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Review

Imaging of calcific tendinopathy: natural history, migration patterns, pitfalls, and management: a review

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Abstract

Calcific tendinopathy is characterized by the deposition of calcium hydroxyapatite crystals in various tendons of the body. Terms like calcium tendinitis, tendinosis, and tendinopathy are used interchangeably. Calcific tendinopathy is a common and well-documented ailment in the literature. Although common, the natural history, aetiology, and progression of calcific tendinitis are poorly understood. The treatment options include conservative and interventional measures. However, these measures cannot be applied as a blanket and are often tailored depending on the stage/phase of the disease. Out of the recognized stages of the disease, the resorptive stage causes the utmost symptoms when the calcium is rather soft and unstable. During this stage, the calcium may migrate beyond expected resorption and get deposited in the adjacent tissues contiguous with the calcium focus. The common destinations include bursal migration, intraosseous migration, muscular migration, and other less common migration sites. Such atypical presentations can lead to dilemmas in the diagnosis, prolongation of the diagnostic pathway, unwarranted apprehension, and treatment delay. Radiologists' role in this situation is to correctly recognize the imaging findings of atypical presentations of calcific tendinopathy and prevent unnecessary diagnostic and interventional studies. In this review article, we describe the pathogenic pathway and natural history of calcific tendinopathy from a radiologist's perspective and discuss different migratory patterns of calcium in calcific tendinopathy not only around the shoulder but also in other areas of the body on different imaging modalities. We also show a few examples of mimics and pitfalls on imaging. Finally, we discuss the appropriate management option of this condition.

Keywords: calcific tendinopathy; ultrasonography; musculoskeletal pain; magnetic resonance imaging; radiography.

Introduction and background

Calcific tendinopathy is a common but poorly understood ailment. Several hypothetical theories have been put forth for the pathogenesis of the condition out of which, the degenerative and cell mediated theories are the most accepted. In the former, the tendon fibre degeneration is followed by calcium deposition. In the latter, a favourable environment permits an active process of cell-mediated cascade resulting in calcium deposition, occasionally followed by spontaneous phagocytic resorption.[1] The human body fluids such as blood, interstitial, and intracellular fluids are supersaturated with calcium phosphate (hydroxyapatite); however, despite this supersaturation, normal crystallization processes such as calcium consolidation in bone and teeth occur as these processes are biologically controlled by calcium homeostasis. The uncontrolled pathological crystallization is seen in various conditions such as tumoural tissue calcification, renal calculi, gallstones, and calcifications in atherosclerosis. Pathological crystallization is thought to occur in favourable situations with the presence of crystallization inducers, the absence of crystallization inhibitors, and/or failure of the immune system.[2]

Calcific tendinopathy is characterized by the deposition of calcium hydroxyapatite crystals in various tendons of the body. Terms like calcium tendinitis, tendinosis, and tendinopathy are used interchangeably. The term "insertional calcific tendinopathy" is used when it affects the bon-tendon junction. The first description of calcium deposition around a joint was made by Painter in 1907, who identified calcium deposition as one of the causes of subdeltoid bursitis.[3] The first description of calcific tendinopathy dates back to 1939 when Bishop described calcification of the supraspinatus tendon.[4] A study describing radiological aspects of calcific tendinopathy published in 1977 by Gerster et al[5] evaluated 52 patients of chondrocalcinosis on conventional radiography and discovered calcium deposits in 7 Achilles tendons, 7 quadriceps tendons, and 1 plantar fascia. The use of ultrasonography to see calcification in the tendon was first described in the patellar tendon by Fornage et al in 1984.[6] Though it can involve any tendon in the body, rotator cuff is the most common site to be involved. Table 1 shows non-rotator cuff tendons in the body affected by calcific tendinopathy as per published literature till date.[7-34]

Prevalence of calcific tendinopathy ranges from 2% to 20% (a range of 10%-40% in chronic painful shoulders and 3% in non-painful shoulders).[35] No study till date has described any racial variation in the occurrence of calcific tendinopathy though it is thought to be more common in

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Table 1. Non-rotator cuff tendons in the body affected by calcific tendinopathy.

Serial number	Site	Muscle/tendon [7–34]
	Neck	Longus colli [Ødegaard KJ 2022, Lim WQ 2022]
	Upper extremity	Common extensor origin at lateral epicondyle of humerus [Abate 2016]
	Upper extremity	Flexor digitorum superficialis [Ali SN 2004]
	Upper extremity	Flexor digitorum profundus [Munjal A et al 2013]
	Upper extremity	Pectoralis major insertion over humerus [Cahir J 2005]
	Upper extremity	Abductor policis longus and brevis [Dilley DF et al 1991]
	Upper extremity	Flexor carpi radialis and ulnaris [Dilley DF et al 1991, Van Demark RE Jr 2022]
	Upper extremity	Distal biceps tendon [Gossner J 2018]
	Upper extremity	Long head of biceps [Goldman AB 1989]
	Upper extremity	Extensor pollicis longus [Hakozaki M 2007]
	Upper extremity	Flexor pollicis longus [Kheterpal A 2014]
	Upper extremity	Flexor hallucis brevis [Lee HO et al 2012]
	Upper extremity	Abductor digiti minimi and pollicis brevis [Lee HO 2012, Shields JS 2007]
	Trunk	Trapezius [Nofsinger CC 1999]
	Trunk	Iliopsoas [Zajonz D 2013]
	Lower extremity	Quadriceps [Abram SGF 2012, Varghese B 2006]
	Lower extremity	Gluteus medius and maximus [Almedghio S 2014, Choudur HN 2006]
	Lower extremity	Adductor magnus [Patil AR 2021]
	Lower extremity	Patellar tendon [Beebe JA 2013]
	Lower extremity	Rectus femoris origin [Braun-Moscovici Y 2006]
	Lower extremity	Achilles [Daniel Sullivan 2021]
	Lower extremity	Popliteus [Doucet C 2017]
	Lower extremity	Tibialis posterior [Harries L 2011]
	Lower extremity	Peroneus longus [Klammer G 2011]
	Lower extremity	Vastus lateralis and Sartorius [Yi SR 2015]

Caucasians. Women are more commonly affected than men in the age group of 30-50 years.[36] Pathogenesis of calcific tendinopathy is multifactorial and poorly understood. The factors such as age, vascular ischemia, overuse, dyslipidaemia, diabetes, hormonal, and genetics factors have been implicated in its etiology.[37-44] Normal tendons are composed of type I collagen, elastic fibres, and ground substance like proteoglycans, glycosaminoglycans, and inorganic elements such as calcium, phosphorus etc. The tenocytes lie between the collagen fibrils and are responsible for formation of the ground substance. Apart from the forementioned degenerative and cell mediated theories of pathogenesis, ischemic injury has also been described.[45-47] The etiological factors are thought to cause metaplastic transformation of tenocytes into chondrocytes with subsequent intratendinous calcification and enchondral ossification.[48,49] A phase of phagocytosis by multinucleated giant cells ultimately results in tendon remodelling, and calcium resolution.[1] Other specific causes such as compression of the tendon leading to formation of fibrocartilagenous matrix in the tendons explaining the reason behind occurrence of calcific tendinopathy in the supraspinatus tendon when chronically impinged have been put forth, however, occurrence of calcific tendinopathy in young patients not involved in manual work raises questions on the association of overuse and degenerative phenomenon with calcific tendinopathy. Another hypothesis is potential of stem cells present in the tendons to undergo differentiation into adipocytes, chondrocytes, and osteocytes. Different pathogenetic pathways play in development of non-insertional and insertional calcific tendinopathies; the first one is described to occur due to repetitive micro-trauma with calcium deposition whereas, the second one is described to occur due to enchondral ossification at the tendon-bone junction.[49,50] Mineralization is an active and complex process involving protein and enzymes leading to apetite crystal deposition, mechanism of which is unclear.[51]

