Patient Age and Surgical Intervention as Risk Factors for the Development of Osteoarthritis After Posterior Shoulder Instability

A Population-Based Study

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Background: Diagnosis and treatment of posterior shoulder instability (PSI) has improved with advances in imaging and surgical technique. However, the relationship between PSI and osteoarthritis (OA) remains unclear.

Purpose: To evaluate a population-based cohort to (1) determine the rate of symptomatic OA, (2) identify patient characteristic risk factors for OA, and (3) evaluate the effect of posterior capsulolabral repair on OA progression.

Study Design: Case-control study; Level of evidence, 3.

Methods: After review of 324 patient records, we included 115 patients (14 female, 101 male) diagnosed with PSI between January 1994 and July 2012 with an average follow-up of 12.5 years (range, 5-23 years). Medical records were reviewed for patient characteristics, injury characteristics, surgical details (if any), and radiographic progression of OA. Kaplan-Meier survival was used to estimate survival free of OA; characteristics associated with OA progression were determined via univariate Cox regression models, and associated 95% CIs and hazard ratios (HRs) are presented.

Results: Overall, 14% (16/115) of patients had radiographic progression of symptomatic glenohumeral arthritis, with 5-year survival of 88.3% (95% CI, 79.7%-97.3%). Older age at the time of instability diagnosis was associated with arthritis progression (10-year unit HR, 1.95; 95% CI, 1.26-3.03). Patients who underwent surgery demonstrated increased radiographic progression of OA (HR, 4.03; 95% CI, 1.23-13.23). There was a trend of increased OA in patients treated with labral debridement compared with repair despite lower baseline levels of OA; however, this difference was not statistically significant (*P* = .09).

Conclusion: Symptomatic glenohumeral arthritis is not seen routinely in patients with PSI. Although uncommon, an age of at least 30 years at the time of diagnosis and surgical intervention were identified as risk factors for developing symptomatic arthritis at long-term follow-up.

Keywords: glenohumeral arthritis; posterior capsulolabral repair; posterior labral repair; posterior shoulder instability; shoulder instability

Posterior shoulder instability (PSI) is relatively uncommon, accounting for only 2% to 10% of all cases of shoulder instability with an incidence of 4.6 per 100,000 person-years.[2,25] Unlike anterior shoulder instability, which usually stems from a traumatic incident, PSI can result from repetitive microtrauma associated with sporting activities or from a single traumatic subluxation or dislocation episode.[5,9]

Patients can present with vague symptoms of persistent shoulder pain, weakness, or a decline in sports performance.[8,16]

Surgical options after failure of conservative treatment include arthroscopic or open capsulolabral repair or debridement of the labrum.[1,4] Capsulolabral repair for PSI has been shown to reliably improve pain and clinical function while also providing a high rate of return to sports for patients who experience recurrent instability, and debridement remains an option for patients who experience primarily persistent pain without symptoms of recurrent

posterior instability.[3-5,14,17] While early and midterm clinical outcomes are excellent, there is a paucity of information on the long-term outcomes in patients with PSI. Specifically, the risk of developing arthritis is not well understood in this population of patients with PSI.

Samilson and Prieto[19] found, in a limited population, that patients with PSI had a higher rate of moderate or severe arthrosis when compared with patients with anterior shoulder instability. In contrast to PSI, the relationship between anterior glenohumeral instability and arthritis has been well established. After surgical stabilization, arthritis is present in as many as 69% of patients at minimum 10-year follow-up.[7,15] Risk factors for the development of arthritis include older age at the time of instability, increased time between diagnosis and treatment, and bony involvement.[7] Furthermore, the severity of dislocation arthropathy has been associated with older age, the number of dislocations, and number of anchors used during surgical fixation.[16]

The purpose of this study was to evaluate a cohort of patients with PSI to (1) determine the rate of progression to symptomatic osteoarthritis (OA), (2) identify patient characteristic risk factors for the development of OA, and (3) evaluate the effect of posterior capsulolabral repair on this progression. We hypothesized that symptomatic OA would be uncommon after PSI and that posterior capsulolabral repair would decrease the rate of OA in the population with PSI.

