Review Article

Glenohumeral Osteoarthritis in the Young Patient

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Abstract

Glenohumeral osteoarthritis in the young patient (aged <60 years) is a difficult condition, given both age and functional demands. Primary osteoarthritis is the most common etiology in this patient demographic, but secondary causes include osteonecrosis, previous trauma (eg, fracture-dislocation), previous infection (eg, septic arthritis), previous capsulorrhaphy, previous arthroscopic surgery (eg, chondrolysis), and glenoid dysplasia. Nonsurgical modalities, including activity modification, pharmacotherapy, physical therapy, and intra-articular injections, are the mainstay of management; however, in young patients who have exhausted nonsurgical management, surgical options include arthroscopic débridement, humeral head replacement with or without glenoid treatment (ie, biologic glenoid resurfacing, glenoid reaming), and total or reverse total shoulder arthroplasty. Unfortunately, failure rates after surgical management are considerably higher in young patients compared with those observed in older, more sedentary patients. Here, we focus on the etiology, evaluation, and management of young patients with glenohumeral osteoarthritis, with a focus on clinical outcomes.

As the population ages, the incidence of glenohumeral osteoarthritis continues to increase. Elderly patients with osteoarthritis of the glenohumeral joint reproducibly have success with current, standard shoulder arthroplasty techniques; however, replacement options are less successful—and less appealing—to the younger patient with osteoarthritis because of limitations imposed postoperatively.[1] Young patients have frequently higher demands for activity and greater functional expectations that may preclude prosthetic replacement, and as a result, surgical and nonsurgical treatment options for this demographic have been pursued with varying results. Of note, the treating physician must consider physiologic and chronologic age when determining treatment algorithms for these patients.

Here, we focus on the etiology, evaluation, and management of young patients with glenohumeral osteoarthritis, with a focus on clinical outcomes.

Etiology

Consideration for the pathogenesis behind arthritic changes in the young patient may affect the surgeon's treatment algorithm and the patient's prognosis. Glenohumeral osteoarthritis can be divided into several etiologies but is broadly classified into primary and secondary osteoarthritis. Primary osteoarthritis affects a broad age range and is associated with posterior glenoid

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wear and progressive internal rotation contractures.[1,2] Although adults aged ≥60 years are typically afflicted with primary glenohumeral osteoarthritis, patients aged <60 years with advanced arthrosis often have more complex pathologies and secondary causes. Secondary forms of glenohumeral osteoarthritis include postinflammatory or infectious, posttraumatic, postoperative, and atraumatic osteonecrosis.[3] Patients aged <50 years are more likely to have diagnoses of posttraumatic arthritis, osteonecrosis, and capsulorrhaphy arthropathy.[4]

Glenohumeral dislocations and subluxations may result in secondary osteoarthritis via affected osteochondral fractures and subchondral bone injury to the glenoid and humeral head.[5] Recurrence of these instability events continues to damage the articular surface.[6,7]

Degenerative changes of the glenohumeral joint are not uncommon after fracture malunion of comminuted or displaced proximal humerus fractures—specifically for Neer three- and four-part fractures—as a result of aberrant joint biomechanics or posttraumatic osteonecrosis.[8] In addition, surgical hardware placed at the time of surgery for fracture fixation can lead to accelerated joint degeneration, as seen with screw cutout and joint breaching after proximal humerus fracture fixation.

Patients with a history of trauma, dysbarism, sickle cell disease, radiation therapy, oral steroid use, alcoholism, HIV, treatment with cytotoxic drugs, lipid metabolism disorders, and lysosomal storage disorders (including Gaucher disease) may be at risk of osteoarthritis resulting from atraumatic osteonecrosis.[1,3]

Iatrogenic causes during surgical intervention about the shoulder can also lead to osteoarthritic sequelae. Mechanisms include direct mechanical injury to the articular surface, such as the effect of the camera or arthroscopic devices against the cartilage, or thermal injury from the use of radiofrequency devices.[9] Rapid postarthroscopic glenohumeral chondrolysis is a rare complication in younger patients in which the articular cartilage undergoes a rapid, irreversible degeneration after shoulder arthroscopy. Implicated risk factors in this setting include indolent infectious origins, mechanical damage (ie, direct/traumatic injury, bioabsorbable suture anchors, knots), thermal injury, and chemical injury (ie, local anesthetic infusion catheters, decreased osmolarity of irrigation fluid).[10]

Overtightening of the capsule during instability surgery in the young patient may lead to pathologic glenohumeral biomechanics and a resultant increase in forces on the articular surface. Specifically, "capsulorrhaphy arthropathy" refers to the rapid posterior chondral wear due to overtightening of the anterior capsule and resultant compressive joint forces and loss of external rotation.[11]

Sequelae from a previously infected joint can lead to generalized degenerative changes as well. Less commonly, glenoid dysplasia may be at the root of otherwise unexplained osteoarthritic changes.

