VITAMIN A REQUIREMENT FOR GENERAL HEALTH & DENTITION: A Review

Beda Nidhi Dahal, Samiksha Chapagain
Integrated Bio-scientific Research Group (BIO-INT)

Dr. Rabindra Man Shrestha
VSOG Dental Clinic, Kathmandu Model Hospital
Public Health Concern Trust (phect-NEPAL)
P.O.Box: 6064, Kathmandu

INTRODUCTION:

Vitamin A is an essential nutrient and its rote in general health and in preventing nutritional defective teeth is well established (Swaminathan 1976). Every year 8 to 10 million children suffer vitamin A deficiency (VAD) in the world (Simon 1990). Nearly 50 percent of the total population and up to 46 percent of pregnant woman are reported to be suffering from VAD in Nepal (OMNI 1996). Moreover, 25,000 children die every year due to VAD in Nepal (OMNI 1998), so VAD is a significant problem in Nepal.

Vitamin A deficiency impairs the structure and functions of epithelial membranes and hence lowers the resistance to infection (Swaminathan 1976). Vitamin A is also necessary for the growth and development of soft tissues through its effect on protein synthesis and differentiation of the teeth cells, so it is required for proper formation and maintenance of the teeth enamel, dentin and healthy gums (manay et al 1987). VAD interferes with the form ation of enamel, hence the teeth formed will be defective and are more likely to contain pits and fissures which predispose the teeth to decay (Swaminathan 1976).

The VAD is primarily concerned with the process of cell differentiation where the epithelial cells fail to differentiate, hence the basal cell layer lose their specificity and tend to from a stratified squamous epithelium with keratin production. The basic change in the body is the keratinizing metaplasia of epithelial cells in mucous membranes of trachea, conjunctiva, ureter and salivary and other glands. In a developing vit-A-deficient tooth, the odontogenic epithelium fails to undergo normal histo-differentiation and morpho-differentiation, and results in increased cell proliferation. In such a case, epithelial invasion of pulpal tissue is characteristic in Vitamin A deficiency (Wolback & Howe).

Current research indicates that Vitamin-A have hormonal function in the regulation of epithelial differentiation. Intracellular receptors have been identified and may transport Vitamin-A molecules to the cell nucleus, where they interact with DNA to direct cellular differentiation (Shafer 1993).

MANIFESTATIONS OF VAD:

Many researchers have suggested the dental anomalies due to VAD predispose the teeth for dental caries. Besides, the eruption rate is retarded, and in prolonged deficiencies eruption ceases completely. The alveolar bone is retarded in its rate of formation and gingival epithelium becomes hyperplastic and in prolonged deficiencies show keratinization, such tissue is early invaded by bacteria which may cause periodontal problems (Shafer 1993).

The major and minor salivary glands undergo typical keratinizing metaplasia (Shafer1993), which causes sicca syndrome or keratoconjunctivitis sicca leading to xerostomia. The clinical features are dryness of mouth with retention of salivary gland, pain in the region and abscess formation in the gland.

VAD has also been associated as the etiological factor for the manifestations of leukoplakia (Shafer 1993), oral cancers (Wahi, Cebar & Lahiri 1965) and cleft lip & cleft palate (Shafer 1993).

The prominent manifestations of VAD in man are night blindness, xerophthalmia and keratomalacia. Vitamin A is necessary for maintaining the eye in healthy conditions and for proper vision in dim light (Swaminathan 1976).

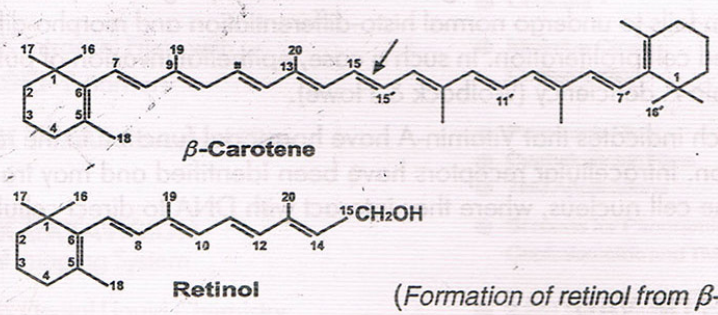
In human infant, keratinizing metaplacia appears in the trachea, bronchi, kidney, pelvis, conjunctiva, cornea, salivary glands and genito-urinary tract. (Wilson & DuBois, Blackfan & Wolbach). Besides, the physical growth in impaired, the skin becomes rough & dry and there is diminished resistance to infections (Mital 1979).

Causes of VAD:

1. Poor intake (dietary lacks due to poverty & ignorance),
2. Malabsorption (steatorrhea & obstructive jaundice),
3. Hypothyroidism,
4. Severe chronic liver disease (cirrhosis of liver)

Chemistry of Vitamin A:

Vitamin A is a fat soluble vitamin and is chemically an alcohol($C_{20}H_{29}OH$), called retinol and metabolically active forms of Vitamin A include the corresponding aldehyde (retinol) and the acid (retinoic acid) (Manay et al 1987). Plants do not contain Vitamin A, but contain its precursors or provitamin A, the carotenoids (α -, β-, χ- carotenes) are C-40 unsaturated hydrocarbons and β-carotene splits into half to give two molecules of Vitamin A. (Manay et al 1987, Rodriguez-Amaya, 1997).



