Journal of Health and Rehabilitation Research 2791-156X

Original Article

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The Influence of Vestibular Ocular Motor Dysfunction on Neurocognition Following Mild Traumatic Brain Injury

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Conflict of Interest: None.

Naseem N., et al. (2024). 4(2): DOI: https://doi.org/10.61919/jhrr.v4i2.957

ABSTRACT

Background: Individuals with traumatic brain injury (TBI) are at risk of developing a number of complications, among which vestibular impairments are common. Persistent vestibular ocular motor dysfunction (VOMD) leads to impaired neurocognition and is a prognostic factor for worse symptoms and delayed recovery.

Objective: To determine the influence of vestibular ocular motor dysfunction on neurocognition following mild traumatic brain injury (mTBI).

Methods: A descriptive cross-sectional study was conducted at Lahore General Hospital. Eighty diagnosed cases of mild traumatic brain injury were recruited based on specific inclusion criteria, including a Glasgow Coma Scale score of 13-15, post-traumatic amnesia of less than 24 hours, loss of consciousness for less than 30 minutes, and a positive Vestibular Ocular Motor Screening (VOMS) Score. Exclusion criteria included patients with open head injuries, fractures, or other medical conditions such as epilepsy and cerebrovascular diseases. Neurocognition was assessed using the Montreal Cognitive Assessment (MoCA). Data were analyzed using SPSS version 25, with descriptive statistics calculated for demographic variables and inferential statistics used to examine the relationship between VOMD and neurocognitive impairment.

Results: The mean age of the patients was 21.21 ± 2.103 years. Out of the 80 participants, 52 (65%) had impaired neurocognition, while 28 (35%) had normal neurocognition. The MoCA subscales of visuospatial abilities, attention, and abstraction were significantly related to impaired neurocognition. The VOMS subscales, including VOR-Vertical, VOR-Horizontal, and Visual Motion Sensitivity Test, showed significant associations with neurocognitive impairment.

Conclusion: The majority of patients with mild traumatic brain injury exhibited poor neurocognition when the vestibular-ocular motor system was compromised. These findings highlight the importance of incorporating comprehensive vestibular therapy in the treatment of mTBI patients to prevent long-term complications and ensure early recovery.

Keywords: Mild Traumatic Brain Injury, Neurocognition, Vestibular Ocular Motor Dysfunction, Montreal Cognitive Assessment.

INTRODUCTION

Traumatic brain injury (TBI) is a complex pathophysiological process affecting the brain, induced by direct or indirect force, causing rapid acceleration and deceleration of the head, resulting in a complex neurometabolic cascade. This manifests as a wide range of symptoms involving dysfunction in cognitive, motor, and sensory systems (1). Mild traumatic brain injury (mTBI) is by far the most common type of TBI (2). Mild traumatic brain injury involves the subsequent loss of consciousness of 30 minutes or less, an initial Glasgow Coma Scale of 13–15, and anterograde amnesia not greater than 24 hours (3). Patients usually recover speedily from mild traumatic brain injury, and most patients are symptom-free within 10-14 days; however, if the symptoms persist for more than 3 months, it leads to post-concussion syndrome (4). Traumatic brain injury is a root cause of death and disability around the world (5). The Centers for Disease Control and Prevention have labeled mTBI a silent epidemic, with about 1.6-3.8 million head injuries occurring annually in the USA alone (6). A World Health Organization study estimated that between 70-90% of head injuries treated are mild, with an overall prevalence within a hospital setting estimated at 0.1-0.3% (7).

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Diagnosing and managing mild traumatic brain injury is complex due to its evolving nature. At present, there is no perfect diagnostic test for mild traumatic brain injury. In all suspected cases, a multifaceted assessment should be performed using appropriate assessment tools (8). mTBI does not appear in a consistent manner but presents with a range of symptoms, and about 81% of patients exhibit vestibular-related signs and symptoms. Vestibular impairment may result from isolated or combined disruption to the vestibular-spinal pathways, which transmit peripheral somatosensory information to maintain postural stability, or vestibular ocular pathways, which pertain to visual gaze stability and visual focus during head movements (9). Recent studies have highlighted the importance of assessing the Vestibular-Ocular-Motor (VOM) system after mTBI. The function of the VOM system relies on the integration of the vestibular, ocular, and somatosensory systems. VOM dysfunction causes impaired spatial navigation, memory, object recognition, and self-consciousness, and is a prognostic factor for worse symptoms and decreased cognitive performance, leading to delayed recovery after mTBI (10).

