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Sir,

Spontaneous hyphaema secondary to a vascularised fragment of persistent pupillary membrane

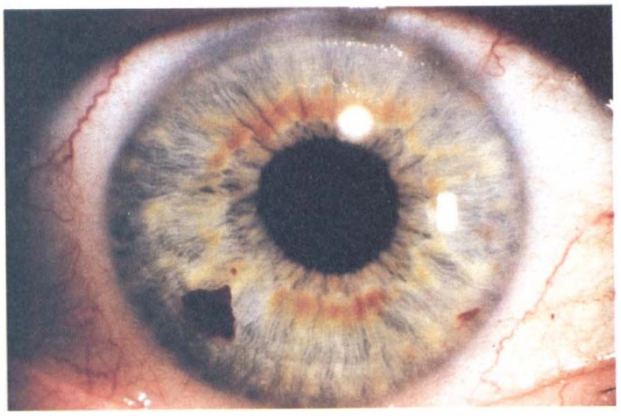
Bleeding from a persistent pupillary membrane has been previously reported in only four patients.[1–4] In each of these cases there appears to have been some predisposing factor such as strenuous physical exertion or hypertension. We report a case of spontaneous hyphaema from a vascularised strand of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

Case report

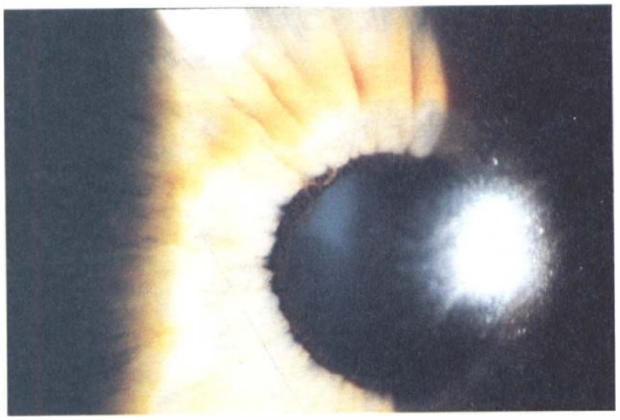
A 57-year-old Caucasian woman was referred to the eye casualty department complaining of the sudden onset of misty vision in her right eye, which had persisted for 18 h. There were no other ocular or systemic symptoms, in particular symptoms suggestive of coagulopathy. There was no history of ocular 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.



(a)



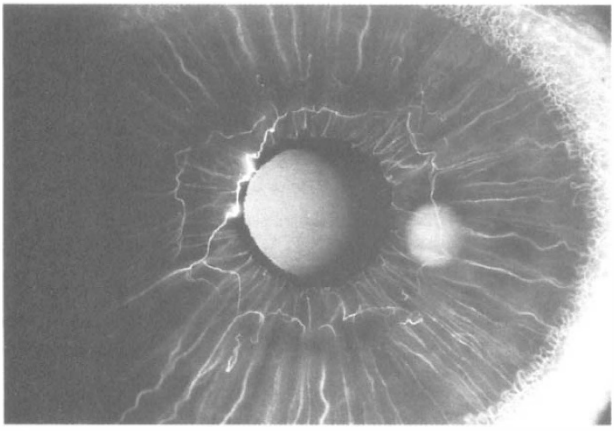
(b)

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.*

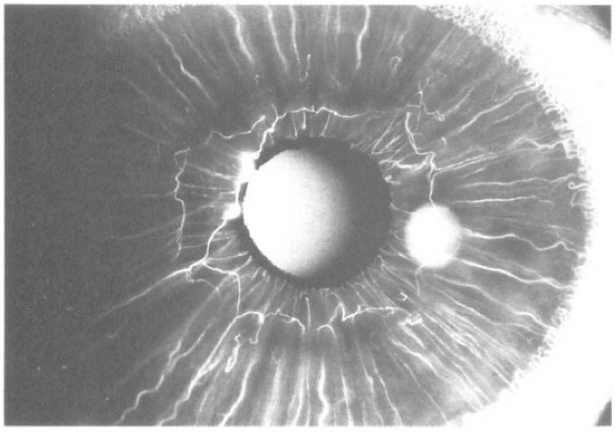
Comment

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



(a)



(b)

Fig. 2. *(a) Iris fluorescein angiogram of the right eye, early phase, demonstrating aneurysmal dilatation of the persistent pupillary membrane. (b) Iris fluorescein angiogram of the right eye, late phase, demonstrating leakage.*

the causation of hyphaema. To our knowledge, this is the first report of a truly spontaneous hyphaema originating from a vascularised persistent pupillary membrane.

Conservative treatment with topical drugs had been the mainstay of treatment in previous cases. However, Rydberg[2] successfully used argon laser to photocoagulate the bleeding vessel in a patient who suffered from recurrent hyphaema.

The patient in this report was treated conservatively with topical steroid drops, reserving laser treatment in the event of recurrence. At 1 year follow-up no further bleeding had occurred and we continue to monitor this patient.

References

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Sir,

Blood-stained keratic precipitates: presenting feature of sarcoidosis with thrombocytopenia

We describe a new clinical sign of blood-stained keratic precipitates which alerted us to the possibility of both thrombocytopenia and sarcoidosis in this patient.

Case report

A 37-year-old British-born West Indian woman presented with a 3 week history of bilateral floaters and blurred vision. This was preceded by 4 months of malaise, dry cough, night sweats, and a weight loss of 19 kg.

Ophthalmic examination revealed visual acuities correcting to 6/6 on the right and 6/12 on the left. Both eyes had granulomatous anterior uveitis, blood-stained keratic precipitates (Fig. 1) and a moderate vitritis with normal fundi. She had mucosal and cutaneous petechiae. A chest radiograph showed bilateral hilar lymphadenopathy and blood tests revealed raised inflammatory markers (ESR = 68 mm/h; CRP = 26 mg/l, normal < 7 mg/l and a platelet count of 6×10^9/l. In view of the latter she was admitted to hospital for a 5 day course of intravenous immunoglobulins at 0.4 mg/kg and was also commenced on topical dexamethasone for the uveitis.

A bone marrow trephine on admission confirmed reactive histology with megakaryocytic hyperplasia and moderate lymphoplasmacytosis. Autoimmune screening revealed a raised IgA anticardiolipin antibody at 17.5 APL u/ml (normal < 7 u/ml) and elevated platelet-associated immunoglobulin confirming an autoimmune basis for her thrombocytopenia. Further investigations relating to the cause of her uveitis included an anergic Heaf test, a serum ACE of 306 u/l (normal 27–82 u/l) and a gallium scan showing markedly increased uptake throughout both lungs and lacrimal glands. A diagnosis of sarcoidosis with secondary immune thrombocytopenic purpura was made.

Within 2 days of completing the course of immunoglobulin the patient's platelet count was 138×10^9/l and she was feeling well. Subsequent pulmonary function tests showed a significant mixed restrictive and obstructive deficit with a decreased transfer factor and, in view of this, increasing breathlessness and persisting extensive mediastinal and hilar lymphadenopathy (confirmed on a chest CT scan), she was commenced on prednisolone 40 mg p.o. daily.