ADHESIVE CAPSULITIS

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. Frozen shoulder is a contracture of the shoulder joint capsule. Although the disease causes a global contracture of the shoulder joint, it appears maximal in the rotator interval area, and particularly around the coracohumeral ligament.

Codman called it frozen shoulder. 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.

Epidemiology

Adhesive capsulitis 2% to 5%
The non-dominant hand is more frequently involved.
30% of those affected will develop the condition in the opposite shoulder.
30% of patients will report a history of minor trauma to the shoulder, but there is no further evidence that this is a posttraumatic condition.
Autoimmune processes have been proposed as the underlying pathophysiology

Predisposing factors

Diabetes mellitus: 20% of all diabetics and 36% of insulin dependent diabetes. 40% of bilateral are diabetic [>10 times normal population] Dupuytrens contractures present in 18%-50% of frozen shoulder Patients with CVA or myocardial infarction have been reported to be at increased risk. Thyroid dysfunction, Autoimmune disease

Pathogenesis

The histologic features of frozen shoulder include a matrix of type I and type III collagen populated by fibroblasts and myofibroblasts that suggest the condition may be modulated by an abnormality in the production of cytokines and growth factors.12 This inflammation cascade, driven by the abnormal cytokine production, has been implicated in the abnormal tissue repair and fibrosis that occurs. TGFb and PDGF to be elevated, and suggested that these cytokines may act as a persistent stimulus causing capsular fibrosis and the development of frozen shoulder. Stimulation of synovitis results in the development of a fibrotic cascade that may involve growth factors such as TGF-beta.

Although adhesive capsulitis is described to be a self-limiting disorder, this condition can sometimes last for years. Ten percent of the patients report persistent pain

Codman's criteria for diagnosis

Insidious in onset Pain is felt near the insertion of deltoid Inability to sleep on the affected side Painful and incomplete elevation Limitation of external rotation Restriction of both spasmodic Atrophy of the spinati No localizing tenderness IO. X-rays negative except for bony atrophy

Codman stated, 'even the most protracted cases get better, with or without treatment, in about 2 years'. It is this statement that has got into the textbooks, has become dogma, and has become established, without question, through three generations of orthopaedic surgeons.

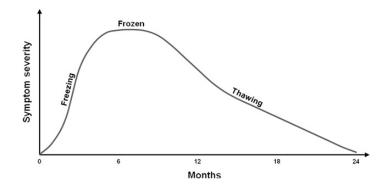
Clinical Signs

1.Painful and incomplete external rotation. D/D conditions that restrict external rotation are a.arthritis,

b. locked posterior dislocation, c. Milwaukee shoulder d. frozen shoulder.

2. Frozen shoulder shows an entirely normal radiographic appearance of the shoulder compared to other 3 causes.

In pragmatic terms, if the radiograph is normal and the joint shows passive restriction, then this can only be caused by contracture of the ligaments.



Imaging

X ray: normal

2. **MRI**: Thickening of the joint capsule and synovium in frozen shoulder. gadolinium-enhanced dynamic MRI, which has shown an increased blood flow to the synovium.

1. Thickening of the coracohumeral ligament and joint capsule in the rotator interval.

2. Capsular thickening of the axillary recess is often a useful sign

- 3. **The MRI arthrogram** is pathognomonic. The joint has a diminished volume, there is absent filling of the infraglenoid recess and the subscapular recess and bicipital tunnel are obliterated in frozen shoulder.
- 4. Ultrasound can show thickening of the coracohumeral ligament

5. Arthroscopy

In the early stages the major finding is angiogenesis, or new blood vessel formation. This can be quite spectacular, with fan shaped areas of blood vessel formation, villi formation. Within the infraglenoid recess the vessels line up in a radial fashion that we term the 'lava flow'. Granulation tissue that is red, highly vascular, with a villous synovium occurs in the rotator interval area. It is interesting that angiogenesis is a feature of diabetes, for frozen shoulder is common in diabetics. In the late stages the angiogenesis diminishes. The joint is less red, but thick bands of scar tissue can be found. The superior glenohumeral ligament becomes thickened, obliterating the rotator interval.

6. Pathology

'The coracohumeral ligament is converted into a tough inelastic band of fibrous tissue spanning in the rotator interval acts like checkrein. Division of the coracohumeral ligament allows early restoration of ROM.

1. Thickened, tight glenohumeral joint capsule with adhesions obliterating the normally patulous axillary fold.

2. Overall joint volume is diminished. Normal shoulder joint volumetric capacity is 28 to 35 mL of injected fluid, whereas in adhesive capsulitis, the joint accepts only 5 to 10 mL.

3. Biopsy of the capsule demonstrates a chronic inflammatory infiltrate, absence of synovial lining, and moderate to extensive subsynovial fibrosis. Perivascular lymphocytic reactions are noted, as well.

4. Four stages of disease have been described based on the arthroscopic appearance

5. Biopsy specimens from patients in the first three stages demonstrate a clear progression from perivascular mononuclear inflammatory infiltrates to reactive capsular fibrosis, confirming an inflammatory origin. Increased levels of transforming growth factor- β

and other profibrotic cytokines are present in capsular biopsy.

6. The inciting cause of the inflammation is unknown.

C/F: Patients typically present with pain of insidious onset of several months' duration.

