adults.[2] Brimonidine lowers IOP through a dual mechanism of action: it reduces aqueous humour production and increases uveoscleral outflow.[3] Significant systemic absorption of brimonidine eye drops occurs in adults, peak plasma concentrations occurring within 1–4 h with a half-life of approximately 2.5 h.[2] Systemic metabolism of brimonidine is primarily by the liver and urinary excretion is the major route of elimination of the drug and its metabolites.[2] Brimonidine, like most drugs, has not been formally tested on children but has, along with other side-effects, caused drowsiness in adults.[4] There have been several unpublished reports of floppy episodes (J.A. Bradbury, Bradford Royal Infirmary, personal communication) and severe life-threatening reactions to brimonidine drops in infants (Allergan, USA, personal communication). The marked sensitivity of infants to brimonidine may be due to their size, immature metabolism and excretion of drugs or an increased receptor sensitivity. To our knowledge this is the first published report of a severe adverse reaction to brimonidine in an infant.

This infant presented with spontaneous hyphaema and raised IOP. The most common cause for hyphaema in any age group is trauma.[5] Non-accidental injury should be excluded in any infant presenting in this way. The most frequently presenting sign in iris juvenile xanthogranuloma is hyphaema, although other causes should be sought.[5] Once the diagnosis of iris juvenile xanthogranuloma is established treatment should be initiated rapidly. Several modes of treatment have been advocated including topical steroids,[5] subconjunctival steroids,[6] systemic steroids[7] and radiotherapy.[8] Not all cases resolve with local or systemic steroids and treatment should be adjusted according to clinical status.[5] If hyphaema and raised IOP is present, as in our case, aggressive antiglaucoma medication with eye drops and oral acetazolamide, if necessary, should be instituted.[5]

In summary, this infant's eye with presumed juvenile xanthogranuloma was controlled medically with topical steroids and with topical and systemic anti-glaucoma medication. However, the child has been left with dense amblyopia and a lens opacity. This case illustrates the potential pitfalls in prescribing adult drops, especially some of the newer anti-glaucoma medications, in infants and we would recommend that great care be taken when using these drops in infants and children.

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Sir,

Hormone replacement therapy and retinal vein occlusion

The use of sex hormone preparations is a risk factor for cardiovascular and cerebrovascular disease.[1–3] A number of isolated case reports and a recent hospital-based retrospective study of retinal vein occlusions have implicated the oral contraceptive pill (OCP) but not hormone replacement therapy (HRT) as an independent risk factor for retinal vein occlusion.[4] However, as an increasingly large proportion of the population are using HRT, reporting cases of retinal vein occlusion in patients on this therapy may have management implications.[4] We report a case of central retinal vein occlusion in a patient on HRT.

Case report

A 43-year-old woman presented with a 3 day history of reduced visual acuity. There was no previous ophthalmic history. This patient had started HRT comprised of low-dose oestrogen and progesterone 5 weeks prior to presentation to alleviate menopausal symptoms. Of note was that she smoked 20 cigarettes a day, but there was no other significant medical or family history.

On examination visual acuity was 1/60 in the right eye and 6/18 in the left. There was no relative afferent pupillary defect and intraocular pressures were normal. Fundal examination demonstrated in the right eye a swollen optic disc, tortuous retinal vessels and multiple intraretinal haemorrhages consistent with a diagnosis of central retinal vein occlusion. The left eye was normal. Fluorescein angiography did not demonstrate any signs of retinal ischaemia. A comprehensive medical examination revealed no abnormalities and the patient was normotensive (125/80 mmHg). A range of haematological (including a thrombophilia screen),

Table 1. *Hormone replacement therapy and retinal vein occlusion: summary of haematology, biochemistry and immunology data*

