Giant intracranial aneurysm with thrombus and calcification

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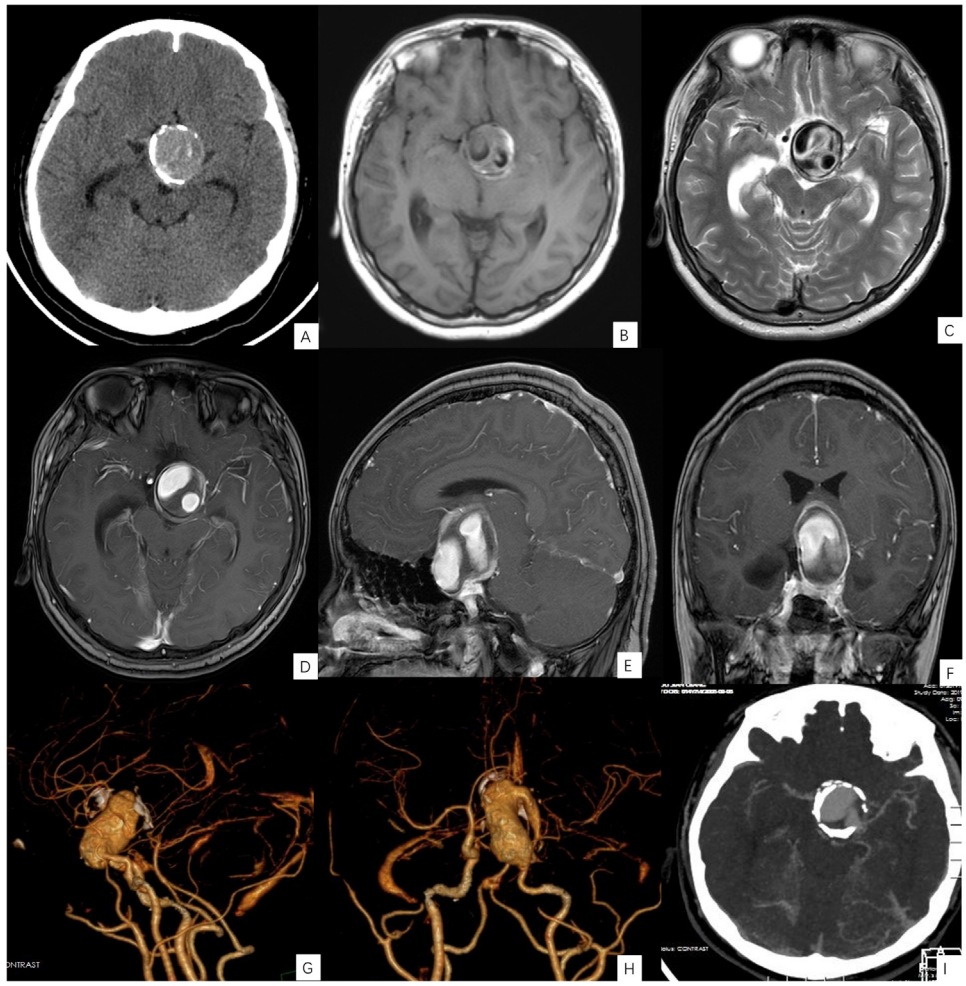

FIGURE.1.

CASE PRESENTATION

A 25-year-old man reported to our department with a history of fever (about 39°F), headache, nausea, vomiting, sporadic convulsions of the limbs lasting up to 10 days and blindness of the left eye for the past 1 month. The computed tomography (CT) scan revealed a high-density mass measuring 3.5 × 2.8 × 4.3 cm, with multiple calcifications in the suprasellar cisterna region (Fig. 1A). The initial assessment indicated craniophrayngioma. Following this the patient underwent further magnetic resonance imaging (MRI) examination, which detected mixed high and low signals with annular hypointensity at the margin. Moreover, the bilateral frontal lobes and brainstem were compressed (Fig. 1B, C). Enhanced MRI revealed that the mass had thick strip enhancement, marginal annular enhancement, and few patchy areas without enhancement (Fig. 1D, E, F). Since the image showed the mass in close proximity to the neighboring vessels, it was suspected to be a thrombotic aneurysm. Subsequently, a CT angiography confirmed that the lesion was likely to have originated from the ocular segment of the left internal carotid artery

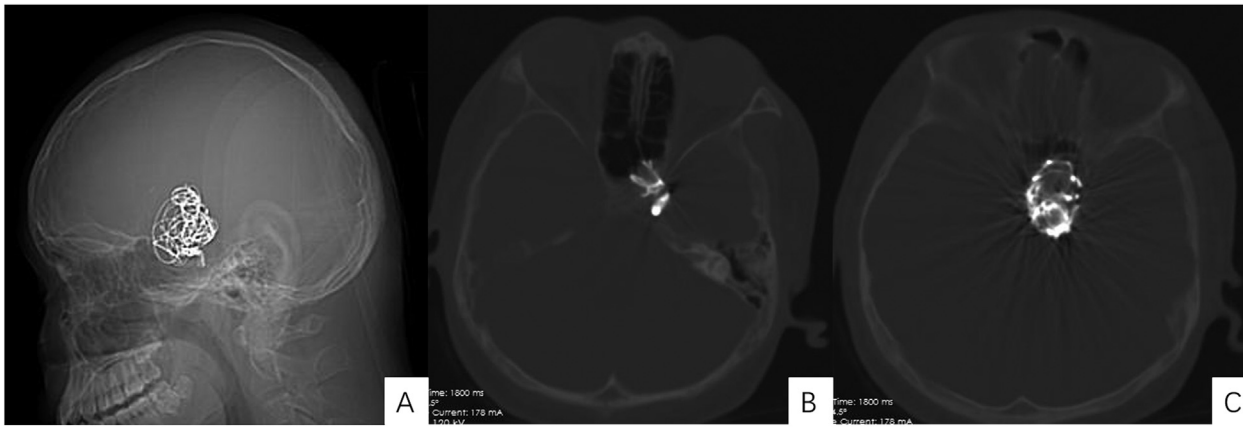

FIGURE 2.

(Fig. 1G, H, I). The patient underwent interventional embolization of the aneurysm (Fig. 2) and recovered without any complications.

The general prevalence rate of intracranial aneurysms is about 3%–5%, with a 1.2% risk of rupture within 5 years of diagnosis, with the circle of Willis being the most common site. Only 2%–5% of intracranial aneurysms enlarge up to a maximum diameter of ≥2.5 cm, called as giant intracranial aneurysms, and are located in the anterior cerebral vessels in 3% of cases.[1,2] These aneurysms usually have a considerable mass effect on the brain, shifting the brain matter, leading to neurological deficits. This mass effect associated with perianeurysmal edema aggravates the patient's condition, complicating treatment outcomes. The direct contact between the partially thrombosed surface of the giant intracranial aneurysms and the brain parenchyma may be crucial for perianeurysmal edema.[3] The risk of subarachnoid hemorrhage increases with aneurysm size. A subarachnoid hemorrhage from an intracranial aneurysm rupture can lead to serious accidents, and less than one-third patients can lead normal lives after this. Currently, the clinical treatment strategies for intracranial aneurysm are mainly restricted to endovascular therapy and surgical clipping.[2]

CONFLICTS OF INTEREST/SOURCE OF FUNDING

All authors have no conflict of interest and grant to declare.

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