Modern management of calcifying tendinitis of the shoulder

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KEYWORDS
Calcifying tendinitis; Shoulder; Extracorporeal shock wave therapy; Needle aspiration irrigation; Arthroscopic shoulder surgery

Summary
Calcifying tendinitis of the rotator cuff is a common disorder and the underlying cause is still not fully understood. About 90% of patients can be treated non-operatively but some are resistant to conservative treatment and surgery is indicated. Non-operative treatments include non-steroidal anti-inflammatory drugs, subacromial injection of steroid, physiotherapy, extracorporeal shockwave therapy and needle aspiration irrigation. When conservative treatment fails, arthroscopic excision of calcium, sometimes combined with an acromioplasty and/or rotator cuff repair, reliably produces excellent results with high patient satisfaction. In this article, an up-to-date review of the published papers evaluating each treatment modality is presented.

Introduction
The term 'calcifying tendinitis' was first coined by De Seze and Welfling and is preferred to calcific tendinitis as this reflects better the continually changing nature of the disease. It is a common disorder of the rotator cuff and accounts for approximately 10% of all consultations for painful shoulder. It affects women more often than men; its peak incidence is in the fifth decade. The prevalence among asymptomatic individuals was reported to be 2.7% by Bosworth, who studied 6061 volunteers from an insurance office.

The histopathological findings of calcifying tendinitis have been extensively reported by Uhthoff, who described three distinctive stages through which the disease process progresses. The first stage is the precalcific stage, characterised by metaplasia of the tendinous tissue into fibrocartilage. This is followed by the calcific stage, which consists of a phase of formation and a phase of resorption. In the post-calcific stage, following resorption of the calcium deposit, tendon reconstitution occurs. For a detailed description of the pathological stages of calcifying tendinitis the reader is advised to refer to a previous issue of this journal, where it is well described.

Radiological classification
The radiographic findings of calcifying tendinitis were first described by Painter in 1907. Since then, several authors have proposed various classification systems based on the size of the deposit on radiographs, stage of the disease process and its morphological appearance. As calcifying tendinitis is a multifocal and polymorphic disease, with
different parts of the tendon simultaneously undergoing varying stages of the evolutionary process, these classification systems serve as a useful guide to treatment and ensure that therapy is targeted according to the individual and to the stage of the disease (Figs. 1–3, Tables 1–4).

**Non-operative treatment**

Conservative treatment is usually successful in up to 90% of patients. The main treatment modalities are:

- non-steroidal anti-inflammatory drugs,
- subacromial injection of steroid,
- physiotherapy,
- extracorporeal shockwave therapy,
- needle aspiration and irrigation.

Non steroidal anti-inflammatory drugs are the mainstay of non-operative treatment. Although steroid injections are commonly used in the treatment of calcifying tendinitis, there is still no conclusive evidence that they promote resorption of the calcium deposit. Uthhoff and Sarkar\(^8\) believe that steroids actually impair the cell-mediated resorption of carbonated apatite crystals. Noel et al.\(^9\) found that steroid injections administered before needle aspiration had no effect on the clinical outcome.

The efficacy of physiotherapy in the form of therapeutic ultrasound, in the treatment of calcifying tendinitis, remains uncertain. The Cochrane Musculoskeletal Database Review of twenty six trials found that both ultrasound and pulsed electromagnetic field therapy resulted in significant improvement in pain, compared to placebo, in calcific tendonitis.\(^10\) However, a further meta-analysis of 35 randomised controlled trials, of which 10 were suitable for inclusion, found that only 2 studies supported the use of therapeutic ultrasound over placebo. The remaining 8 showed that therapeutic ultrasound is no more effective than placebo.\(^11\)

**Extracorporeal shock wave therapy**

Extracorporeal shock wave therapy utilises acoustic waves to induce fragmentation of the mechanically hard crystals. Its use as an alternative treatment for calcifying tendinitis...
has gained increasing popularity in the last few years, especially in Europe. The efficacy of extracorporeal shock wave therapy has been confirmed in several prospective studies and favourable results have been reported in terms of patient satisfaction, improvement in functional scores and disappearance of calcific deposit confirmed radio-graphically.\textsuperscript{12–15} A recent single-blind, randomised controlled study of 90 patients with radiographically verified calcific tendinitis found that extracorporeal shock wave therapy led to complete disappearance of calcifications in 86.6\% of the subjects in the treatment group and reduction in size of deposit in 13.4\% of subjects.\textsuperscript{13} In the control group, only 8.8\% of the subjects displayed partially reduced calcifications and none disappeared totally. There was significant reduction in pain and improvement of shoulder function after 4 weeks, with no adverse effects reported.

