

Chapter

Calcific Tendinitis: Limited Role of Surgery

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Abstract

Calcific tendinitis is not an uncommon condition, although many patients may experience no symptoms, and calcification was found accidentally through imaging studies. Even so, in some cases, calcific tendinitis may arise with bothersome symptoms that can lead to diminished function of the affected joint. Calcific tendinitis is usually a self-limiting disease, where in its course, it may resolve on its own, may not need further interventions. In symptomatic cases, conservative treatment is the main option. More aggressive treatment such as percutaneous lavage may be needed in acute or unresponsive chronic pain. Surgical intervention may be needed to help resolve the symptoms, but it is rarely indicated.

Keywords: calcified tendinitis, surgery, arthroscopy, ultrasonography, lavage

1. Introduction

Calcific Tendinitis (CT) is a condition of abnormal deposition of calcium salt inside of the tendon. It is also known as calcific periarthritis, which implies that the calcification is not happening within the joint, and calcifying tendinitis, describe the transient nature of the disease [1, 2].

It may occur in many regions of the body, but it is mostly found in the shoulder region, usually the rotator cuff in which the supraspinatus tendon is most frequently affected, followed by the infraspinatus and the subscapularis. Hip girdle region is the second most affected region, including rectus femoris and gluteus Medius tendon. CT also has been reported to occur at proximal hamstring, biceps brachii, longus colli, Achilles, flexor carpi ulnaris, and many other sites [1].

Number of incidences varies between 2.7 to 22% in individuals without symptoms detected by X-Ray with bilateral incidence in about 10–20% of cases [3–5]. A study conducted by Bosworth et al. in 1941 found incidence of 2.7% out of 6061 patients have CT in which 34.7% were symptomatic [4]. A newer descriptive study reported the incidence of calcific tendinosis in the shoulder can be found in 7.8% of asymptomatic patients and 42.5% of patients with subacromial pain syndrome [6]. Both studies have shown that CT affected more women than men, in the age group of 30 to 60.

2. History

Duplay was accredited as the first to describe CT of the shoulder in 1872. He defined it as “painful periarthrititis of the shoulder” [7]. Later, Painter rendered the first case report of calcific deposit about the shoulder in 1907, misinterpreting it as thickening of the walls of the bursa [8]. This was followed by other authors who proposed the accumulation of scar tissue [9], hemorrhage under pressure [10] and metamorphosis of fat deposits [11], which suggest subacromial bursa pathology.

In 1908, Codman reported the surgical removal of deposits composed chiefly of calcium, in or on the supraspinatus tendon. In his classic textbook on the shoulder (1934), he then made the following definitive statement: “The deposits do not arise in the bursa itself, but in the tendons beneath it.” The critical area, a concept of there being a specific vulnerable area in the supraspinatus tendon susceptible to calcification was first proposed by Codman [12]. Other authors in agreement like Bishop and Sandstrom suggested that some kind of degeneration of the rotator cuff due to overuse or ischemia leads to calcific deposits in the tendon. The process may begin with necrosis of tenocytes along with intracellular accumulation of calcium [13, 14]. This view was then supported by Refior in a later paper, using cadaveric study [15].

In contrast, Uhthoff and Loehr think that the transient, self-healing nature of calcifying tendinitis did not fit the degenerative disease characteristic. They proposed the multiphasic disease theory, which suggests that deposition of calcium in the tissues will be followed by spontaneous resorption of the calcific deposit. They believe that the process of calcification is actively mediated by cells in a viable environment [16]. This view becomes more prevalent in recent years, with imaging studies and classifications adopting different stages of the disease.

Most of our understanding of this disease comes from observation of CT of rotator cuff of the shoulder.

3. Symptoms

CT can present in three different clinical scenarios. It can be an asymptomatic incidental finding, a condition involving chronic low-grade pain, or a very painful acute condition that affects the range of movement and function of the joint. In shoulder, pain is often aggravated by abduction of the arm above shoulder height or by lying on the affected shoulder. Point exquisite tenderness is found in the calcific region, especially in acute phase [17, 18].

