Calcific periarthritis: A love for the shoulder...yet fondness for other joints too! - a multimodality review

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Learning objectives

The diagnosis of calcific periarthritis and calcium hydroxyapatite crystal deposition (CHCD) is well recognized around the shoulder joint, with the supraspinatus tendon being the most commonly involved site. These features can lead to acute attacks of periarthritis and bursitis. Less well-known, nonetheless significant when it comes to patient symptoms and disability, are similar calcific deposits identified at other multiple joint sites.

This poster highlights the appearances of calcific periarthritis at multiple sites outside the shoulder with the aid of multimodality imaging, and how the condition can be managed, thus helping to reach the correct diagnosis and ensure prompt and appropriate treatment is commenced for the patient.
Background

Periarticular calcific tendinitis is an acute inflammatory reaction and one of a subgroup of calcium hydroxyapatite crystal deposition (CHCD) disease processes.\(^1\) This is commonly seen in the shoulder joint (Fig 1-3) but there are also a significant number of patients that present with CHCD in tendons and soft tissues involving other joints too. The disease process is poorly understood outside the shoulder joint and misdiagnoses have been reported which leads to inappropriate treatment and can adversely affect patient recovery.\(^2\)

Calcium hydroxyapatite is the most abundant form of calcium in the human bone and seen in most pathological calcifications in the body.\(^3\) Many authors classify hydroxyapatite deposition as a crystal deposition disorder, belonging in the same category as gout and calcium pyrophosphate dehydrate (CPPD) deposition disease, hence there now appears to be significant overlap among these crystal deposition diseases.

Aetiology

The aetiology of calcium hydroxyapatite crystal deposition is not clearly understood although a number of theories have been proposed in the past. One such theory suggests calcium deposition takes places in areas of degenerated tendon fibres particularly in the rotator cuff of the shoulder, secondary to abnormal pressure and compression.\(^4\) Other proposals suggest repetitive trauma as a cause for increased CHCD in joints with moderate physiological range of movement.\(^5\)

An initiating event for calcific periarthritis was thought to be hypoxia in a critical area of tendon,\(^6\) followed by fibrocartilaginous metaplasia of the tendon, with a propensity to calcify. Some authors believe metabolic disorders or hereditary factors contribute to CHCD, particularly when evidence has suggested increased calcific deposits in patients with renal disease. So far one has seen a few theories proposed but the fact that so many hypothesizes have been generated suggests that no convincing case for the aetiology of CHCD has been made.

Signs & Symptoms
Calcific periarthritis most commonly affects middle-aged persons and patients can present with acute or chronic symptoms of pain and disability with restriction of movement to the affected joint, fever, localized oedema and elevated levels of acute phase reactants. Outside the shoulder and hip joints, periarticular structures tend to be more superficial and clinical signs of erythema, swelling, and localized tenderness can be prominent and thereby mimic appearances of infection which has been a diagnostic pitfall.

**Misdiagnosis / Differential Diagnosis**

Sometimes patients do not have radiographs taken at clinical presentation particularly in the absence of trauma, but when radiographs have been taken, soft tissue calcification has been misinterpreted as accessory ossicles or avulsion fractures, and sometimes even entirely missed.

The differential diagnosis to consider amongst other causes of calcific periarthritis include tumoral calcinosis, synovial sarcoma, myositis ossificans, and synovial chondromatosis.

**Management**

Calcific periarthritis is generally a self-limiting condition that can spontaneously resolve with time although in the chronic form, symptoms can last anything from 2 to 24 months. Treatment options are tailored to the patient and their symptoms. These include conservative management with rest and use of concurrent oral and/or application of topical anti-inflammatories (NSAIDs); or ultrasonic treatment. Orthopaedic interventional management would include arthroscopic debridement or local surgical excision after appropriate diagnostic confirmation.

Dry needling is a recognized technique that is less invasive than surgical intervention, where under ultrasound guidance, a needle is repeatedly introduced into the periarticular calcific foci in an effort to break up the calcification followed by injection of local steroid and long-acting anaesthetic within the affected soft tissues. This latter approach is commonly used in our practice to good effect with favourable outcomes recorded in our patients who have undergone dry needling under the guidance of the radiologist.
Images for this section:

Fig. 1: XR Right Shoulder - Supraspinatus tendon curvilinear calcification

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Fig. 2: CT Right Shoulder Coronal Reformat - Calcification of supraspinatus tendon

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Fig. 3: Ultrasound Right Shoulder - calcification of supraspinatus tendon (curvilinear echogenicity with posterior acoustic shadowing)

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Imaging findings OR Procedure details

Figures 4 and 5 demonstrate calcification involving the left gluteal medial tendinous insertion over the greater trochanter.

