Frozen Shoulder – Unfreezing the Mystery

Abstract
Frozen shoulder is a common condition in the UK, and has historically been poorly understood. This condition of capsular contracture has received increased attention in the past few years, leading to a better understanding of pathology and course. There are several known associations of this disease that can promote development of the condition, but the causes of primary, or idiopathic frozen shoulder are hotly debated. Advances in treatment have recently been made, and with these advances has come a better chance of a quicker, more effective resolution of this troubling condition.

The term frozen shoulder is used to describe a glenohumeral joint that has undergone pathologic change that results in a stiffened, less mobile joint. Generally the movements that are impaired are rotation and abduction. It is usually a reversible condition. The frozen shoulder continues to be poorly understood in terms of pathology; indeed the very name has become the topic of scrutiny in recent years, with Bunker, Neviaser and others calling for a renaming of the condition to the more accurate contracture of the shoulder. Duplay, who coined the term periarthritis, which described an array of specific shoulder pathology, including frozen shoulder, initially identified the condition in the late 19th century. The condition is one of the most common GP presentations of shoulder pain, second only to impingement syndromes, but is in fact less common that previously thought. Research by Bunker shows that the real incidence of capsular contracture (the pathology behind the frozen shoulder) is 0.75% of the population. Codman, who first coined the term frozen shoulder stated that the condition is “difficult to define, difficult to treat, and difficult to explain”. This remains true today, with a lack of unity of opinion on definition and treatment of this condition.

To begin to understand the disease, it is first useful to briefly outline the anatomy and physiology of the shoulder, and more specifically which structures are affected in the case of frozen shoulder. The shoulder is composed primarily of three bones, the clavicle, the acromion and the humerus. The clavicle serves as the connection between the upper limb and the trunk and has two articulating ends, the sternal end,
and the acromial end. The sternal end communicates with the manubrium of the sternum at the sternoclavicular, or SC joint, and the acromial end with the acromion of the scapula at the acromioclavicular, or AC joint. The scapula is a flat, triangular bone, with several articulating surfaces: the acromion, articulating with the clavicle, and the glenoid cavity, which articulates with the humeral head at the glenohumeral joint. The muscles of the shoulder include the four muscles of the rotator cuff, and deltoid. The teres major is also a scapulohumeral muscle. All of the four rotator cuff muscles, namely the supraspinatus, infraspinatus, teres minor and subscapularis originate from various surfaces of the scapula; and all except the subscapularis insert onto the greater tubercle of the humerus. In the glenohumeral joint itself, there are two bursae (the subacromial and subscapular bursae) and several ligaments (the coracohumeral, transverse humeral and coracoacromial ligaments). The glenohumeral joint is visible in Figure 1.

Figure 1 - Glenohumeral joint. 1-subacromial bursa, 2-long head of biceps, 3-coracoid process, 4-superior glenohumeral ligament, 5-subscapularis, 6-middle glenohumeral joint, 7-inferior glenohumeral joint, 8-inferior joint capsule, 9-glenoid, 10-teres minor, 11-glenoid labrum, 12-infraspinatus, 13-deltoid.
Physiologically, the tendons of the rotator cuff blend with the joint capsule, which, along with bursae in the joint reduce friction on the tendons passing over the bone\textsuperscript{vii}. This organisation allows a vast array of movement, but this high level of mobility of the joint comes at the price of a certain level of instability. Although the joint is well supported by the rotator cuff tendons and glenohumeral ligaments, there is a deficiency inferiorly. The joint capsule is not supported inferiorly, so forms a structure called the inferior recess, visible in Figure 2, which comes under regular tension when the arm is abduced or elevated forward. After having briefly discussed the anatomy and physiology of the shoulder, it is now possible to describe the structures involved in frozen shoulder. The glenohumeral joint is the primarily affected site, as well as the glenohumeral bursae.

Figure 2 - Lax inferior recess visible on sagittal section of shoulder. 1-subdeltoid bursa, 2-humeral head, 3-tendon of biceps, 4-lax inferior recess.

