Review

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Calcified tendonitis of the rotator cuff. A review of this common shoulder pathology

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Abstract

Calcified tendonitis is an acute or chronic painful condition due to the presence of calcium phosphate crystals within or around the tendons that form the rotator cuff.

The precise mechanism leading to the deposition of calcium crystals remains unclear. Factors that appear to contribute to this pathology are metabolic diseases (diabetes, thyroid hormone abnormalities), medication factors (e.g., corticosteroids), genetic predisposition of the individual, and overuse injuries.

Clinically, calcified tendonitis is usually asymptomatic. However, in some cases it is characterized by severe pain, which is more addressed in the morning. Many times, the intensity of the pain is so high that it inhibits movements, resulting in stiff shoulder joints. A simple x-ray is the most appropriate imaging method to diagnose the disease. As far as concerns the course of the disease, in many cases, an automatic resorption of calcium is observed while the symptoms recede. The precise mechanisms associated with absorption are unknown.

Numerous treatment options have been reported in the literature that are applied on a case-by-case basis and vary in efficacy.

Keywords: Calcified Tendonitis, Shoulder Stiffness, Shoulder Treatment, Rotator Cuff, Shoulder Pain.

Introduction

In 1872, Duplay was the first who described calcified tendonitis as a "painful shoulder periarthritis" [1]. In 1934, Codman was the first time who found that deposits were growing either inside or around the tendon of the rotator cuff, contrary to the earlier theory that the deposits had been developing in the subacromial bursa. [2] (**Figure 1**)

Shoulder tendonitis is the most frequently occurring painful disorder, characterized by the presence of calcified deposits in the rotor cuff tendons. The tendon of the supraspinatus muscle is the one mainly affected; rarely we find these problems in the tendon of the infraspinatus and the subscapularis muscle (**Table 1**) [3,4].

The prevalence of the disease is 2.7% in people without symptoms. It mainly attacks people aged from 30 to 60 years. Women appear to have the dis-

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ease at a higher rate than men. Indeed, contrary to what is expected, the disease mainly attacks people who do sedentary work [5,6].

Calcified tendonitis is treated with the use of non-steroidal anti-inflammatory (NSAIDs), ultrasound-guided needling (UGN), physiotherapy, subachromial outburst, and extra-corporeal shock wave therapy (ESWT) [7,8]. In cases where conservative treatment does not work, surgical removal of calcium deposits is recommended [9,10].

Histopathology

According to Uhthoff, the evolutionary course of calcified tendonitis is divided into three stages: the pre-calcific, the calcifying, and the post-calcific [11].

In the pre-calcification stage, numerous factors stimulate the transformation of tendon cells into chondrocytes. The one that follows, the calcification stage, is subdivided into three different phases: the formation, the phase of rest and the absorption one. In general, the disease progresses with a pattern. Initially, amorphous calcium phosphate is deposited. Then, angiogenesis is developed and the calcium crystals are absorbed and the collagenosis of fibroblast damage takes place during the post-calcification stage [11].

Samples collected from the affected area either had the form of sandy hard mass or liquid-like toothpaste, or were an amorphous mass consisting of many small round or oval particles [12]. The material of these deposits, it is recognized as calcium carbonate (apatite) [13]. Carbonate apatite has been further classified as type A and B. Classification is based on positions occupied by carbonate ions in the molecule [14]. Studies on the chemical composition of calcification have found that, in different proportions, both types of apatite carbonate coexisted during the formation, resting and absorption phases [15].

Histochemical studies have shown the presence of extracellular vesicles, also known as microvesicles (MVs), in the area of the rotator cuff calcification. MVs vesicles are normally required for the formation of cartilage, bone and dentin. The attempt to correlate the specific finding with the pathogenesis of calcifying tendonitis has led to discovering that the deposition of calcium crystals is the result of an active and complex process involving the participation of alkaline phosphatase, proteins and enzymes, [11,16,17]. However, the expression of alkaline phosphatase and the presence of MVs are controversial in the international bibliography. On the other hand, it has been shown that despite osteopontin (OPN) is found in the formation phase its role has not been yet clarified. However, IL-1 and IL-18, as well as the inflammatory factor NLRP3, appear to play a critical role in absorption [16, 17]. The exact mechanism of the specific pathology has not yet revealed its data and thus all the information has not been clarified in its whole.

