Dislocation and Instability After Arthroscopic Capsular Release for Refractory Frozen Shoulder

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Frozen shoulder is a condition of unclear etiology characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder.[1] Incidence of frozen shoulder is higher in people with diabetes (up to 36% in patients with insulin-dependent diabetes) than in the general population.[2-4] As a group, people with diabetes tend to be younger and to have a more painful course of frozen shoulder versus people without diabetes.[5] Still, even for people with diabetes, the natural history of this disorder is usually benign, resolving over 1 to 3 years.[6]

Operative treatment for the frozen shoulder is reserved for patients who do not improve with conservative management and are not in the inflammatory phase of this disease. Arthroscopic release provides a controlled and systematic release of needed structures under direct visualization, without the morbidity of an open approach or having to repair the subscapularis tendon. Despite obvious concerns about releasing the principal static stabilizers, instability and dislocation have not been reported as complications.

We report the case of a patient with diabetes and frozen shoulder who had failed conservative therapy, closed manipulation, and 2 arthroscopic releases and subsequently developed frank shoulder dislocation. This patient later developed symptoms of shoulder instability. Dislocation and instability after arthroscopic capsular release of a frozen shoulder have not been reported in the English-language literature.

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Am J Orthop. 2007;36(12):672-674.

Case Report

A 30-year-old right-hand–dominant woman with a history of insulin-dependent diabetes was referred for evaluation of left shoulder pain. She complained of 2 years of worsening pain and stiffness despite use of nonsteroidal anti-inflammatory drugs (NSAIDs), physical therapy, and multiple corticosteroid injections.

At the time of initial examination, the patient's shoulders were symmetric in appearance, without atrophy. There was severe loss of both passive and active motion. Motion in all planes elicited pain. Motor, sensory, deep tendon reflex, and vascular examinations were otherwise unremarkable.

X-rays did not show any evidence of fracture, dislocation, or arthrosis, and magnetic resonance imaging was normal. A bone scan demonstrated some increased nonspecific uptake in the proximal humerus.

Given the persistence of symptoms after almost 2 years of conservative treatment, the patient elected to proceed with arthroscopic capsular release in an attempt to expedite recovery.

After interscalene block was administered, examination conducted under anesthesia revealed external rotation of 0° with the arm to the side and 30° of abduction. With the arm maximally abducted, there was a 10° arc of motion. Internal rotation was only to the greater trochanter. Closed manipulation was gently performed, and some adhesions were released, restoring approximately 60% of motion.

Given the patient's persistent stiffness, arthroscopic capsular release was then performed.[7] Diagnostic arthroscopy revealed a diffusely inflamed capsule both anteriorly and posteriorly with extensive synovitis and a thickened rotator interval. The rotator cuff was intact. Articular surfaces and biceps tendon were normal. The glenohumeral joint was débrided, and the anterior capsule was released from the rotator interval down to the 7-o'clock position. The posterior capsule was subsequently released from the 12-o'clock position to the 5-o'clock position. The remaining inferior capsule was released with gentle manipulation.

After capsular release, evaluation of range of motion (ROM) revealed 120° of abduction and 170° of forward elevation with the arm in neutral. With the arm abducted 90°, there were 90° of external rotation and 60° of internal rotation. The patient was admitted for 48 hours for aggressive pain control and physical therapy, including continuous passive motion, and was then discharged with daily aggressive physical therapy, including full passive and active-assisted ROM.

Two weeks after surgery, shoulder ROM was significantly decreased from that obtained at surgery, and the patient complained of pain. There were 80° of forward flexion, 70° of abduction, and 10° of external rotation. The patient was encouraged to resume her NSAIDs, to continue physical therapy, and to start wearing a static progressive stretching brace (JAS Shoulder Brace; Joint Active Systems, Inc, Effingham, IL).

Two months after surgery, the patient had a marked decrease in shoulder motion and reported persistent pain requiring narcotic medications for relief. On examination, she had restricted passive and active motion with external rotation to 10° in neutral abduction and only 20° of lateral abduction. She was given an intra-articular injection of corticosteroid with local anesthetic in an attempt to alleviate her pain and decrease capsular inflammation. A pain service consult was obtained to help her manage her pain medication regimen. There were no signs of infection.

Five months after capsular release, the patient continued to complain of pain and stiffness, and shoulder motion showed no significant improvement. Again, there was no sign of infection. Options for managing recalcitrant adhesive capsulitis were reviewed with the patient, and she elected to proceed with repeat arthroscopic capsular release.

