

Elbow Arthroscopy: Capitellar Osteochondritis Dissecans and Radiocapitellar Plica

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Abstract

The combination of excessive radiocapitellar compressive forces and the limited vascularity of the capitellum are responsible for the development of osteochondritis dissecans. Repetitive compressive forces are generated by throwing or racket swinging motions or from constant axial compressive loads on the elbow, which are common in athletes such as gymnasts. Symptoms include activity-associated pain and stiffness. Physical examination findings show tenderness over the radiocapitellar joint and, commonly, loss of extension. Plain radiographs may show flattening and sclerosis of the capitellum, lucencies, and possibly intra-articular loose bodies. MRI can detect bone edema early in the disease process and further delineate the extent of the injury. The management of osteochondritis dissecans lesions is primarily based on the demands of the patient, the size and location of the lesion, and the status and stability of the overlying cartilage. Possible treatments include transarticular drilling; removing detached fragments or loose bodies, followed by drilling; and mosaicplasty.

Radiocapitellar plica can cause chondromalacic changes on the radial head and capitellum, with symptoms including painful clicking and effusions. Arthroscopic plica resection is indicated when nonsurgical treatment fails.

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The radiocapitellar compartment of the athlete's elbow is subject to significant stresses during repetitive activities, such as throwing, or upper extremity weight-bearing sports, such as gymnastics.¹ Radiocapitellar compression can lead to Panner disease in pre-adolescent children or capitellar osteochondritis dissecans (OCD) in adolescents or young adults; it is important to differentiate these two disorders because they have different natural histories and treatments.²⁻⁵ Elbow arthroscopic techniques can be used to treat the OCD lesion with débridement, drilling, and even mosaicplasty and can be used to treat loose bodies that develop secondary to the OCD lesion.

Panner Disease

Panner disease predominantly affects boys younger than 10 years.⁶ Patients initially present for treatment with reports of activity-related pain and stiffness in the elbow. Tenderness over the lateral elbow and the capitellum is found on physical examination. Radiographs initially show fissuring, lucencies, fragmentation, and irregularity of the capitellum. Subsequent

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Figure 1 AP radiograph showing an OCD lesion (arrows) in the capitellum of the elbow. (Reproduced with permission from Ahmad CS, EIAttrache NS: Treatment of capitellar osteochondritis dissecans. *Tech Shoulder Elbow Surg* 2006;7:169-174.)

radiographs show larger radiolucent areas followed by reossification, with a corresponding resolution of symptoms. One to 2 years after the initial presentation, the epiphysis regains its normal contour and appearance.⁴ MRI scans typically show edema localized to the chondral surface, with less involvement of the subchondral bone in comparison with OCD.

Treatment involves ceasing the activities causing elbow stress, and the use of ice and anti-inflammatory medication. For severe symptoms, the elbow may be immobilized for 3 to 4 weeks. In general, symptoms usually resolve within 6 to 8 weeks, although they occasionally persist for months. Activities are resumed as tolerated. Panner disease has an excellent long-term prognosis, although some patients may experience loss of motion.^{4,6}



Figure 2 MRI scan showing capitellar OCD lesion (white arrow) with and associated loose body (black arrow). (Adapted with permission from Ahmad CS, EIAttrache NS: Treatment of capitellar osteochondritis dissecans. *Tech Shoulder Elbow Surg* 2006;7:169-174.)

Osteochondritis Dissecans

OCD of the capitellum is characterized by noninflammatory degeneration of subchondral bone occurring in the context of repetitive loading to the lateral compartment of the elbow. Panner disease and OCD may represent two different stages of the same disorder, but they differ in the patient's age at onset and natural history.⁴ Panner disease affects children younger than 10 years, whereas OCD commonly affects older athletes.⁷ OCD is not always self-limiting; if untreated it may result in profound destruction of the capitellum.⁷

