

Calcific tendinopathy of the shoulder with intraosseous extension: outcomes of ultrasound-guided percutaneous irrigation

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Abstract

Objectives Rotator cuff calcific tendinopathy (RCCT) with intraosseous extension is a rare complication of tendinous and peritendinous involvement. The purpose of our study is to evaluate the outcome of ultrasound-guided percutaneous irrigation of calcific tendinopathy (US-PICT) in patients with intraosseous involvement.

Materials and methods From January 2011 to June 2014, patients with a clinical and imaging diagnosis of RCCT were prospectively categorised in two groups based on imaging findings: group A (10 patients) with intraosseous RCCT and group B (control group 35 patients) without osseous involvement. US-PICT followed by subacromial injection was applied to all patients in groups A and B. During a 1-year follow-up, treatment outcome in terms of pain and functional improvement was evaluated at 3 weeks, 3 months, 6 months, and 1 year, with the use of a four-grade scale. The study has been approved by our hospital's ethics committee.

Results Mean improvement scores of group A were significantly lower than those of group B at all time points ($p < 0.0001$). Improvement of group B was noted mainly within the first 3 months post-treatment ($p = 0.016$).

Conclusion Outcomes of ultrasound-guided treatment in cases of RCCT with intraosseous extension are significantly less favourable than in purely tendinous or peritendinous disease.

Keywords Rotator cuff calcific tendinopathy treatment · Hydroxyapatite deposition disease/treatment · Intraosseous · Ultrasonography · MRI diagnosis · Shoulder · Calcifications

Abbreviations

RC	Rotator cuff
BME	Bone marrow oedema
NSAIDs	Non-steroidal anti-inflammatory drugs
SSP	Supraspinatus
ISP	Infraspinatus
US-PICT	Ultrasound-guided percutaneous irrigation of calcific tendinopathy
RCCT	Rotator cuff calcific tendinopathy
ESWT	Extracorporeal shockwave therapy

Introduction

Rotator cuff calcific tendinopathy (RCCT) is a clinical entity commonly affecting the shoulder. It can be found in asymptomatic individuals with an incidence of around 3% and it has a predilection for women aged between 40 and 60 years [1]. It is usually related to the deposition of hydroxyapatite crystals and although the development of intratendinous calcifications has been attributed to various aetiologies, the pathophysiology of the disease has yet to be clarified [2]. According to Uthoff and Loehr the clinical course of the disease consists of three distinct stages: “pre-calcific”, “calcific” and “post-calcific”. The calcific stage can be further subdivided into the formative, the resting and the resorptive phases, a division that is also clinically relevant, as pain usually occurs during the resorptive phase [2, 3]. One of the complications of RCCT is the

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migration of the calcifications to the adjacent bone, which causes disruption of the cortex and an osteolytic lesion of the humeral head at the insertion site of the initially affected tendon, usually the supraspinatus (SSP). Despite its uncommon nature, this complication is of high importance because it can be mistaken for a bone tumour, whereas arthroscopic treatment has been found to be less effective than in a classic location within or adjacent to a tendon [4, 5].

The preferred treatment for RCCT remains a subject of debate [6]. Physical therapy and non-steroidal anti-inflammatory drugs (NSAIDs) are regarded as the first-line treatment in self-limited cases [7]. Extracorporeal shockwave therapy (ESWT) represents an alternative non-surgical treatment, being associated with satisfactory outcomes; however, it is not preferable during the hyperalgetic crisis because of unbearable pain [8]. Arthroscopy has been used to treat RCCT with satisfactory mid-term results [9] and is considered the method of choice once non-surgical treatments have failed. Ultrasound-guided percutaneous procedures, by means of ultrasound-guided percutaneous irrigation of calcific tendinopathy (US-PICT), are considered an effective treatment for RCCT and are associated with short- and long-term clinical improvement [6, 10]. Our primary objective was to compare the outcomes of US-PICT and subacromial injection in patients with and without osseous extension of RCCT.

Materials and methods

Patients

From January 2011 to June 2014, a total of 124 patients with a clinical and radiological diagnosis of RCCT were prospectively studied. All patients signed informed consent to undergo the MRI examination and the US-PICT procedure. The study has been approved by our hospital's ethics committee and in accordance with the Declaration of Helsinki.

