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Outpatient PT Management of Patient with Left Shoulder Calcific Tendinitis

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Outpatient PT Management of Patient with Left Shoulder Calcific Tendinitis

by

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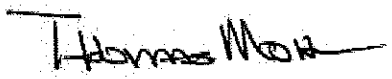
This Scholarly Project, submitted by Austin Stueve in partial fulfillment of the requirements for the Degree of Doctor of Physical Therapy from the University of North Dakota, has been read by the Advisor and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.



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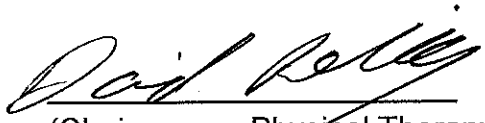

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ABSTRACT

Background and Purpose: This case report describes a 5 week outpatient trial of conservative management of calcific tendinitis of the left shoulder. The patient presented with limited, left shoulder ROM, strength, glenohumeral joint restriction, pain with shoulder plane motions, and shoulder point tenderness. The purpose of this case report is to describe the interventions used for this patient and the outcomes of following the interventions used. **Description.** The treatment of this patient included ROM exercises, rotator cuff strengthening exercises, manual therapy, iontophoresis, and ultrasound **Outcomes.** Following PT intervention, the patient achieved minimal improvements in ROM, strength, and pain. The patient did, however, demonstrate improvements with glenohumeral joint restriction. Following trial of conservative management, the patient opted for arthroscopic surgery following consultation with orthopedic surgeon. **Discussion.** The treatment of this patient primarily included improving glenohumeral joint biomechanics, which included rotator cuff strengthening, scapular stabilization, and manual therapy as well as treating the patient's shoulder pain symptoms. Treatment was progressed and/or regressed based on the patient's response.

Key Words: Calcific Tendinitis, Shoulder impingement, Shoulder Interventions.

CHAPTER I

BACKGROUND AND PURPOSE

Review of the Literature:

Calcific Tendinitis (CT) of the shoulder is classified as a self-limiting disorder characterized by deposition of calcium salts in one or more of the rotator cuff muscles. The presenting symptom is pain associated with activity that persists for months with spontaneous regression in most cases. Incidence of calcific tendinitis is variable from different authors ranging from 2.7 to 22% in studies taken from asymptomatic patient population[1] Bilateral involvement occurs in 10-20% of cases. The common age group affected is 30-50 years, with the supraspinatus tendons most often affected (80%).[1] The etiology of this pathology remains unclear. Several competing theories of the pathogenesis of calcific tendinitis are present and the exact cause is unclear likely due to biopsies taken at the end of natural history of the disease. Deposits associated with this pathology are semisolid and consist of calcium carbonate hydroxyapatite.

Calcific tendinitis occurs in three stages: the pre-calcific stage, the calcific stage, and the repair stage. The calcific stage has the most pain symptoms and can be broken down in to three subdivisions including: the formative phase, the resting phase, and the resorptive phase. The mild pain most often associated with the formative stage is due to fragmented deposits. The pain in the resting stage associated with nodular deposits and the resorptive phase associated with cystic deposits present with severe pain[1]. Both the pre-calcific and repair stages present with mild/moderate pain.

Specific clinical manifestations include pain in the shoulder with or without restriction of movement. Although symptoms often resolve on their own, they can sometimes persist. Complete resolution occurs at a rate of 6.4% of deposits in a year, with 9.3% resolving within 3 years.[1] Clinical features other than pain include ROM limitations, functional limitations, and decreased shoulder strength. Structural causes of calcific tendinitis pain include chemical irritation of tissues caused by calcium, tissue edema causing pressure, bursal thickening due to irritation causing impingement, and pain by chronic stiffening of the glenohumeral joint.[1]

Treatments begin as non-operative with a trial of conservative management; 50.4% of these cases have resolved pain and return to function at the end of 6 months.[1] Following non-operative management, arthroscopic surgery with debridement is typically performed. However, non-invasive therapies are beginning to emerge as options following conservative management including extracorporeal shock wave therapy and ultrasound guided needling. Presenting symptoms and provider preference tend to determine the course progression and management.

