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# Clinical evaluation and management of calcific tendinopathy: an evidence-based review

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**Abstract:** Calcific tendinopathy (CT) is an important musculoskeletal condition most commonly seen in the shoulder girdle, accounting for 10–42% of all shoulder pain. Despite the high prevalence within the shoulder region, CT has been demonstrated in many tendons throughout the axial and appendicular skeleton. Unlike degenerative tendinopathies, CT appears to be a self-limiting condition that affects otherwise-healthy tendon with deposition of calcium hydroxyapatite crystals between healthy tenocytes. In patients with functionally limiting symptoms or pain, the clinical course may be accelerated through a multitude of treatments including physical therapy and pain management, extracorporeal shock wave therapy, ultrasound-guided percutaneous lavage (UGPL), and operative debridement. Currently, the most efficacious and frequently utilized treatment for shoulder CT is UGPL due to its ability to effectively reduce calcium burden and pain while limiting soft-tissue damage. However, more evidence regarding the treatment and course of CT is needed before determining the most appropriate treatment at all potential sites of CT.

**Keywords:** calcific tendinopathy; calcific tendonitis; evaluation; treatment; ultrasound.

Calcific tendinopathy (CT) is characterized by the deposition of calcium hydroxyapatite crystals within a pathologically healthy tendon [1]. This calcification differs from the calcification seen in degenerative tendinopathy, which is composed of a heterogeneous mixture of calcium salts diffusely scattered throughout the tendon in areas of collagen degeneration or tear [1]. In contrast, CT calcification presents as a focal collation of calcium hydroxyapatite crystals in the ‘critical zone’, which is 1–2 cm from the insertion of a tendon [2], where there is thought to be high shear and stress forces that initiate the development of a focal calcific deposit between healthy collagen fibers [1].

CT has been described in innumerable tendons throughout the appendicular and axial skeleton [3]. However, CT has received renewed attention due to the high prevalence in the shoulder girdle, affecting 10–42% of the population [4–6], representing at least 33 million people in the United States alone [4–6]. Of the shoulder tendons, the supraspinatus tendon of the rotator cuff is most frequently affected, representing 80% of shoulder CT [7]. CT of the infraspinatus and subscapularis are seen less frequently, in 15 and 5%, respectively [7]. Hip girdle tendons, including the reflected head of the rectus femoris and gluteus medius, are the second most commonly affected region, with a reported prevalence of 5.4% [8]. Case studies have described CT in the proximal hamstring and biceps brachii as well as occasional descriptions in the longus colli, Achilles, and innumerable others [3, 9–12]. The high prevalence of shoulder CT has facilitated an improved understanding of the pathogenesis as well as the best practice approach to evaluation and evidence-driven treatment, which can be extrapolated to less frequently affected tendons [13].

Due to its presentation in otherwise-healthy tendons, CT is typically seen in younger patients, with most presenting in their 30s to 50s. Women are more commonly affected with CT than men, and associated conditions include diabetes, hypothyroidism, and hyperlipidemia. This is in comparison to degenerative tendinopathy, whereby patients are typically in their late 50s to 60s, with 65% of patients reported to

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have imaging evidence of rotator cuff tendon degeneration at age 65 [14–17].

Patients with CT present in one of four pathological stages (formative, resting, resorptive, or reparative) of disease that tend to loosely correlate with clinical presentation. As patients progress through the pathological morphologies, symptoms progress from diffuse pain to focal impingement, and finally, severe localized pain prior to resolution. The introduction of point-of-care ultrasound (POCUS) has allowed for the individual tailoring of interventions to the pathological state, such as oral medical treatments, corticosteroid injection, ultrasound-guided percutaneous lavage (UGPL), extracorporeal shockwave therapy (ESWT), and surgical debridement and decompression. However, with ongoing technological and biomedical enhancements, the treatment of CT continues to evolve and presents an opportunity for ongoing improvements in diagnosis and treatment.

This paper aims to focus on the clinical evaluation of CT by reviewing the anatomy, pathophysiology, clinical presentation, examination, and approach to imaging as well as by providing an overview of evidence-based treatment options.

