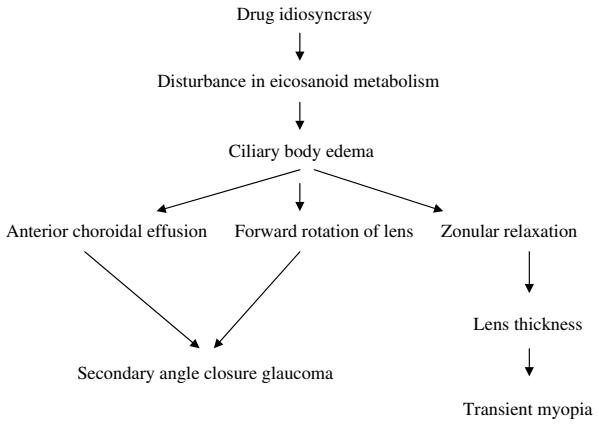

Figure 3 Flow chart for the mechanism of drug-induced secondary angle closure glaucoma.

and d) with clear cornea, normal AC depth and IOP of 18 mm Hg in each eye (Figure 1c and d). He achieved final best-corrected visual acuities of 6/6.

Comment

Acetazolamide-induced transient myopia and angle closure glaucoma has been reported in nonglaucomatous patient.[1,2,6] Forward shift of crystalline lens caused by ciliary body oedema has been postulated as the mechanism.[1,2,5] Ciliary body oedema has been based on an imbalance in eicosanoid (prostaglandin–thromboxane) metabolism (Figure 3).[5]

In this patient, the cloudy cornea precluded laser iridotomy and cycloplegia was avoided as angle closure was a differential diagnosis. Dexamethasone was used empirically as an inflammatory aetiology was suspected.

The failure of laser iridotomy and cycloplegia was reported as the mechanical effect following forward rotation of ciliary body and displacement of iris–lens diaphragm secondary to choroidal effusion.[6] Only when the latter resolved could the angle be opened. Corticosteroids have been shown to cause choroidal effusion[3,4] and are ineffective in managing this complication. Other methods of controlling IOP might have been considered to avoid this unusual complication.

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Eye (2007) **21,** 870–872; doi:10.1038/sj.eye.6702741;
published online 9 February 2007

Sir,

Angle-closure triggered by orgasm: a new provocative test?

Case

Sexual arousal has been reported to lead to angle-closure glaucoma[1] and to blurred vision in women with narrow angles.[2] We treated a 34-year-old Caucasian woman with uniocular pain, blurred vision, and haloes since 2 days. She had a past history of recurrent episodes occurring immediately after orgasm, resolving spontaneously and that occurred only when she was having sex in the dark while in the top position, with her upper torso prone. Her mother and maternal grandmother had had angle-closure.

On examination, best-corrected acuity was 20/25 OU with +5.00 sph +1.00 cyl × 90 OD and +5.25 sph +1.75 cyl × 85 OS. Intraocular pressure was 12 mm Hg OD and 17 mm Hg OS. Gonioscopy revealed plateau iris configuration OU and appositional angle-closure OD with scattered peripheral anterior synechiae and a slit angle OS. Axial lengths were 20.72 mm OD and 20.38 mm

OS. Anterior chamber depths were 3.06 mm OD and 3.01 mm OS. Lens thicknesses were 4.31 mm OD and 4.35 mm OS. Symptoms responded to laser iridotomy and peripheral iridoplasty OU with widening of the angles.

