

Case Report

Atypical site of calcific tendinopathy around the shoulder with Pectoralis major insertion

Raghav Aggarwal¹, Riya Samanta², Geetanjali Nanda³, Nafisa Shakir Batta²

¹Department of Radiology, Dr. ML Aggarwal Imaging Centre Pvt. Ltd, ²Department of Radiology, Mahajan Imaging and Labs, Safdurjung Hospital Sports Injury Centre, ³Department of Radiology, Mahajan Imaging and Labs, SDA, New Delhi, India.

*Corresponding author:

Riya Samanta,
Department of Radiology,
Mahajan Imaging and Labs,
Safdurjung Hospital Sports
Injury Centre, New Delhi,
India.

samanta.ria1011@gmail.com

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ABSTRACT

Calcium deposit tendinopathy of the shoulder is a commonly observed condition, often presenting as non-traumatic shoulder pain. It typically resolves spontaneously, characterized by the deposition of calcium hydroxyapatite at the tendon insertion site, followed by natural regression and subsequent tendon healing. Proper identification of the radiological characteristics of this condition is crucial to avoid unnecessary procedures, such as biopsy. This report presents the case of a 26-year-old female diagnosed with calcific tendinitis affecting the pectoralis major tendon at its humeral insertion—a rare site of involvement, with only a few cases reported in the literature.

Keywords: Calcium deposit tendinopathy, Calcium deposit-related tendinitis, Calcium hydroxyapatite, Hydroxyapatite deposition disease, Shoulder

INTRODUCTION

Calcium deposit tendinopathy is a frequent cause of atraumatic shoulder pain. It represents a self-limiting disorder of uncertain etiology, marked by calcium hydroxyapatite deposition at the tendon insertion, followed by gradual regression and tendon repair.^[1] The supraspinatus tendon is most often affected, though less typical sites such as the pectoralis major, biceps brachii, and deltoid tendons may also be involved.^[2] In particular, tendinitis of the pectoralis major at its humeral insertion is exceedingly uncommon, with only a handful of cases reported.^[3-8]

At these unusual sites, the abnormality may mimic a neoplastic lesion because of its appearance, surrounding inflammation, and potential for cortical erosion or destruction, leading to misdiagnosis and unnecessary procedures such as biopsy.^[9] The underlying mechanism remains uncertain, but proposed explanations include tendon degeneration, necrosis, trauma, or metaplasia of hypoxic tendon insertion into fibrocartilage, ultimately resulting in calcification.^[8] A cell-mediated process has also been suggested, with the disease progressing through four distinct phases:^[10]

1. Pre-calcific stage: Fibrocartilaginous transformation of the tendon due to local hypoxia.
2. Calcific stage: Cellular secretion of calcium producing chalk-like deposits, readily visible on radiographs.
3. Resorptive stage: Liquefaction of deposits with acute inflammation, bursitis, cortical erosion, and marrow edema, producing the most severe pain and radiographic appearances mimicking aggressive disease.

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4. Reparative stage: Restoration of the tendon collagen structure by fibroblasts, with residual thin or linear calcifications sometimes persisting on imaging.

Clinically, patients present with pain and focal tenderness, often correlated with calcific deposits seen on X-ray. Ultrasonography (USG) helps localize the symptomatic calcification and allows image-guided treatment, such as barbotage. Computed tomography (CT) and magnetic resonance imaging (MRI) provide additional detail, showing cortical erosion, intraosseous calcification, and marrow edema.^[9] We describe an unusual presentation of acute calcific tendinitis at the humeral insertion of the pectoralis major tendon.

CASE REPORT

A 26-year-old woman presented with a 1-week history of severe pain in the left shoulder and upper arm, without preceding trauma. The pain developed spontaneously, worse in the mornings and after exertion. Shoulder motion was preserved, but internal rotation and end-range adduction/abduction were painful. Neurovascular status was intact, and no swelling, warmth, or skin changes were noted. A provisional diagnosis of rotator cuff pathology was made, and the patient underwent USG and MRI.

USG showed a lobulated calcific deposit at the humeral insertion of the pectoralis major tendon ($7.3 \times 6.6 \times 6.0$ mm) with marked posterior acoustic shadowing and surrounding edema, while the tendon remained intact. Mild supraspinatus thickening with altered echotexture indicated tendinosis [Figure 1]. There was a mild increase in Doppler vascularity with biphasic low-amplitude arterial flow. No frank hypervascularity was evident.

MRI confirmed the calcification, hypointense on all sequences with prominent blooming on gradient-echo, consistent with calcific tendinitis. Edema was seen on proton density fat-saturated (PDFS) images in the adjacent tendon and soft tissues. The pectoralis major tendon was continuous, with feathery myofascial edema in the distal anterior deltoid and intratendinous PDFS hyperintensity in the distal supraspinatus tendon, suggestive of mild tendinosis [Figure 2]. Other rotator cuff tendons were intact, with no muscle atrophy or fatty infiltration. The glenohumeral joint and labrum were normal. The patient was managed conservatively with analgesics, showing good recovery at 6 weeks.

