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ROLE OF VITAMINS IN EYE HEALTH AND THEIR DEFICIENCES

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ABSTRACT

Indriyas are the organs which are meant to perceive the sense of object. They are originated with the predominancy of their respective five mahabhutas. The eyes are said to be most important than all other Indriyas. They are considered as the reflectors of the mind. It is unique organ in the body where the course as well as patho-physiology of disease process is visible, effect of therapeutic agents can very well be assessed and if needed documented. Every person should try to protect his eye always throughout his life, because the world is useless for the day is as night for persons who are blind, though they might possess plenty of wealth. Nutrition plays a big role in vision health and its function depends on the nutrients from foods. Most people are aware of role of vitamins and nutrition can play in preventing health problems like cancer and heart diseases. But not has many people are aware that most chronic eye diseases are also related to foods we eat. Our eyes benefit from good nutrition in the same way other parts of our body .Vitamins are organic compounds which are not synthesized in the body hence have to be obtained from food. Deficiency of certain eye vitamins may cause eye disorders and impair vision.

KEYWORD: Vitamin and eye diseases.

INTRODUCTION

VITAMINS are essential constituents present in minute amounts in natural foods. If these constituents are removed or deficient such foods are unable to support nutrition and symptoms of deficiency or actual disease develop. Although vitamins are unconnected with energy and protein supplies, yet they are necessary for complete normal metabolism. They are, however, not invariably present in the diet under all circumstances. Childhood and periods of growth, heavy work, childbirth, and lactation all demand the supply of more vitamins, and under these conditions signs of deficiency may be present, although the average intake is not altered. The criteria for the fairly accurate diagnosis of vitamin deficiencies are evidence of deficiency, presence of signs and symptoms, and improvement supplying the deficient vitamin. Vitamin deficiency is usually multiple and may at times be associated with general sub-nutrition and an unbalanced diet. It is customary to consider each vitamin with its specific manifestations separately, although there is really no sharp demarcation between one vitamin deficiency and the other. Xerophthalmia, for instance, is generally associated with rickets in cold climates.

Role of vitamin A in regulation of physiologicalfunctions of eye

The retina is located at the back of the eye. When light passes through the lens, it is sensed by the retina and converted to a nerve impulse for interpretation by the brain. Retinol is transported to the retina via the circulation and accumulates in retinal pigment epithelial cells. Here, retinol is esterified to form a retinylester, which can be stored. When needed, retinyl esters are broken apart (hydrolyzed) and isomerized to form 11-cisretinol, which can be oxidized to form 11-cis-retinal. The latter can be shuttled across the interphotoreceptor matrix to the rod cell where it binds to a protein called opsin to form the visual pigment, rhodopsin (also known as visual purple). Rod cells with rhodopsin can detect very small amounts of light, making them important for night vision. Besides the biological formation of 11-cis retino, much attention has been given to the interaction ofmetarhodopsin II with a G-protein of the rod outer segment, termed transducin. This interaction sets a sequence of amplifying events leading to an electrical signal at the membrane of the rod outer segment. Absorption of a photon of light catalyzes the isomerization of 11-cis-retinal to all-trans-retinal and results in itsrelease. This isomerization triggers a cascade of events, leading to the generation of an electrical signal to the optic nerve. The nerve impulse generated by the optic nerve is conveyed to the brain where it can be interpreted as vision. Once released, all-trans retinal is converted to all-transretinol, which can be transported across the inter photoreceptor matrix to the retinal epithelial cell, thereby completing the visual cycle (12).

Inadequate retinol available to the retina results in impaired dark adaptation, known as "night blindness."

Vitamin A deficiency and vision
Since the unique function of retinyl group is the lightabsorption in retinylidene protein, one of the earliest andspecific manifestations of vitamin A deficiency isimpaired vision, particularly in reduced light (night blindness). Vitamin A deficiency among children indeveloping nations is the leading preventable cause of blindness. Persistent deficiency gives rise to a series ofchanges, the most devastating of which occur in the eyes.Some other ocular changes are referred to asxerophthalmia. First there is dryness of the conjunctiva(xerosis) as the normal lacrimal and mucus secretingepithelium is replaced by a keratinized epithelium. This isfollowed by the build-up of keratin debris in small opaqueplaques (Bitot's spots) and, eventually, erosion of theroughened corneal surface with softening and destructionof the cornea (keratomalacia) and total blindness.

B Complex vitamins: These are responsible for a variety of metabolic and physiological functions.Deficiency can leads to changes in vision, changes in IOP of eye and development of eye factors.

Vitamin B12 (also known as cobalamin) is an essential vitamin for neurological function.Vitamin B12 deficiency optic neuropathy is a rare complication of this deficiency that resultsin progressive, bilateral, painless vision loss that is often associated with reduced color visionand central or cecocentralscotomas.

Vitamin B12 is one of eight components of the vitamin B complex. Itplays a key role in DNA synthesis, maintaining normal brain function, protein metabolism and erythropoiesis. The best sources of vitamin B12 are eggs, milk, cheese, meat, fish, shellfish and poultry. Therecommended daily requirement is 6-9 mcg. Most individuals indeveloped countries consume this amount. The liver can store approximately 500 times the recommended daily allowance; therefore, vitamin B12 deficiency due to dietary insufficiency alone is quite rare.

