Resorption of calcific tendinitis of the shoulder after trauma: a case report focusing on resorptive factors

Reabsorção da tendinite calcária do ombro após trauma: relato de caso focado nos fatores reabsortivos

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Abstract

This is a case report of calcific tendinitis of the shoulder that began a resorptive process after a low-energy trauma. A 55-year-old female patient, previously asymptomatic, developed intense pain in the first days, which was difficult to manage with analgesics. The initial radiographic evaluation revealed an 8mm calcification of the left supraspinatus tendon, classified as Gartner type 1, which evolved into the resorptive phase immediately after the trauma. Radiological imaging on the ninth day showed that the calcification was in the final absorption stage. After effective pain control and physical rehabilitation, the patient fully restored the function of her left shoulder.

Keywords: Tendinopathy; Pain; Shoulder/surgery.

Resumo

Relato de um caso de tendinite calcária do ombro que iniciou quadro reabsortivo após trauma de baixa energia. Paciente feminina, 55 anos, previamente assintomática, evoluiu com dor de forte intensidade nos primeiros dias com difícil controle analgésico. Na primeira avaliação radiográfica, apresentou uma calcificação do tendão do supraespinhal esquerdo de 8mm, Gartner tipo 1, que evoluiu para fase reabsortiva no pós-trauma imediato. Na imagem radiológica do 9º dia a calcificação se apresentava em fase final de absorção. Após controle analgésico efetivo e reabilitação, evoluiu com restauração completa das funções do ombro esquerdo.

Palavras-chave: Tendinopatia; Dor; Ombro/cirurgia.

Introduction

Calcific tendinitis (CT) is a common painful condition of the shoulder initially described by Codman in the 1930s[1]. It accounts for 7% of shoulder pain cases and can present as single or multiple calcium hydroxyapatite deposits. It tends to resolve spontaneously, with interventional measures employed for refractory cases[1-4].

Treatment is usually clinical, focusing on symptom control. Other modalities such as barbotage injection, shockwave therapy, and surgery are used in refractory cases[4].

Study performed at the Clínica DORTO, Sete Lagoas, MG, Brazil.
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Conflict of Interest: None.
Source of funding: None.
Received: March 19, 2025.
Accepted: March 30, 2025

Citation: Fin M, Liu IAW. Resorption of calcific tendinitis of the shoulder after trauma: a case report focusing on resorptive factors. J Braz Musculoskelet Pain Soc. 2025;1(2):e11

Case Report

A 55-year-old right-handed female patient with no previous pathological history.

The patient underwent treatment for calcific tendinitis of the right shoulder between 2015-2017 with medication, subacromial corticosteroid injection, and physical rehabilitation, achieving good clinical evolution and resolution of the disease.

In 2024, she suffered a low-energy trauma to the left shoulder. In an emergency evaluation, there was no fracture and/or glenohumeral dislocation on clinical and radiographic assessment (Figure 1A); however, an 8 mm dense calcification was observed in the supraspinatus tendon, classified as Gartner type 1. She was prescribed symptomatic medications at the emergency department and for home use. Prior to the trauma, she was asymptomatic. Forty-eight hours after the trauma, despite analgesic use, she experienced progressive pain, reaching high intensity with moderate movement limitation. Due to intense pain, she returned for evaluation on the ninth day. A new radiological evaluation showed the evolution of the calcification into the resorptive phase (Figure 1B). At this point, pain management was optimized, and outpatient follow-up was scheduled.

At six weeks post-trauma, she presented with mild pain during shoulder elevation and was no longer using regular medication. She had partial restriction in the left shoulder range of motion, with anterior elevation of 120º, and was referred for physical rehabilitation.

At six months post-trauma, after rehabilitation, the patient was pain-free, with a slight limitation in the final phase of shoulder elevation and had returned to her work activities without restrictions. Radiography showed total resorption of the calcification (Figure 1C).

