JSES International 5 (2021) 282–287

Contents lists available at ScienceDirect

JSES International

journal homepage: www.jsesinternational.org

Glenoid retroversion associates with deltoid muscle asymmetry in Walch B-type glenohumeral osteoarthritis

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ARTICLE INFO

Keywords:
shoulder replacement
shoulder arthroplasty
glenohumeral osteoarthritis
glenoid retroversion
etiology
deltoid

Level of evidence: Anatomy Study; Imaging

Background: The etiologies of glenohumeral osteoarthritis (GHOA) and eccentric glenoid wear within GHOA are unknown, but muscular imbalance may play a role. The purpose of the present study was to determine the relationship between deltoid muscle area, GHOA, and eccentric glenoid wear. We hypothesized that patients with GHOA would have overall deltoid atrophy as compared with controls and that increasing posterior deltoid areas would associate with glenoid retroversion in the Walch B-type (eccentric) GHOA group.

Methods: The study was a retrospective review of computed tomography imaging studies. We included a control group of subjects without GHOA and a group of individuals with GHOA before undergoing total shoulder arthroplasty. We assigned Walch types via consensus. Cross-sectional area was measured for the anterior and posterior deltoid musculature demarcated via the scapular line, normalized to the total deltoid area. Absolute and normalized total, anterior, and posterior deltoid areas were compared between controls and the entire GHOA group. Normalized anterior and posterior deltoid areas were compared between Walch A-type and B-type GHOA patients within the GHOA group. Univariate linear regression was used to evaluate for an association between glenoid retroversion and normalized posterior deltoid areas in controls, Walch A-type, and Walch B-type patients. Multivariate linear regression analysis was used to evaluate the effects of normalized posterior deltoid area, age, sex, and height on glenoid retroversion within the Walch B-type subgroup.

Results: We included 99 patients with GHOA and 47 controls. The control and GHOA patients did not differ in absolute deltoid areas ($21.8 \pm 8.8 cm^2$ vs. $20.6 \pm 7.9 cm^2$; $P = .488$). Patients with GHOA had a statistically significant increase in normalized posterior deltoid area (0.50 ± 0.10 vs. 0.46 ± 0.10; $P = .032$) and a reciprocal decrease in normalized anterior deltoid area (0.50 ± 0.10 vs. 0.54 ± 0.10; $P = .040$) compared with controls. Walch A-type and B-type patients did not differ in normalized posterior deltoid areas (0.50 ± 0.11 vs. 0.50 ± 0.10; $P = .780$). Normalized posterior deltoid area positively associated with glenohumeral retroversion in Walch B-type GHOA ($R^2 = 0.102$; $P = .020$), a relationship maintained in multivariate linear regression, using gender, age, and height as covariates (standardized beta = 0.309, $P = .027$).

Conclusion: GHOA is not associated with deltoid atrophy, calling into question the suggestion that periarticular muscular atrophy in GHOA is secondary to disuse. Increasing normalized posterior deltoid area associates with increased glenoid retroversion in patients with Walch B-type glenoid morphology. Muscular imbalance may play a role in the etiology or progression of the glenoid deformity observed in eccentric GHOA.

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Each author certifies that his or her institution approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

This study was performed under the University of Utah Institutional Review Board approved protocol 71740.

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https://doi.org/10.1016/j.jseint.2020.10.012
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Walch B-type glenohumeral osteoarthritis (GHOA), which describes posterior subluxation of the humeral head and associated glenoid retroversion and/or biconcavity, has been suggested to influence total shoulder arthroplasty (TSA) survival.[4,6,13,14] The etiology of eccentric GHOA is not completely understood and is likely multifactorial.[8] However, multiple authors have suggested that posterior subluxation of the humeral head precedes eccentric glenoid wear and may drive the development of glenoid retroversion and biconcavity.[8,12] The underlying cause of this posterior subluxation remains unknown.

