Frozen Shoulder: A Brief Review

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Abstract
Frozen shoulder, also known as adhesive capsulitis of the shoulder, or arthrofibrosis shoulder,¹ It is a pathological process in which there is excessive formation of scar tissue or adhesions in the shoulder in the glenohumeral joint. This leads to stiffness, pain, and dysfunction.² Simon-Emmanuel Duplay is recognized as the first physician to describe this pathology, which he called “scapulohumeral periarthritis.”² Earnest Codman later composed the term “frozen shoulder” in 1934 to emphasize the debilitating loss of shoulder motion in patients suffering from this condition.³ Although for over a hundred years this disease is being treated, the definition, diagnosis, pathology, and most efficacious treatments are still very much ambiguous.⁴

Incidence
Frozen shoulder have an incidence of 3–5% in overall population but it has a higher incidence in diabetic patients with an incidence of about 20%.⁵ Its peak incidence is between the ages of 40 and 60 and it is fairly rare outside these ages.⁶ It is more in manual workers and women. Some predisposing factors are identified as trauma, hemiplegia, cervical discopathy, hyperthyroidism, hypercholesterolemia, etc.⁵

Natural Course
As per Neviser,⁶ the natural disease progression of adhesive capsulitis has been broken down into four stages. These stages are divided according to the basis of clinical presentation and arthroscopic appearance.¹⁶

In stage I, there is a primary complaint of shoulder pain, especially at night, but the patients have preserved motion in this stage. Arthroscopically, there is evidence of synovitis only but there are no adhesions or contractures inside the joint.¹,⁶

In stage II, the patients start to develop stiffness along with pain. Synovitis is again observed on arthroscopy, there is also some loss of the axillary fold, which suggests formation of early adhesion and contracture of the joint capsule.¹,⁶

Stage III is characterized by profound global loss of range of movement. There is pain at the extremes of active and passive motion. This stage is also known as the maturation stage. Here the synovitis of the joint is resolved but the axillary fold is obliterated, which indicates significant adhesions.¹,⁶

Finally, in stage IV or the chronic stage, the patient experience persistent stiffness but minimal pain, which indicates that the synovitis has resolved. With reduced pain, patients begin to exhibit gradual improvement in shoulder mobility. In this stage, advanced adhesions in the glenohumeral joint with restriction of the joint space are observed arthroscopically.¹,⁶

Applied Anatomy
The glenohumeral joint is a multiaxial synovial ball and socket joint. This joint functions as a diarthrosis, which allows a wide range of motion (ROM) for the upper extremities. Stability of the glenohumeral joint is balanced with the wide ROM by both static and dynamic mechanisms.⁷ The humeral head lies within the glenoid fossa, a cavity that is lined by the glenoid labrum. For the shallow nature of the glenoid fossa, the glenohumeral joint gets an increased ROM but this compromises the stability of the joint.
The bony structures are surrounded by the glenohumeral capsule, a fibrous network that is attached medially to the margin of the glenoid cavity and laterally to the anatomic neck of the humerus. Thickenings within the glenohumeral capsule include the superior, middle, and inferior glenohumeral ligaments (IGHLs). These are critical static stabilizers of the joint. The rotator cuff muscles cover the glenohumeral capsule and create balanced forces that play a pivotal role as dynamic stabilizers of the humeral head during movement (Fig. 1).  

Pathoanatomy

Contracture of the glenohumeral capsule is the main feature of adhesive capsulitis. The pathological findings include loss of the synovial layer of the capsule, adhesions of the axillary part of the capsule to itself and to the anatomical neck of the humerus, and overall decreased capsular volume. In particular, a thickened and fibrotic rotator interval is associated with adhesive capsulitis. The rotator interval is bordered superiorly by the supraspinatus tendon, inferiorly by the subscapularis tendon, laterally by the transhumeral ligament, and medially by the coracoid process. The rotator interval contains the coracohumeral ligament (CHL), biceps tendon, and the glenohumeral capsule. A contracted CHL is often considered as the essential finding in adhesive capsulitis. The CHL ligament is placed under tension with maximal external rotation; therefore, it is the main target of operative and interventional treatment of adhesive capsulitis. Patients with adhesive capsulitis have stiffer, thicker CHL ligament in the affected shoulder compared to the nonaffected side. The capsule in the rotator interval is thicker and the volume of the axillary recess is smaller in frozen shoulders compared to normal (Fig. 2).  

Pathogenesis

Typically frozen shoulder starts with a painful phase that leads to stiffness. This indicates that there is an initial inflammatory response, which later turns into a fibrotic reaction. Initially, there is active fibroblastic proliferation in the capsule of the shoulder joint, which in the later stage is accompanied by some transformation of fibroblasts to myofibroblasts. This causes an inflammatory contracture of the shoulder reducing the capsular volume and ultimately restricting glenohumeral movements. Current researches consider the role of matrix metalloproteinases as the key factor in the construction of the extracellular matrix. Various cytokines also control collagen deposition. The primary pathology can be correlated to contractures of individual structures in the capsule.  

Types

Frozen shoulder or adhesive capsulitis can be primary or secondary.  
- Primary (or idiopathic) adhesive capsulitis is one that occurs spontaneously without any history of trauma or inciting event.  
- Secondary adhesive capsulitis on the other hand has an underlying etiology that attributes to the development of frozen shoulder.  

