CASE REPORTS

Direct Bone Marrow Injections for Avascular Necrosis of the Talus: A Case Report

Ross A. Hauser, MD & Shelby Ostergaard

INTRODUCTION

Avascular necrosis (AVN) of the talus is a debilitating condition, often leading to arthritis and arthrosis of the subtalar and ankle joints.[1] There are three basic categories of cause for AVN. Approximately 10% of AVN of the talus is considered idiopathic: 15% is medication induced and 75% from trauma.[2,3] The most feared complication of talar injuries is AVN.

The talus has several anatomic features that predispose it to AVN. The body of the talus is wider anteriorly than posteriorly and contains the talar dome, which forms the talocrural joint with the tibia. This joint bears more weight per unit of area than any other joint in the body, contributing to its propensity to get injured with ankle trauma.[4] The talus is weakest at the neck, where the bone is recessed to allow for dorsiflexion. In addition, the talar neck has a scarcity of cartilage and numerous ligamentous attachments. Talar neck fractures represent 50% of all talar injuries and are responsible for 90% of all traumatic AVN.[5]

While arthroscopy with or without core decompression is the standard of care for unresolved cases of AVN of the talus, we present a case of AVN of the talar dome where symptoms resolved satisfactorily with direct bone marrow injections into structures into and around the ankle.

CASE REPORT

MD, a 59 year old school administrator from Alaska, had chronic right lateral ankle pain for three years after a severe ankle sprain. Initial x-rays were unremarkable. Her ankle inverted while dancing, significantly increasing her pain. She was treated with an extensive period of guarded weight bearing, crutches, and non-weight bearing for several months but noted no improvement. A corticosteroid shot produced no results. An MRI on 7/7/2010 then showed prominent AVN of the talar dome involving the central to posterior aspect. She was offered various surgical options including arthroscopy with debridement, allograft osteochondral transfer, core decompression and ankle fusion through external fixation. The prognosis for complete pain relief with these options was guarded so MD decided to seek more conservative treatment.

In February 2011, MD decided to seek treatment of her condition by Prolotherapy. She presented complaints of severe pain, stiffness, crepitation, and extremely limited ankle motion to Caring Medical and Rehabilitation Services. Her pain would dramatically increase with any weight bearing and continue to increase throughout the day. She could not even walk around her house without pain. She stopped all extraneous walking, exercise, and hiking. Her pain was further aggravated merely by standing on her feet. She complained of swelling around the ankle. On physical examination an obvious limp was noted. Notable tenderness was observed in the anterior and posterior talofibular, calcaneofibular, deltoid, and tibiotalar ligaments. Her active range of motion was as follows: dorsiflexion 10 degrees; plantar flexion 15 degrees; subtalar eversion 5 derees; and subtalar inversion 10 degrees.

The following areas in the right ankle joint and its medial and lateral bony attachments were injected with a 15% dextrose, 0.1% procaine and 10% Sarapin solution: medial and lateral malleolus, talus, navicular, calcaneus, cuboid, and calcaneus, deltoid, plantar calcaneonavicular, talonavicular, anterior and posterior tibiofibular, anterior and posterior talofibular and calcaneofibular ligaments. A total of 36cc of solution was utilized in 22 separate injections. (*See Figure 1.*)

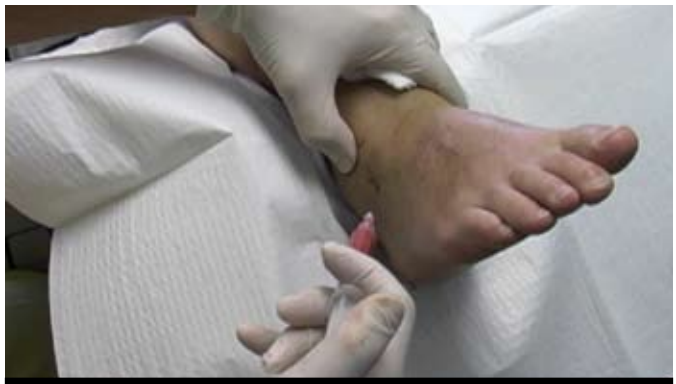

Figure 1. Direct bone marrow injection to the ankle.

For the bone marrow aspiration, an EZIO-10 drill with 28mm bone marrow needle was used. Once the periosteum was reached, anesthesia in the area was confirmed. The EZIO-10 drill was turned on and once the periosteum was pierced, the drill was turned off. The stylet was removed from the neelde. A 12cc luer lock syringe with 2,000 IU of heparin (2cc) was attached to the cannula hub, then 10cc of bone marrow was extracted. The syringe was detached and an empty 10cc syringe was attached and with negative pressure the needle was removed. Pressure over the area was then applied with gauze for two minutes. A pressure dressing was then placed on the area and secured with tape.

