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.

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. 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. In 1945, Naviesar coined the term “adhesive capsulitis.” 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. The peak age is 56, and the condition occurs slightly more often in women than men. In 6-17% of patients, the other shoulder becomes affected, usually within five years, and after the first has resolved.

The non-dominant shoulder is slightly more likely to be affected.

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. Recurrence is highly unusual.

Clinical presentation and examination
A patient with frozen shoulder traditionally progresses through three overlapping phases (box).
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. Confirming that 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 16–36 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

**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 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 perarticular osteopenia as a result of disease.

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. Arthrogram 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. 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.

Pathogenesis
The aetiology of frozen shoulder remains unclear. The disease process particularly affects the anterosuperior joint capsule and the coracohumeral ligament. Arthroscopy shows a small joint with loss of the axillary fold and tight anterior capsule, mild or moderate synovitis, and no adhesions. Neviaser and Neviaser have described an arthroscopic four stage classification for the frozen shoulder, and Hannafin et al 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, a fibrosing condition, or even an algoneuromyodystrophic process.

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 capsule 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. 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. An association between frozen shoulder and Dupuytren's disease has been identified, and this may be related to matrix metalloproteinase inhibitors.

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. They found better results with exercise performed within the limits of pain (64% reached 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.

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

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. 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.

Other treatment modalities
Oral steroids have been proposed as a treatment for frozen shoulder: Buchbinder et al 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 may be beneficial in terms of pain relief (but not movement), and repeated joint distension may improve movement.

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.
Clinical review

Misunderstandings

Misunderstandings happen in every profession, and, as an anatomy teacher, I have experienced a few. We have a counter in the 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 Claren’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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