Osteopathic Family Physician (2012) 4, 72-80

ELSEVIER

acofp Osteopathic Family Physician

"Frozen Shoulder"—A Difficult Clinical Problem

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KEYWORDS:
Frozen shoulder;
Adhesive capsulitis;
Shoulder;
Osteopathic manual medicine;
Manipulation under anesthesia

Frozen shoulder is a clinical condition characterized by a relatively sudden onset, variable degrees of pain, restricted range of motion, and normal radiographic imaging techniques. Although the disease occurs in a small segment of the population, its management presents the clinician with an opportunity to use all of his or her skills to alleviate the pain and restore function of the shoulder. This review article discusses contemporary ideas on etiology, diagnosis, and noninvasive treatment including manual medicine techniques, surgical options, and prognosis.

A frequent visitor to the primary care office is the patient who spontaneously develops shoulder pain and stiffness. Because nothing can be related to the pain, the patient's symptoms are a source of anxiety and fear. The rapidity of the onset of their condition usually prompts the visit, and the practitioner is viewed as the source for relief. Except for shoulder stiffness and pain, the medical history and physical examination are often unremarkable, and the subsequent work-up may leave the patient and their physician puzzled. "What is causing this problem?" "Why am I having pain?" "Why can't I move my shoulder?" This condition, adhesive capsulitis, or "frozen shoulder," is not an uncommon condition and presents many challenges to the health care team. This article will review the presentation, proposed etiologies, differential diagnoses, treatment options, and prognosis for this frequently seen and often disabling condition. In preparing this review, every effort was made to include recent (within the past 10 years) sources of information found in Medline, Ovid, and Quertle using "frozen shoulder" and "adhesive capsulitis" as search terms.

Presentation

"Frozen shoulder" was first described in the French literature in 1872 by Duplay as "Periarthritis of the Shoulder."[1] The interchangeable, contemporary terms "frozen shoulder" and "adhesive capsulitis" were coined by Codman in 1934 and Neviaser in 1945, respectively.[2,3] Drs. Neviaser[4] aptly described the diagnosis of "frozen shoulder" as a "waste can" diagnosis. They felt that the diagnosis was overused and misunderstood. While mentioning both frozen shoulder and the stiff and painful shoulder, they stated, "The diagnosis must be established because the treatment of each is different," and *"Every patient with a painful shoulder and apparent limitation of motion does not have adhesive capsulitis"* (their italics).[4]

This clinical condition characterized by a relatively sudden onset, variable degrees of pain, restricted range of motion (especially external rotation), and normal radiographs is seen in an undetermined percentage of the general population. An estimated frequency of occurrence is difficult to determine from a careful review of the literature.

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1877-573X/$ -see front matter © 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.osfp.2011.12.001

Estimates range between 2% and 5% of the population over the age of 40.[3] It is a disease of middle age, with females and those with diabetes having a predilection for development of this problem.[5-7] Clinical association has been made with low back pain, cervical radiculitis, clinical depression, anxiety disorder, Parkinson's disease,[8] mastectomy, hyperthyroidism,[9] hypertension, and migraine headaches,[10] as well as histological similarities to Duypuytren's disease.[11] The painful and stiff shoulder caused by osteoarthritis, chronic subacromial bursitis, or rotator cuff tendonopathy/tear should not be confused with the frozen shoulder. Trauma may or may not be associated with its onset.

The clinical course usually consists of progressive stages of uncertain duration. These stages represent a continuum of disease and are not discrete. Neviaser described four stages: Stage 1, the preadhesive stage, is characterized by deltoid insertion pain and night pain; stiffness along with persistent night pain develops in stage 2, the acute adhesive synovitis stage; profound stiffness with pain only at the end of range of motion is stage 3, the stage of maturation; and during stage 4, the chronic stage, the pain is minimal and range of motion improves.[4] Today it is commonly accepted that the process may progress through three phases. Reeves[12] and Dias et al.[13] described these as pain, stiffness, and recovery phases. The first phase, painful freezing, is well named for the most intense pain, often noticed at night, as well as progressive stiffness. After 2 to 9 months, this phase gives way to the stiffness or adhesive phase, in which stiffness rather than pain is the characteristic finding. This phase may last for another 1 to 3 months. Finally, the shoulder begins to recover with a spontaneous but often incomplete improvement in the range of motion.[12,13] Depending on the stage at presentation, recovery may take from months to 3.5 years. With effective treatment, progression of the disease can be stopped during any of these three stages.