One of the most accredited etiological theory is from Uhthoff et al[50] who described 3 stages of calcific tendinopathy:

- Precalcific stage
  - It is a preconditioning phase where the "critical area" of future calcification undergoes fibrocartilaginous metaplasia.
- Calcific stage
  - Formative phase: Aided by the chondrocytes of the fibrocartilaginous metaplasia, a matrix of calcification is laid in the tendon which coalesce over a period of time to form a rather multifocal areas of calcium deposition.
  - Resting phase: This is a variable phase of inactivity where no inflammation or vascular infiltration occurs.
  - Resorptive phase: Thin-walled vascular channels at the periphery of the deposits develop which get surrounded by macrophages and multinucleated giant cells that phagocytose and remove the calcium.
- Postcalcific stage/repair stage
  - The repair phase begins with granulation tissue replacement of the space created by calcium resorption and this phase happens concurrent to the resorptive phase of the calcific stage. The granulation tissue matures to a scar; the collagen fibres and the fibroblasts align along the longitudinal axis of the tendon.

In the resorptive phase of the calcific stage, calcium crystals may migrate to the adjacent tissues. Majority of literature available on migration pattern of the calcium in calcific tendinopathy is based on the scattered case reports.[52-55] There are a few review articles on this topic, one of which focuses

only on ultrasonographic features of the pathology[56]; while, one review article focuses on multimodality approach around shoulder.[57] In this review article, we describe the pathogenic pathway and natural history of calcific tendinopathy from a radiologist's perspective and discuss different migratory patterns of calcium in calcific tendinopathy not only around shoulder but also in other areas of the body on different imaging modalities. We also show a few examples of mimics and pitfalls on imaging. Finally, we discuss the appropriate management option for this condition.

Natural history of calcific tendinopathy and clinical features

As discussed above, the enchondral ossification, chondral metaplasia, stem cell differentiation, and various other factors can lead to deposition of calcium crystals in the tendons. Degenerative process and overuse may or may not aid this process. During resorptive phase, where the calcium is expected to be swept away by the phagocytic process, the calcium crystals may migrate to the adjacent tissues due to mechanical forces or deficient areas of the tendon or osseotendinous architecture such as tendon tears, intraossous ganglion formation etc. Different migration patterns have been described in calcific tendinopathies by various authors.

Calcium migration can be,

- Migration within the tendon
- Migration to myotendinous junction
- Migration to the muscle belly
- Intraosseous migration
- Sub bursal migration
- Bursal migration
- Other soft tissue migrations

It is important to know and understand the migratory patterns as the intraosseous and intramuscular migration can often mimic inflammatory or neoplastic process on ultrasound and MRI and patient may undergo unnecessary invasive investigations like biopsy.[53] Intrabursal migration is the most common pattern of migration followed by intraosseous, intramuscular, and rarer unusual migration patterns. In the rotator cuff, where the calcific tendinopathy is very common, calcium can often migrate into adjacent contiguous tendons.

Clinical features of the patient largely depend on the stage of calcification.[50] Patients are generally asymptomatic in pre-calcific and postcalcific stages and hence foci of tendon calcifications can be detected in asymptomatic patients incidentally. During the formative and resting phases of calcific stage, there could be variable amount of pain, however, the resorptive stage is predominantly characterized by pain and the patients may present with acute symptoms. In the resorptive phase, calcium crystals migrate to the adjacent tissues due to mechanical forces and this leads to local inflammation that may cause excruciating pain and limitation of range of motion of the joint involved. If the crystals migrate to the superficial structures close to skin, hyperesthesia and hyperaemia of the skin can occur and the patients can present with low grade fever. Symptoms are usually worse in the first few days and slowly regress over a period of 2-3 weeks which corresponds to the beginning of the repair phase. Though most common in rotator cuff tendons, calcific tendinopathy can involve virtually any tendon in the body, and unspecified regional pain in those areas can show an incidental focus of calcification, which may or may not be responsible for the symptoms.

Imaging modalities for evaluation of calcific tendinopathy

Conventional radiography

Conventional radiography is generally the first line of imaging modality used to evaluate patient presenting with joint pain and limited range of motions, irrespective of the chronicity. Radiographs are usually obtained in 2 orthogonal views with special views taken depending on the joint wherever required. Classification proposed by Gärtner and Heyer[58] correlates with the histological stage;

- Type 1—Well circumscribed, dense calcification
- Type 2—Ill-defined border, dense calcification
- Type 3—Indistinctive border, relative translucent/cloudy calcification

Specifically, the foci of calcification in the resorptive phase (type 3) may barely be seen on conventional radiography and that is the main limitation of this modality in symptomatic patients. Radiographs can be obtained in follow up to see resorption of calcium (Figure 1).

Ultrasonography

All stages of calcific tendinitis are best evaluated by ultrasonography as compared to conventional radiography and MRI.[59] Ultrasonography shows calcific deposits as foci with hyperechogenic rim and internal hypo- or hyperechogenicity with or without posterior acoustic shadowing depending on the phase of calcification. Based on the ultrasound appearance, 3 types of calcifications are described in calcific tendinopathy (Figure 2).

- Type 1 calcification corresponding to formative and resting phase appear as hyperechoic foci with distinct distal acoustic shadowing like a gallstone.
- Type 2 calcification corresponding to early resorption has faint distal acoustic shadowing.
- Type 3 calcification corresponding to established resorptive phase shows no distal acoustic shadowing.[56]

There is usually no neovascularity within and surrounding the calcification in the calcific phase, but as the calcium undergoes resorption with development of surrounding inflammatory changes, some Doppler signals suggesting neovascularity can be seen in the periphery of the calcium fragments. Resorption of calcium and Doppler signal findings correlate strongly with pain in the affected region. The literature shows that the patients with fragmented and cloudy calcification may have significantly worse pain scores and Doppler grades.[60] The incidental recognition of a coexisting tear is not unusual in calcific tendinopathy (Figure 3). Imaging appearance determines potential treatment of a particular focus of calcification such as lavage, barbotage, or surgical decompression. Defining the stage of calcification helps avoid unnecessary and often long-term physiotherapy and

