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ORIGINAL STUDY

Management of diabetic neuropathic ankle arthropathy by arthrodesis using an Ilizarov frame

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Charcot neuroarthropathy is a peripheral and autonomic neuropathy that typically presents as a hyperaemic event (e.g. a red, swollen foot). The diabetic Charcot ankle and foot is a potentially limb-threatening disorder that is being recognized with increasing frequency in persons with longstanding diabetes and concomitant peripheral sensory neuropathy. While considered a rare complication of diabetes, it can be a devastating complication requiring months of treatment to arrest its progression. The main problems encountered in this process are osteopenia, fragmentation of the bones of the foot and ankle, joint subluxation or even dislocation, ulceration of the skin and the development of deep sepsis. Arthrodesis using an Ilizarov external fixator is regarded as an optimal choice for the treatment of Charcot arthropathy.

Keywords : Charcot joint ; diabetes ; ankle ; arthrodesis ; Ilizarov.

INTRODUCTION

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 Jordan noted the connection in 1936 (*21*). Diabetic neuroarthropathy is a destructive process affecting the bony components of a denervated joint. Diabetes mellitus is now the main cause of Charcot neuroarthropathy in both the developed and developing worlds (*16*). Diagnosis of Charcot neuroarthropathy is made clinically after observing a lack of normal foot sensation, presence of ulceration, and the presence of foot deformity because of joint subluxations / dislocations (*25*).

Many factors contribute to the destruction of the bones and joints in patients with Charcot neuroarthropathy. These factors include peripheral neuropathy (loss of protective sensation), autonomic neuropathy, unrecognized injury, weight bearing stress (osseous malalignment/soft tissue imbalance), continued repetitive stress on injured structures or incidental trauma, increased blood flow, metabolic abnormalities, renal disease/transplant, osteoporosis, and glycosylation of bone proteins and collagen (*3,23*).

The mechanisms for the development of Charcot neuroarthropathy are not completely understood. However, two theories that address the causes of Charcot neuroarthropathy have been described : a neurovascular theory and a neurotraumatic

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theory (*5*). 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 blood flow increases the arterio-venous shunting, which causes hyperaemia, demineralization and bone resorption (*2,14,15*)

Excessive osteoclastic activity without a concomitant increase in osteoblastic function has also been documented in Charcot foot (*13*). 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 (*5*).

Charcot neuroarthropathy is also characterized by uncontrolled inflammation in diabetic patients because of the presence of an abnormal expression of nuclear transcription factor NF-(kappa) B, associated with the increased release of proinflammatory cytokines, such as tumour necrosis factor-alpha and interleukin-1 beta, with an increased osteoclastogenesis which results in bone softening and destruction, and in turn, potentiates the inflammatory process (*11*).

Eichenholtz believed that “the appearance of the roentgenological features were sufficient to establish the diagnosis” of Charcot arthropathy (*10*). He presented a classification system for Charcot arthropathy, based more on radiographic than on clinical findings, which described three “well defined” stages (*23*).

Stage 1 : Development phase, characterized by fragmentation of bones and cartilage, joint effusions, subluxation and dislocation, soft tissue oedema, hyperaemia, bone resorption, and intra-articular fractures.

Stage 2 : Coalescence phase, characterized by decreased soft tissue oedema, healing of fractures, and organization of bone fragments.

Stage 3 : Reconstruction phase, characterized by new bone formation and remodeling of bone (*5*).

Shibata *et al* (*22*) added another stage : Stage 0 : Acute phase, characterized by swelling, warmth, joint instability, and normal radiographic anatomy of the foot and ankle. Chantelau *et al* (*6*) have recommended that magnetic resonance imaging (MRI) used as a diagnostic tool at this stage is the most sensitive indicator of the underlying “bone stress injury” that can lead to a fully manifested Charcot arthropathy if left undiagnosed or untreated.

Deformities of the foot and ankle resulting from a neuropathic fracture or dislocation cause difficulty with shoe-fitting and marked alteration in the load applied to various parts of the plantar surface of the foot during weight-bearing. These changes lead to an increased propensity for ulceration in high-pressure areas. These ulcers may become a portal of entry for bacteria and thus may result in superficial or deep infection. A deformity may also be associated with joint instability, which is accentuated by weight-bearing, especially with involvement of the hindfoot or ankle. These changes lead to the loss of the plantigrade position of the foot and development of progressive varus, valgus, equinus, or calcaneus deformity (*16*). The current belief is that an operation done during the acute developmental stage will result in inadequate internal fixation because of fragmentation of bone and may contribute directly to destruction of the bone architecture. Arthrodesis is regarded as an option for the treatment of Charcot arthropathy in the coalescence and reconstruction phases only (*17,19*).

