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Volume 153 · Number 11 · November 2011

Acta
Neurochirurgica

The European Journal
of Neurosurgery

The Official Organ
of the European Association
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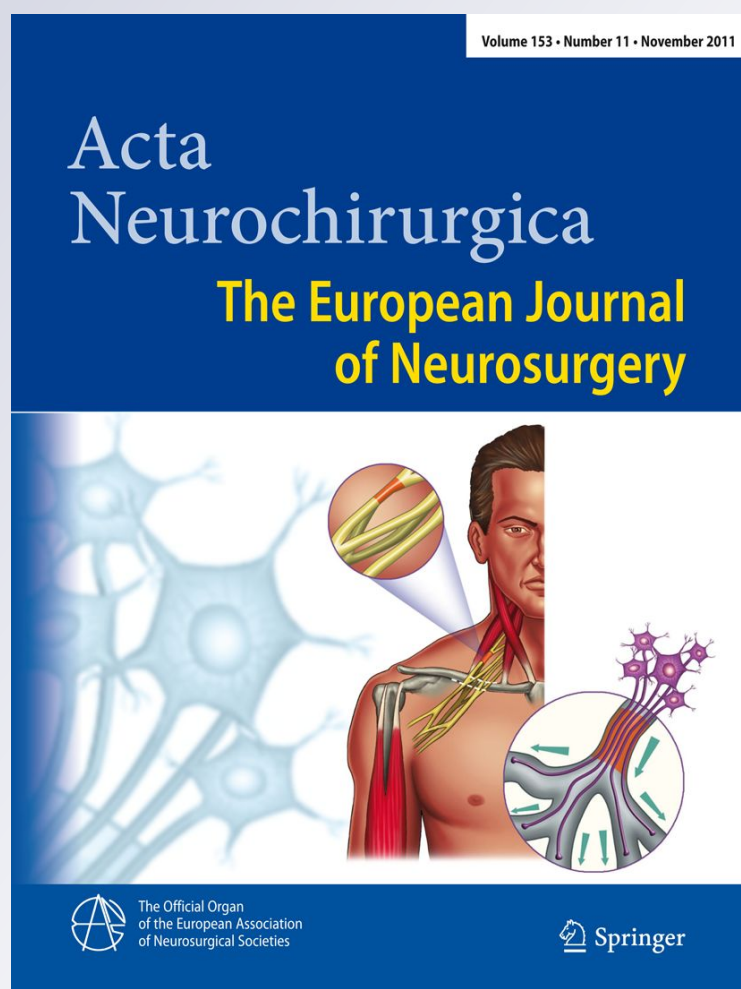Springer

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CLINICAL ARTICLE

Late ophthalmological assessment of patients with subarachnoid hemorrhage and clipping of cerebral aneurysm

Iwona Obuchowska · Grzegorz Turek · Zenon Mariak ·
Jan Kochanowicz · Zofia Mariak

Abstract
Purpose To estimate prospectively late ocular manifestations in patients after aneurysmal subarachnoid hemorrhage (SAH) treated with aneurysm clipping.
Methods Forty-six patients (12 men and 34 women), 23–69 years of age, were included in this study. A conventional ophthalmological examination, visual evoked potentials (VEPs), and static perimetry were performed on all patients. The mean interval between the onset of SAH and the aforementioned examination was 1.9 ± 1.3 years (range 0.5-5 years). The following were compared between patients with affected and non-affected visual fields as well as between those with normal and abnormal VEPs: sex, age, time from SAH to surgery, Hunt and Hess scale, Glasgow Coma Scale, Glasgow Outcome Scale, grading of SAH according to the Fisher scale, and the size and site of aneurysm.
Results Visual field defects were found in 23 patients (50%). In all of these patients, both eyes were affected. The most frequent type of visual field defects were: constricted field (47.8%), multiple peripheral foci (26.1%), and superior field defect (17.4%). There was no significant relationship between the analyzed factors and the occurrence of visual field defects, although statistical significance was almost observed in respect to the Fisher scale ($p=0.055$).

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Deterioration in VEPs was observed in nine patients (19.6%). In the group of patients with abnormal VEPs, the time from onset of SAH to surgery was 2.6 ± 1.8 days, whereas in the group of patients with normal VEPs this time amounted to 6.4 ± 2.4 days ($p=0.02$). In patients with no changes in VEPs, the mean Fisher score was significantly higher than in the group with abnormal VEPs (2.8 ± 0.6 vs 2.0 ± 0.4 respectively, $p=0.04$).
Conclusion Visual field defects and VEP deterioration are frequent late ocular manifestations of SAH treated with aneurysm clipping. Damage to the visual pathway correlates with the severity of SAH and timing of aneurysmal surgery.

