Adhesive Capsulitis : A Review of Current Treatment

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A Review of Current Treatment

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Adhesive capsulitis is characterized by a painful, gradual loss of both active and passive glenohumeral motion resulting from progressive fibrosis and ultimate contracture of the glenohumeral joint capsule. Variable nomenclature, inconsistent reporting of disease staging, and a multitude of different treatments have created a confusing and contradictory body of literature about this condition. Our purpose is to review the evidence for both nonsurgical and surgical management of adhesive capsulitis with an emphasis on level I and II studies when available. Significant deficits in the literature include a paucity of randomized controlled trials, failure to report response to treatment in a stage-based fashion, and an incomplete understanding of the disease's natural course. Recognition that the clinical stages reflect a progression in the underlying pathological changes should guide future treatments.

Keywords: stiff shoulder; frozen shoulder; adhesive capsulitis; periarthritis scapulae; Duplay disease

Adhesive capsulitis is characterized by a painful, gradual loss of both active and passive glenohumeral motion resulting from progressive fibrosis and ultimate contracture of the glenohumeral joint capsule. Patients suffering from this condition face months to years of pain and disability. Codman[14] described his cases as "difficult to define, difficult to treat and difficult to explain from the point of view of pathology." Variable nomenclature, inconsistent reporting of disease staging, and a multitude of different treatments have created a confusing and contradictory body of literature.[48] The true natural history has also not been definitively established, making evaluation of treatment outcomes difficult. Our purpose is to review the evidence for both nonsurgical and surgical management of adhesive capsulitis with an emphasis on level I and II studies when available.

The term "frozen shoulder" was first used by Codman to describe a condition of tendinitis with secondary involvement of the subacromial bursa. He noted that "the term applies to many other conditions which cause spasm of the short rotators or adhesions about the joint or bursae."[14] In 1945, Neviaser[47] described the pathological lesion of fibrosis, inflammation, and capsular contracture responsible for idiopathic frozen shoulder and suggested adhesive capsulitis as a more appropriate descriptor of the pathoanatomy. Although frozen shoulder and adhesive capsulitis are frequently used interchangeably, recognition that many conditions can cause a stiff and painful shoulder while adhesive capsulitis is a distinct pathological entity is essential for evaluating both patients and the literature.[49]

Adhesive capsulitis occurs in 2% to 5% of the population, and a majority of patients are female.[5] Ages range from 40 to 60 years,[39] and the nondominant hand is more frequently involved.[26,37] About 20% to 30% of those affected will develop the condition in the opposite shoulder.[5] The causes remain unclear. It has been proposed that stimulation of synovitis results in the development of a fibrotic cascade that may involve growth factors such as TGF-beta.[65] Twenty percent to 30% of patients will report a history of minor trauma to the shoulder,[26,37] but there is no further evidence that this is a posttraumatic condition. Autoimmune processes have been proposed as the underlying pathophysiology, but evidence for this is contradictory. The incidence of HLA-B27 does not appear to be increased in patients with adhesive capsulitis as was initially reported by Bulgen et al.[10,63]

The development of adhesive capsulitis has been associated with diabetes mellitus,[43,45,52] thyroid dysfunction,[7,77] Dupuytrens contractures,[70] autoimmune disease,[10] and the treatment of breast cancer.[13,76] Patients with cerebrovascular accident or myocardial infarction have been reported to be at increased risk.[45,46] It is more common in those with sedentary vocations than in manual laborers.[49] The subset of diabetic patients who suffer from this condition has proved to be a group that is difficult to treat. It is associated with increasing age in type I and II diabetic patients and with autonomic neuropathy, history of myocardial infarction, and the duration of diabetes in type I patients.[2]

Diagnosis can be challenging as factors both intrinsic and extrinsic to the shoulder can cause stiffness and pain. The term *primary* adhesive capsulitis has been

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TABLE 1
Stages of Adhesive Capsulitis

	Symptoms	Signs	Arthroscopic Appearance	Biopsy
Stage 1				
	Pain referred to deltoid insertion Pain at night	Capsular pain on deep palpation Empty end feel at extremes of motion Full motion under anesthesia	Fibrinous synovial inflammatory reaction No adhesions or capsular contracture	Rare inflammatory cell infiltrate Hypervascular, hypertrophic synovitis Normal capsular tissue
Stage 2				
	Severe night pain Stiffness	Motion restricted in forward flexion, abduction, internal and external rotation Some motion loss under anesthesia	Christmas tree synovitis Some loss of axillary fold	Hypertrophic, hypervascular synovitis Perivascular, subsynovial capsular scar
Stage 3				
	Profound stiffness Pain only at the end range of motion	Significant loss of motion Tethering at ends of motion No improvement under anesthesia	Complete loss of axillary fold Minimal synovitis	Hypercellular, collagenous tissue with a thin synovial layer Similar features to other fibrosing conditions
Stage 4				
	Profound stiffness Pain minimal	Significant motion loss Gradual improvement in motion	Fully mature adhesions Identification of intra-articular structures difficult	Not reported

used to describe the idiopathic process of global capsular inflammation and fibrosis occurring in the absence of other lesions. *Secondary* adhesive capsulitis has been used to describe a constellation of conditions resulting in a stiff shoulder.[41] Some of these conditions demonstrate isolated areas of capsular contracture that are indistinguishable from idiopathic adhesive capsulitis but occur concurrently with other known injuries or diseases. Others have an extra-articular cause of shoulder stiffness without involvement of the joint capsule. Calcific tendinitis, rotator cuff injury, biceps tendinitis, as well as glenohumeral or acromioclavicular arthritis, all can cause shoulder stiffness in the absence of capsular limitation of motion. Distinguishing between primary or idiopathic disease and pain due to other causes can be difficult and there is frequent overlap. Yoo et al[78] reported that 62% of patients with stage 2 idiopathic adhesive capsulitis were found to have supraspinatus lesions, most commonly partial-thickness tears, after undergoing magnetic resonance imaging (MRI).

