Calcific tendinitis is the leading cause of shoulder pain. Among patients with calcific tendinitis, 2.7%–20% are asymptomatic, and 35%–45% of patients whose calcific deposits are inadvertently discovered develop shoulder pain. If symptoms are present, complications such as decreased range of motion of the shoulder joint should be minimized while managing pain. Patients with acute calcific tendinitis respond well to conservative treatment and rarely require surgery. In contrast, patients with chronic calcific tendinitis often do not respond to conservative treatment and do require surgery. Clinical improvement takes time, even after surgical treatment. This review article summarizes the processes related to the diagnosis and treatment of calcific tendinitis with the aim of helping clinicians choose appropriate treatment options for their patients.

**Keywords:** Calcification; Tendinitis; Shoulder joint; Conservative treatment; Surgical treatment
icle is intended to help clinicians choose the appropriate treatment options for patients with calcific tendinitis.

ETIOLOGY

The etiology of calcific tendinitis of the shoulder remains controversial between two theories: degenerative calcification and reactive calcification. The theory of degenerative calcification was proposed by Codman and Akerson [4] in 1931. It posits that degenerative changes of the tendon accumulate with age, leading to decreased distribution of blood vessels and reduced local oxygenation of the tissue, which in turn produces hypoxia, thinning and tearing of the tendon, necrosis, and eventually calcification. However, that theory cannot explain why calcific tendinitis has a peak incidence in patients aged 50 years or why it is a self-limiting disease. In 1997, Uhthoff and Loehr [5] proposed the theory of reactive calcification, a series of processes that occur in precalcific, calcific, and postcalcific stages. Among them, the calcific stage consists of formative, resting, and resorptive phases. In the precalcific stage, tenocytes change into chondrocytes, a process called metaplasia, and fibrocartilaginous transformation occurs within the tendon. In the formative phase of the calcific stage, calcium deposits form and increase in size. Calcium deposition then stops at the resting phase of the calcific stage. During the resorptive phase of the calcific stage, calcific deposits are absorbed by cell-mediated phagocytosis, which is performed by cells such as macrophages and giant cells. Acute pain is mainly present in this phase. In the postcalcific stage, the spaces remaining in the tissue where calcium deposits were absorbed is replaced by granular tissue, and remodeling occurs. Calcific tendinitis eventually progresses to bursitis and inflammatory synovitis caused by chemical irritation due to the calcific deposits. Chemical furuncles are formed by swelling and increased local pressure in the tissue. Thickening of the bursa causes collisions in the subacromial space. All of these processes produce various forms of shoulder pain.

CLASSIFICATIONS

Calcific tendinitis is classified as acute (within 2 weeks), subacute (3 to 8 weeks), and chronic (more than 3 weeks), according to the duration of clinical symptoms [1]. Depending on the degree of invasion, calcium deposits are classified as localized or diffused. The diffused form is usually more painful and persists for a longer time than the localized form. Idiopathic calcific tendinitis, type I, is not accompanied by endocrine disease, whereas secondary calcific tendinitis, type II, is accompanied by endocrine diseases such as diabetes. Patients with secondary calcific tendinitis often do not respond to conservative treatment, and they require surgical treatment more commonly than patients with idiopathic calcific tendinitis. Bosworth [6] classified calcific tendinitis based on the size of calcium deposits, with small deposits being less than 0.5 cm, medium deposits being 0.5–1.5 cm, and large deposits being > 1.5 cm.

Neer [7] classified four types of calcific tendinitis based on pain and calcium deposits. Their first type is characterized by pain caused by chemical irritation as a result of the calcium deposits. The second type involves pain caused by increased local pressure within the tissue as it swells. The third type causes impingement-like pain through bursal thickening and irritation by prominent calcium deposits. The fourth type reflects pain caused by chronic stiffness of the glenohumeral joint, such as frozen shoulder.