Etiology

The etiology of frozen shoulder remains unclear. Duplay believed that an inflammatory process of the subacromial burse was the causative agent.[1] About 50 years later, Meyer felt that various degenerative conditions of the long head of the biceps brachii was responsible.[14] Pasteur[15] in 1932 supported this notion by finding "tenobursite" or tenosynovitis of the long head of the tendon, followed by Lippman[16] in 1943, who advocated open tendon surgery to address an "adhesive tenosynovitis." In 1934, Codman was of the opinion that the rotator cuff tendons were responsible for frozen shoulder.[2] In 1952, DePalma postulated that the problem was caused by a diffuse inflammatory process implicating all soft tissue structures of the shoulder joint but especially the long head of the biceps tendon.[17] In 1977, Turek hypothesized that inflammation, infection, or trauma could cause thickening of the cuff and the intraarticular biceps tendon, which would lead to generalized capsular thickening and subsequent stiffness.[18] As a result of limited range of motion, he went on to say that periscapular muscle atrophy would develop and exacerbate the condition. In 1990, Neer et al. studied the coracohumeral ligament and found contracture of this ligament to be associated with restriction in external rotation. By dividing this ligament, external rotation motion that was severely restricted in frozen shoulder was improved.[19] Bunker and Anthony, in 1995, found no inflammation but described histological and immunocytochemical findings of the coracohumeral ligament and rotator interval, which were very similar to those seen in Dupuytren's disease of the hand, a condition characterized by thick palmar contractures that inhibit finger range of motion.[20] In contradistinction, analyses of biopsy specimens taken from frozen shoulder surgical patients were tested by Hand et al. in 2007 and showed evidence of chronic inflammatory responses. These specimens also showed high vascularity and nerve tissue that may help to explain the stiffness and pain associated with frozen shoulder.[21] Most recently, in 2010, Kabbabe et al. considered that frozen shoulder was caused by a controlled inflammatory response evolving into a fibrotic condition mediated by cytokine messengers.[22] Medical therapy directed at lowering these messengers may prove valuable in altering the course of this disease.

Diagnosis

A thorough history and physical examination to eliminate other causes of pain and restricted motion is the foundation for correctly diagnosing frozen shoulder. Usually the patient is middle-aged and develops shoulder pain suddenly, without trauma or antecedent illness. In the initial phase of the process, their pain is significant, anterior in location, and often situated directly over the biceps groove. The pain is often worse at night. Range of motion is limited in abduction and external rotation, although all ranges of motion may be affected. Passive in addition to active motion is noticeably altered. Although it is not pathognomonic for frozen shoulder, limited external rotation is often found in this condition. At the end of the range of motion, the examiner senses a mechanical blockage to further motion rather than voluntary guarding as a result of pain. There may be localized tenderness directly over the coracoid process that, according to recent original research, may be pathognomonic for frozen shoulder.[23] Typically, the neurovascular examination is unremarkable. Laboratory values prove to be of little help, although C-reactive protein and erythrocyte sedimentation rate may be elevated. Plain radiographs of the shoulder appear normal. Magnetic resonance imaging or arthrography are not usually done unless there is a suspicion of rotator cuff pathology with a previous history of shoulder pain or a lifestyle/occupation that may affect the cuff, e.g., in an overhead worker. However, a recent radiological study suggested a positive correlation with magnetic

resonance imaging findings and clinical stages of frozen shoulder that may serve as a useful adjunct to diagnosis and facilitate treatment for the primary care physician.[24]

Treatment

A recent extensive review of different treatment modalities including 758 papers showed "insufficient evidence to draw firm conclusions about the effectiveness of treatments commonly used to manage a frozen shoulder."[25] With that in mind, what is the clinician to do?

