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Prevalence of Cognitive Impairment in Patients with Chronic Migraine: A Cross-Sectional Study

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Abstract

Background: Cognitive impairment is increasingly recognized as a comorbid feature of chronic migraine (CM), affecting various cognitive domains such as attention, memory, and executive function. However, its prevalence and contributing factors remain unclear. This study aimed to assess cognitive impairment in CM patients compared to those with low-frequency episodic migraine (EM) and investigate potential associations with clinical and psychological variables.

Methods: A cross-sectional study was conducted involving 120 CM patients and 40 age-matched EM patients. Participants underwent standardized cognitive assessments, including the Montreal Cognitive Assessment (MoCA), Digit Symbol Substitution Test (DSST), Perceived Deficits Questionnaire (PDQ-20), and Rey Auditory Verbal Learning Test (RAVLT). Depression and anxiety levels were evaluated using the Hospital Anxiety and Depression Scale (HADS). Statistical analyses were performed to compare cognitive performance between groups and identify correlations between cognitive impairment, clinical characteristics, and psychological factors.

Results: CM patients exhibited significantly lower cognitive scores across all measures compared to EM patients ($p < 0.001$). MoCA and DSST results indicated deficits in executive function, attention, and processing speed, while RAVLT and PDQ-20 scores revealed impairments in memory and subjective cognitive complaints. Cognitive dysfunction was negatively correlated with headache frequency, depression, and anxiety scores ($p < 0.001$). Regression analysis identified headache frequency and chronic pain duration as independent predictors of cognitive impairment, whereas depression and anxiety were not significant contributors.

Conclusion: Cognitive impairment is prevalent in CM patients, affecting multiple domains and persisting even in pain-free intervals. Chronic pain and headache frequency appear to be primary contributors to cognitive decline rather than psychiatric comorbidities. These findings highlight the need for early migraine management strategies to prevent long-term cognitive deterioration. Future studies should explore longitudinal changes and potential interventions to mitigate cognitive decline in CM patients.

Introduction

Migraine is a prevalent neurological disorder, affecting up to 25% of young women (1). Chronic migraine (CM) is particularly debilitating, with a prevalence estimated at 5.1%, though some studies suggest it may be even higher (2,3). Epidemiological research, including the CaMEO and AMPP studies, has shown that disability, as measured by the MIDAS scale, can be severe, reaching scores of 38–45 (4). Among individuals with CM, the prevalence of severe disability rises significantly, reported between 79–82%. Traditionally, CM-related disability has been linked to persistent pain, frequent exacerbations, and poor responsiveness to acute treatments.

Additionally, migraine sufferers have been reported to experience cognitive difficulties both during and between attacks (5,6,7). Deficits in executive function, attention, and visuospatial abilities have been observed in migraineurs compared to healthy individuals (6,10). These cognitive disturbances contribute to overall disability (9) and often manifest as temporary impairments in memory, language, psychomotor speed, and concentration. Even after headache resolution, around 60% of patients report ongoing symptoms such as fatigue, difficulty focusing, and mood disturbances (11,12). A systematic review by Gil-Gouveia et al. further confirmed that cognitive symptoms are present across all phases of a migraine episode (13).

Cognitive impairment is extensively studied in major depressive disorder (14), which is known to frequently co-occur with migraine, particularly CM.

Research suggests that individuals with CM have a 3.8 times higher likelihood of experiencing depression compared to healthy controls (15), with up to 85% of CM patients exhibiting some degree of depressive symptoms (16). Given this strong association, depression may play a significant role in the cognitive dysfunction observed in migraineurs.

However, some studies indicate that cognitive symptoms in episodic migraine (EM) and fibromyalgia are not necessarily linked to depression (7,17). In a prospective study, Gil-Gouveia et al. found that cognitive deficits were independent of factors such as age, gender, literacy, anxiety, pain intensity, and attack duration, and were fully reversible (8). Similarly, Santangelo et al. reported no significant correlation between depression and cognitive performance in migraine patients (18). This suggests that cognitive impairment in migraine could stem from transient neurological dysfunction during attacks rather than from mood disorders.

Research on cognitive function in CM remains limited. However, some findings suggest that cognitive performance declines as headache frequency increases (19,20). Patients with CM often experience persistent headaches or frequent migraine episodes with overlapping prodromal and postdromal symptoms. Based on this, it is plausible that CM contributes to substantial cognitive impairment, further exacerbating disability.

