

Treating Charcot Arthropathy Is a Challenge

Explaining Why My Treatment Algorithm Has Changed



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KEYWORDS

- Charcot • Neuroarthropathy • Charcot reconstruction • Staged approach to Charcot
- Surgical algorithm for Charcot • Charcot challenges • Diabetic reconstruction
- Diabetic limb salvage

KEY POINTS

- The background, pathophysiology, and the conservative care options available for the Charcot patient are discussed.
- Foot and ankle surgeons attempting reconstruction and ultimate salvage of Charcot deformed limbs face challenges.
- The staged protocol used by the senior author, experiences, and outcomes of this approach are discussed, as well as the benefits versus a single-staged approach.

INTRODUCTION

First described in 1703 by Sir William Musgrave¹ but brought into recognition by Jean-Martin Charcot in 1868,² osteo-neuroarthropathy was most commonly caused by tabes dorsalis (Figs. 1–13). Because of this, Charcot himself dubbed the term tabetic foot in 1883 even after obtaining his eponym.¹ However, tabetic foot has been replaced by the term diabetic foot as the leading cause of Charcot neuroarthropathy. With a reported prevalence of 0.1% to 0.4%^{1,3} in the general population, Charcot neuroarthropathy prevails in up to 3.0% in patients with diabetes.⁴

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Fig. 1. Case 1: (A, B) Preoperative anteroposterior (AP) and lateral radiographs. The lateral radiograph demonstrates the sequela of an equinus contracture resulting in a negative calcaneal inclination angle and a dislocation of the midfoot. The AP view demonstrates a midfoot overload secondary to a metatarsal adducts, resulting in overload of the lateral column, resulting in a long-standing diabetic foot ulcer that lead to osteomyelitis.



Fig. 2. Case 1: A clinical view following wound and bone debridement. An osteotome was used to fracture, or perform, an osteotomy of the midfoot and harvest bone for a culture and pathologic testing. Note the skin wrinkles in the midfoot as the foot was manipulated and anatomically reduced, resulting in offloading the wound or ulcer. Negative pressure therapy was used for granulation of the wound while the patient received 6 weeks of intravenous antibiotics.



Fig. 3. Case 1: Postoperative well-healed wound following a posterior muscle lengthening, bone debridement, reduction of the deformity into anatomic alignment, application of external fixation, negative pressure therapy, and intravenous antibiotics. The wound has healed well and the soft tissue envelope is optimized for reconstructive surgery.

PATHOPHYSIOLOGY

Charcot neuroarthropathy is characterized by the gradual progressive destruction of bone and joints,² yet the exact pathophysiology is still unknown. The disorder involves episodes of active and inactive phases⁵ with a rapid onset of swelling, increased temperature, and sometimes discomfort. Typically, many patients with Charcot neuroarthropathy have long-standing diabetes for more than 10 years and often have good perfusion.¹ Classic hypotheses, along with several recent theories, are discussed.^{1-3,6}

The neurovascular theory, or French theory, was proposed by Charcot in 1868.^{5,7} In his study of more than 5000 subjects, he suggested that autonomic neuropathy was the main cause because of changes in spinal trophic centers of the anterior horn.⁷ This autonomic neurogenic loss of vasomotor tone, or autotomy, results in arteriovenous shunts opening into Volkmann and haversian canals.¹ An approximately 30% to 60% increased blood flow into bone allows a hyperemic demineralization of bone to occur with osteoclastogenesis. With minerals being washed out and uncontrolled stimulation of osteoclasts, bone becomes osteopenic with a high propensity for breakdown in the insensate foot.^{1,2} Evidence of a vascular-inflammatory connection is seen with Charcot neuroarthropathy secondary to revascularization.⁸

A competitive philosophy is the neurotraumatic, or German theory, relating to unperceived trauma.¹ Reportedly, diabetic patients have a higher incidence of fractures compared with persons without diabetes.⁹⁻¹¹ Sensory neuropathy permits repetitive microtrauma resulting in inconspicuous stress fractures and joint destruction.

