

Case Report

Unruptured aneurysm of the middle cerebral artery presenting with psychomotor seizures: case study and review of the literature

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Abstract

An intracranial aneurysm would be low on the differential diagnosis of a patient presenting with behavioral or emotional changes. Nonetheless, complex partial seizures (CPS) may cause such symptoms and result from an unruptured intracranial aneurysm. Failure to diagnose and treat this condition in a timely manner increases the patient's risk of catastrophic aneurysmal rupture. This report describes a 55-year-old woman who presented following two CPS which began with the perception of a strange smell and culminated in a brief loss of consciousness. She had no history of seizure disorder or recent trauma. Magnetic resonance imaging (MRI) revealed a space-occupying lesion over the right temporal lobe near the amygdala. Magnetic resonance angiography (MRA) confirmed a 1.5-cm right middle cerebral artery aneurysm, with a dome projecting toward the amygdalohippocampal region. Following surgical ablation, the patient's CPS were well controlled. A review of the literature is performed on this unusual etiology and management strategies are discussed.

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1. Introduction

In patients who present with behavioral or emotional symptoms, the index of suspicion of an intracranial aneurysm as the etiology is low. Complex partial seizures (CPS) may cause such symptoms and can result from unruptured intracranial aneurysms. While the importance of early diagnosis of an unruptured aneurysm is inarguable, long periods frequently elapse before their discovery, even if they are symptomatic. This is the result of nonspecific symptoms and inherent difficulties in diagnostic testing.

Unruptured aneurysms that result in seizures usually involve the middle cerebral artery (MCA), likely secondary to its location adjacent to the temporal lobe. Our patient presented with mental status changes secondary to CPS as the first manifestation of what was found to

be a MCA aneurysm. The aneurysm was clipped successfully and the patient's seizures were well controlled postoperatively. This demonstrated the importance of including unruptured aneurysms in the differential diagnosis of patients with acute-onset behavioral changes.

2. Case report

A 55-year-old woman presented following two CPS, which began with the perception of a strange smell and culminated in a brief loss of consciousness. She had no history of seizure disorder or recent trauma. Magnetic resonance imaging (MRI) revealed a space-occupying lesion involving the right temporal lobe near the amygdala. There also seemed to be increasing signal intensity surrounding the lesion, suggesting a several-week-old subclinical hemorrhage, i.e., a small leak from the aneurysm that did not result in obvious symptomatology (Figs. 1 and 2). Because MRI was suggestive of a vascular lesion, magnetic resonance angiography

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Fig. 1. Coronal T1 magnetic resonance image demonstrating right mesiotemporal abnormality. Increasing signal intensity surrounding the lesion suggests a previous subclinical hemorrhage.

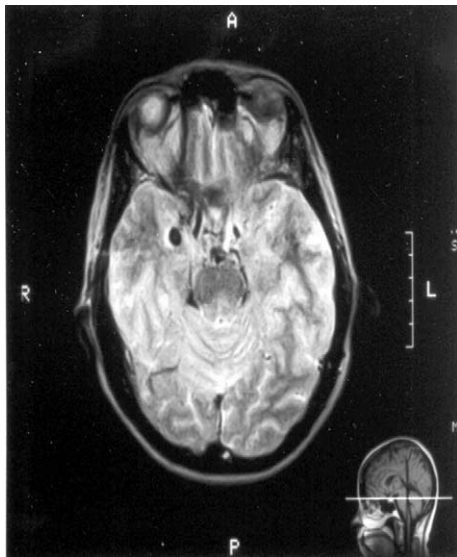


Fig. 2. Axial T2 magnetic resonance image demonstrating right mesiotemporal abnormality.

(MRA) was performed and confirmed a 1.5-cm right MCA aneurysm, with a dome projecting in the lateral, inferior, and slightly posterior direction toward the amygdalohippocampal region. There appeared to be some intraluminal thrombi and the MCA gave a proximal trunk, which subsequently branched into a third M2 segment, as well as an anterior temporal branch. Immediately after the origin of this vessel the large, broad-based aneurysm arose.

The patient was brought to the operating room and a distal transsylvian approach craniotomy was performed.

