Clinical review

Frozen shoulder

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Frozen shoulder is a painful, often prolonged, condition that requires careful clinical diagnosis and management. Patients usually recover, but they may never regain their full range of movement.

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Introduction

Frozen shoulder is a disabling and sometimes severely painful condition that is commonly managed in the primary care setting. True frozen shoulder has a protracted natural history that usually ends in resolution. In this article we consider how to diagnose frozen shoulder and how to distinguish it from other painful shoulder conditions. We also look at the current aetiological theories and the effectiveness of conservative and operative management. We reviewed the current literature on this topic and discussed papers of historical interest with consultants in our department. We have also made reference to key papers cited in *Clinical Evidence* (www.clinicalevidence.com).

What is frozen shoulder?

The term "frozen shoulder" was first introduced by Codman in 1934.[w1] He described a painful shoulder condition of insidious onset that was associated with stiffness and difficulty sleeping on the affected side. Codman also identified the marked reduction in forward elevation and external rotation that are the hallmarks of the disease.

Long before Codman, in 1872, the same condition had already been labelled "peri-arthritis" by Duplay.[w2] In 1945, Naviesar coined the term "adhesive capsulitis."[w3] Although still in use, this more recent term is unfortunate since, although a frozen shoulder is associated with synovitis and capsule contracture, it is not associated with capsular adhesions.

In clinical practice, the tendency is to label any patient with a stiff, painful shoulder as a case of frozen shoulder. This should be resisted. Frozen shoulder is a specific condition that has a natural history of spontaneous resolution and requires a management pathway that is completely different from such distinct shoulder conditions as a rotator cuff tear or osteoarthritis.

Who gets it?

Frozen shoulder patients usually present in the sixth decade of life, and onset before the age of 40 is very uncommon.[w4] The peak age is 56, and the condition occurs slightly more often in women than men.[1 w4] In 6-17% of patients, the other shoulder becomes affected, usually within five years, and after the first has resolved.[1 w4] The non-dominant shoulder is slightly more likely to be affected.[1 w4]

Few attempts have been made to calculate the cumulative lifetime risk of frozen shoulder. In the Scandinavian population at risk, it has been estimated at a minimum of 2% per year.[w4 w5] Recurrence is highly unusual.[w6]

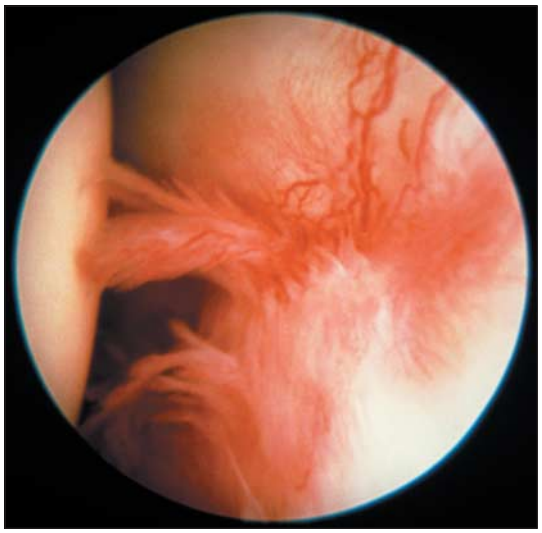
Summary points

- True frozen shoulder is a clinical diagnosis
- The three hallmarks of frozen shoulder are insidious shoulder stiffness; severe pain, even at night; and near complete loss of passive and active external rotation of the shoulder
- Lab tests are normal
- Frozen shoulder is rare under the age of 40; the peak age is 56
- Frozen shoulder progresses through three clinical phases
- It lasts about 30 months, but recovery can be accelerated by simple measures
- Physiotherapy alone is of little benefit, although steroid injection is effective and best combined with physiotherapy
- Refractory cases can be referred for manipulation under anaesthesia and, rarely, arthroscopic release
- Nearly all patients recover, but normal range of movement may never return

Clinical presentation and examination

A patient with frozen shoulder traditionally progresses through three overlapping phases (box).[2]

Additional references w1-w39 are on bmj.com



Arthroscope view of a shoulder with synovitis

When examining any joint, it is useful to apply the well known axiom of the late Alan Apley, a popular orthopaedic speaker and teacher: "Look, Feel, Move."

