

Anterior and Posterior Ankle Impingement

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Chronic anterior ankle pain is commonly caused by talotibial osteophytes at the anterior portion of the ankle joint. Morris [1], McMurray [2], and later Biedert [3], named this condition “athlete’s” ankle or “footballer’s” ankle, although it occurs in other sports, such as running, ballet dancing, high-jumping, and volleyball. The term “footballer’s ankle” has been appropriately replaced by “anterior ankle impingement syndrome.” Chronic posterior ankle pain is commonly caused by an os trigonum or other bony impediment, such as a hypertrophic posterior process of the talus.

The pain from impingement lesions is caused by synovial tissue being caught between the talus and the ankle mortise. Osteophytes or ossicles facilitate this process, because they limit the arc of motion. A differentiation has been made between soft tissue impingement and bony impingement lesions [3–5].

In general, osteophytes are the secondary manifestation of osteoarthritic changes [6,7]. However, repetitive minor trauma in the ankle, as seen in athletes, can induce spur formation [2], with radiographic characteristics similar to osteophytes.

In patients with anterior ankle impingement, recognizable pain on palpation is the most important clinical finding, while, for posterior impingement, recognizable pain on forced hyperplantarflexion is pathognomonic. When standard radiographs do not disclose the cause of an anterior impingement, special projections are used to disclose medially located bony spurs. When conservative management fails, surgery involves removal of bony or soft tissue impediments.

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Etiology of anterior ankle impingement

Repeated capsuloligamentous traction by repetitive kicking with the foot in full plantarflexion may induce traction spurs formation [2]. This hypothesis is supported by the fact that these spurs are frequently found in athletes [2-4,8,9] who repetitively force their ankle in hyperplantarflexion actions, with repetitive traction to the anterior joint capsule [10]. It assumes, however, that the capsular attachment is located at the anterior cartilage rim at the location where spurs originate.

The anterior joint capsule attaches on the tibia at an average of 6 mm proximal to the joint level [11]. On the talar side, the capsule attaches likewise approximately 3 mm from the distal cartilage border. The distance of capsular attachment to the most frequent location of bony spurs is thus relatively large. Hence, the hypothesis of formation of talotibial traction spurs from recurrent traction to the joint capsule is not plausible. This is supported by observations at arthroscopy [12]. In patients with bony impingement, the location of tibial spurs is reported to be at the joint level, and always within the confines of the joint capsule [12,13]. On the talar side, the typical osteophytes are found proximal to the notch of the talar neck. Tibial and talar osteophytes can easily be detected at arthroscopy with the ankle in forced dorsiflexion. The capsule does not need to be detached to locate these osteophytes.

O'Donoghue [14] considered the osteophytes to be related to direct mechanical trauma associated with the impingement of the anterior articular border of the tibia and the talar neck during forced dorsiflexion of the ankle. Here, bone formation is considered to be a response of the skeletal system to intermittent stress and injury, as evidenced by Wolff's law of bone remodeling [15]. According to Hawkins [16] runners, dancers and high-jumpers are prime examples of athletes who may be predisposed to this type of sports-related repeated trauma. Even though this etiologic factor is widely cited [4,5,9,16], scientific evidence for either is scarce.

Along the distal tibia, the width of the nonweight-bearing cartilage rim extends up to 3 mm proximal to the joint line. It is this nonweight bearing anterior cartilage rim undergoes development of osteophytes [5,11]. Damage to this anterior cartilage rim occurs in the majority of supination trauma [17,18]. Depending on the amount of damage, chondral and bone cell stimuli will initiate a repair reaction may cartilage proliferation, scar tissue formation, and calcification. Additional damage by ankle sprains, due to recurrent instability or forced dorsiflexion movements, will further enhance this process [3]. Recent studies demonstrated that chronic ankle instability is indeed significantly correlated with osteophytic formation in the medial ankle compartment [18,19]. Another etiologic factor in the development of spurs is recurrent microtrauma. In soccer players, spur formation is related to recurrent ball impact, which can be regarded as repetitive microtrauma to the anteromedial aspect of the ankle [20]. Repetitive trauma to the

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