DISCUSSION

Calcium deposition in the periarticular soft tissues of the shoulder is frequently seen, particularly within the joint capsule, tendons, ligaments, and bursae. Deposits may develop at the attachment sites of several muscles, including the pectoralis major, gluteals, gastrocnemius, iliopsoas, and vastus lateralis tendons.^[5] The condition is most often encountered

in younger women and typically arises in the absence of a clear traumatic insult. The local inflammatory response to the calcific deposits produces pain and point tenderness.^[11]

Characteristic imaging clues include the recognition of rounded or lobulated calcific foci, demonstration of the deposit within a tendon, adjacent to a joint, or within a bursal sac, together with the absence of a soft-tissue mass or joint effusion, and an abrupt onset of symptoms.^[12]

Multiple explanations have been advanced for how calcific deposits arise. Some authors favor a degenerative-inflammatory process with subsequent necrosis of tendon fibers, whereas Uthoff *et al.*^[10] proposed that the process begins with focal metaplasia of tendon tissue. In the supraspinatus tendon, a relatively avascular zone is particularly prone to hypoxia, which may trigger fibrocartilaginous transformation. Chondrocytes within this metaplastic tissue then promote calcium deposition, a finding best appreciated on radiographs and CT scans. During the resorptive stage, phagocytic activity leads to dissolution of the deposits and neovascularization within the tendon, resulting in pain and tenderness. MRI is especially useful in this phase because of its ability to demonstrate inflammatory edema. Once the deposits are resorbed, the tendon generally returns to its normal structure.^[10]

Spontaneous resolution is the rule, typically within 6–10 weeks.^[7,11,13] In most patients, symptomatic management with non-steroidal anti-inflammatory drugs is sufficient. Cortical destruction is unusual in this disorder; if

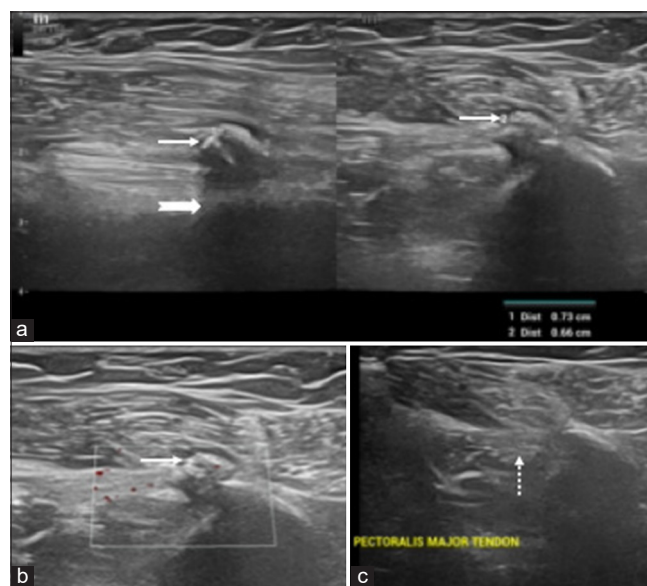


Figure 1: (a and b) Ultrasonographic images showing lobulated calcification (white arrows) with prominent posterior acoustic shadowing (notched white arrow) at the insertion of pectoralis major tendon into the humerus. There was mild increase in Doppler vascularity with biphasic low amplitude arterial flow. No frank hypervascularity seen. (c) The pectoralis major tendon otherwise appears intact (dashed white arrow).

present, it should raise concern for alternative diagnoses such as metastasis. Importantly, metastatic disease would not be associated with tendon calcification.^[14]

Changes in the marrow are poorly depicted on plain radiographs, so MRI and CT offer better characterization, though correlation with radiographic findings remains essential.^[5] USG is particularly helpful for pinpointing the symptomatic deposit and guiding interventions such as

barbotage. Familiarity with the imaging appearance of this entity is critical to avoid misinterpretation and unnecessary invasive procedures such as biopsy.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for this case is mentioned in Table 1 under Differential diagnosis.

