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**SHORT COMMUNICATION**

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**Case report**

# Sympathetic ophthalmia after complicated cataract surgery and intraocular lens implantation

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**PURPOSE.** *To report a case of sympathetic ophthalmia (SO) associated with cataract surgery and intraocular lens (IOL) implantation.*

**METHODS.** *Case report.*

**RESULTS.** *A 50-year-old man developed SO two months after complicated cataract surgery and IOL implantation. Adequate and prompt use of immunosuppressive medications and removal of the IOL by pars plana vitrectomy techniques resulted in control of the uveitis with significant visual improvement.*

**CONCLUSIONS.** *Sympathetic ophthalmia should be included as one of the devastating complications of IOL insertion. A high index of suspicion must be maintained whenever inflammation occurs in the fellow eye of an eye that has undergone intraocular surgery. (Eur J Ophthalmol 2001; 11: 193-6)*

**KEY WORDS.** *Sympathetic ophthalmia, Intraocular lens, Cataract, Complications*

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*Accepted: March 6, 2001*

## INTRODUCTION

Sympathetic ophthalmia (SO) is a bilateral granulomatous panuveitis that occurs after a penetrating injury that involves the uvea of one eye; it is produced by either trauma or surgery. The disease can begin as early as several days after the penetrating insult to decades later, the clinical diagnosis becoming apparent in 80% of cases within three months of injury to the exciting eye (1). The disorder was reported in 0.1% to 0.3% of patients after accidental trauma and in 0.015% after ocular surgery (2-5). A recent prospective population-based study found that ocular surgery, particularly retinal surgery, is a greater risk for SO than accidental trauma (6). Several studies indicated that the visual prognosis is good if diagnosis is made early and prompt, aggressive immunotherapy is started (6-8).

This report describes the development of SO after complicated cataract surgery with intraocular lens (IOL) implantation.

## Case Report

A 50-year-old man presented to the emergency room of King Abdulaziz University Hospital with the chief complaint of sudden loss of vision and severe pain in the left eye of four days duration. His ocular history was significant only for having had cataract extraction with implantation of an IOL in the right eye two months earlier. His medical history was unremarkable. The patient reported no visual improvement after cataract surgery in the right eye.

Examination revealed visual acuity of hand motions, with good projections in all quadrants in the right eye and counting fingers at 1 meter in the left eye. Intraocular pressure was normal in both eyes. Slit-lamp biomicroscopy of the right eye showed a well-healed limbal wound with iris and vitreous incarceration, mutton-fat keratic precipitates, fibrinous exudate in the anterior chamber, fibrous membrane covering the anterior hyaloid face, and a small

irregular pupil with posterior synechiae. The upper lens haptic was wrapped in fibrous tissue and adherent to the iris at the superior pupillary border (Fig. 1). The retina could not be visualized. Slit-lamp examination of the left eye showed 1+ flare and cells. There was a moderate number of vitreous cells.

Fundus examination of the left eye showed hyperemic disc, serous detachment at the posterior pole, and yellow-white lesions in the pigment epithelium at the posterior pole (Fig. 2). In fluorescein angiography these lesions obscured the background choroidal fluorescence during the early phases (Fig. 3) and stained during the late phases, with dye pooling under the neurosensory retina (Fig. 4). The optic nerve head showed leakage and stained in the later stages of angiography (Figs. 3 and 4). Ultrasonography of the right eye showed diffuse thickening of the retina-choroid layer, and the retina was attached.

The clinical diagnosis was sympathetic ophthalmia after complicated cataract surgery. The patient was immediately treated with oral prednisone 1 mg/kg/day, and cyclosporine A 5 mg/kg/day. Topical corticosteroids and cycloplegics were also given. On the tenth day after the start of treatment, the left eye showed a clinical response to treatment; visual acuity improved to 20/60, the posterior pole lesions faded, and the serous detachment resolved. At this time, the patient underwent right eye pars plana vitrectomy, removal of a pupillary membrane, release of the fibrous adhesion between the lower haptic of the implant and the inferior pars plana close to the ora serrata, injection of heavy liquid to float the IOL anteriorly, release of fibrous adhesion between the upper haptic and iris, and removal of the IOL after opening the old cataract wound. Two weeks after surgery, an inferior rhegmatogenous retinal detachment was noted due to oral dialysis. The retina was flattened using a scleral buckling procedure and gas tamponade.

