Acromioclavicular Joint Pain in Patients With Adhesive Capsulitis: A Prospective Outcome Study

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Abstract

Diagnosis of adhesive capsulitis is a clinical diagnosis based on history and physical examination. Afflicted patients exhibit active and passive loss of motion in all planes and a positive capsular stretch sign. The effect of adhesive capsulitis on acromioclavicular biomechanics leading to tenderness has not been documented in the literature. This study reports on the incidence of acromioclavicular tenderness in the presence of adhesive capsulitis. Furthermore, we note the natural history of such acromioclavicular joint pain in relation to that of adhesive capsulitis.

Over a 2-year period (2005-2007), 84 patients undergoing initial evaluation for adhesive capsulitis were prospectively examined with the use of validated outcome measures and physical examination. Acromioclavicular joint tenderness results were compared and analyzed on initial evaluation and final follow-up of at least 1 year. Forty-eight patients (57%) with adhesive capsulitis had acromioclavicular joint pain on examination. At final follow-up, as range of motion improved, a significant increase in American Shoulder and Elbow Surgeons/Penn shoulder score and decrease in number of patients with acromioclavicular pain was noted with only 6 patients with residual pain ($P<.05$).

In the presence of adhesive capsulitis, there is not only compensatory scapulothoracic motion but also acromioclavicular motion. This often results in transient symptoms at the acromioclavicular joint, which abate as the frozen shoulder resolves and glenohumeral motion improves. This is important to recognize to avoid unnecessary invasive treatment of the acromioclavicular joint when the patient presents with adhesive capsulitis.
The shoulder, one of the most mobile joints in the human body, moves in a complex 3-dimensional pattern accomplished through coordinated interactions between 3 diarthrodial articulations: the glenohumeral, acromioclavicular, and sternoclavicular and scapulothoracic joints. While adhesive capsulitis (frozen shoulder) is a condition of the shoulder of unknown etiology, it is characterized by pain and restriction of both passive and active range of motion (ROM). The pathologic anatomy was described by Neviaser in 1945, who described findings of inflammation of synovial lining and contraction of the joint capsule.

Regardless of the biologic cause, adhesive capsulitis is characterized by thickening and contracture of the joint capsule, which results in decreased intra-articular volume and capsular compliance so that glenohumeral motion is limited in all planes. The natural history of primary adhesive capsulitis is well described and has been termed benign because it tends to resolve over the course of 1 to 3 years.

Loss of glenohumeral motion will not only profoundly restrict overall upper extremity function but also alter the normal kinematic relationship of the glenohumeral and scapulothoracic joints. A compensatory increase in scapulothoracic motion can create additional symptoms, most commonly described by patients as discomfort medial to the scapula. The true incidence and natural history of acromioclavicular joint tenderness in the presence of adhesive capsulitis has not been previously evaluated. The goal of our study was to address this issue. We hypothesized that patients with frozen shoulder are susceptible to acromioclavicular joint symptoms, as a result of a compensatory increase in acromioclavicular motion as scapulothoracic motion is diminished, and that such symptoms should improve with resolution of the frozen shoulder.

**Materials and Methods**

After institutional review board approval, data were collected prospectively on all patients with idiopathic frozen shoulder who presented from 2005 to 2007. This included 93 new patients (97 shoulders) who were evaluated by a fellowship-trained shoulder surgeon (J.A.A.). Inclusion criteria were as follows: atraumatic onset of shoulder pain that was present for at least 6 weeks, marked loss of active and passive shoulder motion (>50% loss of external rotation), pain at the extremes of all motions, absence of osseous abnormalities on true anteroposterior and axillary lateral radiographs of the glenohumeral joint, and magnetic resonance imaging (MRI) examination of the affected shoulder without evidence of significant rotator cuff pathology (acute or chronic full thickness tear or high grade partial tear). Exclusion criteria included patients treated at any time during the study with manipulation under anesthesia and/or arthroscopic capsular release, intrinsic glenohumeral pathology such as glenohumeral arthritis, a history of substantial shoulder trauma, workers’ compensation patients, previous documented acromioclavicular joint pain or treatment, and previous shoulder surgery.

