European Journal of Radiology 120 (2019) 108653

Contents lists available at ScienceDirect

European Journal of Radiology

journal homepage: www.elsevier.com/locate/ejrad

Calcium hydroxyapatite deposition disease: Imaging features and presentations mimicking other pathologies

Patcharee Hongsmatip[a], Karen Y. Cheng[b,*], Christopher Kim[c], David A. Lawrence[d], Robert Rivera[e], Edward Smitaman[b,1]

[a] Queen Savang Vadhana Memorial Hospital, 290 Jermjomphol Road, Sriracha, Chonburi, 20110 Thailand
[b] University of California, San Diego, 200 W. Arbor Drive, Mail code 8226, San Diego, CA, 92103, United States
[c] University of California, San Francisco, 505 Parnassus Avenue, M-391, San Francisco, CA, 94143, United States
[d] Department of Radiology and Medical Imaging, University of Virginia, 1218 Lee Street, Box 800170, Charlottesville, VA, 22908, United States
[e] St. Joseph's Hospital, 350 W. Thomas Road, Phoenix, AZ, 85018, United States

ARTICLE INFO

Keywords:
Calcium hydroxyapatite depositional disease
HADD
Trauma
Infection
Neoplasm

ABSTRACT

Objective: Calcium hydroxyapatite depositional disease (HADD) is usually asymptomatic and self-limiting; however, when there is an associated inflammatory process or HADD occurs in an unusual location, it may mimic trauma, infection, or neoplasm. The purpose of this article is to review the imaging features of HADD and how to distinguish it from more worrisome entities that can have similar appearances.

Conclusion: An understanding of the presentations of HADD is important to allow early and confident diagnosis. In particular, familiarity with presentations that resemble more ominous pathologies is essential to avoid costly and time-consuming workup or intervention.

1. Introduction

Calcium hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$, is the most common type of calcium in bone and pathologic calcifications [1]. Calcium hydroxyapatite depositional disease is also known as calcific tendinosis, calcific periarthritis, peritendinitis calcarea, calcific peritendinitis, calcific bursitis, and hydroxyapatite rheumatism. There is a female predominance [2], with one cohort study of HADD indicating that 71.6% of affected individuals were female [3]. The peak incidence occurs in the fourth through sixth decades of life [2,4,5]. Although the pathogenesis remains unknown, HADD is common—with reports of deposits in 2.7–20% of adult shoulders [2,5].

HADD is a self-limited process and is often discovered incidentally on radiographs, with up to 20% of asymptomatic individuals found to have HADD; however, during its resorptive phase, HADD has an associated inflammatory process that is an important cause of joint pain, and represents the major diagnosis in up to 7% of painful shoulders [2]. HADD can involve any joint. The most commonly affected location is the shoulder, followed by hip, elbow, wrist, and knee [1]. Other less common locations include the ankle/foot and fingers [1,6]. When symptomatic, HADD may present with pain, redness, swelling, and/or limited range of motion of the affected joint.

There are numerous clinical presentations of HADD which mimic pathologies that require additional work-up or aggressive treatment. For example, symptoms of longus colli muscle involvement can mimic a retropharyngeal abscess [7]. Similarly, if unsuspected, HADD may be misdiagnosed as a neoplasm [8]. In this paper, we review HADD with an emphasis on distinguishing it from more ominous pathologies with similar imaging appearances. Familiarity with these cases will allow early and confident diagnosis, thereby reducing costly and time-consuming workup or intervention.

1.1. Pathogenesis and predisposing factors

Although HADD has been studied extensively since its first description by Painter in 1907 [8], its pathogenesis remains unknown. Four pathogenic pathways have been proposed [9]: (1) degenerative calcification, in which local tendon damage secondary to vascular ischemia or repetitive trauma is thought to promote deposition of calcified material [5,10]; (2) reactive or cell-mediated calcification, wherein chondrocytes mediate calcium deposition and phagocytosing cells contribute to resorption [11,12]; (3) a process similar to

* Corresponding author.
E-mail addresses: patcharee@somdej-mec.or.th (P. Hongsmatip), kcheng@ucsd.edu (K.Y. Cheng), christopher.kim@ucsf.edu (C. Kim), dl3ra@virginia.edu (D.A. Lawrence), Robert.Rivera@DignityHealth.org (R. Rivera), esmitaman@ucsd.edu (E. Smitaman).
[1] Senior author.

https://doi.org/10.1016/j.ejrad.2019.108653
Received 9 April 2019; Accepted 19 August 2019
0720-048X/ Published by Elsevier B.V.

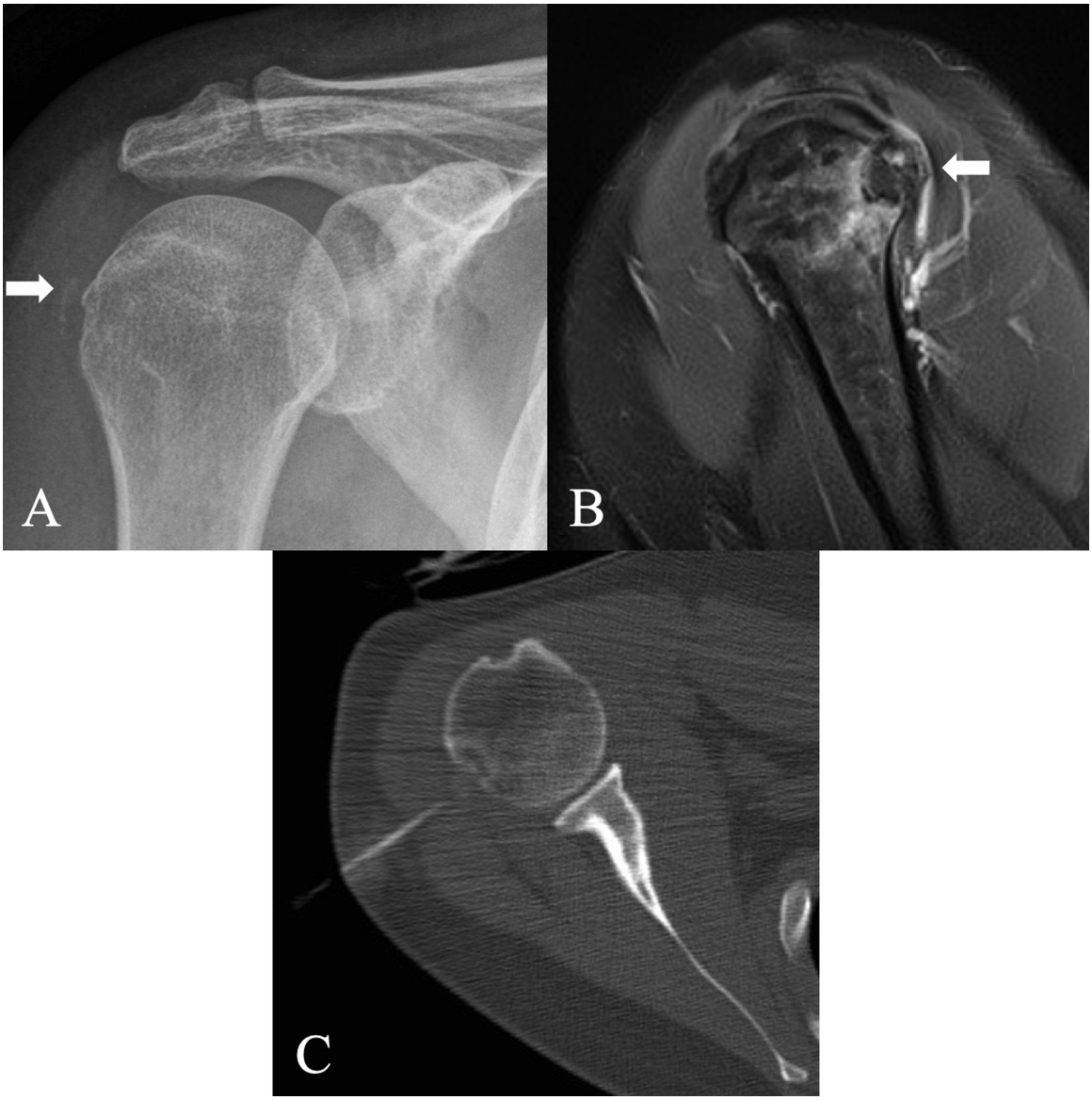

Fig. 1. Adult man referred to orthopedic oncology clinic for an initial diagnosis of tumor. AP radiograph with internal rotation (A) was read as normal; however, in retrospect there is a faint calcification (arrow) overlying the posterior aspect of the oblique facet and mainly the vertical facet of the greater tuberosity, that is consistent with the resorptive phase of HADD. Subsequent sagittal T2-weighted fat-suppressed image (B) of the shoulder shows a hypointense focus of calcium (arrow) with surrounding marrow and soft tissue edema. Biopsy was requested and axial CT with bone windows (C) shows HADD with subcortical extension. Using a spinal needle this was aspirated and confirmed histologically, and steroids were also administered.

endochondral ossification [13]; and (4) erroneous differentiation of tendon-derived stem cells into calcium-depositing chondrocytes or osteoblasts [14].

