Shoulder Dislocation After Mobilization Procedures for Adhesive Capsulitis

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Loss of shoulder motion may occur as a result of primary or secondary capsulitis.[1] Primary adhesive capsulitis (idiopathic frozen shoulder) generally occurs in women between 40 and 50 years. These patients demonstrate a loss of active and passive range of motion (ROM), usually with ≤90° of forward flexion and decreased external rotation when compared to the contralateral side. Secondary capsulitis may occur following surgery or trauma and may have varying degrees of motion loss. This may occur after rotator cuff repair, fracture fixation, or instability. These patients tend to be refractory to conservative management, and the timing of intervention depends on the degree of loss and the lack of progress. Generally, after 6 months of conservative treatment if there is minimal improvement, the motion is essentially fixed and more aggressive interventions are needed to regain mobility.

Although the course of this disease has been elucidated by the work of Hannafin et al,[2,3] its natural history is still not completely defined. Miller et al[4] reported on 50 patients with adhesive capsulitis treated conservatively, all of whom regained a significant amount of motion and returned to activities of daily living without pain over a 10-year period. Shaffer et al[5] reported on 62 patients (68 shoulders) treated nonoperatively for idiopathic frozen shoulder with an average

7-year follow-up; 31 (50%) patients still had either mild pain or stiffness of the shoulder or both. However, only 7 patients (11%) reported functional limitation.

Conservative measures are reported to have failed in 0% to 41% of cases.[2, 4-11] However, in cases when these measures fail, more aggressive interventions may be appropriate to restore ROM and reduce pain. A closed manipulation under general anesthesia typically achieves and maintains a sufficient improvement in motion. Hamdan and Al-Essa[12] reported on 100 patients who were treated with manipulation with or without injection of steroid after a failure of conservative treatment; 66 of 86 shoulders (77%) had fair to good results. Placzek et al[13] reported on 31 patients who underwent manipulation for adhesive capsulitis and had good long-term results with respect to mobility at final follow-up. However, uniformly good results have not been reported. In a study by Holloway et al,[7] 81 of 135 patients (60%) who underwent manipulation under anesthesia for adhesive capsulitis underwent subsequent surgical release due to a failure of long-term improvement in ROM.

The current surgical management of refractory adhesive capsulitis includes both arthroscopic and open techniques, with 84% to 100% good to excellent results.[7,9,11,14-16] Although it was initially thought that open surgery was the optimal means to address

this problem, most patients today will have an arthroscopy.[11,16] Our preferred method is to perform an arthroscopic release for fixed contractures followed by manipulation. This controlled release is less likely to result in humeral fracture or cuff rupture. However, little if any has been published about the potential complications of surgery.

Holloway et al[7] reported that complications included persistent stiffness, recurrent pain, and postoperative biceps tendonitis. To our knowledge, dislocations of shoulders treated operatively for refractory capsulitis have not been reported. The purpose of this study is to report a small series of patients who had postoperative dislocations after arthroscopic release with manipulation procedures for their capsulitis. In each case there was a delay in the diagnosis potentially due to low suspicion, which ultimately affected the clinical outcome.

CASE REPORTS
Patient 1

A 54-year-old, right-hand-dominant woman was initially evaluated for left shoulder pain

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Financial Disclosure.

