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Case report

Quadrigeminal cistern arachnoid cyst as a probable cause of hemifacial spasm ☆☆☆

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ABSTRACT

Arachnoid cysts arising in the quadrigeminal cistern (ACQCs) are uncommon. A 68-year-old woman presented with an unsteady gait, facial spasm, and cerebellar ataxia. Non-contrast head computed tomography showed a cystic mass centered in the quadrigeminal cistern accompanying ventriculomegaly. On MRI, the cyst appeared hypointense on T1- and hyperintense on T2-weighted sequence. There was no restricted diffusion on diffusion-weighted imaging. The cerebral aqueduct was obstructed and the prepontine cistern was narrowed. The left vertebral artery (VA) coursed adjacent to the facial nerve at its origin. The patient underwent neuroendoscopic fenestration of the posterior wall of the third ventricle and ventral wall of the ACQC. Postoperatively, the patient's symptoms resolved. MRI showed a considerable reduction in the ACQC and expansion of the prepontine cistern, whereas the relationship between the left VA and the proximal segment of the facial nerve did not change. We assumed that the pre-existing close relationship between the VA and facial nerve might have been aggravated by the anterior displacement of the brainstem, thus causing the facial spasm.

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Introduction

Arachnoid cysts arising in the quadrigeminal cistern (ACQC) are uncommon, comprising 5%–18% of all intracranial arachnoid cysts. They are frequently accompanied by obstructive hydrocephalus in the aqueduct, often necessitating neuroendoscopic fenestration with satisfactory outcomes [1,2–9]. In rare instances, ACQCs have caused trigeminal

neuralgia and trochlear nerve palsy [10,11]. Due to the complex vascular structures surrounding the quadrigeminal cistern, microsurgical approaches to the ACQCs pose a great challenge [12,13]. To the best of our knowledge, no patient with ACQC has presented with hemifacial spasm. Here, we document a successfully treated ACQC case, presumably caused by an elongated vertebral artery (VA) segment coursing adjacent to the facial nerve at its origin on the brainstem.

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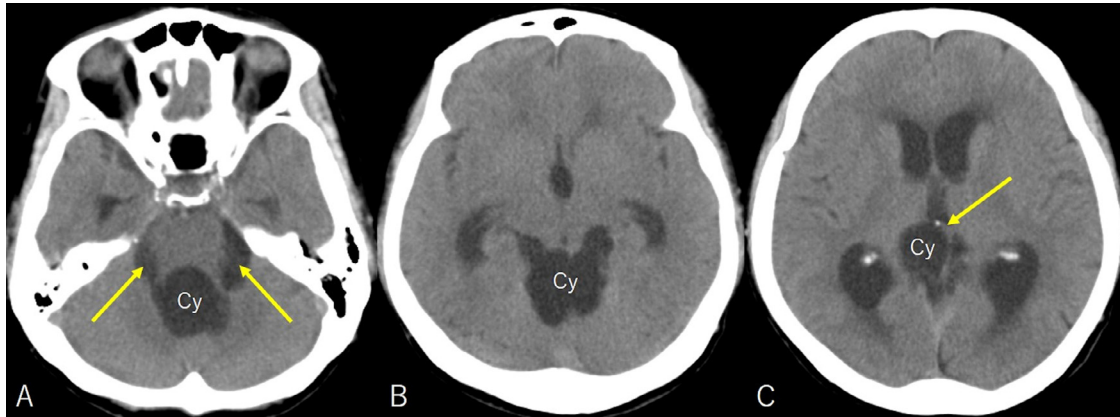


Fig. 1 – (A–C) Non-contrast axial CTs at presentation showing a well-demarcated, hypodense cystic mass (Cy) centered in the quadrigeminal cistern with lateral extension into the ambient cisterns (A, arrows), superior extension to the level of the pineal gland, and inferior extension into the posterior cranial fossa (C, arrow) with accompanying ventriculomegaly

