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Current review of adhesive capsulitis

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Adhesive capsulitis, also known as frozen shoulder, is a common condition involving scapulohumeral pain and loss of motion. This condition was termed "peri-arthritis scapulohumerale" by Duplay in 1896.[36] Codman,[29] in 1934, characterized the diagnosis of "frozen shoulder" as a condition characterized by pain and reduced range of motion in the affected shoulder. Neviaser,[75] in the pre-arthroscopic era, subsequently used the term "adhesive capsulitis" to describe the findings of chronic inflammation and fibrosis of the joint capsule, although arthroscopic examination would support the term "fibrotic capsulitis" with the absence of adhesions. The current consensus definition of the American Shoulder and Elbow Surgeons is "a condition of uncertain etiology characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder."[69,113]

Despite the large number of patients affected by this condition, our understanding of the condition is limited. In the past, the term "frozen shoulder" has often been used to describe other shoulder conditions such as subacromial bursitis and calcific tendonitis. Neviaser and Neviaser[77] described the term "frozen shoulder" as a "waste-can diagnosis," because it was often overused and misapplied to patients with a stiff and painful shoulder. Conditions such as calcific tendonitis, bicipital tenosynovitis, glenohumeral and acromioclavicular arthritis, and tears of the rotator cuff can lead to a stiff and painful shoulder,[76] causing apparent limitation of active range of motion, but they lack true capsular contracture and restriction in passive range of motion and, therefore, should not be labeled as adhesive capsulitis. Accurate diagnosis is essential because of dissimilar treatment approaches for these separate entities.

This misunderstanding and confusion over the definition and diagnosis of adhesive capsulitis reflected our poor grasp of the etiology, diagnosis, and management of this condition. Our understanding of this disease has slowly progressed, and a number of surgical and nonsurgical approaches to treating this often obstinate disease have been investigated. This article aims to review the natural history, pathogenesis, diagnosis, and management of adhesive capsulitis, with emphasis on the most recent evidence regarding etiology and treatment.

Epidemiology and classification

The true incidence of adhesive capsulitis is difficult to assess because many individuals who have the disease will not seek medical attention because of the vague character and insidious onset of the disease. It is a common condition that is often quoted by many studies to affect approximately 2% to 5% of the general population,[12,46,110] this incidence, however, is likely inflated, because many studies include populations with specific comorbid conditions with a higher incidence of frozen shoulder than the general population.[12,110] It most commonly affects women aged between 40 and 60 years.[12,46] Frozen shoulder often presents bilaterally and commonly affects the contralateral side years after onset of symptoms in the first shoulder,[12,46,90,96] but it does not affect the same shoulder twice. The condition has not been reported to have a predilection for race.

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Adhesive capsulitis is usually classified as primary or secondary. Patients are classified as having primary or idiopathic adhesive capsulitis if no findings on history or examination explain the onset of disease. These cases may be related to immunologic, biochemical, or hormonal imbalances. Secondary adhesive capsulitis develops from known causes of stiffness and immobility, such as previous shoulder trauma or surgery, and may represent an entirely different condition. Many conditions and procedures cause the upper extremity to be in a dependent position for extended periods of time, but it is unknown whether development of frozen shoulder in many of these cases is related to pain and immobility.

Frozen shoulder commonly occurs in patients with certain medical comorbidities and is often correlated with increased pain and dysfunction with these comorbid medical factors.[110] Most well-known is the strong association between diabetes and adhesive capsulitis. Bridgman[12] first described this association after observing a 10.8% incidence among 800 diabetic patients and only a 2.3% incidence in 600 nondiabetic patients; the rate of bilateral frozen shoulder was high among diabetic patients, and subsequent studies have supported this observation.[2,40,102] Various cardiac, endocrine, and neurologic disorders have similarly been found to have a higher incidence than in the general population (Table I).

Whether a genetic predisposition for frozen shoulder exists is controversial. Twin studies have shown that adhesive capsulitis occurs 2 to 3 times more frequently than by chance,[44] but this result may be because of individual-specific environmental factors rather than a true genetic component. Studies on human leukocyte antigen B27 (HLA-B27) prevalence in patients with frozen shoulder have been mixed. Interestingly, there are a number of studies reporting a strong association between adhesive capsulitis and Dupuytren disease,[19,97] which is also believed to have a heritable component. Although Smith et al[97] reported that Dupuytren disease is 8.27 times more common in patients with frozen shoulder compared with the general population, this strong association is not well understood.

Anatomy and pathogenesis

The underlying etiology and pathophysiology of adhesive capsulitis are poorly understood. Basic science studies attempting to clarify the pathogenesis of frozen shoulder are limited, but the role of various biologic factors,[19,21,92] mechanical stress,[55] and neovascularization[19,94] has been supported. Most information stems from recalcitrant cases requiring arthroscopic and open treatment.

