

Calcifying Tendinitis of the Rotator Cuff: Diagnosis, Treatment and Complications

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Abstract

Calcifying tendinitis of the shoulder is a disorder characterised by either single or multiple deposits in the rotator cuff (RC) tendon. Most of calcifications undergo spontaneous resorption while a subpopulation of patients continue to complain for pain and shoulder dysfunction and the deposits do not show any signs of resolution. Although several treatment options have been proposed, the ideal approach is still controversial. In this review we report on the current literature evidence on the calcific tendinitis of the RC, with special interest in the diagnosis, treatment and complications.

Keywords: *Calcifying tendinitis; Shoulder; Diagnosis; Treatment; Complications*

Background

The term calcifying tendinitis (CT) of the shoulder was coined by [1] in 1952 to describe a painful condition characterised by either single or multiple deposits in the rotator cuff tendon (RC) or in the subacromial bursa [2]. Many terminologies have been used for this disease [3], where the authors showed the calcium deposits radiographically; Codman., *et al.* [4] was the first to demonstrate the deposits in the RC tendons. The disease subsides spontaneously in majority of the cases and they can be managed with conservative therapy, nevertheless, a subset of patients complain for a persistent painful shoulder with the deposits not showing any signs of resolution. In this subpopulation the choices of the appropriate treatment remain a dilemma. Recent conservative therapies, like ultrasound guided needling and extracorporeal shock wave have come up adding increased management options. In this review we analyze the current literature evidence on the diagnosis and management of CT of the shoulder and we discuss when to use a conservative approach and when to intervene surgically.

Epidemiology

Incidence of CT range from 2.7% to 20% [2,5,6,7], and in about 10-20% of patients the deposits are bilateral [2,6,8,9]. Woman are more affected compared to men [2,8,9]. The average age in most of the studies was falling between 30-50 years [6,8,9], while the deposits in the elderly are uncommon to find in elderly [6,10,11]. Allocation of deposits is prevalent in the supraspinatus [2,5,1,8], followed by infraspinatus [2,5,8] and rarely by subscapularis and teres minor [2,5]. Most of the patients were sedentary workers or housewives [8,9]. Right shoulder was most commonly affected [8,9,12].

Etiopathogenesis and histopathology

In spite of the disease being described since more than hundred years back the etiopathogenesis of CT is still controversial. Various authors have tried to decode the mystery behind the disease process and speculating how it starts [4]. Hypothesized that the overuse degeneration of RC leads to calcific deposits in the tendon and this was also supported by [13], whereas [14] proposed that the degeneration in the tendon was following local ischaemia which led to calcium deposition. More recently [15] considered the process begins with apoptosis and necrosis of tenocytes but more detailed description was given by [16] who proposed 3 stages of the disease: precalcific,

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calcific and postcalcific. In the precalcific stage there is fibro cartilaginous metaplasia in the tendon, this stage is rarely symptomatic. It is followed by calcific stage which is further divided into formative, resting and reabsorption phases. It is in the reabsorptive phase there is infiltration of the tendon with macrophages leading to tissue oedema and increased pressure within the tendon hence patients are mostly symptomatic in this phase. The postcalcific phase is the healing phase in which there is reabsorption of the deposit and realignment of the tendon fibres [17]. Postulated that incorrect differentiation of stem cells, into osteoblasts or chondrocytes, could be the basis of calcification. Disorders of the thyroid (thyroxine) or estrogen metabolism may be related to the onset of the disease [18], as well as insulin-dependent diabetes [19] so that we may distinguish idiopathic forms and those secondary to endocrine disorders. Biochemical features included glycosylation of extracellular tendon matrix of diabetic patients [20]. Genetics factors have been advocated in the formation of calcific deposits [21] found an increased frequency of human leukocyte antigen serotype class A1 in patients with CT. Relationship of human homologue of the murine progressive ankylosis gene (ANKH) and the tissue nonspecific alkaline phosphatase (TNAP) gene has been shown in cuff tear arthropathy (CTA) [22] and more variant genotypes of these two genes were found in patients with CTA than those in controls. Mutations in ANKH gene has also been reported in patients of hereditary chondrocalcinosis leading to altering the picture of extracellular inorganic pyrophosphate [23]. Oliva, et al. [24] found significantly increased expression of precursors of osteopontin, that was detected in the calcific areas in subjects with calcific tendinopathy; the same authors also found a significant decrease in mRNA expression of bone morphogenetic proteins in the calcific area and they concluded that a variation in expression of these genes may be characteristic of this form of tendinopathy.