Etiology

The combination of abnormal radiocapitellar compressive forces and the limited vascularity of the capitellum supplied by end arteries are likely responsible for the development of OCD.^{2,5,8,9} Repetitive compressive forces are generated by either large valgus stresses on

the elbow during throwing or racket swinging or from constant axial compressive loads on the elbow, such as those experienced by gymnasts.^{7,10,11}

The capitellum is supplied by two end arteries coursing from posterior to anterior, which are branches of the radial recurrent and interosseous recurrent arteries.¹² Local blood flow to the capitellum may be disrupted by both repetitive microtrauma or a single traumatic event leading to subchondral bone injury.^{13,14}

Presentation

Patients with OCD will initially present reporting activity-related pain and stiffness in the elbow. Mechanical symptoms of locking or catching, caused by intra-articular loose bodies, may be present. The physical examination shows tenderness over the radiocapitellar joint. Loss of range of motion with a 15° to 20° flexion contracture is common. The active radiocapitellar compression test suggests an OCD lesion when pain is elicited in the lateral compartment of the elbow and when the patient pronates and supinates the forearm with the arm in extension.

Imaging

Full-extension AP, 45°-flexion AP, and lateral radiographic views of the elbow should be obtained; however, results may be negative early in the disease process (**Figure 1**). As the condition progresses, flattening and sclerosis of the capitellum, typically on its anterolateral aspect, will become apparent. Irregular areas of lucency and intra-articular loose bodies may be seen. It should be noted if the capitellar physis is open or closed. In patients with suspected OCD, an MRI scan of the elbow should always be obtained (**Figure 2**). MRI can detect bone edema early in the disease process.¹⁵ A magnetic resonance arthrogram can further

Table 1
Classification and Treatment of Capitellar Osteochondritis Dissecans Lesions

Stability	Stage	Radiographic Findings	Arthroscopic Findings	Treatment
Stable lesion	I	Normal radiographs T1-weighted MRI: abnormal T2-weighted MRI: normal	Intact articular cartilage Subchondral bone edema but structurally sound	Hinged elbow brace Physical therapy Nonsteroidal anti-inflammatory drugs Follow-up radiograph and/or MRI at 3 to 6 months
Unstable lesion	II	Abnormal radiographs T1- and T2-weighted MRIs: abnormal Contrast shows margin around the lesion	Partially detached fragment Cartilage fracture Subchondral bone collapse Lateral buttress involved; poorer prognosis	Acute: Consider fragment fixation but higher success using treatment for chronic Chronic: (a) < 6 to 7 mm lateral buttress involved/radial head does not engage: fragment removal plus microfracture drilling (b) > 6 to 7 mm lateral buttress involved/head engages: removal plus osteochondral allograft/synthetic graft
	III	Loose bodies Associated radial head deformity	Completely detached loose bodies Any of the above	Loose body removal Treat as stage II lesion < 30% radial head involvement: treat as stage II lesion > 30% radial head involvement: no osteochondral grafting; microfracture/drilling are OK

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delineate the extent of the injury because the contrast agent can reveal separation of a detached or partially detached fragment from the subchondral bone.

Management

The management of OCD lesions is based primarily on the demands of the patient and the status and stability of the overlying cartilage. The size and location of the lesion and the status of the capitellar growth plate also influence treatment.¹⁶⁻¹⁸ Several classification systems based on radiographic and arthroscopic findings have been proposed;^{16,19,20} however, none has been universally adopted because the systems are cumbersome to use.^{21,22}

Table 1 shows a simplified, succinct, three-stage classification system that provides a template for managing capitellar OCD lesions.

Stage I

In stage I lesions, the osteochondral fragment is intact, stable, and nondisplaced. Radiographic findings are often negative. The signal findings on MRI are variable, typically abnormal on T1-weighted MRIs and normal on T2-weighted MRIs, although the T2 signal may also be abnormal. Arthroscopy shows that the articular cartilage is intact, and subchondral stability is generally preserved. Treatment is non-surgical and includes resting the elbow in a hinged elbow brace for 3 to 6 weeks. Progressive physical therapy should ensue as symptoms abate. Return to sports activities usually can be expected at 3 to 6 months. Follow-up radiographs and MRIs should be obtained at 2- to 3-month intervals to track progress. If symptoms return, an additional rest period is mandated. With persistent refractory symptoms,

pitchers may have to change throwing positions, and gymnasts may need to elect a different sport.

