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Radiographic progression of arthritic changes in shoulders with degenerative rotator cuff tears

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Abstract

Background—Very little longitudinal information has been available regarding the relationship of cuff tears and arthritis. The purpose of this study was to determine the midterm risk of and risk factors for rotator cuff tear arthropathy progression in a cohort of subjects with an asymptomatic rotator cuff tear.

Methods—Baseline (visit 1), 5-year (visit 2), and most recent follow-up (visit 3) radiographs were reviewed in a cohort of 105 subjects enrolled for longitudinal surveillance of asymptomatic degenerative rotator cuff tears and 33 controls. The radiographs were assessed in a blinded, randomized fashion by 3 observers who graded glenohumeral arthritic changes using the Hamada scores, Samilson-Prieto (SPO) scores, and acromiohumeral interval (AHI).

Results—Osteoarthritis (SPO classification), cuff tear arthropathy (Hamada classification), and AHI progressed between visits 1 and 3 (median, 8 years; $P < .001$ in all cases). SPO progression was not significantly different for partial- vs. full-thickness vs. control baseline tear types ($P = .19$). Both full-thickness and partial-thickness tears had greater progression in Hamada scores than controls did in the first 5 years of follow-up ($P = .02$ and $P = .03$, respectively), but scores did not differ between partial- and full-thickness tears. Tears with and without enlargement did not differ in progression in SPO grade, Hamada grade, or AHI.

Conclusions—Glenohumeral arthritic changes progress significantly but remain minimal within an 8-year period in early to moderate degenerative cuff disease. Whereas the presence of a rotator

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cuff tear influences progression in Hamada grade, the magnitude of radiographic progression is not influenced by tear severity or enlargement at midterm time points.

Level of evidence—Level II; Prognosis Study

Keywords

Rotator cuff tear; rotator cuff tear arthropathy; osteoarthritis; natural history; shoulder pain; degenerative joint disease

The time line and risks of progression of degenerative rotator cuff disease to advanced rotator cuff tear arthropathy (RCTA) are unknown. An improved understanding of the risk factors for radiographic progression of cuff tears may improve treatment paradigms for patients with degenerative cuff tears. For many patients, rotator cuff tears progress over time in both size and symptoms.^{12,17,19} The end stage of this progression is the development of a characteristic set of degenerative changes including proximal humeral migration, acromial acetabularization, and glenohumeral chondral and bone loss, collectively described as RCTA.^{10,20,21} Although historically few reliable surgical options existed for this condition,⁵ the release of modern reverse total shoulder arthroplasty has led to an ever-increasing number of patients undergoing arthroplasty for the degenerative sequelae of rotator cuff tears.^{6,15,25}

RCTA was originally described by Neer >3 decades ago²⁰; however, the natural history remains poorly understood. Small, retrospective studies have described worsening radiographic deterioration in 39% to 92% of patients with long-term follow-up of surgically treated patients with massive rotator cuff tears.^{8,21,31,32} Alternatively, some retrospective series with long-term follow-up have described durable clinical results with rotator cuff repair.^{1,3,7,23,26} No study has prospectively examined rotator cuff tears radiographically to describe the progression of RCTA in a nonsurgical cohort. The risk factors for progression thus remain unknown.

Understanding the likelihood of progression of RCTA and the clinical attributes that modulate this progression is of fundamental importance to defining the natural history of rotator cuff disease. Identification of tear-related risk factors that correlate with arthropathy progression may help focus preventive treatment and direct appropriate counseling of the patient regarding the potential need for shoulder arthroplasty. The purpose of this study was to determine the rate of progression of RCTA in a cohort of subjects with an asymptomatic rotator cuff tear and to analyze factors associated with arthropathy progression.

