

Effective Reduction of Adhesive Capsulitis Pain With a Suprascapular Nerve Block Given in a Primary Care Clinic

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ABSTRACT

Adhesive capsulitis or “frozen shoulder” is a shoulder joint condition defined by pain with decreased active and passive range of motion. The etiology is poorly understood, but the pathological process involves a local inflammatory state, followed by fibrosis or contracture. The condition is often a self-limiting process that resolves within 18-24 months with benign neglect or conservative treatment. Patients failing to improve may require more invasive surgical procedures. The goal of conservative treatment is to reduce pain and improve function; usually this consists of analgesics, non-steroidal anti-inflammatory drugs and physical therapy. Invasive procedures used in non-responsive cases carry significant risks, including humeral fracture, infection, and general anesthesia complications. Suprascapular nerve block is a new treatment where the nerve innervating the shoulder joint is blocked with local anesthetic. Currently, this procedure is not the standard secondary treatment for adhesive capsulitis, but in the future may prove to be an effective way to avoid the risks of more invasive procedures in non-responsive patients. In this case study we describe the clinical presentation, diagnosis, and treatment of adhesive capsulitis in a 52 year-old female who received suprascapular nerve block treatment after failure of conservative primary treatment.

KEYWORDS: *Suprascapular nerve block, Adhesive capsulitis*

ADHESIVE CAPSULITIS

Adhesive capsulitis (AC) is a shoulder condition defined by an insidious onset of pain and a gradual loss of both passive and active range of motion (ROM) in the affected shoulder.^{1,2} Some cases of AC may occur subsequent to injury or immobilization, but most are idiopathic in origin.² AC is characterized by inflammation of the synovial lining and capsule with subsequent generalized contracture of the glenohumeral joint.¹ The natural history of AC is usually self-limiting and often improves with benign neglect in 18-24 months.³ The condition often appears in the fifth and sixth decades of life, but is rare in patients under 40 years of age.⁴ The prevalence of AC is thought to be 2% in the general population, although this is increased with other comorbid conditions such as diabetes, hyperthyroidism, hypertriglyceridemia, and Dupuytren’s contracture.^{4,5}

HISTORY AND CLINICAL FINDINGS

Patients will most commonly present with gradual onset of aching pain over the insertion of the deltoid or acromion with sleep disruption.^{5,6} Pain occurs with shoulder movement and results in loss of ROM, especially abduction and external rotation.^{4,6} Physical examination reveals limited active and passive ROM of

the affected shoulder. Laboratory investigations are unremarkable with the exception of elevated C-reactive protein in early stages.⁵ Plain radiographs are normal with AC, but are often ordered to exclude other pathologies.⁵

DIAGNOSTIC CRITERIA

As AC shares many clinical findings with other shoulder conditions, the diagnosis is one of exclusion.⁵ Differential diagnoses include impingement, partial and full-thickness rotator cuff tears (RCT), arthritis, and locked posterior dislocation.⁵ A list of clinical features of AC defined by Codman criteria include: insidious onset, pain at the insertion of the deltoid, poor sleep due to pain, incomplete elevation and external rotation, reduced active and passive ROM, atrophy of the spinati, and negative radiograph.^{5,6}

Two of these features are crucial for distinguishing AC from other shoulder pathologies. First, reduced external rotation is found with AC, arthritis, locked posterior dislocation, fullthickness RCT, but AC does not show any radiographic changes.⁵ Second, limited passive shoulder ROM is only caused by joint surface abnormality as found in arthritis or contracture of the glenohumeral ligaments as found in AC.⁵ In practice, AC diagnosis is made if physical exam and history include a positive Codman’s criteria with confirmation by normal radiograph. Ultrasound may also be used to rule out RCT if it is suspected.

