Management and Surgical Options for Articular Defects in the Shoulder

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KEYWORDS

• Shoulder • Glenohumeral • Chondral • Cartilage • Defects • Articular
• Autologous chondrocyte implantation • Osteochondral autograft

KEY POINTS

- The natural history of isolated, full-thickness chondral lesions of the glenohumeral joint is less clear than those of the knee or ankle.
- Often, the diagnosis can be difficult to make clinically because of vague, nonlocalized complaints, and a history and physical examination similar to other common shoulder pathologies.
- It is imperative that the surgeon obtain as much information as possible from the clinical evaluation so as to avoid treatment of an incidental, truly asymptomatic lesion.
- No firm consensus exists as of yet on the most appropriate operative treatment options for glenohumeral focal articular defects.
- Possible treatment measures include arthroscopic debridement, microfracture, autologous chondrocyte implantation, osteochondral allograft, and osteochondral autograft transfer, as well as biologic resurfacing or metallic replacement.

INTRODUCTION

Isolated, full-thickness chondral lesions of the glenohumeral joint are a significant pathology encountered by laborers, athletes, and the elderly.[1] They may be a result of genetic and/or degenerative changes to the joint, posttraumatic lesions, postoperative changes, loose bodies, osteonecrosis (iatrogenic, corticosteroid or alcohol use), shoulder instability or microinstability, inflammatory arthritis, osteoarthritis, infection, intra-articular pain pump placement, rotator cuff arthropathy, or osteochondritis dissecans.[2–4] The incidence of 5% to 17%[5] is less common than the knee joint, likely

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related to weight bearing and impact loading that is less in the shoulder joint. This is probably why many are well-tolerated and asymptomatic.[6] Diagnosis of full-thickness chondral defects can be challenging, and the outcomes following nonoperative and operative treatment less predictable.[2] Additionally, the natural history of full-thickness chondral lesions in the shoulder is less clear than those of the knee or ankle.[7]

The management of focal chondral lesions of the glenoid or humerus remains challenging.[8,9] These defects have a limited capacity to heal because of a lack of direct vascular supply and direct access to undifferentiated, pluripotent cells to assist with native healing capacity.[10] Thus, many treatment options have been refined to provide pain relief, create reparative tissue, or restore the articular surface.[8] Although shoulder arthroplasty is a reliable option for those with more diffuse degenerative changes, it can impose significant, debilitating activity restrictions for a younger individual and includes a limited implant life span. Joint-preserving procedures are therefore particularly important to identify for those young patients with focal cartilage defects with continued pain and decreased function.

CLASSIFICATION

No specific classification scheme pertains to articular lesions in the shoulder; as such, the Outerbridge system,[11] as is used to describe lesions in the knee, is conventionally used for the glenohumeral joint as well. In this, Grade 0 refers to normal cartilage, grade I is softening of the articular cartilage, grade II involves fibrillation of half the depth of the articular surface, grade III involves fissuring of more than half of the articular surface depth, and finally grade IV is full-thickness cartilage loss to the subchondral bone. Descriptive characteristics are otherwise pertinent, including location (humerus or glenoid), position (peripheral or central), size, depth, and degree of containment.

GLENOHUMERAL ARTICULAR ANATOMY

The anatomy of the glenohumeral joint can make evaluation and treatment of articular defects difficult. The mean articular depth of the glenoid fossa cartilage is 1.88 mm and that of the humerus cartilage is 1.24 mm.[12] The glenoid articular cartilage is thickest along the periphery and tapers toward the bare area in the center where no cartilage is present. By contrast, the humeral head chondral surface is thickest in the center (at approximately 1.2–1.3 mm thick) and thins to less than 1 mm at the periphery.[13] Knowledge of these characteristics is important when considering on patient imaging or arthroscopic evaluation whether a defect in the cartilage is present, or if it is just the native patient anatomy. In addition, the geometry of the glenohumeral joint is such that the glenoid radius of curvature is within 2 to 3 mm of the humeral head so that they remain relatively congruent with the interposed chondral surfaces and labral rim.[3] Glenoid version (1.5° retroversion) and inclination (4.2° superiorly) are important to consider for as well for approaching the joint surgically.[14]

HISTORY AND PHYSICAL EXAMINATION

A thorough history should be obtained in any patient presenting to the office with shoulder pain and concern for the etiology being an articular cartilage defect. Often the diagnosis can be difficult to make clinically because of vague, nonlocalized complaints and a history and physical examination similar to other common shoulder pathologies.[7] Because of the complex nature of the shoulder joint anatomy, careful

consideration must be entertained for additional pathology to the rotator cuff, acromioclavicular joint, biceps, labrum, or capsule that may otherwise be the root of the patient's complaints. Not infrequently, intra-articular cartilage injury may be an incidental finding when another pathology is truly causing symptoms.

Traumatic events to the shoulder, including previous fractures, subluxations, or dislocations, should be investigated.[15] It is important to question the patient on prior surgical intervention on the glenohumeral joint; it can be very helpful to obtain prior operative reports and intraoperative imaging as well to review. It is imperative to note the nature and onset of symptoms as well as the progression of symptoms.[3] The quality of the patient's pain should be elucidated, as pain due to chondral defects are often dull and achy but with exacerbation by increased use. Sleep is also often affected.[15] Patients may additionally complain of mechanical symptoms, such as swelling, catching, and locking, in addition to a deep, activity-related pain as is also true for the knee joint.[1,15] The patient's age, current and desired activity level, and expectations/goals of treatment should be elucidated as well before discussing any potential therapeutic options.[3]

It is imperative that the surgeon obtain as much information as possible from the clinical evaluation so as to avoid treatment of an incidental, truly asymptomatic lesion. Physical examination should ensue as for any shoulder evaluation, including a documentation of passive and active range of motion, neurologic, and strength testing of the rotator cuff. The latter is pertinent given the reported increased incidence of articular cartilage injury in the presence of rotator cuff deficiency.[16] All examination findings should be compared with the contralateral "healthy" shoulder. Previous surgical scars should be documented. The presence of crepitus with motion may be indicative of an irregular joint surface and the possibility of chondral injury, whereas pain with compressive loads applied to the glenohumeral joint can additionally indicate the presence of an articular defect.[15] Special shoulder-specific tests should be performed to elicit any findings of concomitant pathology.[3] Unlike patients with osteoarthritis, these patients typically do not have significant limitations in motion.

