

DIABETES ,ELLITUS AND THE FROZEN SHOULDER OR CAPSULAR FIBROPLASIA -- THE MYSTERY UNFOLDING

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INTRODUCTION

"This is a class of cases that I find difficult to define, difficult to treat and difficult to explain from the point of view of pathology." E.A. Codman, 1934[1]

The father of shoulder surgery expressed his frustration on the subject of frozen shoulder with these words. Sixty five years have passed since, however, little had been achieved until the arthroscope entered the shoulder joint and threw some light on the pathology. It is now well accepted that majority of painful shoulders develop stiffness secondary to an identifiable primary pathology e.g. rotator cuff tendonitis, rotator cuff tear or calcifying tendonitis. A relatively small percentage of patients, develop painful stiff shoulder without any obvious cause, what is known as Primary or True Frozen Shoulder. True frozen shoulder is an agonizing experience for the patient and a challenge to the attending doctor. Inability to locate the area of pathology has been the primary cause for the lack of effective and predictable treatment. The purpose of this paper is to provide, clinical data and arthroscopic findings of true frozen shoulder in order to help differentiate it from secondary capsulitis and report the results of excision of coracohumeral ligament for severe, refractory true frozen shoulder.

MATERIAL & METHODS

Records of two hundred and eighty painful stiff shoulders seen between 1994 and 1996 were available for review. Thirty (10.7%) of these were diagnosed as true frozen shoulder by the criteria described by Zukerman et al, 1994[2]. Four patients with true frozen shoulder were lost to follow up and were excluded from the study. There were 12 male and 14 female patients and the average age was 48.6 years (range 34 to 63 years). Eighteen patients were of type A (introvert, anxious) personality and 8 patients were type B (extrovert, care free). Non dominant arm was affected in 18 of the 26 patients (69.2%). The average duration of symptoms at the time of first consultation by the author was 6.7 months (range, 3 to 18 months). Eleven of the 26 patients were diabetic (42.6%). Eight of these diabetic patients underwent surgical treatment, one

improved with conservative treatment and two refused any intervention. None of the patients had a Dupuytren's contracture.

All the patients initially had conservative treatment including injecting 40 mg Triamcinolone acetate + 0.25% bupivacaine in the subacromial space followed by physiotherapy. Of the 26 patients, six responded to conservative treatment and the remaining 20 patients were offered surgical treatment. Six patients refused any form of intervention, whereas 14 patients that did not respond to minimum three months of conservative treatment, underwent surgery. Examination of range of movement under general anaesthesia and routine shoulder arthroscopy followed by an anterior deltopectoral approach for open excision of the coracohumeral ligament was performed, The rotator interval area was closed in all the patients, Histopathologic examination with hemotoxylin eosin stain of the excised coracohumeral ligament was performed. Pre and post operative Constant functional score of all the patients was documented (Constant and Murley, 1987}[3].

Constant score : Total 100 points

Pain	=	15 points
Range of Movement	=	40 points
Activities of daily living	=	20 points
Power	=	25 points

RESULTS

Conservative treatment group: Of the six patients that responded to conservative treatment, five were of type B and one patient was type A personality. None of these patients were diabetic. The average Constant score improved from 46(40-58) to 72 at the end of one year of conservative treatment. The patients that improved with conservative treatment were in the very early phase of the disease, the average duration of symptoms being less than four months. These patients were well motivated and were willing to perform exercises after some pain relief was provided by the local injection. Though the pain relief and improvement in movement was acceptable to the patient, the shoulders were not completely normal at the latest follow up.

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Operated group: 12 of the 14 patients (85.7%) that underwent surgery were of type A personality and two were type B. The duration of symptoms was more than six months. 57% of the operated patients were diabetic. The range of movement under anaesthesia, particularly external rotation, was the same as without anaesthesia.

Arthroscopy revealed difficulty in entering the joint. The capsule felt firm and tough to penetrate. Only 15-20 ml of fluid could be filled in the joint. There was villous synovitis in the rotator interval area in all the 14 patients. Rest of the joint capsule appeared red and congested. There was no obliteration of the inferior axillary pouch. No adhesions were found between the capsule and the articular surface.

By a small anterior approach the deltoid muscle was split. The coracoacromial ligament was partially cut in order to fully visualize the coracohumeral ligament. The coracohumeral ligament felt like a thickened cord, which tightened further on attempted external rotation. As soon as the coracohumeral ligament was excised, the improvement in external rotation was obvious. Pain relief, especially night pain, was immediate and dramatic, in all the operated patients. The range of movement improved gradually over six weeks of physiotherapy. The pre-operative Constant score of 38 (range 10-40) improved to 82 at the end of three months and was 94 at the end of 12 months. At three months post op, external rotation was 20 degrees (range 10-40) and forward elevation was 140 degrees (130-170). This improved to 40 degrees and 170 degrees respectively at the end of one year. Examination of the excised coracohumeral ligament revealed predominantly proliferation of fibroblast rather than of inflammatory cells.

At the end of one year, six patients that refused surgery were evaluated. There was no significant difference in the average Constant score. The range of movement was same as before. Pain had worsened in one patient, who was diabetic.