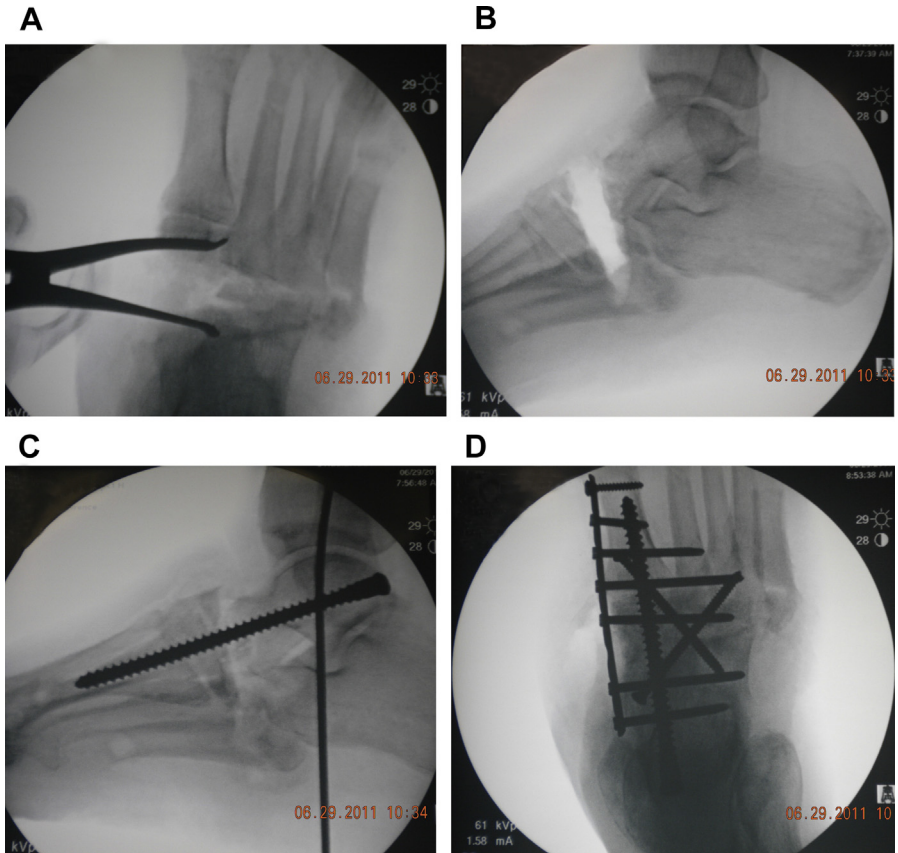


Fig. 4. Case 1: (A, B) Intraoperative radiographs of an internal amputation resecting the diseased Charcot bone and osteomyelitis, providing a surgical, as well as a medical, cure via intravenous antibiotics. (C, D) Intraoperative radiographs of deformity reduction with rigid internal fixation. C demonstrates a full-threaded solid 6.5 bolt realigning Meary's angle. D demonstrates the midfoot aligned into anatomic alignment with a 6.5 solid full-threaded bolt coupled by independent screw fixation and plating. Note that talar first metatarsal angle is re-established. The bone voids were packed with bone graft, giving the mid-foot an excellent chance to successfully fuse. By performing the bony resection, placing the foot into anatomic alignment, and obtaining an arthrodesis, the deforming force is removed and the patient should experience a permanent long-term correction.

Persistent degeneration becomes permanently deformed, showing the importance of neural control of skeletal homeostasis.¹² Obesity has been speculated to overload joints and accelerate the formation of Charcot. Although it is associated with Charcot, elevated body-mass indices do not increase the risk of acute Charcot neuroarthropathy.³ Additionally, some case reports followed diabetic neuropathic patients who underwent successful bariatric surgery who developed acute Charcot.¹³ It was concluded that, even though diabetes had gone into remission, the end-organ damage it had caused (eg, peripheral neuropathy) did not change. These patients became more ambulatory, increasing the stress to their numb feet.

Excessive osteoclastic activity has been reported during acute Charcot neuroarthropathy.⁷ Osteoclastogenesis is mediated by receptor activator of nuclear factor

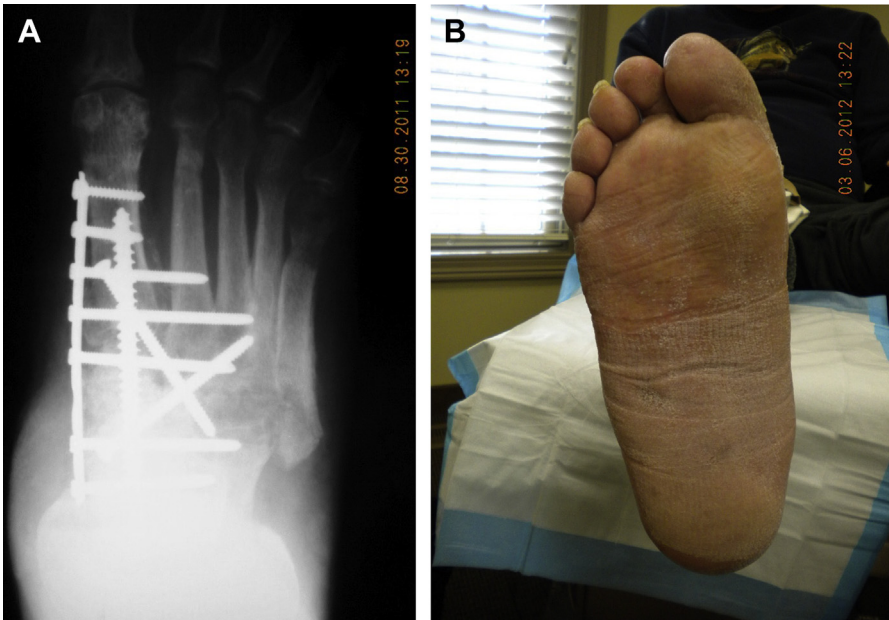


Fig. 5. Case 1: (A) A 6-year postoperative AP view demonstrating good anatomic alignment and a successful arthrodesis. (B) A 6-year postoperative clinical view demonstrating good anatomic alignment, a well-healed wound, and the deforming force permanently removed, leading to a good long-term outcome.

kappa- β ligand (RANKL) and modulated by the RANKL–osteoprotegerin balance. If this equilibrium is disrupted, osteoclastic activity will have no negative feedback and osteopenia will arise.¹ However, a study¹⁴ showed a RANKL-independent pathway revealing osteoclastic precursor cells primed with aggressive behavior in patients with Charcot neuroarthropathy. This pathway was filled with proinflammatory factors that stimulated osteoclast formation, separate from RANKL.^{1,15} Prolonged inflammation and osteolysis induced by Charcot neuroarthropathy resembles that of rheumatoid arthritis and periodontal disease.² Advanced glycation end products from hyperglycemia also affect cortical bone by increasing RANKL activation and osteoblast apoptosis.¹⁶