4. Investigation

Calcific deposits appear as irregular punctuate, circular, linear, or plaque-like radio-dense areas that do not possess a trabecular or cortical structure. In the rotator cuff tendons, it can be localized using the anteroposterior radiographs of the shoulder in internal and external rotation and axillary lateral radiographs. The French Arthroscopic Society using the anteroposterior view of X-rays which then becomes the widely used classification defined four types of deposits. Type-A calcifications are sharply delineated, dense, and homogenous. Type B are sharply delineated and dense in appearance, with multiple fragments. Type C are heterogeneous in appearance, with

a fluffy deposit (**Figure 1**). Type D are dystrophic calcifications at the tendon insertion. The last two types are associated with the resorptive stage of the disease [19, 20].

The second useful investigation is ultrasonographic evaluation. It is sensitive to detecting the calcium deposits in soft tissue. Although in the presence of calcifications, shadows may cause false-positive and false-negative findings, ultrasonographic is still beneficial to detect the cuff tears nearby, and concomitant impingement syndrome. On ultrasound, calcification was classified as arc-shaped (an echogenic arc with clear shadowing), fragmented or punctate (at least 2 separated echogenic spots or plaques) with shadowing, fragmented or punctate without shadowing, and nodular (an echogenic nodule without shadowing) [21]. A study reported that an arc-shaped and hyperechoic deposit indicates calcification in the resting phase, whereas on-arc shaped deposit, may be fragmented, cystic or nodular indicating the resolving phase of the calcification [22]. Bianchi and Martinelo described three types of calcifications based on the percentage of calcium content found by ultrasonography. They described Type I as hyperechoic foci with well-defined acoustic shadowing due to high calcium amount, Type II as hyperechoic foci with mild acoustic shadowing due to reduced calcium amount, and Type III as foci which is isoechoic to the tendon (**Figure 2**) [23].

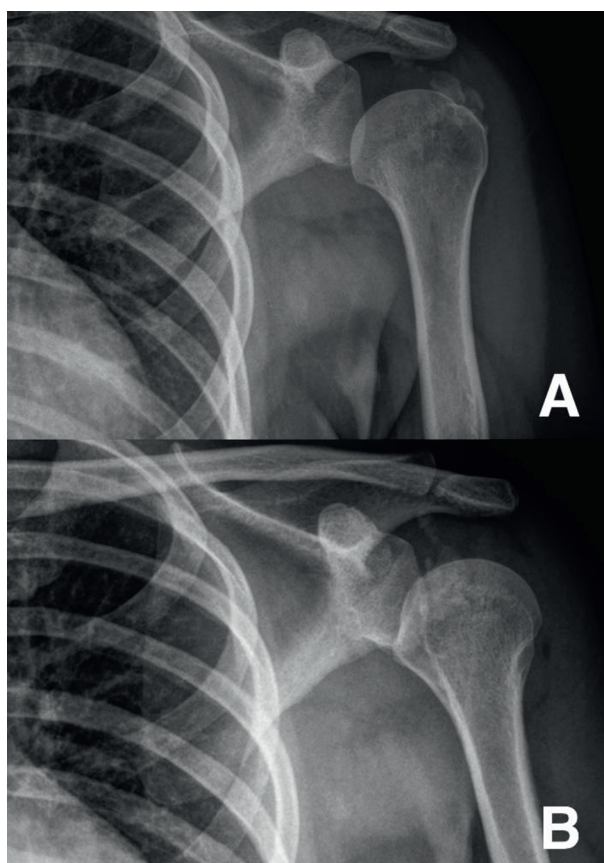


Figure 1.
Anteroposterior (A) and internal rotation (B) view of the left shoulder showing Type C calcification according to French Arthroscopic Association classification within the supraspinatus tendon of a 41-year-old woman.

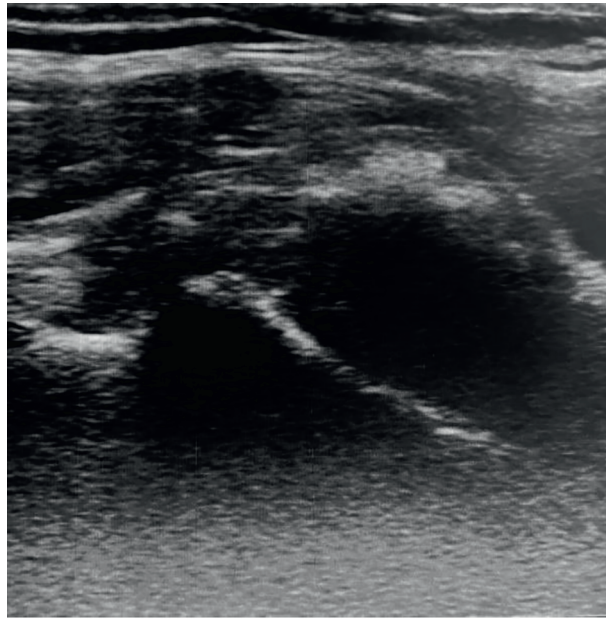


Figure 2.

