

CASE REPORT

Aneurysms and epilepsy: an increasingly recognised cause

ABDUL WADUD KAMALI, O. C. COCKERELL & P. BUTLAR

Department of Neurology, The Royal London Hospital, London, UK

Correspondence to: Abdul Wadud Kamali, Intercalating Medical Student, 44 Anson House, Shandy Street, London E1 4SR, UK. *E-mail:* awkamali@hotmail.com

Intracranial aneurysms have rarely been reported in association with complex partial seizures. We report five cases of complex partial seizures where aneurysms were found either accidentally or through investigations in these patients. MRI scans and angiograms revealed these aneurysms and in one patient several aneurysms were found. Four patients were treated with GDC coil embolisation with follow-on anti-epileptic medication. Two of the patients have reported a few minor seizures despite the treatment. The seizures in the other two patients have resolved completely after the treatment. The fifth patient is on anti-epileptic medication. We review the possible pathogenesis for the link between aneurysms and seizures. The treatment of intracranial aneurysms is also reviewed.

© 2003 BEA Trading Ltd. Published by Elsevier Science Ltd. All rights reserved.

Key words: intracranial aneurysms; seizure; coil embolisation.

INTRODUCTION

Epilepsy is rarely caused by unreptured intracranial aneurysms. Rare presenting symptoms of unreptured cerebral aneurysms include headache or/and cranial nerve compression or mass effects¹ and the main signs and symptoms have been related to bleeding or mass lesion². So far we have come across five independent case reports^{1–5} describing a total of 14 epilepsy patients in whom intracranial aneurysm was accidentally found. In a survey of 666 patients with temporal lobe epilepsy Currie *et al.*⁶ reported only one patient having an aneurysm in the middle cerebral artery. Cerebral aneurysms are not generally considered in the differential diagnosis of lesions causing seizures.

We report five cases of unreptured cerebral aneurysms that presented with seizures. We review the relevant literature on the possible pathogenetic mechanism for these seizures and the treatment outcome.

CASE 1

A 57-year-old lady presented in March 2001 with an episode of loss of consciousness. She remembers photocopying at work then waking up surrounded by ambulance crew. She did not have any headaches, loose urinary or fecal continence or bite her tongue. She recovered consciousness in 20 minutes. Witnesses say she was shaking her arms and legs and she was dribbling from her mouth. She hit the right side of her head.

She has a 3–4-year history of complex partial seizures manifesting in clinical symptoms of remembering or half remembering things. This is associated with light-headedness. General, systemic and neurological examinations were unremarkable and a CT scan (Fig. 1) showed a 1.2 cm slightly enhancing well defined lesion related to the medial aspect of the right middle cranial fossa and abutting the right internal carotid artery. It also showed surrounding low

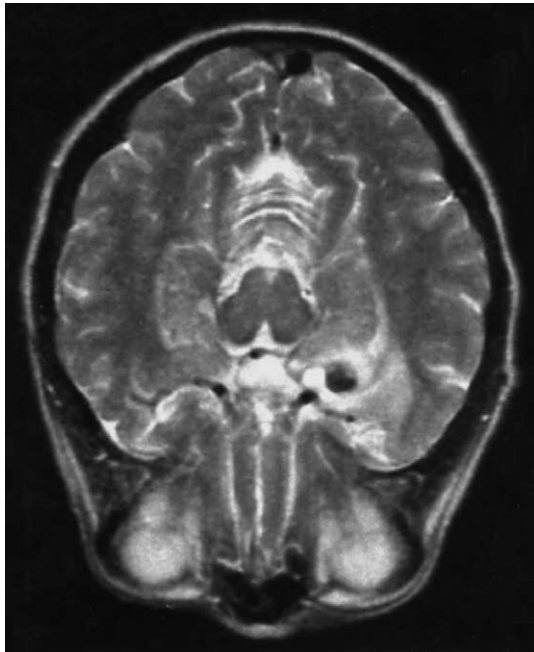


Fig. 1: Computed tomography scan showing enhanced lesion abutting the right internal carotid artery.

density in the right temporal region compatible with oedema. Cerebral angiography (Fig. 2) revealed a large aneurysm in the right posterior-communicating artery. It also revealed aneurysms in the left internal carotid artery, left posterior-communicating artery, right internal carotid artery and the anterior segment of the right anterior communicating artery. Her last seizure was in March 2001 and she had a coil embolisation of the large right posterior-communicating



Fig. 2: Cerebral angiography showing a large aneurysm in the posterior communicating artery.

artery aneurysm performed in April 2001 which was thought to have been giving rise to the epileptic neuronal discharges. There were no post-operative complications. She has had no further epileptic seizures and remains on carbamazepine.