Introduction

Data pertaining to the late ophthalmologic outcomes of aneurysmal subarachnoid hemorrhage (SAH) treated surgically are very scarce. This is in contrast to the amount of data pertaining to early ophthalmologic sequelae of intracranial aneurysmal rupture. In 1881, Moritz Litten described retinal, preretinal, and vitreous bleedings associated with SAH [10]. A number of subsequent studies have shown that most of these effusions clear within months of onset, except massive bleedings to the vitreous body, known as Terson's syndrome [1, 2, 4, 12–14, 20]. This condition often necessitates that the ophthalmologist must have an active approach to treatment (e.g., extraction with vitrectomy). As such, there is plenty of literature pertaining to the late follow-up of patients with Terson syndrome, although this literature pertains to one of

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the least frequent types of ocular hemorrhagic complications of SAH [3, 5, 6, 9, 17, 18]. We did not find any clinical series pertaining to the late general ophthalmological assessment of a group of patients after aneurysmal SAH.

This problem is not trivial since up to 30% of patients after aneurysmal SAH and surgery become dependent patients, while 30-70% of patients suffer from disturbed executive, visual-spatial, or social cognitive functions (amongst other disturbed cognitive functions) [7, 8]. Usually, these problems are by default attributed to neuropsychological deficits; however, lesions at the sensory level may also contribute to these aforementioned problems. In any case, visual deficits can heavily aggravate the complications affecting SAH survivors, especially when the patient and his or her relatives are not aware of these visual deficits. It is a well-known ophthalmological phenomenon that subjects with even severe peripheral visual defects often are not aware of their disability, providing that their central vision/visual acuity is spared [15].

In this study, we describe the long-term ocular manifestations of aneurysmal SAH in a series of patients in whom aneurysmal clipping was performed by one neurosurgeon. The aim of our study was to asses permanent lesions within the eyeball itself and/or within the visual pathways which were directly exposed to the hemorrhage and to surgical manipulations. Visual evoked potentials allowed us to identify the extent of the defects and their localization at a given level of the visual pathways (optic nerve, chiasm, optic tract, etc.), whereas visual field testing (perimetry) additionally revealed which fibers had been damaged at the horizontal cross section of the nerve. The incidence and possible mechanisms of visual pathway damage are discussed.

Patients and methods

Between 2006 and 2008, 79 patients with aneurismal SAH underwent surgical clipping at our institution. All of these procedures were performed by the same surgeon (Ze.M.). Sixty-four patients were discharged with a Glasgow Outcome Scale (GOS) score of 5 (~81%), 13 patients with GOS 4 (~16%) and two patients with GOS 3 (~3%). The examined sample consisted of 46 patients who responded to our invitation to take part in this study and fulfilled the inclusion criteria. Three patients were excluded from this follow-up because of data in the file indicating the presence of glaucoma.

Finally, the study group included 12 men (26.1%) and 34 women (73.9%). The median age of the patients was 50 years (range of 23–69 years, mean of 47.8±10.6 years). As indicated in Table 1, 83% of patients were GOS 5, 15% of patients GOS 4 and 2% of patients GOS 3. Thus, despite

a relatively high drop-out rate, the composition of the sample appears to correspond well with the general population consisting of all patients operated during this period.

The study was approved by the University Institutional Review Board (according to the guidelines of the Helsinki Declaration), and all patients gave written consent for the use of their clinical material in this publication.

The factors analyzed included: age at admission, sex, clinical status at the time of surgical intervention (according to Hunt and Hess, and Glasgow scales), location and size of the aneurysm, timing of surgery in days since the hemorrhage (the day of ictus recorded as day 0), grading of SAH according to the Fisher scale. Ruptured aneurysms were classified into three groups in terms of their site: (1) anterior communicating artery (ACoA), (2) internal carotid artery (ICA), and (3) middle cerebral artery (MCA). Precise localization of the nine ICA aneurysms is shown in Table 1. In our material there were no posterior circulation aneurysms because these lesions are almost exclusively treated with embolization at our institution. The outcome at discharge was categorized using the Glasgow Outcome Scale. These characteristics in all patients are gathered in Table 1. Precise localization of projection of the aneurysm is shown in Table 2.

Additionally, patients were examined with MRI which excluded hydrocephalus as well as any other pathologies that might be incidentally found, in all cases.

Ophthalmological examination

Each patient underwent ophthalmological examination of both eyes by an experienced ophthalmologist (I.O.). The mean interval between the onset of SAH and examination was 1.9±1.3 years (range 0.5-5 years). The comprehensive ophthalmic evaluation included: the recording of visual acuity and color perception, intraocular pressure measurement, slit-lamp examination of the anterior segment, lens and vitreous, direct and indirect ophthalmoscopy, investigation of the function of cranial nerves I-VII, visual field and visual evoked potentials testing. Cranial nerve function was determined based on: the position and motility of the eyelids and eyeballs (the 3rd, 4th, 6th and 7th nerves), status of pupils (the 3rd nerve), along with corneal and skin sensitivity (the 5th nerve).

