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Case report

Intraventricular cavernous hemangioma at the foramen of Monro: Case report and literature review

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Abstract

Cavernous hemangiomas rarely occur in the cerebral ventricles. Those occurring at the foramen of Monro are even less frequent. So far, only eight cases of cavernous hemangioma at the foramen of Monro have been reported in the literature. Here, we present a similar case and correlated the radiographic with the histopathologic findings of the patient.

A 51-year-old woman was admitted with obstructive hydrocephalus-related symptoms. The computed tomography (CT) and magnetic resonance imaging (MRI) revealed a partly calcified lesion with slight contrast enhancement located in the area of the right foramen of Monro. The lesion was completely removed by surgical resection with a transfrontal transventricular approach. The resected mass was histologically diagnosed as cavernous hemangioma. The patient's symptoms resolved immediately after operation.

Cavernous hemangioma at the foramen of Monro in the present case had common MRI features as previously reported. Although MRI can provide initial diagnosis for such unusually localized tumor, it should be confirmed histopathologically.

Keywords: Cavernous hemangioma; Foramen of Monro; Obstructive hydrocephalus

1. Introduction

Cavernous hemangioma is defined as a vascular malformation composed of thin-walled sinusoidal spaces lined with endothelia. It can be found in any part of the brain and is classically an intraparenchymal lesion. Cavernous hemangiomas appear less commonly in the subarachnoid, subdural or epidural space [1–3] and rarely occur in the cerebral ventricular system. Those localized at the foramen of Monro are even more rare. Since the first description of intraventricular cavernomas by Finkelnburg [4] in 1905, only eight cases of cavernous hemangiomas arising directly from the area of the foramen of Monro have been reported in the literature [5–9]. Here, we present another case of cavernous hemangioma in this unusual location and discuss it with review of the literature.

2. Case report

The following material is presented with the informed consent of the patient. A 51-year-old female presented with severe headache for 3 months. She had suffered from gait ataxia and short-term memory deficits for 2 weeks before admission to another hospital. Severe headache and vomiting gradually increased with consciousness disturbance. Then she was referred to our hospital for hydrocephalus. The physical examination demonstrated lack of initiative, disorientation in space and time, and gait disturbance. Computed tomographic (CT) scanning showed obstructive hydrocephalus of both lateral ventricles caused by a mildly contrast-enhanced nodular mass, 8–10 mm in diameter and with calcified rim, on the right side of foramen of Monro (Fig. 1). The magnetic resonance imaging (MRI) confirmed a well-delineated intraventricular mass with heterogeneously high signal intensity on both short and long TR/TE spin-echo sequences (Fig. 2). The mixed-signal core appeared as a "popcorn-like"

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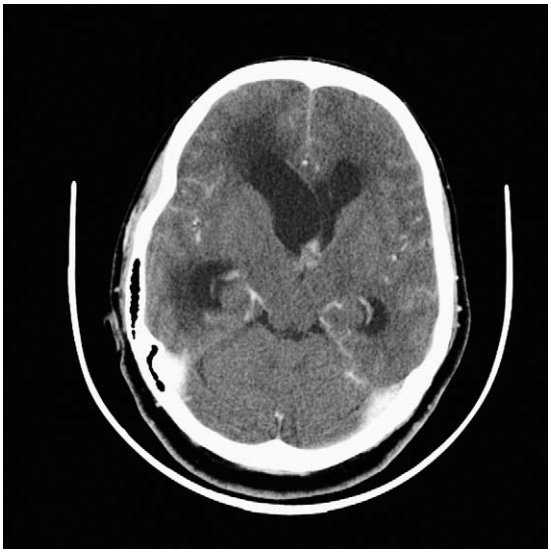

Fig. 1. Preoperative computed tomographic (CT) scan demonstrating a mildly contrast-enhanced nodular mass, 8–10 mm in diameter and with calcified rim, on the right side of foramen of Monro. The mass caused obstruction of both lateral ventricles, but the third and fourth ventricles were not affected.

