Calcifying nucleopathy mimicking infectious spondylodiscitis

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ABSTRACT

Spinal hydroxyapatite deposition disease is an uncommon condition. It can be misdiagnosed. Clinical signs may be remarkable mimicking infectious spondylodiscitis.

We report a case of a 53-year-old man with acute febrile inflammatory back pain. Magnetic ressonance imaging (MRI) showed spondylodiscitis of T12-L1 intervertebral disc without abscesses. Spine radiography revealed a calcifying nucleopathy with a complete disappearance of this calcification during the follow-up.

The diagnosis of hydroxyapatite deposition disease should be considered in patients with acute inflammatory back pain. We highlight the importance of the relevance of imaging features in making the diagnosis. A total disappearance of the calcification is possible during the follow-up.

Keywords: Calcific nucleopathy; Apatite rheumatism; Spine; Spondylodiscitis.

INTRODUCTION

Hydroxyapatite deposition disease (HADD) also known as hydroxyapatite rheumatism is a condition characterized by periarticular deposition of basic calcium phosphate (BCP) crystals. Spinal involvement is scarce. It can be responsible for calcifying nucleopathy (CN)[1]. Symptomatic disc calcification occurring in the spine can be misdiagnosed. Clinical signs may be remarkable mimicking infectious spondylodiscitis. We report a case of acute symptomatic disc calcification in an adult. We emphasize the clinical and radiological features with a complete resolution in the follow-up.

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CASE REPORT

A 53-year-old man, with no relevant past medical history, presented with a two-day history of intense inflammatory back pain. The onset of pain was sudden and nocturnal. There was no report of recent trauma or falls. He developed a single peak of fever (39°C). Physical examination revealed tenderness over the area of the spinal process of T12, restricted back movement (Schöber test was at 2cm). The neurological examination was unremarkable. Blood cell counts, C-reactive protein level and erythrocyte sedimentation rate were normal. Serum levels of calcium, albumin, and phosphorus were within the normal range. Liver tests and renal function were unremarkable. Spine MRI showed hypointense T1 and hyperintense T2-weighted signal in inferior T12 and superior L1 vertebral endplate and the T12-L1 disc. No abscesses were observed (Figure 1).

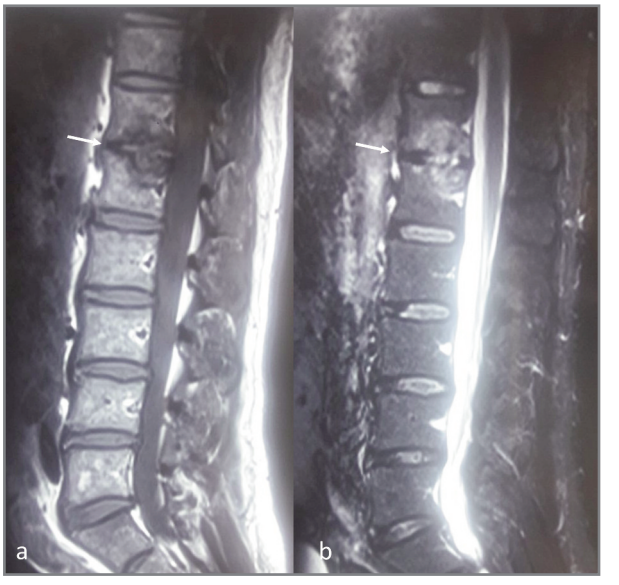

FIGURE 1. Spine MRI showing low T1-weighted images (a) and high signal in T2-weighted images (b) in inferior T12 and superior L1 vertebral endplate and in the T12-L1 disc (arrow)

