Adhesive Capsulitis

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Abstract: Adhesive capsulitis is a common problem seen in the general population by orthopedic surgeons. It is a problem that causes patients pain and disability, and symptoms can last up to 2 years and longer. The questions of when and how to treat the frozen shoulder can present challenges. Most treatments are conservative; however, indications for surgery do exist. Arthroscopic capsular release has gained popularity over the years and offers a predictably good treatment in patients with adhesive capsulitis. The purpose of this paper is to review the orthopedic literature on adhesive capsulitis, to provide background information on this topic, and to describe our technique in arthroscopic capsular release.

Key Words: adhesive capsulitis, frozen shoulder, shoulder stiffness, manipulation under anesthesia, arthroscopic capsular release

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Adhesive capsulitis of the shoulder is a very unique entity in that the shoulder is the only joint in the body that is affected by this type of disease process. The term "frozen shoulder" is defined as a clinical condition with restricted active and passive range of motion (ROM) in all directions, including flexion, abduction, and rotation. Frozen shoulder was first described in 1934 by Codman. In 1945, Neviaser described synovial changes seen in the glenohumeral joint and coined the term "adhesive capsulitis."[1] Lundberg[2] categorized the frozen shoulder into idiopathic or primary adhesive capsulitis and secondary adhesive capsulitis. The pathogenesis of the idiopathic form remains unclear, although there are many proposed mechanisms. Harryman and Neviaser[3] suggest endocrine, immunologic, inflammatory, and biochemical changes as possible causes. Janda[4] described how there is an increased incidence in patients with diabetes.

Secondary adhesive capsulitis develops when there is a known intrinsic, extrinsic, or systemic cause. Possible causes of secondary frozen shoulder include macrotrauma, microtrauma, or postsurgical intervention, combined with prolonged immobilization of the shoulder. Causes of posttraumatic adhesive capsulitis might include acute fractures, missed fractures, and dislocations. Specific shoulder procedures can cause persistent shoulder stiffness. These can include arthroscopic or open stabilization and rotator cuff repairs. These present a challenging dilemma to the surgeon when even a potential manipulation or release can jeopardize the original procedure. Other causes include cervical spine pathology, RSD, chronic obstructive pulmonary disease, thyroid disorders, various medications, and ischemic heart disease. A relationship with Dupuytren disease has been documented. Bunker and Anthony[5] showed that the microscopic changes seen in the anterior capsule and coracohumeral ligament are similar to Dupuytren disease of the hand.

EPIDEMIOLOGY

In the United States, shoulder pain ranks as the third most common cause of musculoskeletal disability. Frozen shoulder is felt to have a prevalence rate of 2% in the general population; however, an 11% prevalence rate is reported in diabetics. Patients with type I diabetes have a 40% chance of developing a frozen shoulder in their lifetimes. Frozen shoulder might affect both shoulders in up to 16% of patients; however, a relapse is uncommon. An increased incidence of frozen shoulders has been noticed in patients with hyperthyroidism and hypertriglyceridemia. Adhesive capsulitis is more common in the fifth and sixth decades of life, and other medical problems should be investigated in patients below 40 years of age. No racial predilection has been described in the literature; however, women are affected more than men with a ratio of 58:42.[6]

HISTORY AND PHYSICAL

Most patients with primary frozen shoulder have no history of shoulder trauma. A careful history of trauma, cervical radiculopathy, brachial plexus injury, and cardiac ischemia should be documented by the physician. Most patients present with an insidious onset of pain, followed by a loss of motion. Most of the pain seems to be neurologically mediated. Peripheral α-adrenoreceptor hyperresponsiveness in the somatosensory neurons of both nociceptive and proprioceptive fibers in the shoulder joint seems to mediate the pain response in many shoulder conditions, including adhesive capsulitis.[6] Physical examination shows loss of both passive and active ROM. Early on in the disease process, the only physical examination finding might be pain produced at the end range of the shoulder motion.[7] As the disease progresses,

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loss of motion will be seen in the shoulder. Up to 80% of shoulder motion might be lost, with external rotation and abduction being the most commonly affected, and to a lesser degree, flexion. Extension and horizontal adduction motion are least affected.[6]

