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Pattern of Colour Vision Anomalies Among Patients Presenting to a Tertiary Eye Center of Nepal

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ABSTRACT

Introduction

Colour vision deficiency (CVD) is the inability to clearly differentiate colour differences under normal lighting condition. People are unaware of colour vision defect due to which they suffer in various aspects of their career. The purpose of this study was to explore the colour vision defect pattern among patients attending tertiary eye centre, Kathmandu, Nepal.

Methods

A hospital based, retrospective study was conducted to evaluate the defective colour vision pattern in patients attending B.P. Koirala Lions Centre for Ophthalmic Studies, Institute of Medicine, Kathmandu, Nepal where total of 692 subjects medical case sheets were reviewed and included in the study from September 2018 to April 2019.

Results

Among 692 patients enrolled in the study, 272 (39.30%) patients were found to have CVD . Among 272 CVD,227(83.45%) were males and 45(16.54%) were females. Congenital colour vision defect was found in 139 (26.73%) males and 21 (13.81%) females. Acquired color vision defect was found in 88 (16.92%) males and 24 (15.78%) females . When congenital CVD was observed, deutan (28.3%) CVD was more prevalent than protan (22.79%) and tritan defect (7.72%). For acquired CVD, non-specific type of CVD (8.92%) was more prevalent followed by tritan CVD (5.35%).

Conclusion

Significant proportion of patient presenting for eye care at tertiary eye hospital have color vision defects. Congenital CVD was found more in males than females whereas acquired color vision defect was found almost in same proportion in both males and females.

Keywords
Colour vision deficiency, Fransworth D15, Ishihara, pseudoisochromatic chart

INTRODUCTION

Colour vision deficiency (CVD) is the inability to distinguish different colours under normal lighting condition.[1] Congenital colour vision defect is the commonest X-linked recessive disorder.[2] The acquired deficiencies are caused by neurological diseases, metabolic disorders, drug toxicity, diabetic retinopathy, hypertension, glaucoma, macular degeneration, yellowing of lens due to aging.[3]

Acquired CVDs can be classified as Type 1 or Type 2 red-green deficiencies and Type 3 blue yellow color deficiency.[4] Congenital CVD are stationary and often affect both eyes equally, whereas acquired deficiencies may be progressive and may affect just one eye depending on the underlying cause.[5] CVDs at the retinal level occur when the development of photopigments in one or more of the cone cells are defective.[6] The highest prevalence of color CVD in general population occurs with red-green color defects which is usually X-linked recessive, thus occurring predominantly in males, but transmitted by females with 8% of the female population being carriers.[4] Colour blind individuals should be advised against training for occupations like pilots, certain jobs and armed forces, electrical jobs, navigators and police, medical scientific officers.[7,8] This study was done to evaluate the color vision pattern in tertiary eye hospital of Nepal.

METHODS

Ethical clearance was taken from the institutional review committee of the Institute of medicine (Ref.53(6-11)E[2] 078/079. Retrospective review of medical case sheets was done to evaluate the defective colour vision in patients attending B.P Koirala Lions Centre for Ophthalmic Studies, a tertiary eye centre at Kathmandu. A total of 692 subjects case sheets were reviewed who had undergone colour vision test for (driving license/ national or foreign study/employment/or for ophthalmological disease evaluation) and were included in the study from September 2018 to April 2019. Colour vision was assessed using Fransworth D15 and Ishihara Pseudoisochromatic chart. Color vision scoring done was based on participants arrangement of caps which was categorized as blue-yellow, red-green or nonspecific color vision defects which follows Vingrys and King-Smith method[9] which is adapted by orthoptic department of BPKLCOS. It takes into account three factors- first confusion angle which identifies the type of color defect; the second is the Confusion index which quantifies the degree of color loss relative to a perfect arrangement of caps; and the third is the Selectivity index which quantifies the amount of polarity or lack of randomness in a cap arrangement. The case sheets were thoroughly reviewed for completeness of data. All the data were entered in the Excel spreadsheet and descriptive analysis was performed. Mean and standard deviation and percentage was calculated.

RESULTS

Out of 692 patients enrolled in the study,77.4% (n=520) were male and 22.6% (n=152) were female. The mean age of the subjects was 31.42±10.8 (12-61) years. Among them, 39.30% (n=272) patients were found to have colour vision defects out of which 83.45% (n=227) were male and 16.54% (n=45) were female. Congenital colour vision defect was found in 26.73%(n=139) males and 13.81%(n=21) females.

