# 19 Charcot Foot: Surgical Management and Reconstruction

Dane K. Wukich and Venu Kavarthapu

## Introduction

The surgical management of Charcot neuroarthropathy (CN) has evolved over the past several decades. Historically, CN has been treated nonsurgically with offloading and accommodative footwear. Early attempts at surgical reconstruction were associated with high complication rates, and this may have contributed to a longstanding bias against surgical reconstruction. In 1967, Johnson [1] reported on 118 cases of neuropathic fractures and joint injuries. At that time he recognized that inadequately protected fractures, soft tissue sprains, and effusions of neuropathic joints were the precursors of joint destruction. He further stated that trauma in the form of surgery during acute stage could stimulate further resorption. Consequently, Johnson did not advocate surgery until resolution of the acute inflammatory response. Johnson did not advocate surgery specifically for diabetes-related neuropathic foot injuries because of the concern for "circulatory problems."

D. K. Wukich (✉)
Orthopaedic Surgery, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Dane.Wukich@UTSouthwestern.edu

V. Kavarthapu
Odense University Hospital, University of Southern Denmark, Odense, Denmark

King's College Hospital, London, UK
e-mail: venu.kavarthapu@nhs.net

The growth of surgical intervention in treating CN) appears to parallel the introduction and acceptance of new methods of skeletal fixation. In 1958, "Arbeitsgemeinschaft für Osteosynthesefragen" or AO (translated to Association for the Study of Internal Fixation) was founded in Switzerland. The AO method of employing rigid internal fixation for the treatment of fractures was revolutionary and considered radical by some surgeons, particularly in the USA. After two to three decades of basic science research and outcome studies, AO became widely adopted globally. Specially designed plates and screws offered a significant improvement in managing fractures and dislocations [2]. Contemporaneously, in the Soviet Union, Professor Gavril Abramovic Ilizarov developed an equally revolutionary and radical method of treating musculoskeletal injuries and deformities [3]. Ilizarov's method utilized circular external rings and fine wire transosseous fixation to stabilize fractures and deformities. The true genius of Ilizarov was to create an "external ring fixator" that enabled not only static correction, but gradual dynamic correction of major deformities. Ilizarov's technique greatly expanded the surgical management in treating osteomyelitis, and the recognition that distraction osteogenesis was possible, contributed to success in limb salvage surgery. Ilizarov's methods remained isolated to the Soviet Union and eastern bloc countries until the 1980s when surgeons from Italy were able to


C. E. Attinger, J. S. Steinberg (eds.), *Functional Limb Salvage*,
https://doi.org/10.1007/978-3-031-27725-2_19

visit his center in Kurgan, Siberia. Subsequently, the methods of Ilizarov were disseminated across the globe, and circular external fixation has become a widely adopted technique that is often employed in the treatment of CN. Finally, another major advancement in skeletal fixation was developed by Professor Gerhard Kuentscher in Germany. Professor Kuentscher pioneered the intramedullary nailing method for the treatment of long bone fractures. His original book was published in 1947, and the full English translation of "The Marrow Nailing Method" in 2006 [4]. This text was previously unknown until 2004 and gives a remarkable account of his treatment of World War II injuries from the perspective of a German surgeon. Similar to AO principles and the methods of Ilizarov, intramedullary fixation of long bone injuries was initially viewed with skepticism in the USA. Over the past four decades, intramedullary fixation has been universally adopted as the treatment of choice for lower extremity long bone fractures. This technique has been translated to use in CN) reconstruction, and retrograde intramedullary fixation is widely used for complex reconstructions of the ankle and hindfoot. Originally implants designed for the femur were inserted, but now multiple specially designed retrograde ankle arthrodesis nails are available [5].

An early study from the Mayo Clinic (1990) reported on ankle fusion in diabetic neuropathic joints, citing complication rate of 62% including non-union, amputation, and death [6]. The authors cautioned that neuropathic arthropathy contributed to the inordinate complication and failure rates, and ankle arthrodesis should be considered with caution in the diabetic patient. It is now well recognized that patients with CN) may have peripheral artery disease; however, it rarely results in critical limb ischemia. Using noninvasive arterial testing, approximately 13% of patients with CN have ischemia as defined by a great toe pressure of less than 60 mmHg, and only 2.4% of patients had critical limb ischemia defined as a toe pressure of less than 30 mmHg [7]. While adequate perfusion is necessary for successful surgical outcomes, we now recognize the neuropathy and poorly controlled diabetes contribute to high rates of postoperative complications [8–10].

Commensurate with the expanded methods of fixation, enthusiasm for the surgical management of Charcot neuroarthropathy has grown over the past 25 years. Historically, surgical intervention was not recommended until the acute inflammatory response had resolved and consolidation had occurred. One of the earliest reports from Thompson and Clohisy [11] recommended surgical reconstruction in patients whose deformity could not be accommodated in a load sharing orthosis. They also advocated that the skin should be free of ulceration at the time of the procedure.

A systematic review on the surgical management of Charcot neuroarthropathy was published in 2012 [12]. The authors searched databases from 1960 until 2009 and identified 96 articles that met the inclusion criteria of surgical management of Charcot neuroarthropathy. Forty-two of the 96 articles were expert opinion or case reports (44%) and 54 articles were retrospective case series without a control group (56%). The level of evidence for the surgical management of Charcot neuroarthropathy was therefore based on level IV and level V evidence, and there were no controlled retrospective studies or prospective randomized studies. Interestingly, four centers accounted for 51% of all patients reported in this systematic review. The authors offered several conclusions from their study: (1) the evidence for performing or not performing surgery during the acute phase of Charcot neuroarthropathy was inconclusive, (2) the most common locations requiring surgical intervention were the midfoot followed by the ankle, (3) exostectomy was found to be useful to relieve bony pressure that could not be accommodated with orthotics and prosthetics means, (4) Achilles tendon lengthening or gastrocnemius recession reduced forefoot pressure and improved alignment of the ankle and hindfoot relative to the midfoot and forefoot, (5) arthrodesis was indicated for instability, pain, or recurrent ulceration that failed nonsurgical treatment, (6) there was inconclusive evidence to recommend one type of fixation over another (internal versus external fixation).

