Foraminal deposition of calcium pyrophosphate dihydrate crystals in the thoracic spine: possible relationship with disc herniation and implications for surgical planning

Report of two cases

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The authors report two cases of nodular calcium pyrophosphate dihydrate (CPPD) crystal deposition close to the thoracic neural foramens, which caused chronic radiculopathy. Preoperatively, the lesions were interpreted as calcified disc herniations. Both patients underwent surgery in which an extended transfacet pedicle-sparing approach was used. Incision of the posterior longitudinal ligament released soft degenerated material. In both cases, histological examination showed abundant degenerative debris along with CPPD crystals. Spinal CPPD deposition is a comparatively rare disease that almost invariably involves the posterior aspect of the spinal canal, typically the ligamentum flavum. The exceptional foraminal location of the lesions reported here, combined with the surgical findings, indicated that the CPPD crystals were deposited on a laterally herniated disc fragment. A distinctive feature in both cases was the soft consistency of the resected tissue. The consistency of the disc material and the location of the lesion in the axial plane (that is, median compared with lateral) are key factors in determining the optimal surgical approach to thoracic disc herniations. In describing consistency, terms such as “calcified” and “hard” have been used interchangeably in the literature. In the cases reported here, what appeared on computerized tomography and magnetic resonance imaging studies to be densely calcified lesions were shown intraoperatively to be soft herniations.

The authors’ experience underscores that not all densely calcified herniated discs are hard. Although detection of this discrepancy would have left surgical planning for the lateral disc herniations unchanged, it could have altered planning for centrally or centrolaterally located disc herniations.

KEY WORDS • calcification • calcium pyrophosphate dihydrate • disc herniation • foramen • thoracic spine

Abbreviations used in this paper: CPPD = calcium pyrophosphate dihydrate; CT = computerized tomography; MR = magnetic resonance; PLL = posterior longitudinal ligament.

Case Reports

Case 1

This 36-year-old woman presented with a 1-month history of unremitting left-sided thoracic pain. Before admission, an MR imaging study had demonstrated a T9–10 lateral disc herniation (Fig. 1 left). The disc appeared hy-
pointense on T1- and T2-weighted MR images. Neuro-
logical examination yielded normal findings. Axial CT
scanning revealed a completely calcified herniated disc
(Fig. 1 center). The patient underwent surgery. The lesion
was exposed via a transfacet pedicle-sparing approach
extended with a T-9 hemilaminectomy. Incision of the
PLL released a glistening, finely granular material that
was easily removed piecemeal. No parts of the mass were
hard (Fig. 1 right). Histological examination showed de-
generating material and CPPD crystals (Fig. 2 upper).
After surgery, the radicular pain abated. At 15 months the
patient was symptom free.

Case 2

This 43-year-old woman underwent MR imaging to dis-
cern the source of intractable burning pain, of approxi-
mately 6 months’ duration, in the left hemithorax. A nodu-
lar lesion, hypointense on T1- and T2-weighted images,
was found to efface the left neural foramen, at the T9–10
segment (Fig. 3 upper). Axial CT scanning demonstrated
that the lesion had completely calcified (Fig. 3 lower). On
admission neurological examination yielded normal find-
ings except for mild hypesthesia specific to the left T-9
dermatome. The patient underwent a transfacet pedicle-
sparing approach that was extended medially via a T-9
hemilaminectomy. When the foramen was unroofed, we
observed a bulging subligamentous mass that displaced
the nerve root posteriorly. Incision of the PLL released
soft degenerated material, which was removed, mostly by
using suction. Histological examination showed massive
deposition of CPPD crystals and abundant degenerating
debris (Fig. 2 lower). After surgery the patient reported
that the radicular pain had completely resolved. At the 1-
year follow-up examination the patient was well.

Discussion

Calcium pyrophosphate dihydrate spinal deposition is a
comparatively rare disease only occasionally observed in
the cervical and the lumbar spine8,20 and, rarely, in the tho-
racic spine.12,13 In previously reported cases, the crystal
deposition caused diffuse or nodular thickening of the liga-
mentum flavum, thus compressing the spinal cord dorsally.
To our knowledge, a foraminal location has not been de-
scribed. In our patients, the foraminal location, the apparent
continuity between the lesions and the disc space, and the
intraoperative findings (an intact PLL covered the lesions)
all indicated that CPPD crystals were deposited on a loose
disc fragment herniated within the foramen. This possible
sequence of events receives support from autopsy7 and
clinical8 studies in which CPPD deposition tropism was
documented in intervertebral discs.

