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CASE REPORTS

Post-concussion Syndrome Light Sensitivity: A Case Report and Review of the Literature

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ABSTRACT

Photophobia is considered the second most common symptom of both concussion and post-concussion syndrome. Soldiers on duty experience photophobia after blast-related concussions or mild traumatic brain injury in 60–75% of instances. In addition, soldiers report other symptoms, such as asthenopia, squinting, dry eyes and headaches, for which they are considered to be at high risk. According to the International Brain Injury Association, some concussed patients report indirect symptoms such as multi-tasking difficulties, dizziness, vertigo, and fatigue. Moreover, some concussed individuals experience photophobia for approximately 6 months or indefinitely. We present the case of a 23-year-old soldier who presented with severe photophobia after a mild traumatic head injury. His photophobia was alleviated after the administration of topical anaesthetic drops in the eyes in the absence of any ocular surface pathology. He was diagnosed with post-concussion syndrome light sensitivity and was managed successfully with rose-coloured special photophobia glasses tinted with FL-41. Photophobia is a common neurological symptom in military personnel that needs more attention as it affects body and mind. We have reported an uncommon pathway of photophobia, which may unveil an unrecognised mechanism that may play a role in post-concussion photophobia.

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Introduction

Photophobia is defined as painful intolerance or discomfort due to exposure to light levels that are not usually unpleasant. It is considered the hallmark of ocular conditions such as uveitis and particular types of retinal dystrophies. Nevertheless, there is a suggestion that photophobia is nothing other than a functional symptom with no organic causes.[1]

Photophobia presents alongside many classes of headaches, such as migraine, tension headache, meningitis, and subarachnoid haemorrhage.[2,3] However, it may be present in conditions with no obvious association with headache or injury to the eyes, such as blepharospasm, trigeminal neuralgia, multiple sclerosis, traumatic brain injury (i.e. concussion) and other neuro-ophthalmological disorders.[4,5] The aetiologies of photophobia can be subdivided into: ocular; orbital and visual pathway pathology; neurological abnormalities; psychiatric disorders (depression, panic attacks); and certain drugs (e.g., barbiturates, benzodiazepines, chloroquine, methylphenidate, and zoledronate).[1,6,7]

Traumatic brain injury

Mild traumatic brain injury (mTBI) or concussion represents 70–90% of all documented brain injuries in which accidents and sports-related injuries are considered the most common causes, especially in athletes and military personnel. mTBI has a prevalence of nearly 600 per 100,000 individuals.[5,8,9] Headache, irritability, and dizziness are the most commonly reported symptoms shortly after head trauma. Symptoms such as photophobia, sound sensitivity, impaired cognitive function, anxiety, depression, and visual problems have also been reported by some individuals.[10] Although it can take up to 3 months for these symptoms to subside, some patients have impairment of cognitive function even for a prolonged period of time or indefinitely.[9,10] Moreover, the under diagnosis of mTBI is very common, as there are no objective methods available for its diagnosis.

Post-concussion syndrome can develop after head trauma, even after mTBI, due to neurophysiological influences and psychological factors that

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may exaggerate or increase the persistence of chronic symptoms due to stressful life situations or depression.[1]

Head trauma that induces a concussion or post-concussion syndrome is associated with various visual symptoms in 69–82% of patients.[11] Researchers suggest that trauma can lead to structural changes, irritation or injury to specific pain-sensitive areas in the brain that could be involved in increased light sensitivity and photophobia. Thus, the brain's ability to adjust to numerous different lights is diminished, which results in symptoms such as eye strain, headaches, and a lack of concentration.[1]