Predisposing risk factors for HADD include diabetes [15], thyroid and estrogen metabolism disorders [3,16], and genetic factors, such as the HLA-A1 genotype [17].

1.2. Stages of HADD

HADD changes with time and several classification schemes have been proposed, dividing it into various stages based on chronicity as described by DePalma [18] or radiographic appearance of the calcifications as in the French Society of Arthroscopy classification [19,20]. A frequently utilized system was first described by Uhthoff and Sarkar [11,12,21], which defines three stages: precalcific, calcific, and postcalcific.

In the precalcific stage, impaired perfusion and resultant focal hypoxia are thought to trigger fibrocartilaginous transformation at the site of future calcification.

The deposition of calcium crystals is termed the calcific stage, which is further subdivided into formative, resting, and resorptive phases. During the formative phase, the fibrocartilage is replaced by a calcific deposit. This deposit then dwells without associated inflammation or vascularity during the “resting phase”, which can be a variable length of time. Eventually, the focus may undergo the resorptive phase, during which the calcium deposit is surrounded by small vascular channels and invaded by macrophages, polymorphonuclear cells, and fibroblasts which phagocytose and remove the calcium and result in an ill-defined, irregular appearance of the calcification. It is during the resorptive phase, in which the calcific deposit can rupture into nearby tissues and trigger an acute inflammatory response, that HADD is acutely painful. Correlative magnetic resonance imaging (MRI) demonstrates edema or fluid signal surrounding the calcific focus [22].

The postcalcific stage occurs when the void from the resorptive

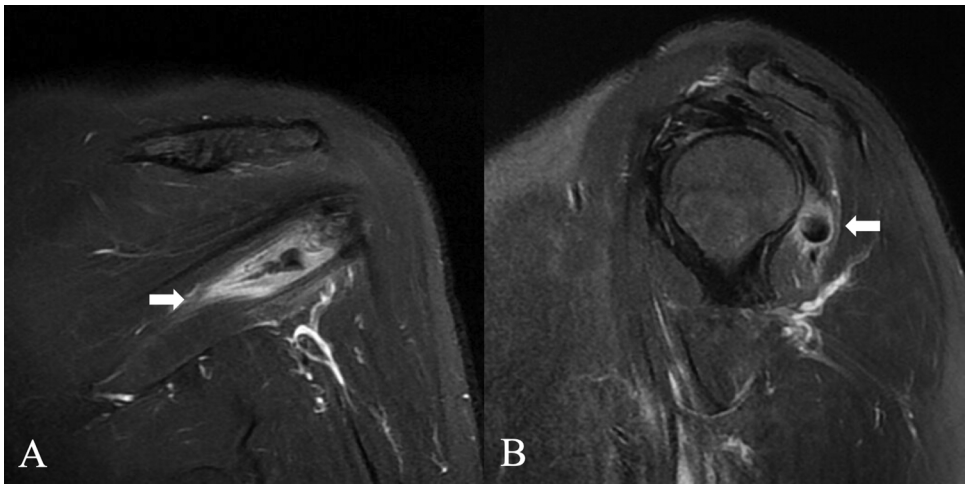

Fig. 2. 47-year-old woman with acute posterior shoulder pain. Coronal (A) and sagittal (B) T2-weighted fat suppressed images show intercompartmental migration of HADD (arrows) between the infraspinatus and teres minor muscles, owing to variant continuous fascia enveloping both muscles, and surrounding inflammation. (Courtesy of Donald Resnick, M.D., University of California San Diego, San Diego, Calif.).

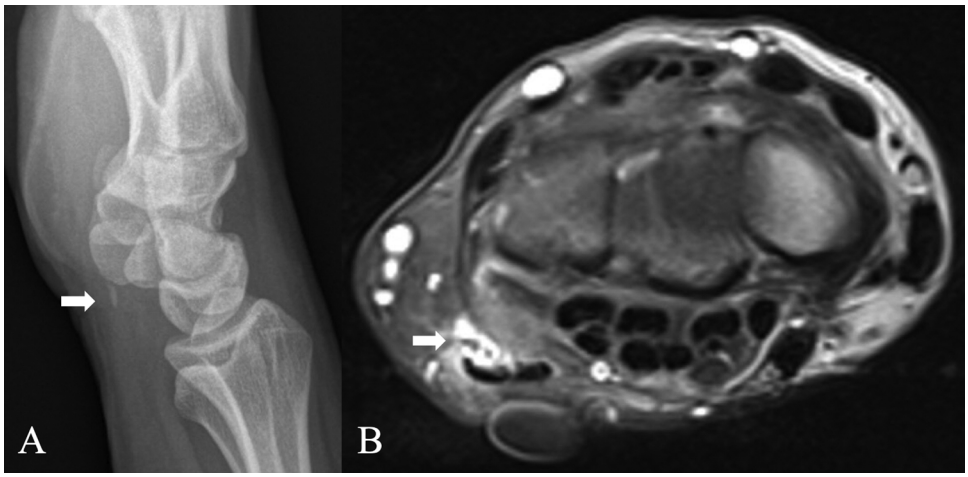

Fig. 3. 45-year-old woman with acute voloulnar wrist pain. Lateral radiograph (A) of the right wrist shows a subcentimeter calcification (arrow) near the flexor carpi ulnaris tendon insertion on the pisiform that was initially misdiagnosed as a fracture. Two days later, an axial T1-weighted fat-suppressed post contrast image (B) shows fragmentation of the calcium with surrounding inflammation (arrow), consistent with resorption of HADD.

phase of the calcific stage is replaced by granulation and scar tissue.

1.3. Imaging of HADD

Radiography is the ideal initial modality for evaluating HADD, as it is sensitive for the detection of calcifications, and can distinguish them from mature ossifications which will show a distinct cortical and/or trabecular bone pattern, or foreign bodies which will have sharp/geometric borders [23]. Calcifications within tendons are most frequently secondary to HADD or calcium pyrophosphate dihydrate deposition (CPPD), the latter of which typically has a more linear and delicate appearance and likely has associated articular calcification, i.e. chondrocalcinosis.

Once a radiodensity has been identified as HADD, it can be further characterized by its radiographic appearance. To this end, several classification systems based on size, morphology, and density of the calcification have been described [5,18,20,24,25]. Pragmatically applying Uhthoff and Sarkar's work to radiographs, HADD can be broadly categorized into the formative and resorptive phases. In the formative phase, which is typically asymptomatic or associated with chronic pain secondary to mechanical impingement, HADD will present as a well-defined, homogeneous and amorphous calcification. In the resorptive and acutely symptomatic phase, on the other hand, HADD typically appears ill-defined, inhomogeneous, and fluffy [22,26].

Computed tomography (CT) is superior to conventional radiography for detecting calcifications and is particularly useful for identifying osseous erosions or migration of calcium into nearby structures. CT may provide additional information to distinguish calcification from ossification based on Hounsfield unit (HU) values, with calcifications typically demonstrating lower HU values of 100–400, as compared to 700 and over 1500 for trabecular and cortical bone, respectively [23]. Additionally, HADD can have a characteristic "comet tail" appearance on CT, which results from the longitudinal orientation of calcified deposits along tendons such that they seem to taper away from a site of bone involvement [27].

Ultrasonography (US) is also useful in the evaluation of HADD. In a study of 951 patients who underwent bilateral shoulder sonography with correlative radiographs, calcifications were detected with ultrasound in 87 patients and radiographs in 93 patients [28]. As with radiography, multiple classifications have been proposed to describe the appearance of the calcifications [25]. In general, calcifications appear hyperechoic with or without acoustic shadowing. In keeping with Uhthoff, the sonographic appearance of HADD can be categorized as well-defined, arc-shaped, hyperechoic foci with posterior acoustic shadowing and no significant vascularity in the formative phase. During the symptomatic resorptive phase, calcifications appear ill-defined, non-arc shaped (fragmented, punctate, cystic, or nodular), less homogeneously hyperechoic with less well-defined posterior acoustic shadowing. Typically, symptomatic resorptive HADD will also demonstrate vascularity on color Doppler images [25,29–31]. The presence of vascularity distinguishes HADD calcifications from degenerative non-HADD calcifications, which occur in non-viable and dysvascularized tendons, typically at a tendon insertion or the edge of a tendon tear [19]. Degenerative calcifications will also tend to worsen over time, while HADD will typically resolve.