CLINICAL ASSESSMENT

Laboratory data are usually normal; however, in patients with other medical issues, thyroid-stimulating hormone, lipid levels, and fasting blood glucose might be elevated.[8] Plain films are usually normal, but can show calcification of the rotator cuff. Arthrography of the shoulder shows decreased shoulder volume, and technetium bone scan shows increase uptake; however, Binder et al[9] found in their study that bone scan and shoulder arthrography do not contribute to the assessment of the painful stiff shoulder. Mengiardi and Gerber found the thickening of the coracohumeral ligament and joint capsule in the rotator cuff interval to be characteristic magnetic resonance (MR) arthrographic findings in the frozen shoulder.[1] MR imaging scans can also be useful in diagnosing other disease processes presenting with shoulder pain and stiffness, such as infection, rotator cuff tears, Pancoast tumor, and other shoulder pathology. MR imaging, however, should not be routinely ordered in the evaluation of the frozen shoulder, as Manton Geoffrey et al's[10] study showed no useful MR arthrographic signs of adhesive capsulitis.

PATHOANATOMY AND HISTOLOGY

Pathoanatomy shows decreased volume of the glenohumeral joint with restricted ROM. The arthroscopic examination shows a reduced volume with a tight capsule, synovial hypertrophy, and neovascular proliferation. In the first stage or freezing stage, the early inflammatory stage, hypervascular synovitis is seen. In the second stage or frozen stage, there is a decrease in hypervascularity and synovitis; however, capsular contraction and thickening is noted on arthroscopic evaluation. In the third stage or thawing phase, no synovitis is seen, and there is a decrease in the thickness of the capsule. Arthroscopy is rarely indicated in the first or third stage of adhesive capsulitis. Although the glenohumeral joint synovial capsule is involved, much of the disease process involves structures outside the shoulder joint, including the coracohumeral ligament, rotator interval, subscapularis musculotendinous unit, and the subacromial bursae.[11]

Histologic studies show chronic fibrosis of the capsule, with the predominant cells involved being the fibroblast and myofibroblast. Bunker et al's[12] study showed an increase in fibrogenic growth factors and matrix metalloproteinases and their inhibitors. Rodeo et al[13] demonstrated elevated levels of cytokines in frozen shoulders. The findings from these studies are compared with the histologic findings of Dupuytren disease.

NATURAL HISTORY AND CLASSIFICATION

The natural course of a frozen shoulder is usually self-limiting. It is a disease that improves over an 18 to 24 month period. In 2004, Diercks and Stevens[14] showed that there is an increase in constant shoulder scores with time when it was treated with "supervised neglect." Multiple studies have demonstrated an improvement with different types of treatment. Dominant arm involvement has been shown to have a good prognosis; associated intrinsic pathology or insulin-dependent diabetes of more than 10 years are poor prognostic indicators.[15]

Three stages of adhesive capsulitis have been described, with each phase lasting for about 6 months. The first stage is the freezing stage in which there is an insidious onset of pain. At the end of this period, shoulder ROM becomes limited. The second stage is the frozen stage, in which there might be a reduction in pain; however, there is still restricted ROM. The third stage is the thawing stage, in which ROM improves, but can take between 12 and 42 months to do so. Most patients regain a full ROM; however, 10% to 15% of patients suffer from continued pain and limited ROM.[3] In 2004, Dudkiewicz et al[16] showed that some patients with a frozen shoulder might show improvement 10 years after the onset of symptoms. Rowe and Leffert[17] in 1988, along with Cameron et al[18] in 2000, showed that recurrence of the frozen shoulder is extremely rare.

TREATMENT

Treatment of adhesive capsulitis is mainly nonoperative, with most patients improving over a time period of 18 to 24-months. Nonoperative treatment consists of physical therapy, intra-articular steroid injections, and nonsteroidal anti-inflammatory drugs (NSAIDs). Physical therapy consists of a supervised home-based stretching and strength maintenance program with the use of electroanalgesia and warm or cool pads for pain relief. Rizk et al[19] showed that TENS does help to diminish pain and showed improvement in pain and ROM in his study. A recent review of the literature showed that there is little evidence to support the use of more common physiotherapeutic modalities such as bipolar interferential electrotherapy, pulsed ultrasound, and magnetotherapy. In 2000, Griggs showed that most patients with phase II idiopathic adhesive capsulitis could be successfully treated with a specific 4-direction shoulder exercise program. He did, however, show that patients with more severe pain and functional limitations, as well as those with pending litigations and workers' compensations, had worse outcomes, and often needed manipulation or capsular release.[20]

NSAIDs, acetaminophen, and a short course of prednisolone for treatment of adhesive capsulitis can have the benefit of pain relief and a decrease in the inflammation of the shoulder. Most of the benefits take the form of pain relief rather than an improvement in ROM. Lee et al[21] showed that patients had improvement when analgesics were added to a stretching program.