The optimum energy level for extracorporeal shock wave therapy, for successful treatment of calcifying tendinitis, was evaluated by Peters et al.\textsuperscript{14} who compared extracorporeal shock wave therapy at two different energy levels (0.15\,mJ/mm\textsuperscript{2} and 0.44\,mJ/mm\textsuperscript{2}) with placebo. Those treated with a lower energy level of 0.15\,mJ/mm\textsuperscript{2} had significantly less pain during treatment but required more treatments and had a significantly higher recurrence of calcification at the 6 months follow-up. On the other hand, those treated with a higher energy level of 0.44\,mJ/mm\textsuperscript{2} had no residual calcification or recurrence of pain. It seems that the effectiveness of extracorporeal shock wave therapy is directly related to the energy level. Overall, there were no major side effects with either treatment, except for a small number of haematomas.

Most of the studies demonstrating the effectiveness of extracorporeal shockwave therapy in the treatment of calcific tendinitis have the common limitation of having only a short term follow up. Daecke et al.\textsuperscript{15} carried out a prospective long-term follow-up study and found that 4 years after the shockwave therapy, 20\% of the study population had undergone surgery on the involved shoulder. Thus, it seems that the failure rate following extracorporeal shockwave therapy is higher than previously reported.

**Table 2** De Palma and Kruper classification.\textsuperscript{5}

<table>
<thead>
<tr>
<th>Type</th>
<th>Radiological appearance</th>
<th>Correspondence to Uhthoff’s pathological stages</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Fluffy, fleecy with ill-defined periphery</td>
<td>Resorptive phase</td>
</tr>
<tr>
<td>II</td>
<td>Homogeneous with clearly defined periphery</td>
<td>Formation phase</td>
</tr>
</tbody>
</table>

**Table 3** French Arthroscopic Society classification based on appearance.\textsuperscript{6}

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Homogeneous calcification with well-defined limits (Fig. 1)</td>
</tr>
<tr>
<td>B</td>
<td>Heterogeneous and fragmented calcification with well-defined limits (Fig. 2)</td>
</tr>
<tr>
<td>C</td>
<td>Heterogeneous calcification with poorly defined limits and sometimes with a punctate appearance (Fig. 3)</td>
</tr>
<tr>
<td>D</td>
<td>Dystrophic calcification of the tendon insertion</td>
</tr>
</tbody>
</table>

**Table 4** Patte and Goutallier classification based on morphology.\textsuperscript{7}

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Sharp and dense</td>
</tr>
<tr>
<td>II</td>
<td>Blunt and dense</td>
</tr>
<tr>
<td>III</td>
<td>Sharp and translucent</td>
</tr>
<tr>
<td>IV</td>
<td>Blunt and translucent</td>
</tr>
</tbody>
</table>

Figure 4 Ultrasound image of a needle traversing into the calcium deposit.
Operative treatment

Whilst there is still controversy regarding the optimal operative treatment, most would agree that in patients with severe disabling symptoms which have persisted for more than 6 months and are resistant to conservative treatment, surgery is indicated. The first case of operative removal of calcific deposit was carried out by Harrington and Codman in 1902. Since then, favourable results have been reported by numerous authors with a subjective improvement of 82% and 71% achieving excellent objective results following open excision of the calcium deposit via a deltoïd split approach combined with an acromioplasty. Proponents of the open approach argue that this is technically simpler to perform and the defect within the tendon can also be repaired easily and quickly. With advances in technology, these superior results have also been reproduced arthroscopically.

The procedure involves a glenohumeral arthroscopy with special attention to the ‘critical zone’ of the rotator cuff. A cherry red spot is often visible on the articular side of the rotator cuff close to the footprint and represents an area of increased vascular proliferation (Fig. 5). This is a useful landmark for the location of the calcium deposit and some surgeons recommend marking out this lesion with a suture to aid subsequent identification of the deposit in the subacromial space. Next, a subacromial bursectomy and bursectomy is carried out to adequately visualise the rotator cuff. When an acromioplasty is indicated, the coracocado-mial ligament is released using electrocautery and a subacromial decompression carried out using a burr. The calcific deposit is usually self-evident and is most commonly found in the supraspinatus, 1.5–2 cm from its attachment to the greater tuberosity. In cases where it is difficult to identify the calcium deposit a spinal needle can be used to probe the rotator cuff. Intraoperative fluoroscopy is sometimes helpful in locating the deposit and confirms that the evacuation is complete at the end of the procedure. Once the calcium deposit is identified, the capsule is carefully incised with an arthroscopic knife in line with fibre orientation of the tendon (Fig. 6). To minimise tendon damage a blunt instrument, such as a curette, is then used to milk out the toothpaste-like contents (Fig. 7). When the calcium is of a hard chalky texture, the arthroscopic rotating blade can be used to decompress the deposit, creating a typical snowstorm appearance. At the end of the procedure, a thorough washout of both the glenohumeral joint and subacromial space is necessary to prevent leaving behind any calcium fragments and some surgeons also give an intra-articular steroid injection. Washout is thought to be important to prevent the development of secondary stiffness, which is relatively common following calcium deposit removal and has been reported in 9–15% of cases. It is thought to be caused by residual calcium fragments provoking an inflammatory reaction within the subacromial bursa, triggering the so-called hyperalgesic crisis.

