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CASE REPORT

Calcium hydroxyapatite crystal deposition with intraosseous penetration involving the posterior aspect of the cervical spine: a previously unreported cause of neck pain

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Abstract
Purpose Calcific tendinitis is a frequent disorder caused by hydroxyapatite crystal deposition; however, bone erosions from calcific tendinitis are unusual. The spinal manifestation of this disease is calcific tendinitis of the longus colli muscle; this disease has never been described in the posterior aspect of the spine. We report a case of calcium hydroxyapatite crystal deposition involving the posterior cervical spine eroding the bone cortex.
Methods A 57-year-old woman presented with a 5-month history of left-sided neck pain. Radiographs showed C4–C5 interspinous calcification with lytic compromise of the posterior arch of C4. Magnetic resonance imaging confirmed a lytic lesion of the posterior arch of C4, with a soft tissue mass extending to the C4–C5 interspinous space; calcifications were observed as very low signal intensity areas on T1 and T2 sequences, surrounded by gadolinium-enhanced soft tissues. A computed tomography (CT) scan confirmed the bone erosions and the soft tissue calcifications.
Results A CT-guided needle biopsy was performed; it showed vascularized connective tissue with inflammatory histiocytic infiltration and multinucleated giant cells; Alizarin Red stain confirmed the presence of hydroxyapatite crystals. The patient was treated with anti-inflammatories for 2 weeks. She has been asymptomatic in a 6-month follow-up; a CT scan at the last follow-up revealed reparative remodeling of bone erosions.
Conclusion This is the first report of calcium hydroxyapatite crystal deposition with intraosseous penetration involving the posterior aspect of the cervical spine. Considering that this unusual lesion can be misinterpreted as a tumor or infection, high suspicion is required to avoid unnecessary surgical procedures.

Keywords Calcific tendinitis · Cervical spine · Hydroxyapatite · Crystal deposition disease · Neck pain

Introduction

Calcific tendinitis is a common condition that occasionally causes acute pain when calcifications dissolve and compromise adjacent soft tissues. This condition is caused by hydroxyapatite crystal deposition; these deposits are frequently asymptomatic, but they may sometimes be associated with pain of variable intensity, acute painful attacks, erythema, swelling, and even low-grade fever [1, 2]. It usually affects patients 40–70 years old and is slightly more frequent in women. This disease most commonly involves the shoulders, hips, elbows, wrists, or knees; spinal involvement has been reported, but is uncommon [1, 3, 4].

Even though calcific tendinitis itself is a frequent disorder, intraosseous calcium penetration from calcific tendinitis is very unusual. This condition was first described by Hayes et al. [4]; these cases may represent a diagnostic challenge if the underlying cortex is eroded or if marrow changes are observed in computed tomography (CT) scans or magnetic resonance imaging (MRI) because they may be confused with a neoplasm. Since publication of the report

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from Hayes et al., further publications have mostly included case reports and small case series of intraosseous involvement. Only two large series discussing calcific tendinitis with intraosseous involvement have been reported. In one series, only 1 out of 50 patients exhibited spinal involvement [1]. The other series, published recently, also described only 1 case with spinal compromise among 35 patients [5].

The spinal manifestation of calcium hydroxyapatite crystal deposition described in the literature is acute calcific tendinitis of the longus colli muscle. This well-characterized condition occurs in the cervical prevertebral space, most frequently at the C1–C3 vertebral level. However, more caudal locations have been described [6, 7]. Calcific tendinitis of the longus colli muscle is frequently misdiagnosed as a retropharyngeal abscess, which sometimes results in unnecessary surgical procedures. To the best of our knowledge, no cases of posterior (dorsal) cervical spine calcium hydroxyapatite crystal deposition with bone involvement have been described in the literature. In this report, we describe a patient with calcium hydroxyapatite crystal deposition in the posterior aspect of the cervical spine that eroded the bone cortex, thus mimicking a vertebral tumor.

