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## Calcific tendinitis of the shoulder

### Abstract

Calcific tendinitis of the shoulder is a process involving crystal calcium deposition in the rotator cuff tendons, which mainly affects patients between 30 and 50 years of age. The etiology is still a matter of dispute. The diagnosis is made by history and physical examination with specific attention to radiologic and sonographic evidence of calcific deposits. Patients usually describe specific radiation of the pain to the lateral proximal forearm, with tenderness even at rest and during the night. Nonoperative management including rest, nonsteroidal anti-inflammatory drugs, subacromial corticosteroid injections, and shock wave therapy is still the treatment of choice. Nonoperative treatment is successful in up to 90 % of patients. When nonsurgical measures fail, surgical removal of the calcific deposit may be indicated. Arthroscopic treatment provides excellent results in more than 90 % of patients. The recovery process is very time consuming and may take up to several months in some cases.

### Keywords

Rotator cuff • Tendons • Tendinitis • Calcific tendinitis • Calcification, pathologic

This article explains the causes and development of calcific tendinitis of the shoulder, with special focus on the diagnosis of the disorder and the existing therapy options. It also describes common surgical procedures employed in the management of the condition and discusses the results of surgical and conservative treatment presented in the literature.

The prevalence of calcific tendinitis of the shoulder in asymptomatic patients is about 10 % of the population.

► **Supraspinatus tendon**

► **Diabetes mellitus**

In younger patients, rotator cuff defects are generally not associated with calcific tendinitis of the shoulder.

The formation of calcific tendinitis of the shoulder is not a process of tendon tissue degeneration.

Different diagnostic techniques, classifications and follow-up periods translate into significant differences in the epidemiological information given in the literature. The prevalence of calcific tendinitis of the shoulder (also referred to as calcifying tendinitis or tendinosis calcarea) in asymptomatic patients is about 10 % of the population. It mainly affects individuals between 30 and 50 years of age [27, 28, 30, 51].

About 80 % of the calcific deposits are located in the supraspinatus tendon, 15 % in the infraspinatus tendon and 5 % in the subscapularis tendon. The predilection site in the ► **supraspinatus tendon** is 1.5 to 2 cm proximomedially from the greater tuberosity [35]. Based on the research results available to date, the incidence of the disorder is gender-neutral, although some literature sources reveal that males tend to be affected at higher rates [15, 30] than females [28, 51, 52]. There are striking differences between publications in terms of the rate of bilateral occurrence, which varies between 8.8 and 40 % [33, 45]. Various studies suggest that over 50 % of patients with calcific deposits develop clinical symptoms as the disorder progresses [4].

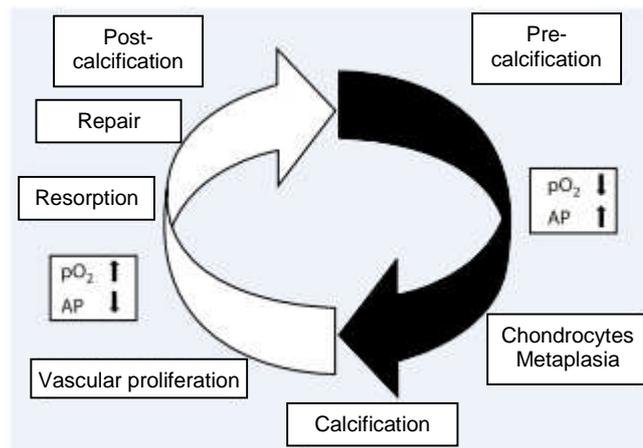
Patients with ► **diabetes mellitus** are more likely to develop calcific deposits. In fact, over 30 % of patients with insulin-dependent diabetes mellitus have calcifications of the rotator cuff [22].

In general, rotator cuff defects are not associated with calcific tendinitis of the shoulder. A European/American study found a co-prevalence of only 1 % [36]. However, in a study conducted by Jim et al. [24] with the help of shoulder arthrography, 25 % out of 81 patients with an average age of 61 years revealed rotator cuff defects [24]. This significantly higher number of defects is assumed to be attributable to the advanced age of the subjects. It is known that the incidence of asymptomatic rotator cuff defects in individuals over 60 years of age is over 25 % and increases at an even higher rate with increasing age, whereas the occurrence of the disorder in people under 50 years of age is below 5 % [32].

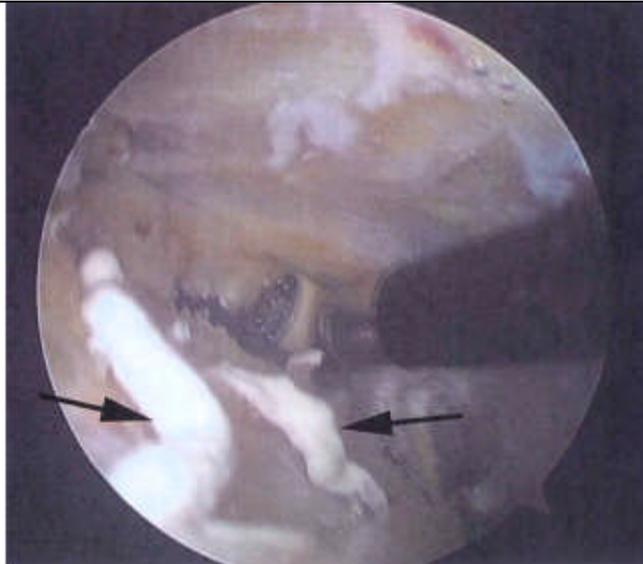
**Etiology and pathogenesis**

Several theories about the etiology of calcific tendinitis of the shoulder will be discussed in this article. Codman [6] was one of the first researchers to investigate the pathogenesis of the disorder as early as in 1930. His theory is based on the degenerative calcification of the tendon tissue. However, as demonstrated by Uthoff and Loehr [50], degenerative calcification of tendon tissue is not responsible for the formation of the condition. This may be explained by the fact that in patients of advanced age calcifications of the tendon are less frequent, while tendon degeneration and tendency to rupture increase substantially.