DISCUSSION

Frozen shoulder is difficult to define, particularly because the etiology is not known and the natural history can be varied. However there are definite predisposing factors and distinct clinical findings, which permit diagnosis of a true frozen shoulder with reasonable consistency. A typical patient is an anxious person, in fourth or fifth decade, with commonly non dominant arm involved, often diabetic, with insidious onset of true shoulder pain, particularly night pain, with painful limitation of

active and passive external rotation (<20 degree) and forward elevation (<90 degree) and a normal radiograph. Limitation of external rotation is most severe and consistent. On passive external rotation a sudden or abrupt block is appreciable in true frozen shoulder, as against a mushy or gradual limitation in secondary capsulitis.

It is rare to see a true frozen shoulder in young patients, less than 30 years of age. Non dominant arm was affected in almost 70% of the patients in this series. DePalma (1983)[4] described inactivity to be a major contributing factor. Painful stiff shoulder developing in paralytics is a reasonable evidence of dependency or inactivity having an important role. Correlation of frozen shoulder with diabetes mellitus is well documented[5, 6]. In this series 46% of the patients were diabetic. Diabetic patients have a 10% to 20% incidence of frozen shoulder and this rises to 36% in insulin-dependent diabetics. Diabetic patients with frozen shoulder are less likely to respond to conventional treatment[7]. This accounts for the 72.7% of the diabetic patients in our series requiring surgical intervention. Bunker et al (1995)[8] has implicated frozen shoulder to be a Dupuytren's like disease, however none of the patients in our series had either Dupuytren's disease or diabetic cheirarthropathy.

Though the clinical course can be very variable, the acute stage seems to pass through two phases. The first 3-4 months are very painful however, reasonable passive movement, including external rotation, may still be possible. During this phase, it may not be easy to differentiate true frozen shoulder from other causes of painful shoulder. The second half of the acute phase is as painful but is associated with limitation of movement, particularly external rotation. The duration of the second phase of the acute state is unpredictable. In some patients the disease process may halt and the shoulder gradually reverts back to near normal. However, in others it may continue to be painful for several years, as many as eight years have been reported[4]. Though the condition is thought to be self limiting, the author is not entirely sure if all the patients become completely normal.

Almost all the structures of the shoulder joint have been blamed to be involved in the pathology of frozen shoulder. Duplay[9] implicated the subacromial bursa; Pasteur (1934) and Lippman (1943)[10] the biceps tendon; Codman (1934)[1] the supraspinatus; Macnab(1973)[11] and others[12] the rotator cuff; and Neviasser(1945)[13] the inferior capsule. All of these have been disproved as the primary focus of pathology, except Neviasser's

theory of adhesive capsulitis. However, arthroscopy has revealed that neither there is obliteration of the inferior capsular pouch nor there are adhesions between the articular surface of humeral head and the capsule, as suggested by Neviaser(1945)[12]. The arthroscopy findings of this study are similar to those described by others[14. 15]. Observations made from this study point towards the coracohumeral ligament as the focus of pathology. It matches what DePalma stated in 1952[16], "the coracohumeral ligament is converted into a tough inelastic band of fibrous tissue spanning the interval between the coracoid process and the tuberosities of the humerus. It acts as a powerful check-rein... division of the coracohumeral ligament allows early restoration of scapulohumeral motion." Primary involvement of this structure explains restriction of external rotation more than any other movement, It is interesting to note that the coracohumeral ligament is a double fold of the capsule and is not a true ligament by definition[17]. Rest of the capsule becomes involved later as the disease progresses.

Manipulation under anaesthesia is a crude method which does not specifically tackle the area of pathology. It can rupture the rotator cuff and can occasionally fracture the humerus[7]. The treatment should be aimed at eliminating or reducing pain. This gives an opportunity to the patient to attempt activity and thereby overcome stiffness and possibly arrest the disease process. In order to reduce pain it may be worth injecting the shoulder during the first three months of the disease. Once stiffness sets in, the benefit of injecting the shoulder reduces. Our results of excision of the coracohumeral ligament have been satisfactory, both subjectively and objectively. Similar results have been reported by Ozaki et al, 21986[18] and Bunker 1995[8]. Why excision of the coracohumeral ligament relieves pain is difficult to explain, but the possibility of heavy concentration of pain receptors in that region remains to be explored.

By convention, most painful stiff shoulders are labeled as frozen shoulders and this tradition condemns the patient to a form of treatment which is neither scientific nor logical. The term frozen shoulder neither reflects the underlying pathology nor suggest the prognosis. Capsular Fibroplasia of the shoulder might be a more appropriate and meaningful term to correctly describe true frozen shoulder.

Lastly, why many of these frozen shoulders, improve spontaneously, while some progress? Why inactivity (due to pain or otherwise) leads to proliferation of fibroblasts? These are some of the

questions that still remain to be answered and should be the focus of further research.

CONCLUSION

True frozen shoulder is an uncommon condition. Arthroscopy reveals villous synovitis, mainly in the rotator interval area. The primary area of pathology seems to be the coracohumeral ligament and rotator interval area. For those refractory to conservative treatment, excision of the coracohumeral ligament provides dramatic relief of pain. Proliferative fibroplasia is predominant on histology.

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