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Late ophthalmological manifestations in patients with subarachnoid hemorrhage and coiling of cerebral aneurysm

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ABSTRACT

Late ocular manifestations of aneurysmal subarachnoid hemorrhage (SAH) have not been previously investigated except for one study which demonstrated that one half of patients subjected to aneurysm clipping suffer from symptoms of visual pathway impairment. We assessed ophthalmological status of patients after 1–4.5 years from SAH and aneurysm embolization to identify predictors of damage to the visual pathways. Complete ophthalmological examination, static perimetry, and visual evoked potentials (VEPs) were performed in 74 patients (26 men, 48 women, aged 19–76 years), who constituted a consecutive sample of 129 patients treated with aneurysm embolization in the years 2008–2010. The following independent variables: sex, age, time from SAH to embolization, size and site of aneurysm, score in Glasgow Coma Scale, Glasgow Outcome Scale, Hunt-Hess and Fisher scales were subject to univariate and multivariate statistical analyses to study their influence on the ocular outcome. 40 patients (54%) demonstrated visual field defects appearing as multiple peripheral foci and constricted field, affecting both eyes. Among these subjects, 12 patients had severe defects in the visual field, 20 had deterioration in VEPs, and 9 had decreased visual acuity. Older age and high score in Hunt-Hess and Fisher scales were identified as predictors for visual field defects and disturbances in VEPs. More than half of the survivors of SAH and aneurysm embolization suffer from a permanent defect in visual function. Damage of visual pathway correlates with severity of SAH and older age of patients.

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1. Introduction

Both common knowledge and results of numerous well documented clinical studies show that spontaneous subarachnoid hemorrhage (SAH) from cerebral aneurysm has a disproportionally high impact on psychosocial outcome of its survivors and their health-related quality of life (HRQOL) [1–4]. The problem is of particular importance, because SAH affects much younger persons than other forms of stroke.

In spite of hitherto vast research on SAH outcome, it is still unclear which particular factors predict the loss of psychosocial performance, as such potential predictors as age, sex, bleeding severity, cognitive impairment, etc., were found to have relatively small effect on mental and physical HRQOL [1,4]. On the other hand, possible lesions at the sensory level have not been studied thus far with regards to their influence on the condition of SAH survivors. Moreover, even the mere incidence of sensory damage in this population has not been conclusively defined or studied.

Recently, we described long-term ocular manifestations of aneurysmal SAH in a series of patients in whom aneurysmal clipping was performed by one neurosurgeon [5]. This study demonstrated that as many as 50% of these patients suffer from varying degrees of visual pathway impairment, with main level of damage located at the optic nerves and/or chiasm. Importantly, most of them, including patients with severe visual field defects, were unaware of their disability.

Naturally, a question arises whether the damage to the visual pathway is an effect of SAH itself or is produced during surgical manipulation during the clipping of the aneurysm. To address this problem we extended our study into a group of patients with SAH in whom aneurysm was secured by intravascular coiling. A conventional ophthalmological examination, visual evoked potentials, and static perimetry were performed in a period of 1–4.5 years after the onset of SAH.

2. Materials and methods

Between 2012 and 2014, one hundred twenty nine patients with aneurysmal SAH underwent aneurysm embolization at our institution. Among these patients, 9 died or were in vegetative state after SAH and embolization, while another 11 were excluded from the study because they underwent combined endovascular/surgical procedures for multiple aneurysms. All the remaining 109 patients discharged within the abovementioned period as independent were invited to take part in the study; however, only 92 patients responded to the invitation. 12 patients were excluded from this primary sample because of the presence of glaucoma, cataract, macular degeneration, or diabetic retinopathy as diagnosed from data on file and based on the result of current ophthamological examination. Another 6 patients had problems with cooperating during examination of the visual field and were also excluded because their results did not fulfill the criteria of reliability.

