Sir,

Bilateral endogenous *Candida* endophthalmitis as the presenting manifestation of diabetes mellitus

Endogenous *Candida* endophthalmitis (ECE) typically occurs in patients with systemic risk factors, including long-term intravenous treatment, debilitating diseases, or recent surgery or childbirth.[1] We present a case of bilateral ECE with concurrent *Staphylococcus aureus* bacteraemia in an otherwise healthy man without any relevant history. Systemic evaluation revealed diabetes mellitus (DM) as the only factor that predisposed to the development of ECE.

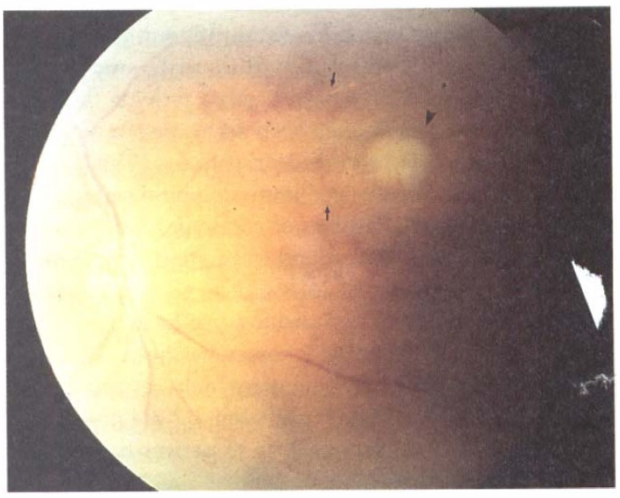
Case report

A 55-year-old robust man experienced rapid vision deterioration in both eyes within 1 day. Corrected visual acuity was 6/20 in the right eye and counting fingers in the left eye. Biomicroscopy showed 4+ cells in the anterior chamber in both eyes and 2 mm hypopyon in the left eye. Ophthalmoscopy disclosed a creamy-white round mass protruding from the superonasal retina into the hazy vitreous with surrounding retinal haemorrhage in the right eye. The retina was obscured by dense vitreous opacity in the left eye.

The patient had no history of drug abuse, surgical or medical disorders. The physical examinations were unremarkable. Blood routine showed leucocytosis with neutrophil predominance. Intravenous cefazolin sodium and amikacin were administered after aqueous humour and blood samples were obtained for evaluation. Fever and dysuria developed 8 h later and urine analysis showed 2+ white blood cells and 3+ occult blood, but no pathogen was identified.

The clinical course deteriorated and on the second day the vision dropped to 1/60 in the right eye and hand motions in the left eye. Spasmo-Euvernil (sulphaniyl carbamide and phenazophyridine) and flomoxef sodium were administered instead for the probable urinary tract infection and suspected *Klebsiella pneumoniae* endophthalmitis. *K. pneumoniae* is the most common pathogen responsible for bilateral endogenous endophthalmitis in our hospital as a complication of liver abscess.[2] The pathogen identified in two sets of blood culture turned out to be *Staphylococcus aureus* that was sensitive to flomoxef sodium. Ocular and urinary conditions began to improve, fever subsided and blood culture turned negative after shifting antibiotics.

Except for high blood sugar and HbA_{1c}, the immune profile, serological tests for herpes virus and tumour marker surveys were all insignificant. Results of a chest radiograph, brain CT scan, echocardiography and whole abdomen ultrasonography were unremarkable. The patient's vision recovered to 6/20 in the right eye and 5/60 in the left eye 2 weeks later. With resolution of the inflammation, creamy-white vitreous opacities and yellowish granulomatous retinal masses with surrounding retinal haemorrhage and sheathing vessels became identifiable in both eyes (Fig. 1). On the 16th day, *Candida albicans* was isolated from the culture of aqueous humour and oral fluconazole was then administered. Two weeks later, the vision improved to 6/7.5 in the right eye and 6/30 in the left, but mild background diabetic retinopathy became evident in both eyes. The left eye was also complicated by tractional retinal detachment, so vitrectomy and focal laser photocoagulation was performed. Although there was no recurrent chorioretinitis or retinal detachment, the diabetic retinopathy progressed rapidly (Fig. 2) and panretinal photocoagulation was performed in the right eye 3 months later. The vision was 6/8.6 in the right eye and 5/60 in the left eye 10 months after the event.