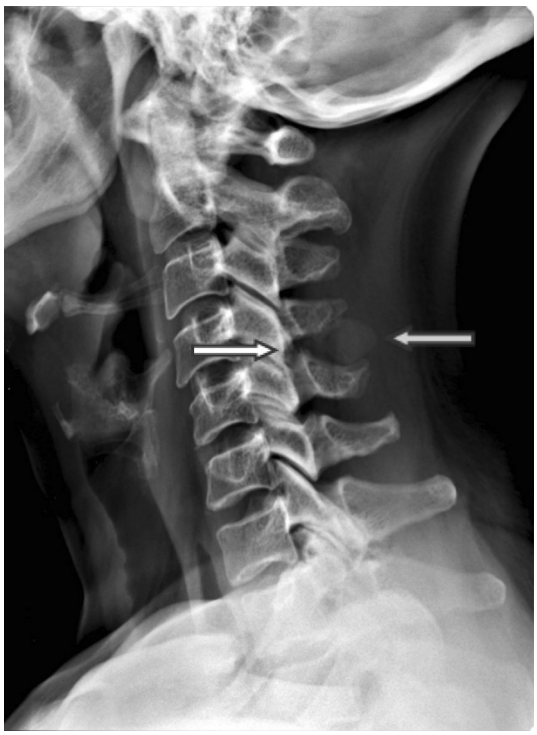

Fig. 1 Plain radiograph showing C4–C5 interspinous calcification and a lytic lesion in the posterior arch of C4 (*arrow*)

Case report

Institutional review board approval was obtained to report this case.

An otherwise healthy 57-year-old female patient presented with a 5-month history of left-sided neck and scapular pain. The pain did not radiate to the arms, and she had no neurological symptoms in her extremities. The pain had no clear onset, and she had temporary pain relief when she used anti-inflammatory medications. On physical examination, the patient had pain during palpation of the cervical spine and paraspinal musculature. Additionally, cervical spine extension and lateral flexion were limited. Neurological examination of the four extremities was normal.

Plain radiographs showed a C4–C5 interspinous calcification and a lytic lesion in the posterior arch of C4 (Fig. 1). The MRI of the cervical spine confirmed a lytic lesion in the posterior arch of C4, with bone marrow changes in the posterior arch of C4. Additionally, the MRI revealed a soft tissue mass dorsal to the lytic lesion, extending to the C4–C5 interspinous space. Notably, calcifications were observed as areas of very low signal intensity on both T1 and T2 sequences, surrounded by areas of gadolinium enhancement in the surrounding soft tissues (Fig. 2). A bone scan was obtained, which showed intense focal tracer uptake on the left side of the dorsal aspect of C4. The scan ruled out abnormal uptake in other skeletal locations.

The patient underwent a CT-guided needle biopsy of the cervical spine lesion. The CT scan confirmed the bone erosions as well as the multiple calcifications within the soft tissue mass; additionally, severe erosion and degeneration of the left C4–C5 facet joint was observed (Fig. 3). Of note, the patient had no previous procedures involving such degenerated joint. A needle aspiration was first performed and a cloudy fluid was collected, which was sent for analysis; additionally, a bone sample was obtained. The birefringence analysis of the aspirate did not show urate or calcium pyrophosphate crystals. The pathology study, performed by an experienced orthopedic pathologist, showed vascularized connective tissue with inflammatory histiocytic infiltration. This tissue was surrounded by multinucleated giant cells, suggesting a foreign body reaction. Considering that radiologists had postulated that the hydroxyapatite crystal deposit with intraosseous involvement was part of the differential diagnosis, an Alizarin Red stain was performed [8]. The staining confirmed the presence of hydroxyapatite crystals.

The patient was treated only with anti-inflammatory medications for 2 weeks, and she was asymptomatic at her 6-month follow-up. A new CT scan obtained 6 months after the rest of the study showed reparative remodeling of