In general, calcific tendinitis is self-limiting and can be divided into 3 stages (► **Fig. 1**, [49]):



**Fig. 1** ◀ Formal pathogenesis of calcific tendinitis;  $pO_2$  partial oxygen pressure, AP alkaline phosphatase. (modified from [49, 50])



**Fig. 2** ▶ Spontaneous extravasation of calcifications under pressure after electrothermal bursectomy; calcium with toothpaste-like consistency: typical of resorptive phase

- pre-calcification stage,
- calcification stage,
- post-calcification stage.

**Pre-calcification stage.** This stage is initiated by chondral metaplasia of tenocytes within the tendon. This causes increased production of proteoglycans and formation of fibrocartilaginous tissue at the predilection sites.

**Calcification stage.** This is the stage in which the disorder becomes clinically apparent. It can be divided into the following phases:

- formative phase,
- resting phase,
- resorptive phase.

During the formative phase, hydroxyapatite crystals accumulate between the chondrocytes and the fibrocartilaginous tissue, forming small calcium deposits. These are primarily separated by fibrocartilaginous septa, but merge with each other as the disorder progresses. In this phase, the calcifications have a chalk-like, crumbly consistency.

During the resting phase, the deposits with fibrocartilage are walled off, but there are no signs of an inflammatory process.

The resorptive phase is initiated by increased vascularisation of the encapsulated deposit. The macrophages and multinucleated giant cells surrounding it form granulomas in which the calcific deposits are resorbed. During this process, the calcifications are liquefied and change to a ▶ **toothpaste-like consistency**.

The histomorphological changes give rise to an increase in volume resulting in higher pressure which may cause ▶ **spontaneous perforation of the calcific deposit** and leakage into the subacromial space (■ Fig. 2).

**Post-calcification stage.** The post-calcification stage follows the resorptive phase. It is characterised by the migration of fibroblasts into the defect zone. This leads to the formation of ▶ **scar tissue** consisting primarily of type III collagen which is replaced by type I collagen in the course of the remodelling process.

The typical pathological mechanism can be easily explained on the basis of the histomorphology of the individual stages. However, the mechanisms that actually initiate these stages are still not fully known. One theory discusses ▶ **local hypoxia** caused by tissue hypoperfusion, which triggers the transformation of tenocytes into chondrocytes. This theory is supported by the fact that the number of vessels found at the predilection sites of calcification is very low [41].

In the formative phase, the calcifications have a chalk-like, crumbly consistency. The histomorphological changes in the calcification stage give rise to an increase in volume resulting in higher pressure.

▶ **Toothpaste-like consistency**

▶ **Spontaneous perforation of the calcific deposit**

▶ **Scar tissue**

▶ **Local hypoxia**

Several phases may exist in parallel within one calcific deposit, and the chronological sequence of the individual stages is not always regular.

Many patients experience excruciating pain during the resorptive phase.

- ▶ **Sudden pain at rest**
- ▶ **Crystal-induced bursitis**

Large calcific deposits may evoke typical impingement symptoms.

- ▶ **Pain at rest during the night**

Radiography of the affected shoulder in three planes is the standard imaging procedure.

**Table 1 Classification by radiotransparency and appearance (acc. to [16])**

Type	X-ray morphology	Stage
I	Dense calcific deposit Circumscribed, well-defined periphery	Formative/resting phase with macroscopic chalk-like hard consistency (■ Fig. 3)
II	Transparent or fluffy, poorly defined periphery Morphologically not classifiable as type I or III	Early resorptive phase
III	Fluffy, transparent, poorly defined periphery	Late resorptive phase (■ Fig. 4)

Despite wide recognition, the theory developed by Uthoff and Loehr [49, 50] is not undisputed in all of its details. Histomorphological examinations of intraoperative preparations revealed that several phases may exist in parallel within one calcific deposit. These findings cast doubts on the theory of a temporal sequence of the disorder [45]. They are corroborated by irregularities in the time profile as the chronological sequence of the individual stages is not always regular, which may cause incomplete resorption of the calcific deposit and persisting residual complaints [45].

### Clinical symptoms

The clinical symptoms of calcific tendinitis vary significantly and primarily depend on the stage the disorder has reached. Moreover, the severity and nature of the pain perceived is correlated with the calcification size and location. While clinical symptoms may be absent in the pre-calcification stage and in the formative and resting phase, many patients experience excruciating pain during the resorptive phase. This is attributable to the fact that this phase is characterised by a concomitant severe inflammatory reaction with release of pain mediators and by rising pressure in the deposit caused by increasing liquefaction [45]. The predominant feature of this phase is ▶ **sudden pain at rest** which may worsen during movement.

