

Corneal Stigmata of Acute Malnutrition: A Case Series

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ABSTRACT

Childhood malnutrition is one of the leading causes of under-five mortality due to increased susceptibility of infections and slow recovery from illness, making it a major public health problem. Ocular involvement in malnutrition is common and more severe in early childhood. Acute malnutrition is an important cause of preventable blindness. Deficiencies of micronutrients and retinol, folate and minerals like iron, magnesium and potassium as well as other vitamins are usually seen in acute malnutrition. Vitamin A deficiency is particularly important, as it can lead to keratomalacia and permanent blindness. The present case series of children (under-five years) elaborates on the ocular consequences of acute malnutrition like conjunctival xerosis, corneal ulceration, keratomalacia, corneal abscess, corneal perforation, desmetocele, corneal scarring and xerophthalmia associated with vitamin A deficiency and their management. It emphasises the importance of preventing xerophthalmia to reduce early childhood blindness. Active corneal xerophthalmia is a medical emergency that should be treated immediately with high dosage of vitamin A. The high-dose vitamin A treatment is recommended for infants and young children with xerophthalmia, severe malnutrition, and measles. Prompt diagnosis and timely intervention are required to prevent the ocular morbidities associated with vitamin A deficiency and acute malnutrition. Vitamin A supplementation programs should be made universally available in all populations where vitamin A deficiency is a public health problem.

Keywords: Keratomalacia, Ocular morbidity, Vitamin A deficiency, Xerophthalmia

INTRODUCTION

Malnutrition among children under five years of age is very high and a major health problem affecting the development of children in India [1]. Acute malnutrition is a nutritional deficiency resulting from inadequate energy or protein intake. Paediatric malnutrition is defined as an imbalance between nutrient requirement and intake, resulting in cumulative deficits of energy, protein or micronutrients that may negatively affect growth, development and other relevant outcomes [2]. Acute malnutrition pertains to a group of linked disorders that includes kwashiorkor, marasmus and intermediate states of marasmic kwashiorkor.

Severe Acute Malnutrition (SAM) is one of the main public health problems worldwide and in addition to general symptoms, is responsible for vitamin A deficiency, which can cause serious ocular damage including keratomalacia [3]. Vitamin A deficiency is the leading cause of preventable childhood blindness in the developing world. Vitamin A deficiency has a wide range of ocular manifestations including night blindness, conjunctival xerosis, Bitot's spots, corneal xerosis, corneal ulceration and keratomalacia, corneal scarring and xerophthalmic fundus [4].

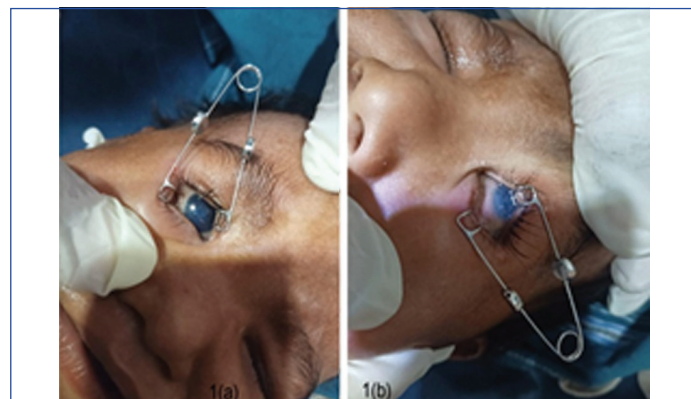
A case series depicting corneal stigmata of acute malnutrition due to vitamin A deficiency is described herewith. In all the cases, ophthalmological evaluation was done by holding the child on the baby cot, instillation of topical anaesthetic drops - Proparacaine eye drops and insertion of ultra-light weight paediatric speculum. There was no need for sedation or examination of the child under general anaesthesia.

CASE SERIES

Case 1

A 10-month-old male child presented with complaints of loose stools and cough since 7 days, diagnosed as complicated SAM with acrodermatitis. He weighed 5.25 kg and complained of inability to open the eyes and increased watering, and was referred for ophthalmological evaluation by the paediatrician.

