Cerebral venous sinus expansion in post-lumbar puncture headache

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Accepted for publication: October 10, 2003

Summary

Cerebrospinal fluid (CSF) leak at the lumbar puncture (LP) site may induce intracranial hypotension, a common cause of post-LP headache (LPH). We present the case of an 18-year-old man who developed a severe and continuous positional headache 24 hours after LP. CT scan and MRI showed abnormal, intense, dural venous sinus enhancement, indicating a compensatory venous expansion. The patient recovered fully within a fortnight. This report supports the hypothesis that venodilation may be involved in the pathogenesis of LPH. Brain MRI, to detect possible dural venous sinus abnormalities, should therefore be performed in patients with severe and prolonged LPH, as this may allow a prompt recognition and treatment of this disturbing condition.

KEY WORDS: CT scan, dural venous sinus, headache, lumbar puncture, MRI.

Introduction

Lumbar puncture (LP) is a relatively safe procedure and is routinely performed during the diagnostic work-up of a broad spectrum of neurological disorders. The most common complication is post-LP headache (LPH), which is estimated to occur in 0.4-70% of cases, depending on the technique used, age of the patient and the size of needle (1,2). The headache usually begins within 24-48 hours of LP and is postural, the pain being relieved upon reclining and worsened by sitting or standing. Symptoms often include other disabling features, such as nausea, tinnitus and blurred vision. The pathogenesis of the LPH may be related to CSF hypotension, which has been suggested to induce acute distension of the intracranial veins and pain (3,4). A recent report describes a case of LPH and MRI evidence of abnormal dural meningeal and dural venous sinus enhancement (5).

In this report, we present a patient with LPH in whom non-invasive imaging (CT and MRI) showed dural venous sinus enhancement, suggesting compensatory venodilation. Thus, our case further supports the hypothesis that CSF hypotension and acute vein expansion may play a role in the development of LPH.

Case report

An 18-year-old man was admitted to our hospital in January 2003 with a several-day history of gait disturbance and dysarthria. Onset of these symptoms had occurred following a week of mild fever, cough and pharyngodynia. His family and medical history was negative. Neurological examination showed mild gait and limb ataxia and dysarthria. An infection of the nervous system was suspected, and a brain magnetic resonance imaging (MRI, 0.5 T unit), with and without *i.v.* Gd-DTPA (gadolinium diethylene triamine penta-acetic acid) contrast injection, was performed that did not show pathological signal enhancements (Fig. 1A). He then underwent an extensive blood, serum and urine biochemical work-up that was within normal ranges.

A LP, performed in the sitting position using a 20G Quincke needle (Artsana, Como, Italy), revealed clear CSF with a normal opening pressure, seven cells, normal protein and glucose levels, and unremarkable routine cultures. No oligoclonal bands were found.

The patient was treated with corticosteroids (betamethasone, 8 mg/day) which led to a rapid and significant improvement of his ataxia and dysarthria. Some 48 hours after the LP, however, he developed a post-LP headache, which rapidly progressed to a severe and continuous positional headache. The pain was relieved upon reclining and worsened by sitting or standing. The patient also complained of tinnitus in the right ear and nausea. He continued the corticosteroid therapy, and also received common analgesics without significant benefit. EEG was normal.

A non-contrast head CT scan revealed diffuse dural venous sinus hyperintensity, which was more evident in the sagittal and transverse sinus (not shown). Brain MRI scans, performed six days after the LP, demonstrated abnormal, intense enhancement and expansion of the cerebral venous sinus (Fig. 1B). Sagittal and coronal images of the brainstem and cerebellar tonsils (not shown) did not show pathological displacements. MR angiography allowed us to exclude venous sinus thrombosis. A diagnosis of LPH was thus made; an epidural blood patch therapy was offered, which the patient refused. He was therefore invited to maintain a clinostatic position and was treated with low doses of heparin. The corticosteroids were discontinued. The clinical outcome was favourable with a full recovery within 15 days.