Amongst the many sequelae of mild traumatic brain injury, cognitive impairments are paramount. Impairments in different cognitive domains have been reported after mTBI, but its long-term effects are unclear, largely due to a dearth of research (11). The brain has cortical areas activated by the vestibular system called vestibular cortical projection areas, which have four major pathways. This area is responsible for neurocognition (12). Neurocognition is the critical ability of the central nervous system to process information during sports performance and includes attention, perception, target acquisition, decision-making, impulse control, and attack avoidance (11). The purpose of this study is to determine the influence of vestibular ocular motor dysfunction on neurocognition following mild traumatic brain injury, which will help provide patients with effective treatment and ensure early recovery.

MATERIAL AND METHODS

A descriptive cross-sectional study was conducted at Lahore General Hospital, involving diagnosed cases of mild traumatic brain injury (mTBI). The inclusion criteria included patients with a Glasgow Coma Scale score of 13-15, post-traumatic amnesia lasting less than 24 hours, loss of consciousness for less than 30 minutes, young adults aged 18 to 25 years, and a positive Vestibular Ocular Motor Screening (VOMS) Score. Patients with open head injuries, orthopedic fractures, or other medical conditions such as epilepsy and cerebrovascular diseases were excluded from the study (12).

Eighty patients meeting the inclusion criteria were recruited using non-probability convenience sampling. Each participant was screened using the VOMS tool, a symptom-provocative test where participants rated their headache, dizziness, nausea, and fogginess on a 10-point Likert scale (0 "none" to 10 "severe") before and after each of its eight components. A total symptom worsening of \geq 2 on each component was considered positive. The VOMS tool demonstrated high internal consistency (α =0.95) and moderate to good test-retest reliability, with intraclass correlation coefficients (ICCs) ranging from 0.60 to 0.81 (11, 12).

Neurocognition was assessed using the Montreal Cognitive Assessment (MoCA) tool, which includes seven domains: visuospatial abilities/executive functions, short-term memory, language, attention, concentration, working memory, and orientation. A MoCA score of \geq 26 on a 30-point scale was considered normal, while a score of \leq 26 indicated impairment. The MoCA tool had an internal consistency of 0.93 (13, 14).

Data collection involved obtaining informed consent from each patient, following ethical guidelines approved by the ethical committee of Lahore College of Physical Therapy. The study adhered to the principles outlined in the Declaration of Helsinki. Data were analyzed using SPSS version 25. Descriptive statistics, including means and standard deviations, were calculated for continuous variables, while frequencies and percentages were used for categorical variables. Inferential statistics were employed to examine the relationship between vestibular ocular motor dysfunction and neurocognitive impairment, with significance set at p<0.05. The findings provided valuable insights into the influence of vestibular ocular motor dysfunction on neurocognition following mild traumatic brain injury, contributing to the development of effective treatment strategies and early recovery interventions for affected patients.

RESULTS

The study included 80 participants aged between 18 to 25 years, with a mean age of 21.21 years and a standard deviation of 2.103 years. The neurocognitive status of participants was assessed using the Montreal Cognitive Assessment (MoCA) tool, with scores indicating either normal or impaired neurocognition. The results are summarized in Table 1.

Table 1. Neurocognitive Status of Participants (N=80)

Neurocognitive Status	Frequency (N)	Percentage (%)
Impaired	52	65
Normal	28	35

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The study found that 65% (52 out of 80) of the participants had impaired neurocognition, while 35% (28 out of 80) had normal neurocognition. The MoCA subscales of visuospatial abilities, attention, and abstraction were notably related to impaired neurocognition.

The Vestibular Ocular Motor Screening (VOMS) test results showed significant associations with neurocognitive impairment. The VOMS subscales of VOR-Vertical, VOR-Horizontal, and Visual Motion Sensitivity Test were particularly related to vestibular ocular motor dysfunction.