Neviaser[75] initially used the term ''adhesive capsulitis'' based on his findings during open surgery of capsular and synovial inflammation and adhesions, leading to adherence of the axillary fold to itself and the anatomic neck of the humerus. More recent evidence supports thickening and contracture of the inferior capsule rather than adherence of the axillary fold.[108] Anatomically, contracture of the rotator interval, coracohumeral ligament, and anterior capsule restricts movement of the shoulder. Neer et al[74] have previously shown that the coracohumeral ligament was contracted and required release to restore restricted external rotation of the shoulder, and one clinical study has shown that release of this ligament restored external rotation in all patients with adhesive capsulitis.[84] Measurements of shoulders with and without adhesive capsulitis show significantly different rotator interval dimensions[56] and thickening of the axillary pouch.[30]

The disease is thought to be a combination of synovial inflammation and capsular fibrosis. Capsular and synovial biopsy specimens from patients with adhesive capsulitis have suggested that cytokines such as transforming growth factor β, platelet-derived growth factor, interleukin 1β, and tumor necrosis factor α are involved in synovial hyperplasia and capsular fibrosis.[92] Increased neovascularization and a strong immunostaining to vascular endothelial growth factor in the synovium of diabetic frozen shoulders have been shown,[94] and vascular endothelial growth factor has been implicated to play an important role in the pathogenesis of frozen shoulder. Histologic and immunohistochemical examination of adhesive capsulitis tissue shows a vascular, collagenous tissue with high cellularity, composed primarily of fibroblasts and myofibroblasts. The active fibroblastic process that occurs in adhesive capsulitis is similar to that which occurs in Dupuytren disease of the hand.[19] Interestingly, patients treated with a synthetic matrix metalloproteinase inhibitor for gastric carcinoma had high rates of frozen shoulder and Dupuytren disease,[51] suggesting that abnormal expression of matrix metalloproteinase inhibitors is involved in the pathogenesis of the disease.[21]

The biochemical rationale for the high incidence of frozen shoulder in patients with diabetes is speculative. It is thought that excessive glucose concentration in diabetic patients can lead to a faster rate of collagen glycosylation and cross-linking in the shoulder capsule,[13,14] restricting shoulder range of motion. This collagen cross-linking may also be responsible for the higher incidence of Dupuytren contractures and trigger finger in diabetic patients.

Natural history

Frozen shoulder is a painful, debilitating disease with an often protracted course. The natural history of the frozen shoulder has been characterized by many authors but remains controversial. Although it has been reported that the majority of patients will have complete resolution of the disease,[71] many others report long-term pain and residual loss of motion.[8,46,80,96] Miller et al[71] followed 50 patients for 10 years, with each patient receiving moist heat and

Table I Conditions associated with adhesive capsulitis

Condition	Author	Description
Trauma/surgical		
Upper extremity trauma	Hand et al[47]	22% of patients report minor trauma to limb before symptoms
Radial neck dissection	Patten and Hillel[85]	31 of 44 patients who underwent neck dissections
Hormonal		
Diabetes	Bridgman[12]	Incidence of 10.8% in diabetic patients and 2.3% in nondiabetic patients
	Thomas et al[102]	Incidence of 4.3% in diabetic patients and 0.5% in nondiabetic patients
	Arkkila et al[2]	Incidence of 10.3% in patients with type I diabetes and 22.4% in patients with type II diabetes
ACTH deficiency	Choy et al[27]	Case of bilateral frozen shoulder in isolated ACTH deficiency
Thyroid disease	Wohlgethan[109]	Case of bilateral frozen shoulder in hyperthyroidism
	Bowman et al[10]	Case of bilateral frozen shoulder in hypothyroidism
	Cakir et al[22]	10.9% incidence in patients with thyroid disease
Cardiac		
Cardiac disease	Tuten et al[103]	3.3% incidence in male cardiac surgery patients (7 of 214)
	Bridgman[12]	10 of 14 nondiabetic patients with frozen shoulder had ischemic heart disease or hypertension
Diabetes and cardiac disease	Boyle-Walker et al[11]	Diabetes and heart disease more prevalent in patients diagnosed with adhesive capsulitis compared with controls
Neurologic		
Parkinson	Riley et al[91]	Incidence of 12.7% in Parkinson patients and 1.7% in controls
Stroke	Lo et al[63]	50% incidence in patients with hemiplegic shoulder pain after first stroke
Neurosurgery	Bruckner and Nye[15]	25.3% incidence in neurosurgical patients
Aneurysm surgery	Tanishima and Yoshimasu[101]	41% of patients undergoing acute aneurysm surgery
Other		
Malignancy	Gheita et al[42]	9 of 60 patients with malignant disease
Hyperlipidemia	Bunker and Esler[20]	Higher triglyceride and cholesterol levels in frozen shoulder patients vs controls
	Hand et al[46]	17% had hypercholesterolemia
Drug related	Hutchinson et al[51]	12 patients treated with matrix metalloproteinase inhibitor for gastric carcinoma
	Grasland et al[43]	8 patients treated with protease inhibitor (indinavir)
	De Ponti et al[32]	6 patients treated with antiretrovirals (stavudine, lamivudine, indinavir)
	Bodor and Montalvo[9]	2 cases after influenza and pneumococcal vaccine
	Freiss et al[41]	2 cases after fluoroquinolones
Dupuytren	Smith et al[97]	52% of patients with frozen shoulder were found to have Dupuytren
	Degreef et al[33]	45% of patients with Dupuytren diagnosed with frozen shoulder