If the tendon is perforated and extravasation of liquefied deposits into the subacromial space occurs, the resulting ▶ **crystal-induced bursitis** will dominate the clinical picture. The patient inevitably holds the affected arm in internal rotation to evade pain and complains about pain radiation in ventrolateral direction to the proximal upper arm and partly to the wrist or neck, depending on the location of the deposits. Owing to the pain reflectory inhibition of muscular function, active abduction of the affected shoulder is significantly restricted in some patients and may present the clinical symptoms of supraspinatus tendon rupture. Large calcific deposits may evoke typical impingement symptoms upon arm abduction, either indirectly because of concomitant inflammation of the bursa or directly due to the narrowing of the subacromial space. These symptoms are characterised by pain radiation to the proximal upper arm with positive painful arc between 60 and 120° and by ▶ **pain at rest during the night** when lying on the affected shoulder.

### Imaging techniques

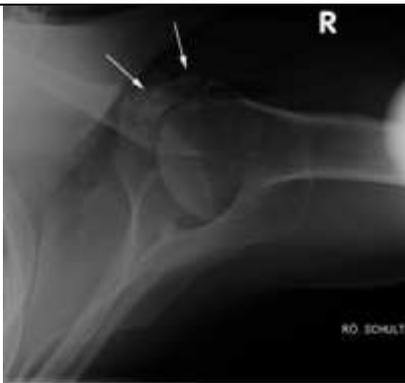
#### X-ray diagnosis

Radiography of the affected shoulder in three planes (true AP, axial, Y-view [22]) is the standard imaging procedure in the diagnosis of calcific tendinitis. The true AP projection can be combined with an additional X-ray examination with the arm in internal rotation to ensure that the head of the humerus does not interfere with the calcific deposits in the infraspinatus tendon region, causing them to form borders. The axial view is especially useful in the diagnosis of calcific deposits in the subscapularis tendon region (■ Fig. 3).

A variety of classifications have been proposed in the X-ray diagnosis of calcific deposits. However, in many cases these classifications do not match the clinical symptoms and intraoperative findings. DePalma and Kruper [7] distinguish between two X-ray morphologies according to periphery definition and radiotransparency [7]:

- Type I: transparent deposits with poorly defined periphery,
- Type II: dense deposits with well-defined periphery.

The classification defined by Gärtner and Simons [16], generally used in German-speaking countries, reflects the chronological sequence of the individual stages of calcific tendinitis (■ Table 1, ■ Figs. 3 and 4, [16]).



**Fig. 3** ▲ Axial view of right shoulder with large calcific deposit (*arrows*) in the subscapularis tendon region; stage I according to Gärtner and Simons [16], homogeneous and dense appearance



**Fig. 4** ▲ Calcific deposit in resorptive phase (type III according to Gärtner and Simons [16]), located above the greater tuberosity; poorly defined periphery



**Fig. 5** ▲ Multilobular calcific deposit (*arrows*) in the supraspinatus tendon reaching far below the acromion

It ensures precise anatomical localisation relative to other landmarks such as the greater tuberosity and biceps tendon [25]. This enables faster and easier intraoperative localisation and eliminates the need for time-consuming large-area removal of the bursa.

In practice, however, type II of this classification is difficult to delimit and does not allow accurate correlation with one of the stages of the disorder.

Regrettably, experience gathered in practice confirms again and again that the radiological classification does not reliably match the macroscopic findings. Radiological type I calcifications according to Gärtner and Simon [16], for example, may not have a chalk-like, crumbly consistency, but may be found to be pasty, which is generally more typical of deposits in the resorptive phase (■ Fig. 2). This observation has been confirmed by other authors [45]. Another weak point of this classification is that the size of the calcification is not considered in the prognosis.

Bosworth [4], on the other hand, proposed a classification of calcific deposits according to their size, distinguishing between small (<0.5 cm), medium (0.5 – 1.5 cm) and large (>1.5 cm) deposits, and suggested that especially the larger ones are clinically relevant.

Yet another classification into four categories (A to D) is used by the French Society for Arthroscopy (SFA) [35]. But again, in many cases the radiological diagnosis cannot be reliably correlated with intraoperative findings [31]. However, this is the only classification in which multilobular or multiple calcific deposits are classified in a separate category (B) (■ Fig. 5). Whether this is relevant in the clinical development of the disorder, remains to be established.

### Ultrasound imaging

Ultrasound imaging is still an important procedure in the diagnosis of shoulder tendinitis. In part, this is because ultrasound imaging enables ► **dynamic examination** and ► **pre-operative localisation** and marking of the calcific deposit site [13]. Farin [11], Farin and Jaroma [12] and Farin et al. [13] were able to prove that there is a high correlation between the consistency and sonomorphology of the calcification. They demonstrated that in the formative phase deposits with solid consistency lead to complete sonographic sound extinction and are therefore well delimited from the surrounding tendon tissue (■ Fig. 6). Deposits with an increasing degree of liquefaction in the resorptive phase generate a much weaker acoustic shadow and are more difficult to discern from the surrounding tissue. This is attributable to the fact that in the formative phase calcific deposits have a high density of hydroxyapatite crystals and thus cause strong ultrasound reflection. In the resorptive phase, however, the inflammatory reaction causes the amount of fluid and the cell count to increase, which leads to a substantially reduced reflection of ultrasound waves.