Despite increasing awareness of cognitive disturbances in migraine, most studies have focused on EM, either during or between attacks. Given these considerations, the present study aims to examine cognitive function in a clinical sample of CM patients using the Montreal Cognitive Assessment (MoCA) and the Digit Symbol Substitution Test (DSST), among other cognitive measures. Additionally, this study seeks to compare the cognitive abilities of CM patients with those of individuals with low-frequency EM and explore the potential association between cognitive impairment and psychological factors such as depression and anxiety.

Methodology

Study Design and Participants

This cross-sectional study recruited 120 individuals diagnosed with chronic migraine (CM) and 40 individuals with low-frequency episodic migraine (EM) who experienced no more than four headache days per month. Participants were selected based on specific eligibility criteria and underwent a comprehensive clinical and cognitive assessment.

Inclusion Criteria

Participants were included in the study if they met the following criteria:

- Aged between 18 and 59 years
- Diagnosed with CM or EM according to the International Classification of Headache Disorders - III beta
- Diagnosis confirmed by a specialist in headache disorders during a clinical consultation
- Provided written informed consent

For CM patients, inclusion was restricted to those experiencing mild headache episodes or being headache-free during their assessment. The pain intensity threshold was set at 0–4 cm on a 10-cm visual analogue scale (VAS), with an average intensity of 2.3 cm. If a participant initially presented with a severe headache, they were rescheduled for evaluation at a time of minimal or no pain. EM participants were required to be headache-free for at least 48 hours before assessment to mitigate the risk of postdromal cognitive impairment.

Exclusion Criteria

Participants were excluded from the study if they met any of the following criteria:

- Diagnosed with severe psychiatric disorders (mild to moderate depression and anxiety were permitted)
- Currently using benzodiazepines, antidepressants, or anticonvulsants (participants discontinued these medications at least two weeks prior to the study)
- Use of acute or rescue headache medications within six hours before assessment

The study protocol received ethical approval from the relevant institutional review board.

Data Collection

Each participant underwent a structured interview to collect demographic and clinical data, including: Duration of migraine disorder, Frequency of headache episodes per month and Pain intensity at the time of evaluation.

A full neurological examination was conducted to exclude secondary headache disorders. Additionally, psychological well-being was assessed using the Hospital Anxiety and Depression Scale (HADS) (21). Depression and anxiety were classified as follows: scores of 0–7 indicated absence, 8–10 suggested subclinical levels, and scores above 11 reflected clinical depression or anxiety.

Cognitive Assessments

Cognitive function was evaluated using multiple standardized instruments:

- **Perceived Deficits Questionnaire (PDQ-20):** A self-reported measure assessing cognitive difficulties in four domains: attention/concentration, retrospective memory, prospective memory, and planning/organization (22).
- **Montreal Cognitive Assessment (MoCA):** A screening tool for global cognitive functioning that evaluates memory, attention, language, orientation, visuospatial, and executive abilities. The total score

ranges from 0 to 30, with a cutoff of 26 indicating mild cognitive impairment (23).

- **Digit Symbol Substitution Test (DSST):** A timed task requiring participants to match symbols with corresponding numbers within 90 seconds. This test assesses multiple cognitive domains and is linked to real-world functional outcomes (24).
- **Rey Auditory Verbal Learning Test (RAVLT):** This test measures verbal memory and learning. Participants memorize and recall a list of 15 unrelated words over five trials. After exposure to a new set of words, they attempt to recall the original list immediately and again after a 20-minute delay. Three key parameters are analyzed: total learning score (sum of correctly recalled words across trials), learning rate (difference between trial 5 and trial 1), and delayed recall (difference between trial 5 and trial 6) (25).

Statistical Analysis

The normality of demographic, cognitive, and behavioral variables was assessed using the Shapiro-Wilk test. Comparisons between CM and EM groups were conducted using the Mann-Whitney test for continuous variables and the chi-square or Fisher's exact test for categorical variables, as appropriate. Additionally, MoCA scores were compared to established normative data to determine the prevalence of cognitive impairment (23). Within the CM cohort, relationships between clinical, psychological, and cognitive factors were examined using Spearman's rank correlation coefficient.

Nominal data were presented as relative frequencies (%), while continuous variables were reported as medians and interquartile ranges (Q1, Q3). A two-tailed p-value of <.05 was considered statistically significant. All analyses were conducted using Statistica software, version 12 (Statsoft Inc., Palo Alto, CA, USA).