Clinical symptoms

The main clinical symptom is pain. Pain, in this condition, is considered to be a complication, as the latter remains primarily asymptomatic in most cases [18]. When calcified tendonitis becomes symptomatic, pain is acute and usually does not reflect below the middle part of the arm [19]. In the acute phase, the disease is so painful that it restricts shoulder's mobility. In the chronic or subacute phase, the pain may be acute but generally allows movement [20].

The occurrence of pain is due either to the inflammatory response to local chemical pathology or to direct mechanical irritation [21]. Four mechanisms that stimulate the onset of pain have been described: the chemical irritation of the tissue by calcium, the pressure of the tissues due to swelling, the impact on the subacromial bursa due to deposition and the chronic stiffness of the glenohumeral joint in a patient's effort to avoid severe pain [22].

Peptide P (neurotransmitter) is involved in the transmission of pain caused by Aδ / C fiber stimulation. It is expressed in the small aesthetic neurons of the tissues of the affected area. It

Is released from sensory neurons and plays an important role in mediating neurogenic inflammation [23]. Researchers who studied the relation between the amount of P in the subacromial bursa and the pain in patients with calcified tendonitis, have discovered an increase in the number of immunoreac-

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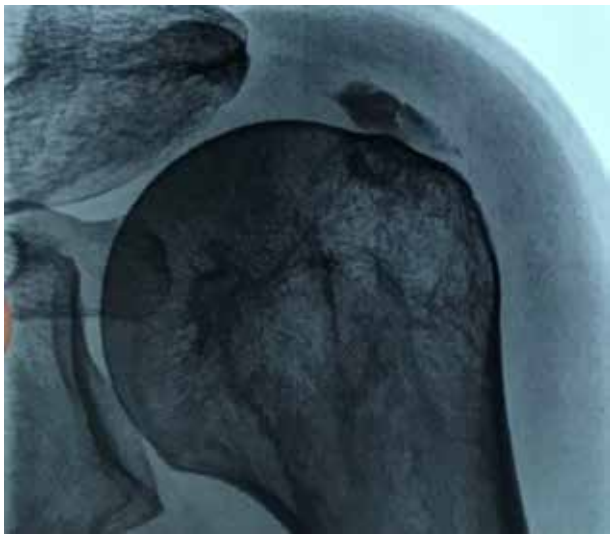

Figure 1. *Shoulder plain x-ray showing calcification of the distal end of supraspinatus tendon.*

tive nerve fibers in the tissues around calcification. These fibers were mainly located around the blood vessels, suggesting an active role of the peptide in regulating subsequent inflammation. They also hypothesize that both the mechanical (subacromial impingement) and the chemical agents (subacromial bursitis) could be a source of harmful stimuli, which cause increased amounts of substance P in the affected nerves. Therefore, it is found that inflammation of the subacromial bursa is the complication associated with pain of the shoulder girdle in patients with calcified tendinitis [23].

Imaging

The preferred imaging methods for the identification of calcified tendinitis is plain radiography (Xray) and ultrasound (US), mainly because calcium deposits are easily recognizable in both of the processes. In X-rays, calcium deposits appear to be homogeneous, amorphous, without rough distribution, allowing the differentiation from ectopic osteogenesis [24]. Ultrasound (US) is particularly effective for the diagnosis of calcified tendonitis, as it detects other pathological conditions that co-exist in the area (e.g. tendon injuries) [25]. High resolution ultrasound in combination with color Doppler allows the distinction between the state of formation and the absorption of calcium [26].

Figure 2. *Needle aspiration of soft calcification of infraspinatus tendon during the initial inflammatory phase. The location of the calcification is identified easily above the skin by palpation of a bulky painful inflammatory area or by U/S guidance. (I.K.T. personal archive)*

On the other hand, computed tomography offers excellent resolution for detecting calcium deposition as a compact or amorphous high-density hob; however, its cost and exposure limit its use. Finally, magnetic tomography should not be used as a first imaging form because the deposits appear as vague low signal regions at T1 and T2 and may not be recognized. In general, CT and MRI should only be selected for controversial cases [27].

