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Review article

Osteochondritis dissecans of the knee

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ABSTRACT

Osteochondritis dissecans (OCD) of the knee is an idiopathic, focal, subchondral-bone abnormality that can cause instability or detachment of a bone fragment and overlying articular cartilage, with subsequent progression to osteoarthritis. The diagnosis is usually made during adolescence. Mechanical factors play a major role in the pathophysiology of OCD. When the radiographic diagnosis is made early in a patient with open physes, healing can often be obtained simply by restricting sports activities. The degree of lesion instability can be assessed by magnetic resonance imaging. When the lesion remains unstable and the pain persists despite a period of rest, surgery is indicated. Arthroscopic exploration is always the first step. Drilling of the lesion produces excellent outcomes if the lesion is stable. Unstable lesions require fixation and, in some cases, bone grafting. Defects must be filled, depending on their surface area. Although many surgical techniques are available, the therapeutic indications are now standardized.

1. Introduction

Osteochondritis dissecans (OCD) of the knee is an idiopathic, focal, subchondral-bone abnormality that can cause instability or detachment of a bone fragment and overlying articular cartilage, with subsequent progression to osteoarthritis. The diagnosis may be made in childhood or adulthood. Data reported at the 2005 SoFCOT symposium indicated a mean age at diagnosis of 16.5 years [1]. The terms "open physes" and "closed physes" should be preferred over the terms "juvenile" and "adult". Despite advances in our understanding of this enigmatic condition, many issues remain unresolved, particularly regarding the pathophysiology, indications for magnetic resonance imaging (MRI), MRI signs of instability, current treatment methods, and feasibility of developing a treatment algorithm.

2. Epidemiology

A recent epidemiological study failed to identify any cases in children younger than 6 years of age, and the diagnosis was made 3.3 times more often between 12 and 19 years than between 6 and 11 years [2]. The incidence was 9.5/100,000. The risk of developing OCD was 3.8 times higher in boys than in girls. Most patients are athletes [3]. The most common site is the medial femoral condyle, particularly on its lateral surface. The lateral femoral condyle and patella were affected less often and the tibial plateau very rarely.

3. What is the pathophysiology?

3.1. Histology

A histological study evaluated osteochondral plugs from the center of OCD lesions [4]. A cleft was visible between the fragment and the surrounding trabecular bone. Interestingly, the fragments had not always been rated as unstable at arthroscopy despite being clearly separated from their bed. Thus, arthroscopic instability may be a delayed finding compared to the natural course of the condition. The subchondral bone was fractured and the fragment necrotic, although which of these two abnormalities occurred first was unclear. The fragment was composed of necrotic trabecular bone, viable trabecular bone, or cartilage with no bone. The deep surface of the fragment was covered with dense fibrous or cartilaginous tissue, resembling a non-union. Active bone remodeling was apparent beneath the surface of the basal side of the lesion. Thus, the necrotised fragment either undergoes re-ossification followed by incorporation or remains separated, as in a non-union, and eventually detaches.

3.2. Micro-trauma hypothesis

This hypothesis is, by far, supported by the best level of evidence. The initiating factor may be impingement on the anterior tibial

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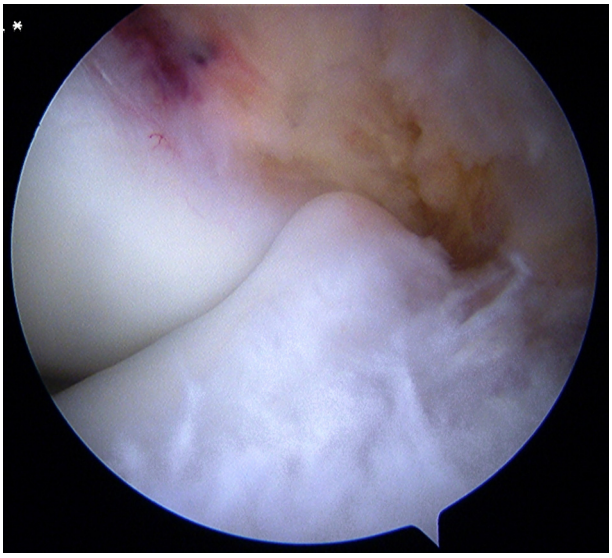

Fig. 1. Arthroscopic view of the anterior tibial spine in contact with the lateral surface of the medial femoral condyle during anterior cruciate ligament reconstruction.

