CASE REPORT

Non-arteritic Anterior Ischaemic Optic Neuropathy after Uneventful Cataract Extraction

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ABSTRACT

We present the case of a 74-year-old Caucasian female who suffered sudden visual loss after routine phacoemulsification cataract surgery. The patient was subsequently diagnosed with non-arteritic anterior ischaemic optic neuropathy. The case is described in detail, and a concise review of the literature is presented together with the authors' view on the subject outlined. This is a very rare complication after cataract surgery even in high-risk patients with associated systemic comorbidities. We suspect that the previous history of obesity, coronary artery disease, and arteriosclerosis contributed to the development of this serious ocular complication. We suggest appropriate measures to reduce the risk of its occurrence.

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Introduction

The incidence of non-arteritic ischaemic optic neuropathy (NAION) is estimated to be 10 in 100,000 in the population aged 50 years and older.¹ In previous studies, the incidence of NAION has been suggested to increase after cataract extraction (CE), to approximately 1 in every 2000 cases. Although the incidence was low, it was statistically higher than the overall incidence rates of NAION as stated above.²

Two categories of CE-associated NAION have been reported: an immediate type occurring hours after CE and a delayed type seen days to weeks after surgery. The incidence of the delayed type was found to be higher than the immediate type.^{2,3}

We present a case of delayed-type NAION occurring after uneventful phacoemulsification cataract surgery in a susceptible individual.

Case report

A 74-year-old Caucasian female presented in our clinic with bilateral moderate cataracts and visual acuity of 6/18 (20/60) in both eyes. She underwent right uneventful phacoemulsification cataract

surgery under sub-Tenon's anaesthesia. Five days postoperatively she experienced acute painless visual loss. She reported waking up with blurred vision in the operated eye.

Previous medical history included body mass index (BMI) of over 30, two coronary artery angioplasties, and current treatment for hypertension and hypercholesterolaemia. She was also treated with oral aspirin 75 mg daily.

On her routine postoperative visit 2 weeks following surgery, she presented with visual acuity of counting fingers and presence of a relative afferent pupillary defect (RAPD) in the operated eye. Dilated fundal examination showed optic disc swelling with the presence of flame-shaped haemorrhages (Figure 1).

After appropriate investigations, including erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) for the exclusion of temporal arteritis, the patient was diagnosed with NAION. No ocular risks for NAION such as small optic disc were identified.

The patient was awaiting cataract surgery in the fellow eye. Due to the vascular incident affecting

Figure 1. Fundus images from both eyes following cataract surgery highlighting the striking difference in the appearance of the optic discs between the right affected eye and left unaffected site. Note the presence of haemorrhages inferiorly in the right optic disc partially obscured by the peripapillary retinal nerve fiber layer oedema (black arrows).

the first eye, the associated systemic vascular risk factors, and her relatively good visual acuity of 6/12 (20/40) in the other eye, surgery has been postponed at present and could be reconsidered should further functional visual deterioration occurs.

Discussion

NAION is associated with a number of systemic risk factors, such as older age, arterial hypertension, nocturnal hypotension, cigarette smoking, diabetes mellitus, hypercholesterolaemia, ischaemic heart disease, cerebrovascular disease, thrombophilia, migraine, and sleep apnoea syndrome.^{1,4,5,6} Non-arteritic anterior ischaemic optic neuropathy has also been associated with blood pressure fluctuation after major surgeries.¹ An ocular risk factor for NAION is a small, “crowded” optic disc with a small cup-to-disc ratio.^{1,4,5,6}

The optic nerve infarct in NAION has been previously described as a form of compartment syndrome that causes tissue ischaemia. The rigidity of the sclera confines optic disc swelling causing the compartment syndrome, with resultant ischaemia.⁴

The pathophysiological mechanism of CE-associated NAION is unclear, but as a causative factor, intraocular pressure increase during surgery has been postulated mainly for the immediate type. Theoretically, perfusion pressure reduction of the posterior ciliary artery results in acute ischaemia of the laminar, prelaminar, and retrolaminar optic nerves as a defining event for NAION.^{1,2}

Cataract surgery is a common, effective, and safe procedure, and vascular complications are

unusual. The literature suggests that after cataract surgery, NAION has a higher incidence in susceptible individuals. This could be related to intraocular pressure fluctuations, locally released vasoactive peptides and systemic prothrombotic and/or proinflammatory factors all somehow influenced by the surgical procedure.^{1–3} However, the prevalence of hypertension and small cup-to-disc ratio was notably lower in patients with NAION following lens-related intraocular surgery than in those with spontaneous NAION, indicating that risk factors for NAION in these settings may be different.⁷

The incidence of NAION occurring in the contralateral eye was found to vary between 24% and 48%, within a period of 5–11 years.⁸

