of this particular association, the mass also involved the cornea and caused significant astigmatism.

CASE REPORT

A 66-year-old male farmer noticed a red mass that grew rapidly within 2 weeks on his left eye. He also reported that his left visual acuity decreased progressively during the same period. He did not have any previous ocular surgery and did not recall any prior complaints related to his eyes.

On examination, the best corrected visual acuity was 20/40 in the right eye and 20/100 in the left. He had moderate nuclear sclerotic cataracts in both eyes. The right eye was otherwise normal. There was a non-tender, soft, reddish cystic mass on the nasal conjunctiva of the left eye (Fig 1). The surface of the mass was minimally keratinised due to exposure. There were dilated and distended blood vessels over the tumour. The mass measured 20 × 18 × 22 mm and protruded through the palpebral fissure preventing the closure of the eyelids. The tumour was attached to an underlying pterygium and extended about 4 mm onto the cornea. Transillumination confirmed the cystic nature of the tumour. Keratometry gave measurements of 50D at 35 degrees and 41D at 135 degrees.

During the excisional biopsy, the cyst could be separated easily from the pterygium that formed its base. The pterygium was also excised. The conjunctiva was then primarily closed. Postoperatively, his best corrected visual acuity rose to 20/40 in the left eye and the irregular corneal astigmatism disappeared.

Histopathologically there was one large cystic cavity lined by single or more layered flattened epithelium in which rare goblet cells were observed (Fig 2). The squamous nature of the lining epithelium could be observed focally. Few chronic inflammatory cells were scattered under the lining epithelium and substantia propria. The surface epithelium displayed hyperplastic changes. A clinicopathological diagnosis of conjunctival epithelial inclusion cyst spontaneously arising from a pterygium was made.

COMMENT

Epithelial inclusion cysts are frequently encountered benign conjunctival lesions. However, they can, rarely, involve the limbus or cornea.² In one large survey of biopsy proved lesions of the conjunctiva in an adult population, epithelial cysts comprised 22.5% of all acquired epithelial lesions and they constituted 80% of all cystic lesions.³ They occurred with almost equal frequency in men as in women and at an average age of 47 years.³

It is believed that conjunctival epithelial cysts can occur in the conjunctiva following

the stagnation of mucus in epithelial spaces formed by amalgamations of mucosal folds that result from irregularly elevated surface epithelium in inflammatory conditions.⁴ This may explain the development of an inclusion cyst over a pterygium as was the case in our patient. These cysts have also been reported to arise from dislocated epithelium below the surface of the conjunctiva or cornea secondary to trauma⁵ or scleral buckling surgery.⁶ Most cysts remain small and asymptomatic but occasionally they may reach large proportions and cause significant corneal astigmatism and visual impairment.⁷

Treatment consists of excisional biopsy, cryotherapy, or cauterisation with trichloroacetic acid.⁸ Nd-YAG laser has also been shown to be effective for acquired inclusion cysts of the conjunctiva.⁹

Conjunctival epithelial inclusion cysts can occur spontaneously over a pterygium and thus involve secondarily the cornea causing significant functional impairment.

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Figure 2 Histopathological section of the cyst showing the surface covered by multilayered squamous epithelium (short arrow). There is marked oedema in the stroma with congested veins and a few scattered inflammatory cells (long arrow). The cavity is lined by flattened squamous epithelium (double arrows) (haematoxylin and eosin, × 10).

Figure 1 A smooth, cystic mass on the nasal bulbar conjunctiva extending on to the cornea of the left eye. The surface vessels are engorged.

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Racemose haemangioma of the iris

EDITOR,—Benign vascular tumors are well known to occur in the retina and choroid where they can be classified into capillary, cavernous, and racemose types.^{1,2} Vascular neoplasms of the iris are very rare and most reported cases have been of the cavernous type. We report our observations of a patient with an arteriovenous lesion in the iris compatible with a racemose haemangioma.

CASE REPORT

An asymptomatic 40-year-old man was found on routine examination to have a vascular lesion in the iris of his left eye. His past medical history, family history, and systemic findings were entirely normal. Specifically he had no history or findings compatible with Wyburn-Mason syndrome, the von Hippel-Lindau syndrome, or other systemic vascular entities. There was no history of systemic or ocular vascular problems.

Slit-lamp biomicroscopy revealed three blood vessels that extended from the anterior chamber angle temporally to terminate in a small complex of intertwining vessels on the temporal aspect of the pupil. Although the vessels could be easily visualised with slit-lamp biomicroscopy, they were difficult to document photographically because they were in the iris stroma. However, fluorescein angiography demonstrated the vascular lesion more clearly. The central vessel was a distinct feeding artery that passed from the peripheral portion of the iris at the 3 o'clock location (Fig 1) and rapidly fed the complex mass of vessels near the pupillary margin. Two distinct veins, one at 2 o'clock and one at 4.30 o'clock, appeared to drain from the lesion and exit into the anterior chamber angle (Fig 2). There was no detectable leakage of fluorescein from the blood vessels. The diagnosis was racemose haemangioma of the iris. The lesion has remained stable for about 12 months since it was initially observed.

