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Calcium Pyrophosphate Dihydrate Crystal Deposition Disease of the Cervical Spine Accompanied by Acute Vertebral Body Collapse and the Crowned Dens Syndrome

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Introduction

Calcium pyrophosphate dihydrate crystal deposition (CPPD) disease usually affects the peripheral joints in elderly patients. Albeit less frequently, it also occurs in and around the vertebral structures, including the intervertebral discs, joint capsules, synovium, articular cartilage, bursae, and ligaments1. The crowned dens syndrome (CDS) is a rare form of crystal disposition disorder characterized by acute and recurring neck pain due to the deposition of hydroxyapatite or calcium pyrophosphate dihydrate around the odontoid process. Herein, we describe a case of CPPD disease with the CDS, which developed unusually severe destructive changes in the cervical spine.

Case Report

A 42-year-old Japanese male presented with neck pain, bilateral shoulder pain, and left finger numbness. Past medical history was not contributory.

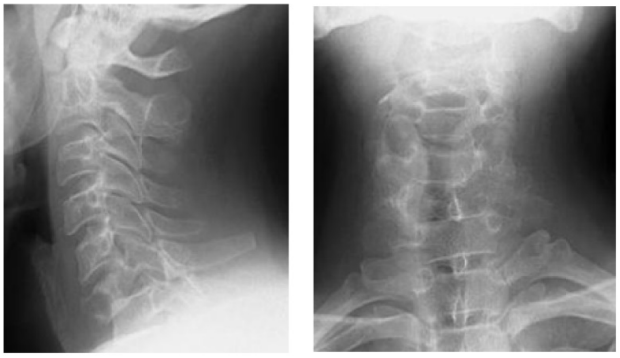

Figure 1: X-P ray images of the cervical spine at the initial visit.

Cervical extension and lateral flexion were significantly limited. Although there was no motor disturbance, the patient had a slight sensory disturbance in the left fingers. Laboratory data showed no abnormalities. X-rays taken at the initial visit showed osteolytic destructive changes in the left side of the C6 vertebral body (Figure 1).

Computed tomography (CT) images demonstrated a collapse of the C6 vertebral body and marked destructive changes in the C6-7 facet joint. Calcific deposition around the odontoid process, which is characteristic to the CDS, was also noted (Figure 2).

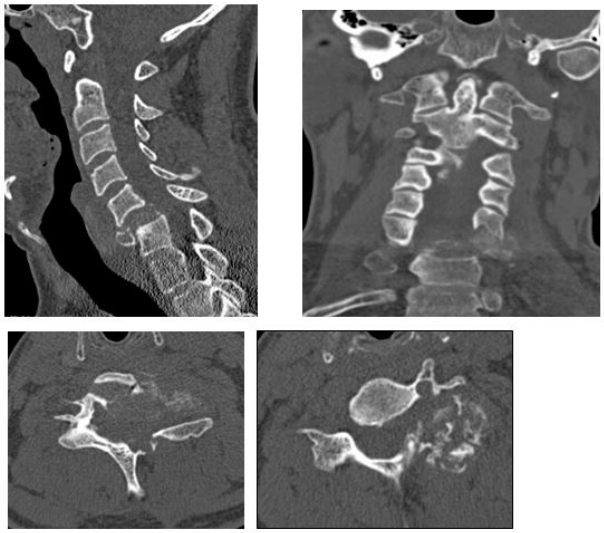

Figure 2: CT images of the cervical spine at the initial visit.

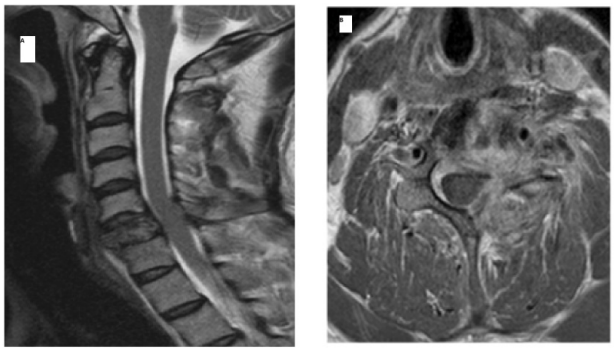

Figure 3: MRIs of the cervical spine at the initial visit; A: T2-weighted iammage; B: T1-weighted image with Ggadolinium contrast enhancement on T1.

Magnetic resonance imagings (MRIs) showed a collapse of the C6 vertebral body and disappearance of the C6/7 disc. Soft tissue

surrounding the C6 vertebra showed heterogeneous intensity on T2-weighted images and low intensity on T1-weighted images, suggestive of edema and inflammation (Figure 3).

