

Intradural Calcium Pyrophosphate Deposition Mimicking Calcified Disc Herniation: A Lumbosacral Case Report

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Abstract:

Calcium pyrophosphate dihydrate deposition disease (CPPD) is primarily a peripheral crystal arthropathy, whereas spinal and especially intradural involvement are extremely rare. We report a 54-year-old man presenting with a 10-year history of low-back pain and progressive left-sided radiculopathy. Imaging suggested L5–S1 calcified disc herniation or intradural tumor. Intraoperatively, a chalk-white mass was identified within the dural sleeve of the left S1 nerve root. Histopathological examination confirmed calcium pyrophosphate crystal deposition. After conservative excision, neurological symptoms improved markedly without short-term recurrence. This case highlights the diagnostic difficulty of intradural CPPD and emphasizes the importance of histopathological confirmation and limited decompressive surgery.

Keywords:

Pseudogout, Spine, Intradural Mass, Calcium Pyrophosphate Dihydrate, CPPD

1. Introduction

Calcium pyrophosphate dihydrate deposition disease (CPPD) is a metabolic crystal-induced disorder characterized by the accumulation of calcium pyrophosphate crystals in cartilage, fibrocartilage, and periarticular soft tissues. The condition has long been recognized in rheumatology practice, where it most commonly presents as acute pseudogout or chronic inflammatory arthropathy involving large peripheral joints. According to the updated 2023 ACR/EULAR classification criteria [1], CPPD encompasses a wide clinical spectrum, ranging from asymptomatic chondrocalcinosis to recurrent acute attacks and progressive structural joint degeneration. The knee and wrist are the most frequently affected sites, followed by the shoulder and ankle [2]. Epidemiologic studies estimate that chondrocalcinosis is present in approximately 10–15% of elderly individuals, though symptomatic CPPD is less common, with a predilection for women and older age groups. The disease burden is expected to rise with global aging trends, making awareness of its atypical presentations increasingly important.

Although peripheral joint involvement is well documented, axial skeletal manifestations are comparatively uncommon. When the spine is affected, previously reported cases have described crystal deposition in the intervertebral discs, facet joints,

ligamentum flavum, and epidural structures [3]. These locations share structural similarities with fibrocartilaginous tissues found in peripheral joints, which may partly explain their susceptibility to crystal deposition. However, involvement of the intradural compartment is distinctly rare. Only isolated case reports have described intradural CPPD lesions worldwide [4], and the true incidence remains unknown. A systematic review of spinal CPPD published in 2022 identified fewer than ten intradural cases, underscoring the exceptional nature of this presentation. The lumbar spine appears to be the most common site for intradural deposition, possibly due to greater mobility and degenerative changes.

The rarity of intradural CPPD poses significant diagnostic challenges. Intraspinial calcified lesions are more frequently attributed to degenerative disc disease, ossification of ligaments, meningioma, schwannoma, metastatic tumors, or chronic infectious processes [5]. Imaging findings such as hyperdensity on computed tomography and low signal intensity on T2-weighted magnetic resonance imaging are not specific to crystal deposition. Consequently, clinicians often favor more common etiologies in the differential diagnosis, especially when the lesion is located near a nerve root and associated with degenerative changes of the spine. In many instances, the preoperative diagnosis is calcified disc herniation or neoplasm, and the true nature of the lesion is only revealed during surgery or after histopathological analysis. This diagnostic ambiguity can lead to either overtreatment or undertreatment, both of which carry potential risks for the patient.

Misdiagnosis is not merely a theoretical concern. An incorrect assumption of neoplasm may lead to unnecessarily aggressive resection or extensive dural exploration. Conversely, interpreting the lesion as a simple calcified disc fragment may result in incomplete decompression if its intradural location is not appreciated. Therefore, awareness of intradural CPPD as a potential albeit rare entity is important for both diagnostic accuracy and surgical planning. Furthermore, because CPPD is a metabolic disorder, its recognition may prompt evaluation for associated conditions such as hyperparathyroidism, hemochromatosis, or hypomagnesemia, even though localized spinal deposition can occur in metabolically normal individuals.