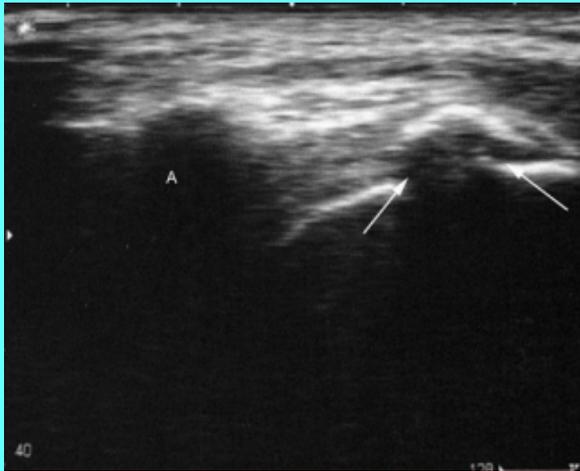
Ultrasound imaging should be performed in the course of the clinical development of the disorder and especially during pre-operative preparations.

In practice, the radiological classification according to Gärtner and Simons does not always reliably match the macroscopic findings.

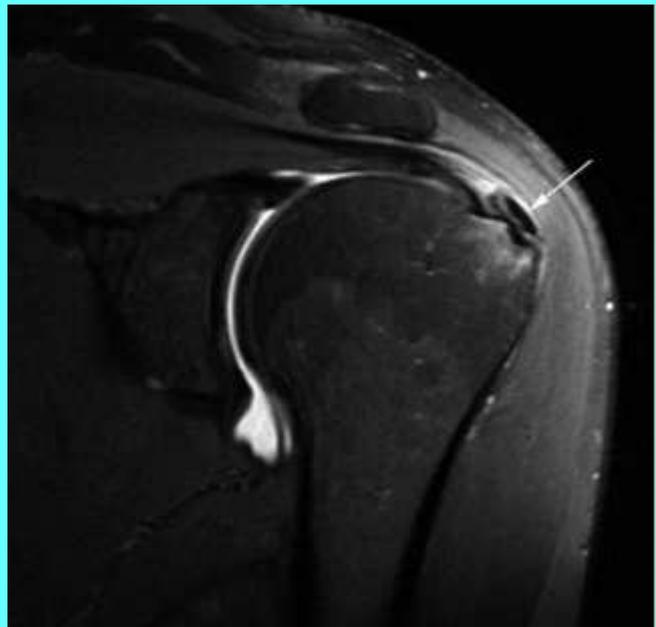
- **Dynamic examination**
- **Pre-operative localisation**

High correlation between the consistency and sonomorphology of calcific deposits

Ultrasound imaging should be performed in the course of the clinical development of the disorder and especially during pre-operative preparations.



**Fig. 6** ▲ Ultrasound image of a calcific deposit (*arrows*) near the supraspinatus tendon attachment with complete sound extinction of the subjacent greater tuberosity, A acromion



**Fig. 7** ▲ Paracoronal magnetic resonance image of left shoulder with hypointense oval calcific deposit above the greater tuberosity and hyperintense surrounding oedema

▶ "Magic-angle" phenomenon

▶ Dystrophic calcification

▶ Septic arthritis

▶ Subacromial bursitis

▶ Supraspinatus tendon rupture

### Magnetic resonance imaging

Magnetic resonance imaging is not one of the imperative standard procedures in the diagnosis of calcific tendinitis because the little extra information it delivers does not justify the high costs it generates [22]. T1- and T2-weighted sequences show a hypointense calcific deposit, while T2-weighted images additionally reveal a hyperintense surrounding oedema (■ Fig. 7). Calcific deposits shown on T1-weighted images must be analysed very carefully as the so-called ▶ "magic-angle" phenomenon may simulate a calcific deposit or supraspinatus tendinitis [10]. Loew et al. [29] performed MRI scans of a number of calcific tendinitis patients, but found only few associated concomitant pathologies that could be co-responsible for symptoms similar to those caused by impingement syndrome.

### Differential diagnosis

▶ **Dystrophic calcifications** at the insertion of the supraspinatus tendon may be misinterpreted as calcific deposits. However, they are substantially smaller and are located directly above and occasionally in contact with the greater tuberosity. In general, calcium deposits in calcific tendinitis patients are located 1 to 2 cm cranially from the greater tuberosity and have no bone contact.

It is also necessary to perform differential diagnosis against ▶ **septic arthritis**. This pathology can usually be diagnosed on the basis of higher inflammation levels, by puncture or history after possible infiltration.

Moreover, ▶ **subacromial bursitis** with or without concomitant pathology may also imitate the symptoms generally caused by calcific tendinitis.

Owing to the pain reflectory loss of strength, calcific tendinitis may simulate the clinical picture of ▶ **supraspinatus tendon rupture**.

Differential diagnosis against a lesion of the postural muscles of the long biceps tendon in the sulcus (pulley lesion) must also be considered because many calcific deposits are located in close vicinity. Whether pain caused by calcific tendinitis covers up complaints associated with the long biceps tendon may be difficult to evaluate.

## Treatment

### Conservative therapy

There is general agreement that the treatment of calcific tendinitis should be started with conservative therapy methods [22, 39, 45]. The therapy of choice is the administration of NSAIDs (non-steroidal anti-inflammatory drugs). If patients suffer excruciating pain, especially in the resorptive phase, central-action analgesics may be used in addition to oral medication. Moreover, patients, especially those with extreme pain in the resorptive phase, may need to wear an arm sling for ► **shoulder relief**.

Physiotherapy exercises do not have top priority in the acute pain setting as they would further aggravate pain. In fact, physiotherapy cannot influence pain activators such as mechanical irritation or pressure increase in the deposit during the acute phase.