Many classifications have been attempted based on the morphology of the calcific deposits as observed in simple radiography. In 1961, DePalma and Kruper [1] classified two types of calcium deposits on radiography. Type 1 has a fluffy shape with an ill-defined margin and mainly appears in the resorptive phase of the calcific stage, in which patients complain of acute pain. This disease state is acute calcific tendinitis. Type 2 has homogenously dense calcium deposits with a well-defined margin, and most patients with this type have little or no pain. These deposits appear in the formative or resting phase of the calcific stage, and they reflect subacute or chronic calcific tendinitis.

The French Arthroscopic Society classification divides calcific tendinitis into four types based on the morphology of calcium deposits on radiology [8]. Type A calcium deposits show dense, homogenous, and sharp contours; type B deposits show dense, segmented, and sharp contours; type C shows heterogeneous and soft contours; and type D shows dystrophic calcification at the insertion of the rotator cuff tendon. Loew et al. [9] classified three types of calcific tendinitis based on the pattern of calcium deposits observed on magnetic resonance imaging (MRI). Type A appears as a dense, uniform, and well-defined single deposit; type B is uniform and well-defined with two or more deposits; and type C appears as heterogeneous, widely spread, and ill-defined deposits.

CLINICAL EVALUATION

Calcific tendinitis is diagnosed through patient history, physical examination, and imaging examination. Among patients with calcific tendinitis, 2.7%–20% are asymptomatic, and 35%–45% of patients whose calcific deposits are discovered inadvertently on
simple radiographs develop symptoms [10,11]. The formative phase generally does not show clinical symptoms and is therefore often found by chance, although chronic intermittent pain is occasionally observed. Chronic pain occurs during shoulder forward flexion. In the resorptive phase, severe acute pain mainly occurs suddenly and worsens at night. Patients experience difficulties in lying on the affected side, and shoulder joint movement becomes limited. Patients consciously maintain a posture with internal rotation of the shoulder to relieve pain, and prior to diagnosis, most patients visit an emergency room due to the sudden onset of symptoms and pain. In addition, calcific tendinitis can be accompanied by local heat, redness, and oppressive pain. Therefore, it needs to be differentiated from septic arthritis, which presents with similar symptoms.

Simple radiographic images of the shoulder anteroposterior view, internal and external rotation views, supraspinatus outlet views, and axillary views should be acquired to determine the location of calcific deposits and predict the possibility of collision symptoms. If follow-up images are acquired, changes in the disease stage can be assessed. In general, the size of the calcific deposits does not change significantly over time, although a previous study reported that 18% of patients experienced an increase in the size of calcific deposits after follow-up for an average of 16 months [12]. According to the classification of Depalma and Kruper [1], radiological findings that show a type 1 pattern, with unclear margins and a fluffy or fleecy appearance, can be judged to be in the resorptive phase in which patients complain of acute pain. On the other hand, if a type 2 pattern with a clear margin and uniform density of calcific deposits is shown, most patients will report little or no pain because they are in the formative or resting phase.

In addition to simple radiographs, ultrasonography can be used to assess calcific deposits. It shows hyperechoic areas and an obvious posterior acoustic shadow in the formative or resting phase. In the resorptive phase, on the other hand, hyperechoic areas are relatively reduced, and the posterior acoustic shadow is also reduced or not observed. MRI is not a routine evaluation; however, it is helpful in identifying lesions in the shoulder joint, including the location of calcific deposits and the condition of the rotator cuff. In T1-weighted images, calcific deposits show a low signal intensity, whereas in T2-weighted images, the edema pattern surrounding the calcific deposits can show a high signal intensity.

Generally, calcific deposits appear to have a fluffy shape on radiography and a toothpaste-like appearance on arthroscopic findings in the resorptive phase of calcific tendinitis, whereas they appear homogeneously dense on radiography and have a chalk-like appearance on arthroscopic findings in the formative or resting phase of calcific tendinitis (Fig. 1).

