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Ocular Manifestations of Vitamin A Deficiency in Bundelkhand Region

Dr. Jitendra Kumar[1], Dr. Pradeep kumar tiwari[2], Dr. kanhaiya prasad[3]
1 Associate Professor & Head, Department of Ophthalmology, MLB Medical College Jhansi, India 2 Junior Resident, Department of Ophthalmology, MLB Medical College Jhansi, India
3 senior Resident, Department of Ophthalmology, MLB Medical College Jhansi, India

Purpose: *To evaluates the various ocular effect of vit-A deficiency and Various factor which increases the VAD susceptibility.*
Method: *A total of 120 Patients who were diagnosed as a case of vit-A deficiency (any sign or symptoms of VAD), were included in this cross sectional study. An assessment of present complaints, detailed clinical history, BMI was recorded. Ophthalmological check up as external examination of the eyes, visual acuity, torch light examination, slit lamp examination, Fluorescein eye staining, Schirmer's test, refraction, direct ophthalmoscopy, was done.*
Results: *In this study, the male female ratio was 1:1.2. Vit-A deficiency was most commonly observed in children with 5-9 years of age group, who belongs to poor socio-economic, rural and illiterate family background. Incompletely immunized and underweight children were more susceptible to VAD.*
Conclusion: *Vitamin A deficiency remains widely prevalent in the developing world. India is currently at a stage when universal vitamin A supplementation must immediately transit to a targeted supplementation programme*
Keywords: *BMI, direct ophthalmoscopy, Fluorescein eye staining, Schirmer's test, VAD.*

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I. Introduction

Vitamin A deficiency remains a leading public health problem in the developing world [1], with its health consequences most apparent and severe among infants, young children, and women of reproductive age. Over the past two decades, tremendous advances have been made in developing indicators of vitamin A status, and defining the magnitude of vitamin A deficiency by region, metabolic and health consequences of deficiency that respond to adequate vitamin A intake, and approaches to effective prevention. Scientific knowledge about vitamin A deficiency and its prevention continues to be translated into effective policies and programs [2] that reflect growing resolve across governments, multilateral and nongovernmental agencies, academia, private industry, and in the community to achieve results. Vitamin A is a fat-soluble vitamin ingested in the diet in two forms-as retinol itself from animal sources, such as milk, meat, fish, liver, and eggs, or as provitamin carotene from plant sources such as green leafy vegetables, yellow fruits, and red palm oil.[3,4] Vitamin A deficiency is a major cause of childhood blindness in India and other developing countries.[5,6] Vitamin A deficiency exists as a public health nutrition problem among preschool-aged children in 118 developing countries around the globe, with the South East Asian region harboring the maximum number of cases.[7] The consequences of vitamin A deficiency include all active clinical stages of xerophthalmia, impaired mechanisms of host resistance, increased severity of infection, anemia, poor growth, and mortality. Certain systemic illnesses are associated with vitamin A deficiency, such as measles, pneumonia, diarrhea, malabsorption due to cystic disease, liver disease, pancreatic disease, or inflammatory bowel disease. Various studies have shown that mortality rate can be decreased if Vitamin A is taken prophylactically. Vitamin A is necessary for preserving integrity and maintaining the functions of several organs in the body. This review is an attempt to highlight the ocular manifestations and prevalence of vitamin A deficiency and to discuss the physiological indicators available for its early diagnosis and prevention strategies.

Prevalence of Vitamin A deficiency

The ocular manifestations of vitamin A deficiency are collectively called xerophthalmia. It can affect any age group, but its most severe blinding complications affect children aged 6 months to 3 years. It was estimated that 127 million preschool children under 5 years of age are vitamin A deficient, of whom 4.4 million have xerophthalmia.[7] There are also an estimated 7.2 million pregnant women with Vitamin A deficiency (serum retinol < 0.7 μmol/l) at any one time in the developing world, of whom around 6 million are night blind,

a condition attributed to vitamin A deficiency.[8] Even amongst school-aged children, the prevalence of vitamin A deficiency was found to be 23.4% in a study done in the countries within World Health Organization (WHO) South East Asian region.[9]

Ocular manifestations of Vitamin A deficiency

Vitamin A deficiency affects the retina, conjunctiva, and cornea, and the signs and symptoms tend to occur in a reliable sequence. The WHO classification of vitamin A deficiency is as follows:[10]

Classification of xerophthalmia (ocular signs).

