CHAPTER **41**

STAGED SURGICAL MANAGEMENT OF THE CHARCOT FOOT

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Since it was first described by Musgrave in 1703, in regard to joint degeneration associated with venereal disease (1), and then detailed by Jean-Martin Charcot in 1868, in regard to cases associated with syphilis (2), Charcot neuroarthropathy (CNA) has been a challenging condition for surgeons to manage. A strong association exists between neuropathic degeneration of the foot and diabetes mellitus, and prevalence of this relationship was described by Jordan in 1936 (1), although a wide range of sensory and autonomic neuropathies are known to lead to the development of CNA. In fact, conditions such as meningomyelocele, spinal cord injury, syringomyelia, long-standing alcoholism, chronic renal failure, as well as syphilis and diabetes mellitus, have all been associated with the Charcot foot.

Despite the numerous conditions associated with CNA, the precise cause of neuropathic bone and joint degeneration remains unclear. Two main theories, however, have been used to explain the development of CNA, namely, the neurotraumatic and neurovascular hypotheses (3, 4). The neurotraumatic theory purports that unperceived microtrauma or injury to an insensate foot results in articular instability, subluxation, and eventual degeneration with bone fragmentation. The neurovascular theory purports that autonomic neuropathy and hyperemia, combined with an imbalance between osteogenesis and osteolysis, results in osteopenia, making bone incapable of sustaining mechanical loads, with resultant articular degeneration. In all likelihood, the combination of autonomic neuropathy leading to abnormal bone formation, and sensory neuropathy leading to insensitivity and susceptibility to trauma, sets the stage for articular degeneration secondary to weightbearing. CNA patients have also been shown to display significantly lower circulating soluble receptor for advanced glycation end products (sRAGE), with an accompanying increase in serum markers of bone turnover, and reduced bone stiffness in the calcaneus not accompanied by reductions in bone mineral density, which suggests the failure of RAGE defense mechanisms against oxidative stress in diabetes (5).

The prevalence of CNA has been reported to be as high as 13% in foot and ankle clinics, and the incidence ranges from 0.15% to 2.5% overall (6). Furthermore, bilateral CNA affecting both feet occurs in <10% of cases, and same site recurrent exacerbation (flare up) occurs in <5% of cases. Still further, the male-to-female ratio of Charcot foot has been reported to range from 1:1 to 3:1 (7). CNA usually presents clinically as an edematous and deformed foot (Figure 1), usually 3° to 7° warmer than the contralateral non-Charcot foot (8). Clinically, it is important to distinguish between Charcot arthropathy, deep vein thrombophlebitis (DVT), and infection, since all three of these conditions usually present with a warm, swollen lower extremity, and the presence of neuropathy can mute some of the pain that is typically associated with thrombosis and infection. Despite the influence of neuropathy, wherein touch-pressure sensation is diminished, deep, aching pain is often localized to the hindfoot and ankle in patients with Charcot arthropathy. Moreover, the adverse combination of sensory neuropathy and gross structural deformity, notably the rocker bottom foot (Figure 1), predisposes to diabetic neuropathic foot ulceration (DNFU), which has been reported to occur in as much as 40% of Charcot foot cases (9). Importantly, CNA is not only associated with foot deformity and DNFU, it is also associated with lower extremity amputation (LEA). In fact, the rate of LEA is 4.1 and 4.7 per 100 person-years ($P > 0.05$), for Charcot foot and DNFU, respectively; and, in patients <65 years of age, the risk of LEA in a patient with a DNFU alone is 7 times greater, and the presence of a DNFU with Charcot is 12 times greater, in comparison to the presence of CNA alone (9).

