CHAPTER

7

THE LATERAL ANKLE LIGAMENTOUS COMPLEX COULD BE THE "MISSING LINK" IN CLINICAL POSTERIOR ANKLE IMPINGMENT

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The *lateral ankle ligamentous complex could be the "missing link" in clinical posterior ankle impingment*

Posterior ankle impingement in athletes: Pathogenesis, imaging features and differential diagnoses.
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Hayashi D, Roemer F, D'Hooghe P, Guermazi A

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ABSTRACT

Posterior ankle impingement is a clinical diagnosis which can be seen following a traumatic hyper-plantarflexion event and may lead to painful symptoms in athletes such as female dancers (‘en pointe’), football players, javelin throwers and gymnasts.
Symptoms of posterior ankle impingement are due to failure to accommodate the reduced interval between the postero-superior aspect of the talus and tibial plafond during plantar flexion, and can be due to osseous or soft tissue lesions. There are multiple causes of posterior ankle impingement. Most commonly, the structural correlates of impingement relate to post-traumatic synovitis and intra-articular fibrous bands-scar tissue, capsular scarring, or bony prominences. The aims of this pictorial review article is to describe different types of posterior ankle impingement due to traumatic and non-traumatic osseous and soft tissue pathology in athletes, to describe diagnostic imaging strategies of these pathologies, and illustrate their imaging features, including relevant differential diagnoses.

INTRODUCTION

The term “impingement” represents painful limitation of motion. Posterior ankle impingement is a clinical diagnosis which may complicate an acute traumatic hyper-plantar flexion event or may relate to repetitive low-grade trauma associated with hyper-plantar flexion, e.g., in female dancers (‘en pointe’ or the‘demi-pointe’), downhill running, football players, javelin throwers and gymnasts[1-3]. The forceful plantar flexion that occurs during these activities produces compression at the posterior aspect of the ankle joint and can put extreme pressure on the anatomic structures normally present between the calcaneus and the posterior part of the distal tibia. Through exercise, the joint mobility and range of motion may gradually increase, progressively reducing the distance between the calcaneus and the posterior portion of the distal tibia[4].

Symptoms of posterior ankle impingement are due to failure to accommodate the reduced interval between the postero-superior aspect of the talus and tibial plafond during plantarflexion[5]. There are multiple causes of posterior ankle impingement. These include bony lesions, posteromedial and posterolateral soft tissue lesions, and anomalous and accessory muscles.

Multimodality imaging including radiography, CT, ultrasound and MRI is useful for assessing the structural correlates of ankle impingement. MRI is particularly

valuable for identifying or rule out other causes of persistent ankle pain that may mimic or coexist with ankle impingement, like for example occult fractures, cartilage damage, intra-articular bodies, osteochondral talar lesions, tendon abnormalities, and ankle instability. MRI features supportive of impingement maybe present in asymptomatic individuals and therefore accurate diagnosis requires careful clinical correlation.

The aims of this pictorial review article are to describe different types of posterior ankle impingement due to traumatic and non-traumatic osseous and soft tissue pathology, and to describe diagnostic imaging strategies of these pathologies and illustrate their imaging features, including relevant differential diagnoses.

Anatomy relevant to posterior ankle impingement

In athletes presenting with posterior ankle impingement symptoms, radiologists should pay specific attention to the presence of os trigonum, Stieda process (posterolateral talar process) (Fig. 1), posterior capsule and the posterior talofibular, intermalleolar, and tibiofibular ligaments (Fig. 2), and the flexor hallucis longus tendon[6]. Additionally, posteromedial tibiotalar capsule and posterior deltoid fibers (specifically those of the posterior tibiotalar ligament between the talus and medial malleolus) should be assessed for abnormality related to posteromedial impingement[7] (Fig. 3). In the diagnostic report, in addition to standard dictation based on a generic template used in each institution, the above structures should be specifically mentioned to describe whether they are normal or abnormal.

For interested readers, the Radiological Society of North America Radiology Reporting Initiative published a clear and concise structured report template for MRI of the ankle Fig. 3. Schematic illustration of the medial view of the ankle. [Table 1] (available online at http://www.radreport.org/template/0000041). Note; however, this template is not specifically tailored for posterior ankle impingement.

One should add description of specific details related to aforementioned anatomical structures when reporting a posterior ankle impingement case.

Table 1
MRI ankle structured reporting template, published by the Radiological Society of North America Radiology Reporting Initiative (available online at http://www.radreport.org/template/0000041).

MR ankle

[Right ankle | left ankle]
Clinical Information []
Comparison [None]

Findings
Alignment
[Normal | Talo-navicular uncovering | talar fault | hindfoot valgus]

Medial compartment
Medial malleolus: [Normal]
Posterior tibial tendon: [Normal]
Flexor digitorum longus: [Normal]
Deltoid ligament complex – superficial: [Intact]
Deltoid ligament complex – deep: [Intact]
Spring (plantar calcaneo-navicular) ligament: [Intact]

Lateral compartment
Lateral malleolus: [Normal]
Retromalleolar groove: [flat | concave | convex]
Peroneus longus: [Normal]
Peroneus brevis: [Normal]
Peroneal retinaculum: [Intact]
Peroneus quartus: [Present]
Anterior inferior tibiofibular ligament: [Intact]
Posterior inferior tibiofibular ligament: [Intact]
Anterior talofibular ligament: [Intact]
Calcaneofibular ligament: [Intact]
Posterior talofibular ligament: [Intact]

Posterior compartment
Posterior talus: [Normal]
Flexor hallucis longus: [Normal]
Intermalleolar ligament: [Intact]
Achilles tendon: [Normal]
Plantar fascia: [Normal]

Anterior compartment
Anterior tibial tendon: [Normal]
Extensor hallucis longus: [Normal]
Extensor digitorum longus: [Normal]
Peroneus tertius: [Present]

Joints
Tibiotalar joint [Normal]
Subtalar joint: [Normal]
Tarsal joints: [Normal]

Other findings
Bones (other than subarticular marrow): [Normal]
Muscles: [Normal]
Tarsal tunnel: [Normal]
Sinus tarsi: [Normal]

Impression []

Imaging strategies for posterior ankle impingement

The first step in the imaging assessment of posterior ankle impingement is to identify anatomical variants using conventional radiography. Routine anteroposterior (AP) ankle view typically do not reveal abnormalities related to posterior impingement. On the lateral view, a prominent Stieda's process or os trigonum may be identified in the posterolateral aspect of the ankle. This posterolateral part is often superimposed on the medial talar tubercle on the lateral projection and thus visualization of an os trigonum on a standard lateral view may not be possible.
Likewise, calcifications can sometimes not be detected by this standard lateral view. Obtaining additional lateral radiographs with the foot in 25◦of external rotation in relation to the standard lateral projection may be helpful to identify these[4].

Differentiation between an accessory ossicle and a fracture may be difficult or impossible solely based on radiographs, but in general the surfaces of a fracture appear irregular on radiographs and CT[8].

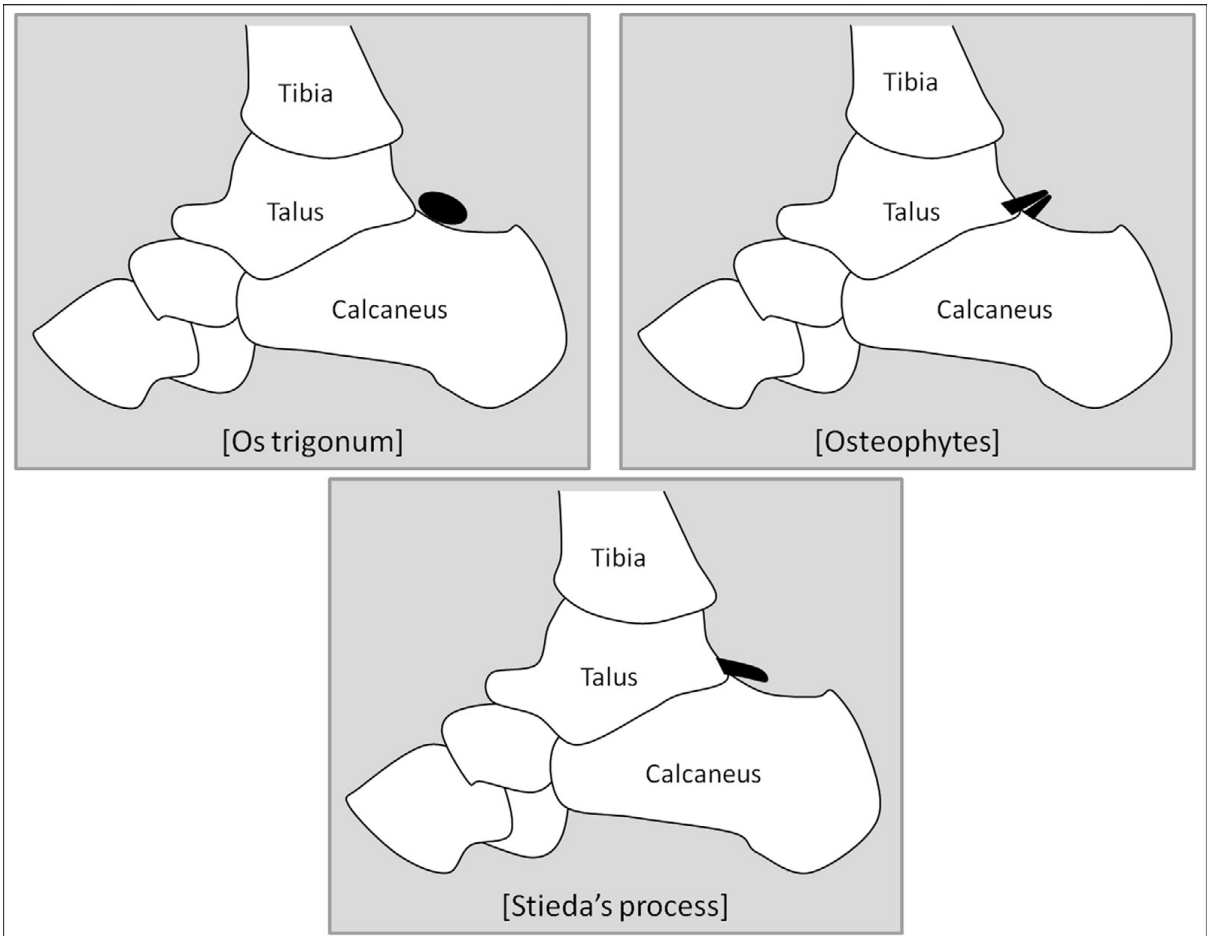

Fig. 1. Schematic illustration of os trigonum, osteophytes and Stieda's process.

CT allows evaluation of anatomical details of osseous structures of the posterior ankle and detection of fractures, loose bodies and osteochondral lesions that may be associated with posterior impingement. 3D surface reformations will aid orthopaedic surgeons during surgical planning. MRI plays an important role because of its excellent soft-tissue delineation. MRI protocols should include at least one fat-suppressed T2-weighted (T2W) or proton density-weighted (PDW)sagittal sequence (Figs. 4 and 5), and the ankle should be imaged in three planes. An example of ankle MRI protocol for assessment of impingement includes sagittal and coronal T1-weighted(T1W) spin-echo (SE) and short tau inversion recovery (STIR) or fat-suppressed PDW images, plus axial T2W or PDW turbo spin-echo sequences[9]. A combination of PDW and fat-suppressed PDW sequences may also be used. MRI in the sagittal plane using T1-weighted and fat-suppressed PDW or STIR sequences can afford optimal visualization of an os trigonum, a Stieda process, a downward sloping posterior malleolus, or a prominent calcaneal tubercle[8].

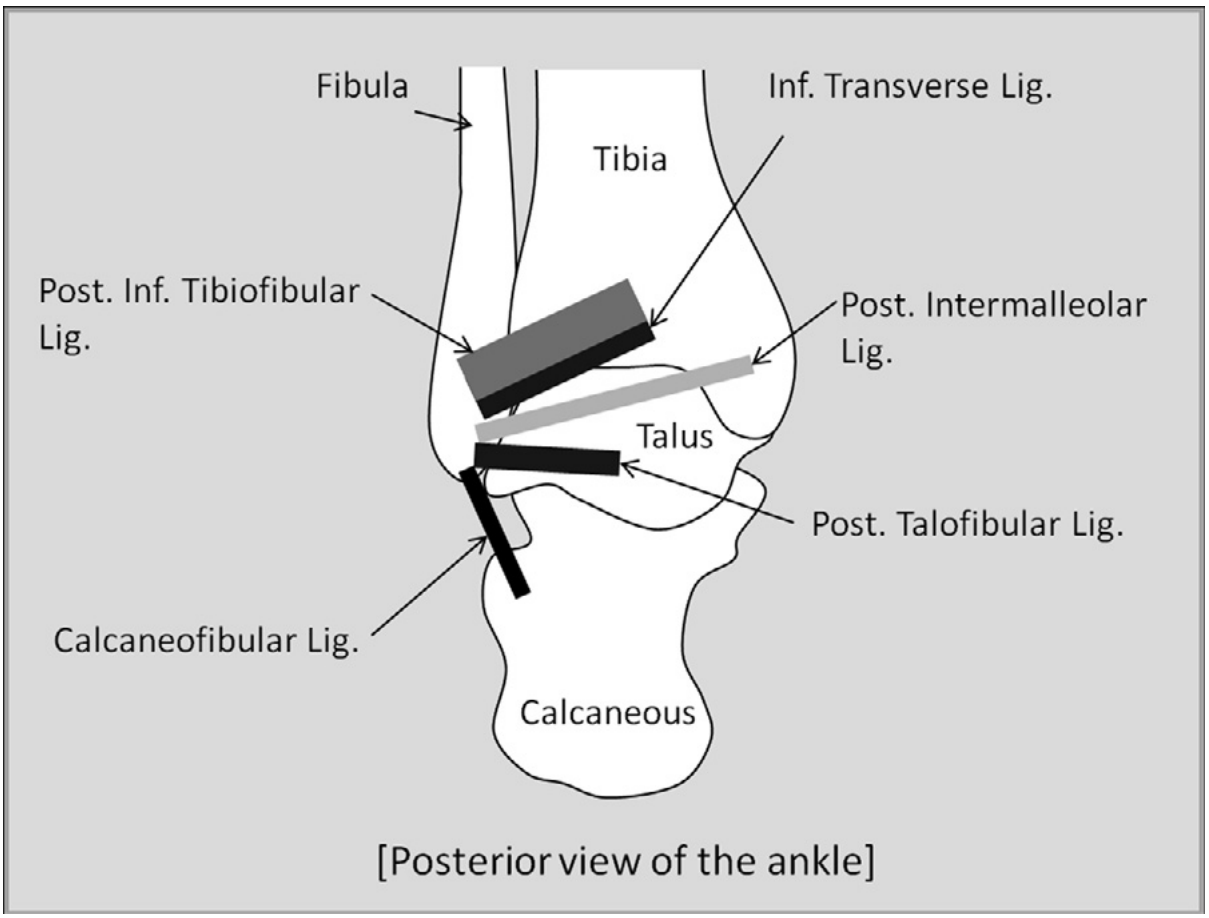

Fig. 2. Schematic illustration of the posterior view of the ankle.

By imaging in neutral and in plantar flexion, one may appreciate interposition or abutment of these osseous structures at the posterior ankle, then determine if there is motion at the synchondrosis of the os trigonum or at a pseudarthrosis or non-united fracture[10]. Associated bone marrow oedema may be present in the region of an os trigonum, about a non-united fracture of the lateral talar tubercle, or at a prominent, downward sloping lip of the posterior malleolus.

The combined presence of bone marrow oedema and posterior ankle synovitis, best demonstrated using STIR or fat-suppressed PDW sequences, may suggest the diagnosis of posterior ankle impingement[8]. It is equally important that MRI can specifically identify the wide range of pathology that may contribute to posterior ankle pain that might be clinically mimic posterior impingement. These include Achilles tendonitis/tear, ankle or subtalar arthritis, flexor hallucis longus tenosynovitis, Haglund's deformity, osteochondral lesion, retrocalcaneal bursitis, peroneal tendon subluxation, post-traumatic instability, sprain, or tarsal tunnel syndrome[8]. In the assessment of posterior ankle pain, use of intravenous gadolinium enhanced fat-suppressed T1W SE images, acquired in

the sagittal and axial planes, is helpful, since contrast-enhanced MRI is the ideal way of detecting synovitis and enables easier differentiation of inflamed synovium from effusion[11] (Fig. 6). This is particularly important in foot and ankle MRI because high T2W/STIR signal in the flexor hallucis longus tendon sheath may be secondary to an effusion within the tibiotalar joint rather than a primary flexor hallucis longus tenosynovitis. Likewise, high T2W/STIR signal in the peroneal tendon sheath may be a reflection of communication with the tibiotalar joint following a lateral collateral ligament injury[9].

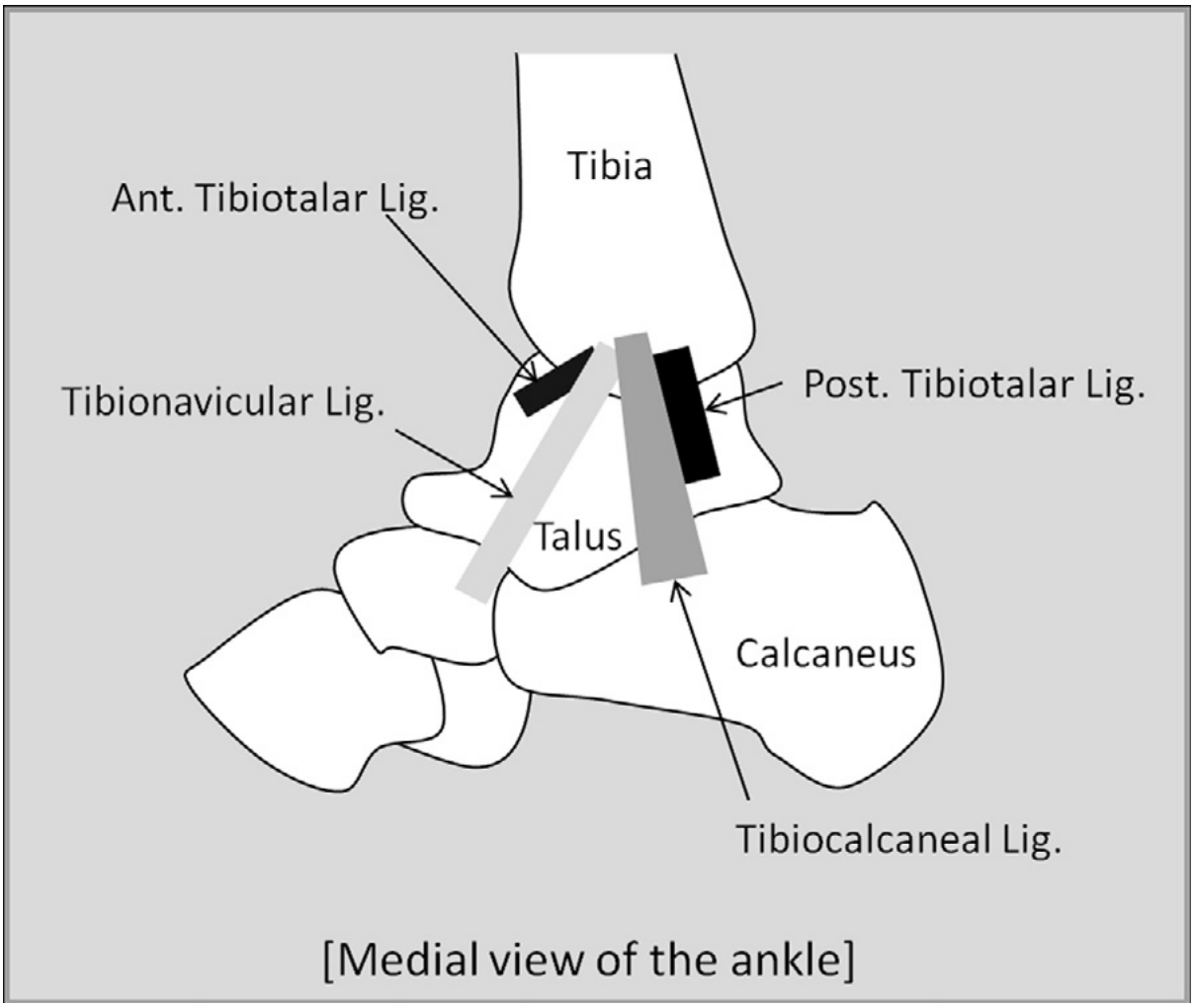

Fig. 3. Schematic illustration of the medial view of the ankle.

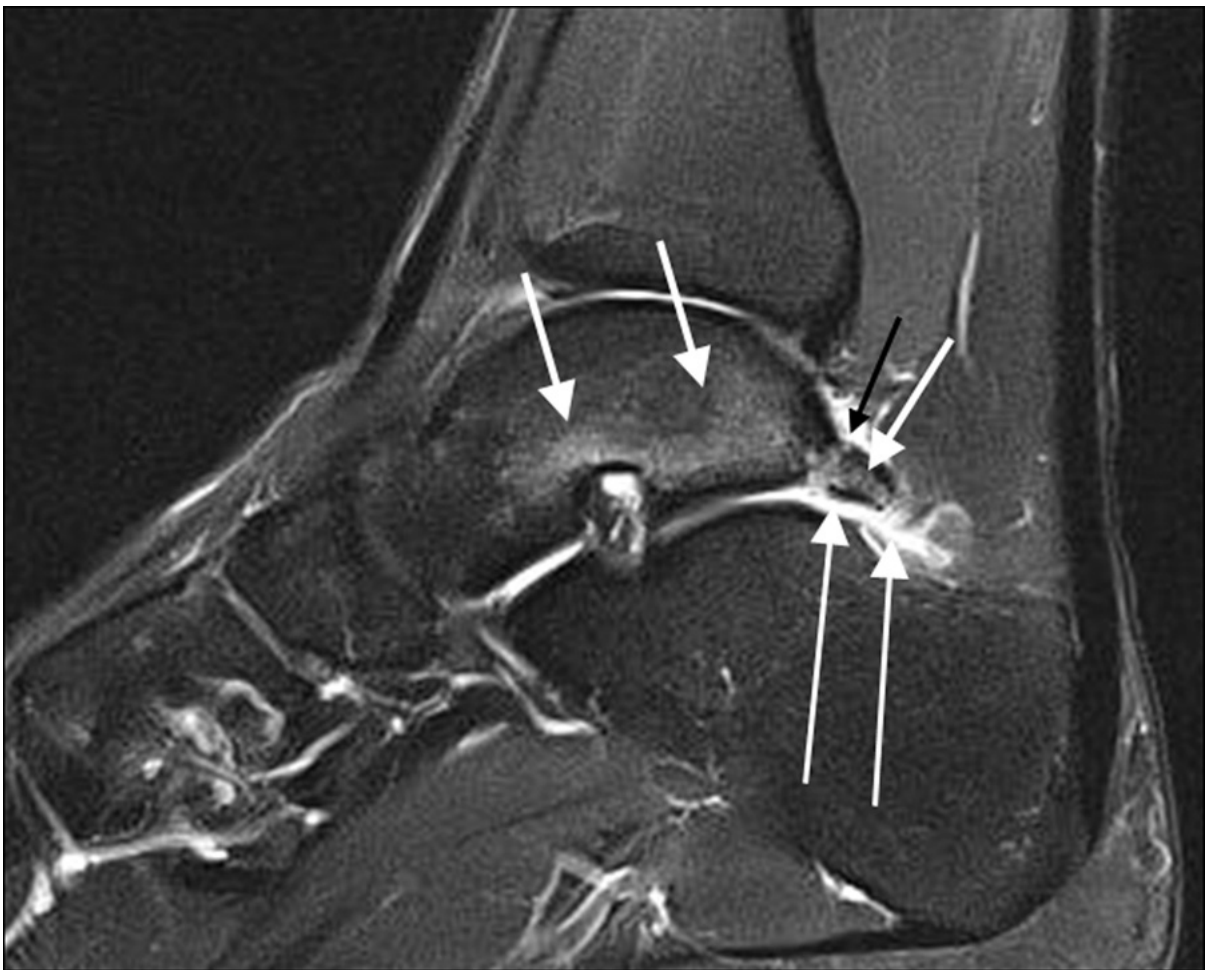

Fig. 4. A 25-year-old female ballet dancer with posterior ankle impingement due to presence of an Os trigonum. Sagittal proton density-weighted fat-suppressed MRI shows moderate bone marrow oedema in the Os trigonum but also the talar body(short white arrows). There is fluid-equivalent signal at the synchondrosis indicating a partial destabilization (black arrow). In addition there is marked synovitis surrounding the Os trigonum and extending to the subtalar joint consistent with an acute inflammatory response due to chronic irritation of the posterior subtalar joint and surrounding structures (long white arrows).

Application of 3D isotropic acquisition (3D gradient echo type sequences such as Spoiled Gradient Echo and Balanced Steady-state Free Precession, and 3D fast spin echo sequences) for imaging of ankle joint has been described in the literature[12,13] but their specific application to imaging of posterior ankle impingement has not been well established. Ultrasound has a main role as a tool for ultrasound-guided therapeutic injection of steroids and anaesthetics into a posterolateral capsule abnormality in athletes with clinical posterior impingement[14]. In most patients, ultrasound will show hypoechoic, nodular capsular thickening localized to the lateral aspect of the lateral talar process or the os trigonum[7].