Stage II

In stage II OCD of the capitellum, the osteochondral fragment is partially separated as documented both radiographically and arthroscopically. Radiographs will show fissuring, lucencies, and fragmentation. On MRI, both T1- and T2-weighted sequences will show abnormal signals and a margin around the fragment, denoting its instability. CT scans may also show the partially separated fragment. Arthroscopic findings include a broken cartilage surface and unstable and partially displaced subchondral bone. Surgical treatment is needed to return athletes to their sports activity or allow resumption of the activities of daily living as soon as possible.

The size and location of the lesion govern treatment. For smaller lesions, débridement is an option. Patients typically have immediate relief of symptoms; however, early arthritis is associated with the long-term natural history of the disease. Fragment fixation has been advocated by some physicians for stage II lesions, although the healing potential of fixed fragments and the clinical results of the procedure are often unpredictable.^{19,20,23,24} Osteochondral autografts or synthetic grafts can be used to treat large defects that engage the radial head and involve the lateral buttress of the capitellum.

This chapter's authors believe that the location of the lesion may be more important than size in guiding treatment. A lesion that extends into the lateral margin of the capitellum, as described by Ruch et al,²⁵ is associated with a potentially poorer prognosis. The lateral column of the capitellum supports large compressive forces when the elbow is stressed in valgus or with axial loading. Lesions that do not involve a significant portion of the lateral buttress of the capitellum and that do not engage the radial head on arthroscopic examination (pronation and supination with the elbow in extension) have been successfully treated with microfracture or drilling. In this situation, the defect is relatively protected, and healing (predominantly fibrocartilage healing) may occur.

Conversely, lateral column involvement of more than 6 to 7 mm cannot be acceptably treated with microfracture. In this instance, the absence of a lateral buttress allows engagement of the radial head in the defect, which inhibits healing and may lead to accelerated radiocapitellar arthrosis. For these larger, engaging defects or those that extend substantially (more than 6 to 7 mm) into the lateral buttress, this chapter's authors recommend removing the loose fragment and achieving osteo-

chondral restoration by means of mosaicplasty or an osteochondral autograft transfer. If there are partially detached fragments, the detached portion (usually central) should be débrided centrally to laterally. Once stable osteochondral borders have been obtained, the lesion is carefully evaluated arthroscopically to ascertain the extent of the lateral column involvement and determine if the radial head is engaged within the defect. Lesions larger than 1 cm² (mean, 1.32 cm²) with no lateral column involvement were treated successfully with microfracture, whereas those involving the lateral column did well with osteochondral grafting (JD Chappell, MD, and NS ElAttrache, MD, unpublished data presented at the American Orthopaedic Society for Sports Medicine Annual Meeting, Orlando, FL, 2008). Fragment fixation is also an option, but this chapter's authors have had superior and more consistent results with grafting.

Stage III

In stage III capitellar OCD, the fragment is fully displaced and has become a loose body. Patients may present with mechanical symptoms related to loose bodies, such as locking. In this stage of the disease, débridement, drilling, or osteochondral replacement is indicated. If the loose osteochondral fragment is shown to be acutely displaced in a patient with previously documented OCD, fixation to its donor site can be attempted; however, fixation results are inconsistent.²⁴ Chronic loose bodies (documented by serial radiographs or MRI) should be removed, and the donor bed should be débrided in preparation for one of the previously described treatment options, following the same algorithm.

Radial Head Involvement

Degenerative changes in the radial head, in addition to capitellar pathology, indi-

cate advanced disease and do not generally occur in athletes. If the radial lesion is less than 30% of the size of the radial head, treatment of the capitellar OCD should proceed as previously described. For radial lesions greater than 30% of the size of the radial head, treatment of the capitellar lesion should be limited to débridement, drilling, and microfracture.⁶ Severe radiocapitellar degenerative arthritis is a relative contraindication to mosaicplasty.