COMMENT

Intraocular haemangiomas usually occur in the retina or the uveal tract. Retinal haemangiomas can be subdivided into capillary, cavernous, and racemose, and acquired non-familial types, each of which has distinctive ophthalmoscopic features and systemic associations.¹ Uveal haemangiomas occur almost exclusively in the posterior choroid and are mostly of the cavernous type.²

Haemangiomas of the iris are generally considered to be quite rare. In 1972, Ferry³ obtained and reviewed histopathological sections of reported cases of iris haemangioma and found that virtually all of them had been misdiagnosed histopathologically. Many of the reported cases were found to be highly vascularised melanomas, juvenile xanthogranulo-

mas, and inflammatory granulomas. Ferry questioned the existence of iris haemangioma.³

A review of the literature suggests that there are some well documented cases of both cavernous and capillary haemangioma of the iris. The cavernous haemangioma, or microhaemangioma, which is more common, appears as a venous tuft at the pupillary margin. It can bleed and produce a spontaneous hyphaema.⁴ Capillary haemangioma has also been reported to occur in the iris. This rare tumour can be associated with a cutaneous capillary haemangioma and it can show spontaneous resolution coincidental with the natural regression of associated cutaneous lesions.⁵

To our knowledge, racemose haemangioma has not been previously reported to affect the iris. The lesion that we report here appears to be entirely compatible with a racemose haemangioma as seen in the retina.⁶ In contrast with a cavernous haemangioma, a racemose haemangioma looks like a small cluster of grapes and fills slowly with fluorescein. The racemose haemangioma of the retina is actually a complex arteriovenous communication that fills rapidly with fluorescein but does not usually show appreciable leakage of fluorescein. Our case in the iris showed similar angiographic features.

Racemose haemangioma of the retina can be associated with the Wyburn-Mason syndrome, characterised by arteriovenous communications in the midbrain, mandible, maxilla, and other adjacent structures. However, our patient, with an identical vascular lesion in the iris, did not have any clinical manifestation

of that entity. Since our patient is entirely asymptomatic, he has not been inclined to undergo additional studies to detect the presence of other arteriovenous communications.

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'Creeping' cytomegalovirus retinitis in AIDS

EDITOR.—Cytomegalovirus retinitis (CMVR) is the most common sight threatening infection in AIDS. Despite antiviral therapy, recurrent infection is common and leads to progressive visual loss. The classic signs of uncontrolled CMVR include extension of existing lesions, the occurrence of new lesions, fresh haemorrhage or necrosis at the borders of atrophic lesions, or new perivascular infiltrates—early recognition of these signs should prompt increased antiviral therapy. 'Smoldering' CMVR has been described as slow but definite progression of the borders of previously inactive retinitis, and the only sign of activity may be a thin white or grey line between atrophic and unaffected retina.¹ Conversely, border opacification may persist on the edge of healed CMVR without

Figure 1 Retinal vascular closure in the left fundus of patient 1 which progressed over a 3 month period without evidence of retinal opacification.

advancement,² and this should not be confused with active infection. Close attention to the border site, serial retinal drawings, and fundus photographs will confirm movement of the border; however, this may be difficult if the retinal vascular landmarks become obliterated or if border activity is absent.

We report on two patients with CMVR as the AIDS defining diagnosis who exhibited recurrent infection over a prolonged period characterised by progressive retinal vascular closure and retinal atrophy with very minimal sign of border activity.

CASE REPORTS

A 39-year-old HIV positive woman complained of blurred vision in her right eye. The ophthalmoscopic appearance of CMVR at the right posterior pole prompted induction intravenous therapy with ganciclovir at 10 mg/kg for 2 weeks. Severe neutropenia interrupted maintenance therapy allowing CMVR progression. Eighteen months later CMVR was diagnosed in the left eye. Despite intravenous foscarnet therapy, progression continued over a 9 month period with little clinical evidence of border activity (Fig 1). She died more than 2 years after diagnosis of CMVR.

A 30-year-old HIV positive heterosexual man complained of floaters in the left eye. Ophthalmoscopy revealed foci of CMVR in the right peripheral nasal retina and left inferior hemiretina. Intravenous induction ganciclovir therapy failed to prevent progression; therefore, intravenous induction foscarnet was used. Progressive retinal scarring and enlarging bilateral scotoma were documented over an 18 month period with minimal clinical evidence of border activity (Fig 2). Foscarnet toxicity during the maintenance phase led to periods of subtherapeutic dosing during this

Figure 1 Fluorescein angiogram at 26.4 seconds after injection, showing filling of the prominent artery at the 3 o'clock position temporally. Note that the area adjacent to the artery is relatively void of blood vessels.

Figure 2 Fluorescein angiogram at 30.9 seconds showing filling of the vascular complex temporal to the pupillary margin and beginning filling of the two draining veins. There was no leakage of dye from the blood vessels in the late angiograms.

Figure 2 Progressive retinal vascular closure in the left fundus over a 6 month period. Minimal retinal opacification was only observed after creeping disease had been present for a further 8 months. The arrowhead indicates the position of a major arteriole for comparison.