On MRI, HADD is hypointense on all pulse sequences, which can

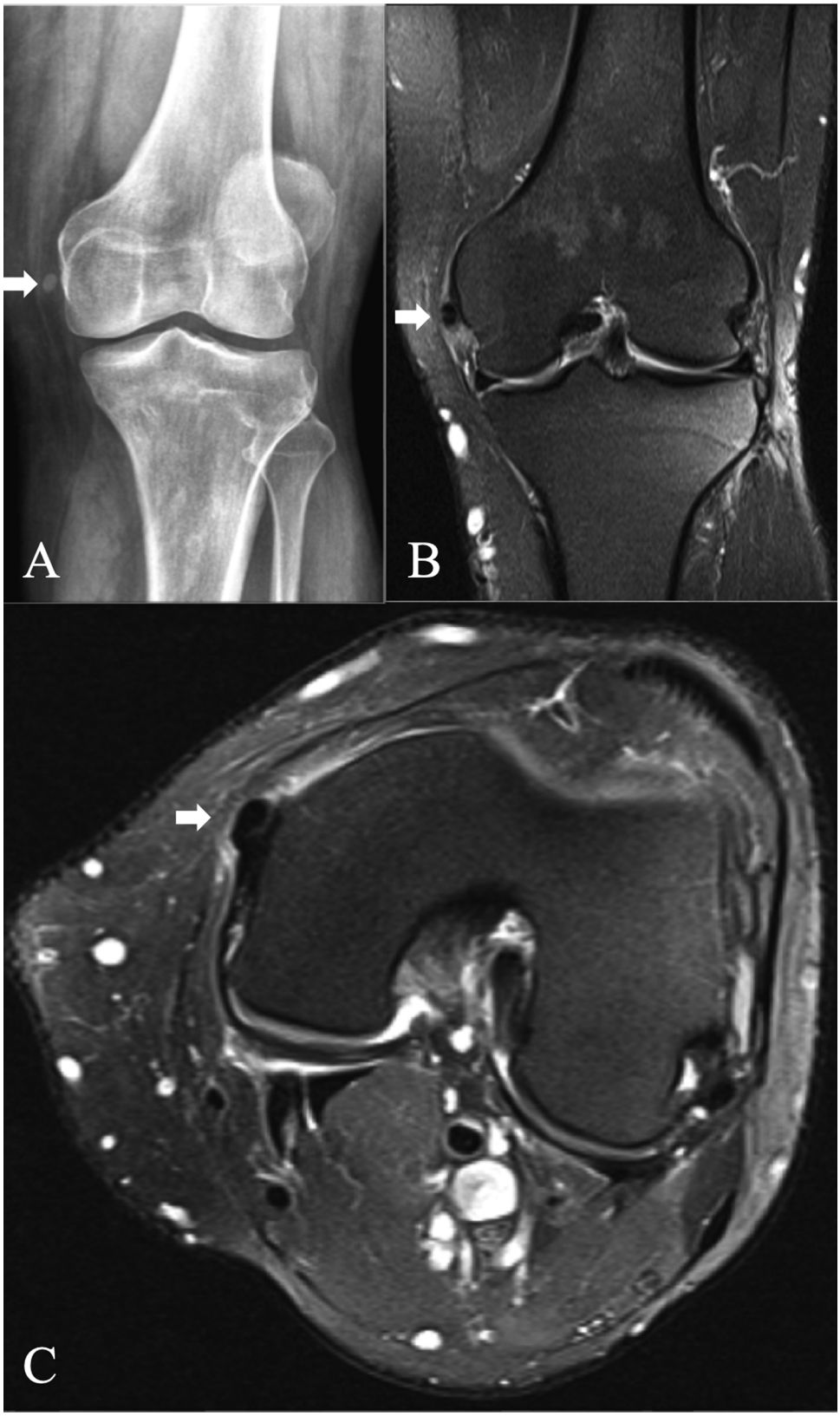

Fig. 4. 50-year-old man with medial knee pain. AP radiograph (A) of the left knee shows an ovoid calcification (arrow), consistent with HADD but initially read as a remote injury of the tibial collateral ligament. Coronal (B) and axial (C) T2-weighted fat suppressed sequences demonstrate HADD (arrows) within the proximal portion of the tibial collateral ligament with a mild amount of surrounding soft tissue edema.

make it difficult to identify without correlative radiographs. MR does not add significant diagnostic information in most cases [30], may not visualize calcifications [32,33], and can lead to a false positive diagnosis, for example, when normal hypointense parts of the rotator cuff mimic calcific deposits [34]. MRI can be helpful in detecting the pattern of migration during the resorptive phase, if any: bursal or

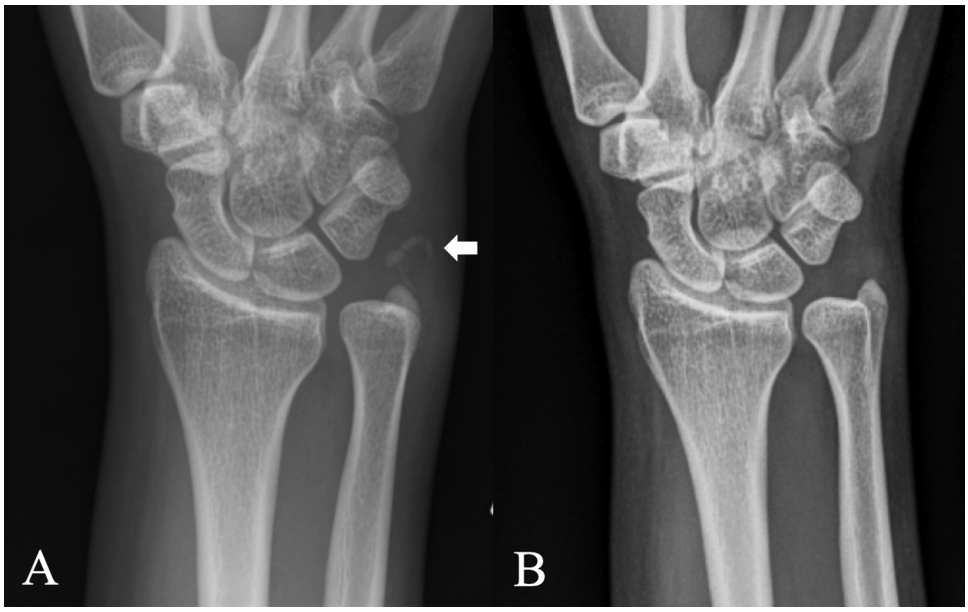

Fig. 5. 25-year-old woman volleyball player with a painful wrist mass. Initial AP radiograph (A) shows a C-shaped calcific aggregate just distal to the ulnar styloid (arrow). It was uncertain whether this was HADD or zonal organization seen in myositis ossificans/heterotopic ossification. A follow-up radiograph (B) was recommended and obtained 28 days later; there is near complete resolution of the calcifications—supporting the diagnosis of HADD.

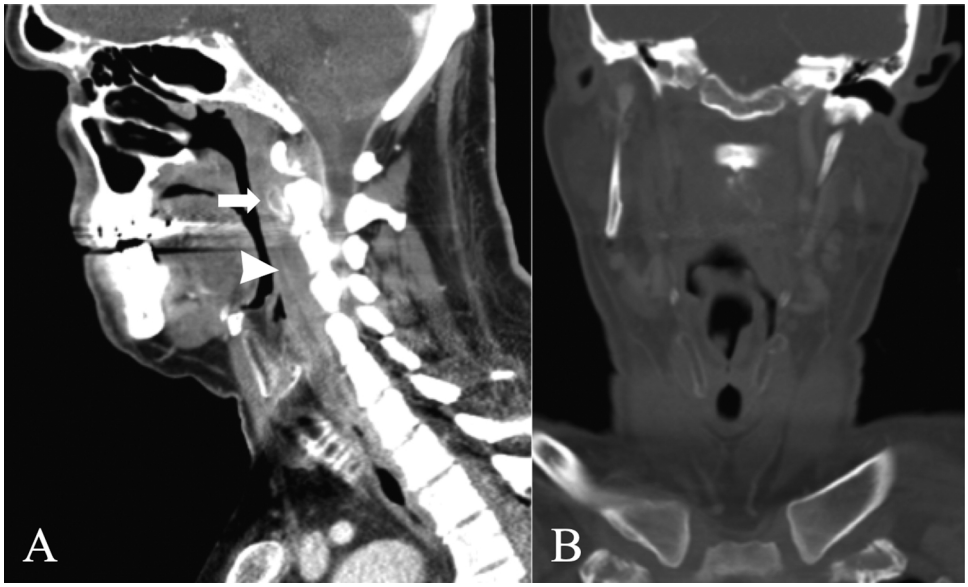

Fig. 6. 49-year-old man with acute neck pain and concern for retropharyngeal or peritonsillar abscess. Sagittal (A) and coronal (B) post contrast CT images of the neck show an ovoid peripherally calcified mass (arrow) inferior to the anterior arch of C1 and anterior to the body of C2 oriented along course of the superior oblique portion of the left longus colli muscle. Note also the subtle soft tissue edema (arrowhead) anterior to C3.

intratendinous toward the myotendinous junction [35], intramuscular [36], or intraosseous [8,27,37–39]. Migration of HADD into nearby tissues triggers an acute inflammatory response that is frequently seen on MRI as bursal fluid, soft tissue edema, or marrow edema about the deposit(s) [36] (Fig. 1). If prior radiographs are not available at the time of the initial MRI interpretation, the inflammatory findings of migratory HADD may mimic trauma or infection [36], and the cortical erosion due to intraosseous migration and/or presence of calcification may raise suspicion for a chondroid neoplasm [8]. When the diagnosis of HADD is suspected but not definitive, particularly in an uncommon location or unusual presentation, radiographic or CT correlation can be obtained to confirm the diagnosis.