IMAGING

The first-line imaging should always include plain radiographs of the glenohumeral joint, including a true anteroposterior view, scapular-Y, and axillary view to assess for any obvious osteophyte formation, subchondral sclerosis or cysts, or additional lucencies within the bone along the joint. The degree of joint space narrowing should be assessed.[15] The Stryker notch view and West Point view are helpful in evaluating Hill-Sachs lesions and glenoid bone loss, respectively, in a patient with history of instability.[17]

MRI provides the best evaluation of the chondral surface and surrounding soft tissues. It thus also allows for assessment of concomitant musculotendinous or labral pathology. The finding of a focal cartilage defect on the articular surface of the humeral head is often overlooked on MRI,[18] with rates as high as 45% for grade IV defects[19] due to the relatively thin cartilage in the shoulder. Standard sequences to evaluate articular cartilage include a T2-weighted image with or without fat suppression, and a T1-weighted fat-suppressed cartilage-sensitive sequence (ie, spoiled gradient-recalled echo or fast spin echo), which can demonstrate chondral fissuring, delamination, and focal loss.[20,21] Additionally, quantitative and semiquantitative techniques including delayed gadolinium-enhanced MRI of cartilage (dGEMRIC), T1rho, T2*, and T2 mapping techniques exist to better evaluate the composition of articular cartilage.[22] Subchondral bone marrow edema at the site of focal articular defects implies a

possible traumatic origin to the injury, or may be suggestive of a full-thickness defect if the diagnosis is not clear.[18] Traumatic humeral head cartilage defects may be found medial to the expected location of a typical Hill-Sachs lesion, potentially due to shearing or compression from the undersurface of the acromion.[18]

Computed tomography (CT) imaging is helpful to evaluate glenohumeral joint alignment, glenoid bone loss, and version for those patients who may be indicated for osteochondral grafting to treat an articular defect.

NONOPERATIVE TREATMENT

Typically, the initial treatment of glenohumeral chondral disease is nonsurgical. This is similar to what is performed to relieve symptoms in other joints, and includes a trial of activity modification, physical therapy, oral nonsteroidal anti-inflammatory medications, and corticosteroid injections.[23] Physical therapy should focus on scapulothoracic and glenohumeral strengthening, stretching, and range of motion improvement.[3] Glenohumeral injection can be diagnostic as well as therapeutic when injected with lidocaine.[3]

BASIC SCIENCE RESEARCH

Few basic science efforts have evaluated the management of focal articular defects in the glenohumeral joint. Van Thiel and colleagues[24] evaluated the use of autologous matrix-induced chondrogenesis (AMIC) in rabbit glenohumeral cartilage defect models. AMIC involves use of a collagen I/III matrix with microfracture to promote the formation of nativelike cartilage architecture. In the 12 rabbit models, no statistically significant differences in micro-CT–determined total cartilage volume or average cartilage thickness were present in in those shoulders treated with microfracture alone, microfracture and AMIC, or control. However, a trend existed toward increased defect fill and thickness in the microfracture and AMIC groups.

Wang and colleagues[25] developed a rabbit shoulder animal model to study glenoid cartilage repair strategies and chondral healing. The investigators compared 45 rabbits in 3 groups: untreated glenoid articular surface defects, microfracture, and microfracture plus type I/III collagen scaffold (AMIC). At 32 weeks after surgery, the investigators demonstrated increased fibrous tissue deposition via micro-CT and a more hyalinelike histologic repair tissue with microfracture alone, whereas additional improvements with AMIC were seen only with MRI signal findings.

PALLIATIVE, REPARATIVE, AND RESTORATIVE SURGICAL OPTIONS

No firm consensus exists as of yet on the most appropriate operative treatment options for glenohumeral focal articular defects. Importantly, the presence of symptoms, not just that an articular defect exists, is what must guide the decision to intervene. Inappropriate surgical candidates include those tumors or infection of the glenohumeral joint, systemic cartilage disease, or inflammatory arthropathy.

Treatment measures may be palliative, reparative, restorative, or reconstructive.[24] Virtually all cartilage restoration options used in the knee can be applied to the shoulder as well.[20] Given the level of activity in these patients, joint-sparing surgery is preferred when nonoperative modalities fail. Nonarthroplasty options for active, young patients include debridement alone or with microfracture (with or without augmentation strategies), autologous chondrocyte implantation, and osteochondral transplantation. Indications are less well defined than in the knee joint, as the shoulder tolerates cartilage pathology better as a result of the relatively load-sparing nature

of the joint and its wide arc of motion.[1] One must take into consideration the patient's defect location, size, depth, and containment, and the presence of any concurrent pathology that should be addressed at the time of intervention (**Table 1**).[3]

Gross and colleagues[26] reported that satisfaction rates are high in the literature (ranging from 66%–100%) for each of these procedures. The morbidity of the procedure must be considered in the decision-making process. Positive prognostic factors for this genre of intervention include lesion size less than 2 cm^2, unipolar lesions, less advanced lesions, and isolated lesions of the humerus. Negative prognostic variables included lesions greater than 2 cm^2, bipolar lesions, and prior surgical intervention.[26]

Arthroscopic Debridement

Palliative treatment with arthroscopic debridement, lavage, and loose body removal provides a relatively low morbidity procedure that does not burn bridges with future cartilage restoration procedures. At the very least, initial arthroscopic evaluation may provide a diagnostic assessment for the presence of chondral lesions to adequately stage and size the defect and determine the need for future intervention.[15] Its role in management specifically for chondral defects of the shoulder is limited in the literature, despite several studies indicating successful outcomes when used in the setting of generalized osteoarthritis.

Typical components of this procedure include removal of loose bodies, synovectomy, capsular release for motion loss, and subacromial decompression if felt to be a component of the patient's symptoms. The removal of chondral flaps with arthroscopic curettes and motorized shavers may help decrease mechanical symptoms.[3] It can be biomechanically beneficial as well to create a stable, vertical transition zone between full-thickness cartilage defects and the surrounding normal cartilage.[3]

Cameron and colleagues[19] reported on the results of 61 patients (mean age, 49.5 years) who underwent arthroscopic debridement with or without capsular release with grade IV articular lesions of the glenohumeral joint. Of the 45 patients with minimum 2-year follow-up, the mean patient satisfaction score improved significantly from 0.67 to 6.28 ($P<.0001$) with 87% of patients indicating that they would have the surgery performed again. Significant improvements in patient pain and function were noted in 88% of all patients ($P<.0001$) despite workers' compensation patients having inferior results. The onset of pain relief was noted for most patients by 5 weeks postoperative, and lasted for more than 28 months. The investigators found an association with return of pain and procedural failure for lesions larger than 2 cm^2.