Investigation	Patient value	Normal values
Haematology		
Full blood count (FBC)		
Haemoglobin	14.7 g/dl	12.0–18.0 g/dl
White cell count	8.7×10^{-9}/l	$4.0–11.0 \times 10^{-9}$/l
Erythrocyte sedimentation rate (ESR)	12 mm/h	< 20 mm/h
C-reactive protein (CRP)	< 0.1 mg/ml	< 0.6 mg/ml
Plasma viscosity	1.72 mPas	1.52–1.72 mPas
Thrombophilia screen		
Activated protein C resistance ratio	2.9	2.1–3.7
Activated protein C resistance ratio (factor V modified)	3.6	2.1–3.8
Prothrombin time (PT)	14 s	13–17 s
Activated partial thromboplastin time (APPT)	37 s	26–39 s
Dilute Russell viper venom time	40 s	29–51 s
Lupus anticoagulant	None detected	None
Protein C	100%	70–150%
Protein S	101%	65–140%
Antithrombin III	89%	80–120%
Factor VIII	81%	50–150%
Fibrinogen	2.8 g/l	1.5–4.0 g/l
Biochemistry		
Fasting blood glucose	6.0 mmol/l	3.5–6.0 mmol/l
Fasting cholesterol	5.4 mmol/l	3.1–6.5 mmol/l
High-density lipoproteins (HDL)	1.2 mmol/l	0.8–2.0 mmol/l
Low-density lipoproteins (LDL)	3.6 mmol/l	< 4.5 mmol/l
Fasting triglycerides	1.3 mmol/l	0.5–1.5 mmol/l
Immunology		
Rheumatoid factor (Latex)	< 20	< 20
Autoantibodies		
Anti-nuclear	< 40	< 40
Anti-parietal	< 40	< 40
Anti-mitochondrial	< 40	< 40
Anti-smooth muscle	< 40	< 40
Anti-reticulin	< 40	< 40

biochemical and immunological parameters were normal (Table 1). The patient was advised to stop smoking and the HRT was discontinued on the advice of the gynaecology service.

Four weeks later at routine follow-up rubeosis iridis was noted and, although there was no clinical finding of ischaemia, repeat fluorescein angiography demonstrated retinal neovascularisation. Despite full treatment with panretinal photocoagulation at the 7 month follow-up visual acuity in the right eye is no perception of light, intraocular pressure remains elevated at 45 mmHg and there is total optic atrophy. The patient is no longer smoking or on HRT.

Comment

The use of the oral contraceptive pill is a risk factor for cardiovascular disease including central retinal vein occlusion.[1–4] A large number of factors have been implicated in the aetiology of central retinal vein occlusion with a number of proposed mechanisms of action.[5] Furthermore the presence of a number of risk factors strongly augments their effects when coexistent.[5]

The role of HRT in the aetiology of central retinal vein occlusion is not clear although the number of reported cases is small. Sex hormone effects on the cardiovascular system are dose dependent and compared with the OCP, the hormonal dose is small in HRT. However, the combination of HRT and smoking, as in the patient reported here, may have augmented their effects as risk factors. While patients on HRT are usually older than those on the OCP and age itself may predispose to the development of retinal vein occlusion, the patient reported here was relatively young.

A recent report has highlighted the need to report the rare cases where HRT is implicated in retinal vein occlusion as an increasingly large number of the population are using this medication.[4] While HRT when used in isolation is relatively safe, prescribing HRT in older patients with other known risk factors for cardiovascular disease such as smoking and hypertension may result in an increasing incidence of reported side-effects, including central retinal vein occlusion.

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Sir,

Ischaemic retinal vasculitis in biopsy-proven sarcoidosis

Retinal periphlebitis is a well-recognised feature of ocular sarcoidosis, but ischaemia and vascular occlusions are only rarely reported. We present a case of biopsy-proven sarcoidosis with ischaemic retinal vasculitis.

Case report

A 47-year-old man presented in 1993 after 1 month of blurred vision in both eyes. He also had a history of malaise, tiredness, loss of weight and non-productive cough of 9 months duration. Examination showed a visual acuity of 6/9 in each eye. There was a granulomatous anterior uveitis, vitreous cells and snowballs, and multiple retinal perivenous infiltrates. On investigation abnormal results included a raised serum angiotensin converting enzyme (ACE) at 217 IU/ml, raised γGT and AST levels, and a chest radiograph which revealed bilateral hilar lymphadenopathy and increased interstitial markings. A transbronchial biopsy showed non-caseating granulomata consistent with a diagnosis of sarcoidosis. The patient was treated with topical steroids and systemic prednisolone, the latter commencing at 40 mg/day and reducing according to disease activity. Substantial improvement ensued and systemic treatment was discontinued by the end of 1996, at which time the minimal uveitis was well controlled using only topical steroids. The visual acuity was 6/6 in both eyes.

