Arthroscopic Approach to Posterior Ankle Impingement

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KEYWORDS

• Posterior ankle impingement • Arthroscopic • Endoscopic • Os trigonum

KEY POINTS

- Posterior ankle pain can occur for many reasons. If it is produced by forced plantarflexion of the foot, it is often a result of impingement from an enlarged posterior talar process or the presence of an accessory ossicle, known as an os trigonum. This condition may present in either the acute or chronic state.
- Management is initially nonoperative but, if pain remains refractory, then surgical treatments are available. Because this is a condition often seen in athletes, procedures that limit surgical trauma and allow early return to activity are ideal.
- Arthroscopic approach for this disorder has been shown to produce good to excellent outcomes with limited complications. Understanding the indications, local anatomy, and surgical technique allows good reproducible outcomes.

Video content accompanies this article at http://www.podiatric.theclinics.com.

INTRODUCTION

Posterior impingement syndrome is often synonymous with the terms posterior talar compression syndrome, os trigonum syndrome, posterior ankle block, nutcracker-type impingement, and posterior tibiotalar impingement syndrome. Frequently, it is associated with the presence of an accessory ossicle known as the os trigonum. Pain can be elicited acutely with a forced plantarflexion injury or chronically in those individuals performing repetitive plantarflexory moments of the foot and ankle, such as ballerinas or soccer players. The latter presentation is more common.

The os trigonum develops as a secondary cartilaginous center during the second month of fetal development, between the ages of 7 and 11 years in girls and 11 and 13 years in boys. Through enchondral ossification, the center fuses to the posterolateral talus with a cartilaginous synchondrosis.[1] One year after its appearance, the

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secondary center unites to the talar body producing the Stieda process.[2] Failure to unite has been identified in 1.7% to 49%[3] of the general population. Intimate to the presence of this accessory ossicle, is the flexor hallucis longus (FHL) tendon. As a result, the tendon is susceptible to a stenosis tenosynovitis. Chronic entrapment of this tendon can occur resulting from low-lying muscle tissue, impingement from an os trigonum, and incongruity of maximum plantarflexion and dorsiflexion of the ankle and great toe joint resulting in compression of the tendon. This condition often occurs in the fibro-osseous tunnel posterior to the medial malleolus. This condition can be seen in athletes who require forceful plantarflexion of the foot, such as soccer players, swimmers, and ice skaters.

ANATOMY

The posterior ankle joint complex is well defined by soft tissue and osseous structures. Medially, it is bounded by the flexor tendons of the leg, including (from superficial to deep) the posterior tibial tendon, the flexor digitorum longus, and the FHL. The FHL tendon is a critical medial boundary during arthroscopy because the neurovascular bundle lies just medial or posterior to this structure (**Fig. 1**). Posteriorly, the prominent Achilles tendon is appreciated. Laterally, the peroneal tendons serve as an outside boundary to protect the sural nerve and small saphenous vein during arthroscopy. Anteriorly lies the tibiotalar and talocalcaneal joints. Inferiorly, is the tuber of the calcaneus. Within the contents of this vault is adipose tissue frequently refer to as the Kager triangle.

In 2002, Sitler and colleagues[4] published an anatomic study regarding structures at risk when performing posterior ankle arthroscopy through both medial and lateral portals. They examined 13 fresh frozen cadavers and placed plastic cannulas filled with oil in portals to serve as landmarks when performing MRI. Imaging studies were compared with open dissection of the specimens to confirm correlation of proximity of vital structures. Portals were placed immediately adjacent to the Achilles tendon. It was appreciated that, on average, the sural nerve was 3.2 mm from the portal, 4.8 mm to the small saphenous vein, 6.4 mm to the tibial nerve, 9.6 mm to the posterior tibial artery, 17 mm to the medial calcaneal nerve, and 2.7 mm to the FHL tendon. There was little discrepancy with MRI studies with the exception of the tibial nerve, which could not always be appreciated on MRI.[4]

Balci and colleagues[5] similarly studied cadaveric specimens, placing posteromedial and lateral portals but adjusting the position of the ankle, assessing neutral, 15° of dorsiflexion, and 30° of plantarflexion. In neutral, the anterolateral portal was 6 mm from the sural nerve and 1.6 mm from the peroneals. The distance between the medial portal and the FHL was 2.11 mm and from the tibial artery was 6 mm. With increased dorsiflexion, the distance between the posterior portal and the neurovascular bundle medially increased. This finding may suggest that dorsiflexion of the ankle during portal placement may better protect medial vital structures.[5]

PATHOGENESIS

Although a rare condition, os trigonum syndrome may occur acutely through hyperplantarflexion injury or chronically by repetitive plantarflexion stress moments.[6] As the talus rotates plantarly, the accessory ossicle or prominent lateral talar process is impinged between the calcaneus inferiorly and the tibial plafond superiorly. It must also be appreciated that hyperdorsiflexion of the foot can produce avulsion of the lateral talar process by increased tension to the posterior talofibular ligament.[7] This condition has also been referred to as a Shepherd fracture (**Figs. 2–4**).[8]