Diagnosis

Clinical presentation

The natural history of the disease can be divided in three distinct clinical stages: acute, sub acute and chronic. The main symptom is pain which may or may not be associated with progressive limitation of active range of motion (ROM) [5,25]. Acute pain is associated with the onset of the disease in symptomatic cases [8] and can be fostered by muscle spasm, subacromial bursitis and long head of the biceps (LHB) tendinitis. Episodes of acute pain can also be related to flare-ups of chronic tendinopathy or onset of complications not related to the evolution of the disease such as adhesive capsulitis, RC tears, LHB tendinopathies, greater tuberosities osteolysis (TO) [26,27].

Imaging

Radiology

Anterior-posterior (AP), outlet and axillary views radiographs allow the analysis of the calcifications and assess the texture and morphology of the deposits [28,29] (Figures 1, 2, 3, 4 A-B). Radiographic aspects of CT were first reported by [3] and subsequently many authors have tried to classify the deposits in terms of size [2], and morphology [8,30,31] (Table 1). The numerous classifications proposed imply that none of them perfectly correlates with the radiologic picture and clinical symptoms, also, there is significant inter-observer variability [32]. The location of the deposits is variable [2,28] as reported in the Table 2.



Figure 1: AP view of a large calcium deposit (> 1.5 cm) in the acute stage at the insertion of the supraspinatus tendon, in touch with the greater tuberosity. The calcification is dense and sharply delineated.



Figure 2: AP view of medium size chronic calcification located on lateral aspect of the greater tuberosity. The patient had persistent shoulder pain resistant to conservative therapies. The deposit is heterogeneous and irregularly outlined.



Figure 3: AP view of a large calcification in the acute stage. The deposit is glided on the lateral aspect of the greater tuberosity along the deltoid bursa.



Figure 4 A-B: A) Radiograms of a patient with a history of long pain and shoulder stiffness. The AP view do not show calcium deposit but the humeral head is inferiorly subluxate due to the deltoid hypotonia. B) Axillary view of the same patient showed a small calcium deposit close to the greater tuberosity. This case was diagnosed as “painful stiff shoulder” following chronic calcific tendinitis of the rotator cuff.

Author	Subtype	Description
Bosworth [2]	Small	< 0.5 cm
	Medium	0.5-1.5 cm
	Large	1.5 cm
DePalma, <i>et al.</i> [8]	Type I	Fluffy, amorphous and ill defined
	Type II	Defined and homogeneous
Molè, <i>et al.</i> (French Arthroscopy Association) [30]	Type A	Dense, rounded, sharply delineated
	Type B	Multilobular, radiodense, sharp
	Type C	Radiolucent, heterogeneous, irregular outline
	Type D	Dystrophic calcific deposit
Gartner, <i>et al.</i> [31]	Type I	Well demarcated, dense
	Type II	Soft contour/dense or sharp/transparent
	Type III	Soft contour/translucent and cloudy

Table 1: Radiographic classification of calcifying tendinitis of the shoulder.

Tendon	Percentage
Supraspinatus	51%
Infraspinatus	44.5%
Teres Minor	23.3%
Subscapularis	3%

Table 2: Percentage of rotator cuff tendon involvement in calcifying tendinitis of the shoulder.

Ultrasound

Ultrasound (US) examination is a fundamental tool in the diagnosis and treatment of CT [33,34]. The role of US has changed from diagnostic (Figure 5 A-B) to an important therapeutic tool especially for carrying out bursal lavage and tendon needling. The use of high resolution US shows the presence of deposits and also defines its location in the tendon, size, texture and precisely shows RC tears and also staging of the deposits by the appearance of shadow cones [35,36]. In the resting phase the deposits appear hyperechoic and arc shaped whereas they appear non arc shaped (fragmented/punctate, cystic, nodular) in the resolving phase [35]. These appearances can also be correlated to the symptomatic and asymptomatic phases of the disease [37]. Farin, *et al.* [38] divided the deposits into three types: (1) hyperechoic focus with a well-defined shadow, (2) hyperechoic focus with a faint shadow and (3) hyperechoic focus with no shadow. Doppler examination during the nodular or cystic phase shows increased vascularity around the deposits [39], this correlates with the histo-pathological findings of [40] that showed how during the reabsorption phase the deposits are surrounded by phagocytes and there was concomitant proliferation of vascular channels around the deposits.

Magnetic Resonance Imaging

Magnetic Resonance Imaging (MRI) is additional but not an essential imaging tool because it does not give any additional information in most of the cases [41,42]. Calcific deposits have low signal intensity in all MRI sequences (Figure 6). Although area of increased signal intensity can be found around the deposits in T2 images signifying oedema around the deposits in the restorative phase. This area of increased signal intensity can be misinterpreted as RC lesion [43,44]. Accuracy of MRI in identifying calcific deposits is around 95% but it is more useful in cases of chronic CT which could be associated with RC tears (Figure 6), adhesive capsulitis and TO [26,43,45,46]. All these investigations and accurate clinical examinations are of critical importance especially when CT disease is associated with stiffness, RC tears and TO [47].

