AMERICAN ORTHOPAEDIC FOOT & ANKLE SOCIETY

Foot & Ankle International
34(3) 459–465

DOI: 10.1177/1071100713477609
fai.sagepub.com

Posterior Ankle Impingement

Sandro Giannini, MD[1], Roberto Buda, MD[1], Massimiliano Mosca, MD[1], and Alessandro Parma, MD[1], and Francesco Di Caprio, MD[2]

Abstract

Posterior ankle impingement is a common cause of chronic ankle pain and results from compression of bony or soft tissue structures during ankle plantar flexion. Bony impingement is most commonly related to an os trigonum or prominent trigonal process. Posteromedial soft tissue impingement generally arises from an inversion injury, with compression of the posterior tibiotalar ligament between the medial malleolus and talus. Posterolateral soft tissue impingement is caused by an accessory ligament, the posterior intermalleolar ligament, which spans the posterior ankle between the posterior tibiofibular and posterior talofibular ligaments. Finally, anomalous muscles have also been described as a cause of posterior impingement.

Keywords: ankle, impingement, arthroscopy

Posterior ankle impingement is a common cause of chronic ankle pain. It is the result of compression between bony or soft tissue structures during terminal plantar flexion. The cause and pathological anatomic features are heterogeneous, and several pathological conditions can result in posterior impingement. As such, it is now considered a syndrome. Although nonoperative treatment measures are often effective, the traditional surgical management of patients with recalcitrant symptoms has involved open decompression. Nevertheless, arthroscopic decompression has recently increased in popularity.[7,10,15,19,22,26,27,35,37,38] The current article reviews the various causes of posterior ankle impingement, with a focus on the pertinent pathoanatomy, diagnostic workup, and treatment options.

Posterior Bony Impingement

Pathoanatomy and Diagnosis

The bony structures responsible for posterior ankle impingement lie in the tibiocalcaneal interval. These include the posterior malleolus, the posterolateral talar process (trigonal or Stieda's process), an os trigonum, the posterior subtalar joint, and the posterior calcaneal tuberosity. In early anatomic studies, Cloquet and Shepherd independently described the presence of an os trigonum. Both authors attributed this bony variation to a fracture.[13] Although it is sometimes still referred to as a Shepherd's fracture, Shepherd later realized that this structure was actually a separate ossification center.[13] An os trigonum is an accessory bone that represents a developmental analogue of the secondary ossification center of the posterolateral talus. The latter mineralizes between 7 and 13 years of age and fuses within 1 year. In 7% to 14% of adults it remains as a separate accessory bone,[17] which is bilateral in 1.4% of cases (Figure 1). This structure is usually asymptomatic, but it may become painful in individuals participating in sports involving repeated plantar flexion.[13]

The first clinical description of bony posterior ankle impingement was by Howse[14] in 1982. He described ankle pain in dancers secondary to the presence of an os trigonum or prominent trigonal process. Although Howse coined the phrase "posterior block of the ankle,"[14] Brodsky and Khalil subsequently used the phrase "talar compression syndrome"[3] and emphasized the etiological role of the *en pointe* and *demi-pointe* positions in ballet.[4] The authors recognized the role of plantar flexion in generating compression of the os trigonum or posterolateral process (Figure 2).

The posterolateral process may be acutely fractured by a single plantar flexion event. In the setting of an os trigonum, such an injury results in disruption of the synchondrosis that connects the accessory bone to the native talus. Alternatively, chronic fracture of these structures may result from repetitive stress. In some cases, forced dorsiflexion may increase tension on the posterior talofibular ligament, which can result in avulsion of the os trigonum or posterolateral tubercle.[24]

[1]University of Bologna, Rizzoli Orthopaedic Institute, Bologna, Italy
[2]Ospedale Ceccarini, U.O. Ortopedia e Traumatologia, Riccione, Italy

Corresponding Author:
Francesco Di Caprio, Ospedale Ceccarini, U.O. Ortopedia e Traumatologia, Viale Frosinone, Riccione, 47838, Italy
Email: fra.dicaprio@gmail.com

