sciendo Romanian Journal of Orthopaedic Surgery and Traumatology, Volume 1, Issue 2, July-December 2018. pp:112-120
doi:10.2478/rojost-2018-0089

REVIEW

Treading on tricky ground: reconstructive approaches to Charcot neuropathic arthropathy of the foot

Daniela–Elena Ion
"Carol Davila" University of Medicine and Pharmacy Bucharest, Romania

Correspondence to: Ion Daniela–Elena, 5th year student,
"Carol Davila" University of Medicine and Pharmacy Bucharest,
8 Eroii Sanitari Bld., Code 050474, District 5, Bucharest, Romania,
Mobile phone: +40730 988 804, E-mail: daniela.ion1@stud.umfcd.ro

Abstract

Introduction and purpose:Charcot neuroarthropathy defines a cluster of progressive lesions affecting the joints and bones, as well as the soft tissues of the foot in the context of diabetes, a pivotal role being attributed to peripheral neuropathy. Loss of sensation and proprioception, subsequent repeated trauma, muscle and autonomic nervous system impairment contribute to the alteration of the foot's architecture and distribution of pressure, ultimately triggering ulceration and gangrene. The urge to avoid amputation has fueled the development of conservative and reconstructive techniques capable of delaying, if not preventing such negative outcomes. The purpose of this review was to present the most frequently used reconstruction procedures and the challenges arising in adapting them to particular foot morphologies and lesion stages.

Methods:Literature search was conducted using PubMed, resulting in around 90 articles, multicenter studies and reviews, 26 of which were considered most relevant in providing the guidelines for orthopedic reconstruction and postoperative care in Charcot foot patients with diabetic neuropathy prevailing over arteriopathy.

Results:The tarsometatarsal and metatarsophalangeal joints are most frequently affected. Closed reduction, arthrodesis, and tendon lengthening are key features of an efficient correction, alternatively accompanied by resections and tenotomies. Ulceration and callus debridement may also be necessary, while prolonged casting and immobilization remain obligatory.

Conclusions:Most authors agree that stabilizing the deformities, optimizing the pressure on the soft tissues, and promoting the healing of potential lesions are the main purposes of the interventions. Prompt recognition and correction of Charcot foot deformities improve life quality and minimize the prospects of amputation.

Keywords: Charcot foot, Charcot neuropathic arthropathy, reconstruction, diabetes, foot deformity

Introduction

Charcot neuropathic arthropathy is a complex foot pathology encountered in diabetic patients, which can develop unnoticed for long periods of time, but severely affects their quality of life by increasing the risk of amputation. There are two main theories regarding the etiology of Charcot neuropathy (CN): the neurovascular and the neurotraumatic theory [1]. To summarize, vascular and Schwann cell metabolic changes lead to progressive degeneration of proprioceptive, followed by tactile, autonomic, and thermoalgic nerve fibers. The foot's protective sensitivity is annihilated and autosympathectomy causes

excessive vasodilatation with subsequent bone demineralization. With the final degeneration of motor fibers, muscle atrophy occurs, affecting the balance between flexion and extension forces, which, in the context of decreased sensory input and neglected minor trauma, results in altered walking, abnormal plantar pressure distributions, dislocations and fractures, promoting calluses and ulcers that almost inevitably advance towards osteomyelitis and amputation (the risk for CN patients being 7 times greater compared to those with isolated neuropathic foot ulcerations and 12 times greater if secondary ulcers occur) [2].

Traditionally, surgical treatment was reserved for patients in whom orthopedic treatment (including total contact casts, orthosis, CROW walkers and custom-made shoes and braces) [3] had failed, but, as amputation is no longer considered an acceptable outcome, new surgical techniques and devices have been devised to allow the intervention and correction in the earlier stages of deformity. The aim of this review was to outline the most frequently employed reconstruction procedures in CN and provide some guidelines for establishing an algorithm for patient-tailored treatment.

