Case Report

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Pathology Section

Lumbar Spine Pseudogout Mimicking Disc Prolapse and Radiculopathy: A Rare Case Report

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ABSTRACT

Calcium Pyrophosphate Dihydrate (CPPD) deposition disease, also known as pseudogout, is a crystal-induced arthropathy that primarily affects larger, weight-bearing joints such as the knees, hips, and shoulders. CPPD can present with a wide spectrum of clinical manifestations, ranging from asymptomatic chondrocalcinosis to acute inflammatory arthritis. CPPD in the spine is rare and can lead to calcification of the ligamentum flavum, potentially causing spinal cord compression, myelopathy, and significant neurological impairment. The absence of chondrocalcinosis on imaging does not rule out the diagnosis of spinal CPPD. On microscopy, CPPD is characterised by rhomboid-shaped, positively birefringent crystals, in contrast to the needle-shaped, negatively birefringent crystals observed in gout. The present case report of a 78-year-old female highlights a rare and isolated clinical presentation of CPPD causing lumbar spine myelopathy, which was discovered incidentally on histopathology after spinal decompression and discectomy surgery. Although imaging modalities may raise suspicion for CPPD by revealing chondrocalcinosis, they often lack the specificity to differentiate it from other degenerative or inflammatory conditions. Histopathological analysis and polarised microscopic examination remain the cornerstones in diagnosing CPPD, even in atypical clinical presentations or ambiguous imaging findings. This underscores the importance of including pseudogout in the differential diagnosis of lumbar myelopathy, particularly in elderly patients, to provide early, targeted treatment and prevent severe irreversible neurological deficits.

Keywords: Calcium pyrophosphate, Lower back pain, Lumbar myelopathy, Serum calcium

CASE REPORT

A 78-year-old female patient presented to the Orthopaedic Outpatient Department with complaints of chronic, progressively severe lower back pain radiating to the left lower limb, accompanied by tingling and numbness for six months. There was no history of trauma, past illnesses, or known co-morbidities.

Her physical examination revealed a limited range of motion in the spine and tenderness along the left side of the back. A neurological examination indicated weakness, hyperreflexia, and reduced sensation in the left lower extremity. The laboratory workup showed mild anemia (haemoglobin 9.2 gm/dL) with a normal total leukocyte count (5600/mm^3) and platelet count (210,000/mm^3). Serum Thyroid-Stimulating Hormone (TSH) (4.10 mIU/L) and serum calcium (8.6 mg/dL) were within normal limits.

Magnetic Resonance Imaging (MRI) of the whole spine detected a posterior disc bulge with osteophyte complexes from C3-C4 to C5-C6, causing anterior thecal sac indentation, as well as circumferential disc bulges with posterocentral protrusions at L4-L5 and L5-S1. There was compression of the cauda equina nerve roots, bilateral severe lateral recess narrowing, compression of bilateral exiting nerve roots, and severe spinal narrowing at the L4-L5 spinal level [Table/Fig-1a]. The radiological findings suggested features of disc prolapse, leading to a diagnosis of a prolapsed intervertebral disc at L4-L5 with associated left lower limb myeloradiculopathy.

Therapeutic management required immediate surgical intervention, which consisted of Unilateral Biportal Endoscopic (UBE) decompression and discectomy at the L4-L5 spine. Postoperatively, the spinal tissue was sent for routine histopathological evaluation.

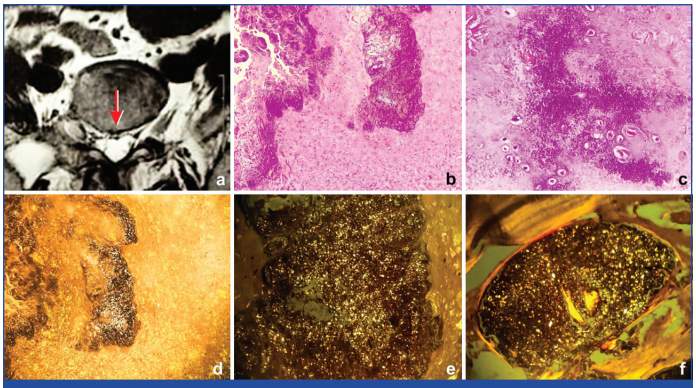
On gross examination: Multiple, tiny, greyish-white, soft to firm tissue fragments were observed, with the largest measuring 1.5×0.8×0.5 cm and the smallest measuring 0.3 cm in dimension. Microscopic examination at low magnification showed fibrocollagenous tissue and disc material, including nucleus pulposus and annulus fibrosus, exhibiting focal deposits of bluish to grey-brown material [Table/Fig-1b]. High magnification revealed aggregates of basophilic to grey-brown crystalline material within the tissue [Table/Fig-1c]. The surrounding tissue displayed chronic inflammatory and histiocytic reactions, with no amyloid-like deposits, granulomatous reactions, or giant cells.