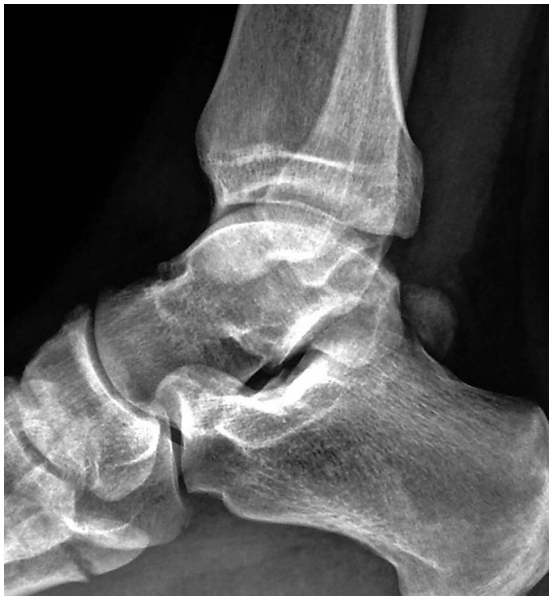

Figure 1. Lateral radiograph demonstrating an os trigonum.

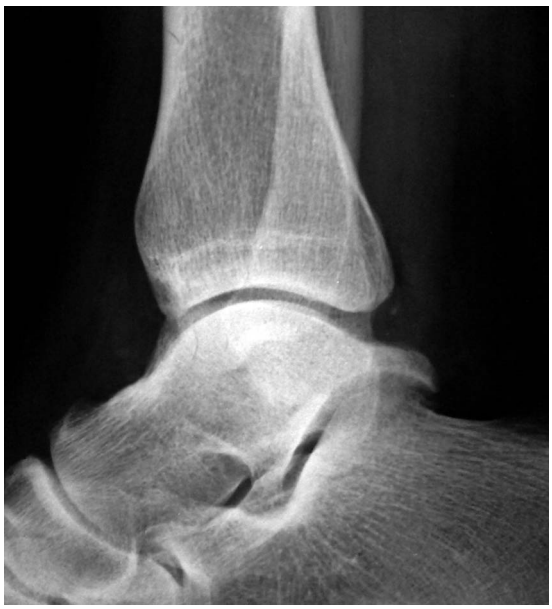

Figure 2. Lateral radiograph demonstrating a prominent trigonal (Stieda's) process.

Finally, posterior impingement may result from previous fractures or degenerative arthritis that can lead to the formation of ankle and subtalar osteophytes (Figure 3). Impinging calcifications in the posterior aspect of the ankle may also result from chronic soft tissue inflammation.

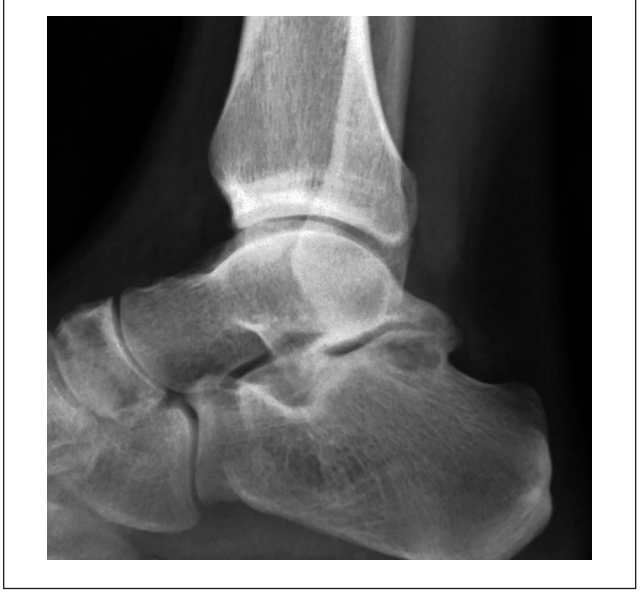

Figure 3. Lateral radiograph demonstrating subtalar arthritis with posterior osteophytes.

The diagnosis of posterior ankle impingement is based primarily on the clinical history. The patient usually reports chronic or recurrent posterior pain caused or exacerbated by forced plantar flexion or push-off activities, such as dance, kicking sports, walking or running downhill, and wearing high heels. The pain is usually deep and may have a mechanical component. There may be a recent or remote history of acute ankle trauma, but overuse must also be considered. On physical examination there typically is posteromedial or posterolateral tenderness. Passive terminal plantar flexion may reproduce the patient's symptoms. If passive hallux motion causes pain, flexor hallucis longus abnormality may also be present.

Marotta and Micheli[21] posited that athletes affected by posterior impingement may attempt to compensate for the loss of plantar flexion by assuming an inverted foot position. This may predispose to frequent ankle sprains, calf strains and contractures, plantar foot pain, and toe curling.

