contraceptives, optic disc vasculitis and thrombophilic factors. A number of coagulation disorders increase the risk of systemic venous thrombosis,[3–9] Factor V Leiden being the most frequently identifiable cause.[7] The Factor V gene is located in the short arm of chromosome 1. It acts as a cofactor for activation of prothrombin by factor Xa. Factor V Leiden is a thrombophilic genotype characterised by homozygosity or heterozygosity for a point mutation in the factor V gene, where glutamine is substituted for arginine at position 506. The resultant mutation makes factor V resistant to degradation by APC, thereby increasing thrombotic tendency. Factor V Leiden is responsible for about 95% cases of APC resistance.[6] It is found in 1–7% of Caucasian alleles.[8] It is already known to be a risk factor in systemic thromboembolism including deep venous thrombosis. Although this patient had hypertension as an important risk factor for developing RVO, the recurrence of RVO in spite of adequate blood pressure control prompted us to investigate for other uncommon identifiable causes. This case demonstrates the need of thrombophilic screening in patients with recurrent venous occlusion.
Haematological opinion should be sought in cases of young patients, patients with history of systemic thrombo-embolic disease, family history of thromboembolism, recurrent foetal loss. Timely institution of antithrombotic therapy may help prevent further potentially fatal thrombo-embolic accidents in these cases.

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Sir,

Diagnosis of pre-existing posterior capsule defect in traumatic white mature cataract with intact anterior capsule

In cases of blunt ocular injury, cataract may result from the impact of trauma with or without anterior and/or posterior capsular defect. We report an intralenticular sign to anticipate a pre-existing posterior capsule defect (PPCD) in white mature cataract with an intact anterior capsule following blunt trauma.

Case report

A 17-year-old male presented with the history of trauma to the left eye with a plastic ball 10 days back and progressive dimness of vision since then. He had an accurate projection with normal pupillary reactions. Slit-lamp examination revealed a white mature cataract with intact anterior capsule and a 'sinking cortex' sign. Intraocular pressure was normal. Fundus details were not visible. A-Scan examination was un-confirmatory. B-Scan ultrasonography revealed floating vitreous echoes of moderate density just behind the posterior capsule (Figure 1). Right eye examination revealed no abnormality on detailed slit-lamp examination after dilatation of pupils.

At the time of surgery, on the following day, slit-lamp examination on the operating table also revealed 'sinking cortex' sign (Figure 2a). There was no postural difference in the appearance on slit-lamp examination in sitting

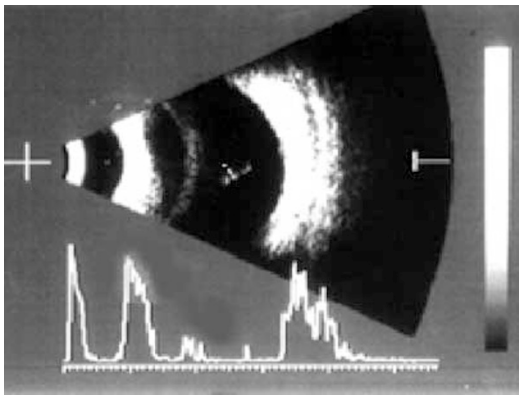

Figure 1 B-Scan photograph showing vitreous floaters.

position as compared to examination on slit lamp mounted on the operating microscope in supine position. Surgery was performed using principles of the close chamber technique under peribulbar anaesthesia as described elsewhere.[1] A single piece AcrySof® (AcrySof®; Alcon laboratories, Fort Worth, TX, USA, model SA60AT) IOL was implanted in the sulcus.

On first postoperative day the anterior segment was quiet with normal IOP of 20 mmHg. On 1-month follow-up UCVA was 20/30, with quiet anterior segment and a centred IOL (Figure 2b).

Comment

Posterior capsule visualization is difficult in white mature cataracts. Traumatic cataracts are often associated with PPCD.[2–6] In white mature cataracts, B-Scan ultrasonography is a useful tool for indirect documentation of PPCD. PPCD is recognized by floating echoes behind the posterior capsule. An accurate preoperative clinical diagnosis of PPCD in white mature cataracts with intact anterior capsule still remains uncertain.

The hallmark of identifying PPCD in white mature cataracts is by 'sinking cortex' sign. As a result of the defect in the posterior capsule, the posterior cortex sinks behind in the vitreous cavity. Moreover, there is always some absorption of the lens matter. All these together will create an empty space in between the intact anterior capsule and the anterior cortex. This empty space appears as a dimple in the anterior cortex of the cataract, which we call 'sinking cortex' sign (Figure 2a). A swift total maturation of the cataract, the 'sinking cortex' sign, and floating echoes in the B-Scan are a sure indication of PPCD.

Contusion cataract may develop due to contra-coup damage following a blow to the orbital area. Shock waves pass through the eye, possibly rupturing the anterior or posterior lens capsule with subsequent lens opacification.

