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Unraveling the Risk Landscape of Mild Cognitive Impairment: A Pilot QEEG Study With *Z*-Score and Cordance Analysis

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Abstract

Introduction. Mild cognitive impairment (MCI) is the decline in cognitive function among individuals aged over 60, and the transitional phase between normal aging and dementia. The Mini-Mental State Examination and Montreal Cognitive Assessment (MoCA) may not detect early dementia, hence the importance of identifying MCI or early dementia through biomarkers, such as EEG. **Objectives**. Evaluating EEG quantification in raw values, EEG quantification in *z*-scores, and cordance measures as potential differential biomarkers to discriminate MCI. **Method**. The study involved 20 subjects: 10 healthy individuals and 10 with memory complaints. An EEG was obtained from each participant and raw scores, *z*-scores, cordance, and three-dimensional data were analyzed. **Results**. No differences were found in absolute power in raw scores, three-dimensional analysis and cordance variables. A significant difference was found between the groups regarding the Delta1 *z*-scores at the F7 location, where the memory complaints group exhibited a higher *z*-score. **Conclusions**. Normalized EEG quantification data, converted into *z*-scores, could serve as potential markers to distinguish between cognitively healthy individuals and those at risk of MCI. Using qEEG normative databases may reveal useful differences for identifying subjects at risk of MCI. Further research into intermediate states, between normal cognitive function and established MCI, is needed to clarify this aspect.

Keywords: qEEG; mild neurocognitive disorders; *z*-score; cordance; sLORETA

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Introduction

Mild neurocognitive disorder (NCD) due to unknown etiology, previously known as mild cognitive impairment (MCI), is the decline and deterioration in cognitive functions among people over 60 years (5th ed.; DSM-5-TR Neurocognitive Disorders Supplement; American Psychiatric Association [APA], 2022). It is also viewed as the preclinical and transitional stage between healthy aging and dementia. Currently, the overall worldwide prevalence of mild NCD is 15.56% in adults aged 50 years and older (Bai et al., 2022).

According to Zhuang et al. (2021), mild NCD is a clinical condition with a high risk of progression to dementia. Hence, the importance of early detection, particularly at the mild NCD stage as a critical strategy in disease management, could potentially affect long-term outcomes (Zhuang et al., 2021). In this sense, the advice is to have a clinical neuropsychological examination that facilitates early detection of dementia, pointing to a pathway for care planning and disease education (Weintraub, 2022).

The detection of mild NCD is a complex clinical task because of its different manifestations or subtypes (amnestic, multidomain, etc.). However, the evolutionary nature of the pathological aging process, especially in the preclinical stage, advises

the use of screening strategies to determine the early risk of developing dementia. Since the sentinel units of epidemiologic surveillance are the primary health care systems, the use of rapid and simple screening tools (brief cognitive tests) is required to objectively identify patients at risk (Jorm & Jacomb, 1989).

However, there is controversy regarding the most advisable instruments for screening in primary care, depending on the time, the cognitive processes evaluated, and the applicability according to the educational level (Carnero-Pardo et al., 2022), or the psychometric quality and discriminative sensitivity among the tests (Costa et al., 2022; Jannati et al., 2024).

For this reason, it is necessary to use combined strategies that evaluate the older adult (cognitive functionality) and contrast the evolution with a key informant, whether a family member or a caregiver. In addition, face-to-face or teleneuropsychology modalities should be used to reach the highest percentage of the population (Sánchez Cabaco et al., 2023).

On the other hand, in the prodromal or earliest stages of dementia, especially in those with high prior levels of cognitive achievement and education, the routine screening accomplished with such tests as the Mini-Mental State Examination (MMSE), the Montreal Cognitive Assessment (MoCA), or the cursory bedside clinical mental status examination may yield no abnormalities (Nasreddine et al., 2005). Hence, the importance of differencing mild NCD from early stages of dementia through biomarkers, being EEG techniques, is helpful in this matter. The sensitivity of EEG to detect brain disorder correlates has been enhanced through quantitative methods of analysis such as quantitative EEG (qEEG). Additionally, qEEG data can be logarithmically transformed to achieve Gaussianity and undergo age regression and transformation into *z*-scores relative to population reference norms (normative databases), thereby mitigating intersubject variations due to variables such as age (Deslandes et al., 2004).