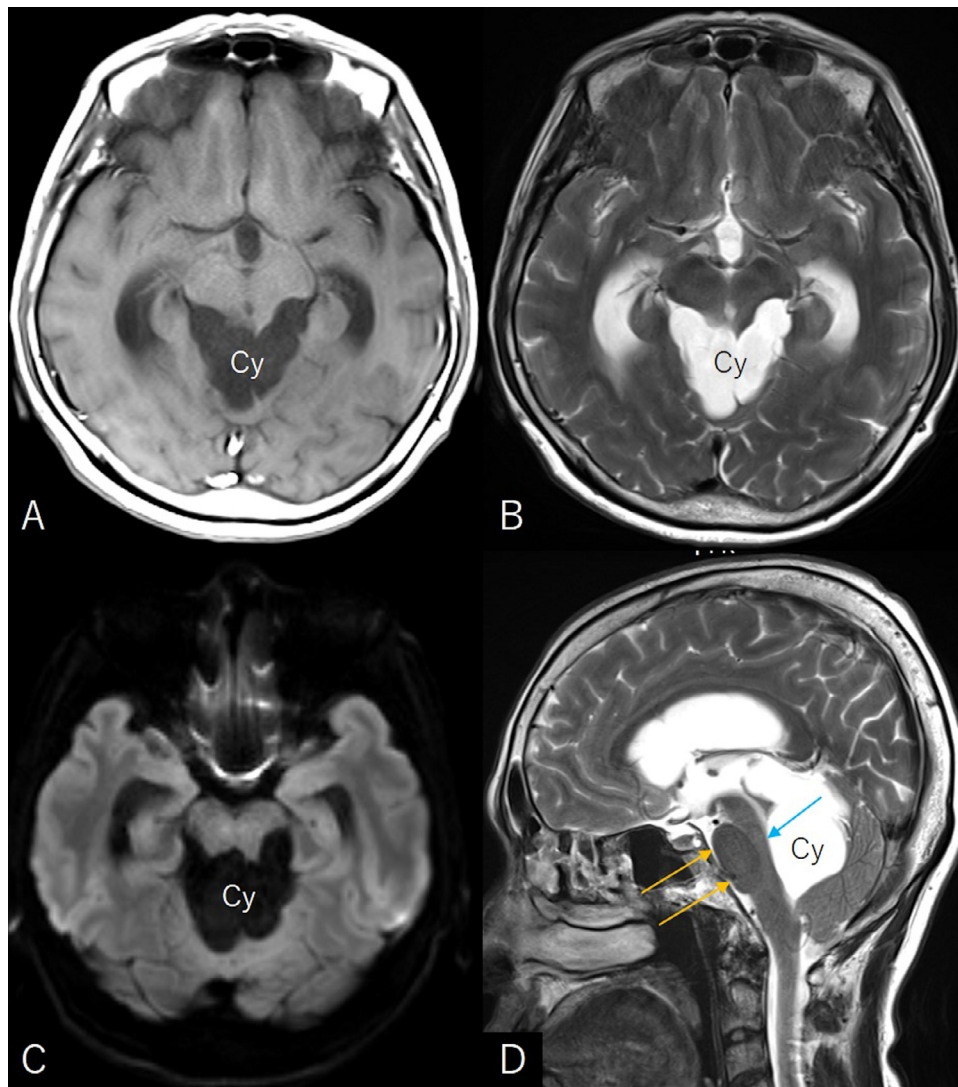


Fig. 2 – Axial T1-(A), axial (B), and sagittal (D) T2-, and axial diffusion-weighted (C) MRI showing the cyst (Cy) appearing hypointense on T1- and diffusion-weighted imaging and hyperintense on T2-weighted sequence. Compressed by the cyst, the aqueduct is obstructed (D, orange arrows) and the prepontine cistern narrowed (D, blue arrow) by the displaced brainstem.

