reduction in size of the tufts that had bled, were observed 6 months after retinal laser photocoagulation in the absence of similar changes in the other tufts. These were most probably signs of involution induced either by the retinal photocoagulation or by the bleeding, which have not been reported before.

We conclude that spontaneous hyphaema from pupillary vascular tufts may indicate a recent onset of a retinal venous occlusion and that their presence can be a risk factor for the development of hyphaema during the acute stage of an ischaemic retinal venous occlusion. Also, we believe that, as much as the ischaemia could lead to iridal vascular tuft engorgement, bleeding from the tufts or retinal laser photocoagulation of the ischaemic retina might initiate their involution.

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Sir,

Cytomegalovirus in aetiology of Posner–Schlossman syndrome: evidence from quantitative polymerase chain reaction

Posner–Schlossman syndrome (PSS) (glaucomatocyclitic crisis) was characterized in 1948 by recurrent episodes of hypertensive iridocyclitis.[1] The aetiology is unknown but current theories favour an infective origin especially herpes simplex virus (HSV).[2] We present a case in which CMV DNA was identified following an acute relapse of PSS.

Case report

A 35-year-old immunocompetent Chinese man with a history of recurrent bilateral Posner–Schlossman syndrome (PSS) presented with acutely raised intraocular pressure (IOP) in the right eye. Prior to this, IOP was controlled with topical brimonidine to both eyes. He was diagnosed with a relapse of PSS and treated

with topical steroids and latanaprost. After 1 week, the left eye suffered a similar relapse. Simultaneously, the right eye developed stromal oedema in the inferior cornea with anterior uveitis and the appearance of keratitic precipitates in a linear pattern typically seen in herpetic corneal endotheliitis (Figures 1 and 2). Oral acyclovir was commenced. The IOP in the left eye remained recalcitrant despite maximal topical therapy including latanaprost, timolol, and oral acetazolamide 250 mg qid for control, eventually requiring Ahmed tube implant surgery.

The endotheliitis in the right eye waxed and waned with topical medications but failed to resolve completely. After about 5 weeks, an anterior chamber paracentesis was performed. Aqueous humour sent for polymerase chain reaction (PCR) was strongly positive for cytomegalovirus (1.5×10^6 copies/ml) but negative for HSV and varicella zoster virus (VZV). The PCR assay for the CMV DNA quantitation was based on the fluorescent resonance energy transfer (FRET) principle and had two primers and two probes. The sense primer had the sequence of 5′-CACTTCGGGGTCGCAAT and the antisense primer of 5′-CGGGTATCAACAACAGCAAGGA. The hybridization probes were the fluorescein-tagged 5′-GTTAAGGCTGCGTTCCACACCGT-(FL) and the LC Red 640-tagged 5′-LCRed640-CCCGAAAAGTA GCCGATCTG. The assay was performed on the Light Cycler™ real-time PCR system (Roche Molecular Diagnostics). A concurrent blood serology for CMV IgM was *negative,* but IgG was positive. Complement fixation antibody was not significant at 1 : 16. Complete blood count (CBC) showed normal total white and differential counts with no lymphocytosis. The patient was otherwise well and apyrexial with no systemic symptoms throughout the whole period of follow-up.

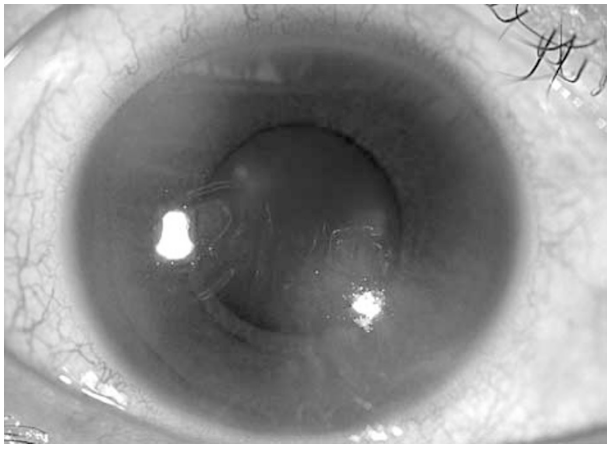

Figure 1 Marked inferior corneal stromal oedema consistent with clinical herpetic endotheliitis.