Table 1. Gender based distribution of colour vision defect

Type of colour vision defect	Total number	Male	Female
Congenital	160 (23.12%)	139 (26.73%)	21 (13.81%)
Acquired	112 (16.18%)	88 (16.92%)	24 (15.78%)

Acquired color vision defect was found in 16.92% (n=88) males and 15.78% (n=24) females (Table 1). Mean spherical equivalent ranged from -3.25D to +1.50D. Out of 296 (44%) patients having refractive error, 26.58% (n=184) of them were able to get the best corrected visual acuity of 6/6 bilaterally. Whereas, there was no improvement in visual acuity in 16.18% (n=112) of the subjects. Majority of patients with congenital colour vision defect were in the second (29.41%) followed by third (11.02%) and first (7.72%) decade of life. Similarly, majority of patients with acquired colour vision defect were in the fourth (11.76%) followed by second (9.56%) and third (7.72%) decade of life. (Table 2).

On the basis of ethnicity, Newars were found to have more colour vision defect (60.37%) followed by Brahmins (49.62%), Chhettris (37.25%), Magar (34.48%) and Lama (33.33%) (Table 3). When

Table 2. Distribution of age in patients with congenital and acquired colour vision defect

Age (in years)	Frequency (%) of Congenital CVD	Frequency (%) of Acquired CVD
10-20	21 (7.72%)	19 (6.98%)
20-30	80 (29.41%)	26 (9.56%)
30-40	30 (11.02%)	21(7.72%)
40-50	10 (3.67%)	32 (11.76%)
50-61	19 (6.98%)	14 (5.14%)

Table 3. Ethnic group distribution of congenital and acquired color vision patients

Ethnic group	Total number	Affected number		Percentage	
		Congenital	Acquired	Congenital	Acquired
Brahmin	264	177	64	29.17	24.24
Chhetri	102	22	16	21.56	15.68
Newar	53	20	12	37.73	22.64
Magar	58	10	7	17.24	12.06
Tamang	45	6	4	13.33	8.88
Gurung	32	5	3	15.62	9.37
Lama	37	5	3	13.51	8.10
Madhesi	60	10	8	16.67	13.33
Others	41	5	3	12.19	7.31

Table 4. Distribution of congenital and acquired colour vision defect

Color vision defect type	BE (congenital CVD)	BE (Acquired CVD)	RE (Acquired CVD)	LE (Acquired CVD)
Protan	62 (22.79%)	0	6 (2.20%)	0
Mild deutan	36 (13.23%)	0	0	0
Strong deutan	41 (15.07%)	0	6 (2.20%)	18 (6.61%)
Tritan	21 (7.72%)	6 (5.35%)	11 (4.04%)	20 (7.35%)
Non-specific	0	10 (8.92%)	17 (6.25%)	18 (6.61%)

congenital colour vision defect was observed, deutan colour vision defect (28.3%) was more prevalent than protan (22.79%) and tritan defect (7.72%). Whereas in acquired color vision defect, non-specific colour vision defect (8.92%) followed by tritan colour vision defect (5.35%) was more noted (Table 4).

The major causes for acquired CVD was papillitis (33.03%) followed by optic neuritis (16.07%) ,optic atrophy(14.28%) and disc pallor (10.71%). Other causes were, traumatic optic neuropathy, disc pallor, pituitary macroadenoma, chloroquin toxity, ethambutal induced toxicity, craniopharyngioma, myasthenia gravis, aplastic anemia, optic neuritis with 6th nerve palsy and retinitis pigmentosa (Table 5).

DISCUSSION

Color vision defects are frequently observed in ophthalmic practice. They are mostly genetic disorder. However, color vision defects can also be observed in various optic nerve ,retinal diseases as well as drug induced.

Previous studies have been done in color vision defect in Nepal but they mainly focused on school children or targeted population.[10,11] As congenital color vision defect is a genetic problem, its incidence does not have relationship with age. However, acquired color vision defect can be seen in varied age group and varied population. Our study included both the children and adults.