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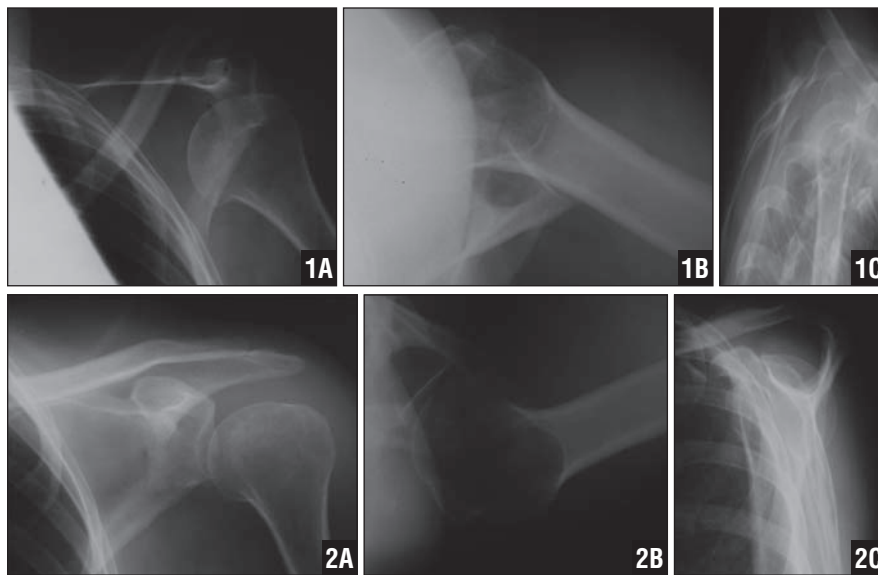
Figure 1: Postoperative AP (A), lateral (B), and axillary (C) radiographs of patient 1's shoulder. **Figure 2:** AP (A), lateral (B), and axillary (C) radiographs of patient 1's shoulder postreduction.

and stiffness. The patient reported an atraumatic onset of shoulder discomfort and progressive limitation of motion for approximately 10 months. The patient had been seen by an outside physician who injected her with cortisone 4 times, which gave her only temporary relief. She also had a short course of physical therapy. She had no significant past medical or surgical history.

Physical exam revealed a tender acromioclavicular joint, active forward flexion to 140°, external rotation to 20°, and internal rotation to L2 on the affected side. The patient had normal rotator cuff strength. Magnetic resonance imaging (MRI) of the left shoulder revealed pericapsular inflammation with no evidence of rotator cuff pathology. A diagnosis of adhesive capsulitis was made, and the patient underwent a course of aggressive physical therapy and anti-inflammatories.

Despite this the patient's symptoms worsened, with weakness and decreased ROM. The patient was indicated for examination under anesthesia, manipulation under anesthesia, and arthroscopic release of adhesions. Examination under anesthesia revealed forward flexion to 60° and external rotation to −20°, compared with full ROM of the contralateral extremity (forward flexion to 180°, external rotation to 60°). After manipulation under anesthesia, arthroscopy was performed and a marked synovitis was visualized and debrided. The anterior capsule was released in the region of

the rotator interval extending inferiorly. A formal posterior release was not performed. In addition, the patient did have some thickening of the subacromial bursa, and a subacromial decompression and acromioplasty was performed. After manipulation under anesthesia and release, the patient had forward flexion to 170° and external rotation to 90°.

The patient was then placed in sling. She was given an intravenous patient-controlled anesthesia machine. Physical therapy was begun immediately postoperatively, with shoulder continuous passive motion and passive ROM to progress to active assisted ROM. The patient was placed in an overhead position to minimize postoperative contracture.

On postoperative day 1, the patient was observed to have decreased sensation in the axillary, median, and ulnar nerve distribution and decreased motor function in her left upper extremity. This was thought to be due to a long-acting regional interscalene block, which had been performed 12 hours prior and could last 18 hours. However, when the patient's condition did not improve, radiographs showed that she had an anterior shoulder dislocation (Figure 1). While she had not experienced a traumatic event, her arm was in the overhead position and may have dislocated during manipulation of her bed during routine nursing care.

The patient underwent emergent closed reduction under anesthesia with no complications (Figure 2). The patient was unstable when externally rotated to 45°. She was therefore placed in a sling and swathe. However, her neurological symptoms were slow to resolve. She was evaluated by a neurologist and diagnosed with a brachial plexopathy. A subsequent MRI of the brachial plexus was unremarkable for injury or impingement of the plexus. Due to the low-energy nature of the injury, her neurologic symptoms were felt likely to improve.

Upon discharge, the patient had dysesthesias in her left upper extremity in the axillary, median, and ulnar nerve distribution. She had 3/5 deltoid strength with 0/5 triceps/biceps and grip strength. She had preservation of some neurologic function and was treated with a combination of splinting and physical therapy. At the time of most recent follow-up, 6 months postoperatively, the patient had no further dislocations. Her passive ROM was as follows: forward flexion to 145°, abduction to 113°, and external rotation to 45°, with forward flexion strength 4/5, abduction 3/5, and biceps 3/5. Sensory and motor functions have continued to improve.

