forceps-assisted delivery makes birth trauma the most likely cause although it can only be proven by a histological study.[4] It is also not possible to prove that this eye had a low endothelial cell count preoperatively. However, it is known that corneal birth trauma causes endothelial cell loss[3] and can lead to late endothelial decompensation in adult life.[5] This case highlights the risk of cataract surgery in an eye with a cornea compromised by birth trauma, a risk we failed to recognise preoperatively. We suggest that a patient with signs suggestive of corneal birth trauma should have an endothelial cell count before undergoing any intraocular procedure. A low endothelial cell count will be an objective indicator of the increased risk of cataract surgery in such a patient. Intraoperative measures such as the soft shell technique[6] should then be employed to reduce the risk of postoperative bullous keratopathy.

References

1 Yi DH, Dana MR. Corneal edema after cataract surgery: incidence and etiology. *Semin Ophthal* 2002; **17**(3–4): 110–114.
2 Goodman DF, Stark WJ, Gottsch JD. Complications of cataract extraction with intraocular lens implantation. *Ophthal Surg* 1989; **20**: 132–140.
3 Mcdonald MB, Burgess SK. Contralateral occipital depression related to obstetric forceps injury to the eye. *Am J Ophthal* 1992; **114**: 318–321.
4 Tetsumoto K, Kubota T, Rummelt V, Holbach LM, Naumann GO. Epithelial transformation of the corneal endothelium in forceps birth-injury-associated keratopathy. *Cornea* 1993; **12**(1): 65–71.
5 Duke-Elder SS. *System of Ophthalmology*. CV Mosby Co: St Louis, 1965, pp 707–711.
6 Arshinoff SA. Dispersive-cohesive viscoelastic soft shell technique. *J Cataract Refract Surg* 1999; **25**(2): 167–173.

V Chadha, AH Taguri and HC Devlin

Princess Alexandra Eye Pavilion,
Edinburgh, UK

Correspondence: V Chadha,
11 West Holmes Place,
Broxburn EH52 5NJ, UK
Tel: +44 786 508 1415;
Fax: +44 131 536 1574.
E-mail: vchdh@aol.com

Eye (2006) **20,** 1428–1429. doi:10.1038/sj.eye.6702288;
published online 24 February 2006

Sir,

Screening for visual impairment in elderly patients with hip fracture: validating a simple bedside test

Dr Squirrell's study involves a comparison of the visual screening test conducted by ophthalmologists and nursing staff in a group of elderly with history of fall.[1] They reported a high level of consistency of the bedside test by nurse screeners.[1] We have got queries for authors' enlightenment.

First, the sensitivity of the test in picking up the potentially remedial visual impairment was reported to be only 70%, which was attributed to misinterpretation of the red reflex.[1] From the data cited, there was a significant number of patients who suffered from age-related macular degeneration (10 patients). The status of the age-related macular degeneration has not been elucidated fully and the proportion of early against advanced macular degenerative disease was not clear. Using the proposed bedside test, how the nurse screener could confidently detect the presence of age-related macular degeneration, which may or may not be coexisting with the cataracts?

Second, geriatric study has shown that diabetes mellitus is an independent risk factor for fall among elderly, and the prevalence of diabetes is ever rising worldwide.[2] Therefore, in such a selected group of patients with history of fall, detection of diabetes mellitus from either history, physical examination, or investigation is not unusual. Surprisingly, the authors did not report any diabetic retinopathy or maculopathy affecting the vision. We were keen to learn the initial demographics and past health of the enrolled patients.

References

1 Squirrell DM, Kenny J, Mawer N, Gupta M, West J, Currie ZI *et al*. Screening for visual impairment in elderly patients with hip fracture: validating a simple bedside test. *Eye* 2005; **19**: 55–59.
2 Maurer MS, Burcham J, Cheng H. Diabetes mellitus is associated with an increased risk of falls in elderly residents of a long-term care facility. *J Gerontol A* 2005; **60**: 1157–1162.

DTL Liu, C-L Li and VYW Lee

Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong

Correspondence: DTL Liu,
Tel: +852 2632 2878;
Fax: +852 2648 2943.
E-mail: david_tlliu@yahoo.com

Financial and proprietary interest: Nil
Financial support: Nil

Eye (2006) **20,** 1429–1430. doi:10.1038/sj.eye.6702289; published online 17 February 2006

Sir,

Bilateral choroidal detachments due to massive pulmonary embolism

Choroidal detachment (in the absence of haemorrhage) is caused by the transudation of serum into the suprachoroidal space. This is most commonly due to an increase in orbital transmural pressure, because of trauma, serum exudation, or inflammation.[1] To the best of our knowledge, choroidal detachment is unreported in association with pulmonary embolism (PE). We report this previously serious occular complication of acute massive PE.