Calcific tendinitis is diagnosed via imaging evaluation primarily done by X-Ray. Radiograph evaluation is completed by AP view (Rockwood View) and shoulder outlet view. The locations of deposits, view of subacromial space, and length and breadth of deposits are observed in AP view. The depth is obtained from the outlet view to obtain full radiographic volume of calcium deposits. Radiologic classifications of calcific tendinitis using the Gartner and Hayer classification system can be classified as 3 types, where deposits are classified based on density and calcification border. Type 1: dense calcifications with well-defined borders; Type 2: dense calcifications with

indefinite borders; and Type 3: transparent with indistinct borders.[1] In general, it is found that radiologic measures have moderate to good reliability with no prognostic value.[3] Ultrasonography may also be used to classify deposits but is less common. MRI may be utilized in identifying deposits along with associated edema and subacromial bursitis.

There are 4 widely accepted theories of pathogenesis. The first is degenerative calcification where intracellular calcium accumulates from old, damaged, and necrotic tenocytes. The second is reactive calcification involving metaplastic fibrocartilage with calcium deposited through an inflammatory mechanism. The third type is endochondral ossification where metaplastic fibrocartilage becomes vascular from bone marrow and calcium deposits without inflammation similar pathogenesis as bone spur formation. The last type is chondral metaplasia, which is described as the erroneous differentiation of tenocytes into bone cells mediated by BMP-2, a bone development protein.[2] No one theory appears superior to another in current literature, as it is proposed that there are genetic components that predispose certain populations to calcific tendinitis.[2]

Initial conservative management of calcific tendinitis involves a formal physical therapy program. Range of motion exercise and improving scapular mechanics can benefit patients with calcific tendinitis. No studies are available that outline specific protocol for patients with calcific tendinitis, but it is often treated similarly to subacromial impingement, a common sequela of calcific tendinitis. Scapular dyskinesis contributes to shoulder pain by altering the position of the humeral head in the glenoid. Scapular muscles including trapezius, rhomboids, levator scapulae, latissimus dorsi, and serratus anterior are active during overhead motion. If movements are not coordinated properly, decreased subacromial space during overhead motion can occur, especially with a

calcium deposit in the rotator cuff. Therapy should be directed at regaining optimal scapular mechanics for better clearance of the supraspinatus tendon and subacromial bursa.[2] Standard therapy that utilizes activation of the middle trapezius, lower trapezius, and serratus anterior helps restore proper balance of scapular movement.

Well organized therapy programs begin with range-of-motion, flexibility, open chain strengthening, and closed chain exercises that lead to reduction of shoulder pain. Common physical therapy practices utilized in conservative management of calcific tendinitis include acetic acid iontophoresis and therapeutic ultrasound. In theory, when acetic acid is applied, it helps dissolve hydroxyapatite crystals in this environment. However, there is little to no evidence that acetic acid iontophoresis affects the improvement of calcium deposit size.[2] Ultrasound studies were conducted displaying reduction in pain and improved quality of life following 6 weeks of treatment. However, pain reduction was not found to be statistically significant which decreased at the 9 month follow-up.[2]

Long-term outcome studies have conflicted results with an overall general consensus that calcific tendinitis is generally a self-limiting disease. A 14-year study that looked at shoulder functional outcomes in individuals with dominant arm involvement revealed that bilateral disease, a large number of calcifications, female gender, and longer duration of self-limiting symptoms were associated with inferior outcomes compared to other counterparts in studied populations.[3] Chronic calcific tendinitis surgery is often indicated after 6 months of conservative management trials.[4] Additional non-invasive therapies are available but are not part of standard practice and are currently being researched. These are minimally invasive non-operative therapies to

reduce pain and remove calcium deposits. Non-operative therapies generally considered are ultrasound guided needling (UGN) and high-energy extracorporeal shockwave therapy (ESWT). UGN is a single treatment procedure that only requires local anesthetics. UGN is a minimally invasive treatment that utilizes a needle to lavage with or without aspiration and is usually combined with subacromial steroid injection.[4] ESWT is an interventional modality applied throughout multiple sessions. It can be described as high-energy sonic pulses measured in millijoules that are applied over calcific deposits and points of tenderness without local anesthetics.[5] ESWT and its biological effects have been reported including denervation of pain receptors, deposit fragmentation, phagocytosis, and neovascularization.[5] Both UGN and ESWT modalities result in reduced pain and significantly improved functional outcome measures after 6 months.[5]

