

Cognitive Impairment during Migraine Attacks: An Analysis from the American Registry for Migraine Research (ARMR)

Meesha Trivedi

Mayo Clinic in Arizona

Richa Chirravuri

Mayo Clinic in Arizona

Todd J. Schwedt

Mayo Clinic in Arizona

Ryotaro Ischii

Kyoto Prefectural University of Medicine

Gina Dumkrieger

Mayo Clinic in Arizona

David Dodick

Atria Research and Global Health Institute

Christopher H. Gottschalk

Yale University

Stewart J. Tepper

Dartmouth Medical School

Kathleen Digre

University of Utah

Catherine D. Chong

chong.catherine@mayo.edu

Mayo Clinic in Arizona

Research Article

Keywords: migraine, cognition, executive function, language, prodrome, postdrome

Posted Date: October 18th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11118845/v1>

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Additional Declarations: Competing interest reported. Meesha Trivedi has no disclosures. Richa Chirravuri has no disclosures. Todd J. Schwedt, within the prior 24 months, received compensation for consulting with Lundbeck and Salvia, and royalties from UpToDate. His employer received compensation for his consulting with AbbVie. He has stock options/equity in AllevaLux and Nocira. His institution received research grants on his behalf from AbbVie, American Heart Association, Flinn Foundation, Henry Jackson Foundation, National Headache Foundation, National Institutes of Health, Patient Centered Outcomes Research Institute, Pfizer, and United States Department of Defense. Ryotaro Ishii has served as a consultant for Amgen K.K., Eli Lilly Japan K.K., Daiichi Sankyo Co., Ltd., Pfizer Japan Inc., and Otsuka Pharmaceutical Co., Ltd. He has received speaker honoraria from Amgen K.K., Daiichi Sankyo Co., Ltd., and Otsuka Pharmaceutical Co., Ltd. Gina Dumkrieger has no disclosures in the previous 24 months. David Dodick: Discloses the following (2025): Consulting: AbbVie, Vedana. Honoraria: Wolters Kluwer, Oxford University Press, Cambridge University Press. Stock options/shareholder/patents/board of directors: ExSano (options), Palion (options), Keimon Medical (Options), Man and Science, Healint (options), Theranica (options), Epien (options), Nocira (options), Matterhorn (shares), Ontologics (shares), King-Devick Technologies (options/board), EigenLyfe (Options/Board), NuvieBio (options/board), Cephalgia Group (options/board), Atria Health (options/employee). Patent 17189376.1-1466:vTitle: Onabotulinum Toxin Dosage Regimen for Chronic Migraine Prophylaxis (Non-royalty bearing). Christopher H. Gottschalk has received research funding from Lundbeck, Merz, Rehaler and Relivion. He has served as a consultant and/or on Advisory Boards (honoraria) for Abbvie, Aspeya, Axsome Therapeutics, Guidepoint Global, Impel, Lundbeck, Pfizer, Satsuma, Theranica, Tonix, Vectura Fertin. He is a board member of the Headache Cooperative of the Northeast (HCNE) and the Alliance for Headache Disorders Advocacy (AHDA). Stewart J. Tepper has received research funding from Aeon, Amgen, Annovis, Axsome, Bristol Myer Squibb, Cassava, Cognition, Eli Lilly, Genentec, Hoffman LaRoche, Inhibikase, Ipsen, Johnson and Johnson/Intracellular, Karuna, Lundbeck, Merz, Neurocrine, Novartis, Pfizer, PrecisionMed, Revance, Sanofi, Suven, UCB, and Upsher Smith. He has

served as a consultant and/or on Advisory Boards for Abbvie, Aeon, Alphasights, Amgen, Amneal, Aruene/eNeura, Atheneum, Axsome Therapeutics, Bausch Health, Becker Pharmaceutical Consulting, Catch Therapeutics, ClearView Healthcare Partners, ClickTherapeutics, CoolTech, CRG, Decision Resources, Defined Health, DRG, DocDelta, Dr. Reddy's, Eli Lilly, ExpertConnect, FCB Health, Fenix, Gilmartin Capital, GLG, Guidepoint Global, Health Advances, Health Science Communications, HMP Communications, Impel, InteractiveForums, IQVIA, Keyquest, KLJ Associates, Krog and Partners, Lundbeck, M3 Global Research, Magellan Health, Magnolia Innovation, MJH Holdings, Neurofront Therapeutics, Nocira, P Value Communications, Pain Insights, Inc, Palion Medical, Paragon Therapeutics, Perfood, Pfizer, Putnam Associates, Rehaler, SAI MedPartners, Satsuma, Scilex, Slingshot Insights, Spherix Global Insights, Sprigings Intellectual Property Law, Strategy Inc, Synapse Medical Communication, System Analytic, Taylor and Francis, Tegus, Theranica, Third Bridge, Tonix, Trinity Partners, and Vial. He received salary from Thomas Jefferson University and Ki Health Partners. He has served on the speakers bureau for AbbVie, Amneal, Axsome, Dr. Reddy's, Eli Lilly, Impel, Lundbeck, Pfizer, Scilex, and Tonix. He received CME honoraria from the American Headache Society, Consortium for Research Education Social Awareness and Training In Neurosciences, Diamond Education Foundation, Forefront Collaborative, HMP Global, Medical Education Speakers Network, Medical Learning Institute Peerview, Miller Medical Education, National Association for Continuing Education, North American Center for CME, Physicians' Education Resource, PlatformQ Education, Primed, Vindico Medical Education, and WebMD/Medscape. He received travel assistance from AbbVie, American Headache Society, Amneal, Axsome, Diamond Educational Foundation, Dr. Reddy's, Eli Lilly, Impel, Lundbeck, Pfizer, and Sanofi. He received book royalties from Springer Health. Kathleen Digre is supported in part by an unrestricted grant to the Department of Ophthalmology and Visual Sciences from Research to Prevent Blindness, New York, New York. She has no conflict of interests. Catherine Chong receives research support from the National Institutes of Health, the American Heart Association and the Department of Defense. She serves as a Board Member of the Headache Cooperative of the Pacific.

Abstract

Objective

To characterize the prevalence, severity, and clinical correlates of subjective ictal-phase cognitive impairment across the migraine attack cycle in a large, specialty-center cohort.

Background

Cognitive dysfunction is a prevalent and disabling feature of migraine attacks, yet standardized tools for measuring migraine attack-related cognitive impairment are limited, and the clinical predictors of cognitive burden remain incompletely characterized. Prior work has largely focused on the ictal phase, although cognitive symptoms are reported across the prodromal, ictal, and postdromal phases of the migraine attack.

Methods

This is a cross-sectional analysis of data from the American Registry for Migraine Research (ARMR), a multicenter registry enrolling patients with migraine diagnosed by headache specialists using ICHD-3 criteria. Participants completed the Migraine-Specific Cognitive Impairment Scale (Mig-SCog; range 0–18, higher scores indicating greater impairment). Associations between cognitive impairment and clinical variables were evaluated using correlation analyses, group comparisons, and multivariable linear regression.

Results

Among 1,231 patients (87.2% women; mean age 46.0 ± 13.7 years; 71.5% chronic migraine), 1,175/1,231 (95.5%) endorsed cognitive symptoms, with a mean Mig-SCog total score of 8.2 ± 4.8 . Executive dysfunction accounted for $71.1\% \pm 18.6\%$ of total cognitive burden, but the proportional contribution of language impairment increased with migraine frequency category, from $22.4\% \pm 18.9\%$ in low-frequency episodic migraine to $28.8\% \pm 19.5\%$ in chronic migraine ($p = 0.003$). In multivariable regression, migraine frequency classification was not independently associated with cognitive impairment. The strongest independent predictors of cognitive impairment were anxiety symptom score ($\beta = 0.15$, $p < 0.001$) and depression symptom score ($\beta = 0.13$, $p = 0.002$), with standardized coefficients exceeding those of all migraine frequency classification categories. Women reported significantly greater cognitive impairment than men across all domains (8.4 ± 4.8 vs. 6.8 ± 4.7 ; $p < 0.001$, small effect). Word-finding difficulty demonstrated the largest ictal symptom-frequency association, with scores rising from 3.3 ± 2.6 among those never reporting word-finding difficulty to 13.6 ± 3.6 among those always reporting it (adjusted $p < 0.001$, large effect). In phase-based analyses, word-finding difficulty and impaired concentration were associated with 4.6- and 3.7-point higher cognitive impairment scores than in patients who did not report these symptoms during the prodrome and 5.1- and 4.0-point higher scores during the postdrome, respectively (all $p < 0.001$, large effects). Among patients with chronic migraine and available disease duration data ($n = 661$), cognitive burden was lower among those within the first 12 months of chronicity compared with those with longer-standing chronic migraine (7.7 ± 4.9 vs. 9.0 ± 4.6 ; $p = 0.010$, $d = 0.27$). Sound hypersensitivity ($\beta = 0.25$) and cutaneous allodynia ($\beta = 0.20$) were the strongest independent sensory predictors of cognitive impairment (both $p < 0.001$).

Conclusion

Migraine-related cognitive dysfunction was nearly universal in this specialty-center cohort and was not adequately captured by migraine frequency classification alone. Anxiety and depression symptom burden, language impairment, full-cycle cognitive symptoms spanning prodrome through postdrome, and sensory sensitization are clinically meaningful markers of cognitive burden that should be assessed alongside more traditional migraine characteristics when evaluating patients who have migraine.

INTRODUCTION

Cognitive dysfunction is a prevalent and disabling symptom among patients with migraine, with patients often reporting impairments in memory, attention, language, and information processing during their migraine attacks.¹ Objective neuropsychological studies corroborate these subjective reports, demonstrating reversible, transient declines in verbal learning, recall, and processing speed during spontaneous attacks compared to interictal baselines, and smartphone-based ambulatory testing confirmed that these deficits are present in real-world settings on migraine days relative to headache-free days, although these studies have largely been conducted in small samples.^{2–4} Despite this, the objective measurement of ictal cognitive burden in clinical populations remains challenging, as most existing assessment tools are not migraine-specific and may fail to capture the transient, functionally debilitating deficits that characterize the migraine attack.⁵ The Migraine-Specific Cognitive Impairment Scale (Mig-SCog) was developed to address this gap by providing a structured, validated framework for quantifying executive dysfunction and language impairment during migraine attacks in clinical and research settings.⁵

Despite growing evidence linking migraine to cognitive dysfunction, variability in study methodologies, sample sizes, and assessment tools has led to inconsistent findings regarding this symptom's prevalence, severity, and associated predictors. Research suggests that multiple factors, including attack frequency, migraine disease duration, sensory hypersensitivity, and psychiatric comorbidities, may contribute to cognitive impairment, but the relative contribution and interplay of these predictors remain poorly understood, particularly in large, well-characterized clinical cohorts.^{1,6–8} Importantly, cognitive symptoms in migraine are not confined to the headache phase. Qualitative data from the MiCOAS project, which systematically evaluated patient-reported cognitive experiences across all phases of the migraine attack cycle in 40 adults with episodic or chronic migraine, found that 90% of participants reported cognitive symptoms during the prodromal phase, 88% during the ictal phase, and 68% during the postdrome, with language and speech, sustained attention, executive function, and memory each affected across phases.⁹ Despite this, large-scale clinical characterization of cognitive burden across the full attack cycle and identification of which patients are most affected remains lacking.

This study uses the Mig-SCog questionnaire to assess self-reported subjective cognitive impairment in a large cohort of patients with migraine enrolled from headache specialty clinics to address these gaps. The objectives of this study were to characterize the prevalence and nature of subjective cognitive impairment during migraine attacks, to evaluate cognitive symptom associations across the prodromal, ictal, and postdromal phases of the migraine attack, and to determine the clinical and demographic characteristics independently associated with greater cognitive burden.

METHODS

This cross-sectional cohort analysis utilized data from the American Registry for Migraine Research (ARMR), a multicenter, longitudinal registry established by the American Migraine Foundation. The American Registry for Migraine Research (ARMR) study received Institutional Review Board (IRB) approval at each of the nine participating academic headache specialty clinics across the United States and was conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent prior to study enrollment and data collection. ARMR contains data from patients diagnosed with headache disorders by specialty clinicians using the International Classification of Headache Disorders (ICHD) criteria, who were receiving care at tertiary headache centers across the United States of America.¹⁰ Registry data contain patient questionnaires and headache diaries, as well as physician assessments and electronic health record information. Complete ARMR protocols have been reported previously.¹¹ Each participating institution obtained Institutional Review Board approval of ARMR protocols.