Leptin has been found to be related to bone mass.⁹ However, in the diabetic patient, leptin hormone levels are increased, which in turn substantially reduces bone mass. This is a result of a receptor mutation, blocking the hormone and its signaling. Solid organ transplantation has likewise been reported with Charcot neuroarthropathy.^{17–21} One study showed 5% of simultaneous pancreatic-renal transplant patients acquired Charcot neuroarthropathy within their first post-transplant year.¹⁷ Situations such as this may be due in part by heavy corticosteroid use, impaired renal function, or nutritional deficiency that cause bone resorption.²²

A combination theory has moreover been described by blending the different processes. For example, the autonomic neuropathy causes bone demineralization because the sensory neuropathy is amenable to uncompensated microtrauma.¹ During the acute stages of the disease, Charcot neuroarthropathy typically is made up of soft bone, whereas later stages show hard, brittle bone. In a recent study comparing trabecular quality histologically, Charcot patients demonstrated thin trabeculae with

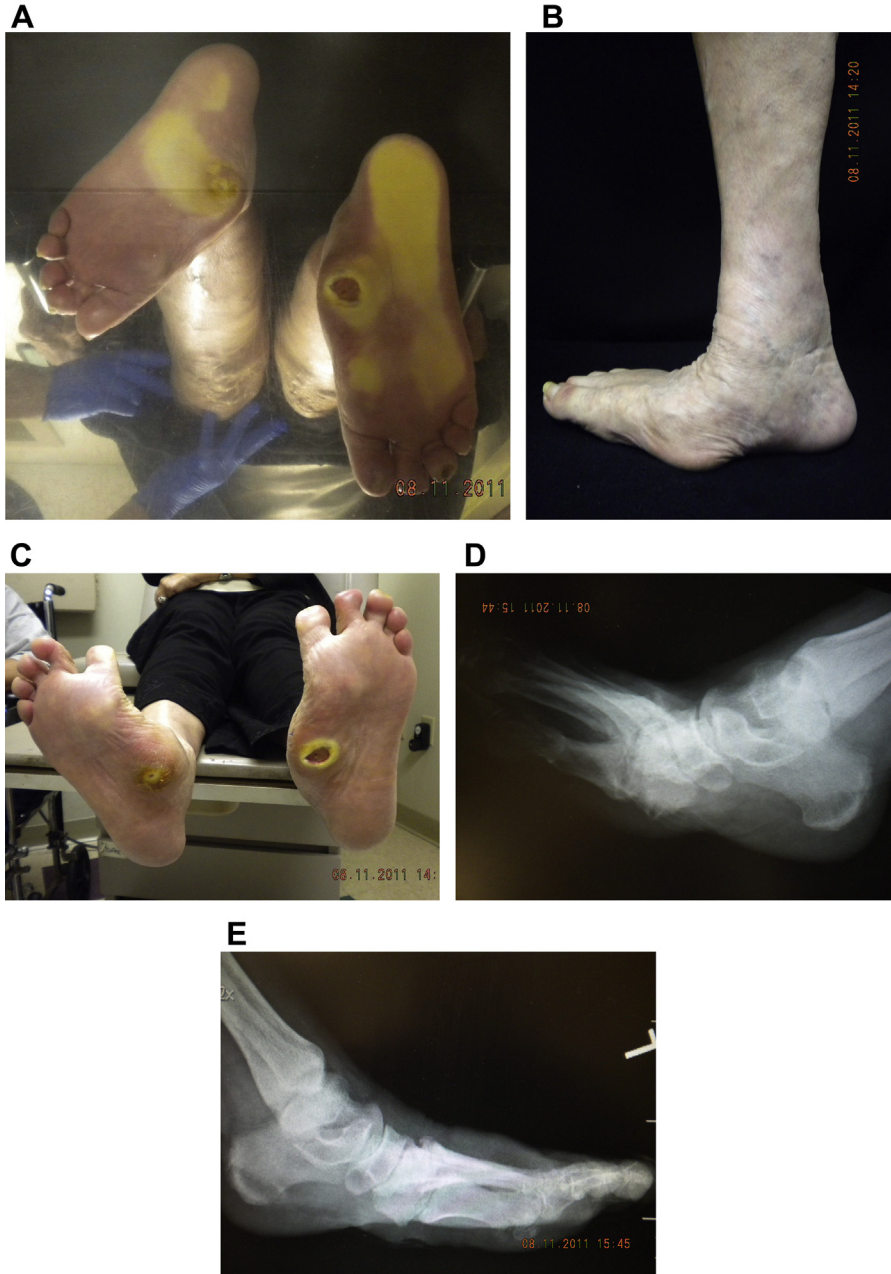


Fig. 6. Case 2: (A) Bilateral midfoot Charcot arthropathy. Unfortunately the patient has experienced chronic bilateral diabetic foot ulcers secondary to the midfoot deformity, resulting in a chronic ulceration with osteomyelitis, making the patient susceptible to limb loss bilaterally. Note the abnormal weightbearing pattern of the feet, leading to chronic ulceration and infection. (B, C) Significant deformities of bilateral feet causing long-standing deformities and chronic ulcers with osteomyelitis. (D, E) Radiographs demonstrating a lack of integrity throughout midfoot with significant Charcot arthropathy of both feet.

