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Early postoperative visual impairments after surgery for tumors of the chiasmal and sellar region and the ways for correcting them

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Purpose: to assess the frequency of visual function deterioration in the early postoperative period after the removal of tumors of the choroidal and sellar region (CSR), and develop effective treatment methods for postoperative local vasospasm.

Material and Methods. We retrospectively reviewed the medical records of 438 patients treated for compressive optic neuropathy due to tumors of the CSR at the Department for Endonasal Cranial Base Endosurgery, SI «Romodanov Neurosurgery Institute, National Academy of Medical Sciences of Ukraine», in 2018-2024. The main group (group 1) was formed of 33 patients (66 eyes) with postoperative visual deterioration to assess the proposed strategy of treatment for postoperative local vasospasm. The retrospective control group (group 2) was used for comparison and comprised 34 patients (68 eyes) with a similar deterioration who underwent tumor surgery in 2014-2018 and did not receive the restorative treatment. Patients underwent clinical neurological and ophthalmological examinations and neuroimaging and functional studies.

Results. We analyzed the outcomes of treatment for postoperative vasospasm which included hemodilution, calcium channel blockers and peripheral vasodilators. In group 1, at day 1 and month 1 after surgery, the mean visual acuity (VA) was 0.44 ± 0.05 and 0.59 ± 0.04 , respectively, and the mean mean deviation (MD), -15.34 ± 0.77 dB and -11.51 ± 0.79 dB, respectively ($p < 0.05$). In group 2, the mean VA improved from 0.38 ± 0.05 to 0.43 ± 0.04 , and the mean MD improved from -15.46 ± 0.73 dB to -13.68 ± 0.69 dB, at month 1 compared to day 1 after surgery ($p > 0.05$). In group 1 and group 2, the mean VA was 0.44 ± 0.05 and 0.38 ± 0.04 , respectively, at day 1 after surgery ($p > 0.05$), and 0.59 ± 0.04 and 0.43 ± 0.04 , respectively, at month 1 after surgery ($p < 0.05$). Additionally, the mean MD was -15.34 ± 0.77 dB and 15.46 ± 0.73 dB, respectively, at day 1 after surgery ($p > 0.05$), and -11.51 ± 0.79 dB and -13.68 ± 0.69 dB, respectively at month 1 after surgery ($p < 0.05$).

Conclusion. With the proposed therapeutic treatment, VA improved from 0.44 ± 0.05 at day 1 after surgery to 0.59 ± 0.04 at one month after surgery, whereas MD improved from -15.34 ± 0.77 dB to -11.51 ± 0.79 dB ($p < 0.05$).

Key words:

tumors of the chiasmal and sellar region, compressive optic neuropathy, chiasmal blood supply, endoscopic endonasal surgery for tumors of the chiasmal region, local vasospasm, restorative treatment

Introduction

The impact of mass effect on the optic nerve/chiasm complex (ONCC) is typical for the tumors of the chiasmal and sellar region (CSR) which make up about 15% of primary brain tumors and about 25% of primary intracranial benign tumors [1]. Benign tumors (like pituitary adenoma (PA), parasellar meningioma (PM) and craniopharyngioma (CP)) are much more common than malignancies (chorioma, glioma, astrocytoma and cancer metastases) [1, 2].

Owing to close proximity of the CSR to the visual pathways, there is potentially a high risk of compressive optic neuropathy (CON) which is accompanied by reduced visual acuity (VA), visual field defects, and descending optic atrophy (OA). The latter develops in a substantial portion (16-72%) of patients with tumors of the CSR and may cause blindness in 3.5-25% of cases [3, 4]. Visual impairments emerge in early disease and are the basic clinical findings in most tumors of the CSR [2, 5].

Endoscopic endonasal excision is a current standard surgical approach for tumors of the CSR, aiming to decompress the anterior visual pathway and improve, restore or stabilize the visual function. Some proportion of patients may exhibit the emergence, no change in or worsening of, VA and/or visual field abnormalities despite successful surgery [6-10].

Worsening of visual function is observed in 1.5-25% of patients after decompression of the optic nerve/chiasm [11-16]. The causes of early postoperative loss of vision include the use of transsphenoidal approach, a chiasmal prolapse into the empty sella, postoperative hematoma at operation site, ischemia, direct injury to (or devascularization of) the optic apparatus, excessive fat grafting, and cerebral vasospasm [17, 18].

Secondary mechanisms of visual loss include hypotension causing hypoperfusion of the already damaged system of branches; optic pathway edema caused by vascular factors; and reperfusion damage [19].

The topic is important since there is a need for the development of effective methods of treatment and strategies for reducing negative postoperative sequelae and improving the postoperative quality of life.

The purpose of this study was to assess the frequency of visual deterioration in the early postoperative period after the removal of tumors of the CSR, and develop effective treatment methods for postoperative local vasospasm.

