Management of cartilage defects in the shoulder

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Abstract Articular cartilage lesions of the glenohumeral joint are an especially difficult clinical problem to manage, particularly in the younger, more active patient. Left untreated, these lesions may progress in the long-term, leading to further pain and disability. While shoulder arthroplasty remains a viable option in older patients with glenohumeral arthritis, concerns over component longevity and loosening in younger patients make it less attractive in that age group. Arthroscopic joint debridement with loose body removal, often with capsular release, has been successful in select, more sedentary patients. More recent techniques, including autologous chondrocyte implantation (ACI), osteochondral grafting (allograft versus autograft), interpositional arthroplasty, and microfracture surgery, have been evaluated for use in the shoulder. These procedures have experienced success in weight bearing joints, including the knee and ankle. Despite the good clinical results in the shoulder with short-term follow-up reported in some small series, the treatment of chondral injuries in the glenohumeral joint remains a challenging problem.

Keywords Glenohumeral arthritis · Microfracture · Autogenous chondrocyte transfer · Osteochondral graft · Interpositional arthroplasty · Sports non-ACL

Introduction

Articular cartilage damage, ranging from focal lesions to a global loss of cartilage, is a common source of pain and disability in the aging population. While these occur most frequently in weight-bearing joints such as the hip, knee, and ankle, they have also been found to occur in the shoulder. Symptomatic chondral lesions of the glenohumeral joint have been reported to have an incidence of 5% to 17% [1, 2]. They often arise secondary to trauma, recurrent shoulder instability, and prior surgical intervention [3–5]. Frequently, they are found incidentally during open or arthroscopic joint exploration for other shoulder pathology such as a rotator cuff or labral tear [3, 6, 7]. These lesions are particularly problematic because of their virtual absence of a viable blood supply and reserve of pluripotent cells. The body's natural response to trauma is therefore absent, making spontaneous healing impossible. If untreated, these lesions may progress to degenerative osteoarthritis [8, 9]. The use of total shoulder arthroplasty for glenohumeral osteoarthritis provides excellent symptomatic relief; however, concern over glenoid component loosening, as well as decreased satisfaction rates, make this procedure a last resort option for younger, more active patients [10]. Consequently, focus has shifted to non-arthroplasty techniques to repair or reconstruct the articular surface of the joint. These techniques have previously been categorized as palliative, reparative, restorative, or reconstructive [4].

Autologous chondrocyte implantation, osteochondral autograft and allograft transplantation, interposition arthroplasty,

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and microfracture comprise the major non-arthroplasty techniques currently under investigation. These procedures may be performed either open or arthroscopically and have been adapted from their use in other joints, in particular the knee. Increasing interest has been shown in the use of microfracture surgery. Its success rates in the knee (approaching 95%) [11, 12] and relatively low morbidity have made it a popular technique for focal unipolar humeral head and glenoid lesions. The glenohumeral joint represents a unique situation given the relatively thin cartilage of the humeral head, with a mean thickness reported from 0.89 mm [13] to 1.44 mm [14]. While published outcomes of each individual procedure have demonstrated promising results, no long-term comparative studies are available. As such, no consensus for treatment exists amongst orthopaedic surgeons when faced with chondral injuries in this challenging subset of patients.

Clinical examination

Chondral lesions of the glenohumeral joint are especially difficult to diagnose using common clinical examination techniques. Furthermore, patients rarely present with isolated cartilage lesions of the shoulder. Oftentimes, they have coexisting injuries, such as rotator cuff tears, that contribute to their pain and disability [3, 6, 7]. Typically, they complain of non-specific symptoms of pain, swelling, catching, and clicking. Any history of trauma or prior surgical intervention on the shoulder should be noted. All patients should undergo a comprehensive physical examination to evaluate for other more common causes for shoulder pain. In particular, range of motion of the affected shoulder should be compared with the contralateral side. Glenohumeral chondral lesions may be clinically confusing because they tend to mimic other pathologies, especially impingement syndrome. The “compression-rotation” test was developed as a means to help differentiate between the two [15]. The patient is placed on the normal side in the lateral decubitus position, with the symptomatic arm at their side and the elbow flexed. The examiner then compresses the affected humeral head into the glenoid and has the patient internally and externally rotate their shoulder. A positive result occurs when pain is elicited during rotation. The specificity of the test is increased after subacromial injection of a local anesthetic (Neer impingement test); if there is no longer pain during forward flexion, but the compression-rotation test remains positive [15], a chondral lesion should be suspected.