Kerr and McCarty[27] reported on the outcomes of patients with either unipolar or bipolar chondral defects of the shoulder who received arthroscopic debridement. Most of these patients, however, had concurrent procedures during the debridement (16 of 19 patients including 36% with capsular release), including 2 with microfracture. Ultimately, patients at 28 months postoperative had significant pain relief (88%) and 87% were satisfied with the procedure. The investigators noted that patients with unipolar lesions had superior outcomes, and outcomes worsened with articular defect size greater than 2 $\times$ 2 cm^2.

Microfracture

Although much more commonly reported in the knee because of its ease as a first-line treatment option with low surgical morbidity and successful clinical outcomes,[1] the use of microfracture in the glenohumeral joint provides an option for isolated full-thickness cartilage defects that is minimally invasive, technically nondemanding, and potentially fruitful. Microfracture creates a channel to the underlying marrow to

Table 1
Summary of reparative and restorative interventions for chondral defects in the glenohumeral joint

Surgical Intervention	Indications	Contraindications	Technical Pearls	Rehabilitation
Microfracture	• Failed conservative management • Potential first-line option for small defects • Borrowed from knee literature: size <2 cm^2, BMI <30 kg/m^2, age <45 y, symptoms >12 mo • Unipolar • Congruent glenohumeral joint	• Generalized DJD • Hyperlaxity • Bipolar lesions • Presence of concomitant intra-articular pathology (unless concurrently addressed) • Uncontained lesions • Partial-thickness lesions • Chondral lesions with associated relevant bony defects • Violated subchondral plate • Relatively larger or bipolar lesions	• Beach chair or lateral decubitus • Careful consideration for anterior portal placement to facilitate perpendicular access to defect (more lateral for glenoid lesions, more medial for humeral lesions) • Diagnostic arthroscopy and concurrent procedures performed first • Be certain the cartilage defect is contained • Curette or shaver to create stable vertical walls and debride calcified cartilage layer • 30° or 90° awl or PowerPick to create microfracture holes perpendicular to the joint	• CPM vs no CPM • Gentle WBAT after surgery • Phase I: protective passive ROM weeks 0–8 • Phase II – active ROM and strengthening weeks 9–14 • Phase III – return to sport weeks 15–17 • No heavy lifting for 3 mo • Full activity allowed at 4 mo • No overhead athletic competition for 6 mo

Augmented microfracture (ie, micronized allogeneic cartilage matrix implantation)	(As for microfracture alone)	(As for microfracture alone)	• Best to have positioned in lateral decubitus to avoid gravity effects on implantation • Turn off arthroscopic fluid and suction joint dry • Prepare BioCartilage mixture with PRP • Thin layer of fibrin glue in the prepared, dried defect bed • Introduce BioCartilage mixture paste onto the defect so that it is slightly recessed when compared with surrounding chondral margins • Freer elevator can be used to smooth over the surface of the BioCartilage, and a thin layer of fibrin glue is used to seal over the top of this and the neighboring cartilage	• Sling for 4–6 wk with removal for active and active-assisted ROM activities • Otherwise as for microfracture alone

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Surgical Intervention	Indications	Contraindications	Technical Pearls	Rehabilitation
Autologous chondrocyte implantation (ACI)	• Young adult (<40 y old) • Isolated lesion • Large lesion not amenable to OATS • Full-thickness defect • Smaller, contained superficial defects	• Relevant subchondral bone edema	• Cartilage biopsy (harvest) site either at the intercondylar notch of the knee, or at healthy cartilage near the shoulder defect • Open approach (deltopectoral) • Obtain hemostasis at the base of the defect • Debride the defect but do not violate the subchondral bone plate • Periosteal patch harvest from medial tibia, or instead a type I/III collagen-based membrane matrix can be used • Fibrin glue along the periphery of the periosteal patch	• CPM machine suggested, 6–8 h daily • At 4 wk, 90° elevation and 20° ER active-assisted motion allowed • At 6 wk, 140° elevation and 40° ER active-assisted motion allowed • At 12 wk, no restrictions • Questionable return to overhead throwing

Osteochondral autograft transfer	• Relatively smaller (10–20 mm diameter) defect sizes for autografting are proposed • Combined cartilage and bone loss (osteochondral injury)	• Young patients • As a first-line treatment	• Open, open-assist arthroscopic, or all-arthroscopic means • Autografting harvest site is the ipsilateral sulcus of the lateral femoral condyle • Press-fit technique for graft to recipient site • Bio-Compression screw for backup fixation	• Sling initially postoperative (<1 wk) • Active-assisted and passive ROM exercises POD#1 • At 3 wk, active ROM allowed • At 5 wk, strengthening exercises introduced • At 6 mo, return to overhead sport
Osteochondral allograft	• Larger, full-thickness defects • Combined cartilage and bone loss (osteochondral injury)	• Young patients • As a first-line treatment	• Open, open-assist arthroscopic, or all-arthroscopic means • Press-fit technique for graft to recipient site • Bio-Compression screw for backup fixation	• Sling initially postoperative (<1 wk) • Active-assisted and passive ROM exercises POD#1 • At 3 wk, active ROM allowed • At 5 wk, strengthening exercises introduced • At 6 mo, return to overhead sport

Abbreviations: BMI, body mass index; CPM, continuous passive motion; DJD, degenerative joint disease; ER, external rotation; OATS, osteochondral autograft transfer system; POD, postoperative day; PRP, platelet-rich plasma; ROM, range of motion; WBAT, weight bearing as tolerated.

encourage chondral resurfacing with fibrocartilage at the site of a focal defect through an introduction of mesenchymal stem cells, growth factors, fibrin, and platelets.[28] Given that the scapula and humerus have excellent vascular supply, it would seem plausible that the glenohumeral joint could expect similar success to the knee with microfracture surgery.[7] It appears to be a viable option for both acute and chronic articular cartilage lesions,[9] and its use avoids the harvest site morbidity of autografting procedures for chondral defect repair yet does not compromise the surgeon's performance of more aggressive subsequent procedures.[3]

There is no formal defect size limit for microfracture, as is quoted consistently through the knee literature, due to the relative paucity of microfracture literature in the glenohumeral joint.[1] However, smaller lesion size is preferred for treatment.[9] Patients should have a focal, symptomatic lesion that has failed conservative management, and the joint should be congruent. From the literature of microfracture use in the knee, considerations of patient chondral defect size (<2 cm^2), age (<45 years), body mass index (<30 kg/m^2), and symptom duration (>12 months) are helpful to identify a patient who will maximally benefit from the intervention.[29]

