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Case Report

A case of a cerebral cavernous malformation of the third ventricle that caused the syndrome of inappropriate secretion of antidiuretic hormone

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Abstract

Background: Cerebral cavernous malformations (CCMs, also known as cavernous hemanigiomas) of the third ventricle are uncommon. Here, we present a rare case of a CCM that caused the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).

Case Description: A 68-year-old man presented with acute-onset cognitive and memory disturbance. Endocrinological examinations revealed hyponatremia due to SIADH. Computed tomography indicated a high-density mass in the third ventricle that caused left unilateral hydrocephalus due to obstruction of the foramen Monroe. On magnetic resonance imaging, the mass showed high intensity in both T1 and T2-weighted images and low intensity in susceptibility-weighted images, suggesting subacute intralesional hemorrhage. We completely excised the mass via a basal interhemispheric translamina terminalis approach. Intraoperatively, the mass adhered tightly to the left hypothalamus, which was supposed to the origin and was well circumscribed from the surroundings. The histopathological diagnosis was CCM, and his SIADH improved after the operation.

Conclusion: We presented a rare case of a CCM in the third ventricle that caused SIADH, which improved after complete excision of the mass via a basal interhemispheric translamina terminalis approach.

Key Words: Cavernous hemangioma, cerebral cavernous malformation, SIADH, third ventricle

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INTRODUCTION

The prevalence of cerebral cavernous malformations (CCMs, also known as cavernous hemanigiomas) has been reported to be 0.4–0.6%.[4] The frequent sites of CCMs are the cerebral hemisphere, brain stem, basal ganglia, and cerebellar hemisphere.[4] CCMs rarely occur in the third ventricle; therefore, the clinical characteristics of such a CCM remain unclear. Here,

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we present a rare case of a CCM in the third ventricle that caused the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).

CASE REPORT

A 68-year-old man presented with acute-onset cognitive and memory disturbance 10 days before admission. He experienced disorientation and had a score of 12 points on the mini-mental state examination (MMSE). He had no headache, nausea, or visual disturbance, including a visual field defect. Endocrinological examinations revealed hyponatremia (119 mEq/L), serum hypo-osmolality (242 mOsm/L), urinal hyper-osmolality (475 mOsm/L), presence of serum antidiuretic hormone (1.6 pg/mL), continued renal excretion of sodium (93.6 meq/L), normal adrenocortical function, absence of clinical evidence of volume depletion, absence of other causes of hyponatremia, and correction of hyponatremia with fluid restriction, which met the criteria of SIADH. Computed tomography (CT) indicated a high-density mass located in the third ventricle that caused left unilateral hydrocephalus due to obstruction of the foramen Monroe [Figure 1]. Intraventricular hemorrhage was not apparent. On magnetic resonance imaging (MRI), the tumor showed high intensity in both T1-weighted images (T1WIs) and T2-weighted images (T2WIs), low intensity in susceptibility-weighted images (SWIs), and a low intensity peripheral rim in T2WIs, suggesting subacute intralesional hemorrhage. The mass was not enhanced with gadolinium. Cerebral angiography revealed no mass stains.

The mass was removed via a basal interhemispheric translamina terminalis approach [Figure 2]. An old hematoma was suctioned after incision of the capsule. The mass adhered tightly to the left hypothalamus, which was supposed to the origin and was well circumscribed from the surroundings. We carefully separated the mass from the left hypothalamus and completely excised the mass. Histopathological examination indicated that the specimen was composed of variant vessels, hematomas, and hemosiderin according to hematoxylin-eosin staining [Figure 3]. Elastica van Gieson staining showed thin blood vessel walls containing endothelium as well as a collagenous adventitia. CD34-immunoreactivity was identified in the endothelial-like cells. The histopathological diagnosis was CCM.