Methods

A search of medical databases was performed to locate relevant literature. PubMed queries revealed around 90 articles, multicenter studies and reviews related to the diabetic CN, using keywords such as "Charcot foot", "neuropathy", "neuropathic arthropathy" and derivatives, as well as filters including "diabetes", "reconstruction", "orthopedics". Exclusion criteria consisted in inclusion of non-diabetic patients, diabetic patients without CN or with prevalent arteriopathy without reporting outcomes separately; focusing on plastic surgery, ulcer removal, or amputation techniques; confusion with Charcot-Marie-Tooth disease; cadaveric studies. A total of 26 articles were selected for the present review.

Results

1. Classification and deformities

Assessing and understanding the sequence of multiplanar deformity is crucial in decifring the surgical approach and fixation methods in diabetic CN, which must be minimally aggressive and provide maximum stabilization of a foot with altered nutritional status and diverse inflammatory and immune changes (involving glycation products, leptin, RANKL) affecting the bone and soft tissue quality that will dictate the outcomes of arthrodesis and wound healing [3-5].

Harris and Brand [6] first proposed a collapse pattern in the neuropathic foot, in which collapse of the posterior pillar led to subtalar incongruity, central foot pressure, talus disintegration with collapse of the anterior pillar followed by the medial and lateral arches. The Eichenholtz (Table 1) [7], Schon (Table 2) [8], Brosdky (Table 3) [9] classifications are currently used, but those of Sanders and Frykberg (Table 4) [10], Sella and Barrette [11] are also mentioned.

Table 1. Eichenholtz classification with Shibata modification

Clinical and Radiograph Findings	
Stage 0	Absence of injury or osteoarticular disorders
Stage 1	Acute phase
	Inflammation, erythema, swelling, and a temperature difference present clinically
	Fracture or dislocation initially subtle
	Later the calcaneal inclination angle is reduced and the talo-first metatarsal angle is broken
	Calcification of the medial arteries evident
Stage 2	Coalescence or subacute phase
	Reduction in temperature, swelling and inflammation
	Reparative process on radiograph
Stage 3	Consolidation or chronic phase
	Resolved inflammation, consolidated fractures

Table 2. Schon midfoot classification

Radiograph findings	
Type I	Lisfranc pattern
Type II	Naviculocuneiform pattern
Type III	Perinavicular pattern
Type IV	Transverse tarsal (Chopart) pattern
Alpha	No additional features
Beta 1	A dislocation is present
Beta 2	The lateral first metatarsal angle is 30° or greater
Beta 3	The lateral calcaneal-fifth metatarsal angle is 0° or greater
Beta 4	The anteroposterior talar-first metatarsal angle is 35° or greater
Stage (A)	The midtarsus is above the metatarsocalcaneal plane
Stage (B)	The midtarsus is coplanar with this plane
Stage (C)	The midtarsus is below this plane

Table 3. Brodsky classification

Stability based on anatomic localization, without differentiating the various mid-tarsal patterns or severity	
Type 1	Midfoot: tarsometatarsal joint
Type 2	Hindfoot: subtalar, talonavicular, calcaneocuboid joints
Type 3a	Ankle: tibiotalar joint
Type 3b	Calcaneus: tuberosity fracture
Type 4	Multiple regions
Type 5	Forefoot: metatarsophalangeal joints

Table 4. Sanders and Frykberg anatomic classification

Locations with associated frequency of onset	
Pattern I	Phalanges, interphalangeal and the metatarsophalangeal joints (15%)
Pattern II	The tarsometatarsal joints (40%)
Pattern III (30%)	The cuneonavicular, talonavicular, and calcaneocuboid articulations
Pattern IV	The talocrural joint (10%)
Pattern V	The posterior calcaneal involvement (5%)

The tarsometatarsal joint and midtarsal joints are most commonly involved [29], the talus often collapses due to avascular necrosis or neuropathic fracture resulting in prominent malleoli [12], varus hindfoot, valgus forefoot and the classically described rocker-bottom deformity accompanied by dorsiflexion of the midfoot onto the hindfoot ("bayonet" effect) [13]. Lateral column involvement (calcaneocuboid luxation) is rarer, but associated with a worse prognosis than medial column involvement (peritalar dislocation of the navicular-cuneiform joint) [14-16] and as the apex of deformity moves more proximally, the deforming forces increase, stressing the affected area and any fixation construct applied [17]. Forefoot deformities comprise claw or hammer toes and prominent metatarsal heads due to proximal interphalangeal joint flexion, which transfers the pressure allocated to the fingers backwards, and diabetic cheiroarthropathy [14,16,17]. Long flexor and triceps surae domination, as well as anterior crural and plantar muscle atrophy contribute to equinus and forefoot deformity, respectively [17].

