resultant disseminated intravascular coagulation. This may be responsible for choroidal ischaemia and consequent serous retinal detachment.[7]

Spontaneous serous retinal detachment in toxaemia of pregnancy can occur both ante partum and post partum causing marked reduction in visual acuity. In most cases the detachment resolves with a return to normal visual function within the first few weeks post partum. However, some patients may develop residual macular retinal pigment epithelial change, which may represent areas of infarction of the choriocapillaries (Elschnig's spots). These changes can mimic a macular dystrophy or tapetoretinal degeneration and infrequently may result in permanent visual impairment.[8]

Our case points out that retinal detachment should always be considered within the differential diagnosis of sudden loss of vision in cases complicated with HELLP syndrome and more generally toxaemia of pregnancy.

It is useful for the ophthalmologists to be aware that retinal detachment may present in the absence of other hypertensive retinal changes.

Finally the favourable prognosis and natural course of this clinical condition should always be emphasised to the patient relieving them from unnecessary distress.

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Sir,

Vitreous amyloidosis and secondary glaucoma—a case report

Eye (2002) **16,** 492–494. doi:10.1038/sj.eye.6700075

We present a case of familial ocular amyloidosis currently without systemic involvement. Ocular features included vitreous opacities and secondary glaucoma. The patient had a transthyretin mutation (TTR-glycine 54) previously reported in his family only.

Case report

A 38-year-old man presented to Eye Casualty with a 7-week history of floaters in both eyes. The patient's father and paternal uncle both died from systemic complications of amyloidosis. His paternal grandmother was blind 20 years prior to her death. The family history is plotted in Figure 1.

The visual acuities were 6/5 and 6/9 in the right

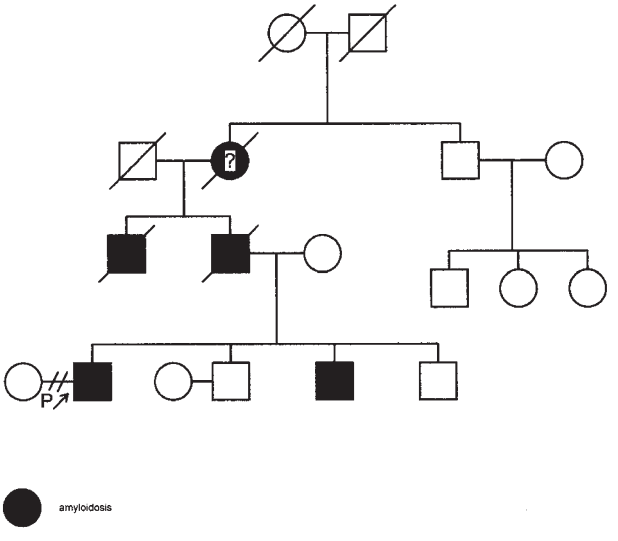

Figure 1 Gene sketch of the family.

and left eye respectively. The anterior segment was normal with open angles on gonioscopy. The intraocular pressures were raised (40 mmHg in the right and 42 mmHg in the left eye). Dilated fundoscopy revealed thin, wispy vitreous opacities more prominent in the left eye, and bilateral posterior vitreous detachments, Figure 2. The optic discs were cupped, C/D ratio of 0.6 in the right eye and 0.7 in the left eye. Threshold visual fields showed early glaucomatous changes. The intraocular pressure was controlled with a topical beta-blocker and latanoprost.

A mutation was found in the Transthyretin gene (TTR-glycine 54), which is capable of producing amyloidosis.

Ten months after presentation the visual acuity had dropped to 6/24 in the right eye and counting fingers at 0.5 m in the left eye. The fundal view also deteriorated due to increased density of vitreous opacities. The intraocular pressures remained under control with topical medication. The patient was offered a vitrectomy but has so far declined further treatment.

Comment

Amyloidosis is a disorder characterised by deposition of amyloid protein in one or more tissues of the body. Ocular involvement is often familial, and may occur with or without systemic abnormalities.

Currently, the disease is sub-classified according to clinical features, ancestry or geographic origin, into four types of Familial Amyloidotic Polyneuropathy (FAP). Type I FAP is associated with polyneuropathy starting in the lower limbs and severe autonomic dysfunction. Type II FAP is associated with polyneuropathy in the lower limbs and mild autonomic dysfunction (Swiss–German families). Type III FAP polyneuropathy is associated with renal failure and cranial neuropathy. Type IV FAP is associated with lattice corneal dystrophy II (Finnish, Irish, American and Japanese families).[1] Types I and II are associated with transthyretin variants; type III is related to apolipoprotein A1 and type IV is related to variants of gelsolin.