Ultimately, the study group included 74 patients: 26 males (35%) and 48 females (65%). The median age of the patients was 52 years (range of 19–76 years, mean 49.6 ± 12.3 years).

The study was approved by the Institutional Review Board, and all patients gave written consent for the use of their clinical material in this publication.

2.1. *Embolization procedure*

Endovascular procedures were performed using biplane Siemens Artis angiographic unit under general anesthesia. Aneurysms were coiled with detachable Guglielmi coils (GDC, Boston Scietific, Microvention), which were platinum coils with a volume-expanding hydrogel coating (Microvention). While most aneurysms were secured with electrolytically detachable coils, some were coiled with mechanically detachable coils (MDS-Balt); detailed indication for a given technique depended on aneurysm size and shape. In a few wide neck aneurysms, balloon remodeling was used as a supporting technique (Balt).

A follow up angiography was scheduled at 6 months and 1 year after endovascular coiling. Complete or near-complete (i.e. satisfactory) occlusion was stated in 86.5% of all aneurysms (64 patients), as classified by using the modified Raymond classification scale [6]. 10 patients needed re-embolization, in 2 of them repeated more than once.

2.2. *Independent factors*

The analyzed factors included: age at admission, sex, clinical status at attempted obliteration of aneurysm (according to Hunt-Hess and Glasgow Coma scales), timing of aneurysm obliteration in days since the hemorrhage (the day of ictus recorded as day 0), and grading of SAH according to the Fisher scale. The size of the aneurysms was dichotomized into <10 mm and >10 mm, whereas their location was classified into four groups: (1) anterior communicating artery (ACoA), (2) internal carotid artery (ICA), (3) middle cerebral artery (MCA), and (4) basilar/vertebral artery (BVA). The outcome at discharge was categorized using Glasgow Outcome Scale. All patients were examined with MRI and/or CT to check for hydrocephalus and/or for other intracranial pathologies that might influence their ophthalmological status. The relevant results of tests performed for all patients and size/localization of the aneurysms are presented in Table 1.

2.3. *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 1.8 ± 1.2 years (range 1.0–3.0 years). The comprehensive ophthalmic evaluation included: 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, and visual field and visual evoked potentials testing. Cranial nerve function was determined based on the position and motility of the eyelids and eyeballs (3rd, 4th, 6th, and 7th cranial nerves), status of the pupils (3rd cranial nerve), along with corneal and skin sensitivity (5th cranial nerve).

Table 1 – Clinical characteristics of the study group.

Parameter		n
Mean age in years ± SD		49.6 ± 12.3
Gender: M/F		26/48 (35/65%)
Gender ratio (M:F)		1:1.85
Glasgow Coma Scale	14–15	65
	9–13	6
	≤8	3
Hunt and Hess grade	I	51
	II	17
	III	5
	IV	1
	V	0
Fisher scale	I	9
	II	38
	III	13
	IV	14
Time of embolization from SAH (days)	1–3	59
	4–10	14
	≥11	1
Glasgow Outcome Scale	5	65
	4	9
	3	0
	≤2	0
Size of aneurysm	<10 mm	51
	≥10 mm	23
Site of aneurysm	Anterior communicating artery	33 (44.6%)
	Internal carotid artery:	20 (27%)
	- Ophthalmic part	2
	- Anterior choroidal part	8
	- Communicating part	6
	- Bifurcation	4
	Middle cerebral artery	10 (13.5%)
	Basilar/vertebral artery	11 (14.9%)

SD – standard deviation; M/F – male/female; SAH – subarachnoid hemorrhage.

Visual fields were assessed twice with automated perimetry (Medmont Model M700) during two session of examination using full threshold strategy. The second results were analyzed in this study to avoid the effect of learning. 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-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 and were as follows: normal field, constricted field (generalized constriction of the entire isopter, Fig. 1), and multiple peripheral foci defects (multiple peripheral defect that may appears in each part of field, Fig. 2). The visual field of patients with small symmetrical superior visual field defects was classified as normal whenever a lid artifact was suspected. For the purpose of statistical analyses, patients were categorized as those with an affected visual field (Field+) and those with 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 [7]. 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 sub-divided into a group with normal VEPs (VEPs−) and abnormal VEPs (VEPs+).

