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Case Report

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# Endoscopic Third Ventriculostomy for Hydrocephalus Associated with Cerebellar Arteriovenous Malformation: A Case Report and Literature Review

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## ABSTRACT

Arteriovenous malformations (AVM) are abnormal arteriovenous shunt lesions that predominantly occur in the brain or spinal cord. However, obstructive hydrocephalus resulting from an unruptured AVM occurs rarely. Herein, we report the case of a patient with obstructive hydrocephalus caused by an unruptured cerebellar AVM that was treated with an endoscopic third ventriculostomy (ETV), and further, we present a literature review. An 11-year-old girl presented to our department with headache, nausea, and vomiting. Magnetic resonance imaging and angiography revealed a cerebellar AVM. Additionally, we used the findings of digital subtraction angiography and confirmed a Spetzler–Martin grade 5 cerebellar AVM fed by branches of the bilateral posterior cerebral and bilateral anterior and posterior cerebellar arteries.

We initiated conservative therapy; however, 10 years after her initial presentation, the patient's acute obstructive hydrocephalus due to aqueductal occlusion had worsened. Therefore, we performed an endoscopic third ventriculostomy that resolved the hydrocephalus and improved the patient's clinical condition. In our review of the literature, we observed that, in most cases, hydrocephalus resulted from deep-seated AVM; furthermore, ETV effectively resolved the hydrocephalus. To summarize, ETV can be an effective alternative to emergent ventriculoperitoneal shunting to treat acute obstructive hydrocephalus caused by unruptured intracranial AVM.

**KEYWORDS:** Arteriovenous malformation, Aqueductal occlusion, Hydrocephalus, Endoscopic third ventriculostomy

**ABBREVIATIONS: AVM:** Arteriovenous malformation, **CSF:** Cerebrospinal fluid, **ETV:** Endoscopic third ventriculostomy, **ICP:** Intracranial pressure, **ICV:** Internal cerebral vein, **MRA:** Magnetic resonance angiography, **MRI:** Magnetic resonance imaging, **SM:** Spetzler–Martin, **VP:** Ventriculoperitoneal

## INTRODUCTION

Brain arteriovenous malformations (AVMs) associated with acute occlusive hydrocephalus occur rarely. In this report, we present the case of a patient with obstructive hydrocephalus caused by a cerebellar AVM that was treated with endoscopic third ventriculostomy (ETV). Additionally, we provide a literature review of reported cases of hydrocephalus associated with brain AVMs.

## CASE REPORT

An 11-year-old girl initially presented to our department with a one-month history of headache, nausea, and vomiting. Brain magnetic resonance imaging (MRI) and magnetic resonance angiography revealed a cerebellar AVM (Figure 1A–C). Digital subtraction angiography revealed a high-grade [Spetzler–Martin (SM) grade 5] AVM fed by branches of the bilateral pos-