Eichenholtz (10) classified Charcot arthropathy based on the clinical and radiographic phases of the process, namely: Stage 1 development, consisting of inflammation, and radiographic dislocation and fragmentation; Stage 2 coalescence, consisting of the resolution of inflammation, and radiographic sclerosis and absorption; and Stage 3 remodeling, consisting of the absence of acute inflammation, and radiographic joint consolidation with gross deformity. Furthermore, the multilevel classification described by Schön et al (11) is comprised of 4 patterns of Charcot joint destruction, and is based on the location (I Lisfranc, II cuneonavicular, III perinavicular, and IV transverse tarsal) and

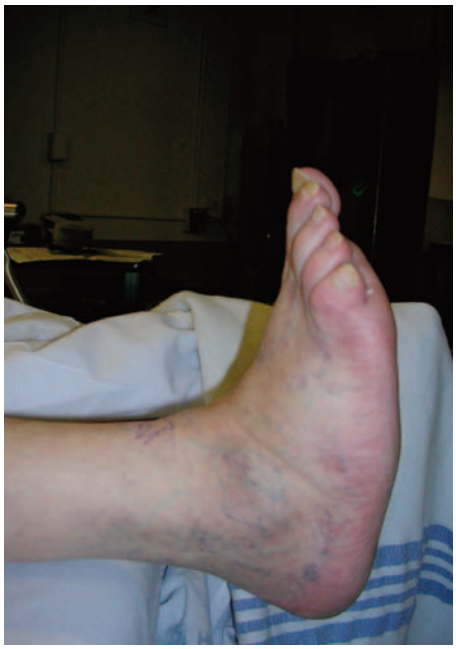

Figure 1A. Clinical appearance of Charcot rocker bottom foot with edema (lateral view).

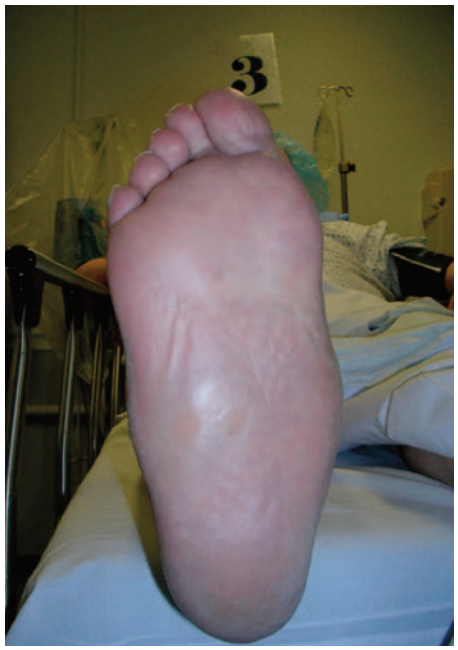

Figure 1B. Plantar view of Charcot foot.

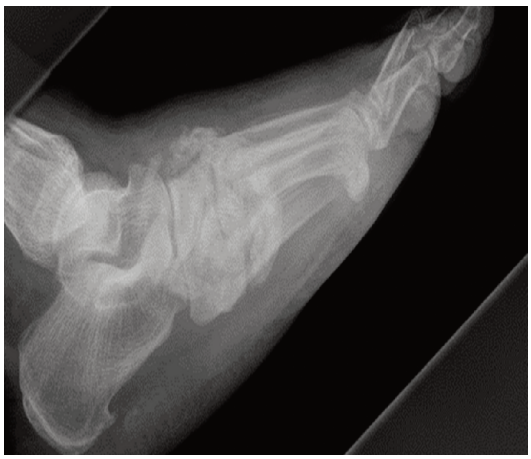

Figure 1C. Lateral radiographic view.

degree (A mild, B moderate, and C severe) of arthrosis. A critical point to keep in mind relative to surgical reconstruction of the Charcot foot is to abstain from elective reconstruction when the foot is acutely inflamed. Moreover, laboratory testing may reveal an elevated erythrocyte sedimentation rate and serum alkaline phosphatase in association with CNA, however the white blood cell count may elevate if osteomyelitis (OM) or soft tissue infection is also present. Imaging studies, such as standard radiographs and magnetic resonance images are important components of the diagnostic evaluation of CNA, however they are rarely definitive in regard to the presence or absence of OM, and bone and synovial biopsies are usually required in order to accurately determine if bone infection is present. Standard radiographs are particularly useful in ascertaining the location and stage of CNA.

