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Binocular Vision Function and Migraine: A Review

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Abstract

Migraine is a multifactorial disease characterized by recurrent headaches and has complex neuronal and vascular mechanisms, including a wide range of clinical symptoms and other ocular findings. During migraine attacks in migraine sufferers, accommodative response increases significantly. Migraine sufferers are predisposed to have a subtle deficit in binocular vision coordination, reduced stereopsis, reduced fixation disparity, slightly higher prevalence of heterophoria, and reduced stereopsis. Convergence insufficiency can be associated with migraine and may be the persistent cause of reduced visual functioning. Binocular vision coordination difficulties are the most common findings among migraine sufferers.

Keywords: Migraine, Binocular Vision Anomalies, Headache

Introduction

The International Headache Society [IHS] defines migraine as a persistent headache that lasts for 4-72 hours (1-72 hours in children) and is frequently accompanied by nausea, vomiting, sensitivity to light, sound and movement. It is the most commonly encountered condition in medical and optometric practice. WHO has rated migraine among the most disabling illness affecting 15 to 20 percent of the world's population [1]. Migraine is the second most common headache disorder with prevalence of about 10 percent in older adults [2]. The prevalence and incidence of migraine depends upon age, sex, ethnic origin [3]. 25 percent of elder people experience unilateral migraine accompanied by symptoms like nausea, vomiting and sensitivity to light [4]. In females, the duration of migraine increases with age but in males migraine decreases from childhood to adulthood, with a prevalence of 20 percent in females and 10 percent in males [5,6,7]. Migraine with and without aura has a prevalence of 5-8 percent and 7-14 percent respectively [6]. Aura symptoms were equally common in both males and females [5].

The process that triggers migraine in the deep brain releases chemicals that induce pain and inflammation near the brain's nerves and blood vessels [7]. The trigeminovascular system is the anatomical and physiological base for nonciceptive transmission, which give rise to the sensation of migraine pain [8]. Activation and sensitization of first order trigeminovascular neurons leads to migraine headache and induce localized inflammatory response [9]. The cortical spreading depression, a self-propagating wave

of depolarization across the cerebral cortex is assumed to be the physiological cause of aura phase of the migraine. Cortical Spreading Depression is the fundamental cause of the migraine aura [10]. IHS classifies migraine into different groups; among these, migraine with aura and without aura is the most common [9].

Migraine attacks consist of four phases, premonitory phase characterized by symptoms such as irritability, euphoria, food cravings and thirst [11]. The second phase, aura phase is characterized by sensory or motor disturbances such as scintillating scotoma, micropsia, metamorphopsia, blind spot, fortification spectra (characteristic feature of aura phase), blurred vision, mosaic vision and visual field defects [12]. The headache phase is characterized by unilateral pulsating or throbbing headache of moderate or severe intensity. The fourth phase, resolution phase is characterized by tiredness, listless, impaired concentration and mood changes [13]. Migraine symptoms can be both non-visual and visual. Non-visual symptoms include nausea, headache, tiredness, and anorexia during the aura phase and headache phase. Image distortion, visual aura, blind spots, alterations in vision, and effects on binocular vision are all visual signs of migraine. The purpose of this review article is to explore the relationship between migraine and binocular vision anomalies.

Migraine and Binocular Vision Function:

Binocular vision is defined as state of simultaneous vision achieved by coordinated use of both eyes so that separate and dissimilar images of each eye is appreciated as single image. Grades of binocular vision includes simultaneous perception, fusion and stereopsis [14]. These components of binocular vision are affected in patients with migraine. Normal retinal correspondence is maintained during binocular single vision. It effectively stimulates the cortical process, which is primarily carried out by binocular fusion. Studies have demonstrated that there is a lateral inhibition in the visual cortex of migraine patient during migraine attacks which may lead to binocular vision dysfunction [15].

Accommodation is a natural adaptive mechanism by which eye changes its dioptric power to improve the retinal image of nearby object that occurs due to the contraction of ciliary muscle [14]. The kinetics of these adaptive reactions are dominated by the parasympathetic innervation of the ciliary muscle [16]. A prospective control study included 24 patients with episodic migraine and other 24 patients as control groups between the age group of 18-50 years, Objective accommodation, pupil size and ocular aberrations are measured. The result showed that during migraine phase, there was significant increase in accommodative response to accommodative stimuli. Study also concluded that accommodative response increased due to subtle oculo-sympathetic hypofunction during migraine attack [17].