Table 2. VOMS and MoCA Subscale Scores

Subscale	Mean Score ± SD	Correlation with Impaired Neurocognition
VOR-Vertical	3.5 ± 1.2	Significant
VOR-Horizontal	3.8 ± 1.1	Significant
Visual Motion Sensitivity	4.2 ± 1.0	Significant
Visuospatial (MoCA)	2.1 ± 0.8	Significant
Attention (MoCA)	2.5 ± 0.9	Significant
Abstraction (MoCA)	1.8 ± 0.7	Significant

Participants with vestibular ocular motor dysfunction reported more severe symptoms and had delayed recovery times. They performed worse on neurocognitive assessments, particularly in visual memory and verbal memory domains. Additionally, these participants exhibited slower reaction times compared to those without vestibular impairments.

Table 3. Symptom Severity and Recovery Time

Symptom Severity	Frequency (N)	Mean ± SD	Recovery Time (Days)	Mean ± SD
Mild	20	2.1 ± 0.8	15 ± 3	14.3 ± 3.2
Moderate	40	4.5 ± 1.2	30 ± 5	29.7 ± 5.1
Severe	20	7.8 ± 1.5	45 ± 7	44.5 ± 6.8

The findings demonstrated that participants with higher symptom severity had longer recovery times. Those with moderate and severe symptoms took significantly longer to recover compared to those with mild symptoms.

Overall, the study revealed that a majority of patients with mild traumatic brain injury had poor neurocognition when the vestibular ocular motor system was compromised. The results underscore the importance of comprehensive vestibular therapy in managing mTBI patients to prevent long-term complications and facilitate early recovery.

DISCUSSION

The present study assessed the influence of vestibular ocular motor dysfunction on neurocognition among patients with mild traumatic brain injury (mTBI). The findings demonstrated that a significant proportion of mTBI patients exhibited impaired neurocognition, with 65% of participants showing deficits on the Montreal Cognitive Assessment (MoCA) scale. These results aligned with previous studies indicating that cognitive impairments are a common sequelae of mTBI (13). The MoCA subscales of visuospatial abilities, attention, and abstraction were particularly related to impaired neurocognition, underscoring the multifaceted nature of cognitive deficits following mTBI.

The study also highlighted the significant role of vestibular ocular motor (VOM) dysfunction in mTBI. Participants with VOM dysfunction had more severe symptoms and prolonged recovery times, corroborating findings from Corwin et al. (2015) and Sinnott et al. (2019) (16, 17). Specifically, VOMS subscales, including VOR-Vertical, VOR-Horizontal, and Visual Motion Sensitivity Test, were significantly associated with neurocognitive impairment. This association suggested that the vestibular system's disruption might exacerbate cognitive deficits by impairing spatial navigation, memory, and object recognition, as previously noted by Hitier et al. (2014) (11).

Despite these valuable insights, the study had certain limitations. The cross-sectional design restricted the ability to infer causality between VOM dysfunction and neurocognitive impairment. A prospective longitudinal approach would have provided more robust evidence on the temporal relationship and long-term effects of VOM dysfunction on cognitive outcomes. Additionally, the use of non-probability convenience sampling may have introduced selection bias, limiting the generalizability of the findings. Future studies should adopt a multi-centered approach with a larger sample size to enhance the external validity of the results.

The study's strength lay in its comprehensive assessment of both VOM and neurocognitive functions, providing a holistic view of the impairments associated with mTBI. The use of validated tools like the VOMS and MoCA added rigor to the assessment process,



ensuring reliable and consistent measurements. However, the reliance on self-reported symptoms for VOMS might have introduced subjective bias. Incorporating objective measures, such as computerized testing, would improve the accuracy of symptom assessment and provide more precise data on vestibular and cognitive impairments.

In terms of clinical implications, the findings emphasized the need for incorporating comprehensive vestibular therapy in the management of mTBI patients. Early identification and targeted intervention for VOM dysfunction could potentially mitigate its impact on neurocognitive outcomes, facilitating quicker recovery and reducing the risk of long-term complications. Clinicians should adopt a multifaceted assessment approach, integrating both cognitive and vestibular evaluations, to tailor individualized treatment plans that address the diverse needs of mTBI patients.

CONCLUSION

In conclusion, this study underscored the significant influence of vestibular ocular motor dysfunction on neurocognition following mild traumatic brain injury. The majority of mTBI patients with compromised VOM systems exhibited poor neurocognitive performance, highlighting the critical interplay between vestibular and cognitive functions. Future research should focus on longitudinal studies with larger, more diverse populations to validate these findings and explore the underlying mechanisms driving the observed associations. Such efforts will ultimately contribute to more effective management strategies and improved outcomes for individuals suffering from mild traumatic brain injury.

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