Presentation and Conservative Management of Acute Calcific Tendinopathy: A Case Study and Literature Review

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**Background:** The efficacy of a variety of noninvasive, conservative management techniques for calcific tendinopathy has been investigated and established for improving pain and function and/or facilitating a decrease in the size or presence of calcium deposits. Surprisingly, few have reported on the use of traditional therapeutic exercise and rehabilitation alone in the management of this condition, given the often spontaneous resorptive nature of calcium deposits. The purpose of this case is to present the results of a conservative approach, including therapeutic exercise, for the management of calcific tendinopathy of the supraspinatus, with an emphasis on patient outcomes. **Case Description:** The patient was a self-referred 41-y-old man with complaints of acute right-shoulder pain and difficulty sleeping. Imaging studies revealed liquefied calcium deposits in the right supraspinatus. The patient reported constant pain at rest (9/10) and tenderness in the area of the greater tuberosity. He exhibited a decrease in all shoulder motions and had reduced strength. The simple shoulder test (SST) revealed limited function (0/12). Conservative management included superficial modalities and medication for pain and a regimen of scapulothoracic and glenohumeral range-of-motion (ROM) and strengthening exercises. **Outcomes:** At discharge, pain levels decreased to 0/10 and SST scores increased to 12/12. ROM was full in all planes, and resisted motion was strong and pain free. The patient was able to engage in endurance activities and continue practicing as a health care provider. **Discussion:** The outcomes with respect to pain, function, and patient satisfaction provide evidence to support the use of conservative therapeutic interventions when managing patients with acute cases of calcific tendinopathy. Successful management of calcific tendinopathy requires attention to outcomes and an understanding of the pathophysiology, prognostic factors, and physical interventions based on the current stage of the calcium deposits and the patient’s status in the healing continuum.

**Keywords:** calcium deposits, shoulder, simple shoulder test, supraspinatus, therapeutic exercise

Calcific tendinitis may present as an acute or chronic condition, often associated with the rotator-cuff tendons of the shoulder. Of the rotator-cuff tendons, the supraspinatus is most commonly affected, followed by the infraspinatus, teres minor, and subscapularis. Calcific tendinitis typically affects people between the ages of 30 and 60 and has been reported at higher rates in women than in men, with clinically based studies often noting similar trends. The prevalence of the condition has been widely described. In 1941, Bosworth noted an incidence rate of 2.7% when fluoroscopically examining 6,061 patients over a 2-year period. Speed reported rates as high as 7.5% to 20% in asymptomatic shoulders and 6.8% in symptomatic patients, while Refior et al noted calcium deposits in 21% of 195 shoulder autopsies. While the prevalence of calcific tendinopathy has been well documented, data regarding the presentation rate of primary acute calcific tendinitis are absent. Similarly, clinical experience suggests that patients with calcific tendinopathy typically present during the “silent” phase of the condition, as a result of other comorbidities, or during the final phases of the condition. As prevalent as calcific tendinopathy is, reports describing the evaluation and management of this condition from its initial stages are rare, with only 1 case currently being reported in the literature.

There is conflicting evidence regarding the occurrence of rotator-cuff tears in the presence of calcific tendinitis. Similarly, our understanding of the pathogenesis of calcific tendinitis continues to evolve. In 1931, Codman and Akerson postulated that rotator-cuff tears may owe some of their existence to calcium deposits and the defects in the tendon that are left behind on removal or resorption of the deposits. Refior et al identified calcification in >50% of the autopsied shoulders presenting with rotator-cuff tears and suggested that
the pathogenesis of both conditions is identical. While attempting to identify the presence of calcium deposits and rotator-cuff tears, Jim et al.\textsuperscript{13} noted this coexistence in 27% of the patients examined arthrographically. Further discussion has followed suggesting that the development of this condition may be degenerative in nature, resulting from hypovascularity, fibrosis, and ultimately necrosis of the rotator-cuff tendons, which ultimately precedes the development of the calcium deposit.\textsuperscript{1,15,17} Uhthoff and Loehr\textsuperscript{18} suggest that calcific tendinopathy is a multiphase process that allows for not only the depositing of calcium in the tissues but also the spontaneous resorption of the calcium, calling into question the degenerative nature of calcium deposits. Instead, they proposed that calcific tendinopathy involves a reactive calcification process relying on cellular mediation, allowing for both the infiltration and removal of calcium from the affected area.\textsuperscript{18} Further histochemical and biochemical analyses have indicated the potential involvement of calcium-depositing chemicals, the presence of inflammatory and cellular proliferation agents, metabolic factors, and mutations in gene expression for calcium-deposit formation in tendinous tissues, lending support to the theory presented by Uhthoff and Loehr.\textsuperscript{1,10,15,17}

