Revue de chirurgie orthopédique et réparatrice de l'appareil moteur (2008) **94S**, S398—S408

Disponible en ligne sur
ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com

TRAINING CONFERENCE, CONFÉRENCE D'ENSEIGNEMENT, CONGRÈS SFA, LYON, DÉCEMBRE 2007

Treatment of osteochondral defects of the talus
Traitement des lésions ostéochondrales de l'astragale

C.J.A. van Bergen, P.A.J. de Leeuw, C.N. van Dijk*

Department of Orthopaedic Surgery, Academic Medical Center, University of Amsterdam, PO Box 22700, 1100 DD Amsterdam, The Netherlands

Available online 11 November 2008

KEYWORDS
Osteochondral defect;
Osteochondral lesion;
Talus;
Ankle;
Treatment

Summary This review article provides a current concepts overview of osteochondral defects of the talus, with special emphasis on treatment options, their indications and future developments. Osteochondral defects of the talar dome are mostly caused by a traumatic event. They may lead to deep ankle pain on weight-bearing, prolonged swelling, diminished range of motion and synovitis. Plain radiographs may disclose the lesion. For further diagnostic evaluation, computed tomography (CT) and magnetic resonance imaging (MRI) have demonstrated similar accuracy. Computed tomography is preferred for preoperative planning. Treatment options are diverse and up to the present there is no consensus. Based on the current literature, we present a treatment algorithm that is mainly guided by the size of the lesion. Asymptomatic or low-symptomatic lesions are treated nonoperatively. The primary surgical treatment of defects up to 15 mm in diameter consists of arthroscopic debridement and bone marrow stimulation. For large cystic talar lesions, retrograde drilling combined with a bone graft is an important alternative. In adolescents or in (sub)acute situations, in which the fragment is 15 mm or larger, fixation of the fragment is preferred. Osteochondral autograft transfer and autologous chondrocyte implantation (ACI), with or without a cancellous bone graft, are recommended for secondary cases as well as large lesions.

Introduction

An osteochondral defect (OD) is the collective term for focal lesions involving the articular cartilage and subchondral bone. If only cartilage is involved in the pathology, the term chondral defect is used. Many synonyms are used, including osteochondritis dissecans [1], transchondral fracture [2], flake fracture [3], talar dome fracture [4], osteochondral fracture [5], osteochondral lesion [6] and osteochondral defect [7]. A differentiation should be made between traumatic and nontraumatic origin (i.e. osteochondritis dissecans). A traumatic event may lead to (partial) detachment of an (osteo)chondral fragment, which may further evolve in the formation of a subchondral cyst with or without osteonecrosis. There is sometimes confusion

* Corresponding author.
E-mail addresses: c.j.vanbergen@amc.uva.nl (C.J.A. van Bergen), c.n.vandijk@amc.uva.nl (C.N. van Dijk).

0035-1040/$ – see front matter © 2008 Elsevier Masson SAS. All rights reserved.
doi:10.1016/j.rco.2008.09.003

between traumatic and nontraumatic because a nontraumatic OD may become symptomatic after trauma.

In 1856, Monro first reported the presence of cartilaginous bodies [8]. In 1888, König used the term osteochondritis dissecans for loose body formation associated with articular cartilage and subchondral bone fracture [9]. He referred to an inflammatory process, although this has never been proved to be involved in the pathology. It was not until 1922 that the first report on osteochondritis dissecans in the ankle was published [10]. The talar dome is the secondly most common location in the human body; most occur in the knee [11].

An OD is often not recognized and therefore not adequately treated. The nonrecognition is mainly due to the fact that the lesion produces symptoms of previous trauma, and it cannot always be identified on plain radiographs [12]. After standard treatment for acute ankle sprains, residual symptoms are reported in 33% of patients [13]. In these cases the possibility of an OD should be considered.

The talus has a limited reparative capacity because of its restricted vascular supply [14]. Inappropriate treatment of OD may result in chronic ankle pain, functional impairment, subchondral cyst formation, and eventually osteoarthritis of the ankle [14—16].