Inclusion criteria comprised:

1. Available X-rays showing calcifications around rotator cuff (RC)
2. MRI according to our institutional protocol
3. Persistent shoulder pain, refractory to conservative treatment with oral analgesics and NSAIDs, for at least 6 weeks, but less than 6 months
4. Ultrasound-guided treatment consisting of US-PICT and subacromial injection
5. One-year follow-up after treatment

We included patients with pain duration of less than 6 months, to avoid adhesive capsulitis development due to chronic immobility, interfering with the interpretation of the outcome.

Patients with a history of trauma, clinical or imaging findings suggestive of other/concomitant causes of shoulder pain (RC tear, symptomatic impingement syndrome, infection, adhesive capsulitis), previous interventions (arthroscopy/surgery, ESWT, injections), specific medication (insulin, anticoagulant therapy) or allergy to lidocaine, were excluded from the study (Fig. 1).

According to the X-ray and MRI findings, the patients were classified into two groups: group A included patients with findings of subacromial-subdeltoid bursitis, tendinous or peritendinous RC calcifications with surrounding oedema on fat-suppressed MR images, intraosseous extension of the RC calcifications through cortical disruption, and humeral head bone marrow oedema (BME), located at the greater or lesser humeral tuberosity; group B, the control group, included patients as in group A, without intraosseous extension of the RC calcifications through cortical disruption and/or humeral head BME.

A clinical evaluation was performed by using a score measuring the clinical improvement in terms of pain and functional impairment compared with the immediate pre-treatment period, at 3 weeks, 3 months, 6 months and 1 year. Namely, patients were asked to score their clinical improvement as follows:

1. Grade 1—no improvement
2. Improvement of less than 50%
3. Improvement of between 50 and 70%
4. More than 70% improvement

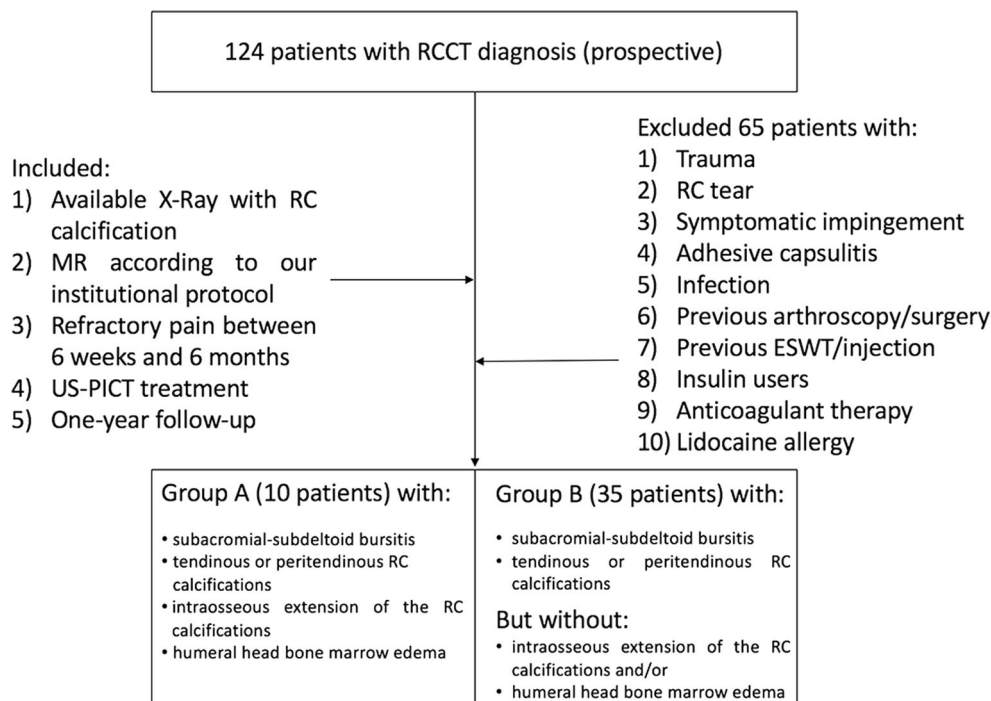
No follow-up ultrasound examinations were performed. Patients were self-evaluated according to the above-mentioned criteria and delivered the score to the senior author by phone contact.