Methods

The subjects included in the study are part of a larger cohort of patients with asymptomatic rotator cuff tears who presented for evaluation of shoulder pain secondary to rotator cuff disease in the contralateral shoulder.^{12,16} Control subjects with ultrasound scans demonstrating a normal rotator cuff in the study shoulder and a painful rotator cuff tear in the contralateral shoulder were also enrolled. Inclusion criteria for study subjects were (1) bilateral shoulder ultrasonography performed to investigate unilateral rotator cuff-based

pain, (2) painful rotator cuff disease in the symptomatic shoulder, (3) a rotator cuff tear in the asymptomatic shoulder at the time of study enrollment, (4) no history of trauma to either shoulder and no traumatic episode through the study period, and (5) a minimum of 5 years of radiographic follow-up. Exclusion criteria were (1) any past or current pain in the “asymptomatic” shoulder at the time of enrollment, (2) continuous use of narcotic or nonsteroidal anti-inflammatory drugs in the 3 months before enrollment, (3) a traumatic episode affecting the symptomatic shoulder, (4) inflammatory arthritis, (5) radiographic evidence of osteoarthritis in the asymptomatic shoulder at the time of enrollment, (6) upper extremity weight-bearing demands, and (7) a subscapularis tendon tear in the asymptomatic shoulder.

Study protocol

Subjects were enrolled from the clinical practices of 3 surgeons during a 30-month period. The study protocol has been described in depth in previous studies.^{12,16} In brief, a trained research nurse performed annual standardized physical examinations with measurement of active range of motion and isometric strength. Patients completed standardized outcome scores including a visual analog scale for pain, the American Shoulder and Elbow Surgeons score, and the Simple Shoulder Test. Patients were specifically questioned about pain at each visits and asked to contact the study coordinator if they developed any new onset shoulder pain. Subjects also underwent standardized annual ultrasound examinations to describe tear width, length, type, fatty atrophy, and enlargement as previously defined.¹² Shoulder ultrasonography, as previously described,^{14,16,29} was performed for each shoulder at baseline and annually thereafter. Sonographic data used for this study included baseline, 5-year, and final follow-up data. Tear enlargement was defined as increase in tear width or length of 5 mm from baseline values for full-thickness tears only.

Radiographic analysis

Radiographs were standardized as previously described¹³ by specifically selected and trained technicians. Radiographs were saved in the picture archiving and communication system (Siemens, Munich, Germany). Baseline, 5-year, and final follow-up radiographs were analyzed. Our radiology department then randomized and anonymized both the initial and final follow-up radiographs to eliminate bias. These images were then exported into the raw Digital Imaging and Communications in Medicine (DICOM) format. Each radiograph was then evaluated in this anonymized and randomized fashion by 3 independent observers (2 fellows [P.N.C. and D.H.S.] and one attending surgeon [J.D.K.]) in a DICOM viewer (OsiriX; Pixmeo, Geneva, Switzerland).

Each radiograph was graded using the Hamada classification of RCTA.¹⁰ Each radiograph was also graded using the Samilson and Prieto (SPO) classification of glenohumeral osteoarthritis.²² This classification has been demonstrated to be reliable.⁴ Acromiohumeral interval (AHI) was also measured on each true anteroposterior radiograph. The radiograph was performed with the shoulder in slight abduction and neutral rotated but was not fluoroscopically controlled. This distance was defined as the shortest measurable distance between the sclerotic line of the undersurface of the acromion and the superior humeral head articular surface. In those cases in which a traction enthesophyte at the origin of the

coracoacromial ligament could be visualized on the scapular Y view, all attempts were made to measure from the native acromion with this enthesophyte excluded. AHI has been demonstrated to have an acceptable if imperfect interobserver reliability.^{2,18,24,30}

