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CLINICAL PICTURE

Night blindness, Bitot's spot and vitamin A deficiency

A 19-year-old male, known case of non-transfusion dependent thalassemia (NTDT), hailing from poor socioeconomic status presented with gritting sensation in both the eyes and decrease vision during the night for 1 month. No history of diplopia, ptosis or swelling and there was no history of any ocular injury. Examination of eyes revealed whitish, foamy lesions on the temporal side of the ocular conjunctiva (Bitot's spot) (Figure 1). Examination on Snellen chart showed visual acuity of 6/9 on the right and 6/12 on left eye with normal color vision. Rest of the ocular and neurological examination was unremarkable. On investigation, his vitamin A level was 0.2 µmol/l (normal range 0.7 µmol/l to 1.04 µmol), which confirmed the diagnosis of vitamin A deficiency with xerophthalmia. The patient was treated with oral vitamin A 200 000 IU on day 0, 1 and at 2 weeks. One month later, he was asymptomatic with near complete resolution of Bitot's spot.

Vitamin A deficiency is a worldwide cause of avoidable blindness but more rampant in developing countries like India.¹ Vitamin A deficiency (VAD) is more prevalent in children, and

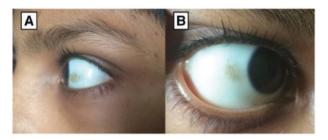


Figure 1. Clinical photography of the left eye (A) and right eye (B) showing whitish, foamy lesions on the temporal side of the ocular conjunctiva (Bitot's spot).

most common causes are malnutrition followed by malabsorption.² Clinical symptoms and signs range from mild eye discomfort, night blindness to total loss of vision, which is grouped under a single entity, called as xerophthalmia. Bitot's spots are a specific manifestation of Vitamin A deficiency. These are triangular dry, whitish, foamy appearing lesions which are located more commonly on the temporal side.³ They mainly composed of keratin admixture with gas-forming bacteria Corynebacterium xerosis, lead to foamy appearance. It is a fully reversible condition with oral vitamin A replacement. However, if untreated, it heralds the complete loss of vision.

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References

- 1. Kapil U, Sachdev HPS. Massive dose vitamin A programme in India–Need for a targeted approach. *Ind J Med Res* 2013; **138**: 411–7.
- 2. Laxmaiah A, Nair MK, Arlappa N, Raghu P, Balakrishna N, Rao KM, et al. Prevalence of ocular signs and subclinical vitamin A deficiency and its determinants among rural pre-school children in India. Public Health Nutrition 2012; 15:568–77.
- 3. Ferrari G, Vigano M. Images in clinical medicine. Bitot's spot in vitamin A deficiency. N Engl J Med 2013; 368:e29.