Calcific Tendinosis Mimicking Septic Arthritis and Myositis: Case Report and Literature Review

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ABSTRACT

Hydroxyapatite deposition disease (HADD) is a crystal-induced condition that most commonly affects the shoulder, leading to rotator cuff tendinopathy. It rarely involves the hip joint and causes gluteus medius tendinitis, making diagnosis in such cases particularly challenging.

We report the case of a 54-year-old female patient who presented with acute, severe right hip pain and restricted mobility. Imaging and laboratory findings suggested right coxofemoral arthritis and focal myositis. Further evaluation revealed multifocal calcific tendinopathies, including a ruptured calcific tendinopathy of the gluteus medius tendon. Ultrasound-guided arthrocentesis ruled out septic arthritis.

The patient responded dramatically to colchicine, with rapid improvement in both pain and mobility. This case highlights the importance of considering HADD in the differential diagnosis of acute monoarthritis, particularly in atypical sites, and underscores the diagnostic value of bimodal imaging.

CASE REPORT

BACKGOURND

This article describes a rare case of gluteus medius calcific tendinitis presenting with features suggestive of both septic arthritis and focal myositis. It highlights the critical role of multimodal imaging in establishing the correct diagnosis. Additionally, it underscores the rapid and effective response to colchicine, supporting its therapeutic value in acute crystal-induced inflammation.

CASE PRESENTATION

A 54-year-old woman presented to the emergency department with a two-day history of progressively worsening

right hip pain, which had become severe enough to prevent weight-bearing on the affected side. Her medical history included recently diagnosed arterial hypertension. Her surgical history included a previous cholecystectomy for recurrent biliary colic. She had no known drug allergies and reported a 30 pack-year smoking history.

On presentation, the patient was afebrile, and her vital signs were within normal limits. Physical examination revealed a markedly limited range of motion in the right hip, with tenderness on palpation and inability to bear weight. Straight leg raise testing was negative bilaterally, and the remainder of the physical examination was unremarkable. The pain was constant and not relieved by simple analgesics (paracetamol and ibuprofen).

Investigations

Pelvic radiography showed the presence of calcifications at the greater trochanters and ischia bilaterally (Figure 1). Pelvic magnetic resonance imaging (MRI) revealed a moderate right coxofemoral joint effusion with intense edematous infiltration of the periarticular soft tissues, (involving gluteus medius, gluteus minimus, iliopsoas and obturator internus muscles), moderate tendinopathy of the gluteus medius tendon and mild peritrochanteric edema (Figure 2A,2B). Laboratory investigations showed evidence of inflammation, with a C-reactive protein (CRP) level of 23.6 mg/L (reference <5 mg/L), a mildly elevated white blood cell (WBC) count of 11.400/mm³ (reference 4.800–10.800/mm³), an elevated lactate dehydrogenase (LDH) level of 267 IU/L (reference 91–248 IU/L), and a creatine phosphokinase (CPK) level of 245 U/L (reference 26–174 U/L).

To exclude septic arthritis, ultrasound-guided arthrocentesis of the right hip was performed, but no synovial fluid was obtained. After saline injection, 1.5 mL of the lavage fluid was sent for culture. Gram stain and culture were negative. Shoulder radiographs showed large calcifications of the supraspinatus tendons (Figure 3).

Treatment and Outcome

Based on these findings, a presumptive diagnosis of ruptured calcific tendinosis of the gluteus medius tendon was made, in the context of a multifocal calcific tendinosis syndrome. The patient was started on colchicine 1mg daily. Within 24 hours, pain significantly improved, and by 48 hours the patient was ambulatory. She was discharged after 72 hours on a short course of colchicine. At day 7, synovial fluid culture remained sterile. Follow-up MRI of the pelvis and ultrasound of the hips showed marked reduction in edema and periarticular inflammation, confirming gluteus medius tendinopathy resolution.

