Evaluation of the Stiff Shoulder

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10.1 Introduction

Shoulder stiffness, presenting with or without pain, is a common expressing complaint in the routine orthopedic practice. Despite this, the actual definition of shoulder stiffness remains elusive. Most clinicians would agree that shoulder stiffness equates with loss of passive motion and does not describe the patient who cannot move it, or is unable to move the shoulder because of pain [1]. While shoulder pain can have a variety of sources outside the shoulder joint itself [1], true shoulder stiffness is invariably caused by intrinsic shoulder pathology. Codman was one of the first to explore the subject of atraumatic shoulder stiffness, coining the term "frozen shoulder" [2] to describe the condition later called "adhesive capsulitis" by Neviaser [3]. While Zuckerman [4] surveyed the American Shoulder and Elbow Society members to determine a consensus definition for this syndrome, others have used the Delphi consensus method [5]. The ISAKOS shoulder committee published a detailed guideline describing the terminology recommended for stiff shoulders [6]. This group stated that the term "stiff shoulder" should be used to describe the patient who presents with a restricted range of motion (ROM). The etiology of the stiff shoulder can be due to primary or secondary causes" [6]. Diercks et al. note a specific definition for the actual amount of stiffness required: "A range of motion of less than 100° in forward flexion, less than 10° in external rotation, and less than L5 level in internal rotation is indicative" [5].

Among the causes of stiff shoulder, common are idiopathic (adhesive capsulitis or frozen shoulder), rotator cuff pathology, glenohumeral arthritis, post-trauma, and post-surgery. While numerous causes for the stiff shoulder can be identified, the history, physical examination, and basic laboratory and radiographic studies can be used to diagnose and direct treatment of these patients accurately.

10.2 Patient History

Several authors have noted the importance of taking a thorough history to evaluate the patient presenting with a stiff shoulder [1, 5, 6]. This should include information regarding the initial onset

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and development of the stiffness and/or pain. Any history of trauma is essential in the characterization of the problem, as well as the onset being gradual or abrupt in nature. The history identifying any significant past medical history (e.g., diabetes, hypothyroidism, malignancy, neurological disease, cardiovascular disease, cerebrovascular accident, hyperlipidemia, and Dupuytren's contracture) and previous injuries should be noted. Accompanying symptoms of weakness and paresthesia are important as they indicate neurological etiology [3]. Night pain is common to many shoulder conditions consistent with the stiff shoulder and signifies substantial impairment in the patient's daily life. Prior treatment received is equally important, such as analgesics, physical therapy, steroid injection, or surgery, and the presence or absence of any benefit should be discussed. This history will help to direct the subsequent examination and evaluation.

10.3 Physical Examination

As with all orthopedic conditions, physical examination of the stiff shoulder requires observation of the shoulder, palpation, ROM, strength, and specialized examination tests. It must be emphasized that visual inspection should be performed from the front, back, and side of the patient, unclothed to the waist. The findings during shoulder examination should be compared to the opposite side [7]. Like the orthopedic examination of any joint, the joints proximal and distal to the involved one should be examined [8]. The resting posture of the shoulder and the pattern of any wasting that is present are important examination findings. Wasting points typically towards the chronic nature of the underlying disease and involved structures. The inspection will also reveal scars from prior injury or surgery. Any abnormalities in the form of weakness or sensory loss should trigger a complete neurologic examination. It is critical to rule out referred pain from the neck as a source of shoulder pain.

Passive and active ROM must be assessed, as this will differentiate the patients with a primary motor problem from those with a primary articular problem [6]. The ROM examination should always be compared to the normal shoulder. The four ROMs usually assessed are flexion, abduction, internal rotation, and external rotation. While these are generally evaluated with the patient sitting upright, flexion, abduction, and external rotation can be evaluated supine especially if pain is suspected to limit the examination.

Differentiation between pain-induced reduction in ROM and true "capsular" pathology can be difficult [5]. In idiopathic frozen shoulder, the loss of passive motion tends to be global. In contracture secondary to other causes such as trauma or spasticity, the movements may be restricted in one plane yet relatively preserved in another [6]. Documenting the unrestricted planes will help prevent an inappropriate release of tissues at the time of any surgical intervention. For some diagnoses, the assessment of internal and external rotation with the arm at 90° of abduction can also be helpful [1]. For example, glenohumeral internal rotation deficit (GIRD) can be common in throwing athletes. Fixing the scapula with one hand while performing active and passive motion can allow the examiner to eliminate scapulothoracic substitution in measuring motion [8]. Observing active motion allows assessment of the deltoid and peri-scapular muscle control. The presence of spasticity can be elicited during the evaluation of the passive motion. The strength of all rotator cuff muscles is assessed and the presence of any lag sign is noted [6]. Specialized tests for rotator cuff muscles like painful arc sign, Jobe's test, drop arm sign, belly press, and bear hug test should be performed [9].