Presentation of a frozen shoulder is often confused with a tendinopathy of one or more of the rotator cuff tendons. Patients often complain of difficulty reaching above
head height, and rotational difficulty manifesting in being unable to reach behind their back. These complaints also manifest in tendinopathy, but the reason for the lack of mobility is different. In tendinopathy patients are generally restricted by pain, rather than stiffness.

A frozen shoulder causes chronic pain. This is severely limiting in terms of movement, and far from indolent; indeed the painful manifestations of this disease can be the most distressing aspect of this condition and are commonly the reason for presentation. It most commonly presents between the ages of 40 and 60, with slightly more females than males affected. A presentation before age 40 is extremely rare, as is recurrence of frozen shoulder in the same shoulder.

The pathogenesis of frozen shoulder is poorly understood, but several theories prevail. Many authors have suggested an autoimmune component, but the majority of evidence has pointed to a fibroblastic condition, with inappropriate deposition of fibroblasts. The active process of fibroblast proliferation in frozen shoulder is very similar in appearance and pathogenesis to Dupuytren’s, and the expression of growth factors, such as VEGF promotes angiogenesis. This angiogenesis goes hand in hand with dense collagen deposition in the capsule. Research by the Oxford group suggested that an inflammatory trigger could possibly initiate the pathological developments.

Although the origination of these changes is unclear, there are definite pathological changes that can be described. Overall, the dense fibroblastic deposition leads to a thickened, contracted joint capsule. A reduced volume in the joint space is often noted, as well as a lax, under filled subscapular bursa and axillary fold. A decreased joint space can lead to impingement of the rotator cuff tendon as it traverses the subacromial space between the head of the humerus and the lateral acromion. Classically, this is the supraspinatus tendon.

The course of the condition is commonly divided into three phases:

1. **Freeze Phase**: This is the initial phase where the shoulder joint is painful and stiff. The movement of the shoulder is severely restricted, and the patient experiences significant pain.
2. **冻结期**: 这是关节开始疼痛和僵硬的最初阶段。肩关节的活动范围受到严重限制，患者感到剧烈的疼痛。
3. **Thaw Phase**: In this phase, the shoulder becomes less painful and more flexible. The patient starts to regain some range of motion, but still experiences some discomfort.
4. **恢复期**: 在这个阶段，肩关节变得不那么疼痛，更灵活。患者开始恢复一些活动范围，但仍会感到一些不适。
5. **Rest Phase**: This is the final phase where the shoulder is fully recovered and has regained its full range of motion. The patient is able to perform daily activities without any significant discomfort.
6. **恢复期**: 在这个阶段，肩关节完全恢复，活动范围完全恢复正常。患者能够进行日常活动而不会感到显著的不适。
1. **Painful freezing phase**: commonly lasts 10-36 weeks. The predominating symptom is pain, often with no obvious causative factor. This pain is exacerbated by essentially all movements of the shoulder, and pain is often worst at night when lying on the affected side. The mainstay of treatment in this phase is NSAIDs. Opiates are also prescribed in more serious cases.

2. **Stiff phase**: lasts for 4-12 months. During this phase the painful aspect of the condition eases, and the patient is left with severe stiffness. This can still be extremely debilitating, as a gross reduction of shoulder mobility is still present. Again, external rotation is most affected, with $0^\circ$ of rotation not uncommon.

3. **Resolution phase**: lasts 6-9 months, with a gradual return of mobility and decrease in pain.

It is a common myth that the resolution phase indeed brings total resolution. This is very often not the case, as demonstrated by various studies, including a study by Shaffer\textsuperscript{xvii}, which showed that 50% of his patients still had stiffness and/or mild pain after 7 years. Reeves concurs with this view in his findings, showing that a mere 39% of patients had a full recovery after a 5-10 year follow up\textsuperscript{xvi}.

