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MIGRAINE AND COGNITIVE FUNCTION: IMPACT ON ATTENTION, MEMORY, EXECUTIVE FUNCTION AND LONG-TERM COGNITIVE OUTCOMES- A STRUCTURED LITERATURE REVIEW

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ABSTRACT

Migraine is a highly prevalent and disabling neurological disorder whose burden extends beyond headache pain and sensory symptoms. Cognitive complaints, frequently described by patients as “brain fog,” difficulties with concentration, slowed thinking, forgetfulness, or impaired decision-making, occur across the migraine cycle and may substantially contribute to disability. This structured literature review synthesizes current evidence on cognitive function in migraine, with particular attention to attention and processing speed, memory, executive function, differences between ictal and interictal states, migraine subtype and chronicity, neurobiological mechanisms, treatment-related effects, and long-term cognitive outcomes. A structured search of PubMed/MEDLINE, Scopus, and Web of Science was designed to identify peer-reviewed studies, systematic reviews, meta-analyses, and relevant clinical guidance published primarily between January 2015 and September 2026, with selected earlier landmark studies included where necessary. The literature consistently supports reversible cognitive impairment during migraine attacks, whereas interictal findings are more heterogeneous. Recent meta-analyses suggest measurable deficits in complex attention, executive function, spatial cognition, and working memory, particularly in clinic-based and higher-burden populations. Chronic migraine, medication overuse, sleep disturbance, mood symptoms, and preventive medications may modify cognitive performance. Emerging neuroimaging and digital-assessment studies further support dynamic network-level changes across the migraine cycle. Long-term dementia risk remains debated despite recent meta-analytic signals. Cognitive symptoms should therefore be recognized as a clinically meaningful dimension of migraine and incorporated into patient-centered assessment and treatment evaluation.

KEYWORDS

Migraine, Cognitive Dysfunction, Attention, Memory, Executive Function, Brain Fog

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1. Introduction

Migraine is a chronic neurological disorder characterized by recurrent attacks of headache and associated symptoms such as nausea, photophobia, phonophobia, and, in a subset of patients, aura. It affects more than one billion people worldwide and remains one of the leading causes of years lived with disability, particularly among working-age adults (Eigenbrodt et al., 2021; GBD 2021 Headache Collaborators, 2025). The clinical burden of migraine, however, cannot be fully captured by headache intensity or attack frequency alone. Patients frequently describe cognitive problems that interfere with communication, work, learning, planning, family responsibilities, and social participation. These experiences have historically received less attention than pain, despite evidence that they are among the most disabling non-pain manifestations of the disease (Gil-Gouveia et al., 2016; Gerstein et al., 2023).

Cognitive symptoms in migraine span several domains. Patients may report reduced concentration, difficulty sustaining attention, slower processing, problems retrieving words, forgetfulness, reduced working-memory capacity, impaired multitasking, and difficulty making decisions. The colloquial term “brain fog” is increasingly used to describe this combination of slowed and inefficient cognition. Importantly, cognitive symptoms are not confined to the headache phase. They may emerge during the premonitory phase, intensify during pain, persist into the postdrome, and in some individuals remain detectable between attacks (Gerstein et al., 2023; Christensen et al., 2023). This temporal variability complicates research because studies performed at different phases of the migraine cycle may reach different conclusions even when investigating similar patient populations.

The distinction between subjective cognitive complaints and objective neuropsychological impairment is also important. Patient-reported difficulties are frequent and clinically relevant, but objective testing has yielded heterogeneous results. Earlier systematic reviews found that clinic-based samples more often showed abnormalities in memory, attention, and processing speed, whereas population-based studies were less consistent (Araújo et al., 2012; Lo Buono et al., 2017). More recent quantitative syntheses have strengthened the evidence for interictal deficits in selected domains, particularly complex attention, executive functioning, spatial cognition, and memory, while simultaneously emphasizing methodological heterogeneity and the potential effects of mood, sleep, medication exposure, and disease severity (Braganza et al., 2022; Pizer et al., 2024).

Migraine provides an especially interesting model for cognitive research because it is a cyclic brain disorder. Neural function changes across the preictal, ictal, postictal, and interictal periods, and brain regions implicated in cognition—including the prefrontal cortex, thalamus, hippocampus, hypothalamus, cerebellum, and large-scale attention and salience networks—are also involved in migraine pathophysiology (Fernandes et al., 2024; Cankaya et al., 2025). Consequently, cognitive dysfunction may reflect transient attack-related network disruption, accumulated disease burden, comorbid conditions, treatment effects, or a combination of these mechanisms.

A further clinical question concerns whether migraine is associated only with transient cognitive inefficiency or with progressive cognitive decline. Traditional longitudinal studies have generally been reassuring, and several reviews concluded that migraine does not clearly cause progressive cognitive deterioration (Vuralli et al., 2018; Gil-Gouveia & Martins, 2019). However, more recent large observational datasets and meta-analyses have reported modest associations between migraine and dementia, including Alzheimer disease and vascular dementia, with some analyses suggesting higher risk in migraine with aura (Jiang et al., 2022; Zhu et al., 2025). These findings require cautious interpretation because residual confounding, diagnostic misclassification, vascular risk, and ascertainment bias may influence the observed associations.