The traditional course of treatment for calcific tendinopathy involves conservative measures followed by surgical intervention if conservative management fails, particularly in unique cases.\textsuperscript{7,10,11,18,19} Given the circumstances under which patients often present with calcific tendinopathy, the development of other comorbidities including adhesive capsulitis is possible. The efficacy of a variety of noninvasive, conservative management techniques for calcific tendinopathy has been investigated and established for improving pain and function and/or facilitating a decrease in the size or presence of calcium deposits. These conservative methods have often included traditional therapeutic modalities and exercise,\textsuperscript{18,19} ultrasound,\textsuperscript{20} a combination of ultrasound and mesotherapy,\textsuperscript{21} microwave diathermy,\textsuperscript{22} radial shock-wave therapy,\textsuperscript{23} extracorporeal shockwave therapy,\textsuperscript{24–26} guided barbotage,\textsuperscript{27–33} and barbotage with steroid injection.\textsuperscript{28} Surgical alternatives have also been suggested, as surgery allows for complete removal of the calcium deposit and surgical augmentation and/or repair of the affected area.\textsuperscript{11,12,35–37} There has also been some discussion that surgical intervention may enhance pain relief associated with treatment, when compared with conservative care.\textsuperscript{14} While the use of various electrical modalities and surgical interventions has been supported for the management of calcific tendinopathy of the shoulder, few have reported on the use of traditional therapeutic exercise and rehabilitation in the management of this condition, given the often spontaneous and resorptive nature of these calcium deposits. Therefore, the purpose of our report is to present the results of a conservative approach for the management of calcific tendinopathy of the supraspinatus, with an emphasis on patient outcomes.

**Case Description**

The patient was a self-referred 41-year-old man (175 cm, 75 kg, left-hand dominant) with complaints of acute right-shoulder pain. He reported an inadvertent episode of forceful right-shoulder abduction and external rotation 36 hours prior. Chief complaints at the time of injury included constant pain at rest (9/10) and significant tenderness in the area of the greater tuberosity. The patient further reported that while he was able to perform internal rotation of the shoulder and place his hand on his abdomen, he was unable to actively elevate and perform external rotation (ER) of the shoulder 5 hours postinjury. To manage the pain the patient initiated the use of nonsteroidal anti-inflammatory medication (600–800 mg ibuprofen, 3 times daily) and local ice massage. Even with limited shoulder motion and pain, he was able to ride a bike on a stationary trainer for 1 hour at an average heart rate of 130 beats/min the next day. Past medical history included lower cervical-spine osteoarthritis and no report of previous shoulder injury. The patient is employed as a physical therapist and faculty member at a local university and is active competitively in endurance-related activities (running and cycling).

When the patient presented to the clinic he was holding his arm in an adducted and internally rotated position. On inspection, the humeral head appeared to be centered in the glenoid, and while ecchymosis was not evident, local swelling over the greater tuberosity was apparent. Questioning and a brief evaluation ruled out the cervical spine as a cause of symptoms. For the shoulder, the patient was unable to initiate active elevation or place the arm behind his back. Passive elevation in the scapular plane was limited to 90° due to pain. ER at 0° of abduction was possible to 0°. Resisted isometrics revealed weak and painful abduction and ER, while adduction, internal rotation, and elbow flexion were strong and painless. The patient was exceptionally tender over the greater tuberosity. He completed a simple shoulder test (SST), received a score of 0/12, and continued to report high pain levels (8–9/10).

