IJMPR 2019, 3(2), 09-12

International Journal of Modern Pharmaceutical Research
www.ijmpronline.com

ISSN: 2319-5878
IJMPR
Research Article

SJIF Impact Factor: 3.458

PREVALENCE OF VITAMIN A DEFICIENCY DISORDERS IN CHILDREN

Dr. Muhammad Umar Farooq* and Mehwish

[1]Aziz Fatima Medical and Dental College Faisalabad, Pakistan.
[2]Institute of Molecular Biology and Biotechnology Bahuddin Zakariya University Multan.

Received on: 28/01/2019
Revised on: 18/02/2019
Accepted on: 08//03/2019

***Corresponding Author**
Dr. Muhammad Umar Farooq
Aziz Fatima Medical and Dental College Faisalabad, Pakistan.

ABSTRACT

Almost 4.4 million children of preschool Age have xerophthalmia throughout the world. Due to these reasons it is now crucial to prevent its deficiency. Deficiency of vitamin A is common problem worldwide causing a number of disorders like there to prevent the lack of vitamin A highlighting the proper nutritional diet, direct vitamin A supplementation, supplementation of preformed retinol and dietary counselling.

KEYWORDS: Disorders, Vitamin A, Deficiency, Anemia, Xerophthalmia, Nutrition.

INTRODUCTION

Vitamin A deficiency continues to be a major common health problem in the underdeveloped world (Sommer et al. 1996) with its health effect most visible and severe among young children, infants and women of generative age. From the past 20 years, enormous advances have been devised for developing symptoms of vitamin A position, and explaining the magnitude of its deficiency by region, health and metabolic consequences of the deficiency that further leads to proper intake of vitamin A, and access to effective prevention (Sommer &Davidson 2002) Effective translation of inquiry into action and policy requires an uninterrupted procedure of "taking stock," by modernizing estimates of the extent in populations under high level of uncertainty, clarifying disease burden linked with the deficiency of vitamin A, exploring much better prevention through the implementation of epidemiologic advances, and assuring progression in the field. With that aspect, the curious reader is subjected to comprehensive, recent supplements that have manifested on the deficiency of vitamin A and its control. This paper is about the current extent of deficiency of vitamin A and its order of different health risks in the young children, as well as highlighting the continued, critical significance of restraining this micronutrient shortage in disadvantaged populations.

Epidemiology of the vitamin A deficiency

Estimates of magnitude and acuteness of its deficiency are flawed, as they rely upon the frequency of using reliable indicators of status of vitamin A throughout the populations, and taking ideas from various origins of data from diverse representativeness. By the studies dependent upon xerophthalmia in children, highly precise data can be obtained underlining the deficiency of vitamin A (mild to severe) in chronically defective regions (Sommer &Davidson 2002). Dark adaptometry offers a recently standardized, logical access to quantify the commonness of moderate scotopic vision loss assignable to the deficiency of vitamin A (Sommer et al. 1983) but has been inadequately utilized to date for approximating the burden of deficiency. Use of stable isotope allows validation of other symptoms of the status and quantitative analysis of total body storages of vitamin (Haskell et al., 2003) while the comparative dose response continues to be a beneficial research tool for evaluating hepatic retinol adequacy in a roundabout manner(Solon et al. 1978). However, by these means, evaluation of liver or entire body storage of vitamin A remains troublesome, expensive, and are not commonly used in studies pertaining to large populations. For estimating population prevalence of this deficiency, CIC (Conjunctival impression cytology) has been widely used, specifically when serologic data may not be collected. These types of recent measures of the deficiency status have been practiced by WHO (World health organization) (Organization, 1995) (West Jr, 2002), IVACG (The International Vitamin A Consultative Group) (West Jr, 2002) and MI (The micronutrient initiative) (Initiative, 1998) (Oomen 1974) to evaluate or display alteration in the burden of this vitamin deficiency in children. About 50% of the children of the world having xerophthalmia dwell in Southeast and south Asia, of which more than 85% reside in India (Organization 1982). These calculations are able to be compared in extent to those of 10 years ago, in spite of remarkable population growth throughout the interim, which apparently reflects degrees of achievements in controlling vitamin A deficiency (West

Jr and Sommer, 1985). Countries like Bangladesh and Nepal have lower levels of xerophthalmia accompanied by biochemical/persistent vitamin A deficiency, perhaps reflecting known high effectiveness of the distribution of vitamin A capsule for the purpose of preventing xerophthalmia (≈ 90%).

