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SCIENTIFIC LETTER

Reversible night blindness – A reminder of the increasing importance of vitamin A deficiency in the developed world

Ceguera nocturna reversible – recordatorio de la importancia creciente de la deficiencia de vitamina A en el mundo desarrollado

Introduction

Essential vitamins are those which must be obtained from diet or supplementation. Malabsorption of various aetiologies, including bowel resection and bariatric surgery, can lead to deficiencies of these vitamins. We report a case of reversible night blindness (nyctalopia) secondary to vitamin A deficiency, which serves as a reminder of the condition and also as a warning that its incidence may be on the rise in the developed world as the popularity of bariatric surgery increases.

Case description

A 55-year-old woman presented with a two-year history of progressive night blindness. She was asymptomatic in daylight, but almost blind in the dark to the extent where she found driving difficult unless weather conditions were clear and bright. She had also experienced bumping into street furniture and parked cars while walking through the town after dark. There was no personal or family history of ophthalmic disease.

She had a medical history of Crohn's disease and three previous bowel resections, which subsequently led to the malabsorptive state of short bowel syndrome. Other than regular vitamin B12 injections, she received no other vitamin supplementation. Her diet included lots of vegetables but she did not eat fruit. She smoked 10 cigarettes daily and drank 5–6 units of alcohol weekly.

On examination, her corrected visual acuity was 6/5 bilaterally. Anterior segment examination was unremarkable, with no evidence of corneal pathology. There were no pupillary abnormalities and Humphrey automated visual fields were full. Fundal examination revealed healthy discs and maculae, but intraretinal white flecks were present in the peripheral retina of both eyes.

The differential diagnosis of nyctalopia was considered, including choroïdæmia, retinitis pigmentosa, gyrate atrophy and vitamin A deficiency. Given her history of bowel resections, vitamin A deficiency was felt to be the most probable underlying cause. Further investigations revealed a reduced serum vitamin A level of 0.3 µmol/l (normal range 1.5–4.2 µmol/l) and full field electroretinography (ERG) demonstrated sub-normal amplitude responses consistent with vitamin A deficiency.

Initial treatment with oral vitamin A supplementation was ineffective, suggesting that the deficiency was related to malabsorption rather than dietary insufficiency. She was commenced on 50,000 units parenteral vitamin A monthly and over the following 18 months, her night vision significantly improved. The white flecks previously seen on fundal examination had resolved. Repeat mixed cone/rod electroretinography was normal bilaterally with bright flash 'a' waves of 205 µV OD, 216 µV OS (normal range 148 ± 45 µV) and 'b' waves of 268 µV OD, 274 µV OS (normal range 290 ± 76 µV), objectively confirming her symptomatic improvement. There was a tendency for right eye ERG readings to be slightly lower than the left eye, but overall both eyes were well within the normal range for independent testing of both the cone and rod-mediated systems. Pattern electroretinography was also normal in each eye.

Discussion

Vitamin A is essential in the eye for corneal and conjunctival epithelial cell RNA and glycoprotein synthesis. Retinal (vitamin A aldehyde) is a vital component of the photo-transduction process which underlies human vision. The systemic manifestations of hypovitaminosis A are varied, but among the ocular complications are conjunctival and corneal xerosis, keratomalacia and retinopathy. However, nyctalopia is the earliest and most common symptom. These complications can lead to significant morbidity or permanent visual loss if untreated.

If recognised early, recovery of visual function may be achieved with adequate and sustained repletion therapy. However, due to the presence of considerable liver stores of vitamin A, development of deficiency-related symptoms can occur many years after surgery.¹ This situation, combined with the relative rarity of vitamin A deficiency in the developed world, can lead to delays in diagnosis and treatment.^{2,3}

Vitamin A deficiency is primarily a problem in developing countries due to malnutrition.² It remains uncommon in developed nations, occurring primarily in patients with liver disease, severe malabsorption or who have had small bowel surgery. However, the incidence may be on the rise with the recent increase in bariatric surgery, which is gaining popularity worldwide due to increased obesity.⁴ This leads to a similar malabsorptive state, with deficiency in fat soluble vitamins having been demonstrated in 69% patients four years post-bariatric surgery.⁵ There is an argument that patients with a history of short bowel or bariatric surgery should undergo regular screening for vitamin deficiencies in order to prevent late complications.

As the ophthalmic complications of vitamin A deficiency tend to be reversible with adequate treatment, it is a condition which all professionals involved in the assessment of visual dysfunction should be aware of. In particular, vitamin A deficiency should be prominent in the differential diagnosis of night blindness, which can be confirmed or refuted with a simple blood test.

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Conflicts of interest

The authors declare not to have any conflicts of interest.

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