Int J Med Rev 2024 December;11(4): 820-829

10.30491/IJMR.2024.482011.1297

INTERNATIONAL JOURNAL OF MEDICAL REVIEWS

Narrative Review

Management and Diagnosis of Adhesive Capsulitis of Shoulder: Current Review

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Received October 5, 2024; **Accepted** December 7, 2024; **Online Published** December 30, 2024

Abstract

Adhesive capsulitis, commonly known as frozen shoulder, is a painful disorder that causes the shoulder joint to become very rigid. One kind is primary (idiopathic), while the other is secondary (caused by something else, such as trauma, surgery, or being immobile) that is related to inflammation. The incidence rates may be approximately 20% in people with diabetes, far greater than the general population frequency of 2-5%. Limitations in shoulder mobility are the result of an inflammatory process that starts within the joint capsule and progresses to fibrosis and adhesion development. When there are no significant radiological abnormalities and the patient experiences a gradual decrease in active and passive shoulder mobility, a clinical diagnosis is usually made. In most instances, the disease's symptoms will go away after a while, but in others, they may linger for years. Ultrasound and magnetic resonance imaging (MRI) may aid in the diagnosis of more complicated situations. Treatment options include non-invasive methods such as physiotherapy, NSAIDs, and injections of steroid medication into the affected joint. Surgical procedures, including capsular release or manipulation while under anesthesia, may be necessary for more serious or long-lasting instances. Although adhesive capsulitis usually resolves on its own, untreated symptoms might cause impairment in the long run.

Keywords: Adhesive Capsulitis, Frozen Shoulder, Shoulder Stiffness, Inflammation

Introduction

Adhesive capsulitis is an inflammatory illness that produces pain and stiffness in the shoulder joint. It is more often known as frozen shoulder. The American Academy of Orthopedic Surgeons has identified a gradual decrease in shoulder range of motion without significant changes in radiological findings as the characteristic that must be present in order for a diagnosis to be made. A correct diagnosis of significant passive range of motion loss is of the utmost importance.[1,2] The following is the American Shoulder and Elbow Society's (ASES) widely accepted definition of ACS: “a condition characterized by functional restriction of both active and passive shoulder motion for which radiographs of the glenohumeral joint are essentially unremarkable”.[3]

Both primary and secondary adhesive capsulitis may develop. Adhesive capsulitis, also known as primary or idiopathic capsulitis, may develop on its own, unprompted by any external factor. Secondary adhesive capsulitis often develops after significant articular trauma, such as a glenohumeral joint fracture dislocation or another kind of periarticular fracture. Shoulder arthroplasty, rotator cuff repair, and other arthroscopic and open procedures may all lead to this serious complication. Adhesive capsulitis affects up to 20% of diabetic people, compared to 3% to 5% of the general population. Although bilateral involvement has been seen in as many as 40 to 50% of instances with idiopathic adhesive capsulitis, the nondominant extremity is more often affected. In most cases, the symptoms of adhesive capsulitis go away on their own within a year or two. Nonetheless, research suggests that 20% to 50% of people may have symptoms that continue for a long time. To achieve satisfactory functional results in this patient group, a combination of non-operative and surgical therapies is required.[4]

Epidemiology

Primary and secondary types of adhesive capsulitis are distinguished. Idiopathic primary adhesive capsulitis usually develops slowly. Common co-occurring disorders include diabetes, thyroid issues, medication side effects, hypertriglyceridemia, and cervical spondylosis. Shoulder trauma, injuries (e.g., rotator cuff tears, fractures, surgeries, or extended immobilization) are the usual causes of secondary adhesive capsulitis.[4,5]

Approximately 2% to 5% of the population has adhesive capsulitis, and the average age at which symptoms first appear is 55. The non-dominant hand is also affected, and the female hand is somewhat more prevalent (1.4:1). Adhesive capsulitis is more common in patients with autoimmune comorbidities, including thyroid problems and diabetes mellitus. Treatment results for people with diabetes may also be worse, and this is related to how long they have had the disease.[6]

Hypothyroidism and diabetes mellitus have been linked to adhesive capsulitis. The incidence of adhesive capsulitis was five times higher in diabetic patients than in the control group, according to a meta-analysis conducted in 2016. Using a 95% confidence range [CI] of 24% to 37%, the same meta-analysis determined that 30% of individuals with adhesive capsulitis also had diabetes. Adhesive capsulitis was associated with a much greater incidence of hypothyroidism diagnoses (27.2% vs. 10.7%; $P = 0.001$) in a 2017 case-control study.[7]

Pathogenesis

The exact mechanism by which adhesive capsulitis develops is not yet known. The current thinking is that synovial lining adhesions and reactive fibrosis occur when inflammation starts in the joint capsule and synovial fluid. Early inflammation of the capsule causes pain, and fibrosis and adhesions in the capsule restrict movement.[8]

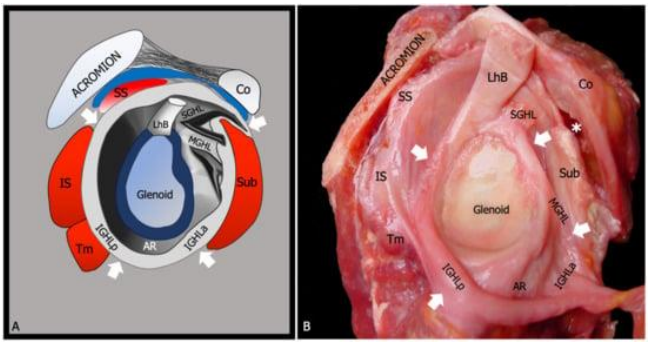

Figure 1. (A) Illustration and (B) Anatomic Dissection of the Glenohumeral Joint Capsule (white arrows) and its Relationship with the Overlying Rotator Cuff Tendons. Superior glenohumeral ligament (SGHL), middle glenohumeral ligament (MGHL), anterior and posterior bands of the inferior glenohumeral ligament (IGHLa and IGHLp). Supraspinatus tendon (SS), infraspinatus tendon (IS), teres minor tendon (Tm), subscapularis tendon (Sub), long head of the biceps tendon (LhB), coracoid process (Co), axillary recess (AR).