**TREATMENTS**

**Conservative Treatment**

The primary treatment for calcific tendinitis is conservative, and it has a success rate of 30% to 80%. Non-steroidal anti-inflammatory analgesics are used to relieve acute pain, and the affected shoulder joint needs to be rested using an arm sling. When there are signs of collision or the patient is in the resorptive phase, sub-acromial steroid injections are effective in alleviating pain. The ultrasound-guided barbotage technique can relieve pain with decompression effects by aspirating and washing out calcific deposits using an 18-gauge or 22-gauge needle. A 3–5-mL mixed solution of normal saline and lidocaine can be administered to locations with calcific deposits, and the aspiration can be repeated until the deposits are washed away. Afterward, an injection of an additional 1 mL of steroid and 2 mL of lidocaine into the bursa around the calcific deposits can enhance pain relief. A previous study reported that the ultrasound-guided barbotage technique

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**Fig. 1.** Radiographic and arthroscopic findings of resorptive and formative or resting phase of calcific tendinitis. (A) In the resorptive phase of calcific deposits (arrows) appear fluffy-like shape on shoulder anteroposterior (AP) view and (B) toothpaste-like appearance on macroscopic findings observed by arthroscopy. (C) In the formative or resting phase of calcific deposits (arrow) appear homogeneously dense on shoulder AP view and (D) chalk-like appearance on macroscopic findings observed by arthroscopy.
achieved satisfactory results in 70% of patients [13]. It can also be performed under fluoroscopy. When performing the barbotage technique, anesthetics and steroids can be injected into the deposition sites and the subacromial space to enhance the effect of the technique.

A randomized controlled study found that ultrasound therapy improved quality of life and helped relieve pain [14]. The study was conducted at a 0.89 MHz frequency and 2.5 W/cm² intensity for 15 minutes per session. The first 15 treatments were performed five times per week for a total of 3 weeks. The remaining nine treatments were performed three times per week for a total of 3 weeks. Furthermore, steroidal and nonsteroidal anti-inflammatory analgesic were not administered during the study. Ultrasound therapy show effects similar to those of surgery.

Extracorporeal shock wave therapy (ESWT) is also widely used and is one of the most effective treatments for pain relief. A prospective study showed that high-energy ESWT in chronic calcific tendinitis patients had a high treatment success rate and few side effects, however, 20% of the patients underwent surgical treatment due to treatment failure during 4 years [15].

In another study, surgical treatment was more effective than ESWT for homogeneous calcification; however, in heterogeneous calcification, ESWT and surgical treatment showed similar effects [11]. During the 1-year follow-up after ESWT in that study, calcific deposits were not observed in 47% of patients, were resorbed in 33% of patients, and showed no changes in 20% of patients [11]. Thus, ESWT is a treatment that can be performed before surgical treatment. Other studies have also reported ESWT as a successful treatment [16,17].

In sum, various conservative treatment options (ultrasound-guided barbotage and injection, ultrasound therapy, and ESWT) show effects similar to those with surgical treatment and are noninvasive. Therefore, conservative treatment is recommended before surgical treatment. Patients with acute pain should begin passive exercise of the shoulder joint to restore range of motion (ROM) after managing the pain with conservative therapy for 1 to 2 weeks and continue until they experience pain relief. In most patients with chronic pain, the ROM of the shoulder joint is close to the normal range. Thus, strengthening exercises need to be started within the range that is comfortable for the patient. If stiffness is observed in patients with chronic calcific tendinitis, adhesive capsulitis should be ruled out. Pain should be controlled first, followed by passive ROM exercises and pendulum exercises.

**Surgical Treatment**

When conservative treatment does not improve pain, shoulder function can decline, making daily activities difficult to perform. For patients who do not respond to conservative treatment after 6 months, surgery should be considered. One study reported that surgery was performed due to conservative treatment failure in approximately 10% of patients, and it showed the best effects in patients with chronic calcific tendinitis in whom the onset of symptoms was more than a year prior to surgery [18].