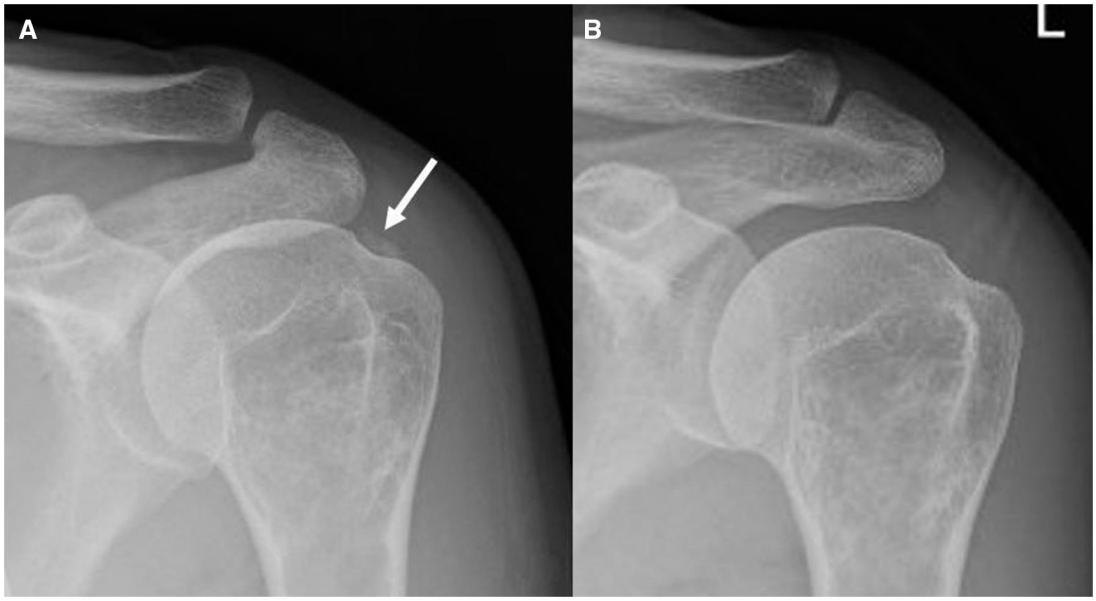

Figure 1. Conventional frontal radiograph of the left shoulder showing faint calcification (arrow) at the foot print of supraspinatus tendon (A). Repeat radiograph 6 months later shows complete resolution (B).

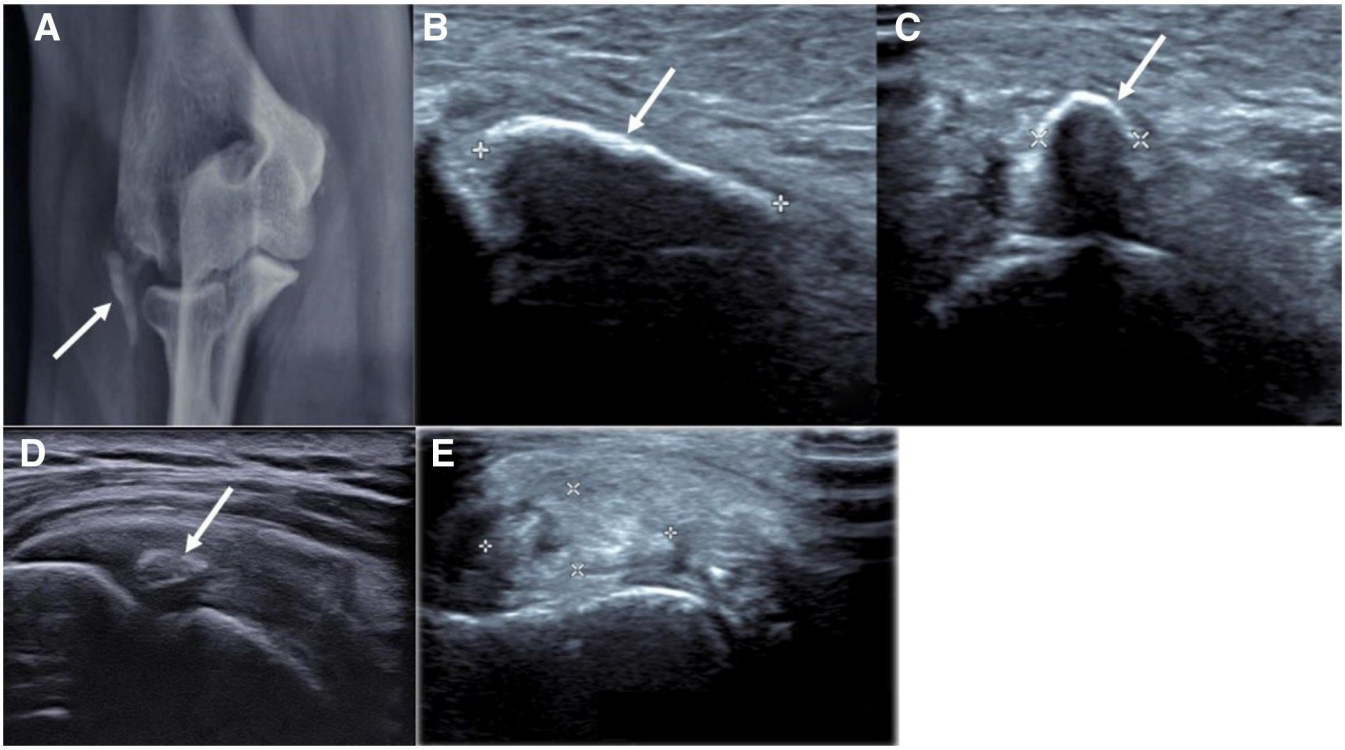

Figure 2. Three types of calcifications; Type 1 calcification (A, B, and C), conventional radiograph (A) shows distinct well circumscribed dense calcification (arrow), ultrasound in long axis (B) and short axis (C) showing well defined hyperechoic focus with distinct distal acoustic shadowing like a gallstone (arrow) in common extensor tendons of the elbow. Type 2 calcification (D) Ultrasound showing Type 2 calcification (arrow) in supraspinatus tendon corresponding to early resorption having faint distal acoustic shadowing. Type 3 calcification (E) grey scale ultrasound showing Type 3 calcification (calipers) corresponding to established resorptive phase showing no distal acoustic shadowing.

helps introducing an alternative effective treatment option, depending on the stage of the disease.[60]

Computed tomography

Computed tomography (CT) is superior to radiography when evaluation of complex sites is required. It is also superior to ultrasound to see intraosseous migration of calcium. It is not a routine practice to consider CT *per se* to detect or monitor calcific tendinopathy. The radiation exposure limits its use and most of the clinical queries related to calcific tendinopathy are generally answered by other imaging modalities like radiography, ultrasonography, and MRI.

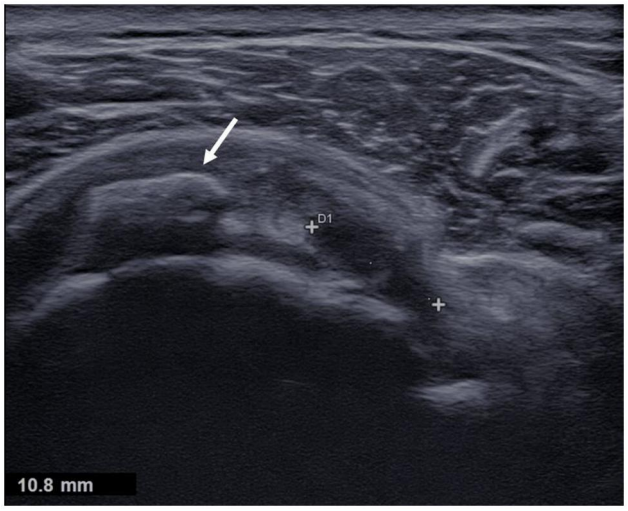

Figure 3. Ultrasound image of the shoulder in transverse plane showing a focus of calcification (arrow) within the supraspinatus tendon with adjacent full thickness tear of the supraspinatus tendon fibres (calipers).

Magnetic resonance imaging

Calcium appears as signal void on all the pulse sequences of MRI and so its visualization can be limited in the hypointense tendons, nevertheless tendon heterogeneity and oedema of the surrounding tendon often helps highlight the calcium focus (Figure 4). Nörenberg et al[61] explored the diagnostic performance of susceptibility-weighted imaging and standard pulse sequences for identification of calcium in cases of calcific tendinopathy and compared them with conventional radiography; and, they found susceptibility weighted imaging to be reliable in identifying calcium with slight overestimation of size. Zubler et al[62] explored the role of MR arthrography to diagnose calcific tendinopathy in rotator cuff tendons and found it to be unreliable as normal hypointense areas in the tendons could falsely be interpreted as calcification and recommended that MRI should not be interpreted without correlating radiographs. MRI can show associated rotator cuff tears, bursitis, and can potentially show the marrow oedema in cases of intraosseous migration of calcium. Nonetheless, MRI is good in determining acuity but not so accurate in measuring the size of calcification. Table 2 summarizes the imaging appearance of calcific tendinopathy of different stages on various imaging modalities.

