# Ocular Menifestation of Malnutrition

*A.N.G. Ahamed Khan[1], Zeenat Ara Rahman[1], Shah Md. Keramat Ali[2],*

1. Dhaka Medical College Hospital
2. Institute of Nutrition and Food Science, University of Dhaka, Bangladesh

## Introduction

The deleterious effects of malnutrition may be seen in all age groups, but most commonly in children. Visual defects may be incremineted to mother's under nutrition during pregnanacy, state of nutrition during early childhood, and in advanced age. The possible effects of nutritional deficiency on the eye are many. There are about forty individual nutrients like vitamins, mineral elements, amino acids etc. which are required for the proper structure and function of ocular tissue. Again the structure and function of the tissues which makes up the eye are themselves manifold varied. Thus one nutrient may produce abnormalities in serveral tissues, and one tissue may be affected, perhape even in identical ways, by deficiency of more than one nutrient. A brief review of ocular manifestations of malnutrition is presented here.

### *Vitamin A Deficiency*

Vitamin A deficiency affect the anterior and posterior segment of the eye. In the former, the epithelium of the conjunctiva and cornea undergoes keratinization and in the later, there is interference with regeneration of visual pigments and specially with synthesis of the rhodopsin[1]

A. Anterior Segment (Conjunctiva and Cornea):
Deficiency of vitamin A leads to the follwoing characteristic changes:-

## Conjunctiva

1. Xerosis. The bulber conjectiva appears dry and lusterless.It is easily wrinkled, forming concentric folds around the limbus.

Bangladesh Journal of Nutrition, Vol. 2, No. 1, 46-50, December, 1988. Printed in Bangladesh. Institute of Nutrition &Food Science, University of Dhaka.

patches are usually located within the lid aperture and are frequently covered by fomy material.

3. Pigmentation. Melanin material exhibiting a yellowish or gray colour may be observed in the lower bulber conjunctiva, lower fornix, semiluner folds, upper lid, and fornix.

## Cornea

In the mild stage of involvement, there is loss of normal luster, with drying and reduced corneal sensitivity. Patchy areas resembling Bitot's spots of the conjunctiva can also be seen on the comeral surface near the limbus.

As the disease progresses, the cornea becomes progressively duller and dryer. In the severe stages, infiltrates are formed and the peripheral cornea becomes vascularized. Epithilial erosion, ulceration, infection, and ultimately perforation may develop.

Keratomalacia, the last stage of vitamin A deficiency is characterized by desication and necrosis of the cornea.

### *B. Posterior Segment (Retina)*

The earliest sign of vitamin A deficiency is night blindness[2]. In patients with night blindness (nyctalopta), vision is adequate in moderate lighting, but poor in dim illumination such as in a movie theater. The eye became incapable of adapting to lower light intensities and visual field may become constricted[3]. Night blindness also occurs as heriditery and congenital defects, in pigmentary degeneration of retina, and with retinal changes found in Oguchi's disease[1].

### *Vitamin B Complex Deficiency*

The vitamin B complex is composed of

serveral water soluble substances (thiamine, riboflavine, nicotinic acid, pantothionic acid, pyridoxin, choline, inositol, cyanocobalamin etc.) and it is difficult to relate eye changes to any one of these specific entities.

Patients with vitamin B complex deficiency present a multiplicity of complaints. Clinically, one may find a wide spectrum of eye involvement, ranging from mild sysptoms of photophobia, lacrimation, angular blepherokerat oconjunctivities and circumcorneal inection to servere corneal vascularization, optic neuritic, atrophy, night blindness, and nutritional amblypia [4,5,6] (Table-2).

### *Corneal vascularization*

Corneal vascularization caused by deficiency in riboflavin has been documented in animals, but in humans, covincing evidence has been lacking.

### *Corneal Epithelial Dystrophy*

Clinically, this appears as a form of superficial keratitis. The corneal epithelium has discrete grayish-white dots arranged in partterns of double lines traversing the cornea at the lower pupillary level[4] Central scotomas have been reported in about 50% of the cases. Frequently the corneal lesion is associated with nutritional amblyopia, however, the exact cause of corneal lesion is still controversial [1].

