Cavernous hemangioma of the third ventricle: a case report and review of the literature

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Abstract

Background: Although cavernous hemangiomas (CHs) can be found anywhere in the central nervous system, CHs of the third ventricle have been reported in only 29 patients (including our case). In the current case report, we discuss the clinical characteristics and surgical outcome of CHs of the third ventricle.

Case presentation: A 64-year-old female was admitted to our emergency room with a sudden decreased level of consciousness. Brain imaging studies demonstrated a multi-lobulated hemorrhagic mass in the third ventricle. The lesion was removed via the transcallosal-interforniceal approach and pathologically diagnosed as CH. Postoperatively, the patient had a transient neurological deficit due to hypothalamic injury and recovered to the normal status at 2 months after the operation. In the review of 29 cases, the mean age of the patients was 40 years with a slight female preponderance (female/male, 17/12). The majority of the patients complained of a mass effect with signs of increased intracranial pressure; only one case was asymptomatic. Gross total resection was achieved in 81% of the cases. Around 80% of the patients were asymptomatic or improved from the initial symptoms. Mortality rate was 6.9% and the most common complication was hydrocephalus.

Conclusions: As demonstrated in the review of the previous reports, the outcome is favorable after surgical excision for CH of the third ventricle. Hence, surgical excision appears to be the treatment of choice for CH located in the third ventricle, which tends to grow rapidly resulting in a mass effect.

Keywords: Cavernous hemangioma, Complication, Outcome, Surgery, Third ventricle

Background

Cavernous hemangiomas (cavernomas, cavernous angiomas, cavernous malformations; CH) are vascular hamartomas that are reported to be found at any location in the central nervous system (CNS). Due to the increased use of computerized tomography (CT) scan and magnetic resonance imaging (MRI), more CHs have been diagnosed in recent years. However, intraventricular location of CHs is uncommon, and the incidence of intraventricular CHs has been reported to be only about 2.5 to 10.8% of all intracranial CHs [1,2]. The most frequent location of intraventricular CHs is the lateral ventricle and involvement of the third ventricle is quite rare. Based on the review of Medline database (PubMed, http://www.ncbi.nlm.nih.gov/PubMed), only 29 cases (including our case) of CH of the third ventricle have been reported [3-20].

We present the case of a patient who had a CH in the third ventricle that was resected through the transcallosal interforniceal approach. In addition, we also review the previously reported cases and discuss their clinical characteristics and surgical outcomes.

Case presentation

A 64-year-old female was admitted to our emergency room with a sudden decreased level of consciousness. Except for an intermittent and mild degree headache, there was no specific history of head trauma and medical illness. On neurological examination, she showed a drowsy mentality with Glasgow Coma Scale score of 14/15 and the right homonymous hemianopsia. She did not have motor/sensory and cranial nerve deficits, and cerebellar signs. There were no abnormal laboratory findings. Non-contrast CT scan showed a heterogeneous

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hyperattenuated hemorrhagic mass within the third ventricle compressing the hypothalamus, without definitive hydrocephalus (Figure 1). Brain MRI revealed a 40 × 30 × 28 mm sized multi-lobulated mass with a recent hemorrhage in the third ventricle, which extended to the foramen of Monro and hypothalamus. There was no definite contrast enhancement (Figure 2).

Right-side interhemispheric, transcallosal interfornicial approach was used for removal of the lesion. At surgery, the lesion was found to be a red colored, multi-lobulated mass, which had numerous vascular channels and multistaged hemorrhage. Although there were severe adhesions between the base of the lesion and the basilar arterial system, gross total removal of the lesion was possible due to the presence of the discrete sticky hemosiderin rim, which allowed differentiation of the lesion from the surrounded normal parenchyma (Figure 3). To prevent hypothalamic injury, the resection of hemosiderin-stained tissue was restricted to the minimum.

Histopathological examination of the lesion revealed a CH composed of large, irregularly dilated, blood-filled vascular channels lined by flat endothelium (Figure 4). Postoperatively, the patient developed transient diabetes insipidus, somnolence, and general weakness due to hypothalamic injury, but these symptoms gradually disappeared with conservative treatment. Finally, she recovered to the normal status at 2 months after the operation.

Discussion

CHs are vascular hamartomas which are reported to be found anywhere in the CNS. However, intraventricular CHs are rare and their incidence was reported to be only about 2.5 to 10.8% of all cerebral cavernous malformations [1,2]. CHs may be diagnosed based on symptoms of acute hemorrhage, seizures, or progressive neurologic deficits. Chadduck et al. [21] reported that there was no

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**Figure 1** Axial non-contrast CT images show a large and heterogeneously hyperattenuated hemorrhagic mass in the suprasellar area, with dilatation of the anterior part of the third ventricle (arrow).

