Acute posterior multifocal placoid pigment epitheliopathy associated with pulmonary tuberculosis

EDITOR,—The hallmark of acute posterior multifocal placoid pigment epitheliopathy (APMPE) is the appearance of multiple discrete, yellow, retinal lesions with ill-defined margins concentrated in the posterior pole and deep within the retina. The condition has recently been reviewed1 and has been associated with a wide range of disorders. These include adenovirus type 5 infection,2 vaccination for swine flu,3 cerebral vasculitis,4 Lyme disease,5 sarcoidosis,6 photic damage to the retina,7 and lead toxicity.8 We report a patient presenting with a clinical picture of APMPE in whom the asymptomatic lung infection by tuberculosis was also identified.

CASE REPORT
A 34-year-old woman reported to an ophthalmic casualty department with a history of progressive blurring of vision of the left eye over the previous 24 hours. Systemic inquiry was otherwise negative. There were no respiratory symptoms. She was married with an 8-month-old child, and had never smoked cigarettes. General physical examination was normal. Visual acuities were 6/60 left eye and 6/5 right eye. Multifocal lesions of pale yellow appearance with indistinct margins were found on retinal examination of the left eye (Fig 1). These lesions were positioned within the vascular arcades with other smaller peripheral lesions. Fluorescein angiography showed early masking with late fluorescein.

The chest radiograph exhibited a round 2 cm lesion in the right middle third of the lung field. Subsequent computerised tomography showed the lesion to be in the right lower lobe. It was clearly defined with no evident calcification. The mediastinum was normal.

Figure 1 Left fundus appearance on presentation showing multifocal pale yellow lesions with indistinct margins.

A tuberculin test (1/1000) produced 25 mm induration. There was no scar on any of the usual vaccination sites to suggest previous BCG. Haemoglobin was 14.8 g/dl, white cell count 6.2 x 10^9/l with normal differential count, erythrocyte sedimentation rate was 18 mm in the first hour. Serum electrolytes, creatinine, calcium, phosphate, glucose, liver function tests, albumin, and urate were normal as were serum angiotensin, anti-nuclear factor, rheumatoid factor, and syphilis serology.

Pulmonary tuberculosis was suspected and treated with isoniazid, rifampicin, and pyrazinamide. One month later she developed a productive cough. Numerous acid and alcohol fast bacilli were seen on direct microscopy of the sputum but these did not grow on culture medium during 16 weeks of incubation. The non-viability of these organisms presumably resulted from antituberculous treatment. Her lung shadow subsequently cleared in a manner typical of early tuberculous lesions which fully resolves with appropriate therapy.

From the respiratory aspect, her health remains excellent and she successfully completed her planned 6 month course of antituberculous therapy. Progressive pigmentation of the fundal lesions took place (Fig 2) and in this case there has been no significant improvement in vision.

COMMENT
We are not aware of any previous case published in which the clinical appearance of APMPE has been associated with other wise asymptomatic infection by tuberculosis.

The alternative diagnostic label of multifocal choroiditis could, of course, be applied but in the case described it was a clinical appearance indistinguishable from APMPE which led to the systemic diagnosis.

The clinical features on presentation of our patient accord with those typical of APMPE. Our patient was young. Her visual symptoms were of rapid onset and she developed ‘multiple postequatorial, circumscripted, flat, grey-white, subretinal lesions involving the RPE’9 on examination by biomicroscopy and fluorescein angiography. The late features of the disorder in our patient accord with those described for APMPE8 but also resemble those of other white spot syndromes10 in which the spots are of larger size and tend to coalesce.

It is of interest that in the original description of APMPE by Gass,11 of the four cases described, two had a positive tuberculin skin test and one a family history of TB although none had overt tuberculosis. This patient’s case (which has now been formally notified on the TB register) highlights that patients with the clinical appearance of APMPE warrant careful investigation for underlying tuberculosis.

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References

Disappearance of opticiliary shunt vessels after optic nerve sheath decompression

EDITOR.—Opticiliary shunt vessels were described by Salzmann in 1879. They have been reported in association with many ophthalmic conditions including central retinal vein occlusion, optic nerve sheath meningo ma, and chronic papilloedema. The common mechanism appears to be obstruction to blood outflow in the central retinal vein which results in shunts between retinal and choroidal veins, usually at the edge of the optic disc. We report a patient with optic hydrocephalus causing chronic papilloedema associated with opticiliary shunts which resolved after optic nerve
sheath decompression on each side. This suggests that optociliary shunts are dynamic structures which can appear and disappear with changes in central retinal venous pressure.

