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Case Report

Small Calcification, Severe Chest Pain: Pectoralis Major Calcific Tendinopathy Masquerading as Cardiac Emergency

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Abstract:

Background: Chest pain is a frequent cause of emergency department consultations, often requiring exclusion of life threatening conditions. We present a rare case of calcific tendinopathy of the pectoralis major tendon mimicking a cardiac emergency.

Case Presentation: A 70 year old man presented with severe atraumatic left sided chest pain radiating to the shoulder and arm. Initial clinical and laboratory findings excluded cardiac and vascular causes. Computed tomography and ultrasound revealed pronounced calcification at the humeral insertion of the pectoralis major tendon. Conservative therapy was insufficient, while ultrasound guided percutaneous lavage with triamcinolone infiltration led to marked symptom resolution.

Conclusion: Calcific tendinopathy of the pectoralis major represents an extraordinarily rare cause of acute chest pain. Recognition of this condition is essential to avoid unnecessary cardiac work up and to provide targeted therapy.

Key words: calcific tendinopathy; pectoralis major; chest pain; emergency; musculoskeletal disorders

Introduction

Chest pain is one of the most common presenting complaints in the emergency department. After exclusion of acute coronary syndrome, pulmonary embolism, and aortic dissection, musculoskeletal causes are often considered. Calcific tendinopathy is usually localized in the rotator cuff, whereas involvement of the pectoralis major tendon is exceptionally rare. We report a case of acute calcific tendinopathy of the pectoralis major presenting as severe chest pain.

Case Presentation

A 70-year-old man was referred to the emergency department by his primary care physician for refractory left-sided atraumatic chest pain. Symptoms began acutely the prior evening without trigger and worsened overnight. Pain was sharp, persistent (VAS 10/10), and exacerbated by minimal left shoulder movement. Immobilizing the left arm provided slight relief. Pain radiated from the left chest to the entire shoulder, scapula, and arm.

Examination

On arrival the patient was afebrile; blood pressure was 145/97 mmHg (left) and 150/97 mmHg (right), pulse 92/min, and oxygen saturation 93% on room air. Cardiopulmonary examination was unremarkable, with palpable peripheral pulses. Marked tenderness was noted in the left pectoralis major and axillary region. Active and passive left shoulder movement was impossible due to pain. Strength was limited by pain, sensation remained intact, and the skin was normal.

Diagnostics

Electrocardiogram showed normal sinus rhythm without ischemic changes. Laboratory tests including CRP, leukocyte count, hs-troponin, creatine kinase, and D-dimer were all normal. Because of persistent severe pain despite analgesia, CT angiography was performed to exclude aortic dissection. Imaging instead revealed pronounced soft-tissue calcification at the humeral insertion of the left pectoralis major tendon (Figure.1). Ultrasound confirmed the calcification at the insertion site with adjacent inflammatory reaction (Figure.2).

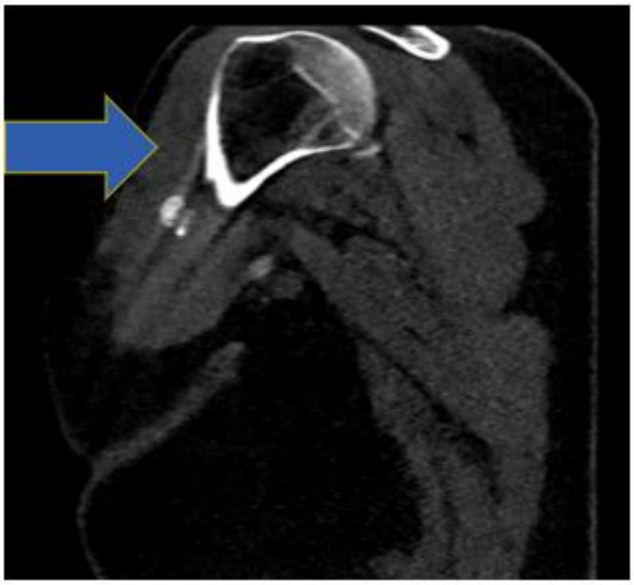

Figure 1: Chest CT: Faint soft- tissue calcification (arrow) at the humeral insertion oft he left pectoralis major muscle

Ultrasound imaging confirmed the location oft the described calcifications at the insertion site oft he left pectoralis muscle (Figure.2).