PATIENTS AND METHODS

Between 2004 and 2009, 12 patients (12 ankles) with diabetic neuropathic arthropathy of the ankle (Eichenholtz stage 2 and 3) were treated by tibio-talar or tibio-calcaneal fusion in a prospective study. Resolution of the acute development stage (stage 1) was determined radiologically and clinically by the lack of local swelling, erythema, and elevated skin temperature, and by comparison with the contralateral ankle. All the patients had type II diabetes, there were 8 women and 4 men, the right side was affected in 7 patients, the left side in 5 patients. They all presented with hypermobile, deformed ankles with positive anterior drawer, varus and valgus stress tests. No patient gave a history of major trauma to the foot and ankle more than twisting injury.

No one presented with chronic ulcer, only two presented with hyperaemia from contact with the brace. All patients had peripheral neuropathy with loss of deep sensations. No patient had had any surgical intervention for treatment of the ankle problem, but bracing was tried in 7 patients with unsatisfaction for all of them. Plain antero-posterior and lateral radiographs were done for all patients. CT was done when needed to delineate the bony architecture of the ankle. MRI was also performed when needed and feasible to exclude avascular necrosis of the talus or occult infection. Peripheral circulation was assessed preoperatively using Doppler ultrasound. The presence of a pulsatile flow in the posterior tibial artery and an ankle-brachial index of 0.65 or more signified adequate perfusion (3).

The contraindications to arthrodesis include (1) infection of the soft tissue or bone except when the arthrodesis is performed as a staged procedure after the infection has been treated, all osteomyelitic bone has been resected, and the soft tissues has healed ; (2) a fracture that is in the acute (development) phase of the neuropathic disease process (Eichenholtz Stage 1) ; (3) uncontrolled diabetes or malnutrition ; (4) peripheral vascular disease ; (5) insufficient bone stock to obtain fixation ; and (6) the inability of the patient to comply with the post-operative regimen (e.g. because of mental illness) (5).

Operative technique

Prophylactic antibiotics were administered intravenously, with the patient lying supine under spinal, epidural or general anaesthesia, Oxygen saturation was measured using a pulse oximeter. A pneumatic tourniquet was applied about the thigh in all cases. The ankle joint was accessed through the anterolateral approach. The ankle was extensively debrided in all patients, with removal of the articular cartilage and excision of any loose or avascular bone until healthy bleeding bone was exposed from distal tibia and talus (when healthy). Four patients had the talus fractured, fragmented and unhealthy, so it was excised and tibio-calcaneal fusion was done. The resulting defect was packed with cortico-cancellous bone graft harvested from the ipsilateral iliac crest. Temporary tibio-talar or tibio-calcaneal fixation was obtained by 2 crossed (3 mm) Steinman pins with the ankle and foot in 10° equinus, external rotation, and 5° valgus.

The frame typically consists of 2 tibial rings, plus foot plate or 5/8 ring for the hind foot, and 1/2 ring for the forefoot may be added. In the 4 cases with tibio-calcaneal fusion we applied a third ring proximally to allow for proximal osteotomy and leg lengthening. In the early cases we used to fix the frame to bone by tensioned wires and 5 mm Schanz screws till we had fracture of the tibial shaft in case number 4 at the site of pin loosening, so in later cases we avoided using Schanz screws in the tibial shaft when possible, and when needed we used 3-4 mm Schanz screws. While the correction was generally done at the time of operation and a static frame was applied, compression was started after 6 weeks during the course of bone consolidation according to healing.

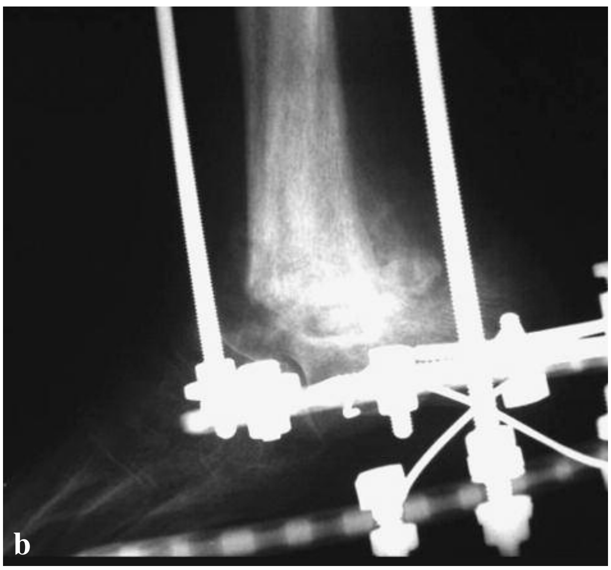
Walking with partial weight bearing was allowed 3 days postoperatively for 8 weeks, and then full weight bearing was allowed till consolidation of arthrodesis, followed by dynamization of the fracture for at least 2 weeks before removal of the frame.

