

R E V I E W

Rotator cuff calcific tendinopathy: from diagnosis to treatment

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Summary. Rotator cuff calcific tendinopathy (RCCT) is a very common condition caused by the presence of calcific deposits in the rotator cuff (RC) or in the subacromial-subdeltoid (SASD) bursa when calcification spreads around the tendons. The pathogenetic mechanism of RCCT is still unclear. It seems to be related to cell-mediated disease in which metaplastic transformation of tenocytes into chondrocytes induces calcification inside the tendon of the RC. RCCT is a frequent finding in the RC that may cause significant shoulder pain and disability. It can be easily diagnosed with imaging studies as conventional radiography (CR) or ultrasound (US). Conservative management of RCCT usually involves rest, physical therapy, and oral NSAIDs administration. Imaging-guided treatments are currently considered minimally-invasive, yet effective methods to treat RCCT with about 80% success rate. Surgery remains the most invasive treatment option in chronic cases that fail to improve with other less invasive approaches. (www.actabiomedica.it)

Key words: calcific tendinopathy, rotator cuff, US, MRI, percutaneous treatments

Introduction

Rotator cuff calcific tendinopathy (RCCT) is a very common condition caused by the presence of calcific deposits in the rotator cuff (RC) or in the subacromial-subdeltoid (SASD) bursa when calcification spreads around the tendons (1-5). This condition has been reported in 2.5%-7.5% of healthy shoulders in adults (6-10), occurring in women in about 70% of cases, especially during the 4th and 5th decades of life (11-15) and seems not to be correlated to physical activity (16-20). In about 10-20% of patients, calcific deposits are bilateral.

In the RC, the most affected area is the critical zone of the supraspinatus tendon (80%), followed by the lower side of the infraspinatus (15%), and the preinsertional area of the subscapularis tendon (5% of cases) (5, 21-25). However, other sites in the body may be affected, being the extrarotator tendons of the hip the most frequently involved.

Deposits may be asymptomatic in 20% of cases or determine low-grade pain, which may be associated with acute or gradual restriction of range of movements (26-30). Classically, this condition tends to have spontaneous resolution and it is uncommon to

see associated degenerative tendon changes (31-35). If symptomatic, RCCT usually responds to relative rest, oral nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids injection within the SASD bursa, and physical therapy (36-42). However, RCCT may represent a highly disabling condition in some cases, with pain resistant to high doses of NSAIDs, usually increasing at night (43-47).

No standard of care has been established for RCCT and its management includes surgery (48), shock wave therapy (27), and image-guided procedures (43, 49-52).

Pathophysiology

The pathogenetic mechanism of RCCT is still unclear. It seems to be related to cell-mediated disease in which metaplastic transformation of tenocytes into chondrocytes induces calcification inside the tendon of the RC (1). Burkhead (53) and Gohlke (54) postulated a pathogenesis based on a degenerative process involving tendon fibers with necrotic changes that progress into dystrophic calcification. However, this theory seems not to be compatible with the *restitutio ad integrum* that happens when RCCT spontaneously resolves (55). Rui et al suggested that calcification process could be the result of erroneous differentiation of tendon stem cells into bone cells (56); this theory was supported by Hashimoto et al who showed that injection of recombinant human bone morphogenetic protein-2 into the rabbit's tendon induces ectopic ossicle formation (57).

Other authors correlated RCCT with a decrease of intratendinous oxygen concentration that can promote tendon fibrocartilaginous metaplasia and cellular necrosis, ultimately followed by calcium deposition (16, 58-63).

At any rate, pathogenesis of RCCT can be divided into three stages, as reported by Uthoff et al. (16):

1: *precalcific stage*, with tendon transformation in fibrocartilaginous tissue which acts as a substrate for calcium deposition.

