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Tobacco-alcohol amblyopia: magnetic resonance imaging findings

Sir: Tobacco-alcohol amblyopia (TAA) is a condition occurring in smokers which causes progressive bilateral visual deterioration. It is commonest in middle-aged and elderly men and malnutrition and excessive alcohol consumption tend to increase the incidence and severity of the condition.¹ The characteristic visual deficit is a centro-caecal scotoma, with loss of colour vision being an early feature.¹

It has been suggested that TAA may be a consequence of the toxic effect of the cyanide constituent of tobacco smoke.¹ Cessation of smoking and hydroxocobalamin replacement usually result in visual improvement.² Pathological studies in TAA have failed to establish whether the primary location of the defect is retinal or in the optic nerve itself.³

Recent advances in magnetic resonance imaging (MRI) of the optic nerve have shown great sensitivity in the detection of optic nerve lesions. For example, in optic neuritis, lesions are found in 84% of clinically affected nerves.⁴ We recently reported abnormal MRI signal in 12 of 15 affected optic nerves in Leber's optic neuropathy (LON),⁵ a maternally inherited condition characterised by the acute or sub-acute onset of bilateral visual loss.⁶ To search for evidence of optic nerve involvement in TAA we have performed optic nerve and brain MRI.

Five patients with TAA were studied. All had typical bilateral centro-caecal scotoma, high tobacco and alcohol consumption,

negative findings for other causes of visual loss and improvement in visual acuities after reduction in smoking. In two patients some improvement in vision had already occurred by the time of MRI (table). Scanning was performed on a Picker 0.5T Superconducting MRI, with axial SE_{2000/60} brain images (5 mm contiguous slices), and coronal STIR (IR_{-1500/40/150}, 5 mm contiguous slices) optic nerve images, as in the Leber's study.⁵

Scans were examined by an experienced neuro-radiologist (BEK). All optic nerve images were normal, in contrast to the findings in LON where most optic nerve images showed either definite or equivocal abnormalities.⁵ One patient (Case 4) had small multiple high signal areas in the white matter, a non-specific finding in this age group.⁷

The results suggest that the optic nerve is not damaged in TAA, or that such damage that occurs is unlikely to produce abnormal MRI signal. However, given the sensitivity of MRI in other conditions, our findings do not provide evidence to support the suggestion that the primary insult in TAA is the optic nerve itself, and are consistent with a proposal from electrophysiological studies that retinal damage makes a significant contribution to the visual loss in TAA.⁸ From the point of view of differential diagnosis we are able to conclude that MRI in TAA is usually normal and therefore may be useful to exclude other causes of visual loss when indicated.

It is interesting to compare these results with those mentioned above in LON.⁵ Recently a specific point mutation in mitochondrial DNA has been proposed as the cause of LON.⁹ This would result in an energy deficit similar to that caused by chronic poisoning with respiratory inhibitors, such as the cyanides⁹ which are found in tobacco smoke.

There are significant clinical differences between TAA and LON. The results reported here provide further evidence against a common aetiology for the two disorders.

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Opsoclonus-myooclonus following the intranasal usage of cocaine

Sir: A wide variety of neurological complications have been reported with cocaine usage. Opsoclonus-myooclonus is an uncommon syndrome thought to be of cerebellar-brain-

Table Visual acuities (VA) and colour vision on Ishihara plates refer to the worst affected eye.

At presentation	Age	VA	Correct plates Ishihara	VA At scan	Duration of symptoms At scan	Final VA
1 M	57	1/60	0/14	6/36	7 months	6/36
2 M	50	6/60	3/12	6/60	5 months	6/6
3 F	42	6/12	6/14	6/5	2 months	6/5
4 M	54	6/36	0/14	6/36	9 months	6/6
5 M	39	6/24	9/14	6/12	10 months	6/12