

ADHESIVE CAPSULITIS OF THE SHOULDER: A COMPREHENSIVE STUDY

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BACKGROUND

For well over a century, adhesive capsulitis of the shoulder has remained a complex disorder with variable clinical outcomes. In 1872, Duplay¹ described a condition he called peri-arthritis scapulo-humeral, and later Codman² described this disorder as a frozen shoulder and stated adhesive capsulitis of the shoulder remains “a class of cases which are difficult to define, difficult to treat, and difficult to explain from the point of pathology.” This remains true even today. It has been suggested that the frozen shoulder is precipitated by degeneration of the supraspinatus tendon and is associated with chronic inflammation of the subacromial bursa and glenohumeral capsule. Furthermore, immunological pathogenesis and chromosomal abnormalities of this condition have also been postulated. Despite several theories and hypotheses, the pathogenesis and pathophysiology of this disorder continues to be poorly understood.

Adhesive capsulitis of the shoulder is clinically described as having three phases: the freezing or painful stage, the frozen or progressive stiffness phase, and the thawing or resolution phase. Codman² stated that even the most protracted cases recover with or without treatment in approximately two years. However, other authors³ have noted that this disorder may not be self-limited with long-term follow up revealing persistent loss of motion in many patients.

In 1995, we set out upon a comprehensive study to determine the prevalence of this disorder as well as the natural history of adhesive capsulitis of the shoulder. Furthermore, we evaluated the surgical outcome of those patients undergoing arthroscopic capsular release for refractory adhesive capsulitis

of the shoulder. We are now in the early stages of evaluating the cellular biology of this orthopaedic unknown.

PREVALENCE OF ADHESIVE CAPSULITIS OF THE SHOULDER

Three hundred consecutive patients were studied prospectively at the Diabetes Center and primary care practices at the Massachusetts General Hospital. Patients completed a questionnaire and were examined by the same investigator in order to minimize interobserver variability. Strict criteria were used to determine the presence of a frozen shoulder.

The overall prevalence of adhesive capsulitis in the diabetic population and nondiabetic population was 12% and <1%, respectively. Separating the diabetic patients into type I and type II diabetics, prevalence was noted to be 16% and 7%, respectively. An increased prevalence of adhesive capsulitis was noted in female patients as well as those who had a longer duration of diabetes mellitus. However, after correction for duration of diabetes, no causation was noted between frozen shoulder and hemoglobin (Hb) A1c values. This was true despite not only the last known HbA1c value, but over the average HbA1c value when followed for a mean of four years. These findings support the hypothesis that adhesive capsulitis of the shoulder is associated with duration of diabetes but not associated with long-term glycemic control. There are several theories as to why diabetic patients are more prone to a frozen shoulder. We believe diabetes may affect fibroblastic proliferation and increased expression of cytokines, thereby making this disorder more likely in this select population.

NATURAL HISTORY

The natural history of adhesive capsulitis remains variable. After reviewing 50 patients over a 10 year period, Rockwood and his colleagues⁴ found “without exception, every patient regained a significant amount of motion and returned to activities of daily living without pain.” In contrast, Shaffer et al,³ evaluated 62 patients and found that 30% of patients had measurable restriction motion at an average of seven years follow up. This diversity in the literature led us to embark on a prospective evaluation of the natural history of this disorder in the general population. We have prospectively followed well over 150 patients referred to our offices over the past five years. Preliminary evaluation of these patients reveal 78% of patients were female and the dominant arm was affected in 52% of patients. Complete resolution of pain and return to full motion has been noted in the vast majority of patients. However, diabetic patients have been reported to have poorer

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outcomes when compared to the nondiabetic population. Our findings have confirmed this as nearly 90% of nondiabetics were noted to have complete resolution of this disorder compared to only 15% of diabetic patients. Duration of symptoms were seen to typically last 12 to 18 months prior to resolution of symptoms. Based on our preliminary findings, we have proposed a treatment plan (Figure 1). However, our study remains ongoing. We plan on completing this study when 200 patients have been enrolled with two-year minimal follow-up.

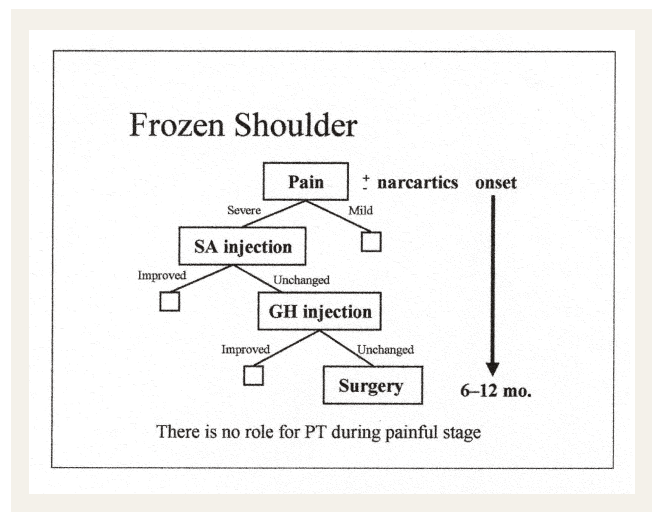


Figure 1. Treatment protocol for adhesive capsulitis of the shoulder.

