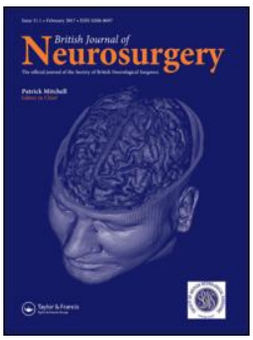

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SHORT REPORT

Delayed ischaemia due to vasospasm after fenestration of a large arachnoid cyst

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ABSTRACT

An 18-year-old patient developed multiple infarcts, nine days after endoscopic fenestration of a large arachnoid cyst. We consider vasospasm to be the most likely cause, presumably triggered by a chemical meningitis. Although mostly seen after subarachnoid haemorrhage, vasospasm can also occur after traumatic brain injury, brain surgery or meningitis.

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Meningitis; cerebral vasospasm; arachnoid cyst; neuroendoscopy

Clinical details

An 18-year-old female presented to our hospital after a mild head trauma. She had fallen off her scooter, after which an epileptic seizure was reported by a nurse, who coincidentally was present at the scene. The patient was unconscious for ten minutes and suffered from posttraumatic amnesia. Neurological examination showed no deficits. The patient had a history of migraine-like headaches. According to her parents, she had always had some difficulties with memory and learning in school. Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) showed a large left-hemispheric arachnoid cyst, which caused significant compression on the brain (Figure 1). There were no signs of subarachnoid haemorrhage or other posttraumatic injuries. Given the large size and the mass effect of the cyst, and the fact that it was a probable cause of the seizure, it was decided to perform surgery. Three days later an endoscopic fenestration of the cyst was performed via a navigated temporal burrhole. The fluid of the cyst appeared slightly clouded. During the procedure we continuously irrigated with Ringer's fluid. The interpeduncular cistern was opened using micro-scissors and coagulation. There were no complications during surgery.

Postoperatively the patient suffered from headaches and nausea. Three days after surgery the intensity of the headache increased and there was occasional vomiting. These complaints were related to a pneumocephalus that was seen on CT scan. We noted a small blood clot in the cavity that we considered asymptomatic (Figure 2). Nine days after surgery, the patient developed a fluctuating left hemiparesis, predominantly of her leg. At some moments she was able to walk around the room, but at other moments there was significant weakness in her leg. Initially, we found no explanation for these phenomena, and even considered a functional disturbance. The next day however, there was also a decline in consciousness. CT showed that the pneumocephalus had diminished. No clear signs of haemorrhage or ischaemia were noted, and the cyst had decreased somewhat in volume. Electroencephalography (EEG) showed diffuse abnormal patterns indicative of an encephalopathic brain, without signs of epileptiform activity. Subsequent blood tests ruled out a metabolic disturbance as a cause for the encephalopathy; serum tests for electrolytes, renal function and liver function were all normal. Puncture of the cyst yielded a pressure of 7 cm H_2O. After drainage of 40 cc of fluid there was no neurological improvement. Biochemical analysis of the cystic fluid showed an increased level of RBCs (12000×10^6/L) and total protein (3.96 g/L), a slightly increased WBC count (238×10^6/L) and a normal glucose concentration (3.4 mmol/L). A culture and gram stain revealed no micro-organisms. PCR for HSV 1, HSV 2, VZV, enterovirus and parechovirus were negative. These results are indicative of a chemical meningitis.

MRI (day 12) unexpectedly showed multiple areas of ischaemia (bifrontal and left temporal) on FLAIR and diffusion imaging weighted series (Figure 2). MR angiography showed severe intracranial vasospasm. The patient was then treated with induced hypertension and nimodipine. At first this did not result in significant clinical improvement, although there was a remarkable decline of the hypodense zones on repeated CT and MRI, suggesting that only part of the hyperintensities on the first MRI eventually resulted in definitive infarction. In later days, the patient seemed to 'awaken' a bit more, although she remained mute for the first six weeks. Afterwards there was very slow neurological recovery. Then, at day 43, the patient was increasingly somnolent again. CT showed a right-sided subdural hygroma and an enlarged ventricular system (hydrocephalus), which was treated with a ventricular-peritoneal shunt. One year after fenestration of the cyst, the patient has significantly recovered. Speech functions have almost normalized and the patient is able to walk, despite a remaining paresis of her left leg. Unfortunately, there are still grave behavioral and cognitive disturbances (e.g. inappropriate and childish behavior).

