vasculitides. Upper respiratory tract and eye involvement occur in 75% and 14% of cases respectively, according to one study.³ Ocular manifestations include conjunctivitis, episcleritis, scleritis and uveitis. Corneal ulceration has also been reported. The orbit may be involved by granuloma formation causing pseudotumour and proptosis or vasculitis causing internal and external ophthalmoplegia. Optic nerve may be affected by occlusive vasculitis which may lead to optic nerve ischaemia, compressive granulomatous lesion or direct spread of inflammation from the sinuses. Treatment of WG includes corticosteroids and a cytotoxic agent. Cyclophosphamide is the drug of choice.

Although in this case the neuritis showed excellent therapeutic response to oral prednisolone, it must be emphasised that close liaison with physicians is required as these patients need pulsed cyclophosphamide to halt disease activity.

It is important to mention that although the initial results of immunofluorescence testing supported the diagnosis of Goodpasture's syndrome, it is likely that the patient had WG all along, especially since the ANCA test was not available at the time to exclude the diagnosis of WG. There are cases reported in which patients were initially diagnosed as having Goodpasture's syndrome on the basis of renal failure but later proven to have WG.^{4,5} However, it should be borne in mind that some overlap does exist between WG and Goodpasture's syndrome.⁶

Thus, although it is known that optic nerve involvement may occur with contiguous granulomatous sinus disease in WG,⁷ ophthalmologists must be particularly aware when a patient who has previously had serious renal disease presents with sinusitis and orbital pathology. In this scenario, WG must be excluded.

References

- 1 Bennet C, Plum F, Gill EN. In: *Cecil Textbook of Medicine*, 20th edn. Vol 2. Saunders: Philadelphia, 1996, pp 1495–1498.
- 2 Sternberg ST, Antonioli DA, Carter D *et al.* *Diagnostic Surgical Pathology*, 3rd edn. Vol 1. Lippincott, Williams and Wilkins: Philadelphia, 1999, pp 1040–1043.
- 3 Anderson G, Coles ET, Crane M *et al.* Wegener's Granulomatosis: a series of 265 British cases seen between 1975 and 1985. *Q J Med* 1992; **83**: 427–438.
- 4 Hensley MJ, Feldman NT, Lazarus JM *et al.* Diffuse pulmonary haemorrhage and rapidly progressive renal failure: an uncommon presentation of Wegener's granulomatosis. *Am J Med* 1979; **66**: 894–898.
- 5 Schackter EN, Finkelstein FO, Bastl C *et al.* Diagnostic problems in pulmonary–renal syndromes. *Am Rev Res Dis* 1977; **115**: 155–159.
- 6 Kalluri R, Meyers K *et al.* Goodpasture syndrome involving overlap with Wegener's granulomatosis and anti-glomerular basement membrane disease. *J Am Soc Nephrol* 1997; **8**: 1795–1800.
- 7 Haynes BF, Fishman ML, Fauci AS *et al.* The ocular manifestations of Wegener's granulomatosis: fifteen years experience and review of the literature. *Am J Med* 1977; **63**: 131–141.

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Sir,

Visual field defects in adults secondary to pre-term delivery

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It is well recognised that a prematurely born child can present with problems in adult life.¹ Here we present two cases with repeatable visual field defects noticed on routine visual field assessment at the optician. No immediate cause was apparent, but extensive history-taking revealed premature birth in both cases.

Case reports

Case 1 A 35-year-old woman was noted to have a repeatable visual field defect on routine visual field testing at the optician. She was referred to the hospital by her general practitioner for the same problem. Her visual acuity was 6/6 right and 6/5 left with correction. No significant past ocular or general medical history was present and she did not have any history of familial disease. On examination her pupils were normal. Her ocular movements were full. She was orthophoric. Threshold visual field testing on the Humphrey automated visual field analyser showed repeatable field defects suggesting right homonymous hemianopia. Dilated fundus examination did not show any abnormality. A CT scan showed ill-defined areas of low attenuation close to the inferior horn of the left lateral ventricle, indistinguishable from its margin and

not associated with any mass effect. There was no lesion enhancement on contrast. A subsequent MRI scan showed high signal on the FLAIR, axial and T2-weighted images, extending away from the left lateral ventricle towards the periventricular white matter. It was poorly defined and did not appear to cause any mass effect. There was a minor enlargement of the left lateral ventricle. It was suspected to be focal damage involving the internal capsule, particularly the anterior limb, genu, globus pallidus and caudate nucleus tail. The pattern was consistent with either a haemorrhage or infarction in early life. It was likely that the optic radiation was interrupted close to the lateral geniculate body.

