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Light Bulb Procedure for the Treatment of Tarsal Navicular Osteonecrosis After Failed Percutaneous Decompression: A Case Report



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ABSTRACT

Tarsal navicular osteonecrosis in adults is a rare condition with unclear etiology, and the optimal treatment has not been established. Here we report a case of tarsal navicular osteonecrosis with a complete course of treatment and comprehensive imaging studies starting at an early stage. A 37-year-old female diagnosed with tarsal navicular osteonecrosis was first treated with percutaneous decompression, but her symptoms persisted postoperatively. The tarsal navicular showed no further collapse, but follow-up magnetic resonance imaging (MRI) at 6 months postoperatively revealed persistent osteonecrotic changes. Debridement of the necrotic bone with preservation of the cortical shell and bone substitute packing for the defect (light bulb procedure) were performed. The symptoms resolved by 3 months postoperatively, and the patient could return to work. At a 6-year follow-up visit, the patient was free of symptoms, and MRI showed remodeling of the tarsal navicular without further collapse.

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Adult-onset tarsal navicular osteonecrosis is a rare disease. Its etiology is unclear, and the optimal treatment has not been established. Most studies reported to date have indicated that core decompression is a safe and effective procedure for treating osteonecrosis of the femoral head. More recent studies have reported better results with innovative multiple percutaneous drilling techniques at the earliest, precollapse disease stages (1–3). Percutaneous drilling and core decompression are also used in early-stage osteonecrosis of the ankle, with promising outcomes (4,5). In 2011, Janositz et al (6) reported a case of tarsal navicular osteonecrosis successfully treated at an early stage with percutaneous decompression.

The light bulb procedure for osteonecrosis of the femoral head was first described by Rosenwasser et al (7) in 1994. It is a head-preserving procedure with thorough debridement and bone grafting through a window at the junction of the femoral head and neck. After thorough debridement of sclerotic necrotic bone, light is able to transilluminate through the femoral head resembling a light bulb. Tosun et al (8) reported a similar method for treating a patient with spontaneous tarsal navicular osteonecrosis.

In this report, we present a case of tarsal navicular osteonecrosis initially treated with percutaneous decompression, with a subsequent

second operation using a light bulb procedure owing to the failure of the first operation to relieve symptoms. The final result was satisfactory. The treatment course and serial imaging changes are described.

Case Report

A 37-year-old female presented at our clinic with chronic left ankle and midfoot pain of 6 months duration. Osteochondral lesion of the talus (OLT) and tarsal navicular osteonecrosis were diagnosed by magnetic resonance imaging (MRI) (Fig. 1). The patient received conservative treatment with cast immobilization for 3 months. However, she had persistent pain and disability, and her American Orthopaedic Foot and Ankle Society (AOFAS) midfoot scale score was 42 (9,10).

Arthroscopic surgery for OLT with removal of osteochondral fragment, transsalleolar multiple drilling, and percutaneous decompression for tarsal navicular osteonecrosis were performed. The patient received cast immobilization for 2 months, after which weightbearing was started gradually. Her ankle pain improved, but pain in the midfoot and hindfoot persisted (AOFAS midfoot scale score, 39). Follow-up MRI at 6 months postoperatively revealed persistence of osteonecrosis without collapse, and computed tomography (CT) scan showed sclerotic changes and loss of trabecular structure of the navicular bone (Fig. 2).

We performed the light bulb procedure, an operation used in early stages of osteonecrosis of the femoral head, after failed percutaneous decompression. This second operation was performed using a dorsal approach between the extensor hallucis longus and tibialis anterior tendons, with removal of necrotic bone through a bone window.

