Osteochondritis dissecans (OCD) involves partial or complete detachment of a fragment of articular cartilage and subchondral bone from a joint surface. OCD was originally thought to be caused by trauma, but other etiologies have been postulated, such as isch-emia, an inflammatory condition, impaired ossification, and genetics. Other causes have been suggested in younger patients, such as an epiphyseal abnormality or endocrine disturbance. OCD occurs in various locations, including the talus, patella, capitellum, femoral head, distal tibia, and wrist. The most common location for OCD is the lateral aspect of the medial femoral condyle. A possible reason that this area is vulnerable may be its contact with the odd facet of the patella in full flexion. The condition rarely affects the trochlea. However, trochlea involvement has been reported, with one case involving an ancient specimen in a museum. Only four cases of bilateral OCD of the trochlea have been reported. We report a case of bilateral OCD of the trochlea, which involved two injuries that occurred within three months of one other.

Case History

A 16-year-old female high school varsity basketball player sustained an injury to her left knee during a basketball camp. She planted her left foot while sprinting and immediately felt a “pop” in her left knee. She had immediate antero-medial pain and swelling. She remained at the camp, but was not able to perform normal activities due to sharp and stabbing pain. Her symptoms were relieved by rest, elevation, ice, and nonweight bearing ambulation. She did not experience joint locking, but felt frequent clicking. No previous knee injury was reported. Her knee injury was not evaluated by an athletic trainer or a physician at the time of the injury.

Due to continued pain, swelling, and clicking, the athlete saw her family physician approximately 10 days after returning from the basketball camp. The physician ordered an MRI and referred her to an orthopedic surgeon. When evaluated by the orthopedic surgeon one week later, her knee motion was limited to 20 degrees of flexion and 130 degrees of extension, without patello-femoral crepitus. She had nonspecific tenderness in response to palpation of the medial joint line and the anterior aspect of the medial femoral condyle, but no tenderness was elicited by palpation over the medial retinaculum or the medial patello-femoral ligament. The patellar...
Grind test was positive, but she exhibited normal and symmetric patellar mobility without apprehension. Lachman’s test, valgus and varus stress tests, and the pivot-shift test were negative. Hip, ankle, and foot functions were normal. Because the patient’s family physician had ordered an MRI in advance of her visit to the orthopedic surgeon, no radiographs were obtained. The MRI revealed an osteochondral defect on the anterolateral aspect of the trochlea, without evidence of a reciprocal lesion on the patella (Figure 1).

Arthroscopic surgery was performed approximately 24 days after the initial injury. A 10 mm × 15 mm loose body with bony fragments was removed, and a chondral defect was identified on the lateral aspect of the femoral trochlea (Figure 2), which articulated with the patella at 30 degrees of flexion. The base of the lesion was debrided and a microfracture procedure was administered (i.e., a sharp awl was used to create holes in the bone that created a bleeding bed within the lesion).9,10

Due to the uniqueness of the injury, a conservative approach to postsurgical management was prescribed to allow healing of the lesion site. The athlete was initially placed in a knee immobilizer for 3 weeks to avoid patella contact with the site of the lesion, and weight-bearing was permitted as tolerated.10 Continuous passive motion was performed at home, which was subsequently progressed to active and active-assistive ROM with a therapist. At 6 weeks postsurgery, the athlete had full knee ROM. A patello-femoral rehabilitation program was initiated in an effort to minimize contact pressure on the microfracture site (Table 1).10,11

At 3 months postsurgery, the athlete rose from a seated position and experienced sudden pain in her contralateral (right) knee. The pain was similar to that which she experienced when she injured her left knee. An immediate effusion developed, and she was unable to fully extend her knee. One week later, physical examination by an orthopedic surgeon identified a mild-to-moderate suprapatellar effusion, without signs of patellar instability or ligamentous injury. An anteroposterior radiograph demonstrated the presence of a loose body in the intercondylar notch (Figure 3). Arthroscopic surgery was performed on her right knee approximately one week after the onset of symptoms. Two loose bodies (13 mm × 10 mm and 10 mm × 5 mm) were removed. A chondral defect was visualized on the supero-lateral portion of the trochlea (Figure 4). A microfracture procedure was performed as previously described, and the same rehabilitation program used earlier for the left knee was administered.9-11