Patient 2

A 52-year-old, right-hand-dominant man presented with a stiff, painful right shoulder. He had a history of a right rotator cuff tear 1 year prior to presentation and had undergone a right rotator cuff repair at an outside institution that had been complicated by a stiff shoulder. Subsequently, he had a manipulation and capsular release, which improved his ROM minimally. In addition, the procedure was complicated by a deep vein thrombosis. The patient had no other contributory past surgical or medical history.

Physical examination revealed that the patient had supraspinatus and infraspinatus atrophy. He was unable to actively elevate his arm. His active ROM was as follows: forward flexion to 45°, external rotation to 0°, abduction to 30°, and internal rotation T12. A motor examination revealed that his forward flexion, external rotation, and elbow flexion and extension were all 4/5. He had some dysesthesias on the lateral aspect of his forearm and an absent biceps reflex. An electromyogram demonstrated an upper trunk
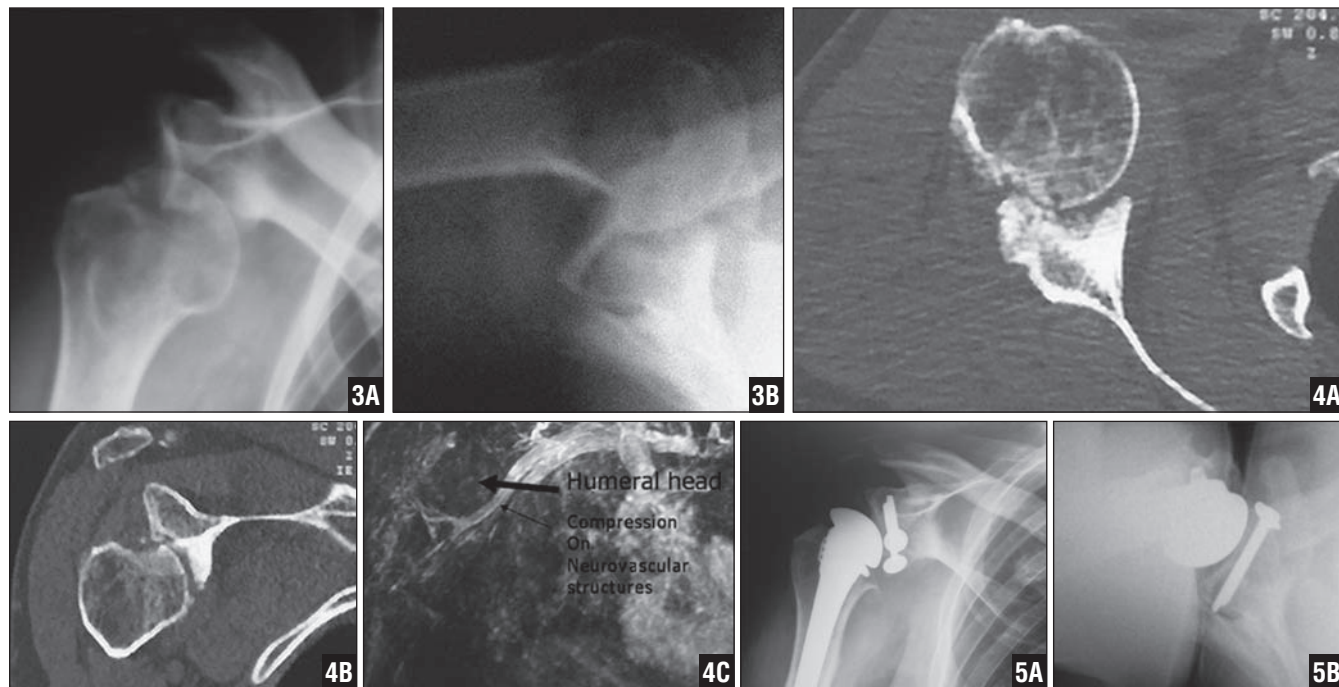
Figure 3: Preoperative AP (A) and axillary (B) radiographs of patient 2's shoulder. **Figure 4:** Preoperative coronal (A) and axial (B) CT images and MRI (C) demonstrating loss of glenoid bone stock and compression of the neurovasculature of the axilla in patient 2. **Figure 5:** Postoperative AP (A) and axillary (B) radiographs of hemiarthroplasty with bone grafting of the glenoid in patient 2.

