



Case Report

Communicating hydrocephalus after resection of a meningioma ventral to the foramen magnum

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ABSTRACT

Background: Tumors in or near the foramen magnum may cause communicating or non-communicating hydrocephalus (HC), depending on their size and location. Here, an 81-year-old female developed communicating HC following the resection of a meningioma ventral to the foramen magnum.

Case Description: An 81-year-old female presented with numbness in the left neck and left hemiparesis. The magnetic resonance revealed an 18-mm tumor ventral to the foramen magnum that significantly enlarged over the past 6 months. She underwent total tumor resection but then presented with progressive HC both clinically (i.e., instability of gait with confusion) and radiographically (computed tomography). Following placement of a lumboperitoneal (LP) shunt, symptoms markedly improved. Further, the cerebrospinal fluid (CSF) analysis showed elevated cell counts and protein concentrations, indicating likely "leakage" of intratumoral contents postoperatively contributing to the progressive HC.

Conclusion: Patients presenting with acute meningiomas ventral to the foramen magnum may develop postoperative communicating HC attributed to tumor-related CSF leakage of necrotic intratumoral components that can be successfully treated with a LP shunt.

Keywords: Cerebrospinal fluid malabsorption, Foramen magnum meningioma, Lumbar peritoneal shunt, Postoperative hydrocephalus, Tumor resection

INTRODUCTION

Depending on their size and location, tumors arising in/near the foramen magnum can cause communicating or non-communicating hydrocephalus (HC). This is largely attributed to tumoral leakage of necrotic components (i.e., elevated protein) into the cerebrospinal fluid (CSF). Here, an 81-year-old female, 2 weeks following gross total resection of a foramen magnum meningioma, developed acute communicating HC successfully treated with a lumboperitoneal (LP) shunt.

CASE DESCRIPTION

An 81-year-old female presented with 6 months of dizziness and numbness in the left neck/body and 2 weeks of acute left hemiparesis. The brain magnetic resonance (MR) showed an 18-mm tumor located anterolaterally to the right of the foramen magnum that had enlarged over the

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past 6 months; there was no accompanying HC [Figure 1]. The cerebral angiogram revealed a tumor stain consistent with a meningioma fed by the right anterior meningeal, right ascending pharyngeal, and left ascending pharyngeal arteries; embolization of the former two vessels was the day before direct tumor resection [Figure 2a and b].

Surgery

In the right park-bench position, hemisection of C1 and partial osteotomy of the right foramen magnum and right condylar fossa

were performed. A dural entry point clearly separated from the right vertebral artery was identified. The tumor was exposed once the dura and dentate ligament were incised, along with retraction of the posterior root of C1 [Figure 3a]. The caudal side of the tumor (i.e., including the ventral portion of the C2 posterior root) was separated carefully from its dural attachments and readily fragmented/aspirated using the SONOPET® (ultrasonic aspirator) [Figure 3b]. Gross total excision was achieved of this lesion which proved histopathologically to be a World Health Organization Grade I meningioma [Figure 3c].

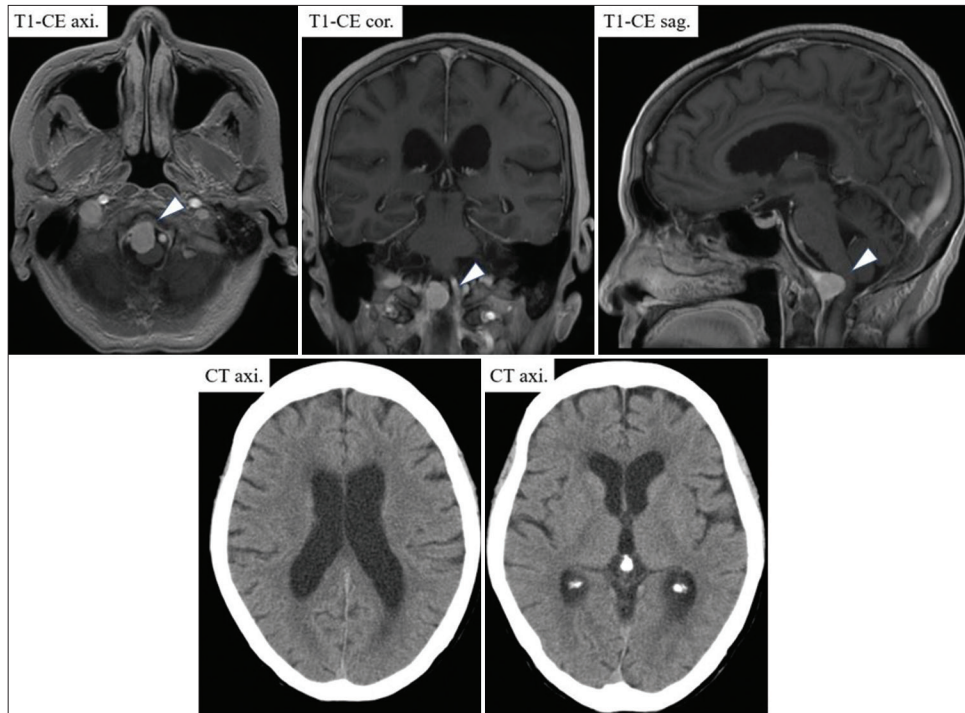


Figure 1: Spinal magnetic resonance imaging revealed an 18-mm tumor lesion with a contrast effect on the ventral side of the foramen magnum (arrowhead); moreover, computed tomography showed no hydrocephalus (Evans index: 0.29).