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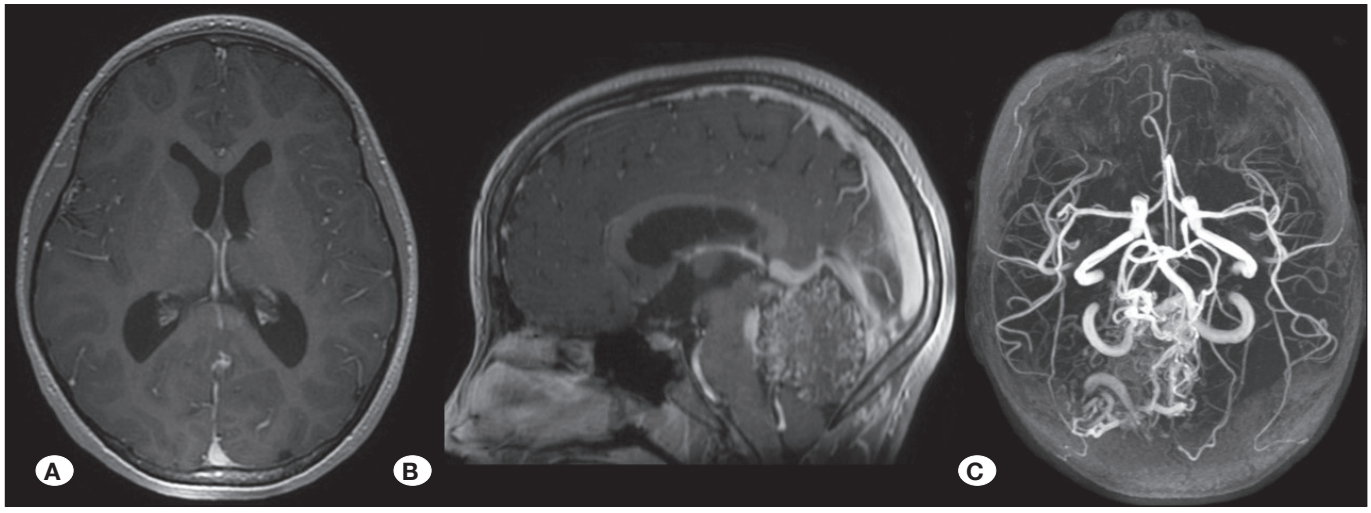


**Figure 1:** Brain magnetic resonance image shows no eminent ventricular enlargement **(A)** and a cerebellar arteriovenous malformation (AVM) stenosing aqueduct **(B)**. Magnetic resonance angiography shows cerebellar AVM **(C)**.

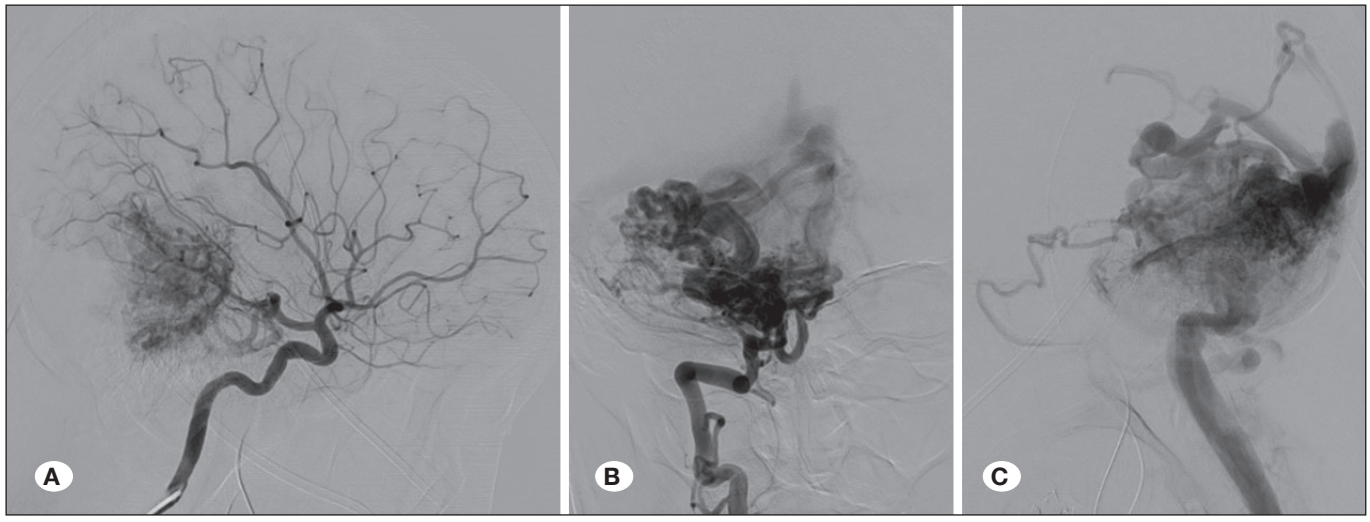


**Figure 2:** Digital subtraction angiography showing a high-grade arteriovenous malformation (AVM) (Spetzler and Martin grade 5) fed by branches of bilateral posterior cerebral artery (PCA) and bilateral anterior and posterior cerebellar artery (AICA and PICA). Right internal carotid angiography shows AVM fed by right PCA and draining into enlarged deep cerebral veins (**A:** Arterial phase in lateral view). Right vertebral angiography shows AVM fed by right PCA, AICA, and PICA that then drains into enlarged deep cerebral veins (**B:** capillary phase in anterior posterior view, **C:** capillary phase in lateral view).

terior cerebral and bilateral anterior and posterior cerebellar arteries (Figure 2A–C) and draining into enlarged deep cerebral veins, internal cerebral veins (ICVs), and the vein of Galen.

