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A rare case of long-standing intracranial hypertension secondary to a lumbosacral extradural tumor

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Introduction
Intracranial hypertension (ICH) caused by a spinal cord tumor is a rare but well-known condition. Identifying the disease is particularly challenging when characteristic spinal symptoms or signs are absent.
Objective
To report a rare case of long-standing intracranial hypertension secondary to a lumbosacral extradural tumor.
Case Report
A 76-year-old male patient, with a 10-year history of headache associated with visual impairment and bilateral papilledema, which was diagnosed as idiopathic intracranial hypertension. One year ago, he developed mental confusion, imbalance, and gait disturbances. Sixty days before admission, he presented weakness in the lower limbs, and 15 days ago he experienced new-onset seizures. On examination, anisocoria (right larger than the left), sluggish pupillary reaction to light in the left eye, and visual acuity of counting fingers at 4 meters in the right eye and 20/80 in the left eye. Fundoscopy showed optic nerve pallor (grade 2+), papillary edema with 360-degree blurring (Frisen grade 4) in the right eye. Cerebrospinal fluid (CSF) analysis, collected via suboccipital puncture, with an opening pressure of 35 cmH2O and a protein level of 81 mg/dL. Xanthochromia was present on this CSF sample and had been described in previous ones, though not investigated because the patient lost follow-up. A angiography of cranial vessels excluded any source or intracranial bleeding.Magnetic resonance imaging (MRI) of the brain showed a partially empty sella,diffuse proportional cerebral atrophy and some spots of superficial siderosis over the cerebellum. The patient underwent optic nerve fenestration and subsequently a ventriculoperitoneal shunt to relieve intracranial pressure and reduce optic nerve suffering. An MRI of the spine revealed an intradural lesion between L4-S1, approximately 6.1 cm in the craniocaudal axis,inferring compression of the cauda equina roots, without involvement of the conus medullaris. Spinal arteriography confirmed a hypervascular lesion, with blood supply from the anterior spinal artery and ectasia of the medullary drainage veins. After neurosurgical evaluation, the patient, in clinical stability, chose not to further stratify the lesion.
Conclusion
Since ICH can lead to a permanent optical loss, spinal MRI may be considered soon after the initial investigation to exclude atypical and rare cases of intracranial hypertension.