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REVIEW ARTICLE

# Rotator cuff calcific tendinopathy: an educational review on diagnosis and treatment

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**Abstract**
Rotator cuff calcific tendinopathy (RCCT) is a common condition characterized by the pathological deposition of calcium hydroxyapatite crystals over fibrocartilaginous metaplasia of tenocytes, most commonly affecting the supraspinatus tendon. RCCT is one of the most frequent causes of chronic shoulder painful and is more common in patients aged between 30 and 60 years, particularly premenopausal women, and seems not to be correlated to physical activity. Although the pathogenetic mechanism of RCCT is still unclear, it is probably a multifactorial disease with multiple stages: pre-calcific, calcific (including formative and resorptive phases) and post-calcific; pain occurs during the resorptive phase. It is easily identified using conventional radiography or ultrasound. RCCT is a self-limiting condition that may be asymptomatic, thus non requiring any treatment. When painful, the initial management strategy consists of conservative treatment, including rest, oral non-steroidal anti-inflammatory drugs, and physical therapy. If symptoms persist, non-operative treatment may be considered, such as ultrasound-guided percutaneous irrigation, corticosteroid injection into the subacromial-subdeltoid bursa, extracorporeal shockwave therapy, and platelet-rich plasma injection. Surgery is reserved for chronic cases that are refractory to these less invasive therapeutic modalities.

**Keywords** Calcific tendinopathy · Rotator cuff · Ultrasound · Conventional radiography · Magnetic resonance · Percutaneous treatments

## Introduction

Calcific tendinopathy of the rotator cuff (RCCT) is a common condition, with prevalence estimates ranging from 2.7% to 22%, most frequently affecting individuals between 30 and 60 years of age, especially in women before menopause [1–5]. RCCT is often clinically silent and has been observed in 2.5%-7.5% of asymptomatic adult shoulders [6, 7]. However, in patients with chronic shoulder pain, RCCT is detected in 10–42% of cases [4]. Beyond pain, rotator cuff calcific tendinopathy commonly presents with night pain, acute or subacute symptom onset, and reduced active and passive range of motion, especially during abduction and forward flexion, often resulting in difficulty with overhead activities [8]. When one shoulder is affected, the contralateral shoulder is involved in approximately 14% of cases [9]. The supraspinatus tendon is the most commonly involved (approximately 80%), followed by the infraspinatus (15%) and the subscapularis (5%). Less frequently, involvement has been reported in atypical sites such as the teres minor, deltoid, and the long head of the biceps tendon (LHBT) [10–15]. Sedentary lifestyles increase the risk of developing RCCT [6] and it is associated with several medical conditions, including ischemic heart disease, hypertension, diabetes, and thyroid disorders, although the underlying mechanisms remain unclear [16]. There is a recognized

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correlation between RCCT and endocrine disorders. Patients with these comorbidities tend to experience symptom onset at an earlier age [17]. Interestingly, RCCT is rarely linked to calcium or phosphorus metabolism disorders [9].

## Pathophysiology

RCCT is characterized by the abnormal deposition of calcium hydroxyapatite crystals within the tendon matrix. The pathogenesis of RCCT remains incompletely understood, with multiple overlapping and possibly interrelated mechanisms proposed. One widely discussed theory suggests a cell-mediated process, in which metaplastic transformation of tenocytes into chondrocytes leads to intramural tendon calcification [5]. Rui et al. suggested that RCCT pathogenesis may involve chondral metaplasia, wherein the ectopic deposition of calcium is driven by the aberrant differentiation of tendon-derived stem cells into osteogenic lineages [18]. Supporting this theory, Hashimoto et al. demonstrated that BMP-2 injection into tendons induces ectopic bone formation, indicating that tendon stem cells are responsive to proteins thought to induce bone growth [19].

A degenerative theory, proposed by Burkhead [20] and Gohlke [21] attributes RCCT to progressive tendon fiber degeneration and necrosis due to aging and compromised vascularity, resulting in dystrophic calcification. This model, however, is challenged by clinical observations of *restitutio ad integrum* in spontaneously resolving cases, which is inconsistent with irreversible degenerative damage.

Another proposed mechanism is endochondral ossification, supported by Benjamin et al.'s findings in rat Achilles tendons. This process involves fibrocartilaginous metaplasia followed by vascular invasion and mineralization, leading to bone-like formation within the tendon, resembling a bone spur. Notably, this process occurred in the absence of any observable inflammatory response. However, Benjamin et al. did not describe the resorption phase or subsequent histological changes in the rat tendons [2, 22].