Patient and Data Selection

Data for this primary analysis were obtained from clinician-entered diagnostic information and cross-sectional electronic questionnaires completed by patients. Participants included in the analysis had an ICHD-3 diagnosis of migraine and completed the Mig-SCog questionnaire. No statistical power calculation was performed prior to the study. Instead, analyses were conducted using all available data from adult participants meeting inclusion criteria.

Measures

Migraine-Specific Cognitive Impairment (Mig-SCog Questionnaire)

The Migraine-Specific Cognitive Impairment Scale (Mig-SCog) is a 9-item, self-administered patient questionnaire developed to quantify cognitive complaints during the ictal phase of migraine attacks within two domains, executive function and language. Patients rated the frequency of each symptom as "Often" (2 points), "Sometimes" (1 point), or "No" (0 points), yielding a total score from 0 to 18. The executive dysfunction subscore includes five items assessing dysfunction in attention/processing speed/orientation (examples of items include "do you have trouble performing tasks at your normal speed?") and dysfunction in planning/attention ("do you have trouble maintaining the thread of your thoughts?"). Executive dysfunction subscores range from 0 to 10. The language domain includes four items which assess general language impairment ("do you have difficulty organizing a sentence or conversation?") and naming-specific language impairment ("do you have trouble remembering the correct names of objects?"). In its validation study cohort of patients diagnosed with episodic migraine, the mean Mig-SCog score was 8.63 (SD 4.04; study cohort of 93 patients aged 18 to 83 years old, with a mean age of 39.2 ± 11.6 years, and 93% female).⁵

Validation studies evaluated the Mig-SCog for its reliability and capacity to measure cognitive dysfunction associated with migraine attacks. Convergent validity was demonstrated through associations between Mig-SCog scores and patients' self-reported cognitive complaints during migraine attacks. Construct validity was supported by correlations between the 9-item Mig-SCog and both a comprehensive 43-item draft questionnaire and the previously validated Cognitive Failures Questionnaire, which measures the frequency of everyday cognitive errors.⁵

Analysis of the scale's diagnostic performance showed that it accurately differentiated migraine-related cognitive symptoms from those experienced during pain-free periods, non-headache pain episodes, and during tension-type headache episodes. Test-retest analyses confirmed consistency in total scores and item-level agreement across administrations. Retrospective scores aligned with real-time scores from the same attacks, supporting the Mig-SCog's reliability as a retrospective measure.

Patient Background & Headache History

Patient age and sex were obtained from registry data. Headache specialists diagnosed patients with migraine, and these diagnoses were entered by clinicians or research staff into ARMR. Participants with a clinician-confirmed diagnosis of chronic migraine were classified as chronic migraine (CM) per ICHD-3 criteria. Among participants without chronic migraine, patient-reported monthly headache frequency (average over the prior three months) was used to classify low-frequency episodic migraine (LFEM; 0–7 days/month) and high-frequency episodic migraine (HFEM; ≥8 headache days/month). Prior ARMR data support the clinical relevance of the frequency thresholds used in this study. Ishii and colleagues demonstrated that patients with 8–14 migraine days per month, and those with 15–23 days per month had similar burden across most disability, productivity, and psychiatric measures, which supports the 8 headache days per month threshold used in this study to distinguish low-frequency from high-frequency episodic migraine.¹²

Patients completed a non-validated headache history questionnaire which asked patients to self-report their monthly headache frequency as an average over the past three months. Participants also reported the year their headaches began, allowing for the calculation of their migraine disease duration at the time of the survey. The questionnaire additionally assessed the frequency with which patients experienced headache-associated symptoms across the prodromal, ictal, and postdromal phases of their migraine attacks. Prodromal and postdromal symptoms were assessed as two checklists of symptoms, beginning with “Before you feel headache pain, do you experience any of the following symptoms (choose all that apply),” and “After your headache pain ends, do you experience any of the following symptoms (choose all that apply)...,” respectively. Prodrome and postdrome data were recorded as binary variables reflecting the presence or absence of each symptom during the respective phase. Ictal symptoms were assessed using an ordinal frequency scale with five response categories: “never,” “rarely,” “less than half the time,” “half the time or more,” and “always.” For ictal analyses, symptom frequency was analyzed as an ordinal variable. For phase-based analyses, ictal symptoms were dichotomized as absent (“never”) versus present (any frequency) to align with binary prodromal and postdromal variables.

Average Headache Intensity

The Migraine Disability Assessment (MIDAS) test is a widely used questionnaire used to evaluate the disability of migraine and impairment of functional ability.¹³ MIDAS contains an additional non-scored question that assesses how painful on average a patient’s headaches were over the past three months on a scale from zero to ten (with 0 being no pain and 10 being the most painful headache imaginable). Patients’ response to this question was used in this analysis to quantify headache intensity.

Photophobia

The Photosensitivity Assessment Questionnaire (PAQ) is a validated, 16-item measure, divided into 8 questions evaluating light-avoiding behaviors that are scaled into a photophobia score from 0 to 1, and 8 questions evaluating light-seeking behaviors that are scaled into a photophilia score from 0 to 1.¹⁴ Only photophobia subscores were used in this analysis. Importantly, the PAQ assesses general photosensitivity and is not specific to changes in light sensitivity caused by migraine. Mean photophobia scores for healthy controls in prior studies ranged from 0.11 (SD 0.13) to 0.12 (SD 0.13).^{15,16}

Phonophobia

Phonophobia was assessed by the Hyperacusis Questionnaire (HQ), which asks patients to self-report how often they experience 14 sound sensitivity behaviors. Responses are rated from 0 (“no”) to 3 (“yes, a lot”), with a final score between 0 and 42.¹⁷ Scores above 28 are interpreted as pronounced auditory hypersensitivity, and population-based samples have reported mean scores near 15 (SD 6.7).^{17,18}

Cutaneous Allodynia

The Allodynia Symptom Checklist (ASC) contains a series of 12 behaviors indicative of cutaneous allodynia and has been validated in patients with migraine.¹⁹ Each item reflects a behavior suggestive of tactile hypersensitivity and is rated as 0 (“does not apply,” “never,” or “rarely”), 1 (“less than half the time”), or 2 (“half the time or more”), resulting in total scores ranging from 0 to 24. Scores of 0–2 denote no allodynia, 3–5 mild allodynia, 6–8 moderate allodynia, and 9 or higher severe allodynia.¹⁹

Depression

Depressive symptoms were assessed using the Patient Health Questionnaire-2 (PHQ-2), a validated two-item screener for depression comprising the first two items of the PHQ-9. The PHQ-2 has been validated in patients with migraine.²⁰ It asks respondents how often, over the preceding two weeks, they have experienced depressed mood (“feeling down, depressed, or hopeless”) and anhedonia (“little interest or pleasure in doing things”). Each item is scored from 0 (“not at all”) to 3 (“nearly every day”), producing a total score from 0 to 6, with a score of 3 or greater suggesting depression.²¹

Anxiety

Anxiety symptoms were assessed using the Generalized Anxiety Disorder-7 (GAD-7), a validated and widely used seven-item questionnaire asking respondents how often, over the preceding two weeks, they have exhibited behaviors indicative of generalized anxiety such as nervousness, excessive

worrying, restlessness, and irritability. The GAD-7 has been validated in patients with migraine.²² Each item is scored from 0 ("not at all") to 3 ("nearly every day"), producing a total score from 0 to 21; scores of 0–4 suggest minimal anxiety, 5–9 mild, 10–14 moderate, and 15–21 severe.²³

Statistical Analysis

This study is an exploratory cross-sectional analysis of clinical correlates of ictal-phase subjective cognitive impairment. Analyses were performed using IBM SPSS Statistics (Version 31). Statistical significance was defined as a two-tailed $p < 0.05$. Listwise deletion was applied on a per-analysis basis. A Bonferroni correction for multiple comparisons was applied within each analysis, and results that remained significant after correction are denoted with a dagger (†).

Migraine-specific cognitive impairment (Mig-SCog total score; range 0–18) was analyzed as a continuous outcome. Executive dysfunction (range 0–10) and language impairment (range 0–8) subscores were evaluated as secondary outcomes. Among participants with endorsement of one or more subjective cognitive symptoms (total score ≥ 1), each subscore was expressed as a proportion of the individual's total Mig-SCog score.

Continuous variables are reported as mean $\pm$ standard deviation, and categorical variables as frequency and percentage. Between-group comparisons were performed using independent-samples t-tests for two groups and one-way analysis of variance (ANOVA) for comparisons across three or more groups. Effect sizes for two-group comparisons were estimated using Cohen's d , except for sex comparisons, where Hedges' g was used due to unequal group sizes.

Associations between continuous clinical variables (age, monthly headache frequency, disease duration, depression score, and anxiety score) and Mig-SCog outcomes were evaluated using Pearson correlation coefficients.

Cognitive symptoms across migraine frequency classification (low-frequency episodic migraine (LFEM), high-frequency episodic migraine (HFEM), and chronic migraine (CM)) was evaluated using one-way ANOVA with Tukey HSD post-hoc testing. Analyses were restricted to participants with clinician-confirmed CM to evaluate cognitive symptoms across chronic migraine duration. Mig-SCog total scores were compared across six CM duration categories (1–2 months, 3–12 months, 1–2 years, 3–5 years, 5–10 years, and > 10 years) using one-way ANOVA, with Games–Howell post-hoc testing. A pre-specified binary comparison evaluated ≤ 12 months versus > 12 months of disease duration. Effect sizes for pairwise comparisons were estimated using Cohen's d .

Multivariable linear regressions were used to evaluate the association between migraine frequency classification and subjective cognitive symptoms. Mig-SCog total score was specified as the dependent variable, with LFEM as the reference category for migraine frequency. Models were adjusted for age, sex, disease duration (years since headache onset), average headache pain intensity, depression score (PHQ-2), and anxiety score (GAD-7).

To evaluate the relationship between ictal symptom frequency and cognitive symptoms, ordinal symptom-frequency variables ("never," "rarely," "less than half the time," "half the time or more," and "always") were analyzed using general linear models with Mig-SCog total score as the dependent variable. Models were adjusted for age, sex, headache frequency, headache disease duration, PHQ-2 depression score, and GAD-7 anxiety score. Statistical significance and adjusted partial eta squared values were derived from these models. Descriptive values in the corresponding table are reported as mean $\pm$ SD with n (%). Effect sizes were estimated using partial eta squared (η^2).

For analyses across migraine phases (prodrome, ictal, and postdrome), ictal symptom-frequency variables were dichotomized as absent ("never") versus present (any frequency). Mig-SCog total scores were compared between groups using independent-samples t-tests, with effect sizes estimated using Cohen's d .

Associations between validated sensory hypersensitivity measures (photophobia, phonophobia, and cutaneous allodynia) and Mig-SCog total score were evaluated using Pearson correlations and multivariable linear regression. All sensory measures were entered simultaneously as continuous predictors, adjusted for age, sex, headache frequency, headache disease duration, PHQ-2 depression score, and GAD-7 anxiety score in the multivariable sensory hypersensitivity model.

RESULTS

The American Registry for Migraine Research enrolled 2,708 patients over the course of the study, and 1,379 (50.9%) were diagnosed with migraine per ICHD-3 criteria and completed baseline questionnaires. The Mig-SCog questionnaire was completed by 1,304 (94.6%) at baseline; 1,231 additionally had complete migraine frequency classification data and comprised the final study cohort. Sample characteristics are presented in Table 1.

