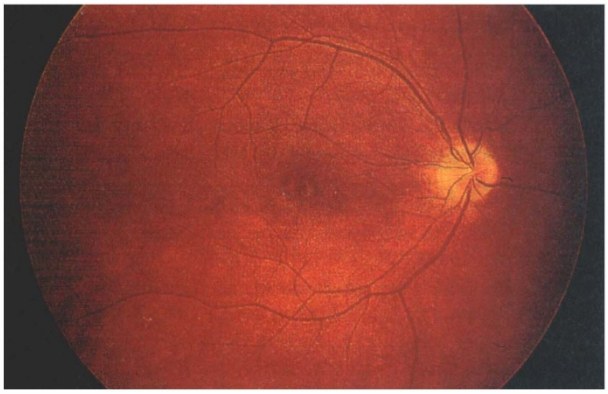
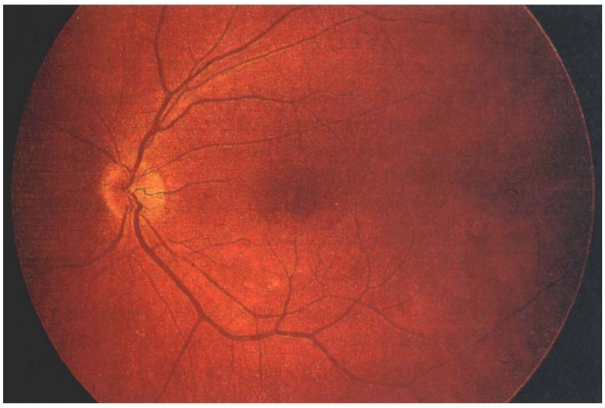


(a)



(b)

Fig. 2. *Five months after presentation. (a) The right optic atrophy has developed and the arteriolar changes have reversed. b) The haemorrhage, cotton wool spot and arteriolar narrowing have all reversed in the left fundus.*

Comment

Accelerated hypertension can cause a retinopathy, choroidopathy and optic disc swelling. Clinical presentation is variable, reflecting the severity and speed of onset of the hypertension as well as the presence of pre-existing vascular disase. Accelerated hypertension produces changes in the autoregulation of the retinal and optic nerve head circulation. Autoregulation maintains a relatively constant blood flow during changes in perfusion pressure. The retina and optic nerve head autoregulate their circulation while the choroidal vascular bed does not.[1]

Optic nerve head ischaemia is produced by a combination of two factors: (i) involvement of the peripapillary choroid by vasoconstriction and vaso-occlusive changes, and (ii) diffusion of angiotensin II and other endogenous vasoconstrictors into the optic nerve head from the peripapillary choroid. Disc oedema is thought to occur due to ischaemia interfering with axoplasmic flow in the optic nerve head.[2]

Hayreh's studies[3] on experimental renovascular accelerated hypertension in rhesus monkeys showed that hypertensive optic neuropathy is a distinct entity, that it is not simply a part of hypertensive retinopathy and that it represents one form of anterior ischaemic optic neuropathy. Talks *et al.*[4] suggested that during the acute stage of accelerated hypertension the ischaemic optic neuropathy may be reversible. Reversibility is clearly demonstrated by this case.

The regulatory mechanisms controlling optic nerve circulation are not fully understood. Factors affecting the reversibility of anterior ischaemic optic neuropathy in this patient may include age, lack of coexisting disease, early presentation and immediate treatment. The absence of any previous history of hypertension in this patient emphasises the importance of checking the blood pressure in all patients with anterior ischaemic optic neuropathy. Precipitous reduction of the blood pressure in patients with hypertensive optic neuropathy may cause complete permanent blindess.[5]

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M.J. Gallagher
R.S. Haslett
R.P. Phillips
Arrowe Park Hospital
Wirral, UK

R.S. Haslett, BM, MRCP, FRCOphth ✉
Arrowe Park Hospital
Arrowe Park Road
Upton
Wirral
Merseyside L49 5PE, UK

Tel: +44 (0)151 678 5711
Fax: +44 (0)151 604 7152

Sir,

External ocular myiasis due to *Oestrus ovis* in a tourist returning from North Africa

The sheep nasal botfly *Oestrus ovis* is the commonest of several species of dipteran fly whose larvae are obligatory parasites in the nasal cavities and frontal sinuses of sheep but may cause infestation (myiasis) in man. Ocular myiasis is rarely reported in the UK. We report a case of imported external ocular myiasis due to *O. ovis* presenting in London.