1. Night blindness (XN)
2. Conjunctival xerosis (X1A)
3. Bitot spots (X1B)
4. Corneal xerosis (X2)
5. Corneal ulceration/keratomalacia <⅓ corneal surface (X3A)
6. Corneal ulceration/keratomalacia ≥⅓ corneal surface (X3B)
7. Corneal scar (XS)
8. Xerophthalmic fundus (XF)

Night blindness

Because of the essential role of vitamin A in photoreceptor function, defective dark adaptation is the most characteristic early clinical feature, resulting in night blindness. Night blindness is the earliest clinical symptom and in children it presents as inability to find their way in dim light. Its occurrence reflects a failure of rod cells in the retina to maintain peripheral vision under dim light. Patient cannot read or drive a car in poor light. Electroretinography and dark adaptation can help in diagnosis of retinal function in early stage even if the person is asymptomatic.

Conjunctival manifestations

Xerosis (X1A) is the term used to describe dryness. Vitamin A deficiency leads to a loss of mucus-secreting goblet cells and eventually to squamous cell metaplasia of the conjunctival epithelial cells. Conjunctiva becomes dry, thick, and wrinkled. It gets keratinized, loses its normal transparency, and acquires a smoky appearance. Conjunctival xerosis typically is found on the temporal and interpalpebral bulbar conjunctiva

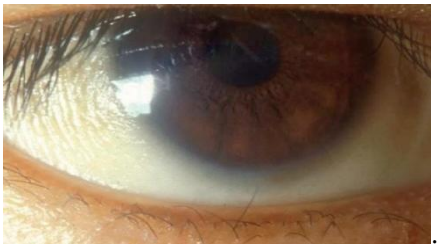

Fig-1: conjuntival xerosis temporal side

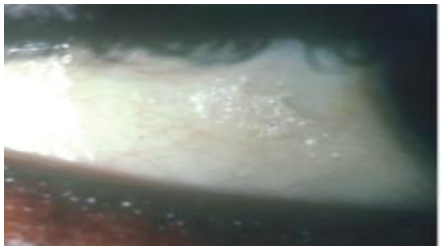

fig-2 :Bitot's spots

Bitot's spots appear as grayish-white triangular plaques, firmly adherent to the conjunctiva due to increased thickness of conjunctiva in certain areas. They stain intensely with Rose Bengal dye. Conjunctival xerosis including Bitot's spots respond favorably to vitamin A and could be a marker to identify those communities with severe and longstanding vitamin A deficiency.[10,11]

Corneal manifestations

The earliest corneal manifestation of xerophthalmia is instability of the precorneal tear film, which can lead to a dull and lusterless corneal appearance. The disorder is present in a majority of people who exhibit night blindness and Bitot's spots. The condition is almost always bilateral. If treatment is initiated at this point, healing usually takes place over the course of perhaps 1 week. If xerophthalmia persists for a long time, it progresses to keratomalacia (a full-thickness liquefactive necrosis of the (cornea). Characteristically, these ulcers are small, partial, or of full thickness, located in the periphery in the early stages. With progression, they can extend to obscure the pupillary axis and develop secondary bacterial infection.[12] The corneal stroma can slough, either leaving a descemetocele (herniation of Descemet's membrane, through a corneal wound or deep ulceration) or, in severe cases, causing perforation and loss of the anterior chamber. Vitamin A supplementation speeds healing. Often, keratomalacia is associated with a preceding systemic stressor, such as measles, diarrhea,

or respiratory infection, or with concurrent severe protein–energy malnutrition.[13] Corneal scar resulting from corneal ulceration due to vitamin A deficiency could potentially lead to blindness. In severe cases, there can be total loss of vision due to formation of anterior staphyloma (weakening and bulging of cornea lined anteriorly by epithelium and posteriorly by iris).

Xerophthalmic fundus

Xerophthalmic fundus is uncommon, but it representsstructural damage to the fundus in the form of white dots in the retinal periphery. Fluorescein angiogram reveals these dots to be focal retinal pigment epithelium defects.[14] Rarely, patients can present with scotomas corresponding to the area of retinal involvement. [15] These changes can respond to vitamin A therapy, with the scotoma disappearing in 1–2 weeks and retinal lesions fading in 1–4 months.[16]

II. Material And Method

A total of 120 Patients who were diagnosed as a case of vit-A deficiency, were included in this cross-sectional study conducted in the Department of Ophthalmology, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh, India over a period of 10 months from Jan. 2018 to Oct. 2019. The procedures followed were in accordance with the ethical standards committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000. The necessary permission from the Ethical and Research Committee was obtained for the study.

