

polyclonality, lymphoplasmacytoid differentiation or incomplete light chain restriction of the lymphoid infiltrate.⁶⁻⁹

Our patient presented with anterior uveitis, a choroidal mass, no visible epibulbar mass, and subsequently an optic neuropathy with a mass surrounding the optic nerve and thickening of the choroid. Histopathological examination revealed small monoclonal B cells with slightly angulated nuclear profiles that were light chain restricted. Although we did not biopsy the choroidal component of the mass it is likely that our biopsy of the tumour adjacent to the optic nerve was representative and our patient had a mantle cell lymphoma involving the choroid at presentation. Our patient's tumour resembles the picture of ULI in some respects. The histopathological findings support a diagnosis of lymphoma. Distinction between benign and well-differentiated malignant lymphoid infiltration may be difficult and at times somewhat academic, but attempts to classify these lymphoid tumours are important and may prove useful in terms of treatment planning and prognosis. We are not aware of other literature reports of primary mantle cell lymphoma presenting as a choroidal mass. Interestingly, our patient showed no evidence of anterior epibulbar involvement, which is a common feature of ULI, and did have uveitis, which is not common in the early presentation of ULI but common in large B cell NHL involving the eye.

In summary, this patient with biopsy-proven mantle cell orbital lymphoma presented with a choroidal mass and uveitis. Our case considered with other reported cases of ocular lymphoid infiltration highlights the spectrum of lymphoid infiltration that may involve the choroid from reactive lymphoid hyperplasia, through well-differentiated and mantle cell lymphoma to large cell high-grade lymphoid tumours. The full spectrum of lymphoid infiltration of the eye should be considered in the differential diagnosis of a yellow choroidal lesion. Our patient's tumour did not respond to systemic steroids but proved to be very responsive to orbital radiotherapy, and the patient has remained disease-free almost 3 years following radiotherapy. Long-term follow-up is, of course, essential because a further manifestation of lymphoma may present.

References

1. White WL, Ferry JA, Harris NL, Grove AS Jr. Ocular adnexal lymphoma: a clinicopathologic study with identification of lymphomas of mucosa-associated lymphoid tissue type. *Ophthalmology* 1995;102:1994-2006.
2. Nakata M, Matsuno Y, Katsumata N, et al. Histology according to the Revised European-American Lymphoma Classification significantly predicts the prognosis of ocular adnexal lymphoma. *Leuk Lymphoma* 1999;32:533-43.
3. Cahill M, Barnes C, Moriarty P, Daly P, Kennedy S. Ocular adnexal lymphoma: comparison of MALT lymphoma with other histologic types. *Br J Ophthalmol* 1999;83:742-7.
4. Rankin GA, Jakobiec FA, Hidayat AA. Intraocular lymphoproliferations simulating uveitis. In: Albert DM, Jakobiec FA, editors. *Principles and practice of ophthalmology*. Philadelphia: WB Saunders, 1994:524-48.

5. Banks P, Chan J, Cleary M, et al. Mantle cell lymphoma: a proposal for unification of morphologic, immunologic and molecular data. *Am J Surg Pathol* 1992;16:637-40.
6. Ryan SJ, Zimmerman LE. Reactive lymphoid hyperplasia: an unusual form of intraocular pseudotumour. *Trans Am Acad Ophthalmol Otol* 1972;76:652-70.
7. Jakobiec FA, Sacks E, Kronish JW, et al. Multifocal static creamy choroidal infiltrates: an early sign of lymphoid neoplasia. *Ophthalmology* 1987;94:397-406.
8. Ben-Ezra D, Sahel JA, Harris NL, et al. Uveal lymphoid infiltrates: immunohistochemical evidence for a lymphoid neoplasia. *Br J Ophthalmol* 1989;73:846-51.
9. Grossniklaus HE, Martin DF, Avery R, et al. Uveal lymphoid infiltration: report of four cases and clinicopathologic review. *Ophthalmology* 1998;105:1265-73.