ACTH, adrenocorticotropic hormone, *MUA*, manipulation under anesthesia, *ROM*, range of motion.

anti-inflammatory medication and undergoing a home rehabilitation protocol; pain resolved and range of motion significantly improved in all patients. However, a similar study by Shaffer et al[96] with a mean of 7 years' follow-up on 62 patients reported that 50% of patients still had residual pain and/or loss of motion at the time of latest follow-up, challenging Codman's statement that ''recovery is always sure and may be confidently expected'' in patients afflicted with frozen shoulder.[29] The results of these studies are difficult to interpret and compare, however, because of the differences in criteria for what constitutes restriction in range of motion. Although at first glance, ''average range of motion markedly increased without exception'' as stated by Miller et al may seem contradictory to the description of ''residual restriction of motion in at least one plane'' of Schaffer et al, these may in fact represent a similar outcome. A number of studies do acknowledge long-term residual motion restriction and symptoms, but this may not always be within the functional limitations of the patient. Commonly, external rotation is the predominant plane of motion restricted at latest follow-up, which may not always result in functional impairment. For a lower-demand patient, decreased range of motion may not limit activities of daily living.

In describing the natural history of frozen shoulder, clinicians refer to the continuum of 3 phases (Fig. 1)

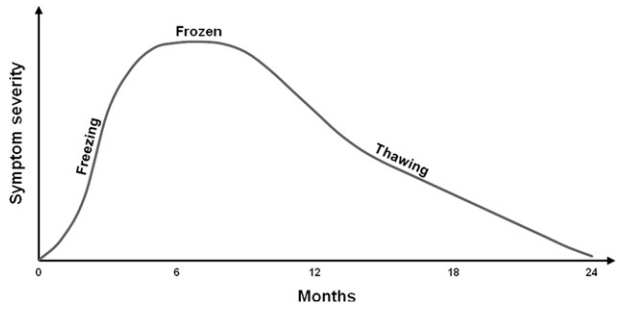

Figure 1 Continuum of phases in adhesive capsulitis.

originally described by Reeves[90] in 1975. Neviaser and Neviaser[77] also described a similar continuum of frozen shoulder based on physical and arthroscopic examination, with subsequent studies by Hannafin and Chiaia[48] providing histologic progression throughout the various stages. The disease begins in the painful, or ''freezing,'' phase, in which the patient has progressive, involuntary stiffness. Patients first note pain with activities and may attribute this to a trivial injury. Typically, pain precedes the restriction in motion, but in some instances, loss of range of motion may be the first symptom. This phase has been documented to last between 10 and 36 weeks. The freezing phase is followed by the stiff, or ''frozen,'' phase, in which the pain gradually decreases but there continues to be a reduction in the range of motion. This phase can last anywhere from 4 to 12 months. Finally, the recovery, or ''thawing,'' phase involves the gradual spontaneous improvement of shoulder mobility and function over the course of 5 to 26 months, with a longer freezing phase associated with a longer thawing phase. The full duration of the disease was described by Reeves to last anywhere from 1 to 3.5 years, with a mean of 30 months, but subsequent reports have described a longer and more protracted course in many patients. Frozen shoulder often subsequently affects the contralateral shoulder between 6 months to 7 years after initial onset of symptoms of the first shoulder.[90]

Clinical evaluation

The majority of patients with adhesive capsulitis do not seek medical attention until weeks to months after the onset of pain and stiffness. Because the onset of the disease is usually gradual rather than acute, patients will not see a physician until activities of daily living become severely limited. Underlying conditions such as glenohumeral and acromioclavicular arthritis, rotator cuff disease, and cervical and peripheral radiculopathy must be evaluated for in patients with shoulder pain and stiffness, although only osteoarthritis, locked posterior dislocation, and frozen shoulder can lead to limitations in passive range of scapulohumeral motion. The criteria for inclusion in most studies of frozen shoulder include insidious onset, pain elicited by isolated scapulohumeral passive motion, night pain, painful restriction of passive scapulohumeral elevation to less than 100° and of external rotation to less than one half of normal, and a radiograph excluding other pathologic processes. The insidious onset of frozen shoulder is usually associated with primary rather than secondary frozen shoulder. In addition, frozen shoulder affects isolated scapulohumeral elevation, which normally is limited to about 120°; in affected patients, this elevation can be severely limited, often much less than the 100° cutoff frequently described in the literature.