The reactive calcification model, as described by Uhthoff et al. [23], views RCCT as a dynamic, multi-phase process. It begins with a precalcific stage, in which tenocytes undergo metaplasia into fibrocartilaginous tissue, followed by the calcific stage, subdivided into formative (deposition of calcium crystals mediated by chondrocytes) and resorptive phases (following a variable asymptomatic phase, vascular ingrowth occurs within the affected area, facilitating macrophage-mediated phagocytosis and resorption of the calcium deposits) [24]. This phase is marked by edema and increased intratendinous pressure, clinically associated with acute, severe pain that may be refractory to standard analgesic treatment. The final post-calcific phase is characterized by tendon remodeling, mediated by fibroblast proliferation and granulation tissue formation. This phase commonly results in full recovery and structural normalization of the affected tendon tissue.

## Imaging

### Conventional radiography (CR)

CR usually represents the first imaging modality, particularly in patients presenting with shoulder pain, and is often the initial diagnostic tool employed when RCCT is suspected, especially in emergency settings [25, 26]. It allows for the detection of calcific deposits in the soft tissues around the humeral head and within the subacromial space, even in asymptomatic patients undergoing imaging for unrelated reasons [7]. It also allows excluding other conditions which may mimic the presence of a calcification.

Standard CR views for RCCT assessment include the anteroposterior (AP), outlet, and axillary projections. These provide information regarding location, morphology, and extent of the calcium deposits [27, 28]. Typically, RCCT-related calcifications appear as homogeneous, amorphous opacities with smooth or ill-defined margins without trabeculation [29] (Fig. 1).

Several radiographic classification systems have been proposed to stratify calcium deposits based on their size, shape, and radiographic appearance. One of the most commonly used is the Gartner and Heyer classification [11], which correlates radiologic findings with histopathologic stages of the disease:

- Type I: well-circumscribed, dense calcifications indicating the formative phase;
- Type II: calcifications with soft contours or a transparent/fragmented appearance;
- Type III: translucent, cloud-like deposits without clear borders, typically observed in the resorptive phase.

Deposits in types I and II are generally well visualized, while those in type III may be barely detectable due to their low density.

Mole et al. [30] further expanded radiographic classification into four types:

- Type A: sharply defined, homogenous and dense;
- Type B: sharply defined, dense in appearance and fragmented;
- Type C: heterogeneous with a dawny deposit;
- Type D: dystrophic calcifications at the tendon insertion.

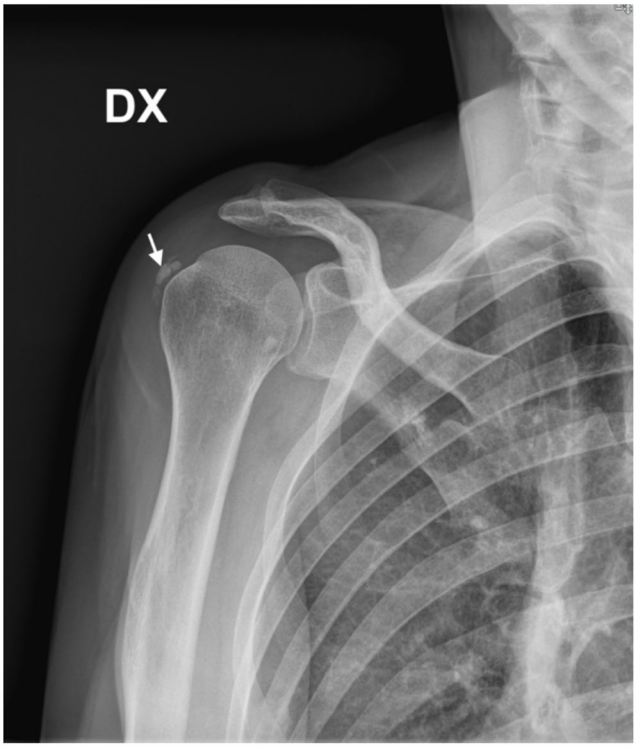


**Fig. 1** A 42-year-old female presents with a 2-day history of progressive right shoulder pain and functional limitation, without prior trauma. Partial response to NSAIDs. Imaging shows a large calcification over the humeral head (arrow)

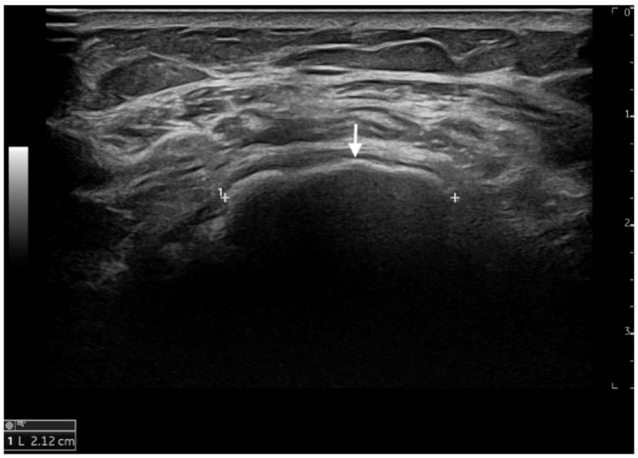


**Fig. 2** A 55-year-old female patient with intense left shoulder pain resistant to oral anti-inflammatory drugs and no history of trauma. The US shows a hard calcification of approximately 2 cm in the supraspinatus tendon, with hyperechoic rim and strong posterior acoustic shadow (arrow)

Notably, types C and D are typically associated with the resorptive phase, during which radiographic visibility of calcifications significantly diminishes.

