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Epilepsy After Operative Treatment of Ruptured Cerebral Aneurysms

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Summary

A retrospective analysis of 183 consecutive patients operated on for ruptured cerebral aneurysms and surviving at least one year revealed appearance of postoperative epilepsy in 14 cases (8 per cent) on an average of 10 months (range 0–23 months) after the operation. Factors associated with the development of secondary epilepsy were localization of the aneurysm on the middle cerebral artery, temporary clipping intraoperatively, wrapping technique to treat the aneurysm, and vasospasm seen on the postoperative control angiogram. Intraoperative and/or postoperative ischaemia seems to be the crucial phenomenon favouring the development of epilepsy. Identification of the risk factors may help to focus the anti-epileptic prophylaxis in cases prone to develop seizures.

Keywords: Aneurysm surgery; secondary epilepsy; temporary clipping; wrapping; vasospasm.

Introduction

Secondary epilepsy complicates many neurosurgical diseases, for example subarachnoid haemorrhage (SAH), in which postoperative seizures are reported to occur in 4.5 to 27.5 per cent of cases[10]. Among the diseases with marked incidence of secondary epilepsy, SAH and head injuries are good models for investigating the development of the epilepsy de novo in the sense that the onset of the primary disorder can be exactly determined. Although epilepsy in association with these diseases is usually a smaller problem than neurological defects or neuropsychologic disturbances, repeated seizures may crucially complicate both the acute phase and the postoperative period[14].

The aim of the present study was to clear aetiological factors, which may have influence on the development of epilepsy after aneurysm surgery. Special attention was paid to the appearance of first seizures, to the localization of the aneurysm, and to intraoperative details.

Patients and Methods

The series consists of 183 consecutive patients operated on for ruptured supratentorial aneurysms in Oulu University Central Hospital during 1984–1988 and surviving at least one year postoperatively. The data were collected retrospectively from preoperative records, and from control notes made by the neurosurgeon 2–3 months after the operation. The condition of the patients was followed by questionnaire and checking patient journals later to get information from their actual situation. The mean follow up time was 3.6 years (range 1.5 to 5.5. years). All patients underwent surgery through a standard frontotemporal or pterional craniotomy using the microscope. Aneurysms were clipped with various types of Sugita clips in 161 cases and in 22 cases were additionally wrapped using temporal fascia, cotton and/or methylmethacrylate glue. Temporary clipping was needed in 11 operations. In other 11 patients SAH was complicated by intracerebral haematoma, which was evacuated at the same operation. As prophylactic anticonvulsant we used routinely diphenytoin for at least 3 months. Nimodipine was started intraoperatively and continued for 3 weeks after the operation in all patients.

Neuropsychological tests (parts of WAIS, symmetry, 100 minus 7 -test, memory tests) were performed in cases with problems of working capacity 3–6 months postoperatively. Control carotid angiography was done routinely 1–2 weeks after the operation for the first years of the study, but later only selectively, so that altogether angiograms of 123 patients could be evaluated.

For statistical processing Fisher's two tailed exact test was used.

Results

Postoperative epilepsy was regarded as repeated seizures, which were encountered in 14 patients (8 per cent). The most common type was primarily generalized tonic-clonic convulsions. The mean age of patients with epilepsy was 37 years. There were nine males and five females. Nine of these patients were preoperatively in grade I–II (Hunt and Hess), and five in grade III. There were no statistical differences in sex, or age distribution, nor in clinical grades between the epileptic and non-epileptic groups. In 11 out of the 14 patients

Table 1. *Types and Timing of Epileptic Attacks After Aneurysm Surgery*

Patient	Type of attack	Timing of first attack (months)
1	GM	6.5
2	GM	9
3	focal, secondarily generalized	7
4	focal, secondarily generalized	13
5	focal, secondarily generalized	14.5
6	focal, secondarily generalized	21
7	GM	12
8	GM	12
9	abcences	0
10	GM	7
11	focal, secondarily generalized	7
12	GM	23
13	GM	11
14	GM	1
		mean 10 months

GM = primarily generalized tonic-clonic convulsions.

Table 2. *Clinical Data of Patients with Epilepsy*

Patient	Sex	Age	Localization	Preoperat. grade	Operation day
1	M	33	MCA sin.	I	4
2	F	58	MCA dx.	II	9
3	M	33	MCA sin.	III	7
4	M	40	MCA dx.	III	6
5	F	26	MCA sin.	I	2
6	M	44	MCA sin.	I	5
7	M	45	MCA dx.	III	9
8	F	21	ICA sin.	I	4
9	F	46	MCA dx.	I	7
10	M	37	MCA dx.	III	8
11	M	35	ICA dx.	I	5
12	M	36	MCA sin.	III	12
13	M	37	MCA dx.	I	2
14	F	26	CoA dx.	I	6
					mean 6.1

Table 3. *Details of the Operation Technique in Patients with Postoperative Epilepsy*

Patient	Operation technique	Temporary clipping
1	clipping	4 minutes
2	clipping + wrapping	5 minutes
3	clipping	5 minutes
4	clipping + wrapping	–
5	clipping + wrapping	–
6	clipping + wrapping	–
7	clipping + wrapping	–
8	clipping + wrapping	–
9	clipping + wrapping	–
10	clipping + evacuation	–
11	clipping	–
12	clipping + evacuation	–
13	clipping	–
14	clipping	–

Wrapping = reinforcing with fascia, cotton and/or methylmethacrylate glue; evacuation = evacuation of intracerebral haematoma.