Treatment

Conservative management of patients with calcified tendonitis of the shoulder is always the first treatment option. According to the literature, it utilizes the following means: non-steroidal anti-inflammatory (NSAIDs) to relieve pain, physiotherapy to maintain shoulder joint range of motion,

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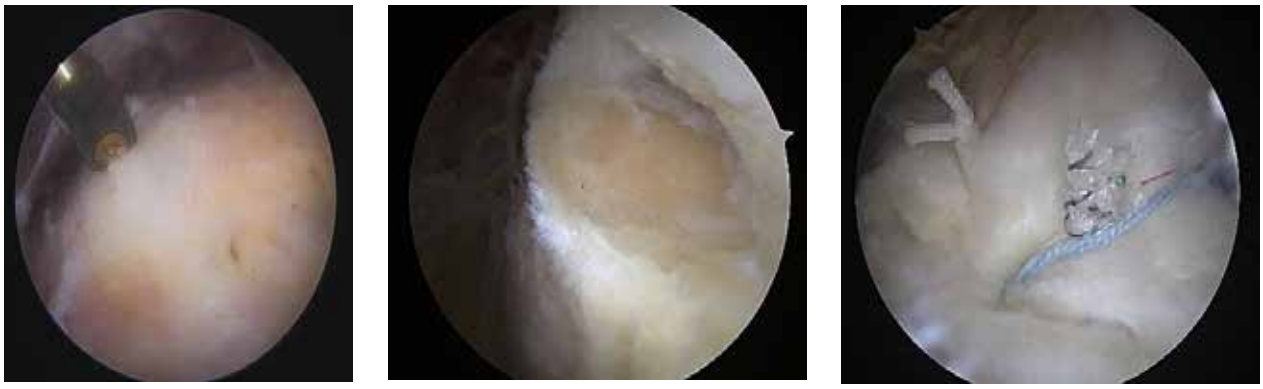

Figure 3. *Arthroscopic identification of the calcified tissue (a). Removal of the degenerated tendon (b). Repair of the tendon with re-attachment to its footprint (c) (I.K.T. personal archive)*

extracorporeal shock waves therapy (ESWT), PRP (Platelet Rich Plasma), ultrasound-guided needling (UGN) and Surgical Treatment [28].

NSAIDs

Non-Steroidal Anti-Inflammatory Drugs are commonly used as the primary choice in the acute phase of calcified tendonitis of the rotator cuff both due to analgesia they offer and for their anti-inflammatory properties. Reducing pain is a key factor for maintaining the shoulder's motion range. As far as the Non-Steroidal Anti-Inflammatory Drugs concern, there is some thought give on the fact that they may adversely affect healing of the tendon-bone adhesion [29,30].

Determining the adverse effects of non-steroidal anti-inflammatory drugs, in terms of healing proves to be rather complicated; this is a mixture of interconnections between the mechanisms found in cellular level, including inhibitors of cyclooxygenase-1 (COX-1), the cyclooxygenase-2 (COX-2) and ways they are affected by the time of administration of the drugs [31].

The existing bibliography does not fully clarify whether the use of non-steroidal anti-inflammatory drugs adversely affects healing or not.

Corticosteroid injections

Cortisone injections may be applied mainly subacromialy and rarely intra-articularly. It should be noted that steroid infusion during the acute phase is often chosen. However, the works on this subject reflect the ambiguity of scientific community. Some studies showed that this method has positive results [32], other works label it as ineffective [33] while it has also been mentioned that it negative consequences because absorption of deposits is inhibited [34].

Finally, surgical removal is selected only in cases where conservative treatment will not produce the desired results.

Physiotherapy

The main role of physiotherapy in calcified tendonitis of the rotator cuff is to reduce pain in the affected and peripheral area of the lesion, as well as to maintain the range of motion of the shoulder joint.

Shock waves in the treatment of the disease are described below so no reference will be made to this chapter.

Passive mobilization by a physiotherapist or a continuous passive movement machine (CPM) device during the acute phase is an important tool for maintaining range of motion.

Myofascial therapy and trigger point therapy play an important role in reducing pain. [35] Natural remedies such as LASER, microwave diathermy, and TENS significantly help reduce pain and expand range of motion. In the second year, it is really important to strengthen the muscles of the shoulder girdle to maintain shoulder stability. [36]

It is significant to train the patient to avoid movements that cause subachromial friction, as well as to learn exercises that maintain the range of motion.

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Table 1. The frequency of calcification in each tendon of the rotator cuff	
Prevalence rates of calcification in the anatomical structures of the rotator cuff [4]	
Supraspinatus tendon	63%
Supraspinatus Subscapularis tendon	20%
Subscapularis tendon	3%
Infraspinatus tendon	7%
Subacromial Bursa	7%

Ultrasound-Guided Needling and Decompression

Ultrasound-guided needling was first performed using a radiograph by Comfort et al. [37]. Farin et al. first attempted to use ultrasound for saline wash [38]. (**Figure 2**)

Ultrasound-guided needling (UGN) is a treatment that can be performed easily with local anesthesia and at a low cost. In 2014, a study was published that involved 121 patients who applied the UGN technique. It was found that within three months of applying the method the results were satisfactory [39]. In a 2013 study, patients were divided into two groups, the first of which used UGN wash and corticosteroid infusion, and the second only corticosteroid infusion. Both groups showed improvement, but patients in the first group performed better [40]. However, a more recent comparative study concluded that the two groups had no significant statistically variance [41].

