JKFAS Journal of Korean Foot and Ankle Society
Case Report
pISSN 1738-3757 eISSN 2288-8551
J Korean Foot Ankle Soc 2026;30(1):38-42
https://doi.org/10.14193/jkfas.2026.30.1.38

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중족부 중증 샤르코 신경병증에서 초구조(superconstruct) 기법과 데노수맙 병용을 이용한 성공적인 재정렬 관절 유합술: 증례 보고

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Successful Realignment Arthrodesis using a Superconstruct Technique and Adjuvant Denosumab in Severe Midfoot Charcot Neuroarthropathy: A Case Report

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Severe midfoot Charcot neuroarthropathy (CN) presents a significant surgical challenge because of extensive bone loss and poor bone quality. This report presents the outcome of a case treated with realignment arthrodesis using a superconstruct technique, supplemented by the postoperative use of denosumab. A patient with Eichenholtz Stage III CN (Brodsky Type 1 and 2) underwent a single-stage surgical reconstruction. Postoperatively, denosumab was administered to enhance bone stability. The 1-year and 1-month follow-up showed that the severe deformity had been successfully corrected to a stable, plantigrade foot, confirmed by radiographic and clinical evaluation. This case suggests that a combined surgical approach using a superconstruct with adjuvant denosumab can be an effective treatment for severe midfoot CN.

Key Words: Joint disease, Arthrodesis, Foot, Diabetic neuropathies, Denosumab

Received November 3, 2025 **Revised** November 11, 2025
Accepted November 12, 2025
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Financial support: None.
Conflict of interest: Yeo Kwon Yoon and Dong Woo Shim are reviewers, and Seung Hwan Han and Kwang Hwan Park are editorial members of Journal of Korean Foot and Ankle Society but they were not involved in the review process of this manuscript. Except for that, no potential conflict of interest relevant to this article was reported.

Charcot neuroarthropathy (CN) is characterized by a local inflammatory cascade with elevated cytokines (e.g., TNF-α, IL-1β, IL-6) and up-regulation of the RANKL-RANK-OPG pathway, which drive excessive osteoclastogenesis and accelerated bone resorption triggered by peripheral neuropathy and repetitive micro-trauma.[1-3] As a result, CN in patients with diabetic neuropathy is a progressive and destructive joint disease leading to severe deformity, instability, and significant functional impairment.[1] Midfoot involvement frequently results in a "rocker-bottom" deformity from the collapse of the longitudinal arch, which complicates bracing and often leads to recurrent ulceration and infection.[4] For patients with severe deformities refractory to conservative treatment, the primary goal of surgery is to establish a stable, plantigrade, and braceable foot, thereby restoring functional

Figure 1. Initial plain right foot radiographs. (A) The anteroposterior view shows severe destruction of the Lisfranc and naviculocuneiform joint. (B) The lateral view demonstrates collapse of medial longitudinal arch, resulting in a rocker-bottom deformity.

ambulation.[5] Surgical management is exceptionally challenging due to poor bone quality, extensive bone loss, and compromised soft tissues.[6] The concept of a "superconstruct," involving rigid internal fixation that spans the zone of injury to engage healthier bone, has become a standard approach.[7] However, risks of fixation failure and nonunion remain significant.

Because the pathophysiology involves unchecked bone destruction, it provides a rationale for using additional anti-resorptive therapy such as denosumab, a monoclonal antibody that inhibits RANKL and thereby blunts osteoclast activity. Early pilot studies in CN suggest denosumab can reduce bone and joint destruction, supporting its potential as adjunctive treatment in this challenging condition.[8,9]

This paper reports a case of severe midfoot CN treated with extensive realignment arthrodesis and supplemented with denosumab postoperatively, which achieved a satisfactory clinical and radiographic outcome.