The aneurysm, clearly projecting in the aforementioned direction, was pushing into the amygdaloid region. A large arterial branch that gave rise to the aneurysm was identified, and the third M3 branch and anterior temporal artery were dissected from the neck of the aneurysm, which had a quite broad base. The patient was placed in a burst suppression coma and a temporary clip was applied to the trunk of origin, as well as on the anterior temporal and the M3 branches. The aneurysm was then deflated and a curved Yasargil clip applied. The operation concluded without incident and the patient was discharged in excellent condition, with her CPS well controlled with carbamazepine.

3. Discussion

3.1. Presentation of unruptured intracerebral aneurysms

Although intracerebral aneurysms commonly come to the attention of the neurosurgeon when they hemorrhage, a significant proportion of unruptured aneurysms go undetected. These are usually diagnosed incidentally or when they produce nonspecific symptoms, and weeks to years may elapse before diagnosis [1]. Often, seizures are the first presenting feature of unruptured intracranial aneurysms, and a significant number of patients who suffer subarachnoid hemorrhage (SAH) have been previously diagnosed with epilepsy [1–3]. A study in 1980 [4] reported on 38 intracranial aneurysm cases that became apparent by nonhemorrhagic signs and symptoms. In 31 of 38 cases, the aneurysm was directly related to the clinical manifestation. Two of these cases presented with seizures. In 1999, two patients were reported who had seizures as a presenting feature of unruptured intracranial aneurysms in which the seizures became well controlled after surgical ablation of the aneurysm [5]. In 1966 a 2-year-old girl was reported with an unruptured posterior cerebral artery aneurysm that presented with seizures [6]. Following pterional craniotomy and clipping of the aneurysm, the seizures became well controlled.

3.2. Complex partial seizures

Complex partial seizures are characterized by a change in alertness or awareness, with behavioral or emotional symptoms and the temporary loss of memory. They can have motor, sensory, somatosensory, autonomic, or psychic components and are prevalent in approximately 1–5 of 1000 people. A rare cause of these seizures is unruptured cerebral aneurysm (Tables 1 and 2). CPS can present with psychiatric symptoms such as schizophrenic behavior [7–13]. In 1996 there was a report of a patient who was arrested for attempted rape and confessed to the act, but claimed partial amnesia

Table 1
Unruptured intracranial aneurysms presenting with CPS

Author(s)	No. of aneurysms	Age/sex	Size	Location	Proposed mechanism ^a	Treatment	Seizure resolution	Seizure type	Evidence of bleeding ^b	Duration of symptoms
Whittle et al. [14]	1	50/F	Giant	L MCA	Compression, subclinical hemorrhage	Clipping/decompression	Complete	CPS	Yes	10–15 years
	1	52/M	Giant	L MCA	Compression	Clipping/decompression	Complete	CPS, GTC	No	8 years
	2	59/M	Giant 5 mm	L MCA L ACom	Compression	Wrapping	No resolution	CPS, TIAs	No	4 years
	2	48/F	Giant small	R MCA R MCA	Compression	Wrapping	No resolution	CPS, focal Seizures	Yes	7 years
Yacubian et al. [28]	1	30/M	Giant	R PCA	Compression, partially thrombosed giant aneurysm	Selective amygdalohippocampectomy, PCA occlusion	Complete	CPS, GTC	Yes	12 years
Tanaka et al. [17]	1	2/F	15 × 8 mm	R MCA	Compression, subclinical hemorrhage, thromboembolism	Spontaneously thrombosed	Complete	CPS	No	4 months
Stewart et al. [38]	1	40/M	12 × 20 mm	L MCA	Compression, thromboembolism	Wrapping then clipping	Yes	CPS, motor seizure	No	8 months
Bull [25]	1		Giant			None		CPS		4 years
	1		Giant			None		CPS		5 years
	1		Giant			None		CPS		12 years
Kamrin [20]	1	45/M	>1 cm	R MCA	Compression	R temporal lobectomy	Yes	CPS, GTC	No	1.5 years
Locksley et al. [16]	1			ACA						
	1			PCA						
	1			PCA						
	1			MCA						
Currie et al. [48]	1			MCA				CPS		
Miyagi et al. [49]	1		Giant	MCA		Excision of aneurysm	Complete	CPS		
Huang et al. [6]	1	2/F		PCA	Compression	Clipping	Well controlled	CPS, GTC	Yes	6 months
Merva et al. [50]	1	57/M	Giant	R PCoA	Subclinical hemorrhage	Clipping	Well controlled	CPS, GTC	Yes	9 years
Frankel and Alpers [51]	1	49/M	4 cm	R MCA	Compression	Surgical ligation	Poor control	CPS, GTC		3 years
	1		8 mm	L MCA		None		CPS	No	6 months