RESULTS

A tibio-talar fusion was done in 8 feet, and a tibio-calcaneal fusion was done in 4 feet after resection of the talus. Osseous fusion was considered to be present when there was definite radiographic evidence of trabeculae crossing the site of arthrodesis and when the gap at the site of arthrodesis had disappeared (fig 1).

Fusion was confirmed in 9 patients (75%), 2 had a stable pseudarthrosis and 1 had an unstable pseudarthrosis. The mean follow-up period was 19.3 months (8-36 months) and no patient was lost to follow-up.

The mean frame time was 21.2 weeks (13-27 weeks) with a mean dynamization period of 3 weeks (2-4 weeks). Pin track infection occurred in 10 patients ; all were grade I and were treated by improving pin care, oral and topical antibiotics. No wound infection, dehiscence, or skin ulceration occurred. One patient (case number 4) had a fracture of the tibial shaft at the level of the proximal Schanz screw in the 13th week of frame time before consolidation of the arthrodesis ; this was managed by frame removal and application of an above-the-knee cast for 6 weeks till healing of the fracture, as the patient refused continuation of the frame and managing the fracture. This particular case ended up with an unstable pseudarthrosis after complete healing of the tibial fracture (fig 2). Patients that had tibio-talar arthrodesis had shortening of 1.5-2.5 cm (mean of 2 cm). In patients that had



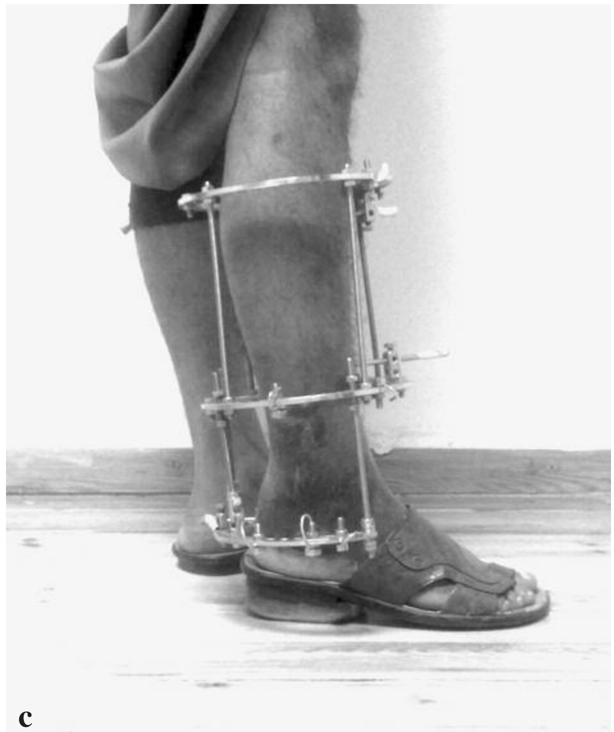


Fig. 1. — Case no. 8 a) preoperative radiograph : A-P view showing complete ankle joint destruction and dissolution of talus. b) postoperative radiograph : lateral view showing full bony fusion before frame removal. c) clinical photograph with full weight bearing before frame removal.

tibio-calcaneal arthrodesis we did a proximal tibial osteotomy for lengthening to equalize the leg lengths.

DISCUSSION

The management of patients who have diabetic neuropathic arthropathy of the foot and ankle is challenging. Education of patients about prevention, early recognition of arthropathy, and prompt institution of protective treatment are clearly the most effective means available to diminish the impact of this problem. The mainstay of treatment for neuropathic arthropathy, once it has been diagnosed, continues to be prolonged external immobilization in a plaster cast or a brace (*7,18*).

Occasionally, however, there are patients who, when they are first seen, have a disabling deformity or severe instability for which treatment with a brace or a plaster cast alone is destined to failure (*19*). For those patients, reconstruction of the foot and ankle is a valuable technique. The goals of operative treatment are to allow the patient to wear a shoe or a brace and to prevent amputation.

