Child's Nervous System (2019) 35:695–699
https://doi.org/10.1007/s00381-018-4011-7

CASE REPORT

Cerebral vasospasm after endoscopic fenestration of a temporal arachnoid cyst in a child—a case report and review of the literature

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Received: 21 August 2018 / Accepted: 20 November 2018 / Published online: 28 November 2018

Abstract

Introduction Intracranial arachnoid cysts (ACs) represent rare extra-axial CSF-containing lesions. Surgical management mainly depends on the cyst location and its size. Nevertheless, pure endoscopic fenestration represents a relatively straightforward and safe technique, and—in most cases—the treatment of choice for symptomatic intracranial ACs. The postoperative complication rate of the procedure is low including subdural hematomas, hygromas, and intraparenchymal hemorrhages. Symptomatic cerebral vasospasm after endoscopic treatment of ACs is a very uncommon event.

Case report/Results To the authors' knowledge, this adverse event in children has not yet been reported in the literature yet. The authors present a case of a 9-year-old child developing an early symptomatic cerebral vasospasm with an insignificant secondary ischemia following endoscopic fenestration of a large temporal arachnoid cyst.

Discussion The clinical approach, possible pathogenesis, and the therapeutic strategy is discussed particularly with regard to the literature.

Keywords Vasospasm · Arachnoid cyst · Arterial ischemic stroke · Endoscopic fenestration · Cystocisternostomy · Neuroendoscopy

Abbreviations

AC	Arachnoid cyst
CSF	Cerebrospinal fluid
FLAIR	fluid attenuated inversion recovery
ICA	Internal carotid artery
ICU	Intensive Care Unit
MAP	Middle arterial pressure
MRI	Magnetic Resonance Imaging
SAH	Subarachnoid hemorrhage
SPECT	Single photon emission computed tomography

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00381-018-4011-7) contains supplementary material, which is available to authorized users.

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Introduction

Arachnoid cysts (ACs) are the most common congenital intracranial cysts which account for approximately 1% of all intracranial lesions [1–3]. Regarding cyst location and size, the surgical approach and technique still remain under debate. Nevertheless, endoscopic fenestration represents a straightforward and safe technique and can be considered as the first-line option for surgical treatment in most cases [2–6]. Regarding the literature [1–8], the postoperative complication rate is low; however, symptomatic cerebral vasospasm following endoscopic treatment of ACs has not yet been reported in the literature.

Here, the authors present an unusual case of an early cerebral vasospasm leading to cerebral ischemia and its late, prolonged recurrence after pure endoscopic fenestration of a temporal arachnoid cyst in a 9-year-old child and discuss its treatment strategy.

Case report

History and clinical presentation

A 9-year -old boy with a 2-month history of intermittent headaches, blurry vision, and progressive double vision was

admitted to the authors' hospital. In the neurological and ophthalmological examination, a right sided abducens nerve palsy and a bilateral papilledema were objectivized. The magnetic resonance (MR) imaging revealed a large temporal AC Galassi Type II in the left middle cranial fossa and also a left hemispheric subdural hygroma (see Fig. 1). The complaints might be caused by the subdural hygroma additionally to the temporal AC in our case [9–11]. Irrespective of this aspect, a decision to perform pure endoscopic cystocisternostomy was made based on the images and the experience in neuroendoscopy in the authors' institution.

Surgical treatment

The patient was placed in the supine position with the head fixed in a Mayfield clamp rotated to the right. After skin incision, a left temporal burr hole was placed according to the neuronavigation trajectory. Under endoscopic view, the isolated AC was identified (Fig. 2A). The endoscopic cystocisternostomy to the left optico-carotid and interpeduncular cistern was performed (Fig. 2B, C, D). The neurovascular structures within the basal cistern, such as left optic and oculomotor nerve and the supraclinoid segment of the left ICA as well as the MCA with its main branches were identified. The thick arachnoid membranes covering basal neurovascular structures were gently perforated and resected. By lateral and caudal widening of the cystocisternostomy to the prepontine area, thin arachnoid vessels were injured causing a significant bleeding (Fig. 2F, G). The bleeding was controlled by permanent irrigation for 30 min. During the final inspection, there was no evidence of further active bleeding or significant hematoma remnants in the subarachnoid space (Fig. 2H) and confirmed a sufficient cystocisternostomy (details see supplementary video).