MRI technique and analysis

All MRI examinations were performed on a 1.5-Tesla scanner (Vision Hybrid; Siemens, Erlangen, Germany) according to the standard shoulder MRI protocol utilised in our department. Patients were scanned in a supine position. The MRI shoulder protocol consisted of fat-suppressed (FS) oblique coronal and sagittal T2-weighted turbo spin echo (TSE) images (TR/TE: 3,050/75; slice thickness: 4 mm; matrix: 192 × 192), oblique coronal and oblique sagittal T1-weighted TSE images (TR/TE: 502/15; slice thickness: 4 mm; matrix: 320 × 240) and axial proton density (PD) FS TSE images (TR/TE: 1,600/13; slice thickness: 4 mm; matrix: 192 × 192). A field of view (FOV) of 15 cm was used. Fat suppression was achieved with spectral presaturation.

All images were analysed on an EVORAD research RIS/PACS system by a musculoskeletal radiologist with 29 years of experience in musculoskeletal imaging and intervention

Fig. 1 Patient selection strategy. *RCCT* rotator cuff calcific tendinopathy, *RC* rotator cuff, *ESWT* extracorporeal shockwave therapy, *US-PICT* ultrasound-guided percutaneous irrigation of calcific tendinopathy



and a 5th-year radiology resident and all decisions were reached in consensus. The rotator cuff and associated structures (bursa, musculature, acromioclavicular joint), the osseous structures, the long bicipital tendon and the glenohumeral joint (joint fluid, cartilage, labrum, support structures) were assessed. Bursitis was defined as distension of the subacromial–subdeltoid bursa above 2 mm [11]. Intraosseous extension of RCCT was defined as the presence of calcifications within the bone along with other bone marrow changes, with or without any continuity with concomitant RCCT. Cortical erosions were defined as a gap/discontinuity in the bony cortex.

Treatment methods

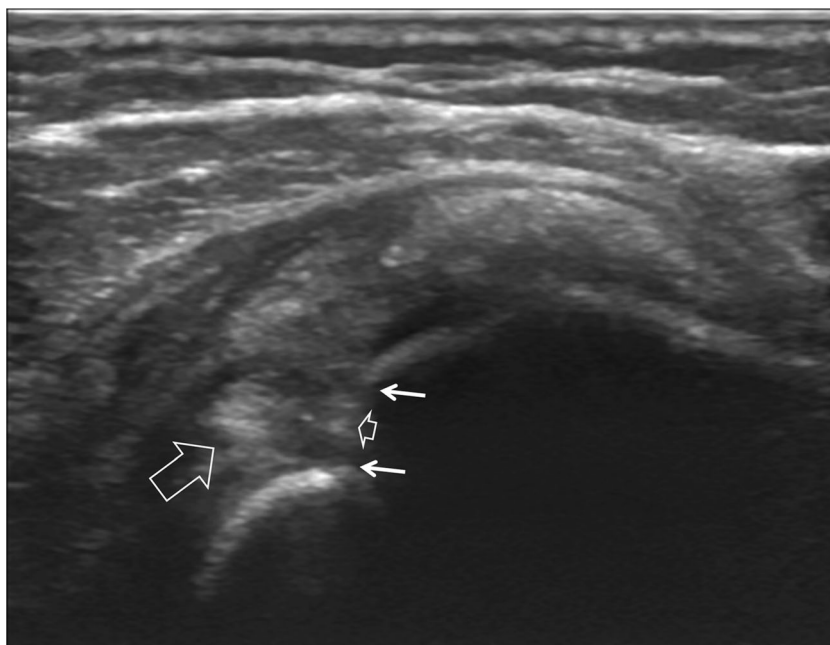
All patients in both groups underwent the US-PICT procedure under a musculoskeletal radiologist with 29 years of experience in musculoskeletal imaging and intervention. A second operator, a 5th year radiology resident, and a nurse were present to assist with the procedure. No anxiolytic medication was used to reduce the risk of vagal reaction. The radiologist explained the procedure to the patients in detail at the time of the clinical examination before the ultrasound examination.

An initial diagnostic ultrasound examination according to guidelines issued by the European Society of Musculoskeletal Radiology [12] was used to confirm the X-rays and MRI findings and to guide the treatment (Fig. 2). Patients were treated either in the sitting or in the lateral decubitus position with the painful shoulder upwards depending on their sensitivity to interventions. In either position, the shoulder was

internally rotated with the arm directed to get a wallet from the back pocket, when calcifications were located within or around the SSP tendon and in neutral position for all other locations. The US scanners used in the study were the GE Logic 7 and 9 provided with 10 to 15MHz linear array transducers. A two-stage US-guided treatment protocol consisting of an initial single needle US-PICT procedure followed by a subacromial-subdeltoid steroid/analgesic injection with sterile technique was applied.