With patients usually going to the hospital in these advanced stages, it is not surprising that the initial, inflammatory stage of the CN, characterized by a non-tender, warm and edematous, disproportionately swollen foot with progressive dislocations, is overlooked. While it is known that immobilizing the foot now in a non-weight-bearing cast until these symptoms decline prevents deformity, conversely, there is no benefit to early surgical intervention [14]. In this stage, the bone softens and inflammation might hinder determining the real extent of deformity, whereas later stages show hard, brittle bone, made up of thin trabeculae with inflammatory infiltrates and hypervascular myxoid tissue [18]. Even later, a plantigrade foot with collinear hindfoot and forefoot may rarely develop an ulcer, so surgery is not necessarily advisable [19].

The main indications for surgical reconstruction are:

- nonbraceable deformity associated with instability
- impending ulceration or inability to heal an ulcer
- recurrent ulcers
- presence of osteomyelitis and/ or significant pain
- acute displaced fractures in neuropathic patients with adequate circulation [3,15,20].

2. Preoperative investigations

Apart from radiographies, CT (or 3D-CT) and MRI imaging are commonly used to

evaluate the bone stock, joint malalignment [21] and, respectively, osteomyelitis. Triple-phase bone scan, combined with white-labeled scan, bone mineral density and biopsy can confirm osteomyelitis, but congestion, minimal pain and a temperature difference above 2°C characterize the inflammatory phase of CN, which is the main differential diagnosis [14]. Determining the vascular status of the foot is essential for the postoperative evolution and success of the reconstruction in diabetic patients: pedal pulses, the ankle-brachial index (falsely elevated in case of non compressible, calcified vessels), toe segmental pressures, transcutaneous oxygen tension (unreliable in edematous feet), as well as MR angiography can serve to either refer the patients for revascularization or exclude them as candidates for limb salvage or reconstruction [3,14, 16,22]. Additionally, some authors prefer to delay surgery until HbA1c is below 8% [15,23].

3.Alignment and complementary interventions

Acute alignment can be achieved through wedge resection (guided by transversal K-wires) [14,17] and fixation, but gradual correction is preferred because it allows maintaining foot length and bone mass and reduces the risk of neurovascular compromise [13]. In case of major deformity or osteomyelitis, double-stage reconstruction is advisable, and the remission of inflammation is obligatory (except for ankle interventions) [15]. Proximal deformities are approached first and the medial column is corrected before the lateral column and subtalar joint, through arthrodeses and ostectomies, aiming for stability rather than compression.

If present, ulcers and equinus deformities are solved first, through excision and, respectively, tendon lengthening procedures. The necessity for the latter is assessed using the Silfverskiold test (mentioned in 5 articles, although 85% of the authors commence with lengthening, with 10.3% equinus prevalence in diabetics and 50% associated ulcers) [24,25]. Removing a major deforming force also allows improved intraoperatory mobility and plantigrade positioning. Options include: the Hoke triple hemisection or open Z-plasty for Achilles tendon lengthening, gastrocsoleus release (Strayer technique), transection of gastrocnemius aponeurosis and soleus fascia, occasionally a peroneal longus or tibialis posterior tendon lengthening; postoperative concerns are overcorrection, rupture and poor blood supply [12,17,26-28]. Claw and hammer toes require percutaneous flexor (long ± short) tenotomy procedures [29,30] extensor hallucis longus tendon Z-lengthening or metatarsophalangeal arthrodesis in the opportunity of immobile joints [14,17].