Calcific tendinitis is primarily treated conservatively, and often fails. Surgery is the next option. There are three major surgical strategies that are commonly used by orthopedic surgeons with no superiority of one technique above the other. The first is acromioplasty with removal of calcific deposits. The second is acromioplasty without removal of calcific deposits. The third is to solely debride calcium deposits. Research comparing all three techniques shows no preferred surgical strategies; all three techniques show good functional and clinical results with low complication rates.[6]

There are multiple complications that can occur with calcific tendinitis. Since calcific tendinitis is considered asymptomatic in most patients, any symptom is considered a complication. Pain is the most common complication and is one of the premier symptoms of calcific tendinitis. Pain is usually a more severe and shooting type

of pain in the shoulder with generally no symptoms radiating beyond the elbow or hand. In acute phases, the pain is severe, drastically limiting shoulder range-of-motion with distinct marked tenderness upon tendon insertion. In chronic and subacute cases, pain can be moderate to severe, but shoulder range of motion is often allowed.[7]

Other pathologies that can be considered as a secondary sequela to calcific tendinitis are rotator cuff tears, adhesive capsulitis, and greater tuberosity osteolytis. The most important and most common secondary sequela to calcific tendinitis is adhesive capsulitis, commonly known as 'frozen shoulder'. The etiology for adhesive capsulitis remains relatively unknown. However, changes in glenohumeral capsule stiffness or development of adhesive capsulitis as a secondary result of calcific tendinitis and its pathology progression remain pertinent. Shoulder stiffness resulting from adhesive capsulitis with primary calcific tendinitis is not tolerated well with patients' and must be treated with standard manual therapy. A study done on post-operative arthroscopy patients with calcific tendinitis reported 18% incidence of adhesive capsulitis following the procedure.[7] This prevalence lead to prolonged recovery phases in regaining functional strength and range of motion.

Another complication is the coexistence of calcific tendinitis and rotator cuff tears. The probability of coexistence of these conditions is 28%, with calcium deposits commonly being associated in rotator cuff repair surgeries.[7] In addition to the coexistence there is also an association of rotator cuff tears following the surgical removal of calcium deposits. It is not uncommon for complete and partial tears in rotator cuff bellies to occur following the removal of medium to large calcium deposits. Although

controversial among surgeons, some believe these tears require immediate repair after deposit removal.

Greater tuberosity osteolytis is an additional, but rare, complication of calcific tendinitis. Greater tuberosity osteolytis contains a form of calcification deep on the insertion of the tendon with very severe and persistent symptoms. The calcification build-up can penetrate the bone, developing lesions into the greater tuberosity. These cases often present as severe pain and are likely to be resistant to conservative management, resorting to surgical treatment

CHAPTER II

CASE DESCRIPTION

This case report describes a conservative management trial of calcific tendinitis of the left shoulder. The Patient was a 60-year-old female referred to PT from the orthopedic walk-in clinic for a trial of conservative management to treat diagnosed calcific tendinitis with x-ray imaging. The patient reported ongoing shoulder pain approximately a year-ago and had received cortisone injection relieving pain for multiple months. The patient received another cortisone injection over the summer which helped initially, but had recurring symptoms for the last month, including radiating pain running down the side of her left arm. The patient received another cortisone injection as well as a dexamethasone injection at site of calcific deposits.

The patient reported her worst pain occurred when lifting baking trays at her part-time job at the bakery, and when she accidently bumps into something with her shoulder. The patient also reported pain with overhead activities such as putting items in her cupboard, combing her hair, and putting on a jacket. The patient reported that she avoids all quick shoulder movements and uses her right arm instead of her left whenever she can.