Finally, given the limitations and controversies mentioned above, the need to use neurophysiological markers (EEG measurements) combined with brief neuropsychological tests should be emphasized to increase the safety of screening (avoidance of false positives and negatives). These markers are an effective complementary tool because of their simplicity, noninvasiveness, few limitations in the measurement environment, and ease of use. Recent studies in this direction have shown that they detect specific changes in elderly people with MCI (Katayama et al., 2023).

The objective of this study was to examine certain quantitative features derived from EEG recordings in both healthy individuals and those with subjective memory complaints. Specifically, we aimed to evaluate EEG quantification in terms of raw values, EEG quantification in terms of *z*-scores, and cordance measures as potential differential biomarkers that could discriminate between healthy individuals and those experiencing subjective memory complaints.

Methods

Participants

The focus of this study is on biomarkers, but participants were recruited from the MCI Screening Unit. So, it is important to note that *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5-TR; APA, 2013) diagnostic criteria for mild NCD due to unknown etiology are based on four criteria and two specifiers. The four criteria relate to cognitive changes, functional activities, and excluding delirium and competing mental disorders. The two specifiers are the presumed etiologies of mild NCD and the presence or absence of behavioral problems (Stokin et al., 2015).

The inclusion criteria for this study were as follows. Participants must be:

- registered in the city of Salamanca, Spain.
- at least 60 years of age at the time of selection.
- able to give informed consent.
- absent of medical conditions that could significantly affect participation in the study.
- willing to attend the scheduled appointments and complete the required questionnaires.
- fluent in Spanish to understand and complete the assessment tools used in the study.
- screened for MCI.

Participants were selected from the population already attending the Mild NCD Screening Unit of the Pontifical University of Salamanca. Twenty participants were invited to participate in this study (mean age 75.4 years) with the sociodemographic characteristics described below (Table 1). Although 20 participants were invited, not all of them were able to complete the EEG measurements due to various circumstances such as poor understanding

of the task during the test (producing nonreductible muscular and movement artifacts which created massive noise to the recordings), nonattendance at the appointment, or illness, among others, including 14 for data analysis.

Study participants were divided into two categories: (a) cognitively healthy and (b) with subjective memory complaints (SMC) or with mild NCD indicator according to MoCA scores (Table 2; Rosenzweig et al., 2023). The Yesavage scale was used to get a better context of the emotional status of the participants, and its implication on the qEEG measurements (Table 3; Greenberg, 2023).

This study protocol was reviewed and approved by the Institutional Review Board of Universidad Pontificia de Salamanca (ei-MEMO+AYsal 03/11/2023). Before the experiment, written informed consents were obtained from all the participants according to the Declaration of Helsinki. Neuropsychophysiology assessments were conducted in one of our neuropsychophysiology laboratories, properly equipped and isolated during the EEG recording.

Table 1
Demographics and Psychometric Data

Variable	HC	MCG	*P*
Gender (M:F)	4:5	1:4	.360
Age	74.56 (4.79)	77.80 (4.32)	.147
Education (years)	19 (5.61)	16.80 (3.63)	.518
GDS	1.11 (2.42)	0.20 (0.44)	.898
MoCA	27.22 (1.39)	16.60 (6.02)	**.040**

F = female; M = male; HC = healthy controls group; MCG = memory complaints group; GDS = Yesavage Geriatric Depression Scale; MoCA = Montreal cognitive assessment. Gender: chi-square test of independence. Age/Education/GDS/MoCA: Mann-Whitney test.