4. Resections and bone grafts

Bone resections are indispensable, either for preparing fusion surfaces (accompanied by subchondral bone plate, cartilage and synovium removal) [12] relaxing the soft tissues [27] eliminating pressure points or osteomyelitis. Aggressive resections termed "internal amputations" [18] are sometimes necessary, but simple ostectomies can be performed even percutaneously (using a Gigli saw [31] or bone shaving [17]); they are never enough to prevent deformity progress [28], but allow subtle adjustments even after final fixation. Side foot incisions prevent disturbance of blood supply, contamination and scarring (nerve damage is almost irrelevant in CN feet) [17]. Open procedures (by curette or rongeur) address the apex of the deformity (mainly on the plantar surface), going as far as the calcaneum and tibia [32] or even fibula (lateral approach in ankle reconstruction) [15,33]. Biopsies are harvested from the resected, but also the remaining proximal and distal bones.

Healthy fragments can be further used as autografts via morselization and decortication. Contralateral iliac crest, proximal and distal tibial autografts are also popular. Autogenous cancellous bone is typically used

as a nonstructural graft in arthrodesis, whereas cortical autografts provide structural support in spanning defects. Disadvantageous due to donor-site morbidity, limited supply, and increased surgical time, they can be combined with allografts to facilitate incorporation. The latter include cadaveric bone, demineralized and synthetic bone grafts such as inorganic bioceramics, calcium phosphate and calcium sulfate or hydroxyapatites, capable of filling bone voids and augmenting other materials for onlay grafting. Antibiotic-impregnated substitutes are both structurally and microbiologically efficient: Hong et al. [32] used an antibiotic-impregnated cement spacer to complete the defect between the fore- and hindfoot, devoid of a destroyed and avascular talus, fixating it with locking screws through the hindfoot arthrodesis nail concurrently implanted. Some residual foot motion was also granted by the pseudarthrosis between the forefoot and cement spacer in an otherwise immobilized foot.

Bone morphogenetic proteins in the shape of demineralized bone matrix, bone marrow aspirates or platelet-rich plasma further enhance the environment to assure fusion and healing [13,34,35].

5. Stabilization

Sammarco [3] popularized the concept of superconstructs – a set of rules that improve stability and surgical healing in case of bone loss, osteoporosis, dysvascular bone, major deformity correction, and multiple medical comorbidities:

- fusion beyond the zone of injury, sacrificing normal joints,
- bony resection for shortening and adequate deformity reduction without excessive soft tissue tension,
- use of the strongest possible device tolerated by the soft tissue,
- application of the fixation device in a position that maximizes its mechanical function.

From the classical examples - axial screw placement, plantar plating and locking plates – the last 2 are rarely used as standalone constructs because of extensive dissection and periosteal stripping, poor bone quality causing loss of purchase, difficult placement in the desired locations [3,22]. Nevertheless, poly-axially inserted screws, both non locking and locking, in medial or plantar fixation in strong cortical metatarsal bone on the tension side of the fusion mass protects intramedullary fixation devices in the first stages of weight-bearing [18,21].

Intramedullary anterograde or retrograde fixation through axial screws or midfoot fusion bolts (MFB) serves to reestablish the medial (talus-navicular-medial cuneiform-1st metatarsal) and lateral (calcaneus-cuboid-4th/ 5th metatarsal base/ head) columns. No stress is placed on the cortical bone, especially with 6.5 to 8.0 mm diameters (larger medially) and yet the devices accept pressure on both the plantar and dorsal surfaces; limited incisions and deperiostation, unexposed hardware in case of soft tissue wounds and easy reduction by the aid of guidewires and cannulated screws (under fluoroscopy; definitive devices are placed last because they impede proper visibility) strongly advise the use of these methods, but at least 2 diverging implants must be used to avoid creating centers of rotation [12,14,36]. Complications include cannulated screw failure (5,6% to 36,4%) and implant migration, avoidable by fully/ longer threaded or hydroxyapatite coated implants.

A retrograde intramedullary nail can be used in tibiotalocalcaneal arthrodesis, with proximal double fixation or cross-locking through the tibia and, supplementary, distal fixation through the calcaneus and subtalar joint [14,19] 300-mm-long nails are preferred because shorter ones are associated with proximal fractures due to the modulus of elasticity between the bone implant interface and the loss of ankle and subtalar joint motion [19].