Standard therapy for CNA entails offloading the affected foot, wound care if neuropathic ulceration is present, and medical management of the patient's metabolic disorders. Unlike focal foot ulcer care, in order to adequately reduce weight-bearing loads on the Charcot foot, the entire foot usually requires protection. This is, in practice, often difficult, as patient compliance is frequently less than optimal (12). Implementation of nonweight-bearing measures, such as bed rest, the use of a wheel chair, as well as crutches or a walker, can be helpful, but are often used by the patient in just a limited fashion due to comorbidities, including obesity and loss of proprioception, and sometimes visual deficits and cardiac disease, which impede ambulation, particularly when off loading is desired. Braces that "float" the foot and transfer some of the ground reactive forces to the leg can be very helpful, as long as the patient uses them correctly. Such devices include total contact casts, Charcot retraining orthotic walkers (CROWs) and other patellar tendon bearing (PTB) braces (13), and the Zero-G (Universal Medical, http://www.universalmedreps.com/Default.aspx) ankle-foot orthosis (AFO). As a rule, these devices only transfer a portion, but not all, of the weight bearing forces to the leg. It is also important for the clinician to be on the lookout for transfer lesions to the leg when these devices are used, especially if the majority of ground reactive force is redirected from the foot to the leg. Transfer lesions to the

leg, in our experience, have been relatively easy to manage and promotion of healing usually requires additional padding of the leg when the AFO is applied.

Furthermore, stabilization of the foot and ankle by means of external fixation and nonweightbearing can also be useful, especially when concomitant wounds and osteomyelitis exist, or when exostectomy is indicated to remove a focal bony prominence. Useful adjunct therapies that may be indicated in the treatment of CNA include systemic bisphosphonate (14,15) and electrical (16,17) or, possibly, ultrasonic bone growth stimulation. It is important, too, to understand that surgery in patients with CNA is associated with a greater prevalence of complications in comparison to non-Charcot arthropathy patients (12).

Assuming that associated cutaneous wounds heal, offloading efforts, in our opinion, need to be vigorously employed for 6 to 12 weeks initially, in order to resolve acute Charcot inflammation, and to invite coalescence and remodeling in a suitable (plantigrade) alignment. Thereafter, sustained use of an extra-depth shoe with a tapered roller outer sole and an accommodative inlay is usually required (18). Recurrent Charcot flare up, or the development or recurrence of neuropathic foot ulceration, serve as indicators, in some cases, to proceed with staged reconstruction of the involved foot and ankle by means of intraosseous transarticular beaming. Grant and colleagues (19) retrospectively described the results of this method in 71 Charcot feet in 70 patients, treated between January 1994 and January 2008. Their cohort consisted of 22 (31%) isolated hindfoot, 20 (28%) isolated Lisfranc, 29 (41%) combined hindfoot and Lisfranc deformities, which were followed for mean of 39 ± 23 months. Their patients underwent beaming of the medial and lateral columns of the foot with or without concomitant subtalar joint arthroereisis or fusion, and showed statistically and clinically significant radiographic improvement that was maintained throughout the observation period. Observed complications included 5 (7%) procedures (feet) that developed a first submetatarsal ulceration secondary to prominence of the screw used to beam the medial column, 1 (1.4%) foot that developed a postoperative transfer lesion under the second metatarsophalangeal joint after ambulation was resumed postoperatively, 6 (8%) pin tract infections, 4 (6%) broken pins, 8 (11%) cases of osteomyelitis, 10 (14%) medial incision dehiscences, and 4 (6%) medial column broken screws with resultant nonunion of the column.