Postoperative course

The child was postoperatively monitored on pediatric ICU. He showed no neurological deficits and the visual complaints resolved completely within the first hours after surgery. On the first postoperative day, the child suddenly presented with a temporary, right-sided, moderate hemiparesis for approximately 20 min. The immediately performed MRI including MR angiography showed a satisfying result with regard to the fenestrated cyst. Additionally, the preoperative left-sided hygroma was no longer evident; however, a vasospasm in the distal supraclinoid segment of the left ICA and in the medial cerebral artery with its branches resulting in acute small infarctions in the left basal ganglia and thalamus was depicted (details see Fig. 3).

The authors decided for anti-vasospastic therapy with oral nimodipin administration (30 mg every 6 h). Middle arterial pressure (MAP) was continuously monitored and kept above 70 mmHg. On the third postoperative day, the clinical neurological deficits and imaging signs (duplex sonography) of vasospasm resolved completely; however, another MRI on the 15th postoperative day showed progression of vasospasm in the distal segment of the left ICA and its branches (see Fig. 4A). The authors continued the oral administration of nimodipin for the next 14 days up to the 26th postoperative day. During this period and until complete disappearance of radiological signs of vasospasm, serial follow-up MRIs were performed. The final MR imaging on the 26th postoperative day demonstrated a complete resolution of vasospasm in the MR angiography and no new infarctions in the MR imaging (details see Fig. 4). Clinically, no further neurological deficits were found in the further neurological examination.

Discussion

Surgical treatment of ACs

The indication for surgical treatment is still the subject of substantial debate [2–4, 6–8, 12] and neuroendoscopy represents a minimally invasive and safe first-line option in most of the cases of ACs [2–6]. Despite the minimally invasive nature of cyst fenestration, a variety of possible complications may occur after the procedure including subdural hematoma from cyst decompression, CSF leakage, CSF circulation impairment with consecutive hydrocephalus, and postoperative bleeding induced by limited ability in controlling bleedings

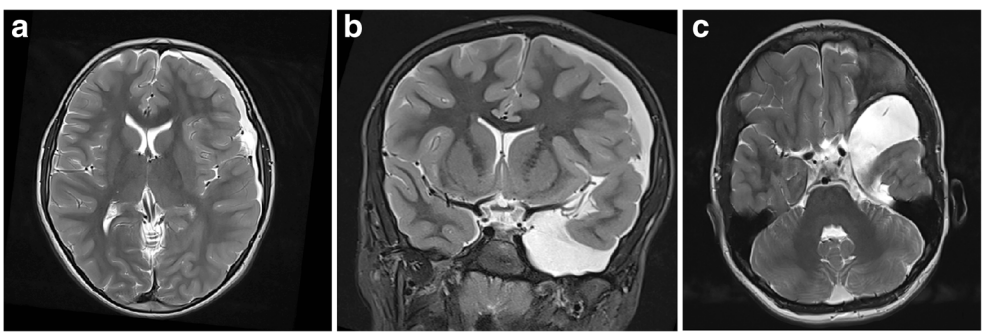

Fig. 1 Demonstrates the MR imaging of a 9-year-old boy with a 2 months history of intermittent headache, blurry vision, and progressive double vision. The MR imaging revealed a large temporal arachnoid cyst in the left middle cranial fossa corresponding to Galassi Type II and also a left hemispheric subdural hygromain axial (**A**, **C**) and coronal (**B**) slides