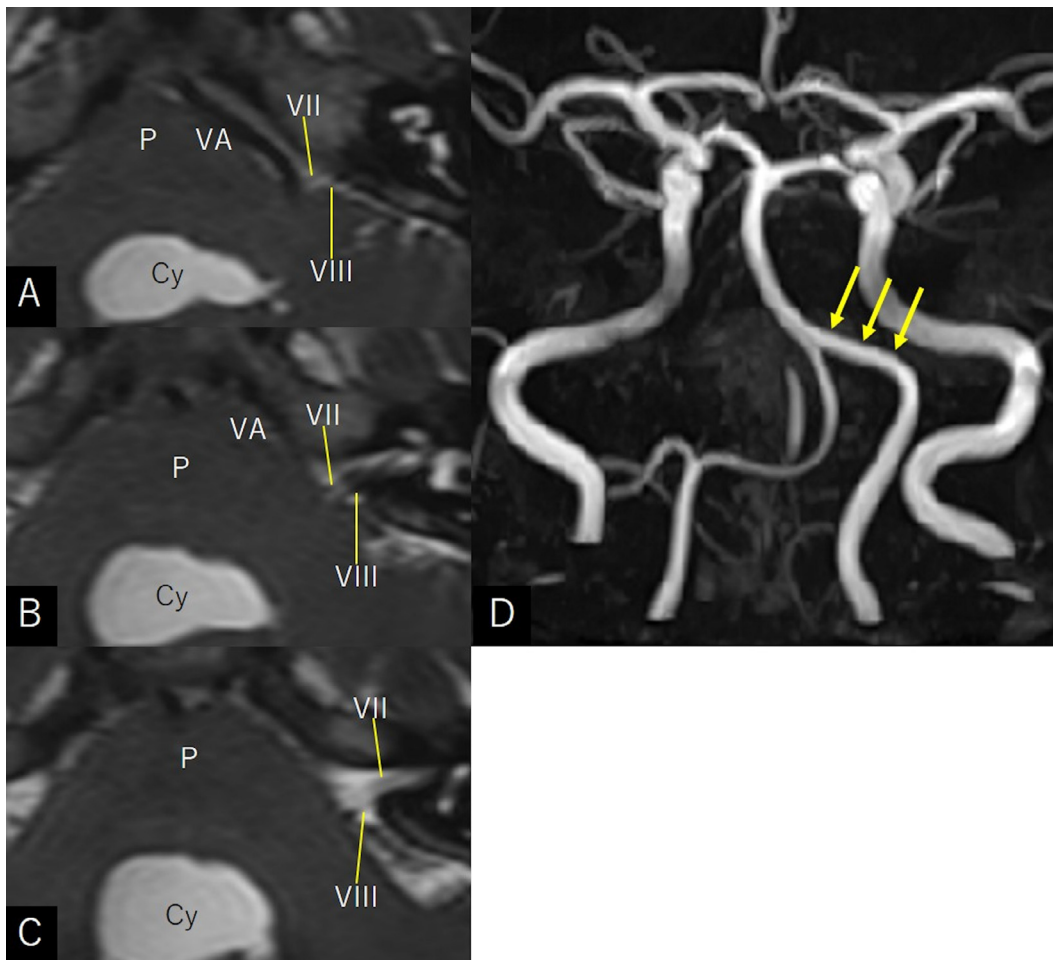


Fig. 3 – (A–C) Serial constructive interference in steady-state images showing the left vertebral artery adjacent to the facial nerve (VII) at the original site of the brainstem. (D) Cerebral MRA, anteroposterior view, showing the elongated left vertebral artery (arrows). Cy, cyst; P, pons; VIII, vestibulocochlear nerve.

