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A case of lumbar intraspinal schwannoma presenting as positional headache

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ABSTRACT

The association between spinal tumors with hydrocephalus and intracranial hypertension is rare, and the exact mechanism has remained uncertain. We present a case of L4/5 intraspinal schwannoma presenting as positional headache, with the aim to discuss the underlying pathophysiology and alert clinicians of the possibility of lower spinal tumor as a differential diagnosis of headache.

Keywords: spinal tumor, intracranial hypertension, hydrocephalus, positional headache

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Introduction

Although there have been few reports of benign spinal tumors associated with hydrocephalus and increased intracranial pressure(IICP) [1, 2], to the best of our knowledge there have been no reported case of positional headache as the presenting symptom of lower lumbar spinal tumor. It is easy to comprehend that in neoplasms of the upper cervical canal or craniovertebral junction, increased intracranial pressure may be caused by mechanical obstruction of the spinal fluid pathway [3, 4]. However, the causes of the pressure changes are less clear when the tumor is situated in the lower spine. Herein, we report a case of L4/5 intraspinal schwannoma presenting as positional headache and discuss the underlying pathophysiology.

Case Presentation

A 68-year-old man presented as positional headache for more than one year. The character of headache was dull and pressure-like in the whole cranium. The headache was aggravated by sitting for more than half hour, and could be relieved by lying down or standing up. Otherwise, the pain was not associated with nausea, vomiting, dizziness, gait disturbance or visual obscurations. There was no recent history of trauma, surgery or infection. However, about one year prior the presentation, he had experienced temporary back pain for weeks with spontaneous recovery, and lumbar spondylosis was diagnosed at local clinic then.

The patient had first presented to a neurologist. Physical examinations were normal except hyporeflexia of knee jerk. He received brain magnetic resonance imaging (MRI), which revealed borderline ventriculomegaly (Fig.1). In addition, a spine MRI was also performed, showing an intradual extramedullary tumor, about 2.5cm in diameter, at L4/5 level (Fig.2). Finally, the patient received operation and complete tumor removal, after which the positional headache was immediately alleviated. The tumor was later proven pathologically to be a schwannoma.