2.4. *Statistical analyses*

Statistical analyses were performed with Systat® software for Windows (Systat, Evanston, IL). All of the used continuous variables were found to be normally distributed after testing with the Shapiro–Wilk's test. To examine associations between clinical variables and the ophthalmological outcome, univariate analyses were performed using Person's correlation, Mann–Whitney *U* test for continuous variables, or χ^2 test for categorical variables. Binary logistic regression analysis was then used to dichotomize outcome as a variable dependent on variables found to be significant on the univariate testing. The odds ratios (ORs) were calculated with a 95% confidence interval (CI). The level of significance was set at $p < 0.05$.

3. Results

In general, ophthalmological examination revealed three types of functional disturbances: visual acuity decrease, visual field defects, and VEPs abnormality reflecting impaired conductivity of the optic nerve.

3.1. *Visual acuity*

Decreased visual acuity (VA) in one eye (5 persons) or both eyes (4 persons) was found in 9 patients (12.2% of the whole group). The best corrected visual acuity in these patients ranged from 0.2 to 0.8. The remaining 87.8% of patients had undisturbed visual acuity, normal intraocular pressure, and no abnormalities were found in the anterior segments of their eyes. Temporal pallor of the optic disc in one or both eyes were observed in 3 patients with normal VA.

Among the patients with decreased VA, two developed Terson's syndrome immediately after SAH with massive hemorrhage into the vitreous body. Fundoscopy revealed the presence of vitreous opacity and temporal pallor of the optic disc in the affected eyes. Another five patients presented with decreased visual acuity and slight temporal pallor of the optic nerve head seen with fundoscopy – usually more evident in one eye. The remaining two patients in this group had only

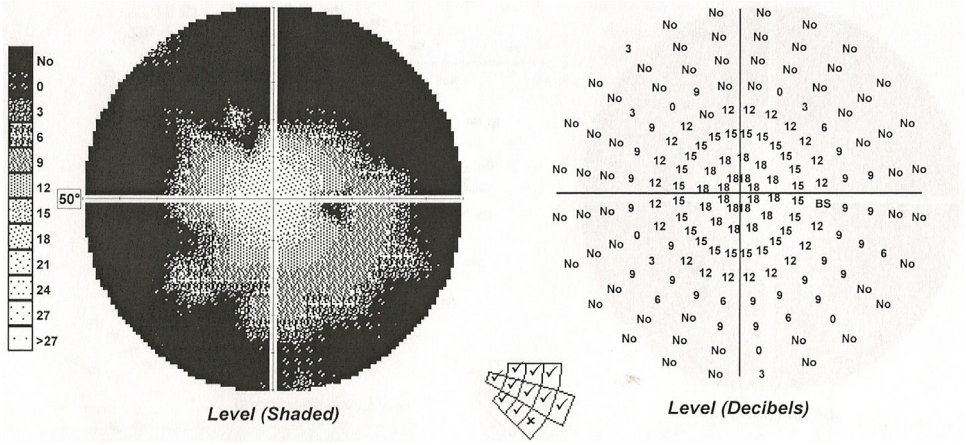

Fig. 1 – Right-sided visual field defect in a 48-year-old woman who underwent coiling of an AcoA aneurysm. Automated perimetry revealed an extensive peripheral constricted field.