MATERIALS AND METHODS

Table 1: Questionnaire to evaluate awareness about etiology of Vitamin A deficiency.

Vitamin A deficiency is a	Yes	No
1. Viral disease		
2. Bacterial disease		
3. Fungal disease		
4. Genetic disease		
5. Metabolic disease		
Ever suffered from		
6. You		
7. Your family		
8. Your relative		
9. Your neighbor		
10. Your friend		
Vitamin A deficiency is transmitted by		
11. Contacts or blood transfusion		
12. From parents to offspring		
May be treated by		
13. Medicines		
14. Surgery		
15. Do not worry, it is easily curable		

Table 1: Questionnaire to evaluate awareness about etiology of Vitamin A deficiency.

Is a	Yes	No
1. Viral disease		
2. Bacterial disease		
3. Fungal disease		
4. Genetic disease		
5. Metabolic disease		

Table 2: Questionnaire to evaluate views about prevalence of Vitamin A deficiency.

Ever suffered from	Yes	No
1. You		
2. Your family		
3. Your relative		
4. Your neighbor		
5. Your friend		

3: Questionnaire to evaluate views about transmission of Vitamin A deficiency.

Is transmitted by	Yes	No
1. Contacts Table or blood transfusion		

Table 4: Questionnaire to evaluate views about Hope for Vitamin A deficiency.

may be treated by	Yes	No
1. Medicines		
2. Surgery		
3. Do not worry, it is easily curable		

RESULTS

Table 5: Awareness about etiology of Vitamin A deficiency: Views of Postgraduate Biology Students.

Questions	Male		Female		Total	
	Yes	No	Yes	No	Yes	No
1. Viral disease	0%	100%	0%	100%	0%	100%
2. Bacterial disease	0%	100%	0%	100%	0%	100%
3. Fungal disease	0%	100%	0%	100%	0%	100%
4. Genetic disease	10%	90%	20%	80%	20%	80%
5. Metabolic disease	0%	100%	33.3%	67%	28%	71%

DISCUSSION

Common Disorders caused by Vitamin A deficiency

Over all, health consequences referable to the deficiency of vitamin A are termed as vitamin A deficiency disorders and abbreviated as VADD. These conditions vary from ocular demonstrations of xerophthalmia, comprising its blinding consequence, to less approximate disorders of impaired processes of host resistance, poor growth, mortality referable to the deficiency of vitamin

A and acute infectious illnesses, in a population (West Jr and Sommer, 1985) Because of their specificity, the levels of xerophthalmia are comprised of both of the indicator variables and VADD for evaluating prevalence and acuteness of deficiency in a specific(Organization 1982). Long-term improper dietary intake affects the status of vitamin A and is a powerful determinant causal factor of VADD (Sommeret al. 1996) (Fawzi et al., 1995). The comprehensive, but plausible, definition of VADD offers a framework for enhancing status assessment, better knowhow about the deficiency, health burden related to the deficiency and figuring out public health advantages of the prevention.

Vitamin A deficiency disorders in preschool children:
Preschool children are vulnerable to the deficiency of vitamin A and its health outcome (xerophthalmia, poor growth, infection, anemia) because of the high nutrient requirement to support growth, usual exposure to the disease and nutritionally demanding sickness, and constant lack of proper nutrition and health care in the poor areas.(Petersen *et al.*1968).