METHODS

This population-based study was conducted using the Rochester Epidemiology Project, a medical records linkage system that provides complete medical records for all Olmsted County residents in the state of Minnesota between 1966 and the present.[18] Details and validity of the epidemiological tool have previously been reported.[22,23] Institutional review board approval was obtained from the study institution and participating institutions in Olmsted County, Minnesota, and consent was provided by the patients.

We queried this system for all patients between January 1, 1994, and July 31, 2012, with an International Classification of Diseases, 9th Revision, diagnosis code for PSI for the study. For each patient, medical records from all health care providers in Olmsted County were reviewed to confirm the diagnosis of posterior labral pathology. Patients were included if they had (1) a clinical diagnosis of PSI, (2) at least 1 imaging test (radiograph, magnetic resonance imaging [MRI] scan, and/or computed tomography [CT] scan) consistent with posterior instability, and (3) a minimum of 5 years of follow-up from diagnosis. Confirmatory imaging included axillary radiograph showing posterior glenohumeral dislocation, an MRI/ magnetic resonance angiography scan showing a posterior labral tear, or a CT scan showing posterior glenoid injury and/or a reverse Hill-Sachs defect on the anterior humeral head. Operative reports (for those undergoing surgery) were also reviewed to confirm injury to the posterior labrum. Patients were excluded if they did not meet all 3 of the inclusion criteria above or if they had a history of seizure, multidirectional instability, anterior-only instability, or superior labrum anterior-posterior tear pathology without evidence of instability.

Descriptive information, past medical history, imaging findings, and treatment details were analyzed. Progression to symptomatic arthritis was identified via (1) the new development of OA symptoms such as pain, decreased motion, and stiffness; (2) evidence of progressive joint space narrowing or other stigmata of OA on shoulder radiographs compared with baseline imaging; and (3) attribution of the shoulder symptoms to progressive OA by the treating clinician.

For patients who underwent surgery, the group was further stratified based on operative procedure undertaken such as simple debridement, limited posterior capsulolabral repair (1-2 anchors), or more extensive posterior capsulolabral stabilization (≥3 anchors). This delineation allowed for assessment of potential association between technique and progression to OA. While labral repair was generally performed for patients who progressed to surgery primarily because of persistent instability, simple debridement was more common in patients who progressed to surgery because of persistent pain without recurrent instability.

Statistical Analysis

Data were collected and stored in Microsoft Excel (2010; Microsoft Corp) and analyzed using JMP software (Version 13; SAS Institute Inc) or R Statistical Software (R Core Team, Foundation for Statistical Computing). After analyzing for parametric/nonparametric assumptions, we compared

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continuous variables between groups using *t* test or Wilcoxon rank sum test. Categorical variables were compared using chi-square analysis or Fisher exact test. The 95% CIs were calculated, and *P* values <.05 were considered significant. Categorical data are presented as numbers (%), whereas continuous variables are presented as means ± SD. Kaplan-Meier estimate was used to calculate survival from symptomatic OA. Univariate Cox proportional hazards regression model was used to evaluate potential predictors of OA progression, and hazards ratios (HRs) are reported.

RESULTS

After review of 324 records, 115 patients (14 female, 101 male) with new-onset PSI between January 1, 1994, and July 31, 2012, met the inclusion criteria. Characteristics are summarized in Table 1. Patients had an average follow-up of 12.5 years (range, 5-23 years), with at least 10 years of follow-up in 78 of 115 patients. The average body mass index (BMI) was 27.5 (range, 16.9-40.6). In this population, there were 59 nonsmokers and 54 current or former smokers. There were 5 patients with diabetes, 22 laborers, and 93 nonlaborers. In terms of age at instability, 59 (51.3%) patients were diagnosed with instability before age 30 years, whereas 56 (48.7%) patients were diagnosed at or after the age of 30 years. There were 32 (27.8%) contact athletes (defined as weightlifting, football, rugby, wrestling, etc), 33 (28.7%) overhead athletes (defined as volleyball, throwing, swimming, rock climbing, etc), and 49 (42.6%) nonathletes.

