Nonarteritic ischemic optic neuropathy developed after capsular block syndrome

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A 65-year-old man developed capsular block syndrome in the early postoperative period, following phacoemulsification surgery. After neodymium-doped yttrium aluminum garnet (Nd:YAG) laser anterior capsulotomy, the intraocular pressure remained elevated for 4 days despite antiglaucomatous medication. On the postoperative fifth day, nonarteritic ischemic optic neuropathy was diagnosed. To the best of our knowledge, this is the first report of a case with nonarteritic ischemic optic neuropathy associated with early postoperative capsular block syndrome after phacoemulsification surgery.

Key words: Capsular block syndrome, optic neuropathy, phacoemulsification

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Capsular block syndrome (CBS) is a rare complication of modern cataract surgery, which presents with fluid accumulation in the capsular bag.[1] It can occur in the intraoperative, early postoperative and late postoperative periods, and is characterized by intraocular pressure (IOP) elevation, myopic shift and anterior displacement of the iris-lens diaphragm.[2] Nonarteritic anterior ischemic optic neuropathy (NAION) is another rare complication of intraocular lens (IOL) surgery which is presented with loss of vision, ipsilateral relative afferent pupillary defect and disc edema.[3] In this paper, we report the clinical findings and treatment results of a case with NAION that developed after CBS. To the best of our knowledge, this is the first case report of a patient with NAION associated with CBS in the early postoperative period after phacoemulsification surgery.

Case Report

A 65-year-old man with a preoperative best corrected visual acuity of 20/40 had an uneventful phacoemulsification surgery and IOL implantation in the right eye in May 2006. The diameter of the anterior capsule opening was 5 mm and a 23-diopter Sensar IOL (Abbott Medical Optics, Inc., Santa Ana, CA, USA) was implanted using sodium hyaluronate (Healon). Preoperative ophthalmologic and systemic examination was unremarkable. On the first postoperative day, the patient presented with ocular pain. On slit-lamp examination, there

was corneal edema and shallow anterior chamber. The IOP was 40 mmHg and the patient was given topical dorzolamide hydrochloride and timolol maleate combination with 250 mg bid oral acetazolamide. On the postoperative second day, corneal edema decreased and the IOP was 30 mmHg. On slit-lamp examination, backward displacement of posterior capsule and the anterior displacement of IOL were noted [Fig. 1]. The patient's refractive error was −2.00 −1.50 × 105. An early postoperative CBS was diagnosed and neodymium-doped yttrium aluminum garnet (Nd:YAG) laser (Visulas YAD II Plus, Carl Zeiss Meditec AG) anterior capsulotomy was performed to the temporal part of the anterior capsule. As soon as the capsulotomy was carried out, the capsular bag collapsed and the retained ophthalmic viscosurgical device (OVD) was released into the anterior chamber.

On the postoperative fourth day, IOP was still 30 mmHg and anterior chamber venting was performed with the depression of the posterior lip of the paracentesis incision with a sterile forceps. On the postoperative fifth day, the cornea was clear and there was no capsular bag distention. The refractive error was plano −1.25 × 105 and the IOP was 16 mmHg. The visual acuity was noted to be finger counting from 4 m. Fundoscopic examination revealed optic disc edema and fluorescein angiography showed hyperfluorescence of the optic disc [Fig. 2]. There was prolonged P100 latency in the pattern visual-evoked potentials (Roland Consult RETIscan System, Wiesbaden, Germany). A diagnosis of NAION was made and the patient was given 1000 mg intravenous pulse methylprednisolone per day for 3 days, followed by 64 mg oral prednisolone. Two weeks postoperatively, the visual acuity improved to 20/40. Eighteen months after the surgery, optic disc was pale, visual acuity was 20/40 and there was nasal step in the visual field [Fig. 3].

Discussion

In our patient, the rise in IOP in the postoperative period was caused by CBS. It is known that CBS in the early postoperative period is developed by retained OVD behind the IOL and the occlusion of continuous curvilinear capsulorhexis opening by the optic of the IOL. It is proposed that the high osmolarity produced by the retained OVD in the capsular bag results in further accumulation of water inside the capsule.[4] Nd:YAG laser anterior capsulotomy allows the trapped OVD to pass into the anterior chamber and corrects the myopic shift and the anterior displacement of the IOL. In our patient, displacement of the retained OVD into the anterior chamber which prolonged the rise in IOP acted as an additional factor in the development of NAION.