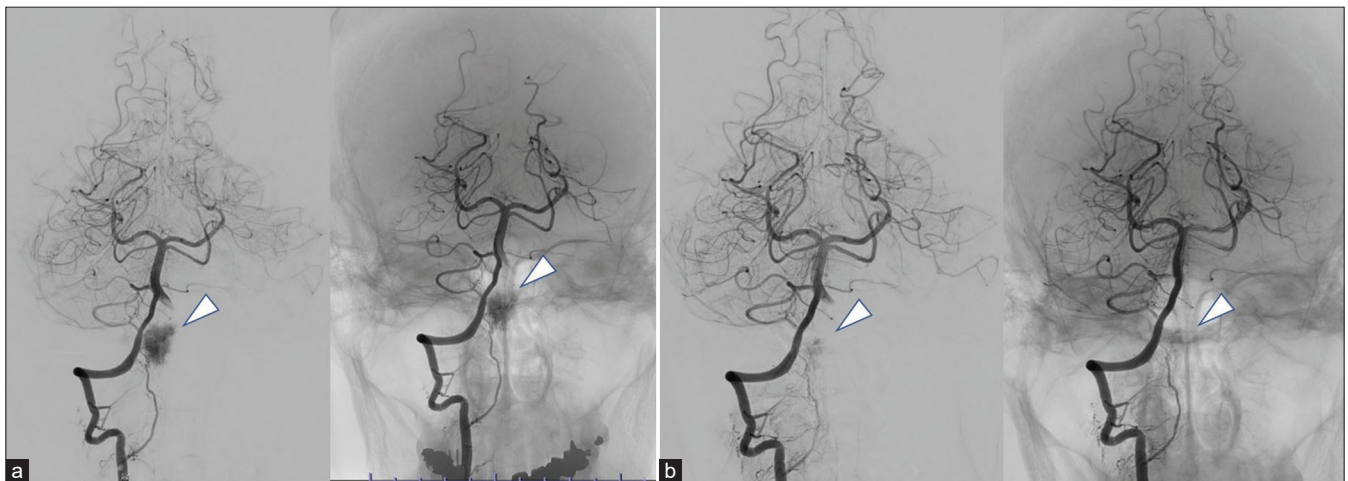


Figure 2: (a) Tumor staining image obtained through head angiography. (arrowhead). (b) Tumor staining disappeared due to the embolization of nutrient vessels (arrowhead).

Postoperative course

Three days later, the patient only had a mild residual right lower extremity paresis and the repeat MR confirmed complete tumor removal [Figure 4a]. At 2 postoperative weeks, however, the patient developed recurrent staggering of gait and confusion. When the brain computed tomography confirmed the development of HC, a LP shunt was placed [Figure 4b]. Subsequent CSF samples documented high spinal fluid cell counts (i.e., 337/ μ L) and a high protein level (i.e., 86 mg/dL) responsible for the development of the patient's communicating HC. Lumbar peritoneal (LP)

shunting was performed, which alleviated the patient's symptoms [Figure 4c].

DISCUSSION

The incidence of HC following resection of foramen magnum meningiomas ranges from 4.5% to 20%.^[1,4] Risk factors potentially contributing to postoperative HC include postoperative CSF leak, prior craniotomy, prior radiation therapy, postoperative CSF infection, scarring of the CSF pathways, increased CSF blood products, and impaired hydrodynamics in the basal cisterns resulting from elevated

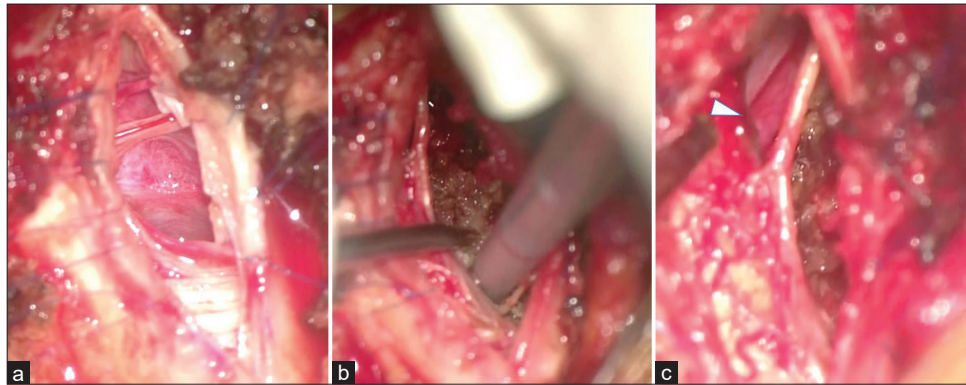


Figure 3: (a) The right-sided dura was incised, the dentate ligament and the posterior root of C1 were cut, and the tumor was exposed. (b) The tumor was carefully fragmented and completely removed by ultrasonic aspiration. (c) The tumor was completely removed, and the contralateral vertebral artery was identified (arrowhead).