DISCUSSION

Hydroxyapatite deposition disease (HADD) is a condition characterized by the deposition of hydroxyapatite crystals in and around joints, particularly in the periarticular soft tissues such as tendons and bursae. This crystal-induced disease encompasses a wide clinical spectrum, ranging from asymptomatic calcific deposits to acute inflammatory tendinopathies and periarthritis [1]. The disease most commonly affects the shoulder, particularly the rotator cuff tendons, where it is a well-recognized cause of shoulder pain and dysfunction. Less frequently, extra-shoulder localizations, such as the hip, elbow, and wrist, may occur, often presenting a diagnostic challenge due to their rarity and nonspecific clinical manifestations [2,3]. The hip joint represents the second most frequent site of involvement after the shoulder, with the rectus femoris and gluteal tendons constituting the primary targets for calcific deposition [2]. A comprehensive literature review reported only 42 cases of gluteus medius and 36 cases of gluteus maximus calcific tendinopathy, underscoring the rare nature of these localizations [2].

Clinically, symptomatic HADD may present acutely, simulating infectious or inflammatory arthropathies, with severe pain, functional impairment, and elevated inflammatory markers [3]. The inflammatory reaction is typically secondary to rupture of a calcific deposit into the surrounding soft tissues, triggering an intense local response mediated by macrophages and neutrophils. This pathophysiological overlap with septic arthritis or myositis can lead to diagnostic uncertainty, unnecessary invasive procedures, and inappropriate antimicrobial therapy.

Here, we describe a rare case of gluteus medius calcific tendinitis in a 54-year-old woman who presented with acute hip pain mimicking septic arthritis and focal myositis. The case emphasizes the importance of considering HADD in the differential diagnosis of acute monoarthritis, the critical role of multimodal imaging in diagnostic clarification, and the potential therapeutic benefit of colchicine in crystal-induced inflammation.

Our case uniquely documents gluteus medius calcific tendinitis presenting with features mimicking both septic arthritis and focal myositis, a combination not previously reported in the literature. It emphasizes the diagnostic challenge of atypical HADD localizations and the critical role of multimodal imaging for accurate identification of calcific deposits and exclusion of infectious or inflammatory mimics. Additionally, the rapid and robust response to colchicine highlights its potential as a targeted therapy in the acute inflammatory phase. By integrating these clinical, radiological, and therapeutic insights, this report expands the understanding of atypical HADD presentations and provides a practical reference for managing complex cases of acute hip pain.

Epidemiology and Pathophysiology

Calcific tendinitis, calcific tendinopathy, and hydroxyapatite deposition disease (HADD) are synonymous terms describing a crystal-induced disorder characterized by deposition of hydroxyapatite crystals within periarticular soft tissues, most commonly tendons and bursae [4,5]. The condition typically

affects individuals between the fourth and seventh decades of life, with a clear female predominance, and is usually unilateral and monoarticular [5,6]. Although the shoulder joint, particularly the rotator cuff tendons, is the most frequently involved site, extra-shoulder localizations, including the hip, elbow, wrist, and knee, have been reported [4,5]. Within the hip region, the rectus femoris and gluteal tendons represent uncommon but clinically relevant sites of involvement. Calcific tendinitis of the gluteus medius remains rare, with only sporadic cases described in the literature [5-8].

The precise pathogenesis of HADD remains incompletely elucidated but is widely considered multifactorial. Proposed contributing mechanisms include repetitive microtrauma, local ischemia, endocrine and metabolic abnormalities (notably diabetes mellitus and thyroid disorders), and genetic predisposition [5,9]. It is now recognized as an active, cell-mediated process rather than a passive degenerative phenomenon. Histopathologic studies demonstrate that tenocytes undergo fibrocartilaginous metaplasia, transforming into chondrocyte-like cells capable of initiating matrix vesicle formation, which subsequently serve as nucleation centers for hydroxyapatite deposition [4,9].

Dalla-Torre et al. [10] delineated four distinct stages in the natural history of HADD: (1) a *precalcific* stage characterized by fibrocartilaginous metaplasia, (2) a *formative* stage marked by crystal deposition, (3) a *resorptive* stage corresponding to acute inflammation, and (4) a *repair* stage during which fibroblastic proliferation and tissue remodeling occur. The resorptive phase is clinically relevant, as it represents the period of maximal inflammation and symptom severity, often leading to diagnostic confusion with infectious or inflammatory arthropathies [9-11].