Table 4. *Postoperative Outcome in Patients with Epilepsy*

Patient	Neurological defect	Neuropsychologic disturbance	Outcome
1	dysphasia	memory, visual	G
2	–	–	G
3	dysphasia, hemiparesis	memory, visual	M
4	hemiparesis	memory	G
5	–	memory	E
6	–	slowness of thinking	G
7	hemiparesis	–	G
8	–	memory, writing	G
9	hemiparesis	–	G
10	–	memory, slowness	G
11	hemiparesis	slowness, visual	G
12	dysphasia, hemiparesis	nonverbal activity	M
13	–	memory, learning	G
14	–	intellectual flexibility	E

E = excellent, working.
G = good, self sufficient.
M = moderate, needs help in some daily activities.

with postoperative epilepsy, the aneurysm was situated on the middle cerebral artery (79 per cent), whereas in those without epilepsy this localization was statistically significantly less common (34 per cent, $p = 0.0015$, Fisher's test). Table 1 summarizes the types and timing of postoperative seizures, and a detailed clinical data of patients with postoperative epilepsy is shown in Table 2.

Preoperatively intracerebral haematoma was documented in 9 out of the 169 patients without secondary epilepsy and in 2 out of 14 suffering from it. The difference in incidences of intracerebral haematoma in these groups is not statistically significant. Additional wrapping was needed in 7 out of the 14 patients with epilepsy and only in 15 out of the 169 without it. This difference is statistically significant ($p = 0.0003$). Temporary clipping was used in 3 out of 14 patients with epilepsy but only in 8 out of 169 patients without epilepsy, the difference being significant ($p = 0.041$). Details of the operative technique are shown in Table 3.

Postoperative control carotid angiograms were performed one week after the operation. Angiographic spasm, i.e. diffuse or local narrowing of the lumen of the arteries by 25 per cent or more was seen in 8 patients out of 12 with epilepsy and in 37 out of 111 patients without epilepsy. The difference between these groups is statistically significant ($p = 0.027$). Postoperative outcome was evaluated one year after the operation and is summarized in Table 4.

Discussion

In the present study factors associated with the development of secondary epilepsy were 1) localization of the aneurysm on the middle cerebral artery, 2) temporary clipping intraoperatively, 3) wrapping technique to treat the aneurysm, and 4) vasospasm on the postoperative check angiogram. On the basis of these findings, especially 2) and 4), intraoperative and/or postoperative ischaemia seems to be the crucial phenomenon favouring the development of epilepsy.

In a recent study on patients with head injury[8], posttraumatic epilepsy was observed to appear on average within one year after the trauma. Similar latency was seen in the present study also after SAH and aneurysm operation as there was a median latency of 10 months from the bleeding to the first attack. In head injuries the occurrence of traumatic intracerebral haematoma and primary seizures were significant factors associated with the development of epilepsy[8], but not in the patients of the present investigation. Both head injuries and SAH cause great changes in local cerebral blood flow: trauma patients suffering from post-traumatic epilepsy presented significantly impaired local cerebral blood flow both in the acute stage and several months after the injury; in aneurysm patients with secondary epilepsy check carotid angiography revealed significantly more often spasm than in the group without epilepsy. Although angiographic spasm does not directly mean clinically important vasospasm; it seems to predict well the occurence of postoperative epilepsy and may be an important sign of ischaemia which later on leads to clinical seizures.

Nimodipine is reported to be able to prevent ischaemic neurological deficits, although its effect on angiographic spasm is not clear. In addition to the prevention of vasospasm on capillary level, the mechanism of action of nimodipine might include also a protective effect on brain cells against harmful agents released by ischaemia[7]. Further, nimodipine is shown to exhibit anticonvulsive activity[2], which might be important in preventing postoperative epilepsy.

Lesions in the territory of the middle cerebral artery seem to lead most commonly to secondary epilepsy[10]. According to Glaser[6] the temporal lobe is susceptible to various physical distortions, mainly due to its vascular supply consisting of peculiar "rake-like" branchings. Certain parts, particularly the hippocampus, have very high density of neurons. Therefore, interaction between neurons becomes possible and accumulation of enhanced $K+$ may happen in the extracellulary space, so that seizures can be generated even in the absence of synaptic transmission[4]. In the present study, epileptic attacks as well as memory deficits seemed to be concentrated on the patients with MCA aneurysms. This is in accordance with the results of Larsson *et al.*[11] showing that neuropsychological deficits and their specific nature depend on the localization of the ruptured aneurysm. Both neuropsychological problems and epilepsy must be regarded as milder sequelae than neurological deficits.

The frequency of 8 per cent of secondary epilepsy in the present series is low compared with the 10–27 per cent rate in many earlier surgical series[10]. This may be a result of improvement in surgical and anaesthetic techniques including the use of the microscope, better aneurysm clips and instruments, better medication against spasm, and better appreciation of the microanatomy. Also the routinely given prophylatic diphenytoin medication could have contributed to the lower incidence of epilepsy.

Postoperative scarring and atrophy may be minimized by avoiding cortical damage, by using minimal gyrus resection, compression by retractor for as short a time as possible, and taking care of perforating vessels in order of minimize parenchymal brain damage. Wrapping and temporary clipping are sometimes necessary, but the price of these techniques seems to be an increased risk of epilepsy. Even in these cases with high risk, intensified medical therapy consisting of use of nimodipine pre- and postoperatively and routine anti-epileptic prophylaxis[3, 12] at least for one year is anticipated to lead to a clinically acceptable result.

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