At this time, differential diagnosis included anterior glenohumeral subluxation, greater tuberosity fracture, axillary-nerve injury, calcific tendinitis, and rotator-cuff tear. During the initial clinic visit the patient was instructed to continue icing the shoulder and was issued a TENS unit. He was also referred to a general orthopedic surgeon for additional consultation. Radiographic images were obtained and revealed 3 liquefied calcium deposits in the supraspinatus tendon (Figure 1). No degenerative changes or other significant abnormalities were noted. The patient received an intra-articular cortisone injection in conjunction with Marcaine using a lateral approach, which resulted in no change in pain. The surgeon prescribed a sling and narcotic medication (Vicodin) for pain, referred the patient to physical therapy, and suggested additional consultation with a shoulder specialist if symptoms failed to improve.
The patient experienced significant difficulty sleeping and was forced to sleep in an upright position. In addition, the patient, who works as a health care provider in a clinical setting, was unable to function normally with respect to work-related activities. He continued to note no changes in pain level, range of motion, or function. Attempts to perform passive range-of-motion exercises resulted in increased symptoms and were discontinued. However, the patient was able to maintain cardiovascular fitness and intensity levels on the bike as noted previously.

Due to the persistence of acute symptoms, a reassessment of the shoulder by an orthopedic shoulder specialist occurred 4 days after the patient was initially seen by the general orthopedist, 5 days postinjury. Observation revealed a swollen and shiny shoulder. The patient continued to note an inability to elevate the arm and to place the arm behind his back. He did, however, report that pain levels in general had declined slightly but continued to remain constant (7/10). Attempts at movement would often result in pain returning to its previously elevated levels (9/10). A reevaluation of the initial radiographs revealed an uncharacteristic location for one of the suspected calcium deposits, suggesting a possible greater-tuberosity avulsion. The physician ruled out tears of the subscapularis, infraspinatus, and teres minor, as the belly press was negative and light force could be generated for ER. Special tests for the supraspinatus and anterior shoulder instability were not possible due to pain and range limitations. Sensory and motor function of the axillary nerve were assessed via sensation over the lateral aspect of the proximal humerus and performance of an isometric deltoid contraction, respectively. With normal sensation and a small contraction noted, injury to the axillary nerve was ruled out. Due to the patient’s reported levels of persistent pain, a second cortisone and Marcaine injection was performed. This time the injection was given with a posterior approach. Despite incorporation of the anesthetic, once again there was no immediate change in pain. Magnetic resonance imaging (MRI) was ordered for further evaluation of the shoulder soft tissues, specifically the integrity of the supraspinatus, and to rule out a greater-tuberosity avulsion fracture.

At 5 days postinjury, the patient initiated use of Vicodin, which resulted in a significant decline in resting pain (1/10) but did not allow for effective active or passive shoulder motion. Scores from the SST confirmed this level of function (1/12). The greater tuberosity remained acutely point tender, and concerns over the development of adhesive capsulitis surfaced. Rehabilitation exercises consisted of step-out isometric exercises using blue surgical tubing for shoulder internal rotation and ER. Dumbbell curls were also incorporated using a 1-pound weight. In addition, active assisted range-of-motion exercises were performed for flexion, with the patient achieving 90° of shoulder flexion, due predominantly to scapular motion. The patient continued to use the sling regularly and maintained cardiovascular fitness on the bike. Ice massage continued to be incorporated to help mitigate pain in conjunction with the Vicodin only. Six days postinjury, notable declines in swelling and point tenderness were appreciated. While active motions were still difficult, resting pain levels remained low (3/10). Local ice massage was used several times during the day while sling use was reduced. Due to improvements in overall pain, the patient discontinued use of all pain medications and was able to return to sleeping in a supine position.