Although photophobia is caused mainly by light, some blind patients have been documented to have photophobia.[1] Furthermore, clinical observation shows that both post-traumatic headache and photophobia often occur together, which suggests a common pathway. The cardinal eye sensors are the rods and cones. A third type of photoreceptor, intrinsically photosensitive retinal ganglion cells, was discovered recently (ipRGCs). The ipRGCs are non-image-forming photosensors containing melanopsin that connect to and function by projecting light signals onto the olivary pretectal and specific thalamic nuclei involved in somatosensory and nociceptive perception, respectively.[1] Notably, this pathway is also activated in response to painful stimulation generated in the dura mater. Trauma may induce cortical injury, ocular vasodilation, or other dysfunction in any part of these pathways. These findings reflect a relationship between cortical damage and post-traumatic headache symptoms.[12] This integration of light and pain stimuli within these specific thalamic nuclei most likely accounts for light perception as a source of pain or photophobia.[4,13] Finally, intrinsically photosensitive melanopsin-containing cells have been discovered in non-retinal tissues such as the cornea, corneal nerve fibres, iris and trigeminal nerve branches.[5,14,15]

Case presentation

A 23-year-old male soldier with no known medical conditions presented with a 2 week history of severe bilateral photophobia with lacrimation following a minor head injury caused by a wooden box falling on his head 1 month prior to the presentation. This condition prevented him from working. At presentation, he covered his eyes with both hands over the top of a pair of sunglasses. His photophobia subsided following the application of a topical anaesthetic (benoxinate hydrochloride 0.4%) to both eyes. His best-corrected visual acuity was 20/20 and he had normal ocular surfaces. Fluorescein staining of the cornea and conjunctiva was normal and did not show any scratches or foreign bodies. Eversion of the upper eye lids did not show any dry eye or foreign bodies. The remainder of the ocular examination was within normal limits in both eyes. He was instructed to wait until the topical anaesthetic wore off, as there was no ocular finding that might account for his acute light sensitivity. The photophobia returned 20 minutes later. To determine if the eyes were dry, topical sodium hyaluronate (0.18% VISMED®) eye drops were applied. However, the photophobia persisted. Re-application of the topical anaesthetic immediately alleviated the photophobia.

As a result, a more detailed history of neurological symptoms including facial pain or tingling associated with trigeminal neuropathy, concussion, loss of consciousness, vertigo, difficulty multi-tasking, and cognitive dysfunction, was taken in addition to a comprehensive neurological and ophthalmological examination, which revealed no abnormal results.

The application of dark coloured filters (as used in Low-Vision Aids testing) resulted in near-complete relief of photophobia. This result confirmed our suspicions regarding post-concussion syndrome-related light sensitivity and left us perplexed about the action of topical anaesthetics on photophobia.

In addition to lubricant eye drops, special rose-coloured FL-41 glasses were prescribed, and he was referred to a neurologist for further evaluation. He had a normal neurological examination and magnetic resonance imaging (MRI) of his brain revealed no pathological changes, confirming the diagnosis of post-concussion syndrome with light sensitivity.

He returned to work wearing FL-41 spectacles. Additionally, he had monthly follow-up consultations to monitor his symptoms and his medical officer reported that his symptoms improved substantially during a 4 month follow-up period.

Discussion

Pathways of photophobia and headaches

Photophobia is strongly connected to pain and discomfort in the eyes. The trigeminal nuclei and nerves are considered the main moderators of pain sensation in the head and eyes. The trigeminal ganglion's afferent ophthalmic (V1) branch is responsible for transmitting pain signals of several eye structures, including the cornea, conjunctiva, sclera, and uvea. These structures are richly innervated with nerve fibres and immensely sensitive to pain. Additionally, the optic nerve carries trigeminal afferents within the blood vessels and the dura, which can explain why arteritic anterior ischaemic optic neuropathy and optic neuritis cause pain.[1]

The majority of orbital structures are sensitive to pain since the extraocular muscles contain nociceptive afferents that run close to the third, fourth, and sixth cranial nerves, which explains why myositis is painful. Additionally, the fact that orbital blood vessels are innervated by the trigeminal nerve explains the pain associated with orbital inflammation.[1]

Trigeminal ganglion neurons are both autonomic and sensory. The trigeminovascular reflex regulates the dilatation of innervated vessels by mediators, such as calcitonin gene-related peptide (CGRP) and nitric oxide, which are released after being activated by nociceptive sensory stimuli. The activation of superior salivatory and Edinger-Westphal nuclei by collaterals from the caudal trigeminal nucleus is known as the trigeminal-autonomic reflex, which is considered a multi-synaptic reflex. Parasympathetic activity in the pterygopalatine ganglion is activated by superior salivatory signals that dilate blood vessels and mediate lacrimation via the ciliary ganglion, in contrast to pupillary constriction, which is mediated by Edinger-Westphal output. These two reflexes explain the injection of conjunctival vessels and excessive tearing along with periorbital pain in patients with migraine or cluster headaches, which mostly occur with photophobia.[1]