1.4. Shoulder: the primary site of HADD involvement and usual presentation

As mentioned previously, HADD most commonly affects the shoulder. Specifically, the supraspinatus tendon is the most frequently involved rotator cuff tendon, which is involved in approximately half of cases with shoulder involvement [5]. Radiographs with the humeral head in external rotation readily show the calcific deposit in profile [4,40].

Infraspinatus, subscapularis, and teres minor tendon involvement are less common and better appreciated on internal rotation or axillary views of the shoulder [25,36,41,42]. HADD in the shoulder may be associated with a rotator cuff tear in 27–28% of patients [43,44]. Intercompartmental migration of HADD between the infraspinatus and teres minor can be seen in rare instances owing to variant continuous fascia enveloping both these muscles [45] (Fig. 2).

Other sites of HADD about the shoulder include the long head of the biceps musculotendinous junction, which can resemble a loose body within the long head of the biceps tendon sheath [46]; the pectoralis major tendon insertion [38,47], which may resemble the reactive cortical irregularity of the “ringman’s lesion” [48]; the insertion of the latissimus dorsi simulating a “pseudotumor” [49]; and the subacromial-subdeltoid bursa, which can give the appearance of a “skull cap” [50].

1.5. HADD presentations mimicking other pathologies

Given the variable appearance of HADD, it can be misdiagnosed as

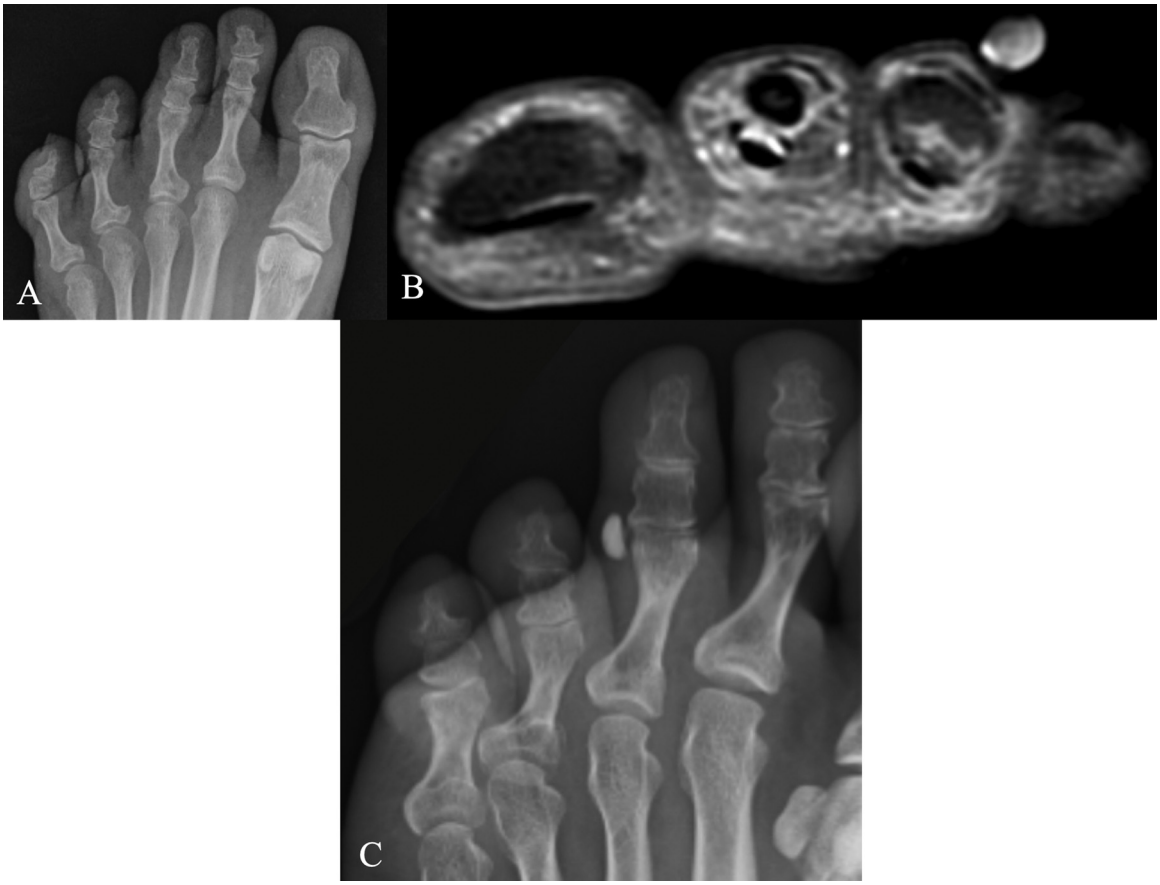

Fig. 7. 58-year-old woman with pain and swelling of the third toe. Initial radiograph of the foot (A) shows a mild amount of nonspecific soft tissue swelling of the third toe. Short axis T2-weighted fat-suppressed image (B) shows a mild amount of nonspecific soft tissue edema of the third toe. Note the fiduciary marker at the dorsolateral aspect of the third toe. The patient had mentioned an outside diagnosis of a "rock" in her toe; those radiographs were obtained (C) and confirms the diagnosis of HADD, which has undergone resorption.

trauma, infection, or tumor. This may result in unnecessary additional imaging, intervention (e.g. biopsy), and patient angst.

1.5.1. Mimic of acute or subacute trauma

The diagnosis of HADD may be confounded by the setting of a recent trauma. It can be mistaken for a small acute avulsion fracture fragment, especially when the calcific deposit is small and occurs in uncommon locations, such as the origin of the long head of the triceps tendon [51], hand [52–54] and wrist (Fig. 3). The two entities can be differentiated by a history of trauma, the homogenous appearance of HADD, and the partially corticated jigsaw puzzle piece appearance of a fracture fragment that "fits" into the defect of the parent bone [26].

Osseous—cortical, subcortical, or intramedullary—migration of HADD coupled with a history of recent trauma can mislead one to the diagnosis of bone contusion or fracture [55]. For instance, substantial subcortical involvement of the greater tuberosity of the humerus with underlying inflammatory marrow edema on MRI may be mistaken for a Hill-Sachs lesion, which often presents as a notched defect of the posterolateral aspect of the humeral head with associated intermediate to high subcortical signal intensity [56]. In addition, punctate residual HADD in the nearby tendon on an accompanying radiograph or CT may be mistaken for fracture fragments. HADD can also extend further into the intramedullary space [55], which may lead to its misdiagnosis as a subacute fracture with partial healing; however, HADD is typically less dense and organized as compared to callus formation.

When intratendinous migration of HADD occurs, the striated pattern of reactive edema in the surrounding rotator cuff musculature can mimic injury of the myotendinous junction [36]. This is further complicated by the globular appearance of torn and retracted tendon fibers, which can resemble HADD, and by concomitant tendon calcifications occurring in up to 11% of myotendinous injuries [57]. An accurate history of acute trauma, patient demographics, and correlative radiographs are helpful in differentiating myotendinous injury from HADD, as the former typically occurs in younger patients with a history of trauma and no visible calcifications on radiographs.

1.5.2. Mimic of chronic trauma and normal ossicles

HADD can mimic chronic fracture fragments, myositis ossificans (MO)/heterotopic ossification (HO), or normal accessory ossicles. A small focus of HADD in the tibial collateral ligament or tibial collateral ligament bursa may be confused for a remote avulsion fracture, the so-called Pellegrini-Stieda lesion [58–61] (Fig. 4); however, Pellegrini-Stieda lesions are typically curvilinear, while HADD is rounded and lobate in appearance [58]. Additionally, Pellegrini-Stieda lesions represent ossification rather than calcification of the tibial collateral ligament and should demonstrate a distinct trabecular pattern on radiographs with marrow signal on MRI (hyperintense on T1 weighted images and intermediate to low signal on fat-suppressed images) [62], while HADD appears homogeneously dense [58]. HADD in the symptomatic phase will also demonstrate surrounding edema on MRI.