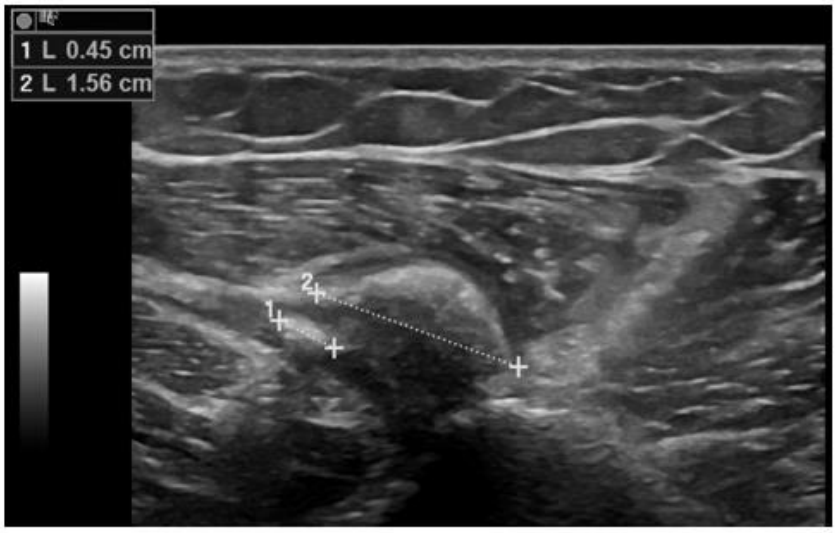

Figure 2: Ultrasound left shoulder: Large calcification in the left ventral region, closely associated with the long head of the biceps tendon (2), most likely at the insertion site of the pectoralis major muscle with adjacent inflammatory reaction (1) in cross-section

The diagnosis of acute calcific tendinopathy oft he left pectoralis major muscle was established.

Therapy and Course

The patient was hospitalized for pain management and initially treated with opioids, non-steroidal anti-inflammatory drugs, and paracetamol. After five days without sufficient improvement, a 7-day course of oral prednisolone (30 mg/day) was initiated but proved ineffective. Subsequently, ultrasound-guided percutaneous lavage (barbotage) combined with local triamcinolone infiltration was performed, resulting in rapid clinical improvement and complete resolution of symptoms.

Discussion

Calcific tendinopathy (synonyms: calcific tendinitis, periarthritis, tendinosis) is characterized by calcium hydroxyapatite crystal deposition within tendons. While most prevalent in the rotator cuff tendons (3-10% population prevalence [2]), 50% of calcifications remain asymptomatic. Among patients with shoulder pain, 7-17% demonstrate calcific tendinopathy [3]. The supraspinatus tendon is affected in approximately 80% of cases, followed by the infraspinatus and subscapularis tendons (10% each). Rare locations include the hip [4], elbow, wrist, knee, and neck (longus colli muscle) [5]. Pectoralis major tendon involvement is exceptionally rare [1], with only few documented cases [6-8]. Presentation with acute, treatment-resistant pain as in our case is unusual.

The etiology remains unknown. Calcifications develop in viable tendons, excluding degenerative processes. No association exists with occupational activities, sports, or trauma. Reported comorbidities include diabetes, thyroid disorders, and nephrolithiasis [10,11]. A genetic predisposition remains unproven. Females are more commonly affected, peak age: 30-50 years.

The pathophysiological mechanisms are not yet fully understood. The disease model proposed by Uhthoff and Loehr (2), describing sequential pathological phases, remains widely accepted today. During the precalcific phase, chondroid metaplasia occurs in tendon tissue,

transforming tenocytes into chondrocytes. These cells subsequently generate the calcium deposits during the formative phase. The triggering mechanism for this process remains unclear. A resting phase follows, where calcium deposits remain stable but may become symptomatic through mechanical interference - for instance, causing clinical impingement symptoms in the subacromial space when larger calcifications are present in the supraspinatus tendon. The resorptive phase features neovascularization and infiltration by macrophages and giant cells that dissolve the calcifications. This process may lead to tendon perforations and extrusion of calcific material into bursae or surrounding soft tissues, potentially triggering intense inflammatory reactions with severe pain. The post-calcific phase completes the cycle with resorption of crystals and tendon remodeling by fibroblasts, ultimately resulting in full structural restoration (restitutio ad integrum).

Diagnosis of calcific tendinopathy is typically straightforward using ultrasound examination of the affected tendon. Conventional radiography also clearly visualizes the calcifications. The clinical course shows considerable variability. The most acute presentation, as seen in our case, is termed acute calcific tendinopathy. Among rotator cuff calcific tendinopathies, 50% become asymptomatic within 3 months either spontaneously or with conservative therapy. An additional 20% of patients require up to one year to achieve pain-free status, while only 30% experience persistent or recurrent symptoms [12].

Therapeutic decisions must account for the condition's strong tendency toward spontaneous resolution. Initial treatment is conservative, employing NSAIDs and other analgesics along with possible physical therapy measures. When significant inflammatory reactions are present (e.g., subacromial bursitis), local steroid injections combined with anesthetics can be highly effective. For persistent symptoms, additional interventions include extracorporeal shockwave therapy (ESWT), ultrasound-guided percutaneous lavage (UGPL), and arthroscopic calcific deposit removal (ACDR). UGPL has emerged as the preferred method due to demonstrated superiority over ESWT (13), while ACDR is reserved for rare cases refractory to UGPL.

Key Clinical Points

- Calcific tendinopathy of the pectoralis major represents an extraordinarily rare cause of acute-onset chest pain.
- Diagnosis can be easily established through ultrasonography.
- The condition frequently follows a favorable natural course
- Acute Acute management involves analgesics and steroid injections, with ultrasound-guided percutaneous lavage being the primary option for treatment-resistant cases,

Statements

Ethics Approval

Written informed consent for publication was obtained from the patient.

Conflict of Interest

The authors declare no potential conflict of interest.

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