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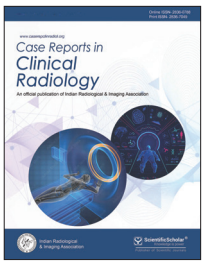

Case Series

Hydroxyapatite deposition disease beyond the shoulder: A case series on unusual musculoskeletal locations

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ABSTRACT

Hydroxyapatite deposition disease (HADD) is a condition where calcium hydroxyapatite crystals build up in soft tissues such as tendons and joints, most frequently observed in the shoulder. This specific case series highlights HADD occurrences in atypical locations. This is particularly important because HADD in these unusual areas can easily be mistaken for an infection or a tumor. By improving our awareness that HADD can present in these uncommon sites, clinicians can make a confident diagnosis, leading to better patient care.

Keywords: Calcific periarthritis, Calcific tendinosis, Crystal arthropathy, Hydroxyapatite deposition disease, Hydroxyapatite rheumatism

INTRODUCTION

Hydroxyapatite deposition disease (HADD) is a crystal-induced arthropathy characterized by calcium hydroxyapatite deposition in periarticular soft tissues, tendons, or bursae. It affects approximately 3% of adults aged 30–60 years, with a higher prevalence in women. Although the shoulder remains the most common site, extra-shoulder manifestations – including the hip, elbow, wrist, knee, ankle, and hand – are increasingly reported.[1,2]

The disease progresses through three phases: Pre-calcific, calcific, and post-calcific. While often self-limiting, acute inflammation during the resorptive phase can mimic infection, gout, or soft-tissue neoplasm.[3] Recognizing this imaging pattern is essential to prevent invasive procedures and ensure timely, conservative management.

CASE SERIES

Case 1

A 44-year-old woman presented with acute right thigh pain for 5 days without trauma or fever. Magnetic resonance imaging (MRI) hip and thigh [Figure 1] revealed small amorphous calcifications along the posterior aspect of the proximal femoral diametaphysis, at the attachment of the vastus lateralis muscle onto the linea aspera. Adjacent soft-tissue and fascial edema were evident, with no periosteal reaction or bone marrow involvement. Findings were consistent with acute calcific tendinitis (HADD) of the linea aspera. The patient was managed conservatively with non-steroidal anti-inflammatory drugs (NSAIDs) and rest, showing symptomatic improvement.

Case 2

A 28-year-old woman presented with acute neck pain and right upper limb paresthesia for 5 days. MRI cervical spine [Figure 2] demonstrated a small amorphous calcific focus (7 × 6 mm) involving the central fibers of the right longus colli muscle at the C5–C6 level, with associated soft-tissue edema and thin prevertebral fluid extending from C1 to C5. Computed tomography (CT) correlation confirmed calcification without evidence of infection or abscess. These findings supported acute calcific tendinitis of the longus colli muscle (HADD). The patient responded well to NSAIDs and physiotherapy within 10 days.

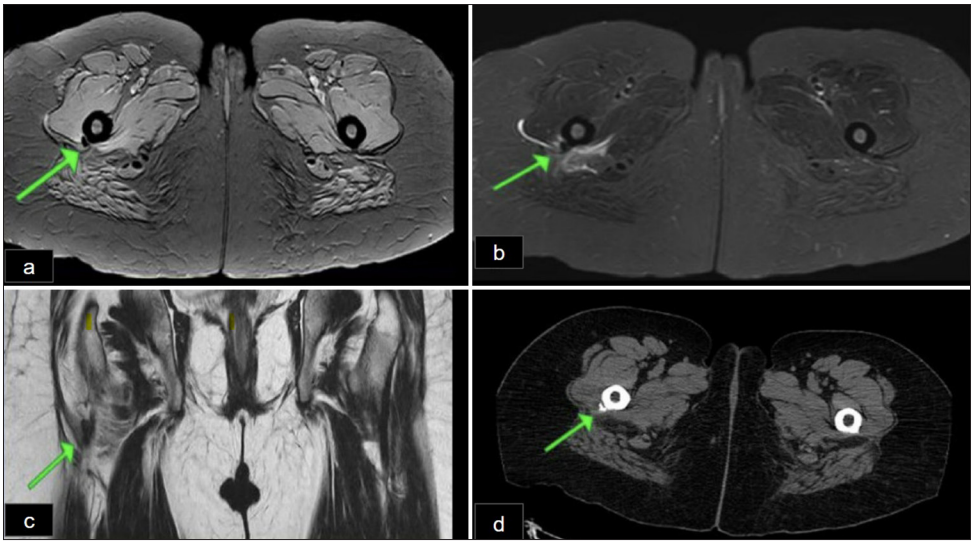

Figure 1: (a) Axial GRE image of the right thigh showing tiny hypointense foci (calcific deposits) along the posterior aspect of the proximal femur at the attachment of the vastus lateralis (green arrow). (b and c) Axial STIR and Coronal T2 weighted images showing localized peritendinous inflammation along linea aspera with surrounding soft tissue and fascial edema (green arrow). (d) Axial computed tomography confirming amorphous calcific foci at the muscle insertion (green arrow).

