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Bilateral glaucoma attack after flow-diverting stent placement in the left inner carotid artery in the projection of the aneurysm neck: a case report

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Tashkent (Uzbekistan)**Key words:**supraclinoid internal carotid artery aneurysm,
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We report a case of the development of bilateral glaucoma after flow-diverting stent placement in the left internal carotid artery (ICA) in the projection of the aneurysm neck. The case confirms the importance of a multidisciplinary approach to the surgical treatment of patients with a supraclinoid ICA aneurysm, and may be of interest not only to ophthalmologists, but also to neurosurgeons and vascular surgeons.

Introduction. Supraclinoid inner carotid artery (ICA) aneurysms comprise 28-36% of all brain aneurysms. The supraclinoid ICA has a close anatomic relationship with the optic nerve, oculomotor nerve, basal temporal and frontal lobes, and cavernous sinus [1].

A supraclinoid ICA aneurysm is manifested by a complex of neuroophthalmological symptoms including ocular pain, dilated pupils, blurred vision, diplopia, unilateral facial paralysis, insomnia, dizziness, and tinnitus [2]. It is not uncommon that patients with supraclinoid ICA aneurysm require a surgical intervention which consists of premedication, induction of anesthesia, and surgery itself [3]. There are few reports which consider a relationship between premedication, anesthesia, surgery itself and elevated intraocular pressure (IOP) [4, 5]. A correlation between the use of anesthetics and elevated IOP has been also reported [8].

Animal studies have demonstrated that an increase in systolic blood pressure can result in elevated IOP [7], and a relationship between increased systolic blood pressure and IOP has been confirmed [6].

We report a case of the development of an ophthalmic complication (bilateral ophthalmic hypertension) after flow-diverting stent placement in the left ICA in the projection of the aneurysm neck. The case confirms the importance of a multidisciplinary approach to the surgical treatment of patients with a supraclinoid ICA aneurysm, and may be of interest not only to ophthalmologists, but also to neurosurgeons and vascular surgeons.

Description of the case

We report a case of 69-year-old female patient who developed an ophthalmic complication (bilateral ophthalmic hypertension) after an intravascular implant (a flow-diverting stent) was placed in her left ICA in the projection of the aneurysm neck.

Premedication with 0.5 mg of intravenous (IV) atropine sulfate 0.1%, 0.7 mg of intramuscular (IM) atropine sulfate 0.1%, and 2.0 ml of fentanyl 0.005% was administered to her before aneurysm surgery.

Anesthesia included 0.5 mg of IV atropine sulfate 0.1% and 0.1 mg of fentanyl 0.005%.

Lisinopril dehydrate 10 mg was administered postoperatively.

On post-operative day 3, the patient began complaining of reduced vision in both eyes and marked pain in the projection of the globes.

She underwent eye examination including visual acuity assessment, Goldmann tonometry, biomicroscopy, gonioscopy, ophthalmoscopy and biometry.

Uncorrected visual acuity was 0.1 in the right eye (OD) and hand motion in the left eye (OS).

IOP was 53 mmHg OD and 32 mmHg OS.

On slit lamp examination of both eyes, the cornea was edematous, the anterior chamber shallow, and the lens showed mild opacification.

The anterior chamber angle was closed on gonioscopy.

The patient experienced dilated pupils (OD = OS; 7 mm).

Ocular motility was full in both eyes.

On ophthalmoscopy, the right optic disc showed pallor temporally and had sharp margins, moderately dilated veins were seen in the right fundus and the artery-vein ratio was 1:3; the left optic disc showed pallor and had sharp margins, severely dilated arteries and moderately dilated veins were seen in the left fundus, the artery-vein ratio was 1:4, and the macula and visible peripheral retina were unremarkable.

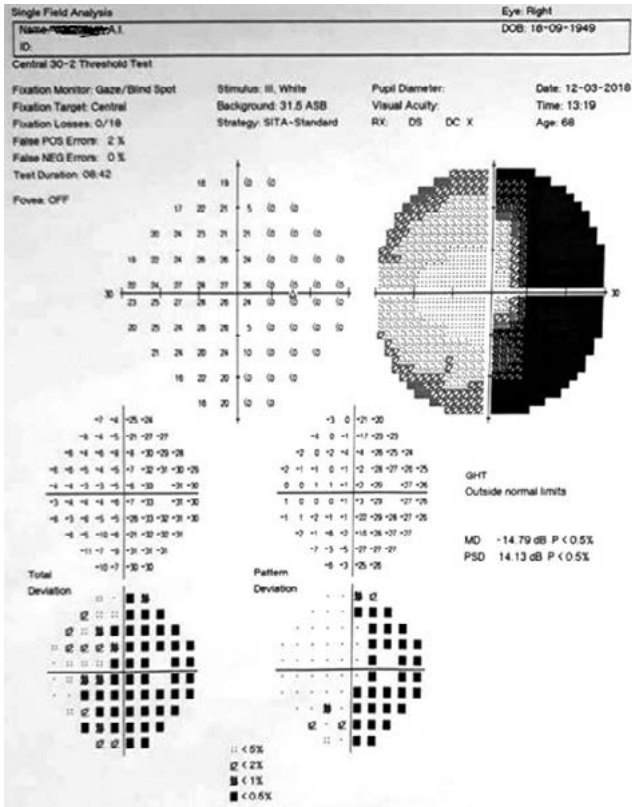


Fig. 1. Automated static perimetry of the right eye shows an absolute temporal hemianopia

Ultrasonic ocular biometry found that anterior chamber depth (ACD), lens thickness (LT) and axial length (AL) were 1.62 mm, 4.9 mm and 22.54 mm, respectively, OD and 1.57 mm, 5.03 mm and 22.58 mm, respectively, OS.

