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Sir,

Intravitreal ganciclovir injections in aqueous cytomegalovirus DNA positive hypertensive iritis

Teoh *et al*[1] suggested that Posner–Schlossman Syndrome (PSS) may represent a spectrum of anterior chamber inflammatory responses to local viral reactivation of members of herpesviridae family such as CMV and HSV. We would like to share a similar case of CMV associated hypertensive iritis treated with intravitreal ganciclovir.

A 42-year-old Chinese man initially presented with a 3-day history of right eye redness and pain. Best-corrected visual acuity was 6/9 and intraocular pressure was 45 mmHg. Right eye slit-lamp examination showed mild corneal oedema, medium-sized stellate keratic precipitate, and mild anterior chamber reaction. Gonioscopy showed open angle. No glaucomatous optic disc changes were present and Humphrey visual fields were normal. The rest of the ocular examinations of both eyes were normal. On direct questioning, patient admitted having two mild episodes of visual blurring, discomfort, and redness in his right eye, each lasting 1–2 days, over the past 2 years. A tentative diagnosis of PSS was made. Topical corticosteroids and timolol were prescribed for ocular hypertension and inflammation. The right eye returned to normal after 2 weeks.

After 1 year, he presented with a recurrence. The right intraocular pressure was again high at 50 mmHg. The condition was again controlled with topical steroids and timolol.

A relapse recurred 2 months later when the frequency of topical steroid was reduced. To exclude an infective cause, the aqueous was aspirated for polymerase chain reaction (PCR) analysis. The right aqueous had CMV DNA measured at 7.16×10^3 copies per ml. The left aqueous was negative for CMV. HIV test on the patient was negative. His CD4 and CD8 counts were normal. The serum IgG but not the IgM to CMV was positive, the serum CMV antigen was negative. He was recommended to take ganciclovir but was unhappy with the diagnosis and the expenses involved. He subsequently agreed to try an intravitreal injection of ganciclovir. Right eye aqueous was retested 2 weeks later and the CMV DNA count had been reduced to 1.3×10^3 copies per ml. The patient had a quiet anterior chamber and intraocular pressures were within normal range. We think our case adds to the evidence of herpesviridae viruses playing a significant role in the etiology of PSS. We look forward to further interesting reports on this subject.

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Sir,

Intravitreal triamcinolone acetonide as an adjunct in the treatment of severe ocular toxoplasmosis

Toxoplasmosis lesions in the posterior pole are generally treated with anti-Toxoplasma drugs in association with high-dose oral corticosteroids.[1] Our standard therapeutic regimen consists of a 6-week course of pyrimethamine, sulphadiazine, folinic acid, and oral prednisone, along with topical drops. However, there are situations in which systemic corticosteroids are contraindicated. Herein we report two such cases, treated with anti-Toxoplasma drugs and an intravitreal triamcinolone acetonide (IVTA) injection.

Case reports

A 30-year-old woman had central serous chorioretinopathy OD associated with high-dose corticosteroid use and active macular toxoplasmosis OS (Figure 1). At 7 days before presentation, she was started on pyrimethamine 25 mg daily, sulphadiazine 2 g daily, folinic acid 5 mg twice weekly, and prednisone 60 mg daily. Visual acuity was 20/50 OD and counting fingers OS. Oral prednisone was discontinued and 4 mg of triamcinolone acetonide was injected via pars plana OS under topical anesthesia. After 5 days, marked amelioration of the vitritis was seen, with visual acuity of 20/200 OS. At 1 month following the injection, visual acuity was 20/20 OD and 20/100 OS. Funduscopy was normal OD and disclosed a scarred toxoplasmosis lesion in the superotemporal macula OS (Figure 2). The medication was switched to oral trimethoprim-sulphamethoxazole (160/800 mg) three times a week for 3 months. At 18 months after IVTA injection, no signs of recurrence were noted.

A 74-year-old difficult controlled diabetic man with active macular toxoplasmosis, along with severe anterior chamber reaction and vitritis OS, had visual acuity of 20/20 OD and hand movements OS. Pyrimethamine, sulphadiazine, and folinic acid were started. After 1 week, the picture was unchanged. In total, 4 mg of triamcinolone acetonide was injected via pars plana OS. After 7 days, marked diminution of anterior chamber and vitreous cells was noted. Treatment was switched to trimethoprim-sulphametoxazole at the 5th week and discontinued 3 months later. At 18 months following IVTA injection, visual acuity was CF at 3 m and signs of scarring were seen in the retinochoroidal lesion. No evidence of recurrence or active inflammation was detected.

Discussion

Kishore *et al*[2] reported treating four patients with clindamycin and dexamethasone given intravitreally

