

Unruptured cerebral aneurysms causing seizure disorder (report of two cases)

H. ELLAMUSHI, L. THORNE & N. KITCHEN

University Department of Neurosurgery, Institute of Neurology UCL, Queen Square, London, WC1 3BG UK.

Correspondence to: H. Ellamushi, University Department of Neurosurgery, Institute of Neurology, Queen Square, London WC1 3BG, UK

Seizures as a presenting feature of unruptured cerebral aneurysm are unusual. We report two cases of unruptured cerebral aneurysm presented with seizures. In both cases the seizures controlled following surgical ablation of the aneurysm.

Key words: unruptured cerebral aneurysm; seizure; cerebral angiography.

INTRODUCTION

Cerebral aneurysms present most commonly with sub-arachnoid haemorrhage. Rare presenting symptoms of unruptured cerebral aneurysms include headache or/and cranial nerve compression or mass effects. Seizures as a presenting symptom of unruptured giant cerebral aneurysms are unusual and even more unusual as the presenting symptoms of small unruptured cerebral aneurysms. Thus cerebral aneurysms are not generally considered in the differential diagnosis of lesions causing seizures. We report two cases of unruptured cerebral aneurysms which presented with seizures and in one of them the aneurysm was small and in a peculiar position, arising from a peripheral branch of the middle cerebral artery. In both these cases the diagnosis of the aneurysm was made some time after the onset of the seizures.

CASE ONE

A 38-year-old left-handed woman presented in January 1997 with an episode of loss of consciousness. She bent over at work, felt peculiar, and subsequently walked out of the building and was witnessed to have collapsed.

Five months later she developed a dull aching headache when cycling. She had an epigastric aura

* E-mail: H.Ellamushi@ion.ucl.ac.uk

associated with a feeling of *jamais vu* and was in a dream-like state. She was witnessed getting off her bi-

cycle, then walking off before collapsing with generalized tonic-clonic convulsions with tongue biting and incontinence. The epileptic attack lasted 5–10 minutes, following which the patient was in a post-ictal state, confused and drowsy. The patient had a past history of migraine and at the age of 20 she was diagnosed as having viral meningitis. There was no history of head trauma, cardiac disease, drug or alcohol abuse and no family history of epilepsy or cerebrovascular disease. General, systemic and neurological examination was unremarkable and standard scalp EEG showed epileptic waves arising from the left temporal lobe. An MRI scan of the head revealed a 7 mm rounded lesion within the left superior temporal gyrus with a low signal in T1 and T2 images with a surrounding high signal in T2 images (Fig. 1). Full blood count, ESR, C-reactive protein and blood culture were normal. Cerebral angiography showed an aneurysm arising from the periphery of the temporal branch of the middle cerebral artery (Fig. 2). With the help of a frameless stereotactic technique (Philips Easyguide Neuronavigator system) using magnetic resonance images, the site of the aneurysm was accurately localized. A small linear scalp incision was performed and the aneurysm exposed using a small trephine craniotomy. The aneurysm was found embedded in the superior temporal gyrus very close to the cortical surface. There was no evidence suggesting re-

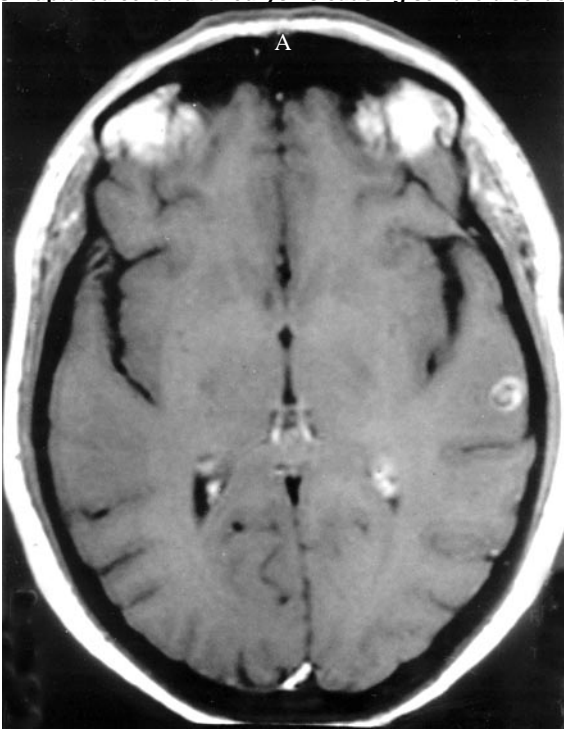


Fig. 1: Enhanced T1-weighted axial MR image showing a rounded lesion within the left superior temporal gyrus.



