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Conservative and Surgical Treatment of Diabetic Charcot Neuroarthropathy (Literature Review)

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Abstract: The basis of treatment for Charcot neuroarthropathy is long-term immobilization in the form of a plaster cast or corset, as well as the use of antiresorptive drugs in the acute stage. However, some patients already have a disabling deformity or severe instability at the time of presentation for whom conservative treatment is doomed to failure. Although conservative treatment using TCC followed by appropriate bracing and footwear is considered the gold standard treatment for NS, surgical treatment is necessary when conservative treatment is ineffective. Surgery includes exostectomy, Achilles tendon lengthening, and arthrodesis.

Keywords: type 2 diabetes mellitus, Charcot foot, treatment.

The purpose of the study is to review the literature on the issues of conservative and surgical treatment of diabetic Charcot neuroarthropathy.

The research material is publications over the past 10 years on this topic.

Conclusions.

1) Early detection and prevention of collapse are the best treatment options for patients with diabetic Charcot arthropathy

2) In patients with diabetes and lower extremity neuropathy, any minor injury requires close monitoring due to the developmental tendency of the limb toward a Charcot process, so appropriate education, improved clinical judgment, and early intervention are necessary to control the disease.

3) In the presence of collapse in the acute stage, the use of unloading TSS and antiresorptive drugs is recommended.

Introduction. Charcot neuroarthropathy (CN) is a severe joint disorder of the foot and ankle that can lead to fractures, permanent deformity, and limb loss. This is a serious and potentially limb-threatening late complication of diabetes mellitus.

NS was first described by neurologist Jean-Martin Charcot in 1868 in a group of patients with syphilis, but the pathophysiology remains a mystery [1-3]. NS is considered to be a destruction of bones and joints secondary to underlying neuropathy, trauma, and disorders of bone metabolism. Although NS is a clinical diagnosis, recent advances in diagnostic imaging have

made the clinical task of deciphering the infection based on Charcot changes easier. Limb salvage is a new option in modern medical and surgical treatment. Pharmacological therapy has also shown promise for treatment.

The above formed the basis for this study.

The purpose of the study is to review the literature on the issues of conservative and surgical treatment of diabetic Charcot neuroarthropathy.

Prevalence of Charcot foot. NS is a progressive non-infectious neuroosteoarthropathy of bones and joints in patients with sensory neuropathy, leading to the destruction of the architecture of the foot [1, 2]. NS is an inflammatory condition that leads to osteolysis and is indirectly responsible for the progressive fractures and multiple joint dislocations that characterize its manifestations. It is usually located in the midfoot, but also occurs in the forefoot and rearfoot. NS is also associated with autonomic neuroarthropathy, infections (leprosy, HIV), toxic exposures (ethanol, drugs), rheumatoid arthritis, multiple sclerosis, congenital neuropathy, sicca, traumatic injury, metabolic disorders and syringomyelia [3, 4]. However, in recent years, diabetes mellitus has become the most common etiology. The exact incidence of NS in people with diabetes has previously been estimated to be 0.1–0.4%, but the incidence has recently been estimated at 0.08–0.13% [1, 5, 6]. In NS with foot and ankle deformities associated with diabetes and neuropathy, the incidence rate is up to 7.5%, with more than 9% of them having bilateral involvement [5, 6]. Moreover, its prevalence is less than the actual figure due to misdiagnosis or delay in diagnosis [1].

The diagnosis of NS is reported to be delayed due to misdiagnosis such as gout, deep vein thrombosis, soft tissue injury, rheumatoid arthritis, or infection. The consequences of a delay in diagnosis are serious and debilitating, such as structural foot deformity. This subsequent deformity in the presence of peripheral neuropathy significantly increases the risk of skin ulceration and lower extremity amputation [10–12]. NS requires emergency medical care, since with early diagnosis, treatment can prevent the destructive process [7-10].