Migration of calcium in calcific tendinopathy

Intrabursal migration

It is the most common pattern of calcium migration in a case of calcific tendinopathy in resorptive phase. Intrabursal migration in subacromial bursa in a case of rotator cuff calcific tendinopathy is well described in literature.[57,63] Patient may present with acute pain and swelling over rotator cuff with painful limitation of motion. Other peri-tendinous bursae may also get affected for example trochanteric bursa, retrocalcaneal bursa, pre-patellar/infra-patellar bursa etc. (Figure 5). On conventional radiography, amorphous calcific densities can be seen in the expected location of the tendon as well as bursa with soft tissue swelling and obliteration of fat planes between the tendon, muscles, bursa, and subcutaneous fat signifying ongoing inflammation. Calcium-fluid level may also be seen rarely if the radiograph is acquired in position where calcium migrates in the dependent location confirming the intrabursal location of the calcium. On Ultrasound examination, bursal distention with anechoic fluid containing hyperechoic foci of calcium can be easily seen in addition to the confirmation of calcific tendinopathy in the adjacent tendon as inciting factor. Sometimes the bursa gets distended with homogenous hyperechoic fluid giving an appearance of milk of calcium (Figure 6). By applying gentle pressure with an ultrasound probe, calcium mixed fluid can be seen moving from one site to the other. Fluid calcium level can also be visualized on ultrasound. Bursal inflammation appears as thickening of wall of the bursa with increased vascularity which can be easily appreciated on power Doppler study. MRI can show foci of hypointensity (calcium) located within the tendon and bursa with inflammation appearing as hyperintensity on T2W fat suppressed sequence. Chemical bursitis due to release of calcium into the bursa following lavage or barbotage of the calcific tendinopathy is well recognized and hence such procedures are usually concluded with injection of corticosteroids into the bursa to deal with postprocedural inflammation.[64]

Intraosseous migration

Intraosseous migration of calcium to the adjacent bones is not infrequent and is described in the radiology literature. The pathological mechanism of migration include inflammation at the enthesis and mechanical traction by the tendon. Hayes et al[65] first reported the radiographic evidence of bone erosion in 5 cases of calcific tendinopathy at unusual locations (pectoralis major, gluteus maximus, and adductor magnus insertions) with histological evidence of calcium migration into the cortex of the underlying bone. Ultrasound examination has inherent limitation in visualizing intraosseous migration of calcium, however, evidence of calcific tendinopathy with underlying cortical erosions and extension of calcium in the erosion can be seen on ultrasound scan. MRI, CT, and radiography can help to identify intraosseous migration of calcium; MRI in addition can show associated marrow oedema which may correlate with the patient's symptoms (Figure 7).

Intramuscular and musculotendinous junction migration

Intramuscular migration of calcium is very rare and is described only in some case reports in the literature.[54] The pathological mechanism of intramuscular migration is tracking of calcium into the musculotendinous junctions and muscle bellies along delaminating intrasubstance tendon tears. Ultrasound can easily demonstrate hyperechoic calcification in the background of hypoechoic muscles (Figure 8) except in cases of severe fatty atrophy of muscles where it can become difficult to visualize. Doppler can show increased vascularity surrounding the calcification signifying ongoing inflammation. Conventional radiography and MRI can help in confirming the ultrasound imaging findings (Figure 9). It is important to be mindful of intramuscular migration of calcium and related chemical myositis which can potentially be mistaken for other inflammatory and neoplastic pathologies to avoid unnecessary apprehension.

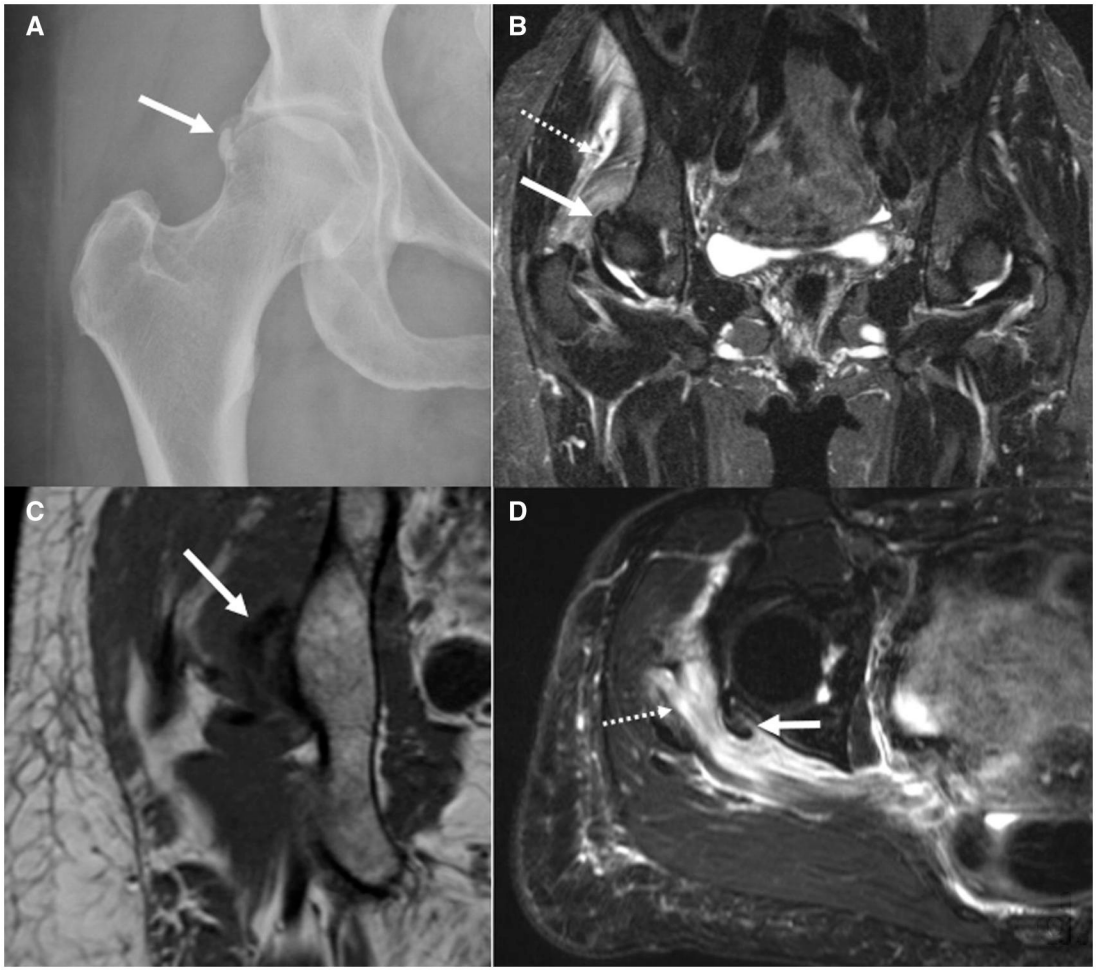

Figure 4. Conventional frontal radiograph of the right hip (A), coronal STIR and coronal T1W MRI (B and C), and axial fat-suppressed proton density MRI images (D) showing a curvilinear focus of calcification (white arrow) along the path of the rectus femoris tendon appearing as hyperdense structure on radiograph and as signal void on MRI. Extensive oedema and calcium debris (dashed arrow) of the adjacent gluteus minimus is seen in keeping with associated chemical myositis.