Case report

On her return from North Africa a 42-year-old female tourist presented to the accident and emergency department of St George's Hospital, London, with a 3 day history of a painful red watery left eye. Her symptoms had begun while walking on a beach in Tunisia, when she reported a sudden onset of foreign body symptoms that she had presumed to result from sand blown into her eye. On examination there was conjunctival hyperaemia with moderate papillae and a mild punctate keratitis in the left eye. On tarsal eversion a single motile larva, measuring 1 mm in length, was observed on the palpebral conjunctiva in the superior fornix. Ocular examination was otherwise normal. The larva was removed with a sterile cotton bud and identified by light microscopy as the larva of the sheep nasal botfly *Oestrus ovis*. The patient was prescribed g. chloramphenicol 0.5% q.d.s. to the affected eye. Her symptoms and signs had resolved 3 days later.

Comment

The larvae of *Oestrus ovis* are hatched from their eggs in the vagina of the adult female, who ejects them into the nostrils of sheep[1] in a stream of milky white fluid, possibly without direct contact.[2] Migration to the frontal sinuses is followed by maturation for 8–12 months. The larvae are subsequently sneezed out and pupate on the ground for a period of 3–6 weeks. The lifespan of the adult fly is about 4 weeks. The human is an incidental host and becomes involved when larvae are ejected onto the ocular surface instead of the nasal mucosa of sheep. In humans the larvae are unable to mature and survive for up to 10 days. Sudden onset of foreign body symptoms is followed by pain and inflammation. A punctate keratitis is common and small conjunctival haemorrhages may be seen. Single or multiple larvae are observed in the conjunctival sac. The condition is normally benign and self-limiting.[3] Invasion of the orbit or globe, more typical of other species, is rarely reported due to *O. ovis*[4] but the resulting panuveitis may be severe. Management of external ocular myiasis involves careful removal of the organisms with forceps aided by topical anaesthetic to slow their motility. Topical administration of corticosteroids for symptomatic relief and antibiotics to prevent bacterial contamination have been recommended.[3]

Oestrus ovis is widespread in Africa and the Middle East, where the annual incidence of ocular myiasis is estimated to be 10 per 100 000,[5] but is also reported in Australia,[6] North America,[3,7] and Southern Europe.[8] Reports of external ocular myiasis due to *O. ovis* in the UK, either indigenously acquired[9,10] or imported,[11] are rare. A history of recent travel to endemic areas should prompt a high index of suspicion and careful examination of the conjunctival fornices for larvae.

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James Bainbridge
Chad Rostron
Department of Ophthalmology
St George's Hospital
London, UK

James W.B. Bainbridge, MA, FRCOphth ✉
Department of Ophthalmology
Charing Cross Hospital
London W6 8RF, UK

Sir,

Marked thickening of Bruch's membrane in a 17-year-old patient with angioid streaks

Angioid streaks represent linear, narrow subretinal streaks radiating several millimetres from a peripapillary ring. They appear around the second decade of life, and are accompanied by a high incidence of macular degeneration at a young age.[1] The earliest angioid streaks can be detected at age 8 years with findings of narrow, short, radial, discontinuous hypopigmented streaks.[2] Thereafter angioid streaks enlarge in length and width. The end stage is disciform macular degeneration, helicoid peripapillary atrophy or diffuse choroidal sclerosis.[3,4] Little is known about the histological findings in young eyes with angioid streaks. We present the youngest patient with angioid streaks whose eyes were examined at autopsy.

Case report

A 17-year-old white man of Yugoslavian extraction was admitted in January 1968 because of a coma of several hours' duration. Past medical history revealed normal