Although the glenohumeral joint is known to be dependent on the periarticular musculature for stability, the contributions of these structures to the development of posterior subluxation of the humeral head and eccentric GHOA is unknown.[9] Pediatric brachial plexus injuries resulting in persistent muscular imbalance cause glenoid retroversion and posterior subluxation of the humeral head to develop over time, suggesting that soft tissue imbalance can precede bony deformity.[16] Rotator cuff atrophy and fatty infiltration are common findings in patients with GHOA and have been shown to affect outcome after TSA.[7,11] Several recent studies have supported the hypothesis that GHOA is associated with a unique pattern of rotator cuff asymmetry in the axial plane.[1,5,9,15]

Along with the rotator cuff, the deltoid is the major contributor to shoulder stability and function.[2] It is unclear whether GHOA is associated with changes in deltoid muscle area and/or architecture, as is seen in the rotator cuff.[7,11] Furthermore, whether or not deltoid dysfunction contributes to the development of GHOA has not been fully established. It is plausible that deltoid muscular imbalance within the axial plane could also associate with eccentric GHOA, similar to the findings in the rotator cuff discussed previously. This could provide an explanation for posterior subluxation, with glenoid deformity developing as a consequence.

Thus, the purpose of the present study was to determine the relationship between deltoid muscle area and symmetry and GHOA. We hypothesized that patients with GHOA would have deltoid atrophy as compared with controls and that increasing normalized posterior deltoid areas would associate with glenoid retroversion in the Walch B-type (eccentric) GHOA group.

Methods

Patient selection and data collection

This was a retrospective cohort study performed at the University of Utah (Salt Lake City, UT, USA). Institutional review board approval was obtained before data collection. We included two patient groups: (1) patients with primary GHOA who underwent primary TSA and had a recent preoperative computed tomography (CT) scan, (2) control patients who had CT scans with no evidence of glenohumeral arthritis.

All patients were identified from a GHOA database maintained at our institution. During database creation, a query was made within the electronic medical record to identify patients who underwent primary shoulder arthroplasty for primary GHOA from 2012 to 2017 and who had a preoperative (CT) scan within three months before surgery. A control cohort of patients with CT imaging of the shoulder and no evidence of glenohumeral arthritis was identified. Control patients were imaged for one of the following reasons: to rule out a fracture, evaluation of nearby structures, evaluation for metastasis/tumor, or research imaging. Exclusion criteria for both groups included nonfunctioning deltoid, known rotator cuff tear, rotator cuff tear arthropathy, previous surgical rotator cuff repair, history of inflammatory arthritis, or prior proximal humerus fracture.

Demographic data were collected from the electronic medical record, including; age, sex, operative side (if applicable), self-reported height and weight, and body mass index. Demographic data for the GHOA group were collected at the time of surgery for TSA. Control cohort demographics were documented at the time of imaging.

Deltoid muscle area measurement

Preoperative CT scans from the osteoarthritis cohort and control CT scans were downloaded into a third-party DICOM viewer (Horos, Purview, Annapolis, MD, USA). Each scan was reoriented into the plane of the scapula using the center of the glenoid face, trigonum spinae of the scapula, and inferior angle of the scapula to form reliable axes. Cross-sectional deltoid muscle area measurements were performed on axial images. The scapular line, defined as the superoinferior plane from the trigonum spinae through the center of the glenoid face, was used to bisect the deltoid muscle into distinct anterior and posterior portions. Next, the axial slice used for area measurements was identified using the superior-most slice of the scan containing the greater tuberosity of the humerus. This methodology was selected as it has been previously demonstrated to best correlate with whole deltoid volume.[10] The anterior and posterior deltoid segments were calculated for area using the closed polygon tool (Figure 1). Total deltoid area was calculated as the sum of anterior and posterior deltoid areas. Each deltoid muscle area measurement was performed by two researchers independently and the average area was used for this study. In an attempt to minimize the effects of patient size on the analysis, anterior and posterior deltoid areas were divided by total deltoid area to create normalized anterior and posterior deltoid areas, which were used in the analysis.

