

Charcot Neuroarthropathy of the Foot and Ankle

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INTRODUCTION

Jean Martin Charcot, in 1883, first described Charcot neuroarthropathy as occurring in patients with tabes dorsalis. Charcot neuroarthropathy was not known to be associated with diabetes until William Reily Jordan noted the connection in 1936 (1). Although two-thirds of patients with Charcot neuroarthropathy have type 2 diabetes, many other causes have been reported [e.g., syphilis (i.e., tabes dorsalis), leprosy, alcoholism, peripheral nerve injuries, spina bifida, folate deficiency (e.g., pernicious anemia), and spinal cord lesions (e.g., syringomyelia, post-traumatic cord injury, meningomyelocele, and disk herniation)] (2).

Charcot neuroarthropathy is a peripheral and autonomic neuropathy that typically presents as a hyperemic event (e.g., a red, swollen foot). The diagnosis of Charcot neuroarthropathy is made clinically after observing a lack of normal foot sensation, presence of ulcerations, and presence of foot deformity because of joint subluxations/dislocations. Many factors contribute to the destruction of the bone and joint in patients with Charcot neuroarthropathy. These factors include loss of protective sensation (peripheral neuropathy), autonomic neuropathy, weight-bearing stress (osseous malalignment/soft-tissue imbalance), trauma (repetitive or incidental), metabolic abnormalities, renal disease/transplant, osteoporosis, and glycosylation of bone proteins and collagen (2,3).

The mechanism(s) for the development of Charcot neuroarthropathy is not completely understood. However, two theories that address the causes of Charcot neuroarthropathy have been described: a neurovascular theory and a neurotraumatic theory (2,3). Both mechanisms likely contribute to Charcot neuroarthropathy. The neurovascular theory is based on increased blood flow to the limb from dilation of the blood vessels because of sympathetic denervation. The loss of vasomotor control allows blood vessels to dilate, thus increasing the peripheral blood flow. The increased peripheral blood flow increases the arteriovenous shunting, which causes hyperemia and bone resorption. Contrary to common misconceptions, most patients (90%) with Charcot neuroarthropathy of the foot have excellent blood supply to the foot (4).

The neurotraumatic theory is based on the patients experiencing an overuse injury because of an absence of protective sensation. Either acute trauma or repetitive trauma can initiate Charcot neuroarthropathy. Absence of protective sensation limits the body's protective mechanisms such as shifting body weight, limiting activity, and muscle guarding (2,3).

The best treatment results for Charcot neuroarthropathy of the foot and ankle are achieved when treatment is initiated during the early stages of the disease. The typical treatment for acute Charcot neuroarthropathy attempts to stabilize the condition by placing the foot in a total contact cast and immobilizing it. Non-weight bearing in a total contact cast can produce osteopenia of the ipsilateral foot and an increased load on the contralateral foot, which can lead to ulceration and Charcot neuroarthropathy in the contralateral foot. Maintaining non-weight-bearing status is difficult for this patient population for various reasons (e.g., obesity, diminished proprioception, and muscle atrophy). A new method for treating acute Charcot neuroarthropathy is to apply a static external fixator that acts like a cast by immobilizing the affected joints and bones (5).

After the Charcot foot is stabilized, the bones and joints halt disintegration and subluxation and coalesce to become fixed in a deformed position. Abnormal osseous prominences are potential areas for ulceration. During gait, the position of the patient's deformed foot and the resultant altered muscle-tendon balance produce aberrant weight-bearing forces that increase the risk for ulceration. When ulcers occur, osteomyelitis should be ruled out as a diagnosis and, if present, removed. If osteomyelitis is concurrent with Charcot neuroarthropathy, the patient has a poor prognosis. Therefore, early recognition and appropriate treatment is paramount to avoid osteomyelitis.

The typical surgical management for Charcot neuroarthropathy includes procedures such as Achilles tendon lengthening, ostectomy, debridement, osteotomy, arthrodesis, and open reduction with internal fixation. Open reduction with plantar plating or rigid internal fixation and an Achilles tendon lengthening are methods that are frequently used for reconstruction (6). Recently, static external fixation has been used for reconstruction of Charcot deformities, but gradual correction with the use of an external fixator is a new treatment option (2,3).

CLINICAL EVALUATION

Charcot deformities of the foot and ankle are vast. Acute, chronic, or acute-on-chronic Charcot neuroarthropathy can be observed at multiple anatomic locations with varying degrees of severity. Typically, the Charcot foot is wider and larger in size than a normal foot and, if untreated, will become even more deformed. The clinical presentation varies depending on the anatomic location and the stage of the disease. Acute Charcot neuroarthropathy might seem to be an infection because it presents as a red, hot, swollen foot (stage 1). Acute Charcot neuroarthropathy can present with or without foot and ankle deformities. A chronic Charcot deformity presents as a severe foot or ankle deformity, typically with ulceration (stages 2 and 3).

Ulcerations, infections, and osteomyelitis commonly are associated with Charcot neuroarthropathy and generally occur as related sequelae. Plantar ulcers commonly occur in patients with Charcot neuroarthropathy of the foot, and the location of the ulcer usually correlates to the anatomic location of the Charcot neuroarthropathy. For example, medial column ulcers of the foot are generally associated with tarsometatarsal Charcot neuroarthropathy that results in a medial column collapse. Tarsometatarsal Charcot deformities typically become stable (by coalescing) and are successfully treated conservatively or with a limited surgical approach (ostectomy or wedge resection with stabilization) (7). However, lateral column ulcers are associated with a more proximal Charcot deformity of the midfoot that typically does not fully coalesce. Instability of the lateral column leads to recurrent ulcers. In such cases, conservative treatment generally is futile and surgical reconstruction often becomes necessary.