Ultrasonographic of a 49-year-old woman showed a Type II calcific deposit in the subscapularis tendon according to Bianchi and Martinoli, who later underwent ultrasound-guided percutaneous lavage with dramatic improvement of acute pain.

The use of magnetic resonance imaging in cases of CT has shown to be unessential as it tells no specific additional information and does not alter the treatment plan. The deposit can be found in MRI imaging as a low-intensity lesion in the T1-weighted images. High intensity may be found in T2 sequence indicating edema around the deposit which correlates to the resorptive phase of calcification. MRI also allows better evaluation of coexisting pathology when conservative treatment fails and surgery is indicated [24].

Computed tomography allowed better localization of the deposits but is rarely indicated.

In contrast to metastatic calcification, laboratory findings are usually within normal limits. Some metabolic abnormal findings may be found as a predisposing factor to CT such as type 2 diabetes and hypothyroid conditions [16].

5. Pathomechanism

There are two most accredited proposed mechanisms for CT: degenerative, which highlights similarities with degenerative lesions of the rotator cuff, and reactive, proposed by Uhthoff and Loehr, which suggests that deposition of calcium in the tissues is a cell-mediated process that is followed by spontaneous resorption [15, 16].

The degenerative calcification theory proposes that CT is a form of dystrophic calcification of the tendon that follows an ischemic, degenerative, and necrotic phase, secondary to wear and tear, overuse and microinjury all attributable to aging. In the shoulder, it is suggested that the pathogenesis of both cuff tears and calcific tendinopathy are identical. The concept of there being a specific area in the

supraspinatus tendon susceptible to calcification and tearing was first proposed by Codman. This is supported by the observation that CT seldom affects people before the fourth decade [25–27], although studies on correlation between rotator cuff tear and CT showed conflicting results. Another objection is that CT can be observed, not only in the supraspinatus and subscapular tendon within the “avascular critical zone,” but also in the subscapularis and teres minor tendon outside the “avascular critical zone”.

In contrast, Uhthoff proposed a different mechanism, which is an active calcification followed by resorption and tendon remodeling. The reactive calcification theory involves four phases (pre-calcific, formative, resorptive, and healing). In pre-calcific phase, the fibrocartilaginous focus was developed on extracellular matrix of tendon. Matrix that will be filled with calcium in the calcific phase. In resorptive phase, macrophage and giant cell migrates surrounding the calcification site and begins phagocytosis. In the post-calcific phase, fibroblast proliferation occurs, followed by remodeling of the affected tendon. Uhthoff regards the transformation of fibrocytes into chondrocytes as the initial stage. He was not able to find any similar metaplasia in the case of rotator cuff rupture. Therefore, he concluded that CT and rupture of the rotator cuff have no common degenerative preconditions. The data shows peak at 50 years of age and fail to show that trauma and overuse as risk factor; dominant hand is not more frequent than non-dominant, and it is not more prevalent in heavy jobs. This view is also supported by a variety of imaging studies demonstrating a complete resolution of the calcium deposits. This is confirmed later by several studies [1, 28, 29]. The concept has been challenged by Refior, who was able to prove the simultaneous incidence of rotator cuff ruptures and intra-tendinous calcification in 13 of 22 cases of rotator cuff ruptures. The coexistence of CT and rotator cuff tear is not as rare as previously mentioned. A total of 28% of patients with CT revealed rotator cuff tear with arthrography study, and five of these were confirmed at surgery [30].

It has been reported in subsequent studies that endocrine disorders (thyroxine, estrogen, insulin) and genetic factors may also be related to the development of CT and affect resolution process.

Long-term data on the natural history of CT vary greatly. Gärtner et al. reported an 85% chance of natural resolution after 3 years for type III deposits, as opposed to 33% for type I and II deposits. In his classic study, Bosworth reported that 6.4% of calcific lesions showed spontaneous resorption.