## Clinical summary

### Anatomy and pathophysiology

The composition of tendon architecture has been well described through animal models, surgical specimens, and cadaveric studies. Tendons are comprised of three basic building blocks: tenocytes, extracellular matrix, and collagen (primarily Type I). Collagen is arranged in a hierarchy of tightly packed linear fibers surrounded by connective tissue sheaths of both the endotendon and epitendon [18, 19]. Although tendons are primarily avascular with most of their nourishment coming extrinsically through diffusion from the surrounding connective tissues, tenocytes aid in tendon health by surveying for injury and secreting extracellular matrix when stimulated. Unlike degenerative tendinopathy, which results in the breakdown of type I collagen and subsequent diffuse intratendonous calcification [20], CT typically involves the formation of a single foci of calcium hydroxyapatite crystals embedded between grossly healthy fibrils of collagen [2]. The prevalent theory for the development of CT is a ‘failed cell-mediated healing theory’ [2, 21], whereby excessive loading conditions and repetitive microtrauma results in an aberrant healing response and focal calcification formation. As such, commonly loaded

or injured tendons, including the supraspinatus, are more vulnerable. However, due to commonalities in structure and pathogenesis, any tendon throughout the body can be affected with CT.

The development of CT has been described by Uhthoff and Loerr [2] and Gosens et al. [22] to follow distinct stages of progression. First, the precalcific stage is characterized by the fibrocartilaginous metaplasia of tenocytes into chondrocytes, creating an environment in which calcifications can develop. The second, calcific stage, is subdivided into formative, resting, and resorptive phases. In the formative phase, calcium crystals are formed and coalesce into large foci of calcification, typically with a chalk-like appearance [2, 22]. The resting phase is characterized by a stable presence of mature calcifications surrounded by a fibrocartilaginous tissue border or ‘cap’. Finally, in the resorptive phase, an inflammatory reaction to the calcific deposits occurs and vascularized tissue develops at the calcification periphery or cap [2, 22]. Resorption is mediated through macrophages and multinuclear giant cells, which infiltrate and phagocytose the calcific deposits. The calcification at this phase resembles toothpaste consistency and can leak into nearby bursae, bone, or muscle, causing severe pain [2, 22]. The last stage is the postcalcific/reparative phase, in which fibroblasts remodel the space previously occupied by calcium with Type III collagen. The Type III collagen is then replaced by Type I collagen, ultimately resulting in complete healing of the affected tendon and the restoration of tendon architecture [18].

### Clinical presentation and examination

The clinical presentation and examination of CT is highly variable and depends on both the stage of the disease as well as the anatomic location [23]. Due to the higher prevalence of shoulder and hip CT, the stage-to-clinical correlations are based on these locations but can be extrapolated to any anatomic location in the body where CT develops [23].

As demonstrated on numerous imaging studies [24], a large percentage of patients with CT are asymptomatic, with little to no positive examination findings throughout the entire course of disease. Although the true prevalence of asymptomatic CT is unknown, asymptomatic shoulder CT has been proposed to affect 35–50% of the population [24]. As such, symptomology may vary significantly from patients who are completely asymptomatic to those presenting with acute, debilitating pain. In addition, patients

may present at any stage of disease without symptoms of previous or subsequent stages. For example, one may present with acute-onset severe pain in the resorptive phase, with no prior symptoms in the formative phase. As always, clinical correlation with imaging findings is critical to ensure an accurate diagnosis.

Patients who present in the formative stage, in which calcium hydroxyapatite crystals form, typically describe a poorly localizable, subacute, low-grade pain that may be more pronounced or noticeable at night or with increased pressure in the area [2, 25]. Physical examination in this phase may demonstrate vague pain that is provoked by shoulder movements, without localizable or specific findings [2, 25]. Notably, for unknown reasons, a small subset of patients (~10%) may remain in a chronic formative phase with symptoms persisting for months to years unless treated [15, 23]. However, most patients progress into the resting stage after 3–6 months. As stabilization of CT develops into a single calcific mass, symptoms become more mechanical secondary to the size of the lesion and the limited elasticity of the tendon. Patients presenting in the resting phase may complain of clicking, snapping, or catching sensations with joint motion and may have impingement and localizable pain with either stretch or recruitment of the affected tendon (Table 1) [26, 27]. As an example, those with supraspinatus CT may present with signs of rotator cuff impingement demonstrated by pain with abduction or with over-head activities in which the supraspinatus tendon, and the associated large calcific mass, become caught between the acromion and humeral head.