lesion delineated by a low-signal hemosiderin rim (Fig. 2A and C). There was no surrounding edema on the FLAIR (fluid-attenuated inversion recovery) images (Fig. 2E) and only minimal enhancement of the mass was found in the gadolinium-contrasted T1-weighted image. (Fig. 2B). Craniotomy with total resection of the nodular mass through a transfrontal transventricular approach was performed. The resected mass, 1.5 cm × 0.9 cm × 0.3 cm in size, was reddish and lobular, and consisted mainly of clotted blood vessels and xanthochromic tissue. The histological examination revealed a vascular malformation with multiple hyalinized vessels and hemorrhagic residua at different stages (Fig. 3A). Neither muscular nor elastic tissue component was found in the vascular walls (Fig. 3B). These histologic findings were compatible with characteristics of cavernous hemangioma. After operation, the patient's symptoms including nausea, vomiting and headache gradually disappeared and further therapy was not required. Postoperative MRI confirmed complete removal of the tumor (Fig. 4).

3. Discussion

Vascular malformations of the central nervous system (CNS) are typically divided into three major categories: venous and arteriovenous malformations, capillary telangiectasia, and cavernoma (also known as cavernous malformation, cavernous angioma or cavernous hemangioma), the latter two being angiographically occult [10]. Cavernous hemangiomas are histologically benign, consisting of lobulated sinusoidal vascular channels which are lined with thin endothelia. Unlike capillary telangiectasia, cavernous hemangioma has surrounding gliosis that is not separated by intervening neural tissue. Hemorrhages at all stages of evolution are present within the lesion and cause occlusion and thrombosis of the vascular channels. Organization of the hematoma results in hyaline-degenerative changes, chronic granulation and scar formation, and induces progressive pseudotumorous evolution of the mass [11]. Further bleeding may occur in the immediate vicinity of cavernous hemangioma, leading to hemosiderin deposits and gliosis [12–16]. Unlike arteriovenous malformations, cavernous hemangiomas usually do not have obvious, well-formed vessels supplying or draining them. Therefore, they are often angiographically occult [1,15].

Cavernous hemangiomas are typically located in the subcortical areas, deep white matters and basal ganglia. Supratentorial areas account for 80% of locations where cavernous hemangiomas occur. In the infratentorial regions, cavernous hemangiomas mostly occur in the brainstem and cerebellum [1,2,17]. Instead, they are rarely seen in the intraventricular or extraaxial spaces [18]. Finkelnburg reported the first intraventricular cavernous angioma in 1905. Later on, Voigt and Yasargil [11] reported six out of their 164 cases of intracerebral cavernous angiomas to be located in the paraventricular or intraventricular areas. Lobato et al. [19] reviewed 82 cases of cavernous hemangioma and found only five lesions located in the third or lateral ventricles. Cavernous hemangioma may manifest by symptoms of acute hemorrhage, seizures or progressive neurologic deficits [20]. Katayama et al. [6] and Tatagiba et al. [21] demonstrated that the major clinical symptoms of intraventricular cavernous hemangioma at the time of diagnosis were focal neurological deficits similar to those elicited by mass effect. Signs of increased intracranial pressure and bleeding caused by intraventricular cavernous hemangioma were not rare [6,15,21–22]. In the present case, the main symptom, chronic headache, resulted from raised intracranial pressure due to obstructive hydrocephalus. Repetitive intralesional hemorrhages induced pseudotumoral growth of the intraventricular mass and led to occlusion of the foramen of Monro.

Eight cases of cavernous hemangioma located at the foramen of Monro were reviewed in comparison with our case [5–9,23–25]. The clinical data of all nine patients are summarized in Table 1. There were three males and six females, suggestive of female predominance. At symptom onset, most of the patients were middle-aged (mean 41.7 years) except one subject aged 64 years. The initial symptoms were caused by hydrocephalus in seven cases and by massive extralesional hemorrhage in the other two. All patients underwent surgical removal of the cavernous hemangioma. The surgical approaches used included transcallosal in three patients, transfrontal transventricular in four patients, and translamina terminalis approach in one patient. In the remaining subject, the surgical approach was not available. Seven patients demonstrated full recovery of neurological function after surgery, one fell into persistent vegetative state due to massive hemorrhage at symptom onset, and one retained mild neurological deficits due to postoperative hydrocephalus.