Chronic ulcerations eventually lead to deep infection and osteomyelitis. Patients with osteomyelitis, as diagnosed in a clinical setting by probing to bone through the ulcer, undergo surgical debridement to remove all infection. In cases of osteomyelitis, the infected bone should be resected before reconstruction or the infection can be addressed concurrently with reconstruction by using external fixation.

In addition to noting the stage of Charcot neuroarthropathy, the surgeon should thoroughly assess the ulcerations, osteomyelitis, and anatomic location of the deformity. Radiographic assessment aids in determining the stage of the disease and the location of the deformity. Radiographs of the foot and ankle in cases of Charcot neuroarthropathy can be difficult to decipher; the bones of the hindfoot and midfoot are superimposed because of the subluxation/dislocation of these joints. Bone fragmentation and proliferation of new bone during the early and late stages of Charcot neuroarthropathy, respectively, add to the complexity of radiographic interpretation. Radiographs that show the patient during weight bearing should be obtained in all planes to more easily locate the Charcot deformity. Axial view radiographs can be helpful to evaluate Charcot neuroarthropathy of the midfoot, hindfoot, and ankle. Combined Charcot joint deformities are not uncommon. Using normal foot and ankle radiographic reference points and angles provides a quantitative assessment of the degree of deformity present (8). Major ankle, hindfoot, and forefoot axes, angles, and reference points should be used because of the osseous obscurity of the region affected by Charcot neuroarthropathy. Thus, forefoot-to-midfoot or midfoot-to-hindfoot relationships can be assessed and compared with normal relationships that aids in surgical planning.

CLASSIFICATION

Overall, Charcot neuroarthropathy is characterized by identifying the stage and the anatomic location of the deformity.

Stages of Charcot Neuroarthropathy

Eichenholtz (9) defined the stages of Charcot neuroarthropathy based on clinical examination and radiographic findings.

- Stage 1—Development phase, characterized by fragmentation of bones and cartilage, joint effusions, subluxation and dislocation, soft-tissue edema, hyperemia, bone resorption, and intra-articular fractures.
- Stage 2—Coalescence phase, characterized by decreased soft-tissue edema, healing of fractures, and organization of bone fragments.
- Stage 3—Reconstruction phase, characterized by new bone formation and remodeling of bone.

Shibata et al. (10) added another stage:

- Stage 0—Acute phase, characterized by swelling, warmth, joint instability, and normal radiographic anatomy of the foot and ankle.

Location of Charcot Deformity

Authors have described the patterns of Charcot neuroarthropathy based on the anatomic locations of the affected joints in the foot and ankle (2,11,12). The six regions affected by Charcot neuroarthropathy are the metatarsophalangeal, tarsometatarsal, midfoot, ankle, subtalar, and calcaneal regions. Each anatomic location of Charcot deformity has a particular pattern of osseous and soft-tissue deformity.

A patient with Charcot neuroarthropathy with or without ulceration who experiences a collapse that involves the lateral column has a poor prognosis for treatment (12). Various patterns of Charcot neuroarthropathy of the midfoot cause a lateral column deformity. Catanzariti et al. (13) found that osteotomy is successful in healing ulcers of the medial column but not of the lateral column. A collapsed lateral column creates an unstable foot, thereby producing aberrant and propulsive gait. During gait, the ground reaction force vector ends just proximal to the calcaneocuboid joint and produces a constant deforming force on an already unstable lateral column (14). This pedal instability creates significant treatment difficulties. Catanzariti et al. (13) summarized by recommending that patients with lateral column Charcot deformity require a more complex surgical reconstruction.

TREATMENT OPTIONS

Conservative Treatment Options

Local wound care, total contact casting, custom-molded shoes with inserts, and specialized braces are commonly used to obtain or maintain a closed tissue envelope in patients with Charcot neuroarthropathy of the foot and ankle. The goals of these treatments are to off-load the ulcer and to support the foot and ankle. These conservative treatments can prevent or delay surgical intervention. However, surgical intervention is indicated when off-loading and supportive shoe gear is unable to stabilize the foot and ankle, or when recurrent ulceration is present. Even after surgical intervention, these conservative treatments are implemented.

Surgical Treatment Options

The goals of surgical intervention for the Charcot foot and ankle are to restore alignment and stability, prevent amputation, prepare for a shoe or brace, and allow the patient to be ambulatory. Achilles tendon lengthening, osteotomy, debridement, osteotomy, arthrodesis, open reduction with internal or external fixation, and gradual correction with external fixation

are surgical procedures that should be considered when reconstructing the Charcot foot and ankle. The surgical procedure selection depends on the stage of the disease, the anatomic location of the deformity, the presence or absence of osteomyelitis, the stability of the Charcot deformity, and multiple other factors.

Equinus deformity imparts a major deforming force on the foot and is almost always concurrent with Charcot foot deformities. Surgical strategies to correct equinus deformity of the ankle are based on clinical evaluation. The Silfverskiöld test is the gold standard for determining whether a patient has gastrocnemius equinus deformity or gastrocnemius-soleus equinus deformity. With most Charcot deformities, a severe gastrocnemius-soleus equinus contracture is present; therefore, maximum lengthening is necessary. Percutaneous Achilles tendon Z-lengthening is preferred. However, when possible, gastrocnemius-soleus recession is performed to minimize the loss of Achilles tendon strength (15).

