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Ophthalmology

NIGHT BLINDNESS CAUSES: A HOSPITAL BASED STUDY

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ABSTRACT **Purpose-** The aim of this study was to provide a retrospective analysis of causes and clinical profile of patients of complaining of night blindness in the Department of Ophthalmology at M.L.B. Medical College, Jhansi. **Materials And Methods-** This was a retrospective observational study that involved 20 patients who presented to us with chief complaints of night blindness in the Department of Ophthalmology at M.L.B. Medical College, Jhansi. **Results-** In this study the maximum patients were in the age group of 20-40 years. Majority of patients who presented early had Vitamin A deficiency. Retinitis Pigmentosa presented to us in the age group of mainly 20-40 years and were only males in our study. In Vitamin A deficiency majority of patients were males. **Conclusions-** Nyctalopia may be the first presenting symptom of inherited conditions such as retinitis pigmentosa or acquired diseases such as vitamin A deficiency. Other caused of night blindness are pathological myopia, cortical cataract and other retinal causes. Vitamin A deficiency is a treatable condition thus early diagnosis can prevent its burden and improve the visual outcome. For retinitis pigmentosa genetic counselling is advised.

KEYWORDS : Retinitis Pigmentosa, Nyctalopia, Atypical Retinitis Pigmentosa, Vitamin A Deficiency, Pathological Myopia, Cortical Cataract.

INTRODUCTION:

Nyctalopia refers to night blindness or difficulty of the eye in visualizing under dim light or at night. It is due to the eye's inability to adapt quickly from lightness to darkness. The principle cell-type associated with Nyctalopia is rod cells. Rods have a singular photopigment, rhodopsin, which utilizes the protein scotopsin and the Vitamin A-derived cofactor, retinol.[1] This cascade is essential for the body's ability to regulate the pupillary light reflex. The pupillary light reflex allows unilateral afferent detection of changes in light energy entering the eye, and efferent adjustments in the pupillary sphincter and dilator pupillae muscles to initiate consensual constriction and dilation of the eyes.[2] Night blindness is the physical manifestation of impaired functioning of these processes. There are both acquired and congenital forms of night blindness. Acquired forms of night blindness include an insufficiency of vitamin A and paraneoplastic syndromes (melanoma-associated retinopathy and cancer-associated retinopathy). Congenital forms include both stationary (in which the severity remains relatively constant throughout life) and progressive (in which severity increases over time) forms of night blindness. Stationary forms can result from genetic mutations in rod photoreceptors or rod bipolar cells. Progressive forms include retinitis pigmentosa, choroideremia, and gyrate atrophy.

MATERIALS AND METHODS:

This study was a retrospective observational study that involved 20 patients who presented to us with chief complaints of night blindness. The patients were recruited from the OPD in the Department of Ophthalmology at Maharani Laxmi Bai Medical College, Jhansi between June 2022 to February 2023. It was performed under the Helsinki Declaration of 1997 revised in 2000. The necessary permission from the Ethical and Research Committee was obtained for the study.

Inclusion Criteria:

All patients who presented to us with chief complaints of night blindness

Exclusion Criteria:

- Age less than 1year or more than 70 years.
- Patients with CORTICAL cataract.
- Patients with pathological myopia.
- Patients with cognitive or language impairment.
- Patients with other debilitating systemic condition.
- Pregnant or breastfeeding women.
- Refusal to provide consent.

All patients who were included in the study were subjected to proper history taking ad examination which included visual acuity testing using Snellens visual acuity charting, colour vision, refraction both subjective and retinoscopy. Fundus examination was done using Indirect Ophthalmoscopy and 20 D LENS, and later fundus photo was taken using Zeiss Fundus Camera. Further SD-Optical Coherence Tomography was also done to study the further complications.