SURGICAL OUTCOMES

Over the past 5 years, we have been evaluating outcomes following arthroscopic capsular release in the refractory frozen shoulder. Fifty-two patients with idiopathic adhesive capsulitis of the shoulder have undergone arthroscopic capsular release at the Massachusetts General Hospital. Patients have been studied using the Constant and Murley⁵ score system as well as the American Shoulder and Elbow Society Standardized Shoulder Assessment and Shoulder Score Index.⁶

OPERATIVE TECHNIQUE

After administration of anesthesia, gentle manipulation is performed by forwardly flexing the arm slowly with gradually increasing force. Care is taken to support the humerus just distal to the axilla. The manipulation is often associated with an audible and palpable sensation of tissue rupture. This process is then repeated with lateral elevation; however, internal and external rotation often remains limited. Attempts of achieving increased rotation may be performed, however, it is difficult to control the humerus during this manipulation, and attempting to achieve such increased rotation may be complicated by a fracture of the humerus.

An anterior and posterior portal is established in the usual fashion. An arthroscopic capsular release is then performed utilizing an electrocautery device. It is imperative that systemic muscle relaxants are not used by the anesthesiologist in order to be certain that if the release approximates the axillary nerve, the surgeon will see abrupt contraction of the deltoid

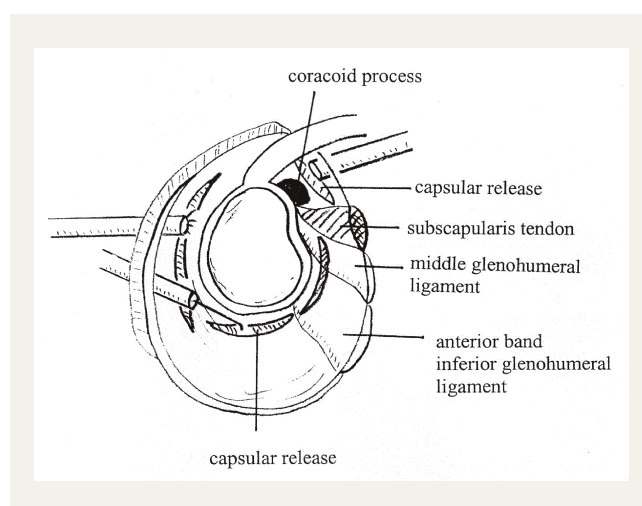


Figure 2-A. Arthroscopic capsular release diagram. Note dual posterior portals for access to axillary recess for inferior capsular release.



Figure 2-B. Arthroscopic photograph in lateral decubitus position. Note subscapularis muscle belly visible following anterior capsular release.

muscle. The release begins at the middle glenohumeral ligament and continues inferiorly until external rotation with the arm at the side is noted to improve. Typically, the inferior glenohumeral ligament complex has been disrupted during the flexion component of the manipulation.

If internal rotation remains limited, the arthroscope is then transferred to the anterior portal and utilizing the posterior portal as a working portal, a posterior capsular release is performed. Finally, if flexion remains limited, an inferior capsular release is performed through a second posterior portal, which is established one centimeter inferior to the initial portal (Figure 2). This gives excellent access to the axillary recess, although care must be taken to avoid injury to the axillary nerve during this portion of the release.

After release of the anterior and posterior structures, attention is then turned to the rotator interval. While visualizing the interval posteriorly, a rotator interval release is performed releasing all capsule in-between the subscapularis and the biceps tendon. Release extends superiorly to the anterior margin of the supraspinatus tendon. The coracoid process can be palpated just medial to the anterior arthroscopic portal. The coracohumeral ligament is divided by first dividing the rotator interval and then gently dividing all tissue lateral to the coracoid process as the electrocautery device is slowly brought from inside the joint towards the deltoid. Often the coracohumeral ligament is not identified as a discreet structure, however, dividing all tissues lateral to the coracoid process will release this ligament at its coracoid origin. Finally, if external rotation remains limited, one may consider releas-

ing the intra-articular portion of the subscapularis tendon; however, caution should be taken to avoid excessive release of the subscapularis.

RESULTS

Pre-operative and post-operative results were compared using the paired *t*-test. A regression analysis was used to evaluate any correlation between length of follow-up and change in outcome. Preliminary findings with minimal follow up at one year has revealed statistically significant improvement in range of motion, pain, and objective shoulder scores following arthroscopic capsular release.⁷ We paid particular attention to the outcomes following the use of an indwelling interscalene catheter. It has several theoretical advantages. It decreases pain in the postoperative period thereby improving compliance with physical therapy. Our findings have supported the use of an indwelling interscalene catheter. We have found a significant improvement in the objective shoulder scores system in those patients who underwent release following placement of an interscalene catheter. We now are at the beginning stages of a prospective randomized study evaluating the use of this catheter.

CELLULAR BIOLOGY

The cellular biology and pathogenesis of adhesive capsulitis remains poorly understood. Rodeo and colleagues⁸ reported increased receptors for certain growth factors in those patients with adhesive capsulitis. Similar findings have been noted by Bunker and Anthony⁹ This prompted us to begin a study of the cellular biology of adhesive capsulitis. Monoclonal antibodies will be used to localize cytokines in the shoulder capsule of normal and frozen shoulder patients. We plan on studying the presence and concentration of platelet-derived growth factor (PDGF), basic fibroblast growth factor (bFGf), insulin-like growth factor (IGF), as well as leucocyte common antigen, and macrophage/synovial antigen in the shoulder capsules of these patients.

SUMMARY

We hope our research efforts will shed some light on this interesting and often disabling disorder. Understanding the cellular biology of adhesive capsulitis may have important treatment implications in the future.

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