Etiopathogenesis of the disease. Recognizing the pathogenesis of NS is important for deciding treatment strategies. The pathogenesis of the disease is not well understood, and there is no consensus regarding the pathological process that causes NS. Therefore, it is likely that the pathogenesis of this disease is multifactorial [1, 10]. The pathogenesis of NS is explained by two main theories. Neurovascular theory suggests that joint destruction is secondary to an autonomic stimulated vascular reflex, causing hyperemia and periarticular osteopenia with concomitant trauma. On the other hand, neurotraumatic theory suggests that NS is an overuse injury in which desensitized joints are subjected to either repeated microtrauma or a single traumatic event that results in typical Charcot changes [1, 2, 10-12]. The lack of protective sensation delays the recognition of bone injuries, which can overload the unconscious limb and lead to an active Charcot process [13-15]. Loss of sensation prevents the victim from adopting normal protective mechanisms, such as unloading and changing activity, or from seeking medical help. Diagnostic clinical findings include autonomic dysfunction, neurological, vascular, musculoskeletal and radiographic components.

In chronic NS, patients experience reduced symptoms of heat and swelling, and inflammation is usually absent. On X-ray examination, osteophytes, joint consolidation and arthrosis are signs of chronic NS. Dislocation of the tarsometatarsal joint with a rupture of the talo-first metatarsal line and a decrease in the angle of the calcaneus can be seen on a lateral radiograph in the late stage of chronic NSH deformity. The deformity begins from the medial column and in the later stages of NS spreads to the lateral column. Abduction of the arch of the foot and development of bony prominences lead to deformation and ulceration of the foot. Rocker foot deformity with or without plantar ulceration indicates severe chronic deformity typical of chronic NS [7, 8, 15]. Rocker floor deformity begins with collapse of the scaphocuneiform joint, which participates in the scaphocuneiform pattern. The perinavicular pattern begins with osteonecrosis or fracture of

the scaphoid. In later stages, the talus dislocates completely from the scaphoid and ulceration of the calcaneocuboid space begins.

Stages of Charcot's foot. Many systems have been proposed for the classification of Charcot foot. Eichenholtz proposed a systematic classification based on radiological data in 1966 [16]. Its assessment was mainly the radiological evolution of the condition over time and did not include clinical manifestations [1, 16]. Although he described 3 stages, “Stage 0” is defined by a hyperthermic, usually painful and swollen foot in which radiographic changes are absent but magnetic resonance imaging shows bone marrow edema [17–19]. The physician may often suspect either a deep infection or cellulitis [20-22].

The classical staging system proposed by Eichenholtz has been refined over time and is supported by the following clinical findings: Stage I is the fragmentation phase (acute stage) when plain radiographs demonstrate osteopenia, periarticular fragmentation, and subluxation or frank dislocation of the joints. Clinically, the foot continues to be warm and swollen and there may be increased ligamentous laxity [1, 16]; Stage II, fusion stage, subacute Charcot stage (reparative stage) is the early phase of healing. Swelling and warmth decrease, and other typical changes include resorption of bone debris, fusion of bone fragments, formation of new bone, and/or sclerosis of bone ends; and stage III, the consolidation stage, the chronic Charcot stage (reconstructive or bone healing stage) is characterized by a lack of inflammation and progression to a more stable, although often deformed, foot or ankle [1, 16].

Brodsky described an anatomical classification based on the four areas most commonly affected by NS. Brodsky type I (midfoot) includes the navicular-cuneiform and metatarsocuneiform joints, which are the most common (60%). The second most common type, type II (hindfoot), involves the subtalar, talonavicular, or calcaneocuboid joints. Type II accounts for approximately 30–35% of anatomical incidence. Type III is divided into "A" and "B". Type IIIA involves the tibiotalar joint and associated bones, and type IIIB involves a pathologic fracture of the calcaneal tuberosity. [23, 24].

Current classification systems for Charcot foot are primarily based on radiographic findings and anatomical location. Thus, these systems are not sufficient to detect disease in early stages, provide prognostic data, or guide the clinician to specific treatment options. New clinically based staging systems that also classify the Charcot patient as active or inactive based on inflammatory status should be carefully considered.

Treatment of Charcot's foot. The goal of treatment for NS is to achieve a plantigrade, stable foot that can fit into a shoe, and to prevent recurrent ulceration. Treatment depends on many factors, including location, phase of the pathological process, deformity, presence or absence of infection and other concomitant diseases. The severity of the disease determines goals that must be specific and realistic to achieve, while the treatment plan can range from basic shoe modification to limb amputation [1, 25].