CASE REPORT
A 21-year-old man presented in 1981 with a history of right earache with discharge, headache, and obstructions of vision in both eyes. Examination showed bilateral papilloedema but no other neurological abnormality. Cerebrospinal fluid (CSF) had normal constituents but pressure was raised at greater than 400 mm H₂O. Computed tomography scan showed petrous osteomyelitis and small cerebral ventricles. He was treated by right mastoidectomy and during this procedure the lateral dural venous sinus was visibly thrombosed. The headache and obstructions did not resolve so he underwent lumbarperitoneal CSF shunting in 1982. He required numerous shunt revisions to maintain CSF pressure control over the next 4 years. The shunt was removed at the time of a lumbar 4/5 laminectomy in 1986. Headache and visual obscurations recurred and the shunt was reinserted. However, during the following year, the shunt became infected and was removed again. His headache worsened and visual obscurations were occurring 15 times a day in each eye.

In April 1988 he underwent right optic nerve sheath decompression via a medial orbital approach under general anaesthesia. The procedure was without complication and the frequency of obstructions immediately reduced to roughly twice per week. One week after surgery the optociliary shunt that had been evident on the right disc before surgery was much reduced in calibre (Fig 1).

The left eye continued to suffer frequent obstructions and by later in the year, these were occurring 20–30 times a day. In January 1989 the left optic nerve was decompressed using the same technique. Again surgery went smoothly and by 4 days postoperatively the optociliary shunt vessel on this disc was much reduced in diameter (Fig 2). Both optociliary shunts continued to undergo further involution with time and, late in 1992, there was no swelling of either disc. Both discs were flat and pale with no conspicuous optociliary shunts.

At last follow up in early 1993, vision was preserved at 6/5 N5 in both eyes and, although visual fields were smaller than a normal individual of his age, his vision was functionally normal. Repeated visual field assessment showed residual enlargement of the blind spots although the optic discs were clinically not swollen.

COMMENT
Optociliary shunts may be congenital or acquired.1,2 Acquired optociliary shunts are classically associated with optic nerve sheath meningioma3 although central retinal vein occlusion is the most common cause in clinical practice. Dowhan et al1 give a list of causes including glaucomatous optic atrophy, chronic papilloedema, optic nerve glioma, arachnoid cyst of optic nerve, neurofibromatosis, benign intracranial hypertension (pseudotumour cerebri), optic disc drusen, optic nerve coloboma, and osteosclerosis.

Optociliary vessels develop when dilatation of collateral channels connecting retinal and choroidal venous systems occurs as central retinal venous pressure (CRVP) rises.2,4 The increase in CRVP may follow obstruction in the vessel lumen (central retinal vein occlusion), nerve itself (glioma), subarachnoid space (raised CSF pressure), or by disease of the nerve sheath (meningioma).

Previous reports of resolution of optociliary shunts are scarce and we have only been able to locate one case in which resolution followed optic nerve sheath decompression. Perlmutter et al5 described a patient with benign intracranial hypertension (pseudotumour cerebri) where optociliary shunt vessel calibre was reduced after bilateral optic nerve sheath decompression. Other reports of resolving CSF or intracranial pressure have rarely been reported to cause resolution of optociliary shunts. Dowhan et al1 reported two patients with neonatal hydrocephalus where optociliary shunts disappeared following CSF shunting procedures. Tyson and Lessell6 described optociliary shunt vessels associated with chronic atrophic papilloedema which resolved when a massive meningioma was removed. The case reported here and the other quoted demonstrate that optociliary shunts due to raised CSFP with chronic papilloedema are reversible when CSFP is reduced. The reduced CSFP reduces CRVP and pressure in venous drainage by reducing the calibre of the central retinal vein, reducing the calibre of the blood column in the optociliary shunt vessels. This regulation of calibre appears to occur rapidly with normalisation of CSFP and study of optociliary shunt vessels provide longitudinal information regarding CSFP in the optic nerve sheath in patients with chronic papilloedema.

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Paroxysmal superior rectus and levator palpebrae spasm: a unique presentation of multiple sclerosis

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