Nonoperative Treatment of Osteochondritis Dissecans of the Knee

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KEYWORDS

• Osteochondritis dissecans • Juvenile osteochondritis dissecans
• Nonoperative treatment • Open physis • Stable lesions • Activity modification
• Return to play

KEY POINTS

- Osteochondritis dissecans is potentially devastating cause of knee pain in adolescents and adults.
- Prognosis and treatment is dependent on the stability of the lesion and the age of the patient. Skeletally immature patients with stable lesions are amenable for nonoperative treatment.
- Nonoperative treatment is less predictable in skeletally mature patients and patients with unstable lesions.
- Lesion size, location, and stability, along with symptomatology, should all be considered before initiating treatment.
- Modalities of nonoperative treatment can range from activity modification to complete immobilization. Close follow-up is recommended to monitor healing progression and symptom resolution.

INTRODUCTION

Osteochondritis dissecans (OCD) is an infrequent but potentially devastating cause of knee pain in adolescents and adults. First described by Paget in 1870, OCD involves a focal, idiopathic alteration of subchondral bone with risk for instability and disruption of adjacent articular cartilage that may result in premature arthritis.[1] Prognosis and treatment is dependent primarily on the stability of the lesion and the age of the patient. Although juvenile OCD generally presents as a stable lesion amenable to

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conservative treatment, symptomatic adult OCD lesions have diminished healing potential, are frequently unstable, and often require operative intervention. Numerous publications regarding the etiology, natural history, and treatment of OCD have been put forth; however, a general consensus remains elusive.

ETIOLOGY

The subchondral bone is the primary site of pathology in OCD lesions. The term osteochondritis dissecans, coined by Konig in 1888, is now recognized as a misnomer, as no evidence of an inflammatory process has ever been demonstrated in these lesions. Necrosis in the subchondral bone is a frequent but inconsistent histologic finding and the overlying articular cartilage is often normal.[2,3] Absent in the literature is a consensus as to whether subchondral bone necrosis is primary or secondary to the progression of OCD. These issues remain extremely relevant to the successful management options reviewed here.

The cause of OCD remains unknown, with several etiologies, including genetic predisposition, defective skeletal development, vascular insult, and trauma proposed in the literature. Usually no single causative factor can be identified and the true etiology is likely multifactorial. In discussing OCD lesions, one must be cautious to differentiate between the idiopathic type (topic of this review) and lesions resulting from osteonecrosis secondary to other factors, such as hemoglobinopathies, steroid use, and chemotherapy.

Familial inheritance has been suggested in several small studies involving multiple family members with OCD lesions.[4] In many of these familial cases, an association between dwarfism and short stature has been reported.[4–6] A sporadic form also has been identified, with no evidence of genetic predisposition or association with short stature.[7]

Ischemia was traditionally believed to be the primary cause of OCD lesions.[1] Issues such as emboli from blood or fat and/or disorders in subchondral vascular anatomy were felt to result in relative ischemia of the subchondral bone and subsequent subchondral fracture with fragment formation.[1,8] Trauma, specifically repetitive trauma, is currently the most accepted causative factor of OCD lesions. The association between OCD lesions and patient activity level supports this theory. In a study of more than 105 patients with OCD lesions involving the knee, 60% reported high-level participation in athletic activity.[9,10] The prevalence of OCD lesions has been shown to coincide with increased sports participation.[10] Repetitive trauma is believed to induce a stress reaction and subsequent stress fracture within the subchondral bone. In the setting of continued trauma or impaired healing capacity, fractured subchondral bone undergoes necrosis, leading to separation between the bone and overlying articular cartilage.

Altered joint mechanics also have been proposed as a factor contributing to the development of OCD lesions.[1,11] Lesions of the medial femoral condyle have been associated with varus alignment of the knee, whereas lesions of the lateral femoral condyle are more often seen in patients with valgus alignment.[12] The development of OCD lesions in the lateral femoral condyle also has been reported following saucerization of a discoid lateral meniscus.[13]

EPIDEMIOLOGY

The incidence of OCD is approximately 15 to 29 per 100,000 patients.[10,14] A steady rise in this number is likely secondary to increased provider awareness, more frequent use of advanced imaging, such as magnetic resonance imaging (MRI), introduction of