Glenoid version measurement

Glenoid version measurements used in the study were collected from the GHOA research database maintained at our institution, which has previously demonstrated measurement reliability in a prior study.[5]

Walch type determination

Axial images were used. Two surgeons who have completed fellowship training in shoulder and elbow surgery (R.Z.T. and P.N.C.) independently evaluated each glenoid. Each surgeon assigned a Walch type to each glenoid using standard Walch type glenoid classification.[3] Owing to the documented reliability concerns of this method,[3] each patient for whom there was disagreement (41 of 146 cases) of Walch type were evaluated by both surgeons together to assign a final consensus Walch glenoid type, which was used in the final analysis.

Statistical analysis

Statistical analyses were performed in Excel X (Microsoft, Redmond, WA, USA) and SPSS 25 (IBM, Armonk, NY, USA). Descriptive statistics were calculated for all variables and normality was analyzed using the Kolmogorov-Smirnov test. Continuous variables were compared between groups using an independent samples t-test or a Mann-Whitney u-test depending on the normality of the sample. Categorical variables were analyzed using Pearson's chi-squared test if all cells had an expected value of greater than or equal to five. Fisher's exact test was used for categorical variables if the expected value of any cell was less than 5. Univariate linear regression analysis was used to evaluate the association between glenoid retroversion and normalized posterior deltoid area in

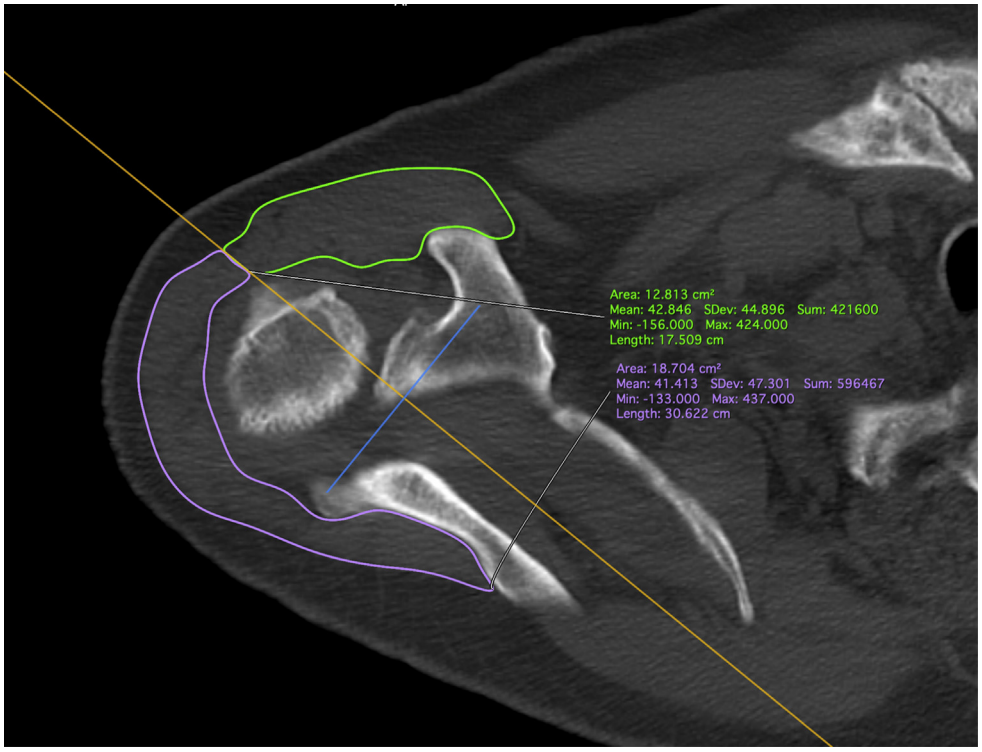

Figure 1 Depiction of deltoid muscle area measurement. Shown is a right shoulder computed tomography (CT) scan with outlined anterior and posterior deltoid area measurements (Horos, Purview, Annapolis, MD, USA).

control shoulders, Walch A-type shoulders, and Walch B-type shoulders. Multivariate linear regression was used to repeat this analysis for Walch B-type shoulders while controlling for age, height, and gender.