Open biopsy was performed from the C6-7 facet joint under general anesthesia. Pathological diagnosis of the biopsy specimens was compatible with crystal deposition disease without any findings indicative of malignancy (Figure 4).

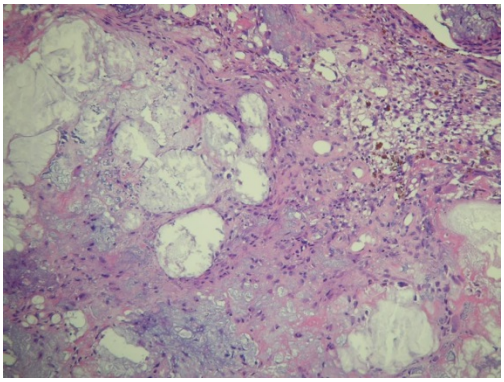

Figure 4: A section of the biopsy specimen Microscopicstained with hematoxylin and eosin finding (HE).

The patient was conservatively treated with non-steroidal anti-inflammatory drugs and his symptoms had temporally relieved. However, the deformity and immobility of the neck became significantly worsened afterwards. Clinical images taken 3 months after the initial diagnosis showed that the bone destruction at C6 level had progressively aggravated, resulting in severe lateral tilt of the cervical spine (Figure 5).

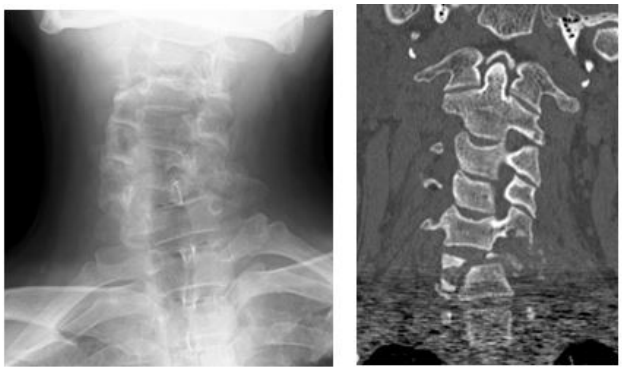

Figure 5: X-ray and CT images of the cervical spine taken P and CT after 3 months after the initial visit.

Subsequently, anterior cervical plate fusion was performed to correct and stabilize the cervical spine. Three years after surgery, the patient is currently well without any symptoms or pain. Clinical images showed remodeling of the vertebral body and the facet joint (Figure 6).

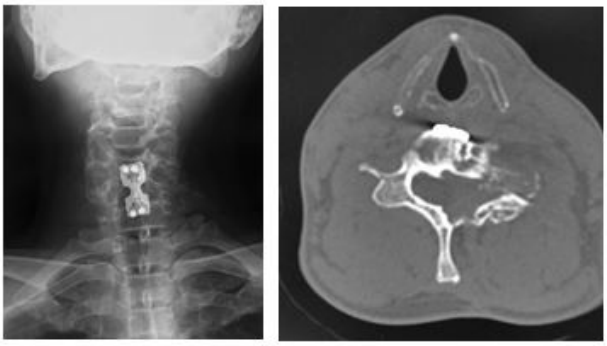

Figure 6: X-rays and P and CT images post. Op. 3 years after surgery.

To the best of our knowledge, the present report may describe the first case of CPPD disease which was accompanied by cervical spine destruction and the CDS. The CDS is related not only to CPPD disease but also to hydroxyapatite crystal deposition disease (HADD) [1,2]. Clinically, it is not feasible to make a differential diagnosis between HADD and CPPD disease based solely on the clinical images. Joint cartilage calcification is a hallmark of CPPD diseases, and peripheral X-ray examinations are of help in identifying this condition. Nevertheless, a definite diagnosis of CPPD disease can be established by examining the joint fluid for the presence of calcium pyrophosphate crystals. On the other hand, HADD in the spine is characterized by calcific tendinitis of the longus colli and discal calcinosis1. In addition, hydroxyapatite calcifications are usually dense and amorphous in appearance, and the crystal deposition often disappear within a few weeks [3]. In our case, although we could not prove the presence of calcium pyrophosphate crystals in the biopsy specimens, clinical images and histological findings were compatible with CPPD disease. Furthermore, clinical presentations (such as the acute destructive changes in the affected vertebrae) and a relatively long clinical course favor the diagnosis of CPPD disease over HADD. Based on the above information and findings, we made the final diagnosis of CPPD disease.

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