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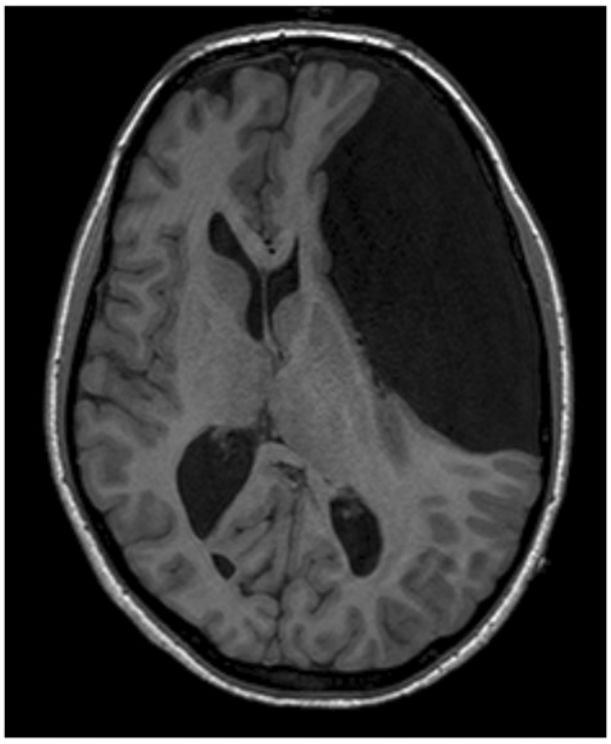

Figure 1. Pre-operative MRI showing a large arachnoid cyst in the left hemisphere with mass effect and compression of the ventricular system.

Discussion

Our patient unexpectedly suffered from delayed cerebral ischaemia after an endoscopic fenestration of a large arachnoid cyst. The cerebral ischaemia was caused by vasospasm as was evident on MR angiography. We found no firm clues about the etiology of the cerebral vasospasm, but consider a chemical meningitis to be the most likely cause, perhaps related to the cystic contents or the breakdown of blood products within the basal cisterns.

Cerebral vasospasm is a major cause of morbidity and mortality after subarachnoid haemorrhage. Incidentally, it also occurs after traumatic brain injury, brain surgery or meningitis.[1] After brain tumour removal, several mechanisms have been postulated in the pathogenesis of vasospasm: accumulation of blood in the basal cisterns, manipulation or damage of blood vessel walls, and release of tumour material.[2] A number of studies specifically reported on vasospasm after resection or rupture of an intracranial cyst (craniopharyngioma, colloid cyst, dermoid tumour).[3] It is suggested that in these cases the content of the cyst caused a sterile inflammation, which subsequently led to the vasospasm. Inflammation, and in particular leukocyte-endothelial cell interactions, have been hypothesized to play a critical role in the pathogenesis of vasospasm.

In our patient, the symptoms of cerebral vasospasm started nine days after fenestration of a large arachnoid cyst. Mixing of the cystic contents with the cerebrospinal fluid (CSF) in the subarachnoid space after fenestration may have induced a sterile inflammation that caused vasospasm and ischaemia. Perhaps the