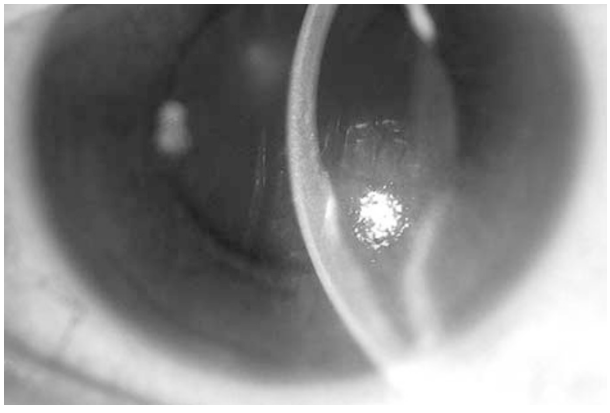

Figure 2 High magnification showing linear keratitic precipitates demarcating superior border.

Discussion

There are numerous postulations for the pathogenesis of Posner–Schlossman including abnormal vascular reactivity, autonomic dysregulation and infection.[2–5] Infective agents that have been most commonly implicated are HSV and CMV via immunologic response or direct infection. Bloch-Michel *et al*[2] postulated the role of CMV from detection of antibodies in aqueous humour, but Yamamoto[3] demonstrated only HSV DNA by PCR in patients during acute relapses of PSS. We present a case where acute relapse is associated with CMV detected by quantitative PCR. As far as we are aware, this has not been previously reported. These findings support an infective trigger and suggest that PSS may represent a spectrum of anterior segment manifestations of the herpesviridae family including HSV and CMV.

Evidence of an infective origin is suggested by the presentation of keratouveitis with endotheliitis, features that are reminiscent of classic 'herpetic disciform-like keratitis', soon after the acute relapse of PSS. The simultaneous and consecutive occurrence of inflammatory endotheliitis with onset of acute hypertensive uveitis compatible with relapse of PSS suggests a similar aetiological agent.[6] We postulate that CMV, possibly a latent reactivation, was a trigger inciting an immunologic response responsible for both presentation of corneal endotheliitis and acute trabeculitis resulting in an intractable glaucoma. It has been shown that in relapse of PSS, HSV may cause trabeculitis resulting in elevation of IOP and corneal endotheliitis, possibly due to viral reactivation via the trigeminal nerve (V1).[2,7] It is likely that a similar mechanism for CMV reactivation accounted for our observations in this case.

Keratitic precipitates (KPs) in PSS are typically described as medium- to large-sized and stellate, a

feature which is almost pathognomonic. Walter *et al*[8] also demonstrated similar stellate KPs in CMV uveitis due to fibrin deposition around inflammatory cells. Under specular microscopy in patients with PSS, Pillai *et al*[9] also observed a similar fibrin deposition around an individual KP resulting in a large conglomeration, giving rise to the classical 'stellate' appearance. These similarities further support a common aetiology between PSS and CMV or herpetic keratouveitis.

Conclusion

We postulate that PSS is not a distinct entity but may represent a spectrum of inflammatory responses to members of the herpesviridae family including CMV and HSV. An acute relapse may present with classic hypertensive cyclitis with or without other clinical manifestations of anterior segment inflammation indistinguishable from or even pathognomonic for 'herpetic' infections including corneal endotheliitis. This observation has implications on future treatment of this condition. Further investigations are necessary to confirm these postulations in large patient series.

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Sir,

Reply to ocular pathology in congenital heart disease

We read with interest the paper of Mansour *et al*[1] on ocular pathology in congenital heart disease.

In a recently published study,[2] we have focused our attention on the relationship between heart and ocular defects in Down's syndrome (DS) patients.

Our study based on 65 DS patients (aged between 1 month and 15 years old), followed up with an ophthalmological examination at birth and one each year, showed that in 17 cases (26%), congenital heart disease (CHD) and ocular anomalies (OA) were significantly associated (χ^2 test, $P<0.01$).

We also found a recurrent association between nystagmus (4/6) and congenital cataract (3/3) with atrial septal defects and between myopia and severe CHD (three with atrioventricular canal and two with Fallot tetralogy on six cases), suggesting a possible specific pattern of association.

Moreover, Bromham *et al*[3] observed that in children with DS, heart defects were associated with both myopia and nystagmus and not with other ocular anomalies.

We have searched for possible association between ocular and heart anomalies in the Sicilian Registry of Congenital Malformations database and we found 15 cases of nonsyndromic congenital cataract. In five cases (30%; $P=0.45$), CHD was also reported (four with atrial septal defect and one with ventricular septal defect).

This second set of data confirms the hypothesis of a link between congenital cataract and atrial septal defects, even if this type of CHD is common and a causal relationship is difficult to assess with small sample size.

On the basis of these observations, it is possible to reinforce the hypothesis that susceptibility genes for specific CHD and OA may be contiguous or reciprocally influenced.