The athlete returned to sports at 6 months after the second surgical procedure. She had full ROM and normal physical examination findings for both knees (27 weeks after the first surgical procedure and 24 weeks after the second surgical procedure). She was completely asymptomatic and capable of performing all activities at full speed.
Discussion

OCD frequently occurs on the lateral aspect of the medial femoral condyle, and only 2% of cases involve the lateral trochlea.\(^2,6,7,12,13\) Including this report, a total of only five cases of bilateral OCD of the trochlea have been reported in the literature.\(^7\) The classic description of OCD was first reported by Paget in 1870.\(^7\) Several possible etiologies of OCD have been proposed, most of which involve trauma.\(^1,4,6,7,12,13\) Aichroth\(^7\) described two trochlear lesions that were believed to have been related to a patellar dislocation. The patient in this report demonstrated no signs of patellofemoral malalignment or instability. Others attribute OCD lesions to ischemic necrosis, disturbance of the normal ossification process, and genetic susceptibility.\(^5,13\)

The patient’s left knee lesion reported in this case was associated with running deceleration. While recovering from surgery for management of the left knee lesion, she had a sudden onset of the same symptoms in her right knee without having experienced a traumatic event. Some atraumatic etiology may have predisposed her to OCD, which may have been associated with repetitive minor trauma, shear forces, overuse, weak articular cartilage, or a congenital malformation. The possibility that repetitive trauma may induce a stress reaction in the underlying subchondral bone has been suggested.\(^14\) When stress

<table>
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<tr>
<th>Left Knee Rehabilitation Progression</th>
<th>Right Knee Rehabilitation Progression</th>
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<tr>
<td><strong>Weeks 0–3</strong>: In knee immobilizer, weight bearing as tolerated. CPM was utilized at home beginning with 0–30 degrees with 10 degrees daily flexion increments as tolerated.</td>
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</tr>
<tr>
<td><strong>Weeks 4–5</strong>: Progressed to active/active assistive ROM with a therapist.</td>
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<tr>
<td><strong>Weeks 6–7</strong>: Began isometric exercises, patellar joint mobilization, SLR, BAPS board, UBE, core exercises, and treadmill walking.</td>
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</tr>
<tr>
<td><strong>Weeks 8–9</strong>: Began short arc quads and continued PRE weeks 6–7 exercises.</td>
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</tr>
<tr>
<td><strong>Weeks 9–12</strong>: Began and progressed isoinertial closed kinetic chain exercises, resisted running.</td>
<td><strong>Weeks 9–12</strong>: Began and progressed isoinertial closed kinetic chain exercises, resisted running.</td>
</tr>
<tr>
<td><strong>Weeks 12</strong>: Injured contralateral knee – discontinued current rehabilitation.</td>
<td><strong>Weeks 12–16</strong>: Began and progressed running and continued PRE closed chain.</td>
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<tr>
<td><strong>Weeks 16–24</strong>: Began and progressed agility drills and plyometrics.</td>
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Loading exceeds the subchondral bone’s ability to heal, necrosis of the fragment may occur, with fragment dissection, separation, and progression to non-union. This mechanism is supported by histologic studies that have shown OCD lesions to have a subchondral area of bone necrosis, which is replaced by cartilage that lacks bone trabeculae. The information presented in this case report is consistent with others that have not involved a traumatic event. The cumulative stress imposed on subchondral bone by exercise or sport participation may lead to vascular compromise that ultimately produces an OCD lesion.

The patient’s left knee presentation was similar to that of another published report of repetitive minor trauma that resulted in bilateral OCD lesions. The 15-year-old female basketball player whose case we report began to have simultaneous bilateral knee pain at the age of 12. Her basketball career had begun at a very young age. Kocher et al have suggested that an increasing frequency of OCD in young patients may be caused by increasingly competitive sports participation at an early age. Patello-femoral joint mechanics could play a role, because an association between axial alignment and the location of OCD has been reported.

Conclusion

The reported case of OCD is unique in terms of lesion location, symptom development, and bilateral involvement. Although the patient did not report any anterior knee pain prior to the onset of OCD symptoms, adolescent athletes who report anterior knee pain should be examined to rule out OCD. MRI may reveal the presence of an OCD lesion in a patient with a history of refractory anterior knee pain.

References


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