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Acute posterior multifocal placoid pigment epitheliopathy associated with pulmonary tuberculosis

EDITOR.—The hallmark of acute posterior multifocal placoid pigment epitheliopathy (APMPPE) is the appearance of multiple discrete cream-coloured 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 APMPPE in whom 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 fluorescence.

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.

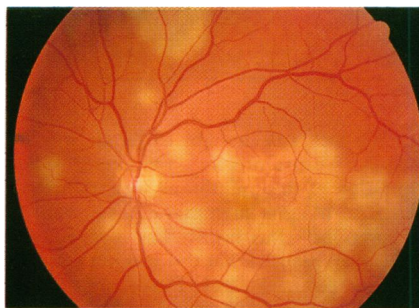


Figure 1 Left fundus appearance on presentation showing multifocal pale yellow lesions with indistinct margins.

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 $6.2 \times 10^9/l$ 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, antinuclear 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 tuberculosis 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 APMPPE has been associated with otherwise 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 APMPPE which led to the systemic diagnosis.

The clinical features on presentation of our patient accord with those typical of APMPPE.⁹ Our patient was young. Her



Figure 2 Left fundus appearance following resolution of acute lesions. Extensive areas of retinal pigment epithelial atrophy and pigment clumping are seen.

visual symptoms were of rapid onset and she developed 'multiple postequatorial, circumscribed, 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 APMPPE⁹ 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 APMPPE 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 APMPPE warrant careful investigation for underlying tuberculosis.

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Disappearance of opticiliary shunt vessels after optic nerve sheath decompression

EDITOR.—Optociliary shunt vessels were described by Salzman in 1893 and have been reported in association with many ophthalmic conditions including central retinal vein occlusion, optic nerve sheath meningioma, 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 otitic hydrocephalus causing chronic papilloedema associated with opticiliary shunts which resolved after optic nerve



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