

Posterior Ankle Impingement: Os Trigonum Syndrome

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Six types of ankle impingement are described in the literature, and each takes its name from its location about the ankle joint. The 3 most common impingement syndromes are anterior impingement, anterolateral impingement, and posterior impingement. Three other ankle impingement syndromes—anteromedial impingement, posteromedial impingement, and syndesmotic impingement—are less well characterized in the literature.

Posterior impingement syndrome is characterized by posterior ankle pain with forceful plantar flexion. Soft tissues, bony processes, unfused ossicles, or osseous fragments entrapped between the posterior tibial plafond and the superior calcaneus lead to symptoms. Structures implicated in the etiology of posterior impingement of the ankle include the os trigonum, a prominent posterolateral process of the talus, fractures of the ossicle or talar process, enlarged posterior process of the calcaneus, the posterior intermalleolar ligament, soft-tissue impingement, loose bodies, ganglia, calcified inflammatory tissue, a low-lying flexor hallucis longus muscle belly, and anomalous muscles.¹ Pathology of the os trigonum–talar process is the most common cause of posterior impingement syndrome.²

Os trigonum syndrome is a subset of posterior ankle impingement caused by pathology at the posterolateral process of the talus. Os trigonum syndrome is seen in athletes who engage in forceful plantar flexion or push-off maneuvers, such as jumping, downhill running, or kicking. Clinical presentation may be either acute secondary to trauma or chronic as a result of repetitive stress. Ballet dancers are commonly afflicted, and female ballerinas are particularly susceptible, given the extreme plantar flexion of the en pointe position.²

The os trigonum, a secondary ossification center at the posterolateral corner of the talus, mineralizes between ages 7 and 13 years. Usually, this ossicle fuses to the talus to form the posterolateral process of the talus. Incomplete ossification may occur in up to 14% of the population, leaving a syn-

chondrosis or syndesmosis between the talus and the ossicle. When an os trigonum is present, it is bilateral in 50% of patients. There are 4 anatomical variants of the posterolateral talus: I (normal posterolateral talar process), II (elongated posterolateral talar process, or Stieda process), III (accessory bone or os trigonum), and IV (os trigonum fused with posterolateral talus by synchondrosis or syndesmosis).

Diagnosis of os trigonum syndrome is based primarily on clinical examination and history. Imaging findings support the diagnosis. Patients typically complain of posterior ankle pain between the Achilles and peroneal tendons exacerbated by forced plantar flexion or wearing of high-heeled shoes. There may be a history of antecedent trauma. Some patients may have coincident symptoms of flexor hallucis longus tenosynovitis manifesting as posterior ankle pain radiating into the arch. On physical examination, ankle and subtalar range of motion should be fully assessed. Care must be taken to differentiate between Achilles-tendon–related pain and posterior ankle impingement. In the passive forced plantar flexion test, the patient sits with knee flexed at 90°, and multiple quick hyperplantar-flexion movements are performed at the ankle.

Imaging findings of posterior impingement may vary according to the etiology of the symptoms. The most common plain radiograph finding is presence of os trigonum or the prominent elongated posterolateral process of the talus known as the Stieda process (Figure 1). There may be degenerative change across or frank disruption of the synchondrosis of the os trigonum (Figure 2). A fracture of the os or the Stieda process may be encountered. Bone scans may show abnormal radionuclide

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Figure 1. Radiograph shows large os trigonum (asterisk).



Figure 2. Sagittal T₁-weighted (A) and short tau inversion recovery (B) images show large os trigonum with marrow edema, cystic change, and sclerosis at os trigonum and at adjacent posterior talus (arrow heads) across synchondrosis. There are no fluid between ossicle and talus and no displacement of os. Synchondrosis remains intact. There is nodular synovial reaction (white arrow) involving posterior recess of ankle joint.

uptake at the posterior ankle; however, a study of active-duty soldiers found uptake to be a frequent finding and of limited value in detecting the symptomatic os trigonum.³ Magnetic resonance imaging (MRI) is excellent in showing both bone and soft-tissue changes. MRI can reveal bone marrow edema as well as sclerosis and cystic change of the os trigonum and the adjacent talus (Figure 3). The posterior ankle joint capsule may demonstrate fluid distention, posterior capsular thickening, and nodular synovitis. Fluid in or edema of the adjacent soft tissues, fluid within the synchondrosis, or a fracture of the Stieda process may be present. Flexor hallucis longus tenosynovitis is also not uncommonly encountered. MRI may be of aid in

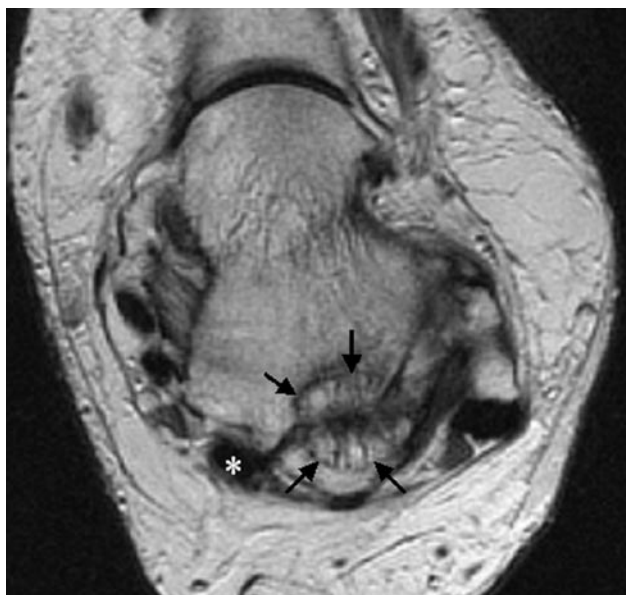


Figure 3. Axial proton density-weighted image shows reactive cystic marrow changes at os trigonum and posterior talus across intact intervening syndesmosis. Note proximity of flexor hallucis longus tendon (*).

demonstrating other processes that mimic posterior impingement clinically, such as fractures or Achilles tendon pathology.⁴ Fluoroscopically controlled injection can help confirm the suspected diagnosis of os trigonum syndrome.⁵

Treatment of posterior ankle impingement is initially conservative, with rest, icing, and use of nonsteroidal anti-inflammatory drugs producing good results. Steroid injections may lead to temporary symptom relief. A study of professional soccer players with posterior impingement found that ultrasound-guided injection of steroids and local anesthetics was well tolerated, which allowed for rapid return to athletic activity in all patients studied.⁶ Patients for whom conservative management fails may benefit from open surgical or arthroscopic resection of the Stieda process or os trigonum.⁷

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

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