Sir,
Authors' reply: 'Optical coherence tomography in a patient with tobacco-alcohol amblyopia'

We thank Dr Grzybowski for his interest in our article,¹ and his comment that the term, 'tobacco-alcohol amblyopia' is inappropriate, because 'it is in fact just 'nutritional optic neuropathy' related to deficiencies of vitamin B and foliate acid'. In addition, Dr Grzybowski suggested that Leber's hereditary optic neuropathy (LHON) should be considered.

We completely agree that the use of amblyopia is inaccurate, and also that there is a nutritional component to tobacco-alcohol amblyopia.² Nevertheless, many studies have confirmed the toxic effects of tobacco smoke and ethanol, and the pathogenesis of tobacco-alcohol amblyopia has yet to be elucidated.

Tobacco smoke contains a range of toxins, such as carbon monoxide and cyanide, that are capable of damaging the optic nerve and retina.³ Cyanide in cigarette smoke is not properly metabolized in patients with tobacco-alcohol amblyopia because of alcohol-related hepatic dysfunction and a lack of vitamin B₁₂.⁴ Moreover, cyanide blood levels are elevated,⁵ and smoking is known to impair vitamin B₁₂ absorption in tobacco-alcohol amblyopia patients.⁶

Alcohol consumption can cause electroretinographic abnormalities,^{7,8} and prolonged exposure to ethanol during fetal development is known to have adverse effects on the retina that may persist into adulthood.⁹ Moreover, by-products generated during the metabolism of ethanol affect metabolic processes in neural tissues.¹⁰ Several underlying mechanisms, associated with the toxic effects of tobacco smoke and alcohol or related to nutritional deficiency, may determine the development of tobacco-alcohol amblyopia.¹¹ Thus, in our opinion, tobacco-alcohol amblyopia cannot be straightforwardly classified as a nutritional optic neuropathy.

Regarding the possibility of LHON, four mutations at nucleotides 11778, 14484, 3460, and 4171¹² previously found in Koreans were not found in our patient. Moreover, although there is a possibility that some other LHON mutation may have been present, we emphasize that the clinical features of our patient were incompatible with a diagnosis of LHON. We would have preferred to have added details of our mitochondrial DNA mutation analysis results, but unfortunately, could not do so owing to the word count restriction.

Regarding blood levels of B₁₂ and folic acid, when the patient was referred, he was in good nutritional condition, and therefore these levels were not determined. However, as we described in the article, our patient admitted not eating when he had been drinking 4–5 bottles of Korean gin per day for 10–14 days, once or twice a year for the past 4 years. We appreciate Dr Grzybowski for having given us another opportunity to clear these issues.

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Sir,

Tobacco amblyopia: does it really exist?

With reference to Kee and Hwang's article entitled 'Optical coherence tomography in a patient with tobacco-amblyopia', I doubt whether the presented case of optic neuropathy could be appropriately named 'tobacco amblyopia'.

In the last two centuries it has been believed that tobacco consumption might result in toxic neuropathy called 'tobacco amblyopia'.¹ Tobacco neuropathy has been supposed to originate from excessive cigar and pipe smoking (or tobacco chewing or snuffing) rather than from cigarette smoking.¹ Presently, this disorder is less frequent. Some even argue that 'tobacco amblyopia' does not exist. Nevertheless, some data collected during the

twentieth century should be considered with regard to this problem.

Firstly, paradoxically, in spite of the explosion of cigarette smoking during the twentieth century, the number of tobacco amblyopia cases significantly decreased. The discussion related to this phenomenon can be found elsewhere.²

Secondly, it was shown that optic neuropathy in excessive alcohol consumers—called ‘alcohol amblyopia’ previously or often ‘tobacco-alcohol amblyopia’—is in fact just ‘nutritional optic neuropathy’ related to deficiencies of vitamin B and folic acid.³ In different studies, it was shown that a supplementation diet improves the prognosis even without tobacco abstinence.⁴

Thirdly, it was proposed and documented that some cases of ‘tobacco amblyopia’ were in fact Leber’s hereditary optic neuropathies (LHON) and, possibly, which is less understood, epidemic optic neuropathies in Tanzania, Cuba, or Nigeria.⁵

The patient presented in the article had probably some major nutritional deficiencies, although there was no description of tests for vitamin B12 or folic acid in serum, etc, which could confirm this diagnosis. The LHON and other epidemic causes of optic neuropathy are probably very unlikely in this case. Thus, in my opinion, the patient should be diagnosed as having nutritional optic neuropathy.

I believe that the term ‘tobacco optic neuropathy’ should be reserved only for such cases, where the other—that is nutritional, genetic, as well as toxic or infectious—causes can be excluded. There is also one more argument against using the name ‘tobacco amblyopia’, neither is it caused by tobacco itself (it might be an additional or accompanying factor), nor is it real amblyopia.

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Sir,

An unusual ocular manifestation in fanconi anaemia: anterior ischaemic syndrome

Fanconi’s anaemia (FA) is a rare autosomal recessive disorder.^{1,2} We report this case, hitherto unreported, to emphasize that anterior segment ischaemia may develop as a complication of uncontrolled anaemia.

Case report

A 23-year-old male, a known case of FA, presented with decreasing left eye vision of 15 days duration. On examination, the best-corrected visual acuity (BCVA) was 6/6 and light perception in the right and left eye respectively. Intraocular pressure was 20 and 6 mmHg in the right and the left eye respectively. Right eye examination was unremarkable. Left eye showed sub-conjunctival haemorrhage, diffuse corneal oedema with Descemet’s folds, pigmented keratic precipitates, distorted pupil, and a complicated cataract (Figure 1). The fundus could not be visualized. Ultrasound B-Scan of the left eye revealed an echo-free vitreous cavity with attached retina. Integrity of the visual pathway was confirmed with ERG and VEP.

Haematological evaluation revealed haemoglobin of 7.2 g%, haematocrit 13.30%, total leukocyte count 2100 and low platelet count. Blasts were absent. Systemic vasculitic disorders were ruled out. Doppler scan of the carotids was normal.

The patient received alternate day blood transfusions and oral steroid therapy, for a period of 10 days, monitored by a haematologist. Topically prednisolone acetate 1% eye drops 6 times/day, in weekly tapering doses, and atropine sulphate 1% eye drops 3 times/day was administered. On follow-up, 1 month later, BCVA was 20/600, intraocular pressure was 9 mmHg, corneal oedema had reduced, and iris showed generalized atrophy. There was no evidence of iris neovascularization. The patient was subsequently lost to follow-up.

Figure 1 Clinical photograph of the left eye showing dispersed sub-conjunctival haemorrhage, diffuse corneal oedema with Descemet’s folds, pigmented keratic precipitates, distorted pupil, and a complicated cataract.