Plain radiographs may detect an acute or chronic fracture of the trigonal process as well as the presence of an intact or fractured os trigonum. Nevertheless, the presence of an os trigonum on conventional radiographs is not necessarily clinically relevant.[17] Calcifications may be detected about the posterior ankle and result from previous trauma, arthritis, or chronic soft tissue inflammation.[13]

With regard to advanced imaging, magnetic resonance imaging (MRI) may reveal marrow edema in the os trigonum or the trigonal process as well as adjacent soft issue inflammation.[5] Other findings that may be demonstrated on MRI include chondral injury and flexor hallucis longus

tenosynovitis.[5] Computed tomography can detect or confirm the presence of a nondisplaced or minimally displaced fracture. Finally, local anesthetic injections may also be of diagnostic value.

Treatment

The initial treatment of bony impingement includes rest, ice, nonsteroidal anti-inflammatory medication, and the avoidance of extreme ankle plantar flexion. Immobilization is indicated if there is evidence of an acute fracture.[13] Subsequent physical therapy and protective dorsiflexion taping may be helpful. Such nonoperative treatment has a reported success rate of 60%.[13] Corticosteroid and anesthetic injections have been shown to provide clinical relief in 84% of cases.[24]

Operative intervention is indicated if symptoms persist despite 3 to 6 months of nonoperative care. Typically, surgery entails excision of the trigonal process or the os trigonum. The approach may be either posterolateral or medial. The posterolateral approach between the peroneal and flexor hallucis longus (FHL) muscle is more direct and avoids the neurovascular bundle.[21] However, the medial approach allows direct visualization of the FHL tendon. This is useful if the latter needs to be released, as in the setting of stenosing tenosynovitis. The medial approach also reduces the risk of peroneal tendinitis and adhesions, which have been reported to be a complication of the posterolateral approach.[13]

In the series reported by Hedrick and McBryde,[13] open excision of a symptomatic os trigonum provided good to excellent results in 88% of patients. In a series of 37 dancers, 26 open procedures were performed for FHL tendinitis and posterior impingement, 9 for isolated tendinitis, and 6 for isolated posterior impingement syndrome.[12] In this series, good to excellent results were reported in 76% of cases, with full return to dance 5 months after the operation for isolated posterior impingement. In another case series, all patients were able to resume dance within 3 months after surgery; however, 67% of patients reported occasional recurrence of their symptoms.[21]

Arthroscopic/endoscopic decompression has recently been described for the treatment of posterior ankle impingement, demonstrating good results with low complication rates and early return to sports.[10,26,35,37] In a series of 16 posterior ankle arthroscopies evaluated at a mean follow-up of 32 months, all patients had good to excellent health-related quality of life and functional outcome scores, with a high rate (93%) of return to preinjury athletic level.[37] In another series of 36 patients treated by hindfoot arthroscopy for symptomatic os trigonum or osteophytes, scores on a visual analog scale for pain improved significantly from 7.2 points to 1.3 points, although impaired sensitivity of the sural nerve was reported in 2 cases.[10] In addition to the removal of an os trigonum (Figure 4) or trigonal process,[38] posterior ankle arthroscopy has been used to resect a prominent calcaneal tuberosity[35] and for tendoscopy of the flexor hallucis longus.[35]

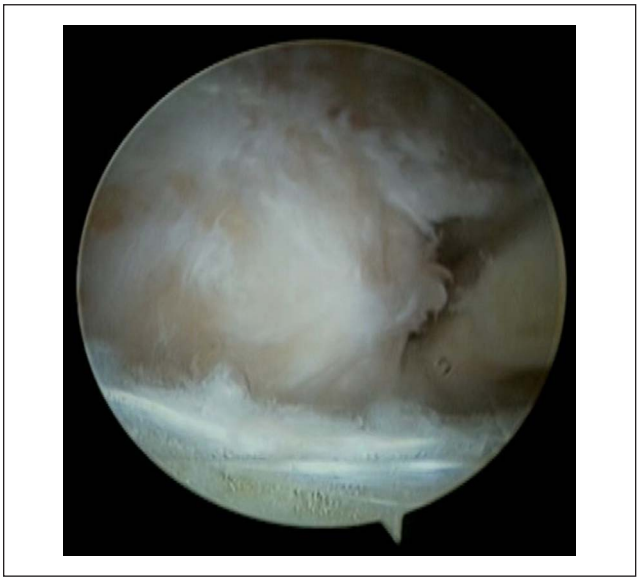

Figure 4. Arthroscopic view of an os trigonum (posterolateral portal).