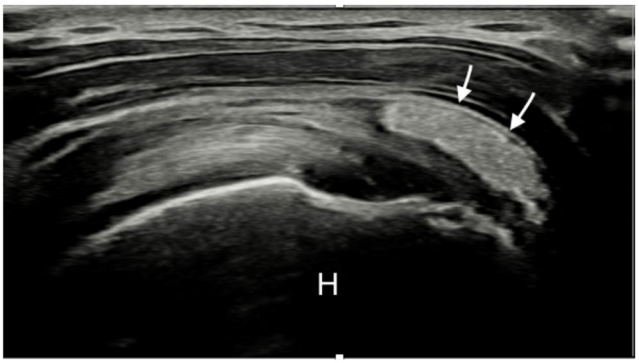


**Fig. 3** A 47-year-old male patient with atraumatic shoulder pain due to RCCT, showing bursal migration of a calcification (arrows) with a maximum diameter of 15 mm. H=humerus

## Ultrasound (US)

US is a well-established imaging modality for the evaluation of soft tissue structures and has proven to be highly effective in the diagnosis of RCCT, demonstrating a sensitivity of 98% and specificity of 94% [31]. It allows for accurate detection and localization of calcific deposits within rotator cuff tendons. On US, calcific deposits predominantly present as hyperechoic foci with well-demarcated posterior acoustic shadowing; however, variations in disease stage and calcium content may cause these deposits to appear as hyperechoic clusters with reduced or absent acoustic shadowing [32]. Multiple US classification systems have been developed based on the morphology and echogenicity of the calcifications. Bianchi and Martinoli [32] described three types based on calcium content:

- Type I: hyperechoic foci with strong posterior shadowing, consistent with the formative phase;
- Type II: hyperechoic with mild shadowing due to lower calcium content;
- Type III: nearly isoechoic to the tendon and lacking acoustic shadowing;

Type II and III correspond to the resorptive phase and may be more difficult to identify.

A complementary classification by Sconfienza et al. [33], tailored to interventional US procedures, categorizes RCCT into:

- Hard calcifications: characterized by a hyperechoic rim and strong posterior acoustic shadow (Fig. 2);
- Soft calcifications: appearing as homogeneously hyperechoic or isoechoic to tendon tissue without shadowing (Fig. 3);
- Fluid calcifications: showing a thin hyperechoic rim with a hypoechoic or anechoic core.

These sonographic patterns can evolve over time, especially during the resorptive phase, where calcifications may display irregular contours, focal discontinuities (Fig. 4), and progressive fragmentation (Figs. 5 and 6).

Chiou et al. highlighted the potential utility of color Doppler ultrasound in differentiating between the formative and resorptive phases of calcific deposits, demonstrating a strong correlation between Doppler signal patterns and the patient's symptoms [34].

## Magnetic resonance imaging (MRI)

MRI is a well-established modality for the evaluation of shoulder pathology. However, its role in the diagnosis of RCCT is limited due to the intrinsic imaging characteristics of calcium; the low number of resonating protons within calcific deposits results in poor visibility of the calcifications themselves [35] (Fig. 7). Non-acute calcific deposits appear as areas of signal void on all pulse sequences, which can be difficult to distinguish from the naturally hypointense tendon tissue. Despite this limitation, MRI may still contribute to the diagnosis by revealing secondary signs such as surrounding tendon edema, heterogeneity, and associated inflammatory changes, especially in the acute phase, which may help localize the calcific focus.

Recent studies explored advanced MRI techniques to improve diagnostic accuracy. Nörenberg et al. [36] assessed the performance of susceptibility-weighted imaging (SWI) in detecting calcific deposits and found it to be highly reliable, with a sensitivity of 98% and specificity of 96% compared to conventional radiography. However, they noted a tendency for SWI to slightly overestimate the size of calcifications. Conversely, standard MRI sequences demonstrated significantly lower diagnostic accuracy, with reported sensitivities and specificities of approximately 65% and 58%, respectively [36–39].