Examination and Evaluation:

The evaluation was based on Dutton's Orthopaedic Examination, Evaluation, and Intervention textbook.[1] An Upper extremity functional index was given to the patient in

the physical therapy lobby prior to her PT evaluation. She scored a 74/80 on the upper extremity functional index. Upon observation, the patient appeared to be in no acute distress, demonstrated a forward headed posture with no left arm swing during walking.

Her examination revealed no obvious deviations of shoulder symmetry or muscle tone and no apparent spine misalignment. Initial AROM was observed with signs of pain and apprehension in mid arc with bilateral shoulder abduction. Bilateral ROM using goniometric measurements are shown in Table 1. Results from the Apley scratch test for internal and external rotation are also presented in Table 1.

PROM had normal end-feels in all motions without pain including bilateral extension and external rotation. PROM was limited and she demonstrated muscle spasm end-feels due to muscle guarding including left sided flexion, abduction, and internal rotation.

Table 1. Initial Shoulder Range of Motion (in Degrees).

	Right	**Left**
Flexion	175	163 (pain noted)
Extension	40	40
Abduction	175	155 (pain noted)
External Rotation (Scratch Test)	T4	T3
Internal Rotation (Scratch Test)	T6	T10 (pain noted)

Right shoulder strength was tested with gross manual muscle testing and showed 5/5 (R) in all motions. Left-sided strength was graded as 4/5 with pain noted in all motions. Scapular strength was tested with manual muscle testing and graded as 5/5

for the lower trapezius, middle trapezius, rhomboids, upper trapezius, and serratus anterior. Glenohumeral Joint play showed the presence of a moderate restriction with GH posterior glides and GH distraction. Anterior and Inferior glides were tested and showed normal joint play.

Multiple diagnostic tests were used to provide diagnostic confidence. The individual special tests along with their sensitivity/specificity values are shown in Table 2. Upon palpation, tenderness and pain were noted over the bicipital groove, greater tuberosity, and supraspinatus tendon on left side. There were no significant deviations of muscle tone when compared bilaterally.

Table 2. Initial Evaluation Special Test results and Sensitivity/Specificity.

Special Tests	**Results**	**Sensitivity Range[8,9]**	**Specificity Range[8,9]**
Neers Impingement	Negative-Bilaterally	0.60-0.84	0.35-0.51
Empty Can/Jobe	Negative-Bilaterally	0.78-0.94	0.40-0.55
External Lag	Negative-Bilaterally	0.35-1.0	0.89-0.98
Speed's Test/Palm up test	Positive-Left, Negative-Right	0.49-0.71	0.60-0.85

Prognosis and Plan of Care:

The initial examination confirmed the presence of calcific tendinitis of the anterior shoulder musculature; potentially of long head of biceps and/or supraspinatus muscles. The patient presented with limited ROM, strength, positive speeds test, and moderate GH joint restriction of the left shoulder. The plan of care included shoulder resistance band plane exercises and supine/side lying AROM exercises including flexion, abduction, external rotation, and internal rotation to increase ROM. Theraband rows and

GH joint mobilization were included in POC to address strength deficits and GH joint restrictions. Imaging was taken by orthopedic clinic but was not available in the patient's chart prior to evaluation but was available the next treatment session.

