Clinical Article

Frozen shoulder: A review

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Abstract
A 'frozen shoulder' has often frustrated both orthopaedic surgeons and patients. A review of the literature has shown clear clinical and pathological stages of the disorder. No clear cause has yet been found for the idiopathic type of frozen shoulder. Management options and recent clinical results are discussed. It is clear, however, that research in the treatment of this condition is difficult and can result in misleading outcomes.

Introduction
The term 'frozen shoulder' was first used by Codman in 1934[1] and thereafter Neviaser[2] noted that the pathology of this condition was actually located in the capsule of the shoulder joint and therefore called it 'adhesive capsulitis'.
A stiff and painful shoulder is often casually labelled as a frozen shoulder. This type of generalisation should be avoided, as one could miss other more serious conditions that need to be treated urgently. By taking a thorough history and performing a proper examination, the surgeon will expose certain facts that are typical of a frozen shoulder. The typical findings are pain and a global restriction of movement, with limited passive external rotation being the most notable.

Epidemiology
Research has shown a prevalence rate of 2% in the general population.[3] It affects persons older than 40 years[4] of age more commonly, and 70% of patients presenting with a frozen shoulder are women.[5] The condition affects diabetic (type 1) patients more often than healthy ones, with a prevalence of almost 11% in this population group.[6] No racial predilection has been described.

Griggs *et al*[7] suggested with their results that female patients who do not have an intrinsic emotional, psychological or personality disorder can overcome adhesive capsulitis better than those who do.

Pathology of 'frozen shoulder'
The pathophysiological process is believed to involve synovial inflammation and fibrosis of the shoulder joint capsule.[8] With microscopic examination of the tissue one will find the majority of cells to be fibroblasts with some mast cells also present. Cytokines such as transforming growth factor β and platelet-derived growth factor may contribute to the inflammatory process. Hand *et al*[9] suggested the process to be immunomodulated, as they found a chronic inflammatory response with fibroblastic proliferation to be present.

Although the glenohumeral joint synovial capsule is involved, much of the disease also involves structures outside the glenohumeral joint. These structures can include the coracohumeral ligament, rotator interval, subscapularis musculotendinous unit and the subacromial bursa.[6]

Aetiology
The cause of the idiopathic form of the disease to date is unknown. Secondary frozen shoulder develops when there is a possible cause identified. See *Table I* for a list of causes.

The typical findings are pain and a global restriction of movement, with limited passive external rotation being the most notable

Table I: Conditions that can lead to secondary frozen shoulder

Systemic	Extrinsic	Intrinsic
Diabetes mellitus	Cardiopulmonary disease	Rotator cuff tears
Hypothyroidism	Cervical spine pathology	Rotator cuff tendinitis
Hyperthyroidism	CVA (stroke)	Biceps tendinitis
Hypoadrenalism	Parkinson's disease	Calcific tendinitis
	Humerus fracture	AC joint arthritis

Clinical picture

The diagnosis is made on the basis of the medical history, clinical and radiological examination and the exclusion of other shoulder pathologies. Impingement syndrome can often be confused with early stages of frozen shoulder.

In 1934 Codman[1] proposed the following diagnostic criteria for frozen shoulder:

- shoulder pain with slow onset
- pain felt at deltoid insertion
- inability to sleep on affected side
- atrophy of the supra- and infraspinatus muscles
- sometimes minimal local tenderness
- restriction of active and passive ROM
- painful and restricted: elevation and external rotation.

The patient's history is especially important so as to determine the patient's current disease stage to help guide one in the appropriate treatment.

History

Most patients with primary frozen shoulder have no history of shoulder trauma. They usually give a history of insidious onset of pain, followed by a loss of motion. Night and rest pain are common in the early stages.

Patients who suffer from secondary frozen shoulder often give a history of known diabetes mellitus.[10] The incidence is reported to be between 10 and 36%[10] in diabetic patients. Other conditions that have shown an association with frozen shoulder and which might give a clue to the diagnosis are the following: hyperthyroidism, hypothyroidism, hypoadrenalism, Parkinson's disease, cardiac disease and a history of stroke.[11] A history of recent surgery, such as cardiac surgery, neurosurgery and radical neck dissection has also been associated with the development of secondary frozen shoulder.