Imaging

Standard diagnostic imaging is safer and more cost effective than arthroscopy, but it frequently fails to diagnose

glenohumeral cartilage lesions. Radiographs of the shoulder are an appropriate first imaging step; however, radiographic changes associated with osteoarthritis, such as joint space narrowing and subchondral cysts, typically do not appear until late in the disease process [6]. Computed tomographic scans with 3D reconstruction may be useful in select cases to better appreciate the underlying bony architecture. Magnetic resonance imaging (MRI) should be the next imaging modality obtained if there is suspicion for a chondral lesion, because it allows evaluation of both chondral and soft tissue pathology. While it has been shown to be useful in elucidating chondral lesions of the knee where the articular cartilage is much thicker (3–4 mm) [16, 17], it often fails to accurately diagnose glenohumeral lesions. Guntern et al [18] found only a fair association in the interobserver ability to diagnose glenohumeral articular cartilage lesions based on magnetic resonance arthrography in patients with impingement syndrome. Most errors in diagnosis in this study were false positives, indicating an overestimation of cartilage lesions based on MR arthrography.

The difficulty in accurately determining articular cartilage thickness on the humeral head has been demonstrated with the use of MRI. In a cadaveric study, Hodler et al [19] found that various routine MR sequences both with and without contrast tended to overestimate cartilage thickness on the humeral head in areas of thin cartilage (periphery), and to underestimate this measurement in areas of thick cartilage. Furthermore, areas in which there was no anatomic presence of articular cartilage tended to have up to 1.6 mm of cartilage measured from the imaging. Using cadaveric shoulders, Yeh et al [20], similarly found that MR arthrography tended to overestimate thin cartilage and underestimate thick cartilage on the humeral head. Interestingly, the accuracy of the measurements was significantly better for the glenoid than for the humeral head.

Currently, the gold standard for diagnosing chondral lesions of the shoulder is via arthroscopy. It allows for direct visualization and size measurement of the lesion with limited morbidity. Additionally, it allows for a more thorough treatment of any concomitant shoulder pathology. As with the knee, chondral lesions of the shoulder are most often classified according to the Outerbridge classification system [21]. Lesions are scored from 0 to IV by increasing severity. Grade 0 refers to normal cartilage; I to cartilage softening and swelling; II to fragmentation and fissuring involving up to half the depth of the cartilage; III to fragmentation and fissuring involving more than half the depth of the cartilage; and IV to cartilage loss reaching or going through the subchondral plate. The lesions have been described as unipolar (affecting one side of the joint) versus bipolar (affecting both the humeral head and glenoid).

Nonoperative treatment

Individuals with confirmed symptomatic chondral lesions in the shoulder should be treated nonoperatively initially. Most commonly, patients may be managed with a regimen of nonsteroidal, anti-inflammatory medications. Intra-articular corticosteroid injections are also reasonable options, but only after patients have failed a trial of oral pharmacotherapy. The liberal use of intra-articular cortisone injections, alone or in combination with local anesthetics, should be avoided given recent evidence for chondrotoxicity of these agents. [22] Physical therapy, as an adjunct to pain medication, may also be utilized, with the goal of maintaining/improving their range of motion and providing symptomatic relief [5]. Viscosupplementation, currently FDA approved for use in the knee, is being investigated for use in the shoulder. Blaine et al [23], in a randomized controlled trial of 660 patients with persistent shoulder pain of various etiologies, found that patients who were managed with intra-articular injections of sodium hyaluronate had significantly greater pain relief than the controls (saline solution). Patients receiving 5 weekly injections also had greater decrease in pain as the study progressed. Both Silverstein et al [24] and Noel et al [25•] examined the effects of intra-articular injections of Hylan G-F 20 for the treatment of symptomatic shoulder osteoarthritis. Silverstein et al noted significant improvements in shoulder function, while both reports noted significant improvements in pain level.

Operative treatment

Non-arthroplasty options in the management of glenohumeral chondral lesions include palliative (debridement), reparative (microfracture/abrasion chondroplasty), restorative (ACI/MACI and osteochondral allograft and autograft transfer), and reconstructive (interposition arthroplasty and prosthetic resurfacing) procedures. (Table 1) Patient characteristics—age, activity level, occupation, and coexisting pathology—determine which surgical treatment may be most appropriate. Palliative treatment via debridement, capsular release, bursectomy, and/or acromioplasty has been reported following failure of nonoperative management [6, 26–30]. The goal of palliative treatment is not to repair the cartilage lesions, but to remove damaged flaps of cartilage or loose bodies in order to provide pain relief and functional gains (Fig. 1a). These patients typically have lower activity demands and tend to be older. While not a permanent solution to glenohumeral arthritis, it can be used as a means to delay permanent prosthetic management.