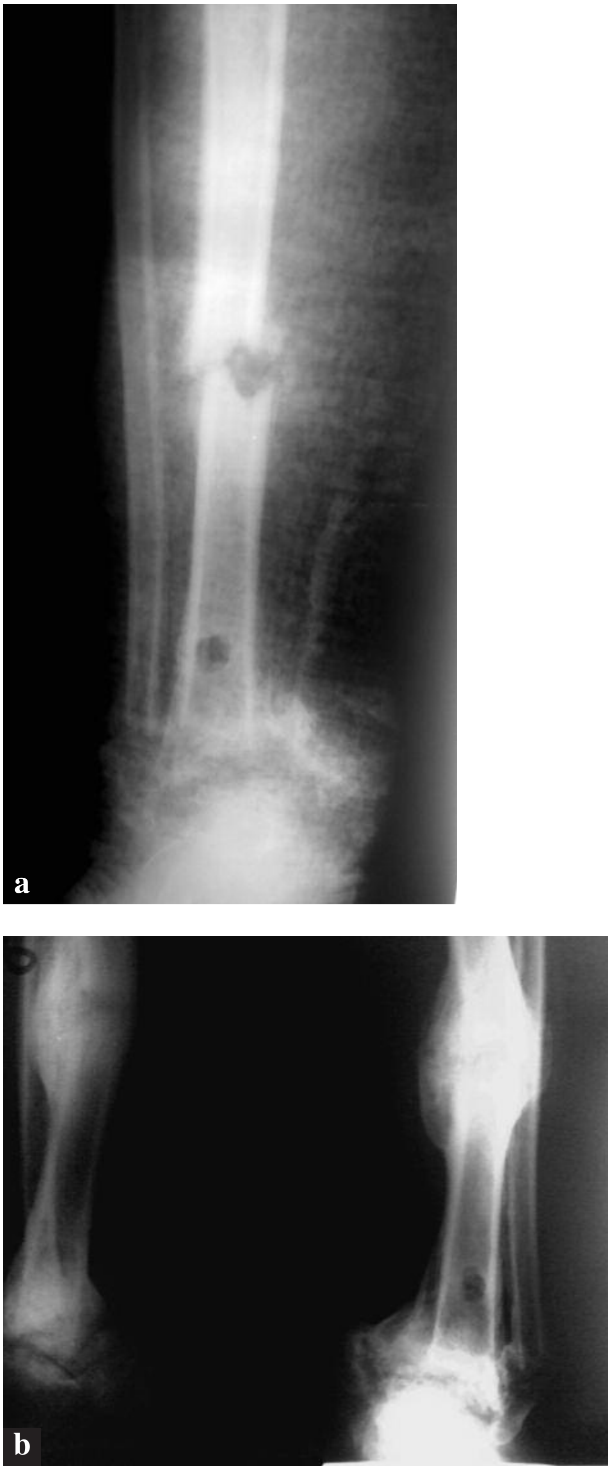


Fig. 2. — a) Case no. 4 with fracture of the mid shaft of the tibia before full consolidation of arthrodesis. b) healing of fracture 6 weeks later after cast removal

Despite complications, the overall rate of success is more than 91.6% (*1,4,9,20*).

Stability and appropriate alignment are more important than union for achieving a successful result.

Open reduction with internal fixation for Charcot osteoarthropathy is associated with a high rate of complications and failure because of infection, bone softening, resorption, fragmentation and breakage of the implant (*5,11*).

Complex reconstructive procedures with arthrodesis are more frequently reserved for realignment and stabilization of severely deformed feet and ankles in an effort to avoid amputation. The choice of internal or external fixation depends on the quality of the bone. Generally in Charcot disease, the bone stock is poor and external fixation provides better compression with fewer fixation failures and soft tissue complications. Due to its ability to correct multiplanar deformities in osteopenic bone even in the presence of open wounds, the circular (Ilizarov) external fixation is preferred for most of Charcot foot and ankle reconstructions (*12*).

In the present study 12 patients (12 ankles) with diabetic neuropathic arthropathy of the ankle (Eichenholtz stage 2 and 3) were treated by tibio-talar or tibio-calcaneal fusion using Ilizarov external fixator after open debridement of the joint and dead bones, and realignment and stable fixation.

We had a 75% rate of solid fusion in this study, which compares favourably with results obtained by Papa *et al*, Shibata *et al*, and by Stuart and Morrey (*19,22,24*) Furthermore, since two of the pseudoarthroses were clinically stable at the most recent follow-up examinations, the overall success rate was 91.6%.

Patients in this study had a mean frame time of 4.9 months which is comparable to the studies done by El-Gafary *et al*, who had fusion in their patients within 5 months including frame dynamization period (*11*), Wang *et al* who had fusion within 3.1 months (*26*), and Cooper who had fusion within 4.2 months (*8*).

Surgery is most often reserved for those patients with severe or unstable deformities that are not amenable to long term bracing alone. By achieving a plantigrade foot with a stable foot and ankle during weight bearing, it greatly reduces the chances of recurrent ulcerations and infections in a neuropathic limb.

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