Chapter 13
Frozen Shoulder

Junji Ide

Abstract Diagnosis of frozen shoulder is based upon the recognition of the characteristic features of the pain and limitation of both active and passive elevation, external rotation, and internal rotation. The macroscopic and histological features of the capsular contracture are well defined; however, the underlying pathological processes remain poorly understood. Furthermore, clearly defined diagnostic criteria are lacking. Contracture may cause protracted disability. Most patients are still managed by medication of nonsteroidal antiinflammatory drugs and physiotherapy in primary care, and only the more refractory cases are referred for specialist intervention. Targeted therapy is not possible, and treatment remains predominantly symptomatic. However, during the past 10 years, more active interventions that may shorten the clinical course, such as manipulation under anesthesia and arthroscopic capsular release, have become more popular.

Keywords Frozen shoulder • Manipulation under anaesthesia • Arthroscopic capsular release • Continuous passive motion machine

13.1 Introduction

Frozen shoulder is a common disorder in orthopedic practice, characterized by pain in the shoulder and physical restriction of movements of the glenohumeral joint. Frozen shoulder is a term coined by Codman in 1934 [1], who described the common features of a slow onset of pain felt near the insertion of the deltoid muscle, inability to sleep on the affected side with restriction in both active and passive elevation and external rotation, yet with normal radiographic appearance. Synonyms include pe'riarthrite scapulohume'rale [2] and adhesive capsulitis [3]. Although identification of the syndrome rests on the recognition of characteristic clinical features, clearly defined diagnostic criteria are lacking. Frozen shoulder may arise spontaneously without an obvious preceding cause, or may be

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Table 13.1 Definition of frozen shoulder

Symptoms	True (deltoid insertion) shoulder pain
	Night pain of insidious onset
Signs	Painful restriction of active and passive motion
	Passive elevation less than 100°
	Passive external rotation less than 30°
	Passive internal rotation less than L5
	All other shoulder conditions excluded
Investigations	Plain radiographs normal
	Arthroscopy shows vascular granulation tissue in the rotator interval

Table 13.2 Classification of frozen shoulder

Primary/idiopathic frozen shoulder
An underlying etiology or associated condition cannot be identified
Secondary frozen shoulder
An underlying etiology or associated condition can be identified
Intrinsic
In association with rotator cuff disorders (tendinitis and partial-thickness or full-thickness tears), biceps tendinitis, or calcific tendinitis
Extrinsic
In association with previous ipsilateral breast surgery, cervical radiculopathy, chest wall tumor, previous cerebrovascular accident, or more local extrinsic problems, including previous humeral shaft fracture, scapulothoracic abnormalities, acromioclavicular arthritis, or clavicle fracture
Systemic
Diabetes mellitus, hyperthyroidism, hypothyroidism, hypoadrenalism, etc.

associated with local or systemic disorders. The need for detection in diagnosis has recently been emphasized, and a system of terminology and classification based on consensus would be advantageous. A survey of the members of the British Elbow and Shoulder Society overwhelmingly agreed with the definition of frozen shoulder as seen in Table 13.1 [4]. In the United States, Zuckerman proposed to classify frozen shoulder into primary and secondary, and subdivided secondary frozen shoulder into intrinsic, extrinsic, and systemic ones [5] (Table 13.2). Diabetes mellitus is the condition most commonly associated with frozen shoulder, secondary systemic frozen shoulder. Diabetics have a 10–20 % lifetime risk of developing a frozen shoulder [6], with a risk two to four times greater than the general population [7].

13.2 Natural History

Many studies suggest that frozen shoulder is a benign condition, passing through phases of pain, pain and stiffness, stiffness and resolution, and typically leading to a functional recovery after 2–3 years [8, 9]. However, it is now accepted that up to 50 % of patients continued to have mild pain or stiffness 7 years after the initial symptoms as well as a deficit in shoulder range of motion compared with the contralateral shoulder [10]. It is estimated that approximately 7–15 % have some degree of permanent loss of movement, although few have persistent functional disability [11].

13.3 Pathology

The most common cause of painful restriction of movement is an idiopathic frozen shoulder, which is characterized by an inflammatory contracture of the capsule and ligaments, which reduces the available intraarticular volume, limiting glenohumeral movement. Macroscopically, the capsule has a glassy appearance with acute vasculitis, inflammation, and thickening, progressing to a more indolent fibrotic appearance with time.

