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MINIREVIEWS

Calcific tendinitis of the rotator cuff

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Abstract

Calcific tendinitis within the rotator cuff tendon is a common shoulder disorder that should be differentiated from dystrophic calcification as the pathogenesis and natural history of both is totally different. Calcific tendinitis usually occurs in the fifth and sixth decades of life among sedentary workers. It is classified into formative and resorptive phases. The chronic formative phase results from transient hypoxia that is commonly associated with repeated microtrauma causing calcium deposition into the matrix vesicles within the chondrocytes forming bone foci that later coalesce. This phase may extend from 1 to 6 years, and is usually asymptomatic. The resorptive phase extends from 3 wk up to 6 mo with vascularization at the periphery of the calcium deposits causing macrophage and mononuclear giant cell infiltration, together with fibroblast formation leading to an aggressive inflammatory reaction with inflammatory cell accumulation, excessive edema and rise of the intra-tendineous pressure. This results in a severely painful shoulder. Radiological investigations confirm the diagnosis and suggest the phase of the condition and are used to follow its progression. Although routine conventional X-ray allows detection of the deposits, magnetic resonance imaging studies allow better evaluation of any coexisting pathology. Various methods of treatment have been suggested. The appropriate method should be individualized for each patient. Conservative treatment includes pain killers and physiotherapy, or "minimally invasive" techniques as needling or puncture and aspiration. It is almost always successful since the natural history of the condition ends with resorption of the deposits and complete relief of pain. Due to the intolerable pain of the acute and severely painful resorptive stage, the patient often demands any sort of operative intervention. In such case arthroscopic removal is the best option as complete removal of the deposits is unnecessary.

Key words: Rotator cuff; Calcific tendenitis; Prevalence; Pathogenesis; Natural history; Classification; Clinical picture; Imaging; Treatment

Core tip: This review article discuss calcific tendinitis of the rotator cuff regarding the definition, prevalence, pathology, pathogenesis, natural history, clinical presentation, classification, diagnosis and various treatment modalities.

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INTRODUCTION

Calcium deposits within the rotator cuff tendon is a common shoulder disorder[1]. Calcium deposits may be in the form of calcific tendinitis or dystrophic calcification.

Calcific tendinitis is calcification within a viable and well vascularized rotator cuff. It occurs within the midsubstance of the cuff, 1 to 2 cm proximal to its insertion. Classically, the condition will end by spontaneous resolution and it is uncommon to see other signs of degenerative changes[2].

Dystrophic calcification is calcification within a non-viable and poorly vascularized rotator cuff. It occurs at the insertion site or at the edges of a cuff tear. Classically, the condition worsens by time and it is common to see other signs of degenerative changes[2].

PREVALANCE

Calcific tendinitis usually occurs during the fifth and sixth decades of life. It occurs within the supraspinatus tendon in almost 50% of cases. It is more common in females (60%). It occurs more commonly with sedentary workers than with heavy labor workers (45% are house wives)[3-5].

PATHOGENESIS AND NATURAL HISTORY

Controversy exists over the exact cause of calcific tendinitis. Burkhead[6] and Gohlke[7] proposed that it is a degenerative process that involves necrotic changes of tendon fibers that progress into dystrophic calcification.

Mclaughlin[8] believed that it proceeded from focal hyalinization of the fibers that become fibrillated and detached from the tendon, thus wounding up into rice-like bodies that later undergo calcification.

On the other hand, Uhthoff[9] pointed out that this may be true regarding dystrophic calcification. But regarding calcific tendinitis they believed that this explanation was most unlikely. They argued that calcific tendinitis occurred in viable and well vascularized tissues and thus could not be a degenerative process, instead they suggested that it was a reparative process progressing through a predictable disease cycle[9-12].

The calcium deposits may have a chalk-like consistency or a fluid consistency or a mixed one[13].

CLASSIFICATION AND CLINICAL PRESENTATION

Many classifications have been suggested to describe calcific tendinitis. Some classified it according to the severity of the symptoms into acute, subacute and chronic[14]. Others classified it according to the radiological form into two categories. The first with localized, discrete, dense and homogenous deposits with spontaneous healing tendency and the second with diffuse, fluffy and heterogeneous deposits characterized by delayed and slow healing[15]. The French society of arthroscopy divided the condition into four types: Type A (20%) with homogenous deposits with well defined edges; Type B (45%) with heterogeneous fragmented deposits with well defined edges; Type C (30%) with heterogeneous deposits with ill defined edges and Type D which is not calcific tendinitis but degenerative dystrophic calcifications at the rotator cuff insertion[16].

Uhthoff *et al*[12] were the ones who described the complete cycle of the calcium deposits and explained the development of its natural history. They divided the condition into formative and resorptive phases. Lying within the two phases most authors motioned the presence of three stages; pre-calcification (silent), calcification (impingement) and post-calcification (acute)[17-20].