10.4 Diagnostic Investigations

10.4.1 Laboratory Studies

There are no specific laboratory tests for the frozen shoulder [10]. Laboratory studies can elucidate secondary causes of shoulder stiffness. Measures of glucose metabolism, such as fasting blood sugar can detect diabetes. Serum hemoglobin A1C test can reveal poor glycemic control over a period of last 3 months. Elevated triglycerides can similarly be associated with shoulder

stiffness [10]. White blood cell count and inflammatory markers such as C reactive protein and erythrocyte sedimentation rate can provide a clue to subtle infection. While numerous other markers of the disease have been described, these are not generally clinically useful. Serum levels of ICAM-1, TIMP1, TIMP-2, and TGF-β1 were significantly higher, but MMP-1 and MMP-2 levels were significantly lower in frozen shoulder [10, 11].

10.4.2 Radiographs

Radiographs in both true anteroposterior (Grashey) and axillary views are essential for optimizing investigation of shoulder problems in general. Several potential diagnoses that lead to shoulder stiffness should have normal radiographs, including idiopathic adhesive capsulitis and rotator cuff tear. Two other common causes of stiff shoulders, osteoarthritis and calcific tendonitis, are easily diagnosed with plain radiographs. The physicians should be mindful of getting radiographs in patients with stiff shoulders as the literature continues to demonstrate cases followed conservatively with a late diagnosis of primary or metastatic malignancy [12]. Radiographs are especially of use in patients with post-surgical stiffness, where hardware placement often gives valuable clues to treatment.

10.4.3 Arthrogram

Although currently of limited use, Neviaser et al. have described the classic findings on arthrogram of loss of the inferior pouch and limited injection volume available when performing the arthrogram [3]. These findings are pathognomonic for adhesive capsulitis although they are also consistent with advanced glenohumeral arthritis which should be ruled out.

10.4.4 Magnetic Resonance Imaging (MRI) and Ultrasound Findings

MRI is rarely required to diagnose adhesive capsulitis [3]. Rather, it is used to exclude other etiologies of stiffness to assist the physician in the correct diagnosis and treatment. While not required, MRI findings in stiff shoulders in general, and idiopathic adhesive capsulitis in particular, have been well established [13–20]. The MRI findings that suggest adhesive capsulitis include soft tissue thickening in the rotator interval, which may encase the coracohumeral and superior glenohumeral ligaments, and soft tissue thickening adjacent to the bicipital groove. The typical MRI findings in patients suffering adhesive capsulitis depend on the stage of the disease. Peri-articular swelling and capsule inflammation are found at the initial period of the freezing stage (Fig. 10.1). Also, fibrosis of the rotator interval is