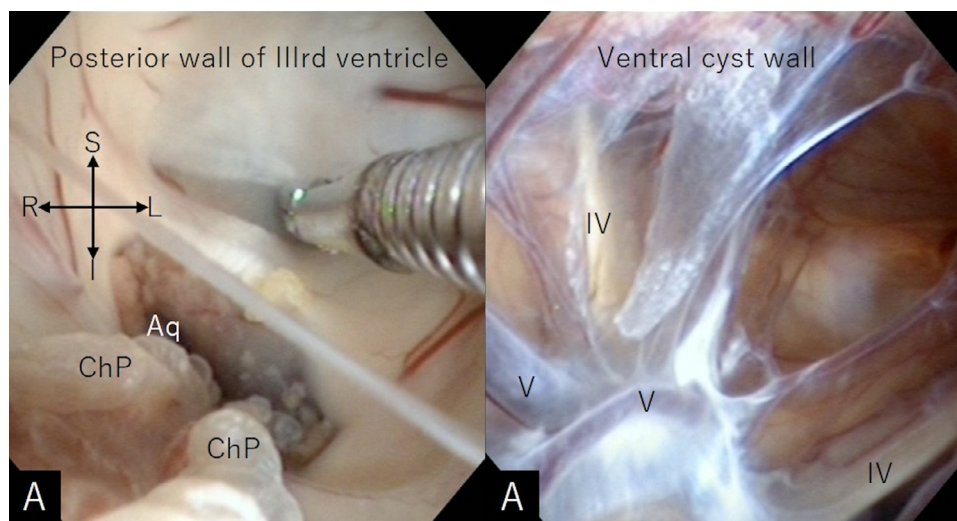


Fig. 4 – Intraoperative photos showing endoscopic perforation of the posterior wall of the third ventricle (A) and after perforation of the ventral cyst wall (B). Note that the bilateral trochlear nerves (IV) are observed through the arachnoid membrane-like cyst wall. Aq, obstructed aqueduct; ChP, choroid plexus; I, inferior; L, left; R, right; S, superior; V, vein.

Case presentation

A 68-year-old previously healthy woman developed progressively worsening gait unsteadiness for 3 months. At presentation, the patient showed intermittent spasms in the left orbicularis oculi and oris muscles, as well as cerebellar ataxia in the left upper and lower extremities. Noncontrast head computed tomography showed a well-demarcated cystic mass centered in the quadrigeminal cistern with lateral extension into the ambient cistern, superior extension to the level of the pineal gland, and inferior extension into the posterior cranial fossa, with moderate ventriculomegaly (Fig. 1). On MRI, the cyst appeared hypointense on T1-weighted imaging and hyperintense on T2-weighted sequence. There was no restricted diffusion on diffusion-weighted imaging. The cere-

bral aqueduct was obstructed and the prepontine cistern was narrowed by the displaced brainstem (Fig. 2). The constructive interference steady-state sequence and magnetic resonance angiography revealed an elongated left VA coursing adjacent to the facial nerve at its origin on the brainstem (Fig. 3). The patient underwent neuroendoscopic procedures for symptomatic ACQC. Following ventriculostomy in the anterior horn of the right lateral ventricle, a flexible neuroendoscope was introduced into the third ventricle. The posterior wall of the third ventricle and ventral wall of the ACQC were then perforated, followed by dilation of the stoma using a balloon. The cyst wall was composed of arachnoid-like membranes (Fig. 4). The ventricle floor was thick and tough with no signs of downward displacement; hence, an additional third ventriculostomy was not performed. Postoperatively, the patient's symptoms resolved. MRI showed a considerable