Understanding cognition in migraine has practical implications. Cognitive complaints may affect adherence to therapy, medication use, productivity, driving, education, and the ability to manage complex daily tasks. They may also influence treatment choice; for example, some preventive drugs can worsen word finding, attention, or memory, whereas successful migraine prevention may improve cognitive efficiency by reducing attack burden (Öztürk et al., 2019; Chowdhury et al., 2024). New digital approaches that repeatedly assess cognition using smartphones are beginning to capture day-to-day variation more precisely than conventional single-visit neuropsychological testing, potentially creating new patient-centered outcomes for trials and clinical care (Khorsand et al., 2026).

The aim of this structured literature review is to synthesize contemporary evidence regarding cognitive function in migraine, focusing on patient-reported symptoms, objective neuropsychological findings, variation across phases of the migraine cycle, the influence of aura and chronicity, neurobiological mechanisms, treatment-related effects, and long-term cognitive outcomes. Particular attention is given to areas in which evidence is convergent, areas in which results remain inconsistent, and methodological priorities for future research.

2. Methodology

This structured literature review was developed through a targeted search of PubMed/MEDLINE, Scopus, and Web of Science. The principal search window covered publications from January 1, 2015 to September 12, 2026. Earlier landmark studies and systematic reviews were included when they provided foundational evidence regarding migraine-related cognition, attack-phase cognitive changes, or neuropsychological methodology. Search concepts were combined using Boolean operators and included terms such as “migraine,” “migraine with aura,” “migraine without aura,” “chronic migraine,” “cognitive function,” “cognitive dysfunction,” “brain fog,” “attention,” “processing speed,” “memory,” “working memory,” “executive function,” “neuropsychological testing,” “ictal,” “interictal,” “postdrome,” “dementia,” “cognitive decline,” “neuroimaging,” “hippocampus,” “CGRP,” “preventive treatment,” “topiramate,” “onabotulinumtoxinA,” and “digital cognition.”

Eligible publications included peer-reviewed original studies, cohort studies, cross-sectional studies, qualitative studies, randomized or prospective treatment studies, systematic reviews, meta-analyses, and high-quality clinical reviews or consensus documents. Studies were prioritized when they clearly identified migraine according to accepted diagnostic criteria, used standardized cognitive or patient-reported outcome measures, differentiated migraine phase or subtype, or examined clinically relevant modifiers such as attack frequency, chronicity, medication overuse, psychiatric comorbidity, sleep, or preventive treatment. Studies focused exclusively on secondary headache disorders were excluded. One recent 2026 preprint was retained only to illustrate an emerging digital-assessment approach and is explicitly identified as a preprint in the reference list. Pediatric studies were not a primary focus because developmental differences in cognitive assessment would reduce comparability with the adult literature.

Because the objective was a structured thematic synthesis rather than a formal systematic review with quantitative re-analysis, no pooled effect estimates were calculated independently for this manuscript. Instead, evidence was organized according to recurring themes: subjective cognitive symptoms and functional impact; cognition across migraine phases; interictal cognitive performance; domain-specific findings in attention, processing speed, memory, and executive function; the effects of aura, chronic migraine, and medication overuse; neurobiological mechanisms and neuroimaging; modifying factors and treatment effects; long-term cognitive outcomes; and emerging digital assessment approaches. Particular emphasis was placed on systematic reviews and meta-analyses, while representative primary studies were used to illustrate clinically meaningful or methodologically important findings.

The literature search was updated immediately before manuscript preparation to capture publications available through September 12, 2026. Reference lists of key systematic reviews and contemporary narrative reviews were also screened to identify influential studies not retrieved by the primary keyword combinations. Given the heterogeneity of cognitive instruments, patient populations, migraine definitions, and attack-phase timing, results were interpreted qualitatively and with attention to study design rather than ranked solely by statistical significance.

3. Results

3.1. Patient-reported cognitive symptoms and functional burden

Patient-reported cognitive symptoms are among the most consistent findings across the migraine literature. Qualitative and diary-based studies indicate that patients frequently experience difficulty concentrating, slowed thinking, word-finding problems, forgetfulness, reduced ability to organize tasks, and impaired decision-making. These symptoms are clinically important because they interfere with functioning even when conventional neurological examination is normal. In a diary study evaluating 229 migraine attacks, cognitive symptoms were among the most intense and disabling features after pain and were closely related to the overall disability attributed to the attack (Gil-Gouveia et al., 2016). This observation helped shift attention from cognition as a secondary complaint to cognition as a meaningful component of migraine disability.

The Migraine Clinical Outcome Assessment System qualitative study provided a more detailed patient-centered description. Among 40 people with migraine, at least one cognitive symptom was reported by 90% during the pre-headache phase, 88% during headache, 68% during the post-headache phase, and 33% during interictal periods. Patients described disturbances in sustained attention, memory, receptive and expressive language, articulation, planning, information processing, and decision-making (Gerstein et al., 2023). The

breadth of these complaints supports the concept that migraine-related cognitive dysfunction is multidimensional rather than restricted to memory alone.