The history and physical examination are essential to differentiating between the stiff and painful shoulder and the shoulder with true adhesive capsulitis. Patients will often describe an insidious onset of vague, dull pain at the deltoid insertion, a pain pattern that may be due to innervation of the joint capsule by the axillary nerve. Night pain is a very common feature, and sleeping on the affected shoulder is usually symptomatic. Painful and restricted elevation and external rotation are common. As the patient progresses from the freezing to frozen stage, the pain becomes more severe, and the restriction in elevation and rotation increases.

On examination, the patient will usually have tenderness at the deltoid insertion and over the anterior capsule and posterior capsule with deep palpation. In those with longstanding disease, increased compensatory scapulothoracic motion can create additional pain around the medial scapula. Most critical in the physical examination of the patient is the evaluation of passive range of motion. Forward flexion, abduction, and internal and external rotation should be assessed with control of scapulothoracic motion. This is often done in the supine position. True mechanical restriction with passive motion that occurs with capsular contraction is characterized by a firm, reproducible endpoint and must be differentiated from resistance due to pain. An intra-articular lidocaine injection can assist in differentiating limited motion due to pain versus contracture. Monitoring shoulder motion over the course of the disease can determine the efficacy of treatment, and if surgical release is eventually needed, the pattern of motion loss can assist in determining the location of contracture.

Radiographic findings

Adhesive capsulitis is primarily a clinical diagnosis. Radiographic studies are used to exclude other causes of shoulder pain and can be complementary in the diagnosis. Calcific tendonitis, neoplastic processes, impingement, and osteoarthritis of the glenohumeral and acromioclavicular joints can be assessed. Many reports have associated osteopenia of the proximal humerus with frozen shoulder.[66,81]

Arthrography of the shoulder can also be done to assess the volume of the joint; in frozen shoulder, the axillary fold

will be contracted, reducing the normal joint volume. The normal glenohumeral joint capacity is around 15 mL, and because of the decreased size of the axillary recess, a reduced volume can be an indicator of adhesive capsulitis. The severity of the shoulder range restriction may also be associated with reduction of joint volume.[65] A normal joint volume, however, cannot be used to rule out adhesive capsulitis, given that the presence of a concurrent full-thickness rotator cuff tear and flow of fluid into the subacromial space can result in a seemingly normal intra-articular volume; the volume of the inferior recess measured on magnetic resonance (MR) arthrography would still be diminished in these cases.

Currently, plain magnetic resonance imaging (MRI) and MR arthrography can provide reliable imaging indicators of adhesive capsulitis, and these findings on MRI have been shown to correlate well with surgical findings.[30,70,99] Potentially useful MR findings in frozen shoulder are thickening of the coracohumeral ligament and joint capsule in the rotator interval.[24,100] Capsular thickening of the axillary recess is often a useful sign,[38] although some studies in the radiologic literature do not support this.[24,67] There is often obliteration of the fat triangle, or subcoracoid fat, between the coracohumeral ligament and the coracoid process, often referred to as the ''subcoracoid triangle sign.''[70] Despite these potentially useful findings, plain MRI and MR arthrography are not initially indicated to diagnose frozen shoulder and should only be used to rule out other intra-articular pathology such as rotator cuff tears or early chondral damage.

Ultrasound has also been shown to have utility in diagnosing frozen shoulder.[50] Measurements of coracohumeral ligament thickness can be performed in the majority of shoulders, and thickening can be suggestive of adhesive capsulitis. The presence of a hypoechoic region with increased vascularity in the rotator interval correlates with the fibrovascular inflammatory tissue that is usually present and can provide early and accurate diagnosis.

Management of adhesive capsulitis

Treatment regimens for adhesive capsulitis include a trial of conservative therapy, followed by more invasive procedures for recalcitrant cases. The level of evidence of studies regarding the various treatments for frozen shoulder is limited, especially with regard to surgical treatments. Controlled prospective studies are difficult to design and carry out because of the difficulties in diagnosing the condition and because of the various stages of the disease at which patients present; therefore, the majority of studies are case series describing a single therapeutic approach. Often, study results describe outcomes at time points greater than 1 year, which clinically for patients is too long of a time period for relief of symptoms. The goal of therapy is to reduce pain and allow the patient to sleep shortly after choosing a particular intervention.

