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Case Report

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Sensorimotor Radiculopathy Caused by Intracranial Hypertension Treated with CSF Diversion: Case Report

BOS Diversiyonu ile Tedavi Edilen İntrakraniyal Hipertansiyona Bağlı Sensörimotor Radikülopati: Olgu Sunumu

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ABSTRACT

Idiopathic intracranial hypertension or pseudotumor cerebri is defined as increased intracranial pressure without radiologic findings of intracranial masses or obstructive hydrocephalus. Typical symptoms include headache, nausea, visual disturbances, and papilledema. In some cases, radiculopathy may present as acral paresthesias, back pain, and radicular pain. We present here an extremely rare case of sensorimotor radiculopathy caused by idiopathic intracranial hypertension; manifested by drop foot and treated with cerebrospinal fluid diversion.

Keywords: Cerebrospinal fluid diversion; Idiopathic intracranial hypertension; Pseudotumor cerebri; Radiculopathy.

ÖZET

İdiyopatik intrakraniyal hipertansiyon veya psödotümör serebri, radyolojik intrakraniyal kitle veya obstrüktif hidrosefali bulguları olmadan kafa içi basıncın artması olarak tanımlanır. Tipik semptomlar baş ağrısı, mide bulantısı, görme bozuklukları ve papilödemdir. Bazı durumlarda, radikülopati akral paresteziler, sırt ağrısı ve radiküler ağrı olarak ortaya çıkabilir. İdiyopatik intrakraniyal hipertansiyonun neden olduğu, düşük ayak ile prezente ender bir sensöri-motor radikülopati vakasını ve beyin omurilik sıvısı diversiyonu ile tedavisini sunuyoruz.

Anahtar sözcükler: Beyin omurilik sıvısı diversiyonu; İdiyopatik intrakraniyal hipertansiyon; Psödotümör serebri; Radikülopati.

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Idiopathic intracranial hypertension (IIH), also known as pseudotumor cerebri, is defined as increased intracranial pressure without radiologic findings of intracranial masses or obstructive hydrocephalus.[1,2] The most common symptoms include headache, nausea, and papilledema.[2–4] The incidence of radiculopathy as a minor sign in the form of acral paresthesias, back, and radicular pain was reported to be less than 15%.[3] We herein present a unique case of sensorimotor radiculopathy caused by IIH and treated with cerebrospinal fluid (CSF) diversion.

Case Report

A 38-year-old obese female patient presented to our neurosurgery clinic with pain in her back and left leg that had been present for 15 days. She had been diagnosed with IIH 2 years ago and had been taking acetazolamide. On physical examination, the patient had a positive Lasegue test at 20 degrees on the left side, indicating compression of the nerve root. Dorsiflexion muscle strength of the left foot was 2/5. According to the patient, this weakness began

at the same time as the leg pain. Deep tendon reflexes were normal, and the cranial nerves were intact. Electromyography was performed, which showed nonspecific chronic subacute involvement of the left L5 nerve root. Magnetic resonance imaging (MRI) of the lumbar spine revealed an L4-L5 disk protrusion, after which the patient underwent 10 sessions of physiotherapy (Fig. 1). The patient showed no improvement in her motor symptoms but also complained of severe headaches and visual disturbances. The neurologic examination revealed bilateral papilledema. Cranial MRI findings were nonspecific with normal sella turcica and normal appearance of the optic nerve; only slit-like ventricles were seen (Fig. 2). MR venography showed a narrowing of the right transverse sinus (Fig. 3). To test whether the patient would respond to CSF drainage, the tap test was performed 1 month after the onset of her symptoms. A lumbar puncture was performed; with the opening pressure measured at 420 mm H_2O; and 30 ml of CSF was collected. After this first puncture, her back pain was relieved, and the motor deficit in the dorsiflexion muscle strength of the left foot improved to 3/5. Two days later, a second puncture was performed with an opening pressure of 320 mm H_2O and a CSF collection of 35 ml. Her headaches and the pain in her left leg had disappeared. A lumbo-peritoneal shunt (LPS) was placed, the motor radiculopathy improved immediately to a muscle strength of 4/5. Her visual disturbances had also disappeared. The patient achieved full muscle strength on the 2nd postoperative day. No concordant pathologies were found at the 2-year follow-up.