CASE 2

A 50-year-old man presented in November 2000 with an episode of sudden collapse. These blackouts have been happening from 1998 but no real cause has been found. When these attacks started they were quite frequent and very stereotyped in their appearance. He always gets a warning that is a rising nausea and anxiety type feeling that moves up from his stomach to his throat. This is accompanied by an unpleasant taste in his mouth. He then collapses and then falls on the ground and afterwards he is very confused for about 5 minutes. A diagnosis of complex partial seizures was made. General, systemic and neurological examination was unremarkable. CT and EEG scans were normal but a MRI scan showed a small (5–6 mm) low signal lesion within the right uncus consistent with an aneurysm. An MRA confirmed the presence of a small aneurysm arising from the right posterior-communicating artery. His last complex seizure was in July 2001. Clipping was performed in October 2001 and he has been put on sodium valproate since then. Despite the treatment he has had several minor seizures and one complex partial seizure.

CASE 3

A 37-year-old right-handed man first presented in September 2001 complaining of complex partial seizures. In October 2000 while at work his eye-balls started rolling, he span and fell from standing. He then lost consciousness and had a tonic-clonic seizure, which lasted for a few minutes. There was no tongue biting or incontinence. Two months later while playing squash he stood still repeating the same activities having another similar seizure. He was treated with phenytoin and improved. Since then he has had no full-blown complex partial seizures. He has a 4-year history of unrelated tension headaches, pressure all over the head, which usually disappears. On examination he was generally well and showed no neurological deficits. His speech was normal. He had a CT scan (Fig. 3) in July 2001 showing a mass enhancing lesion on the right temporal fossa. A MRI of the brain and an MRA showed an aneurysm of the right middle cerebral artery at the bifurcation. It also showed a cyst like lesion in the parenchyma adjacent



Fig. 3: Computed tomography scan showing a mass enhancing lesion on the right temporal fossa region.

to the aneurysm. In December 2001 he had a cerebral angiogram done (Fig. 4) and embolisation of the right middle cerebral bifurcation aneurysm was also performed. Post-operatively he has been on phenytoin and carbamazepine but has had a few localised seizures since then.



Fig. 4: Cerebral angiography showing an aneurysm of the right middle cerebral artery.

CASE 4

A 44-year-old bus driver presented in August 2000 with simple and complex partial seizures. These started with an episode of feeling 'dread' and difficulty in breathing. Earlier that day he was driving his bus when he suddenly got an electric shock starting from the top of his head and spreading straight through his body over a split second. He got an intense feeling of dread and he began to panic and had difficulty in breathing. He got out of the bus but then got back in and drove it back to the depot. In the depot he went into the office and broke down in tears. He has no recollection of driving the bus back. He was taken to A&E and discharged that day.

He had two further episodes of 'dread' in December 2000 lasting 30 seconds. He may not have been able to speak during the attack but this is not definite. He has had no olfactory auras or epigastric rising pain. In between these events he had episodes of his right arm feeling very empty with dystonic posturing of the hand lasting 2–3 minutes. This can occur up to six times a night. He has a family history of epilepsy. On examination his general systematic and neurological examination was unremarkable. Cranial MRI shows a right middle cerebral artery aneurysm measuring 10 mm. His last seizure was in early December 2001. He was started on carbamazepine, which has completely controlled his seizures. He insisted on having elective coiling of his aneurysm which was performed in late December 2001. He was discharged from hospital and has had no episodes of seizures since then.

CASE 5

A 57-year-old right-handed woman presented in December 2001 with complex partial seizures. Earlier that day while shopping, she went to the toilet, felt strange and the next thing she knew, she had collapsed and found herself on the floor. She was out for approximately 2 minutes and remembers when coming round that her legs were shaky and felt tired. Within 10 minutes she was back to normal. She has had a few more incidents in February and March 2002 when she felt strange in the head with a sensation as if to pass out, but she managed to sit down, otherwise she feels that she might have fallen. These incidents are preceded immediately by a funny smell and she knows that this is a warning of another attack.

One of the incidents in February was while she driving. She managed to pull over for a few minutes and then continued her journey back home. Since then she has been having frequent episodes of feeling odd and detached for about a minute. But she has lost the *déjà vu* for smell. On examination her cardiovascular

system was normal with no carotid bruits, fundi normal with no focal neurological deficit. Her ECG is normal but her MRI and MR angiography showed a small aneurysm just below the bifurcation of the carotid siphon on the right side. She has been started on carbamazepine in July 2002.

DISCUSSION

When a patient has epileptic seizures, the possibility of an intracranial aneurysm is rarely raised. In a series of 202 cases of unreptured cerebral aneurysms, epilepsy was detected in 5% of the patients¹. Morley and Barr⁷ reported 3 patients having epilepsy in a series of 21 patients with giant aneurysms. In all, the aneurysms were located at the middle cerebral artery. Liang-fu and Da-jie⁸ similarly reported epilepsy in 2 of 21 cases of giant aneurysms, both again located at the middle cerebral artery. In contrast, Sahs *et al.*⁹ recorded that 8% of unreptured posterior-communicating artery aneurysms and 36% of middle cerebral artery aneurysms presented with seizures. Most of the reports in the literature link complex partial seizures with giant aneurysms of the middle cerebral artery⁵ although there are a small number of reports linking seizures with aneurysms originating in other parts of the cerebral circulation¹⁻³. In our report, two cases had aneurysms originating from the middle cerebral artery and the other two cases had the aneurysm originating from the posterior-communicating arteries. Two/three of them were small aneurysms, which is even rarer as a cause of epilepsy.

