Vitamin A Deficiency-induced Keratomalacia Due to Malabsorption Syndrome

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Abstract

Background: Keratomalacia is commonly reported in regions with high levels of malnutrition. We present a rare case of Vitamin A deficiency-induced keratomalacia due to malabsorption syndrome in Brunei Darussalam, a country with one of the highest per-capita gross domestic products in Southeast Asia - highlighting the importance of considering nutritional deficiencies even in affluent settings.

Case presentation: An 18-year-old Malay male presented with right eye redness, congestion and hand movement vision. He had atopic dermatitis, asthma, hyper Ig E syndrome, and multiple vitamin deficiencies, including vitamin D, vitamin B12, and iron. He was HLA dQ8 and DQ7 positive, DQ7 being a risk allele for coeliac disease. On initial examination, there was a right shield ulcer, which was treated for vernal atopic keratoconjunctivitis. Over a period of one month his skin and eye condition worsened, with xerosis of the conjunctiva, corneal thinning, perforation of corneal shield ulcer with an iris plug and shallowed anterior chamber. The clinical signs strongly suggest Vitamin A deficiency causing keratomalacia. He was admitted for systemic steroid treatment and given conservative intensive treatment of lubricants and topical antibiotics (moxifloxacin, tobramycin, and atropine). At six months review, and through a comprehensive treatment approach including Vitamin A supplements, carotenoid-rich food, the patient demonstrated a remarkable restoration of visual acuity of 6/9 in the affected eye.

Conclusion: Although Vitamin A deficiency is more common in developing countries, this case highlights that it can occur in a socioeconomically developed setting and may present with atypical features. Thorough history taking and clinical correlations are important to guide timely diagnosis and management in similar cases. Early recognition and prompt intervention are crucial to prevent irreversible visual loss.

Keywords: Vitamin A Deficiency, Keratomalacia, Xerophthalmia, Ulcer

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Introduction

Vitamin A deficiency (VAD) remains a significant global public health problem, disproportionately affecting populations in low-income and developing countries.[1] In developed nations, it can present in patients with malabsorption syndrome or those who have undergone extensive large bowel resections.[2] Its profound impact on ocular health has earned it a reputation as one of the leading causes of preventable blindness, particularly in regions where access to diverse and nutritious diets is limited. The World Health Organization (WHO) estimates that more than 250 million preschool children worldwide have VAD, and about three million have xerophthalmia, with about 25% at risk of going blind.[2] Vitamin A is needed in rod production in the retina as well as in maintenance of ectoderm derived tissues such as epithelium as in the conjunctiva and cornea. Deficiency of the vitamin can lead to xeropthalmia, corneal melt or keratomalacia and eventually cornea perforation if not treated urgently. Beyond ocular manifestation, VAD is associated with a range

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of systemic complications, including xerosis of the skin, follicular hyperkeratosis, increased susceptibility to respiratory tract infections, delayed growth development, and even infertility. The treatment is straightforward with oral Vitamin A but in cases where malabsorption becomes an issue, it has provided a challenge to treat these patients. Moreover, as Vitamin A deficiency is a rare occurrence in developed countries, clinicians tend to overlook this. We aim to increase awareness of the importance of early diagnosis, and prompt management in preventing irreversible visual impairment and blindness, irrespective of the geographical and socioeconomic context.

Case report

An 18-year-old Malay male with atopic dermatitis presented with right eye redness and congestion with blurring of vision. The right eye visual acuity (VA) was hand movement and 6/9 in his left eye. He has a hyper IgE syndrome with multiple vitamin deficiencies including Vitamin D, Vitamin B12 and iron due to his restricted diet. He is HLA dQ8 and DQ7 positive, DQ7 being a risk allele for coeliac disease. He was also a known asthmatic. On examination, there was a right shield ulcer and was initially treated for vernal atopic keratoconjunctivitis with sub tarsal dexamethasone injection, topical 1.5% levofloxacin and topical dexamethasone. A bandage contact lens was placed to prevent further deterioration of the corneal epithelial defect. The left eye was clinically unremarkable with no signs of conjunctival or corneal involvement.