Posteromedial Soft Tissue Impingement

Pathoanatomy and Diagnosis

Posteromedial impingement is caused by entrapment of fibrotic scar tissue in the posteromedial ankle gutter (Figure 5).[16] This condition was first recognized by Liu and Mirzayan[18] in 1993. These authors reported the case of a patient with chronic posteromedial ankle pain following multiple inversion and eversion ankle sprains. The authors felt that the underlying cause was hypertrophic scar tissue that resulted from a partial tear of the deep posterior fibers of the deltoid ligament.

Posteromedial impingement most commonly arises from an inversion injury with the ankle plantar flexed.[34] With this, the fibers of the posterior tibiotalar ligament (Figure 6) are compressed between the medial malleolus and talus and subsequently hypertrophy. Initially, this injury is often unnoticed because lateral ligamentous disruption dominates the clinical presentation. A less common mechanism includes tearing of the posterior tibiotalar ligament in the setting of an eversion injury. It should be considered, however, that this structure is the strongest ligament of the ankle, and isolated injury is relatively uncommon.

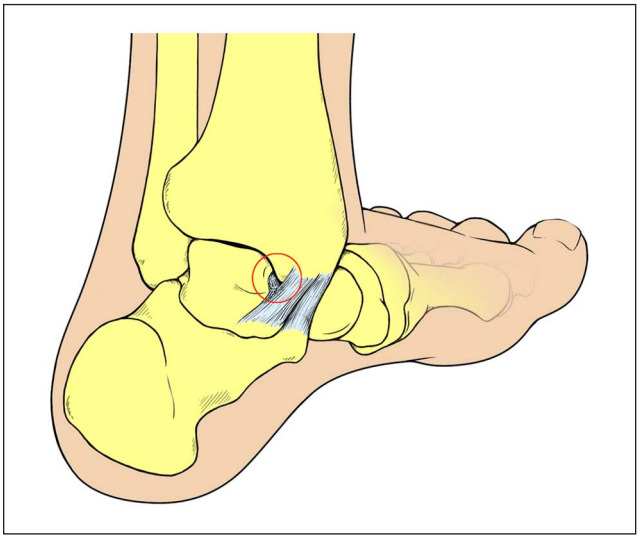

Figure 5. Illustration of posteromedial impingement, with entrapment of fibrotic scar tissue in the posteromedial ankle gutter (circled).

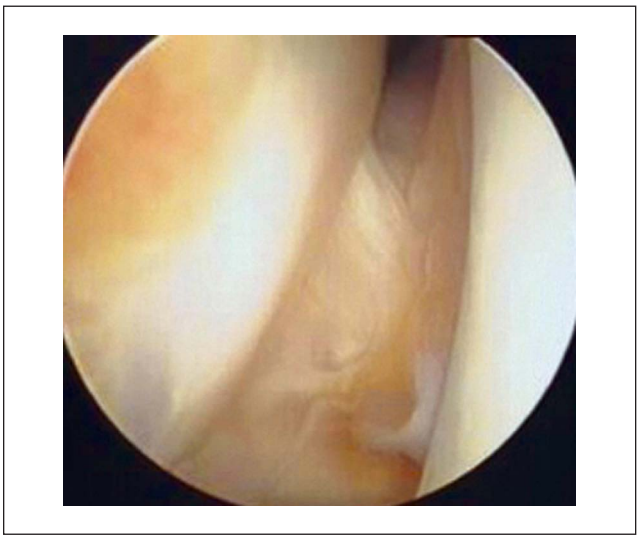

Figure 6. Arthroscopic view of the posterior tibiotalar ligament (anteromedial portal), running from the medial malleolus (left) to the posterior aspect of the talus (right).

