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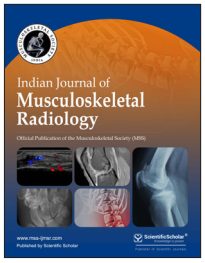

Case Series

Hydroxyapatite deposition disease – Think beyond the shoulder!! A case series with unusual sites of involvement

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ABSTRACT

Hydroxyapatite deposition disease (HADD) can affect any location of the body and present with a variety of clinical symptoms. Here, we describe three unusual sites of involvement of HADD around the hip as well as the wrist joint. Magnetic resonance imaging along with computed tomography correlation was performed in these cases, which demonstrated the calcific deposits as well as their associated soft-tissue inflammatory changes. Its imaging findings can look very aggressive and empowering, similar to infection or even malignancy. This makes this entity a great mimicker. Through these cases, we emphasize the need to think about HADD in places other than the classical shoulder girdle and always keep it on the differential list.

Keywords: Hydroxyapatite, Deposition, Crystal

INTRODUCTION

Deposition of hydroxyapatite crystals, which can occur intra- or periarticular, is the hallmark of the condition known as hydroxyapatite deposition disease (HADD). The most typical presentation is calcific tendinitis, which is caused by hydroxyapatite crystal deposition within tendons. Peak incidence occurs between the ages of 30 and 60, and it affects up to 3% of adults.[1] HADD's cause is unknown; however, it is thought to be a cell-mediated reactive process that is different from degenerative tendinopathy.[2-4]

The hip is the second most often affected region after the shoulder, affecting 5% of patients with HADD.[5] The greater trochanter, where the gluteal tendons insert, is the most frequently involved site.[6] HADD, however, can develop in any tendon or muscle.

The flexor and extensor tendons of the hand can also be involved; the most frequent location is where the flexor carpi ulnaris (FCU) tendon attaches to the pisiform bone.[7,8]

Calcium hydroxyapatite deposition disease may occasionally involve the superolateral portion of the longus colli muscle. Clinical symptoms include headache, neck pain, and dysphagia.[9]

Patients may have no symptoms or exhibit discomfort, swelling, pain, or a limited range of motion. The symptoms are occasionally accompanied by a low-grade temperature and elevated inflammatory markers, and they can be very severe. Imaging may also reveal edematous soft tissues and accompanying bone abnormalities. Its accurate diagnosis is therefore crucial.

CASE SERIES

Case 1

A 34-year male presented with right-sided hip pain for 3 days. The pain was sudden in onset. There was no history of any significant trauma. On clinical examination, there was a painful external rotation of the right hip. The patient was referred for magnetic resonance imaging (MRI) right hip joint. The MRI showed the presence of a few ill-defined nodular foci appearing hypointense on T1- and T2-weighted images in the region of the musculotendinous junction of the right inferior gemillus [Figure 1a and b]. These nodular foci showed calcific density on limited computed tomography (CT) correlation [Figure 2a and b]. There was associated significant edema and inflammatory changes in the surrounding external rotator group of muscles including the gemelli, obturator internus, and pyriformis [Figure 3a and 3b]. These deposits were in close proximity to the right hip joint capsule. A diagnosis of calcium hydroxyapatite deposition disease was made on CT and MRI findings.

