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Original Article

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Association of Cognitive Impairment with Clinical Variables in Migraine Patients

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ABSTRACT

Background: This study explores the relationship between migraine and cognitive impairment, aiming to recognize deficits during ictal and interictal phases. This study examines how migraine frequency and severity affect understanding and evaluates the competence of present treatments in reducing these impairments. Understanding these relations may help improve management approaches and improve the quality of life for migraine sufferers. This study aims to measure the prevalence of cognitive impairment in migraine patients and discover its association with clinical variables, including migraine severity, duration, psychological factors, and treatment outcomes.

Methods: This cross-sectional observational study included 100 migraine patients aged 18 to 60 years, diagnosed per ICHD-3 criteria. Participants underwent assessments using MoCA, MIDAS, and HADS scales, alongside serum vitamin B12 and TSH testing. Cognitive impairment was defined as a MoCA score <26. Data were analysed using SPSS v26 with appropriate statistical tests; $p<0.05$ was considered significant.

Results: Out of 97 migraine patients assessed, 19.6% showed cognitive impairment (MoCA<26). Females predominated (89.7%), and photophobia/phonophobia was universal ($p<0.001$). While 81.4% had low vitamin B12, no significant association was found with cognition ($p=0.72$). Cognitive status did not significantly correlate with gender, headache duration, migraine severity (MIDAS), depression, anxiety, or vitamin B12 levels ($p>0.05$). However, 66% showed cognitive improvement after 3 months of treatment ($p=0.002$), suggesting potential reversibility.

Conclusion: Cognitive impairment was present in 19.6% of migraine patients, with no significant correlation to clinical variables. Effective treatment improved cognition, highlighting the need for holistic migraine management.

Key-words: Migraine, Cognitive Impairment, MoCA, Migraine Disability (MIDAS), Neurological Assessment

INTRODUCTION

Migraine is a prevalent neurological disorder affecting approximately 1 billion people globally, and it remains one of the leading causes of disability, especially in young adults and middle-aged women. Traditionally, migraine has been conceptualized primarily as a disorder of pain; however, recent studies have shown that the impact of migraine extends beyond headache into the domain of cognitive functions [1]. Cognitive complaints reported by migraine sufferers include difficulty with concentration, memory lapses, decreased attention span, and mental fatigue both during migraine attacks (ictal phase) and in headache-free periods (interictal phase). These deficits significantly affect quality of life, occupational productivity, and social functioning [2].

Migraine is a complex neurological disorder characterized by recurrent episodes of headache, which can be moderate to severe in intensity. It is often accompanied by nausea, vomiting, photophobia, and phonophobia and can significantly impair the quality of life of sufferers. The prevalence of migraine varies globally, affecting approximately 1 billion individuals. It is

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more common in women than in men, and typically affects individuals between the ages of 25 and 55 years [1,2]. Recent research has begun to explore the cognitive aspects of migraine, moving beyond the traditional focus on pain. Migraine sufferers often report transient cognitive disturbances such as difficulty concentrating, memory lapses, and slowed thought processes during and between migraine episodes. These cognitive symptoms can be as disabling as the pain itself, impacting daily activities and occupational performance [3,4].

The pathophysiology of migraine is complex and involves multiple neurological pathways. It is hypothesised that migraine is related to dysfunctional cortical excitability and altered cerebral blood flow. The concept of cortical spreading depression (CSD) is central in understanding the mechanisms underlying migraine aura and possibly the headache phase itself. CSD refers to a wave of neuronal and glial depolarisation that travels across the cortex, leading to transient changes in neural activity that could affect cognitive functions [5,6].

Furthermore, chronic migraine sufferers might experience more pronounced cognitive deficits due to frequent and prolonged pain episodes, which can lead to structural changes in the brain. Studies using neuroimaging techniques have shown that migraineurs, particularly those with chronic forms, may have alterations in brain areas involved in pain processing and cognitive functions, such as the frontal and temporal lobes, and the thalamus [7,8].

Pathophysiological Mechanisms

The pathophysiology underlying cognitive impairment in migraine is complex and multifactorial. One of the primary mechanisms is Cortical Spreading Depression, a self-propagating wave of neuronal and glial depolarisation followed by a period of suppression of brain activity. CSD is not only responsible for migraine aura but is also hypothesised to contribute to transient cognitive dysfunction by altering neuronal excitability and affecting regions involved in memory and attention processing [9].

