

Development of Night Blindness in Cirrhotic Patients¹Parveen Malhotra, Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India²Ankit Chahal, Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India³Janvi, Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India⁴Rahul Siwach, Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India⁵Manav, Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India⁶Kshitez, Department of Medical Gastroenterology, PGIMS, Rohtak, Haryana, India**Corresponding Author:** Parveen Malhotra, Department of Medical Gastroenterology, PGIMS, Rohtak & 128/19, Civil Hospital Road, Rohtak, Haryana, India**How to citation this article:** Parveen Malhotra, Ankit Chahal, Janvi, Rahul Siwach, Manav, Kshitez, “Development of Night Blindness in Cirrhotic Patients”, IJMACR– September – 2026, Volume – 9, Issue – 5, P. No. 914 – 917.**Open Access Article:** © 2026 Parveen Malhotra, et al. This is an open access journal and article distributed under the terms of the creative common’s attribution license (<http://creativecommons.org/licenses/by/4.0>). Which allows others to remix, tweak, and build upon the work non - commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.**Type of Publication:** Original Research Article**Conflicts of Interest:** Nil**Abstract**

Introduction: Night blindness i.e. nyctalopia in cirrhotic patients is most commonly caused by a severe deficiency of vitamin A, an essential nutrient processed and stored by liver. It normally stores a large supply of vitamin A, so night blindness usually does not appear until the late stages of liver disease when these reserves run out. Cirrhosis impairs bile production, which the body needs to absorb fat-soluble vitamins like vitamin A. Advanced liver disease often involves low zinc levels, which reduces the carrier proteins (**retinol-binding proteins**) needed to transport vitamin A to the eyes. Patients feel difficulty in seeing or driving in the dark, or a long adjustment time when moving from light into dark areas. Many patients do not realize they have night blindness until specifically enquired for, as they adapt their habits

to avoid dim lighting. The diagnosis relies on serum vitamin A and retinol-binding protein levels, and an electroretinogram (ERG) to measure retinal function. High-dose oral vitamin A is often much more effective than intramuscular shots in cirrhotic patients to restore dark adaptation.

Aims and Objectives: To determine prevalence of Night blindness in cirrhotic patients at tertiary care center of Northern India.

Materials & Methods: It was prospective study conducted at Department of Medical Gastroenterology, Post Graduate Institute of Medical Sciences (PGIMS), Rohtak, over a period of two years from 1st September, 2024 to 31st August, 2026. The 300 confirmed liver cirrhosis patients were enrolled in the study and all were cirrhotic (fibroscan greater than 12.2 Kpa) or proven on

ultrasound abdomen and biochemical labs. Only those patients who had at-least three years or more duration of being diagnosed with cirrhosis, were enrolled in the study. This was done with aim of giving ample time for developing night blindness, if any, so as to decrease false negative rate.

Results: Our all 300 cirrhotic patients were predominantly males. The cirrhotic patients had mixed aetiology including alcoholic liver disease, chronic hepatitis B & C, metabolic syndrome associated and, in few cases, autoimmune liver disease or primary sclerosing cholangitis. The maximum percentage (78%) were above 40 yrs of age; the peak age group was between 41-60 yrs (54%). Out of these 300 patients, 66 (22%) developed night blindness. Out of these 66 patients who developed night blindness, 53 (80.30%) were decompensated and only 13 (19.70%) were in compensated stage. The average time period for developing night blindness after being diagnosed as cirrhotic varied between 2- 6 years.

Conclusion: The cirrhotic patients developed night blindness predominantly in decompensated stage and prevalence increased with progression of decompensation. Hence, it's anticipation by the treating team is mandatory which can be achieved by regularly testing for the same. Moreover, patients should also be motivated for being watchful for their vision in nighttime or in dark conditions. Timely detection and appropriate treatment of the same can lead to reduction in morbidity and mortality associated with it.

Keywords: Night blindness, Cirrhosis, Vitamin A, Decompensation, Retinol

Introduction

Vitamin A is fat soluble retinoids that include retinol, retinal and retinyl esters which is essential for vision,

growth, reproduction and immunity. Liver plays pivotal role in absorption of vitamin A as its absorption is mediated by bile salt induced micelles formation. In a normal liver, hepatic stellate cells are major storage sites for vitamin A. Hepatic stellate cells following activation lose vitamin A and transformed into myofibroblasts resulting in fibrosis and cirrhosis development through multiple pathways¹. Intake, diseases of gastrointestinal tract, pancreas and liver can lead to deranged serum vitamin A level and consequently clinical manifestation of vitamin A deficiency. Vitamin A deficiency leads to night blindness and xerophthalmia in the form of Bitot's spots, xerosis, keratomalacia, and even retinopathy^{2,3}. Data related to vitamin A status in non-cholestatic chronic liver diseases and relation between low serum retinol level and clinical manifestation in the form of xerophthalmia are limited and inconclusive. In one study, out of 63 patients of cirrhosis of liver being evaluated for liver transplant, Venu M et al. reported vitamin A deficiency in 69.8 percent but none had clinical manifestation of night blindness⁴.