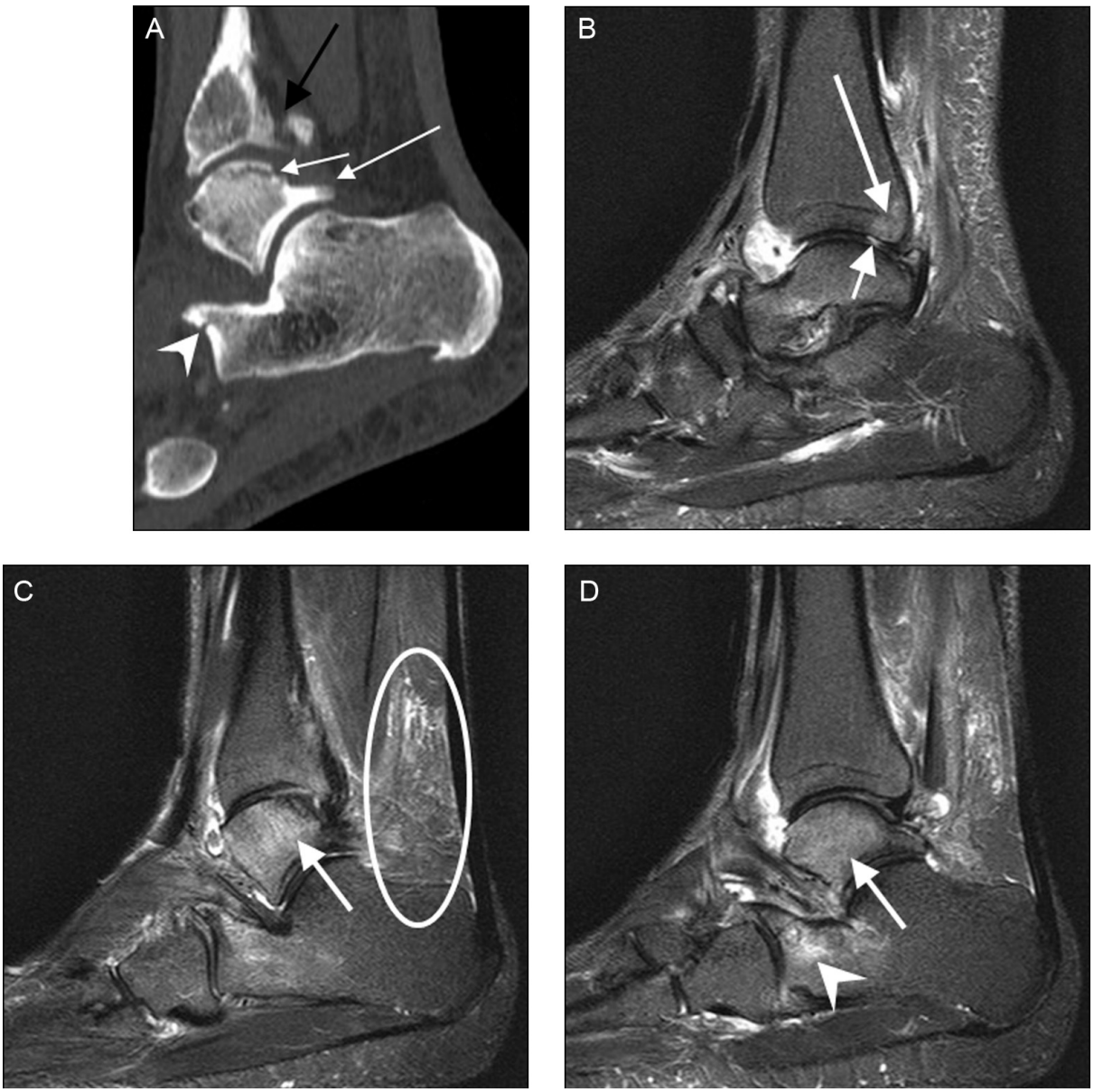

Fig. 5. A 28-year-old male football player. (A) Sagittal reformatted CT image of the ankle shows an intra-articular fracture (short white arrow) adjacent to the Stieda's process (long white arrow) associated with non-displaced fracture of the anterior process of the calcaneus (arrowhead) and distal fibula (black arrow). (B–D) Sagittal proton density-weighted fat-suppressed MRI demonstrates the osteochondral nature of the posterolateral fracture of the talus with large bone marrow oedema of the talus (white arrow, C and D). There is a posterior focal cartilage defect of the tibia (short white arrow, B) with subchondral bone marrow oedema (long white arrow, C). There is a Kager's fat pad synovitis (oval). The oedema in the calcaneal bone (arrowhead, D) is secondary to a small fracture that is shown on the CT (arrowhead, A).

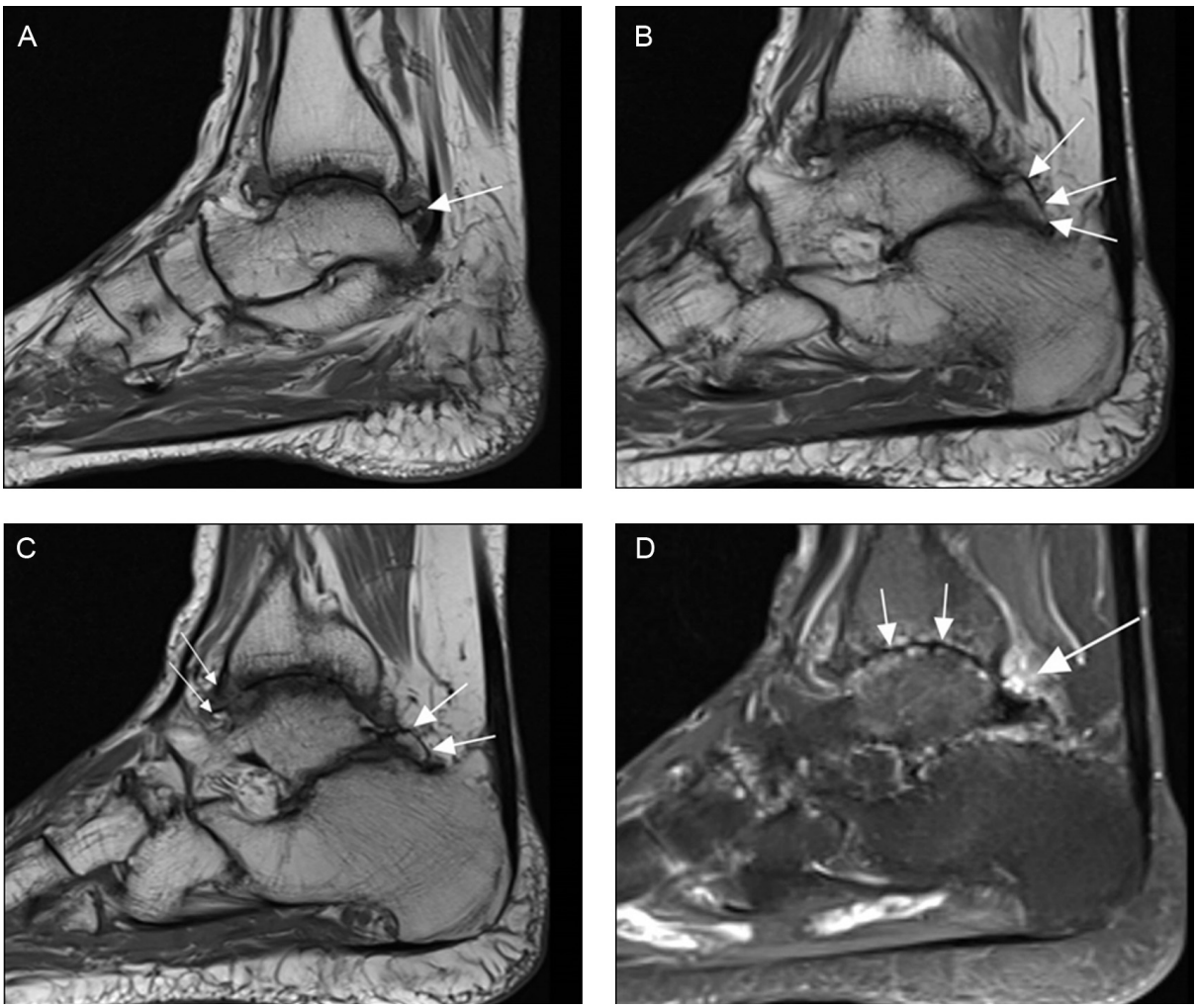

Fig. 6. A 58-year-old male active runner with advanced post-traumatic osteoarthritis of the ankle joint and posterior pain. (A) Sagittal T1-weighted MRI shows advancedosteoarthritis of the tibio-talar joint and a large posterior talar osteophyte that impinges into the posterior joint capsule especially on plantar flexion (arrow). (B) An additionallarge osteophyte is seen at the posterior margin of the talus leading to impingement at the calcaneo-talar joint (arrow). (C) The osteophyte reaches far laterally (large arrows)and in addition anterior tibia-talar osteophytes are observed (small arrows). (D) Sagittal contrast enhanced T1-weighted fat-suppressed MRI shows that reactive synovitis isprimarily observed posteriorly (large arrow), which correlates well with the clinical picture. There is complete obliteration of the tibiotalar joint (small arrow).

Nuclear medicine bone scintigraphy has been used as an adjunct to radiography since it shows increased radiotracer uptake due to hyperemia and bone repair in the posterior ankle in the setting of impingement[15].
Although highly sensitive, bone scintigraphy lacks specificity and cannot distinguish between radiotracer uptake related to fracture, pseudarthrosis, bone contusion, or posterior subtalar arthritis[8]. In comparison to bone scintigraphy, SPECT-CT allows superior anatomical correlation of radiotracer activity and symptoms. Use of SPECT-CT has been described for diagnosis of os trigonum syndrome in an athlete with posterior ankle pain[16].

Bony lesions

The bony structures responsible for posterior ankle impingement lie in the possible narrowing of the tibiocalcaneal interval and include os trigonum, posterolateral talar process (Stieda's or trigonal process), osteophytes, loose bodies, posterior malleolus, posterior subtalar joint and posterior calcaneal tuberosity (Fig. 1).

Os trigonum

Os trigonum is an ossicle located posterior to the talus[17]. The reported prevalence of the os trigonum is quite variable, from 7 to25%[18,19]. Os trigonum is ideally assessed on lateral radiographs of the foot and ankle and may be round, oval or triangular and may have a synchondrosis with the posterolateral talus[19]. On MRI, a small os trigonum may be sclerotic without central fatty marrow, and pericapsular fat may mimic os trigonum[5]. However, by performing both T1-weigted and fat-suppressed T2-weightedimaging this differentiation should be easy. A common differential for the os trigonum is a Shepherd fracture, which will be described later in this review. The presence of os trigonum may cause posterior impingement symptoms which may or may not be associated with a trauma. Symptoms may relate to destabilization of the cartilaginous synchondrosis of an os trigonum, compression between the os trigonum and tibia, or compression between os trigonum and calcaneus.

There may be entrapment of the adjacent soft tissues and secondary synovitis may develop, often centred on the posterior talofibular ligament. Synovitis may extend to involve the posterior recess of the ankle or the subtalar joint and the flexor hallucis longus tendon sheath. The synchondrosis of the os trigonum may vary in orientation from coronal to oblique sagittal plane. When present, synchondrosis between the talus and os trigonum should be evaluated for the presence of fluid signal intensity, indicating instability. Assessment should also be made for bone marrow edema at the synchondrosis margins and adjacent synovitis in the posterior recesses of the ankle and posterior subtalar joint, which may be a cause of posterior impingement symptoms (Fig. 4).

Sclerosis and cystic change at the synchondrosis margins indicates a degree of chronic stress across the synchondrosis. A small os trigonum may be seen only on a single axial or sagittal image. One should look for bone marrow signal on T1W or PDW sequences and assess for corticated margins.

Disruption of the os trigonum synchondrosis can be difficult to define on conventional MRI. In this situation, fluoroscopically guided arthrography of the synchondrosis will help define its integrity and also allow us to perform therapeutic intervention, like for example injection of local anesthetic and steroid[6,20]. It needs to be mentioned that an os trigonum per se in not a structure of pathologic relevance as long as it is asymptomatic.

Stieda's process fracture

Stieda's process is the posterolateral process of the talus. Acute fractures have been described eponymously as a Shepherd fracture, which may result in acute posterior impingement symptoms. Such fractures are particularly common in football players (Fig. 5). Nonunion of such fracture can result in chronic posterior impingement symptoms. It is best evaluated on sagittal MRI.

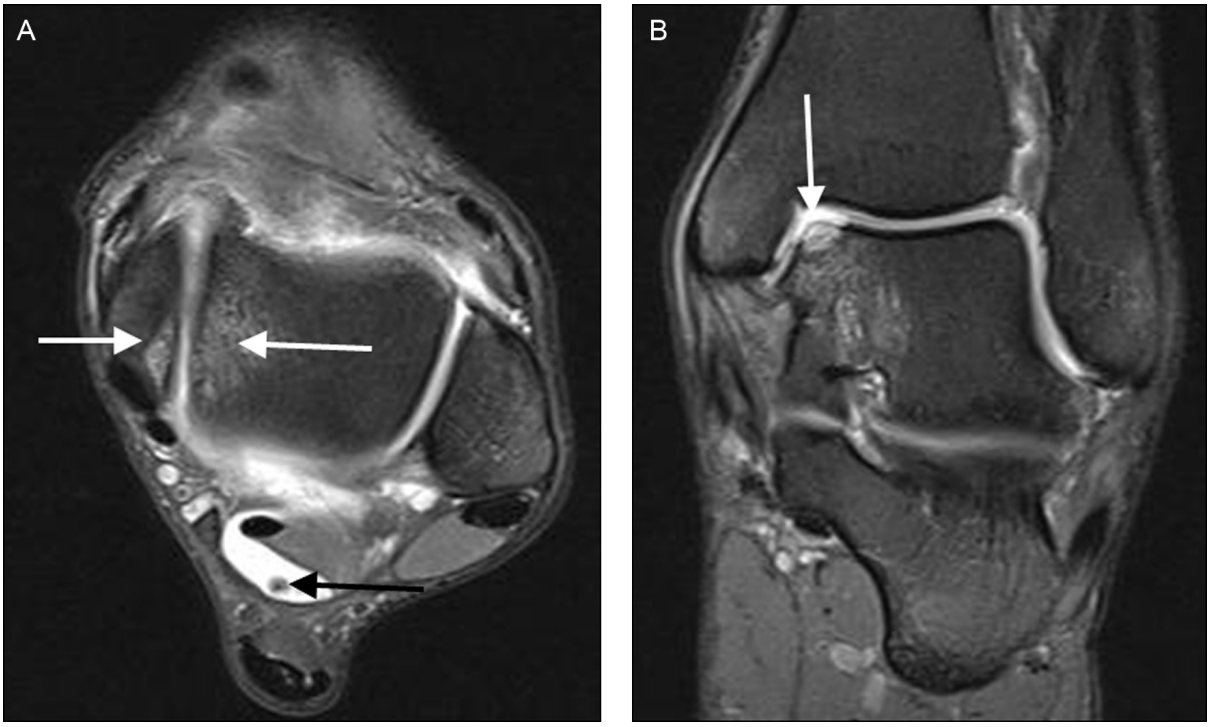

Fig. 7. A 29-year-old female gymnast with posteromedial impingement syndrome. (A) Axial proton density-weighted fat-suppressed MRI shows bone marrow oedema at the medial talar dome and malleolus medialis (white arrow). Note a small loose body posteriorly (black arrow) within a small fluid collection posterior to the flexor halluces longus tendon. (B) Coronal proton density-weighted fat-suppressed MRI shows an osteochondral lesion of the talus at the medial talar shoulder with full thickness cartilage loss (white arrow).

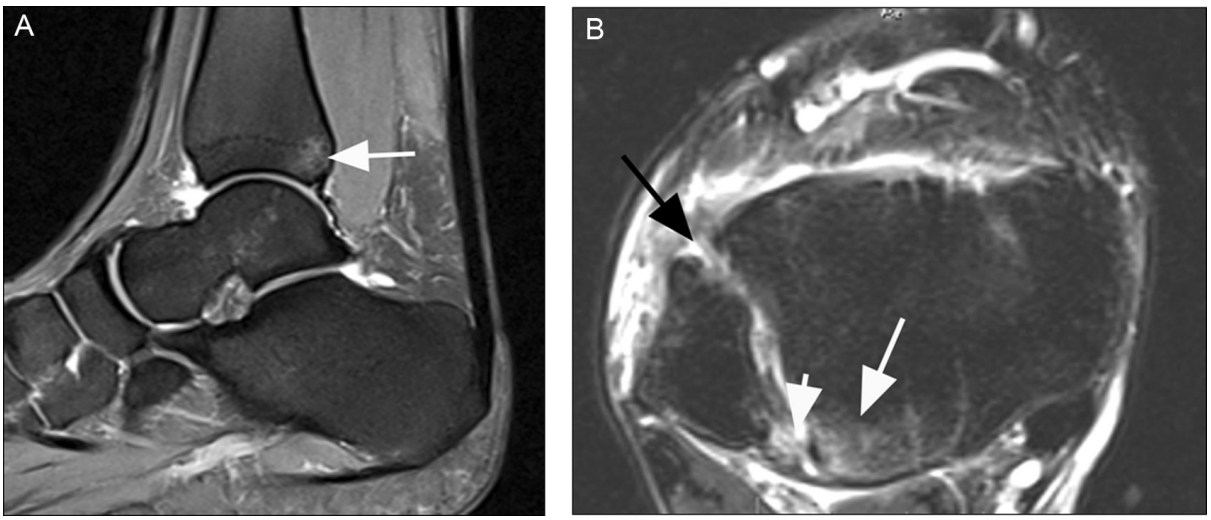

Fig. 8. A 22-year-old male football player with acute ankle sprain. (A) Sagittal proton density-weighted fat-suppressed MRI shows an avulsion oedema at the posterior periarticular tibia near the attachment of the posterior inferior tibio-fibular ligament (arrow). (B) Axial proton density-weighted fat-suppressed MRI shows interstitial oedema of the ligament itself (short white arrow). Injury to the posterior syndesmosis always involves the posterior aspect of the tibia (long white arrow). In addition there is a complete tear of the anterior syndesmotic ligament (black arrow) and surrounding oedema. Consecutive scarring of the posterior syndesmosis may lead to posterior impingement syndrome.

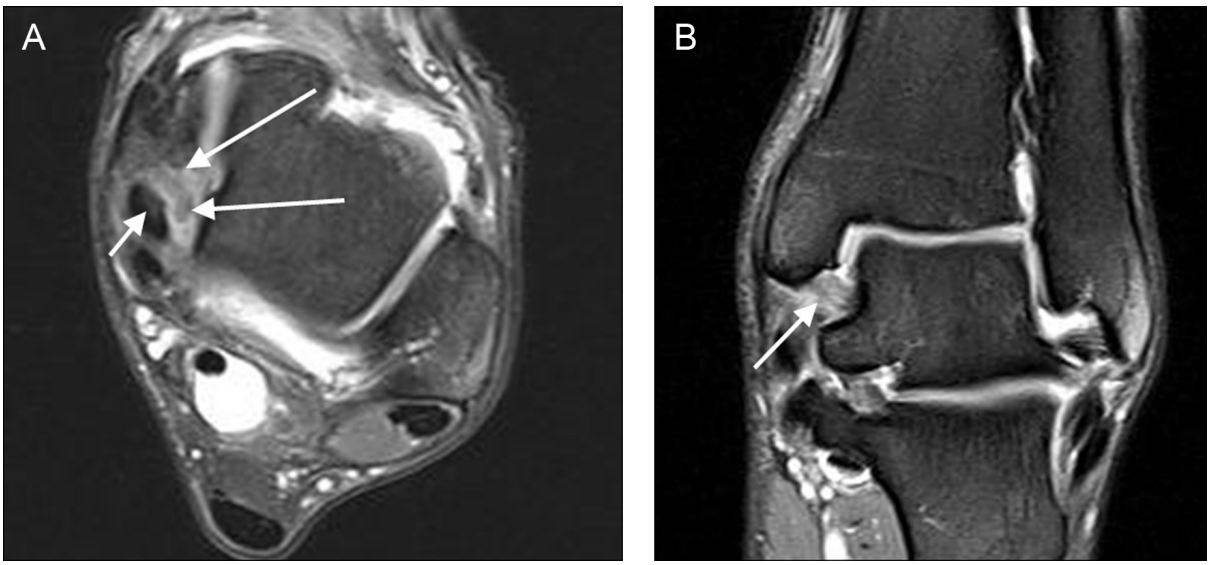

Fig. 9. A 24-year-old male marathon runner with posteromedial impingement. (A) Axial proton density-weighted fat-suppressed MRI shows marked synovitic changes around the tibialis posterior tendon (long white arrows). Tendon itself shows intra-tendinous signal change consistent with degeneration (short white arrow). (B) Coronal proton density-weighted fat-suppressed MRI shows hyperintensity of the posterior tibiotalar ligament (white arrow) consistent with ligament oedema and loss of normal striated morphology due to remote trauma and now clinical instability. In addition, there is marked surrounding hyperintensity representing concomitant synovitis.

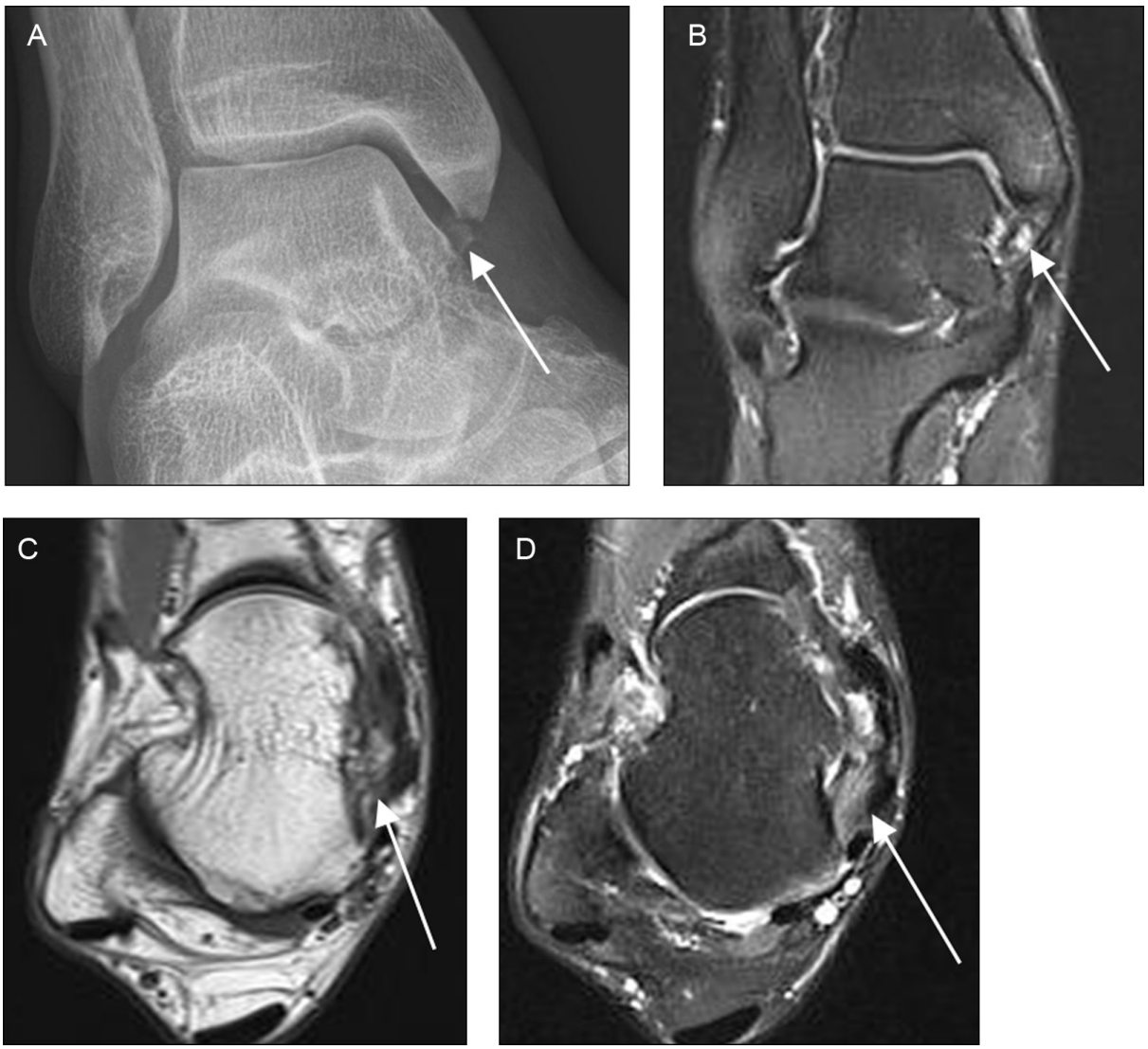

Fig. 10. A 33-year-old male rugby player with posteromedial impingement. (A) Anteroposterior X-ray shows an avulsion-fracture of the tip of the medial malleolus (arrow). (B) Coronal proton density-weighted fat-suppressed MRI shows bone marrow oedema at the tip of the medial malleolus and sprain of the posterior tibiotalar ligament(arrow). (C) Axial T1-weighted and (D) proton density-weighted fat-suppressed MRI show there is immature scarring of the posterior tibiotalar ligament and posttraumatic synovitis (arrow).

The Stieda's process is considered prominent if it extends posterior to the arc of curvature of the talar dome in the sagittal plane. Fractures of the posterolateral process are usually readily demonstrated on MRI[5]

Other bony lesions

These include osteophytes, talar osteochondral lesion and loose body, and posterior syndesmotic injury. These pathologic processes are shown in Figs. 6–8. Generally, osteophytes are the secondary manifestation of osteoarthritic changes[21,22]. However, repetitive minor trauma in the ankle can induce spur

formation, with radiographic features similar to osteophytes[4]. Loose bodies are frequently small and their presence should be confirmed in three planes on MRI. The deep fibers of the posteroinferior tibiofibular ligament, pericapsular fat and a fibrous band may mimic a loose body[5].

Posteromedial soft tissue lesions

Posteromedial soft tissue impingement is caused by entrapment of granulation tissue or fibrotic scar formations in the posteromedial ankle gutter — posterior tibiotalar ligament (deep, posterior component of the deltoid ligament) and posteromedial gutter synovitis and scar (Figs. 3, 9–11). The posteromedial gutter is a recess defined anteriorly by the posterior border of the medial malleolus and the posterior tibiotalar ligament. The posteromedial border of the talar dome-body and posteromedial process of the talus lie at the deep margin, and the posteromedial capsule lies at the superficial and posterior margin.

On MRI, the posteromedial gutter is normally identified as a small recess containing minimal fluid, with a thin overlying capsular layer, and is readily visualized on axial images as the recess lying deep to the interval between the flexor hallucis longus and the flexor digitorum longus tendons[5]. Axial MRI findings include loss of the normal striated appearance of the posterior tibiotalar ligament, protrusion of scar response and synovitis into the medial gutter posteriorly, loss of the normal clear space in the posteromedial gutter between the levels of the flexor digitorum longus and flexor hallucis longus tendons, and thickening of the posteromedial ankle capsule[5].

Concurrent injury to the flexor retinaculum may result in partial scar encasement of the posterior tibial tendon between the retinaculum and the scarred posterior tibiotalar ligament. Avulsion fractures of the posteromedial process of the talus can also cause posteromedial impingement, but the avulsion fragment often being difficult to identify on MRI, and CT scan may be required for confirmation[5].