The hypertrophied ligament may come into contact with the flexor tendons and partially encase the tibialis posterior (40% of cases), the flexor hallucis longus (16%), or the flexor digitorum longus (8%).[16] Concomitant talar dome chondral injuries have been described in 16% of the patients.[16]

Patients typically complain of posteromedial activity-related ankle pain. In the absence of concomitant lateral ligament abnormality, instability is usually not an issue. In some cases, symptoms may persist for weeks or months after the initial injury. Physical examination often reveals localized fullness and tenderness at the posteromedial aspect of the ankle. Posteromedial tenderness with passive ankle inversion and plantar flexion is a reliable and consistent finding.[28]

Radiographs are usually normal, although in the series reported by Koulouris et al,[16] bone fragments avulsed from the medial malleolus were detected in 12% of cases. Stress radiographs may be useful to exclude instability. MRI has been shown to be most helpful in establishing the diagnosis and demonstrates signal within the posterior tibiotalar ligament with loss of its normal striated appearance and varying degrees of hypertrophy.[16] Ultrasound examination may demonstrate deltoid abnormalities that correspond to MR findings.[16]

Treatment

Conservative treatment includes activity restriction, immobilization, nonsteroidal anti-inflammatory medications, cortisone injection, and physical therapy. Currently, there is insufficient evidence to support the use of one modality over another.[20]

Surgery is indicated for recalcitrant pain. In a series of 6 cases treated by open decompression, complete or near-complete relief was noted in all patients.[28] The authors emphasized the importance of an accurate diagnosis to exclude other causes of pain, such as lateral ligament insufficiency and chondral injury. A posteromedial approach was used, with the posterior tibial tendon dislocated anteriorly and a capsulotomy performed through the tendon bed.[28] Residual fibrotic thickening of the posterior tibial tendon sheath was reported as a potential surgical complication.[28] If concomitant ligament insufficiency is present, a ligament reconstruction should be considered. The lesion is accessible through anterior arthroscopic portals only in the setting of advanced ligamentous laxity.[28] Otherwise, posterior portals should be considered.[35]

Posterolateral Soft Tissue Impingement

Pathoanatomy and Diagnosis

Posterolateral soft tissue impingement is caused by an accessory ligament, the posterior intermalleolar ligament (PIML). The PIML was described by Rosenberg et al[31] in 1995, who identified it in 56% of cadaver specimens. This variant of normal ankle anatomy, also referred to as a marsupial meniscus, spans the posterior ankle between the posterior tibiofibular and posterior talofibular ligaments, from the malleolar fossa of the fibula to the posterior tibial cortex (Figure 7). The PIML may protrude further into the joint during plantar flexion, becoming entrapped and torn. The resulting impingement syndrome consists of ankle locking and pain.

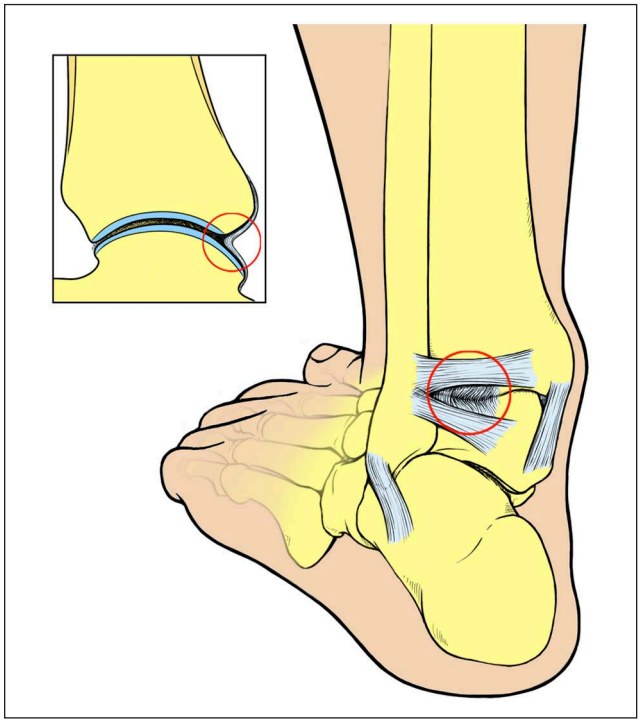

Figure 7. Illustration of posterolateral impingement, with the marsupial meniscus extending intra-articularly between the posterior tibiofibular and posterior talofibular ligaments.