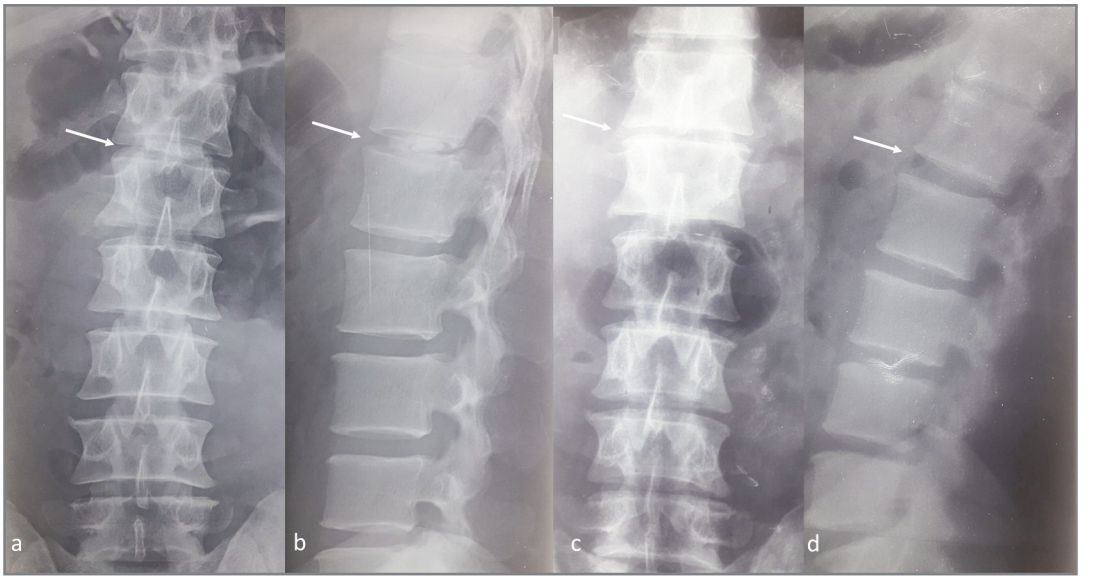

FIGURE 2. Spine radiography showing well-defined central calcification of T12-L1 disc (a, b). And a complete disappearance of the calcifying nucleopathy after one-year (c, d)

The radiography revealed calcifying nucleopathy of the T12-L1 disc (Figure 2 a, b). Radiography of the shoulders, wrists, and knees did not show calcifications. The diagnosis of disc calcification due to HADD was established. A non-steroidal anti-inflammatory treatment based on a 200-mg daily dose of ketoprofen was prescribed for 10 days leading to the improvement of symptoms within two days. After one year of follow-up, the radiography revealed complete disappearance of the calcification (Figure 2 c, d). The spine computed tomography showed the fragmented calcification being resorbed associated with vertebral endplate erosions (Figure 3).

DISCUSSION

Hydroxyapatite deposition disease involves typically the appendicular skeleton. The shoulder is the most affected joint (60% of cases)[2]. Disc calcification is uncommon. It occurs usually in the inferior thoracic discs and dorsolumbar junction in adults and the lower cervical spine in children.[2] The BCP crystals may deposit in several anatomic structures such as interspinous bursae, apophyseal joints, and intervertebral discs.

The deposition of BCP crystals undergoes three stages: precalcific, calcific, and postcalcific stage[3]. In the precalcific stage, no calcification is visible whereas histological finding shows fibrocartilaginous metaplasia. The calcific stage has a three-phase evolution: formative, resting and resorptive phase. The two first phases may be asymptomatic or induce minor symptoms. Disc calcification can be due to HADD or calcium pyrophosphate dihydrate crystals (CPPD) deposition. BCP crystals deposits in the central part of the disc (calcifying nucleopathy (CN)). However, CPPD deposit in the annulus fibrosus[4].

The resorptive phase is due to the acute inflammatory response resulting from the migration of BCP crystals to adjacent tissues. CN can migrate with a herniated disc. Then, this calcified disc herniation can migrate to the endplate of adjacent vertebra[1,5-7]. It can also migrate in the spinal canal, the intervertebral foramen[8-13] or front of the intervertebral space[14] or adjacent costovertebral joint[15].

The resorptive phase is characterized by intense symptoms including pain, stiffness, paravertebral muscular contracture, and limitation of motion of the spinal segment. Symptoms may also include, depending on the site of migration, radiculopathy, and acute paraplegia due to spinal cord compression[13].