Material and Methods

We retrospectively reviewed the medical records of 438 patients treated for CON due to tumors of the CSR at the Department for Endonasal Cranial Base Endosurgery, SI "Romodanov Neurosurgery Institute, National Academy of Medical Sciences of Ukraine" (RNI), in 2018-2024. All surgeries aimed at removing tumors and decompressing the ONCC, were performed through the endoscopic endonasal approach and were radical (i.e., patients had a subtotal or total tumor excision).

To assess the proposed strategy of treatment for postoperative local vasospasm, we formed the main group of 33 patients (66 eyes) with a preoperatively impaired visual function and postoperative unilateral or bilateral worsening of VA and/or visual fields. The control group comprised 34 retrospective cohort patients (68 eyes) treated at the Department for Endonasal Cranial Base Endosurgery, the RNI, in 2014-2018. These patients had visual function impairment preoperatively and unilateral or bilateral worsening of VA and/or visual fields postoperatively but were not treated for postoperative local vasospasm.

Inclusion criteria were age 18 years or older and worsening of visual function (VA and/or visual fields) after endoscopic endonasal subtotal or total resection of a tumor of the ONCC. The groups were matched for refractive error (myopia or hyperopia of less than 3.0D).

Exclusion criteria were patients with continued tumor growth, signs of intracranial hypertension, ocular comorbidity, previous history of radiation therapy, surgery for tumor biopsy, a chiasmal prolapse into the empty sella, postoperative hematoma at operation site, direct injury to the optic nerve, and excessive fat grafting.

The following types of diagnostic methods were used: clinical neurological, ophthalmological and laboratory methods (including total blood cell count, clinical urinalysis and biochemical methods and serum pituitary hormone studies), and neuroimaging and functional studies. Preoperative and postoperative neuroimaging studies included non-enhanced and contrast-enhanced magnetic resonance imaging (MRI) and multislice computed tomography (MSCT).

A 1.5-T MRI system (Inera 1.5T/I system, Philips Medical Systems, Best, the Netherlands) was utilized to obtain non-enhanced and contrast-enhanced T1-weighted

and T2-weighted images in the axial, coronal and sagittal planes.

A Philips Brilliance 64 multislice CT system (Philips Medical Systems, Best, the Netherlands) was utilized to obtain brain MSCT images in the three planes (slice thickness, 0.5 mm). The brain mode was used for improved visualization of bony structures, and the soft tissue mode was used to determine the severity of cerebrospinal fluid (CSF) dynamics abnormalities and the involvement of the parasellar neurovascular structures.

A Toshiba Aplio 400 (Toshiba Medical Systems, Otawara-Shi, Japan) or Canon Aplio i800 (Canon Medical Systems Corporation, Tokyo, Japan) ultrasound system was used for duplex ultrasonography of the head and neck vessels. Blood flow velocities in the internal carotid artery (ICA) and intracerebral arteries were recorded and vasospasm ratios were calculated.

Eye examination included VA assessment, biomicroscopy, kinetic and static perimetry, and direct and indirect ophthalmoscopy. The first eye examination was performed on the day of or after admission, while the second and third examinations were performed on day 1 and month 1 after surgery. The second eye examination was performed on day 1 after surgery due to patients' complaints of visual function worsening after being awakened from medication-induced sleep condition.

Best-corrected VA (BCVA) was classified as normal (1.0), mild impairment (0.7-0.9), moderate impairment (0.4-0.6), severe impairment (0.1-0.3), very severe impairment (< 0.1) and blindness (zero) [19].

Negative dynamics in BCVA was defined as a one-half reduction in the BCVA (for BCVA < 0.1) or at least a 0.2 reduction in the BCVA (for BCVA > 0.1).

Static automated perimetry (SAP) was performed with the Centerfield 2 Perimeter (Oculus, Wetzlar, Germany) using the neurological 30-2 threshold test program and Neuro screening program. Aside from defect localization, the arithmetic mean of the sensitivity loss, the mean deviation (MD), was used to assess visual field loss severity. Visual field loss severity was classified as mild visual field loss (Grade 1; MD, -2 dB to -4 dB), moderate visual field loss (Grade 2; MD, -4 dB to -12 dB), severe visual field loss (Grade 3; MD, -12 dB to -20 dB), and very severe visual field loss (Grade 4; MD, worse than -20 dB).

Negative dynamics in MD was defined as a reduction of 15% or more in visual field extent, an increase in the size of a scotoma or emergence of a scotoma, or any worsening in the grade of visual field loss severity (e.g., a change from grade 3 to grade 4).

Restorative treatment was initiated within the first day after surgery, following MSCT of the brain, to prevent early postoperative complications necessitating urgent surgical intervention, and after no evidence of signs of the involvement of cerebral arterial vasospasm was found by head and neck ultrasound.

Preoperative and postoperative brain MRI views are presented in Fig. 1.

