was more likely that our patient had developed a scleral granuloma as a result of local $CD8^+$ infiltration and that this regressed with commencement of HAART and topical steroids.

Scleral granulomas have been found in association with different systemic conditions.[6–10] To our knowledge this is the first report of scleral granuloma occurring in the presence of presumed DILS. During HIV infection the immune system becomes dysfunctional because of the coexistence of immunodeficiency and immune hyperactivity, and a disregulated production or activity of cytokines, thereby explaining the development of the DILS. Clinicians need to be aware of this entity, which is gaining in significance.

References

1 Nopachai A, Garwacki CP, Moll S. Diffuse infiltrative lymphocytosis syndrome. *Am J Hematol* 2004; **75**(3): 173–175.
2 Henderson HW, Davidson F, Mitchell SM. Intraocular inflammation and the diffuse infiltrative lymphocytosis syndrome. *Am J Ophthalmol* 1998; **126**(3): 462–464.
3 De Silva DJ, Obi AA, Mitchell SM. Bilateral panuveitis in HIV-1-infected patients with CD8 lymphocytosis. *Ocul Immunol Inflamm* 2005; **13**(4): 311–316.
4 Kestelyn P, Lepage P, Karita E, Van de Perre P. Ocular manifestations of infection with the human immunodeficiency virus in an African pediatric population. *Ocul Immunol Inflamm* 2000; **8**(4): 263–273.
5 Schuval SJ, O'Reilly ME, Bonagura VR. Increased frequency of HLA-DR11 in pediatric human immunodeficiency virus-associated parotid gland enlargement. *Clin Diagn Lab Immunol* 1997; **4**(3): 258–260.
6 Saito W, Kotake S, Ohno S. A patient with sarcoidosis diagnosed by a biopsy of scleral nodules. *Graefes Arch Clin Exp Ophthalmol* 2005; **243**(4): 374–376.
7 Gopal L, Rao SK, Biswas J, Madhavan HN, Agarwal S. Tuberculous granuloma managed by full thickness eye wall resection. *Am J Ophthalmol* 2003; **135**(1): 93–94.
8 Janknecht P, Mittelviefhaus H, Loffler KU. Sclerochoroidal granuloma in Wegener's granulomatosis simulating a uveal melanoma. *Retina* 1995; **15**(2): 150–153.
9 Kielar RA. Exudative retinal detachment and scleritis in polyarteritis. *Am J Ophthalmol* 1976; **82**(5): 694–698.
10 Hatsuda TA, Tanaka J. Bilateral necrotizing scleritis. *Am J Ophthalmol* 1978; **86**(5): 710–714.

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Sir,

Dengue retinopathy manifesting with bilateral vasculitis and macular oedema

Ocular involvement is uncommon in dengue fever, most commonly presenting as blot haemorrhages.[1–4] We report an unusual manifestation of dengue fever presenting bilaterally with extensive panretinal vasculitis and severe macular oedema, which resolved spontaneously without anti-inflammatory treatment.

Case report

A 37-year-old female with dengue fever diagnosed on dengue immunoglobulin (Ig)M and IgG serology and polymerase chain reaction experienced sudden, bilateral blurring of vision with metamorphopsia 6 days after the onset of fever. Her platelet count was at its nadir (26×10^9/l). Visual acuity was 20/400 OD and counting fingers OS. There was bilateral extensive retinal vasculitis at the maculae extending outwards to the peripheral retinae, complicated by macular branch vein occlusion OD. Numerous yellowish retinal infiltrates and blot haemorrhages were scattered throughout both maculae, and there was bilateral macular oedema (Figure 1). Fundus fluorescein angiogram (FFA) demonstrated leakage indicative of extensive panretinal vasculitis, more severe at, but not limited to the maculae (Figure 1). Optical coherence tomography (OCT) showed central foveal thickening of 503 μm OD and 843 μm OS.

After platelet transfusion, her platelet count improved to 67×10^9/l and gradually returned to normal levels over the next few days. Treatment with anti-inflammatory agents for her posterior retinitis and vasculitis was considered but withheld in consultation with her physician in view of her pyrexic state.