In order to achieve instant temporary alleviation of pain, subacromial injections of long-acting local anaesthetics can be given. When used in combination with corticosteroids, this method may even provide long-term pain relief [22, 45, 54].

Wölk and Wittenberg [54] report good or even excellent results in 70 % of patients treated with conservative therapy methods over a 60-month follow-up period. Still, 30 % of the subjects in this study continued to have complaints and required additional treatment [53, 54]. Noel [37] achieved good or even excellent improvement in pain symptoms in 50.4 % out of 125 patients treated conservatively.

There is general agreement that the conservative therapy approach should extend over at least 3 to 6 months [39]. However, the specific clinical picture in each patient and the individual severity of pain determine how well and for how long the treatment can be tolerated. Consequently, ► **prognostic factors** concerning the development of the disorder over time, which may be important when taking decisions for or against surgery, play a crucial role. In a study conducted on 420 patients, Ogon et al. [39] were able to demonstrate that very large calcific deposits located below the anterior margin of the acromion or, in a lateral view, medially below the acromion have a very poor prognosis. This also applies to bilateral calcific deposits. On the other hand, deposits in the resorptive phase, which have a fluffy appearance with poorly defined periphery in the X-ray image and do not produce sonographic sound extinction, have a much better prognosis [39]. As far as the negative prognostic factors are concerned, it is to be noticed that due to the size or location of the deposit the sliding capacity of the rotator cuff below the coracoacromial arc is diminished, which, consequently, contributes to inducing extrinsic impingement. The local growth of the calcific deposit, accompanied by a swelling of the tendon tissue during the resorptive phase, or the bursitis resulting from this process may aggravate the pain symptoms experienced by the patient.

### Extracorporeal shock wave therapy

If conservative therapy methods fail to provide a substantial improvement of pain symptoms, extracorporeal shock wave therapy (ESWT) can be used as an additional non-invasive procedure [18].

Physically speaking, shock waves are ► **high-energy pressure pulses** characterised by ultrafast pressure rise (within a few nanoseconds) and high peak pressure (>10 MPa) in their focus, followed by a tensile wave component (negative pressure).

Basically, one distinguishes between high-energy and low-energy shock waves and between focused and radial shock waves. They are generated by different types of systems (ballistic, electromagnetic, electrohydraulic and piezoelectric principles (■ Fig. 8).

The biological effects produced by shock waves in the treatment of calcific tendinitis result not so much from the direct mechanical pressure pulse, but rather from the indirectly induced cellular reaction, which leads to hyperaemia and neovascularisation and, consequently, to the dissolution of the calcific deposits [17, 18]. The energy applied during the treatment, measured in mJ/mm<sup>2</sup>, depends on the energy flux density in the shock wave focus. In principle, treatment can be performed at low energy levels [40]; the energy flux densities at these settings may reach up to 0.08 mJ/mm<sup>2</sup>. However, high-quality clinical studies have revealed that high-energy focused shock wave therapy provides significantly better results than low-energy treatments [18].

The standard therapy of choice is the administration of NSAIDs.

#### ► Shoulder relief

The conservative therapy approach should extend over at least 3 to 6 months.

#### ► Prognostic factors

Deposits that diminish the sliding capacity of the rotator cuff below the coracoacromial arc result in a negative prognosis.

#### ► High-energy pressure pulse

The biological effects produced by ESWT in the treatment of calcific tendinitis result primarily from the indirectly induced cellular reaction.

In general, patients receive 2 to 3 shock wave treatments at weekly or fortnightly intervals.

► **Application pain**

Shock wave treatment is an evidence-based therapy with an evidence level of I.

► **Puncture of calcific deposit**

The success rates of surgical removal of calcifications is highest among patients with calcific deposits in the formative phase.

► **Subacromial decompression**

In general, patients receive 2 to 3 shock wave treatments at weekly or fortnightly intervals [26]. In individual cases, common analgesic measures are adopted to eliminate the ► **application pain**. Treatment must be performed under X-ray or ultrasound control.

Contrary to other procedures, the effectiveness of shock wave therapy in the treatment of calcific tendinitis is supported by strong scientific evidence from high-quality randomised controlled studies. As a result, ESWT is an evidence-based therapy with an evidence level of I [1, 18].

### Needling

Needling means ► **puncturing the calcific deposit** repeatedly with a needle under direct ultrasound guidance. This is done under local anaesthesia. During the procedure, doctors may try to inject saline solution into the calcification and then aspirate the fluid with the fragmented deposits [13].

The literature dealing with this treatment option has produced vastly differing results so that no verified evidence is available that would confirm the effectiveness of this method, all the more so because the stage of the condition has not been considered in most of the publications. The results of needling, especially when assessed after long follow-up periods, can be compared to the outcome of conservative therapy without needling [45, 47].

### Surgery

If the disease remains unresponsive to conservative management for a longer period of time, surgery may be considered to resolve the condition. Indications for surgery are progressive pain, strain-related persistent pain in daily activities or failure of conservative treatment over at least 6 months [20]. These criteria apply to about 10 % of calcific tendinitis patients [42].

The success rates of surgical removal of calcifications is highest among patients with calcific deposits in the formative phase.

### Results of calcific deposit removal by open surgery

Harrington and Codman [6] were the first surgeons to perform surgical removal of calcific deposits in the 1930s.