Ide and Takagi reported intraarticular findings at arthroscopic capsular release [12]. All 42 patients (44 shoulders) manifested reduced intraarticular volume and highly vascular papillary infolding of the synovium (Fig. 13.1). Pathological findings, categorized as traumatic and nontraumatic frozen shoulder, are shown in Table 13.3. Regardless of etiology, all shoulders had similar intraarticular findings.

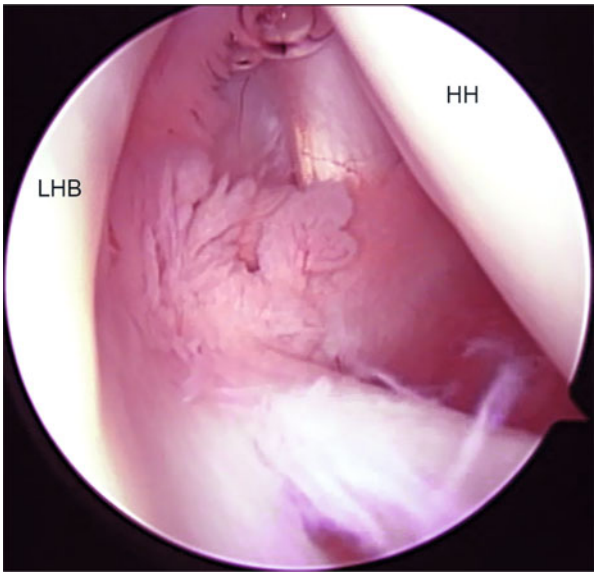

Fig. 13.1 Arthroscopic view of a right shoulder in patients with frozen shoulder from a posterior portal. Note the highly vascular papillary infolding of the synovium at rotator interval. LHB; long head of biceps tendon, HH; humeral head

Table 13.3 Arthroscopic findings of refractory frozen shoulder

	Nontraumatic ($n = 37$)	Traumatic ($n = 7$)
Highly vascular papillary infolding of		
Synovium	37 (100 %)	7 (100 %)
Rotator interval	36 (97 %)	7 (100 %)
LHB	25 (68 %)	6 (86 %)
Axillary pouch	22 (59 %)	4 (57 %)
Rotator cuff	21 (57 %)	2 (29 %)
Adhesion between LHB and rotator cuff		
	6 (16 %)	0 (0 %)
Incomplete rotator cuff tears	4 (11 %)	2 (29 %)
Labral lesions	3 (8 %)	1 (14 %)
Traumatic lesions of articular cartilage		
	0 (0 %)	0 (0 %)

After capsular release, capsule thickening was observed. The pathology of this condition is a soft tissue fibrosing and inflammation. There are no 'adhesions' within the joint. Future studies should be directed to give light on the initiator of inflammation, as well as of fibrosis, with the final aim to better treat or prevent frozen shoulder.

Histological examinations of synovial and capsular biopsies in patients with frozen shoulder demonstrate synovial hyperplasia with a normal underlying capsule (Fig. 13.2).

13.4 Imaging

Radiographic appearance is normal in patients with frozen shoulder. Anteroposterior radiograph in active elevation indicates that there is no glenohumeral joint movement (Fig. 13.3). The decrease of joint volume at arthrography indicates shortening of the joint capsule (Fig. 13.4). Magnetic resonance imaging (MRI) can detect thickening of the joint capsule. particularly in the axillary region [13, 14]. MRI also demonstrates thickening of the coracohumeral ligament [15]. MR arthrography may show obliteration of the subcoracoid fat triangle, resulting from shortening or fibrosis of the rotator interval capsule [15, 16]. Using dynamic MRI enhanced with intravenous gadolinium administration, Tamai et al. [17] demonstrated a greater increase of signal intensity in the glenohumeral joint synovium in frozen shoulder. This finding indicates an increased perfusion of gadolinium from the vessels to the synovium, which most probably is the result of synovial inflammation. The bone mineral density usually returns to near normal with the improvement of clinical symptoms [18]. A bone scan generally shows positive, which indicates increased local blood flow in frozen shoulder [19].