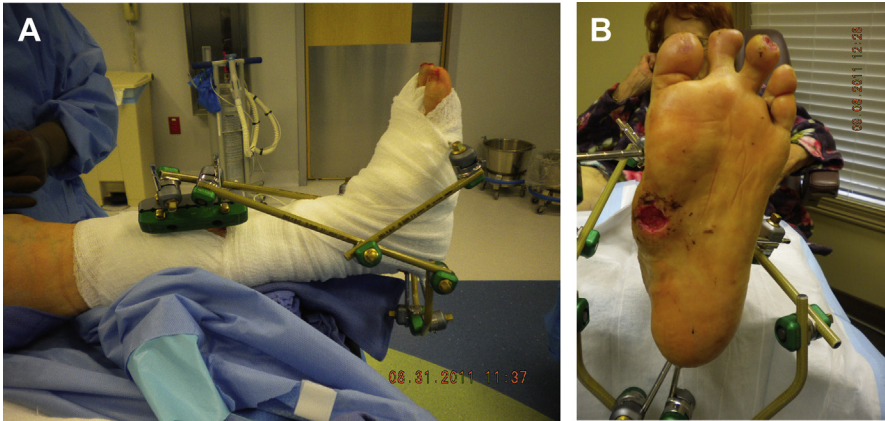


Fig. 7. Case 2: (A) Intraoperative view of the patient having a compressive bandage applied, following the posterior muscle lengthening, bone debridement for cultures and pathologic testing, manipulation and reduction of the foot deformity, application of an external fixator, wound negative pressure therapy, and a compressive postoperative bandage. (B) A post-operative visit demonstrating a wound that is granulating nicely with no evidence of infection. Note the foot is anatomic alignment and stable allowing the wound to be off-loaded with easy access to the wound.

inflammatory infiltrates and hypervascular myxoid tissue.² Other publications indicate that bone in diabetic patients is weaker with 1 study comparing young diabetic animal models with older nondiabetic animals. The results found similar structural changes in both groups and suggested that diabetes may increase the normal aging process.²³ Interestingly, osteoporotic bone, whether in a geriatric or a long-standing diabetic patient, will generally be stiff but brittle with increased risk for fracturing, whereas poorly mineralized bone in a pediatric patient will be very flexible, which decreases the risk of fracture.

CONSERVATIVE CARE

Whether surgical or nonoperative, the treatment objective for Charcot neuroarthropathy is to achieve a plantigrade foot with bony stability.² Traditionally, nonoperative treatment has been used with offloading devices, including total contact casting, Charcot restraint orthotic walkers (CROWs), and bracing.^{1,6,24,25} Although some patients will have a debilitating fixed deformity or gross instability that may not respond to bracing or casting alone,⁵ 1 investigator noted that 60% of patients with midfoot Charcot neuroarthropathy attained a desirable outcome without surgical intervention.²⁶ Serial radiographs are important to carefully observe further fragmentation in the acute stage or coalescence with adequate immobilization.

Because a rigid bony deformity is very likely in patients with Charcot collapse, evidence clearly shows that effective offloading reduces the likelihood of ulceration, as well as amplifies the odds of healing an ulcer.²⁷ Pressure relief is essential. In a small study,²⁸ ulceration rates of subjects with and without Charcot neuroarthropathy were followed using custom orthotic treatment. Before orthotics, the ulceration rate of Charcot subjects was 73% (compared with 31% of non-Charcot). After 1-year follow-up, rates reduced dramatically to 9.8%, which was statistically significant. Yet in the acute phase, a total contact cast is preferred. Duration of offloading is guided by clinical assessment of edema, erythema, and skin temperature changes.¹

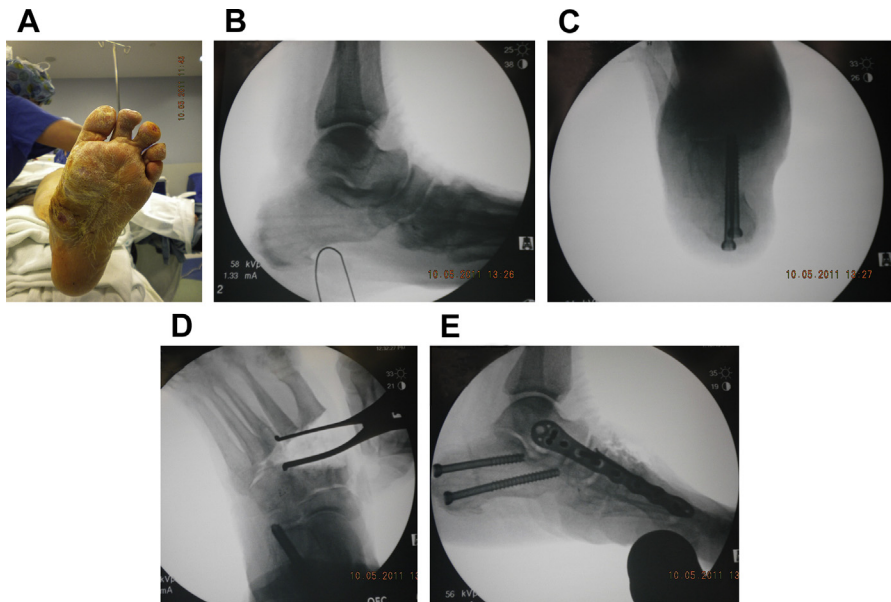


Fig. 8. Case 2: (A) Clinical view of the right foot after the external fixator is removed. Note the wound completely healed and soft tissue envelope is intact with no stress or inflammatory effects of the disease process, resulting in the patient being optimized for surgical reconstruction. (B) Lateral intraoperative view demonstrating a percutaneous calcaneal osteotomy with a Gigli saw. (C) Intraoperative view of a calcaneal axial view demonstrating medialization of the calcaneus with 2 large cancellous screws for fixation. (D) Intraoperative view demonstrating an internal amputation, an aggressive bone resection of the diseased midfoot removing the pathologic bone. (E) Intraoperative lateral view following a gastrocnemius recession, a percutaneous calcaneal displacement osteotomy, an aggressive bone resection (an internal amputation) with realignment and rigid internal fixation. Note the increase in the calcaneal inclination angle, the well-aligned Meary's angle and a recreation of the arch off-loading ulceration and the original deformity.