There are several findings on examination that help define a diagnosis of frozen shoulder. One of the hallmarks of this condition is the restriction of external rotation, as well as a generalized restriction of motion as noted by Neviaser\textsuperscript{ii}, caused in part by anterior and inferior extraarticular contractures, described by MacKay\textsuperscript{xviii}. Using imaging techniques to diagnose this disease have limited use, as frozen shoulder is not visible on x-ray, however an x-ray may show periarticular osteopenia purely as a result of the lack of use of the joint\textsuperscript{xix}. The best method of direct visualisation is by using keyhole surgery. Arthrographically, these transformations are visible as a reduced capacity of the joint space\textsuperscript{xix}.

The causes and associations of frozen shoulder are disputed. According to Lundberg\textsuperscript{xx}, frozen shoulder can be primary, in the case of idiopathic, or secondary, with an obvious traumatic or surgical history prior to the development of their symptoms. Secondary frozen shoulder can also be linked to an associated systemic illness. Several associations have been noted. There is a well-identified link with
diabetes, and Bridgman states that more than 10-36% of diabetics develop a frozen shoulder\textsuperscript{xxi}. Indeed Bridgman is keen to suggest that all patients presenting with periarthritis of the shoulder should be screened for diabetes, especially if the pathology is bilateral\textsuperscript{xxi}. It is also worth noting that a diabetic frozen is generally much more serious and resistant to treatment\textsuperscript{xxii}.

Bunker is keen to emphasise the link between frozen shoulder and Dupuytren’s disease, stating that the pathophysiology of the two diseases are remarkably similar. As mentioned previously, the fibroblastic proliferation is seen to be very similar in nature to that of Dupuytren’s disease\textsuperscript{x}. In the same study, Bunker states that there is also an increased incidence of Dupuytren’s in diabetics, leading to the assumption that the three conditions are inherently linked.

Various authors suggesting a link between frozen shoulder and thyroid problems present less convincing evidence. Oldham described several patients with “periarthrosis of the shoulder” and hyperthyroidism\textsuperscript{xxiii}. Surgery for the affected thyroid soon lead to swift respite of the shoulder pain, compared to other patients who were treated solely with medication\textsuperscript{xxiii}.

The treatment of frozen shoulder is varied, the ultimate goal of all treatment being a relief from pain, a re-establishment of function, and a restoration of any quality of life lost. Management also depends entirely on the phase of the disease. In the initial painful phase, analgesics are the basis of treatment, and intensive physiotherapy in this phase has been shown to worsen and prolong overall recovery. This is indicated in a study by Stevens\textsuperscript{xxiv}, who separates the disease into four stages: inflammation, proliferation, fibrosis, and remodelling. He goes on to postulate that the aggressive physiotherapy may well have had a negative effect on the natural progression of the disease in the early “active” stages of the disease, previously referred to in this article as the “painful phase”. He also proposes the causative factor to be inflammation, aggravated by the physiotherapy.

Another common treatment is a steroid injection. This is a controversial treatment, as a recent study by Buchbinder showed no significant difference between the steroid and a placebo\textsuperscript{xxv}. However, another study by Blanchette et al\textsuperscript{xxvi} showed that a
combination of a capsular steroid injection and supervised physiotherapy did provide an improvement in shoulder mobility. This study also supported the proposition that the use of physiotherapy as the sole treatment is of little use, and perhaps contraindicated.

For pain relief in the active phase, an interesting development in treatment is the suprascapular nerve block. When combined with other methods this can provide extremely effective pain relief with a significant increase in passive movement immediately after the procedure xxvii.