Subtle clues in the history and physical examination allow discrimination of primary adhesive capsulitis from these other conditions. Treatment of so-called secondary adhesive capsulitis should be directed toward the associated condition causing immobilization of the shoulder, and outcomes differ based on that condition.[30,50] Only the treatment of primary adhesive capsulitis will be discussed here.

Adhesive capsulitis progresses through 4 stages described by Neviaser and Neviaser[49] based on the correlation of physical examination and arthroscopic examination of affected joints. Hannafin et al[28] demonstrated the histopathological progression of disease in capsular biopsies from patients with Neviaser stages 1 to 3. Recognition of these stages is essential to applying appropriate treatment, communicating prognosis, and establishing the expectations of both patient and physician (Table 1). The arthroscopic and histopathological appearances of the shoulder capsule in the various stages of disease are discussed below. These are not required to identify the stages of disease. Diagnosis and staging of adhesive capsulitis are determined clinically based on symptoms and physical examination. Intra-articular anesthetic injection can be used to discriminate between the first 2 stages.

Stage 1 is characterized by a gradual onset of pain typically referred to the deltoid insertion. It is usually achy at rest and sharper with movement. Pain at night is common, and patients frequently report an inability to sleep on the affected side. Duration of symptoms is generally less than 3 months. Capsular pain on deep palpation or passive

stretch is common. There is an empty end feel at the extremes of motion. Patients may report limitation of movement; however, motion is fully restored when pain is relieved by intra-articular anesthetic injection. Discriminating stage 1 disease from other shoulder conditions can be difficult, as the symptoms are not specific. Early loss of external rotation with intact rotator cuff strength is a hallmark of adhesive capsulitis that is less common in other disease processes.[16] Further diagnostic testing may be required to exclude other diagnoses (ie, radiographs to rule out calcific tendinitis and early osteoarthritis or MRI to rule out rotator cuff injury). Arthroscopy in this stage reveals a fibrinous synovial inflammatory reaction without adhesions or capsular contracture (Figure 1A). Biopsy of the joint capsule demonstrates rare inflammatory cell infiltrates; hypervascular, hypertrophic synovitis; and normal capsular tissue (Figure 1B).

Stage 2 represents a combination of acute synovitis and progressive capsular contracture, which some have called the freezing stage.[60] Pain persists and may be more severe, particularly at night. Motion is restricted in forward flexion, abduction, and internal and external rotation. Limitation of motion is improved but cannot be fully reversed with intra-articular anesthetic injection.[42] Arthroscopy demonstrates a thickened, hypervascular synovitis described as having a Christmas tree appearance (Figure 2A).[49] There is early loss of the dependent axillary pouch causing the restricted motion. Hypertrophic, hypervascular synovitis with perivascular and subsynovial scar formation is seen on capsular biopsy (Figure 2B).

In stage 3, the stage of maturation,[49] also referred to as the frozen stage,[60] the predominant patient complaint is significant stiffness. Pain may still be present at the end range of motion and occasionally at night. Physical examination reveals a sense of mechanical block or tethering at the ends of motion. No improvement in motion is seen with intra-articular anesthetic injection or examination under anesthesia.[27,42] Symptoms have typically been present for 9 to 15 months at this point. Loss of the axillary recess is seen on arthroscopic examination, and minimal synovitis is present (Figure 3A). Capsular biopsy demonstrates dense, hypercellular, collagenous tissue with a thin synovial layer exhibiting features similar to other fibrosing conditions (Figure 3B).

Stage 4, the chronic stage,[49] has also been termed the thawing stage.[60] Pain is minimal, and a gradual improvement in motion can occur. The amount of improvement typically seen is controversial. Long-term objective assessments demonstrate more significant motion deficits than patients tend to self-report, and the natural history of the disease has not been clearly described.[69] Arthroscopy demonstrates fully mature adhesions, making identification of intra-articular structures difficult.[49] Histopathology has not been reported for this stage.

Roentgenograms done at any stage reveal no pathological changes other than the osteopenia of disuse. It is critical to understand that these stages are not distinct but rather represent a continuum of disease. Interpretation of the majority of literature regarding treatment is difficult to assess because few papers report the duration of disease as an independent variable in response to treatment, and the ultimate course of this disease remains controversial.