Visual fields were assessed during one session of examination with automated perimetry (Medmont Model M700) using full threshold strategy. A standardized grid of 164 static targets within the central 50 degrees of the visual field was applied with decreased stimulus intensity in steps of 3 dB, until threshold sensitivity was reached. Reliability criteria were established along with recommended standards, i.e. less than 20% fixation losses, false-negative error and false-

Table 1 Clinical characteristics of the study group

Characteristic		Number of patients
Mean age in years ± SD		47.8±10.6
Gender: M/F		12/34 (26/74%)
Gender ratio (M:F)		1:2.8
Glasgow Coma Scale	14-15	39
	9-13	7
	≤8	0
Hunt and Hess grade	I	26
	II	14
	III	6
	IV	0
	V	0
Fisher scale	I	19
	II	11
	III	14
	IV	2
Time of surgery (days)	1-3	26
	4-10	15
	≥11	5
Glasgow Outcome Scale	5	38
	4	7
	3	1
	≤2	0
Size of aneurysm	<10 mm	32
	≥10 mm	14
Site of aneurysm	ACoA	17 (36.9%)
	ICA	9 (19.6%)
	- ophthalmic part	3
	- anterior choroidal part	2
	- communicating part	1
	- bifurcation	3
	MCA	20 (43.5%)

SD standard deviation, *M* male, *F* female, *ACoA* anterior communicating artery, *ICA* internal carotid artery, *MCA* middle carotid artery

positive error rates below 33% each. Moreover, appropriate correction for close vision was included. The patterns and binocular characteristics of the defects in the visual fields were evaluated. Visual field defects were classified according to their predominant pattern. They were categorized as follows: normal, constricted field (Figs. 1 and 2), superior field defect (Fig. 3), inferior field defect (Fig. 4), and multiple foci defect (Fig. 5). The visual field of patients with small superior visual field defects was classified as normal whenever a lid artifact was suspected. Definitions of the types of visual field defects are summarized in Table 3. For the purpose of statistical analyses, patients were categorized as those with an affected visual field (Field+) and those with a normal visual field (Field−).

Visual evoked potentials (VEPs) were recorded with the Vision Monitor Model Mon EL2 (Metrovision). All of the procedures applied to record VEPs were performed in accordance with the standards of the International Society for Clinical Electrophysiology of Vision [11]. Patterns of VEPs (PVEPs) were recorded monocularly with needle-electrodes. The active electrode was attached to the skull in the midline, 2 cm above the inion. The reference electrode was placed on the vertex and the ground electrode was placed on the forehead. Sixty responses were averaged. All of the VEP recordings were repeated twice. For the analysis of PVEPs, latency (ms) and amplitude (μV) values for the P2 wave were used. The obtained values of the PVEPs were assessed as abnormal when they did not fall between the ranges of normal reference values used in our laboratory. As in visual field defect, patients were subdivided into the group with normal VEPs (VEPs−) and abnormal VEPs (VEPs+).

Statistical analysis

The data were analyzed using statistical software Systat for Windows, (Systat, Evanston, IL). All of the continuous variables used were found to be normally distributed after testing with the Shapiro-Wilk's test. Chi-squared or Fisher's exact tests were used for testing the statistical hypotheses pertaining to the categorical variables. Pearson's correlation and *t*-test were calculated for continuous variables. Levels of probability were considered statistically significant when $p<0.05$.

Results

Ophthalmological examination

From among all of the patients examined, only one (2.2%) demonstrated decreased visual acuity (Visus=0.08). This patient was a woman who developed Terson's syndrome with massive hemorrhage into the vitreous body immediately after SAH. Fundoscopy revealed the presence of a macular scar in the affected eye. All the remaining patients had undisturbed visual acuity, normal intraocular pressure and no abnormalities were found in the anterior and posterior segments of the eye. In some older patients, both in those with intact and with defected visual field, slight temporal pallor of the optic nerve head was seen. This appearance was judged to be rather within normal range according to their age, the more so the both optic discs were of similar look.

A wide, unreactive pupil (dysfunction of the right oculomotor nerve) was found in one patient who underwent clipping of an aneurysm pointing infratentorially and

Table 2 Projections of aneurysms

Projection	Number of patients				
	Total	Field(+)	Field(−)	VEPs(+)	VEPs(−)
ACoA:					
forward + upward	10	4	6	2	8
downward	7	3	4	1	6
MCA:					
to frontal lobe	9	5	4	3	6
to temporal lobe	5	3	2	1	4
to the Sylvian fissure	6	2	4	1	5
ICA – ophthalmic part:					
backward	3	3	0	1	2
ICA – posterior communicating part:					
backward	1	0	1	0	1
ICA – anterior choroidal part:					
embedded in brain	2	1	1	0	2
ICA – bifurcation:					
embedded in brain	3	2	1	0	3

Field(+) visual field defects, *Field(−)* no visual field defects, *VEPs(+)* abnormal visual evoked potentials, *VEPs(−)* normal visual evoked potentials

situated at the posterior communicating artery (PCoA) segment of the right internal carotid artery.