The characteristic sign of adhesive capsulitis is a tightening of the glenohumeral capsule. Decreased capsular volume, axillary adhesions to the humeral neck and to one another, and synovial layer loss are all findings. Because it is crucial to the glenohumeral joint's health, the thickening and fibrosis of the rotator interval that is associated with adhesive capsulitis is remarkable. The rotator interval is bordered by the tendon of supraspinatus on top, the tendon of subscapularis on the bottom, the trans humeral ligament on the side, and the coracoid process in the center. The rotator interval is comprised of the glenohumeral capsule, biceps tendon, and CHL. A significant finding in adhesive capsulitis is a contracted CHL. The CHL ligament is the main target of adhesive capsulitis surgery because of the tension it undergoes during full external rotation. The CHL ligament in the injured shoulder of adhesive capsulitis patients is shown to be stronger compared to the non-affected shoulder, as revealed by shear-wave elastography. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) scans of patients with adhesive capsulitis indicate CHL thickening (4.1 mm vs. 2.7 mm). The control group had a smaller axillary recess capacity of 0.53 mL and a bigger rotator interval capsule of 7.1 mm compared to 4.5 mm.[4]

Histological examination of materials from patients with adhesive capsulitis often reveals fibroblasts along with type I and type III collagen, lending credence to the long-held belief that the condition is mostly fibrotic and comparable to Dupuytren's disease. Capsular

contracture is thought to be caused by the fibroblasts' transformation into myofibroblasts, which exhibit characteristics of smooth muscle. Changes in the levels of matrix metalloproteinases (MMPs) are shown; these enzymes are involved in the remodeling of scar tissue. Those who suffer from adhesive capsulitis are unable to produce MMP-14, in contrast to healthy controls. Because of its function as an activator of MMP-2, a protein involved in collagen degradation, MMP-14 may lead to an excess of collagen rather than its breakdown. The expression of matrix metalloproteinases (MMP)-1 and MMP-2 decreases in adhesive capsulitis patients, whereas the expression of tissue inhibitors of metalloproteinases (TIMP)-1 and TIMP-2 increases. The findings indicate that disorders in the breakdown, remodeling, and regeneration of the extracellular matrix tissue might lead to adhesive capsulitis. Possible future therapeutic approaches include promoting remodeling of fibrotic tissue or directly inhibiting fibrogenesis.[4]

Inflammation and fibrosis are now universally acknowledged as factors in the development of adhesive capsulitis. Research has shown that when compared to normal subjects, capsular and bursal tissues from people with adhesive capsulitis exhibited elevated levels of inflammatory cytokines such as interleukin (IL)-1α, IL-1β, tumor necrosis factor (TNF)-α, cyclooxygenase (COX)-1, and COX-2. Adhesive capsulitis, which develops into fibrotic changes, is therefore primarily caused by inflammation. Almost all rotator interval samples collected from persons with adhesive capsulitis include inflammatory cells, including T cells, B cells, macrophages, and mast cells. In vivo, mast cells control fibroblast growth and may mediate the transition from inflammation to fibrosis. More recent research has attempted to establish a connection between adhesive capsulitis' molecular etiology and established risk factors and hereditary predisposition. Research using cytogenetic analysis has shown that individuals with adhesive capsulitis had higher levels of inflammatory (IL-6) and fibrogenic (MMP-3) cytokines.[4]

People often think of fibrotic conditions like Dupuytren's disease when they see fibroblasts mixed with type I and type III collagen in tissue microscopy from AC sufferers. It appears that the capsular contraction is induced by the fibroblasts transforming into myofibroblasts, which display smooth muscle traits. It seems that the sickness is advancing due to the availability of adhesion-related cytokines, including transforming growth factor Ω (TGF-β) and platelet-derived growth factor (PDGF). TGF-β has been associated with fibrosis, the development of an extra-capsular thick collagen matrix, elevated PDGF levels, and, as per some research, it might potentially stimulate AC. Using an adenovirus vector that had a high concentration of TGF-β1, Watson et al. discovered that AC manifested 5-10 days after treatment with rats. Because TGF-o and PDGF were detected at higher concentrations in the contracted tissue, their effects were inhibited when cell cultures were established using both normal and contracted patient tissues. Blocking increased chemotaxis and decreased collagen production and dispersion. Tissue samples from both AC patients and healthy controls were analysed in another study to determine the frequency of matrix metalloproteinases (MMPs) and inhibitors of these enzymes. Scar tissue remodeling involves matrix metalloproteinases (MMPs). The AC group does not include MMP-14. Matrix remodeling is controlled by membrane metalloproteinases. Since MMP-14 promotes MMP-2, which is involved in collagen degradation, its absence may cause collagen to be overproduced rather than broken down. The blood was tested for levels of AC-related proteins, MMP-1 and MMP-2, as well as TIMP-1, TIMP-2, and TGF-β1, in an independent study. The AC group had significantly lower levels of MMP-1 and MMP-2 and significantly higher levels of TIMP-1, TIMP-2, and TGF-β1. These results lend support to the theory that AC results from an inappropriate interaction between the aging, remodeling, and regeneration of extracellular matrix (ECM) tissues. Several proteins, including as vascular endothelial growth factor, CD29, NF-κB, JNK, ERK, and Jun N-terminal kinases, have been found in tissues collected from individuals with AC at significant amounts.[9]

Three or four clinical phases have historically been used to explain the natural history of AC; these stages correlate to certain histologic abnormalities that may be seen in the joint capsule of individuals afflicted.[9,10]

- In the first three months, you may have a sudden, vague discomfort around your deltoid region; this pain is more pronounced at night. This is known as the painful stage. Additionally, you may notice a slight limitation in the range of motion for the glenohumeral joints. Histologically speaking, the glenohumeral joint capsule becomes more inflamed,

synovitis develops, and hypervascularity occurs at this stage.

