The Role of the Anterior Shoulder Joint Capsule in Primary Glenohumeral Osteoarthritis

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Abstract

The pathogenesis of primary glenohumeral arthritis (GHOA) is mediated by a complex interaction between osseous anatomy and the surrounding soft tissues. Recently, there has been growing interest in characterizing the association between the anterior shoulder joint capsule (ASJC) and primary GHOA because of the potential for targeted treatment interventions. Emerging evidence has shown substantial synovitis, fibrosis, and mixed inflammatory cell infiltrate in the anterior capsule of osteoarthritic shoulders. In addition, increased thickening of the ASJC has been associated with greater posterior glenoid wear and humeral head subluxation. While these findings suggest that anterior capsular disease may play a causative role in the etiology and progression of eccentric GHOA, further studies are needed to support this association.

The purpose of this article is to review the pathogenesis of primary GHOA, contextualize current hypotheses regarding the role of the anterior capsule in the disease process, and provide directions for future research.

Level of Evidence: Review Article

Keywords: anterior shoulder joint capsule, glenohumeral osteoarthritis, glenoid erosion, fibrosis, thickening, pathogenesis

Primary glenohumeral osteoarthritis (GHOA) is one of the most common causes of shoulder pain [3, 18, 26] and is the most common indication for anatomic total shoulder arthroplasty [56]. While the true prevalence of GHOA is difficult to determine, a recent review estimates that 16-20% of the middle-aged and elderly population have radiographic evidence of shoulder
osteoarthritis [25]. After the hip and knee, the shoulder is the third most commonly affected large joint [26].

Shoulder arthroplasty is a highly successful procedure for symptomatic GHOA [27, 55, 59], although the generally excellent outcomes may be less consistent in cases with substantial posterior glenoid deformity and posterior humeral head subluxation (PHHS) [23, 24, 54]. While a variety of surgical techniques have been described to address these challenges, there are a paucity of studies that explore the etiology and progression of arthritic changes in the glenohumeral joint [11, 53, 56]. Further understanding of this process may aid in the development of disease-modifying strategies to delay or reduce the need for shoulder arthroplasty, which is of particular importance in young and active patients [6, 11, 53].

The purpose of this article is to review the pathogenesis of primary GHOA, contextualize current hypotheses regarding the role of the anterior capsule in the disease process, and provide directions for future research.

**Primary GHOA: Pathogenesis**

A landmark study by Harryman et al [19] in 1990 described the effects of anterior capsular tightness on translation of the humeral head with passive motion of the osteoarthritic shoulder. When the anterior capsule is tight and contracted, external rotation of glenohumeral joint produces a posteriorly directed force that causes posterior decentering of the humeral head on the glenoid [19, 34] (Figure 1). Over time, posterior subluxation of the humeral head causes erosion of the posterior glenoid, which in turn limits external rotation of the glenohumeral joint and
subsequently leads to more stiffness of the anterior capsule – thus continuing this “vicious cycle” [33] (Figure 2).

Walch et al [53] was among the first to define, quantify, and categorize the concept of static posterior subluxation of the humeral head. In their study, 13 males (mean age, 40 years) with symptomatic GHOA were evaluated by computed tomography (CT) imaging and found to have PHHS without evidence of posterior glenoid bone erosion. Each patient underwent various surgical procedures in attempt to correct the subluxation, including arthroscopic Bankart repair, the Bristow-Latarjet procedure, arthroscopic synovectomy, a posterior bone-block procedure, posterior opening wedge osteotomies of the scapula neck, arthrolysis of the anterior compartment with lengthening of the subscapularis tendon and posterior capsulorrhaphy, or removal of hardware used in previous stabilization surgery. At 46 months postoperatively, all patients had progression of arthritis with subsequent development of posterior glenoid wear and recurrent posterior humeral subluxation. It was suggested that static PHHS in the young adult could be the first step in the development of primary GHOA. This theory was corroborated nearly two decades later. In 2018, Walker et al [56] evaluated the longitudinal history of primary GHOA in 65 shoulders by comparing CT findings at an average time interval of 74 months. It was found that the humeral head remained centered in most Walch A-type glenoids, with rare progression to PHHS or asymmetric posterior glenoid bone loss. In contrast, B1 glenoids were associated with progression to B2 and B3-type glenoids, indicating that the presence of posterior humeral head translation may lead to the development of posterior glenoid bone loss and its progression over time.
While the etiology and progression of GHOA is not fully understood [11, 21], it appears to be mediated by a complex interaction between osseous anatomy and the surrounding soft tissues. While most of the initial research efforts focused on the influence of the rotator cuff musculature on glenoid deformity [1, 12, 39, 56], there has been increased interest in characterizing the role of the anterior capsule in primary GHOA because of the potential for early targeted treatment interventions to prevent and manage arthritis.