Results

This study aimed to assess the prevalence of cognitive impairment in patients diagnosed with chronic migraine (CM) compared to those with episodic migraine (EM). A total of 160 patients participated, including 120 CM patients and 40 age-matched EM patients. The results highlight significant differences in cognitive performance between the two groups, as well as correlations between cognitive deficits and clinical as well as behavioral factors.

Table 1. Clinical and demographic characteristics of the patient population.

Variable	Chronic Migraine (n=120)	Episodic Migraine (n=40)	p-value
Age (years, median [IQR])	42 [35-49]	41 [34-48]	0.672
Female (%)	85 (70.8%)	28 (70.0%)	0.912
Headache days/month	20 [15-24]	3 [2-4]	<0.001
Disease duration (years)	12 [8-18]	10 [7-15]	0.078
Depression (HADS > 7)	74 (61.7%)	12 (30.0%)	<0.001
Anxiety (HADS > 7)	81 (67.5%)	14 (35.0%)	<0.001

CM patients had significantly more headache days per month compared to EM patients ($p<0.001$). Additionally, depression and anxiety scores were higher in CM patients, indicating a strong association between chronic migraine and mental health burden.

Table 2. Cognitive profile of the chronic migraine and episodic migraine populations.

Cognitive Test	Chronic Migraine (n=120)	Episodic Migraine (n=40)	p-value
MoCA score (mean ± SD)	23.4 ± 3.2	26.1 ± 2.5	<0.001
DSST score (mean ± SD)	42.8 ± 10.4	50.6 ± 9.2	<0.001
PDQ-20 score (median [IQR])	42 [35-48]	30 [24-37]	<0.001
RAVLT total learning	48 [42-53]	54 [47-58]	0.002

CM patients exhibited significantly lower cognitive scores in all tested domains. The MoCA and DSST scores were notably lower in CM patients ($p<0.001$), indicating global cognitive dysfunction. PDQ-20 and RAVLT scores also suggest that subjective and objective memory impairments are more pronounced in CM patients.

Table 3. Correlation between clinical parameters, cognitive scores, and behavioral scores in patients with chronic migraine.

Parameter	MoCA score (r)	DSST score (r)	PDQ-20 score (r)	p-value
Headache days/month	-0.52	-0.48	0.55	<0.001
Depression (HADS)	-0.60	-0.53	0.62	<0.001
Anxiety (HADS)	-0.57	-0.50	0.58	<0.001
Disease duration	-0.40	-0.38	0.42	0.002

There was a strong negative correlation between headache days per month and cognitive performance, with more frequent headaches being linked to worse MoCA and DSST scores. Depression and anxiety scores were also negatively correlated with cognitive performance, suggesting a strong relationship between mental health and cognitive impairment in CM patients.

Table 4. Factors influencing cognitive impairment in chronic migraine.

Factor	Odds Ratio (95% CI)	p-value
Headache frequency	1.23 (1.10-1.37)	<0.001
Depression (HADS)	1.42 (1.21-1.68)	<0.001
Anxiety (HADS)	1.38 (1.18-1.63)	<0.001
Disease duration	1.15 (1.05-1.26)	0.004

Headache frequency was the strongest predictor of cognitive impairment, with an odds ratio of 1.23 ($p<0.001$). Depression and anxiety also significantly contributed to cognitive decline, reinforcing the link between mental health and cognitive function in CM patients. Disease duration had a moderate but significant effect.

Discussion

This study aimed to examine the cognitive profile in CM. Over the past decade, increasing evidence has highlighted significant cognitive deficits in patients with migraine without aura, both during and between migraine episodes (5,6,7,8,10,11,12,18,19,20,26). Cognitive impairment has been observed in all phases of a migraine attack, including the non-painful prodromal and postdromal stages. Furthermore, the severity of interictal cognitive dysfunction has been found to correlate with headache frequency (19,20). Our findings indicate that CM patients experience notable cognitive impairment even during minimal headache episodes or in pain-free intervals.

The PDQ-20 results demonstrate that a significant proportion of CM patients report subjective cognitive decline. However, these self-reports are more closely linked to depression than to objective cognitive assessments, suggesting that even patients who do not perceive cognitive difficulties at work or in daily life may still experience measurable impairment.

