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## A possible case of unruptured middle cerebral artery aneurysm presenting as epileptic seizures

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**Abstract** We report the case of a 73-year-old man with an unruptured aneurysm of the left middle cerebral artery. The initial sign was complex partial seizures. A standard scalp electroencephalogram was normal while neuropsychological tests revealed a slight deficit of episodic memory. Brain MRI showed an aneurysm at the left middle cerebral artery bifurcation. Cerebral angiography confirmed the presence of a saccular aneurysm at the left middle cerebral artery bifurcation, with a maximum diameter of 12 mm. This case had two main characteristic features: seizures had a quite late onset and were the only symptom the patient experienced.

**Key words** Middle cerebral artery aneurysm • Unruptured aneurysm • Seizure

### Introduction

Cerebral aneurysms present most commonly with subarachnoid hemorrhage. Unusual presenting symptoms of unruptured cerebral aneurysms include headache, cranial nerve compression or mass effects. Seizures as presenting symptoms of unruptured giant cerebral aneurysms are rare, and they are even more rare as the first and only symptoms of small unruptured cerebral aneurysms [1–8]. Hence cerebral aneurysms are not generally considered in the differential diagnosis of lesions causing seizures. In this paper we describe a patient with complex partial seizures associated with an unruptured intracranial aneurysm of the left middle cerebral artery (MCA).

### Case report

A 73-year-old right-handed man complained in February 1999 of brief episodes of confusion preceded by epigastric aura with perception of a metallic taste and oral (masticatory) and manual automatisms involving both hands; sometimes déjà-vu were also reported. The first episode occurred six months before admission and subsequently there was a frequency of almost one attack per month. Seizures lasted 2–3 minutes and were followed by a postictal state, with confusion and drowsiness. There was no medical history of febrile convulsions in early life, head trauma, cardiac disease or drug or alcohol abuse, and there was no family history of epilepsy.

General and neurological examinations were unremarkable except for an osteotendineal reflex prevalence at the right side. Slight arterial hypertension was found and the patient was treated with enalapril (20 mg daily). Full blood count and hematologic analysis were normal.

Scalp electroencephalography (EEG) was normal and neuropsychological tests revealed a slight deficit of episod-