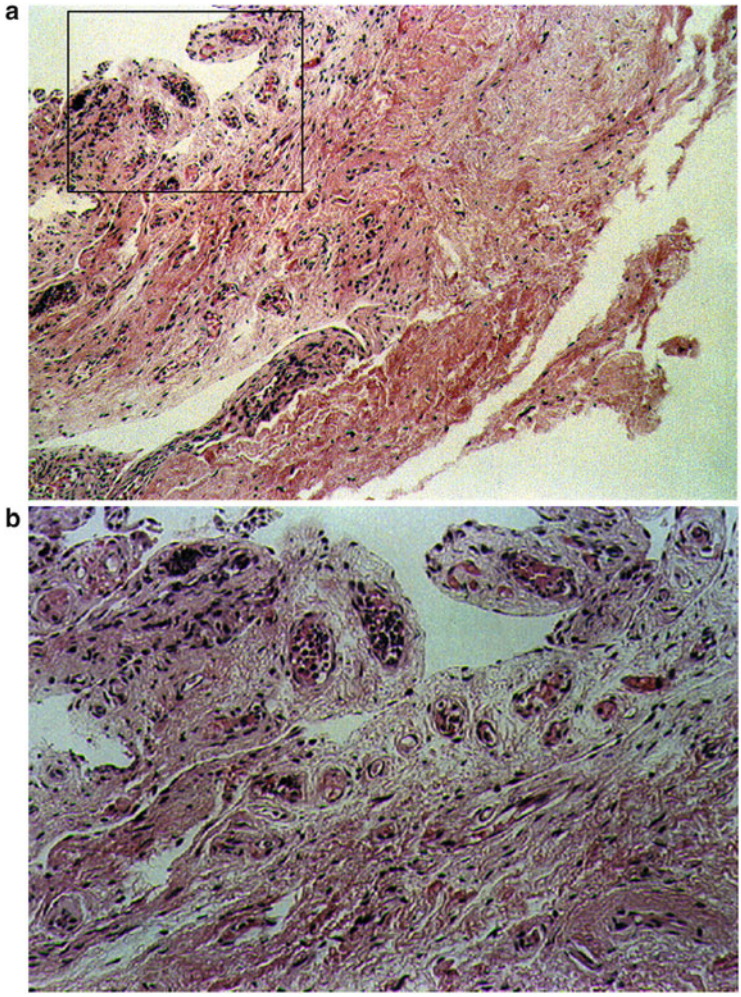

Fig. 13.2 Histological findings of synovial and capsular biopsies in patients with frozen shoulder demonstrate synovial hyperplasia with normal underlying capsule. (**a**) Hematoxylin and eosin staining, ×100. (**b**) Hematoxylin and eosin staining, ×400

13.5 Conservative Treatment

The goal of treatment is to relieve pain, restore movement, and regain function of the shoulder. There are many alternative forms of treatment for this condition, but evidence of their efficacy is not well established from clinical trials [20], and it is unclear if several interventions used in combination are better.

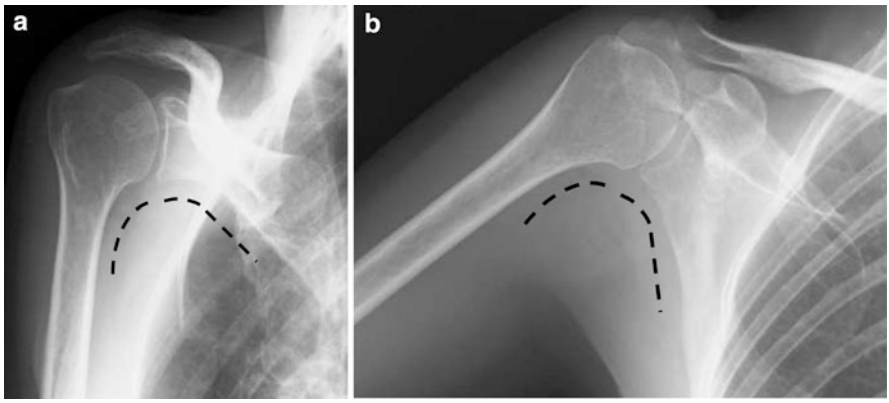

Fig. 13.3 (**a**) Radiographic appearance is normal in patients with frozen shoulder. (**b**) Anteroposterior radiograph in active elevation, indicating that there is no glenohumeral joint movement

