The Journal of Foot & Ankle Surgery 52 (2013) 295–297

Contents lists available at ScienceDirect

The Journal of Foot & Ankle Surgery

journal homepage: www.jfas.org

Reconstruction of Lisfranc Joint Dislocations Secondary to Charcot Neuroarthropathy Using a Plantar Plate

Dave Garchar, DPM [1], Lawrence A. DiDomenico, DPM, FACFAS [2], Kaj Klaue, MD [3]

[1] *Ryan Foot and Ankle Clinic, Charlotte, NC*
[2] *Ankle and Foot Care Centers, Boardman, OH*
[3] *Clinica Luganese, Sede Moncucco, Lugano, Switzerland*

A R T I C L E I N F O

Level of Clinical Evidence: 3
Keywords:
arthrodesis
Charcot foot
diabetes mellitus
foot
internal fixation
neuropathy
surgery

A B S T R A C T

Lisfranc joint dislocation secondary to Charcot arthropathy is a debilitating condition that often leads to ulceration and infection. After conservative treatment, such as bracing and appropriate shoe wear fail, the only option might be amputation. However, we have seen good clinical outcomes from applying a plate to the plantar (tension) side of the medial midfoot. In our retrospective study, 24 consecutive patients (25 feet) from April 1999 through July 2004 underwent Charcot reconstruction for Lisfranc dislocation. Clinical and radiographic follow-up examinations were performed every 3 weeks during the postoperative course. Union was achieved in 24 (96%) of the 25 feet. The average time to ambulation was 11.68 (range 7 to 20) weeks for the 24 patients. The average follow-up period was 38.0 (range 17 to 64) months. The union and interval to ambulation rates showed that a plate applied to the plantar aspect of the medial midfoot provides a strong, sturdy construct for arthrodesis and ambulation.

Neuropathic arthropathy was first described in association with tabes dorsalis in 1868 by Jean Martin Charcot, with bone and joint changes secondary to a trophic effect (1). Thereafter, similar bone and joint changes have been described in patients with diabetes mellitus, syringomyelia, leprosy, scleroderma, hereditary sensory neuropathy, Lyme disease, and alcoholic neuropathy (2–5). Currently, Charcot neuroarthropathy is characterized by episodes of active and inactive periods. An edematous, erythematous, warm foot that shows progressive destruction and dislocation of the bone and joints characterizes the active period. Charcot neuroarthropathy is a very common complication of diabetes mellitus, with a recent prevalence range of 0.08% in the general diabetic population to 13% in the high-risk diabetic population (6). Because diabetes mellitus is becoming an epidemic, especially in the United States, it can be expected that the prevalence of Charcot neuroarthropathy will increase.

The exact etiology of neuropathic arthropathy remains undefined; however, 2 theories have been postulated. The neurotraumatic theory, experimentally confirmed by Eloesser (7) in 1917 and later developed by Johnson (2) in 1967, proposed that a decreased protective sensation permits cumulative microtrauma, resulting in fracture and joint destruction. The neurovascular theory was proposed by Brower and Allman (8) in 1981 and suggested that a loss of vasomotor tone or autosympathectomy leads to increased blood flow and active bone resorption. Both theories are believed to play a role in the development of Charcot neuroarthropathy (9).

The staging of neuropathy, as described by Eichenholtz (10), uses both clinical and radiographic criteria. In stage I, the foot exhibits progressive edema, hyperemia, and an increased temperature. Radiographically, debris, joint dislocation, and bone fragmentation are noted. In stage II, the swelling, warmth, and redness decrease. Radiographically, sclerosis, absorption of fine debris, and fusion of large bone fragments are seen. In the final stage, stage III, the clinical inflammation has resolved, with radiographic bony consolidation. An additional stage, stage O, was later added by Shibata et al (11), in which the patients exhibits the same clinical signs as in stage I, but the radiographic changes are absent or minimal. Although the clinical and radiographic criteria are important, the anatomic location and incidence are essential to determine the treatment and outcomes. The tarsometatarsal articulation is the most common location for the development of neuropathic arthropathy, with 60% to 70% of cases at this location (12).

No definitive treatment option is available for neuropathic arthropathy, and both conservative and surgical options are available (13). Conservative treatment usually consists of a lengthy course of immobilization, achieved with the use of contact casting, removable casts, an ankle-foot orthosis, or molded shoes (14). Associated ulcers are

Financial Disclosure: None reported.
Conflict of Interest: None reported.

Address correspondence to: Lawrence A. DiDomenico, DPM, FACFAS, Ankle and Foot Care Centers, 8175 Market Street, Boardman, OH 44512.
E-mail address: ld5353@aol.com (L.A. DiDomenico).