Visual field

Visual field defects were found in 23 patients (50%). In all of these patients, both eyes were affected. Five patients had different patterns of visual field defects in the right and the left eye. The typical patterns of visual field defects are shown in Figs. 1, 2, 3, 4 and 5. The most frequent type of defect was that of a constricted field, which was found in 47.8% of cases. The next two common patterns of visual field loss were: multiple peripheral foci (26.1%) and

superior field defect (17.4%). An inferior field defect was found in 8.7% of cases.

In eight patients (17.4%), the defect involved about 50% of the visual field and therefore could be defined as severe. Noticeably, these severe deficits affected only one eye and occurred rather in older patients: the mean age of this subgroup amounted to 53.4±8.1 years, whereas the mean age of the whole group was 47.8±10.6 years. Interestingly, these patients were usually unaware of their incapacity as none of them had sought ophthalmological assistance before they were invited to this examination.

Table 4 presents the patterns of visual field defects found in patients with different localizations of the aneurysm. It is
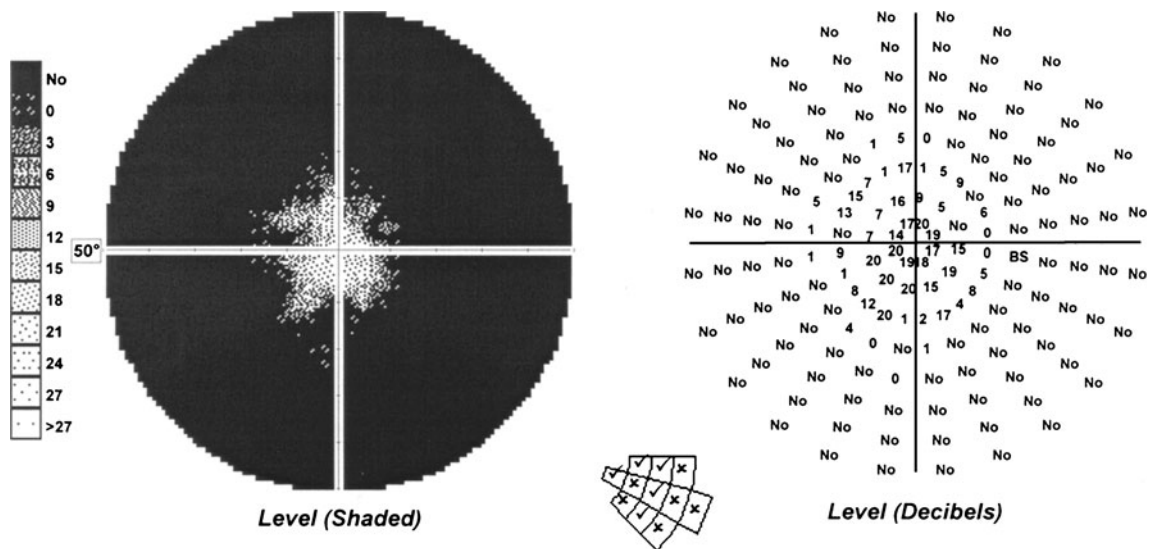

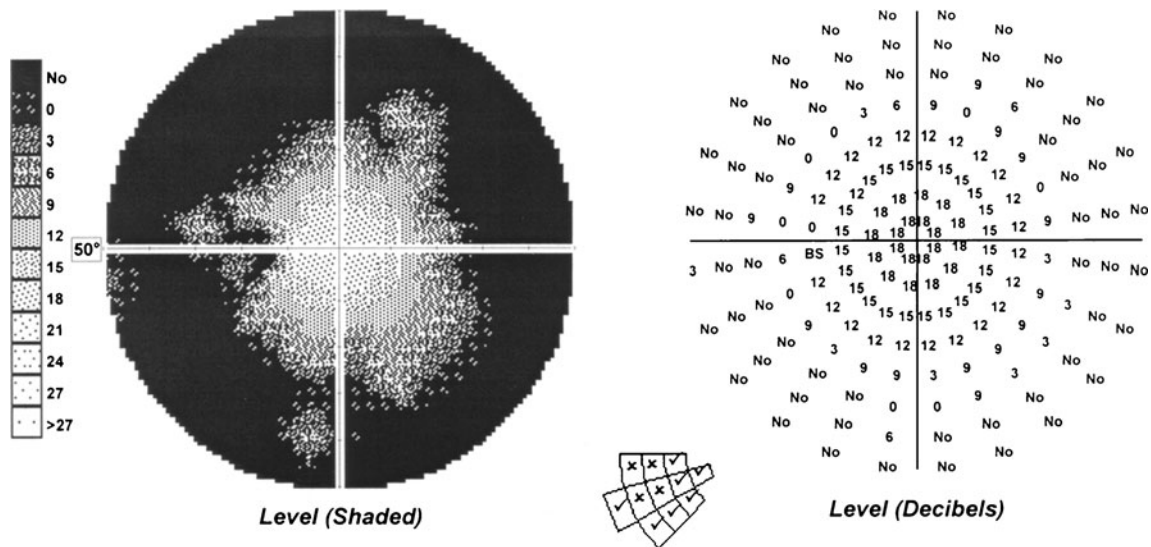**Fig. 1** Constricted field with only central island field present