Two readers made all deltoid area measurements. To assess reliability between observers, intraclass correlation coefficients (ICCs) were calculated using a two-way mixed effects model with absolute agreement. ICC values of greater than 0.8 were considered strong agreement and ICC values between 0.6 and 0.8 were considered good agreement

Results

Comparison of control patients to patients with primary glenohumeral osteoarthritis

Ninety-nine patients with primary GHOA and appropriate CT imaging were identified. Forty-seven control patients with CT imaging of the shoulder were identified (Table I). Compared with controls, patients with GHOA were older and had a higher body mass index. Morphologically, patients with GHOA had significantly higher mean glenoid retroversion. There were no differences in absolute total deltoid area between controls and patients with GHOA ($21.8 \pm 8.8 cm^2$ vs. $20.6 \pm 7.9 cm^2$; $P = .488$). However, patients with GHOA had a statistically significant increase in normalized posterior deltoid area (0.50 ± 0.10 vs. 0.46 ± 0.10; $P = .032$) and a reciprocal decrease in normalized anterior deltoid area (0.50 ± 0.10 vs. 0.54 ± 0.10; $P = .040$) compared with controls.

Comparison of patients with Walch A-type GHOA with patients with Walch B-type GHOA

Within the GHOA cohort, 40 patients with Walch A-type GHOA and 53 patients with Walch B-type GHOA were identified (Table II). Six patients from the GHOA cohort were excluded from the concentric versus eccentric GHOA subanalysis because they had Walch C- or D-type glenoid morphology. The two groups were similar demographically with the exception that a higher percentage of patients in the Walch B-type group were male compared with the Walch A-type group (62% vs. 40%; $P = .033$). As expected, the Walch B-type group demonstrated significantly higher glenoid retroversion than the A-type group ($19.6 \pm 10.2°$ vs. $8.0 \pm 8.4°$; $P < .001$). There were no differences in normalized anterior or posterior deltoid area between groups.

Table I
Comparison of control patients with patients with primary glenohumeral osteoarthritis (GHOA)

Variable	Control (N = 47)	GHOA (N = 99)	*P* value
Side (% left)	41.0%	56.0%	.110
Gender (% male)	55.0%	54.0%	.840
Age (yr)	39.3 ± 17.8	66.6 ± 9.7	**<.001**
Height (cm)	175.0 ± 10.5	172.6 ± 10.6	.228
Weight (kg)	73.5 ± 17.2	92.3 ± 10.6	**<.001**
BMI (kg/m^2)	24.1 ± 5.4	39.4 ± 5.9	**<.001**
Anterior deltoid area (cm^2)	11.4 ± 4.5	10.0 ± 3.9	.620
Posterior deltoid area (cm^2)	10.4 ± 5.4	10.6 ± 5.1	.725
Total deltoid area (cm^2)	21.8 ± 8.8	20.6 ± 7.9	.488
Normalized anterior deltoid area	0.54 ± 0.10	0.50 ± 0.10	**.040**
Normalized posterior deltoid area	0.46 ± 0.10	0.50 ± 0.10	**.032**
Glenoid retroversion (°)	4.2 ± 4.4	14.6 ± 11.5	**<.001**

Patients with glenohumeral osteoarthritis were older and had a high BMI than controls. No differences in absolute area were identified between groups. Normalized posterior deltoid area was larger in the glenohumeral osteoarthritis group. Normalized anterior deltoid area was smaller in the glenohumeral osteoarthritis group.
Bolded values indicate statistical significance for a *P* value cut-off ≤.05.