Epidemiology

CT affects 2.5%-7.5% of asymptomatic shoulders and most commonly affects the right side of women aged 30-50 years old. The gender ratio is 3:7, and bilateral cases account for 10-20%. The most affected tendon is the supraspinatus (80%) followed by the infraspinatus (15%) (Figure 2; Graph 1)[2,3,5].

Jomaa et al.[6] in his series, investigated the association of calcific tendinitis (CT) with lithiasis. He reported a history of nephrolithiasis in 22% and choledocholithiasis in 27% of patients. Other factors such as older age, higher body mass index, and glycemic alterations also contributed positively to this increase[6].

Endocrinopathies have some influence on the incidence of CT. In his study, Su et al.[7] demonstrated that patients with diabetes had a 27% higher risk of developing calcific tendinopathy of the shoulder within eight years after the initial diagnosis of the disease[7].

Pathophysiology

The mechanisms involved in CT formation are not officially standardized. Theories such as metaplasia of tenocytes to chondrocytes; tendon degeneration with fiber necrosis and subsequent calcification; erroneous differentiation of tendon stem cells into bone cells are postulated and contested in medical literature (Figure 3)[4,5].

Endocrinological and hormonal factors are associated with an increased incidence of calcific tendinitis (CT). Diabetic patients present calcifications three times

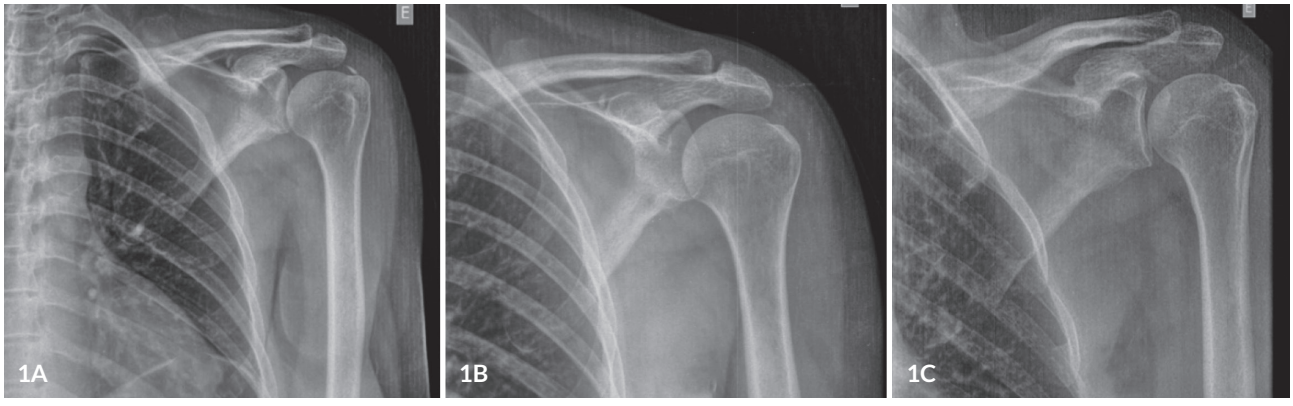


Figure 1. Radiological evolution of the calcification. Day of trauma (1A); 9th day after trauma (1B); 6 months after trauma (1C).

more frequently than control groups, while up to two-thirds of patients in some series presented endocrine alterations (Table 1)[1]. These patients experience an earlier onset of symptoms, tend to develop chronic symptoms, and more frequently require surgical treatment[4].

