Original Article

Epileptic Disord 2004; 6: 41-4

Unruptured intracranial aneurysm as a cause of focal epilepsy: an excellent postoperative outcome after intra-arterial treatment

Robert Kuba[1], Petr Krupa[2], Lenka Okáčová[1], Ivan Rektor[1]

[1] Department of Neurology, [2] Dpt. of Radiology, Brno Epilepsy Centre, Masaryk University, St Anne's Hospital, Brno, Czech Republic

Received August 11, 2003; Accepted November 27, 2003

ABSTRACT – This report involves a patient suffering from focal epilepsy caused by an unruptured, intracranial aneurysm (UIA), and her treatment using intravascular embolisation, with an excellent postoperative outcome in terms of the epilepsy. A 52 year-old, right-handed woman had been suffering for three years from focal seizures, characterized by loss of consciousness, and oroalimentary and hand automatisms. The epilepsy was not controlled by carbamazepine monotherapy. Magnetic resonance imaging and digital subtraction angiography revealed a saccular aneurysm at the bifurcation of the middle cerebral artery, in contact with the cortex of the temporal opercular region. An intra-arterial embolisation, using a detachable, mechanical spiral (MDS, Bold Platinum), was performed, with no complications. The purpose of embolisation was the treatment of the aneurysm. The patient had two more, complex partial seizures (CPS) in the early postoperative period, but was seizure-free between September 1998 and September 2002. This is the first case report that presents the successful treatment of UIA-related epilepsy by means of intravascular embolisation.

KEY WORDS: temporal lobe epilepsy, focal seizures, aneurysm, intravascular embolisation

Unruptured intracranial aneurysm (UIA) is a rare cause of epilepsy. Several cases have been reported in the literature [1-5]. Several types of seizures caused by UIA were described, mostly complex partial seizures (CPS) and sensory auras. In rare cases, the epilepsy caused by UIA can be refractory to antiepileptic drug (AED) treatment [3]. In general, clipping or ablation of the aneurysm has been effective in stopping epileptic seizures [4, 5]. We have no knowledge of a documented case of UIA associated with epilepsy that has been successfully treated by the use of intra-arterial embolisation.

Case study

This report concerns a 52 year-old, right-handed woman without any medically significant past history, and without perinatal trauma or febrile seizures. She had experienced complex

Correspondence:
Robert Kuba M.D.,
Department of Neurology,
Brno Epilepsy Centre, Masaryk University,
St Anne's Hospital, Brno, Pekarska 53,
Czech Republic 656 91
Tel.: +42 5 4318 2626
Fax: +42 5 4318 2624
E-mail: robert.kuba@fnusa.cz

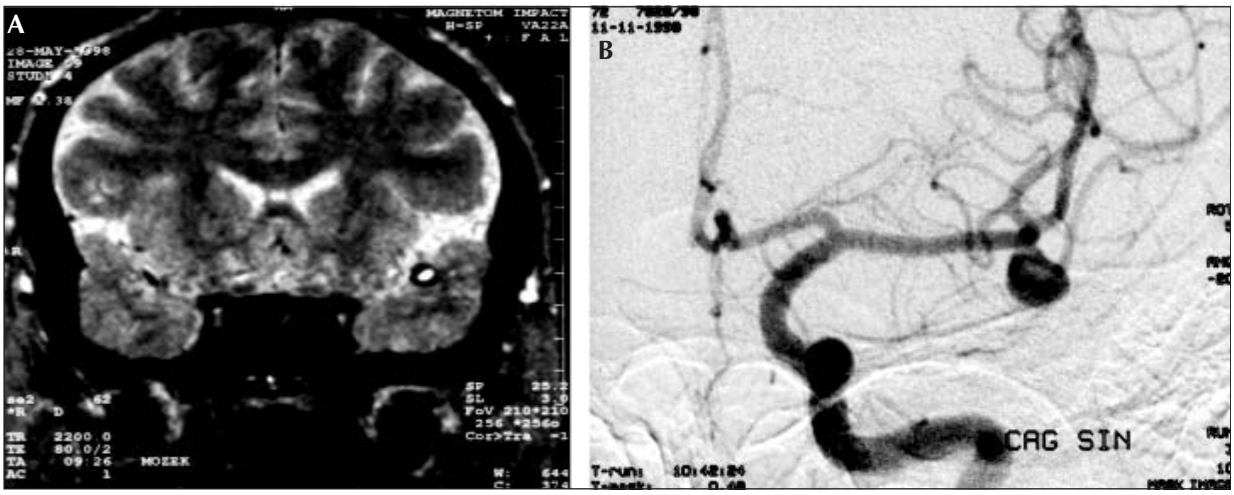

Figure 1. **A.** *T2-weighted MR (coronal view) – saccular aneurysm in contact with the temporal operculum.* **B.** *Digital subtraction angiography – saccular aneurysm on the bifurcation of the middle cerebral artery.*