Abrasion chondroplasty/microfracture

Cartilage itself lacks the vascularity that would allow it to heal spontaneously following injury. It has been demonstrated, however, that if cartilage damage extends into and beyond the subchondral bone, the resultant bleeding and release of marrow elements can possibly provide the ideal environment for fibrocartilage interposition [9]. This concept is exploited in two similar procedures: abrasion chondroplasty and microfracture. Abrasion chondroplasty requires removal of the superficial layer of subchondral bone over the entirety of the cartilage lesion, commonly using a motorized shaver or burr. This creates an area of roughened and bleeding bone. A clot forms over the roughened surface and is potentially remodeled into a layer of fibrocartilage. As with microfracture, a strict rehabilitation program must be employed in order to prevent clot dislodgement and to promote cell maturation/differentiation. [31] While these two procedures promote similar concepts, microfracture surgery has become more popular and merits further attention.

The technique, indications, and contraindications of microfracture in the shoulder mirror those for the knee. The ideal patient has a focal, unipolar Outerbridge grade IV lesion that does not violate the subchondral plate and has failed conservative therapy. Patients who have partial-thickness lesions, bipolar lesions, lesions associated with bony defects, systemic immunocompromise such as rheumatoid arthritis, and crystalline deposition diseases, should not undergo this procedure. The goal of microfracture is to release pluripotent cells from the marrow via small holes forming a subsequent clot. Through a strict rehabilitation program, the marrow clot eventually matures into fibrocartilage [12]. Operative positioning of the patient depends on surgeon preference. Lesion location determines portal placement. Anterosuperior glenoid lesions are accessed most effectively via a more laterally placed anterior portal. More inferior lesions require lower portals placed just proximal to the subscapularis. A posterior 7-o'clock portal is useful for posterior glenoid lesions. Humeral lesions can be treated using a standard anterior portal and simply rotating the arm. Portal placement is critical to achieving perpendicular orientation to the joint surface.

Following portal placement, any concomitant shoulder pathology is addressed prior to initiating microfracture. This ensures visualization of the marrow elements and prevents clot dilution. Once the lesion is identified, an arthroscopic shaver is used to remove cartilage flaps to confirm that the lesion is contained. A vertical wall of viable cartilage surrounding the lesion to contain the clot is created using curettes. The calcified cartilage layer should be abraded with care not to penetrate the subchondral bone. The roughened surface facilitates adherence of the clot to the joint surface.

Table 1 Outcomes following non-arthroplasty treatment for glenohumeral cartilage defects

Study	No. of patients	Type of treatment	Assessment Measures	Mean Follow-up (months)	Outcomes
Romeo et al ³⁸	1	ACI	Pain, ROM	12	Painless full ROM
Connor et al ⁴²	1	OAT	ROM	24	Returned to work at 3 months without limitations ROM completely restored with no posterior instability
Ivkovic et al ⁴³	1	OAT	CMS, Pain, ROM	36	Autograft shoulder generally painless ROM markedly increased Allograft CMS: 86 Allograft CMS: 86 Hemiarthroplasty CMS: 55
Scheibel et al ⁴¹	8	OAT	CMS	32.6	CMS significantly improved Six patients completely pain free and returned to full work and sport activities
Frank et al ¹	16 (17 shoulders)	Microfracture	ASES, ROM, SANE, SF12, SST, UCLA, VAS	27.8	ASES, SST, VAS, ROM significantly improved Postop SANE=85.6 UCLA=32.4 respectively Mean CMS=73.2
Millett et al ³⁶	30 (31 shoulders)	Microfracture	ASES, Satisfaction, VAS	47	Humeral head only lesions greatest ASES increase Worse outcomes when ≥2 additional pathologies and history prior surgery Lesion size and ASES negatively correlated Prior surgery is predictor of lower ASES scores
Siebold et al ⁸	5	Microfracture	CMS	25.8	CMS significantly increased in all patients
Savoie et al ⁵⁷	23	Interposition Arthroplasty	ASES, CMS, Rowe, ROM, UCLA, VAS	36–72	ASES, CMS, UCLA, Rowe, VAS, ROM, and 6/8 SF12 significantly increased Overall success rate: 75%

ROM = Range of Motion; CMS = Constant—Murley Score; ASES = American Shoulder and Elbow Score; SF12 = Short-Form 12; SANE = Single Assessment Numeric Evaluation; SST = Simple Shoulder Test; UCLA = University of California, Los Angeles; VAS = Visual Analog Scale; OAT = Osteochondral Autograft Transfer

Arthroscopic awls with varying angulation (30°, 45°, and 90°) are used to penetrate the subchondral plate. The initial holes should be placed at the periphery of the lesion. (Fig. 1b) Subsequent holes are placed 3–4 mm apart and 2–3 mm deep. Spacing is crucial to prevent fracture between holes. The arthroscopic pump pressure is decreased to confirm the release of marrow elements. (Fig. 1c) Postoperative rehabilitation is important for clot maturation. Patients are placed in continuous passive motion (CPM) devices following knee microfracture surgery [32–34]. CPM induces the production of synovial fluid, thus stimulating the differentiation and maturation of the pluripotent cells in the clot. Patients also maintain a toe-touch weight bearing status. The shoulder does not

require similar limitations in weight bearing. Further, CPM is not used following shoulder microfracture surgery. Patients are encouraged to begin pendulum exercises immediately. Current regimens recommend 600–800 rotations per day for the first 6 weeks [1, 35]. Therapy progresses to light weight training and stretching for the next six weeks and ultimately unrestricted weight training at 12 weeks. Full activity can be resumed at 16 weeks. Certainly, any associated pathology addressed during the surgical procedure may influence postoperative management.