Inclusion criteria:

1. Patients with any sign or symptoms of Vit.-A deficiency with or without affected visual acuity.
2. Both male and female patients were included in the study.
3. The age group of the patients to be studied was between 1 month to 14 years

Exclusion criteria:

1. Patients with Vit.-A deficiency already on medications, were not included in the study.
2. Patients above the 14 years of age were excluded from the study.
3. Patients, who had previous history of any ocular surgery, were excluded from the study.
4. Patients with history of corneal ulcer, trauma, and other ocular pathology, were excluded from the study.

An assessment of present complaints, detailed clinical history (present and past), and history of any ocular surgery, Age, sex, occupation, socio-economic status, immunization status, antenatal, intranatal, postnatal history and BMI were recorded. Ophthalmological check up as external examination of the eyes, visual acuity, torch light examination, slit lamp examination, Fluorescein eye staining, Schirmer's test, refraction, direct ophthalmoscopy, was done.

III. Result

A total of 120 Patients which was diagnosed as a case of Vit-A deficiency, were included in this cross-sectional study

Table 3.1: Gender ratio of patients with corneal opacity (n=120)

No. of patients	56	64
Percentage (%)	46.66	53.33

Table 3.2: Age and sex wise distribution of patients (n=120)

Age groups (in years)	No. of patients	Percentage (%)
0-4	21	17.5(%)
5-9	77	64.16(%)
10-14	22	18.34(%)
Total	120	100(%)

Table 3.3: BMI distribution of patients (n=120)

BMI	No. of patients	Percentage (%)
Less than 18.5	65	54.17(%)
18.5 to 24.9	45	3.75(%)
More than 25	10	8.33(%)
Total	120	100(%)

Table 3.4: Immunization status of patients (n=120)

Immunization status (Including MMR vaccine & Vit-A suppl.)	No. of patients	Percentage (%)
Fully immunised	20	16.67(%)
Not completely immunized	100	83.33(%)
Total	120	100(%)

Table 3.5: Family Socioeconomic status of patients (n=120)

Revised Kuppuswamy's Socioeconomic Status	No. of patients	Percentage (%)
Upper class	5	4.16(%)
Upper middle class	12	10(%)
Lower middle class	19	15.84(%)
Upper lower class	24	20(%)
Lower class	60	50(%)
Total	120	100(%)

Table 3.6: Patients presented with xerophthalmic ocular sign andSymptoms

Xerophthalmia by ocular signs.	**No. of patients**	**Percentage (%)**
1. Night blindness (XN)	**11**	**9.16**(%)
2. Conjunctival xerosis (X1A)	**93**	**77.5**(%)
3. Bitot spots (X1B)	**10**	**8.33**(%)
4. Corneal xerosis (X2)	**18**	**15**(%)
5. Corneal ulceration/keratomalacia <⅓ corneal surface (X3A)	**11**	**9.16**(%)
6. Corneal ulceration/keratomalacia ≥⅓ corneal surface (X3B)	**4**	**3.33**(%)
7. Corneal scar (XS)	**4**	**3.33**(%)
8. Xerophthalmic fundus (XF)	**00**	**0 %**

IV. Discussion

In this study, the male female ratio of vit-A deficiency was 1:1.2, which was most commonly observed in children with 5-9 years of age group (64.16%), who were belongs to poor socio-economic (almost 70%), rural and illiterate family background. The most common sign of vit-A deficiency was conjunctival xerosis (77.5%) and most common cause of blindness (economical or social blindness) in vita-A deficiency was corneal scar (3%). In this study it was also observed that underweight (54.17%) and incompletely immunized (83.33%) children were more susceptible to vit-A deficiency. Though the prevalence of severe forms of vitamin A deficiency such as corneal ulcers/ Keratomalacia has in general become very rare, the milder forms such as Bitot spots, conjunctival xerosis and night blindness are prevalent in varying magnitudes in different region of the country.

V. Conclusion

Vitamin A deficiency can be controlled as a public health problem by maintaining adequate intakes of the nutrient in high-risk groups through direct supplementation, fortification, agronomic programs, marketing, and educational efforts to improve diet. The prevalence of severe vitamin A deficiency is Widespread in children aged 6–9 years in rural areas. vitamin A deficiency in younger children to a level not considered to be of public health significance are encouraging but a challenging task remains in countries where the number of children affected is larger than previously expected. Nutrition education, social marketing, and other food-based approaches can be equally developed from epidemiologic evidence. Guiding mothers to breast-feed infants through the third year of life rests on a consistent association of protection against xerophthalmia. Vitamin A deficiency remains widely prevalent in the developing world. India is currently at a stage when universal vitamin A supplementation must immediately transit to a targeted supplementation programme.

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