Table II
Comparison of Walch A-type GHOA patients to Walch B-type GHOA patients.

Variable	Walch A-type (N = 40)	Walch B-type (N = 53)	*P* value
Side (% left)	55.0%	58.0%	.736
Gender (% male)	40.0%	62.0%	**.033**
Age (yr)	65.3 ± 9.5	68.5 ± 9.1	.167
Height (cm)	171.2 ± 10.0	173.1 ± 10.2	.231
Weight (kg)	90.5 ± 23.5	89.1 ± 19.8	.748
BMI (kg/m^2)	30.5 ± 5.9	29.6 ± 5.5	.409
Anterior deltoid area (cm^2)	10.0 ± 4.3	9.5 ± 3.7	.981
Posterior deltoid area (cm^2)	10.8 ± 5.4	10.2 ± 5.1	.991
Total deltoid area (cm^2)	20.8 ± 9.4	20.0 ± 7.0	1.000
Normalized anterior deltoid area	0.50 ± 0.11	0.50 ± 0.10	.664
Normalized posterior deltoid area	0.50 ± 0.11	0.50 ± 0.10	.780
Glenoid retroversion (°)	8.0 ± 8.4	19.6 ± 10.2	**<.001**

GHOA, glenohumeral osteoarthritis.
Walch A-type and Walch B-type patients were similar across the majority of study variables. Walch B-type patients were more likely to be male. As expected, Walch B-type shoulders had a significantly larger amount of retroversion than Walch-A type shoulders.
Bolded values indicate statistical significance for a *P* value cut-off ≤.05.

Regression analysis

Univariate linear regression analysis was performed to evaluate the relationship between glenoid retroversion and normalized posterior deltoid area in controls, Walch A-type GHOA, and Walch B-Type GHOA (Figure 2). Results of univariate regression were insignificant for controls ($R^2 = 0.003$; $P = .569$) and for Walch A-type GHOA ($R^2 = 0.001$; $P = .860$). Results of univariate regression were significant for Walch B-type GHOA ($R^2 = 0.102$; $P = .020$).

Given the results of the univariate analysis, a multivariate linear regression analysis was performed for the Walch B-type subgroup to control for the effects of age, gender, and height on glenoid retroversion (Table III). While controlling for the above covariates, the association between glenoid retroversion and normalized posterior deltoid area remained (standardized beta = 0.309; $P = .027$).

Reliability analysis

Two readers measured anterior and posterior deltoid areas for all patients in the study. Intraclass correlation coefficients were 0.886, 0.919, and 0.948 for anterior, posterior, and total deltoid areas, respectively. These values represent strong agreement between readers for all area measurements.

Discussion

The purpose of this study was to determine if there were differences in cross-sectional deltoid muscle area between control and GHOA patients and, furthermore, to determine whether or not an association exists between the proportion of the deltoid that is posterior to the scapular line and glenohumeral retroversion in patients with GHOA. We showed that there was no difference in overall deltoid muscle area between control and arthritic shoulders but that these groups differed in the distribution of deltoid muscle area, with arthritic shoulders demonstrating larger normalized posterior deltoid area. In addition, we demonstrated that there was a positive association between glenoid retroversion and normalized posterior deltoid area in Walch B-type shoulders. Taken together, our findings support the hypothesis that muscular imbalance may contribute to the development of eccentric glenohumeral arthritis.