Calcific deposits involving the gluteus medius tendon are typically located lateral or superior to the greater trochanter [11]. Acute presentations are characterized by sudden-onset lateral hip pain, tenderness, and functional limitation. Although most acute episodes are self-limited, resolving within one to two weeks, chronic cases can persist for several months, with residual calcifications visible radiographically for up to eight months [7].

Imaging Features

Accurate diagnosis of HADD relies on a multimodal imaging approach. Plain radiography remains the first-line modality, capable of identifying calcific deposits as homogeneous or amorphous radiopaque densities adjacent to tendinous insertions [12]. The morphology and density of these deposits correlate with disease stage, dense and sharply circumscribed during the formative phase, but ill-defined and cloud-like during resorption.

Radiographs are particularly valuable for documenting resolution following conservative therapy. Ultrasonography offers superior sensitivity for detecting small or early deposits and allows dynamic assessment of tendon integrity and vascularity. Typical sonographic findings include hyperechoic foci with or without posterior acoustic shadowing; Doppler imaging may demonstrate perilesional hyperemia during the acute inflammatory phase [11]. Ultrasound also serves as a guidance tool for aspiration, lavage, or corticosteroid injection when indicated.

Magnetic resonance imaging (MRI) provides detailed evaluation of the soft-tissue and bone marrow response to inflammation. On MRI, calcific deposits appear as focal signal voids surrounded by hyperintense edema on T2-weighted images and hypointense areas on T1-weighted sequences [11]. MRI also assists in excluding differential diagnoses such as infection, neoplasm, or avascular necrosis. Sakai et al. [7] emphasized that bone marrow edema, rather than the calcific deposit itself, is the principal source of pain, reinforcing the inflammatory basis of acute HADD. The integration of radiography, MRI, and ultrasonography thus provides a comprehensive assessment, allowing accurate diagnosis and exclusion of mimicking conditions.

Differential Diagnosis

Septic Arthritis

The clinical presentation of acute calcific tendinitis may mimic septic arthritis due to overlapping features: acute pain, swelling, restricted motion, and elevated inflammatory markers. However, septic arthritis is a rare but serious cause of hip monoarthritis, with high potential for rapid joint destruction if missed. In our case, prompt ultrasound-guided arthrocentesis ruled out infection, thus avoiding unnecessary antibiotic therapy and invasive procedures.

Focal Myositis

Focal myositis, another differential diagnosis, is a benign pseudotumoral inflammatory disorder of skeletal muscle that can mimic infectious or neoplastic disease [12]. It typically presents with pain and limited range of motion without systemic symptoms, and muscle enzyme levels (LDH, CPK) are often normal. Yoo et al. reported three cases, two resolving with NSAIDs and one requiring colchicine with rapid improvement [12]. Although rare, calcific tendinitis can present with features resembling acute myositis due to the migration of hydroxyapatite crystals into adjacent muscles, provoking localized inflammation. Published cases describe patients with subscapular [13] or gluteus medius involvement [14], in whom crystal-induced inflammation led to muscle edema, pain, and restricted mobility, closely mimicking myositis on clinical and imaging assessments. In our patient, mild elevations of muscle enzymes, periarticular edema on MRI, and localized tenderness initially suggested focal myositis. Such overlap reflects the inflammatory resorptive phase of HADD, during which peritendinous edema and myogenic irritation can mimic myositis both clinically and radiologically. The identification of gluteus medius calcifications, absence of systemic features,

and prompt response to colchicine, however, substantiated the diagnosis of calcific tendinitis. This case underscores the need to recognize HADD as a myositis-mimicking entity in acute periarticular inflammation, emphasizing careful correlation of imaging, laboratory findings, and response to targeted therapy.

Crystal Arthropathies

Gouty arthritis may also mimic calcific tendinitis but is distinguished by monosodium urate crystals, often polyarticular involvement, and characteristic erosive changes on imaging, which occur in about 44% of patients, especially those with chronic or tophaceous gout. Synovial fluid analysis remains the key diagnostic tool for differentiation [15].