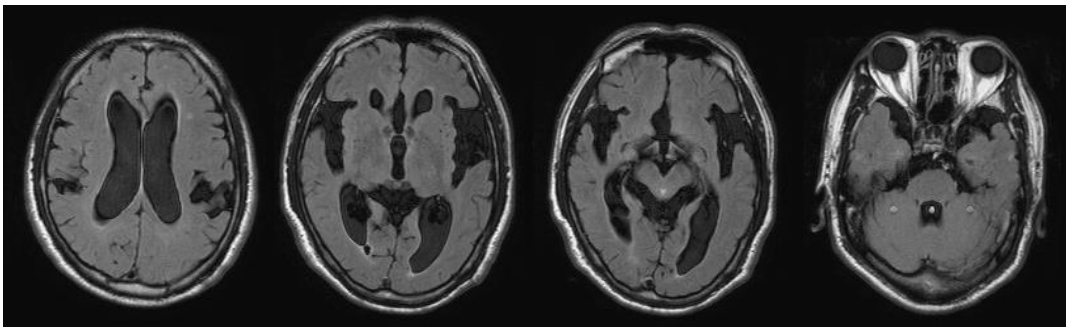

Fig.1. FLAIR axial MRI revealed borderline ventriculomegaly

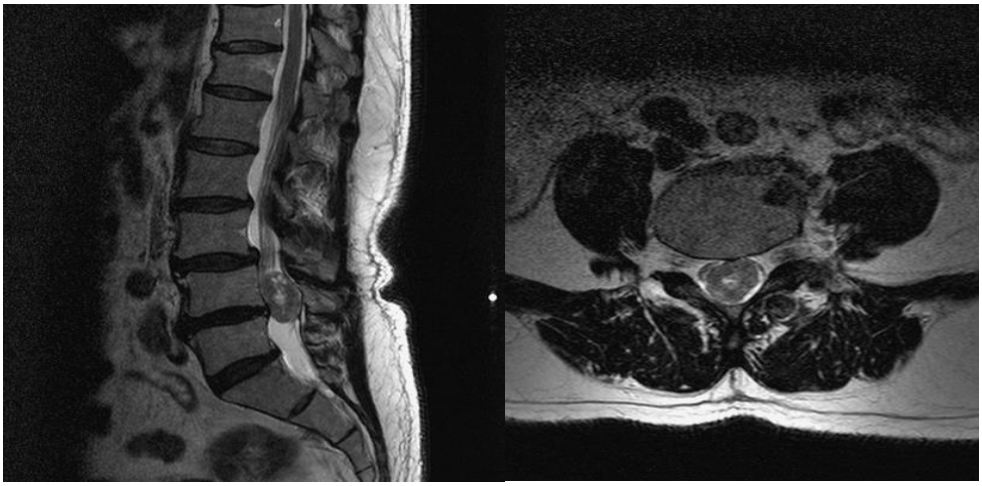

Fig.2. Lumbar MRI demonstrated an intradual extramedullary tumor at L4/5 level

Discussion

To date, various mechanisms have been proposed to explain the underlying pathophysiology of IICP in cases with benign spinal tumors. Some authors believe that spinal tumors with excessive protein secretion accounts for increased CSF viscosity, which in return decreases CSF absorption at the arachnoid villi and retards CSF flow. However, not all reports of hydrocephalus in patients with spinal tumors are associated with high protein levels [1]. Arachnoiditis may be another factor, possibly due to tumor releasing products, such as fibrinogen and inflammatory cytokine transforming growth factor (TGF)-β, subarachnoid bleeding from the tumor, or intracranial seeding, which may cause irritation of the arachnoid membrane [5]. Others proposed the hydrodynamic theory. The lumbar portion of the spinal sac serves as reservoir for the compensation of CSF flow changes. When a spinal lesion blocks the subarachnoid space, this reservoir function eliminates, and thus ventricular dilatation or raised intracranial pressure might develop. Moreover, high pulse pressure alone has been reported to create ventriculomegaly by a “water hammer effect” [3]. Another explanation is that compression of the spinal venous plexus may increase the intracranial venous pressure, which contributes to intracranial hypertension [2].

In our case, the immediate improvement in headache after surgery suggested an immediate improvement in intracranial pressure and made increased CSF viscosity or inflammatory etiologies unlikely deciding factors. We therefore suspect the underlying pathophysiology to be predominantly related to the dynamic theory. During systole, the increase in intracranial CSF volume is dissipated by a cranio-caudal CSF flow into the spinal subarachnoid space, and a reversal of CSF flow direction occurs in diastole. The compliance of the spinal compartment therefore plays a crucial role in the CSF circulation. The spinal compartment compliance is in turn based on an increased elasticity of the spinal dura mater, a wide epidural space with an extended compressible epidural venous plexus, and sub-atmospheric epidural pressure. A benign spinal tumor could therefore decrease the spinal compartment compliance through obstruction of subarachnoid space, compress the epidural venous plexus, and cause an increase in intracranial pressure. Under such conditions, other factors such as changes to CSF protein concentration or CSF flow resistance due to arachnoiditis may potentially have added effects on CSF dynamics. We also speculate that in our case, the positional nature of the headache is related to the significant size and lower lumbar location of the tumor, which resulted in diminished spinal subarachnoid space during kyphosis.

Conclusion

Although benign lower spinal tumors associated with headache or intracranial hypertension are not commonly encountered, this possibility should be kept in mind as a differential diagnosis, especially if the patient also presented with concomitant upper or lower motor neuron signs. Detail neurological and neuroradiological investigations should be performed to ensure correct diagnosis and optimal treatment.

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