By 2 weeks, the retinal perivascular sheathing had spontaneously resolved, macular oedema had decreased, and visual acuity improved to 20/200 bilaterally. By 10 weeks, without any specific treatment, her vision had recovered to 20/20 bilaterally with no residual inflammation or macular oedema. However, there was

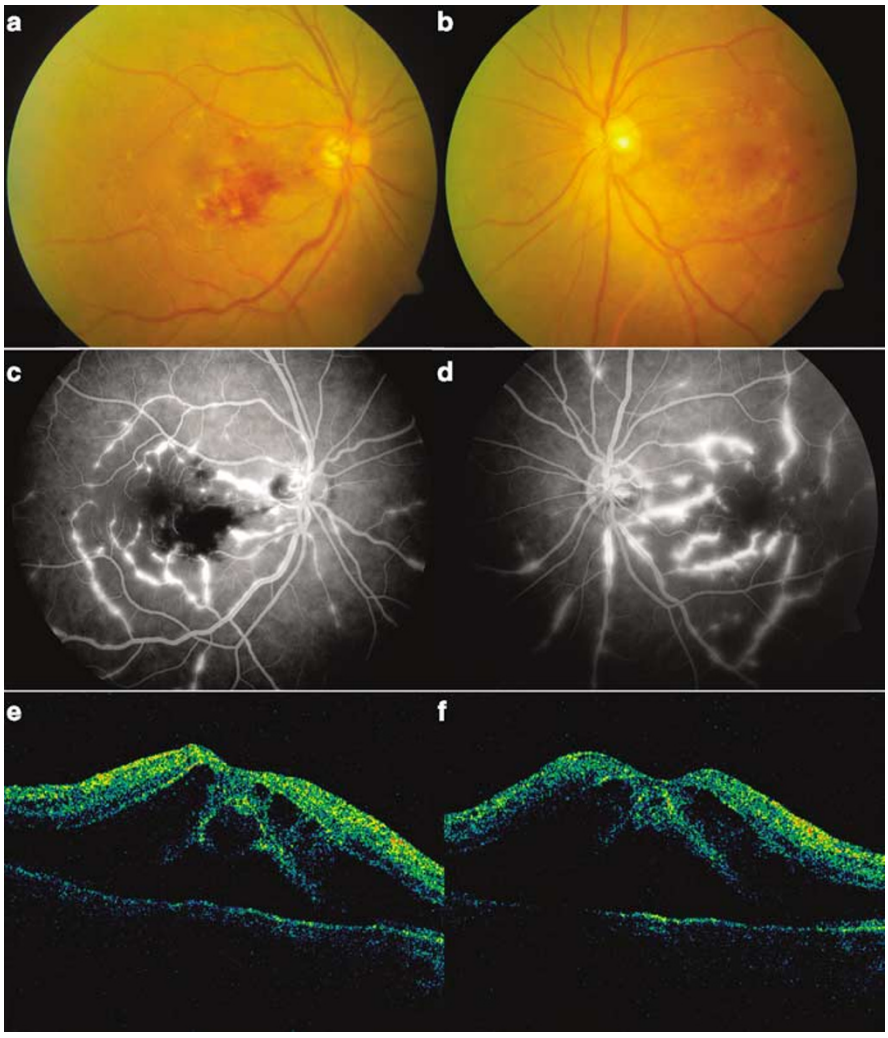

Figure 1 (a, b) Colour fundus photographs at the onset of visual symptoms showing perivascular sheathing around the foveal vessels, scattered blot haemorrhages and exudates. The flame haemorrhages and cotton wool spots in the right eye are caused by a branch macular vein occlusion. (c, d) Fundus fluorescein angiography demonstrates extensive panretinal vasculitis, more severe at, but not limited to the maculae. There is leakage from the perifoveal vessels in both eyes. (e, f) OCT of both eyes demonstrate severe macular oedema.