In 1998 the patient presented urgently with sudden blurring of vision in the right eye. Examination showed a visual acuity of 6/6 in each eye. There was no afferent pupillary defect. Colour vision was marginally reduced in the right eye, the patient being able to read 19/21 Ishihara plates. The left eye was entirely normal. The right anterior segment was uninflamed but there was active vitritis and some areas of substantial perivenous exudate. There were multiple small poorly defined pale retinal lesions at the posterior pole. Reactivation of sarcoidosis was presumed and prednisolone 80 mg/day was commenced. The visual acuity fell to 2/60 within 3 weeks, the optic disc becoming hyperaemic and cotton-wool spots becoming evident at the posterior pole (Fig. 1). There were no retinal haemorrhages. A mild afferent

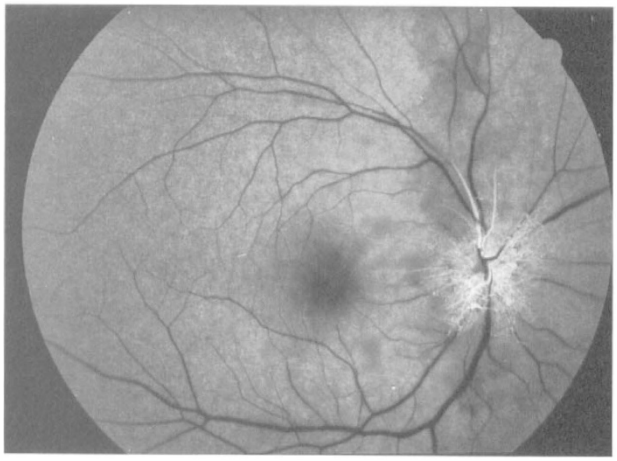

Fig. 2. *The onset of the arterial phase fluorescein angiogram (10.2 s) shows filling of optic nerve head vessels prior to complete choroidal filling.*

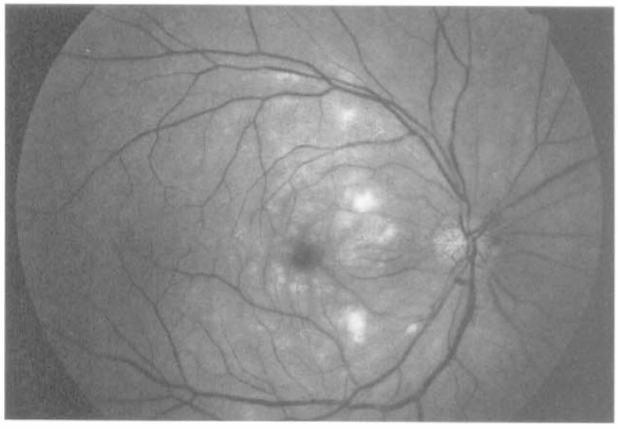

Fig. 1. *A red-free photograph of the right posterior pole shows multiple cotton-wool spots and minor calibre changes to the superotemporal venous circulation.*

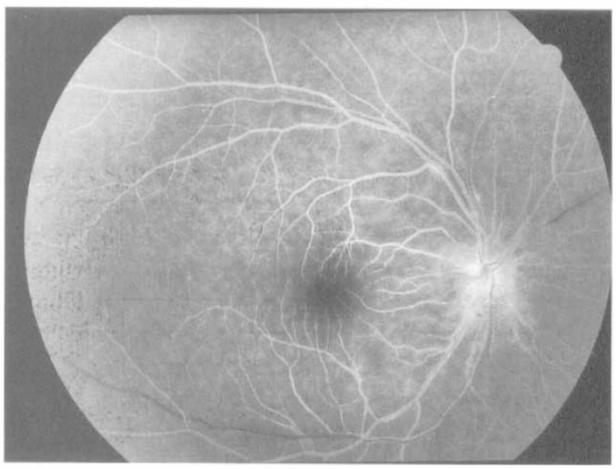

Fig. 3. *A late phase angiogram (70 s) shows that venous filling remains incomplete. Two areas of phlebitis are seen in the superotemporal vessels. There is some leakage from the optic disc.*