CASE REPORT

This study was approved by the Institutional Review Board of Yonsei University Health System, Severance Hospital (IRB No.4-2024-1498). A 57-year-old female presented with a 1-year history of progressive deformity, swelling, and limping in her right foot. She had been diagnosed with diabetes mellitus 5 years prior and reported no history of significant trauma. A physical examination revealed a collapsed arch and significant midfoot swelling, but an absence of significant dependent erythema or localized heat.

Figure 2. Preoperative computed tomography images of right foot. (A) Coronal view. (B) Axial view. (C) Saggital view. The extensive bony destruction, fragmentation, and multiplanar deformity of the midfoot were observed.

Initial plain radiographs revealed severe destruction of the Lisfranc and naviculocuneiform joints, with a resulting collapse of the medial longitudinal arch creating a classic rocker-bottom deformity (Fig. 1). A subsequent computed tomography (CT) scan was performed to better define the anatomy, which confirmed extensive bony destruction, fragmentation, and severe multiplanar deformity throughout the midfoot (Fig. 2). Magnetic resonance imaging revealed extensive bone marrow edema and joint destruction; however, the possibility of acute osteomyelitis was considered unlikely based on the patient's clinical presentation (Fig. 3). Laboratory tests, including C-reactive protein and bone turnover markers including bone alkaline phosphatase, osteocalcin, procollagen 1N-terminal, b-crosslaps, and deoxypyridinoline were within normal limits, and a bone mineral density scan showed osteopenia (femur -2.1, spine -2.2). The patient was diagnosed with midfoot CN, classified as Brodsky Types 1 and 2, and in

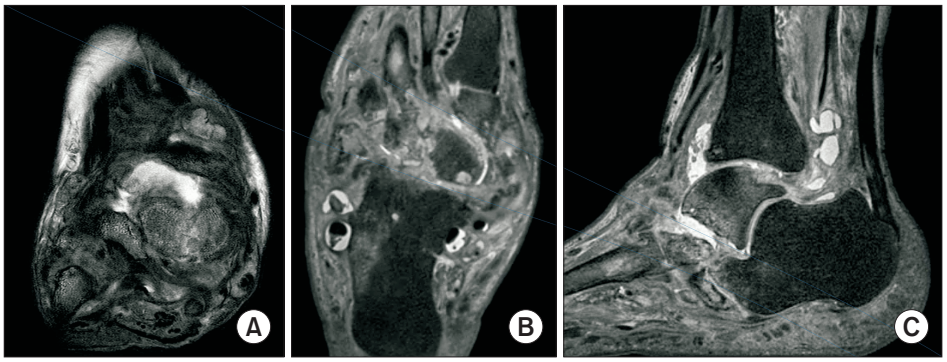

Figure 3. Preoperative T2-weighted magnetic resonance imaging showing bone marrow edema and joint destruction. Fluid collection and edematous change were also observed.

a chronic, reconstructive phase corresponding to Eichenholtz Stage III. Given the severe, unbraceable deformity and the patient's desire for improved ambulation, a single-stage surgical reconstruction was planned.[8]

The surgical procedure involved an extensive midfoot bone resection (cuneiforms, navicular, cuboid, and talar head) via both medial and lateral approach to shorten the foot and allow for realignment without excessive soft-tissue tension. An open Achilles tendon tenotomy was performed to correct the equinus contracture. A subtalar arthrodesis was performed using two 6.5-mm cannulated screws. Subtalar joint arthrodesis is incorporated in Charcot midfoot reconstruction to enhance hindfoot stability by limiting pronation-supination motion, thereby creating a more rigid structure and reducing postoperative complications and progression to hindfoot collapse.[6,9] The resulting bone defect in Chopart joint was filled with an allogenous femoral head strut bone graft, and a "superconstruct" was created using a beaming screw from the first metatarsal head to the talus, augmented with a medial locking plate. A second beaming screw was placed from the fifth metatarsal base to the calcaneus to stabilize the lateral column.