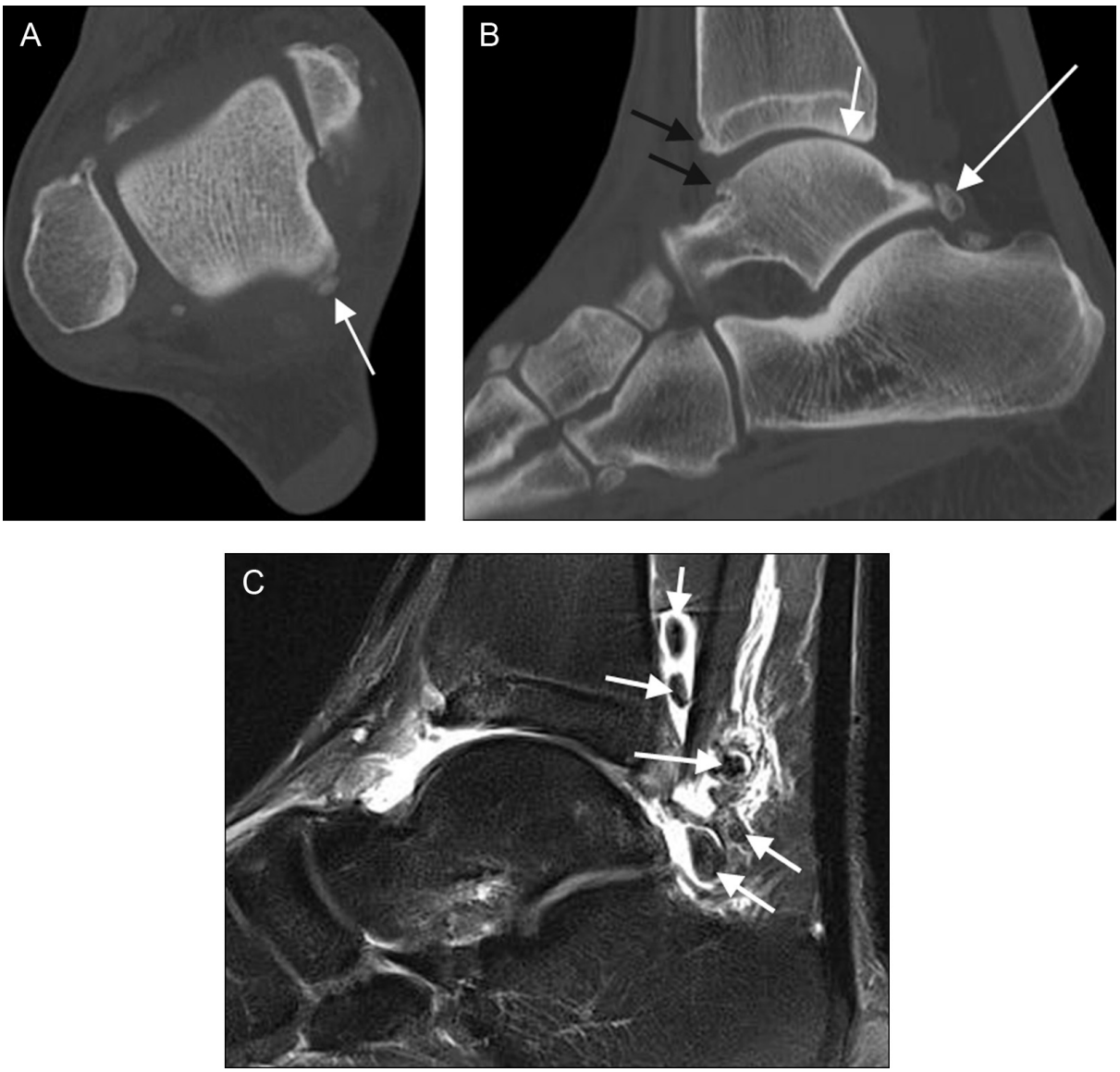

Fig. 11. A 40-year-old male triathlete with posteromedial impingement. (A) Axial CT image shows an old avulsion fracture at the posteromedial process of the talus at the insertion of the posterior tibiotalar ligament (arrow). (B) Sagittal CT reformat shows a prominent posterior talar process and adjacent loose body (long white arrow) possibly due to remote fracture. There are also osteophytic spurs at the anterior tibiotalar joint (black arrows) and joint space narrowing with subchondral sclerosis posteriorly indicating moderate osteoarthritis changes (short white arrow). (C) Sagittal proton density-weighted fat-suppressed MRI depicts multiple loose bodies (white arrows) with marked surrounding synovitis posteriorly and joint effusion in the tibiotalar joint.

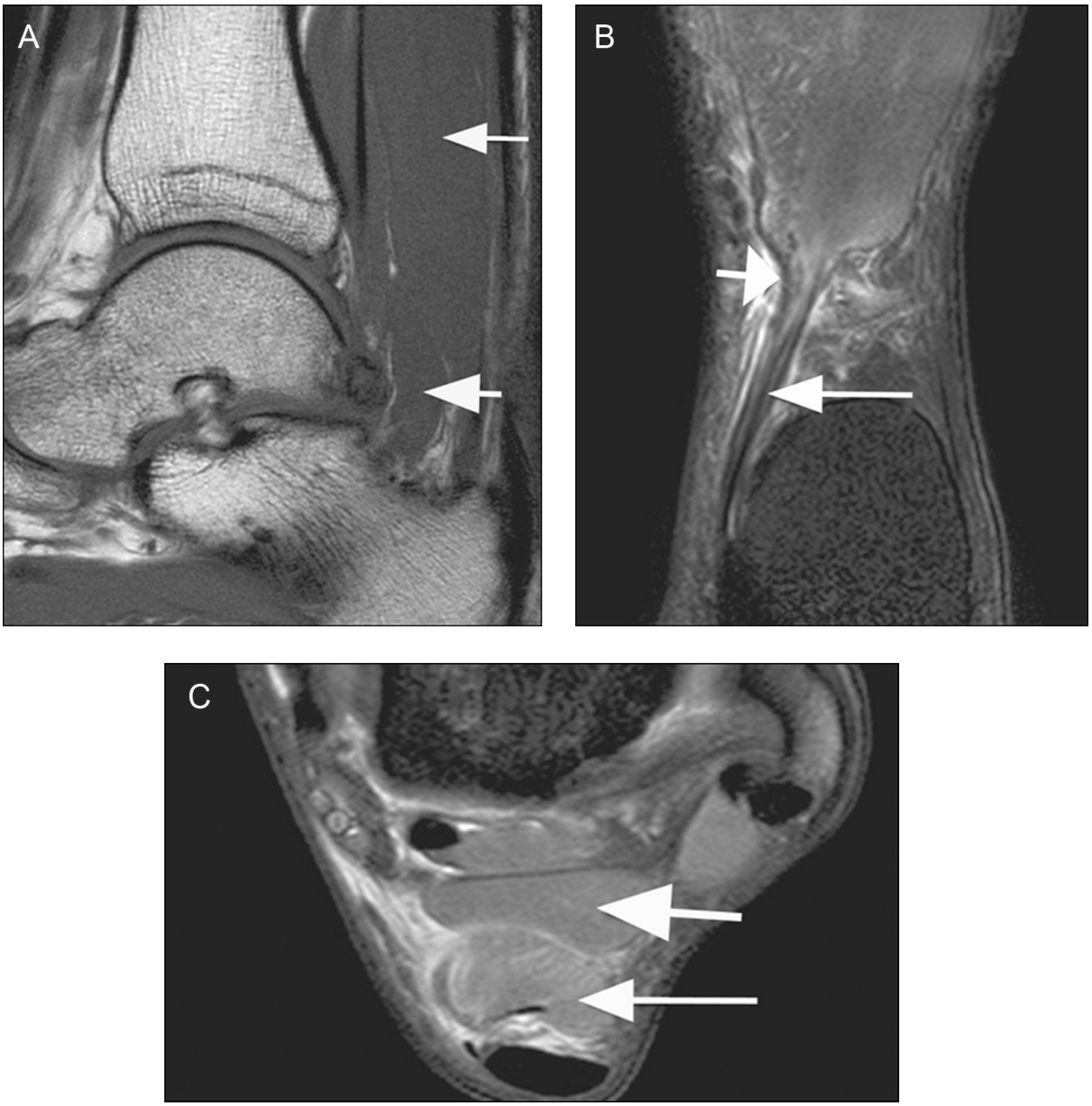

Fig. 12. A 27-year-old male football player with accessory muscles causing posterior ankle pain. (A) Sagittal T1-weighted MRI shows co-existence of an accessory soleus muscle (long thin arrow) and an accessory flexor digitorum longus muscle and tendon (short arrow). (B) Coronal proton density-weighted fat-suppressed MRI shows tendon of the accessory soleus muscle (long arrow) and oedema at the myotendinous junction (short arrow). (C) Axial proton density-weighted fat-suppressed MRI depicts a normal accessory flexor digitorum muscle (short arrow) and a strain at the myotendinous junction of the accessory soleus (long arrow).

Posterolateral soft tissue lesions

Posterolateral soft tissue impingement is caused by an accessory ligament, the posterior intermalleolar ligament (Fig. 10). 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. The posterior intermalleolar ligament may protrude further into the joint during plantarflexion, becoming entrapped and torn.

The resulting impingement consists of ankle locking and pain. The intermalleolar ligament is best assessed on axial and coronal MRI with the ankle in neutral position, since it usually cannot be identified as a separate structure in ankle plantar flexion[5]. Important MRI findings in the setting of posterior impingement due to intermalleolar ligament pathology are thickening and tear and adjacent synovial thickening, demonstrated as intermediate signal intensity tissue with ill-defined margins on PDW or fat-suppressed PDW sequences[5]. Frequently, this is the only sign of a non-acute tear. In contrast, redundant torn ligament and focal ligament fiber discontinuity maybe seen in the acute setting[5].

Anomalous and accessory muscles

These are unusual causes of posterior ankle impingement. Muscles that can cause impingement include peroneus quartus, flexor accessories digitorum longus, accessory soleus, peroneus-calcaneus internus muscle, tibiocalcaneus internus, and low-lying flexor hallucis longus muscle belly (Fig. 12)

Differential diagnoses

Pathologies that cause inflammatory changes in the posterior ankle can also cause posterior ankle pain and may mimic impingement syndromes. Such pathologies that can be seen in athletes include posterior capsulitis and rheumatoid arthritis (Figs. 13 and 14).

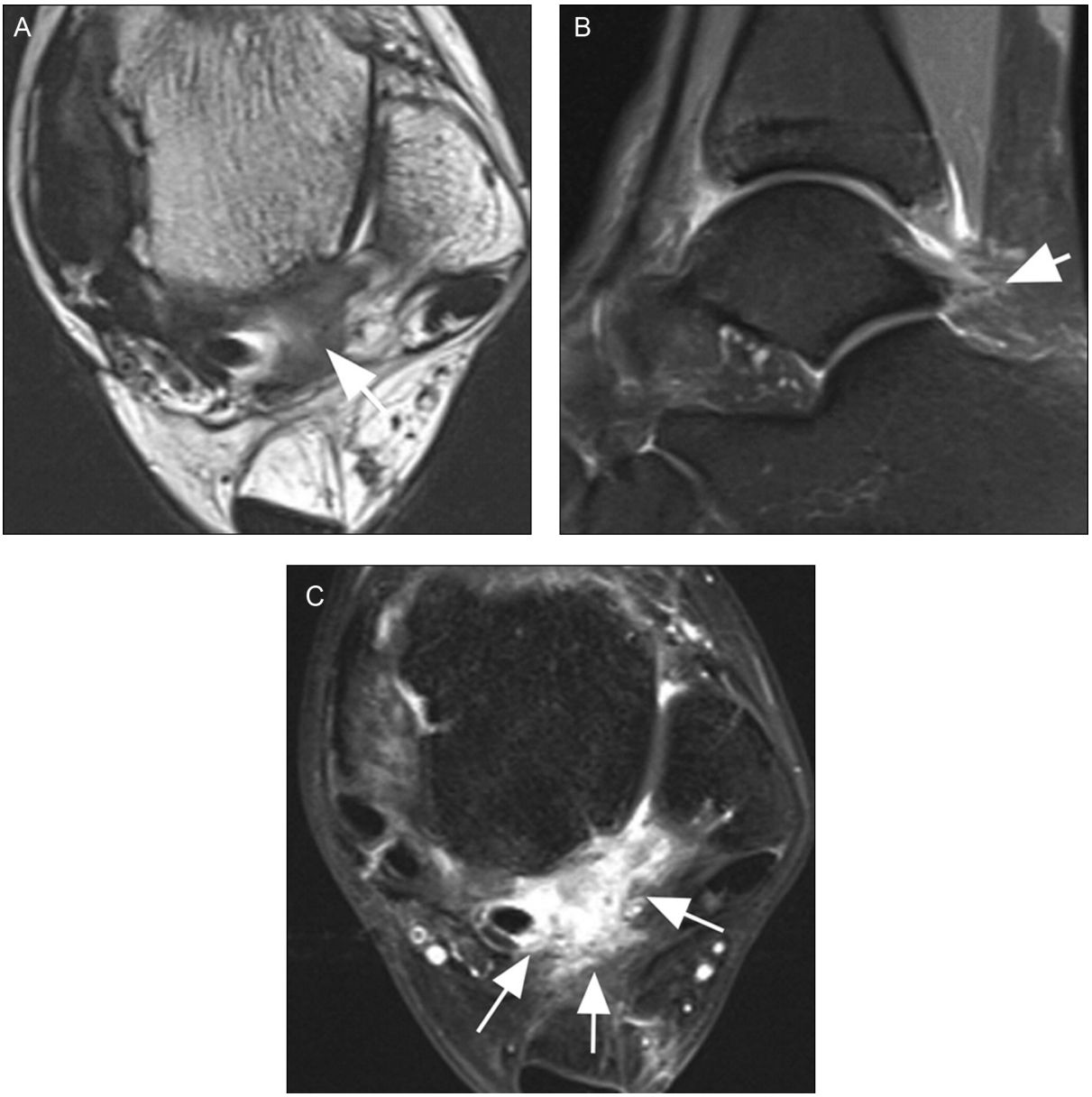

Fig. 13. A 23-year-old professional female dancer with posterior ankle pain. (A) Axial T2-weighted MRI shows diffuse hypointensity posteriorly to the talus likely representing circumscribed fibrosis (arrow). (B) Sagittal proton density-weighted fat-suppressed MRI depicts minimal hyperintensity in the same anatomical region (short arrow). (C)Axial contrast-enhanced T1-weighted fat-suppressed MRI shows intense enhancement demonstrating focal posterior capsulitis as the source of the pain (arrow). Note that no os trigonum or other anatomical predisposing structures are present in this patient.

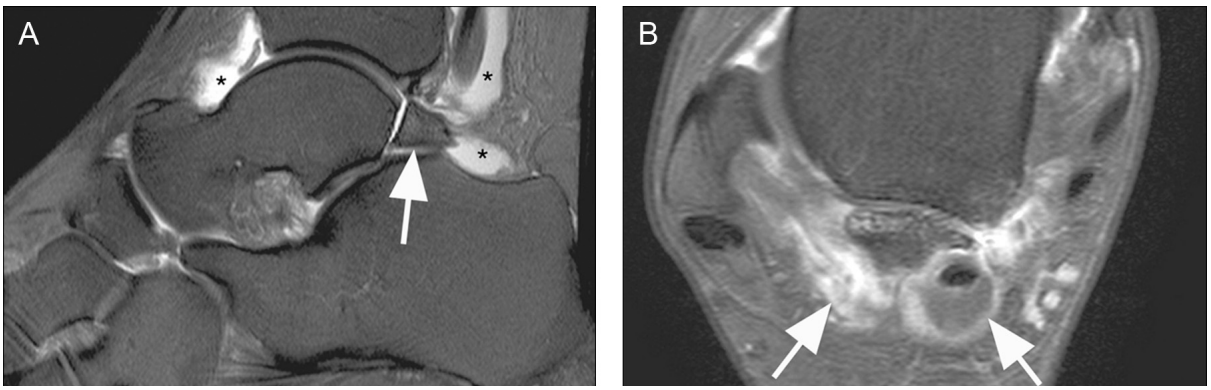

Fig. 14. A 46-year-old female active runner with chronic posterior pain suggesting posterior impingement syndrome. (A) Sagittal proton density-weighted fat-suppressed MRI shows an os trigonum (arrow) and marked synovitis and effusion posteriorly, anteriorly and around the flexor tendons (*). (B) Axial contrast-enhanced T1-weighted fat-suppressed MRI superiorly differentiates the synovial thickening from fluid (arrow). In this case the inflammation was due to diffuse synovitis of the ankle in recently diagnosed rheumatoid arthritis. Note almost normal signal within the os trigonum.

CONCLUSION

Ankle impingement syndromes are important causes of persistent ankle pain. There are multiple potential sites and etiologies of posterior impingement symptoms.

Most commonly, impingement lesions relate to post-traumatic synovitis and intra-articular fibrous bands-scar tissue, capsular scarring, or bony prominences, although radiologists need to be aware of even rare differential diagnoses such as posterior capsulitis, gouty tophus and rheumatoid arthritis. Multimodality imaging, particularly MRI, is useful in confirming the diagnosis, evaluating patients with an uncertain clinical diagnosis, and planning surgery. These pathologies are readily identified on MRI and, when present, should be interpreted as predisposing to impingement symptoms, accepting that the diagnosis of impingement is clinical.

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Posterior ankle arthroscopy: current state of the art
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ABSTRACT

The most common hindfoot pathologies seen in clinical practice and sports medicine are posterior ankle impingement and osteoarthrosis (OA). Both these – and other pathologies such as insertional tendinitis and Haglund's disease - may cause significant disability, in both everyday life and during sporting activities. Posttraumatic OA alone causes a healthcare burden of over 3 billion US dollars per year. An adequate approach of these pathologies is required to minimize this healthcare burden and additionally to maintain patients' economic productiveness.

The aim of this article is to outline the most important evidence-based indications concerning posterior ankle arthroscopy focusing on diagnostics, surgical techniques, complications, geographical differences and future developments in the field of hindfoot arthroscopy. Initially, the treatment of hindfoot pathology is conservative. If adequate conservative treatment does not result in a good response, surgery may be indicated. Over the last three decades, arthroscopy of the ankle joint has become a standardized and important procedure, with numerous indications for both anterior and posterior pathology.
Since 2000, a two-portal hindfoot arthroscopic approach has been described and used globally in clinical practice.

Some of the indications that may be addressed using this approach are the treatment of posteriorly located osteochondral defects, posterior ankle impingement, pathology of the deep portion of the deltoid ligament, Cedell fracture, tarsal tunnel release, loose bodies and tibiotalar or subtalar arthrodesis. Tendon pathology can also be treated using posterior portals; however, this is beyond the scope of this review.

HISTORY of ANKLE ARTHROSCOPY

The field of arthroscopic foot and ankle surgery has progressed tremendously since its inception in 1939.[3] Access to the posterior compartment of the ankle and subtalar joint historically has been performed in combination with a two-portal anterior approach, with the patient in the supine position.
A third posterolateral portal was used mainly for irrigation or for the introduction of a grasper in order to remove a loose body in the posterior compartment. A posteromedial portal was regarded as dangerous because of potential nerve damage and damage to the posterior tibial artery and

posteromedial tendons.[4] In 2000, van Dijk et al[5] developed a two-portal technique for hindfoot arthroscopy with the patient in the prone position. This approach is currently used as the standard approach for posterior pathology (figure 1). This technique provides excellent access to the posterior ankle compartment, subtalar joint and also the extra-articular structures, thus allowing for the inspection and treatment of posterior ankle pathology such as posterior ankle impingement and flexor hallucis longus (FHL) tendinopathy.[5-8] This approach can also be used to treat talar osteochondral defects (OCDs), removal of loose bodies or to perform arthroscopic ankle fusion, subtalar fusion or a combined ankle and subtalar fusion. Additional procedures are tarsal tunnel release and peroneal groove deepening for recurrent peroneal tendon dislocation. Ankle arthroscopy has expanded to become an important therapeutic technique in the management of disorders of the ankle joint.[3,9,10] As the indications for hindfoot arthroscopy have increased, so has its usage. It is the procedure of choice for the treatment of chronic and post-traumatic pathologies due to low morbidity rates, more rapid rehabilitation and favorable cosmetic results compared with conventional open surgical procedures.[8,11]

Reviews and state-of-the-art or current concept Articles

Box 1 highlights six articles that the authors profess to be key in the development of posterior ankle arthroscopy.

Box 1 Key articles on posterior ankle arthroscopy

- First description of the two-portal technique with the patient in the prone position by van Dijk *et al*[5] showing good results with a 2-year follow-up.
- In 2006, Pau Golanó published two articles providing detailed insight in the anatomy of the ankle with the focus on the salient facts for arthroscopists.[58 59]
- The low complication rate per technique was outlined by the research group of Zengerink *et al*.[23]
- A recent overview on causes, diagnosis, surgical technique, outcomes and complications is provided by Smyth *et al*.[18]
- In 2015, Hayashi *et al*[60] provided a radiographic overview on posterior ankle impingement, including the differentials that should be taken into account.
- To assess the safety of the posterior approach of ankle arthrodesis, Kerkhoffs *et al*[61] performed a cadaveric study to assess iatrogenic damage after posterior ankle arthroscopic procedure.

This article is the first state-of-the-art overview on ankle arthroscopy of the posterior ankle joint that discusses the diagnostics prior to arthroscopic treatment, the technique for posterior ankle arthroscopy, the complications, the pitfalls, any regional or geographical differences and future directions.

CURRENT STATE of the ART

Diagnostics

Thorough history taking and physical examination are the key to ensure a working hypothesis. For each pathology, specific indications may be found, which can be confirmed or excluded by means of history taking and physical examination (table 1).
A patient with subtalar pathology has deep ankle pain which cannot be easily reproduced by physical examination. Locking is a sign of a loose body. Hindfoot pain, which aggravates with plantar flexion, is typical for posterior impingement. Numbness is a sign of a tarsal tunnel syndrome. Each examination begins with inspection and malalignment must be looked for specifically. The location of the pain is an important indicator. On physical examination, it is important to look for recognizable tenderness on palpation (figure 2). Not all disorders of the hindfoot can be diagnosed on palpation, but recognizable tenderness over one of the tendons guides the diagnosis in the direction of a tendon disorder.

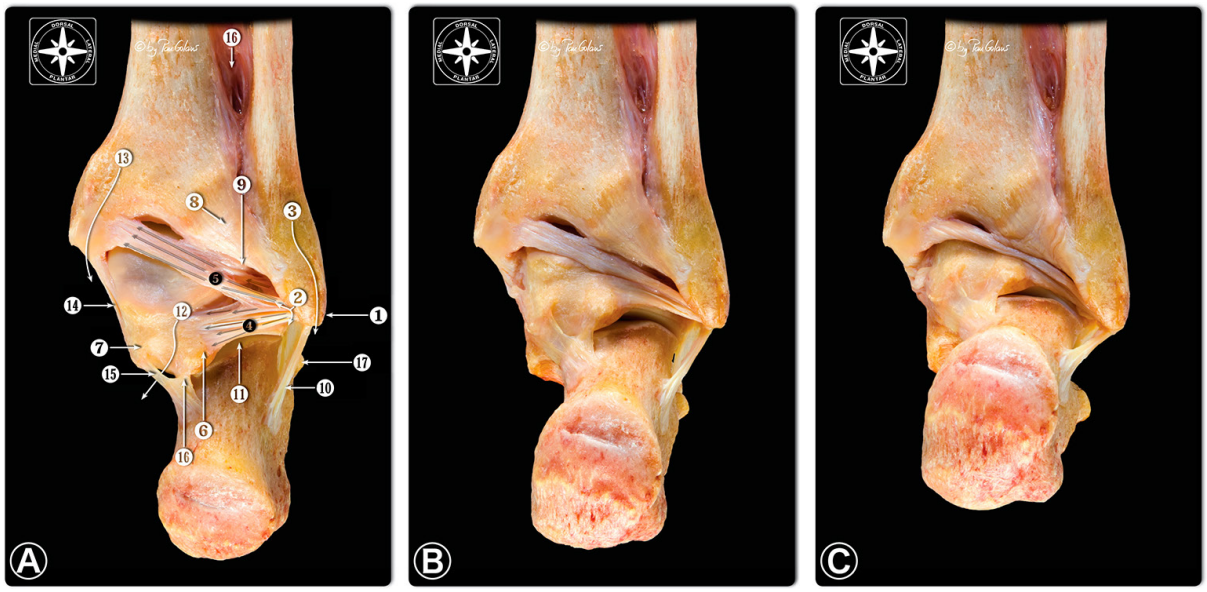

Figure 1. Posterior view of the anatomical dissection of the ankle ligaments showing the posterior intermalleolar ligament with its relation to the surrounding anatomy. The capsule was removed. (A) Dorsal flexion, (B) neutral position and (C) plantar flexion. The posterior intermalleolar ligament tenses during dorsiflexion and relaxes during plantar flexion. Trauma that causes forced dorsiflexion of the ankle can be assumed to produce injury to—or rupture of—this ligament or osteochondral avulsion. Plantar flexion would cause it to relax and become susceptible to trapping between the tibia and the talus, leading to impingement (from van Dijk, CN, Ankle Arthroscopy, 2014, Springer; reproduced with permission of van Dijk57) ©Dr. Pau Golano. 1. Lateral malleolus. 2. Malleolar fossa. 3. Peroneal groove of the fibula and peroneal tendons traject. 4. Posterior talofibular ligament. 5. Posterior intermalleolar ligament. 6. Lateral talar process. 7. Medial talar process. 8. Superficial component of the posterior tibiofibular ligament. 9. Deep component of the posterior tibiofibular ligament or transverse ligament. 10. Calcaneofibular ligament. 11. Subtalar joint. 12. Tunnel for the flexor hallucis longus tendon. 13. Tibialis posterior tendon traject. 14. Medial collateral ligament. 15. Flexor hallucis longus retinaculum. 16. Posterior talocalcaneal ligament. 17. Interosseous membrane.

The posterolateral talar process can be palpated on the posterolateral side of the ankle with the ankle in 15–20 degrees of plantar flexion. The posterior medial talar process can be palpated on the posteromedial side of the talus. It is important to determine the range of motion (ROM) of both the ankle joint and the subtalar joint and to compare both sides. At the conclusion of the examination, a posterior impingement test is performed (figure 2).

Recognisable posterior pain, confirms the diagnosis of posterior ankle impingement. Finally, the neurological and vascular status of the foot must be determined.

For posterior impingement, a lidocaine injection can be used for diagnostics, as it should result in a negative hyperplantar flexion test. The os trigonum is visible on lateral ankle radiographs, but it can be better visualized using a posterior impingement view— made with the ankle in 25 degrees of external rotation (PIM-view).[12,13]

Deep ankle pain is the main symptom of an OCD. Often routine ankle radiographs are negative.[14–16] To determine the extent and location of an OCD and to determine if an anterior or a posterior ankle arthroscopic approach is required, a CT scan can be used. Verhagen has shown that both CT scan and MRI have a similar accuracy in detecting an OCD.[17] For preoperative planning, a CT scan is preferable to determine the location and extent of the lesion and location of bony fragments.

MRI is the imaging method of choice for evaluating soft tissue injury and bone bruises, but may overestimate the size of an OCD due to bone oedema. Ultrasonography is a relatively inexpensive and reliable alternative to MRI for detecting focal soft tissue damage.[18] In case the diagnosis remains unclear in spite of all additional diagnostics the patient will likely not benefit from (diagnostic) arthroscopy.[19]

In case of suspicion of joint degeneration or OA, a standing radiograph may show joint space narrowing.[20]
Preoperative severity of complaints may be assessed using patient reported outcome measures (table 2). These may additionally be used to evaluate postoperative recovery.