At time of repeat surgery, diagnostic arthroscopy demonstrated moderate adhesions in the glenohumeral joint with labral fraying and extensive synovitis. Débridement was performed to remove adhesions and synovium. The rotator interval was released again, followed by release of the anterior capsule to the 6:30 position. The arm was placed in internal rotation, and the posterior capsule was released, dissecting the capsule from the overlying posterior rotator cuff. Examination under anesthesia demonstrated 170° of forward flexion and 140° of abduction. With the arm in 90° of abduction, there were 90° of external rotation and 90° of internal rotation. The patient received aggressive physical therapy while remaining in the hospital for 5 days. She was discharged on a supervised physical therapy program that included use of a continuous passive ROM machine.

Six weeks after repeat capsular release, the patient presented urgently complaining of pain and loss of motion. She had had a low-energy fall 10 days earlier as a precipitating event. She stated that she had slipped while walking down 2 steps and landed on her left elbow. She had experienced an immediate decrease in ROM but had not sought medical attention.

On examination, there was obvious deformity of the left shoulder, and the humeral head was palpable anteriorly through the anterior deltoid. There was no evidence of edema, erythema, or ecchymosis. Active ROM was 75° of forward flexion in abduction, 30° of internal rotation, and 0° of external rotation with the arm in neutral. The left upper extremity was otherwise neurovascularly intact.

X-rays showed an anteroinferior dislocation of the left humeral head with a Hill Sachs deformity. Closed reduction under intravenous sedation was unsuccessful. The patient then underwent general anesthesia, and the left shoulder was reduced easily under fluoroscopic guidance to ensure that the humeral head was safely unlocked from the glenoid during the reduction maneuver. Under fluoroscopy and complete muscle paralysis, the shoulder began to dislocate with abduction of 90° and external rotation beyond 30°. There was no bony crepitus or cracking noted, and no fracture was appreciated. The patient was placed in a sling immobilizer with the shoulder in neutral rotation for 1 week before restarting physical therapy. She was allowed to gradually progress her motion over the next several weeks.

The patient had no further shoulder dislocations over the next year but did have symptoms of instability for 6 to 8 months. She gradually improved active and passive ROM to 120° of forward flexion, 30° of external rotation, and 90° of abduction.

Discussion

Dislocation after arthroscopic capsular release has not been reported in the literature. In this case report, we report this complication and the details of how it was managed.

Treatment of patients with frozen shoulder is often difficult and not without complications. Even patients who have successful resolution of the stiffness with physical therapy and time may have some residual loss of motion or pain.[8] According to studies on the natural history of this condition, 10% or more of patients fail conservative therapy.[5,9-11] This subset of patients faces the options of closed manipulation, arthroscopic release, and even open capsular release if they decide to pursue further treatment.[12-17]

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Reported complications for closed manipulation of frozen shoulders include humeral fracture, nerve injury, and dislocation.[18-23] Dislocation after manipulation for frozen shoulder is rare, and a case has not been reported in the English-language literature since a 1982 report of 2 cases after 612 closed manipulations.[18]

For surgical release, complications include nerve damage, infection, and incomplete release.[12,15] Although the English-language literature documents 1 case of subjective instability on a self-reported patient questionnaire,[22] our report is the first of frank anterior dislocation after arthroscopic capsular releases for frozen shoulder.

An advantage of arthroscopic release is that it is more precise—it cleaves the capsule at the appropriate interface between the labrum and the capsule. Manipulation may cause the labrum or capsule to tear in nonanatomic tissue planes.

It is difficult to establish the precise cause of dislocation in our patient's case, but several variables—poor tolerance of pain, failed surgery necessitating second release, traumatic fall after surgery—are all potential contributing factors. However, it seems unlikely that the low-impact trauma the patient experienced when she slipped on 2 steps and braced herself with the treated arm would have caused a frank dislocation. Obviously, releasing the capsule destabilizes the shoulder, as the biomechanical roles of the capsule and glenohumeral ligaments are well defined.[24] Despite the dislocation, conservative nonoperative treatment was effective in managing the patient's subsequent symptoms of instability; within 6 to 8 months, these symptoms resolved. Given this experience, we advocate 6 to 12 months of conservative treatment for instability after capsular release before considering surgical intervention. Clearly, the idea that it is more difficult to treat frozen shoulders in patients with diabetes is supported in this case.

Arthroscopic release does not always result in complete resolution of symptoms, and significant complications may be associated with this type of management. In the case of our patient with diabetes, the natural history of frozen shoulder appeared to be unaltered by the wide array of treatment modalities, and subjective resolution of symptoms was seen only 3 years after onset of symptoms. This may simply reflect the natural history of the disease. The best treatment modality for the recalcitrant frozen shoulder in patients with diabetes is yet to be determined. It is a challenge suited for a prospective study comparing established modalities, such as manipulation and surgical release, with conservative treatment or "natural history." We hope that this case report will help other surgeons to be aware of the possibility of this complication after an arthroscopic capsular release.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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