Night pain is common

A mechanical restraint to passive motion is the hallmark of adhesive capsulitis. This finding is best appreciated on passive external rotation with the arm at the side.

Discriminating stage 1 disease (ie, before adhesion formation) from other pathology can be difficult because the signs

MRI: 4 mm thickening of the axillary fold on MRI :highly specific for adhesive capsulitis;Changes in the coracohumeral ligament were not a consistent feature.

Stage I

Nevasiar Stages

Stage 1Gradual onset of painPain at night is common,

Duration of symptoms is generally less than 3 months.

Patients may report limitation of ROM but fully restored

when pain is relieved by intra-articular anaesthesia

Early loss of ER with intact rotator cuff strength is a hallmark of adhesive capsulitis

Arthroscopy :

Fibrinous synovial inflammatory reaction

without adhesions or capsular contracture

Biopsy

Hypervascular, hypertrophic synovitis; and Normal capsular tissue

Stage II

Acute synovitis and capsular contracture [The freezing stag].

Pain persists and may be more severe, particularly at night.

Motion is restricted in Flexion, abduction, and rotations.

Limitation cannot be fully reversed with intra-articular anesthetic injection.

Arthroscopy

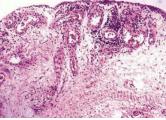
A thickened, hypervascular synovitis : a Christmas tree appearance There is early loss of the dependent axillary pouch Biopsy

Hypertrophic, hypervascular synovitis with perivascular and subsynovial scar

Stage III

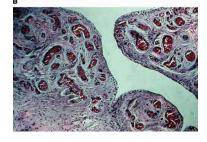
The stage of maturation, also referred to as the frozen stage, the predominant patient complaint is significant stiffness. Pain may still be present at the end of ROM No improvement in motion is seen with intra-articular anesthetic injection or examination May present for 9 to 15 months at this point. Arthroscopy Loss of the axillary recess with minimal synovitis is present Biopsy Dense, hypercellular, collagenous tissue with a thin synovial layer exhibiting features similar to other fibrosing conditions





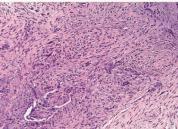
Stage II





Stage III





Stage 4 the chronic stage [thawing stage]
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.
Arthroscopy demonstrates fully mature adhesions, making identification of intra-articular structures difficult.

Natural course [Orth Trauma 25:1: 16]

Griggs confirmed these findings and stated that 'even amongst the patients who were satisfied, a substantial number were not pain free'; 10% had mild pain at rest, and 27% had mild or moderate pain with activity. 40% of the satisfied patients had abnormal shoulder function.

Present study: showed that although 86% had an improvement in their level of pain, this did not mean that they had no pain. Only 53% had no pain, 33% had an occasional pain and 14% had marked residual pain.

Treatment

1.Steroid [J Shoulder Elbow Surg (2010) 19, 172-179]

Prospective Randomized 40 patients with idiopathic adhesive capsulitis, treated with an oral corticoid Rx or 3 intra-articular injections of corticosteroids. Follow-up was after 4, 8, and 12 weeks, and 6 and 12 months. Intra-articular injections of glucocorticoids showed superior results in objective shoulder scores, range of motion, and patient satisfaction compared with a short course of oral corticosteroids. Significant superior range of motion for the patients treated with intra-articular injections was seen in shoulder flexion after 4, 8, and 12 weeks of follow-up and for the abduction after 8 weeks and 6 and 12 months of treatment. External rotation showed significant differences at 4 and 8 weeks of follow-up.

2.NSAID Treatment: No difference with different NSAID

3. Oral Steroid Treatment: 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.

4.Physical therapy: Physical therapy is the most consistently prescribed treatment. The level I studies by Carette found no differences between patients treated with physiotherapy and no-treatment controls.

A level III study by Diercks et al19 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. Patients in

the neglect group engaged in pendulum exercises and active exercises within their pain threshold throughout the study.

4. Manipulation under anesthesia, (MUA)

Professor Sir John Charnley, 70% said they would never perform a manipulation, as all would eventually get better, and some could be harmed.

Sneppen shown that 79% of patients with frozen shoulder are relieved of their pain, and 75% regain a near normal range of movement after manipulation.

We have arthroscoped patients before and after manipulation to discover exactly what is happening.1 Essentially, what we found was that elevation, or abduction, tears the capsule from the neck of the humerus, releasing the inferior capsule, and this occurs with relative ease. It is much harder to free rotation, but forced external rotation tears the coracohumeral ligament. This is an extra-articular ligament, so what is seen arthroscopically is haemorrhage in the rotator interval. Often, the coracohumeral ligament is so contracted that it will not tear and the patient is left with limitation of external rotation.

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. Following this, 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.

Kivimaki a level I study : Compare MUA Vs a home-based exercise 125 patients were randomized The MUA 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. Farrell reported on the long-term results of MUA and showed sustained improvement in both pain and motion

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.

5.Arthroscopic release, in the hands of the expert shoulder surgeon, has transformed the management of capsular contracture.

Harryman and Matsen published a year later and demonstrated fantastic results. The range of motion went from 41% of the opposite side to 78% on the first postoperative day and 93% at the end of the study. Before surgery 6% could sleep and after 73%. Recent long term results [>10 years] showed good outcome is well maintained.

Arthroscopic release appears to show great promise for it delivers what the patient wants; relief of pain, undisturbed nights and improved function.

6. 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.