Hyponatremia and serum hypo-osmolality improved (Na: 137 meq/L, serum osmolarity: 279 mOsm/L) without fluid restriction 7 days postoperatively. Diabetes insipidus did not appear. Postoperative T1WIs showed that the mass was completely excised and that the left unilateral hydrocephalus improved [Figure 4]. However, disorientation and memory disturbance did not recover after the operation, and he was transferred to another hospital for rehabilitation of higher brain function. His MMSE score improved to 21 points, and he could perform indoor activities of daily living, however, he could not resume work at 1 year after the operation.

DISCUSSION

Intraventricular CCMs represent only 2–10% of CCMs in the literature;[7] Kivelev *et al*. reported that CCMs in the third ventricle accounted for 38% of all intraventricular CCMs.[6] CCM in the third ventricle usually manifest when the mass enlarges by repeated bleeding, compresses the surrounding structures (optic pathway, hypothalamus, and thalamus), and/or obstructs the flow of cerebrospinal fluid.[8] The common clinical symptoms are visual disturbance, memory disturbance, and increased signs of

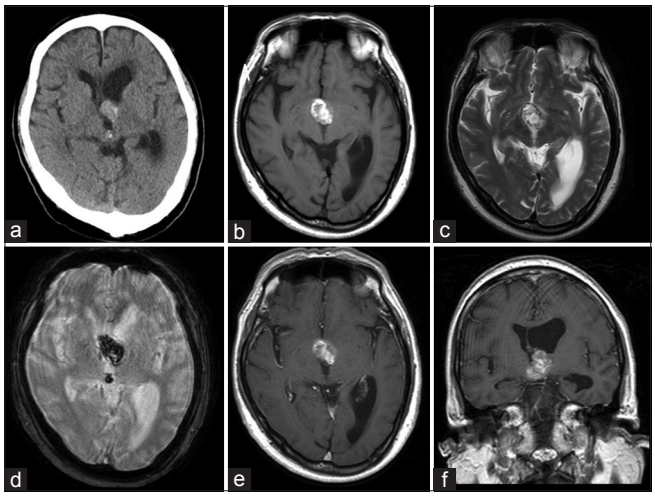

Figure 1: Preoperative computed tomography scan (a) shows a high-density mass in the third ventricle, which obstructed the foramen Monroe, causing left unilateral hydrocephalus. On MRI, the mass shows high intensity in both T1-weighted images (b) and T2-weighted images (c) and low intensity in susceptibility-weighted images (d), suggesting subacute intralesional hemorrhage. The mass is not enhanced with gadolinium (e: Axial, f: Coronal image)

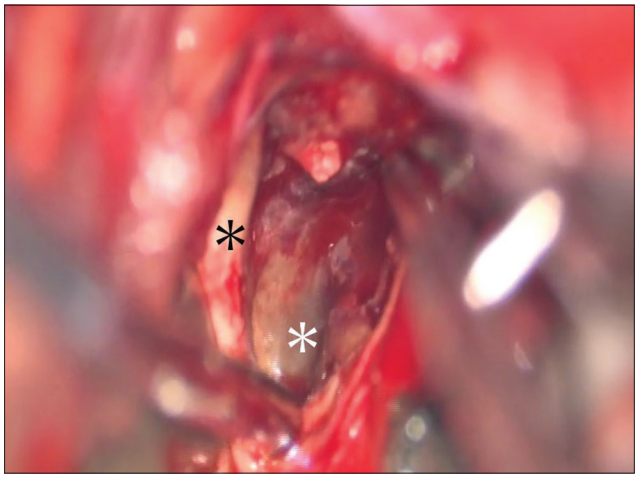

Figure 2: Surgical view shows that a xanthochromic and multilobulated tumor (white asterisk) in the third ventricle, which adhered to the left hypothalamus (black asterisk)

