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Masterclass

Frozen shoulder contracture syndrome – Aetiology, diagnosis and management

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ABSTRACT

Frozen shoulder is a poorly understood condition that typically involves substantial pain, movement restriction, and considerable morbidity. Although function improves overtime, full and pain free range, may not be restored in everyone. Frozen shoulder is also known as adhesive capsulitis, however the evidence for capsular adhesions is refuted and arguably, this term should be abandoned. The aim of this Masterclass is to synthesise evidence to provide a framework for assessment and management for Frozen Shoulder. Although used in the treatment of this condition, manipulation under anaesthetic has been associated with joint damage and may be no more effective than physiotherapy. Capsular release is another surgical procedure that is supported by expert opinion and published case series, but currently high quality research is not available. Recommendations that supervised neglect is preferable to physiotherapy have been based on a quasi-experimental study associated with a high risk of bias. Physiotherapists in the United Kingdom have developed dedicated care pathways that provide; assessment, referral for imaging, education, health screening, ultrasound guided corticosteroid and hydro-distension injections, embedded within physiotherapy rehabilitation. The entire pathway is provided by physiotherapists and evidence exists to support each stage of the pathway. Substantial on-going research is required to better understand; epidemiology, patho-aetiology, assessment, best management, health economics, patient satisfaction and if possible prevention.

1. History and nomenclature

Duplay (1896) described the disabling combination of shoulder pain and restricted movement as péri-arthrite scapulo-humérale, attributing the condition to inflammation of the subacromial bursa. The term periarthritis of the shoulder has been used by others, both as a diagnosis and to explain the pathology (Dickson and Crosby, 1932; Wright and Haq, 1976). With the advent of radiographs, calcific deposits were observed, and for a period of time, the pain and stiffness was attributed by some to this newly observed phenomenon (Baer, 1907).

Codman, initially considered the condition to be an 'adherent subacromial bursitis', but after 15 years of clinical observation he rejected this in favour of the term frozen shoulder (Codman, 1934). He believed the condition involved a non-calcifying tendinitis of the rotator cuff, arguing that calcification represented a different pathology. During a one year period (approximately 1933), Codman treated four people suffering from frozen shoulder, and described the symptoms to consistently involve; slow onset (typically insidious, although trauma or strain may predispose), pain near the insertion of deltoid, inability to sleep on the affected side, painful and incomplete shoulder elevation and external rotation, and, with the exception of possible bone atrophy, normal shoulder radiographs. He added that although the aetiology remained uncertain, and the condition difficult to treat, the disorder would almost certainly resolve. To treat frozen shoulder, Codman advocated hospitalisation, with the arm constrained in elevation for one to two weeks. Patients were permitted to get up, out of bed, once a day to perform pendular exercises.

Lippmann (1943) supported many of Codman's observations, but argued that periarthritis or frozen shoulder resulted from inflammation of the long head of biceps tendon that eventuated in firm adhesions of the tendon to the bicipital sheath and bicipital groove. On the basis of intra-operative findings in 12 people, Lippmann argued the condition should be called bicipital tenosynovitis and clinically should be regarded as being similar to de Quervain's disease.

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Soon after this and based on a case series of 10 patients and observations of inflammation, fibrosis and contraction of the shoulder capsule, and with the axillary fold becoming 'adherent' to the humeral head, Neviaser (1945) suggested the term adhesive capsulitis better described the pathology. The adhesion was described as being similar to that of an adhesive plaster applied to the skin. Rotation and manipulation of the humerus was advocated to separate the adherent capsule from the humeral head. Later evidence suggested that thickening and contracture of the glenohumeral joint capsule was associated with frozen shoulder, *without* adhesions to the humerus (Wiley, 1991). Capsular adhesions have also not been reported in other investigations (Uitvlugt et al., 1993; Bunker and Anthony, 1995). The term adhesive capsulitis appears not to appropriately describe the condition and arguably should be abandoned.

Lungburg (1969) introduced the terms primary and secondary frozen shoulder, with primary being associated with idiopathic onset and secondary occurring following trauma, and forced inactivity following trauma. Others have further classified secondary frozen shoulder into; intrinsic, extrinsic and systemic categories (Zuckerman and Rokito, 2011). Conditions such as; calcific tendinosis, rotator cuff and biceps tendinopathy precede intrinsic secondary frozen shoulder. Shoulder surgery may result in iatrogenic intrinsic secondary frozen shoulder. Secondary extrinsic frozen shoulder is diagnosed when the condition is preceded by pathology remote from the shoulder, such as; humeral or clavicular fractures, cervical radiculopathy, ipsilateral breast surgery, chest wall tumour or cerebrovascular accident. Systemic secondary frozen shoulder occurs in the presence of conditions such as; diabetes, thyroid abnormalities, and heart disease. It is important to emphasise that currently a definitive relationship between many of these conditions, and frozen shoulder remains uncertain.