The few reports on outcomes following glenohumeral microfracture have demonstrated promising results. Frank et al [1] retrospectively evaluated outcomes of 16 patients

Fig. 1 Intra-operative images. **a** View from superolateral portal demonstrating an Outerbridge 4 chondral lesion on the anterior-inferior glenoid rim, and illustrating the difficulty in establishing vertical walls in the glenohumeral joint. **b** Debridement of an Outerbridge 4 chondral lesion of the inferior glenoid using a motorized shaver in preparation of microfracture. **c** Confirmation of marrow element release following microfracture by reducing fluid inflow

(17 shoulders) with a mean age of 37 years. An isolated glenoid lesion was found in six shoulders, an isolated humeral lesion in ten shoulders, and one shoulder had a bipolar lesion. The mean size of the humeral lesions was 5.07 cm² and the mean size for the glenoid lesions was 1.66 cm². The authors reported significant improvements in VAS, ASES, and SST scores. Statistically significant improvements in abduction and external rotation were found during the clinical evaluation of eight patients who underwent a clinical exam. Three patients required subsequent shoulder surgery, including osteochondral allograft placement and hemiarthroplasty and were deemed clinical failures. Overall, 92% of patients felt that they would undergo microfracture surgery again.

Similar findings were reported by Millett et al [36•] in a retrospective review of 30 patients (31 shoulders). The VAS score, ASES score, and range of motion all improved significantly at a mean follow-up of 47 months. In their series, 13 patients had isolated glenoid lesion (mean size 137 mm²), 12 had isolated humeral lesions (mean size 422 mm²) and 6 had combined humeral and glenoid lesions (mean size 442 mm² and 273 mm², respectively). Patients who had two or more concomitant procedures performed in addition to microfracture had outcome scores lower than those who did not. Furthermore, prior shoulder surgery was found to predict a lower postoperative ASES score. Lesion location was also found to be an important predictor of ASES score improvement. Patients who had microfracture surgery for humeral head lesions alone showed the greatest improvement (+32 points), while patients who had combined humeral head and glenoid lesions or glenoid lesions alone showed less improvement (+19 and +17 points respectively). Three of the six patients who underwent subsequent shoulder surgery required total shoulder replacements and were deemed failures.

Sieblod et al [8] prospectively evaluated five patients with Outerbridge IV humeral lesions with a mean size of 311 mm² who underwent a combination of microfracture surgery and a periosteal flap harvested from the proximal humerus. At a mean follow-up of 26 months, they found significant improvements in both Constant and VAS scores. The ability to perform activities of daily living also improved, although not significantly. The authors did not find any significant improvement in shoulder range of motion. Three patients also underwent a second-look arthroscopy, which showed a significant reduction of the lesion size.

Autologous cartilage implantation (ACI)

Autologous cartilage implantation (ACI) is a two-stage procedure whereby healthy articular cartilage is harvested, cultured and expanded, and implanted more than 3–4 weeks later [5, 37]. Patients with large lesions not suitable for other reparative techniques and who have failed conservative management may be appropriate for this technique. ACI is time consuming, expensive, and requires multiple surgeries to complete. To date, it has been used almost exclusively in the knee joint. The current literature on ACI for the shoulder is limited to a single case report of a 16-year-old male overhead athlete with a large, full-thickness lesion (3.3×1.5 cm) of the humeral head. Following the procedure, the patient regained painless full range of motion at 12 months [38].

Recent technical advances include matrix-induced autologous chondrocyte implantation (MACI) [38]. Chondrocytes are cultured and grown directly onto type I and III collagen membranes. The membranes with cultured cells can then be

glued directly to the defect rather than the surrounding healthy tissue. It reduces the possibility of cell leakage, which can occur when the overlying patch seal is not watertight. Arthroscopic second-look surgeries have revealed normal-appearing or near-normal-appearing cartilage at the lesions [39]. This procedure has yet to be applied to lesions in the shoulder.