Ninety-three patients were eligible for our study. Of these patients, 1 had died and 8 could not be located for follow-up. The remaining 84 patients (86 shoulders) were available for examination and formed the basis of the study. Thirty-three men and 51 women had a mean age of 51 years (range, 38-74 years). The mean duration of shoulder pain prior to initial evaluation was 4.6 months (range, 1.3-16 months). Demographics and medical comorbidities are summarized in Tables 1 and 2, respectively.

A validated shoulder outcomes questionnaire was used to determine the shoulder function of the patients (American Shoulder and Elbow Surgeons [ASES]/Penn shoulder score). At the initial evaluation and at each follow-up visit, these scores were attained. Patients were seen and examined initially and then at 3 weeks, 4 months, and 1 year. Magnetic resonance imaging examinations were reviewed and findings recorded on initial or second office visit.

In this study, the physician measured ROM with a goniometer. Passive flexion, abduction, and internal rotation were measured with the patient standing, and external rotation was determined with the patient lying supine. In flexion and abduction, the angle between the arm and body was measured. In internal rotation, the arm was raised behind the back as high as possible. In the evaluation of the range of external rotation, the arm was kept close to the patient’s side with the elbow flexed to 90º, and at the end point of the movement, the angle between the vertical line and forearm line was measured. Although goniometers may be designed and used to assess glenohumeral motion, they are actually measuring both glenohumeral and scapulothoracic motion. Previous studies

### Table 1
Patient Demographics

<table>
<thead>
<tr>
<th>No. patients</th>
<th>84</th>
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<tr>
<td>No. men/women (%)</td>
<td>33 (39)/ 51 (61)</td>
</tr>
<tr>
<td>Average patient age, y (range)</td>
<td>51 (38-74)</td>
</tr>
<tr>
<td>No. dominant extremity (%)</td>
<td>53 (63)</td>
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### Table 2
Medical Comorbidities

<table>
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<tr>
<th>Comorbidity</th>
<th>No.</th>
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<tbody>
<tr>
<td>Diabetes</td>
<td>12</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>8</td>
</tr>
<tr>
<td>Stroke</td>
<td>5</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>16</td>
</tr>
<tr>
<td>Cervical disease</td>
<td>2</td>
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<tr>
<td>Lung disease</td>
<td>3</td>
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have shown that motion of the scapula can have a significant effect on both goniometric and vertebral level measurements. As a result, glenohumeral motion was isolated and recorded.

Patients’ acromioclavicular joints were examined. Each patient who had pain localized to the acromioclavicular joint that increased with direct palpation and pain with passive cross-body adduction with a negative contralateral examination was considered as having a positive clinical acromioclavicular joint examination. Direct palpation of the sternoclavicular joint, anterior leading edge of acromion, and medial scapula was also performed and response recorded. Patients with more diffuse shoulder pain, not localized to the acromioclavicular joint, were not considered to have acromioclavicular pain.

Power calculations carried out before the study to attain a power at least equal to 0.80 at the .05 significance level revealed that a sample of 70 patients were needed. Student t test was used for the statistical testing of continuous variables. An analysis of variance was used in the evaluation of the effect of the baseline information on changes in shoulder symptoms and shoulder mobility and in testing the significance of differences during follow-up.

RESULTS

The mean duration of follow-up was 16 months (range, 12-20 months). On initial examination, 48 shoulders (56%) with adhesive capsulitis were found to have a positive clinical examination for acromioclavicular joint arthritis. In contrast, 38 shoulders had an asymptomatic acromioclavicular joint examination. All patients were treated with a home-based standard stretching program, and in phase 1 of the disease process, 32 shoulders (38%) underwent therapeutic intraarticular glenohumeral injection of lidocaine and depomedrol; all injections were administered without ultrasound or fluoroscopic guidance through the posterior portal location, approximately 1 inch inferior and medial to the posterolateral corner of acromion and into the “soft spot.” There was no relationship between these injections and resolution of acromioclavicular joint pain. In addition, we did not note a difference in shoulder ROM between the patients who had injections and those who did not. None of the patients received acromioclavicular joint injections as part of the workup or treatment of the acromioclavicular pain.