Patient Evaluation

History

In the setting of primary osteoarthritis, the presentation can be similar among affected individuals, independent of patient age. Patients may provide a history of pain with so-called deep motion and posteriorly on the shoulder and can report difficulty sleeping due to pain. Alternatively, osteoarthritic pain may be described as dull and diffuse at rest, yet sharp and stabbing with activities that bring the arm into an end–range of motion position. Range of motion is frequently limited. Conditions of loading such as sports or manual labor may exacerbate symptoms. Locking or catching with attempted motion can be indicative of a loose body in the joint or a partially detached osteoarticular fragment.[3] A careful history must assess for pain generators from additional pathology of shoulder joint anatomy; while complaints of weakness may suggest rotator cuff pathology and pain in the acromioclavicular joint indicates acromioclavicular joint pathology, differentiating between pain generated by other structures (ie, biceps, labrum, capsule, extra-articular anatomy) and by osteoarthritis of the joint can be difficult.

Clinicians should obtain a careful history delineating the onset, character, and duration of symptoms, as well as any previous history and timing of shoulder injuries, pathology, or surgical intervention. Descriptions of previous fractures, subluxations, or dislocations must be

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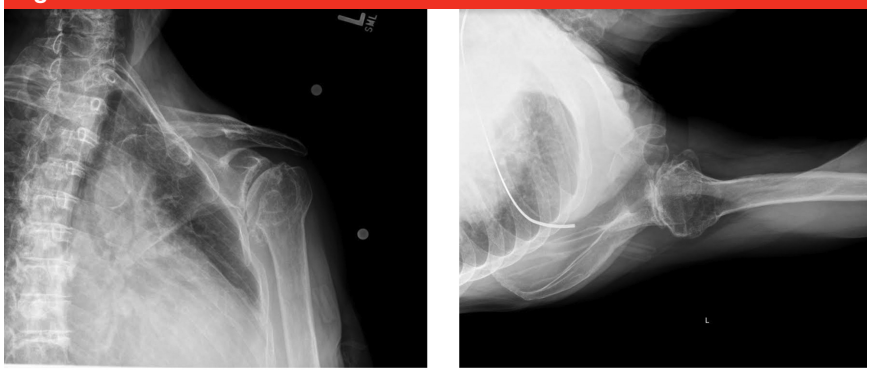
investigated. Obtaining surgical reports and intraoperative imaging for review can be helpful in the evaluation of patients who have previously undergone surgery. Finally, in determining a course of intervention, it is imperative to understand the patient's activity level, hobbies, occupation, and treatment goals because these can affect the surgeon's decision making. Whether the patient performs manual labor or participates in sports, for example, has implications for different therapeutic strategies.

Physical Examination

Examination will likely demonstrate limitations in range of motion, typically in forward flexion and external rotation, particularly when compared with the contralateral, unaffected side. Pain at the end ranges of the motion arc is typical of the osteoarthritic process. The passive and active motion arc should be documented. A formal neurologic and strength assessment of shoulder motions should be performed as well. Note should be made of previous surgical incisions, which can be pertinent for understanding the patient's history and for planning future surgery.

Figure 1



A B

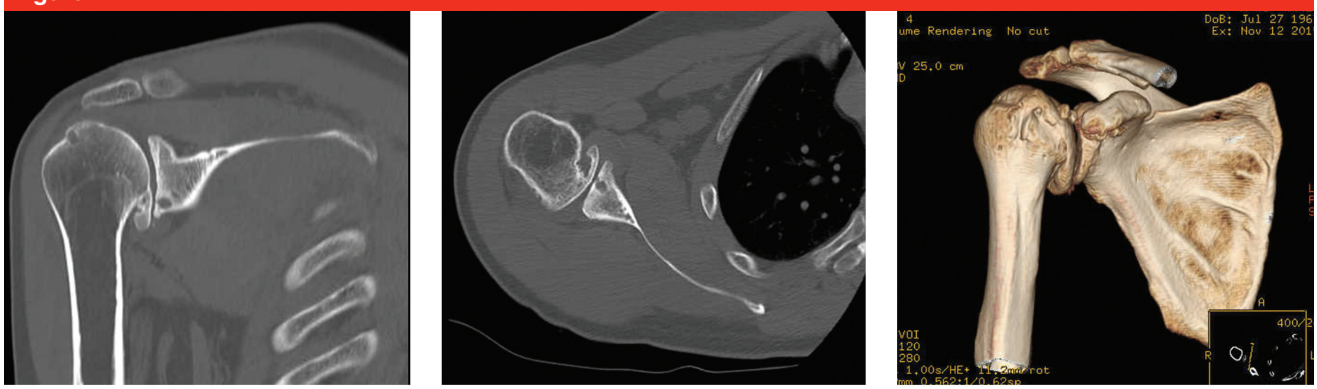
AP (**A**) and axillary plain (**B**) radiographs demonstrating findings typical of a young individual with left-sided glenohumeral osteoarthritis.