2: *calcific stage*, with real calcium deposition. It is composed of the formative and resorptive phase. The first is characterized by calcium crystals deposition into the tendon, which is mediated by the chondrocytes of

the fibrocartilaginous metaplasia. The resorptive phase begins after a variable time period of silent course of disease in which vascular weaving develops in the affected area with subsequent macrophages phagocytosis of the calcium deposit (36). This phase is characterized by edema and increased intratendinous pressure with possible extravasation of calcium crystals in the SASD bursa. The resorptive phase is usually associated with the development of acute pain that can be highly disabling and unresponsive to common analgesics.

3: *Postcalcific stage*, with tendon tissue remodelling by fibroblasts after calcium deposition, which can last several months. The postcalcific stage and the resorptive phase of the calcific stage seem to occur simultaneously, with the replacement of calcium deposits by granulation tissue. This process usually ends with complete healing of the involved tendon.

Imaging

Conventional radiography (CR)

CR is widely used in the musculoskeletal system to evaluate a number of different conditions (64-68). When dealing with RCCT, CR may detect the presence of calcifications in the soft tissues around the humerus and in the subacromial space, thus confirming the clinical suspect. CR can also detect the presence of a calcification in patients who perform the examinations for other reasons. Standard radiographs used for diagnosis and follow-up of RCCT are the antero-posterior (AP), outlet, and axillary views, as they provide useful information regarding location and morphology of the calcium deposits (69-71).

Several authors tried to classify the calcium deposits on the basis of the size, morphology, or radiographic appearance. Mole et al classified the deposits in four classes (72): (i) type A, sharply defined, homogenous and dense calcification; (ii) type B, sharply defined, dense in appearance, with multiple fragments; (iii) type C, heterogeneous calcification in appearance, with a dawning deposit; (iv) type D, dystrophic calcification in the tendon insertion. The type C and D are associated with the resorptive phase and calcific deposits are barely visible on radiographs in this phase (73). Gartner

and Heyer (21) classified RCCT into: (i) type I, well circumscribed, dense calcification, formative phase; (ii) type II: soft contour/dense or sharp/transparent; and type III; translucent and cloudy appearance without clear circumscription, resorptive phase (Fig. 1).

Ultrasound (US)

US is a well-accepted modality for the evaluation of soft tissue structures throughout the body (21, 34, 38, 74-93). On US, the calcium deposits usually appear hyperechoic with or without acoustic posterior shadowing. Bianchi and Martinoli (94) described three different calcifications types, based on their different percentage of calcium. In type I, calcifications appear as hyperechoic foci with a well-defined acoustic shadowing due to their substantial quantitative of calcium. In type II, calcification appears as hyperechoic foci with a mild acoustic shadow due to the reduced amount of calcium; in type III, calcification appears almost isoechoic with the tendon, without an acoustic shadow and often they are difficult to diagnose. Type I correspond to formative phase while type II and III calcifications correspond to the resorptive phase. Sconfienza et al. (95, 96) proposed a different classification of RCCT at ultrasound: (i) hard calcifications, hyperechoic with strong acoustic shadowing; (ii) soft

calcifications, hyperechoic, almost isoechoic with the tendon, without acoustic shadowing; and (iii) fluid calcifications, hypo/anechoic, without acoustic shadowing (Fig. 2). This classification is mainly related to the