Hwang et al. [40] demonstrated that zero echo time (ZTE) sequence had the highest detection rate for calcific deposits (100%) compared to conventional sequences such as fat-suppressed proton density-weighted imaging (84.7%) and T2-weighted imaging (55.9–66.1%). ZTE also showed superior sensitivity (100%), with comparable specificity (91.7%) and high positive (93.5%) and negative predictive values (100%), outperforming conventional sequences in overall diagnostic performance. ZTE sequence also improves the detection of calcific deposits and differentiation between resorptive and formative phases; in the resorptive phase, a greater percentage of calcific deposits showed irregular shapes, ill-defined margin, heterogeneous compositions on ZTE images (Fig. 8). At any rate, ZTE sequences are generally not part of routine standard protocol.

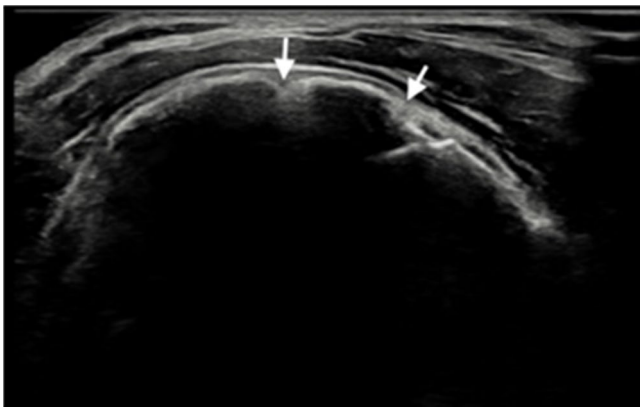

**Fig. 4** Shoulder US of a 56-year-old female with calcific tendinopathy of the supraspinatus tendon shows a large 30 mm calcification in the resorptive phase, presenting irregular profiles and focal breaks (arrows)

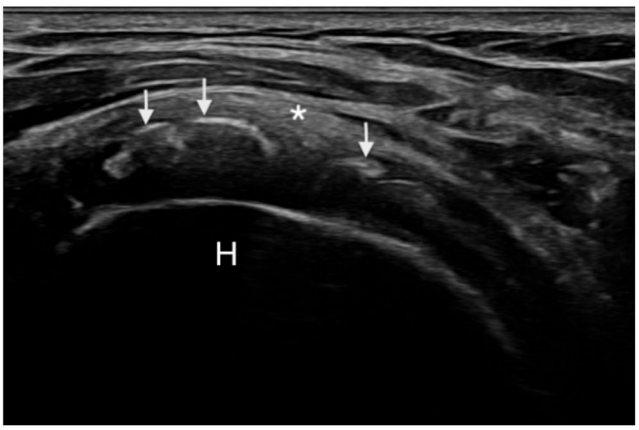


**Fig. 5** Shoulder US of a 45-year-old female patient with recent onset of acute right shoulder pain unresponsive to analgesics. The US shows a large calcification of supraspinatus tendon in the resorptive phase, presenting irregular margins and fragmented hyperechoic calcification(arrows), associated with subacromial bursitis (asterisk). H=humerus

Zubler et al. [39] evaluated MR arthrography and concluded that it is unreliable for detecting RCCT, as small calcium deposits may be missed, and hypointense areas within the tendon may be misinterpreted as calcifications, leading to both false-negative and false-positive results. Accordingly, it is recommended that MRI findings be interpreted in conjunction with radiographs to avoid diagnostic errors.

In addition to identifying soft tissue involvement, MRI can be useful in evaluating complications of RCCT, such as intraosseous migration of calcium [41]. In such cases, bone marrow edema and cortical erosions may be evident. Porcellini et al. [42] and Klontzas et al. [43] reported that intraosseous extension of calcium is associated with more significant structural damage and poorer clinical outcomes following treatment.

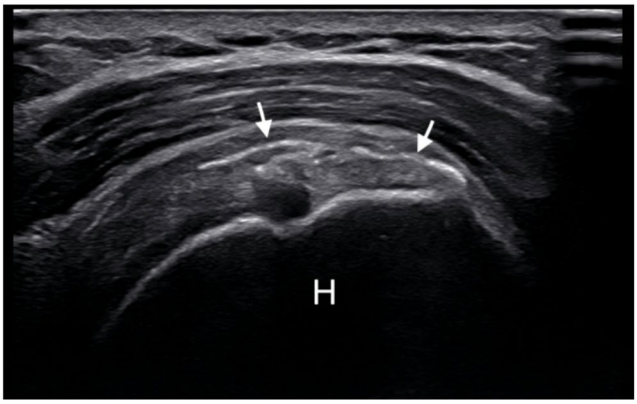


**Fig. 6** A 64-year-old male patient with calcific tendinopathy of the supraspinatus tendon, showing a markedly fragmented calcification (arrow), partially migrated into the subacromial-subdeltoid bursa. The calcification is in the resorptive phase, showing no distal acoustic shadowing. Associated with reactive thickening of the subacromial-subdeltoid bursa due to chronic bursitis(asterisk).H=humerus

