The Frozen Shoulder: Myths and Realities

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Abstract: Frozen shoulder is a common, disabling but self-limiting condition, which typically presents in three stages and ends in resolution. Frozen shoulder is classified as primary (idiopathic) or secondary cases. The aetiology for primary frozen shoulder remains unknown. It is frequently associated with other systemic conditions, most commonly diabetes mellitus, or following periods of immobilisation e.g. stroke disease. Frozen shoulder is usually diagnosed clinically requiring little investigation. Management is controversial and depends on the phase of the condition. Non-operative treatment options for frozen shoulder include analgesia, physiotherapy, oral or intra-articular corticosteroids, and intra-articular distension injections. Operative options include manipulation under anaesthesia and arthroscopic release and are generally reserved for refractory cases.

Keywords: Frozen shoulder, adhesive capsulitis, arthroscopic release.

INTRODUCTION

Frozen shoulder, also referred to as adhesive capsulitis, is a common condition affecting the glenohumeral joint characterised by progressive inflammation of the joint capsule and subsequent stiffness of the shoulder. The term frozen shoulder has in the past been used inaccurately as a general diagnosis for shoulder pain and stiffness. Precise definitions of this condition vary, as the aetiology, pathophysiology, and treatment are subjects of continued debate [1-3]. Associated with a protracted clinical course, but usually ending in resolution, the slow rate of recovery from what is frequently a disabling condition can be frustrating for patients as well as health care professionals [4, 5].

The term ‘frozen shoulder’ was first used by Dr Codman [6] in 1934 in his book named “The Shoulder: Rupture of the Supraspinatus Tendon and other Lesser Lesions in or about the Subacromial Bursa” in which he described frozen shoulder. He was the first to describe the classic diagnostic criteria for the condition, which include: idiopathic aetiology, global restriction in the range of movement of the shoulder, severe restriction of external rotation, painful at the outset, and normal plain X-ray findings.

Duplay was the first to recognize the condition as a pathology in its own right, referring to “periarthritis scapulohumeralae” leading to the widespread but inappropriate use of the term “periarthritis” [7]. Neviasier, in 1945, described “adhesive capsulitis” using the term “adhesive” to describe the texture and integrity of the inflamed capsule, which he thought was similar to sticking plaster [8]. The term is also inaccurate, as this condition is not associated with adhesions of the capsule, but rather is related to synovitis and progressive contracture of the capsule.

Shoulder pain is a common presentation to primary care doctors, with approximately one out of forty patients seeking medical advice for a painful shoulder. Frozen shoulder is one of the causes, as it is disabling and often presents with such severe pain that patients find it difficult to cope with. The incidence of frozen shoulder is approximately 4% in the population but affects up to 36% of patients suffering from diabetes. Female patients are more commonly affected, typically in the 5th to 7th decades of life. The condition affects both sides equally frequently, and rarely presents bilaterally in a simultaneous fashion. However, sequential bilateral occurrence may be found in up to 50% of patients [9-13].

Frozen shoulder can be classified either as primary (idiopathic) or secondary. The first group has a gradual onset and slow development of symptoms, where no obvious trigger mechanism can be found. Secondary cases are in general due to trauma or prolonged immobilization which may be due to a range of pathologies e.g. stroke [4, 12-14].

NATURAL HISTORY

Typically there three phases are seen as frozen shoulder progresses, described as “freezing, frozen and thawing”. These stages last for approximately two years, with a gradual initial onset over days or weeks. The initial phase (freezing) is characterised by marked pain and lasts approximately three months. The frozen (adhesive) phase lasts for 3-9 months, with significant stiffness and pain at the extremes of movement. The thawing (resolution) phase lasts for 9-18 months, is relatively painless, with stiffness improving steadily during this phase. Several authors have described frozen shoulder as a self-limiting condition that resolves in 12-36 months; [4, 5, 9, 12, 13, 15]. However, other reports have shown a more variable prognosis, with resolution in many cases being incomplete within this timeframe, and up
to 50% of patients remaining symptomatic for up to ten years. In a prospective study of 41 patients followed up for up to 10 years, Reeves [4] reported that approximately 40% of patients demonstrated a full recovery. However, more than 50% had some clinical limitation of movement, without restriction of function. A further 7% had restriction of shoulder function [4]. Similar results were reported by Shaffer et al, who studied 61 patients in a prospective longitudinal study, with a mean follow up time of 7 years. They reported 50% of patients complained of pain or stiffness, and 60% had a reduction in range of motion on clinical review. 11% of cases demonstrated a functional deficit [16].

