Idiopathic Adhesive Capsulitis: Long-Term Results of Conservative Treatment

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Abstract

Background: Adhesive capsulitis, also termed "frozen shoulder," is controversial by definition and diagnostic criteria that are not sufficiently understood. The clinical course of this condition is considered self-limiting and is divided into three clinical phases. Several treatment methods for adhesive capsulitis have been reported in the literature, none of which has proven superior to others.

Objectives: To evaluate the long-term follow-up of patients with idiopathic adhesive capsulitis who were treated conservatively.

Methods: We conducted a long-term follow-up (range 5.5–16 years, mean 9.2 years) of 54 patients suffering from idiopathic adhesive capsulitis. All patients were treated with physical therapy and non-steroidal anti-inflammatory drugs.

Results: An increased statistically significant improvement ($P < 0.00001$) was found between the first and last visits to the clinic in all measured movement directions: elevation and external and internal rotation.

Conclusions: Conservative treatment (physical therapy and NSAIDs) is a good long-term treatment regimen for idiopathic adhesive capsulitis.

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Adhesive capsulitis, also termed "frozen shoulder," is well-known but is inadequately understood and controversial in terms of definition and diagnostic criteria. This clinical condition was first described in 1872 by Duplay [1], who used the term "periarterite scapulo humerale" and considered manipulation under general anesthesia to be the treatment of choice. In 1934, Codman [2] used the term "frozen shoulder" to describe this condition. He stated that most cases resolved within 2 years without treatment. In 1945, J.S. Neviaser [3] coined the term "adhesive capsulitis" to reflect his findings at surgery and autopsy in patients treated for a painful, stiff shoulder. He reported two main findings: a) a tightly contracted, thickened joint capsule with relative absence of synovial fluid, and b) cellular changes of chronic inflammation with fibrotic and perivascular infiltration in the synovial layer of the capsule, consistent with a reparative inflammatory process. There were no specific abnormalities in the synovial layer itself. Neviaser's findings were subsequently confirmed by others [4] and contributed to our understanding of the pathology of adhesive capsulitis. The clinical course of this condition is considered self-limiting and is divided into three clinical phases:

- The painful phase – in this phase the main symptom is a gradual increase in pain that is typically worse at night and exacerbated by lying on the affected side. The patient prefers not to use the affected shoulder and by the time the condition has improved and is less painful, there is a marked deterioration in the global motion of the shoulder joint. This phase usually lasts between 2 and 9 months.
- The stiffness phase – a marked loss of global motion of the shoulder joint usually accompanied by alleviation of pain. This phase lasts between 4 and 12 months.
- The thawing phase – gradual improvement of the shoulder range of motion. This phase may last weeks or months.

Numerous clinical conditions and diseases are considered to be risk factors that contribute to the initiation of adhesive capsulitis. These include prolonged periods of immobility of the shoulder due to different etiologies, such as exacerbation of cervical pain, pain after overuse, among others [5,6]. Many patients suffering from adhesive capsulitis do not have any known risk factors and are considered to have idiopathic adhesive capsulitis.

Numerous methods of treatment for adhesive capsulitis have been reported, including benign neglect, chiropractic manipulation, oral corticosteroids, physical therapy exercises, manipulation under general anesthesia, scalenes block, intraarticular injection of corticosteroids, intraarticular injections of fluid volume, arthroscopic and open arthrolysis [7–18]. None of these treatments has proven superior to others. The present study was conducted to evaluate the long-term follow-up of patients with idiopathic adhesive capsulitis who were treated conservatively.

Patients and Methods

During the years 1984–1995, 110 patients (59 women and 51 men) with phase 1 and phase 2 idiopathic adhesive capsulitis in 118 shoulders were treated in our clinic. The average age at onset of the condition was 51.8 ± 9.7 years (range 31–82 years, median 51). Fifty-four patients (28 females and 26 males) with idiopathic adhesive capsulitis in 58 shoulders were available for long-term evaluation. The mean follow-up period was 9.2 ± 2.5 years (range 5.5–16 years, median 8.9). The mean time interval between onset of pain due to "frozen shoulder" and the first visit to the clinic was 9.7 ± 6.7 months (range 1.5–24 months, median 9). The treatment

NSAIDs = non-steroidal anti-inflammatory drugs

regimen for all patients included at least one non-steroidal anti-inflammatory drug and a long-term course of physical therapy. No manipulation under general anesthesia was performed. Eleven patients who had had intraarticular steroid injections prior to the first checkup in the clinic were excluded from the study.

The inclusion criteria were: a) no history of major trauma or injury to the shoulder, although minor trauma to the shoulder or to the distal part of the affected upper extremity was acceptable; b) pain at the extreme of all motions; c) marked loss of active and passive global shoulder motion concomitant with at least 50% loss of range in external rotation; d) globally restricted glenohumeral translation; and e) absence of radiographic pathologic findings. Exclusion criteria were: a) any known risk factors, such as type 1 or 2 diabetes mellitus, history of minor trauma to the affected shoulder, or trauma to the distal part of the affected limb (elbow, forearm, wrist or hand) prior to the clinical appearance of adhesive capsulitis; b) any acute coronary event, post-coronary artery bypass or catheterization less than 3 months prior to the clinical appearance of adhesive capsulitis; and c) thyroid disease.

Each patient was evaluated clinically and by radiographs on the first visit to the clinic and during the subsequent follow-up period. Fifty percent of the patients were also evaluated by a Tc^{99} bone scan.

The pain was evaluated according to a grading scale of 0 to 15. The range of movement was compared to that of the contralateral shoulder and the results were expressed as a percentage of the normal shoulder. The patients were evaluated according to the constant score.

