Subdural fluid collections after decompression for Chiari malformation

To The Editor: We read with great interest the recent article by Bahuleyan at al. (Bahuleyan B, Menon G, Harihar E, et al: Symptomatic posterior fossa and supratentorial subdural hygromas as a rare complication following foramen magnum decompression for Chiari malformation Type I. Report of 2 cases. J Neurosurg 114:510–513, February, 2011) in which they described 2 patients experiencing symptomatic subdural hygromas after foramen magnum decompression (FMD) for Chiari malformation. The authors suggest that this unusual complication can be prevented “by widely opening the arachnoid” “regardless of whether or not the arachnoid is damaged at surgery.” In addition, they recommend surgical intervention for symptomatic patients “with either a bur hole (posterior fossa or supratentorial) or a VP [ventriculoperitoneal] shunt.”

We would like to comment briefly on their report. In our opinion, the term “subdural hygroma” is confusing and does not fully describe the occurrence of subdural fluid collections (SFCs) after FMD for Chiari malformation. The term subdural hygroma is generally used to describe SFCs in elderly patients with atrophic brains after a traumatic tear in the arachnoid membrane. In these patients, when the subdural collection does not resolve spontaneously and the ipsilateral ventricle is compressed, the subdural external drainage is usually curative. The occurrence of symptomatic SFCs after FMD is a rare entity, and its pathophysiology is complex and still unclear. A tear in the arachnoid membrane at the time of surgery plays a pivotal role in the development of this condition. In 2008, we provided an extensive review of the literature and found that the wide fenestration of the arachnoid at the time of surgery does not prevent the occurrence of SFCs after FMD. Subdural fluid collections after FMD can be transient or can progress to become excessive CSF accumulations in different intracranial compartments. The SFC can shift from infratentorial to supratentorial subdural space and from one side to the contralateral side during neuroradiological monitoring or after bur hole evacuation, which is generally not curative for these patients. In addition, SFCs should be monitored using serial CT scanning as these collections may be complicated by the occurrence of hydrocephalus. The development of ventricular dilatation despite the presence of SFCs indicates the formation of external hydrocephalus, as a result of disturbed CSF flow and/or absorption. Like external hydrocephalus after subarachnoid hemorrhage, the blood-contaminated CSF in patients with an arachnoidal rent that occurred during FMD can interfere with the absorption of CSF, leading to its accumulation. It is noteworthy that about 50% of reported patients with SFCs after FMD presented with pseudomeningocele, which seems to prevent normalization of CSF flow at the craniovertebral junction because of dissipation of the systolic pulse pressure into the distensible pseudomeningocele cavity.

These concepts are exemplified by a case that we recently encountered. A 4-year-old boy with a Chiari I malformation without syringomyelia (Fig. 1A) underwent suboccipital craniectomy and C-1 laminectomy by using an extraarachnoidal technique. During the operation, a small tear in the arachnoid was accidentally made. Ten days after the initial surgery, the patient developed progressive swelling on the wound site that was associated with severe headache. A CT scan revealed a left frontal SFC with prominent midline shift (Fig. 1B), bilateral infratentorial SFCs, and a significant pseudomeningocele (Fig. 1C). Following left frontal bur hole evacuation and CSF aspiration from the pseudomeningocele, the symptoms rapidly improved, and a CT scan disclosed complete resolution of midline shift. On postoperative Day 10 after bur hole evacuation, the patient experienced increasing headache, and a CT scan demonstrated an SFC on the contralateral side (Fig. 1D). The patient was treated conservatively and exhibited progressive clinical improvement. Two weeks after the initial operation, the symptoms recurred and repeat imaging disclosed a recurrent left SFC along with dilation of all 4 ventricles. The patient underwent VP shunt insertion with rapid clinical improvement. A postoperative CT scan revealed resolution of SFC and decrease in ventricular size (Fig. 1F). At the 6-month follow-up the patient is symptom free.

We look forward to additional studies to advance in the understanding of the pathophysiology and clinical course of this unusual complication after FMD for Chiari malformation.

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References


RESPONSE: We appreciate the interest of Drs. Perrini and Di Lorenzo on this controversial topic and thank them for their valuable remarks. We agree that postoperative CSF collections in the subdural space are best referred to as SFCs rather than subdural hygromas, the latter being a different clinicopathological entity as mentioned by Yoshimoto et al.1

The controversial issues as we understand them are summarized as follows: the mechanism of SFC formation, which can appear both in the supratentorial compartment and in the infratentorial compartment and can shift from one side to another, and whether a wide arachnoid opening along with a lax duraplasty can prevent this complication; the occurrence of hydrocephalus despite the presence of a symptomatic SFC producing mass effect; and the best means to treat symptomatic SFCs.

The general policy at our institution is to open the arachnoid widely and perform a lax duraplasty. The patients we described in our report were the first 2 cases at our institution in which an attempt was made to keep the arachnoid intact. In our series of 252 patients, we observed symptomatic SFC only in these 2 patients in whom an arachnoid preserving technique was attempted.1,2 We were therefore inclined to believe that postoperative SFC develops due to the slow leakage of CSF through an iatrogenic arachnoid defect, which probably acts like a one-way valve. For the same reason, we proposed that this complication can be prevented by a wide opening of the arachnoid. We thank Drs. Perrini and Di Lorenzo for sharing their experience with a similar complication in a patient of theirs in whom the arachnoid was widely opened at surgery.3 We accept that the exact etiology for the development of this complication following FMD is unclear and that leaving the arachnoid wide open may not prevent this complication.

Interestingly, in our series of 252 patients, we have

Fig. 1. Preoperative MR image (A) and serial postoperative noncontrast-enhanced CT scans (B–F) obtained in a 4-year-old boy with a Chiari malformation. A: Sagittal T1-weighted image demonstrating Chiari I malformation. B and C: A left frontal SFC causing prominent midline shift and pseudomeningocele (arrow) was noted 10 days after FMD. D: Image obtained 10 days after bur hole evacuation of the left SFC. A low-density fluid collection is detected on the contralateral side. E: Hydroce- phalus developed 2 weeks after FMD. Note the recurrent left frontal and the interhemispheric SFCs. F: Image obtained after shunt insertion. Note the paradoxical response of SFCs to ventricular shunting.