Understanding frozen shoulder in the hemiparetic arm after stroke

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Abstract

Frozen shoulder is more common in the weakened hemiparetic shoulder post-stroke than in the general population. Increasing age, micro-vascular co-morbidities and the presence of subluxation make the soft tissue of the hemiparetic shoulder more susceptible to injury and inflammation. Inflammation can trigger fibrosis of the shoulder ligamentous capsule due to a disruption of joint homeostasis. It is this fibrosis that results in the common presenting features of frozen shoulder, namely restriction of passive shoulder external rotation, abduction and internal rotation.

Frozen shoulder is still largely a clinical diagnosis after assessment and exclusion of other possibilities. Spasticity of shoulder adductors and internal rotators is also common in hemiparesis and is hard to differentiate from frozen shoulder. Pectoralis Major is a common contributor to adductor and internal rotator tone and so diagnostic lateral pectoralis nerve blocks (DNBs) may help to establish if this is main cause of restriction. However, several muscles often contribute to shoulder adductor and internal rotator spasticity, so there is a risk of misinterpretation of DNB results.

In cases of frozen shoulder, daily movements of the shoulder joint within tolerable pain limits can help to restore joint homeostasis and reduce pain. Steroid injection (either alone or as part of a hydrodilatation injection) when inflammation is present can also reduce pain and improve range when used in combination with physiotherapy.

Background

Post-stroke shoulder pain (PSSP) is an umbrella term that includes all forms of pain that is perceived in the hemiparetic (weakened) shoulder and upper arm post-stroke [3,30]. It can affect around 50% of those with moderate weakness and around 80% of those with severe weakness[1]. PSSP should be detected and managed as soon as possible to reduce pain and avoid disengagement with early rehabilitation that can have a devastating impact on long term outcomes [2]. The

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terms post stroke shoulder pain and hemiplegic shoulder pain (HSP) are commonly used but are simply descriptive and do not point to the underlying contributory pathologies that represent potential therapeutic targets [3]. Stroke clinicians often lack confidence in identifying the underlying causes of PSSP, which in turn hampers effective management [4,5].

Shoulder pain results from a complex interaction of biopsychosocial processes [6], but for practical purposes the best starting point is to consider pathologies that are either local or remote (including referred pain) to the shoulder complex (Figure 1). This article will focus on understanding and treating frozen shoulder in the hemiparetic arm, a common pathology that influences structures local to the shoulder complex.

Frozen Shoulder clinical presentation

Frozen shoulder affects 2-5% of the general population and is most common in people between 40 – 60 years old [7,8,38]. In the hemiparetic shoulder after stroke it has been shown to account for between 41% to 88% of cases of pain [9,10,11]. Frozen shoulder is usually a clinical diagnosis characterised by (i) pain on movement and (ii) at least 50% restriction in passive external rotation of the shoulder compared to the non-paretic side [12]. Another diagnostic criterion that has been used is; at least 30% restriction in 2 out of 3 of passive movements; external rotation, internal rotation and abduction of the shoulder [13]. It is important to exclude bone pathology as a differential diagnosis of passive shoulder restriction with plain shoulder x-rays, especially if the subject has experienced trauma.

Frozen shoulder usually starts with a pain predominant phase, likely inflammatory, characterised by pain on movement and/or at night [8,14]. This is followed by a restriction predominant phase with reduced pain but with significant passive joint restriction [8,14]. Often, frozen shoulder is self-limiting and improves with time, though time scales can be highly variable and can be several years [14]. This means early intervention to treat frozen shoulder in stroke survivors is vital to prevent disruption to rehabilitation and helping to optimise upper limb recovery.

Frozen shoulder pathophysiology

What causes the observed restriction pattern?