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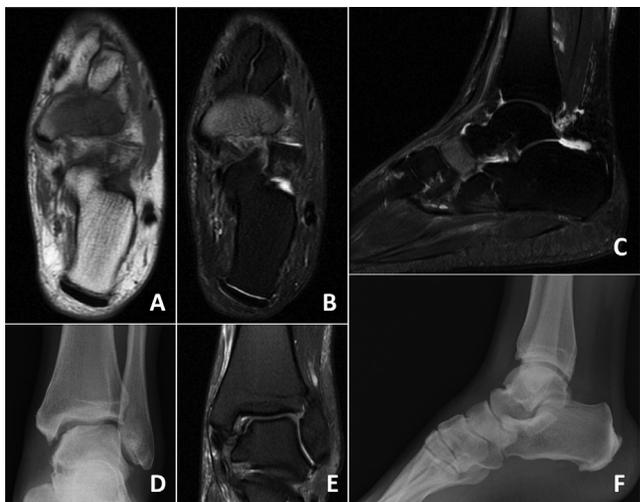


Fig. 1. Initial radiographs and magnetic resonance imaging (MRI) of the ankle and foot at presentation. (A–C) Low T1 signal and high T2 signal of the tarsal navicular on MRI. (D and E) Osteochondral lesion of the talus on radiography (D) and MRI (E). (F) Radiography revealed no collapse or sclerosis of the tarsal navicular.

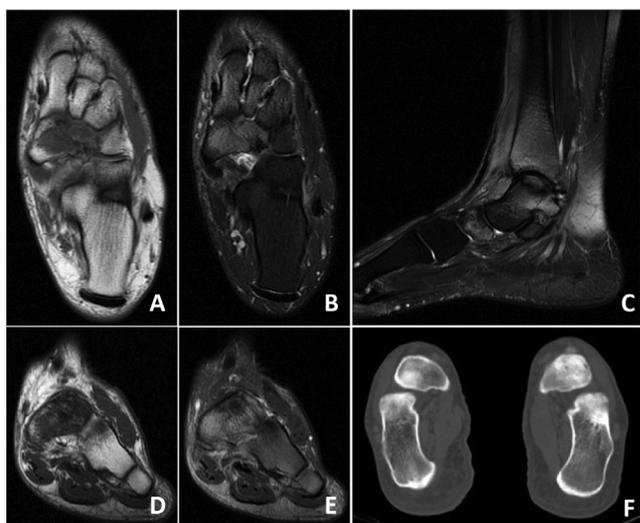


Fig. 2. Imaging studies at 6 months after percutaneous decompression. (A–E) Persistent marrow edema over the navicular bone. (F) Computed tomography scan showing sclerosis and loss of left navicular trabecular structure.

Sclerotic changes in the affected bone were observed intraoperatively and were completely debrided with a high-speed burr. Ample saline was used for cooling, and perforation of the navicular was avoided.

The depth of debridement was checked with fluoroscopy and transillumination with the arthroscopic light source over the medial aspect of the navicular. The pneumatic tourniquet was released, and bleeding points over the inner surface of the bone confirmed the adequacy of debridement. The bony defect was filled with bone substitute (calcium sulfate, OSTEOSET® Pellets; Wright, Memphis, TN), and the bone window was closed using the original cortical bone without internal fixation (Fig. 3).

Microscopically, the debrided tissue showed fragmented necrotic bone with discernable focal new bone formation. Hematoxylin and eosin staining revealed fat necrosis (i.e., lipid-filled spaces and lack of nuclei), pyknosis (i.e., condensation of chromatin in the nucleus), and karyorrhexis (i.e., fragmentation of the nucleus) in bone marrow (Fig. 4).