Fig. 2 Constricted field

of interest to note that the incidence of abnormal visual fields was lowest in the group of patients with ACoA aneurysms and greatest in the group of patients with aneurysm of the ICA, although this difference was not statistically significant.

Visual field deficits were found in six out of nine patients with ICA aneurysms. It could be of interest to point out that such defects were present in all three patients with aneurysms located in the ophthalmic segment of the ICA, although they were not more pronounced than deficits in patients with other aneurysms. In one subject the visual field defect was significant and was accompanied by VEP disturbances.

The relationships between other analyzed variables and the occurrence of visual field deficits are shown in Table 5.

From amongst the different demographic and clinical factors, only the severity of the SAH (expressed as the Fisher scale scored) was related to the appearance of visual field deficits.

Visual evoked potentials

Moderate to severe deteriorations in PVEPs in one or both eyes were observed in nine patients (19.6%). In four patients, both a reduction in amplitudes and an increase in latency of P2 waves were seen in both eyes. A reduction in only the amplitude was observed in both eyes of three patients and in one eye of two patients. VEP responses recorded bilaterally from the visual cortex showed inter-hemispheric symmetry during stimulation of each eye—a
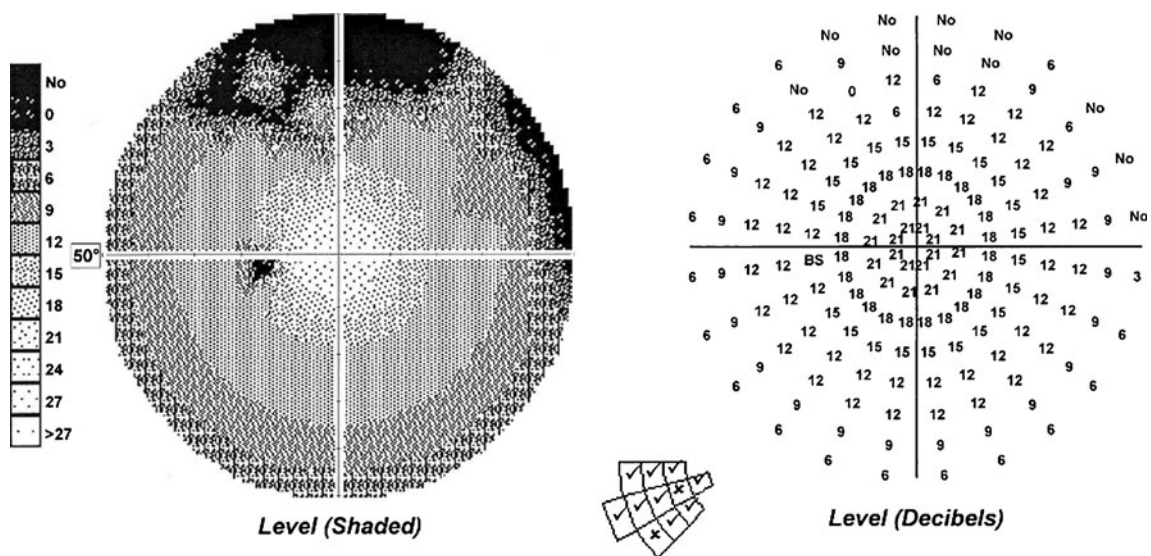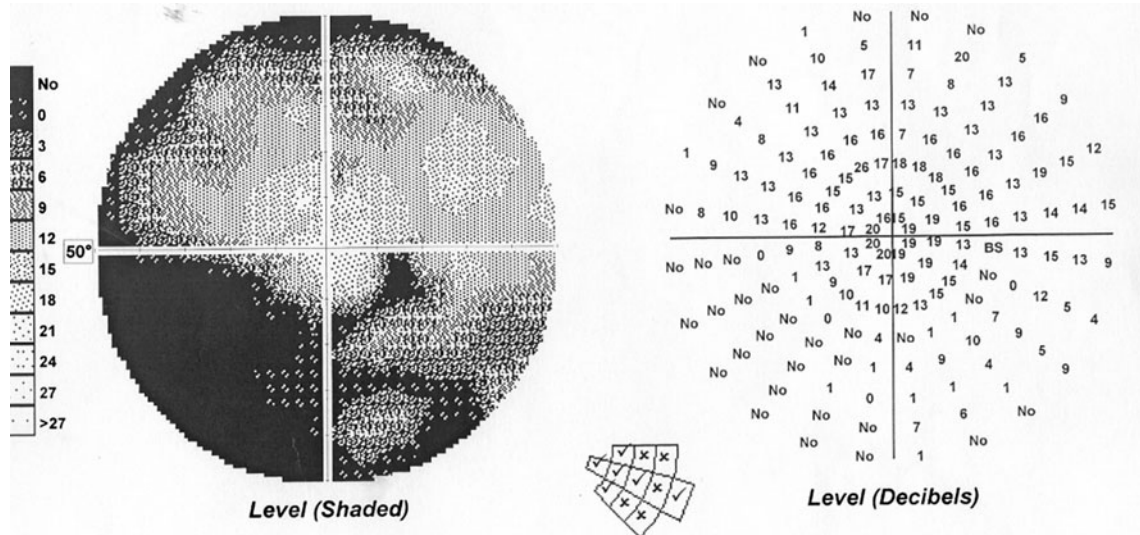