The shoulder joint is anatomically complex. The glenohumeral joint has multiple degrees of freedom because of a small area of bone articulation [16] and joint stability is largely provided by muscle control and a ligamentous capsule [16]. For example, the axillary pouch of the capsule becomes tense in abduction and elevation to prevent excessive movement [17]. The coracohumeral ligament provides anterior stability [17]. In cases of frozen shoulder, fibrosis of these structures reduces the capsule volume and results in passive shoulder external rotation and abduction restriction [10]. In regard to other potentially affected structures; fibrosis of the superior glenohumeral ligament results in restriction of shoulder external rotation when the humerus is abducted to 90 degrees [18]. Inferior posterior capsule fibrosis can result in restriction of shoulder internal rotation [18].

Contrast enhanced MRI and arthrogram imaging of the hemiparetic frozen shoulder often show thickening in the coracohumeral ligament and joint capsule axillary pouch [9,10,].

What causes capsule fibrosis?

Rotator cuff tendinopathies and tears are more common in the hemiparetic shoulder [19,11] and associated age-related tendinosis may make tendons even more susceptible to injury [20]. Soft tissue injury creates a pro-inflammatory environment which can disrupt joint homeostasis within the shoulder capsule, especially in those with micro-vascular co-morbidities such as diabetes mellitus [21,22]. Advanced glycation end products (AGEs) which accumulate in shoulder tissues during ageing and as a result of diabetes, may also promote a pro-inflammation environment [38]. Inflammation can disrupt the balance of enzymes that regulate the joint extracellular matrix [21]. For example, matrix metalloproteinase (MMP) is responsible for degrading collagen as part of the joint remodelling process [21] and is regulated by the tissue inhibitor of matrix metalloproteinase (TIMP) [21]. In cases of frozen shoulder, MMP/TIMP ratios are reduced [23] resulting in collagen fibrils being continuously laid down leading to thickening of the ligamentous joint capsule. As well as fibrosis, inflammation may also trigger neo-angiogenesis and neo-innervation around the joint capsule [14,38]. This hyper-vascular synovitis near nerve endings may explain why frozen shoulder is so painful in the early stages [7].

The challenge of identifying cases of frozen shoulder in hemiparetic shoulder

Diagnostic criteria for frozen shoulder have been developed in the non-neurological population, but there are additional diagnostic challenges in the post-stroke hemiparetic arm. After stroke, another major cause of restriction of external rotation and abduction of the shoulder after stroke is spasticity in the shoulder internal rotators [10], which can develop within days of stroke when the arm is very weak [24]. Restriction can also be due to non-neural muscle and soft tissue shortening as well as altered movement patterns [25,26] making a single diagnosis difficult. To make things more challenging some stroke survivors with proximal shoulder motor activity can develop guarding in shoulder internal rotators/adductors. This guarding has been shown to mimic frozen shoulder restriction patterns in the general population [46]. These different causes of restriction often coexist and so a diagnostic hypothesis is usually a 'best guess' of the primary cause after careful assessment. Hypotheses can then be updated by