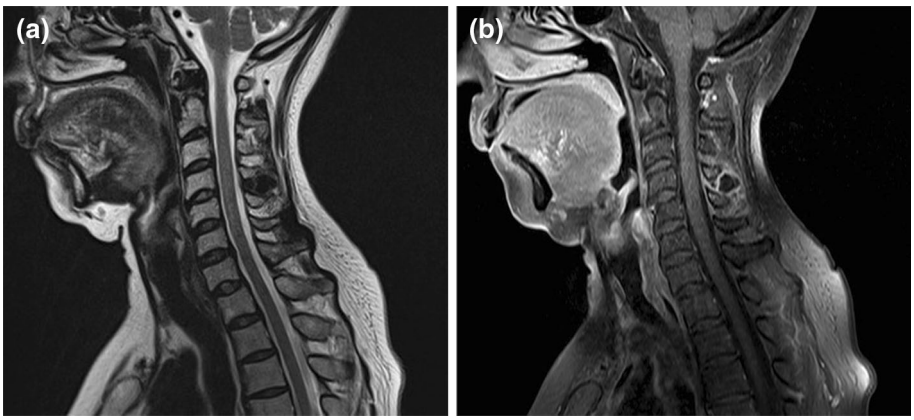

Fig. 2 **a** Sagittal T2 MRI image showing C4–C5 interspinous calcium deposit as an area of low signal intensity; **b** sagittal gadolinium-enhanced T1 MRI image showing interspinous calcium deposit (area of low signal intensity) and surrounding soft tissue enhancement

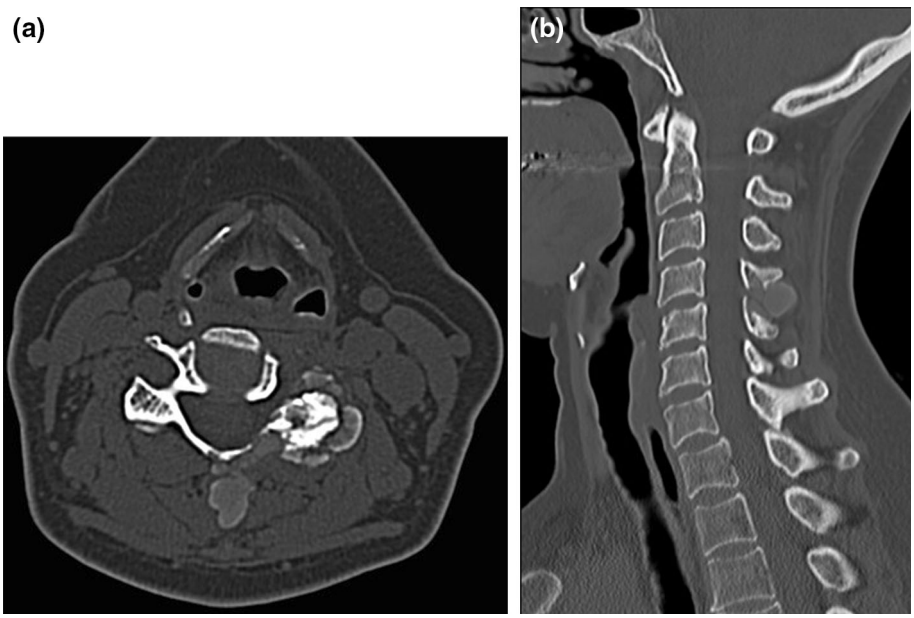

Fig. 3 **a** Axial and **b** sagittal CT scan showing laminar erosion and soft tissue calcifications

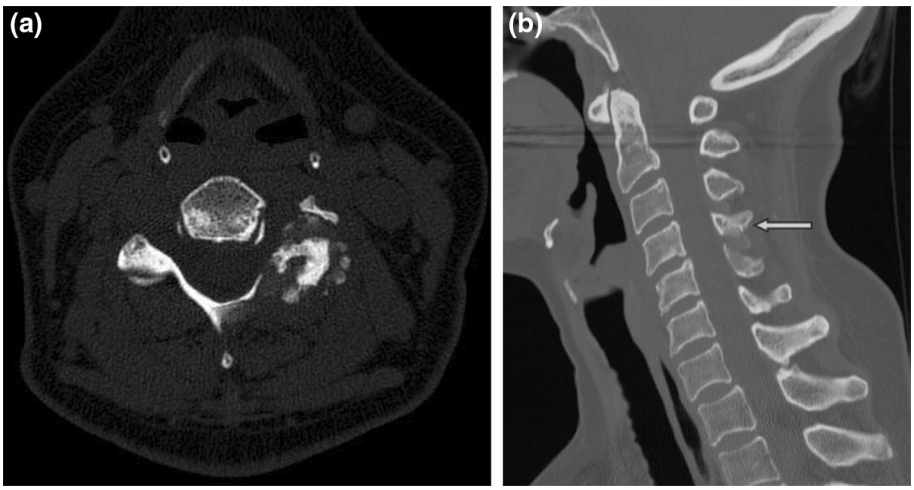

Fig. 4 **a** Axial and **b** sagittal CT scan exhibiting reparative remodeling of the posterior arch of C4

the posterior arch of C4, with decrease in the size of soft tissue calcification (Fig. 4a, b); however, the left C4–C5 facet joint degeneration did not change in the follow-up (Fig. 4a).