Discussion

This case demonstrates that cerebral venous sinus dilation, evidenced by MRI, can be a feature of LPH. This association underlines the importance of performing non-invasive imaging studies in patients with LPH. Interestingly, while uncomplicated LP alone is unlikely to cause abnormal meningeal/venous sinus enhancement on MRI (6), the frequency of dural venous dilation in moderate LPH, a condition that often occurs in adults, is still unknown. It is to be noted, however, that long-lasting LPH can be associated with Gd-DTPA meningeal enhancement (7).

The pathophysiological relationship between LPH, venous sinus expansion and intracranial hypotension has still to be clearly defined. Recent evidence indicates that lumbar puncture increases the compliance at the caudal end of the CSF compartment, thus leading to fluid displacement downward (8). In susceptible individuals, as may be the case of our patient, this mechanism may lead to intracranial hypotension which, in turn, induces a compensatory dural sinus dilation with venous engorgement (5,8-11). This has been suggested to be the cause of the pain (4), even though other factors may also play a role (e.g., stretching of sensitive cranial nerves, such as V-IX-X, and/or pachymeninges).

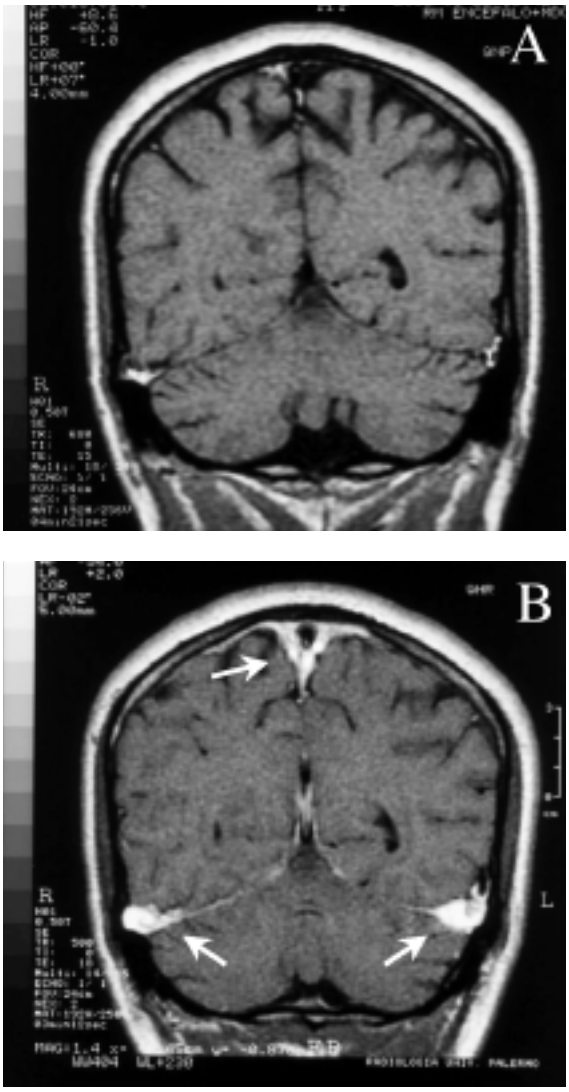

Figure 1 - Coronal post-contrast MRIT1WI (0.5T) before (A) and one week after onset of LP (B). Note the meningeal enhancement, involving the tentorium. The abnormal dilatation of the transverse and sigittal superior sinus is marked by arrows.

The post-LP brain MRI in our patient did not show parenchymal abnormalities, with the notable exception of a dural sinus expansion. In the case reported by Bakshi et al. (1999), MRI demonstrated only a slight downward displacement of the cerebellar tonsils (5). It is likely that our case documents a mild condition of intracranial hypotension, without the appearance of brain displacement and/or brainstem sagging.

In conclusion, the presence of a venous sinus enhancement on MRI, indirectly suggesting the presence of intracranial hypotension (5), can be considered the imaging marker of severe and continuous LPH. Our case report thus adds to the very limited literature on this topic, and supports the hypothesis that intracranial hypotension-associated venous dilatation might be causally related to LPH.

To improve recognition and allow prompt treatment of this disturbing condition, MRI investigations, to detect meningeal and venous sinus abnormalities, should therefore be performed in any patient presenting severe and prolonged LPH.

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