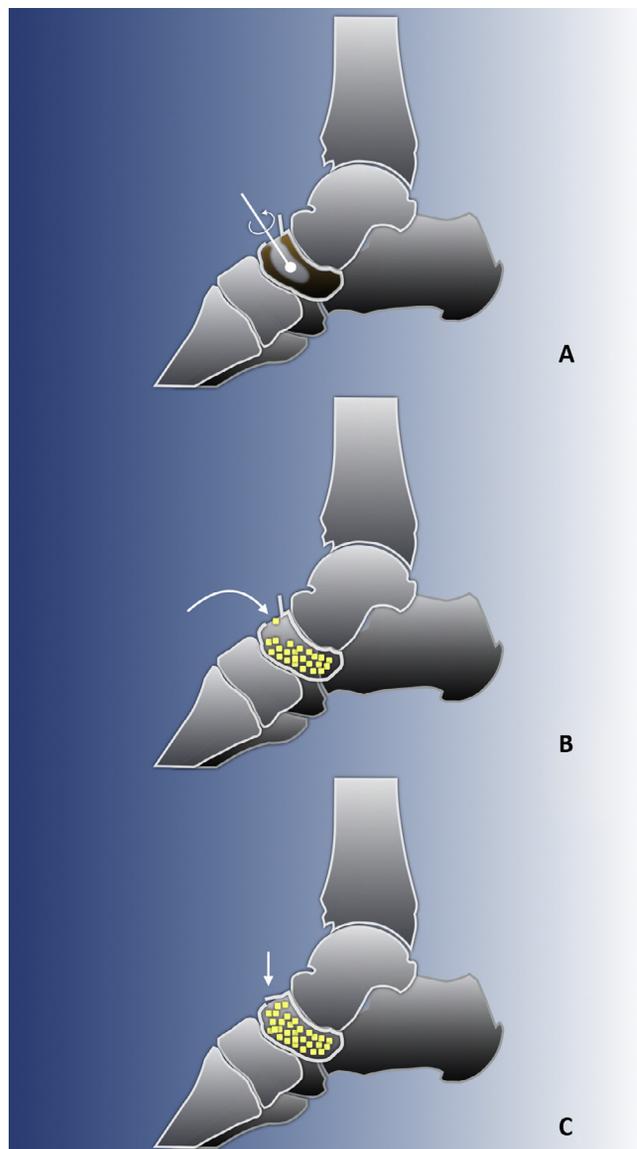


Fig. 3. The light bulb procedure in tarsal navicular osteonecrosis. (A) The necrotic area was debrided with a high-speed burr through a bone window. (B) The cortical shell was filled with bone substitute. (C) The bone window was closed with the original cortical bone.

Postoperatively, the patient received cast immobilization for 2 months, and weightbearing of the affected leg was encouraged thereafter. Her symptoms resolved, and she returned to her daily work at 3 months postoperatively (AOFAS midfoot scale score, 72). MRI at 3 months postoperatively showed signal changes in previously necrotic areas (Fig. 5).

Follow-up MRI at 6 years postoperatively showed remodeling of the navicular with small areas of low T1 signal (Fig. 6). The AOFAS midfoot scale score was 97, and radiography showed low-grade perinavicular arthritis without further collapse or deformity (Fig. 7).

Discussion

Tarsal navicular osteonecrosis in adults is rare and complex. It can be secondary to trauma or idiopathic, and the affected area can be partial or total.

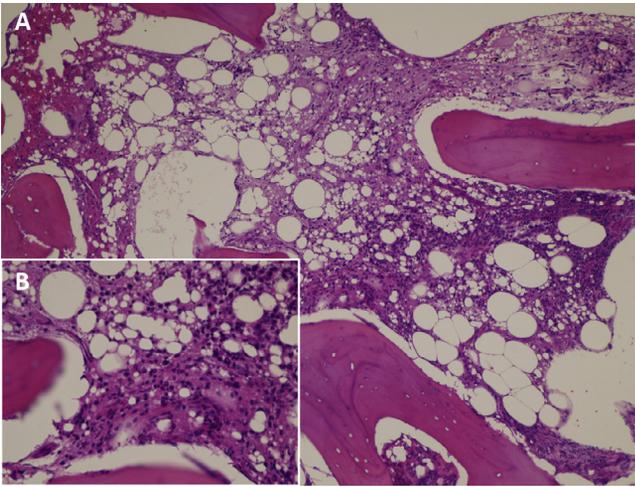


Fig. 4. Hematoxylin and eosin staining of the operative specimen showing fat necrosis, pyknosis, and karyorrhexis in bone marrow. (A) Original magnification, 100 \times . (B) High-power magnification, 400 \times .



Fig. 5. Imaging studies at 3 months after the light bulb procedure. (A–E) Signal changes in the previously necrotic area. (F) Remodeling of osteochondral lesion of the talus and linear marrow edema of a previous drilling track over medial malleolus.

Müller-Weiss disease presents with chronic midfoot and hindfoot pain and is characterized by navicular collapse and fragmentation on radiography. Müller and Weiss independently described this condition in 1927, and the disease was named after both authors (11,12). Maceira and Rochera (13) proposed a combination of delayed ossification of the tarsal navicular and abnormal force distribution as the primary causative factors of Müller-Weiss disease. Numerous other etiologic factors have been proposed, including osteonecrosis, trauma, congenital dysplasia, and a normal variant or migration of an accessory cuboid (12,14–19). Although whether Müller-Weiss disease is caused by osteonecrosis is controversial, most of the literature related to tarsal navicular osteonecrosis actually discusses cases of late-stage Müller-Weiss disease (13,20,21).