The ultrasound probe was cleaned before the procedure, but not wrapped with a sterile cover. At the beginning of the procedure, aseptic cleaning utilising surgical gloves with povidone–iodine solution (10%, 2 times) and application of anaesthesia locally and around the calcifications (23G needle, 2% lidocaine) were performed. Using sterile contact gel, a single 18G needle barbotage was performed under real-time ultrasound guidance. After confirming the location of the needle within the calcifications, an injection of lidocaine, fragmentation of the calcifications by rotating and moving the needle tip inwards and outwards, the injection of saline, and aspiration were performed (Fig. 3). The US-PICT was repeated until the aspirated fluid was clear without visible calcific material and the tendinous or peritendinous calcifications were not visible or showed a substantial reduction in size. In cases of hard calcifications, the procedure ceased when the scratching diminished substantially. After the end of US-PICT, a subacromial–subdeltoid bursal injection under real-time ultrasound guidance was performed with 1 mL (40 mg) of triamcinolone mixed with 6 mL of 0.5% bupivacaine and 1 ml of 2%

Fig. 2 Ultrasound supraspinatus image showing intratendinous calcifications (*long open arrow*) in continuity with migrating calcifications intraosseously (*short open arrow*) through cortical disruption (*thin arrows*)



lidocaine. As a rough estimation, the maximum longitudinal diameter of intraosseous calcifications ranged from 1 to 3 cm. However, owing to the ill-defined borders of the majority of the lesions, no analysis of the dimensions was attempted. The large volume of the solution attempted diffusion of triamcinolone throughout the large bursal space whereas the addition of lidocaine was preferred for rapid pain relief, because of its relatively immediate onset of action [13]. Reduced pain and increased range of motion

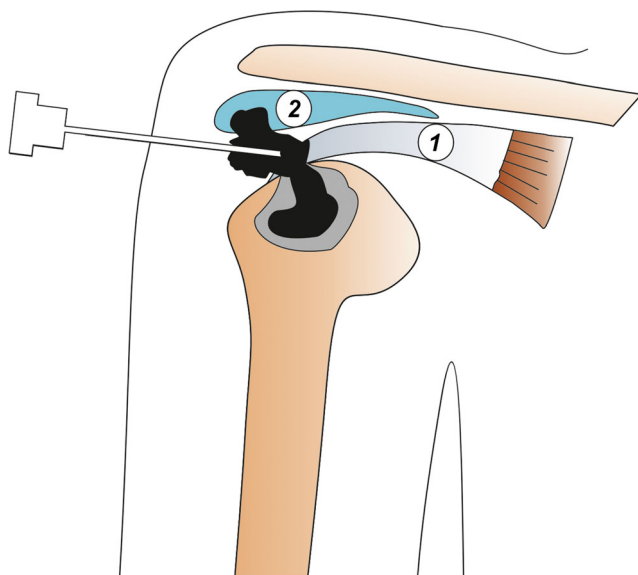


Fig. 3 Illustration presenting the ultrasound-guided treatment technique. A single-needle barbotage of the intra- and peritendinous calcifications (1) is performed, followed by a subacromial, bursal (2) injection

provided a first indication of success immediately after the procedure. No attempt at dry needling was performed within the areas of tendon degeneration. No second attempt at US-P ICT was performed in any of the patients in the two groups. All patients were instructed to remain in the department for 30 min and receive paracetamol in case of pain exacerbation for the 48 h after treatment. In addition, recommendations of protection from overloading for 3 to 4 days were provided. After the procedure and for the following 4–6 weeks, all patients underwent a shoulder exercise program, which was taught by the senior radiologist who performed the intervention. Adherence to the training schedule was the responsibility of each individual patient and all patients included in the study reported full compliance with the recommendations. The program took place twice-a-week and included posterior capsular stretches and active-assisted range-of-motion exercises with a suggestion for further exercise with swimming and avoiding overhead movements. Follow-up did not include any imaging and was based solely on the clinical outcome [10].