The patient continued to show marked improvement at 9 and 10 days postinjury. The MRI results were negative for a Bankart lesion or greater-tuberosity avulsion, and no obvious structural damage to the supraspinatus or other soft-tissue structures was apparent (Figure 2). It was therefore concluded that symptoms were likely the result of several dormant supraspinatus calcium deposits that were aggravated with inadvertent shoulder abduction and lateral rotation. This combined position places the supraspinatus in a vulnerable location where it may be pinched between the greater tuberosity and posterosuperior aspect of the glenoid labrum. Despite a negative MRI related to a Bankart lesion, anterior glenohumeral instability was further ruled out later using the apprehension test when range of motion permitted proper positioning for this test. The patient reported that resting pain levels remained low (0–1/10) while palpation revealed minimal tenderness of the greater tuberosity. The patient noted that he...
was continuing to start the car and shift the automatic transmission with his left hand due to the right-shoulder injury. Scores for the SST continued to reflect his level of function, rating shoulder function as a 2/12. Improvements in range of motion were also noted, with the patient being able to actively flex and abduct the shoulder to 90° and 60°, respectively. In addition, he was able to actively reach to the spinous process of L5 with his thumb. Passive range of motion also demonstrated significant improvements, as the patient was able to achieve 135° of flexion and 45° of ER at 0° of abduction; however, pain in the deltidoid region continued to limit all ranges of motion. Strength testing revealed that internal rotation was strong and painless (3+/5), ER was strong and painful (3+/5), and abduction was weak and painful (2-/5). As a result of the improvements, additional exercises were added in an effort to continue improving range of motion and strength while still controlling for pain. Physioball flexion, wall slides, gentle ER stretches, and supine cane exercises were added for range of motion. Scapular retraction exercises, ER walkouts, and bicep curls were also prescribed as tolerated for strength, and the patient was instructed to ice after exercise and again at the end of the day.

Continued improvements were observed at day 12, as the patient reported no pain at rest and rated his pain 4/10 with active shoulder motion. Improvements in daily activity were also noted as he reported using his nondominant affected extremity to start and shift the car and brush his hair with only slight discomfort. SST scores continued to reflect improvement as the patient scored his level of function as 8 out of 12, with limitations only with ball-toss activities, lifting weight to shoulder height, and performing tasks that involved horizontal adduction. Palpation revealed no point tenderness. Range of motion continued to improve as the patient was able to reach fully behind his back, to the level of L2, with slight pain only at end range. Flexion and true abduction showed improvements, which were mirrored by full humeral elevation in the scapular plane. Eccentric lowering did elicit a painful arc, and the patient remained somewhat limited with horizontal adduction, as he was only able to reach the contralateral acromion. Passive flexion; ER at 0°, 45°, and 90° of abduction; and horizontal adduction continued to show improvement, as well. Internal- and external-rotation strength were both noted to be strong and pain free (4+/5). Strength continued to be limited with respect to abduction against gravity, particularly >90° abduction, yet, in a gravity-eliminated position, abduction was within normal limits. Furthermore, at lower degrees of elevation abdution strength was painful but strong. During the visit, it was decided that abduction strength was likely impaired due to impingement of the supraspinatus between the greater tuberosity and coracoacromial arch. A positive Hawkins-Kennedy test for pain and symptom reproduction and a positive empty-can test (4–/5) for pain confirmed this secondary diagnosis. Due to the patient’s improvements, rehabilitation continued to include physioball flexion, wall slides, cane flexion, and ER. Wall stretches for ER at 0° of abduction and passive stretching in all planes using the contralateral unaffected upper extremity were also incorporated. Scapular-retraction exercises and bicep curls were continued, while ER walkout exercises were replaced with side-lying ER using a 1-lb (~0.5-kg) weight.