We determined that there was no difference in absolute anterior, posterior, or total deltoid muscle areas between control patients and patients with primary GHOA. This finding contrasts with GHOA-related changes demonstrated in the rotator cuff.[1,5,7,9,11,15] Multiple authors have shown that GHOA is associated with a high prevalence of rotator cuff atrophy and fatty infiltration.[7,11] Based on the present study, it appears that the deltoid does not experience similar atrophy in the setting of end-stage GHOA. The pathogenesis of rotator cuff dysfunction in GHOA has not been fully described. It is possible that rotator cuff atrophy and fatty infiltration are related to disuse secondary to pain and decreased range of motion in OA. Lapner et al demonstrated improvements in rotator cuff fatty infiltration after TSA, indirectly supporting this hypothesis.[11] It is also possible that rotator cuff dysfunction precedes end-stage arthritis and contributes to its development by destabilizing the glenohumeral joint and reducing shock absorption.[5] The finding that the deltoid muscle is not atrophied in patients with GHOA relative to controls may indirectly support the hypothesis that rotator cuff dysfunction may be cause instead of consequence of primary GHOA. However, this question will ultimately be best answered with longitudinal studies documenting changes in the rotator cuff and deltoid in arthritic shoulders over time.

The present study demonstrated that there was a difference between controls and patients with GHOA in normalized deltoid area, defined as the proportion of deltoid that was either anterior or posterior to the scapular line, with GHOA patients demonstrating a higher proportion of posterior deltoid muscle area than control subjects. This finding is similar to previous research demonstrating changes in rotator cuff muscle areas between control and GHOA patients. Chalmers et al showed that patients with GHOA have changes in the rotator cuff characterized by a proportional decrease in supraspinatus area and a proportional increase in subscapularis area relative to controls.[5] Taken together, these data suggest that GHOA is associated with changes in the distribution of muscle area in both the deltoid and the rotator cuff relative to non-arthritic shoulders. These data further contribute to the hypothesis that muscle dysfunction may precede end-stage arthritis and contribute to its development by destabilizing the glenohumeral joint and reducing shock absorption. Again, longitudinal studies may be helpful to fully elucidate this relationship.

Within the present study, there were no differences in absolute or normalized deltoid areas between patients with Walch A-type (concentric) and Walch B-type (eccentric) GHOA. However, glenoid retroversion associated with normalized posterior deltoid area in Walch B-type shoulders. There was no association between glenoid retroversion and normalized posterior deltoid area in Walch A-type or control shoulders. These findings are similar to prior research on the rotator cuff.[1,5,9,15] Most similar to the current study, Chalmers et al showed that there were not proportional differences in rotator cuff muscle area between Walch A-type and B-type osteoarthritis patients. However, within the Walch B-type subgroup, glenoid