In another published report, Lamm and colleagues (20) described an effective minimally invasive surgical technique for the staged treatment of Charcot deformity, which they performed on 11 feet in 8 neuropathic patients. In their case series, osseous realignment was achieved through gradual distraction of the joints with adjustable external fixation, after which minimally invasive arthrodesis was performed with rigid internal fixation. The feet were operated on at various stages of Charcot deformity, including Eichenholtz stage I (1 foot), Eichenholtz stage II (6 feet), and Eichenholtz stage III (4 feet). When they compared the mean change in preoperative and postoperative radiographic angles, the transverse plane talar-first metatarsal angle ($P = 0.02$), sagittal plane talar-first metatarsal angle ($P = 0.008$), and calcaneal pitch angle ($P = 0.001$) were all found to be statistically significant. Observed complications included 3 (27.3%) operative adjustments of external or internal fixation, 4 (36.4%) broken wires or half-pins, 2 (18.2%) broken rings, and 11 (100%) pin tract infections. Notably, however, they observed no deep infection, no screw failure, no recurrent ulcerations, and no amputations were indicated over the mean 22-month follow up observation period.

In our own prospective case series, consisting of 9 feet in 7 patients, treated between June of 2009 and January of 2011, staged reconstruction, consisting of: Stage 1—offloading, wound care, removal of OM and packing with antibiotic impregnated calcium sulfate beads, removal of bony prominences, and stabilization with external fixation; and Stage 2—removal or modification and maintenance of the external fixation frame and transarticular beaming of the medial and lateral columns (Figures 2, 3), satisfactory results were observed in 8 (88.9%) of the 9 feet. The mean age of the patients in the series was 55 years (range 41 to 68 years), and their mean body mass index was 28 (range 25 to 33). The series included 6 (85.71%) males. Overall, the duration of diabetes prior to Charcot foot reconstruction was 14 years (range 9 to 41 years), and the mean HbA1c was 9.6 (5.8 to 9.3) mg%. The patients had a median of 3 comorbid disorders (range 0 to 6).

Categorization of the 9 feet, using the classification described by Schön et al (11), revealed 4 (44.4%) I-C (Lisfranc, severe), 2 (22.2%) II-C (cuneonavicular, severe), 2 (22.2%) III-C (perinavicular, severe), and 1 (11.1%) IV-C (transverse tarsal, severe) degenerations. All of the patients received systemic bisphosphonate therapy (intravenous 4 mg zoledronic acid), and bone growth stimulation, 5 (55.6%) received low-intensity ultrasound, and 4 (44.4%) induction electrical stimulation. The mean duration of time between the first and second surgical stages was 12 weeks (range 7 to 21 weeks), and the mean time to radiographic consolidation of the beamed feet was 15.4 (range 5.25 to 27) months. Furthermore, the mean time to full weight bearing in accommodative shoe gear after beaming was 13 (range 9 to 20) weeks. The incidence of complications was 67% (6 of the 9 feet), and included at least one pin tract infection in every operated foot, hardware migration in 2 (22.2%) feet,

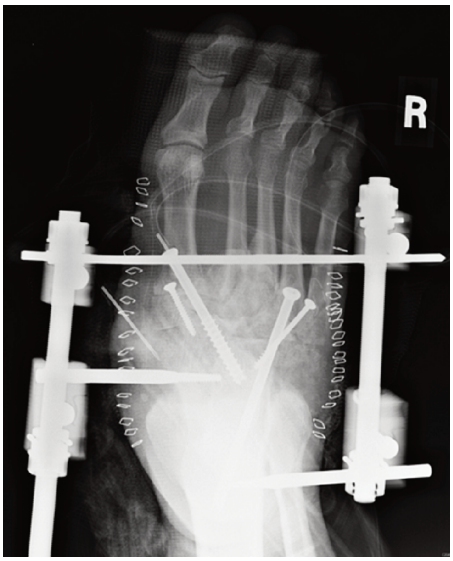

Figure 2A. Anteroposterior view of the patient shown in Figure 1 after application of internal fixation devices (beams), and continued use of the external fixation frame, as well as a posterior splint.