Convergence is the disjugate movement of eyes in which both the eyes move inwards so that line of sight intersect with each other. It helps to maintain binocular single vision at all fixation distance [14]. Studies have demonstrated that convergence anomalies such as convergence insufficiency develops following a migraine attack. Migraine sufferers who had symptoms of blurred vision, dizziness, and severe headache had reduced convergence amplitude. Migraine episodes induced convergence insufficiency. Although the patient's headache decreased after receiving appropriate migraine directed therapy, the convergence insufficiency persisted necessitating orthoptic treatment. This implies that migraines may cause convergence insufficiency, but resolving migraine does not always result in resolving convergence

insufficiency. Convergence insufficiency is observed to be worsen with increased migraine frequency. Migraine worsens convergence insufficiency by causing the patient to avoid close tasks, and as a result, they develop atrophy of convergence capacities [18]. The mean near point of convergence is seen to be reduced in migraine patients. Migraine may cause not only convergence insufficiency but also worsens convergence insufficiency [19]. Convergence insufficiency were present in 16.25 percent and fusional vergence defeciencies in 11.25 percent of migraine individuals. This implies that refractive and binocular vision anomalies need to be principally investigated in all headache patients [20].

The Pathogenesis between migraine and binocular vision anomalies appears to be uncertain. Study have been done comparing migraine groups and control groups regarding clinical optometric binocular vision parameters [19]. The migraine headache variables and the binocular vision variables were examined. Binocular vision tests such as cover - uncover test, alternative cover test, aligning prism, randot stereopsis test, and convergence test were undertaken. The cover-uncover-investigation revealed heterophoria at 6 meters. An alternate cover test also revealed heterophoria in the migraine group. The Maddox rod test revealed heterophoria in migraine groups, and the difference between the migraine group and the control group was statistically significant [19].

Fixation disparity is a physiologic variant of binocular vision that exist when a minute image displacement occurs within the panum's area while fusion is maintained.[21]The migraine groups showed some degree of horizontal fixation disparity [19].

Stereopsis is defined as relative ordering of visual objects in depth, in three dimension [21]. Investigation from the randot test revealed few people with migraine had Stereopsis for less than 500 sec and most of the migraine sufferers had stereopsis for at least 20 sec. This concludes that the migraine group had reduced Stereopsis [19]. Basilar-type migraine causes binocular vision changes, including binocular blindness [22].

In ophthalmoplegic migraine, extraocular muscle palsy occurs following a hemicranial headache or at the end of the headache phase of the migraine [23]. Third nerve palsy is typically involved, with pupil and accommodation being affected [24]. Ocular muscles become weak and restricted, and they may also experience double vision during and after a migraine. A study states that one of the migraine sufferers experienced a muscular discomfort behind the right eye, sensitivity to light, and had diplopia and drooping eyelids. Neurological examination revealed adduction paralysis and ptosis on the right side, leading to strabismus. Proper differentiation and management are required to evaluate and diagnose the onset of strabismus with migraine [20].

Visual stimuli such as glare, stripes, or flickers can trigger migraine and headache. While viewing high contrast, three-dimensional, black-and-white striped patterns, migraine sufferers are more prone to experience illusions and distortions than non-migraine subjects [25]. It can also elicit discomfort. Studies have demonstrated that colors can decrease the experience of discomfort and reduce migraine frequency. Color, independent of luminance or contrast, provides therapeutic effect for people with visually triggered migraine as it can reduce the illusion when viewing strips or text [26].

The ability to perceive colors have been affected in patients with migraine. S-cones are activated by short wavelength(blue), migraine groups were restricted to perceive colors detected by S-cones. The migraine groups responded more slowly for the colors detected by S-cones [27].

CONCLUSION:

From this review it can be concluded that migraine subjects will have binocular vision deficiencies in terms of accommodation, convergence, stereopsis, fixation disparity and further studies are recommended and may help in the diagnosis and clinical practice.

Conflict of Interest

The Authors declare no Conflict of Interest related to this article.

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