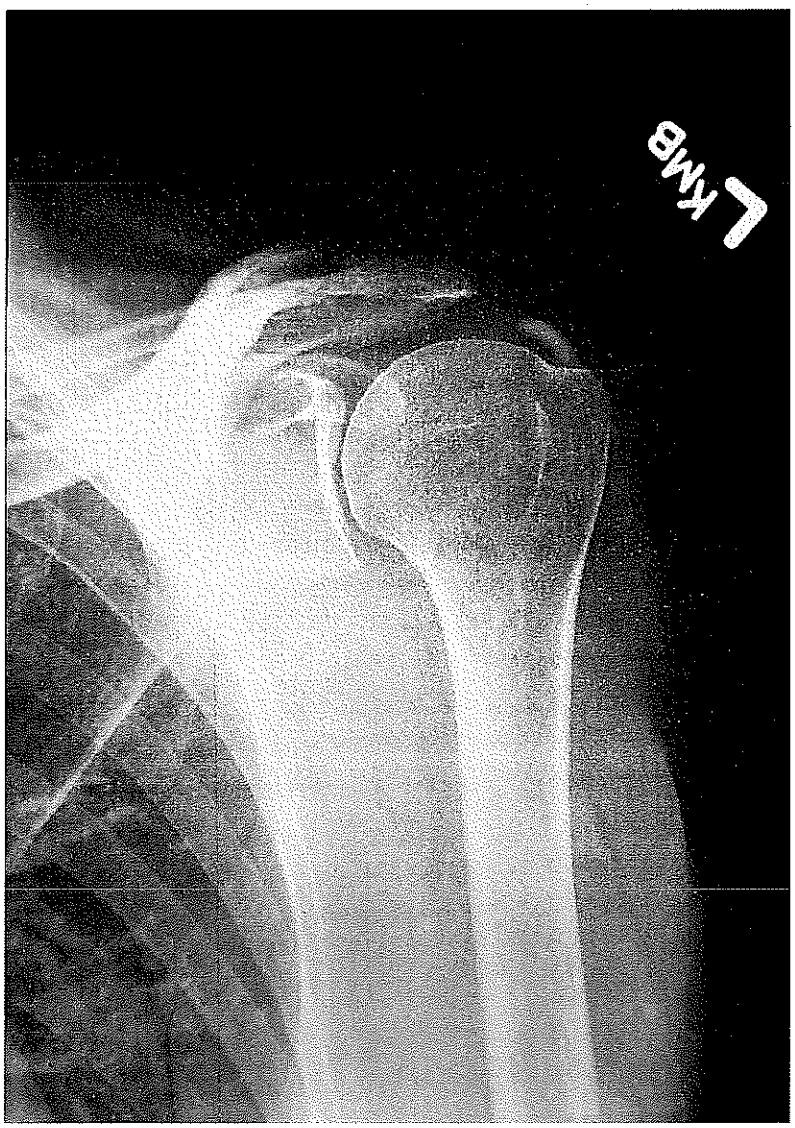

Figure 1. Shoulder X-Ray AP View, Calcific Tendinitis.

CHAPTER III

INTERVENTION

The patient was seen 2 times a week for 30 minutes each session for 5 weeks before follow-up with orthopedics. The therapeutic exercises included ROM exercises, strengthening exercises, manual therapy. Modalities included, iontophoresis, and ultrasound.[10] Iontophoresis was specifically ordered to be included in treatment by orthopedic PA-C and was applied throughout the entire treatment duration.

Weeks 1 and 2 of the intervention was primarily aimed at controlling inflammation, avoiding movements that caused pain, and beginning ROM exercises that do not aggravate the affected tendon/s. A trial of shoulder plane exercises was initiated upon evaluation but were not tolerated by the patient. The POC was altered to address the control of inflammation and pain and ROM.

The new POC included AROM exercises including supine flexion, abduction, supine reverse pendulums, external rotation/internal rotation, elbow flexion exercises, and PROM exercises. The resistance exercises included standing rows with orange Theraband and scapular stabilizing exercises that did not exacerbate painful symptoms.

Manual therapy was performed and included glenohumeral distraction and posterior glides, grades 1-4, with moderate capsular restriction. Normal joint play was attained by the third visit during the second week.

Iontophoresis was applied every session and was tolerated well by patient with no burning or side-effects. Iontophoresis was applied over the area of greatest tenderness which was a portion of the bicipital groove and greater trochanter near the insertion of supraspinatus. The iontophoresis dosage included 1.5 mL fill volume, using 4 mg/mL dexamethasone at 80 mAmps with a 6-hour wear time.

