(v) Chronic ankle instability

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Abstract

Injuries to the ligaments of the ankle are common, especially in athletes. Symptomatic ankle instability develops in as many as 10–40% following an acute injury. The causes of symptoms are multifactorial, encompassing pre-existing patient factors predisposing to instability, functional instability and mechanical instability. Chronic ankle instability occurs when patients suffer recurrent episodes of ankle sprains and the majority can be successfully treated with a functional rehabilitation programme. Those that fail require consideration of surgical intervention. A full history, clinical examination, radiological investigation and an understanding of the pathomechanics involved are vital to ensure that the most appropriate surgical strategy is adopted. Pain and swelling are commonly associated symptoms and may be more disabling than the episodes of instability. Concurrent intra and extra-articular pathologies must be addressed to achieve a successful functional outcome. Surgical options include arthroscopy, ligament reconstruction techniques, hindfoot alignment procedures and gastrocnemius release. This article focuses on the anatomy, pathomechanics and treatment of chronic lateral ankle instability. Medial, syndesmotic and subtalar instability are also discussed.

Keywords ankle injuries; ankle joint; joint instability; lateral ligament ankle; subtalar joint

Introduction

Injuries to the lateral ankle ligament complex or "ankle sprains" are the commonest sports related injury, accounting for 16–21% of all musculoskeletal injuries. The incidence in the UK of 52.7/10 000/year equates to 300 000 injuries/year.[1] Following ankle injury the majority of patients undergo a functional rehabilitation programme, which is usually successful in returning patients to functional normality. There is little role for surgery in the acute phase.[2] However, some patients develop residual symptoms of pain and/or instability as a consequence, which may be underestimated in clinical practice. Symptomatic ankle instability can develop in as many as 10–40% of patients following an acute event, even after adequate conservative treatment.[3] Chronic ankle instability does not exist as a single pathologic entity and the symptoms of "*sprained ankle syndrome*" are frequently multifactorial. Treatment is not only based upon a proper history, physical examination and radiological investigations but consideration of the pathomechanics of the condition.

Ankle joint anatomy and biomechanics

The ankle joint complex consists of three articulations: the talocrural, subtalar and distal tibiofibular joints. The three joints work together to allow coordinated movement of the hindfoot in three cardinal planes: the sagittal plane (plantarflexion–dorsiflexion), the frontal plane (inversion–eversion) and the transverse plane (internal and external rotation). Hindfoot motion does not occur in isolation but rather in a coordinated, coupled motion best described as pronation (dorsiflexion, eversion and external rotation) and supination (plantarflexion, inversion and internal rotation).[4]

The talocrural joint or '*mortice*' is formed by the articulation of the dome of the talus, the medial malleolus, the tibial plafond and the lateral malleolus. In isolation, the ankle joint is a modified hinge joint allowing dorsiflexion and plantarflexion. The sagittal plane motion of the ankle joint passes through the tips of the malleoli. Since the lateral malleolus is longer and is posterior to the medial malleolus, the plane is oblique to the plane of the floor and also to the transverse plane. As the ankle dorsiflexes, it also rotates externally and *vice versa*.[5]

The biomechanics of the ankle is complex, with three factors contributing to stability. In a loaded ankle the osseous anatomy is the most critical as the talus compresses into the bony mortice resulting in primary stability. In an unloaded ankle a combination of static ligamentous restraints and musculotendinous units play more vital roles, with each ankle ligament contributing a different function depending of the position of the foot and ankle in space.

Lateral ligaments

The lateral ankle ligament complex is composed of three main ligaments: the anterior talofibular, calcaneofibular and posterior talofibular ligaments (Figure 1). The anterior talofibular ligament (ATFL) is the weakest of the lateral ligaments. It has a load to failure 2–3.5 times lower than the calcaneofibular ligament (CFL) and two times lower than the posterior talofibular ligament

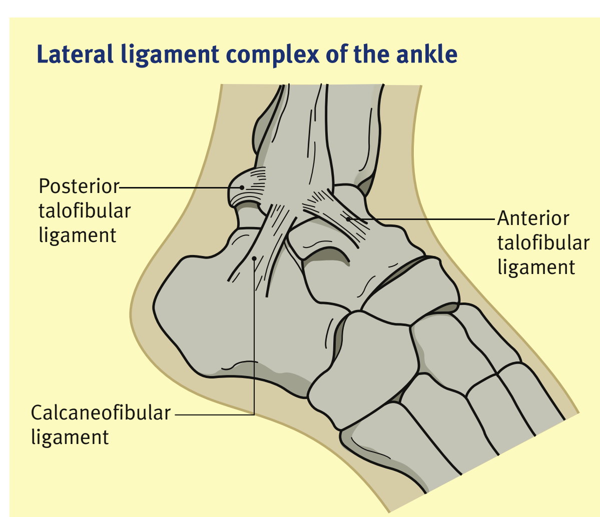

Figure 1 The lateral ligament complex of the ankle (to be redrawn).

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(PTFL).[6] The ATFL is intra-capsular and originates 1 cm proximal from the tip of the lateral malleolus just anterior to the fibular facet. It extends distally and medially inserting onto the neck of the talus and functions as a check-rein when the foot is in an equinus or inverted position. It is therefore most vulnerable when the ankle is in plantarflexion and is the most frequently injured ligament. It is usually disrupted through its midsubstance. The ATFL is taut in plantarflexion and acts to prevent anterior displacement of the talus from the ankle and excessive inversion and internal rotation.