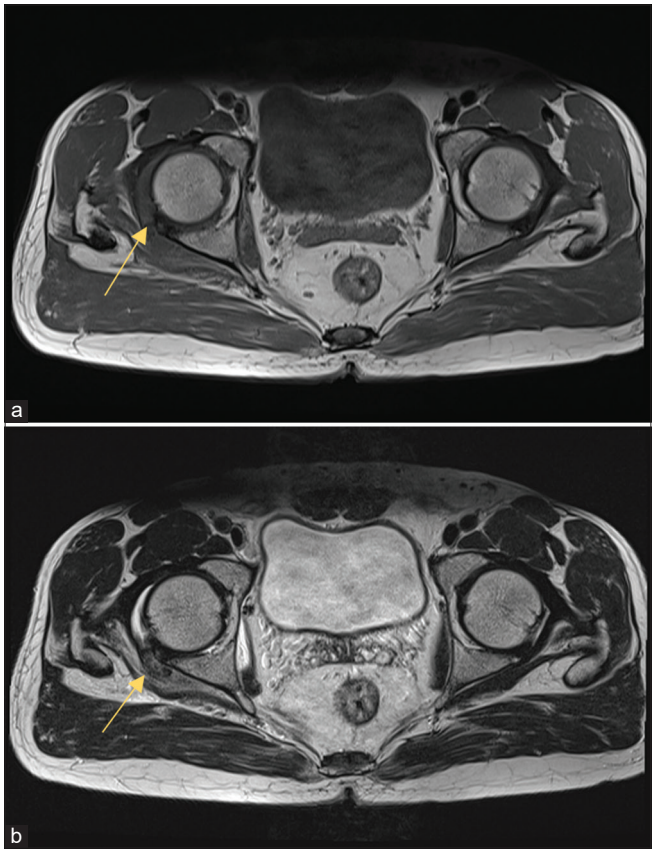

Figure 1: Axial T1W (a) and T2W (b) images of the hip joint demonstrate ill-defined hypointense nodular foci (arrows) in the region of the musculotendinous junction of the right inferior gemillus muscle.

Case 2

A 61-year-old male presented with left-sided hip pain for 7–10 days. The pain was maximum in intensity during waking up. No history of fever or trauma was noted. On clinical examination, there was painful and restricted abduction of the left hip. The patient was referred for evaluation of left hip pain. The MRI showed the presence of multiple nodular to confluent foci appearing hypointense on T1- and T2-weighted images within the gluteus medius and minimus tendon, close to their insertion over the greater trochanter [Figure 4a and b]. These foci showed calcific density on limited CT correlation [Figure 5a and b]. There was associated significant edema in the surrounding soft tissues. Fluid was also noted in the greater trochanteric bursa with changes in trochanteric bursitis [Figure 6a and b]. These features were consistent with calcium hydroxyapatite deposition disease involving the left abductor tendons in the region of the greater trochanter, which is most common site of HADD around the hip joint.

Case 3

A 42-year-old male with no significant medical history presented with acute severe pain in the right wrist that had increased gradually over the course of 1 week. There was no history of trauma and the patient was afebrile. Clinically, he had tenderness and swelling over the volar aspect on the ulnar side of the wrist. The hand and wrist motions were restricted. The pain was aggravated by ulnar deviation or flexion against resistance. MRI was ordered to find out the cause of ulnar sided wrist pain. MRI showed bulky and

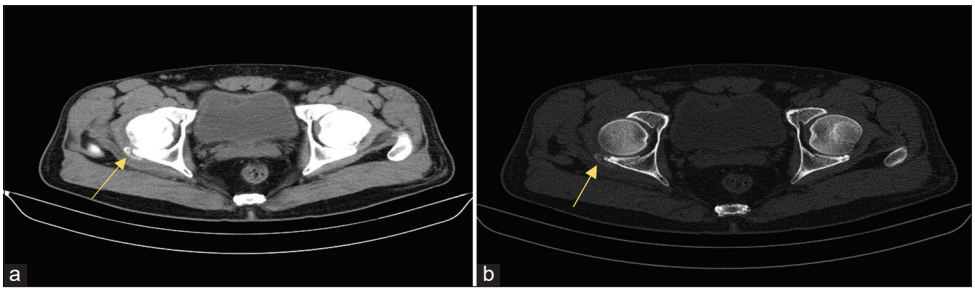

Figure 2: Axial CT scan soft-tissue window (a) and bone window (b) show nodular foci of calcium density (arrows), suggesting calcific deposits.