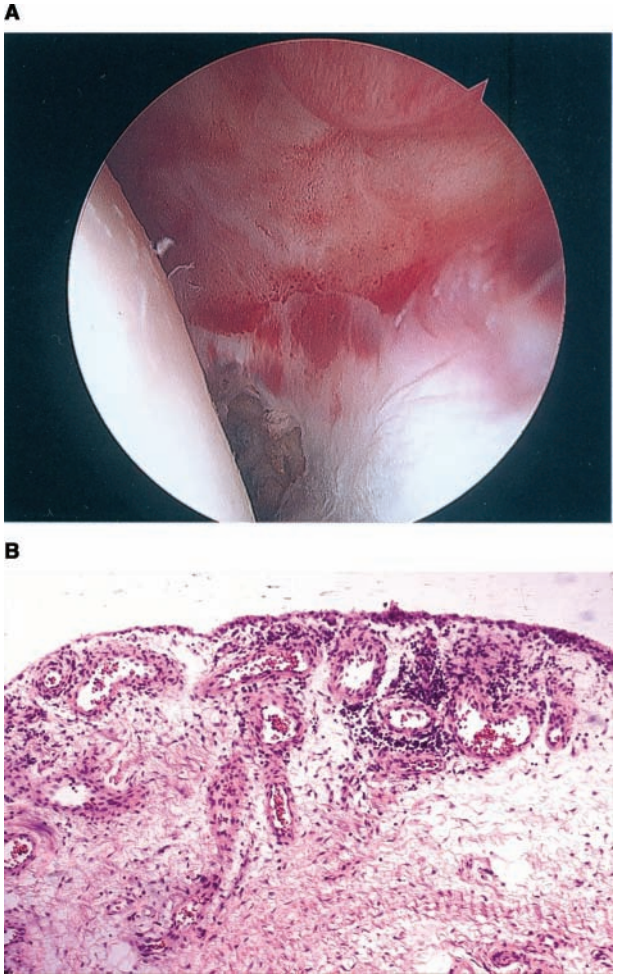

Figure 1. A, Stage 1 adhesive capsulitis is characterized by fibrinous synovial inflammatory reaction without adhesions or capsular contracture. B, Histologic findings of stage 1 adhesive capsulitis demonstrate rare inflammatory cell infiltrate; hypervascular, hypertrophic synovitis; and normal capsular tissue.

NATURAL HISTORY

Adhesive capsulitis has been described as self-limiting[24]; however, there are no true natural history studies in the literature. In all series, some form of treatment is given, although in several instances, this is minimal. A series of level IV studies describe the course of adhesive capsulitis with minimal intervention. Grey[24] reported 24 of 25 patients, with a minimum 2-year follow-up, returned to "absolutely normal function" after treatment with simple analgesics and reassurance. Miller et al,[45] in a retrospective review of 50 patients treated with home therapy, moist heat, and oral anti-inflammatory medications, found that

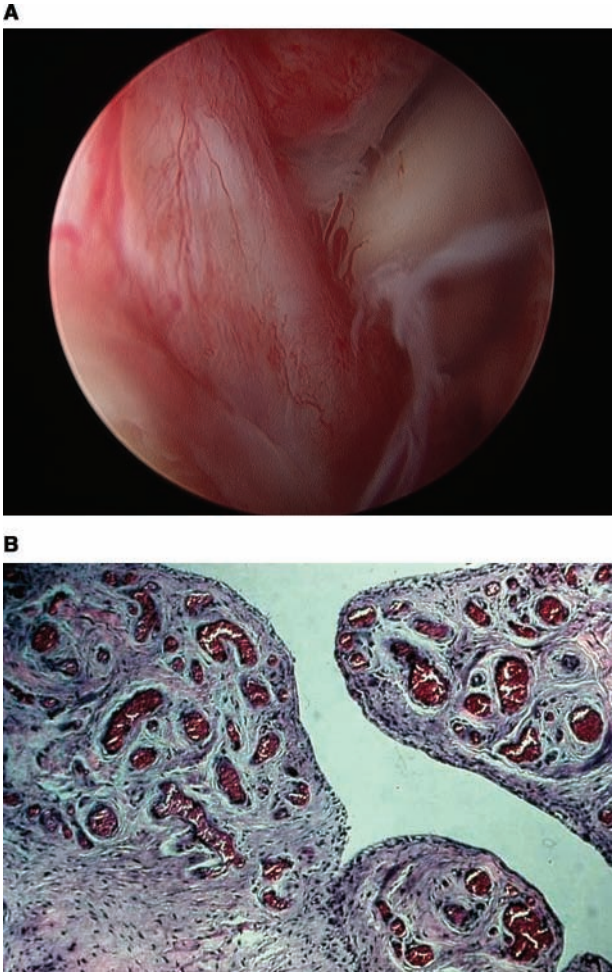

Figure 2. A, Stage 2 adhesive capsulitis demonstrates a thickened, hypervascular synovitis described as having a Christmas tree appearance. B, Hypertrophic, hypervascular synovitis with perivascular and subsynovial scar formation is seen on histologic examination for stage 2 capsulitis.