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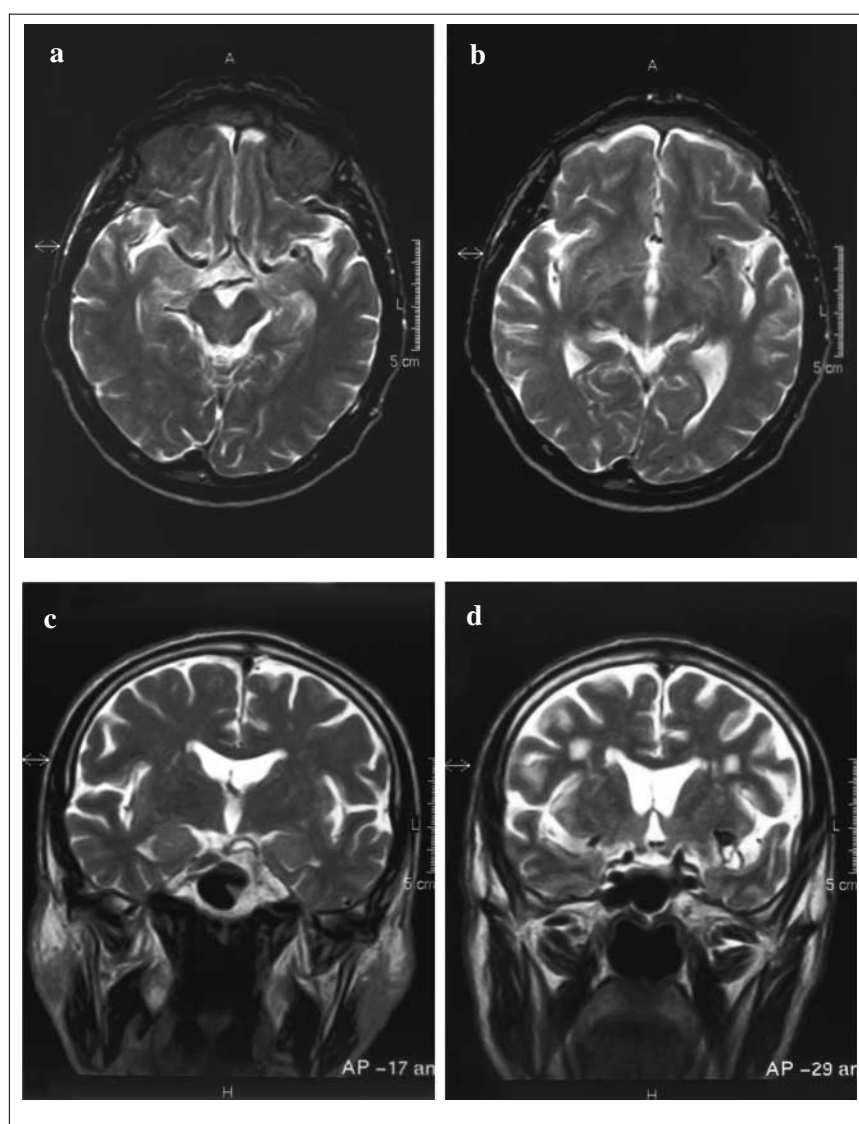
ic short-term memory. Magnetic resonance imaging (MRI) of the brain showed an aneurysm at the left MCA bifurcation, located in the depth of the sylvian fissure, in contact with the claustrum cranially and adjacent to the mesial temporal structures inferiorly (Fig. 1). Furthermore, no cortical malformations or other signal abnormalities were detectable in the mesial temporal structures. Cerebral angiography confirmed the presence of a saccular aneurysm at the left MCA bifurcation, with a maximum diameter of 12 mm (Fig. 2).

A diagnosis of complex partial seizures was made and the patient was treated with 300 mg carbamazepine (CBZ) daily. A decision was made to proceed with surgery. Two weeks later, clipping of the aneurysm was performed; after surgical intervention there was a transient right hemiparesis and, two days later, brain computed tomography (CT)

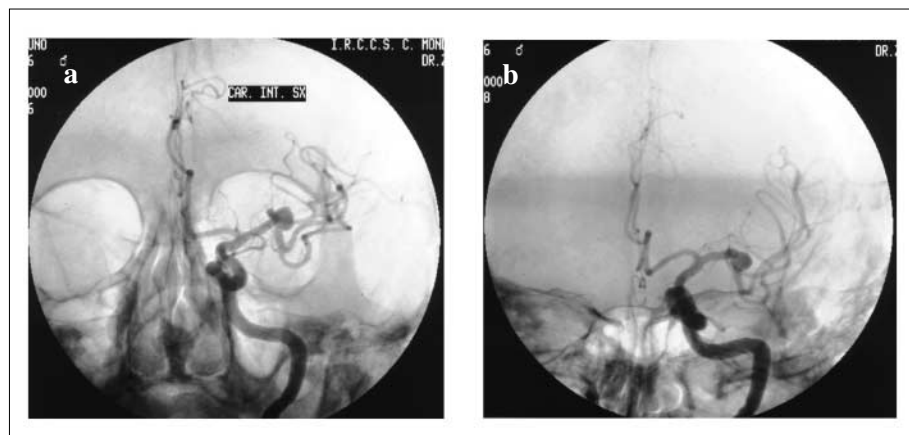
showed the existence of a right subdural hygroma. Within a few days motor disability was resolved and the patient was treated with 100 mg phenytoin daily.

One month later antiepileptic drugs were withdrawn by the patient and three brief (less than 30 seconds), partial seizures with epigastric aura and oral automatisms occurred; neither a postictal state nor impairment of consciousness during the seizure was referred. After the reintroduction of CBZ (400 mg daily) seizures were well controlled.