Absolute contraindications include the presence of generalized degenerative joint osteoarthritis, high-grade ligamentous laxity, partial-thickness lesions or lesions associated with large bony defects, and subchondral plate violation.[1,9] Relative contraindications included lesions of larger size or those with untreated bipolar counterparts,[15] the latter of which may be better treated with glenoid microfracture but biologic restorative means (such as osteochondral allografting) of the humerus.[15] Microfracture should additionally not be performed in isolation if intervention is needed to address concomitant rotator cuff injury, labral or biceps disease, or shoulder instability, in which repeated postoperative subluxations/dislocations may affect the healing capacity from microfracture (see **Table 1**).[1,15]

With the surgical technique, the patient can be placed either in beach chair or lateral decubitus position based on surgeon preference. A standard posterior portal is used; the position of the anterior working portal is judged based on the location of the chondral defect being addressed so that a more direct, perpendicular route is attainable to the lesion.[1] That is, a more lateral position of the anterior portal will be beneficial to work at the anterosuperior glenoid, whereas a more inferior portal can help reach a defect in the inferior glenoid. Conversely, a more medial position for the portal will benefit access to the humerus, and internal and external rotation of the head will help enable the approach. A posterior 7-o'clock portal may allow easier access to the posterior glenoid.[1]

After diagnostic arthroscopy, any additional concurrent pathology should be addressed first so as to maintain clarity in visualization for these concomitant interventions that can be lost after microfracture.[1] It is necessary to confirm containment of the chondral defect before proceeding.[1] Standard procedure for microfracture is then performed as in other joints throughout the body: debridement with an arthroscopic shaver, ring curette, or basket forceps can ensure.[1] A combination of arthroscopic elevator, Q7 shaver, and curette can be used to create stable vertical walls circumferentially to facilitate the fibrous clot formation after microfracture. The calcified cartilage layer is debrided in its entirety, typically with an arthroscopic curette or the shaver run in forward or reverse direction, with confirmation through punctate bleeding in the bone base.[9] As the concavity of the glenoid can make it difficult to place the microfracture awl tip perpendicular to its surface, it is suggested to use a 90° awl for superior positioning. The 30° awl is often more appropriate for the humeral head, which is typically easier to access.[1] The PowerPick instrumentation can otherwise be used if preferred.[4]

Augmentation strategies including micronized allogeneic cartilage matrix (BioCartilage) implantation can be considered in an attempt to restore the glenohumeral joint surface with a more hyaline-type cartilage as opposed to the fibrocartilage generated from microfracture alone.[4] When this is deemed appropriate, the arthroscopic pump is shut off and the joint fluid suctioned to thoroughly dry the defect site. The BioCartilage mixture paste is mixed with platelet-rich plasma (PRP) and placed over a thin layer of fibrin glue to fill the defect almost to the level of the surrounding healthy cartilage. Another layer of fibrin glue is placed over the top of the smoothed BioCartilage surface.[4]

Well-defined rehabilitative protocols following microfracture of the shoulder joint are limited. The shoulder differs from the knee in terms of its decreased joint volume and synovial lining, and increased range of motion; thus, some investigators[1] have advocated against use of continuous passive motion (CPM) machines, as patients can often move their shoulder appropriately after surgery to stimulate synovial fluid production. As the shoulder is not a load-bearing joint, patients can bear weight as tolerated, with avoidance of heavy overhead lifting and competitive overhead athletics for 3 and 6 months, respectively.[1]

Hensley and Sum[30] provide a detailed postoperative rehabilitation protocol including a 3-phase approach. Phase I includes protective passive range of motion from 0 to 8 weeks postoperatively. Phase II includes active range of motion and strengthening from 9 to 14 weeks postoperatively. Phase III is a return to sport phase from weeks 15 to 17 postoperatively, with a focus on advanced strengthening, control, and introduction of resistance activities while maintaining and improving shoulder motion.

Siebold and colleagues[31] reported on 5 patients who underwent a combination of open microfracture and periosteal flap for the treatment of focal full-thickness humeral head chondral lesions. Three of the patients had undergone instability treatment previously, and 2 again at the time of microfracture surgery. Mean patient age was 32 years, and mean lesion size was 311 mm^2. Patients had significant improvements at a mean follow-up of 25.8 months in Constant score (43.4% to 81.8%) and pain reduction (to 18.6 points). Second-look arthroscopy in 3 patients at a mean 8 months postoperative demonstrated significantly reduced chondral lesion sizes.

Snow and Funk[7] evaluated 8 patients who underwent arthroscopic microfracture to treat full-thickness chondral lesions smaller than 4 cm^2. The mean age was 37 years, and 7 patients (87.5%) underwent concurrent procedures. Five of the treated defects were at the humeral head, and the remaining 3 at the glenoid. The investigators ultimately saw significant improvements in Constant and Oxford scores, with no complications. Two second-look arthroscopic surgeries demonstrated good lesion filling with fibrocartilage.

Millett and colleagues[9] described the results of microfracture in 30 patients (31 shoulders), including 6 patients with bipolar lesion treatment, 13 with glenoid defect treatment, and 12 with humeral head microfracture. Although the investigators reported a failure rate of 19% (6 of 31 shoulders), at a mean 47 months of follow-up, pain scores, patient ability to work, and performance of activities of daily living and sport activities all significantly improved. The mean American Shoulder and Elbow Surgeons (ASES) score improved by 20 points as well, and patients expressed a mean satisfaction of 7.6 of 10. The investigators found the greatest significance in improvement for those patients with isolated humeral head lesions that received treatment; a negative correlation was found between lesion size and ASES improvement, and patients with bipolar treated lesions were least improved.

Slabaugh and colleagues[1] described a case report of a patient in his early 40s with a 10-year history of right shoulder pain who was successfully treated with microfracture

of a 25 × 25-mm focal chondral defect on the humeral head. He regained full shoulder motion, complete satisfaction, and full strength postoperatively.

Frank and colleagues[5] reported on 17 shoulders in 16 patients who underwent microfracture of the glenoid (n = 6), humerus (n = 10), and both surfaces (n = 1) at a mean follow-up of 27.8 months. The mean patient age was 37 years, with average humeral and glenoid defect sizes of 5.07 cm^2 and 1.66 cm^2, respectively. The investigators reported failure (by means of subsequent surgical intervention) in 3 patients. The investigators reported significant improvements in visual analog scale (VAS) pain scores (5.6–1.9), Simple Shoulder Test score (5.7–10.3), and ASES score (44.3–86.3). They reported that 12 patients (92.3%) said they would have the procedure performed again.

Hensley and Sum[30] reported on a 46-year-old male powerlifter with grade IV chondral lesions of the humeral head and articular surface of the superior glenoid rim measuring 2 to 3 cm in diameter each. He underwent microfracture of both defects with concurrent debridement of a type I superior labral tear from anterior to posterior (SLAP) and subacromial decompression. The patient at 2 years postoperatively was very satisfied with his outcome, with substantial improvements in all QuickDASH subscores and a return to lifting, although at much lower weight quantities (**Table 2**).