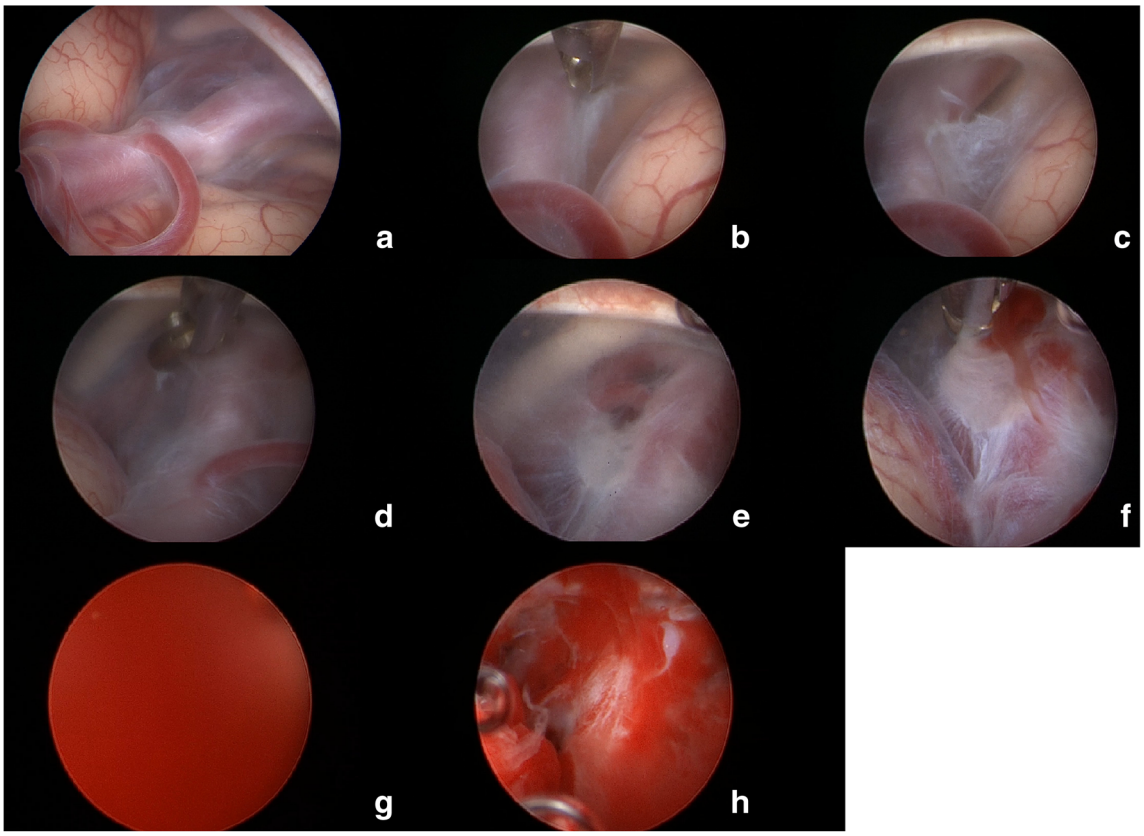

Fig. 2 Shows the different steps of the endoscopic procedure. **a** Endoscopic inspection of the arachnoid cyst. The neurovascular structures within the basal cistern, such as left optic nerve, oculomotor nerve, supraclinoid segment of the left internal carotid artery, and medial cerebral artery with its branches were identified. **b** Cystocisternostomy into the optico-carotid window with a forceps. **c** Final inspection of the stoma. There is a sufficient opening into the basal cistern. **d** Additional cystocisternostomy between the oculomotor nerve and the supraclinoid segment of the left internal carotid artery. **e** Inspection of the stoma. There is still a small arachnoid layer visible. **f** During further procedure a small vessel in the arachnoid membrane was injured by the forceps. **g** Loss of vision during the procedure after bleeding from the injured vessel. **h** Final endoscopic inspection after 30 min permanent irrigation revealed an adequate cyst fenestration and sufficient hemostasis

endoscopically [3, 4, 6]. In the presented case, the authors performed an endoscopic cystocisternostomy in an AC with a left-sided hygroma. A significant intraoperative bleeding was likely to cause delayed vasospasm of the cerebral arteries.