The same group updated their systematic review searching articles from 2009 until 2014 [13]. An additional 30 manuscripts met the criteria for inclusion, demonstrating that 6.6% of studies were level II prospective comparative studies, 13.3% were level III retrospective case-control study, and 80% were level IV retrospective case series. This updated review demonstrated that the ankle (38.4%) and hindfoot (41.6%) were the most common locations reported for surgery followed by the midfoot (29.6%). The conclusion of this updated systematic review suggested that the published surgical data for Charcot neuroarthropathy was improving as evidenced by higher level studies during the preceding 5 years. The authors also reported that despite improved methods of fixation and improved patient selection, approximately 9% of patients with Charcot neuroarthropathy who underwent surgery required a major amputation.

In 2017 Safavi et al. [14] also performed a systematic review on the outcomes of surgical treatment of midfoot CN. Nine studies were identified, and the authors reported a fusion rate of 91%, amputation rate of 6%, and hardware complication rate of 16%.

One of the most important determinants of a successful Charcot surgical team is to be part of a multidisciplinary program. Cates et al. [15] reported on outcomes of Charcot reconstruction in patients with and without diabetes. The authors are a part of the Georgetown University Diabetic Limb Salvage Program, globally viewed as an excellent multidisciplinary program. Despite their large experience, Charcot reconstruction in diabetic patients was associated with high rates of wound dehiscence (16%), delayed healing (34%), and major lower extremity amputation (26%). When they evaluated their cohort of diabetic patients who were deemed to be well controlled (hemoglobin A1c ≤6.5), the rate of major lower extremity amputation was only 10%. There was little difference in the rate of wound dehiscence (15%) or delayed healing (30%) in well-controlled patients.

The King's College Hospital Program in London UK is also widely globally recognized as an outstanding multidisciplinary diabetic foot care team (MDFT) [16]. They have reported their experience in the management of complex midfoot, hind foot, and ankle Charcot neuroarthropathy as well as their protocol for dealing with infected Charcot joints [17–19]. Both the Georgetown and King's College Programs have a collaborative service that includes specialists in reconstructive surgery, vascular surgery, wound care, and internal medicine (diabetologists) with a mission of providing outstanding patient care and service.

## Controversies of Surgical Management

Many questions regarding the surgical management of Charcot neuroarthropathy remain unanswered or debated. The decision to proceed with surgical intervention is often debated, and often the anatomic location of Charcot determines whether or not surgery will be done. For example, nonsurgical treatment of midfoot Charcot has been reported to be as high as 60% in a large series [20]. Success was defined as being able to wear standard, commercially available therapeutic depth inlay shoes and custom fabricated accommodative foot orthosis. Deformities at the ankle are less well tolerated and more prone to ulceration, particularly when deformity in the coronal plane is present. A consensus document recommended that for severe Charcot deformity of the ankle, surgical management should be considered a primary treatment because coronal deformity of the ankle is poorly tolerated [21].

Traditionally, surgical intervention was not recommended until the acute inflammatory response had subsided and consolidation occurred. There is little evidence to support this, but the dogma was that surgical intervention during the hyperemic phase was associated with higher complication rates. In 2000 Simon et al. [22] reported on a series of 14 patients with midfoot Charcot who underwent early operative treatment during Eichenholtz stage I. All 14 arthrodesis procedures were successful and the meantime to return to assisted weight-bearing was approximately 10 weeks. In 2010, Mittlemeier et al. [23] reported on the outcomes in 22 patients (26 ft) that underwent primary surgical reconstruction. The indications for realignment arthrodesis were instability, non-plantigrade foot, and deformity with ulcer or impending ulceration. They experienced nine complications, five hematomas, and four with postoperative instability. Despite this, all patients achieved a stable and plantigrade foot and no recurrent ulcerations occurred. The authors suggested that surgical reconstruction of the Charcot foot should not be limited to salvage procedures, but early surgical intervention in high-risk patients should be considered. One of the potential limitations of this retrospective study is that only four of 22 patients (18%) were treated during Eichenholtz stage 1. Five years later the same center reported that 19 of 21 patients (90%) who underwent late corrective arthrodesis experienced at least one complication [24].

The choice of fixation (i.e., internal or external fixation) depends on several factors, not the least of which is surgeon preference. One of the major determinants is the presence or absence of active bone or soft tissue infection. Most authors agree that internal fixation is not recommended in the setting of active infection. Some surgeons utilize internal fixation in patients with clinically uninfected wounds, while others prefer to achieve ulcer healing prior to operative reconstruction. Our general approach is to use internal fixation in patients without active infection. In patients with open wounds associated with active infection we prefer external fixation, as a primary procedure or staged. Patients with active infection are also treated with culture directed antibiotics and negative pressure wound therapy if indicated. In select cases, a hybrid type of construct is utilized combining both internal and external fixation, particularly in patients with poor bone quality. While external fixation is an invaluable tool in CN, its use can be associated with a high complication rate in patients with diabetes [25].

Dayton et al. [26] published a systematic review comparing the outcomes of CN using internal or external fixation. Procedures using internal fixation achieved an overall successful outcome rate of 87% of patients and 6.5% required major amputation. Procedures on the foot achieved a higher rate of success than the ankle (93% vs. 84%). In contrast, external fixation was associated with successful outcomes in 93% of patients and the major amputation rate was 3.5%. Procedures on the foot achieved a higher rate of success than the ankle (90% vs. 88%).

Additional data demonstrated from Dayton's review included the fact that internal fixation was used in patients with uncomplicated wounds or osteomyelitis. Screws were preferred for the foot and intramedullary fixation for the ankle. Dayton also acknowledged that external fixation was used primarily in more complicated cases with infection. Pooling of data from the 23 studies found that the odds ratio of successful outcome using internal fixation was significantly less likely than when external fixation was used (OR 0.52, 95% CI 0.30–0.90, $p < 0.05$). The conclusions drawn from this study must be viewed in the context of selective bias.