Clinical examination

The only sign found in the early stages of the disease process is pain experienced at the end range of shoulder motion. Patients presenting with stages 1 and 2 have pain on palpation of the anterior and posterior capsule and describe pain radiating to the deltoid insertion. Later on in the disease process, one can note mild disuse atrophy of the deltoid and supraspinatus muscles.

A diffuse tenderness with palpation over the glenohumeral joint can extend to the trapezius and interscapular area.[11] The extension of this tenderness into the neck and upper back is due to the splinting of the painful shoulder.

It has been shown that a complete loss of external rotation is a pathognomonic sign of frozen shoulder.[12] It is important to distinguish whether this loss of external rotation occurs both actively and passively. If passive external rotation is full but active external rotation is absent, a possible rotator cuff tear should rather be considered. Most of the movement in a severely affected frozen shoulder occurs at the scapulothoracic joint.

The disease process least affects extension and horizontal adduction motion.[13]

Special examinations

The diagnosis of frozen shoulder should be based on clinical examination findings with limited dependence on specific laboratory findings or radiological examinations. Most references therefore suggest only a plain X-ray to be requested. Often these might be reported as normal but some may show periarticular osteopaenia due to disuse.[14] These X-rays can also assist in excluding other potential causes of a stiff shoulder, such as glenohumeral arthritis, calcific tendonitis or rotator cuff disease.[15]

MRI arthrography is not routinely needed for the diagnosis of adhesive capsulitis but if performed will show a slight thickening in the joint capsule and the coracohumeral ligament.[11] Lee *et al*[16] showed a decrease in the filling ratio of the fluid-distended axillary recess when compared to controls in patients with arthroscopically proven adhesive capsulitis.

MRI alone can also be helpful in identifying other causes of a stiff shoulder, such as infection or tumours.

Laboratory investigations are not routinely required, but can be helpful in patients with other medical issues that may lead to secondary frozen shoulder. These include thyroid-stimulating hormone levels, lipid levels and fasting blood glucose.

The patient's history is especially important so as to determine the patient's current disease stage

Natural history

It is important to realise that this condition is self-limiting and therefore improves with time. The majority of the literature states that this period lasts for between 18 and 24 months. Recent literature seems to suggest that recovery might take even longer. In 2008 Hand *et al*[17] showed in their population group that 40% of patients still suffered from moderate to severe pain at 4 years' follow-up. This is supported by Dudkiewicz *et al*,[18] who showed that some patients experienced an improvement in symptoms only up to 10 years after the onset. Shaffer *et al*[19] showed in their group of 61 patients with frozen shoulder that some degree of pain and stiffness was present in 51% of patients at an average of seven years after onset. It therefore appears as if a large percentage of patients have persistent pain and stiffness in the long term (two years or longer).

Poor prognosis has been associated with factors such as insulin-dependent diabetes and associated intrinsic pathology such as calcifying tendonitis.

In 1987 Neviaser and Neviaser[5] described four stages in the disease process. Hannafin *et al*[20] subsequently used these four stages and were able to correlate the clinical examination with the histological appearance of capsular biopsy specimens for the first three stages. It is important to note that these stages represent a continuum of disease rather than discrete, well-defined stages.

Stage 1 (inflammatory):

The patient complains of pain with active and passive range of motion. The pain is described as an ache at rest and sharp with motion and is usually worse at night. Range of motion is still well maintained. These symptoms are usually present for less than 10 weeks. Intra-articular injection of local anaesthetic and a corticosteroid gives significant improvement in range of motion.

Stage 2 ('freezing'):

The patient gives a history of chronic nagging pain over the previous 10 to 36 weeks. The pain is worse at night. No history of injury is present. There is progressive loss of range of motion. Arthroscopic findings include diffuse pedunculated synovitis and a rubbery/dense feel at insertion of arthroscopy cannula.

Stage 3 ('frozen'):

This occurs at 4 to12 months. Pain gradually subsides and is only present at the extreme range of movement. Gross reduction of movement is present with almost no external rotation possible.

Stage 4 ('thawing'):

This occurs usually from 12 months after onset and can last for up to 42 months thereafter. A spontaneous improvement in range of movement occurs with minimal pain. Histology has not yet been correlated because patients usually do not undergo surgery at this stage as they are improving.

Treatment

Educating patients will help to reduce frustration and encourage compliance. Time should be taken to explain the condition to the patient. Specific information on the natural history of the disease and an explanation that it will resolve spontaneously should be provided. It should be remembered, though, that the full range of motion might never be restored as shown in some of the aforementioned studies (see 'Natural history').