Treatment options are suggested on the basis of the stage at presentation, but even so, there is disagreement about what constitutes the best protocol for dealing with this disabling problem. The practitioner must be aware of not only the physical limitations caused by the disease but also the psychological ramifications of the lifestyle changes. The literature includes numerous treatment options including observation and reassurance, rest, analgesics, nonsteroidal antiinflammatory medication, oral steroids,[26] home exercise programs, structured physical therapy, osteopathic manipulative techniques, corticosteroid injections, myofascial trigger point injections,[27] capsular distension with saline, steroid injections with distension,[28] electromagnetic therapy, manipulation under anesthesia,[29-32] open and arthroscopic capsular release, acupuncture,[33] and Ayurvedic approaches.[34]

The stage of the disease dictates the optimum treatment. For example, if the patient presents with a one-week history of severe pain caused by adhesive capsulitis, then rest and analgesics are appropriate. On the other hand, if the symptoms have existed for three months and stiffness is the main problem, then physiotherapy may be effective. Clinical discretion is most important. Supportive care and overall treatment guidance is the responsibility of the primary care practitioner, who will address associated dysfunctions, both physically and mentally. A team approach is warranted, bringing in the orthopedist as a consultant along with the physiotherapist, who plays an integral, active role in treatment.

Narcotic analgesics should be prescribed judiciously because of the protracted course of the disease and the potential for dependence. Nonsteroidal antiinflammatories as well as oral steroids have the potential for serious side effects but are usually well-tolerated. Oral steroids such as prednisolone may be prescribed during the painful or early phases of the disease. A review of studies on oral steroids for adhesive capsulitis showed a possible benefit for short-term use; however, each study included the use of physiotherapy and had population sizes of fewer than 50 subjects.[26]

Lee et al studied intraarticular steroids injected with and without the use of ultrasound and suggested an increased efficacy using ultrasound. A single injection of 20 mg triamcinolone mixed with 1.5 mL xylocaine and 4 mL normal saline was used. The patients then received five weekly injections of sodium hyaluronate. All of the patients in this study were instructed in a home program of physiotherapy. By the second week postinjection, the improvement in pain intensity, range of motion, and shoulder function was significantly greater in the ultrasound-guided injection group, although these differences did not extend beyond the third week.[35] A systematic review of three high-quality trials using multiple corticosteroid injections demonstrated no benefit to using more than six injections during the course of treatment.[36] A similar paper suggested the effectiveness of corticosteroid injections in the short term compared with physiotherapy, but this difference was small in the long term.[37]

Twenty-six studies were included in a recent review of physiotherapy interventions for shoulder pain. Green et al. stated ". . . substantial clinical heterogeneity with respect to the intervention tested existed . . . making it difficult . . . to reach an overall conclusion about the effect of physiotherapy interventions for shoulder disorders."[38] It seems reasonable, however, to recommend physiotherapy in the vast majority of patients with frozen shoulder.

Osteopathic approach to treatment

A mobile shoulder girdle consists of a series of joints, including glenohumeral, sternoclavicular, acromioclavicular, and the first rib, as well as scapulothoracic and suprahumeral articulations, all of which contribute to the maximum motion of the upper extremity.[39] Therefore, in an osteopathic approach to the patient with adhesive capsulitis, we must consider the relationship of both the structure and function of the joint and attempt to restore the normal structure/functional relationship that occur in that region. Treatment must not be limited solely to the glenohumeral joint. By addressing the shoulder region or girdle, we seek to reestablish articular and soft tissue structure and the functional relationships to restore flow in the arterial, venous, and lymphatic systems to positively affect the inflammatory and fibrotic cascade that has been described previously.[3,40]

Current evidence suggests that inclusion of manipulative interventions, both thrusting and nonthrusting type of techniques, indeed may be helpful in the treatment of individuals with shoulder pain,[41-44] and may speed recovery, which is sustained at least one year out.[45] A number of different types of treatment approaches may be used including soft tissue (myofascial, muscle energy, strain-counterstrain, lymphatic); articulatory (high velocity–low amplitude, low velocity–high amplitude); and trigger point treatment, directed at the shoulder girdle (especially subscapularis muscle), thoracic and cervical spines, and ribs, avoiding the significant "pain zone" because this may slow progress. Indirect techniques may be especially effective in the initial treatments.[46]

Figure 1 Jones technique.