Neer proposed four types of pain on CT in shoulder. The first type is characterized by pain caused by chemical irritation as a result of the calcium deposits. The second type involves pain caused by increased local pressure within the tissue as it swells. The third type causes impingement-like pain through bursal thickening and irritation by prominent calcium deposits. The fourth type reflects pain caused by chronic stiffness of the glenohumeral joint, such as frozen shoulder [31].

6. Treatment

No treatment is indicated in incidental findings of CT without symptoms.

6.1 Acute stage

This stage represents resorption phase, characterized by acute exquisite focal tenderness with restriction of joint movement, possibly from chemical irritation

of calcific deposit. This is associated with The French Arthroscopy Association radiologic type C, with fragmented or nodular appearance on ultrasonography [32]. Although anti-inflammatory drugs, analgesics, and steroid injections had been recommended, ultrasound-guided percutaneous lavage should be the main treatment option, since it has been largely successful in reducing the pain dramatically and removing calcium deposits. ESWT is not feasible because of the associated pain, and there is no indication for more invasive therapeutic measures such as surgery in this stage [33, 34].

6.1.1 Percutaneous lavage of rotator cuff calcific tendinitis (barbotage)

Patients sit whilst putting the affected forelimb behind the back in cases of supra and infraspinatus, and externally rotated in for subscapularis CT. The affected shoulder is facing the operator and the ultrasonography monitor is placed on contralateral side in line with the operator. With aseptic technique, deposits are identified using the Ultrasound linear probe, 12–17 MHz, and entry is made into the deposit, with inline technique, after anesthetizing the pathway. Procedure can be done using large bore needle 18–20G especially if the calcific lesion is hard, or regular 23G needle in cases of soft lesion, with syringe filled with lidocaine. The tip of the needle should be placed at the soft spot of calcific deposit, and the needle should be aimed upward to allow gravity to help facilitate calcific material not returning into the deposit cavity. Some amount of lidocaine is injected by pulsating technique into the lesion, and the dissolved calcium will enter syringe, passively. Repeat injections until the syringe fluid is saturated with the material. Further same procedure continued with saline until no calcific material can be extracted anymore. The puncture and aspiration can be done with 1 or 2 different needles according to preferences. Furthermore, the needle is pulled out slowly until it reaches subdeltoid bursa space, and 20–40 mg triamcinolone mixed with 2 cc lidocaine 2% and 10 cc normal saline is injected. After treatment, the patient is advised to rest his shoulder for several days and avoid above-the-shoulder activity for approximately 3 weeks.

6.2 Chronic stage

Chronic stage of the disease may represent calcific, resorptive, and remodeling phases of the disease. Patient complained of chronic, inconsistent pain that sometimes interferes with daily living and can last for years, especially with shoulder abduction. Tenderness can be felt on the calcific deposit site. Some combined with mechanical impingement pain due to bursal thickening that is best shown with dynamic ultrasonography examination (**Figure 3**).

On newly diagnosed untreated CT with mild symptoms and of radiologic type A, conservative measures such as anti-inflammatory drugs, analgesics, and physiotherapy can be initiated with the aim of stimulating vascularization of the tendon and hence improving the conditions for resorption of the calcium deposit. Subacromial steroid injection could be offered in cases with combined impingement syndrome (**Figure 4**).

In calcification radiologic types A and B, complementary focused ESWT or UGN can be offered to disintegrate the calcium deposit. Both methods had shown clinically significant improvement in function and pain, although UGN shows superiority in calcium resorption [35–37].