Surgical Technique

Although alternate surgical patient positioning is possible, the supine position is preferred by the senior author (NSE) because it facilitates general anesthesia, provides an easy conversion to an open procedure if needed, and orients the elbow in an anatomically familiar way. Standard arthroscopic portals are created beginning with the diagnostic arthroscopic procedure in the anterior compartment. The anterior capitellum is inspected and is most often normal, with the pathology residing out of view on the more posterior aspect. In throwing athletes, a valgus stress test with the elbow flexed to 70° can be performed while visualizing the medial ulnohumeral joint. A 1- to 2-mm opening indicates pathologic laxity of the medial collateral ligament, although clinical correlation is mandatory. Diagnostic arthroscopy then proceeds in the posterior compartment, with standard portals to assess for loose bodies and other pathology. Detached OCD fragments are often located in the olecranon fossa.

To visualize the posterior capitellum, a midlateral portal (lateral soft spot) is created in line with the lateral epicondylar ridge and entered with the arthroscope (**Figure 3**). This area often is better suited to a small 2.7-mm arthroscope. The radial head, the capitellum, the trochlear notch, and the trochlear ridge are best seen through

this portal. Care should be taken to avoid injury to the posterior antebrachial cutaneous nerve located near this portal. A working portal is created adjacent and slightly ulnar to the midlateral portal. A cadaver study showed that carefully placed, dual, direct lateral portals do not damage lateral ligaments and provide excellent exposure to the capitellum.²⁶ A thickened radiocapitellar plica is occasionally found in patients with OCD and lateral compartment symptoms; if found, the plica should be resected.

The OCD lesion is then visualized and graded. Grade II and III lesions are treated by removing any loose fragments, shaving loose fragments of cartilage down to subchondral bone, and establishing healthy cartilage borders (**Figure 4**). The size of the lesions is determined using a calibrated probe. The ability of the capitellum to buttress the radial head is determined. At this point, specific procedures are performed as indicated.

Microfracture and Subchondral Drilling

Stage I lesions with cartilage fibrillation and fissuring are treated with transarticular drilling with a small 0.045-inch Kirschner wire. For stage II and III lesions, detached fragments or loose bodies are removed. With the arthroscope in the direct lateral portal, a 0.045-inch or 0.062-inch Kirschner wire is inserted through the accessory lateral portal and used to create vascular channels in the lesion separated by 2 mm (**Figure 5**). Multiple holes are made in the lesion, and efflux of marrow elements is observed to induce a fibrocartilage healing response. Excellent results at 3-year follow-up were obtained in 11 patients treated with this technique; all returned to their previous activity level (JD Chappell, MD, and NS ElAttrache, MD, unpublished data presented at the American



Figure 3 Anterolateral (white arrow) and midlateral (black arrow) portals (soft-spot portals) are outlined. (Reproduced with permission from Ahmad CS, ElAttrache NS: Treatment of capitellar osteochondritis dissecans. *Tech Shoulder Elbow Surg* 2006;7:169-174.)