Involvement of extrinsic ocular muscle leading to nystagmus and ophthalmoplegia (Ptosis, occular muscle palsy) is also found to be associated with thiamin deficiency. Experimentally cataracts can be produced in deficiency of vitamin $B_2$ (riboflavine)[7]. Riboflavine deficiency probably acts by interference of normal glucose metabolims[8].

In tobacco amblyopia, deficiency of vitamin $B_{12}$ has been implicated[9] and there is in fact sosme association between this type of embloypia and that seen in pernicious anaemia.

### *Vitamin C Defieieny*

Vitamin C deficiency affects the eye as a hemorrhagic diathesis[1,4]. The sites of hemorrhage are found in the lids, conjectiva, anterior chamber, iris, the retina. Bleeding into skin of the lids appears as petechial hemorrhage. Intraorbital hemorrhage in infantile scurvy manifest clinically as spontenous proptosis[10].

Deficiency of vitamin C is also responsible for delayed healing of corneal wounds and ulcers, due to failure of formation of collagen [4,11].

### *Vitamin D Drficiency*

Although eye signs caused by vitamin D deficiency are rare, lameller cataract has been mentioned in Duka - Elder's text[4]. In children with rickets and in adults with osteomalacia, the lens should be searched for opacities that may be responsible for the diminution of vision.

Vitamin D intoxication: It is a special interest to note that large doses of vitamin D (150, 000 to 160,000 I.U. daily) have led to eye disturbances, clinically mainfested as photphobia and calcific deposits in the conjunctiva and cornea[4,12]. Imporvement was noted by withdrawal of the vitamin.

### *Vitamin K Deficiency*

Deficiency of vitamin K in late pregnancy is probably responsible for higher incidence of retinal hemorrhage in newborn[13].

### *Vitamin E Deficiency*

Deficiency of vitamin E have recently been found to be responsible for electro-physiological retinal dysfunction, visual field defect and pigmentary degeneration of the retina[14,15,16,17]. Supplementation of large doses of vitamin E has been found to be effective in preventing retinopathy of prematurity[18,19] retinopathy of abetalipoproteinaemia[20] and early cataract formation[21], Vit. E is also effective in

preventing and reducing the severity of retrolental fibroplassia in premature infants exposed to prolonged high oxygen tension in incubator[22].

The mode of action of vitamin E in the retina is poorly understood. Vitamin E is an antioxident which can scavange free radicles and thus protect membrance lipids from peroxidation. It is normally present in high concentration in the memberanes of rod outer segments of the retina[23] an area which is particularly susceptable to peroxidation. This is becuase it contains high concentration of poly unsaturated fatty acids[24] is exposed to a plentiful supply of oxygen, and light is known to enhance peroxidation[25]. A deficiency of vitamin E is therefore likely to accentuate peroxidative damage to the retina and also increase oxidative destruction of vitamin E[17].

Combine deficiency of vitamin A and E has been found to be resulted in a synergistically delecterius effect on the retina of rats[15].

Deficiency of certain minerals like calcium, zinc, copper, chromium and has been reported to produce eye changes in experimental animals[8].

***Protein Deficiency***

Protein deficiency is seen endemically in the tropical and subtropical countries where malutrition commonly occurs, and else where in times of famine. Young patients are particularly prone to servere protein deficiency (Kwashiorkor), even though they may getting adequate supply of calories, such as on a rice or maize diet[4].

In protein deficiency, the conjunctival vessels may, appears balanced, giving rise to a procelain-like appearance of the sclera. The incidnence of marginal blepharitis and stye is increased[1].

Polymorphic Superficial Keratopathy: The corneal lesions observed in protein deficiency consists to patches of degenerative changes in the epithelial cells, associated with round or oval zones of infiltration in the subepithelial tissues. Ulceration may occur, but usually does not penetrate Bowan's membbrance[4].