There are two primary surgical treatments for frozen shoulder: manipulation under anaesthetic, and arthroscopic release, the latter having become the gold-standard treatment of choice. A basic illustration of the technique for arthroscopic release is shown in Figure 3. Manipulation has historically been the treatment of choice for a frozen shoulder that fails to respond to non-surgical intervention for an extended period of time. There is significant evidence supporting this method x, but more recently arthroscopic release has become more favoured. The increase in popularity of this method is for a number of reasons, including a greater reported relief of pain and increase in function with arthroscopy than manipulation xviii. Other studies have also found that the related complications associated with pure manipulation are almost entirely eliminated with the use of arthroscopic release xxviii. These include fractures of the humeral head and a risk of damage to intra-articular structures xxviii. It is important to be selective with the phase of the condition when considering arthroscopic surgery.
Figure 3 - (A) The arthroscope has been introduced through the posterior shoulder. The interval area between subscapularis and supraspinatus has been filled with inflammatory tissue, extending up the biceps tendon. The arthroscopic powered synovial resector has been introduced anteriorly to resect the inflammatory tissue. 1-humeral head, 2-biceps tendon, 3-inflamed tissue in interval area, 4-powered resector, 5-subscapularis, 6-anterior superior and middle glenohumeral ligaments, 7-glenoid, 8-infraglenoid recess, 9-inferior capsule, 10-anterior inferior glenohumeral ligament. (B) The synovial resector has been used to divide the superior and middle glenohumeral ligaments (6). The tendinous portion of the underlying subscapularis tendon can be seen (5). (C) The tendinous portion of subscapularis has been divided (5). The muscle fibers of the remaining 3 quarters of the subscapularis can be seen. (D) The inferior glenohumeral ligament has been divided (10). The synovial resector tip is now in the inferior glenoid recess (8). The inferior capsule is being divided carefully so as not to exit the shoulder capsule.
Arthroscopic release continues to gain popularity as a safe, effective procedure with fewer complications. It is particularly indicated in cases of frozen shoulder that is resistant to treatment. This is especially true with diabetic frozen shoulders, who tend to benefit more from arthroscopy than manipulation\textsuperscript{xviii}. However, arthroscopic release is not effective in every case, and some patients are resistant to all treatment. Prognostic indicators show that the rate of resolution of frozen shoulder has been historically overestimated. This is shown in a study by Shaffer, who found that 50\% of his patients maintained a loss of function, and continued pain after 7 years of onset of frozen shoulder\textsuperscript{xxx}.

In conclusion, it can be stated that this common condition has benefited significantly from an increased understanding in recent years. The involvement of fibroblastic proliferation in the thickening and contracture of the glenohumeral capsule has been proven, and treatment is widely available. It is a manageable condition, primarily governed in a GP setting where auto-resolution of the condition is the normality. Where necessary, the surgical treatment of choice is now arthroscopic capsular release which benefits more resistant cases as well as those with associated conditions. Despite an effective treatment regimen, a total return of mobility is not assured, despite common misconceptions.
Case Report - Mrs W

Mrs W is a healthy woman who last year developed a painful, stiff right shoulder. At age 55 she considered the possibility of osteoarthritis as a cause, but was reluctant to seek medical treatment. She took regular co-codamol to lessen her symptoms, but rarely found much relief. Having lived with worsening symptoms for several months, Mrs W opted to have treatment for the condition. This was in the time frame associated with the second phase of frozen shoulder, namely around the 6 month time period. She was successfully operated on in late December 2009, with an arthroscopic capsular release. She has, in the last couple of weeks, noticed a distinct improvement in her symptoms, more specifically an increase in mobility and a decrease in pain.

Being a manageress of a pub, Mrs W leads a very active lifestyle, with much use of the shoulder joint in her everyday activities. She described the onset of her symptoms as an ‘overworked arm’, with a tender lump on the top of the shoulder joint. This slowly progressed to a ‘tooth-ache’ like gnawing pain, with soreness radiating all the way down the arm to her knuckles. The symptoms worsened considerably following a family holiday abroad, where Mrs W made a conscious effort to avoid the use of her affected shoulder, effectively rendering it immobile for an extended period of time. On her return to the UK, there was a marked increase in the pain, and a significant decrease in the amount of mobility afforded to the joint. She found the pain to be worst after work and just before bed, with many sleepless nights, with minor improvement after having taken 2-3 co-codamol tablets.