For the last decade great developments have been made in the surgical treatment. Despite advancements in options like osteochondral autograft transfer system (OATS) or autologous chondrocyte implantation (ACI), arthroscopic debridement and bone marrow stimulation remain the best treatment that is currently available for defects up to 15 mm in diameter [17,18]. In larger (cystic) defects this treatment is less successful, and hence there is more debate [19,20].

The aim of this article is to provide an overview of treatment options and their indications for ODs of the talus, based on the current evidence.

Etiology

In 1985, trauma was described in 98% of lateral lesions and in 70% of medial lesions [1]; more recently, 93% for lateral and 61% for medial lesions were reported [18]. As not all patients report a history of ankle injury, a subdivision can be made in the etiology of nontraumatic and traumatic defects.

Ischemia, subsequent necrosis and possibly genetics are etiologic factors in nontraumatic ODs [14]. Furthermore, ODs in identical twins [21] and in siblings [22] have been described. Less reported possible causes are metabolic, vascular, endocrine and degenerative factors, as well as morphologic abnormalities [2,11,23].

In the etiology of traumatic ODs, ankle sprains play the largest role. A severe ankle sprain may cause a small fracture and subsequent impaired vascularity, leading to the formation of an OD. Alternatively, the cause may not be a single event but may consist of a series of repeated, less intense injuries [14,23]. Microtraumas caused by repetitive surface loading or excessive stress can lead to cartilage cellular degeneration or apoptosis and thickening of the subchondral bone [24].

Mechanism of injury

During an ankle sprain the talus twists inside the ankle mortise, which may lead to a bruise and subsequent softening or even delamination of the cartilage. Separation may occur in the upper layer, as a result of shearing forces, or may occur in the subchondral bone. Osteocartilaginous fragments either remain partially attached or become loose bodies in the ankle joint. The subchondral fracture has no soft tissue attachments and is highly susceptible to subsequent avascular necrosis [11]. The repetitive forcing of synovial fluid into the underlying cancellous bone with every step of walking may create a subchondral cyst [25]. The repetitive fluid pressure may prevent healing of a subchondral cyst.

Berndt and Harty clearly described the trauma mechanism in cadaver ankles [2]. They were able to reproduce a lateral defect by strong inversion of a dorsiflexed ankle, leading to compression of the lateral border of the talar dome against the face of the fibula. Partial detachment of the chip occurred when the lateral ligament ruptured. They reproduced medial lesions by plantar flexion and inversion of the ankle combined with slight anterior displacement and lateral rotation of the tibia upon the talus.

The lateral lesions are typically shallow and wafer-shaped, indicating the shear mechanism of injury [15]. Because of their shape, lateral lesions are more frequently displaced than medial lesions. In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of torsional impaction [15]. These lesions are usually larger than lateral lesions [26].

Epidemiology

With the increased awareness and newer diagnostic techniques, the incidence of OD seems to increase [27]. In 1955, Bosien et al. described an incidence of 7% in 113 patients conservatively treated for acute lateral ankle ligament ruptures [13]. Later, van Dijk et al. reported 4% fresh talar dome lesions and 67% fresh chondral lesions of any kind in 30 patients who had operative repair of acute ruptures of lateral ligaments [28]. More recently, an even higher incidence was reported, namely 41% of 86 patients with anterior talofibular ligament disruptions and 71% of 92 patients with distal fibular fractures [29]. However, the majority of these reported lesions were located at the cartilage covering the anterior aspect of the medial malleolus and the opposite medial talar facet or the anteromedial rim of the tibiaplafond [28,29]. Accordingly, talar ODs were found in 28% [30] and 40% [31] of patients with ankle fractures treated by arthroscopically assisted open reduction and internal fixation. In these series the highest incidence was found in patients with distal fibular fractures [30,31].

Most ODs are localized on the posteromedial (58%) or anterolateral (42%) talar dome (Fig. 1) [18], although anteromedial, posterolateral and central lesions also occur [26]. In a large magnetic resonance imaging (MRI) survey of 428 affected ankles 53% of the lesions were localized centromedially and 26% centrolaterally [26].

In 4—7% of patients the occurrence of the defect is bilateral [2,6,15], suggesting nontraumatic osteochondritis