At a minimum of 5 years after diagnosis, 16 of the 115 patients (14%) demonstrated progression to symptomatic OA. Kaplan-Meier survival analysis showed survival free from OA of 97.9% at 1 year (95% CI, 94.8%-100%), 88.3% at 5 years (95% CI, 79.7%-97.3%), and 80.8% at 10 years (95% CI, 56.3%-88.4%) (Figure 1). Only 1 person progressed to end-stage OA, necessitating a total shoulder arthroplasty at age 53 years.

When evaluating risk factors associated with progression of OA, we found that instability at age ≥30 years was significantly associated with radiographic progression of shoulder arthritis with an HR of 0.08 (95% CI, 0.01-0.59) (Figure 2). In addition, the 10-year unit HR was significant at 1.954 (95% CI, 1.26-3.03) (Table 2). BMI, smoking status, diabetes, heavy labor occupation, number of dislocations, and sport were not associated with OA progression (Figure 3 and Table 2). If the patient underwent surgery, the number of anchors was not associated with OA progression.

In this population, 37 of 115 (32%) patients were treated nonoperatively, while 78 of 115 (68%) underwent surgery. In those patients who did not undergo surgery, 3 of 37 (8%) showed radiographic progression of symptomatic OA. Of the 78 patients who underwent surgery, 13 (17%) showed radiographic progression of shoulder arthritis; the rate of OA progression for those who underwent surgery was greater than for those treated nonoperatively, with an HR of 4.03 (95% CI, 1.23-13.2).

The surgical cohort was further divided into 3 groups: labral debridement only with no anchor placement (22/78;

TABLE 1
Patient Characteristics[a]

	Full Cohort (N = 115)	Progressed to OA (n = 16)
Sex		
Male	101	15
Female	14	1
Age at instability, y		
<30	59	1
≥30	56	15
BMI		
<25	38	4
25-29.9	44	6
30-34.9	21	3
≥35	10	3
Smoker		
Current/former	54	8
Never	59	8
Diabetes mellitus		
No	110	14
Yes	5	2
Work type		
Laborer[b]	22	4
Nonlaborer	93	12
Dislocation		
Never	87	13
Once	13	1
Multiple	14	2
Sport[c]		
None	49	8
Throwing	33	2
Contact	32	6
No. of anchors	(n = 78)	(n = 13)
0	22	6
1	15	1
2	19	2
≥3	22	4

[a]Data are reported as No. of patients. BMI, body mass index; OA, osteoarthritis.
[b]Laborer was defined as occupation where <50% of work hours were spent sedentary or that is well known to require significant physical energy expenditure.
[c]Contact sport was defined as athletic activity that allows for regular body contact between participants and/or objects of significant weight.

28%), posterior capsulolabral stabilization with 1 to 2 anchors (34/78; 44%), and posterior capsulolabral stabilization with ≥3 anchors (22/78; 28%). When stratified by number of anchors, 6 of 22 (27%) of those who underwent labral debridement alone had OA progression, while 3 of 34 (8.8%) of those who underwent limited capsulolabral fixation and 4 of 22 (18%) of those who underwent complete posterior capsulolabral stabilization also showed progression of OA (Table 3). None of the differences between these groups reached statistical significance, although patients who underwent labral debridement alone showed a trend toward increased OA progression (*P* = .09).