Figure 1 shows the visual field OD. A visual field assessment of the left eye was not possible due to low visual acuity.

The patient was diagnosed with a bilateral acute attack of glaucoma, complicated cataract, chiasmal syndrome, and partial descending optic atrophy.

It is noteworthy that, prior to the presentation to us, she had been followed by her ophthalmologist for ischemic optic neuropathy of the left eye. From 2014 to 2018, she had been complaining of a gradual reduction in vision in her left eye from 0.6 to 0.2, but elevated IOP had not been found in that period.

The patient was administered topical timolol maleate 0.5% solution, topical pilocarpine hydrochloride 1% solution, and oral acetazolamide 250 mg to reduce her IOP. This treatment resulted in a reduction in the IOP to 12 mmHg OD and 18 mmHg OS.

On the second day after the attack was relieved, the IOP was 18 mmHg OD and 25 mmHg OS, with the patient being under an ocular hypotensive treatment regimen with brinzolamide 1% twice and timolol maleate 0.5% both administered twice daily.

On slit lamp examination of both eyes, the conjunctiva was unremarkable, the cornea clear, the anterior chamber moderately shallow, and the lens showed mild opacification.

Ocular motility was full in both eyes.

An OptoYag&SLT M laser system (Optotek Medical, Ljubljana, Slovenia) was used to perform laser iridotomy in both eyes because of the anterior chamber angle closed on gonioscopy in both eyes.

On day 4, uncorrected visual acuity was 0.6 OD and 0.1 OS. In addition, the IOP was 17 mmHg OD and 18 mmHg OS. Moreover, on gonioscopy of both eyes, the anterior chamber angle was open, and the patient had a narrow anterior chamber angle profile.

Discussion

Two conceptual models serve as the basis for understanding IOP [7]. One model treats IOP in terms of the ocular pressure-volume relation, which is an exponential function of the total ocular volume and the elasticity of the corneoscleral coat. The other model treats the steady-state IOP as a function of aqueous flow and outflow resistance (Fig. 2).

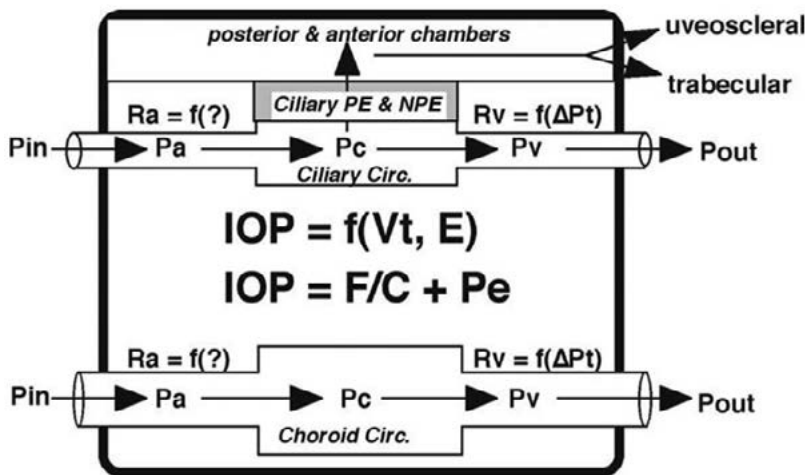


Fig. 2. Schematic of IOP generation (P_{in} : extraocular arterial pressure; P_a : intraocular arterial pressure; P_c : intraocular capillary pressure; P_v : intraocular venous pressure; P_{out} : extraocular venous pressure; R_a : arterial resistance; R_v : venous resistance; ΔP_t : transmural pressure gradient; PE: pigment epithelium; NPE: nonpigment epithelium; V_t : total ocular volume; E : elastance or “rigidity” of the ocular surface; F : aqueous flow; C : outflow conductance or “facility”; P_e : episcleral venous pressure) [7]

If the corneoscleral elastance is constant, changes in IOP must involve changes in ocular volume. The main contributors to the total ocular volume are the vitreous, lens, aqueous and blood. The volumes of the vitreous and lens are relatively stable, whereas the volumes of blood and aqueous are more labile and cause most variations in IOP.

Because most IOP measurement techniques are discontinuous, the effect of blood volume and gradient on IOP generally goes unnoticed [9].