Cognitive symptoms also contribute to the broader real-world burden of migraine. Qualitative studies of patient experience have identified concentration and communication difficulties as important mechanisms through which migraine affects work, family roles, social relationships, and perceived independence (Buse et al., 2021). Population-level research similarly demonstrates that increasing headache frequency is associated with lower quality of life and progressively greater work and activity impairment (Ishii et al., 2024). Although such studies do not isolate cognition as the only determinant of functional loss, they provide important context: cognitive inefficiency occurs in a disease already associated with major productivity and participation costs.

The discrepancy between subjective complaints and objective test results should not be interpreted as evidence that patient-reported problems are unreliable. Subjective cognition captures everyday performance under conditions of fatigue, sensory hypersensitivity, pain, anxiety, multitasking, and time pressure, whereas laboratory tests typically isolate single cognitive domains under controlled conditions. Current reviews therefore argue that both perspectives are necessary. Patient-reported cognitive outcomes may be particularly sensitive to attack-phase changes and functional consequences, while formal neuropsychological assessment is better suited to identifying specific deficits and separating cognitive inefficiency from global impairment (Fernandes et al., 2024).

3.2. Cognitive function across the migraine cycle

One of the most reproducible findings is that cognition worsens during migraine attacks. A systematic review of neuropsychological testing performed during attacks found reversible impairment across several domains, with executive dysfunction appearing frequently, although the small number of studies and heterogeneity of cognitive instruments limited firm conclusions (Gil-Gouveia et al., 2015). In a prospective crossover design, patients tested during an untreated migraine attack performed worse than during headache-free periods on word reading speed, verbal learning, short-term verbal recall, and delayed recall, demonstrating that at least part of the cognitive burden is state dependent (Gil-Gouveia et al., 2015).

Attack-related cognitive symptoms are not limited to the pain phase. A systematic review of clinical descriptions of migraine attacks identified cognitive complaints in prodromal, headache, and postdromal phases, with cognitive symptoms among the most frequently reported prodromal and headache manifestations (Gil-Gouveia & Martins, 2018). More recent work on the postdrome has shown that concentration difficulties are among the most common symptoms after headache resolution. A 2023 systematic review and meta-analysis estimated that postdromal symptoms occur in a large majority of patients in clinic-based studies, with concentration difficulty reported in a substantial subgroup (Christensen et al., 2023). These findings are clinically relevant because patients may remain functionally impaired even after pain has improved.

The cyclic nature of migraine means that the definition of “interictal” is critical. Participants tested one day after an attack may still be in a postdromal state, while those tested shortly before an attack may already be in a preictal state. Conventional cross-sectional research may therefore misclassify dynamic cognitive changes as stable interictal deficits. This problem is increasingly recognized as one explanation for inconsistent findings across studies (Fernandes et al., 2024).

Recent digital assessment provides a method for overcoming this limitation. In the MIND cohort, repeated smartphone-based testing across thousands of person-days demonstrated greater subjective brain fog and task forgetting on ictal days, together with slower performance on measures of attention and processing speed and modest impairment of working-memory accuracy. Because repeated assessment allows each patient to serve partly as their own control, digital phenotyping can capture within-person fluctuation more precisely than one-time clinic testing (Khorsand et al., 2026). These findings reinforce the view that migraine-related cognitive dysfunction is dynamic and strongly phase dependent.

Clinically, this suggests that cognition should be discussed as part of the entire migraine cycle. A patient may have adequate cognitive performance during a conventional headache-free consultation yet experience substantial impairment during prodrome, headache, or postdrome. For occupational and educational planning, the duration of cognitive impairment may therefore exceed the duration of severe pain itself.

3.3. Interictal cognitive performance

Whether objective cognitive impairment persists between attacks has been debated for decades. Earlier population-based studies frequently found little or no difference between migraine and control groups, whereas clinic-based studies more often detected abnormalities in attention, memory, executive functioning, or processing speed (Araújo et al., 2012; Vuralli et al., 2018). Referral bias provides a plausible explanation: patients attending specialist headache clinics tend to have higher attack frequency, greater disability, more comorbidities, and more medication exposure than people with migraine identified in population samples.

Quantitative synthesis has strengthened evidence that at least some interictal deficits are measurable. Braganza et al. (2022) included 17 studies in a meta-analysis of interictal neuropsychological performance and found moderate negative effects in complex attention, immediate and delayed memory, spatial cognition, and executive functioning. Importantly, the authors emphasized limitations including incomplete assessment of mood, medication exposure, and performance validity. A broader 2024 multidomain meta-analysis by Pizer et al. similarly concluded that migraine is associated with measurable neuropsychological differences across multiple domains, while highlighting substantial variability among studies. Together, these meta-analyses suggest that interictal cognitive differences are real at the group level but are not universal, severe, or sufficiently specific to define an individual patient.