Table 2
MoCA Scores

Interpretation	Score range
Normal cognition	26–30 points
Mild NCD	18–25 points
Moderate NCD	10–17 points
Major NCD	Under 10 points

Table 3
Yesavage Depression Scale Scores

Interpretation	Score range
Normal condition	0–4 points
Mild depression	5–8 points
Moderate depression	9–11 points
Severe depression	12–15 points

EEG Acquisition and Preprocessing

An EEG was obtained from each participant. For the collection of the EEGs each patient was fitted with an electroencephalography cap, Electro-Cap, (Electro-cap International) with 19 channels located according to the 10–20 International System (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2) and using a linked-ear montage. For 5 min, EEG signals from all 19 channels were simultaneously obtained and collected using a Discovery24 amplifier from BrainMaster Technologies, Inc. Impedances of less than 5 kΩ were maintained, and a constant temperature and humidity of less than 25 °C and 50%, respectively, were maintained in the laboratory. EEG recordings were made in the closed-eye state with the use of BrainMaster Technologies, Inc. Brain Avatar 4.6.4 software, where artifacts were visually inspected and removed.

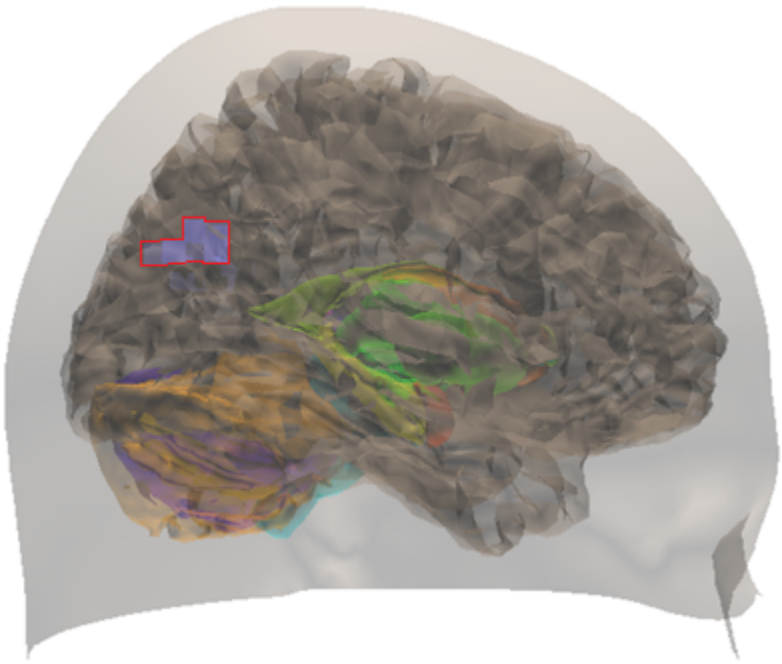
EEG Analysis and Quantification

EEG recordings were processed using a linked-ear montage and compared to the BrainDx normative database (John et al., 1977; John et al., 1987), from

which surface *z*-score values were obtained. Ten frequency bands were selected with the following ranges: Delta1 = 0.5–1.5 Hz, Delta = 1.7–3.7 Hz, Theta = 3.7–7.7 Hz, Alpha = 7.7–12.7 Hz, Beta = 12.7–25.2 Hz, Beta-2 = 25.2–35.2 Hz, Gamma = 35.2–50 Hz, Alpha1 = 7.7–10.2 Hz, Alpha2 = 10.2–12.7 Hz, and Sum = 1.7–25.2 Hz, as they appear in the Brain Avatar 4.6.4 BrainDx normative database implementation. Relative power was excluded because it was a calculation of the absolute power distribution of the entire spectrogram. This avoided redundant data (Stoller, 2011) and allowed each wave to be treated as a variable independent of the other variables.