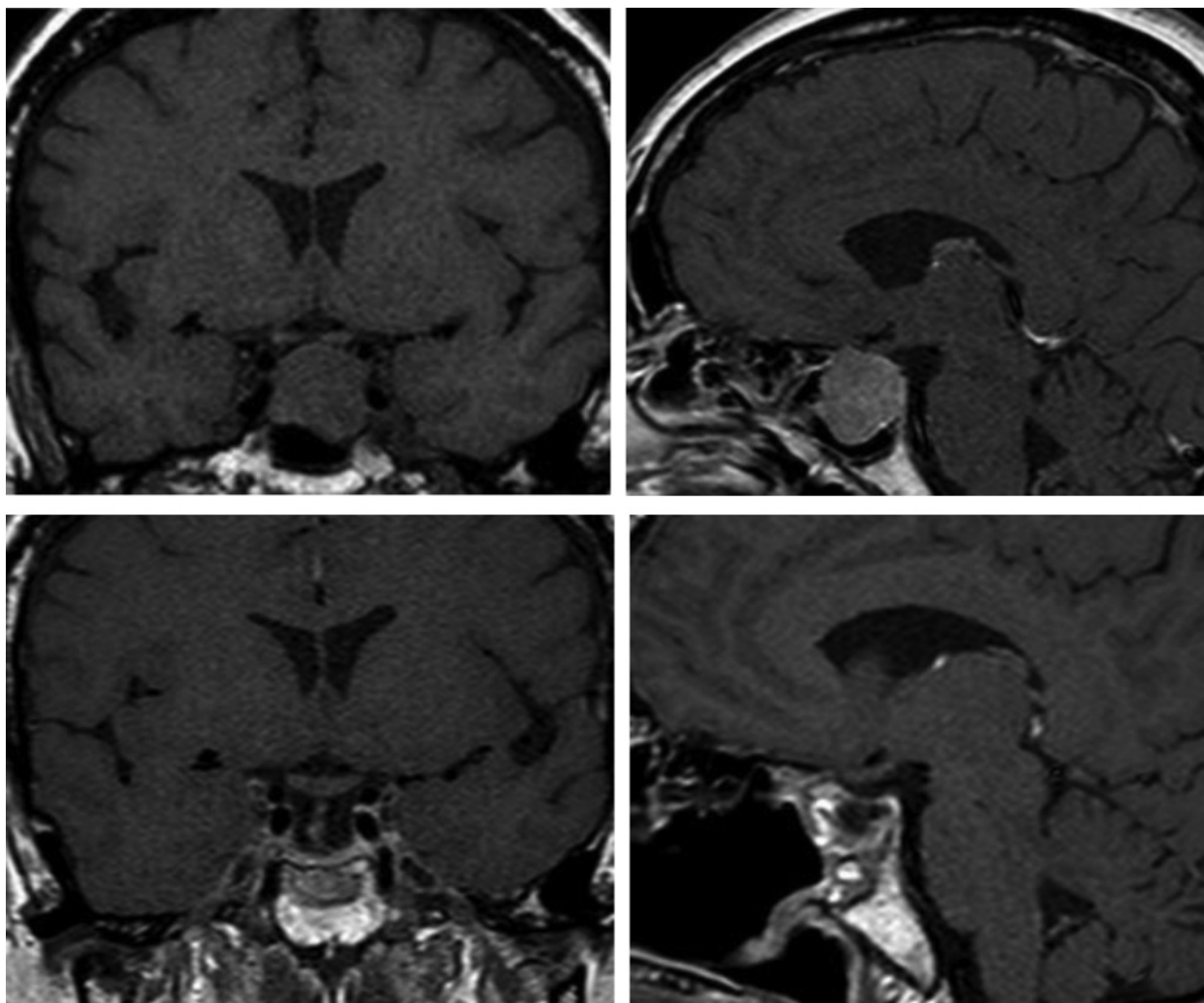


Fig. 1. MRI of the brain of a female patient who underwent total excision of endosuprasellar hormonally inactive pituitary adenoma. (A) Preoperative coronal view. (B) Preoperative sagittal view. (C) Postoperative coronal view. (D) Postoperative coronal view.

The main group was treated with infusion therapy including hemodilution, calcium channel blockers (nimodipine) and peripheral vasodilators (pentoxifylline). Hemodilution was achieved with electrolyte infusion solution (containing Na, 145.0 mMol/l; K, 4.0 mMol/l; Ca, 2.5 mMol/l; Mg, 1.0 mMol/l; chloride, 127.0 mMol/l; acetate, 24.0 mMol/l; and malate, 5.0 mMol/l), 1000 ml daily, and saline, 1000 ml daily, for 10 days. Nimodipine was administered orally, 60 mg six times daily for 10 days. Pentoxifylline was administered initially as intravenous infusion at a dose of 100 mg daily for 10 days, and then switched to oral pentoxifylline at a dose of 600 mg daily for three weeks.