- The second stage, also known as the freezing stage, begins in the third month and continues until the ninth month, during which the patient feels a worsening of the pain and stiffness in their joints. Flexion, abduction, and additional rotation are the movements in which joint stiffness is most noticeable. The synovial membrane thickens and becomes more vascular, collagen deposits in an irregular pattern, and adhesions form at the macroscopic level, indicating a change in the capsule. More recent accounts have combined the first and second phases.
- During this phase, which may last until the fourteenth month and is marked by a major restriction of joint motions, the pain is initially less visible while at rest but becomes much worse during passive mobilisation of the glenohumeral joint. The third stage, sometimes called the frozen stage, is where we are today.
- Stage four, the thawing phase: In this final stage, which may continue for as long as two years, you will notice that your stiff joints and aches are going away on their own. In the third and fourth phases, histological analysis of the capsular tissue reveals the presence of mature and adherent hypercellular collagen, with fewer obvious symptoms of inflammation.

Clinical Examination

Adhesive capsulitis evaluations begin with comprehensive history taking of the shoulder. It is common to mention slight trauma as an aetiology for shoulder discomfort. Though it can be something completely unrelated to the procedure, the patient might remember anything that is associated with beginning it. It is common for the non-dominant limb to be affected by adhesive capsulitis. This occurs because the dominant extremity can carry out the necessary tasks, and it is more comfortable to avoid using the uncomfortable extremity altogether. Holding the limb close to the body, as if to "protect it," allows the process to continue unabated. This is further highlighted when there is an abnormally high level of discomfort and guarding together with passive range of motion. Codman described this entity as having a gradual development of discomfort at the deltoid insertion, difficulty sleeping on the afflicted side, and restrictions in external rotation, active and passive elevation, and pressure, but with normal radiologic appearance. This patient's history and radiographs point to adhesive capsulitis rather than degenerative joint degeneration.

Adhesive capsulitis is characterized by limited range of motion and pain in the affected shoulder. A dull ache, not localized at all and maybe spreading to the biceps, is one of the symptoms. Reaching up or behind you could help ease any stiffness or pain you may be experiencing. Fever, night sweats, malaise, or unexplained weight loss are signs that a doctor should look out for in order to rule out neoplasm or autoimmune sickness. When neuropathy symptoms manifest, a cervical radiculopathy diagnosis should be investigated.[12] Shoulder pain caused by adhesive capsulitis often develops gradually over many weeks or months. Restriction of shoulder motion is the subsequent stage. Restrictions in active and passive ROM, especially in the directions of internal and external rotation, abduction, and forward flexion, are hallmarks of adhesive capsulitis. In extreme cases, further symptoms may include muscular dystrophy and an abnormality in the way the arms swing while walking. When you palpate the affected shoulder, you will feel widespread pain around the joint. The safety of the distal nervous system must be ensured. The pain and severe limitation felt when the shoulder cannot move freely are comparable to those of a rotator cuff tear. The Apley scratch test is a way to evaluate internal rotation.[11]

Patients suffering from adhesive capsulitis may show signs of reduced glenohumeral range of motion (ROM) and discomfort during physical examination tests. When you are in pain, it is hard to do a full physical. Relative to the afflicted side, the range of motion (ROM) in two or more planes, both active and passive, is often significantly reduced. Loss of range of motion (ROM) often occurs in a predictable sequence, beginning with external rotation and progressing through abduction, internal rotation, and forward flexion.

In many cases, specialized tests that look for impingement (Neer and Hawkins) or biceps tendinopathy (Speed's test) come out positive. The diagnosis is mostly clinical, based on the patient's history and physical examination findings. Adhesive capsulitis cannot be diagnosed with any one set of laboratory tests. If there is reason to suspect a systemic illness is contributing to

the condition, more laboratory testing may be conducted. Adhesive capsulitis is usually best diagnosed clinically rather than with imaging. If another possible diagnosis is being examined or if underlying disease, such as a fracture, has to be evaluated, imaging procedures like a shoulder X-ray may be considered.

The injection test is a diagnostic tool that may be used when the underlying reason for shoulder pain cannot be identified. During the test, a local anesthetic injection of 5 milliliters of 1% lidocaine is usually administered into the subacromial area. Adhesive capsulitis patients report little relief from their discomfort or limited range of motion after receiving injections. People with subacromial pathologies, including rotator cuff tendinopathy or subacromial bursitis, may find that injections help with pain and increase their range of motion.