Autologous Chondrocyte Implantation

At present, there is not definitive evidence for the use of autologous chondrocyte implantation (ACI) in the shoulder, as the literature is much more scarce than is present for the knee joint. Some investigators believe it to be a promising avenue for treatment, at least in part because of the low loads experienced by the joint. The autologous chondrocytes produce anabolic growth factors to promote cell survival and induce chondrocyte proliferation at the site of implantation; however, concerns exist for its use because of the relatively high levels of shear stress during shoulder rotational motions, such as overhead throwing, which could affect the integrity of the ACI procedure.[8] Considering the positive results demonstrated in the use of ACI for articular defects in the knee, however, its use in the shoulder has begun to be evaluated.

The optimal indication is for a contained, unipolar, superficial, or surface defect devoid of subchondral bone involvement/edema in the humerus or glenoid in a relatively young patient (age <40 years) who failed cartilage reparative techniques (ie, microfracture).[20,32] It otherwise may be implemented in larger lesions not amenable to osteochondral autograft transplantation or more superficial lesions in which violating the subchondral surface (as occurs with osteochondral grafting) is to be avoided. Potential morbidity may be introduced by the open approach required for the procedure, or the 2-step approach required to harvest and subsequently implant the chondrocytes (see **Table 1**).

Autologous cartilage can be harvested from the knee at the intercondylar notch, in the location of a typical notchplasty during anterior cruciate ligament reconstruction. ACI can be performed within 1 month thereafter.[8] Other sources have included at the location of macroscopic healthy cartilage near the defect site.[32] An open approach using the deltopectoral interval is most appropriate for surgical visualization and performance of the procedure. The articular edges of the defect are debrided to stable vertical walls, such as with a ringed curette, with careful hemostasis obtained at the base of the defect. The subchondral bone plate does not need to be violated with this debridement.

A periosteal patch can be harvested from the medial tibia just distal to the pes anserine, and sutured to the remaining cartilage using 6 to 0 Vicryl sutures with a small opening left for injection of the chondrocyte suspension.[8] A collagen I/III-based matrix

membrane can be used instead of a periosteal flap to avoid donor site morbidity, excessive suturing, and the rate of graft hypertrophy.[33] Fibrin glue is used along the circumference of the periosteal patch to create a watertight seal. The suspension is injected, and the remaining defect sutured and sealed.

Some investigators have suggested use of CPM machine after this procedure for 6 to 8 hours daily with initial weight-bearing restrictions.[8] Active-assisted motion to 20° of external rotation and 90° of elevation is typically allowed at 4 weeks postoperative; this is increased to 40° and 140°, respectively, at 6 weeks postoperative. Restrictions are eliminated at 12 weeks postoperative for strengthening and range of motion. Return to overhead throwing activities is questionable.[8]

Only 2 published studies exist on the use of ACI in the shoulder. Romeo and colleagues[8] described a case report of a 16-year-old boy with a 2-year history of insidious-onset right shoulder pain related to throwing a baseball. After an outside surgeon had performed an arthroscopic subacromial decompression and thermal shrinkage, he began to develop increased mechanical symptoms in the subsequent months, and clinical evaluation suggested a posterosuperior labral tear. At the time of revision arthroscopic stabilization, the patient was noted to have a 3.3 × 1.5-cm full-thickness chondral defect in the anterosuperior humeral head. After this procedure, the patient had continued symptoms and was deemed appropriate for autologous cartilage harvest from the knee with subsequent ACI performance in the shoulder 1 month later. At 12 months postoperative, the patient demonstrated full and painless range of motion with no complaints of pain at rest.

Buchmann and colleagues[32] reported on 4 consecutive male patients (mean age, 29.3 ± 6.2 years) who underwent ACI for large symptomatic glenoid (1 measuring 2.0 cm^2) or humeral (3, measuring each 6.0 cm^2) full-thickness chondral defects. At a mean follow-up of 41.3 ± 24.9 months, all patients had satisfactory shoulder function with mean postoperative VAS scores of 0.3, mean unweighted Constant scores of 83.3 ± 9.9, and mean ASES index of 95.3 ± 8.1. Patients additionally underwent MRI, which indicated satisfactory coverage of the defect locations with signs of fibrocartilaginous repair tissue formation (see **Table 2**).

Osteochondral Autograft Transfer

Advantages of osteochondral autograft transfer include the ability to restore the glenohumeral architecture with a viable "organ" of live cartilage and bone through a single-stage procedure. It additionally provides the opportunity to achieve osseous integration while preserving the articular tidemark.[3] Disadvantages of osteochondral autograft include specifically donor site morbidity, and allograft as well as autografting risks dead space between circular grafts, graft integration, and the differing mechanical properties and geometry between the recipient and donor cartilage sites.[34]

Some investigators believe that osteochondral graftings are less appealing as an initial treatment option in young patients because of the destruction it requires to the healthy subchondral bone and lack of good salvage procedures should it fail.[8] It is typically used in the genre of anterior shoulder instability repair, for those shoulders with Hill-Sachs lesions caused by the impact of the posterolateral aspect of the humeral head on the anterior aspect of the glenoid at the time of dislocation, or in others with large, uncontained defects with subchondral bone loss.[20] Often, as with ACI, this is considered a second-line procedure after failed cartilage reparative techniques (ie, microfracture), but can be used as a first-line procedure.

The ideal osteochondral defect size for osteochondral autologous transplantation to the shoulder is between 10 and 20 mm in diameter or an area of 1.0 to 1.5 cm^2.[34] Osteochondritis dissecans of the humeral head, although an uncommon disorder in

Table 2
Clinical outcome studies on reparative and restorative treatments for glenohumeral chondral defects