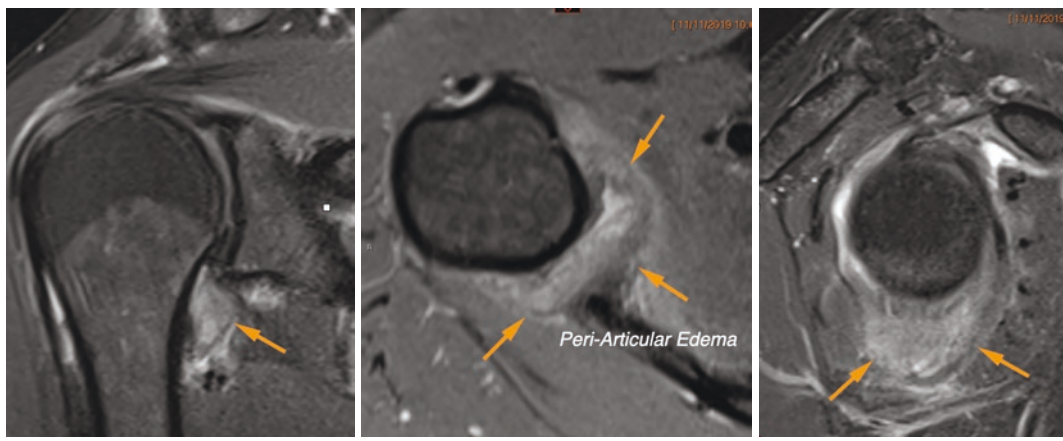

Fig. 10.1 Right shoulder MRI. Adhesive Capsulitis. DP-FS slices. Coronal, axial, and sagittal oblique cuts. The orange arrows indicate the ligaments swelling and capsular inflammation of the initial stage of adhesive capsulitis. (Courtesy F Idoate, Spain)

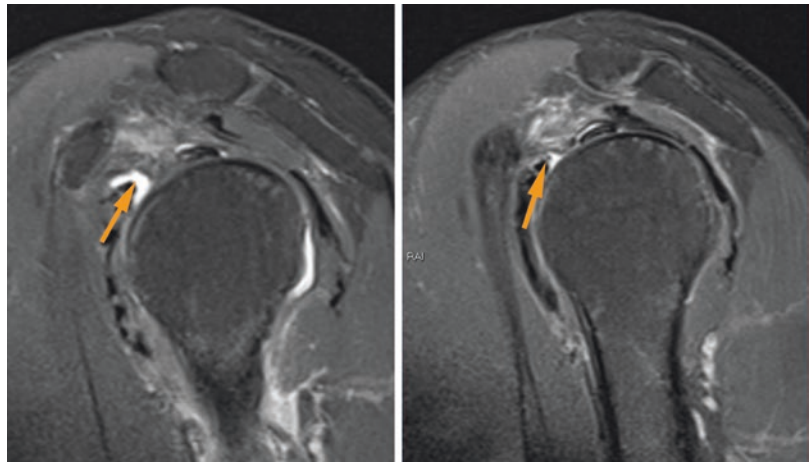

Fig. 10.2 Right shoulder MRI. Adhesive Capsulitis. Sagittal oblique DP-FS cuts. The orange arrows signpost the rotator interval tissue fibrosis between the coracoid tip and the bicipital groove. The coracohumeral ligament, which arises from the coracoid base to the proximal humerus, is also involved. (Courtesy F Idoate, Spain)

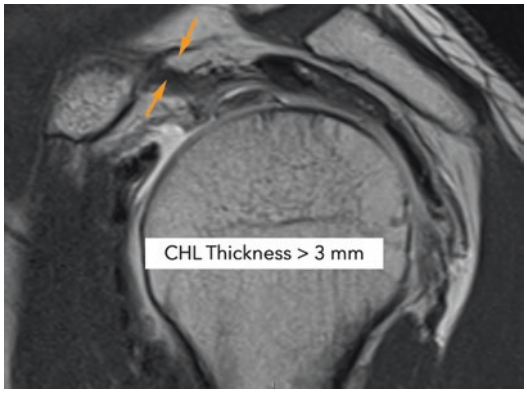

Fig. 10.3 Right shoulder MRI. Adhesive Capsulitis. Sagittal oblique T1 cut. The orange arrows indicate the thickened coracohumeral ligament. It runs from the coracoid to the bicipital groove, and when it has more than 3 mm wide strongly suggests a frozen shoulder syndrome. (Courtesy F Idoate, Spain)

a common finding at the sagittal fast spin-echo cuts demonstrating the origin of pain and the beginning of the motion loss (Fig. 10.2). After a few weeks of this freezing period, the coracohumeral ligament, a key component of the motion restriction, is shown thicker than usual. According to Homsi et al. [21], if the coracohumeral ligament is thicker than 3 mm at the sagittal oblique T1 cut, the diagnosis of frozen shoulder syndrome is ensured (Fig. 10.3) [21]. The range of motion is remarkably reduced in the final frozen stage by a thick and rigid inferior glenohumeral ligament (IGHL) and capsule. The average width of the capsule at the IGHL level is 2 mm. In severe adhesive capsulitis cases, the capsule thickness can reach between 4 and 7 mm wide (Fig. 10.4), [22–25]. It is important to note that the MRI in the patient with clinically established frozen shoulder can confuse, rather than assist, as Loeffler et al. has noted the high number of false-positive MRI scans in the presence of adhesive capsulitis [26]. Given a consistent clinical picture of the etiology of a stiff shoulder, MRI findings, in general, should not direct treatment. However, they need to be carefully ruled in or out during arthroscopic evaluation. Rotator cuff pathology in the presence of a clinical frozen shoulder is uncommon although the radiologic reading of partial rotator cuff tear is more so [19]. In the authors' experience, this finding is often false positive at arthroscopic evaluation secondary to cuff edema from the synovitis of frozen shoulder.