## Radiographic Evaluation

Standing X-rays of the ankle and foot should be obtained in all patients to include three views. Hindfoot alignment views are also essential to identify subtle varus and valgus deformities. Contralateral radiographs can be especially helpful to assess the normal anatomy. In some cases, osseous anatomy can be so distorted that radiographs are not optimal for preoperative planning in which case advanced imaging is beneficial. Malalignment of the ankle is typically obvious; however deformities of the hindfoot and midfoot can be less obvious. Measurement of certain angles of the foot can be helpful in preoperative planning and predicting the potential for ulceration. In 2008, Bevan and Tomlinson [27] reported that lateral talar-first metatarsal angle measured on weight-bearing radiographs was a simple means of monitoring patients' risk of development of midfoot ulceration. Another radiographic study found that sagittal plane deformities are more likely to be associated with foot ulcerations than transverse plane deformities

[28]. Lateral column involvement was identified by a decrease in the cuboid height, decreased calcaneal pitch, and decreased lateral calcaneal fifth metatarsal angle. Meyr and Sebag [29] recommended against using a single radiographic parameter to predict midfoot ulceration because of significant positive and negative correlations among various angles that could be measured. Given that limitation, excellent reliability for radiographic measurement of cuboid height on subjects with midfoot Charcot neuroarthropathy has been reported [30].

CT scans provide improved osseous visualization and can identify bone loss and dislocations not seen on radiographs. MRI can identify bone injury beyond the suspected area of Charcot as evidenced by increased signal. MRI can also be useful in cases of suspected osteomyelitis in patients with active or healed wounds as well as following the course of CN.

Nuclear medicine can also be helpful in the evaluation of suspected for infected CN by labeling Leukocytes with either (99m)Tc-HMPAO or (111)In-oxine [31]. In experienced centers accuracy in detecting bone infection can be greater than 95%. Another benefit is that white cell labeled scans may be able to differentiate soft tissue infection versus aseptic inflammation. Single-photon emission computed tomography/computed tomography (SPECT/CT) and bone marrow scanning can also improve anatomic resolution of the foot and ankle [31].

## Preoperative Evaluation

Patients with CN often have multiple comorbidities that can increase the perioperative risks of surgery to include cardiovascular disease (hypertension, coronary artery disease, compensated or uncompensated heart failure), diabetic nephropathy or autonomic neuropathy, poorly controlled diabetes, and tobacco use. A thorough preoperative evaluation can predict the risk [32].

Patients undergoing Charcot reconstruction benefit from a structured multidisciplinary evaluation prior to the procedure. At King's College Hospital diabetic foot unit, the preoperative assessment is commenced during their visit to a dedicated "Foot School Clinic" (see figure). The group of patients undergoing Charcot reconstruction procedures are seen along with their personal care providers or family members in the clinic. Detailed and interactive audio-visual presentations are made to the attendees by the members of the MDFT (physician, surgeon, podiatrist, physiotherapist, and occupational therapist), covering the generic information on their perioperative care. This is followed by individual assessment and counseling of each patient, separately, by each member of the MDFT. The physiotherapist provides the information on the prehabilitation regime (PREHAB) whereas the occupational therapist goes through the microenvironment setup at the patient's residence and the regimens used for postoperative mobilization due to the limitation of weight-bearing. All routine perioperative assessments and investigations are completed at this stage.

Routine blood investigations, including CBC, renal and liver profiles, and inflammatory markers (C-reactive protein, ESR, procalcitonin) will provide adequate assessment of surgical fitness. Blood vitamin D levels are often low in this group of patients and it is recommended to routinely provide vitamin D replacement. In the presence of raised inflammatory markers, radiological features of osteomyelitis or a history of previous infection in the Charcot affected region, it is advisable to perform bone biopsies for a definitive diagnosis and microbiological sensitivities.

Bone biopsy can be performed as an outpatient procedure in most patients. The patient should be off antibiotics for at least 2 weeks prior to the biopsy procedure. The location of the bone biopsy target material is determined based on the imaging studies. Local anesthetic infiltration can be applied to the area of skin penetration if the skin sensation is intact. Using aseptic technique, the assembled trocar and cannula of the biopsy instrument is pierced into adequate depth and in the direction based on imaging studies. If an ulcer is present, the biopsy entry point is chosen about 1 cm away from the edge of the ulcer, avoiding areas of active inflammation. Using a standard bone core biopsy technique, the specimen is harvested and sent for microbiology (culture and sensitivities) and histological studies.

Vascular studies are routinely considered prior to Charcot reconstruction, to rule out any significant vascular compromise. If identified, this is best addressed by performing the revascularization procedure about 4–6 weeks prior to the deformity correction. The method of revascularization is beyond the scope of this chapter and will be discussed in other chapters.

## Charcot Bone

Surgeons should recognize that bone is a dynamic organ, and in normal homeostasis, bone resorption and bone formation are in relative balance. It is well recognized that during the active phase of Charcot neuroarthropathy, circulating osteoclasts are significantly elevated and metabolically active resulting in a net loss of bone. Osteoclasts also express inflammatory cytokines such as interleukin-1 beta, interleukin-6, and TNF alpha which facilitate the recruitment, proliferation, and differentiation of osteoclasts. A histopathological and immunohistochemistry study of bone retrieved from patients undergoing Charcot reconstruction demonstrated that even though patients were beyond Eichenholtz stage 1 (active) and in the remodeling phase, expression of proinflammatory cytokines was still present on pathological examination [33]. This finding has implications in planning surgical reconstruction.

Surgeons planning to reconstruct Charcot neuroarthropathy should have an understanding of the quality of the involved bone. Herbst at al. [34] classified the bone injury pattern as either a fracture, dislocation, or fracture dislocation. Bone mineral density was measured in the contralateral femoral neck or contralateral distal radius, and not measured in the involved foot. The authors found that patients who presented with a fracture pattern had significantly lowered *T*-scores in bone mineral density compared to the dislocation group. In fact, the age adjusted odds ratio of a patient with osteopenia according to the World Health Organization criteria as having a fracture rather than dislocation was 9.5. Dislocations and fracture dislocations had normal bone mineral density as measured in their study. The fracture pattern was more likely to be seen in the ankle and foot, while the midfoot mostly involved dislocations. The hindfoot was represented by fractures, dislocations, and fracture dislocations. The authors opined that the osteopenia seen in the fracture group was not a result of regional Charcot neuroarthropathy, because the decreased bone mineral density was observed in the contralateral extremities. They further stated that the success of midfoot reconstruction may be related to the fact that the dislocation pattern has more of a normal bone mineral density compared to the fracture pattern. Limitations of this article included few patients in the combination fracture dislocation group and inclusion of patients presenting at different Eichenholtz stages of the disease. Nonetheless this study highlights an important point, namely that identification of peripheral osteopenia may be a potentially modifiable systemic risk factor in patients with diabetes and neuropathy.

