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REVIEW ARTICLE

# Patterns of ankle injury in soccer: MRI clues to traumatic mechanism

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## Abstract

Understanding the traumatic mechanisms of ankle injuries in soccer is crucial for an accurate and complete MRI diagnosis. Many ankle injuries share universal mechanisms seen in other athletic activities, but certain patterns are found to be more specific and relatively unique to soccer. Ankle impingement syndromes encountered in soccer encompass a spectrum of disorders that include anterior and posterior impingement categories, with anterior impingement representing pathology relatively specific to soccer. Lateral ligamentous sprains are one of the most common injuries; however, there is a higher rate of injuries to the medial structures in soccer as compared to other sports. Ankle fractures are uncommon in soccer while bone contusions and chondral and osteochondral injuries frequently accompany ligamentous sprains. Tendon abnormalities in soccer most commonly result from overuse injuries and typically affect peroneal tendons, posterior and anterior tibialis tendons, and Achilles tendon. Acute Achilles tendon ruptures occur in both recreational players and elite soccer athletes. Tibialis anterior friction syndrome may mimic tibial stress fractures. Long-term sequelae of acute traumatic and chronic overuse ankle injuries in professional soccer players manifest as ankle osteoarthritis that is more prevalent compared to not only the general population, but also to former elite athletes from other sports. This article examines the most common and specific injuries in soccer in order of their frequency.

**Keywords** Ankle injury · Soccer · Football · Ankle impingement syndromes · MRI

**Key points**
- Lateral ligamentous sprains are one of the most common ankle injuries in soccer. The medial structures are injured at a higher rate compared to other sports.
- Anterior ankle impingement syndrome results from direct microtrauma to the anteromedial aspect of the ankle related to recurrent ball impact.
- Anterolateral impingement syndrome is produced by instability and morphologic changes in the ligaments following lateral ligamentous sprain.
- Tendon abnormalities in soccer most commonly result from overuse and affect peroneal, posterior and anterior tibialis tendons, and Achilles tendon.

## Introduction

The ankle is one of the most frequently injured joints in soccer, competing for highest incidence with the injuries of the knee. Depending on the studied population and analysis of preseason training versus match injuries, ankle trauma is reported to account for 13–36% of all soccer-related injuries [1–5]. The spectrum of ankle injuries includes high impact contact trauma, non-contact acute trauma, chronic repetitive microtrauma, and overuse injuries [6]. While many ankle injuries share universal mechanisms seen in other athletic activities, certain patterns are found to be more specific and relatively unique to soccer. The long-term clinical significance of ankle injuries in soccer can be inferred from the data showing that the prevalence of ankle osteoarthritis in professional soccer players is greater than in not only the general population, but also in former elite athletes from other sports [7]. This article examines the most common and specific injuries in soccer in order of their frequency. We will review ligamentous sprains, ankle impingement, osteochondral injuries, and tendon injuries in context of traumatic mechanisms encountered in soccer.

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## Ligamentous injuries

Ankle ligamentous sprains are very common in soccer accounting for 72–98% of all soccer-related ankle injuries [1, 2]. Video analysis of match injuries in professional soccer players reveals two main patterns of injuries: (1) indirect contact injuries after a collision with an opponent with direct impact on the medial side of the leg, causing the player to land with the ankle in an inverted position and (2) contact injuries from direct forces against the ankle [8, 9]. A higher incidence of ankle sprains is seen in the dominant leg, probably due to frequent forced inversion in jumping and kicking while being tackled by an opponent. Ankle sprains are also significantly more common in players who sustained sprains in the past [2]. As in the general athlete population, the ankle is most susceptible to inversion injury and, as a result, damage to the *lateral ligamentous complex* [2]. However, Morgan et al. emphasized that the demands of soccer also place the *medial foot and ankle* at greater risk than those of any other sport. Passing the ball is most often achieved by striking the ball with the medial aspect of the foot which puts the ankle in an abducted and externally rotated position and subjects medial structures to stress [4] (Fig. 1).

When analyzing the lateral ligamentous complex on MRI, it is helpful to keep in mind that most injuries occur in inversion and plantarflexion. Both of these movements are stabilized by the anterior talofibular ligament (ATFL) which makes it most vulnerable to injury [10–12]. Extreme inversion leads to combined tears of the ATFL and calcaneofibular ligament (CFL) and in the most severe cases, is followed by the disruption of the posterior talofibular ligament (PTFL) (Fig. 2). In authors' experience, ATFL tears in soccer players are frequently associated with injuries of the inferior extensor retinaculum.

The CFL serves as a stabilizer to both the ankle joint and the subtalar joint, resisting ankle inversion in the dorsiflexed position and subtalar joint inversion [11]. Indirect in vivo evaluation of the length of the ligament through the arc of motion suggests that the CFL may also be susceptible to injury in dorsiflexion and pronation [12]. Although uncommon, isolated injuries of the CFL sparing the ATFL can be encountered in ankle dorsiflexion sprains as well as in subtalar sprain [13–15] (Fig. 3).

Acute strains of the extensor digitorum brevis muscle commonly occur in soccer players. These injuries may mimic lateral ligamentous sprain on clinical exam.

