Original Article

Retina Amacrine cell study by flash Electroretinogram test in patients with color blindness

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ABSTRACT

Objective: this study tended to investigate retina amacrine by flash electroretinogram (FERG) test in patients with color blindness referred to Qods Clinic during 2016-2017. Materials and Methods: In this observational, cross-sectional descriptive study, 25 patients with color blindness referred to Qods Clinic in 2016-2017 were selected by convenient sampling and subjected to FERG. Voltage and latency were determined in the test and were compared with the observed findings in the control group. Results: The voltage recorded in ERG was 73.8±10.03 μV in the case group and 109.2±8.60 μV in the control group, which showed a significant difference between the two groups (P = 0.0001). The latency of ERG was 56.4±6.05 ms in the case group and 43.9±1.86 ms in the control group, which showed a statistically significant difference between the two groups (P = 0.0001). Conclusion: Based on the results of this study, it can be concluded that the presence of color blindness causes a significant change in ERG, which is a decrease in voltage and increase in latency.

Keywords: electroretinogram, color blindness, flash stimulus.

Introduction

Color blindness or insensitivity of vision system to color is a problem that affects about 2.3-2.7% of the general population [1, 2], and interestingly, this disorder is more common in industrial societies, accounting for 4% of reported prevalence [2]. This disorder has a genetic aspect, cardiovascular diseases, hyperlipidemia,and is most commonly seen in some families and races; however, it should not be forgotten about the role of environmental factors such as occupational injuries and acquired diseases, such as glaucoma and cataracte, which cause color blindness [3-5]. Retina is the light sensitive part of the eye that contains cones and cylinders. Cones are responsible for color vision and cylinders are primarily responsible for vision in dark. When cylinders and cones are stimulated, messages are sent through sequential neurons of the retina itself and optic nerve fibers that ultimately reach the cortex [6]. Black pigment melanin in the pigmentation layer prevents light reflection inside the sphere of the eye; this is important for clear vision. This pigment does the same thing as black color inside the camera film compartment. Light beams will be reflected in all directions in the eye in the absence of melanin; the whole retina will be lit instead of natural contrast between the dark and bright spots that are needed to create accurate images. We recognize the importance of melanin in the choroid pigmentation layer when it comes to the effects of its absence in "albinism", which inherit no melanin in all parts of the body. Pigmentation layer also stores large amount of vitamin A; this vitamin A is exchanged in and out in membrane of the outer cylinders and cones that are buried in the pigmentation layer. The cylinders and cones contain chemicals that are decomposed in exposure to light through which the nerve fibers which come out of the eye are stimulated. The cylindrical chemical is called rhodopsin; the light-sensitive chemicals in the cones, which are slightly different from rhodopsin, are called cone pigments [7].

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Destruction of the entire optic nerve causes blindness of the affected eye. Destruction of optic chiasm prevents the passage of impulses from nasal side halves of two retinas towards vision routes of the other side. Thus, nasal side half of both retina is blind. In other words, both temporal vision fields are blind, because image of the vision field falls inversely on the retina; this is called bitemporal hemianopsia. These lesions are often due to pituitary gland tumors which push up optic chiasm [8].

Although various methods are used for diagnosing color blindness disorder, especially in early-stage patients, methods with higher diagnostic accuracy can allow faster diagnosis of this disorder, prevent its progress, and improve performance of the patients [9].

One of these methods and electrophysiologic tests is electroretinogram (ERG), which allows objective evaluation of visual function in people with color blindness [10]. ERG is an electrical activity or electrical potential derived from the retina which results from its optical stimulation. ERG measures integrity of the retinal layer. In other words, ERG, which is obtained from retina after stimulation, is one of the electrophysiologic methods for diagnosing retinal functional disorders. This technique is particularly suitable for tracking diseases associated with retinal receptors and retinal pigment epithelium which reduces response [11].

Stimulation or optical flash is wide and creates the acting potential and adds it the eye resting potential. To record ERG, an electrode is connected to contact lens on the cornea and normal saline solution is used to fill its distance with cornea and another electrode is placed on the forehead. The retina is then simulated by light and a small voltage is generated that is reflected after amplification on oscilloscope screen and the oscilloscopic image is taken. The retinal stimulation is scientifically in two modes, one after getting used to scotopic darkness and one after getting used to photopic light. After initiating optical stimulation, a latency phase is initially followed by a negative wave called wave a. The wave a represents the stimulation of optical receptors (cylindrical and conical cells). After wave a, a positive wave, called wave b, is produced [12].

Therefore, this study tends to evaluate the performance of retinal amacrine cells by flash ERG in patients with color blindness referred to Qods Clinic in 2016-2017.