Table 2. Summary of the imaging appearance of calcific tendinopathy of different stages on various imaging modalities.

Stages of calcification	Appearance on different imaging modalities			
	Conventional radiography	Ultrasonography	Computed tomography	MRI
Pre-calcific stage	Ill-defined border, faint calcification	Hyperechoic foci with faint distal acoustic shadowing	Faint hyperdense foci	Faint hypointensity on both T1 and T2W sequences
Calcific stage	Well circumscribed, dense calcification	hyperechoic foci with distinct distal acoustic shadowing like a gallstone	Distinct hyperdense foci	Distinct hypointensity on both T1 and T2W sequences
Postcalcific/repair stage	Indistinctive border, relative translucent/cloudy calcification, mostly barely visible	Hyperechoic foci with faint/no distal acoustic shadowing	Faint hyperdense foci	Faint hypointensity on both T1 and T2W sequences with adjacent oedema, best visualized on fat-suppressed T2W sequence

Other soft tissue migration

Calcium from calcific tendinopathy can migrate to the adjacent soft tissue planes such as sub-bursal space, rotator interval, biceps tendon sheath/other tendon sheaths etc. (Figures 10-12 and Figure S13). Intra-articular migration of calcium is not described in the radiological literature, however, in author's experience reactive effusion in the adjacent joint space is often seen and it is possible due to intra-articular release of calcium. There is evidence of enough literature on soft tissue migration of calcium from longus colli calcific tendinopathy into the retropharyngeal space, mimicking retropharyngeal abscess.[7,8]

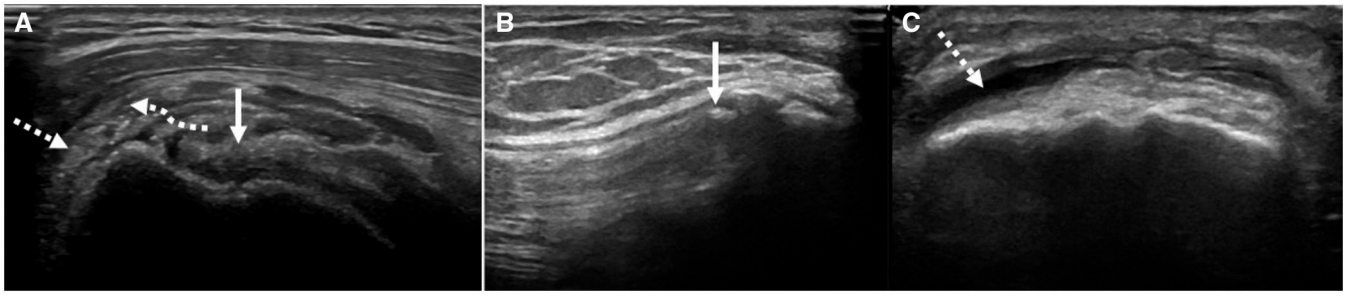

Figure 5. Calcific tendinopathy with bursal migration: (A) ultrasound image showing type 3 calcification in the supraspinatus tendon (white arrow) with migration of calcium (curved dashed arrow) into the subdeltoid bursa (dashed arrow), (B) grey scale ultrasound image of quadriceps tendon showing calcific tendinopathy (white arrow) with reactive bursal thickening with bursal effusion (dashed arrow) suggesting prepatellar bursitis (C).

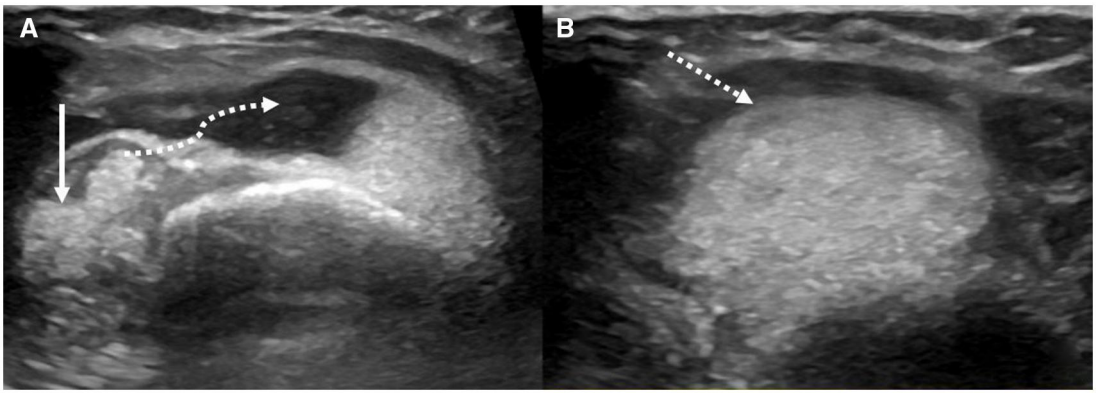

Figure 6. Ultrasound images of the shoulder in transverse plane (A and B) showing supraspinatus calcific tendinopathy (white arrow) with migration of calcium (curved dashed arrow) into the subacromion subdeltoid bursa with homogenous hyperechoic bursal fluid giving an appearance of milk of calcium (dashed arrow), please note fluid-fluid level due to sedimented calcium in the dependent area of the bursa.

Pitfalls and mimics of calcific tendinopathy and calcium migration

Calcific enthesopathy

Calcific enthesopathy is the common pitfall and mimic of calcific tendinopathy. It is the result of degenerative process and is characterized by deposition of linear lamellar calcification at bone tendon interface with underlying cortical irregularity (Figure S14). Unlike calcific tendinopathy, calcific enthesopathy does not resolve over time; in fact, it can progress to ossification or to erosion of the underlying bone.[66]

Avulsion fracture at tendon attachment

Avulsion fractures generally present in post-traumatic settings. Radiographically, a fragment of bone with trabecular markings will be seen instead of dense calcification which is devoid of any trabecular markings and donor site can also be identified, however, in atypical situations, the bone fragment fails to unite and may mimic a calcium focus along the perienthesial tendons (Figure S15).[67]

Tendon calcifications in calcium pyrophosphate dihydrate crystal deposition disease

Calcium deposition in calcium pyrophosphate dihydrate (CPPD) deposition disease is small in size and generally in linear fashion along the tendon fibres. Calcium deposition in the fibrocartilage tissues or articular hyaline cartilage can be identified on imaging and it helps in confirming the diagnosis of CPPD deposition disease over calcific tendinopathy. Calcium deposition can affect superficial joints like acromioclavicular joint (Figure S16).[68]

Gout

Monosodium urate crystals can deposit in tendons, cartilage, and soft tissues as hyperechoic foci with distal acoustic shadowing. This condition can be differentiated from calcific tendinopathy by presence of typical clinical, pathological, and radiological profile of gout (Figure S17).[69]

Neoplastic intraosseous or soft tissue lesions with calcification

Intraosseous and intramuscular migration of calcium can incite an inflammatory response which can mimic an intraosseous or soft tissue malignancy. However, care must be exercised not to disregard presence of neoplastic lesions containing calcification and surrounding reactive oedema such as aggressive intraosseous chondroid lesions, osteogenic soft tissue sarcoma within muscles etc.[70-72]