Imaging

First-line imaging includes plain radiographs of the glenohumeral joint with orthogonal views (ie, AP, axillary, scapular Y) to evaluate for symmetric joint space narrowing, subchondral cyst formation or sclerosis, and/or osteophyte formation (Figure 1). These images will help assess for a missed acute fracture or dislocation, which could lead to a similar symptomatic profile. Findings can also be characteristic of rotator cuff arthropathy with elevation of the humeral head or the absence of large osteophytes in the setting of rapid progressive arthritic changes seen after postoperative chondrolysis.[1]

Advanced imaging options including CT and MRI could be used as second-line means for visualizing greater detail on bony abnormality or deformity and a closer examination of the articular cartilage, respectively (Figure 2). Magnetic resonance arthrography is helpful to more closely evaluate the articular cartilage for osteochondral defects, the rotator cuff for partial or full tears, the glenoid labrum for tears, the biceps tendon for lesions, and for the presence of intra-articular loose bodies.

Figure 2



A B C

Coronal (**A**) and axial (**B**) CT scans and a three-dimensional CT reconstruction (**C**) from a patient with glenohumeral osteoarthritis, demonstrating fine bony detail of subchondral cyst formation, sclerosis, joint space narrowing, and osteophyte formation.

Treatment

Nonsurgical

Nonsurgical modalities, including activity modification, pharmacotherapy, physical therapy, and intra-articular injections, are the mainstay of conservative management. The treatment algorithm for the young, arthritic patient must additionally begin with patient education regarding the arthritic process. Changes in daily activities, sports participation, and/or occupation can be considered if these activities generate shoulder pain.

Pharmacotherapy

Oral NSAIDs, salicylates, and acetaminophen should be included in the initial conservative treatment scheme. However, many of these options have an adverse effect profile, which includes most commonly gastric irritation or renal toxicity, in addition to liver toxicity and cardiac disorders. Oral steroids can be used in an attempt to combat acute worsening of chronic pain from osteoarthritis. Narcotic medications have a limited role and should be used sparingly, if at all.

Physical Therapy

Physical therapy treatment includes manual physical therapy, exercises, and progressive functional activities tailored specifically to the patient's presentation. We feel that patients with less advanced arthritic changes and more preserved range of motion may be more likely to benefit from these options. Physical therapy should focus on periscapular and shoulder kinetics, strengthening, stretching, distraction, and range of motion improvements.[1,12] Of note, evidence on physical therapy for glenohumeral osteoarthritis is scarce, and in some patients, strengthening exercises may result in exacerbation of symptoms.

Intra-articular Injection

Intra-articular injection of local anesthetic alone (with lidocaine or marcaine) can be useful to confirm an intra-articular cause of the patient's symptoms. However, level I and II evidence to support the use of intra-articular corticosteroids in the glenohumeral joint is lacking. Much of its use in the shoulder is as a result of the efficacy demonstrated with use in the knee.[13] Viscosupplementation—although not FDA approved for use in the shoulder—similarly continues to receive increased attention for use in glenohumeral osteoarthritis, given reports of its efficacy with arthritis of the knee. Several other high-level studies have confirmed the efficacy and safety of hyaluronic acid preparations for symptomatic glenohumeral osteoarthritis, suggesting that it be considered as a part of the first-line treatment regimen of standard multimodal shoulder osteoarthritis.[14,15] However, other authors question whether its effect meets minimal clinically important differences.[16] Mesenchymal stem cells and autologous bone marrow concentrate have also shown promising results for the treatment of degenerative joint conditions, with early success in the glenohumeral joint, but additional studies are required before definitive conclusions can be drawn.[17,18] No outcomes studies have been published on the use of platelet-rich plasma and other more experimental therapies.