Buchbinder et al[22] showed in his study that a 3-week course of 30 mg of prednisolone daily has a significant short-term benefit, but this is not maintained beyond 6 weeks.

Intra-articular steroid injections are also useful in the treatment of the inflammatory phase of adhesive capsulitis and are the second most common medical intervention, second to NSAIDs. Many people have demonstrated improvement in symptoms with intra-articular steroid injections. Carette et al[23] showed significant improvement after treatment with corticosteroid injections plus exercise versus exercise alone. More recent controlled clinical trials have failed to show good results with the injection of steroids in the shoulder joint. One of the main concerns with shoulder injections is the delivery of the steroids into the glenohumeral joint. Eustace et al's[24] study and other studies have showed that 68% of the shoulder injections administered by experts without radiologic guidance failed to enter the glenohumeral joint. With a posterior injection technique and an over-90% accuracy rate, we have had positive results in the form of a decrease in pain and an increase in ROM, both in the short and the long terms, in over 80% of the patients.

Intra-articular joint distention or brisement has also proved to be of benefit. Distention of the capsule to the point of capsular disruption has been shown to help with both pain relief and the increase in ROM. Most studies have shown good short-term benefits for 1 to 3 months with this treatment; however, no difference was evidenced in the long-term outcomes when compared with other treatment modalities.[3]

Surgical treatment of adhesive capsulitis, including manipulation and arthroscopic capsular release, should be reserved for patients who do not respond to conservative treatment after a minimum of 6 months of appropriate nonoperative treatment. Manipulation can prove to be effective; however, this does not allow for controlled release of pathologic tissue and has an increased risk of causing a humeral shaft fracture. Contraindications and relative contraindications to manipulation under anesthesia (MUA) are (i) no improvement or worsening in ROM or comfort after previous manipulation and (ii) patients with significant osteopenia, a rotator cuff tear, or long-term insulin-dependent diabetes.[3] Kessel showed that patients do better with MUA if they have been symptomatic for more than 6 months.[25] Reported results of MUA are variable, with a range of 25% to 90% of patients improving 3 months after manipulation, and an average of 70% improving after 6 months. Dodenhoff et al[26] found that 94% of the patients in his study who had undergone MUA were satisfied with their results, but 12.8% still had persistent disability. Fox et al[27,28] showed in 2005 that MUA under interscalene block results in sustained improvement in function and movement at the 12-month follow-up.

Open capsular release is not very common owing to its high complication rate. It is technically difficult to achieve a complete posterior release, and postoperative pain and the need to protect the lengthened subscapularis tendon inhibit the unrestricted ROM needed to maintain motion. Braun et al[29] recommend open release in patients with severe restriction in motion secondary to head injuries or strokes. Open release might occasionally be indicated in posttraumatic and postsurgical cases of adhesive capsulitis in which extensive subdeltoid scarring and also extensive intra-articular and extra-articular contractures have occurred, which are not amenable to arthroscopic release.

Arthroscopic surgical release was first described in 1979 by Conti. Since then, it has become the main operative treatment of adhesive capsulitis. Ogilvie-Harris and D'Angelo[30] resected the inflamed synovium and divided the anterior capsule, inferior capsule, and subscapularis tendon. They found good results when using arthroscopic release in diabetic patients.[30] Segmuller et al[31] found in their study that arthroscopic release is safe and effective in treating adhesive capsulitis. Warner and associates[32] showed in their study that arthroscopic capsular release improves motion, with little operative morbidity, in patients who have loss of shoulder motion that is refractory to closed manipulation. Pearsall et al[33] described releasing the intra-articular portion of the subscapularis; however, most studies show excellent results without subscapularis release. The advantages of this approach include the complete release of the contracted capsule in a controlled manner. Also complete synovectomy is possible. Patients have minimal postoperative pain, and aggressive active and passive motion can be started immediately. One can also identify other shoulder pathology that can cause shoulder pain and disability. Some of the risks of arthroscopic capsular release include recurrent stiffness, anterior dislocation immediately after the operation, and axillary nerve palsy; however, these complications are rare.