Sengupta et al. [18]	1	52/M	Small	L MCA	Subclinical hemorrhage	Clipping	Complete	CPS	Yes	5 months
	1	56/F	Large	R MCA	Subclinical hemorrhage	Clipping and excision	Complete	CPS	Yes	30 years
Mizobuchi et al. [52]	1	49/F	Large	R MCA	Compression	Clipping	Complete	CPS	Yes	6 months
Pasqualin et al. [21]	1	31/F	Giant	R MCA	Compression	Clipping and excision	Complete	CPS	No	9 years
Sahs et al. [43]	1	65/M	10 mm	L ICA	Compression, local gliosis	Clipping	Complete	CPS, secondary generalization	No	1 day

^aMechanism proposed by author.

^bEvidence of bleeding such as hemosiderin deposits and CT/MRI signs.

and had no explanation for the offense. A witness had seen the subject shortly before the event in “a poor state of orientation.” Three months following the subject’s conviction, he suffered a massive SAH from an anterior communicating artery aneurysm [13]. Whittle et al. [14] described four patients who experienced partial epileptic seizures for several years that were the result of giant cerebral aneurysms of the MCA. None of the patients had any history of SAH. The author reported operative findings of medial temporal and subfrontal cortex compression, infarction of the superior temporal gyrus, and evidence of previous subclinical aneurysmal hemorrhage. Two of the patients had resolution of seizures with clipping of the aneurysm and resultant decompression of the adjacent temporal lobe. The remaining two patients’ aneurysms could only be wrapped, and their seizures continued postoperatively and responded poorly to anticonvulsant medications.

3.3. Location

MCA aneurysms seem to be the most commonly involved with seizures [15,16]. This is likely due to the proximity of the arteries to the temporal lobe. Another possible explanation for the higher incidence of seizures related to unruptured MCA aneurysms could be that posterior cerebral artery (PCA) aneurysms may be diagnosed earlier secondary to focal symptoms or precocious hemorrhage. Raps et al. [1] found that aneurysms of the internal carotid artery and posterior circulation (including the PCA) were more likely to cause focal symptoms from mass effect than were anterior cerebral artery (ACA) and MCA aneurysms.

Tanaka et al. [17] reported a 20-month-old girl with an unruptured right MCA aneurysm who initially presented with CPS. This saccular aneurysm was not treated and the right MCA spontaneously thrombosed within 3 months, causing no neurological deficits. The patient’s seizures were controlled using anticonvulsant medications. The author postulated mechanisms of direct pressure effect, subclinical hemorrhage, and ischemia from thromboembolism as the cause of the seizures. Sengupta et al. [18] reported six cases of seizures related to unruptured aneurysms. These seizures occurred anywhere from 1 day to 30 years prior to discovery of the aneurysms, and all of the aneurysms involved the MCA. Of these patients, one had CPS and another psychic seizures. The seizures completely resolved in five of the patients who were treated with operative ablation of the aneurysm (they did remain on postoperative anticonvulsant medications). The only patient not to be operated on expired within 6 months secondary to a SAH. McCulloch and Ashworth [19] reported two cases of unruptured cerebral aneurysms presenting with temporal lobe epileptic attacks. In both of these cases the aneurysms were large and of the MCA. The authors

