

## Intracranial Infectious Aneurysms from Postoperative Meningitis: Two Case Reports

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**Abstract:** Infectious intracranial aneurysms from postoperative meningitis is extremely uncommon. This study aims to alert that infectious intracranial aneurysms and lethal subarachnoid hemorrhage can be following the un standard sterilized surgery due to postoperative meningitis, by providing the general clinical profiles of 2 cases. Two cases of infectious intracranial aneurysms from postoperative meningitis were reported. One was about an adolescent male, who contracted meningitis after debridement of head trauma and whose aneurysm was located on ACOM and saccular in shape, while the other one was about an old male who was afflicted meningitis after microvascular decompression surgery for hemifacial spasm and whose aneurysm was located on the initial segment of basilar and was fusiform in shape. Both patients died from repeated subarachnoid hemorrhage. Infectious intracranial aneurysms from postoperative meningitis are catastrophic and therefore, aggressive approaches should be taken for treatment.

**Key words:** Infectious intracranial aneurysms, postoperative meningitis, subarachnoid hemorrhage, microvascular decompression, anterior communicating artery, neck rigidity, headache

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### INTRODUCTION

Infectious intracranial aneurysms are infrequent, with vast majority of them occurring in the setting of bacterial endocarditis and localized on the distal segments of cerebral arteries (Kannoth *et al.*, 2007; Phuong *et al.*, 2002; Clare and Barrow 1992; Chun *et al.*, 2001). Here, we report 2 cases where infectious intracranial aneurysms were situated on the trunks of cerebral arteries and caused by refractory meningitis following Microvascular Decompression (MVD) for hemifacial spasm and incompletely wound cleaning of cranial wound.

### MATERIALS AND METHODS

**Case 1:** This was a 13 years old right-handed boy who was admitted into the hospital for having headache and fever for 7 days. On Feb. 9, 2006, the boy was hit in the left frontotemporal area of the head by a sharp pointed tip of a forked metal farming tool, which was used for picking up feces of cattle, leading to scalp laceration of the region. He did not lose his alertness with this accident. Debridement and closure of the scalp wound was carried out at the regional county hospital of his living. No imaging investigations were given. The scalp wound healed well 7 days later.

On Feb. 26, 2006, 17 days after the accident, the boy began to have irregular fever and headache. The body

temperature reached 38.6°C. His headache was sometimes associated with vomiting. He was treated with penicillin  $8 \times 10^7$  unit day<sup>-1</sup> intravenously, for 7 days without remarkable effect. So, on March 5, 2006, 26 days after the accident, he was admitted into our hospital complaining of headache and fever.

On admission, the boy was alert without local neurological deficit, but he was found having neck rigidity and positive Kernig's sign. The body temperature was 38.9°C.

Lumbar puncture was done and CSF investigation showed increased protein and decreased sugar and chloride concentrations. White cells counting of CSF was  $800 \times 10^9$  L<sup>-1</sup> with neutrophil 80%. Routine CT scanning showed mildly depressed skull fracture in the left lateral frontal area, which did not need to elevate. Initial MRI done on March 6, 1 month after the accident, showed local cerebral contusion and edema in the lateral frontal lobe adjacent to the lateral fissure without aneurysmal changes shown in the Anterior Communicating (ACOM) artery complex (Fig. 1a).

He was diagnosed with bacterial meningitis and thereafter, treated with multiple antibiotics, but his intracranial infection could not be controlled effectively with temperature over 38°C.

On March 14, 2006, 38 days after the accident, he suddenly developed blindness with his left eye and thus, another MRI was again performed showing a suprasellar