Cerebral vasospasm after endoscopic treatment

Cerebral vasospasm is a very rare event observed in the pediatric population and there is no serious knowledge about

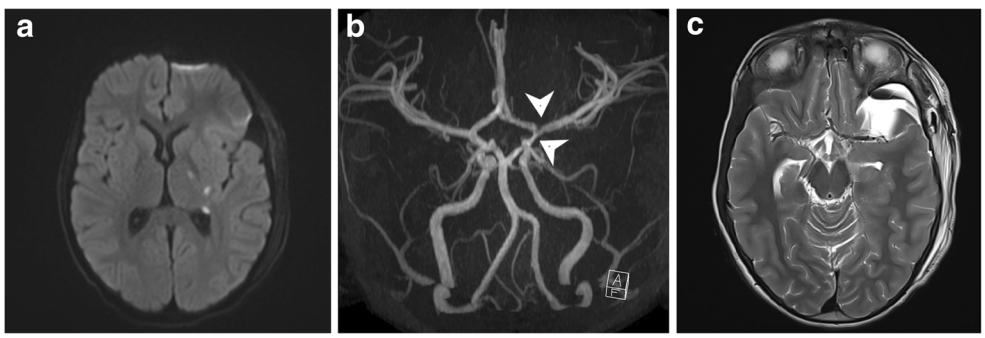

Fig. 3 Demonstrates the postoperative MR imaging on the first postoperative day. **a** FLAIR-MR sequence showing acute small infarction of the left basal ganglia and thalamus. **b** MR angiography depicting vasospasm in the distal supraclinoid segment of the left internal carotid artery and the M1 segment. **c** Axial T2-weighted image showing satisfying result after fenestration of the arachnoid cyst and no postoperative hemorrhage in the surgical field. There is some air collection in the fenestrated arachnoid cyst. The subdural hygroma has completely disappeared

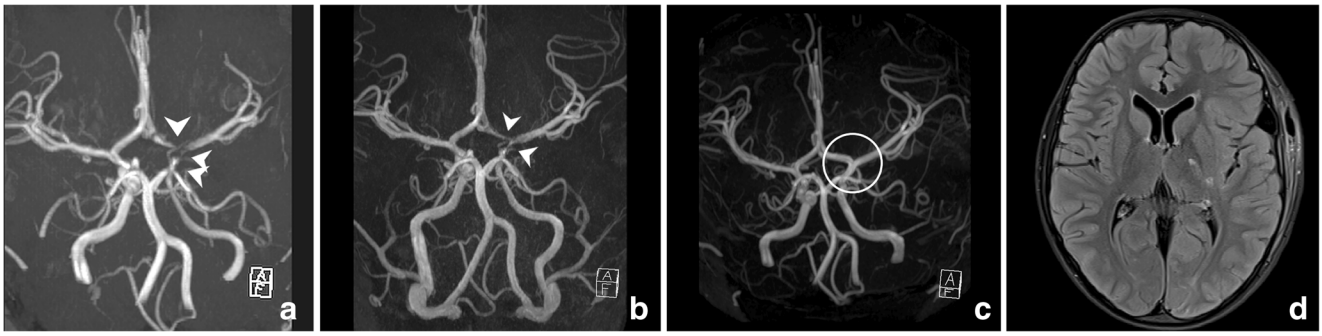

Fig. 4 Demonstrates the follow-up in the MR angiography in the first postoperative month: There was still a significant left-sided vasospasm of the distal supraclinoid ICA segment and its branches on the 15th postoperative day (**A**) and also on 18th postoperative day (**B**); however, the child revealed no symptoms. The final control on the 26th postoperative day demonstrated a complete resolution of vasospasm in the MR angiography (**C**) and no new infarctions in the FLAIR-sequence (**D**)

postoperative vasospasm after AC fenestration and no literature yet. The only case of a delayed ischemia due to vasospasm after microsurgical fenestration of a large AC was described by Auschwitz et al. [1] in 2015. Thereby, chemical meningitis was proposed as possible explanation triggering the occurrence of vasospasm; however, cerebral vasospasm after endoscopic cyst fenestration might be an even more unusual event and is not reported in the literature. In the presented case, the injury of a thin arachnoid vessel induced a significant bleeding. This bleeding could be controlled by permanent irrigation for 30 min; however, the bleeding and its small blood clot remnants might have induced the postoperative vasospasm.