In both groups, patients received antibacterial therapy and thromboprophylaxis. If required, patients received fluid and electrolyte correction. Additionally, in the early postoperative period, corticosteroids were given to the patients who had developed postoperative edema caused by brain matter traction.

The study involved human subjects, adhered to the tenets of the Declaration of Helsinki, and was approved by the Ethics Committee of the Romodanov Neurosurgery Institute (minutes no. 5 of 13.12.2017). Informed consent was obtained from all patients. This study was not conducted on animals.

The data were input into an Excel spreadsheet. Statistical analyses were conducted using SPSS Statistics v.30 (IBM Corp, Armonk, NY) software. The Shapiro-Wilk test was used to assess the normality of study variables. Results are presented as the mean and standard deviation ($M \pm SD$) or standard error of mean (m). Two independent groups were compared using Student t-test for normally distributed variables.

Results

Of the 483 patients, 285 (59%) were women and 198 (41%) were men. Patient age ranged from 18 to 80 years (mean \pm SD, 51.2 ± 12.7 years). Of the 483 patients with tumors of the CSR, 361 (722 eyes) had PA, 64 (128 eyes)

had suprasellar supradiaphragmatic CP, and 58 (116 eyes) had PM.

Visual field impairment was the most common complaint, reported by 205 patients (42.4%), followed by reduced VA (185 patients or 38.3%), headache (104 patients or 21.5%) and diplopia (40 patients or 8.3%). Aside from headache, other general cerebral symptoms complained of were nausea (34 patients or 7%), vomiting (17 patients or 3.5%), cognitive abnormalities (11 patients or 2.3%), instability of gait (6 patients or 1.2%), and urinary incontinence (4 patients or 0.8%).

Pituitary adenomas were hormonally inactive in 298 (82.6%) patients, and prolactin-, growth hormone- and adrenocorticotrophic hormone-secreting, in 34 (9.4%), 23 (6.4%), and 6 (1.7%), respectively.

Hypopituitarism was found in 34 (53.1%) of patients with CP.

All 483 (100%) patients had postoperative worsening of VA and/or visual fields. Of these patients, 37 (7.7%) had unilateral, and 446 (92.3%) had bilateral worsening of VA and/or visual fields. Additionally, 428 (88.6%) patients had unilateral or bilateral worsening of VA. All 483 (100%) patients had postoperative visual field defects. Of these patients, 439 (878 eyes; 90.9%) had bilateral, and 44 (44 eyes; 9.1%) had unilateral visual field defects. Temporal hemianopia with central scotoma was the commonest

visual field defect (267 eyes; 27.6%), followed by temporal hemianopia (either complete or partial) alone (252 eyes; 30.7%), and central scotoma with temporal visual field loss (132 eyes; 13.7%). Ophthalmoscopy found primary descending OA in 348 (72.1%) patients, including 238 patients (476 eyes) with bilateral OA and 110 patients (110 eyes) with unilateral OA.

All patients received a surgical treatment aimed at decompressing the ONCC, which was performed through the endoscopic endonasal approach and was radical (i.e., patients had a subtotal or total tumor excision).

Postoperatively, VA and visual fields in both eyes restored in 60 (12.4%) patients, improved in 303 (62.7%) patients, remained unchanged in 80 (16.6%) patients, and worsened in 40 (8.3%).

The group used for assessing the effectiveness of the proposed strategy of treatment for postoperative local vasospasm was composed of 33 patients.

The effectiveness of the proposed restorative treatment strategy was assessed in the postoperative period after the removal of tumors of the CSR, with patients in group 1 (33 patients, 66 eyes) receiving, and patients in group 2 (34 patients, 68 eyes) not receiving the treatment.

Major characteristics of patients in these two groups are presented in Table 1. Age, gender, histological tumor type, duration of visual impairment, and tumor volume

Table 1. Clinical characteristics of study groups of patients with tumors of the chiasmatal and sellar region in the preoperative and postoperative periods

Clinical data	Group 1, 33 patients (66 eyes)	Group 2, 34 patients (68 eyes)	P value
Age, M ± SD, years	51.6 ± 11.2	51.9 ± 8.2	0,13 p = 0.9006*
Female, n	24	18	2.803 p = 0.095**
Male, n	9	16	2.803 p = 0.095**
Pituitary adenoma, n	18	25	2.625 p = 0.106**
Parasellar meningioma, n	5	3	0.638 p = 0.425**
Supradiaphragmatic craniopharyngioma, n	10	6	1.476 p = 0,225**
Preoperative visual acuity, M ± SD	0.57 ± 0.38	0.56 ± 0.37	0.14 p = 0.88*
Postoperative visual acuity, M ± SD	0,44 ± 0,37	0,38 ± 0,38	0,85 p = 0.39*
Preoperative MD, M ± SD, dB	-11.97 ± 6.59	-11.35 ± 6.61	0.54 p = 0.58*
Postoperative MD, M ± SD, dB	-15.34 ± 6.26	-15.46 ± 6.07	0.11 p = 0.91*
Duration of visual impairment, M±SD, months	11.72 ± 16.5	12.89 ± 16.1	0.30 p = 0.76*
Tumor volume, M ± SD, cm ³	22.14 ± 19.8	19.73 ± 9.87	0.36 p = 0.71*