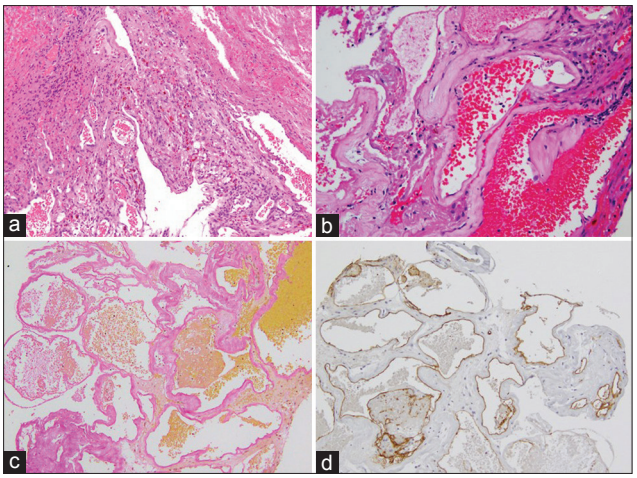

Figure 3: Photomicrograph of the surgical specimen stained with hematoxylin and eosin shows variant vessels, hematomas, and hemosiderin (a and b). Elastica van Gieson staining shows thin blood vessel walls containing endothelium and a collagenous adventitia (c). CD34-immunoreactivity is identified in the endothelial-like cells (d)

intracranial pressure, such as headache, nausea, vomiting, and consciousness disturbance.[6,8,9] Few cases of endocrine dysfunction caused by CCMs in the third ventricle have been reported, but they were unspecific.[6,8,9] In our case, endocrine examinations showed hyponatremia due to SIADH before surgery.

Antidiuretic hormone is produced by the supraoptic and paraventricular nuclei of the hypothalamus and is released at the posterior pituitary gland. SIADH due to suprasellar tumors, such as Rathke's cleft cysts, craniopharyngiomas, germ cell tumors, arachnoid cysts, and pituitary adenomas, sometimes occurs before surgery, because the mass might cause inappropriate ADH release by direct mechanical stimulation and ischemic changes at the osmoreceptor and ADH-secreting neurons.[1,2] In our case, SIADH might have resulted from compression of the bilateral hypothalamus by rapid enlargement of the CCM due to intralesional hemorrhage, and it improved by decompression of the hypothalamus after surgery.

CCMs in the third ventricle have been reported to show rapid growth compared with those at other sites, and they can lead to severe morbidity.[3,5,8,9] Complete resection of the CCMs is recommended because partial resection might result in regrowth or rebleeding.[3,5] In the literature, CCMs in the third ventricle were removed via the transcallosal, transcortical, or translamina terminalis approach by microsurgery, or via the transventricular approach by endoscopic surgery.[5,10] The selection of the approach is dependent on the location of the mass. We selected the basal interhemispheric translamina terminalis approach, which might be the most noninvasive procedure for a mass in the anterior segment of the third ventricle from the viewpoint of damage to brain parenchyma. In our case, removal of the tumor improved hyponatremia and hydrocephalus,

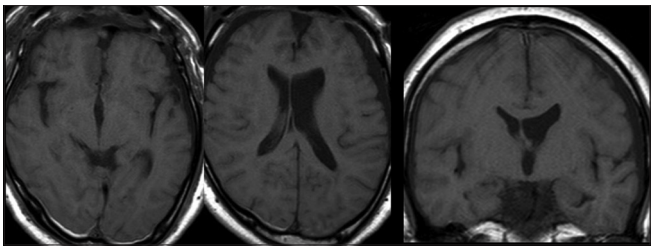

Figure 4: Postoperative T1-weighted images show that the mass is completely excised and that left unilateral hydrocephalus improved

however, the patient had persistent cognitive disorder resulting from damage to the hypothalamus and fornix. Intraoperatively, the mass adhered tightly to the left hypothalamus, which was supposed to the origin. The damage might have been caused by the rapid growth of the mass owing to intralesional hemorrhage, and a surgical procedure has the potential to deteriorate the condition. Therefore, careful dissection is important at such sites.

CONCLUSION

We presented a rare case of a CCM in the third ventricle that caused SIADH. The patient's condition improved after removal of the mass via a basal interhemispheric translamina terminalis approach.

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Conflicts of interest
There are no conflicts of interest.

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