Table 1 Key issues of patient selection

Indication	History	Physical examination	Additional diagnostics
OCD treatment	Deep ankle pain	The pain cannot be provoked by palpation with the ankle in a neutral position	► In case an X-ray does not show an OCD, a CT scan or MRI may be used. ► CT scan for preoperative planning and determination of the size of the lesion
Posterior ankle impingement	Activity associated posterior ankle pain	Hyper plantar flexion test	Posterior impingement view to identify an os trigonum
Deltoid ligament/Cedell fracture	► Hyperdorsiflexion or eversion trauma ► Posteromedial ankle pain aggravated by running or walking on uneven ground	Recognisable pain by palpation of the posteromedial (retromalleolar) region	CT scan for affirmation of avulsion/fracture/ calcification
Tarsal tunnel syndrome	Posteromedial ankle pain	Sensory and motor nerve examination	Electromyography
Loose body	Activity associated ankle pain or locking	No specific findings	CT scan for affirmation of loose bodies and their location
Arthrodesis	► Deep ankle pain ► Failed conservative treatment ► No other appropriate surgical option (shared decision-making)	ROM limitation Crepitation Deformation	Standing X-ray to confirm joint space narrowing

OCD, osteochondral defect; ROM, range of motion.

Non-operative treatment

Most ankle injuries are primarily treated non-operatively. If conservative treatment fails, surgery can be considered. Athletes require a quick return to play and may be eligible for acute surgical treatment.[18]

Surgical Techniques

The operative approach to hindfoot pathology can be performed by means of open or arthroscopic surgery. Hindfoot pathology concerning tendons may also require a hindfoot approach.
The best approach for this category of pathology is by tendoscopy. Tendoscopy is however, not the focus of this review. For hindfoot and posterior ankle arthroscopy, a two-portal hindfoot approach is used and is routinely performed as a day care procedure. Generally, no prophylactic antibiotics are given. A 4 mm 30 degrees angle arthroscope or an 11 cm length 2.7 mm scope with high-volume sheath (4.6 mm) is used.[5] The procedure is performed under general or neuraxial anaesthesia. A tourniquet is placed around the upper thigh, but arthroscopic surgery can also be performed without the use of a tourniquet.[21]
Kim et al[22] described a technique for the treatment of concurrent anterior and posterior ankle impingement, in which the patient was placed in a prone position, with the ankle hung in a shoulder-holding traction frame and the application of non-invasive ankle distraction.

Posterior ankle arthroscopy

For posterior ankle arthroscopy, the patient is placed in the prone position, with the ankle overhanging the end of the table, or with a triangular cushion under the distal tibia (figure 3).[23,24]
Using a two-portal approach, posterior ankle pathology can be visualized and subsequently treated.[19] For subtalar arthrodesis and for a fibular groove deepening procedure, an additional third portal is used.[14]
The posterolateral portal is initially created at the level of the tip of the lateral malleolus and the arthroscope is introduced, with the initial view direction being 30 degrees to the lateral side.[25] Care must be taken to avoid damage to the sural nerve. The posteromedial portal is then made at the same level (figure 4). A vertical stab incision is made and a mosquito clamp introduced. If scar tissue or adhesions are present, the mosquito clamp is exchanged for a 4.5 mm or 5.5 mm full radius shaver. Surgical debridement to improve the view is then commenced laterally—at the level of the subtalar joint, subsequently moving slowly towards the FHL. The FHL tendon is important to identify, as the neurovascular bundle lies just medial to it. For this reason, the area lateral to the FHL tendon is considered 'safe'. Passive motion of the great toe may help to identify the tendon. After removal of the thin ankle joint capsule, the joint itself can be entered, inspected and treated.[12,18,19,23,24]

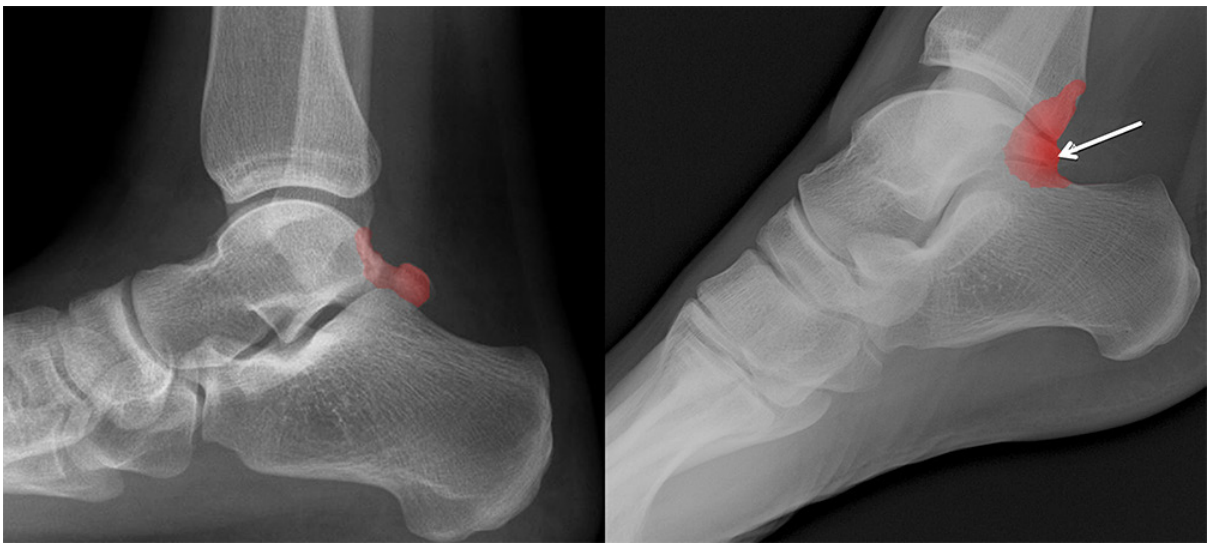

Figure 2. Pain experienced with posterior ankle impingement, intensified by maximal plantar flexion due to entrapment of soft and bony tissue.

Table 2 Validated outcome measures and classifications

Indication	Outcome measure	Classification
All indications	FAOS* SF-36* AOFAS VAS-FA FAAM	NA
Impingement ► Posterior impingement		Osteoarthritis scale, van Dijk *et al.*[10 62]*
Osteoarthritis	Ankle osteoarthritis scale[9 63]	Osteoarthritis scale, van Dijk *et al.*[10 62] *
OCD		CT classification, Ferkel[64]* MRI classification, Hepple[65]

*Approved by the ISAKOS scientific committee.
AOFAS, American Orthopaedic Foot and Ankle Society Score[67]; FAAM, Foot and Ankle Ability Measure[68]; FAOS, Foot and Ankle Outcome Score[66]; NA, not available; OCD, osteochondral defect; SF-36, Short Form (36) Health Survey[66]; VAS-FA, Visual Analogue Score Foot and Ankle.[69]

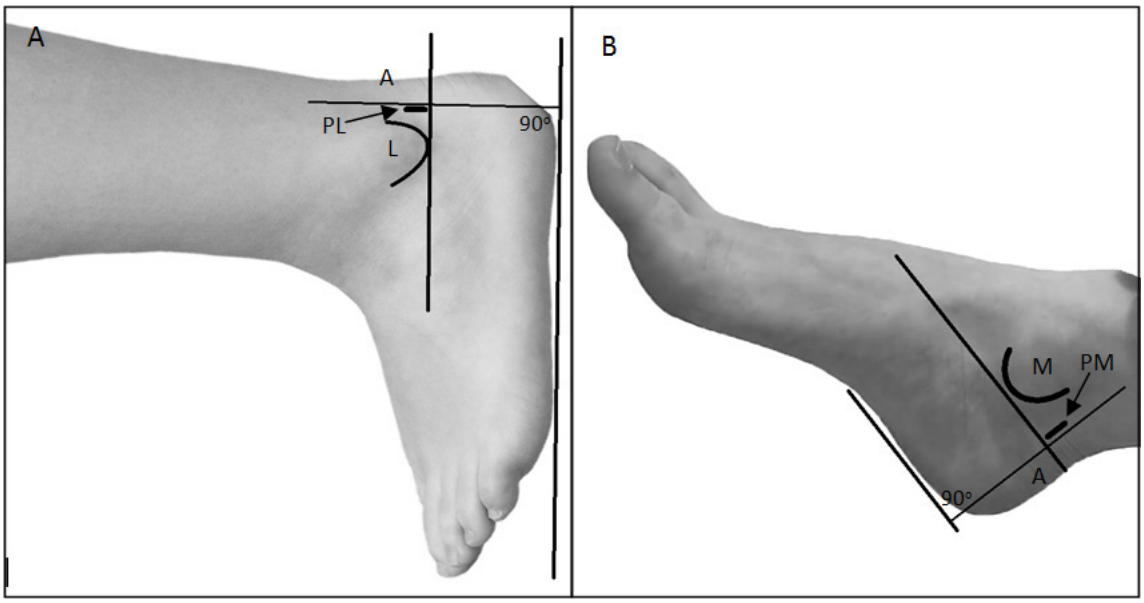

Figure 3. (A) The ankle is kept in a neutral position for making the anatomic landmarks that are needed for portal placement. To define the portal location, a straight line is drawn from the tip of the lateral malleolus to the Achilles tendon, parallel to the sole of the foot. (A=Achilles tendon, L=lateral malleolus and PL=posterolateral portal). The posterolateral portal is made just above the line from the tip of the lateral malleolus to the Achilles tendon. (B) The posteromedial portal (PM) is located at the same level as the posterolateral portal, just in front of the medial aspect of the Achilles tendon. (M=medial malleolus). Caption is copied from the figure 7 of de Leeuw et al.[14]

Surgical indications

Arthroscopic surgery offers advantages such as direct visualization of structures, improved assessment of articular cartilage, decreased postoperative morbidity, improved rehabilitation (both more rapid and also in terms of better functionality), earlier resumption of sports and improved day care treatment.[19] The main pathologies that can be treated with hindfoot arthroscopy are posteriorly located OCDs, loose bodies, ossicles, post-traumatic calcifications, avulsion fragments, posterior tibial rim osteophytes, chondromatosis and chronic synovitis (table 3).[26]

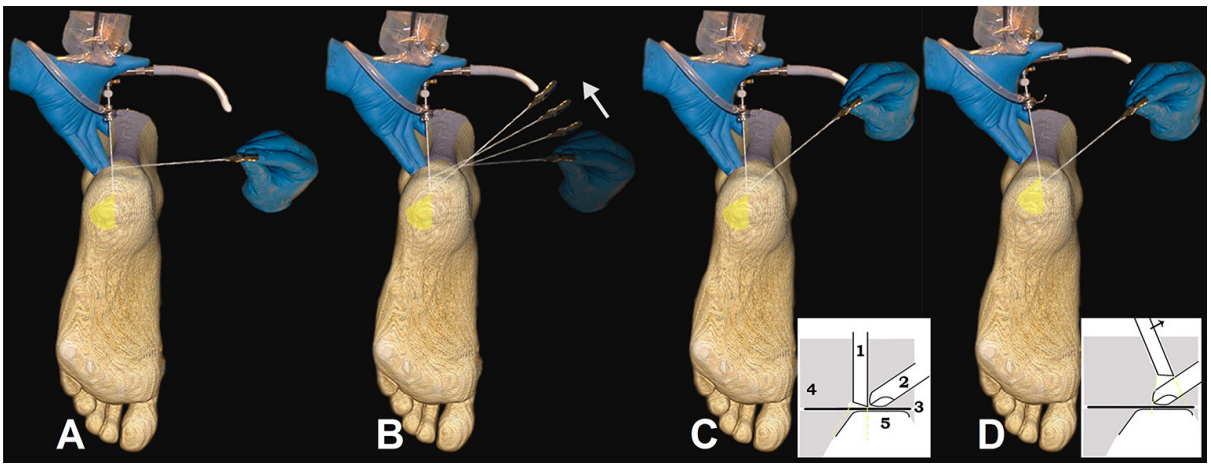

Figure 4. Technique to orient the arthroscope and instrument for posterior ankle arthroscopy with the aim of approaching the posterior aspect of the talocrural joint for excision of the os trigonum or posterior bony spurs. (A) The arthroscope is inserted in the posterolateral portal in the direction of the space between the first and second toe. The arthroscope touches the bone. The direction of view is 30° to the lateral side. The shaver is introduced through the medial portal and pushed anteriorly to the Achilles tendon to touch the shaft of the arthroscope. (B) The tip of the shaver is moved down along the shaft of the arthroscope to meet the arthroscope where it touches the bone. (C) 1—arthroscope; 2—shaver; 3—Rouviere ligament; 4—fatty tissue and 5—talus/ calcaneus. (D) The arthroscope is moved backward and tilted to the medial side to bring the tip of the shaver into view. From this position, the shaver can be used to carefully remove the Rouviere ligament and fatty tissue to create a working area with a clear view of the posterior aspect of the talus and talocrural and subtalar joints.

Pathology of the subtalar joint like osteophytes or loose bodies can also be treated by means of hindfoot arthroscopy. Extra-articular structures that can be treated with hindfoot arthroscopy are the hindfoot ankle tendons, the deep portion of the deltoid ligament and a symptomatic os trigonum .[12,25,27,28] Phisitkul et al[29] showed that, in patients with early-stage OA, arthroscopic debridement was useful in the treatment of impingement syndromes. Additional indications for which hindfoot arthroscopy may be used is the treatment of posteriorly located intraosseous talar cysts, talar body fractures and pigmented villonodular synovitis. Finally hindfoot endoscopy can be used to perform ankle arthrodesis or a subtalar arthrodesis.

Table 3 Essential and typical features of diagnostics and surgical procedures

Indication	Diagnostic findings	Surgical treatment
OCD treatment	Deep ankle pain Bone marrow oedema on MRI Identify cysts and loose fragments	Arthroscopy first choice (anterior, posterior or both, depending the location of the lesion)
Posterior ankle impingement	Os trigonum or prominent posterior talar process	Posterior arthroscopy
Deltoid ligament/Cedell fracture	Calcifications and avulsion fragments on CT scan	Removal avulsion fragments and calcifications Ligament reconstruction in case of persistent medial instability
Tarsal tunnel syndrome	Space occupying lesions Deformities	Local corticosteroid injections Orthoses for foot deformities Physiotherapy Release of tibial nerve (endoscopic)
Loose body removal	Chondral or post-traumatic origin Avulsion fragments Concomitant OCD	Posterior arthroscopy
Arthrodesis ► Tibiotalar fusion ► Subtalar fusion ► Double fusion	► Tibiotalar osteoarthritis ► Sequelae of a fracture ► Subtalar osteoarthritis ► Symptomatic combined arthrosis	► Standard portals with accessory anteromedial portal in case of 'bowler-hat' shaped talus ► Standard posterolateral and medial portal ► Possible use of two accessory para-Achilles tendon portals ► Minimal resection joint surface to avoid destabilisation

OCD, osteochondral defect.

Osteochondral defect

Talar OCD's are mainly post-traumatic. Due to the post-traumatic origin concomitant ankle instability is not uncommon.[30–32]
Post-traumatic OCDs may remain asymptomatic or may even heal. A significant number however, progresses to deep ankle pain exacerbated during weight bearing. Symptoms include prolonged swelling, joint stiffness, recurrent synovitis, catching and locking. Non-surgical therapy has a success rate of 45%.[12,18] The treatment of choice for OCDs, in lesions less than 15 mm in diameter, is arthroscopic debridement associated with subchondral bone penetration (curettage, drilling or microfracture).[12,18,19,33]

No firm recommendations on the treatment of lesions greater than 15 mm can be made at present, due to the lack of evidence currently in the literature.[34]
If an OCD is diagnosed, ankle CT scan may help determine the exact size and location of the lesion (table 4). A CT scan in plantar flexion may also help to determine if an anterior or a posterior approach is indicated.[17] Generally, treatment of an OCD is based on one of the following three principles:

1. debridement and bone marrow stimulation, potentially in combination with loose body removal (microfracture, abrasion arthroplasty or drilling);
2. securing a lesion to the talar dome (retrograde drilling, bone grafting or fragment fixation);
3. stimulating the development of hyaline cartilage (osteochondral autografts mosaicplasty, allografts or autologous chondrocyte implantation).[35]

As described by van Dijk et al,[12] in an update on arthroscopic techniques, he cites 'In these procedures, a 4.0 mm scope and a 4.5- or 5.5 mm shaver are routinely used. If synovitis is present, a local synovectomy is performed with the ankle in dorsiflexion. The lesion is identified in forced plantar flexion by palpating the cartilage with a probe. A soft tissue distractor can be applied if needed. The full-radius resector is then introduced into the defect. In some cases, identifying the defect by introducing a spinal needle, probe, or curette can be useful before introducing the resector.

To increase the chance of success, it is important to identify the full defect (including the anterior part) and to remove unstable cartilage and necrotic subchondral bone. Hyaluronic injections may improve surgical results after performing microfracture and debridement.[36] After the procedure a compression dressing is applied.[12] Good to excellent results have been reported for arthroscopic treatment of posteriorly located OCDs in 80%–87% of patients.[17,23]

Table 4 Essential and typical features of imaging techniques used in the diagnostic process of an OCD[17 70 71]

Imaging tool	CT scan	MRI
Lesion size	High accuracy of both size and location	Overestimation if size due to bone oedema
Concomitant damage	Additional bony fragments and osteophytes	Additional soft-tissue lesions
Preoperative planning	A CT scan in forced plantar flexion (sagittal reconstruction) helps to determine whether anterior or posterior approach is required	
Negatives	No cartilage visualisation	No loose body detection No correct estimation of the size of the bony lesion
Radiation exposure	Ionising radiation	No ionising radiation
Diagnostic accuracy	Sensitivity 81% Specificity 99% Positive predicting value 96% Negative predicting value 94%	Sensitivity 96% Specificity 96% Positive predicting value 89% Negative predicting value 99%

OCD, osteochondral defect.

Van Dijk CN, *et al*. *JISAKOS* 2017;**2**:269–277. doi:10.1136/jisakos-2016-000082

Posterior ankle impingement

Posterior ankle arthroscopy has highlighted the need for specific anatomical knowledge, modified the classic arthroscopic tools and skills and has introduced a broad spectrum of new indications in posterior ankle pathology.[12]
Posterior ankle impingement is not always caused by bony pathology. It frequently presents as a soft tissue impediment, with or without a bony component.[12]
Posterior ankle impingement syndrome consists of a group of pathologies characterized by posterior ankle pain in plantar flexion, frequently occurring in ballet dancers, downhill runners and football players.[12,18,28] The mechanism of

injury can be overuse or trauma. Differentiation between these two groups is important, as the prognosis for posterior impingement from overuse is better, and these patients are more satisfied after arthroscopic treatment.[19,37] The hyperplantar flexion test is considered positive for posterior impingement if it causes recognizable posterior ankle pain. Additionally, the physician can perform a diagnostic injection with lidocaine. Surgical resection is indicated in case of failure of conservative treatment.[18,28]

The main procedures are resection of an os trigonum, reduction of a prominent posterior talar process, and removal of a soft tissue impediment.[12,38–40] In addition to the standard instruments for treatment of osteophytes and ossicles, a 4 mm chisel and small periosteal elevator can be used.[12]

In order to be able to remove the posterior process or os trigonum, the surgeon will need to release the posterior talocalcaneal ligament and flexor retinaculum and partially detach the posterior talofibular ligament. This creates enough working area to lift the os trigonum from the subtalar joint using a small-sized bone elevator. Subsequently, a grasper can be used to remove the fragment. Postoperatively, a sterile compression dressing is applied around the ankle.[12]

In the series of Spennachio et al,[26] arthroscopic treatment of posterior impingement provided excellent results and clinical improvement in all cases. In the series of Lopez et al,[41] the Visual Analogue Scale for pain showed a decrease in reported pain from 7.5 preoperatively to 0.8, 1 month postoperatively.

Overall, posterior ankle arthroscopy is considered safe and effective in the treatment of posterior ankle impingement in the elite football player, with an expected return to training of 5 weeks.[42]

Deep portion of the deltoid ligament and Cedell fracture

Two conditions often seen together are rupture of the deep portion of the deltoid ligament (posterior talotibial ligament (PTTL)) and a Cedell fracture. Both originate from a hyperdorsiflexion- or eversion trauma leading to an avulsion of the PTTL at its insertion (medial talar tubercle). Calcifications are not an uncommon result after this trauma. The most distinctive symptoms include posteromedial ankle pain, worsening by activities such as running and walking on uneven ground. Avulsion fragments, post-traumatic calcifications or ossicles in the deep portion of the deltoid ligament can be treated by removal of these bony structures.[19] The avulsion fragment lies medial and anterior to the FHL;

thus, care has to be taken to protect the neurovascular bundle. It is important to stay close to the bone and use blunt dissection with a small sized periosteal elevator. If persistent chronic medial instability is present, ligament reconstruction can be carried out using a free graft of the plantaris tendon, with tendon allograft being an alternative. Given the continuing evolution of the anatomical understanding of this ligament, current treatment protocols for deltoid injuries require further standardization, with an emphasis on proper diagnosis.[43]

Tarsal tunnel syndrome

Tarsal tunnel syndrome is an entrapment neuropathy of the tibial nerve. The cause is often ideopathic but factors such as trauma, space-occupying lesions and deformities of the foot have been shown to be related to its development.[44] Clinically, patients may report symptoms that are difficult to localize, and physical examination findings vary greatly leading to underdiagnosis and misdiagnosis.
A thorough history taking should ascertain any causative factors. Additionally, electromyography may support the diagnosis; however, a normal electromyogram does not exclude a tarsal tunnel syndrome. It has been shown that sensory nerve examination has a higher sensitivity compared with motor nerve examination.[45,46]
Conservative treatment consists of local corticosteroid injections, orthoses for foot deformities and physiotherapy. If conservative treatment fails, endoscopic decompression is a good option. After identification of the FHL tendon, the fascia covering the tibial nerve is opened with a haemostat. The fascia is opened over its full length thereby exposing the tibial nerve. Adhesions are identified and the nerve is freed along its entire length.

Loose bodies

Loose bodies may be chondral or osteochondral in origin and can be post-traumatic or result from an OCD. Multiple loose bodies may develop in case of chondromatosis or synovial osteochondromatosis. Loose bodies can cause pain, swelling, decreased ROM and locking. They do not necessarily float freely within the joint capsule but may also be fixed to synovium, thereby being semi-loose bodies or ossicles. On a standard ankle radiograph, loose bodies can easily be missed. A CT scan is the additional investigation of choice. Localization is important in order to facilitate and determine the best surgical approach for extraction.

Arthrodesis

Tibiotalar arthrodesis

For arthroscopic tibiotalar arthrodesis, the standard two-portal technique used in hindfoot arthroscopy is used. For debridement, routine instruments are used: a 5.5 mm Bone-cutter shaver blade, a curved curette and a 5.0 mm osteotome. An accessory anteromedial portal may provide outcome in case of a 'bowler-hat' shaped talus to ensure complete debridement of the anterior part of the talus and distal tibia.
After full cartilage removal, including the joint gutters, the subchondral bone is removed until a bleeding bone surface is attained. The contour of the talus (and distal tibia) has to be kept intact. Two 6.5 mm cancellous compression screws are inserted through a midline incision and through the Achilles tendon. The excellent intra-articular operating area provides the ability to optimize hindfoot alignment and the easy orientation of the two screws intraoperatively. For this reason, the posterior approach is preferred over the anterior procedure. Fluoroscopy may be used as guidance for screw insertion.
After surgery, patients are kept in a non-weight bearing cast for 6 weeks. Depending on clinical and radiographic assessment, a walker—or weight-bearing cast—is applied for another 4–6 weeks. After radiographic fusion, the patient is allowed to wear normal shoes and resume activities, as tolerated.

In summary, posterior arthroscopic fusion has proven to be an effective and safe option at mid-term follow-up in the treatment for progressed post-traumatic ankle osteoarthritis.[47]

Subtalar arthrodesis

The main indications for subtalar arthroscopy are sequelae of a fracture, a sprain or subtalar osteoarthritis. The first arthroscopic subtalar arthrodesis was described by Parisien et al[48] which was a lateral and posterolateral approach. Later popularised by van Dijk et al[5] an exclusively posterior approach was described. Preoperatively, an anteroposterior (AP) and lateral weight-bearing ankle X-ray should be performed and a CT scan can also be useful in preoperative planning.[49] To perform a subtalar arthrodesis using posterior ankle arthroscopy, the patient is positioned prone with a tourniquet around the thigh. The foot is left free, hanging over the end of the table with the ability to place

the ankle in a 90 degrees angle. Routine posterolateral and posteromedial portals are created as previously described. After identification of the subtalar joint, all cartilage is removed using curved curettes and a shaver system.[50] A third sinus tarsi portal facilitates debridement of the anterior compartment of the subtalar joint. Fixation is performed, with two large-diameter (>6 mm) cannulated screws which are introduced through a separate incision at the level of the posterior calcaneus.[5]

Postoperatively, the ankle is immobilized using a removable non-weight bearing cast for 4–6 weeks, followed by a weight bearing cast for another 4–6 weeks. Arthroscopy has improved the results when compared with open arthrodesis-significantly reducing non-union rates and with fewer neurovascular complications.[49]

Double fusion

Through a posterior arthroscopic approach, double fusion (combined tibiotalar and subtalar arthrodesis) can be performed. Double fusion may be indicated in case of symptomatic combined arthrosis of the ankle joint and subtalar joint. Bernage et al[51] described the double fusion using two additional para-Achilles tendon portals. Minimal resection of the joint surfaces of both the subtalar and tibiotalar joint are described to avoid destabilization of the joint. A tibial hindfoot nail is used for fixation, which is introduced through an incision on the sole of the foot. First, a transplantar K-wire is introduced which allows subsequent introduction of reamers and the nail. Final osteochondral resection is performed with the K-wire still in place. Postoperative treatment is similar to that of the subtalar and ankle arthrodesis.

Contraindications to surgery

Absolute contraindications for any form of ankle arthroscopy include local (soft-tissue) infection, severe degenerative joint disease and poor vascularity of the leg. Moderate degenerative changes with diminished ROM, severe oedema, joint space narrowing and diabetic vascular disease account for relative contraindications.[14,18,24]

Complications

Overall hindfoot arthroscopy is regarded to be a safe procedure. Nevertheless, adequate preoperative planning and the use of a careful, precise technique is

important. The most common complications include neurological problems, sinus tract formation, vascular damage, synovitis and wound infection.[23] The proximity of the medial neurovascular bundle to the posteromedial portal is the major concern for posterior ankle arthroscopy. Not only the use of the posteromedial portal is a risk factor for postoperative complications, but also the posterolateral portal which is close to the sural nerve. Inadequate portal positioning may cause damage to this nerve (box 2 and 3).[18] Knowledge and understanding of the ankle joint anatomy help reducing these complications.[19]

Donnenwerth et al[52] reported complications in 3.8% of cases after hindfoot arthroscopy and Blazquez Martin et al[53] reported complications in 12.06% of cases. Zengerink reported 2.3% of complications for hindfoot arthroscopy alone.[23]

Box 2 Tips and tricks for successful posterior arthroscopy

- For correct orientation and reproducibility, always start with the arthroscope in the posterolateral portal.
- Instruments introduced through the posteromedial portal are inserted perpendicular to the arthroscopic shaft. The shaft is subsequently used as a guide to direct instruments anteriorly. The direction of the arthroscopic view (30° angulation) is routinely towards the lateral side for optimal and consistent orientation throughout the procedure.
- It may be helpful to enlarge the entry point through the crural fascia using a punch or scissors.
- Identify the flexor hallucis longus (FHL) tendon. The FHL serves as a landmark. Avoid instrumentation medial to the FHL.
- In OCD treatment, it is advised to regularly switch portals.