A distinctive finding in our two cases was the mislead-
ing imaging appearances of the lesions. The high density
of the lesions on CT scans and lack of signal on T1- and
T2-weighted MR imaging sequences indicated typical in-

S. Paolini, et al.

FIG. 1. Case 1. Left: Sagittal T1-weighted MR image revealing a herniated disc close to the left T9–10 neural fora-
men. The low signal intensity indicates the presence of calcifications. Center: Axial CT scan demonstrating the bone-
like intensity of the lesion. Right: Intraoperative photograph obtained at the end of the procedure. The lesion, on the left
side of the dural sac (arrow), has been emptied of its soft contents and its capsule retracted by a dissector.

FIG. 2. Photomicrographs obtained in Cases 1 (upper) and 2 (lower), showing amorphous crystalline deposits of CPPD and
degenerating disc material. H & E, original magnification × 250.
Foraminal CPPD deposition in the thoracic spine

Sagittal T₁-weighted MR image revealing a round hypointense lesion within the left T₉–₁₀ neural foramen, indicating a calcified disc herniation. Lower: Axial CT scan revealing the lesion’s bright intensity, which confirms the presence of massive calcifications.

Assuming that calcium deposition affects the consistency of the disc, authors have invariably emphasized the role of intradiscal calcifications in surgical planning in the management of thoracic disc herniations.⁴,¹⁰,¹⁴,¹⁷,²²,²₄,²₅,²₇ Whereas soft-disc herniations can be grasped and removed piecemeal despite their oblique orientation, hard disc herniations generally must be demolished using a microdrill. Because an unobstructed ventral view of the canal is essential to control the entire base of the lesion, centrally located hard-disc herniations are best exposed via an anterolateral approach.

In our cases, the imaging appearance of the lesions had no bearing on the operative strategy. Because of the lesions’ lateral position and the lack of overlying dural draping, we could have safely undertaken a posterolateral approach regardless of the consistency of the disc.²⁴ The unexpected intraoperative finding of a soft consistency nevertheless indicates that the presumed role of intradiscal calcifications in the surgical planning hinges on a misconception. The structural feature contraindicating a posterolateral approach to a central disc herniation should probably be defined as “ossification,” rather than “calcification.” Calcification alone does not exclude the presence of massive degenerative phenomena that can soften the disc material. Accordingly, degenerative phenomena have already been found in association with CPPD crystals and attributed to the proteolytic action of disease-related inflammatory mediators.¹² Consequently, a calcified disc that would normally require transthoracic surgery could instead be treated via a posterolateral approach alone, if preoperative imaging studies could differentiate degenerative calcified tissue from ossified herniated disc fragments. Our experience therefore raises a new problem with preoperative diagnosis. As a working hypothesis, we conjecture that contrast-enhanced MR imaging might be used to detect inflammatory tissue,¹⁹ a marker of degeneration related to CPPD deposition. Uptake of contrast agent, however, is a common imaging finding in posterior disc herniations.³ Additionally, the precise relationship between the intensity of contrast enhancement and the presence of macroscopically visible proteolytic phenomena would be hard to establish. A more effective method might be to disclose the presence of an amorphous matrix directly in the setting of a calcified formation. Diffusion-weighted MR imaging, a modality used only for central nervous system lesions despite its high sensitivity to hydrophilic components, has the drawback of calcium-related artifacts. The best solution would be to use strongly T₂-weighted MR imaging sequences that are scarcely influenced by calcified tissue and highly sensitive to soft tissue (namely, pulse sequences with a long echo time and a long echo train). Should it prove effective, this modality might be used as an adjunct in the routine diagnostic imaging workup of calcified herniated discs, especially when the patient’s medical history comprises various endocrine or metabolic conditions potentially associated with the development of pseudogout.

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S. Paolini, et al.