Additionally, neuroinflammation and oxidative stress play a significant role in migraine-associated cognitive changes. Animal model studies have demonstrated that repeated migraine episodes can lead to elevated levels of inflammatory cytokines and reactive oxygen species, causing both structural and functional brain alterations that impair cognition [10]. Structural neuroimaging studies have further shown that chronic migraine is associated with grey matter volume reductions, particularly in regions such as the prefrontal cortex, hippocampus, and temporal lobes, all of which are critical for cognitive processes including executive function and memory consolidation [11]. Emerging evidence also implicates the P2X7 receptor/NLRP3 inflammasome signaling pathway in migraine-related cognitive deficits. Activation of this pathway promotes neuroinflammatory responses, leading to neuronal dysfunction and contributing to the cognitive impairments observed in chronic migraine models [12].

The study aims to investigate the association between cognitive impairment and migraine. The objectives are to identify cognitive deficits associated with migraine in both the interictal and ictal phases, to assess the impact of migraine frequency and severity on cognitive functions, and to evaluate the effectiveness of current migraine treatments in mitigating cognitive impairments.

MATERIALS AND METHODS

Research Design- This study was designed as a cross-sectional observational study, conducted at the Department of Neurology, Himalayan Institute of Medical Sciences, Dehradun. The study period extended from January 2023 to December 2024. Ethical approval for the study was obtained from the Institutional Ethical Committee, and informed consent was collected from 97 patients before their inclusion in the study, ensuring adherence to ethical research practices. The study population consisted of patients diagnosed with migraine, with or without aura, who presented to the Neurology Outpatient Department during the study period. The diagnosis of migraine was established according to the criteria defined in the International Classification of Headache Disorders, 3rd Edition. A total of 100 participants were enrolled in the study. The sample size was calculated based on previous literature reporting a prevalence of cognitive impairment in migraine patients ranging between 15% to 25%, ensuring a 95% confidence level and 10% margin of error to provide adequate statistical power for detecting significant associations. The sample size was set at 100 participants based on preliminary estimates required to

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achieve adequate power for detecting significant cognitive differences.

Inclusion Criteria

- Age between 18 and 60 years.
- Diagnosed case of migraine (with or without aura) according to ICHD-3.
- Migraine history of at least 6 months.
- Patients capable of understanding and completing the cognitive and psychological assessment tools.

Exclusion Criteria

- Patients with other neurological disorders (e.g., stroke, epilepsy, multiple sclerosis).
- Individuals with psychiatric illnesses such as schizophrenia or bipolar disorder.
- Patients currently on psychotropic medications, anti-epileptics, or cognitive-enhancing drugs.
- History of head trauma or post-traumatic migraine.
- Presence of chronic systemic illnesses (e.g., chronic kidney disease, chronic liver disease, hypothyroidism, malignancy).
- Substance abuse or dependence history.

Study Procedure and Evaluation- Patients were evaluated using a structured questionnaire that included the Migraine Disability Assessment Score (MIDAS), the Hospital Anxiety and Depression Scale (HADS), and the Montreal Cognitive Assessment (MoCA). Blood tests included measurements of vitamin B12 and thyroid-stimulating hormone. Clinical and questionnaire data were processed along with laboratory results. All participants underwent a comprehensive clinical assessment using standardised tools. Cognitive function was evaluated using the Montreal Cognitive Assessment, a widely accepted screening tool for cognitive impairment. A MoCA score below 26 was considered indicative of mild cognitive impairment. To assess the severity and disability associated with migraine, the Migraine Disability Assessment Score was administered, categorising patients into various grades based on the level of disability. Psychological evaluation was performed using the Hospital Anxiety and Depression Scale to identify comorbid anxiety and depression, conditions known to influence cognitive performance and quality of life in migraine patients. In addition to clinical assessments, laboratory investigations were conducted, including measurement of serum Vitamin B12 levels and Thyroid Stimulating Hormone levels, to exclude other potential causes of cognitive dysfunction such as vitamin B12 deficiency or hypothyroidism.

Statistical analysis- The statistical analysis for this study was conducted using SPSS version 26. Descriptive statistics were applied to summarise the data, with means, standard deviations, medians, and interquartile ranges calculated for continuous variables, while frequencies and percentages were used for categorical variables. For comparative analysis, the Chi-square test or Fisher's exact test was employed to evaluate associations between categorical variables, such as the presence of cognitive impairment and migraine severity or gender.