In this report, we describe a case of intradural calcium pyrophosphate deposition located within the dural sleeve of the S1 nerve root at the lumbosacral junction. The lesion was initially interpreted radiologically as calcified disc herniation or intradural tumor. Definitive diagnosis was established only after histopathological examination. By presenting detailed clinical, radiological, operative, and pathological findings, and by integrating relevant literature, we aim to clarify the diagnostic considerations and management principles associated with this unusual presentation. This case also adds to the growing body of evidence that CPPD should be included in the differential diagnosis of any calcified intraspinal lesion, regardless of its location or the patient's metabolic status.

2. Case Presentation

2.1. Clinical history

A 54-year-old man was admitted to our department with complaints of persistent low-back pain accompanied by radiating discomfort along the posterior aspect of the left lower extremity. The symptoms began approximately ten years earlier after a lumbar sprain sustained while lifting a heavy object at work. Initially, the pain was

intermittent and manageable, occurring mainly after prolonged physical activity or prolonged standing. Over time, however, the frequency and severity gradually increased, interfering with daily activities such as walking and sitting for extended periods. The patient, a construction worker by occupation, had to modify his work duties and eventually take sick leave due to incapacitating pain.

Six months prior to admission, the patient noticed a distinct change in the character of his symptoms. The radiating pain intensified and extended from the buttock to the posterior thigh, calf, and medial ankle. Walking for moderate distances—approximately 200 meters—triggered a burning or electric-like sensation, forcing him to stop and rest. These episodes were consistent with neurogenic claudication. In addition, he reported occasional paresthesia in the left foot, described as “tingling” and “numbness,” particularly in the little toe and lateral border of the foot. Conservative treatments, including oral analgesics (nonsteroidal anti-inflammatory drugs and acetaminophen) and physiotherapy, provided only transient relief. Epidural steroid injections were not administered.

The patient denied any history of gout, hyperparathyroidism, chronic kidney disease, hemochromatosis, rheumatoid arthritis, or other systemic metabolic or inflammatory disorders. There was no prior history of spinal surgery or malignancy. Family history was unremarkable for rheumatic diseases or spinal disorders. He had no known allergies and was not taking any regular medications aside from occasional over-the-counter pain relievers.

The prolonged and progressive nature of the symptoms suggested chronic nerve root compression rather than acute inflammatory pathology. The absence of constitutional symptoms such as fever, weight loss, or night sweats argued against infectious or malignant processes.

2.2. Physical examination

On examination, the patient ambulated with a mild antalgic gait favoring the left side. Lumbar lordosis was reduced. Palpation revealed localized tenderness over the lower lumbar region, particularly at the L5/S1 level, with mild paraspinal muscle spasm. Lumbar flexion and extension were moderately restricted due to pain, while lateral bending and rotation were preserved.

The straight-leg raise test was positive at 30° on the left side, reproducing radicular pain radiating to the calf and ankle. Contralateral straight-leg raise was negative. The crossed straight-leg raise test was also negative. Sensory examination revealed diminished light-touch and pinprick sensation along the S1 dermatome, including the lateral aspect of the foot and the little toe. Proprioception and vibration sense were intact. Motor strength in both lower extremities was preserved (Medical Research Council grade 5/5) for hip flexion, knee extension, ankle dorsiflexion, and great toe extension. However, subtle weakness in left ankle plantarflexion (grade 4+/5) was noted on repeated testing, though the patient could still walk on tiptoes. Deep tendon reflexes were normal at the knees (2+) but absent at both ankles. No Babinski sign or other upper motor neuron signs were detected. Rectal tone and perianal sensation were normal, excluding cauda equina syndrome.