spine. Wechter et al. found greater posterior and medial tibial slope in patients with medial condyle OCD, suggesting a role for micro-trauma from the anterior tibial spine [5]. The smaller intercondylar notch width in patients with OCD may promote impingement [6]. We found that the anterior tibial spine was larger in patients with OCD [7], in keeping with reports by Fairbank and Smillie (Fig. 1). In young baseball catchers, who frequently assume a crouched position, McElroy et al. showed that the OCD lesions were located more posteriorly on the femoral condyle [8]. A more distal location of the footprint of the posterior cruciate ligament (PCL) may add repetitive traction loading to the impingement [9]. Smillie [10] then Cahuzac et al. [11] previously pointed out the close relationships between the OCD lesion and the femoral PCL footprint. In an MRI study, Laor et al. found disruption of the secondary physis, i.e., the edge of the enchondral ossification center of the condyle [12]. This micro-trauma-induced disruption, akin to epiphysiodesis, may result in uneven growth with the production of irregular subchondral bone that may predispose to OCD. Patients with lateral condyle OCD often have a discoid meniscus responsible for repetitive abnormal loading. Complete discoid menisci were associated with central OCD and incomplete discoid menisci with peripheral OCD [13].

3.3. Other hypotheses

Other hypotheses have been put forward but failed to garner support. As suggested by the designation "osteochondritis dissecans" used in 1888 by König, a moderate inflammatory foreign-body response has been described. A role for endocrine disturbances has been suggested. Reports of familial cases [14], particularly in identical twins, prompted genome-wide association studies, which recently identified several candidate loci in humans [15]. Ischemia responsible for subchondral bone necrosis is often incriminated. Causes of ischemia may include trauma or arterial micro-thrombosis resulting in focal disruption of the blood supply.

3.4. In brief

OCD is clearly a multifactorial condition in which micro-trauma plays a major role. Multicenter studies and task forces such as Research in Osteo Chondritis of the Knee (ROCK) will provide further insights in the near future.

4. Clinical diagnosis

There are three main presentations:

- incidental discovery in an asymptomatic individual;
- mechanical pain during sports (the most common);
- nearly continuous mechanical pain with swelling and locking of the joint.

Pain upon weight bearing is the predominant symptom in 80% of cases [1]. Patients may also report swelling, catching, or locking of the joint. In patients with an unstable or loose fragment, transient quadriceps blocks may result in buckling of the knee. A systematic physical examination must be performed, as the pain may be unrelated to the OCD, which is then discovered incidentally.

The physical findings are often limited. Femoro-tibial alignment in the coronal plane should be assessed. Medial condyle OCD is associated with varus and lateral condyle OCD with valgus of the knee [16]. Finger pressure on the femoral condyle at various degrees of knee flexion may elicit the patient's usual pain. The Wilson test consists in bending the knee at 90° then passively moving it to 30° of flexion while rotating the foot medially. If the usual pain occurs during the test and resolves when the foot is rotated laterally, the test is positive. The Wilson test detects only medial condyle lesions and is interpretable only when positive. It is a helpful follow-up tool. A joint effusion [3] or a sudden increase in pain intensity suggests an unstable lesion.

The functional impact of OCD is usually moderate. Thus, the International Knee Documentation Committee (IKDC) score is higher than in other knee disorders of adolescence [17].

5. Imaging studies

5.1. Radiographs

Radiography is the first step. An antero-posterior view, a lateral view, and a tunnel view with the knee flexed at 60° should be obtained. A skyline view is required if an OCD lesion of the patella or trochlea is suspected. As OCD is bilateral in about 15% of cases, radiographs of the contralateral knee should be taken.

5.1.1. Stages of OCD

Bedouelle described a classification during an instructional course lecture delivered in 1988 [18] (Fig. 2). Although this classification is extremely accurate, distinguishing stages Ia and Ib and stages IIa and IIb can be difficult [1,11]. Furthermore, in addition to the radiographic changes, intraoperative findings contribute to define the stages. Consequently, a simpler and strictly radiographic three-stage classification was suggested at the 2005 SoFCOT symposium. The stages are focal lucency, attached fragment, and detached fragment, defined based on the bone trabecula abnormalities, irrespective of the condition of the overlying cartilage and viability of the fragment. A more accurate evaluation can now be obtained by MRI [1].

5.1.2. Location and surface area

The Cahill and Berg classification separates the antero-posterior view into five segments, from medial to lateral. On the lateral view, the Harding classification distinguishes anterior (A), middle (B), and posterior (C) sites, based on the Blumensaat line and on the tangent to the posterior femoral cortex (Fig. 3). The surface area is relevant to the prognosis and follow-up and is now more easily measured due to the widespread availability of digitized imaging systems. Finally, skeletal maturity should be assessed and the patient classified as having open or closed physes.

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