Fig. 2: Left carotid angiogram showing a small aneurysm arising from the temporal branch of the middle cerebral artery.

cent haemorrhage or rupture, however, the surrounding brain tissue looked abnormal in colour and was gliotic. A swab for microbiological testing sent from the site of the aneurysm was negative. The aneurysm was successfully clipped using two fenestrated straight aneurysm clips.

The patient had an uneventful post-operative recovery and remains seizure free on no antiepileptic med-

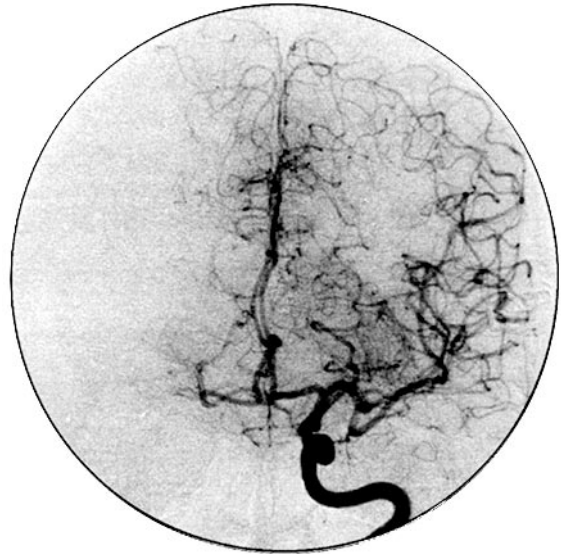


Fig. 3: Post-operative cerebral angiography showing complete ablation of the aneurysm with the temporal branch distal to the clipped aneurysm is patent.

ication 2 years following surgery. The post-operative angiogram showed complete ablation of the aneurysm (Fig. 3).

CASE TWO

A 61-year-old male presented in June 1997 with a sudden collapse at work. He remained unconscious for 10 minutes and was briefly confused following the episode. There has no history of headache before or following the collapse. Following this episode he has had several attacks of presyncope associated with pins and needles sensation down the right side. Four weeks later he had a generalized tonic-clonic seizure whilst asleep with tongue biting and incontinence. During the neurological examination the patient was drowsy, confused and irritable but displayed no other abnormalities. Scalp EEG showed frequent burst of sharp waves over the left anterior temporal lobe indicating seizure activity arising from this area. A CT scan of the brain was normal. The patient was commenced on antiepileptic medication and discharged from hospital. Despite medical treatment, he continued having nocturnal seizures (2-3 seizures every night). Three months later an MRI scan of the brain was performed which showed a 2.5 cm lesion invaginating the uncus part of the temporal lobe and involving the amygdala and the hippocampus (Fig. 4).

A cerebral angiogram confirmed a giant aneurysm arising from the left internal carotid artery near the origin of the posterior communicating artery (Fig. 5).

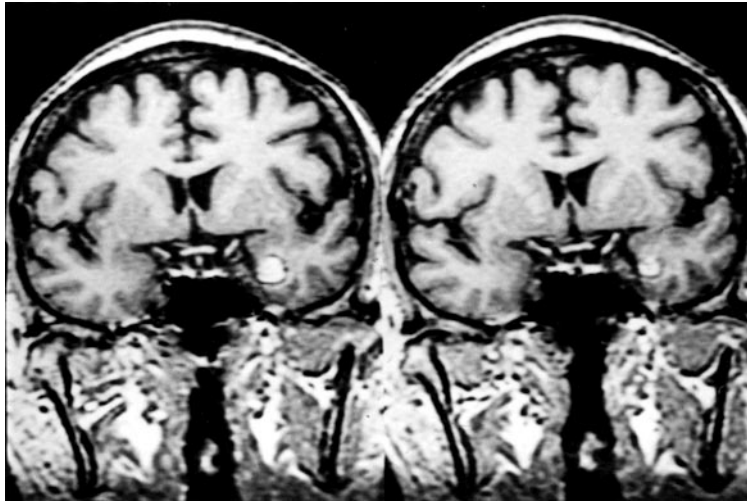


Fig. 4: Contrast enhancing T1-weighted coronal MR image showing a rounded hyperintense lesion invaginating the left temporal lobe.