Petrova at al. [35] studied 36 consecutive patients who were treated for acute Charcot neuroarthropathy. They measured bone mineral density of the calcaneus from the involved foot and compared it to bone mineral density from the contralateral uninvolved foot. The authors found that the bone mineral density of the involved foot was significantly decreased when compared to the contralateral foot at presentation, after 3 months of casting and at clinical resolution. There was a significant decrease in bone mineral density from the time of presentation until the time of casting at 3 months. After 3 months no significant further decrease in bone mineral density occurred. Contrary to the opinions of Herbst et al. [34], the authors felt that this reduced ipsilateral bone mineral density was secondary to proinflammatory induced osteolysis.

Greenhagen et al. [36] prospectively studied central (core) and peripheral bone mineral density in a cohort of diabetic and non-patients. Peripheral bone density was measured in the calcaneus of the extremity affected by CN, while the core bone density was measured in the lumbar spine. The diabetic cohort was comprised of two groups, one who had Charcot neuroarthropathy and a control of diabetes patients without Charcot. The bone mineral density of the Charcot group was significantly lower in the calcaneus compared to the control group, and there was a strong trend that the Charcot group bone quality was lower than the diabetic control group ($p = 0.08$). Interestingly the core bone mineral density as measured in the lumbar spine was not significantly different between the three groups.

It is important to recognize that tools that measure BMD are quantitative in nature and do not measure the qualitative aspect of bone. Patients with diabetes are at risk for fragility fractures due to decreased bone material strength even in the setting of normal BMD [37]. This increased risk of fracture is secondary to greater cortical porosity, smaller cortical area, and decreased bone strength. The implication in Charcot patients is the obvious potential for stress fractures even without observed trauma. Given the alteration in bone remodeling and turnover seen in diabetic bone, healing after arthrodesis could be impacted as well due to decreased bone quality and reduced biomechanical properties.

It has also been demonstrated that inflammatory and bone turnover markers and acute Charcot neuroarthropathy are elevated in peripheral serum [38]. Inflammatory markers such as C-reactive protein, TNF alpha, and interleukin-6 were found to be significantly higher in patients with Charcot neuroarthropathy when compared to diabetic patients without Charcot neuroarthropathy. Markers of bone turnover such as C-terminal telopeptide, bone alkaline phosphatase, and osteoprotegerin were also significantly elevated at presentation. TNF alpha and interleukin-6 declined significantly after 3 months of casting but did not change during the resolution phase. Markers of bone turnover did not decline significantly after 3 months of casting or at final resolution. Surgeons should recognize that the potential for ongoing bone remodeling can occur regardless of the timing of surgical intervention, even after resolution of Eichenholtz Stage 1.

## Indications for Surgery

Traditionally the indications for surgical intervention include:

- Non-braceable deformities
- Instability
- Impending ulceration of the skin
- Non-healing ulcers
- Recurrent ulcers
- Osteomyelitis of the midfoot, hindfoot, and ankle
- Pain

Although symptomatic pain is relatively uncommon, a subset of patients with Charcot neuroarthropathy will complain of significant pain due to instability and deformity. Some patients complain of difficulty ambulating with a rocker bottom deformity. Patients whose foot is non-plantigrade are at high risk of ulceration. For the purposes of this chapter, a non-plantigrade foot/ankle is defined as one in which the patient is bearing weight on skin that is not meant to bear weight. For example, the plantar arch, lateral and medial borders of the foot dorsal to glabrous skin and skin over the medial and lateral malleoli are not designed to bear weight. Midfoot CN commonly results in collapse of the arch with potential skin compromise medially or laterally on the plantar surface. Collapse of the medial column can involve subluxation/dislocation of the talonavicular, naviculocuneiform, or tarsometatarsal joints. Laterally, the prominence is typically a result of subluxation or dislocation of the calcaneo-cuboid joint. Hindfoot and ankle deformities can result in significant varus or valgus malalignment that jeopardizes non-plantar skin (see figure). Final surgical planning and goals of surgery are best discussed by the multidisciplinary team, taking into consideration the factors that have been previously reviewed. While each Charcot case is unique, reconstruction should follow a logical and reproducible plan. We recognize that customization of the approach may be necessary, but in general the following principles guide reconstruction.

1. Perioperative medical optimization measures. A high portion of patients are on anticoagulation treatment and it is often not safe to stop this medication preoperatively.
2. The choice of anesthesia: Peripheral nerve block for pain relief is not considered in most patients, due to the degree of sensory neuropathy. Tranexamic acid administration during induction is usually considered in most patients.
3. Decision on the usage of tourniquet: This is based on the vascular status and previous revascularization procedures. It should be recognized that many patients have medial artery calcinosis that can prevent occlusion with a tourniquet, resulting in a venous tourniquet.
4. Prophylactic antibiotics administration: This is delayed until the intraoperative bone and deep tissue samples are harvested.
5. Surgical approaches: The location of the surgical approaches and their effect on the vascular supply to the soft tissue envelope and bones is discussed. It is preferred to perform the reconstruction using one main surgical approach, supplemented with additional small approaches as required. Foot and ankle surgeons must be comfortable with a 360-degree approach to the pathology, as many patients have compromised skin from previous surgery.
6. Soft tissue releases: The associated soft tissue contractures are assessed, and plans are made for lengthening or release for deformity correction. Commonly performed soft tissue lengthenings in Charcot surgery include Achilles (sagittal plane), posterior tibial (varus deformities), and peroneal (valgus deformities).
7. Bone corrections: The location of the bone osteotomies and the size of the bone wedge/rhomboid resections are discussed based on the assessment of clinical deformity (shape and flexibility) and imaging studies. CT imaging with 3D reconstruction, weight-bearing CT, and 3D printed model of the bone deformity of the foot are useful tools used for this assessment.
8. The fixation devices: Due to the presence of significant bone loss, Charcot foot reconstruction procedures often require a combination of fixation devices to achieve a long-segment and rigid fixation construct. Hindfoot nail fixation may require additional cannulated screw fixation across the hindfoot, and midfoot beams may require supplementary locking plate fixation, to enhance the rotational rigidity to the construct.
9. Wound closure: On occasions, it may not be possible to achieve tension-free primary wound closure, particularly when the degree of the deformity is severe or if concomitant ulcer debridement was done. The need for performing a local rotational flap or other appropriate plastic surgical procedure or usage of NWPT is anticipated. Soft tissue complications are common, and one method of minimizing these complications is to approach the deformity from the convex side. Once the deformity is corrected, the convex side is no longer under tension while the concave side is subjected to tension.
10. Antibiotic regimen: In the presence of an ulcer or previous history of infection, a biodegradable, osseoconductive and local antibiotic eluting calcium sulfate preparation can be used to fill the bone voids and achieve high concentrations of the antibiotic. The postoperative antibiotic regimen can be based on the preoperative bone biopsy microbiology sensitivities and modified according to the sensitivities of intraoperative specimens.
11. Mobility: Postoperative weight-bearing status and the duration is determined based on the complexity of the reconstruction procedure. Consideration is given on the status of the opposite foot, as excessive load bearing carries a risk of activation or re-activation of Charcot changes in this foot.