In addition, *z*-scores for voxels, structures, Brodmann areas, and networks were calculated using BrainMaster standardized low-resolution electromagnetic tomography (sLORETA). This software allows the source of EEG activity within the cortex to be triangulated. Its bark model consists of 6,239 voxels, cubes of 5 mm^3. In each voxel, the current density source (CDS) is 1–45 Hz, whose estimates can be converted into a three-dimensional image (Gracefire, 2016). Each structure is formed by a set of voxels (see Figure 1) that are considered or designated as regions of interest (ROI) and displayed for monitoring through the screen (Collura, 2012, 2017).

Figure 1. *Brain Avatar sLORETA Projector*.



Note. The angular gyrus is displayed as an ROI (blue-colored and red-highlighted marked area).

Additionally, concordance was calculated using the method proposed by Leuchter et al. (1994). Cordance is known to be a metric highly correlated with metabolism and perfusion (Leuchter et al., 1994; Leuchter et al., 1999). This was important to this study, given the fact that different studies (Austin et al., 2011; Sperling et al., 2010) have revealed reductions in metabolism and perfusion, along with the accumulation of amyloid deposits, in regions such as parietal areas, lateral temporal areas, precuneus, posterior cingulate gyrus, and the dorsolateral frontal cortex in patients with cognitive decline.

Statistical Analyses

Mann-Whitney *U*-test and *t*-test were used to analyze continuous variables (age, absolute power, absolute power *z*-scores, etc.) computing the means, standard deviation, and chi-square (χ^2) for gender and cognitive abilities distribution between groups. Statistical significance was set at $\alpha = 0.05$ for all analyses. The effect size was calculated using Hedges' *g*.

Results

For an overview of the results of group comparisons for demographic data and screening psychometric scale data, refer to Table 1. In the healthy controls (HC) group, there were nine subjects (55.55% female) and five in the memory complaints group (MCG) group (80% female). No statistically significant differences in the distribution were found either by gender or condition (χ^2 = .837, p = .360). There were no differences between the groups for age (U = 33.50, p = .0147), years of education (U = 17.00, p = .518), or GDS scale (U = 21.00, p = .898). A statistically significant difference between the groups was found in the MoCA scores (U = 2.00, p = .004), in which the MCG group scored lower than the HC.

In terms of neurometric variables, no differences were found between the groups in any variable of absolute power in raw scores, cordance (Tables 4 and 5) or sLORETA. Regarding the *z*-scores, a single statistically significant difference was found between the groups (see Table 6), particularly in Delta1 activity at the F7 location, where the MCG group exhibited a higher *z*-score (U = 4.00, p = .048, ES = .573).

Discussion

This pilot study focused on exploring the potential of brain mapping, including analysis of cordance and transformations in *z*-scores of qEEG variables, in the detection of MCI. Our sample consisted of cognitively normal individuals and individuals with subjective memory complaints, suspected of cognitive impairment. No significant differences were found in the demographic variables, but differences were observed in the performance on the MoCA screening test, with subjects reporting memory complaints scoring lower. This finding was expected, as previous literature documented similar results (Hong & Lee, 2023). Subjective memory complaints in healthy individuals are a risk factor for the development of cognitive impairment, and individuals with cognitive complaints have been found to perform worse on memory tests (Han et al., 2021; Mills et al., 2020; Xiao et al., 2021).

Regarding the neurometric measures, we found no significant differences in the raw qEEG values between the groups. In this regard, it is worth mentioning that previous studies have found differences in raw qEEG between healthy subjects and those with Alzheimer's disease (Musaeus et al., 2018; Shim et al., 2022; Tomasello et al., 2023), or between healthy subjects and patients with established cognitive impairment (Musaeus et al., 2018; Tomasello et al., 2023). One possible explanation for this discrepancy in results between our study and previous research is that our subjects with memory complaints did not have a confirmed diagnosis of cognitive impairment but only a suspected diagnosis.