Comment

Provocative testing for angle-closure includes dark room, prone, and mydriatic tests. The prone test causes anterior displacement of the lens and pupillary block. On prone position UBM (Figures 1 and 2), eyes exhibit shortening and an increase in iris thickness associated with greater anterior convexity.[3]

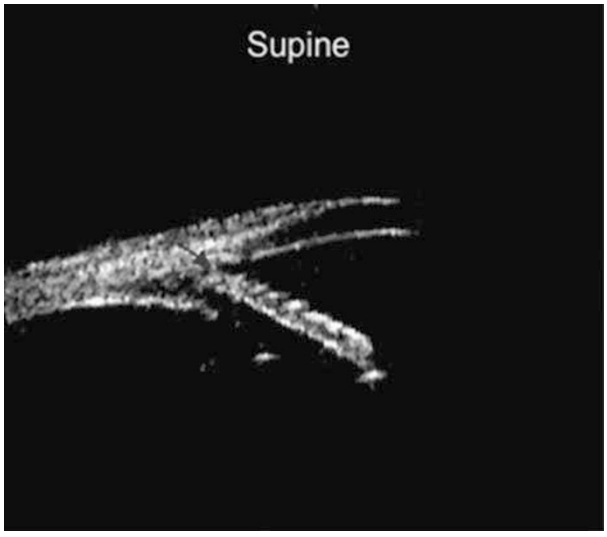

Figure 1 UBM image of superior angle in supine posture during dark condition from a normal subject. Arrow indicates scleral spur.

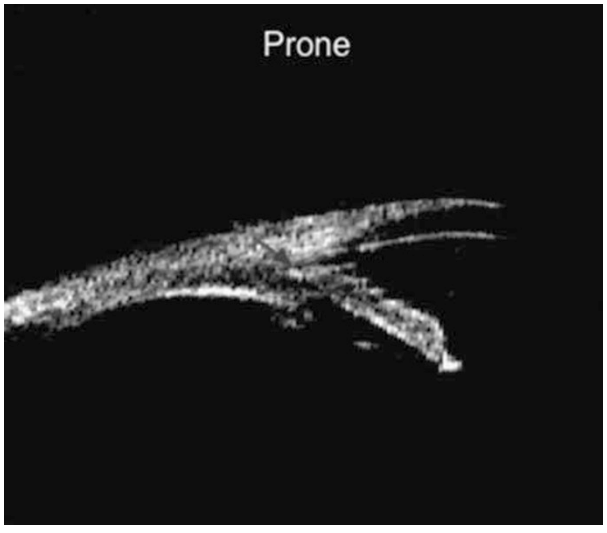

Figure 2 UBM image of superior angle in prone posture during dark condition from a normal subject. Note the apposition of peripheral iris to cornea with thickening of the peripheral iris. Arrow indicates scleral spur.

Activation of the sympathetic and parasympathetic nervous systems can affect mydriasis.[4] Sphincter muscle stimulation and pupillary dilation in the dark cause centripetal and posterior forces, respectively, increasing iridolenticular apposition.[5] Parasympathetic-induced ciliary contraction causes zonular relaxation, anterior lens movement, increased lens thickness and curvature, and pupillary block. Increased iris thickness, owing to sympathetic stimulation of the iris dilator muscle and its resulting circumferential folding, may prompt angle-closure if a narrow angle is present.

Our patient was a hyperope with short globe and normal anterior chamber depth and lens thickness. Both the dark room and orgasm caused pupillary dilation, with angle crowding and iridotrabecular apposition, while the prone position presumably led to anterior lens movement. The combination of these three factors triggered angle-closure.

Acknowledgements

This work was supported by the Joseph and Geraldine LaMotta Research Fund of the New York Glaucoma Research Institute, New York, NY, USA.

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Eye (2007) **21,** 872–874; doi:10.1038/sj.eye.6702744; published online 16 February 2007

Sir,

Scleral granuloma associated with presumed diffuse immune lymphocytosis syndrome

Diffuse infiltrative lymphocytosis syndrome (DILS) is a rare manifestation of human immunodeficiency virus (HIV) infection. It is characterised by HIV seropositivity, a $CD8^+$ lymphocytosis, visceral gland infiltration with $CD8^+$ lymphocytes, bilateral parotid gland swelling, xerostomia, and xerophthalmia.[1] Peripheral neuropathy, interstitial pneumonitis and a granulomatous panuveitis have also been described in association with this condition.[2] We present a case of scleral granuloma occurring in the presence of presumed DILS.