Most of the anterior segment surgeons are familiar with CBS. On the other hand, NAION is a very rare postoperative complication of cataract surgery. The incidence of NAION after cataract extraction was reported to be 1 in 2000 surgeries. It was reported to be associated with high IOPs resulting in microvascular hypoperfusion and infarction of the optic nerve head.[3,5] Patients who develop NAION without documented IOP elevations are accepted to be associated with transient increases in IOP.[6] Although these studies document the pathogenesis of the development of NAION in our case, NAION after CBS has never been reported before.

Because of the limited publications about the treatment of

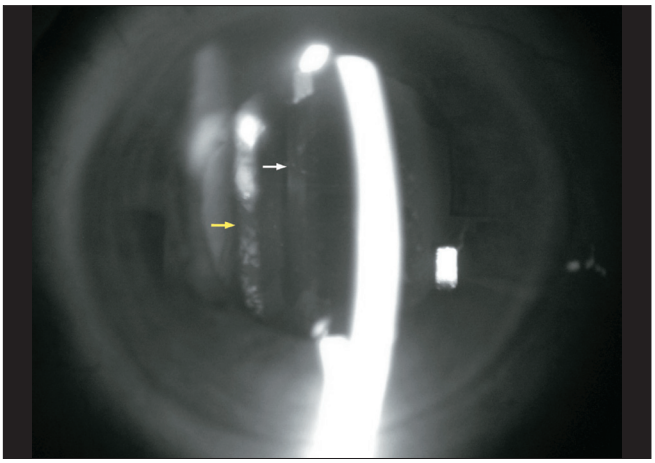

Figure 1: Slit-lamp photography demonstrates the posterior edge of the intraocular lens (arrow on the left) and distended posterior capsule (arrow on the right)

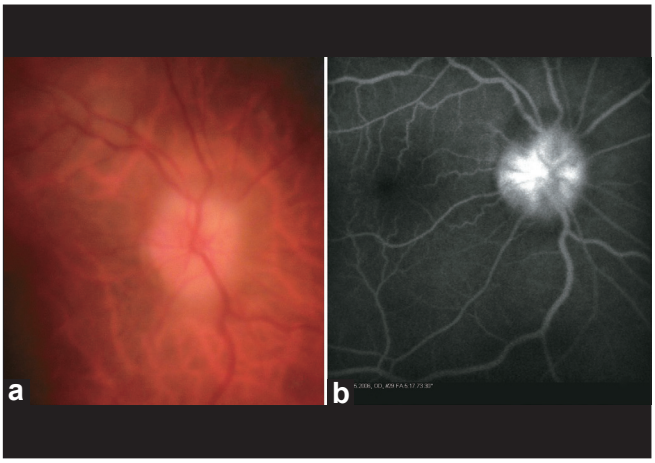

Figure 2: (a) Fundus photography shows nonarteritic ischemic optic neuropathy; (b) fluorescein angiography shows hyperfluorescence of the optic disc

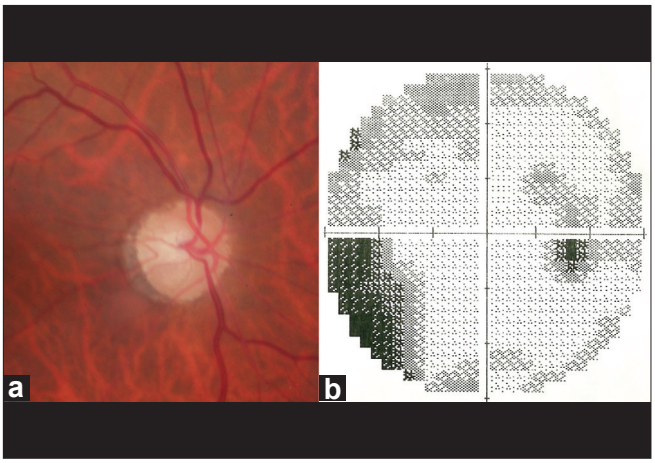

Figure 3: (a) 18 months postoperative fundus photography demonstrates optic disc pallor; (b) visual field test with nasal step

NAION after cataract surgery, we treated the patient like a spontaneously developed NAION. We preferred megadose intravenous pulse methylprednisolone therapy and the best corrected visual acuity was increased to 20/40, 14 days after the surgery. The final visual acuity at the end of the 18 months was unchanged despite pallor of the optic disc. We believe that further studies are needed to demonstrate the effect of pulse steroid therapy in postoperative NAION cases.

To conclude, we have described a patient who developed NAION after CBS. We believe that early control of IOP elevation in CBS will prevent the development of NAION after phacoemulsification. Anterior segment surgeons must be aware of the risk of developing NAION in cases with early postoperative CBC.

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