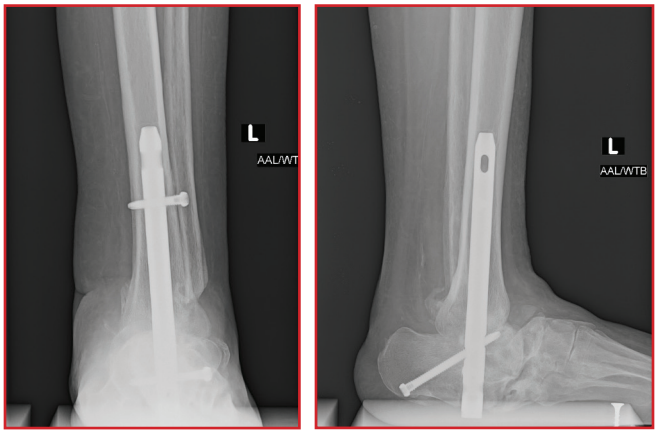

Figure 2: Postoperative AP and lateral x-rays with ankle realigned and nail in position resulting in stable plantigrade foot at 6 months.

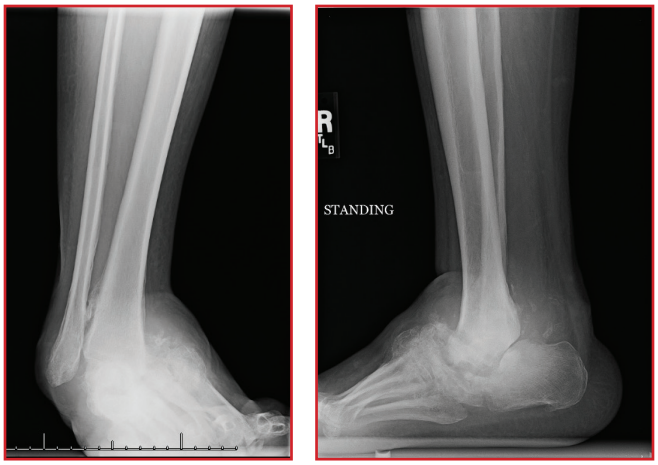

Figure 3: Weight-bearing AP and lateral x-rays of the ankle demonstrating prominent fibula from varus deformity and unstable ankle.

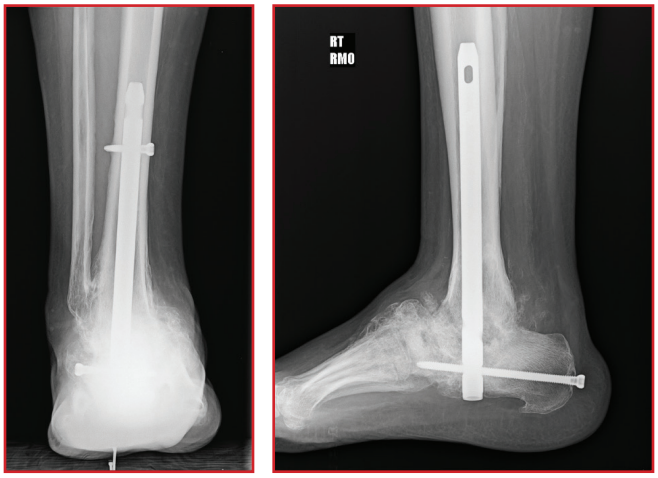

Figure 4: Weight-bearing AP and lateral x-rays of the ankle 6 month status post surgery demonstrating a stable well aligned foot

Discussion

The goal of treating Charcot neuroarthropathy is to obtain a plantigrade foot which is stable and able to be put in a shoe. This allows ambulation and improved functional use of the limb. Additionally it corrects excessive deformity which can lead to ulceration. Initially this is done with orthoses. While many times these goals are achieved, this was not attainable with these two patients. The bone destruction progressively lead to instability, deformity and bony prominence which prevented bracing and ambulation. By virtue of the concomitant diabetes, these two individuals were also predisposed to ulceration, infection, and the eventual threat of amputation. Surgical correction consisting of realigning and fusing the ankle is at times an alternative to amputation when Charcot destruction is severe. Such was the case for our two patients.

Candidates for this procedure should have sufficient vascular supply and potential for healing and ambulation. Though Charcot joint does not directly involve the blood supply to the foot and ankle, there is a high prevalence of peripheral artery disease in patients with diabetes. An ischemic Charcot joint would necessitate revascularization prior to surgical treatment.[8] Candidates should also be free of infection prior to surgical consideration and correction. Many of the signs and symptoms of Charcot joint are similar to infection, including swelling, erythema, elevated temperature and inflammatory laboratory markers. Additional studies may help differentiate osteomyelitis from Charcot joint.[9]

Given the odds of complications inherent with these procedures, patients must be compliant with the required postoperative care to obtain the best chance possible for a functional outcome. While these two patients have had a good short term outcome, it is important to consider that surgical complications are relatively high in this patient population. Even with a successful surgery, constant vigilance is required to prevent the underlying diabetes complications of associated wounds, infection and prolonged treatment.

Despite the high rate of complications in certain situations, arthrodesis might allow for limb salvage. The future may be more promising as salvage options for failed arthrodesis using a blade plate have recently been reported as well.[10] Research into the pathophysiology may lead to new targeted treatments for treatment and perhaps prevention.[7]

References

1. Van der Ven A, Chapman C, Bowker J. Charcot neuroarthropathy of the foot and ankle. *J Am Acad Ortho Surg.* 2009;17:562-571.

2. Eichenholtz SN. *Charcot Joints.* C.C. Thomas; 1966.

3. Shibata T, Tada K, Hashizume C. The results of arthrodesis of the ankle for leprotic neuroarthropathy. *J Bone Joint Surg.* 1990;72A:749-756.

4. Caputo G, Ulbrecht J, Cavanagh P, Juliano P. The Charcot foot in diabetes: six key points. *Am Fam Physician.* 1998 Jun 1;57(11):2705-2710.

5. Shen W, Wukich D. Orthopaedic surgery and the diabetic Charcot foot. *Med Clin N Am.* 2013;97:873-882.

6. Chisolm K, Gilchrist J. The Charcot joint: A modern neurologic perspective. *J Clin Neuromusc Dis.* 2011;13:1-13.

7. Jeffcoate W. Theories concerning the pathogenesis of the acute Charcot foot suggest future therapy. *Curr Diabetes Rep.* 2005;5:430-435.

8. Palena L, Brocco E, Manzi M. Critical limb ischemia in association with Charcot neuroarthropathy: complex endovascular therapy for limb salvage. *Cardiovasc Intervent Radiol.* 2014;37:257-261.

9. Varma A. Charcot neuroarthropathy of the foot and ankle: a review. *J Foot Ankle Surg.* 2013;52:740-749.

10. DiDomenico L, Brown D. Limb salvage: Revision of failed intramedullary nail in hindfoot and ankle surgery in the diabetic neuropathic patient. *J Foot Ankle Surg.* 2012;51:523-527.