Xerosis and Keratomalacia have been described in young children with Kwashiorkor[26]. However, in these cases vitamin deficiencies may also be the contributing factors. The corneal manifestation becomes more evident in the terminal stages.

Prolonged dietary deficiency of certain amino-acids such as tryptophan, phenylalanine and histidin, can contribute to the production of cataractus changes in the lens of experimental animals[7,8,27].

Other instances of eye involvement such as subconjunctival hemorrhage, faulty dark adaption, night blindness, optic atrophy, and increased myopia, are thought to be related to protein dificiency, but deficiency in other nutrients can also be cosntributing factor.

## Carbohydrate Malnutrition

Ocular changes can occur in defective utilization of carbohydrate rather then actual deficiency, as seen in diabetes mellitus, hypergalactosemia etc.

Cataract can developed in infantile hyoglycemia, a group of disease in which low plasma glucose levels are present[28]. However, evidence to support the widely circulated concept that human senile cataracts may caused by decreased glucose metabolism or lens anoxia is not available[29].

Galactose induced cataract is found in animals fed a diet rich in lactose or galactose and in young human being with a congential deficiency of the enzyme involved in galactose metabolism. Simoons [30] has noted

Table I. *Ocular Manifestation of Malnutrition*

| Anatomy | Pathology | Vitamin Deficiency | | | | | | | | Protdin deficiency |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | B Complex | | | | | | | |
| | | A | $B_1$ | $B_2$ | Ni cin | $B_6$ | $B_{12}$ | C | D | E |
| Lids | Hyperkeratosis of skin and mucus membrane | + | | | | | | | | |
| | Angular blephero conjunctivitis.. | | | + | | + | | | | |
| | Hemorrhage | | | | | | + | | | |
| Conjunctiva | Wrinkling | + | | | | | | | | |
| | Xerosis | ++ | | | | | | | | |
| | Bitot's spots | ++ | | | | | | | | |
| | Pigmentation | + | | | | | | | | |
| | Hemorrhage | | | | | | + | | | |
| Cornea | Keratomalacia | + | | | | | | | | |
| | Vascularization | | | ++ | | | | | | |
| | Epith. Keratitis | | | ++ | | | | | | |
| | Ulceration | + | | + | | | | | | |
| | Hypopyon | | | + | | | | | | |
| | Xerosis | + | | | | | | | | |
| | Leukoma | + | | | | | | | | |
| | PSK* | | | | | | | | | |
| Anterior Chamber | Hemorrhage | | | | | | + | | | |
| Iris | Hemorrhage | | | | | | + | | | |
| Lens | Lamellar Cat. | ± | | ± | | | + | + | | + |
| Fundus | Macular Stripling | + | | | | | | | | |
| | Macular pallor | + | | | | | | | | |
| | Optic atrophy | + | | | + | | | | | |
| | Optic neuritis | | + | + | | ± | ± | | | |
| | Retinal Hamorrhage | | | | | | + | | | |
| | degeneration | | | | | | | | + | |
| Orbit | Hemorrhage | | | | | | + | | | |
| | Proptosis | | | | ± | | + | | | |
| Vision | Nactalopi | + | | + | | | | | | |
| | Photophobia | + | | + | | | | | | |
| | Amblyopia | | + | | | + | | | | |

(+) Indicates presence of correlation.
(±) Indicates equivocal presence of correlation.
*Polymorphic Superficial keratopathy.

Table 2. *Correlation of Nutritional Deficiencies with Ocular and Systemic Manifestation*