The patient continued to make improvements as his treatment plan progressed. At 15 days postinjury, he continued to report no pain while at rest, slight pain with activity, and an associated “click” during elevation at or near 90° of abduction. He also noted only inability to toss a ball overhead and lift weight to shoulder height as limitations according to the SST, resulting in an overall score of 10/12. Functionally, the patient exhibited full range of motion for flexion, ER, internal rotation, and horizontal adduction. Abduction in the frontal plane was still limited slightly by pain and a noticeable pinching sensation in the critical zone. Passive range of motion was equal bilaterally, and resisted range of motion was strong and pain free for all ranges except abduction, which continued to present with weakness due to pain. Tenderness of the greater tuberosity continued to be minimal, while Hawkins-Kennedy and the empty-can tests both remained positive. The score for the SST remained high, with the patient achieving 10 out of 12, with limitations only with throwing a ball and lifting a load to shoulder level. As the patient’s level of function increased, additional exercises were incorporated that focused on the rotator-cuff and scapula-stabilizing musculature.

**Outcome**

At discharge, 20 days postinjury, the patient reported with excellent results. Seven treatment appointments were completed and focused on decreasing pain, improving range of motion, and reestablishing glenohumeral and
scapulothoracic kinematics and strength over an 18-day period. Pain levels at rest and with activity decreased to 0/10. Scores for the SST had progressively increased to 12/12. While the painful clicking sensation remained, (~5° arc) with abduction at ~90° of humeral elevation, active and passive range of motion was full in all planes. Resisted tests of shoulder strength, including abduction, were strong and pain free. The only limitation that remained was a mildly positive Hawkins-Kennedy test for impingement. Throughout the treatment plan, the patient had been able to maintain stationary cycling activities for cardiovascular fitness. While activities of daily living and the ability to work had been affected by the shoulder condition, his abilities to perform all activities of daily living and return to unrestricted work as a health care provider were restored during the rehabilitation process.

Additional follow-up with the patient 22 months postinjury continued to reveal successful patient outcomes. He noted SST scores of 12/12 and full unrestricted function with no recurrence of pain. He also reported that the clicking sensation noted at discharge had resolved itself within 1 month of the last appointment. All range-of-motion measures were equal bilaterally, and strength was full and painless. Shoulder-impingement tests were also negative, with the patient again noting resolution of those symptoms shortly after discharge and attributing the improvements in part to the rotator-cuff and scapular-musculature rehabilitation exercises. Furthermore, the patient continued to note no difficulty with activities of daily living and has been able to participate in multiple endurance-related training and racing activities. We obtained permission from the patient to document and publish the findings specific to this case and assured him that his confidentiality would be maintained.

Discussion

The prevalence of primary acute calcific tendinopathy is currently unknown. However, the natural progression of calcium-deposit resorption has led many investigators and clinicians to consider the role of conservative, noninvasive, nonsurgical methods of managing patients with calcific tendinopathy.4,5,10,14,19–31,33,34,39 While many of the conservative treatment methods have focused on improving function, reducing pain, and eliminating calcium deposits within the affected tendinous structures, few have emphasized traditional rehabilitation or therapeutic exercises to manage this condition.18,19 In fact, much of the emphasis has been directed at examining the efficacy of therapeutic interventions designed to break up or disrupt the integrity of a calcium deposit by way of shock-wave therapy or needling to facilitate the natural resorption of the calcium deposit.4,5,10,21,23–31,33,34,39

While these studies provide minimal direct evidence to support the use of therapeutic exercise, their efforts continue to fuel the theory presented by Uhthoff and Loehr18 regarding calcium resorption and the potential benefit of conservative management techniques. When considering the diagnostic evidence, the acute nature of the condition, and the signs and symptoms of the patient at the time of injury, it was evident that the integration of these more aggressive therapeutic modalities was not necessary due to the presence of liquefied calcium deposits, which have been described as part of the natural calcium-resorption process.6,17 As such, the approach used with this patient focused on pain management, range-of-motion and strength restoration, and a return to normal function, with special attention aimed at therapeutic exercise integration.