External fixation tolerates osteomyelitis and permits immediate weight-bearing for patients having difficulty with long cast immobilization while also forcing patient compliance [3,18]. It allows direct wound visualization, offloading, and assistance in major flap closure procedures while protecting soft tissue and neurovascular structures. It may be employed independently (hexapods or distraction osteogenesis rings in double stage procedures to distract the bones gradually using computer programs based on daily radiographies) [13,31,37] or to protect internal constructs until radiographic bony consolidation occurs (Ilizarov, bar-clamp fixators) [18,33], together with a kickstand apparatus for leg elevation and surgical offloading. The use of smooth wires versus half pins apparently has fewer complications in the diabetic population [38]. Hybrid external fixation combining circular and uni/ biplane constructs is applicable in case of large soft tissue reconstructions or poor bone quality.

Osteomyelitis can be a consequence or a trigger of CN [39]. It often requires two-stage interventions and compels the use of external fixation after debridement for stabilization after the first phase [3]. Local treatment (antibiotic beads through biodegradable - calcium sulfate/ phosphate or non-biodegradable systems - polymethylmethacrylate, removed in the next stage) [31,40] is accompanied by 6 weeks of culture-guided antibiotic i.v. treatment, usually targeting S. aureus, Group A β-hemolytic Streptococcus, Enterobacteriaceae, and P. Aeruginosa [41]. In associated ulcers the surface polymicrobial flora does not necessarily correspond to the bacteria in deeper layers, which stresses the importance of intraoperatory biopsies [28]. Eventually, an infection disease specialist assesses the inflammation and identifies the optimal moment for the second stage of reconstruction [18].

6. Post-operative care and complications

Internal fixation procedures require below-knee or short leg non-weight-bearing split/ univalve/ bivalve casting or an orthosis for a medium of 8 weeks (4 to 12), incision healing being sometimes monitored through windows in the plaster. Radiographic checkup is scheduled around the 6th week, followed by partial and full weight-bearing (cast, orthosis, CROW or Böhler walker) extending to 7-15 months; weight-bearing load control is complicated in CN patients owing to the lack of sensation and other patient morbidities (obesity, arthritis, visual deficits, cardiovascular disease) [42]. Major procedures or infection development require prolonged postoperative use of support devices to aid bone remodeling [18]. Stimulation of bone repair with ultrasound and negative-pressure wound therapy might be helpful, too [14,18].

The following complications may arise: loss of correction, infection, and amputation, nonunion, implant break or migration, with the need for symptomatic hardware removal, fractures, damage to tendons and joints, swelling and lymphedema, burred bone or bone graft substitute leakage through the incision sites, transfer ulcers developing on adjacent areas because of shifted pressure [12,14,17,29,37]. External fixation constructs might additionally develop pin tract infections (at 4 and 8 weeks, due to a loss of wire tension over time owing to premature or excessive frame loading) [33], pin breakage, tibia fracture after device removal [15].

Discussion

Charcot neuropathic arthropathy is among the pathologies that should be approached by a multidisciplinary team including an orthopedic and a plastic surgeon, a vascular surgeon, diabetes and an infectious diseases specialist, as well as a podiatrist, if available. The orthopedic intervention is central and decisive for the progress of the deformities, but the control of the underlying cause (maintaining adequate glycemias, HbA1c and a satisfactory nutritional status) and assuring adequate

vascularization of already compromised tissues are quintessential in order to achieve union, correction and neutralize abnormal pressure distributions. Osteomyelitis and infected, intractable superficial ulcers demand increased intraoperative and postoperative care and thorough debridement, but also applying culture-based antibiotic treatment protocols. Osteomyelitis should be differentiated from the active, inflammatory phase of CN, in which invasive procedures are not indicated. Some authors believe that conservative treatment through immobilization in this phase is as efficient as reconstruction in preventing the consequences of the disease.