The recommended treatment for Eichenholtz stage 0 is frequent surveillance with periodic radiographs to monitor the development of stage I NS and patient education in diabetic foot care [1, 26]. Eichenholtz stage I NS is successfully treated with immobilization and non-weight-bearing in a total contact plaster cast [27, 28]. The duration of treatment is usually determined by the practitioner's judgment of the ability of the affected joints to withstand physiological stress. At this stage, frequent follow-up and radiographic studies with serial castings are essential until the erythema, color and inflammation resolve [1, 2, 16, 22]. Stage II (subacute fusion phase) is usually treated with protected weight-bearing using a total contact cast or a molded polypropylene full-contact ankle prosthesis [7, 22]. At stage III (reconstructive-chronic), if the foot is plantigrade, the patient can wear special shoes with inlays [21, 22, 25]. If the patient has a nonplantigrade foot or a history of recurrent ulcers, debridement, exostectomy, correction, or fusion with internal fixation may be an option. Additionally, in stage III osteomyelitis, the recommended treatment is debridement with staged reconstruction with or without internal or external fixation, or amputation [1, 2,10, 17, 21, 22, 29].

Non-surgical treatment of NS is usually considered in the acute stage and includes offloading the affected foot, treating bone disease and preventing further fractures and/or dislocations [2,10, 22, 5, 30-31]. Additionally, this treatment can also be used in some patients with chronic NS and foot ulcerations. Offloading is the most important step in the treatment strategy for acute NS to consolidate the progression of the deformity. Total contact casting (TCC) is considered the gold standard for the treatment of neuropathic diabetic plantar foot ulcers. The use of TCC reduces mechanical forces, inflammation and swelling; redistributes plantar pressure; limits the destruction of bones and joints; and may consolidate the progression of the deformity. Its overall goal is to preserve the foot, which can then support weight bearing in a shoe or brace [23,30].

The TCC should cover the entire foot and ankle, with all major bony prominences well covered with cotton-based bandages. Frequent cast changes are critical to reduce complications as placement can lead to instability and ulceration of the cast [1, 2,10, 22] and patients should be closely monitored weekly. The cast or device should be inspected and removed at each visit. Wounds should be examined, carefully treated if necessary, measured and photographed [30, 32-35]. At the end of the active phase, the patient can be put on an orthopedic walker with a Charcot fixator, and later - special shoes or orthoses. The average duration of casting for chronic CN with ulceration is 5 weeks, with transition to therapeutic shoes after 12 weeks. Some patients may require a cast for more than a year, and complications may include simple maceration of the skin [33-35].

Therefore, patients with chronic Charcot foot ulcers can be treated with TCC or removable walkers [23, 25]. In some studies, removable walkers have been successfully used to treat acute or chronic ulcerative NS. Removable walker orthoses are also effective in relieving pressure on the sole of the foot and treating diabetic foot ulcers. The low cost of removable walkers compared to multiple TCC replacements makes them a viable treatment option. Disadvantages of removable walker braces include the inability to accommodate severe deformities and potentially limited compliance [33–35].

Drug therapy of Charcot's foot. In NS, the available treatment options are based on the balance between bone resorption and bone formation [36]. There is good evidence that CN is associated with increased osteoclast activity, and antiresorptive therapy has been used with some success. Bisphosphonates and calcitonin have been used in the treatment of NS. Bisphosphonates can inhibit osteoclastic bone resorption, so they are usually used in the treatment of conditions characterized by abnormal metabolism, especially in the acute active phase of NS, and sometimes in the chronic phase [36-39]. Some patients with CN may not tolerate oral bisphosphonates but may benefit from intravenous pamidronate or zoledronic acid. The most commonly used is pamidronate, which acts on hydroxyapatite crystals in the newly synthesized bone matrix, blocking the access of osteoclast precursors to the matrix [39, 40]. Pitocco et al showed significant reductions in bone resorption markers with another bisphosphonate-alendronate and clinical improvement in foot NS at 6 months. Intranasal calcitonin is another antiresorptive agent that has been studied in the treatment of NS. This therapy has shown fewer complications compared to the use of bisphosphonates [36–40]. However, there is little evidence to guide the use of available pharmacological treatments for the treatment of CN. Most pharmacological treatments are still theoretical, with most studies evaluating only secondary clinical markers [20, 36, 37, 40–42].