Overall, the neurological findings were consistent with chronic compression of the left S1 nerve root, with both sensory and motor involvement.

2.3. Laboratory findings

Routine laboratory tests, including complete blood count, erythrocyte sedimentation rate, and C-reactive protein, were within normal limits. Serum uric acid was 347 $\mu\text{mol/L}$ (reference range 200–420 $\mu\text{mol/L}$), and serum calcium was 2.31 mmol/L (reference range 2.10–2.55 mmol/L). Serum phosphate, magnesium, and alkaline phosphatase were also normal. Tests for rheumatoid factor, anti-cyclic citrullinated peptide antibodies, and antinuclear antibodies were negative. Tumor markers (carcinoembryonic antigen, alpha-fetoprotein, CA19-9) were within reference ranges. Thyroid function tests and parathyroid hormone level were unremarkable.

Although systemic metabolic disorders are frequently associated with CPPD [7], the absence of laboratory abnormalities does not exclude localized crystal deposition. Indeed, several previously reported spinal CPPD cases have occurred in metabolically normal individuals. Screening for hemochromatosis (ferritin, transferrin saturation) and Wilson's disease was not performed due to lack of clinical suspicion.

2.4. Imaging studies

Lumbar computed tomography (CT) revealed a 0.7×0.5 cm nodular hyperdense lesion adjacent to the left S1 nerve root, just medial to the pedicle (Figure 1). The lesion exhibited well-defined margins and homogeneous calcification, with a density of approximately 450 Hounsfield units, consistent with calcium deposition. No bony erosion, vertebral body destruction, or foraminal widening was observed. The adjacent intervertebral disc at L5–S1 showed mild height loss and vacuum phenomenon, indicative of degenerative disc disease.



Figure 1. Sagittal (A) and axial (B) lumbar CT images obtained in February 2025 showing a $0.7 \text{ cm} \times 0.5 \text{ cm}$ calcified lesion adjacent to the left S1 nerve root.

Magnetic resonance imaging (MRI) of the lumbar spine demonstrated degenerative disc changes at L5–S1, including disc desiccation and mild bulging. The calcified lesion appeared as a low-signal-intensity structure on T2-weighted sequences, and on T1-weighted images it was isointense to the nerve root. After gadolinium administration, no significant enhancement was noted within the lesion, though mild enhancement of the surrounding dura was observed. The lesion was located within the spinal canal, apparently compressing the left S1 nerve root (Figure 2). There was no evidence of associated soft tissue edema or contrast-enhancing mass suggestive of infection or tumor.

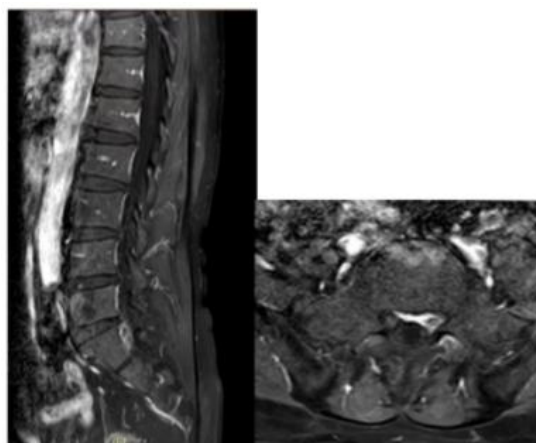


Figure 2. Contrast-enhanced lumbar MRI: sagittal and axial views.

Although the imaging findings were compatible with calcified disc herniation, the lesion's relatively discrete and encapsulated appearance, together with its intradural location suggested by its relationship to the thecal sac, raised suspicion of possible intradural pathology. However, given the presence of degenerative disc changes at the same level, calcified disc extrusion remained the leading preoperative diagnosis.

