Osteoarthritis of the ankle

Andrew H N Robinson

Troy Keith

Abstract

Ankle arthritis is a debilitating condition. Primary ankle joint osteoarthritis is much rarer than hip or knee arthritis. The majority of cases are post-traumatic secondary to previous articular trauma or ligamentous instability. Treatment methods available are guided by disease severity and the patient's functional demands. Weight loss, activity modification, the wearing of an ankle brace or high topped-lace up boots and orthoses should be the first line of treatment. Joint sparing procedures such as arthroscopic debridement, supramalleolar osteotomy and distraction arthroplasty have been trialed with low levels of supporting evidence. The demand for joint preservation has led to the development of total ankle allografting procedures, but these have fallen out of favour. Despite advancements, the gold standard for end stage arthritis remains arthrodesis, although evidence for total ankle arthroplasty in certain groups of patients is increasing. The current third generation implants have improved survivorship although their results remain inferior to arthroplasty of the hip and knee. There are currently two prospective randomized control trials of ankle replacement versus arthrodesis under way as well as several national joint registries, which will hopefully better define those patients who will gain most benefit from replacement as opposed to fusion.

Keywords ankle; arthritis; arthrodesis; arthroplasty; total ankle arthroplasty

Introduction

The incidence of ankle osteoarthritis (OA) in the UK has recently been estimated to be 47.7 per 100 000. Of the 29 000 symptomatic cases referred to specialist surgeons in the UK each year, approximately ten percent undergo arthrodesis or arthroplasty.[1] The societal impact of ankle arthritis is significant with the degree of physical impairment having been estimated as equivalent to end stage kidney disease or congestive heart failure. The ankle is rarely affected by primary OA with 80% of all cases of ankle OA being trauma related.[2] The available treatments for ankle arthritis are diverse. An appreciation of the patient's expectations and the severity of disease is paramount in deciding management.

Andrew H N Robinson ***BSc FRCS (T & O)*** *Consultant Orthopaedic Foot and Ankle Surgeon, Cambridge University Hospitals NHS Trust, Cambridge, UK. Conflicts of interest: The author is a paid consultant to Orthosolutions ltd.*

Troy Keith ***FRACS(Orth) MBBS (Hons)*** *Senior Orthopaedic Foot and Ankle Fellow, Addenbrookes Hospital, Cambridge University Hospitals NHS Trust, Cambridge, UK. Conflicts of interest: No conflicts of interest.*

Structure and function

The ankle's bony anatomy confers a high degree of stability and congruence when it is loaded. It is composed of three articulations: the tibiotalar, fibulotalar, and tibiofibular joints. The talar body has a convex dome which is wider anteriorly than posteriorly. When the talus is plantarflexed, its narrowest portion occupies the ankle mortise and allows for rotation between the talus and mortise. However, when the talus is maximally dorsiflexed, the tibiofibular syndesmosis accommodates the wider portion of the talus in the ankle mortise, allowing little or no rotation to occur and stabilizing it.

The ankle joint does not simply act as a hinge, nor is its primary axis fixed, but changes direction and position throughout the range of ankle motion. Accordingly, its motion is coupled, with external rotation and eversion occurring with dorsiflexion and internal rotation and inversion with plantarflexion. The talus is a frustum of a cone with its apex oriented medially. A frustum is a segment of a solid lying between two parallel lines, which cut it (Figure 1). Despite the arcs of curvature either side of the trochlear being different they subtend the same angle.[3]

It has been estimated that ankle degeneration is one ninth as common as hip or knee osteoarthritis, despite the ankle being the most frequently injured weight-bearing joint. The reason for this is uncertain, although ankle cartilage does differ from that of the hip and knee. The articular cartilage of the hip and knee is said to be at least 3 mm thick in load bearing areas, and may exceed 6 mm. The maximal articular cartilage thickness in the ankle measures 2.7 mm on high resolution MRI.[4] It was previously thought the thickest cartilage was found at the centre of the talar dome and tibial plafond, but the thickest cartilage is now thought to be anterolateral and posteromedial, on the talar shoulders.[3,4] On the tibial side, the thickest cartilage areas are seen on the central and anterior tibial plafond and the curved area of transition between the plafond and medial malleolus.[4]

The relative rarity of primary ankle osteoarthritis, compared to the hip, may be a consequence of its ability to sustain higher peak contact stresses, resulting in a more favourable linear age related decline. Specifically, ankle articular cartilage tensile fracture stress and tensile stiffness have been shown to deteriorate less rapidly with age than in hip articular cartilage. Hip articular cartilage is also felt to exhibit an exponential decline with age.[2]

Buckwalter and Saltzman hypothesized that post-traumatic ankle osteoarthritis is the result of excessive articular surface contact stress resulting from post-traumatic joint incongruity and instability, such that the cartilage's capacity to repair is exceeded. Their contention is that post-traumatic ankle OA progresses through three stages as shown in Table 1.[2]