brachial plexopathy with abnormal increased polyphasic potential in the deltoid, rotator cuff, and biceps. Radiographic evaluation revealed a chronic anterior dislocation of the shoulder with erosion of the glenoid (Figure 3). The patient also had a chronic Hill-Sachs lesion and some heterotopic bone formation about the shoulder. Magnetic resonance imaging and computed tomography (CT) demonstrated a 60% defect of the glenoid and rotator cuff tendinosis without a discrete tear. In addition, a mild compression of the axillary nerve from the dislocation was noted (Figure 4).

Intraoperative evaluation confirmed a massive defect of the glenoid precluding glenoid replacement. The humeral head had grade IV arthritic changes with large osteophytes at the inferior margin. There was some supraspinatus tendon present. A hemiarthroplasty was performed with grafting of the glenoid defect using a section of the humeral head with an interpositional Achilles tendon allograft placed onto the glenoid (Figure 5).

Postoperatively the patient was placed in a sling and started on a shoulder rehabilitation protocol with Codman's and pendulum exercises only. At most recent follow-up, the patient could forward flex to 80° without pain. In this case the diagnosis had been delayed for several months, which resulted in progressive glenoid erosion as therapy continued. The glenoid bone loss necessitated a bone grafting procedure, and ultimately a glenoid replacement may be considered if pain develops.

Patient 3

A 59-year-old, right-hand-dominant woman presented with a 4-day history of left shoulder pain after tripping and falling on an outstretched left upper extremity. She developed pain and discomfort without any neurologic findings. Physical examination revealed tenderness to palpation over the greater tuberosity and markedly decreased ROM and strength secondary to pain. She was neurologically intact distally. Radiographic evaluation revealed a nondisplaced fracture of the greater tuberosity.

The patient was placed in a sling and started on Codman's and pendulum exercises. She progressed to more active exercises 6 weeks postinjury. Despite these measures, the patient developed stiffness. She had a subacromial injection, which did not alleviate her symptoms. Her ROM was as follows: forward flexion 70°, external rotation 20°, and internal rotation L5. After conservative measures failed, she became a candidate for arthroscopic debridement and release of adhesions.

Examination under anesthesia revealed abduction to 70°, forward flexion to 80°, internal rotation to 25°, and external rotation to 15° and 0° at 90° of abduction. Arthroscopy demonstrated a significant amount of synovitis and some capsular adhesions anteriorly and posteriorly. A capsular release was performed from the 9:30 position to the 6:30 position. There were also a large number of subacromial adhesions, necessitating a subacromial decompression. The rotator cuff was intact. Postoperatively the patient was placed in an overhead position to maximize the gains obtained after release.

