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Sir,

Haemorrhagic hypertensive hypopyon uveitis in early immune reactivation uveitis

We report an atypical presentation of immune reactivation uveitis (IRU) with haemorrhagic hypertensive hypopyon uveitis after highly active antiretroviral therapy (HAART) in AIDS.

Case report

A 60-year-old male recently diagnosed with AIDS presented with a week of pain, redness, blurred vision, and floaters in his right eye. Diagnosis of AIDS was confirmed by an infectious diseases physician when he presented with persistent odynophagia due to oral candidiasis. Medical review did not reveal any opportunistic illnesses or evidence of any extraocular cytomegalovirus (CMV) infections. He was started on HAART (efavirenz, lamivudine, and zidovudine), with a baseline CD4 T-lymphocyte count <20 cells/μl ($<1\%$) and 2 weeks before onset of ocular symptoms. Of note, he was not treated with rifabutin or cidofovir.

Presenting Snellen visual acuity (VA) was 6/12 in the right eye and 6/6 in the left eye. Slit-lamp examination revealed a severe right haemorrhagic hypopyon anterior uveitis, large granulomatous keratic precipitates (Figure 1) and an intraocular pressure of 22 mm Hg. Fundus examination showed active vitritis 3+ (BIO score 2) and retinitis with flame haemorrhages, clinically consistent with CMV retinitis. Anterior chamber tap PCR confirmed CMV DNA. He was treated with intravitreal ganciclovir (2 mg/0.04 ml), oral valganciclovir, intensive prednisolone acetate 1% and twice daily timolol 0.5% eye drops with clinical improvement within 3 days. Three months later, VA improved to 6/9. There was still 0.5+ anterior chamber activity and 1+ vitritis but clinical resolution of CMV retinitis. CD4 T-lymphocyte count was 38 cells/μl (4%). His left eye remained quiescent. Throughout the period of follow-up, there were no complications of cystoid macular oedema or epiretinal membrane noted. At the last follow-up, 8 months after diagnosis of IRU, VA was 6/7.5, and both eyes were quiescent.

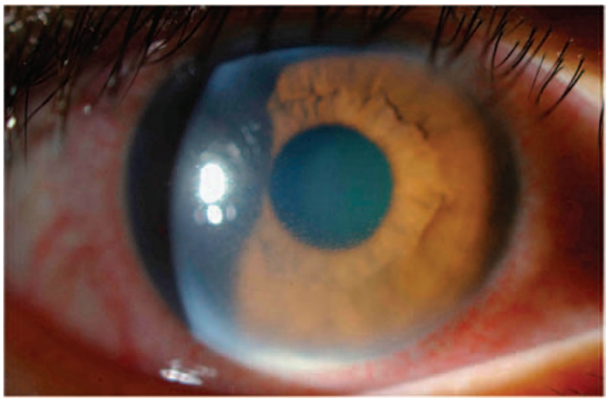

Figure 1 Right eye at presentation showing a haemorrhagic hypopyon uveitis with large granulomatous keratic precipitates.

Comment

IRU typically presents with a paucity of inflammation in the anterior as compared with the vitreous. Literature review did not reveal any cases of IRU presenting with a hypopyon or haemorrhagic hypopyon.[1] Most cases of anterior uveitis were reported as flare or cells up to 2+ activity.[2,3]

Intraocular inflammatory responses typically commence 1 to 6 months after HAART, paralleling rise in the CD4 T-lymphocyte count of >100 cells/μl or an increase of >50 above the observed nadir level.[4] This case is unusual as IRU developed just 2 weeks after, with a rise of <50 cells/μl. We postulate that the high load of intraocular CMV antigens before the initiation of HAART, as evidenced by the detection of CMV DNA in the anterior chamber tap; and specifically an increased CMV antigen load in the trabecular meshwork that contributed to the slightly raised intraocular pressure at presentation, are possible reasons for the early and aggressive inflammatory hypopyon reaction. A case of CMV panuveitis was reported earlier with evidence of cytomegalic transformation of corneo-trabecular endothelial cells.[5]

CMV uveitis in AIDS typically presents with minimal inflammation. The gradual improvement seen with specific anti-CMV agents, however, suggests absence of other endogenous infections.

IRU should be considered in patients with anterior segment inflammation even within a few days of commencing HAART.

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NY Gan and SC Teoh

National Healthcare Group Eye Institute, Department of Ophthalmology, Tan Tock Seng Hospital, Singapore
E-mail: stephen_teoh@ttsh.com.sg

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