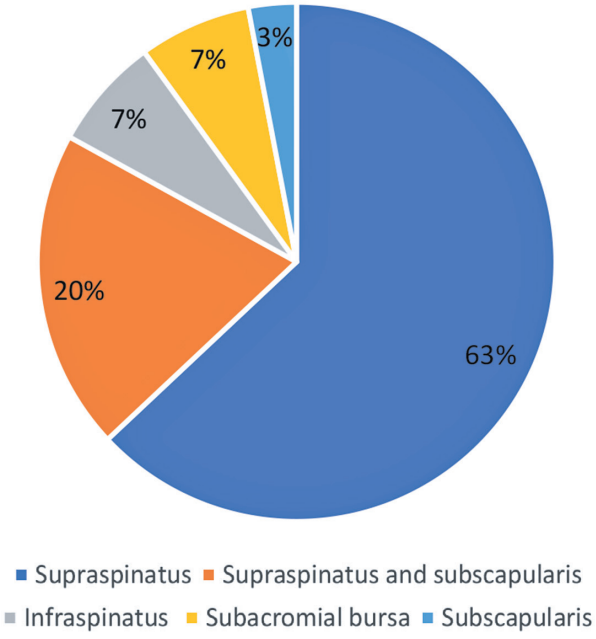
Uhthoff Sarkar[8] described CT in stages: pre-calcific, with the transformation of the tendon into fibrocartilaginous tissue; calcific, with the actual deposition of calcium and subsequent resorption; and postcalcific, with remodeling of the tendon tissue by fibroblasts.

The resorptive phase occurs after a latency period with no defined pattern. The calcified area undergoes a peripheral neovascularization process, facilitating the phagocytic system, mediated by macrophages, to remove the accumulated material. However, this "trigger" still lacks official literary references[4].

More recent findings also show some neoinnervation and an eight-fold increase in the presence of local monocytes and macrophages, in addition to tendon neovascularization. These factors directly contribute to pain, since a greater presence of nerves is positively correlated with more neovascularization and also with a higher frequency of intense pain. Neovascularization is associated with more frequent and more intense pain during sleep[9].

Clinical Manifestations

Clinical calcifications present in three scenarios (Figure 4), being asymptomatic in up to 20% of cases. When symptomatic, they manifest as sudden and poorly



Graph 1. Distribution of calcifications around the shoulder[8].
Source: Adapted from DE Carli et al.[20]

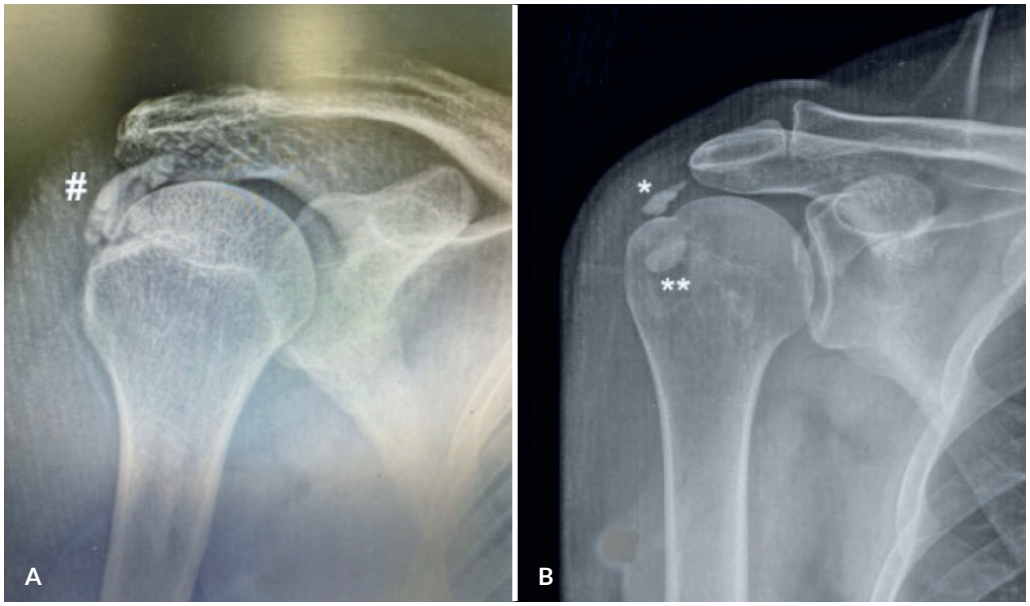


Figure 2. A. (#) Single calcification of the supraspinatus tendon. B. Multiple calcifications (*) supraspinatus and (**) infraspinatus.

delimited pain of variable intensity tending to high intensity, normally related to the resorptive phase[1,10].