Although it is not clear how cerebral aneurysms give rise to seizures, several mechanisms have been postulated. Falconer *et al.*¹⁰ demonstrated that a significant proportion of temporal lobe epilepsy is caused by pathologically identifiable hamatomas. Calcification in the walls of aneurysms act in the same way as hamatomas and other reports have supported this theory^{4,9}. Other theories include compression of, or ischaemic changes in, the adjacent brain tissue^{4,9}, subclinical hemorrhage causing gliosis in the adjacent brain tissue^{2,6} and emboli from distant cerebral aneurysms travelling to other parts of the brain which result in micro infarction and subsequent development of epileptogenic focus¹.

Surgical clipping has been the mainstay of treatment of both reptured and unreptured cerebral aneurysms until 1991 when the Guglielmi detachable coil (GDC) embolisation as an alternative method for treating selected patients was introduced. Ellamushi *et al.*¹ reported that the best treatment option for unreptured aneurysms was surgical obliteration which both controlled the seizures and prevents the risk of future haemorrhage. Several reports back this report but all

were carried out prior to the introduction of the GDC embolisation. Lot *et al.*¹¹ showed that in selected patients with aneurysms appropriate for this procedure, coil embolisation has better outcome than surgical reduction. Unsatisfactory results were seen in 7% of surgical cases of aneurysm reduction and 9% after embolisation but after retreatment, they were 3 and 0.70%, respectively. The advantage of this procedure in selected patients is also suggested in other reports^{12,13}. GDC embolisation was performed in four of the patients in this study who were suitable for the procedure alongside oral medication. Two of the patients were still reporting minor episodes of seizures where the other two were doing very well. The last patient hasn't had GDC embolisation performed and it is too early to comment on her response to medication. Therefore, treatment of aneurysms with clipping has shown some improvement but a larger study is needed to get a clearer picture of the effects of such a treatment.

ACKNOWLEDGEMENT

The authors would like to thank Dr Hewazy, Consultant Neurologist, Southend General Hospital for his assistance with the case reports.

REFERENCES

1. Ellamushi, H., Thorne, L. and Kitchen, N. Unreptured cerebral aneurysms causing seizure disorder (report of two cases). *Seizure* 1999; **8**: 310-313.
2. Yacubain, E. M. T., Rosenberg, S., Da Silva, H. C. A., Jorge, C. I., De Olivera, E. and De Assis, L. M. Intractable complex seizures associated with posterior cerebral artery giant aneurysm: a case report. *Epilepsia* 1994; **35**: 1317-1320.
3. Leibrock, L. G., Bennet, D. R. and Bloch, S. Complex partial seizures associated with unreptured thrombosed basilar artery apex aneurysm. *Surgical Neurology* 1983; **19**: 17-20.
4. Singupta, R. P., Saunders, M. and Clarke, P. R. R. Unreptured intracranial aneurysms—an unusual source of epilepsy. *Acta Neurochirurgica* 1978; **40**: 45-53.
5. Whittle, I. R., Allsop, J. L. and Halmagyi, G. M. Focal seizures: an unusual presentation of giant intracranial aneurysms: a report of four cases with comment on the natural history and treatment. *Surgical Neurology* 1985; **24**: 533-540.
6. Currie, S., Heathfield, K. W. G., Henson, R. A. and Scott, D. F. Clinical course and prognosis of temporal lobe epilepsy: a survey of 666 patients. *Brain* 1971; **94**: 173-190.
7. Morley, T. P. and Barr, H. W. K. Giant intracranial aneurysms: diagnosis, course and management. *Clinical Neurosurgery* 1969; **16**: 73-94.
8. Liang-fu, Z. and Da-jie, J. Large and giant intracranial aneurysms-diagnosis and surgical treatment. *Chinese Medical Journal* 1987; **100**: 392-397.
9. Sahs, A. L., Perret, G. E., Locksley, H. B. and Nishroka, H. *Intracranial Aneurysms and Subarachnoid Haemorrhage: A Co-operative Study*. Philadelphia, PA, Lippincott, 1969.
10. Falconer, M. A., Serafetimides, E. A. and Corsellis, J. A. N. Etiology and pathogenesis of temporal lobe

- epilepsy. *Archives of Neurology (Chicago)* 1964; **10**: 233–248.
11. Lot, G., Houdart, E., Cophignon, J., Casasco, A. and George, B. Combined management of intracranial aneurysms by surgical and endovascular treatment. Modalities and results from a series of 395 cases. *Acta Neurochirurgica* 1999; **141** (6): 557–562.
 12. Dovey, Z., Misra, M. and Thornton, J. Guglielmi detachable coiling for intracranial aneurysms: the story so far. *Archives of Neurology* 2001; **58** (4): 559–564.
 13. Murata, T., Tsuruno, T., Shimotake, K. et al. Management strategies for unruptured cerebral aneurysms. *No Shinkei Geka-Neurological Surgery* 2001; **29** (10): 943–949.