The differential diagnosis included:

Calcified disc herniation (extruded or sequestered)

Intradural schwannoma with calcification

Meningioma (psammomatous type)

Tophaceous gout

Metastatic tumor (e.g., from lung, breast, or prostate)

Infectious processes such as tuberculoma or calcified abscess

Synovial cyst with calcification

Given the progressive neurological symptoms and radiologic evidence of nerve root compression, surgical exploration was recommended.

2.5. Surgical findings

Under general anesthesia and operating microscope guidance, a posterior approach to the L5–S1 level was performed. The patient was positioned prone on a Jackson table with careful padding. A midline incision was made, and the paravertebral muscles were dissected subperiosteally to expose the laminae of L5 and the upper sacrum. Partial laminectomy of L5 and medial facetectomy were carried out to expose the left S1 nerve root. The ligamentum flavum was removed, revealing a normal-appearing epidural space without any extruded disc material.

Upon further exploration of the left S1 nerve root region, the dura was noted to be tense. A small durotomy was performed longitudinally over the nerve root sleeve, and a firm, chalk-white, encapsulated mass measuring approximately 0.5×0.4 cm was discovered within the dural sleeve, intimately adherent to the nerve root fibers (Figure 3). The lesion was clearly intradural and distinct from disc material. It was non-vascular, had a crystalline appearance, and was easily separated from the nerve root

with microdissection. There was no evidence of infiltration into surrounding neural structures, and the nerve root appeared otherwise healthy.



Figure 3. Intraoperative view showing a white calcified nodule within the S1 nerve-root dural sleeve.

Meticulous microsurgical dissection allowed en bloc excision of the mass while preserving the integrity of the nerve root (Figure 4). The dura was repaired primarily with 6-0 Prolene sutures, and a Valsalva maneuver confirmed a watertight closure. Fibrin glue was applied to reinforce the repair. The wound was closed in layers over a subfascial drain. The 2.3-cm incision healed uneventfully (Figure 5).



Figure 4. Calcified material completely excised during surgery.



Figure 5. Post-operative incision length approximately 2.3 cm.

2.6. Histopathological examination

The excised specimen was sent for histopathological analysis. Grossly, it appeared as a white, firm nodule. Hematoxylin and eosin staining revealed amorphous basophilic deposits surrounded by multinucleated giant cells, histiocytes, and chronic inflammatory infiltration, consistent with a foreign-body reaction. Under polarized light microscopy, rhomboid crystals exhibiting weak positive birefringence were observed, confirming the presence of calcium pyrophosphate dihydrate crystals [11]. No evidence of malignancy or infection was seen.

The pathological findings confirmed the diagnosis of intradural CPPD.

2.7. Postoperative course

The patient reported immediate relief of radicular pain following surgery. Mild residual numbness in the left S1 distribution gradually improved over the subsequent

weeks. A small postoperative cerebrospinal fluid leak occurred on day 2, presenting as clear fluid oozing from the incision. This was managed conservatively with bed rest, pressure dressing, and placement of a lumbar drain for 48 hours. The leak resolved without further intervention. The surgical incision healed without infection.

At short-term follow-up (3 months), neurological function remained stable, with complete resolution of motor weakness and only mild residual sensory symptoms. The patient returned to his normal activities, including light work duties. Follow-up MRI at 6 months showed no evidence of residual or recurrent mass, and the patient remained asymptomatic. Longer-term follow-up is planned annually to monitor for possible recurrence.

3. Discussion

3.1. Pathophysiological considerations

CPPD results from abnormal pyrophosphate metabolism and subsequent crystal deposition within connective tissues [6]. Aging, cartilage degeneration, and local biochemical changes contribute to crystal nucleation. At the molecular level, excess extracellular inorganic pyrophosphate promotes crystal formation, a process regulated by enzymes such as ANKH (progressive ankylosis protein homolog) and tissue-nonspecific alkaline phosphatase. Genetic mutations in the ANKH gene have been linked to familial forms of CPPD, though sporadic cases are more common. Epidemiologic studies have demonstrated associations with hyperparathyroidism, hemochromatosis, chronic kidney disease, osteoarthritis, and hypomagnesemia [7]. These conditions alter calcium or phosphate homeostasis, creating a microenvironment conducive to crystal precipitation.