A decision regarding the best treatment option depends on the stage of the disease and clinical symptoms. There is however still no consensus on a standard management protocol. This is understandable if you consider the opinion of Lubiecki *et al*[21] who came to the conclusion that there is no conclusive evidence on the superiority of any of the known published interventions. They based this on the fact that there is no 'robust data' available that compared any specific treatment with the natural history of the disease.

Non-surgical treatment

Medication

Oral non-steroidal anti-inflammatory medication can be initiated in patients who present with painful limited range of motion during the painful freezing phase. This can be supplemented with other analgesics as necessary. There is, however, no randomised, controlled study to confirm the effectiveness of NSAIDs in frozen shoulder.

Intra-articular steroids

It is suggested in the literature[21] that an intra-articular injection has better pain relief than physiotherapy, analgesics or placebo. A Cochrane database review furthermore showed that it might be beneficial in the short term but that the effect might be small and not well maintained.[22] It is, however, more effective when used in combination with other treatment options, as proved by Carrette *et al*.[23] They were able to show that intra-articular steroids combined with physiotherapy were more effective in improving shoulder range of motion than when each of these was used individually. In more recent research, Jacobs *et al*[24] also showed that a combination of steroids (triamcinolone) and distension (21 ml per injection) had the same outcome at two years as manipulation under anaesthesia. This suggests that their proposed outpatient procedure gives similar results without exposing the patient to the risks associated with a manipulation under anaesthesia.

Physiotherapy

During **stage 1**, the focus should be on interrupting the cycle of inflammation and using modalities that can relieve the pain. Educating the patient in activity modification and positioning should be a priority.

Physical therapy and stretching are most effective in patients presenting with **stage 2** frozen shoulder. The goal should be to stretch the capsule sufficiently to allow normal glenohumeral biomechanics. Pain should, however, be the guide as to how intensive this should be. Diercks *et al*[25] compared the outcome of 77 patients after some received intensive physiotherapy (passive stretching and manual mobilisation) and the other supervised neglect (active exercises within pain-free range and pendulum exercises). The supervised-neglect group showed the best results with 89% of patients having normal painless shoulders as compared to the intensive group with only 63% of patients achieving the same results. It is important to note that both treatments were more than 50% effective and that there was no long-term evidence of efficacy of either method.

Hydrodilation

This has been suggested as an outpatient procedure. It was first described by Andren and Lundberg[26] in 1965 and involved the intra-articular injection of a large amount of normal saline to distend and rupture the capsular adhesions. A randomised study by Quraishi *et al*[27] showed better results with hydrodilation than manipulation under anaesthesia. They reported that at six months' follow-up the Constant score showed a statistically significant improvement. The range of movement had, however, not improved.

Oral steroids

These have been proposed as a treatment for frozen shoulder, but Buchbinder *et al*[28] found that, although it did improve the symptoms initially, the effect did not last beyond six weeks. In light of the adverse reactions, some authors suggest that it should not be routinely used for this condition.

Sheridan and co-authors[29] noted in their review of frozen shoulder that 'in most trials, there was no long-term difference (two-year follow-up) between treatment groups, as might be suspected in a self limiting condition.'

Most patients have significant improvement by 12 to 16 weeks. Some do not, however, improve and may get worse. These patients will then need surgical intervention after three to six months of conservative treatment.

Surgery

Manipulation under anaesthesia

Duplay initially recommended this kind of manipulation as treatment in 1872, when he described *periarthrite scapulohumerale.* It is generally indicated when the functional disability persists in spite of adequate non-operative treatment for four to six months. Kessel *et al*[30] supported this when they showed that patients do better if they have been symptomatic for more than six months. A complete evaluation of the passive range of motion of both shoulders should be performed while the patient is under anaesthesia so as to give the surgeon an idea of how much range is required, and to prevent overmanipulation. The manipulation should be performed in a systematic way so as to release all the relevant structures. Short lever arms should be utilised to prevent iatrogenic fractures. Some authors[29,31] recommend that an arthroscopic examination be performed before a closed manipulation, as they have shown that it helps to reduce stiffness and pain. Physiotherapy is recommended for two to six weeks post-surgery.

(See *Table II* for the contraindications of manipulation under anaesthesia.)