Ours was also a hospital-based study. A similar hospital-based study was carried out by Godar et al.[5] It was done in tertiary care hospital-based study in western Nepal where color vision was tested by Ishihara Pseudo isochromatic chart. They have found 74.72% males and 25.27% females in their study. Similar was found in other studies done in Nepal.[10,11] This was also seen in our study too with with male outnumbering females in ratio 2:1 in congenital type. The increased prevalence of congenital colour vision defect in male as comparison to females is due to x-linked recessive pattern of inheritance in males

Table 5. Distribution of causes of acquired color vision defect

Characteristics	Number (%)
Papillitis	37 (33.03)
Optic neuritis	18 (16.07)
Optic atrophy	16 (14.28)
Traumatic Optic Neuropathy	3 (2.67)
Disc pallor	12 (10.71)
Glaucoma	3 (2.67)
Pituitary macroadenoma	10 (8.92)
Chloroquin toxicity	4 (3.57)
Ethambutal induced toxicity	2 (1.78)
Craniopharyngioma	2 (1.78)
Myasthenia gravis	1 (0.89)
Aplastic anemia	1 (0.89)
Optic neuritis with 6th nerve palsy	1 (0.89)
Retinitis pigmentosa	2 (1.78)

and transmission by females.[12] Unlike congenital defects, acquired colour vision anomalies seemed evenly distributed between males and females. Overall, colour vision defect was found to be higher among males in our study, this could be attributed to males working in outdoors conditions more than females leading to change of color of crystalline lens from transparent to yellow which acts as filtering and affect short wavelength.[13] Like wise, the prevalence of CVD in European causacians, Chinese and Japanese ethnicity has been found to be higher among males.[14] However the prevalence of CVD (congenital) is much higher in our study as compared to other studies.[5,10,11] It might be because of purposive sampling carried out only in subjects who came for colour vision examination.

In our study, majority of patients with congenital colour vision defect were in the second (29.41%) followed by third (11.02%) and first (7.72%) decade of life. Whereas, majority of patients with acquired colour vision defect were in the fourth (11.76%) followed by second (9.56%) and third (7.72%) decade of life. In a study done by Godar et.al, colour vision defect was more prevalent in second decade (31.86%) of life followed by third decade (23.07%).[5] Colour vision defect was noticed later in life due to patients unawareness about the colour vision defect. Aware ness of color vision defect was also reported to be poor among majority study subjects of Ethopia.[15] Screening for CVD is important so that appropriate career advice can be given to these individuals, especially during the period when they are receiving their education rather than later life.[16]

In our study, colour vision defect was observed more in Newars followed by Brahmins and Chhettris. Similar finding was found in another study done in Nepal.[17] The results were contrary to the study done by Godar et.al, in which there was more prevalence of colour vision defect in Chhetri followed by Brahmin and Magar.5 More prevalence of colour vision defect in Newars in our study could be due to a greater number of Newar inhabitants (31.8%) in Kathmandu valley.[18] To find out exact incidence of color blindness among the different ethnic groups, a multicentric study with larger sample size is necessary.

When congenital colour vision defect was observed, deutan colour vision defect was more prevalent than protan and tritan defect which was similar to the other studies.[1,5,15] Whereas in acquired colour vision defect, non-specific colour vision defect followed by tritan (blue-yellow) colour vision defect was more noted. According to Kollner's rule, acquired blue yellow colour defects are the result of changes in the ocular media, choroid and diseases occurring in the outer retinal layers, whilst acquired red-green defects are the result of changes in the optic nerve and more inner parts of the visual pathway.[13] Also we had found various causes for acquired color vision defect as in a similar study conducted in Nepal.[5]

Observing the laterality in acquired colour vision defect, LE (20.57%) was found to be more defective than the RE (14.69%) and tritan defect (7.35%) was more observed than non-specific (6.61%), deutan (6.61%) and protan defect in LE. As acquired colour vision is mostly due to secondary cause, its incidence does not have relationship with eye laterality.

The major limitation of this study is that it was a retrospective study and hospital-based study. However, being a hospital-based study, varied causes could be isolated for acquired color vision defect in our study along with congenital type. In future, a multicentric prospective study could highlight the actual scenario of color vision defect in Nepal.

Findings of our study suggested that colour vision defect is important as it is a frequent finding and the detection and classification of acquired CVD should be done to look into various causes of it.

CONCLUSION

In our study, majority of those attending for color vision screening had normal color vision. When color vision defect was found, it was more prevalent in males than females for congenital type and in acquired type the proportion was almost same. Deutan color vision defect was common congenital color vision defect. In acquired color vision defect nonspecific color vision defect was common along with left eye involvement.

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CONFLICT OF INTEREST

The author(s) declare that they do not have any conflicts of interest with respect to the research, authorship, and/or publication of this article.

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