Intracranial hypotension caused by leakage of cerebrospinal fluid from the thecal sac after lumboperitoneal shunt placement

Case report

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The authors describe a newly recognized complication of lumboperitoneal (LP) shunt placement, namely, intracranial hypotension from leakage of cerebrospinal fluid (CSF) through a defect in the lumbar dura created by the shunt catheter. They report on a 47-year-old obese woman with idiopathic intracranial hypertension who underwent routine placement of an LP shunt. Following surgery, her headache became worse. Two radionuclide shunt studies showed no anterograde tracer flow, suggesting either obstruction or a leak. After shunt reservoir manometry indicated low pressure, spinal magnetic resonance (MR) imaging was performed. The MR images revealed a CSF leak from the lumbar thecal sac. A computed tomography (CT) myelogram, performed by injection into the shunt reservoir, confirmed the presence of a leak by showing extravasation of contrast agent into the epidural space. The patient was treated by application of a CT-guided blood patch at the leak site. Catheter-associated CSF leak is an unusual cause of intracranial hypotension that can occur following LP shunt placement. This case report outlines the clinical features of this condition, documents the neuroradiological findings, and demonstrates successful treatment with a blood patch.

Key Words • blood patch • idiopathic intracranial hypertension • papilledema • pseudotumor cerebri • radionuclide shuntogram

When weight loss and carbonic anhydrase inhibitors fail in the treatment of patients with IIH, surgical intervention should not be delayed. The operations performed most commonly are optic nerve sheath fenestration and LP shunt placement. The latter procedure has the advantages of lowering ICP and relieving papilledema, whereas optic nerve sheath fenestration does not reduce ICP. In principle, therefore, LP shunt placement accomplishes both of one’s major therapeutic goals: preventing vision loss and curing headache. Unfortunately, some patients continue to report headache after LP shunt placement. In these patients, the diagnostic challenge is to determine why headache persists.

We treated a patient with IIH who had severe, intractable headache after uneventful placement of an LP shunt. The patient’s headache was due to a newly recognized complication, namely, leakage of CSF from the lumbar thecal sac caused by the shunt catheter. Here we document the radiological appearance of CSF leak associated with an LP shunt catheter and describe the successful application of an epidural blood patch.

Case Report

History and Presentation. This 47-year-old woman developed headache, papilledema, and blurred vision. The results of an MR imaging study were normal. Elevated ICP was documented on numerous occasions by measurement of the lumbar subarachnoid opening pressure. Analysis of the CSF was normal. Idiopathic intracranial hypertension was diagnosed in the patient and her symptoms were controlled with acetazolamide and analgesics for several years. She was eventually referred to our institution for LP shunt placement because of advancing peripheral visual field loss and medically refractory headache.

Examination. The patient weighed 90 kg and her height was 165 cm, for a body mass index of 33.1 (obese). Her visual acuity was 20/20 in each eye. Computerized Humphrey
visual fields showed peripheral constriction and enlarged blind spots in the visual fields of both eyes. Chronic papilledema and early optic atrophy were observed on fundus inspection. The results of the neurological examination were otherwise normal.

First Operation and Postoperative Course. An LP shunt was placed without difficulty, employing a horizontal-vertical lumbar valve model #903-330A with a horizontal valve closing pressure of 50 to 80 mm H2O and a vertical valve closing pressure of 230 to 320 mm H2O (Integra Neurosciences). During laparoscopically guided insertion of the abdominal end of the catheter, multiple intraabdominal adhesions were noted, including one from the liver to the anterior abdominal wall. To avoid this adhesion, the catheter was placed closer than usual to the midline.

Within a few days after surgery, the patient reported worsening headache, which required treatment with acetaminophen and hydrocodone. On the fifth day after surgery, an abdominal radiograph (Fig. 1) was obtained to assess the integrity of the shunt. The radiograph showed no evidence of shunt migration or disconnection. Two days later a radionuclide study was performed by injecting ⁹⁹mTc into the shunt reservoir. Dynamic imaging over the next 15 minutes showed no ⁹⁹mTc flow from the reservoir into the abdomen. Instead, there was reflux of tracer from the reservoir through the catheter toward the lumbar spine. The results of the study were interpreted as evidence of shunt obstruction between the reservoir and the abdomen, resulting from mal-function of the valve, blockage of the catheter tubing, or occlusion of the abdominal opening by one of the adhesions seen during shunt placement.