RESULTS

In this study the maximum patients were in the age group of 20-40 years. Patients in the age group of 1-20years had Vitamin A deficiency in 40% cases rest 10% patients were in age group of 20-30 years. There were 40% patients of Typical Retinitis Pigmentosa in this study which were primarily from the age group 20-40 years. 10% patients in our study had Atypical Retinitis Pigmentosa. 80% patients who presented to us were males and rest 20% females. In this study in patients of Vitamin A deficiency 20% were females and rest 30% males., while in Retinitis Pigmentosa whether Typical or Atypical were all males.

Table1: showing age wise distribution

AGE(years)	n(%)
1-5	06(30%)
5-20	05(25%)
20-40	07(35%)
40-60	02(10%)
60-80	00(00%)

Table 2: Distribution of patients according to diagnosis.

Diagnosis	MALES	FEMALES
Vitamin A deficiency	06	04
Retinitis Pigmentosa	08	00
Atypical Retinitis Pigmentosa	02	00

DISCUSSIONS:

Nyctalopia may be the first presenting symptom of inherited conditions such as retinitis pigmentosa or acquired diseases such as vitamin A deficiency. Night blindness is sensitive and specific for serum retinol levels and is the earliest clinical manifestation of vitamin A deficiency. Night blindness may present with recurrent nighttime falls and difficulty with nighttime driving.[03]

Myopia, or nearsightedness, is refractive error pathology, which can cause nyctalopia. Myopia occurs due to an “elongated” eye in which the focal point converges in front of the retina, creating a progressively blurrier image of far distance objects. This blurriness may be accentuated in dim light, manifesting as a common etiology of nyctalopia. Corrective lenses and prescription eyeglasses based on calculated refractive error improve dim light vision.[4][5]

Hereditary retinal dystrophies are a rare, but significant, cause of nyctalopia. In congenital stationary night blindness (CSNB), there is

impaired photoreceptor transmission leading to impaired dark adaptation. Complete type (CSNB1) and incomplete type (CSNB2) are rare heterogeneous conditions, most commonly X-linked. CSNB1 results from a diseased gene in the region between DXS556 and DXS8083 in Xp11.4-p11.3. CSNB1 characteristically results from mutations in genes involved in neurotransmitter detection by bipolar cells and reduced rod sensitivity up to 300x. A different locus is responsible for CSNB2, localized to the region between DXS722 and DXS8023 in Xp11.23; CSNB2 demonstrating membrane defects involved in neurotransmitter release by photoreceptor cells.[6][7]

Vitamin A deficiency is among the leading causes of blindness worldwide, particularly in developing countries. Xerophthalmia, Bitot spots, keratomalacia, conjunctival and corneal xerosis, retinopathy, developmental defects, and nyctalopia are among the associated clinical ocular findings. Vitamin A is necessary for normal visual function and maintenance of the corneal epithelium. Vitamin A is a visual pigment precursor, and subnormal levels of 11-cis-retinal may lead to a decline in the visual sensitivity of peripheral rod photoreceptors. [8] Nyctalopia associated with vitamin A deficiency is reversible and managed with retinal supplementation.[9]

Retinitis pigmentosa (RP), also known as hereditary retinal dystrophy, refers to a group of disorders with progressive loss of vision representing the most common inherited retinal disease. Nyctalopia is generally the first symptom of RP, followed by a gradual narrowing of the visual field or "tunnel vision" and eventually, total vision loss. Vitamin A supplementation may slow RP progression.[10]

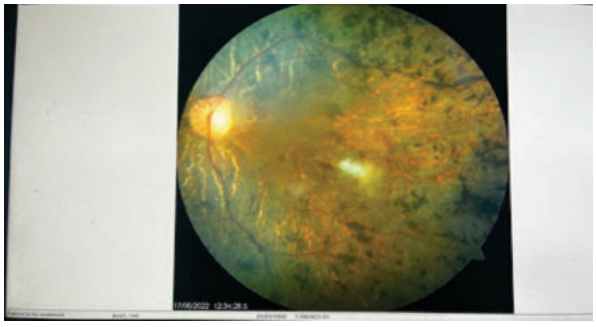

Image1: showing fundus photo of Retinitis Pigmentosa

Image2: showing fundus photo of Xerophthalmic fundus

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