Over a period of one month, his skin condition deteriorated, and he was hospitalized for systemic steroid therapy. His eyes condition also worsened leading to xerosis of the conjunctiva, corneal thinning, and eventually spontaneous perforation of corneal shield ulcer with an iris plug and shallowed anterior chamber (Figure 1). He was treated with intensive topical antibiotics moxifloxacin, tobramycin, atropine, and lubricants. Conservative management was opted over surgical intervention in view of active systemic disease, underlying immunodeficiency (hyper IgE syndrome), and the high risk of poor wound healing and infection. Additionally, the corneal perforation healed spontaneously, accompanied by deepening of the anterior chamber and progressive healing of the corneal ulcer.

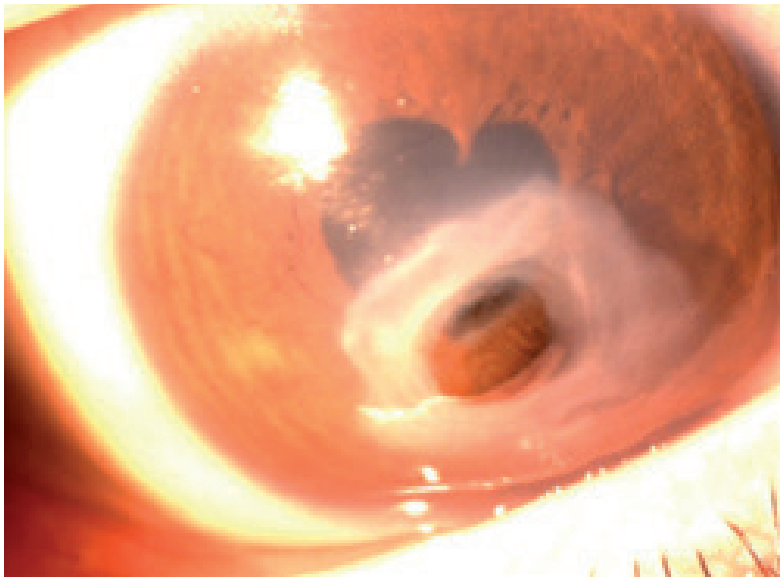

Figure 1: Shield ulcer - perforated corneal

Two weeks later, he returned to see us with severe dry eyes (xerophthalmia) and worsening of vision, this time in the left eye. Visual acuity in the right eye was 3/60 and, in the left, 6/18. The conjunctiva and cornea of both eyes had begun to keratinise. The patient reported that he had reduced the frequency of the lubricating gel from hourly to four times a day due to unavailability of stock. After reviewing the history and clinical findings particularly the rapidly resolving punctate keratopathy, conjunctival and corneal xerosis, following consumption of Vitamin A and carotenoid-rich foods-Vitamin A deficiency-related xerophthalmia was suspected. The initial differential diagnoses included worsening vernal keratoconjunctivitis, severe dry eye disease secondary to atopic dermatitis, and infectious keratitis. However, the presence of conjunctival and corneal xerosis, along with rapid improvement after nutritional intervention, supported Vitamin A deficiency as the underlying cause.

Within a week, his symptoms significantly decreased. Gradually, the corneal scar reduced in size and the dryness of eyes improved considerably. Review at six months showed significant improvement of visual acuity to 6/9 with reduced corneal scarring (Figure 2).