Others

Dystrophic muscular calcification in tissues because of ischemic, inflammatory, traumatic, or neurological causes can mimic intramuscular migration of calcium; but differentiation becomes easy when complete medical history of the patient is taken into consideration before concluding migrated calcium (Figure S18).[73,74]

Calcific tendinopathy might be erroneously identified as immature myositis ossificans. While mature myositis ossificans typically exhibits distinctive zonal mineralization after around 6-8 weeks, immature myositis ossificans may manifest with faint calcifications. Follow-up radiographs prove most valuable in distinguishing between the 2 conditions, as

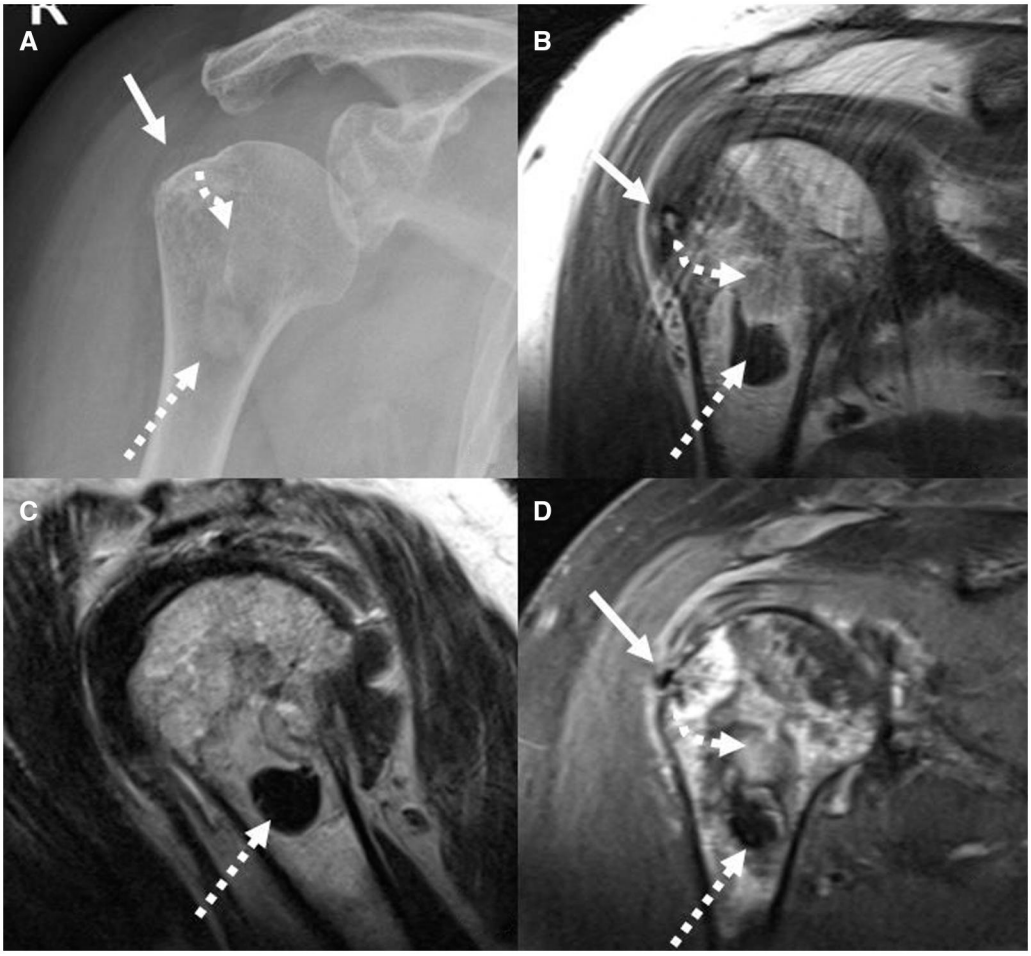

Figure 7. Conventional frontal radiograph of the shoulder (A) showing faint calcification in the region of supraspinatus tendon suggesting calcific tendinopathy (white arrow) and there is another well define focus of calcium within the proximal humerus (dashed arrow) suggesting intraosseous migration (curved dashed arrow). Corresponding coronal MRI T1W (B), sagittal T2W (C), and coronal fat-suppressed proton density (D) images showing supraspinatus calcific tendinopathy and confirms the presence of intraosseous calcium. The path of migration is well depicted (curved dashed arrow) with extensive reactive bone marrow oedema (D).

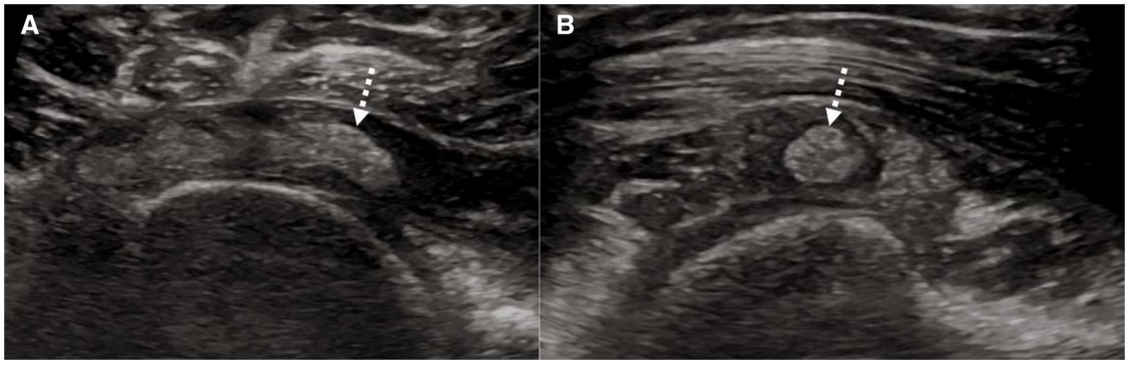

Figure 8. Ultrasound images of the shoulder showing longitudinal (A) and transverse (B) images of the subscapularis tendon showing calcium extension (dashed arrow) from the tendon to its corresponding muscle belly in keeping with intramuscular migration of calcium.

calcific tendinopathy is likely to remain unchanged (in the resting phase) or resolve (in the resorptive phase), whereas myositis ossificans will undergo maturation over time.[75]

Management

Calcific tendinitis is considered a self-healing condition in which calcifications resolve spontaneously. Management options of calcific tendinopathy include conservative measures such as non-steroidal anti-inflammatory drugs to alleviate pain and inflammation followed by physiotherapy to increase range of movement of the joint. In cases where patients do not progress spontaneously from the calcific to the postcalcific stage and experience recurrent pain and disability, nonoperative treatments are initially recommended. These may involve therapeutic exercise, nonsteroidal