Box 3 Pitfalls in posterior ankle arthroscopy

- Identify the flexor hallucis longus tendon and work lateral to this tendon, as the neurovascular bundle is situated just medial to it.
- When microfracturing, an osteochondral defect, loose bony particles can be created with the microfracture which may act as loose bodies if not properly removed.
- After peroneal groove deepening for recurrent peroneal tendon dislocation, the ankle is manipulated to check whether sufficient bone has been removed. Removing too much bone may result in a fracture of the lateral rim. To avoid damage to the tendon, the lateral edge of the groove should be smoothed.

Geographical differences

There have been reports of additional portals used in the technique of posterior arthroscopy. Two additional posterolateral portals have been described for the removal of os trigonum. Ferkel described removal of os trigonum by standard subtalar portals.[54] These approaches have not met with great acceptance.
The posterior approach cannot be regarded as state of art for ankle arthrodesis. The majority of current literature reports on performing an ankle arthrodesis through two anterior portals. For endoscopic double fusion, the posterior approach can be considered as state of art.

For subtalar fusion in Europe, the two-portal hindfoot approach is mainly used. In the USA, some perform the two-portal hindfoot approach, but others report using the classic subtalar portals with the patient in lateral decubitus position.[55] In South America, arthroscopic surgeons use the two classic posterior portals as previously described. Most ankle surgeons in this region do not use dedicated instruments specifically designed for the ankle joint. The same problem occur with drills. In Argentina, for example, 95% of drills used in surgeries, are not specifically designed for arthroscopy.

A common issue in the Middle East is the high incidence of athletes with vitamin D deficiency.[56] In combination with triggering sports, this potentially can lead to early initiation of bony posterior impingement syndromes.

Future directions

Currently, the two-portal hindfoot arthroscopy technique is safe and accepted for most indications. In the future, more indications will undoubtedly arise, given the ongoing studies in combination with the use of hindfoot arthroscopy and new higher quality evidence emerging. This will strengthen current recommendations and further help orthopaedic specialists in evidence-based practice.[26] Expansion of endoscopic soft tissue techniques in the hindfoot will likely be a major avenue of study.

Currently, arthroscopy is being performed for assessment and treatment of disorders of the peroneal tendon, posterior tibial tendon and Achilles tendon. For the Achilles tendon in particular, these techniques have been shown to be beneficial and future expansion in this direction can be anticipated.

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Chronic lateral ankle increases the likelihood for surgery in athletes with an os trigonum syndrome.

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D'Hooghe P, Alkhelaifi K, Almusa E, Tabben M, Wilson M, Kaux JF

Chronic lateral ankle instability increases the likelihood for surgery in athletes with os trigonum syndrome.

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ABSTRACT

Purpose

The etiology and incidence of os trigonum syndrome in professional athletes is highly variable. There is a paucity of data to ascertain why some athletes evolve towards surgery whilst others remain asymptomatic.

Aim

We hypothesized that a lateral ligament ankle injury would increase the likelihood for surgery in those athletes with os trigonum syndrome.

Methods

Eighty professional athletes with clinical and radiological signs of os trigonum syndrome were identified to ascertain the incidence of injury to the lateral ligamentous ankle complex (acute and chronic) by magnetic resonance imaging (MRI). This cohort was subdivided into 2 groups; a surgical (n = 40) and a non-surgical (n = 40) cohort. Surgical division was decided if (1) the clinical hyper-plantar flexion test was positive, (2) a positive diagnostic ultrasound-guided injection and (3) no improvement was observed after 6 weeks of conservative rehabilitation.

Results

From the surgical cohort, 37 players (94.1%) had a chronic lateral ankle ligament injury on MRI, whilst 3 players (5.9%) had an acute lateral ankle ligament injury. Binary logistic linear modelling revealed that having a chronic lateral ligament injury increases the likelihood of os trigonum syndrome surgery by ten times compared to those with an acute lateral ligament injury.

Conclusion

Professional athletes with chronic lateral ligament ankle injury have an approximate ten times greater risk for os trigonum syndrome surgery compared to athletes with acute lateral ligament ankle injury.

INTRODUCTION

Os trigonum ankle syndrome refers to a posterior ankle impingement pathology, often characterized by posterior ankle pain in plantar flexion. It is frequently observed in athletes where the mechanism of injury is either overuse

or direct trauma[15,18,21]. The clinical prognosis appears to be better in those presenting with overuse injuries rather than trauma[12,19]. The incidence of os trigonum syndrome in the athletic population is variable, ranging between 1.7 and 50%. Available data also suggests that between 33 and 50% of athletes present bilaterally. There does not appear to be an increased prevalence between men and women, nor between different age groups[15]. Athletes who participate in dynamic agility sports such as football, require a high degree of plantarflexion strain, and are thus more likely to symptomatic if having os trigonum syndrome[12,15,18,19,21].

The etiology of os trigonum ankle syndrome in professional athletes is highly variable. Although blunt trauma is considered a primary etiological factor, there is a paucity of data to why certain athletes evolve towards surgery whilst others remain asymptomatic for this condition. Most commonly, symptomatic os trigonum may be attributed to repetitive microtrauma due to impingement of the ossicle between the calcaneus and the postero-inferior aspect of the tibia[12,15,18,19,21]. Understanding why some athletes evolve towards surgery, and others not, have significant implications for those athletes with os trigonum syndrome regarding strategies for prevention, diagnosis, therapy and rehabilitation.

Diagnosing os trigonum syndrome is accomplished via a number of procedures. First, a clinical hyperplantar flexion test is considered positive for posterior impingement if it causes posterior apprehensive ankle pain. Secondly, a diagnostic ultrasound-guided injection may prove helpful in identifying the exact location of the pain[21]. Finally, radiological imaging often reveals a bony cause typical of os trigonum, that, if restricting athletic participation through impingement pain, is a clear indication for arthroscopic surgical resection[1,4,8,15,18,21]. Arthroscopic treatment of posterior impingement provides excellent results and clinical improvement in the athlete's ankle[9] with an expected post-operative return to training from 5 weeks onwards[3,22]. Accordingly, the aim of the study was to ascertain if chronic lateral ankle instability is a contributing factor that leads an athlete with os trigonum syndrome towards surgery.

Our hypothesis was that lateral ligament injury to an athlete's ankle can increase the likelihood for surgery in those with os trigonum syndrome.

MATERIALS & METHODS

The pre-operative magnetic resonance images (MRI) of 80 professional athletes who were referred for surgical consultation at the Aspetar Hospital Orthopaedic Surgery Department during the past 5 years (2013–2017), were evaluated for the presence of chronic or acute lateral ligament complex injury. Inclusion criteria were the confirmation of an os trigonum on MRI imaging, together with having a positive hyper-plantar flexion clinical test and being a registered professional athlete with the Qatar Olympic Committee.
Exclusion criterium was having previous ankle surgery or a malleolar fracture (lateral or medial).
A radiologist blinded to the research question at Aspetar Hospital was asked to review all ankle MRI images. The radiologist was asked to answer the following five radiological questions for each athlete[22]:

1. Confirmation of os trigonum in the ankle: yes/no.
2. Anterior talo-fibular ligament status: Grade 1–3.
3. Calcaneo-fibular ligament status: Grade 1–3.
4. Posterior talo-fibular ligament status: Grade 1–3.
5. Acute (< 6 weeks after trauma) or chronic ligament injury (≥ 3 months after trauma) along the current radiological MRI guidelines[22].

This cohort was subdivided into two groups; a surgical (n = 40) and a non-surgical (n = 40) cohort. The division was decided if clinical hyper-planter flexion test was positive, if the ultrasound-guided injection was positive, and if no improvement was observed after 6 weeks of conservative rehabilitation.
All athletes were clinically evaluated by the same orthopaedic ankle expert and all were diagnosed with a positive clinical posterior ankle impingement test and a positive os trigonum finding on MRI. This study received IRB approval (#E2017000260) from the Qatar Anti-Doping Laboratory IRB.

STATISTICAL ANALYSIS

Data were analyzed using SPSS software (IBM-SPSS statistics, v23, Chicago, Illinois). Data were divided into two groups: surgical vs. non-surgical. Data are presented as count (percentage) of chronic vs. acute lateral ankle ligament injury in each group. No calculation of sample size was performed as this is a case–control study in which we included all possible cases from 2013 to 2017.
A binary logistic linear model was used to analyze the association between the lateral ankle ligament injury (acute vs. chronic) and surgery outcome.

Regression coefficients are presented as odds ratios with 95% confidence intervals (CI). P values < 0.05 were considered as statistically significant.

RESULTS

All forty professional athletes that required os trigonum surgery had some involvement of lateral ligament injury to the ankle on MRI; 37 players (94.1%) had a chronic lateral ligament injury [Table 1].

Table 1 Surgery outcome vs. lateral ligament of the ankle (acute/chronic) cross-tabulation

	Ligaments state		Total
	Count of chronic case	Count of acute case	
Surgery			
No	28 (60.9%)	18 (39.1%)	46
Yes	32 (94.1%)	2 (5.9%)	34

Three (5.9%) athletes that underwent os trigonum surgery, had an acute lateral ankle ligament injury on MRI. Binary logistic linear model revealed that professional athletes with os trigonum syndrome were 10-times more likely to require surgery if presenting with a chronic lateral ligament injury of ankle for compared to those athletes with an acute lateral ligament injury (Table 2).

Table 2 Lateral ligament of the ankle (acute / chronic) risk factor for surgery in athletes with os trigonum syndrome

	Odds ratio	95% CIs	*P* value
Lateral ligament of the ankle			
Acute	1		
Chronic	10.3	2.2–48.3	0.003

Regression coefficients are presented as odds ratio with 95% CI

DISCUSSION

The most important finding of this study was that athletes presenting with chronic lateral ankle instability and os trigonum syndrome were ten times more likely to undergo surgery than athletes with an acute ankle injury and os trigonum syndrome. Although overuse and repetitive trauma (contact, plantar flexion and supination) are considered primary etiological factors for os trigonum syndrome, there is a paucity of data as to why certain athletes evolve towards surgery whilst others remain asymptomatic with this condition[5,6,16,17].

The os trigonum syndrome mechanism of injury has been described as a "nut in a nutcracker" because the posterior talus and surrounding soft tissues are compressed between the tibia and the calcaneus during plantar flexion of the foot[2,17]. Due to the repetitive plantar flexion movements in dynamic sports such as football, the chronic stress imposed on the posterior ankle increases the risk of developing osseous and soft-tissue injuries[2,6,17].

The two bony structures that are involved in the os trigonum syndrome impingement mechanism are the posterior tibial malleolus (which may have a prominent downward slope) and the superior surface of the calcaneal tuberosity (which may have a prominence)[13,14]. The soft-tissue components of this anatomic interval, include the synovial sheath of the Flexor Hallucis Longus (FHL) and the posterior synovial recess of the tibiotalar and subtalar joints, all of which may be involved in this impingement syndrome. In fact, the reported prevalence of FHL tenosynovitis is greater in athletes with posterior ankle pain[6,13,16].

The differential diagnosis of os trigonum syndrome is considerable, with lateral ankle instability considered one of many conditions accounting for hindfoot ankle pain[2].
There are, however, reports suggesting that lateral ankle instability might be linked to mechanisms leading towards posterior ankle impingement[17]. In those athletes with a deficient lateral ligament complex as a result of an ankle inversion sprain for example, the talus can rotate more anteriorly under the tibial plafond.
This can lead to an increased osseous impingement between the posterior tibia and the talus, resulting in os trigonum syndrome[13,14]. A number of studies have demonstrated the link between lateral ankle instability and anterior impingement, but the link with posterior impingement has not been well documented[3,10,17,21,22] Consequently, the difference between acute vs. chronic lateral instability and posterior impingement has never been fully proven. Biomechanical testing has demonstrated that a severe ankle sprain creates a pathological anterior translation of the ankle (Fig. 1), but not a posterior translation[7].
The implication of this means that the hyper plantar-flexion position of the ankle could create a greater mechanical posterior ankle conflict in the presence of a combined lateral ankle instability.

These two important findings could be the reason why chronic lateral ligament instability is a key variable in the development of os trigonum syndrome. It might also explain why bony anterior impingement is frequently observed in combination with a restricted ankle range of motion where this is rarely the case in bony posterior impingement. An MRI study in athletes with os trigonum syndrome (sagittal T1-weighted and fat-suppressed T2-weighted images) observed abnormal signal intensity in the lateral talar tubercle and/or os trigonum, consistent with bone marrow edema[11].

Considering the mechanisms of injury, this abnormal signal intensity could be the result of bone impaction which represents micro-trabecular fractures, edema, and/or hemorrhage of the bone marrow without disruption of the cortex. Most ankle sprains occur in plantar flexion and inversion[10,20]. When considering the repetitive dynamic movements involved in agility sports, this ankle position in those athletes with a deficient lateral ligament might explain why the os trigonum undergoes increased mechanical overload, becoming painful compared to incidental findings of os trigonum. This is supported with MRI findings of bone marrow edema over the os trigonum after hyper plantar flexion injury of the ankle (Fig. 2a, b).

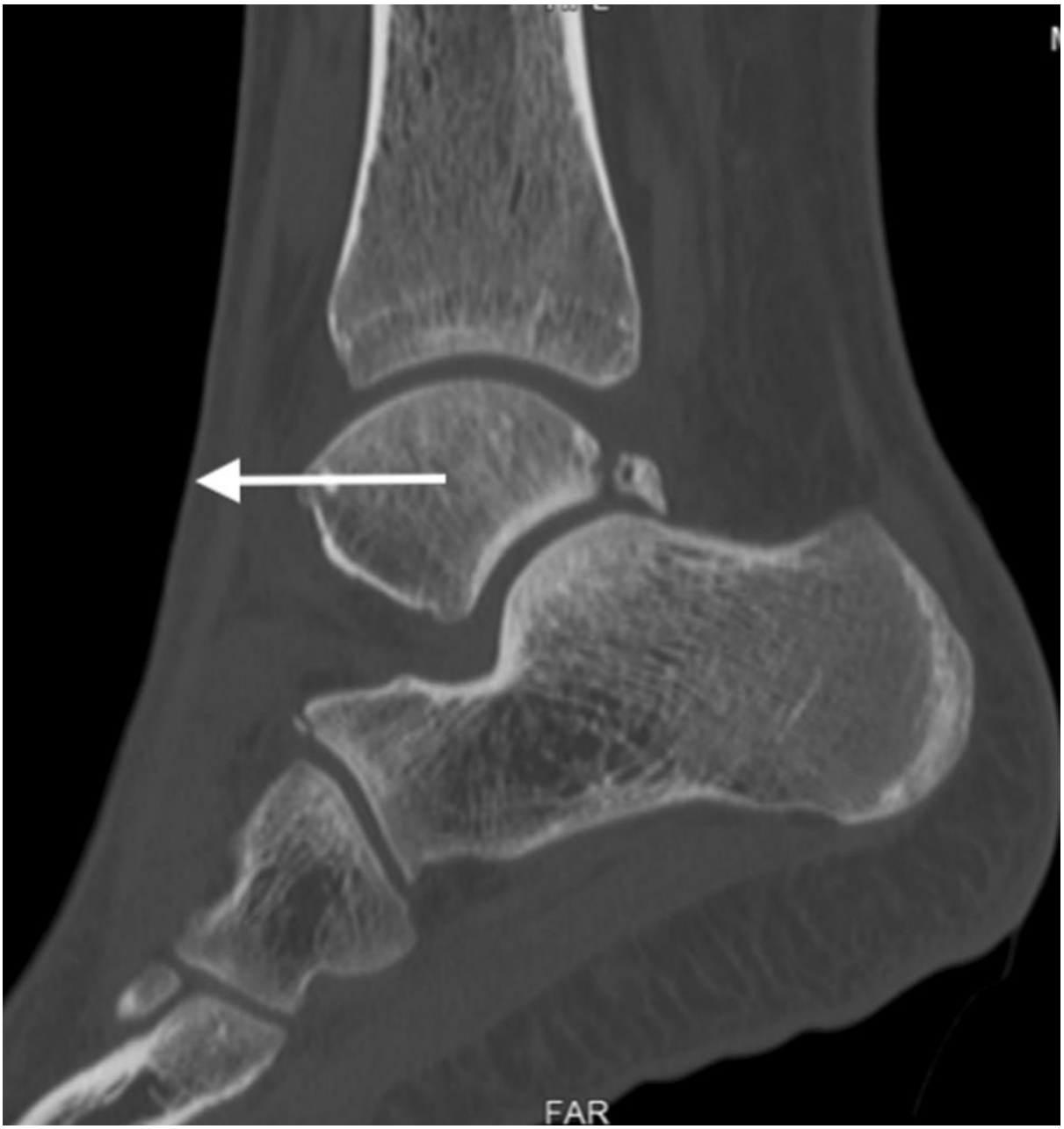


Fig. 1 Anterior translation of the ankle (white arrow) in an athlete with os trigonum syndrome and combined chronic lateral ligament instability of the ankle

In the present study, chronicity of ankle instability correlates with a significantly greater likelihood of os trigonum surgery compared to athletes with acute instability. We suspect that the initial swelling and overall dysfunction after acute ankle sprain limits the hyper plantar-flexion position of the ankle, and therefore, restricts the chance of posterior conflict. In addition to that, the

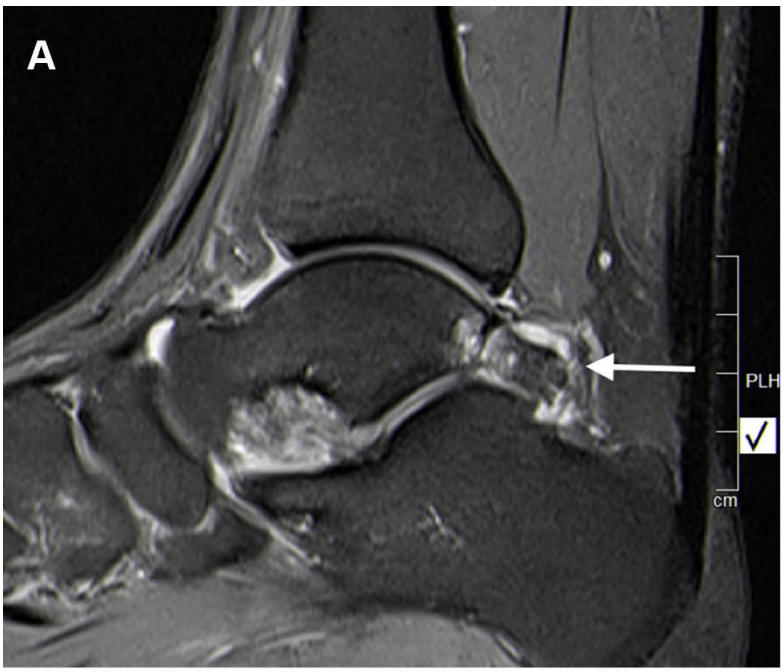
ankle may also still count on its neuromuscular compensatory mechanisms in an acute ankle sprain.

This study is limited by its respective cross-sectional design. Further, no follow-up MRI scans were performed to ascertain the post-operative evolution of MRI signal intensity over time. Finally, no sample size calculation was undertaken and no test–retest reliability (clinical examination and MRI interpretation) was measured.

The clinical relevance of this study is that athletes with os trigonum syndrome should be investigated for combined chronic lateral ligament instability.

By preventing ankle injuries in athletes with os trigonum evolving towards chronic lateral ankle instability, the likelihood of surgery might be significantly reduced.

To our knowledge, no previous studies have examined the association between the injury to the lateral ligament complex of the ankle (acute and chronic) and the clinical os trigonum syndrome. Consequently, this study offers new insights into the etiology and pathophysiology of posterior impingement in the athlete's ankle. It also provides new evidence-based diagnostic indications for os trigonum syndrome surgery.
More studies are necessary to evaluate the exact role of ankle instability in os trigonum syndrome, especially in the professional athletes and whether preventing an acute ankle injury from progressing into a chronic syndrome reduces the likelihood of surgery in those athletes with os trigonum syndrome.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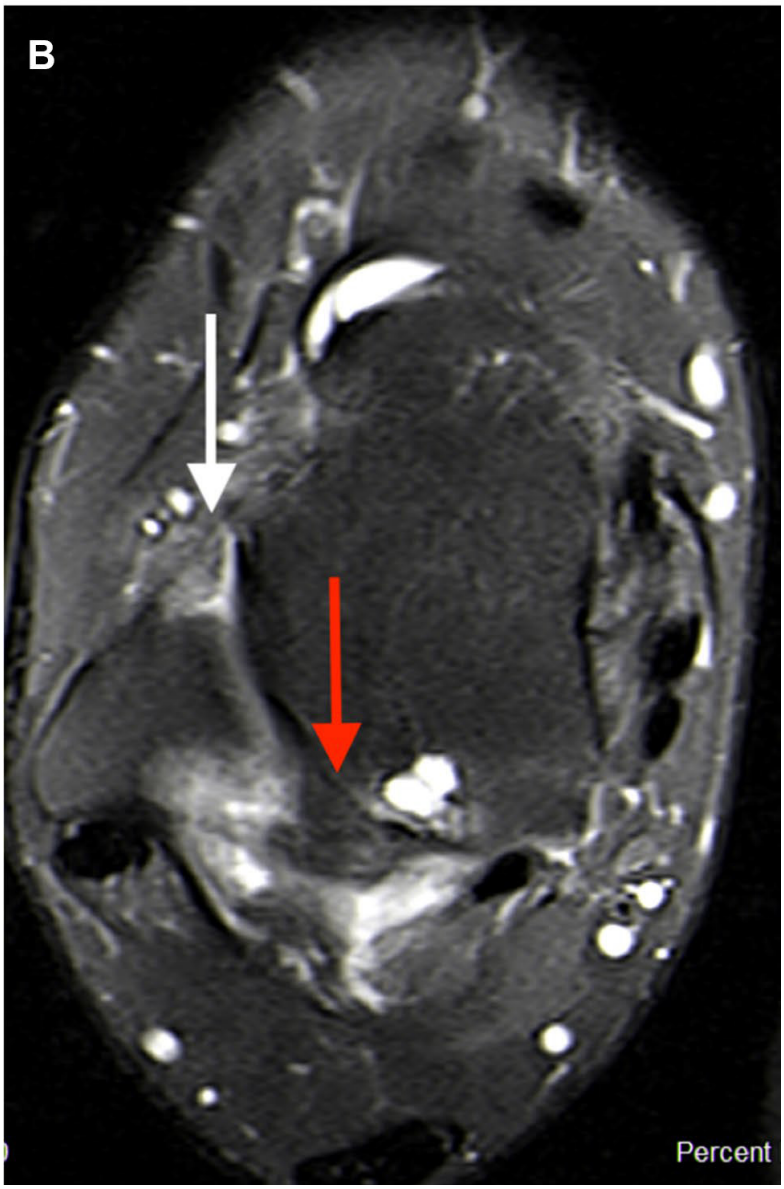
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Fig. 2 a Sagittal T2 MRI image depicting inflammatory signs and bony oedema over the os trigonum complex (white arrow). b Axial T2 MRI image depicting inflammatory signs and bony oedema over the os trigonum (red arrow) in an athlete with a combined chronic injury to the lateral ligament complex (white arrow)

CONCLUSION

Professional athletes who have os trigonum syndrome and a chronic lateral ligament ankle injury have an approximate 10 times greater risk for surgery compared to athletes with os trigonum syndrome and an acute lateral ligament ankle injury.

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CHAPTER

8

RETURN TO PLAY (ANKLE SPRAIN & HIGH ANKLE SPRAIN)

CHAPTER 8:
Return To Play (Ankle Sprain & High Ankle Sprain)

Return to play after isolated unstable syndesmotic ankle injuries in professional football players.
British Journal of Sports Medicine (in review 2019)

D'Hooghe P, Grassi A, Alkhelaifi K, Calder J, Kaux JF, Baltes T, Zaffagnini S, Ekstrand J

ABSTRACT

Objectives
Current epidemiological data indicates a significant increase in the incidence of isolated syndesmotic ankle injuries in male professional football players. We evaluated the typical time to return to play following surgical stabilization for isolated unstable ankle joint distal syndesmosis injuries amongst a cohort of professional male football players.

Methods
Between January 2012 and December 2017, 110 male professional football players were treated surgically for an isolated unstable ankle joint distal syndesmosis injury. We followed these players longitudinally until they returned to play.

Results
The mean age at surgery of the footballers was 24.9 ± 4.0 years; according to arthroscopic evaluation, 75 (68%) were graded as a Westpoint grade IIB injury and 35 (32%) were graded as a Westpoint grade III injury, while concomitant cartilage injury was observed in 23 patients (21%). The mean post-operative time required to begin the on field rehabilitation (completing exercises on grass) was 37 ± 12 days, while the mean time to return to team training was 72 ± 28 days. The first official match was played on average 103 +/- 28 days post-operatively. After 1 month post-operatively, 55% of players had started the on field rehabilitation, while only 4% had returned to team training and none had participated in an official match. At 2 months post-operatively, almost all of the players (97%) had started on field rehabilitation, 47% were participating in team training and 4% had already participated in an official match. At 3 months post-operatively, 73% were allowed to partake in team training and nearly half (44%) had played an official match. The proportion of players who had returned to team training and match play were 95% and 76% at 5 months post-operatively and 98% and 95% at 6 months post-operatively.

Multivariate analysis revealed that the severity of injury, the concomitant presence of cartilage injury and the age of the player were significant predictors ($p<0.00001$) of time to return to on field rehabilitation, team training and match play.
An injury graded as a Westpoint grade III lengthened the time required to start on field rehabilitation by 16 days, while the presence of a concomitant cartilage injury resulted in a 4-day delay. The time to return to team training was delayed

by 43 days in the presence of a Westpoint grade III injury, while the presence of a concomitant cartilage injury resulted in a 10-day delay. Finally, return to official match play was delayed for 44 (SE 3.8) days in the presence of a Westpoint grade III injury and 9 (SE 4.2) days in the case of a cartilage injury respectively. A correlation between age and rehabilitation times was observed, whereby an additional day of rehabilitation and team training is required for each year younger a player is.

