model of ocular inflammation. In the case described, the patient was commenced on topical timolol in order to treat the secondary glaucoma; however, this had no effect on the inflammatory process. It is possible that that oral metoprolol was effective because it had a higher bioavailability within the posterior segment, as well as anteriorly. It was thus effective in suppressing ocular inflammation through the mechanisms described above.

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Sir,

Metoprolol responding uveitis: reply

We thank Dr Masood for his useful comments. The mechanism he suggested is tempting, however, it is speculative. It should be emphasized that at the time the patient received metoprolol tartrate he was no longer on timolol. Both $beta_1$- and $beta_2$-adrenergic receptors have been identified in the human iris and ciliary body.[1] Activation of $beta_2$ receptors increases the formation of cyclic adenosine monophosphate and stimulation of Na^+, K^+, Cl^- cotransport in the foetal nonpigmented ciliary epithelium.[2] Metoprolol may as well have an indirect effect on the Na–K pump via adrenergic receptors. This may either result in changes in aqueous production or in concentration of inflammatory mediators in the anterior segment and explains its clinical effect in the specific individual with type A personality. It should be clinically determined if metoprolol has a similar effect on different individuals and in which dosage. The drug may have different activities in different concentrations. The molecular mechanism of metoprolol effect should be evaluated also in cell cultures. Our study is intended to provoke research in these directions.

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Sir,

Reversible night blindness in a patient with neuroendocrine tumour of pancreas

Nutritional vitamin A deficiency is a common cause of blindness in developing countries. Around 2.8 million preschool children are affected with vitamin A deficiency in over 60 countries and subclinical vitamin A deficiency is considered a problem for at least 251 million people.[1] However, in Western world it is very rare. Most cases of vitamin A deficiency in developed countries are caused by malabsorption secondary to intestinal disorder or defective storage and metabolism due to liver disease.[2] We report a case of night blindness secondary to vitamin A deficiency in a patient with neuroendocrine tumour of pancreas.

Case report

A 79-year-old lady presented to the eye clinic with a 4-week history of poor vision in dimly illuminated

conditions. She described difficulty seeing objects in poorly lit cupboards, around the corridors of her house, and difficulty seeing under streetlights in her village. She did not have any problem with daytime vision. She had no history of previous eye problems apart from a left extracapsular cataract extraction with posterior chamber lens implant a year ago. She had undergone a whipple procedure (pancreaticoduodenectomy) 4 years ago for a neuroendocrine tumour of head of pancreas. As the tumour had spread into peripancreatic tissue, entire removal was not accomplished. Immunohistochemistry identified the predominant hormone type to be somatostatin. She was treated with a course of postoperative radiotherapy. Following this she led an active life. Her appetite was good and she maintained a steady weight. She was taking oral creon (pancreatic enzyme supplements) and loperamide for loose stools. At 2 years postoperatively, she presented with an epigastric mass, which was diagnosed as recurrence of pancreatic tumour. She was not given any active treatment as potential benefit was thought to be limited.

On examination, her best-corrected visual acuity was RE 6/9, LE 6/9. Her colour vision tested with ishihara plates was within normal limits. There was no conjuctival or corneal xerosis. Fundal examination showed normal looking optic disc, macula, and peripheral retina in both eyes. Comprehensive electroretinographic tests were undertaken according to IFCN guidelines.[3] These tests revealed profound retinal dysfunction, preferentially involving the rod function but with some significant cone dysfunction as well. Photopic and scotopic ERG amplitudes were reduced in both eyes (Figure 1 and Table 1). Laboratory investigations revealed low serum vitamin A level at 0.13 μmol/l (normal range 0.99–3.35 μmol/l). Antiretinal antibodies were negative for cancer-associated retinopathy (CAR). The Goldmann visual field test showed mild peripheral constriction to I4e. Hypovitaminosis A, most probably secondary to poor absorption, was diagnosed as the cause of her night blindness. The patient was treated with an injection of vitamin A 100 000 IU by intramuscular route. This was repeated after 10 days. A