Steven A. Rowley¹

Gerald T. Fahy¹

Lawrence J.R. Brown²

¹Department of Ophthalmology

Leicester Royal Infirmary

Leicester, UK

²Department of Histopathology

Leicester Royal Infirmary

Leicester, UK

Gerry T. Fahy, MD, FRCOphth ✉

Department of Ophthalmology

University Hospital

Galway, Ireland

Sir,

Spontaneous resolution of bilateral macular haemorrhage in a patient with kala-azar

Kala-azar is caused by *Leishmania donovani*, a haemoflagellate protozoan endemic in the Mediterranean basin, Asia, Africa (Old World) and South America (New World).¹ Ocular lesions in kala-azar include post kala-azar uveitis,² subacute focal retinitis and retinal haemorrhages,³ in addition to cutaneous leishmaniasis causing ulcerative or interstitial keratitis, ulcerating granulomatous lid lesions and blepharoconjunctivitis.¹ Retinal haemorrhage was first described as one of the ocular manifestations of kala-azar by Ling and Lee in 1924 in 6 Chinese patients.³ Following this report, only three further cases have been reported.⁴

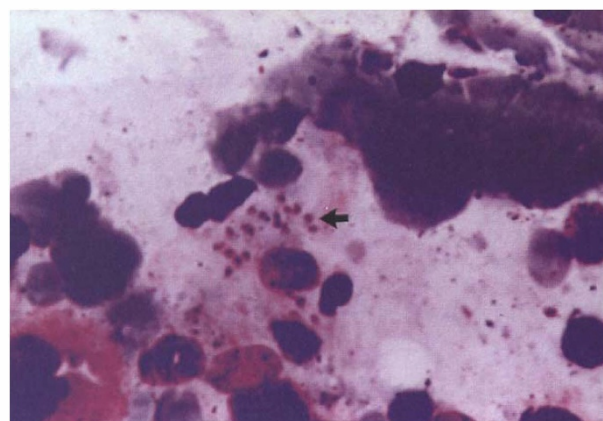
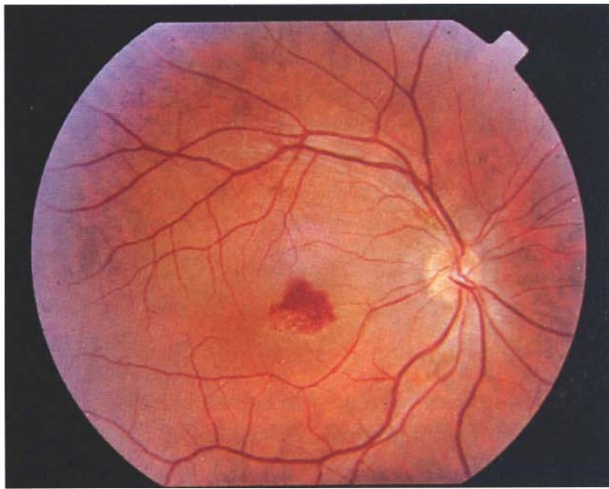
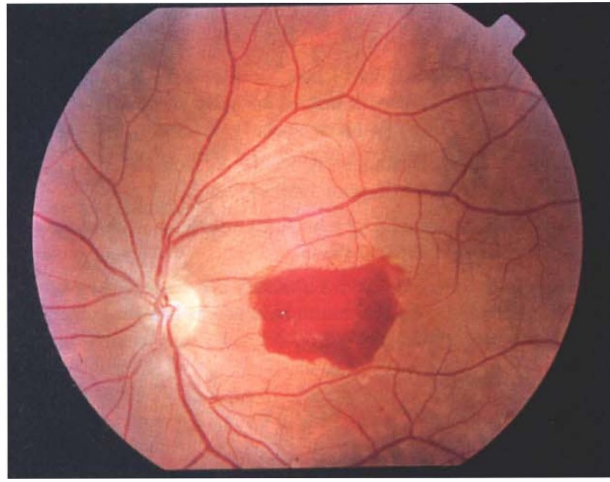


Fig. 1. Microphotograph showing *Leishman-Donovan* bodies in bone marrow aspirate (arrow).



(a)



(b)

Fig. 2. Fundus photographs showing premacular haemorrhage in the right (a) and left (b) eyes.

We now report a patient from Bihar (an endemic area of kala-azar), India, who developed isolated bilateral macular haemorrhage, which resolved spontaneously.