In general, acute calcific tendinitis responds well to conservative treatment. However, chronic calcific tendinitis often requires surgical treatment, which can take the form of open surgery or arthroscopic surgery. Both surgical methods remove calcific deposits and have shown satisfactory clinical outcomes. Between them, arthroscopic surgery has the advantages of a short rehabilitation period and cosmetic superiority, and it is a less invasive method that helps protect surrounding tissues and can be used to treat comorbidities such as frozen shoulder and rotator cuff tears. Previous studies have reported no significant differences in the clinical outcomes after complete and incomplete removal of calcific deposits [19]. Other studies reported that patients whose radiographic findings after surgery indicated a removal or reduction of calcific deposits showed better prognoses than those whose calcific deposits remained unchanged [20]. This suggests that it is essential to remove as many calcific deposits as possible while minimizing damage to the rotator cuff. If signs of collision are observed, an acromioplasty procedure is effective.

Whether rotator cuff repair after the removal of calcific deposits affects clinical outcomes remains controversial. In general, patients with rotator cuff repair do not show different clinical results from patients who do not receive such a repair, and further progression of a rotator cuff tear is rarely observed. However, several authors have suggested that rotator cuff repair can facilitate rehabilitation treatment in patients with a combined full-thickness rotator cuff tear [21]. In addition, one study reported that clinical outcomes were satisfactory when using both side-side sutures and suture anchors for rotator cuff tears [22].

**SURGICAL TECHNIQUES**

**Glenohumeral Joint**

A posterior portal is made 2 cm inferior and 1 cm medial to the posterolateral corner of the acromion. After inserting the arthroscope into the joint, an anterior portal is made lateral to the coracoid process and anterior to the acromioclavicular joint. When frozen shoulder is present, arthroscopic capsular release should be performed. Expansion, swelling, or fibrillation of the articular side of a supraspinatus tendon that could have calcific deposits needs to be assessed in detail. If a suspicious area on the joint surface is observed, an 18-gauge spinal needle is passed.
through the supraspinatus tendon at the lesion site 1 cm outside the acromion and placed behind the long head of the bicep tendon. Polydioxanone (PDS) can then be passed through the needle to the supraspinatus tendon for marking, and the spinal needle can be removed while maintaining the PDS at its location. This helps locate calcific deposits in the subacromial space.

**Subacromial Space**

After the appropriate glenohumeral joint exploration, an arthroscope moves into the subacromial space. Arthroscopic treatment of calcific tendinitis is mostly performed in the subacromial space. In general, moderate inflammatory changes, as well as hyperproliferation and thickening of the bursa, can be observed. Afterward, a lateral working portal is made at the anterolateral area of the acromion.

**Bursectomy and Decompression**

If PDS marking was performed to visualize a lesion in the articular joint, decompression is conducted by thoroughly removing the bursa using a shaver with a suction opening through the lateral working portal. The PDS should not be cut during the bursectomy. If no lesion is visible in the articular joint or if calcific deposits are located on the bursal side, comprehensive bursectomy is performed first. Adequate hemostasis using electrocautery is required to obtain a proper surgical field. Edema and swelling can be seen macroscopically in areas with calcific deposits, which helps to find them.

**Calcific Deposit Removal**

After identifying the location of calcium deposits, PDS and the needle used for marking are removed. In the acute or resorptive phases, calcific deposits have a toothpaste-like appearance. In the chronic or formative phases, they have a chalk-like appearance. The deposits can be visually identified using an arthroscope (a 16-gauge needle is most often used). When needling is performed in an area with calcific deposits, calcific deposits can be observed as creamy or snowy. If the deposits are large or if there are difficulties removing them, a small incision can be made in the long axis of the tendon using a scalpel. A probe can be used to remove residual calcific deposits in the tendons. It is unnecessary to completely remove the calcific deposits, which could damage the tendon, because the effect of decompression is more important than complete removal of the calcific deposits. After removing the calcific deposits in the tendon, debridement of the surrounding tissue and removal of floating residual calcific deposits is performed using a shaver.