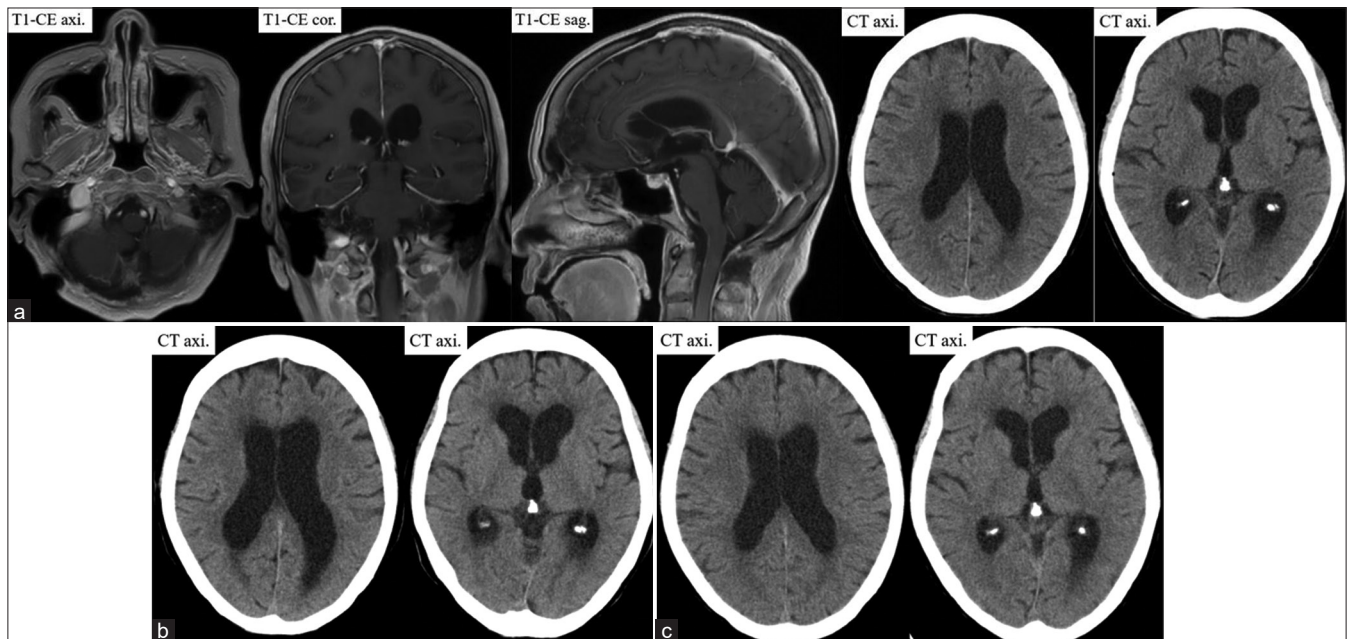


Figure 4: (a) Spinal magnetic resonance imaging showed complete tumor removal, and computed tomography (CT) showed no hydrocephalus (HC) (Evans index: 0.30). (b) CT showed the appearance of HC (Evans index: 0.36). (c) After lumboperitoneal shunt procedure, CT showed improvement in HC (Evans index: 0.32).

Table 1. A summary of the literatures reporting on Postoperative communicating hydrocephalus after tumor resection.

Reference No.	Study	Year	Tumor Type	Location	Mean Age / Number of Patients	Incidence of Post-treatment Hydrocephalus (%)	Risk factors
1	Benoit P, <i>et al.</i>	2010	Meningioma	Foramen magnum	47 (N=1)	4.5	Intraoperative transient CSF blockage
2	Burkhardt JK, <i>et al.</i>	2011	Meningioma	skull base	73/ (N=18)	7.9	Advanced patient age, Longer duration of surgery
3	Duong DH, <i>et al.</i>	2000	Meningioma, Schwannoma, etc	Cranial fossa	-	8.0	CSF leak, Prior craniotomy, Prior radiation therapy, CSF infection, Blood products, Elevated protein levels
4	Maick W, <i>et al.</i>	2021	Meningioma	Foramen magnum	37.8 / (N=20)	20.0	-

CSF protein levels [Table 1].^[2,3] Here, the subsequent onset of communicating HC was most likely attributed to CSF malabsorption (i.e., due to the release of tumor necrotic components and proteins into the CSF). Meticulous piecemeal removal of the tumor, ultrasonic aspiration/debulking of tumor, and careful lysis of tumor/dural adhesions (i.e., especially lateral to ventral) were likely to be major contributors to elevated CSF protein levels and the development of communicating HC in this patient.

CONCLUSION

Following resection of a foramen magnum meningioma, patients may acutely develop communicating HC attributed to high CSF protein levels/tumor debris, subsequently requiring placement of LP shunts.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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