In terms of recurrence, within the whole cohort, 4 patients had explicitly documented continued instability, while 16 had recurrent pain. Of those who experienced recurrent
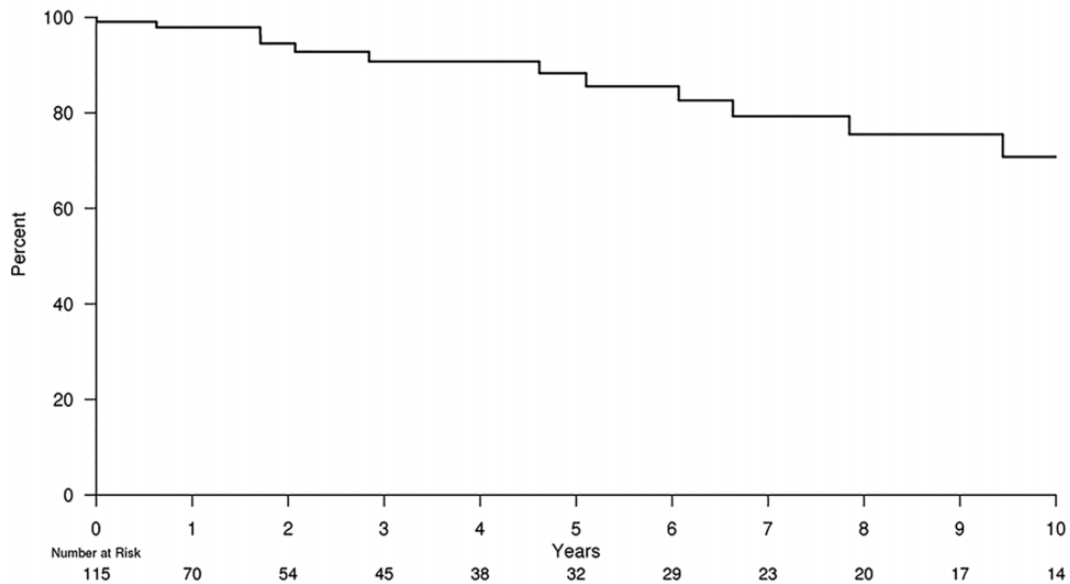
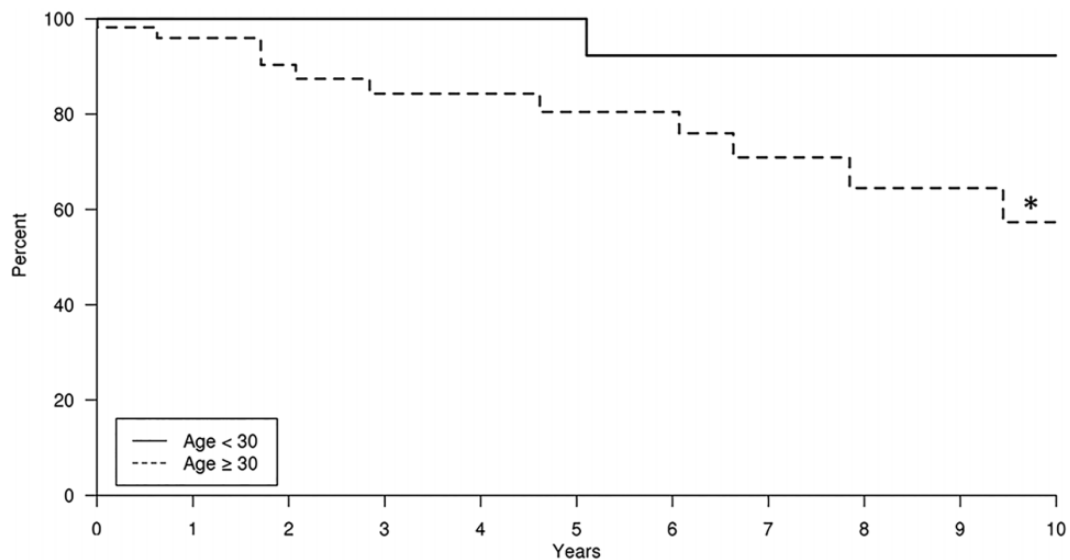**Figure 1.** Rate of survival free from progression to osteoarthritis.