Note: M, mean value; MD, mean deviation; n, number of patients; P value, significance of difference between groups (*, P-value as assessed by Student's t-test; (**, P-value as assessed by Pearson's chi-squared test); SD, standard deviation

Table 2. Mean values of visual acuity ($M \pm m$) and mean deviation ($M \pm m$) in groups of patients at day 1 and month 1 after surgery

No.	Group of patients, n (eyes)	Visual acuity ($M \pm m$)		MD ($M \pm m$) dB	
		Day 1 after surgery	Month 1 after surgery	Day 1 after surgery	Month 1 after surgery
1.	Group 1, n = 66	0.44 \pm 0.05	0.59 \pm 0.04	-15.34 \pm 0.77	-11.51 \pm 0.79
2.	Group 2, n = 68	0.38 \pm 0.05	0.43 \pm 0.04	-15.46 \pm 0.73	-13.68 \pm 0.69
	p_1	$p = 0.02$		$p = 0.00$	
	p_2	$p = 0.44$		$p = 0.06$	
	$p_{1-2 \text{ day } 1}$	$p = 0.39$		$p = 0.91$	
	$p_{1-2 \text{ month } 1}$	$p = 0.00$		$p = 0.04$	

Note: M, mean value; m, error of mean; MD, mean deviation; p_1 , comparison of intragroup data from day 1 after surgery to month 1 after surgery for group 1; p_2 , comparison of intragroup data from day 1 after surgery to month 1 after surgery for group 2; $p_{1-2 \text{ day } 1}$, comparison of data between groups at day 1 after surgery; $p_{1-2 \text{ month } 1}$, comparison of data between groups at month 1 after surgery; p, P-value as assessed by Student's t-test; SD, standard deviation

demonstrated a homogeneous distribution among the two groups ($p > 0.05$).

Preoperatively, patients in both groups exhibited reduced VA and visual field defects. In group 1, 20 (30.3%) eyes had a VA of 1.0, 9 (13.6%) eyes had a VA of 0.7-0.9, 13 (19.7%) eyes had a VA of 0.4-0.6, 13 (19.7%) eyes had a VA of 0.1-0.3, and 11 (16.7%) eyes had a VA lower than 0.1. Additionally, temporal hemianopia with central scotoma was the commonest visual field defect (21 eyes; 31.8%), followed by central scotoma with temporal visual field loss (16 eyes; 24.2%), complete temporal hemianopia (9 eyes; 13.6%), partial temporal hemianopia (6 eyes; 9.1%), residual nasal visual field (5 eyes; 7.6%), and homonymous hemianopia (4 eyes; 6.1%). Visual field showed no change in 3 (4.5%) eyes and was not measurable in 2 (3.1%) eyes. Moreover, ophthalmoscopy found primary descending OA in 29 (87.9%) patients, including 25 patients (50 eyes) with bilateral OA and 4 patients (4 eyes) with unilateral OA.

In group 2 (34 patients, 68 eyes), 21 (30.9%) eyes had a VA of 1.0, 8 (11.8%) eyes had a VA of 0.7-0.9, 15 (22.1%) eyes had a VA of 0.4-0.6, 14 (20.5%) eyes had a VA of 0.1-0.3, and 10 (14.7%) eyes had a VA lower than 0.1. Additionally, temporal hemianopia with central scotoma was the commonest visual field defect (21 eyes; 30.9%), followed by complete temporal hemianopia (14 eyes; 20.6%), central scotoma with temporal visual field loss (13 eyes; 19.1%), partial temporal hemianopia (6 eyes; 8.8%), residual nasal visual field (6 eyes; 8.8%), and homonymous hemianopia (2 eyes; 2.9%). Visual field showed no change in 5 (7.4%) eyes and was not measurable in 1 (1.5%) eye. Moreover, ophthalmoscopy found OA in 30 (88.2%) patients, including 27 patients (54 eyes) with bilateral OA and 3 patients (3 eyes) with unilateral OA.

Preoperatively, in groups 1 and 2, mean VA was 0.57 \pm 0.05 and 0.56 \pm 0.05, respectively, and mean MD was 11.97 \pm 0.81 dB and 11.35 \pm 0.8 dB, respectively, with no significant difference between the groups ($p > 0.05$). On day 1 after surgery, in both groups, VA worsened, but not significantly ($p > 0.05$), and visual fields worsened significantly ($p < 0.05$).