The chronic formative phase results from transient hypoxia that is commonly associated with repeated microtrauma and sometimes with a significant single trauma. This results in increased proteoglycan levels that induce tenocyte metaplasia into chondrocytes. This is followed by calcium deposits, mainly into the matrix vesicles within the chondrocytes. These deposits develop into bone foci that later coalesce.

During the acute resorptive phase the periphery of the calcium deposits shows vascularization with macrophage and mononuclear giant cell infiltration together with fibroblast formation. This produces an aggressive inflammatory reaction with inflammatory cell accumulation, excessive edema and rise of the intratendineous pressure. This leads to severe pain which is attributed by some to secondary impingement resulting from the increased tendon size, or due to rupture of the deposits into the subacromial space or into the bursa.

During the post-calcification stage the fibroblasts lay down collagen (mainly type Ⅱ) that fills the gap. This will maturate into collagen type Ⅰ within 12 to 16 mo[9,11,12].

The clinical presentation is highly variable and depends on the phase the patient is passing through. During the chronic formative phase that may extend anywhere from 1 to 6 years, the patient may be completely asymptomatic. In some cases the condition will only be discovered accidentally. Some patients may present with symptoms that mimic mild impingement. However during the acute resorptive phase, the patient usually presents with severe symptoms that may extend from 3 wk up to 6 mo. In general, the more severe the symptoms are, the shorter the duration of the condition is. The patient presents with tremendous pain all over the shoulder with tenderness over the supraspinatus insertion. Pain commonly extends to the root of the neck

with difficulty during overhead activity associated with muscle spasm. It is very difficult to perform any of the special tests due to the unbearable pain.

IMAGING

Radiological investigations confirm the diagnosis and may even make the diagnosis in asymptomatic cases. It also suggests the phase of the condition and is used to follow its progression.

They include conventional X-ray in true antero-posterior, lateral and outlet views. Deposits within the subscapularis may be detected by anteroposterior view in external rotation. In internal rotation, the deposits within the infraspinatus and teres minor may be detected. "Skullcap appearance" indicate rupture of the deposits within the bursa[21].

Ultrasonographic examination was reported to be more sensitive in detecting the calcium deposits within the cuff[22].

Computed tomography allowed better localization of the deposits[21]. Although routine conventional X-ray allowed detection of the deposits, magnetic resonance imaging (MRI) studies allow better evaluation of any coexisting pathology. The deposits present with a low intensity signal in the T1 weighted images. In the T2 weighted images there may be perifocal low intensity signal denoting surrounding edema[23].

The thinned out cuff lateral to the deposits may be falsely interpreted as a cuff tear. MRI arthrography was more beneficial to avoid such false conclusions[23].

TREATMENT

Various methods of treatment have been suggested. The appropriate method should be individualized for each patient depending on proper understanding of the pathophysiology and natural history of the condition, as well as proper clinical and radiological assessment of the patient, and finally accurate determination of the stage at which the patient presents.

The treatment may be "conservative" including pain killers and physiotherapy, or "minimally invasive" as needling and puncture and aspiration, or "operative" whether arthroscopic or open.

Due to the intolerable pain of the acute and severely painful resorptive stage, the patient often demands any sort of intervention despite explaining to him that the condition is probably resolving.

Since the natural history of the condition ends with resorption of the deposits and complete relief of pain, usually conservative measures are successful in most of cases, reaching 80% in some studies and even 99% in others[12,24].

During the acute stage the aim is to relief of pain. The efficacy of non-steroidal drugs may be doubtful with frequent need to narcotic medications.

Physiotherapy

Some authors suggested physiotherapy including range of motion exercises to avoid gleno-humeral stiffness and idiopathic frozen shoulder. However there is no evidence that calcific tendinitis causes gleno-humeral capsular contracture[25].

There is no solid evidence that different physical modalities including infrared, ultrasound, or deep heat have any effect on the natural history of the condition.

Extracorporeal shock wave

Extracorporeal shock wave (ECSW) has been used to treat symptomatic patients passing through the chronic formative phase with definite radiological evidence of calcium deposits[26].

Most authors report short term symptomatic improvement[27]. But ECSW was not free from complications, that included transient bone marrow edema and even reported cases of humeral head necrosis[28,29].

Most authors reported that the improvement is dose dependant, with better results following one or two sessions of high energy applications[30].

Needling or puncture and lavage

Minimally invasive techniques include needling or puncture and aspiration. These techniques were suggested by many authors aiming at decompressing the deposits and thus relieving the pain. They suggested that direct puncture of the deposits would shorten the natural history of the condition and accelerate resorption in 50% of cases[31].

Since the fifties of the last century some authors recommended blind needling of the deposits with intralesional local anesthetic injection reporting pain relief in 85% of cases. They reported that the amount of deposits removed didn't affect the outcome and accordingly concluded that pierce opening of the deposits was the essential step and not the calcium removal[32,33]. In the sixties Depalma and Kruper[14] popularized blind needling of the deposits without any radiological localization, with good results. Clement reported pain relief within 24 h following repeated blind needling of the deposits (15 to 20 times), after local anesthetic and corticosteroid injection into the subacromial space. He referred the patients to ultrasonic treatment within a few days. He claimed that this would cause active hyperemia that would enhance deposit absorption[34]. Most authors reported very good results after performing needling under fluoroscopic and, or ultrasonographic guidance[35,36].