Figure 2. Rates of survival free of osteoarthritis according to age at instability. *P = .003 for validity of the survivorship curve.

instability, 3 had been treated nonoperatively, and 5 of those with recurrent pain had undergone debridement only. Four patients who experienced recurrent pain—all of whom were from the surgically treated group—had documented radiographic progression of OA. Only 2 of these individuals underwent additional surgery: 1 posterior labral debridement and 1 posterolabral repair with 2 anchors.

DISCUSSION

In this population, there was a 14% rate of development of symptomatic OA after a diagnosis of PSI. Although the overall 5-year survivorship from symptomatic OA was 88.3%, the risk for OA was increased in patients aged ≥30 years at the time of diagnosis (HR, 1.22). This risk continued in 10-year units, indicating that overall increasing age at time of diagnosis is associated with increased risk of OA development (HR, 1.95). No other patient characteristics, including BMI and smoking, demonstrated a statistically significant influence on risk for progression to OA. Patients treated operatively demonstrated increased risk for OA development compared with nonoperatively treated patients (HR, 4.03). There was an increase in the development of OA in patients who underwent debridement alone compared with capsulolabral stabilization; however, this did not reach statistical significance.

TABLE 2
Predictors for OA Progression[a]

	Hazard Ratio (95% CI)	P	Overall P
Sex			
Male	3.5 (0.45-27.54)	.234	
Female	Reference		
Age, y			
<30	0.078 (0.01-0.59)	**.014**	
≥30	Reference		
Age per 10 y	1.954 (1.26-3.03)	**.003**	
BMI			.6829
25-29.9	1.416 (0.40-5.04)	.591	
30-34.9	1.096 (0.24-5.01)	.906	
≥35	2.415 (0.53-11.02)	.255	
<25	Reference		
Surgery			
Yes	4.03 (1.23-13.2)	**.040**	
No	Reference		
Smoker			
Current/Former	0.944 (0.35-2.52)	.908	
Never	Reference		
Diabetes mellitus			
Yes	1.609 (0.36-7.18)	.533	
No	Reference		
Work type			
Laborer	1.094 (0.35-3.46)	.878	
Nonlaborer	Reference		
Dislocation			.6891
Once	0.912 (0.12-7.11)	.930	
Multiple	1.945 (0.41-9.16)	.400	
Never	Reference		
Sport			.1014
None	0.323 (0.10-1.02)	.054	
Throwing	0.236 (0.04-1.29)	.095	
Contact	Reference		
No. of anchors			.7749
1	0.329 (0.04-2.87)	.314	
2	0.660 (0.13-3.50)	.626	
≥3	0.878 (0.24-3.29)	.847	
0	Reference		

[a]Boldface P values indicate statistically significant difference compared with reference variable (P < .05). BMI, body mass index; OA, osteoarthritis.

With an average follow-up of 12.5 years, the rate of symptomatic arthritis progression reported in this study is similar to, or lower than, values previously reported for recurrent anterior glenohumeral instability.[11] Hovelius et al[11] reported a 20% rate of arthritis after anterior glenohumeral instability at 10 years of follow-up, and this increased to a rate of 61% at 25 years.[12] Marx et al[13] reported a 10-fold increased risk of developing shoulder arthritis in patients with a history of shoulder dislocation compared with those without it, although the direction of instability was not addressed. The lower rate of arthritis in patients being treated for PSI may be a reflection of disease progression, as these patients may have radiographic findings but no clinical symptoms; thus, they do not seek care and remain undiagnosed, despite disease presence.

Indeed, this finding may reflect a more fundamental difference between PSI and that of anterior instabilities.

Specifically, while recurrent anterior glenohumeral instability is almost always associated with a high-energy traumatic dislocation, the majority of patients seeking treatment for symptoms of PSI have not reported a major traumatic event.[24] Instead, these patients experience repetitive microtrauma and a more gradual decline in shoulder function, both of which would suggest a lesser degree of cartilage damage.[6] This is further supported by Singer et al,[21] who hypothesized that recurrent injury does not have the same deleterious effect as the initial traumatic injury. Continued follow-up in this population may be useful to determine even longer-term effects of PSI.