Rectifying pressure points across the plantar aspect of the foot often require additional procedures such as correcting claw and hammer toes, prone to hyperkeratosis on bony prominences, which also count as pre-ulcerative lesions. A major role is played by the reduction of the equinus deformity, caused either by muscular contraction or capsular and tendon retraction, several procedures being applicable at the beginning of the surgical procedure, but complications such as tendon rupture might be triggered by inconsistencies between weight-bearing particularities in lengthening procedures and osteoarticular procedures.

The surgeon's armamentarium includes numerous internal and external fixation devices, but no consensus has been reached as to which type is most advantageous in CN patients. Limb salvage is achieved in 91-97% of the cases by using internal fixation, up to 96% with external fixation and 91-93% in combined fixation. One article reported a 100% salvage rate [43], so it can be considered that amputation rates have been significantly decreased by correcting the anomalies associated with CN in time, using devices adapted to the patient's particular deformity. However, infection rates are increased (up to 45%), which is not unexpected in diabetic patients, but postoperative wound and especially external fixation care should be enhanced. Another objective is to decrease postoperative non/ partial weight-bearing intervals and maintain patient compliance.

All the articles provided retrospective case reports, but prospective studies are difficult to conceive due to the immense variability of the deformities. However, it seems that detailed clinical and imaging assessment and classification allow orthopedic surgeons to report cases in a manner that provides useful indications for colleagues facing similar CN patterns across the world.

In conclusion, the treatment of the Charcot neuropathic arthropathy requires patient-adapted techniques, which, despite reducing the risk of amputation imminent once ulcers start developing, involve high risks of complications and therefore it is essential to have knowledge of all the available fixation methods and complementary procedures in order to achieve satisfactory outcomes.

Conflicts of interest

No conflicts of interest are acknowledged.

References

1. Gouveri E, Papanas N. Charcot osteoarthropathy in diabetes: a brief review with an emphasis on clinical practice. World J Diabetes. 2011; 2:59–65.
2. Sohn M, Stuck RM, Pinzur M et al. Lower-extremity amputation risk after Charcot arthropathy and diabetic foot ulcer. Diabetes Care. 2010; 33(1):98–100.
3. Sammarco VJ. Superconstructs in the treatment of Charcot foot deformity: plantar plating, locked plating, and axial screw fixation. Foot Ankle Clin. 2009 Sep; 14(3):393-407. doi: 10.1016/j.fcl.2009.04.004.
4. Witzke KA, Vinik AI, Grant LM et al. Loss of RAGE defense: a cause of Charcot neuroarthropathy?. Diabetes Care. 2011; 34(7):1617–21.
5. Baumhauer JF, O'Keefe RJ, Schon LC et al. Cytokine-induced osteoclastic bone resorption in Charcot arthropathy: an immunohistochemical study. Foot Ankle Int. 2006; 27(10):797–800.
6. Harris JR, Brand PW. Patterns of disintegration of the tarsus in the anaesthetic foot. J Bone Joint Surg Br. 1966 Feb; 48(1):4-16.
7. Eichenholtz SN. Charcot joints. 1966, Springfield (IL): Charles C Thomas.
8. Schon LC, Easley ME, Weinfeld SB. Charcot neuroarthropathy of the foot and ankle. Clin Orthop

