



Reversible Night Blindness in an Adult – A Case Report

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ABSTRACT

Vitamin A deficiency is rare in adults, still rarer is the fundus changes due to it. We report a case of 55 years old male with post gastric bypass surgery status, presenting with defective distant vision for 2 years whom on investigation revealed fundus changes, defective colour vision, abnormalities in OCT and ERG and low serum retinol levels who was treated successfully with Vitamin A therapy.

KEY WORDS: Fundus, Vitamin A

INTRODUCTION

Vitamin A deficiency due to malnutrition is the leading cause of childhood blindness in the developing world. It is rare in adults and can occur as a result of malabsorption, poor vitamin metabolism due to liver disease or cystic fibrosis. Recently, in developing countries like India, due to increased number of bariatric procedures and malabsorption syndromes, vitamin A deficiency in adults is on the rise. We report two cases with reversible night blindness due to vitamin A deficiency.

CASE REPORT

A 55 year old male presented with complaints of defective distant vision in both eyes for 2 years, which started initially with difficulty in night vision. He was a known diabetic for past 1 year, on insulin therapy. He underwent gastric bypass surgery for peptic ulcer disease 10 years back. On

Examination, his best corrected visual acuity in both the eyes was 5/60 NIG NIP. Anterior segment was normal in both eyes. Fundus examination of both eyes showed mild disc pallor with diffuse retinal pigment epithelial mottling (fig 1 & 2).

Right eye

Left eye

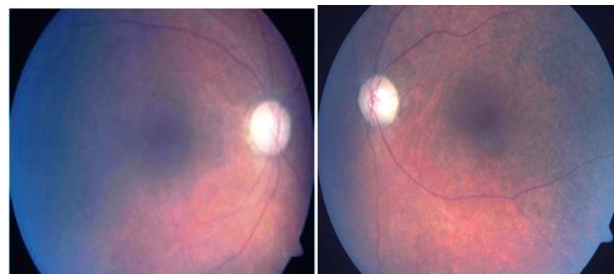


Fig-1

Fig-2

On Colour vision testing he could not read any plate. Central fields of both his eyes showed tubular vision with peripheral constriction of fields. Optical Coherence Tomography (OCT) showed thin epi-retinal membrane with disruption

of inner segment–outer segment junction. Electro-Retinogram (ERG) showed diminished rod and cone responses (fig-3)

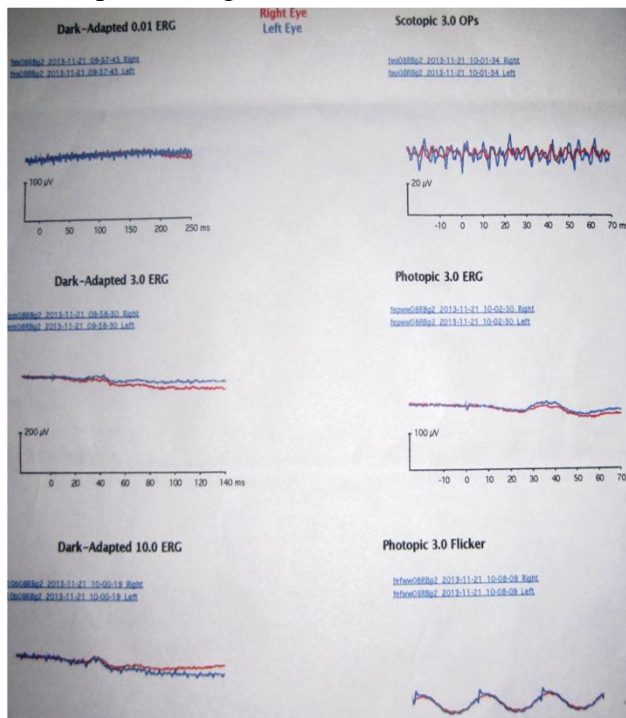


Fig-3: ERG picture before treatment

A differential diagnosis of Albinism, Cataract, Glaucoma, Gyrateatrophy, Oguchi's Disease, Optic atrophy, Retinitis Pigmentosa, Siderosis, Uncorrected myopia, Vitamin A deficiency and Zinc deficiency were considered. After ruling out other possibilities Vitamin A deficiency was strongly considered. His serum retinol level was $<0.1\text{mg/l}$ (N- $0.3\text{-}0.6\text{mg/l}$). Hence the same diagnosis of was made and he was started on injection Vitamin A5000 IU intramuscularly for 10 days and oral Vitamin A capsules 10,000 IU twice daily for 1 month. After one month of treatment, his vision had returned to normal, visual acuity had improved to 6/6 in both the eyes and fundus examination was normal in both the eyes. He also showed improvement in colour vision. His serum vitamin A level was still below normal but had increased to 0.26 mg/L . A repeat visual field was performed at that visit which showed an improvement in both visual fields. Repeat ERG showed improved rod and cone responses (fig-4 & 5).

