Sir,

Spontaneous iris sphincter ruptures due to lens–iris diaphragm retropulsion syndrome during phacoemulsification

Cataract surgery, especially extracapsular cataract extraction (ECCE), has been known to be associated with pupillary abnormalities following surgery. Yap *et al.*¹ reported macroscopic and microscopic iris sphincter ruptures in 43.75% and 30.09% of patients respectively as the commonest type of pupillary abnormality following ECCE, while direct iris trauma in 5.3% attributed to direct mechanical damage was the commonest pupillary abnormality during phacoemulsification. Cataract surgery in a highly myopic eye has historically been associated with many unforeseen complications.^{2,3} We describe a hitherto unreported complication of spontaneous multiple iris sphincter ruptures associated with lens–iris diaphragm retropulsion syndrome (LIDRS; a term first described by Wilbrandt *et al.*²) during phacoemulsification in a highly myopic eye.

Case report

A 76-year-old man with a diagnosis of nuclear cataract and high myopia was listed for phacoemulsification and intraocular lens implant surgery in his right eye. Pre-operative refractive error was –10.25 DS/–0.75 DC in the right eye and –8.25 DS–0.1 DC in his left eye, while biometry had revealed axial lengths of 26.56 mm and 26.19 mm respectively. He underwent phacoemulsification surgery in his right eye in February 2000, with the Alcon Legacy 20000 system. Intraoperatively, frequent deepening of the anterior chamber with simultaneous dilatation of the pupil was noted especially with initiation of infusion; this was also associated with significant episodes of retrodisplacement of the lens–iris diaphragm. At the end of the apparently uneventful surgery, the presence of multiple small radial iris sphincter tears was noted in the pupillary margin by the surgeon (Fig. 1). The patient had a very good visual recovery, regaining Snellen 6/5 visual acuity in the operated eye. In addition there were no symptoms

Fig. 1. Post-operative photograph showing multiple iris sphincter tears in the pupil margin.

suggestive of glare and the patient did not report any cosmetic awareness of the defect. Pupil reactions were normal.

Comment

Although pupillary abnormalities are uncommon during phacoemulsification in routine patients, highly myopic eyes as in our patient predispose to LIDRS (a similar entity has been described in previously vitrectomised eyes in the literature^{2,3}). The pathophysiology of LIDRS in highly myopic eyes has been ascribed to zonular laxity, which is thought to be acquired by previous prolonged voluntary disuse of accommodation causing zonular spring exhaustion and/or elongation. The iris sphincter tears can be explained by the physical trauma due to sudden enlargement of the pupil caused by the infusion surge during phacoemulsification compounded by the lax zonules. The traditional method of managing this problem was by a substantial lowering of the infusion bottle height, which unfortunately also caused an undesirable reduction in infusion volume. This problem has been circumvented to some extent by the newer phacoemulsification probes which combine an undersized ultrasonic tip and oversized phacoemulsification sleeve, thereby increasing the infusion volume in spite of lowered bottle heights.^{2,4,5} However, as illustrated by our case, where such a phacoemulsification system was used, present phacoemulsification systems are not entirely infallible in preventing LIDRS and its consequent complications.

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Sir,

Retinal toxicity due to Efavirenz

Efavirenz is a non-nucleoside reverse transcriptase inhibitor recently licensed for the treatment of AIDS. The drug has proven efficacy in increasing CD4⁺ counts and

reducing HIV RNA plasma cells when combined with nucleoside reverse transcriptase inhibitors and/or protease inhibitors. The most common side-effects related to Efavirenz are headache, insomnia, fatigue and dermatological effects such as maculopapular rash.¹

We report a case of bilateral maculopathy in an HIV-positive patient which, we believe, was secondary to the use of Efavirenz.

Case report

A 37-year-old HIV-positive air steward was referred for an ophthalmological examination due to impaired vision in both eyes. He denied any previous ocular problem or family history. He initially received Nevirapine 200 mg b.d. but he developed a generalised rash and Nevirapine was discontinued. In March 1999, he received Efavirenz 600 mg o.d. with Combivir. His CD4⁺ count was 379 cells/mm³ and viral load 230 RNA copies/ml.