Histopathological differentials considered included crystal deposition diseases like gout, CPPD, and fungal infections. Isolated fungal hyphae can be missed on routine staining or appear as empty spaces, and aggregates of hyphae can also present as bluish deposits. Special stains, Periodic Acid-Schiff (PAS) and Grocott-Gomori Methenamine Silver (GMS), were performed and yielded negative results, ruling out fungal etiology. Additionally, polarising microscopy was conducted to confirm and differentiate the type of crystal deposition disease.

Polarising microscopy revealed multiple rhomboid-shaped crystals showing positive birefringence, confirming the presence of calcium pyrophosphate dihydrate crystals [Table/Fig-1d-f], which differ from those of gout. Based on the clinico-radiological, histopathological, and polarising microscopy findings, a diagnosis of CPPD or pseudogout at the L4-L5 spinal level was established. Soon after decompressive surgery, the neurological deficits diminished. However, the patient was lost to follow-up and could not be evaluated for other inflammatory and serum markers associated with primary disease etiology or recurrence.

DISCUSSION

CPPD or pseudogout is a crystal-induced arthropathy that primarily affects larger, weight-bearing joints such as the knees, hips, and shoulders [1]. CPPD can present with a wide spectrum of clinical manifestations, ranging from asymptomatic chondrocalcinosis to acute inflammatory arthritis, often mimicking conditions such as gout or rheumatoid arthritis. Although CPPD most commonly affects peripheral joints, involvement of the spine is unusual and clinically



[Table/Fig-1]: a) MRI axial section of lumbar spine at L4-L5 showing circumferntial disc bulges and compression of cauda equine nerve roots, bilateral exiting nerve roots with severe bilateral lateral and spinal recess narrowing at suggestive of prolapsed intervertebral disc at L4-L5 spinal level; b) Photomicrograph of histopathology {Haematoxylin and Eosin (H&E), 100x} showing basophilic to bluish-grey crystalline material representing CPPD deposits in a background of lumbar disc with round to oval chondrocytes; c) Photomicrograph of histopathology (H&E, x400) showing basophilic to bluish-grey crystalline material representing CPPD deposits in a background of lumbar disc with round to oval chondrocytes; d) Photomicrograph under polarisedlight microscopy (100x) showing rhomboid-shaped, birefrengent crystals confirming CPPD deposits; e) Photomicrograph under polarised light microscopy (x400) showing rhomboid-shaped, birefrengent crystals confirming CPPD deposits; f) Photomicrograph under polarised light microscopy (x400) showing rhomboid-shaped, birefrengent crystals confirming CPPD deposits.

significant. CPPD deposits in the spine can lead to calcification of the ligamentum flavum, resulting in spinal cord compression and myelopathy [2].

The present case showcases an elderly woman with an unusual location of CPPD in the lumbar spine. Pseudogout in this case manifested clinically as severe lower back pain and neurological symptoms of tingling and numbness in the left lower limb. These findings were consistent with other reported cases of spinal CPPD by I Kh Almadhoun MK et al., Chen SL et al., Kobayashi T et al., and Wilkinson BM et al., who encountered patients with similar ages, genders, and clinical symptoms. In their research, the site for spinal CPPD was the cervical spine, whereas in our case, it was the L4-L5 lumbar spine. Except for the instance reported by Wilkinson BM et al., all cases demonstrated neurological signs [Table/Fig-2] [3-6].

While the underlying mechanism behind spinal CPPD is idiopathic, metabolic dysfunctions and genetic predispositions are contributing factors. Risk factors for CPPD include aging, osteoarthritis, metabolic disorders, and hereditary conditions such as hyperparathyroidism, hypomagnesaemia, and hypophosphatasia [2].

Imaging is essential for diagnosis, with Computerised Tomography (CT) scans being sensitive for detecting calcific deposits and chondrocalcinosis. MRI is preferred for its optimal soft-tissue contrast and ability to assess neural compression in patients with myelopathy [7,8]. CPPD nodules typically present as areas of low signal intensity on T1-and T2-weighted images, surrounded by high-intensity and medium-intensity signals indicative of edematous changes [9].

In the present case, the diagnosis of pseudogout was incidentally confirmed through pathological examination, as imaging showed no evidence of CPPD and revealed features of disc prolapse only. Similar rare cases have been reported in the literature, such as the case by Chen SL et al., involving C3-C5 cervical spine CPPD, where MRI findings suggested ossification of the posterior longitudinal ligament. Another case by Wilkinson BM et al., involved C5-C6 cervical spine CPPD, where the CT scan showed C5-C6 disc prolapse with endplate destruction and features of osteomyelitis or discitis [4,6]. In both cases, imaging alone did not provide conclusive evidence of CPPD deposits, which were later demonstrated on pathological examination. This suggests that pseudogout can occur without radiological evidence of chondrocalcinosis.