Second Operation and Postoperative Course. Nine days after shunt placement the patient was taken back to the operating room. The flank incision was reopened to remove the reservoir and valve. The free end of the lumbar catheter was placed in a dependent position, resulting in a slow trickle of CSF. From this observation it was concluded that the portion of the LP shunt from the lumbar spine to the shunt reservoir was functioning properly. A new reservoir and valve were installed, and the portion of the shunt from the valve to the peritoneal cavity was replaced. The free end of the catheter in the abdomen was noted by laparoscopy to be positioned satisfactorily. The patient was discharged from the hospital 5 days later, with limited improvement in her headache.

Further Examination. A week later the patient was re-admitted to the hospital because of worsening headache, which could not be controlled by hydromorphone and topiramate. Another ⁹⁹mTc shuntogram was performed (Fig. 2). As before, the study showed no anterograde flow from the reservoir to the abdomen. At this point, the pressure in the reservoir was measured directly with a manometer. The pressure was 0 to 4 mm H2O with the patient supine, and fell to 9 to 12 mm H2O when she sat up at a 45° angle. These values were unexpectedly low, alerting us to the possibility of intracranial hypotension. An MR imaging study of the brain showed bilateral subdural fluid collections (Fig. 3), supporting the diagnosis of postoperative intracranial hypotension. There was no evidence of an acquired Chiari malformation type I, a complication associated with LP shunts.¹²

A spinal MR imaging study was performed to search for
a leakage site. It demonstrated the expected course of the LP shunt catheter. The shunt pierced the dura mater dorsally, entered the subarachnoid space at the L2–3 level, and ascended parallel to the ventral aspect of the spinal cord. There was an epidural fluid collection located ventrally at the level of the LP shunt entry into the thecal sac (Fig. 4A and B). To pinpoint the locus of the apparent CSF leak, a CT myelogram was performed with injection of a contrast agent into the shunt reservoir. The injection led immediately to focal epidural extravasation of contrast material at the L2–3 level, with subsequent pooling of the contrast agent in the ventral epidural space (Fig. 4C and D).

These imaging studies demonstrated the presence of a leak in the thecal sac at the level where the shunt was inserted, but the contrast material seemed to emanate from the ventral, rather than the dorsal, side of the thecal sac. It was unclear whether the leak arose from the actual catheter insertion site, located dorsally, or whether a separate hole was present in the ventral dura mater. Such a hole might have been caused by inadvertent puncture of the dura during the shunt insertion or by erosion of the catheter through the dura. In any event, the leakage of CSF and contrast agent seen on the spinal studies explained the patient’s headache. It also explained the lack of 99mTc clearance from the reservoir on both shunt studies. The leak reduced the pressure gradient for anterograde tracer flow from the reservoir to the abdomen.

Additional Treatment and Posttreatment Course. To close the leak, a 15 cc blood patch was administered. The patient reported only transient improvement in her headache. Three days later the procedure was repeated with 28 cc of blood, again without permanent relief. These unsuccessful patches were applied at the bedside without the benefit of neuroradiological guidance. A third blood patch was applied, on this occasion using CT to confirm that the needle was located in the L2–3 epidural space near the leak site (Fig. 5). The patch was delivered by injecting 5 cc of blood mixed with contrast agent. After the injection was made, CT images showed formation of a blood clot and compression of the thecal sac.

The next day the patient reported nearly complete resolution of her headache. She was instructed to remain in bed for 2 days. Four days after application of the third blood patch she was discharged from the hospital in good condition. A year later the patient reported no recurrence of headache on a routine office visit. A neuroophthalmological examination showed no visual field loss or papilledema.