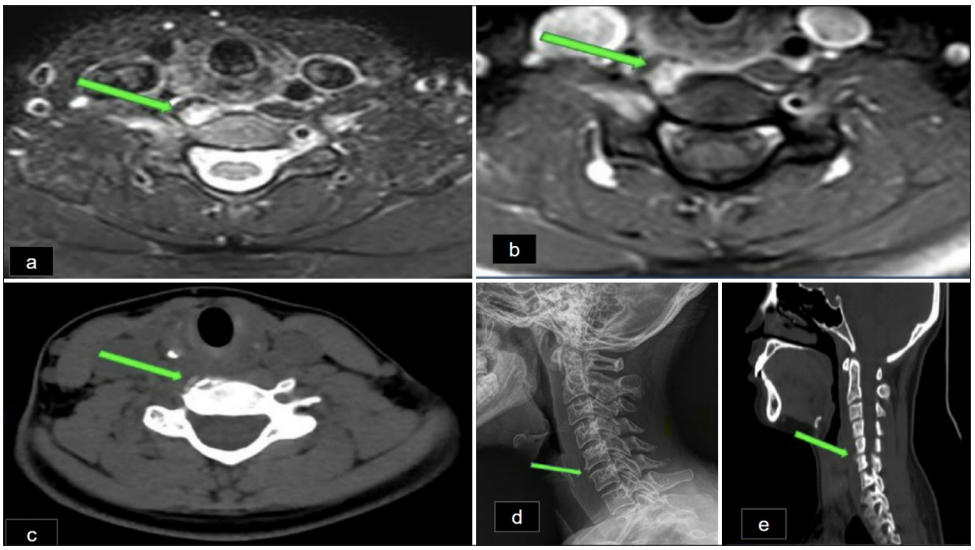

Figure 2: (a) Axial STIR image showing prevertebral soft-tissue edema at the C5–C6 level with focal hyperintensity in the right longus colli, (green arrow). (b) post-contrast T1-weighted image demonstrating thin linear prevertebral enhancement extending from C1 to C5, (green arrow). (c and d) axial computed tomography (CT) and sagittal radiograph images showing a small amorphous calcific focus within the right longus colli muscle at C5-6 level, (green arrow) and (e) sagittal CT image confirming central calcification with surrounding soft-tissue swelling (green arrow).

Case 3

An 81-year-old male presented with pain and swelling over the left 1st metatarsophalangeal (MTP) joint for one week. Serum uric acid was normal. MRI foot [Figure 3] revealed a small amorphous calcific focus in the periarticular region of the 1st MTP joint involving the medial capsuloligamentous structure, with mild marrow edema and subtle cortical erosion. Dual-energy CT (DECT) excluded uric acid deposition. Imaging features were consistent with acute calcific periarthritis (HADD). The patient improved with conservative management and short-term NSAIDs.