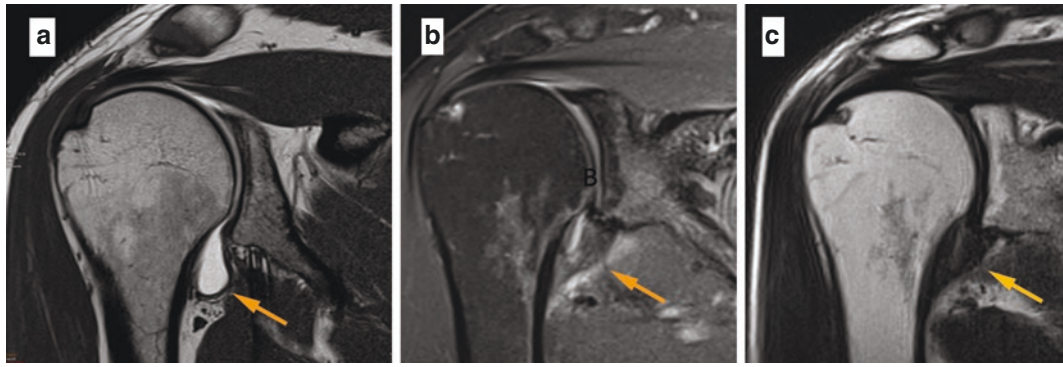

Fig. 10.4 Right shoulder MRI. Coronal slices. (**a**) Normal inferior glenohumeral ligament (IGHL) and capsule width of approximately 2 mm. (**b** and **c**) The orange arrows signpost the stiffened capsule and the thickened IGHL between 4 and 7 mm wide. (Courtesy F Idoate, Spain)

10.5 Etiology of Stiff Shoulder

As well described by Armstrong [1] the stiff shoulder can be broadly classified based on etiology into atraumatic and traumatic etiology. Other authors have divided joint stiffness into intrinsic (joint) and extrinsic (outside joint) [4, 27]. While this classification has some advantages, the primary cause of the stiff shoulder is alterations to the capsule. Bain et al. [6] further divided the causes of stiffness into intracapsular, capsular, and extracapsular (Table 10.1). The anatomic etiology of shoulder stiffness remains a point of discussion, the clinical classification into traumatic and atraumatic remains useful.

Bain et al. note that "The extra-articular causes are outside of the joint and include rotator cuff tendon and muscle. 'Other causes' are entirely separate from the shoulder. The systemic causes will affect a specific anatomical structure, e.g. diabetes causes capsulitis" [6].

In summary, the stiff shoulder can have multiple etiologies. While the diagnosis can be challenging, this can generally be accomplished with a thoughtful history and physical examination, given the etiologies described above. Plain radiographs are required. Increasingly, the pathology creating the stiff shoulder can be further evaluated with ultrasound and MRI imaging.

Table 10.1 Patho-anatomical classification of shoulder stiffness (Reproduced from Bain et al. 2015 [6])

Intra-articular (bearings)	Capsular (constraints)	Extra-articular (motor, cable, levers)	Neurological (control, electrics, sensors)
Articular surface	*Labrum and ligaments*	*Muscles*	*Central*
Osteochondral defect	Deficient	Myopathy	Behavioral, dyskinesia
Degeneration	Tear	Fatty infiltration	Dystonia
Subchondral	*Capsule*	*Tendons and bursa*	*UMN and LMN*
Dysplasia, fracture	Patulous capsule	Tear, calcification	Spasticity
AVN, degeneration	Capsulitis, contracture	degeneration, bursitis	Flaccid paralysis
Synovium	*Congruity*	*Other (external to shoulder)*	*Sensory and autonomic*
Inflammatory	Subluxation	Fracture, malignancy, HO	Charcot joint
Crystalopathy	Dislocation	Skin contracture	Chronic regional pain

AVN avascular necrosis; *HO* heterotopic ossification; *UMN* upper motor neuron; *LMN* lower motor neuron

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