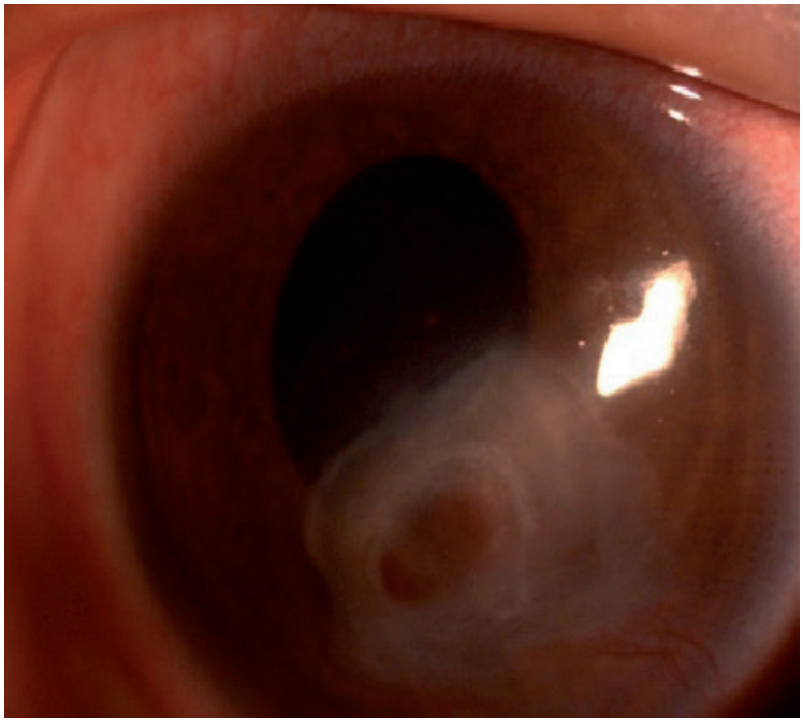

Figure 2: Sealed corneal perforation

Systemic investigations, including serum Vitamin A levels and autoimmune or gastrointestinal screening, were not conducted due to limited access and a primary focus on acute ophthalmic management. The patient's parents independently initiated oral Vitamins A and C supplementation without a documented dose. Additional treatments such as oral doxycycline was not prescribed. While doxycycline can be beneficial in managing inflammatory corneal melts due to its anti-collagenolytic effects, it was not initiated, as the clinical course here was consistent with nutritional keratopathy, and the patient responded well to conservative and nutritional therapy alone.

Discussion

Vitamin A is a fat-soluble vitamin that is mainly found in liver and dairy products, or otherwise can be formed in the small intestines from the splitting of carotenoids of plant origin. It is stored in the liver as retinol palmitate and then released and transported to target cells as retinol in a one-to-one molar association with retinol-binding protein. Cellular receptors bind this holo-RBP complex, allowing the retinol to enter the cell. There are other forms of vitamin A, such as retinoic acid, which circulates in small amounts, have specific cellular receptors, and plays important roles in other physiologic functions. Vitamin A is required in the maintenance of mucus-secreting epithelia. Keratinisation of mucosal epithelium is the most prominent presentation in VAD.[3]

Low levels of Vitamin A results in xerophthalmia of increasing severity. Since Vitamin A is involved in rod vision, the earliest clinical manifestation is impaired dark adaptation and night blindness. However, this was not observed and tested in this patient. Subsequently an oval or triangular patch of temporal conjunctiva develops a typical dry, non-wettable, xerotic appearance. A Bitot's spot is formed when the area is covered by a superficial foamy substance. Histopathologic examination of the lesion will show typical keratinizing metaplasia, loss of surface infolding and microvilli, absence of goblet cells, and formation of prominent keratin and granular cell layers. The superficial foamy substance is composed of desquamated keratin and saprophytic diptheroids. However, it has been found that generally goblet cells are absent throughout the conjunctiva in VAD cases.[4]

The three main ocular effects of VAD are night blindness, xerophthalmia and keratinisation. It can also cause anaemia through various biological mechanisms and is associated with low serum iron and transferrin concentrations with excess iron in stores and a characteristically low lymphocyte count.[3] This explains the anaemia observed in our case. With adequate Vitamin A, the anaemia improves due to more efficient utilisation of iron stores for haematopoiesis. Vitamin A levels are assessed by measuring serum retinol concentration, which accounts for the majority of Vitamin A in serum. However, this does not reflect the total body status of Vitamin A unless levels are extremely high or low, as the serum retinol concentration is kept constant over a wide range of intakes.[5]

The pathogenetic mechanisms responsible for corneal ulceration and necrosis in VAD are unclear, involving a cascade of structural and biochemical changes. Vitamin A helps maintain healthy mucosal surfaces; without it, the corneal epithelium becomes keratinized, and goblet cells are lost, reducing mucin and destabilizing the tear film.[3] This dry, damaged surface is prone to infection and enzyme activity. In response, neutrophils and epithelial cells release matrix metalloproteinases (especially MMP-9 and MMP-2), which break down stromal collagen, causing softening and liquefactive necrosis seen in keratomalacia.[6]

Clinically, xerophthalmic ulcers have a classical appearance. Most are small, round to ovoid punched-out craters or full thickness perforations. Sometimes, they resemble trephine lesion, located peripherally in the inferior or nasal quadrants, and respond rapidly to vitamin A. Smaller lesions usually resolve quickly, with viable stroma, and usually visual axis spared. In larger lesions much of the necrotic stroma sloughs, leaving a descemetocele that scars slowly.