On MRI, you could see glenohumeral joint capsule thickness in the axillary pouch, subcoracoid fat triangle loss, glenohumeral ligament hypertrophy, and rotator interval synovitis. These are all hallmarks of adhesive capsulitis, but unfortunately, they do not indicate a disease. Arthrography may reveal joint capsule contracture if the usual axillary recess is absent.[12]

Imaging

Using imaging to diagnose adhesive capsulitis (AC) is still a contentious matter. The inclusion of a plain film negative for calcific tendinitis and osteoarthritis in the diagnostic criteria for frozen shoulder, as outlined in a highly referenced article, provides an illustrative scenario. Imaging tests have traditionally been reserved for unusual or resistant patients, and while there is no firm agreement on their function in the diagnostic process, they are often considered helpful but not essential. But many studies have looked at imaging modalities as a possible way to diagnose AC, and several radiological findings have been shown to be quite sensitive and specific for this disease. Traditional arthrography was useful for diagnosing AC in the past, but now MRI and US are more commonly used because they can directly show the thickening of the glenohumeral capsule and reveal typical pathological changes, while conventional arthrography can only show indirect findings like reduced capsular distension and extravasation of contrast agent. This method is becoming more attractive for the diagnosis and treatment of AC due to the fact that US may be used to target percutaneous interventions (such as hydrodistension and medication injection).[13,14]

Magnetic resonance imaging (MRI) is often considered the gold standard in the diagnosis of suspected AC because of its exceptional contrast resolution, which allows it to show the entire glenohumeral capsule and pericapsular soft tissue (Figure 2). Figure 3 shows the six main MRI findings that were recently identified as the most accurate for AC diagnosis in a recent meta-analysis. Here are some of the findings: The coracohumeral ligament thickens, the rotator interval is fat-obliterated, the inferior glenohumeral ligament is hyperintensified and thickens, and the axillary joint capsule and the rotator interval are enhanced with contrast.[13]

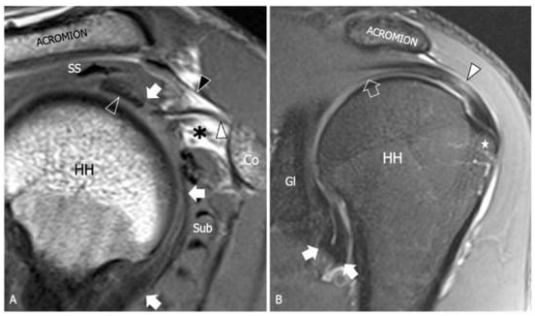

Figure 2. Glenohumeral Joint Capsule, Normal MRI Findings. Gl, scapular glenoid; HH, humeral head; star, greater tuberosity.In figure 2, (A) Sagittal tSE T1-weighted MRI image shows the joint capsule (arrow) as a structure of intermediate signal located deep to the rotator cuff tendons. Note the intra-articular part of the long head of the biceps tendon (outlined arrowhead) running on the inner surface of the capsule. The coracohumeral ligament (white arrowhead) is demonstrated as a thin and low-signal fibrillar structure running from the coracoid (Co) to the humeral head (HH). The subcoracoid fat triangle (asterisk) is a fat-filled space delimited by the coracohumeral ligament, the joint capsule, and the subscapularis (Sub) muscle. SS, supraspinatus muscle; black arrowhead, coracoacromial ligament. (B) Coronal tSE fat-suppressed T2-weighted MRI scan shows the inferior part of the joint capsule (arrow) as a low signal folding that delimits the axillary recess. Note the thin superior capsule (outlined arrow) located underneath the supraspinatus muscle and tendon (arrowhead).[15]

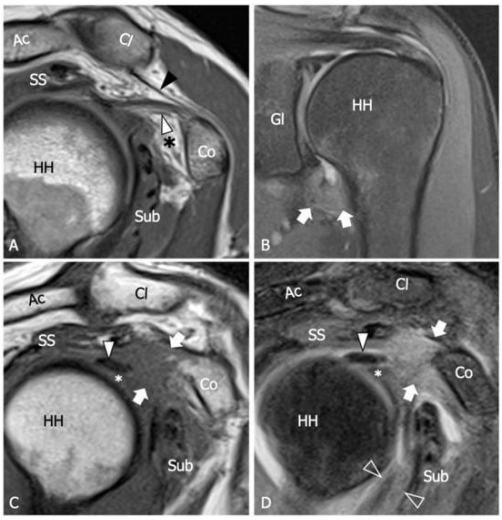

Figure 3. Adhesive Capsulitis, Spectrum of MRI Findings. (A) Sagittal tSE T1-weighted MRI scan from a 42-year-old woman with a three-month history of shoulder pain demonstrates mild thickening of the coracohumeral ligament (white arrowhead) and initial effacement of the subcoracoid fat triangle (asterisk) by hypointense synovium. Black arrowhead, coracoacromial ligament. (B) Coronal tSE proton density MRI scan from a 68-year-old woman with recent onset of pain and progressive limitation of glenohumeral ROM shows a marked thickening of the inferior capsule (arrows), which appears edematous and demonstrates increased signal intensity in fluid-sensitive sequences. (C) Sagittal tSE T1-weighted and (D) Sagittal tSE fat-suppressed T2-weighted MRI images from a 70-year-old male with a one-year history of severe limitation of active and passive shoulder motion demonstrate complete obliteration of the subcoracoid fat triangle by synovial tissue (arrows), which is also extended underneath the long head of the biceps tendon (arrowhead) in the area of the pulley (asterisk). The coracohumeral ligament appears embedded by the synovium. Note severe thickening and hyperintensity of the anteroinferior capsule (outlined arrowhead). Ac, acromion; Cl, clavicle; Co, coracoid; SS, supraspinatus; Sub, subscapularis; GL, scapular glenoid; HH, humeral head.[15]