Charcot neuroarthropathy causes the bones of the foot to undergo subluxation and dislocation, which produces a misshapen foot with osseous prominences. Ostectomy is performed to minimize or remove an osseous prominence. When an ulcer and an exostosis are present, the spur can be resected by using a direct or an indirect approach. A direct approach is performed by excising the ulcer and removing the underlying spur through the same incision. The indirect approach is performed by removing the spur from a remote site that is then closed separately from the site of the ulcer excision to avoid cross-contamination. For longstanding and deep ulcers with underlying exostosis, the indirect approach might not be possible because osteomyelitis is typically present. Surgical debridement of the infected bone and a six-week course of intravenously administered antibiotics that are specific to the organisms revealed by bone culture are mandatory.

Although limb preservation is the goal, the patient might undergo amputation of the Charcot foot because of related comorbidities, the severity of the Charcot deformity, or the extent of the infection. Surgical amputation is most commonly performed in patients who have severe Charcot neuroarthropathy of the foot and ankle with extensive osteomyelitis.

Surgical reconstruction typically is performed during stages 2 or 3 of the disease (9). Often, patients present during the later stages (i.e., coalescence and remodeling phase) of Charcot neuroarthropathy because they notice ulcerations or changes to the shape of the foot. It is unusual for patients to present during the acute stages of Charcot neuroarthropathy. During stage 0 or 1, the acute phases, surgical application of static external fixation has been advocated as an alternative to the traditional treatment option of total contact casting. External fixation has been used for early stabilization of acute Charcot joint subluxations and dislocations. Early stabilization with an external fixator off-loads the ulcerations and maintains the anatomic position of the bones, which avoids further deformity and allows for early partial weight bearing (5,16).

The anatomic locations of Charcot deformities have been defined by multiple authors (2,11,12). The exact locations of the deformities, especially in the midfoot joints, are difficult to determine based on plain radiographs because of the osseous superimposition. This distinction is critical because a Charcot deformity in the tarsometatarsal region typically is a stable deformity whereas a Charcot deformity in the midfoot region is an unstable deformity that leads to plantar-central and plantar-lateral ulcerations. Charcot neuroarthropathy in the ankle and subtalar regions also produces an extremely unstable deformity. When multiple locations are affected, the foot becomes severely unstable and difficult to manage.

A Charcot foot that is stable can be placed in a shoe or a brace while off-loading or accommodating osseous prominences. When Charcot deformity leads to an unstable foot, ulcerations are inevitable. Recurrent ulceration, osseous deformity, instability, and infection (e.g., osteomyelitis) are indications for surgical intervention. Eradicating the ulcer and infection and realigning the dislocated/subluxated foot joints with subsequent stabilization are the goals of reconstructing a Charcot foot and ankle. These factors and the patient's medical and surgical histories are critical to formulating an appropriate surgical plan for accurate correction.

An osteotomy is performed in a coalesced Charcot foot that is stable and can undergo osseous realignment. Shortening of the foot typically occurs when a midfoot wedge resection is performed. For example, a dorsally and medially based wedge is typically removed in patients with midfoot rockerbottom deformity. In patients with tarsometatarsal Charcot deformity, a similar type of wedge osteotomy can be performed. External fixation can be used

to gradually correct an osteotomy to realign the osseous segments. A percutaneous Gigli saw technique is preferred for midfoot osteotomies (14).

Arthrodesis is an important surgical step that should be used in all reconstructions of the Charcot foot and ankle (7). Open reduction with internal fixation is the traditional surgical technique that is used to stabilize the deformities. Internal fixation failure and recurrent breakdown of the Charcot joint have caused surgeons to develop new techniques to treat the effects of Charcot neuroarthropathy. Plantar plating and the use of additional hardware were introduced to better stabilize the Charcot foot and ankle (6). Arthrodesis with rigid internal fixation has remained an important treatment that is used to maintain a stable plantigrade foot.

SURGICAL TECHNIQUES (ACUTE VS. GRADUAL CORRECTION)

The surgical principle of obtaining a correction and then maintaining the correction applies not only to fracture reduction but also to deformity correction with the use of the Ilizarov device. The first stage of reconstructing a Charcot foot is to obtain a correction (i.e., acute or gradual soft-tissue correction and osseous realignment), and the second stage is to maintain the correction (i.e., arthrodesis or stabilization). As previously noted, almost all corrections require Achilles tendon Z-lengthening or gastrocnemius-soleus recession.

Acute Correction

Historically, open reduction with internal fixation was the mainstay for treatment of Charcot foot deformities. Large open incisions were made to remove the excess bone and to reduce the fragmented or dislocated bone. In addition, screw fixation or plantar plating was traditionally performed in an attempt to stabilize the Charcot joint. These invasive surgical procedures typically resulted in a nonanatomic correction (e.g., shortening of the foot or incomplete deformity correction) and occasionally resulted in neurovascular compromise, incision healing problems, infection, and the use of casts or boots for non-weight-bearing patients. Although performing open reduction has disadvantages, in cases of tarsometatarsal Charcot deformity, it is advantageous. Typically, Charcot neuroarthropathy of the tarsometatarsal joints is associated with mild-to-moderate deformities because the tarsometatarsal joints are structurally interlocked. Acute realignment achieved by performing a wedge resection and applying internal fixation produces a stable foot.