A 2024 longitudinal controlled study provides clinically relevant evidence that interictal deficits may be at least partly reversible. Preventive-treatment-naïve patients with migraine performed worse than controls on backward digit span, Trail Making Tests, and Stroop measures, suggesting impairment in working memory, attention, vigilance, and executive control. After six months of successful preventive treatment, performance improved across cognitive measures (Chowdhury et al., 2024). Although practice effects and treatment heterogeneity must be considered, improvement after migraine control argues against interpreting all interictal deficits as fixed neurodegeneration.

Recent data also indicate differences between episodic and chronic migraine. A 2026 study comparing episodic migraine, chronic migraine, and healthy controls found more pronounced interictal deficits in chronic migraine, particularly in attention and working memory, with additional within-person worsening during attacks (Kizilkilic et al., 2026). This aligns with the clinical impression that cognitive complaints become more persistent as attack frequency increases and the boundaries between migraine phases become less distinct.

The most defensible interpretation of current evidence is therefore not that every person with migraine has interictal cognitive impairment, but that measurable deficits are more likely in selected patients—particularly those with frequent or chronic migraine, higher disability, comorbid sleep or mood symptoms, medication overuse, and specialist-clinic ascertainment. This distinction is important when communicating results to patients because the literature does not support a generalized claim of permanent cognitive impairment in all migraineurs.

3.4. Attention, processing speed, and executive function

Attention and executive function are among the cognitive domains most consistently implicated in migraine. These functions are closely related: efficient attention is required to select relevant information, sustain concentration, switch between tasks, inhibit competing responses, and maintain goal-directed behavior. Deficits in these processes correspond closely to patient descriptions of difficulty multitasking, slower thinking, and reduced ability to work under cognitive load.

Studies using the Attention Network Test have provided a more granular view of attentional function. Patients with migraine without aura have shown slower executive-control responses even when alerting and orienting networks were relatively preserved, suggesting that executive attention may be selectively vulnerable (Huang et al., 2019). Similar studies have reported poorer performance on Stroop and trail-making tasks, which depend on inhibitory control, processing speed, and cognitive flexibility. The relationship with attack frequency is not entirely consistent, but some studies have found greater impairment in patients with more frequent or prolonged attacks.

Task-switching paradigms further support executive vulnerability. Migliore et al. (2022) compared patients across low-frequency episodic, high-frequency episodic, and chronic migraine and found evidence of altered task-switching performance, providing a mechanistic framework for the everyday complaint that rapidly alternating between cognitive demands becomes more difficult as migraine burden increases. These findings complement meta-analytic evidence for reduced executive performance between attacks (Braganza et al., 2022; Pizer et al., 2024).

Decision-making represents another executive domain of interest, particularly in chronic migraine with medication overuse. Lau et al. (2023) found impaired decision-making under ambiguity in patients with chronic migraine and medication-overuse headache compared with both chronic migraine without medication overuse and healthy controls, while decision-making under explicit risk and standard executive performance were less affected. This dissociation suggests that altered emotional feedback or reward processing, rather than generalized executive failure, may contribute to repeated analgesic overuse. Earlier work also linked medication overuse to orbitofrontal dysfunction and disadvantageous decision-making (Gómez-Beldarrain et al., 2011; Biagianti et al., 2012).

Executive deficits must nevertheless be interpreted cautiously. Performance on tasks such as the Stroop test, Trail Making Test, or Iowa Gambling Task is influenced by fatigue, depression, anxiety, sleep quality, pain expectancy, and medication. Moreover, a statistically significant group difference may be modest and may not translate into clinically meaningful impairment for every individual. The strongest conclusion is that attention, processing speed, and executive control are recurrently implicated domains, particularly during attacks and in patients with high disease burden.

3.5. Memory and language

Memory complaints are common in migraine, but the pattern is more specific than a generalized amnesic syndrome. Patients often report forgetting recently presented information, losing track of tasks, difficulty retrieving words, or needing more time to encode and manipulate information. These experiences may reflect disrupted attention and working memory as much as impaired storage.

A 2026 systematic review and meta-analysis focused specifically on short-term and working memory found no clear evidence of impaired short-term memory in adults with migraine compared with healthy controls, including analyses of migraine with and without aura. In contrast, working-memory performance was significantly poorer in migraine, suggesting selective vulnerability in higher-order processes requiring active manipulation and cognitive control (McKay & Kelly, 2026). This distinction is important because it provides a plausible explanation for “brain fog”: basic storage may remain intact while tasks that require holding, updating, and using information become less efficient.

Attack-phase studies have demonstrated reductions in verbal learning and recall, but these abnormalities may improve outside attacks (Gil-Gouveia et al., 2015). Interictal meta-analyses have also found small-to-moderate differences in immediate and delayed memory, although heterogeneity is substantial (Braganza et al., 2022). Memory performance therefore appears sensitive to both migraine phase and task complexity.

