floating in the midvitreous near the hyaloid system,[15] but is also found in the retrolental space[16] and more posteriorly near the optic disc.[12]

One histological examination of a vitreous cyst in a patient with previous retinal detachment surgery showed gliotically changed retinal tissue embedded in a collagen matrix.[6] The authors theorized that the cyst might have been derived from displacement of retinal tissue into the vitreous cavity secondary to the retinal detachment or the subsequent surgery. They did not rule out that the cyst occurred congenitally from a pathological retinal process.

Another report of a vitreous cyst revealed cells with positive carbonic anhydrase and actin activity.[15] This suggested that these cells are similar to pigment epithelial-type cells of the retina, ciliary body, or iris. These cells also contained melanosomes and premelanosomes. The authors suggest that the presence of premelanosomes would argue against an acquired traumatic cause, as the pigment epithelia are melanized at birth and no further pigment granules are formed after. As these cysts were associated with remnants of the hyaloid system, and sometimes were located in Cloquet's canal, they believe that vitreous cysts represent congenital remnants of the primary hyaloidal system.

Vitreous cysts are generally not symptomatic and can be followed conservatively.[11] Occasionally, patients report symptoms of floaters,[14] visual field defects,[13] or intermittent blurring of vision when the cyst crosses in and out of the visual axis.[13] Argon laser photocoagulation[13] and Nd : YAG laser[14] have been described to rupture these cysts. Two reports had symptomatic cysts removed by pars plana vitrectomies,[6,15] while one patient had his cyst aspirated via a pars plana approach.[8]

There has been no prior association with congenital or juvenile-onset cataract and our case represents the first such occurrence. We also believe our patient is the youngest reported case of a vitreous cyst. This supports the idea that these cysts are congenital in nature. A fibrous structure was also noted anterior to the optic disc in close proximity to the cyst in our patient, and this may represent a persistent hyaloid structure. As vitreous cysts may be a remnant of the hyaloid system, they may represent part of a spectrum with persistent hyperplastic primary vitreous on one end and other hyaloid remnants like Mittendorf's dot and Bergmeister's papilla on the other.

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Sir,

Autosomal dominant retinitis pigmentosa in a five-generation pedigree in People's Republic of China
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The reported prevalence of retinitis pigmentosa (RP) in China is 25 per 100 000,[1] which is similar to the rate of 19–27 per 100 000 observed in Western countries.[2] Genes causing RP have been identified by a combination of linkage mapping, cloning, and candidate testing. At present, close to 100 rhodopsin mutations have been identified in adRP patients and the existence of other

adRP loci has been established.[3] Genetic heterogeneity, allelic heterogeneity, and clinical heterogeneity have been demonstrated among patients with adRP, arRP, and X-linked patterns of inheritance.[4–6] Digenic RP caused by the simultaneous presence of a mutation in the peripherin/RDS gene and a mutation in the Rom1 gene, and one family with RP involving mitochondrial inheritance, have also been described.[7,8]

Case report

RP has been clinically diagnosed in 32 members of a five-generation family of 111 persons, aged 3–82 years, from Hubei Province, People's Republic of China (Figure 1). The proband is a 58-year-old male (III17) diagnosed with night-blindness in early childhood, and showing gradual deterioration of peripheral sight and almost total loss of vision by 52 years of age. Symptoms in the various affected family members include night-blindness, impaired vision, and visual field loss. Their visual deterioration commenced between 20 and 30 years of age, with symptoms usually starting in one eye and progressing bilaterally. Progressive myopia and astigmatism, and retinochoroidal dystrophy are found in all patients. The family members underwent clinical examination, testing for visual acuity (VA) and field loss, fundus examination and fundus photography in Tongji Medical College, Huazhong University of Science and Technology, China. The earliest ocular changes in members of the family were a pale fundus with pigment granularity, followed by narrowed vessels and bone spicule-like pigment accumulation by 10–20 years of age. The pigment deposits increased and approached the posterior pole by 30–50 years of age (Figure 2). Eight patients over 30 years of age have severe retinochoroidal atrophy and a bull's-eye macula is seen in most patients. Teenage patients in the family have peripheral lens opacities, and young adult patients have early nuclear sclerosis confirmed by lens opacitometry. A 23-year-old patient (IV61) with VA of 0.02 also has posterior subcapsular cataracts and retinal detachment in his left eye. Several persons over the age of 30 years old are completely blind (III22, III23, III24, and IV39), and other individuals over the age of 50 years (II8, II9, and III12) are severely visually handicapped (VA=0.00). Besides visual impairment, other abnormalities including dementia (III30), digital abnormalities (II4, III16, and III18), deaf-mutism (IV45), and mental retardation (IV78 and V109) have also been observed in this pedigree.