Relat Res. 1998; 349:116–31.
9. Brodsky JW, Rouse AM. Exostectomy for symptomatic bony prominences in diabetic Charcot feet. Clin Orthop Relat Res. 1993; 296:21–6.
10. Sanders LI, Frykberg RG. The Charcot foot. In Levin and O'Neal's The Diabetic Foot (7th edn), 2007, JH B, MA P (eds). Mosby Elsevier: Philadelphia, 258.
11. Sella EJ, Barrette C. Staging of Charcot neuroarthropathy along the medial column of the foot in the diabetic patient. J Foot Ankle Surg. 1999; 38(1):34–40.
12. Richter M, Mittlmeier T, Rammelt S, Agren PH, Hahn S, Eschler A. Intramedullary fixation in severe Charcot osteo-neuroarthropathy with foot deformity results in adequate correction without loss of correction - Results from a multi-centre study. Foot Ankle Surg. 2015 Dec; 21(4):269-76. doi: 10.1016/j.fas.2015.02.003.
13. Siddiqui NA, LaPorta GA. Midfoot Charcot Reconstruction. Clin Podiatr Med Surg. 2018 Oct; 35(4):509-520. doi: 10.1016/j.cpm.2018.07.003.
14. Miller RJ. Neuropathic Minimally Invasive Surgeries (NEMESIS): Percutaneous Diabetic Foot Surgery and Reconstruction. Foot Ankle Clin. 2016 Sep; 21(3):595-627. doi: 10.1016/j.fcl.2016.04.012.
15. Wukich DK, Raspovic KM, Hobizal KB, Sadoskas D. Surgical management of Charcot neuroarthropathy of the ankle and hindfoot in patients with diabetes. Diabetes Metab Res Rev. 2016 Jan; 32 Suppl 1:292-6. doi: 10.1002/dmrr.2748.
16. Canale ST, Beaty JH, Campbell WC. Campbell's operative orthopaedics. 2013, Philadelphia, PA: Elsevier/ Mosby.
17. Botezatu I, Laptoiu D. Minimally invasive surgery of diabetic foot - review of current techniques. J Med Life. 2016 Jul-Sep; 9(3):249-254.
18. DiDomenico L, Flynn Z, Reed M. Treating Charcot Arthropathy Is a Challenge: Explaining Why My Treatment Algorithm Has Changed. Clin Podiatr Med Surg. 2018 Jan; 35(1):105-121. doi: 10.1016/j.cpm.2017.08.012.
19. Wukich DK, Mallory BR, Suder NC, Rosario BL. Tibiotalocalcaneal Arthrodesis Using Retrograde Intramedullary Nail Fixation: Comparison of Patients With and Without Diabetes Mellitus. J Foot Ankle Surg. 2015 Sep-Oct; 54(5):876-82. doi: 10.1053/j.jfas.2015.02.019.
20. Garchar D, DiDomenico LA, Klaue K. Reconstruction of Lisfranc joint dislocations secondary to Charcot neuroarthropathy using a plantar plate. J Foot Ankle Surg. 2013 May-Jun; 52(3):295-7. doi: 10.1053/j.jfas.2013.02.019.
21. Stapleton JJ, Zgonis T. Surgical reconstruction of the diabetic Charcot foot: internal, external or combined fixation?. Clin Podiatr Med Surg. 2012 Jul; 29(3):425-33. doi: 10.1016/j.cpm.2012.04.003.
22. Lamm BM, Siddiqui NA, Nair AK, LaPorta G. Intramedullary foot fixation for midfoot Charcot neuroarthropathy. J Foot Ankle Surg. 2012 Jul-Aug; 51(4):531-6. doi: 10.1053/j.jfas.2012.04.021.
23. Pinzur MS, Gil J, Belmares J. Treatment of osteomyelitis in Charcot foot with single-stage resection of infection, correction of deformity, and maintenance with ring fixation. Foot Ankle Int. 2012 Dec; 33(12):1069-74. doi: 10.3113/FAI.2012.1069.
24. Lavery LA, Armstrong DG, Boulton AJ. Ankle equinus deformity and its relationship to high plantar pressure in a large population with diabetes mellitus. J Am Podiatr Med Assoc. 2002; 92(9): 479–82.
25. Frykburg RG, Bowen J, Hall J, Tallis A, Tierney E, Freeman D. Prevalence of equinus in diabetic versus nondiabetic patients. J Am Podiatr Med Assoc. 2012; 102: N2 84–N2 88.
26. Tagoe MT, Reeves ND, Bowling FL. Is there still a place for Achilles tendon lengthening?. Diabetes Metab Res Rev. 2016 Jan; 32 Suppl 1:227-31. doi: 10.1002/dmrr.2745.
27. Rios-Ruh JM, Martin-Oliva X, Santamaría-Fumas A, Domínguez-Sevilla A, López-Capdevila L, Vilà Y Rico J, Sales-Pérez JM. Treatment algorithm for Charcot foot and surgical technique with circular external fixation. Acta Ortop Mex. 2018 Jan-Feb; 32(1):7-12.
28. Kučera T, Šponer P, Šrot J. Surgical reconstruction of Charcot foot neuroarthropathy, a case based review. Acta Medica (Hradec Kralove). 2014; 57(3):127-32. doi: 10.14712/18059694.2014.51.
29. Scott JE, Hendry GJ, Locke J. Effectiveness of percutaneous flexor tenotomies for the management and prevention of recurrence of diabetic toe ulcers: a systematic review. J Foot Ankle Res. 2016 Jul 29; 9:25. doi: 10.1186/s13047-016-0159-0.
30. Rasmussen A, Bjerre-Christensen U, Almdal TP, Holstein P. Percutaneous flexor tenotomy for preventing and treating toe ulcers in people with diabetes mellitus. J Tissue Viability. 2013 Aug; 22(3):68-73. doi: 10.1016/j.jtv.2013.04.001.
31. LaPorta GA, D'Andelet A. Lengthen, Alignment, and Beam Technique for Midfoot Charcot Neuroarthropathy. Clin Podiatr Med Surg. 2018 Oct; 35(4):497-507. doi: 10.1016/j.cpm.2018.05.008.
32. Hong CC, Jin Tan K, Lahiri A, Nather A. Use of a definitive cement spacer for simultaneous bony and soft tissue reconstruction of mid- and hindfoot diabetic neuroarthropathy: a case report. J Foot Ankle Surg. 2015 Jan-Feb; 54(1):120-5. doi: 10.1053/j.jfas.2014.10.009.
33. Hegewald KW, Wilder ML, Chappell TM, Hutchinson BL. Combined Internal and External Fixation for Diabetic Charcot Reconstruction: A Retrospective Case Series. J Foot Ankle Surg. 2016 May-Jun; 55(3):619-27. doi: 10.1053/j.jfas.2015.04.016.
34. Ramanujam CL, Facaros Z, Zgonis T. An overview of bone grafting techniques for the diabetic Charcot foot and ankle. Clin Podiatr Med Surg. 2012 Oct; 29(4):589-95. doi: 10.1016/j.cpm.2012.07.005.
35. Pinzur MS. Use of platelet-rich concentrate and bone marrow aspirate in high-risk patients with Charcot arthropathy of the foot. Foot Ankle Int. 2009; 30(2):124–7.
36. Cullen BD, Weinraub GM, Van Gompel G. Early results with use of the midfoot fusion bolt in Charcot