Language-related symptoms are less extensively quantified but are prominent in patient reports. The MiCOAS qualitative study documented difficulties with receptive language, expressive language, and articulation across migraine phases (Gerstein et al., 2023). These symptoms overlap with word-finding problems reported as adverse effects of some preventive medications, particularly topiramate, making medication history essential when assessing language complaints. In clinical practice, new, abrupt, or persistent language deficits still require appropriate evaluation for secondary neurological causes rather than automatic attribution to migraine.

3.6. Aura, chronic migraine, and medication overuse

The extent to which migraine subtype predicts cognitive dysfunction remains uncertain. Studies directly comparing migraine with and without aura have produced mixed results. In a neuropsychological study of 100 patients with migraine, both aura and non-aura groups demonstrated abnormalities in selected domains, with differences in delayed memory and set shifting compared with healthy controls, but no simple pattern establishing aura as uniformly associated with worse cognition (Santangelo et al., 2019). Meta-analytic work has similarly failed to establish a consistent broad cognitive disadvantage attributable solely to aura (Braganza et al., 2022; McKay & Kelly, 2026).

Chronicity appears more clinically relevant. Patients with chronic migraine have a higher cumulative burden of headache, postdrome, sleep disruption, medication use, affective symptoms, and functional impairment. Cross-sectional research has reported lower global cognitive scores and greater impairment of language fluency, memory, or executive function in chronic migraine than in controls or episodic migraine (Cai et al., 2019). The 2026 comparison of episodic and chronic migraine likewise found more pronounced interictal deficits in chronic migraine (Kizilkilic et al., 2026).

Medication overuse introduces an additional layer of complexity. Cai et al. (2019) found that chronic migraine was associated with greater cognitive impairment, while nonsteroidal anti-inflammatory drug overuse itself did not explain the cognitive differences after adjustment. In contrast, studies of decision-making suggest that medication-overuse headache may be associated with specific frontostriatal or orbitofrontal abnormalities rather than generalized cognitive decline (Lau et al., 2023). Metacognitive research has also identified altered monitoring of cognitive performance in episodic and chronic migraine, suggesting that the relationship between objective ability and self-perceived performance deserves further study (Zucca et al., 2020).

Taken together, current evidence supports a burden model rather than a single-subtype model. Aura may influence particular cognitive or neuroimaging findings, but frequent attacks, chronic migraine, medication overuse, prolonged symptoms, and comorbidities more consistently identify patients likely to report or demonstrate cognitive problems.

3.7. Neurobiological mechanisms and neuroimaging

The mechanisms underlying cognitive dysfunction in migraine are likely multifactorial. Migraine is increasingly conceptualized as a disorder of distributed brain networks rather than an isolated vascular phenomenon. Regions involved in nociception, sensory processing, arousal, memory, and executive control show dynamic functional changes across the migraine cycle. Contemporary reviews implicate the hypothalamus, thalamus, hippocampus, prefrontal cortex, cerebellum, brainstem, salience network, default-mode network, and executive-control networks in the interaction between migraine and cognition (Fernandes et al., 2024; Vuralli & Ayata, 2026).

The thalamus is particularly relevant because it integrates sensory information and participates in attention. Abnormal thalamocortical processing could contribute simultaneously to sensory hypersensitivity and inefficient allocation of cognitive resources. Similarly, hypothalamic changes associated with premonitory symptoms may affect arousal, sleep, appetite, and attention before pain begins. During an attack, competition between pain processing and cognitive networks may further reduce available attentional capacity.

The hippocampus has attracted increasing interest because of its role in memory, stress, and pain modulation. A 2025 resting-state fMRI study found lower cognitive scores in patients with migraine and reported differences in hippocampal subfield connectivity and volumes compared with controls (Cankaya et al., 2025). These findings support a biological substrate for some cognitive differences, although cross-sectional imaging cannot establish whether structural or functional alterations are causes, consequences, compensatory responses, or incidental correlates of migraine burden.

Cortical hyperresponsiveness, deficient sensory habituation, altered synaptic plasticity, neuroinflammation, energy-metabolism disturbances, and dysregulated monoaminergic or CGRP-related signaling have also been proposed. These mechanisms may interact. For example, repeated attacks could increase allostatic load and disrupt sleep, while sleep loss independently reduces attention and working memory. Anxiety and depression can further alter cognitive efficiency and network function. The resulting cognitive phenotype may therefore reflect the combined effect of intrinsic migraine neurobiology and external modifiers rather than one single lesion or pathway.

Neuroimaging differences between migraine with and without aura add complexity. Recent imaging reviews describe both shared and distinct patterns, including greater involvement of visual regions, thalamic networks, and cerebellar systems in aura (Visual Neuroimaging Review Group, 2025). However, imaging biomarkers are not currently specific enough for individual diagnosis of migraine-related cognitive dysfunction. The most promising role of neuroimaging is therefore mechanistic: linking changes in network connectivity or regional structure with well-characterized cognitive outcomes and migraine phases.

3.8. Comorbidities, medications, and treatment-related cognitive change

Cognitive performance in migraine cannot be interpreted without considering comorbidities and treatment. Sleep disturbance, insomnia, anxiety, depression, fatigue, and medication use are common in migraine and independently affect attention, memory, processing speed, and executive function. These factors can either mediate genuine migraine-related cognitive problems or confound comparisons between patients and healthy controls. Contemporary reviews therefore emphasize careful measurement of mood and sleep rather than excluding cognitive symptoms as “psychological” when such comorbidities are present (Fernandes et al., 2024).