Fig. 3 Superior field defect

Fig. 4 Inferior field defect

finding which suggests a prechiasmal localization of the defect. It is worth noticing that all patients with abnormal VEPs also had vast defects in their visual fields, whereas up to 14 patients with visual fields defects did not show any abnormalities in VEPs.

Abnormal VEPs were found in three out of 17 patients with an aneurysm of the ACoA, in four out of 20 patients with an aneurysm of the MCA, and in two out of nine patients with an aneurysm of the ICA.

The relationship between other analyzed variables and the occurrence of VEP abnormalities is shown in Table 6. From amongst the many factors of interest, two appeared to relate to the incidence of VEP abnormalities: the severity of SAH (according to the Fisher scale) and the timing of surgical intervention.

Discussion

Our study has demonstrated that as many as 50% of patients who experienced a SAH and underwent aneurysm clipping suffer from varying degrees of visual pathway impairments, with the main level of damage localized to the optic nerve and chiasm. Furthermore, 35% of our patients (17% of the entire group) had severe defects in their visual field, and/or abnormal patterns of VEP. Often, our patients were unaware of their visual incapacity.

Except for a patient who suffered from Terson's hemorrhage at the onset of SAH, no other patient showed any damage to the optic structures within the eyeball, and the visual acuity of the whole group was within the normal range. Severely defected visual field with the relatively
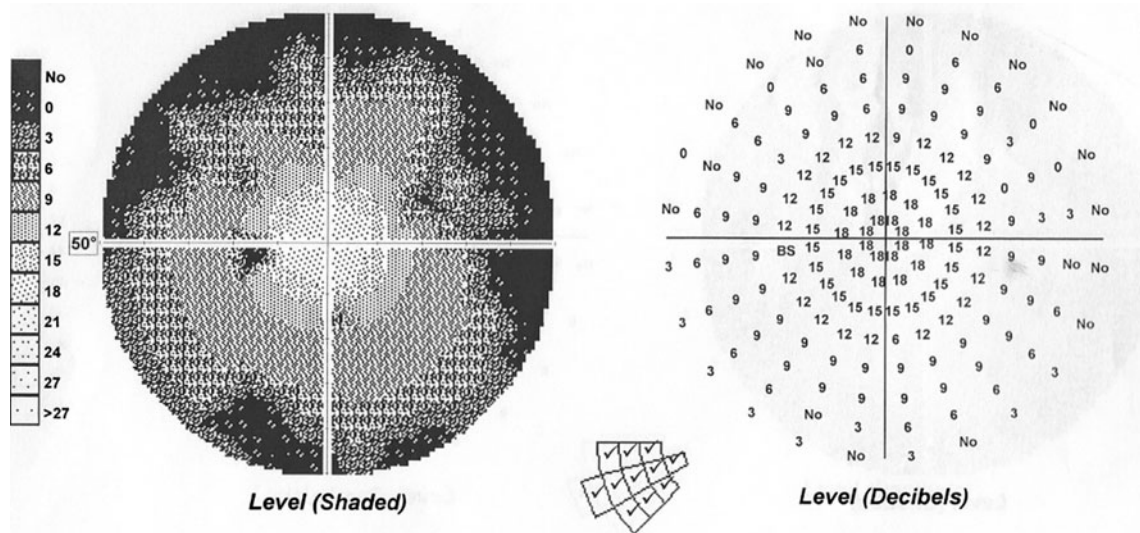

Fig. 5 Multiple foci defect

Table 3 Patterns of visual field defects

Type pattern	Definition
Normal	None or only a few depressed points in no specific pattern
Constricted field	Generalized constriction of the entire isopter
Superior field defect	Peripheral defect in one or both superior quadrants
Inferior field defect	Peripheral defect in one or both inferior quadrants
Multiple foci defect	Multiple peripheral defect that may appears in each part of field

normal optic head and preserved visual acuity can appear contradictory. We can explain this by the peripheral nature of these deficits, which leave a relatively vast central region of the retina intact. Preserved macular nerve fibers are overrepresented in the optic nerve, thus ensuring good visual acuity and relatively normal appearance of the optic disc. Discrete temporal paling seen in some older patients with normal and abnormal perimetry were considered as remaining within normal range for their age because the appearance of the optic discs was similar in both eyes, whereas the visual field defects were highly asymmetrical.