Figure 3 Green's technique. Second position.

An indirect technique (taking the tissues the way they "like to go" or away from the restricted barrier) aimed at the glenohumeral joint is described by Jones et al. (Fig. 1).[47] It has been described that treatment of this type corrects the aberrant alpha-gamma loop misinformation as well as other mechano-receptor misinformation and restores normal tone to the tissues. In this treatment, the patient's arm is held in the adducted/internal rotation position, and a compression force is applied on the elbow along the shaft of the humerus, which lifts it. This will externally rotate the scapula and produce more adduction of the humerus in the glenoid. The patient can be taught how to do this at home, by leaning the elbow on a lower table and then lowering the trunk, forcing the shoulder into the same position.

Bergman describes Green's technique, or a glenoidal labrum technique, to enhance movement of the humeral head in the glenoid and the labrum (Figs. 2-4).[45] The first step is to have the patient lie in the prone position with the painful arm off the edge of the table (Fig. 2). The physician next takes the arm and applies an anterior and caudal traction with internal and external rotation 2 to 3 times (Fig. 3). Finally, the physician takes the humeral neck with thumbs on the greater tuberosity, and the remaining fingers surrounding the proximal shaft. Movement is applied through the humeral head in an anterior-posterior, cephalad-caudad, medial and lateral traction–distraction, figure of eight, and circular directions to improve overall motion (Fig. 4).

The Spencer technique (or "Seven Stages of Spencer") was developed by Charles H. Spencer, DO. In his initial description in 1916, he noted that he had particular success in applying a series of manipulative treatments to baseball players and others who had suffered trauma to the shoulder, and that were most effective in those with decreased shoulder motion and pain.[48] The Spencer technique has undergone several modifications since then, most notably by the addition of an isotonic muscle contraction to many of the steps. This treatment combines Spencer's positioning, sequence, and slow stretching with a patient-active muscle energy technique to enhance both the stretching of the soft tissues as well as mobilization of fluids, adding to the effectiveness of the treatment.[49] The various steps (Figs. 5-12) will evaluate the separate ranges of motion, both in

Figure 2 Green's technique. Initial position.

Figure 4 Pump.

Figure 5 Spencer technique—Extension. The patient's arm is carried into extension to the restrictive barrier to stretch the tissues and held for a few seconds. Then the patient can be instructed to flex against resistance for 3 to 5 seconds, followed by a second of relaxation. This flexion against resistance is repeated 3 to 5 times.

Figure 7 Circumduction with compression. The arm is abducted to 90°(as pain allows), with the elbow flexed. With slight compression through the humerus, a circumduction motion is applied, starting with small circles and progressing to larger ones in a clockwise motion for about 15 to 30 seconds. The same is repeated in a counterclockwise motion. Note: No muscle energy at this step.

the pain-free and total ranges. It is typically described in the following sequence to address the glenohumeral motion in a pattern that treats the most pain-free (or best preserved motion) first, followed by the more restricted motion. The patient is lies in the lateral recumbent position with the affected shoulder up. The physician's cephalad hand is used to stabilize the shoulder girdle, including the scapula and clavicle, while the caudal hand is providing the force on the patient's affected arm.

The myofascial etiology to frozen shoulder as described by Travell and Simons would suggest the initial insult is to the subscapularis muscle, thus initiating trigger points, which are typically in the lateral aspect of the muscle in the posterior axillary fossa along the lateral border of the scapula. Subscapularis trigger points are activated by several potential mechanisms. The most relevant ones to the discussion of adhesive capsulitis are the following: (1) Unusual repetitive exertion requiring forceful internal rotation when the patient is not conditioned (e.g., overhead stroke of the crawl during swimming, or throwing motion overhead); (2) repeated forceful overhead lifting while exerting a strong adduction force (e.g., swinging a small child back and forth from between an adult's legs, up overhead, and down again); (3) sudden stress overload (e.g., reaching back to break a fall); (4) prolonged immobilization of the shoulder joint in the adducted and internally rotated position (e.g.,

Figure 6 Flexion. The arm is carried into flexion to the restrictive barrier to stretch the tissues and held for a few seconds. The patient is instructed to extend against resistance for 3 to 5 seconds, followed by a second of relation. This extension against resistance is repeated 3 to 5 times.