Osteochondral autograft transfer (OAT)

Osteochondral autograft transfer (OAT) is used in small lesions that have failed first-line therapy. Promising results have been shown in the knee and ankle joints [40]. The procedure involves harvesting cylindrical grafts of bone and cartilage from non-weight bearing surfaces (such as the femoral condyle) and transplanting them to areas of cartilage loss. It requires only one stage to complete. Scheibel et al [41] performed this technique on eight patients (seven humeral lesions, one glenoid) with Outerbridge IV lesions. Grafts were taken from the lateral femoral condyle. At a mean follow-up of 32.6 months, all patients noted significantly less pain and increased ability to perform activities of daily living. Only two patients were unable to return to full occupational and recreational levels. MRI revealed congruent cartilage surfaces and excellent graft viability and consolidation in all but one patient. There were no complications reported as a result of the procedure. One patient, however, had persistent donor site pain and recurrent effusions necessitating two subsequent arthroscopies. Donor site morbidity, while unusual, is a serious complication that the surgeon needs to take into account.

Two case reports also report on the application of this procedure to humeral head lesions. Connor et al [42] and Ivkovic et al [43] both reported on patients who received OAT following traumatic posterior shoulder dislocations. Both patients underwent hemiarthroplasty in one shoulder, while the other less deformed joint received autograft from the contralateral humeral head. The articular surfaces were reconstructed in both patients. Both cases resulted in excellent clinical and radiographic results.

Osteochondral allograft implantation

Allograft transplantation is becoming a viable option for the repair of various musculoskeletal disorders [44]. The technique works similar to autograft transplantation with two major advantages: a lack of donor site morbidity and its application to more extensive defects. Grafts are typically size-matched and side-matched, although they may come from unrelated bones, and do not require immediate transplantation. It is recommended that grafts be stored and transplanted within 21 days [45], but no later than 28 days following graft harvesting [3, 4]. Beyond this time point,

cell viability and density decreases precipitously [45]. While the use of allograft remains attractive, clinical studies on this technique are limited mostly to its use in large Hill—Sachs lesions for recurrent dislocators [46–50]. This procedure carries the potential for disease transmission [51]. However, due to the advances in transmissible disease screening, there have been no cases of HIV transmission from an allograft reported since 1985 [5].

Arthroscopic interposition arthroplasty

Arthroscopic interposition arthroplasty employs biologic and synthetic acellular materials to resurface the glenoid. These materials serve as a scaffold for cellular infiltration by native fibroblasts and vascular tissue with minimal inflammation [52]. The procedure is most frequently performed in association with hemiarthroplasty of the humeral head [53–56, 57•]. Krishnan and colleagues [54], using Achilles tendon allograft, anterior capsule, or fascia lata autograft, reported significant increases in ASES scores and functional outcomes. Satisfactory results were reported in 31/36 shoulders at a mean follow-up of two to 15 years. The majority of unsatisfactory results were in patients receiving anterior capsule grafts. Nicholson et al [55] used meniscal allograft to resurface the glenoid in 30 young patients undergoing hemiarthroplasty. Significant improvements in ASES, VAS, and range of motion were reported after a mean follow-up of 18 months. Others reported similar clinical outcomes.. [56] In contrast, Elhassan et al [57•] reported no significant changes in VAS, Constant—Murley, or subjective scores in a series of 13 patients undergoing hemiarthroplasty with meniscal allograft. Eleven patients continued to have severe pain and ten progressed to revision total shoulder arthroplasty at a mean of 16 months. At revision, the graft was found to be completely replaced by scar tissue.

In an investigation of glenoid resurfacing with porcine allograft without concomitant hemiarthroplasty in a series of 23 patients with end stage glenohumeral arthritis, Savoie et al [58•] noted significant improvements in ASES, UCLA, and Constant—Murley scores at final follow-up. In addition, all SF-12 subset scores except general health and role-emotional improved significantly. Five patients ultimately proceeded to surface replacement arthroplasty, with 15 patients remaining satisfied at final follow-up.

Partial joint resurfacing

Partial joint resurfacing is a fairly new technique. Rather than replacing the entire articular surface of the humeral head, the procedure simply replaces a segment. These prostheses are stemless, which leaves most of the proximal humeral anatomy intact. This provides the surgeon with

multiple options for future prosthetic resurfacing if necessary. These implants are available in various sizes and allow the surgeon to better fit the contours of the humeral head. They may be used as an alternative to osteochondral allograft and autograft transfers, particularly with large engaging Hill—Sachs lesions [59, 60]. While these implants offer an attractive option for younger patients, there have been no outcome studies presented in the literature.

Authors' preferred treatment

The senior author encourages careful history taking and physical examination of any patient in whom a chondral injury is suspected. Conservative management of these injuries should be attempted as first-line treatment. In older, sedentary patients, consideration may be given to debridement of any loose cartilage flaps, while addressing other shoulder pathology including capsular contracture. Because only a few small series of the treatment of chondral lesions in the glenohumeral joint are currently available, only general guidelines for operative management can be provided. In the case of a smaller focal unipolar lesion of the glenoid or humeral head, microfracture of the lesion is recommended, utilizing a technique as outlined above. In addition, the OAT procedure may also be considered for smaller lesions. For larger lesions, osteochondral grafting or ACI may be considered. When faced with bipolar lesions, consideration should be given to reconstructive techniques, including interposition arthroplasty to resurface the glenoid. The humeral head lesion should then be addressed based on the individual characteristics of that lesion.