Xerophthalmia
Xerophthalmia continues to be the major known reason of blindness in teenage children that can be prevented (West Jr, 2015). As a result of acute vitamin A deficiency, ulceration, necrosis (tissue death) and Corneal xerosis have been observed often when there is inadequate nutrition usually termed as malnutrition (Foster and Sommer, 1987) (BLOEM *et al.* 1989). Corneal xerophthalmia is still rare, because of the integrated effects of low occurrence and high case lethality, and seems to be in a downslope (Solon *et al.* 1979) Moderate, non-blinding xerophthalmic levels include night blindness, pertaining to impaired dark adaptation because of vitamin A shortage in Bitot's spots and in the photoreceptors of rod cells come into being from keratinizing tissue alteration of the conjunctival layer of bulb. (Humphrey *et al.* 1992).

Infectious diseases
Vitamin A deficiency adds to the vulnerability to infection, which, successively, may further exacerbate the deficient state, disclosing a classical "synergism" nearly forty years ago that our maximum understanding of mechanisms goes on to support (Van Horn et al. 1980) Children with moderate xerophthalmia (Bitot's spots or night blindness) show cell-mediated immunity (Semba et al., 1993) and impaired antibody (Semba et al., 1992) raised levels of severe phase plasma proteins (Semba et al., 2000) (Semba et al., 2000) that tend to suggest infection, poor growth (West Jr et al., 1997) (Sommer et al., 1984) in addition to much higher risks of diarrhea (Khatry et al., 1995), mortality (Van Hornet al. 1980) and respiratory diseases. Contrary to this, children with low respiratory infection and diarrhea living in regions having vitamin A deficiency endemically are more probable to go through xerophthalmia (Sommer et al., 1987). Supplementation of vitamin A has been revealed to minimize the acuteness of malaria, measles and diarrhea. Supplementation has also been revealed to minimize the acuteness of measles complications and remarkably lower case lethality, by ≈ 50% as demonstrated by the clinical trials (Van Hornet al. 1980). Due to the fact that not all reports have shown alike effects, extra studies are required to verify the prophylactic measure of vitamin A in treating falciparum malaria. Shigella dysentery and diarrhea are linked with increased losses of retinol pertaining to urine, which may intensify the infection (Stephensen, 2001) (Mitra et al., 1998). Supplementation of vitamin A has been revealed to reduce the acuteness of dysentery and diarrhea in ill-fed populations (Barreto et al., 1994) presumably by improving epithelial repair and role in the intestine while serving to preserve body storages of vitamin A. In some of the trials, marks and indications of intensified respiratory disease have resulted from vitamin A supplementation, perhaps reflecting improved immune-mediated airway action, transient toxicity or inflammation (Dibley et al., 1996) (Latham et al., 2003). Harmful effects of supplying vitamin A, when summarized, tend to occur unexpectedly more frequently in properly nurtured groups of children and infants, since increase in prophylactic and therapeutic benefits in poorly nurtured or infected individuals or populations is obvious (West Jr et al., 1995). Mechanisms to properly elaborate these interactions are currently lacking.

Anemia
In the late 19th century, a relation between night blindness and anemia was identified (Semba and Bloem, 2002). From that time, children having xerophthalmia have been observed suffering from anemia, and experimentally, animals showing depleted levels of vitamin A have been discovered to lesser hematopoietic capacity. According to the reports from childhood surveys in ill-fed populations of Africa, South Asia and America, there are obvious correlations (r) of approximately 0.20–0.50 between plasma retinol and hemoglobin concentrations. Perhaps, Anemia of the deficiency of vitamin A ascribed to several conceivable processes which may respond to much better vitamin A nourishment, comprising impaired mobilization and transfer of body iron, interrupted erythropoietin synthesis and imperfect formation of blood cells in the bone marrow (Fishman et al., 2000).