In the current case, the patient had a supraclinoid ICA aneurysm measuring 27 x 23 x 22 mm, for which she underwent surgical intervention (flow-diverting stent placement in the projection of the aneurysm neck). The intervention facilitated the restoration of blood pressure in the ophthalmic artery, and the patient was administered vasodilation agents postoperatively. This sequence of events and measures taken can partially explain the pathophysiological process of the development of bilateral ophthalmic hypertension after flow-diverting stent placement in the left ICA in the projection of the aneurysm neck.

Of interest are reports on the impact of blood pressure and medications administered before, during and/or after anesthesia on IOP, with evidence of the mechanisms explaining elevated IOP (autoregulatory myogenic mechanisms, anesthesia, pupillary block, premedication and mydriasis) [8].

An acute increase in arterial pressure by occluding the aorta causes only a small increase in IOP due to choroidal vasoconstriction under control conditions. The myogenic mechanism regulating hydrostatic pressure is explained in the following way: if arterial or venous pressure rises, the arterial myogenic vasoconstriction would act to preserve capillary hydrostatic pressure [10, 11].

It is, however, important to note that an abrupt and long-term increase in IOP may occur if the regulation of vasoconstriction is blocked by vasodilators (in our patient, lisinopril 10 mg was administered postoperatively).

An experimental study in rabbits demonstrated that raising arterial pressure elicits a modest increase in IOP under control conditions and a much larger increase when choroidal regulation is impaired by systemic vasodilation (Fig. 3). Choroidal vasoconstriction is blocked in the presence of vasodilation, which could occur in our patient [6].

There was a mean increase of 0.44 mmHg for those whose systolic blood pressure increased by 10 mmHg and a decrease of 0.59 mmHg in IOP for those whose systolic blood pressure decreased by 10 mmHg or more [9].

Minimum IOP was 6 mmHg for normotensive patients (those with systolic and diastolic pressures being < 120 mmHg and < 80 mmHg, respectively) and 8 mmHg for hypertensive patients (those with systolic and diastolic pressures being \geq 140 mmHg and \geq 90 mmHg, respectively), and maximum IOP was 26 mmHg for the former patients and 36 mmHg for the latter patients [11].

The pathophysiological basics of the relationship between elevated IOP and increased systolic blood pressure has not been completely elucidated. It is theorized that in-

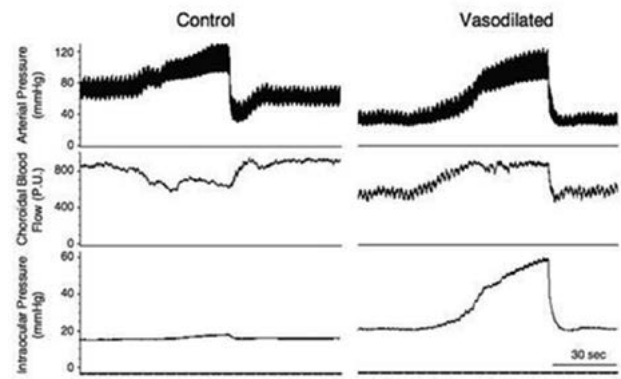


Fig. 3. Intraocular pressure responses to acute increases in arterial pressure in an anesthetized rabbit. Raising arterial pressure to 110 mmHg elicits a modest increase in IOP under control conditions (left) and a much larger increase when choroidal regulation is impaired by systemic vasodilation with hydralazine (right)

creased systemic blood pressure leads to an increase in ciliary body pressure, which can in turn increase the filtration fraction of aqueous humor and lead to elevated IOP [8].

Fentanyl, an opioid analgetic, causes myosis; alpha adrenoreceptor agonists can cause mydriasis, and their simultaneous use with fentanyl increases the duration of iridolenticular contact and may provoke pupillary block [8]. Given that our patient was administered atropine sulfate 0.1%, the involvement of postoperative medical mydriasis in the development of ophthalmic hypertension in this patient cannot be excluded.

Therefore, on the basis of the patient's examination findings and imaging studies, as well as consideration of the potential risk factors for the development of glaucoma reported in the literature, we may suggest a multifactorial cause for the development of bilateral glaucoma after flow-diverting stent placement in the left ICA in the projection of the aneurysm neck.

In our case, we believe that the following factors contributed to the development of bilateral ophthalmic hypertension in our patient:

- patient's ocular anatomic features (ACD, LT and AL were 1.62 mm, 4.9 mm and 22.54 mm, respectively, OD and 1.57 mm, 5.03 mm and 22.58 mm, respectively, OS);
- premedication drugs (0.5 mg of IV atropine sulfate 0.1%, 0.7 mg of IM atropine sulfate 0.1%, and 2.0 ml of fentanyl 0.005%);
- anesthesia drugs (0.5 mg of IV atropine sulfate 0.1% and 0.1 mg of fentanyl 0.005%);
- the surgical intervention (flow-diverting stent placement in the projection of the aneurysm neck with restoration of the blood flow), and postoperative vasodilators (Lisinopril dehydrate 10 mg).

Given the above, this case confirms the importance of a multidisciplinary approach (with examination and IOP control by an ophthalmologist) to the surgical treatment of supraclinoid ICA aneurysm.

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