Calcific tendinitis of the shoulder

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Calcific tendinitis of the shoulder is an acute or chronically painful condition that is caused by inflammation around calcium deposits located in or around the rotator cuff tendons. The process consists of a multifocal, cell-mediated calcification of a living tendon that is usually followed by spontaneous phagocytic resorption\cite{1}. After resorption or surgical removal of the deposit, the tendon reconstitutes itself\cite{2}. Calcium deposition also can be latent, however, and asymptomatic in many individuals. When it becomes painful, it usually has an abrupt onset and can severely limit activity even though it is not necessarily activity-dependent. It is believed that the disease only becomes acutely painful when the calcium is undergoing resorption. The diagnosis is made by history and physical examination with specific attention to radiographic evidence of calcification. Patients usually exhibit specific tenderness over the greater tuberosity and symptoms similar to impingement syndrome.

Treatment usually consists of rest and nonsteroidal anti-inflammatory drugs together with localized disruption of the calcifications using a “needling” technique. Failure of these modalities may lead to the need for surgical excision of the lesion, which can be done either open or arthroscopically.

\textbf{Historical review}

In 1907 Painter was the first to describe the radiographic findings in patients with calcific tendinitis\cite{3}. Codman\cite{4} discussed the problem in the 1930s and proposed that degeneration of tendon fibers preceded calcification. Bosworth\cite{5} reviewed radiographs and examined 6,061 employees of an insurance company and found an incidence of 2.7\% of calcific deposits in the shoulder. Of these individuals, 35\% had been symptomatic previously, and 51.5\% of the deposits were in the supraspinatus tendon. In 1978 Bateman\cite{6} observed that deposits at the site of tendon attachment were in a “zone of stress” and an area of hypovascularity. More recently Uhthoff and Loehr\cite{2} proposed a progressive reactive calcification process to describe the disease cycle.

Over the years treatment aimed at decreasing the pain has consisted of rest, heat, nonsteroidal medication, physical therapy, “needling,” and localized injections of anesthetic and corticosteroids. Failure to respond to nonoperative treatment may necessitate surgical intervention. In 1902 Harrington and Codman performed the first reported operative removal of a calcific deposit\cite{4}. More recently arthroscopic treatment for these deposits has been described with successful results\cite{7}. This method continues to be the trend in surgical treatment.

\textbf{Pathogenesis}

The cause of calcium deposition in the rotator cuff is unknown. Decreased local oxygen tension and hypoxia have been suggested as a possible factor\cite{4}. The most common site of occurrence is within the supraspinatus tendon and at a location 1.5–2.0 cm away from the tendon’s insertion on the greater tuberosity\cite{2} (Figs. 1 and 2). It usually has onset in individuals who are more than 30 years old, and it affects up to 10\% of the population. As many as 10\%
of those affected have bilateral deposits, and women seem to be affected more often than men [8]. Most individuals with deposition are asymptomatic. Patients with diabetes are more likely to develop asymptomatic deposits; more than 30% of patients with insulin-dependent diabetes have tendon calcification. According to an arthrographic study [9], a rotator cuff tear may coexist in approximately 25% of patients presenting with calcific tendinitis; small rather than large amounts of calcification are more likely to be associated with cuff tears.

Pain is believed to be an inflammatory-induced response to the local chemical pathologic disorder and to direct mechanical irritation. Neer describes four types of pain associated with calcium deposits [10]. First is pain that is caused by the calcium’s ability to irritate the tissue chemically. The second type is pain caused by pressure within the tissue as it swells. The third is an impingement-like pain caused by bursal thickening and irritation and occasionally by deposit prominence. The fourth type of pain is caused by a chronic stiffening of the glenohumeral joint (“frozen shoulder”) that occurs when the arm is held continuously at the side to avoid irritating the calcium deposit with abduction or overhead activities.

Degenerative calcification

The earliest proposed mechanism for calcific tendinitis was calcification that occurred secondarily within a degenerative tendon [4]. This theory was accepted for some time by many investigators [13–15]. McLaughlin [16] described focal hyalinization of the tendon fibers that led to fibrillation and eventual detachment from the surrounding normal tendon fibers. This produced necrotic debris that then became calcified. More recently Mohr and Bilger [17] described the process as beginning with the necrosis of tenocytes with concomitant intracellular accumulation of calcium, often in the form of microspheroliths or psammomas. Uhthoff [2] points out, however, that calcifying tendinitis peaks in the fifth decade, whereas a degenerative process without healing potential continues to increase with age. He believes the histologic and ultrastructural features of degenerative calcification and calcifying tendinosis are different.