Conclusion
The majority of isolated, unstable syndesmotic injuries occurred during match-play were non-contact injuries (64%). A majority of these injuries were classified as Westpoint grade IIB (68%), with the remaining being graded as Westpoint grade III. Ninety-five percent of injured players return to match-play within 6 months post-operatively. Grade III syndesmotic injuries are 4 times more likely to involve cartilage injury than grade IIB injuries. Therefore, players who sustain a grade IIB injury will return to match play on average 48 days earlier than those players who sustain a grade III injury. The grade of isolated unstable ankle joint distal syndesmosis injury, the presence of concomitant cartilage injury as well as player age at the time of injury all influence the outcome of time to return to match play.

What are the new findings?
This study establishes the average time required to start with on field rehabilitation, team training and official match play in professional football players who were surgically treated for isolated unstable ankle joint distal syndesmosis injuries. We also identified 3 specific injury characteristics (a grade III injury, a combined cartilage lesion and young age) as predictors for a delayed return to match play.

How might it impact on clinical practice in the near future?
This study informs the football player with an unstable syndesmosis ankle injury on the expected postoperative time to return to play. It also presents 3 specific injury characteristics (a grade III injury, a combined cartilage lesion and young age) that can predict a prolonged rehabilitation time.

INTRODUCTION

Although the incidence of isolated syndesmosis injury among professional football players is low, with an incidence of 0.05 per 1000hours of exposure, it

constitutes a time loss injury in football with a mean absence of 39 days and an injury burden of 1.8 days absence per /1000 hours of exposure.[1,2] Diagnosis of syndesmosis injury is often difficult as physical examination is of limited value.[3,4] However, with the use of magnetic resonance imaging (MRI), early treatment is increasingly being initiated to prevent long-term sequala such as anterolateral ankle soft-tissue impingement (24%), chronic pain (33%), heterotopic ossifications (3,4%), recurrence (7%) and even chronic instability (20,3%) associated with this type of injury.[5-8]

Current treatment algorithms subdivide syndesmotic injury based on MRI findings (tear of the AITFL, IOL and PITFL) and physical examination (squeeze test & external rotation stress test).[3-5,7] Based on MRI findings syndesmosis injury can be classified as Westpoint grade I (sprain of the antero-inferior talofibular ligament [AITFL]) and Westpoint grade III (complete disruption of all syndesmotic ligaments with frank diastasis).[5] MRI findings for Westpoint grade IIA (stable) & IIB (unstable) include complete tear of the AiTFL and injury of the Interosseous ligament [IOL] and are differentiated based on a positive squeeze test.

Despite limited evidence most clinicians advocate conservative measures for stable low-grade syndesmosis injuries (Westpoint ≤IIB), while surgical stabilization of unstable high-grade (Westpoint ≥IIB) isolated syndesmotic injuries is the current standard treatment in elite athletes.[3,9]
The adoption of surgical stabilization for unstable syndesmotic injuries in elite athletes is the result of claims that surgical stabilization for syndesmotic instability might result in a shortened return to play.[10,11] Thus far, studies investigating return to play after surgical treatment of unstable syndesmosis injuries are scarce. A recent publication by Calder et al. reported the results of 36 athletes who underwent surgical stabilization of a Westpoint grade IIB unstable syndesmotic injury. Athletes were able to return to play after a mean 65 days (range, 27 to 104 days).[12] Despite the methodological quality of this study, the evidence from this study is limited as this cohort consisted of athletes participating in various sports and there's a great variability in the outcome of clinical testing (like the squeeze test).

The aim of this study was to determine the typical time to return to on field rehabilitation, team training and official match play following surgical stabilization for isolated unstable ankle joint distal syndesmosis injuries amongst a cohort of 110 professional male football players.

METHODS

Patient selection

In this retrospective cohort study, we evaluated the time required to return to sport-specific rehabilitation, team training and to first match play, in a consecutive series of professional football players that underwent surgical fixation of an isolated, unstable ankle joint syndesmosis injury at our institution between January 2012 and December 2017.

Patients were eligible for this study (Anti-Doping Lab Qatar review board #E2017000259) if they were registered professional football players with the QFA (Qatar Football Association, compromising around 480 players per season), had sustained a syndesmotic ankle injury, graded as Westpoint (Clinical) IIB or III, and had completed a minimal post-operative follow up of 6 months. The pre-operative classification of syndesmotic injury was based upon clinical examination (squeeze test and external rotation test) and a detailed magnetic resonance imaging (MRI) report by a radiologist at our Center that was blinded to the patient's details. Only lesions graded by MRI to be isolated IIB and III were included in the study and indicated for surgical fixation (Figure 1).

Football players with injuries older than 6 weeks, any previous ankle surgery or with a concomitant lateral or medial malleolar ankle fracture at the time of surgery were excluded from the study. A total of one hundred twenty-three players were initially indicated for arthroscopy of which six were excluded from the study for occult concomitant fracture on MRI, three excluded for having previous ankle surgery and four were excluded as they were identified as stable lesions during surgery. No patients were lost to follow up thanks to the direct contact with the player's physiotherapists and football club team doctors.

After surgery, the time to return to sport-specific rehabilitation was documented and retrieved from the player's physiotherapist while the time to return to team training and official first match was documented and retrieved from the player's football club team doctor.

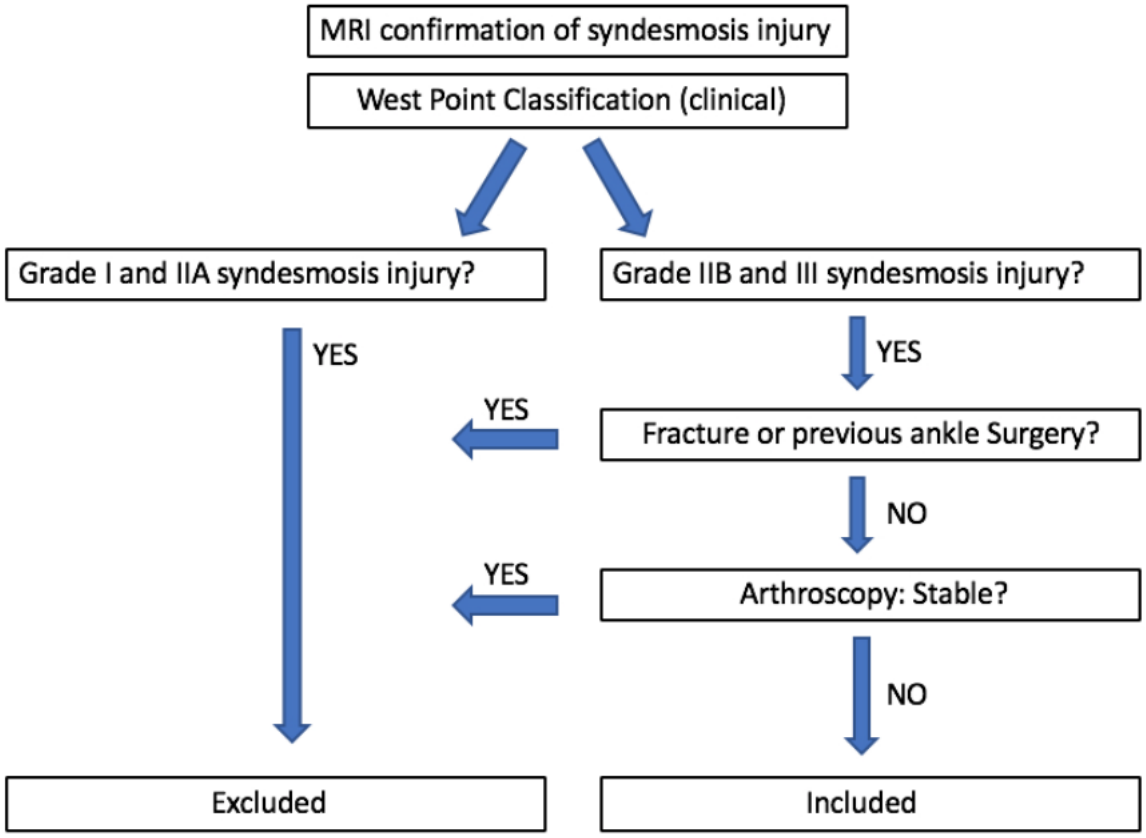

Figure 1. Selection algorithm to decide on inclusion in the study.

The criteria to return to sport-specific rehabilitation that we used were: normal gait, stability in single-leg balance stance, able to perform painfree a single-leg calf raise, a deep "catcher squat", a single-leg hop and jog limp-free without any shortened stride or step length.

Patient Involvement

All football players who participated in the study signed a pre-operative surgical informed consent mentioning they agreed to participate in the study. The players were all involved in the conduct of the study since they were all contacted to report their exact dates of return to play. The research question did not change during the study progress and all players were informed on the results of their personal study data.

Surgical Technique

A fellowship-trained Sports Medicine Surgeon (PdH) performed all surgical procedures. General anesthesia was used in all procedures. Patients were positioned in the supine position. Standard 2-portal anterior ankle arthroscopy (anteromedial and anterolateral) was performed to confirm syndesmotic instability. The distal syndesmosis was considered unstable if a 5.0 mm shaver blade could be gently introduced into the distal syndesmosis, 1 cm above the talocrural joint. In our study, 2 patients had a negative shaver blade test during arthroscopy. Consequently, these 2 patients were excluded from the study. Lateral incision over the distal fibula 2-3 cm proximal to the tibiotalar joint was performed after confirmation of an isolated unstable syndesmosis.

In order to achieve correct fibular reduction into the tibial incisural notch, a reduction Weber clamp was used under fluoroscopic control in anteroposterior and lateral views. A Kirschner wire (K-wire) was inserted from the distal fibula (incision) to the distal tibia under fluoroscopy in anteroposterior and lateral views with the ankle in neutral position. The K-wire was overdrilled by a 4-mm cannulated drill and the suture button (Tightrope, Arthrex®) was inserted. The suture button was passed through the medial tibial cortex under fluoroscopic control and the tibiofibular joint reduction was completed after tightening the pulley.
After confirmation under fluoroscopy that the suture button is located flush on the bone, the construct was hand tied on the lateral side with the ankle in neutral position. A second suture button was placed 1 cm proximal to the other one in case of a combined posterior malleolar fracture or deltoid ligament rupture.

Post-operatively, all players were allowed to mobilize immediately and partial weightbearing in a pneumatic cam boot was initiated for the first 10 days post-operatively. Range of motion (ROM) was not restricted from day 1 post-operatively. After 10 days of partial weightbearing, the boot was removed and full mobilization of the ankle was then allowed. Strengthening and running were allowed after 3 weeks. Full return to competitive sports was permitted along the individual progression.

Statistical Analysis

Statistical analysis was performed with the software MedCalc. Continuous variables were expressed as mean and standard deviation (SD), while

categorical variables were expressed as the percentage of the entire group. The comparison between different subgroups was performed with the independent sample t-test for continuous variable, and with the Fisher exact test for categorical variables.
A Kaplan-Meier curve was calculated using three different end-points: the time to on field rehabilitation, the time to return to train with team and the time to return to first match. For each of these outcomes, the rate and standard error (SE) of the player that reached the endpoint at weekly time-points were calculated for the whole series and for the subgroups based on injury severity. The Log-rank test was used to compare the curves of the two subgroups based on injury severity.
A multivariate analysis was performed in a stepwise fashion using as outcomes: the time to on field rehabilitation, the time to return to train with team and the time to return to first match. This multivariate analysis used as independent variables: the patients' characteristics, age, injury severity, presence of concomitant cartilage injury, re-injury, dominant leg, match injury and contact injury. The multivariate analysis was corrected in order to have 25 years as the reference age of the players to calculate the constant coefficients. Values were considered as statistically significant with a $p<0.05$.

RESULTS

Population and injury characteristics

A total of one hundred ten male football players with a mean age at surgery of 24.9 ± 4.0 years old were included in this study. All of them (100%) were available for the evaluation and their mean follow up duration was 11.3 ± 3.0 months.

The syndesmotic injury involved predominantly the dominant leg (60%) and most commonly resulted from a non-contact mechanism (64%) such as twist (36%) or landing from a jump (16%).

The majority of the injuries occurred during match play (69%). Only 7% of them were classified as re-injury. According to arthroscopic evaluation, 75 (68%) were graded as a grade IIB and 35 (32%) were as a grade III injury.

Concomitant cartilage injury was observed in 23 patients (Table I).

Table I: Patients and injury characteristics				
	Total	Grade IIB (n=75)	Grade III (n=35)	P-value
Age	24.9 ± 4.0	25.4 ± 4.1	23.8 ± 3.3	=0.0586
Dominant leg				=1.0000
No	45 (40%)	31 (41%)	14 (40%)	
Yes	65 (60%)	44 (59%)	21 (60%)	
Grade				
IIB	75 (68%)	75 (100%)	0 (0%)	NA
III	35 (32%)	0 (0%)	35 (100%)	
Cartilage injury				=0.0019*
No	87 (79%)	66 (88%)	21 (60%)	
Yes	23 (21%)	9 (12%)	14 (40%)	
Reinjury				=1.0000
No	102 (93%)	69 (92%)	33 (94%)	
Yes	8 (7%)	6 (8%)	2 (6%)	
Event				
Training	34 (31%)	25 (33%)	9 (26%)	=0.5091
Match	76 (69%)	50 (67%)	26 (74%)	
Mechanism				=0.0001*
Contact	40 (36%)	15 (20%)	25 (71%)	
Non contact	70 (64%)	60 (80%)	10 (29%)	
Situation				=0.0001*
Tackled	40 (36%)	15 (20%)	25 (71%)	
Twist	40 (36%)	33 (44%)	7 (20%)	
Landing from jump	17 (16%)	14 (19%)	3 (9%)	
Unknown	13 (12%)	13 (17%)	0 (0%)	

Table I: Patient and Injury Characteristics

Return to sport outcomes

The mean time required to begin the sports-specific rehabilitation was 37 ± 12 days, while the time to return to train with team was 72 ± 28 days. The first official match after surgical syndesmosis fixation was played on average after 103 ± 28 days (Table II).

Table II: Return to sport				
	Time landmarks	Return to on field rehabilitation	Return to train with team	Return to first match
0 months	**7 days (1 week)**	**-**	**-**	**-**
	14 days (2 weeks)	0% (SE 0%)	-	-
	21 days (3 weeks)	4% (SE 2%)	0% (SE 0%)	-
	28 days (4 weeks)	19% (SE 4%)	1% (SE 1%)	-
1 month	**35 days (5 weeks)**	**55% (SE 5%)**	**4% (SE 2%)**	**0% (SE 0%)**
	42 days (6 weeks)	76% (SE 4%)	10% (SE 3%)	0% (SE 0%)
	49 days (7 weeks)	86% (SE 3%)	20% (SE 4%)	1% (SE 1%)
	56 days (8 weeks)	94% (SE 2%)	35% (SE 5%)	2% (SE 1%)
2 months	**63 days (9 weeks)**	**97% (SE 2%)**	**47% (SE 5%)**	**4% (SE 2%)**
	70 days (10 weeks)	98% (SE 1%)	56% (SE 5%)	5% (SE 2%)
	77 days (11 weeks)	99% (SE 1%)	67% (SE 4%)	16% (SE 3%)
	84 days (12 weeks)	100% (SE 0%)	69% (SE 4%)	29% (SE 4%)
3 months	**91 days (13 weeks)**	**-**	**73% (SE 4%)**	**44% (SE 5%)**
	98 days (14 weeks)	-	79% (SE 4%)	53% (SE 5%)
	105 days (15 weeks)	-	86% (SE 3%)	60% (SE 5%)
	112 days (16 weeks)	-	93% (SE 2%)	66% (SE 5%)
4 months	**119 days (17 weeks)**	**-**	**95% (SE 2%)**	**76% (SE 4%)**
	126 days (18 weeks)	-	97% (SE 2%)	85% (SE 3%)
	133 days (19 weeks)	-	97% (SE 2%)	91% (SE 3%)
	140 days (20 weeks)	-	98% (SE 1%)	93% (SE 2%)
5 months	**147 days (21 weeks)**	**-**	**98% (SE 1%)**	**95% (SE 2%)**
	154 days (22 weeks)	-	98% (SE 1%)	97% (SE 2%)
	161 days (23 weeks)	-	100% (SE 0%)	97% (SE 2%)
	168 days (24 weeks)	-		97% (SE 2%)
6 months	**175 days (25 weeks)**	**-**	**-**	**98% (SE 1%)**
	182 days (26 weeks)	-	-	98% (SE 1%)
	189 days (27 weeks)	-	-	99% (SE 1%)
	196 days (28 weeks)	-	-	100% (SE 0%)
Mean ± SD		**37 ± 12 days**	**72 ± 28 days**	**103 ± 28 days**

Table II. Return to Sport

Mean time-loss for respectively unstable grade IIB injuries and grade 3 injuries are presented in Table III.

Table III: Return to sport according to injury severity

Time landmarks		Grade IIB injuries (n=75)			Grade III injuries (n=35)		
		Return to on field rehabilitation	Return to train with team	Return to first match	Return to on field rehabilitation	Return to train with team	Return to first match
0 months	**7 days (1 week)**	- -	- -	- -	- -	- -	- -
	14 days (2 weeks)	0% (SE 0%)	- -	- -	- -	- -	- -
	21 days (3 weeks)	7% (SE 3%)	0% (SE 0%)	- -	- -	- -	- -
	28 days (4 weeks)	28% (SE 5%)	1% (SE 1%)	- -	0% (SE 0%)	- -	- -
1 month	**35 days (5 weeks)**	**79% (SE 5%)**	**7% (SE 3%)**	- -	**3% (SE 3%)**	- -	- -
	42 days (6 weeks)	96% (SE 2%)	15% (SE 4%)	0% (SE 0%)	34% (SE 8%)	- -	- -
	49 days (7 weeks)	99% (SE 1%)	29% (SE 5%)	1% (SE 1%)	57% (SE 8%)	0% (SE 0%)	- -
	56 days (8 weeks)	100% (SE 0%)	49% (SE 6%)	3% (SE 2%)	80% (SE 7%)	3% (SE 3%)	- -
2 months	**63 days (9 weeks)**	- -	**65% (SE 6%)**	**7% (SE 3%)**	**91% (SE 5%)**	**9% (SE 5%)**	- -
	70 days (10 weeks)	- -	77% (SE 5%)	8% (SE 3%)	94% (SE 4%)	9% (SE 5%)	- -
	77 days (11 weeks)	- -	95% (SE 3%)	23% (SE 5%)	97% (SE 3%)	9% (SE 5%)	- -
	84 days (12 weeks)	- -	97% (SE 2%)	43% (SE 6%)	100% (SE 0%)	9% (SE 5%)	0% (SE 0%)
3 months	**91 days (13 weeks)**	- -	**100% (SE 0%)**	**63% (SE 6%)**	- -	**14% (SE 6%)**	**3% (SE 3%)**
	98 days (14 weeks)	- -	- -	76% (SE 5%)	- -	34% (SE 8%)	3% (SE 3%)
	105 days (15 weeks)	- -	- -	84% (SE 4%)	- -	54% (SE 8%)	9% (SE 5%)
	112 days (16 weeks)	- -	- -	93% (SE 3%)	- -	77% (SE 7%)	9% (SE 5%)
4 months	**119 days (17 weeks)**	- -	- -	**100% (SE 0%)**	- -	**86% (SE 6%)**	**26% (SE 7%)**
	126 days (18 weeks)	- -	- -	- -	- -	91% (SE 5%)	51% (SE 8%)
	133 days (19 weeks)	- -	- -	- -	- -	91% (SE 5%)	71% (SE 8%)
	140 days (20 weeks)	- -	- -	- -	- -	94% (SE 4%)	77% (SE 7%)
5 months	**147 days (21 weeks)**	- -	- -	- -	- -	**94% (SE 4%)**	**83% (SE 6%)**
	154 days (22 weeks)	- -	- -	- -	- -	94% (SE 4%)	91% (SE 5%)
	161 days (23 weeks)	- -	- -	- -	- -	100% (SE 0%)	91% (SE 5%)
	168 days (24 weeks)	- -	- -	- -	- -	- -	91% (SE 5%)
6 months	**175 days (25 weeks)**	- -	- -	- -	- -	- -	**94% (SE 4%)**
	182 days (26 weeks)	- -	- -	- -	- -	- -	94% (SE 4%)
	189 days (27 weeks)	- -	- -	- -	- -	- -	97% (SE 3%)
	196 days (28 weeks)	- -	- -	- -	- -	- -	100% (SE 0%)
Mean ± SD		32 ± 7 days	57 ± 14 days	88 ± 16 days	49 ± 11 days	105 ± 22 days	136 ± 21 days

Table III. Return to sport according to injury severity

One month post-operatively, 55% of players had started on field rehabilitation, 4% had already returned to team training and none had participated in an official match.

At 2 months, almost all of the players (97%) had started sport-specific rehabilitation, 47% were training with their team and 4% had already participated in an official match. At 3 months 73% of the players were allowed to train with their team and nearly half (44%) had played an official match. The return to training and to match play were respectively 95% and 76% at 5 months and 98% and 95% at 6 months (Figure 2).

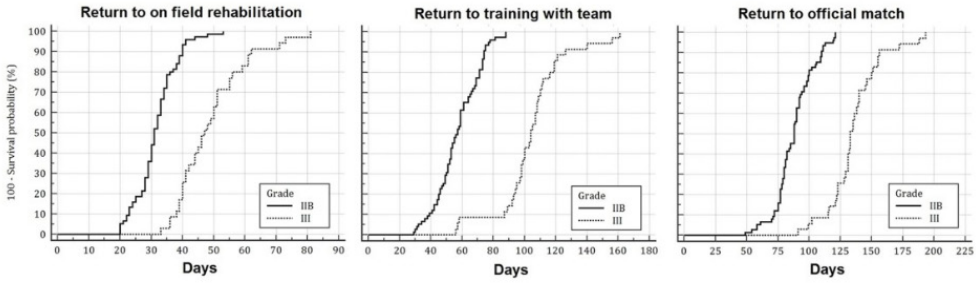

Figure 2. Graphic image on the time towards return to on field rehabilitation, training with team and first match.

During the post-operative follow-up, 5 patients (5%) experienced delayed wound closure. None of these patients required additional treatment and all healed within 50 days after surgery.

Analysis based on injury severity

Patients that were diagnosed as having a grade III injury (32%) presented with a significantly higher rate of concurrent cartilage injury (p=0.0019), almost a 4-fold increase compared to patients with a grade IIB injury (68%). Moreover, in patients with grade III injury, the mechanism of injury was predominantly due to contact with another player during a tackle (p=0.0001); more than a 3-fold increase compared to patients with grade IIB injuries. Patient's age, reinjury-type and match or training injury were not different between these two subgroups.

Regarding the sport-related outcomes, a longer time to sport-specific rehabilitation (p<0.0001), return to team training (p<0.0001) and return to match play (p<0.0001) were observed in those patients diagnosed with a grade III injury (Table 3).

In particular, all players with grade IIB injuries had started sport-specific rehabilitation after 8 weeks, compared to 80% with grade III injuries. Similarly, all players classified as having sustained a grade IIB injury, had returned to team training after 3 months, compared to only 14% of the athletes with a grade III injury.

Finally, all athletes with a grade IIB injury had played an official match within 4 months compared to only 26% of those with a grade III injury (Figure 3).

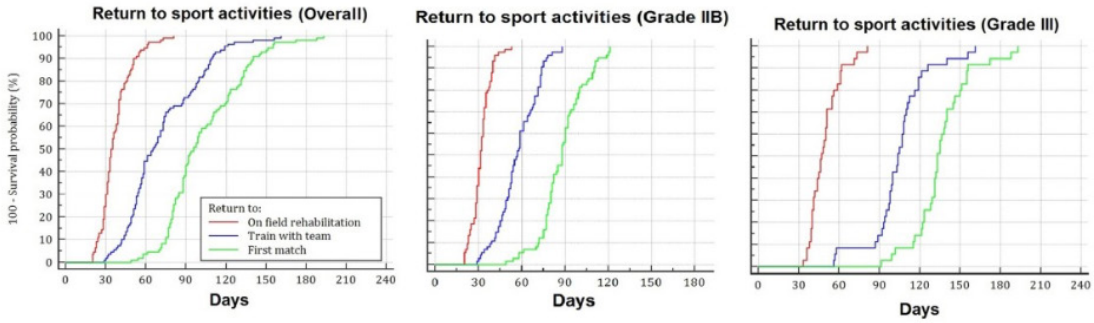

Figure 3. Graphic Image on the specific characteristics of return to sports (overall, Grade 2B, Grade 3)

According to the multivariate analysis, the injury severity, the presence of cartilage injury and the age at surgery were significant predictors (p<0.0001) in all the three models of return to sport-specific rehabilitation, return to team training and return to match play (Table IV).

Table IV: Multiple regression models for return to activity outcomes

	Return to on field rehabilitation			Return to train with team			Return to first match		
Independent variables	**Coeff.**	**SE**	**P-value**	**Coeff.**	**SE**	**P-value**	**Coeff.**	**SE**	**P-value**
(Constant)	31.2			56.4			87.5		
Grade III injury	15.7	1.8	<0.0001	43.0	3.6	<0.0001	43.8	3.8	<0.0001
Cartilage injury	4.4	2.0	=0.0277	10.7	4.0	=0.0096	9.2	4.2	=0.0311
Age (years)	-0,4	0.2	=0.0460	-0.8	0.4	=0.0475	-1.1	0.4	=0.0121
Injury during training	-	-	n.s.	-	-	n.s.	-	-	n.s.
Reinjury	-	-	n.s.	-	-	n.s.	-	-	n.s.
Contact injury	-	-	n.s.	-	-	n.s.	-	-	n.s.
Dominant leg	-	-	n.s.	-	-	n.s.	-	-	n.s.
Significance of the model	P<0,0001			P<0,0001			P<0,0001		
Residual standard deviation	8.0			16.3			16.9		

Table IV. Multiple regression models for return to sport outcomes

The average time expected to begin sport-specific rehabilitation for a 25 years old player, with a grade IIB injury and no cartilage involvement was 31.2 days. This is lengthened by an average of 16 days in the presence of a grade III injury, 4 days in the case of cartilage injury, and 0.4 days every year younger than 25, or shortened by 0.4 days every year older than 25.
Similarly, the expected time to return to team training for the same profile of player (25 years old, type IIB injury, no cartilage injury) was 56.4 days, which is delayed by a mean 43 days in the case of a grade III injury, 10 days in the case of a cartilage injury and 0.8 days every year younger than 25, while shortened 0.8 days for every year older than 25. Finally, the return to official match play was expected after an average of 87.5 days, which is delayed of 44 days in the case of a grade III injury, 9 days in the case of a cartilage injury and of 1.1 day for every year younger than 25, while shortened by 1.1 day for every year older than 25.

DISCUSSION

The most important finding in this study were that the mean time required to begin sport-specific rehabilitation was 37 ± 12 days, the time to return to team training was 72 ± 28 days and the first official match was played on average after 103 ± 28 days, with ninty-five percent of injured football players returned to match-play within 6 months after surgery. In addition, we identified that

injury severity, concomitant cartilage injury and the age at time of surgery are associated with a prolonged rehabilitation.