Patients in group 1 received treatment according to the proposed scheme after they underwent postoperative follow-up imaging and duplex ultrasound scanning. There was no duplex ultrasound evidence of cerebral arterial vasospasm in the groups: the peak systolic blood flow velocity was within the normal range for the age of this cohort, and the Lindegaard ratio for the right and the left sides were 2.25 \pm 0.3 and 2.33 \pm 0.3, respectively, for group 1, and 2.18 \pm 0.2 and 2.21 \pm 0.3, respectively, for group 2, with normal values being less than 3.0.

Table 2 shows mean VA and MD values at day 1 and month 1 after surgery for groups 1 and 2.

In group 1, at day 1 and month 1 after surgery, the mean VA was 0.44 \pm 0.05 and 0.59 \pm 0.04, respectively, and the mean MD was -15.34 \pm 0.77 dB and -11.51 \pm 0.79 dB, respectively, with a significant difference between time points ($p < 0.05$).

In group 2, the mean VA improved from 0.38 \pm 0.05 to 0.43 \pm 0.04, and the mean MD improved from -15.46 \pm 0.73 dB to -13.68 \pm 0.69 dB, at month 1 compared to day 1 after surgery, with no significant difference between time points ($p > 0.05$).

In group 1 and group 2, the mean VA was 0.44 \pm 0.05 and 0.38 \pm 0.04, respectively, at day 1 after surgery ($p > 0.05$), and 0.59 \pm 0.04 and 0.43 \pm 0.04, respectively, at month 1 after surgery with a significant difference between groups ($p < 0.05$).

Additionally, the mean MD was -15.34 ± 0.77 dB and 15.46 ± 0.73 dB, respectively, at day 1 after surgery ($p > 0.05$), and the mean MD was -11.51 ± 0.79 dB and -13.68 ± 0.69 dB, respectively, at month 1 after surgery ($p < 0.05$).

Discussion

This study assessed the incidence of early postoperative visual impairments in patients that underwent the removal of tumors of the CSR. On the basis of the data obtained, we developed the algorithm of visual restorative treatment which efficiency was assessed in study groups of patients.

In the early postoperative period after the removal of tumors of the CSR, the rate of visual function worsening compared to preoperative status was 6.8%, which is in agreement with the reports that the resection of tumors of the CSR is associated with a 1.5-25% risk of post-operative visual impairment [11-16].

Visual deterioration can occur immediately or early (within a few hours) after surgery, resulting either from direct damage to, or devascularization of the optic nerve [19, 21].

Diffuse cerebral vasospasm is known to occur as a result of acute neurosurgical pathology accompanied by subarachnoid hemorrhage [22].

Cerebral vasospasm is a rare complication of cranial base surgery, with the diagnosis being difficult due to a delayed development of the disease and variable clinical manifestations. The pathogenetic mechanisms of clinical complications are likely to involve the development of lesions of the microcirculation of perforating arteries supplying the ONCC [10, 19, 23, 24].

The blood supply of the chiasm and the ONCC is characterized by individual anatomic variability, a variety of sources and well-developed network of perforating arteries. It can be divided into ventral, dorsal, superior and inferior blood supply.

The dorsal blood supply of the ONCC is provided by branches of the ICA, anterior cerebral and anterior communicating arteries. The ventral blood supply is provided by branches of the posterior communicating and basilar arteries. The blood supply of the superior chiasm is provided by branches of the ICA, anterior cerebral and anterior communicating arteries, whereas that of the inferior chiasm is provided by branches of the ICA, basilar and posterior communicating arteries [25].

Blood is supplied to the intracranial portion of the optic nerve and chiasm by the arteries arising from the ophthalmic, posterior communicating, and choroidal branches of the C4 segment of supraclinoid internal carotid artery. The superior hypophyseal and infundibular arteries play the most important roles. The superior hypophyseal arteries (SHA) are a group of one to five small branches that arise from the C4's ophthalmic segment and terminate on the pituitary stalk and gland, but also send branches to the optic nerve and chiasm. The largest of the branches is often referred to as the SHA. The infundibular arteries are a group

of arteries that originate from the posterior communicating artery to the pituitary stalk. The SHA and infundibular arteries intermingle and form an anastomotic plexus called the circuminfundibular anastomosis, and it is the small ascending arteries arising from the plexus that supply the inferior surface of the optic chiasm [26].

Hemodynamic augmentation (with hemodilution, hypertension, and hypervolemia), systemic pharmacotherapy, intra-arterial antispasmodic medications/vasodilation, and angioplasty have been reported for the treatment of cerebral vasospasm. Intraarterial treatment most commonly included papaverin, verapamil, nimodipine and milrinone [10, 19, 23].

The excision of tumors of the CSR is accompanied by local vasospasm resulting from damage to the blood supply of the optic nerve and chiasm. Local microvascular response affects the perfusion of the ONCC. Vascular injury, intraoperative vascular manipulations, or surgical removal of a tumor along with blood vessels (especially when the tumor is in close contact with the vascular wall) can result in impaired vasodilation, which is considered a mechanism of the development of postoperative vasospasm.