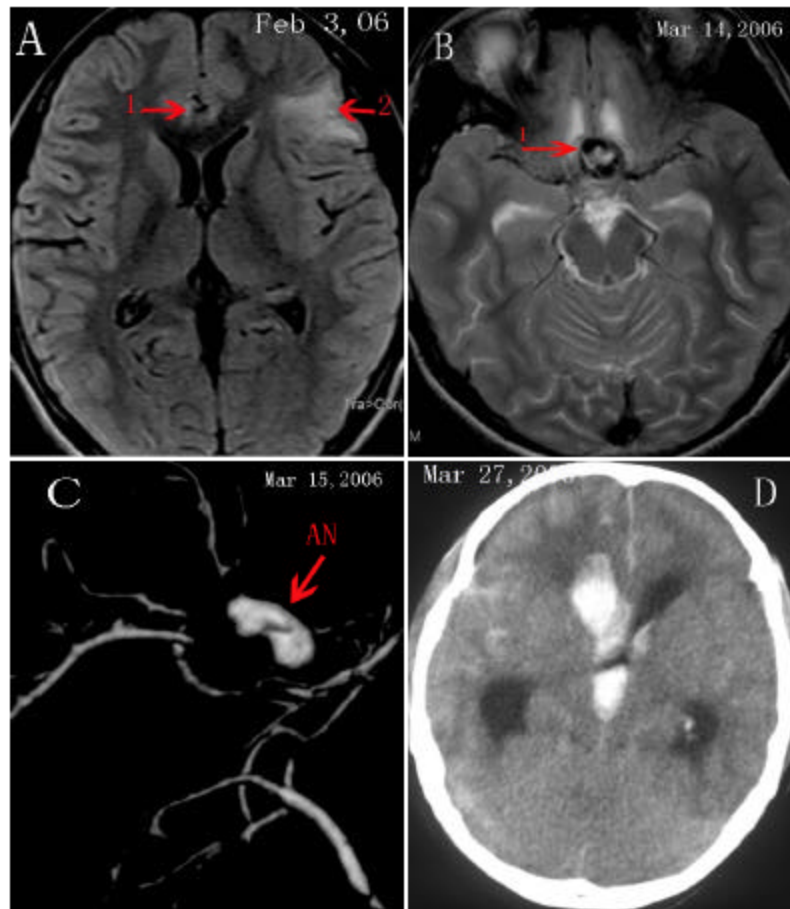


Fig. 1: Serial images showing the chronological process of an infectious aneurysm at the Anterior Communicating artery (ACOM) complex in a 13-years old boy, a) MRI done 30 days after head trauma due to meningitis showing local cerebral contusion and edema (2 arrow) in the lateral frontal lobe adjacent to the lateral fissure without aneurysmal changes shown seen at ACOM (1 arrow), b) MRI done 8 days later when the patient suddenly developed blindness with his left eye, showing a suprasellar lesion of 1.0×1.2 cm highly suspected to be an aneurysm of ACOM (1 arrow), c) CTA done the next day showing a saccular aneurysm of ACOM (AN arrow). D: CT done 1 month after having manifestations of meningitis when the patient suddenly developed severe headache and vomiting after a sneeze, showing SAH that ruptured into the ventricular system leading to his death 3 h thereafter

lesion of 1.0×1.2 cm in size, with long-short T1, long T2 and high Flair signal, highly suspected to be an aneurysm of ACOM. The surrounding rectus gyrus of the lesion was edematous (Fig. 1b). The next day, a CTA was carried out showing a saccular aneurysm of ACOM (Fig. 1c).

Considering that the aneurysm in this boy resulted from refractory meningitis and thus, was infectious in etiology and the wall of it could be too fragile to be clipped. So cerebral angiography and possible endovascular embolization was planned to be done as soon as possible, but was postponed due to uncontrolled meningitis.

On the morning March 27, 2006, 1 month after having manifestations of meningitis, the boy suddenly developed severe headache and vomiting after a sneeze. He got into coma with pupil on both sides enlarged without light reflex. Another CT was done showing SAH that ruptured into the ventricular system (Fig. 1d) leading to his death 3 h thereafter. No autopsy was done due to the refusal of the parents of the boy.

**Case 2:** This is a 60 years old male was admitted into our hospital on May 30, 2008 for persistent headache and irregular fever after MVD for hemifacial spasm.