Authors	Operative Treatment	Defect Location	Study Type/ Cohort Size	Patient Information	Clinical Outcomes
Slabaugh et al,[1] 2010	Microfracture	Humerus	Case report N = 1	• Early 40-something year old, 10-y history of shoulder pain, failed nonsurgical management • Lesion size: 25 × 25 mm	• 3/10 pain → 0/10 pain (on VAS scale) • ASES score 62 → 100 • Full ROM, strength • Complete satisfaction
Hensley et al,[30] 2011	Microfracture	Glenoid AND humerus	Case report N = 1	• 46 y-old male power lifter • Full-thickness humerus lesion, lesion of articulating surface of superior glenoid rim, both 2–3 cm in diameter • Concurrent SLAP debridement, SAD	• 2-y postoperative • QuickDASH sport 100 → 25; work 56.25 → 6.25; ADLs 40.9 → 4.5 • Minimal, intermittent stiffness • Very satisfied
Siebold et al,[31] 2003	Microfracture (+periosteal flap)	Humerus	Case series N = 5	• Grade IV defects, mean size 311 mm^2 (range, 225–400 mm^2) • Mean age, 32 y (range, 16–56 y) • Concurrent surgeries: posterior capsule shift (2), anchor removal (2), labral augmentation (1) • 3 with prior surgeries (open or arthroscopic Bankart repairs)	• Mean follow-up 25.8 mo • 3 patients with second-look scope at mean 8 mo, all with significantly reduced lesion sizes • Constant score significantly improved 43.4% → 81.8% • Pain reduced significantly to 18.6 points • Radiography and MRI showed progression of arthritis in 2 patients
Snow et al,[7] 2008	Microfracture	Glenoid OR humerus	Case series N = 8	• 6 men, 2 women • Mean age, 37 y (range, 27–55 y) • Lesion size <4 cm^2 • 1 isolated surgery, 7 with concurrent procedures (2 SAD, 2 capsular plication, 3 anterior stabilization) • 5 humeral head defects, 3 glenoid defects	• Mean follow-up of 15.4 mo (range, 12–27 mo) • Mean Constant score 43.88 → 90.25 (P<.005) • Mean Oxford score 25.75 → 17 (P<.005) • No complications • 2 second-look operations, both showed good filling with fibrocartilage

Millett et al,[9] 2009	Microfracture	Glenoid AND/OR humerus	Case series N = 31 shoulders (30 patients)	• 25 men, 5 women • Mean age, 43 y • 6 both humeral (mean 442 mm^2) and glenoid (mean 273 mm^2); 13 just glenoid (mean 137 mm^2); 12 just humeral head (mean 422 mm^2) • Concomitant procedures: 6 instability procedures, 10 SADs, 7 capsular releases or manipulations under anesthesia, 7 SLAP lesion debridements/repairs, 3 biceps releases	• Mean final follow-up, 47 mo • Mean pain scores 3.8 → 1.6 • Significant improvements in patients' ability to work, ADLs, sports activity ($P<.05$) • Painless use of involved arm improved ($P<.05$) • Mean ASES score improved by 20 points ($P<.05$) • Mean satisfaction 7.6 of 10 • No association between age/gender and outcomes • Greatest improvements when isolated lesion to humerus • Worst with bipolar lesions • Failure in 6 of 31 (19%): 3 shoulder replacements at mean 41 mo, 1 shoulder instability procedure, 1 biceps/instability, 1 unknown procedure
Frank et al,[5] 2010	Microfracture	Glenoid AND/OR humerus	Case series N = 17 shoulders (16 patients)	• Mean age 37.0 y (range, 18–55 y) • 7 men, 5 women in final analysis (2 lost to follow-up, 3 failures) • Average humeral defect size, 5.07 cm^2 (range, 1.0–7.84 cm^2) • Average glenoid defect size, 1.66 cm^2 (range, 0.4–3.75 cm^2)	• Mean 27.8-mo follow-up • Three failures (subsequent shoulder surgery) • Significant VAS pain improvement 5.6 → 1.4 ($P<.01$) • Significant Simple Shoulder Test improvement 5.7 → 10.3 ($P<.01$) • Significant ASES improvement (44.3 → 86.3) • 92.3% said they would have the procedure performed again

(*continued on next page*)

Table 2
(*continued*)

Authors	Operative Treatment	Defect Location	Study Type/ Cohort Size	Patient Information	Clinical Outcomes
Buchmann et al,[32] 2012	ACI	Glenoid OR humerus	Case series N = 4	• 4 men • Mean age, 29.3 ± 6.2 y • 3 humeral full-thickness defects (each 6.0 cm^2), 1 glenoid full-thickness defect (2.0 cm^2) • Humeral locations: anterior-superior, posterior-central, central • Glenoid location: posterior • Concomitant surgeries: 2 loose body extraction, 1 anchor extraction, 1 tenodesis of long head of biceps, 1 microfracture of anterior glenoid	• Final follow-up, mean 41.3 ± 24.9 mo • Mean VAS 0.3 of 10 • Mean unweighted Constant score 83.3 ± 9.9 • Mean ASES index 95.3 ± 8.1 • MRI with satisfactory defect coverage with signs of fibrocartilaginous repair tissue
Romeo et al,[8] 2002	ACI	Humerus	Case report N = 1	• 16 y old, 2-y history of shoulder pain, failed arthroscopic SAD and capsular thermal shrinkage • Lesion size: 33 × 15 mm	• At 12 mo, full ROM without pain • No further complaints, no pain at rest
Camp et al,[43] 2015	OA	Glenoid	Case report N = 1	• 25-y-old former multisport athlete • 6-y history of pain • 15-mm-diameter defect • Medial tibial plateau osteochondral allograft source	• At 1 y postoperative, subjective shoulder value score 40% → 99% • QuickDASH score 36 → 2 • ASES score 46 → 92 • Articular surface restoration maintained at 6-mo MRI

Park et al,[35] 2006	OATS	Humerus	Case report N = 1	• 13-y-old boy • Defect on posterosuperior head, 9 mm • Harvest site, ipsilateral sulcus of the lateral femoral condyle • All arthroscopic	• At 5-mo postoperative, second-look arthroscopy demonstrated healed and covered with congruent hyaline cartilage • Final follow-up 31 mo, no symptoms and good functional results with radiographic resolution
Kircher et al,[38] 2009	OATS	Glenoid OR humerus	Case series N = 7	• Age range, 23.4–57.1 y (mean 43.1 y) • 6 men, 1 woman • Defects on anterocentral glenoid (1); central (3), posteromedial (1), posterocentral (1) and anterocentral (1) humerus • Mean 1.86 osteochondral cylinders used • Mean size of affected area 150 mm^2 • 4 isolated procedures; 3 with concurrent labral augmentation and capsular shift • Harvest site on ipsilateral knee	*Note: The investigators reported outcomes at mean 32.6 mo (Scheibel et al,[34] 2004), where patients had significant increases in mean Constant score and MRI evidence of good osseointegration and congruent cartilage site in all but 1 patient* • Mean final follow-up 8.75 y • 100% very satisfied • No reoperations • Mean Constant score 76.2 → 90.9 (P = .018) • Mean Lysholm score 100 → 99.3 • One patient had marginal decline in knee function • From first to final follow-up, 3 patients showed no change in pain but 3 showed an increase in their pain score (P = .257) • 100% increased level of ADLs (P = .018) • All but one with significant strength increase (P = .028) • All patients with increased OA classification at final follow-up • All but 1 patient with congruent joint surface on final MRI; all grafts fully integrated into surrounding bone