HADD may also be mistaken for immature MO/HO in an atypical location such as the shoulder [63] or hand [64,65]. Although mature MO/HO demonstrates characteristic zonal mineralization (i.e. cortical bone peripherally and internal trabeculae) after approximately 6–8 weeks [1,66,67], immature MO/HO may present with only faint calcifications. Follow-up radiographs are most useful in distinguishing the two entities, as HADD will likely remain the same (resting phase) or resolve (resorptive phase), while MO/HO will mature over time (Fig. 5).

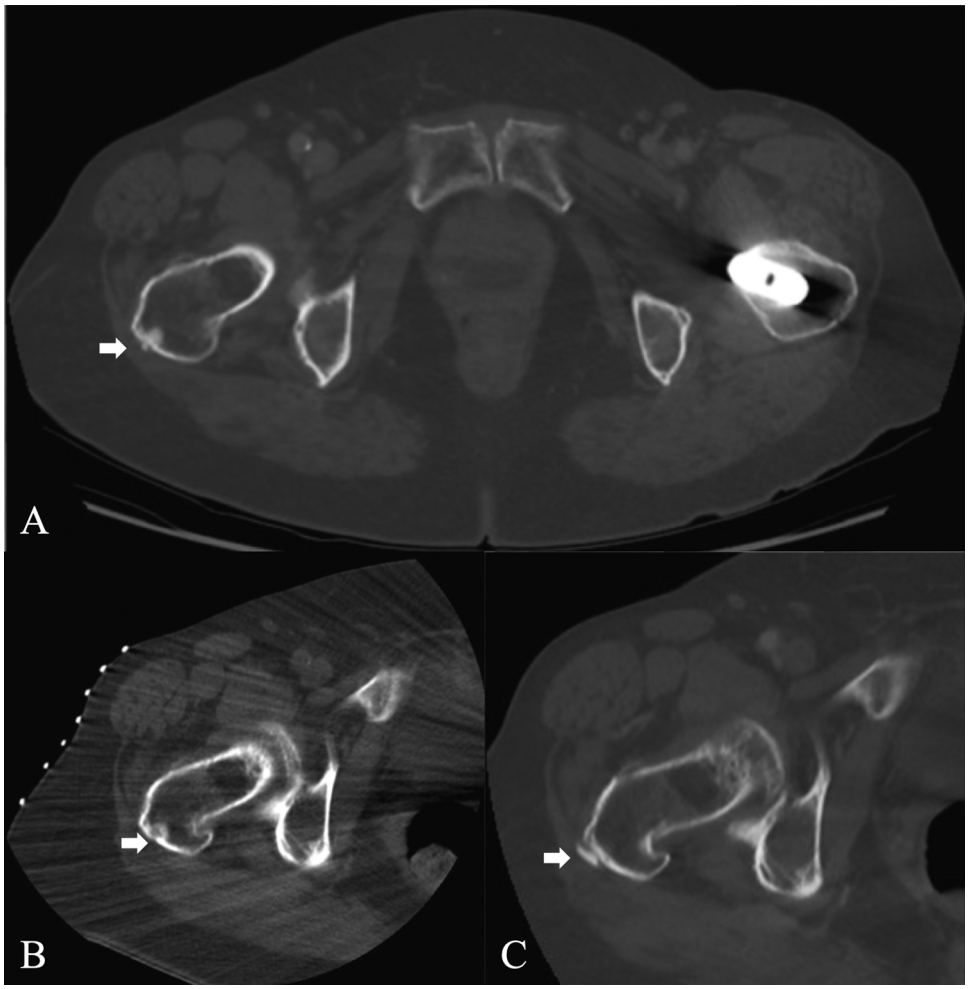

Fig. 8. 79-year-old woman with known breast cancer and right hip pain initially misdiagnosed with osseous metastasis involving the right greater trochanter. Initial axial CT image (A) shows a subcentimeter calcification (arrow) at the lateral facet of the greater trochanter. Also note the subtle focus of calcification beyond the cortex, which would be unusual for metastatic disease and suggests the so-called "comet tail" sign of HADD instead. At the time of biopsy (note the CT-guidance markers along the anterolateral right hip), an axial CT image (B) shows disappearance of the calcification (arrow), consistent with the resorptive phase of HADD. Prior CT image (C) was requested and confirms HADD of the gluteus medius tendon (arrow) before intraosseous migration.

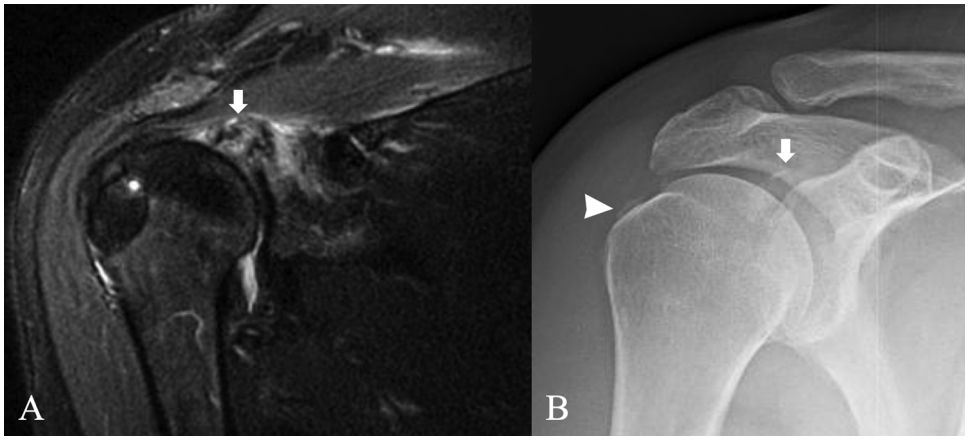

Fig. 9. 38-year-old man initially misdiagnosed with a tumor of the superior glenoid. Coronal T2-weighted fat-suppressed image (A) shows a heterogeneous mass in the region of the biceps-labral complex (arrow) with nearby marrow edema within the superior glenoid. Prior radiograph (B) was requested and confirms the diagnosis of HADD involving the biceps-labral complex (arrow), as well as the infraspinatus footprint (arrowhead), which was difficult to discern from tendon on the MRI (not shown).

Finally, HADD may be misperceived as a normal accessory ossicle, but ossicles will have a cortical rim with internal trabeculae and occur in typical locations [1].

1.5.3. Mimic of infection

During the symptomatic phase, the clinical presentation of HADD may mimic infection with low-grade fever, soft tissue swelling, erythema, limited range of motion of the affected joint, and abnormal laboratory values (including mild leukocytosis, elevated erythrocyte sedimentation rate, and elevated C-reactive protein) [68–73]. For instance, involvement of the superior oblique portion of the longus colli muscle often results in neck pain/stiffness and elevated inflammatory markers that clinically mimics neck infections, such as retropharyngeal abscess or meningitis [7,74–76]. The diagnosis of HADD of the longus colli muscle is readily made on a well-aligned lateral radiograph or CT of the neck or cervical spine, and the calcification is usually anteroinferior to the anterior arch of C1 but is occasionally seen anterior to C2 [77](Fig. 6).

HADD may also mimic pyogenic myositis as deposits migrate into nearby muscles [36]. Although pyogenic myositis typically presents with an associated abscess, early infection may demonstrate only T2 hyperintense signal and heterogeneous contrast enhancement of the

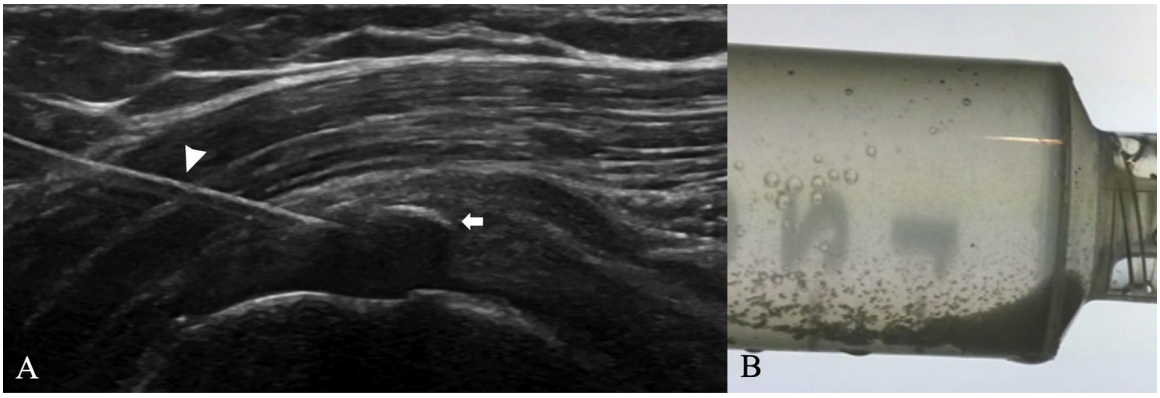

Fig. 10. 42-year-old woman with acute exacerbation of chronic shoulder pain undergoing ultrasound-guided lavage of HADD. Grayscale ultrasound image (A) shows hyperechoic superficial rim with posterior acoustic shadowing (arrow) representing HADD. An 18-gauge spinal needle (arrowhead) was used to lavage this deposit of calcium hydroxyapatite using a lidocaine and sterile saline mixture followed by steroid injection. Photograph of syringe (B) shows layering calcium hydroxyapatite.

affected muscle [76,78], which can be seen with HADD migration [36]. In such cases, history of predisposing factors for infection such as intravenous drug use or immunocompromised state, and careful inspection for calcification on radiographs may be the only clues to distinguishing these two entities.