## Exostectomy

Several retrospective case series have described medial or lateral exostectomy to decompress bony deformities of CN and promote healing of recalcitrant ulcers [39–44]. Advocates of exostectomy cite high healing rates of ulcers; however, Catanzariti et al. [40] reported higher success with medial column exostectomy versus lateral column exostectomy. Molines-Barroso et al. [42] found that sagittal plane radiographic measures worsened after lateral column exostectomy as manifested by a significantly decreased calcaneal inclination angle and significantly increased talar declination angle.

Plantar ulcers due to bone prominence that do not respond to surgical debridement can be considered for exostectomy. The infected ulcer is excised, removing all infected and necrotic tissue, down to the bone prominence. Any associated tendon contractures, particularly of the Achilles tendon is released or lengthened. The area of bone resection is identified by careful palpation and under the guidance of fluoroscopy. All bone prominence is excised completely using an oscillating saw or a sharp osteotome. If there are any areas of residual bone necrosis or bone changes consistent with osteomyelitis, the resection is continued until all these areas are removed. Care is taken not to leave any bone projections or loose bone fragments as this will interfere with ulcer healing. Where available, antibiotic loaded calcium sulfate preparation can be inserted into drill hole channels created in the exposed bone as an injectable form or applied on the bone surface as beads (see figure). Local elution of high concentrations of antibiotic can potentially eradicate any residual infection. The foot is examined after completion exostectomy for the presence of instability due to bone resection. This requires a temporary stabilization of this area with threaded wires or an external fixator for the duration of bone healing. The open wound is managed either with a local rotation flap, free flap, or negative pressure wound therapy and appropriate offloading.

## Reconstruction of Active Charcot Foot

Acute (active) CN of foot normally responds to immediate offloading in a total contact cast (TCC) or a well-fitting brace until it reaches an inactive phase. Despite adequate offloading some Charcot deformities continue to progress secondary to the degree of bone fragmentation or joint dislocations. Progressive deformities make the foot and ankle vulnerable to friction and shear forces which can lead to ulceration. If the foot is at risk of ulceration, and consequently infection due to the presence of marked deformity and or instability, it is advisable to perform surgical reconstruction in the active phase of the disease. Although ideally performed after resolution of foot swelling and normalization of local warmth, the degree and location of deformity may accelerate the surgical plan despite the presence of active inflammation. The use of a preoperative compression dressing incorporating cast padding in conjunction with elevation can result in substantial reduction in edema. The reconstruction is performed using internal or external fixation, using the principles described later in this chapter.

## Reconstruction of Inactive Charcot foot

Severe Charcot deformity carries a high risk of developing ulceration even with adequate offloading. Associated instability, often noticed in severely affected feet due to non-union of bone fragments effected by the Charcot process, provides additional challenge in preventing a skin breakdown. Ulceration often progresses to developing infection and a chain of events resulting in a major amputation. An ulcerated Charcot foot is 12 times more vulnerable to undergo a major lower limb amputation [45]. Due to the high rate of mortality following a major lower limb amputation in the diabetic population, a functional limb salvage of Charcot foot can potentially save the life of some of these patients.

The aim of Charcot foot reconstruction is to achieve a plantigrade and stable foot that is infection and ulcer free and allows full weight-bearing in a modified shoe or a brace. The incidence of non-union following Charcot foot reconstructions is high, and in general a stable non-union or pseudoarthrosis has been considered as an acceptable outcome. Some fibrous non-unions achieve adequate stability and are still desirable; however, mobile and unstable non-unions should be avoided in a neuropathic foot, as this can result in recurrence of deformity over a period of time. Ideally, the surgical aim should be to achieve a full bone fusion or a stable fibrous union in every procedure.

## Deformity Correction

Charcot foot and ankle deformity correction is achieved by achieving adequate soft tissue balance, through release or lengthening of contracted tendons and other soft tissues, and performing wedge or rhomboid bone resections on the convexity of the deformity, based on surgical planning. The choice of incision and surgical approach depends on the location of the deformity and the vascular status of the angiosomes. Multiple major surgical incisions should be avoided as they carry a significant risk of wound breakdown and infection. Careful deep dissection of the incisions developing thick and deep soft tissue flaps and protecting vascular structures is critical. All joints intended for bone fusion are exposed and thoroughly prepared. Following the desired deformity correction, stabilization of the correction is achieved by using either internal or external fixation methods. Recent systematic review studies revealed no significant advantage of one method over the other (see figure) [46]. With the recent introduction of Charcot-specific internal fixation devices, there has been a recent increase in the usage of this internal fixation method for reconstructions.

## Hindfoot and Midfoot Stabilization Using Internal Fixation

The surgical principles for Charcot foot and ankle internal fixation method have evolved since Sammarco et al. [47] described a decade ago, when the term "Super-construct" was introduced for this fixation. This advocated extension of bone fixation beyond the zone of injury, the usage of the strongest fixation device that is tolerated by the soft tissue envelope and application of the fixation devices in a novel position that maximizes its mechanical function. Subsequent studies identified the additional need for the fixation construct to provide rigidity against axial, bending, and rotational forces to improve the fixation outcomes. The current established principle of internal fixation for Charcot reconstruction is "durable long-segment rigid fixation with optimal bone opposition."

