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Case Reports and Series

Tibiocalcaneal Fusion for Charcot Ankle With Severe Talar Body Loss: Case Report and a Review of the Surgical Literature

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ABSTRACT

Severe bone loss resulting from talar body necrosis in the Charcot ankle can be challenging to treat. In particular, the Charcot ankle will demonstrate progressive instability and deformity, causing protrusion of the medial or lateral malleolus, which will mostly lead to skin ulcers or osteomyelitis and, in some cases, will ultimately require transtibial amputation. Problems such as bone fragility, poor compliance with load-bearing restrictions, susceptibility to infection, and circulatory disorders cause difficulties in the surgical treatment of the Charcot ankle. We believe that tibiocalcaneal fusion is a reliable method to obtain satisfactory outcomes in these difficult cases. However, no study has reported on the use of a locking plate for tibiocalcaneal fusion. Therefore, we report on tibiocalcaneal fusion using a locking plate in 3 patients with Charcot ankle and severe talar body loss. All patients achieved bony union with a plantigrade foot and without any skin complications. We have concluded that a locking plate provides rigid fixation and easier insertion of additional screws, when necessary.

Charcot neuroarthropathy can occur because of diabetes, myelophthisis, myelomeningocele, and congenital insensitivity to pain with anhidrosis, with diabetes the most common cause (1). This neuroarthropathy will initially be characterized by local inflammation, the causes of which are often unclear, with progression to bone destruction that leads to subluxation or luxation. The deformity observed in Charcot neuroarthropathy will be exacerbated by repeated microtrauma, accelerated bone resorption, bone fragility resulting from autonomic imbalances, and/or the influence of inflammatory cytokines (1).

Charcot neuroarthropathy occurs most frequently in the foot and ankle joint and is anatomically defined using the Brodsky classification system. Type 1 involves the tarsometatarsal and naviculocuneiform joints and comprises approximately 70% of reported cases; type 2 involves the Chopart and subtalar joints and accounts for approximately 20% cases; and type 3 involves fractures of the ankle joint (type 3a) and the posterior calcaneal tuberosity (type 3b), comprising approximately 10% of cases, mainly in the ankle. Instability will be marked in types 2 and 3, with severe deformity believed to accompany type 3a in particular (2). The progression of Charcot neuroarthropathy will most often follow predictable clinical and radiographic patterns and is a widely recognized Eichenholtz classification (Table 1). Casts or orthotic treatments have generally been used according to the Eichenholtz stage; however, surgery should be considered when manipulating or maintaining an appropriate position becomes difficult. Clear standards have not been established for the timing of surgery, but most procedures have been conducted when the neuropathy has progressed to stage II or later, after inflammation has subsided and bony consolidation has begun (2,3). The most serious problem related to the Charcot ankle (Brodsky classification type 3a) is that instability and progressive deformity cause protrusion of the medial or lateral malleolus, which mostly leads to skin ulcers or osteomyelitis and, in some cases, leads to transtibial amputation. Thus, the aims of treatment of Charcot ankle are to reduce inflammation, prevent further deformity, and allow walking without plantar callosity or ulceration.

Talar body necrosis is a feature of the Charcot ankle. It is difficult to treat the severe bone loss resulting from talar body necrosis. Moreover, the Charcot ankle is particularly challenging because of bone fragility, poor compliance with load-bearing restrictions, susceptibility to infection, and circulatory disorders.

Usually, tibiocalcaneal fusion using intramedullary nails will be performed to treat the Charcot ankle. However, recently, some

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Table 1
Eichenholtz classification for the progression of Charcot neuroarthropathy

Stage	Clinical and Radiographic Findings	Recommended Treatment
0	Normal radiographic findings	Frequent follow-up with serial radiographs to monitor development of stage I Charcot neuroarthropathy
	Loss of protective sensation with swelling and erythema	Protected weightbearing
	Clinical instability	Patient education on diabetic foot care
I	Osteopenia, periarticular fragmentation, fracture, subluxation	Non-weightbearing or protected weightbearing using a total contact cast
	Continued warmth and swelling, increased ligamentous laxity	Frequent follow-up and radiographic evaluations, with serial cast changes until erythema, calor, and inflammation resolve
II	Absorption of debris, early fusion, and sclerosis	Protected weightbearing with a total contact cast, Charcot restraint orthotic walker, or clamshell ankle-foot orthosis
	Decreased warmth and swelling	
III	Joint arthrosis, osteophytes, subchondral sclerosis	Plantigrade foot: custom inlay shoes
	Absence of inflammation, stable on examination	Nonplantigrade foot or recurrent ulceration: debridement, exostectomy, correction, or fusion with internal fixation
		Osteomyelitis: debridement with or without staged reconstruction with internal or external fixation, or amputation

studies have reported the use of a locking plate during tibiotalocalcaneal fusion to improve the union rate (4,5). In the present study, we report the outcomes of tibiocalcaneal fusion using a proximal humeral locking plate in 3 patients with Charcot ankle and severe talar loss.

