

Vitamin A deficiency, a potential threat to sight in transplant recipients

Fatema Ali Lanewala

Vitamin A (Vit A or Retinol) is an essential nutrient for proper human body functioning including embryogenesis, vision, cell proliferation and differentiation, immune regulation, and glucose and lipid metabolism. It is mainly acquired through diet rich in Retinol or Retinyl esters; or its precursors like carotenoids, Provitamin A, and β carotene.^{1,2}

Plant-derived carotenes and animal-derived retinyl esters are mixed with bile salts in the digestive tract and made available for absorption in the small intestine. Retinyl ester hydrolases (REHs) in the gut lumen release retinol from retinyl esters, after which it is absorbed by enterocytes while carotenes are taken up by membrane-bound transporters, and metabolized to retinol inside enterocytes. Next, retinol is re-esterified to retinyl esters, sequestered into chylomicrons (CM) and secreted to the circulation. CM that contain most of the retinyl esters are taken up by hepatocytes, and the retinyl esters are then hydrolyzed to retinol through the action of REHs. This inactive product is then transferred to the circulation by binding with retinol binding protein 4 (RBP4) and transthyretin (TTR). Through blood circulation, retinol is directed to the peripheral tissues in demand of retinol where it is converted to bioactive retinoic acids. The remaining retinol is taken up by the hepatic stellate cells (HSC) that convert it back again to retinyl esters and store it in large cytoplasmic lipid droplets. Approximately 60–95% of the whole body's reservoir of vitamin A is found in the liver of a healthy individual, but significant amounts may also reside in adipose tissue, the pancreas, the intestines, and the eyes.²

Vitamin A deficiency results from an inadequate dietary intake of vitamin A that is unable to fulfill an individual's physiological needs. Vitamin A deficiency may be exacerbated by high rates of infection, especially diarrhoea and measles. It is common in low and middle income countries, and may rarely be seen in the high-income countries. Vitamin A deficiency is a public health problem in a large part of the world, especially those in Africa and South-East Asia.^{4,5} The most severe effects of this deficiency are seen among young children and pregnant women in these parts of the world.³

Department of Ophthalmology, Sindh Institute of Urology and Transplantation (SIUT), Karachi, Pakistan.

Correspondence: Fatema Ali Lanewala e-mail: fatemali92.110@gmail.com
ORCID ID: 0000-0002-9024-0475

Xerophthalmia is the clinical spectrum of ocular manifestations of vitamin A deficiency; these range from the milder stages of night blindness and Bitot spots to the potentially blinding stages of corneal xerosis, ulceration and necrosis (keratomalacia).⁶ The various stages of xerophthalmia are regarded both as disorders and clinical indicators of vitamin A deficiency.

Night blindness (also called Nyctalopia, in which it is difficult or impossible to see in relatively low light) is one of the clinical signs of vitamin A deficiency. It is common during pregnancy in developing countries.³ Due to multiple reasons, logistical and practical, night blindness is most effectively assessed by taking a history.⁷ There are many other factors responsible for poor night vision. These include defective transmission of light through the lens, impairment of pupillary dilatation, nearsightedness, congenital or inherited development of the retina being few of the important ones.

While the most common population affected by night blindness include children and pregnant women, our experience at a transplant centre in Karachi, Pakistan has demonstrated its high prevalence among kidney transplant recipients. It is believed that the main reason among such a population is maladaptation of rod function due to Vitamin A deficiency.^{8,9}

Our study published in the Journal of Pakistan Medical Association April 2023 is the first to ever report vitamin A deficiency as a possible cause of nyctalopia in renal transplant population all over the world.⁹ While conducting

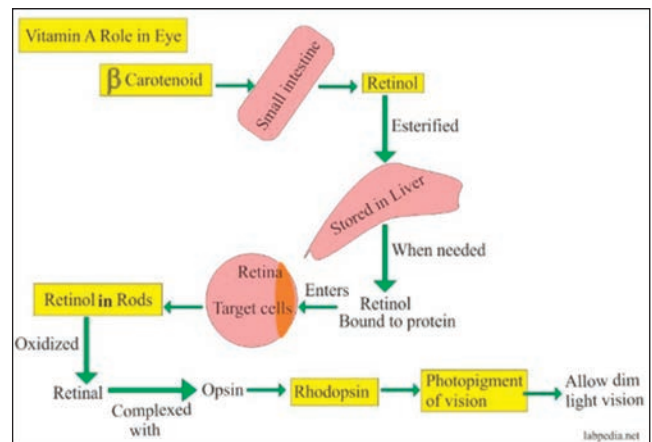


Figure-1: Vitamin A metabolism and its role in the eye.¹

routine transplant follow up clinics at the Department of Ophthalmology at Sindh Institute of Urology and Transplantation (SIUT), frequent complaint of night blindness was reported in otherwise stable recipients who were generally doing well. These patients were clinically seen to have dry eyes and complained of poor night vision causing hindrance in their routine activities.

The serum Vitamin A levels were requested, but could not be performed due to logistical constraints. After taking detailed history, and thorough clinical examination, this situation was discussed with the transplant team. After careful consideration and nutritionist's consultation, Vitamin A rich diet and oral tablets were prescribed. In addition to other nutritional supplements, one capsule Seven Seas was given daily which contains 750µg RE of Vitamin A.¹⁰ Marked improvement was observed in patient's symptoms including dry eyes complaint leading to a healthier ocular surface. Patients reported an improvement in the quality of their life over time.

An Ethiopian study has demonstrated high prevalence of dry eyes in transplant recipients, but they have attributed it to the local climate.¹¹ In our opinion, this could also have been secondary to Vitamin A deficiency as the particular vitamin is essential in maintaining a moist and healthy ocular surface.

On the basis of our study, we are now trying to establish Vitamin A deficiency as the cause of night blindness in renal transplant recipients in Pakistani population. For this purpose, we encourage other transplant centers to take an initiative and manage Vitamin A deficiency in a proactive manner. We, in the meantime, are trying to evaluate serum Vitamin A levels in our transplanted population in order to establish this deficiency as a definitive cause of night blindness.

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Conflict of Interest: None.

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