Medication effects are particularly important. Topiramate is an effective preventive therapy but is well known to cause cognitive adverse effects in a subset of patients, especially word-finding difficulty, slowed processing, reduced verbal fluency, and attention problems. Reviews suggest that cognitive adverse effects are often dose dependent and may improve with slower titration, dose reduction, or discontinuation (Mula, 2014). A migraine-specific study also demonstrated measurable cognitive effects during topiramate therapy, highlighting the need to separate treatment-emergent symptoms from the underlying migraine phenotype (Öztürk et al., 2019).

Conversely, successful prevention may improve cognition by reducing attack frequency and cumulative symptom burden. Chowdhury et al. (2024) observed improvement in interictal attention and executive measures after six months of preventive treatment. A prospective real-world study of onabotulinumtoxinA in chronic migraine reported improvement in cognitive speed and working-memory accuracy alongside reductions in headache burden, anxiety, and depression (Ho et al., 2020). More recent work comparing responders and non-responders to anti-CGRP monoclonal antibodies or onabotulinumtoxinA suggests that treatment response may be accompanied by improvement in executive functions, although causal pathways remain to be clarified (Martins et al., 2026).

These findings have two implications. First, cognitive symptoms should be documented before preventive treatment so that new complaints can be distinguished from baseline migraine-related dysfunction. Second, cognition may represent an additional treatment outcome beyond headache days. Current international recommendations emphasize individualized preventive treatment and consideration of tolerability and comorbidities (Puledda et al., 2024). For patients whose occupation or education relies heavily on verbal fluency, rapid processing, or sustained attention, cognitive tolerability may be particularly important in treatment selection.

3.9. Long-term cognitive decline and dementia risk

The question of long-term cognitive decline is clinically sensitive because patients with recurrent subjective memory problems may fear that migraine is causing permanent brain damage or dementia. Historically, longitudinal studies and narrative reviews were generally reassuring, finding no consistent evidence of progressive cognitive deterioration attributable to migraine (Vuralli et al., 2018; Gil-Gouveia & Martins, 2019). However, more recent epidemiological meta-analyses have reported modest associations between migraine and dementia.

A 2022 systematic review and meta-analysis including nine studies and more than 290,000 participants found that migraine was associated with increased risk of all-cause dementia and vascular dementia (Jiang et al., 2022). A larger 2025 meta-analysis of 11 cohort studies involving approximately 6.96 million participants reported elevated adjusted risks for all-cause dementia, Alzheimer disease, and vascular dementia, with subgroup analysis suggesting a stronger association for migraine with aura (Zhu et al., 2025). The very large sample size increases precision, but observational data cannot fully eliminate residual confounding.

Several mechanisms could theoretically connect migraine to later-life cognitive disease: shared vascular risk factors, endothelial dysfunction, stroke, white-matter lesions, chronic inflammation, sleep disturbance, depression, or common genetic susceptibility. Yet the absolute risk for an individual patient may remain low, and the observed associations are not proof that repeated migraine attacks directly cause neurodegeneration. Diagnostic surveillance bias is also possible because people with frequent neurological symptoms may interact with healthcare systems more often and therefore receive dementia diagnoses earlier.

The apparent tension between neuropsychological longitudinal studies and epidemiological dementia studies may reflect different outcomes. A younger or middle-aged migraine cohort may show stable cognitive performance over years, while a population-level association with dementia could emerge later through vascular or comorbidity pathways. Alternatively, heterogeneity among studies may produce inconsistent pooled signals. Recent mechanistic reviews explicitly note this discrepancy and call for cohorts that combine standardized migraine phenotyping, repeated objective cognitive assessment, imaging, vascular risk measurement, and long follow-up (Vuralli & Ayata, 2026).

For current clinical practice, the evidence does not justify telling patients that migraine inevitably leads to dementia. A more accurate message is that recent observational research suggests a modest association that remains under investigation, and that management of established vascular and lifestyle risk factors remains appropriate for general brain health.

3.10. Digital cognitive assessment and future biomarkers

Traditional cognitive research in migraine has relied on one or two clinic-based assessments, an approach that is poorly suited to a fluctuating disorder. Digital testing creates the possibility of brief, repeated assessments during preictal, ictal, postictal, and interictal periods. The 2026 MIND cohort demonstrated the feasibility of smartphone-based measurement of reaction time, attention, working-memory accuracy, and subjective brain fog over thousands of observation days. Objective changes were modest but directionally consistent with patient-reported worsening during attacks (Khorsand et al., 2026).

This approach has several advantages. Repeated within-person measurement reduces between-subject confounding, allows adjustment for practice effects, and may identify individualized cognitive signatures of impending attacks. In the future, digital cognition could be integrated with electronic headache diaries, sleep metrics, heart-rate variability, physical activity, and treatment exposure. Such multimodal data may help distinguish attack-related cognitive change from medication effects or chronic baseline impairment.