Aims and Objectives- To determine prevalence of Night blindness in cirrhotic patients at tertiary care center of Northern India.

Material and Methods- It was prospective study conducted at Department of Medical Gastroenterology, Post Graduate Institute of Medical Sciences (PGIMS), Rohtak, over a period of two years from 1st September, 2024 to 31st August, 2026. The 300 confirmed liver cirrhosis patients were enrolled in the study and all were cirrhotic (fibroscan greater than 12.2 Kpa) or proven on ultrasound abdomen and biochemical labs. Only those patients who had at-least three years or more duration of being diagnosed with cirrhosis, were enrolled in the study. This was done with aim of giving ample time for

developing night blindness, if any, so as to decrease false negative rate.

Statistical Analysis- All the data was entered in Microsoft Excel and was analysed using SPSS 15.0 version.

Observations & Results- Our all 300 cirrhotic patients were predominantly males. The cirrhotic patients had mixed aetiology including alcoholic liver disease, chronic hepatitis B & C, metabolic syndrome associated and, in few cases, autoimmune liver disease or primary

sclerosing cholangitis. The maximum percentage (78%) were above 40 yrs of age; the peak age group was between 41-60 yrs (54%). Out of these 300 patients, 66 (22%) developed night blindness. Out of these 66 patients who developed night blindness, 53 (80.30%) were decompensated and only 13 (19.70%) were in compensated stage. The average time period for developing night blindness after being diagnosed as cirrhotic varied between 2- 6 years.

Table 1: Showing Sex, Geographical and Age Distribution in Cirrhotic Group with Night Blindness

Cirrhosis Patients	Male	Female	Rural	Urban	20-40 yrs	41-60 yrs	61-80 yrs
300 (100%)	180 (60%)	120 (40%)	177 (59%)	93 (31%)	66 (22%)	162 (54%)	72 (24%)

Table 2: Showing prevalence of Development of Night Blindness in Cirrhotic Patients

Total Cirrhotic Patients	Developed Night Blindness	Not Developed Night Blindness
100	66 (22%)	234 (78%)

Discussion

Vitamin A is now widely recognized to have multiple health related functions. Vitamin A and its retinoid derivatives are essential for physiological functions, including vision, cellular proliferation and differentiation and immune system activity. The liver is one of the major organs involved in its metabolism. In present study, 350 patients with liver cirrhosis of various etiologies were evaluated for vitamin A status. Alcohol is the most common etiology of cirrhosis (50%) followed by HBV infection in our study. Out of 300 patients, 66 (22%) developed night blindness. Prevalence of vitamin A deficiency was reported to be 54.3% in 140 patients of hepatitis C patients by Peres et al⁵. Paula et al⁶ found vitamin A deficiency in 60% of 58 patients of cirrhosis of liver. There were multiple mechanisms for vitamin A deficiency in chronic liver disease. One of them was

because of poor nutrition. Low intake of animal sources also contributed to the reduced serum retinol levels observed in chronic liver disease (CLD) patients. Dietary beliefs, taboos and constraints associated with liver disease leads to reduced intake of protein and fat and, consequently, compromise the intake and absorption of preformed vitamin A, as well as other micronutrients⁷. There occurs malabsorption of vitamin A and other fat-soluble vitamins. There is reduction in hepatic synthesis of the retinol carrier protein due to dysfunction of the organ or protein-energy malnutrition^{8,9}. The conversion of β -carotene to retinol occurring in the liver might be deficient in these patients, contributing to the lowered levels of serum retinol in these patients¹⁰. Vitamin A deficiency can cause xerophthalmia, which can manifest in an array of signs including night blindness, corneal and conjunctival xerosis, bitot spots, corneal ulceration, and

keratomalacia. Recognizing these signs can be very helpful in identifying a deficiency in vitamin A as well as prompting tests of both serum levels of vitamin A and serum levels of other nutrients because the most common causes of vitamin A deficiency are also likely to cause deficiencies in other nutrients¹¹.

Conclusion

The cirrhotic patients developed night blindness predominantly in decompensated stage and prevalence increased with progression of decompensation. Hence, it's anticipation by the treating team is mandatory which can be achieved by regularly testing for the same. Moreover, patients should also be motivated for being watchful for their vision in nighttime or in dark conditions. Timely detection and appropriate treatment of the same can lead to reduction in morbidity and mortality associated with it.

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