Non-arteritic anterior ischaemic optic neuropathy and cataract surgery

John Elston

Can cataract surgery trigger NAION in susceptible individuals, and, if so, how?

Non-arteritic ischaemic optic neuropathy (NAION) is a relatively common disorder with an estimated annual incidence of around 10 per 100 000 population aged ≥ 50 years.¹ Small case series have previously suggested that cataract surgery can cause or be associated with the development of NAION, irrespective of previous occurrence of contralateral NAION. In most cases described, vision deteriorated in the immediate postoperative period or within a few weeks after surgery.

In the current issue (Lam B, *et al*², (see page 585)), a retrospective study of 325 cases of unilateral NAION diagnosed between 1986 and 2001 is reported. A total of 17 patients underwent uncomplicated cataract surgery on the contralateral eye in the study period, 9 (53%) then developed NAION in the operated eye, two-thirds in the first 6 months postoperatively. Of the 308 patients who did not undergo surgery, 59 (19%) developed contralateral NAION. Can cataract surgery trigger NAION in susceptible individuals, and if so how?

The pathophysiology of NAION is only partly understood. The defining event is acute ischaemia of the laminar, prelaminar and immediate retrolaminar optic nerve owing to a critical reduction of perfusion pressure through the posterior ciliary arteries. Anatomical factors are an important predisposing factor—a small or “crowded” optic disc is frequently described but not invariable. Systemic vasculopathic and prothrombotic risk factors similar to those for small-vessel occlusive cerebrovascular diseases—for example, diabetes mellitus,

hypertension, cigarette smoking and others—may be present. Nocturnal hypotension could be important. Increasing age is a risk factor (and a risk factor for the development of visually significant cataract). NAION after major surgery has been associated with fluctuations in blood pressure, especially hypotension, and specific operations such as cardiopulmonary bypass procedures and back surgery, particularly with a prone posture. Nevertheless, NAION can occur at any age with any optic disc morphology and without any recognised risk factors.

Cataract surgery is a common, highly effective and safe procedure, and vascular occlusive complications in the operated eye are extremely unusual. The study under discussion was retrospective, although the design is good, with rigorous diagnostic criteria and follow-up data. Nevertheless, there is a risk of overdiagnosis of postoperative NAION inherent in the study design if vision deteriorated after a cataract operation. The possibility that other conditions, such as cystoid macular oedema, could be responsible for reduced vision should be considered. NAION occurring in the immediate postoperative period could relate to intraocular pressure fluctuations, but these are less with modern small incision procedures, and the rate of postoperative NAION was constant over the study period. A non-specific effect of the surgical intervention on systemic prothrombotic and/or proinflammatory factors seems unlikely.

Local vasoactive peptide release during and after uncomplicated cataract surgery may be responsible for the development

of postoperative cystoid macular oedema and possibly the progression of ischaemic diabetic intraocular complications. It seems unlikely that such peptides could affect posterior ciliary artery function, but could conceivably influence optic nerve head perfusion by an effect on intravascular resistance in a predisposed individual.

It is very difficult to ascribe the occurrence of NAION more than a few weeks after a cataract operation to the surgery. Moreover, the development of contralateral NAION in those who did not undergo cataract surgery in this study was low compared with other similar studies. Other studies show a much higher risk of contralateral NAION developing over 10 years—34%–76%, depending on risk factors—compared with 19% over up to 15 years in this patient group.³

However, at least some of the cases of post-cataract NAION reported are likely to be due to factors relating to surgery. If it occurs after a previous contralateral NAION, the effect on overall visual function will be devastating for the patient and upsetting for the surgeon. The absolute risk is undetermined and unlikely to be as high as this study suggests. The mechanism remains undetermined. On the basis of this work and other previous reports, however, a surgeon has the responsibility to discuss this possible risk with an individual with previous NAION considering cataract surgery.

Br J Ophthalmol 2007;**91**:563.

doi: 10.1136/bjo.2006.110007

Correspondence to: Mr J Elston, Oxford Eye Hospital, Walton Street, Oxford OX2 6HE, UK; mary.spearman@orh.nhs.uk

Competing interests: None declared.

REFERENCES

- 1 Hattenhauer MG, Leavitt JA, Hodge DO, *et al*. Incidence of non-arteritic ischaemic optic neuropathy. *Am J Ophthalmology* 1997;**123**:103–7.
- 2 Lam B, J-H Haneen, A-S Nabih, *et al*. Risk of nonarteritic anterior ischaemic optic neuropathy after cataract extraction in the fellow eye of patients with prior unilateral nonarteritic anterior ischaemic optic neuropathy. *Br J Ophthalmol* 2007;**91**:585–7.
- 3 Beri M, Klugman MR, Kohler JA, *et al*. Anterior ischaemic optic neuropathy, VII, incidence of bilaterality and various influencing factors. *Ophthalmology* 1987;**94**:1020–6.