Three months after starting the new anti-HIV therapy he started to complain of blurred vision in both eyes without any other symptoms. On examination he had corrected visual acuities of 6/9 in both eyes without signs of anterior segment inflammation and normal intraocular pressures. Fundus examination of his left eye

showed only a small macular lesion, which appeared to be at the level of the retinal pigment epithelium (RPE). The fundus of his right eye was completely normal. Efavirenz was discontinued a week after this visit.

A month later (July 1999) the patient complained of deterioration in visual acuity. Visual acuities at this time were 6/60 in the right eye and 6/9 in the left eye. He had no signs of inflammation in either eye. Ophthalmoscopy showed an area of retinal opacification occupying most of his macula in both eyes (Fig. 1), with no abnormalities in the periphery. These lesions were primarily outer retinal, and involvement of the choriocapillaris was excluded by indocyanine green angiography (ICG), which showed normal perfusion of the inner choroid. Fluorescein angiography revealed a ring-like lesion in the

(a)

(b)

Fig. 1. (a), (b) Colour photographs of both fundi showing a large area of retinal opacification of the macular region.

(a)

(b)

Fig. 2. (a), (b) Fluorescein angiography of both fundi showing a ring-like lesion in the macular region, representing transmission of fluorescein through a defective retinal pigment epithelium (window defect).

macular region, compatible with a transmission defect at the level of the RPE in both eyes (Fig. 2). A visual field test (Humphrey 30-2) failed to reveal any specific abnormality. In September 1999, in spite of the similarity of the retinal findings, his visual acuities were 6/36 in the right eye and 6/9 in the left. His CD4⁺ count was 450 cells/mm³ and his viral load was 36 000 RNA copies/ml.

Two months later (November 1999) the macular lesions were unchanged, and despite the fact that Efavirenz had been discontinued for 5 months vision had dropped to hand movements in the left eye. Electrodiagnostic tests were performed, with the pattern and focal ERGs showing no response. The rod ERG and electro-oculogram were normal. The cone ERG was mildly delayed. The conclusion was of a profound bilateral maculopathy. The following baseline serological investigations were negative: toxoplasma serology, VDRL/TPHA, cryptococcal antigen and CMV IgM.

At his last assessment the patient presented the same changes in the macula and his visual acuities were 6/60 in both eyes.

Comment

Ocular manifestations in AIDS patients are classically related to low CD4⁺ counts, especially the HIV-related microangiopathy and the cytomegalovirus retinitis.² Our patient never presented with very low CD4⁺ counts, which makes the possibility of an opportunistic disease in his case very unlikely.

Few cases have been published regarding retinal drug toxicity in patients with AIDS. A well-established correlation between didanosine (DDI) and peripheral retinal changes has been shown. Didanosine toxicity is related to high doses of the drug and affects children more frequently; however, cases in HIV-positive adults have been reported.^{3,4} Didanosine lesions produce electroretinographic and visual field defects without changes in visual acuity because of their peripheral occurrence. Histologically DDI was shown to affect primarily the RPE with secondary damage to the neurosensory retina and to the choriocapillaris.⁵ Although no histopathological information was available, we can, based on the clinical appearance, the angiographic and electroretinographic findings, conclude that the damage in our patient was also primarily to the RPE.

As the other drugs used by the patient, including azathioprine and 3TC (Combivir), have been available for a long time and no cases of retinal toxicity have been associated with their use, we believe that retinal damage in this case was due to Efavirenz. Since the experience with Efavirenz is still limited it is possible that more cases will appear and physicians should be aware of this possible side-effect, which may have devastating effects on vision.

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Sir,

Angle closure risk from proprietary medicines

Acute angle closure glaucoma is uncommon in the Caucasian population with a prevalence of 0.1%. However, it must be diagnosed and managed quickly and effectively to prevent permanent visual loss.^{1,2} Pharmacological agents capable of causing mydriasis and cycloplegia can precipitate acute angle closure glaucoma in a predisposed patient with pre-existing narrow angles. These patients are usually unaware of their risk of angle closure glaucoma so that drug data sheets advising patients to 'speak to your doctor if you have glaucoma' are likely to go largely unheeded. The appropriate advice should be to stop the preparation and seek medical review if blurred vision is associated with significant eye pain, headache, or nausea and vomiting.

Case report

A 48-year-old man presented to eye casualty with a 3 day history of pain, redness and blurred vision of the right eye. In the previous 2 months he had experienced several episodes of ocular pain whilst watching television. There was no other significant history. For