Optic neuritis following measles/rubella vaccination in two 13-year-old children

Editor.—During November 1994 following the Joint Committee on Vaccination and Immunisations recommendations a widespread school based immunisation campaign was set up for measles and rubella. The objectives were to negate the need for a single antigen rubella vaccine to be given to 11-year-old girls and to promote herd immunity to measles.1

We describe two 13-year-old children who developed optic neuritis 2–3 weeks after measles/rubella vaccination.

Case 1

A 13-year-old boy of Asian origin was seen on the 23 December 1994 with a 2–3 week history of deteriorating vision. Initially this began in the left eye but was bilateral within a few days and was associated with photophobia and pain on eye movements. Three weeks before the symptoms appeared he had received a measles/rubella vaccination at school. There was no family history of Leber's optic neuropathy, multiple sclerosis, or other neuromuscular disease.

General and neurological examination excluding the ocular findings was normal. Visual acuities were corrected to right eye 1/60, N14, and left eye 6/36, N8. Ishihara plates for colour vision were right eye 1/17 and left eye 4/17. Visual fields demonstrated bilateral central scotomas. Funduscoppy revealed mild bilateral optic disc hyperaemia with a single flame-shaped peripapillary haemorrhage on the left. The peripheral retina, macula, vitreous anterior segments, and extracocular movements were normal for each eye.

Investigations revealed normal full blood count, biochemistry profile, clotting studies, erythrocyte sedimentation rate, viral B12, and serum anticonversion enzyme. Autoantibodies, the VDRL test, and heavy metal screen were negative. Magnetic resonance imaging (MRI) (axial T2, coronal T1, and T1 contrast) showed mild signal change in the posterior part of the optic nerves and chiasm; this was felt to be consistent with inflammation or demyelination. There were no white matter lesions elsewhere in the brain. Fundus autofluorescence angiography demonstrated mild tortuosity of the retinal vessels but there was no disc leakage or evidence of subclinical periphlebitis. CSF examination revealed normal constituents; oligoclonal bands were negative. Tests for the pathological mutations of mitochondrial DNA associated with Leber's hereditary optic neuropathy (LHON) at the 11778, 3460, and 14484 sites were negative.

The patient was treated with vitamin B12 injections and intravenous methylprednisolone but made no recovery. On review in September 1995 visual acuities were 6/60 and N4/60 for the left eye with bilateral central scotomas and pale discs. Repeat MRI (without contrast) of the brain and orbits was normal.
Letters

LETTERS

Retinal arterial occlusion associated with resistance to activated protein C

EDITOR,—Resistance to activated protein C (APC) was described in 1994 as a thrombophilic factor responsible for deep venous thrombosis and pulmonary embolism.1 It represents one component of thrombophilic disorder in the normal population (5%). APC is an important component of the physiological anticoagulant system that inhibits factors Va and VIIIa. The resistance to APC is related to a single mutation in the factor V gene which causes the switch from arginine to glutamine at position 506. This mutation of factor V blocks the site of cleavage by APC.2 Reported thrombophilic manifestations include venous thromboembolism and some cases of central retinal vein thrombosis.3 Arterial occlusion is not clearly associated with resistance to APC. Ischaemic stroke was recently reported in three patients with resistance to APC.2 We described a case of branch retinal artery occlusion associated with resistance to APC.