Tears of the deltoid ligament are often associated with tears of the lateral collateral ligament complex, tibiofibular syndesmosis, and flexor retinaculum [16, 17] (Fig. 4). Inversion injuries often produce contusions of the deltoid ligament which manifest as loss of normal striations of the posterior tibiotalar component of the deep deltoid ligament [18]. Crim et al. reported that MRI findings of focal detachment of the superficial deltoid origin or detachment of the fascial sleeve of the medial malleolus yielded a high sensitivity and specificity for superficial deltoid ligament tears [17] (Fig. 2). In adolescent soccer players, chronic repetitive injury to the deltoid ligament complex may lead to delayed fusion of the medial malleolar apophysis manifesting as fragmentation of medial malleolus on MRI [19].

*Syndesmotic sprains* constitute the minority of ankle sprains but have a prolonged recovery period [20]. Some authors speculate that a lower reported rate of syndesmotic sprains may be a result of true lower rate of occurrence, as well as of underdiagnosis due to similarities in clinical presentation of syndesmotic injury and inversion sprain [4, 20, 21]. Regarding the mechanism of injury, syndesmotic sprains are thought to result from either external rotation or a hyperdorsiflexion of the ankle and foot [20, 21]. Due to shared external rotation and eversion mechanism of injury, syndesmotic sprains are often accompanied by deltoid ligament injury [21–23]. On MRI, evaluation of syndesmotic complex includes analysis of anterior and posterior tibiofibular ligaments, inferior transverse ligament, and interosseous ligament, a distal thickening of

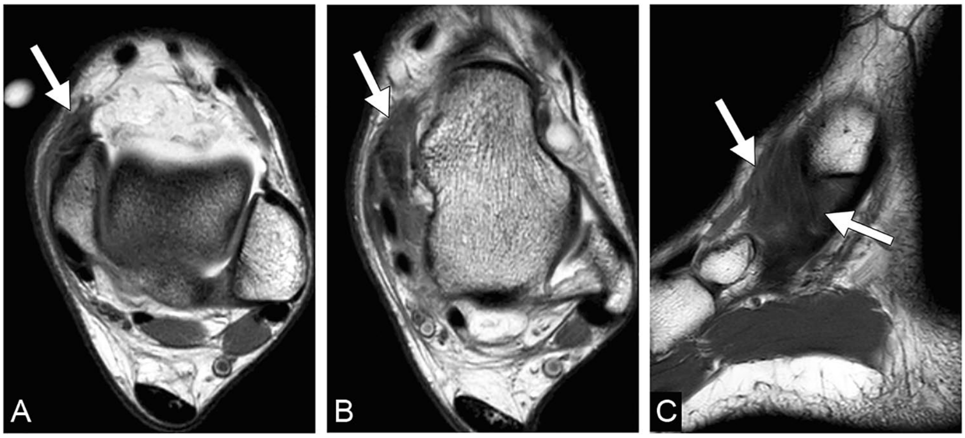

**Fig. 1** Chronic medial ankle sprain in a 22-year-old man with anterior medial ankle pain 1 month after a soccer injury. Sequential axial proton-density-weighted (**A**, **B**) and sagittal T1-weighted (**C**) MR images show a marked thickening of the superficial deltoid ligament (arrows)

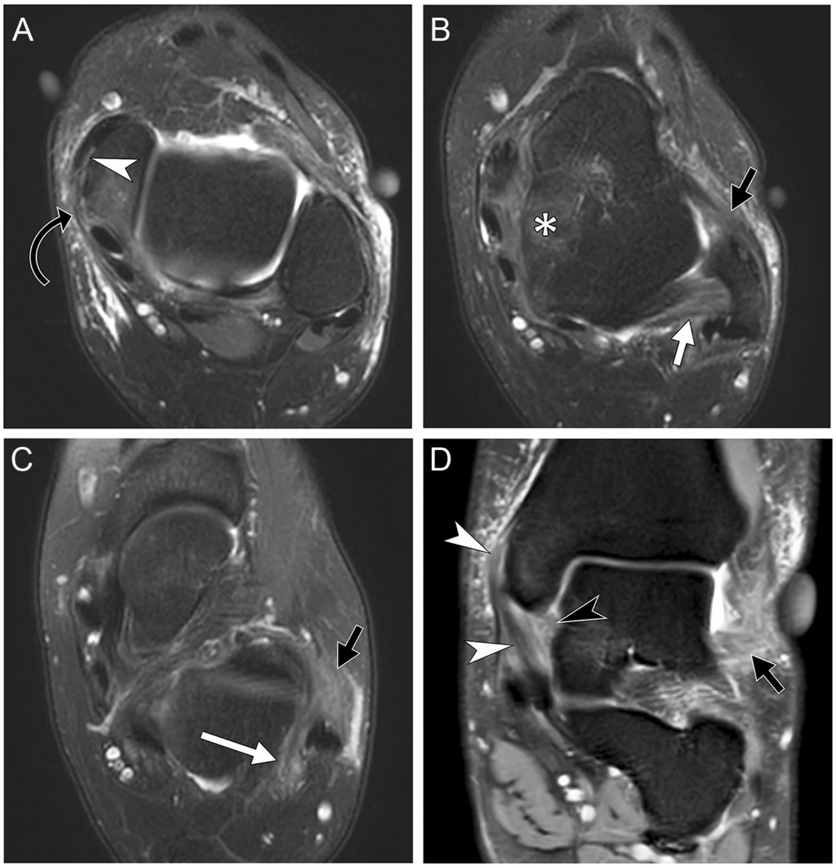

**Fig. 2** Ankle sprain in a 24-year-old woman. Axial fat-suppressed T2-weighted (**A**–**C**) and coronal proton-density-weighted (**D**) MR images demonstrate stripping of the tibial attachment of the superficial layer of deltoid ligament complex (arrowhead, **A**), contiguous with detachment of the periosteum and flexor retinaculum (curved arrow), bone contusion at the medial aspect of the talar body (asterisk), sprains of the ATFL (black arrow), PTFL (short white arrow), CFL (long white arrow), and superficial (white arrowheads, **D**) and deep (black arrowhead) layers of the deltoid