Conservative treatment involving pressure-offloading devices can be effective biomechanically. On the pharmacologic aspect, bisphosphonate mechanism of action is used to decrease osteoclastic resorption and increase osteoblastic activity. A small study^{29,30} was conducted comparing alendronate against placebo. Both treatment groups were managed with standard offloading regimens. Although there was no report on clinical differences, investigators noticed a statistically significant decline in markers of bony turnover. As an alternative to bisphosphonates, calcitonin can be used in patients with renal insufficiency.¹ Vitamin D levels should also be tested because this deficiency can aid in bone loss.²²

However, unless acute Charcot neuroarthropathy is well-managed from beginning, most patients will have some instability or bony prominence that will be amenable to surgical intervention. In cases that are mismanaged or missed, reconstruction or amputation may need to be pursued. The argument of limb salvage versus primary amputation has yet to be settled, and evidence-based outcomes on the matter are sparse. To truly evaluate reconstruction versus amputation, one must consider that 85% of patients with a diabetic foot ulceration and/or deformity report being unable to ambulate independently, which limits their physical activity and quality of life.³¹ The claim that primary amputation leads to shorter recovery and quicker return to



Fig. 9. Case 2: A clinical view following bilateral, reconstructive, staged surgery of both feet. The patient's left foot was done initially, followed by the right foot. The feet are in more normal anatomic alignment, therefore eliminating the underlying pathologic condition, creating plantigrade, stable feet, resulting in long-term correction of a severe deformities that are free of ulceration and infection.

function with a prosthesis is grim. Only 47% to 67% of patients who undergo a primary amputation secondary to ischemia or ulceration are able to rehabilitate to independent function with their prosthesis.³² Additionally, patients with a primary lower extremity amputation secondary to diabetes have been shown to have a 30% increase

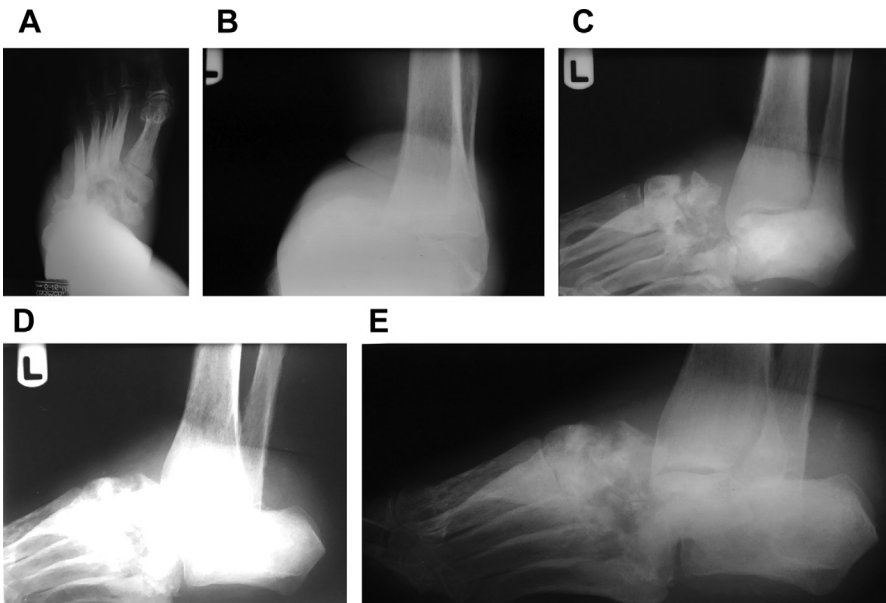


Fig. 10. Case 3: (A–E) Preoperative radiographs of significant Charcot arthropathy involving the tibial talar joint, talar calcaneal joint, and the midfoot.



Fig. 11. Case 3: An intraoperative lateral view of half pins placed in the calcaneus from posterior to anterior preceded by half pins placed in the tibia and midfoot. Placing the half pins in the calcaneus from posterior to anterior provides advantages because this allows more contact of half pin to bone and provides more leverage when attempting to get the ankle out of equinus. Two pins provide the surgeon more control of the hind foot in all 3 cardinal planes.

incidence of depression.³³ Mortality rates following primary amputation reach up to 40% 1-year postoperatively, and 80% after 5 years.³⁴

From an economic standpoint, collected data showed the mean reimbursement for all Medicare services for a patient with diabetic foot ulceration was \$16,700 in 2008, compared with \$36,500 for someone who underwent a major lower extremity amputation.³⁵ Recent retrospective cost analysis has shown that lifelong costs for major lower extremity amputations can average \$509,275, which is about 3 times higher than the costs for patients who undergo reconstruction.³² Given the high level of morbidity and excessive costs associated with amputation, reconstruction by a multi-discipline team has been shown to be effective.^{31–33,35,36}