Conservative treatment

Codman[29] originally described a self-limited course of frozen shoulder, and many patients are treated with supervised neglect with analgesia, supervised physical therapy, and/or steroid injection. The majority will experience resolution when treated conservatively, but some patients may have long-term pain and restriction of motion several years after initial symptoms. A multitude of conservative treatment options exist, and all have been reported to have positive short-term results (Table II). Regardless of choice, a minimum of 6 months of supervised conservative treatment should be attempted before any other more invasive treatments are considered.

Although there is no literature to support the use of nonsteroidal anti-inflammatory drugs in the treatment of frozen shoulder, they are often prescribed in the early, inflammatory phases of the disease to provide short-term pain relief. Use of nonsteroidal anti-inflammatory drugs for frozen shoulder has never been shown to improve pain or function when compared with placebo. Oral corticosteroids can also be prescribed and have been shown to improve pain, especially night pain,[7] and range of motion in the short term.[7,17,18] Injection of corticosteroids into the glenohumeral or subacromial space is also commonly used and has been reported to have similar outcomes to physiotherapy alone and to more invasive measures such as manipulation and hydrodilatation.[53,93,104] The use of ultrasound or fluoroscopy to guide injections has been shown in multiple studies to improve the accuracy of these injections compared with a blind technique.[60,73,95,112]

It is widely accepted that physical therapy and stretching should be used in the conservative management of frozen shoulder. Simple home exercise programs with analgesia have been shown to be effective, whereas other clinicians suggest more intensive supervised physical therapy. A prospective study with 2-year follow-up compared intensive physical therapy including passive stretching and manual mobilization versus supportive therapy and exercises within pain limits, or ''supervised neglect.''[34] Those patients receiving intensive physical therapy reached a Constant score of 80 less often (63%) than those treated with supervised neglect (89%). Moist heat, anti-inflammatory medication, and a physician-directed rehabilitation program were shown to consistently result in resolution of symptoms.[71]

More invasive procedures such as hydrodilatation and nerve blockade have been supported.[23,31,54,89] First described by Andren and Lundberg,[1] hydrodilatation aims to rupture capsular contractures by distension of the joint with a large amount of normal saline solution while avoiding the complications of manipulation under

Table II Studies of conservative therapy for adhesive capsulitis

Author	Year	Technique	No. of patients	Follow-up	Results
Physiotherapy/home therapy					
Leung and Cheing[61]	2008	1. Stretching only 2. Superficial heating + stretching 3. Deep heating + stretching	10/group		Improvement of pain relief greater in deep than superficial heating
Miller et al[71]	1996	Home therapy with moist heat and anti-inflammatory medications	50	10 y	All patients regained motion and returned to activities of daily living without pain
Nicholson[79]	1985	1. Active only for 4 wk 2. Active + passive for 4 wk	20 active + passive 20 active only		Similar results between 2 groups
Supervised neglect					
Diercks and Stevens[34]	2004	1. Supportive therapy ("supervised neglect") 2. Intensive physical therapy	77	24 mo	Greater proportion had normal shoulder function with supervised neglect
Steroid injection					
Jacobs et al[53]	2009	1. Intra-articular steroids injection 2. MUA	53	2 y	No difference in outcomes
Buchbinder et al[16]	2004	1. Steroid + saline solution distension 2. Placebo distension	48	12 wk	Short-term efficacy of steroid injection
Hydrodilatation					
Tveita et al[104]	2008	1.Corticosteroids with hydrodilatation 2. Corticosteroids without hydrodilatation	76	6 wk	No difference in treatment groups
Quraishi et al[89]	2007	1. Hydrodilatation 2. MUA	20 hydrodilatation 18 MUA	6 mo	Hydrodilatation better than MUA
Piotte et al[86]	2004	1. Repeated distension arthrography 2. Home exercise	15		Decreasing effectiveness with number of distension arthrography
Bell et al[4]	2003	Hydrodilatation	109		33% still painful at 2 mo
Nerve blockade					
Dahan et al[31]	2000	1. Bupivacaine suprascapular nerve block 2. Saline solution control	34	1 mo	Effective in reducing pain but no difference in shoulder function
Jones and Chattopadhyay[54]	1999	1. Suprascapular nerve block 2. Intra-articular injection	30	12 wk	Faster pain resolution and restoration of range of motion
Other					
Buchbinder et al[17]	2006	Oral steroids	Cochrane review		Oral steroids provide short-term benefits (<6 wk) in pain and ROM

MUA, manipulation of anesthesia, *ROM*, range of motion, *DASH*, Disabilities of the Arm, Shoulder, and Hand, *VAS*, visual analogue scale, *SST*, Simple Shoulder Test, *ASES*, American Shoulder and Elbow Surgeons.

anesthesia. A recent study comparing hydrodilatation and manipulation under anesthesia showed that Constant scores at 6 months were higher and patients were more satisfied after hydrodilatation than manipulation.[89] Suprascapular nerve blockade has also been suggested to be an effective treatment,[16,31,54] but more supporting data from prospective studies are needed.