Conclusion

Articular cartilage lesions of the glenohumeral joint are difficult to identify and manage. They are often overlooked due to concomitant shoulder pathology and their non-specific presentation. Often, they are encountered during open or arthroscopic treatment for other shoulder problems. Initial management includes conservative treatment with oral pharmacotherapy and physical therapy. If conservative management fails and the lesions are left untreated, they can progress, leading to permanent deformity, loss of function, and pain. Shoulder arthroplasty for these lesions allows for symptomatic relief and functional improvement. However, this is not necessarily the best option for younger patients, in whom the implant longevity may be an issue. Non-arthroplasty procedures like microfracture surgery, osteochondral autograft and allograft transplant, and autologous chondrocyte implantation allow surgeons to restore or repair the joint anatomy, while preserving the surgeon's ability to

perform more definitive procedures like arthroplasty at a later date. These techniques have only recently been applied towards lesions in the shoulder and little literature exists on their outcomes. The results of the case reports and small retrospective reviews have been encouraging. Future case-control studies are necessary in order to determine the true long-term efficacy of these procedures in the management of glenohumeral articular cartilage defects.

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References

Papers of particular interest, published recently, have been highlighted as:

- Of importance

1. • Frank RM, Van Thiel GS, Slabaugh MA, et al. Clinical outcomes after microfracture of the glenohumeral joint. *Am J Sports Med.* 2010;38:772–81. *Retrospective review of outcomes of microfracture surgery of the shoulder in 14 patients. Patients displayed significant improvements in subjective pain and functional status and objective shoulder range of motion.*
2. Elser F, Braun S, Dewing CB, Millett PJ. Glenohumeral joint preservation: current options for managing cartilage lesions in young, active patients. *Arthroscopy.* 2010;26:685–96.
3. McCarty III LP, Cole BJ. Nonarthroplasty treatment of glenohumeral cartilage lesions. *Arthrosc.* 2005;21:1131–42.
4. Cole BJ, Yanke A, Provencher MT. Nonarthroplasty alternatives for the treatment of glenohumeral arthritis. *J Shoulder Elbow Surg.* 2007;16(Suppl):231S–40.
5. Provencher MT, Barker JU, Strauss EJ, et al. Glenohumeral arthritis in the young adult. *Instr Course Lect.* 2011;60:137–53.
6. Cameron BD, Galatz LM, Ramsey ML, Williams GR, Iannotti JP. Non-prosthetic management of grade IV osteochondral lesions of the glenohumeral joint. *J Shoulder Elbow Surg.* 2002;11:25–32.
7. Page RS. Managing chondral lesions of the glenohumeral joint. *Int J Shoulder Surg.* 2008;2:77–8.
8. Siebold R, Lichtenberg S, Habermeyer P. Combination of microfracture and periosteal-flap for the treatment of full thickness articular cartilage lesions of the shoulder: a prospective study. *Knee Surg Sports Traumatol Arthrosc.* 2003;11:183–9.
9. Mankin HJ. The response of articular cartilage to mechanical injury. *J Bone Joint Surg Am.* 1982;64:460–6.
10. Sperling JW, Cofield RH, Rowland CM. Neer hemiarthroplasty and neer total shoulder arthroplasty in patients fifty years old or less. Long-term results. *J Bone Joint Surg Am.* 1998;80:464–73.
11. Miller BS, Steadman JR, Briggs KK, et al. Patient satisfaction and outcome after microfracture of the degenerative knee. *J Knee Surg.* 2004;17:13–7.
12. Steadman JR, Briggs KK, Rodrigo JJ, et al. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. *Arthrosc.* 2003;19:477–84.
13. Fox JA, Cole BJ, Romeo AA. Articular cartilage thickness of the humeral head: an anatomic study. *Orthopedics.* 2008;31:216.
14. Soslowky LJ, Flatow EL, Bigliani LU, Mow VC. Articular geometry of the glenohumeral joint. *Clin Orthop Relat Res.* 1992;285:181–90.