We deemed the surgical treatment of this high-grade AVM to be challenging; therefore, we initiated the patient on conservative therapy. However, 10 years after initial admission, she developed severe headaches and moderate confusion. Imaging studies revealed obstructive hydrocephalus caused by aqueductal occlusion (Figure 3A–C). We suspected this aqueductal occlusion resulted from a minor intraventricular hemorrhage. Initially, we treated the increased intracranial pressure (ICP) by administering concentrated glycerin and inducing a 48-hour-barbiturate coma. However, the severe headache and increased ICP persisted, and the patient developed mild disturbance in consciousness. We promptly performed an endoscopic third ventriculostomy (ETV) (Figure 4A–C), which resolved the mild disturbance in consciousness and severe headache. Postoperative brain MRI revealed a mild decrease in ventricular size (Figure 5A–C). After undergoing ETV, the patient did not experience recurrence of hydrocephalus or its resultant clinical symptoms.

Written informed consent was obtained from the patient's family for publishing this clinical report.

## DISCUSSION

In most reported cases (15), 50% of patients with posterior fossa AVM presented with or developed hydrocephalus,

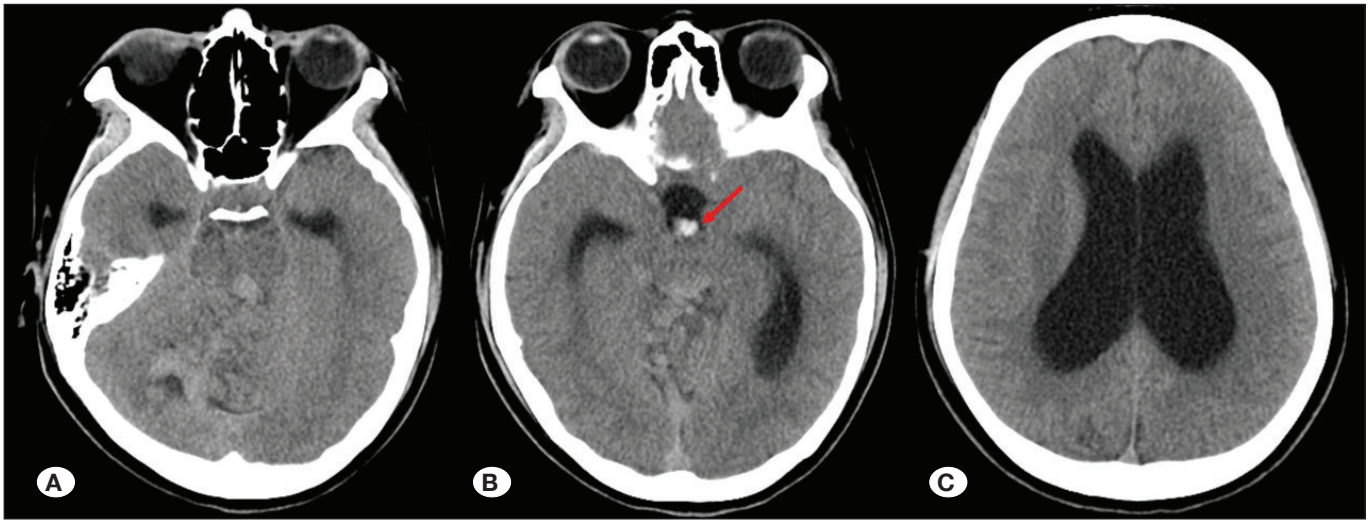


**Figure 3:** At 10 years after the first admission, acute obstructive hydrocephalus is noted following aqueductal occlusion; minor bleeding is suspected to be the cause (red arrow) that worsened the condition.