Treatment and Prognosis

Most cases of calcific tendinitis are self-limiting, resolving spontaneously or with conservative management such as rest, NSAIDs, and physical therapy [5,7,11]. Corticosteroid injections or ultrasound-guided aspiration may be considered for persistent pain. Shock wave therapy is a safe and effective noninvasive treatment option that facilitates fragmentation and resorption of calcium deposits, promotes healing, and improves function, with success rates reported up to 90% in some studies [16]. Surgical treatment, typically arthroscopic removal of calcific deposits, is considered when conservative management and shock wave therapy fail, especially in chronic cases with persistent pain or loss of function. Surgery can involve either open or arthroscopic techniques, with arthroscopy favored for shorter rehab and less invasiveness. Although recovery may take several months, surgery generally results in satisfactory clinical outcomes [17,18]. In our patient, colchicine (1 mg daily) led to a dramatic reduction in pain within 24 hours and full functional recovery within 48 hours. This response supports colchicine's anti-inflammatory mechanism, which involves inhibition of neutrophil migration, suppression of the NLRP3 inflammasome, and reduction of crystal-induced inflammation, pathophysiological features also implicated in HADD.

CONCLUSION

Hydroxyapatite deposition disease represents an under-recognized cause of acute periarticular inflammation, capable of closely mimicking septic arthritis or inflammatory myopathies when occurring at atypical sites such as the gluteus medius tendon. This case reinforces several key clinical principles. First, clinicians should maintain a high index of suspicion for HADD in patients presenting with acute monoarthritis and periarticular calcifications, even in the absence of shoulder involvement. Second, a multimodal imaging strategy, combining radiography, ultrasonography, and MRI, remains indispensable for accurate localization of calcific deposits, assessment of soft-tissue inflammation, and exclusion of infectious or neoplastic mimics. Third, therapeutic responsiveness to colchicine underscores its value as a targeted anti-inflammatory option, exploiting its inhibitory effects on neutrophil chemotaxis and inflammasome activation, thereby mitigating crystal-induced inflammation.

In summary, early recognition of HADD is essential to prevent misdiagnosis and overtreatment. The present case contributes to the limited body of literature on gluteus medius calcific tendinitis, highlighting the diagnostic utility of imaging, the clinical efficacy of colchicine, and the necessity for awareness of this rare but important differential diagnosis in acute hip pain.

TEACHING POINTS

HADD can mimic septic arthritis and myositis, especially in atypical localizations such as the gluteus medius tendon, multimodal imaging (X-ray, ultrasound, MRI) essential for diagnosis. Finally, Colchicine may provide rapid symptom relief by inhibiting crystal-induced inflammation.

QUESTIONS

Question 1: What is the composition of calcifications in calcific tendinitis?

A. Calcium pyrophosphate dihydrate
B. Monosodium urate crystals
C. Hydroxyapatite crystals
D. Basic calcium oxalate

Correct answer: C. Hydroxyapatite crystals

Explanation: Calcific tendinitis (HADD) is a "crystal-induced disorder characterized by deposition of hydroxyapatite crystals within periarticular soft tissues."

Question 2. Which elements point to a non-mechanical joint effusion in the case described?

A. Pain only during passive movement
B. Absence of periarticular edema on MRI
C. Elevated CRP/WBC, periarticular soft-tissue edema, and moderate joint effusion on MRI
D. Normal inflammatory markers with no muscle involvement

Correct answer: C.

Explanation: Mechanical joint effusions do not cause any systemic effects nor does it affect surrounding tissues.

Question 3. What are the MRI-based differential diagnoses when muscle edema suggests myositis?

A. Bursitis, osteoarthritis, and simple strain
B. Tendon rupture, ligament sprain, and ganglion cyst
C. Septic arthritis/myositis, neoplasm, autoimmune myositis, and crystal-induced inflammation from calcific tendinitis
D. Fibromyalgia and chronic tendinopathy without inflammation

Correct answer: C.

Explanation: MRI findings of muscle edema may mimic septic arthritis, pyomyositis, neoplasm, and autoimmune myositis, but can also arise from crystal migration and inflammation in HADD, making these the key differentials.

Question 4. What treatment options for calcific tendinitis are described in the article?