The clinical presentation of HADD of the fingers or toes can also be confused for acute septic arthritis, but can be distinguished by calcific deposits about the affected joint(s) [69,70] (Fig. 7). Alternatively, if MR is performed, absence of synovial enhancement and thickening or joint effusions, characteristic of septic arthritis [79], may distinguish HADD from infection.

1.5.4. Mimic of tumor

The imaging appearance of HADD as it migrates into nearby bone may raise concern for an aggressive neoplasm, as osseous erosion with periosteal reaction and soft tissue calcification are also seen with juxtacortical chondroma or chondrosarcoma and with secondary bone invasion by soft tissue sarcomas [8,27,55,80]. In the setting of cortical erosion, two key distinguishing features of HADD are (1) continuity with a tendon calcification [55] and (2) a tapered configuration of the calcification producing a “comet tail” appearance [27] (Fig. 8), which are best appreciated on CT. Other findings favoring HADD include the absence of a discrete soft tissue mass, HADD involving another tendon or joint (Fig. 9), and the characteristic appearance of resorptive phase calcification with “snowy” density on CT or central high signal intensity on T1 weighted images of MR [8,80].

When classic imaging features are not present and biopsy is unavoidable, it is crucial to communicate the suspected diagnosis of HADD to the pathologist because histologic findings of chondroid metaplasia, seen in HADD, can be misdiagnosed as a chondroid neoplasm [8].

1.6. A brief word on the treatment of HADD and the role of the radiologist

When HADD presents acutely in the resorptive phase, it is a self-limited process and frequently resolves spontaneously within two to three weeks [81]. During the symptomatic phase, the primary management is conservative with non-steroidal anti-inflammatory drugs (NSAIDs) or physiotherapy [22,25,26,30,38,82]. Non-invasive treatment with acetic acid iontophoresis, ultrasound therapy [83], and extracorporeal shock wave therapy [84–87] have also been described. However, the mainstay of treatment is one which can be performed by the radiologist: percutaneous needle aspiration and lavage of the calcific deposits (i.e. barbotage) under ultrasound guidance (Fig. 10), which has been described in detail by Aina et al. [88] and others [25,61,89,90]. Ultrasound-guided barbotage has been shown in a large-scale meta-analysis of 920 studies to be more effective than extracorporeal shock wave therapy or subacromial corticosteroid treatment [83]. For recalcitrant cases, arthroscopic removal of calcific deposits may be considered [91], although there is no consensus on optimal surgical technique [92].

2. Conclusion

HADD can mimic a variety of pathologies. It may be confused for trauma or infection due to its associated inflammatory response or resemble neoplasm due to its propensity for cortical erosion. It is important to remember the diagnosis of HADD, particularly its characteristic radiographic appearance, to avoid additional unnecessary workup or intervention.

Financial disclosure(s)

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Grant support

None.

IRB statement

No IRB approval was required for this review.

Declaration of Competing Interest

None.

References

[1] C.W. Hayes, W.F. Conway, Calcium hydroxyapatite deposition disease, RadioGraphics 10 (November (6)) (1990) 1031–1048.
[2] C.A. Speed, B.L. Hazleman, Calcific tendinitis of the shoulder, N. Engl. J. Med. 340 (May (20)) (1999) 1582–1584.
[3] P. Harvie, T.C.B. Pollard, A.J. Carr, Calcific tendinitis: natural history and association with endocrine disorders, J. Shoulder Elbow Surg. 16 (March (2)) (2007) 169–173.
[4] C.S. Resnik, D. Resnick, Crystal deposition disease, Semin. Arthritis Rheum. 12 (May (4)) (1983) 390–403.
[5] B.M. Bosworth, Calcium deposits in the shoulder and subacromial bursitis: a survey of 12,122 shoulders, J. Am. Med. Assoc. 116 (May (22)) (1941) 2477–2482.
[6] K.-B. Lee, K.-J. Song, H.-S. Kwak, S.-Y. Lee, Acute calcific periarthritis of proximal interphalangeal joint in a professional golfer’s hand, J. Korean Med. Sci. 19 (December (6)) (2004) 904–906.
[7] B. Suh, J. Eoh, J. Shin, Clinical and imaging features of longus colli calcific tendinitis: an analysis of ten cases, Clin. Orthop. Surg. 10 (June (2)) (2018) 204–209.
[8] D.J. Flemming, M.D. Murphey, K.M. Shekitka, H.T. Temple, J.J. Jelinek, M.J. Kransdorf, Osseous involvement in calcific tendinitis: a retrospective review of