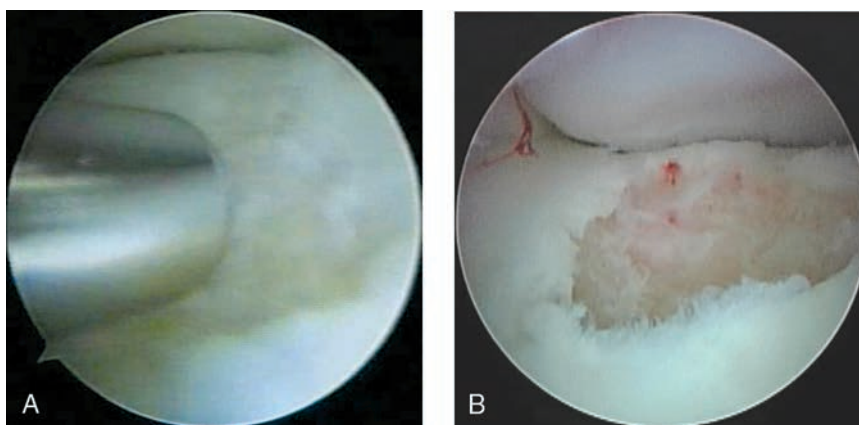


Figure 4 A shaver is used to débride an OCD lesion (A) to stable borders (B). (Reproduced with permission from Ahmad CS, ElAttrache NS: Treatment of capitellar osteochondritis dissecans. *Tech Shoulder Elbow Surg* 2006;7:169-174.)

Orthopaedic Society for Sports Medicine Annual Meeting, Orlando, FL, 2008). The size of the OCD lesions ranged from 7 × 6 mm to 17 × 15 mm.

Mosaicplasty

In mosaicplasty, small-size osteochondral grafts are obtained arthroscopically from the knee at the lateral periphery or the trochlear edge of the

femoral condyles and transplanted into prepared osteochondral defects on the capitellum.²⁷ Mosaicplasty is indicated when a large capitellar lesion engages the radial head, as observed while rotating the extended arm during arthroscopy, or when there is significant (more than 6 to 7 mm) lateral column involvement. Radial head degeneration and severe deformities of



Figure 5 Microfracture technique. **A**, A 0.062-inch Kirschner wire (arrow) is inserted through the accessory midlateral portal and used to perforate the lesion. **B**, Holes (arrows) in the lesion allow marrow elements to produce a fibrocartilage healing response. **C**, The bleeding response from the drilling visualized after the tourniquet is deflated. (Reproduced with permission from Ahmad CS, ElAttrache NS: Treatment of capitellar osteochondritis dissecans. *Tech Shoulder Elbow Surg* 2006;7:169-174.)



Figure 6 Osteochondral plug reconstruction of a capitellar OCD lesion. **A**, The OCD recipient site is created. **B**, The donor osteochondral graft is harvested from the lateral intercondylar notch. **C**, Final appearance of the capitellum after two osteochondral plugs are placed. (Reproduced with permission from Ahmad CS, ElAttrache NS: Treatment of capitellar osteochondritis dissecans. *Tech Shoulder Elbow Surg* 2006;7:169-174.)

the capitellum are relative contraindications to mosaicplasty.

A midlateral working portal is used to débride the lesion to stable cartilage borders in preparation for drilling. In lesions with a partially detached fragment, the detached region is often located centrally. In this situation, the senior author (NSE) recommends débriding the partially detached portion, beginning centrally and proceeding laterally toward the lateral column. Débridement proceeds until an area of bony integrity, consisting of an osseous

connection between the fragment and the subchondral bone, is encountered (if present). The extent of the posterolateral column involvement is then determined, and an arthroscopic evaluation (consisting of supination and pronation of the extended forearm) of radial head engagement in the defect is performed. If more than 6 to 7 mm of the lateral column is involved or the radial head is engaged in the defect, osteochondral grafting proceeds. The goal is to restore a bony buttress to prevent radial head subluxation into the

defect; it is not necessary to replace every millimeter of the lesion.

Elbow flexion is increased to 90° to 100°, and a spinal needle is introduced through the anconeus to gauge the feasibility of a perfectly perpendicular approach to the lesion. An incision is created to provide access for a 4- to 6-mm diameter plug. The recipient site is drilled perpendicular to the chondral surface using commercially available osteochondral autograft transfer instrumentation (**Figure 6, A**). The donor osteochondral plug is then ar-

arthroscopically harvested from the intercondylar notch (**Figure 6, B**). The arthroscope is placed into an anterolateral portal. Using an anteromedial portal, a donor plug of an appropriate size is then harvested with 1 cm of depth from the medial edge of the lateral femoral condyle toward the notch. The donor plug is introduced into the recipient site and impacted flush with the surrounding cartilage (**Figure 6, C**). The goal is to reconstitute the lateral buttress so that the radial head does not engage into the defect. The process of osteochondral grafting is repeated until lateral column integrity is adequately restored. If some corners of the lesion cannot be fully replaced, they are treated with drilling. If the surgeon does not choose to use autograft, allograft or synthetic scaffolding can be used.