Poor growth
For mammalian growth, vitamin A is necessary (De Sole et al. 1987). Children having moderate xerophthalmia are stunted in many cases and may display some extent of wasting. The more acute the deficiency or disease, the more the possibility that supplementation will enhance either ponderal or linear growth. Outcomes, where present, still may be seasonal (Sommer &Davidson 2002) sex or age-specific (Villamor et al., 2002) (Donnen et al., 1998) and perhaps controlled by disease factors, like intestinal worm burden and duration of respiratory disease (Hadi et al., 1999) which causes the

growth response troublesome to foretell as a common health advantage for controlling vitamin A deficiency.

Preventive measures for vitamin A deficiency

As Vitamin A deficiency is questioning public health, it must be prevented. For this purpose, there must be some maintenance of sufficient intake of the proper nutritional diet in groups at higher risks through, fortification, direct supplementation, marketing, agronomic programs, and educational struggles to improve diet. In recent years, by conducting well-organized campaigns every 6 months, higher supply of vitamin A has been achieved. Polio vaccination by the NIDS (National Immunization Days) have provided chances to encase preschoolers one time every year (Goodman et al., 2000). Social marketing, diet education and other nutrition-based approaches may be originated from epidemiologic evidence to the same degree (De Soleet al. 1987). A diet that evenly provides provitamin A carotenoids or vitamin A which has been created in advance is strongly linked with protection from the disorders caused by the deficiency of vitamin A among children. Thus, dietary counseling must be there (West et al., 2002). In recent times, plant foods, specifically dark green leaves have been observed to produce provitamin A carotenoid which further can be converted to vitamin A (Copper, 2001). Development of inexpensive packaging may rise to be necessary for prosperous fortification of vitamin A' products in markets having minimal qualities.

CONCLUSION

Vitamin A deficiency continues to be widespread in the under-developed world. In children, it is followed by different disorders including xerophthalmia having potential to cause blindness; damaged host resistance to disease with consequent higher risks of acute measles, diarrhea, malaria, and other illnesses pertaining to fever and poor growth in the children. These disorders may have a remarkable effect on the survival and health of the child. Thus, it suggests a need to improve effective preventive approaches in high-risk individuals thereby broadening the numbers and proportion requiring vitamin A in ill-fed societies. Nutritional policies developed must be evidence-based, sufficient in coverage and marketed to the populations at higher risks.

REFERENCES

1. BLOEM MWWedel MEgger RJSpeek AJChusilp KSaowakontha S,Schreurs W A prevalence study of vitamin a deficiency and xerophthalmia in northeastern thailand, American journal of epidemiology, 1989; 129: 1095-1103.
2. De Sole GBelay Y,Zegeye B Vitamin a deficiency in southern ethiopia, The American journal of clinical nutrition, 1987; 45: 780-784.
3. Humphrey JWest Jr K,Sommer A Vitamin a deficiency and attributable mortality among under-5-year-olds, Bulletin of the World Health Organization, 1992; 70: 225.
4. Oomen H Vitamin a deficiency, xerophthalmia and blindness, Nutrition reviews, 1974; 32: 161-6.
5. Petersen RAPetersen VS,Robb RM Vitamin a deficiency with xerophthalmia and night blindness in cystic fibrosis, American Journal of Diseases of Children, 1968; 116: 662-665.
6. Solon FFernandez TLLatham MC,Popkin BM An evaluation of strategies to control vitamin a deficiency in the philippines, The American journal of clinical nutrition, 1979; 32: 1445-1453.
7. Solon FSPopkin BMFernandez T,Latham MC Vitamin a deficiency in the philippines: A study of xerophthalmia in cebu, The American journal of clinical nutrition, 1978; 31: 360-368.
8. Sommer AHussaini GTarwotjo I,Susanto D Increased mortality in children with mild vitamin a deficiency, The Lancet, 1983; 322: 585-588.
9. Sommer A,Davidson FR Assessment and control of vitamin a deficiency: The annecy accords, The Journal of nutrition, 2002; 132: 2845S-2850S.
10. Van Horn DLSchutten WHHyndiuk RA,Kurz P Xerophthalmia in vitamin a-deficient rabbits. Clinical and ultrastructural alterations in the cornea, Investigative ophthalmology & visual science, 1980; 19: 1067-1079.