Postoperative radiographs confirmed satisfactory alignment (Fig. 4). The rehabilitation protocol began with a short leg splint for the first 3 weeks to manage postoperative swelling and for wound management, during which the patient remained strictly non-weightbearing. The patient was allowed to perform only active ankle and toe range of motion exercises during this period. At 3 weeks postoperatively, she was transitioned to a short leg walking cast and initiated partial weightbearing. Weightbearing was gradually advanced as tolerated. The cast was changed at 7 weeks and again at 10 weeks. The cast was removed at 14 weeks postoperatively,

Figure 4. Immediate postoperative plain radiographs. (A) Anteroposterior view. (B) Lateral view. Alignment correction is satisfactory and screw positions are well maintained.

after which she transitioned to wearing a Charcot restraint orthotic walker boot for outdoor ambulation and an ankle brace while indoors. She was subsequently fitted for custom-molded footwear at 7 months postoperatively.

To enhance bone stability, the patient received subcutaneous injections of denosumab (60 mg) postoperatively and again at 6 months in an off-label manner. At the 13 months follow-up, radiographs demonstrated a well-maintained correction with signs of bony consolidation (Fig. 5). Postoperative clinical photographs also showed a well-corrected plantigrade foot shape (Fig. 6). The patient was ambulating comfortably in custom-molded footwear and reported significant functional improvement and resolution of her preoperative pain.

Figure 5. Plain foot radiographs at 13 months postoperatively.

Figure 6. Right foot gross photos at 13 months postoperatively. A stable and plantigrade foot is maintained without evidence of ulceration or recurrent deformity.

DISCUSSION

The surgical reconstruction of severe CN aims to create a stable, plantigrade foot rather than restore normal anatomy.[10] In cases of severe deformity with bone loss, less rigid fixation methods, such as screw-only constructs or simple dorsal plating, often fail to provide adequate stability to resist the substantial mechanical forces and underlying poor bone quality, leading to a high rate of hardware failure and loss of correction. The "superconstruct" approach, as utilized in this case, is therefore essential. By spanning the entire midfoot with long, large-diameter beaming screws and a rigid locking plate, fixation is achieved in healthier bone proximal and distal to the area of destruction, providing a durable structure that maintains alignment during the healing process.[10]

A novel aspect of our treatment strategy was the adjuvant use of denosumab. The pathophysiology of CN is driven by a local inflammatory cascade that up-regulates the RANKL-RANK-OPG pathway, leading to excessive osteoclastogenesis and the rapid, pathological bone destruction that defines the disease.[3] Denosumab, a monoclonal antibody that inhibits RANKL, directly and potently suppresses this osteoclast-mediated resorption.[3] In the context of a surgical reconstruction, this underlying cellular activity can persist, threatening the surgical outcome by causing nonunion, allograft resorption, or hardware failure. The rationale for adjuvant denosumab is to create a more favorable biological environment for fusion by rapidly inducing disease quiescence. This approach is supported by emerging clinical evidence. Shofler et al.[9] demonstrated that a single 60 mg denosumab injection was well-tolerated and appeared to accelerate the transition from the acute inflammatory phase, with patients achieving skin temperature normalization (a marker of quiescence) at an average of 52 days. Furthermore, Carvès et al.[6] studied patients with refractory active CN and found that denosumab treatment led to clinical improvement in all subjects, a significant decrease in metabolic activity on ^{18}F-Fluorodeoxyglucose Positron Emission Tomography-CT scans, and stabilization of structural damage on radiographs. Although the literature is still based on small cohorts, the data is encouraging. Our strategy was to combine rigid mechanical stabilization with this potent biological modulation. The stable maintenance of alignment and clear signs of bony consolidation at 13 months in our patient suggest a potential synergistic benefit of this combined approach, which may be particularly valuable in severe, high-risk reconstructive cases.