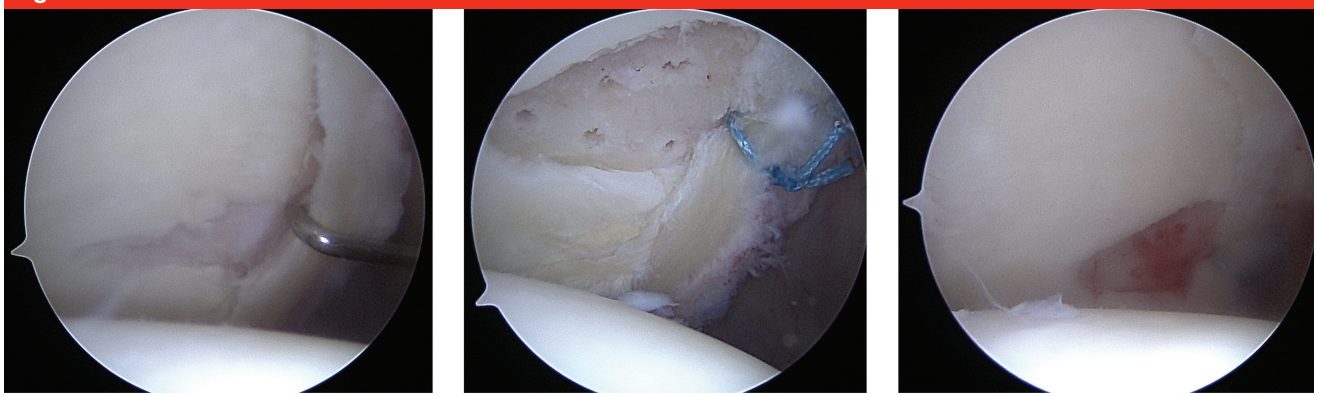
Surgical

The decision to operate on these young patients is particularly challenging because of higher patient expectations and greater durability needs of the reconstructive efforts.[1,19] The concerns for implant longevity in the face of increased life expectancy and higher levels of activity leave concerns for a particularly high risk of mid- and long-term complications with arthroplasty implants.[20,21] Although the ultimate goal is to provide durable pain relief with improvement in function and limited risk to the patient, the following should be considered in the surgical decision-making process when determining the optimal intervention: patient age, occupation, activity level and impact loads, sports participation, disease level, focality of disease (ie, unipolar versus bipolar, superficial versus deep), concomitant shoulder pathology, and patient expectations.[1] A wide range of treatment options exists, from simple arthroscopic débridement to total arthroplasty of the glenohumeral joint (see Table 1, Supplemental Digital Content 1, http://links.lww.com/JAAOS/A111).

Arthroscopic Débridement and Capsular Release

Recently, arthroscopic débridement procedures have received increased attention in this patient demographic because of the high rates of revision surgery with arthroplasty performed in young, active patients.[22] Patients with mild osteoarthritis are most likely to benefit from arthroscopic intervention. Thus, whereas arthroscopy is indicated for patients with minimal osteophyte formation and subchondral sclerosis or cysts,[23] outcomes tend to be worse in patients with bipolar disease. Although, some authors suggest that patient age, sex, duration of symptoms, previous surgical procedures, and radiographic stage of osteoarthritis may not correlate with successful arthroscopic débridement.[24] Coexisting pathology, including capsular contracture and subacromial inflammation, can be addressed at the time of arthroscopy as well with subacromial decompression and distal clavicle excision, removal of loose bodies, débridement of the labrum or chondral flaps, biceps tenotomy, synovectomy, capsular release, or osteophytectomy.[24] In general, arthroscopic intervention

Figure 3



A B C

Arthroscopic images demonstrating a contained glenoid chondral defect (**A**) and subsequent microfracture of the chondral defect (**B** and **C**).

allows for stabilization of chondral lesions and elimination of mechanical symptoms.[25] Arthroscopic lavage alone may be helpful in removing proinflammatory enzymes and proteins within the synovial fluid.[23]

Most patients can expect short-term relief, with a substantially lower risk of complications compared with humeral arthroplasty (HA) or total shoulder arthroplasty (TSA).[26] Arthroscopic débridement does not prevent osteoarthritic progression, but it may provide a temporizing option to avoid prosthetic replacement and allow earlier return to recreational activities and physically demanding occupations.[25]

The various techniques for arthroscopic procedures to treat glenohumeral arthritis are well described. Uniformly, authors suggest examination under anesthesia of the affected and unaffected extremities.[23] Capsular release should be considered in patients in whom there is a 20° side-to-side difference, particularly in external or internal rotation, because progressive arthritis can constrict the anterior capsule and alter joint contact forces, causing exponential arthritis progression.[1] Removal of loose chondral fragments from cartilaginous defects can help reduce mechanical irritation and inflammation of the synovium.[1] Chondroplasty should include débridement down to a stable rim of cartilage and subchondral bone; débridement of these irregular articular surfaces and frayed labrum may additionally reduce mechanical symptoms.[27] Inferior osteophytes may limit abduction by tensioning the axillary pouch and compressing the axillary nerve, which can contribute to posterior shoulder pain. Therefore, osteophyte removal and transcapsular axillary nerve decompression at the inferior glenohumeral capsule have been proposed to decrease pain.[25,28-30]