A. Long-term antibiotics and open surgical exploration

B. Radiation therapy and immunosuppressants

C. NSAIDs, colchicine, corticosteroid injections, ultrasound-guided aspiration/lavage, shockwave therapy, and surgery for refractory cases

D. Only rest and physical therapy

Correct answer: C.

Explanation: Calcific tendinitis is usually managed with conservative therapy (NSAIDs), colchicine (rapid improvement in the presented case), corticosteroid injections, ultrasound-guided aspiration, shockwave therapy, and arthroscopic removal if symptoms persist.

Question 5: What is the best imaging modality for identifying calcific tendinitis?

A. MRI

B. PET scan

C. Plain radiography (X-ray)

D. Bone scintigraphy

Correct answer: C. Plain radiography (X-ray) **Explanation:** Xray's are the first-line imaging modality and effectively shows calcific deposits as homogeneous or amorphous radiopaque densities, making it the best tool for detecting calcifications themselves. MRI and ultrasound provide complementary soft-tissue information.

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FIGURES

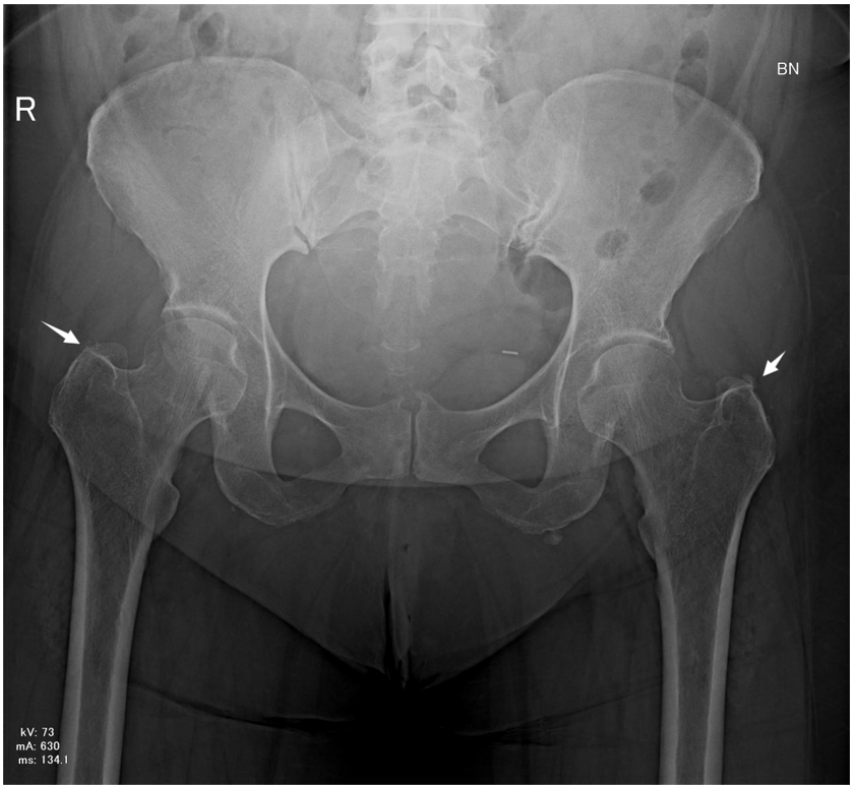
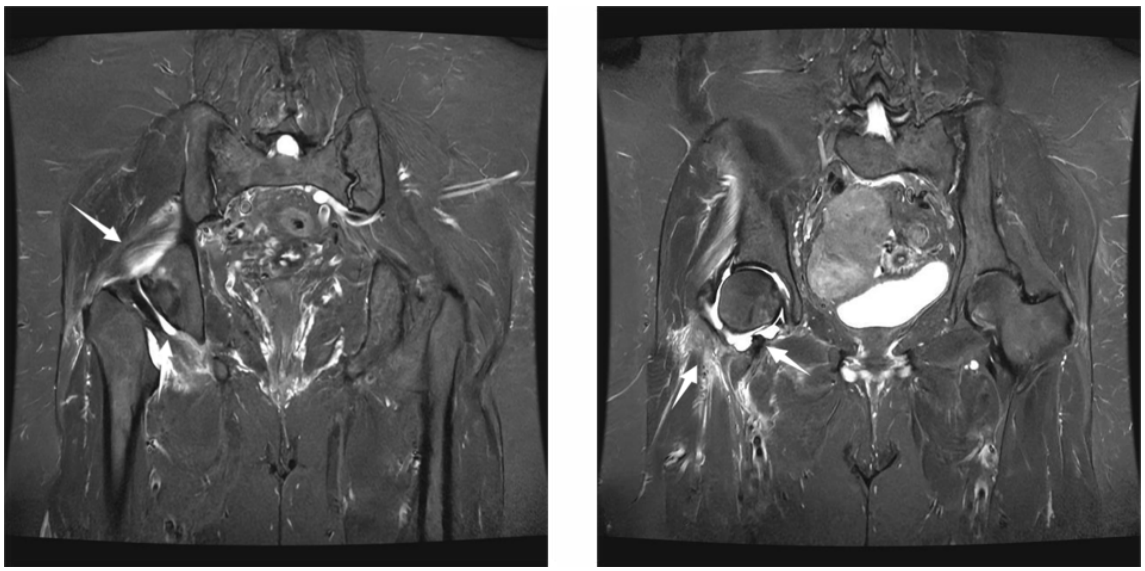