Table 1
Cohort characteristics

CHARACTERISTIC	n	PERCENTAGE (%)	
Sex			
Female	1071	87.20%	
Male	157	12.80%	
Migraine Frequency Classification			
LFEM (0–7 days/month)	103	8.40%	
HFEM (8–14 days/month)	248	20.10%	
Chronic Migraine (≥ 15 days/month)	880	71.50%	
CHARACTERISTIC	n	RANGE (MIN–MAX)	MEAN ± SD
Demographics			
Age (years)	1231	18.0–86.0	46.0 ± 13.7
Clinical Headache Phenotype			
Duration of Disease (years)	1011	0.0–79.0	21.0 ± 15.8
Headache Frequency (days/month)	1231	0.0–30.0	18.4 ± 9.4
Psychological Comorbidity			
GAD Score (Anxiety)	1226	0.0–21.0	6.1 ± 5.2
PHQ Score (Depression)	1118	0.0–6.0	1.4 ± 1.7
Ictal Cognitive Impairment			
Total Ictal Cognitive Impairment Score	1231	0.0–18.0	8.2 ± 4.8
Ictal Executive Dysfunction Subscore	1231	0.0–10.0	5.4 ± 2.9
Ictal Language Impairment Subscore	1231	0.0–8.0	2.7 ± 2.3
Subscore Contribution to Ictal Cognitive Impairment Total Score (%)			
Ictal Executive Dysfunction Subscore Contribution	1175	0.0–100.0%	71.1% ± 18.6
Ictal Language Impairment Subscore Contribution	1175	0.0–100.0%	28.9% ± 18.6

Continuous variables are reported as mean $\pm$ standard deviation (SD); categorical variables as frequency (n) and percentage. Migraine frequency classification was defined as follows: low-frequency episodic migraine (LFEM), 0–7 headache days/month; high-frequency episodic migraine (HFEM), ≥ 8 headache days/month without a diagnosis of chronic migraine per ICHD-3 criteria; chronic migraine (CM), clinician-diagnosed per ICHD-3 criteria. Duration of disease reflects years since headache onset and was calculated from patient-reported year of headache onset; sample size is lower due to missing onset year data. Subscore contribution values reflect each domain score expressed as a percentage of the individual's total Mig-SCog score and are reported among participants with any cognitive impairment (total score ≥ 1). Mig-SCog = Migraine-Specific Cognitive Impairment Scale (total score range 0–18; executive function subscore range 0–10; language subscore range 0–8). GAD = Generalized Anxiety Disorder 7-item Scale. PHQ = Patient Health Questionnaire.

The study cohort was predominantly women (1,071/1,228, 87.2%), with a mean age of 46.0 $\pm$ 13.7 years. The majority of participants had chronic migraine (880/1,231, 71.5%), with smaller proportions classified as high-frequency episodic migraine (HFEM; 248/1,231, 20.1%) and low-frequency episodic migraine (LFEM; 103/1,231, 8.4%). Patients reported a mean migraine frequency of 18.4 $\pm$ 9.4 days per month and had lived with migraine for a mean of 21.0 $\pm$ 15.8 years (n = 1,011, reflecting missing headache onset year data). Mean GAD-7 and PHQ-2 scores were 6.1 $\pm$ 5.2 (n = 1,226) and 1.4 $\pm$ 1.7 (n = 1,118), respectively.

Mig-SCog total ictal cognitive symptom scores ranged from 0 to 18, with a mean of 8.2 $\pm$ 4.8 (Table 1). The mean executive dysfunction subscore was 5.4 $\pm$ 2.9 and the mean language impairment subscore was 2.7 $\pm$ 2.3. Among the 1,175/1,231 patients (95.5%) with any cognitive symptoms (total score ≥ 1), executive dysfunction accounted for 71.1% $\pm$ 18.6% of the total Mig-SCog score and language impairment accounted for 28.9% $\pm$ 18.6%; subscore percentages were undefined for patients with a total score of 0 and were excluded from these calculations.

Women reported significantly greater total ictal cognitive symptoms than men across all Mig-SCog domains (Table 2). Total scores were higher in women than men (8.4 $\pm$ 4.8 vs. 6.8 $\pm$ 4.7; $p < 0.001$, g 0.33), as were executive dysfunction subscores (5.6 $\pm$ 2.8 vs. 4.6 $\pm$ 2.9; $p < 0.001$, g 0.36), and language impairment subscores (2.8 $\pm$ 2.3 vs. 2.3 $\pm$ 2.2; $p = 0.009$, g 0.22). Effect sizes for all comparisons were small, as estimated by Hedges' g .

Table 2
Cognitive impairment by patient sex

Total Ictal Cognitive Impairment Burden	Female (n = 1071)	Male (n = 157)	p-value
	Mean ± SD	Mean ± SD	
	8.4 ± 4.8	6.8 ± 4.7	< 0.001* ^{†A}
Executive Function Subscore	5.6 ± 2.8	4.6 ± 2.9	< 0.001* ^{†A}
Language Subscore	2.8 ± 2.3	2.3 ± 2.2	0.009* ^{†A}

Group differences evaluated using independent-samples *t*-tests (two-tailed). Mig-SCog: Migraine-Specific Cognitive Impairment Scale. SD = standard deviation. *: significant at *p* < 0.05. [†]: significant after adjustments. ^A: small effect sizes estimated using Hedges' *g*.

Table 3
Associations between cognitive impairment scores and clinical variables

Variable	Total Cognitive Impairment (r)	Executive Dysfunction Subscore (r)	Language Impairment Subscore (r)
Monthly Frequency	0.2 (<i>p</i> < 0.001* [†])	0.2 (<i>p</i> < 0.001* [†])	0.1 (<i>p</i> < 0.001* [†])
Age	-0.2 (<i>p</i> < 0.001* [†])	-0.2 (<i>p</i> < 0.001* [†])	-0.1 (<i>p</i> < 0.001* [†])
Disease Duration	-0.1 (<i>p</i> < 0.001* [†])	-0.1 (<i>p</i> < 0.001* [†])	-0.1 (<i>p</i> = 0.003* [†])
Depression Score (PHQ)	0.3 (<i>p</i> < 0.001* [†])	0.3 (<i>p</i> < 0.001* [†])	0.2 (<i>p</i> < 0.001* [†])
Anxiety Score (GAD)	0.3 (<i>p</i> < 0.001* [†])	0.3 (<i>p</i> < 0.001* [†])	0.2 (<i>p</i> < 0.001* [†])

Values represent Pearson correlation coefficients (*r*). All correlations are two-tailed. Monthly headache frequency reflects average headache days per month over the preceding three months. Disease duration reflects years since headache onset. GAD: Generalized Anxiety Disorder 7-item Scale score. PHQ: Patient Health Questionnaire-2 score. Mig-SCog: Migraine-Specific Cognitive Impairment Scale. *: significant at *p* < 0.05. [†]: significant after adjustments.

Pearson correlations between Mig-SCog outcomes and continuous clinical variables are presented in Table 3. PHQ-2 depression score and GAD-7 anxiety score demonstrated the largest observed correlations with Mig-SCog total score and executive dysfunction subscore (*r* = 0.3, *p* < 0.001 for both), with weaker associations observed for language impairment (*r* = 0.2, *p* < 0.001 for both), suggesting that the relationship between psychiatric symptom burden and cognitive symptoms during migraine attacks is more strongly expressed through executive dysfunction than through language impairment. Monthly migraine frequency was positively correlated with total cognitive symptoms, executive dysfunction, and language impairment scores. Age and migraine disease duration were each inversely associated with all three outcomes.

Patients with chronic migraine reported more cognitive symptoms than those with low-frequency or high-frequency episodic migraine in unadjusted comparisons (Table 4).

Table 4
Cognitive impairment by migraine frequency classification

Variable	LFEM	HFEM	Chronic	p-value
	Mean ± SD (n = 103)	Mean ± SD (n = 248)	Mean ± SD (n = 880)	
Mig-SCog Total Score	7.1 ± 5.0	7.7 ± 5.0	8.4 ± 4.7*	0.009* [†]
Executive Function	4.9 ± 3.1	5.2 ± 3.0	5.6 ± 2.8	0.036*
Language Subscore	2.2 ± 2.4	2.5 ± 2.4	2.9 ± 2.3*	0.006* [†]
Cognitive Impairment Subscore Contribution (%)				
Executive Function	66.0% ± 29.5	70.7% ± 23.4	67.2% ± 23.4	0.139
Language	22.4% ± 18.9	25.3% ± 19.1	28.8% ± 19.5	0.003* [†]

Values are reported as mean ± standard deviation. Group differences were evaluated using one-way analysis of variance (ANOVA). Pairwise comparisons were performed using Tukey HSD post-hoc testing. Migraine frequency classification was defined as follows: low-frequency episodic migraine (LFEM), 0–7 headache days/month; high-frequency episodic migraine (HFEM), ≥ 8 headache days/month without a diagnosis of chronic migraine per ICHD-3 criteria; chronic migraine (CM), clinician-diagnosed per ICHD-3 criteria. Domain contribution values reflect each subscore expressed as a percentage of the individual's Mig-SCog total score and were calculated only among participants with Mig-SCog total score ≥ 1. Mig-

SCog: Migraine-Specific Cognitive Impairment Scale; SD: standard deviation. *Overall ANOVA significant at $p < 0.05$; asterisked group values indicate significant pairwise difference from LFEM on Tukey HSD post-hoc testing. [†]: significant after adjustments.

Mean Mig-SCog total scores were 7.1 ± 5.0 in LFEM, 7.7 ± 5.0 in HFEM, and 8.4 ± 4.7 in CM ($p = 0.009$), with the CM group differing significantly from LFEM on Tukey HSD post-hoc testing. Language impairment subscores increased similarly across groups (LFEM: 2.2 ± 2.4 , HFEM: 2.5 ± 2.4 , CM: 2.9 ± 2.3 ; $p = 0.006$), again with a significant pairwise difference between CM and LFEM. Executive dysfunction subscores differed more modestly across groups (LFEM: 4.9 ± 3.1 , HFEM: 5.2 ± 3.0 , CM: 5.6 ± 2.8 ; $p = 0.036$) without significant pairwise differences after post-hoc correction. Notably, the relative contribution of language impairment to total cognitive burden increased with migraine frequency (LFEM: $22.4\% \pm 18.9$, HFEM: $25.3\% \pm 19.1$, CM: $28.8\% \pm 19.5$; $p = 0.003$), while the executive dysfunction contribution did not differ significantly across groups ($p = 0.139$).

Displays total cognitive impairment burden among patients diagnosed with chronic migraine by clinicians per ICHD-3 criteria ($n = 661$). Percentages reflect proportion of the chronic migraine stratum ($n = 661$). Group sizes: 1–2 months, $n = 27$ (4.1%); 3–12 months, $n = 79$ (12.0%); 1–2 years, $n = 78$ (11.8%); 3–5 years, $n = 129$ (19.5%); 5–10 years, $n = 151$ (22.8%); >10 years, $n = 197$ (29.8%). Mig-SCog = Migraine-Specific Cognitive Impairment Scale. Points represent Mig-SCog total score group means; error bars represent 95% confidence intervals. The dashed vertical line demarcates the early chronicity phase (≤ 12 months) from the phenotypic plateau phase (> 12 months), shown in gray. One-way analysis of variance across the six duration categories approached but did not reach significance ($F[5, 655] = 2.17$, $p = 0.056$). Pairwise post-hoc comparisons were performed using the Games-Howell test. Participants in the 1–2 month group ($n = 27$, 4.1%) reported significantly lower Mig-SCog scores than those in the 1–2 year ($n = 78$, 11.8%; $p = 0.044$, $d = 0.61$), 3–5 year ($n = 129$, 19.5%; $p = 0.018$, $d = 0.63$), and > 10 year ($n = 197$, 29.8%; $p = 0.048$, $d = 0.55$) groups; all pairwise effect sizes were in the medium range. The 3–12-month group ($n = 79$, 12.0%) did not differ significantly from any longer-duration group (all $p > 0.39$), suggesting cognitive burden may begin to stabilize within the first year of chronic migraine. No significant pairwise differences were observed among groups with > 12 months of chronic migraine duration. A pre-specified binary contrast confirmed significantly lower Mig-SCog scores among participants with ≤ 12 months versus > 12 months of chronic migraine duration (7.7 ± 4.9 vs 9.0 ± 4.6 ; $p = 0.010$, $d = 0.27$). Effect sizes reported as Cohen's d , calculated using the pooled within-group standard deviation derived from the ANOVA mean square error; interpreted as small (≥ 0.2), medium (≥ 0.5), and large (≥ 0.8).

Among patients with clinician-confirmed CM and available CM duration data ($n = 661$), cognitive burden was lowest in the earliest phase of chronic migraine and appeared to plateau after approximately 12 months (Fig. 1). Patients in the 1–2 month CM duration group (27/661, 4.1%) reported significantly lower Mig-SCog scores than those in the 1–2 year group (78/661, 11.8%; $p = 0.044$, $d = 0.61$), 3–5 year group (129/661, 19.5%; $p = 0.018$, $d = 0.63$), and > 10 year group (197/661, 29.8%; $p = 0.048$, $d = 0.55$), with all pairwise effect sizes in the medium range on Games-Howell post-hoc testing. No significant pairwise differences were observed among groups with > 12 months of CM duration. In a pre-specified binary contrast, patients with ≤ 12 months of CM duration reported lower Mig-SCog scores than those with > 12 months (7.7 ± 4.9 vs 9.0 ± 4.6 ; $p = 0.010$, $d = 0.27$, small effect). The overall one-way ANOVA across six duration categories approached but did not reach statistical significance ($F[5,655] = 2.17$, $p = 0.056$).