Classically, in shoulder and hip CT, patients present in the resorptive stage, the most painful stage of CT, and may present acutely, without symptoms at previous stages [27, 28]. In the resorptive stage, due to inflammation with associated increased vascular flow and extrusion of calcium into nearby structures, patients tend to have localized swelling, erythema, and pain, with limited range of motion in their joints. The significant inflammatory reaction at this stage may mimic more aggressive osseous and articular conditions including septic arthritis [29, 30]. During the transition from the resorptive to the reparative stage, patients will experience diminishing signs and symptoms until normal tendon structure and joint mechanics are restored.

## Diagnostic imaging

### Plain radiographs

Plain radiographs with standard joint-specific views can visualize CT in most cases, in addition to ruling out more

ominous osseous abnormalities. Radiographic morphology can also be utilized in evaluating the stage of CT (Table 1, Figure 1) [22, 28].

There are multiple radiographic classification systems for CT [31, 32]. The Gartner and Heyer classification system describes three unique deposit morphologies that correlate well with the histologic stage: (1) well circumscribed and dense (resting phase); (2) well circumscribed with an inhomogeneous structure or poorly circumscribed with a homogeneous structure (formative); and (3) poorly circumscribed and translucent (resorptive phase) [12, 27]. Plain radiographic findings may not correlate with the degree of symptoms or clinical improvement as similar initial symptoms and prognosis has been found regardless of location, size, and classification of calcifications on radiographs [33].

### Ultrasound

Ultrasound is the other diagnostic imaging modality of choice for CT due to its superior ability to visualize CT at all stages and to guide therapeutic interventions [3, 34]. Sonographically, CT appears as a hyperechoic focus within the fibular pattern of healthy tendon, with posterior acoustic shadowing. Calcifications in CT can take on multiple forms that have been shown to correlate with pathological state. Hyperechoic arc-shaped deposits are characteristic of the resting phase and typically require more aggressive treatment such as UGPL or ESWT, owing to lower rates of spontaneous resolution. Non-arc shaped, fragmented, or nodular calcifications with increased color Doppler signal are indicative of the resorptive phase, in which spontaneous resolution is common and management with nonsteroidal anti-inflammatories (NSAIDs) and physical therapy is often successful. Ultrasonographic findings associated with symptomatic CT include fragmentation, power Doppler signal, and distention or extrusion of calcium into surrounding bursal structures [35, 36]. However, similar to plain radiographs, although the appearance of calcifications on ultrasound may correlate with pathological stage and help guide treatment, findings may not correlate with the degree of symptoms [37]. In addition, although ultrasound is most commonly utilized for CT surrounding the shoulder and distal extremities due to the superficial locations [38], it may still be diagnostic for deeper structures, including those in the hip region or axial skeleton [38].

Newer scanning techniques, including compound scanning technology and elastography, hold promise for