Questioning about trauma, fever, and endocrine disorders helps in the differential diagnosis, including: capsulitis, infection, rotator cuff rupture, tenosynovitis, bursitis, arthritis, etc. A physical examination assessing amplitude and strength completes this clinical evaluation. Imaging tests such as simple radiography confirm calcification in most cases, and laboratory tests (C-reactive protein and blood count) help rule out infection[4]. Other imaging options are ultrasound (US) and magnetic resonance imaging. Currently, US is not only diagnostic, but has also been a powerful tool in treatment, guiding therapeutic interventions[1].

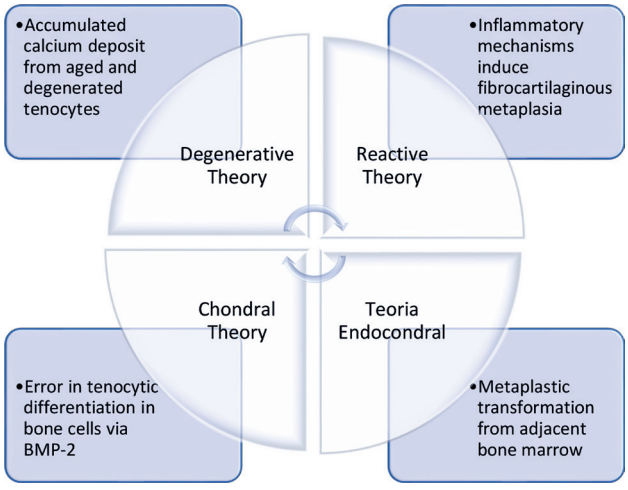


Figure 3. Theories of calcium deposition development. BMP (bone morphogenetic protein)[4].
Source: Adapted Greis et al.[4]

Table 1. Risk factors for calcific tendinopathy of the shoulder[1,4,6].

✓ Sedentary lifestyle
✓ Ischemic heart disease
✓ Systemic arterial hypertension
✓ Diabetes
✓ Thyroid diseases
✓ Alterations in estrogen metabolism
✓ Lithiasis
✓ Human leukocyte antigen serotype class A1
✓ Mutation in the human homologue of the murine progressive ankylosis gene (ANKH)
✓ Increased expression of tissue transglutaminase (tTG2) and its substrate osteopontin

Classification

CT is classified based on several factors, such as: pathophysiology, temporality, size (Table 2) and homogeneity of the deposit (Tables 3 and 4). The Gartner classification gained popularity due to its application in the prognosis of the disease, and is even used in the approach with shock wave therapy (Table 3)[11].

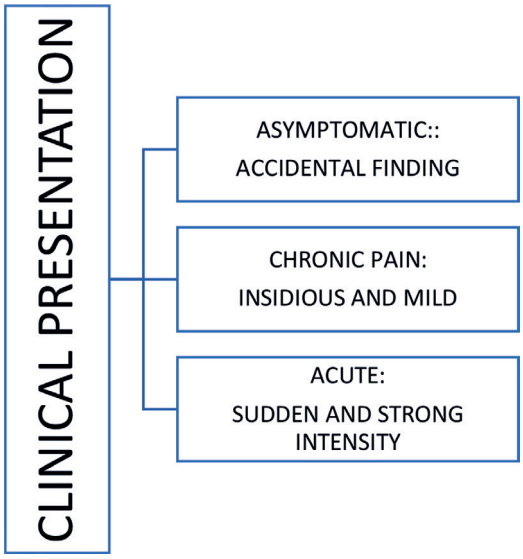


Figure 4. Clinical presentations of calcific tendonitis[9].
Source: Adapted Hackett et al.[9]

Table 2. Bosworth classification by CT size[21].

Small: up to 5 mm
Medium: 5–15 mm
Large: greater than 15 mm

Table 3. Gartner classification[11].

Type I - Well-demarcated dense
Type II - Smooth/dense or sharp/transparent contour
Type III - Smooth/translucent and blurred contour

Table 4. Molé Classification[22].