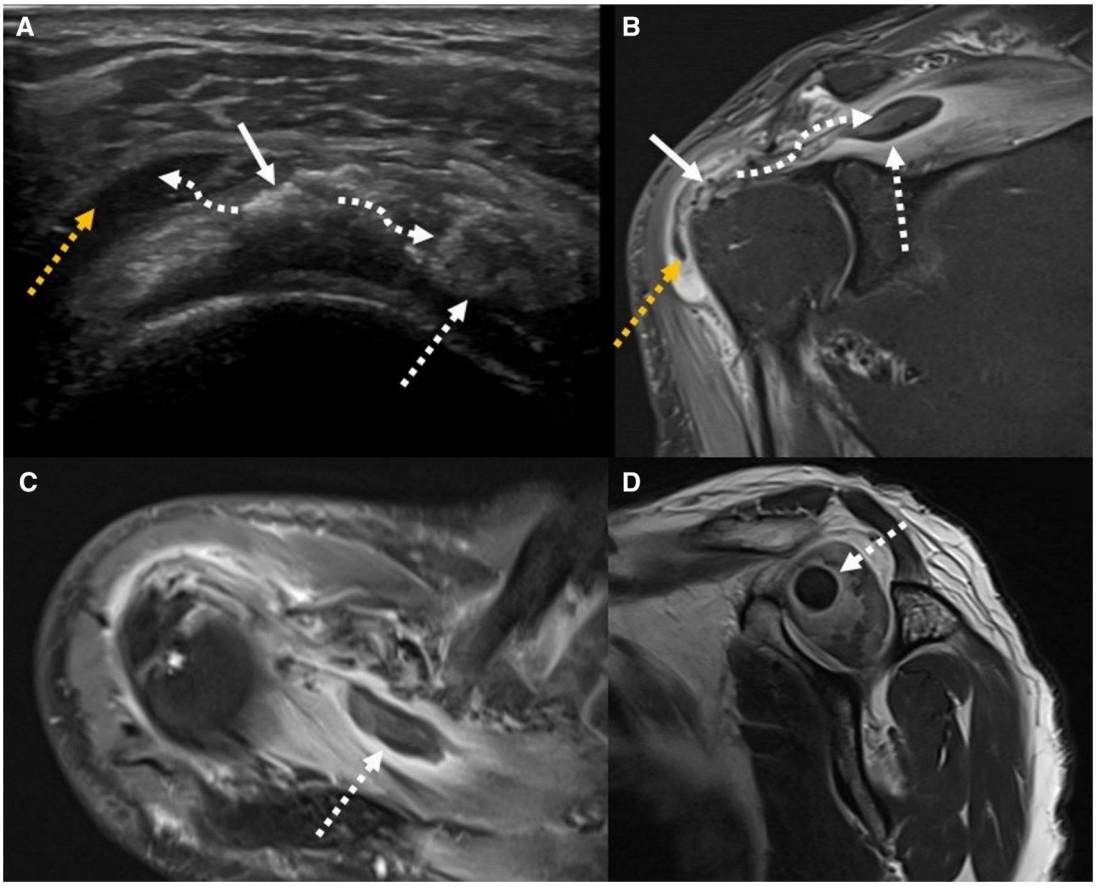

Figure 9. Supraspinatus calcific tendinopathy with intrabursal and intramuscular migration; Ultrasonography (A) showing calcification in the supraspinatus tendon suggesting calcific tendinopathy (white arrow) with migration of calcium (curved dashed arrows) into the bursa (yellow dashed arrow) and muscle (white dashed arrow), MRI T2W fat suppressed coronal (B), fat-suppressed T2W axial (C), and T2W sagittal (D) sequences showing supraspinatus calcific tendinopathy as signal voids (white arrow) with migration of calcium (curved dashed arrow) into the bursa (yellow dashed arrow) and muscle belly (white dashed arrows).

anti-inflammatory drugs for pain relief, extracorporeal shockwave therapy, and corticosteroid injections. For patients not responding to nonoperative treatments, the removal of calcification can be facilitated through ultrasound-guided needle lavage. This minimally invasive procedure utilizes 1 or 2 needles for lavage, with or without aspiration of calcium deposits, guided by an ultrasound probe for real-time imaging. This is usually combined with intra-bursal injection of corticosteroid to prevent further inflammation. Sonography offers high sensitivity, allowing clinicians to visualize and localize rotator cuff calcifications accurately without exposing patients to harmful radiation. The procedure enables fragmentation and removal of calcifications through saline lavage (barbotage) (Figure S19). Ultrasound also allows easy assessment and documentation of calcification size and softness, facilitating objective comparisons. The surrounding tendon's status, including procedure-related tears, can be accurately evaluated, with such tears being infrequent. The limited use of ultrasound-guided needle lavage may be attributed to a lack of comparative effectiveness studies with alternative interventions and the absence of a standard of care with long-term findings.

Sconfienza et al[76] in their randomized controlled trial found that in the treatment of calcific tendinopathy of the rotator cuff, addition of warm saline in the barbotage appears to reduce procedure duration and improve calcification dissolution while reducing the frequency of postprocedural bursitis. Extracorporeal shock wave therapy and Kinesio taping can also be tried that elicit an analgesic, anti-inflammatory effect and promote tissue regeneration.[77,78] Surgical decompression of the calcium is usually reserved for resistant cases.[79] Medina-Gandionco et al[80] have described the use of Acetic Acid Iontophoresis for successful treatment of rotator cuff calcific tendinopathy. No prescribed specific treatment regimen exists for migrated calcium. The migrated calcium should be treated based on the destination of deposition, that is, bursal calcium can be easily aspirated, whereas intraosseous calcium is usually left alone with supportive conservative treatment if symptomatic.

Conclusion

Calcific tendinopathy is a common and well-documented ailment in the literature. Although common, the natural history, aetiology, and progression of calcific tendinitis is poorly understood. The treatment options include conservative and interventional measures, however, these measures cannot be applied as a blanket and often tailored depending upon the stage/phase of the disease. Out of the recognized stages of the disease, the resorptive stage causes utmost symptoms when the calcium is rather soft and unstable. During this stage, the calcium may migrate beyond expected resorption and get