1067-2516/$ - see front matter
http://dx.doi.org/10.1053/j.jfas.2013.02.019

treated with local wound care and antibiotic therapy, as needed. If conservative treatment is ineffective, patients become at risk of skin/bone breakdown, which can become limb or life-threatening. Therefore, surgical treatment aimed at correcting the deformity could be indicated. The primary goal of surgical treatment is to create a functional plantigrade foot, allowing the patient to be return to functional activity, prevent additional breakdown, and decrease the medical costs.

Patients and Methods

Indications

The indications for surgical treatment include the chronic ulcers associated with bony deformities or contractures, unstable joints of the foot and ankle that cannot be treated with a shoe or brace, recurrent infected ulcers with bony prominences, and acute displaced fractures in neuropathic patients with adequate circulation (Fig. 1) (3,15). Traditionally, surgical treatment has been limited to the inactive phase of the disease. All of the patients were identified in the senior author's (L.A.D.) practice and consecutively enrolled between April 1999 and July 2004. The coauthors identified the patients based on operative records and abstracted the data. Subsequent surgical management, identification of bony union, and identification of postoperative complications was determined by the attending surgeon (L.A.D.).

Gross instability at the tarsometatarsal articulation will lead to the characteristic symptomatic medial and plantar bony prominences, which can cause ulceration and infection, often resulting in amputation of the limb (16). Ostectomy alone does not address the biomechanical instability and thus does not provide long-term benefit (17). However, amputation, in addition to the effect of the loss of limb, puts the patient at additional risk of amputation of the contralateral limb (18). Pinzur (19) demonstrated that energy consumption with a unilateral amputation is directly proportional to the number of functional joints remaining and inversely proportional to the length of the remaining limb. Additionally, because of the energy costs, patients often adapt their lifestyle by becoming less active, resulting in reduced physical conditioning of the muscles of the remaining lower extremity (18,20,21). Furthermore, Waters et al (21) demonstrated that energy consumption increases 10% to 40% with a unilateral below the knee amputation and 50% to 70% with a unilateral above the knee amputation.

Limited information is available from published reports concerning midfoot arthrodesis using the planter plate for neuropathic joints. However, we believe that benefits exist for those who fit the appropriate clinical history and surgical criteria.

Procedure

The patients were taken to the operating room and placed in the supine position. After administration of general anesthesia, cotton padding was placed over the upper thigh of the foot undergoing surgery, and a pneumatic thigh tourniquet was applied. The foot was then prepared and draped in the usual sterile manner. The tourniquet was applied and elevated. Attention was then directed to the Achilles tendon, where a linear incision was made over the distal one third of the lower leg. Tendoachilles lengthening was performed with an open Z-plasty, achieving approximately 2 cm of lengthening. Attention was then directed to the planter medial aspect of the foot. A straight incision was made, beginning at the talonavicular joint and extending to the distal one third of the first metatarsal shaft. The incision was deepened by sharp and blunt dissection down to the first metatarsal, medial cuneiform, and navicular. A full-thickness tissue flap was then retracted off the tarsometatarsal joints. Attention was directed toward the base of the Lisfranc articulation. An osteotome was used to remove an approximately 1-cm block of necrotic bone across the Lisfranc joint, down to good, healthy, bleeding bone. If all the Charcot bone could not be resected through the medial incision, a second incision was made on the lateral aspect of the foot between the fourth and fifth metatarsals. This incision was deepened down to the base of the metatarsals and cuboid. All necrotic bone from the lateral to the medial aspect was removed, completing the resection of the Charcot bone across the Lisfranc joint. Depending on the extension of the Charcot destruction, the same procedure was performed through the innominate (ie, naviculocuneiform joints) to restore the medial arch of the foot. The Lisfranc joint was adducted and held in a plantarflexed position using two 0.062-in. Kirschner wires for temporary fixation. Next, a 3.5-mm reconstruction plate was eccentrically loaded and applied to the plantar aspect of the first metatarsal, medial cuneiform, and navicular (Fig. 2). One 3.5-cm cortical screw was placed outside the plate in an oblique fashion, seating on the medial wall of the first metatarsal and aiming at the lateral edge of the navicular. Its length depended on the extension of the debrided region. A second cortical screw was inserted outside the plate from the medial cuneiform or navicular into the second or third metatarsal base. No fixation was used on the fourth and fifth rays. Autogenous or allogenic cancellous bone can be used to fill any void or space at the arthrodesis site. The incisions were then dressed using Betadine-soaked adaptic dressings (Purdue Fredrick Co, Stamford, CT), 4 × 4 dressings, and Kling bandages (Johnson & Johnson, New Brunswick, NJ). A dry sterile dressing was applied, followed by a posterior splint or Jones compression dressing. The postoperative course included serial radiographs every 3 weeks, and the patients were kept non-weightbearing, depending on the radiographic and clinical appearance (Figs. 3 and 4). The patients were placed in a walking cast or controlled ankle motion walker for an additional 2 to 3 months, after the initiation of weightbearing with physical therapy.