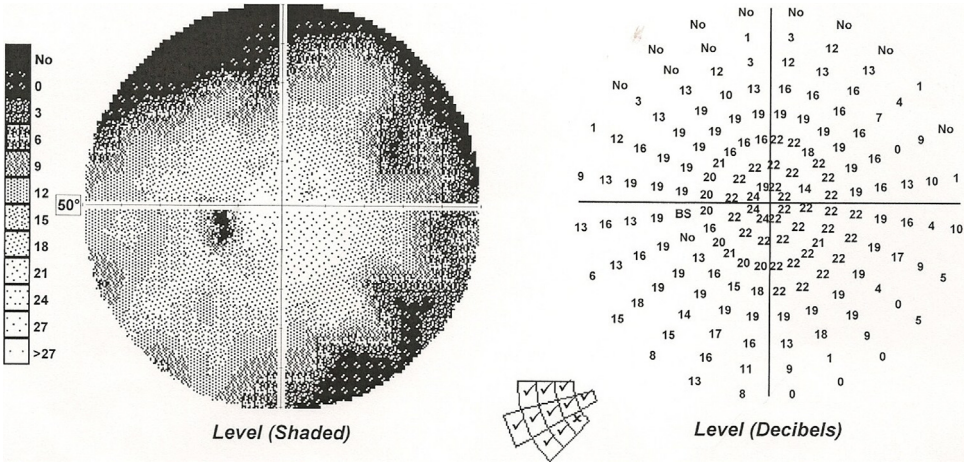

Fig. 2 – Left-sided visual field defect in a 52-year-old man who underwent coiling of an MCA aneurysm. Automated perimetry revealed peripheral multiple foci defect.

slight vision impairment in one eye (0.7–0.8), without abnormalities in the anterior and posterior segments of the eye.

Clinical characteristics of the patients with affected visual acuity were as follows: there were 8 women and 1 man, mean age 56.3 years, Fisher scale on average 3.0, average GCS 13.3, average score in Hunt-Hess scale – 1.6. Five patients had aneurysm of the ICA, 3 – of the AcoA, and 1 of the MCA. Decreased visual acuity was accompanied by visual field defects in all affected eyes (6 patients had constricted field and 3 patients were revealed to possess multiple peripheral foci defects) and abnormal VEPs in 6 affected eyes. The patients with reduced visual acuity were generally older than the rest of the group, whereas other clinical parameters were similar to those in patients with unaffected vision.

Importantly, 9 patients had hydrocephalus diagnosed after SAH and aneurysm embolization. Ophthalmological examination revealed that three of the previously mentioned subjects had disturbed VA with slight temporal pallor of the optic nerve head; no abnormalities in the anterior and posterior segments of their eyes were found in the remaining 6 participants. Virtually all patients with hydrocephalus had visual field defects and five of them had also abnormal VEPs.

Ophthalmological examination in the group of 10 patients who needed re-embolization revealed that 50% of this group had visual field defects and 1 of them had also decreased visual acuity and abnormal VEPs.

3.2. *Visual field*

Visual field defects were found in 40 patients (54%), in all of them both eyes were affected. Generally, the form of the defects could be dichotomized into multiple peripheral foci type, which was found in 60% of the affected patients (Fig. 1), and constricted visual field type, detected in the remaining 40% of patients (Fig. 2). In 12 patients (constituting 16.2% of the whole group and 30.0% of patients with affected visual field), the defect involved at least 50% of the visual field and could, therefore, be defined as severe.

Table 2 presents the patterns of visual field defects found in patients with different localizations of aneurysm. It should be noted that the incidence of visual field disturbances was lower

Table 2 – Visual field defects and localization of aneurysm.

Visual field status	Number of patients				
	Total ($n = 74$)	ACoA ($n = 33$)	MCA ($n = 10$)	ICA ($n = 20$)	BVA ($n = 11$)
Normal	34	14	5	8	7
Abnormal	40	19	5	12	4
Constricted field	16	5	3	7	1
Multiple peripheral foci	24	14	2	5	3
Percentage of abnormal fields	54%	58%	50%	60%	36%

ACoA – anterior communicating artery; MCA – middle cerebral artery; ICA – internal carotid artery; BVA – basilar/vertebral artery.

in the group of patients with aneurysms located in the posterior circulation than in those with supratentorial aneurysms; however, this difference did not reach the level of statistical significance on χ^2 test.