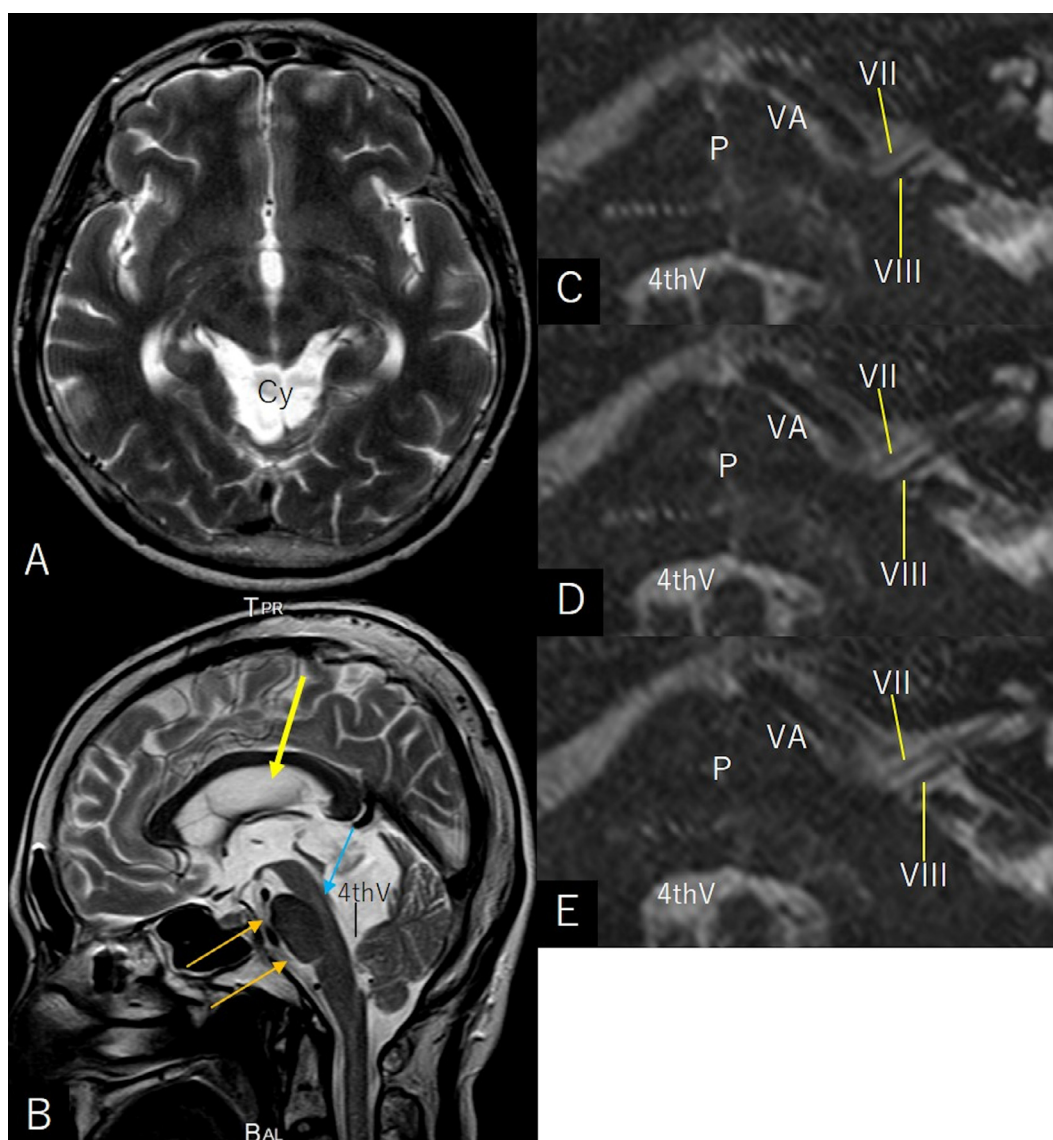


Fig. 5 – Axial (A) and sagittal (B) T2-weighted MRI showing reduction of the cyst (Cy), ambient cisterns, and ventriculomegaly (yellow arrow) and expansion of the prepontine cistern (orange arrows) with the patent aqueduct (blue arrow). (C–E) Serial constructive interference in steady-state images showing no significant changes in the relationship between the left vertebral artery and the proximal segment of the facial nerve (VII). P, pons; VIII, vestibulocochlear nerve; 4thV, fourth ventricle.

reduction in the ACQC, ambient, and ventriculomegaly, in addition to an expansion of the prepontine cistern. The close relationship between the left VA and the proximal segment of the facial nerve that was found on the preoperative imaging did not show any significant change (Fig. 5).

Discussion

Arachnoid cysts arising in the cerebellopontine angle have been documented to cause hemifacial spasm and trigeminal pain [14–16]. However, to the best of our knowledge, ACQC-associated hemifacial spasm has not been reported. In this case, an elongated left VA was found to course adjacent to the origin of the left facial nerve. While this relationship did not change on postoperative imaging, the patient's facial spasm resolved after surgery with an expansion of the prepontine cistern. This close approximation of the VA and the proximal segment of the facial nerve may have been aggravated by the anterior displacement of the brainstem due to compression by the ACQC, resulting in facial spasm. This case suggests that ACQCs may cause hemifacial spasm under certain circumstances.

ACQCs are rare but frequently require intervention for management, including less invasive neuroendoscopic fenestration with cystocisternostomy or cystoventriculostomy combined with the third ventriculostomy that is as effective as open microsurgery [1–7,9]. Due to the complex anatomy in this area, open microsurgical techniques are difficult [12,13]. In this case, the floor of the third ventricle was thick, tough, and lacking downward displacement; hence, a safe third ventriculostomy would not be possible. If the fenestration fails, a shunt surgery/procedure can be performed.

Conclusion

ACQCs may cause hemifacial spasm indirectly. Neuroendoscopic fenestration is useful for the treatment of ACQCs complicated by hemifacial spasm.

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