Ophthalmological evaluation revealed conjunctival xerosis with corneal ulceration in the right eye, and in the left eye, corneal ulceration involving more than one third of corneal circumference, with congestion with conjunctival xerosis and keratomalacia [Table/Fig-1a,b]. The fundus in the right eye was normal. Serum retinol and retinol binding protein tests were not conducted due of financial constraints.



[Table/Fig-1]: (a) Corneal ulcer with conjunctival xerosis (right eye) and (b) Keratomalacia with conjunctival xerosis (left eye).

He was given 1,00,000 IU of vitamin A on days 1,2 and 14. He was advised to use antibiotic eye drops (tobramycin) every hour, lubricating eye drops-Carboxymethyl Cellulose (CMC) every hour and lubricating eye ointment gel at night and atropine eye drops three times a day. The patient showed dramatic improvement in right eye after one week and in the left eye after two weeks. Another week later, the congestion had disappeared in the left eye, and ulcers in both eyes had healed on medical management. On follow-up at six weeks, the right eye was completely normal with no sequelae, while the left eye had a residual corneal opacity.

Case 2

A two year and six-month-old female child complaining of diarrhoea and vomiting and was diagnosed with complicated severe acute

malnutrition by the paediatrician. She weighed 4.5 kg and was referred for ophthalmological examination as she complained of pain, watering and blepharospasm.

Ophthalmological evaluation revealed conjunctival xerosis with central corneal abscess in right eye, and conjunctival xerosis with an inferior corneal abscess and iris incarceration in the left eye [Table/Fig-2a,b]. Ultrasonography (USG) B scan was normal in both eyes. Serum retinol and retinol binding protein tests were not conducted due to financial constraints.



[Table/Fig-2]: (a) Corneal abscess with conjunctival xerosis (right eye) and (b) Corneal abscess with iris incarceration with conjunctival xerosis (left eye).

The patient was informed about the possibility of perforation in left eye and the urgent need of penetrating keratoplasty at higher centre, which was refused. She was advised to take 2,00,000 IU of vitamin A on days 1,2 and 14. Corneal scrapping was not attempted due to the risk of impending perforation. She was advised antibiotic eye drops (tobramycin) hourly, antifungal eye drops-natamycin hourly, cycloplegics (atropine) eye drops three times a day and lubricating eye drops three times a day. After two weeks, there was marked clinical improvement, and after four weeks, there was leucomatous corneal opacity in the right eye and adherent leucoma in the left eye. The patient was explained about the possibility of amblyopia and was advised penetrating keratoplasty in both eyes but the patient was not willing to go to higher centre for the further treatment.

Case 3

A three-year-old female presented with chief complaints of fever with cold and diarrhoea since five days and was diagnosed as severe acute malnutrition with severe dehydration. The patient weighed 6 kg. She was unable to open her eyes and was referred for ophthalmological evaluation.

Ophthalmological examination showed conjunctival xerosis and an epithelial defect with corneal haze in the right eye, with a corneal ulcer involving more than 1/3 of corneal surface (keratomalacia). In the left eye, there was conjunctival xerosis with total corneal abscess with desmetocele [Table/Fig-3a,b]. USG B-scan was normal in both eyes. The possibility of perforation in the left eye was explained to



[Table/Fig-3]: (a) Keratomalacia (right eye) (b) LE Total corneal abscess with descemetocoele (left eye).

the patient. Urgent penetrating keratoplasty should be performed in such patients. The patient was poor and did not wish to go to higher centre. Serum retinol and retinol binding protein tests were not done because of financial constraints and corneal scrapping was not attempted in left eye because it might lead to corneal perforation.

The patient was advised to take 2,00,000 IU of vitamin A on days 1,2 and 14. The patient was advised to use antibiotic eye drops (tobramycin) hourly in both eyes, antifungal drops (natamycin) eye drops hourly in the left eye, cycloplegic atropine eye drops three times a day in both eyes, lubricating eye drops hourly in both eyes. There was dramatic improvement in the right eye after two weeks, and decrease in infection in the left eye. After four weeks, corneal opacity was developed in right eye and a leucomatous corneal opacity developed in left eye. The infection subsided in the left eye leaving a dense leucomatous corneal opacity. Efforts were made to convince the patient regarding penetrating keratoplasty in the left eye at higher center, and the possibility of amblyopia was explained but the patient was reluctant to go.