Additionally, in contrast to the well-described cerebral vasospasm in adulthood [13–15], there is rare evidence regarding the course and natural history of pediatric vasospasm. According to selected reports of SAH in pediatric population [16–18], the extravascular hemolyzed blood distributed in the subarachnoid space after surgery might have triggered the disadvantageous cascade. Furthermore, reports of vasospasm after surgical treatment of craniopharyngeoms or transsphenoidal surgery can be found in the literature [19, 20]. Thereby, the postoperative vasospasm might be induced most likely by maneuvering around the major vessels, blood in the subarachnoid space and rapid shifts of fluid intraoperatively.

Another statement of possible mechanism of perfusion impairment related to middle cranial fossa ACs was presented by Sgouros et al. [21] using SPECT scanning. The authors suggested that pure existence of large middle fossa ACs may cause global brain ischemia. Their findings indicate that the presence of middle fossa ACs may cause chronic cerebral hypoperfusion. The sudden decompression of an AC might lead to a reperfusion damage in some cases.

Treatment strategy

Although there is a lack of therapeutic algorithm for pediatric patients, the consequent anti-vasospastic therapy with nimodipin might be an effective and preventive approach. Medication should directly be implemented after the occurrence of subarachnoid blood extravasation and consequently administered during the critical postoperative days.

Conclusions

Cerebral vasospasm after endoscopic fenestration of arachnoid cysts is a very uncommon event, which might induce secondary ischemic events. Once the diagnosis is established, early and consequent medical treatment can contribute to good clinical outcome.

Financial disclosure Joachim Oertel acts as consultant to Karl Storz SE Company. Stefan Linsler received honorarium for presentations from Karl Storz SE Company. Besides this, the authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest, or non-financial interest in the subject matter or materials discussed in this manuscript. No funding was received for this research.

References

1. Auschwitz T, DeCuypere M, Khan N, Einhaus S (2015) Hemorrhagic infarction following open fenestration of a large intracranial arachnoid cyst in a pediatric patient. J Neurosurg Pediatr 15:203–206. https://doi.org/10.3171/2014.9.PEDS14126
2. Oertel JM, Baldauf J, Schroeder HW, Gaab MR (2009) Endoscopic cystoventriculostomy for treatment of paraxial arachnoid cysts. J Neurosurg 110:792–799. https://doi.org/10.3171/2008.7.JNS0841
3. Oertel JM, Wagner W, Mondorf Y, Baldauf J, Schroeder HW, Gaab MR (2010) Endoscopic treatment of arachnoid cysts: a detailed account of surgical techniques and results. Neurosurgery 67:824–836. https://doi.org/10.1227/01.NEU.0000377852.75544.E4
4. El-Ghandour NM (2014) Endoscopic treatment of intraparenchymal arachnoid cysts in children. J Neurosurg Pediatr 14:501–507. https://doi.org/10.3171/2014.7.PEDS13647
5. Hendrix P, Senger S, Griessenauer CJ, Simgen A, Linsler S, Oertel J (2018) Preoperative navigated transcranial magnetic stimulation and tractography to guide endoscopic cystoventriculostomy: a technical note and case report. World Neurosurg 109:209–217. https://doi.org/10.1016/j.wneu.2017.09.185