Histopathology and polarised microscopy in this case confirmed the diagnosis of CPPD by demonstrating rhomboid-shaped, positively birefringent crystals, thereby distinguishing pseudogout from other potential differential diagnoses. This finding was consistent with all other cases where spinal CPPD was diagnosed [3-6]. On histopathology and polarised microscopy, CPPD is characterised by rhomboid-shaped, positively birefringent crystals, in contrast to the needle-shaped, negatively birefringent crystals observed in gout [1]. Gout shows characteristic tophi, which are aggregates of conspicuous, long, needle-like, negatively birefringent crystals surrounded by histiocytes, whereas rheumatoid arthritis has synovial hyperplasia with pannus formation, prominent lymphoplasmacytic infiltration, and no crystal deposits. Other inflammatory and septic arthritic disorders show a dense neutrophilic infiltrate in the absence of crystal deposition.

The majority of CPPD cases are sporadic; however, some forms are associated with diseases such as osteoarthritis, chronic kidney disease, hemochromatosis, hyperparathyroidism, Wilson's disease, hypothyroidism, hypophosphatasia, and hypomagnesaemia [10-13].

Management of CPPD varies and depends on the course of the disease. Although CPPD can cause inflammation, many patients do not present with elevated inflammatory markers [14]. Patients with abnormally high inflammatory factors, such as C-reactive protein levels, respond well to conservative treatments such as Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), corticosteroids, and low-dose colchicine [15,16]. However, cases with negative inflammatory markers, severe chronic manifestations, and neurological impairment necessitate operative intervention via open decompression through either laminectomy with fusion or laminoplasty [17,18]. Early surgical treatment yields better long-term outcomes and facilitates the return

Study	Age (years)	Sex	Symptoms	Site	Radiology	Histopathology	Polarising microscopy
I Kh Almadhoun MK and Ta'amneh OO [3]	Early 60s	Female	Neck stiffness, bilateral upper limb tingling numbness	C1-C2 cervical spine	MRI: C1-C2 cervical stenosis. CT scan: calcified mass	Nodular clusters of crystal deposits noted	Positive birefringence
Chen SL et al., [4]	69	Female	Neck pain, right upper limb weakness	C3-C5 cervical spine	MRI: C3-C5 disc prolapse and ossification of posterior longitudinal ligament	Purple aggregates of small rhomboidal crystals	Positive birefringence
Kobayashi T et al., [5]	70	Female	Neck pain, no neurological manifestations	C5-C6 cervical spine	CT: High-density area at C5-C6 laminae with fluid collection and calcification of yellow ligament	Crystals noted	Positive birefringence
Wilkinson BM et al., [6]	80	Female	Right lower limb weakness	C5-C6 cervical spine	CT: C5-6 disc space collapse with endplate destruction and possible osteomyelitis ordiscitis	Calcium pyrophosphate crystal deposits	Positive birefringence
Present case	78	Female	Lower back pain, left limb tingling numbness	L4-L5 lumbar spine	MRI: L4-L5 stenosis and disc prolapse	Basophilic to gray-brown crystalline material	Rhomboid-shaped crystals with positive birefringence

[Table/Fig-2]: Comparison among rare cases of spinal CPPD [3-6].

of neurological function. Furthermore, patients should be monitored over time to identify CPPD in other areas [19,20].

This case highlights a rare and isolated clinical presentation of CPPD causing lumbar spine myelopathy, which was discovered incidentally on histopathology after spinal decompression and discectomy surgery. CPPD or pseudogout should be considered a differential diagnosis, especially in elderly patients presenting with back pain associated with lumbar myelopathy. While imaging modalities may raise suspicion for CPPD by revealing chondrocalcinosis, they often lack the specificity to differentiate it from other degenerative or inflammatory conditions. The absence of chondrocalcinosis on imaging does not exclude the diagnosis of spinal CPPD. The demonstration of basophilic to greyish-brown crystals on histopathology and characteristic positively birefringent rhomboid-shaped crystals on polarised microscopy provides the gold standard for a definitive diagnosis.

CONCLUSION(S)

The present case report emphasises that histopathological examination, along with polarised microscopy, is essential in diagnosing CPPD disease despite an atypical clinical presentation or ambiguous imaging findings. The absence of chondrocalcinosis on imaging does not rule out the diagnosis of spinal CPPD. This underscores the importance of including pseudogout in the differential diagnosis of lumbar myelopathy, particularly in elderly patients. A timely and precise diagnosis can reduce diagnostic delays and facilitate early, targeted treatment strategies to prevent irreversible neurological impairments.

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