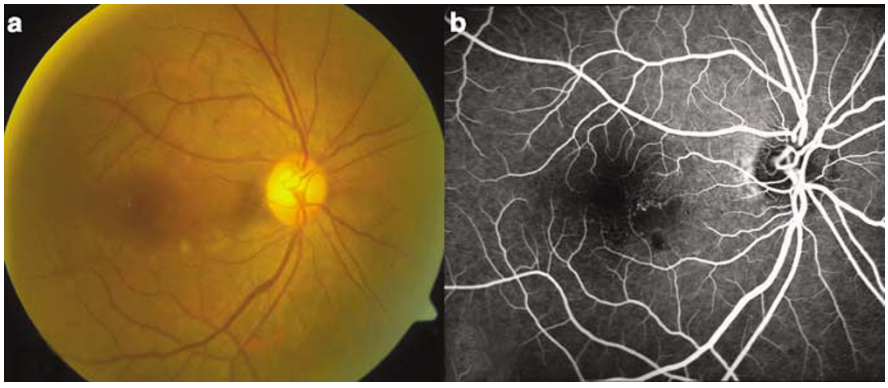

Figure 2 (a) Fundus photograph of the right eye at 10 weeks, when visual acuity has improved to 20/20. (b) Fundal fluorescein angiography (FFA) of the right eye shows an area of ischemia infero-nasal to the right fovea with telangiectatic vessels at the margins.

mild-red desaturation in the right eye and visual distortion on Amsler grid monitoring. This corresponded to an area of retinal capillary nonperfusion at the infero-nasal margin of the foveal avascular zone on repeat FFA (Figure 2).

Comment

In the few reports of dengue patients with ocular involvement, common symptoms were blurred vision, central scotoma, floaters, photophobia, and haloes.[1–4] Ocular findings included anterior chamber and vitreous inflammatory cells, intraretinal and peripapillary haemorrhage, Roth's spots, intraretinal lesions, maculopathy with diffuse oedema, vasculitis, and blurring of the optic disc margins.[1–4]

The precise ocular pathophysiology in dengue fever is unknown.[1] Our patient's ocular manifestations occurred 1 week after dengue fever, when her platelet count was at its nadir. This time interval is consistent with those previously reported[1–3] and may suggest an immune-mediated process,[1] possibly coinciding with the onset of IgG production. The retinal haemorrhages may be explained by thrombocytopaenia and a transient bleeding diathesis. In contrast to another series of patients,[1] our patient's ocular manifestations were not confined to the vascular arcades in the maculae region, but extended outwards to the peripheries, suggesting a more diffuse inflammatory process.

Unlike other vasculitic disorders, despite conservative management, there was complete resolution of the fundal signs and normal visual acuity by 10 weeks.

This case report demonstrates that dengue fever may manifest as severe panretinal vasculitis and macula oedema, which coincides with the nadir of the thrombocytopaenia on day 7 of the illness. Although severe, these conditions may be self-limiting and may resolve spontaneously without specific anti-inflammatory or antiviral therapy.

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References

1 Lim WK, Mathur R, Koh A, Yeoh R, Chee SP. Ocular manifestations of dengue fever. *Ophthalmology* 2004; **111**: 2057–2064.

2 Wen KH, Sheu MM, Chung CB, Wang HZ, Chen CW. The ocular fundus findings in dengue fever. *Gaoxiong Yi Xue Ke Xue Za Zhi* 1989; **5**: 24–30.

3 Siqueira RC, Vitral NP, Campos WR, Orefice F, de Moraes Figueiredo LT. Ocular manifestations in dengue fever. *Ocul Immunol Inflamm* 2004; **12**: 323–327.

4 Haritoglou C, Dotse SD, Rudolph G, Stephan CM, Thurau SR, Klauss V. A tourist with dengue fever and visual loss. *Lancet* 2002; **360**: 1070.

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Sir,

Bilateral non-arteritic ischemic optic neuropathy associated with pegylated interferon for chronic hepatitis C

It is recognized that standard interferon therapy can rarely cause ocular toxicity, including retinopathy and optic neuropathy,[1] but the frequency and severity of adverse effects of the pegylated form is not known.

This 46-year-old man with a 4-month history of hepatitis C, whose risk factor for this infection was past blood transfusion, had been treated with PEG interferon alpha 2B at 0.5 ml subcutaneously per week. He was not taking any other medications. After 3 weeks of treatment, he presented with acute bilateral visual loss. He described sudden blackout of his vision upon awakening without any headache or ocular pain. He had no diabetes, hypertension, heart disease, or past history of alcohol, smoking or drug abuse.

His visual acuity was 20/400 OD and 20/100 OS without relative afferent pupillary defect. Extraocular motility was full. Automated perimetry revealed bilateral inferior altitudinal defects. Slit-lamp examination and intraocular pressures were normal. He had bilateral swollen optic discs without hemorrhages or cotton wool spots. Macular and peripheral retinal exam was normal.