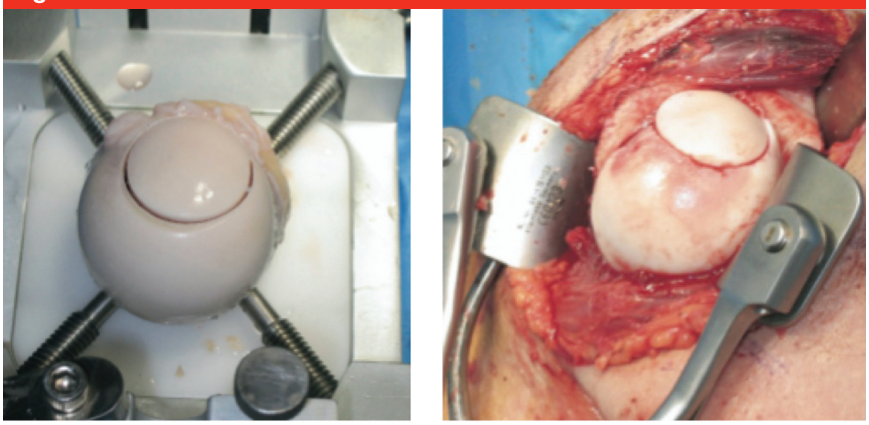
Millett and Gaskill[28] proposed a surgical technique called comprehensive arthroscopic management, which includes synovectomy, axillary nerve decompression and osteophytectomy, and inferior/anterior/posterior capsular release. Arthroscopic débridement has additionally been paired successfully with arthroscopic biological glenoid resurfacing with a biologic membrane.[31] Porcellini et al[32] reported on 47 patients between the ages of 30 and 55 years who underwent arthroscopic circumferential capsulotomy and either microfracture (36 patients) or placement of Hyalograft C for glenoid chondral lesions (11 patients). Significant improvements were seen in 93.6% of all patients at 24 months postoperatively.

Biologic Replacement

Although the use of biologic replacement is more limited in the setting of diffuse osteoarthritic changes, it may be appropriate for young patients with focal, contained chondral lesions in whom the subchondral plate is maintained and the lesion is <2 cm^2 in size. These restorative surgical options—which can be entertained at the time of arthroscopic débridement—include microfracture, osteochondral allografting/autografting, and autologous chondrocyte implantation (ACI) (Figures 3-5). The goal with these procedures is to re-establish hyaline or hyaline-like cartilage at the articular surface. However, these options are less successful in the shoulder than in the knee, in which the articular cartilage is substantially thicker.[1]

Autologous osteochondral transfer may have a role in full-thickness defects because it allows transfer of both cartilage and bone, although it risks donor site morbidity at the knee, where the graft is commonly harvested. By comparison, osteochondral allograft allows the

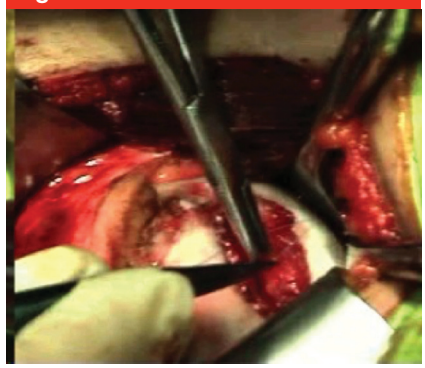
Figure 4



A B

Intraoperative images demonstrating osteochondral allograft preparation (**A**) and placement into the osteochondral defect in the humeral head (**B**).

Figure 5



Intraoperative photograph demonstrating a chondral defect of the humeral head after autologous chondrocyte implantation.

surgeon to address larger lesions without the risk of donor site morbidity.[1] In ACI, autologous cartilage is harvested through in vitro cell growth to apply to a chondral defect; thus, although it avoids donor site morbidity (and may preserve more native subchondral bone, making revision procedures easier), it requires two surgical procedures in a staged approach to harvest and subsequently implant the cultured cells.[1,25]

Relatively little has been published on the use of microfracture or osteochondral grafting for symptomatic glenoid or humeral head articular cartilage lesions, and the use of ACI in the shoulder is investigational and limited to case reports.[33-35] Millett et al[33] reported on 31 shoulders in 30 patients aged <60 years (mean age, 43 years; range, 19 to 59 years) who underwent microfracture for full-thickness glenohumeral joint chondral lesions. At a mean follow-up of 47 months, six shoulders (19%) had progressed to another surgery. Of the remaining 25 shoulders, mean pain scores had decreased from 3.8 to 1.6, and significant improvements were reported in patients' activities of daily living, sports activity, and ability to work ($P < 0.05$). Mean American Shoulder and Elbow Surgeons scores improved significantly from preoperative to postoperative time points, by 20 points. Neither age nor sex was associated with surgical outcomes, but microfracture of isolated humeral lesions was associated with the greatest improvements.

