

Left Deep Frontal Cavernous Angioma Mimicking a Glioma in an Adult Patient

Abstract

Cerebral cavernous angiomas are vascular malformations characterized by large adjacent vessels. Usually, these lesions are smaller than 3 cm, the mean age at presentation occurs between 20 and 40 years, and the neuroradiological findings are well described, especially for magnetic resonance imaging, where the “popcorn balls” appearance is due to the presence of locules containing blood. Among these, the giant cavernous angiomas are very rare, particularly in adults. We collected clinical and neuroradiological data from clinical file and hospital diagnostic archive. A comprehensive review of similar cases was performed. We describe the clinical, diagnostic, and surgical management of a giant cerebral cavernous angioma located in the left deep frontal lobe mimicking a high-grade glioma in an adult Chinese patient. Giant cerebral cavernous angioma may be misdiagnosed and should be considered as differential diagnosis.

Keywords: *Cavernoma, cavernous angioma, glioma, giant angioma, mimicking*

Background

Cerebral cavernous angiomas are vascular malformations characterized by large adjacent vessels. Usually, these lesions are smaller than 3 cm, the mean age of presentation is between 20 and 40 years^[1] and the neuroradiological findings are well described, especially for magnetic resonance imaging (MRI).^[2] Among these, the giant cavernous angiomas (GCA) are very rare, particularly in adults. In our case report, we describe the case of a GCA mimicking a high-grade glioma in an adult patient.

Case Report

A 49-year-old Chinese female was admitted to the emergency department of our center for a sudden severe headache, and she was successfully referred to our institution after an enhanced CT scan and a magnetic resonance (MR) showing a round hemorrhagic mass lesion. The mass of 6 cm × 4.8 cm × 6 cm was located in the left deep frontal region with an irregular peripheral enhancement and perilesional edema determining a contralateral midline shift of 8 mm [Figure 1]. The clinical history revealed two previous episodes of a mild headache (2 months before) and

a tubal ligation performed several years before.

At the admission, the patient was awake, alert, with a GCS score of 15 and a moderate right hemiparesis. No cranial nerve palsy was observed. Desametasone 8 mg twice/day, mannitol 125 ml, levetiracetam 500 mg, and pantoprazole 40 mg were administered.

The patient underwent a surgical intervention for the excision of the lesion 2 days later. After the dural opening, some ectatic pathological cortical veins were observed within the left upper Sylvian area, in the fronto-opercular region, close to the Broca’s area. The arachnoid was dissected along the margin of the vessels and after the left frontal inferior sulcus was widened a polylobate, capsulated vascular mass was detected, which was surrounded by coagulated blood. A circumferential dissection of the capsule was performed, the mass was completely removed and the specimen sent to our pathology department for histologic examination. The postoperative computed tomography (CT) scan showed no tomographic alterations.

The histological examination was performed with hematoxylin and eosin section [Figure 2] revealed ectatic and fragmented

**Andrea Boschi,
Arturo Consoli¹,
Annamaria
Buccoliero²,
Giovanni Barbagli,
Salvatore
Mangiafico¹,
Franco Ammannati**

*Department of Neurosurgery,
¹Interventional Neuroradiology
Unit, Careggi University
Hospital, ²Pathology Unit Meyer
Hospital, Florence, Italy*

Address for correspondence:

*Dr. Andrea Boschi,
Department of Neurosurgery,
Careggi University Hospital,
Largo Palagi 150139,
Firenze, Italy.
E-mail: a.boschi.md@gmail.com*

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vascular structures. These findings were suggestive for a cavernous malformation.

The postoperative course was regular. No complications occurred, and the patient was discharged 6 days later with a good neurological outcome and improvement of the moderate right hemiparesis. A 2 months' MR follow-up was performed [Figure 3].