A systematic review of the bibliography on the efficacy of the UGN method in calcified tendonitis of the shoulder shows that studies do not provide strong evidence for the efficacy of the method. In addition, there is an extensive variation in the results, indicating that more reliable studies is a necessity [41]. In another systematic review, the UGN method is suggested as an alternative treatment when the initial conservative treatment has failed [42]

Extracorporeal Shock Waves Therapy

Extracorporeal shock waves therapy (ESWT) has been used to treat calcified tendinitis since the 1990s. This method is becoming increasingly popular and new research is being published on its dosing, duration and the number of sessions. Shock waves therapy can be generated by electromagnetic or piezoelectric devices. The categorization of shock waves in relation to energy appears as follows:

(a) low energy (below 0.08 mJ / mm2)
(b) medium energy (0.08-0.28 mJ / mm2)
(c) high energy (0.28-0.60 mJ / mm2) [43].

In a research study in which the control group received placebo treatment, it was found that the results were better in the shock wave treatment group [44]. Various doses of ESWT energy have been indicated occasionally for the treatment of calcified tendonitis, and most authors have described positive clinical results with low and medium energy waves [43,47,48]. In a study comparing a dose of 0.3 mJ / mm2 versus two doses of 0.2 mJ / mm2, the former proved to be more effective [44]. Another study revealed that a dosage of 0.20 mJ / mm2 is more effective than 0.10 mJ / mm2 [45]. Albert et al. found positive results with high doses of ESWT. However, in this case the follow-up of patients lasted only three months and no significant decrease in the size of the deposition was observed on radiographs [46].

A comparative study between the UGN and ESWT methods showed that the UGN technique had better radiological and clinical results, although both groups showed improvement compared to the original findings [49]. In a 2018 research study, 66 patients with LCT were given a needle-guided xylocaine infusion followed by a five-session ESWT session over a month. The results showed clinical improvement in patients and disappearance of deposition at the end of the fifth treatment [50].

PRP (Platelet Rich Plasma)

The use of PRP in the treatment of calcified tendonitis of the rotator cuff, based on the existing literature, does not appear to have positive results [51] although it should be noted that current research so far is not as extended as need.

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Surgical Treatment

Arthroscopic treatment of rotator cuff calcified tendonitis has been described by many researchers as a treatment with positive results [52,53,54,55] (**Figure 3**) However, it is a fact that many heterogeneous techniques have been used as well as short-term comparative studies that have not convinced the scientific community which is the best technique that can effectively tackle the problem.

In the international bibliography there is a controversy as far as concerns the need for complete removal of calcium. Several researchers appear to associate successful outcomes of surgery with complete absence of calcareous tendons [57,58,59]. However, the view that total removal of calcium deposits is not necessary is strongly supported, since cellular absorption begins from the surgical incision site of the affected tendon. Several studies even confirm that there was no difference in functional outcome between patients with calcified residues and patients who had been fully calcified [54,55,56].

The issue of surgical repair of the tendon also causes controversy. Researchers argue that the tendon should not be repaired in all cases, as it results in natural healing [57]. However, researchers who studied a group of patients who underwent complete removal of calcium deposits without tendon repair after 24 months of ultrasound monitoring [56] found that 31% of those patients developed defective healing in the rotator cuff.

The necessity of acromioplasty is another controversial issue regarding the surgical treatment of shoulder calcified tendonitis. Many studies have considered acromioplasty as a routine treatment for rotor pedal calcifications [60,61]. However, other studies have not confirmed the benefit of additional subachromial decompression [52]. Marder et al. performed a comparative study between a group of 25 patients who underwent arthroscopic calcification, and a group of 25 patients who underwent calcification and simultaneous acromioplasty. Finally, acromioplasty was found beneficial because it delayed both the elimination of pain and ultimately the recovery [63].

Conclusion

Calcified tendonitis of the rotator cuff, although commonly found in clinical practice, has many areas yet to be discovered in terms of the factors that play a significant role in its formation. At the same time, there is great controversy about the most appropriate therapeutic method to achieve full functional recovery of the patient.

Conflict of interest

The authors declare no conflicts of interest.

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