This case report has two primary limitations. First, while the patient showed excellent progress at the 13 months fol-

low-up, this is a relatively short-term period for evaluating a Charcot reconstruction. A longer-term follow-up is necessary to definitively confirm the durability of the arthrodesis and to monitor for the longevity of the "superconstruct" and late-stage complications, such as stress fractures or the transfer of disease to adjacent joints.[11] Second, our assessment of the surgical outcome relied on radiographic evidence and subjective patient-reported function. The absence of objective data, such as pre- and postoperative plantar pressure analysis, quantitative gait studies or functional outcomes, limits our ability to fully characterize the functional improvements in foot biomechanics.

In conclusion, extensive midfoot resection and realignment arthrodesis using a rigid superconstruct of plates and beaming screws is an effective technique for managing severe Charcot midfoot deformities. The adjuvant use of an anti-resorptive agent like denosumab represents a theoretically sound strategy to enhance bone stability and may support successful surgical outcomes in this challenging patient population.

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REFERENCES

1. **Johnson-Lynn SE, McCaskie AW, Coll AP, Robinson AHN.** *Neuroarthropathy in diabetes: pathogenesis of Charcot arthropathy. Bone Joint Res. 2018;7:373-8. doi: 10.1302/2046-3758.75.BJR-2017-0334.R1.*
2. **Larson SA, Burns PR.** *The pathogenesis of Charcot neuroarthropathy: current concepts. Diabet Foot Ankle. 2012;3. doi: 10.3402/dfa.v3i0.12236.*
3. **Greco T, Mascio A, Comisi C, Polichetti C, Caravelli S, Mosca M, et al.** *RANKL-RANK-OPG pathway in Charcot diabetic foot: pathophysiology and clinical-therapeutic implications. Int J Mol Sci. 2023;24:3014. doi: 10.3390/ijms24033014.*
4. **Trieb K.** *The Charcot foot: pathophysiology, diagnosis and classification. Bone Joint J. 2016;98:1155-9. doi: 10.1302/0301-620X.98B9.37038.*
5. **Wukich DK, Sung W.** *Charcot arthropathy of the foot and ankle: modern concepts and management review. J Diabetes Complications. 2009;23:409-26. doi: 10.1016/j.jdiacomp.2008.09.004.*
6. **Carvès S, Bourgeon-Ghittori M, Henry J, Belkhir R, Besson FL, Levante S, et al.** *Denosumab in active Charcot neuro-osteoarthropathy of the foot. Joint Bone Spine. 2021;88:105241. doi: 10.1016/j.jbspin.2021.105241.*
7. **Manchanda K, Wallace SB, Ahn J, Nakonezny P, Liu GT, Raspovic KM, et al.** *Charcot midfoot reconstruction: does subtalar arthrodesis or medial column fixation improve outcomes? J Foot Ankle Surg. 2020;59:1219-23. doi: 10.1053/j.jfas.2020.07.001.*
8. **Mateen S, Thomas MA, Jappar A, Wu S, Meyr AJ, Siddiqui NA.** *Progression to hindfoot Charcot neuroarthropathy after midfoot Charcot correction in patients with and without subtalar joint arthrodesis. J Foot Ankle Surg. 2023;62:731-6. doi: 10.1053/j.jfas.2023.03.004.*
9. **Shofler D, Hamedani E, Seun J, Sathananthan A, Katsaros E, Liggan L, et al.** *Investigating the use of denosumab in the treatment of acute Charcot neuroarthropathy. J Foot Ankle Surg. 2021;60:354-7. doi: 10.1053/j.jfas.2020.09.018.*
10. **Ford SE, Cohen BE, Davis WH, Jones CP.** *Clinical outcomes and complications of midfoot Charcot reconstruction with intramedullary beaming. Foot Ankle Int. 2019;40:18-23. doi: 10.1177/1071100718799966.*
11. **Schade VL, Andersen CA.** *A literature-based guide to the conservative and surgical management of the acute Charcot foot and ankle. Diabet Foot Ankle. 2015;6:26627. doi: 10.3402/dfa.v6.26627.*