**Table 1:** Outline of expected physical examination findings, suggested radiographic views and CT mimics.

Location	Positive examination findings	Radiograph views	CT mimics
<b>Shoulder</b>	Painful range of motion	– Anteroposterior	<b>Adhesive capsulitis:</b>
Supraspinatus	– Empty can	– Internal and	– May mimic CT when patients have severe pain in the resorptive phase
	– Painful arc	external rotation	
Infraspinatus	– Resisted external rotation pain: tested with elbows against side and flexed to 90 degrees	– Supraspinatus outlet	– Passive range of motion normal in CT but limited in adhesive capsulitis
		– Axillary	
Subscapularis	– Belly press pain		<b>Long head biceps tendinopathy:</b>
	– Lift off pain		– May present similarly when the anterior supraspinatus or subscapularis has CT
Long head biceps	– Speed's test pain		– Normal radiographs for biceps tendinopathy
	– Uppercut test pain		<b>Degenerative tendinopathy:</b>
			– Pain with resisted recruitment
			– Pain with passive stretch
			– Pain to palpation
			– Negative plain radiographs with signs of tendon degeneration on ultrasound
			<b>Acute synovitis or septic arthritis:</b>
			– Severe pain and motion limitations for both
			– Reflected head of rectus femoris involvement may present with intra-articular findings due to its close proximity to the hip joint
			– Infectious/inflammatory labs usually normal in CT
			<b>Tendon tear:</b>
			– Acute severe pain can be seen in both
			– In CT, active range of motion should be intact albeit painful
<b>Hip</b>	Painful range of motion	– Anteroposterior	<b>Acute synovitis or septic arthritis:</b>
Rectus femoris	– Anterior inferior iliac spine tenderness	– Lateral	– Severe pain and motion limitations for both
	– Resisted hip flexion and internal rotation		– Reflected head of rectus femoris involvement may present with intra-articular findings due to its close proximity to the hip joint
Gluteus medius or minimus	– Greater trochanteric tenderness		– Infectious/inflammatory labs usually normal in CT
	– Side-lying hip abduction pain		
	– FABER pain		
Proximal hamstring	– Ischial tuberosity tenderness		
	– Supine single straight-leg bridge pain		
<b>Other sites</b>	Painful passive motion, point tenderness	– Anteroposterior	<b>Chronic tendinopathy:</b>
<u>Upper extremity</u>			– Pain with resisted recruitment
<u>Common extensor and flexor tendons of the elbow</u>	<u>Extensor:</u>		– Pain with passive stretch
	– Resisted wrist extension (Cozen's or Thomsen test)		– Pain to palpation
	– Maudsley's (resisted 3rd digit extension)		– Negative plain radiographs with signs of tendon degeneration on ultrasound
	<u>Flexor:</u>		<b>Tendon tear:</b>
	– Resisted wrist flexion: with elbow extended and forearm supinated		– Acute severe pain can be seen in both
	– Passive wrist extension		– In CT, active range of motion should be intact albeit painful
<u>Lower extremity</u>			
Quadriceps tendon	– Resisted knee extension	– Lateral	
	– Active straight leg raise		
	– Preference to keep knee flexed		
	– Pain with stairs and squatting		

Table 1: (continued)

Location	Positive examination findings	Radiograph views	CT mimics
Patellar tendon	<ul style="list-style-type: none"> <li>– Resisted knee extension</li> <li>– Pain with squatting and jumping</li> </ul>	– Lateral	
Achilles tendon	<ul style="list-style-type: none"> <li>– Calf raises</li> <li>– Pain with running and possibly walking</li> </ul>	– Lateral	

CT, calcific tendinopathy.

evaluation of CT but need more investigation before routine use [35, 36].

## Magnetic resonance imaging or arthrography (MRI/MRA)

Unlike the superiority of MRI/MRA for the diagnosis of many musculoskeletal conditions, CT is difficult to visualize with standard MRI/MRA due to the similar signal hypointensity of calcifications compared to normal tendon. This leads to false-negatives and missed deposits or false-positives of normal hypointense, healthy tendons [39, 40]. MRI is often utilized to rule out other local joint and soft tissue pathology that may cause similar symptoms including tendon tears, osteoarthritis, and chondral or labral injury.

The advent of specialty sequences including susceptibility weighted imaging (SWI) has allowed for the improved diagnostic ability of MRI because calcifications appear hyperintense in contrast to the hypointensity of healthy tendon. Similar to ultrasound, calcifications are typically best seen in the coronal plane whereby the length of the tendon and the positioning of CT are best demonstrated. The use of SWI MRI is most commonly utilized for preoperative planning, whereby a diagnosis of CT has already been made and a precise three-dimensional (3D) localization of calcifications is needed for operative resection. Additionally, MRI has a high specificity for tendon and ligamentous tears as well as cartilaginous injuries that are important to identify and may be amenable to surgical repair or addressed intraoperatively.

## Treatment

The treatment of CT follows a stepwise algorithm that begins with a nonoperative regimen of activity modification, anti-inflammatory medications, and physical therapy, and may progress to minimally invasive treatments including