The patient gradually had all medications tapered and discontinued four months after the start of treatment. Four months later, his best-corrected visual acuity was 20/80 in the right eye and 20/30 in the left eye. The intraocular pressure was 20 mmHg in the right eye and 18 mmHg in the left eye and the patient remained in sustained remission.

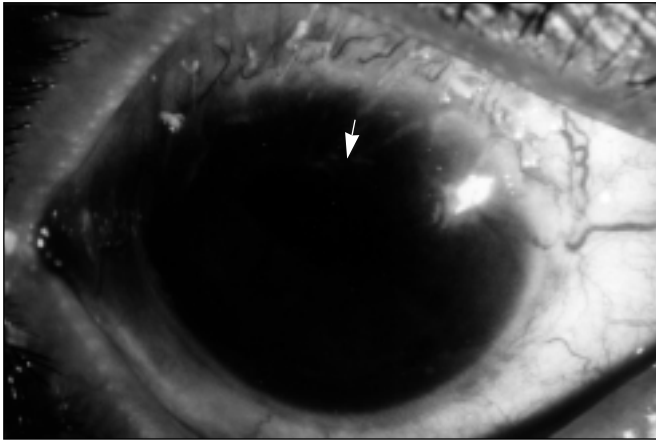
## DISCUSSION

Sympathetic ophthalmia is a rare bilateral granulomatous inflammation arising after penetrating injury to the eye. Although mostly seen after traumatic ocular perforation, it is a rare, but dreaded, complication of intraocular surgery. In two recent retrospective studies, ocular surgery was the sole cause of SO in 28% (7), and 17% (8) of the cases. A prospective population-based study found that ocular surgery, particularly retinal surgery, was the sole cause of SO in 56% of cases (6). Only two cases of SO after cataract extraction with lens implantation have been reported (9, 10).

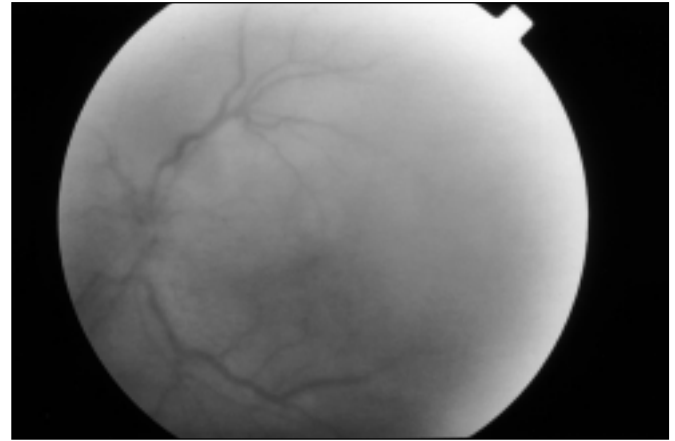
The eye is regarded as an immune privileged site with its blood-tissue barriers at the levels of the retinal vascular endothelium and the retinal pigment epithelium, and the lack of intraocular lymphatic drainage (11). Classically, the development of SO requires scleral perforation, allowing previously sequestered intraocular autoantigens access to conjunctival lymphatics and then to the regional lymph nodes, which subsequently induce an immunological reaction towards the previously tolerated antigens (12). Rao and colleagues reported that subconjunctival injection of retinal S antigen produced marked local inflammation, brisk antibody, and cell-mediated immune responses to S antigen, and sympathetic uveitis in the fellow eye, whereas the animals receiving intraocular injections had no immune responses to retinal S antigen, and none developed contralateral inflammation (12).

The exact pathogenesis of SO in the present case is not known. One may speculate that uveal incarceration at the cataract wound, a known immunogenic risk (13), exposed uveoretinal antigens to the conjunctival lymphatics, thereby inducing an immunopathological response. In addition, it is possible that the IOL had a major precipitating role. The haptics of the IOL were adherent to the iris and the ciliary body, disrupting uveal tissue and inducing a fibrinous reaction. Breakdown of the blood-ocular barrier induced by the IOL, with release of sequestered uveoretinal antigens, is an additional immunogenic risk. In one report, removal of the IOL relieved the uveitis which was not controlled with medical management (9).