15. Ellman H, Harris E, Kay SP. Early degenerative joint disease simulating impingement syndrome: Arthroscopic findings. *Arthrosc.* 1992;8:482–7.
16. Hall FM, Wyshak G. Thickness of articular cartilage in the normal knee. *J Bone Joint Surg Am.* 1980;62:408–13.
17. Cohen ZA, McCarthy DM, Kwak SD, et al. Knee cartilage topography, thickness, and contact areas from MRI: in-vitro calibration and in-vivo measurements. *Osteoarthr Cartil.* 1999;7:95–109.
18. Guntern DV, Pfirrmann CWA, Schmid MR, et al. Articular cartilage lesions of the glenohumeral joint: diagnostic effectiveness of MR arthrography and prevalence in patients with subacromial impingement syndrome. *Radiology.* 2003;226:165–70.
19. Hodler J, Loreda RA, Longo C, et al. Assessment of articular cartilage thickness of the humeral head: MR-anatomic correlation in cadavers. *Am J Roentgenol.* 1995;165:615–20.
20. Yeh LR, Kwak S, Kim YS, et al. Evaluation of articular cartilage thickness of the humeral head and the glenoid fossa by MR arthrography: anatomic correlation in cadavers. *Skeletal Radiol.* 1998;27:500–4.
21. Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br.* 1961;43:752–7.
22. Braun HJ, Fogel NW, Kim HJ, et al. The effect of local anesthetic and corticosteroid combinations on chondrocyte viability. *Knee Surg Sports Traumatol Arthrosc* 2011, Epub ahead of print.
23. Blaine T, Moskowitz R, Udell J, Skyhar M, et al. Treatment of persistent shoulder pain with sodium hyaluronate: A randomized, controlled trial. *J Bone Joint Surg Am.* 2008;90:970–9.
24. Silverstein E, Leger R, Shea KP. The use of intra-articular hylan G-F 20 in the treatment of symptomatic osteoarthritis of the shoulder. A preliminary study. *Am J Sports Med.* 2007;35:979–85.
25. • Noël E, Hardy P, Hagen FW, et al. Efficacy and safety of Hylan G-F 20 in shoulder osteoarthritis with an intact rotator cuff. Open-label prospective multicenter study. *Joint Bone Spine.* 2009;76:670–3. *Prospective evaluation of patients receiving intra-articular injections of Hylan G-F 20 for shoulder osteoarthritis. There was a significant improvement in pain in the subgroup of patients requiring only 1 injection. Product safety was demonstrated via the minimal adverse reactions/effects..*
26. Weinstein DM, Bucchieri JS, Pollock RG, et al. Arthroscopic debridement of the shoulder. *Arthrosc.* 2000;16:471–6.
27. Safran MR, Baillargeon D. The role of arthroscopy in the treatment of glenohumeral arthritis. *Sports Med Arthrosc Rev.* 2004;12:139–45.
28. Guyette TM, Bae H, Warren RF, et al. Results of arthroscopic subacromial decompression in patients with subacromial impingement and glenohumeral degenerative joint disease. *J Shoulder Elbow Surg.* 2002;11:299–304.
29. Ellowitz AS, Rosas R, Quick DC, et al. The benefit of arthroscopic subacromial decompression for impingement in patients found to have unsuspected glenohumeral osteoarthritis. *Orthop Trans.* 1997;21:1135–6.
30. Bishop JY, Flatow EL. Management of glenohumeral arthritis: a role for arthroscopy? *Orthop Clin North Am.* 2003;34:559–66.
31. Johnson LL. Arthroscopic abrasion arthroplasty. A review. *Clin Orthop Relat Res.* 2001;391(Suppl):S306–17.
32. Steadman JR, Rodkey WG, Singleton SV, Briggs KK. Microfracture technique for full-thickness chondral defects: technique and clinical results. *Oper Tech Orthop.* 1997;7:300–4.
33. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res.* 2001;391(Suppl):362–9.
34. Sledge SL. Microfracture techniques in the treatment of osteochondral injuries. *Clin Sports Med.* 2001;20:365–77.
35. Salata MJ, Kercher JS, Bajaj S, Verma NN, Cole BJ. Glenohumeral microfracture. *Cartil.* 2010;1:121–6.
36. • Millett PJ, Huffard BH, Horan MP, et al. Outcomes of full-thickness articular cartilage injuries of the shoulder treated with microfracture. *Arthrosc.* 2009;25:856–63. *Retrospective review of 30 patients receiving microfracture surgery for articular cartilage lesions in the shoulder. Lesion location, size, and prior surgery all correlated with ASES scores. Worse outcomes were noted when 2 or more additional pathologies were also addressed..*
37. Peterson L, Minas T, Brittberg M, Lindahl A. Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation. Results at two to ten years. *J Bone Joint Surg Am.* 2003;85:17–24.
38. Romeo AA, Cole BJ, Mazzocca AD, et al. Autologous chondrocyte repair of an articular defect in the humeral head. *Arthrosc.* 2002;18:925–9.
39. Enea D, Cecconi S, Buisliacchi A, et al. Matrix-induced autologous chondrocyte implantation (MACI) in the knee. *Knee Surg Sports Traumatol Arthrosc* 2011, Epub ahead of print.
40. Hangody H, Füles P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints. *J Bone Joint Surg Am.* 2003;85(Supplement 2):25–32.
41. Scheibel M, Bartl C, Magosch P, et al. Osteochondral autologous transplantation for the treatment of full-thickness articular cartilage defects of the shoulder. *J Bone Joint Surg Br.* 2004;86:991–7.
42. Connor PM, Boatright JR, D'Alessandro DF. Posterior fracture-dislocation of the shoulder: Treatment with acute osteochondral grafting. *J Shoulder Elbow Surg.* 1997;6:480–5.
43. Ivkovic A, Boric I, Cicak N. One-stage operation for locked bilateral posterior dislocation of the shoulder. *J Bone Joint Surg Br.* 2007;89:825–8.
44. Ho JY, Miller SL. Allografts in the treatment of athletic injuries of the shoulder. *Sports Med Arthrosc Rev.* 2007;15:149–57.
45. Allen RT, Robertson CM, Pennock AT, et al. Analysis of stored osteochondral allografts at the time of surgical implantation. *Am J Sports Med.* 2005;33:1479–84.
46. Gerber C, Lambert SM. Allograft reconstruction of segmental defects of the humeral head for the treatment of chronic locked posterior dislocation of the shoulder. *J Bone Joint Surg Am.* 1996;78:376–82.
47. Yagishita K, Thomas BJ. Use of allograft for large Hill—Sachs lesion associated with anterior glenohumeral dislocation. A case report. *Injury.* 2002;33:791–4.
48. Chapovsky F, Kelly 4th JD. Osteochondral allograft transplantation for treatment of glenohumeral instability. *Arthrosc.* 2005;21:1007.e1–4.
49. Kropf EJ, Sekiya JK. Osteoarticular allograft transplantation for large humeral head defects in glenohumeral instability. *Arthrosc.* 2007;23:322.e1–5.
50. McCarty LP, Cole BJ. Reconstruction of the glenohumeral joint using a lateral meniscal allograft to the glenoid and osteoarticular humeral head allograft after bipolar chondrolysis. *J Shoulder Elbow Surg.* 2007;16:e20–4.
51. Buck BE, Malinin TI, Brown MD. Bone transplantation and human immunodeficiency virus. An estimate of risk of acquired immunodeficiency syndrome (AIDS). *Clin Orthop Relat Res.* 1989;240:129–36.
52. Adams JE, Steinmann SP. Soft tissue interposition arthroplasty of the shoulder. *J Shoulder Elbow Surg.* 2007;16(Suppl): 254S–60.
53. Burkhead WZ, Hutton KS. Biologic resurfacing of the glenoid with hemiarthroplasty of the shoulder. *J Shoulder Elbow Surg.* 1995;4:263–70.
54. Krishnan SG, Nowinski RJ, Harrison D, Burkhead WZ. Humeral hemiarthroplasty with biologic resurfacing of the glenoid for glenohumeral arthritis. *J Bone Joint Surg Am.* 2007;89:727–34.
55. Nicholson GP, Golstein JL, Romeo AA, et al. Lateral meniscus allograft biologic glenoid arthroplasty in total shoulder arthroplasty