RESULTS

Total of 97 participants were assessed after accounting for data completeness. The general characteristics of the study population are summarised in Table 1. A significant female predominance was observed, with 89.7% of the participants being female and only 10.3% male, reflecting the well-documented higher prevalence of migraine in women ($p<0.001$). Regarding headache duration, most patients (66%) reported headaches lasting less than one day, while 34% experienced longer-lasting headaches (>1 day), and this difference was statistically significant ($p=0.002$). Photophobia and phonophobia were universally present in the study population, further confirming their diagnostic relevance in migraine ($p<0.001$). When analysing cognitive status using the Montreal Cognitive Assessment (MoCA), 3.1% of patients exhibited low cognitive performance, 16.5% had mild cognitive impairment (MCI), and 80.4% had normal cognitive function ($p<0.001$). Additionally, 19 out of 97 participants (19.6%) were classified as having cognitive abnormalities ($p<0.001$). Evaluation using the MIDAS score revealed that the majority of patients (54.6%) fell into Grade II disability, 37.1% into Grade I, while 4.1% each were categorised into Grade III and IV. A significant proportion of the study population (81.4%) was found to have low serum vitamin B12 levels, which may be relevant to cognitive performance ($p<0.001$). Follow-up cognitive assessment after 3 months showed that 66% of participants improved in MoCA scores, whereas 34% showed no change, suggesting some

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reversibility of cognitive dysfunction with management (p=0.002).

Table 1: Baseline Characteristics of the Patients in this Study

Parameter	Sub-Parameter	Number of Patients	Total	Proportion	p-value
Gender	Male	10	97	0.10	<0.001
	Female	87	97	0.89	<0.001
Headache Duration	Less than one day	64	97	0.66	0.002
	More than one day	33	97	0.34	0.002
Photophobia	Yes	97	97	1	<0.001
Aura	No	90	97	0.92	<0.001
	Yes	7	97	0.07	<0.001
Cognition	Low	3	97	0.03	<0.001
	MCI	16	97	0.16	<0.001
	Normal	78	97	0.80	<0.001
Cognition Abnormality	No	78	97	0.80	<0.001
	Yes	19	97	0.19	<0.001
Midas	1	36	97	0.37	0.014
	2	53	97	0.54	<0.001
	3	4	97	0.04	<0.001
	4	4	97	0.04	<0.001
B12 Level	Low	79	97	0.81	<0.001
	High	18	97	0.18	<0.001
Moca After 3months	Improved	64	97	0.66	0.002
	Same	33	97	0.34	0.002

Note: H_a is proportion ≠ 0.5

Table 2 shows the correlation between cognitive impairment and gender distribution in this study. Among the total 97 patients, 10.3% (10 patients) were male, and 89.6% (87 patients) were female. Among the male participants, 90% (9 patients) did not experience cognitive impairment, and 10% (1 patient) had cognitive impairment. In contrast, among the female participants, 79.31% (69 patients) did not report cognitive impairment, while 20.68% (18 patients) had cognitive impairment. The overall percentage of participants without cognitive impairment was 80.4%, while 19.57% experienced cognitive impairment. The p-value of 0.420 indicates that there is no statistically significant relationship between cognitive impairment and gender distribution, as the p-value is greater than the commonly used threshold of 0.05.

Table 2: Correlation between cognitive impairment and gender distribution in this study

Cognitive Impairment	Males	Females	Total	p-value
Not present	9(90%)	69(79.31%)	78(80.4%)	0.42
Present	1(10%)	18(20.68%)	19(19.57%)	
Total	10(10.30%)	87(89.6%)	97(100%)	

Table 3 and Fig. 1 explore the correlation between cognitive impairment and various factors such as headache duration, depression, and vitamin B12 levels in migraine patients, with 97 participants in total. The p-value for the relationship between cognitive impairment and headache duration is 0.17, indicating no significant association.

Among those with headaches lasting >1 day, 84.3% had no cognitive impairment, while 15.6% did, compared to 72.7% and 27.27%, respectively, for those with headaches lasting <1 day. Regarding depression, the p-

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value is 0.82, suggesting no significant link with cognitive impairment, as 82.3% of participants with depression In comparison, 80% of those without depression had no impairment, and 20% did. Similarly, the p-value for vitamin B12 levels is 0.72, showing no significant correlation between cognitive impairment and vitamin B12 status, as 79.74% of participants with low vitamin B12 levels did not have cognitive impairment, and 83.33% of those with high vitamin B12 levels did not have impairment either.