(Formation of retinol from β-carotene)

Review of the Literature:

Vitamin A & D deficiency with protein energy malnutrition have been associated with dental abnormalities in malnourished populations. It has been observed that children in underprivileged groups have a high prevalence of dental dysplasias and that have been

associated with inadequate nutrition during tooth development. Baume & Meyer in 1966, described two types of dental dysplasias; Odontoclasia in deciduous dentition and a "yellow teeth" condition seen in permanent teeth. Both of the defects were highly susceptible to rampant tooth decay. They further described a third dysplastic condition referred to as "infantile melanodontia", which was seen as mottled enamel in children living on distant islands, however these conditions were caries resistant. Baume made an extensive study of these epidemiological findings and suggested that the enamel alterations were related to malnutrition experienced in early life especially within 6 years of age, when the teeth were undergoing mineralization (Shafer 1993).

The nutrients like proteins, Vitamin A and minerals and involved in the process of calcification. Vitamin A (Navia & Harris, 1985) & Mn affect in glycosaminoglycan metabolism. They are essential for thc formation and maintenance of calcified tissues like bones & teeth. Severe and even mild deficiencies of these nutrients during the mineralization of teeth may cause the deposition of defective during the mineralization of teeth may cause the deposition of defective enamel & dentin. In contrast to bone, it has only single opportunity to be formed normally. Hence the defects during development con not be cellularly repaired and constitute irreversible lesions in the teeth. Vitamin A deficient diet has been considered as highly influential in caries activity and can act preeruptively (Harris & Navia) and posteruptively it enhances the implantation and metabolic activity of cariogenic microorganisms. The posteruptive influence of diet can predispose the teeth for rampant caries with contributory preeruptive cariogenic effect. In a study, in which rat pups were made Vitamin A deficient, specially during critical periods of tooth development, it was concluded that there was and increased caries susceptibility of rat molars in experimental rats compared to that of control group (Harris & Navia, 1980).

The VAD results in keratinizing metaplasia of the epithelium. In a study with rats with VAD, hyperplasia and hyperkeratinization of the gingival epithelium with proliferation of the gingival epithelium was observed (Boyle, Bessey 1941) with retardation of gingival wound healing (Frandsen 1963). In the presence of local irritants, Vitamin A deficient rats develop periodontal pockets (Boyle 1947) that are deeper than those in non-VitA-deficient rats and exhibit associated epithelial hyperkeratosis (Glickman, Stoller 1948). Deficiency of Vitamin A also interferes with the production of lysozyme which is an antibacterial agent in saliva for the control of oral microbial flora.

VAD has been associated with cleft palate, which has been experimentally produced in newborn rats by feeding diets deficient in Vitamin A to maternal rats during pregnancy (Shafer 1993). The author suggested that the VAD could be a predisposing factor for the multifactorial developmental defect like cleft lip & cleft palate.

VAD has also been associated with salivary gland disorder like keratoconjunctivitis sicca or sicca syndrome leading to xerostomia. Wolback reported VAD in human leading to squamous metaplasia of the salivary gland duct epithelium with retention of salivary secretion as well as inflammation with abscess formation. Similar conditions have also been shown in animals with experimental induction of avitaminosis-A.

Low serum Vitamin A was found in 76.2% of patients with oral carcinomas and was considered an adjuvant in the cariogenic process (Wahi, Kebar & Lahiri 1965). A diet of rice

and tapioca, extremely deficient in protein and vitamin A, might predispose to oral cancer. This condition was found among the Indian coolies working in Travancore where oral cancer rates are extremely high (Orr 1933). It has also been suggested that the deficiency of Vitamin A is related to the development of leukoplakia (Shafer 1993).

Requirement of Vitamin A:

The daily intake of Vitamin A as recommended by the FAO is 250-400 retinol equivalents (RE) for children, 575-725 RE for adolescents, 750 RE for adults with additional 200-300 RE for pregnant and lactating women (FAO 1982).

The recommended dietary allowances (RDA) of Vitamin A (revised 1980) by the Food & Nutrition Board, National Academy of Sciences & National Research Council of US for the different age and sex groups are as follows.

	Age (years)	Weight (kg)	Vitamin A (μgRE)*
Infants	0.0-0.5	6	420
	0. 5-1.0	9	400
Children	1-3	13	400
	4-6	20	500
	7-10	28	700
Males	11-14	45	1000
	15-18	66	1000
	19-22	70	1000
	23-50	70	1000
	51+	70	1000
Females	11-14	46	800
	15-18	55	800
	19-22	55	800
	23-50	55	800
	51+	55	800
Pregnant			+200
Lactating			+400

(*Retinol Equivalents: I retinol equivalent = 1 μg retinol or 6μg β – carotene)

Discussion:

The dire situation of the Nepalese population is large part due to low Vitamin A intake, because of insufficient awareness, lack of knowledge about the importance of Vitamin A, food habits and cultural traditions that constrain the consumption of balanced diet, insufficient production and utilization of food items containing vitamin A, and insufficient public intervention to ensure good nutrition.