Nevertheless, intradural involvement remains poorly understood. Unlike articular cartilage, the intradural compartment does not contain typical fibrocartilaginous structures. It is possible that chronic mechanical stress, microtrauma, or degenerative changes in adjacent structures create a microenvironment conducive to ectopic crystal deposition. The nerve root sleeve, composed of dura mater and arachnoid, may undergo repeated tensile and compressive forces during spinal movement, leading to microscopic tears and subsequent crystal nucleation. Additionally, age-related degeneration of the intervertebral disc and facet joints may release inflammatory mediators that promote crystal formation in nearby tissues.

In the present case, the patient had a history of long-standing lumbar strain from heavy lifting. Repetitive mechanical stress may have contributed to local tissue alteration and facilitated crystal accumulation within the nerve root sleeve. The absence of systemic metabolic abnormalities suggests that local factors played a predominant role in this patient's disease.

3.2. Radiologic challenges

Imaging findings of spinal CPPD are often nonspecific. CT hyperdensity and low T2 signal intensity are characteristic but not pathognomonic [10]. Calcified tumors (e.g., meningioma, schwannoma), chronic infections (e.g., tuberculoma), and degenerative lesions (e.g., calcified disc herniation, ossified ligamentum flavum) may present similarly. In this case, the lesion's location adjacent to a degenerated disc naturally led to the presumption of a calcified disc fragment. However, certain features may raise suspicion for CPPD: the presence of well-circumscribed,

homogeneous calcification without associated bone destruction or edema, and the lack of significant contrast enhancement. In retrospect, the lesion's intimate relationship with the nerve root sleeve rather than the disc space could have suggested an intradural origin.

Previous literature has documented comparable diagnostic difficulties. López-Navarro et al. described lumbar intraspinal CPPD initially interpreted as disc herniation [8]. Wilkinson et al. reported cervical CPPD mimicking osteomyelitis and epidural abscess [9]. In both instances, the correct diagnosis was only established after histopathological examination. A recent review by Tekaya et al. emphasized that spinal CPPD should be considered in any patient with chronic radiculopathy and imaging evidence of calcified intraspinal lesion, particularly when the clinical course is indolent and metabolic workup is unrevealing.

Recognition of well-circumscribed calcification without aggressive bone destruction may provide a subtle clue favoring benign crystal deposition. However, the rarity of intradural CPPD means that most radiologists and spine surgeons will encounter it only once or twice in their careers. Therefore, a high index of suspicion is necessary when atypical features are present, such as an encapsulated appearance or lack of continuity with the disc space.

3.3. Histopathology as definitive diagnosis

Definitive diagnosis relies on histopathological examination. Under polarized light microscopy, CPP crystals demonstrate weak positive birefringence, distinguishing them from monosodium urate crystals, which exhibit strong negative birefringence [11]. The rhomboid morphology of CPP crystals is also characteristic. In this case, the presence of multinucleated giant cells and chronic inflammation reflects a foreign-body reaction to the crystals, a common finding in tumoral CPPD. Similar pathological patterns have been reported in tumoral CPPD of cranial structures [12], as well as in spinal locations.

It is important to differentiate CPPD from other crystal deposition diseases, such as gout and basic calcium phosphate (hydroxyapatite) deposition. Hydroxyapatite crystals are not birefringent and appear as non-specific calcific deposits on histology. The clinical context and imaging may help, but polarized light microscopy remains the gold standard for identifying CPP crystals. In some cases, electron microscopy or Raman spectroscopy can provide additional confirmation, though these are rarely needed in routine practice.