Table 5
Association between migraine frequency classification and cognitive impairment: adjusted linear regression

Predictor	B (SE)	β	t	p-value
Migraine Frequency				
HFEM (vs. LFEM)	-0.5 (0.7)	-0.04	-0.8	0.451
CM (vs. LFEM)	0.3 (0.6)	0.03	0.4	0.658
Demographics				
Age	-0.0 (0.0)	-0.12	-3.1	0.002* [†]
Sex (Male)	-1.3 (0.5)	-0.09	-2.7	0.007*
Clinical Covariates				
Disease Duration (Years)	0.0 (0.0)	0.0	0.1	0.922
Pain Intensity (NRS)	0.2 (0.1)	0.05	1.7	0.094
Psychological Factors				
Anxiety Score (GAD-7)	0.1 (0.0)	0.15	3.6	<0.001* [†]
Depression Score (PHQ-2)	0.4 (0.1)	0.13	3.1	0.002* [†]

Model Statistics: Adjusted $R^2 = 0.106$; $p < 0.001$.

LFEM (low-frequency episodic migraine; 0–7 headache days/month) is the reference category. HFEM: high-frequency episodic migraine (≥ 8 headache days/month without a diagnosis of chronic migraine per ICHD-3 criteria); CM: chronic migraine (clinician-diagnosed per ICHD-3 criteria). GAD-7: Generalized Anxiety Disorder 7-item Scale. PHQ-2: Patient Health Questionnaire-2. NRS: Numeric Rating Scale. B: unstandardized regression

coefficient; SE: standard error; β : standardized coefficient. Model adjusted for migraine frequency classification, age, sex, disease duration, pain intensity, GAD-7 anxiety score, and PHQ-2 depression score. *: significant at $p < 0.05$. [†]: significant after adjustments.

In the adjusted linear regression model, migraine frequency classification was not independently associated with Mig-SCog total score after accounting for age, sex, migraine disease duration, depression score, and anxiety score (Table 5). Compared with LFEM, neither HFEM ($B = -0.5$, $SE = 0.7$, $\beta = -0.04$, $p = 0.451$) nor CM ($B = 0.3$, $SE = 0.6$, $\beta = 0.03$, $p = 0.658$) significantly predicted Mig-SCog total score. By contrast, higher GAD-7 anxiety score ($B = 0.1$, $SE < 0.1$, $\beta = 0.15$, $p < 0.001$) and higher PHQ-2 depression score ($B = 0.4$, $SE = 0.1$, $\beta = 0.13$, $p = 0.002$) were both independently associated with greater cognitive burden, and with standardized coefficients exceeding those of all migraine frequency classification categories. Younger age was also independently associated with greater Mig-SCog burden ($\beta = -0.12$, $p = 0.002$), and male sex was associated with lower Mig-SCog total score ($B = -1.3$, $SE = 0.5$, $\beta = -0.09$, $p = 0.007$). The overall model significantly predicted Mig-SCog total score (adjusted $R^2 = 0.106$, $p < 0.001$).

Table 6
Association between ictal migraine-related symptom frequency and subjective cognitive impairment burden

SYMPTOM	Never (Mean ± SD; n (%))	Rarely (Mean ± SD; n (%))	Less than half the time (Mean ± SD; n (%))	Half the time or more (Mean ± SD; n (%))	Always (Mean ± SD; n (%))	Adjusted p-value
COGNITIVE						
Difficulty finding words, expressing yourself verbally or in writing, or fully understanding what others are saying	3.29 ± 2.57; 245 (20.8%)	5.92 ± 3.17; 231 (19.6%)	8.27 ± 3.54; 264 (22.4%)	11.00 ± 3.81; 302 (25.6%)	13.64 ± 3.62; 137 (11.6%)	< .001* ^{†C}
Routine mental activity worsens headache	4.90 ± 3.90; 63 (5.4%)	5.78 ± 4.00; 165 (14.0%)	6.66 ± 4.26; 203 (17.2%)	8.82 ± 4.65; 420 (35.7%)	9.87 ± 4.75; 326 (27.7%)	< .001* ^{†B}
MOOD						
Restlessness/agitation/irritability	7.00 ± 4.47; 639 (54.5%)	8.55 ± 4.56; 198 (16.9%)	9.00 ± 4.73; 167 (14.2%)	10.77 ± 4.93; 124 (10.6%)	10.25 ± 4.91; 44 (3.8%)	< .001* ^{†A}
AUTONOMIC & GASTROINTESTINAL						
Thermal dysregulation	5.89 ± 4.42; 224 (19.1%)	6.73 ± 4.33; 216 (18.4%)	8.52 ± 4.25; 275 (23.4%)	9.32 ± 5.01; 321 (27.4%)	9.98 ± 4.63; 137 (11.7%)	< .001* ^{†B}
Nausea	5.52 ± 4.55; 56 (4.7%)	6.63 ± 4.44; 265 (22.4%)	8.32 ± 4.56; 308 (26.0%)	8.67 ± 4.81; 384 (32.5%)	9.52 ± 4.81; 170 (14.4%)	< .001* ^{†A}
Non-vertigo dizziness (unsteadiness, lightheadedness, motion sickness)	4.65 ± 3.94; 122 (10.3%)	6.35 ± 4.43; 280 (23.7%)	8.02 ± 4.24; 293 (24.8%)	9.43 ± 4.50; 334 (28.3%)	11.28 ± 4.55; 152 (12.9%)	< .001* ^{†B}
Vertigo	5.77 ± 4.18; 330 (28.0%)	8.04 ± 4.67; 399 (33.8%)	9.07 ± 4.49; 228 (19.3%)	10.46 ± 4.31; 170 (14.4%)	11.30 ± 4.80; 53 (4.5%)	< .001* ^{†B}
SENSORY						
Light hypersensitivity	4.06 ± 3.64; 34 (2.9%)	5.62 ± 4.19; 115 (9.7%)	7.35 ± 4.87; 139 (11.8%)	8.13 ± 4.79; 348 (29.5%)	9.05 ± 4.57; 544 (46.1%)	< .001* ^{†B}
Sound hypersensitivity	4.47 ± 3.58; 45 (3.8%)	5.85 ± 4.24; 129 (11.0%)	6.96 ± 4.47; 158 (13.4%)	8.15 ± 4.76; 345 (29.3%)	9.33 ± 4.67; 500 (42.5%)	< .001* ^{†B}
Odor hypersensitivity	6.09 ± 4.54; 219 (18.6%)	7.23 ± 4.55; 201 (17.1%)	8.39 ± 4.62; 175 (14.9%)	8.56 ± 4.71; 268 (22.8%)	9.49 ± 4.69; 315 (26.7%)	< .001* ^{†A}
SOMATIC & FOCAL NEUROLOGIC SYMPTOMS						

SYMPTOM	Never (Mean ± SD; n (%))	Rarely (Mean ± SD; n (%))	Less than half the time (Mean ± SD; n (%))	Half the time or more (Mean ± SD; n (%))	Always (Mean ± SD; n (%))	Adjusted p-value
One-sided numbness	7.10 ± 4.45; 779 (66.2%)	9.07 ± 4.56; 173 (14.7%)	10.05 ± 4.71; 111 (9.4%)	11.15 ± 4.89; 91 (7.7%)	12.87 ± 4.26; 23 (2.0%)	< .001* ^{†B}
One-sided weakness	6.93 ± 4.41; 763 (64.8%)	9.09 ± 4.55; 203 (17.2%)	10.11 ± 4.26; 96 (8.1%)	11.94 ± 4.61; 84 (7.1%)	12.97 ± 4.83; 32 (2.7%)	< .001* ^{†B}

Mig-SCog total cognitive impairment scores across ordinal ictal symptom-frequency categories. Values are reported as mean ± SD; n (%) reflects the proportion of participants in each symptom-specific analytic sample reporting that symptom. Statistical significance and partial eta squared values were derived from general linear models adjusted for age, sex, headache frequency (days per month), headache disease duration (years), Patient Health Questionnaire-2 depression symptom score, and Generalized Anxiety Disorder-7 anxiety symptom score. *p < 0.05. [†]: significant after adjustments. Superscripts denote adjusted partial eta squared effect-size: A: small, B: medium, C: large. Mig-SCog: Migraine-Specific Cognitive Impairment Scale.

Mean Mig-SCog total cognitive impairment scores are shown across ordinal ictal symptom-frequency categories for selected migraine-associated symptoms. Symptoms are grouped by clinical domain: cognitive and mood, autonomic and gastrointestinal, sensory hypersensitivity, and focal neurologic. Each point represents the mean Mig-SCog score for participants reporting the corresponding symptom frequency category. Bubble size reflects the proportion of participants within each symptom row reporting that frequency category. Lines connect mean scores across symptom-frequency categories for each symptom. Effect sizes are reported as partial eta squared (η^2) from unadjusted univariate ANOVA models.

Patient-reported ictal symptom frequency was significantly associated with Mig-SCog total score across all symptom categories examined (Table 6, Fig. 2). The largest effect size was observed for the symptom “difficulty finding words, expressing oneself verbally or in writing, or fully understanding what others were saying,” assessed as a single Mig-SCog item, with mean total cognitive symptom scores rising from 3.3 ± 2.6 among patients who never reported this symptom (245/1,179, 20.8%) to 13.6 ± 3.6 among those who always reported it (137/1,179, 11.6%; adjusted p < 0.001, large effect). Routine mental activity worsening migraine showed a medium-effect association (adjusted p < 0.001), with scores rising from 4.9 ± 3.9 to 9.9 ± 4.8 across frequency categories. Among vestibular symptoms, both non-vertigo dizziness and vertigo showed medium-effect associations (adjusted p < 0.001 for both); scores for non-vertigo dizziness rose from 4.7 ± 3.9 among patients never reporting the symptom (122/1,181, 10.3%) to 11.3 ± 4.6 among those always reporting it (152/1,181, 12.9%). Thermal dysregulation showed a medium-effect association with Mig-SCog total score (adjusted p < 0.001), with mean scores rising from 5.9 ± 4.4 among patients who never reported it (224/1,177, 19.1%) to 10.0 ± 4.6 among those who always reported it (137/1,177, 11.7%). Focal neurologic symptoms were associated with the highest Mig-SCog scores in the highest-frequency categories, although these groups were small: patients who always reported ‘one-sided numbness’ (23/1,177, 2.0%) had a mean score of 12.9 ± 4.3 , and those who always reported ‘one-sided weakness’ (32/1,178, 2.7%) had a mean score of 13.0 ± 4.8 (both adjusted p < 0.001, medium effects). Light and sound hypersensitivity each showed medium-effect associations, and odor hypersensitivity a small-effect association (all adjusted p < 0.001).