Figure 1: Pelvic radiograph showing calcific deposits at the gluteus medius insertion (arrows).



Figures 2A,2B: MRI of the pelvis showing effusion in the right coxofemoral joint and right gluteus medius tendinopathy with surrounding edema involving gluteus medius, gluteus minimus, iliopsoas and obturator internus muscles (arrows).

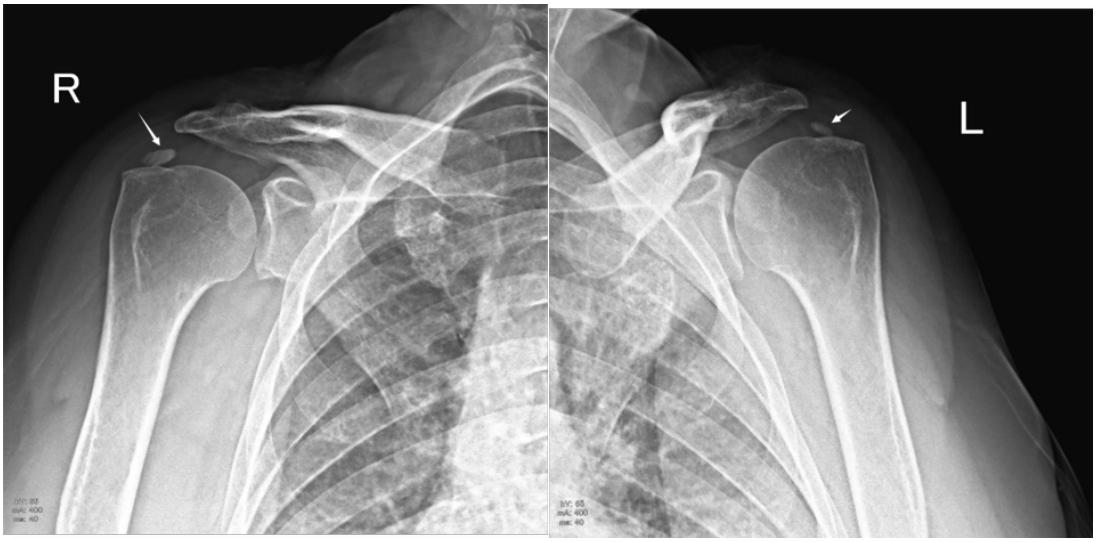


Figure 3: Shoulders radiographs showing supraspinatus tendon calcifications on both sides (arrows).

KEYWORDS

Calcific tendinosis, Hydroxyapatite deposit, Septic arthritis, Myositis, Case report.

ABBREVIATIONS

HADD = Hydroxy Apatite Deposition Disease
MRI = Magnetic Resonance Imaging
CRP = C-Reactive Protein
WBC = White Blood Cell
LDH = Lactate Dehydrogenase
CPK = Creatine Phosphor Kinase
NSAIDs = Non Steroidal Anti-Inflammatory Drugs