Sympathetic efferents (via the short and long ciliary nerves) reach the orbital structures and carry innervation to the ocular and orbital blood vessels and pupils, respectively. Furthermore, the ophthalmic division of the trigeminal nerve via the long ciliary nerves innervates the cornea.[4] The long ciliary branches of the nasociliary nerve also innervate the bulbar conjunctiva. The superior palpebral conjunctiva is innervated by both the frontal and lacrimal branches of the ophthalmic division, while the inferior palpebral conjunctiva is innervated by both the infraorbital branch of the maxillary nerve and the lacrimal branch of the ophthalmic nerve.[16] When the superior cervical ganglion is stimulated, pain results.[1]

Post-traumatic headache

Photophobia is considered one of the most commonly encountered symptoms of concussion and post-concussion syndrome. There is a suggestion that approximately 43% of individuals experience photophobia after even mild head trauma. Furthermore, the majority of patients rate their photosensitivity as severe.[1] Other common symptoms of mTBI and concussion light sensitivity may include asthenopia, squinting and headaches. Photophobia is the most severe 1 to 3 weeks following trauma. However, light sensitivity may last up to 6 months or years after head trauma.[1]

Post-traumatic headache is associated with a number of distinct and overlapping pathophysiologies. Injuries to the central nervous system's microglia result in the inflammatory release of toxic or degradative substances that harm normal cells. Furthermore, disruption of the blood-brain barrier can amplify these effects. Increased levels of CGRP were associated with photophobia in one mouse model study, while the administration of CGRP antagonists relieved the symptoms.[12] In mTBI, the same model displayed a selective increase of nociceptive responses to cranial structures. This demonstrates that post-traumatic headache may originate from peripheral pain pathways.[12] Nonetheless, trauma can cause structural damage along pain pathways, which may account for the central origin of pain, as seen by MRI findings of white matter injury in migraine patients.[8]

Moreover, there is another explanation for post-traumatic headache. This involves the role of excess glutamate release, which can result in receptor

overstimulation, leading to ion imbalances and a worsening of symptoms.[17] What is interesting is that genetic factors appear to play a role in this condition. Carriers of the calcium voltage-gated channel subunit alpha1 A gene mutation may complain of severe symptoms even after mTBI.[12]

Light wavelength effect on photophobia

Photophobia is strongly affected by the wavelength of light. Amini, et al. demonstrated that patients with migraines were more annoyed by shorter wavelength (blue) light than by longer wavelength (red) light in comparison to those with tension headaches or controls.[9] Another study revealed that when the wavelength of the light stimulus was changed, visually evoked beta brain activity demonstrated a significant difference in cortical activity. The results indicated that blue light enhanced and red light suppressed migraine symptoms in patients.[14] The reasons for these effects were unclear. Schmidt, et al. found that ipRGCs were preferentially sensitive to blue light.[18]

Fluorescent lights are the main source of bothersome light. This may be due to the invisible flicker induced by fluorescent bulbs that is unrecognisable to the eye but can be received by the brain. Additionally, fluorescent lights as well as daylight have a significant amount of blue light, which is considered the most uncomfortable wavelength for individuals with photophobia and plays a role in worsening concussion-related symptoms.[4] Patients with persistent photophobia after 6 months of head injury or mTBI showed a reduced tolerance for light and sound. This is another explanation why these patients find the light brighter and more painful than non-injured individuals.[11]

Optical filters have been utilised in the treatment of photophobia. Rose-coloured tinted lenses, referred to as FL-41 tinted lenses, were found to successfully reduce migraine frequency in over 50% of children.[1] These glasses effectively filter out the noxious blue-green light emitted by fluorescent lighting in the visible light spectrum.[1] However, these glasses have also been shown to be effective at ameliorating photophobia associated with a variety of other conditions, including migraine, light-triggered seizures, benign essential blepharospasm, and digital eye strain.[1] Tinted lenses have also been shown to benefit concussion-related photosensitivity. Clark, et al. (2017) concluded that approximately 85% of photophobic patients reported some improvement in their symptoms following the use of tinted lenses.[3] The soldier in this case report was diagnosed with light sensitivity due to post-concussion syndrome and was successfully treated with FL-41 tinted glasses.