**Parallel with Capsulorrhaphy Arthropathy**

The role of the ASJC in the development of primary GHOA may be closely correlated with the concept of capsulorrhaphy arthropathy, which describes the onset of glenohumeral degeneration following the surgical treatment of recurrent anterior shoulder instability [41]. Long term follow-up studies have shown high rates of GHOA of up to 62% after a Putti-Platt repair, Bankart repair, and Bristow procedure [22, 29, 51, 63]. With many of these procedures, the anterior capsule is tightened with or without shortening or transfer of the subscapularis tendon. This tightening is performed to limit external rotation and prevent the shoulder from reaching positions in which it is prone to anterior instability [41]. However, tightening of the anterior soft tissue structures may result in obligate posterior translation of the humeral head and rapid posterior chondral wear as a result of compressive joint forces [19]. These points were further corroborated in a cadaveric study by Werner et al [57], in which the effects of selective capsular plication on humeral head translation were studied. That study found that selective anterosuperior plication, anteroinferior plication, or total anterior plication significantly increased posterior translation of the humeral head in both neutral and 90 degrees of internal rotation, inferring that anterior capsular tightening may influence the development of static
subluxation and osteoarthritis. The posterior humeral head subluxation and posterior erosion in capsulorrhaphy arthropathy is indistinguishable to that in primary GHOA with posterior glenoid erosion, making anterior capsule plication essentially a “human animal model” for the development of GHOA.

**Histopathology of the Anterior Capsule in the Osteoarthritic Shoulder**

There is evidence to suggest that the pathogenesis of primary GHOA involves thickening and asymmetric contracture of the ASJC [6]. Similar capsular abnormalities have been shown to occur in adhesive capsulitis, where contracture and hypertrophy are likely mediated by synovial hyperplasia and hypervascularity with perivascular and capsular fibrosis [20, 43]. Chainani et al [6] performed histopathologic comparison of the ASJC harvested from shoulders with primary GHOA at the time of total shoulder arthroplasty (TSA) and compared them to that of cadaveric donors with no history of shoulder pathology. Compared with cadaveric controls, the synovial lining and ASJC in those with primary GHOA had significant synovitis and fibrosis--confirmed by increased expression of alpha smooth muscle actin (αSMA; a marker of myofibroblast activation) and transforming growth factor β1 (TGFβ1; a fibrosis-related growth factor). TGFβ1 has been shown to stimulate αSMA production and is involved in the transition of synovial fibroblasts to myofibroblasts [36]. TGFβ signaling pathways have also been implicated in the pathogenesis of other joint capsule pathologies, including carpal tunnel syndrome, adhesive capsulitis, and post-traumatic joint contractures of the hip and shoulder [38, 61, 62].

Furthermore, the synovial lining and ASJC in those with primary GHOA had a significant increase in the expression of M1 and M2 macrophage lineage markers. While macrophages are
normally present within healthy joint capsule and serve an important role in the attenuation of osteoarthritis [60], their function is dysregulated in osteoarthritis [30]. For example, the M2 macrophage subset expression data led Chainani et al [6] to believe that both the synovial lining and ASJC of primary GHOA patients may be in a dynamic state of inflammation, proliferation, tissue stabilization, and remodeling without resolution of the inflammatory state. In addition, it was found that the immunoreactivity of several transient receptor potential (TRP) channels (TRPA1, TRPV1, and TRPV4) was greater in the ASJC of arthritic shoulders. As mechanosensitive transcriptional upregulators of TGFβ and myofibroblast differentiation [9], these channels serve an important function in the development of fibrotic tissues [58], and could provide a mechanism for initiation of fibrosis and thickening of the ASJC.