The relationships between variables analyzed in this study and the incidence of visual field deficit are shown in Table 3. The following factors appeared to influence the visual deficit: age, clinical status of the patient at admission according to Hunt-Hess scale, and severity of SAH.

3.3. *Visual evoked potentials*

Moderate to severe deterioration of VEPs in one or both eyes were observed in twenty patients (27%). VEP responses recorded bilaterally from the visual cortex showed interhemispheric symmetry during stimulation of each eye. Virtually all patients with abnormal VEPs had also defects in their visual fields.

Abnormal VEPs were found in 8 out of 33 patients with aneurysm of the ACoA (24%), in 2 out of 10 patients with aneurysm of the MCA (20%), in 7 out of 20 patients with aneurysm of the ICA (35%), and in 3 out of 11 patients with aneurysm of BVA (27%). In a contrast to visual field defects, disturbances in VEP appeared to affect both patients with supratentorial and infratentorial localization of aneurysm with a similar frequency. The relationship between analyzed variables and the occurrence of VEP abnormalities is shown in Table 4. The results appeared similar to those found in visual field deficits with the exception of Fisher scale, which did not reach the threshold of statistical significance as a variable.

Table 3 – Clinical characteristics of patients with visual field defects (Field+) and with no defects (Field−).

Characteristic	Field+	Field−	p
Mean age (years)	53.7	44.9	0.02*
Gender: M/F (M:F ratio)	12/28 (1:2.33)	14/20 (1:1.4)	0.73
Time of embolization (days from SAH)	3.0	2.3	0.32
Fisher scale	2.8	2.0	0.01*
GCS	13.6	15.0	0.45
Hunt-Hess scale	1.7	1.1	0.01*
Size of aneurysm (mm)	8.0	7.5	0.41
GOS	4.8	5.0	0.07

M/F – male/female; SAH – subarachnoid hemorrhage; GCS – Glasgow Coma Scale; GOS – Glasgow Outcome Scale.
* Statistically significant.

Table 4 – Clinical characteristics of the patients with abnormal (VEPs+) and normal (VEPs−) visual evoked potentials.

Characteristic	VEPs+	VEPs−	p
Mean age (years)	56.4	47.1	0.003*
Gender: M/F (ratio M:F)	5/15 (1:3.0)	22/32 (1:1.45)	0.25
Time of embolization (days from SAH)	3.4	2.4	0.20
Fisher scale	2.7	2.3	0.07
GCS	12.9	14.6	0.49
Hunt-Hess scale	1.9	1.2	0.04*
Size of aneurysm (mm)	7.9	8.8	0.52
GOS	4.6	5.0	0.08

M/F – male/female; SAH – subarachnoid hemorrhage; GCS – Glasgow Coma Scale; GOS – Glasgow Outcome Scale.
* Statistically significant.

Table 5 – Binary logistic regression analysis of factors potentially predicting ophthalmological outcome after SAH and embolization of aneurysm.

Variable	Visual field defect		Abnormal visual evoked potentials	
	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
Mean age	2.80 (0.98–7.87)	0.05	6.65 (0.81–54.36)	0.03
Fisher scale	2.76 (1.47–5.16)	<0.01	1.64 (0.94–2.86)	0.08
Hunt-Hess scale	5.57 (1.81–17.14)	<0.01	4.37 (1.82–10.47)	<0.01

3.4. *Risk factors for damage of the visual pathways after SAH and aneurysm embolization*

To identify the factors reliably correlating with the visual field and VEP disturbances, a two-step statistical analysis was performed. The first step was univariate analysis aimed at identifying variables potentially influencing the outcome. The second step included multivariate analysis of these variables to find positive predictors for damage to the visual pathways.