Closed manipulation

After 6 months of refractory pain and stiffness in patients with primary frozen shoulder, manipulation under anesthesia can be considered. In this procedure, the patient can be placed supine or in the seated beach-chair position, and the shoulder is gently passively stretched in forward flexion, abduction, and adduction while the scapula is being stabilized. With the elbow at a right angle, the upper arm is finally gently rotated through extremes of internal and external rotation by use of a short lever arm. Tearing of the contracted capsule may be palpated and even audibly confirmed by the physician. Closed manipulation should not be attempted in more resistant cases of post-traumatic and postsurgical frozen shoulder because of increased risk of fracture.

The results of manipulation have mostly been reported to be excellent (Table III),[35,39] but comparative studies have shown equivocal benefit when compared with hydrodilatation[89] or home exercise therapy.[58] A long-term follow-up of patients who underwent closed manipulation for frozen shoulder showed that the majority of patients achieved sustained improvements in range of motion at a mean of 15 years after manipulation.[39] However, a recent blinded, randomized, controlled trial comparing patients undergoing manipulation under anesthesia with a control group did not show a benefit to manipulation: improvement in the 2 groups was similar at all time points up to 12 months.[58] Of note, patients in this study were symptomatic for a mean of 7 months and had not received previous treatment at the time of diagnosis. Additional studies are needed to show whether manipulation under anesthesia has value in patients who have refractory primary frozen shoulder.[52]

A number of complications can occur as a result of excessively forceful manipulation, such as iatrogenic fractures of the humerus, glenohumeral dislocation, rotator cuff and labral tears, brachial plexus injuries, and hemorrhagic effusions and hematomas, which can be detrimental to articular cartilage. A study in which arthroscopy was performed after manipulation after anesthesia showed a number of intra-articular lesions, including hemarthrosis in all shoulders, iatrogenic superior labrum anterior-posterior lesions, partial subscapularis tendon tears, anterior labral detachments, and tears of the middle glenohumeral ligament.[64] Typical findings during post-manipulation arthroscopy are hemarthrosis and capsular tearing; any ligament or tendon tears suggest a need for improvement in the manipulation technique.

Surgical intervention

Arthroscopic release

Despite initial recommendations that arthroscopy has no role in the treatment of adhesive capsulitis,[77] arthroscopic release has become more commonplace. The techniques have been well-described and limit the risk of intra-articular damage.[88,106] Numerous studies in the past few years have supported the role of arthroscopic release as a safe and effective treatment for recalcitrant frozen shoulder (Table IV).[3,6,28,37,98]

After documenting scapulohumeral range of motion and giving consideration to manipulation under anesthesia, the surgeon establishes a standard posterior portal and performs a diagnostic sequence. The long head of the biceps is inspected, and the rotator interval is defined by the anterior edge of the supraspinatus and the superior border of the subscapularis (Fig 2). The rotator interval is typically opened up, and scar tissue is typically released from the undersurface of the subscapularis. This permits translation of the humeral head inferiorly and laterally and allows for complete release of the anterior capsule. Partial release of the subscapularis is not usually necessary. Capsular release will then continue along the inferior capsule. The surgeon must be careful while releasing the inferior portion of the capsule, because the axillary nerve courses just inferiorly from medial to lateral in an anterior-to-posterior direction. Posterior capsular release can then be performed by placement of the camera anteriorly and by use of a posterior working portal. As with any capsular release, the complications of arthroscopic capsular release include shoulder dislocation and instability, which can occur with an overly aggressive technique.

Open release

Open release for frozen shoulder can be considered after failed release with manipulation, but with current arthroscopic techniques, open release is rarely performed and is largely considered a historical treatment option. This method of release is more commonly adapted in the treatment of patients with post-traumatic adhesive capsulitis, especially in the setting of retained hardware used in open reduction−internal fixation. Ozaki et al[84] first reported the results of open release for recalcitrant cases of frozen shoulder, with complete relief of pain and restoration of full range of motion occurring in 16 of 17 patients. Omari and Bunker[82] also reported on a series of 25 patients with adhesive capsulitis and found open release to be a useful operation in patients with severe disease.