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PATHOPHYSIOLOGY

The pathological process of AC remains unclear, but it is hypothesized that it begins with synovial inflammation followed by capsular fibrosis and contracture.^{5,7} The current pathophysiological understanding is described in the context of AC stages. Four stages of AC are described as 1) Inflammatory, 2) Freezing, 3) Frozen, and 4) Thawing.^{5,7}

In stage 1, passive ROM is increased with anesthesia indicating that passive ROM is pain limited. Histological specimens demonstrate inflammatory infiltrates and hypervascular synovitis with a normal underlying capsule.⁷ Stage 2 differs in that passive ROM is similar with or without anesthesia. Histological specimens indicate hypertrophic, hypervascular synovitis with subsynovial and capsular scarring.⁷ In stage 3, pathological specimens show reduced synovitis and dense scar formation in the underlying capsule.⁷ Stage 4 represents resolution and no pathological specimens have been described.⁷

CASE PRESENTATION

A 52 year-old female patient first presented with achy left shoulder joint with pain that radiated down her arm with a severity of 7/10. Range of motion (ROM) in the affected shoulder was reduced such that she could not dress herself. She also had disrupted sleep due to pain. This patient had a history of hypertension and type II diabetes mellitus. Physical examination showed no signs of erythema or edema in the shoulder or elbow and no scapular winging. She was referred for physical therapy and shoulder ultrasound to rule out RCT.

Within the next five months she noted some reduction in pain and increased ROM. She found mild relief with cryotherapy and ibuprofen. No rotator cuff tears were identified on ultrasound.

“...in the future [suprascapular nerve block] may prove to be an effective way to avoid the risks of more invasive procedures in non-responsive patients.”

reach to S1 on the left and T7 on the right. A clinical diagnosis of adhesive capsulitis (AC) was made and the patient was to remain in physical therapy, and return if pain worsened.

Pain severity at rest was 3/10, and 9/10 with shoulder movement. Active shoulder ROM in the left (AL) and right (AR) shoulder and passive ROM in the left (PL) and right (PR) were as follows: Forward Flexion AL 110°, PL 120°, AR 170°, PR 170°; Abduction AL 90°, PL 100°, AR 160°, PR170°; and External Rotation AL 5-10°, PL 10°, AR 70°, PR 70°. With internal rotation the patient could

SOAP Note

Subjective

- 52 year-old female patient presents with a 5 month history of achy left shoulder that radiates down arm
- pain and decreased range of motion limit ability to dress herself
- pain rated as 3/10 at rest, 9/10 with shoulder movement
- patient found minimal pain relief with cryotherapy and ibuprofen
- physical therapy mildly successful at reducing pain
- patient has hypertension and diabetes mellitus

Objective

- no signs of erythema or edema in affected shoulder
- no scapular winging
- patient has reduced active and passive range of motion in the affected shoulder, especially external rotation
- no rotator cuff tears identified on ultrasound

Assessment

- history, clinical findings, and ultrasound are consistent with adhesive capsulitis

Plan

- continue physical therapy and NSAIDs
- if pain worsens, patient is to return to discuss further treatment options including:
 - manipulation under anesthesia
 - open surgery
 - arthroscopic capsular release
 - suprascapular nerve block

TREATMENT

Initial treatment for AC is non-operative, consisting of benign neglect, non-steroidal anti-inflammatory drugs (NSAIDs), oral corticosteroids, and intra-articular corticosteroid injections.⁹ The main benefit from these treatments appears to be pain reduction with little improvement in ROM.¹⁰ Physical therapy provides a non-invasive approach to stretching the contracture.⁵ Within non-operative treatments, success varies with many patients opting for multiple treatment modes. Regardless of mode, 89.5% of AC cases will respond to non-operative treatment.⁹

Invasive treatment may be required for AC cases that are non-responsive to conservative treatment after 6 months.⁷ The most common invasive treatment is manipulation under anesthesia in which the shoulder is manipulated to break the contracture.⁵ Success rates are variable with some risk of humeral fracture or dislocation.^{5,10} Open surgical capsular release is a last resort due to complications, but is effective in severe cases.⁴ The more common surgical technique is arthroscopic capsular release which improves ROM with reduced operative morbidity relative to open release.^{11,12}

CASE REPORT

SUPRASCAPULAR NERVE BLOCK (SSNB)

SSNB is not recognized as standard treatment for AC, but has shown greater relief from symptoms compared to placebo and a faster and more complete reduction in symptoms when compared to intra-articular corticosteroid injections.^{13,14} The suprascapular nerve originates from the superior trunk of the brachial plexus and passes posterolaterally through the suprascapular notch and suprascapular fossa to innervate the glenohumeral joint (Figure 1). Blockade of this nerve has been described by Wertheim and more recently by Dangoisse.^{15,16}