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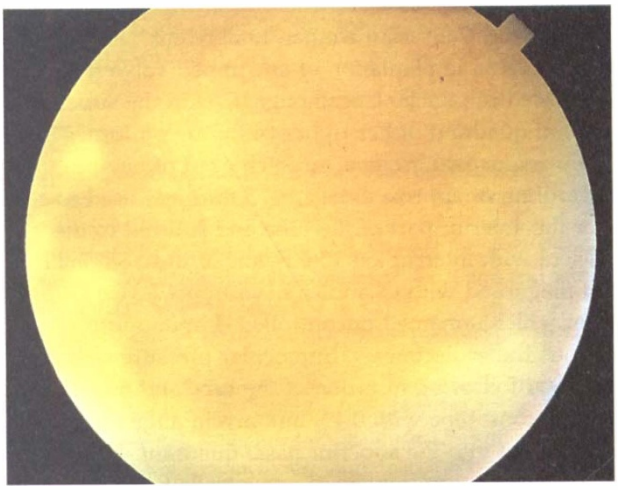
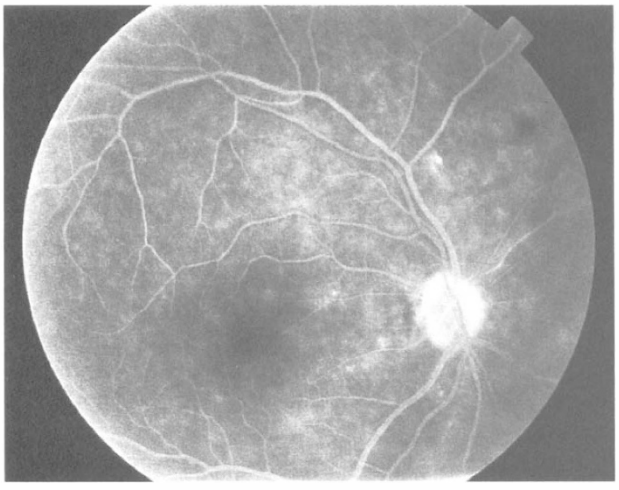


(b)

Fig. 1 *(a) A granulomatous lesion (arrowhead) over the superonasal retina with surrounding retinal haemorrhage and sheathing vessels (arrows) in the right eye. (b) Creamy-white snowball-like vitreous opacities in the left eye.*

Comment

DM is a well-known risk factor for endogenous fungal endophthalmitis;[1,3] however, almost all diabetic patients with ECE have other complicating conditions, such as surgery or deep tissue infection, which contribute to the development of endophthalmitis.[3,4] It is unusual for ECE



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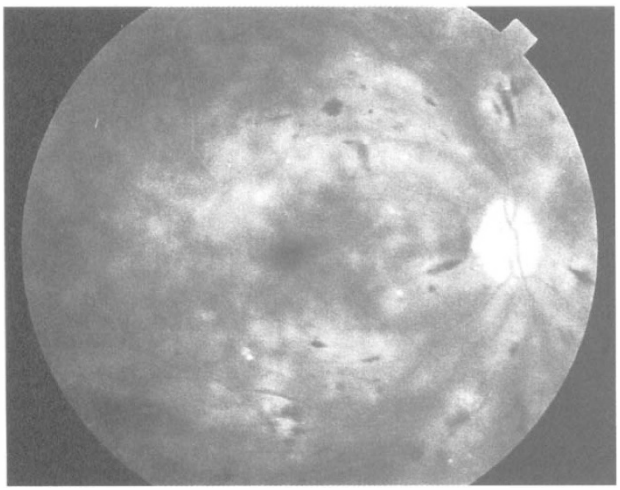

(b)

Fig. 2. *Fluorescein angiography of the right eye showed rapid progression of diabetic retinopathy. (a) Twelve days, (b) 2 months after clinical onset.*

to develop in an apparently robust man. To the best of our knowledge, ECE has never been reported as the presenting manifestation of DM as found in this case.

Diabetics are susceptible to both staphylococcal and *Candida* infection because their monocytes are decreased in number and the killing activity of their monocytes and neutrophils against these two pathogens is impaired.[5,6] Willcox *et al.*[7] have reported that DM is the most common underlying disease for community-acquired *S. aureus* bacteraemia in the absence of intravenous drug abuse, but no patients in their study developed endogenous endophthalmitis.

In this case, bacterial endophthalmitis was first diagnosed based on the findings of blood routine and blood culture, as well as improved ocular condition with antibacterial antibiotics. However, the manifestations that became evident after resolution of vitreous haze and the result of aqueous culture confirmed the diagnosis of *Candida* endophthalmitis. The characteristic findings of ECE are creamy-white, circumscribed chorioretinal lesions that may be associated with retinal haemorrhage, perivascular sheathing, and yellow-white vitreous opacities. Differential diagnoses include necrotising retinopathies, *Toxoplasma* retinochoroiditis, cryptococcosis and choroidal granuloma.[3]

Since *C. albicans* has a propensity to localise in the choroid and to spread towards the retina and vitreous cavity,[1] endophthalmitis can result from transient candidaemia, which is not uncommon among diabetics.[3] In addition, superinfection with *S. aureus* exerts a synergistic effect on *Candida* pathogenicity, as demonstrated by Carlson.[8] With antibacterial antibiotics, eradication of staphylococcal bacteraemia may nullify the synergistic effect of bacteria on *Candida* infection.[9] Furthermore, since both bactericidal and fungicidal systems depend mostly on phagocytosis and cell-mediated immunity, eradication of *Staphylococcus* would lighten the burden of the immune system and facilitate spontaneous resolution of ECE.

Diabetic patients may present themselves with bilateral ECE, though it is rare. Searching for predisposing risk factors associated with ECE is imperative even in an apparently healthy patient. Because diabetic retinopathy may progress rapidly after endophthalmitis,[10] the diabetic patient should be followed up carefully and closely so that timely intervention can be initiated.