Iwasaki et al²⁷ reported good or excellent results in seven of eight teenage baseball players with OCD who were treated with osteochondral autograft transplants. Yamamoto et al²⁸ reported that six of nine adolescents with grade III OCD lesion and eight of nine with grade IV lesions returned to competitive baseball after an osteochondral autograft transplantation procedure. Five baseball players with OCD were treated using an osteochondral autograft transplantation procedure (JD Chappell, MD, and NS ElAttrache, MD, unpublished data presented at the American Orthopaedic Society for Sports Medicine Annual Meeting, Orlando, FL, 2008). All five patients returned to competitive baseball and were still playing 5 years postoperatively. Osteochondral autograft transplantation is particularly recommended when more than 6 to 7 mm of the lateral column is involved, and radial head engagement with the lesion is seen during supination-pronation and flexion-extension of the forearm during a careful arthroscopic examination.

Fragment Fixation

Fragment fixation has been used to treat unstable, partially detached OCD lesions.²³ Kuwahata and Inoue²⁴ described using cancellous bone grafts and a Herbert screw with an open technique in seven patients. At 32-month follow-up, all patients were free of pain and returned to their sport activities. Takahara et al^{19,20} used bone pegs harvested from the lateral olecranon to fix partially detached lesions using an open approach. Newer bioabsorbable implants may facilitate fragment fixation with an arthroscopic approach. Although encouraging results have been reported, the bone quality on the fragment often has limited healing potential. This chapter's authors therefore recommend excision and drilling or grafting for partially detached lesions.

Postoperative Management

Postoperatively, the elbow should be protected for 2 to 3 weeks with a hinged brace. Passive and active assisted motion is performed to avoid postoperative stiffness. Gentle resistance exercises are initiated at 3 months, progressing to greater resistance at 4 months. For throwing athletes, a throwing program is started at 5 months. Return to full participation in sports is usually achieved at 6 months after surgery. Athletes who are treated with simple débridement and drilling or microfracture can usually return to sport activities 1 to 2 months sooner than those treated with open procedures and grafting, depending on the progress of rehabilitation.

Prognosis

Takahara et al^{19,20} retrospectively reviewed 106 patients with capitellar OCD with an average follow-up of 7 years. The authors found stable lesions healed completely with nonsurgical treatment when three common characteristics were present: an open

capitellar growth plate, localized flattening or radiolucency of the subchondral bone, and good elbow motion. The prognosis for OCD of the capitellum is better for patients with stage I disease compared to those with stage II or III OCD. Early stage OCD responds better to nonsurgical treatment than advanced stages, so identifying the disease promptly can have a significant impact on a patient's prognosis. Matsuura et al²⁹ reported that 91% of early-stage OCD but only 53% of advanced-stage OCD improved after nonsurgical treatment.

Unfortunately, most capitellar OCD lesions are diagnosed at stage II. Although surgery will usually alleviate symptoms and allow a return to sports, these patients have a less favorable long-term outcome. Longitudinal studies have documented that osteoarthritis will develop in 50% of patients with radiocapitellar OCD.³⁰ Newer techniques, including osteochondral grafting (mosaicplasty) may change the long-term degenerative process.

Fragment stability also affects the final outcome. If the fragment is stable, Mitsunaga et al³¹ reported that less than 50% of those lesions will become unstable in the long term. However, Takahara et al¹⁹ showed that fragments that become unstable have a low rate of healing. Patient age has not been correlated with the likelihood of healing in some studies.^{25,32} Mihara et al¹⁸ noted a significant correlation between open capitellar growth plates and healing, reporting that 94% of patients with early-stage OCD with open growth plates healed, whereas only 71% with closed growth plates healed.