Statistical analysis

For SPO and Hamada scores, interobserver reliability was calculated using weighted κ values, and consensus scores were used in the analysis. For AHI, interobserver reliability was calculated using intraclass correlation coefficients, and mean values were used in the analysis. Radiographs were divided into baseline (visit 1), 5-year follow-up (visit 2), and most recent follow-up (visit 3). Progression in SPO and Hamada scores over time was statistically compared using generalized estimating equations with a multinomial distribution, a cumulative logit link function, and an independent correlation structure; progression in AHI was analyzed using a mixed model repeated-measures analysis of variance with a heterogeneous autoregressive covariance structure. Using similar models, progression was compared between tear types (for SPO and Hamada grades) and between tears that do and do not enlarge (for SPO grade, Hamada grade, and AHI), in which the focus was on the interaction between visit and tear type and between visit and tear enlargement, respectively. Before the interaction analyses, Hamada category was dichotomized (Hamada 1 vs. Hamada > 1) because of nonunique infinite estimates. Statistical contrasts within the generalized estimating equations and repeated-measures analysis of variance models were used for specific pairwise comparisons and are Šidák adjusted. The Wilcoxon 2-sample test was used to compare tear severity by dichotomized Hamada category. The association of baseline tear length and width with change in SPO and Hamada grades was assessed with Spearman correlations. Spearman correlations also assessed the association between change in tear size and change in SPO grade, Hamada grade, and AHI.

Results

There were 138 patients who met inclusion criteria, with a total of 397 radiographs evaluated. Interobserver agreement was excellent. For SPO scores, weighted κ was 0.68 (95% confidence interval, 0.63–0.72). For Hamada scores, weighted κ was 0.72 (0.67–0.78). For AHI, intraclass correlation co-efficient was 0.88 (0.86–0.90). Of the included patients, 24% were control patients, 28% had partial-thickness tears at baseline, and 49% had full-thickness tears at baseline. Final radiographic follow-up occurred at median of 8 years (range, 5.9–12 years). Our cohort mostly included patients with small to medium-size tears. Baseline tear size for partial-thickness tears was 7.1 mm (interquartile range, 2.8) in length and 8.9 mm (2.7) in width, and final tear size was 12.7 mm (7.9) in length and 12.5 mm (8.5) in width. Baseline tear size for full-thickness tears was 13.2 mm (8.3) in length and 12.4 mm (7.2) in width, and final tear size was 21.9 mm (9.5) in length and 18.9 mm (9.4) in width.

SPO grade, Hamada grade, and AHI all progressed during the initial 5 years of the study period but not between the 5-year and most recent follow-up visits. Within SPO grades, whereas 87% of patients were grade 0 at visit 1, only 70% were by visit 3. SPO grade

significantly progressed from visit 1 to 2 and visit 1 to 3 but not visit 2 to 3. Similarly, within Hamada grades, 93% were grade 1 at visit 1 whereas 77% were grade 1 at visit 3. Hamada grade significantly progressed from visit 1 to 2 and visit 1 to 3 but not visit 2 to 3. Similar to SPO and Hamada grades, AHI progressed from visit 1 to 2 and visit 1 to 3 but not visit 2 to 3 (Table I).

Radiographic progression was compared with baseline tear severity. Baseline tear severity grouping (control vs. partial-thickness tear vs. full-thickness tear) did not influence SPO progression (Table II). Baseline tear severity grouping (control vs. partial-thickness tear vs. full-thickness tear) did not influence Hamada progression (Table II), but in post hoc analyses when the control group was compared with either the partial-or full-thickness tears between visits 1 and 2, progression did differ (Table II). Tear size at visits 2 and 3 was larger for shoulders with Hamada grade >1 changes compared with those with Hamada grade 1 changes in all dimensions (Table III). Change in AHI did not correlate with change in either tear width or length in any visit comparison ($P>.12$ in all cases).

Tear enlargement during follow-up was not associated with radiographic progression as measured with SPO grades, Hamada grades, or AHI ($P>.09$ in all cases; Table IV).

Discussion

No prior studies have described the natural history of RCTA or the tear-related risk factors for progression of RCTA. Identification of tear-related risk factors is critical for prevention and prognosis and may play a role in surgical indications. The purpose of this study was to determine the rate of progression of RCTA in a cohort of subjects with an asymptomatic rotator cuff tear and to analyze factors associated with arthropathy progression. Although our cohort mostly included patients with small to medium-size tears, SPO grade, Hamada grade, and AHI all progressed during the initial 5 years of the study period. Although progression in Hamada and SPO grades was not associated with baseline tear severity grouping, in post hoc analyses, full- and partial-thickness tears had more progression in Hamada grades than the control group did during the first 5 years of follow-up. Tear enlargement in full-thickness tears was not associated with radiographic progression as measured using SPO grades, Hamada grades, or AHI.