Results

A total of 25 feet in 24 patients underwent the procedure. The mean age of the 14 (58%) males and 10 (42%) females in our study was 58.8 (range 42 to 74) years. All the patients were neuropathic, 22 (91.67%) had diabetes, and 12 (50%) were insulin dependent. One (4.17%) patient had alcoholic neuropathy, and 1 (4.17%) patient had idiopathic neuropathy. The 24 patients were either in late stage II or stage III of Charcot deformity. Postoperative complications, including infection (n = 4 [16%]), ulcers (n = 2 [8%]), Charcot developing into other joints (n = 3 [12%]), wound dehiscence (n = 1 [4%]), and heel decubitus ulcers owing to a poorly fitted cast (n = 1 [4%]), were noted in 10 (42%) patients. Union, determined by a clinically stable foot with no evidence of sclerosis or gapping at the fusion site, maintenance of solid hardware, and trabeculation identified on radiographs, was achieved in all but 1 foot. The patient whose foot did not consolidate was the patient who had undergone bilateral surgery. The 50-year-old male, a noninsulin-dependent diabetic smoker, experienced a good outcome with the right foot and was ambulating within 8 weeks.

Fig. 1. Rocker bottom deformity noted on lateral radiograph.

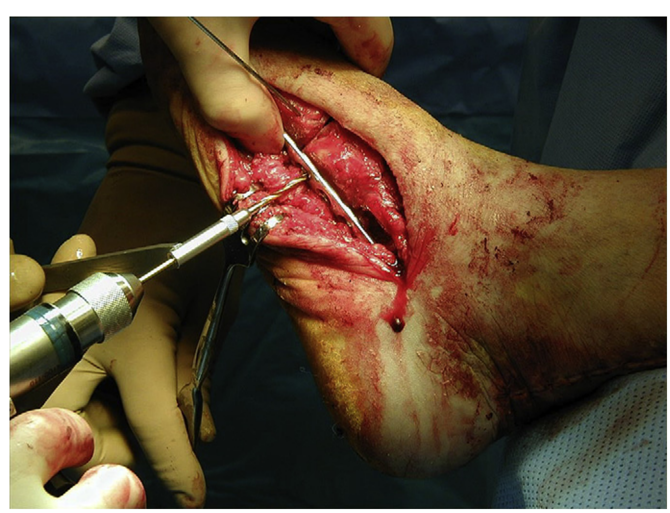

Fig. 2. Reconstruction plate applied to plantar aspect of medial column.

Fig. 3. Postoperative anteroposterior radiograph at 24 months revealing consolidation across Lisfranc joint.

However, the left foot posed problems with infection and recurrent ulceration. Tobacco use was not uncommon in the present cohort, with 10 (42%) reporting active smoking. The mean interval to ambulation in a walking boot was 11.68 (range 7 to 20) weeks. The average length of follow-up was 38.0 (range 17 to 64) months.

Discussion

The accepted treatment of neuropathic arthropathy has historically been immobilization in a cast or brace until consolidation has occurred. However, some patients will have a disabling fixed deformity or severe instability that will not respond to bracing or casting alone (22). Research has shown that a plate applied to the plantar (tension) aspect of the medial midfoot provides a stronger, sturdier construct than midfoot fusion with screw fixation (23). Studies have also revealed that a plantar plate allows significantly less initial displacement and maintains stabilization at a much greater load (23). By maintaining correction during high and repetitive loading, the patient should be able to ambulate earlier while maintaining the arthrodesis. To date, data have not been published concerning the outcome of using a plantar plate for Charcot reconstruction and the return to earlier ambulation. Using the plantar plate on the tension site and employing AO principles, we have found that a sturdy construct on which to ambulate markedly increases the stability of the reduction and helps to facilitate arthrodesis and early ambulation.

In conclusion, various methods of fixation can be used for arthrodesis of a midfoot dislocation. The modification of applying a plate to the plantar medial foot for midfoot fusion provides a more stable construct than simple screw fixation, enhancing the arthrodesis (24). Our results have indicated that arthrodesis with a return to early ambulation can be achieved using this technique. In patients with an unstable deformity or those experiencing recurrent ulcerations, this is a viable alternative to amputation. As with all observational investigations, this study was limited by potential biases related to surgeons determining the outcomes of interest, the absence of an explanatory analysis, and the small sample size, and we realize that future prospective cohort studies and randomized controlled trials will be needed in order to better understand the surgical repair of the neuropathic foot.