External Fixation

During the last decade, important advances have been achieved in the technology, preoperative deformity planning, and basic science of external fixation, especially regarding its use for deformity correction. Increased knowledge and improved technology have tremendously expanded the indications and applications of external fixation, including the use of external fixation and deformity-correction principles to treat Charcot neuroarthropathy of the foot and ankle.

External fixators allow for fine-tuning of residual deformity correction outside the operating room. With internal fixation, the precise plan for deformity correction must be obtained at the time of surgery and cannot be altered during the postoperative period. External fixator constructs can allow early weight bearing, which can lessen the severity of disuse osteoporosis, and allow for access to the soft tissues for wound care (16).

The disadvantages of external fixation include the length of treatment, complications such as pin-site infections, and the special surgical expertise required for construction of the frame. Most associated complications are minor and can be addressed nonoperatively. Typically, when operative intervention is required, the treatment can continue while the complication is being treated (16).

External fixation can be used for stabilization of an acute correction or for gradual deformity correction. The initial use of external fixation was for static fixation purposes only after acute correction was obtained with either open osteotomy or arthrodesis (7,17-19). The disadvantages of this approach are the large amount of bone resection required, which shortens the foot, and the large incisions required, which increase the rate of infection and the potential for wound-healing problems.

Gradual Correction

Gradual deformity correction with external fixation is preferred for large deformity reductions of the dislocated Charcot joint(s). Correction with external fixation allows for gradual and accurate realignment of the dislocated/subluxated Charcot joints. One advantage of using an Ilizarov apparatus to gradually correct the deformity is that the technique is minimally invasive, especially for patients with multiple previous incisions. Gradual correction also allows for anatomic correction without loss of foot length or bone mass. External fixation allows for partial weight bearing and limits neurovascular compromise because the correction occurs slowly over a period of time.

A stable or coalesced foot with Charcot deformity will require an osteotomy for correction of the deformity. Osteotomy can be performed by using the percutaneous Gigli saw technique. Midfoot osteotomies can be performed across three levels (i.e., talar neck and calcaneal neck, cubonavicular osseous level, and cuneocuboid osseous level). Performing a proximal osteotomy across multiple metatarsals is best avoided because of the disturbance of the interossei, risk of neurovascular injury, and multiple bones that require stabilization (14).

For an unstable Charcot foot or an incompletely coalesced Charcot foot, correction can be obtained through gradual distraction. Despite the radiographic appearance of coalescence, majority of patients with Charcot deformities can undergo distraction without osteotomy to realign the anatomy of the foot. After realignment, the correction is maintained by creating an osseous fusion with rigid internal fixation that is inserted percutaneously. This two-stage correction is a new technique that was developed by the senior author (Paley). The first stage consists of osseous realignment achieved by performing ligamentotaxis. Distraction and realignment restore the osseous structure and allow for soft-tissue healing. The second stage consists of removing the external fixator while simultaneously performing minimally invasive arthrodesis of the affected joints with percutaneous insertion of internal fixation. We prefer to use multiple large diameter, fully threaded, cannulated, intramedullary metatarsal screws that are inserted percutaneously through the head of the metatarsal by dorsiflexing the metatarsophalangeal joint. Recently, headless screws [e.g., Acutrak Fusion screws (Acumed, Beaverton, Oregon, U.S.A.)] have been used to achieve compression. Typically, three screws are used: medial and lateral column screws and one central screw. These screws span the entire length of the metatarsals to the calcaneus and talus, provide compression across the minimally invasive arthrodesis site, and stabilize adjacent joints. For example, a Charcot midtarsal joint is realigned with gradual external fixation, fused with minimally invasive techniques, and fixated with intramedullary metatarsal screws, which compress the arthrodesis site and stabilize the tarsometatarsal joint. The intramedullary metatarsal screws cross an unaffected joint, the tarsometatarsal joint, thereby protecting the tarsometatarsal joints from experiencing future Charcot neuroarthropathy (Fig. 1).

We have used this gradual distraction technique during the past four years and have achieved good to excellent success. The short-term results are promising considering that neither recurrent ulceration nor deep infection have occurred. Our results have been reproduced by Dr. Guido Laporta who used a similar protocol (oral communication, September 2006). The advantages of our method when compared with the resection and plating method reported by Schon et al. (6) or the resection and external fixation method reported by Cooper (17) are preservation of foot length, soft tissue and osseous anatomy, and cosmesis. Furthermore, our method is much less invasive.

Frame Constructs

A static frame (i.e., a Charcot stabilization construct) should include a distal tibial ring with a closed foot ring. This construct is generally used to treat patients with Charcot neuroarthropathy of the ankle or Charcot neuroarthropathy of the ankle combined with subtalar and/or midfoot Charcot deformity. A forefoot 6 × 6 butt frame (Fig. 2) should be used when gradually correcting a midfoot Charcot deformity with the use of a Taylor spatial frame (TSF) (Smith & Nephew, Memphis, Tennessee, U.S.A.) (20). The butt frame corrects the forefoot on a fixed hindfoot. In cases in which midfoot Charcot deformity is combined with subtalar joint Charcot deformity that cannot be acutely reduced, a forefoot 6 × 6 miter frame or forefoot 6 + 6 frame can be used. The miter or forefoot 6 + 6 frame can correct both hindfoot and forefoot deformities simultaneously. A forefoot 6 × 6 butt frame can also be used to correct both hindfoot and