3.4. Surgical strategy

When neurological compression is present, surgical decompression is warranted. Conservative en bloc excision aimed at relieving neural compression is sufficient in most cases. Because CPPD lesions are typically benign and non-infiltrative, complete excision is not necessary as long as the neural elements are adequately decompressed. Aggressive resection may increase the risk of nerve root injury, cerebrospinal fluid leak, or postoperative instability.

Previously reported intradural cases [13] achieved favorable outcomes with limited resection. Aggressive or radical excision is unnecessary and may increase morbidity. In our patient, en bloc excision was possible due to the lesion's well-circumscribed nature and lack of adherence to neural tissue. The use of an operating microscope facilitated meticulous dissection and preservation of nerve root fibers. The subsequent

cerebrospinal fluid leak, though a known complication of intradural surgery, was managed conservatively and did not affect the long-term outcome.

Postoperative management should include monitoring for recurrence, though data on long-term outcomes are sparse. In peripheral joints, CPPD can recur after synovectomy, but spinal recurrence appears to be rare based on limited case reports. Nevertheless, periodic clinical and radiological follow-up is advisable.

3.5. Clinical implications and literature integration

The present case adds to the limited body of literature documenting intradural CPPD. Compared with previously reported cases, our patient demonstrated a prolonged clinical course (10 years) and isolated nerve root involvement without systemic metabolic disorder. This pattern is consistent with the hypothesis that local mechanical factors contribute to crystal deposition. Notably, the lesion was located within the dural sleeve of the nerve root, a site not previously emphasized in the literature. This location may represent a predilection for intradural CPPD, possibly due to the unique biomechanical forces acting on the nerve root sleeve during spinal movement.

A comparison with other intradural CPPD cases reveals both similarities and differences. Chakravorty et al. [4] reported a case of intradural CPPD at the lumbar level in a patient with back pain and radiculopathy, similar to our patient. However, their lesion was located in the cauda equina rather than a single nerve root sleeve. Gewolb et al. [14] described a case involving the filum terminale. These variations suggest that intradural CPPD can occur anywhere within the spinal canal, and the clinical presentation depends on the specific neural structures involved.

This case reinforces the importance of integrating clinical history, imaging interpretation, intraoperative findings, and histopathology in establishing diagnosis. Increased awareness among spine surgeons and radiologists may reduce misdiagnosis and guide appropriate surgical management. Furthermore, recognition of CPPD may prompt evaluation for underlying metabolic disorders, even though many cases are idiopathic. From a therapeutic standpoint, conservative excision is safe and effective, and patients can expect excellent neurological recovery.

4. Conclusions

Intradural spinal CPPD represents a rare but clinically significant cause of radiculopathy. Imaging findings may mimic calcified disc herniation or neoplasm, making preoperative diagnosis challenging. Histopathological confirmation is essential for accurate diagnosis. Compared with previously reported cases, the present case further supports that conservative surgical excision provides satisfactory neurological recovery. This study contributes to the limited body of literature by reinforcing the importance of including CPPD in the differential diagnosis of calcified intradural lesions.

Theoretically, this case expands understanding of atypical spinal manifestations of metabolic crystal disease. Clinically, it emphasizes careful integration of imaging and intraoperative findings to guide management. However, long-term follow-up data remain limited, and further case accumulation is needed to clarify recurrence risk and optimal postoperative strategies. Increased awareness among spine surgeons and radiologists may reduce misdiagnosis and prevent unnecessary aggressive intervention. Future research should focus on the molecular mechanisms underlying

ectopic crystal deposition in neural tissues and the potential role of local mechanical factors.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Author Contributions

Tao Peng contributed to patient management and manuscript drafting. Daming Peng contributed to data collection. Hui Zhang supervised the study and critically revised the manuscript. All authors read and approved the final manuscript.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was waived because this is a single case report without experimental intervention.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Availability of Data and Materials

All data generated or analysed during this study are included in this published article.

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