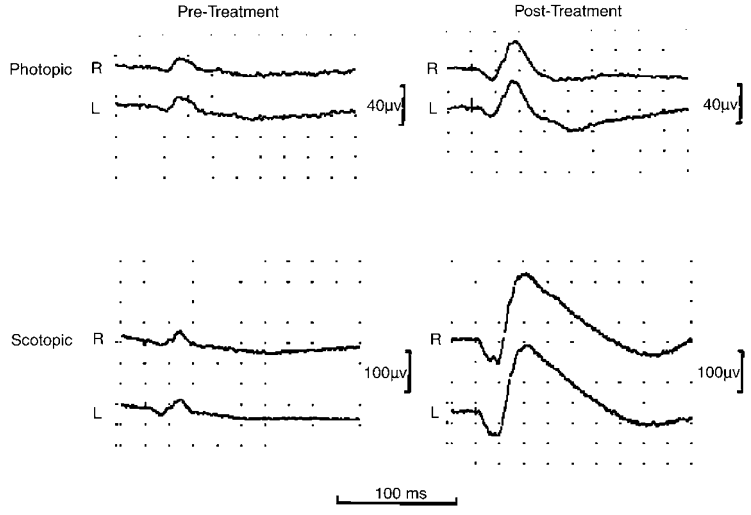

Figure 1 Flash electroretinogrphic responses under photopic and scotopic condition. Pre- and post-treatment with vitamin A. R—Right eye, L—Left eye.

Table 1 Flash electroretinographic studies, pre- and post-treatment with vitamin A

Stimulus used	*Amplitude wave a to b (μV)*				*Normal*
	Pre treatment with vitamin A		*Post-treatment with vitamin A*		
	RE	LE	RE	LE	
Light adapted white flash	14	13	132	92	>50
Light adapted red flash	14	13	38	35	>30
Dark adapted white flash	34	35	220	217	>150
Dark adapted blue flash	13	18	200	200	>150

week after the first injection patient started noticing improvement in her night vision. Serum vitamin A level rose to 0.85 μml/l. Her post-treatment (9 weeks after first vitamin A injection) electrodiagnostic tests showed marked improvement in retinal function. Flash ERG responses under both scotopic and photopic conditions had returned to normal (Figure 1 and Table 1). Our patient continues to remain symptom free with vitamin A injection supplements.

Comment

Vitamin A deficiency is well documented in patients suffering from chronic pancreatic insufficiency (alcoholic pancreatitis, cystic fibrosis) and in intestinal bypass surgery involving jejunum, ileum, and colon.[4–7] However, night blindness due to vitamin A deficiency in relation to pancreatic neoplasm is probably rare and to our knowledge there are only a couple of case reports in medical literature.[2,8]

Vitamin A (retinol) is ingested mainly as retinyl esters and a range of carotenoids, the most abundant of which β carotene is cleaved in the enterocyte to retinaldehyde and then reduced to retinol. Carotenoids are obtained mainly from green vegetables and vitamin A (retinol) from animal products. Vitamin A is mostly absorbed in the upper small intestine. Since hydrolysis is necessary to liberate retinol before it can be absorbed, pancreatic insufficiency may be associated with vitamin A deficiency.[9]

Vitamin A is necessary for the synthesis of visual pigments of rods and cones. In our case, scotopic ERG was affected to a greater extent than photopic ERG indicating more marked involvement of rods over cones. This differential susceptibility to vitamin A deficiency is well documented in previous reports.[5,6]

If night blindness occurs in a patient with a pancreatic neoplasm one should suspect vitamin A deficiency. Another possibility is CAR although there are no reports suggesting its association with pancreatic malignancy and also the electrophysiological findings in CAR are different from vitamin A deficiency-related night blindness.[10] The useful investigations in this setting are serum vitamin A level, antiretinal antibodies, and electrodiagnostic tests. Night blindness due to vitamin A deficiency responds very well to vitamin A supplements.

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Sir,

Conjunctival necrosis and bleb leakage secondary to an adherent conjunctival foreign body

A case of conjunctival necrosis and bleb leakage secondary to an adherent conjunctival foreign body is presented.

Case Report

A 72-year-old gentleman presented to ophthalmic causality complaining of irritation watering