50 cases, Am. J. Roentgenol. 181 (October (4)) (2003) 965–972.
[9] F. Oliva, A.G. Via, N. Maffulli, Physiopathology of intratendinous calcific deposition, BMC Med. 23 (August (10)) (2012) 95.
[10] W.A. Bishop, Calcification of the supraspinatus tendon: cause, pathologic picture, and relation to the scalneus anticus syndrome, Arch. Surg. 39 (August (2)) (1939) 231–246.
[11] H.K. Uhthoff, Calcific tendinopathy of the rotator cuff: pathogenesis, diagnosis, and management, J. Am. Acad. Orthop. Surg. 5 (July (4)) (1997) 183–191.
[12] H.K. Uhthoff, K. Sarkar, J.A. Maynard, Calcifying tendinitis: a new concept of its pathogenesis, Clin. Orthop. 118 (August) (1976) 164–168.
[13] M. Benjamin, A. Rufai, J.R. Ralphs, The mechanism of formation of bony spurs (enthesophytes) in the Achilles tendon, Arthritis Rheum. 43 (3) (2000) 576–583.
[14] Y. Rui, P.P. Lui, L. Chan, K. Chan, S. Fu, G. Li, Does erroneous differentiation of tendon-derived stem cells contribute to the pathogenesis of calcifying tendinopathy? Chin. Med. J. (Engl.) 124 (February (4)) (2011) 606–610.
[15] M.E. Mavrikakis, S. Drimis, D.A. Kontoyannis, A. Rasidakis, E.S. Moulopoulou, S. Kontoyannis, Calcific shoulder periarthritis (tendinitis) in adult onset diabetes mellitus: a controlled study, Ann. Rheum. Dis. 48 (March (3)) (1989) 211–214.
[16] V. Wright, A.M. Haq, Periarthritis of the shoulder. I. Aetiological considerations with particular reference to personality factors, Ann. Rheum. Dis. 35 (June (3)) (1976) 213–219.
[17] D.P.S. Sengar, R.J. McKendry, H.K. Uhthoff, Increased frequency of HLA-A1 in calcifying tendinitis, Tissue Antigens 29 (3) (1987) 173–174.
[18] A.F. DePalma, J.S. Kruper, Long-term study of shoulder joints afflicted with and treated for calcific tendinitis | Ovid, Clin. Orthop. 20 (1961) 61–72.
[19] M.T. ElShewy, Calcific tendinitis of the rotator cuff, World J. Orthop. 7 (January (1)) (2016) 55–60.
[20] D. Molé, J.F. Kempf, P. Gleyze, B. Rio, F. Bonnomet, G. Walch, Results of endoscopic treatment of non-broken tendinopathies of the rotator cuff. 2. Calcifications of the rotator cuff, Rev. Chir. Orthop. Reparatrice Appar. Mot. 79 (7) (1993) 532–541.
[21] H.K. Uhthoff, K. Sarkar, Calcifying tendinitis, Baillières Clin, Rheumatol. 3 (December (3)) (1989) 567–581.
[22] D.S. Siegal, J.S. Wu, J.S. Newman, J.L. del Cura, M.G. Hochman, Calcific tendinitis: a pictorial review, Can. Assoc. Radiol. J. 60 (December (5)) (2009) 263–272.
[23] V. Freire, T.P. Moser, M. Lepage-Saucier, Radiological identification and analysis of soft tissue musculoskeletal calcifications, Insights Imaging 9 (June (4)) (2018) 477–492.
[24] J. Gärtner, A. Heyer, [Calcific tendinitis of the shoulder], Orthopade 24 (June (3)) (1995) 284–302.
[25] V. Chianca, D. Albano, C. Messina, F. Midiri, G. Mauri, A. Aliprandi, M. Catapano, L.C. Pescatori, C.G. Monaco, S. Gitto, A. Pisani Mainini, A. Corazza, S. Rapisarda, G. Pozzi, A. Barile, C. Masciocchi, L.M. Sconfienza, Rotator cuff calcific tendinopathy: from diagnosis to treatment, Acta Bio-Med. Atenei Parm. 89 (January (1-S)) (2018) 186–196.
[26] N.M. Beckmann, Calcium apatite deposition disease: diagnosis and treatment, Radiol. Res. Pract. [Internet] (2016) [cited 2019 Feb 14]; Available from: https://www.hindawi.com/journals/rrp/2016/4801474/abs/.
[27] C. Hayes, D. Rosenthal, M. Plata, T. Hudson, Calcific tendinitis in unusual sites associated with cortical bone erosion, Am. J. Roentgenol. 149 (November (5)) (1987) 967–970.
[28] P.U. Farin, H. Jaroma, Sonographic findings of rotator cuff calcifications, J. Ultrasound Med. 14 (1) (1995) 7–14.
[29] H.-J. Chiou, Y.-H. Chou, J.-J. Wu, C.-C. Hsu, D.-Y. Huang, C.-Y. Chang, Evaluation of calcific tendonitis of the rotator cuff, J. Ultrasound Med. 21 (3) (2002) 289–295.
[30] G. Merolla, S. Singh, P. Paladini, G. Porcellini, Calcific tendinitis of the rotator cuff: state of the art in diagnosis and treatment, J. Orthop. Traumatol. Off. J. Ital. Soc. Orthop. Traumatol. 17 (March (1)) (2016) 7–14.
[31] B. Le Goff, J.-M. Berthelot, P. Guillot, J. Glémarec, Y. Maugars, Assessment of calcific tendonitis of rotator cuff by ultrasonography: comparison between symptomatic and asymptomatic shoulders, Joint Bone Spine 77 (May (3)) (2010) 258–263.
[32] G.F. Bachmann, C. Melzer, C.M. Heinrichs, B. Möhring, M.B. Rominger, Diagnosis of rotator cuff lesions: comparison of US and MRI on 38 joint specimens, Eur. Radiol. 7 (March (2)) (1997) 192–197.
[33] M. Maier, A. Stäbler, C. Schmitz, A. Lienemann, S. Köhler, H.R. Dürr, M. Pfahler, H.J. Refior, On the impact of calcified deposits within the rotator cuff tendons in shoulders of patients with shoulder pain and dysfunction, Arch. Orthop. Trauma Surg. 121 (July (7)) (2001) 371–378.
[34] C. Zubler, B. Mengiardi, M.R. Schmid, J. Hodler, B. Jost, C.W.A. Pfirrmann, MR arthrography in calcific tendinitis of the shoulder: diagnostic performance and pitfalls, Eur. Radiol. 17 (June (6)) (2007) 1603–1610.
[35] M.R. Abreu, M. Recht, MR imaging of the rotator cuff and rotator interval, in: J. Hodler, R.A. Kubik-Huch, G.K. von Schulthess (Eds.), Musculoskeletal Diseases 2017-2020: Diagnostic Imaging [Internet], Springer International Publishing, Cham, 2017[cited 2019 Feb 19]. p. 203–14. Available from: https://doi.org/10.1007/978-3-319-54018-4_19.
[36] B.P.G. Pereira, E.Y. Chang, D.L. Resnick, M.N. Pathria, Intramuscular migration of calcium hydroxyapatite crystal deposits involving the rotator cuff tendons of the shoulder: report of 11 patients, Skeletal Radiol. 45 (January (1)) (2016) 97–103.
[37] W.C. Sola, G.N. Drake, C.H. Ramos, A. Gomes, G.M. Gartsman, Calcific tendinitis of the rotator cuff associated with intraosseous loculation: two case reports, J. Shoulder Elbow Surg. 18 (November (6)) (2009) e6–e8.
[38] E.J. Kraemer, G.Y. El-Khoury, Atypical calcific tendinitis with cortical erosions, Skeletal Radiol. 29 (December (12)) (2000) 690–696.
[39] P. Fritz, T. Bardin, J.-D. Laredo, J.-M. Ziza, G. D'Anglejan, J. Lansaman, B. Bucki, M. Forest, D. Kuntz, Paradiaphyseal calcific tendinitis with cortical bone erosion - Fritz - 1994 - Arthritis & Rheumatism - Wiley Online Library, . 37 (May (5)) (1994) 718–723.
[40] B.M. Bosworth, Examination of the shoulder for calcium deposits: technique of fluoroscopy and spot-film roentgenography, JBJS 23 (July (3)) (1941) 567.
[41] B. Umamahesvaran, S.N. Sambandam, V. Mounasamy, P.P. Gokulakrishnan, M. Ashraf, Calcifying tendinitis of shoulder: a concise review, J. Orthop. 15 (September (3)) (2018) 776–782.
[42] M. Becciolini, G. Bonacchi, S. Galletti, Intramuscular migration of calcific tendinopathy in the rotator cuff: ultrasound appearance and a review of the literature, J. Ultrasound 19 (March (3)) (2016) 175–181.
[43] H.-C. Hsu, J.-J. Wu, Y.-F. Jim, C.-Y. Chang, W.-H. Lo, D.-J. Yang, Calcific tendinitis and rotator cuff tearing: a clinical and radiographic study, J. Shoulder Elbow Surg. 3 (May (3)) (1994) 159–164.
[44] Y.F. Jim, H.C. Hsu, C.Y. Chang, J.J. Wu, T. Chang, Coexistence of calcific tendinitis and rotator cuff tear: an arthrographic study, Skeletal Radiol. 22 (April (3)) (1993) 183–185.
[45] D. Chafik, L.M. Galatz, J.D. Keener, H.M. Kim, K. Yamaguchi, Teres minor muscle and related anatomy, J. Shoulder Elbow Surg. 22 (January (1)) (2013) 108–114.
[46] A. Goldman, Calcific tendinitis of the long head of the biceps brachii distal to the glenohumeral joint: plain film radiographic findings, Am. J. Roentgenol. 153 (November (5)) (1989) 1011–1016.
[47] J. Cahir, A. Saifuddin, Calcific tendonitis of pectoralis major: CT and MRI findings, Skeletal Radiol. 34 (April (4)) (2005) 234–238.
[48] M.N. Fulton, J.P. Albright, G.Y. El-Khoury, Cortical desmoid-like lesion of the proximal humerus and its occurrence in gymnasts (ringman's shoulder lesion), Am. J. Sports Med. 7 (January (1)) (1979) 57–61.
[49] S.E. Anderson, R. Hertel, J.O. Johnston, E. Stauffer, E. Leinweber, L.S. Steinbach, Latissimus dorsi tendinosis and tear: imaging features of a pseudotumor of the upper limb in five patients, Am. J. Roentgenol. 185 (November (5)) (2005) 1145–1151.
[50] G.D. Vigario, T.E. Keats, Localization of calcific deposits in the shoulder, Am. J. Roentgenol. 108 (April (4)) (1970) 806–811.
[51] J. Earwaker, Isolated avulsion fracture of the lesser tuberosity of the humerus, Skeletal Radiol. 19 (February (2)) (1990) 121–125.
[52] D.F. Dilley, M.A. Tonkin, Acute calcific tendinitis in the hand and wrist, J. Hand Surg. Br. Eur. 16 (May (2)) (1991) 215–216.
[53] R.E. Carroll, W. Sinton, A. Garcia, Acute calcium deposits in the hand, J. Am. Med. Assoc. 157 (January (5)) (1955) 422–426.
[54] A. Munjal, P. Munjal, A. Mahajan, Diagnostic dilemma: acute calcific tendinitis of flexor digitorum profundus, Hand N. Y. N. 8 (September (3)) (2013) 352–353.
[55] J. Malghem, P. Omoumi, F. Lecouvet, B. Vande Berg, Intraosseous migration of tendinous calcifications: cortical erosions, subcortical migration and extensive intramedullary diffusion, a SIMS series, Skeletal Radiol. 44 (October (10)) (2015) 1403–1412.
[56] T.L. Workman, T.K. Burkhard, D. Resnick, W.B. Goff, Z.N. Balsara, D.J. Davis, J.M. Lapoint, Hill-Sachs lesion: comparison of detection with MR imaging, radiography, and arthroscopy, Radiology 185 (December (3)) (1992) 847–852.
[57] A.K. Taneja, S.V. Kattapuram, C.Y. Chang, F.J. Simeone, M.A. Bredella, M. Torriani, MRI findings of rotator cuff myotendinous junction injury, Am. J. Roentgenol. 203 (July (2)) (2014) 406–411.
[58] Y. Kamawal, A.F. Steinert, B.M. Holzapfel, M. Rudert, T. Barthel, Case report - calcification of the medial collateral ligament of the knee with simultaneous calcifying tendinitis of the rotator cuff, BMC Musculoskelet. Disord. [Internet] (2016) [cited 2019 Feb 20];17. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4944491/.
[59] H.L. Mansfield, A. Trezies, Calcific tendonitis of the medial collateral ligament, Emerg. Med. J. 26 (July (7)) (2009) 543.
[60] M. Muschol, I. Müller, W. Petersen, J. Hassenpflug, Symptomatic calcification of the medial collateral ligament of the knee joint: a report about five cases, Knee Surg. Sports Traumatol. Arthrosc. 13 (October (7)) (2005) 598–602.
[61] F. Del Castillo-González, J.J. Ramos-Álvarez, J. González-Pérez, E. Jiménez-Herranz, G. Rodríguez-Fabián, Ultrasound-guided percutaneous lavage of calcific bursitis of the medial collateral ligament of the knee: a case report and review of the literature, Skeletal Radiol. 45 (October (10)) (2016) 1419–1423.
[62] L.F.A. Mendes, M.L. Pretterklieber, J.H. Cho, G.M. Garcia, D.L. Resnick, C.B. Chung, Pellegrini–stieda disease: a heterogeneous disorder not synonymous with ossification/calcification of the tibial collateral ligament—anatomic and imaging investigation, Skeletal Radiol. 35 (December (12)) (2006) 916–922.
[63] D.K. Martin, S.P. Zadow, S. Pandit, M.S. Falworth, Myositis ossificans of the subscapularis: an unusual case of restricted internal rotation, J. Shoulder Elbow Surg. 17 (July (4)) (2008) e21–e24.
[64] M. Chadha, A. Agarwal, Myositis ossificans traumatica of the hand, Can. J. Surg. 50 (December (6)) (2007) E21–E22.
[65] S. Kusuma, G.M. Lourie, R.E. Lins, Myositis ossificans of the hand, J. Hand Surg. Br. Eur. Vol. 24 (February (1)) (1999) 128–130.
[66] M.J. Kransdorf, J.M. Meis, From the archives of the AFIP. Extraskeletal osseous and cartilaginous tumors of the extremities, RadioGraphics 13 (July (4)) (1993) 853–884.
[67] L.S. Steinbach, Calcium pyrophosphate dihydrate and calcium hydroxyapatite crystal deposition diseases: imaging perspectives, Radiol. Clin. North Am. 42 (January (1)) (2004) 185–205.
[68] D.J. McCarty, R.A. Gatter, Recurrent acute inflammation associated with focal apatite crystal deposition, Arthritis Rheum. 9 (6) (1966) 804–819.
[69] A.G. Pollen, Calcareous deposits about the metacarpo-phalangeal joints, J. Bone Joint Surg. Br. 43-B (May (2)) (1961) 250–255.
[70] C.L. Selby, Acute calcific tendinitis of the hand: an infrequently recognized and