6. Oertel JM, Baldauf J, Schroeder HW, Gaab MR (2009) Endoscopic options in children: experience with 134 procedures. J Neurosurg Pediatr 3:81–89. https://doi.org/10.3171/2008.11.PEDS0887
7. Bilginer B, Onal MB, Oguz KK, Akalan N (2009) Arachnoid cyst associated with subdural hematoma: report of three cases and review of the literature. Childs Nerv Syst 25:119–124. https://doi.org/10.1007/s00381-008-0728-z
8. Wester K, Helland CA (2008) How often do chronic extra-cerebral haematomas occur in patients with intracranial arachnoid cysts? J Neurol Neurosurg Psychiatry 79:72–75. https://doi.org/10.1136/jnnp.2007.117358
9. Abbas M, Khairy S, AlWohaibi M, Aloraidi A, AlQurashi WW (2018) Bilateral temporal extradural hematoma on top of bilateral temporal arachnoid cyst: first case report and extensive literature review. World Neurosurg 115:134–137. https://doi.org/10.1016/j.wneu.2018.04.040
10. Aydogmus E, Hicdonmez T (2017) Spontaneous intracystic haemorrhage of an arachnoid cyst associated with a subacute subdural hematoma: a case report and literature review. Turk Neurosurg. https://doi.org/10.5137/1019-5149.JTN.20885-17.2
11. Yuksel MO, Gurbuz MS, Senol M, Karaarslan N (2016) Spontaneous subdural haematoma developing secondary to arachnoid cyst rupture. J Clin Diagn Res 10:PD05–PD06. https://doi.org/10.7860/JCDR/2016/21056.8708
12. Bahl A, Connolly DJ, Sinha S, Zaki H, McMullan J (2012) Rapid brain shift, remote site hemorrhage, and a spinal hematoma after craniotomy for a large arachnoid cyst. J Pediatr Neurosci 7:106–108. https://doi.org/10.4103/1817-1745.102568
13. Konczalla J, Kashefiolasl S, Brawanski N, Lescher S, Senft C, Platz J, Seifert V (2016) Cerebral vasospasm and delayed cerebral infarctions in 225 patients with non-aneurysmal subarachnoid hemorrhage: the underestimated risk of fisher 3 blood distribution. J Neurointerv Surg 8:1247–1252. https://doi.org/10.1136/neurintsurg-2015-012153
14. Konczalla J, Kashefiolasl S, Brawanski N, Senft C, Seifert V, Platz J (2016) Increasing numbers of nonaneurysmal subarachnoid hemorrhage in the last 15 years: antithrombotic medication as reason and prognostic factor? J Neurosurg 124:1731–1737. https://doi.org/10.3171/2015.5.JNS15161
15. Weir B, Grace M, Hansen J, Rothberg C (1978) Time course of vasospasm in man. J Neurosurg 48:173–178. https://doi.org/10.3171/jns.1978.48.2.0173
16. Dorsch NW (1994) A review of cerebral vasospasm in aneurysmal subarachnoid haemorrhage part III: mechanisms of action of calcium antagonists. J Clin Neurosci 1:151–160
17. Dorsch NW, King MT (1994) A review of cerebral vasospasm in aneurysmal subarachnoid haemorrhage part I: incidence and effects. J Clin Neurosci 1:19–26
18. Solenski NJ, Haley EC Jr, Kassell NF, Kongable G, Germanson T, Truskowski L, Torner JC (1995) Medical complications of aneurysmal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Participants of the multicenter cooperative aneurysm study. Crit Care Med 23:1007–1017
19. Osterhage K, Czorlich P, Burkhardt TR, Rotermund R, Grzyska U, Flitsch J (2018) Symptomatic vasospasms as a life-threatening complication after transsphenoidal surgery. World Neurosurg 110:180–188. https://doi.org/10.1016/j.wneu.2017.10.027
20. Singh A, Salunke P, Rangan V, Ahuja CK, Bhadada S (2017) Vasospasm after craniopharyngioma surgery: can we prevent it? World Neurosurg 101:208–215. https://doi.org/10.1016/j.wneu.2017.01.115
21. Sgouros S, Chapman S (2001) Congenital middle fossa arachnoid cysts may cause global brain ischaemia: a study with 99Tc-hexamethylpropyleneamineoxime single photon emission computerised tomography scans. Pediatr Neurosurg 35:188–194. https://doi.org/10.1159/000050420