Table 5 summarizes independent predictors of outcome as identified with binary logistic regression analysis. High score in Hunt-Hess and Fisher scales and older age revealed to be independent predictors for visual field deficit. The same factors predicted disturbances in VEPs with some exception of the Fisher scale, which did not reach statistical significance.

4. Discussion

Our study has demonstrated that more than half of patients who experienced SAH and underwent aneurysm embolization suffer from varying degrees of visual pathway impairments. This figure appeared to be even a little higher than the one we once reported in patients who had been treated by aneurysm clipping (54% versus 50%, respectively) [5]. All the affected patients demonstrated visual field deficits of different pattern

and severity whereas defects involving at least 50% of the field (i.e. severe defects), occurred in 30% of the subjects. It is of interest to add that all patients who had severe defect of the visual field demonstrated also disturbances in VEPs.

An even more striking difference between the groups of patients with clipped and coiled aneurysm pertains to visual acuity –2.2% versus 12.2% of patients with decreased VA, respectively. Moreover, also abnormal VEPs were observed more often in patients with aneurysm embolization than in those with aneurysm clipping (27% vs. 19.6% respectively). It can be admitted that apart from the discussed above severe visual field deficit, also decreased vision and impaired conductivity of the optic nerve testify to ''considerable'' or ''functionally significant'' degrees of visual pathway damage. As many as 22 patients (close to 30% of the entire group) appeared to fulfill such working criterion of significant damage to the visual pathway.

The rate of ophthalmological complications after SAH and aneurysm embolization (which at least matches the rate seen after surgical clipping) allows to speculate on the possible character and mechanism of such damage and thus to address the question set in the introduction. As to the character of the damage, we argued in our earlier paper [5] that the pattern of visual deteriorations seen in this group of patients corresponds to damage of the visual pathways and, more specifically, the optic nerves and chiasm. Such notion is supported by peripheral, bilaterally asymmetrical, and generally constricting character of these visual field defects. Lesion located at the retrochiasmatic level of the optic pathways is less probable as no symmetric homonymous defect seemed to emerge in any of the patients. Decreased visual acuity and/or abnormal VEPs seen in nearly one third of our patients speaks for relatively considerable damage to the visual pathways. It must be additionally stressed here that VEPs' examination in particular fulfills all criteria of objectivity, because the patient lacks any means of influencing the results.

The above findings indicate that the visual deterioration seen after aneurysmal SAH does not depend on the way in which the aneurysm has been secured. Consequently, the nature of SAH itself ought to be inspected for a mechanism responsible for this effect. Obviously, vascular insufficiency is first to be considered. According to the anatomical studies [8], several minute arteries arising mainly from the ophthalmic segment of the ICA run to the optic nerves and chiasm from below. They both perforate these cerebral structures and give off tiny and extremely thin-walled (and thus fragile) branches, which traverse around the whole surface of the optic nerves and chiasm (Fig. 3). It is apparent that these tiny, vulnerable superficial vessels come into direct contact with extravasated blood of subarachnoid hemorrhage and may develop spasm with consequent ischemia within their territory of supply. Somewhat different is anatomical situation of the above-mentioned main feeders to the nerves/chiasm. In its ophthalmic segment, the ICA and the optic nerve remain in a close, intimate contact. Therefore, the feeders to these structures remain to some extent hidden and less exposed to blood or its degradation products known to produce vasospasm [9–11]. Also, the perforating branches of these arteries can maintain their function and continue feeding the core of the nerves