Surgical treatment of Charcot's foot. Surgical treatment of NS foot and ankle joints primarily depends on the opinion of the doctor. Patient comorbidities, compliance with treatment, location and severity of the deformity, presence or absence of infection, pain, or instability are factors considered when deciding whether to undergo surgical treatment. In chronic NS, well-known surgical methods for realigning and stabilizing deformed diabetic Charcot feet include Achilles tendon lengthening, plantar osteotomy, bone debridement, leveling osteotomy, selective or extended arthrodesis, and open reduction with various forms of internal fixation with or without external fixation. There have been no comparative studies on the surgical choice of CN. [43].

Recent studies have suggested the benefits of earlier surgical correction of the deformity and arthrodesis, based on the assumption that surgical stabilization will improve the patient's quality of life [43]. Recurrent ulcers, joint instability, pain, associated displacement, severe exostosis, and potential skin complications due to inability to fixate the foot or missing foot are the most important indications for surgery in chronic NS. Due to the increased risk of wound infection and mechanical failure of fixation, surgical intervention should be avoided at the stage of active inflammation. A series of studies by Mittlmeier et al showed the benefits of early deformity correction in combination with arthrodesis [44]. Early surgical series showed improvement in restoring the plantigrade foot and preventing recurrence of ulceration, although nonunion, failure, and loss of initial correction were common. The number of patients with NS who undergo minor or major surgery varies from 14 [45] to 51% [46, 47]. According to a study by Salzman et al [48], the rate of lower extremity amputation ranged from 3 to 9%, primarily depending on the prevention of ulceration.

Thus, in the era of evidence-based medicine, NS of the foot and ankle remains a poorly understood disease, although recent clinical and basic science research has increased our knowledge regarding its etiology and treatment. NS is a serious complication of diabetes mellitus. It often appears suddenly and can quickly develop into severe and permanent foot deformity, leading to ulceration and amputation. An uncontrolled cycle of inflammation leads to destruction of the foot and ankle and severe deformities. Unloading is the most important initial treatment recommendation. Most patients with NS can be treated with immobilization and protected weight bearing. The use of TCC is the preferred method of non-surgical treatment. The basis of treatment for Charcot neuroarthropathy of the ankle joint is long-term immobilization in the form of a plaster cast or corset, as well as the use of antiresorptive drugs in the acute stage. However, some patients already have a disabling deformity or severe instability at the time of presentation for whom conservative treatment is doomed to failure. Although conservative treatment using TCC followed by appropriate bracing and footwear is considered the gold standard treatment for NS, surgical treatment is necessary when conservative treatment is ineffective. Surgical treatment is indicated for chronic recurrent ulcers, intractable deformities, acute fractures, dislocations or infections. Surgery includes exostectomy, Achilles tendon lengthening, and arthrodesis. Arthrodesis can be performed with internal fixation, or combined with external fixation simultaneously or with external fixation alone. Additional osteotomies may also be used. Regardless of the treatment pathway chosen, all treatment protocols must be adjusted to suit the patient based on lower extremity pathology, general health, and ability to comply with treatment.

Conclusions:

1) Early identification and prevention of collapse continue to be the best treatment options for patients with diabetic Charcot arthropathy of the ankle.

2) In patients with diabetes and lower extremity neuropathy, any minor injury requires careful monitoring due to the developmental tendency of the limb to undergo a Charcot process, so appropriate education, improved clinical assessment and early intervention are necessary to control the disease.

3) In the presence of collapse in the acute stage, the use of unloading TSS and antiresorptive drugs is recommended. In the following stages, foot and ankle preservation procedures with external fixation are valuable options that should be tried before deciding to amputate unless there is significant vascular compromise or uncontrolled infection.

REFRENCES

1. van der Ven A, Chapman CB, Bowker JH. Charcot neuroarthropathy of the foot and ankle. J Am Acad Orthop Surg. 2009;17:562–71. [PubMed] [Google Scholar]
2. Armstrong DG, Peters EJ. Charcot's arthropathy of the foot. //J Am Podiatr Med Assoc. 2002;92:390–4.

