The management of stiff shoulder with rotator cuff lesions from pathomechanical point of view

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There are few reports on the management of rotator cuff tears with shoulder stiffness. Some authors suggest that in patients with rotator cuff tears with secondary shoulder stiffness, the shoulder stiffness should be treated initially because a rotator cuff repair is a “shoulder-tightening” procedure and might increase stiffness postoperatively. However, patients usually need to wait a significant length of time for improvement of shoulder motion before rotator cuff tears can be repaired. In addition, shoulder stiffness may not be relieved, especially in the presence of rotator cuff lesions.

The choice of treatment for shoulder stiffness should be tailored according to the duration and severity of symptoms. There are six treatment modalities including (1) supportive treatment, such as immobilization, heat, ice, ultrasound, massage, and others, (2) medications given orally, topically, or parenterally (local and intra-articular), such as nonsteroid anti-inflammatory agents, analgesics, narcotics, corticosteroids, and hyaluronates, (3) stretching exercises or traction, (4) injections of fluid, arthrographic dye, or medication for joint distention to release capsular contracture, (5) manipulative therapy with or without anesthesia to release adhesions or contracted structures, and (6) surgical release of adhesions by open or arthroscopic surgery.

Intrinsic degeneration and outlet acromial spur impingement are reportedly prominent pathological reactions in rotator cuff lesions. Most cases of complete tears of the rotator cuff are mainly complicated by partial tears in the lesions. We prospectively reported the etiology of rotator cuff lesions and demonstrated the pathologic changes in the acromion and rotator cuff and the surgical results of patients with partial tears of the rotator cuff. MRI of the rotator cuff has shown more severe pathologic changes in articular side tears than bursal side tears. On the contrary, bursal side tears have more severe histological changes in the acromion. The clinical vignettes indicate that articular side tears of the rotator cuff mainly associate with intrinsic degenerative changes, whereas bursal side tears are mainly caused by subacromial impingement on the underlying more
mildly degenerated rotator cuff.

Most pathomechanical studies have focused on the development of primary frozen shoulder. Lundberg found increased collagen deposits in the joint capsule and further proposed inflammation as an important reaction leading to stiffness, pain, and capsular fibrosis. Some investigators associated the fibrotic changes in the glenohumeral capsule to Dupuytren’s contracture in the palm, and mentioned the presence of an inflammatory component in the synovial and subsynovial layers, but absence of this component in the shoulder capsule. Bunker and Anthony reported active fibroblastic proliferation in association with myofibroblasts in the coracohumeral ligament and rotator interval in patients with frozen shoulder. During surgery, the release of a contracted rotator interval and coracohumeral ligament increases external rotation of the shoulder and excision of the adhesive subacromial bursa and lysis of the subacromial adhesions promotes shoulder motion. Adhesions are more severe in the subacromial space than in the glenohumeral joint. These intraoperative findings suggest that adhesions in the subacromial bursa are a potent cause of rotator cuff lesions with shoulder stiffness.

We found that patients with shoulder stiffness had increased IL-1β expression in the lesion tissue, in association with increased joint fluid IL-1β, IL-6 and TNF-α concentrations. Increased subacromial bursa and joint fluid IL-1β expression correlated with a preoperative deficit in shoulder motion and preoperative Constant scores. Patients with shoulder stiffness had increased myofibroblasts, however, those without shoulder stiffness had decreased myofibroblasts but increased apoptotic myofibroblasts in the subacromial synovium of the rotator cuff. We provided the first cellular and molecular indications that an increased IL-1β level and myofibroblast recruitment in the subacromial bursa at least partly contribute to rotator cuff lesion with shoulder stiffness.

There are increasing amount of publications reporting concomitant treatment of rotator cuff lesions and shoulder stiffness. In 2007, we reported a combined procedure of manipulation, lysis and debridement of adhesions, anterior acromioplasty, and repair of rotator cuff in 43 patients (47 shoulders) with a minimum follow-up of 2 years. Ten of these patients (11 shoulders) had diabetes mellitus. Twenty-seven shoulders displayed partial tears of the rotator cuff, 15 shoulders displayed complete tears and 5 shoulders showed massive tears. After a postoperative follow-up of 48.6 ± 18.0 months, each patient showed significant improvement in subjective, objective, and strength scoring as well as in the total Constant score. Comparison of the scores among
the three types of rotator cuff tears revealed that our modalities significantly improved the total function score. Moreover, patients with partial tears of the rotator cuff had significantly better scores than did those with complete tears or large tears. We also found increased interleukin-1β levels in the subacromial fluid in diabetic patients with rotator cuff lesions compared with nondiabetic patients. This finding may explain the likelihood of pain and shoulder stiffness developing in these patients.

Based on the clinical investigation, we suggest that aggressive non-surgical treatment is feasible first for patients with rotator cuff lesions and shoulder stiffness. A combined procedure of manipulation, lysis of adhesions, acromioplasty and repair of the rotator cuff is optionally recommended if symptoms do not improve after 3 months of aggressive rehabilitation.