Table 1: Differential diagnosis

S. No.	Differential diagnosis	Differentiating features
1.	Traumatic bony avulsion of the pectoralis major tendon	The clinical presentation is consistent with an injury commonly observed in individuals involved in weightlifting or bodybuilding and is often associated with a specific traumatic event.
2.	Conditions that cause periarticular calcification such as collagen vascular disease, hyperparathyroidism, renal osteodystrophy, hypervitaminosis D, milk alkali syndrome, and sarcoidosis	Biochemical workup helps in differentiation.
3.	Malignant bone tumour sharing the atypical location and extended cortical erosion	Often associated with a soft-tissue mass, more prolific osteolysis and erosion, not seen in calcific tendinitis.
4.	Myositis ossificans	Formation of new bone within the muscle tissue following traumatic injury, whereas calcific tendinitis is the deposition of calcium within a tendon and is of non-traumatic etiology. The initial stages of myositis, however, show overlap features; assessment of clinical history and temporal evolution of symptoms is key.

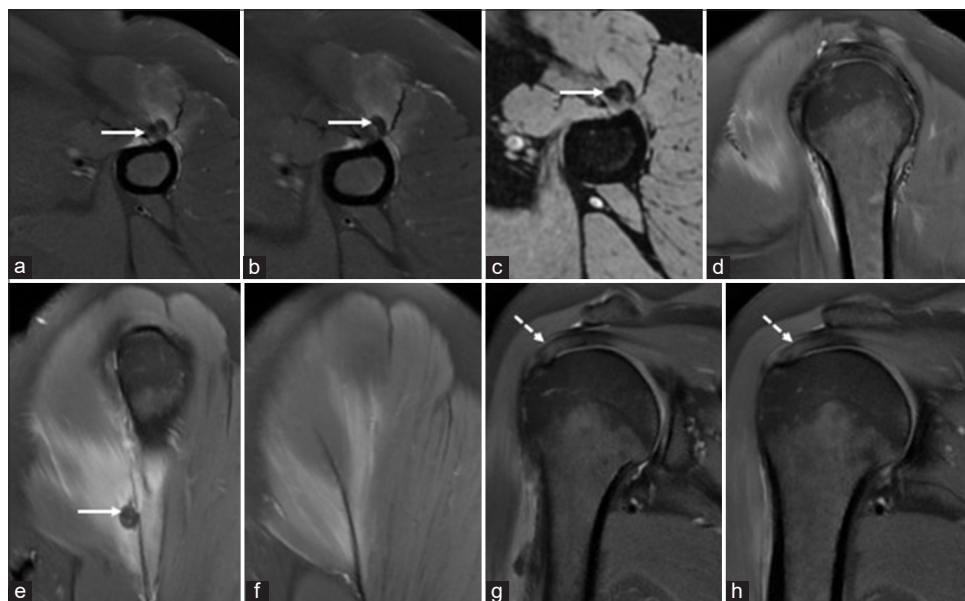


Figure 2: Magnetic resonance (MR) imaging of the left shoulder and upper arm. (a and b) axial proton density fat-saturated (PDFS) MR images reveal calcification (white arrows) towards the insertion of pectoralis major tendon into the humerus appearing hypointense on all sequences. Edema is seen in the surrounding tendon and soft tissues as well as feathery myofascial edema in the distal anterior fibers of the deltoid muscle. (c) Axial gradient image showing prominent blooming at the site of calcification (white arrow). (d, e and f) Serial sagittal PDFS images from medial to lateral showing calcification (white arrow) at the insertion of pectoralis major tendon into the humerus with edema in the surrounding tendon and soft tissues as well as feathery myofascial edema in the distal anterior fibers of the deltoid muscle. (g and h) coronal PDFS images showing intrasubstance hyperintense signal in the distal supraspinatus tendon, suggesting mild tendinosis (dashed white arrows).

CONCLUSION

We present the USG and MRI findings of calcific tendinitis involving the pectoralis major tendon. A comprehensive biological evaluation to identify possible systemic causes is crucial and should be standardized. Diagnostic biopsy can generally be avoided, as radiological imaging confirms the benign nature of the condition. Surgical intervention may be considered in specific cases to facilitate the rehabilitation process.

TEACHING POINTS

1. Calcium deposit-related tendinitis of the pectoralis major at its insertion can be misleading
2. A biological assessment to look for a systemic etiology is mandatory and standardized. Biopsy is not required; radiological examinations are sufficient to make a diagnosis
3. Surgical management may be offered in specific cases to shorten the necessary rehabilitation time.

MCQs

1. Calcium deposit tendinopathy occurs as a result of
 - a) Trauma
 - b) Deposition of calcium hydroxyapatite at the site of insertion of a tendon
 - c) Deposition of bone within the muscle belly
 - d) Hyperparathyroidism

Answer Key: b

2. The most common site of calcific tendinitis in the shoulder is:
 - a) Rotator cuff
 - b) Deltoid
 - c) Pectoralis major
 - d) Coracobrachialis

Answer Key: a

3. The most painful stage of calcific tendinitis is
 - a) Pre-calcific or formative stage
 - b) Calcific stage
 - c) Resorptive stage
 - d) Reparative or post-calcific stage

Answer Key: c

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