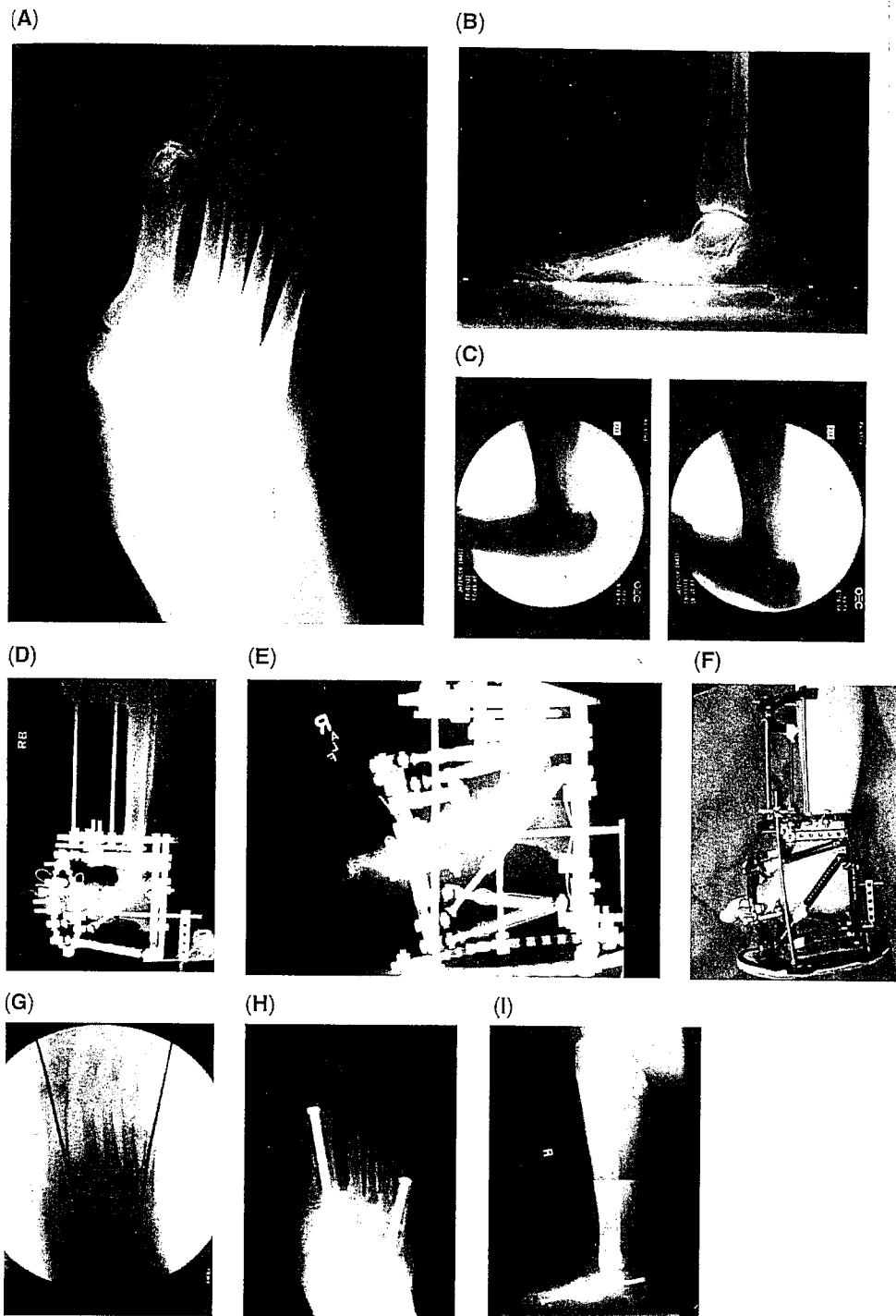


Figure 1 Case of midfoot Charcot neuroarthropathy with hindfoot equinus deformity (Eichenholtz stage 3, stable, without ulceration, or osteomyelitis). **(A)** Anteroposterior view radiograph shows the patient during weight bearing. Note the midfoot abduction. **(B)** Lateral view radiograph shows the patient during weight bearing. Note the dorsal translation midfoot deformity with equinus. **(C)** Percutaneous Achilles tendon Z-lengthening was performed to acutely correct the hindfoot equinus deformity. A lateral view still image obtained by using video fluoroscopy shows the complete correction of the equinus deformity. The hindfoot and ankle were then fixed in the corrected position with the Taylor spatial frame (forefoot 6 × 6 butt) (Smith & Nephew, Memphis, Tennessee, U.S.A.). **(D)** Lateral view postoperative radiograph shows the initial forefoot position. **(E)** Lateral view postoperative radiograph shows the forefoot position after gradual distraction to realign the forefoot to the hindfoot. **(F)** Clinical photograph shows the final midfoot realignment. Immediately after the external fixator was removed, a minimally invasive fusion of the midtarsal joint was performed. **(G)** Intramedullary metatarsal cannulated screws were inserted percutaneously to stabilize both the medial and the lateral columns of the foot. **(H)** Anteroposterior view radiograph shows the patient during weight bearing. Note the accurate anatomic reduction. **(I)** Lateral view radiograph shows the patient during weight bearing. Note the accurate anatomic reduction. The patient has not experienced a recurrence of ulceration or deformity (four-year follow-up).