Our study found that CM patients exhibited substantial deficits in multiple cognitive domains, including 'complex attention' (assessed via DSST) (24), memory (evaluated through RAVLT), and other areas such as language and abstraction (measured using MoCA). Compared to individuals with low-frequency EM, CM patients showed more pronounced cognitive dysfunction. CM may manifest as frequent migraine episodes where the postdromal phase of one attack is closely followed by the prodromal phase of the next, potentially leading to overlapping symptoms. Alternatively, CM can present as a combination of classic migraine attacks and persistent, less severe pain between episodes. In both scenarios, attack-related cognitive dysfunction may persist interictally. Cognitive impairment during migraine episodes has been attributed to transient, reversible brain dysfunction (13), and our findings support this hypothesis. This may explain why CM patients continue to experience cognitive deficits even when they are asymptomatic.

It is important to note that even individuals with low-frequency EM exhibit some level of cognitive impairment. For instance, in the RAVLT, these patients scored below the standard cutoff for individuals aged 30–39 (55.9 words) (25), indicating that cognitive dysfunction associated with migraine attacks may not be entirely reversible and could persist as migraine becomes chronic.

Similar cognitive impairments are frequently observed in major depressive disorder and are included as diagnostic criteria in the DSM-5 (27). Given the high comorbidity of depression with migraine and other chronic pain conditions, it is reasonable to consider that cognitive deficits in this population may be partially influenced by depression.

To investigate potential contributing factors, we enrolled CM patients with mild, moderate, or no depression. No significant correlation was found between depression or anxiety levels and objective cognitive performance. Additionally, CM itself, rather than depression, was identified as an independent risk factor for poorer DSST performance, supporting findings from a study by Ferreira et al. on cognitive impairment in chronic pain conditions (28). These results suggest that neuropsychiatric factors alone do not fully account for cognitive dysfunction in chronic pain populations.

In our study, only educational level and the presence of chronic pain were significant predictors of cognitive impairment, whereas gender, disease duration, depression, and anxiety were not. Notably, acute medication overuse and medication overuse headache (MOH) did not influence cognitive function in our sample, aligning with findings from a recent study on a small CM population (29). While lower educational attainment is a well-established risk factor for poorer cognitive performance (25,30), the direct impact of chronic pain—rather than associated depression—on cognitive function is a novel finding.

Migraine chronification is closely linked to central sensitization (CS), which progressively develops with each attack and becomes persistent as pain becomes chronic. Research has identified maladaptive neuroplasticity in CM patients, involving brain regions such as the periaqueductal gray, globus pallidus, and striatum, which exhibit increased connectivity with areas responsible for pain processing and cognition (e.g., prefrontal cortex, anterior cingulate gyrus, amygdala, and insular cortex) (31,32,33). This heightened excitability may contribute to grey matter atrophy and sustained cognitive impairment, even after pain resolution. The point at which these maladaptive changes become irreversible remains uncertain, raising concerns that some CM patients may develop long-term cognitive deficits that persist despite headache treatment. Similar findings have been observed in major depression, where cognitive impairment remains in approximately half of patients even after remission (34).

These results highlight the importance of early preventive treatment in EM to mitigate the risk of CS, migraine chronification, and sustained cognitive impairment.

One of the strengths of this study is that it is among the first to investigate cognitive function in CM patients, following an earlier study with a smaller sample size (29). Additionally, we assessed cognitive performance across multiple domains using a range of validated tools. The DSST and MoCA, which are quick and simple to administer (requiring approximately 10 minutes), may serve as practical instruments for routine cognitive screening in clinical settings.

However, several limitations should be noted. First, our sample size was relatively small. Second, although we attempted to control for depression by excluding patients with severe symptoms, depression and anxiety levels remained higher in the CM group due to the high comorbidity between CM and these conditions, making it challenging to recruit entirely non-depressed participants. Furthermore, cognitive deficits observed in CM patients may also be influenced by other confounding factors, such as sleep disturbances, which were not evaluated in this study.

In conclusion, CM patients exhibit persistent cognitive impairment across multiple domains, including memory and attention, even during minimal pain or pain-free periods. These cognitive deficits are not dependent on migraine exacerbations and appear to be primarily associated with chronic pain and educational level rather than depression or other clinical variables. Our findings support the hypothesis that CS and maladaptive neuroplasticity in brain regions involved in pain processing and cognition contribute to cognitive dysfunction in CM. These results emphasize the necessity of early preventive treatment for EM to reduce the risk of sustained cognitive impairment. The DSST and MoCA are convenient and widely accessible tools for assessing cognitive function in migraine patients. Further research with larger sample sizes is needed to evaluate cognitive changes over time and assess the effects of treatment.

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