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Ankle Arthritis: Etiology and Epidemiology

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The ankle joint is subject to symptomatic arthritis at a much lower rate than the other major weight-bearing joints in the lower extremity. When degenerative changes do occur, they are most commonly secondary to trauma, chronic ankle malalignment, ankle instability, inflammatory conditions, infection, recurrent hemarthrosis, neuropathic and neoplastic conditions.
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The ankle joint has a much lower incidence of symptomatic arthritis compared with other major joints.[1,2] This is despite the fact that the articular cartilage in the ankle experiences the greatest contact force per unit area of any major joint in the body[3-5] and the ankle joint is one of the most commonly injured areas in orthopedic surgery.[6] The tibia, fibula, and talus articulate very precisely to form the ankle mortise. Medial, lateral, and syndesmotic ligaments provide further stabilization and serve to maintain even distribution of force on the articular surface throughout normal ankle motion. The articular cartilage of the ankle is specialized for a joint that is highly congruous and must transmit high forces over a relatively small area. Compared with the hip and knee, the articular cartilage in the ankle is more uniform, averaging 1 to 1.7 mm.[7] It is also stiffer and has less diminution with aging.[8] Despite the unique and highly functional design of the ankle joint, it can still become afflicted with arthritis, and the purpose of this article is to review the prevalence and outline the various causes of ankle arthritis.

Symptomatic ankle arthritis can develop from a variety of causes (Table 1), including ankle trauma; conditions that increase joint loading, such as malalignment or hind foot fusions; ligament disruption leading to instability; inflammatory arthritis, such as rheumatoid arthritis; crystal arthritis, such as gout; infectious processes; and even neoplastic conditions, such as pigmented villonodular synovitis (PVNS). The most common cause of ankle arthritis is trauma that directly or indirectly accounts for approximately 75% of all cases of ankle arthritis.[4,9,10] Another common cause of ankle arthritis is altered force transmission across the ankle joint with subsequent cartilage degeneration secondary to malalignment. Alignment abnormalities in the coronal and sagittal plane stemming from deformities at the knee, tibia, or foot can lead to ankle arthritis over time and will be reviewed. Finally, we will review less common causes of ankle arthritis, such as inflammatory syndromes, septic insult to the join, neoplastic, neuropathic, and hemophilic degeneration.

Posttraumatic Injury

It has been well established that degeneration of the ankle joint most commonly occurs secondary to trauma.[4,9,10] Trauma causes arthritis either by (1) direct cartilage injury; (2) uneven joint loading caused by intra-articular fractures with joint incongruity; (3) altered loading caused by malalignment (eg, from tibial malunion); or (4) a combination of these factors. Direct trauma refers to the direct injury to the cartilage occurring at the time of the trauma. It has been reported that clinically significant cartilage damage was present in more than 70% of operatively treated ankle fractures that underwent arthroscopy before definitive fracture treatment.[11-13] Even without macroscopic cartilage damage it has been shown that a direct blow to the cartilage surface can lead to marked cartilage cell death.[14] Therefore, depending on the mechanism and level of energy of the insult, a variable extent of cartilage cell death will occur after major ankle trauma. There is a clear relationship between the type of injury (eg, rotational ankle fracture vs axial load Pilon fractures), the degree of comminution, and the amount of energy imparted to the ankle joint at the time of injury. Pilon fractures have a much greater component of compression at the level of the articular surface compared with rotational ankle

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Table 1 Causes of Ankle Arthritis

Trauma	Direct cartilage injury
	Incongruent ankle joint
	Trauma leading to malalignment
Chronic ankle malalignment (causing increased focal joint pressure)	Primary ankle malalignment
	Secondary (altered mechanical axis in the hip, femur, knee, tibia and foot)
Ankle instability (causing repetitive joint injury and/or increased focal joint pressure)	Lateral instability
	Deltoid insufficiency
	Syndesmotic disruption
Inflammatory arthritis	Rheumatoid arthritis
	Psoriatic arthritis
	Ankylosing spondylitis
	Crystalline arthropathy (gout, pseudogout)
Infectious	Septic arthritis
	Osteomyelitis
Hemorrhagic	Hemophilia
	Hemochromatosis
	Recurrent traumatic hemarthroses
Neuropathic	Charcot arthropathy (eg, diabetes)
Neoplastic	Benign (eg, PVNS)
	Malignant

fractures. This in turn produces greater cartilage damage and a subsequently higher rate of ankle degeneration.