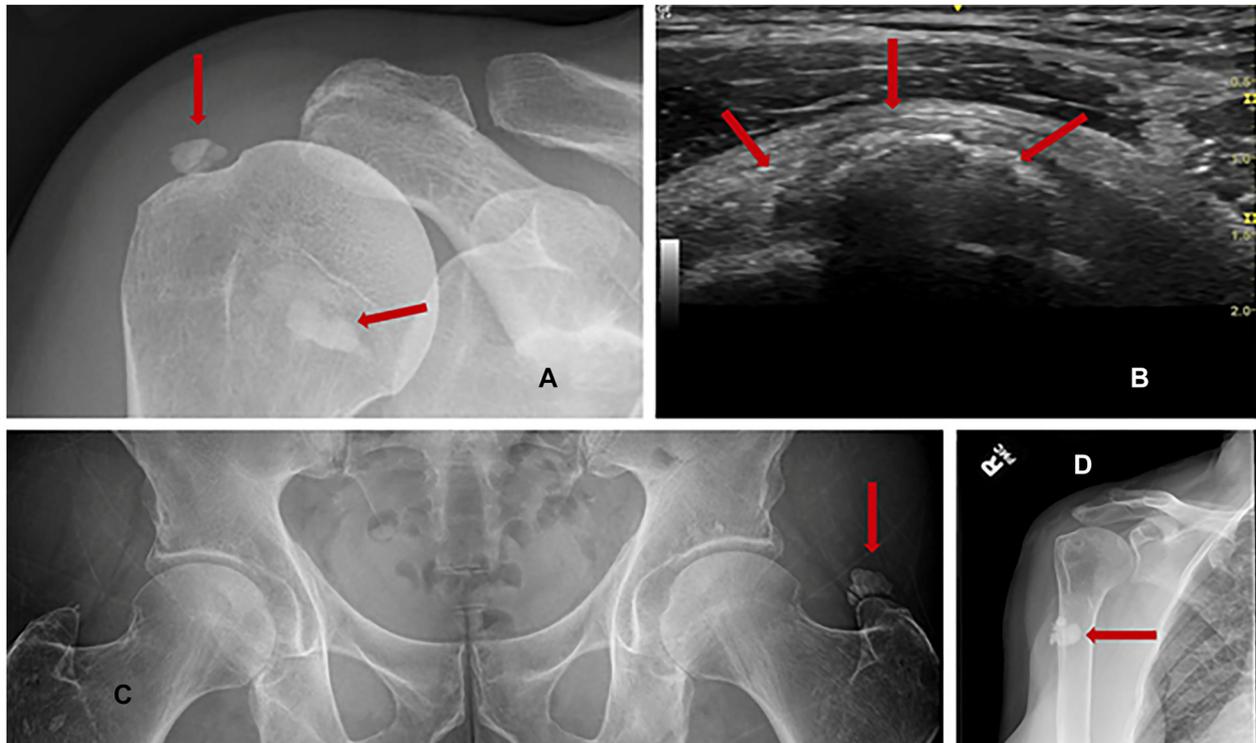
UGPL and ESWT. For those with refractory symptoms, operative debridement and potential tendon repair are considered (Figure 2) [41].

## Nonoperative treatments

Given the natural history of CT, whereby the natural course of disease concludes in complete resolution without residual tendon damage or degeneration, a trial of conservative therapy focused on symptomatic relief and functional improvement is utilized by most practitioners [42]. Although the following treatments are frequently utilized due to their low risk profile [43], there is little evidence regarding the individual efficacy or superiority of any particular treatment with regards to pain relief, improvements in function, or patient satisfaction.

Oral NSAIDs are commonly utilized due to their ability to provide relief through analgesia and reduction in inflammation [44]. Given no direct comparison studies, any NSAID and approved dosage can be considered, including topical NSAIDs, which have been demonstrated to result in lower systemic complications [45, 46]. Duration of NSAID therapy should be limited, when possible, given the long-term gastrointestinal, renal, and cardiovascular side effects of chronic NSAID use [44]. Cimetidine is another oral medication that has been evaluated for CT and is theorized to alter parathyroid hormone activity and promote absorption of calcifications [47, 48]. However, its use has only been reported in case series, and high-quality evidence for use in CT is currently lacking. Common side effects include headaches and drowsiness, as well as a rare risk of gynecomastia.

Despite the common use of physical therapy in CT, there has not been a specific exercise regimen that has been shown to be efficacious in CT. Only a handful of studies have investigated physical therapy in CT as a sole intervention [49–51]. However, due to the ubiquitous nature of shoulder pathologies, physical therapy has been utilized as a co-intervention or control group in the large majority of publications [34, 52, 53]. Described



**Figure 1:** Plain radiograph and ultrasonographic images of common calcific tendinopathy. (A) Plain anteroposterior (AP) radiograph of the shoulder demonstrating supraspinatus (superior arrow) and subscapularis (inferior arrow) CT seen as a radiopaque lesion near the greater tuberosity and lesser tuberosity, respectively. (B) Ultrasonographic image of supraspinatus CT demonstrating hyperechoic structure with posterior shadowing within the supraspinatus tendon. (C) Plain anteroposterior radiograph of the pelvis demonstrating gluteus medius CT seen as a radiopaque lesion near the greater trochanter. (D) Plain AP radiograph of the shoulder demonstrating biceps CT as a radiopaque lesion within the bicipital groove/anterior humerus. CT, calcific tendinopathy.