Statistical analysis

Data were analysed with the use of IBM SPSS Statistics for Windows v.22 (IBM, Armonk, NY, USA) and differences between mean improvement scores for each follow-up time point were evaluated with the use of the Mann–Whitney *U* test. Statistical significance was denoted by a *p* value less than 0.05. Patient ages were expressed as mean values (\pm standard deviation).

Results

The age range in group A (3 men, 7 women) was 35–60 years (mean 44.4 ± 7.5), and in group B (24 men, 11 women) it was 33–59 years (mean 49.8 ± 6.7). Three patients did not undergo US-P ICT because of large humeral intraosseous cystic lesions that required surgical intervention with bone grafting. Moreover, 6 patients who were lost in follow-up beyond 6 months were not included in the study. According to our inclusion and exclusion criteria (Fig. 1), 10 patients were included in group A and 35 patients were included in group B, whereas 65 patients were excluded from our study.

Descriptive statistics showed that in 80% of group A patients ($n = 8$) the SSP tendon was affected, whereas the infraspinatus (ISP) was involved in only 20% of the patients ($n = 2$). In addition, in 2 patients with SSP calcifications, bursal calcification was depicted, whereas in 2 patients, diffuse SSP and ISP oedema was also evident (see Fig. 8). Women were affected more than men (70 vs 30%) and the mean age of patients with osseous involvement was 44.4 ± 7.5 years.

The mean improvement score of group A was found to be significantly lower at all time points during the follow-up ($p < 0.0001$) compared with group B (Table 1). There was no clinical improvement in group A over the course of 1 year as proved by a non-significant comparison between the mean improvement score at 3 weeks and at 1 year ($p = 0.2781$). Treatment of the control group (group B) resulted in a significant improvement in the patients' score, with a median value score at 3 weeks of 3 (interquartile range [IQR] 2–3) compared with a median score at 1 year of 4 (IQR 4–4; $p < 0.0001$). However, the score increased significantly only between 3 weeks and 3 months ($p = 0.016$) and not between 3 months and 1 year ($p = 0.058$).

Calcifications as shown on X-rays, were present on MRI in all patients of group A and in 30 patients of group B (86%). On MRI, tendinous and peritendinous calcific deposits appeared as low or mixed low and intermediate signal intensity lesions on both T1- and T2-weighted images, demonstrating an oedematous surrounding rim and soft-tissue oedema, in all our patients (Figs. 4, and 5). On fat-suppressed PD-/T2-weighted images, intraosseous extension of RCCT was depicted as an area of high signal intensity ($n = 2$) or heterogeneous signal intensity ($n = 2$) lesion with cystic components and low signal foci, representing calcification (Figs. 6, 7). In 6 patients, it appeared as a low signal intensity lesion, on all

Table 1 Mean improvement scores of the two groups

Group	3 weeks	3 months	6 months	1 year
A	1 (1-2)	1 (1-2)	1 (1-1.25)	1 (1-1)
B	3 (2-3)	4 (2-4)	4 (3-4)	4 (4-4)

Values represent median scores (interquartile range)

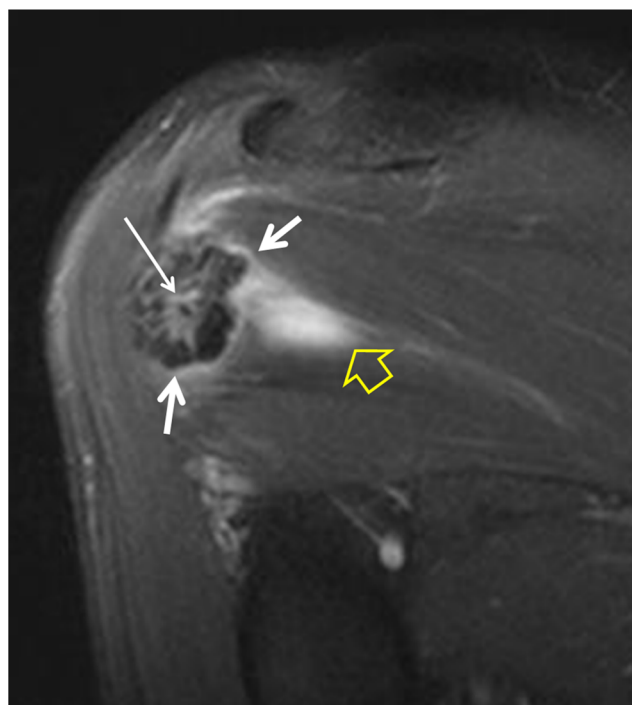


Fig. 4 A 60-year-old female patient with a 4-month history of pain. The fat-suppressed oblique coronal PD-weighted MRI, shows the perilesional rim of oedema (arrows), the soft-tissue oedema (open arrow) and the internal high signal intensity (long arrow) in keeping with an active calcific deposit lesion

