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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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

growth until age 5 years, when he developed lower extremity weakness and was found to have multiple bone deformities compatible radiologically with Paget's disease of bone. Systemic blood pressure was mildly elevated. The bony deformities progressed further at age 9 years. Antihypertensive medication was started at age 10 years.

He was lost to follow-up until the age of 17 years when he presented to the Montefiore Medical Center with visual loss in the left eye. Angioid streaks and subretinal macular haemorrhages were noted bilaterally. He was not taking the hypertensive medication and his blood pressure was 200/170 mmHg. The patient and his family refused hospitalisation. Two months later he was brought to the emergency room in a coma from a cerebral bleed (bloody cerebrospinal fluid) and malignant hypertension. He died 8 h after admission.

Clinicopathological correlation

At autopsy, an external examination revealed the typical gooseflesh skin of pseudoxanthoma elasticum. No skin biopsy was performed. A piece of optic nerve measuring 22 mm with attached posterior pole was obtained via an intracranial route. The specimen was fixed in 10% formaldehyde and embedded in paraffin. Light microscopic examination of the haematoxylin and eosin section showed extreme and uniform thickening of Bruch's membrane (Fig. 1). This thickening was accentuated compared with an age-matched eye with no ocular or systemic problems (Fig. 2). In the posterior pole, the thickness of Bruch's membrane was 2.5 μm in the control eye versus 7.4 μm (3 times thicker) in the eye with angioid streaks. There were no breaks in Bruch's membrane in the available sections. Sections through the optic disc and angioid streaks were not available. The choriocapillaris, retinal pigment epithelium and retina were unremarkable. The paraffin block was not available for further studies.

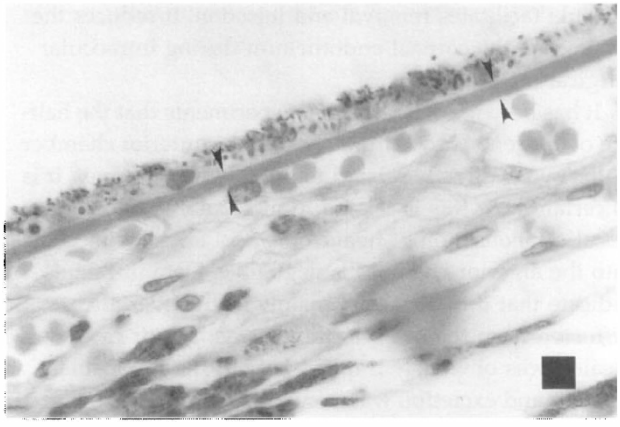

Fig. 1. *Section through the posterior pole of the eye of the 17-year-old man with angioid streaks. There is severe thickening of Bruch's membrane (arrowheads). The choriocapillaris is patent. H&E. Scale block represents 15 μm.*

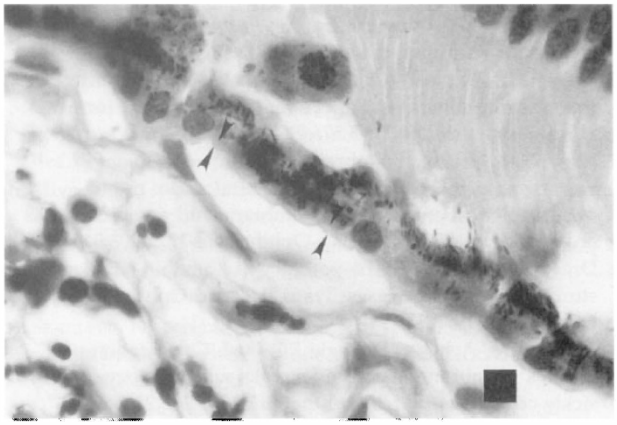

Fig. 2. *Section through the posterior pole of a control eye of the same age reveals a very thin Bruch's membrane (arrowheads). H&E. Scale block represents 15 μm.*

Discussion

The present case demonstrates severe thickening of Bruch's membrane in a 17-year-old man with angioid streaks. Histopathologically, angioid streaks correspond to a full-thickness break in Bruch's membrane with disruption of the underlying choriocapillaris and atrophy of the overlying retinal pigment epithelium.