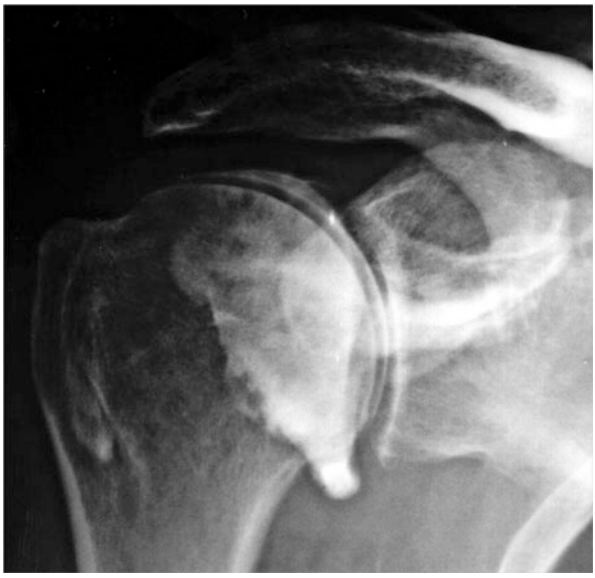

Fig. 13.4 Arthrography in patients with frozen shoulder, indicating the decrease of joint volume and shortening of the joint capsule

13.5.1 Physiotherapy

A recent Cochrane review concluded that the existing literature was insufficient to prove that physiotherapy alone was beneficial, with two small clinical trials concluding that physiotherapy alone did not offer any benefit when compared with no-treatment controls [21].

13.5.2 Steroid Injection

Although some studies have shown improvement with intraarticular steroid injection, others have found that this treatment produces little benefit [22]. A recent meta-analysis showed little evidence of benefit from steroid injection [23]. Steroid injections appear to provide earlier relief from pain, when compared with placebo, but whether this is sustained in the long term is unknown.

13.5.3 Distension Arthrography

This local anesthetic has the advantage of producing rapid improvement in movement, without recourse to a more interventional surgical procedure. Under fluoroscopic control, an arthrogram is initially performed to exclude a rotator cuff tear. The diagnosis of frozen shoulder is supported by the characteristic arthrographic appearance of a contracted capsule. Sterile water is then injected under pressure sufficient to cause capsular rupture. Data from a small placebo-controlled trial suggested that arthrographic distension provides significant short-term benefit, which is maintained in the medium term [24]. Further comparative studies are required to evaluate the efficacy of this technique.

13.5.4 Manipulation Under Anaesthesia

Manipulation under anesthesia (MUA) has been used extensively if physiotherapy fails. It has been successfully used alone or combined with a steroid injection or with an arthroscopic capsular release, and usually results in a rapid return of movement of the shoulder [25].

13.6 Surgical Treatment

My indications for arthroscopic treatment of shoulder stiffness are as follows [12]: limitation of active and passive range of motion, pain and dysfunction, at least 6 weeks of conservative treatment without progress, and symptoms for at least 3 months. Patients were subjected to arthroscopic capsular release if a closed manipulation did not restore at least 80 % of the range of motion of the normal, contralateral shoulder in all planes. It is my opinion that the major role of an arthroscopic treatment for shoulder stiffness is fast recovery and long-term efficacy.

I released the capsule using electrocautery from the anterior portal after diagnostic arthroscopy from the posterior portal in the lateral position. I did not release