Approximately 3 weeks postoperatively, the patient presented in severe pain with a significantly decreased ROM. Radiographs revealed an anterior dislocation (Figure 6). The dislocation was fixed and could not be reduced by closed means. The patient underwent an open reduction and a capsular shift procedure. A deltopectoral approach was used. The conjoint tendon fibers were excised to gain access to the
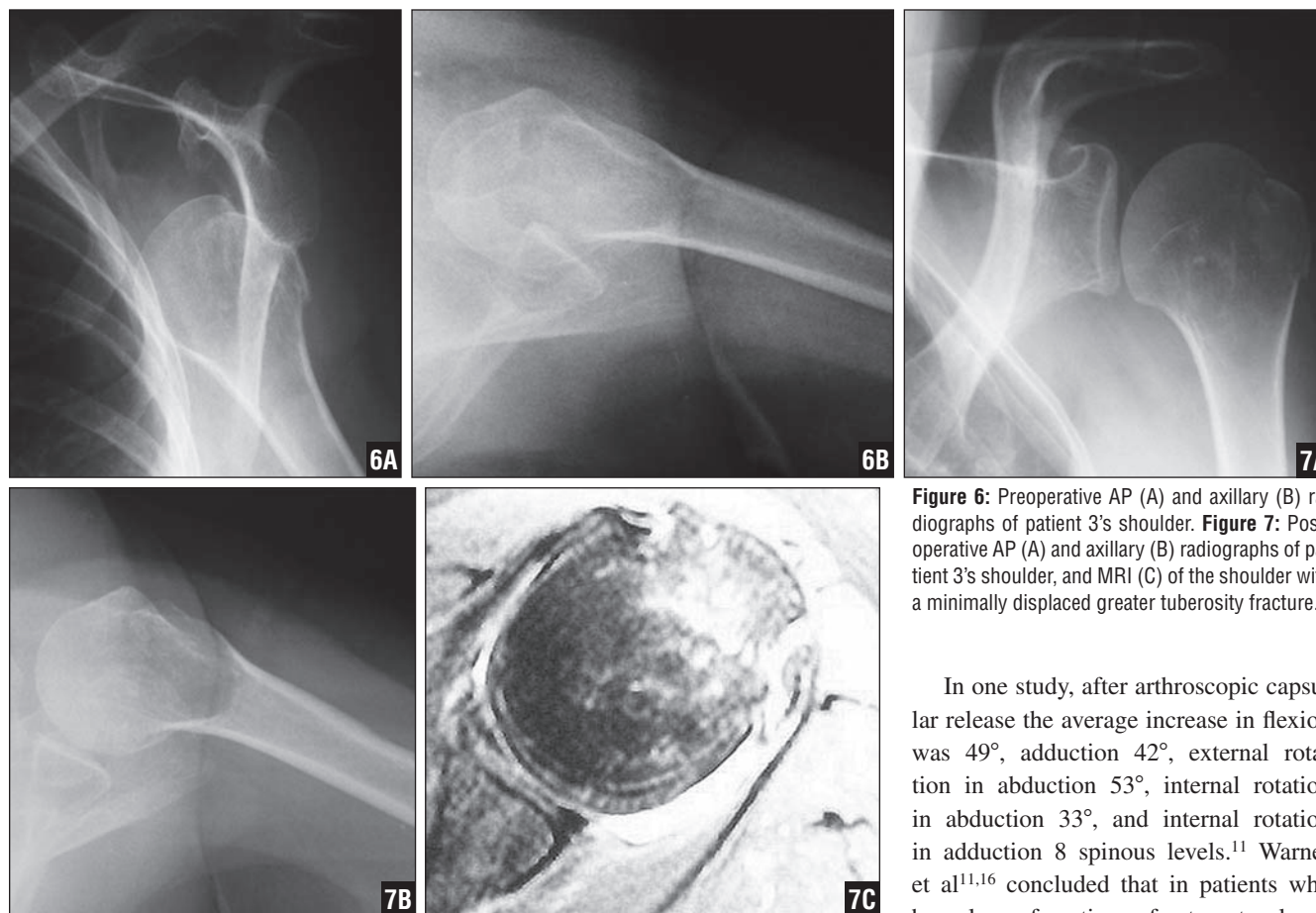
Figure 6: Preoperative AP (A) and axillary (B) radiographs of patient 3's shoulder. **Figure 7:** Postoperative AP (A) and axillary (B) radiographs of patient 3's shoulder, and MRI (C) of the shoulder with a minimally displaced greater tuberosity fracture.

humeral head. Tagging sutures were placed in the subscapularis, and the tendon was incised at its insertion. During inspection, a defect in the capsule was appreciated inferior to the subscapularis. Furthermore, some of the anteroinferior capsule had been torn off the glenoid. A capsular shift and repair to the glenoid was performed using 4 suture anchors. Because of the poor tissue quality, a TissueMend collagen matrix (Stryker Orthopaedics, Mahwah, New Jersey) was added to the inferior aspect of the capsule for structural support. The capsule was appropriately tensioned, the rotator interval was closed, and the conjoint tendon was repaired. A minimally displaced greater tuberosity fracture was also appreciated but not addressed (Figure 7).

At most recent follow-up, 7 months postoperatively, the shoulder was located and the patient was continuing active physical therapy. She had forward flexion to 150°, external rotation to 40°, and internal rotation L5 with 4+/5 strength in all 3 areas.