Table 7
Total cognitive impairment by symptom endorsement across phases of migraine attack

SYMPTOM CATEGORY	Prodromal Without Sx (Mean $\pm$ SD; n (%))	Prodromal With Sx (Mean $\pm$ SD; n (%))	p-value	Ictal Without Sx (Mean $\pm$ SD; n (%))	Ictal With Sx (Mean $\pm$ SD; n (%))	p-value	Postdromal Without Sx (Mean $\pm$ SD; n (%))	Postdromal With Sx (Mean $\pm$ SD; n (%))	p-value
COGNITIVE									
Impaired concentration	7.1 $\pm$ 4.5; n = 734 (68.4%) + 1	10.8 $\pm$ 4.5; n = 339 (31.6%) + 1	< 0.001 ^{†C}	--	--	--	6.9 $\pm$ 4.4; n = 669 (63.6%)	10.9 $\pm$ 4.4; n = 383 (36.4%)	< 0.001 ^{†C}
Difficulty finding words, expressing yourself verbally in writing, or fully understanding what others are saying	7.6 $\pm$ 4.5; n = 915 (85.3%)	12.2 $\pm$ 4.2; n = 158 (14.7%)	< 0.001 ^{†C}	3.3 $\pm$ 2.6; n = 245 (20.8%)	9.4 $\pm$ 4.4; n = 934 (79.2%)	< 0.001 ^{†C}	7.8 $\pm$ 4.6; n = 946 (89.9%)	12.9 $\pm$ 4.0; n = 106 (10.1%)	< 0.001 ^{†C}
Routine mental activity worsens headache	--	--	--	4.9 $\pm$ 3.9; n = 63 (5.4%)	8.3 $\pm$ 4.8; n = 1114 (94.6%)	< 0.001 ^{†B}	--	--	--
MOOD									
Depression	8.0 $\pm$ 4.7; n = 949 (88.4%)	10.7 $\pm$ 4.5; n = 124 (11.6%)	< 0.001 ^{†B}	--	--	--	7.9 $\pm$ 4.7; n = 878 (83.5%)	10.5 $\pm$ 4.7; n = 174 (16.5%) ^B	< 0.001 ^{†B}
Anxiety	7.8 $\pm$ 4.7; n = 837 (78.0%)	10.0 $\pm$ 4.6; n = 236 (22.0%)	< 0.001 ^{†A}	--	--	--	8.0 $\pm$ 4.7; n = 906 (86.1%)	10.6 $\pm$ 4.9; n = 146 (13.9%)	< 0.001 ^{†B}
Restlessness/agitation/irritability	7.6 $\pm$ 4.7; n = 700 (65.2%)	9.6 $\pm$ 4.6; n = 373 (34.8%)	< 0.001 ^{†C}	7.0 $\pm$ 4.5; n = 639 (54.5%)	9.3 $\pm$ 4.8; n = 533 (45.5%)	< 0.001 ^{†B}	7.9 $\pm$ 4.8; n = 831 (79.0%)	9.9 $\pm$ 4.6; n = 221 (21.0%)	< 0.001 ^{†A}
Euphoria	8.1 $\pm$ 4.7; n = 1010 (94.1%)	11.5 $\pm$ 4.6; n = 63 (5.9%)	< 0.001 ^{†B}	--	--	--	8.3 $\pm$ 4.8; n = 1019 (96.9%)	9.6 $\pm$ 5.3; n = 33 (3.1%)	0.121 ^A
AUTONOMIC / GI									
Drowsiness	7.2 $\pm$ 4.5; n = 731 (68.1%)	10.5 $\pm$ 4.6; n = 342 (31.9%)	< 0.001 ^{†B}	--	--	--	6.8 $\pm$ 4.5; n = 507 (48.2%)	9.8 $\pm$ 4.6; n = 545 (51.8%)	< 0.001 ^{†B}
Yawning	8.0 $\pm$ 4.7; n = 869 (81.0%)	9.6 $\pm$ 4.8; n = 204 (19.0%)	< 0.001 ^{†A}	--	--	--	8.2 $\pm$ 4.8; n = 976 (92.8%)	9.5 $\pm$ 5.1; n = 76 (7.2%)	0.029 ^A
Thermal dysregulation	7.9 $\pm$ 4.7; n = 857 (79.9%)	9.6 $\pm$ 4.8; n = 216 (20.1%)	< 0.001 ^{†A}	5.9 $\pm$ 4.4; n = 224 (19.1%)	8.6 $\pm$ 4.7; n = 949 (80.9%)	< 0.001 ^{†B}	8.1 $\pm$ 4.8; n = 956 (90.9%)	10.8 $\pm$ 4.4; n = 96 (9.1%)	< 0.001 ^{†B}
Nausea	8.0 $\pm$ 4.7; n = 814 (75.9%)	9.0 $\pm$ 4.9; n = 259 (24.1%)	0.004 ^A	5.5 $\pm$ 4.5; n = 56 (4.7%)	8.2 $\pm$ 4.7; n = 1127 (95.3%)	< 0.001 ^{†B}	8.2 $\pm$ 4.8; n = 931 (88.5%)	9.6 $\pm$ 4.8; n = 121 (11.5%)	0.001 ^{†A}
Appetite loss	8.0 $\pm$ 4.7; n = 969 (90.3%)	10.5 $\pm$ 4.9; n = 104 (9.7%)	< 0.001 ^{†B}	--	--	--	8.1 $\pm$ 4.7; n = 950 (90.3%)	10.5 $\pm$ 4.9; n = 102 (9.7%)	< 0.001 ^{†B}
Food craving	8.0 $\pm$ 4.7; n = 971 (90.5%)	11.0 $\pm$ 4.5; n = 102 (9.5%)	< 0.001 ^{†B}	--	--	--	8.2 $\pm$ 4.7; n = 986 (93.7%)	9.8 $\pm$ 5.2; n = 66 (6.3%)	0.009 ^A
Vomiting	--	--	--	7.1 $\pm$ 4.6; n = 429 (36.3%)	8.6 $\pm$ 4.8; n = 753 (63.7%)	< 0.001 ^{†A}	--	--	--
Non-vertigo dizziness (unsteadiness, lightheadedness, motion sickness)	--	--	--	4.6 $\pm$ 3.9; n = 122 (10.3%)	8.5 $\pm$ 4.7; n = 1059 (89.7%)	< 0.001 ^{†C}	--	--	--

SYMPTOM CATEGORY	Prodromal Without Sx (Mean $\pm$ SD; n (%))	Prodromal With Sx (Mean $\pm$ SD; n (%))	p-value	Ictal Without Sx (Mean $\pm$ SD; n (%))	Ictal With Sx (Mean $\pm$ SD; n (%))	p-value	Postdromal Without Sx (Mean $\pm$ SD; n (%))	Postdromal With Sx (Mean $\pm$ SD; n (%))	p-value
Vertigo	--	--	--	5.8 $\pm$ 4.2; n = 330 (28.0%)	9.0 $\pm$ 4.7; n = 850 (72.0%)	< 0.001 ^{†B}	--	--	--
SENSORY									
Light hypersensitivity	7.3 $\pm$ 4.7; n = 527 (49.1%)	9.2 $\pm$ 4.7; n = 546 (50.9%)	< 0.001 ^{†A}	4.1 $\pm$ 3.6; n = 34 (2.9%)	8.2 $\pm$ 4.7; n = 1146 (97.1%)	< 0.001 ^{†C}	7.8 $\pm$ 4.7; n = 750 (71.3%)	9.7 $\pm$ 4.7; n = 302 (28.7%)	< 0.001 ^{†A}
Sound hypersensitivity	7.6 $\pm$ 4.7; n = 693 (64.6%)	9.5 $\pm$ 4.8; n = 380 (35.4%)	< 0.001 ^{†A}	4.5 $\pm$ 3.6; n = 45 (3.8%)	8.2 $\pm$ 4.8; n = 1132 (96.2%)	< 0.001 ^{†B}	7.7 $\pm$ 4.6; n = 807 (76.7%)	10.4 $\pm$ 4.8; n = 245 (23.3%)	< 0.001 ^{†B}
Odor hypersensitivity	7.7 $\pm$ 4.6; n = 775 (72.2%)	9.7 $\pm$ 4.9; n = 298 (27.8%)	< 0.001 ^{†A}	6.1 $\pm$ 4.5; n = 219 (18.6%)	8.6 $\pm$ 4.7; n = 959 (81.4%)	< 0.001 ^{†B}	8.0 $\pm$ 4.7; n = 879 (83.6%)	10.2 $\pm$ 5.0; n = 173 (16.4%)	< 0.001 ^{†A}
Skin hypersensitivity	8.0 $\pm$ 4.7; n = 973 (90.7%)	11.2 $\pm$ 4.3; n = 100 (9.3%)	< 0.001 ^{†B}	--	--	--	8.0 $\pm$ 4.7; n = 967 (91.9%)	11.7 $\pm$ 4.2; n = 85 (8.1%)	< 0.001 ^{†B}
SOMATIC / FOCAL NEUROLOGIC SYMPTOMS									
Neck pain	--	--	--	5.8 $\pm$ 5.2; n = 69 (5.8%)	8.2 $\pm$ 4.7; n = 1111 (94.2%)	< 0.001 ^{†B}	--	--	--
One-sided numbness	--	--	--	7.1 $\pm$ 4.5; n = 779 (66.2%)	10.0 $\pm$ 4.8; n = 398 (33.8%)	< 0.001 ^{†B}	--	--	--
One-sided weakness	--	--	--	6.9 $\pm$ 4.4; n = 763 (64.8%)	10.2 $\pm$ 4.7; n = 415 (35.2%)	< 0.001 ^{†B}	--	--	--

Table 7 shows Mig-SCog total cognitive impairment scores among participants with versus without migraine-associated symptoms across the temporal evolution of the migraine attack, including prodromal, ictal, and postdromal phases. Ictal symptoms originally assessed on an ordinal frequency scale were dichotomized as absent (“never”) versus present (any reported frequency). Values are reported as mean $\pm$ SD; n (%) reflects the proportion of participants within each phase-specific symptom sample with versus without the symptom. P-values compare Mig-SCog total scores between participants with versus without each symptom within the corresponding attack phase. Superscripts denote effect-size magnitude for the phase-specific between-group comparison using Cohen’s d: A: small, B: medium, C: large. Mig-SCog: Migraine-Specific Cognitive Impairment Scale. [†]: significant after adjustments.

Figure 3. Increase in cognitive burden associated with symptoms across the prodrome and postdrome

Horizontal bars show the mean difference in Mig-SCog total cognitive impairment score between participants with versus without each symptom during the prodromal and postdromal migraine attack phases. Values represent the absolute point increase in mean Mig-SCog score associated with symptom endorsement within each phase.

Cognitive and language symptoms showed the largest point increases in Mig-SCog total score associated with symptom endorsement across migraine attack phases (Table 7, Fig. 3). During the prodrome, patients reporting “difficulty finding words” had Mig-SCog scores 4.6 points higher than those without this symptom (12.2 $\pm$ 4.2 [158/1,073, 14.7%] vs. 7.6 $\pm$ 4.5 [915/1,073, 85.3%]; $p < 0.001$, large effect), and those reporting “impaired concentration” had scores 3.7 points higher than those without it (10.8 $\pm$ 4.5 [339/1,073, 31.6%] vs. 7.1 $\pm$ 4.5 [734/1,073, 68.4%]; $p < 0.001$, large effect). During the ictal phase, “difficulty finding words” again showed the largest contrast, with patients endorsing this symptom scoring 6.1 points higher than those not endorsing it (9.4 $\pm$ 4.4 [934/1,179, 79.2%] vs. 3.3 $\pm$ 2.6 [245/1,179, 20.8%]; $p < 0.001$, large effect), and “non-vertigo dizziness” was associated with a 3.9-point higher score among patients endorsing it (8.5 $\pm$ 4.7 [1,059/1,181, 89.7%] vs. 4.6 $\pm$ 3.9 [122/1,181, 10.3%]; $p < 0.001$, large effect). During the postdrome, “difficulty finding words” again showed the largest between-group difference, with patients endorsing this symptom scoring 5.1 points higher than those not endorsing it (12.9 $\pm$ 4.0 [106/1,052, 10.1%] vs. 7.8 $\pm$ 4.6 [946/1,052, 89.9%]; $p < 0.001$, large effect); patients endorsing “impaired concentration” scored 4.0 points higher than those without it (10.9 $\pm$ 4.4 [383/1,052, 36.4%] vs. 6.9 $\pm$ 4.4 [669/1,052, 63.6%]; $p < 0.001$, large effect), and

those endorsing postdromal “skin hypersensitivity” scored 3.7 points higher than those without it (11.7 ± 4.2 [85/1,052, 8.1%] vs. 8.0 ± 4.7 [967/1,052, 91.9%]; $p < 0.001$, medium effect).

Among mood symptoms, prodromal and postdromal depression, anxiety, and restlessness/agitation/irritability were each associated with higher Mig-SCog scores (Table 7). Euphoria showed a phase-specific pattern, and patients reporting prodromal euphoria had Mig-SCog scores 3.4 points higher than those without it (11.5 ± 4.6 [63/1,073, 5.9%] vs. 8.1 ± 4.7 [1,010/1,073, 94.1%]; $p < 0.001$, medium effect), whereas postdromal euphoria was not significantly associated with cognitive symptom burden (9.6 ± 5.3 [33/1,052, 3.1%] vs. 8.3 ± 4.8 [1,019/1,052, 96.9%]; $p = 0.121$). Euphoria was the only mood symptom listed in Table 7 that was significantly associated with cognitive burden during the prodrome but not during the postdrome.