Physical characteristics

What truly constitutes these calcific deposits is still under scrutiny, but they are believed to consist of water (H₂O), carbonate (CO₃), and phosphate (PO₄), and contain basic calcium phosphate crystals. Hamada and coworkers [11] analyzed calcium deposits from patients with calcific periarthritis and concluded that the deposits are composed of carbonate apatite, and that hydroxyapatite, octacalcium phosphate, and tricalcium phosphate were not present. Rowe describes calcific deposits as appearing in one of three forms [12]. The first is a dry, powdery deposit that is the chronic quiescent form. The second form is a soft, putty, or toothpaste deposit that may produce a mild chronic discomfort that is painful or aggravated by impinging in abduction under the acromion. The third is a milky or creamy collection that is usually under pressure. This corresponds to the acute, painful phase in which the deposit is surrounded by inflammation and an acute synovitis or bursitis.
Reactive calcification

Several investigators have elucidated the process of calcifying tendinitis to be actively mediated by cells in a viable environment [18–21]. Uhthoff proposes that the evolution of the disease can be divided into three distinct stages: precalcific, calcific, and postcalcific [2] (Fig. 3).

In the precalcific stage, fibrocartilaginous transformation begins within the tendon at the site of predilection for calcification. This metaplasia of tenocytes into chondrocytes is accompanied by metachromasia, indicating the elaboration of proteoglycan.

The calcific stage that follows is subdivided into three phases: formative, resting, and resorptive. Separated by chondrocytes and fibrocartilaginous tissue septae, calcium crystals are deposited primarily in matrix vesicles that coalesce to form large foci of calcification. In this formative phase, the deposits exhibit a chalk-like consistency (Fig. 4). The resting phase occurs when fibrocollagenous tissue borders the foci of calcification without evidence of inflammation, thereby indicating termination of deposition. The beginning of the resorptive phase is marked by the appearance of thin-walled vascular channels at the periphery of the deposit (Fig. 5). Macrophages and multinucleated giant cells then surround the deposit and phagocytose debris with calcium removal. In this phase the deposit exhibits a thick, creamy, or toothpaste-like material that is often under pressure.

As the tissue undergoes healing during the postcalcific stage, new vascular channels promote fibroblasts to form type III collagen that becomes replaced by type I collagen. The tendon is healed subsequently with fiber realignment and resolution of the calcium deposit.

Radiographic evaluation

Painter first identified radiographic calcifications in the periaricular soft tissue of the shoulder in 1907 [3]. Calcium deposits can be seen on plain radiographs with a full series of shoulder films that include AP, internal and external rotation, scapular Y, and axillary lateral views (Figs. 6–8). These views help to localize the deposit to a specific tendon and show signs of possible impingement. Follow-up radiographs are important to assess disease stage with possible progression or resorption of the deposits. Degenerative radiologic signs are rare in individuals with calcific tendinopathy [2].

DePalma and Kruper [8] classified two radiographic types of calcium deposits in the shoulder. Type I deposits have a fluffy, fleecy appearance with a poorly defined periphery and usually are seen in individuals suffering from acute pain in the resorptive phase. Type II deposits exhibit homogeneous density within a discrete, well circumscribed lesion. Patients with these Type II radiographic findings usually have little to no pain and are believed to be in the late formative or resting phase.

Magnetic resonance imaging (MRI) evaluation is not indicated routinely. Calcifications appear as areas of decreased signal intensity on T1-weighted images, whereas T2-weighted images may show a site of increased signal intensity surrounding the lesion similar to edema (Fig. 9). Loew et al found little correlation between calcifying tendinitis and additional findings associated with subacromial impingement with 16% of patients having a type III acromion [22].