Humeral Arthroplasty Without Glenoid Treatment

HA avoids complications related to prosthetic loosening of the TSA glenoid component.[2,26] The optimal candidate is a young patient with unipolar involvement of the humeral head and a relatively preserved glenoid articular surface[2] or one that is not unevenly worn.[36] Treatment options include full resurfacing, partial resurfacing, and stemmed or stemless humeral head replacement.[2] Stemmed components are more commonly used in the traditional setting.[2] However, stemless HA leads to relatively little bone loss because there is no need to ream the humeral canal in preparation for arthroplasty acceptance.[1]

Soft-tissue balancing is important when performing HA to re-create more anatomic glenohumeral motion, and contracted structures about the shoulder may require aggressive intraoperative surgical release to avoid uneven glenoid wear from its articulation with the metallic humeral head replacement.[2,36] However, persistent pain resulting from bony erosion and arthrosis at the native glenoid are complications following HA, and patients in whom these conditions develop may require conversion to TSA if glenoid bone stock is adequate.[2,20,37] It is important to note that results after conversion from HA to TSA are inferior to results after primary TSA.

Levine et al[36] reviewed 31 young patients (mean age, 56 years) with primary (10 patients) or secondary (21 patients) glenohumeral osteoarthritis who underwent HA. They reported that 74% of shoulders achieved satisfactory results, with outcomes correlated most significantly with the status of posterior glenoid wear (63% satisfactory results in type II glenoids versus 86% satisfactory results in type I glenoids). The authors thus suggested that HA be reserved for patients with a concentric glenoid because this affords a superior fulcrum for glenohumeral motion. These patients were reevaluated at an average follow-up period of 17.2 years.[38] At that time, only 25% of patients were satisfied with their outcome.

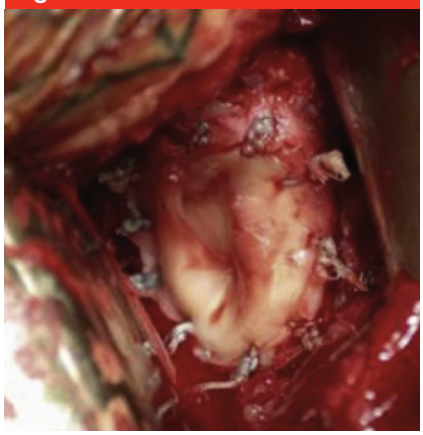
Humeral head resurfacing is similar to traditional HA with identical indications but instead replaces the humerus with a metallic cap. This is a more technically demanding procedure than the placement of a traditional stemmed humeral head because replication of the position and size of the native humeral head can be challenging. Because of its lack of modularity, the resurfacing procedure can make the revision setting more difficult because access to the glenoid is limited without careful explantation.[2] However, the decreased bone stock lost with the resurfacing is beneficial should revision to a TSA be necessary.[2] Iagulii et al[39] reviewed 48 young patients (mean age, 48 years) who underwent humeral head surface replacement arthroplasty to meet continued high-activity demands. Acceptable results were demonstrated at midterm follow-up.

Partial humeral head resurfacing can be used to treat more focal cartilage defects and preserve the remaining intact native cartilage; this technique requires coring a circular trough around a focal chondral defect and replacing it with an implant of equal curvature and core diameter. However, reports suggest an unacceptable failure rate with this option.[40]

Humeral Arthroplasty With Glenoid Treatment

HA with glenoid treatment was developed because of concerns for glenoid component loosening in TSA and for the risk of progressive arthrosis in HA without intervention at the glenoid. Such treatment may be useful for younger patients who are not appropriate candidates for TSA, while avoiding the potential complications from a glenoid prosthesis.[2] High-demand patients (eg, persons who perform manual labor) and those who anticipate a return to overhead activities are candidates for HA with glenoid treatment.[2,41]

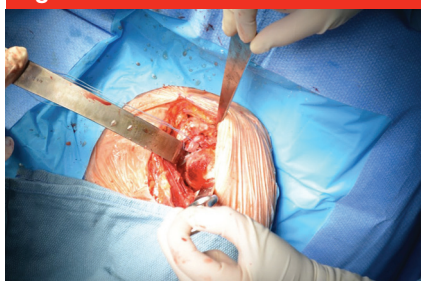
Figure 6



Intraoperative photograph demonstrating biologic interpositional resurfacing of the glenoid using allograft meniscus at the time of humeral head arthroplasty.