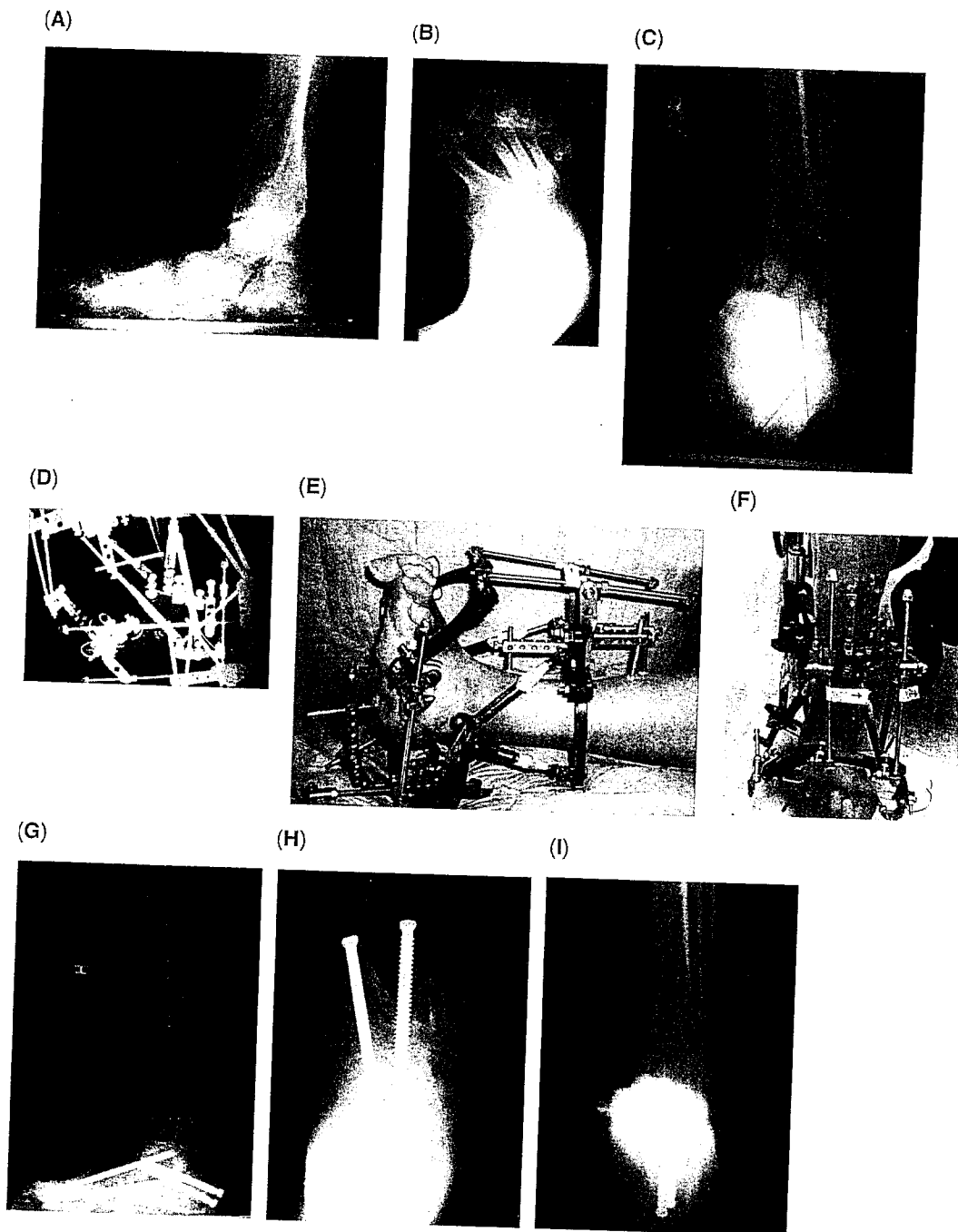


Figure 2 Case of midfoot Charcot neuroarthropathy with hindfoot equinovarus deformity (Eichenholtz stage 3, stable, with superficial ulceration and previously removed osteomyelitis). **(A)** Lateral view radiograph shows rocker-bottom and equinus deformities. **(B)** Anteroposterior view radiograph shows midfoot adduction deformity. **(C)** Axial (Saltzman) view radiograph shows hindfoot varus deformity. A percutaneous Achilles tendon Z-lengthening was performed to aid the hindfoot correction before applying the external fixator. However, complete equinus correction was not achieved acutely. A percutaneous midfoot Gigli saw osteotomy was performed across the neck of the calcaneus and talus. A Taylor spatial frame (long bone) was used for gradual soft-tissue correction of the hindfoot (equinovarus) while the forefoot was gradually corrected with the use of an Ilizarov apparatus through the midfoot osteotomy. **(D)** Lateral view postoperative radiograph shows the midfoot osteotomy with the external fixator in place. The hindfoot equinus deformity was corrected first through gradual soft-tissue realignment. The external fixator was then modified while the patient was in the clinic by backing out the half-pin that crossed the sinus tarsi into the talus so that the half-pin was located only in the calcaneus. The hindfoot varus deformity was then gradually corrected through the subtalar joint. **(E)** Lateral view and **(F)** anteroposterior view clinical photographs show the Ilizarov pusher and puller rods that were used to gradually correct the rockerbottom and adduction midfoot deformities. External fixation was removed after realignment was achieved. Immediately after removal of the fixator, a minimally invasive fusion of the midtarsal and subtalar joints was performed with the use of intramedullary metatarsal cannulated screws inserted percutaneously. **(G)** Lateral view radiograph shows the patient during weight bearing. **(H)** Anteroposterior view radiograph shows the patient during weight bearing. **(I)** Axial view radiograph shows the patient during weight bearing. Note the accurate anatomic reduction. The patient has not experienced recurrence of ulceration or deformity (three-year follow-up).