Materials and Methods

In this observational, cross-sectional descriptive study, 25 patients with color blindness referred to the Qods Clinic in 2016-2017 were selected by convenient sampling and subjected to FERG. Voltage and latency were determined and were compared with the observed findings in the control group. Data was collected by field method using ERG and data collection form. Finally, the collected data from all the subjects studied was analyzed using SPSS version 13 software. Mean and standard deviation were reported for latency and voltage. The test used in this study was independent t-test ($p<0.05$).

Results

Table 1: distribution of frequency of the voltage recorded in ERG

	Group	Mean	Std. Deviation
Amplitude	Case	7384	10.032
	Control	109.20	8.607

According to Table 1, the voltage recorded in ERG was 73.8±10.03 μV in the case group and 109.2±8.60 μV in the control group, which indicated a significant difference between two groups (P=0.0001).

Table 2: distribution of frequency of the latency recorded in ERG

	Group	Mean	Std. Deviation
Latency	Case	56.40	6.055
	Control	43.92	1.869

According to Table 2, the latency recorded in ERG was 56.4±6.05 ms in the case group and 43.9±1.86 ms in the control group, which indicated a significant difference between two groups (P=0.0001).

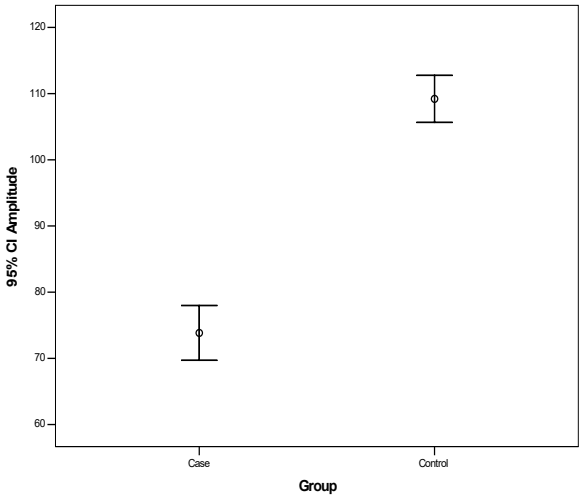

Figure 1: distribution of frequency of the voltage recorded in ERG

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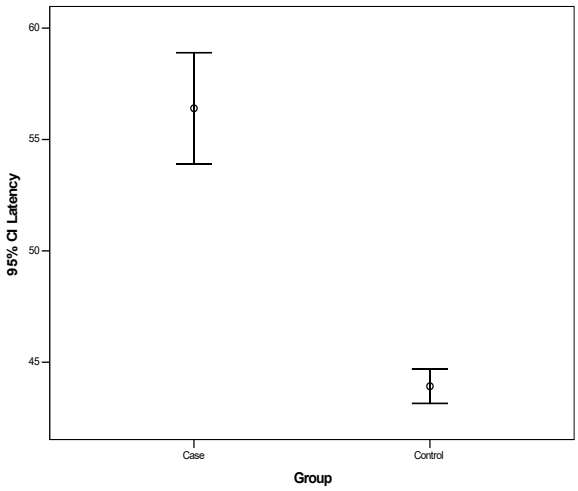

Figure 2: distribution of frequency of the latency recorded in ERG

According to Table 2, the latency recorded in ERG was 56.4±6.05 ms in the case group and 43.9±1.86 ms in the control group, which indicated a significant difference between two groups (P=0.0001).

Discussion

Although various methods are used for diagnosing color blindness disorder, methods with higher diagnostic accuracy can allow faster diagnosis of this disorder, prevent its progress, and improve performance of the patients [9]. One of these methods and electrophysiologic tests is ERG, which allows objective evaluation of visual function in people with color blindness. Thus, this study tended to examine performance of the retina amacrine cell by FERG in people with color blindness referring to the Qods Clinic during 2016-2017.

In this cross-sectional study, the voltage recorded in ERG was 73.8±10.03 μV in the case group and 109.2±8.60 μV in the control group, which indicated a significant difference between two groups (P=0.0001). the latency recorded in ERG was 56.4±6.05 ms in the case group and 43.9±1.86 ms in the control group, which indicated a significant difference between two groups (P=0.0001). In a study by Ziemssen et al. (2008) conducted in Germany, it was asserted that ERG could be used to evaluate the performance of the retina amacrine cell in patients treated with Avastin for evaluation of drug side effects; in cases color-blinded by drug effects, up to 90% of cases were affected [13], which was consistent with our findings that latency was longer in color blindness cases and voltage was reduced. In a study conducted by Pojda et al (2004) in Poland, it was reported that the voltage recorded in the ERG decreased in families with genetic disorders associated with color blindness and disorder in retinal amacrine cell function [14], which confirms our findings. In a study by Seeliger et al. (1999) in Germany, it was reported that ERG showed a decrease in voltage in patients with color blindness and disorder in retinal amacrine cell function [15], which is consistent with our study. In a study by Miyake et al. (1989) in Japan, it was stated that ERG would be almost normal in cases where retinal disorder was only Maculopathy and retinal amacrine cell was not involved [16]. This was not observed in our study. In a study by Krastel et al. (1979) in Germany, it was stated that ERG is a test that can be used to diagnose both nyctalopia and color blindness to evaluate the function of the retinal amacrine cell [17], which is similar to the results of our study.