Case report

A 28-year-old man from Bihar, India visited our institute on 28 February 1998 with complaints of dimness of vision in both the eyes of 1½ months' duration. The patient gave a history of fever in October 1997. The patient subsequently had a relapse of fever associated with chills, rigors and dry cough on 15 December 1997. He was initially suspected to have re-occurrence of typhoid; however, the Widal test was negative. Systemic examination revealed fever and moderate hepatosplenomegaly. Investigations for tuberculosis were negative. No malarial parasite was seen. As the patient had a low haemoglobin level (7.6 g%), a bone marrow examination was done, which revealed 3+ Leishman Donovan (L.D.) bodies. The peripheral smear was negative for malarial parasite. The patient was put on intravenous amphotericin B 50 mg/day for 15 days and oral haematinics. Although the fever subsided with the above treatment, the patient developed sudden painless blurring of vision. On examination by a local ophthalmologist he was found to have bilateral macular haemorrhage. The patient was re-evaluated by an internist who did not find any systemic evidence of kala-azar. A splenic aspiration was negative.

Ophthalmic examination at our centre showed his vision to be 6/18 in the right eye and 6/60 in the left eye. Slit-lamp examination revealed no abnormalities. Fundus examination revealed macular haemorrhage 1 1 disc diameters (DD) in size in the right eye and 2 2 DD premacular haemorrhage in the left eye. The patient was afebrile with no hepatosplenomegaly. Haemoglobin was 12 g%, ESR was 32 mm in the first hour, and total and differential leucocyte counts were normal. Peripheral smear revealed absence of L.D. bodies and abnormal

blood cells. Coagulation tests were normal. The patient was offered no treatment and explained the chance of spontaneous resolution of macular haemorrhage.

At the last follow-up after 11 months the patient had a visual acuity of 6/6 in the right eye and 6/9 in the left eye. Fundus examination of the right eye revealed complete resolution of haemorrhage over the macula leaving a little fine pigmentation over the macula. Fundus examination of the left eye also showed complete resolution of the retinal haemorrhage with only a few specks of pigmentation in the parafoveal area.

Comment

In tropical countries the common causes of macular haemorrhage are malaria, kala-azar and acute anaemia, especially megaloblastic anaemia. The cause of haemorrhage in a case of kala-azar can be secondary to thrombocytopenia, a fall in the fibrinogen level in the plasma, or an increase in fibrinolytic activity in the plasma.⁴ There is also a possibility of septic retinitis in the active stage of the disease causing fragility of superficial capillaries leading to retinal haemorrhage. De Cock *et al.*⁴ found that retinal haemorrhages resolve spontaneously with restoration of vision. An unusual aspect in the presentation of this patient is that the haemorrhages involved only the macular area and occurred in the convalescent stage of kala-azar (which was proved by a negative splenic aspirate). Treatment for such haemorrhage is observation for spontaneous resolution. In case such resolution does not occur, YAG or argon laser posterior hyaloidotomy of premacular haemorrhage can be attempted.

Although kala-azar is endemic in certain parts of India, an increase in travel and migration can result in presentation of such cases to ophthalmologists elsewhere in the country and abroad. Awareness of this rare cause of macular haemorrhage can ensure proper diagnosis and management.

References

1. Kean BH, Sun T, Ellsworth RM. Leishmaniasis. In: Kean BH, Sun T, Ellsworth RM, editors. Colour atlas and textbook of ophthalmic parasitology. New York: Igahu-Shoin, 1991:60–5.
2. Dechant W, Rees PH, Kager PA, Klauss V, Adala H. Post kala-azar uveitis. Br J Ophthalmol 1980;64:680–3.
3. Tassman WS, O'Brien DD, Hahn K. Retinal lesions in kalaazar. Am J Ophthalmol 1961;50:161–3.
4. De Cock KM, Rees PH, Klauss V, Kasili EG, Kager PA. Eeftink Schattenkerk JKM. Retinal haemorrhages in kala-azar. Am J Trop Med Hyg 1982;31:927–30.