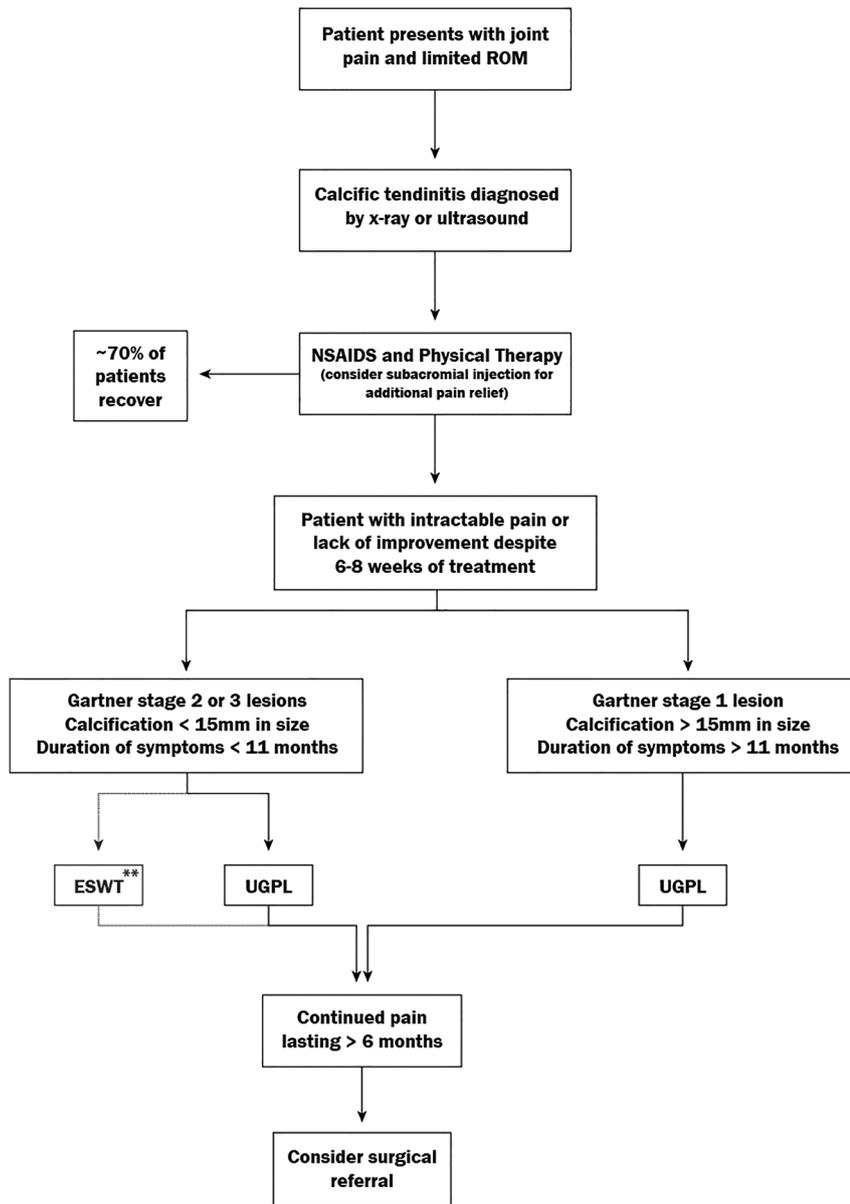
techniques include a wide range of treatments consisting of range-of-motion exercises and aspects of periscapular and rotator cuff muscular strengthening [54–56]. The combination of these techniques is thought to preserve articular and tendon mobility while optimizing joint mechanics thereby decreasing dynamic tendon impingement. These techniques may be effectively extrapolated to any tendon affected by CT, but there is currently not sufficient evidence to guide rehabilitation protocols.