Acquired conditions, such as pernicious anemia, also cause inadequate absorption of B12. Pernicious anemia is a condition in which the body does not produce enough red blood cells due to decreased intrinsic factor, a glycoprotein secreted by the stomach. Intrinsic factor is commonly reduced in atrophic gastritis, autoimmune diseases, and secondary to gastric surgery.

Pathophysiology of B12 deficiency
Vitamin B12 has several vital roles in the body. It contributes to the formation of methionine, an important part of DNA synthesis in cells that undergo rapid turnover, including those of the hematopoietic system and of the enteric lining that produces intrinsic factor. A deficiencyin vitamin B12 can lead to megaloblasticerythropoiesis, a form of anemia that ischaracterized by large red blood cells that fail to divide via mitosis. Additionally, vitamin B12plays a crucial role in limiting the amount of plasma homocysteine, which in high amountscan lead to endothelial cell toxicity. Vitamin B12 deficiency also leads to elevated levels ofmethylmalonylCoA, which interferes with fatty acid synthesis and contributes to abnormalmyelin formation.Of particular relevance to this case report, vitamin B12 acts as a cofactor in the formation ofsuccinylCoA, an integral part of the Krebs cycle that ultimately produces adenosine triphosphate (ATP). This impaired oxidative metabolism causes a depletion of ATP. Due tothe high metabolic demand of the papillomacular bundle in the retina, it is believed that thisdepletion of ATP damages these sensitive papillomacular bundle fibers resulting in thecommonly noted bitemporal optic nerve atrophy and cecocentralscotoma seen in B12deficiency optic neuropathy.

Optic neuropathy secondary to vitamin B12 deficiency occurs in less than 1% of B12 deficient patients. It results in progressive, bilateral, painless vision loss that is often associated with reduced color vision and central or cecocentralscotomas. The optic nerve may appear normal in the early stages of disease until optic atrophy develops. In approximately 30% of cases, the visual deficiencies precede other neurological and hematological signs often due to the presence of folic acid. Optic nerve head involvement (in the form of optic atrophy) is rare, but can lead to significant visual decline.

Vitamin B1 - (thiamine) Deficiency of Thiamine can cause corneal and an aesthesia, conjunctival and corneal dystrophy and acute retrobulbar neuritis.

Vitamin B2 - (RIBOFLAVIN) Deficiency of vitamin b2 can produce burning sensation in eyes due to conjunctivalirritation,mistyvision,watering,photophobia in the earlier stages and vascularization of cornea.

Vitamin C - Vitamin C is associated with hemorrhages in the conjunctiva, lids, anterior chamber, retina and orbit. Delayed healing of wounds and ulcers of eyes.Increases the risk for the development of cataract.

Vitamin D - Deficiency of vitamin D may cause increased lacrimation.

Vitamin E - Vit. E acts as an antioxidant and plays a critical role in protecting the eye from development of cataract.

Vitamin K - Vit. k leads to coagulation defect and bleeding disorders in various parts of eyesubconjunctival and vitreous hemorrhage.

Early identification and proper management, along with a comprehensive vaccination programme can help to

prevent blindness due to vitamin deficiency.Corneal scars and blindness can be prevented if identified and treated early.

Eat foods rich in vitamins such as dark green leafy vegetables, yellow vegetables and fruits like Carrots, sweet potato, papaya, and mango.

DISCUSSION AND CONCLUSION

Vitamins are essential micronutrients whose role in visual function has been known for thousands of years. The role vitamin A plays in basic physiologic processes, such as growth, reproduction, immunity, and epithelialtissue maintenance, has been also known for a long time.Although vitamin A is essential throughout the entire life span, yet its influence is particularly critical during periods when cells proliferate rapidly and differentiate, such as during pregnancy and early childhood. Vitamin A and its precursors are available in several food items as well as in commercial preparations. Actions of vitamin A have been shown to be mediated through binding to particular nuclear receptors that regulate synthesis and physiological functions of different hormones. This process may also be facilitated through transport of the vitamin by certain plasma binding proteins.Deficiency of vitamin A may be associated with serioushealth problems such as night blindness, reduced immunity, increased risk of respiratory infections and hematological disorders. Such health problems can be successfully managed through the use pharmacological preparations of vitamin A or fortification of diet with vitamin A rich foods. On the other hand, chronic overuseof the vitamin may lead to more serious disorders especially affecting the bone and central nervous system; and may even precipitate birth defects. Furthermore, interactions of vitamins with certain drugs especially alcohol and nutrients may result in either vitamin deficiencies or risk of toxicities particularly affecting the liver and the lung tissues. Therefore, in spite of the undeniable role of vitamin in maintenance of healthy bodily functions, yet the use of this particular vitamin should be optimized to achieve the best prophylactic and therapeutic goals and, in the meantime, avoid serious adverse consequences.

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