Week 3 interventions included the introduction of free weights into the AROM exercises. The patient subjectively reported that her arm was feeling better, and that her shoulder was feeling better with increased ease of reaching overhead and reaching behind her back. The exercises were progressed to 1 pound dumbbell AROM exercises including supine flexion, side lying abduction, supine reverse pendulums, side lying external rotation/internal rotation, and elbow flexion exercises.

Resistance exercises including standing rows with orange theraband and scapular stabilizing exercises were maintained. At the next treatment, the patient reported shoulder pain exacerbation following the introduction of the 1 pound weight. She was now unable to reach behind her back without pain and had less elevation at the next session 4 days later. Treatment was regressed to the initial treatment level, as the patient was unable to tolerate exercises with a 1 pound weight.

Weeks 4-5 interventions included similar AROM exercises and scapular stabilization exercises with no addition of weight. The patient reported no improvement during these sessions and became frustrated with the lack of results. Ultrasound was included during these treatment sessions to help reduce pain and was given at the patient's request. Ultrasound was applied at 1 MHz, 100% duty cycle, and 1.5 W/cm^2 for

8 minutes over the bicipital groove and anterior shoulder musculature near greater tuberosity.

CHAPTER IV

OUTCOMES

At discharge, the patient demonstrated 5/5 strength on right side and 4/5 strength on left side with pain noted in flexion, abduction, internal rotation, external rotation, and extension. The patient demonstrated normal glenohumeral joint mobility bilaterally. The patient demonstrated minimal ROM improvements at discharge (Table 3). The patient still had occurring pain with shoulder at the end of treatment. The patient scored 75/80 on the Upper extremity functional index.

Table 3. D/C Shoulder Range of Motion (in Degrees).

	Right	**Left**
Flexion	175	168 (pain noted)
Extension	40	40
Abduction	175	165 (pain noted)
External Rotation (Scratch Test)	T4	T3
Internal Rotation (Scratch Test)	T6	T9 (pain noted)

CHAPTER V

DISCUSSION

Throughout the 5-week intervention period, the patient had minimal ROM improvements gaining only 3 degrees of flexion, 10 degrees of abduction, and one measurable spinal segment of Internal rotation during the scratch test. Glenohumeral joint restriction was improved from moderate restriction to normal joint play. Point tenderness remained upon palpation of superior aspect of bicipital groove, greater tuberosity, and supraspinatus tendon.

The patient's chart was left open after the last session pending follow-up with orthopedics; she would be discharged if no further contact was made. After follow-up with orthopedics, this patient was unsuccessfully treated with conservative management of calcific tendinitis. During her visit to orthopedics, she elected arthroscopic subacromial debridement, decompression, and calcium deposit removal to be scheduled at a later date.

It is important to note during this episode of care that the patient inaccurately completed the Upper Extremity Functional Index, which is scored reliable and valid for scoring functional outcomes for shoulder pathology patients.[11] At discharge the patient explained that she completed the questionnaire indicating activities she could complete when using both right/left arm versus just left arm function as I instructed. The inaccurate reporting resulted in skewed results of this patient's functional assessment.

Reflective Practice:

Reflecting upon the results from this case has allowed me to gain a better understanding of the disease process and how to better help manage calcific tendinitis for future patients. Through research and this patient's clinical manifestations, I have gained a better understanding of the pathogenesis and stages of calcific tendinitis which better prepares me for calcific tendinitis cases as a future clinician.

An important topic to note that was not understood are the distinct clinical manifestation differences of calcific tendinitis when compared to tendinitis of rotator cuff musculature. A major component to take into consideration with calcific tendinitis in the general population is that a significant number of patients in this population present as asymptomatic. The key clinical manifestation to differentiate between regular tendinitis and rotator cuff tendonitis is that patients with regular tendinitis present with pain that has persisted for months and spontaneously regresses; rotator cuff tendinitis is commonly considered a chronic overuse injury or traumatic injury. Other notable manifestations include the increased prevalence in female patients, it contains minimal losses in range of motion, and is considered a "self-limiting" disease in literature. This newfound information will aid in creating a differential diagnosis to correctly diagnose calcific tendinitis if a case is presented without diagnostic imaging.