Look: On inspection, the arm is held by the side in adduction and internal rotation. Mild disuse atrophy of the deltoid and supraspinatus may be present.

Feel: On palpation, there is diffuse tenderness over the glenohumeral joint, and this extends to the trapezius and interscapular area owing to attempted splinting of the painful shoulder.

Move: In true frozen shoulder there is almost complete loss of external rotation. This is the pathognomonic sign of a frozen shoulder.[1 2 w1-w3] Confirming that external rotation is impossible with active and passive movements is important. For example, if external rotation was easily possible with the help of the doctor, we would consider the diagnosis of a large rotator cuff tear, which would require completely different management. In frozen shoulder, all other movements of the joint are reduced, and if movement occurs this usually comes from the thoracoscapular joint.

Three phases of clinical presentation

Painful freezing phase
Duration 10-36 weeks. Pain and stiffness around the shoulder with no history of injury. A nagging constant pain is worse at night, with little response to non-steroidal anti-inflammatory drugs

Adhesive phase
Occurs at 4-12 months. The pain gradually subsides but stiffness remains. Pain is apparent only at the extremes of movement. Gross reduction of glenohumeral movements, with near total obliteration of external rotation

Resolution phase
Takes 12-42 months. Follows the adhesive phase with spontaneous improvement in the range of movement. Mean duration from onset of frozen shoulder to the greatest resolution is over 30 months

What's the natural history of frozen shoulder?

Although the natural history of frozen shoulder is for ultimate resolution, this may not be complete. Reeves, in a prospective study of 41 patients with 5-10 years' follow-up, found that 39% had full recovery, 54% had clinical limitation without functional disability, and 7% had functional limitation.[2] Shaffer et al showed that 50% of his 61 patients with frozen shoulder had some degree of pain and stiffness an average of seven years after onset of the disease.[3]

Secondary frozen shoulder

Frozen shoulder can be a primary or idiopathic problem or it may be associated with another systemic illness. By far the most common association of a secondary frozen shoulder is diabetes mellitus.[4 w7 w8] The incidence of frozen shoulder in diabetes patients is reported to be 10%-36%.[4–6] The incidence in type 1 and type 2 diabetes is similar.[5] Unfortunately, frozen shoulder in diabetes is often more severe and is more resistant to treatment.[7] Moren-Hybbinette et al reported on the natural history of the diabetic painful stiff shoulder and found a restriction in the range of motion in 35 (65%) of 54 shoulders at a mean follow-up of 29 months.[8]

Bunker et al have shown an association with Dupuytren's disease in the hand, proposing that the contracting shoulder tissue itself represents a form of fibromatosis.[6 w9] Much more rarely, secondary frozen shoulder may be associated with conditions such as hyperthyroidism,[w10] hypothyroidism,[w11] and hypoadrenalism.[w12]

Additional associations include Parkinson's disease,[w13] cardiac disease, pulmonary disease, and stroke,[w14-w16] although the pathological condition here may be different from idiopathic frozen shoulder. Clearly, in the case of stroke, shoulder stiffness may be simply the result of muscle spasticity in the shoulder region.