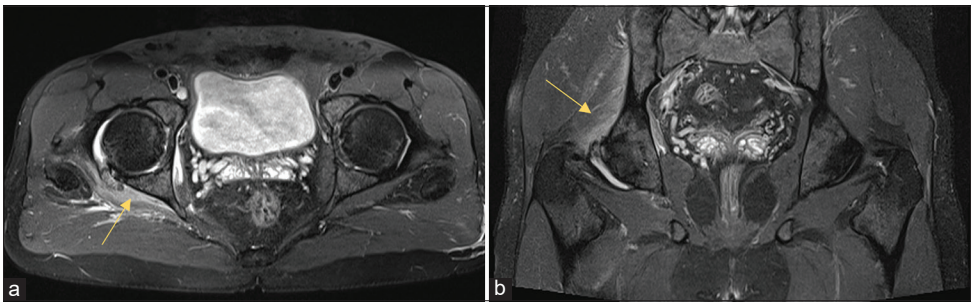

Figure 3: Proton density fat-saturated axial (a) and coronal (b) magnetic resonance imaging images show increased signal intensity in the surrounding soft tissues (arrows), suggesting acute inflammatory changes.

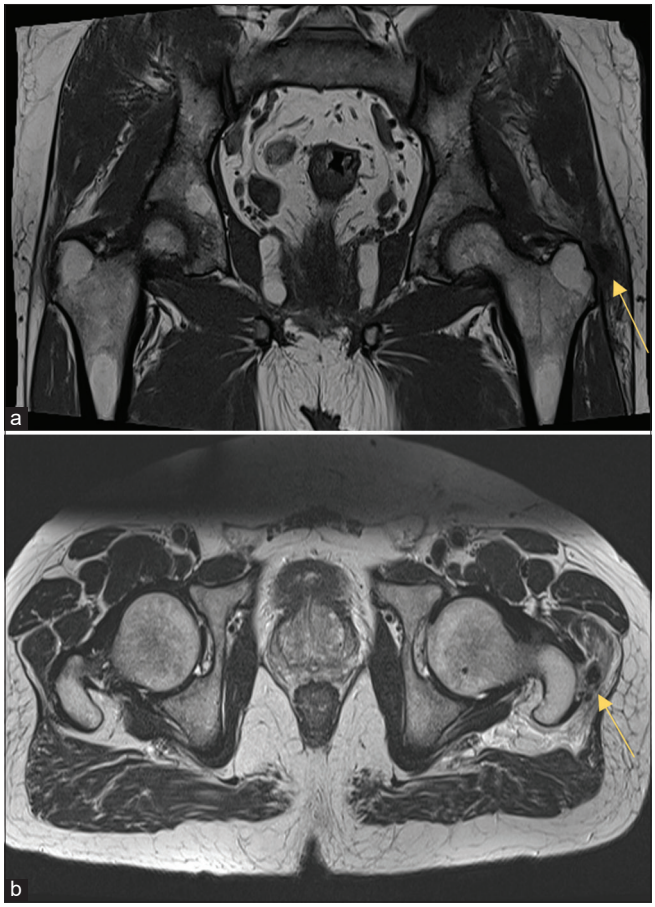

Figure 4: Coronal T1W (a) and axial T2W (b) image of the hip joints show few confluent hypointense foci (arrows) in the region of tendons of the left gluteus medius and minimus, close to their attachment over greater trochanter.

swollen FCU tendon close to its attachment over the pisiform carpal bone with increased signal intensity on fluid sensitive sequences [Figure 7a and b]. There were surrounding edematous and inflammatory changes. There was evidence of few foci appearing hypointense on T2-weighted and Proton density fat-saturated images [Figure 8a-c] as well as Gradient echo sequences images [Figure 8d] in the vicinity –

