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Cranial Neuropathy due to Intradural Disc Herniation

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Abstract

Background and Importance—Herniated intervertebral disc fragments rarely penetrate the thecal sac, and intracranial hypotension attributable to such penetrating fragments is even more unusual. We describe the first reported case of a cranial neuropathy due to intradural herniation of a disc fragment, in which intracranial hypotension from a resulting cerebrospinal fluid leak caused bilateral abducens palsies.

Clinical Presentation—A 45-year-old man presented with a positional headache after having experienced a “popping” sensation in his back while lifting a heavy object. He also complained of blurred vision and was noted to have lateral gaze palsies bilaterally. Magnetic resonance imaging (MRI) of the brain revealed bilateral subdural collections, abnormal pachymeningeal enhancement, and cerebellar tonsillar herniation, suggesting intracranial hypotension. T₂-weighted MRI of the spine revealed extrusion of the T₁₂–L₁ disc and suggested the presence of a disc fragment in the intradural space, displacing the caudal nerve roots. A myelogram demonstrated a filling defect extending into the subarachnoid space adjacent to the disc herniation, consistent with a free disc fragment in the intradural space. A diagnosis of intracranial hypotension due to a cerebrospinal fluid leak resulting from an intradural herniated disc was made. The diagnosis was confirmed intraoperatively.

Conclusion—Surgical removal of the herniated disc fragment and repair of the dural defect resulted in complete resolution of the cranial neuropathy. This rare etiology of a cranial neuropathy, arising from pathology in the thoracolumbar spine, illustrates the clinical teaching that the sixth cranial nerves are highly sensitive to deformation induced by intracranial hypotension.

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Keywords

Abducens nerve palsy; Cranial neuropathy; CSF leak; Disc herniation; Herniated disc; Intracranial hypotension; Intradural disc

Background and Importance

Herniation of intervertebral discs is a common phenomenon in adult populations, and imaging studies suggest that approximately half of asymptomatic adults demonstrate radiographic evidence of lumbar disc bulges, while one quarter exhibit lumbar disc protrusions.¹ Among such cases, intradural herniated discs are rare, having an estimated incidence ranging from 4 to 150 per 10,000 disc protrusions.²⁻³ Intradural herniated discs tend to cause more severe symptoms than extradural herniations at corresponding levels, and in some instances intradural disc fragments have been mistaken at clinical presentation for tumors of the spinal cord.⁴

In a small number of cases, intradural herniated discs have been reported to cause cerebrospinal fluid (CSF) leaks, resulting in intracranial hypotension and postural headaches.⁵⁻⁶ However, no cases have been reported in which the CSF leak has caused a cranial neuropathy. Here we present the first reported case of an intradural herniated disc in which an associated dural tear and cerebrospinal fluid leak presented as a low-pressure headache and bilateral sixth-nerve palsies. Surgical repair led to complete resolution of the cranial neuropathy.

Clinical Presentation

History and Examination

A forty-five-year-old man presented to his local emergency department complaining of headache, neck pain, nausea, and vomiting. Two weeks earlier he had experienced a “popping” sensation in his back while lifting a heavy object. His symptoms resolved, and he was discharged. Several days later, however, his symptoms returned; his headache was frontal and positional. He was evaluated and noted to have lateral gaze palsies bilaterally (Fig. 1). Magnetic resonance imaging (MRI) of the brain demonstrated several signs consistent with intracranial hypotension, including small bilateral subdural collections (Fig. 2A and Fig. 2C), abnormal pachymeningeal enhancement (Fig. 2B), and caudal displacement of the brainstem with cerebellar tonsillar herniation (Fig. 2D). Sagittal T₂-weighted MRI of the thoracic and lumbar spine revealed an extrusion of the T₁₂-L₁ disc (Fig. 3A), and the axial T₂-weighted sequence suggested the presence of a free disc fragment in the intradural space at the same level, displacing the nerve roots of the cauda equina (Fig. 3B). A myelogram was performed, demonstrating a filling defect extending into the subarachnoid space adjacent to the disc herniation, consistent with the presence of a free fragment in the intradural space.

Operation

The patient underwent T₁₂-L₁ laminectomy to expose the thecal sac posteriorly at the level of the presumed dural tear. The dura was incised dorsally to reveal an intradural disc

fragment 2 cm caudal to the conus medullaris (Fig. 4A). After removal of the fragment, a 1-cm anterior dural defect was observed in the ventral side of the thecal sac (Fig. 4B), and was repaired primarily.