Multiple studies have shown that thickening and hyperintensity of the inferior shoulder capsule are hallmarks of AC. For the hyperintensity in the axillary pouch/inferior glenohumeral ligament complex, MRI using non-arthrography T2-weighted fat-suppressed sequences has a high sensitivity (85.3-88.2%), and a kappa value of 0.85 suggests that there is minimal interobserver variability. A preliminary MRI investigation using a limited sample size found that joint capsule measures greater than 4 mm on T1 oblique coronal MR images are associated with a 70% sensitivity and a 95% specificity for the diagnosis of AC. This discovery is related to thin capsules. According to research by Jung et al. using conventional MRI, the most accurate way to diagnose AC was by measuring the thickness of the axillary recess capsule on T1 oblique coronal images. This threshold value of 4.5 mm had a sensitivity of 91%, specificity of 90%, and overall accuracy of 90%. Another study found that the thickness of the axillary recess was associated with the clinical stage. The hyperintense capsule signal on proton density sequences collected with conventional MRI was similarly statistically linked to Stage 2 and this clinical stage, according to Sofka et al., who found an average axillary pouch thickness of 7.5 mm. There are a variety of risks and complications that should be considered, including the potential for bleeding and septic arthritis, even if magnetic resonance angiography (MRA) can estimate inferior capsular thickness more precisely. Furthermore, a recent meta-analysis found no significant difference in the sensitivity and specificity of MRI and direct MRI for diagnosing inferior glenohumeral thickness. Additional findings that can be detected by magnetic resonance angiography include contrast agent leakage anterior to the medial margin of the scapula, decreased distension of the axillary recess, pseudo-synovitis over the cranial border of the subscapularis tendon and the biceps anchor, and widening of the subscapular recess (Figure 4). Contrast agent intra-articular injections, however, do not seem to be necessary for individuals with AC.[15–17]

Similarly, there is some disagreement about whether or not intravenous gadolinium is useful for assessing vascularization and thickness of the joint capsule. While Ahn et al. found no association between axillary recess capsule enhancement and forward elevation, external, or internal rotation limitation, they did find a statistically significant positive linear correlation

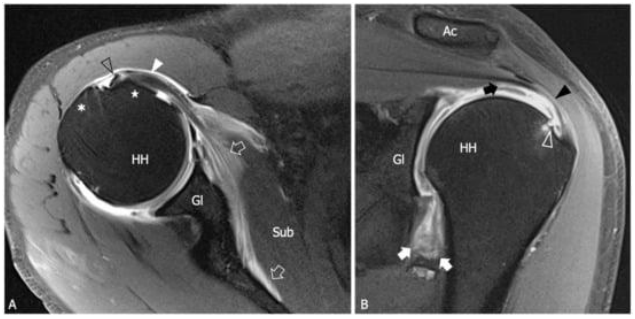

Figure 4. Adhesive Capsulitis in a 45-year-old Man with Severe Limitation of the Glenohumeral ROM after a Trauma who was Submitted for an MRA for a Suspected Labral Tear. (A) Axial and (B) Coronal tSE fat-suppressed T1-weighted MR images obtained after intraarticular injection of gadolinium demonstrate anterior extravasation of the contrast medium (outlined arrows) into and underneath the subscapularis muscle (Sub) as a consequence of capsular stiffness and fissuration. Note the abnormally low distension of the axillary recess (arrows) and its markedly thickened walls. In (B) a partial thickness tear (white outlined arrowhead) of the articular side of the supraspinatus tendon (black arrowhead) is also evident. In effect, the tear involves both the inner fibers of the supraspinatus and the joint capsule, which are merged at this level to form the superior complex. As a consequence, note the superior migration of the contrast outside the joint cavity (black arrow). Black outlined arrowhead, long head of the bicep tendon; white arrowhead, subscapularis tendon; asterisk greater tuberosity; star, lesser tuberosity; HH, humeral head; Gl, glenoid; Ac, acromion.[15]

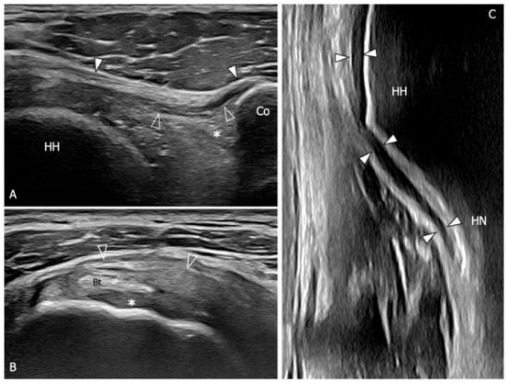

Figure 5. Glenohumeral Joint Capsule and Pericapsular Ligaments, Normal US Findings. (A) Oblique transverse 18–5 MHz US image shows the normal thin and fibrillar appearance of the coracohumeral ligament (outlined arrowheads), which is demonstrated connecting the coracoid (Co) and humeral head (HH) in a deeper position respective to the coracoacromial ligament (arrowheads). Note the homogeneous and hyperechoic appearance of the subcoracoid fat (asterisk). (B) Short-axis 18–5 MHz US image shows the distal part of the coracohumeral ligament (outlined arrowheads) in the area of the rotator interval and the biceps pulley (asterisk). Bt, long head of the biceps tendon. (C) Longitudinal 18–5 MHz US obtained by orienting the probe parallel to the humerus in the axillary region shows the inferior capsule (arrowheads) overlying the humeral head (HH) and folding over the humeral neck (HN).[15]

between pain intensity, joint capsule thickness, and axillary recess capsule enhancement grade in AC patients. Even while the contrast agent seemed to boost the reader's confidence in measuring the joint capsule after intravenous injection, another study discovered no significant differences in the diagnosis of AC between regular MRI and gadolinium-enhanced MRI. While MRA and MRI with intravenous contrast injection may provide additional information regarding the capsule's condition and enhance the reader's confidence in diagnosing AC, conventional MRI without contrast administration is recommended owing to its lesser invasiveness and expense.[18]