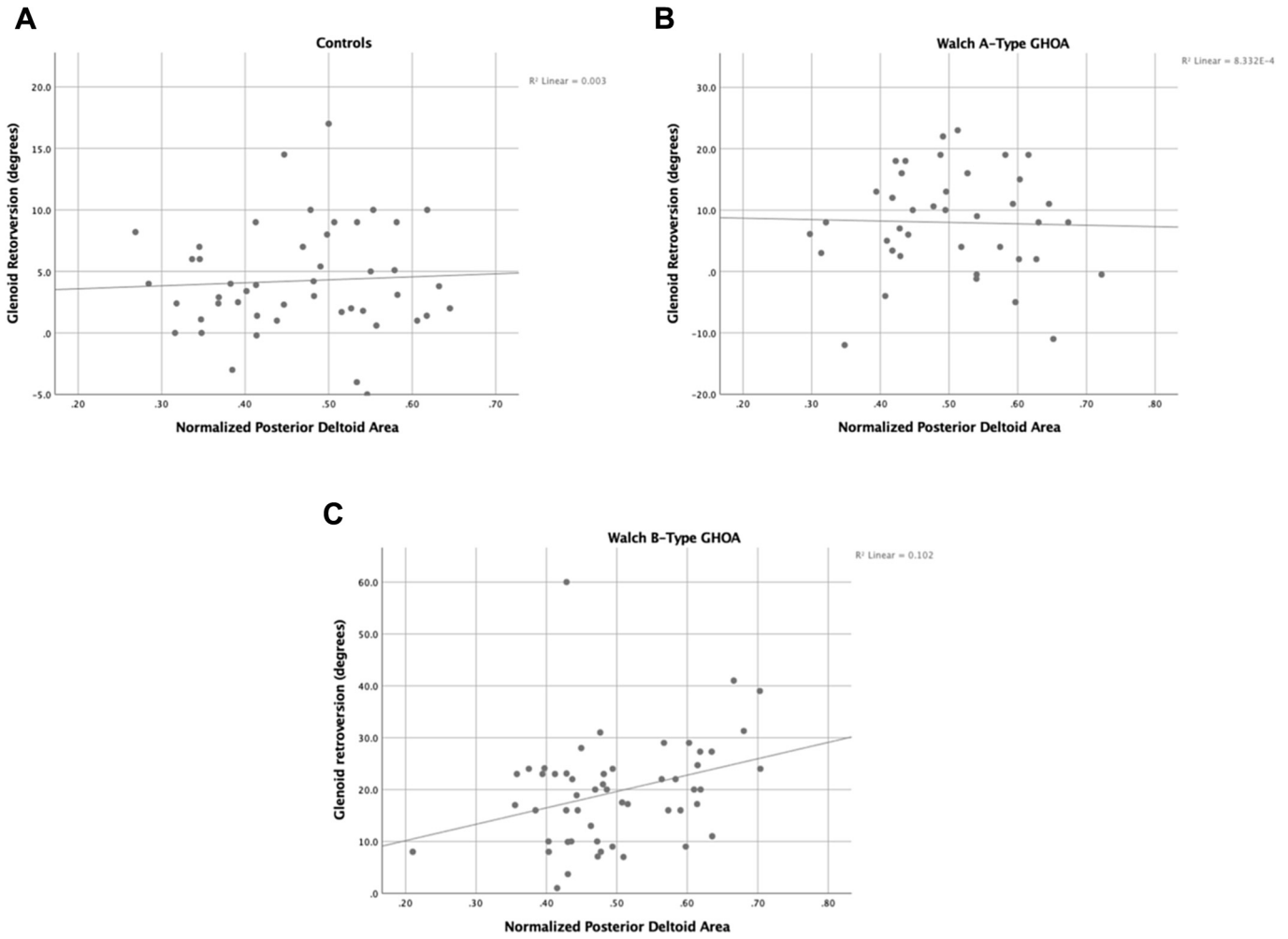

Figure 2 Univariate regression analysis. Scatterplots demonstrating the association between normalized posterior deltoid area (x-axis) and glenoid retroversion (y-axis) for controls (**A**), Walch A-type glenohumeral osteoarthritis (**B**), and Walch B-type osteoarthritis (**C**). Results of univariate regression were insignificant for controls ($R^2 = 0.003$; $P = .569$) and for Walch A-type GHOA ($R^2 = 0.001$; $P = .860$). Results of univariate regression were significant for Walch B-type GHOA ($R^2 = 0.102$; $P = .020$)

Table III
Multivariate linear regression analysis for glenoid retroversion in Walch B-type GHOA.

Variable	Beta	Standard error of beta	Standardized beta	*P* value	Change in R^2
Normalized posterior deltoid area	30.549	13.35	0.309	**.027**	0.102
Age (yr)	0.014	0.165	0.013	.933	0.013
Height (cm)	0.309	0.201	0.309	.131	0.041
Gender	−7.118	4.326	−0.342	.106	0

GHOA, glenohumeral osteoarthritis.
Normalized posterior deltoid area was associated with glenoid retroversion using age, height, and gender as confounding variables. The association between normalized posterior deltoid area and glenoid retroversion remained significant (Standardized beta = 0.309; $P = .027$). None of the other covariates in the model demonstrated statistical significance.
Bolded values indicate statistical significance for a *P* value cut-off $\leq .05$.