In a study conducted on 22 patients, Rochweger et al. [42] were able to demonstrate that after calcific deposit removal by open surgery with concomitant ► **subacromial decompression** the Constant score improved from 52 to 89 points within a 23-month follow-up period. The best results were achieved in patients who had suffered from the disorder for over one year and had exhibited progressive symptoms [42].

Rubenthaler and Wittenberg [43], too, reported about 122 patients who after concomitant open subacromial decompression had Constant score improvement to 100 % within a 4.5-year follow-up period. In this context we should add that even in cases of a spontaneous course of the disorder without any therapy 67 % out of 145 patients no longer showed any sonographic evidence of calcific deposits after 5 years [54].

In another prospective study conducted on 47 patients with type I or II calcific tendinitis according to the classification by Gärtner and Simons [16], 32 % of the calcium deposits had been resorbed either completely or by over 50 % 6 months after symptomatic therapy [45].



**Fig. 8 ▲** Extracorporeal shock wave system (Duolith SD1 from Storz Medical, Kreuzlingen, Switzerland). Handpieces with different diameters and application methods are shown on either side of the touch screen (*right*: handpiece for focused shock waves; *left*: handpiece for radial pressure waves).

**Table 2 Results of arthroscopic removal of calcific deposits in calcific tendinitis<sup>a</sup>**

	Year	Patients treated	Follow-up (months)	Complete/partial removal (%)	Constant score (%)	Good/excellent results (%)
Mole et al. [35]	1993	112	21	91.5	83	89
Jerosch et al. [23]	1998	48	21	77	76	
Seil et al. [46]	2006	58	24	100	91	94
Boyer [5]	2006	275	12	84	-	92
Rebuzzi et al. [40]	2008	22	24	86	-	82
El Shewy et al. [8]	2011	54	84	98	98	-

<sup>a</sup>) All studies found a significant improvement in the average post-operative Constant score, with a high percentage of patients who considered the outcome to be good or even excellent.

### Results of arthroscopic removal of calcific deposits

Ellman [9] was the first to describe arthroscopic removal of calcific deposits with concomitant subacromial decompression in 1987.

In 1991, Ark et al. [2] performed arthroscopic resection of calcific deposits in 22 patients who had undergone unsuccessful conservative therapy for over a year. After a 26-month follow-up period, 11 (50 %) out of the 22 patients reported complete disappearance of complaints and unrestricted range of motion. In 9 subjects (41 %) the range of motion had been fully restored, but they still had occasional residual pain. Two patients (9 %) complained about persisting pain. The time required to achieve complete pain relief varied considerably among patients and depended on the severity of the concomitant bursitis. A total of 91 % of the patients had almost no symptoms for six months after the surgery.

In a study conducted by Jerosch et al. [23], 48 patients had arthroscopic removal of calcific deposits. Concomitant subacromial decompression was only performed when bony impingement was present. Patients who had undergone complete or partial resection of the calcific material achieved far better post-operative clinical results than those exhibiting radiological persistence of the deposit. According to the observations of the authors, subacromial compression as such did not provide any further improvement of the therapy outcome.

These results are confirmed by more recent studies (■ **Table 2**). After arthroscopic removal of calcific deposits in 54 patients, El Shewy et al. [8] reported a Constant score of 98 % after a 7-year follow-up period. A total of 29 patients had complete removal, the remaining 25 subjects had partial removal.

Seil et al. [46] performed arthroscopic management of calcific deposits in the supraspinatus tendon of 58 patients and observed an improvement in the Constant score from 33 % before surgery to 91 % within a 2-year follow-up. Despite these excellent results, only 15 % of the subjects in this study reported immediate post-operative pain relief within the first three months after the procedure. Most patients experienced gradual reduction in residual post-operative pain, which persisted for up to one year. Post-calcification tendinitis plays a key role among such residual pain conditions.

Up to 10 % of the patients complained about persisting post-operative shoulder pain, which is assumed to result from incomplete remodelling of the defect and a persisting inflammatory reaction [38]. The only serious complication that may occur is ► **secondary shoulder stiffness** [7].

Not always is it possible to completely remove the calcific deposit. According to Barchilon and Gazielly [3], residual deposits are found in an average 27 % of patients. Despite such incomplete resection, all calcific deposits are resorbed almost completely during the first year after surgery [46].

The results of arthroscopic removal of calcific deposits are equivalent to those achieved by open surgery [44]. However, the benefits of arthroscopic management are ► **low surgical morbidity** and minimal post-operative pain symptoms. This allows the procedure to be performed on an outpatient basis. Subacromial infiltration of a local anaesthetic can additionally enhance the effects of post-operative pain therapy. Contrary to inpatient treatment, outpatient pain management with catheter techniques, such as the interscalene block, must be viewed sceptically because possible complications such as pneumothorax or diaphragmatic irritation with breathing difficulties may necessitate post-operative hospitalisation [14, 34].

Arthroscopic removal of calcific deposits provides good results.

Most patients experience gradual reduction in residual post-operative pain after arthroscopic removal of calcium deposits.