Figure 8 Circumduction with distraction. The arm is positioned as in Figure 7 but the elbow is extended, and a distracting force is applied through the arm. Circumduction maneuvers are performed as in Figure 7. Note: No muscle energy at this step.

Figure 9 Abduction. The arm is moved into abduction to the point of stiffness and held for a few seconds to stretch the tissues. The patient is then asked to adduct against resistance for 3 to 5 seconds, then rest for a second. This adduction against resistance is repeated 3 to 5 times.

Figure 11 Adduction with internal rotation. To engage the adduction barrier, the elbow is gently pushed toward the table to the point of stiffness to stretch the tissues. The patient is asked to push upward against resistance for 3 to 5 seconds, then rest for a second. This resistance against upward pushing is repeated 3 to 5 times.

arm in a sling); and (5) overload stress after shoulder dislocation or fracture.[50] These points, in turn, may cause associated trigger points in the surrounding muscles. The activation of subscapular trigger points restricts abduction of the shoulder, which sensitizes the pectoralis, latissimus dorsi, and triceps. Restriction of external rotation leads to sensitization of the anterior deltoid and teres major. The posterior deltoid may also be involved because of referred pain from the subscapularis. Eventually, most or all of the shoulder girdle muscles may be involved, leading to a frozen shoulder. Trigger points may be treated with a spray and stretch or local injection with anesthetic. The spray and stretch procedure involves vapocoolant spray (either Fluori-Methane or ethyl chloride) swept over the affected muscle at about a 30° angle, parallel to the muscle fibers, followed by an immediate passive stretch. If injection is to be pursued, Travell and Simon recommend procaine because it has a short duration of action (<30 minutes), has minimal systemic toxicity, and is the least myotoxic of the local anesthetics. Lidocaine has twice the potency and twice the duration of activity compared with procaine and can also be used if procaine is not available. Often the response is immediate resolution of spot tenderness and referred pain

Figure 10 Adduction with internal rotation. To engage the internal rotation barrier, the arm is adducted and internally rotated by placing the dorsum of the hand on the small of the back. The patient's arm is gently pulled forward to the point of stiffness to stretch the tissues. The patient is asked to pull the elbow back against resistance for 3 to 5 seconds, then rest for a second. This resistance against the elbow motion is repeated 3 to 5 times.

Figure 12 Glenohumeral pump. Finally, the arm is abducted and placed on the physician's shoulder. The physician's fingers are interlaced and placed just distal to the glenohumeral joint. A gentle, rhythmic scooping or translation motion is applied to the humeral head in an anterior-inferior motion to stretch the tissues. While maintaining slight traction, the patient is asked to press down against the physician's shoulder for 3 to 5 seconds, relax for a second, and repeat 3 to 5 times.

with release of the muscle's restricted range of motion. As trigger point activity lessens, treatment may be advanced. Initially, passive stretching followed by stabilization exercises, and finally active strengthening exercises, should be incorporated.[50]

Other recommendations for the patient to do at home include modifications of sleep position and posture, and compliance with their home exercise program. While sleeping, the patient should avoid prolonged full adduction and internal rotation. Modification while sleeping on the painful side or back can include using a small pillow between the elbow and the side to maintain some arm abduction. While sleeping on the pain-free side, the pillow can be moved in front of the body to better support the painful arm and prevent the arm from folding across the chest. When standing for longer periods of time, the patient can hook the thumb of the painful side into a belt or on the hip to prevent the arm from resting at one's side.[50]