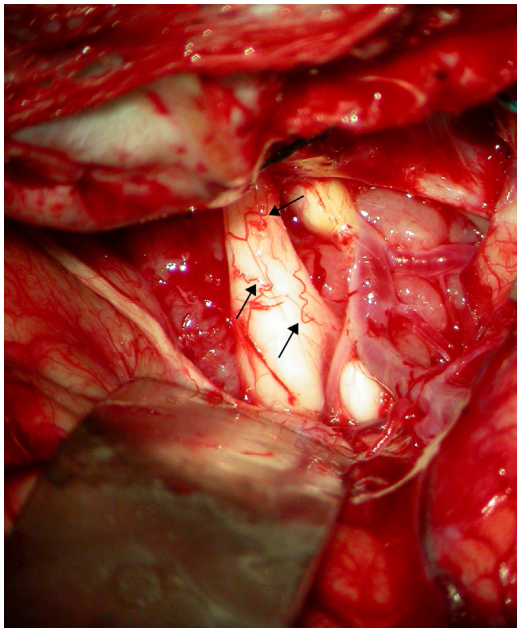

Fig. 3 – Intraoperative photograph showing minute arterioles on the optic nerve surface (arrows), which are naturally exposed to cerebro-spinal fluid within chiasmatic cistern.

(where the majority of macular fibers are positioned). Only in this way can we speculatively explain the pattern of visual field defects seen in patient after SAH: spared central zone and peripherally located deficits.

Unlikely the studies on predictors of neuropsychological deficit after aneurismal SAH, our investigation did reveal factors which potentially determine the outcome related to visual sensory deficit. Both univariate and binary logistic regression analysis have pointed out three factors associated with the occurrence of visual field defects and abnormal VEPs. Two of them i.e. high score in Hunt-Hess and Fisher scales, reflect the severity of SAH and the amount of blood present in the subarachnoid space. This finding strengthens our hypothesis of the vascular mechanism underlying the damage to the visual pathway discussed above. It also supports the notion that visual disturbances which we once found in patients in whom aneurysm was clipped were caused rather by SAH itself and not by surgical manipulation on and around the optic nerves [5].

An additional factor to influence the outcome with respect to visual deterioration was age of the patient. Patients who had visual field deficit and VEP disturbances were on average 9 years older than those who did not develop such sequelae. It could be also of interest to note that patients with abnormal VEP (and also those with decreased VA) were on average 3 years older than those affected by visual field restriction.

The results of our study need to be considered in the context of some potential limitations. Our present and formerly published results indicate that the impairment of visual functions occurs in patients with SAH irrespective of the mode of treatment: coiling and clipping of the aneurysm. While it is tempting to conclude that surgical manipulations do not add to the rate of damage to the visual pathway after

aneurismal SAH, it must be remembered that our set-up does not allow for any (except speculative) considerations about how the intravascular manipulation itself affects the visual pathways. A further investigation of patients who had undergone embolization of unruptured aneurysm would help in addressing this question.

Another limitation pertains to the fact that in the literature there is virtually no data available on the late ophthalmological status of patients after aneurismal SAH. For this reason, we could not use literature references in the present study, except the one we have published previously [5]. It must be noted, however, that our two groups of patients after SAH are not fully homogenous. Main difference pertained to classification in Fisher scale, (i.e. massiveness of SAH) and to distribution of aneurysm location. The distribution of patients in Fisher scale was shifted to its higher scores among the group treated with endovascular coiling in comparison to those treated with aneurysm clipping. Also, in the group of coiling there was a subgroup of 11 aneurysms of the vertebro-basal arteries; no such aneurysm was clipped in our material. Nevertheless, it must be said that the incidence of damage to the visual pathways was substantially lower among patients with BVA aneurysms in comparison to the patients with supratentorial aneurysms (36% vs. 56%, respectively).

5. Conclusions

Our study indicates that more than one half of patients with SAH and aneurysm embolization suffer from varying degrees of late visual deteriorations. The pattern of those disturbances corresponds to damage to the visual pathway; specifically, the optic nerves and chiasm. Visual deterioration occurs more frequently in patients with higher score in Hunt-Hess and Fisher scales – both of them reflecting the severity of SAH and the amount of blood present in the subarachnoid space.

Conflict of interest

None declared.

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Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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