Within our cohort, during a median of 8 years of follow-up, AHI decreased from 8.9 ± 1.7 to 7.7 ± 2.6 . Many previous studies have measured AHI progression. Gerber et al described a decrease in AHI from 7.4 to 4.9 mm with 10-year follow-up of 46 patients who underwent latissimus dorsi transfer for massive rotator cuff tears.⁸ Jost et al observed 20 patients who sustained imaging-documented structural failure after a rotator cuff repair, noting progression of proximal humeral migration between 3.2-year follow-up (8.4 mm) and 7.6-year follow-up (7.4 mm).¹¹ Keener et al performed an in-depth analysis of risk factors for proximal humeral migration within the cohort used in this study. These authors found proximal humeral migration to be significantly greater in tears with symptoms, tears with involvement of the infraspinatus, and tears with larger size, although the last applied only to larger tears. In multivariate analysis, tear size was the strongest predictor of migration.¹³

Within our cohort, 17% of patients progressed from SPO grade 0 to 1, and 16% of patients progressed from Hamada grade 1 to 2; however, most patients had small or medium-size tears and minimal arthritic changes (93% Hamada grade 1 and 87% SPO grade 0) at baseline and thus may have been at low risk for arthritic progression. Patients who present with larger tears and more advanced arthritic changes may have more accelerated progression. In addition, within our cohort, more patients had full-thickness tears (48%) than partial-thickness tears (28%). Thus, these findings may be more representative of the natural history of full-thickness tear than of partial-thickness tear, which may progress more slowly. Numerous previous studies have documented progression of RCTA with time. Gerber et al followed up 46 shoulders for a minimum of 10 years after latissimus dorsi transfer for massive rotator cuff tears and found that 48% of patients progressed at least 1 SPO grade from a mean of 0.4 to 1.0 and 57% of patients progressed at least 1 Hamada grade from a mean of 1.2 to 2.0, all of which were significant.⁸ Jost et al followed up 20 patients with imaging-documented structural failure of a rotator cuff tear for a mean of 7.6 years, of whom only 3 patients progressed between 3.2 and 7.6 years of follow-up, with no significant increase in degeneration grade, although only the SPO system was used in this study.¹¹ Paxton et al followed up 15 patients for a minimum of 10 years after a repair of a massive rotator cuff tear, all of whom had an imaging-documented retear, and found that 93% of patients progressed at least 1 Hamada grade, with 53% of patients having grade 3 or greater changes.²¹ Zumstein et al followed up 23 patients for a mean of 9.9 years after open repair of a massive rotator cuff tear, with a 57% retear rate and significant progression from a mean SPO grade 1.3 to 2.0, with 61% of patients having grade 2 changes.³²

Progression in Hamada and SPO grades was not associated with baseline tear severity grouping; in post hoc analyses, full- and partial-thickness tears had more progression in Hamada grades than the control group did during the first 5 years of follow-up. However, progression of SPO grade, Hamada grade, and AHI was similar for tears that did and did not enlarge. No previous comparative studies have examined for a correlation between tear characteristics or clinical findings and the progression of RCTA, although noncomparative series have suggested that large, irreparable recurrent tears have rapid progression rates.^{21,31,32} The Hamada system is specific to RCTA and thus may be a more sensitive system than the SPO system. Overall, our results confirm these findings, suggesting that RCTA may be more rapidly progressive in patients with tears compared with those without tears.

