A giant middle fossa arachnoid cyst with spontaneous rupture into the subdural space: conservative observation or neuroendoscopic fenestration

Siyi Xu, Chunlong Zhong*, Yong Wang, Qizhong Luo, Jiyao Jiang

Department of Neurosurgery, Renji Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200127, China
drchunlongzhong@126.com

Abstract: Middle fossa arachnoid cyst (MFAC) is the most common of arachnoid cysts. However, the treatment of MFAC is still a matter of controversy. We report a case of a giant left-sided MFAC with spontaneous rupture into the subdural space that was discovered in a 4-year-old boy who had symptoms of an occasional headache. Initially, we chose to pursue conservative observation. Unfortunately, nine months later, the boy suffered from an aggravated headache and severe vomiting without any apparent causes. At that time, we performed surgery with a neuroendoscope to drain and fenestrate the cyst to obtain nearby cystocisternal communications. The patient made a complete recovery, and the substantial reduction of the cyst was revealed postoperatively on radiography. The thickened cyst membrane and yellowish-green cerebrospinal fluid that we observed intraoperatively led us to conclude that this type of MFAC may not be self-limiting through its spontaneous rupture into the subdural space; on the contrary, the intracranial pressure fluctuation and potential bleeding when these cysts burst may be life threatening to the patient. Neuroendoscopic fenestration may be an acceptable and minimally invasive option for the management of symptomatic MFACs.

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Key words: middle fossa arachnoid cyst; neuroendoscopic fenestration; conservative observation; minimal invasion

1. Introduction

Arachnoid cysts are CSF-containing cystic lesions of an unclear etiology (Gupta et al., 2004). Middle fossa arachnoid cysts (MFACs) are the most common of the arachnoid cysts, accounting for approximately one-half of adult cases and one-third of pediatric cases (Li et al., 2013). However, the treatment of MFAC is still a matter of controversy. In this report, we report a case of a giant left-sided MFAC with spontaneous rupture into the subdural space that was discovered in a 4-year-old boy who had symptoms of an occasional headache. Initially, we chose to pursue conservative observation. Unfortunately, nine months later, the boy suffered an aggravated headache and severe vomiting without any apparent causes. At that time, we performed a neuroendoscopic fenestration for the cyst to obtain nearby cystocisternal communications and the patient made a complete recovery.

2. Clinical Data

This case involves a 4-year-old boy who occasionally complained of headaches. CT and MRI scans were performed and showed that there was a giant left-sided MFAC filling the entire middle fossa, with displacement of the ipsilateral frontal and parietal lobes. There was also mass effect with substantial midline shift of the intracranial contents (Fig. 1 a, b, c and d, e, f). In the T2 weighted images, a layer of membrane that separated the cyst cavity was observed and may have been composed of the ruptured arachnoid membrane, indicating that the cyst may have ruptured into the subdural space (Fig. 1 e). Considering the patient's young age, the lack of serious symptoms, and the reports that some MFACs could resolve spontaneously by rupturing, we chose conservative observation. Unfortunately, nine months later, the boy began to suffer from a severe headache without any apparent causes. Seven days later, the headache worsened and was associated with two episodes of severe vomiting before the patient was sent to the emergency room. A new CT scan showed no changes with the cyst compared to the previous CT images.

To relieve the symptoms, we performed a neuroendoscopic fenestration with a 0° lens rigid neuroendoscope (HOPKINS II, Karl Storz, Germany) for the patient under general anesthesia. A temporal 5 cm curved incision was made, and a bone window that measured 2 cm × 2 cm was created above the zygomatic arch. We observed that both the inner membrane and the outer membrane of the cyst were markedly thickened and opaque, appearing to be a sort of pseudo-membrane. We also found that there was a gush of yellowish-green cerebrospinal fluid (CSF) released under high pressure when the outer membrane was incised. Furthermore, we noticed that a layer of ruptured arachnoid membrane, which was thin and hyaline, separated the cyst's cavity as the MRI scan had

shown previously. We detected some laceration on the ruptured arachnoid membrane through which the original middle fossa arachnoid cyst communicated with the subdural space and both of them formed the whole cyst cavity. Some sections of the outer membrane and most of the ruptured arachnoid membrane were resected. Subsequently, several non-vascularized areas were selected and 3 openings of more than 1 cm each were made to produce sufficient communication with the neighboring CSF spaces toward the basal cisterns (Fig. 2). A successful cystocisternostomy was confirmed by the visualization of the CSF pulsations. Finally, the endoscope was removed, and closure was performed conventionally without any drainage.

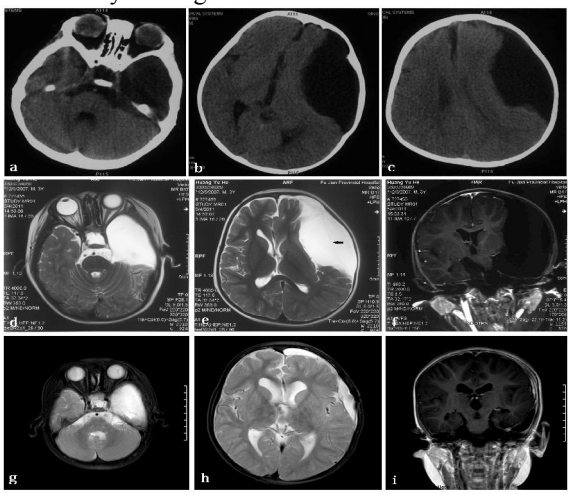

Fig. 1 Brain CT and MRI images: (a, b, c): CT scans showing a giant left-sided middle fossa arachnoid cyst at the initial evaluation. (d, e, f): Axial T2-weighted and coronal T1-weighted MRI showing a giant left-sided middle fossa arachnoid cyst at the initial evaluation. There is a layer of ruptured arachnoid membrane separating the cyst cavity from the subdural space (arrow). (g, h, i) : Axial T2-weighted and coronal T1-weighted MRI approximately 3 months postoperatively showing the substantial reduction of the middle fossa arachnoid cyst and the almost complete resolution of the mass effect.