DISCUSSION

Ocular involvement in severe acute malnutrition is common due to vitamin A deficiency, and it is more serious during early childhood. Vitamin A deficiency due to acute malnutrition is endemic in developing countries such as southeast Asia and sub-Saharan Africa where it is leading cause of childhood blindness [5]. Vitamin A deficiency is leading cause of preventable blindness in developing and underdeveloped nations [6].

The findings in the present series, as in cases one and three, were similar to findings in a photo essay by Madan S et al., in which a one-and-half-month-old infant, immunised for age, developed bilateral keratomalacia following an episode of diarrhoea. The right eye had a corneal abscess and the left eye had a near total corneal haze with a central 1.5×1.5 mm ulcer. Management included prompt administration of vitamin A 50,000 IU (day 0,1 and 14), oral and topical antibiotics, and a cycloplegic. After 14 days, the cornea of the right eye appeared hazy; however the cornea left eye cleared significantly. After one week, the congestion had disappeared, and the ocular surface appeared dry. Over a period of four weeks, the ocular surface appeared moist, and ulcers in both eyes healed with medical management, avoiding the need for urgent keratoplasty [5].

A case of bilateral corneal melting in a two-year-old paediatric patient with severe vitamin A deficiency was reported by Roheen ZU et al., The patient was taken for Exam Under Anaesthesia (EUA), which revealed chemotic and keratinised conjunctiva in both eyes with xerosis but no Bitot's spot. There was corneal melting with collapsed anterior chamber in both eyes, and the right eye had a purulent iris that was affixed to the lens by extensive posterior synechiae. The patient's right eye was eviscerated because of poor visual potential and risk of sympathetic ophthalmia, and the left eye was treated with temporary tarsorrhaphy and referred to a specialised eye institute for keratoplasty. In present case series, as compared to this case report, there was no corneal melt or chemosis of the conjunctiva, and there was no need of evisceration or tarsorrhaphy [7]. In a study by Raichandani D et al., which included a total of 100 severely acute malnourished children, vitamin A deficiency signs were observed in 17 eyes (8.5%). Conjunctival xerosis was the most common finding observed in 10 (58.9%) eyes. Corneal xerosis, keratomalacia, and corneal perforation were each documented in 2 (11.8%) eyes each. No cases presented with Bitot's spots [1]. In a study by Boro A et al; 6.9% cases reported ocular involvement due to vitamin A deficiency including one case of Bitot's spot, four cases of conjunctival xerosis and one case of keratomalacia. The patients received a dose of vitamin A; all ocular lesions had regressed after evaluation in one week, with the exception of the case of keratomalacia. This eye unfortunately lost visual function [3].

Anandakumar TS et al., documented conjunctival xerosis in 36% cases of severe acute malnutrition. Bitot's spots were seen in 24% cases, corneal scar was seen in one case. Night blindness was not noted in any children [8].

Systemic affections such as pneumonia, diarrhoea, jaundice, vomiting and septicaemia and measles may also precipitate vitamin A deficiency [5]. Other factors responsible for vitamin A deficiency are malabsorption, impaired transport and storage of vitamin A, which may develop following gastrointestinal surgery, malabsorption syndrome, or due to dietary factors [5]. By improving immune function, vitamin A reduces mortality associated with measles, diarrhoea and other illness [9].

Vitamin A regulates corneal epithelial cell proliferation, goblet cell differentiation, and mucin production, thereby improving tear film stability [10]. Its deficiency leads to epithelial keratinisation, making tissue harder and resistant to wetting [11]. Corneal xerosis is the drying of the cornea and is a sign of sudden, acute deficiency. The cornea becomes dry because conjunctival glands are not functioning normally, and the goblet cells which secrete mucous to maintain moistness is reduced [11]. This leads to loss of tears and mucous, both of which acts as wetting agents. The lack of mucous and tears not only leads to dry appearance but also increases the risk of infection [4].