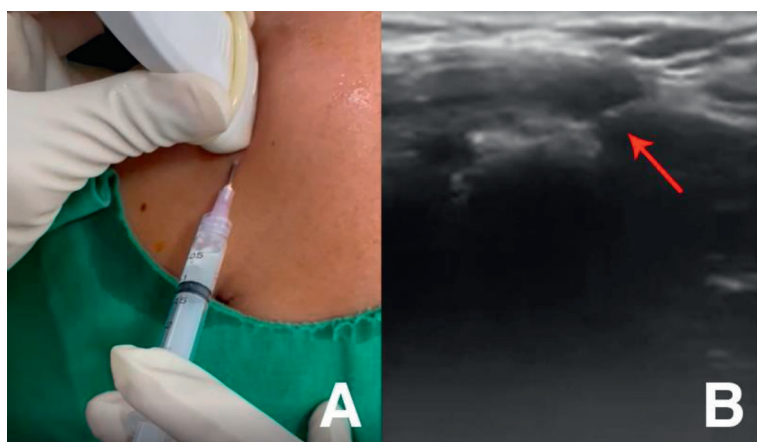


Figure 3.
 (A) Clinical image and (B) ultrasonographic image showing needle positioning (red arrow) towards calcium deposit during ultrasound-guided percutaneous lavage of subscapular tendon of the patient from **Figure 2**, showing saturated syringe content.



Figure 4.
 Post percutaneous lavage shows saturated saline-filled syringes (A), (B), (C) consecutively and (D) saturated lidocaine-filled syringe.

Ultrasonography-guided needling could be repeated after 3 months if the calcific deposit remains, and symptoms persist even if it were previously possible to partially extract the calcium. Finally, steroid injections may be recommended when the calcification has become poorly defined on imaging and it is no longer possible to puncture it. The disappearance of the calcification between 3 and 12 months is also observed in one-third of patients, without any additional treatment [38].

6.2.1 Surgery

Since surgery has a significant risk of iatrogenic tendon tear, it is usually reserved for failed conservative treatment only, especially if it is combined with concomitant lesions such as intraarticular, rotator cuff tear, and impingement syndrome. It consists of open or arthroscopic removal of calcific deposit, with or without tendon

repair and decompression with excellent short and mid-term clinical outcomes. But it has shown to have slow recovery of functional scores with majority of patients needing 6 to 12 months to recover [39–42].

6.2.1.1 Arthroscopy removal of calcific deposit

Patient is positioned in either lateral decubitus or beach chair position under general anesthesia, according to the surgeon's preference. Firstly, diagnostic arthroscopy of the glenohumeral joint is performed to address any intra-articular pathology with standard posterior portal. Then, the arthroscope is moved to the subacromial space. A lateral portal is then created above the calcific deposit and bursectomy is performed around the suspected calcific deposit. The calcium deposit is located using probe or by percutaneous needling. Hypervascularization, bulging, or calcific substance might be identified from the bursal site. The overlying tendon is then carefully incised longitudinally using a No. 11 blade and the calcific deposit is then removed by using a probe and motorized shaver. Inaccurate identification of the site of the calcification may result in iatrogenic tendon tears [43]. Intraoperative ultrasound has been reported to increase accuracy of calcific location. The arthroscopic surgery should be converted to mini-open surgery in case of unsuccessful identification of calcific deposit [17].

The surgeon should avoid overzealous calcium deposit removal and should err to incomplete removal to avoid unnecessarily large defects that may be challenging to repair. The remaining tendon after excision of the calcium deposits may not hold the repair. It also may result in excessive tension during margin convergence of the cuff. Although several authors reported shoulder better function in whom complete removal had been achieved [24, 44, 45], recent studies showed that it was not important to remove all the calcific deposit. Residual calcification was resorbed within 6–12 months of the surgical treatment and there were no significant differences in outcomes between patients with and those without complete removal of calcific deposits [39, 40, 42]. The postoperative spontaneous resorption of the remnant calcification may explain the similarity in clinical outcomes between patients whose calcific deposits were completely removed and those whose were not.

Whether it is necessary to repair rotator cuff defect after calcific debridement is debatable. Although rotator cuff repair had been reported to have superior functional outcomes, it had been also related to prolonged recovery [42, 46, 47], and stiffness. Most authors still recommend repairing large defects or encompassing significant thickness of tendon cuff width [48]. That is why surgeon should aim to remove calcific deposit conservatively, using combination of shaver and probe, preserving surrounding healthy tendon as much as possible with longitudinal opening. If large defects resulted despite all efforts, repairing the tendon with side-to-side suture is preferable, which is feasible in majority of cases (**Figure 5**).

6.2.2 Rehabilitation

Since surgery on CT had been shown to have a slow recovery, it is imperative to inform patients before treatment that pain may persist for up to 6 months of the treatment.