One option to treat the glenoid at the time of HA is via biologic interpositional resurfacing (Figure 6). Biologics used include Achilles tendon allograft, meniscus allograft, fascia lata, and acellular, dermal matrix-based scaffold grafts.[2] With this technique, glenoidplasty is performed first to decorticate the articular surface and burr to a bleeding subchondral level of bone.[23] This step may require efforts to increase the anteversion of the native, diseased glenoid when typical posterior glenoid osteoarthritic wear patterns are present, but the technique should be tailored to the patient's anatomy. The allograft is thawed and, using sutures or suture anchors passed circumferentially about the native glenoid rim, it is tacked down into place.[42] In the case of meniscal allograft resurfacing, a lateral meniscus is used so that the anterior and posterior horns can be brought together at the anterior aspect of the native glenoid, with the thick portion of the graft covering the posterior glenoid.[41] Conflicting reports regarding the outcomes after humeral head replacement and glenoid soft-tissue interpositional resurfacing exist in the literature.[41-46]

Figure 7



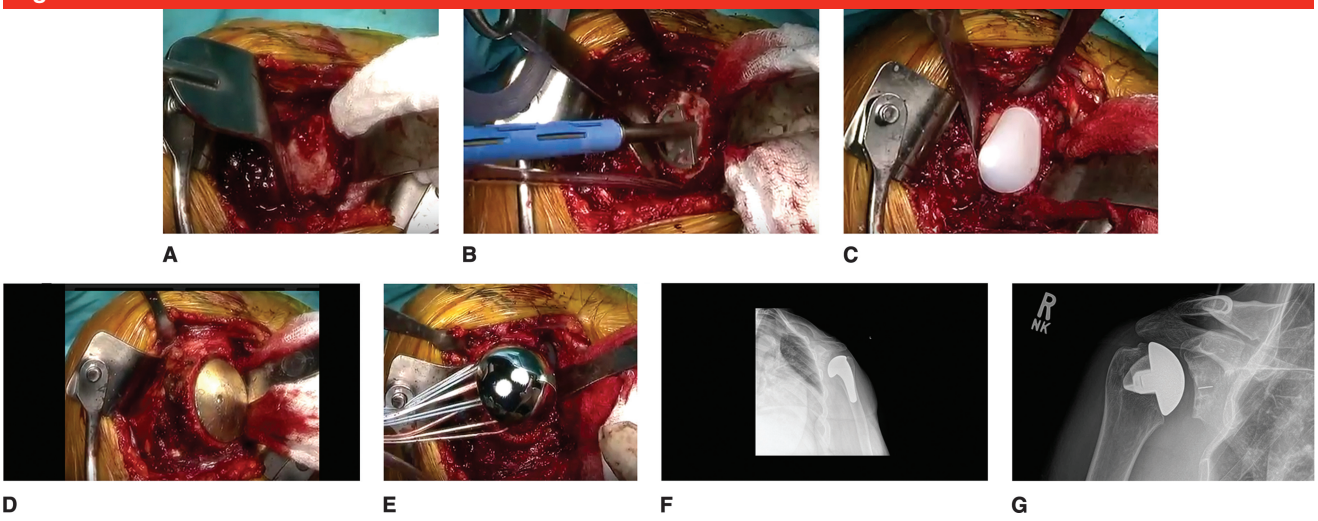
Intraoperative photograph of a left shoulder with the glenoid exposed after reaming for the ream and run technique of humeral head arthroplasty with glenoid treatment.

Another option to address the glenoid is the "ream and run" technique, which is used to restore a concentric glenohumeral articulation while preserving the glenoid bone stock[2] and avoiding risks of polyethylene component wear or the complexities of soft-tissue interposition[19] (Figure 7). In this technique, the glenoid is superficially reamed to a bleeding subchondral bone to recreate an articular surface that is concentric and slightly greater in radius of curvature than the prosthetic humeral head. The reaming process stimulates fibrocartilaginous growth and thus creates a thick fibrous covering at the glenoid at around 24 weeks postoperatively while maintaining its concavity.[1]

Anatomic Total Shoulder Arthroplasty

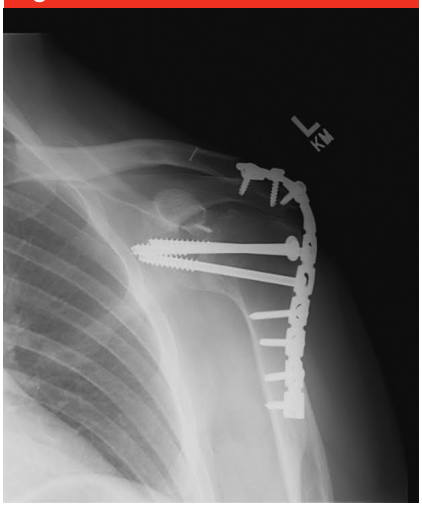
Although originally thought to be more suited for lower-demand patients, TSA remains the standard for treatment of diffuse glenohumeral arthritis (Figure 8). The American Academy of Orthopaedic Surgeons (AAOS) clinical practice guidelines support its use.[47] In comparison with HA, TSA leads to a significantly better pain score and range of motion improvements but with similar satisfaction and revision needs.[26,37] However, despite the good initial pain

Figure 8



Intraoperative photographs of total shoulder arthroplasty (TSA) demonstrating an osteoarthritic glenoid (**A**), reaming of the glenoid (**B**), and in situ fixation of the glenoid (**C**) and humeral head components (**D** and **E**). Postoperative AP radiographs demonstrating placement of stemmed (**F**) and stemless (**G**) TSA humeral components.