Table 2
Unruptured intracranial aneurysms presenting with seizures

Author	No. of aneurysms	Age/sex	Size	Location	Proposed mechanism ^a	Treatment	Seizure resolution	Seizure type	Evidence of bleeding ^b	Duration of symptoms
Provenzale et al. [47]	1	39/M	12 mm	R PCoA	Compression, subclinical hemorrhage	Clipping	Complete	Olfactory hallucinations	Yes	Longstanding
Raps et al. [1]	1		Large to giant							
	1		Large to giant							
Daly [53]	1		Large to giant							
Jomin et al. [54]	1									
	1							Uncinate fits		
	1							Seizure		
	1							Seizure		
Hook and Norlen [55]	1	11/F	1 cm	MCA		None		Seizure		
	1	24/F	1 cm	L MCA		Ligation	Complete	Visceral, aphasic		
Allegre and Vogouroux [56]	1							Uncinate		
Walton [57]	1					None		Uncinate		3 months
McCulloch and Ashworth (1982) [19]	1	41/F	4 × 1.8 cm	R MCA		Excision of aneurysm	Complete	GTC	No	5 months
	1	47 M	2.5 cm	R MCA		None		GTC	No	14 years
Sengupta et al. [18]	1	57/F	Large	L MCA	Subclinical hemorrhage	Clipping	Complete	Vertiginous seizure	Yes	1 year
	1	15/M	Large	R MCA	Subclinical hemorrhage	Clipping	Complete	GTC	Yes	2 months
	1	42/M	Large	L MCA	Subclinical hemorrhage	Ligation	Complete	GTC	Yes	1 day
	1	43/M	Large	R MCA	Subclinical hemorrhage	None		GTC	Yes	1 year
Liang-fu and Da-jie [30]	1		Giant	MCA				Seizure		
	1		Giant	MCA				Seizure		
Oka et al. [58]	1		Large			Autothrombosed		Seizure		
	1					Surgical ablation	Well Controlled			
Ellamushi et al. [5]	1					Surgical ablation	Well controlled			
Kamrin [20]	1	48/F	>1 cm	R MCA	Compression	Clipping	Decreased frequency	Uncinate, GTC, auditory	No	1 year
	2	53/F	2 × 1 cm, 1 × 1 cm	R MCA	Compression	Excision	Decreased frequency	Uncinate, auditory	No	10 years
	1	33M	4 × 5 cm	R MCA	Compression	Surgical ablation	Yes	Uncinate, visual	No	1 year

Author	Age	Sex	Location	Size	Basilar apex	Aneurysmal emboli	Surgical treatment	Complete	Transient neuro deficits	Autonomic seizure, GTC	Outcome
Leibrock et al. [31]	60	F	Giant	MCA	Basilar apex	Aneurysmal emboli	None	None	None	None	6 years
Morley and Barr [23]	1		Giant	MCA	MCA		Surgical ablation				years
Stewart RM [38]	1		Giant	MCA	MCA		Surgical ablation				years
	34	F	3 × 4 mm	R Ant choroidal	Thromboemboli	Clipping	Complete	Transient neuro deficits	No	No	1 month
	39	F	3 × 4 mm, 4 × 4 mm	R PCoAR MCA	Subclinical hemorrhage	Clipping	Yes	Transient neuro Deficits	Yes	Yes	8 months
	71	F	14 × 19 mm	L Carotid siphon	Subclinical hemorrhage, thromboemboli	None	None	Transient neuro deficits	No	No	36 months

^aMechanism proposed by author.

^bEvidence of bleeding such as hemosiderin deposits or CT/MRI signs.

clipped the aneurysm in one of the patients, and her seizures resolved. The second patient was not treated operatively and expired approximately 10 years later, at the age of 64 years, of unrelated causes. Together Kamrin [20], McCulloch and Ashworth [19], and Pasqualin et al. [21] reported 13 patients with epilepsy secondary to unruptured MCA aneurysms.

3.4. Size

The probability of CPS related to unruptured intracranial aneurysms increases with size [22]. Giant aneurysms, defined as being greater than 2.5 cm in diameter, account for approximately 5% of all verified intracranial aneurysms [21,23,24]. Because of their size more are detected before hemorrhage. They often present with transient ischemic attacks (TIA), dementia, or seizures [23–26]. Their mass effect could result in direct compression of the adjacent cortex [18,27], which can lead to injury, and sclerotic changes. These changes could become seizure foci, especially if the temporal lobe is involved. In a case of intractable CPS associated with a giant right PCA aneurysm reported by Yacubian et al. [28], neuropathologic findings demonstrated severe neuronal loss, gliosis, and an abundance of macrophages with hemosiderin. This damage was similar to that reported by Fried et al. [29] in patients with mesiotemporal lesions. While the mass effect associated with larger aneurysms contributes to the initiation of seizures, the etiology is likely multifactorial. Seizures from unruptured MCA aneurysms may also be the result of local irritation secondary to the pulsatile aneurysmal wall against the brain or from hamartomatous changes in local tissue. If an aneurysm becomes thrombosed, the walls can become calcified, causing irritation of the underlying cortex [18,30]. Another explanation for these seizures could be small hemorrhages resulting in adjacent cortex damage and glial scarring, which could develop into an epileptic focus. The intraoperative evidence of hemorrhage by-products in some patients supports this theory, as does an increasing signal around the aneurysm on MRI, as observed in our patient. Another possible cause of this condition is ischemic degeneration of the temporal lobe secondary to thromboemboli produced by the unruptured aneurysm [31–38].