Postoperative Course

Postoperatively, the patient reported prompt resolution of his headaches. His lateral gaze palsies and diplopia were still present in the immediate postoperative period, but improved over the following days. By the time he was seen for follow-up examination three weeks after the operation, his lateral gaze palsies had resolved and his vision had returned to normal.

Discussion

We present the first reported case of intradural disc herniation causing a cranial neuropathy, presenting as bilateral abducens palsies. Though it is well known that spinal CSF leak and intracranial hypotension can lead to sixth nerve palsies, the mechanism underlying this phenomenon may be worth revisiting in the context of the unusual presentation reported here. Abducens palsy is commonly observed in association with changes in intracranial pressure, including both intracranial hypertension and intracranial hypotension, and the specific anatomy of the abducens nerve is thought to render it susceptible to such changes in intracranial pressure. In particular, the sixth nerve courses rostrally after it emerges from the pontomedullary junction, within the subarachnoid space between the pons and the clivus. It then pierces the dura, enters Dorello's canal (the bony groove it shares with the inferior petrosal sinus), and turns anteriorly to enter the cavernous sinus. The intra- to extradural transition and pinning of the abducens within Dorello's canal render the nerve sensitive changes in the buoyant suspension system of the brain. Such changes generate tension on the intradural side of the nerve when the brainstem shifts rostrally or caudally while the extradural side of the nerve remains tethered. In cases of intracranial hypotension due to CSF leaks, loss of CSF compromises the natural suspension system of the brain, which depends on neutral buoyancy within the fluid-filled intracranial space. Reduction of the buoyant force normally exerted on the brain by the CSF causes the brain to sag within the cranial cavity. This phenomenon may be confirmed radiographically.⁵ The sagging brain places arachnoid fibers under tension, transmitting a ventral, stretching force to the rostral periosteal dura; this tension can cause symptoms of frontal headache, as observed in the case reported here, due to sensory innervation of the dura by meningeal branches of the trigeminal nerve. Deformation of the abducens nerve as it exits the dura between the rostral clivus and the entrance to Dorello's canal, resulting from this sagging, may be responsible for the diplopia and lateral gaze palsies that often accompany intracranial hypotension.

The phenomenon of intradural disc herniation was first described by Dandy, as one of six complications in his first series of approximately three hundred operations for ruptured intervertebral discs.⁴ The mechanism by which intervertebral discs penetrate the thecal sac has not been firmly established, but a number of authors have suggested, on the basis of intraoperative observations and gross pathological examinations of retrieved disc fragments, that adhesions between the ventral dura and posterior longitudinal ligament may predispose

patients to intradural herniation²; this hypothesis is perhaps supported by the observation in one series that approximately one-third of patients with intradural herniations had previously undergone surgery for lumbar disc herniation.³

Conclusion

Penetration of the thecal sac by an intervertebral disc fragment is rare, and it is even more uncommon for a penetrating fragment to cause intracranial hypotension. We describe the first reported case of intradural disc herniation causing bilateral lateral gaze palsies. Surgical removal of the herniated disc fragment and repair of the dural defect resulted in complete resolution of the clinical deficits. This rare etiology of a cranial neuropathy lends support to the clinical teaching that the sixth cranial nerves are highly sensitive to deformation induced by intracranial hypotension.

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Figure 1.
Bilateral abducens palsies revealed by attempted left (*A*) and right (*B*) lateral gaze.

Figure 2.

MRI findings consistent with intracranial hypotension. Coronal T₂-weighted short-tau inversion recovery (STIR) (A) and axial T₂-weighted fluid-attenuated inversion recovery (FLAIR) (C) images demonstrate small bilateral subdural collections (arrows). Abnormal diffuse linear pachymeningeal enhancement (arrows) is present on the coronal post-gadolinium T₁-weighted image (B). Sagittal T₁-weighted image (D) demonstrates caudal displacement of the brainstem and mild cerebellar tonsillar herniation.

Figure 3. Sagittal T₂-weighted MRI of the thoracolumbar spine reveals an extrusion of the T₁₂-L₁ disc (A, arrow). Axial T₂-weighted imaging suggests the presence of a T₂ hyperintense, edematous free disc fragment extending intradurally at the same level (B, arrows), displacing the nerve roots of the cauda equina.

Figure 4. Intradural disc fragment among the caudal nerve roots, 2 cm caudal to the conus medullaris (A), and the corresponding 1-cm anterior dural defect (B).