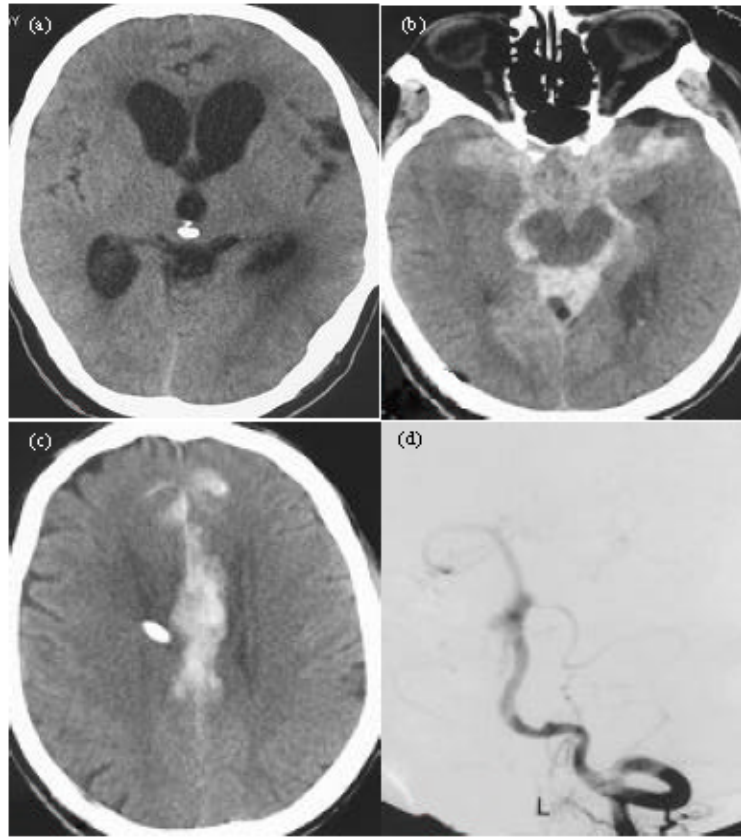


Fig. 2: Serial images showing the chronological process of an infectious basilar artery aneurysm in a 60-year-old male. a: CT done 8 months (June 17) after MVD showing hydrocephalus due to meningitis leading to the placement of external right lateral ventricular drainage b and c: CT done on July 8 showing SAH with rupturing into ventricular system. d: DSA done the next day after the occurrence of SAH showing a fusiform aneurysm with irregular configuration at the initial segment of the basilar artery

In October, 2007, about 7 months before admission, this patient underwent MVD surgery via right retro sigmoid approach at his county hospital for right hemifacial spasm, which he had suffered from it for 7 years. According to the patient, his wife and his son, the surgery regarding to relieve facial spasm was completely successful and no neurological complications such as hearing loss, tinnitus and facial paralysis followed the surgery. The wound healed on time and suture was removed on the 7th day after surgery. There was no Cerebrospinal Fluid (CSF) leakage. The patient was discharged after suture removal. After discharge, he had been on pain-killer pills for persistent mild-to-moderate headache and thus, according to his son, no fever had been discerned until Feb. 2008 when persistent irregular fever, headache and occasional vomiting began. Then, he was admitted into the same county hospital where lumbar puncture and CSF analysis was done showing evidence of bacterial meningitis. He was treated with multiple antibiotics but ineffective for the meningitis.

In April 2008, 6 months after MVD, the patient was transferred to the General Hospital of the People's Liberation Army of China in Beijing. There, multiple MRI scans were done and multiple CSF tests and bacteriology investigations were carried out, also pointing to bacterial meningitis. He was tried a large variety of antibiotics again, but failed for the treatment of meningitis.

Then, on May 30, 2008, the patient was transferred to our hospital for the refractory meningitis. On admission, he continued to have headache, irregular fever and occasional vomiting. The fever fluctuated around 38°C, sometime, exceeded 39°C. He had apparent neck rigidity and positive Kernig's sign. Serial MRI scans and CT scans including with contrast were carried out, but failed for possibly finding a small abscess in the MVD region as in a case of its kind before. MRA was normal. Blood cell analyses showed mild anemia, otherwise normal. Multiple blood and CSF cultures for bacteriology study showed negative results for Gram-positive, Gram-negative and

anti-acid staining bacteria and fungi. Serial aerophobic bacterial studies were also done with negative results. Systemic investigations including chest CT scans, abdominal and pelvis CT scans showed no evidence with infection. Serial CSF analysis showed that white cell count was at  $80 \times 10^9 \sim 2100 \times 10^9 \text{ L}^{-1}$ , with neutrophils dominating, accompanied by increased protein and chloride concentrations. Multiple antibiotics and steroids were tried again therefore, but failed.

On June 17, external right lateral ventricular drainage was put on due to hydrocephalus with increased intracranial pressure as shown on CT (Fig. 2a). The drainage had to be in place continually because of uncontrolled meningitis, which prevented the placement of shunt system for hydrocephalus.