Abbreviations: ACI, autologous chondrocyte implantation; ADL, activities of daily living; ASES, American Shoulder and Elbow Surgeons; OA, osteochondral allograft; OATS, osteochondral autograft transplantation system; ROM, range of motion; SAD, subacromial decompression; SLAP, superior labral tear from anterior to posterior; VAS, visual analog scale.

young patients, is another pathology that may warrant osteochondral transplantation. This involves a localized involvement of part of the subchondral bone and overlying articular cartilage that results in separation of the two and a resultant defect in the chondral surface (see **Table 1**).[35]

The procedure can be performed through an open approach, or by all-arthroscopic means.[36] With autografting, a donor plug can be harvested through an open approach from the lateral trochlea of the knee just proximal to the sulcus terminalis. Osteoarticular bone is reamed at the recipient site, to match the sized core of osteochondral graft. Fixation can be achieved through press-fitting, partially threaded cancellous screws, or headless compression screws.[37]

Postoperative rehabilitation varies after allograft or autograft transplantation. Sling use for the first week after surgery is advised by some investigators, with active-assisted and passive range of motion exercises allowed as soon as postoperative day 1. At 3 weeks postoperative, active range of motion is initiated, and strengthening exercises are introduced at 5 weeks from surgery. Return to overhead sport may be feasible at 6 months from the date of surgery.

Park and colleagues[35] performed an arthroscopic osteochondral autograft transfer in treatment of an osteochondral defect of the humeral head of a 13-year-old boy with an osteochondral lesion measuring 9 mm in diameter. The investigators obtained a bony graft from the ipsilateral sulcus of the lateral femoral condyle, and transplanted the tissue through arthroscopic means to the posterosuperior defect site. At a second-look arthroscopic surgery 5 months postoperative, the defects at the harvest site and pathologic site were completely healed and covered with congruent articular hyaline cartilage. With final 31-month follow-up, the patient had no symptoms and good functional results, with radiographic resolution of the defect.

Scheibel and colleagues[34] reported on 8 patients at medium-term follow-up of 32.6 months after osteochondral autologous transplantation to the humerus and/or glenoid. The mean patient age was 43.1 years. The patients had a mean defect size of 150 mm^2. Four patients underwent concurrent procedures at the time of the index intervention (labral augmentation and capsular shift). The investigators reported significant improvements in the mean Constant score, with MRI demonstrating good osseointegration of the osteochondral plugs and congruent articular surface at the site of transplantation for all but 1 patient. Macroscopic appearance in 2 patients who underwent second-look arthroscopy showed an intact surface as well. Kircher and colleagues[38] reported on 7 of the aforementioned patients (6 humeral, 1 glenoid) at a mean long-term follow-up of 8.75 years as well. Patients significantly improved in terms of mean Constant score and Lysholm score, although a significant progress of osteoarthritic changes was present from preoperative to final follow-up, unrelated to the defect size, number of cylinder use, or the Constant score. Postoperative imaging demonstrated congruent joint surfaces at the defect in all but 1 patient, with full bony integration of all osteochondral grafts. Ultimately, the investigators suggested a satisfactory outcome over a long follow-up period from the surgery with very good subjective and objective findings.

Osteochondral Allograft

The use of osteochondral allografts to address chondral articular defects in the knee has been well established,[39] but its utility in the shoulder has been evaluated only more recently. The goal with osteochondral grafting is to recreate the congruency of the articular surface (**Fig. 1**).[3] It requires a thorough appreciation for the morphology of the native glenohumeral joint to ensure proper placement and sizing.[40] Concern exists

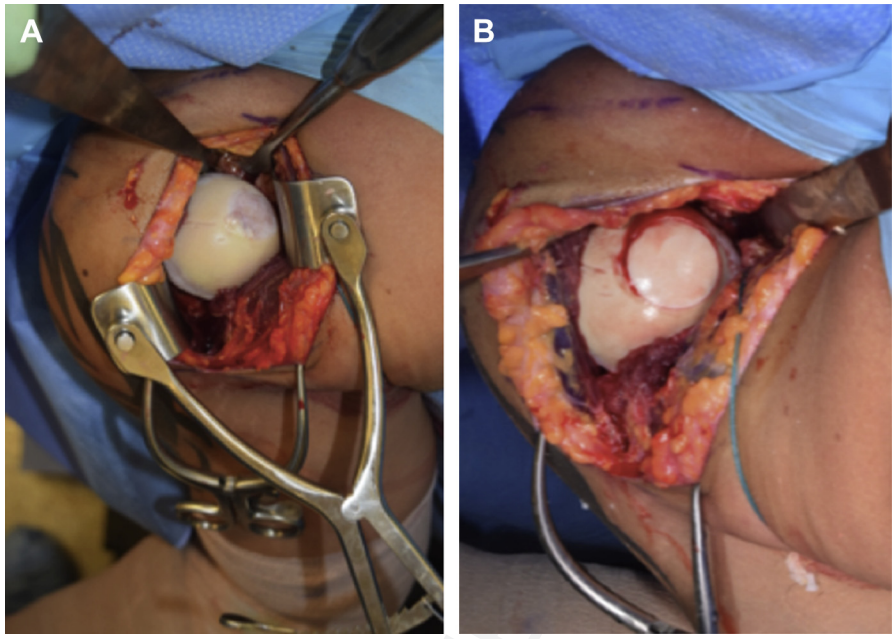

Fig. 1. (*A*) Symptomatic chondral lesion in a 30-year-old active man having failed prior arthroscopic debridement. (*B*) Image of OA plug in place of defect in same 30-year-old man.

at the glenoid as to whether reaming may cause a cortical blowout, and thus whether adequate depth of reaming can occur to provide a stable press-fit of an osteochondral graft. Accommodation of graft size decreases significantly as the reaming depth is increased above 4 mm.[41]

The procedure is performed similarly to the aforementioned approach for osteochondral autografting. The source of osteochondral allografts can be fresh or fresh-frozen, and include femoral head allograft or humeral head allograft sources. Recent data suggest that the talar dome has a high degree of surface congruency when compared with the humeral head, with maximal graft sizes of 30 × 10 mm; this may be a potential future source option as an alternative to a size-matched humeral head allograft.[42] Postoperative rehabilitation is similar to the aforementioned protocol recommendations for osteochondral autografting.