Table III Results of manipulation under anesthesia for adhesive capsulitis

Author	Year	Technique	No. of patients	Follow-up	Results	Outcome scores
Ng et al[78]	2009	MUA + early physiotherapy	50	?	MUA with early physiotherapy alleviates pain and facilitates recovery	DASH, 15.84 VAS, 1.88
Wang et al[105]	2007	MUA	51	82 mo	Less improvement in post-surgery shoulders vs idiopathic or post-traumatic	Adjusted Constant, 70.1 (of 75)
Farrell et al[39]	2005	MUA	18	15 y	Sustained improvement after MUA at 15 y	SST, 9.5 ASES, 80
Hamdan and Al-Essa[45]	2003	1. MUA 2. MUA + saline solution injection 3. MUA + steroid injection	88	6-8 mo	MUA with saline solution injection did best	
Massoud et al[68]	2002	1. MUA 2. MUA, then arthroscopy 3. MUA, then arthroscopic release	47	35 mo	Good functional recovery with MUA in diabetic patients	Constant, 63.7
Othman and Taylor[83]	2002	MUA	74	33 mo	MUA effective in restoring ROM	Adjusted Constant, 72.4 (of 75)
Kivimaki and Pohjolainen[57]	2001	1. MUA with steroid injection 2. MUA without steroid injection	24		MUA not enhanced with steroid	
Dodenhoff et al[35]	2000	MUA	37	3 mo	94% patients satisfied with MUA, restored ROM	Median Constant, 69
Placzek et al[87]	1998	MUA	31	14.4 mo	Safe and effective for improving ROM	

Table IV Studies of operative treatment for adhesive capsulitis

Author	Year	Technique	No. of patients	Follow-up	Results	Outcome scores
Open						
Omari and Bunker[82]	2001	Open release of rotator interval and coracohumeral ligament	25	19.5 mo	Useful option for shoulder that fails therapy with MUA	VAS, 1.1
Ozaki et al[84]	1989	Open release of rotator interval and coracohumeral ligament	17	6.8 y	16/17 recalcitrant cases had full ROM and no pain	
Arthroscopic						
Snow et al[98]	2009	Anterior + inferior release vs anterior, inferior, and posterior release	48	5 mo	No difference with addition of posterior release	Constant: Standard, 66, + posterior, 71
Musil et al[72]	2009	RI, CH ligament, S/M/AIGHL release, anterior GH joint capsule	27		23 gained normal shoulder function	Constant, 80.3 UCLA, 28.6
Elhassan et al[37]	2010		115	46 mo	Idiopathic and post-traumatic stiffness have better outcomes than postsurgical stiffness	Constant, 86
Cinar et al[28]	2010		14 idiopathic 14 diabetic	48.5 mo 60.2 mo	No difference in duration of pain relief or ROM	UCLA Diabetic, 29.0 Idiopathic, 32.7 Constant Diabetic, 82.0 Idiopathic, 93.6
Liem et al[62]	2008	Release of subscapularis and capsule	22	53 mo	No loss of internal rotation strength	Constant, 82.8 ASES, 76.8
Baums et al[3]	2007		30	36 mo	Reliable improvement in arthroscopic release with gentle manipulation	ASES, 91 VAS, 2 SST, 10
Castellarin et al[25]	2004	MUA, then arthroscopic release	40	42 mo	MUA, followed by arthroscopic release, is effective for resistant cases	Constant, 91.7 SST, 10.8
Berghs et al[6]	2004	Arthroscopic release	25	14.8 mo	Arthroscopic release yields rapid relief of pain and improvement in function	Constant, 75.5 SF-36, 48.7
Yamaguchi et al[111]	2002	Arthroscopic release + intra-articular pain catheter	23	22.4 mo	19/20 achieved near complete ROM without pain	SSI, 90.9 VAS, 1.2
Klinger et al[59]	2002	Arthroscopic	36	18 mo	Safe and effective, no complications	Constant, 66 (median)
Holloway et al[49]	2001	Arthroscopic	50	20 mo	Less improvement in subjective scores in postoperative stiffness compared with post-traumatic and idiopathic	ASES, 57 (postoperative) ASES, 82, 86 (post-traumatic, idiopathic)
Bennett[5]	2000	Arthroscopic	31	>18 mo	30/31 retained intra-operative ROM gain, no difference in primary vs secondary stiffness	ASES, 82 Constant, 78
Watson et al[107]	2000	Arthroscopic	73	12 mo	11% with recurrent pain/stiffness	

MUA, manipulation under anesthesia, *ROM*, range of motion, *RI*, rotator interval, *CH*, coracuhumeral, *S/M/IGHL*, superior/medial/inferior glenohumeral ligament, *VAS*, Visual Analogue Scale, *SST*, Simple Shoulder Test, *ASES*, American Shoulder and Elbow Surgeons.