Return to Sport Activities

Return to sport activities following treatment of capitellar OCD has varied. Historically, gymnasts have inferior outcomes compared with throwing athletes, perhaps because of the signifi-

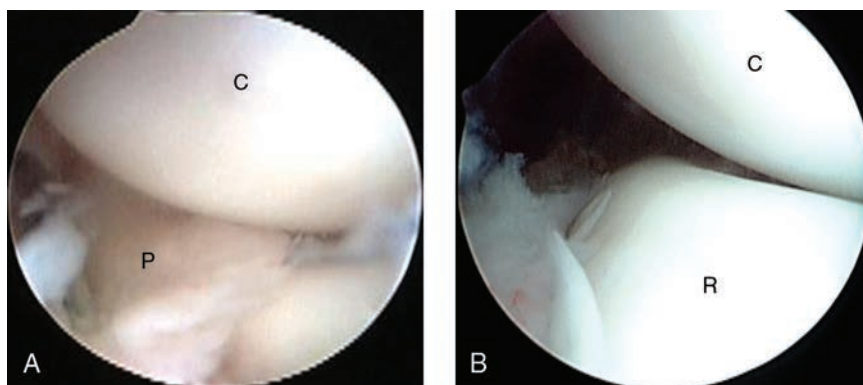


Figure 7 Arthroscopic image of the radiocapitellar plica. **A**, Radiocapitellar plica (P) adjacent to the capitellum (C). **B**, After plica excision of radial head (R) and capitellum (C), the articulation is unobstructed.

cantly increased axial elbow loads. In a study by Jackson et al,³ 10 female gymnasts were treated with removal of loose bodies and drilling after failed nonsurgical treatment for capitellar OCD; only 1 patient returned to gymnastics. More recently, Bojanić et al³³ reported that all three female gymnasts in their study successfully returned to their previous sports level after loose body removal and microfracture. Byrd et al³⁴ reported that 4 of 10 adolescent baseball players returned to competitive baseball after arthroscopic débridement, whereas Yamamoto et al²⁸ reported return to sports in 14 of 18 juvenile baseball players after osteochondral autograft transplantation. One hundred percent (8 of 8) of male baseball players returned to their previous level of sport activities at an average 3-year follow-up after either microfracture or osteochondral autograft transplantation (JD Chappell, MD, and NS ElAttrache, MD, unpublished data presented at the American Orthopaedic Society for Sports Medicine Annual Meeting, Orlando, FL, 2008).

Radiocapitellar Plica

Posterolateral elbow impingement can be caused by a thickened radiocapitellar plica and can occur in combination with capitellar OCD.³⁵⁻³⁷ The plica can

cause chondromalacic changes on the radial head and capitellum.³⁵ Symptoms include painful clicking or catching and effusions. If there is snapping, it often occurs with elbow flexion greater than 90° with the forearm in pronation.^{35,37} If identified during arthroscopic evaluation, the plica should be resected. Kim et al³⁶ reported excellent results in throwing athletes and golfers after plica débridement.

A camera is introduced in the proximal anteromedial portal, and a proximal anterolateral working portal is established. Synovitis surrounding the radial neck and the anterior capsule is débrided. The lateral plica is visualized as a fibrous band folding over the radial head. During elbow flexion and extension, the plica will snap back over the radial neck and head. An area of chondromalacia is often present on the anterolateral radial head. A combination of basket resection instruments and shavers are used to resect the plica back to the normal annular ligament (**Figure 7**). The scope is then placed in the posterolateral portal and/or direct midlateral portal, and the plica excision is continued from the midlateral portal (if viewing from the posterolateral portal) or from an accessory midlateral portal (if viewing from the midlateral portal).

Summary

Young male throwing athletes and female gymnasts are at highest risks for the development of capitellar OCD, which must be distinguished from Panner disease. Advanced OCD lesions require surgical treatment. The size and location of the lesion as well as its functional relationship to the radial head should help guide surgical treatment decisions. Factors that influence results include open growth plates, the size and stability of the lesion, and good elbow motion at the initial patient presentation.

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