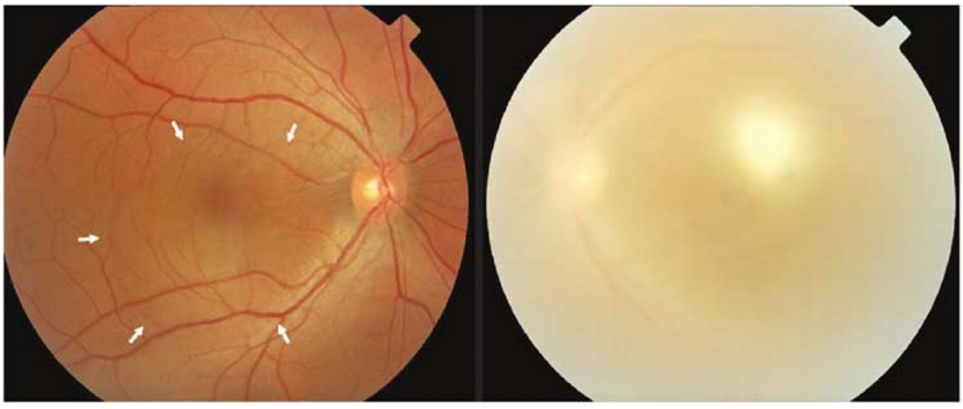

Figure 1 Initial presentation. (Left) Fundus photography of the right eye (OD) revealing a circumscribed neurosensory retinal detachment in the posterior pole (arrows). (Right) Fundus photography of the left eye (OS) showing a whitish, fluffy lesion in the superotemporal macula, as well as marked vitritis. Fundus details are barely visible.

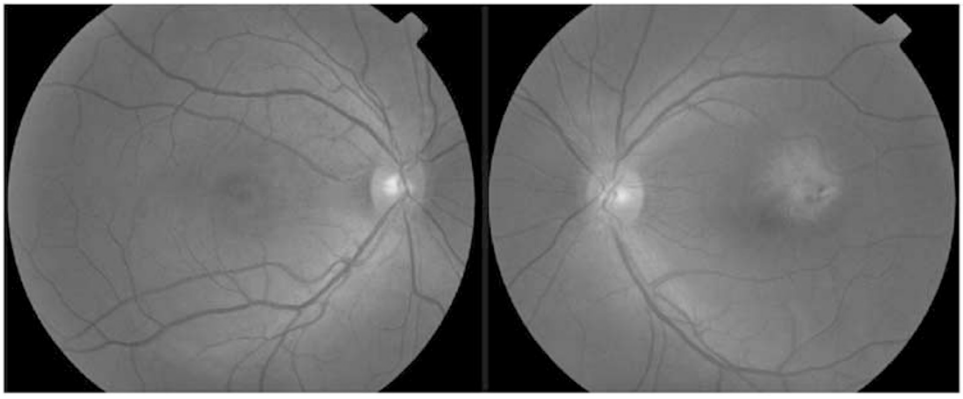

Figure 2 At 1 month following IVTA injection. (Left) Fundus photography OD (normal). (Right) Fundus photography OS disclosing a scarred toxoplasmic retinochoroiditis lesion in the superotemporal macula.

under general or peribulbar anesthesia. However, up to four injections were required to control the disease, and recurrence occurred in one case during a mean follow-up time of 17.5 months. Three patients, including the one with recurrence, continued one systemic antitoxoplasmic medication during follow-up.

IVTA has been efficient in rapidly decreasing inflammation in acute sight-threatening noninfectious uveitis.[3,4] In our study, a single injection of IVTA was used as an adjunct in the treatment of severe ocular toxoplasmosis, with rapid and successful control of the intraocular inflammation in both cases and no signs of recurrence during an 18-month follow-up period. Trimethoprim-sulphamethoxazole three times a week was used as a prophylactic treatment against the parasite in the period in which some triamcinolone acetonide residue might still be present in the vitreous cavity, potentially suppressing the immune response.

Conclusion

We suggest that IVTA should be considered as a potential benefit to patients with severe ocular toxoplasmosis or in which the use of systemic corticosteroids is contraindicated and should be investigated further. To our knowledge, this is the first report of IVTA in the treatment of infectious uveitis.[4]

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Sir,

The natural history of Vigabatrin-associated visual field defects in patients electing to continue their medication

We read with interest the paper by Best and Acheson[1] regarding the natural history of Vigabatrin-associated visual field defects in patients electing to continue their medication. The first cases of concentric bilateral visual field defects in patients taking Vigabatrin were reported in the late 1990s and since then numerous cases have been reported worldwide. The association has grown stronger and is now a well-accepted adverse drug effect of Vigabatrin therapy.[2–4]

In 2001, the Royal College of Ophthalmologists (RCO) published screening guidelines entitled 'The ocular side-effects of Vigabatrin, information and guidelines for screening'.[5] For adults, they recommend pretreatment baseline visual field using either static suprathreshold 2 or 3 zone perimetry (Humphrey 120 point or Octopus 07) to at least 45 radius eccentricity, or Goldman kinetic perimetry (IIIe and I4e or I2e stimuli, as appropriate). All patients should have 6 monthly follow-up assessments for the first 3 years of treatment, which can then be extended to annually in patients in whom no visual field defects are found.

We used a questionnaire survey in the South-West of England and Wales to investigate views of consultant ophthalmologists on the RCO guidelines and to review current clinical practice.

Out of 97 consultants, 54 contacted responded to the questionnaire (response rate 56%). Consultant ophthalmologists were asked about their experience with Vigabatrin-related visual field screening in the year 2002–2003. More than a third (35%) of those surveyed were unaware that the RCO had published guidelines relating to Vigabatrin-associated visual field defects. Only 15% had received new referrals for baseline visual field documentation prior to patients starting on Vigabatrin and 41% of respondents had performed or arranged visual field screening for patients already on Vigabatrin. With respect to screening intervals, 51% thought that the screening interval should be 12 months or longer, 45% agreed with a screening interval of 6 months and 4% felt