Fever occurs usually in children. However, it's uncommon in adults.[1] In our case, the patient developed a single peak of fever. However, a normal CRP level made the diagnosis of infectious spondylodiscitis very unlikely. Raised inflammatory markers may be ob-

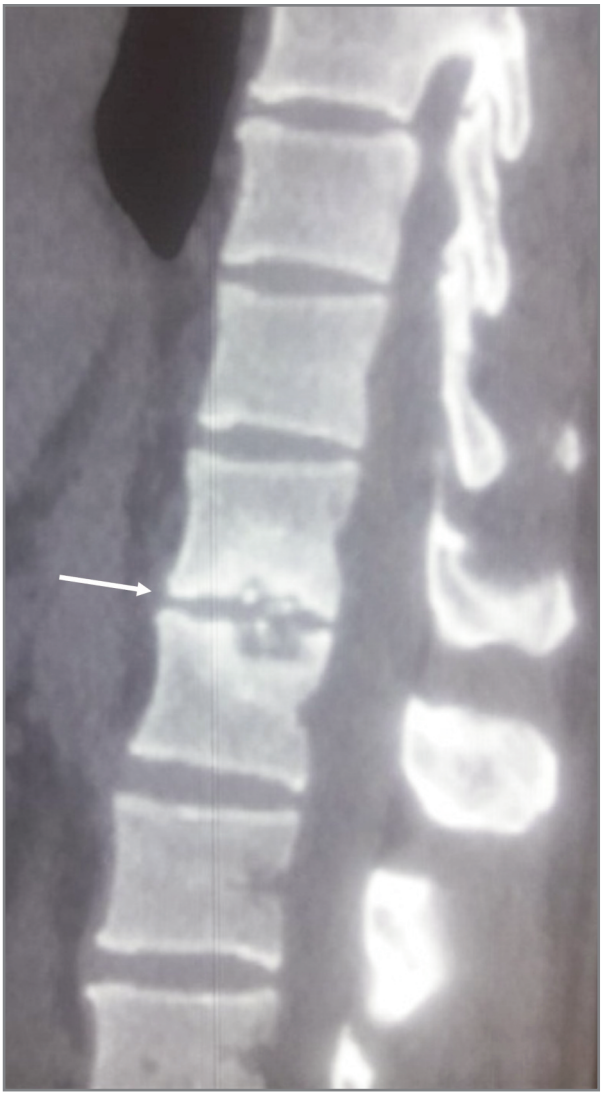

FIGURE 3. CT-scan of the dorso-lumbar junction showing residual fragmented hydroxyapatite crystals associated with vertebral endplate erosions, undetectable on radiography

served in this phase and can mimic infectious spondylodiscitis. This phase usually subsides within a few days. Sadok *et al.*[14] reported only 9 cases of acute symptomatic form of CN. Computed tomography should be considered in case of migration of the calcification. As in our case, it can show a migration of calcification to the endplate of adjacent vertebra[6] resulting in vertebral endplate erosion.

In MRI, crystals appear in hyposignal T1 and T2. MRI may detect inflammation explained by the resorption of calcifications. However, the non-visualization of calcification within inflammatory phenomena affecting the disc and adjacent endplates, including hypointense signal in T1-weighted and hyperintense signal in T2-weighted images, can induce errors in diagnosis.

The postcalcific stage is characterized by the improvement of the inflammatory symptoms. The spine radiography can reveal a complete disappearance of the calcification. To our knowledge, the complete disappearance of the spinal calcification in plain radiography has been reported in only three cases[10,14,15]. In other cases, only partial resolution of the calcification was observed[5-7,9]. Hence, it is essential to perform radiography during the resorptive phase and the follow-up.

HADD is a self-limiting process and spontaneous remission is usually obtained. However, the intensity of symptoms often requires the use of treatment such as non-steroidal anti-inflammatory drugs. The effect of colchicine is not defined. Physiotherapy and immobilization of the affected spine segment can be prescribed. Surgical treatment is indicated in case of neurological complications.

CONCLUSIONS

The diagnosis of HADD occurring in the spine is sometimes difficult. It can be misdiagnosed. It should be considered in patients with acute inflammatory back pain. Clinical signs may be remarkable mimicking infectious spondylodiscitis. We highlight the importance of the relevance of imaging features in making the diagnosis. A complete resolution and total disappearance of the CN is possible during the follow-up. We suggest that a new radiography is mandatory during the resorptive phase.

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