Rotational ankle fractures are very common orthopedic injuries and frequently require open reduction with internal fixation. A series of more than 300 ankle fractures reported by Lindsjo et al[15] is commonly cited with reference to the rates of posttraumatic ankle arthritis. They found that the rates of symptomatic arthritis in operatively treated Weber A, B, and C ankle fractures was 4%, 12%, and 33%, respectively. The presence of a posterior maleolar fracture pointed towards greater likelihood of developing arthritis. This finding suggests that some element of direct cartilage injury occurs in many seemingly benign ankle fractures.

Malreduction of ankle fractures will increase the long-term rate at which ankle arthritis develops. It has been reported that even 1 mm of increased lateral talar shift will decrease the tibiotalar contact area by 42%.[16] This finding highlights the high degree of congruity that the ankle joint possesses and its sensitivity to anatomic derangement. Similarly, failure to anatomically reduce intra-articular ankle fractures will also lead to increased ankle joint loading. Over time, this increased focal tibiotalar contact pressure will lead to cartilage breakdown and arthritis.

However, even with an anatomic reduction ankle injuries associated with high-energy compression forces, such as Pilon fractures, commonly develop ankle arthritis as the result of direct cartilage injury.[3] The propensity to develop posttraumatic arthritis is greatly influenced by the energy level of the fracture and the degree of comminution. Therefore, despite the tremendous effort taken to restore or rebuild the articular alignment in severe fractures of the plafond, patients with these fractures are still at high risk for developing significant posttraumatic ankle arthrosis.[1,17,18]

Talar fractures occur much less frequently than those of the malleoli or plafond. Most of the talus is covered with articular cartilage, making it particularly susceptible to posttraumatic changes in the tibiotalar and subtalar joints. In addition, blood supply can be compromised from fractures, leading to osteonecrosis and subchondral collapse. Inokuchi et al[19] defined talar neck fractures as those with an inferior fracture line anterior to the lateral talar process on a lateral radiograph. Fractures extending into or posterior to the lateral process were said to involve the body.

With increasing displacement of talar neck fractures and subsequent adjacent joint dislocations, there is an increase in the rates of osteonecrosis.[20] The classification by Hawkins et al[20] and later modified by Canale et al[21] is centered on this principle. Rates of osteonecrosis following talar neck fractures has been reported to range from 0% to 15% for Type I, 20%-50% for Type II and 80%-100% for Type III and IV fractures.[20-24] Worse outcomes have been found with greater comminution[25] and open talar fractures.[23,24,26] Varus malunion is a potential pitfall that leads to subtalar joint stiffness and increased force transmission occurs through the lateral aspect of the foot and ankle. Talar body fractures can be isolated or occur in conjunction with talar neck fractures. Such fractures can be difficult to classify and can also be complicated by osteonecrosis and arthropathy of the tibiotalar joint.[27,28]

Malalignment

The articular cartilage of the ankle is thinner on average than that of the knee and hip and is more uniform in thickness across the mortise.[7,8,29] Such properties highlight the efficiency of the ankle under normal condition but may make it more susceptible to degeneration from even minor changes in joint loading. Compared with direct traumatic injuries to the ankle joint that lead to a more global degeneration, alignment abnormalities in the coronal and sagittal plane produce more focal pathology.[30,31] With alteration in the mechanical axis, there is increased loading per unit area and asymmetric wear can occur. The ankle joint and subtalar joints have the capacity to accommodate to some degree, but with greater angulation, the joint begins to decompensate. It is important to assess the mechanical and anatomic alignment of the lower extremities in patients with ankle degeneration because the ankle pathology may be secondary to other underlying conditions. In many patients who were previously thought to have developed "idiopathic" ankle arthritis it has subsequently become apparent that their ankle arthritis developed because of focal joint loading secondary to malalignment.

Medial or varus ankle arthritis tends to occur when the mechanical axis of the limb falls to the medial half of the ankle joint. There are numerous conditions that can lead to such pathologic loading of the joint, including genu varum and varus tibial malunions, both of which lead to a varus alignment of the ankle. Iatrogenic causes include malposi-