Due to these reasons, most of the population in Nepal is suffering from VAD during critical periods of tooth development, there was found to be increased caries susceptibility and it has been associated with dental anomalies in malnourished population. The deficiency is also associated with periodontal problems and oral cancers. Besides salivary

gland disorders, skin and mucosal problems and problems related to eye like night blindness are also considered to be due to VAD. Therefore, most of the patients who are suffering from such diseases might be caused of vitamin A malnutrition, hence this vitamin is essential for general health especially for dental health. However per capita consumption of vitamin A is still a topic of research in Nepal that has not been done so far.

Recommendations:

To fulfil the requirements of Vitamin A, food like carrot, pumpkin, mango, papaya, sweet potato, squash etc from plant origin and fish, meat, egg, butter, milk from animal origin should be consumed balancedly (Swaminathan 1976, Rodriguez- Amaya 1997 and Simon 1988). Most of these vitamin sources are available in hill and plain terai regions of Nepal, but not balancedly consumed by the VAD sufferers, which is a major cause of VAD in Nepal.

Further, high carotene carrot containing 30,000 micrograms of carotene per 100 gram has been developed and tested in Nepal (Simon 1988). Only 30 grams of raw carrot (3 grams dried) can provide a sufficient vitamin A to meet the daily requirement for an adult or a child (Simon 1988).

Retinol content of selected foods (in retinol equivalents,RE (mcg/100g)):

Halibut liver oil	900,000	Carrot	1167
Cod liver oil	18,000	Spinach	607
Liver	16,500	Amaranth	515
Butter	825	Green leave	300
Margarine	900	Mango,ripe	313
Cheese	350	Papaya	118
Egg	140	Orange	25
Cow milk	38	Tomato	84
Fish	40		

Distribution of Vitamin A:

Ministry of Health and many non-governmental organizations (NGO's) have supported extensive distribution of high dose vitamin A capsule (20,000 IU) donated by OMNI to Nepalese children in target areas. This effort has been helpful in reduction of VAD, however it is not a long term sustainable solution for developing country like Nepal. Therefore, the problem should be solved initiating the production and balancedly utilizing and consuming locally available cheaper resources, which are rich in Vitamin A or provitamin A like green leafy vegetables, yellow & red fruits and vegetables, meat, fish, egg, butter and milk (Manay et al 1987, Swaminathan 1976, Stallard 1982, Rodriguez-Amaya 1997).

Conclusion:

In conclusion, dental caries, dental anomalies, periodontal problems and oral cancers due to VAD can be solved to an extent by proper utilization of locally available vitamin A sources in Nepal. For this purpose, mass education & awareness as well as production and utilization in people is essential, whereby VAD dental problems would be reduced in a sustainable way in Nepal.

Reference:

- FAO, 1982: Management of group feeding programs. FAO, UN, Rome. FAO Food & Nutrition paper 23.
- Rodriguez – Amaya D.B; 1997: Carotenoids & Food Preperation : The Retention of provitamine A carotenoids in prepared, processed & stored food. USAID/JSI/OMNI
- Simon, RW, 1988: Genetic improvement of carrots for meeting human nutritional needs. In Proc. 1st Intl. Symp. Horticulture & Human Health. B. Quebedeaux and F.A. Bliss, eds, Prentice Hall Pub. Co., Englewood Cliffs, NJ.
- Simon, P.W., 1990: Carrots & other horticultural crops as a source of provitamin A corotenes. Hortsci. 25:1495-1499.
- Manay, N.S. & Shadakshara swamy, M. 1987: Foods, Facts & Principles, publiched by Wiley Eastern Ltd., India.
- Swaminathan M & Bhagavan R.K, 1976: Our food, published by Ganesh & Co., Madras-17, India.
- OMNI (opportunities for Micronutrient Interventions), 1996: Micronutrient facts, Nepal country series, published by USAID & JSI.
- OMNI, 1998: The OMNI Experience, A USAID funded project ,Managed by John Snow Inc., Final Repot 1993-1998,
- Stallard, R.E, 1982: A Texbook of preventve dentstry, 2nd Ed, W.B. Saunders co
- Shafer, 1993: A textbook of oral pathology, 4th Edn., WB. Saunders Co.
- Carranza, Newman, 1996: Clinical Periodontology, 8th Edn., WB. Saunders Co.
- Macphee & Cowley, 1981: Essentials of periodontology & Periodontics, 3rd Edn., Blackwell Scientific publications.
- Datay, Shah, 1979: API Textbook of Medicine, 3rd Edn, API.
- Park. K., 1997: Park's Textbook of Preventive & Social Medicine, 15th Edn., B.B.Publishers, India
- Edwards, Bouchier,1994: Davidson's Principles & Practice of Medicine, 16th Edn, ELBS Churchill Livingstone, UK

❑