Using the Dangoisse technique a 21G x 1.5" needle is inserted 2cm superior to the bisection of the scapular spine in the plane of the blade of the scapula and directed inferiorly toward the suprascapular fossa floor (Figure 2). The needle is advanced in this plane until bony contact is made with the fossa floor. The needle must be aspirated to eliminate the risk of intravascular needle placement. Once in place, 10cc of local anesthetic plus corticosteroid is injected into the floor of the fossa, bathing the suprascapular nerve. The benefit of corticosteroid in addition to local anesthetic alone has been debated as the injection point is not a site of inflammation.¹⁷ However, there is evidence that a combined local anesthetic and corticosteroid may increase the duration of nerve blockade.¹⁸ Aside from vasovagal episodes, the risk of SSNB appears to be low, with no significant complications reported in over 2000 procedures.¹³

RETURN TO CASE

The patient returned two months later, seven months after initial onset of symptoms, with increased pain. Her pain severity was 7-8/10 at rest. Active ROM in the shoulders was: Flexion L 130°, R 170°; Abduction L 70°, R 170°; External Rotation L 10°, R 50°; and internal rotation reach was L S1 and R T7. After consultation, the patient was interested in SSNB treatment.

A SSNB of 9 cc of 0.5% bupivacain and 1 cc of 0.4% dexamethasone using the Dangoisse technique was provided with

the patient's consent. She noted some immediate pain relief within 5 minutes of the injection and her pain scores were followed for six weeks after the injection.

Pain was evaluated with a 100mm visual analogue scale (VAS) and disability with a validated shoulder dysfunction questionnaire [Shoulder Pain and Disability Index (SPADI)]¹⁹ before treatment and 1,3 and 6 weeks post-injection. VAS and SPADI scores through the 6-week duration are presented in Table 1.

Symptoms were reduced throughout this period with the patient also requiring reduced use of analgesic medication. Physical examination was completed at six weeks post injection with active ROM showing improvement: Flexion L 140°, R 170°; Abduction L 150°, R 180°; External Rotation L 30°, R 70°; and internal rotation reach was L L3 and R T5. Passive ROM was not recorded at this time. Pain severity was significantly improved six weeks post injection with a reported value of 1-2/10.

CONCLUSION

SSNB was an effective treatment for this patient, providing a simple, safe and non-invasive alternative to manipulation under anesthesia, arthroscopy, or open surgery. The patient's AC symptoms were greatly reduced with SSNB after seven months of unsuccessful physical therapy and ibuprofen. Her symptoms continued to diminish up to three weeks post-injection and remained reduced at six weeks follow-up. She did not return for further treatment of recurrent symptoms after such time.

SSNB carries less risk of complications than the aforementioned invasive treatments and can be administered in a clinic setting by a primary care physician rather than in an operating room by a surgeon so there is a reduction in patient discomfort and possibly some reduction in treatment cost. However, further research is required to evaluate the effectiveness of this procedure and the necessity for corticosteroid co-injection with SSNB as some debate exists over the treatment efficacy, ability for symptom reduction and mechanism of action. 

Table 1. Visual Analogue Scale (VAS) and Shoulder Pain and Disability Index (SPADI) Summary

Measure	T1	T2	T3	T4
VAS (mm)	74	58	17	15
SPADI (%)	84	55	33	32

This table represents the patient's reported visual analogue scale (VAS) pain measures and shoulder pain and disability index (SPADI) measures throughout 6 weeks of treatment monitoring: pre-injection (T1), 1 week post-injection (T2), 3 weeks post-injection (T3), and 6 weeks post-injection (T4). *Note:* VAS score 0 represents no shoulder pain and 100 represents most severe shoulder pain. SPADI score represents combined pain and disability: score 0 represents no shoulder pain or disability and 100 represents maximum shoulder pain and disability.