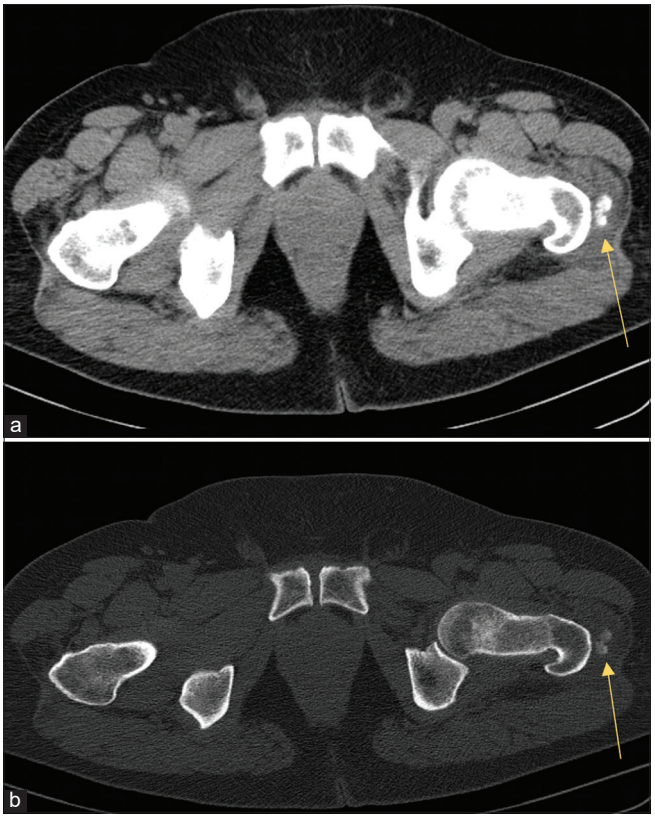

Figure 5: Axial computed tomography scan soft-tissue window (a) and bone window (b) show the nodular foci of calcium density (arrows), suggesting calcific deposits.

suggesting calcific deposits. Based on these imaging findings, a diagnosis of acute calcific tendinitis of the FCU tendon was made. The patient was treated with rest, immobilization, and splinting. There was a good response with near-complete resolution of symptoms in 5–7 days.

DISCUSSION

Calcific tendinitis typically affects people between the ages of 30 and 60.[10] Men and women are equally affected.[11] Calcium

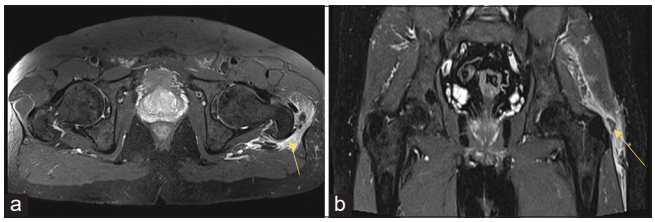

Figure 6: Axial (a) and coronal (b) proton density fat-saturated images of the hip show significantly increased signal intensity in the soft tissues surrounding the left greater trochanter due to the calcific deposits (arrows). Note is also made of mild fluid in the left trochanteric bursa.

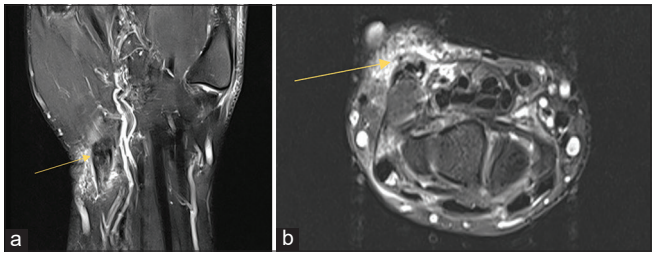

Figure 7: A coronal (a) and axial (b) proton density fat-saturated image of the wrist joint demonstrates bulky, swollen, and edematous flexor carpi ulnaris tendon (arrows) with significant surrounding inflammatory changes.