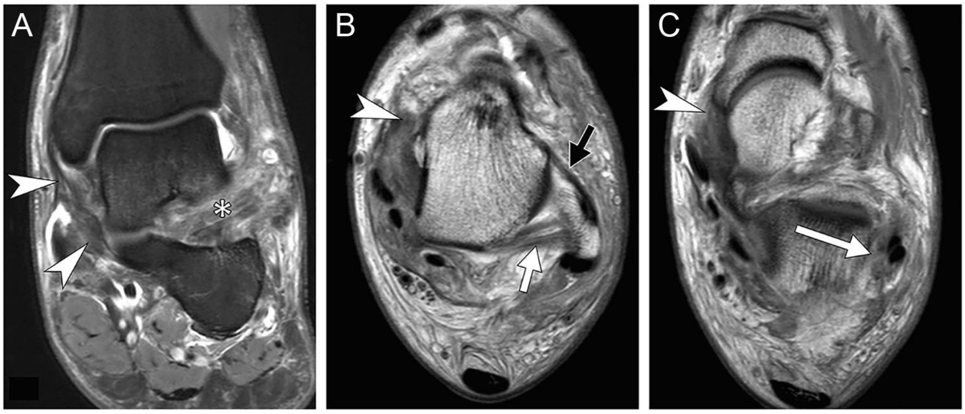

**Fig. 3** Subtalar dislocation in a 33-year-old man sustained after landing on an uneven surface while playing barefoot soccer. Coronal fat-suppressed (**A**), axial (**B**, **C**) proton-density-weighted MR images show sprain of the tibiocalcaneal (arrowheads, **A**) and tibionavicular (arrowheads, **B**) components of the superficial deltoid complex, sprain of the superomedial calcaneonavicular ligament of the spring ligament complex (arrowhead, **C**), disruption of the sinus tarsi ligaments (asterisk), intact ATFL (black arrow, **B**) and PTFL (white arrow, **B**), and full thickness tear of the CFL (white arrow, **C**)

the interosseous membrane. Additional structures include commonly present and variable in appearance posterior intermalleolar ligament, sometimes referred to as tibial slip of the posterior talofibular ligament [24–26]. Some authors distinguish an accessory anteriorinferior tibiofibular ligament, also referred to as “Bassett’s ligament” [27, 28]. Conventional radiographs demonstrate moderate accuracy, high specificity, and low sensitivity for syndesmotic injury and instability. MRI has sensitivity and specificity of nearly 100% for detection of syndesmotic injuries; however, its utility in diagnosis of subtle syndesmotic instability needs further investigation [29–32]. Syndesmotic

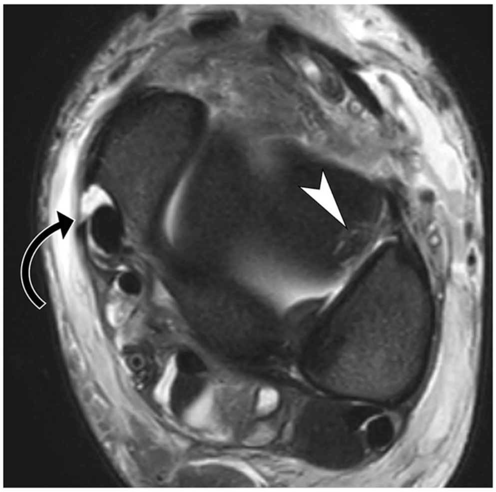

**Fig. 4** Multiligamentous injury in a 15-year-old boy sustained while playing soccer. In addition to tears of the ATFL, CFL, deep, and superficial deltoid (not shown), an axial fat-suppressed T2-weighted MR image of the ankle depicts tear of the flexor retinaculum (curved arrow) with subluxation of the posterior tibialis tendon, as well as osteochondral fracture of the lateral talar dome (arrowhead)

**Table 1** Mechanisms of ankle ligamentous injuries in soccer

| Ankle ligamentous complexes | Mechanisms of injury in soccer |
|---|---|
| Lateral | • Inversion and plantarflexion of the ankle, increasing in severity with sequential injuries to ATFL, CFL, and PTFL<br>• Ankle dorsiflexion or subtalar joint injury producing isolated CFL injury (uncommon) |
| Medial | • Abduction and external rotation, with medial complex injured in isolation or in association with syndesmotic injuries<br>• Inversion and plantarflexion, in association with lateral ligamentous injuries |
| Syndesmosis | • External rotation<br>• Hyperdorsiflexion |

*ATFL* anterior talofibular ligament, *CFL* calcaneofibular ligament, *PTFL* posterior talofibular ligament

interval can be assessed in detail by CT. Some studies evaluating the benefits of axial loading or weight bearing CT (WBCT) in diagnosis of syndesmotic instability demonstrated conflicting results [33, 34]. Recent investigations of CT assessment of instability include evaluation of various methods of measurements of the syndesmotic region including distance, area, and volume on WBCT as well as CT with stress maneuvers [35–37].