**Figure 2** Preoperative MRI images. Sagittal T₁-weighted (A) and post-contrast T₁-weighted (B) images show a non-enhancing hemorrhagic mass in the anterior third ventricle and hypothalamic area. Axial T₁-weighted (C), T₂-weighted (D), and susceptibility weighted (E) images demonstrate a typical cavernous malformation with heterogeneous signal intensity and hemosiderin rim indicating mixed acute or subacute stage hemorrhage.
difference between the clinical symptoms and signs of intraventricular CHs and parenchymal CHs. However, because of the rarity, the natural history and clinical features of CHs located in the third ventricle have not been fully investigated and there are no definite recommendations for its management.

Overall, 29 patients with a well-described CH in the third ventricle have been reported in the literature, including our case [3-20] (Table 1), with a slight female preponderance (female/male ratio, 58/42%). The median age of the patients was 40 years (range, 8–64 years) and 6 patients were of the pediatric age group (21%). The most frequent initial clinical symptoms included a mass effect, with signs of increased intracranial pressure (headache, nausea, vomiting, visual disturbance, memory impairment and signs of hypothalamic dysfunction) in 23 patients (79%). Intraventricular hemorrhage from the lesion occurred in 3 cases (10%) and seizures in 2 cases (7%); only one patient was asymptomatic. This higher incidence of mass effect symptoms may be because of the direct compression of the surrounding
| Authors            | Publication year | Age (year) | Sex | Symptom                                      | Size (cm) | Approach | Extent of resection | Outcome | Postoperative complication |
|--------------------|------------------|------------|-----|----------------------------------------------|-----------|----------|---------------------|---------|--------------------------|
| Vaquero et al. [3] | 1980             | 18         | F   | Diplopia                                     | –         | TC       | GTR                 | Improved|                          |
| Pozzati et al. [4] | 1980             | 31         | F   | Headache, vomiting                           | –         | TV       | GTR                 | Improved|                          |
| Lavyne et al. [5]  | 1983             | 48         | F   | Headache, memory impairment                 | 1.5       | TC + TV SC| PR                  | Not improved | HDC, IVH                |
| Amagasa et al. [6] | 1984             | 40         | M   | Homonymous hemianopsia, endocrine function deficit | –         | IH + TLT | GTR                 | Improved|                          |
| Harbaugh et al. [7]| 1984             | 44         | F   | Headache, vomiting, IVH                      | 2         | TC + TV  | GTR                 | Improved| HDC                      |
| Yamasaki et al. [8]| 1986             | 9          | M   | Headache                                     | 2.5       | –        | GTR                 | Improved|                          |
|                   |                  | 15         | F   | Lower temporal quadrantopsia                 | 1.5       | –        | PR                  | No symptom |                          |
|                   |                  | 36         | M   | Headache, vomiting, mental change            | 2.5       | –        | PR                  | Improved|                          |
| Voci et al. [9]    | 1989             | 19         | F   | IVH                                          | –         | TC       | GTR                 | Improved|                          |
| Ogawa et al. [10]  | 1990             | 16         | M   | Headache, nausea                             | 2         | IH + TLT | GTR                 | No symptom|                          |
|                   |                  | 40         | M   | Homonymous hemianopsia, endocrine function deficit | 2         | IH + TLT | GTR                 | Improved|                          |
| Katayama et al. [11]| 1994            | 9          | F   | Seizure                                      | –         | IH + TLT | PR                  | Death   |                          |
|                   |                  | 50         | –   | –                                            | –         | –        | –                   | Improved|                          |
|                   |                  | 45         | F   | IVH                                          | –         | –        | –                   | Not improved | Vegetative state |
|                   |                  | 49         | M   | Visual field defect, endocrine function deficit | 2         | –        | –                   | Improved|                          |
|                   |                  | 47         | F   | Memory impairment                            | 3         | SC + TVI | GTR                 | Improved| Transient DI, Recurrence |
| Sinson et al. [12] | 1995             | 43         | F   | Headache, memory impairment                 | 3         | IH + TC IF | GTR                | Death   |                          |
|                   |                  | 36         | F   | Memory impairment, weight gain               | 3         | IH + TC IF | GTR                | Not improved | HDC |
|                   |                  | 52         | F   | Headache, nausea                             | 3.5       | TCo      | GTR                 | Improved|                          |
|                   |                  | 32         | F   | Headache, vomiting, diplopia                 | 2         | IFT + SCbll | GTR                | Improved|                          |
| Reyns et al. [13]  | 1999             | 42         | M   | Seizure                                      | 2.5       | TCo + TVI | PR                  | Improved| Recurrence              |
| Crivell et al. [14]| 2002             | 38         | M   | Memory impairment, gait disturbance, headache, vomiting | – | TCo + TVI | GTR                | Improved|                          |
| Wang et al. [15]   | 2003             | 62         | F   | Gait disturbance                             | –         | TCo + TV | GTR                 | Not improved | ICH on thalamus, CNS infection |
| Milenkovic et al. [16]| 2005         | 56         | M   | Headache, memory impairment, bizarre behavior | –         | TC + TV + TF | GTR               | Improved|                          |
| Darwish et al. [17]| 2005             | 47         | F   | No symptom                                  | 1.5       | TC + TV + TF | GTR                | No symptom| HDC                      |
| Longatti et al. [18]| 2006            | 35         | M   | Headache, vomiting, neck pain                | 1.2       | TV       | GTR                 | Improved|                          |
Table 1 Summarized surgically resected cavernous hemangioma of the third ventricle (Continued)