Association between Injury and Return to Play

A recent prospective study by Calder et al reported on 36 athletes with an unstable grade IIB ankle injury. In their surgically treated cohort, they observed a mean return to sport of 64 days. No comparison with grade III injuries was described but they did find an additional increase in return to sports of 21.5 days if a concomitant deltoid ligament lesion was apparent. No differentiation was made on whether return to sports meant sport-specific rehabilitation, team training or first official match.[12]

In division I collegiate American football players following high ankle sprains, there is a clear association between injury severity as measured by physical examination and the time to return to unrestricted play. Although these injuries are less common than isolated lateral ankle sprains, their clinical course is shown to be more protracted and unpredictable.[13]
When compared to time loss to play with lateral ankle sprains, numerous authors report a significantly greater time loss to play with high ankle sprains.[12-16]

In professional football players this results in a higher inability to train (11.7 vs 3.5 training sessions) and play official matches (1.4 vs 0.3 matches) compared to those with simple lateral ankle sprains only.[15] Furthermore, unstable syndesmotic injuries are associated with persistent ankle pain (40%) and premature ankle arthritis (42%) if left untreated.[16]
A report on collegiate athletes with syndesmotic sprains also found an association between injury height on physical examination and time lost from athletic participation.[17] Although this study was not restricted to football players only (without the effect of sex or sport in their results), similar mean times lost from athletic participation are reported (13.4 vs 15.5 days).[17]

Ideal Rehabilitation Strategy

Furthermore, the ideal treatment regimen on the rehabilitation of high ankle sprains remains undefined due to the paucity of publications on the topic. The few studies that have presented rehabilitation protocols generally follow 3 phases:
(1) an early phase of pain and inflammation control;
(2) a middle phase focusing on mobility, function, and strengthening;

(3) a final phase of advanced training to prepare the athlete to return to competition.[18,19]

Another study suggested to establish a prediction model based on the association between, injury severity and time to recovery. [20] The data was collected over a period of 3 consecutive college football seasons and involved the clinical evaluation of the athlete's status together with a musculoskeletal ankle ultrasound to more clearly define the window for return to play in syndesmotic injuries.[20] Another study concluded that injury severity as measured by physical examination is more closely correlated with time to return to play than diagnostic ultrasound.[21]

Study Limitations

The most important limitation of our study is its retrospective design, as collection of return to play outcome data might have been influenced by recall bias. Furthermore, the treating physiotherapists and team medical staff had access to the imaging studies (MRI) and surgical notes, potentially biasing their progression through rehab and return to play decision. The return to play information might have been confounded by heterogeneity of the ankle pathology as well, as patients with concomitant osteochondral defects were included in this study. Also, the follow up was restricted to the time to return to official match play without further information on long-term functional outcome data.
Despite obvious shortcomings, this study is of major clinical significance. It is the first study to report on return to play outcome after surgical stabilization of isolated unstable syndesmosis injuries in a homogenous cohort of professional football players. In addition, it is the largest study looking at return to play after surgical stabilization of syndesmosis injuries overall.
Future studies should aim to prospectively collect return to play data (together with recurrence, revision and injury risk post return to unrestricted sports) in a similar sized cohort of surgically treated football players. In addition, future studies should blind the threating physiotherapist in order to eliminate any potential bias and patients with concomitant osteochondral lesions should be excluded to minimize confounding. Furthermore, a randomized prospective controlled trial comparing functional outcome and return to play outcome after surgical stabilization or conservative treatment of acute isolated unstable Westpoint grade IIB syndesmosis injuries is warranted.

CONCLUSION

In our cohort of professional football players, surgical stabilization of acute isolated unstable syndesmosis injury allowed for relatively quick return to field rehabilitation, team training and match play. We also identified that injury severity, concomitant cartilage injury and the age at time of surgery are predictive of a prolonged rehabilitation.

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Return to sport following lateral ankle ligament repair is under-reported: A systematic review

Journal of ISAKOS (2016)

Hunt K, Fuld R, Sutphin B, Pereira H, D'Hooghe P

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ABSTRACT

Importance:
Ankle sprains are the most commonly occurring musculoskeletal injury. Reconstruction of the lateral ligament complex is often required for athletes with recurrent instability, or high-grade acute sprains, in order to return to their preinjury level of sport.

Objective:
The purpose of this systematic review was to evaluate the spectrum, prevalence and quality of evidence regarding return to sport timeline following lateral ligament surgery.

Evidence review:
A search was conducted of Embase and Medline databases from the earliest possible entry to November 2016. Studies reporting a timeline regarding return to play (RTP) following lateral ankle ligament reconstruction were included in this review.

Findings:
Of 3184 total articles, 20 articles evaluating 489 athletes met the criteria and were included for review. Thirteen of the 20 papers were used to calculate a weighted mean time to RTP of 4.7 months. Overall, both the frequency and quality of RTP criteria and reporting were very low.

Conclusions and relevance:
The current review identifies a clear deficiency in the literature pertaining to consistent, meaningful postoperative RTP timeline following lateral ankle ligament repair. Published studies vary considerably in the metrics used for measuring patient-reported outcomes, and very few actually track them. Further studies on outcomes following ankle ligament repair should include clear and consistent metrics for return to sport and level of play. Standardized and reproducible criteria for reporting RTP for athletes will improve the utility and applicability of outcomes data as surgical and rehabilitative techniques continue to advance.

What is already known?
►There is extensive literature describing techniques and outcomes for lateral ankle ligament repair and reconstruction.

►There is existing literature to support the ability of athletes to return to play (RTP) following lateral ankle ligament stabilization, but timeline of return is generally not included in those reports.
►There is a need to understand rates and timing of return to sport following lateral ligament repair in order to compare treatments, procedures and rehabilitation techniques.

What are the new findings?
►These articles suggest a rate of return to sport of 85% following lateral ankle ligament repair in athletes, at an average of 4.7 (+/−1.5) months.
►There are more than 360 manuscripts describing a postoperative clinical outcome of lateral ankle ligament repair, yet only 5.5% of these detail an RTP timeline as a reported outcome metric, indicating a clear deficiency in the literature.
►We propose a better defined structure and protocol for assessing the readiness of athletes to RTP.

INTRODUCTION

Ankle sprains (particularly inversion sprains involving the lateral ligament complex) are exceedingly common injuries, with an incidence of 2.15 per 1000 person-years in the US general population.[1] The incidence of injury increases with exposure to sport, with ankle sprains comprising 31% of all injuries in soccer and 45% of all injuries in basketball.[2] In a cohort of 10393 basketball participations, the rate of ankle injury was 3.85 per 1000 participations.[3] Although more common in collision sports, ankle sprain remains the most common musculoskeletal injury regardless of sport or exposure type.[3–6]

Ankle sprains are most commonly caused by inversion of a plantar flexed foot, resulting in injury to the lateral ligament complex of the ankle joint.[7] Sandelin et al[8] observed that 75% of ankle ligament sprains were of the lateral ligament complex. Hawkins et al[9] had similar findings, with 80% of sprains being to the lateral ligament complex. Lateral ankle stability is ordinarily maintained by the surrounding ligamentous structures, including the anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL) and the posterior talofibular ligament.

The ATFL prevents anterior talar displacement and is the primary structure injured in an ankle sprain; it has been observed that 2/3 of all ankle injuries are isolated to the AFTL.[10] The CFL prevents excessive inversion and is the second

structure injured during an ankle sprain.[10] Broström[11] observed combined injury of the ATFL and CFL in 20% of ankle sprains and that the CFL was never ruptured alone.

Several surgical techniques have been described for repair of the lateral ligament complex. In 1966, Broström[12] was the first to describe direct repair of a remnant ATFL ligament with suture. In 1980, Gould et al[13] modified this procedure with advancement of the inferior extensor retinaculum; this procedure was further modified by Hu et al,[14] who used bone tunnels or suture anchors to repair both the ATFL and CFL back to the fibula. Patients whose ligament's remnants are preserved are good candidates for reconstruction with the modified Broström-Gould procedure. Additional techniques describe reconstruction with an alternative tendon graft, such as allograft or autograft, and arthroscopic techniques for primary ligament repair.

Surgical intervention of acute ankle sprains has not consistently been shown to improve long-term function.[15] However, in 20%–40% of ligamentous ankle injuries, chronic ankle instability develops, resulting in short-term and long-term functional deficits[16] and risk to other structures,[17] necessitating surgical correction.[18,19] In these cases, without surgical intervention, improvement in symptoms is unlikely.[20] Patients intending to return to their preinjury level of sports participation may elect for surgery in order to maximize recovery and function. Surgical outcomes for lateral ligament reconstruction are highly favorable in the general population; however, data regarding rates of return to preinjury sports participation following lateral ankle ligament injury and surgery are limited. Even less often reported is the timeline of return to play (RTP). The highly competitive nature of modern sports and the associated multifaceted pressures for rapid RTP following injury underscore the importance of understanding surgical procedures and rehabilitative techniques that may lead to a consistent and predictable return protocol during management of injured athletes. Moreover, our ability to compare novel surgical and rehabilitation techniques is predicated on consistent use of outcome measures and RTP metrics. Proper diagnosis and early intervention may facilitate an earlier return to sport and decreased reinjury rates.

This systematic review aims to evaluate all available literature regarding postoperative return to sport following surgical repair of the lateral ankle ligament complex. The primary purpose was to evaluate the current literature describing an RTP timeline following lateral ankle ligament repair, including how

often an RTP timeline is being measured and the metrics being used to describe the RTP timeline.

METHODS

Literature search

A systematic literature search was conducted for articles on surgical repair of lateral ankle ligaments for acute sprain or chronic instability. Articles for review were obtained from a search of Medline and Embase databases, from 1953 up to November 2016, using the search headings 'ankle ligament surgery' and 'ankle sprain instability repair'. Information collected included year of publication, number of athletes, surgical technique, RTP metric, RTP timeline, RTP performance data, patient-reported outcomes measures and functional outcome measures.

Study selection

We independently identified and screened published studies by their title and abstract. Only articles written in English were considered. Initial exclusion criteria included:
(1) no abstract;
(2) no reported clinical outcomes (basic science, radiographic, anatomical study);
(3) a review paper (review or meta-analysis).

Manuscripts with abstracts including clinical outcomes from surgical stabilization (varying procedures) of the lateral ankle ligaments were read fully to assess for RTP timeline metrics. To be included in this review, the study must have contained:

(1) patient(s) who participate in athletic activities;
(2) RTP timeline as an outcome metric or result;
(3) clinical outcome(s) following lateral ankle ligament repair.
Once inclusion criteria were met and papers read fully, those bibliographies were searched for additional relevant papers.

Statistical analysis

Using descriptive statistics from the articles included in this review, the weighted mean and weighted SD for time to RTP were calculated. Rate of RTP was described by percentage of all athletes who were able to return fully.

RESULTS

Literature search

The Medline search yielded a combined 2481 results and the Embase search yielded an additional 703 independent results. Of the 3184 total results, 360 articles (11.3%) were identified as having any clinical outcome post lateral ankle ligament stabilization.
Articles that did not report an RTP timeline were then excluded, leaving 54 papers (15.0%) that discussed an RTP timeline in some capacity. Twenty (5.5%) of the RTP timeline papers met the secondary criteria for inclusion in this review article. The other 34 papers were excluded primarily because they did not report an RTP timeline as a result or outcome metric. Most commonly, the RTP timeline was only described as part of a postoperative protocol without report of outcome, which did not meet the standards for inclusion in this review (figure 1).

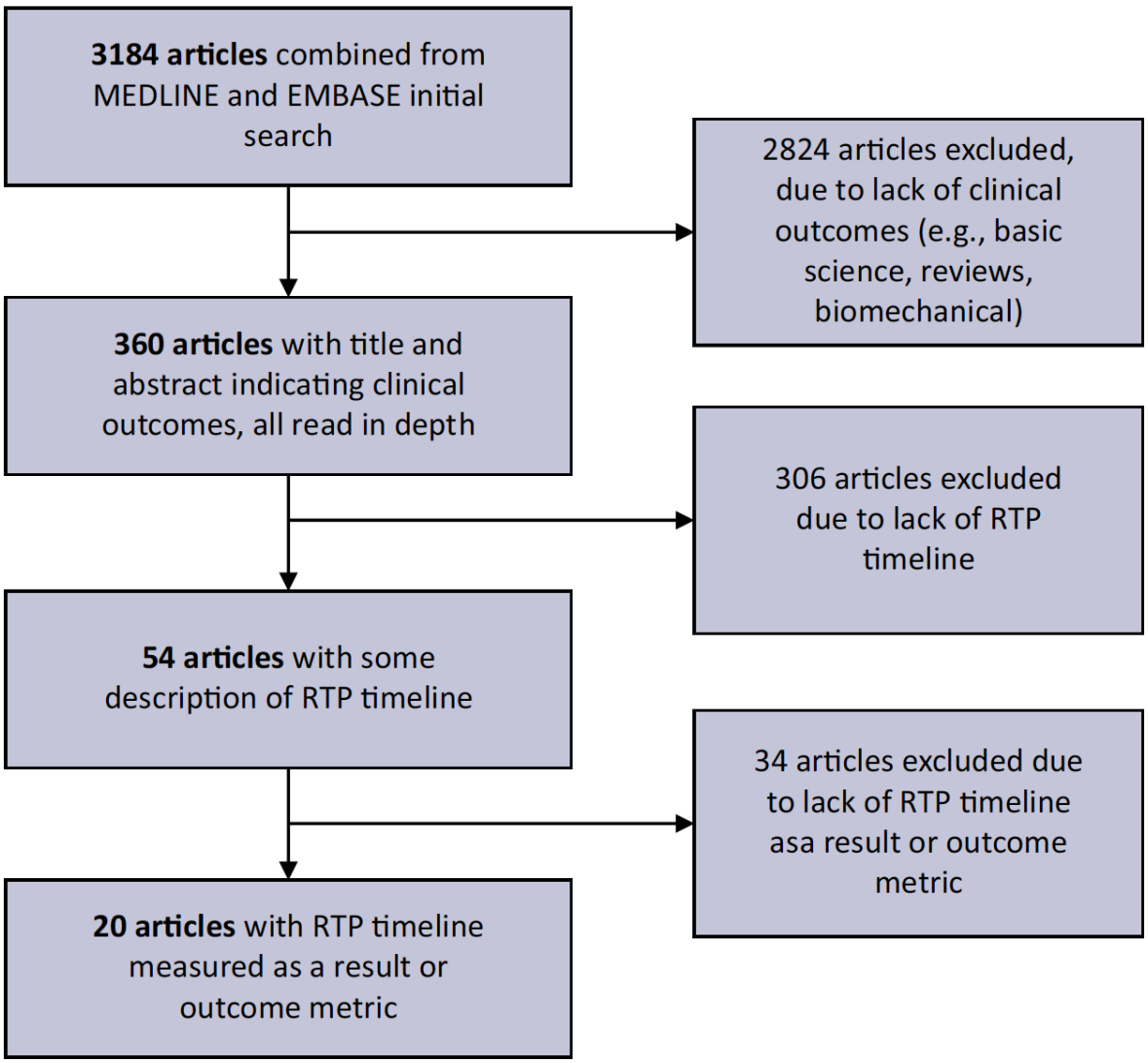

Figure 1. Literature search results ('RTP = return to play').

RTP metrics

Studies were not uniform in their descriptions of RTP timeline. Twelve studies reported an average time to return to sport for all athletes (10 studies[21–30] included a range, 2 studies[31 32] did not). Three studies[33–35] described mean time to returning to various different exercises (eg, jogging, jumping and running) for all athletes but lacked explicit report of return to sport. Two studies[36 37] reported the specific return time for each individual athlete. Two studies[38 39] reported the number of athletes returning to sport at specified time intervals. One study[40] reported a range time for all athletes. RTP metrics and results for all included studies are shown in table 1.

For the purposes of calculating a weighted mean and weighted SD for time to RTP, 13 of the 20 papers provided adequate data to include in this review. Three papers, all written by the same author, were excluded from statistical analysis because they only measured time to return to specific activities, not to sport.[33–35] One article[38] was excluded because RTP was measured as the number of athletes able to return by a specified time (without providing a mean time), and three papers[21 25 29] were excluded because the numbers of athletes returning to play were not specified.

For those articles that reported on whether or not athletes were able to return to preinjury level of participation, only those reporting athletes who were able to return fully were included in the weighted mean. Thirteen articles with a total of 281 athletes
resulted in a weighted mean time to RTP of 4.7 months, with a weighted SD of 1.5.

Table 1 Summary of articles and RTP metrics used in systematic review

Author	Year	RTP metric	RTP timeline data	RTP performance data/notes
Matsui	2016	Mean time for all athletes (with range)	1. Arthroscopic group: 16.5 weeks (range 12–22) 2. Open group: 17.1 weeks (range 13–22)	No recurrence of instability in either group; possibly first study to compare open versus arthroscopic for lateral instability of ankle
White	2016	Median time to training and full sport (with range)	1. Median time to return to training: isolated injuries (57 days, range 49–110), associated injuries (86 days, range 63–152), p<0.001 2. Median time to return to play: isolated injuries (72 days, range 56–127), associated injuries (5 days,* range 82–178), p<0.001	All professional athletes returned to preinjury levels (2/42§)
Yoo	2016	Number returned to sport at specified time intervals	Percent returned to sports activity at 12 weeks: 1. with internal brace (81.8%) 2. without internal brace (7%†); p<0.001	N/A
Cho*	2015	Mean time to return to specific exercises, based on VAS	Jogging (0.2 weeks*), sprint running (3.8 weeks*), jumping (11.4 weeks)	N/A
Cho†	2015	Mean time to return to specific exercises, based on VAS	Jogging (8.4 weeks), spurt running 2.5 weeks*), jumping (9.2 weeks)	N/A
Cho‡	2015	Mean time to return to specific exercises, based on VAS	1. suture bridge: jogging (9.8 weeks), spurt running (3.8 weeks*), jumping (0.6 weeks*) 2. suture anchor: jogging (0.4 weeks*), spurt running (4.5 weeks*), jumping (13.8 weeks)	Statistically significant difference in jumping; 44/45 athletes returned to preinjury level
Giannini	2015	Mean time for athletes (with range) back to preinjury level	(6 months — for 26/31 patients) (–8 months§)	5/31 did not return to preinjury level; 3 professional players resumed full activity
Jung	2015	Mean time for all athletes (with range)	3.1 months (6 weeks–12 months)	N/A - no specifics on number of athletes
Buerer	2012	Mean time for all athletes (with range)	.7-month average§ (–12 months†)	No correlation with sport and RTP time; no specifics on number of athletes
Takao	2012	Mean time for all athletes (with range)	1. With external support — functional treatment (6.3 weeks, range 3–12 weeks), surgery (5.7 weeks, range 3–8 weeks) 2. Without external support: functional treatment (6.0 weeks,* range 8–48 weeks), surgery (0.1 weeks,* range 8–15 weeks)	N/A — no specifics on number of athletes in each group
Ibrahim	2011	Mean time for all athletes (with range)	6.8 months (range 4–11 months)	N/A
Kramer	2011	Number returned to sport at specified time intervals	28 of 35 athletes at a median of 6 months (95% CI, 2.8–7.2 months) breakdown: 3 months,† 4 months (9), 5 months (0*), 6 months (9*), 7 months (0†), 9 months (1†), 12 months (28)	9 not at the same level as before their injury, 6/9 due to voluntary discontinuation of sport; soccer players less likely to return and more likely to return later
Morelli	2011	Mean time for all athletes (with range)	12 of 14 patients RTP after mean follow-up of 6 months (range 4–8 months)	The six professional athletes all reported a full recovery of their preoperative activity level; two reported a return to lower demanding sports activity, because of residual occasional pain occurring during high-level performances
Jones	2007	Specific time for individual athletes	3 months for 4/4 patients	2/2 athletes returned to sport, 2/2 recreational fitness returned to previous activity level
Coughlin	2004	Mean time for all athletes (with range)	(6.5 months) (–12 months‡)	4/28 patients had injury recurrence — still achieved eventual return to full activity
Solakoglu	2003	Mean time for all athletes	6 months	14/14 patients returned fully to amateur sports (basketball or football) or basic military training
Paterson	2000	Mean time for all athletes (with range)	Average time until return to sport was 12 weeks and all patients were able to return to sport, including 23/26 who were able to return to their preinjury level	Three did not return to preinjury level due lack of confidence in the ankle (2 people) or persistent pain (1 person)
Agoropoulos	1997	Range for all athletes	–6 months§	48/48 patients returned to sports without limitation
Hoy	1994	Mean time for all athletes	10 weeks, 18/25 athletes returned to preinjury level	Two patients did not return to sport
Leyshon	1982	Specific time for individual athletes	5 months, one athlete (case report)	Light training at 4 months, return to competition at 5 months

*Minimal invasive suture-tape augmentation for chronic ankle instability.
†A ligament reattachment technique for high-demand athletes with chronic ankle instability.
‡A prospective outcome and cost-effectiveness comparison between two ligament reattachment techniques.
RTP, return to play; VAS, Visual Analogue Scale for pain.

Articles that reported time in weeks and days were converted to months using the following conversions: 1 day is 0.033 months; 1 week is 0.23 months. Full results are reported in table 2.

Table 2 Mean time to return to play (RTP)

Author	Year	Athletes	RTP (months)
Matsui*	2016	19	3.8
Matsui†	2016	18	3.9
White	2016	30	2.4
Giannini	2015	26	6.0
Ibrahim	2011	14	6.8
Kramer	2011	28	6.0
Morelli	2011	12	6.0
Jones	2007	2	3.0
Coughlin	2004	28	6.5
Solakoglu	2003	14	6.0
Paterson	2000	23	2.8
Agoropoulos‡	1997	48	5.0
Hoy	1994	18	2.3
Leyshon	1982	1	5.0
Mean±SD			4.7±1.6

*Arthroscopic group.
†Open group.
‡Midpoint of 4–6 range chosen.

Data specifying ability to RTP, independent of timeline, were available for a total of 489 athletes (16 articles), of which 414 were reported to have returned to preinjury level of sports participation at follow-up, producing a pooled RTP rate of 85%, as shown in table 3.

Table 3 Rate of return to play

Author	Year	All athletes	Full return
Matsui	2016	37	37
White	2016	30	30
Yoo*	2016	22	18
Yoo†	2016	63	17
Cho‡	2015	45	44
Giannini	2015	31	26
Takao§	2011	54	54
Ibrahim	2011	14	14
Kramer	2011	35	28
Morelli	2011	14	12
Jones	2007	2	2
Coughlin	2004	28	28
Solakoglu	2003	14	14
Paterson	2000	26	23
Agoropoulos	1997	48	48
Hoy	1994	25	18
Leyshon	1982	1	1
Totals		489	414
Return percentage		85%	

*Returned at 12 weeks with internal brace.
†Returned at 12 weeks without internal brace.
‡Ligament reattachment technique.
§All surgical patients, including those using external bracing.

Comparison by intervention, injury and sport

Five of the 20 articles included some type of comparison between different groups and the outcome on RTP timeline.
Four articles discussed differences in surgical techniques and one article discussed differences in injury pattern. Cho et al[34] compared suture anchor and suture bridge group reattachment techniques for mean period to return to exercise and found that the suture bridge group returned to jumping earlier

when compared with the suture anchor group (10.6 weeks vs 13.8 weeks; p=0.038). Takao et al[29] compared functional treatment alone (F) with functional treatment with surgical repair (RF), for RTP both with and without the external support of soft ankle orthosis. With external support, the elapsed time between injury and return to full athletic activity was 6.3 weeks (F) and 5.7 weeks (RF) (p=0.0498). Without external supports, the elapsed time between injury and return to full athletic activity was 16.0 weeks (F) and 10.1 weeks (RF) (p<0.0001).

Matsui et al[26] compared arthroscopic (A) and open (O) repairs, and found no significant difference in mean time to return to sports activities for the two groups (16.5 weeks (A) vs 17.1 weeks (O); p=0.07). Yoo and Yang[38] compared arthroscopic Broström with an internal brace to arthroscopic Broström without an internal brace. They found a significant difference in the rate of returning to sports at 12 weeks (81.8% with internal brace vs 27% without internal brace; p<0.001). White et al[30] compared isolated lateral ligament injuries with lateral ligament injuries with associated injuries (eg, OCL, deltoid). They found median time to return to training was different for isolated (57 days) and associated (86 days) injuries (p<0.001). Additionally, median time to RTP was different for isolated (72 days) and associated (105) injuries (p<0.001).

There were extremely limited data comparing RTP by sport, as articles generally reported aggregated mean time to RTP across all athletes included in the study, regardless of sport. Kramer et al[39] noted in their study that soccer players were less likely to RTP, and those who were able to return did so later than athletes who return to other sports. Buerer et al[21] found no relationship between RTP timeline and sport, yet they did not describe the types of sports included in their study.

Patient-reported outcome measures

For the purposes of evaluating the use of patient-reported outcomes, we analyzed all articles that mentioned an RTP timeline, including the 20 articles considered for this review and the 34 articles that only discussed a postoperative protocol without providing data on return to sport. In these 54 articles, no single patient-reported outcome measurement was used more than 50% of the time. The most often used was the American Orthopedic Foot and Ankle Society Scales (AOFAS) (n=22; 41%), followed by the Karlsson scale (n=11; 20%), the Visual Analogue Scale (VAS) (n=8; 15%), the Foot and Ankle Outcome

Score (n=7; 13%) and the Sefton scale (n=5; 9%). An additional 12 outcomes metrics were used in other papers. Full results are in table 4.

DISCUSSION

In general, the RTP timeline is primarily of concern for elite-level athletes. However, collecting data on all patients participating in sport is necessary due to the dearth of available literature solely on professional athletes. RTP timeline data are necessary and useful for three purposes:
(1) expectations for physicians and patients;
(2) tracking milestone progression towards RTP post-surgery;
(3) linking RTP timeline with eventual outcomes.

Table 4 Patient-reported outcome scales

AOFAS	22	41%
Karlsson	11	20%
VAS	8	15%
FAOS	7	13%
Sefton	5	9%
Tegner	4	7%
JSSF	3	6%
FAAM	2	4%
Good	2	4%
Ahlgren and Larsson	1	2%
CAIS	1	2%
CAIT	1	2%
Elerud and Molander	1	2%
Hamilton	1	2%
Kaikkonen	1	2%
Modified Weber	1	2%
SF-12	1	2%

AOFAS, American Orthopedic Foot and Ankle Society Scales; CAIT, Cumberland Ankle Instability Tool; CAIS, Chronic Ankle Instability Score; FAAM, Foot and Ankle Ability Measure; FAOS, Foot and Ankle Outcome Score; JSSF, Japanese Society for Surgery of the Foot ankle-hindfoot score; SF-12, Short-Form 12 Health Survey; VAS, Visual Analogue Scale for pain.