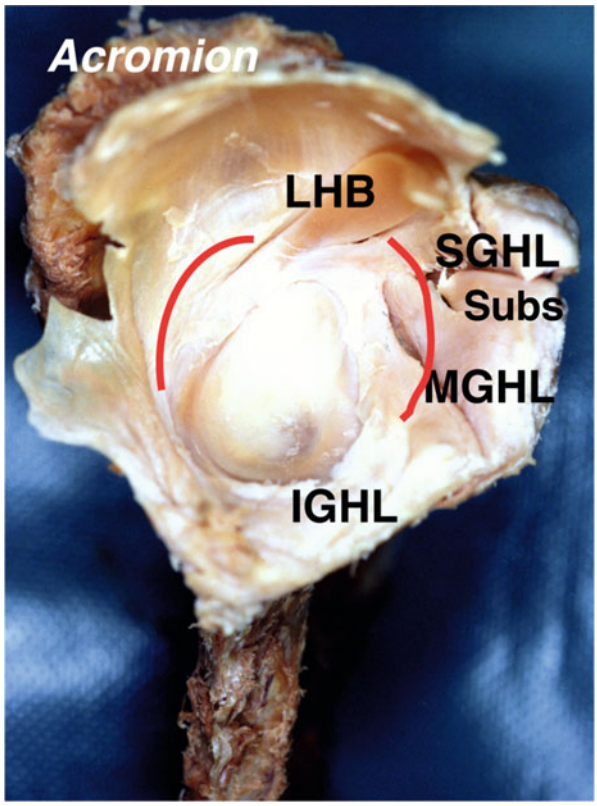

Fig. 13.5 Arthroscopic capsular release. Cadaveric specimen demonstrating capsular release (*red line*). Using electrocautery, we released the capsule including the superior glenohumeral ligament (*SGHL*) rotator interval, the middle glenohumeral ligament (*MGHL*), the anterior band of the inferior glenohumeral ligament (*IGHL*), the coracohumeral ligament extraarticularly, and/or the intraarticular portion of the subscapularis (*SubS*). To avoid axillary nerve injury, we did not release the inferior portion of the IGHL

the whole portion of the capsule, especially the inferior portion, if adequate range of motion was restored. A release of the superior and middle glenohumeral ligaments, the rotator interval, the coracohumeral ligament extraarticularly, or the intraarticular portion of the subscapularis was performed for loss of external rotation; a release of the anterior-inferior capsule including the anterior band of the inferior glenohumeral ligament was performed for loss of elevation; and a posterosuperior capsular release was performed for loss of internal rotation [12] (Fig. 13.5).

Preoperative pain and function of the shoulder joints in my patients were significantly improved at 4 weeks after the operation, and 91 % continued to be in good condition for a mean of 7.5 years [12]. There were no complications related to the arthroscopic procedure. I recommend selective arthroscopic capsular release for shoulder stiffness for which physiotherapy and manipulation under anesthesia have failed.

Intensive rehabilitation should begin immediately postoperatively with daily stretching exercises. Continuous passive motion (CPM) machines may also be useful to maintain movement, although controversy persists concerning the use of CPM machine exercise in patients with restricted shoulder motion. CPM exercise

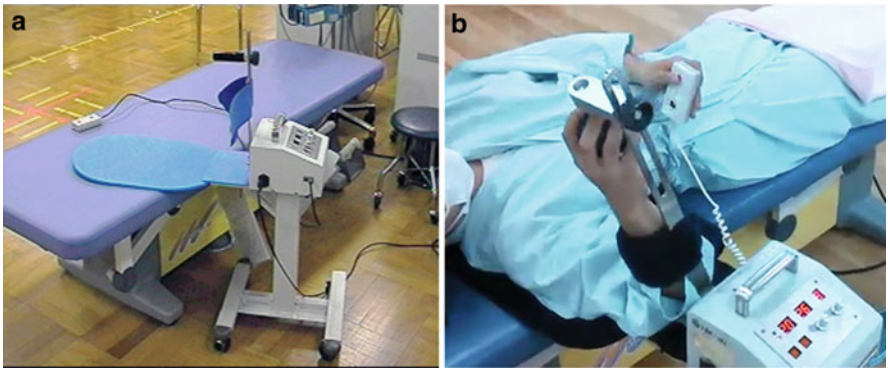

Fig. 13.6 (**a**) Continuous passive motion (CPM) machine providing for external and internal rotating motion of the shoulder joint (**b**) in abduction in the supine position, which supports scapular stability

may be useful in retaining the range of motion of the shoulder joint after restoring it by arthroscopic capsular release [12]. The CPM machine design in this study provides for external and internal rotating motion of the shoulder joint in abduction in the supine position, which supports scapular stability (Fig. 13.6).

The extent of capsular release depends on clinical judgement: some authors have advocated routinely performing a '360-degree' release [26], whereas others have adopted a more cautious approach [12, 27, 28]. Le Lievre and Murrell reported that shoulder range of motion at 7 years after 360° arthroscopic capsular release in patients with idiopathic adhesive capsulitis was equivalent to that in the contralateral shoulder, in contrast to results reported for nonoperative treatment [29]. Further investigation is needed to determine the optimal extent of the release.

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