Discussion

IIH, also known as pseudotumor cerebri, is characterized by increased intracranial pressure without localized neurological symptoms and radiological findings.[1] IIH is more common in obese young women, as shown in a large prospective study of untreated patients.[4] Headache was the most common symptom (84%), while transient visual disturbances occurred in 68% and pulsatile tinnitus in 52% of patients.[5] The incidence of radiculopathy as a minor sign was less than 15%.[4] Radiculopathy due to IIH had different manifestations; usually sacral paresthesias, neck stiffness, distal extremity paresthesias, back, and radicular pain.[1,5,6] Previous studies have reported IIH-induced radiculopathy only in the context of radicular pain. The uniqueness of our case is the presence of radicular pain in combination with the motor deficit, a manifestation not previously reported in the literature.

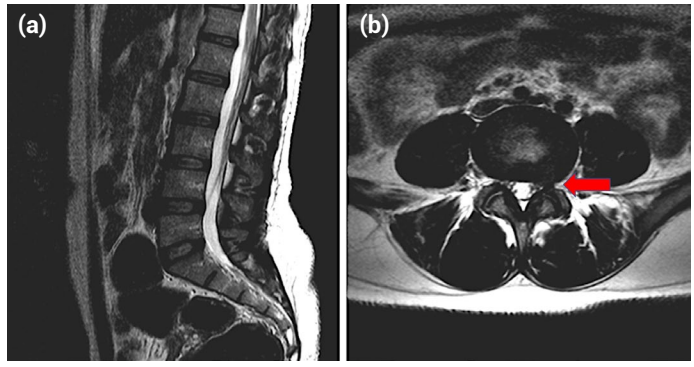

Figure 1. Lumbar MRI sagittal **(a)** and axial **(b)** view: Left L4-L5 disc protrusion (arrow).

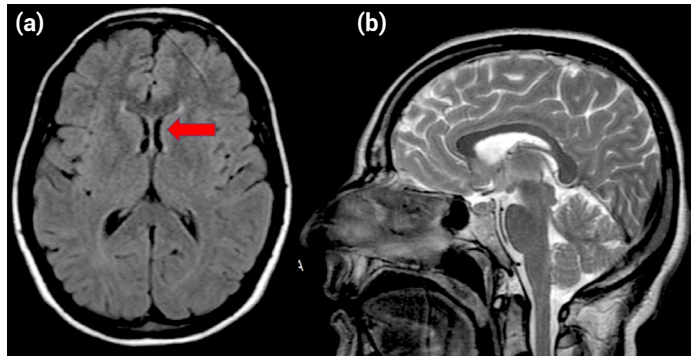

Figure 2. Cranial MRI axial **(a)** and sagittal **(b)** view: Normal sella tursica and normal appearance of the optic nerve. Axial T1-weighted MRI shows slit-like ventricles (arrow).