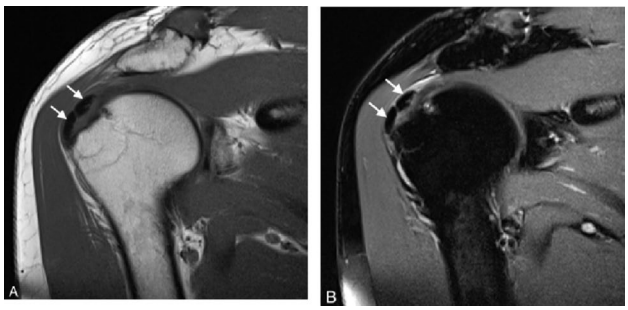


**Fig. 7** Right shoulder MR of a 45-year-old male patient with painful RCCT, images show a calcification appearing as focal area of low signal on all sequences (arrows) within the supraspinatus tendon. **A** Coronal T1-weighted. **B** Fat-suppressed protondensity weighted

While MRI is not optimal for directly assessing the size or density of calcific deposits, it remains valuable for determining the acuity of the condition, detecting associated rotator cuff tears, bursitis, and bone involvement.

## Computed tomography (CT)

Computed tomography is not routinely indicated in rotator cuff calcific tendinopathy, as conventional radiography and ultrasound are usually sufficient for diagnosis and procedural planning; CT may be useful only in selected atypical cases, particularly when unusual localization, osseous involvement, or calcific migration require further anatomical definition [44].

## Treatment

Although RCCT is a common cause of shoulder pain, it often presents as a self-limiting condition. Thus, optimal treatment should be rapidly acting and minimally invasive. Conservative management typically includes rest, physical therapy, and oral administration of nonsteroidal anti-inflammatory drugs (NSAIDs) or analgesics. Subacromial corticosteroid injections may be added to manage pain. Asymptomatic calcifications generally do not require treatment; however, when symptoms persist or become disabling, further intervention is warranted. If symptoms continue beyond 8 weeks, the condition is considered chronic, and nonoperative alternatives such as extracorporeal shock wave therapy (ESWT) or ultrasound-guided percutaneous irrigation of calcific tendinopathy (US-PICT) can be considered. These nonoperative therapies demonstrated consistent clinical efficacy, resulting in favorable outcomes in approximately 70% to 80% of patients with RCCT [45–47]. In approximately 20% to 30% of cases where symptoms persist despite nonoperative management, surgical management may be considered for symptom relief [45, 48]. Surgical intervention has been associated with superior improvements in functional outcome measures, while achieving pain reduction comparable to that observed with nonoperative management [49].

### Conservative treatment

There is broad consensus that conservative treatment should be the first-line approach for RCCT. This typically includes rest, NSAIDs, physical therapy, and, in selected cases, subacromial corticosteroid injections during later stages. Pain control is the primary goal in the acute phase, with NSAIDs being the most commonly used agents, although their use should be carefully considered in patients with gastrointestinal or cardiovascular comorbidities [50].

Yokoyama et al. explored an alternative pharmacological approach, demonstrating that cimetidine could reduce symptoms in patients with RCCT. Although the mechanism is not fully understood, it is hypothesized to involve serum calcium reduction. However, this study is limited by low sample size [51].

Physical therapy remains a cornerstone of conservative management, focusing on range-of-motion and scapular stabilization exercises to prevent joint stiffness and improve biomechanics. Matsen et al. [52] suggested that range-of-motion exercises may prevent glenohumeral stiffness and adhesive capsulitis, although a direct link between RCCT and capsular involvement remains unproven. Scapular dyskinesia, often associated with subacromial impingement, can worsen shoulder pain; therefore, rehabilitation aimed at