Table 8 Association between sensory hypersensitivity and cognitive impairment during migraine attacks				
Ictal-phase sensory hypersensitivity score	Unadjusted r	Adjusted B (SE)	β	p-value
Sound hypersensitivity	0.45	0.12 (0.02)	0.25	$< 0.001^{*†}$
Skin hypersensitivity	0.39	0.21 (0.03)	0.2	$< 0.001^{*†}$
Light hypersensitivity	0.32	1.19 (0.52)	0.08	0.021*

Table 8 shows the relationship between validated sensory hypersensitivity scores and Mig-SCog total cognitive impairment score. Unadjusted correlations reflect bivariate associations between each sensory hypersensitivity measure and Mig-SCog total score. Adjusted coefficients were derived from a single multivariable linear regression model including all sensory hypersensitivity scores simultaneously as continuous predictors, with Mig-SCog total score as the dependent variable, adjusting for age, sex, headache frequency, headache disease duration, Patient Health Questionnaire-2 depression symptom score, and Generalized Anxiety Disorder-7 anxiety symptom score. * $p < 0.05$. Mig-SCog: Migraine-Specific Cognitive Impairment Scale. †: significant after adjustments.

Validated sensory hypersensitivity scores were each positively correlated with Mig-SCog total score in unadjusted analyses, with sound hypersensitivity showing the strongest association ($r = 0.45$), followed by skin hypersensitivity ($r = 0.39$) and light hypersensitivity ($r = 0.32$; Table 8). A multivariable model including all three sensory hypersensitivity scores simultaneously and adjusted for age, sex, monthly migraine frequency, migraine disease duration, PHQ-2 depression score, and GAD-7 anxiety score, evaluated sound hypersensitivity ($B = 0.12$, $SE = 0.02$, $\beta = 0.25$, $p < 0.001$, small effect) and skin hypersensitivity ($B = 0.21$, $SE = 0.03$, $\beta = 0.20$, $p < 0.001$, small effect), and both remained independently associated with greater ictal cognitive impairment. Light hypersensitivity also remained independently associated with Mig-SCog total score, although with a smaller standardized coefficient ($B = 1.19$, $SE = 0.52$, $\beta = 0.08$, $p = 0.021$); this association should be interpreted with caution, as light hypersensitivity was endorsed by 97.1% of patients in the symptom-specific sample, leaving a comparison group of only 34 patients without this symptom and limiting the interpretability of its independent contribution in this model.

DISCUSSION

Ictal cognitive impairment, although frequently reported and substantially disabling during migraine attacks, remains under-characterized in clinical practice relative to cardinal symptoms such as headache and photophobia. This study cohort of 1,231 patients with migraine diagnosed by headache specialists using ICHD-3 criteria, and 95.5% endorsed some degree of subjective cognitive dysfunction during attacks, a prevalence consistent with prior work demonstrating that cognitive complaints during the ictal phase are nearly universal in clinically-ascertained migraine populations.^{5,6} This has a direct clinical implication, that cognitive symptoms are not an occasional complaint or an incidental finding in patients with migraine. They are a near-universal feature of the disease in specialty care populations, and they warrant the same systematic inquiry that headache severity and attack frequency do. This study includes one of the largest cohorts to use the Mig-SCog; the concordance between total cognitive symptom scores and related peri-attack symptom measures, particularly prodromal and postdromal word-finding difficulty and impaired concentration, provides additional real-world support for the instrument's construct validity in a substantially larger cohort than the original validation study.⁵

The distribution of cognitive burden was not uniform across the migraine frequency spectrum, as unadjusted cognitive symptoms increased across LFEM, HFEM, and CM, with post-hoc testing showing a significant difference between CM and LFEM. Importantly, the relative contribution of language impairment to total cognitive burden increased progressively with migraine frequency, from 22.4% in LFEM to 28.8% in CM, while executive dysfunction contribution did not differ across groups. This shift is clinically meaningful, because in patients with more chronic migraine, language-related symptoms such as word-finding difficulty, impaired verbal expression, and language comprehension represent a more prominent component of ictal burden. Clinicians who ask only about attention and memory may underestimate the degree to which patients with chronic migraine are struggling with language-specific cognitive demands at work and in daily communication.

A central finding of this study is that migraine frequency classification alone does not adequately explain the distribution of cognitive impairment in patients with migraine. After accounting for age, sex, migraine disease duration, anxiety symptoms, and depression symptoms, neither HFEM nor CM significantly predicted total cognitive symptoms relative to LFEM. The strongest independent predictors were anxiety and depression symptom scores, with standardized coefficients exceeding those of all migraine frequency classification categories (anxiety: $\beta = 0.15$; depression: $\beta = 0.13$). This pattern held consistently across multiple levels of analysis. In unadjusted bivariate correlations, depression and anxiety symptom scores demonstrated the

largest observed associations with total cognitive symptoms and executive dysfunction of any continuous clinical variable examined, with the relationship between psychiatric symptom burden and cognitive impairment more strongly expressed through executive dysfunction than through language impairment, a distinction worth examining in future work given well-established links between depression, anxiety, and prefrontal network function.^{24,25} Restlessness, agitation, and irritability were significantly associated with higher cognitive burden across ordinal frequency categories in ictal symptom-frequency analyses. In analyses that considered the phase of migraine attacks when symptoms were occurring, prodromal and postdromal depression, anxiety, and restlessness were each associated with substantially higher total cognitive symptom scores, persisting after the migraine attack's headache phase had resolved. Taken together, affective dysregulation during migraine, whether captured as a validated psychiatric comorbidity, a subjective ictal symptom, or a peri-attack mood feature, tracked cognitive burden more consistently than migraine frequency classification in this cohort. This does not render attack frequency irrelevant to cognition, as unadjusted group comparisons were directionally consistent with prior literature showing worse cognitive performance with greater migraine burden.^{8,26} Rather, these findings raise the question of whether the apparent cognitive burden of chronic migraine is partially mediated or confounded by the substantially higher rates of anxiety and depression that accompany chronification.^{24,25} The direction of this association cannot be established from cross-sectional data, as elevated anxiety and depression may arise as consequences of chronic migraine rather than acting as sole contributors to cognitive burden. The practical clinical implication is specific, that a patient with LFEM and high anxiety and depression may report worse cognitive burden than a patient with CM who does not. Migraine frequency classification alone cannot predict cognitive burden, and clinicians who rely on headache diary data alone to assess disease severity will be unlikely to accurately estimate cognitive burden. Cognitive complaints should prompt systematic assessment of the broader affective migraine phenotype, including baseline anxiety and depression and peri-attack mood symptoms, because these measures tracked cognitive burden more consistently than migraine frequency classification alone in this cohort. These findings support routine screening for anxiety and depression when patients report cognitive symptoms. Future longitudinal and interventional studies should determine whether treating these comorbidities reduces perceived cognitive burden.

Within the chronic migraine stratum, cognitive burden was lower among patients within the first 12 months of chronic migraine than among those with longer-standing chronic migraine, with no significant differences observed among groups beyond 12 months of chronic migraine duration. Patients with 12 months or less of chronic migraine duration reported lower total cognitive symptoms than those with more than 12 months in the pre-specified binary contrast (7.7 ± 4.9 vs. 9.0 ± 4.6 ; $p = 0.010$, $d = 0.27$). This pattern is consistent with, but does not prove, a threshold-plus-plateau model, in which the transition into chronic migraine represents the most dynamic window for cognitive impact rather than the start of a period of ongoing progressive deterioration. Because cognitive burden did not continue to rise across years of established chronic migraine, the largest differences appear concentrated around the transition into chronic migraine. This study did not assess preventive treatment, so any implications for the timing of prevention remain hypotheses for future longitudinal work. This should not be interpreted as evidence of longitudinal stabilization within individual patients as cross-sectional data cannot establish how cognitive burden evolves within a given person over time, and longitudinal studies with repeated assessments across the chronification trajectory are needed to confirm this pattern. Whether the observed plateau reflects compensatory neuroplasticity, habituation of pain-cognitive interference, or survivor selection effects is unknown. It is, however, consistent with neuroimaging evidence that structural and functional brain changes in migraine are not strictly proportional to disease duration, with some changes appearing relatively early in the chronification process.^{27,28} Prior longitudinal studies have not consistently supported a model of steadily progressive cognitive decline attributable to migraine alone, although evidence regarding long-term cognitive outcomes and dementia risk remains mixed, particularly in studies considering migraine with aura.^{29–31} These findings should be interpreted with appropriate caution given that the overall ANOVA across six duration categories did not reach statistical significance ($p = 0.056$), and the 1–2 month group was small (27/661, 4.1%).

Among the ordinal ictal symptom-frequency variables examined, "difficulty finding words, expressing oneself verbally or in writing, or fully understanding what others were saying," assessed as a single item, demonstrated the largest effect size and the largest absolute score difference, with total cognitive symptoms 10.3 points higher in patients who always reported this symptom relative to those who never reported it (13.6 ± 3.6 vs. 3.3 ± 2.6). The increasing relative contribution of language impairment to total cognitive burden across LFEM, HFEM, and CM further suggests that language networks carry particular sensitivity to migraine disease burden, consistent with neuropsychological and functional imaging data demonstrating disruption of distributed frontotemporal networks during the ictal migraine state.^{32,33} More broadly, the symptom-frequency analyses suggested a possible neuroanatomical gradient in cognitive burden in which symptoms plausibly reflecting cortical network involvement, including word-finding difficulty, one-sided weakness, and one-sided numbness, demonstrate the largest associations with cognitive symptoms, while symptoms often linked to brainstem or autonomic pathways, such as nausea and vomiting, show comparatively weaker associations despite their central role in ICHD-3 migraine criteria. This dissociation raises the possibility that subjective cognitive impairment during migraine attacks may not be a non-specific consequence of feeling systemically unwell, but may instead reflect greater involvement of cortical networks during attacks.³³ Language difficulty, focal motor symptoms, and sensory disturbances of the extremities also overlap with classical features of cortical aura, raising the possibility that cortical spreading depolarization may contribute to the most cognitively disruptive ictal experiences in some patients, although one-sided weakness and numbness were always reported by small subgroups (2.0% and 2.7% of respective samples), and this hypothesis requires prospective investigation with aura-confirmed phenotyping.³⁴

Vestibular symptoms represented a notable intermediate, as non-vertigo dizziness and vertigo were each associated with total cognitive symptom scores more than 5 points higher in patients who always reported them relative to those who never did, despite being classically interpreted as having brainstem localization. This may reflect the requirement for multimodal cortical integration in the parieto-insular vestibular cortex during vestibular

processing, such that vestibular symptoms during migraine may index cortical involvement beyond what their traditional localization would suggest.^{35,36}

Phase-based analyses demonstrated that cognitive burden associated with migraine is not confined to the ictal phase. These findings align closely with patient-centered qualitative data from the MiCOAS project, in which Gerstein and colleagues found that cognitive symptoms were reported during the pre-headache phase by 90% of participants, during the headache phase by 88%, during the post-headache phase by 68%, and even interictally by 33%, with language and speech, sustained attention, executive function, and memory identified as major affected domains across the migraine attack cycle.⁹ In the present cohort, the consistency of findings across the prodrome and postdrome reinforces that interpretation. Patients reporting prodromal word-finding difficulty scored 4.6 points higher on total cognitive symptoms than those without this symptom (12.2 ± 4.2 vs. 7.6 ± 4.5 ; $p < 0.001$, large effect), and patients reporting postdromal word-finding difficulty scored 5.1 points higher (12.9 ± 4.0 vs. 7.8 ± 4.6 ; $p < 0.001$, large effect). Impaired concentration showed a parallel pattern, associated with 3.7-point higher scores during the prodrome (10.8 ± 4.5 vs. 7.1 ± 4.5 ; $p < 0.001$, large effect) and 4.0-point higher scores during the postdrome (10.9 ± 4.4 vs. 6.9 ± 4.4 ; $p < 0.001$, large effect). During the ictal phase, word-finding difficulty showed the largest between-group difference (9.4 ± 4.4 vs. 3.3 ± 2.6 ; $p < 0.001$, large effect), and non-vertigo dizziness was associated with scores 3.9 points higher in patients endorsing it (8.5 ± 4.7 vs. 4.6 ± 3.9 ; $p < 0.001$, large effect). Postdromal skin hypersensitivity was associated with scores 3.7 points higher in patients endorsing it (11.7 ± 4.2 vs. 8.0 ± 4.7 ; $p < 0.001$, medium effect). The persistence of large-effect cognitive symptom associations into the postdrome has a specific and under-addressed clinical implication, that patients are routinely cleared to return to work, school, and normal activity based on pain resolution, and not cognitive resolution. Patients who report postdromal word-finding difficulty and impaired concentration are a measurably distinct, high-burden subgroup whose cognitive symptoms persists beyond the headache phase, and clinical guidance about return-to-activity should reflect that.