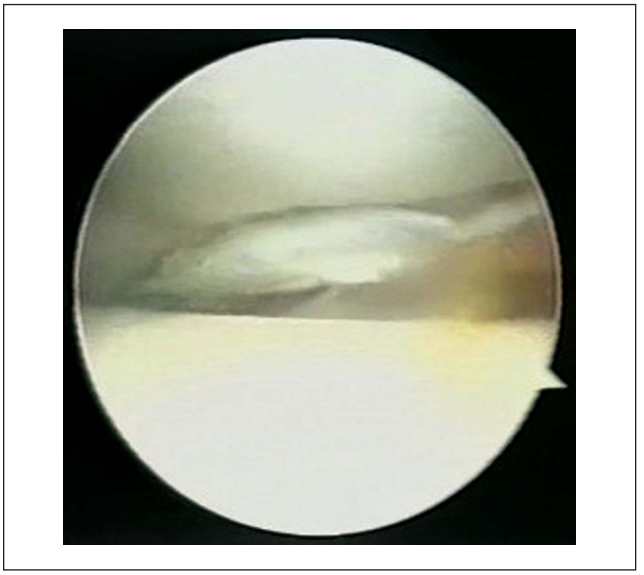

Figure 8. Arthroscopic view of the marsupial meniscus extending intra-articularly (anteromedial portal).

Although the presence of a PIML is common in the general population, posterolateral impingement related to the PIML is rare. In reported cases, symptomatic impingement follows an acute inversion injury with marked plantar flexion. The PIML is likely to be compressed and torn during this initial injury, with subsequent hypertrophy. Activity requiring repetitive forced plantar flexion (eg, ballet, soccer) can then cause chronic posterolateral pain.[9,11] Concomitant lateral ligament insufficiency may cause additional stress on the injured PIML.[20]

On physical examination, range of motion is usually normal. There may be posterolateral tenderness. Symptoms may be reproduced with forced passive plantar flexion and eversion.[20] The ankle should also be checked for ligamentous instability.

Plain radiographs are helpful in ruling out bony posterior impingement, and stress radiographs may be useful in detecting instability. MRI is an effective means to evaluate the PIML as a cause of posterior impingement[9] and demonstrates a thickened meniscoid soft tissue mass in the posterolateral joint space, possibly with bruising of the adjacent chondral surface.

Treatment

As with other causes of posterior impingement, nonoperative treatment may entail activity restriction, immobilization, nonsteroidal anti-inflammatory medications, corticosteroid injection, and physical therapy.[20] Surgery is indicated for recalcitrant pain. In 2 case reports describing posterolateral impingement in high-level athletes, symptoms were completely relieved following arthroscopic resection of the PIML.[9,20] Reports of arthroscopic resection of the PIML demonstrated the presence of a large meniscoid mass extending horizontally across the posterior aspect of the ankle joint (Figure 8).[9,20] If concomitant lateral ankle instability is present, simultaneous ligament repair should be considered.[20]

Posterior Ankle Impingement by Anomalous Muscles

Pathoanatomy and Diagnosis

Although common in the general population, anomalous muscles are an unusual cause of posterior ankle pain.[1,32] The reason why some patients become symptomatic is unclear. One potential explanation is a mass effect leading to transient ischemia[2] or tenosynovitis of adjacent tendons.[11] However, impingement is another potential explanation.[1] The following anomalous muscles have been reported to be possible sources of posterior impingement:

- The peroneus quartus muscle is the most common of these muscles. Also referred to as the peroneus accessorius, peroneus-calcaneus externus, and peroneus externus, it has a reported prevalence of 7% to 22%.[29,33,36,39] It originates from the peroneus brevis muscle, or less commonly from the peroneus longus or posterior aspect of the distal

fibula. It inserts onto the peroneal tubercle of the calcaneus or onto the cuboid or base of the fifth metatarsal.

- The flexor accessorius digitorum longus, also called the second accessorius of Humphrey or long accessory flexor muscle, is present in 1% to 8% of the general population.[25] It arises from the posterior aspect of the tibia, fibula, or interosseous membrane and inserts onto the flexor digitorum tendon at various levels. It may also insert onto the lateral head of the quadratus plantae muscle, lying deep or crossing the neurovascular bundle.[6,25]
- The peroneus-calcaneus internus muscle originates from the distal fibula and runs parallel to the flexor hallucis longus. It inserts onto the under surface of the sustentaculum tali. It was found in 1% of an asymptomatic population.[23]
- The tibiocalcaneus internus originates from the posterior distal tibia and inserts on the medial aspect of the calcaneus.[1]
- The accessory soleus also inserts on the medial aspect of the calcaneus, but it runs outside the tarsal tunnel. It was found in 1% to 6% of the general population.[2,32]
- A low-lying flexor hallucis longus muscle belly is another possible source of posterior impingement.[11]