Our study has several limitations. AHI was used to measure proximal humeral migration. This measurement was selected because it can be easily measured clinically and thus provides the surgeons with the most facile clinicopathologic correlate. However, it is affected by heterogeneities in patient positioning, gantry and plate positioning, humeral rotation, scapular positioning, and acromial morphology, all of which may limit reliability.^{2,9,18,30} Although prior studies have described low interobserver reliability with this measure, within our data set, interobserver reliability was high, probably because a standardized protocol was used for all radiographs to mitigate these effects. Similarly, both the SPO and Hamada systems provide an incomplete understanding of the extent of degenerative changes with poor resolution, although both have been extensively used within the literature. Several other classification systems exist for RCTA, and our findings may not

apply to those systems. Some patients were excluded from our study because of incomplete or inadequate radiographs. Ultrasonography is user dependent and was used to measure rotator cuff tear progression, although this technique has been demonstrated to be highly accurate at our institution.^{27,28} In addition, this study provides only midterm (8-year) follow-up. RCTA is expected to be slowly progressive, and thus longer term follow-up will be necessary to further examine the relationship between initial tear severity and tear progression and the progression of RCTA. We did not examine for any connection between progression of arthritic changes and the patient's symptoms. Finally, because of the relatively small tear size and lack of arthritic changes at baseline within our cohort, these patients may have been at a relatively low risk for arthritic progression, which limits the generalizability of our findings. Studies with cohorts of larger tears with longer follow-up will be necessary to determine if other rotator cuff tear-related risk factors for arthropathy progression can be found.

Conclusion

Glenohumeral arthritic changes progress significantly but remain minimal within an 8-year period in early to moderate degenerative cuff disease. Whereas the presence of a rotator cuff tear influences progression in Hamada grade, the magnitude of radiographic progression is not influenced by tear severity or enlargement at midterm time points.

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Table 1

Radiographic classifications for each visit (baseline, 5-year follow-up, and most recent follow-up at a median of 8 years)

Variable	Score	Visit 1 (No. = 138)	Visit 2 (No. = 138)	Visit 3 (No. = 118)	P values for comparisons across visits
SPO, No. (%)	0	120 (87)	106 (77)	83 (70)	Overall $P < .0001$
	1	18 (13)	29 (21)	30 (25)	Visit 1 vs. 2, $P = .006$
	2	0 (0)	3 (2)	4 (3)	Visit 1 vs. 3, $P < .0001$
	3	0 (0)	0 (0)	1 (1)	Visit 2 vs. 3, $P = .23$
Variable	Score	Visit 1 (No. = 138)	Visit 2 (No. = 138)	Visit 3 (No. = 118)	P values for comparisons across visits
Hamada, No. (%)	1	128 (93)	115 (83)	91 (77)	Overall $P = .0004$
	2	10 (7)	19 (14)	20 (17)	Visit 1 vs. 2, $P = .002$
	3	0 (0)	4 (3)	5 (4)	Visit 1 vs. 3, $P < .0001$
	4	0 (0)	0 (0)	2 (2)	Visit 2 vs. 3, $P = .18$
Variable		Visit 1 (No. = 135)	Visit 2 (No. = 138)	Visit 3 (No. = 118)	P values for comparisons across visits
AHI (mm), mean (SD)		8.9 (1.7)	7.8 (2.2)	7.7 (2.6)	Overall $P < .0001$
					Visit 1 vs. 2, $P < .0001$
					Visit 1 vs. 3, $P < .0001$
					Visit 2 vs. 3, $P = .99$

No., number of patients; SPO, Samilson-Prieto; AHI, acromiohumeral interval; SD, standard deviation.

Comparison of change in Samilson-Prieto (SPO) and Hamada grades by baseline tear type