## Hindfoot Internal Fixation

An intramedullary hindfoot nail (IMHN) is the most accepted method of internal fixation for Charcot hindfoot reconstruction. Biomechanically, IMHN is a load sharing device, provides better mechanical environment, and has higher bending and torsional stiffness compared to other forms of internal fixation. It can also provide intraoperative compression of the bone fragments over the nail and that results in optimal bone opposition. The torsional rigidity of the construct may be suboptimal in the presence of marked bone loss and may require additional fixation (see figure) [18].

A trans-fibular lateral approach provides good access to the ankle and subtalar joints for preparation and wedge resections and is the most common surgical approach. Alternative approaches may be considered for severe valgus deformities or those with compromised lateral soft tissues. Following the soft tissue releases and bone resections, the hindfoot is stabilized temporarily with 2 mm Kirshner wires, to maintain correction. The entry point for the hindfoot nail is determined under fluoroscopy guidance and care is taken to make sure that guide wire goes through the mid-portion of calcaneus. The intramedullary reaming to adequate diameter and depth is performed. The Kirshner wires are then removed and the chosen length and diameter hindfoot nail is inserted, using the recommended standard surgical technique. Correct length and diameter of the nail should be chosen to achieve a good isthmal fit of the nail is the tibial diaphysis. There are varying opinions on whether to use short or long retrograde nails, and IMHN sizes range from 150 to 300 mm in length. Axial compression of the bone fragments over the nail is attained before inserting both proximal and distal locking screws. In the presence of significant bone loss or during severe hindfoot correction that utilizes large bone resections, optimal rotational rigidity cannot be achieved with a standard hindfoot nail construct alone. To enhance the rotational rigidity in such constructs, an additional cannulated screw can be inserted from calcaneum into distal tibia (see figure). Supplemental fixation can also be achieved with a locking plate spanning distal tibial and talus.

## Midfoot Internal Fixation

Charcot midfoot deformities generally fall into one of three patterns:

1. rocker bottom forefoot abduction
2. dorsal subluxation/dislocation
3. forefoot adduction (see figure).

The rocker bottom forefoot abduction deformity is the commonest pattern and results from the involvement of the medical column collapse. Significant deformity often results in marked reduction of calcaneal pitch and contracture of Achilles tendon. Sagittal plane deformity can be quite significant.

Midfoot deformity correction often requires posterior muscle group lengthening to achieve soft tissue balance in the sagittal plane. This can be accomplished with required percutaneous tendo Achilles lengthening, open Achilles tendon lengthening, or gastrocnemius recession. This is performed in conjunction with anatomic restoration by performing bone wedge resections on the convex side. Most deformities are associated with rocker bottom and forefoot abduction components. A medial midfoot approach allows performing a plantar and medial based bone wedge resection, with the apex of the wedge placed in the lateral part of the cuboid bone, thereby preserving the cuboid's lateral cortex. This intact lateral cortex of the cuboid allows controlled correction of the forefoot deformity by closing the wedge and permits the application of tension band plating principle for the medial column fixation (see figure). The deformity correction can be provisionally held with two or more 2 mm Kirschner wires.

The deformity correction can be stabilized with a medial column beam or locking plate or a combination of these. For locking plate fixation technique, initial lag screw fixation with one or two cannulated lag screws across the osteotomy is done before using a strong and low profile contoured locking plate spanning across the medial column for neutralization. More recently an intramedullary medial column beam spanning the first metatarsal and talus, inserted either retrograde through the metatarsal head or antegrade through the posterior body of talus, is favored as it provides excellent compressive fixation and requires smaller surgical approach. If any residual rotational instability is noted, this can be enhanced by supplementing the fixation with a locking plate across the medial column (see figure). Most midfoot deformities, involving the medial column, do not require a lateral column fixation, if the lateral cortex of cuboid is left intact. However, for complex deformities, as noted in some dorsal subluxation patterns and those that involve medial and lateral column rocker bottom deformity, additional lateral column fixation is required. This can be achieved by using additional lateral beams inserted from the third and fourth metatarsal into calcaneus, along with an additional plate fixation spanning the base of fourth metatarsal to the anterior part of calcaneum, if required.

## Two-Stage Reconstruction

Charcot foot reconstruction is typically carried out as a one-stage procedure, even in the presence of a non-infected ulcer. In the presence of an ulcer, thorough surgical debridement is performed at the beginning of the procedure, followed by the reconstruction using the principles enumerated above. Multiple bone and soft tissue specimens are collected during the procedure for microbiological analysis and empirical antibiotic therapy is commenced until the microbiological sensitivity results are obtained. The wound from ulcer debridement is managed with primary closure, a local rotation flap, or NPWT. In some cases, the ulcer wound can be left open and healing occurs rapidly once the osseous deformity has been corrected.

Charcot foot deformity associated with an infected ulcer or deep infection is best managed as a two-stage procedure (to achieve functional limb salvage) [48]. The first stage of this treatment consists of surgical debridement of all infected and necrotic tissues using the principles described above. It is critical that multiple deep tissue and bone specimens from the infected areas are harvested for microbiological culture and sensitivities. Infected bone and prominences are thoroughly excised. In the presence of marked deformity, osteotomy or wedge resection is done to reduce deformity and decompress the soft tissues. The bone voids that are created from debridement and osteotomy are filled with an antibiotic impregnated calcium sulfate preparation, for local antibiotic elution in high concentration to eliminate any residual infection. The choice of the antibiotic used in this preparation is based on the previous microbiological sensitivities. If an osteotomy is done, the associated foot instability is addressed with the application of threaded guidewires or an external fixator temporarily. The open wounds created from ulcer debridement or surgical wounds are managed with negative pressure wound therapy (NPWT).

Infection eradication is achieved by administering empirical intravenous antibiotics that are changed to targeted antimicrobials once the intraoperative specimen microbiology results become available. The duration of antibiotic administration is based on the improvement noted clinically and serologically. After a period of 6–10 weeks of interval treatment that includes advanced wound care and foot offloading, the second stage of treatment is delivered.