All the disorders demonstrated by perimetry and VEP rather appeared to correspond with damage located at the optic nerves and/or chiasm. This notion is supported by the finding that in virtually all affected patients, the pattern of visual field defects was different in both eyes. If the damage had been located beyond the chiasm, a homonymous symmetric defect would have been expected. Nevertheless, such a homonymous hemianoptic pattern might be difficult to pinpoint because it may be overlapped by deficits generated by disseminated damage to the two optic nerves and chiasm. Thus, in contrast to the optic radiation and the visual cortex, partial damage to the retrochiasmatic part of the visual pathway cannot be categorically excluded.

The most conspicuous feature in all patients with visual field defects was the peripheral location of scotomas. The most affected patients (four persons), showed a severely constricted field with merely a central island of 10–15 degrees preserved. Such "lunette" vision usually ensures normal visual acuity and this can explain why these patients did not seek any ophthalmological assistance. Patients with less pronounced damage to the visual pathways showed disseminated visual field defects, which were invariably also located peripherally. These scotomas usually appeared U-shaped or trigonal, typical for ischemic foci. In all likelihood, these defects can be ascribed to damage of the tiny arterial vessels running along the subpial space of the optic nerve and their minute feeders running from the carotid stem and its branches to the nerve within the subarachnoid space of the chiasmatic cisterns [16].

As for potential factors which could have produced these vascular abnormalities, it is obvious that the damage could have originated either from surgical manipulation around the nerve or from interactions between the vessels and the extravasated blood. Primary damage to the visual field can only be produced by large aneurysms that compress the nerve directly. None of the aneurysms observed in this location appeared to compress the nerve or was of a substantial size. Direct compression of the optic chiasm by an ACoA aneurysm was not seen in any of our patients and is generally much less likely in spite of close anatomical proximity of both structures. ACoA aneurysm rarely increases in size before rupture and only large and downward directed aneurysms can expand to the chiasm. Although aneurysm can fuse tightly with the chiasm, actual compression occurs rarely because both structures are not bordered by any firm anatomical structures to produce an anatomical trap (unlike the carotid-ophthalmic aneurysms).

As demonstrated in Table 4, visual field defects were more frequent amongst patients with an aneurysm located in the ICA and MCA, and less frequent when the aneurysms were located in the ACoA. The ACoA lies

Table 4 Visual field defects and localization of aneurysm

Visual field status	Number of patients			
	Total ($n=46$)	ACoA ($n=17$)	MCA ($n=20$)	ICA ($n=9$)
Normal	23	10	10	3
Abnormal:	23	7	10	6
constricted field	11	2	6	3
superior field defect	4	2	1	1
inferior field defect	2	1	1	0
multiple peripheral foci	6	2	2	2
Percentage of abnormal fields	50%	42%	50%	67%

Table 5 Clinical characteristics of patients with visual field defects (*Field+*) and with no defects (*Field–*)

Characteristic	Field+	Field–	p
Mean age (years)	51.2	47.9	0.43
Gender: M/F (M:F ratio)	7/16 (1:2.9)	5/18 (1:3.6)	0.73
Time of surgery (days from SAH)	5.9	7.9	0.52
Fisher scale	2.44 ± 1.0	1.88±0.9	0.055
GCS	14.3	14.5	0.98
Hunt-Hess scale	1.5	1.4	0.51
Size of aneurysm (mm)	8.2	7.0	0.44
GOS	4.8	4.7	0.63

GCS Glasgow Coma Scale, *GOS* Glasgow Outcome Scale

(and often bleeds) above the optic complex, whereas the minute optic feeders approach the nerves and chiasm from below, through the chiasmatic cistern, communicating laterally with the Sylvian cistern. This suggests that the damage to the nerve arose due to direct contact with extravasated blood. This notion is supported by the finding that the occurrence of visual field defects and/or disturbances in VEPs coexisted in a statistically significant manner with Fisher scale scores, i.e., the extent of the hemorrhage (Tables 5 and 6). Direct severance during the hemorrhage and/or delayed vasospasm of these tiny [19], fragile vessels seem to have been responsible for the permanent damage of the optic nerves observed.

Table 5 shows that the average time from the onset of SAH to surgical intervention was shorter in patients with abnormalities of visual fields than in those who were not affected. This difference was not statistically significant. However, we must emphasize that this difference pertained to the entire group of 23 patients, of whom as many as 15 had minor to moderate field defects. In reference to the subgroup of severely damaged patients, persons with significant visual field defects and disturbed VEPs (Table 5, VEP+) appeared to have undergone aneurysm clipping significantly sooner after SAH than those with normal VEPs (2.6±1.8 days vs 6.4±2.4 days, respectively).