pulse sequences (Figs. 8, 9). Intraosseous lesions were surrounded by diffuse or rim-like BME in all 10 patients.

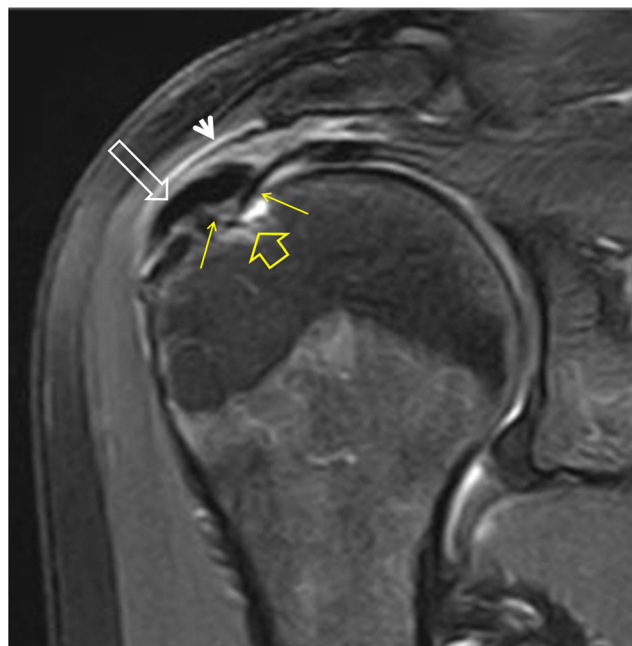


Fig. 5 A 50-year-old male patient with a 5-month history of pain. The fat-suppressed oblique coronal PD-weighted MRI shows the intratendinous calcification (long open arrow), the cortical irregularity (arrows), the subcortical cyst and reactive marrow oedema (short open arrow), and the subacromial bursitis (arrowhead)

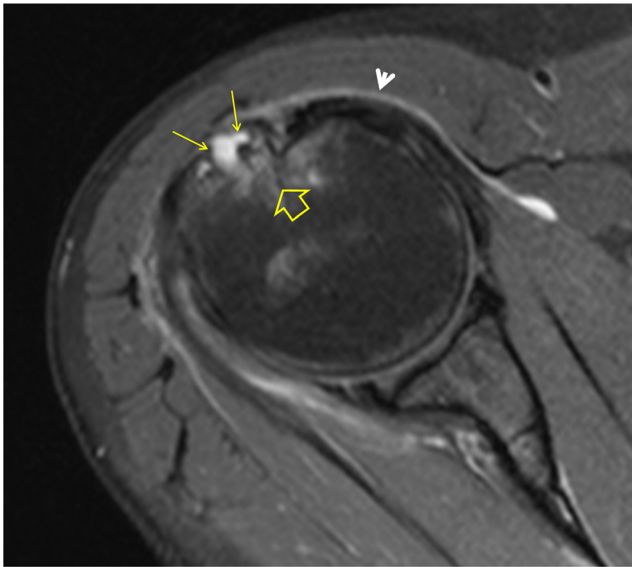


Fig. 6 A 47-year-old female patient with a 7-month history of pain. The fat-suppressed axial PD-weighted MRI shows cortical irregularity and a subcortical cystic change (*arrows*), bone marrow oedema (*open arrow*) and subacromial bursitis (*arrowhead*)

Discussion

There is a scarcity of literature on the comparison of the effect of US-PICT with subacromial–subdeltoid injection between patients with RCCT only and those with osseous