In Japan and China frozen shoulder is known as *the fifty year old shoulder*. This reflects the mean age of onset of the condition (Lundberg, 1969). The condition has been termed; *frozen shoulder syndrome* (Lundberg, 1969; Yang et al., 2007), and Bunker (2009) recommended the term *contracture of the shoulder* arguing this best incorporates the clinical and histological presentation. The multiple nomenclature used to describe this condition reflects poor understanding of the pathoaetiology, with the term frozen shoulder being described as a "waste-can diagnosis"(Neviaser and Neviaser, 1987) as it is often applied to any stiff and painful shoulder. In addition, the appellation frozen shoulder suggests the shoulder will eventually thaw, without the need for treatment. Not only may this belief lead to complacency, it may also be incorrect, as ongoing symptoms, 11 years post onset, have been reported (Shaffer et al., 1992). The recommendation for using the term contracture of the shoulder (Bunker, 2009) may be more suitable, but this may not reflect the often severe pain experienced with this condition, and as such, frozen shoulder contracture syndrome (FSCS), may more appropriately describe the condition.

Earlier clinical, operative and histological findings, often based on observational inferences from small studies with uncertain inclusion and exclusion criteria, have frequently been reported in later publications without critique of the quality of the earlier evidence, and as such, many 'truisms' relating to FSCS have become integrated into current clinical practice and this 'evidence' is often used to inform management and patient education. In fact, there is no certainty that women are affected more than men, and the true incidence and prevalence of FSCS remains unknown, as is certainty that the non-dominant side is more frequently involved than the dominant side. There is no certainty that onset relates to menopause in woman, or personality type (peri-arthritic personality). The number of people being affected bilaterally is often cited as 1 in 6 (17%), but this, alongside the belief that relapse in the same shoulder does not occur, also remain definitively unsubstantiated.

2. Pathoaetiology

The normal intra-articular volume of the glenohumeral joint has been reported to be between 15 and 35 cc and in FSCS the volume may reduce to 5–6 cc (Lundberg, 1969). Neviaser (1945) described an inflammatory (hence capsulitis) process, Lundberg (1969) did not report significant numbers of inflammatory cells, a finding supported by others (Bunker, 1997, 2009). However, others have suggested that the pathology associated with FSCS involves a chronic inflammatory response with fibroblastic proliferation (Hand et al., 2007).

Lundberg (1969) described the capsular changes to resemble Dupuytren's contracture. In addition, he reported that osteopenia was commonly observed in the humeral head of the affected side. Histological investigations of the coracohumeral ligament revealed nodules and laminae of dense tissue reported to be mature type III collagen, with a proliferation of fibroblasts and myofibroblasts (cells associated with contractile scar tissue). These histological and immunochemical changes have also been reported in Dupuytren's contracture. There has been a recent suggestion of an association between Propionibacterium acnes and frozen shoulder (Boyd et al., 2014). If proven, this may lead to a change in the understanding and management of this condition.

A summary of reported abnormalities include; thickening and fibrosis of the rotator interval, obliteration and scaring of the subscapular recess (area between biceps and subscapularis), neovascularity, increased cytokine concentrations, contraction of the anterior and inferior capsule (axillary recess), reduced joint volume, contraction and fibrosis of the coracohumeral ligament, proliferation of fibroblasts and myofibroblasts, presence of contractile proteins, and uncertainty regarding inflammatory changes. Adhesions of the capsule to the humeral head do not occur. The contracted tissue resembles Dupuytren's contracture. Neovascularity is present in the earlier stages of the disease and is found in the rotator interval, superior capsule, posterior capsule and the infraglenoid recess (De Palma, 1952; Lundberg, 1969; Ozaki et al., 1989; Neer et al., 1992; Bunker and Anthony, 1995; Bunker, 1997; Handa et al., 2003; Ryu et al., 2006; Uhthoff and Boileau, 2007; Bunker, 2009).

Dupuytren's contracture is classified under a group of tissue pathologies known as fibromatoses. There appears to be a high incidence of Dupuytren's contracture in people with FSCS (Smith et al., 2001; Degreef et al., 2008). Both FSCS and Dupuytren's contracture may occur without an identifiable precipitating event, and both are more common in people with diabetes (Smith et al., 2012).

Raised serum lipid levels have been reported both in FSCS (Salek et al., 2010) and in people with Dupuytren's contracture (Sanderson et al., 1992). However, this association, as well as the relationship between FSCS and thyroid disease and heart disease, is less certain (Smith et al., 2012).

Significantly greater plasma levels of substance P were reported in people who developed FSCS following shoulder surgery than those that didn't (Franceschi et al., 2008). In addition, substance P has been reported to accelerate angioneogenesis and hypercellularity in tendon (Andersson et al., 2011; Backman et al., 2011). Substance P in combination with interleukin 1α have also been shown to promote angioneogenesis (Fan et al., 1993) and high concentrations of interleukins have been identified in the capsule and subacromial bursa of people with FSCS (Lho et al., 2013). It is possible that these substances are involved in the pathogenesis of FSCS, and directing treatment at the neovascularity may contribute