Mechanisms of ankle ligamentous injuries in soccer are summarized in Table 1.

## Impingement

Ankle impingement syndromes are defined by a painful mechanical limitation of ankle motion when performing specific movements about the joint [5, 38–41].

*Anterior impingement syndrome*, the prototypical impingement in soccer, was first described by Morris in 1943 and later investigated and named “footballer’s ankle” [42, 43]. This condition manifests with anterior ankle joint pain and limited dorsiflexion that results from entrapment of hypertrophied synovial tissue or scar tissue between the distal tibia and talus and is exacerbated by the presence of abnormal osseous proliferation along the anterior tibiotalar joint line.

Initially, it was believed that osseous overgrowth represents spurs from capsular traction produced by the hyperplanarflexion of the foot in the repetitive kicking action [44]. However, several subsequent investigations, including arthroscopic evaluation, cadaveric studies, and biomechanical analysis evaluating impact location and force of kicking the soccer ball challenged this concept and provided evidence for an alternative mechanism [45–47] (Fig. 5). The currently accepted theory is that it is the direct microtrauma to the anteromedial aspect of the ankle related to recurrent ball impact that leads to damage of the articular cartilage and osteophyte formation in anterior ankle impingement syndrome. Subsequently, repeated forced dorsiflexion causes osseous abutment between the talus and tibia, which leads to microfractures and soft tissue entrapment that perpetuate inflammation and formation of the osteophytes, intraarticular bodies, and fibrous bands along the anterior joint line [47, 48]. With respect to the nature and location of talar bony osseous outgrowths, Hayeri et al. reported that bone development occurs in an intraarticular location and represents osteophytes on the medial part of the anterior talus, while laterally the outgrowths develop extraarticularly and may represent enthesophytes [49].

In imaging evaluation of anterior impingement, radiographs and CT are essential in the localization of osseous outgrows and assisting preoperative planning (Fig. 6). MRI provides additional information about the mechanics of chronic ankle impingement rather than an accurate diagnosis of this clinical entity [50] (Fig. 7). Although conventional MRI showed only fair sensitivity and specificity for anterior tibial osteophytes, it had a high sensitivity for synovitis, capsular thickening, intraarticular bodies, and granulation tissue, and also proved useful in detecting extraarticular soft tissue pathology which may eliminate the need for arthroscopy [39, 50]. Newer MR imaging techniques, such as zero

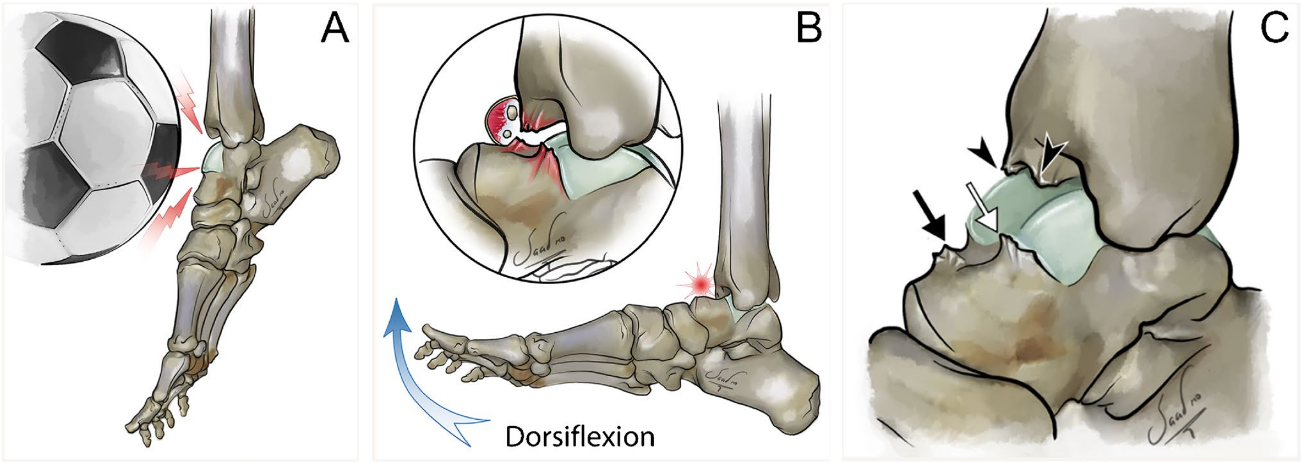

**Fig. 5** Pathogenesis of anterior ankle impingement syndrome in soccer. **A** Direct microtrauma from recurrent ball impact on the anteromedial aspect of the ankle leads to articular cartilage damage and osteophyte formation. **B** Subsequent repeated dorsiflexion causes osseous abutment between the talus and tibia, which leads to microfractures and soft tissue entrapment that perpetuate synovial inflammation and hypertrophy, formation of the osteophytes, and intraarticular bodies along the anterior joint line. **C** Osseous outgrowths occur at various locations including osteophytes along the anterior lip of the tibia (arrowheads) and the medial aspect of the anterior talus (white arrow), while laterally the talar outgrowths may represent enthesophytes (black arrow)