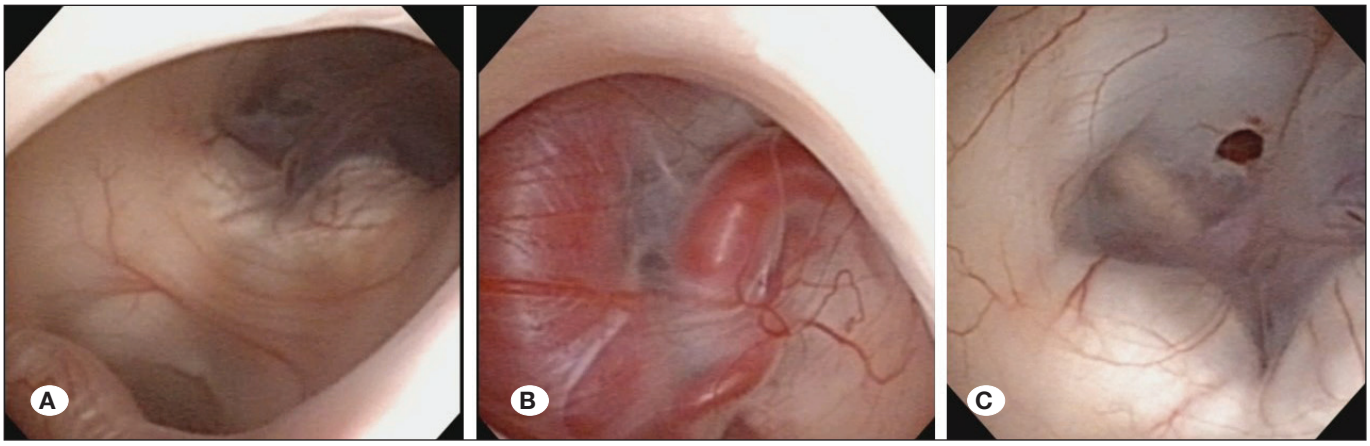


**Figure 4:** Endoscopic third ventriculostomy (ETV) is performed. After confirmation of red dilated drainer packing and aqueduct occlusion (**A:** the view at Monro foramen, **B:** the view to the aqueduct). Third ventriculostomy is undertaken at the center between bilateral mammillary bodies and infundibular recess **(C)**. Intraoperative findings show no hemorrhage from the arteriovenous malformation.