Maceira et al (13) classified Müller-Weiss disease into 5 stages based on radiographic findings. In stages 1 and 2, there are minimal changes in the navicular or dorsal subluxation of the talar head. In stages 3 to 5, radiographic findings may include navicular



Fig. 6. Imaging studies at 6 years after the light bulb procedure showing remodeling of the navicular with small areas of low T1 signal.

compression or fragmentation, and a comma-shaped navicular bone due to lateral collapse and reduction of space between the talar head and the cuneiform. There is no deformity or navicular collapse in stages 1 and 2, which are usually defined as early stages. Maceira stages 3 to 5 are usually defined as advanced stages. The Maceira staging system is based on image findings, but the clinical presentation is not consistently correlated with the severity of navicular deformity.

CT scans may precisely define the site and extent of collapse or fragmentation. MRI is indicated in strongly suspected cases without radiographic changes. It may show intraosseous edema (marked by high signal intensity on T2-weighted MRI and low signal intensity on T1-weighted MRI) in the early stages of osteonecrosis (13,22). It also serves as an important tool for evaluating operative outcomes and remodeling of the navicular. The use of dual-energy CT (or spectral CT) has been expanded to the musculoskeletal system. It allows the evaluation of bone marrow edema and detection of subchondral changes in the early stage. Spectral CT also provides accurate visualization of the morphology of the tarsal navicular and perinavicular joint surfaces (23–26).

For symptomatic patients with Müller-Weiss disease, treatment usually starts with conservative measures. Nonsteroidal antiinflammatory medication and cast immobilization may reduce pain. Foot orthosis with medial arch support and a valgus heel post may improve symptoms and function in most patients with Müller-Weiss disease (14,27–31). Operative treatment is indicated after failed conservative treatment. At present, there are no clear guidelines for treating tarsal navicular osteonecrosis or Müller-Weiss disease in all stages (32). Patients in advanced stages (Maceira stages 3 to 5) have been treated with talonavicular arthrodesis, talonavicular-cuneiform arthrodesis, or triple arthrodesis in most studies (15,16,18,27,29,30,33–36). Hetsronia et al (28) found significantly higher plantar pressures at the midfoot, along with reduced toe pressures, in patients with Müller-Weiss disease. Maceira et al (13) also found an abnormal force distribution on the lateral half of the navicular. This may be at least partially responsible for pain and discomfort, and hindfoot varus correction may play a key role in treating pain and dysfunction. Li et al (31) treated 14 feet of 13 patients with Müller-Weiss disease (5 feet in Maceira stage 2, 9 feet in advanced stages) with a lateral shift calcaneal osteotomy without other associated operative procedures and obtained satisfactory results. As in our patient, if symptoms persist but are relieved by a valgus heel



Fig. 7. Postoperative (PO) radiographs showing serial changes of the navicular and incorporation of the bone graft substitute.

post, calcaneal corrective osteotomy may be considered before proceeding to arthrodesis.

However, the operative treatment of early-stage tarsal navicular osteonecrosis without deformity has been described only in sporadic cases. Yu et al (37) reported a 15-case series of Müller-Weiss disease in which 1 patient with Maceira stage 2 disease was treated with arthrodesis. Arthrodesis of symptomatic tarsal joints can be a useful salvage procedure after failed percutaneous decompression or osteotomy (31). Yet this results in decreased tarsal joint mobility, compromising gait, and degenerative changes in the adjacent joints ensue (38–41). Janositz et al (6) reported a young athlete with early-stage Müller-Weiss disease successfully treated with percutaneous decompression. Tosun et al (8) reported an adult case of spontaneous osteonecrosis of the tarsal navicular with diffuse navicular sclerosis without evident collapse on radiography, erosion of cortex and cystic lesions on CT, and collapse, disorganization, and marrow edema of the navicular bone on MRI. The case was treated with removal of necrotic bone combined with autologous bone grafting. Radiography showed satisfactory incorporation of bone grafts without collapse 3 years postoperatively. The core decompression reported by Janositz et al (6) was performed percutaneously. The technique reported by Tosun et al (8) involved a dorsomedial approach and managed to evacuate the entire navicular content. In this case, we performed debridement of necrotic bone and packing of bone substitute under direct visualization through a limited dorsal approach, assisted by the light bulb technique.