Recent developments in high-frequency transducers and technological advancements in the United States have made it possible to conduct detailed evaluations of the glenohumeral joint capsule and pericapsular ligaments in isolation from the surrounding soft tissues (Figure 5). A large body of research confirms that ultrasonography (US) is a very reliable tool for diagnosing AC. In a recent meta-analysis, it was shown that the US had a combined sensitivity of 88% (95% CI: 74-95) and specificity of 96% (95% CI: 88-99) when assessing for inferior capsule and coracohumeral thickness, rotator interval abnormalities, and limited range of motion (Figure 6).[19–21] According to the study conducted

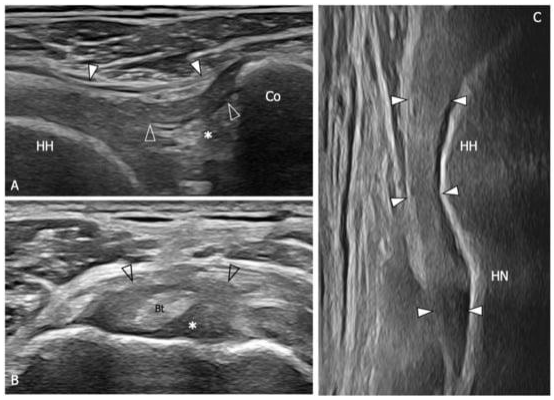

Figure 6. US Findings in a 53-year-old Woman with Adhesive Capsulitis. (A) an Oblique transverse 18–5 MHz US image demonstrates the markedly thickened coracohumeral ligament (outlined arrowheads), which has lost the normal fibrillar echotexture and appears homogeneously hypoechoic due to fibrotic changes and degeneration of the fibers. Note the presence of hypoechoic synovial tissue in the subcoracoid triangle (asterisk). Arrowheads, coracoacromial ligament. (B) Short-axis 18–5 MHz US evidences the thickening and fibrotization of the coracohumeral ligament (arrowheads) and the biceps pulley (asterisks) in the rotator interval. (C) Longitudinal 18–5 MHz US image shows a significant thickening of the inferior capsule (arrowheads). HH, humeral head; Co, coracoid; Bt, long head of the biceps tendon; HN, humeral neck.[15]

by Michelin et al., axillary pouch thickness was measured by ultrasonography to be 4 mm in patients with AC and 1.3 mm in those without symptoms. Later work by Kim confirmed that the US may detect abnormal capsule thickening in the axillary recess in people with unilateral AC. After measuring the capsule at its widest point, which encompasses both the humeral and glenoid layer sides (P<0.001), the researchers discovered a mean value of 4.4 mm for the injured shoulder and 2.2 mm for the intact shoulder.[23] The same study demonstrated a correlation between the MRI and US assessments of capsule thickness (P<0.001, r = 0.83). The axillary pouch cutoff value of 4 mm was associated with a sensitivity of 93.8% and a specificity of 98% for AC in related research. In patients with suggestive symptoms but an axillary recess thickness below 4 mm, the authors suggested that a 60% difference between the afflicted and unaffected sides might help disclose this illness.[23]

Management

Non-Surgical Treatment

Adhesive capsulitis recovers on its own in about 18-30 months in the majority of patients. Reducing symptoms and increasing range of motion are the primary goals of treatment. When it comes to therapy management, there is a lack of research. Some effective treatments are as follows[24]:

- Initially, nonsteroidal anti-inflammatory drugs (NSAIDs) may help manage pain. Evidence for the efficacy of physical therapy is weak, but some treatments can help patients in the recovery process. There is modest evidence that stretching, gradual weight bearing, and light range-of-motion exercises can reduce discomfort and improve function. However, overly intense rehabilitation can worsen symptoms, so it is best to minimize it. To ensure that treatment is well tolerated and does not worsen the disease, patients and their doctors should approach it with caution and evaluate the response regularly. The attached capsule is stretched and physiological accessory movements are improved using joint mobilization methods including glides and traction. If you want to do traction, you should move one joint surface away from the other, and if you want to do glides, you should move one joint surface parallel to the other.[25]
- For temporary pain relief and enhanced range of motion and function, oral corticosteroids are a therapy option to consider. Oral steroids may have beneficial effects, but they usually only last a few weeks. Before prescribing them, doctors should think carefully about the dangers and advantages of using these steroids.
- Steroid injections into the joint have shown promise in enhancing function, decreasing discomfort, and expanding range of motion (ROM). Keep in mind, nevertheless, that the benefits of injectable steroids do not last forever. Both the amount of shots and the methods utilized to administer them

might differ. Twenty to sixty milligrams of Triamcinolone is the typical dosage of this steroid. Injections of corticosteroids are often used in conjunction with a local anaesthetic. This is why medical professionals administering steroid injections need to be alert to the possibility of adverse consequences. Injecting patients early in the course of their illness may improve their chances of a good result. If necessary, it may be necessary to provide several injections to alleviate symptoms.[26]