Conclusion

Based on the results of this study and comparison with other studies, it is concluded that color blindness resulting in disorder in retinal amacrine cell caused a significant change in ERG in the form of a decrease in voltage and increase in latency. Finally, it is recommended that more studies be done in order to confirm the findings of this study in a multicentre form with higher sample size.

References

1. Gupta M, Gupta BP, Chauhan A, Bhardwaj A. Ocular morbidity prevalence among school children in Shimla, Himachal, North India. Indian J Ophthalmol. 2009 Mar-Apr;57(2):133-8.
2. Ceda-Flores RM, Arriaga-Ríos G, Muñoz-Campos J, Bautista-Peña VA, Angeles Rojas-Alvarado M, González-Quiróga G, et al. Frequency of color blindness and glucose-6-phosphate dehydrogenase enzyme deficiency in non-industrialized populations in the state of Nuevo León, México. Arch Invest Med (Mex). 1990 Jul-Sep;21(3):229-34.
3. Kohl S. Genetic causes of hereditary cone and cone-rod dystrophies. Ophthalmologe. 2009 Feb; 106(2):109-15.
4. Alipour-Parsa S, Haybar H, Namazi MH, Safi M, Khaheshi I, Memaryan M, et al. Evaluation of pentraxin-3 level and its related factors in patients undergoing primary percutaneous coronary intervention. ARYA Atheroscler [Internet]. 2017 Mar [cited 2019 Jan 20]; 13(2):73–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/29026413.
5. Jalali T, Eghbalnejad AM. Serum lipid profile and insulin resistance in women with polycystic ovary syndrome (PCOS). 2018; 5(3):107–11.
6. Halliday A. Evoked potential in clinical testing, 1st ed. Churchill Livingstone. New York. 1982
7. Whittakar S, Seigfried J. Origin of wavelets in the visual evoked potential. Electroencephalogr Clinical Neurophysio. 1983.
8. Bajalar AAA, Wrisght CE, vander Viliet VJ. Changes in the human VEP caused by the anticholinergic agent,

hyoshine hydrobromide. J Neural Neurosurgery Psychiatry. 1986; 49: 175-82.
9. Melamud A, Hagstrom S, Traboulsi E. Color vision testing. Ophthalmic Genet. 2004 Sep;25(3):159-87.
10. Kellner U, Kleine-Hartlage P, Foerster MH. Cone dystrophies: clinical and electrophysiological findings. Ger J Ophthalmol. 1992; 1(2):105-9.
11. Shoushtarian, M. M., 1997. evaluating normal values of amplitude and latency in electrical activity of retina or ERG. Journal of medical sciences, Issue 18, pp. 21-27.
12. Vaughan/Asburg/Riovdan- Eva: General ophthalmology. 15th edition, Appleton and Lange. 1999.
13. Ziemssen F, Lüke M, Messias A, Beutel J, Tatar O, Zrenner E, et al. Safety monitoring in bevacizumab (Avastin) treatment: retinal function assessed by psychophysical (visual fields, colour vision) and electrophysiological (ERG/EOG) tests in two subgroups of patients. Int Ophthalmol. 2008 Apr; 28(2):101-9.
14. Pojda-Wilczek D, Makowiecka-Obidzińska K, Herba E. Electroretinogram and electrooculogram in a family with Stargardt's disease. Klin Oczna. 2004; 106(3 Suppl):540-1.
15. Seeliger MW, Biesalski HK, Wissinger B, Gollnick H, Gielen S, Frank J, et al. Phenotype in retinol deficiency due to a hereditary defect in retinol binding protein synthesis. Invest Ophthalmol Vis Sci. 1999 Jan; 40(1):3-11.
16. Miyake Y, Shiroyama N, Horiguchi M, Saito A, Yagasaki K. Bull's-eye maculopathy and negative electroretinogram. Retina. 1989;9(3):210-5.
17. Krastel H, Jaeger W, Spiegelberg A. X-linked night-blindness: clinical and electrophysiological investigations including de-ERG in two families. Albrecht Von Graefes Arch Klin Exp Ophthalmol. 1979 Mar 5; 210(1):55-64.