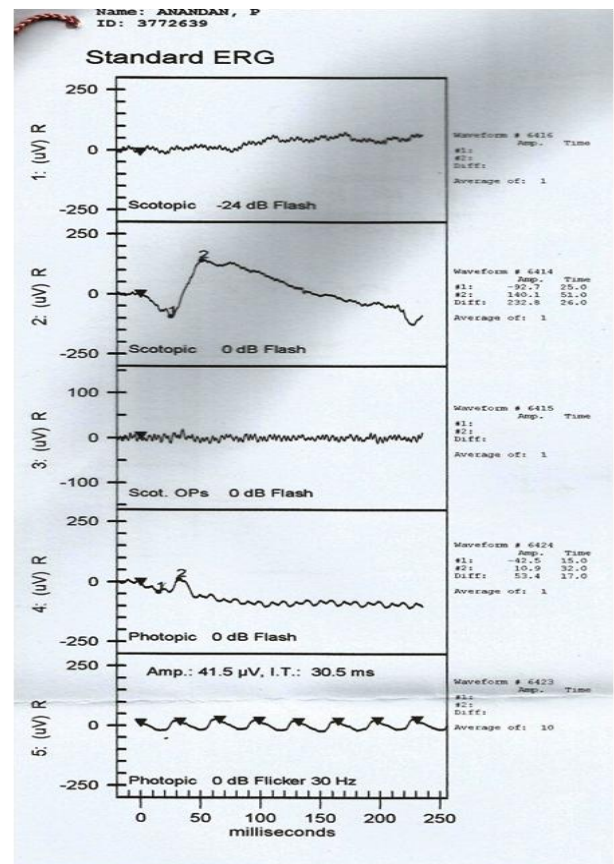


Fig-4

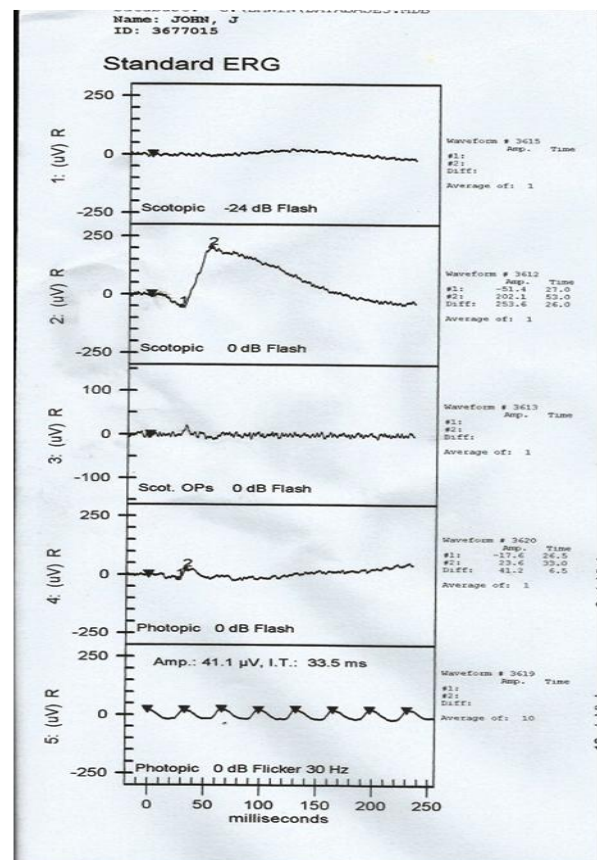


Fig-5

DISCUSSION

Xerophthalmia is a term used to describe the spectrum of ocular disease that occurs from vitamin A deficiency. These changes include dry eye (xerosis), corneal ulceration and melting (keratomalacia), night blindness (nyctalopia), and retinopathy^[1] Vitamin A is also essential for immune function, and affected children are more susceptible to severe infections, such as measles. Vitamin A is ingested in the form of retinaldehyde from milk, meat, fish, liver, and eggs. It is also ingested as carotene from green leafy vegetables, yellow fruits, and red palm oil. These compounds are stored in the liver in the form of retinylpamitate. The aldehyde form of vitamin A, retinaldehyde, combines with the protein opsin in the rods to create rhodopsin, which is a photosensitive pigment. A similar process takes place in the cones. During photo transduction, some retinal is lost so a constant supply of vitamin A is needed.

Vitamin A deficiency can therefore lead to night blindness with associated visual field changes and a depressed ERG. Vitamin A is also necessary for the maintenance of specialized epithelial surfaces. In the conjunctiva, loss of goblet cells and squamous cell metaplasia leads to dryness or xerosis. Bitot's spots are perilimbal gray plaques of keratinized conjunctival debris overlying an area of xerosis^[5]. A full-thickness liquefactive necrosis of the cornea (keratomalacia) can also occur. Finally, retinopathy in the form of yellow or white punctate dots can be seen in the retinal periphery^[4]. Testing for vitamin A deficiency is available in the form of retinol, retinylpamitate, and retinol binding protein levels.

The World Health Organization recommends 200,000 IU of vitamin A for 2 days followed by another dose 2 weeks later for severely malnourished children older than 12 years of age with vitamin A deficiency (WHO 1999);^[6] however, more recent case studies have shown that much less supplementation can lead to reversal of symptoms. Functional improvement of

the retina is likely to occur before ophthalmoscopically evident improvement of structural changes in fundus^[3] Vitamin A can also be given intramuscularly if there is a concern for severe malabsorption or corneal involvement requiring a faster recovery.^[2]

CONCLUSION

Vitamin A deficiency in adults is rare and fundus changes in vitamin A deficiency is still rarer. Our patient had a fairly quick recovery on vitamin A supplementation. This reinforces the importance of considering vitamin A supplementation in all patients who have undergone bowel resection and in patients with underlying liver disease.

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