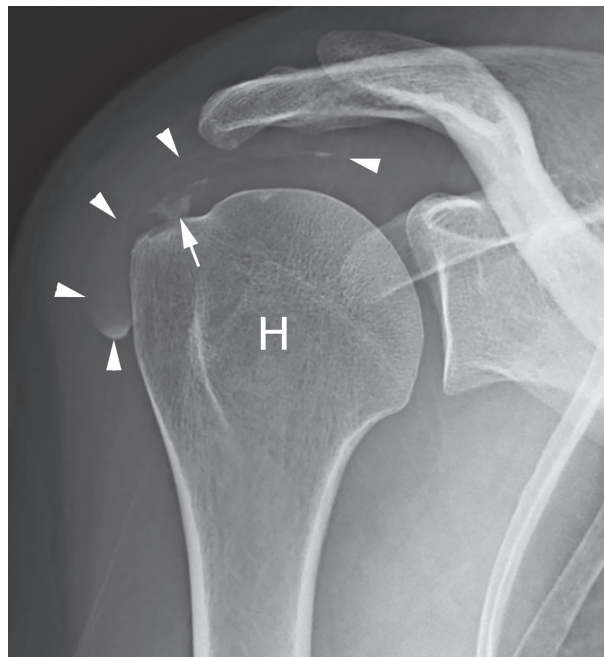


Figure 2. Calcific tendinopathy of the supraspinatus (arrow). In this case, most of the calcification has drained into the subacromial-subdeltoid bursa (arrowheads), which can be easily seen on conventional radiograph. H=humeral

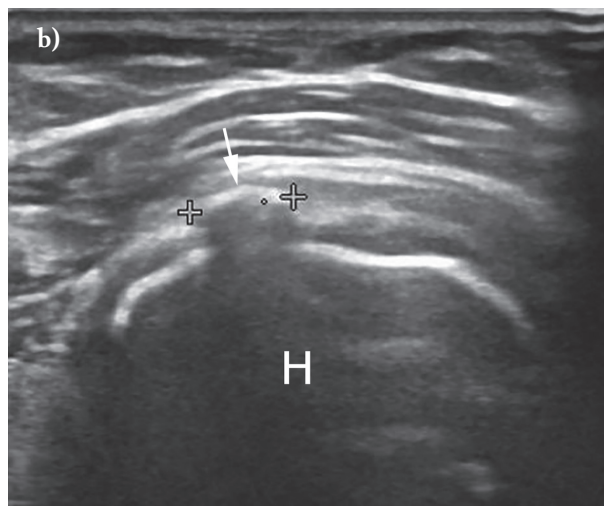
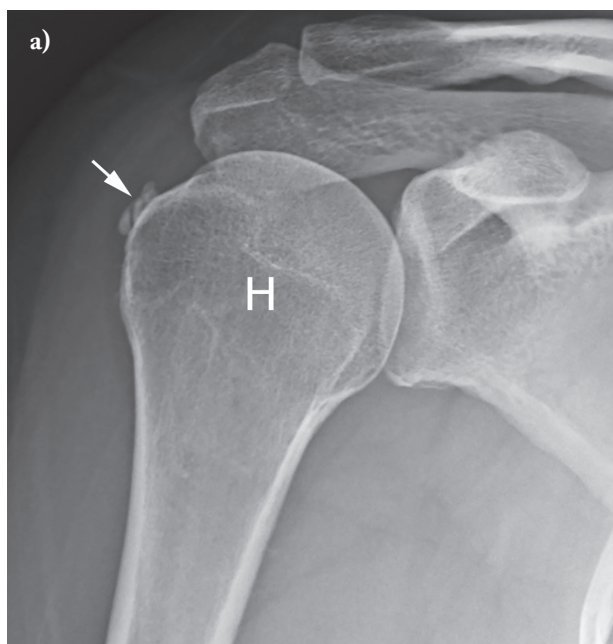


Figure 1. Calcific tendinopathy (arrow) of the supraspinatus tendon evaluated using (a) conventional radiography and (b) ultrasound in the same patient. The calcification has a maximum diameter of 7 mm. H=humeral

three different types of RCCT commonly encountered in patients undergoing interventional procedures (see below). Chiou et al described the potential advantage of color doppler in the identification of the formative and resorptive phase of calcifications with a strong correlation between color Doppler findings and clinical symptoms (97).

Magnetic Resonance Imaging (MRI)

MRI is a well-established imaging technique (98) in the musculoskeletal system and particularly in the shoulder (18, 30, 99-101). However, the low amount of resonating protons contained in calcific deposits usually leads to poor visibility of the calcification itself (80). Conversely, when the calcification is particularly oedematous, signal changes may occur, mimicking the presence of a tendon tear.

In 2016, Nörenberg et al (102) evaluated the diagnostic performance of susceptibility-weighted imaging (SWI) for the detection of shoulder calcific deposits in comparison to that of conventional radiography. SWI showed sensitivity of 98% and specificity of 96% for the identification of calcifications when compared with radiography, thus leading to better diagnostic performance than standard shoulder MRI protocols.

