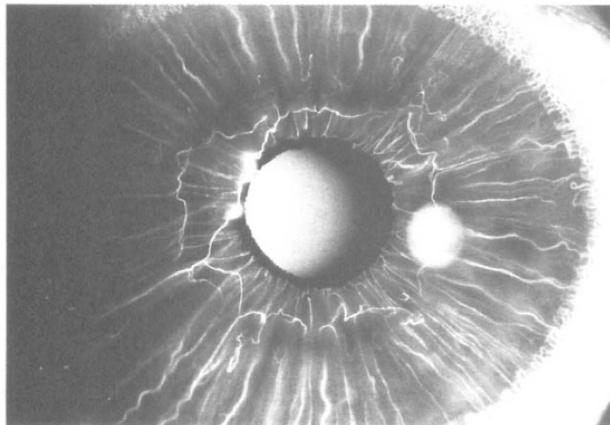


(a)



(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, Rydberg² 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

1. McLean DW. An unusual case of intra-ocular haemorrhage. *Br J Ophthalmol* 1946;30:758.
2. Rydberg M. Haemorrhage from remnants of prepupillary membrane. *Acta Ophthalmol (Copenh)* 1965;43:160-3.
3. Brusini P, Beltrame G. Spontaneous hyphaema from persistent remnant of the pupillary membrane. *Acta Ophthalmol (Copenh)* 1983;61:1099-103.
4. Laidlaw DAH, Bloom PA. Spontaneous hyphaema arising from a strand of persistent pupillary membrane. *Eur J Ophthalmol* 1993;3:98-100.

5. Snell RS, Lemp MA. *Clinical anatomy of the eye*. Oxford: Blackwell Science, 1989:9.

V. Kotamarthi
O. Sarodia
G.H.A. Woodruff
Ophthalmology Department
Leicester Royal Infirmary
Leicester LE2 7LX, UK
V. Kotamarthi ✉
e-mail: ransha14@hotmail.com

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 $6 \times 10^9/l$. 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 $138 \times 10^9/l$ 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.

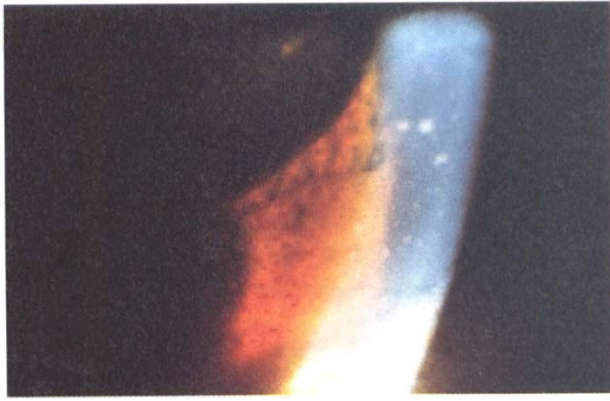


Fig. 1. Anterior segment photograph showing blood-stained keratic precipitates, best seen inferiorly.

Within a further 4 weeks her vision was 6/6 bilaterally with quiet eyes and her inflammatory markers were settling.

Comment

Some degree of thrombocytopenia in sarcoidosis is well recognised. However, severe thrombocytopenia (platelet count of less than $20 \times 10^9/l$) is much rarer, with fewer than 30 cases reported in the literature.^{1,2} Thrombocytopenia may occur in sarcoidosis secondary to bone marrow involvement, hypersplenism or antibody-mediated destruction of platelets. There have been only four reports of the use of intravenous immunoglobulin in thrombocytopenia associated with sarcoidosis, in all of which it was used when systemic steroids with or without vincristine had failed to increase the platelet count.³⁻⁶ In our case steroids were avoided initially due to the fact that uveitis and severe thrombocytopenia were the presenting features of the disease, and infectious causes had not been excluded at that stage. The severity of the thrombocytopenia meant that delay in its treatment pending further results was not advisable.

To our knowledge this is not only the first description of blood-stained keratic precipitates but also the first report of the use of intravenous immunoglobulin to increase the platelet count as a first-line treatment in sarcoidosis with thrombocytopenia.

References

1. Dickerman JD, Holbrook PR, Zinkham WH. Etiology and therapy of thrombocytopenia associated with sarcoidosis. *J Pediatr* 1972;81:758-64.
2. Lawrence J, Greenberg BR. Autoimmune thrombocytopenia in sarcoidosis. *Am J Med* 1985;79:761-4.
3. Henke M, Engler H, Engelhardt R, Lohr GW. Successful therapy of sarcoidosis-associated thrombocytopenia refractory to corticosteroids by a single course of human gammaglobulins. *Klin Wochenschr* 1985;64:1209-11.
4. Lerner AJ. Hammersmith staff rounds. Life threatening thrombocytopenia in sarcoidosis. *BMJ* 1990;300:317-9.

5. Guzzi LM, Kucera RF, Lillis P, Hornstein EH. Human gammaglobulin use in the treatment of severe thrombocytopenia associated with sarcoidosis. *Am J Med Sci* 1991;301:331-4.
6. Roedel C, Flury G. Severe thrombocytopenia in systemic Boeck's disease. *J Suisse Med* 1992;122:1582-5.

A.M.S. Noury
C.C. Bailey
M.R. Stanford
E.M. Graham
St Thomas' Hospital
London, UK

Miss C.C. Bailey ✉
Medical Eye Unit
St Thomas' Hospital
Lambeth Palace Road
London SE1 7EH, UK

Tel: +44 (0)207 928 9292
Fax: +44 (0)207 922 8165
e-mail: ccbailey@doctors.org.uk

Sir,

The use of supplementary blue light during Perkins applanation tonometry in theatre

Accurate intraocular pressure measurement is an important part of a paediatric ophthalmologist's examination of a child. Goldmann applanation tonometry is regarded as the 'gold standard' for intraocular pressure measurement as it has been shown to be consistently reliable and accurate. It is the standard against which all other tonometers are measured.¹ However, Goldmann applanation tonometry is not a portable technique, which makes it impractical in the paediatric setting where pressure measurements are performed in the spine position under general anaesthesia.

The Perkins tonometer (Clement Clarke) is a portable applanation tonometer which is used frequently during examination under anaesthesia. It is based on the Goldmann tonometer but is counterbalanced so that tonometry can be performed in any position. Its accuracy is comparable with the Goldmann applanation tonometer and this has been confirmed by a number of research groups.^{2,3}

One disadvantage of the Perkins tonometer is that the illumination of the tonometer prism by two cobalt blue filtered light sources within the instrument is very dim (5.4 volts total). This makes visualisation of the excited fluorescein within the tear film difficult, even when the room lighting is reduced. This may increase the time required for examination, and can lead to an underestimation of intraocular pressure.⁴ Modifications have been suggested to the tonometer to increase the internal illumination⁵ but these are not easily achievable. Simple pen torches with blue filter caps are widely available and used in the examination of the anterior segment with fluorescein. The average bulb voltage from a pen torch is 2.2 volts. We wished to establish whether the use of external supplementary blue light from a pen torch facilitates measurement of intraocular pressure in theatre using the Perkins tonometer.