Osteopathic manipulative therapy and/or friction massage can be recommended for appropriate patients with secondary somatic dysfunction, myofascial pain, and motion restriction with techniques that include counter-strain and muscle energy [57]. Although there is only preliminary evidence, the available literature has demonstrated a low potential for harm, with utilization in numerous studies for both controls and active groups [43, 57, 58]. In addition, several adjunctive modalities have been studied including therapeutic ultrasound, transcutaneous elective nerve stimulation (TENS), and acetic acid iontophoresis [59, 60]. Although therapeutic ultrasound and TENS have demonstrated promise in providing

pain relief, none of these modalities have definitively demonstrated improvements in pain, function, or time to condition resolution [59, 60]. Overall, many noninvasive treatments have been evaluated for the treatment of CT, but there is limited evidence to recommend one specific treatment over another [61].

### Minimally invasive treatments

Minimally invasive treatments or percutaneous techniques for the treatment of CT include isolated bursal or peritendinous injections, ESWT, and UGPL. For those in the resorptive phase, whereby there is already evidence of calcific resorption but there is continued limitation due to pain, additional pain relief can be achieved through an isolated bursal or peritendinous corticosteroid injection. Evidence for subacromial-subdeltoid corticosteroid injection (SAI) in a study of 21 patients with shoulder CT demonstrated 60% pain relief at 6 weeks and 3 months; however, there was a return to mean pain scores thereafter [37]. Notably, the ability of the tendon to regenerate



**Figure 2:** Treatment algorithm for those with calcific tendinopathy. UGPL is generally the preferred treatment over ESWT based on studies showing greater clinical benefit and greater reductions in the size of calcifications. Additionally, UGPL is typically covered by insurers in the United States, whereas ESWT is often an out-of-pocket expense. ESWT, extracorporeal shockwave therapy; NSAIDs, nonsteroidal anti-inflammatory drugs; UGPL, ultrasound-guided percutaneous lavage.

or resorb calcific deposits is not hindered by the anti-inflammatory or catabolic effects of the corticosteroid [37]. Therefore, the utilization of corticosteroid injections for pain relief in CT does not disrupt the natural disease course of tendon reconstitution.

Several individual studies as well as a network meta-analysis have demonstrated that ESWT, a technique in which sound waves are delivered to mechanically disrupt tissues, results in reduced pain and calcification size compared to nonoperative treatments including physiotherapy in those with shoulder CT [23, 49, 50, 62–64]. A few small studies have demonstrated that ESWT is most efficacious in those with smaller calcifications or at an earlier stage of disease. As such, it has been recommended that

those with Gartner type I calcifications, calcification extent >15 mm, or duration of symptoms >11 months do not receive ESWT because these individuals do not reliably improve following therapy [65]. This may be due to the limitations of ESWT to disrupt or mobilize larger, more stabilized calcifications.

ESWT can be classified into “radial” (R-SWT) or “focused” (F-SWT), with differences in waveform characteristics and subsequent effect on the targeted tissue [52]. In addition, energy intensity levels during ESWT may be classified as low (<0.08 mJ/mm<sup>2</sup>), medium (0.08–0.28 mJ/mm<sup>2</sup>), or high (>0.28 mJ/mm<sup>2</sup>) energy flux density [52]. Currently, there is more evidence needed to determine the most efficacious protocol of ESWT for CT. However, the authors

suggest that based on the current literature, a high-energy protocol utilizing F-SWT, or a combination of F-SWT and R-SWT, may provide the most significant improvement in pain and calcification size [52, 62, 66]. In the United States, ESWT is typically an out-of-pocket expense, and this should be considered when assessing treatment options.

UGPL is perhaps the most studied and utilized treatment for painful cases of CT not responding to nonoperative treatments [67, 68]. Prior meta-analyses have demonstrated that UGPL resulted in statistically and clinically significant improvement in both pain and radiographic resolution when compared to placebo, isolated corticosteroid injection, or ESWT in shoulder CT [53, 69]. Mean reductions in Visual Analogue Scale (VAS) of 3.14 (95% CI 5.64–0.64) were seen, with an increase in resolution demonstrated with complete resorption of calcific deposits in 45% of patients at 6 weeks and 86% of patients at 12 months post-treatment [23, 53, 63].