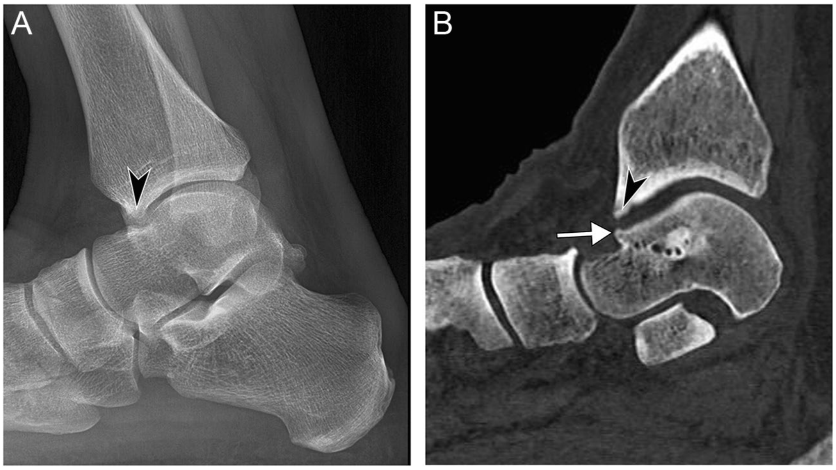

**Fig. 6** Anterior ankle impingement in a 24-year-old college soccer player. Lateral radiograph of the ankle in simulated dorsiflexion (**A**) and sagittal reformatted CT image (**B**) demonstrate osteophytes at the anterior lip of the tibia (arrowhead) abutting osteophytes at the anterior medial aspect of the talus (white arrow)

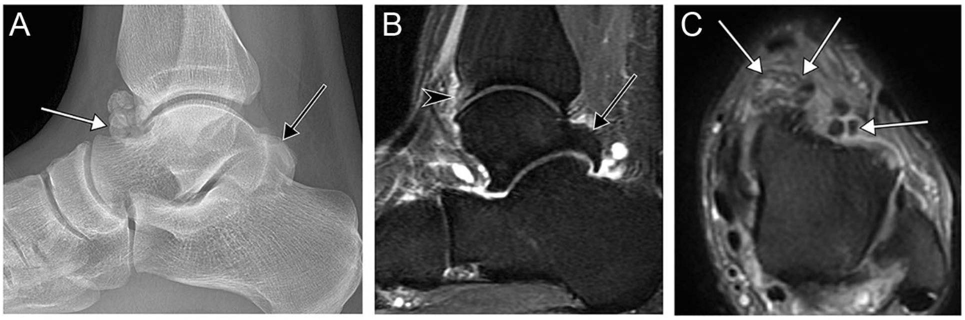

**Fig. 7** Anterior ankle impingement syndrome in a 26-year-old man professional soccer player with anterior pain and limited dorsiflexion of the ankle. Lateral radiograph (**A**) and sagittal STIR (**B**) and axial fat-suppressed T2-weighted (**C**) MR images of the ankle demonstrate intraarticular bodies (white arrows), osteophytes, and marrow edema at the anterior lip of the tibia (arrowhead). Note the elongated lateral tubercle of the posterior talar process (black arrow) and posterior subtalar effusion; although these features may be associated with posterior impingement, the patient did not demonstrate clinical symptoms of PAIS

echo time (ZTE) imaging or a fast field echo resembling a CT using restricted echo-spacing (FRACTURE), produce CT-like images that depict cortical and trabecular bone and mineralized structures [51, 52].

*Anterolateral impingement syndrome*, described by Ferkel et al., can develop after injury to anterior tibiofibular or anterior talofibular ligaments [53]. Ligamentous disruption and edema and associated instability or microinstability leads to capsular thickening, synovitis, scarring, and occasional formation of a hyalinized mass in the anterior lateral gutter referred to as a "meniscoid lesion" [54, 55]. MRI and MRI arthrography have shown high sensitivity and moderate to high specificity and accuracy in diagnosis of anterolateral impingement demonstrating injury to the ligaments and abnormal contour of the anterolateral gutter [56, 57] (Fig. 8).

*Anteromedial impingement syndrome* is rare ankle impingement syndrome that can result from deltoid ligament complex disruption following an inversion injury, eversion injuries, or following medial malleolar or talar fractures [5, 58].

*Combined* *anterior ankle impingement syndrome* (AAIS) encompasses anterior, anterolateral, and anteromedial ankle impingement syndromes. A recent prospective cohort study of 6754 male professional soccer players showed that AAIS is less frequent, but associated with a longer absence and higher re-injury rate as compared to posterior ankle impingement syndrome [59].