Type A: Dense, rounded, clearly delineated
Type B: Multilobular, radiodense and clear
Type C: Radiolucent, heterogeneous with irregular contour
Type D: Dystrophic calcified deposit

Treatments

Described as a self-limiting disease, calcific tendinitis (CT) in most patients resolves completely with only clinical management as the first line of treatment. This includes analgesics, anti-inflammatory drugs, and ultrasound (US)-guided interventions, promoting comfort for the patient. Clinical management also includes physical rehabilitation therapies[1,4].

Ogon et al.[12] defined the failure of surgical treatment as the maintenance of symptomatic CT after a minimum of 6 months of clinical treatment, including at least 3 months of standardized treatment. His studies also highlight prognostic factors-both positive and negative-for treatment outcomes (Table 5)[12].

Non-Surgical Treatment Modalities

Clinical approach is the basis of initial treatment. This includes rest, analgesics, anti-inflammatory medications, and physical therapy. Physical therapy aims to minimize joint contracture and prevent adhesive capsulitis[5].

Acetic acid iontophoresis: The peri-calcification acidification would promote the mobilization of hydroxyapatite by dissolving the deposit[4].

Therapeutic Ultrasound: Pulsed therapeutic ultrasound applied over 24 sessions in six weeks has shown pain reduction and improved quality of life in CT[4].

Extracorporeal shock wave therapy (ESWT): There are two different types of ESWT: focal, where the energy generated converges at an adjustable focal point at a predetermined depth in the body tissues where maximum pressure is reached; and radial, in which the maximum pressure is at the surface of the skin and disperses as it penetrates deeper. The differences in effectiveness are related to the physical characteristics and mechanism of action. Unlike focal ESWT, radial ESWT has a different linear pressure, lower energy values, relatively low propagation speed and short rise time. Both are described as effective in the treatment of CT, with significant improvement in clinical, functional and ultrasound findings after the use of focal, radial or combined therapy. Level 1 studies even indicate that combination therapy is the most effective technique[13].

US-guided procedures: This range of interventions aims, among others: to fragment the calcium deposit (barbotage), washing the deposit with saline solution and/or local anesthetic, aspirate the calcification, injection of the subacromial bursa to reduce the risk of post-procedure bursitis[4].

Platelet-Rich Plasma (PRP): The association of PRP with conventional treatments has not shown improvement in tendon healing after arthroscopic surgical treatment[14]. Moreover, it has not proven superior to needling combined with corticosteroid injection, presenting more complications with similar clinical outcomes after two years of follow-up[15].

Table 5. Prognostic factors related to the success of clinical treatment for calcific tendinitis[12].

Negative Factors	Positive Factors
✓ Bilateral calcified deposit ✓ Location near the anterior portion of the acromion ✓ Subacromial extension ✓ Large volume of calcified deposit	✓ Gartner type III calcified deposit ✓ Lack of ultrasonographic sound extinction of the calcified deposit

Surgical Treatment

Surgical treatment of chronic CT results in higher functional outcome scores and pain reduction, comparable to non-surgical interventions such as US-guided needling. Both operative and non-operative treatment modalities lead to clinically significant improvements in function and pain. Therefore, it is advisable to first attempt less invasive US-guided procedures and ESWT as primary treatments due to their lower morbidity[16].

Complications

Treatment aims to restore the patient's functionality. Managing pain, preventing contractures and maintaining quality of life by reducing suffering are the goals during the progression of the disease. The complications with the greatest clinical impact are pain, adhesive capsulitis, ruptures of the affected tendon, osteolysis of the greater tuberosity and tendonitis[10].

Pain

It is the initial marker of the resorption process and is acute and intense. Special attention should be given to patients with refractory pain, requiring regular clinical and radiological follow-ups[1].

Pain may result from: chemical irritation of the tissue by calcium, pressure on the tissue due to its swelling, impact-like pain caused by bursal thickening or irritation from the deposit itself and chronic glenohumeral joint

stiffness due to prolonged voluntary immobilization to avoid irritation from abduction and elevation[17].