Among mood symptoms, one pattern was particularly notable. Prodromal euphoria was associated with total cognitive symptom scores 3.4 points higher than in patients without it (11.5 ± 4.6 vs. 8.1 ± 4.7 ; $p < 0.001$, medium effect), whereas postdromal euphoria was not significantly associated with cognitive burden (9.6 ± 5.3 vs. 8.3 ± 4.8 ; $p = 0.121$). Euphoria was the only mood symptom to be significantly associated with cognitive burden during the prodrome but not during the postdrome, distinct from depression, anxiety, and restlessness/irritability, which remained significant across both phases. Beyond its neurobiological interest, this finding has a practical dimension, that a patient who feels unusually well or euphoric immediately before migraine onset and then develops cognitive difficulty may be demonstrating a recognizable premonitory signature. Clinicians who counsel patients to identify and act on early prodromal features may find euphoria, particularly when accompanied by word-finding difficulty, a useful behavioral marker for early acute treatment initiation. One possible interpretation is that prodromal euphoria reflects a transient premonitory-phase neurobiological state, potentially involving hypothalamic or dopaminergic pathways, which resolves as the acute attack concludes, while negative affective and cognitive symptoms may persist into the recovery period as cortical excitability and neuromodulatory tone normalize more gradually, although this cannot be tested in the present data.^{37,38} Longitudinal studies with real-time symptom tracking are needed to characterize how mood and cognitive symptoms resolve within individual migraine attacks.

A consistent association between validated sensory hypersensitivity scores and total cognitive symptoms was observed across multiple analyses. Sound hypersensitivity showed the strongest association with total cognitive symptoms ($r = 0.45$), followed by skin hypersensitivity ($r = 0.39$) and light hypersensitivity ($r = 0.32$) in unadjusted correlations. All three sensory measures simultaneously in the multivariable model, adjusted for age, sex, monthly migraine frequency, migraine disease duration, depression symptoms, and anxiety symptoms, resulted in sound hypersensitivity ($\beta = 0.25$) and skin hypersensitivity ($\beta = 0.20$) both independently associated with greater cognitive impairment after accounting for migraine frequency, disease duration, and mood symptom burden.

Light hypersensitivity retained a smaller but statistically significant independent association ($\beta = 0.08$), although this finding warrants particular caution, as light hypersensitivity was endorsed by 97.1% of patients in the symptom-specific sample, leaving a comparison group of only 34 patients without it, which substantially limits the interpretability of its independent contribution in this model. The near-universal prevalence of photophobia limits its clinical utility for identifying patients at higher cognitive risk, whereas phonophobia and cutaneous allodynia, which are prevalent but not universal, showed stronger and more discriminating independent associations with cognitive burden. That is a specific and actionable clinical message, that when assessing cognitive risk in patients with migraine, asking specifically about sound sensitivity and cutaneous allodynia, not only photophobia, may better identify who is carrying a disproportionate cognitive burden. That phonophobia and cutaneous allodynia independently predicted cognitive impairment after full adjustment is consistent with the possibility that sensory hypersensitivity and cognitive dysfunction may reflect overlapping central sensitization, sensory gating, or thalamocortical network mechanisms.^{6,39}

Cutaneous allodynia in particular reflects trigeminal sensitization extending beyond the primary pain site and is associated with greater migraine chronification risk and broader neurological symptom burden.¹⁹ The graded associations between sensory symptom frequency and cognitive burden in ictal analyses, and the clustering of sensory symptoms in the prodrome and postdrome among patients with the highest cognitive symptoms, are consistent with the view that pronounced sensory sensitivity during migraine may in many cases reflect prodromal sensory sensitization accompanying the attack rather than serving as a causal precipitant.^{25,34}

Women with migraine reported significantly greater cognitive impairment than men across all domains, with small but consistent effect sizes, and male sex was independently associated with fewer total cognitive symptoms in adjusted analyses ($\beta = -0.09$). This is consistent with literature suggesting that women with migraine may experience greater symptom burden through overlapping hormonal, neurobiological, and psychosocial

pathways, although sex differences in migraine-related cognition remain incompletely characterized.^{25,33,40} Migraine disproportionately affects women, and cognitive complaints that are systematically greater in women with migraine risk being attributed to mood, fatigue, or other causes rather than recognized as part of the migraine phenotype itself.

Younger age was also independently associated with greater cognitive impairment after adjustment for migraine disease duration, a pattern that likely reflects the greater functional impact of cognitive symptoms during peak occupational and academic years, when attentional demands, processing speed, and multitasking requirements are highest, rather than progressive neurodegeneration.⁴¹ Evidence suggests that as patients with migraine age, attack phenotype may attenuate, including reductions in associated symptoms such as nausea and photophobia that contribute to perceived cognitive difficulty, which may partially account for the inverse age relationship observed here.^{25,42}

Taken together, these findings support a reconceptualization of ictal cognitive impairment as a measurable feature of migraine disease that is not adequately captured by migraine frequency classification alone. The highest cognitive burden clustered with mood symptom burden, sensory sensitization, language and cortical symptoms, and symptoms spanning the prodrome through postdrome, not with migraine frequency class after adjustment in this cohort.

For clinicians, several specific priorities emerge. Cognitive symptoms should be asked about during clinical encounters, not only when patients raise them independently, given their near-universal prevalence in specialty care populations. Cognitive complaints should prompt assessment of anxiety and depression, peri-attack mood symptoms, and sensory hypersensitivity, because these measures tracked cognitive burden more consistently than migraine frequency classification alone. Phonophobia and cutaneous allodynia specifically, more than photophobia, may help identify patients at highest cognitive risk. Patients whose word-finding difficulty and impaired concentration persist into the postdrome represent a high-burden phenotype for whom pain resolution is not an adequate proxy for cognitive recovery, and return-to-activity guidance should reflect this. Early identification of cognitive burden during the transition to chronic migraine may help guide preventive treatment decisions, given that the threshold-plateau pattern observed here suggests the earliest phase of chronification may be the most important window. Integration of a brief, validated instrument such as the Mig-SCog into routine migraine assessment may facilitate systematic identification and longitudinal monitoring of cognitive burden as part of comprehensive migraine care.

Several limitations of this study warrant consideration. The cross-sectional design precludes causal inference, and this is particularly relevant to two of the study's most clinically significant findings. First, the observed plateau in cognitive burden beyond 12 months of chronic migraine duration cannot be interpreted as evidence that cognitive symptoms stabilize over time within individual patients; it reflects a cross-sectional snapshot in which patients at different disease stages were compared at a single timepoint. Longitudinal studies with repeated cognitive assessments across the chronification trajectory are needed to confirm this pattern. Second, while anxiety and depression were the strongest independent predictors of cognitive impairment in adjusted models, the direction of this relationship cannot be established from these data. Psychiatric comorbidity may amplify the perception of cognitive dysfunction, cognitive impairment may exacerbate mood symptoms, or both may reflect shared neurobiological vulnerability, possibilities that cross-sectional regression cannot disentangle. Third, the study cohort was drawn from tertiary headache centers participating in ARMR, and the high proportion of participants with chronic migraine (880/1,231, 71.5%) likely reflects referral patterns rather than the broader migraine population; findings may not generalize to patients managed in primary care or community settings. Fourth, total cognitive symptom scores reflect subjective self-report of ictal cognitive complaints and were not validated against concurrent objective neuropsychological testing in this sample, leaving open the question of how closely subjective cognitive burden corresponds to objectively measurable deficits in this cohort. Finally, listwise deletion was used to handle missing data on a per-analysis basis, and given that missingness in migraine disease duration affected approximately 18% of the study cohort, analyses involving this variable should be interpreted with awareness of potential selection effects.

Declarations

Competing Interests

Meesha Trivedi has no disclosures. Richa Chirravuri has no disclosures. Todd J. Schwedt, within the prior 24 months, received compensation for consulting with Lundbeck and Salvia, and royalties from UpToDate. His employer received compensation for his consulting with AbbVie. He has stock options/equity in AllevaLUX and Nocira. His institution received research grants on his behalf from AbbVie, American Heart Association, Flinn Foundation, Henry Jackson Foundation, National Headache Foundation, National Institutes of Health, Patient Centered Outcomes Research Institute, Pfizer, and United States Department of Defense. Ryotaro Ishii has served as a consultant for Amgen K.K., Eli Lilly Japan K.K., Daiichi Sankyo Co., Ltd., Pfizer Japan Inc., and Otsuka Pharmaceutical Co., Ltd. He has received speaker honoraria from Amgen K.K., Daiichi Sankyo Co., Ltd., and Otsuka Pharmaceutical Co., Ltd. Gina Dumkrieger has no disclosures in the previous 24 months. David Dodick: Discloses the following (2025): Consulting: AbbVie, Vedana. Honoraria: Wolters Kluwer, Oxford University Press, Cambridge University Press. Stock options/shareholder/patents/board of directors: ExSano (options), Palion (options), Keimon Medical (Options), Man and Science, Healint (options), Theranica (options), Epien (options), Nocira (options), Matterhorn (shares), Ontologics (shares), King-Devick Technologies (options/board), EigenLyfe (Options/Board), NuvieBio (options/board), Cephalgia Group (options/board), Atria Health (options/employee). Patent 17189376.1-1466.vTitle: Onabotulinum Toxin Dosage Regimen for Chronic Migraine Prophylaxis (Non-royalty bearing). Christopher H. Gottschalk has received research funding from Lundbeck, Merz, Rehler and Relivion. He has served as a consultant and/or on Advisory Boards (honoraria) for Abbvie, Aspeya, Axsome Therapeutics, Guidepoint

Global, Impel, Lundbeck, Pfizer, Satsuma, Theranica, Tonix, Vectura Fertin. He is a board member of the Headache Cooperative of the Northeast (HCNE) and the Alliance for Headache Disorders Advocacy (AHDA). Stewart J. Tepper has received research funding from Aeon, Amgen, Annovis, Axsome, Bristol Myer Squibb, Cassava, Cognition, Eli Lilly, Genentec, Hoffman LaRoche, Inhibikase, Ipsen, Johnson and Johnson/Intracellular, Karuna, Lundbeck, Merz, Neurocrine, Novartis, Pfizer, PrecisionMed, Revance, Sanofi, Suven, UCB, and Upsher Smith. He has served as a consultant and/or on Advisory Boards for Abbvie, Aeon, Alphasights, Amgen, Amneal, Aruene/eNeura, Atheneum, Axsome Therapeutics, Bausch Health, Becker Pharmaceutical Consulting, Catch Therapeutics, ClearView Healthcare Partners, ClickTherapeutics, CoolTech, CRG, Decision Resources, Defined Health, DRG, DocDelta, Dr. Reddy's, Eli Lilly, ExpertConnect, FCB Health, Fenix, Gilmartin Capital, GLG, Guidepoint Global, Health Advances, Health Science Communications, HMP Communications, Impel, InteractiveForums, IQVIA, Keyquest, KLJ Associates, Krog and Partners, Lundbeck, M3 Global Research, Magellan Health, Magnolia Innovation, MJH Holdings, Neurofront Therapeutics, Nocira, P Value Communications, Pain Insights, Inc, Palion Medical, Paragon Therapeutics, Perfood, Pfizer, Putnam Associates, Rehaler, SAI MedPartners, Satsuma, Scilex, Slingshot Insights, Spherix Global Insights, Sprigings Intellectual Property Law, Strategy Inc, Synapse Medical Communication, System Analytic, Taylor and Francis, Tegus, Theranica, Third Bridge, Tonix, Trinity Partners, and Vial. He received salary from Thomas Jefferson University and Ki Health Partners. He has served on the speakers bureau for AbbVie, Amneal, Axsome, Dr. Reddy's, Eli Lilly, Impel, Lundbeck, Pfizer, Scilex, and Tonix. He received CME honoraria from the American Headache Society, Consortium for Research Education Social Awareness and Training In Neurosciences, Diamond Education Foundation, Forefront Collaborative, HMP Global, Medical Education Speakers Network, Medical Learning Institute Peerview, Miller Medical Education, National Association for Continuing Education, North American Center for CME, Physicians' Education Resource, PlatformQ Education, Primed, Vindico Medical Education, and WebMD/Medscape. He received travel assistance from AbbVie, American Headache Society, Amneal, Axsome, Diamond Educational Foundation, Dr. Reddy's, Eli Lilly, Impel, Lundbeck, Pfizer, and Sanofi. He received book royalties from Springer Health. Kathleen Digre is supported in part by an unrestricted grant to the Department of Ophthalmology and Visual Sciences from Research to Prevent Blindness, New York, New York. She has no conflict of interests. Catherine Chong receives research support from the National Institutes of Health, the American Heart Association and the Department of Defense. She serves as a Board Member of the Headache Cooperative of the Pacific.