Calcific tendinopathy of the shoulder may present as either an acute or a chronic condition and is attributed to a collection of calcium hydroxyapatite or calcium phosphate crystals in the rotator-cuff tendons.1,15,17 Unfortunately, the etiology of this condition is relatively unknown; as such, its genesis has often been attributed to tendon fibrosis and necrosis due to hypovascularity.1,18 Further evidence suggests that calcium-depositing chemicals, cellular-proliferation mediators, metabolic factors, and variations in gene expression for calcium-deposit formation may contribute to the development of this condition.1,10,15,17 It has also been noted that exposure to varying degrees of shoulder trauma is believed to initiate a change in an existing symptomless calcium deposit, resulting in active and acutely symptomatic calcific tendinopathy.1

The development of calcific tendinopathy has been described both as a 3-stage process (precalcific, calcific, postcalcific)19 and as a 4-stage process (precalcific, formative, resorptive, healing),1 with considerable overlap accounting for this variability. Under both models, the precalcific phase is often characterized by metaplasia of the fibrocartilaginous tissue. According to Uhthoff and Loehr,18 the calcific stage involves formative development of the calcium deposit, a resting phase signified by fibrocartilaginous tissue bordering the calcium foci suggesting completion of deposit development, and a resorptive phase. Hallmarks of the resorptive phase include (1) variable lengths of disease dormancy and spontaneous resorption initiated through the influx of vascular tissue along the deposit borders and (2) the infiltration of phagocytic cells. It has also been noted that patients presenting during the resorptive period report considerable pain. After the cellular-assisted resorption, the natural course of tissue healing is observed.1,18

Investigations involving imaging modalities and classifications for calcific tendinopathy would also suggest that the condition is a multistage disease process. Both Gärtner types (types I–III) and classifications according to the French Society of Arthroscopy (types A–D) recognize varying degrees of calcium-deposit delineation, consistency, and homogeneity, with initial types (type I or types A and B) representing dense well-defined deposits consistent with chronic and dormant deposits and later types (type III or types C and D) presenting with indistinct borders, transparency, and fluffy appearance, which is often noted during the resorptive phase of the condition.7 Although the reliability of these classification schemes has been questioned,7 ultrasound deposit grading remains consistent with radiographic...
classification systems, in both appearance and symptoms. In acutely painful, symptomatic patients, Chiou et al. presented ultrasound evidence showing a lack of calcium-deposit definition and deposit heterogeneity consistent with what has been reported in patients experiencing the resorptive phase of calcific tendinopathy. Similarly, while examining the role of conservative calcium-deposit treatment and using the Gartner classification system, Ogon et al. noted a negative prognosis and positive prognosis when patients presented with type I deposits, which are commonly chronic and dormant, and type III deposits that are actively acute and associated with the resorptive phase, respectively.

The roles of the physical evaluation and diagnostic imaging were crucial to ensure appropriate diagnosis and successful management of this patient. The patient presented to the clinic with an acute shoulder injury that included significant pain, decreased range of motion and strength, and overall functional limitations. While the differential diagnosis included possible anterior glenohumeral subluxation, greater tuberosity fracture, axillary-nerve injury, calcific tendinopathy, and possible rotator-cuff tears, the physical examinations alone were able to rule out glenohumeral subluxation, axillary-nerve injury, and injury to 3 of the 4 rotator-cuff tendons. Imaging studies enabled the physicians to further rule out a tear of the supraspinatus, any associated fractures, or other soft-tissue injuries and were able to identify the location and nature of the calcium deposits that were likely the cause of the patient’s presentation. While the calcium deposit was not aspirated, the patient’s symptoms and films were consistent with the radiographic data presented by Chiou et al. and Le Goff et al. suggesting a positive relationship between poorly defined calcium deposits and elevated pain levels. Based on the work of Ogon et al., the identification of liquefied calcium deposits also presented preliminary evidence to suggest a positive prognosis relative to a conservative-management approach to the condition. Furthermore, bench research findings regarding the natural course of the apatite-based calcium deposits would also indicate that resorption of the deposits is likely, which provided additional support for a favorable outcome using a conservative approach. Reexamination, evaluation, and outcome measures throughout the rehabilitation process enabled the clinicians to appropriately modify the course of treatment as improvements in the patient’s pain and function were noted. Moreover, follow-up examination and evaluation of the patient provided opportunities to address comorbidities and concerns that surfaced during the recovery process, including adhesive capsulitis and shoulder impingement. The ability to recognize these potential limitations and their potential impact on the successful management of the calcific tendinopathy provided the clinician with the chance to modify the exercise selection to account for these developments.