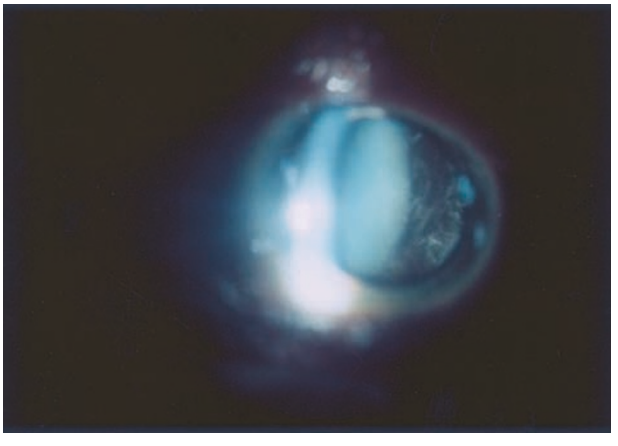

Figure 2 Thin wispy cotton wool-like opacities in the vitreous, left eye.

Eye involvement occurs most commonly with Type I FAP. The main ocular manifestations are: abnormal conjunctival vessels, keratoconjunctivitis sicca due to lacrimal gland infiltration or autonomic neuropathy and pupillary abnormalities including anisocoria and sluggish reactions to light or accommodation. Less common features include vitreous opacities and secondary glaucoma.[1]

Transthyretin is a tetrameric plasma protein (prealbumin), synthesised in the liver, it polymerises into a β-pleated structure of amyloid fibril. TTR is also synthesised by the retinal pigment epithelium[2] and in the choroid plexus of brain. Different mutations in the TTR gene lead to the various hereditary forms of amyloidosis. More than 40 mutations of the TTR gene have been identified but they are not all pathogenic. The most frequent mutation is TTR-met30[3] (methionine replacing valine at position 30 in transthyretin). Our patient, and other affected members of his family, have a rare mutation,[4] TTR-glycine 54.

Isolated vitreous amyloidosis is rare,[5] it is usually associated with CNS manifestations.[6] The appearance of the vitreous has been likened to cotton wool or lace-like veils. The density of these opacities determines the visual acuity and symptoms. Vitreous opacities in contact with the posterior lens surface may form footplates or 'pseudopodia lentis'. Vitreous amyloid is immunologically related to TTR in patients of different FAP kindreds.[7] Results of vitrectomy have in general been satisfactory, but recurrences occur (Doft *et al*).[8]

In FAP type I, phenotypic differences in age of onset, location and severity of lesions occur despite biochemical similarities in different kindreds. Vitreous involvement occurs earlier and is more severe in Portuguese families compared to Swedish or Japanese families with the same TTR-met30 mutation. As one might predict, vitreous involvement is more severe in homozygotes than heterozygotes with TTR mutations (Sandgren *et al*).[9]

Glaucoma in FAP may be due to amyloid infiltrating the trabecular meshwork thus reducing the outflow, or due to raised episcleral venous pressure (Nelson, 1998).[10] Amyloid has been found on histopathological examination of the conjunctiva and trabecular meshwork. Filtration surgery is often necessary because the glaucoma tends to be resistant to topical therapy.[8]

We have presented a case of type I Familial Amyloidotic Polyneuropathy. The underlying transthyretin mutation (TTR-glycine 54) has not been reported in any other family. The case illustrates the importance of taking a careful family history.

Acknowledgements

We thank Professor PN Hawkins PhD FRCP (Clinical Director, National Amyloidosis Centre, Department of Medicine, Royal Free Campus, London).

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Sir,

Secondary posterior chamber intraocular lens implantation in Fuchs' heterochromic uveitis

Eye (2002) **16,** 494–496. doi:10.1038/sj.eye.6700063

In almost all patients with uveitis requiring cataract surgery, phakoemulsification with intraocular lens (IOL) implantation is the procedure of choice.[1,2] The need for stringent peri-operative control of uveitis is well-documented.[3] Some eyes however, will not tolerate IOL implantation and in some it is not possible accurately to predict postoperative reactions, including severe or persistent uveitis, cyclitic membrane formation and phthisis. Sometimes explantation of an IOL becomes necessary, which may be problematic.[4] During the 1980s many surgeons, because of such potential problems, chose not to implant IOLs in eyes with uveitis on the grounds of safety.

Fuchs' heterochromic uveitis (FHU) is a peculiar uveitis which many have perceived to behave less aggressively after cataract surgery. This view is not universally shared,[2] and a higher inflammation rate has been reported from IOL implantation during extracapsular cataract extraction (ECCE)[5] and in comparison with other forms of uveitis, in phakoemulsification surgery.[2] In the past some FHU eyes did not undergo primary IOL implantation but patients may subsequently request secondary IOL implantation, usually on the grounds of contact lens (CL) intolerance.

The outcomes and problems of secondary IOL implantation have been reported.[6–8] However, there are no previous reports on the procedure in eyes with uveitis.

Case reports

The details of the four cases are shown in Table 1. All cases underwent primary surgery elsewhere and were referred to the Manchester Uveitis Clinic for secondary surgery. In all cases posterior capsule integrity was demonstrated preoperatively. A large-optic (7 mm) IOL was inserted under Healon® via a corneal section, into the ciliary sulcus. Two-hourly topical steroid was administered postoperatively, being tapered to zero according to recovery rate.

Case 1

A 43-year-old male bus driver with right FHU underwent ECCE but developed progressive contact