Two factors are likely to be responsible for this patient's disc new vessels. The fluorescein angiogram suggested retinal ischaemia due to retinal vasculitis. Hypoxia stimulates the production of vascular endothelial growth factor (VEGF) by retinal cells as a prelude to overt neovascularisation.[7]

Inflammatory processes can also induce new vessel formation independent of ischaemic vasculitis. The most potent mediators are thought to be metabolites of arachidonic acid, in particular prostaglandins E_1 and E_2.[8,9] Cyclo-oxygenase inhibitors, which inhibit the production of these prostaglandins, reduce new vessel formation.[10,11]

Steroids and cyclosporin decrease the liberation of inflammatory mediators and subsequently improve retinal perfusion, inhibiting ischaemic and prostaglandin-induced neovascularisation. Furthermore, it has been recognised for some time that corticosteroids have an anti-angiogenic effect in active neovascularisation which appears to be independent of their anti-inflammatory activity.[12]

In summary, bilateral vitreous haemorrhage and headache is not pathognomonic of Terson's syndrome. It is suggested that a thorough search for underlying ocular inflammatory disease should be made in cases of apparently 'idiopathic' retinal neovascularisation. Ideally assessment should include fluorescein angiography and ultrasound biomicroscopy where available.

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Sir,

Paroxetine and acute angle-closure glaucoma

Acute angle-closure glaucoma has been associated with tricyclic antidepressants and monoamine oxidase inhibitors. In susceptible patients it would appear sensible to use an anti-depressant with weak anticholinergic activity and the use of selective serotonin re-uptake inhibitors (SSRIs) – now widely prescribed – seems an appropriate choice of drug. However, paroxetine (an SSRI) has been implicated recently in three separate case reports of acute angle-closure glaucoma.[1–3] We report a further case of unilateral acute angle-closure glaucoma related to paroxetine.

Case report

A 53-year-old woman attended the eye clinic with a history of left ocular pain, browache and blurred vision for 1 day. There was congestion of the conjunctiva, shallow anterior chamber and the pupil was mid-dilated with a high intraocular pressure of 61 mmHg on Goldmann tonometry (normal value ⩽21 mmHg). The patient's spectacle prescription was [R] +6.50/+1.00 160 and [L] +7.00/+1.00 30; she had worn glasses for hypermetropia since the age of 5 years. A diagnosis of acute angle-closure glaucoma was made and she was admitted for standard medical treatment. Nd:Yag laser peripheral iridotomies were performed to both eyes and she made an unremarkable recovery with normal intraocular pressure measurements off all topical treatment. A similar, but less severe episode had occurred 6 months previously when she had been prescribed paroxetine 20 mg once daily. She had stopped the medication herself and the symptoms had resolved spontaneously. Interestingly, she had re-commenced the same medication just 3 days prior to her later presentation with acute angle-closure glaucoma.

Comment

Incomplete mydriasis is the trigger for acute angle-closure glaucoma in susceptible eyes and hence dim ambient lighting conditions and the use of drugs with anticholinergic or adrenergic properties (e.g. bronchodilators and tricyclic antidepressants) have been implicated in its aetiology. Three previous case reports[1–3] describe a link between the use of paroxetine and the onset of acute angle-closure glaucoma in patients whose ages ranged from 70 to 91 years. In only one report was the patient's refraction referred to qualitatively as hypermetropic. The patient in our case was a more typical age for suffering an acute angle-closure glaucoma

attack and had good quantitative evidence of refractive status. A common feature of all reports is the onset of symptoms within a few days of commencing paroxetine. The Committee on the Safety of Medicines reports side-effects possibly related to paroxetine as acute angle-closure glaucoma, mydriasis and blurred vision (personal communication).

Iris sphincter muscle in rabbits has been shown to possess receptors to serotonin which produces a relaxation of the constricted muscle in a dose-dependent manner.[4] It is not inconceivable that the human sphincter pupillae has similar receptors, and hence the use of a SSRI could allow the local build-up of serotonin and hence mydriasis.

To the authors' knowledge, this is the first reported case of a patient describing a clear association in time with the use of paroxetine on two separate occasions. Although mydriasis in overdose is noted, neither the British National Formulatory nor the datasheet for paroxetine cite glaucoma as a potential side-effect in susceptible patients. Perhaps the time has come for this to be addressed.

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Sir,

Treatment of recurrent hordeolum with Broncasma Berna

Recurrent hordeolum is an eyelid problem.[1,2] This letter reports on the results of using a vaccination in its treatment.

The author has developed a method that uses an inactivated bacterial vaccine (Broncasma Berna) in the treatment of recurrent hordeolum and has clinically experienced 11 years of effective treatment. Broncasma Berna, which has already been used for the treatment of chronic bronchitis and its complications, asthma[3] of bacterial origin, chronic sinusitis and nasal allergies[4] of bacterial origin, has few or no side-effects.

For this study, 25 patients who suffered from attacks of recurrent styes, despite treatment with warm compresses, surgery, antibiotics or local corticosteroid injections, were selected. Formal consent was obtained from each patient. No patient had diabetes or neoplasm (meibomian gland carcinoma). During the period from 1985 to 1996, each of the patients (16 women, 9 men; median age 32 years) was given a subcutaneous injection of 0.05 ml of Broncasma Berna once every 4–14 days for a total of 4–10 injections. The average frequency of recurrent styes was six times per year. Broncasma Berna was given once every 5 days and an average of 5 times. Other medications for styes were not used. One year and a half after the completion of treatment, each patient was checked for recurrent styes. Of the 25 patients treated, the author confirmed that 21 (84%) of the patients no longer had recurrent styes at that time. A double-masked study was not carried out on ethical grounds; placebo treatment would not alleviate an attack of styes.

Broncasma Berna is a product of the Swiss Serum and Vaccine Institute Berne. A 1 ml quantity of Broncasma Berna is composed of the following: 50 10^6 pneumococcus types I, II and III; 40 10^6 streptococci; 500 10^6 staphylococci; 60 10^6 *Neisseria catarrhalis*; 20 10^6 *Gaffkya tetragena*; 250 10^6 *Pseudomonas aeruginosa*; 40 10^6 *Klebsiella pneumoniae*; 40 10^6 *Haemophilus influenzae*; conservans (maximum 0.4% phenol). The specimen scheme for the dosage is as follows: first to fifth injections: 0.1 ml, 0.3 ml, 0.5 ml, 0.7 ml and 1 ml; then, after an interval of 1 week, five injections of 1 ml. In these trials, however, only 0.05 ml of Broncasma Berna was used.

Although Broncasma Berna has been used in many countries for three decades and millions of doses have been administered, no anaphylactic reactions or other significant complications have ever been reported to the Swiss Serum and Vaccine Institute Berne. Broncasma Berna has been used in Japan for a quarter of a century, and no significant side-effects have ever been reported. As the dose used was relatively small, none of the patients complained of spontaneous pain or fever, although two complained of slight tenderness at the injection point on the following day.

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