Open capsular release

The main aim of this procedure should be to release the coracohumeral ligament and rotator interval. In their study, Ozaki *et al*[32] showed that 94% of patients had relief from pain and had complete range of movement. It does, however, have its disadvantages, which include: postsurgical stiffness, decreased pain control and restrictions on physiotherapy in the early postoperative period. None of the studies that report on this technique included control groups and the results have therefore been questioned. Tasto *et al*[6] suggested that due to the postoperative limitations such as stiffness and pain, this procedure should be limited to those cases that are not ideal for arthroscopic release. These include cases with extensive subdeltoid scarring and extensive intra- and extra-articular contractures.

Arthroscopic capsular release

Since Conti described the first arthroscopic release in 1979, it has become the main surgical option in the treatment of adhesive capsulitis. It is especially helpful in cases where a manipulation has failed, as is often the case in diabetic patients. The advantages of the arthroscopic release include the following:

- the ability to evaluate glenohumeral joint and subacromial space
- possibility of a synovectomy in stage 2
- the facilitation of precise and complete release of the capsule in a controlled manner
- minimal postoperative pain
- the opportunity to start aggressive active and passive motion immediately.

Table II: Contraindications to manipulation under anaesthesia

- Significant osteopaenia
- Recent soft tissue repair in shoulder
- Presence of a fracture
- Neurological injury

It is difficult or even impossible to compare the results of different published series on this procedure since they all differ in technique, length of follow-up and the outcome measured. Most of these showed, however, that a release is more often required in diabetic patients than in non-diabetics, and that conservative treatment should still be the initial treatment of choice. Most of these consider the release of the antero-inferior capsule as the most important structure to release.

There are also risks involved and they include:

- recurrent stiffness
- anterior dislocation immediately after operation
- axillary-nerve palsy.

Some authors argue that a manipulation performed before arthroscopic release can cause excessive bleeding during arthroscopy and should therefore be avoided.[6]

The release itself can be very demanding due to a tight joint and the fact that a proper release of the capsule should be performed in the full 360° around the glenoid. Great care should be taken to avoid damage to the axillary nerve at the 6 o'clock position. Technically, it can be very demanding to release the capsule in this area, especially if the joint is tight. The use of a radio-frequency device is recommended. Some authors[33] have described releasing the m. subscapularis (or part thereof). Most studies do, however, show excellent results without releasing part of m. subscapularis. Nor should the labrum or biceps be released.

After the release, a manipulation can also be performed. Pain pumps are suggested to assist in early pain-free mobilisation the first few days. These should, however, be placed in the subacromial space, as some complications have been reported if placed intra-articular. An interscalene indwelling catheter block is also an option although not popular with some due to nerve-related complications. No results were found when comparing the different pain-management options and therefore personal preference should be the guide.

Patients can be started on physical therapy in hospital and discharged on home exercises that are both passive and active-assisted. CPM (if available) can be helpful, especially in refractory cases. Monitoring of the patient postoperatively is imperative so as to give support and assist in the home programme.

Chambler *et al*[34] came to the conclusion that there are no data to prove that early surgical intervention improves outcomes.

Discussion

When selecting a treatment method for frozen shoulder, it is extremely important to consider the patient's symptoms and stage of the condition because each patient's treatment should be individualised. As mentioned previously, it can be very difficult to place patients in a specific stage as stages often overlap. Suggestions for treatment in the various stages are shown in *Table III*.

Table III: Proposed management options for the different stages of frozen shoulder

Stage 1	Intra-articular steroids Physiotherapy
Stage 2	Intra-articular steroids Arthroscopic release
Stage 3	Arthroscopic release
Stage 4	Monitoring progress Active physiotherapy

It is extremely important to consider the patient's symptoms and stage of the condition because each patient's treatment should be individualised

Much of the data and research results on the active treatment of this condition show satisfactory outcomes, but that is to be expected in view of the natural history of the frozen shoulder. The true role of active treatment as compared to supervised neglect has not yet been fully established. Chambler *et al*[34] also suggested that any orthopaedic surgeon wanting to treat this condition effectively should be able to perform an adequate arthroscopic release of the capsule. The same author concluded, however, that there is limited evidence to show that surgery will truly change the natural course of this disabling condition. It remains true that, irrespective of the treatment given, a surprisingly high percentage of patients do not regain a full range of motion when the disease has run its course, and still suffer from minimal to moderate pain for an extended period.

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