

Rosenthal fibers in cervical enhancing myelopathic lesion: spondylosis or tumor?

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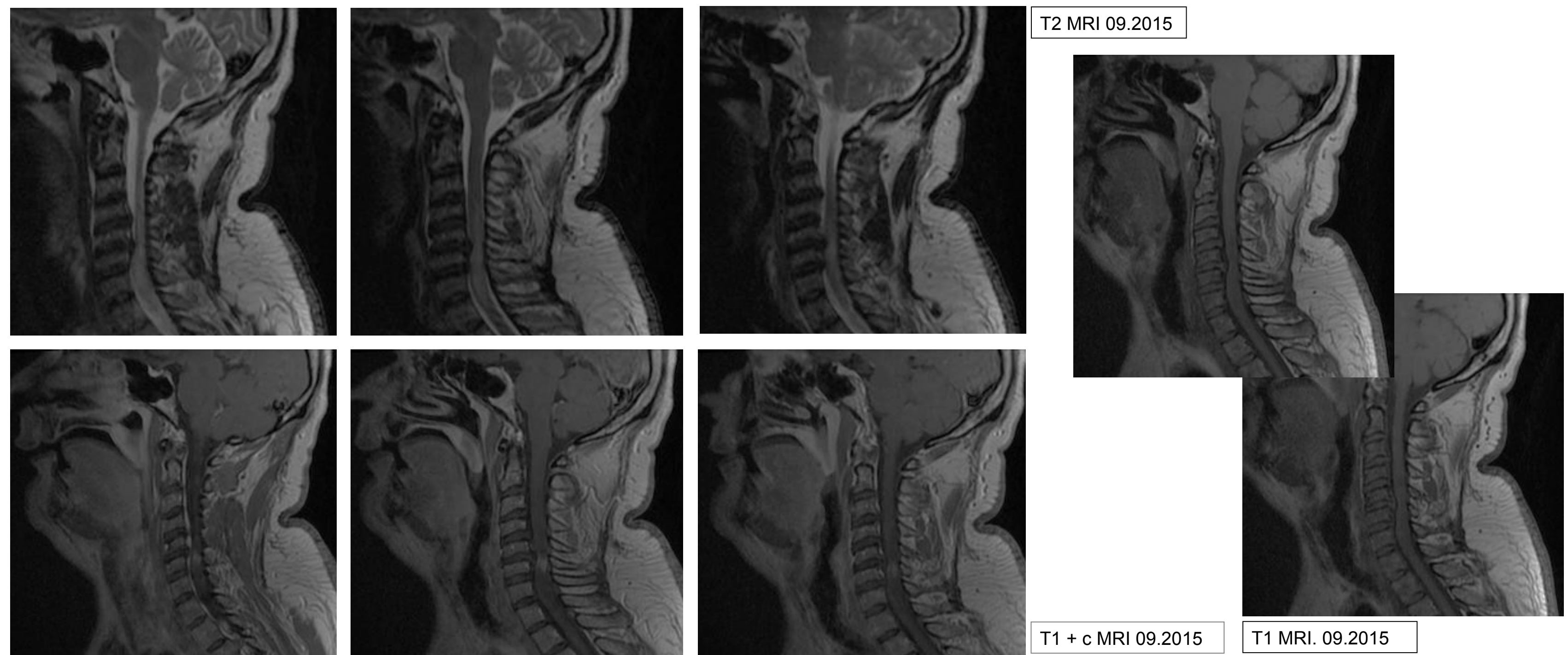
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Objective: We describe the case of a 42-year-old man affected by cervical spondylotic myelopathy, showing a clinical aggressive course after surgical treatment and persistent cervical MRI T2 hyperintense and enhancing lesion, suspicious for oncological lesion.

Case report: Patient's neurological history began three years ago, when he experienced cervicobrachial pain, left upper limb paresthesia and numbness. One year later he developed mild paraparesis and lower limb hypoesthesia. Spinal cord MRI demonstrated a cervical spondylotic myelopathy extended from C3 to C6 and C4-D1 spinal cord T2 hyperintense lesion, associated with C5-C6 contrast enhancement. Spinal cord appeared swollen.

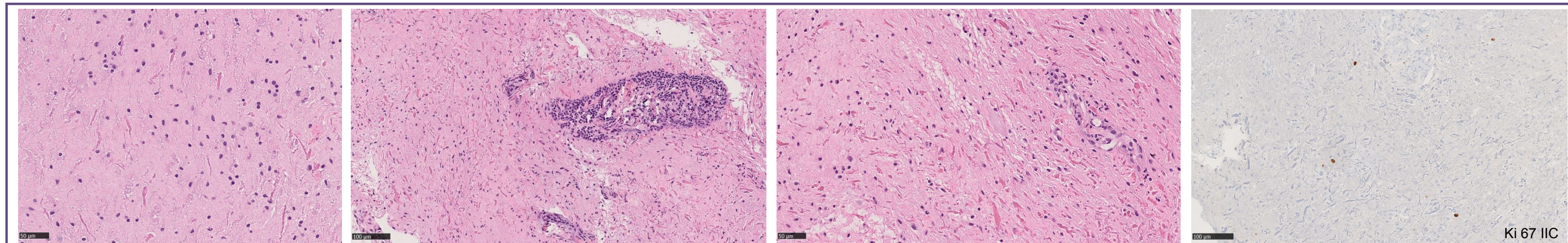
The patient subsequently underwent an anterior C4-C5 and C5-C6 discectomy and arthrodesis. In the following two months symptoms worsened, leading to moderate paraparesis and sphincter dysfunction.



A new MRI showed the persistence of a cervical T2 hyperintense lesion with irregular contrast enhancement. Serum and cerebrospinal fluid (CSF) examination revealed no signs of infectious or inflammatory disease



Cervical spinal biopsy was performed and histological examination revealed mild increase in glial cells, perivascular lymphocytes, eosinophilic granulocytes and Rosenthal fibers, arising the hypothesis of a pilocytic astrocytoma.



After discharge, patient's conditions began to improve, but only temporary, leading to persisting moderate paraparesis.

Spinal cord MRI performed three months after biopsy confirmed a T2 hyperintense C4-C6 lesion, with mildly reduced C5-C6 irregular contrast enhancement.



At subsequent MRI control, at seven months from biopsy, there was a reduction of both diffuse T2 hyperintensity and contrast enhancement on known cervical lesion, that correlated with neurological improvement.



Discussion: It is well known that spinal cord MRI T2 hyperintense lesions correlate with clinical outcome in spondylotic myelopathy (1). Contrast enhancement has been reported infrequently in spondylotic myelopathy and might be a consequence of disturbed blood circulation (2). Enhancement disappearance after surgery is variable, since some case reports show a persistence 1 year after decompression (1).

Clinical and MRI findings in our case were not typical for pilocytic astrocytoma for several reasons: patient age, spinal cord localization (extremely rare) at a discal hernia site, irregular and poor defined contrast enhancement. Rosenthal fibers (RF) can argue against the hypothesis of a spondylotic myelopathy, being a common finding in pilocytic astrocytoma. Nonetheless, RF are described in other pathologies, e.g. in reactive tissue (highly gliotic tissue surrounding cysts or vascular malformations) or Alexander disease, probably representing the by-product of GFAP (glial fibrillary acidic protein) upregulated in response to stress or genetic mutations (3).

Conclusions: Contrast enhancement at the site of discal hernia can represent a sign of severe spondylotic myelopathy. However, other diagnosis must be excluded, especially in patients with rapid neurological worsening.

Bibliography:

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