Delayed Avascular Necrosis of the Talus Following Arthroscopic Os Trigonum Excision

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Introduction
Posterior ankle arthroscopy is a commonly used modality for the treatment of os trigonum, prominent stieda process, posterior tibiotalar ligament impingement, removal of loose bodies, and tenolysis of the flexor hallucis longus. Complications following posterior ankle arthroscopy are rare but include damage to the posterior tibial artery and nerve when using the posterior medial portal.

We present a case of iatrogenic avascular necrosis of the talus after arthroscopic excision of an os trigonum using a 2-incision posterior-medial and posterior-lateral approach. To our knowledge, this complication has not been reported after ankle arthroscopy.

Case
A 67-year-old healthy woman, with no significant medical or operative history, sustained a left inversion ankle sprain injury (no fractures on radiographic imaging) that was treated conservatively with rest, ice, compression, and elevation of the affected extremity. Four years following her injury, the patient continued to complain of posterior ankle pain that was aggravated with walking, navigating stairs, and wearing heels. Physical examination revealed tenderness and swelling of the posterior ankle joint. The patient was treated with a single cortisone injection, a nonsteroidal anti-inflammatory drug, physical therapy, and ankle supports without relief of symptoms. Radiographs and magnetic resonance imaging (MRI) revealed a large os trigonum involving the posterior talus with marrow edema within the posterior talus (Figure 1A-C). No additional corticosteroid injections were performed.

After 6 months of nonoperative treatment, the patient underwent a left posterior ankle arthroscopy with an excision of the os trigonum using the technique proposed by van Dijk. The patient was in prone position and posterior ankle arthroscopy was completed via posterior-medial and posterior-lateral portals with no distraction. The procedure was completed under tourniquet (total time of 40 minutes), with intra-articular fluid pressures maintained between 35 and 40 mm Hg. The flexor hallucis longus tendon was visualized and used as the medial boundary for dissection as the os trigonum was identified lateral to this. Soft tissue dissection medially was limited to avoid risking injury to neurovascular structures (Figure 2). The os was removed without complication and the patient’s neurovascular status remained intact after surgery. The patient did well initially but developed pain approximately 6 months after surgery.

At 6 months following surgery, a plain-film radiograph demonstrated changes to the talus consistent with avascular necrosis (Figure 3). A follow-up MRI showed avascular necrosis (AVN) of the talar body (Figures 4 and 5). In the span of these 6 months, she did not sustain any new injuries or have any new changes in her medical or social history.

Discussion
Avascular necrosis of the talus has been reported following fracture-dislocation, exposure to pressurized atmospheric conditions, use of corticosteroids, Addison disease, Cushing syndrome, alcoholism, sickle cell, peripheral vascular
disease, lupus, and dialysis for chronic renal failure. We report of a case of AVN of the talus that occurred after posterior ankle arthroscopy. Iatrogenic cases are rare and have been associated with clubfoot repair and triple arthrodesis. Two cases of avascular necrosis of the talus following posterior medial clubfoot approach are noted in the literature. A case of AVN following a medial approach talonavicular arthrodesis has also been reported. These cases of iatrogenic AVN of the talus previously have been associated with damage to the artery of the tarsal canal.

Posterior ankle arthroscopy is generally recognized as a safe procedure for removal of os trigonum, decompression of a prominent posterior talar process, tenolysis of flexor hallucis longus, removal of foreign bodies, and arthroscopy. The posterior medial portal is considered to be the highest risk but is found to be 16.5 mm from the posterior tibial artery.

To our knowledge, no previous cases of AVN from ankle arthroscopy have been reported. However, there have been reported cases of AVN in other joints after arthroscopic procedures. Postarthroscopic osteonecrosis of the knee (or osteonecrosis in the postoperative knee) is a rare complication usually seen in elderly patients with recurrent or worsening pain after arthroscopic treatment of meniscal or chondroid lesions. The pathophysiology is still not fully understood but studies have suggested that it could be due to heat generated to the subchondral bone from radiofrequency probes or more likely from the increased biomechanical load causing insufficiency fractures within the femoral condyle after partial meniscectomy within the compartment. There have also been some case reports of AVN after arthroscopic anterior cruciate ligament reconstruction or patellar osteonecrosis after arthroscopy presumably due to excessive disruption of the anterior fat pad.

Osteonecrosis of the femoral head with arthroscopy has been rarely seen and reported. The arthroscopy portals do not risk the key vascular structures, but possibly the combination of the traction and increased intracapsular pressure may increase the risk. Similarly, in the shoulder, few case
series have reported postarthroscopic humeral head osteonecrosis after rotator cuff repairs. A potential cause of this has included the disruption of blood supply from the anchors.\(^1,9\)

AVN of the talus is primarily caused by disruption of the blood supply. In 1940, Phemister noted that the major blood supply to the talus was the dorsalis pedis artery and its branches.\(^12\) The artery of the tarsal canal is another major blood supply to the body of the talus that arises from the posterior tibial artery. The peroneal artery supplies the lateral process of the talus. A significant portion of the blood supply lies in periosteal vessels around the talus.\(^11\)

Vasospasm has been shown to be an important mechanism in causing avascular necrosis. In a study on rabbits, osteonecrosis was induced by injecting corticosteroids around the femoral head. In the treatment group, fasudil, an antispasmodic agent, was injected as well, and this significantly reduced the rate of avascular necrosis.\(^7\) A study on rats by Drescher et al\(^4\) also showed that vasoconstriction was a likely cause of femoral head avascular necrosis.\(^7\) Furthermore, patients with coagulopathy are predisposed to developing avascular necrosis.\(^8\) This all suggests that severance of arteries may not be necessary to cause this condition.

In this article, we have described a woman where the posterior medial and posterior lateral arthroscopic approaches were used for removal of an os trigonum. The MRI taken several months before the procedure failed to show any signs of avascular necrosis, and the MRI after the procedure demonstrates severe avascular necrosis of the talar body. The exact mechanism in this case is unclear as there was nothing to suggest arterial injury during or after surgery, nor was the case performed under tourniquet for an excessive period of time (40 minutes). Additionally, it seems she had no other extrinsic risk factors or causes that could have led to AVN. However, it is documented in animal models that vasoconstriction is a mechanism that can cause AVN. It would seem possible that in this case the arthroscopic procedure triggered a vasospastic event, causing avascular necrosis of the talus.

**Ethics Approval**

Ethical approval was not sought for the present study because this is a case report; however, we complied with HIPAA guidelines and maintained confidentiality and privacy while preparing the report.

**Declaration of Conflicting Interests**

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