AETIOLOGY

The aetiology of primary adhesive capsulitis is still unknown. It is frequently associated with other systemic conditions, most commonly diabetes mellitus. The condition has been reported in 10-36% of diabetics, who are approximately 2-4 times more likely to develop a frozen shoulder than members of the general population. Insulin-dependent diabetics are at the highest risk, and the condition is often particularly severe in these cases [9, 11]. In addition to diabetes, adhesive capsulitis is seen commonly in thyroid disorders, Parkinson’s disease, and a range of cardiac and pulmonary diseases. Surgical procedures, such as radical neck dissection, neurosurgery and cardiac surgery can also trigger frozen shoulder, particular where patients are bedbound for prolonged periods of time [12, 17, 18].

Bunker et al. [19] studied histological samples of capsular tissue from patients with frozen shoulder and found a pathological picture comparable to Dupuytren's disease. The authors demonstrated an increase in local collagen production, myofibroblasts, and fibroplasia. It is worth noting that up to 60% of patients with primary idiopathic adhesive capsulitis have a history of Dupuytren’s disease [3, 12, 15, 17, 18], suggesting a fibroproliferative mechanism for the condition.

The predominant macroscopic findings are synovitis within the rotator interval, with thickening and contracture of the anterior capsule, especially the coracohumeral ligament (CHL) and the middle glenohumeral ligament (MGHL). This process leads to a reduced glenohumeral joint volume, and restricts movements of the shoulder, particularly affecting external rotation in neutral- (CHL) and mid- elevation (MGHL) [12, 19, 20].

CLINICAL FEATURES

On inspection, the patient often presents holding the arm in adduction and internal rotation. Sometimes, atrophy of the shoulder muscles can be found. On palpation, there can be diffuse tenderness along the shoulder joint. There is usually a global restriction of movements of the shoulder, painful in early and middle stages of disease. Of particular importance is an almost complete loss of external rotation, which is almost pathognomonic. This is confirmed by testing the active and, more importantly, the passive ranges of movement. [4, 12, 13, 21]. Adhesive capsulitis is generally a clinical diagnosis and normally does not require extensive investigations. Plain radiographs of the shoulder to exclude osteoarthritis of the joint or other pathology are usually sufficient. Blood tests, including infection markers, are normal in true frozen shoulder [13].

Arthrography historically was used to assess joint volume, and typically shows a reduction in glenohumeral joint volume. Normal glenohumeral joint volume is 25-30 ml, which may be reduced to 5-10 ml in severe cases. In more than 90% of cases, bone scanning demonstrates an increased uptake around the affected joint. Magnetic resonance imaging (MRI) demonstrates a thickening in the joint capsule and the affected ligaments, as well as signs of synovitis [5, 19, 20].

MANAGEMENT

Management is controversial and depends on the phase of the disease. Decision making is often based on quality of life and whether the patients are able to cope with the pain and/ or stiffness till its eventual resolution. Treatment options include a range of conservative and surgical measures. In a recent survey of UK health professionals [14], only 3% recommended surgical procedures for the initial painful “freezing” phase. For the second and third phases of frozen shoulder, nearly 50% of the respondents suggested surgical treatment (including MUA and capsular distension injections). Operative treatment (i.e. manipulation under anaesthesia or arthroscopic capsular release) are generally considered only after conservative measures have failed. Presently there is no consensus as to the appropriate time for intervention.

Conservative treatment options include oral non-steroidal anti-inflammatory preparations (NSAIDs) for symptomatic pain relief; which can be used in any stage of frozen shoulder. However, there is little evidence to suggest that the disease progression is affected. Physiotherapy forms the mainstay of early- and mid-stage disease. The majority of studies that have examined the role of physiotherapy have shown improvement in pain scores, functionality and range of motion [22-28]. Griggs et al. [22] performed a prospective outcome study of non-operative treatment, including physiotherapy and passive stretching, and followed up 77 patients over a two-year period. Nine out of ten patients had favourable results; only 10% were dissatisfied with the outcome. It is worth noting that, compensation claims and pending lawsuits were linked to a need for further operative treatment. These results were echoed by Melzer et al. [23] studied 110 shoulders over a 3.8-year period and found that patients receiving physiotherapy alone had better clinical outcomes than patients undergoing MUA. However, in a prospective randomized study of 77 patients with frozen shoulder, Dierks et al. [17] demonstrated that supervised neglect provided better outcomes at 2 years when compared with an intensive physiotherapy regime, suggesting that physiotherapy may not alter disease progression, particularly if the regime is aggressive.