Mrs W has no previous history of any shoulder pain, but a family history of systemic lupus erythematosis. This is not a condition normally linked to shoulder contracture, but Mrs W was advised to see a rheumatologist to rule out any autoimmunity. She has also received HRT for the past 5 years, which conversely may help to prevent arthritic change. Tests for Sjorgen’s disease were recently carried out, but the result was negative.

It took Mrs W several more weeks to eventually present to the A+E, after being brought in by her daughter in excruciating pain. Radiographs were taken, and Mrs W was informed that she had a classic frozen shoulder, presumably from the lack of
diagnostic information on the aforementioned x-ray. After having been diagnosed with a frozen shoulder, Mrs W decided to have treatment for the condition, and promptly saw a specialist orthopaedic surgeon. Together they decided that surgery was the best course of action, primarily because of Mrs W’s loss of her active lifestyle, and strong desire to regain it. She was made aware of the alternative treatments, including conservative management, but felt that a possible wait for auto resolution of 18-24 months was simply too long. She was subsequently consented for surgery.

Mrs W was operated on in late December 2009, with a full arthroscopic capsular release of the glenohumeral joint. In the days and weeks following the operation, no significant improvement was noticed, and the post-operative physiotherapy was poorly tolerated. However, Mrs W was resigned to ‘fighting through the pain’. This approach has recently been proven to be very beneficial, as in the last few weeks, a significant increase in mobility and decrease in pain and stiffness was reported. Mrs W was very satisfied with the outcome of the surgery, and has returned to working at the pub, as well as active relaxation with a recent swimming holiday abroad.

This case serves to highlight the effective treatment of frozen shoulder with a surgical intervention, namely a capsular release. In this case, surgery was more appropriate than conservative management, because of the significant drop in the patient’s quality of life. Furthermore, Mrs W’s case was a severe case of frozen shoulder, with many capsular contractions. Complete resolution of this pathology with non surgical methods would be expected to take many months or even years.
Case Report – Mrs D

Mrs D is a 61 year old retired woman who has recently been diagnosed with a right-sided frozen shoulder, after several months of suffering. She initially believed the pain to be attributable to a pulled muscle, but the pain worsened considerably over the following weeks and months, and she was eventually referred to a specialist orthopaedic surgeon. She is at the stage where treatment options are being discussed, and states that she will try anything to rid herself of the pain, including surgery.

Mrs D’s initial suspicions of a pulled muscle were thought to be correct when she visited her GP after several weeks of painful suffering. Her main symptom of right-sided shoulder radiating down her arm to her hand was very distressing, keeping her up at night. It also interfered with everyday tasks, such as the housework. She claimed to be constantly exhausted, being unable to obtain a comfortable position in bed. After taking paracetamol for the pain, and seeing only a worsening in her symptoms, Mrs D began to question the working diagnosis of a pulled muscle, and once more returned to her GP. This time she was referred for physiotherapy to try and ease the pain and stiffness. After a significant amount of physiotherapy, Mrs D found that this was only aggravating the pain, and so was referred to a specialist for a clearer diagnosis. The specialist subsequently told her that she was suffering from a frozen shoulder, for which there were a number of treatments.

Mrs D has an interesting past medical history, including hyperthyroidism, which was successfully treated with radioiodine. She is now on a maintenance dose of levothyroxine. The link between hyperthyroidism and frozen shoulder has been alluded to earlier in this article, and may well be of significance in this case. She also suffers from serious migraines, for which she takes regular analgesics. It is also interesting to note that the early physiotherapy indeed aggravated the symptoms of the frozen shoulder, as was shown to be the case in a study quoted earlier in this article. Mrs D is now at the stage where she must choose her path of treatment, and has indicated that she would be very keen to undergo a surgical treatment if it would cure her symptoms. This would most likely involve an arthroscopic capsular release performed under a general anaesthetic.
REFERENCES


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FIGURES

**Figure 1** – Glover J (2010). Redrawn from Surgical Exposures in Orthopaedics, Hoppenfeld, deBoer, Buckley (2009). Published by Lippincott Williams & Wilkins.

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