1. Expectations: The review of current literature has illustrated significant variation in the RTP timeline depending on the injury pattern, surgical technique, sport played and use of external bracing. It is likely that other variations in the RTP timeline exist but have yet to be confirmed through scientific study. Expectations for ability to return to sport, especially when similar pre-existing conditions can be compared with previously established cases, can be of high utility to patients when evaluating their treatment options. The literature is especially weak on this reporting for elite-level athletes. Indeed, in a study on the RTP post acute lateral ankle ligament repair in professional athletes, White et al[30] indicated that there is a lack of available data to guide professional athletes in their recovery timeline.

2. Tracking progression: For those patients who have undergone surgery to repair the lateral ankle ligaments, it would be relevant to know where in their rehabilitation process they are relative to other comparable cases. Clanton et al[41] discussed the need for subjective and objective data in determining ability to RTP. Specifically, they called for assessments in functional testing to include range of motion, balance and proprioception, agility and strength. Pertinent to our subject of interest, the Lower Extremity Functional Scale is an objective score whose use has specifically been validated in athletic subjects with ankle sprains.[42] Rehabilitating postsurgical athletes should be tracked using both subjective and objective assessments to determine the relationship between these assessments and the RTP timeline. As of now, no study reviewed for this article included any measurement of clinical outcome scores at the time of RTP.

3. Linking to outcomes: Early RTP is important to high-level athletes, yet the long-term outcomes of the impact of an earlier or later RTP are not known. In general, there is literature to support the efficacy of repair and the ability of athletes to eventually return to preinjury level of play.[43–45]

In particular, Maffulli et al[44] reported long-term results following a Broström procedure, indicating that 58% of athletes were able to return to their full activity level, while the remaining 42% were still able to be physically active (16% of whom were still able to compete but at a lower level). However, it is not possible to relate these data to when athletes return to play and if that impacted their eventual outcome. Overall, this trend of reporting on ability to RTP but lacking RTP timeline data is also noted by White et al.[30] Generally, there is substantial variability in the measurement of patient-reported outcome measures in the RTP literature. This trend is very similar to

the broader foot and ankle literature, where Hunt and Hurwit[46] found the AOFAS scale was used most frequently (55.9%), followed by the VAS scale (22.9%).

While the AOFAS scale is commonly used, it has not been found to accurately quantify or compare patient outcomes and is not a validated patient outcome measure.[47] In lieu of the AOFAS scale, we suggest a movement towards the use of concise, validated patient-reported outcome measures. The VAS is a widely accepted and validated outcome measure for pain and should continue to be used.[48] In the context of chronic ankle instability, the Foot and Ankle Ability Measure is a validated outcome measure that should be incorporated in the standardization of patient outcome measures,[49] and the PROMIS scales are gaining popularity as an efficient set of outcomes tools in orthopaedics. Having a more consistent use of validated clinical outcome measurements will increase the utility and applicability of data reporting.

The dearth of available data sufficiently describing RTP following orthopaedic surgery applies to other areas as well. For example, in a systematic review of 48 articles describing resumption of sport following ACL repair, only nine studies were found that included an RTP timeline with an average resumption of sport at 7.3 (range 2–24) months.[50] Consistent with our findings, there appears to be a deficiency in consistent reporting of RTP timeline reporting following ACL repair. In addition, while there are no pooled data on RTP following Bankart repair (shoulder stabilization), one paper on 16 athletes reported an average RTP of 4.4 months.[51] It is clear that the paucity of RTP timeline data is not just isolated to ankle ligament reconstruction, but to other common surgically treated sports injuries as well.

LIMITATIONS

This study has several limitations. Lack of consistency in reporting data and outcomes metrics across manuscripts did not allow for complex comparative statistical analysis. None of the papers included in this systematic review reported effect sizes, such as ORs, risk ratios or the associated 95% CIs, which made conducting a meta-analysis unfeasible. Therefore, quantitative analysis was limited to reporting on descriptive statistics. In addition, the overwhelming majority of articles describing outcomes for lateral ligament repair are in the chronic instability population, so we pooled the one paper describing acute repair with the remaining articles that chronic instability. Lastly, the low number of included articles impacts the strength of any findings.

CONCLUSION

The results of this review demonstrate that while 360 manuscripts describe a postoperative clinical outcome of lateral ankle ligament repair, only 20 (5.5%) detail an RTP timeline as a reported outcome metric, indicating a clear deficiency in the literature.

These articles suggest a rate of return to sport of 85% of athletes at an average of 4.7 (+/−1.5) months. In future studies involving athletes, increased attention should be placed on detailing the time until the athletes can RTP and the level of play to which the athletes returned. In addition, these data would optimally be stratified by activity or sport, so that athlete and physician expectations for return to sport can be based on sport-specific data. While some manuscripts broke down their patient populations by sport played, the RTP timeline was reported in an aggregated mean time to RTP across all athletes included in the study, regardless of sport, reducing the utility of their data. In addition, as acute ligament repairs become increasingly common with less invasive techniques, stratifying outcomes by chronicity (acute vs chronic) and by technique has become increasingly important. Moreover, there is currently no well-defined structure and protocol for assessing the readiness of athletes to RTP. We propose that athletes should be returned to play following a scheme similar to what van Eekeren et al[52] have suggested for talar osteochondral lesions, following a four-phase progression of increasing intensity, including walking, jogging, return to non-contact sports and return to contact sports.

Our ability to better describe clinical and return-to-sport outcomes in patients will dramatically improve the science supporting novel advances in ankle ligament repair techniques and rehabilitation.

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Return to Play Following Arthroscopic vs Open Treatment of Lateral Ankle Instability in Recreational/ Athletic Populations: a Systematic Review.

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D'Hooghe P, Axibal D, Fuld R, Sutphin B, Hunt K

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ABSTRACT

Background:
Ankle sprains are very common injuries among athletic populations. Sparse data exists regarding return to play (RTP) following common lateral ligament repairs. Our purpose is to compare RTP timelines and outcomes between open and arthroscopic treatment of lateral ankle instability in athletes.

Methods:
In this systematic review, MEDLINE and EMBASE searches were performed to identify available literature through November 2017 describing open and/or arthroscopic treatment of lateral ankle instability in athletes, their outcomes, and a RTP timeline.

Results:
A total of ten studies met criteria. 174 athletes were treated with open ankle instability procedures (9 studies) and 19 athletes were treated with arthroscopic procedures (1 study). 167/174 patients with open treatment returned to sport (96% RTP rate, weighted mean RTP timeline of 2.85 months). In comparison, all 19 patients in the arthroscopic group returned to sport (100% RTP rate, weighted mean RTP timeline of 3.794 months).

Conclusion:
Very few articles describing outcomes of lateral ligament repair in athletes include return to play metrics. Considering the data available, athletes treated with open ankle ligament repair procedures (nine studies with 167 athletes) returned to play almost 1 month earlier than athletes treated with arthroscopic procedures (1 study with 19 athletes). As timing of return to activities is a valuable metric to compare surgical and rehabilitative techniques, more studies that detail return to sport are needed as part of a description of ankle ligament repairs.

INTRODUCTION

Ankle sprains are very common injuries in the athletic population. At the 2004 Olympic summer games in Greece, ankle sprains accounted for 22% of injuries[1]. In Hootman et al. 16-year study of fifteen sports in all three American collegiate divisions, ankle ligament sprains were the most common injury, accounting for approximately 15% of all injuries[2]. Tenforde et al. study of U.S. cross country

and track and field high-school athletes suggest that nearly one-third of female and one-quarter of male athletes have a history of an ankle sprain[3]. Lievers et al. studied male collegiate American football injuries from the National Collegiate Athletic Association (NCAA) Injury surveillance system during the 2004-2009 seasons. Lateral ankle sprains were the most common foot and ankle injury, accounting for almost half (45%) of all injuries, as well as the greatest total time loss of all injuries (12.726 days)[4].

Acute ankle sprains have been classified based the amount of ligamentous damage:
Grade I entails a stretched anterior talofibular ligament (ATFL) with no laxity on examination.
Grade II consists of a complete tear of the ATFL, with or without partial tearing of the calcaneofibular ligament (CFL). Laxity may be present.
Grade III involves complete disruption of the ATFL and CFL, with or without posterior talofibular ligament (PTFL) or capsular tearing[5].

While Grade I and II ankle sprains can successfully be treated nonoperatively, many grade III injuries require surgical treatment in order to prevent recurrence and facilitate return to full sports participation. Pijnenburg et al. found that when compared to patients treated nonoperatively, fewer patients treated surgically reported residual pain, symptoms of giving way, and recurrent sprains[6] (Figure 1).

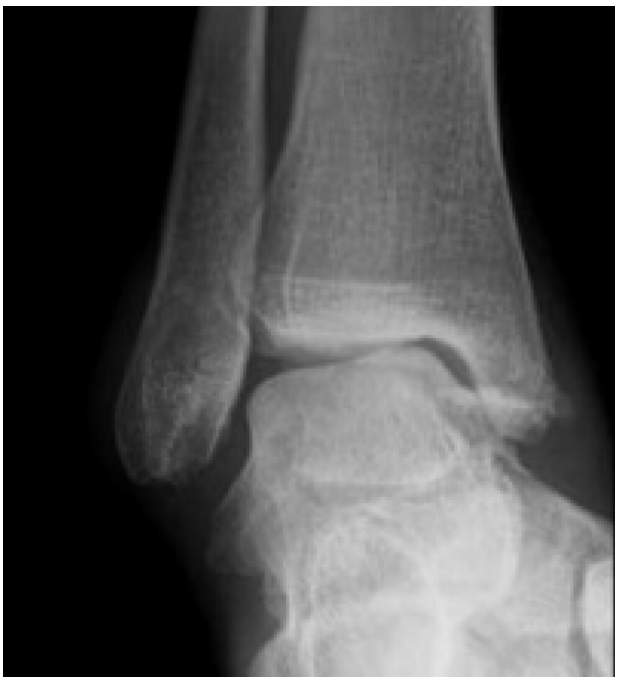

Figure 1. Anteroposterior X-ray of the ankle with significant talar tilt secondary to ankle instability.

Techniques used for lateral ligament repair have evolved over time. The most common surgical procedure to repair the lateral ligaments was described by Brostrom et al. as a mid-substance imbrication and suture of the injured ATFL ends[7]. Gould et al. augmented the Brostrom technique with overlap of the nearby lateral talocalcaneal ligament, and by attaching a mobilized lateral portion of the lateral extensor retinaculum to the fibula (in addition to repair of the ATFL and CFL)[8].

Since then, arthroscopic techniques have been introduced to allow repair of the lateral ankle ligaments. Hawkins et al. described an arthroscopic staple technique: the staple tines gather the damaged ATFL and contiguous capsule. The tissue is then fixed to an abraded area on the vertical surface of the talus, anterior to the fibular tip[9]. Maiotti et al. reported the results of arthroscopic thermal capsular shrinkage in 22 soccer players with ankle instability: 86.3% of patients (n=19) had good or excellent functional outcomes at a mean of 42 months[10]. Lui et al. proposed one of the first arthroscopic assisted lateral ligament reconstructions: An ATFL and CFL reconstruction with a plantaris tendon free-graft via a three-portal approach[11]. The purported advantage of the less invasive arthroscopic repair is faster recovery and earlier return to sport. However, this purported benefit has not yet been well defined.

The purpose of this review is to compare return to play (RTP) timelines and outcomes between open and arthroscopic treatments of lateral ankle instability in athletes.

METHODS

Literature Search

In this systematic review, a literature search was performed for articles on surgical treatment of lateral ligament ankle sprains. Using the search terms ‘ankle ligament surgery’ and ‘ankle sprain instability repair’ in MEDLINE and EMBASE databases, the available literature was obtained up to November 2017. Information obtained included the year of publication, number of athletes, surgical technique, return to play timeline, RTP timeline, RTP performance data, patient reported outcomes measures, and functional outcome measures (Figure 2).

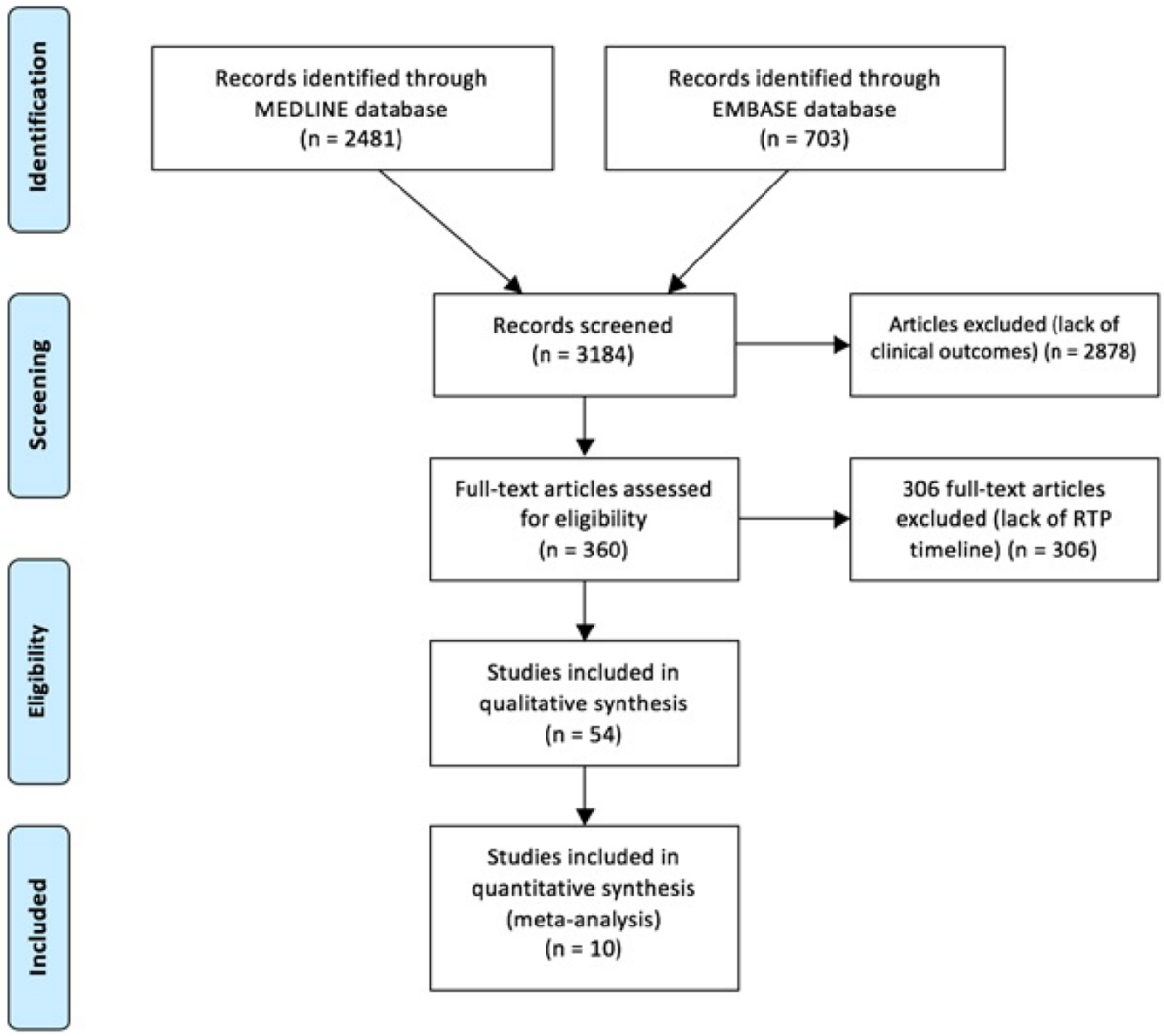

Figure 2. Literature search flow diagram[20].

Study Selection

Studies were independently screened by title and abstract. Initial inclusion criteria included:
(1) Articles available in the English language,
(2) Abstract available,
(3) Reported clinical outcomes (Visual Analog Scores, Japanese Society for Surgery of the Foot, Foot and Ankle Ability Measure, etc.).

Exclusion criteria included basic science articles, anatomic studies, radiographic studies, and review papers. After passing the initial screening, the remaining manuscripts were completely examined to assess for RTP timeline metrics.
To be included in this review, studies needed to contain:

(1) Patients who participate in athletic activities,
(2) Return to play timelines as an outcome metric or result ("patients returned to play at 6 months," not "patients were allowed to return to play at 6 months),
(3) Return-to-play timelines reported in means (not medians).

Statistical Analysis

Using descriptive statistics from the articles included in this review, a weighted mean and weighted standard deviation for time to return to play were calculated. As some studies reported return to play timelines in weeks while others reported in months, a conversion of 4.3 weeks to 1 month was used. Rate of RTP was described by percentage of all athletes who were able to return to play at all. A percentage was also calculated for those who returned to sport at pre-injury level, those who returned to sport below the pre-injury level, and those unable to return to sport.

RESULTS

Literature Search

The EMBASE database search produced 703 independent results, while the MEDLINE database search produced an additional 2481 results. This generated 3184 total results. Articles that did not report clinical outcomes after surgical management of lateral ankle sprains were excluded, leaving 360 eligible articles. Articles that did not report a return to play timeline were also excluded. The remaining 54 papers discussed a RTP timeline in some capacity. Articles that reported return to play as part of a post-operative protocol (per protocol, patients are allowed to return to sports at nine weeks), but did not record actual return to play, were excluded, leaving 20 remaining papers.

Finally, nine studies reported return to play timelines in means, not medians, and were used for inclusion in this review article. This allowed us to calculate a weighted mean. All nine studies included open procedures, while one of those studies also included arthroscopic procedures (Matsui et al. 19 patients). Further details can be found in Table 1[12-20].

Return to Play Metrics

Studies were not uniform in their descriptions of a return to play timeline. Although all included studies reported a mean return to play, three studies also reported a range[12,14,15]. One study reported the specific return to play timeline for each individual athlete[16]. 174 athletes were treated with open ankle instability procedures, but only 167 were able to return to sport (96% return to play rate for open ankle instability procedures). These athletes returned to sport at a weighted mean of 2.85 months (standard deviation-1.89 months). All 19 patients in the arthroscopic group returned to sport, producing a 100% return to play rate. These athletes returned to sport at a weighted mean of 3.794 months[12]. Three studies specified whether patients returned to sport at the athlete's pre-injury level or below the pre-injury level[13,15,19]. There were a total of 71 patients who returned to play in these three studies (Giannini n=31; Paterson n=26; Morelli n=14), with 10 patients returning to lower demand sports (Giannini n=5; Paterson n=3; Morelli n=2).

Author	Year	Procedure	Patients, N (mean age) Activity level/ sport: N	Clinical Outcome (time measured)	Return to play average (range)
Arthroscopic					
Matsui	2016	ATFL repair w/inferior extensor retinaculum reinforcement	19 (28 years)	1. VAS: 12.4 (2 wks) 2. JSSF: 98 (1 year)	3.8 months (2.76–5.06 months)
Open					
Matsui	2016	ATFL repair w/inferior extensor retinaculum reinforcement	18 (24 years)	1. VAS: 19.2 (2 wks) 2. JSSF: 95 (1 year)	3.9 months (2.99–5.06 months)
Giannini	2015	Modified brostrom or anatomic reconstruction with plantaris autograft/ peroneus brevis allograft	31 (25.9 years) Recreational level: 28 Professional: 3	1. AOFAS: 92.2 (5 years)	6 months Return to pre-injury level (n=26) Return to lower-demand sports (n=5)
Ibrahim	2011	Gracilis tendon autograft	14 (25 years) Football: 8 Handball: 3 Basketball: 3	1. AOFAS: 96 (33.5 months) 2. VAS: 6 (33.5 months) 3. Karlssons: 94.7 (33.5 months) 4. Olerud and Molander: 87.5 (33.5 months)	6.8 months (4–11 months)
Morelli	2011	Modified Watson-Jones	14 (22.7 years) Professional: 6	1. Tegner: 5.1 (10.8 years) 2. Good: 1.6 (10.8 years) 3. AOFAS: 92.2 (10.8 years)	6 months (4–8 months) Return to lower-demand sports (n=2) All professionals return to pre-injury level
Jones	2007	Woven polyester tape	4 (42.5 years)	1. Sefton criteria: Grade 1 (24.5 months)	3 months
Coughlin	2004	Direct repair w/gracilis tendon autograft augmentation	28 (31 years) Team sports: 15 Exercise activity: 8 Recreational: 4 No specific sport: 1	1. AOFAS: 98 (23 months) 2. Karlsson score: 95.3 (23 months) 3. VAS: 6 (23 months)	6.5 months
Solakoglu	2003	Colville technique	14 (25 years) Amateur sports: 8 Military: 6	1. Alghren/Larson: 5 (20 months)	6 months
Paterson	2000	Semitendinosis autograft	26	1. ROM (24 months)	2.75 months Return to pre-injury level (n=23) Return to lower-demand sports (n=3)
Hoy	1994	Watson-Jones technique	25	1. Symptoms survey (5 years)	2.29 months 18 returned to sports

Table 1. Summary of articles included in study.

In Paterson et al. study, two patients cited lack of confidence in the ankle while one patient cited persistent pain. Of the available data, this produces an aggregate of 14% of patients treated with open ankle instability procedure return to sport below the preinjury level (of the 71 patients from these three studies).

Patient Reported Outcome Measures

Clinical outcome scores were highly variable among identified articles. Thus, we were unable to make a meaningful comparison of patient reported outcome metrics between arthroscopic and open groups among all the studies. However, Matsui et al. did provide a direct comparison between open and arthroscopic treatment. Matsui found no significant differences between open and arthroscopic groups in Visual Analog Scores (VAS) at two weeks after surgery and Japanese Society for Surgery of the Foot (JSSF) scores 1 year after surgery[12].

No other analysis used the JSSF score. For the studies that did use the VAS outcome, these were measured at a different point in time (not at the two weeks as measured by Matsui). Therefore, we are unable to make a direct comparison between this arthroscopic group and open groups from other papers. Interestingly, the Foot and Ankle Ability Measure (FAAM), which is one of few outcome scores validated for ankle instability, was not used in any of the selected articles that describe return to play following lateral ligament repair[21].

DISCUSSION

The most important finding of this study is the relative paucity of articles that describe return to play following lateral ligament repair. When comparing open repair to arthroscopic repair, 167 athletes treated with open ankle instability procedures returned to play almost 1 month earlier than 19 athletes treated with arthroscopic procedures (open: 2.9 months vs. arthroscopic: 3.8 months). The variability in the data prevented us from performing a meaningful statistical comparative test.

Considering all athletes in this review, the overall return to play rate was 96% for open ankle instability procedures (167/174) and 100% for arthroscopic repairs (19/19). These rates are similar to other studies in the literature (although not included in this analysis because they did not meet criteria). Nery et al. reported on the outcomes of 38 patients treated with combined open and arthroscopic ("arthroscopic-assisted") anatomic reconstruction of the lateral ligament complex.
With an average follow-up of 9.8 years, 96% (29/30) of the active patients were able to return to sport[22]. Our analysis found that only 14% of athletes treated with an open ankle instability procedure returned to sport below the pre-injury level (10/174). Similarly, Nery et al. found that 10% (3/30) returned to sport at a lower level. Our evaluation found that 4% of athletes treated with an open

ankle instability procedure do not return to sport at all (7/174). Nery found comparable rates of 3.3% (1/30).

This review of the literature identified nine eligible studies reporting on 167 athletes treated with open procedures, and only one eligible study reporting on 19 athletes treated with arthroscopic procedures.
Although open procedures tend to be the gold standard for the treatment of lateral ankle instability, arthroscopy can serve as both a diagnostic and therapeutic tool. The advent of arthroscopy has expanded our knowledge of the magnitude of intra-articular pathology associated with ankle instability[23].

Furthermore, arthroscopy has been used for the surgical treatment of lateral ankle instability, as well.
Hawkins first described his arthroscopic technique using a staple for plication of the ATFL[9]. Kashuk et al. described his arthroscopic technique of repairing the lateral ligamentous complex with suture anchors[24]. Maiotti et al. proposed the use of arthroscopic thermal capsular shrinkage to treat ankle instability[10]. Lui et al. detailed a three-portal approach for reconstruction of the ATFL and CFL using a plantaris tendon free graft[11].

We identified substantial variability on the patient reported outcome metrics used. In fact, nine different outcome metrics were used in 9 studies.
In addition, the timing of reporting of these measures was highly variable, making it difficult to make a direct comparison. Although we only had one eligible study reporting on return to play outcomes following arthroscopic management, others have presented the outcomes in the arthroscopic management of this injury.
Corte-Real et al. conveyed their results in 28 patients treated with an arthroscopic-assisted technique: with an average follow-up of 24.5 months, the mean AOFAS score was 85.3 and mean satisfaction was 3.8 (out of 5)[25].
Although we had only one eligible study comparing open and arthroscopic outcomes of lateral ankle instability, the comparison of open versus arthroscopic techniques is prevalent in many other foot and ankle pathologies, as well. Yeap et al. compared the outcomes of calcaneal fractures after open reduction internal fixation versus arthroscopic-assisted percutaneous screw fixation. Although Bohler's angle, Gissane's angle, and AOFAS and SF 36 scores were not significantly different, the arthroscopic-assisted group was able to have surgery earlier, go home faster, and return to work earlier[26].

Many of the weakness of this review stem off the fact that there was only one eligible study for the arthroscopic group. As there was only one study for the arthroscopic group, a standard deviation could not be calculated, and consequently, a t-test to compare the two groups could not be estimated. Furthermore, our sample size of ten total studies (nine open and one arthroscopic) does not meet the criteria for the assumption of normally distributed data, which is also needed for a t-test.

Additionally, a Wilcoxon-Mann-Whitney test for non-parametric data could not be conducted because the underlying assumptions required for this test were not met. Also, the indications for surgery were left to the discretion of the treating provider and often times not reported in the manuscript.
Likewise, the nonsurgical interventions were consistently not detailed. In analyzing the use of clinical outcome measures in our nine studies, no single outcome measurement was used more than 50% of the time. The most often used scores were the AOFAS (n=4), VAS (n=3), and the Karlssons score (n=2). An additional 6 outcomes metrics were reported by the other papers, each used once only by that specific paper[27] (Table 2).

Clinical Outcomes Scores	Frequency
American orthopaedic foot and ankle score	4
Visual analogue scale	3
Karlssons	2
Japanese society for surgery of the foot score	1
Olerud and Molander	1
Tegner	1
Good	1
Sefton	1
Algren/Larson	1

Table 2. Frequency of clinical outcome scores[27]

CONCLUSION

Although the outcomes of open procedures in the management of lateral ankle sprains in athletes are well reported in the literature, the techniques and outcomes of arthroscopic treatment in athletes are sparse. We found that athletes treated with open ankle instability procedures (nine studies with 167 athletes) returned to play almost 1 month earlier than athletes treated with arthroscopic procedures (1 study with 19 athletes).
However, additional prospective studies are needed to document the outcomes and return to play for athletes treated with arthroscopic ankle instability management.

Ideally, these studies would include the following components:
1) consistent and validated patient reported outcome metrics,
2) consistent description of return to play criteria and timing. This would allow meaningful comparison of surgical techniques as they evolve.

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