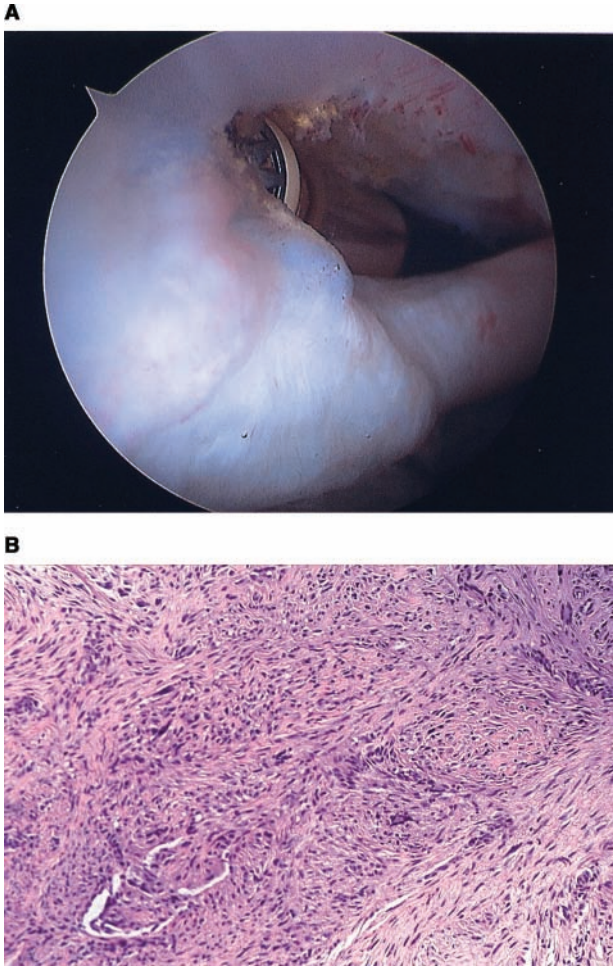

Figure 3. A, Scarring of the superior labrum is seen on arthroscopic examination, and minimal synovitis is present with stage 3 adhesive capsulitis. B, Capsular biopsy demonstrates dense, hypercellular, collagenous tissue with a thin synovial layer exhibiting features similar to other fibrosing conditions.

all 50 reported normal function of their arm and minimal residual pain on questionnaires completed 4 years after diagnosis. O'Kane et al[53] found that patients treated with home exercise alone improved in a self-assessed shoulder rating system, physical function, and pain subscales of the SF-36 at a mean 25-month follow-up. However, 30% to 40% of patients in this study could not place an 8-lb object on a shelf or carry a 20-lb object at their side. In a level III investigation of 42 patients followed for a minimum of 40 months, Binder et al[5] found that 5 patients had severe limitation of motion and 11 had mild limitation compared to age-matched controls. Involvement of the dominant arm, return to manual labor after onset of symptom, and male sex correlated with worse outcome. Others have reported that as much as 50% of patients have residual mild pain and decreased motion in long-term follow-up (average, 4-7 years) (level IV evidence).[26,69]

This discrepancy in results may be due in large part to variable methods of outcome measure. Studies that rely on patient-based outcomes tend to be more favorable than those using objective, physician-based evaluation. Whether the condition resolves on its own or not, the prolonged pain and disability require intervention.

TREATMENT

Treatment for adhesive capsulitis, as is true for any condition, should address the underlying pathology. Nonoperative measures encompass pharmacological treatment of the synovitis and inflammatory mediators and also physical modalities to prevent or modify capsular contracture. Surgery can address both the inflammatory component via synovectomy and the capsular contracture through capsular release and/or manipulation under anesthesia. Optimizing treatment depends on recognition of the clinical stage at presentation because the condition progresses through a predictable sequence.

NSAID Treatment

Despite their widespread use, literature on oral nonsteroidal anti-inflammatory drugs (NSAIDs) for the treatment of adhesive capsulitis is limited. We are unaware of any level I or II placebo controlled studies or comparisons of NSAIDs alone to an untreated group. In many studies, they are permitted in control groups and not considered to alter the course of disease. Thus, NSAID treatment, although of theoretical benefit, has yet to be proven effective as an isolated measure.

Comparisons of different NSAIDs to one another have been published. In a level II study, Rhind et al[61] reported on 41 patients randomized to either naproxen or indomethacin treatment for 4 weeks. Although both groups experienced decreased pain symptoms from baseline, no significant change in objectively assessed motion was found for either treatment. Fourteen patients in the naproxen group and 16 in the indomethacin group reported adverse reactions, with nausea being among the most common. A level I randomized study of 59 patients treated with the same drugs at higher treatment doses produced similar results.[21] The efficacy of COX II inhibitors and other more recently developed oral nonsteroidal anti-inflammatory agents has not been examined.

Oral Steroid Treatment

Two level I studies have compared oral steroid treatment with placebo. In a double-blind, randomized controlled trial, Blockey et al[6] examined a cortisone acetate suspension given in a tapered fashion over 4 weeks' time to a similarly administered placebo.

Thirty-two patients were included, and all began "vigorous" shoulder exercise after 1 week of treatment. Patients were stratified into 2 groups: those who had symptoms of less than 6 months, and those who had symptoms longer. No significant differences in reported pain or objective range of shoulder motion were found at 18 weeks' follow-up, and results were not stratified by duration of symptoms. Two complications were reported, 1 infection and 1 myocardial infarction, both occurring in the treatment group, but the difference in complication rate did not meet statistical significance.

Buchbinder et al[9] compared a 3-week course of oral prednisolone and placebo. This was also a double-blinded randomized trial. Forty-nine patients were included, making it sufficiently powered to detect a difference of 2 on a 0- to 10-point pain perception scale. The treatment group showed statistically less pain and improved function after 3 weeks of treatment. Examination at 6 and 12 weeks, however, failed to show a significant difference between the groups. The authors attributed this to a rebound effect in the treatment group occurring after the course of steroid was concluded. Pain and disability ratings failed to improve or declined at the cessation of treatment, while the placebo group continued to make improvements throughout the 12-week study.

In a level II study, Binder et al[4] compared 40 patients randomized to treatment with oral prednisolone for 6 weeks or no treatment. Examiners were blinded, but participants were not. All patients completed pendulum exercises 3 times per day. A more rapid improvement in pain symptoms was seen in the treatment group at 5 months but was not sustained through the 8-month follow-up.

Oral steroid treatment appears to provide more rapid relief of pain compared with controls (similar to the effects seen with intra-articular steroid injection), but this benefit is not sustained at longer follow-up. None of the described studies reported the stage of disease at the time of treatment so it is unknown if stage targeted treatment would produce better results. The potential for systemic side effects and the inconvenience of daily dosing are disadvantages of systemic treatment. No study of oral steroid treatment for adhesive capsulitis has been of sufficient duration to report long-term complications such as avascular necrosis of the femoral head, but the well-known side effects remain a theoretical concern.