The light bulb procedure, a treatment option for early-stage osteonecrosis of the femoral head, was first described by Rosenwasser et al (7) in 1994. A window was created at the junction of the femoral head and neck via a Watson-Jones or Smith-Petersen approach. All sclerotic bone lesions were debrided with drills, burrs, and curettes under an image intensifier. Autologous iliac bone graft was harvested and packed tightly into the femoral head through the window. The cortical window was replaced. In their report, 13 patients underwent the procedure, and the mean follow-up period was 12 years. Only 2 patients received revision to total hip replacement, and the others remained symptom-free with minimal hip osteoarthritis. Other studies also showed good to excellent results using the light bulb procedure to treat osteonecrosis of the femoral head (42–47).

It is simpler to perform the light bulb procedure for the tarsal navicular, a relatively superficial structure, than for the femoral head. The navicular blood supply comes mainly from the dorsalis pedis artery in the dorsolateral aspect and the medial plantar artery in the plantar medial aspect (32). Using the dorsal approach for the navicular could minimize violation of the feeding vessels. The adequacy of debridement could be evaluated under fluoroscopy or with transillumination of the navicular using arthroscopic light (Fig. 8). There was decreased transillumination in the sclerotic navicular relative to the adjacent tarsal bones, followed by an increase in transillumination after thorough debridement.

Various types of bone grafts and bone substitutes, including autologous iliac bone grafts, allografts, human bone morphogenetic protein-2 with calcium phosphosilicate, and calcium sulfate with calcium phosphate, have been used in core decompression or associated operations for osteonecrosis with favorable outcomes (7,42,45,48). Bone grafting remains the treatment of choice because it provides osteoinduction, osteoconduction, and osteogenesis (49). Synthetic bone substitutes, including calcium phosphate, calcium sulfate, hydroxyapatite, and bioactive ceramics, provide osteoconductive support with limited osteoinductive ability (50). Considering that the volume of the bony defect in the tarsal navicular was relatively small (<5 mL) compared with that of osteonecrosis of the femoral head (30 to 48 mL), and that donor site

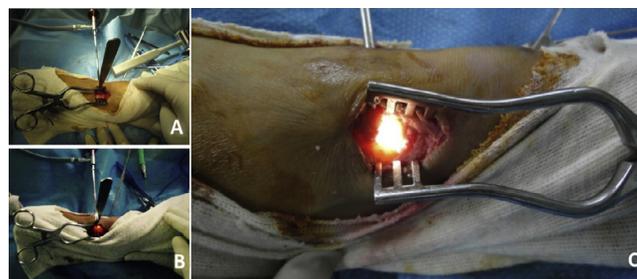


Fig. 8. The light bulb procedure. (A) Transillumination was weak in the sclerotic navicular bone before debridement. (B) Transillumination was observed more easily after switching off the operating light. (C) Increased transillumination of the navicular after thorough debridement.

morbidity was to be avoided, bone substitute was used in this case. Successful bone ingrowth and remodeling were observed on postoperative MRI (45,51–52).

In our present case, sclerotic changes in the navicular were noted on CT scan after failed percutaneous decompression. Bone sclerosis was also described by Tosun et al (8). Although percutaneous decompression was less invasive in that case, debridement of the sclerotic osteonecrotic bone and replacement with autologous bone graft appears to be a rational treatment approach. Thus, evaluating sclerosis of the navicular might be important in treating tarsal navicular osteonecrosis in the early, precollapse stage.

In conclusion, for tarsal navicular osteonecrosis diagnosed at an early stage, with failed conservative treatment and absence of bony collapse, there is no optimal operative treatment, and only a few techniques have been described. Adoption of the light bulb procedure used for osteonecrosis of the femoral head may be a useful method for bone salvage and restoration of function; however, it should be used with caution in early-stage tarsal navicular osteonecrosis because it is currently only supported by a very small number of patients with short duration of follow-up.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1053/j.jfas.2018.08.003.

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