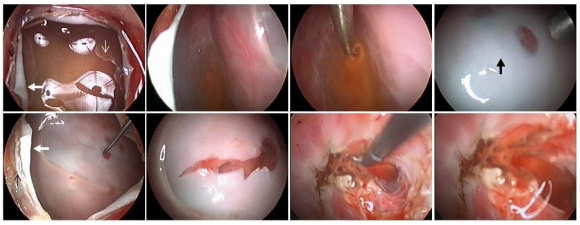

Fig. 2 Intra-operative photographs show the subdural spaces communicating with the cyst cavity through some laceration and the contained yellowish-green CSF. Moreover, the cyst membrane was markedly thickened and opaque (dura matter: small black arrow; outer membrane: large white arrow; media membrane: large black arrow), and the ruptured arachnoid membrane is thin and hyaline (small white arrow). Subsequently, nearby cystocisternal communications were made toward the basal cisterns.

An MRI was performed three months after the operation and revealed that the MFAC was substantially reduced and that the mass effect had almost disappeared (Fig. 1 g, h, i).

3. Discussion

First described by Bright in 1831, MFACs have subsequently been reported by a number of authors; however, their etiopathogenesis and natural history remain obscure (Gupta et al., 2004; Li et al., 2013). A large majority of patients suffering from MFAC remain asymptomatic until symptoms occur as a result of the enlargement of their cysts, obstruction of the CSF pathways with resulting hydrocephalus, or other complications. Acute intracranial hypertension may be precipitated by an intracystic bleed, subdural hematoma (probably due to bleeding from the bridging veins), or a subdural hygroma as the result of cyst rupture. Conversely, the spontaneous resolution of arachnoid cysts has also been reported, both with and without a history of preceding trauma (Spacca et al., 2010).

Initially, the patient had only a minor headache prior to the apostasis, and his arachnoid cyst was detected incidentally. MRI images also revealed a layer of ruptured arachnoid membrane separating the cyst cavity from the subdural space, indicating that the cyst may have ruptured with subdural effusion. The treatment of asymptomatic arachnoid cysts is still a matter of controversy. Based on the patient's age and the lack of serious symptoms, we chose to treat the patient with conservative observation. However, nine months later, the patient developed intracranial hypertension and signs of meningeal irritation. We inferred from these symptoms that the cyst have ruptured again and led to the intracranial pressure fluctuation.

To the best of our knowledge, as MFACs' proximity to the basal cisterns makes them particularly amenable to cystocisternostomy, it has been well accepted that the best operative intervention for symptomatic MFAC is to drain and fenestrate the cyst using neuroendoscopic techniques.

Intraoperatively, we observed yellowish-green cerebrospinal fluid in the cyst cavity, which indicated that prior bleeding had occurred repeatedly and that even a subdural hematoma might form in the future. Therefore, we concluded that this type of MFAC may not be self-healing through spontaneous rupture into the subdural space; on the contrary, the

intracranial pressure fluctuation and potential bleeding when these cysts burst may be life-threatening for patients.

The currently accepted view is that fenestrations for cystocisternostomy of MFACs should be as large as possible without injuring the surrounding structures. However, the pathophysiology and epidemiology appear to support the hypothesis that smaller fenestrations may achieve the same therapeutic goals (Johnson et al., 2011). In some cases, it has been reported that the surgical effect would be improved if the openings were created between the tentorium and oculomotor nerve or between the internal carotid artery and the oculomotor nerve (Greenfield and Souweidane, 2005). However, in this patient, the thickened cyst membrane may have been caused by protein deposition from the bleeding of the ruptured vessels in the arachnoid membrane. As a result, the media cyst's membrane was too thick to clearly visualize the deep vital structures. Therefore, we selected several nonvascularized areas and created 3 openings of more than 1 cm with sufficient communication of the neighboring CSF spaces toward the basal cisterns. Finally, during follow-up, the patient's symptoms subsided, and the cyst's reduction was revealed by radiography.

4. Conclusion

In our opinion, some MFACs will self-heal by cyst rupture, but there may be other patients whose cyst membrane is sufficiently thick to prevent the communication of the cyst with the nearby cisterns and the intracranial pressure fluctuation and potential bleeding when these cysts burst may be life-threatening. We conclude that neuroendoscopic fenestration may be an acceptable and minimally invasive option for the management of symptomatic MFACs.

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***Corresponding Author:**
Dr. Chunlong Zhong,
Department of Neurosurgery,
Renji Hospital, Shanghai Jiao Tong University School of Medicine,
Shanghai 200127, China.
E-mail: drchunlongzhong@126.com

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