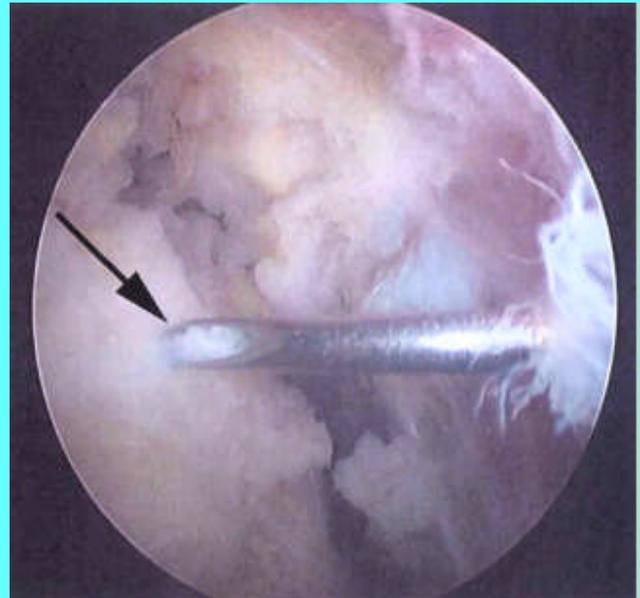
► **Secondary shoulder stiffness**

The results of arthroscopic removal of calcific deposits are equivalent to those achieved by open surgery.

► **Low surgical morbidity**



**Fig. 9** ▲ Patient in beach-chair position with marked landmarks and portals



**Fig. 10** ▲ Localisation of the calcific deposit with a spinal needle in the subacromial space

Surgery can be performed in beach-chair position or in lateral decubitus position.

Localisation of the calcification may be very time consuming.

Intraarticular localisation and marking with PDS thread are not necessary.

In general, the bursa is severely inflamed and thickened.

► **Spinal needle**

### Arthroscopic technique for the removal of calcific deposits

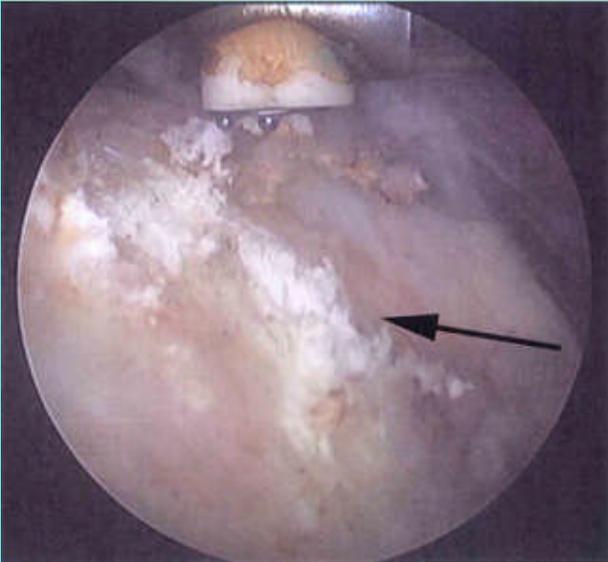
Owing to the reasons outlined above, the arthroscopic technique is our preferred option for the management of calcific deposits. It comprises the following stages: localisation of the calcification, removal of the calcific material and, if necessary, tendon suture. Surgery can be performed in beach-chair position or in lateral decubitus position. After having marked the landmarks (■ Fig. 9), diagnostic arthroscopy of the glenohumeral joint is performed through the standard dorsal portal. Here, close examination of possible (partial) tears of the supraspinatus tendon or lesions of the long biceps is necessary, as these may require treatment, but may be masked by the clinical symptoms of calcific tendinitis.

**Localisation of the calcific deposit.** Localisation of the calcification may be very time-consuming if the deposit is located deep in the tendon and if the surface is not eroded and does not bulge forward. The anterolateral acromial corner can be used as a reference point in the subacromial space. However, surgeons should make sure to memorise or mark the location of the calcific deposit identified by pre-operative X-ray and ultrasound [47]. In many cases, the subacromial bursa may be severely inflamed and thickened and thus have a massively increased tendency to bleed, which makes localisation even more difficult. In the very rare cases in which the calcific deposit cannot be identified, the possibility of using intraoperative X-ray image converter guidance should be considered.

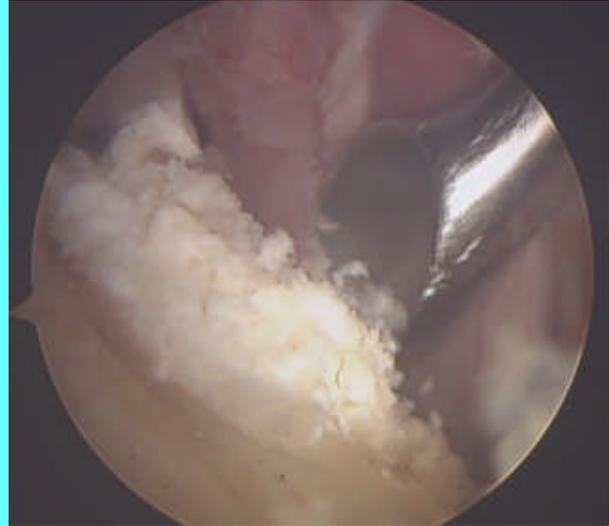
In some cases, the calcific deposit with its pathological vascular pattern can be seen shining through the supraspinatus tendon on the articular side. Owing to vascular hyperproliferation, the lesion resembles the pattern of a strawberry [22].

From our point of view, intraarticular localisation and marking with PDS (polydioxanone) thread, which is then identified in the subacromial space, are not necessary. Moreover, intra-articular puncture of calcific deposits localised at the insertion of the greater tuberosity or medially far below the acromion is very difficult due to the bony structures.