Therefore, in the current study, the visual function worsening after the resection of tumors of the CSR was thought to occur as a consequence of hemodynamic disorders in the anastomotic system of perforators supplying the ONCC. Cases of severe damage to the chiasm or tumor invasion of the chiasm were excluded from the analysis. Therefore, it is the development of local vasospasm due to manipulations of the above arteries and/or reaction to the presence of blood in the chiasmatic cistern that was taken as a working hypothesis. In line with this concept, we used a postoperative treatment scheme similar to those used in cerebral vasospasm.

For the main group of patients, VA and visual field measurements improved significantly ($p < 0.05$) with the restorative treatment. There was a significant difference in the VA and visual field outcomes ($p < 0.05$) between the main group and the retrospective group.

With the proposed restorative treatment, VA improved from 0.44 ± 0.05 at day 1 after surgery to 0.59 ± 0.04 at one month after surgery, whereas MD improved from -15.34 ± 0.77 dB to -11.51 ± 0.79 dB, and these improvements were statistically significant ($p < 0.05$).

The results obtained indirectly indicate that visual function loss after endoscopic endonasal surgery is relatively rare, is associated mostly with a local vascular mechanism and should receive effective medical correction.

References

1. Bresson D, Herman P, Polivka M, Froelich S. Sellar Lesions/ Pathology. *Otolaryngol Clin North Am.* 2016 Feb;49(1):63-93. doi: 10.1016/j.otc.2015.09.004.
2. Gotecha S, Kotecha M, Punia P, Chugh A, Shetty V. Neuro-Ophthalmic Manifestations of Intracranial Space Occupying Lesions in Adults. *Beyoglu Eye J.* 2022 Nov 15;7(4):304-312. doi: 10.14744/bej.2022.50469.