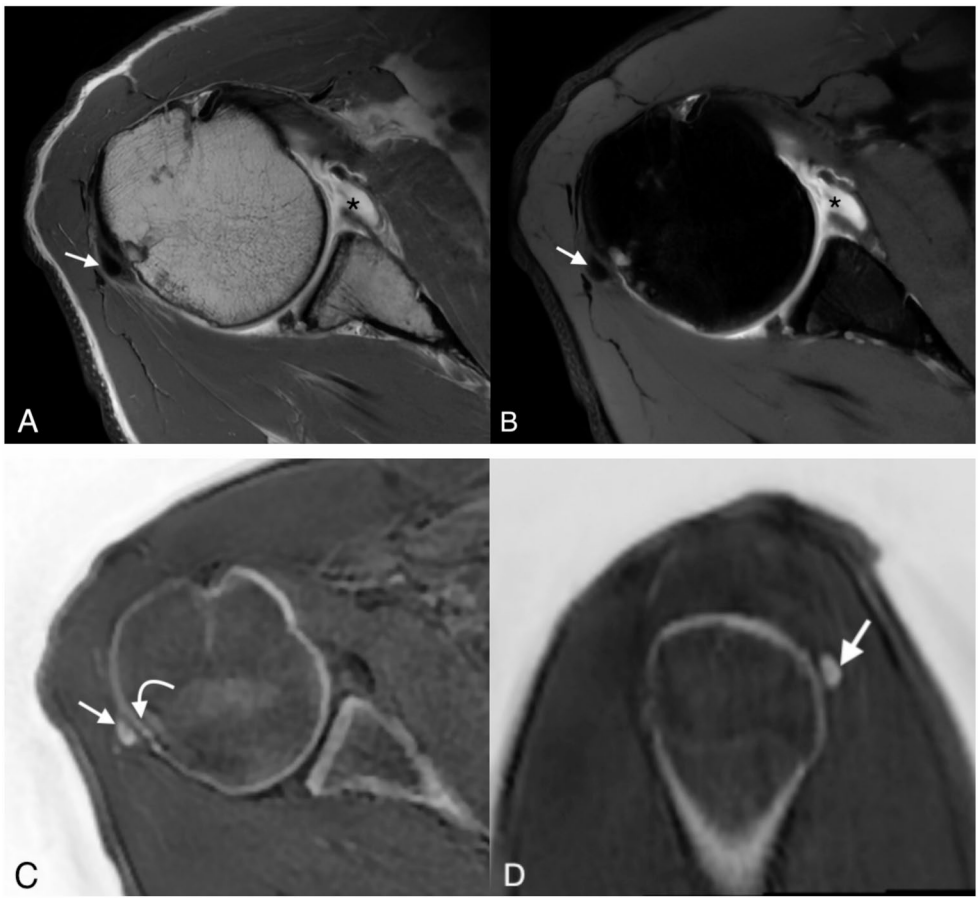


**Fig. 8** MR arthrography of a 35-year-old male patient with painful shoulder instability. MRI images show a lesion of the anteroinferior glenoid labrum (asterisk), signs of a Hill-Sachs lesion (curved arrow), and calcific tendinopathy of the infraspinatus with an intact pre-insertional calcification (arrows) measuring approximately 7 mm. **A** axial T1 FSE (**B**) axial T1 Fat Sat (**C**) axial ZTE/MR bone (**D**) sagittal ZTE

improving scapular kinematics has shown beneficial effects on symptom relief [53, 54].

Ogon et al. [55] evaluated the effectiveness of a multimodal conservative treatment regimen consisting of physical therapy (including heat and cold application, manual therapy, electrotherapy, and iontophoresis), oral analgesics and NSAIDs, and up to three subacromial corticosteroid injections. Nonresponse to treatment was characterized by ongoing clinical manifestations lasting no less than 6 months, including a minimum of 3 months of standardized therapy. The overall failure rate was 27%. Negative prognostic factors included bilateral calcific deposits, anterior acromial localization, medial (subacromial) extension, and large deposit volume. In contrast, favorable outcomes were associated with Gartner type III calcifications and the absence of sonographic sound extinction.

Cho et al. [26] also supported the efficacy of conservative therapy, reporting good to excellent outcomes in 72% of patients treated without surgery.

Subacromial corticosteroid injections are often employed in the acute symptomatic phase, particularly when bursitis or impingement is suspected [3]. Nonetheless, their use remains controversial. While such injections may provide temporary pain relief, some evidence suggests they may interfere with the natural resorption of calcific deposits, potentially exerting a detrimental effect [56, 57].

## Extra-corporeal shock wave therapy (ESWT)

ESWT has been implemented as a noninvasive treatment modality for musculoskeletal pathologies since the 1990s, with established application in RCCT. This technique delivers repetitive pulses to the affected area, with energy generated via electrohydraulic, electromagnetic, or piezoelectric mechanisms. Shock waves can be delivered as focal or radial forms and are categorized by energy flux density into low (< 0.08 mJ/mm²), medium (0.08–0.28 mJ/mm²), and high-energy (0.28–0.60 mJ/mm²) levels [58].

A systematic review and meta-analysis indicated that high-energy ESWT [59] significantly reduces pain and disability, with a higher complete resorption rate of calcifications compared to low-energy ESWT. However, no significant clinical differences have been observed between focal and radial modalities [59]. However, the evidence quality of correlated studies is rated as very low, and ESWT is associated with complications such as acute pain, superficial skin lesions, and local hematomas, particularly with higher-energy shockwaves.

The proposed mechanisms of action include both mechanical and molecular effects. Mechanically, ESWT may fragment calcium deposits by increasing intralesional pressure. On a cellular level, ESWT has been shown to induce neovascularization, enhance anti-inflammatory cytokine production, and stimulate growth factor release, collectively promoting phagocytic activity and calcium resorption [60]. In vitro studies support these biological effects through observed increases in cell proliferation and metabolic activity within tendon tissues [26, 61].

A meta-analysis by Ioppolo et al. demonstrated significantly greater rates of total and partial calcific deposit resorption at 6 months post-ESWT compared to placebo [62].

