Osteochondritis Dissecans of the Knee

Pathoanatomy, Epidemiology, and Diagnosis

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KEYWORDS

• Osteochondritis dissecans • Osteochondrosis • Sports medicine • Knee • Epidemiology

KEY POINTS

- Multiple hypotheses exist regarding the cause of osteochondritis dissecans (OCD); leading theories at this point are those of repetitive microtrauma, disruption of normal endochondral ossification, and genetic factors.
- Male/female ratio is approximately 4:1 and the highest incidence in the United States is seen in the African American population.
- Plain radiographs, especially the flexion notch view, are important in the diagnosis of OCD lesions, whereas magnetic resonance imaging is paramount for OCD lesion characterization.
- Arthroscopy continues to be the gold standard for assessing the stability of OCD lesions.

INTRODUCTION

It has been more than 125 years since German-born, Franz König[1] first described and coined the term osteochondritis dissecans (OCD).[1,2] Since its early characterization, OCD has remained enigmatic and the evolution of its understanding has been slow in the orthopedic community. Confusion about the pathoanatomy and cause of OCD is partially derived from the roots of its etymology. The suffix -itis in

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osteochondritis comes from the Greek root meaning inflammation, despite the current belief that inflammation plays little to no role in the pathoanatomy of OCD. In an attempt to standardize language for discussing OCD lesions, the Research in Osteochondritis Dissecans of the Knee (ROCK) group[3] has defined the term OCD as a focal, idiopathic alteration of subchondral bone with risk for instability and disruption of adjacent articular cartilage that may result in premature osteoarthritis.[4]

Researchers have proposed several hypotheses for the causes of OCD, which have included occult or repetitive microtrauma,[5–8] genetic predisposition and markers,[9–12] inflammatory causation,[1,13] and vascular abnormalities.[14,15] However, despite these hypotheses, there has been no conclusive agreement on the cause. Nonetheless, given its increased incidence in people participating in athletics, a repetitive microtrauma hypothesis is the most popular and can also account for the increased incidence of medial femoral condyle lesions of the knee given the location's proximity to the tibial eminence. However, this hypothesis cannot explain the causal development of OCD in other locations and joints.

Classification of OCD in the knee is broken down by lesion location, characterization of the lesion, status of the overlying cartilage, and skeletal maturity. These variables are elucidated with the use of radiographs, magnetic resonance imaging (MRI), and direct visualization through arthroscopy and/or open arthrotomy in certain cases.

This article provides a detailed review of OCD of the knee, with specific discussions on pathoanatomy, epidemiology, and the diagnosis of OCD.

CAUSES OF OCD

Inflammatory Causes

As previously mentioned, the hypotheses include inflammatory, vascular, trauma/microtrauma, and genetic causes. OCD was first thought by König[1] to have an inflammatory component, hence the suffix itis. Despite describing what König[1] described as "dissecting inflammation," early histologic analysis of loose bodies in OCD suggested that an inflammatory component is unlikely.[16,17]

Vascular/Ischemic Causes

Eighteen years before König[1] coined the term OCD, Sir James Paget[18] described what was later thought to be OCD as "quiet necrosis." In the same vein, Green and Banks[19] also theorized that OCD was caused by ischemia/necrosis of subchondral bone leading to the development of OCD. Researchers in the early twentieth century had several hypotheses for the cause of these ischemic/vascular insults. These hypotheses included emboli from tubercle bacilli,[20] fat emboli,[21] and blood emboli.[22] However, the crux of the hypotheses relied on the supposition that the epiphyseal arterial supply was an end artery construct, which was later shown not to be the case. Moreover, histopathologic analysis of excised OCD specimens has suggested that avascular necrosis is not the cause.[23]

Trauma/Microtrauma

Although both Paget and König discussed trauma in their early works, it was Fairbanks[24] in the early twentieth century who championed the hypothesis of trauma as a cause for OCD. Despite the inability to explain OCD in other joints without clear impaction-type injuries, Smillie[8] strongly supported Fairbanks's[24] so-called tibial spine theory for the cause of OCD. In addition, the work of Cahill[25–27] and Cahill and Berg[28] suggests that earlier sport entry results in juvenile OCD occurring in the weight-bearing portions of the femoral condyle. Perhaps a theory of repetitive

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