partial seizures (CPS) without warning signs since the age of 49 years. Her seizures were described by her husband as states initiated by staring and then followed by oroalimentary and limb automatisms. CPS lasted from one to four minutes and were followed by dizziness and headache. The seizure frequency was from once to twice a week. Carbamazepine administration (800 mg daily) failed to control the seizures. The patient was brought to our department in June 1998 for evaluation. Both neurological and cardiological examinations were normal; scalp interictal EEG showed both focal slowing and epileptic sharp waves and spikes at the left temporal region, scalp ictal EEG revealed the seizure onset in the left temporal region. We noticed certain ictal lateralizing signs during the patient's CPS, which lateralized the seizure origin to the left (dominant) temporal lobe (right-sided, upper limb dystonia, postictal aphasia). Interictal HMPAO-SPECT showed left temporal hypoperfusion; a neuropsychological examination did not reveal any significant deficit in memory function. Magnetic resonance imaging (MRI) showed a small, probably vascular, lesion near the middle cerebral artery (MCA), which was in contact with the superior temporal gyrus (temporal operculum) on the left side *(figure 1A)*. A small saccular aneurysm on the MCA (bifurcation of MCA) was verified by digital subtraction angiography *(figure 1 B)*. No other pathology of the left temporal lobe, including the mesiotemporal structures, was detected by MRI *(figure 2)*. After the control angiography, the intra-arterial embolisation, using a detachable mechanical spiral (MDS, Bold Platinum),

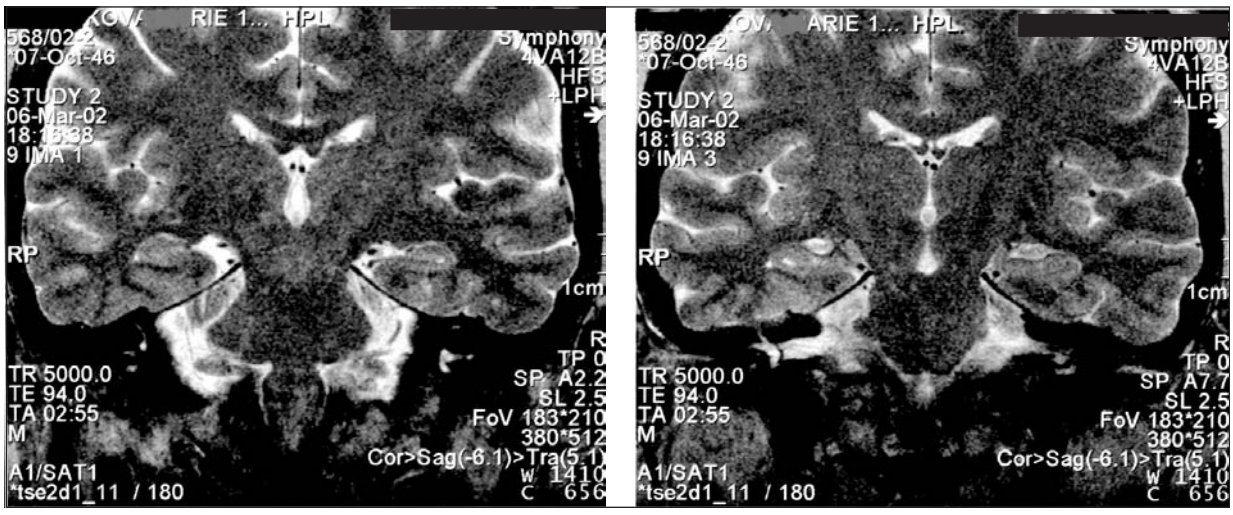

Figure 2. *T2-weighted MR (coronal view) – no other pathology of the left temporal lobe, symmetrical hippocampi with no change in the intensity.*

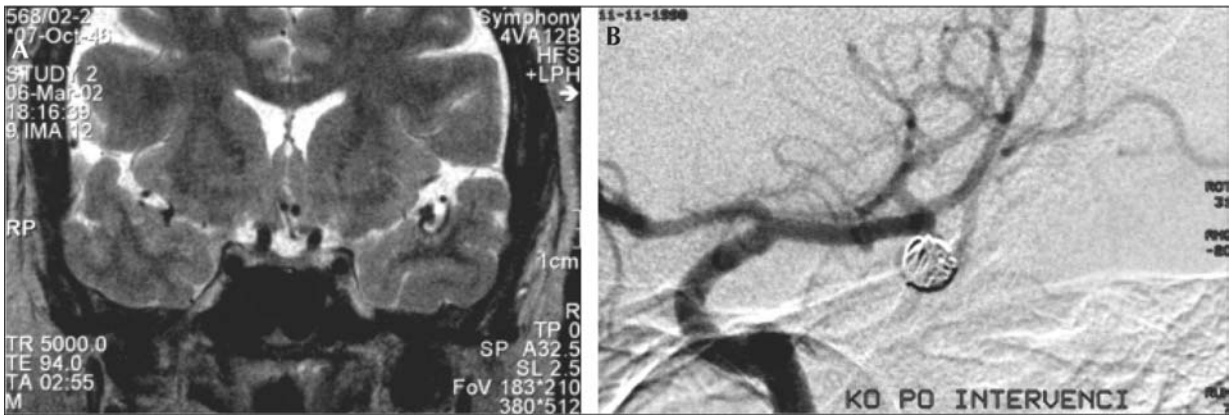

Figure 3. **A.** *T2-wighted MR (coronal view).* **B.** *Digital subtraction angiography. The situation after the intravascular embolisation.*

was performed, with no complications *(figure 3A,B)*. This procedure did not cause any neurological deficit. The patient had two more CPS in the early postoperative period; between September 1998 and September 2002 she was seizure-free. The patient no longer uses antiepileptic medication; carbamazepine treatment was discontinued in 2001.