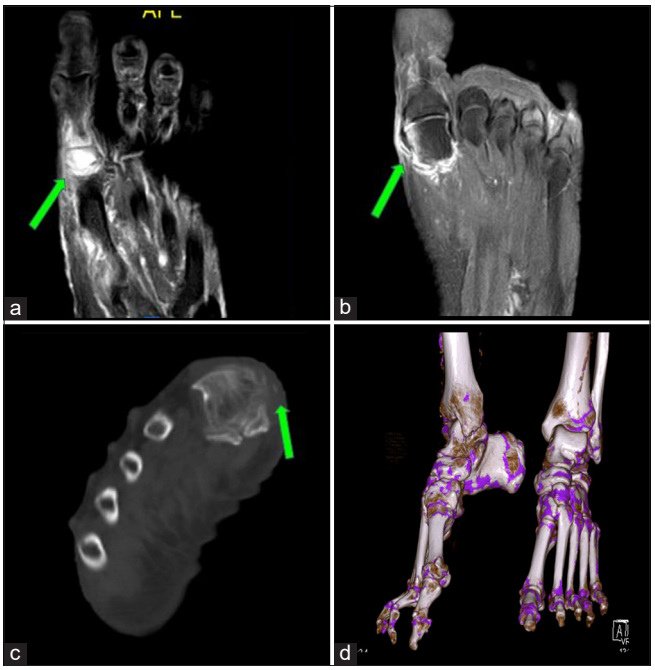

Figure 3: (a) Axial short tau inversion recovery (STIR) image showing hyperintensity around the first metatarsophalangeal joint with surrounding soft-tissue edema, (green arrow). (b) Axial post-contrast T1-weighted image showing enhancement of periarticular soft tissues along the medial capsule (green arrow). (c) Axial computed tomography (CT) showing amorphous calcific focus in the periarticular region of the first MTP joint (green arrow). (d) Dual-energy CT showing absence of uric acid deposition (urate-negative signal) (green arrow).

DISCUSSION

Background

HADD encompasses a group of conditions involving hydroxyapatite crystal deposition in soft tissues, tendons, and periarticular structures. Commonly affecting the rotator cuff, HADD may occur at atypical sites such as the hip, neck, or extremities, leading to diagnostic confusion. The pathogenesis likely involves microtrauma, ischemia, and tendon necrosis, leading to calcium crystal deposition. The disease progresses through pre-calcific, calcific, and post-calcific stages, with symptoms typically most severe during the resorptive phase.[1,2]

Clinical and imaging perspective

When occurring in atypical locations, HADD can mimic infection, trauma, or neoplasm, especially in the acute inflammatory phase. Plain radiography and CT remain first-line modalities, showing amorphous or cloud-like calcifications near tendons or capsule insertions. MRI may reveal low-signal foci (calcific deposits) with surrounding soft-tissue edema and enhancement. The "arc and ring" sign or gradient echo (GRE) blooming supports the diagnosis. DECT is valuable in differentiating HADD from gout by demonstrating the absence of urate deposition. The correlation of imaging findings with absence of systemic inflammatory markers and clinical improvement under conservative therapy confirms the diagnosis.[3-5]

Ultrasonography is also a valuable tool for evaluating HADD. In most cases, calcifications appear as hyperechoic foci with or without acoustic shadowing. During the symptomatic resorptive phase, Color Doppler imaging often reveals increased vascularity. Unlike degenerative calcifications in non-viable tendons, HADD-related calcifications are typically vascular.[3-5]

Outcome and management

Acute HADD is self-limiting, resolving within 2–3 weeks. Conservative measures – NSAIDs, rest, and physiotherapy – are first-line. Ultrasound-guided barbotage or aspiration may be considered for persistent cases, while surgery is reserved for refractory ones.[6]

DIFFERENTIAL DIAGNOSIS

- Gout: Monosodium urate deposits with "rat-bite" erosions and positive DECT signal.
- Heterotopic ossification (HO): Corticated bone formation within soft tissue
- Traumatic calcification: Associated with fracture lines or bone contusions
- Infection: Rim-enhancing abscesses and systemic symptoms.[2]

CONCLUSION

HADD is a dynamic, self-limiting condition that can present in unusual musculoskeletal sites. Recognition of its imaging spectrum and clinical context is key to distinguishing it from infection or neoplasm. Awareness of extra-shoulder manifestations helps avoid misdiagnosis and unnecessary interventions.

TEACHING POINTS

1. HADD can occur beyond the shoulder, mimicking infection or tumor.
2. CT and MRI play complementary roles – CT detects calcification, MRI delineates inflammation.
3. DECT helps exclude gout by identifying urate-negative deposits.
4. Awareness of site-specific patterns ensures correct diagnosis and avoids overtreatment.

MCQs

1. In differentiating HADD from HO on CT, which finding favors HADD?
   a) Presence of peripheral cortical bone formation
   b) Amorphous, cloud-like calcification without cortication
   c) Mature trabecular bone within soft tissue
   d) Central lucent zone with surrounding ossification

Answer Key: b

2. On ultrasonography, which feature differentiates HADD from degenerative (non-HADD) calcification?
   a) Presence of hyperechoic foci
   b) Absence of posterior acoustic shadowing
   c) Associated vascularity on color Doppler
   d) Location near tendon insertion

Answer Key: c

3. HADD at atypical sites, such as the hip or neck, may commonly be misdiagnosed as:
   a) Fibromyalgia
   b) Infective or neoplastic lesion
   c) Stress fracture
   d) Rheumatoid arthritis

Answer Key: b

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