Pediatric Knee Osteochondritis Dissecans Lesions

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KEYWORDS

• Cartilage • Chondral • Knee pain • Knee swelling • Sports medicine

KEY POINTS

- Osteochondritis dissecans (OCD) lesion of the knee is a relatively common cause of knee pain in pediatric patients.
- Most pediatric OCD lesions of the knee will heal with nonoperative treatment, which includes a period of rest or activity modification with or without immoblization.
- Surgical treatment is indicated for patients with closed physes or unstable or unsalvageable lesions.
- The goals of surgical treatment include maintenance of articular cartilage congruity, rigid fixation of unstable fragments, and repair of osteochondral defects with cells or tissue that can adequately replace lost or deficient cartilage.
- High-quality evidence for the optimal evaluation and management of pediatric OCD lesions remains sparse, and continued research is needed.

INTRODUCTION

Osteochondritis dissecans (OCD) of the knee can be a source of pain and dysfunction in pediatric patients. The estimated incidence of OCD ranges from 9.5 to 29 cases per 100,000 population.[1–3] Boys have an approximately fourfold increased incidence of OCD of the knee compared with girls.[3] Most lesions are found within the distal femur, and the most common site is the lateral aspect of the medial femoral condyle.[3,4] OCD is an acquired condition of articular cartilage and subchondral bone that initially manifests itself as a softening of the overlying cartilage (Fig. 1). Without treatment, this can progress to articular cartilage fissuring, separation, partial detachment, and eventually, osteochondral separation.[5–9] OCD of the knee can be subcategorized based on the status of the distal femoral physis. Juvenile OCD occurs in patients with open physes and has a much better prognosis than adult OCD. Greater than 50% of juvenile OCD cases will show healing within 6 to 18 months with nonoperative treatment, whereas patients with adult OCD frequently require operative intervention.[10]

HISTORY AND ETIOLOGY

The etiology of OCD remains unclear, and no theory regarding its cause is universally accepted.[11] Theories on etiology include inflammation, ischemia, ossification abnormalities, genetic factors, and repetitive microtrauma.[11–14] In 1887, König suggested an inflammatory etiology, coining the term "osteochondritis dissecans."[15]

Disclosure Statement: The authors have no relevant disclosures.

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Orthop Clin N Am 47 (2016) 763–775

http://dx.doi.org/10.1016/j.ocl.2016.05.001

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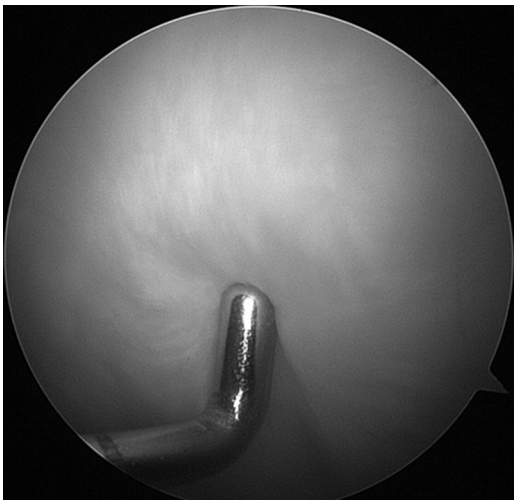

Fig. 1. Arthroscopic photo showing articular cartilage softening found in OCD lesions. (*Courtesy of* Theodore J. Ganley, MD, Philadelphia, PA.)

Further study, however, did not support inflammation as the primary cause of OCD. Ribbing attributed abnormalities in ossification within the distal femoral epiphysis as a cause.[16] A possible vascular etiology has been proposed, with relative ischemia and subsequent necrosis being important components in the development of OCD.[17,18] Other studies, however, have failed to definitively identify avascular necrosis of the OCD fragment or find a relative ischemic watershed area of the lateral aspect of the medial femoral condyle (the most common location of OCD lesions).[19–22] More recent research in the development of OCD lesions in animals has focused on the role that vascular architecture may play in the development of OCD.[23–25] Both pigs and people develop OCD lesions in similar anatomic regions, and the vascular anatomy in both species demonstrates that both species have similar vascular architecture, which may predispose them to the development of OCD of the femoral condyles.[23] This research suggests that vascularity may play a role in the development of OCD in people.

A genetic predisposition for the development of OCD has also been proposed. Several cases of monozygotic twins with OCD lesions have been described,[26–31] as well as large reports of OCD lesions within the same family.[13,14,32] Additionally, there are several genetic diseases that are associated with the development of OCD lesions. Patients with Stickler syndrome have been found to have multiple OCD lesions.[30] OCD lesions are also commonly reported in association with dwarfism.[33–37]

Mechanical factors such as malalignment[38] or repetitive microtrauma[39] have also been implicated in the development of OCD. The frequent occurrence of OCD lesions in patients who are involved in activities and sports with repetitive impact as well as the association with abnormal meniscal anatomy help support the concept of altered knee biomechanics as a cause of OCD.[40–42] This theory holds that OCD occurs as a result of an initial stress reaction, which may then progress to a stress fracture of the underlying subchondral bone. With progressive repetitive loading, the stress fracture fails to heal, and the subchondral bone becomes necrotic,[12] causing the fragment to eventually dissect and separate from the fracture bed.

Although the precise etiology of OCD is unclear, it is known that if these lesions are left untreated and fail to heal appropriately, they have a high potential of contributing to the development of future osteoarthritis.[43,44]

BIOLOGY OF ARTICULAR CARTILAGE AND SUBCHONDRAL BONE

Understanding the anatomy and morphology of subchondral bone is important to adequately evaluate and manage conditions that affect the subchondral bone such as OCD.[45–47] The subchondral zone or the subchondral bone plate refers to the cortical endplate lying adjacent to the calcified zone of the articular cartilage with its accompanying subarticular spongiosa (Fig. 2). The cement line separates the calcified zone from the subchondral bone plate, with the thickness of the subchondral bone plate varying depending on the joint.

The architecture of the subchondral bone plate consists of 2 mineralized layers, which together form a single unit, separating the articular cartilage from the bone marrow. There is a discrete band of calcified cartilage on the articular side of the subchondral bone plate. This band appears as the tidemark on hematoxylin and eosin histologic staining. The tidemark is a complex 3-dimensional structure. The tidemark is significant, because it represents the mineralization front and is a transition zone between 2 dissimilar regions of cartilage[45] (see Fig. 2). The tidemark separates the type II collagen fibrils of the articular cartilage from the type I collagen found deeper (away from the joint surface). The tidemark has significant biomechanical functions and changes in response to microinjury. There is evidence that collagen fibrils cross the tidemark, resulting in a strong link