A retrospective study published in 2007 challenged the previous notion that CE-associated NAION was unrelated to prior NAION in the fellow eye.³ The relevant study included a total 325 patients. Seventeen of them underwent cataract surgery in the fellow eye, and 53% developed NAION. Of those affected, two-thirds of the cases occurred within 6 months after CE, with a median of 2.8 months and a range of 2 days to 6.5 years; in the remaining 308 patients who did not have cataract surgery, only 19% developed NAION in the fellow eye. This postulates that the risk of NAION associated with cataract surgery is 4 times higher in patients with previous fellow-eye NAION, and this risk was not reduced when statistical correction for systemic risk factors was employed. This same study, however, could not determine the risk of CE-associated NAION occurring in the fellow eye after CE-associated NAION in the primary eye. That is because only 2 of the 17 patients

with NAION who had fellow-eye CE had prior unilateral NAION in the primary eye occurring within 6 months after CE.³

The incidence of the delayed-type CE-associated NAION was found to be higher than the immediate type, with the latter being linked to raised intraocular pressure during the operative and perioperative periods in the affected patients. It could not be determined with certainty, however, that the NAION experienced after cataract extraction was not due to coincidental spontaneous NAION.^{2,7,9} Due to the nature of the condition and its rarity, we are confined to retrospective studies to report its incidence, as an adequately controlled prospective study would be difficult.^{2,7}

Our patient experienced a delayed-type CE-associated NAION in the clinical context of additional cardiovascular risk. We could assume this occurring due to hypotension, nocturnal or pharmaceutically induced, or even due to sleep apnoea. As this patient's BMI was over 30, the risk for sleep apnoea is noteworthy. The previous history of angioplasties and hypercholesterolaemia also indicates the presence of arteriosclerosis and increased risk of vascular events. All of the above known risk factors for NAION may also be contributing factors to CE-associated NAION in association with surgery-related postulated mechanisms, thus increasing the overall risk of NAION in these patients.

A summary of the most important literature information in relation to cataract surgery and NAION is shown in Table 1.

Interestingly, the operation in our case was performed under sub-Tenon's anaesthesia, which has previously been linked with posterior segment ocular circulation compromise. All orbital blocks, including sub-Tenon's, cause a marked decrease in

pulsatile ocular blood flow. The exact mechanism of this is elusive, but it has been suggested to be associated with an increase in intraocular pressure (IOP) exceeding the mean arterial pressure in the ophthalmic or central retinal artery that may interfere with the small vessels supplying the optic nerve. The manipulation of the globe could also compromise the retinal perfusion as a result of spikes in IOP. It has also been hypothesised that the decrease in ocular blood flow is the result of vasoconstriction caused by the high local concentration of the local anaesthetic drug or inhibition of endothelium-dependent relaxation of the ciliary arteries or even anaesthetic blockade of the vasodilator nerves.¹⁰

This case report of an isolated delayed CE-associated NAION highlights the importance of careful selection, preoperative assessment, and management of patients at higher risk of developing this complication. Aspirin could be used as a prophylactic measure in such cases at a slightly higher dose (100–325 mg/day) than usually administered on a regular basis (75 mg/day).⁸ This could prove useful especially in cases where the fellow eye needs to undergo surgery as well. In addition, susceptible individuals should be considered to undergo surgery under topical anaesthesia only, if appropriate, to eliminate the potential risk of retinal and choroidal ischaemia previously reported after periocular anaesthesia. Preoperative and postoperative monitoring of blood pressure to exclude hypotension especially when treated and postoperative monitoring for excessive inflammatory response and possible raised intraocular pressure could also contribute to reducing the risk of CE-associated NAION. The surgeon must discuss, counsel, and consent the patient preoperatively, and it is advisable for consent to be tailored to the individual. In cases where CE-associated NAION has taken place in the first eye, or there is increased cardiovascular risk, it is important to highlight the risks to the fellow eye.

Our report does not prove a direct causal relationship between CE and delayed NAION. However, we hypothesise an association between these two entities. The possible underlying mechanisms have been pre-

Table 1. Non-arteritic anterior ischaemic optic neuropathy (NAION) and cataract surgery.^{1,2,3}

Outcome	Intervention		
	After cataract surgery	Fellow eye NAION–No cataract surgery	Fellow eye NAION–After cataract surgery
NAION	1 in 2000	19%	53%
No NAION	–	81%	47%

Note. The incidence of delayed NAION after CE (1 per 2000) is significantly higher than the annual incidence of NAION (2.3–10.3 per 100 000).³

sented to support this assumption, together with potential measures to prevent the development of such a complication. Surgeons should always be aware of the rare but devastating development of NAION after routine cataract surgery, recognise susceptible individuals preoperatively, and act accordingly.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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