Secondary frozen shoulder can be intrinsic, extrinsic, and systemic.  
- Intrinsic—in association with rotator cuff disorders, bicipital tendinitis, or calcific tendonitis.  
- Extrinsic—in association with breast surgery, cervical radiculopathy, chest wall tumor, fracture of glenohumeral joint, scapula thoracic abnormalities, acromioclavicular arthritis, arthroscopic procedure, CVA, etc.  
- Systemic—in association with thyroid disorders, diabetes, hypertension, etc.  

Clinical Features and Diagnosis

Frozen shoulder is mainly a diagnosis of exclusion. It is a clinical diagnosis made on the basis of medical history and physical examination. Other causes of a painful as well as stiff shoulder have to be excluded before a diagnosis of adhesive capsulitis is being reached. The causes may be rotator cuff pathology, septic arthritis, glenohumeral arthrosis, malposition of orthopedic hardware, fracture malunion, or cervical radiculopathy, etc.  

Clinically frozen shoulder can have three phases:  
- Freezing phase—first 10–36 weeks. Pain is main complain specially during night. The range of movement is reducing gradually. Nonsteroidal anti-inflammatory drugs are not much of help in reducing the pain.  
- Frozen phase—4–12 months. Pain gradually diminished but there is persistent stiffness.  
- Thawing phase—12–24 months sometimes up to 7 years. Stiffness gradually disappears and motion restored.
On history—Clinically, most of the patients first present with shoulder pain followed by gradual loss of both active and passive ROM. Pain is normally worse at the extremes of motion, as the contracted capsule is more stretched in this position.

On examination—Inspection and palpation findings are normal in absence of other pathologies like fracture dislocation or septic swollen arthritis. On range of movement findings, the passive range of movement is lost with painful endpoints of motion. Special clinical tests, like empty can test for supraspinatus, can be positive if rotator cuff tear or tendinopathy is present.

Laboratory testing—Routine blood investigation like complete blood count, erythrocyte sedimentation rate, C-reactive protein, thyroid profile, blood sugar, etc., to rule out medical causes.

Imaging—Imaging studies are not mandatory for the frozen shoulder diagnosis but those can be helpful to rule out other causes of a painful and stiff shoulder, which are already discussed.

Magnetic resonance imaging and magnetic resonance arthrography (MRA) may detect thickening of capsular tissues and pericapsular tissues along with a contracted glenohumeral joint space (Fig. 3).

Ultrasonography—Thickening or hypoechoic appearance of the coracohumeral ligament, thick axillary pouch, and presence of power Doppler signal in rotator interval are sometimes found in patient with adhesive capsulitis of the shoulder joint. Dynamic sonography is very helpful in detecting rotator cuff muscles tear or impingements and it can also detect thickening of the joint capsule (Fig. 4).

Arthrography—It involves intra-articular injection of the diluted iodinated contrast medium, which can help in both detecting and treating frozen shoulder. The following findings are suggestive of frozen shoulder:

- Decreased capsular distention, with irregular internal profile and internal septa (medial leakage of contrast)
- Minimum distention of subscapular bursa
- Atypical contrast leakage in the biceps sheath

**Differential Diagnoses**

- Calcifying tendinitis
- Cervical radiculopathy
- Fracture
- Glenohumeral osteoarthritis
- Glenohumeral synovitis
- Malignancy
- Polymyalgia rheumatica
- Polymyositis
- Rotator cuff tendinopathy
- Shoulder impingement
- Subacromial bursitis

![Coronal T1 (left) and PD-fat sat (right) MRI. Thickening (left) and intermediate signal (right) of the joint capsule in the axillary recess in a patient with adhesive capsulitis.](Image)

**Fig. 3:** MRI of frozen shoulder

Figs 4A to D: Ultrasonography of rotator cuff muscles during shoulder movements
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TREATMENT

Nonoperative

• Physiotherapy—Passive mobilization and capsular stretching are two of the most commonly used physiotherapy.17
• Hydrodilation (arthrography distention)—This treatment involves the injection of a local anesthetic into the capsule at a pressure high enough to distend and stretch the joint capsule.18
• Intra-articular platelet-rich plasma injection—Commonly approached through rotator cuff interval under ultrasonographic guidance from anterior approach.19
• Intra-articular hyaluronidase injection.20
• Suprascapular nerve block to reduce intensity of pain.21
• Steroid injection—There is at best a small short-term benefit to steroid injection alone for frozen shoulder but that the evidence base is poor.22
• Oral steroid—There is a mild short-term (under 6 weeks) benefit to oral steroid therapy.22
• Nonsteroidal anti-inflammatory drugs.23

Operative

• Arthroscopic or open capsular release24
• Manipulation under anesthesia25

CONCLUSION

Frozen shoulder being an entity since ages is yet to have fully standardized definition, diagnosis, and treatment. Although advanced imaging techniques give valuable information but cost-effective clinical methods are still the pillar to diagnose frozen shoulder. Also in treatment, physiotherapy is still being used as the first line of management but interventions under ultrasonography guidance and local nerve blocks are very much effective treatment modalities.

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