Hydrocephalus after meningioma surgery

TO THE EDITOR: A retrospective study on postoperative hydrocephalus by Burkhardt and colleagues¹ (Burkhardt JK, Zinn PO, Graenicher M, et al: Predicting postoperative hydrocephalus in 227 patients with skull base meningioma. *Neurosurg Focus* 30(5):E9, May 2011) is really interesting and is focused on a not completely explained phenomenon. Furthermore, postoperative hydrocephalus can seriously affect the postoperative course, resulting in a worsening of patients' neurological performances. Because this form of hydrocephalus is a well-recognized and treatable condition in neurosurgical practice, its incidence and predictive factors are important.

The authors' data demonstrating that de novo postoperative hydrocephalus occurs more often in the skull base than in other locations agree with evidence published in the literature.² Nevertheless, the etiology of postoperative hydrocephalus is still unknown.

Some authors explain the complication of postoperative hydrocephalus as the result of spillage of blood products in the subarachnoid and cisternal spaces, as is known to happen in subarachnoid hemorrhage (SAH). However, the very different rates of hydrocephalus after SAH (range 20%–30%) compared with those after skull base surgery for meningioma (range 2%–13%) seem to indicate that postoperative hydrocephalus has a multifactorial etiology.²

We report an unexpected CSF finding, which could represent one of the multiple etiological factors in postoperative hydrocephalus, in a woman in whom hydrocephalus developed after meningioma surgery. This patient underwent surgery for an MRI foramen magnum meningotheliomatous meningioma without MRI-documented hydrocephalus (Evans' index 0.27). After a macroscopically complete removal and coagulation of the dural attachment (Simpson Grade II), postoperative neuroradiological imaging revealed no hemorrhagic complication, and the patient had an uneventful postoperative course. Two months later, she developed a CSF fistula, severe headache, and vomiting. Laboratory data were within the normal range, and a CT scan showed a communicating hydrocephalus (Evans' index 0.37) and a CSF fistula in the posterior fossa. In order to rule out possible infections, gauge possible alterations of CSF, and verify if CSF subtraction could improve the patient's clinical status, we withdrew and analyzed a CSF sample. The CSF was obtained by a lumbar puncture performed in the lumbar cistern. We also performed a cytological examination because some evidence in the literature

points out the presence of cells in CSF and their connection to hydrocephalus.⁶

It is well known that in some types of tumor there are CSF chemical modifications; for instance, in vestibular schwannomas, postoperative communicating hydrocephalus can occur in up to 42% of cases, and the cause is an elevation of protein levels in the CSF.⁴ Moreover, in glioblastomas after surgery and adjuvant radiochemotherapy, high CSF proteins levels are found in up to 82.8% of cases that develop hydrocephalus.⁵

After the CSF analysis, we had an unexpected observation: we found neither features of infection nor a high level of protein; rather, aggregates of tumoral meningioma cells were demonstrated (Fig. 1). After a few days, when the patient's clinical status had improved, a ventriculoperitoneal shunt with a programmable valve was implanted. We also analyzed a sample of ventricular CSF that was collected when the ventricular catheter was placed, and no meningioma cells were observed. Several days later, the patient was discharged from the hospital without complications (Fig. 2).

We think that these cells may have contributed to an obstruction of absorptive CSF pathways and also contributed to hydrocephalus development. The discrepancy between the lumbar and ventricular CSF cytology agree with some data reported in the literature.³

FIG. 1. Tumoral meningioma cells forming whorls in the CSF. May Grünwald-Giemsa stain, original magnification $\times 20$.

Fig. 2. **A:** Preoperative MR image. **B:** Postoperative MRI after meningioma removal. **C:** Axial CT scan demonstrating hydrocephalus 2 months after meningioma surgery. **D:** Axial CT scan after shunt insertion.

So, in this case, cell aggregations could represent a cofactor in hydrocephalus development. Surely a large study is necessary to highlight the prevalence of this finding in patients' CSF and to correlate cellular aggregate with hydrocephalus.

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Disclosure

The authors report no conflict of interest.

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RESPONSE: We thank Dr. Rossetto and colleagues for their interest in and comments on our recently published article on the occurrence of hydrocephalus after skull base meningioma surgery. The authors additionally report an unexpected CSF finding in a patient after resection of a foramen magnum meningioma with postoperative hydrocephalus. Aggregates of tumoral meningioma cells were found in the CSF after lumbar puncture 2 months postoperatively, and Rossetto et al. suggest the latter as an etiology for the occurrence of hydrocephalus. However, ventricular CSF analysis during ventriculoperitoneal shunting did not reveal any meningioma cells.

We agree that meningioma cell spread as etiology for hydrocephalus after meningioma surgery is an intriguing hypothesis, but it requires further investigation in a larger patient cohort. Spinal meningioma cell spread or spinal drop metastasis is not a new finding and is a well-known complication of intracranial meningiomas, especially when located in the posterior fossa, with a transitional histological characteristic in benign meningiomas or in high-grade meningiomas.² Similar to other CNS tumors with CSF spreading or intracerebral bleeding such as SAH, cells or blood products may follow the CSF path in a sedimentation manner. Although rare, data on benign meningiomas (WHO Grade I) showed intracerebral spreading besides a spinal or extraneural spreading.¹ In this study, none of these patients were symptomatic with hydrocephalus. Therefore, detection of meningioma cells in CSF may not necessarily be a reason for impairment of CSF circulation.

Nevertheless, Rossetto et al. present an interesting case, and further studies should address if meningioma cell spreading and aggregation sufficiently explain the etiology of hydrocephalus in meningioma patients.

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