Discussion

Headache is a common symptom in patients with IIH who require LP shunt placement. Paradoxically, headache is also one of the most frequent symptoms experienced after surgery. In a retrospective study involving 82 patients with IIH whose elevated ICP had been normalized by medication or surgery, 68% subsequently developed a new headache syndrome, consisting of episodic tension-type headache (30%), migraine without aura (20%), chronic tension-type headache (10%), or analgesic overuse headache (8%). In another retrospective study of 42 patients with surgically treated IIH, severe headache recurred in 48%, despite a properly functioning shunt. These data suggest that patients with a history of IIH are predisposed to headache, even after successful placement of a LP shunt.
Faced with the dilemma of a patient with postoperative headache, the neurosurgeon’s main responsibility is to make sure that the LP shunt is functioning properly. Once infection has been excluded, the principal concerns are obstruction or excessive shunting. Any of these shunt complications can produce postoperative headache. In every case series, obstruction remains the most common cause of shunt failure. The problem of excessive shunting has become less prevalent, owing to the routine use of valves with two pressure settings (for erect and recumbent postures). However, it can still occur, even when these devices are used.

A radionuclide shuntogram is useful to differentiate between obstruction and excessive shunting, but results must be interpreted cautiously because testing protocols, normal values, and rates of false positive or negative results vary widely among clinical centers. It should also be emphasized that published data pertain almost exclusively to ventriculoperitoneal shunts, rather than LP shunts. In principle, overdrainage should be easy to recognize because of rapid clearance of the tracer from the shunt reservoir and early spillage into the abdominal cavity. However, tracer clearance can occur rapidly in a patient with a normally functioning shunt, especially if a large tracer volume is administered or the patient is crying or upright during the procedure. For this reason, a radionuclide study cannot always differentiate reliably between a shunt that is overdraining and one that is working properly.

In our patient, the lack of anterograde flow of 99mTc from the shunt reservoir to the abdomen in the first shuntogram was interpreted as evidence for shunt obstruction. Had distal obstruction been present, direct measurement of the pressure in the reservoir would have yielded an elevated reading. Unfortunately, we did not perform a pressure measurement because the results of the shuntogram convinced us that the shunt was obstructed. To our surprise, after replacement of the distal shunt apparatus, a follow-up shuntogram still showed no anterograde flow. At this point, the pressure in the reservoir was measured. The low-pressure reading altered our interpretation of the findings of the shunt study.

A low pressure in the shunt reservoir without anterograde tracer flow is compatible with either shunt breakage or proximal obstruction between the lumbar region and the shunt reservoir. With proximal obstruction, little retrograde flow of tracer should occur from the shunt reservoir because the catheter is blocked. However, some degree of retrograde flow may be detected due to 99mTc reflux into the catheter, especially if a large volume of tracer is injected under sufficient pressure. In our patient, proximal obstruction was deemed unlikely because CSF had been observed to trickle from the open end of the catheter when it was disconnected from the reservoir during the shunt revision procedure.

A single prior case has been reported of a patient with severe postural headache following LP shunt placement, due to a probable CSF leak from the thecal sac. Neuroimaging was not performed in that case, but the patient was treated empirically with a blood patch, resulting in immediate relief. We now report a second case, this time proving the existence of a CSF leak by neuroimaging. The complication of fluid leakage from the thecal sac probably occurs more often than realized after LP shunt placement, but it has probably been confused with a low-pressure syndrome resulting from excessive shunting. Wider recognition that a low-pressure syndrome can also occur from chronic leakage of CSF in the lumbar region may help avoid unnecessary shunt removal or revision. A simple blood patch can suffice to treat this complication. As we have shown, CT can facilitate the application of the blood patch to ensure that it is delivered to the actual leak site.

After routine lumbar puncture, CSF continues to drain into the paraspinal area for several days before the dural puncture eventually seals. In our patient, a 14-gauge Tuohy needle was used to pierce the dura and a 1.6-mm (outside diameter) catheter was threaded into the subarachnoid space. For the defect to close, a watertight seal must develop between the dura and the shunt catheter. Given this requirement, it is rather surprising that a permanent leak does not occur more commonly after LP shunt placement. The fact that the paraspinal area can absorb enough CSF to restore ICP to normal in patients with IIH raises the possibilit...
ity that in the future one might invent a controlled drainage device for implantation in the lumbar dura mater that does not require the shunting of fluid to the peritoneal cavity.

Conclusions

Leakage of CSF from the thecal sac into the epidural space can occur as a complication of LP shunt placement. This problem produces a low ICP syndrome that may be confused with the results of excessive shunting, or even obstruction. To accurately diagnose the condition, it is useful to combine a radionuclide shuntogram with direct measurement of CSF pressure. Both MR imaging and CT studies can also show the leak site in the thecal sac that is associated with the lumbar shunt catheter.

References


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