Table 3: Correlation of Cognitive Impairment with other factors

Cognitive Impairment versus Headache				
Cognitive Impairment	**Headache Duration >1 Day**	**Headache Duration <1 Day**	**Total**	**p-value**
Not Present	54 (84.3%)	24 (72.7%)	78 (80.41%)	0.17
Present	10 (15.6%)	9 (27.27%)	19 (19.58%)	
Total	64	33	97	
Cognitive Impairment versus Depression				
Cognitive Impairment	Depression Present	Depression Absent	Total	p-value
Not Present	14(82.3%)	64(80%)	78(80.41%)	0.82
Present	3(17.6%)	16(20%)	19(19.58%)	
Total	17	80	97	
Cognitive Impairment versus Vitamin B12				
Cognitive Impairment	Vitamin B12 Low	Vitamin B12 High	Total	p-value
Not Present	63(79.74%)	15(83.33%)	78(80.41%)	0.72
Present	16(20.2%)	3(16.66%)	19(19.58%)	
Total	79	18	97	

Table 4 and Fig. 1 present the distribution of cognitive impairment across different levels of migraine severity, as measured by the MIDAS questionnaire. Among the 97 participants, 80.41% did not report cognitive impairment, with 19.58% reporting it. Cognitive impairment was present in 22.22% of participants in Grade I (mild), 16.98% in Grade II (moderate), 0% in Grade III (severe), and 50% in Grade IV (very severe). Despite these variations, the p-value of 0.294 indicates that there is no statistically significant correlation between cognitive impairment and the severity of migraine. This suggests that, based on the data, cognitive impairment does not appear to be strongly associated with migraine severity.

Table 4: Correlation Of Cognitive Impairment and Severity of Migraine (According to Midas Questionnaire)

Cognitive Impairment	**Midas (Grade-I)**	**Midas (Grade-II)**	**Midas (Grade-III)**	**Midas (Grade-IV)**	**Total**	**p-value**
Not present	28(77.77%)	44(83.01%)	4(100%)	2(50%)	78(80.41%)	0.294
Present	8(22.22%)	9(16.98%)	0(0%)	2(50%)	19(19.58%)	
Total	36	53	4	4	97	

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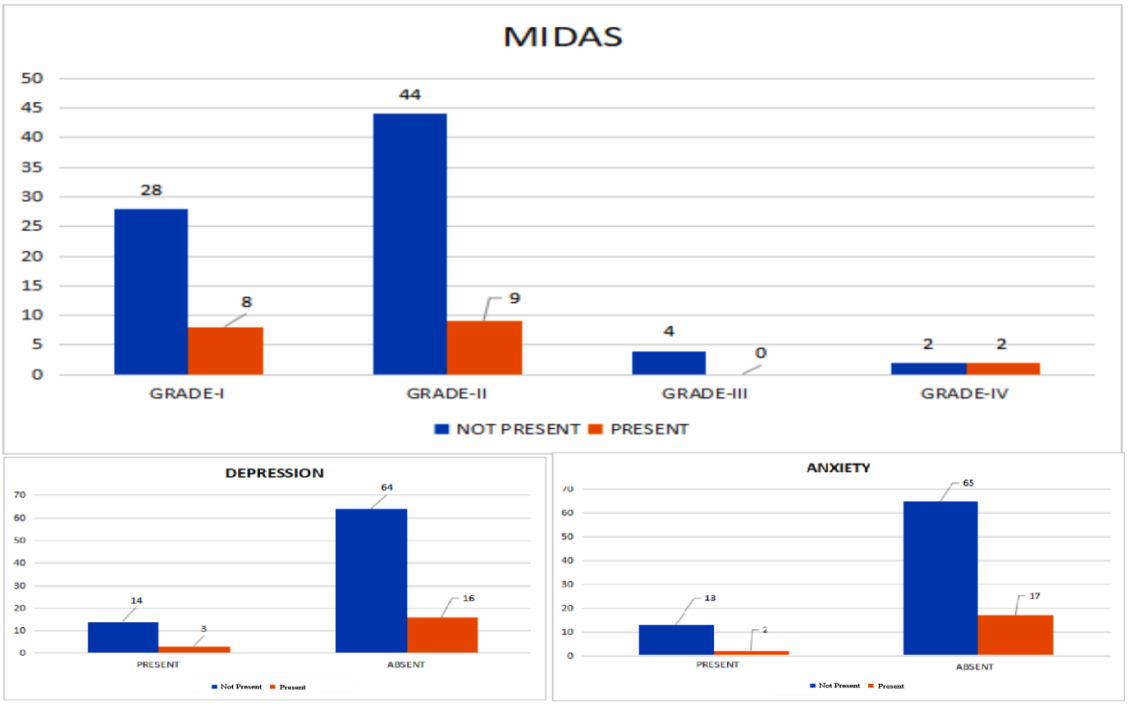