**Rotator Cuff Tendon Repair and Acromioplasty**

Whether the empty space in the tendon left by removing calcific deposits needs to be repaired is controversial. If the rotator cuff tear is small, repair is not needed. However, if the tear is 2 cm or larger and involves more than 70% of the thickness of the tendon, tendon repair is performed. Torn areas near the supraspinatus tendon insertion are generally repaired using suture anchors. If the tear is located within the musculotendinous junction, side-to-side suturing using PDS is performed with a suture lasso (Fig. 2). If the tear is large, rotator cuff repair using suture anchors is necessary (Fig. 3). However, repair should be performed carefully. Calcific tendinitis is a self-limiting disease, and thus repair can increase the pressure at the removal site. In a study by Lee and Shin [23], approximately 26.5% of patients required rotator cuff repair after arthroscopic removal of calcific deposits, and they found no clinical differences between patients who received rotator cuff repair and those who received simple decompression. Acromioplasty is not required in all patients with calcific tendinitis. In patients with impingement syndrome or obvious osteophytes in the acro-

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**Fig. 2.** Arthroscopic decompression and rotator cuff repair using side-to-side sutures. (A) Preoperative fat suppressed T2-weighted magnetic resonance imaging coronal view shows calcific deposits on the supraspinatus tendon within musculotendinous junction. (B) Arthroscopic findings after removal and debridement of calcific deposits lesion and an approximately 1.0×1.0-cm-sized defect is seen. (C) Arthroscopic side-to-side suture is performed using polydioxanone.
Acromioplasty is effective and widens the subacromial space to prevent collisions.

**AUTHORS’ PREFERRED TREATMENTS**

In some patients, it is difficult to identify the location of calcific deposits, even when the bursa has been sufficiently removed. In such patients, calcific deposits can be located by gently needling the rotator cuff tendon at suspicious areas with a 16-gauge spinal needle (after removing the stylet) and assessing the presence of calcific deposits on the needle tip. When the needle passes through calcific deposits, leaking of the calcific deposits into the subacromial space can be observed.

After removing calcific deposits, large partial or small full-thickness rotator cuff tears are not repaired. Rotator cuff repair is performed only for medium or large full-thickness rotator cuff tears. Side-to-side sutures are placed using PDS for tears at the musculotendinous junction. For other types of tears, rotator cuff repair is performed according to the shape of the tear using suture anchors.

**POSTOPERATIVE REHABILITATION**

Shoulder and elbow motion are allowed immediately after the operation as long as the pain is tolerable, and an arm sling is required for 3 weeks for protection in patients with calcific decompression. Passive and active shoulder ROM exercises should be started immediately, and muscle strengthening exercises should be started 6 to 12 weeks after the operation. Patients can immediately perform light office work, and moderate labor can be started from 6 to 12 weeks after the operation.

If the rotator cuff was repaired using a suture anchor at the tendon insertion site, a shoulder abduction brace needs to be used for 4 weeks, and gradual passive shoulder ROM exercises need to be started immediately after the operation. Muscle strengthening exercises can be started from 6 to 12 weeks after the operation, depending on the size of the tear.

**CONCLUSION**

The primary choice of treatment for calcific tendinitis is conservative, especially in patients with acute calcific tendinitis. However, conservative treatment often fails in chronic patients, so surgical treatment is required. Many patients regard operative treatment as a simple procedure and expect rapid recovery. However, the clinical symptoms of many patients do not improve immediately after surgery and require 6 months or more for complete recovery. Therefore, patients should be given sufficient prior explanation that recovery could be delayed and that intermittent pain could occur for 2 years or more after surgery. In addition, continual follow-up for pain control and recurrence of symptoms is necessary after surgery.

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