In this study, 2 factors were predictive of developing symptomatic arthritis. First, age ≥30 years at the time of instability was predictive of progression of symptomatic arthritis. Hovelius and Saeboe[12] reported similar findings, with age >25 years at the time of anterior glenohumeral instability correlated with increased severity of arthrosis. There are changes in the labrum and glenohumeral joint seen on MRI scans in shoulder joints of asymptomatic middle-aged patients.[20] This may reflect a greater degree of preexisting joint pathology in older patients that is subsequently exacerbated by the development of PSI, thus lowering the threshold for degeneration after an instability event.

The second factor predicting the development of symptomatic arthritis was surgical intervention of any type (labral debridement or stabilization). Surgical intervention placed a patient at a 4 times increased risk of having symptomatic arthritis progression (HR, 4.03; 95% CI, 1.23-13.2). One hypothesis is that individuals who choose to undergo surgery represent a self-selected cohort of patients with more severe or advanced instability. However, the number of patients with baseline radiographic arthritis at the time of diagnosis did not differ between the surgical and nonsurgical groups.

In the surgical intervention group, there was a trend toward increased progression of arthritis in the labral debridement group compared with the surgical stabilization group despite no patients having preexisting arthritis in the debridement group and 3 patients having preexisting arthritis in the stabilization group (P = .09). This finding suggests that stabilization affects long-term joint health, but no statistical significance was reached with the number of patients in the study. Although stabilization with ≥3 anchors showed increased arthritis progression compared with stabilization with 1 to 2 anchors, this also did not reach significance). This trend may be attributed to worse baseline instability causing symptomatic arthritis or joint pathology caused by anchor placement or anchor type. With the length of follow-up of this study, there have been changes to surgical technique and equipment that may also factor into the development of arthritis. Further research is needed to determine the influence of surgical technique on the progression of arthritis and to determine whether there is a protective effect provided by posterior glenohumeral stabilization similar to that previously demonstrated in anterior glenohumeral stabilization.[8,9,12] Radiographic parameters including glenoid version and static posterior
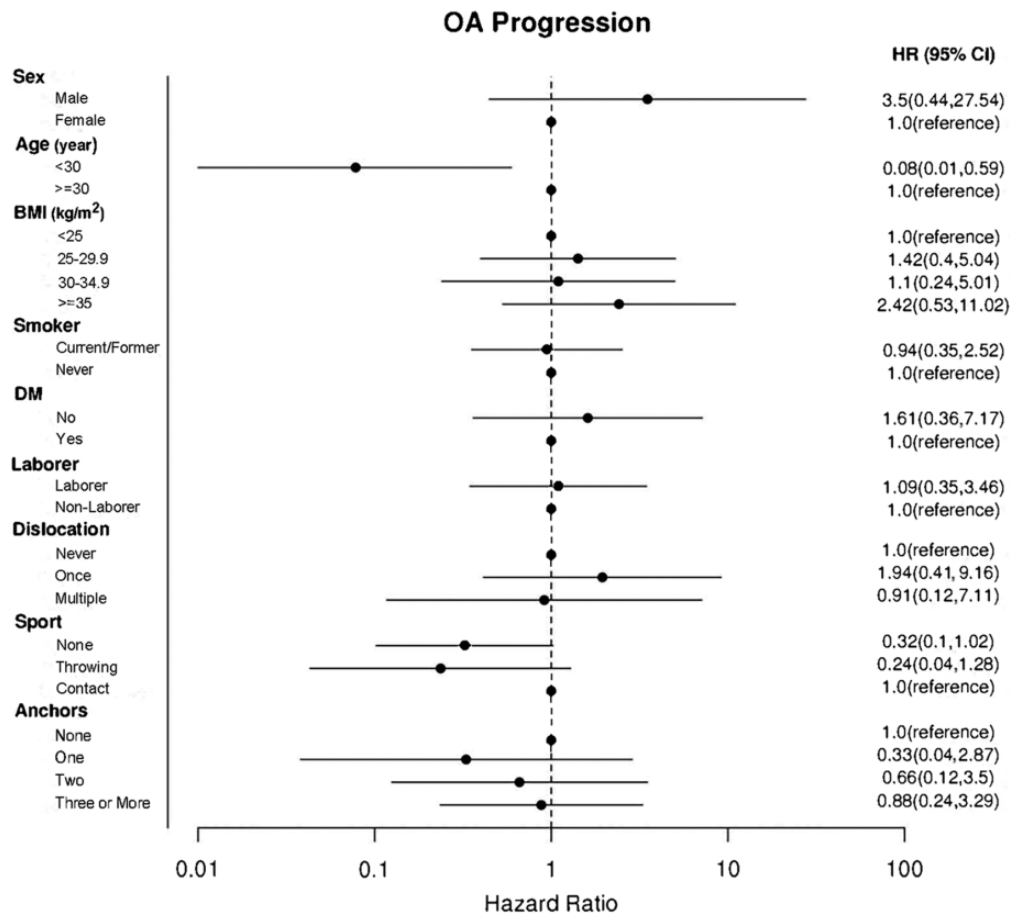
Figure 3. Forest plot demonstrating HRs of risk factors for OA progression. BMI, body mass index; DM, diabetes mellitus; HR, hazard ratio; OA, osteoarthritis.