During the initial examination there were essential questions that were not emphasized that would have been pivotal in creating a more effective plan of care and prognosis for this patient. I asked the patient how long she had been experiencing symptoms but did not create a plan of care for an adequate duration of treatment. The

duration of symptoms is important in determining the prognosis of patient care as the literature states that approximately 50% of the patients reduce pain and regain previous levels of function after 6 months of conservative management. In this 6-week trial, the patient did get the chance to achieve this positive outcome because she did not receive conservative management care at the initial onset of symptoms, nor did she receive a full 6-month duration of care.

An additional question that would have been beneficial to include in the questionnaire is how the severity of the shoulder pain varied throughout the duration of her shoulder pain. This is pertinent as calcific tendinitis occurs in phases and some are more painful than others particularly the calcific phase. This could provide a better gauge as to the phase the patient is in during the pathology process and assist with creating a prognosis and/or treatment by giving an accurate estimation of the expected time based on previous studies. Additionally, I could have ensured that my patient understood the questions on the functional assessment and how to complete it correctly so that the results would have been accurate.

Prior to and throughout the examination process, I had made assumptions without full understanding of the pathology of calcific tendinitis. One major assumption I made included a method of trauma that would likely cause micro-trauma to one of the rotator cuff tendons resulting in conventional tendinitis as well a clear diagnosis with the use of special tests. This assumption caused me to dedicate a sizable amount of time performing special tests and attempting to find activities of daily living that would cause trauma to the patient's rotator cuff. An examination technique I would now incorporate after acquiring further knowledge would be movement assessments including tests from

Saurman Functional Movement Assessment. Utilizing these assessments would allow me to identify scapular and glenohumeral muscle imbalances that would likely be evident with movement. Otherwise, I would complete the physical assessment of the examination similar to what I completed in the clinic, testing range-of-motion, strength of scapular and glenohumeral musculature, joint play, observing shoulder and scapula for potential deviations, and palpation of the affected shoulder.

Furthermore, I did not have access to my patient's radiograph before my evaluation as it had not been signed off on the patient's chart yet. This affected my examination as I did not have knowledge of the large calcium deposit in the supraspinatus and continued through the examination process as instructed by my clinical instructor.

Interventions prescribed following the initial evaluation included glenohumeral resistance band plane exercises along with scapular stabilization exercises which were implemented into a home exercise program. During these initial exercises, I overdosed the initial intensity of the glenohumeral plane exercises. This patient had enough range of motion to initiate resistance exercises. If I had knowledge of the calcium deposit, I would have been more conservative with the initial glenohumeral plane exercises given that the deposit could exacerbate symptoms by irritating other shoulder structures involved. As a result, the patient's pain grew worse by the next session, and I regressed her to simple range of motion exercises after receiving access to the shoulder radiograph. This patient tolerated range of motion exercises much better initially, but symptoms flared up when a light weight was added. Reflecting on the situation, I had thought that the addition of weight exacerbated the patient's symptoms. However, after

expanding my knowledge in this area, it is plausible that these symptoms were a part of the calcific phase of the disease progression.

My thought process while treating this patient was to strengthen the rotator cuff muscle. This would increase blood flow to the painful area, promote healing while strengthening the scapular stabilizer muscles, and promote proper biomechanical scapulohumeral rhythm during glenohumeral joint motion. Other interventions applied were glenohumeral joint mobilizations, iontophoresis application, and ultrasound. Glenohumeral joint mobilizations were successful, returning the left glenohumeral joint play back to normal, which was a likely contributing factor in the improvement during the first few weeks of treatment.

After reviewing the literature for calcific tendinitis, glenohumeral joint restriction is common and is considered part of conservative management practice. Iontophoresis was prescribed by this patient's provider and was applied during every treatment session. Initially, after receiving this order I thought iontophoresis application may affect the calcium deposits. Upon reviewing the literature and the effects of dexamethasone, I learned that iontophoresis has no effect on calcium deposit formation for calcific tendinitis patients. Iontophoresis with dexamethasone application is primarily used for pain and inflammation of affected structures, which was likely its intended use in this case. During the last few treatments ultrasound was applied to reduce pain and avoid furthering exacerbation of symptoms.