Dry eye as a consequence of mTBI

Lee, et al. reported that patients with mTBI have a higher prevalence of dry eye disease and pain disorders, indicating a common underlying pathophysiology.[7] Golar, et al. found that patients with dry eyes and photophobia report more intense symptoms than those with the dry eyes only. Hence, the treatment of dryness helps repair the damaged ocular surface and could help to decrease photophobia.[4,19]

Similarly, other studies have shown benefits of using medications for neuropathic pain and photophobia, such as alpha 2 delta ligand anti-epileptics (e.g., gabapentin) as the first line of treatment and serotonin-norepinephrine re-uptake inhibitors (e.g., duloxetine) and tricyclic antidepressants (e.g., nortriptyline) as second and third-line agents, respectively. In cases of severe eye pain, or when other treatments have failed, opioids (e.g., buprenorphine) can be tried in addition to the previous agents in certain patients.[20] Lastly, aggressive measures such as sympathetic blocks of the superior cervical ganglion have shown significant results in reducing ocular pain and photophobia in refractory cases.[17]

Effect of anaesthesia on photophobia pathways

Regarding this case, we believe that the topical anaesthetic drops likely altered two pathways: the trigeminal pathway via the corneal sensory fibres and the melanopsin-containing cells in non-retinal tissues such as the cornea, corneal nerve fibres, iris and trigeminal nerve branches.[5,15] As a consequence, one of the photophobia pathways was blocked. Although this finding contradicts a study by Lei, et al.,

who reported that ocular topical anaesthetics have little effect on blue or red light-induced pain thresholds, in normal subjects, it indicates that melanopsin-containing ophthalmic trigeminal ganglion cells could play a role in mediating light-induced discomfort.[18] Our findings raise the possibility that trauma disrupts the normal physiological pathways in these cells, thereby mediating photophobia. Furthermore, the current findings do not rule out the possibility that melanopsin-containing ocular trigeminal ganglion cells could contribute to photophobia in pathological conditions.[18] This significant observation lays the groundwork for future research into this subgroup of patients with post-concussion syndrome who exhibit light sensitivity.

Management of photophobia

Photophobia is regarded to be among the most challenging neuro-ophthalmological disorders to manage since there are no large randomised controlled trials to guide management.[1] Dark-coloured and tinted lenses for photosensitivity related to concussion have been tested, with 85% of patients reporting improvement.[3] Additionally, some behavioural adjustments, such as using polarised sunglasses, anti-glare covers for electronic devices, and non-liquid crystal display displays, may help alleviate light sensitivity.[21] Sunglasses may reduce photophobia, but their usage indoors is discouraged since they will worsen dark adaptation. Tinted lenses, such as FL-41, filter out light at the 480 nm wavelength, which has been shown to benefit a large number of patients since this is the wavelength at which ipRGCs are most sensitive.[19] In addition, the frequent use of topical ocular lubricants is advised.[16]

Conclusion

Photophobia has a variety of causes that are not confined to eye disorders, a fact that should prompt ophthalmologists to think about other aetiologies and to obtain a proper history from patients, especially for psychiatric and neurological symptoms, in addition to head injuries and medications. Moreover, the absence of the cardinal neurological symptoms of concussion, such as headache or loss of consciousness, does not exclude photophobia, as some patients may suffer from only excessive light sensitivity weeks after mTBI. Photophobia, in conjunction with a history of head trauma, even mild trauma, should raise the possibility of post-concussion syndrome light sensitivity. What made this case noteworthy was the alleviation of photophobia after using anaesthetic eye drops. Therefore, this observation may provide evidence of an undiscovered mechanism that is either directly or indirectly linked to the light hypersensitivity of the cornea.

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Consent for publication

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