Frozen shoulder has also been reported subsequent to non-shoulder surgical procedures, such as cardiac surgery,[w17] cardiac catheterisation through the brachial artery,[w18] neurosurgery,[w19] and radical neck dissection.[w20]

Laboratory investigations and radiology in frozen shoulder

There are few specific laboratory tests or radiological markers for frozen shoulder, and the diagnosis is essentially clinical. Immunological studies (such as human leucocyte antigen B27), C reactive protein, and erythrocyte sedimentation rate are all normal[w21-w23] and would be measured only to exclude other conditions. Most orthopaedic surgeons would not investigate a frozen shoulder beyond a plain x ray. When plain radiographs of the frozen shoulder are taken they may well be reported as normal, although they may show periarticular osteopenia as a result of disuse.[9 w24]

Contrast technetium-99m diphosphonate bone scan shows an increased uptake on the affected side in 92% of patients compared with the opposite side or with controls.[9] Arthrography shows characteristic find-

ings of limitation of capacity of the shoulder joint (5-10 ml compared with 25-30 ml in the normal joint) and a small or non-existent dependent axillary fold.[9 10 w24] However, in most units, arthrography is a historical investigation in frozen shoulder. Magnetic resonance imaging may show a slight thickening in the joint capsule and the coracohumeral ligament.[w25]

Pathogenesis

The aetiology of frozen shoulder remains unclear. The disease process particularly affects the anterosuperior joint capsule and the coracohumeral ligament.[11] Arthroscopy shows a small joint with loss of the axillary fold and tight anterior capsule, mild or moderate synovitis, and no adhesions.[12 w4 w26]

Neviaser and Neviaser have described an arthroscopic four stage classification for the frozen shoulder,[10] and Hannafin et al[w5] have described a correlation between the arthroscopic stage, the clinical examination, and the histological appearance of the tissues.

Disagreement prevails about whether the underlying pathological process is an inflammatory condition,[w5 w27] a fibrosing condition,[6] or even an algoneurodystrophic process.[w28]

Evidence shows a synovial inflammation with subsequent reactive capsular fibrosis. A dense matrix of type I and type III collagen is laid down by fibroblasts and myofibroblasts in the joint capsule. Subsequently, this tissue contracts.

Increased growth factors, cytokines, and expression of matrix metalloproteinases in capsular biopsy specimens obtained from patients with primary and secondary frozen shoulder indicate that these are involved in the inflammatory and fibrotic cascades seen in frozen shoulder.[w27 w29]

Cytokines and growth factors are involved in the initiation and termination of repair processes in musculoskeletal tissues through regulating fibroblasts, and the remodelling process is controlled by matrix metalloproteinases and their inhibitors.[w29 w30] An association between frozen shoulder and Dupuytren's disease has been identified,[6 13] and this may be related to matrix metalloproteinase inhibitors.[w31]

How should I treat it?

Educating patients helps to reduce frustration and encourages compliance. An explanation that the condition will spontaneously resolve and stiffness will greatly reduce helps. However, it is important to emphasise that the full range of motion may never recover. Ideally, the treatment of frozen shoulder should be tailored to the stage of the disease.

Treatment in the painful freezing phase

During the initial painful freezing stages, treatment is directed at pain relief. The patient is encouraged to use pain as a guide to limit activity, with all pain free activities allowed and all painful activities avoided.

It is traditional to give patients non-steroidal anti-inflammatory drugs (NSAIDs) if they can tolerate these. Where necessary these should be supplemented with other analgesics. There are, however, no randomised controlled trials that confirm the effectiveness of NSAIDs in the specific condition of frozen shoulder.

Physiotherapy

Dierks et al described a prospective study of 77 patients that compared exercise within the limits of pain with intensive physiotherapy in patients with frozen shoulder.[14] They found better results with exercise performed within the limits of pain (64% reached near normal, painless shoulder movements at 12 months and 89% at 24 months) than with intensive physiotherapy (63% achieved a similar result at 24 months).

Steroid injection

Hazelman performed a meta-analysis on the use of intra-articular steroids and reported that the success of the treatment depends on the duration of symptoms—patients who receive the injection earlier in the course of the disease recover more quickly.[15]

Early treatment with a steroid injection into the intra-articular glenohumeral joint may reduce the synovitis, thus shortening the natural history of the disease.[w5] De Jong et al have reported that the response to steroid injection is dose dependent.[16]

In a randomised placebo controlled trial, Carette et al compared the effectiveness of physiotherapy alone with a single intra-articular steroid injection given under x ray control.[17] This study also looked at patients treated with physiotherapy and steroid injection in combination and a fourth, placebo group treated with a saline injection. The authors concluded that when used alone, supervised physiotherapy is of limited benefit, but that a single steroid injection in combination with physiotherapy is effective in reducing both pain and disability associated with frozen shoulder.