Patients complain of posterior ankle pain that is activity related.[1] Symptoms may be bilateral, and tarsal tunnel syndrome may also be present. Physical examination typically reveals full motion and no evidence of instability. There is tenderness with deep palpation of the posterior ankle. The use of MRI has been demonstrated to be effective in the diagnosis of abnormalities in the posterior ankle[5,8,30] and in differentiating accessory muscles from other soft tissue masses.[6]

In the case of a peroneus quartus muscle, longitudinal tears of the adjacent peroneus brevis tendon were found in 18% to 50% of cadaver specimens.[33,39] Because of their proximity to the neurovascular bundle, the flexor accessorius digitorum longus, the peroneus-calcaneus internus, and the tibiocalcaneus internus muscles have been implicated as uncommon causes of tarsal tunnel syndrome.[32]

Treatment

Nonoperative management includes rest, activity restriction, nonsteroidal anti-inflammatory medications, and physical therapy. Surgical resection of the anomalous muscle may be considered for recalcitrant pain. Both posteromedial and posterolateral approaches have been described, with removal of the anomalous muscle and, if necessary, neurolysis of the posterior tibial nerve.[11,13] The posterolateral approach presents the advantage of minimizing the risk of postoperative scarring around the nerve, which is particularly important in athletes.[1]

Conclusion

Posterior ankle impingement is a syndrome that has numerous potential causes. Although bony impingement is common, soft tissue impingement should not be overlooked. An understanding of these various causes is essential to the establishment of an accurate diagnosis and implementation of an effective treatment plan.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

1. Best A, Giza E, Linklater J, Sullivan M. Posterior impingement of the ankle caused by anomalous muscles. *J Bone Joint Surg Am*. 2005;87(9):2075-2079.
2. Brodie JT, Dormans JP, Gregg JR, Davidson RS. Accessory soleus muscle: a report of 4 cases and review of literature. *Clin Orthop Relat Res*. 1997;(337):180-186.
3. Brodsky AE, Khalil MA. Talar compression syndrome. *Am J Sports Med*. 1986;14:472-476.
4. Brodsky AE, Khalil MA. Talar compression syndrome. *Foot Ankle*. 1987;7(6):338-344.
5. Bureau NJ, Cardinal E, Hobden R, Aubin B. Posterior ankle impingement syndrome: MR imaging findings in seven patients. *Radiology*. 2000;215(2):497-503.
6. Buschmann WR, Cheung Y, Jahss MH. Magnetic resonance imaging of anomalous leg muscles: accessory soleus, peroneus quartus and the flexor digitorum longus accessorius. *Foot Ankle*. 1991;12(2):109-116.
7. Egol KA, Parisien JS. Impingement syndrome of the ankle caused by a medial meniscoid lesion. *Arthroscopy*. 1997;13(4):522-555.
8. Erickson SJ, Quinn SF, Kneeland JB, et al. MR imaging of the tarsal tunnel and related spaces: normal and abnormal findings with anatomic correlation. *AJR Am J Roentgenol*. 1990;155(2):323-328.
9. Fiorella D, Helms CA, Nunley JA II. The MR imaging features of the posterior intermalleolar ligament in patients with posterior impingement syndrome of the ankle. *Skeletal Radiol*. 1999;28:573-576.
10. Galla M, Lobenhoffer P. Technique and results of arthroscopic treatment of posterior ankle impingement. *Foot Ankle Surg*. 2011;17(2):79-84.