SST measures and pain scores provided critical information regarding the patient’s functional status and quantitative data to justify our rehabilitation decisions. A variety of shoulder-evaluation instruments and functional scoring systems are available, with many having been compared with one another. Each of the available instruments relies on a combination of self-report (ie, pain, function/disability) and clinical measures (ie, range of motion, strength, function), along with varying amounts of patient and clinician involvement to quantify shoulder-related, functional limitations. The overall scoring, time to complete and score, the measurement properties, reliability, internal consistency, the measures of error estimate, and responsiveness also differ between the assessment tools, making comparisons difficult. While many of these assessment pieces have been used previously to document outcomes in calcific-tendinopathy patients, we selected the SST due to its emphasis on the patient’s perception of function and its comparability to other more complex assessment tools. Although the SST has been criticized for its simplicity, a recent study bolstered its clinical usefulness after an assessment of its responsiveness, noting a clinically important difference of 3.0 SST points for the instrument. With similar information available with respect to pain reports, our ability to track clinically meaningful changes in SST scores and pain enabled us to tailor the rehabilitation progression to continue achieving favorable outcomes.

The reported pain levels and reduced function in this case were similar in nature to an acute case of calcific tendinopathy previously presented in the literature. Wainner and Hasz reported on an acute patient diagnosed with calcific tendinitis as the result of an inoculation. While the cause and onset of symptoms differed between these cases, both presented with point tenderness of the shoulder, significantly reduced range of motion, and reductions in shoulder and rotator-cuff muscle strength due to pain. Wainner and Hasz noted the use of a lidocaine injection and barbotage as part of the initial management of their patient to control pain associated with fragmentation of the calcium deposit, a positive prognostic factor noted on X-ray in our patient. While our patient did not undergo needling, he did receive injections on separate occasions to assist in managing pain, neither of which seemed to significantly alter his symptoms. While the intent of these injections was for pain management, some have noted the potential for slowing the progression of calcium-deposit resorption after corticosteroid injections due to their anti-inflammatory effects. More recent work by Chiou et al. supports the beneficial nature of the inflammatory process in the natural course of calcific tendinopathy; they identified elevated vascularity in areas of successful calcium-deposit resorption. Chiou et al. have also speculated that the increased inflammatory reaction associated with a needling procedure may facilitate calcium resorption. Given the fact that our patient did not undergo barbotage, it is fortunate that the use of corticosteroids did not appear to hamper his full recovery.

The role of pain management through medication appeared to play a considerable role in the progress observed during this conservative rehabilitation approach. Although the patient was initially reluctant to use narcotic
pain medication, he experienced clinically meaningful improvements in pain, which were mirrored by improvements in SST scores. Early efforts to engage in therapeutic exercise were greatly limited by pain. However, noted pain relief enabled the patient to engage in a variety of basic range-of-motion and strengthening exercises at a critical point in the recovery process. The likely onset of adhesive capsulitis was imminent, a development that Wainner and Hasz40 noted as part of their case. The gradual integration of therapeutic exercise mitigated the possibility of adhesive capsulitis and even appeared to facilitate further reduction in discomfort associated with the tendinopathy. Continued improvements were observed in SST scores, justifying the addition of more therapeutic exercises. It should also be noted that in addition to range-of-motion activities, we believe that exercises for the rotator-cuff and scapula-stabilizing musculature, which often serve as the cornerstones of shoulder rehabilitation, were able to contribute to the recovery from both the initial condition and the developing case of shoulder impingement. In retrospect, the exercise selection and progression of this patient’s rehabilitation, particularly while leading up to day 10 when all other soft-tissue injuries were ruled out, were remarkably consistent with general shoulder-rehabilitation strategies.51–53 While the argument could be made that our rehabilitation efforts up to day 10 could have masked the presentation of a patient with a possible anterior shoulder subluxation, we would reference the consistent presentation and progress of a patient in the resorptive stage of calcific tendinopathy, the imaging evidence to support this diagnosis, and the response of the patient to conservative therapeutic intervention, which was again consistent with the potential for positive prognosis noted by Ogon et al.7 Ultimately, over the nearly 3-week period improvements were noted in all areas, full function was restored, and resolution of the calcific tendinopathy’s symptoms and its comorbidities was achieved.