Despite the significant improvement with UGPL [67, 68], protocols vary regarding the technique, lavage solution, and whether SAI is administered, with no definitive evidence regarding superiority of individual techniques. The two predominant UGPL procedural approaches are the single-needle and double-needle techniques. The single-needle technique involves a single puncture of the calcification, at which point the syringe plunger is pulsed to inject saline into the deposit and allow backflow of calcium into the syringe when pressure is released. Syringes can be exchanged, and the technique is continued until the aspirated fluid is clear. Proponents of the single-needle technique contend that use of a single needle puts the patient at lower risk of infection, bleeding, and tendon trauma. The double-needle technique utilizes a second needle to aspirate the fluid introduced by the first needle. This creates a continuous influx and outflow of saline to remove calcium. Proposed benefits of the double-needle technique include greater fragmentation of the calcium deposition due to the introduction of two needles, and lower saline pressures within the calcifications (because saline is removed at the same rate it is injected). Theoretically, lesser intra-deposit pressures may reduce the risk of calcium spreading into the surrounding tendon and bursa, thereby decreasing the risk of postprocedural bursitis. One trial comparing these two techniques in 211 patients found no difference in the ease of calcium dissolution, calcification appearance, or clinical outcomes [70]. Although no statistically significant difference was found between these two techniques and postprocedural bursitis, there was a trend toward increased postprocedural bursitis in the one-needle technique group.

One large randomized trial demonstrated that warm saline (42 °C, 107.6 °F) led to faster procedural times,

easier calcium dissolution, and less subacromial bursitis compared to room-temperature saline in 462 patients [71]. However, a recent systematic review of 18 studies found that there continues to be limited evidence to definitively support the use of warmed lavage solution [72]. Sodium thiosulfate, a medication utilized in the treatment of calciphylaxis, did not provide benefit over normal saline [73]. In addition, although some providers often administer a corticosteroid injection after lavage, it has only been shown to be beneficial in those utilizing a single-needle technique [74, 75]. Although SAI is the most common type of bursal injection due to the predominance of shoulder CT, a bursal (trochanteric for gluteus medius) or peritendinous (common wrist extensors, proximal hamstring, etc.) injection may be performed. Corticosteroid injections are generally not administered around distal load-bearing tendons including the Achilles tendon.

Given the above evidence, the authors utilize and recommend a single-needle technique, UGPL, with a postprocedural bursal injection to reduce the risk of postprocedural bursitis. Utilizing this technique, study outcomes demonstrate complete resorption of calcific deposits in nearly half of patients by 6 weeks and the large majority by 1 year [61, 63, 68].

## Operative treatments

Operative intervention may be considered for patients who fail nonoperative management strategies and suffer from chronic pain lasting greater than 6 months. The goal of operative management is to debride and maximally remove calcifications while preserving tendon integrity. As such, local lavage after debridement is commonly performed in order to wash out any remaining calcium fragments within the joint space or local tissues and prevent secondary joint or tissue stiffness [76, 77]. However, patients should be counseled that improvements postsurgically may be slow because many studies report significant improvements in pain and function starting at 3–6 months postoperatively [76, 77].

Commonly, surgeons opt for arthroscopic approaches over open approaches to limit associated tissue damage; however, this is highly dependent upon anatomic location. Procedures for tendons located more superficially, including the Achilles tendon, have been described utilizing an open operative approach because there is easy tendon access with little intervening muscle or soft tissue [78]. Alternatively, deeper tendons, including the rectus femoris tendon, are often approached arthroscopically to limit local muscle trauma and ensure a shorter recovery

time [23, 79]. Regardless of the operative approach, the use of intraoperative ultrasound has gained popularity due to its ability to localize calcific deposits resulting in reductions in procedural time, number of portals, and length of operative field/incision [80].

## Conclusions

CT is a common pathology characterized by the deposition of calcium hydroxyapatite crystals within a healthy tendon. Although CT is most frequently seen in tendons surrounding the shoulder, it can present in any tendon throughout the axial and appendicular skeleton. CT typically progresses through four stages consisting of the formative, resting, resorptive, and reparative stages. Patients most commonly present in the resorptive phase of the condition, whereby there is resorption of the calcium deposits resulting in significant inflammation associated with pain and limited function. Ultrasound and plain radiographs continue to be the gold standard for the diagnosis of CT and can be useful in guiding staged-treatment. Current nonoperative management strategies consist of oral NSAIDs and physical therapy for those that are in the reparative phase or not significantly functionally limited. Those with resistant disease may require SAI, UGPL, and ESWT. The best results have been demonstrated with UGPL. Operative debridement is reserved for those failing all previous treatments.

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**Competing interests:** None reported.

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