Surgical option

Surgical management of the frozen shoulder includes manipulation under anesthesia with or without either open or arthroscopic release of the contracted tissues. These interventions are usually considered after a six-month course of conservative care has failed to produce meaningful improvement. Manipulation under anesthesia (MUA) is included as a "stand-alone" procedure or combined with surgical release. McLaughlin examined two dozen shoulders between 1936 and 1938 that were exposed by operation before manipulation under anesthesia to locate the mechanism for the "stiffness" and to document the results of the manipulation. He found no adhesions, but "In almost every instance the biceps tendon ruptured, usually just proximal to the bicipital groove." He stated also that in every case either the subscapularis tendon was ruptured or the glenohumeral ligaments were avulsed from the scapula.[51] Subsequent observations by others noted ruptures of different areas of the shoulder joint.[11]

Andersen et al. examined 24 frozen shoulders with arthroscopy followed immediately with manipulation under anesthesia and repeat arthroscopy. They found that in 79% of the cases the capsule was ruptured adjacent to the anterior inferior glenoid rim by the manipulative procedure.[52] Hamdan and Al-Essa manipulated 98 shoulders. His patients were divided into three groups that received manipulation alone or with steroid or normal saline distension. The group receiving manipulation and normal saline distension was described as "effective."[53] In our opinion, it is difficult to interpret these results because there was no group who received only normal saline distension as a control. Klinger et al. found arthroscopic capsular release to be an effective way of shortening the course of the disease. All of their 36 patients tried six months of physiotherapy before the procedure. At a mean of 18 months "all patients noted substantial relief of pain." Improvement in range of motion occurred although it was not determined whether the range had returned to normal.[54] Kivimaki et al. compared results of MUA and home exercises with an equal cohort receiving only home exercises. They found that MUA did not add effectiveness to the home exercise program.[55] Complications, including iatrogenic fracture, may worsen the patient's condition. Chambler and Carr reviewed 12 operative series that included combinations of MUA and arthroscopy with or without steroids and found "limited evidence to show that it (surgery) will truly change the natural course of this disabling condition."[56]

Prognosis

Despite a conscientiously and skillfully applied treatment, residual problems may persist. Objective outcomes, e.g., an improved range of motion, may not always correlate with a patient's subjective complaints. In 1975, Reeves reported on 49 patients who were followed to "their greatest recovery" over a 5- to 10-year period. Half of these patients had slight restriction of movement, but only 3 patients perceived the restriction as a handicap.[57] Several years later, a long-term prospective study was completed on 40 patients by Binder et al. The exact therapeutic regimens were difficult to determine; however, after a mean of 44 months, objective restriction of range of motion was severe in five and mild in 11 patients. Although patients' shoulder range of motion was restricted, the authors reported little functional impairment.[58] Shaffer et al. performed a long-term follow-up study and found that 50% of 62 patients followed for an average of seven years had continued symptoms including stiffness, mild pain, or both.[59] Most recently, Hand et al. studied 269 shoulders in 223 patients over an average of 4.4 years to find that 59% of the patients had normal or near normal shoulders and 41% had ongoing symptoms.[60] From a functional standpoint, these studies indicate a positive outcome in the majority of cases, although patients should be warned that full restoration of movement may not occur.

Conclusion

Frozen shoulder is a clinical entity of uncertain etiology characterized by pain and stiffness presenting in three progressive phases. Numerous treatments have been suggested, with physiotherapy as a cornerstone. Various manipulative techniques have been used to treat associated soft tissue dysfunction and shorten the duration of symptoms. A multidisciplinary approach to this problem using medication, manipulative medicine, psychological counseling, support, physical therapy—and in longstanding severe, refractory cases, orthopedic surgery—will generally improve the majority of patients' function and range of motion, although a small number of patients may experience residual stiffness.

Acknowledgements

We thank David Patch for the photography and Stephanie Simms as the model. We also thank Elaine Powers for reference procurement and Carole Porter for editing.

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March/April 2012 CME Quiz Answers:

1.c, 2.a, 3.b, 4.c, 5.b, 6.b, 7.c, 8.d, 9.b, 10.b