Adhesive Capsulitis

It is a common complication in conditions affecting the glenohumeral joint. If the deposit is in a liquid state, the acute phase presents with severe pain as the main symptom. If the calcification is dry and hard, a chronic form is typically observed, where pain is gradually replaced by fibrosis and stiffness, leading to limited range of motion and secondary freezing of the joint, which is the most functionally limiting sequel[1].

Tendon Rupture

CT can coexist with tendon injuries. Observations indicate that ruptures were associated with smaller deposits. Rotator cuff integrity, rupture pattern, shape, location, and gender were significantly related to the texture of the calcified deposit[18].

Osteolysis

It is an extremely rare complication of CT, associated with prolonged symptoms and greater functional impairment. Osteolytic lesions of the tuberosities may contribute to symptom chronicity. In such cases, prognosis is worse, and patients may be resistant to common conservative therapies, necessitating surgical treatment.

Ossifying Tendinitis

It is a very rare complication of CT, characterized by heterotopic ossification and hydroxyapatite crystal deposition in a mature lamellar bone histological pattern. This often requires surgical treatment[1].

Discussion

The factors influencing the onset of the reabsorptive phase are not fully known and established in the literature. It is possible that internal and/or external factors trigger the deposition site, generating a process of local neovascular sprouting with reactive inflammatory input stimulating phagocytic cells to promote local "cleaning".

The natural course of calcific tendinitis involves a period of hibernation, followed by a phase of intense pain leading to deposit resorption. During the inert phase, the calcifications appear amorphous, surrounded by acellular and avascular fibrocartilaginous tissue, effectively "hiding" them from the immune response. Natural processes or actions that aim to aggravate, irritate, or fragment the calcification should lead to its disappearance by breaking the natural insulation, triggering an inflammatory, resorptive, and reparative process[19].

One theory suggests that apatite crystals themselves can induce an inflammatory reaction, leading to resorption. This process involves multinucleated giant cells resembling osteoclasts, IL-1 family cytokines (e.g., IL-1β and IL-18), and the NLRP3 inflammasome. Macrophage biopsies containing minerals have shown a neovascularization pattern interspersed with calcified tissue. Additionally, multinucleated cells express cathepsin K and tartrate-resistant acid phosphatase (TRAP) on their surfaces, both of which are involved in bone resorption[19].

This report describes a previously asymptomatic patient but with a history of contralateral calcific tendinitis years prior. She had a calcification in the left supraspinatus that, after a low-energy trauma, developed disproportionate pain and dysfunction. Radiological evaluation on the day of the event already showed the calcification. Over time, spontaneous resorption of the deposit occurred without direct medical intervention.

Considering the "protective capsule rupture" theory, the trauma may have initiated the resorptive phase by triggering a local inflammatory/irritative process. Although we cannot yet demonstrate the exact pathophysiological mechanisms involved, the coincidence of events, supported by evidence-based medicine, suggests that external factors-along with individual/disease-related factors-may act as triggers for the resorption and healing process in calcific tendinitis of the shoulder.

At six weeks, the patient had controlled pain without regular medication use. She underwent physical rehabilitation to restore function and ultimately achieved resolution of symptoms without complications.

Considerations

The pathophysiology of calcific tendinitis is not fully understood, from its formation to resolution. However, it is generally a self-limiting condition that naturally resolves, although the resorptive phase is marked by acute and intense pain, requiring adequate analgesia to prevent complications. The use of US-guided interventional techniques for both diagnosis and treatment is increasing. Pain control during

the resorptive phase is crucial to prevent suffering, chronic pain, periarticular fibrosis, and contractures. Surgical management is reserved for refractory cases. The triggers for resorption remain unknown, but external irritation of the calcium deposit may initiate inflammation, promoting healing through neovascularization. In this case report, shoulder trauma may have "broken the protective capsule," leading to a healing process that was managed with analgesia and rehabilitation, ultimately restoring functionality.

Authors' Contribution: MF: Conceived and planned the activities that led to the study, wrote the paper, participated in the reviewing process, approved the final version; IAWL: participated in the reviewing process, approved the final version.

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