3. Tagoe NN, Essuman VA, Fordjuor G, Akpalu J, Bankah P, Ndanu T. Neuro-Ophthalmic and Clinical Characteristics of Brain Tumours in a Tertiary Hospital in Ghana. *Ghana Med J*. 2015 Sep;49(3):181-6. doi: 10.4314/gmj.v49i3.9.
4. Schmalisch K, Milian M, Schimitzek T, Lagrèze WA, Honegger J. Predictors for visual dysfunction in nonfunctioning pituitary adenomas - implications for neurosurgical management. *Clin Endocrinol (Oxf)*. 2012 Nov;77(5):728-34. doi: 10.1111/j.1365-2265.2012.04457.x.
5. Nuijts MA, Veldhuis N, Stegeman I, van Santen HM, Porro GL, Imhof SM, et al. Visual functions in children with craniopharyngioma at diagnosis: A systematic review. *PLoS One*. 2020 Oct 1;15(10):e0240016. doi: 10.1371/journal.pone.0240016.
6. Agosti E, Alexander AY, Leonel LCPC, Van Gompel JJ, Link MJ, Pinheiro-Neto CD, et al. Anatomical Step-by-Step Dissection of Complex Skull Base Approaches for Trainees: Surgical Anatomy of the Endoscopic Endonasal Approach to the Sellar and Parasellar Regions. *J Neurol Surg B Skull Base*. 2022 Aug 25;84(4):361-374. doi: 10.1055/a-1869-7532.
7. Al-Bader D, Hasan A, Behbehani R. Sellar masses: diagnosis and treatment. *Front Ophthalmol (Lausanne)*. 2022 Nov 24;2:970580. doi: 10.3389/fopht.2022.970580.
8. Azab WA, Khan T, Alqunae M, Al Bader A, Yousef W. Endoscopic Endonasal Surgery for Uncommon Pathologies of the Sellar and Parasellar Regions. *Adv Tech Stand Neurosurg*. 2023;48:139-205. doi: 10.1007/978-3-031-36785-4_7.
9. Rawanduzy CA, Couldwell WT. History, Current Techniques, and Future Prospects of Surgery to the Sellar and Parasellar Region. *Cancers (Basel)*. 2023 May 24;15(11):2896. doi: 10.3390/cancers15112896.
10. Budnick HC, Tomlinson S, Savage J, Cohen-Gadol A. Symptomatic Cerebral Vasospasm After Transsphenoidal Tumor Resection: Two Case Reports and Systematic Literature Review. *Cureus*. 2020 May 17;12(5):e8171. doi: 10.7759/cureus.8171.
11. Gnanalingham KK, Bhattacharjee S, Pennington R, Ng J, Mendoza N. The time course of visual field recovery following transphenoidal surgery for pituitary adenomas: predictive factors for a good outcome. *J Neurol Neurosurg Psychiatry*. 2005 Mar;76(3):415-9. doi: 10.1136/jnnp.2004.035576.
12. Mortini P, Barzaghi R, Losa M, Boari N, Giovanelli M. Surgical treatment of giant pituitary adenomas: strategies and results in a series of 95 consecutive patients. *Neurosurgery*. 2007 Jun;60(6):993-1002; discussion 1003-4. doi: 10.1227/01.NEU.0000255459.14764.BA.
13. Schick U, Hassler W. Surgical management of tuberculum sellae meningiomas: involvement of the optic canal and visual outcome. *J Neurol Neurosurg Psychiatry*. 2005 Jul;76(7):977-83. doi: 10.1136/jnnp.2004.039974.
14. Engelhardt J, Namaki H, Mollier O, Monteil P, Penchet G, Cuny E, et al. Contralateral Transcranial Approach to Tuberculum Sellae Meningiomas: Long-Term Visual Outcomes and Recurrence Rates. *World Neurosurg*. 2018 Aug;116:e1066-e1074. doi: 10.1016/j.wneu.2018.05.166.
15. Li-Hua C, Ling C, Li-Xu L. Microsurgical management of tuberculum sellae meningiomas by the frontolateral approach: surgical technique and visual outcome. *Clin Neurol Neurosurg*. 2011 Jan;113(1):39-47. doi: 10.1016/j.clineuro.2010.08.019.
16. Kachhara R, Nigam P, Nair S. Tuberculum Sella Meningioma: Surgical Management and Results with Emphasis on Visual Outcome. *J Neurosci Rural Pract*. 2022 Jun 8;13(3):431-440. doi: 10.1055/s-0042-1745817.
17. Chowdhury T, Prabhakar H, Bithal PK, Schaller B, Dash HH. Immediate postoperative complications in transsphenoidal pituitary surgery: A prospective study. *Saudi J Anaesth*. 2014 Jul;8(3):335-41. doi: 10.4103/1658-354.
18. Dinsmore A, Compton C, Kline L, Bhatti MT. I'm stuffed; visual loss after trans-sphenoidal adenomelectomy. *Surv Ophthalmol*. 2014 Jan-Feb;59(1):124-7. doi: 10.1016/j.survophthal.2013.03.008. Epub 2013 Aug 1. PMID: 23911151.
19. Santarius T, Jian BJ, Englot D, McDermott MW. Delayed neurological deficit following resection of tuberculum sellae meningioma: report of two cases, one with permanent and one with reversible visual impairment. *Acta Neurochir (Wien)*. 2014 Jun;156(6):1099-102. doi: 10.1007/s00701-014-2046-4.
20. Dandona L, Dandona R. Revision of visual impairment definitions in the International Statistical Classification of Diseases. *BMC Med*. 2006 Mar 16;4:7. doi: 10.1186/1741-7015-4-7.
21. de Divitiis E, Esposito F, Cappabianca P, Cavallo LM, de Divitiis O. Tuberculum sellae meningiomas: high route or low route? A series of 51 consecutive cases. *Neurosurgery*. 2008 Mar;62(3):556-63; discussion 556-63. doi: 10.1227/01.neu.0000317303.93460.24.
22. Dicipinigaitis AJ, Feldstein E, Shapiro SD, Kamal H, Bauerschmidt A, Rosenberg J, et al. Cerebral vasospasm following arteriovenous malformation rupture: a population-based cross-sectional study. *Neurosurg Focus*. 2022 Jul;53(1):E15. doi: 10.3171/2022.4.FOCUS2277.
23. Bejjani GK, Sekhar LN, Yost AM, Bank WO, Wright DC. Vasospasm after cranial base tumor resection: pathogenesis, diagnosis, and therapy. *Surg Neurol*. 1999 Dec;52(6):577-83; discussion 583-4. doi: 10.1016/s0090-3019(99)00108-1.
24. Eseonu CI, ReFaey K, Geocadin RG, Quinones-Hinojosa A. Postoperative Cerebral Vasospasm Following Transsphenoidal Pituitary Adenoma Surgery. *World Neurosurg*. 2016 Aug;92:7-14. doi: 10.1016/j.wneu.2016.04.099.
25. Bergland R. The arterial supply of the human optic chiasm. *J Neurosurg*. 1969 Sep;31(3):327-34. doi: 10.3171/jns.1969.31.3.0327.
26. Gibo H, Lenkey C, Rhoton AL Jr. Microsurgical anatomy of the supraclinoid portion of the internal carotid artery. *J Neurosurg*. 1981 Oct;55(4):560-74. doi: 10.3171/jns.1981.55.4.0560.

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Abbreviations: CSR, chiasmal and sellar region; CON, compressive optic neuropathy; CP, craniopharyngioma; DUS, duplex ultrasound scanning; MD, mean deviation; MRI, magnetic resonance imaging; MSCT, multispiral computed tomography; ONCC, optic nerve/chiasm complex; PA, pituitary adenoma; PM, parasellar meningioma