However, digital biomarkers require validation. Smartphone ownership, familiarity with technology, device type, environmental distractions, and motivation can influence performance. Algorithms trained in highly selected cohorts may not generalize across age, education, language, or socioeconomic groups. Privacy and data governance are additional considerations. For these reasons, digital cognitive measures should complement rather than replace clinical history and validated neuropsychological assessment.

A further research priority is to develop a migraine-specific patient-reported cognitive outcome. Generic cognitive questionnaires may not capture the phase-dependent combination of language, attention, memory, and executive symptoms described by patients. The MiCOAS program provides a framework for developing outcomes that reflect what patients themselves consider meaningful (Gerstein et al., 2023). Better cognitive endpoints could then be incorporated into trials of acute and preventive therapies rather than treating headache-day reduction as the only relevant outcome.

Table 1. Representative evidence on cognitive function in migraine

Study	Design	Population / scope	Main cognitive finding	Interpretive value
Braganza et al. (2022)	Meta-analysis	17 interictal studies	Moderate deficits in complex attention, memory, spatial cognition, and executive function	Supports group-level interictal differences while highlighting confounding
Gerstein et al. (2023)	Qualitative study	40 adults with migraine	Cognitive symptoms reported across pre-headache, headache, post-headache, and interictal phases	Demonstrates patient-perceived functional relevance
Pizer et al. (2024)	Multidomain meta-analysis	Neuropsychological studies across multiple domains	Objective differences across several cognitive domains	Broad quantitative synthesis
Chowdhury et al. (2024)	Longitudinal controlled study	150 migraine patients and 150 controls	Interictal attention/executive deficits improved after preventive treatment	Suggests partial reversibility
Cankaya et al. (2025)	MRI / cognitive study	49 migraine patients, 41 controls	Hippocampal connectivity and volumetric differences associated with cognitive status	Provides mechanistic imaging support
Zhu et al. (2025)	Systematic review and meta-analysis	11 cohorts; ~6.96 million participants	Modest increased risk of all-cause, Alzheimer, and vascular dementia	Raises long-term risk question; causality uncertain
McKay & Kelly (2026)	Systematic review and meta-analysis	Adult migraine memory studies	Short-term memory preserved; working memory impaired	Suggests selective higher-order memory vulnerability

4. Discussion

The literature reviewed here supports cognitive dysfunction as a clinically meaningful dimension of migraine, but it also demonstrates why simple statements such as “migraine causes cognitive impairment” are inadequate. The strongest and most consistent evidence concerns transient cognitive worsening during attacks and surrounding phases of the migraine cycle. Patient-reported studies, prospective within-subject testing, systematic reviews, and recent digital assessments converge on a pattern of reduced concentration, slower processing, working-memory inefficiency, language difficulty, and executive dysfunction during symptomatic periods (Gil-Gouveia et al., 2015; Gerstein et al., 2023; Khorsand et al., 2026). This convergence across methodologies strengthens the clinical validity of symptoms often described informally as brain fog.

Interictal cognitive dysfunction is more heterogeneous. Earlier community-based studies frequently failed to detect major deficits, whereas clinic-based studies more often reported abnormalities. Recent meta-analyses now suggest that group-level differences are detectable in complex attention, executive function, spatial cognition, and memory, but effect sizes vary and study quality remains inconsistent (Braganza et al., 2022; Pizer et al., 2024). These findings fit a model in which cognitive dysfunction is not an obligatory trait of migraine but becomes more likely as disease burden and complexity increase.

Several factors probably explain heterogeneity. First, migraine phase is often inadequately defined. A supposedly interictal assessment may occur during the late postdrome or early preictal phase. Second, cognitive tests differ greatly in sensitivity and may measure overlapping domains. Third, migraine populations differ in attack frequency, chronicity, aura status, treatment, medication overuse, sleep quality, anxiety, depression, age, and education. Fourth, selection bias can enrich specialist-clinic cohorts for more severe disease. Finally, many studies are underpowered and conduct multiple cognitive comparisons without prespecified primary outcomes.

The domain-specific pattern is increasingly informative. Working memory and executive control appear more consistently vulnerable than simple short-term storage. The 2026 meta-analysis by McKay and Kelly found preserved short-term memory but impaired working memory, a distinction that aligns with patient complaints of being able to retain information but struggling to manipulate, prioritize, or use it under cognitive load. Attention-network and task-switching studies similarly point toward executive control and cognitive flexibility rather than diffuse global impairment. This pattern is compatible with network-level dysfunction affecting prefrontal, thalamic, and salience systems.

Chronic migraine deserves particular attention. Frequent attacks reduce the duration of truly symptom-free periods and increase exposure to pain, poor sleep, fatigue, acute medication, and psychological stress. Cross-sectional and recent comparative studies generally show more pronounced cognitive problems in chronic than episodic migraine. However, it remains difficult to determine whether chronicity directly causes cognitive dysfunction or serves as a marker for accumulated burden and comorbidity. Medication-overuse headache illustrates this complexity. Some studies find specific abnormalities in decision-making and orbitofrontal function, while broader cognitive differences may be attributable more to chronic migraine than to medication overuse itself (Cai et al., 2019; Lau et al., 2023).

