Letter to the Editor

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Response to Comments on "Subjective Cognitive Decline Patterns in Patients with Migraine, with or without Depression, versus Non-Depressed Older Adults"

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Dear Editor,

We appreciate the thoughtful comments on our manuscript, "Subjective cognitive decline patterns in patients with migraine, with or without depression, versus non-depressed older adults."[1] We are grateful for the opportunity to address these methodological and interpretive concerns, which have helped improve the clarity, scientific rigor, and interpretive balance of our study.

First, we agree with the commentator's assessment.[2] The use of different objective cognitive assessment tools—the comprehensive Seoul Neuropsychological Screening Battery-II (SNSB-II) for the memory clinic cohort and the brief Korean Mini-Mental State Examination (K-MMSE) and Korean Montreal Cognitive Assessment (K-MoCA) for the headache clinic cohort—represents an important limitation. As the commentator noted, brief screening tools may not be sufficiently sensitive to detect subtle, domain-specific cognitive deficits in younger, highly educated individuals and may therefore introduce a ceiling effect. At the time of data collection, these screening tests were used as practical standard measures in our routine headache clinic practice to exclude overt cognitive impairment. We agree that this institutional difference in tool selection represents a limitation. While this was a practical constraint in our routine clinical practice at the time, we explicitly noted in our study that the classification of the migraine cohort might be less rigorously defined. This highlights the clear need for consistent neuropsychological batteries in future prospective trials.

Second, we appreciate the commentator's observation regarding the psychometric and conceptual differences between the Short-form Geriatric Depression Scale (SGDS) and the Patient Health Questionnaire-9 (PHQ-9). We agree that directly comparing absolute scores or applying a uniform "non-depressed" threshold across these distinct scales is methodologically limited. From a clinical and measurement perspective, however, the use of population-specific instruments was a deliberate approach intended to improve measurement accuracy within each cohort.[3] In older adult populations, standard depression scales may be confounded by age-related somatic complaints, which can inflate false-positive findings. The SGDS was designed to reduce this somatic confounding and to better isolate affective symptoms in memory clinic settings.[4] In contrast, for the younger migraine cohort, the PHQ-9 is a reliable, widely accepted, and validated instrument for assessing comorbid affective symptoms in headache clinical practice.[5] If we had assessed both cohorts using a single scale, we may have introduced substantial "somatic symptom overlap bias," either by overestimating

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depression scores in the older adult group through PHQ-9 somatic items or by reducing sensitivity to depressive symptoms in the younger migraine group through SGDS use. Thus, although this dual-scale approach introduces cross-group statistical heterogeneity, it was clinically justified to support the internal validity of depression assessment within each population.

Third, we agree with the commentator that the lack of detailed clinical variables, such as headache frequency, chronicity, and medication overuse (MO), is important for interpreting the absolute severity of subjective cognitive decline (SCD). As noted, MO is common in tertiary headache clinics and may contribute to cognitive complaints, potentially inflating total quantitative SCD Questionnaire (SCD-Q) scores. However, a primary objective of this study was to analyze qualitative differences in domain-specific cognitive complaints across distinct clinical populations. From a methodological perspective, we believe that strictly controlling for all migraine-specific clinical characteristics as covariates could introduce overadjustment bias.[6] In clinical practice, headache frequency, chronicity, and subsequent medication use are intrinsic components of a more severe migraine phenotype and may function as mediators or synergistic factors rather than as baseline confounders alone. Statistically removing the variance attributable to these disease characteristics could therefore create an artificial and clinically nonrepresentative migraine group, such as one effectively stripped of headache burden. This approach could obscure, rather than clarify, the phenomenological profile of SCD experienced by these patients in real-world clinical practice. Therefore, although the lack of adjustment for MO and headache frequency limits quantitative causal inference, our approach helped preserve the qualitative pattern analysis central to the study objective.

Fourth, we are grateful to the commentator for this guidance. Recent large-scale meta-analyses and diagnostic frameworks suggest that elevated depressive symptoms in individuals with SCD do not independently predict accelerated progression to mild cognitive impairment or dementia, whereas anxiety and SCD-related worry may have stronger prognostic relevance.[7,8] Accordingly, the elevated SCD-Q scores in our depressed migraine cohort are best interpreted as a reflection of affective vulnerability rather than a preclinical neurodegenerative signal.[9] As our study relied on a cross-sectional design, we agree with the commentator and the frameworks of Desai et al.[7] and Jessen et al.[8] that longitudinal prognostic inferences should not be drawn from these data.

In conclusion, we thank the commentator for these constructive criticisms, which helped us refine the interpretation of our data and present a more balanced and scientifically rigorous discussion.

AVAILABILITY OF DATA AND MATERIAL

Not applicable.

AUTHOR CONTRIBUTIONS

Conceptualization: SHL, SJC; Data curation: SHL, Investigation: SHL; Writing–original draft: SHL, Writing–review and editing: SHL, SJC. All authors have read and agreed to the published version of the manuscript.

CONFLICT OF INTEREST

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