Primary candidate genes (ie RP1, RP7, RP9, RP11, RP13, Rom1, Rom3, rhodopsin, and peripherin/rds) have been screened by a combination of SSCP and RFLPs techniques in eight affected (III17, III18, III23, III26, IV61,

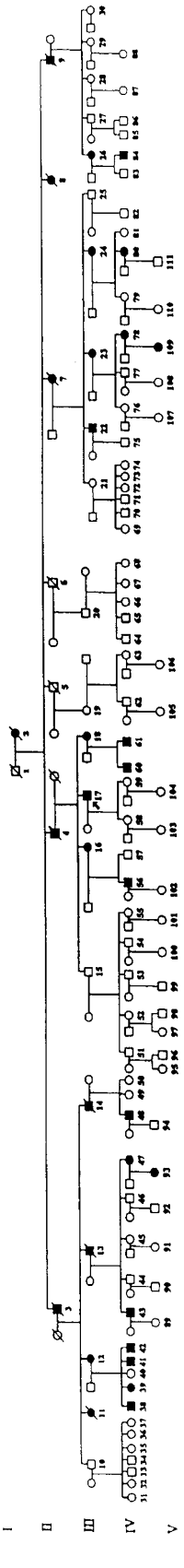

Figure 1 Pedigree of the family with RP patients. Persons who married into the family were not numbered. □, normal male; ○, normal female; ■, affected male; ●, affected female; /, deceased; ↗, proband.

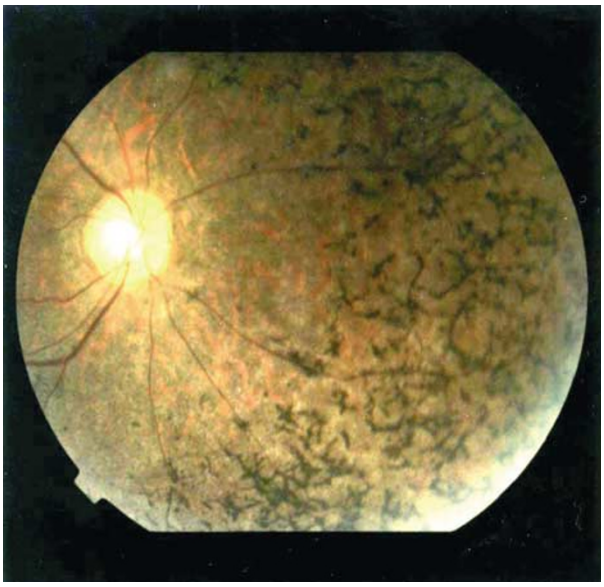

Figure 2 Fundus examination of the proband: pale fundus with pigment granularity, followed by narrowed vessels and bone spicule-like pigment accumulation.

IV78, IV84, and V109) and 10 unaffected family numbers (III19, III20, III21, III28, IV58, IV59, IV62, IV71, IV76, and IV83) available for blood sampling from the pedigree. A previously undescribed missense mutation was identified in the rhodopsin gene at exon 1, codon 52 (TTC→TAC) in all eight affected individuals, resulting in a substitution of phenylalanine (Phe) to tyrosine (Tyr). The mutation was confirmed by direct DNA sequencing.

Comment

Although both Tyr and Phe have similar hydroxyl groups, Tyr is an uncharged polar residue, whereas Phe is nonpolar. The mutation could therefore potentially affect a crucial ligand-binding site or a particular fold. The programme PredictProtein (http://maple.bioc.columbia.edu/pp/) indicated that the mutation did not significantly alter the secondary protein structure. However, it is possible that Tyr interferes with the helical structure encoded by the upstream motif WQFSMLAAYMFLLI.

MaxHom analysis of rhodopsin homologues showed that Phe is conserved across mammalian species,[9] which suggests that it may be critical in ensuring normal visual function. This adds support to the proposition that the Phe→Tyr substitution is the disease mutation. Owing to no restriction enzyme cutting site created or ablated by the sequence change, the initial SSCP method was employed to screen the mutation in 50 unrelated Chinese individuals (25 male and 25 female) from the same Han Chinese ethnic group in Wuhan. The sequence change in the rhodopsin gene was not found in these 100 Chinese control chromosomes, indicating that the mutation is not a polymorphism. Given the level of clinical heterogeneity observed in the family, cytogenetic analysis and mtDNA sequencing will also be undertaken to identify other potential causes associated with additional or alternative candidate regions.