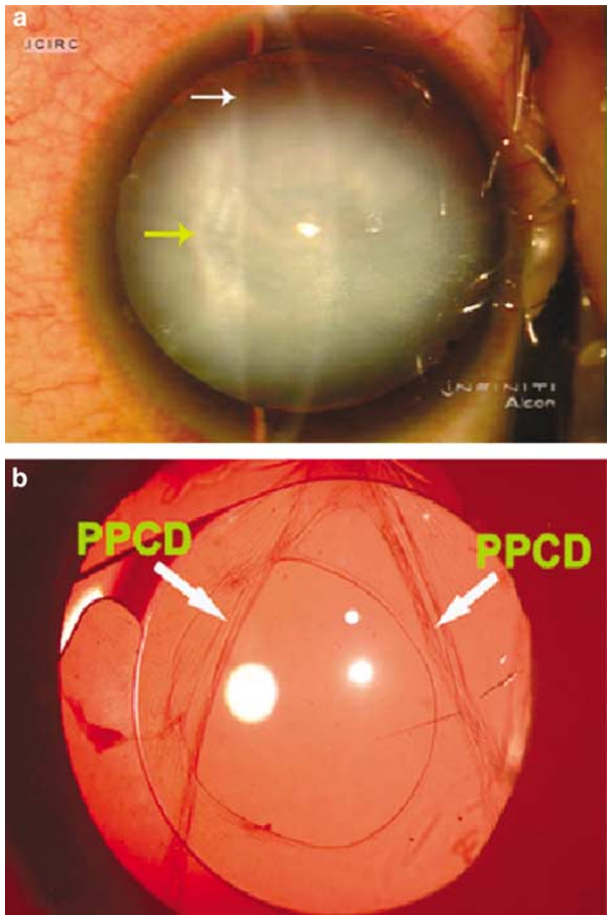

Figure 2 (a) White arrow indicates the normal cortex. Yellow arrow indicates 'sinking cortex' sign, a dimple with a hollow space between the intact anterior capsule and cortex. (b) A postoperative photograph of the same patient with pre-existing posterior capsule defect (PPCD), at 1-month follow-up, showing a small anterior capsulorhexis with AcrySof SA60AT in the ciliary sulcus.

There is an equatorial expansion of the eye that may cause a rupture in the lens capsule.[7] The 'typical' posterior capsule tears in cases of blunt trauma are usually located in the central part of the posterior capsule, the area thinnest and most vulnerable to concussional insult. The typical appearance described in the literature has thickened and fibrosed margins of these posterior capsule tears, which have been attributed to the migration of the hyperplastic epithelial cells that collect in this region.[2] Congenital deformity like posterior lentiglobus was ruled out from the history, detailed slit-lamp evaluation of posterior capsule after dilatation in fellow eye and absence of any amblyopia postoperatively.

An accurate diagnosis helped us in preoperative counselling regarding potential difficulties during surgery. Anticipation of PPCD made us perform a small

anterior capsulorhexis contemplating ciliary sulcus fixation of IOL (Figure 2b) and abandoning hydroprocedures. Use of the principles of close chamber technique[1] prevented vitreous loss and posterior segment complications. We routinely use single piece AcrySof IOL for sulcus fixation. In our experience, symmetry of the placement of this IOL is critical and not the bulk of the haptic. At the root of the iris the bulk and the square edge of the single piece haptic will not produce excessive irritation, as it is not mobile.

In summary, this case emphasizes the importance of 'sinking cortex' sign in predicting PPCD in traumatic white mature cataracts for a suitable surgical strategy to achieve satisfactory technical and visual outcomes.

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Sir,

Safe and effective visualisation of vitreous in the anterior chamber with intracameral fluorescein to facilitate its complete removal

Prolapsed vitreous into the anterior chamber is a commonly encountered surgical problem, either as a result of trauma or in complicated intraocular surgery, such as posterior capsule (PC) rupture during cataract surgery.[1] Indeed, PC rupture is one of the most common complications in cataract surgery especially in the initial stages of training, frequently resulting in herniation of vitreous into the anterior chamber.[2,3] Even though PC rupture is associated with a significant risk of reduced final visual outcome,[4] it has been shown that with prompt and appropriate management of the complications, good visual outcome is possible.[5] Complete clearance of vitreous from the AC is thus essential to prevent further complications such as persistent wound leak with associated pain secondary to vitreous traction on the wound, vitreoretinal traction leading to cystoid macular oedema, retinal tear or retinal detachment, secondary glaucoma, and in some cases corneal endothelial toxicity.[6–9]

The use of fluorescein as a vitreous staining agent was first proposed in the 1980s (Hanemoto, Ophthalmology Times, April 2004), but did not gain much popularity and has never been formally presented or published. It is therefore little known by the younger generation of ophthalmologists. In this study, we revisited this idea and modified the technique for intracameral use.

Materials and methods

Surgical technique

A measure of 1–2 drops ($\sim 60\,\mu$l) of 1% fluorescein minim (Minims, Chauvin Pharmaceuticals) is added to 2 ml of balanced salt solution (BSS®) to give a concentration of 0.03%. This diluted 0.03% fluorescein solution is then injected into the anterior chamber via paracentesis using a 2 ml luer-lock syringe and a 30 G cannula.

The vitreous present in the anterior chamber (Figure 1a) is immediately stained bright yellow-green by the fluorescein and becomes visible (Figure 1b). A standard vitreous cutter (Millennium™) with a separate anterior chamber irrigation port is then used to remove the fluorescein-stained vitreous from the anterior chamber. Given its low toxicity, this fluorescein staining can be repeated several times until the surgeon is satisfied that all the vitreous has been removed.

Intracameral miochol is a useful adjunct to confirm vitreous removal by demonstrating a round pupil without distortion from the vitreous strands, and it was used in some of the patients in our study.