Two-tailed Student's *t*-test was used to evaluate the improvement of motion ranges of the affected shoulder.

Results

The results are summarized in Table 1. Only 6 (6%) of the patients also suffered adhesive capsulitis in the contralateral shoulder, but not simultaneously. A comparison between the first and last visits to the clinic showed a statistically significant improvement ($P < 0.00001$) in all measured movement directions, elevation, and external and internal rotation.

The majority of patients (with the exception of five) returned to their former employment or occupation. All apart from one regained their former normal lifestyle. Bone scans revealed a mild increase in uptake of the affected shoulder in 80% of the patients who were in phase 1 of the disease.

Table 1. Range of movement

		Mean ± SD	Median	Range
Elevation	Start	47.8 ± 18.1	50	20–80
	End	93.2 ± 11.7	100	50–100
	Change	45.4 ± 19.9	50	0–75
External rotation	Start	25.0 ± 14.0	25	0–50
	End	89.4 ± 17.6	90	20–100
	Change	64.8 ± 17.2	65	20–100
Internal rotation	Start	34.5 ± 15.6	40	0–70
	End	91.1 ± 11.9	90	30–100
	Change	56.6 ± 18.7	60	10–100

The results are expressed as a percentage, compared to the normal contralateral shoulder

Discussion

Adhesive capsulitis is a well-known, highly debatable disease. Diverse hypotheses exist regarding the pathologic mechanism of adhesive capsulitis, but the most accepted theory is the one described by J.S. Neviaser [3]. Although consensus exists regarding the three phases of the disease (pain, stiffness, and thawing), controversies still arise regarding the diagnostic criteria, as well as the preferred treatment protocol or modality.

The diagnostic criteria range from combined abduction (<135°) and combined elevation (<135°) coupled with pain and loss of movement in the glenohumeral joint [19], abduction (<45°) and total rotation (<50%) or <50% of external rotation [6,13]. There are numerous descriptions of the criteria, by Rizk et al. [20], R.J. Neviaser [5], and others.

The criteria we used were similar to those described by Binder et al. [7] and Lloyd-Roberts and French [21], i.e., pain at the extreme of all motions, marked loss of active and passive global shoulder motion coupled with at least a 50% external rotation range loss, and globally restricted glenohumeral translation.

The issue of treatment protocols or modalities is even more controversial. As previously mentioned, many known treatment modalities and methods have been reported in the literature, none of which proved superior to the others. Among these modalities are benign neglect, chiropractic manipulation, oral corticosteroids, physical therapy exercises and modalities, manipulation under general anesthesia, scalenes block, intraarticular injection of corticosteroids, and intraarticular injections of fluid volume, arthroscopic and open arthrolysis [7–18].

The treatment regimen for our patients combined physical therapy, mainly self-imposed home protocol exercises, and temporary administration of NSAIDs. The average follow-up period was 9.2 ± 2.5 years (range 5.5–16 years). A marked improvement was noted in all movement ranges (45.4% in elevation, 64.8% of external rotation and 56.6% of internal rotation). Meulengracht and Schwartz [22] reported a 15% disability in 71 patients, and Simmonds [23] described 15 of 21 patients (71%) who were symptomatic after 3 years. However, Dickson and Crosby [24] found that <5% of their patients were symptomatic after 6 months, and Gray [11] reported that 1 of 25 were symptomatic after 2 or more years of follow-up.

Fifty percent of our patients were also evaluated by a Tc^{99} bone scan to exclude other reasons for the condition, such as infections, minor undiagnosed trauma and tumors. The mild increase in uptake of the affected shoulder in 80% of the patients, who were in phase 1 of the disease, might be due to the inflammatory process in the first stage.

To the best of our knowledge, this is the first ever long-term follow-up study performed on patients with adhesive capsulitis. In our series 6% of the patients also suffered from adhesive capsulitis in the contralateral shoulder, but not simultaneously. Although the majority of patients had minor residual objective deficiency in some

range of movements, they were not symptomatic and most (90%) returned to their former work or occupation after recovery. Furthermore, there was no direct relationship between objective findings and subjective complaints, as previously reported by Binder et al. [25]. A point of interest is that 20 patients (37%) did not recall in which shoulder they had suffered pain, a fact that strongly supports the results of good recovery.

In conclusion, our study comprised a long-term follow-up of more than 9 years (range 5.5–16 years) in 54 patients with idiopathic adhesive capsulitis who were treated by physical therapy and NSAIDs. The objective (range of movement), functional and subjective results were very good. Although arthroscopy or other surgical procedures may shorten the morbidity period, they might cause complications. In our experience, the majority of patients who suffer from idiopathic adhesive capsulitis can be treated successfully by conservative means.

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Capsule

A new cytokine involved in dermatitis

Activation of the immune system can guard against pathogens that cause disease, but when they go awry the same mechanisms can lead to autoimmune diseases or allergies. For instance, atopic dermatitis, an inflammatory skin disease that is frequently associated with hay fever or asthma, appears to involve abnormal cytokine production by T helper type 2 (TH2) cells. Dillon et al. identified a TH2 cell-derived cytokine, interleukin 31 (IL-31), and its receptor IL-31 receptor A (IL-31RA) on lymphoid and epithelial cells. Transgenic mice that over-expressed IL-31 in lymphocytes developed pruritis, skin lesions and hair loss, as did mice treated directly with IL-31. In a model of airway hyper-responsiveness, exposure of presensitized mice to antigen stimulated IL-31RA expression in the lung. Thus, IL-31 appears to be involved in mediating dermatitis and hyper-responsive airway disease.

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