Intra-articular Steroid Injections

An alternative to oral treatment is the use of intra-articular steroid injections. The efficacy of these injections has been extensively studied. The following level I investigations are representative of a large volume of literature on this subject.

Rizk et al[64] compared intra-articular methylprednisolone and lidocaine to an intra-articular lidocaine placebo and 2 control groups who received the same injections intrabursally. Inclusion criteria required symptoms of no more than 6 months' duration, and patients averaged approximately 12 to 14 weeks of symptoms before the study began. Follow-up occurred weekly for 11 weeks with additional evaluation at 15 weeks and 6 months post-injection. Blinded assessments of pain and range of motion showed no significant difference in shoulder range of motion between groups. Those treated with the intra-articular steroid did show a more rapid improvement in pain symptoms, but this difference was transient (2-3 weeks).

Bulgen et al[11] randomized 42 patients to 1 of 4 treatment groups: (1) intra-articular injection of methylprednisolone, (2) mobilization with a physiotherapist, (3) ice treatments following proprioceptive exercises, and (4) no treatment. Clinical inclusion criteria were specific but did not report staging or duration of symptoms. All patients reported improved pain at 6 months; however, the majority still had objective deficits in motion. Those treated with steroid injections had the most marked improvement in range of motion at 4 weeks' time. At 6 months, however, there was no difference between the groups. Others have confirmed these results with methylprednisolone injection.[3]

Lee et al[36] randomized 80 patients into 4 groups. Group 1 received a single dose of infrared radiation, group 2 received an intra-articular injection of hydrocortisone acetate (25 mg), and group 3 received the same injection into the biceps tendon sheath. All groups participated in a standardized physical therapy program. The fourth group

served as a control and received only oral analgesics without physiotherapy. Although all treatments produced improved range in motion compared with group 4 controls, no differences between the various treatments could be demonstrated. Improvement in motion occurred only in the first 3 weeks of treatment in all groups and not after. There was no stratification based on duration of symptoms or staging of disease.

Van der Windt et al[72] randomized 109 patients to receive either 40-mg intra-articular injections of triamcinolone acetonide or physiotherapy 2 times per week for 6 weeks. A painful and stiff shoulder due to "capsular syndrome" was required for inclusion. Stage of disease at presentation and duration of symptoms were not reported. Other shoulder conditions were excluded based on physical examination by a general practitioner. Patients could receive more than 1 injection but no more than 3 in the 6-week period. The group averaged 2.2 injections in the treatment period, although indications for repeat injections were not described. The authors reported treatment success in 77% of patients treated with injection compared with 46% of those treated with physiotherapy. Success was defined as patients who rated themselves having had a full recovery or much improvement based on pain and functional scales. This difference was statistically significant and persisted until the final assessment at 1 year.

Carette et al[12] in a placebo controlled trial showed that a single, fluoroscopically guided injection of 40 mg of triamcinolone hexactonide produced significantly improved Shoulder Pain and Disability Index (SPADI) scores as compared with placebo injection and physical therapy or placebo injection alone. There appeared to be an additive effect when steroid injection was combined with stage-specific physical therapy. No differences in SPADI scores were shown beyond 3 months' time, however. All patients had symptoms less than 1 year when the study began and were divided into those with acute and chronic adhesive capsulitis to determine the appropriate physical therapy protocol. The results were not stratified to examine differences in these groups, however. In a well-designed level I study, Ryans et al[68] confirmed these findings of more rapid improvement in patients treated with intra-articular triamcinolone injection as compared to controls, which dissipates after longer follow-up beyond 6 weeks.

Hazelman,[29] in a level IV retrospective review of 130 patients, reported that the efficacy of intra-articular injections of hydrocortisone inversely correlates with the duration of symptoms. This may reflect a greater efficacy in the early, inflammatory stages of the disease. A level IV retrospective case series at our institution of patients who received an injection of 80 mg of depomedrol in stages 1 or 2 showed high rates of recovery over 6 weeks and 7 months, respectively.[42]

Intra-articular injections may be more efficacious in stage 1 or early stage 2 before development of a significant capsular contracture, but this has yet to be proven with higher level evidence. They also do appear to provide earlier relief from pain than placebo, although this has not been shown to change the long-term outcome.

Physical Therapy

Physical therapy is the most consistently prescribed treatment to prevent capsular contracture and to improve motion in the latter stages of disease. Despite ubiquitous use, supporting evidence proving its benefit is very limited. A Cochrane database review of physiotherapy for painful conditions of the shoulder concluded that deficiencies in the literature resulted in little overall evidence to guide treatment and found no evidence that physiotherapy alone is of benefit in adhesive capsulitis.[23] This review contained only 4 (of 26) studies dedicated solely to the treatment of adhesive capsulitis with a combined 308 participants.

The level I studies by Carette et al[12] and Bulgen et al[11] (described above) found no differences between patients treated with physiotherapy and no-treatment controls. The number of patients enrolled in these studies, however, was low (42 and 93, respectively), and both studies randomized patients into 4 groups, increasing the likelihood of a type II error.

A level III study by Diercks et al[19] comparing benign neglect to intensive physical therapy would also appear to support this position. They found almost 90% of those in the "neglect group" had near normal shoulder function at 2 years as compared with 63% in the therapy group. Describing this control group as neglected was not accurate, however. Patients in the neglect group engaged in pendulum exercises and active exercises within their pain threshold throughout the study. The treatment group was subjected to more strenuous active and passive exercise and stretching beyond what was painful. Rather than validating neglect as a treatment, this study supports therapy within the limits of pain as an alternative treatment.