| Nutritional Factors | Eye Involvement | Systemic and other Manifestations |
|---|---|---|
| Vitamin A | Xerosis (nutritional) xerophthalmia, Bitot's spots keratomalacia Night Blindness | Fatty epiphyseal bone formation defective tooth enamel, keratinization of mucous membrane and skin, retered growth. |
| Vitamin B Complex | | |
| Thiamine ($B_1$) | Accommodative Fatigue Nutritional amblyopia | Beriberi, Wernicke's syndrome, nystigmus,peripheral polyneuropathy. |
| Riboflavin ($B_2$) | Ariboflavinosis (Photophobia, blurred vision, burning and itching of eyes, corneal vascularization and cataract formation. | Cheilosis, angular stomatitis, dermatitis. |
| Niacin | Conjunctivitis, Optic atrophy,ocular proptosis. | Pellegra, stomatitis, glositis |
| Pyridoxin ($B_6$) | Angular blepharoconjunctivitis | Seborrhea like skin lesions, nerve inflamation: epileptic form with convulsion in infants; anaemia. |
| Cyanocobalamin ($B_{12}$) | Amblyopia (tobaco amblyopia) | Pernicious anemia |
| Vitamin C | Hemorrhagic diathesis (lids, orbit, conjunctiva, anterior chamber iris, retina). Delayed healing of corneal ulcer and wound. | Scurvy (irritability, slow growth susceptibility to infection, poor wound healing, hemorrhage, loose teeth, gingivitis. |
| Vitamin D | Lamellar cataract | Rickets and osteomalacia |
| Vitamin E | Retinal degeneration early cataract formation. | Infertility and early senile changes. |
| Protein | Polymorphic superficial keratopathy. Cataract formation. | Kwashiorkor (protein malnutrition) |

the high incidence of senile cataract in some group of people who consume large quantities of milk and lactose rich diary products and who in addition have a high frequency of persistant loctose activity in adult life.

### *Lipid Malnutrition*

The lipids are major components of the lens fibre membrances and either decrease in their synthesis or impaired degradation bring about membrance damage and lens opacities. It is observed that cataracts develop in humans treated with anticholesterolemic agents such as triparanol On the other hand, cataracts are present in cholesterolemic Xanthomatosis, an inborn error of cholesterol degradation[29]

## Discussions

The preservation and promotion of eye sight is always affected by malnutrition. Opthalmic manifestation of this conditions are highly prevalent in developing countries like Bangladesh. Since malnutrition is the outcome of serveral factors, the problem can best be solved by taking action simultaneously at Family, Community, National and International levels. It requires a co-ordinated approach like changing of food habit, education of health and nutrition, increasing production of food, nutritional intervention programe, and nutrition related health activity such as immunization, family planning etc. It calls-for a comprehensive programme of socio-economic development of the entire country.

Awarness of the eye changes that occur in theearly and late stages of nutritional deficiency disease would hopefully alert the clinician and the nutritionist to make an early diagnosis, initiate appropriate intervention program and thus prevent reversible disease from drifting into catastrophic sequelae.

## Summary

Deficiencies of vitamin A, the vitamin B complex, vitamin C, vitamin D and protein have been increminated as the causative factors in nutritional diseases associated with ocular manifestations. In recent years, deficiency of vitamin E is also balmed to be responsible for may visual abnormalities.

### Reference

1. Chan G. H., Nutritional deficiency disorder, In Pediatric Ophthalmology (Robinon D. Harley, editor), 2nd ed. Vol. II, W. B. Saunders Company, Philadelphia, 957, 1983
2. Yudkin J., Nutritional deficiency,In Sorsby H.,(ed>); Modern Ophthalmology, 2nd. ed., Vol, II, Butterworths, London, 271, 1972.
3. Duke-Elder, S.; System of Ophthalmology, Vol, IV, The C. V. Mobsy Co.; St. Louis; 589, 1968.
4. Duke-Elder, S., System of Ophthalmology, Vol. VIII, Part 2, The C. V. Mosby Co., St. Louis, 1111, 1965.
5. Venkataswamy G.,Ocular manifestations of vitamin B-complex deficiency, Brit. J. Ophthalmol. 51; 749, 1967.
6. Venkataswamy G., Angular conjunctivitis and ribo lavine deficiency, J. All India Ophthalmol. Soc. 8, 33, 1960.
7. Person's Diseases of the Eye, Stephen J. H. Miller, 7gh ed. Churchill Livingstone, Edinburgh, London, Melbourne, and New York, 174, 1984.
8. K. Seetharam Bhat, Nutrition News, National Institute of Nutrition, Hyderabad, Vol. 4, No. 1, 1983.
9. Heaton, J. M. Mccormick, AJA, and Freeman A.·G.; Tobaco Amblyopia: A Clinical Manifestation of Vitamin B 12 Deficiency, Lancet, 2; 286, 1958.
10. Harley, R. D., Exophthalmos in the newborn from vitamin C deficiency, Am. J. Ophthalmol., 26; 1314, 1943.
11. Robbins, S. L., Cotran, R. S., Kumar, V., Pathologic Basis of Disease, 3rd ed., W. B. Saunders Company, Philadelphia, 419, 1984.
12. Harley, R. D., Ocular changes in idiopathic hypercalcemia, Trans. Acad. Ophthal. and Oto., 69: 879, 1963.
13. Scheie H. G., Albert, D. M. Textbook of Ophthalmology, 9th ed., W. B. Saunders Co. Philadelphia, 456, 1977.