The role of oral corticosteroids has been examined in multiple studies. Most of these demonstrate a short term benefit with reduction in pain, especially during the early stages of frozen shoulder. Blockey [29] published his work on oral cortisone therapy for frozen shoulder in 1954 and found that patients receiving 200 mg cortisone followed by MUA had better results than patients undergoing MUA alone. Binder [5] demonstrated in a prospective study that
the daily administration of 10 mg Prednisolone for four weeks improved pain at night and patients had a more rapid initial recovery; nevertheless, after five months there was no difference when compared to the control group. These results were supported by Buchbinder et al. [30], who performed a prospective, randomized, double blinded, placebo-controlled trial, in 50 patients, administering 30 mg Prednisolone or placebo orally for three weeks. The authors demonstrated significant improvements in pain and functional scores at 3 and 6 weeks in the steroid group, but no benefit was seen beyond the 6 week mark.

The use of intra-articular steroids has been examined in a number of studies. Treatment regimes in these studies differ, but most studies demonstrate a short-term clinical benefit [24,25,31-34]. Bulgen et al. [24] conducted a prospective clinical study and evaluated three treatment regimens in 42 affected shoulders: subacromial corticosteroid injections, physiotherapy, and ice therapy, compared against a control group. They reported early clinical improvement in the steroid group, but no difference was demonstrated after six months. This is supported by the work of Carette et al. [25], who compared intra-articular corticosteroid injections, physiotherapy, and a combination of the two in a prospective comparative study. At six weeks he found that the steroid group has improved more than the other 2 groups, but at 12 months, however, there was no difference between the groups. In a further study, Ryans et al. [31] performed a randomized controlled trial of intra-articular steroid injection and physiotherapy in 80 shoulders, and showed early improvements in function in the steroid group; however, there was no difference at 16 weeks.

In 1965, Andren and Lundberg [35] performed the first arthrographic distension injection for adhesive capsulitis using 20 ml of contrast medium. Since then, different techniques for distension injection have been described using different substances, including combinations of saline, corticosteroids, local anaesthetic, and contrast. A number of randomized trials and published case series have reported promising results [36, 39]. However, the majority of studies examine small volumes of injected fluid, up to 20 ml, but have generally failed to demonstrate a significant lasting benefit.

In a prospective, randomized, double blind, placebo-controlled trial, Buchbinder et al. [36, 37] compared arthrographic distension using a combination of saline and steroids (total volume 30-90 ml) and placebo, at a mean follow up of 12 weeks. Early clinical results favoured the treatment group but differences were not significant by the 6th week and at 12 weeks results favoured the placebo group. A further Cochrane systematic review [36] has supported these results, demonstrating that arthrographic distension appears to have only limited short term benefit.

In a similar study, Jacobs et al. [38] conducted a prospective randomized trial, comparing MUA followed by physiotherapy (gold standard), with intra-articular steroid-with-distension (SWD) injections, at six week intervals, as an outpatient. Patients were followed up for two years. Similar clinical outcomes were seen in both groups, with the cost burden of patients in the surgery group being considerably higher. MUA remains a commonly used technique to improve the ROM of the shoulder in established disease. There is a small risk of humeral fracture, dislocation, rotator cuff injuries, labral tears and brachial plexus injury, as well as increased pain postoperatively, and patients should counselled appropriately. MUA is well accepted, is considered a cornerstone of treatment, and has been shown to allow return to a satisfactory range of movement in theatre. Concomitant physiotherapy is of particular importance following this procedure, as a proportion of patients do not achieve similar ranges of movement post-operatively as were achieved intra-operatively [23, 40-43].

For refractory cases, arthroscopic capsular release is a valuable treatment option. Good results have been provided by various authors, though many are retrospective and of small sample size. A number of systematic reviews on the subject have been largely inconclusive. The technique includes the release of the contracted capsule including the tight coracohumeral ligament, rotator interval and, the axillary pouch, using arthroscopic cautery instrumentation. This procedure can be combined with MUA, which is often necessary to promote access to the joint. Arthroscopy facilitates a direct visual assessment of the shoulder joint, and additional/ causative pathologies may be addressed simultaneously [41, 44, 45].

Frozen shoulder is, in most cases, a self-limiting condition of poorly understood aetiology. In the coming years it is likely that the prevalence of adhesive capsulitis will increase and clinicians will require robust prospective data to guide treatment. Much of the evidence for treatments in current practise suggests a short term benefit for several treatment modalities, but there is little data suggesting that either surgery or more conservative measures may be able to modify disease progression. Patients in the frozen and thawing phase may benefit from more intense physiotherapy and/or distension injections, whilst surgical intervention with MUA or arthroscopic release should be reserved for refractory cases. A greater number of prospective, comparative studies are required, with adequate medium and long-term follow up times, before clinicians will be able to provide reliable evidence-based treatment for a challenging condition.

ABBREVIATIONS

CHL = Coracohumeral ligament
MGHL = Middle glenohumeral ligament
MUA = Manipulation under anaesthesia
MRI = Magnetic resonance imaging
NSAIDs = Non-steroidal anti-inflammatories

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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