The second stage of the reconstruction is typically done using the external fixation option; however, recent reports have shown good results with internal fixation methods using the principles described above (see figure). Repeat debridement of the previously infected areas and further harvesting of deep tissue and bone samples are done during the second stage, followed by soft tissue releases if required, wedge bone resections, joint preparations, and internal fixation using the principle of "long segment and rigid internal fixation with optimal bone opposition," as described above. Gentamycin or Vancomycin impregnated injectable calcium sulfate preparation (Cerament® G or V, Bonesupport, Lund, Sweden) is applied to the bone voids and around the osteotomy sites for local antibiotic elution. Targeted intravenous antibiotics are continued for 2–6 weeks based on the improvements noted clinically and serologically. The postoperative care is similar to the one-stage reconstruction.

## Postoperative Care of Reconstructed Charcot Foot

The leg is elevated postoperatively to reduce swelling and the patient mobilized non-weight-bearing in a well-padded below-knee splint. Closed surveillance of the surgical wound is undertaken, and once the wound is stabilized, a total contact cast is applied. Bivalving of the TCC is especially helpful to facilitate regular wound inspections. The patient is discharged home, when safe mobility levels are reached, non-weight-bearing in a TCC. Postoperative radiographs are taken at 6 and 12 weeks and regularly then after as required. The non-weight-bearing TCC is continued for at least 3 months post-surgery. Progression to partial weight-bearing in the cast can be initiated once radiographs demonstrate signs of osseous healing. Ultimately, progression to custom-made orthotics and/or shoes is fabricated to assist in independent ambulation. Some patients may benefit from additional stability by using a cane to help mitigate the consequences of peripheral neuropathy.

## References


1. Johnson JT. Neuropathic fractures and joint injuries. Pathogenesis and rationale of prevention and treatment. J Bone Joint Surg Am. 1967;49:1.
2. Mueller ME, Allgower M, Schneider R, Willenegger H. Manual of internal fixation: techniques recommended by the AO-ASIF Group. Berlin: Springer; 1991.
3. Kirienko A, Villa A, Calhoun JH. Ilizarov technique for complex foot and ankle deformities. New York, NY: Marcel Dekker, Inc; 2004.
4. Kuntscher G. The marrow nailing method. Kiel: Stryker Trauma GmbH; 1947, translated 2006.
5. Pinzur MS, Kelikian A. Charcot ankle fusion with a retrograde locked intramedullary nail. Foot Ankle Int. 1997;18:699.
6. Stuart MJ, Morrey BF. Arthrodesis of the diabetic neuropathic ankle joint. Clin Orthop Relat Res. 1990;(253):209.
7. Wukich DK, Raspovic KM, Suder NC. Prevalence of peripheral arterial disease in patients with diabetic Charcot neuroarthropathy. J Foot Ankle Surg. 2016;55:727.
8. Myers TG, Lowery NJ, Frykberg RG, Wukich DK. Ankle and hindfoot fusions: comparison of outcomes in patients with and without diabetes. Foot Ankle Int. 2012;33:20.
9. Wukich DK, Crim BE, Frykberg RG, Rosario BL. Neuropathy and poorly controlled diabetes increase the rate of surgical site infection after foot and ankle surgery. J Bone Joint Surg Am. 2014;96:832.
10. Wukich DK, Shen JY, Ramirez CP, Irrgang JJ. Retrograde ankle arthrodesis using an intramedullary nail: a comparison of patients with and without diabetes mellitus. J Foot Ankle Surg. 2011;50:299.
11. Thompson RC Jr, Clohisy DR. Deformity following fracture in diabetic neuropathic osteoarthropathy. Operative management of adults who have type-I diabetes. J Bone Joint Surg Am. 1993;75:1765.
12. Lowery NJ, Woods JB, Armstrong DG, Wukich DK. Surgical management of Charcot neuroarthropathy of the foot and ankle: a systematic review. Foot Ankle Int. 2012;33:113.
13. Schneekloth BJ, Lowery NJ, Wukich DK. Charcot neuroarthropathy in patients with diabetes: an updated systematic review of surgical management. J Foot Ankle Surg. 2016;55:586.
14. Shazadeh Safavi P, Jupiter DC, Panchbhavi V. A systematic review of current surgical interventions for Charcot neuroarthropathy of the midfoot. J Foot Ankle Surg. 2017;56:1249.
15. Cates NK, Wagler EC, Bunka TJ, Elmarsafi T, Tefera E, Kim PJ, Liu GT, Evans KK, Steinberg JS, Attinger CE. Charcot reconstruction: outcomes in patients with and without diabetes. J Foot Ankle Surg. 2020;59:1229.
16. Manu CA, Mustafa OG, Bates M, Vivian G, Mulholland N, Elias D, Huang DY, Deane C, Cavale N, Kavarthapu V, Rashid H, Edmonds M. Transformation of the multidisciplinary diabetic foot clinic into a multidisciplinary diabetic foot day unit: results from a service evaluation. Int J Low Extrem Wounds. 2014;13:173.
17. Butt DA, Hester T, Bilal A, Edmonds M, Kavarthapu V. The medial column Synthes Midfoot Fusion Bolt is associated with unacceptable rates of failure in corrective fusion for Charcot deformity: Results from a consecutive case series. Bone Joint J. 2015;97-B:809.
18. Siebachmeyer M, Boddu K, Bilal A, Hester TW, Hardwick T, Fox TP, Edmonds M, Kavarthapu V. Outcome of one-stage correction of deformities of the ankle and hindfoot and fusion in Charcot neuroarthropathy using a retrograde intramedullary hindfoot arthrodesis nail. Bone Joint J. 2015;97-B:76.
19. Vasukutty N, Jawalkar H, Anugraha A, Chekuri R, Ahluwalia R, Kavarthapu V. Correction of ankle and hind foot deformity in Charcot neuroarthropathy using a retrograde hind foot nail-The Kings' Experience. Foot Ankle Surg. 2018;24:406.
20. Pinzur M. Surgical versus accommodative treatment for Charcot arthropathy of the midfoot. Foot Ankle Int. 2004;25:545.
21. Rogers LC, Frykberg RG, Armstrong DG, Boulton AJ, Edmonds M, Van GH, Hartemann A, Game F, Jeffcoate W, Jirkovska A, Jude E, Morbach S, Morrison WB, Pinzur M, Pitocco D, Sanders L, Wukich DK, Uccioli L. The Charcot foot in diabetes. Diabetes Care. 2011;34:2123.
22. Simon SR, Tejwani SG, Wilson DL, Santner TJ, Denniston NL. Arthrodesis as an early alternative to nonoperative management of Charcot arthropathy of the diabetic foot. J Bone Joint Surg Am. 2000;82-A:939.
23. Mittlmeier T, Klaue K, Haar P, Beck M. Should one consider primary surgical reconstruction in Charcot arthropathy of the feet? Clin Orthop Relat Res. 2010;468:1002.
24. Eschler A, Gradl G, Wussow A, Mittlmeier T. Late corrective arthrodesis in nonplantigrade diabetic Charcot midfoot disease is associated with high complication and reoperation rates. J Diabetes Res. 2015;2015:246792.
25. Wukich DK, Belczyk RJ, Burns PR, Frykberg RG. Complications encountered with circular ring fixation in persons with diabetes mellitus. Foot Ankle Int. 2008;29:994.
26. Dayton P, Feilmeier M, Thompson M, Whitehouse P, Reimer RA. Comparison of complications for internal and external fixation for Charcot reconstruction: a systematic review. J Foot Ankle Surg. 2015;54:1072.
27. Bevan WP, Tomlinson MP. Radiographic measures as a predictor of ulcer formation in diabetic Charcot midfoot. Foot Ankle Int. 2008;29:568.
28. Wukich DK, Raspovic KM, Hobizal KB, Rosario B. Radiographic analysis of diabetic midfoot Charcot neuroarthropathy with and without midfoot ulceration. Foot Ankle Int. 2014;35:1108.