Five ccs of direct bone marrow solution was then instilled into the ankle (tibiotalar) and subtalar joints after 1cc of 8% procaine (80mg of procaine) was injected. MD was instructed to follow-up every two months.

When MD came in for her second visit, she was pleased to report that her ankle felt stronger and more stable. She was able to be up on her feet for longer periods of time without pain and she could now walk about a half mile without pain. At her third visit, MD enthusiastically reported an even greater improvement. She was back to hiking up to three miles on hilly, uneven ground and she said that there were several days over the past couple months where she had absolutely no pain in her ankle. On her fourth visit, she reported being able to do activities of daily living without pain and could hike most days without any pain. She could now hike longer, more often, and go up mountains. Only with extreme strenuous hiking did she have symptoms. On physical examination she had only very minimal tenderness in the medial and lateral ankle ligamentous attachments and her active range of motion was as follows: dorsiflexion 15 degrees; plantar flexion 30 degrees; subtalar eversion 12 degrees; and subtalar inversion 16 degrees. She now walked, jumped, hiked, and ran with only minimal symptoms. She was phoned six months after her last visit and stated her pain on a 0 to 10 scale was 1 was aggressive activity, but had 0 pain with normal daily activities and no pain at rest. Her ankle swelling has completely resolved. She had no limitation of activities and has resumed aggressive mountain hiking. She is on no pain medication.

DISCUSSION

There are several pathophysiological causes for the ischemia that has been postulated as the etiological basis for avascular necrosis. One involves an acceleration of the bone degradation versus synthesis.[6] In the course of avascular necrosis, however, the healing process is usually ineffective and the bone and subsequent soft tissues break down faster than the body can repair them. If left untreated, the disease progresses, the bone collapses and the joint surface breaks down, leading to pain and arthritis.

Avascular necrosis occurs when the blood supply to the talus is compromised and leads to ischemic bone death. About 60% of the talus is covered by cartilage, limiting the area for blood vessels to penetrate.[7] The blood supply to the talus enters the bone through the capsular and ligamentous attachments.[8] The vascular supply to the talar dome is an end-artery system, with blood vessels entering from the talar neck and plantar talar body. Circulation to the talus is supplied by the posterior tibial artery, anterior tibial artery, and peroneal artery, whose branches form a vascular sling around the talar neck and sinus tarsi.[9] The posterior tibial artery reaches the talus through the inferomedial soft-tissue attachments.[10] Because of this delicate vascularization soft-tissue attatchments surrounding the talus are imperative to the blood supply.[11] Cases of injury to the talus without fracture or ligamentous injuries, such as certain anterolateral dislocations, often do not develop avascular necrosis.[12]

MD, suffered from two separate lateral ankle sprains. This type of injury involves inversion and plantar flexion, commonly injuring the lateral ligament complex involving the anterior and posterior talofibular ligaments and calcaneofibular ligament. The common result of this is lateral ankle instability. While fractures are the main

cause of compromised talus blood supply, ankle joint instability, in this case occuring over three years, was the most probable cause. Her injuries to the ligamentous attachments on and surrounding the talus, caused her to develop ankle joint instability and resulted in a disrupted blood supply and the development of AVN.

MD's condition was found by MRI, the standard for diagnosing AVN.[13, 14] Avascular necrosis can be treated in a number of ways. The normal course of treatment begins with limiting weight-bearing on the extremity.[15] The amount of time that a person spends non-weight bearing (crutches) or limited weight bearing depends on the stage of injury.[16] It has also been found that the desirable time and degree of weight bearing is typically dictated by the degree of osteonecrosis.[17, 18]

MD failed weight bearing restrictions and then was offered the common surgical approaches to AVN including core decompression and ankle fusion.[19, 20] While multiple surgical options are available including debridement, osteotomy and bone grafts to treat AVN, their efficacy remains controversial.[21-23]

The first case report of using bone marrow cells for osteonecrosis was reported in 1997.[24] The technique of bone marrow transplantation into areas of avascular necrosis after core decompression has expanded, especially of the hip.[25, 26] Transplanted bone marrow aspirates contain progenitor cells including mesenchymal stem cells, whose levels are depressed in patients with osteonecrosis.[27] A deficiency of osteoblastic cell proliferation has also been shown in AVN.[28] It is this insufficiency of osteogenic cells could explain the inadequate repair mechanism that to AVN.

This is the first case report of using direct bone marrow aspiration into areas of pain in a patient with AVN of the talus that does not involve a core decompression. In this particular case, the bone marrow aspirate was injected into the tibiotalar and subtalar joints. The surrounding painful and injured ligaments on the lateral and medial sides of the ankle were also treated with Prolotherapy. ■

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