**Correlation of Pathologic ASJC with Posterior Glenoid Erosion and Humeral Head Subluxation**

**Subluxation**

In 2022, Menendez et al [37] utilized magnetic resonance imaging (MRI) and CT to study the relationship between anterior capsular thickness and glenoid deformity in 134 osteoarthritic shoulders. Within this study, the anterior capsule was thickest in B2 (5.5 mm, 95% confidence interval (CI) 5.0-6.0) and B3 glenoids (6.1 mm, 95% CI 5.6-6.6), and thinnest in A1 (3.7 mm, 95% CI 3.3-4.2; P<0.001). Furthermore, increased capsular thickness correlated with greater glenoid retroversion (r=0.57; p<0.001) and posterior humeral head subluxation (r=0.50; p<0.001) providing imaging evidence of previous clinical and cadaveric observations described above. For every 1-mm increase in anterior capsular thickening, there was an adjusted mean increase of 3.2 degrees (95% CI 2.4-4.1) in glenoid retroversion and a 3.8% (95% CI 2.7-5.0) increase in posterior humeral head subluxation. However, given the single-point-in-time analysis, despite a
very strong correlation, the authors note that a causal relationship amongst these variables could not be established.

Correlation of Pathologic ASJC with Humeral Head Pattern of Osteoarthritis

The macroscopic appearance of humeral head erosion in glenohumeral osteoarthritis has been colorfully described as the “Friar Tuck” pattern, with central eburnation and a surrounding ring of cartilage and osteophytes [47]. This pattern was recently confirmed at the histological level, as Matson et al [35] showed that chondral wear of the humeral head resected from patients undergoing anatomic TSA for end-stage GHOA is present centrally and centro-inferiorly. Interestingly, many patients with end-stage GHOA had preserved peripheral cartilage but demonstrated central cartilage wear and bony sclerosis (Figure 3). It was also found that higher histopathological scores (indicating worse damage) were associated with decreased preoperative range of motion. This may suggest that a feedback loop of capsular pathologic stretch-fibrosis-contracture-inflammation leading to decreased range of motion may cause abnormal central and inferior humeral head loading leading to osteoarthritis, which in turn leads back to capsular contracture and re-initiation of this process (Figure 4). These findings lead one to consider the potential link between maintaining range of motion and the preservation of humeral head cartilage, especially considering the early success of capsular release in conjunction with partial biologic or prosthetic resurfacing techniques for the treatment of early GHOA [2, 10, 46, 48, 52].

Treatment Options for Anterior Capsular Disease

Arthroscopic joint débridement with capsular release may provide a temporizing treatment option to avoid prosthetic replacement in young patients with mild GHOA [50]. We prefer the
Arthroscopic Circumferential Capsulotomy (ArCC) procedure that involves a complete release of the capsule as well as axillary nerve identification to facilitate a thorough capsular release [28, 49]. This is particularly useful in those with a substantial side-to-side difference in external or internal rotation [4, 45], as a contracted ASJC has been shown to reduce rotation about the glenohumeral joint and has been implicated in the progression of osteoarthritis [7]. Porcellini et al [42] reported on 47 patients aged 30 to 55 years after arthroscopic circumferential capsulotomy with microfracture (n=36) or placement of Hyalograft C (n=11) for glenoid chondral lesions in patients with painful early arthritic shoulders. At 2-year follow-up, significant improvements in the mean Constant score (43.8 to 79.1) and Simple Shoulder Test (SST) score (4.9 to 9.4) were found, and 93.6% of patients achieved clinical improvement. Interestingly, however, it was noted that improved outcomes were only associated with small and centered cartilage lesions, leading the authors to conclude that soft tissue procedures are best suited for those with early arthritis and central wear. This leads one to consider the utility of early joint-preserving interventions in preventing or slowing the progression of GHOA. This is especially plausible in light of the humeral head histopathologic findings described in the previous section, where preserved peripheral cartilage is frequently found in cases of severe stiffness, and the loss of joint motion could have essentially “protected” these more peripheral regions from abnormal load and chondral damage [35].