Management

Nonoperative treatment

Numerous nonsurgical modalities have been used to treat painful calcific tendinitis. Nonsteroidal anti-inflammatory drugs are a mainstay, and subacromial bursal steroid injection may be beneficial if the patient shows impingement signs [23]. Gentle exercises with a physical therapist can help maintain range of motion. There is mixed evidence that active therapeutic ultrasound is more effective than placebo ultrasound for treating people with calcific tendinitis [24]. In a well
Needle lavage has been described as an effective treatment that can be performed either in the operating room or in the radiology suite. This technique is best used in patients with an acutely painful shoulder.
in the resorptive phase, and it can help decrease the intratendinous pressure. Use of inflow–outflow needles is helpful, allowing the elution of calcium particles to be seen as they are washed out through the needle. In 1937 Patterson and Darrach first described a needle irrigation technique [26]. Comfort and Arafiles [27] later described the results at an average of 9 years of follow-up in nine patients with symptomatic calcific deposits in the rotator cuff who underwent fluoroscopically-guided barbotage (needle irrigation and aspiration). They believed it was not essential to remove completely the deposit to have a good outcome and that failures apparently were caused by difficulty in locating the deposit with the needle. Treatment with modified ultrasound-guided fine needle technique has been shown to be an effective therapy with a significant clinical response and perhaps greater precision [28]. Using ultrasound-guided needle puncture, Farin et al [29] found favorable results in more than 70% of patients.

**Extracorporeal shock wave therapy**

Most of the shock wave therapy studies originate in Europe where there is more widespread use of this technique. Loew et al [30] prospectively evaluated 195 patients with chronic calcifying tendinitis treated with extracorporeal shock wave therapy (ESWT). There was random assignment to control, low-energy, high-energy groups, and high-energy groups that received either one or two sessions. The results showed energy-dependent success, with relief of pain ranging from 5% in the control group to 58% after two high-energy sessions. Daecke et al [31] prospectively evaluated 115 patients at 4-year follow-up to determine long-term effects and any complications. They concluded that the level of success was energy-dependent and that there were significant differences in radiologic changes between the groups. By 4 years after shock wave therapy, 20% of the entire patient population had undergone surgery on the involved shoulder. The failure rate after ESWT was high, but the treatment was successful and without long-term complications for 70% of the patients.

**Surgical treatment**

**Open versus arthroscopic surgery**

Approximately 10% of the patients who do not respond to nonoperative treatment require surgery
Surgery is indicated for patients who have progression of their symptoms, constant pain that interferes with activities of daily living, and absence of improvement after conservative therapy [33]. Surgery is helpful for the chronic formative phase patients and especially those with impingement symptoms. Harrington and Codman performed the first operative procedure for removal of a calcific deposit in 1902 [4]. Since the 1930s many investigators have supported this procedure [4,34–36]. Rochwerger et al [32] reported on 22 patients who underwent open removal of a calcific deposit and an acromioplasty and found the Constant-Murley assessment score [37] increased from 52.2 points (out of 100 points) at the preoperative examination to 89.3 points postoperatively, with a mean follow-up of 23 months. They concluded that the most favorable results are obtained in patients with the longest interval between onset of disease and intervention (more than 1 year) and with a progressive course of the disease.

Arthroscopic treatment has been shown to have outcomes that are equivalent to those of open procedures, and it has the added benefits of improved cosmesis and possibly a shorter hospital stay, which leads to decreased cost. Interscalene blocks and subacromial Marcaine injections help with postoperative pain. Rehabilitation consists of a simple home exercise program to regain muscle tone and to prevent adhesive capsulitis.

In 1987 Ellman [38] first described his arthroscopic technique involving blind needle aspiration to locate the deposit, followed by excision, and then acromioplasty. Weber [39] augmented the technique with the addition of intraoperative fluoroscopic guidance for needle localization of deposits with excellent results.

Ark et al [7] described the results of arthroscopic treatment of chronic resistant calcific tendinitis of the shoulder in 22 patients with an average age of 49 years and a minimum of 1 year of unsuccessful conservative management. The average follow-up was 26 months and subacromial bursectomy was performed in all patients. Needling of the deposit was confirmed arthroscopically when a snowstorm-like effect was visualized. Intraoperative fluoroscopy was not used. Results were graded as good in 11 patients (50%) with full motion and complete pain relief, satisfactory in 9 patients (41%) with full motion and occasional episodes of pain, and unsatisfactory in 2 patients (9%) with persistent pain. The interval until relief of symptoms was noted to be variable depending on the degree of bursitis and inflammation, but 41% obtained pain relief shortly after surgery, and by 6 months 91% were relieved of pain. Based on follow-up radiographs, 13 patients had partial calcium removal and 9 had complete removal of calcium. The two patients with persistent pain only had partial removal, but the remaining patients with only partial removal did have significant pain relief. The investigators concluded that complete excision of the calcium deposit was not essential to achieve pain relief. The 91% who achieved good or satisfactory results is comparable to the results of open procedures.