| Study               | Year | Age | Gender | Symptoms                                | Approach | Resection | Outcome     |
|---------------------|------|-----|--------|-----------------------------------------|----------|-----------|-------------|
| Zakaria et al.      | 2006 | 8   | M      | Headache, vomiting, gait disturbance    | TC       | GTR       | Improved    |
| Kivelev et al.      | 2010 | 52  | M      | Headache, vomiting                      | TC + IF(?) | GTR       | Improved    |
| Present study       | 2012 | 64  | F      | Mental change, homonymous hemianopsia    | TC + IF   | GTR       | Improved    |

*; totally resolved at 2 months after the operation.

–, not available; DI, Diabetes insipidus; F, Female; GTR, Gross total resection; HDC, Hydrocephalus; ICH, Intracerebral hemorrhage; IF, Interforniceal; IH, Interhemispheric; IFT, Infratentorial; IVH, Intraventricular hemorrhage; M, Male; PR, partial resection; SC, Subchoroidal; SCbll, Supracerebellar; TC, Transcallosal; TCp, Transcortical; TF, Transforaminal; TLT, Translamina terminalis; TV, Transventricular; TVI, Transvelum interpositum.
structures, due to CH growth. Katayama et al. [5] stated that intraventricular CHs tend to grow rapidly resulting in giant malformation, because of low mechanical resistance caused by lack of the surrounding brain tissue and repeated hemorrhage in the CH. In the literature, the mean size of the lesions was reported to be 23 mm (range, 12–40 mm). Although intraläsional bleeding can frequently occur when CHs grow within the ventricle, bleeding from a CH into the ventricular system is rare as per the previous reports [10].

The radiological findings of the intraventricular CHs do not differ from those of the intraparenchymal type [10]. Generally, on CT scans, the CH is suggested by the presence of a high density area, absence of perilesional edema, and mild or no contrast enhancement because of blood pool effects, calcification, and recent hemorrhage [22]. On MRI images, the CHs usually have mixed signal intensities. High signal intensities correlate with the presence of methemoglobin and low signal intensities correlate with calcifications and fibrosis within the lesion on T1- and T2-weighted images. A peripheral rim of low signal intensity correlates with the paramagnetic effect of hemosiderin [23].

A conservative treatment is appropriate for an asymptomatic CH located in the supratentorial parenchyma. However, CHs located in the third ventricle, surrounded by vital structures, are especially dangerous. It has also been documented that these lesions show a rapid growth [5], resulting in significant morbidity. For these reasons, the third ventricular CH needs to be treated more aggressively. As shown in Table 1, 80% of the patients were asymptomatric or improved from their initial symptoms after the surgical procedure. The most frequent post-operative complication was a hydrocephalus, observed in four patients. Postoperative mortality was 6.9% (2/29). The important point to be noted, as illustrated by our case, is that large-sized lesions frequently involve the hypothalamus [6,10,11]. Therefore, careful dissection of the lesion should be performed to prevent damage to the hypothalamus. To reduce this complication, minimizing the resection of hemosiderin-stained tissue and preservation of associated developmental venous anomalies are the key points, as in surgery for CHs located in the brain stem or cranial nerves [24,25]. Furthermore, during the operation for CHs buried in the parenchyma with a critical neurological function, initial dissection and removal of the lesion should be attempted on the short trajectory after observation of the surface changes caused by the hemorrhage [24,26]. Considering these principles, transcallosal-interforniceal approach can provide a direct, short corridor to the third ventricle with wide exposure of the lesion.

Conclusions
Surgical excision appears to be the treatment of choice for CHs located in the third ventricle, which tend to grow rapidly and cause a mass effect. Using the short corridor to the third ventricle, obtaining wide exposure of the lesion, and minimizing resection of the surrounding hemosiderin-stained tissue can lead to a favorable surgical outcome, as demonstrated in the previous reports, including this report.

Consent
Written informed consent was obtained from the patient for publication of this Case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Abbreviations
CH: Cavernous hemangioma; CNS: Central nervous system; CT: Computed tomography; MRI: Magnetic resonance imaging.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
SMH and KSM drafted manuscript. KHL and SKL revised manuscript critically for important intellectually content. KHL and KSM helped acquisition and interpretation of data. KHL and SKL participated in reviewing literature and helped in conception and design of the study. KSM and SJ conceived the study and participated in its design and coordination. All authors read and approved the final manuscript.

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