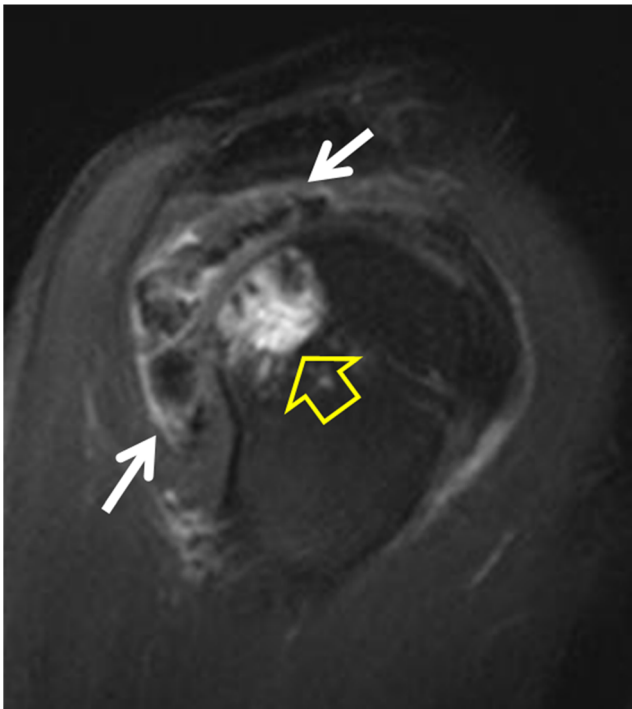


Fig. 7 Same patient as in Fig. 2. The fat-suppressed oblique sagittal T2-weighted MRI shows extensive calcifications within the subdeltoid bursa (*arrows*) and the subcortical bone marrow change with small foci of calcifications (*open arrow*)



Fig. 8 A 48-year-old male patient with a 3-month history of pain. The fat-suppressed axial T2-weighted MRI shows the calcific deposit of the supraspinatus (*arrow*), extending into the bone (*long arrows*) with reactive subcortical bone marrow oedema (*short open arrow*)

involvement. Osseous involvement in RCCT was first reported by Hayes et al. [14] with cortical erosions at the pectoralis major, gluteus maximus, and adductor magnus tendinous insertions. Up to today, several case reports [15–18] and few larger series have described osseous involvement at different sites [4, 5, 19]. Although the pathogenesis of bone erosion remains unclear, it is believed that active inflammation and local vascularisation at the tendon insertion or mechanical effects of muscle traction

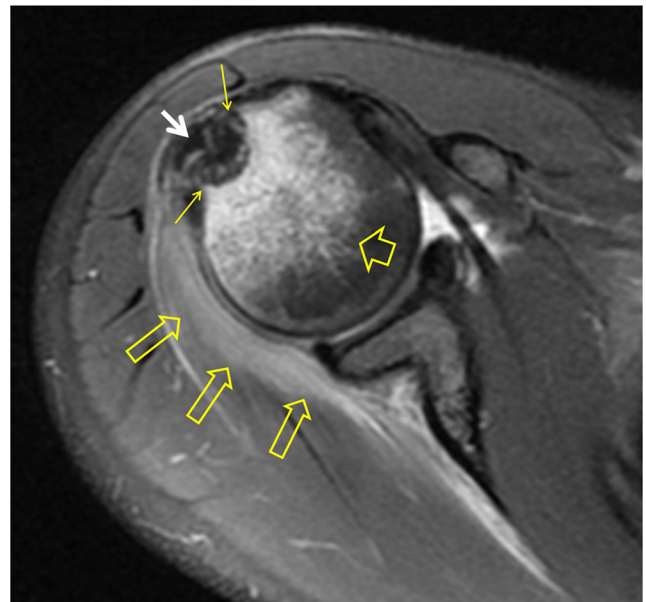


Fig. 9 A 35-year-old female patient with a 6-month history of pain. The fat-suppressed axial PD-weighted MRI shows the anterior calcific deposit of the infraspinatus (*arrow*), extending into the bone (*long arrows*) with reactive subcortical bone marrow oedema (*short open arrow*). Reactive oedema into the infraspinatus muscle is also shown (*long open arrows*)

may play a role in the development of cortical erosions [20]. In patients with intraosseous involvement, many authors have proposed cortical erosions and/or subcortical calcium migration as one of the causes altering the natural disease course and deteriorating patients' prognosis [4, 19, 21, 22].

