Brain stem and cerebellar dysfunction after lumbar spinal fluid drainage: case report

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Lumbar spinal fluid drainage is a common procedure to reduce the risks of cerebrospinal fluid (CSF) fistula after skull base fractures or various transdural neurosurgical procedures. Nevertheless, this simple and effective technique can lead to overdrainage and CSF hypovolaemia. This report describes the case of a young patient who had a lumbar drain inserted, to avoid CSF fistula after a pterional craniotomy with opening of the frontal sinus for the clipping of a ruptured aneurysm. The drain was removed after 48 hours because of underdrainage (<1 ml/h). Three days after drain removal, she developed rapid deterioration of her level of consciousness and signs of cranial nerves involvement, brain stem and cerebellar dysfunction. Intracranial pressure was low (<5 cm H2O) and MRI showed brain sagging and cerebellar foramen magnum herniation. The patient was successfully treated with epidural blood patch, ventricular drainage, and Trendelenburg position. The authors report this case because CSF hypovolaemia attributable to lumbar overdrainage is an insidious and threatening condition not easy to diagnose in the absence of detectable CSF leak. MRI and intracranial pressure monitoring confirm the diagnosis and permit better understanding of the physiopathology of brain sagging.

Cerebrospinal fluid (CSF) leakage is a classic neurosurgical complication after cerebral or spinal transdural surgery or after fracture of the skull base. Continuous CSF lumbar drainage has gained wide acceptance to prevent and treat such fistulas, as it decreases the pressure gradient across the fistula, and offers an attractive alternative to surgical revision that exposes the patient to anaesthetic and procedural risks. One has, however, to be aware of the risks related to this simple and effective procedure. To illustrate the severe complications that can be associated with lumbar drainage, we present a case of brain stem and cerebellar dysfunction attributable to CSF hypovolaemia that developed several days after a lumbar drain had been removed. We discuss the concept of CSF hypovolaemia and propose therapeutic strategies.

CASE REPORT
A 38 year old healthy woman was admitted for a Fisher grade II subarachnoid haemorrhage attributable to a ruptured carotid artery bifurcation aneurysm, without hydrocephalus. Neurological examination was normal except for meningeval signs.

A pterional craniotomy was performed to clip the aneurysm. Because of extensive pneumatisation of the frontal skull base, the frontal sinus was opened during craniotomy. To prevent the development of a CSF fistula, the frontal sinus was cranialised and a lumbar drainage was inserted. Two puncture attempts with an 18 gauge needle were needed to place the drain intradurally.

The patient had normal postoperative neurological examination. Drainage through the lumbar catheter was minimal (<1 ml/h), for this reason it was removed the second postoperative day. Three days after drain removal, she developed headache and drowsiness. On examination, she had difficulties swallowing, absent gag reflex, VIth nerve palsy on the left side, and multidirectional nystagmus.

Cerebral computed tomography showed small lateral ventricles, grey-white matter differentiation in the posterior fossa, and descent of the cerebellar tonsils into the occipital foramen. Cerebral angiogram showed complete exclusion of the aneurysm as well as the absence of cerebral vasospasm.

Intracranial hypotension was in the differential diagnosis, however there was no clinical evidence of CSF leakage. Cerebral MRI (fig 1) confirmed the presence of tonsillar herniation through the foramen magnum and tentorial herniation of the splenium of the corpus callosum and the cingular gyrus. The transtentorial herniation reaches a line drawn tangential to the frontal cranial base. The thin arrow shows the tonsillar herniation through the foramen magnum. The tip of the tonsils descends to the upper part of the arch of C1 as indicated by the lower line (drawn parallel to the upper line). Note also the disappearance of the posterior fossa cisterns and the fourth ventricle.

By that time, the patient was lethargic and was intubated. Intracranial pressure monitoring confirm the diagnosis and permit better understanding of the physiopathology of brain sagging.

Figure 1 T1 gadolinium enhanced mid-sagittal cerebral MRI shows the sagittal sinus without meningeval enhancement. The large arrow shows the tentorial herniation of the splenium of the corpus callosum and the cingular gyrus. The transtentorial herniation reaches a line drawn tangential to the frontal cranial base. The thin arrow shows the tonsillar herniation through the foramen magnum. The tip of the tonsils descends to the upper part of the arch of C1 as indicated by the lower line (drawn parallel to the upper line). Note also the disappearance of the posterior fossa cisterns and the fourth ventricle.
pressure was low (<5 cm H2O). This information confirmed the diagnosis of intracranial hypotension. Even though no CSF leakage or collection was detectable in the lumbar area, the most probable cause was an occult spinal fluid fistula consecutive to the lumbar drain punctures.

To promote dural repair, we performed a lumbar epidural blood patch at the site of puncture. To help posterior fossa structures to raise back to their normal position and to establish CSF normovolaemia (as we continued to drain CSF from the ventricle), but to invert the pressure gradient between the cranial and spinal compartments. This is why we believe that patients are at higher risk to develop pressure gradients between the cranial and spinal compartment, and therefore brain sagging after lumbar drainage, than after ventricular drainage. Mokri’s review on CSF hypovolaemia supports this, as brain sagging was described only in patients with spinal CSF leaks, and never in patients with cranial CSF leaks.3 The dramatic improvement obtained in our patient by combining external ventricular drainage and head down positioning further supports this hypothesis. The clinical recovery was not related to reestablishment of CSF normovolaemia (as we continued to drain CSF from the ventricle), but to the inversion of CSF dynamics between the spinal and the cranial compartments.

Treatment of spinal dural leaks is often successful after one or more epidural patches; if this fails, surgery to repair the dural defect may be necessary.10 11 In the presence of progressive brain sagging, symptoms may become severe and life threatening. In such situation, we recommend the addition of Trendelenburg position and external ventricular CSF drainage to invert the pressure gradient between the spinal and cranial compartment and to decrease the pressure at the level of the fistula.
Furthermore, measurement of low intracranial pressures with the ventricular drain confirms the diagnosis and permits monitoring during the Trendelenburg positioning of a neurologically threatened patient. In conclusion the use of lumbar spinal drainage is a procedure that deserves special attention and careful monitoring as it may be associated with life threatening CSF overdrainage or leak, even after removal of the lumbar drain. The risk of dural tear and subsequent occult CSF fistula is increased after multiple puncture attempts to insert the drain. A therapeutic strategy, combining Trendelenburg positioning and external ventricular drainage was successfully applied in one patient presenting severe life threatening brain sagging attributable to occult CSF leak.

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