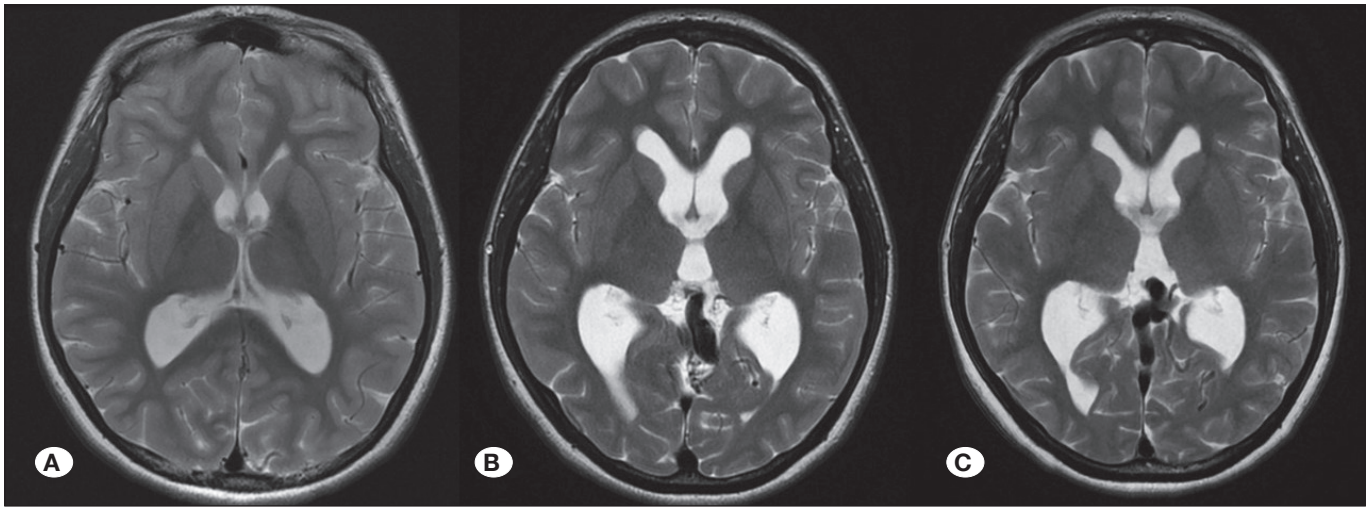


**Figure 5:** After the endoscopic third ventriculostomy (ETV), hydrocephalus disappeared and clinical condition improved (**A:** initial magnetic resonance imaging [MRI], **B:** at the hydrocephalus state MRI, **C:** post-ETV state MRI).

**Table I:** Review of the Cases Treated with ETV for Hydrocephalus Associated with Cerebral and Cerebellar AVMs

| Author | Year | Age (years) | Gender | Location of AVM | Lesion size (cm) | Venous drainage | Spetzler & Martin Grade | Treatment | Angiographical result | Level&cause of obstruction | Treatment of hydrocephalus | Therapy effectiveness | Follow up (month) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Rodrigez and Molet (14) | 2013 | 83 | Male | Posterior fossa | N/A | N/A | N/A | N/A | N/A | Aqueduct (Dilation of Vein of Galen) | ETV | Improved | N/A |
| | | 64 | Female | Posterior fossa | N/A | N/A | N/A | N/A | N/A | Aqueduct (Dilation of Vein of Galen) | ETV | Improved | N/A |
| Tucker et al. (16) | 2013 | 63 | Male | Pineal region | N/A | Lateral perimesencephalic vein → Vein of Galen | 5 | None | Unchanged nidus | Aqueduct (Nidus) | ETV | Improved | N/A |
| Champeaux et al. (2) | 2016 | 54 | Male | Thalamic insular & capsular | 5 | Thalamostriate vein → Rt ICV → Vein of Galen | 5 | None | Unchanged nidus | Aqueduct (Dilation of Vein of Galen) | VP shunt occlusion → ETV | Improved | 8 |
| Ono et al. (12) | 2015 | 56 | Male | Cerebellar vermis | N/A | Cerebellar veins → Vein of Galen | 5 | TAE | Small residual nidus | Aqueduct (Dilation of Vein of Galen) | ETV | Improved | N/A |
| **Present case** | 2024 | 21 | Female | Cerebellum | 7 | Cerebellar veins → Vein of Galen | 5 | None | Unchanged nidus | Aqueduct (Venous pouch) | ETV | Improved | 12 |

***TAE:*** *Transarterial embolization,* ***ICV:*** *Internal cerebral vein,* ***N/A:*** *Not applicapable,* ***AVM:*** *Arteriovenous malformation.* ***VP shunt:*** *Ventricular peritoneal shunt,* ***ICV:*** *Internal cerebral vein,* ***N/A:*** *Not applicapable,* ***ETV:*** *Endoscopic third ventriculotostomy.*

and 40% were treated for it. Approximately 90% of patients presenting with hemorrhage developed hydrocephalus. In 30% of unruptured AVM cases, hydrocephalus developed in the postoperative period after microsurgical AVM removal. In 20% of the posterior fossa AVM cases, temporary external ventricular drainage was sufficient to manage hydrocephalus; however, the remaining 20% required a VP shunt placement.

In children, a deeply located intracranial unruptured AVM with acute obstructive hydrocephalus occurs rarely (5). Although focal neurologic deficits, seizures, and headaches are the most common symptoms, acute neurologic deterioration due to hydrocephalus may be the presenting symptom in children (5).