X ray control is not normally available for a joint injection in primary care. However, in a separate study, Van der Wind et al showed that steroid injection by a general practitioner to be more effective than physiotherapy alone at six weeks.[18]

Other treatment modalities

Oral steroids have been proposed as a treatment for frozen shoulder: Buchbinder et al[19] described a double blind, randomised controlled trial on a series of 50 patients. In this study, oral steroids initially improved the frozen shoulder, but their effects did not last beyond six weeks. The adverse side effects of oral steroids are well documented, and they should not be regarded as routine treatment for this condition.

Suprascapular nerve blocks[20] may be beneficial in terms of pain relief (but not movement), and repeated joint distension may improve movement.[21 w32]

Treatment during the adhesive phase

Intra-articular steroid injections are not indicated in the adhesive phase as the inflammatory stage of the disease has passed. More aggressive stretching exercises will be tolerated and should be the focus of treatment, with the aim of regaining the range of motion. Low load, prolonged stretches produce plastic elongation of tissues as opposed to the high tensile resistance seen with high load, brief stretches.[22 w33]

Manipulation under anaesthesia

For patients who are unable to tolerate the pain and disability associated with the condition, manipulation under anaesthesia[23 24 w34 w35] is the most reliable way to improve the range of movement in a frozen shoulder. It is indicated if the functional disability persists in spite of adequate non-operative treatment for six months.[10 24 w36] Manipulation under anaesthesia generally results in notable improvement in shoulder function and range of motion within three months.[w34]

Surgical release

More recently, arthroscopic release of the capsule has been advocated to allow a more controlled release of the contracted capsule than manipulation under anaesthesia.[25 w37] This is required if manipulation fails to release the capsule, which is a common problem in frozen shoulder in diabetes.[w37 w38] Arthroscopic release also avoids reported complications associated with manipulation, such as fracture of the humerus[24] and iatrogenic, intra-articular shoulder lesions.[w39] Arthroscopic release and synovectomy in the painful freezing phase of the disease may be effective in controlling the progression of the disease, if synovitis is an essential factor in the development of frozen shoulder.[w5]

Conclusions

Frozen shoulder is a common, sometimes painful, but ultimately self limiting, condition that is usually managed in the primary care setting with a combination of analgesics, injections, and physiotherapy. Formal investigations are usually normal, and the diagnosis is essentially clinical. Most cases can be managed in the primary care setting. Educating patients plays an important part in the management of the condition. A minority of patients require referral to an orthopaedic specialist, where manipulation under anaesthesia is the most common method of treatment. Arthroscopic surgical release has proved itself to be useful in refractory cases. Irrespective of the treatment given, a high proportion of patients with frozen shoulder do not regain a full range of motion.

Contributors: RD proposed the article and mapped out an overview and also provided the illustrations. SC wrote the first draft and recruited SM, a consultant surgeon with a special interest in shoulder surgery, who rewrote the first draft and served as senior technical adviser on the project as a whole. SC is guarantor.

Competing interests: None.

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Misunderstandings

Misunderstandings happen in every profession, and, as an anatomy teacher, I have experienced a few. We have a counter in our dissection hall from where the students can borrow bones, books, and dissection equipment to use during their dissections. During one dissection class I asked a student to go and get a skull, atlas, and axis. The student duly returned with a skull, an axis vertebra, and *Grant's Atlas of Anatomy*. Actually, I expected him to get a skull, atlas vertebra, and axis vertebra. Since then, whenever I need the same bones, I ask students to get a skull and the first and second cervical vertebrae.