In more severe cases, during shoulder arthroplasty anterior capsular release in conjunction with version correction has been shown to re-center posteriorly subluxated humeral heads [15, 17]. Gerber et al [15] studied 33 shoulders with PHHS (average subluxation index, 71%) treated with primary TSA using peri-glenoid capsular release and corrective glenoid reaming. At a mean
follow-up of 42 months, PHHS was reversed in 50% of patients. The authors suggested that anterior capsule release may have contributed to re-centering of the glenohumeral joint and that persistent PHHS postoperatively may be due to over tensioning of the subscapularis tendon or excessive rotator interval closure. In a separate study [17] of 77 arthritic shoulders with PHHS and posterior glenoid wear, recentering of the humeral head was achieved in all patients at mid-term follow-up (average, 24 months) by surgical correction of glenoid alignment (eccentric reaming or glenoid bone grafting) with soft tissue balancing (bifocal capsular release, posterosuperior rotator cuff mobilization, coracohumeral ligament release, and pectoralis major tendon release in those with massive contraction). In the most recent study of patients with GHOA with posterior glenoid erosion and posterior humeral head subluxation, Garrigues et al [14] used an augmented anatomic glenoid with either complete antero-inferior capsulectomy or complete antero-inferior capsular releases and showed posterior humeral head subluxation not only corrected, but that the correction was maintained with minimum 2-year follow-up.

Authors’ Preferred Technique of Anteroinferior Capsulectomy for Anatomic Shoulder Arthroplasty

A key component of anatomic TSA is thorough release of the typically thickened and contracted anterior and inferior capsular tissues [32, 40]. This allows for adequate subscapularis mobilization during glenoid exposure, increased external rotation and abduction of the glenohumeral joint postoperatively, and correction of PHHS when performed in conjunction with correction of bony glenoid erosion [14, 16]. While most surgeons routinely perform a longitudinal capsulotomy, some, including our senior author (**blinded for review**), resect the entire anteroinferior capsule when performing a primary anatomic TSA for glenohumeral
arthritis (Figures 5, 6, 7). We have found that use of a lesser tuberosity osteotomy combined with anteroinferior capsulectomy results in optimal soft tissue balancing and postoperative range of motion. However, it is important to note that these results are anecdotal through experience of over 3,200 cases but not tested in a head to head randomized fashion.

In brief, a standard deltopectoral approach is utilized with an incision placed over the coracoid tip extending towards the anterior deltoid insertion. After standard subcutaneous dissection and development of the deltopectoral interval, the axillary nerve is palpated where it passes between the subscapularis and the anterior humeral circumflex artery. This is confirmed by the tug test, which involves a gentle pull on the axillary nerve with one index finger in the subdeltoid space and feeling of the axillary nerve move with the other index finger placed within the subcoracoid space. Next, the long head of the biceps tendon is tenodesed. At this point, the bicipital groove and subscapularis tendon are exposed. With the arm in external rotation, osteotomy of the lesser tuberosity is performed and should resemble an oval of approximately 2 cm in diameter and 1 cm in thickness. Upon release, the subscapularis is tagged at the musculotendinous junction. A plane is then developed between the subscapularis and the anteroinferior capsule, which should remain attached to both the humerus and glenoid. The capsule is then released from the humerus as the arm is adducted and externally rotated to allow for complete circumferential capsular release along the humeral neck. After osteophytes are removed and the humeral head cut is made, anteroinferior capsulectomy is the first step in glenoid exposure. A Fukuda retractor is placed posterior to the glenoid to retract the humeral head posterolaterally, and two blunt Hohman retractors are placed in the plane previously developed between the capsule and the subscapularis, deep to the axillary nerve, while a Kocher clamp is used to securely grasp the
capsule. With the capsule clearly visualized, to ensure that the axillary nerve is not in danger, the capsule is divided from lateral to medial at the 6 o’clock position on the glenoid. This anteroinferior capsule is then removed from inferior extending superiorly to the rotator interval. The resected capsule and synovial lining are universally thickened and contracted, with varying levels of severity. After the capsulectomy is performed, the labrum is circumferentially excised, and the posterior and remaining inferior capsule is released to complete the glenoid exposure. After the glenoid and humeral prosthesis are implanted, the lesser tuberosity osteotomy is repaired using a double row construct.