Jyotirmay Biswas ✉

Baskaran Mani

Muna Bhende

Medical and Vision Research Foundation

18 College Road

Chennai 600 006, India

Tel: +91 8271616, +91 8271036

Fax: +91 44 8254180

e-mail: MDSAAA@giasmd01.vsnl.net.in

Sir,

Squamous cell carcinoma of the conjunctiva as initial presenting sign in a patient with acquired immunodeficiency syndrome (AIDS) due to human immunodeficiency virus type-2

Conjunctival squamous cell dysplasia and neoplasia have been associated with HIV infection and AIDS in the sub-Saharan African population.¹ There has been a 6-fold rise in the incidence of conjunctival malignancy following the epidemic of HIV infection reported in Uganda.² Ocular involvement in AIDS was first described in India in 1995; of the two patients, one had cytomegalovirus retinitis and the other had endogenous endophthalmitis.³ In India there is currently a rapid increase in patients with various ocular manifestations of AIDS. In a series reported by us 45.7% had ocular lesions, the most common being cytomegalovirus retinitis (21.4%). Extraocular lesions were seen in only 4 cases (5.7%).⁴ Ocular involvement was lower than in similar studies in the USA (65%) and Africa (55%).

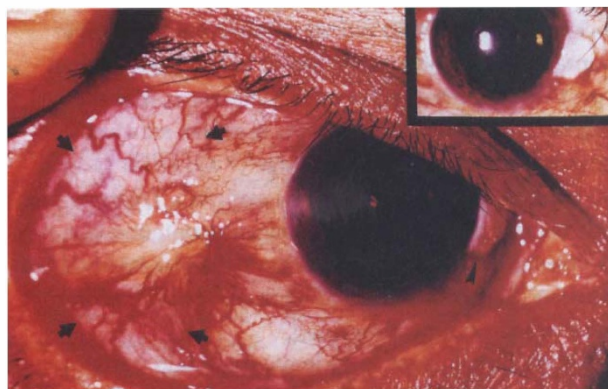


Fig. 1. The mass lesion in the temporal part of the bulbar conjunctiva in the right eye (arrow). Note the multiple dilated conjunctival vessels (patient looking towards the left). An elevated triangular leukoplakic patch was seen on the nasal part of the bulbar conjunctiva (single arrow and inset).

We describe a healthy young patient presenting with leukoplakia and a conjunctival mass as the initial manifestations of HIV infection. Subsequent histopathological study revealed conjunctival dysplasia on the nasal side and squamous cell carcinoma on the temporal bulbar conjunctiva. This is the first report of squamous cell carcinoma of the conjunctiva occurring in AIDS in India.

Case report

A 34-year-old man presented in September 1998 with a 7 month history of a progressive swelling in the right eye associated with redness, watering and diplopia on right gaze. He had received treatment for pulmonary tuberculosis due to *Mycobacterium tuberculosis* 3 years previously and for herpes zoster skin infection 6 years earlier. On examination, his best corrected visual acuity was 20/20 in both eyes. External examination of the right eye revealed conjunctival congestion, an elevated triangular leukoplakic patch at the 4 o'clock limbus (Fig. 1, inset) and a 15 mm × 12 mm mass in the temporal bulbar conjunctiva 4 mm from the limbus, with prominent vessels (Fig. 1). The mass had a firm consistency, was non-tender and fixed to the globe. Intraocular pressure was 16 mmHg and fundus examination revealed the indentation effect produced by the mass lesion at the temporal ora serrata. The left eye was normal.

Ultrasonography of the right eye revealed a well-circumscribed homogeneous mass of low reflectivity measuring 16 mm × 11 mm, situated in the anterior orbit anterior to the insertion of the lateral rectus muscle. Indentation of the globe wall was noted, but the sclera was not infiltrated. The other ocular structures were normal. CT scan did not reveal any changes in the bony wall of the orbit. The patient underwent excision of the leukoplakic lesion and an incisional biopsy of the mass lesion.

Histopathological study of the leukoplakic lesion revealed hyperkeratosis, parakeratosis, acanthosis and loss of polarity of epithelial cells, indicating dysplasia of the conjunctival epithelium (Fig. 2, inset). On histopathological examination the conjunctival mass lesion contained scattered clusters of malignant squamous cells and vascular channels with sparse infiltration of lymphocytes (Fig. 2). In view of the rarity of squamous cell carcinoma in immunocompetent patients, underlying HIV infection was suspected. ELISA testing revealed positive antibody to HIV-2, and Western blot test confirmed the ELISA finding. Total white blood cell count was 7200/mm³, absolute CD4 count was 324/μl, CD8 count was 578/μl, and the CD4/CD8 ratio was 0.56. On questioning, the patient disclosed a positive history of unprotected sexual exposure with a commercial sex worker in 1988. The patient was examined in an AIDS Care Centre by an internist and no systemic opportunistic infection was found.