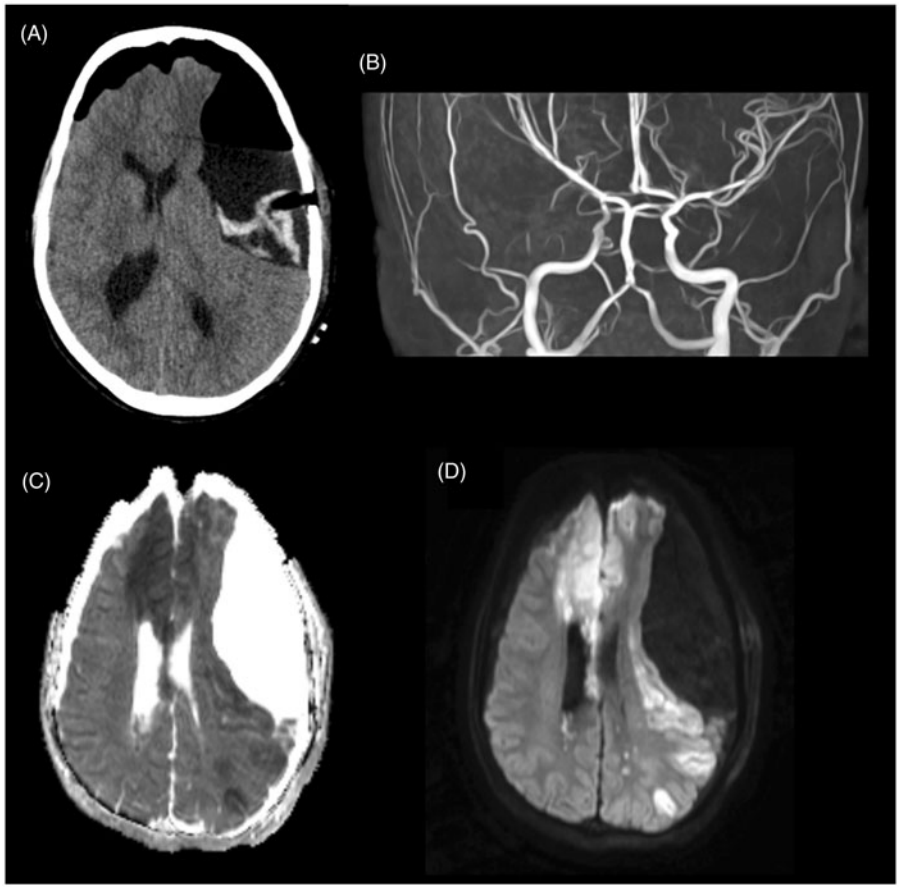

Figure 2. Postoperative images. (A) CT scan at postoperative day 3 showing pneumencephalus. Note also the blood clot under the burr hole. (B) MRA (day 12) indicating vasospasm of vessels in both anterior and posterior circulation. (D) MRI DWI (day 12) showing hyperintense lesions in multiple areas of both hemispheres. (C) Corresponding hypointense lesions on the ADC map, indicative of ischaemia. Note that different vascular territories are involved.

slightly cloudy fluid that was observed during the endoscopic fenestration suggests that the cystic contents contained an increased amount of protein compared to CSF (this fluid was unfortunately not analysed). Alternatively, the presence of blood in the cavity, as unexpectedly seen on the postoperative CT scan, may have contributed to this phenomenon. It is unclear to us why the vasospasm occurred after a delay of nine days, although this time course is well known from patients with a subarachnoid haemorrhage. Interestingly, a similar time course (1-2 weeks) is reported in the literature for patients that clinically declined due to vasospasm after removal of a tumour or colloid cyst.[2,3]

We considered alternative explanations. Post-traumatic vasospasm is not seen in patients with a GCS >12 and a normal CT (i.e. without traumatic blood).[4] Non-traumatic subarachnoid haemorrhage also seems an unlikely cause for the vasospasm, because there was no intraoperative arterial bleeding, and postoperative CT showed no evidence of arterial or subarachnoid haemorrhage. Lastly, we ruled out an infectious meningitis as a likely cause, because cultures remained negative and the CSF showed only a slightly elevated WBC and a normal level of glucose.

To our knowledge this is the first report on vasospasm after fenestration of an arachnoid cyst. The vasospasm likely resulted from a chemical meningitis, perhaps initiated or aggravated by the cystic contents or the presence of blood in the cavity. This case reminds us to remain vigilant in the postoperative period after endoscopic cisternal fenestration.

Acknowledgments

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Statement of ethics

Informed consent was obtained from the patient's parents.

Disclosure statement

No potential conflict of interest was reported by the authors.

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