- Bastille of the suprascapular nerve A technique called suprascapular nerve block (SNB) was first reported in 1941. Its goal is to obstruct the branches of the suprascapular nerve that go to the glenohumeral joint, specifically at the scapular notch. For a total of two to four treatments, you might repeat the block twice a week. When compared to intra-articular corticosteroid injection, a previous study indicated that SNB was a safer, more effective, and more beneficial treatment option. Three weekly SNBs with bupivacaine or placebo were the subjects of a randomized controlled trial. Although there was no increase in range of motion, SNB did provide considerable pain alleviation. Another case report suggested using ultrasound to guide the insertion of a perineural catheter, which would allow for continuous SNB.[26]
- The glenohumeral capsule is injected with a mixture of saline and steroids in hydrodilatation, a therapeutic method that promotes capsule dilation. There has been some evidence that this therapy method may alleviate pain and improve range of motion and function in the near term. Hydro-dilatation and intra-articular steroid injection do not seem to vary much in terms of result, according to the available research.[27]
- For instances of adhesive capsulitis that have not responded to conservative therapies, the next step is to do the manipulation while under anaesthesia. Humerus fractures are possible outcomes. The technique entails delicately moving the shoulder joint in different directions. A little lever arm is placed near the patient's shoulder to support their arm, and their shoulder is delicately moved in several directions, including flexion, abduction, external rotation, and 90° abduction. If you find more resistance, do not apply force. Another option for reducing inflammation during the surgery is to inject a mixture of triamcinolone and bupivacaine.[28]

Surgical Treatment

It is important to identify an alternative treatment strategy in case the illness worsens or development stops.[3,12] When non-surgical treatments, such as prednisone or nonsteroidal anti-inflammatory drugs (NSAIDs), fail to alleviate symptoms after glenohumeral or subacromial injections, or when physical therapy fails to provide the desired results, surgical intervention may be considered. Conversely, surgical procedures are not always appropriate. Some examples of these situations include a lack of response to nonsteroidal anti-inflammatory drugs (NSAIDs) or steroids; no prior conservative treatment; an active infection; the presence of a concurrent shoulder cancer; or a problem with the cervical spine's neurological system.

- When other methods fail, the surgeon may use arthroscopic capsular release. It is advisable to see an orthopedic surgeon if symptoms do not resolve after 10 to 12 months of conservative treatment. To improve mobility, surgery requires the release of certain components within the joint capsule. It may be difficult to penetrate the thick, narrowed joint capsule using conventional posterior or lateral portal entry methods; however, the following are released: the rotator cuff interval, coracohumeral ligaments, middle glenohumeral ligaments, anterior capsule, and posterior capsule. The proximity of the inferior capsule to the axillary nerve makes it an ideal target for direct manipulation during adhesiolysis. During surgery, every effort is made to ensure that adjacent structures are not damaged. Examination of the subacromial space after release, and debridement of inflammatory tissue and bursae may be performed if necessary. Injecting the shoulder joint with a mixture of triamcinolone and bupivacaine helps reduce inflammation. Early range-of-motion exercises, both passive and dynamic, are started after surgery. There is no conclusive evidence in the literature comparing the clinical effectiveness of arthroscopic capsular release with manipulation under anesthesia.[28]
- Open capsular release might be an option for

patients with conditions such as posttraumatic or postsurgical adhesive capsulitis, severe adhesions, contractures that prevent arthroscopic surgery, and brain injuries or strokes. Open release necessitates a larger incision to swiftly access and remove the thicker and more compressed joint capsule. The morbidity associated with the open operation is greater than that of the arthroscopic capsular release treatment.[29]

Conflict of Interest

The authors declare no conflicts of interest.