29. Meyr AJ, Sebag JA. Relationship of cuboid height to plantar ulceration and other radiographic parameters in midfoot Charcot neuroarthropathy. J Foot Ankle Surg. 2017;56:748.
30. Sheth S, Derner BS, Meyr AJ. Reliability of the measurement of cuboid height in midfoot Charcot neuroarthropathy. J Foot Ankle Surg. 2018;57:759.
31. Lauri C, Glaudemans A, Signore A. Leukocyte imaging of the diabetic foot. Curr Pharm Des. 2018;24:1270.
32. Duceppe E, Parlow J, MacDonald P, Lyons K, McMullen M, Srinathan S, Graham M, Tandon V, Styles K, Bessissow A, Sessler DI, Bryson G, Devereaux PJ. Canadian cardiovascular society guidelines on perioperative cardiac risk assessment and management for patients who undergo noncardiac surgery. Can J Cardiol. 2017;33:17.
33. Baumhauer JF, O'Keefe RJ, Schon LC, Pinzur MS. Cytokine-induced osteoclastic bone resorption in Charcot arthropathy: an immunohistochemical study. Foot Ankle Int. 2006;27:797.
34. Herbst SA, Jones KB, Saltzman CL. Pattern of diabetic neuropathic arthropathy associated with the peripheral bone mineral density. J Bone Joint Surg (Br). 2004;86:378.
35. Petrova NL, Edmonds ME. A prospective study of calcaneal bone mineral density in acute Charcot osteoarthropathy. Diabetes Care. 2010;33:2254.
36. Greenhagen RM, Wukich DK, Jung RH, Vardaxis V, Yoho RM. Peripheral and central bone mineral density in Charcot's neuroarthropathy compared in diabetic and nondiabetic populations. J Am Podiatr Med Assoc. 2012;102:213.
37. Lecka-Czernik B. Diabetes, bone and glucose-lowering agents: basic biology. Diabetologia. 2017;60:1163.
38. Petrova NL, Dew TK, Musto RL, Sherwood RA, Bates M, Moniz CF, Edmonds ME. Inflammatory and bone turnover markers in a cross-sectional and prospective study of acute Charcot osteoarthropathy. Diabet Med. 2015;32:267.
39. Brodsky JW, Rouse AM. Exostectomy for symptomatic bony prominences in diabetic Charcot feet. Clin Orthop Relat Res. 1993;(296):21.
40. Catanzariti AR, Mendicino R, Haverstock B. Ostectomy for diabetic neuroarthropathy involving the midfoot. J Foot Ankle Surg. 2000;39:291.
41. Laurinaviciene R, Kirketerp-Moeller K, Holstein PE. Exostectomy for chronic midfoot plantar ulcer in Charcot deformity. J Wound Care. 2008;17:53.
42. Molines-Barroso RJ, Lazaro-Martinez JL, Beneit-Montesinos JV, Alvaro-Afonso FJ, Garcia-Morales E, Garcia-Alvarez Y. Early foot structural changes after lateral column exostectomy in patients with Charcot foot. Int J Low Extrem Wounds. 2019;18:129.
43. Rosenblum BI, Giurini JM, Miller LB, Chrzan JS, Habershaw GM. Neuropathic ulcerations plantar to the lateral column in patients with Charcot foot deformity: a flexible approach to limb salvage. J Foot Ankle Surg. 1997;36:360.
44. Sato T, Ichioka S. Ostectomy and medial plantar artery flap reconstruction for Charcot foot ulceration involving the midfoot. J Foot Ankle Surg. 2016;55:628.
45. Sohn MW, Stuck RM, Pinzur M, Lee TA, Budiman-Mak E. Lower-extremity amputation risk after Charcot arthropathy and diabetic foot ulcer. Diabetes Care. 2010;33:98.
46. Ha J, Hester T, Foley R, Reichert ILH, Vas PRJ, Ahluwalia R, Kavarthapu V. Charcot foot reconstruction outcomes: A systematic review. J Clin Orthop Trauma. 2020;11:357.
47. Sammarco VJ, Sammarco GJ, Walker EW Jr, Guiao RP. Midtarsal arthrodesis in the treatment of Charcot midfoot arthropathy. Surgical technique. J Bone Joint Surg Am. 2010;92(Suppl 1 Pt 1):1.
48. Wukich DK, Sadoskas D, Vaudreuil NJ, Fourman M. Comparison of diabetic Charcot patients with and without foot wounds. Foot Ankle Int. 2017;38:140.