*Posterior ankle impingement syndrome* (PAIS) is manifested by pain with plantar flexion resulting from compression of bone and soft tissues at the posterior ankle. In soccer, PAIS is caused by microtrauma from repetitive plantar flexion, or less commonly, by severe inversion injury that involves the posterior ligaments or a direct trauma of the posterior area from a soccer shoe. Several anatomic variants predispose individuals to posterior impingement [60]. These include prominent os trigonum, an elongated lateral tubercle of the posterior talar process (Stieda process) and posterior intermalleolar ligament. Pathologic conditions such as fractures of the posterior talar process, an avulsion fracture of the posterior ligament complex, instability and pseudoarthrosis of the os trigonum, as well as tenosynovitis of the flexor hallucis longus (FHL) may lead to or exacerbate posterior impingement. Baillie et al. reported that a high prevalence of PAIS-associated MRI morphologic osseous findings, ankle and subtalar joint effusion and synovitis, FHL tenosynovitis, and posterior ankle bone marrow edema was found in dancers and athletes including soccer players. Investigators also reported a lack of association between imaging findings and clinical features of PAIS, emphasizing the need for clinical correlation in PAIS diagnosis [61, 62].

## Osseous and osteochondral injuries

*Fractures* of the ankle are uncommon among soccer-related injuries, representing less than 3% of all ankle injuries and 9% of all soccer related fractures [63, 64]. Most fractures result from a single traumatic event. Because of the low incidence, there is paucity of data about the mechanisms of injury, morphology, and classification of ankle fractures specific to soccer. The mechanism of injury is similar to other sport-related fractures and contusions, commonly resulting from direct trauma during contact with another player. The most common mechanisms resulting in sport-related ankle fractures are supination and external rotation (SER) following the general pattern of Lauge-Hansen SER ankle injuries. Bearing in mind a higher prevalence of medial sided ankle ligamentous injuries in soccer which occur in abducted and externally rotated position, less common Lauge-Hansen injury types must be considered when evaluating MRI. Such patterns include pronation-external rotation (PER) and pronation-abduction (PAB) (Fig. 9).

*Bone contusions* at the medial joint line tend to be associated with more severe lateral ligamentous injuries [65, 66] (Fig. 1). Furthermore, bone contusions and impaction fractures of the medial plantar aspect of the talar head are associated with injury of the transverse tarsal joint

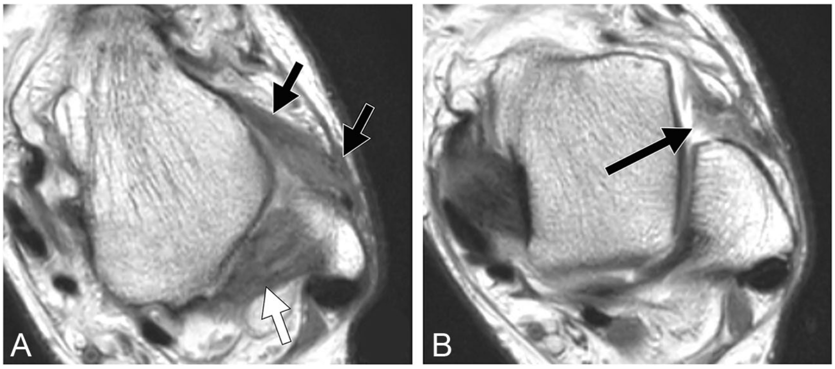

**Fig. 8** Anterior lateral impingement and instability in a 42-year-old man 4 months after sustaining a severe lateral ligamentous sprain. Axial proton-density-weighted MR images (**A**, **B**) show thickened ATFL (black arrows, **A**), PTFL (white arrow, **A**), and hypertrophied synovium in the anterolateral recess resulting in formation of a soft tissue mass with meniscoid configuration (arrow, **B**)

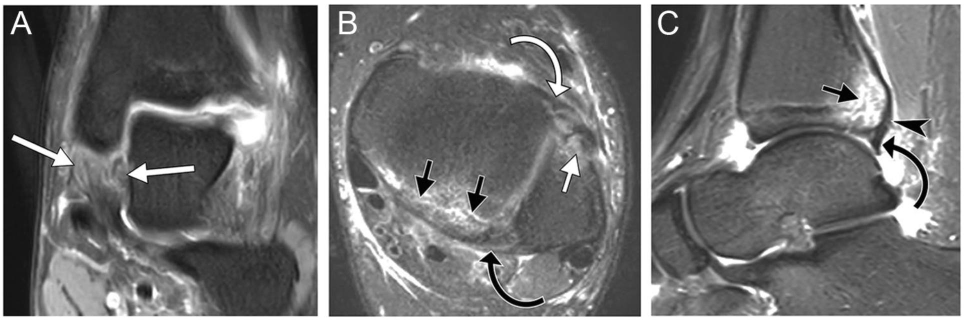

**Fig. 9** Stage II Lauge-Hansen pronation-abduction injury in a 23-year-old male soccer player. Coronal fat-suppressed proton-density-weighted (**A**), axial T2-weighted (**B**), and sagittal STIR (**C**) MR images of the ankle demonstrate tear of the deep and superficial deltoid ligament (white arrows, **A**), avulsion fracture of the distal fibula (straight white arrow, **B**) at the attachment of the anterior tibiofibular ligament (curved white arrow, **B**), fracture of the posterior malleolus (straight black arrows, **B** and **C**) at the attachment of the posterior tibiofibular ligament (arrowhead, **C**), and inferior transverse ligament (curved black arrow, **B** and **C**). Radiographs of the ankle and tibia-fibula demonstrated no fibular shaft fractures

complex [67, 68]. Although bone contusions resolve with time, the acuity of contusions cannot be established on MRI, as time to resolution is highly variable, with reported range from 2 to 16 months [69]. Moreover, bone marrow edema-like signal on MRI has been reported in asymptomatic athletes, further complicating assessment [70].