References

1. McKean D, Yoong P, Brooks R, Papanikitas J, Hughes R, Pendse A, et al. Shoulder manipulation under targeted ultrasound-guided rotator interval block for adhesive capsulitis. Skeletal Radiol. 2019;48:1269-74. doi:10.1007/s00256-018-3105-3
2. Allen GM. The diagnosis and management of shoulder pain. J Ultrason. 2018;18(74):234-9. doi:10.15557/JoU.2018.0034
3. Georgiannos D, Markopoulos G, Devetzi E, Bisbinas I. Suppl-1, M2: Adhesive Capsulitis of the Shoulder. Is there Consensus Regarding the Treatment? A Comprehensive Review. Open Orthop J. 2017;11:65-76. doi:10.2174/1874325001711010065
4. Le HV, Lee SJ, Nazarian A, Rodriguez EK. Adhesive capsulitis of the shoulder: review of pathophysiology and current clinical treatments. Shoulder Elbow. 2017;9(2):75-84. doi:10.1177/1758573216676786
5. Small KM, Adler RS, Shah SH, Roberts CC, Bencardino JT, Appel M, et al. ACR Appropriateness Criteria® shoulder pain-atraumatic. J Am Coll Radiol. 2018;15(11):S388-402. doi:10.1016/j.jacr.2018.09.032
6. Kingston K, Curry EJ, Galvin JW, Li X. Shoulder adhesive capsulitis: epidemiology and predictors of surgery. J Shoulder Elbow Surg. 2018;27(8):1437-43. doi:10.1016/j.jse.2018.04.004
7. Schiefer M, Teixeira PF, Fontenelle C, Carminatti T, Santos DA, Righi LD, et al. Prevalence of hypothyroidism in patients with frozen shoulder. J Shoulder Elbow Surg. 2017;26(1):49-55. doi:10.1016/j.jse.2016.04.026
8. Angelo JMS, Taqi M, Fabiano SE. Adhesive Capsulitis. StatPearls. 2023.
9. Yuan X, Zhang Z, Li J. Pathophysiology of adhesive capsulitis of shoulder and the physiological effects of hyaluronan. Eur J Inflamm. 2017;15(3):239-43. doi:10.1177/1721727X17747439
10. Date A, Rahman L. Frozen shoulder: overview of clinical presentation and review of the current evidence base for management strategies. Future Sci OA. 2020 ;6(10):FSO647. doi:10.2144/fsoa-2020-0145
11. Ramirez J. Adhesive capsulitis: diagnosis and management. Am Fam Physician. 2019;99(5):297-300.
12. Suh CH, Yun SJ, Jin W, Lee SH, Park SY, Park JS, et al. Systematic review and meta-analysis of magnetic resonance imaging features for diagnosis of adhesive capsulitis of the shoulder. Eur Radiol. 2019;29:566-77. doi:10.1007/s00330-018-5604-y
13. Papalexis N, Ponti F, Rinaldi R, Peta G, Bruno R, Miceli M, Battaglia M, Marinelli A, Spinnato P. Ultrasound-guided treatments for the painful shoulder. Curr Med Imaging. 2022;18(7):693-700. doi:10.2174/1573405617666211206112752
14. Rangan A, Brealey SD, Keding A, Corbacho B, Northgraves M, Kottam L, et al. Management of adults with primary frozen shoulder in secondary care (UK FROST): a multicentre, pragmatic, three-arm, superiority randomised clinical trial. Lancet. 2020;396(10256):977-89. doi:10.1016/S0140-6736(20)31965-6
15. Picasso R, Pistoia F, Zaottini F, Marcenaro G, Miguel-Pérez M, Tagliafico AS, et al. Adhesive Capsulitis of the Shoulder: Current Concepts on the Diagnostic Work-Up and Evidence-Based Protocol for Radiological Evaluation. Diagnostics. 2023;13(22):3410. doi:10.3390/diagnostics13223410
16. Jung JH, Kim DH, Yi J, Kim DH, Cho CH. Determination of magnetic resonance imaging criteria for diagnosis of adhesive capsulitis. Rheumatol Int. 2019;39(3):453-60. doi:10.1007/s00296-018-04238-9
17. Suh CH, Yun SJ, Jin W, Lee SH, Park SY, Park JS, et al. Systematic review and meta-analysis of magnetic resonance imaging features for diagnosis of adhesive capsulitis of the shoulder. Eur Radiol. 2019;29:566-77. doi:10.1007/s00330-018-5604-y
18. Erber B, Hesse N, Goller S, Gilbert F, Ricke J, Glaser C, et al. Diagnostic performance and interreader agreement of individual and combined non-enhanced and contrast-enhanced MR imaging parameters in adhesive capsulitis of the shoulder. Skeletal Radiol. 2024;53(2):263-73. doi:10.1007/s00256-023-04391-8
19. Wu H, Tian H, Dong F, Liang W, Song D, Zeng J, et al. The role of grey-scale ultrasound in the diagnosis of adhesive capsulitis of the shoulder: a systematic review and meta-analysis. Med Ultrason. 2020;22(3):305-12. doi:10.11152/mu-2430
20. Al Khayyat SG, Falsetti P, Conticini E, Frediani B, Galletti S, Stella SM. Adhesive capsulitis and ultrasound diagnosis, an inseparable pair: A novel review. J Ultrasound. 2023;26(2):369-84. doi:10.1007/s40477-022-00725-9
21. Fields BK, Skalski MR, Patel DB, White EA, Tomasian A, Gross JS, et al. Adhesive capsulitis: review of imaging findings, pathophysiology, clinical presentation, and treatment options. Skeletal Radiol. 2019;48:1171-84. doi:10.1007/s00256-018-3139-6
22. Kim DH, Cho CH, Sung DH. Ultrasound measurements of axillary recess capsule thickness in unilateral frozen shoulder: study of correlation with MRI measurements. Skeletal Radiol. 2018;47:1491-7. doi:10.1007/s00256-018-2959-8
23. Stella SM, Gualtierotti R, Ciampi B, Trentanni C, Sconfienza LM, Del Chiaro A, et al. Ultrasound features of adhesive capsulitis. Rheumatol Ther. 2022;9(2):481-95. doi:10.1007/s40744-021-00413-w
24. Koorevaar RC, van't Riet E, Ipskamp M, Bulstra SK. Incidence and prognostic factors for postoperative frozen shoulder after shoulder surgery: a prospective cohort study. Arch Orthop Trauma Surg. 2017;137:293-301. doi:10.1007/s00402-016-2589-3
25. Nakandala P, Nanayakkara I, Wadugodapitiya S, Gawarammana I. The efficacy of physiotherapy interventions in the treatment of adhesive capsulitis: A systematic review. J Back Musculoskelet Rehabil. 2021;34(2):195-205. doi:10.3233/BMR-200186
26. Haque R, Baruah RK, Bari A, Sawah A. Is suprascapular nerve block better than intra-articular corticosteroid injection for the treatment of adhesive capsulitis of the shoulder? A Randomized Controlled Study. Ortop Traumatol Rehabil. 2021;23(3):157-65. doi:10.5604/01.3001.0014.9152
27. Poku D, Hassan R, Migliorini F, Maffulli N. Efficacy of hydrodilatation in frozen shoulder: a systematic review and meta-analysis. Br Med Bull. 2023;147(1):121-47. doi:10.1093/bmb/ldad018
28. Xiao Y, Tang H, Meng J, Wu Y, Liu W, Liu P, et al. Similar outcomes between arthroscopic capsular release and manipulation under anaesthesia for frozen shoulder: A meta-analysis. Asian J Surg. 2024. doi:10.1016/j.asjsur.2024.03.055
29. Sarkar A. Adhesive capsulitis: a review of current clinical treatments: Our purpose is to review the evidence for both surgical and non-surgical management of adhesive capsulitis. Sustainability, Agri, Food and Environmental Research-DISCONTINUED. 2023;12. doi:10.7770/safer-V13N2-art3010