Author Contribution

M.T., T.S., G.D, C.C. wrote the main manuscript text. M.T. prepared all figures. All authors contributed to the conception and design of the work. All authors reviewed and edited the manuscript and reviewed the final draft.

Acknowledgement

The authors gratefully acknowledge the American Registry for Migraine Research (ARMR) for the use of registry data to conduct this research. We would like to thank the patients and clinicians who participated in this registry. The data has been supplied by ARMR. The interpretation and reporting of these data are the responsibility of the authors and in no way should be seen as an official interpretation, endorsement by or position of ARMR.

Data Availability

The data used in this study were obtained from the American Registry for Migraine Research (ARMR) repository. The data are not publicly available, and all requests for access to the data or inquiries regarding data availability should be directed to ARMR in accordance with its established data access policies and procedures.

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Figures

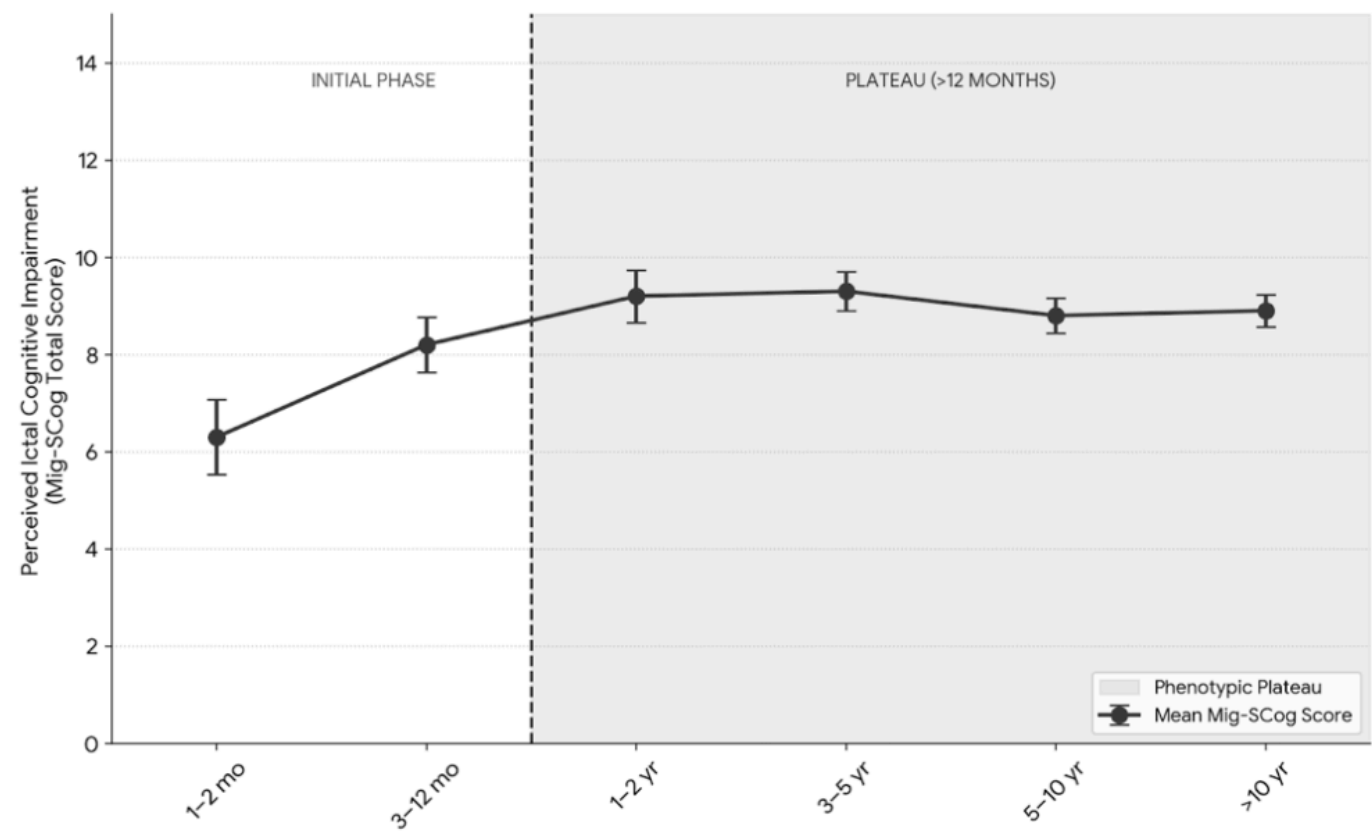


Figure 1

Cognitive impairment by duration of chronic migraine

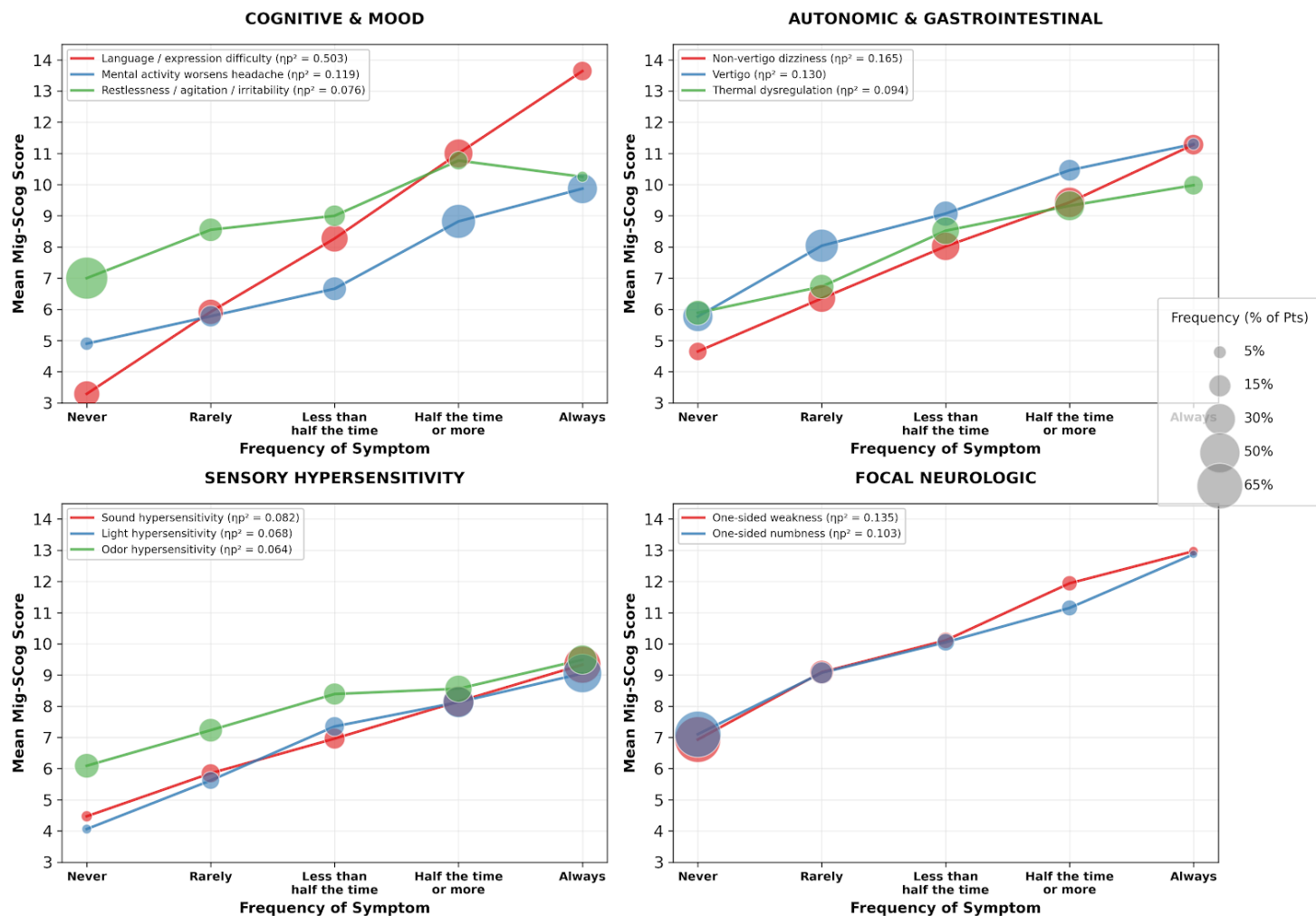


Figure 2

Association between ictal migraine-related symptom frequency and subjective cognitive impairment burden

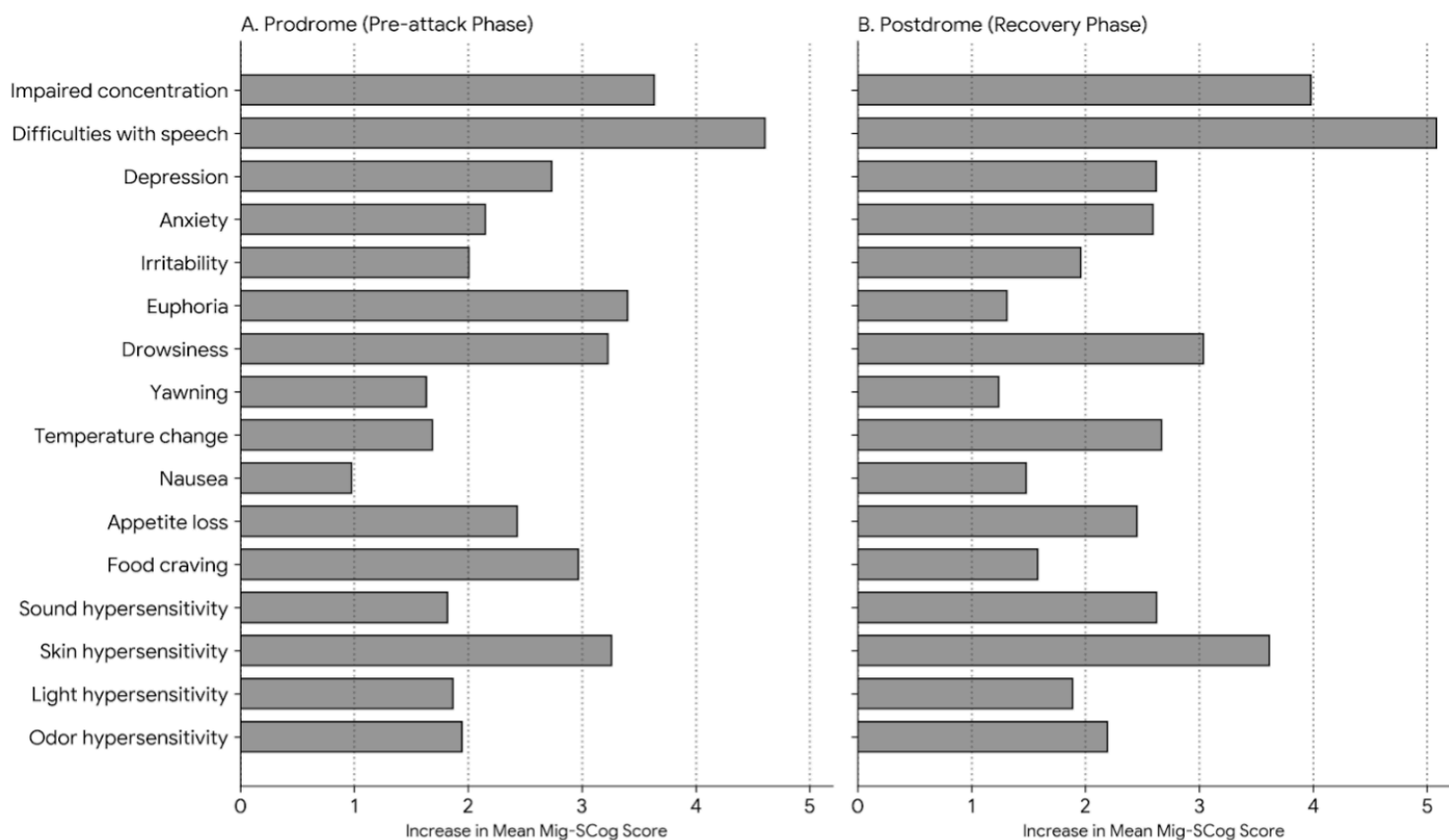


Figure 3

Increase in cognitive burden associated with symptoms across the prodrome and postdrome