*Osteochondral and chondral injuries* commonly accompany both ankle fractures and ligamentous sprains. Early studies showed that up to 95% of severe ankle sprains have concomitant chondral injuries on arthroscopy [71]. Most commonly affected are the centromedial and centrolateral regions of the talar dome. Lateral lesions are typically shallow and wafer shaped, indicating a shear type injury (Fig. 10). In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of impaction [72]. Because of their shape, location, and trauma mechanism, lateral lesions are more often displaced than medial lesions [73].

## Ankle osteoarthritis

Professional soccer players suffer from ankle osteoarthritis at a higher rate compared to not only the general population, but also other athletes. Its reported prevalence in former professional soccer players ranges from 9 to 17% [7]. The pathogenesis of cartilage loss in ankle osteoarthritis is multifactorial and encompasses both acute trauma and chronic overuse. Acute trauma to the cartilage may accompany both fractures and ligamentous sprains. Residual ligamentous laxity after ankle sprains can lead to functional ankle instability and with instability, a pathologic amount of talus translation and rotation within the mortise, as well as varus, or less commonly, valgus malalignment, create shear and compression forces on the articular cartilage that lead to accelerated osteoarthritis. Remarkably, single severe ankle sprains carry a higher risk for development of posttraumatic osteoarthritis and shorter latency time between the initial injury and

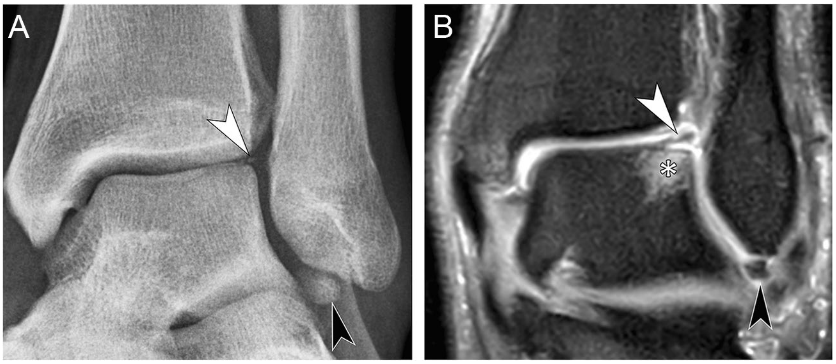

**Fig. 10** Acute osteochondral fracture of the lateral talar dome in a 22-year-old man with acute on chronic lateral ligamentous sprain. The mortise view radiograph (**A**) and coronal fat-suppressed proton-density-weighted MR image (**B**) of the ankle demonstrate an elevated shallow osteochondral fragment composed of articular cartilage and subchondral bone plate (white arrowhead). Note the small ossicle adjacent to the lateral malleolus (black arrowhead) indicative of an old lateral ligamentous injury

symptomatic osteoarthritis as compared to chronic recurrent ligamentous sprains [74]. An analysis of distribution pattern of subchondral bone density in asymptomatic soccer players demonstrated areas of higher stress concentration in the anterolateral tibia, anteromedial and anterolateral talus, and fibula as compared to controls [75]. This data correlates with biomechanical analysis of impact location in soccer [45].

## Tendon injuries

In soccer athletes, most pathologic conditions of the tendons are caused by overuse rather than acute injury. While the balance between pronation and supination is mandatory for normal gait, it is critical for ball control in soccer, particularly in ball reception or dribbling. The key muscles controlling pronation-supination are the posterior and anterior tibial muscles and peroneal muscles, and their tendons are often subjected to overuse injuries [76]. Concomitant instability creates additional strain on the tendons. For example, hindfoot varus and ankle instability are associated with overuse injuries of the peroneal tendons [76].

The classic mechanisms of overuse injury to the *peroneal tendons* are fully applicable in soccer. The most common abnormalities include longitudinal split tear of the peroneus brevis (PB) tendon and peroneal tenosynovitis (Fig. 11). According to the seminal work by Sobel et al., PB split tear represents attrition due to a combination of dynamic mechanical stress and anatomic factors related to (1) the position of the PB tendon, (2) the laxity of the superior peroneal retinaculum, (3) shallow morphology of the fibular groove, (4) an anomalous low-lying PB muscle belly, or (5) accessory peroneus quartus muscle [77]. Chronic PB tendon tear and mucoid degeneration may result in formation of intratendinous ganglion cysts [78, 79] (Fig. 12). The peroneus longus (PL) tendon is most frequently torn at the cuboid tunnel where it is subjected to high shear stresses because of acute tendon turn [80].

*Achilles tendon disorders* result in the longest absences from playing in elite-level soccer [81]. The spectrum of pathology includes both chronic tendinopathy and acute ruptures. Full thickness Achilles tendon ruptures occur in middle age recreational players as well as in elite soccer athletes. While several mechanisms of injury have been reported, performing a “stepping back” movement was found to be the most frequent injury pattern leading to an Achilles tendon rupture in professional male football players. The move is commonly performed to stop a backward movement and to initiate change of direction, and it is characterized by a sudden dorsiflexion of the plantarflexed foot and knee extension. This maneuver applies a high force to the Achilles tendon over a short period of time and may result in acute Achilles tendon rupture [82]. As in general population, the most common site of Achilles tendon tear is located at the relatively avascular “critical zone” region, located 2–6 cm proximal to the calcaneal insertion. Achilles tear may be accompanied by bone contusions along the anterior rim of the tibia, related to a sudden dorsiflexion of the ankle exacerbated by abrupt Achilles tendon failure (Fig. 13).