The difference in ROM between the affected shoulders and the contralateral shoulders at initial and final evaluations and associated ASES/Penn scores are summarized in Tables 3 and 4, respectively. There was a significant increase in ROM in all planes at the final evaluation (P < .001) for patients with and without associated acromioclavicular joint pain (Figure). While the patients with acromioclavicular joint pain, on average, had less ROM in all planes compared to the group without acromioclavicular joint pain, this difference was not statistically significant. On final follow-up, along with improved ROM and ASES/Penn scores, a significant number of acromioclavicular symptomatic patients reported resolution of symptoms (P = .005), with only 6 patients reporting residual acromioclavicular joint symptomatology. At final follow-up, these 6 patients demonstrated significantly improved ROM. Range of motion and asso-
associated number of patients with acromioclavicular joint pain are summarized in the Figure.

DISCUSSION

In this study, we prospectively evaluated the presence of associated acromioclavicular joint symptomatology on initial examination and at least 1 year follow-up of patients with diagnosed adhesive capsulitis. Previously, no study had looked at the presence of associated acromioclavicular joint symptomatology on history, clinical examination, or imaging studies in patients with adhesive capsulitis. We found that while 56% of patients with clinically diagnosed adhesive capsulitis had objective findings of acromioclavicular arthritis on physical examination, the majority of patients improved with no intervention directed specifically at the acromioclavicular joint such as injections or more invasive procedures such as arthroscopic surgery. Of all symptomatic patients, 13% (6/48) continued to have acromioclavicular joint pain at final follow-up. While this group of patients might have benefited from specific acromioclavicular joint intervention, they declined treatment secondary to the marked subjective overall improvement in symptoms.

This study had several limitations. We used the crossover test and palpation examination to establish the clinical diagnosis of acromioclavicular joint pain. Several studies have confirmed the crossover test to provide significant enough joint stress to cause pain in patients with acromioclavicular pathology. None of the patients received diagnostic acromioclavicular injections to fully identify the pathology in the respective joint. This was not presented as a therapeutic choice, as we were optimistic that with improved glenohumeral ROM, the acromioclavicular joint pain would dissipate. Also, while commonly used, there are some limitations when using a goniometer to measure ROM. Some limitations include the fact that end range is determined by clinical feel as opposed to an objective assessment of torque. The specific cause of acromioclavicular pain in the setting of adhesive capsulitis still needs to be fully elucidated via biomechanical testing. We have theorized that in the presence of significantly reduced glenohumeral ROM, in addition to compensatory scapulothoracic motion, there is the potential for stress transfer at the acromioclavicular joint that may occur in isolation or in relation to excessive and repetitive scapular protraction and can manifest as pain and tenderness. Additionally, some patients might be more prone to develop these symptoms as a result of more severe underlying acromioclavicular joint abnormalities (ie, grade III and IV osteoarthritis).

CONCLUSION

Based on the results of this study, we discourage clinicians from aggressive interventions for acromioclavicular joint pain in the presence of adhesive capsulitis. This subset of patients should undergo treatment focused on adhesive capsulitis prior to embarking on any invasive treatment focused on the acromioclavicular joint as there is a high probability of symptom resolution with the improvement of glenohumeral joint motion. Patients who continue to have acromioclavicular joint pain after resolution of adhesive capsulitis may be candidates for formal acromioclavicular joint treatment. To our knowledge, this is the first prospective study of idiopathic adhesive capsulitis to provide detailed information about the association of clinically positive acromioclavicular joint symptoms in patients with adhesive capsulitis. Further biomechanical studies will be needed to further understand the cause of acromioclavicular joint pain in this patient population.

REFERENCES