The CFL originates adjacent to the ATFL, approximately 8 mm from the tip of the fibula, and courses distally and posteriorly across both the ankle and subtalar joints to insert onto the lateral aspect of the calcaneus just behind and above the peroneal tubercle. It forms the floor of the peroneal tendon sheath. When the ankle is dorsiflexed, the ATFL is loose and the CFL is taut.

The PTFL is short, thick and is the strongest of the three ligaments and hence is rarely injured. It originates from the medial surface of the lateral malleolus and inserts into the posterior aspect of the talus. The talar and fibular insertions of the PTFL are broad. The PTFL is under tension only when the ankle is in extreme dorsiflexion, and provides restraint to both inversion and internal rotation when the ankle is loaded.[7]

Medial ligaments

The deltoid ligament has significantly higher load to failure than its lateral ligament counterparts and thus requires much greater force to injure. The anatomy of the deltoid ligament comprises of both superficial and deep components. The superficial deltoid originates from the anterior colliculus of the medial malleolus and inserts into both the navicular and the sustentaculum tali of the os calcis. The deep deltoid ligament is a key component of ankle stability. It originates from the posterior colliculus and inserts into the non-articular medial surface of the talus. Classically the superficial deltoid ruptures first followed by the deep deltoid at its talar insertion due to forced abduction or eversion. The biomechanical function of the deltoid ligament is to resist abduction and lateral translation of the talus. The deep deltoid ligament provides the greatest restraint against talar shift.[8]

Syndesmosis

The syndesmosis refers to the distal articulation between the tibia and fibula, and forms the stable roof of the talocrural joint. The joint is stabilized by a thick interosseous membrane that runs throughout the length of the two bones. There are three ligaments at the ankle: the anterior–inferior tibiofibular ligament (AITFL), the posterior–inferior tibiofibular ligament (PITFL) and the interosseous ligament (IOL). The AITFL is the most commonly injured and results in the so-called '*high ankle sprain*'. The IOL is both stronger and stiffer than the AITFL but is commonly injured in combination with the AITFL. The PITFL is smaller than the AITFL and is composed of both a deep portion, the transverse tibiofibular ligament, and a superficial portion. The PITFL contributes most towards the stability of the syndesmosis,[9] and acts to deepen the talocrural joint by projecting inferior to the tibia, preventing posterior translation of the talus.

Biomechanically, a limited degree of motion is necessary at the syndesmosis for normal ankle function. When the talus is wider anteriorly than posteriorly, as the ankle moves from plantarflexion to dorsiflexion the lateral malleolus externally rotates by 11° and the distance between the tibia and fibula increases by 1.5 mm.

Subtalar joint anatomy and biomechanics

The subtalar joint is formed by the articulation between the talus and os calcis. The joint is divided into anterior and posterior articulations separated by the sinus tarsi and canalis tarsi. The anterior joint consists of the talonavicular joint including the anterior and middle facets of the calcaneum. The posterior joint contains the posterior facet and its corresponding inferior talar surface. The anterior joint lies more medial than the posterior joint and has a higher centre of rotation. This results in a subtalar joint axis of rotation that is 42° upwards in the sagittal plane and 23° medial to the midline of the foot in the transverse plane[10] (Figure 2).

There is debate in the literature regarding the key ligamentous stabilizers of the subtalar joint, in both their terminology and reported functions. It is generally accepted that there are three ligamentous groups; the peripheral, deep and the retinacular ligaments. There are three peripheral ligaments, the calcaneofibular ligament (CFL), lateral talocalcaneal ligament (LCTL) and fibulotalocalcaneal ligament (FTCL). There are two deep ligaments, the cervical ligament (CL) and interosseous ligament (IOL).

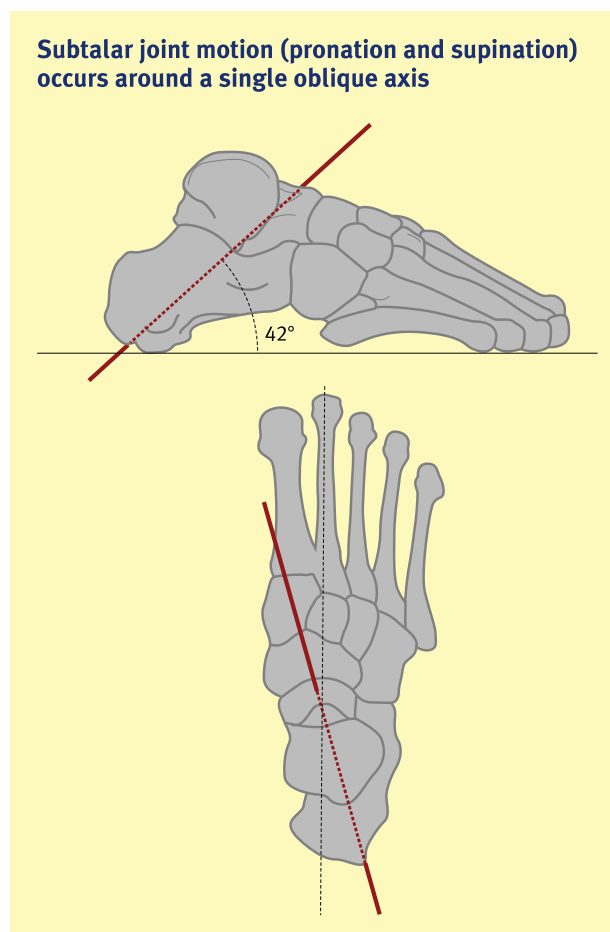

Figure 2 Subtalar joint motion (pronation and supination) occurs around a single oblique axis (to be redrawn).