CT performed five months after the operation showed an almost complete reabsorption of the hygroma and the presence of a metallic clip in the left sylvian area (Fig. 3). EEG of the scalp showed features similar to those seen previously. The patient has been followed for 26 months and seizures have been controlled by the administration of 400 mg CBZ daily (plasma concentration, 7.6  $\mu\text{g/ml}$ ).



**Fig. 1a-d** T2-weighted MR scans show a rounded lesion with high velocity signal loss (flow voids) which arise from the left middle cerebral artery (MCA). **a-c** The berry aneurysm is located inside the sylvian fissure, in contact with the claustrum cranially and adjacent to the mesial temporal structures inferiorly. **d** No malformative or signal abnormalities were evident in the temporal lobe



**Fig. 2a,b** Anterior views of the left internal carotid artery angiogram show a large saccular aneurysm at the level of the trifurcation of the middle cerebral artery. **a** Straight anteroposterior projection. **b** Half-axial anteroposterior projection



**Fig. 3** Plain post-surgery CT scan demonstrating clips in the left sylvian fissure and dilatation of the temporal horn of the homolateral lateral ventricle

## Discussion

Seizures are unusual presenting features of unruptured cerebral aneurysms and are therefore not frequently included in the differential diagnosis of patients presenting with epilepsy. Giant aneurysms are reported to cause epileptic seizures more often than small ones; most published papers report complex partial seizures with giant aneurysms of the middle cerebral artery, maybe because of the low seizure threshold of the mesial temporal lobe structures particularly when they are damaged [9, 10].

In our case, aneurysm was detected by means of MRI and diagnosis was made some time after the onset of seizures. Furthermore, the onset of the seizures in this patient occurred quite late (73 years of age). The most frequently reported causes of epilepsy in the elderly have been excluded in this case: there was no medical history of head injury, drug or alcohol abuse and no family history of epilepsy, nor of infective or metabolic disturbances, tumor, cardiac or respiratory

illness, ischemic or hemorrhagic stroke or dementia. This evidences the need to carry out in-depth diagnostic research on the symptomatic forms of epilepsy in order to find a suitable therapeutic, pharmacological or surgical solution.

In this patient, seizures were supposed to be due to the aneurysm. This hypothesis was supported by the correlation between localization of the aneurysm and ictal semiology and by the decrease in the frequency, severity and duration of seizures after surgery. Seizure onset could be attributed to a recent enlargement of aneurysm, with an irritative action on temporal, particularly insular, structures. It is also interesting to notice that post-clipping seizures occurred in absence of impairment of consciousness, according to the description of seizures with oroalimentary automatism with preserved consciousness reported by some authors [11]. Seizures were controlled after clipping of aneurysm according to previous reports that showed a good prognosis after surgery, often maintaining antiepileptic drug therapy for the follow-up period; in this patient CBZ was sufficient at a quite low daily dosage.

It is still unclear how cerebral aneurysms cause seizures. Most commonly postulated mechanisms include ischemic changes, compression of the adjacent brain tissue, calcification in the wall of the aneurysms and repeated subclinical hemorrhages which lead to gliosis in the near brain tissue [6].

The best management of patients with unruptured intracranial aneurysms remains controversial; in fact it requires an accurate assessment of the risks of different treatment options compared to the natural history of the condition. It is well accepted that symptoms such as mass effect on cerebral or brain stem structures, compression of cranial nerves, or ischemic/embolic phenomena can be treated with surgical clipping and decompression and can serve as an important indication for treatment, as they indicate a sign of aneurysmatic growth [12]. Our case had two main characteristic features: first of all, seizures had a quite late onset, and, second, the seizures were the only symptom experienced by the patient.

Recognition that epileptic seizures can be due to aneurysms has important prognostic implications. In fact,

like other symptoms classically associated to unruptured aneurysm, they can represent an indication for surgical treatment in order to prevent hemorrhage and to maximize the potential recovery of the possible sensory or motor deficit [13].

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