In a level I study, Vermeulen et al[74] examined the issue of rehabilitation intensity and found that there was little difference between those who engaged in low grade mobilization techniques compared with groups using high grade techniques. Low grade is defined as movement within a pain-free zone, while high grade mobilization included movements into the stiff, painful range.

In a level IV investigation, Griggs et al[25] prospectively evaluated 75 patients with stage 2 disease treated with a specific 4-direction stretching program and found 90% achieved a satisfactory outcome. Stretching was limited to the range of tolerable discomfort.

Despite the lack of high grade evidence clearly supporting the use of physical therapy, many lower level studies report its benefit, and its use in the treatment of adhesive capsulitis is almost universal.[23,45] Gentle stretching and active motion within the pain-free range appear to be sufficient, and the treatment need not be unduly painful.

In most series, approximately 10% of patients do not respond to the variety of nonoperative treatments described above.[26,37] In a level IV review, Levine et al[37] identified those patients who failed to improve or were worse after 4 months as most likely to fail nonoperative treatment. However, the indications for more invasive options remain highly subjective and need to be individualized to each patient. Manipulation under anesthesia (MUA), hydrodilation, suprascapular nerve block (SSNB),

and arthroscopic or open capsular release have all been described as possible options when physical and pharmacological therapies have failed.

Suprascapular Nerve Blocks

Suprascapular nerve blocks have traditionally been done by anesthesiologists in hospital pain clinics, but new techniques permit this procedure to be done in the office setting.[18] Advocates of their use in adhesive capsulitis suggest temporary disruption of efferent and afferent pain signaling may allow "normalization" of the pathological, neurological processes perpetuating pain and disability.[17,44] Pain relief may then translate into better shoulder function.

Dahan et al[17] conducted a well-designed, double-blind randomized trial comparing 17 patients treated with a series of 3 bupivacaine SSNBs with 17 patients treated with placebo injections. The treatment group showed a significant 62% improvement in overall pain compared with 13% improvement in controls. There was no difference in shoulder function between the 2 groups. Although this amount of pain relief is impressive, outcome was measured at 1 month only, severely limiting the value of this study.

In a prospective randomized comparison of intra-articular triamcinolone acetonide (20-mg) injection to SSNB (9.5 mL 0.5% bupivacaine and 20 mg triamcinolone) by Jones and Chattopadhyay,[34] the nerve block cohort showed a significantly greater reduction in pain and improved shoulder range of motion at 3 months' follow-up. Pain relief from the SSNB was greater as early as 1 week from the injection. Sleep disturbance showed a trend toward greater improvement, but the difference did not reach statistical significance. This is likely because of the small number of patients enrolled in the study, which was 15 per group.

These investigations suggest promise for SSNB; however, the exact therapeutic mechanism remains unclear. Larger studies with longer follow-up are needed to establish the role for SSNB in treating adhesive capsulitis.

Hydrodilation

Brisement, or hydrodilation, has been used as an alternative to operative procedures. This involves increasing intracapsular pressure and expanding capsular volume through injection of fluid until capsular rupture. It can be done under local anesthetic and takes only 15 minutes to complete. Various liquids have been used, and the procedure can be done in conjunction with arthrography. In the past, results have been variable and confounded by the use of dilation in combination with other treatments such as manipulation.

A recent level II randomized controlled trial of 36 patients (38 shoulders) compared hydrodilation with normal saline to MUA.[59] All patients were described as stage 2. Those who underwent MUA also received a 30-mg intra-articular injection of triamcinolone. Despite small numbers, the authors demonstrated an improvement in Constant score that was statistically greater in those receiving hydrodilation than in the MUA group. Visual analog pain scores were also statistically better in the hydrodilation group. Both differences persisted until the study concluded after 6 months. No differences in range of motion were found between the groups.

This investigation suggests benefit, but experience with this technique is limited, and more investigation is required to determine its ultimate role in the treatment of adhesive capsulitis. A Cochrane database review was unable to draw firm conclusions about the efficacy of this technique because of the small number of trials with few patients and different comparison interventions.[8]

Manipulation

Closed MUA has been used extensively with consistently satisfactory results in both short- and long-term follow-up. Complications of this technique have been reported including humeral fracture, subscapularis rupture, labral tears, and injury to the biceps tendon. These complications are minimized with proper technique.[22,40]

Higher level studies are few. Kivimaki et al[35] performed a level I study to compare MUA with a home-based exercise program. One hundred twenty-five patients were randomized to undergo home exercise or home exercise and MUA. The average duration of symptoms was similar in both groups (~7 months). No description of stage was given nor was a description of the treatment patients had before being enrolled. Inclusion criteria were not limited to patients who had failed more conservative treatment. The manipulation group had slightly better mobility at 3-month follow-up examinations with statistically significant improvement in shoulder flexion, but this was not sustained at 6 months and 12 months.

Dodenhoff et al[20] reported on the results of MUA in 39 shoulders from 37 patients in a prospective level IV study. All patients had primary adhesive capsulitis and were in Neviaser stage 2. Mean Constant scores improved from 24 before the procedure to 63 and 69 at 3 and 6 months, respectively. Ninety-four percent of patients were satisfied at final follow-up. The most common reason for satisfaction was regaining the ability to do daily tasks, which most could do within days of the manipulation.