On 6 July, 8 months after MVD, the patient suddenly had severe headache and then lost consciousness. Blood was showed out in the drainage. An emergency CT scan was done showing widespread severe SAH with rupturing into ventricular system (Fig. 2b, c).

The next day after the occurrence of SAH when the patient recovered his consciousness and was in relatively stable state, a 4-vessel angiography was carried out showing a fusiform aneurysm with irregular configuration at the initial segment of the basilar artery (Fig. 2d).

We did not know how to treat such fusiform basilar aneurysm as a result of long term refractory meningitis as in this case. As, we were hesitating to try to implant sent for reducing the tendency of bleeding of the aneurysm, the patient died of another SAH 3 days later.

## RESULTS AND DISCUSSION

The diagnosis of infectious intracranial aneurysms were established in the cases based on that: meningitis in the 2 cases were confirmative according to history of cranial surgery, persistent fever, headache and meningeal irritating signs after surgery and CSF chemistry and cytology, though CSF bacteriology was negative; imaging studies earlier in the period of the disease showed normal cerebral vascular structures without evidence of preexisting aneurysms and thus, the aneurysms in these 2 patients occurred later in process of meningitis; the irregular configuration and the proximal location of the aneurysms are in agreement with the features of such kind as previously reported (Kannoth *et al.*, 2007, 2008). A very special feature in our cases is that the meningitis was as a result of cranial surgery. In the 1st case, we think the meningitis was due to inappropriate wound cleaning, while in the 2nd case, the problem could be due to unstandard sterilization of inserts (Teflon) used in MDV. The meningitis in these 2 cases could be avoided if

the surgical procedures were carried out by highly trained professionals and in a better condition hospitals.

Infectious intracranial aneurysms can be classified into 2 subgroups according to infectious origins: those related with bacterial endocarditis and those from neighboring septic foci or meningitis (Kannoth *et al.*, 2007; Phuong *et al.*, 2002; Clare and Barrow, 1992). Compared with infectious intracranial aneurysms due to endocarditis, infectious intracranial aneurysms from meningitis is less common (Kannoth *et al.*, 2007; Phuong *et al.*, 2002; Clare and Barrow, 1992) and have different clinical features, imaging characteristics and natural history. Infectious intracranial aneurysms from meningitis tend to occur in the vertebrobasilar territory, are more proximal and bleed, leading to SAH and death, while under medical management, whereas those related to endocarditis are located more often in carotid territory, tend to be distal and are less prone to fatal bleeding (Kannoth *et al.*, 2007). The cases are in agreement with this. The two aneurysms in both cases were proximal with one at the initial segment of basilar artery and one at ACOM, ruptured while on medical treatment leading to death.

Since infectious intracranial aneurysms from meningitis is very fatal, it is important to find them as early as possible for the implementation of appropriate treatment. First of all, it should be given alertness to patients with refractory meningitis that fatal infectious intracranial aneurysms could be developed and attention should be paid to the screening of them. Multi-slice CT angiography with 3 dimensional reconstruction and MR angiography have provided unique opportunity to non-invasively screen high risk patients with meningitis to detect infectious intracranial aneurysms early. For instance, the aneurysm in the 1st case had been shown on MRI done before bleeding.

Modalities of treating infectious intracranial aneurysms include conservative, endovascular and microsurgical (Kannoth *et al.*, 2007; Chun *et al.*, 2001; Eddleman *et al.*, 2008), but actually when these patients present, they generate challenging problem with regard to decision-making about the choice of treatment modes. As reported in the literature (Kannoth *et al.*, 2007) and in our cases, infectious intracranial aneurysms from meningitis is much more fatal than those from endocarditis and leading to death rupture usually occur while on medical treatment, a more aggressive attitude should be taken for the treatment of them. In our 1st case, we put too much effort on the control of meningitis, leading the delay of treatment of the aneurysm that led to the loss of opportunity for treatment; while in the second case, stenting the fusiform aneurysm could have avoided the occurrence of catastrophic rebleeding.

### **CONCLUSION**

Infectious intracranial aneurysms from postoperative meningitis are rare. They are usually located on the proximal segments of cerebral arteries, irregular in configuration and very fatal due to their high tendency of rupture. Aggressive attitude and approaches should be taken for treating them.

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