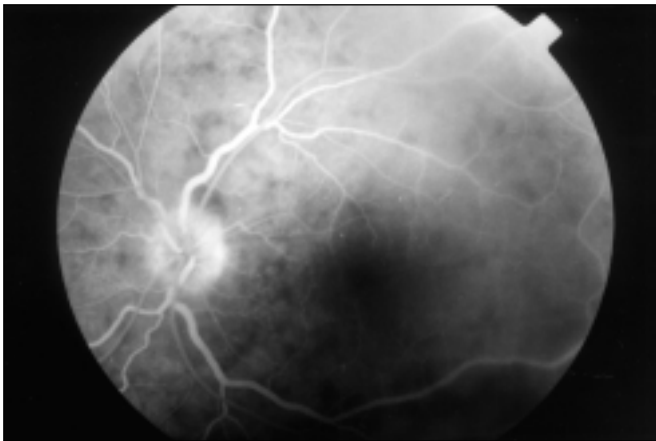
The visual outcome in the present case was good. The patient achieved visual acuity of 20/80 in the right eye and 20/30 in the left. This was presumably because



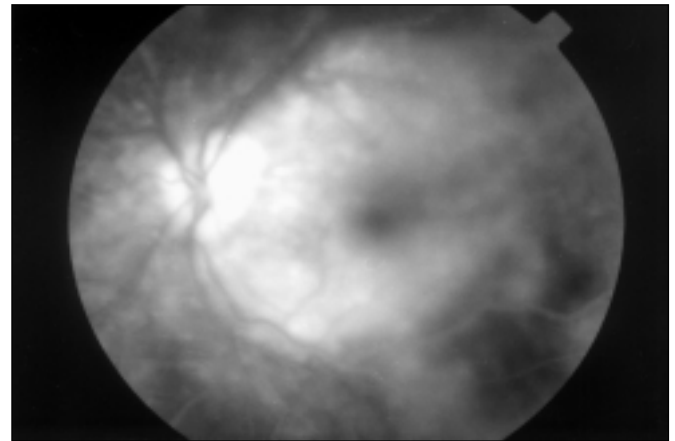
**Fig. 1** - Diffuse illumination photograph of the left eye, showing mutton-fat keratic precipitates, fibrous membrane covering the anterior hyaloid face, and small irregular pupil with posterior synechiae. The upper lens haptic is wrapped in fibrous tissue and is adherent to the iris (arrow).



**Fig. 2** - Fundus photograph of the left eye showing hyperemic optic disc, serous detachment at the posterior pole, and multiple yellow-white lesions in the pigment epithelium.



**Fig. 3** - Fluorescein angiogram, arteriovenous phase, shows blockage of background fluorescence by the lesions, and optic disc leakage.



**Fig. 4** - Fluorescein angiogram, late phase, shows staining of the lesions, dye pooling in subretinal fluid, and staining of the optic disc.

of the prompt and adequate systemic immunotherapy with prednisone and cyclosporine A. In other studies good visual outcome was associated with early, aggressive use of anti-inflammatory drugs (6-8).

Dalen-Fuchs nodules, a typical feature of the disease but not essential for diagnosis (6-8), were seen at presentation in this case. These are collections of lymphocytes, histiocytes, and altered pigment epithelial cells that lie just internal to Bruch's membrane (14). The clinical presence of these nodules may correlate with the severity of the disease (15). In the acute phase of SO, the fluorescein angiogram typically shows mul-

multiple hyperfluorescent sites of leakage. These sites generally correspond to the Dalen-Fuchs nodules (16). In the present case, the angiogram showed multiple hypofluorescent areas during the early phases that stained during the late phases, similar to those in acute posterior multifocal placoid pigment epitheliopathy. This pattern of early hypofluorescence with late staining has been reported (17). It is likely that the status of the pigment epithelium overlying the Dalen-Fuchs nodules determines the hyperfluorescent or hypofluorescent nature of these lesions on angiography.

## ACKNOWLEDGEMENTS

The authors thank Ms. Connie B. Unisa-Marfil for secretarial assistance.

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