frequently misdiagnosed form of periarthritis, Arthritis Rheum. 27 (March (3)) (1984) 337–340.
[71] H. Kuroda, Y. Wada, K. Nishiguchi, T. Ninomiya, A. Takahama, S. Sato, H. Kitagaki, A case of probable hydroxyapatite deposition disease (HADD) of the hip, Magn. Reson. Med. Sci. 3 (3) (2004) 141–144.
[72] J.D. Wiper, A. Garrido, Acute calcific tendinitis, N. Engl. J. Med. 359 (December (23)) (2008) 2477.
[73] M. Hammoudeh, Calcific tendinitis mimicking acute sternoclavicular joint arthritis, Rheumatology 40 (November (11)) (2001) 1316–1317.
[74] T.K. Patel, J.C. Weis, Acute neck pain in the ED: consider longus colli calcific tendinitis vs meningitis, Am. J. Emerg. Med. 35 (June (6)) (2017) 943.e3-943.e4.
[75] A. Shawky, B. Elnady, E. El-Morshidy, W. Gad, A. Ezzati, Longus colli tendinitis. A review of literature and case series, SICOT-J. [Internet] (2019) [cited 2019 Feb 20];3. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5492790/.
[76] A.H. Zibis, D. Giannis, K.N. Malizos, P. Kitsioulis, D.L. Arvanitis, Acute calcific tendinitis of the longus colli muscle: case report and review of the literature, Eur. Spine J. 22 (May (Suppl 3)) (2013) 434–438.
[77] P.D. Holt, T.E. Keats, Calcific tendinitis: a review of the usual and unusual, Skeletal Radiol. 22 (January (1)) (1993) 1–9.
[78] J. Garcia, MRI in inflammatory myopathies, Skeletal Radiol. 29 (August (8)) (2000) 425–438.
[79] M. Karchevsky, M.E. Schweitzer, W.B. Morrison, J.A. Parellada, MRI findings of septic arthritis and associated osteomyelitis in adults, Am. J. Roentgenol. 182 (January (1)) (2004) 119–122.
[80] A. Marinetti, M. Sessa, A. Falzone, S.W. Della Sala, Intraosseous migration of tendinous calcifications: two case reports, Skeletal Radiol. 47 (January (1)) (2018) 131–136.
[81] P.H. Harmon, Methods and results in the treatment of 2,580 painful shoulders: With special reference to calcific tendinitis and the frozen shoulde, Am. J. Surg. 95 (Apr (4)) (1958) 527–544.
[82] G. Hurt, C.L. Baker, Calcific tendinitis of the shoulder, Orthop. Clin. 34 (October (4)) (2003) 567–575.
[83] G.R. Ebenbichler, C.B. Erdogmus, K.L. Resch, M.A. Funovics, F. Kainberger, G. Barisani, M. Aringer, P. Nicolakis, G.F. Wiesinger, M. Baghestanian, E. Preisinger, R. Weinstabl, V. Fialka-Moser, Ultrasound therapy for calcific tendinitis of the shoulder, N. Engl. J. Med. 340 (May (20)) (1999) 1533–1538.
[84] M. Loew, W. Daecke, D. Kusnierczak, M. Rahmanzadeh, V. Ewerbeck, Shock-wave therapy is effective for chronic calcifying tendinitis of the shoulder, J. Bone Joint Surg. Br. 81-B (September (5)) (1999) 863–867.
[85] J. Peters, W. Luboldt, W. Schwarz, V. Jacobi, C. Herzog, T.J. Vogl, Extracorporeal shock wave therapy in calcific tendinitis of the shoulder, Skeletal Radiol. 33 (December (12)) (2004) 712–718.
[86] J.D. Rompe, J. Zoellner, B. Nafe, Shock wave therapy versus conventional surgery in the treatment of calcifying tendinitis of the shoulder, Clin. Orthop. Relat. Res. 387 (June) (2001) 72.
[87] C.-J. Wang, K.D. Yang, F.-S. Wang, H.-H. Chen, J.-W. Wang, Shock wave therapy for calcific tendinitis of the shoulder: a prospective clinical study with two-year follow-up, Am. J. Sports Med. 31 (March (3)) (2003) 425–430.
[88] R. Aina, E. Cardinal, N.J. Bureau, B. Aubin, P. Brassard, Calcific shoulder tendinitis: treatment with modified US-guided fine-needle technique, Radiology 221 (November (2)) (2001) 455–461.
[89] P.B. de Witte, J.W. Selten, A. Navas, J. Nagels, C.P.J. Visser, R.G.H.H. Nelissen, M. Reijnierse, Calcific tendinitis of the rotator cuff: a randomized controlled trial of ultrasound-guided needling and lavage versus subacromial corticosteroids, Am. J. Sports Med. 41 (July (7)) (2013) 1665–1673.
[90] D.L. Gatt, C.P. Charalambous, Ultrasound-guided barbotage for calcific tendonitis of the shoulder: a systematic review including 908 patients, Arthroscopy 30 (Sepetember (9)) (2014) 1166–1172.
[91] R. Seil, H. Litzenburger, D. Kohn, S. Rupp, Arthroscopic treatment of chronically painful calcifying tendinitis of the supraspinatus tendon, Arthroscopy 22 (May (5)) (2006) 521–527.
[92] K. Suzuki, A. Potts, O. Anakwenze, A. Singh, Calcific tendinitis of the rotator cuff: management options, JAAOS – J. Am. Acad. Orthop. Surg. 22 (Nov (11)) (2014) 707.