Occlusive hydrocephalus resulting in brain AVMs, while uncommon, can develop if the lesions are located in and occlude the cerebrospinal fluid (CSF) tract. While reviewing the literature, we observed that, in most cases, hydrocephalus is caused by high-SM-grade AVMs and posterior fossa AVM (15); however, surgical treatment of these AVM types is often challenging, even with recent advances in endovascular technology and radiosurgery that have increased the potential for successful treatment of previously inoperable high-grade AVMs (3,8).

Moreover, hydrocephalus caused by such AVMs should preferably be treated without surgically removing the AVMs. Hydrocephalus is treated by placing a temporary external ventricular drain (EVD) or performing ventriculoperitoneal (VP) shunting or ETV (1,2,5–7,9–16). However, patients with hydrocephalus associated with AVM who were treated with VP shunting have required repeat VP shunt revisions or subsequent ETV (1,2,6,7,9,11,14). In this literature review, we discuss the efficacy of ETV in such cases of hydrocephalus.

We reviewed six cases, which were described in detail, of hydrocephalus associated with cerebral and cerebellar AVMs that were treated with ETV (2,12,14,16) (Table I). In these six cases, the patients were aged 21–83 years, with 67% being men; the hydrocephalus-inducing AVMs were located in the thalamus (n=1; 13%), posterior fossa and midbrain (n=5; 74%), and cerebellum (n=1; 13%). In all cases, the patients presented with dilated veins and drainage of the unilateral thalamo-striate, lateral mesencephalic, and/or cerebellar veins into the ICVs and vein of Galen, which resulted in the obstruction of the aqueduct.

Possible causes of hydrocephalus include venous outflow and hemodynamic imbalance as well as mechanical ventricular obstruction by the dilated vein or nidus (4). The vein of Galen was the most common dilated draining vein causing compression of the ventricular system at the level of the Sylvian aqueduct. Less commonly, the compressing vein was a dilated vein obstructing the foramen of Monro. The superior sagittal sinus was the most common arterialized venous sinus in patients with impaired CSF re-absorption (4).

In the present case, the mechanical obstruction of the aqueduct by the AVM is suspected to have caused the hydrocephalus. However, Ebinu et al. reported a case of AVM-associated hydrocephalus that resulted from the reflux of blood into the periventricular and trans-medullary veins rather than a mechanical obstruction (6). Lobato et al. reported a case of hydrocephalus that resulted from the overproduction of CSF by a choroidal AVM (10).

In our literature review, we found that most cases of hydrocephalus that resulted from AVMs were of higher grades, which required treatment with multimodal therapy, such as endovascular embolization and stereotactic radiosurgery, rather than microsurgical resection. However, in some cases, these treatment modalities can be challenging and therefore unsuitable. In these cases, conservative treatment and monitoring is typically the best course of action, as observed in the present case.

Of the cases of AVM-associated obstructive hydrocephalus that we reviewed, six were primarily treated with ETV.

ETV does not alter the relationship between the supra- and infratentorial pressure and has the advantage of being "shunt-free." Moreover, ETV effectively treated malfunctioning VP shunts and over-drainage following VP shunt placement or revision.

## CONCLUSION

AVM-associated obstructive hydrocephalus occurs rarely, and its surgical treatment is challenging. Hydrocephalus—and its resultant increase in ICP—can be treated by ETV rather than VP shunting.

**Declarations**

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**Availability of data and materials:** The datasets generated and/or analyzed during the current study are available from the corresponding author by reasonable request.

**Disclosure:** The authors declare no competing interests.

**AUTHORSHIP CONTRIBUTION**

Study conception and design: DW
Data collection: DW, YS, ST
Analysis and interpretation of results: DW, YS, ST
Draft manuscript preparation: DW, TK
Critical revision of the article: TK, YS, ST, KM, MN
All authors (DW, YS, ST, KM, MN) reviewed the results and approved the final version of the manuscript.

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