Farrell et al[22] reported on the long-term results of MUA and showed sustained improvement in both pain and motion (level IV evidence). Nineteen shoulders (18 patients) maintained improvement in forward flexion from a mean of 104° before MUA to 168° at 15 years' follow-up. Mean external rotation improved from 23° to 67°. Sixteen patients reported no or minimal pain; 18 required no further surgery.

For refractory loss of motion, MUA appears to be a reliable treatment. The failure to show benefit beyond home therapy by Kivimaki et al[35] results from patient selection. Their cohort was not limited to patients failing other treatments as is the case with most other reports. Those who do not respond to physical therapy appear to benefit most from MUA, whereas it may not be as beneficial in less severe disease.[31]

Arthroscopy

Many surgeons have chosen to address this condition arthroscopically in addition to or instead of closed manipulation. To our knowledge, the earliest description of arthroscopic resection of the shoulder capsule is that of Conti[15] in 1979. It has since become the most popular method of treating refractory adhesive capsulitis. There are several advantages to performing an arthroscopic examination of the shoulder before capsular release or manipulation.[1] Diagnosis and staging of disease can be readily confirmed. If necessary, a therapeutic synovectomy can be performed, and potential secondary causes of symptoms can be recognized.

Several level IV studies have reported on the benefits of using arthroscopy to address capsular contracture. Pollack et al[58] reported 83% excellent or satisfactory results when arthroscopic debridement of the rotator interval was done in conjunction with MUA. Warner et al[75] were able to achieve significant improvement of motion in all planes in patients treated with arthroscopic anterior release who had previously failed MUA. Improvements in pain and motion have been reported to be preserved at long-term follow-up (7.5 years).[32]

In a level III comparison of MUA and arthroscopic division of the joint capsule by Ogilvie-Harris et al,[51] patients who were treated via arthroscopy were twice as likely to be pain free at 2 years' follow-up. Seventeen of the 20 patients treated with arthroscopic division had no functional deficit compared with 9 of 20 in the MUA group. Arthroscopic division included a release of the superior glenohumeral ligament and rotator interval as well as release of the intra-articular portion of the subscapularis (IASS). No significant deficits or instability have been reported with releasing this portion of the tendon.[50,57] However, the need for release of the IASS has not been proven in comparison trials.

How much of the capsule should be released remains a matter of debate. Some authors have advocated performing a 360° release while maintaining the IASS. Jerosch[33] described a release in which anterior, posterior, and inferior portions of the capsule up to the 5-o'clock position were sectioned with electrocautery. The most inferior part of the capsule is then cut with a small angled meniscus cutter to better protect the adjacent axillary nerve, producing a circumferential release. No axillary nerve injuries were reported in the initial series of 28 patients.

The posterior release is thought necessary to improve significant internal rotation deficits.[55] However, a recent level III investigation comparing anterior release with anterior and posterior release did not demonstrate improved range of motion with a more extensive release.[71]

Arthroscopic release has supplanted MUA as the treatment of choice for refractory adhesive capsulitis despite the lack of higher level comparison trials. Patients appear to achieve more significant and rapid improvements in motion and pain than the less precise manipulation and do not face the same risk of complications. Further investigation is needed to determine how much of the capsule requires release.

Open Release

The indications for an open capsular release are very few, and this procedure is rarely performed. Patients who have failed arthroscopic and closed manipulation may benefit from open intervention. This approach carries the morbidity of an open procedure including prolonged recovery, postsurgical stiffness, and restricted postoperative therapy. Small level IV series have reported success with open excision of the coracohumeral ligament. Ozaki et al[56] treated 17 patients who had failed nonoperative measures with an open excision of the rotator interval. Sixteen patients had complete pain relief and a return of motion equal to the other side after 3 months. In a slightly larger series, Omari and Bunker[54] treated 25 patients with the same open technique and found the results to be satisfactory. Pain and range of motion improved in all directions. However, of the 7 patients in the study who had diabetes mellitus, 4 had poor results. The authors cautioned against using this technique in insulin-dependent diabetic patients in light of these results.

AUTHORS' PREFERRED METHOD

We recommend the following stage-based treatment protocol. It is important to note that this protocol is aimed at addressing the progressive pathological changes reflected in clinical stages. Although, in our experience, this treatment method has been successful, it has not been tested in a controlled study and thus has not been validated.

Patients presenting in the early painful stages are given an intra-articular injection of 80 mg of methylprednisolone acetate mixed with lidocaine to disrupt the inflammatory process and to discriminate between stage 1 and 2 disease.[42] We do not routinely treat with oral steroids because the same benefit can be achieved with local injection without systemic side effects. Oral NSAIDs are routinely used in patients at all stages of disease for the analgesic effect and to facilitate both physical therapy and sleep.

Physical therapy remains the mainstay of treatment despite the lack of high grade evidence. As with other treatments, physical therapy is tailored to address the underlying pathological changes. For patients in stage 1, the goals of therapy are to interrupt the inflammation and diminish pain.[27] Education, activity modification, and gentle range of motion exercises are prescribed. Pain can alter glenohumeral mechanics, and therapy should focus on re-establishing proper scapulohumeral rhythm. Therapeutic modalities to relieve pain are employed such as iontophoresis, cryotherapy, and transcutaneous electrical nerve stimulation. Exercises include closed chain scapular stabilization, joint mobilization, continuous passive movement, hydrotherapy, and a home exercise program. Home therapy focuses on passive range of motion and pendulum exercises within the pain-free zone.[73,74]

Patients in stage 2 have the additional goal of minimizing capsular adhesions and restrictions of motion in addition to reducing pain and inflammation. Passive joint glides are used to increase capsular mobility.[66] Home

exercises are expanded to include cane exercises focusing on internal and external rotation range. Active exercises in the plane of the scapula are added to range of motion protocols aimed at preserving motion.