forefoot deformities simultaneously by stabilizing the ankle and talus with the vertical U-plate (reference ring) and then correcting the forefoot (proximal reference program) and the hindfoot (distal reference program) with two sets of TSF struts (combined anterior and posterior forefoot 6×6 butt frames). A miter frame can also correct a supramalleolar deformity combined with a midfoot Charcot deformity. The TSF can also be combined with Ilizarov parts to create the necessary frame construct (Fig. 3).

Creative frame construction is required because the pedal anatomy and the small size of the foot make it difficult to apply external fixation. Bone segment fixation is important; otherwise, incomplete anatomic reduction or failure of osteotomy separation occurs. Stirrup wires are external fixation wires that are inserted through the bone and are bent 90° to extend and attach to a ring distant from the point of fixation. This stirrup wire is not tensioned and captures osseous segments that are distant from a ring. Small wire fixation is preferred in the

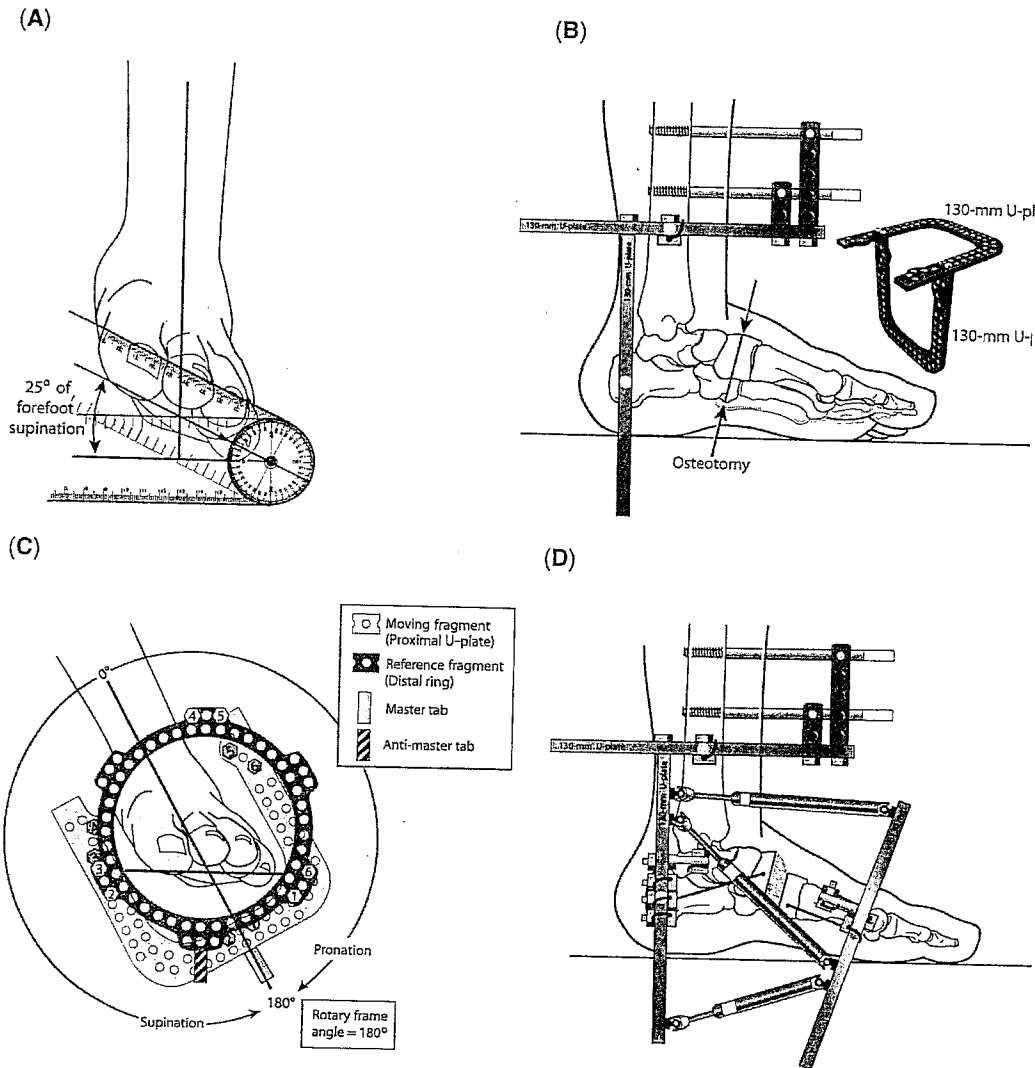


Figure 3 (A) Illustration of a forefoot supination deformity of 25° , as measured clinically. No hindfoot or ankle deformity was present. A percutaneous Gigli saw osteotomy at the cuneocuboid osseous level was performed before application of external fixation. Taylor spatial frame (forefoot 6×6 butt) was mounted (Smith & Nephew, Memphis, Tennessee, U.S.A.). (B) To fix the tibia, talus, and calcaneus, two U-plates were joined and mounted orthogonal to the tibia in the anteroposterior and lateral planes. With the hindfoot held in a neutral position, the calcaneus was then fixed in a neutral position with two crossing 1.8-mm wires. A talar neck 1.8-mm wire was also fixed to the posterior U-plate. Stirrup 1.8-mm wires were inserted just proximal and distal to the osteotomy and were bent 90° to fixate each of them to their respective external fixation ring. A forefoot ring was then mounted orthogonal to the metatarsals in the anteroposterior and lateral planes with two 1.8-mm crossing metatarsal wires. (C) Note the orientation of the rings and the locations of the struts and master tab. The forefoot 6×6 butt frame is shown mounted in a 180° rotary frame offset. Other degrees of rotary frame offset may be used as well. (D) Illustration shows the final correction. Note the slight distraction to allow complete varus correction.

Table 1 Clinical Management

Classification Subgroup	Clinical Evaluation	Surgical Approach	Pearls	Complications/Pitfalls
Eichenholtz stage 0 or 1	Location of Charcot deformity, stability, ulcer, osteomyelitis	Immobilize with total contact cast or static external fixation	Conversion to stage 2 or 3, eradicate osteomyelitis	Nonunion or malunion, malalignment, recurrent ulcer, osteomyelitis, amputation
Eichenholtz stage 2 or 3	Location of Charcot deformity, stability, ulcer, osteomyelitis	Acute correction with internal fixation or gradual correction with external fixation then stabilization	Anatomic realignment, fusion of Charcot joint and stabilization of adjacent joints, eradicate osteomyelitis	Nonunion or malunion, malalignment, recurrent ulcer, osteomyelitis, amputation

foot because of the size and consistency of the bones (Fig. 3). Construction of extremely stable constructs is of great importance when treating a patient with neuropathy (Table 1).

COMPLICATIONS

External Fixation Complications

Patients with neuropathic conditions require close monitoring (i.e., weekly or biweekly) to assess for fixation failure and pin-site infection. Infections around the pin sites of the external fixation device are common and are treated by administering oral antibiotics. The infections rarely require removal of the pins or surgical debridement.

Malalignment