SURGICAL RECONSTRUCTION

Surgical reconstruction and salvage is typically offered when conservative care has failed. Additional indications are ulceration or preulcerative lesions, osteomyelitis, and pain. In recent studies, the anatomic locations of surgical reconstructions have been changing. The most common locations requiring surgical intervention based on a 54-year review (1960–2014) for patients with Charcot was the midfoot (43.5%), followed by the ankle (33.8%). During the past 5 years, the most common locations requiring surgical intervention were the hindfoot (41.6%), followed by the ankle (38.4%). Although midfoot and hindfoot Charcot have a higher rate of occurrence than the ankle, the midfoot is more amenable to bracing and other nonoperative treatment methods than cases in which the ankle is affected. Additionally, surgeons are quicker to address the posterior muscle group, reducing plantar pressures and halting midfoot breakdown.³⁷

Various fixation techniques have been described and analyzed, including internal fixation, external fixation, combination techniques, and superconstructs. One study discussed a comparison between internal and external fixation techniques for surgical

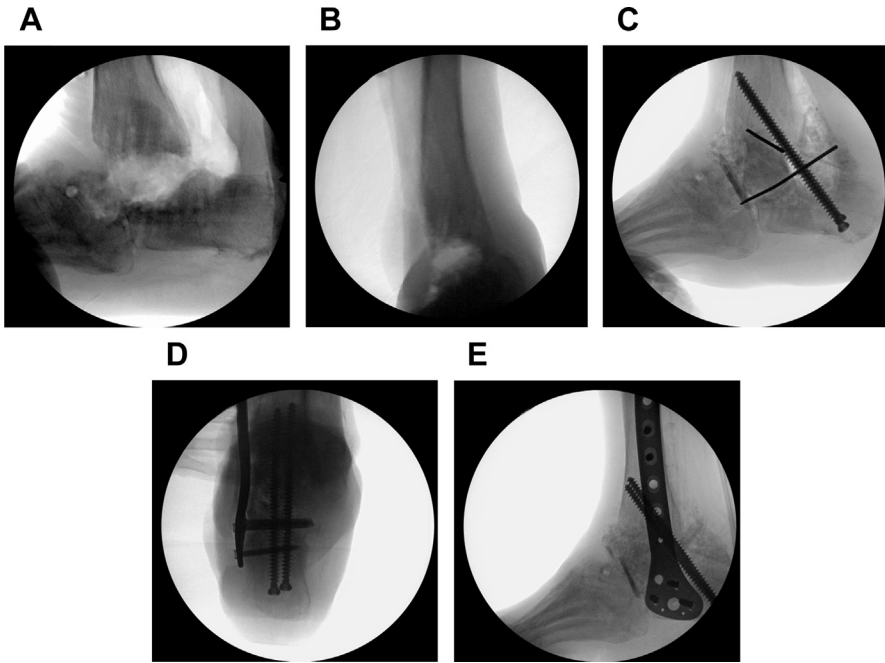


Fig. 12. Case 3: (A, B) Intraoperative view following an internal amputation of the diseased and osteomyelitis bone. (C–E) Intraoperative views following the takedown of the fibula. The distal cortical cancellous block of the distal fibula was used as an inlay graft and the remaining fibula was put into a bone mill and, coupled with allogenic bone, was packed tightly into the bone voids. Fixation is provided through 2 fully threaded solid large cancellous screws and a femoral locking plate.

reconstruction of the foot and ankle in patients who are not infected. The addition of the circular external fixation device did not affect the overall limb salvation rate or complication rate. Overall, the data continue to be too inconclusive to recommend any form of fixation over another.^{36,38} (See discussion of fixation options, in this issue.)

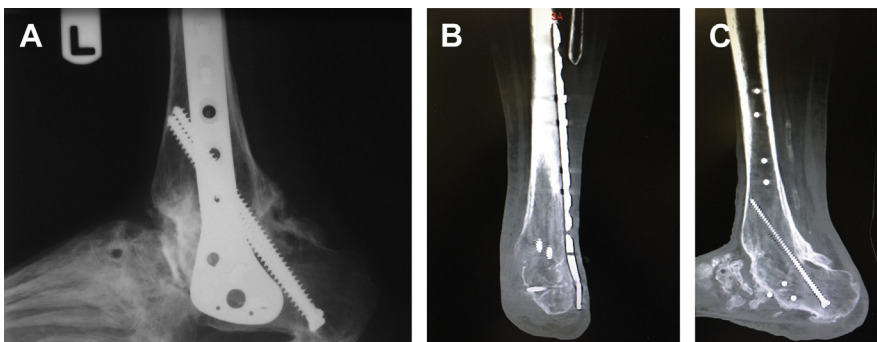


Fig. 13. (A–C) Lateral radiograph postoperatively demonstrating good anatomic alignment and a solid bony union to the tibial talar, talar calcaneal, and midfoot. The computed tomography scan demonstrates a bony union of the hind foot and ankle with anatomic alignment. The bone graft successfully replaced the osteomyelitis and Charcot bone, allowing for good bony union.

The decision on whether to approach surgical reconstruction in a single-stage or a multistage approach is usually based on patient deformity and surgeon preference. Studies have been inconclusive about whether reconstruction during the acute phase is beneficial. Typically, patients requiring reconstruction have had ulcerations, osteomyelitis, or significant deformity. It was these characteristics that led the senior author to use a staged approach.