3.5. Diagnosis

The diagnosis of an unruptured intracerebral aneurysm is difficult if it is asymptomatic or causes nonspecific symptoms. Nonetheless, the advantages of early diagnosis are underscored by the low morbidity and mortality enjoyed today for surgical repair prior to hemorrhage. If undetected, the mortality approaches 25% after the aneurysm's first rupture, with 50% morbidity for patients

who survive [39]. The importance of adequate diagnostic evaluation of this population is increased if one realizes that many patients in seizure clinics likely harbor unruptured intracranial aneurysms [32,33,40]. Because seizures resulting from these aneurysms are difficult to diagnose, their prevalence is likely underestimated and some patients classified as having idiopathic CPS treated with anticonvulsants [41] could be at risk of SAH. MRI has and will continue to be an important imaging technique in patients with new-onset seizures. Deposits of hemosiderin or gliosis may identify local subclinical hemorrhage. Perianeurysmal hyperintense T2 signal attributed to edema has previously been reported by Atlas et al. [42]. Infarctions secondary to emboli may also reveal themselves on MRI. MRI findings suggestive of a vascular lesion should prompt further studies, such as MRA and four-vessel digital subtraction angiography (DSA). It has been reported that 30–40% of aneurysms larger than 15 mm exhibit thrombosis, as do 10–15% of smaller aneurysms [20]. As MRA and computed tomography angiography (CTA) do not rely on flow to the extent that DSA does, the former tests may be preferable during an initial vascular lesion workup, which often includes thrombosed lesions.

3.6. Prognosis

It is unequivocal that the prognosis of aneurysm patients diagnosed and treated before rupture is much better than that of those who hemorrhage before treatment. It is possible that in some patients, seizures may be a sentinel event to a major hemorrhage. As mentioned above, fatal SAH shortly after three patients with giant aneurysms presented with seizures has been reported [18,26]. Sahs et al. [43] reported seizures in 95 of 2621 patients who later suffered SAH.

This patient population's prognosis in relationship to seizure control, however, is less predictable. Using the above-mentioned mechanisms of seizure focus origin, the prognosis of individual patients seizure control may be predicted. Signs of hemorrhage around the aneurysm worsen the prognosis of intractable or poorly controlled seizures secondary to permanent cortical damage. Signs of infarction likewise increase the possibility of difficult seizure control. These patients might require resection of the damaged tissue in addition to aneurysmal obliteration. If only compression is suspected, favorable seizure control following direct surgical obliteration might be expected.

3.7. Treatment

The goal in the management of patients with unruptured intracranial aneurysm-induced seizures is elimination of the aneurysm and seizure foci through either endovascular coiling or clipping, with resection of

abnormal tissue, if necessary. In this patient population obliteration of the aneurysm serves two purposes: it can be used to control seizures and to prevent a future SAH. The results with respect to control of seizures are usually remarkable. The majority of patients whose aneurysms are corrected by direct surgical clipping seem to remain seizure-free. Some studies have reported that wrapping or other indirect methods of correction are not as effective in controlling seizures as direct methods such as clipping and aneurysmectomy [25,26].

Since the favorable outcomes were reported for the endovascular management of ruptured intracranial aneurysms during the International Subarachnoid Aneurysm Trial (ISAT) [44], this treatment has become an important option. Several retrospective analyses have found that patients who undergo this less invasive treatment for unruptured aneurysms have shorter hospital stays and shorter recovery times compared those who undergo traditional surgery [45,46]. When considering this treatment option in patients with aneurysm-related seizures, several factors must be taken into consideration. Basic endovascular qualifications for treatment must be satisfied. The aneurysm must be amenable to endovascular coiling from a morphologic and anatomic standpoint (i.e., Does it have an appropriate dome-to-neck ratio for coil placement and is it reachable by an endovascular catheter?). The patient described in this report was disqualified from endovascular treatment based on the broad neck of the aneurysm. Factors specific to seizure-producing aneurysms must also be addressed. It is likely that larger aneurysms precipitate seizure activity secondary to mass effect. Craniotomy with clipping and/or aneurysmectomy would be more likely to reduce this effect. Aneurysm patients who present with seizures should be examined for peri-aneurysmal MRI signal abnormalities [47]. Such an abnormality would strengthen the argument that permanent cortical changes related to the aneurysm are the precipitant of the seizure. This subset of patients may require not only craniotomy for clipping but also possible resection of abnormal tissue.