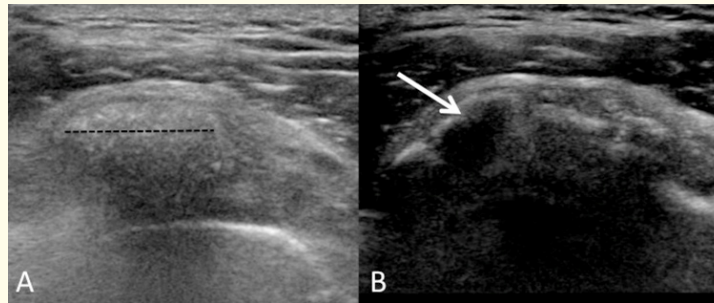


Figure 5 A-B: Ultrasound image of the rotator cuff with the probe on the long axis of the supraspinatus tendon. A) large calcification (> 1.5 mm) above and inside the supraspinatus tendon (hyperchoic areas highlighted by dottedline).
 B) Inflammation of the subacromial bursae and oedema (bursitis) (white arrow) associated with the resorptive phase of the disease.



Figure 6: Coronal fatty suppressed MRI highlights a chronic calcium deposit with associated full thickness supraspinatus tendon tear.

Treatment modalities

Non-surgical measures

Non-operative procedure is the first line of treatment and includes NSAIDs, physical therapy; ultrasound guided needling (UGN) and Extracorporeal Shock Wave Therapy (ESWT) [48]. Described prognostic factors which can address the treatment for a favourable outcome in the early phase of the disease. Non-operative therapy was defined a failure if the symptoms of CT persist after a minimum of 6 months of conservative treatment, including a minimum of 3 months of standardized therapies. They concluded that “negative prognostic factors” were bilateral occurrence of the calcific deposit, localization to the anterior portion of the acromion, medial (sub-acromial) extension, and high volume of the calcific deposit. Prognostic factors that significantly reduced the probability of failure of nonoperative therapy (“positive prognostic factors”) were a Gartner type III calcific deposit and lack of sonographic sound extinction of the calcific deposit. The treatment can be modulated depending upon presence of these prognostic factors. NSAIDs and physiotherapy can be prescribed in the acute phase to relieve the pain and to avoid stiffness of the shoulder. The use of steroid injection in acute phase is controversial as studies have shown it to have positive [40] or no effect [49] or even a negative effect in the form of stopping the re-absorption of the deposits [50,51]. Conservative treatment has been shown to be enough for resolution of symptoms in most of cases, with good to excellent results in 72% to 90% of cases [52,53].

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Ultrasound guided needling

After the early report of UGN under fluoroscopy control [54], Farin., *et al.* [33] described its use for bursal lavage and needling. This procedure is commonly performed in outpatient office, is inexpensive and can be carried out under local anaesthesia (Figure 7 A-B). Gonzales., *et al.* [55] reported satisfactory results after UGN at 3 months in 121 patients with 2 years follow-up. In a randomised trial between UGN with subacromial injection and subacromial injection alone, both the groups showed improvement but the UGN group fared better as compared to injection alone [56]. A recent systemic review of literature for the efficacy of UGN in CT concluded that due to the variation in studies and the low quality of evidence, efficacy of UGN could not be firmly established and additional high quality studies are required [57].

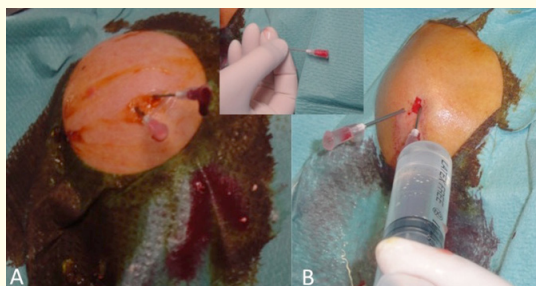


Figure 7 A-B: Ultrasound-guided needling and lavage in acute phase of calcifying tendinitis.

A) Leakage of calcium from the the two needle placed in the subacromial space.

B) The calcium was aspirated with a syringe and an abundant lavage of the subacromial space was performed. The windows up shows the calcium inside the needle.

Extra corporeal shock wave therapy

ESWT has been widely used for medical treatment in the last two decades, but there is lot of disparity in the dosage, duration of the impulses and interval of administration.