TRANSITION TO STAGED PROTOCOL

The complexity of the Charcot patient is often underestimated. This patient population includes some of the most ill patients a foot and ankle surgeon will encounter. Quite often these patients have uncontrolled diabetes, peripheral vascular disease, obesity, hypertension, cardiac issues, and smoke tobacco.^{4,6,8,11,25} Complete lifestyle modification and medical optimization is required to sustain a biologic environment for healing and recovery. Referrals to multiple medical specialties are necessary to correct the metabolic imbalances. Endocrinology, vascular surgery, and infectious disease are involved in virtually all Charcot reconstructions.

In addition to the systemic disease processes, these patients have significant deformity of the foot and ankle, often with severe ulcerations. Data have shown that postoperative complications and infection rates decrease if surgery can be postponed until wounds are closed.³ Additionally, a recent study by Sohn and colleagues³⁹ found that those patients with Charcot deformity and an ulceration were 12 times more likely to undergo a major amputation than those without ulceration. The value of a healthy soft tissue envelope was another driving force to the staged protocol. Controlling the edema, resolving wounds, and proper moisturizing techniques can help reduce future wound complications and decrease the patients' risk for limb loss. In a study by Aragón-Sánchez and colleagues,⁴⁰ major amputation was recommended for 2 cases of severe Charcot deformity complicated by osteomyelitis. Four major steps were used for limb salvage:

1. Partial removal of infected bone by curettage
2. Culture-guided postdebridement antibiotic treatment
3. Bed rest before placement in a total contact cast
4. Stabilization of the unstable foot using a total contact cast with an opening for performing wound care and to check healing.

Although this protocol was successful, most patients requiring reconstruction have significant deformity and soft tissue breakdown. Treatment with an external fixator is more amenable to providing stability and addressing wounds and soft tissues complications, while also forcing patient compliance.

Staged Reconstruction

First stage of the reconstruction surgery

After medical optimization and appropriate consultations, the patient undergoes the first stage of reconstruction. Typically, this patient population experiences an equinus contracture of the posterior muscle group. A Silfverskiöld test is performed once the patient is under anesthesia to determine if the equinus contracture is gastrocnemius or gastrocnemius and soleus in nature. Based on the results of the Silfverskiöld test, a gastrocnemius recession is performed either open or endoscopically.³⁵ Once the posterior muscle group is lengthened, the surgeon can mobilize the deformity much more easily, reduce the deformity, and maintain the ankle out of equinus. The posterior muscle lengthening procedure is mandatory to obtain an adequate

reduction. Attention is directed to the wound and the deformity. This generally consists of wound debridement or excision, and bone debridement if bone is exposed. Bone is sent for pathologic testing, as well as for cultures and Gram stain. Next, manipulation and reduction of the deformity is performed and stabilization of the deformity in anatomic alignment is accomplished with the use of a multilevel external fixator. Application of negative pressure therapy is applied to the wound to assist with wound treatment. All affected bone or bone that has been exposed, is debrided, biopsied, and sent for cultures. Depending on the deformity, an osteotome, rongeur, or curette is used to harvest and resect bone. If the deformity is flexible, then the deformity is reduced and placed into anatomic alignment and fixated with an external fixator. If the deformity is rigid, an osteotome is used to weaken and/or fracture through the deformity, bone is harvested for pathologic testing and cultures, and then manipulation and reduction of the deformity into a more normal alignment is performed. A multilevel external fixator (typically, we use a bar-clamp external fixator) is used to maintain correction in the desired anatomic position. The external fixator provides stability and maintains the deformity in anatomic alignment; therefore, offloading the wound by correcting the deformity that was causing the wound. Additionally, it provides easy access to the wound. The patients are admitted to the hospital and an infectious disease consultation, as well as social service consultation are made in attempt for skilled nursing. Bone and tissue cultures are evaluated and followed by an infectious disease specialist who typically manages any infection with intravenous antibiotic therapy. During the postoperative course, the inflammatory markers are evaluated. The inflammatory markers often mirror the wound. Because of the reduction of the deformity, as well as the offloading of the wound, along with negative pressure therapy, the wound typically goes on to heal uneventfully, closing the soft tissue envelope. In cases of large and more complicated wounds, negative pressure therapy is performed weekly and advanced wound care and/or split-thickness skin grafting is used in attempt to close the wound. Edema control with bandaging is performed at every dressing change to prevent soft tissue complications, as well as pin tract infection. Appropriate moisturizing is also implemented to improve soft tissue integrity. Once wound healing is achieved, preparation for next definitive stage of the surgical reconstruction begins. It has been the senior author's experience that it is best to maintain anatomic alignment of the foot and ankle and to allow several weeks for the soft tissue envelope to remodel and mature so that the soft tissues are optimized for the definitive stage of the reconstructive surgery. Additionally the inflammatory markers are followed by an infectious disease specialist to assess and to identify when the patient is most optimized for the definitive and reconstructive surgery (Figs. 1–3).

Second definitive stage of the surgical reconstruction

This stage consists of removing the external fixator and thoroughly cleansing the entire lower extremity using peroxide, as well as providing a sterile preparation of the limb, before the surgical reconstruction. Once a good sterile preparation and draping is accomplished, all incisions are made full thickness in an attempt to preserve the soft tissue envelope. Aggressive bone resection eliminating the Charcot bone or infected bone, and realignment and correction of the deformity, is performed via arthrodesis with internal fixation. The bone resection is critical and needs to be aggressive in removing the diseased bone and in management of obtaining definitive anatomic alignment. The aggressive bone resection is often referred to as an internal amputation. Each deformity is patient-specific and deformity-specific. It is the senior author's preference to remove any affected bone, especially bone with positive biopsies or culture.