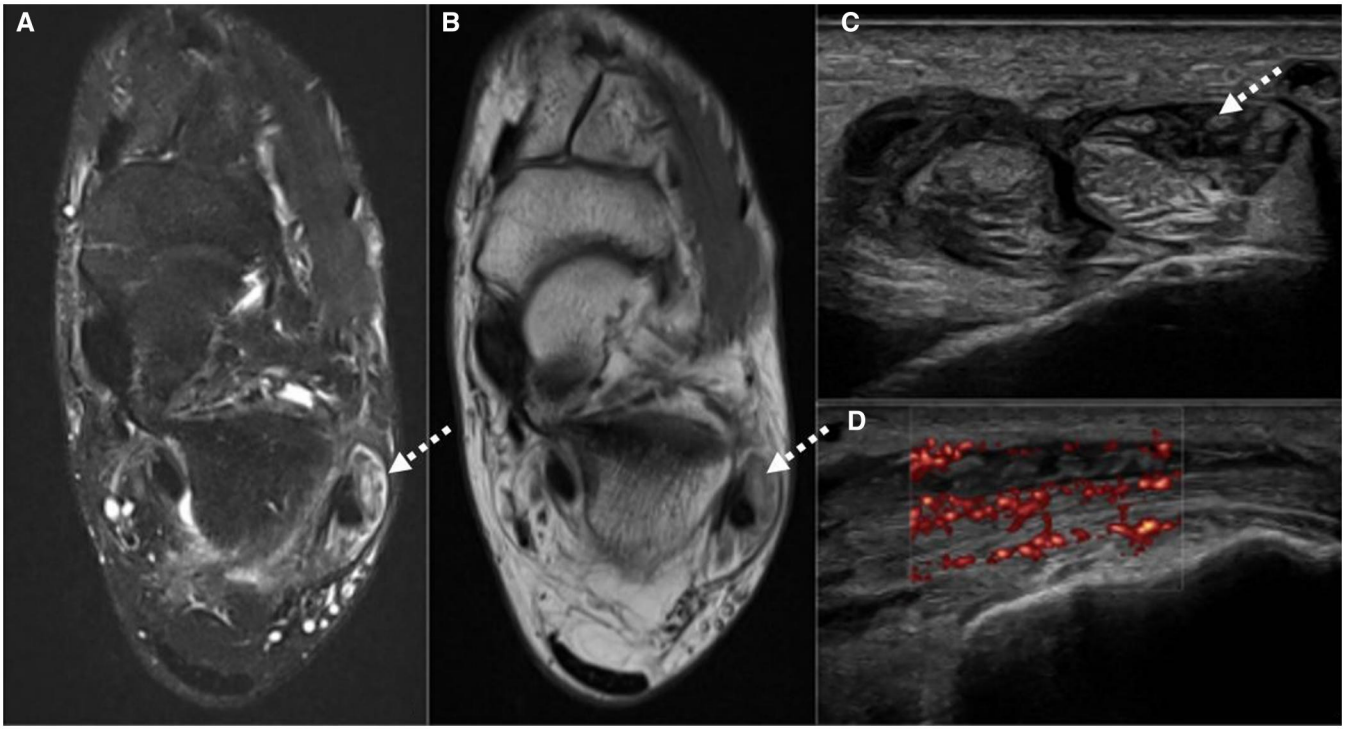

Figure 10. Axial MRI fat-suppressed proton density (A) and T1W (B) images of the ankle demonstrating low signal intensity calcium depositions (dashed arrows) within the peroneal tendon sheath with associated tenosynovitis. Corresponding ultrasound images in short axis (C) demonstrating echogenic material in peroneal tendon sheath (dashed arrow) and long axis image (D) shows neovascularity on power Doppler in keeping with tenosynovitis (D).

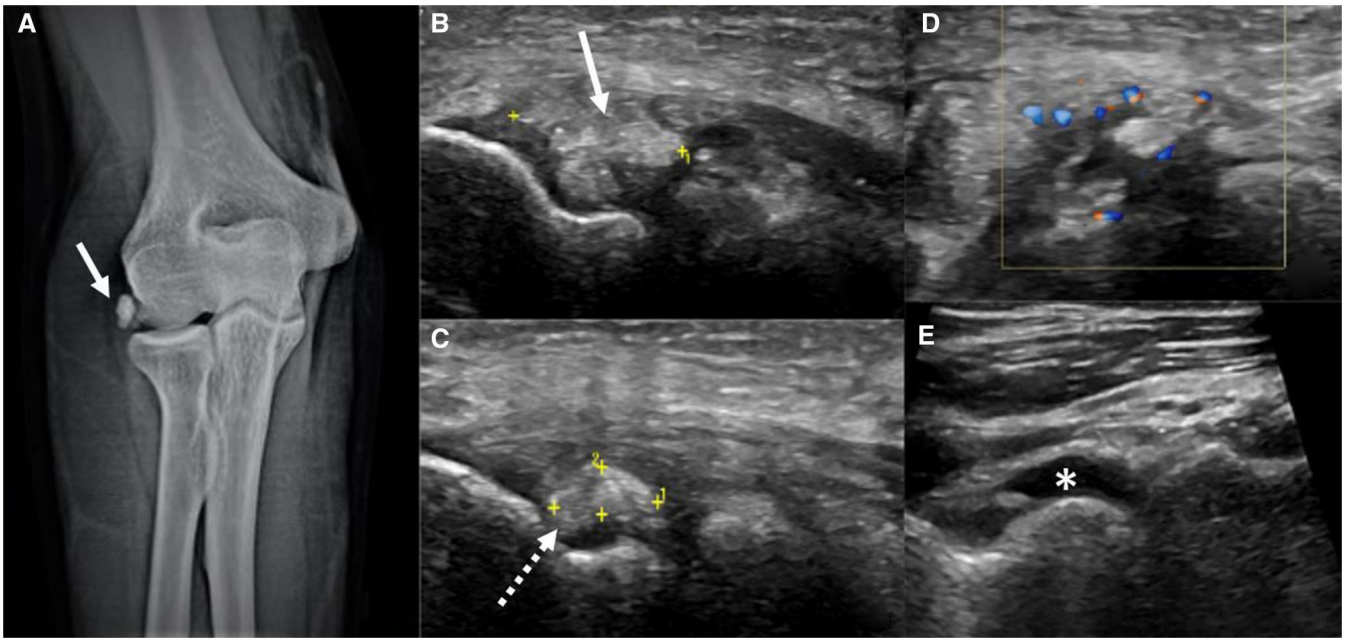

Figure 11. Conventional frontal radiograph of the elbow (A) showing amorphous calcific densities in the region of common extensor origin (white arrow) suggesting calcific tendinopathy. Corresponding ultrasound images in longitudinal plane (B, C, and E) and in transverse plane (D) showing type II/III calcification in the common extensor tendon (white arrow), calcium migration can be seen through the deficient radial collateral ligament (dashed arrow) with adjacent neovascularity (D) and reactive radiocapitellar joint effusion (asterisk) suggesting ongoing inflammation.

deposited in the adjacent tissues contiguous with the calcium focus. The common destinations include bursal migration, intraosseous migration, muscular migration, and other less common sites of migration. Such atypical presentations can lead to dilemma in the diagnosis, prolongation of the diagnostic pathway, unwarranted apprehension, and treatment delay. Radiologists' role in this situation is to correctly recognize the imaging findings of atypical presentations of calcific tendinopathy and prevent unnecessary diagnostic and interventional studies.

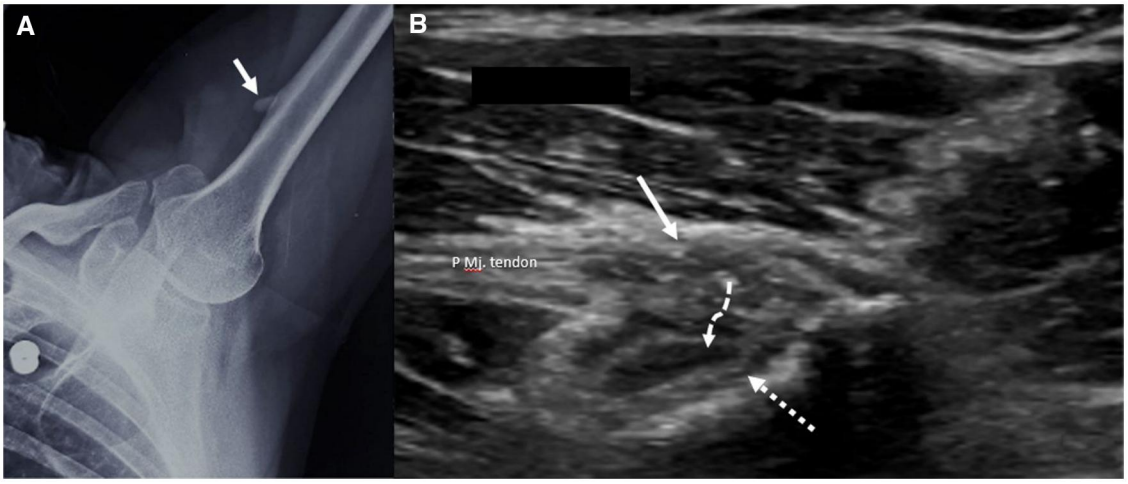

Figure 12. Conventional axial view radiograph of the shoulder (A) showing a small, well-defined focus of calcification at the insertion site of the pectoralis major tendon (white arrow). The ultrasound images of the proximal arm in longitudinal plane (B) show calcific foci at pectoralis major insertion site (white arrow) with potential migration (curved dashed arrow) into floor of the bicipital groove (dashed arrow).

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Supplementary material

Supplementary material is available at *BJR* online.

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Conflicts of interest

None declared.

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Review