Figure 5 Cherry spot—area of increased vascular proliferation.
How important it is to remove all of the calcium deposit remains uncertain. Jerosch has shown that the functional outcome following surgery is inversely related to the amount of calcification remaining. More recent studies, however, suggest that absolute eradication of the calcium deposit is probably not necessary, as cell-mediated resorption is already initiated by the surgery.

Acromioplasty

The question of whether acromioplasty should be performed or not remains controversial. Some surgeons, such as Neer, strongly believe that calcific tendinitis is independent of rotator cuff impingement. This is supported by Uhthoff’s histological findings that the subacromial bursa in calcifying tendinitis usually has minimal signs of inflammation. Other researchers believe that the vascular invasion and influx of phagocytic cells during the acute resorptive phase lead to oedema of the rotator cuff and rise in the intratendinous pressure. This theoretically can lead to secondary impingement as the thickened and indurated calcified tendon bulges into the subacromial space. However, radiological studies have found little correlation between calcifying tendinitis and osseous subacromial impingement, with only 16% of patients with calcifying tendinitis having the so-called type III or hooked acromion on supraspinatus outlet view radiograph.

Interestingly, Resch et al. reported that patients with diffusely spread small (<5 mm) calcium deposits often had fair or poor functional outcome following deposit removal alone and pain was only relieved later by performing an additional acromioplasty. They therefore advocated that patients with diffusely spread small calcium deposits without evidence of substantial surrounding inflammatory changes should also have an acromioplasty. Some authors even advocate performing an acromioplasty alone without excision of the calcium deposit. The study from Tillander et al. showed that by performing a subacromial decompression alone without interfering with the calcific deposit, 79% of patients had disappearance or decrease in the size of the calcific deposit after a mean period of 2 years from surgery. They question whether the calcific deposits disappear more quickly after an acromioplasty as a result of a reduction in the pressure within the subacromial space.

Thus, summarising the currently available evidence, the most commonly accepted indications for performing an acromioplasty are:

1. Radiological evidence of mechanical impingement e.g. type III acromion, sclerosis of undersurface of acromion and greater tuberosity.
2. Intraoperative evidence of mechanical impingement e.g. kiss lesion—partial bursal sided rotator cuff tear with mirror changes on the undersurface of anterior acromion.
3. Type C calcium deposits with an ill-defined contour and heterogeneous appearance on X-ray. This is because the calcium deposit is diffusely infiltrated and even following surgery, some minute microscopic deposits of calcium will inevitably remain within the tendon.

Repair of the rotator cuff

Traditionally, it was thought that calcifying tendinitis progresses through distinct stages, as described by Uhthoff, and the tendon always reconstitutes after calcium deposit removal. Neer recommended excision of the calcifying tendon as ‘a quarter orange’ without the need for complementary suturing. Recent evidence, however, suggests that spontaneous healing of the tendon does not always occur and the cyclical natural history can be interrupted at any stage of the disease. Seil et al. found that 65% of patients 2 years following surgery had persistent discrete flattening of the tendon on ultrasound. The incidence of persistent rotator cuff defects following surgery has been found to be 25% with 7% having persistent pain. This is more common following removal of large (>2 cm) deposits. Some surgeons therefore recommend a

**Figure 6** Incision of the calcium deposit in line with the tendon fibre orientation.

**Figure 7** Arthroscopic view of the toothpaste like contents from the calcific deposit.
primary side to side repair of the rotator cuff defect if the residual defect following excision is large. 37

Summary and conclusions
Calcifying tendinitis of the rotator cuff is a polymorphic disease characterised by multifocal deposition of calcium in non degenerative tissue. The majority of patients can be treated effectively with non-operative measures such as non steroidal anti-inflammatory drugs, subacromial injection of steroid, physiotherapy, extracorporeal shockwave therapy and needle aspiration irrigation. Approximately 10% are resistant to conservative treatment and surgical removal of the calcium deposit is necessary. In selected patients, a concurrent acromioplasty and rotator cuff repair is also indicated. The key to successful management is to understand the natural history of the condition thereby devising the optimum treatment based on the pathology.

References