14. Alverz F., Liandrieu P., Lagetp, Lemonnier F., Odievere M. Alagille D., Nervous and ocular discorder in children with cholestasis and vitamin A and E deficiencies, Hepatology, 410, 1983.

15. Howard, L., Ovesen, L., Satya-Murti S., Chu R., Reversible neurological symptoms caused by vitamin E deficiency in patients with short bowel syndrome, Am J. Clin. Nutr., 36; 1243, 1982.

16. Hayes K. C., Retinal degeneration in monkeys induced by deficiencies of vitamin E or A, Invest. Ophthalmol. Vis. Sci., 13; 499, 1974.

17. Robinson W. B., Kuwabara T., Biori J. G., Deficiencies ofvitamin E and A in the rat. Retinal damage and lipofucin accumulation, Invest. Ophthalmol. Vis. Sci., 19; 1030, 1980.

18. Schaffer D. B., Vitamin E and retinopathy of prematurity, follow-up at one year, Ophthalmology, 92(8); 1005, 1985.

19. Johnson L., Vitamin E and retinopathy of prematurity (letter), Pediatrics, 81(2); 329, 1988.

20. Runge P., Muller D. R. R., Mcallister J., Calver D., Loyd K. J. Oral vitamin E supplements can prevent the retinopathy of abetalipoproteinaemia, Brit. J. Ophthalmol., 70; 166, 1986.

21. Gupfta P. P., Prevention ofexperimental cataract by Alphatocopherol, Indian J. Exp. Biol., 22(11); 620, 1984.

22. Hittner H. M., Retrolental fibroplastia: Efficacy ofvitamin E in a double blind clinical study of preterm infants, N. Eng. J. Med., 305; 1365, 1981.

23. Dilley R. A., Mcconnel D. G., Alpha-tocopherol in theretinal outer segment of bovine eyes, J. Member. Biol., 2; 317, 1970.

24. Daemen F. J. M., Vertibrate rod outer segment membrances, Biochem. Biophys. Acta., 300; 255, 1973.

25. Feeney L., Berman E. R. Oxygen toxicity: Membrane damage by free raticals, Invest. Ophthalmol. Vis. Sci., 15; 789, 1976.

26. Oomen HAPC, Xerophthalmia in presence of Kawshorkor, Brit.J. Nutr. 8; 307, 1954.

27. Dobree, J. H., Cataract, in Sorsby H. (ed.); Modem Ophthalmology, 2nd ed., Vol. IV, Butterworths, London, p. 657, 1972.

28. Mertin S. and Crawford J. S.; Hypoglycemia and infantile cataract. ARCH. ophthalmol. 86; 495, 1971.

29. Cotlier E.; The lens, In Adler's Physiology of the Eye-Clinical Application, Moses RA (ed.) 7th ed. The C. V. Mobsy Company, St. Luis, Toranto, London; 291; 1981.

30. Simoons F. S., A geographic approach to senile cataracts; Possible links with milk conjumption, Lactose activity and Galactose metabolism, Dig. Dis. Sci., 27: 257-64, 1982.