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Sir,

A case of inadvertent ocular perforation and intravitreal injection of depomedrone during peribulbar injection. Good visual prognosis with delayed vitrectomy

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We report a case of inadvertent intraocular injection of depomedrone (methylprednisolone acetate 40 mg/ml). This is a rare complication of peribulbar steroid injection. The need for the procedure to be performed must be strongly assessed in view of the potential complication of perforation.

Case report

A 71-year-old gentleman underwent uneventful left cataract extraction and posterior chamber implant insertion in September 1999. He developed refractory cystoid macular oedema (CMO) 2 months postoperatively, unresponsive to topical/oral steroids and topical nonsteroidal anti-inflammatories. He started to develop osteoporotic joint symptoms while on oral steroids; hence the decision was made to perform a peribulbar steroid injection to reduce systemic steroid sideeffects. A sharp 25-gauge 16-mm needle was introduced transconjunctivally at the junction of the outer third and medial two-thirds of the lower orbital margin. Some resistance to insertion was experienced during needle entry. Aspiration check revealed no blood or fluid. A measure of 1 ml of depomedrone was injected. This was perceived by the patient to be uncomfortable at the time. The eye was then double padded and the patient was asked to keep the pad on till later that day.

The patient presented 24 h later, as an emergency, with reduced vision in the left eye. He reported that he was aware of floaters in the eye soon after the peribulbar injection. Visual acuity prior to the injection was 6/9, and was count fingers 24 h postinjection. Examination findings were a subconjunctival haemorrhage in the left inferior fornix; anterior chamber debris and white globules of depomedrone were noted floating in the vitreous with some coating the retina inferiorly (Figure 1). The IOP was 12 mmHg. He was commenced on oral ciprofloxacin and topical ofloxacin, atropine, and dexamethasone.

An ultrasound B scan was performed, which revealed vitreous detachment with a mobile subhyaloid collection (Figure 2). No retinal elevation was noted.

A pars plana vitrectomy was performed at the earliest available opportunity, which was just over 48 h after the perforation. Laser retinopexy to needle entry site and air/fluid/gas (C_3F_8) exchange was performed. Postoperative posturing was advised.

He underwent an uneventful postoperative recovery and his visual acuity gradually improved to 6/12 and N5 over a 2-month period. FFA performed 2 months postoperatively revealed persisting CMO. Visual acuity

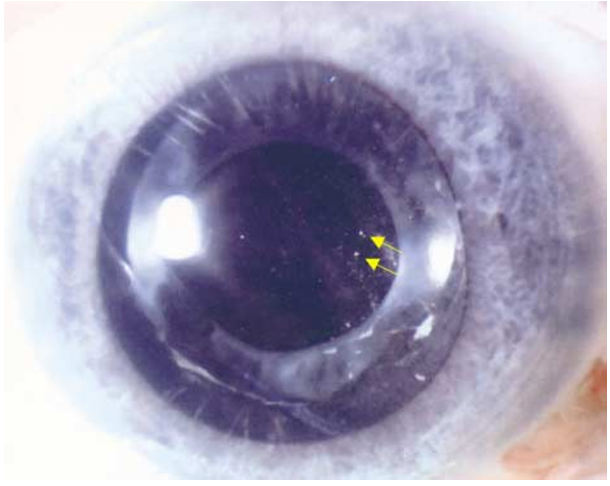

Figure 1 Slit-lamp photograph showing depomedrone globules in the anterior chamber.

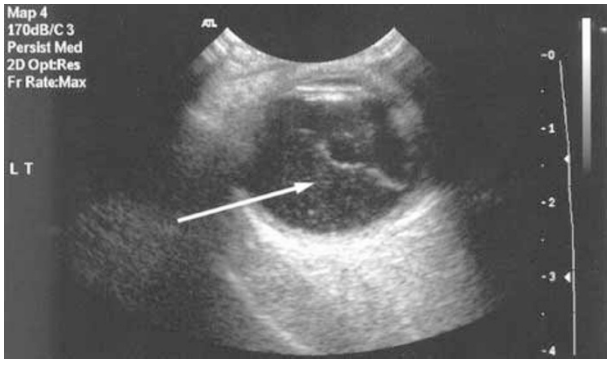

Figure 2 Ultrasound B scan showing subhyaloid collection of depomedrone (arrowhead).