There have been five pathological studies in angioid streaks, mostly in elderly subjects.[5–9] The first histological case was described in 1948,[5] followed by one case with Paget's disease in 1973,[6] one case with pseudoxanthoma elasticum in 1977,[7] 21 cases in 1978,[8] and one case with sickle cell disease in 1987.[9]

Bruch's membrane in angioid streaks is thickened and basophilic from deposition of calcium.[7–9] By elemental analysis, the two kinds of calcification consist of hydroxyapatite and $CaHPO_4$.[7] The deposition of calcium confers an egg-shell brittleness on Bruch's membrane that is modulated by the axis of the optic nerve insertion, resulting in radiating cracks on Bruch's membrane.[2,3] Bruch's membrane measures 1–2 μm at the periphery and 2–4 μm in the peripapillary area.[10] It thickens with age with the accumulation of debris, starting as early as the second decade of life.[11] Visible thickening of Bruch's membrane in the posterior pole begins after age 60 years and can reach 2–3 times its original thickness.[12–15] The present case demonstrates severe thickening of Bruch's membrane in a 17-year-old man with angioid streaks. Thickening of Bruch's membrane may be one of the early histological changes in angioid streaks.

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Sir,

Post-operative myopic shift due to trapped intracapsular Healon

We describe a rare post-operative complication caused by Healon trapped within the capsular bag behind a posterior chamber intraocular lens. This resulted in a shallowed anterior chamber and a myopic shift, both of which were corrected by removal of the trapped Healon.

Case report

A 76-year-old woman complaining of blurred vision in her left eye was diagnosed as having a cortical cataract. Her visual acuities were 6/12 right and 6/18 left. Anterior and posterior segment examination was otherwise unremarkable and in particular she had normal anterior chamber depths (right 2.47 mm, left 2.55 mm). The patient agreed to a left cataract extraction with intraocular lens implantation. Her pre-operative refraction was +3.75/−2.50 in her right eye and +1.50/−1.75 in her left eye. The aim was to make the left eye emmetropic and the posterior chamber lens needed to achieve this was a 23 dioptre lens. An uneventful left phacoemulsification using a foldable intraocular implant (8590B Chiron) inserted under sodium hyaluronate 5000 (Healon) was carried out after a continuous circular capsulorhexis. The Healon was aspirated from in front of the implant at the end of the operation.

Post-operatively she had a visual acuity of 6/6 and a refraction of −1.75/−1.75. A shallow anterior chamber was noted. A greater than normal distance between the posterior surface of the intraocular lens and the surface of the posterior capsule was also noted. The shallow anterior chamber, distended capsular bag and refraction remained unchanged over the following 2 months. It was decided that the Healon needed removing from the capsular bag and that this should be done surgically as the capsulorhexis edge was obscured by the pupil margin (even when the pupil was dilated). The Healon was aspirated after inserting a cannula into the capsular bag between the edge of the intraocular lens optic and the capsulorhexis margin. The procedure was uneventful.

Post-operatively the patient's vision remained at 6/6, but the anterior chamber deepened, the capsular bag distension disappeared and her refraction improved to −0.25/−0.50. At no stage was there excessive intraocular inflammation or raised intraocular pressure and the Healon remained clear throughout this 2 month period.

Comment

Hyaluronic acid is a naturally occurring glycosaminoglycan which, due to its viscosity, elasticity and other properties, acts as an ocular lubricant and an anterior chamber depth maintainer. Formulations of exogenous hyaluronic acid for clinical use are derived from the dermis of rooster combs or by fermentation in streptococcal cultures. The physical properties of hyaluronic acid and its use in ophthalmic surgery have been described in several detailed reviews.[1–4] The high viscosity when stationary permits maintenance of depth and shape of the anterior chamber and manipulation of tissues, and the low viscosity when forced through a cannula facilitates removal and injection. It reduces the damage to the corneal endothelium during intraocular surgical manipulation.

It has been shown in animal experiments that the half-life of exogenous hyaluronic acid in the anterior chamber of the eye is directly related to the injected volume.[5] It is uncertain how this is related to molecular weight.[6,7] Local catabolisation of hyaluronic acid after its injection into the anterior chamber is negligible. Data in rabbits indicate that it is degraded mainly in the liver after diffusion from the eye into the plasma. In our case the Healon was obviously trapped behind the intraocular implant and excretion was occurring extremely slowly, if at all, therefore requiring its removal. Shammas[8] and Holtz[9] have also described similar patients with post-operative myopia, shallow anterior chambers and distended posterior capsular bag due to trapped Healon.