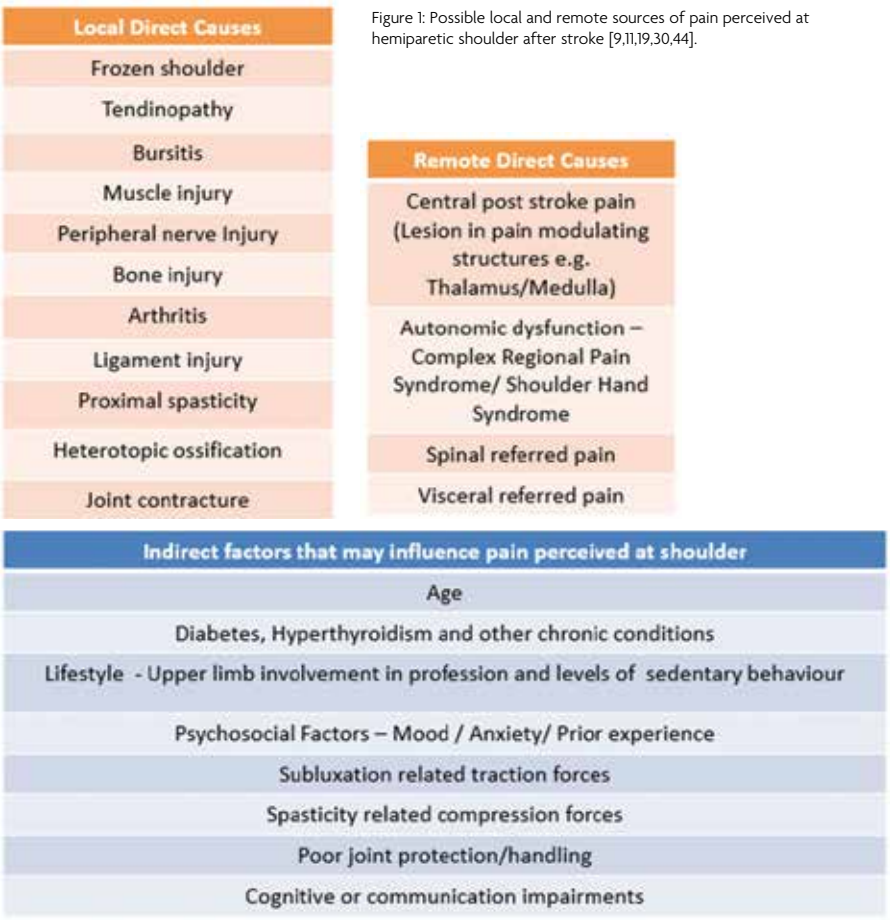

Figure 1: Possible local and remote sources of pain perceived at hemiparetic shoulder after stroke [9,11,19,30,44].

reassessment after initial treatment approaches are trialled.

If frozen shoulder is suspected from clinical assessment, evidence of glenohumeral joint effusion and thickening around the ligamentous capsule on soft tissue imaging (ultrasound or MRI) can add weight to the diagnosis of frozen shoulder [9].

Diagnostic Nerve Blocks

In cases where proximal spasticity of shoulder adductors and frozen shoulder occur together, identifying the dominant pathology may be aided by a diagnostic lateral pectoralis nerve block (DNB) [27]. The lateral pectoralis muscle supplies the pectoralis major muscle and so improvements in range after DNB indicate spasticity in the pectoralis major is the predominant cause of restriction. If range does not improve, frozen shoulder or contracture is then identified as the most likely cause of pain and restriction [27]. However, additional shoulder internal rotators/adductors such as subscapularis, latissimus dorsi and teres major are also known to develop spasticity post stroke [43]. These muscle groups would be unaffected by this nerve block, indicating that there is a risk of misinterpreting the results of DNBs.

Differentiating between frozen shoulder and proximal spasticity of shoulder internal rotators currently still relies on a detailed multidisciplinary assessment, which will establish a working diagnostic hypothesis. This is followed by trialling treatment approaches in a systematic way. Further research is required to refine diagnostic techniques to aide differentiation between these common presentations.

Treating Frozen Shoulder in the hemiparetic arm

Most of the rationale for treating hemiparetic frozen shoulder comes from experience in the general population. Education to prevent or reduce any fear of movement and promoting engagement in regular daily hemiparetic shoulder movements is key to improvements in symptoms and shoulder range [28,42]. These can be conducted independently or with the support of carers. In the general population with frozen shoulder, stretching several times a day into external and internal rotation, flexion and abduction improves pain, joint range and strength and is more effective than passive pendular exercise [23]. Stretching dosage will depend on an individual's pain tolerance, and pain irritability (length of time pain remains after movement) which will usually coincide with the suspected frozen shoulder phase (pain predominant versus restriction predominant) [38]. It may be necessary to start programmes more conservatively, for the first few days to build confidence. Analgesia should also be considered to assist tolerance of regular arm movement [38]. Care should be taken to optimise alignment in the presence of subluxation, with adequate support of the arm. This relies on training of the patient's support network to ensure movements are safe and appropriate.