Fig 1: Cognitive Impairment and MIDAS grading (above); Cognitive Impairment with depression (left below) and anxiety (right below)

DISCUSSION

This study evaluated the prevalence and associations of cognitive impairment in migraine sufferers. Approximately 19.6% of participants exhibited mild cognitive impairment (MCI) based on the MoCA, consistent with findings from Ferreira *et al*., who reported cognitive deficits in attention, memory, and executive function among chronic migraineurs [13]. Despite the lack of statistical significance, the trend indicates that migraine severity and cognitive dysfunction may be interrelated. While the study did not establish a statistically significant link between cognitive impairment and migraine severity, depression, anxiety, or vitamin B12 levels, existing literature suggests potential underlying mechanisms. Research indicates that chronic neuroinflammation and repetitive cortical spreading depression (CSD)—a wave of neuronal depolarisation associated with migraine aura—may contribute to cumulative cognitive deficits in migraine patients. CSD has been shown to activate neuroinflammatory pathways, including the release of pro-inflammatory cytokines and activation of glial cells, which could impair cognitive function over time. Additionally, animal studies have identified the P2X7 receptor-NLRP3 inflammasome signaling pathway as a mediator of neuroinflammation and cognitive impairment in migraine models.

These findings underscore the need for further research to explore the complex interplay between neuroinflammation and cognitive function in migraine patients [14,15].

The association with depression and anxiety was not statistically significant in this study. However, the literature suggests that mood disturbances can exacerbate subjective cognitive complaints, even if not directly linked to objective test results [16]. The role of Vitamin B12 deficiency in cognitive impairment among migraine patients is noteworthy, even though statistical significance was not reached in the study. A high proportion of patients in the study were found to have low Vitamin B12 levels, which may suggest a potential compounding effect on cognitive symptoms. Vitamin B12 plays a crucial role in maintaining myelin integrity and supporting proper neurotransmission, both of which are critical to the functioning of the nervous system. Given that migraines involve complex neurovascular mechanisms, deficiencies in Vitamin B12 could exacerbate cognitive symptoms through its impact on nerve function, potentially influencing the severity of cognitive impairment observed in migraine patients. Although the findings do not establish a direct statistical correlation, the association warrants further exploration in future studies to determine whether Vitamin B12 deficiency may contribute to or worsen cognitive

dysfunction in migraine patients [17,18]. When the correlation of cognitive impairment and gender was made, it was found that 10.30 percent of patients were males and 89.30 percent were females. Among the male patients, 10 percent had cognitive impairment, and among the female patients, 89.30 percent had cognitive impairment. Still, the association between cognitive impairment and gender was not significant [18]. Our findings did not demonstrate a statistically significant association between cognitive impairment and migraine duration. However, studies such as those by Costa-Silva *et al*. [19] indicated that the duration of migraine could be linked to structural brain changes visible on MRI, potentially contributing to cognitive decline.

Our data, which illustrated the impact of headache severity and cognitive impairment, did not find it to be statistically significant. In our study, it was found that 19.5 per cent of patients had cognitive dysfunction in migraine. All patients had either mild cognitive impairment or early dementia based on the MOCA. The conclusions drawn by Chu HT *et al.* [20,21], who reported that increased headache frequency was correlated with poorer cognitive performance in a cohort of migraine sufferers. The findings suggest that repetitive exposure to migraine attacks may contribute to cumulative cognitive decline, potentially through mechanisms involving chronic neuroinflammation or cortical hyperexcitability as outlined by Taheri *et al*. [22,23]. Most 17/99 (89.5%) patients who had cognitive impairment had either anxiety or depression based on the HADS scale. In our study, 84 percent (16/19) were found to have low Vitamin B12 levels.

CONCLUSIONS

The study concluded that 19.58% of participants experienced cognitive impairment; however, no significant correlations were found with gender ($p = 0.42$), headache duration ($p = 0.17$), migraine severity ($p = 0.29$), depression ($p = 0.82$), anxiety, or vitamin B12 levels ($p = 0.72$). These results suggest that cognitive impairment in migraine patients is not strongly associated with these factors. Moreover, our research found that effective migraine treatment not only alleviates physical symptoms but also improves cognitive impairments. This improvement in cognitive function post-treatment underscores the potential for cognitive recovery and the importance of timely and effective management of migraines. This study contributes to the growing body of literature that affirms cognitive impairment as a significant and measurable aspect of migraine, urging healthcare providers to adopt a more holistic approach in the treatment of migraine patients.

CONTRIBUTION OF AUTHORS

Research concept– Dr. Annirudh Srivastava, Dr. Ashwani Bhat
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