We usually opt for direct subacromial localisation of the calcific deposit. In general, the bursa is found to be severely inflamed and thickened. In order to avoid unnecessary bleeding, which would impair the view of the target area, electrothermal systems should be kept ready for use to ensure better control and management of a bleeding bursa and immediate sclerotherapy, when necessary. After partial bursectomy and visualisation of the tendon, the calcific deposit is approached with a ► **spinal needle**. In general, it is located about 1.5 to 2 cm above the greater tuberosity. The tendon section in which the calcification is assumed to be located is systematically perforated with the spinal needle during inner and outer rotation of the tendon until calcium is found in the bevel end of the needle (■ Fig. 10). In some cases, this calcium can already be seen shining through a circumscribed swelling in the tendon.



**Fig. 11** ▲ After electrothermal resection of the subacromial bursa: the calcific deposits eroding the supraspinatus tendon become visible (*arrow*)



**Fig. 12** ▲ Removal of the calcific deposit with a sharp spoon

If the calcific deposits are large and located close to the body surface, the tendon will be found to be eroded by the calcium and the use of a spinal needle will not be necessary (■ **Fig. 11**). In some cases, the overpressure in the calcific deposits results in extravasation of the calcium with toothpaste consistency (■ **Fig. 2**). Calcifications located deeper in the tendon may be more difficult to localise, especially when the tendon is affected by diffuse calcific deposits.

**Removal.** After the precise location of the calcific deposits has been identified, a small longitudinal incision is made in the tendon in line with its fibres. The objective is to remove as much calcium as possible without causing any unnecessary tendon damage. Therefore, instead of using a shaver, the deposits should rather be removed with a sharp spoon (■ **Fig. 12**). This applies especially to diffuse calcifications in the tendon as the use of a shaver might cause iatrogenic tendon damage.

After the defect has been scraped out meticulously, the calcium must be aspirated with a shaver or a suction cannula in order to avoid any risk of post-operative crystal-induced bursitis [45]. Depending on the primary size of the calcification, complete excision of the diffuse calcium at the margins of the deposit should not be performed in order to prevent any unnecessary increase of the defect. The opening of the deposit and the resulting growing-in of granulation tissue for defect closure trigger an ► **intrinsic resorption mechanism** that breaks down the residual calcium. Here, it should be pointed out once again that the post-operative results of partial removal of calcific deposits are equivalent to those achieved with complete removal because residual calcium is resorbed almost completely within one after surgery [46]. In most cases, it is sufficient to reduce the size of the calcification to diminish the mechanical impingement. In general, the mere opening of the deposit will reduce pain and intratendinous pressure.

**Tendon suture.** Depending on the size of the defect resulting from the procedure, a side-to-side suture may be necessary in very rare cases. This is done to bring together the ends of the defect and prevent it from becoming larger. However, this closure should only be performed when absolutely necessary because contrary to rotator cuff tears, which are primarily caused by a degenerative genesis [21], calcific tendinitis is a ► **self-limiting disorder** with high regeneration potential [22]. Also, increased pressure and tension associated with the suture should be avoided [22]. In a recent study conducted by El Shewy [8], arthroscopic closure of the defect was only performed in 2 out of 54 patients. In these two patients, the thickness of the defect was more than 50 % of the total tendon thickness.

The deposits should be removed with a sharp spoon rather than with a shaver.

► **Intrinsic resorption mechanism**

Post-operative results of partial removal of calcific deposits are equivalent to those achieved with complete removal.

► **Self-limiting disorder**



**Fig. 13** ▲ Y-view X-rays (a, b) and arthroscopic image (c) of right shoulder with bony impingement, a pronounced traction osteophyte, b acromion type III according to Bigliani, c intraoperative surgery site after initial removal of bony spur on the ventral acromion with a shaver

### ► Calcium resorption

As strong pain may be present in the resorptive phase, the focus should be on aggressive pain therapy until the deposits have dissolved.

### Subacromial decompression

Subacromial decompression should only be performed in patients with signs of a bony impingement such as an acromial spur (■ Fig. 13). Other indications are small, multilobular deposits which are difficult to remove completely or calcifications that cannot be localised.

There is some speculation that subacromial decompression may induce ► **calcium resorption** [48]. Studies in which calcific tendinitis was treated by subacromial decompression alone support this assumption because 70 % of the calcific deposits were found to have been resorbed after 5.3 years [19]. However, the possibility of spontaneous healing should not be left out of consideration with such a long follow-up period.

### Contraindications to surgical management

Removal of the calcific deposits is no longer indicated in the resorptive phase when phagocytosis of the calcification occurs and tendon remodelling takes place. As strong pain may be present in this phase, the focus should be on aggressive pain therapy until the deposits have dissolved.

### Conclusions for practice

**Calcific tendinitis is a self-limiting disorder with a high spontaneous healing rate. Depending on the severity of pain suffered by patients, primary non-surgical treatment should be performed over at least 3 to 6 months. Here, modern therapy options such as ESWT are gaining increasing importance, all the more so as their effectiveness is supported by scientific evidence from high-quality controlled studies.**

**Only in 10 % of all patients is calcific tendinitis unresponsive to conservative management. In these cases, arthroscopic removal of the calcific deposits should be performed. Owing to the high self-healing capacity of the tendon, reconstruction of defects is generally not necessary.**

**Additional subacromial decompression is only indicated in patients with bony impingement, diffuse multilobular calcifications or calcific deposits that cannot be localised. Owing to the high rate of spontaneous healing and the different classifications used, treatment results are difficult to compare across studies.**

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**Conflict of interests.** The corresponding author declares that there is no conflict of interests.

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