According to the WHO grading, the different ocular signs of VAD are night blindness (XN), conjunctival xerosis (X1A), Bitot's spots (X1B), corneal xerosis (X2), corneal ulcer covering less than a third of the cornea (X3A), corneal ulcer covering at least a third of the cornea, defined as keratomalacia (X3B), and corneal scarring (XS).[3]

In our patient, initial examination revealed a shield ulcer in the right eye, which later progressed to conjunctival xerosis and corneal thinning, eventually resulting in spontaneous perforation of the cornea. These findings correspond to WHO grades X1A (conjunctival xerosis), X2 (corneal xerosis), and X3B (corneal ulceration involving more than one-third of the cornea with signs of keratomalacia). This progression, in the setting of multiple nutritional deficiencies including Vitamin A and an underlying immunodeficiency (hyper IgE syndrome), highlights the severity of his ocular involvement. Night blindness (XN) was not reported by the patient and was not actively assessed at the time of presentation. Following initiation of nutritional therapy,

including Vitamin A supplementation such as orange, yellow fruits and vegetables like carrots, sweet potatoes, apricots and dark green leafy vegetables such as spinach and kale; there was notable improvement, with resolution of corneal xerosis, punctate keratopathy and spontaneous healing of the corneal ulcer. This response is consistent with the known rapid epithelial regeneration following vitamin A repletion, typically occurring within four to seven days, during which keratin and granular cell layers disappear and microvillous projections reappear-a pattern also observed in our patient.[7]

The WHO recommends an oral dose of 200000 IU of Vitamin A for two days consecutively, followed by another dose one to four weeks later. In patients where malabsorption is an issue, or with severe ocular involvement, intramuscular Vitamin A injection, are recommended.[8] This case report, however, emphasises the need of early diagnosis and treatment of VAD, which would have prevented progression to corneal perforation.

Given that Vitamin A deficiency is rare in developed countries, this case also highlights the need for increased clinical vigilance and consideration of targeted screening in high-risk individuals-such as those with restrictive diets, malabsorption syndromes, or chronic inflammatory conditions-to allow timely intervention and prevent sight-threatening complications.

This case is notable for the severe ocular complications of Vitamin A deficiency, including corneal perforation, occurring in a patient with hyper IgE syndrome and multiple nutritional deficiencies but without classic gastrointestinal disease. Vitamin A deficiency generally presents with bilateral and symmetrical clinical signs; however, this patient exhibited an asymmetrical and sequential presentation, which made establishing the diagnosis more challenging. Such a presentation is uncommon and highlights the diverse clinical settings in which Vitamin A deficiency can manifest.

For optimal management, early identification and treatment of at-risk patients are essential. While the WHO oral vitamin A protocol remains standard, intramuscular administration should be considered in cases with malabsorption or severe eye involvement to prevent vision-threatening complications.

Limitations of this report include the lack of long-term follow-up data and absence of objective assessments such as serum vitamin A levels or formal evaluation for night blindness. Despite these, the marked clinical improvement after vitamin A therapy supports the diagnosis and treatment approach.

Conclusion

Diagnosis of VAD depends primarily on a thorough patient history and clinical findings such as night blindness, corneal ulcers and Bitot's spots. In the presence of corneal lesions which constitute a medical emergency, early clinical suspicion is crucial, especially in atypical patients with restrictive diets or malabsorption. Nutritional assessment should be part of managing chronic eye conditions. Prompt treatment with oral vitamin supplements can prevent further deterioration in vision and the resolution of epithelial defects, while patients with poor absorptive states, intramuscular or intravenous Vitamin A supplements may be necessary. In view that VAD is a rare occurrence in developed countries, clinicians tend to overlook this. Through this case report, we hope to raise awareness among ophthalmologists of the importance of early diagnosis and prompt treatment to prevent severe ocular complications.

Patient Consent

Written informed consent was obtained from the patient for participation and publication of this report and related photo.

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Declaration of competing interest

The authors declare that there is no conflict of interest.

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