No special rehabilitation regimen is prescribed in CT debridement without cuff repair. Several days of rest in a sling are followed by gradual passive and active ROM

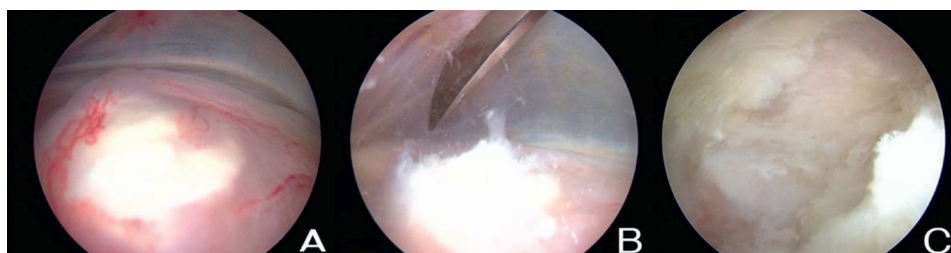


Figure 5.
Subacromial arthroscopic view of (A) supraspinatus calcific tendinitis bulging with hypervascularization on the surface, (B) debridement of calcific deposit, and (C) post debridement.

as tolerated. Patients refrained from excessive load exercises for the first 6 postoperative weeks to avoid disturbance of tendon healing.

In cases with significant defects and cuff repairs, early rehabilitation should be performed to prevent secondary stiffness and shoulder abduction splint is worn for 3 to 6 weeks according to rotator cuff repair protocol [42].

7. Complications

The progression of natural course of untreated disease leads to following complications, although some may also happen postoperatively as well [19].

7.1 Adhesive capsulitis

Adhesive Capsulitis, also known as frozen shoulder can be predisposed by factors inside the shoulder (Fracture, shoulder inflammation, CT) or by extra-articular factors (type 2 diabetes, hyperthyroidism). Some researchers also agree that prolonged immobilization can be a factor predisposing adhesive capsulitis [49, 50]. In symptomatic cases of CT, the calcification may cause severe pain and therefore may induce voluntary immobility. In cases of chronic CT, pain may have subsided, but limited range of shoulder motion is almost always noted, in which adhesive capsulitis becomes the common sequela. On another hand, adhesive capsulitis may be associated with the inflammation that's happening within the surrounding structures of the shoulder, and as previously mentioned, systemic diseases such as diabetes can be a predisposing factor to both CT and adhesive capsulitis. A study done by Jacobs et al. also showed that 18% of patients who underwent arthroscopy removal of the calcification experienced adhesive capsulitis and was suggested due to irritation of shoulder capsule by the residual calcium debris [51].

7.2 Greater tuberosity osteolysis

Greater tuberosity osteolysis is one of the rare complications of CT. A study done by Porcellini et al. shows that calcium deposits that come in contact with the tuberosities have consistently caused cortical lesions. This cortical lesion that happens due to biochemical effects of bone lysis can lead to insertion of calcification into the bone, which may cause further pain [52].

7.3 Ossifying tendinitis

Ossifying tendinitis is also a rare complication of CT. Ossifying tendinitis usually happens due to trauma or surgical intervention in the Achilles tendon, gluteus maximus tendon, and distal biceps. A case series and literature review by Merolla et al. found ossifying tendinitis histologically when performed a CT arthroscopy removal. Two cases were described showing persistent shoulder pain that was previously diagnosed as CT, but when removal was done, the calcification was found to be hard. Histological examination showed bone metaplasia. It was hypothesized that ossification happened because of mesenchymal cell transformation to bone-forming cells as a result of the calcification excision previously done [53].

8. Conclusion

Although CT is a transient disease that has self-healing nature, it can result in severe acute or prolonged chronic pain. X-ray, and ultrasonography are the most essential investigation for diagnosis and treatment plans. Conservative treatment should be the main treatment in newly diagnosed CT with mild symptoms, but more aggressive treatment is frequently needed in acute phase or unresponsive chronic phase. Ultrasound-guided percutaneous needling and lavage had been shown to effectively reduce pain and remove calcific deposits. It can be repeated in the less-responsive cases. Surgery role is limited. It should be reserved only for failed conservative treatment. Preservation of surrounding living tissue of the calcification during debridement should be a priority since residual calcification from incomplete excision can be resorbed spontaneously.

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Conflict of interest


The authors declare no conflict of interest.

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