ELSEVIER

Operative Techniques in

Sports Medicine

Arthroscopic and Open Surgery for Osteochondritis Dissecans

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Osteochondritis dissecans lesions of the elbow occur in 3.4% of adolescent baseball players, and the lesions will inevitably progress without intervention. Fifty percentage of patients who initially presented with advanced lesions had decreased range of motion and pain with activities of daily living. Patients treated early have high return to sports rates. After radiographs are obtained, patients suspected of having an osteochondritis dissecans lesion in the elbow should undergo a magnetic resonance arthrogram to determine the integrity of the cartilage cap. Central lesions with an intact cartilage cap can often be successfully treated with bracing and a course of formal therapy. Patients who fail conservative treatment with a violated cartilage cap can undergo microfracture or fragment fixation. Patients with lesions that extend to the lateral wall require an autograft transfer to restore the structural integrity of the capitellum and prevent instability.

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Definition

(1) Osteochondritis dissecans (OCD) is a noninflammatory lesion in which an area of discrete articular surface begins to separate from the subchondral bone.[1,2]
(2) Within the elbow, OCD lesions most commonly occur in the capitellum, but they have been reported in the radial head, olecranon, olecranon fossa, and trochlea.[3]

Anatomy

(1) The overhead throwing motion generates significant valgus and extension moments in the elbow, which causes tension across the medial side of the elbow and compression laterally.[4] The anterior bundle of the ulnar collateral ligament is the primary restraint to valgus force from 30°-120° of flexion—the flexion range in which overhead throwing occurs. The anterior bundle tries to reduce forces as it resists the valgus moment.[1,4]
(2) Two terminal arteries branching off the radial recurrent and interosseus recurrent arteries provide the blood supply to the epiphysis of the capitellum. The area lacks collaterals or metaphyseal blood supply.[5,6]

Pathogenesis

(1) The late cocking and early acceleration phases of throwing generate significant valgus moments that cause repetitive radiocapitellar compression.
(2) Repetitive valgus loads during the late cocking and early acceleration phase of throwing generates compression and, more importantly, shearing forces at the radiocapitellar joint.[1] These loads result in stress fractures in the subchondral bone, which initiate the progressive separation of the articular surface from the subchondral bone.[5]
(3) The presence of lateral capitellar defects, in contrast to central defects, actually increases radiocapitellar contact

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pressures. As the contract pressures increase the lesion progresses and, in turn, the contact pressures further increase. This sequence creates a constant cycle of lesion progression.[1]

(4) As described earlier, the blood supply to the capitellum is limited, which limits its ability to recover from repetitive insult.[7]

Natural History

(1) OCD lesions in the capitellum are not self-limiting. In the absence of intervention, the natural history of these lesions is to progress in severity.
(2) Most patients wait until the lesions have progressed to seek medical attention. As the lesions progress they become more difficult to treat.[8]
(3) If diagnosed in the early stages then these lesions have a chance to heal. By treating them with strict rest, Mihara et al reported that 88% of their patients with early signs of an OCD lesion went on to heal.[9]
(4) The natural history of OCD lesions that are diagnosed in the late stages is much worse. Studies have reported that at long-term follow-up 50% of patients with advanced lesions at the time of diagnosis experienced decreased range of motion and symptoms that impaired activities of daily living.[10,11]

Patient History and Physical Examination

(1) Patients typically complain of progressive, generalized elbow pain that is relieved by rest.[12]
(2) Patients often report stiffness associated with a loss of terminal extension.
(3) Physical examination.
  (a) Tenderness to palpation at the radiocapitellar joint.
  (b) Tender posterolateral plica.
  (c) Positive active radiocapitellar compression test.[13]
  (d) Loss of terminal extension.

Imaging

(1) Anteroposterior, lateral, 2 obliques, and an anteroposterior view with elbow in 45 degrees of flexion to better visualize the joint.[6,14,15]
  (a) Images will show a discrete area of rarefaction and radiolucency (Fig. 1).[14]
(2) Magnetic resonance (MR) arthrogram (Fig. 2).
  (a) An arthrogram is important to determine the viability of the cartilage cap.
  (b) Early lesions demonstrate low signal intensity on T1-weighted images and appear normal on T2-weighted images.
  (c) Late lesions show high signal intensity on both T1 and T2-weighted images.[3]

Figure 1 An AP radiograph of a right elbow demonstrating an osteochondritis dissecans lesion of the capitellum. Note the area of rarefaction and radiolucency. AP, anteroposterior.

  (d) Lesions in which the cartilage cap has been violated will show a high intensity signal behind the lesion, which indicates that fluid has been able to get behind the fragment.[16,17]

Nonoperative Management

(1) Indicated as the initial management of central lesions with intact cartilage caps as seen on MR arthrogram.
(2) The patient is placed in a hinged elbow brace and may continue to participate in his or her sports as long as he or she plays in the brace.
(3) The patient is started in a formal therapy program.
(4) Continue the brace until the patient can participate in sport without pain.

Techniques

Surgical Indications

(1) Continued pain and dysfunction despite a course of bracing.
(2) An unstable cartilage cap as seen on MR arthrogram.
(3) Lesions extending to the lateral wall.