Twelve weeks of stretching into tolerable pain for 10 seconds, 4 times a day has been shown to improve joint homeostasis in the general population by returning serum levels of MMP and TIMP levels to normal [23]. In stiffness predominant cases where there is no suspected inflammation, heat treatments in combination with passive stretching may help to improve range further [23]. The following injection treatments can provide a window of opportunity for physical interventions.

Intra-articular steroid injection has proved to be effective for pain reduction and subsequent functional improvements in the general population diagnosed with frozen shoulder [29]. Published literature for steroid injection in hemiparetic shoulders has shown mixed results [30]. However, pain presentations in these studies are often poorly defined, with hemiplegic shoulder pain (HSP) used as a blanket diagnosis [30]. Steroid injections may be more effective in pain predominant cases of hemiparetic frozen shoulder when inflammatory processes are particularly active.

Hydrodilatation/hydrodilation injections involve combining a local anaesthetic and a high volume of saline (usually between 20-30ml), with or without a steroid, to distend the capsule [14]. Hydrodilatation has a greater effect on reducing early pain compared to steroid alone in frozen shoulder cases in the general population but long-term functional improvements appear to be similar with both treatments [29]. Steroid injections can result in transient reductions in rotator cuff tensile strength and can influence tendon and collagen cell viability [31], but serious adverse events are rare [36,37]. Deciding whether an injection is appropriate should be guided by levels of pain on movement, at rest and overnight, and whether initial stretching alone is effective.

Suprascapular nerve block (SNB), can provide a window of pain relief to allow engagement in upper limb movement, especially in cases where steroid injection is not possible or indicated. This is because the suprascapular nerve is believed to supply around 70% of sensory innervation of the shoulder [39]. As spasticity is modulated by sensory inputs; SNB may also help to reduce local tone [28,41].

If restriction is significantly impacting on engagement in a stroke survivor's activities of daily living and the treatments discussed are ineffective there may then be a case for referring them for arthroscopic capsular release (ACR) or manipulation under anaesthetic (MUA) [47,48].

Finally, it is important that as pain improves, a progressive shoulder strengthening programme is established, especially involving activation of the rotator cuff [33]. Away from the shoulder, strengthening of the trunk and lower limbs in functional training can also help improve axioscapular muscle recruitment, which will likely help to prevent reactivation of frozen shoulder processes [35]. In cases where strengthening is not possible because of dense weakness, Neuromuscular Electrical Stimulation (NMES) is a good alternative to consider [30].

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Future Research

1. Improving diagnostic accuracy so treatments can be based on mechanistic principles: It would be beneficial for future clinical trials to target the underlying causes of restriction and pain in the hemiparetic arm. This might involve a combination of diagnostic nerve blocks, spasticity assessments, x-rays, ultrasound, blood tests or contrast enhanced MRIs [9,27]. Better understanding of the natural history and clinical presentation of different pain pathologies will help target treatments to appropriate patients based on mechanistic principles.
2. Early intervention: Reducing the time to treat leads to better outcomes and reduces chronicity of hemiparetic shoulder pain after stroke [28]. Future studies should focus on the first 3 months after stroke at a time when successful intervention could help prevent lasting contracture or joint fibrosis.
3. Systematic approaches to shoulder pain: Treatment pathways that guide management after assessment, have been shown to improve outcomes [45]. Published pathways have so far concentrated on rehabilitation settings, where cohorts are generally several months post stroke. It would now be useful to trial systematic approaches earlier in the stroke pathway, sooner after pain onset.
4. Anti-inflammatory drugs: There are no human trials targeting the MMP pathway, although this is an area of active research in preclinical models of frozen shoulder [34].

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