Case report

An 86-year-old man presented following a collapse outside his general practioner (GP) surgery. On arrival of the paramedics, he was cyanosed, with an unrecordable blood pressure. He was normotensive on arrival at hospital, where an initial electrocardiogram (ECG) showed acute right bundle branch block (RBBB). An arterial blood gas on high-flow oxygen showed hypoxia. The alveolar–arterial oxygen gradient was very high at 85.3.

A D-dimer was raised, as was the troponin. A diagnosis of acute massive PE was made and he was anticoagulated with low molecular weight heparin and warfarin. He was not thrombolysed. A computed tomographic pulmonary angiogram (CTPA) scan, 12 h later, confirmed large bilateral pulmonary emboli.

He complained of blurred vision 24 h later. On examination, visual acuity was 6/60 in the right eye (RE) and light perception in the left intraoccular pressures (IOP) was normal. Pupils were reactive, but sluggish on the left. Anterior segment examination showed folds in the descements membrane. The anterior choroid was quiet. Dilated fundus examination revealed bilateral choroidal detachments with kissing choroids seen on B scan, with appearances consistent with suprachoroidal haemorrhage (Figure 1).

The patient was managed conservatively. After 10 days later, right visual acuity had improved to 6/18, with counting fingers in the left eye (LE). By 1 month, there was near resolution of the right choroidal detachment, with an RE visual acuity of 6/6 at 2 months. At 6 weeks following the initial insult, he developed a secondary LE vitreous haemorrhage, which failed to improve with conservative management. Anticoagulation was optimal throughout. He underwent a vitrectomy under local anaesthesia, leaving his LE visual acuity at 6/60 with resolved choroidal detachment, a flat retina, and no macular oedema.

Comment

Serous choroidal detachment involves the exudation of serum into the suprachoroidal space. There are several mechanisms for this, including low intraoccular pressure (IOP), which can be a result of ciliary body hypoperfusion.[1] We consider the most likely mechanism for this patient's choroidal detachment to be a low arterial perfusion pressure. Acute massive PE leads to an acute rise in pulmonary artery pressure. This leads to reduced pulmonary blood flow and impaired left heart filling, leading to a low systemic blood pressure.[2] The low cardiac output state leads to reduced carotid perfusion, and therefore reduced flow in the ophthalmic

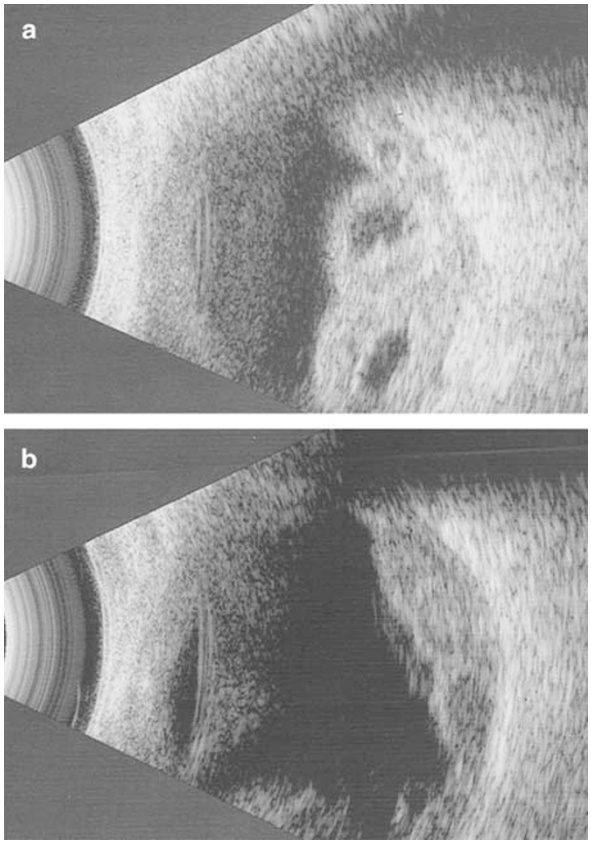

Figure 1 B scan of the RE (top panel, a) and LE (lower panel, b), 4 days post PE, showing choroidal detachments with kissing choroids.