Future Directions
As the role of anterior capsular disease in osteoarthritic shoulders becomes more evident, there is growing research interest on the influence of targeted management strategies to potentially delay and better manage arthritis. Given the recent immunohistologic data showing synovial fibrosis and inflammation in the ASJC of arthritic shoulders [6], there is a great opportunity to investigate the role of cellular therapies in the prevention and management of primary GHOA [44]. While studies are limited in number and sample size, promising reductions in pain and improvements in shoulder function have been reported with use of platelet rich plasma (PRP)[13, 31] and autologous bone marrow aspirate concentrate (BMAC) [5], which both may serve as regulators of the inflammatory cascade [8]. Perhaps targeted pharmacologic therapy of anterior capsular disease could be an important next step in the modulation of the arthritic shoulder pathway. Future studies should also assess the effects of interventions such as capsular stretching/mobilization exercises or focal anterior capsular releases on the progression of primary GHOA. Lastly, it would be beneficial to determine whether there is a difference in
postoperative range of motion and patient reported outcomes after shoulder arthroplasty with routine capsulotomy versus capsulectomy in patients with primary GHOA.

Conclusion

Thickening and fibrosis of the anterior shoulder joint capsule has been implicated in the pathogenesis of glenohumeral osteoarthritis, particularly in cases with posterior glenoid erosion. While it remains unclear whether anterior capsular disease plays a causative role in the etiology and progression of eccentric GHOA, further understanding of this relationship may allow for the study of targeted management strategies or refinement of surgical techniques (e.g., anteroinferior capsulectomy during total shoulder arthroplasty) to improve functional outcomes.

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**Figure Legends**

Figure 1: Obligate posterior translation of the humeral upon external rotation of the glenohumeral joint as a consequence of anterior capsular tightness. (Reproduced with permission from Elsevier)

Figure 2: The “vicious cycle” of glenohumeral arthritis.

Figure 3: Central (A) and peripheral region (B) cartilage thickness and heat map (C). Central (D) and peripheral region (E) subchondral bone area and heat map. Central (G) and peripheral region (H) subchondral bone plate thickness and heat map (I) for zones 1–8 circumferentially around resected humeral heads. Black line p < 0.05 between zones. * Central region significantly different from periphery, p < 0.0001. S = Superior, A = Anterior, I = Inferior, P = Posterior. (Reproduced with permission from Osteoarthritis Research Society International)
Figure 4: The influence of anterior capsule disease on central chondral wear of the humerus and end-stage glenohumeral osteoarthritis.

Figure 5: The anterior capsule (grasped with forceps) remains attached to the humerus, while the subscapularis has been completely dissected off.

Figure 6: The anterior capsule (grasped with a Kocher clamp) is fully separated from the subscapularis muscle, which is reflected medially with a Cobb elevator.

Figure 7: The resected capsule is held so the cross section can be seen. Note it is nearly 1 cm thick.
Obligate posterior translation. When the anterior capsule is tight, external rotation against the tight capsule produces a posteriorly directed force that can push the humeral head in a posterior direction.
Declaration of interests

☐ The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

☒ The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Suleiman Y. Sudah, MD certifies that he had nothing of value related to this study.
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Richard N. Puzzitiello, MD certifies that he had nothing of value related to this study.
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