Discussion

To the best of our knowledge, this is the first report of calcium hydroxyapatite crystal deposition with intraosseous penetration involving the posterior aspect of the cervical spine. The intraosseous compromise, while similar to those described in other anatomic locations, could be confused with an aggressive neoplasm. Therefore, a thorough process is needed to obtain the correct diagnosis.

Stable calcium hydroxyapatite crystal deposits can appear as solid, stippled or amorphous zones of increased density in plain radiographs. They lack the cortical or trabecular structure that is observed in heterotopic ossification or accessory ossicles [4]. However, as the crystals dissolve, their density diminishes and their volume increases, their contours become ill defined and they present a "snowflake" or "fluffy" aspect [5], similar to that seen in our patient (Fig. 1). The disease is easy to diagnose on plain radiographs when soft tissue calcifications are found in typical locations (such as the shoulder). However, it may be confused with an infection or an aggressive neoplastic lesion if the adjacent bone is involved [9]. Therefore, clinical suspicion of this disease is essential for correct diagnosis.

CT scans are particularly valuable to establish the diagnosis of calcium hydroxyapatite crystal deposits when calcifications are not dense enough to be visible on plain radiographs [5]. Moreover, cortical erosions are easier to observe on CT scans than on plain radiographs or MRI [1]. Accordingly, we believe that a CT scan is particularly helpful to establish the diagnosis of calcium hydroxyapatite crystal deposition in the posterior aspect of the cervical spine, as it is essential in the diagnosis of acute calcific tendinitis of the longus colli muscle [6, 7].

MRI may show bone marrow involvement and significant edema in neighboring soft tissues. The calcium deposits may become visible as areas of very low signal intensity on both T1 and T2 sequences [1, 3]. However, because the calcifications can be hard to visualize on MRI [1, 10], the erosive lesions associated with such soft tissue and bone marrow edema may lead to misdiagnosis as an infection or a bone tumor [9]. This is important, as MRI is often used as the first imaging modality to assess patients with suspected neoplasms and is even used to evaluate patients with spinal pain. Accordingly, a thorough diagnosis should combine the different imaging modalities.

Bone scans are frequently requested when a neoplasm is suspected. It has been described that patients having calcific tendinitis with osseous involvement almost always exhibit focal increased radionuclide uptake during bone scanning [1], as our patient did.

It has been hypothesized that the periosteal reaction observed in these cases could be secondary to the local inflammatory response produced by the deposition of calcium hydroxyapatite crystals within the tendinous insertion. The periosteal reaction may be confused with an aggressive neoplasm rather than a manifestation of calcific tendinitis. The biopsy in our patient showed the scattered fibrovascular tissue calcification with associated histiocytes and macrophages, characteristic of acute-phase calcific tendinitis [1, 11]. Of note, we confirmed the presence of hydroxyapatite crystals using Alizarin Red staining [8]. Without staining, pathological findings cannot be differentiated from other conditions such as tumoral calcinosis of the spine [12]. It is noteworthy that even the largest series studying patients presenting calcific tendinitis with osseous involvement did not include special staining to confirm the presence of hydroxyapatite crystals.

Most patients with calcific tendinitis of the appendicular skeleton (as well as in acute calcific tendinitis of the longus colli muscle) only require non-steroidal anti-inflammatory drugs as treatment, because the disease is self-limiting and usually resolves spontaneously. This was the case in our patient, who was pain free in less than a week after using anti-inflammatory medications. A CT scan obtained 6 months after starting treatment showed reparative remodeling of bone erosions.

Calcium hydroxyapatite crystal deposition with intraosseous penetration has been described in several skeletal locations. However, no spinal locations have been described previously, other than the anterior cervical spine involvement [1, 5–7]. Because this unusual lesion can be misinterpreted as a bone tumor or infection, high suspicion is required on the part of radiologists and clinicians for the correct diagnosis and avoidance of unnecessary surgical procedures. We believe this report will help radiologists and clinicians recognize this unusual disease in the future.

Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

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