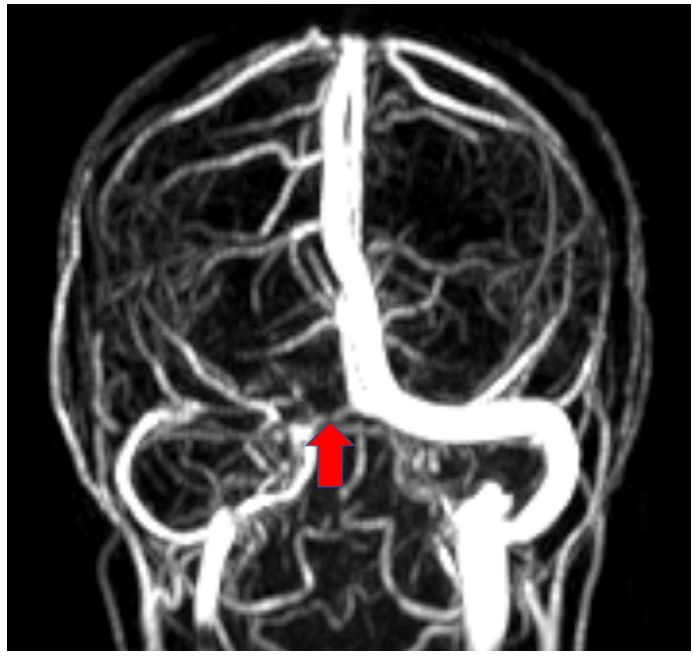

Figure 3. Cranial MR venography. A narrowed right transverse sinus (arrow).

Bortoluzzi et al.[7] presented an obese 34-year-old IIH patient with polyradiculopathy who had impaired CSF flow and absorption demonstrated by cisternography in the set-

ting of an unusual enlargement of the subarachnoid space below L2, with immediate relief of spinal pain achieved by insertion of a LPS as in our case. Murray et al.[8] presented a 4-year-old patient with radicular pain and hypoactive cervical deep tendon reflexes due to IIH, which was treated by lowering the intracranial pressure, resulting in the disappearance of the symptoms. In parallel, Obeid et al.[9] reported two patients with papilledema, marked visual impairment, and flaccid quadriparesis due to IIH who were misdiagnosed as Guillain-Barre syndrome; and given intravenous immunoglobulin, but this was of no benefit as the symptoms disappeared in both patients after LPS surgery. Another case of IIH causing radiculopathy with signs of electrodiagnostic changes returned to normal responses after CSF diversion via a ventriculoperitoneal shunt.[10] Finally, Ragab et al.[11] presented a 17-year-old female patient with acute onset of quadriparesis, headache, and total ophthalmoplegia due to IIH, where LPS surgery resulted in significant recovery.

Hulens et al.[12] suggested draining the CSF to provide temporary relief for patients with unexplained widespread pain and fibromyalgia.[13] Increased CSF pressure irritates the nerve fibers in the nerve root leaflets and causes radicular pain. Dysregulation of CSF pressure can also cause radicular pain via the same pathophysiological pathway.[9] The mechanism of radiculopathy and cranial neuropathies due to increased intracranial pressure is thought to be similar: mechanical compression of the nerve roots due to increased CSF pressure in the subarachnoid space or impaired venous outflow through the radicular veins leading to decreased blood flow to the most vulnerable parts of the nerve root causes ischemia.[7,14] In addition, radicular venous compression impairs the clearance of inflammatory mediators such as substance P, vasoactive intestinal peptide, and calcitonin gene-related peptide, causing progressive intraneural edema.[13]

Initial treatment options for IIH are weight loss or dietary changes and exercise, medications such as acetazolamide and topiramate. Serial lumbar punctures are effective for diagnosis by measuring CSF pressure, while clear reference cut-off values for CSF opening pressure remain a matter of debate. In general, there is a consensus that an opening pressure of >200 mm H_2O in non-obese patients and >250 mm H_2O in obese patients together with symptoms of increased intracranial pressure is sufficient for diagnosis.[15] Severe, refractory, or progressive symptoms are considered for surgical treatment: CSF flow diversion (shunt), stenting of the venous sinus in case of thrombosis, or fenestration of the optic nerve. Diverting the CSF flow through a shunt, as in our patient, to reduce the pressure led to a significant improvement of clinical symptoms, so that surgical placement of a shunt system is nowadays the first surgical approach.[16]

Conclusion

It has been emphasized that mislocalized minor signs can be indicators of serious pathology of the neural pathways, their perception, and the situations in which they occur, facilitating appropriate and timely research and treatment. Finding the underlying cause is crucial for appropriate treatment. In patients with radiculopathy associated with headache and visual disturbances, IIH should be considered. With increased intracranial pressure, enlargement of the spinal subarachnoid space surrounding the nerve roots leads to radicular pain and even motor deficits. Therefore, careful analysis of symptoms and assessment of the CSF pressure should help to identify the cause of the radiculopathy.

Disclosures

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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