Long-term outcomes are less well established, though Daecke et al. [45] reported that 70% of patients remained surgery-free at 4 years following ESWT, while 20% ultimately required operative intervention. Comparative studies have shown that ESWT provides superior clinical outcomes to placebo, and is comparable to arthroscopic intervention in terms of pain reduction and functional improvement, but with fewer complications [63].

Adverse events are typically limited to localized discomfort and transient skin reactions.

Furthermore, combining ESWT with US-PICT may enhance therapeutic success compared to ESWT alone [64]. Kim et al. [60] found US-PICT to be superior to ESWT in both clinical and radiological outcomes, and combined approaches yielded even better results. Despite promising short- and mid-term results, ESWT appears less effective during the acute resorptive phase of RCCT, and further research is warranted to optimize treatment parameters and patient selection.

## US-PICT and needling

US-PICT has emerged as a first-line, minimally invasive treatment for RCCT [65, 66], especially in the acute phase and in cases with soft or fluid calcifications [67]. US-PICT is considered superior to ESWT and subacromial corticosteroid injections in terms of both symptom relief and functional improvement, with a low complication rate [68–70]. The procedure is designed to fragment and flush calcium deposits using one [71, 72] or two [73] needles inserted under US guidance, through the injection of warm saline solution (typically at 42 °C) [33]. The technique typically begins with subcutaneous and subacromial bursal anesthesia (e.g., lidocaine or mepivacaine), followed by the insertion of one or two 14–18 Gauge needles into the calcific deposit under real-time US visualization. Regarding needle length, conventional 5-cm long needles are generally sufficient to reach the deposits. However, in patients with larger body build or with deeper deposits, longer needles may be used [7, 74]. In the single-needle approach (Figs. 9 and 10), fluid is injected and then aspirated back into the syringe, drawing dissolved calcium with it. In the two-needle technique, a lavage circuit is created: the first needle delivers fluid into the calcification, while the second drains it [65, 67, 75]. Warm saline improves dissolution efficacy and reduces procedure time and postprocedural bursitis [33]. The procedure concludes with a corticosteroid injection (typically methylprednisolone 40 mg) into the subacromial-subdeltoid bursa to minimize post-interventional inflammation. US color Doppler can assist in confirming appropriate fluid distribution [76].

US-PICT is generally performed with the patient in a supine position using a high-frequency linear probe, ensuring accurate needle placement and avoiding radiation exposure [7].

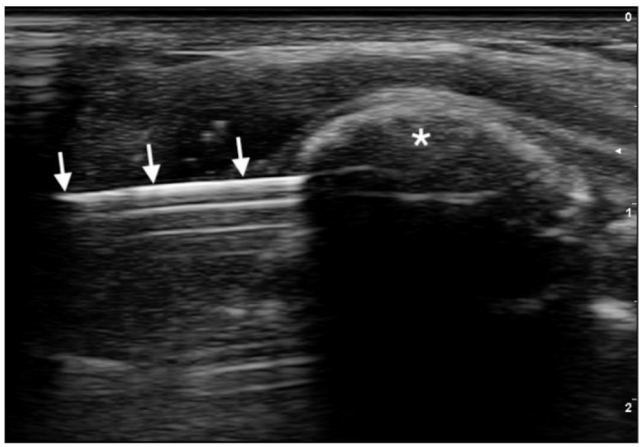

**Fig. 9** Ultrasound-guided percutaneous irrigation of calcific tendinopathy with the single-needle (arrows) procedure. Treatment of left shoulder calcific tendinopathy is performed, with a 20-mm calcification (asterisk) of the supraspinatus tendon

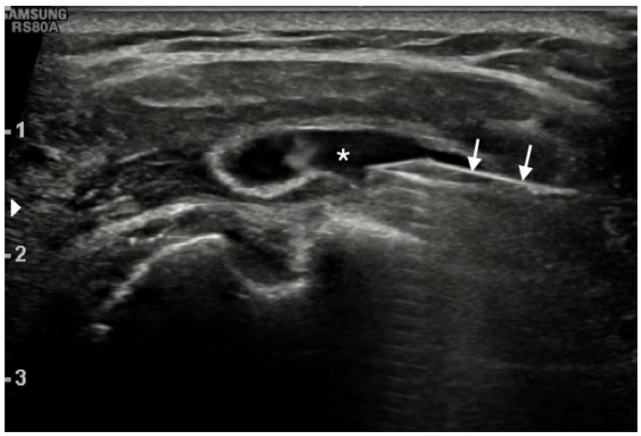

**Fig. 10** End of ultrasound-guided percutaneous irrigation of calcific tendinopathy with the single-needle (arrows) procedure. The calcification is totally empty (asterisk)

Dry-needling is an alternative for hard calcifications or when lavage is not feasible. This involves repeated puncturing of the calcific area to induce mechanical fragmentation, local bleeding, and subsequent resorption [76].