neuroarthropathy. J Foot Ankle Surg. 2013; 52:235–8.
37. Peterson KS, Hyer CF. Posterior approach for medial column beam screw in midfoot Charcot reconstruction: technique and structures at risk. J Foot Ankle Surg. 2015 May-Jun; 54(3):433-6. doi: 10.1053/j.jfas.2014.10.006.
38. Jones CP, Youngblood CS, Waldrop N et al. Tibial stress fracture secondary to half-pins in circular ring external fixation for Charcot foot. Foot Ankle Int. 2014; 35:572–7.
39. Aragón-Sánchez J, Lázaro-Martínez JL, Quintana-Marrero Y, Álvaro-Afonso FJ, Hernández-Herrero MJ. Charcot neuroarthropathy triggered and complicated by osteomyelitis. How limb salvage can be achieved. Diabet Med. 2013 Jun; 30(6):e229-32. doi: 10.1111/dme.12191.
40. Burns PR, Monaco SJ. Revisional Surgery of the Diabetic Charcot Foot and Ankle. Clin Podiatr Med Surg. 2017 Jan; 34(1):77-92. doi: 10.1016/j.cpm.2016.07.009.
41. Tsourvakas S. Local antibiotic therapy in the treatment of bone and soft tissue infections. In: Danilla S, editor. Selected topics in reconstructive plastic surgery. Rijeka (Croatia): InTech Europe; 2012, 17–44.
42. Wiewiorski M, Yasui T, Miska M, Frigg A, Valderrabano V. Solid bolt fixation of the medial column in Charcot midfoot arthropathy. J Foot Ankle Surg. 2013 Jan-Feb; 52(1):88-94. doi: 10.1053/j.jfas.2012.05.017.
43. Siebachmeyer M, Boddu K, Bilal A et al.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(1):76–82.