MRI may be used to evaluate intraosseous migration of calcification in some cases of RCCT (102). Porcellini et al suggested that calcium deposits in contact with the tuberosities consistently produce cortical lesions which determine less improvement after therapeutic treatment (103). This is also confirmed by Klontzas et al (104) who recently reported that patients with intraosseous extension have a less satisfactory outcome compared to patients with uncomplicated RCCT.

Treatment

Conservative treatment

Conservative management of RCCT usually involves rest, physical therapy, and oral NSAIDs administration. Matsen et al supposed that physiotherapy with range of motion exercises could be able to avoid

gleno-humeral stiffness and frozen shoulder, but there is no evidence that RCCT is linked with gleno-humeral capsular impairment (105). Ogon et al defined the failure of conservative treatment as the persistence of clinical symptoms for at least 6 months, including 3 months of standardized treatment (106).

Various injections with different techniques have been used in the management of RCCT, using or not US guidance. An intra-SASD injection of corticosteroids may be used to relieve patient's symptoms due to subacromial impingement and bursitis (107). However, it has been demonstrated that US-guided percutaneous aspiration of calcific tendinopathy (US-PICT) is superior to SASD bursa injections in this setting (108).

Extra-corporeal shock wave therapy (ESWT)

ESWT is an option for the management of RCCT, with reported alternate results. This method is based on the application of repetitive pulses over the affected shoulder. The exact underlying mechanism of the therapeutic effect of ESWT on RCCT is still debated. Regarding the direct mechanical effect, ESWT induce calcium deposit fragmentation due to the increasing pressure inside the deposit itself, while regarding its molecular effect, it seems to be related to the phagocytosis of calcium deposit induced by neovascularization inflammatory response and leukocyte chemotaxis (109). There is evidence in literature that ESWT in association with needling procedure could lead to higher frequency of therapeutic success compared with ESWT alone (110). At any rate, the use of ESWT in patients with acute pain given by RCCT in resorption seems to be suboptimal.

US-PICT and needling

US is widely used as guidance for musculoskeletal procedures (111-117) especially in the shoulder (118), allowing to avoid radiation exposure for both radiologist and patients (83, 119, 120). US-PICT is currently accepted as a first-line, safe, and effective treatment for RCCT (121, 122) with significant pain relief and a low incidence of minor complications such as vasovagal reaction and bursitis (123).

Different approaches have been reported in recent studies and all include the use of a fluid (local anaesthetic or saline solution) to dissolve calcium deposits; one needle (124-126) or two needles (49) are used to inject and retrieve the fluid to dissolve calcium deposits. Recent evidence showed that a double-needle approach might be more appropriate to treat harder deposits, while one needle may be more useful in treating fluid calcifications (96).

US-P ICT is always indicated in the acute phase of the pathology, in presence of soft or fluid calcifications. In cases of hard calcifications in mildly symptomatic patients, elective treatments should be considered (43). Percutaneous treatment is not indicated when patients are asymptomatic, calcification is very small (≤ 5 mm), or it has migrated into the bursal space (127). There is evidence that patients with intraosseous migration of calcification experience worse results (104).

Some advantages of US-P ICT are that the procedure does not require any hospitalization, is performed under local anesthesia, the patient can return home about 30 minutes after the procedure is completed, there is no need of post-procedural immobilization, and the patient can return to work the day after the procedure.

In brief, US-P ICT is performed with patient supine after sterile preparation of the skin and US probe, and the calcification is visualized along its major axis; a small amount of local anaesthetic (up to 10 ml of lidocaine) is injected in the SASD bursa and near the

calcification. If two needles are used, the first is inserted into the lowest portion of the calcification with needle bevel open towards the probe, while the second needle is inserted into the calcification parallel and superficial to the first one, with its bevel opposite to the first needle in order to create a correct washing circuit (128, 129). Applying an intermittent pressure, the calcification is progressively filled with saline solution to dissolve its core and allowing the washing fluid to get out of the second needle, until a complete internal emptying is visualized. If the calcification is treated with one needle only, washing procedure is performed by pushing the syringe plunger to hydrate the deposit and calcium refluxes back together with saline solution or anaesthetic within the same syringe (55).