- for young shoulders with degenerative joint disease. *J Shoulder Elbow Surg.* 2007;16(Suppl):261S–6.
56. Wirth MA. Humeral head arthroplasty and meniscal allograft resurfacing of the glenoid. *J Bone Joint Surg Am.* 2009;91:1109–19.
57. • Elhassan B, Ozbaydar M, Diller D, et al. Soft-tissue resurfacing of the glenoid in the treatment of glenohumeral arthritis in active patients less than fifty years old. *J Bone Joint Surg Am.* 2009;91:419–24. *Thirteen patients received glenoid resurfacing along with humeral head replacement. Overall outcomes were poor with 85% of patients having persistent severe pain and radiographic evidence of graft failure. 77% underwent total shoulder replacement at a mean of 16 months.*
58. • Savoie FH, Brislin KJ, Argo D. Arthroscopic glenoid resurfacing as a surgical treatment for glenohumeral arthritis in the young patient: Midterm results. *Arthrosc.* 2009;25:864–71. *Twenty-three patients received interposition arthroplasty without humeral head replacement. ASES, CMS, UCLA, Rowe, VAS, SF12, and range of motion all significantly improved. The procedure had a 75% overall success rate..*
59. Gomoll AH, McCulloch P, Kang RW, Cole BJ. Treatment of chondral defects in the shoulder. *Oper Tech Orthop.* 2006;16:232–43.
60. Scalise JJ, Miniaci A, Iannotti JP. Resurfacing arthroplasty of the humerus: Indications, surgical technique, and clinical results. *Tech Shoulder Elbow Surg.* 2007;8:152–60.