The most severe form of xerophthalmia is keratomalacia, as seen in the present case series (cases 1 and 3), where more than one third of cornea is affected. The cornea may become oedematous and thickened, and then melt away. This occurs because the collagen of the cornea is affected by necrosis [4]. The cornea can be destroyed within a few days. Lack of immunisation and malnutrition also predispose children to keratomalacia. The end result of corneal ulceration and keratomalacia is corneal scarring, staphyloma or phthisis bulbi [4].

The severity of xerophthalmia is directly proportional to the severity of malnutrition. Another micronutrient that affects the cornea due to malnutrition is riboflavin. In riboflavin deficiency, there is congestion of limbal plexus, invasion of stroma, and corneal neovascularisation, presenting as burning sensation, lacrimation and photophobia. Riboflavin deficiency also produces phlyctenular keratoconjunctivitis, rosacea keratitis and pannus of trachoma [11].

Prophylactic vitamin A supplementation of three groups -infants and young children (0-59 months), pregnant females, and postpartum females within six weeks after delivery is recommended in all populations in which vitamin A deficiency is important public health problem [12]. High doses of vitamin A should not be given to females who could be pregnant because of the risk of teratogenic effects in the foetus. The dosage of vitamin A is as follows: for young infants (0-5 months) is 50,000 IU, older infants (6-11 months) is 1,00,000 IU and for children males (12 months or more) and females (12 months to 12 years and >50 years) is 2,00,000 IU. In severe malnutrition, the above dose is given on day one, measles, on days 1 and 2, and in xerophthalmia, on days 1,2 and 14; and for adults with active corneal lesions 2,00,000 IU on days 1,2 and 14 [12].

Vitamin A deficiency associated with protein-energy malnutrition should be treated for protein deficiency along with vitamin A supplementation, else the symptoms will recur within weeks after therapy.

Improvement in corneal lesions is usually rapid and dramatic. Improvement of active Bitot's spots usually occurs within two weeks, while retina is usually slow to respond to treatment, with night blindness and poor dark adaptation often persisting for at least four weeks [12]. Infants with xerophthalmia are at high risk of death from infectious causes, so treating them with vitamin A will reduce this risk [12]. Night blindness is the earliest symptom of vitamin A deficiency, secondary to depletion of photopigments. Active corneal xerophthalmia is a medical emergency, and most individuals go

blind within 24-48 hours unless they receive effective treatment with vitamin A. The most commonly reported side effects of vitamin A supplementation include irritability, loose stools, headache, fever, nausea, vomiting, and bulging fontanelle in neonates [13]. Complementary interventions needed for vitamin A deficiency control include vitamin A biofortification, micronutrient powders, dietary diversity, nutrition education, and prevention and control of infectious diseases [9]. Breastfeeding should be emphasised as colostrum and breast milk contain vitamin A [11]. The importance of weaning foods rich in Vitamin A, such as mango, papaya, spinach, carrots, sweet potatoes and dark green leafy vegetables, should be elaborated [11]. Surgery may be necessary for corneal perforations; options include tissue adhesive for small perforations, patch graft for medium-sized defects and penetrating keratoplasty for large defects [14].

Paediatric keratoplasty has a high risk of graft failure. Repeated general anaesthesia is a concern in paediatric keratoplasty, for concerns such as loose sutures, broken sutures, anterior chamber reformation, and breaking synechiae in centers with limited general anaesthesia facilities. There is a risk of amblyopia even after successful surgery. The most common postoperative complications are glaucoma, graft rejection, cataract, infection and others such as steroid response, endophthalmitis and peripheral anterior synechiae [15]. Optical iridectomy is effective in cases where penetrating keratoplasty is difficult [5]. The risk of general anaesthesia in malnourished children is also a major concern. Promising results of paediatric keratoplasty have been reported [15].

CONCLUSION(S)

Prevention of malnutrition is a priority for governments and other organisations dedicated to improving child health. Efforts include promoting of nutrient rich weaning foods and increasing the coverage of immunisation and vitamin A supplementation. Proper parental counselling and education about maternal health, immunisation, sanitation, management of malnutrition and prompt ophthalmic intervention in the target population can help prevent ocular morbidity.

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