CASE REPORT
A 35-year-old non-smoking man was referred for a sudden decrease of visual acuity in the left eye. He had no familial or personal history of thrombophilic disorders. At examination, his best corrected visual acuity was 20/20 in the right eye and 20/30 in the left. A superotemporal branched retinal artery occlusion was noted in the left eye with an ocular tumour, whitish, retinal infarction in the affected vessel. A fluorescein angiogram confirmed the branched retinal artery occlusion (Fig. 1). After 2 months, visual acuity improved to 20/25 in the left eye. He had a permanent visual field defect in the area of damaged retina. In addition, 2% lignocaine was used by the patient for pain management. Cardiac rhythm was regular; transthoracic echocardiography, and carotid Doppler studies showed no abnormalities. Red blood cell, white cell, platelet counts, and erythrocyte sedimentation rate were all normal. Platelet aggregation was normal. A search for antinuclear and antiprotein antibodies was negative. Prothrombin time and activated partial thromboplastin time (APTT) were normal. Plasma levels of protein C, S, antithrombin III, fibrinogen, plasminogen, plasminogen activator inhibitor were within normal ranges. APC resistance was determined by evaluating the anticoagulant response of plasma samples to APC with an APTT based assay. Results were expressed as the following APC sensitivity ratio (APTT + APC)/(APTT – APC). The cut off value was 2.2. In our patient, the APC sensitivity ratio was 2.1. Heterozygous factor V Leiden mutation was disclosed by molecular analysis.

COMMENT
Occlusion of the retinal artery is more rarely encountered in younger than in older patients.4 Multiple causes of arterial occlusion in the retina were described. In a recent report, the causes of retinal arterial occlusions in 21 young adults were analysed.5 Emboli were identified in 33% of the patients. Cardiac valvular disease, including atrial myxoma, bacterial endocarditis, and mitral valve vegetation due to lupus anticoagulant, was the mainly recognised condition and was present in 19% of the patients. Other associated risk factors for cerebrovascular occlusion such as cigarette smoking, oral contraceptive use, obesity, pregnancy, and Behçet’s disease were found in 91% of the patients. Antithrombin III, protein S, or protein C deficiencies are hypercoagula-
ble conditions that can lead to recurrent venous or arterial thrombotic events.6 Protein S deficiency was associated with a case of bilateral branch retinal artery occlusion.7 This biological abnormality was detected in only one young woman with diabetes mellitus and pregnancy in the series of Greven et al.8 Antiphospholipid antibody syndromes are thrombophilic factors that occur in patients with either lupus anticoagulants or antibodies to antiphospholipid or dissociated syndys tophils serology. Antiphospholid antibodies can lead to recurrent arterial and/or venous thrombosis.9 APC resistance is clearly related to venous thromboembolism.10 A recent report suggests the possible role of APC resistance in arterial thrombosis.10 APC resistance was not searched for as a thrombophilic factor in retinal arterial occlusions in young adults. In our case, the cause of retinal arterial occlusion could be attributed to the heterozygous mutation of factor V. Owing to the severity of retinal arterial occlusion, long term oral anticoagulant treatment was proposed in our patient for secondary prevention of thrombosis. APC resistance should be considered in patients with retinal arterial occlusion when the usual embolic or thrombotic diseases are ruled out.

Figure 1 Superotemporal branched retinal artery occlusion.

Recurrent septic retinal embolus following dental surgery

EDITOR,—Metastatic bacterial endocarditisms following head and neck surgery is rare.1 To our knowledge, no case of recurrent septic retinal embolism with a presumed dental source has been described before.

CASE REPORT
A healthy 36-year-old white man presented to a dental surgeon with a localised periapical abscess at his right upper first molar, which was confirmed by dental x ray. A volume of 0.2 ml of pus was drained after local periapical infiltration with 2% lignocaine 1 ml with adrenaline 1:80 000 and postoperative irrigation with 0.1% dexamethasone solution. He was prescribed oral amoxicillin/clavulanic acid 375 mg three times daily but did not start antibiotics until 12 hours after the procedure. Three days later, the patient noted sudden blurring of vision and floaters in his right eye but was systemically well.

There was no previous ocular history and the medical history was negative for intravenous drug use, rheumatic fever, or other cardiac disease. He had no risk factors to suggest systemic immunosuppression. On examination, a white, yellow, soft, irregular abscess, significantly, he was afebrile with no cardiac signs or peripheral stigmata of infective endo-

carditis. Uncorrected visual acuity was 6/5 in both eyes and there was no relative afferent