TABLE 3
Progression of OA Based on Surgical Technique[a]

Surgical Technique	Baseline OA	OA Progression
Debridement (0 anchors)	0/22 (0)	6/22 (27)
Stabilization (1-2 anchors)	2/34 (6)	3/34 (9)
Stabilization (≥3 anchors)	1/22 (5)	4/22 (18)

[a]Data are reported as n/total (%). OA, osteoarthritis.

subluxation may also be factors contributing to arthritis progression.[10,24]

PSI is a rare but increasingly recognized pathology that results in pain, decreased function, and a decline in athletic performance.[4] Despite recognition of an association between PSI and the development of arthritis, the rate at which this occurs has not been well defined. In this population-based study, we defined the rate of progression in symptomatic arthritis and identified risk factors for progression.

Limitations

There are several limitations when interpreting the results of this study. First, this was a retrospective, population-based study that relies on International Classification of Diseases, 9th Revision, codes and chart review to capture an accurate patient population. Our population likely underestimated both the number of patients diagnosed with PSI and those with symptomatic progression of OA, as each of these observations is dependent on a patient seeking medical attention. This is particularly true for patients with x-ray evidence of OA, as a patient may have radiographic findings, but no clinical symptoms; thus, they do not seek care and are unaccounted for. Second, this study spanned nearly 2 decades, and considerable changes in both the rate at which patients underwent surgery and the techniques used (ie, number of anchors used, anchor material) evolved over that period. To determine the influence of surgical stabilization most accurately, we defined complex stabilization as a procedure using a minimum of 3 anchors, while simple stabilizations used 1 to 2 anchors. While this allowed us to identify a cohort of patients undergoing repair using more modern techniques, it may have also self-selected for patients with larger labral tears or surgeon bias. Third, the radiographic findings of arthritis were based on radiologist reports rather than surgeon review. Factors including glenoid morphology including retroversion and/or dysplasia were not evaluated.

This study also has several important strengths. This was a population-based study with medical record review of all patients to confirm accurate diagnosis. Second, all medical visits within a geographically defined community were captured and available for review, including primary care evaluation, surgeon follow-up, urgent care, and emergency treatment. Third, although there was considerable variability in treatments patients received, this study design allowed the unique opportunity to evaluate the rate of symptomatic arthritis progression in nonoperatively treated patients in addition to those treated surgically. Future studies evaluating radiographic parameters including glenoid morphology are needed to evaluate their influence on arthritis progression in patients with PSI.

CONCLUSION

Symptomatic progression of glenohumeral arthritis was observed in 14% of patients diagnosed with PSI at long-term follow-up. Age ≥30 years (HR, 1.20) and surgical intervention (HR, 4.03) were identified as risk factors for developing symptomatic arthritis after PSI.

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