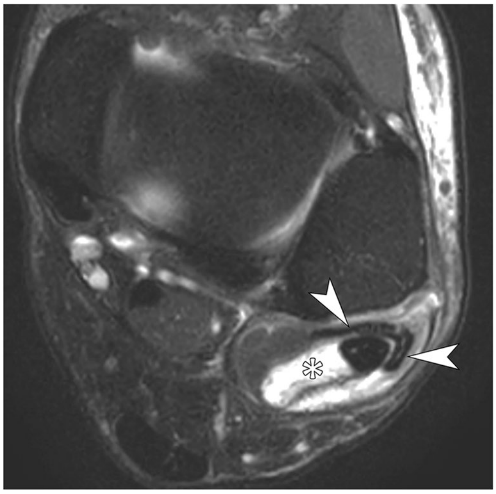

**Fig. 11** Chronic injury to the peroneal tendons in a 36-year-old man presenting with chronic pain and swelling over the lateral malleolus. Axial fat-suppressed T2-weighted MR image of the ankle demonstrates a longitudinal split tear of the peroneus brevis tendon, separating it into two “hemitendons” located both medial and abnormally lateral to the peroneus longus tendon (arrowheads). Note the prominent peroneal tenosynovitis (asterisk)

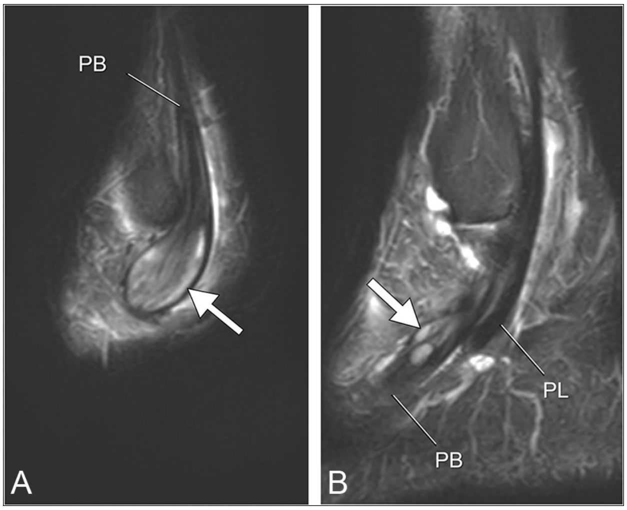

**Fig. 12** Mucoid degeneration with intratendionus ganglion cyst formation in a 46-year-old man. Sagittal STIR MR images (**A**, **B**, lateral to medial) demonstrate mucoid changes within the peroneus brevis tendon (PB) with “shallot-like” enlargement of the tendon (arrow, **A**) proximally to the inferior peroneal retinaculum and smaller intratendious ganglion cysts distally to the retinaculum (arrow, **B**). PL, peroneus longus tendon

Fig. 13 Full thickness Achilles tendon rupture in a 23-year-old man sustained during a soccer match. Sagittal STIR MR image depicts a torn Achilles tendon with a large gap (arrows). Note the bone contusion at the anterior lip of the tibia (arrowhead) suggestive of a dorsiflexion mechanism of injury

*Anterior tibialis tendon* in a soccer player may be subjected to both direct trauma and overuse. Kho et al. described a constellation of MRI findings in soccer players and runners who present with suspected tibial stress fractures [83]. These abnormalities, termed *tibialis anterior friction syndrome*, include peritendinous fluid around an intact tibialis anterior tendon at the level of the mid-to-distal third of the tibia above the level of superior extensor retinaculum, with surrounding subcutaneous, periosteal, and tibialis anterior myotendinous junction edema. The proposed etiology of this entity is friction of the tibialis anterior tendon between the superior extensor retinaculum and the anterior tibia that occurs with repetitive dorsiflexion and plantar flexion.

## Special considerations: soccer refereeing and beach soccer

Functional demands on the ankle of a soccer referee are different from those of a player. The specific ankle injuries in soccer refereeing are due to the high amount of backward running. During a game, a referee typically runs backwards for about 2 km, and this puts a high load on Achilles tendons [84]. MRI depicts the usual range of pathologies of the tendon and associated soft tissue and osseous abnormalities such as retrocalcaneal bursitis, paratendinitis and peritendinitis, and osteitis of the calcaneus.

In beach soccer, ankle injuries are notoriously less common compared to soccer, with reported incidence of ankle sprains ranging from 1 to 5%, and most injuries involve the foot and the toes [85, 86].

## Conclusion

While many ankle injuries in soccer share universal mechanisms with athletic activities, certain patterns are found to be relatively unique to this sport. Understanding these specific traumatic mechanisms of ankle injuries in soccer players in instances of both acute trauma and chronic overuse is crucial for a comprehensive MRI diagnosis.

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## Declarations

**Conflict of interest** The authors declare no competing interests.

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