4. Conclusion

In summary, we are reminded that unruptured cerebral aneurysms, commonly involving the MCA, should be taken into consideration in the differential diagnosis of a patient presenting with new-onset mental status changes or seizures of unknown etiology. These seizures could be the result of several aneurysmal complications including subclinical hemorrhage, a thromboembolic or blood flow-related ischemic process, local irritation, and a direct mass effect on the adjacent brain tissue.

The diagnosis of an unruptured intracerebral aneurysm is difficult owing to no or nonspecific symptoms,

and weeks to years may elapse before its discovery. The importance of early diagnosis is reinforced by the observation that in some patients, seizures may be a sentinel event to a major hemorrhage. Obviously, the prognosis of patients with aneurysms discovered before rupture is much better than that of patients who hemorrhage.

MRI can identify signs of subclinical hemorrhage or local irritation such as deposits of hemosiderin and gliosis. Infarctions secondary to emboli may also reveal themselves on MRI. If there is the suggestion of a vascular lesion during the initial workup of a patient with new-onset seizures, some type of angiography should be performed. Because a significant number of larger aneurysms thrombose, MRA, or CTA may be superior to DSA. Signs of hemorrhage or infarction around the aneurysm increase the likelihood that resection of the damaged tissue, in addition to aneurysmal obliteration, will be required for seizure control.

If the aneurysm is discovered before rupture, the obvious treatment of choice is elimination or repair prior to hemorrhage. This can be accomplished through either endovascular coiling or clipping with resection of abnormal tissue, if necessary. In addition to eliminating the risk of hemorrhage, eradication of the aneurysm usually provides excellent control or elimination of the patient's seizures.