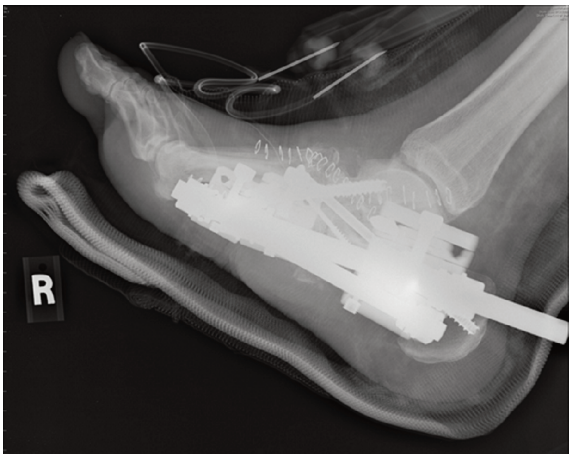

Figure 2B. Lateral radiographic view of the patient shown in Figure 1.

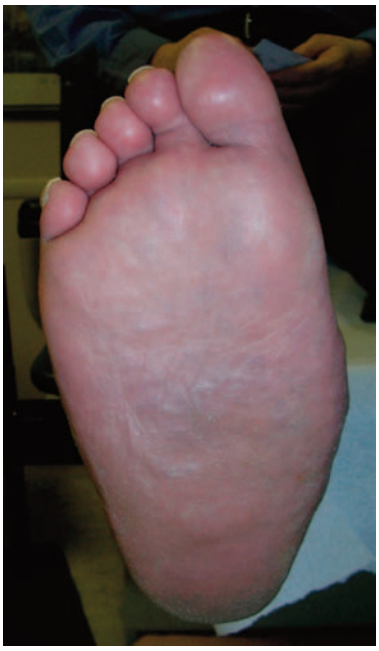

Figure 3A. Postoperative plantar view of the patient shown in Figure 1 more than 1 year after removal of the external fixation of frame and internal transarticular beaming.

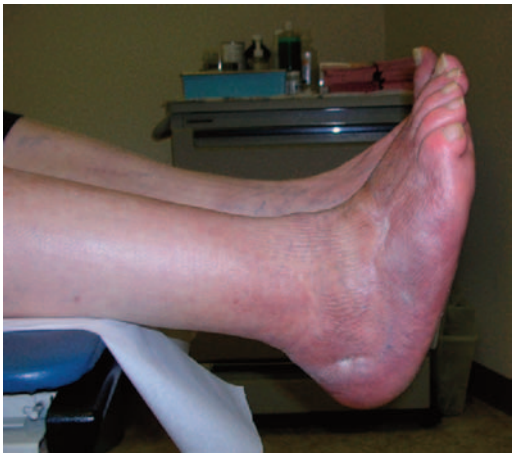

Figure 3B. Lateral view more than 1 year following internal transarticular beaming and removal of the external fixarion frame.

and nonunion in 4 (44.4%) feet (2 medial column, and 2 lateral column) after beaming. There were no cases of deep infection following beaming, during the observation period. Overall, 1 foot (11.1%;14.3% of the patients) required a return to the operating room to remove and replace a displaced medial column fusion bolt that migrated out of the calcaneus. That particular patient recovered fully, and progressed without relapse for >13 months since undergoing the revision.

In conclusion, staged reconstruction of the deformed Charcot foot, consisting of application of an external fixator in order to achieve a quiescent foot, followed by intraosseous transarticular beaming of the pedal columns, appears to be safe and effective. Adjunct therapies that may be employed include wound care and the surgical treatment of OM, as well as gradual or acute realignment, excision of bony prominences, use of bisphosphonates, and bone growth stimulation. Although variations on this approach to the surgical management of the Charcot exist, the method has not be investigated by means of a prospective cohort study, and it has not yet been compared to any other form of treatment in a randomized controlled fashion. For these reasons, it is premature to say that the method described in this report is better than any other surgical procedure. Based on our understanding of the existing literature, as well as our experience with the series of patients described in this report, staged reconstruction is a reasonable operative approach to this complicated deformity.

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