Obtaining proper anatomic alignment in the axial, sagittal, and transverse planes is critical. The severity of Charcot deformities makes it challenging to obtain accurate anatomic correction. Failure to accurately realign the foot and ankle can lead to recurrence of Charcot neuroarthropathy or recurrence of ulceration.

Ulceration/Osteomyelitis

Aggressively debriding the ulceration/osteomyelitis to the level of healthy bleeding tissue should always be performed. External fixation allows for wound healing while the deformity is being corrected.

Fixation Failure

Internal and external fixation (e.g., pin, wire, ring) breakage is not an uncommon complication and can occur when the biomechanical forces of the fixator are exceeded by the lengthening/deformity-correction process or by excessive weight bearing. This complication can be avoided by placing the fixator in a biomechanically advantageous position, using multiple points of fixation, and limiting weight bearing in neurologically compromised patients.

Wound-Healing Problems

Problems with incision healing can be avoided with an atraumatic surgical technique and proper preoperative planning of incision placement. For example, when performing an acute shortening of the midfoot by removing a large bone wedge, a transverse incision should be used to prevent the skin tension that would occur with a longitudinal incision.

FUTURE DIRECTIONS

The aftermath of Charcot neuroarthropathy of the foot and ankle can be devastating for the patient and extremely challenging for a surgeon to manage. Medical advancements to prevent neuropathy and eliminate the disease would be the ideal solution. In the meantime, improvements in technology will provide better fixation options and an enhanced understanding of the metabolic factors of Charcot neuroarthropathy will improve pharmacologic treatment.

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REVIEW OF LITERATURE

Authors	Methods	Results	Conclusions
Wang et al. (5)	28 patients (retrospective review), open Achilles tendon lengthening, hybrid external fixation, monolateral fixation corrected the medial and lateral columns	No patients had further ulceration, consolidation averaged 3.1 mo, longest follow-up 2 yr	Correct equinus, stabilization of Charcot deformity with external fixation is an effective treatment
Cooper (17)	100 patients (retrospective review), acute open reduction with mostly static external fixation	Mean follow-up 22 mo, no recurrent ulcerations at the primary site, three limbs not salvaged, five patients developed ipsilateral Charcot ankle, stabilization of midfoot in a mean of 4.2 mo	Multiplanar deformities should be corrected gradually with a customized external fixator, external fixation offers various advantages over internal fixation
Farber et al. (18)	11 patients (retrospective review), excision of ulcer, open osteotomy with application of static external fixation	Mean follow-up 24 mo, external fixation with concurrent fusion, average fusion time 57 days and then total contact casting for an average of 131 days, No recurrent ulceration at the surgical site	Treatment good for ulcer resolution and prevention of amputation, fibrous vs. osseous union had no effect on clinical outcome
Simon et al. (7)	14 patients (retrospective review), Eichenholtz stage 1 of the tarsometatarsal joint treated with open reduction and fusion with internal fixation and autologous bone graft	Mean follow-up 41 mo, mean time to weight bearing without assistance was 15 wk, mean time to return to regular shoe gear was 27 wk, no repeat ulceration, gait analysis showed no difference to age-matched diabetic patients	Demonstrated the safety and efficacy of early operative intervention to restore foot alignment and function in cases of Charcot neuroarthropathy of the tarsometatarsal region

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