Thoracic disc herniation associated with papilledema

Case report

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The occurrence of papilledema in a patient with progressive spastic paraparesis due to herniation of the T11-12 intervertebral disc is reported. The papilledema resolved following discectomy. The association and possible pathogenetic mechanisms between spinal cord lesions and signs of raised intracranial pressure are reviewed.

KEY WORDS • papilledema • thoracic spine • intervertebral disc • spinal cord • intracranial pressure

The association of spinal cord tumors and papilledema is a rare though well recorded entity. We present here a patient in whom a thoracic spinal cord discogenic compressive lesion was associated with papilledema, which resolved following the removal of the disc. Such an association has not been described previously.

Case Report

This 47-year-old man presented with a 21-year history of progressive weakness in his right leg followed by weakness in the left lower extremity. Several months prior to his hospitalization, he required the use of a cane for ambulation and felt paresthesias in both legs. He also complained of back pain and difficulty in initiating urination. Approximately 1 month prior to his hospitalization he began to suffer from headaches and visual disturbances.

Examination. His visual acuity was 6/30 bilaterally. Funduscopic examination revealed florid bilateral papilledema with hemorrhages (Fig. 1). Spastic paraparesis, hyperreflexia, bilateral patellar clonus, and plantar extensor responses were found. Abdominal reflexes were present only in the upper quadrants. On sensory examination, hypesthesia was present below the T11-12 level.

Computerized tomography (CT) of the brain was within normal limits. Lumbar puncture disclosed an opening pressure of 120 mm H2O. Cerebrospinal fluid (CSF) analysis revealed 94 cells, all of which were lymphocytes, a protein concentration of 150 mg%, and a glucose content of 49 mg%. Myelography (Fig. 2) revealed a filling defect compatible with disc herniation at the T11-12 interspace. Cerebral arteriography was normal, with no evidence of a venous drainage abnormality.

Operation. Using a posterolateral approach, we performed a costotransversectomy with removal of the T11-12 disc. Following surgery there was significant improvement of muscle strength in the lower extremities. The spasticity resolved and the gait improved within a number of days. Significant improvement of the visual acuity and papilledema was observed (Fig. 1 right).

Postoperative Course. By 1 1/2 months postoperatively the papilledema had completely disappeared. At evaluation 1 1/2 years after surgery, the patient was symptom-free with regard to spasticity and to the appearance of the optic disc (Fig. 3), visual acuity, and fields.

Discussion

Thoracic disc herniation is an infrequent cause of spinal cord compression. Only 0.15% to 1.7% of spinal discectomies are performed on thoracic discs. The T11-12 interspace is most frequently involved.110 An-
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FIG. 1. Left: Preoperative funduscopy showing bilateral florid papilledema with hemorrhages on the left. Right: Bilateral improvement of papilledema is seen 2 weeks postoperatively.

FIG. 2. Anteroposterior myelogram showing a filling defect opposite the T11-12 intervertebral space (arrow).

FIG. 3. Funduscopy 1½ years postoperatively showing the residual appearance after resolution of papilledema. The black dot in the region of the disc is due to a technical problem in the Zeiss camera.

To the best of our knowledge, an association of thoracic disc herniation with papilledema has not been described previously. An intracranial cause for the papilledema in our patient was ruled out by CT and cerebral angiography. The ventricles were of normal size, and no pathology was seen in the venous drainage.

The association of papilledema with spinal cord lesions is unusual, with only 53 cases reported in the literature. The common denominator in all these cases was the resolution of the papilledema following surgical removal of the spinal lesion. The majority of the lesions were intradural tumors, with ependymoma being the pathological diagnosis in over half of the cases. There have been only two reports of an extradural lesion.
associated with signs of increased intracranial pressure (ICP): a T-12 meningioma reported by Mittal, et al. and a thoracic sympatoblastoma described by Schijman, et al.

We cannot propose a uniform pathophysiological mechanism to explain the association of signs of raised ICP with spinal lesions. Elevated protein content in the subarachnoid fluid is cited by most authors as a possible mechanism. Gardner, et al., postulated that large protein molecules may mechanically block the pores at the sites of CSF absorption, thus leading to raised ICP. Davson, et al., showed experimentally that infusion of serum into the CSF slowed absorption, although this effect could be observed even after filtering the CSF through a 0.5-μ pore membrane which eliminated most protein particles. Moreover, repeated intracranial injections of serum in monkeys did not bring about papilledema.

The association of papilledema with the Guillain-Barré syndrome may also implicate high CSF protein levels in raising ICP. However, this fails to explain the large number of cases of Guillain-Barré syndrome with very high CSF protein levels without raised ICP, or with papilledema and only mildly elevated or even normal CSF protein values. The same argument applies to cases of spinal lesions. The large majority of such lesions do not cause signs of increased ICP, even in the presence of high CSF protein levels. On the other hand, there are cases in which the association was reported although CSF protein levels were only mildly elevated.

An additional factor cited is the possible irritative effect of hyperproteinorachia and other tumor release products on the arachnoid membranes. Arseni and Maretsis operated on two cases of posterior fossa arachnoiditis associated with cauda equina ependymomas. They blamed the arachnoiditis as the cause of the increased ICP. Our case demonstrated an otherwise unexplained CSF lymphocytosis. However, we are not able to compare this finding with the cases of Arseni and Maretsis or other cases reported in the literature, as CSF cytology has not generally been commented upon.

The high incidence of thoracolumbar lesions among all spinal lesions associated with papilledema may suggest a cause related to this anatomical location. It is conceivable that the mechanical blockage of CSF flow to and from the lumbar cul-de-sac may play a role in the increase of ICP and in the formation of papilledema.

Our case demonstrates a partial spinal block due to an extruded thoracic disc at the T11-12 intervertebral space associated with papilledema. The elevated protein level was presumably due to chronic epidural venous congestion secondary to pressure by the extradural herniated disc and the partial subarachnoid block. It was also associated with what seems to be an aseptic arachnoid irritative reaction. The rapid resolution of the papilledema and the return of normal visual acuity following the thoracic discectomy supports a causal mechanical relationship between the spinal and the ocular findings.

References


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