A major component that may have improved the outcome of this case was the patient education portion of the intervention. Prior to examination, I was unaware of the progression of calcific tendinitis and the variability of the duration of outcomes. Armed

with this information, I will be able to better educate future patients on the progression of the disease, discussing the pre-calcific, calcific, and repair phases of the pathology, to better inform them what to expect throughout the treatment process. This also better prepares them for changes to occur, allowing less frustration and greater confidence for the patient in the treatment they are receiving.

Another important topic on which to educate future patients is that calcium deposits in shoulder musculature is common and that they likely had calcium deposits in their shoulder muscles before experiencing symptoms. This will allow the patient to be more confident in the potential results from therapy as well as allow them to recognize that that their calcific tendinitis is not necessarily limiting, allowing them to be less handicapped from it.

Additionally, patients may not be aware of non-surgical treatments following a trial of conservative management. Ultrasound guided needling and extracorporeal shockwave therapies have produced positive results and are becoming more common in practice. Knowledge of these treatments will allow me to give additional non-surgical treatment options to patients to discuss with their provider if conservative management fails.

Areas in which I would seek further evidence is the effects that the size of the calcium deposits' have on conservative management outcomes. The size of calcium deposits can be determined through plain radiographs since calcium deposits are already taken and graded as part of the confirmation of calcific tendinitis diagnosis. Clinicians could then postulate if patients will have positive outcomes and correlate care according to the size of the deposits. When a patient's radiographs show large calcium

deposits, physical therapy clinicians can refer those patients to appropriate providers. Contrarily, it would allow providers to refer patients with smaller deposits for therapy if there is evidence to support positive outcomes based on deposit size.

The cost associated with this care were fairly reasonable considering it was a failed trial of conservative management. However, based purely on outcomes, the cost of treatment was not worth it given there was not enough time allowed during this trial of care to result in potential positive outcomes. To potentially reduce costs, interventions and modalities for management for this patient would likely have to remain the same. Costs could be reduced based on the number of times the patient is seen per week. For example, this patient was seen 2 times per week for a short period of time. However, the focus of conservative management is to promote function and reduce pain through the duration of the pathology, which could likely be achieved by reducing session frequency to 1 time per week. A proper home exercise program prescription and patient education during initial evaluation and subsequent sessions would likely be successful in managing patients for longer durations.

Information I learned as part of this case will influence my professional development because I will pursue further education into management of calcific tendinitis along with all other joints of the body. Since this pathology is not typically studied, I will need to participate in self-studies of patients with calcific tendinitis and their outcomes with various treatments. These studies will help establish a clinical guideline for patients with calcific tendinitis. This would be beneficial to the medical community, particularly the physical therapy profession, as there is not a clear guideline for students to follow.

When I provide therapy for future patients with calcific tendinitis, I will prescribe more appropriate exercise according to the pain experienced by the patient. I will respect the phases of calcific tendinitis and focus the interventions on increasing function, gaining range of motion, reducing joint restriction, and using proper biomechanics to reduce pain.

Additionally, the biggest takeaway from my newfound information on calcific tendinitis is the prognosis with treatment and the typical duration of the pathology. This trial of conservative management lasted 5-weeks. The patient was referred back to orthopedics and opted for arthroscopic surgery. It is common for calcific tendinitis to heal on its own in 1-3 years, which would be a long time for patients to deal with pain and functional limitations. However, from research it is reported that 50% of conservative management cases reduce pain and return to functional activities following a 6-month period. With this information I now know to allow more time for treatment progressing at a slower rate to improve function. I will be able to give a more accurate prognosis for future patients with calcific tendinitis and inform them that conservative management may take several months. Following a true trial of conservative management, therapists can make appropriate referrals and give patients the option of trying additional non-operative treatments, and/or opt for arthroscopic surgery.

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