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Sir,

Spontaneous hyphaema secondary to a vascularised fragment of persistent pupillary membrane

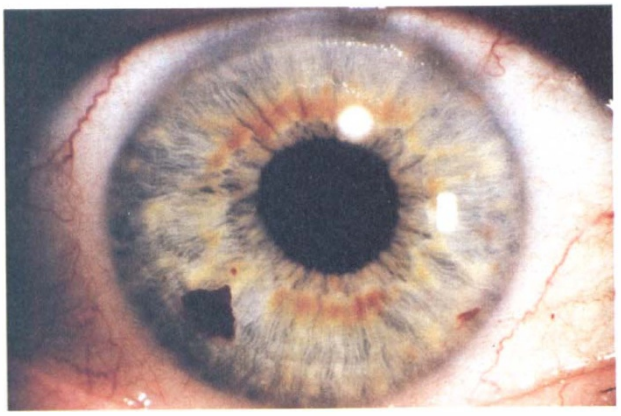
Bleeding from a persistent pupillary membrane has been previously reported in only four patients.[1–4] In each of these cases there appears to have been some predisposing factor such as strenuous physical exertion or hypertension. We report a case of spontaneous hyphaema from a vascularised strand of persistent pupillary membrane without any predisposing factors.

Case report

A 57-year-old Caucasian woman was referred to the eye casualty department complaining of the sudden onset of misty vision in her right eye, which had persisted for 18 h. There were no other ocular or systemic symptoms, in particular symptoms suggestive of coagulopathy. There was no history of ocular trauma or any other strenuous physical activity before the onset of the visual symptoms. There was no significant past medical history and the patient was not on any medication (in particular, she had not been taking anticoagulants or aspirin).

The visual acuity was 6/5 unaided in each eye and slit-lamp examination of the left eye was normal. The right anterior chamber was of normal depth and showed 1+ of suspended red blood cells (RBCs). Small blood clots were adherent to the iris inferotemporally and inferonasally (Fig. 1a). In continuation with the inferotemporal clot was an abnormal vessel, which appeared to be a vascularised strand of persistent pupillary membrane, crossing the collarette of the pupil from the inferotemporal to superonasal region (Fig. 1b). This strand appeared to have an area of aneurysmal dilatation from which the haemorrhage arose. There was no rubeosis iridis. There was no evidence of uveitis or iris atrophy. Gonioscopy showed wide-open angles without any new vessels in the angle. Intraocular pressures were within normal limits. Fundus examination of each eye was normal. The eyelids and orbits on each side were normal to both inspection and palpation. General physical examination was normal. The blood pressure at presentation was 150/80 mmHg. There was no evidence of any carotid bruit.

The patient was treated with topical Predsol forte four times a day. The RBCs in the anterior chamber and the blood clots disappeared over the next 6 days. Anterior segment fluorescein angiography showed a normal iris vascular pattern in the left eye and over most of the right iris, with radial arteries becoming fluorescent from the periphery to the centre without any pupillary border fluorescence. In addition, at the collarette of the right eye, there was an incomplete vascular circular arteriosus minor and from this an anomalous vessel with slight aneurysmal dilatation was seen coursing from the inferotemporal to the superior collarette (Fig. 2). There were no other abnormal vessels or vascular structures noted in the iris.



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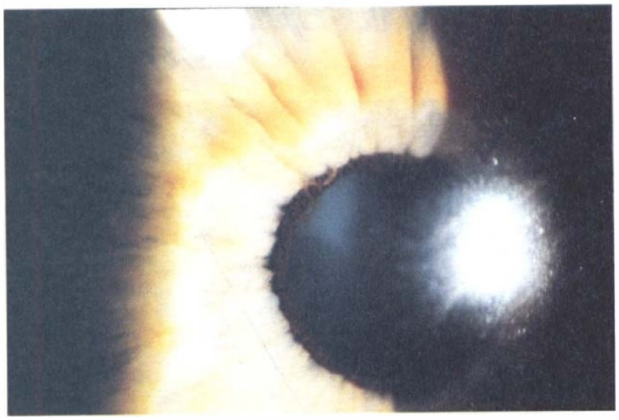

(b)

Fig. 1. *(a) Colour photograph of the right eye at presentation with hyphaema. (b) Colour photograph of the right eye demonstrating the vascularised fragment of persistent pupillary membrane.*

Comment

The vascular connection between the minor cycle and the tunica vasculosa lentis derives from mesodermal tissue and normally disappears before birth. At about the eighth month of gestation the pupillary membrane starts to degenerate and eventually disappears. Fine fibrillary remnants often persist even after birth.[5] Histologically fine strands of mesodermal tissue are seen, rarely with blood vessels. Total persistence of a pupillary membrane is extremely rare and is associated with other ocular anomalies such as microphthalmos.

In all the previously reported cases a predisposing factor, either strenuous effort immediately preceding the bleeding[1,4] or systemic hypertension,[2,3] contributed in