SURGICAL TECHNIQUE

It is imperative that a full examination under anesthesia be completed by examining the free passive ROM of both the affected and the unaffected shoulders. This will give the surgeon a realistic goal as to what can be obtained, and, it is hoped to prevent overmanipulation. This must be done before the patient is placed into the decubitus position, if that is the desired surgical position. Although some authors suggest a gentle MUA before surgery, we do not advocate this because of the fear of excessive bleeding before the arthroscopic procedure.

Positioning the patient is possible with the use of 2 standard techniques. One is the lateral decubitus position, in which the arm is placed in traction/suspension. The other is the beach chair position, in which both arms are free if comparisons are needed, and the patient is placed in an upright position.

The affected extremity is prepped and draped in the usual fashion; we do not feel that it is necessary to give preoperative antibiotics. We use the lateral decubitus

position. The patient is placed in a 60-degree upright position, with the affected extremity being placed in approximately 12 pounds of traction-suspension (Fig. 1). The landmarks of the shoulder are carefully identified with a marking pen.

The posterior portal is established in the usual fashion. Sometimes entry into the shoulder through this portal is difficult because of the restricted joint capsule and diminished space. Additional traction and rotation can aid the surgeon in entering the joint.

Once the joint is successfully entered, it is commonplace to encounter some early bleeding because of the synovitis and reactive capsulitis in the joint (Fig. 2). The control of bleeding needs to be established in the usual fashion, but is probably even more important in this particular operative procedure. We employ a pump and generally start at a 45-mm Hg pressure: this facilitates either increase or decrease as necessary. Epinephrine can be used in the irrigating solution. It is imperative to have hypotensive anesthesia, if medically advisable, with a systolic pressure below 95 mm Hg. It is imperative to have adequate flow, which will be established once the anterior portal is established. Radiofrequency (RF) is used not only to control the bleeding, but for controlled release.

The anterior portal is then established, either outside in or inside out. Sometimes difficulties in carrying out an inside-out method might arise because of the very limited glenohumeral space. Therefore, even if one is used to an inside-out method, an outside-in technique might be necessary.

A bipolar RF device is used for the resection and hemostasis. Mechanical devices can be used, but the bipolar RF device is far more versatile in reaching all the targeted areas in the shoulder. It also has a very controlled cutting and coagulation mechanism that allows the surgeon to be more precise. If the surgeon approaches the axillary nerve and gets too close to it, then the muscle twitch will alert the surgeon to this risk.

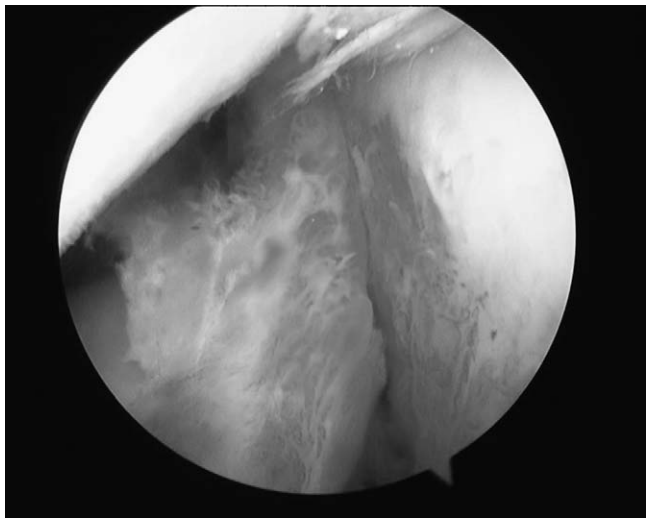

FIGURE 2. Inflammatory synovitis in the patient's glenohumeral joint.