Given the radiologic evidence and acute nature of the patient’s condition, more invasive yet conservative management techniques, including extracorporeal shock-wave therapy and barbotage, were not incorporated. Traditionally, shock-wave therapy is reserved for patients who present with chronic calcific tendinopathy and/or those whose calcium deposits are classified by Gartner et al54,55 as either type I or type II,4,23–26,28,39 with the primary morphological goal being to fragment the existing calcium deposit. Similarly, the incorporation of barbotage is reserved for cases where fragmentation of the deposit is indicated or in instances where a proinflammatory response is desired to facilitate calcium resorption.5,30–32,34 There is considerable evidence regarding the efficacious natures of both shock-wave therapy and barbotage. Extracorporeal and radial shock-wave therapy have been noted to produce favorable short- and long-term reductions in pain, noted changes in calcium deposits, and improvements in shoulder function, with only occasional intensity-related side effects.4,23–28,39 A recent systematic review by Lee et al29 continues to support this notion regarding shock-wave therapy’s effectiveness when managing pain and dysfunction in patients with calcific tendinopathy of the shoulder. However, the need for additional large-scale, prospective, long-term studies remains a limitation associated with this modality.25,26,29 Similarly, while outcomes vary slightly with respect to barbotage, the overall success of needling is favorable,5,30–32,34 albeit slightly uncomfortable for the patient during the treatment.10 The traumatic mechanism of injury and the acute presentation of this patient would suggest an active inflammatory response. Furthermore, the liquefied calcium deposits observed via imaging implied that the natural calcium-resorption process was already underway, not warranting further mechanical fragmentation of the deposit. Therefore, the incorporation of either of these modalities would have been ill advised when reflecting back on the initial presentation of the patient. Had progress during the rehabilitation or failure to achieve optimal long-term outcomes not resulted, one or both of these modalities may have been suitable adjuncts to the treatment plan.

There are many questions regarding the development, progression, and optimal management of calcific tendinopathy. The natural progression concerning the development and resolution of calcific tendinopathy suggests that the condition can resolve without physical intervention. Hence, it is entirely possible that our patient may have recovered without the conservative measures we employed. Though the scientifically based progression of calcium deposits may not warrant physical intervention, the volume of clinical research for both conservative and surgical management of calcific tendinopathy in acute and chronic cases would suggest otherwise. Failure to treat or even a delay in treatment could result in the development and progression of other comorbidities, as were observed in our case, and could result in additional long-term pain and dysfunction. Therefore, a comprehensive conservative rehabilitation strategy aimed at restoring the patient to normal function should be designed to follow the natural progression of an acute calcifying tendinopathy.

**Conclusion**

While a case of calcific tendinopathy itself is not overwhelmingly unique, the acute presentation of this patient, the course of therapeutic exercise, and the documented improvements in pain and function presented here are unlike any others that have been presented in the literature. Knowledge of the pathology, the evaluation and diagnoses, and the importance of routine outcome measures were critical for the implementation of a goal-oriented approach to managing this case of acute calcific tendinopathy. Addressing the concerns of the patient, managing pain, considering the natural progression of this condition, and integrating appropriate modalities and exercise enabled the patient to return to normal functional levels. Certainly other more aggressive operations and invasive yet conservative methods are available to treat...
calcific tendinopathy. However, the positive short- and long-term outcomes of this patient provide support for the use of conservative therapeutic interventions to manage acute calcific tendinopathy of the shoulder.

Acknowledgments

This case was seen at Tri-State Physical Therapy, Seven Fields, PA. The patient was on the service of Dr. Carcia at the time of presentation. The opinions and assertions contained in this manuscript are the view of the authors and do not necessarily reflect the views of Tri-State Physical Therapy.

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