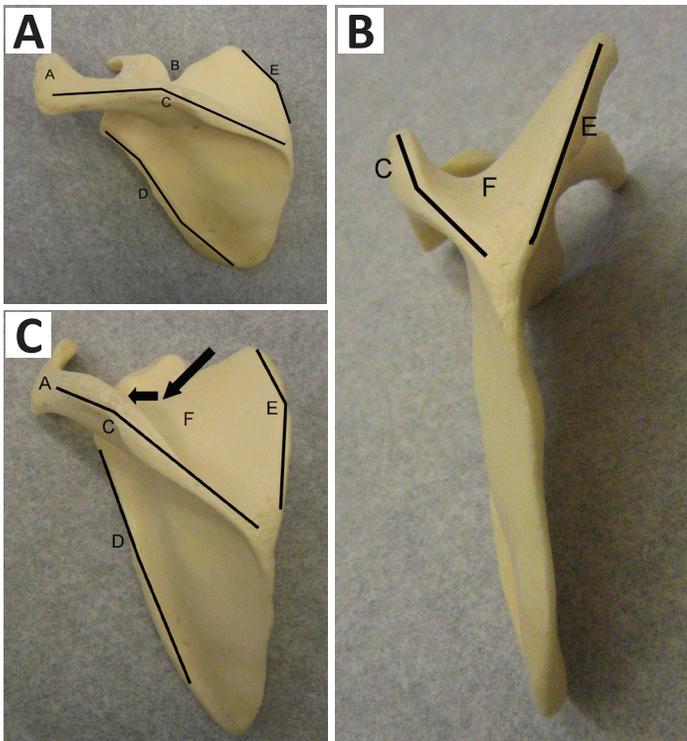


Figure 1. Left scapula viewed posteriorly (Panel A), medially (Panel B), and posteromedially (Panel C). Landmarks are labeled as follows: Acromion (A), Suprascapular notch (B), Spine of scapula (C), lateral border of scapula (D), medial angle of scapula (E), and suprascapular fossa (F). The posterolateral passage of the suprascapular nerve from the brachial plexus, through the suprascapular notch, toward the glenohumeral joint is indicated by two dark arrows (Panel C).

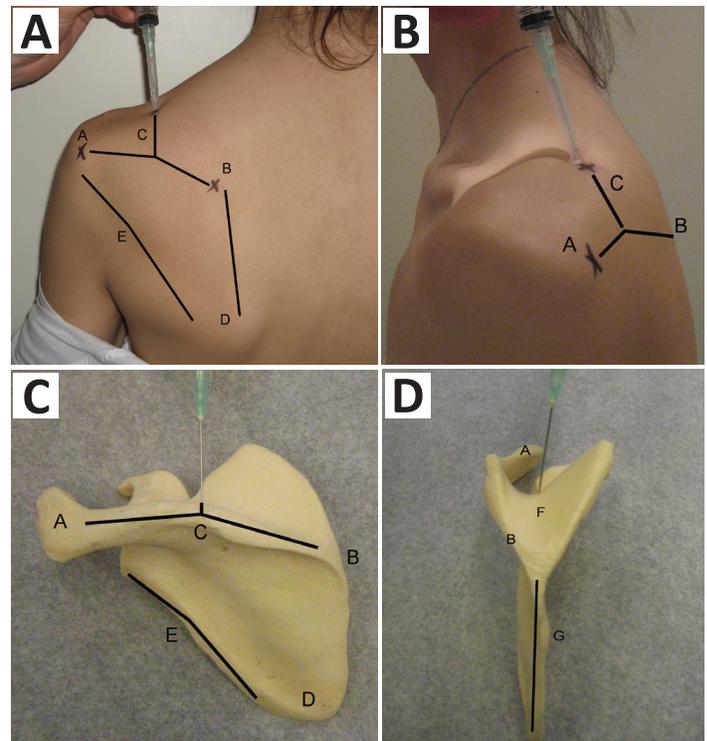


Figure 2. Posterior (Panels A and B) and lateral (Panels C and D) images of SSNB using Dangoisse technique. Landmarks are indicated as follows: Acromion and lateral end of scapular spine (A), medial end of scapular spine (B), midpoint of scapular spine (C), inferior angle of scapula (D), lateral border of the scapula (E), suprascapular fossa (F), and blade of the scapula (G). Note the needle is aligned 2cm superior to the midpoint of the scapular spine parallel to the blade of the scapula.

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