Humeral head osteochondral allograft transplantation has been evaluated in terms of large Hill-Sachs lesions due to instability with significant improvements in shoulder motion and ASES scores as far as 1-year postoperatively, and with high rates of return to work and satisfaction despite substantial complication and reoperation rates.[37] Camp and colleagues[43] reported the use in a 25-year-old male former multisport athlete of a tibial osteochondral allograft to restore a large glenoid osteochondral defect. The investigators had a successful result at 1-year postoperative with significant improvements in the patient's QuickDASH score (from 36 to 2), subjective shoulder value (from 40% to 99%), and ASES score (from 46 to 92). MRI demonstrated maintained congruity of the articular surface at 6 months postoperative (see **Table 2**).

BIOLOGIC RESURFACING AND RECONSTRUCTIVE SURGICAL OPTIONS

For those young patients with advanced bipolar lesions not amenable to reparative or restorative options, biologic resurfacing may be used. This refers to the use of soft tissue interposition within the joint, including fascia lata, allograft tendon, periosteum, porcine small intestine submucosa, anterior shoulder capsule, or allograft meniscus,[20,44] to biologically resurface the glenoid with either biologic or nonbiologic resurfacing of the humeral head. The procedure is most often performed in association with hemiarthroplasty of the humeral head.[45] The use of this technology is to bridge the treatment gap for this demographic of patients who are not yet candidates for total shoulder arthroplasty. The goal is thus to avoid the complications of glenoid component loosening and morbidity of revision procedures for young, typically high-demand patients who can be seen with arthroplasty surgery.[6] Few clinical studies have evaluated these techniques, but results are generally positive.[23,44,46–48]

Additional reconstructive efforts with metallic replacement means are typically reserved for the more diffuse, osteoarthritic shoulder rather than for the management of a focal articular defect. However, these may be required for use as salvage options when failure has occurred, or in the setting of bipolar disease in which the aforementioned options are less appropriate. These include open lateral meniscal allograft or dermal patch resurfacing, the glenoid ream-and-run procedure with humeral head implant resurfacing, and total shoulder arthroplasty.[49] Partial shoulder arthroplasty options include inlay arthroplasty, hemiresurfacing, and stemmed hemiarthroplasty,[50] whereas total shoulder replacement includes total resurfacing, stemmed totals shoulder arthroplasty, and reverse shoulder arthroplasty. Total shoulder arthroplasty remains an option for older patients with more diffuse, symptomatic cartilage disease, but imposes significant limitations on the younger patient with a more focal articular defect. These interventions remain outside of the scope of this review article, but their outcomes in the young adult are well described in the literature.[3]

FUTURE DIRECTIONS: PLATELET-RICH PLASMA?

PRP has shown greater promise as an emerging biological therapy for the treatment of chondral injury and cartilage repair efforts in the knee because it provides numerous bioactive growth factors at the site of application.[51] PRP increases chondrocyte and mesenchymal stem cell proliferation, proteoglycan deposition, and type II collagen deposition, and it has been used as an independent intra-articular injection, or as an adjunct to concomitant surgical management in the knee (ie, microfracture surgery, graft/scaffold/implant insertion).[52] It has also been described in clinic use for Achilles tendon rupture, chronic rotator cuff tendinopathy or tearing, muscle injury, chronic tendinosis, and meniscal repair.[53] The use of PRP in the glenohumeral joint for articular defects has not yet been evaluated in the literature, however.

OVERALL KEY PRINCIPLES IN TREATMENT

Gross and colleagues[26] suggested 5 key principles to guide treatment of focal articular defects of the glenohumeral joint that hold true when considering the most recent literature updates: (1) arthroscopic debridement alone should be considered when a lesion is encountered incidentally; (2) biologic resurfacing should be considered when lesions are bipolar; (3) osteoarticular graft or resurfacing should be considered when the lesion involves bone loss; (4) microfracture and osteochondral autograft transfer system (OATS) should be considered when the lesion is small; and (5) ACI or osteochondral allograft (OA) should be considered when the lesion is large. Q8

CONCLUDING THOUGHTS

Articular cartilage defects in the glenohumeral joint remain a challenging pathology for the treating orthopedic surgeon. A thorough workup of the patient needs to be performed to confirm a symptomatic defect. The patient's articular defect characteristics, symptoms, and activity level all must be taken into consideration when developing a treatment plan for this complex problem.

Gross and colleagues[26] conducted a systematic review of clinical outcomes after many of the aforementioned cartilage restorative and reparative procedures in the glenohumeral joint. In their synthesis of the data, they identified that most studies reported favorable results, but the evidence available for the use of these procedures is considered "very low" and "any estimate of effect is very uncertain." The investigators reported, however, that all of these studies are observational, retrospective case series without control groups.

These investigators highlighted how high-quality evidence is clearly lacking for any of these procedures in the glenohumeral joint. Decision making in this patient demographic should be performed on a case-by-case basis. Long-term clinical evaluation studies and randomized clinical trials are needed for these surgical procedures and their use in the glenohumeral joint to better define surgical indication and efficacy of use, and comparison of efficacy against one another.

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Q4	The following synopsis is the one that you supplied, but edited down to less than 100 words. Please confirm OK, or submit a replacement (also less than 100 words). Please note that the synopsis will appear in PubMed: Isolated, full-thickness chondral lesions of the glenohumeral joint are a significant pathology encountered by laborers, athletes, and the elderly. A thorough history should be obtained in any patient presenting to the office with shoulder pain and concern for the etiology being an articular cartilage defect. The first-line imaging should include plain radiographs of the glenohumeral joint; MRI and CT can be ordered as necessary to provide greater detail. Typically, the initial treatment of glenohumeral chondral disease is nonsurgical; however, many surgical treatment options have been refined to provide pain relief, create reparative tissue, or restore the articular surface.
Q5	Please verify the affiliation addresses.
Q6	If there are any drug dosages in your article, please verify them and indicate that you have done so by initialing this query.
Q7	Phrase does not seem complete "Standard procedure for…basket forceps can ensure."

(*continued on next page*)

Q8	Please verify the expansion for OA.
Q9	Could you please provide the grant number for (1) Nova Science Publishers, (2) Zimmer, (3) Medipost, (4) Regentis, (5) Aesculap/B.Braun, (6) Össur, (7) Arthrex, (8) Cytori, (9) Tornier, (10) DJ Orthopaedics, (11) Postgraduate Institute for Medicine, (12) National Institutes of Health, (13) Smith and Nephew, (14) SLACK Incorporated, (15) Athletico, (16) Saunders/Mosby-Elsevier and (17) Elsevier Publishing, if any?
Q10	One or more sponsor names may have been edited to a standard format that enables better searching and identification of your article. Please check and correct if necessary: (1) Smith and Nephew, United Kingdom; (2) Össur, Iceland; (3) Arthrex, United States.
Q11	Please provide the publisher location for Ref. 20.
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Thank you for your assistance.