It is preferable to use warm saline solution to reduce procedure duration and improve calcium deposit dissolution, particularly in cases of hard calcifications (95). There is also evidence that the use of warm saline could be also associated with a reduction in postprocedural bursitis incidence (95). The final phase of the procedure involves injection of low-solubility steroid into the SASD bursa; color Doppler may be a useful tool to ensure the injected fluids in the correct site (117).

An alternative procedure is to perform dry needling of the calcification. It consists of a series of punctures performed under US guidance on the degenerated area of the tendon to fragment the deposit, induce local bleeding, and promote calcium deposit resorption (117) (Fig. 3).

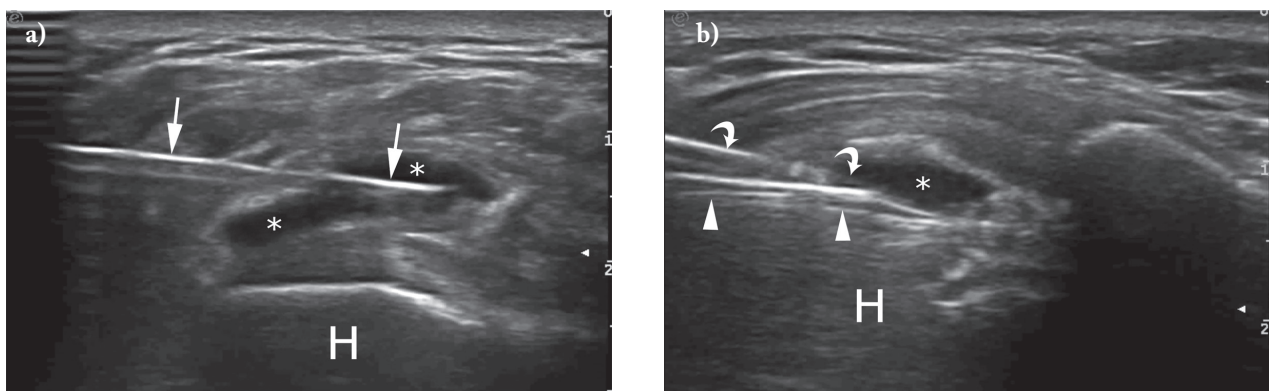


Figure 3. End of ultrasound-guided percutaneous irrigation of calcific tendinopathy procedure performed with (a) one needle (arrow) and (b) two needles (superficial needle, curved arrows; deeper needle, arrowheads). The calcification is totally empty (asterisks) in both cases. H=humeral

Surgery

Arthroscopy is another option to treat RCCT. Currently, it is regarded as the last option in chronic cases in which conservative or less invasive approaches have failed. Calcium deposits under arthroscopic evaluation were identified as a bulge within tendon structure, also known as the “calcific bulging sign” (130). Calcification removal techniques vary regarding the type of tendon incision and the instrumentation used to remove the calcium deposit (131).

One of the great advantages of surgery is that, while removing the calcification, the surgeon may also perform other procedures, such as subacromial decompression and thorough cleaning of the joint (132). However, surgery requires hospitalization, general anesthesia or sedation, and quite a long rehabilitation period after treatment.

Conclusion

RCCT is a frequent finding in the RC that may cause significant shoulder pain and disability. It can be easily diagnosed with imaging studies as CR or US. Although resorption of deposits occurs spontaneously in the majority of cases, some patients show persistent clinical symptoms requiring medical attention. Most patients respond favourably to conservative treatments, but if symptoms persist, other therapeutic options such as US-PICT or ESWT may be considered. Surgery remains the most invasive treatment option in chronic cases that fail to improve with other less invasive approaches.

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