Only limited case reports have described the imaging findings of intraosseous extension of RCCT in the shoulder [14, 19, 21, 23, 24]. Flemming et al. [19] reported "marrow involvement" in all their patients with available MRI examinations. However, they did not clarify whether "marrow involvement" refers to BME or intraosseous lesions other than cortical erosion, as they did not utilise fat-suppressed sequences for the evaluation of the majority of the lesions. In our study, intraosseous extension of RCCT was assessed with MRI. This strategy is in line with Porcellini et al. [4] who reported that MRI showed a sensitivity of 100% in identifying arthroscopically confirmed intraosseous lesions associated with RCCT.

The SSP represents the most common tendinous location of RCCT followed by infraspinatus, teres minor, and subscapularis [2, 6]. This is in keeping with the location of calcific deposits identified in our patients. Moreover, the mean age of our patients with intraosseous disease was found to be similar to that published for sole intratendinous involvement.

Our follow-up method is in accordance with Serafini et al. and Cacchio et al. [10, 25], who state that follow-up imaging studies are not required when the end-point is pain reduction and functional improvement. Furthermore, we share the opinion of Lanza et al. [6], who believe that once it is established that US-PICT is not detrimental to tendons, the imaging follow-up should not be routinely used, but rather only when complications are suspected. Thus, we support the opinion that during follow-up the evaluation of the morphology and the size of the calcific deposits does not provide any clinically relevant information nor does it correlate with the treatment outcome.

Comparison of mean improvement scores in group A at 3 weeks and 1 year showed no significant improvement after ultrasound-guided treatment, whereas outcomes for the control group are constantly improving over the course of the follow-up. However, it is extremely important to note that most of the improvement was reported within the first 3 months, whereas between 3 months and 1 year minimal and non-significant improvement was found that could possibly be attributed to the pharmacokinetics of the drug in the soft tissues of the shoulder.

To the best of our knowledge, there are no other studies comparing the clinical results of US-PICT combined with subacromial–subdeltoid bursa injection for the treatment of RCCT in patients with and without osseous involvement. Two previous reports [4, 5] have provided conflicting results on the surgical outcome of the

arthroscopic removal of intraosseous deposits in patients with RCCT. In accordance with our results, Porcellini et al. [4] showed that arthroscopic removal of the deposits and debridement of the residual lesion improved the Constant scores in patients with and without osseous extension of RCCT. However, the outcomes were significantly less favourable in the osseous extension group. Contrary to that, Seyahi and Demirhan report that arthroscopic removal of intraosseous and intratendinous deposits in patients with RCCT plus osseous involvement seems to be as safe and effective as the arthroscopic removal of intratendinous deposits in cases of tendinous involvement only [5].

Our study has specific limitations. First, the number of patients in group A is limited. However, this is expected as osseous involvement in shoulder RCCT is one of the least common complications of the disease. Another limitation is that in the present study, classification of calcific deposits is not provided. This is supported by various reports. Maier et al. [26] assessed the intra- and interobserver reliability of the various classification systems using X-rays and CT scans and concluded that all the scores showed insufficient reliability and reproducibility and they were statistically insignificant to be recommended as a routine investigation. Moreover, we agree with Seyahi et al. [5] with regard to their statement that the existing classification systems may not be applied to the classification of RCCT with osseous extension, as they do not describe the pattern of osseous involvement. Additionally, as stated by Serafini et al. in their response letter [10], the exclusion of Gärtner type III calcifications [26] has been used mainly in clinical trials of extracorporeal shockwaves. In addition, the fact that continuity between soft tissue and intraosseous calcifications was not observed in all our cases may constitute a limitation of our study. A possible explanation for the less beneficial outcome in patients with intraosseous RCCT may be related to the fact that no attempt was made towards removal of the intraosseous calcifications. Limited experience of an ongoing project of our group (unpublished data) showed that CT-guided removal of intraosseous calcifications was related to a favourable outcome. Finally, the clinical scale that was used to assess treatment outcome could also be considered a limitation. The scale described above is a modified visual analogue scale that has been used in previous studies, to which patients responded well, and that has proved easy to obtain [27].

In conclusion, we evaluated the therapeutic outcome of US-PICT combined with subacromial–subdeltoid bursa injection in this subgroup of patients, compared with patients with RCCT without osseous extension. We concluded that RCCT with osseous involvement should be considered a distinctive form of the disease with a less favourable outcome on US-PICT than in purely tendinous involvement.

Compliance with ethical standards

Conflicts of interest No conflicts of interest to disclose.

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