On specific enquiry the patient gave a history of 6 weeks premature delivery. We recovered from her old notes the information that she had an upper limb paresis at birth, which improved spontaneously. Initially it was thought to be brachial plexus injury due to birth trauma, but it is more likely to have resulted from brain parenchymal damage secondary to her prematurity.

She did not have any problem with driving, when she was unaware of the visual field defect. In the light of her recent diagnosis she was advised to inform the Driver and Vehicle Licensing Agency, who revoked her driving licence.

Case 2 A 32-year-old myopic, contact lens wearer was seen at the optician for a routine eye check. There was a history of glaucoma in her family. On a routine visual field examination there was a repeatable right superior quadrantic field defect. She was referred to the eye clinic by her general practitioner.

On examination her best-corrected visual acuity was 6/5 in both eyes. Her pupils were equal and reacting. Colour vision testing was normal. Her intraocular pressures were 16 mmHg in both eyes. Dilated fundus examination was unremarkable. She had no neurological defects. Her right superior quadrantic hemianopia was repeatable when tested on the Humphrey automated visual field analyser.

On specific enquiry we obtained a history of 6 weeks premature delivery. She had neonatal jaundice and had been treated with phototherapy. An MRI scan of the brain and the visual pathways failed to show any abnormality. Estermann field testing showed full driving visual fields that were up to the standard of holding a driving licence.

Comment

Although there are a few reports of neurological abnormalities presenting as a consequence of

prematurity, some of which have ophthalmological relevance eg cortical visual impairment, refractive error, optic atrophy, strabismus,^{2,6} cases of homonymous hemianopic defects attributed to prematurity have not previously been reported. Brain parenchymal damage (periventricular leucomalacia) due to prematurity is well known to have ocular morbidity,^{4,5} but a delayed presentation such as in these cases is unusual. We would like to highlight this unreported ophthalmic consequence of pre-term delivery.

The second point these cases illustrate is a potential problem of screening visual fields: that is the detection of an unexpected, unpleasant and uncorrectable abnormality. The patient described in case 1 had lost her driving licence as a result of an unrequested field test. She subsequently lost her ability to earn her living—a further consequence of prematurity.

References

- 1 Tasman W, Brown GC. Progressive visual loss in adults with retinopathy of prematurity. *Graefes Arch Clin Exp Ophthalmol* 1989; **27**: 309–311.
- 2 Tuppurainen K, Herrgard E, Martikainen A, Mantyjarvi M. Ocular findings in prematurely born children at 5 years of age. *Graefes Arch Clin Exp Ophthalmol* 1993; **31**: 261–266.
- 3 Peterson JR, Rosenberg T, Ibsen KK. Optic nerve atrophy with particular attention to perinatal damage. *Ugeskr Laeger* 1990; **152**: 3865–3867.
- 4 Phillips J, Christiansen SP, Ware G, Landers S, Kirby RS. Ocular morbidity in very low birth-weight infants with intraventricular haemorrhage. *Am J Ophthalmol* 1997; **123**: 218–223.
- 5 King KM, Cronin CM. Ocular findings in premature infants with grade IV intraventricular haemorrhage. *J Paediatr Ophthalmol Strabismus* 1993; **30**: 84–87.
- 6 Dowdeswell HJ, Slater AM, Broomhall J, Tripp J. Visual deficits in children born at less than 32 weeks' gestation with or without major ocular pathology and cerebral damage. *Br J Ophthalmol* 1995; **79**: 447–452.

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