Local corticosteroid injection (whether intralesional or into the subacromial space) following the needling of the deposits, is recommended by some authors with good results and some suggested two or more injections. Many studies showed no evidence that corticosteroid injection improved the results[36,37]. Some reported that corticosteroids injection was short acting and only symptomatic. Other surgeons argued that corticosteroids

would reduce the tendon healing process[38]. Neer[39] disagreed with this claim.

Many authors recommended dual needling and lavage for cases with calcium deposits[40-42]. Needling is no new technique as it has been described over a century ago by Flint in 1913 as reported by Codman in 1934[6].

After all of the various needling techniques the patient should be instructed to rest the shoulder for a short period (1 to 2 d) followed by gradual return to daily activities.

The patient should also be forewarned that a successful full recovery may take 3 to 6 mo. During the chronic formative stage the symptoms are usually mild and no intervention is needed. Yet some authors suggested needling (whether blind needling or under radiological guidance) suggesting that direct puncture of the deposits would shorten the natural history of the condition and accelerate deposits resorption[43]. Non-steroidal drugs may be used every now and then.

The true debate concerning needling or puncture and lavage is the fact that the acute and severe symptoms are almost always associated with an expedient resolution of the condition. Thus, any form of treatment at this point will ultimately be a "success".

Operative intervention

Indications: Many authors suggested that the indications for operative intervention include progression of symptoms that interferes with the daily activities after failure of conservative measures[44,45].

Neer stated that operative indications include long standing symptoms after failure of conservative measures in the presence of multiple, hard and gritty deposits. He rarely resorted to operative intervention and suggested that residual tendinopathy would follow[39].

Most authors starting from Burkhead[6], passing through Lippmann[1], to Mclaughlin[8], and up till today[45,46] agree that surgical removal of rotator cuff calcium deposits end with good permanent results. They agree that the indications include symptomatic patients after failure of conservative measures with radiological evidence of relatively homogenous calcium deposits.

During the resorptive stage, conservative measures were recommended as the natural history of the condition would end with complete resolution of the deposits and the symptoms. Yet operative intervention is to be considered upon the patient's demand due to the intolerable pain despite the conservative measures.

Open surgery: It is performed through a deltoid splitting incision, the site of which may be modified to allow the best access to the exact location of the deposits. The deltoid fibers are separated and the deposits are split-open along the direction of the cuff fibers. Usually the deposits are readily apparent as a bulge within the cuff. The deposits commonly burst out when opened. Open surgery has a high success rate in complete removal of the calcium deposits but with some intraoperative complications[47-49]. Some authors suggested resuturing of the cuff if a significant gap is left behind. The benefit of this step is unclear[13,50]. This is followed by a period of shoulder rest (5 to 7 d), with gradual return to daily activities over a 4 to 6 wk period of time.

Most authors reported that complete intraoperative removal of the deposits was unnecessary as it didn't significantly affect the final clinical outcome. Thus total removal was not essential and sometimes not possible without substantial damage to the tendon[49,51]. In most cases partial removal of the deposits will finally lead to total resorption. This was reinforced by other studies[46,52].

Arthroscopic management: Nowadays, open surgery is rarely used to remove calcium deposits as arthroscopy offers a much better choice.

Arthroscopic calcium deposits removal was quite effective, although it may fail to completely remove the deposits compared with open surgery[47-49]. But as long as complete removal of the deposits was unnecessary, then arthroscopic removal was clearly a better option. Studies showed that the rate of full-thickness rotator cuff tears after calcium deposits removal was quite low (3.9% after a 9-year follow-up)[53]. Accordingly, rotator cuff repair following calcium deposits removal was not mandatory. However, it was found that the intraoperative status of the rotator cuff had a significant influence on the functional results at follow-up[47]. In one study, the 2 patients of the 54 cases of the study (3.7%) who needed later rotator cuff repair showed obvious degeneration of the rotator cuff during the removal of the deposits. Accordingly, it should be recommended to repair the rotator cuff after the removal of calcium deposits, whenever the cuff appears to be noticeably degenerative[46].

Arthroscopic subacromial bursectomy should be performed to allow better visualization of the rotator cuff. In cases with shoulder impingement, subacromial decompression (acromioplasty) should be performed. The calcium deposits were identified as a bulge within the cuff tendon "calcific bulging sign"[51]. Then, *via* a lateral working portal, a half-moon arthroscopy knife may be used to open up the deposits along the fibers of the cuff. After that, a 3.5-mm motorized shaver was used to remove as much as possible of each deposit, only stopping short of causing any iatrogenic damage to the cuff.

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