The procedure begins with a release of the rotator interval, and then working the RF device down the anterior capsule, staying very close to the labrum, and attempting to reach the 6-o'clock position, having started at the 1-o'clock position (Fig. 3). The device is completely through the capsule when the capsular tissue is seen to separate while the shoulder joint is under constant pressure from the irrigating fluid. Approximately 70% of the time, the underlying muscle tissue is seen, which also indicates complete capsular resection. A 90-degree device seems to be the most versatile one to perform the resection with (Fig. 4). The surgeon can alternate between cutting when resecting the capsule and coagulation when

FIGURE 1. The patient positioned in the right lateral decubitus position with the arm in 12 pounds of traction-suspension.

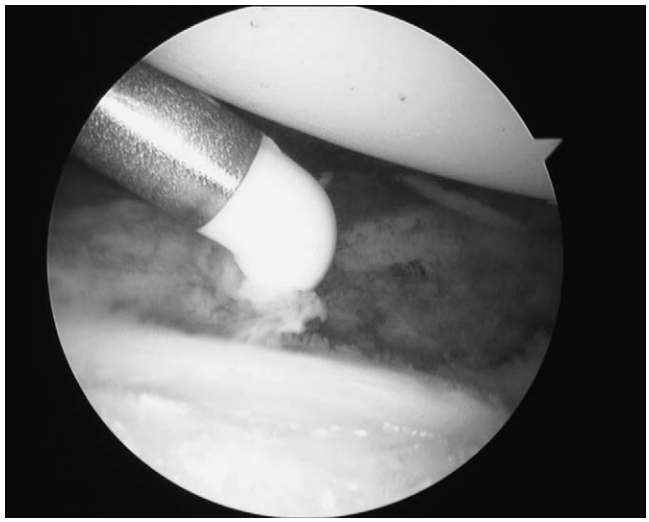

FIGURE 3. A 90-degree bipolar RF device sectioning the capsule in proximity to the glenoid.

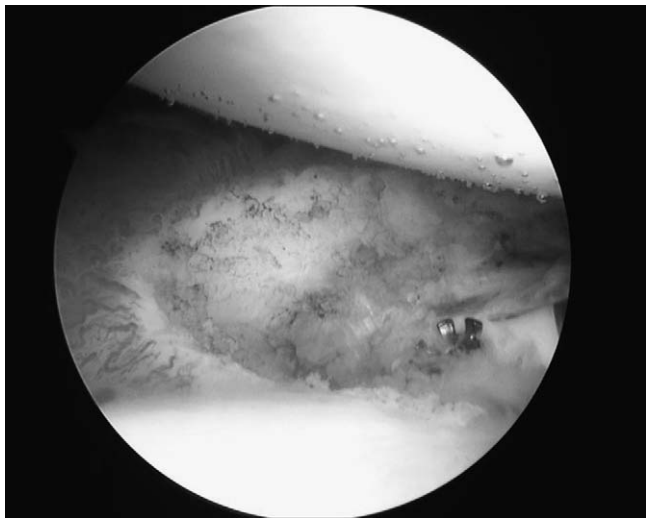

FIGURE 4. Appearance of the capsule after RF resection.

bleeding is encountered. When the 5-0'clock position is approached, great care is needed in spotting the axillary nerve, which might come into view. The distension in the capsule might separate the capsule well enough for the surgeon to actually visualize it. Under most circumstances, a small portion of the tendon of the intra-articular component of the subscapularis (Fig. 5) will have to be released. The rationale behind this is to place a small stress riser in this tendon so that, after the arthroscopic portion of the procedure, during manipulation, the musculotendinous construct undergoes a controlled stretch rather than a rupture. Approximately 5% to 7% of the entire subscapularis tendon is cut near its insertion. This represents about a 3 to 4-mm cut through the tendon. The intra-articular portion of the subscapularis tendon only represents a small fraction of the entire subscapularis tendon. The intra-articular pressure needs to be carefully monitored at this stage because the capsule is now open and extravasation can occur, along with a significant amount of fluid loss into the surrounding tissue. The superior resection of the capsule is then completed from 11 o'clock to 1 o'clock with the same anterior portal. The superior release includes the superior rotator interval and the superior capsule. The biceps and labrum are not released.