The aggressive resection of bone is a surgical cure achieved by removing the diseased bone (Charcot and/or infected bone). It is imperative that the surgeon resects the bone in a manner that leaves only healthy, bleeding, viable bone. The resected bone reassessed with pathologic testing and cultures. The bone resection is performed with the intent of surgical excising of the pathologic bone, creating the planes to allow correction of the underlying deformity, and putting the foot and ankle back into anatomic position. New cultures and biopsies are taken from the remaining (what is suggested as healthy) most proximal and distal locations of the bone before the reduction and fixation. Any remaining voids are either backfilled with autograft and/or allograft bone. In cases of midfoot Charcot, the entire midfoot can be resected with an internal amputation and the foot functionally shortened and stabilized with internal fixation. In cases of hindfoot or ankle Charcot deformity, the same theory and principles are maintained. The fibula can be used as a graft for corticocancellous struts, as a biologic plate to aid in fixation and stability, or put through a bone mill to aid in fusion.

Some patients require reconstruction of both the hindfoot and ankle, as well as the midfoot. In these cases, reconstruction of the most proximal segment is typically performed first. Once osseous fusion is noted via radiograph and/or computed tomography (CT) scan, and the soft tissue envelope has recovered, the midfoot reconstruction is performed barring any complications.

Results of staged protocol

Recently, the authors performed a 4-year follow-up of a retrospective review of our 30 most recent patients who underwent a staged Charcot reconstruction with a multiplanar external fixator. Subjects were excluded if they had a single-stage reconstruction. Twenty-seven subjects were identified and 26 charts were available for review. Inclusion criteria included subjects who underwent a staged Charcot reconstruction, with external fixation, wound negative pressure therapy, and arthrodesis. The average age was 60 years and the average body mass index was 37. There were 13 women 13 men. All of the subjects had underlying diabetes mellitus and diabetic peripheral neuropathy.

Ten of the 27 subjects' (37%) bone biopsies were negative for osteomyelitis. The remaining 17 subjects (63%) had a bone biopsy positive for osteomyelitis. All subjects with positive cultures completed a minimum 6-week intravenous antibiotic course. Twenty-four (92%) of the 26 subjects achieved successful limb salvage. Only 2 subjects (8%) went on to below-knee (BK) amputation.

POSTOPERATIVE MANAGEMENT

Postoperative management consists of a postoperative compressive bandage and an univalve split plaster cast³² while keeping the patient is kept nonweightbearing. Typically, the social services department is consulted in an attempt to place the patient into a rehabilitation facility or skilled nursing facility. The postoperative univalve split plaster cast is left on for 2 weeks unless otherwise indicated. At the 2-weeks postoperative visit, another compressive bandage and a BK fiberglass case are applied. The BK cast typically left on for 6 to 8 weeks based on serial radiographs. A postoperative CT scan is commonly ordered to assess the reconstruction site. Compression support hose and moisturizing agents are ordered and recommended for long-term use to assist in the postoperative edema and to help with maintaining the soft tissues. Once bony union is achieved, a CROW walker is used as the bone continues to remodel and mature. The CROW walker provides continued support as the patient begins to ambulate in anatomic alignment and the bone is remodeling. An ankle-foot orthosis (AFO) is ordered for the months or years of transition after a CROW walker, The AFO provides continued support while bony remodeling continues and until full

weightbearing is permitted. Based on the outcome and the deformity, and once complete remodeling has occurred, the attempt is made to place the patient into their regular shoes, if possible, or special custom accommodative shoes based on the deformity. In cases in which a patient may not achieve a bony union but is clinically stable and maintains anatomic alignment, the patient will remain in a brace or CROW walker. The goal is to get the patient into regular shoes, if possible, because long-term compliance with the use of braces or CROW walkers is not desirable to most patients.

SUMMARY

It has been the authors' experience that staging and being patient with this challenging multifactor complex deformities has yielded the best results. We have found that staging the surgery and being patient with the timing of the stage reconstruction allows the host patient's body to react more positively, and to respond and manage the inflammatory response often experienced by the host patient relative to the infectious process and surgery. Additionally, once relief from the initial inflammatory response of the initial surgery and management of the infection are achieved, it seems the patients respond better compared with those who undergo to acute correction. The combination of an aggressive resection (internal amputation) of the diseased bone (Charcot and/or osteomyelitis) along with intravenous antibiotics provides a definitive way of curing and treating a chronic diseased bony infection with a Charcot joint.

Successful limb salvage can be achieved with proper preoperative vascular evaluation and staged correction of the deformity. We recommend noninvasive vascular testing and, if needed, a referral to a vascular surgeon before reconstruction. Initial surgery consists of a posterior muscle lengthening, bone debridement with biopsy and culture, reduction of the deformity, stabilization with an external fixator, and negative-pressure therapy to underlying open ulcerations. When appropriate, a referral to a infectious disease specialist is made. Once the wound is completely healed, and the patient is optimized, the patient undergoes the surgical definitive reconstruction surgery. The reconstructive surgery consist of aggressive bony resection (internal amputation), deformity correction via arthrodesis with internal fixation. Following reconstruction, the patients are placed in a CROW walker, transitioned to AFO, and returned to accommodative diabetic shoe gear when appropriate.

Despite modern techniques using improved methods of fixation and improved patient selection, approximately 9% of patients with Charcot deformities who undergo surgery will require a major amputation.³⁶

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