A decision was made to proceed to surgery. Through a posteroital craniotomy the giant aneurysm was ex-

DISCUSSION

Seizures are unusual presenting features of unruptured cerebral aneurysms and frequently are not included in



Fig. 5: Left carotid angiography showing a giant aneurysm arising from the internal carotid artery.

posed and defined. The aneurysm was arising from the internal carotid artery distal to the origin of the posterior communicating artery. After defining the aneurysm, two aneurysm clips were applied uneventfully across the neck of the aneurysm. The patient recovered well following the surgery with no neurological deficit. He remained seizure free when he was seen in the follow-up clinic 18 months following the surgery.

The cerebral angiogram performed post-operatively revealed complete obliteration of the aneurysm (Fig. 6).

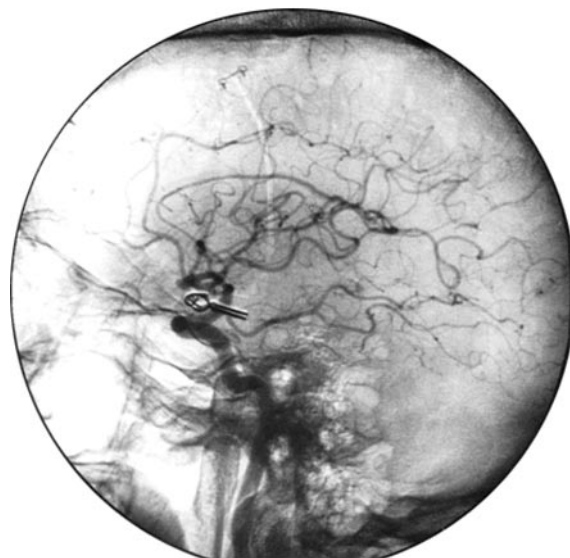


Fig. 6: Post-operative cerebral angiogram shows the clip across the neck of the aneurysm, which is completely obliterated.

the differential diagnosis of patients presenting with seizures. In our two cases the diagnosis of the aneurysm was made some time after the presentation, and in both cases the aneurysm was detected unexpectedly. Most of the reports in the literature link complex partial seizures with giant aneurysms of the middle cerebral artery¹. In a series of 202 patients with unruptured cerebral aneurysms, 5% of the patients presented with seizures, and all had aneurysms larger than 1 cm². In contrast, Sahs *et al.*³ (1969)

recorded that 8% of unruptured posterior communicating artery aneurysms and 36% of middle cerebral artery aneurysms presented with seizures. In our report, one case had an aneurysm in the middle cerebral artery territory which was less than 1 cm in size, and in the second case the (giant) aneurysm was situated on the internal carotid artery.

Although it is not clear how cerebral aneurysms cause seizures, a number of mechanisms are postulated, including compression of, or ischaemic change in, the adjacent brain tissue, calcification in the wall of the aneurysms acting in the same way as a haematoma and repeated subclinical haemorrhage causing gliosis in the adjacent brain tissue^{1,2,7}.

In this report, the aneurysm in the first patient was too small to cause any local pressure effect, however the presence of gliotic-looking brain tissue surrounding the aneurysm indicated that a subclinical haemorrhage might have been the mechanism causing the seizures. In the second patient, the aneurysm was giant, invaginating the mesial temporal lobe with no calcification in the wall of the aneurysm and it is possible that local pressure by the aneurysm on the temporal lobe might explain the cause of the seizures.

In both cases the seizures were controlled after obliterating the aneurysm and both patients remained seizure free after a follow-up period of 24 months in the first case and 18 months in second case. This is similar to the previous reports which showed obliterating the aneurysms was necessary for the post-operative control of seizures and to prevent the risk of bleeding. These two case reports consolidate the previous reports in the literature of unruptured aneurysms causing seizures^{1,5,7,8} and show that the best treatment option was surgical obliteration which both con-

trols the seizures and prevents the risk of future haemorrhage^{1,5,7,8}. We also emphasize that patients presenting with seizures without neurological deficit and without any history suggesting subarachnoid haemorrhage, may still have an aneurysm causing the seizures and therefore should be considered in the differential diagnosis. A brain magnetic resonance imaging (MRI) and in some cases magnetic resonance angiogram (MRA) might be necessary to exclude the possibility of a cerebral aneurysm causing the seizure.

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