On another occasion, I had to explain to the students how to do a dissection. I told them to make an incision, cut the skin and throw it upwards, and then find the structures deep to it, referring to their *Cunningham's Manual of Practical Anatomy*. After a little time into the dissection, I heard a commotion at a table. This was because a student had literally cut the skin and thrown it up in the air, and it had fallen on another student's head. Since then, I tell students to make an incision, reflect the skin upwards, and find the structures deep to it.

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Hollingworth et al(3)compared placement of injections as compared to trigger point injection method in a randomized controlled double blind trial. The method of anatomical injection gave 60% success compared with the method using tender or trigger point localization, giving 20% success. In another study Eustace et al (4) studied the effect of accuracy on the clinical outcome of local steroid injections to the shoulder. There were significant differences in relation to outcome between the accurately placed and the inaccurately placed groups.

Very little is mentioned of treating frozen shoulder by distension in the present article.Treatment of frozen shoulder by distension seems to be an effective treatment. In a randomized controlled trial of 22 participants, Gam et al (5) compared intra-articular steroid injection with capsular distension with steroid and Lignocain by posterior approach. The VAS outcomes showed no difference between the treatments while in the distension group, range of motion (ROM) showed significant improvement in all directions except extension. Buchbinder et al (6) carried out a trial on 48 participants, to determine whether arthrographic distension, in patients with painful stiff shoulder for at least 3 months, is better than placebo in improving function, pain and range of motion at 3, 6, and 12 weeks. Shoulder joint distension was carried out with normal saline and corticosteroid, total volume (30-90 ml) and compared with placebo (artrhrogram). Both at 6 weeks and 12 weeks analysis for both intention to treat and an analysis excluding the four withdrawals demonstrated a significant improvement for the distension group (Buchbinder et al 2004).

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Mr Dias and his colleagues are to be congratulated on a good general overview of Frozen Shoulder. As one of the researchers cited twice in their article I would like to make three brief points.

The first is that the term Frozen Shoulder is still too frequently used and indeed misused. As they quite correctly say the key pathognomonic clinical sign is limitation of global passive movement of the shoulder, but in particular limitation of passive external rotation to less than half the asymptomatic side, in the presence of normal radiographs. This clinical picture is distinct, and with it a diagnosis of idiopathic frozen shoulder, or contracture can be given. The term contracture of the shoulder should be substituted for the old name of frozen shoulder for clinically that is what it is.

The pathology is no longer an enigma. We clearly demonstrated that the pathology is that of capsular fibrosis (Bunker and Anthony 1995), with collagen laid down in bundles and nodules within the shoulder capsule. The tissue was highly cellular with cells identified as fibroblasts and myofibroblasts and this has been confirmed by Killian and co-workers. Understanding the pathology allows us to be rational in terms of management.

In terms of treatment it really is time that we as doctors asked the patient what they would like from treatment. The common, dismissive neglect that is excused by the statement that the condition will get better in two years is no longer acceptable in the 21st Century. Patients do not want to suffer considerable pain, stiffness and in particular night awakening and be told that it will get better in two years. Ask patients what they want. I have and patients are quite consistent that they want four things. They want to be able to sleep at night, they want to be free of pain by day, they want to move their shoulder, but in particular they want this TOMORROW, and not in two years time!

Recent research (Berghs et al 2004) shows that 36% of patients can be relieved of their pain, night pain and stiffness the day following arthroscopic capsular release, and 80% within two weeks. Arthroscopic capsular release has transformed the management of this disease and it is time that this message was disseminated.It is a simple daycase minimally invasive procedure with proven results in the majority of patients who truly have this condition.

Bunker and Anthony, The pathology of frozen shoulder. Journal of Bone and Joint Surgery 1995; 77B: 677-683

Killian, Kreigsman, Berghauser. Die frozen shoulder. Der Chirurg 2001; 72: 1303-1308

Berghs, Sole Molins, Bunker. Arthroscopic release of frozen shoulder. Journal of Shoulder and Elbow Surgery. 2004; 13: 180-185