Acute calcific periarthritis can lead to bursitis. Figures 6-8 demonstrate one case where a patient developed trochanteric bursitis secondary to calcium hydroxyapatite crystal deposition (CHCD). This patient was successfully managed by ultrasound-guided dry needling and steroid injection. The patient was pain free 12 months post injection.

Figures 9-11 demonstrate acute periarthritis involving the proximal medial collateral ligament as evident by the low signal rounded calcification seen on the MRI sequences.

Figures 12 and 13 illustrate CHCD in the foot.

CHCD can also involve the wrist and if calcific deposits are seen involving tendon sheaths, this can lead to tenosynovitis as demonstrated in figures 14-17. This patient was managed successfully by ultrasound-guided dry needling of calcification.

As discussed earlier, awareness of calcific periarthritis outside the shoulder joint is less so amongst general clinicians and sometimes misdiagnoses do occur. However important differentials must be entertained and figures 18 and 19 just highlight the importance of remembering to consider alternative diagnoses.
**Fig. 4:** XR Left Hip - Calcification overlying the greater trochanter involving the gluteal medius tendinous insertion

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Fig. 5: XR Pelvis - Left greater trochanteric calcification of the gluteal medial tendon

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**Fig. 6:** MRI Pelvis (Coronal Proton Density Fat Suppression) - Low signal calcification seen around the left greater trochanter with surrounding high signal oedema in adjacent gluteal muscle in keeping with trochanteric bursitis

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**Fig. 7:** MRI Pelvis (Coronal Proton Density Fat Suppression) - Left greater trochanteric low signal rounded calcification

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Fig. 8: USS Left Hip (Transverse Plane) - calcification over the left greater trochanter (curvilinear echogenicity with posterior acoustic shadowing) in a patient with trochanteric bursitis.

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**Fig. 9:** XR Left Knee - calcification superficial to proximal medial collateral ligament (MCL) insertion. The patient also has osteoarthritis with medial compartment predominance.

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Fig. 10: MRI Left Knee (Coronal Proton Density Fat Suppression) - Low signal calcification superficial to proximal insertion of MCL

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Fig. 11: MRI Left Knee Axial Proton Density Fat Suppression - low signal calcification superficial to proximal insertion of MCL

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Fig. 12: XR Right Foot - calcification in the medial aspect of the base of the 2nd proximal phalanx

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**Fig. 13:** XR Right Foot - calcification in the medial aspect of the base of the 2nd proximal phalanx

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**Fig. 14:** XR Wrist - calcification distal to the tip of the ulna styloid process (Extensor Carpi Ulnaris (ECU) tendon sheath)

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Fig. 15: USS Wrist (Transverse Place) - ECU tendon sheath calcification (arrows) with increased activity seen on Colour Doppler.

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**Fig. 16:** MRI Wrist (Coronal T2-weighted) - foci of calcification seen within the ECU tendon sheath (arrows)

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Fig. 17: MRI Wrist (Axial STIR) - foci of calcification seen within the ECU tendon sheath with tenosynovitis (surrounding high signal change).

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Fig. 18: XR Fingers - Differential Diagnosis of calcification. This patient has a recurrence of a giant cell tumour overlying the distal interphalangeal joint (DIP) of the middle finger.

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Fig. 19: Differential Diagnosis of calcification - This patient has chronic heterotopic ossification following a central slip injury to the left ring finger.

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Conclusion

This poster has illustrated a select few examples of tendinous and periarticular calcification deposits in joints less commonly seen than in the typical locations like the shoulder. Acute calcific periarthritis outside the shoulder is less well recognized by clinicians and can therefore lead to a misdiagnosis. The ability to recognize that these periarticular amorphous calcifications at or adjacent to tendinous insertion sites are due to hydroxyapatite crystal deposition, will lead to the appropriate diagnosis being made and allow suitable and prompt patient treatment to be instigated.
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References