retroversion was positively associated with the proportion of the rotator cuff that was supraspinatus and infraspinatus and negatively associated with the proportion of the rotator cuff that was subscapularis. These associations were not present in Walch A-type GHOA or control patients.[5] Similarly, Aleem et al retrospectively reviewed 370 preoperative scans of patients undergoing TSA and demonstrated that glenoid retroversion was associated with an increased ratio of posterior to anterior rotator cuff muscle area in patients with GHOA.[1] Taken together with prior research, the current data further support the hypothesis that, while concentric arthritis may not be associated with muscle imbalance, eccentric arthritis is associated with muscular imbalance about the shoulder joint characterized by proportionally increased muscle area posteriorly and proportionally decreased muscle area anteriorly. In the axial plane, we speculate that a larger posterior deltoid and infraspinatus pull the humerus posteriorly into posterior subluxation, which, over years of wear, ultimately results in retroversion and biconcavity.

This study has several limitations. This is a retrospective study which relied on data collection from the electronic medical record. GHOA patients and controls were selected based on the availability of imaging and the study data is subject to possible selection bias as a result. Control patients were imaged for a variety of reasons and

do not represent a uniform cohort. Chart review was performed to exclude patients with a nonfunctioning deltoid, known rotator cuff tear, rotator cuff tear arthropathy, previous surgical rotator cuff repair, history of inflammatory arthritis, or prior proximal humerus fracture. However, there is the possibility that chart review did not completely capture this exclusion criteria. Furthermore, there are many medical comorbidities and aspects of the patient history which were not included in the analysis and could theoretically influence cross-sectional muscle area.

There are measurement limitations associated with this study. First, the determination of what constitutes anterior from posterior deltoid must be defined. This is in contrast to similar research in the rotator cuff in which all muscle areas have been measured using the cross-sectional area from anatomically defined individual rotator cuff muscles. We chose to use the scapular line as a logical demarcation for anterior / posterior deltoid, as muscle fibers behind the scapular line should exert a posteriorly directed force on the humerus relative to the glenoid and vice versa. In addition, the center of the glenoid face, which was used along with other scapular landmarks to reliable create the scapular line, may be altered by the formation of osteophytes in the GHOA compared with controls. Thus, variations in the glenoid face due to bone formation in osteoarthritis may influence the determination of anterior and posterior deltoid used in this study. Finally, the study does not assess changes in muscle area and GHOA over time. While the association between deltoid asymmetry and glenoid retroversion in the study are intriguing, the study design is inherently unable to demonstrate causation. Longitudinal data are required to determine whether axial plane imbalance in the deltoid contributes to the development of GHOA.

Conclusions

GHOA is not associated with deltoid atrophy, calling into question the suggestion that periarticular muscular atrophy in GHOA is secondary to disuse. Increasing normalized posterior deltoid area associates with increased glenoid retroversion in patients with Walch B-type glenoid morphology. Muscular imbalance may play a role in the etiology or progression of the glenoid deformity observed in eccentric GHOA.

Disclaimer

Dillon O'Neill, Garrett Christensen, Bradley Hillyard, and Jun Kawakami, their immediate family, and any research foundation with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this article. Robert Tashjian is a paid consultant for Zimmer and Mitek; has stock in Conextions, INTRAFUSE, Genesis, and KATOR; receives intellectual property royalties from Imascap, Shoulder Innovations, and Zimmer; receives publishing royalties from Springer, and serves on the editorial board for the Journal of Orthopaedic Trauma, the American Journal of Orthopedics, the Journal of Shoulder and Elbow Arthroplasty and the Journal of the American Academy of Orthopaedic Surgeons. Peter Chalmers is a paid consultant for Mitek and Arthrex, receives intellectual property royalties from Depuy, and serves on the editorial board for the Journal of Shoulder and Elbow Surgery.

This study did not receive any support.

The work for this article was performed at the University of Utah in Salt Lake City, UT, USA.

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