In an observation of 29 eyes in 27 paediatric RRD patients, Fivgas and Capone[2] had shown that myopia was reckoned as the most common aetiology (34%, 10/29 eyes) with nearly half of the myopic patients labelled as having ROP-related detachments. In the present study, authors did not provide any subgroup analysis and it is not certain how many cases were actually in association with ROP, which is not an uncommon retinal disorders in the prematurity paediatric population.

Conversely, familial exudative vitreoretinopathy (FEVR) is a comparatively uncommon paediatric retinal disorder.[3] A proportion of 13% among other causes for paediatric RRD may be disproportionately high in comparison with other series.[2] Retinal detachment associated with FEVR may not necessarily be rhegmatogenous in nature, which has been found to be responsible for only 57.1% of RD cases whereas exudative together with tractional RD accounted for the remaining 42.9%.[3] In lack of further breakdown of the data and ascertainment of the inclusion criteria, we were wondering the possibility of combined RD such as rhegmatogenous and tractional or rhegmatogenous and exudative entities instead of pure RRD being recruited into the study.

Authors can certainly enlighten us in these regards.

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Sir,

Reply to DTL Liu *et al*

We appreciate the comments made by Dr David TL Liu.

The diagnosis of familial exudative vitreoretinopathy (FEVR) in our study followed the precedent report by Akabane *et al*,[1] in which it was based on abnormal straightening of the retinal vessels, no history of retinopathy of prematurity (ROP), peripheral areas of retinal nonperfusion, and either present or absent of family history. FEVR is genetically heterogeneous. It is not always an autosomal dominant disorder and sporadic cases without family history have been described.[2] Many individuals have asymptomatic abnormal vascular hypermeability, demonstrable only by fluorescein angiography. It is likely that the frequency has been underestimated. Based on similar diagnostic criteria, FEVR composed 12–16%[1,3] of cases of juvenile rhegmatogenous retinal detachment in Japanese population, which are comparable to our report.

As we stated in the Patients and methods section, we excluded rhegmatogenous retinal detachments secondary to ROP from our study. We intended to state that not only retinal detachment due to acute ROP but also ROP-related detachments in school-aged children were also excluded. Therefore, no ROP-related detachment was included in the myopic patients in our study.

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Sir,

Bilateral jugular vein thrombosis: a rare cause of papilloedema

Papilloedema is often the presenting feature of serious intracranial pathology. Commoner causes include space occupying lesions and benign intracranial hypertension. Impaired cerebral venous drainage is also known to increase intracranial pressure and result in papilloedema. Jugular vein thrombosis is uncommon but may rarely cause papilloedema. We present the case of such a patient.

Case report

A 77-year-old man was seen in the ophthalmic clinic with a 6-month history of headache and a 1-month history of deteriorating vision especially in the left eye.

Past medical history included hypertension and previous myringotomy and T-tube insertion for a right middle ear effusion.

Visual acuity was 6/36 left and 6/12 right. There was no afferent pupillary defect and the ocular media were clear. Fundoscopy revealed bilateral optic disc swelling. He underwent an urgent magnetic resonance imaging (MRI) scan; however, no significant pathology was evident.

A neurological opinion was sought and the patient underwent a lumbar puncture. The CSF opening pressure was elevated at 28 cm H_2O. Brain imaging did not reveal intracerebral venous thrombosis; therefore formal angiography was carried out. This showed slow flow within the venous sinuses and was discovered to be due to a complete occlusion of the right jugular vein and severe narrowing of the left jugular vein (Figure 1). Computerised tomography (CT) scanning of the chest did not show any compressive intrathoracic lesion.

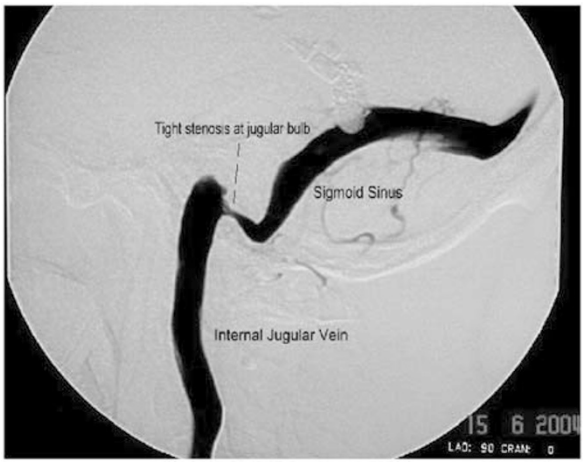

Figure 1 Left jugular venogram showing a narrowing at the jugular bulb.

A full range of blood test were performed including blood count, urea and electrolytes, inflammatory markers, tumour markers, immunological screen, and a thrombophilia screen. These investigations revealed no abnormality apart from a mild anaemia with haemoglobin of 12.9 g/dl and a normal mean corpuscular volume.

The patient was initially heparinised for 6 days to see if that would improve the cerebral venous blood flow, especially if the thromboses were fresh. However, upon repeating the lumbar puncture the cerebro-spinal fluid (CSF) pressure remained elevated at 33 cm H_2O. A neurosurgical opinion was sought and the patient underwent a lumbo-peritoneal shunt procedure. On discharge his visual acuity had improved to 6/9 right and 6/12 left. He has been reviewed in the ophthalmic clinic over the past 6 months and his condition is stable, although he has lost visual field in both eyes due to optic nerve damage secondary to papilloedema. We have still not found an underlying cause for the bilateral jugular vein thrombosis.

Comment

Spontaneous internal jugular vein thrombosis is a rare vascular disorder. In the pre-antibiotic era this condition was a well-known complication of head and neck infection.[1] The leading cause at present is trauma; this can include catheterisation of the internal jugular vein,[2] intravenous drug abuse[3] or head and neck surgery.[4] Other causes include malignancy,[5] thrombophilic[6] states and use of the oral contraceptive pill.[7]

Papilloedema secondary to jugular vein occlusion is rare, and to our knowledge has only been described in two previous cases. One patient developed papilloedema