Table II

Variable	Score by study baseline tear type	Visit 1 (No. = 138)	Visit 2 (No. = 138)	Visit 3 (No. = 118)	P values for comparisons across visits and study baseline tear type
SPO, No. (%)	Control				Overall $P = .19$
	0	29 (88)	25 (76)	23 (85)	Control vs. partial, visit 1 to visit 2, $P = .99$
	1	4 (12)	7 (21)	2 (7)	Control vs. partial, visit 1 to visit 3, $P = .98$
	2	0 (0)	1 (3)	2 (7)	Control vs. full, visit 1 to visit 2, $P = .49$
	Partial				Control vs. full, visit 1 to visit 3, $P = .85$
	0	36 (95)	32 (84)	24 (77)	Partial vs. full, visit 1 to visit 2, $P = .46$
	1	2 (5)	5 (13)	5 (16)	Partial vs. full, visit 1 to visit 3, $P = .18$
	2	0 (0)	1 (3)	1 (3)	
	3	0 (0)	0 (0)	1 (3)	
	Full				
	0	55 (82)	49 (73)	36 (60)	
	1	12 (18)	17 (25)	23 (38)	
	2	0 (0)	1 (1)	1 (2)	
Hamada, No. (%)	Control				Overall $P = .09$
	1	29 (88)	31 (94)	26 (96)	Control vs. partial, visit 1 to visit 2, $P = .02$
	2	4 (12)	2 (6)	0 (0)	Control vs. partial, visit 1 to visit 3, $P = .17$
	4	0 (0)	0 (0)	1 (4)	Control vs. full, visit 1 to visit 2, $P = .03$
	Partial				Control vs. full, visit 1 to visit 3, $P = .12$
	1	38 (100)	32 (84)	26 (84)	Partial vs. full, visit 1 to visit 2, $P = .98$
	2	0 (0)	6 (16)	4 (13)	Partial vs. full, visit 1 to visit 3, $P = .72$
	3	0 (0)	0 (0)	1 (3)	
	Full				
	1	61 (91)	52 (78)	39 (65)	
	2	6 (9)	11 (16)	16 (27)	
	3	0 (0)	4 (6)	4 (7)	
	4	0 (0)	0 (0)	1 (2)	

No., number of patients.

Table III
Tear characteristics at visits 2 and 3 between Hamada grade 1 and Hamada grade >1

Variable	Visit	Statistic	Hamada score = 1	Hamada score > 1	P value
Tear length (mm)	Visit 2	Median (IQR)	10.0 (6.0)	21.0 (16.0)	.0002
		No.	91	21	
	Visit 3	Median (IQR)	13.0 (11.0)	27.0 (20.0)	.002
		No.	76	18	
Tear width (mm)	Visit 2	Median (IQR)	10.0 (7.5)	13.5 (13.0)	.04
		No.	92	22	
	Visit 3	Median (IQR)	11.5 (7.0)	23.0 (20.0)	.001
		No.	74	22	

IQR, interquartile range; *No.*, number of patients.

Comparison of change in Samilson-Prieto (SPO) grade, Hamada grade, and acromiohumeral interval (AHI) by tear enlargement

Table IV

Variable	Score by enlargement ever in the study	Visit 1 (No. = 67)	Visit 2 (No. = 67)	Visit 3 (No. = 60)	P values for comparisons across visits and tear enlargement
SPO, No. (%)	No enlargement (n = 15)				Overall $P = .18$
	0	13 (87)	11 (73)	6 (46)	No enlargement vs. enlargement, visit 1 to visit 2, $P = .80$
	1	2 (13)	3 (20)	6 (46)	
	2	0 (0)	1 (7)	1 (8)	No enlargement vs. enlargement, visit 1 to visit 3, $P = .16$
	Enlargement (n = 52)				
	0	42 (81)	38 (73)	30 (64)	
	1	10 (19)	14 (27)	17 (36)	
Hamada, No. (%)	No enlargement (n = 15)				Overall $P = .57$
	1	12 (80)	10 (67)	7 (54)	No enlargement vs. enlargement, visit 1 to visit 2, $P = .74$
	2	3 (20)	4 (27)	5 (38)	
	3	0 (0)	1 (7)	1 (8)	No enlargement vs. enlargement, visit 1 to visit 3, $P = .50$
	Enlargement (n = 52)				
	1	49 (94)	42 (81)	32 (68)	
	2	3 (6)	7 (13)	11 (23)	
	3	0 (0)	3 (6)	3 (6)	
	4	0 (0)	0 (0)	1 (2)	
AHI (mm), mean (SD)	No enlargement (n = 15)	8.4 (1.9)	7.2 (2.5)	6.9 (3.1)	Overall $P = .80$
	Enlargement (n = 52)	8.6 (1.9)	7.1 (2.4)	7.1 (2.7)	No enlargement vs. enlargement, visit 1 to visit 2, $P = .82$
					No enlargement vs. enlargement, visit 1 to visit 3, $p = 1.0$

No., number of patients; SD, standard deviation.