

Avascular Necrosis of the Talus in a Professional Football Player

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Avascular necrosis, or the death of bone or tissue, results from a diminished blood supply to an area of the body. Usually the direct result of an injury, these cases have been reported in the hip, medial femoral condyle of the knee and sesamoids of the great toe in professional football players. However, the incidence of this type of injury to the talus bone of the ankle is rare. It is even less frequent when there is no direct injury to the bone.

This case study will examine the treatment and rehabilitation of an avascular necrosis of the talus suffered by a professional football player.

Injury

The injury occurred in a divisional playoff game on January 9, 1999, when a 29-year-old running back was tackled from his right side. While turning in an attempt to maintain his balance, he caught his left foot in the turf as he fell to the ground. His body continued to rotate to the right, fracturing his left distal fibula just superior to the lateral malleolus and tearing a small part of the anterior portion of the syndesmotic ligament.

Initial Treatment

Surgeons repaired the fracture on January 16, 1999. He was casted and remained

non-weight bearing until March 4, when he began a rehabilitation program in a physical therapy clinic located away from the 49ers facility.

Soon after, he complained of increased ankle pain. An MRI showed what appeared to be a stress reaction in the talar dome. We believed, at the time, that this may have been caused from repeated forced dorsiflexion while receiving physical therapy at a clinic in his hometown. The MRI also revealed what could have been the initial stages of osteonecrosis,

with approximately one half of all talar avascular necrosis cases being the result of a disruption in the subtalar joint. However, the sinus tarsi and subtalar joint were not compromised in this case, as neither the cervical nor the interosseous talocalcaneal ligaments were torn. As there were no osteochondral defects present on the articular surface of the talus when the ankle was first injured, we were initially unsure what caused his talar necrosis to develop.

It was then hypothesized that the blood circulation through the sinus tarsi region was not adequate enough to provide nutrition to the talus before the injury, so there had to be additional feeders by which the bone and cartilage were being nourished. We believe that this accessory vascular supply came from the posterior tibial artery through the deltoid ligament and that it had been compromised by the injury.

Continued Treatment

A CT-Scan was taken in June to determine the rate of healing, and it was decided to salvage the chondral surface of the talus by cleaning out the necrotic bone tissue and replacing it with graft material. This surgery was performed on July 24.

Following a month in a boot cast, he began rehabilitation exercises, including light distraction mobilizations, soft-tissue massage, cardiovascular work and hyperbaric treatments. Unfortunately, ankle

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Forced rotation in the ankle led to a fractured fibula, torn syndesmotic ligament and an avascular necrosis of the talus in this athlete.

which was found to be the true cause of the changes. A repeat MRI performed in June showed further degenerative changes in the talus.

Talar Necrosis

The primary vascular supply to the talus comes from the sinus tarsi region below it,

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AVASCULAR NECROSIS

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dorsiflexion could not be increased much beyond neutral.

By November, X-rays revealed minimal filling in of the necrotic area.

Additional Surgical Procedures and Follow-up Care

As limited healing was taking place, the following procedures helped speed the rate of recovery:

December 1999

Surgery:

- Arthroscopic debridement of the anterior, medial and lateral gutters of the ankle and joint manipulation under anesthesia to achieve dorsiflexion to match the uninvolved ankle.

Follow up:

- MRI performed January 2000 and CT-Scan done in February showed no significant improvements in the healing of the defect.
- Continued physical therapy and joint mobilizations helped to increase ankle dorsiflexion to 10 degrees, but an April CT-Scan revealed a demarcation line with no change in circulation to the graft.

May 2000

Surgery:

- Medial malleolar osteotomy and debridement of articular cartilage and bone graft material from earlier surgeries.

- Osteochondral grafts from the player's right knee were harvested and packed into the talus to maintain as much of the smooth chondral surface as possible.

Follow up:

- The player was immobilized for eight weeks and was walking in a boot cast by mid-July.
- Joint mobilizations, isotonic strengthening in all four planes of motion, pool exercises, stretching and proprioceptive work was added.
- An August MRI showed that the malleolar osteotomy was not fully healed, the talar graft material was healing and the articular cartilage was still rough.
- In November of 2000, the player returned to practice with the team with complaints of morning stiffness in the ankle. He was inactive for the remaining games of the 2000 season.

Resolution

In January 2001, surgeons went in one last time to:

- Remove the irritating medial malleolar osteotomy screws.
- Eliminate heterotrophic bone from between the distal tibia and fibula.
- Debrided the medial and lateral gutters of scar tissue.

Dorsiflexion improved to 15 degrees passive and 10 degrees active post-op. He continued

physical therapy until reporting for training camp later that summer.

Return to Play

Multiple sessions of daily joint mobilizations, neurodynamic treatments and modifications to the levels of practice and game participation allowed the player to have an extremely productive 2001 season. In addition to rushing for 1,206 yards, he was:

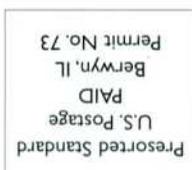
- Named as an NFC Player of the Week.
- Recognized as the NFL Comeback Player of the Year.
- Selected to the 2002 Pro Bowl Squad.

Conclusion

A significant component to the successful outcome of this injury was the player's goal setting, attitude and belief that he was going to recover. The ankle still has a minimal amount of dorsiflexion, yet his hard work and determination have allowed him to overcome a potentially career-ending injury and return to the highest levels of competition.

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