DISCUSSION

In cases of capsulitis where a patient's benefit from conservative management has plateaued, operative intervention is useful to restore a functional shoulder. A gentle manipulation may be attempted initially, but often an arthroscopic capsular release is needed. The capsular scar caused by the fibroplasia is divided, removing the restraint to ROM. The postoperative protocol includes aggressive ROM, stretching, continuous passive motion, anti-inflammatories, and pain control.

Several studies have shown that arthroscopic release for refractory adhesive capsulitis is an appropriate intervention, producing significant increases in ROM compared with the preoperative state.[7,11,15-18]

In one study, after arthroscopic capsular release the average increase in flexion was 49°, adduction 42°, external rotation in abduction 53°, internal rotation in abduction 33°, and internal rotation in adduction 8 spinous levels.[11] Warner et al[11,16] concluded that in patients who have loss of motion refractory to closed manipulation, arthroscopic capsular release reliably improves motion with little operative morbidity. Physical therapy was conducted twice a day starting postoperative day 1, and stretching was limited only by the patient's intolerance to pain. The group did not have any postoperative episodes of instability. The authors felt that this was due to articular compression by the remaining soft-tissue envelope.

Few complications have been reported after capsular release procedures. Most common complications include persistent pain and restricted ROM. To our knowledge this is the first report of shoulder dislocation after surgical release and manipulation procedures for adhesive capsulitis. We have presented 3 patients with a diagnosis of capsulitis of varying etiologies: adhesive, postsurgical, and postfracture. In these cases a manipulation with capsular release was performed, resulting in a dislocation that was

missed in varying intervals from 18 hours to 1 year. The question of limiting the scope of surgical release could be raised in 2 of our patients. We have generally released the capsule from anteriorly to inferiorly and then at times moved the arthroscope to the anterior portal to perform a posterior release. Perhaps avoiding an inferior release could add stability, but elevation would be limited.

Adhesive capsulitis is generally described as a global pathology to the glenohumeral joint. In surgical releases, particularly those performed arthroscopically, the release may not be circumferential and may only include the anterior and inferior portions of the capsule, as in patient 1. Therefore, the remaining unreleased portions of the capsule continue to help stabilize the shoulder joint. This tenet has led many surgeons to be aggressive with respect to mobilizing their patients postoperatively. In each of the 3 cases presented, the patient had a nontraumatic shoulder dislocation postoperatively. It is plausible that in an effort to maximize mobility, one can sacrifice stability.

Another important consideration is the use of regional anesthesia. At our institution, long-acting regional blocks and indwelling interscalene catheters are routinely used to manage postoperative pain and improve a patient's ability to participate in early physical therapy. However, these interventions mask the clinical symptoms of dislocation and paralyze the dynamic stabilizers of the glenohumeral joint. This was a contributing factor to the dislocation and delay in diagnosis in patient 1.

Patient 3 had poor-quality capsular tissue that needed augmentation. In this case, a less aggressive release may have been more appropriate. Furthermore, the postoperative protocol placing these patients in the overhead position may place the shoulder at an increased risk for subluxation/dislocation due to the inherent instability of the position. As a result of these cases, we have discontinued this protocol at our institution.

In patients 1 and 2, the dislocation was associated with a brachial plexopathy. Zanotti et al[17] showed the proximity of the neurovascular bundle to the subscapularis tendon. This study demonstrated that a safe margin between the capsule and the neighboring neurovascular structures can be obtained by releasing the capsule within 1 cm of the glenoid rim. In general, careful dissection and meticulous technique can prevent neurovascular injury during these procedures. The neurologic injury presented in our series may have been due to compression on the brachial plexus from the humeral head for 18 hours to 1 year.

CONCLUSION

The potential for instability and dislocation in patients with stiff shoulders has frequently been an afterthought, the primary focus being on mobilization. Based on our findings, surgeons should have a higher index of suspicion for this complication. It is the authors' opinion that aggressive mobilization in the immediate postoperative period should be tempered by the clinical stability assessment in order to prevent instability episodes. Specifically, patients should be tested for stability as well as ROM in the operating room postoperatively. If the patient dislocates, the rehabilitation program should be adjusted accordingly. Furthermore, careful, early postoperative evaluation by clinical examination and radiograph may prevent a delay in diagnosis, which resulted in a neurologic injury in 2 of our patients and marked joint destruction due to extended physical therapy in our second patient.

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