References

- [1] Raps EC, Rogers JD, Galetta SL, et al. The clinical spectrum of unruptured intracranial aneurysms [see comments]. *Arch Neurol* 1993;50:265–8.
- [2] Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. Based on 6368 cases in the cooperative study. *J Neurosurg* 1966;25(2):219–39.
- [3] Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. *J Neurosurg* 1966;25(3):321–68.
- [4] Aimard G, Confavreux C, Devic M, Vighetto A, Boisson D. Non-hemorrhagic forms of intracranial arterial aneurysms. *Rev Neurol* 1980;136:419–29.
- [5] Ellamushi H, Thorne L, Kitchen N. Unruptured cerebral aneurysms causing seizure disorder (report of two cases). *Seizure* 1999;8:310–2.
- [6] Huang LT, Shih TY, Lui CC. Posterior cerebral artery aneurysm in a two-year-old girl. *J Formos Med Assoc* 1996;95:170–2.
- [7] Frydl V. Giant brain-base aneurysm—manifested by psychotic symptoms. *Fortschr Med* 1983;101:1815–8.
- [8] Okawa M, Maeda S, Nukui H, Kawafuchi J. Psychiatric symptoms in ruptured anterior communicating aneurysms: social prognosis. *Acta Psychiatr Scand* 1980;61:306–12.
- [9] Williams SE, Bell DS, Gye RS. Neurosurgical disease encountered in a psychiatric service. *J Neurol Neurosurg Psychiatry* 1974;37:112–6.
- [10] Storey P. Emotional disturbances before and after subarachnoid haemorrhage. *Ciba Found Symp* 1972;8:337–43.
- [11] Penrose R, Storey P. Emotional disturbance and subarachnoid haemorrhage. *Psychother Psychosom* 1970;18:321–5.
- [12] Thompson GN. Cerebral lesions simulating schizophrenia: three case reports. *Biol Psychiatry* 1970;2:59–64.
- [13] Mielke U, Donauer E, Luthe R. Subarachnoid hemorrhage from the anterior communication artery in a sex offender. considerations regarding liability. *Nervenarzt* 1996;67:781–4.
- [14] Whittle IR, Allsop JL, Halmagyi GM. Focal seizures: an unusual presentation of giant intracranial aneurysms: a report of four cases with comments on the natural history and treatment. *Surg Neurol* 1985;24:533–40.
- [15] Locksley HB, Sahs AL, Knowler L. Report on the cooperative study of intracranial aneurysms and subarachnoid hemorrhage: Section II. General survey of cases in the central registry and characteristics of the sample population. *J Neurosurg* 1966;24:922–32.
- [16] Locksley HB, Sahs AL, Sandler R. Report on the cooperative study of intracranial aneurysms and subarachnoid hemorrhage: 3. Subarachnoid hemorrhage unrelated to intracranial aneurysm and A-V malformation. A study of associated diseases and prognosis. *J Neurosurg* 1966;24:1034–56.
- [17] Tanaka K, Hirayama K, Hattori H, et al. A case of cerebral aneurysm associated with complex partial seizures. *Brain Dev* 1994;16:233–7.
- [18] Sengupta RP, Saunders M, Clarke PR. Unruptured intracranial aneurysms: an unusual source of epilepsy. *Acta Neurochir* 1978;40(1–2):45–53.
- [19] McCulloch DK, Ashworth B. Cerebral aneurysm presenting with epilepsy. *Postgrad Med J* 1982;58:94–7.
- [20] Kamrin RP. Temporal lobe epilepsy caused by unruptured middle cerebral artery aneurysms. *Arch Neurol* 1966;14:421–7.
- [21] Pasqualin A, Da Pian R, Colamaria V, Bardin PG. Giant unruptured aneurysm of the middle cerebral artery manifesting with epilepsy: successful surgical treatment. *J Neurosurg Sci* 1979;23:303–10.
- [22] Solomon RA, Fink ME, Pile-Spellman J. Surgical management of unruptured intracranial aneurysms. *J Neurosurg* 1994;80:440–6.
- [23] Morley TP, Barr HW. Giant intracranial aneurysms: diagnosis, course, and management. *Clin Neurosurg* 1969;16:73–94.
- [24] Whittle IR, Dorsch NW, Besser M. Giant intracranial aneurysms: diagnosis, management, and outcome. *Surg Neurol* 1984;21:218–30.
- [25] Bull J. Massive aneurysms at the base of the brain. *Brain* 1969;92:535–70.
- [26] Drake CG. Giant intracranial aneurysms: experience with surgical treatment in 174 patients. *Clin Neurosurg* 1979;26:12–95.
- [27] Falconer M, Serafetinides E, Corsellis J. Etiology and pathogenesis of temporal lobe epilepsy. *Arch Neurol* 1964;10:233–48.
- [28] Yacubian EM, Rosemberg S, da Silva HC, Jorge CL, de Oliveira E, de Assis LM. Intractable complex partial seizures associated with posterior cerebral artery giant aneurysm: a case report. *Epilepsia* 1994;35:1317–20.
- [29] Fried I, Kim JH, Spencer DD. Hippocampal pathology in patients with intractable seizures and temporal lobe masses. *J Neurosurg* 1992;76:735–40.
- [30] Liang-fu Z, Da-jie J. Large and giant intracranial aneurysms: diagnosis and surgical treatment. *Chin Med J (Engl)* 1987;100:392–7.
- [31] Leibrock LG, Bennett DR, Bloch S. Complex partial seizures associated with unruptured thrombosed basilar artery apex aneurysm. *Surg Neurol* 1983;19:17–20.
- [32] Wiebers DO, Whisnant JP, Sundt Jr TM, O'Fallon WM. The significance of unruptured intracranial saccular aneurysms. *J Neurosurg* 1987;66:23–9.
- [33] Eskesen V, Rosenorn J, Schmidt K, et al. Clinical features and outcome in 48 patients with unruptured intracranial saccular aneurysms: a prospective consecutive study. *Br J Neurosurg* 1987;1:47–52.