Low (below 0.08 mJ/mm²), medium (0.08-0.28 mJ/mm²) and high (0.28-0.60 mJ/mm²) energy shock waves have been defined [58]. The shock waves can be generated through electrohydraulic, electromagnetic, or piezoelectric mechanisms. Farr., *et al.* [59] compared one dose of 0.3 mJ/mm² vs two doses of 0.2 mJ/mm² and found the former dose more effective. Ioppolo., *et al.* [60] found 0.20 mJ/mm² dosage to be more effective than 0.10 mJ/mm². Albert., *et al.* [61] also pitched in favour of high dose therapy though their follow-up was of only 3 months and they didn't find any significant differences in the size of deposits on X-rays. In a randomized trial where the control group was given sham treatment [62] opined that the results were better in ESWT group. Krasny., *et al.* [63] compared ESWT alone and ESWT combined with UGN and found that the combined treatment was more effective in relieving symptoms and lesser patients in the combined treatment group required surgery. Daecke., *et al.* [64] published long term follow-up in patients managed with ESWT tough 20% of overall patients required surgery 70% of patients were treated successfully and no long term complications were seen. Lee., *et al.* [65] did a systematic review to find out midterm effectiveness of ESWT but due to the variability of treatment and reliability of the available studies they were not able to come to conclusion for a particular dosage of treatment. A comparative study between UGN and ESWT by Kim., *et al.* [66] showed better radiological and clinical outcomes in the UGN group tough both the groups showed improvement from the initial findings.

Surgical approach

Surgery is indicated when non-operative treatment fails [67]. Arthroscopy gives similar results as open surgery and is well-tolerated for its less invasiveness [68]. The most debated issues regard the need to repair or leave the defect created, the complete vs incomplete removal of the deposits, removal of deposits vs only acromioplasty (Figure 8 A-B). Some authors [69-71] suggested that

complete removal of the deposits is not essential neither they attempted repair of the defects. Jerosch., *et al.* [72] agreed that repair is not required following removal of the defects but they insisted on complete removal of the deposits. Other researchers [73,74] recommended complete removal of deposits followed by repair of the defect in the tendon using simple side to side sutures or suture anchors depending upon the size of the residual defect. They argued that repair gives similar results without the fear of propagation of the tear and also helps in early rehabilitation of the patients [75] comparing outcomes of acromioplasty in patients which had CT and other had other causes of impingement syndrome, they did not find any significant clinical difference between both groups at 2 years and recommended that the deposits should be left alone. Nevertheless, many authors [69-73] recommended acromioplasty only in cases of visible mechanical impingement during the arthroscopy; it was interesting to note that most of the surgeons [69-71,73] recommended to inform the patients about delayed recovery post-surgery and that suggested to deserve surgical treatment for patients not responding to conservative treatment for at least 6 months.

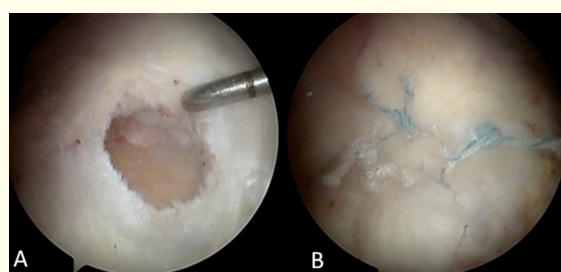


Figure 8 A-B: Arthroscopic findings in a case with chronic calcification of the rotator cuff.
 A) Insertional supraspinatus tendon tear with almost complete spontaneous resorption of the deposit.
 B) Tendon to bone repair with a triple suture anchor at the end of the procedure.

Complications

Various complications associated with CT have been recently described [27]. Pain has been categorised as a complication because the majority of the patients with CT were asymptomatic. Other complications included secondary adhesive capsulitis and RC tears, both of which may occur during the primary disease or post-surgical intervention. Ossifying tendinitis (OT) was described as a very rare finding occurring after surgical removal of the calcium deposits. TO of the greater tuberosity associated with CT of the RC was described by many authors [43,45,46,26,27]. Porcellini., *et al.* [26] suggested that TO should be identified as a different form of CT which is prone for delayed recovery of patients managed conservatively and surgically. During UGN mild vasovagal syncope is reported in 5% of patients [76] also there is reported occurrence of delayed bursitis in patients undergoing UGN [77]. High dose ESWT is associated with pain sometimes requiring local anaesthesia, also local haematoma; erythema and ecchymosis have been reported. Osteonecrosis of the humeral head has also been described [78].

Overview

CT of the RC is a common clinical finding where the treatment depends on the biologic stage of the disease. Although it reabsorbs spontaneously in majority of the cases, a subset of subjects with persistent shoulder pain requires conservative or operative therapy. Some complications recently reported, such as TO, adhesive capsulitis or ossifying tendinitis (very rare), can explain the cases of pain resistant to common conservative therapies. UGN is the preferred therapeutic approach in acute phase but acceptable outcomes have also been described in chronic cases. ESWT can be reasonably used in chronic calcific cases even in combination with UGN. Surgical treatment should be considered when conservative therapies have failed or in cases of US or MRI evidence of RC tears.

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