3. Sanders LJ, Fryberg RG. The Charcot foot. In: Bowker JH, Pfeifer MA, editors. Levin and O'Neal's the diabetic foot. 7th edn. Philadelphia: //Mosby Elsevier; 2007. pp. 257–83
4. Miller DS, Lichtman WF. Diabetic neuropathic arthropathy of feet; summary report of seventeen cases. //AMA Arch Surg. 1955;70:513–18.
5. Sinha S, Munichoodappa CS, Kozak GP. Neuro-arthropathy (Charcot joints) in diabetes mellitus (clinical study of 101cases) //Medicine. 1972;51:191–210.
6. Klenerman L. The Charcot joint in diabetes. //Diabet Med. 1996;13(Suppl 1):52–4
7. Nielson DL, Armstrong DG. The natural history of Charcot's neuroarthropathy. //Clin Podiatr Med Surg. 2008;25:53–62.
8. Pakariennen TK, Laine HJ, Honkonen SE, Peltonen J, Oksala H, Lahtela J. Charcot arthropathy of the diabetic foot. Current concepts and review of 36 cases. // Scand J Surg. 2002;91:196–201.
9. Chantelau E. The peris of procalcitonin: effects of early vs. delayed detection and treatment of incipient Charcot fracture. //Diabet Med. 2005;22:1707–12.
10. Perrin BM, Gardner MJ, Suhaimi A, Murphy D. Charcot osteoarthropathy of the foot. //Aust Fam Physician. 2010;39:117–19.
11. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation. Basis for preventation. //Diabetes Care. 1990;13:513–21
12. Reiber GE, Vileikyte L, Boyko EJ. Causal pathways for incident lower extremity ulcers in diabetes from the two settings. //Diabetes Care. 1999;22:157–62.
13. Tan AL, Greenstein A, Jarrett SR, McGonagle D. Acute neuropathic joint disease: medical emergancy? //Diabetes Care. 2005;28:2962–4.
14. Eloesser L. On the nature of neuropathic affections of the joints. //Ann Surg. 1917;66:201–7
15. Salo PT, Theriault E, Wiley RG. Selective ablation of rat knee joint innervation with injected immunotoxin: a potential new model for the study of neuropathic arthritis. //J Orthop Res. 1997;15:622–8.
16. Eichenholtz SN. Charcot joints. Springfield, IL: //Charles C. Thomas; 1966. pp. 3–8.
17. Shibata T, Tada K, Hashizume C. The results of arthrodesis of the ankle for leprotic neuroarthropathy. // J Bone Joint Surg Am. 1990;72:749–56.
18. Morrison WB, Ledermann HP. Work-up of the diabetic foot. Radiol Clin North Am. 2002;40:1171–92.
19. Morrison WB, Ledermann HP, Schweitzer ME. MR imaging of the diabetic foot. //Magn Reson Imaging Clin N Am. 2001;9:603–13
20. Petrova NL, Foster AV, Edmonds ME. Difference in presentation of Charcot osteoarthropathy in type 1 compared with type 2 diabetes. // Diabetes Care. 2004;27:1235–6.
21. Ramanujam CL, Facaros Z. An overview of conservative treatment options for diabetic Charcot foot neuroarthropathy. // Diabet Foot Ankle. 2011;2:6418
22. Pinzur MS, Lio T, Posner M. Treatment of Eichenholtz stage I Charcot foot arthropathy with a weightbearing total contact cast. //Foot Ankle Int. 2006;27:324–9
23. Trepman E, Nihal A, Pinzur MS. Current topics review: Charcot neuroarthropathy of the foot and ankle//. Foot Ankle Int. 2005;26:46–63
24. Brodsky JW, Rouse AM. Exostectomy for symptomatic bony prominences in diabetic Charcot feet. //Clin Orthop Relat Res. 1993;296:21–6.

25. Rogers LC, Frykberg RG, Armstrong DG, Boulton AJ, Edmonds M, Van GH, et al. The Charcot foot in diabetes. //Diabetes Care. 2011;34:2123–9

26. Jirkovská A, Kasalický P, Boucek P, Hosová J, Skibová J. Calcaneal ultrasonometry in patients with Charcot osteoarthropathy and its relationship with densitometry in the lumbar spine and femoral neck and with markers of bone turnover. //Diabet Med. 2001;18:495–500.

27. Young MJ, Marshall A, Adams JE, Selby PL, Boulton AJ. Osteopenia, neurological dysfunction, and the development of Charcot neuroarthropathy. //Diabetes Care. 1995;18:34–8.