Contraindications for US-PICT include asymptomatic calcifications, very small deposits (< 5 mm), intra-bursal migration, or intraosseous extension, which are associated with poorer outcomes [67]. In cases of bursal migration of calcific material, treatment should also address the associated inflammatory bursopathy, usually with ultrasound-guided subacromial-subdeltoid bursal injection, while residual symptomatic migrated fragments may require targeted needling or lavage in selected cases, which however may not always be possible [77].

Despite different procedural variations, no consensus currently exists on the optimal needle size or number, although some studies suggest two-needle techniques are preferable for hard deposits, while single-needle techniques may be more efficient for fluid calcifications [74].

Sconfienza et al. demonstrated that previous unsuccessful ESWT does not adversely affect the efficacy of US-PICT. Consequently, it remains a valid and effective treatment option for calcific rotator cuff tendinopathy, including ESWT-resistant cases [78].

US-PICT is a safe and effective outpatient procedure that avoids hospitalization and promotes rapid recovery, often allowing a same-day return to normal activities.

Further research is needed to refine technique standardization and explore the role of adjuncts such as platelet-rich plasma (PRP), which has not shown superiority over corticosteroids in post-procedural management [79].

### Platelet-rich plasma (PRP)

PRP therapy has gained attention as a regenerative treatment for musculoskeletal tissues, particularly tendinopathies. PRP delivers high concentrations of growth factors and bioactive molecules to promote tissue healing by stimulating cellular proliferation, differentiation, and migration [80–82]. However, clinical results are mixed, likely due to variability in PRP preparation methods, including differences in platelet concentration, leukocyte content, activation procedures, and PRP volume. Studies indicate that PRP effects may depend on the severity of tissue damage [83].

In vitro and in vivo evidences suggest PRP can enhance tenocyte proliferation and tendon remodeling [84]. Clinically, PRP is considered for cases unresponsive to conservative treatments; limited case studies, such as one involving supraspinatus tendinopathy, show promising results with symptom resolution and calcification disappearance [85]. Yet, high-quality randomized controlled trials are scarce. Comparisons between PRP combined with other therapies (e.g., US-PICT) and standard treatments show mixed outcomes, with some benefits observed at longer follow-ups but increased complications [86]. PRP, prepared from autologous blood via centrifugation to concentrate platelets, releases growth factors essential for tissue repair. Its application in RCCT has been studied in only two RCTs, with conflicting results [87, 88]: one showing superior outcomes over dry needling [89], and another finding no significant difference compared to placebo [90]. Although the efficacy in RCCT remains uncertain, PRP presents a potentially effective option, especially in refractory cases.

### Surgery

Surgical intervention is typically reserved for patients with symptoms persisting beyond six months despite conservative care. Arthroscopic procedures, such as bursectomy and direct removal of calcific deposits, are currently considered the treatment of choice in chronic RCCT cases unresponsive to conservative or less invasive approaches. Arthroscopy is now favored over open surgery due to lower morbidity and comparable outcomes [91–93]. A major advantage of arthroscopy is the opportunity to perform additional procedures, including subacromial decompression and thorough joint debridement during the same session [94].

The extent of calcific deposit removal in RCCT remains a subject of ongoing debate [26]. While several authors emphasize the importance of complete excision, citing an inverse correlation between functional outcomes and the volume of residual calcification [91, 95, 96], others argue that total removal is not essential. Indeed, it has been suggested that the surgical incision alone may be sufficient to initiate a cell-mediated resorption process [26, 92, 97]. Furthermore, partial removal may be advantageous in preserving tendon integrity [92].

Surgical treatment has been associated with greater improvements in functional scores and comparable pain reduction to nonoperative interventions [49].

## Conclusion

RCCT is a multifactorial condition characterized by pathological calcium deposition in tendinous tissue, most commonly affecting the supraspinatus tendon. RCCT is a common cause of shoulder pain and functional limitation, typically diagnosed through CR or US.

While spontaneous resorption of calcium deposits occurs in many cases, persistent symptoms may require medical intervention. Most patients demonstrate a favorable response to conservative management, including rest, NSAIDs, and physical therapy. In chronic or refractory

cases, nonoperative treatments such as ESWT and US-PICT are highly effective. US-PICT offers particular advantages, including lower complication rates and successful outcomes even after failed ESWT. PRP has emerged as a promising adjunctive treatment in selected refractory cases, although current evidence remains limited, heterogeneous, and further validation is needed to confirm its efficacy. Surgery remains a reliable and definitive option in cases unresponsive to less invasive therapies.

**Data availability** No clinical data were used or analysed for the current paper since it is an educational review.

## Declarations

**Conflict of interest** The authors declare that they have no conflicts of interest.



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