In the later stages (3 and 4), we do not use corticosteroid injection because the inflammatory phase of the disease has passed.[29,42] The focus of therapy in stage 3 disease is treatment of the marked loss of motion and abnormal scapulohumeral mechanics. Aggressive stretching should be the mainstay of therapy for this stage. Active warm-up to enhance soft tissue circulation is performed. Heat can be used to promote relaxation of the surrounding musculature. Prolonged, low-load stretching is more effective than brief, high-load stretching.[38,62] The limits of motion can be pushed, but the patient should not have significant pain. Strengthening of the scapular muscles continues, and strengthening of the rotator cuff muscles can be initiated if range of motion permits. Specific therapy for stage 4 does not greatly differ from stage 3. Further cuff strengthening including conditioning is initiated as motion improves.

We treat those patients who have failed nonoperative treatment with arthroscopic capsulotomy. Synovectomy is performed if there is significant synovitis. The indications for surgical intervention are patient specific. There are no clear guidelines that can be established from the literature, in large part because our understanding of the long-term sequelae of this disease is incomplete. We concur with Levine et al[37] that patients who are regressing despite appropriate therapy are likely to require surgical intervention. Generally, we prefer to wait a minimum of 4 months (usually more than 6 months) from the onset of symptoms before offering a surgical option to the patient. The decision to embark on surgery is made by the patient after a frank discussion of his or her prognosis, disability, and desired activity level.

We prefer arthroscopic division of the capsule to traditional MUA because it allows a more precise release. We do not have experience with hydrodilation or SSNB. Despite the findings of Snow et al,[71] the posterior capsule is included in the release because loss of internal rotation is typically significant and the pathological process involves the entire capsule.[67] The morbidity of including this release is, in our experience, insignificant. Regional anesthesia is used. A posterior viewing portal is established, followed by an anterior interval portal. Pump pressure is kept between 20 to 25 mm Hg. In a right shoulder, the release is begun at the 1-o'clock position by placing an electrocautery instrument medial to the superior labrum and directing it inferiorly. Progressing anteriorly and inferiorly, the capsule is released completely. The IASS is preserved. As the capsule is progressively divided, there is a concurrent increase in the available intra-articular space and ease of accessing the remaining capsule. From the anterior portal, release can be completed until the 5-o'clock position. The joint is then viewed from the anterior portal, and the electrocautery is placed posteriorly. The release is again begun superiorly at 11-o'clock and progresses posteriorly and inferiorly to the 7-o'clock position. The remaining inferior capsule is left intact to protect the axillary nerve.

A manipulation maneuver is completed at the end of the procedure to assure adequate release. In our experience, performing the MUA before arthroscopy leads to bleeding within the joint and impairs visualization. The scapula is stabilized by the surgeon with one hand, while the other hand is used to grasp the affected arm above the elbow. The manipulation progresses through a smooth sequence of movements. The arm is first externally rotated, then brought into full abduction above the patient's head. It is then lowered to 90° of abduction and internally rotated. Patients are placed in a sling postoperatively.

Surgery is done as an outpatient procedure, and all patients are scheduled to begin physical therapy the following day. We have found it beneficial to demonstrate the gains in motion to patients in the recovery room by fully abducting the arm overhead while they are still under regional anesthetic block but no longer sedated. This allows the patient to recognize that the mechanical block to motion is gone and allays fears about quickly beginning rehabilitation. Outpatient therapy is begun with a protocol similar to that described for stage 2 treatment. Progression is similar to the preoperative protocol described above but is more rapid and based on the resolution of postoperative pain and return of rotator cuff function.

CONCLUSIONS

The treatment of adhesive capsulitis remains controversial despite an abundance of published literature on the subject. Traditional treatments of physical therapy and NSAIDs have not been shown to alter the natural course of the disease but remain popular. Oral and intra-articular steroids provide early pain relief, but benefit cannot be shown beyond several weeks. Recognition that the clinical stages reflect a progression in the underlying pathophysiology may help tailor treatments more specifically. Lower level reports on intra-articular steroid injections suggest they may be most beneficial in the first 2 stages of disease, but this has not been investigated in higher level studies.

In patients who have failed nonoperative treatments, more invasive therapy is considered. Hydrodilation and SSNB have been reported to be successful, but studies are limited by short follow-up and small numbers. There is extensive experience with MUA. Level IV evidence has shown it to be a reliable means of improving function for patients with refractory disease. Arthroscopic capsuloraphy can provide similar benefits with less pain and allows global assessment of the joint and subacromial space.

Significant deficits in the literature include a paucity of level I and II studies examining more invasive measures, failure to report response to treatment in a stage-based fashion, and an incomplete understanding of the disease's natural course. Patients tend to report a more complete resolution of motion loss than has actually occurred, and many are left with significant deficits. Because we do not know the natural history of this disease, and will likely never know it, there is no clear standard against which treatment outcomes should be measured.

Despite extensive investigation, the causes of adhesive capsulitis remain unknown. Agents designed to specifically target inflammatory cytokines in stages 1 and 2 or agents designed to diminish scar deposition or enhance capsular remodeling in stages 2, 3, and 4 hold theoretical promise but are not yet used clinically. Further prospective controlled investigations of our current treatments are also needed to more accurately define which of those treatments provide the most benefit and to which patients.

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