28. Jeffcoate WJ, Game F, Cavanagh PR. The role of proinflammatory cytokines in the cause of neuropathic osteoarthropathy (acute Charcot foot) in diabetes. // Lancet. 2005;366:2058–61

29. Stuck RM, Soh MW, Budiman-Mak E, Lee TA, Weiss KB. Charcot arthropathy risk elevation in the obese diabetic population. //Am J Med. 2008;121:1008–14.

30. Wukich DK, Sung W. Charcot arthropathy of the foot and ankle: modern concepts and management review. //J Diabetes Complications. 2009;23:409–26.

31. Verity S, Sochocki M, Embil JM, Trepman E. Treatment of Charcot foot and ankle with prefabricated walker brace and custom insole. //Foot Ankle Surg. 2008;14:26–31.

32. McGill M, Molyneaux L, Bolton T, Ioannou K, Uren R, Yue DK. Response of Charcot's arthropathy to contact casting: assessment by quantitative techniques. //Diabetologia. 2000;43:481–4.

33. Sinacore DR. Healing times of diabetic foot ulcers in the presences of fixed deformities of the foot using total contact casting. //Foot Ankle Int. 1998;19:613–8

34. Armstrong DG, Lavery LA, Wu S. Evaluation of removable and irremovable cast walkers in the healing of diabetic foot wounds: a randomized controled trial. //Diabetic Care. 2008;28:551–4

35. Armstrong DG, Short B, Espensen EH. Technique for fabrication for an instant total contact cast for treatment of neuropathic diabetic foot ulcers. //J Am Podiatr Med Assoc. 2002;92:405–8.

36. Selby PL, Young MJ, Boult AJ. Bisphosphonates: a new treatment for diabetic Charcot neuroarthropathy? // Diabet Med. 1994;11:28–31.

37. Rogers MJ. New insights into the molecular mechanisms of action of bisphosphonates. // Curr Pharm Des. 2003;9:2643–59.

38. Jude EB, Selby PL, Burgess J, Lilleystone P, Mawer EB, Page SR. Bisphosphonates in the treatment of Charcot neuroarthropathy: double blinded randomised controlled trial. //Diabetologia. 2001;44:2032–7.

39. Pitocco D, Ruotolo V, Caputo S, Mancini L, Collina CM, Manto A. Six month treatment with alendronate in acute Charcot neuroarthropathy: a randomized controlled trial. //Diabetes Care. 2005;28:1214–5.

40. Bern R, Jirkovska A, Fejfarova V, Skibova J, Jude EB. Intranasal calcitonin in the treatment of acute Charcot neuroosteoarthropathy: a randomized controlled trial. //Diabetes Care. 2006;29:1392–5.

41. Edelson GW, Jensen KL, Kaczynski R. Identifying acute Charcot arthropathy through urinary crosslinked N-telopeptides. //Diabetes. 1996;45(Suppl 2):108.

42. Selby PL, Jude EB, Burgess J. Bone turnover markers in acute Charcot neuroarthropathy. //Diabetologia. 1999;41(Suppl 1):275.

43. Dhawan V, Spratt KF, Pinzur MS, Baumhauer J, Rudicel S, Saltzman CL. Reliability of AOFAS diabetic foot questionnaire in Charcot arthropathy: stability, internal consistency, and measurable difference. // Foot Ankle Int. 2005;26:717–31.

44. Mittlmeier T, Klaue K, Haar P, Beck M. Should one consider primary surgical reconstruction in charcot arthropathy of the feet? //Clin Orthop Relat Res. 2010;468:1002–11.

45. Farber DC, Juliano PJ, Cvanagh PR, Ulbrecht J, Caputo G. Single stage correction with external fixation of the ulcerated foot in individuals with Charcot neuroarthropathy. // Foot Ankle Int. 2002;23:130–4.

46. Pinzur MS. Neutral ring fixation for high-risk nonplantigrade Charcot midfoot deformity. //Foot Ankle Int. 2007;26:961–6.

47. Pinzur MS, Sage R, Stuck R, Kaminsky S, Zmuda A. A treatment algorithm for neuropathic (Charcot) midfoot deformity. //Foot Ankle Int. 1993;14:189–97

48. Saltzman CL, Hagy ML, Zimmerman B, Estin M, Cooper R. How effective is intensive nonoperative initial treatment of patients with diabetes and Charcot arthropathy of the feet? //Clin Orthop Relat Res. 2005;435:185–90