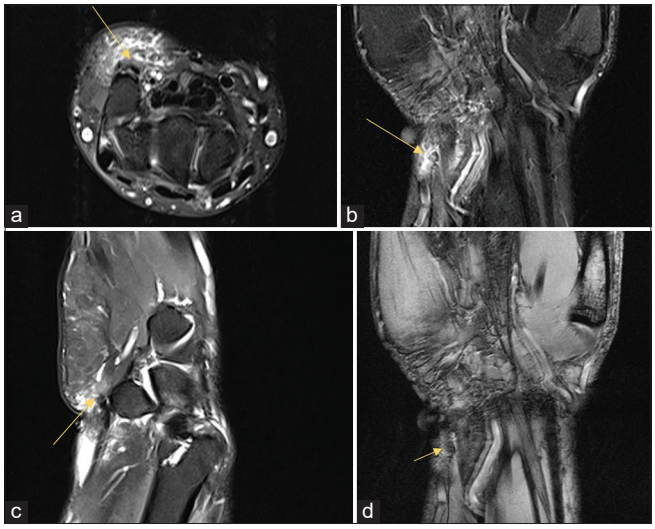

Figure 8: An axial (a), coronal (b), and sagittal (c) proton density fat-saturated images and coronal gradient echo sequences images (d) of the wrist joint show ill defined hypointense foci in the vicinity of the flexor carpi ulnaris tendon (arrows), consistent with calcific deposits.

apatite deposition disease is synonymously/interchangeably used with HADD, both of them representing the same disease process. Although calcific tendinitis frequently has no symptoms in the shoulder, these symptoms are virtually always present in the hand and the hip area. The precise sequence of events that lead to calcific deposition is still unclear and under debate. While some authors have suggested that the calcifications are the result of a cell-mediated reactive mechanism, others have suggested that the disorder is caused by tendon degeneration, hypoxia brought on by inadequate blood flow, and trauma.

According to Uhthoff *et al*., local hypoxia brought on by mechanical, metabolic, or other causes can cause tendon to fibrocartilage metaplasia, degeneration, and chondrocyte calcification.[3] Carroll *et al*.[12] proposed stress necrosis and chronic inflammation as the causes of such calcifications. These conditions are brought on by repetitive strain injuries and trauma. Activities such as quick, repetitive actions, and particular professions such as playing the violin, golfing, or typing are more frequently connected with this entity.[13]

CT is most sensitive in the detection of hydroxyapatite deposits as well as in analyzing the shape and consistency of deposits. This makes it possible to distinguish between them and fragments of ossification or avulsion, which have corticated borders and are more dense (100–400 HU for deposits in comparison to 700–1500 HU for bone).[14]

On all MRI sequences, calcific deposits show up as focal areas of low signal, usually close to tendon insertions. MRI is the best modality for assessing inflammatory changes and other causes of hip pain.[15] Edema that is connected with the acute resorptive phase may be severe and mimic an infection or injury. A malignant or infectious process may be mistaken for cortical erosion, periosteal response, and marrow involvement, which have all been described. The approach could be modified to include gradient echo pulse sequences to use the susceptibility artifact to identify calcification.

It is therefore essential for diagnosis to look for distinctive calcification near or within a tendon together with surrounding soft-tissue edema and inflammatory changes.

The prognosis for acute calcific tendinitis is excellent. According to Carroll *et al*.[12] and Strandell,[16] the time it takes for symptoms to resolve in untreated patients is considerable – roughly 3 weeks. The symptomatic time is reduced to 7–9 days with the use of a splint and NSAIDs.[12] Non-surgical therapies have a high success rate. Although they are also an option, invasive treatments such as local injections of anesthetic or steroid substances, aspiration of calcium deposits, surgical excision of calcific deposition, and lysis of peritendinous adhesions are typically reserved for severe cases that have not responded to conventional therapies and when symptoms last for more than a few weeks.

CONCLUSION

Although shoulder is the most common site of involvement, hydroxyapatite deposition disease (HADD)

can involve any joint or peritendinous soft tissues in the body. This entity should always be considered on the list of differential diagnosis of the pathologies around the joints. CT is the modality of choice to visualize the calcium deposits, whereas the MRI more clearly demonstrates its acute phase changes of soft-tissue edema and inflammation. The patient can be treated conservatively with rest, immobilization, and anti-inflammatory medications with a very good response/prognosis, so its diagnosis is essential and of great clinical importance to the orthopedic clinicians.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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