This finding potentially adds a new dimension to the other more recognized sequelae of early surgical intervention in

patients with aneurysmal SAH. It is quite obvious that surgical procedures during the early period after SAH may be a source of additional harm to the cerebral structures (including the optic nerves). As a matter of fact, except for large ophthalmic and downward directed ACoA aneurysms, there is usually little need to manipulate the optic nerves and chiasm. Nevertheless, the conventional surgical procedure includes disconnection of the frontal lobe from the optic nerves and chiasm. This maneuver, along with wide separation of the Sylvian fissure, enables "mobilization" of the frontal lobe so that minimal pressure is exerted by spatula onto the cerebral structures. Though performed extremely carefully and under microsurgical magnification, these manipulations may potentially injure the surface of the nerve and/or the fine vasculature of the optic structures.

In summary, our study indicates that a considerable proportion of patients with aneurysmal SAH have defects in their visual field and/or abnormal VEPs. The pattern of these abnormalities corresponds to damage of the anterior segment of the visual pathways, namely the optic nerves and/or optic chiasm. It is worth noting that while defects were detected in both eyes, they were not symmetrical, and that severely constricted fields of vision usually concerned one eye. The peripheral localization and typical U-shaped or trapezoid appearance of these defects suggests a vascular origin. This notion is strengthened by the fact that the severity of optic nerves damage corresponded with factors

Table 6 Clinical characteristics of the patients with abnormal (*VEPs+*) and normal (*VEPs–*) visual evoked potentials

Characteristic	VEPs+	VEPs–	p
Mean age (years)	51.4	49.1	0.46
Gender: M/F (ratio M:F)	2/7 (1:3.5)	10/27 (1:2.7)	0.99
Time of surgery (days from SAH)	2.6±1.8	6.4±2.4	0.02
Fisher scale	2.8±0.6	2.0±0.4	0.04
GCS	13.9	14.5	0.21
Hunt-Hess scale	1.7	1.4	0.35
Size of aneurysm (mm)	7.9	8.8	0.73
GOS	5.0	4.8	0.73

known to provoke vasospasm: the massiveness of the SAH and surgical manipulations [19].

Among the limitation of this study is the relatively small size of the sample. Therefore, some noticeable trends could not be demonstrated as being statistically significant, such as the trend suggesting a lower incidence of optic pathway damage in patients with aneurysms of the ACoA. Also, our sample does not represent the whole scale of SAHs and all localizations of aneurysm. No posterior circulation aneurysms were included because they are almost invariably embolized at our institution. At the same time, our clinical material is homogenous because the study group contains mainly patients with a good clinical state, i.e., patients with Hunt and Hess grade I or II, and only six patients with Hunt and Hess grade III. This was due to the fact that in our institution patients with higher Hunt and Hess scores and with suitable aneurysm anatomy are more readily treated with endovascular embolization, including those with Hunt and Hess grade III. Nonetheless, in this homogenous group of patients with a relatively good clinical grade and with a good prognosis the incidence of optic nerve damage appeared to be surprisingly high. One can expect that in patients affected more severely by SAH, the incidence of permanent visual pathways defects is at least similar or even higher.

Another apparent limitation might pertain to the fact that virtually no data on ophthalmic status of our patients before the SAH were available to us. This may raise a question whether all observed deficits were the result of SAH and its consequences or were some of them present previously. To address this question, patients with ophthalmological problems, especially glaucoma, were excluded from the study on the basis of their medical history and ophthalmological examination. Three patients were excluded based on these criteria and therefore the examined sample did not contain any subjects with prior ophthalmological conditions.

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Comment

Patients with SAH have more visual problems than we may recognize as other neurological sequelae may steal the attention initially. But, after the initial recovery, visual problems may disturb daily activities and this has not been well studied in detail in the long run. The current paper shows that visual field defects are quite common after SAH and clipping of aneurysms, but the effects of each can not be separated. We do not know the visual state well enough before the ictus. The paper also addresses the importance of gentle dissection under high

magnification of the operating microscope in slack brain using modern neuroanesthesia, preferably without the use of retractors to avoid iatrogenic brain and nerve damage.

There are biases concerning the examination of the patients affecting the results. In cases of constricted visual fields and normal appearing optic nerve heads, you should take into consideration the huge effect of learning: in some patients the first visual field examination is worse than the later ones and these results should be evaluated carefully. Even though you have a commonly accepted automated perimeter and the examination is performed according to a strict standard methodology, many patients cannot cope with the automated static perimetry as it may be too exhaustive for them, with negative effects on the results. In patients with severe visual field defects, kinetic perimetry by a skilled perimetrist should be performed.

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