Tibial plafond fractures, malleolar fractures, fracture dislocations of the ankle, inadequate fracture reduction and ligamentous injury have been consistently implicated in the development ankle OA. In pilon fractures the energy of the injury, articular comminution and displacement are associated with poorer outcomes. The malleolar fracture pattern attributed with the highest incidence of OA has been Weber C fractures in association with medial malleolar fracture. With greater than one third of patients developing advanced radiographic degenerative changes in a long-term, retrospective review of 102 ankle fractures, at 18

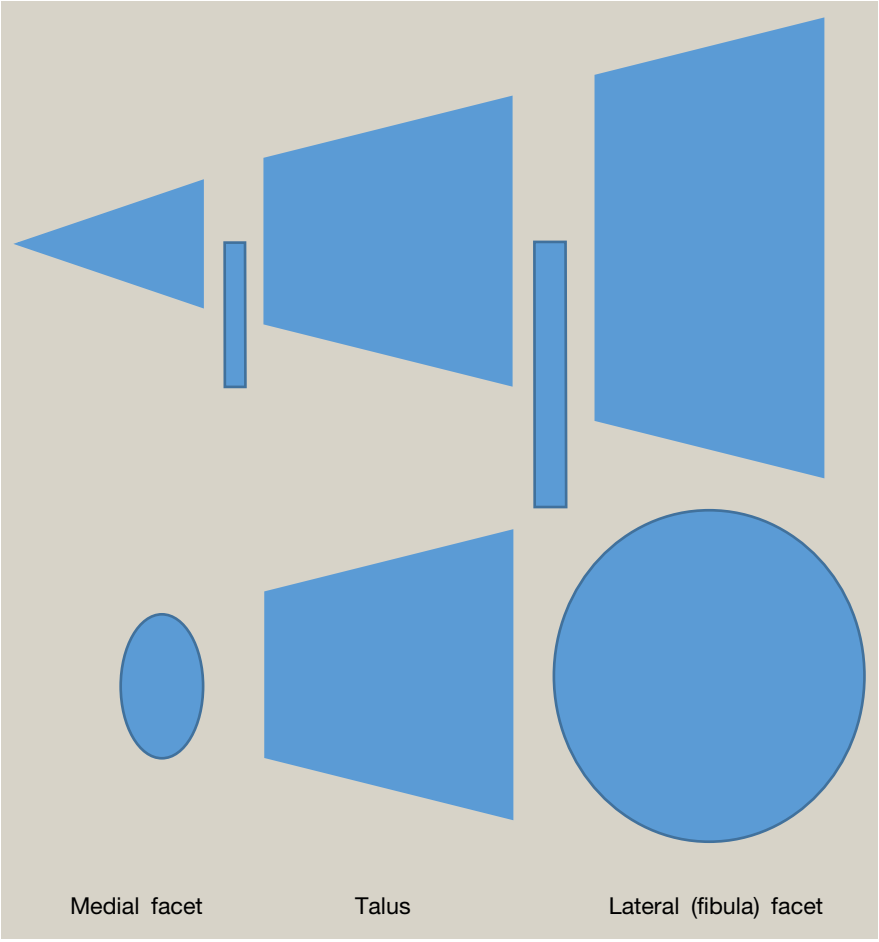

Figure 1 Trochlear of the talus in cross section, or frustum, of a cone. A frustum is a segment of a solid lying between two parallel lines, which cut it. The cross-section of the medial facet creates an elliptical section and the cross-section of the lateral facet (fibula) creates a circular section.

years.[5] The presence of a posterior malleolar fracture has also been shown to correlate with poorer clinical and radiographic outcomes, especially if there is inadequate reduction of the posterior malleolar articular fragment. Following ankle fractures the majority of arthritic change has been reported to occur early, in the first 12–18 months.[6]

Osteochrondral injuries are also linked to the development of OA. However, the determining factor may be the depth and

Stages in development of post-traumatic ankle osteoarthritis

Stage I

Increased contact stress damages articular cartilage

Disruption or alteration of the matrix macromolecular framework associated with an increase in water concentration may be caused by high levels of contact stress. At first, the type 11 collagen concentration remains unchanged, but the collagen meshwork may be damaged and the concentration of aggrecan and the degree of proteoglycan aggregation decrease.

Stage II

Chrondrocyte response to matrix disruption or alteration

When chrondrocytes detect a disruption or alteration of their matrix they respond by increasing matrix synthesis and degradation and by proliferating. Their response may restore the tissue, maintain the tissue in an altered state, or increase cartilage volume. They may sustain an increased level of activity for years.

Stage III

Decline in chrondrocyte response

Failure of the chondrocyte response to restore or maintain the tissue leads to loss of articular cartilage accompanied or preceded by a decline in the chrondrocytic response. The causes for the decline in chrondrocytic response is poorly understood, but it may be the result of mechanical damage to the tissue with injury to chrondrocytes and a down regulation of the chrondrocytic response to anabolic cytokines.

Table 1