Case report

A 10-year-old girl of Congolese origin, who was HIV positive, gave a 1-month history of a red painful right eye. Examination revealed a 15 mm × 15 mm dark scleral nodule above the cornea which was associated with locally injected scleral feeding vessels, as shown in Figure 1. Anterior segments were otherwise unremarkable but dilated fundoscopy revealed a bilateral periphlebitis, with no haemorrhage or neovascularisation.

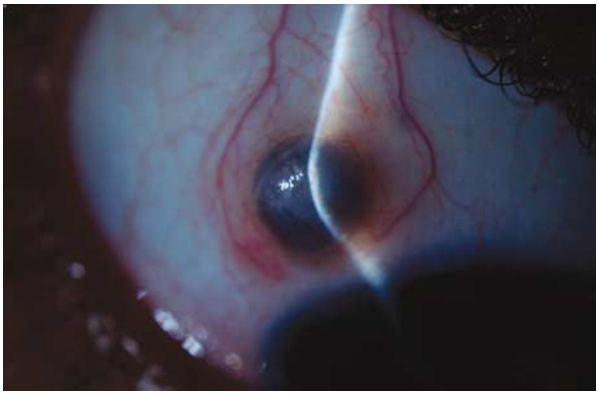

Figure 1 Scleral nodule with surrounding vascularisation.

Systemic examination showed her to be of short stature and to have tinea capitis and tinea unguis, cervical and axillary lymphadenopathy, and bilateral parotid gland enlargement. Serologic analysis revealed absolute $CD4^+$ cell count of 0.38×10^9/l (normal range, $0.30–2.0 \times 10^9$/l), absolute $CD8^+$ cell count of 1.04×10^9/l (normal range, $0.30–1.80 \times 10^9$/l), and peripheral blood film $CD8^+$ positive percentage raised at 47% (normal range, 19–34%). Full blood count was otherwise normal but C-reactive protein was raised at 26 mg/l. Chest radiography findings were consistent with lymphocytic interstitial pneumonitis. Serology for syphilis, cytomegalovirus, and hepatitis B and C showed neither active disease nor previous infection. Measles serology indicated that she had had previous exposure only. Serum angiotensin-converting enzyme (ACE) level was normal.

She was commenced on a course of topical dexamethasone and highly active antiretroviral therapy (HAART) at the following doses: abacavir 450 mg o.d., lamivudine 200 mg o.d., and efavirenz 300 mg o.d. Over 4 months, the scleral granuloma became smaller, less locally injected and the eye comfortable. Subsequent $CD8^+$ positive percentage improved sequentially to 37%.

Comment

A presumptive diagnosis of DILS is likely given this patient's HIV seropositivity, relative $CD8^+$ lymphocytosis (although this could be due to a relatively lower $CD4^+$), bilateral parotid gland enlargement, and normal ACE. Bilateral panuveitis, retinal periphlebitis, and lacrimal gland involvement are recognised ocular associations in DILS,[2–4] however, a literature search shows no previous report of scleral granuloma occurring in the presence of this syndrome. We appreciate that there was no absolute lymphocytosis, only a reversal of the $CD4^+ : CD8^+$ ratio. All cases reviewed in the existing literature have shown absolute lymphocytosis, with numbers much higher than those seen in our patient.

It may be argued that positive tissue diagnosis was required to confirm DILS to show local $CD8^+$ infiltration and to exclude granulomatous pathology, especially given the presence of retinal periphlebitis. However, a negative Mantoux test and a normal chest radiograph and serum ACE excluded both tuberculosis and sarcoidosis. Furthermore, it was deemed inappropriate to subject a 10-year-old girl with resolving parotid swelling to a tissue biopsy. Although typing was not performed in our patient, HLA DR11 has been shown to have a higher association with this condition.[5]

Although a diagnosis of scleral staphyloma with uveal prolapse was considered, in which case ultrasonic biomicroscopy would have been useful, we thought it