Jerosch et al [40] evaluated 48 patients treated arthroscopically with deposit removal, resection of the coracoacromial ligament, and acromioplasty, if there was radiographic evidence of subacromial stenosis. Results showed patients with postoperative radiographic elimination or reduction of the deposits had significantly better outcomes than those without radiographic change and that acromioplasty did not

Fig. 9. (A) Axillary radiograph and (B) MRI showing dense calcific deposit in subscapularis tendon.
improve the results. The investigators’ conclusion was that removal of as much of the calcific deposit as possible would lead to improved outcomes and should be the goal of arthroscopic treatment.

**Authors’ preferred surgical technique**

For surgical treatment of calcific tendinitis, the authors perform an arthroscopic procedure with the patient in the standard lateral decubitus position. Joint distraction of the affected shoulder is accomplished using 10–15 pounds of traction with an arm sleeve, and standard anterior, posterior, and lateral arthroscopy portals are established. The authors carefully evaluate preoperative radiographs to assess the precise location of the deposit. Although it is a good idea to have it available, the authors do not routinely use intraoperative fluoroscopy. A standard intra-articular examination is performed with careful attention to the articular side of the rotator cuff. A strawberry-like lesion is often visible on the undersurface of the area of calcification, representing an area of vascular proliferation. Use of an 18-gauge spinal needle and monofilament suture is helpful to mark this lesion before the transition is made to the bursal side.

It is important to perform an adequate subacromial bursectomy to view all areas of the rotator cuff. As mentioned previously, most patients have calcification within the supraspinatus tendon at an area of 1.5–2.0 cm from the attachment on the greater tuberosity. From the bursal side the calcification within the cuff may appear as an area of indistinct swelling (Fig. 10) or as a bulging blister (Fig. 11). Using preoperative radiographs as a guide and direct viewing during arthroscopy, the calcification area can be disrupted using multiple percutaneous passes with the 18-gauge needle.

![Fig. 10](image1.png)  
**Fig. 10.** Arthroscopic view of bursal side reveals evidence of calcific deposit in rotator cuff tendon.

![Fig. 11](image2.png)  
**Fig. 11.** Arthroscopic view of calcific mass.

![Fig. 12](image3.png)  
**Fig. 12.** Arthroscopic technique of needle visualization.

![Fig. 13](image4.png)  
**Fig. 13.** “Snowstorm” effect often seen with arthroscopic needling or debridement of calcific tendinitis. (From Ark JW, Flock TJ, Flatow EL, Bigliani LU. Arthroscopic treatment of calcific tendinitis of the shoulder. Arthroscopy 1992;8:183–8; with permission.)
spinal needle (Fig. 12). When the correct area is punctured there may be a creamy flow or the release of small granular “snowflakes” (Fig. 13).

The goal of surgery should be to remove as much of the calcification as possible with as little disruption as possible to any normal surrounding tissue. A small-diameter shaver is best used to uncover and debride the area. If the resultant defect is large, a single side-to-side arthroscopic suture may help narrow the defect and prevent tear propagation; however, this should be used with caution because the natural process of the disease is self-healing of the tendon and no increased pressure should be put on the area that was released. After resection of the calcification, a standard acromioplasty should be performed only if subacromial stenosis and true impingement are present. After surgery, the patient’s arm is placed in a sling for comfort, and gentle range of motion exercises are initiated to prevent adhesions.

Summary

Calcific tendinitis of the shoulder is a process involving calcium deposition commonly in the rotator cuff tendons. It is a cell-mediated process that is often chronic in nature, but it is usually self-limiting with regard to its acute pain states. Nonoperative management is still the treatment of choice and is successful in up to 90% of patients. When conservative measures fail, a needling technique or surgical removal may be indicated; the trend is toward arthroscopic management. Acromioplasty should not be performed without radiographic signs of impingement. If a resulting large rotator cuff defect is found after removal of the calcific deposit, it may be worthwhile to close the defect arthroscopically with suture to prevent cuff tear progression and to promote healing.

References