Patients and Methods

Three patients with Charcot ankle (Brodsky classification type 3a) and severe talar body loss were included in the present study. The primary disease was diabetes in 2 patients and lumbar spinal canal stenosis in 1. The mean patient age was 68 (range 66 to 70) years, and the mean observation period was 21 (range 12 to 36) months. Surgery was performed when the Charcot ankle had progressed to Eichenholtz stage III (Table 2).

The patient was placed in the supine position, and the skin was incised directly above the fibula. The distal 10 cm of the fibula was removed after subperiosteal detachment to preserve the peroneal tendon sheath. The distal fibula was resected to expose the talocrural and subtalar joints. Next, the necrotic talar body was removed, the tibial and calcaneal joint surfaces were curetted to prepare them for fusion, and the subchondral bone was perforated with a 2.0-mm Kirschner wire to stimulate the bone marrow. A proximal humerus internal locking system plate (Synthes, Paoli, PA) was placed upside down for tibiocalcaneal fusion. The proximal portion of the proximal humerus internal locking system plate was fixed to the calcaneus using locking screws in multiple planes. Next, a headless screw was inserted anteriorly and across the talar head to the tibia. The resected fibula was morselized and used in the surrounding bone graft (Fig. 1). The patients remained non-weightbearing for 8 weeks postoperatively and then began to progressively bear weight using a patellar tendon-bearing brace. The brace was used until bony union had been confirmed radiographically.

Results

Bony union was obtained in all cases, and all patients could walk without any braces or walking aids. No intraoperative or postoperative complications, such as infection, skin disorders, or plantar callosity, developed.

Table 2
Clinical data from 3 patients who underwent tibiocalcaneal fusion

Patient No.	Age (y)	Sex	Primary Disease	Complications	Follow-up Length (mo)
1	68	Female	Lumbar spinal canal stenosis	None	36
2	70	Male	Diabetes	None	15
3	66	Female	Diabetes	None	12

Case Report

A 66-year-old female presented to our hospital because of idiopathic ankle edema and slight pain while walking. Severe talar body loss was observed by radiography, and Charcot ankle was suspected (Fig. 2). Diabetes was her primary disease, and, subsequently, she began receiving insulin therapy. Because the patient was considered to have a diabetes-related Charcot ankle, she was first immobilized in a cast and instructed to avoid weightbearing. Surgery was performed 5 weeks later, after the local inflammation had resolved and her blood glucose level had reached normal levels. Severe talar body loss was observed on computed tomography; however, the talonavicular and calcaneocuboid joints were preserved. Because magnetic resonance imaging had revealed a viable talar head, tibiocalcaneal fusion preserving the talar head was performed. Radiographs taken at 6 months after surgery showed bony union and good foot alignment. A leg length discrepancy of approximately 3 cm associated with talar body removal was observed, but she reported no subjective symptoms and no difficulty while walking. A plantigrade foot was acquired, and no callosity was seen (Figs. 3 and 4).

Discussion

The patients included in the present study had type 3a Charcot ankle according to the Brodsky classification system. This type is the most likely to progress to instability and severe deformity. Such patients can develop a limp, and those with diabetes as the primary disease are at risk of progressive skin ulcers or osteomyelitis due to protrusion in the deformed area. Thus, surgery is considered necessary to achieve rigid fixation and obtain an appropriate corrective position.

The surgical procedure for severe talar body loss includes talus removal, tibiotalar fusion, tibiocalcaneal fusion, and artificial talus replacement. Previously, talus removal was performed; however, its use has not been continued recently, because it alone will not resolve the instability and pain but, rather, leaves patients with a limp (6,7). Tibiotalar fusion using a sliding tibial graft was described by Blair in 1943, and several variations have since been reported. In this method, after talar body removal, bone fragments from the anterior surface of the tibia are used in a sliding graft on the remaining talar neck. Its benefits include little limb shortening, the possibility of subtalar and Chopart joint preservation, and a relatively normal foot appearance; however, it has been associated with a high rate of pseudarthrosis