- [34] Yamaki T, Yoshino E, Higuchi T. Rapidly growing aneurysm. *Surg Neurol* 1986;26:301–5.
- [35] Antunes JL, Correll JW. Cerebral emboli from intracranial aneurysms. *Surg Neurol* 1976;6:7–10.
- [36] Duncan A, Caplan L, Rumbaugh C. Aneurysms: a source of cerebral emboli. *Neurology* 1979;29:592.
- [37] Taptas JN, Katsiotis PA. Arterial embolism as a cause of hemiplegia after subarachnoid hemorrhage from aneurysm. *Prog Brain Res* 1968;30:357–60.
- [38] Stewart RM, Samson D, Diehl J, Hinton R, Ditmore QM. Unruptured cerebral aneurysms presenting as recurrent transient neurologic deficits. *Neurology* 1980;30:47–51.
- [39] Batjer H. Aneurysmal subarachnoid hemorrhage: pathophysiology and sequelae. *Cerebrovascular disease*. Philadelphia: Lippincott-Raven; 1997. p. 889–99.
- [40] Wiebers DO, Whisnant JP, O'Fallon WM. The natural history of unruptured intracranial aneurysms. *N Engl J Med* 1981;304:696–8.
- [41] Witoonpanich R, Bunyaratavej S, Vejajiva A. Epileptic seizure in intracranial aneurysm. *J Med Assoc Thai* 1980;63:192–5.
- [42] Atlas S, Grossman R, Goldberg H, Hackney D, Bilaniuk L, Zimmerman R. Partially thrombosed giant intracranial aneurysms: correlation of MR and pathologic findings. *Radiology* 1987;162:111–4.
- [43] Sahs A, Perret G, Locksley H, Nishioka H. Intracranial aneurysms and subarachnoid hemorrhage: a cooperative study. Philadelphia, Toronto: Lippincott; 1969.
- [44] Molyneux A, Kerr R, Stratton I, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. *Lancet* 2002;360:1267–74.
- [45] Johnston SC, Wilson CB, Halbach VV, et al. Endovascular and surgical treatment of unruptured cerebral aneurysms: comparison of risks. *Ann Neurol* 2000;48:11–9.
- [46] Johnston SC, Dudley RA, Gress DR, Ono L. Surgical and endovascular treatment of unruptured cerebral aneurysms at university hospitals. *Neurology* 1999;52:1799–805.
- [47] Provenzale JM, Gorecki JP, Koen JL. Cerebral aneurysms associated with seizures but without clinical signs of rupture: seemingly distinctive MR imaging findings in two patients. *AJR Am J Roentgenol* 1996;167:230–2.
- [48] Currie S, Heathfield KW, Henson RA, Scott DF. Clinical course and prognosis of temporal lobe epilepsy: a survey of 666 patients. *Brain* 1971;94:173–90.
- [49] Miyagi J, Shigemori M, Sugita Y, Nishio N, Harada K, Kuramoto S. Giant aneurysm of the middle cerebral artery presenting with complex partial seizure: case report. *Neurol Med Chir (Tokyo)* 1991;31:953–6.
- [50] Merva W, Jamshidi S, Kurtzke JF. Posterior communicating artery giant aneurysm as a cause of seizures [letter]. *Neurology* 1985;35:620–2.
- [51] Frankel K, Alpers B. The clinical syndrome of aneurysm of the middle cerebral artery. *Arch Neurol Psychiatry* 1955;74:46–67.
- [52] Mizobuchi M, Ito N, Tanaka C, Sako K, Sumi Y, Sasaki T. Unidirectional olfactory hallucination associated with ipsilateral unruptured intracranial aneurysm. *Epilepsia* 1999;40(4):516–9.
- [53] Daly D. Uncinate fits. *Neurology* 1958;8:250–60.
- [54] Jomin M, Lesoin F, Lozes G, Favaz A, Villette L, Autricque A. Surgical prognosis of unruptured intracranial arterial aneurysms: 50 cases. *Presse Med* 1987;16:375–7.
- [55] Hook O, Norlen G. Aneurysms of the middle cerebral artery: a report of 80 cases. *Acta Chir Scand* 1958;235:1–39.
- [56] Allegre G, Vigouroux R. Traitement chirurgical des aneurysmes intracranien du systeme carotidien. Paris: Masson & Cie; 1957.
- [57] Walton J. Subarachnoid hemorrhage. Edinburgh: E & S Livingstone; 1956.
- [58] Oka H, Kurata A, Miyasaka Y, et al. Completely thrombosed large aneurysm of the distal middle cerebral artery: a case report. *No Shinkei Geka* 1994;22:677–80.