The relationship between migraine treatment and cognition is equally nuanced. Topiramate demonstrates that an effective preventive therapy can worsen cognitive symptoms in a subset of patients. Conversely, successful prevention with other strategies may improve cognitive performance by reducing attack burden. Improvement following preventive treatment, onabotulinumtoxinA, and newer anti-CGRP therapies suggests that at least part of the cognitive phenotype is reversible (Ho et al., 2020; Chowdhury et al., 2024; Martins et al., 2026). Future trials should therefore include cognitive outcomes, particularly when therapies have different neurocognitive tolerability profiles.

Neuroimaging findings provide biological plausibility but should not be overinterpreted. Changes in hippocampal volume or connectivity, thalamocortical function, and executive networks are intriguing, yet cross-sectional imaging cannot distinguish cause from consequence. Longitudinal imaging linked to repeated cognitive measurements across migraine phases would be substantially more informative. Multimodal approaches that combine imaging, digital phenotyping, sleep and activity data, and biomarkers may ultimately identify subgroups with distinct cognitive trajectories.

The long-term dementia question remains unresolved. Recent large meta-analyses report modestly increased risks of all-cause dementia, Alzheimer disease, and vascular dementia, while older longitudinal cognitive studies were largely reassuring. These results are not necessarily contradictory because dementia risk is influenced by age, vascular disease, genetic factors, mood, sleep, and other comorbidities that may not

be captured in neuropsychological cohorts. For clinicians, it is essential to avoid deterministic interpretation. Migraine should not be presented as a direct pathway to dementia, but the emerging association justifies continued research and reinforces the importance of managing modifiable vascular and lifestyle risks.

From a practical perspective, cognitive symptoms should be incorporated into routine migraine assessment. Asking patients whether attacks affect concentration, language, memory, planning, and ability to work can reveal disability that headache-frequency measures miss. Clinicians should establish the timing of symptoms relative to the migraine cycle, assess sleep and mood, review medication effects, and investigate atypical or persistent deficits when clinically indicated. Cognitive complaints that are abrupt, progressive, focal, or unrelated to the patient's established migraine pattern should not automatically be attributed to migraine.

This review also highlights a measurement gap. Common migraine outcomes-monthly migraine days, pain intensity, acute medication use, and disability scales-capture important dimensions but may miss cognitive recovery. A patient may experience fewer headache days yet continue to report postdromal brain fog or medication-related word-finding difficulty. Conversely, improved cognitive function may represent a meaningful benefit even if headache-day reduction is modest. Patient-centered research initiatives and repeated digital testing offer tools for addressing this gap.

Several limitations should be acknowledged. This manuscript is a structured thematic review rather than a registered systematic review, and therefore does not provide a PRISMA flow diagram, formal risk-of-bias scoring, or original pooled effect estimates. The evidence base itself is heterogeneous, with variable migraine definitions, cognitive tests, attack-phase timing, and control of confounders. Some cognitive domains are supported by many small studies rather than a few definitive large trials. Publication bias toward positive findings is possible. In addition, emerging 2025–2026 studies require replication before their results can be considered definitive.

Future research should prioritize prospective longitudinal designs with repeated testing across the migraine cycle, standardized definitions of ictal and interictal periods, prespecified cognitive outcomes, adequate control of mood and sleep, and careful documentation of preventive and acute medications. Studies should distinguish migraine with and without aura, episodic and chronic migraine, and medication-overuse headache while avoiding excessive subgroup fragmentation. Digital cognitive assessment appears particularly promising because it can capture intra-individual variability at scale. Finally, long-term cohorts integrating cognitive testing, neuroimaging, vascular risk, and dementia outcomes are needed to resolve whether migraine contributes independently to later-life cognitive decline.

5. Conclusions

Cognitive dysfunction is an important but incompletely characterized component of migraine. Evidence is strongest for reversible impairment during migraine attacks and surrounding phases, particularly involving attention, processing speed, working memory, language, and executive function. Interictal deficits are less universal but are increasingly supported by meta-analytic evidence, especially in patients with frequent or chronic migraine and greater overall disease burden. Aura alone does not consistently predict cognitive impairment, whereas chronicity, medication overuse, sleep disturbance, mood symptoms, and treatment effects may modify performance.

Current neuroimaging and mechanistic research supports a network-level explanation involving prefrontal, thalamic, hippocampal, hypothalamic, and other cognitive-pain systems. Treatment may either worsen cognition, as can occur with topiramate, or improve it when migraine control is achieved. Recent observational meta-analyses suggest a modest association between migraine and dementia, but causality remains unproven and results should be interpreted cautiously. Future studies should integrate repeated digital cognitive assessment, standardized neuropsychological measures, treatment exposure, and long-term follow-up. Clinically, cognition should be recognized as a patient-centered outcome and routinely considered when evaluating migraine burden and treatment response.

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