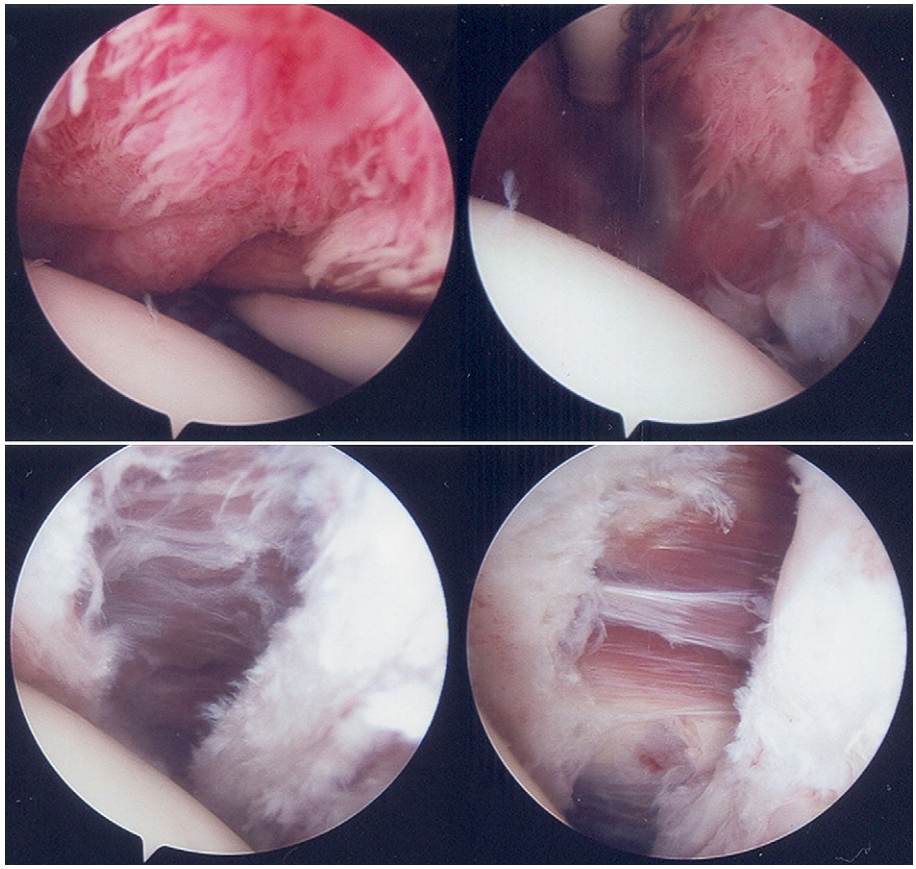

Figure 2 Arthroscopic release for adhesive capsulitis.

In patients with primary adhesive capsulitis undergoing the classic open capsular release, an incision is made from the clavicle to the lateral border of the coracoid. The deltoid is split to expose the coracohumeral ligament, and the ligament is excised with the arm in external rotation. The border of the rotator interval should be identified, along with the long head of the biceps. The tissue between the supraspinatus and subscapularis and under the coracoid process should be excised. Care should be taken to prevent iatrogenic damage to the subscapularis, supraspinatus, and long head of the biceps. If external rotation still remains tight after this release, the middle glenohumeral ligament, inferior glenohumeral ligament, and capsule can be divided as far posteriorly as possible. However, this can be fairly challenging and may require subscapularis tenotomy and repair for adequate visualization. More often than not, the superior capsule and posterior capsule have minimal involvement, and recently, it has been shown that the addition of posterior capsular release is likely superfluous in the treatment of idiopathic frozen shoulder.[26,98] If open release of the posterior capsule and superior capsule is performed, it requires takedown of the subscapularis and careful use of retractors to position the humeral head posteriorly and laterally to provide adequate access to these quadrants of the capsule.

Conclusion

Frozen shoulder is a common clinical disease affecting middle-aged persons, often with comorbid conditions such as diabetes, some cardiac diseases, or other endocrine diseases. It usually has an insidious onset and follows a protracted course. The pathogenesis is unclear, but it is thought to be a progression of inflammation and fibrosis and has many similar features to Dupuytren disease, including the lack of adhesions. Appropriate physical examination and radiologic studies can help in differentiating true adhesive capsulitis from the stiff and painful shoulder. A number of effective conservative treatments exist, and resolution of symptoms can be obtained in the majority of patients. Manipulation under anesthesia and surgical (arthroscopic or open) release can often restore motion and obtain pain relief for patients with refractory cases. Although patients who initially present with frozen shoulder should always be treated nonoperatively while allowing a chance for

spontaneous improvement, larger prospective, comparative studies are needed to determine the optimal treatment for recalcitrant frozen shoulder.

Disclaimer

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