At this stage, the portals are exchanged using switching sticks, and the resection is visualized anteriorly and completed posteriorly. The pump pressure is then lowered to 20 mm Hg, to allow for the visualization of any unusual bleeding. The posterior capsule is resected from 11 o'clock to 6 o'clock, again staying very close to the glenoid. The arthroscopic cannulas and scope are then removed after a thorough irrigation.

The patient then undergoes a formal manipulation in the following fashion and order: forward elevation, followed by external rotation and internal rotation at 0 degrees of adduction. This is followed by extension. The arm is then taken through another forward elevation maneuver, and then the external and internal rotation maneuvers are carried out at 90 degrees of abduction. The final manipulation that is performed is abduction. The manipulation is repeated in the above order on as many occasions as necessary, to achieve as complete an ROM as is possible.

Surgeons who prefer to manipulate first and arthroscope after the manipulation do so to visualize their results, but they usually encounter a significant amount of bleeding (Fig. 6). It is optional to take a relook at this stage, but there is no reason to rearthroscope the shoulder after manipulation.

After the operation, the patient is started on physical therapy and a home exercise program, both passive and active-assisted. In some refractory cases, CPM can be helpful. Pain pumps should be used,

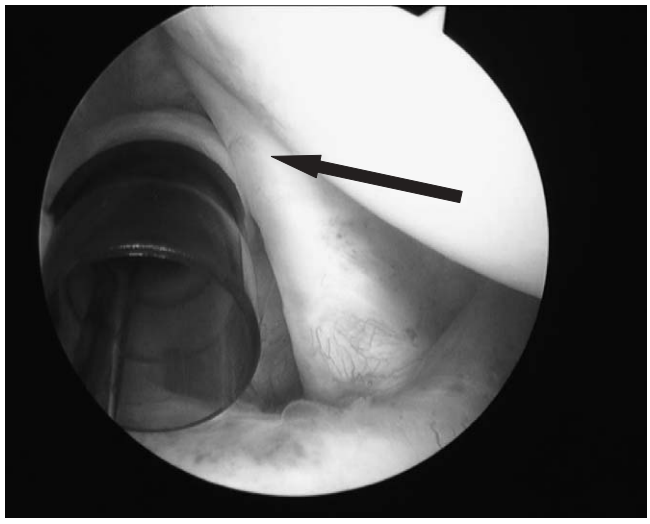

FIGURE 5. The subscapularis tendon with inflammatory changes. The arrow depicting where a partial release has been achieved.

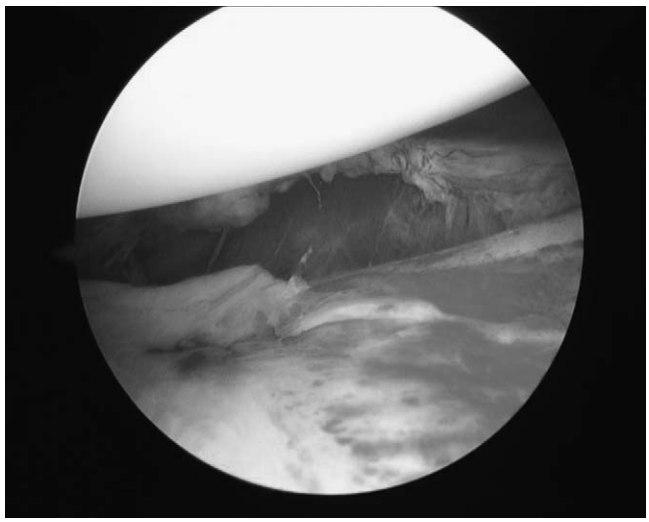

FIGURE 6. Capsular tearing after MUA.

preferably in the subacromial space for 72 hours, as some adverse reactions have been reported when they are placed intra-articularly. We prefer this to an interscalene block or an indwelling interscalene block. It is imperative that the patient is followed up very closely, given support during this most difficult period, and also monitored for home-therapy progress.

SUMMARY

In summary, adhesive capsulitis is a fairly common shoulder problem. It can be debilitating for patients, and most cases take 12 to 24 months to show improvement. It occurs mainly in diabetic women in their fifth decade of life.[34] Most patients can be successfully treated with nonoperatively; however, good results in the refractory cases can be obtained with arthroscopic capsular release followed by controlled MUA.

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