11. Hamilton WG. Foot and ankle injuries in dancers. *Clin Sports Med*. 1988;1:143-173.
12. Hamilton WG, Geppert MJ, Thompson FM. Pain in the posterior aspect of the ankle in dancers: differential diagnosis and operative treatment. *J Bone Joint Surg Am*. 1996;78(10): 1491-1500.
13. Hedrick MR, McBryde AM. Posterior ankle impingement. *Foot Ankle Int*. 1994;15(1):2-8.
14. Howse AJ. Posterior block of the ankle joint in dancers. *Foot Ankle*. 1982;3(2):81-84.
15. Jaivin JS, Ferkel RD. Arthroscopy of the foot and ankle. *Clin Sports Med*. 1994;13:761-783.
16. Koulouris G, Connell D, Schneider T, Edwards W. Posterior tibio-talar ligament injury resulting in posteromedial impingement. *Foot Ankle Int*. 2003;24(8):575-583.
17. Lawson JP. Symptomatic radiographic variants in extremities. *Radiology*. 1985;157(3):625-631.
18. Liu SH, Mirzayan R. Posteromedial ankle impingement: case report. *Arthroscopy*. 1993;9(6):709-711.
19. Liu SH, Raskin A, Osti L, et al. Arthroscopic treatment of anterolateral ankle impingement. *Arthroscopy*. 1994;10(2):215-218.
20. Lohrer H, Arentz S. Posterior approach for arthroscopic treatment of postero-lateral impingement syndrome of the ankle in a top-level field hockey player: case report. *Arthroscopy*. 2004;20(4):e15-e21.
21. Marotta JJ, Micheli LJ. Os trigonum impingement in dancers. *Am J Sports Med*. 1992;20(5):533-536.
22. Martin DF, Curl WW, Baker CL. Arthroscopic treatment of chronic synovitis of the ankle. *Arthroscopy*. 1989;5(2): 110-114.
23. Mellado JM, Rosenberg ZS, Beltran J, Colon E. The peroneocalcaneus internus muscle: MR imaging features. *AJR Am J Roentgenol*. 1997;69(2):585-588.
24. Mouhsine E, Crevoisier X, Leyvraz PF, et al. Post-traumatic overload or acute syndrome of the os trigonum: a possible cause of posterior ankle impingement. *Knee Surg Sports Traumatol Arthrosc*. 2004;12(3):250-253.
25. Nathan H, Gloobe H, Yosipovitch Z. Flexor digitorum accessorius longus. *Clin Orthop Relat Res*. 1975;(113):158-161.
26. Noguchi H, Ishii Y, Takeda M, et al. Arthroscopic excision of posterior ankle bony impingement for early return to the field: short-term results. *Foot Ankle Int*. 2010;31(5): 398-403.
27. Ogut T, Ayhan E, Irgit K, Sarikaya AI. Endoscopic treatment of posterior ankle pain. *Knee Surg Sports Traumatol Arthrosc*. 2011;19(8):1355-1361.
28. Paterson RS, Brown JN, Roberts SNJ. The posteromedial impingement lesion of the ankle. *Am J Sports Med*. 2001;29(5):550-557.
29. Regan TP, Hughston JC. Chronic ankle "sprain" secondary to anomalous peroneal tendon: a case report. *Clin Orthop Relat Res*. 1977;(123):52-54.
30. Robinson P, White LM, Salonen D, Ogilvie-Harris D. Anteromedial impingement of the ankle: using MR arthrography to assess the anteromedial recess. *AJR Am J Roentgenol*. 2002;178(3):601-604.
31. Rosenberg ZS, Cheung YY, Beltran J, et al. Posterior intermalleolar ligament of the ankle: normal anatomy and MR imaging features. *AJR Am J Roentgenol*. 1995;165: 387-390.
32. Seipel R, Linklater J, Pitsis G, Sullivan M. The peroneocalcaneus internus muscle: an unusual cause of posterior ankle impingement. *Foot Ankle Int*. 2005;26(10):890-893.
33. Sobel M, Levy ME, Bohne WH. Congenital variations of the peroneus quartus muscle: an anatomic study. *Foot Ankle*. 1990;11(2):81-89.
34. van Dijk CN, Bossuyt PM, Marti RK. Medial ankle pain after lateral ligament rupture. *J Bone Joint Surg Br*. 1996;78(4):562-567.
35. van Dijk CN, Scholten PE, Krips R. A 2-portal endoscopic approach for diagnosis and treatment of posterior ankle pathology. *Arthroscopy*. 2000;16(8):871-876.
36. White AA III, Johnson D, Griswold DM. Chronic ankle pain associated with the peroneus accessorius. *Clin Orthop Relat Res*. 1974;(103):53-55.
37. Willits K, Sonneveld H, Amendola A, et al. Outcome of posterior ankle arthroscopy for hindfoot impingement. *Arthroscopy*. 2008;24(2):196-202.
38. Yilmaz C, Eskandari MM. Arthroscopic excision of the talar Stieda's process. *Arthroscopy*. 2006;22(2):225.e1-225.e3.
39. Zammit J, Singh D. The peroneus quartus muscle: anatomy and clinical relevance. *J Bone Joint Surg Br*. 2003;85(8): 1134-1137.