Figure 9



Postoperative plain radiographs (AP view, left shoulder) demonstrating a left glenohumeral arthrodesis.

relief,[1] concerns for glenoid component loosening, polyethylene wear, and cement fragmentation temper excitement with its use in younger patients[2] because of the potential need for revision surgery.[20,48]

Although some authors have noted unsatisfactory results despite improvements in pain and motion for this patient demographic, others have considered it a viable treatment option with low complication rates and excellent intermediate- to long-term results.[20,21,26,37] Bartelt et al[37] evaluated 46 TSA and 20 HA in 63 patients (aged ≤55 years) with primary glenohumeral osteoarthritis and reported an implant survival rate of 92% at 10 years for TSA and significantly less pain ($P = 0.01$), greater active elevation ($P = 0.05$), and higher satisfaction ($P = 0.05$) than their counterparts who underwent HA. However, in 10 of 34 TSA, more than minor glenoid periprosthetic lucency or a shift in component position was present.

Raiss et al[20] prospectively evaluated 21 patients (21 shoulders) with a mean age of 55 years (range, 37 to 60 years) who underwent TSA for primary glenohumeral osteoarthritis. At a mean follow-up of 7 years, there were no revision requirements, and 95% of patients were either "very satisfied" or "satisfied" with their outcome. The mean constant and Murley scores increased significantly ($P < 0.0001$), and no clinical or radiologic signs of periprosthetic loosening were reported.

Denard et al[21] examined 52 TSAs in a multicenter study of patients aged <55 years with primary glenohumeral osteoarthritis. At a mean follow-up of 115.5 months postoperatively, forward flexion had improved from 97° to 128°, and adjusted Constant scores had improved from 37.0% to 73.4% ($P < 0.001$ for both). Survivorship of the glenoid component (with the end point defined as revision surgery requirement for glenoid loosening) was 98% at 5-year follow-up and 62.5% at 10-year follow-up. Compaction glenoid preparation technique and anatomic humeral component positioning were factors correlated with glenoid implant survival.

Other

In addition to the aforementioned procedures, several other methods are practiced with less frequency in specific patient populations. Corrective osteotomies, such as open-wedge osteotomies of the glenoid, can be used to treat symptomatic fixed posterior subluxation and posterior glenoid wear.[3] Although use of glenohumeral arthrodesis has declined, it can be a viable option for young patients with

end-stage disease and strenuous physical demands (ie, heavy manual labor), chronic infection, severe neurologic injury, massive rotator cuff tears with concurrent deltoid deficiency, voluntary dislocators, and multiple failed surgeries or failed arthroplasty[3,32] (Figure 9). Low-demand patients with poor preoperative function, who have failed rotator cuff repair or other arthroplasty means, or those who present with rotator cuff arthropathy, may be candidates for reverse total shoulder arthroplasty.[2,49,50]

AAOS Clinical Practice Guideline

In 2009, the AAOS provided a summary of recommendations and evidence-based guidelines regarding the treatment of glenohumeral joint osteoarthritis.[47] They ultimately were unable to recommend for or against the use of pharmacotherapy or injectable corticosteroids for the initial treatment of glenohumeral osteoarthritis. They provided a "limited" recommendation for the use of injectable viscosupplementation for these patients, citing unconvincing supporting evidence. The guidelines found a lack of compelling evidence for or against the use of arthroscopic treatments or nonprosthetic/biologic interposition arthroplasty for patients with glenohumeral osteoarthritis. They provided a "limited" recommendation for the use of TSA or HA in treating such patients, with a moderate recommendation favoring TSA over HA.

These guidelines, despite being from the year 2009, are still representative of the prevailing literature findings of the past decade. Because their intentions are to more broadly group all patients with this pathology, their recommendations are not specific to the younger patient as we highlight in this review. Despite this, the data do not suggest that substantial changes should be made to the original guidelines when considering the young patient demographic with this pathology.

Summary

Glenohumeral osteoarthritis in the young patient is complex. This demographic often has high functional demands, and management must be tailored accordingly. A thorough history and physical examination should be obtained for any patient presenting with shoulder pain and consideration of osteoarthritis. Nonsurgical modalities are the mainstay of management; however, in young patients who have exhausted nonsurgical management options, surgical options may be pursued with varying levels of efficacy. Future research efforts into the current interventions—as well as further investigative work on cartilage reparative, restorative, or replacement options—may help improve outcomes in this challenging demographic.

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