Discussion

The detection of a cavernous angioma is based on typical morphologic and structural features observed with the neuroimaging. Nonenhanced CT scan may be negative in about 30%–50%^[3] except in hemorrhagic ones, although no specific signs may be recognized and the most sensitive imaging technique is the MR. Cavernous angiomas are usually <3 cm, without peripheral contrast enhancement and no mass effect except in acute hemorrhages. The “popcorn balls” appearance is due to the presence of locules containing blood.^[2] These lesions have no specific localizations and may be intra- or extra-axial.^[4] In this unusual case, the MRI showed a giant intra-axial, cortico-subcortical lesion in the left deep frontal lobe with peripheral enhancement. Inhomogeneous mixed signal intensity, mass effect with midline shift and perilesional edema was observed. The diffusion-weighted imaging (DWI) revealed a restriction of the signal with corresponding ADC hypointensity and, therefore, considering all the MR characteristics the lesion was suspected for a high-grade glioma. A few cases of GCA were reported in literature.^[1,5-8] In these cases, the hemorrhagic events were more severe, and the neuroimaging might have been more suggestive for a GCA, and most of them were described in children. Instead, in our case, the patient is an adult female, with few symptoms compared to the size of GCA. The incidence of high-grade glioma in the age group of our patient is not very high, less than 10 cases per 10,000,^[9] but it is higher compared to the incidence of GCA.^[10] Other authors reported cases in which a brain tumor could mimic a different one, and the final diagnosis was provided by histological examination.^[11,12] Finally, also some infectious, inflammatory or demyelinating diseases, such as multiple sclerosis^[13] or tumefactive demyelinating lesions^[14] may present some characteristics that may become confounding factors and may lead to a misdiagnosis.

Conclusion

The treatment of a giant cerebral lesion should be chosen depending on the nature of the lesion. In those cases, which the proper surgical approach is established, especially if a cerebral biopsy is compulsory for subsequent treatment (i.e., radiotherapy-chemotherapy), all the differential diagnoses should be enlisted, and the only morphologic standard neuroimaging should not be considered as sufficient. A neurovascular study (MR-A, CT-A, digital subtraction angiography) should be considered in all the cases, even if it might not provide

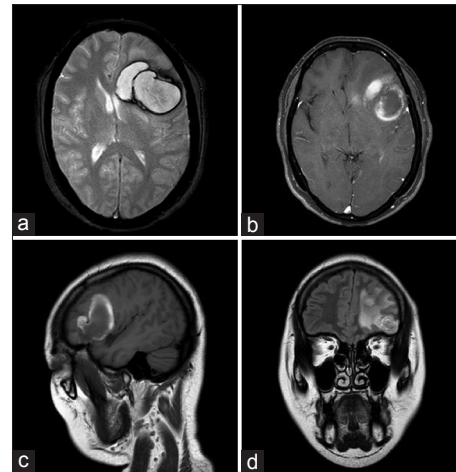


Figure 1: Brain magnetic resonance at the admission. (a) T2* sequence in axial plane showing hemosiderin within the lesion and in the surrounding space. Postcontrast T1 on axial (b) and sagittal (c) planes showing peripheral enhancement of the left frontal lesion. Fluid-attenuated inversion recovery sequence on coronal (d) planes demonstrating perilesional edema and contralateral shift

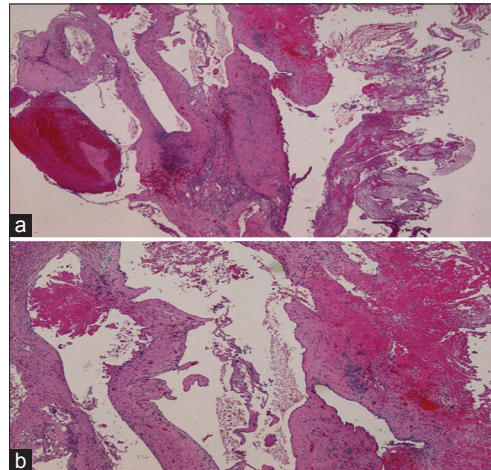


Figure 2: Histological examination with 4x magnification (a) and x2 magnification (b) hematoxylin-eosin section revealed ectatic and fragmented vascular structures

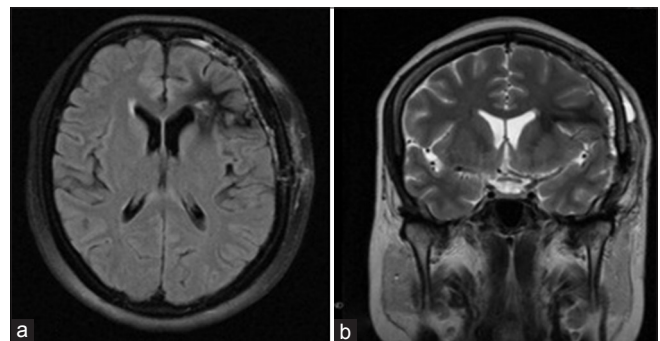


Figure 3: Two months follow-up magnetic resonance imaging. axial Fluid-attenuated inversion recovery sequence (a) and coronal TSE T2 sequence (b) showing the complete excision of the lesion

further information, as well as advanced MRI with DWI-perfusion-weighted imaging spectroscopy.

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Conflicts of interest

There are no conflicts of interest.

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