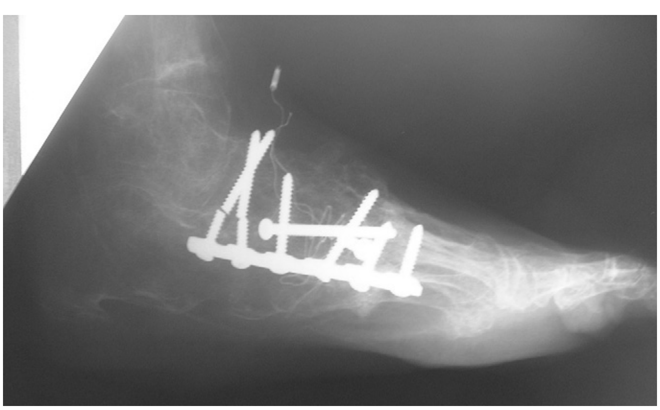

Fig. 4. Postoperative lateral radiograph at 24 months showing medial arch height maintained.

References

1. Charcot JM. Sur quelques arthropathies qui paraissant depredre d'une lesion du cerveau ou de la moelle apiniere. Arch Physiol Normale Pathol 1:161–178, 1868.
2. Johnson JTH. Neuropathic fractures and joint injuries: pathogenesis and rationale of prevention and treatment. J Bone Joint Surg 49-A:1–30, 1967.
3. Alvarez RG, Trevino SG. Surgical treatment of the Charcot foot and ankle. In: *Operative Treatment of the Foot and Ankle*, Appleton & Lange, Stamford, CT, 1999, pp. 147–178.
4. DiDomenico LA, Blasko GA, Adams HB. An unusual case of bilaterally symmetrical neuropathic osteoarthropathy of the midfoot as a result of Lyme disease-induced peripheral neuropathy: a case report. Foot Ankle Int 23:155–157, 2002.
5. Schon LC, Marks RM. The management of neuroarthropathic fracture-dislocations in the diabetic patient. Controversies Foot Ankle Trauma 26:375–392, 1995.
6. Frykber RG, Belczyk R. Epidemiology of the Charcot foot. Clin Podiatr Med Surg 25:17–28, 2008.
7. Eloesser L. On the nature of neuropathic affections of the joints. Ann Surg 66:201, 1917.
8. Brower AC, Allman RM. Pathogenesis of the neurotrophic joint: neurotraumatic vs. neurovascular. Radiology 139:349–354, 1981.
9. Gupta R. A short history of neuropathic arthropathy. Clin Orthop 296:43–49, 1993.
10. Eichenholtz SN. *Charcot Joints*, Charles C. Thomas, Springfield, IL, 1966.
11. Shibata T, Tada K, Hashizume C. The results of arthrodesis of the ankle for leprotic neuroarthropathy. J Bone Joint Surg 72-A:749–756, 1990.
12. Brodsky JW. Management of Charcot joints of the foot and ankle in diabetes. Semin Arthrop 3:58–62, 1992.
13. Pinzur MS, Shields N, Trepman E, Dawson P, Evans A. Current practice patterns in the treatment of Charcot foot. Foot Ankle Int 21:916–920, 2000.
14. Holmes GB Jr, Hill N. Fractures and dislocations of the foot and ankle in diabetics associated with Charcot joint changes. Foot Ankle Int 15:182–185, 1994.
15. Sticha RS, Frascone ST, Wertheimer SJ. Major arthrodeses in patients with neuropathic arthropathy. J Foot Ankle Surg 35:560–566, 1996.
16. Brodsky JW, Rouse AM. Exostectomy for symptomatic bony prominences in diabetic Charcot feet. Clin Orthop 296:21–26, 1993.
17. Early JS, Hansen ST. Surgical reconstruction of the diabetic foot: a salvage approach for mid foot collapse. Foot Ankle Int 17:325–330, 1996.
18. Huang CT, Jackson JR, Moore NB, Fine PR, Kuhlemeier KV, Traugh GH, Saunders PT. Amputation: energy cost of ambulation. Arch Phys Med Rehab 60:18–24, 1979.
19. Pinzur MS. The metabolic cost of lower extremity amputation. Clin Podiatr Med Surg 14:599–602, 1997.
20. Kegel B, Carpenter ML, Burgess EM. Functional capabilities of lower extremity amputees. Arch Phys Med Rehab 59:109–120, 1978.
21. Waters RL, Perry J, Antonelli D, Hislop H. Energy cost of walking amputees: the influence of level of amputation. J Bone Joint Surg 58-A:42–46, 1976.
22. Papa J, Myerson M, Girard P. Salvage, with arthrodesis, in intractable diabetic neuropathic arthropathy of the foot and ankle. J Bone Joint Surg 75-A: 1056–1066, 1993.
23. Marks RM, Parks BG, Schon LC. Midfoot fusion technique for neuroarthropathic feet: biomechanical analysis and rationale. Foot Ankle Int 19:507–510, 1998.
24. Myerson MS, Henderson MR, Saxby T, Short KW. Management of mid foot diabetic neuroarthropathy. Foot Ankle Int 15:233–241, 1994.