Discussion

The combination of UIA and epilepsy is a relatively rare condition. The incidence of epilepsy in patients with UIA varies. Although Currie *et al.* [1] reported one single case in 666 temporal lobe epilepsy patients (0.2%), the percentage of epilepsy in other UIA series, as reported by Morley and Barr [6] and Yomin *et al.* [7] (10.7% and 8%, respectively), is much higher. In these series [1-7], the most frequent localization was on the MCA or on the posterior cerebral artery (PCA), and in most case reports, the aneurysms were large or giant.

The development of epilepsy in patients with intracerebral aneurysms has several explanations. The first explanation suggests that the aneurysm may be the source of thrombotic emboli, which directly cause the focal ischemia [2]. Focal ischemic lesions could be a cause of focal epileptic seizures. The second explanation involves direct cortical compression by the UIA [2, 5, 8]. This can be confirmed during a surgical procedure (aneurysm clipping), and has been reported frequently. The direct compression of the cortex may lead to the focal gliosis of cerebral tissue, and this is associated with epilepsy [3]. A third possible explanation of the epileptogenicity of UIA may be minor recurrent bleeding from the aneurysm, as suggested by Sengupta *et al.* [2]. This may lead to the focal destruction of the cerebral tissue and the further development of the epileptic focus. Direct intraoperative observation of local bleeding or hemosiderin deposit, or an MRI finding of the hemosiderin layer surrounding the aneurysm are the rationale for this explanation. The fourth possible explanation of potential epileptogenicity of UIA is intermittent compression of underlying cortical tissue due to the pulsation of UIA [9]. Nevertheless, the exact mechanism of the epileptogenicity of intracranial aneurysms is unknown, and the combination of more factors may be possible.

We document the case of a small UIA on the MCA, associated with epilepsy, which was, from an epileptologic point of view, successfully treated by means of an intravascular embolisation. Preoperative MRI showed hemosiderin deposits, which were probably caused by recurrent subclinical bleeding. As seen in the preoperative and postoperative MR scans, there was no reduction of aneurysmal size in our patient. This fact may lead to the suggestion that in terms of the pathophysiology, at least two different mechanisms of epileptogenicity of UIA could be involved in this case, i.e. local subclinical bleeding and intermittent pulsation of the aneurysm, which were abolished by coil embolisation.

As has been previously reported, most patients with UIA and epilepsy are seizure-free after surgical procedures such as clipping and ablation of the UIA [4, 5, 8]. At least in these cases, the functional changes caused by the UIA were reversible. Although UIA is not a common cause of focal epilepsy, intravascular embolisation may be a successful treatment for the focal epilepsy, although it is primarily performed for the treatment of aneurysm. □

References

1. Currie S, Heathfield KWG, Henson RA, Scott DF. Clinical course and prognosis of temporal lobe epilepsy: a survey of 666 patients. *Brain* 1971; 94: 173-90.

2. Sengupta RP, Saunders M, Clarke PR. Unruptured intracranial aneurysms – an unusual source of epilepsy. *Acta Neurochir (Wien)* 1978; 40: 45-53.

3. Yacubian EM, Rosemberg S, da Silva HC, Jorge CL, de Oliveira E, de Assis LM. Intractable complex partial seizures associated with posterior cerebral giant aneurysm: a case report. *Epilepsia* 1994; 35: 1317-20.

4. Ellamushi H, Thorne L, Kitchen N. Unruptured cerebral aneurysms causing seizure disorder (report of two cases). *Seizure* 1999; 8: 310-2.

5. Mizobuchi M, Ito N, Tanaka Ch, Sako K, Sumi Y, Sasaki T. Unidirectional olfactory hallucination associated with ipsilateral unruptured intracranial aneurysm. *Epilepsia* 1999; 40: 516-9.

6. Morley TP, Barr HWK. Giant intracranial aneurysms: diagnosis, course, and management. *Clin Neurosurg* 1969; 16: 73-94.

7. Jomin M, Lesion F, Lozes G, Fawaz A, Villette L. Surgical prognosis of unruptured intracranial arterial aneurysms: report of 50 cases. *Acta Neurochir (Wien)* 1987; 84: 85-8.

8. Whittle IR, Allsop JL, Halmagyi GM. Focal seizures: an unusual presentation of giant intracranial aneurysm. *Surg Neurol* 1985; 24: 533-40.

9. Stewart RM, Samson D, Diehl J, Hinton R, Ditmore QM. Unruptured cerebral aneurysms presenting as recurrent transient neurologic deficits. *Neurology* 1980; 30: 47-51.