On the other hand, although there were no differences in age between the groups, it is known that there are developmental changes in qEEG scores, and therefore, age is a crucial factor in interpreting the results of such neuropsychophysiological tests (John et al., 1980; Ko et al., 2021; Matthis et al., 1980). For that, we decided to transform the raw qEEG scores into *z*-scores by comparing each subject to a normative database based on age and using these transformed scores as the dependent variable. The *z*-score minimizes the influence of variables that could affect the EEG in each subject, such as age.

When comparing between groups, we found that subjects in the MCG exhibited a higher *z*-score than controls in the left frontal region, specifically at the F7 location. This was the only noteworthy finding regarding the results of quantification and comparison with normative databases in our study. These results are consistent with previous studies that have found greater EEG slowing in individuals with impairment compared to healthy subjects. For example, Shim et al. (2022), investigated early detection of Alzheimer's disease in individuals with memory complaints and found an excess of delta activity in frontal, temporal, and parietal regions in subjects with subjective complaints and positive amyloid PET. Furthermore, they found that the greatest slowing of the EEG was in the left frontal regions. In another study focused on frontal event-related oscillations using oddball tasks as a reflection of neurodegeneration in the continuum between normality and mild NCD, Yener et al. (2016) found that frontal volume was lower in subjects with mild NCD compared to healthy individuals, and that there was a positive correlation between frontal volume and frontal event-related oscillations. They explain this correlation by assuming that an increase in frontal delta activity at rest could lead to lower frontal event-related oscillations during the performance of tasks like those used in their study.

Table 4
P-Values for Each Frequency Band and Channel of the Raw Scores

	Frontal Lobe							Central Area			Parietal Lobe			Temporal Lobe				Occipital Lobe	
	Fp1	Fp2	Fz	F3	F4	F7	F8	Cz	C3	C4	Pz	P3	P4	T3	T4	T5	T6	O1	O2
Delta1	.833	.833	.435	.622	.435	.524	.065	.524	.222	.127	.171	.724	.524	.354	.354	.435	.093	1.00	1.00
Delta	.622	.724	.943	.943	.833	.435	.435	.622	.284	.524	.354	.943	.724	.833	.724	.622	.127	.622	.724
Theta	.622	.833	.524	.622	1.00	.171	.833	.622	.833	.833	.724	1.00	.833	.833	.833	.724	.943	.724	.833
Alpha	1.00	1.00	.622	1.00	.833	.524	1.00	1.00	.943	1.00	.622	.833	.943	.622	.943	.622	.284	.524	.284
Beta	.622	.524	1.00	.943	.622	.354	.284	.833	.833	.833	.524	1.00	.833	.284	.222	.724	.127	.435	.524
Sum	1.00	1.00	.833	.943	.943	.222	.724	.943	.622	.943	.171	.622	.622	.833	.622	.524	.065	.435	.354
Beta2	.435	.354	.724	1.00	.524	.093	.065	.435	1.00	.724	.724	.724	.524	.435	.127	.622	1.00	.724	.833
Gamma	1.00	.284	.127	1.00	.943	.354	.354	.524	.622	.171	1.00	.524	.833	.524	.524	.943	1.00	.354	.833
Alpha1	1.00	1.00	1.00	.943	.833	.524	1.00	1.00	.943	.943	.435	.724	.724	.435	.833	.724	.354	.524	.222
Alpha2	.724	.524	.284	.724	.524	.354	.724	.354	1.00	.833	.724	.724	1.00	.622	.833	1.00	.354	.724	.435

Table 5
P-Values for Each Channel of the Cordance

	Frontal Lobe							Central Area			Parietal Lobe			Temporal Lobe				Occipital Lobe	
	Fp1	Fp2	Fz	F3	F4	F7	F8	Cz	C3	C4	Pz	P3	P4	T3	T4	T5	T6	O1	O2
Cordance	.524	.354	1.00	.724	.284	1.00	.524	.524	.943	1.00	1.00	.435	.171	.622	.943	1.00	.833	.524	.622

Table 6
P-Values for Each Frequency Band and Channel of the Z-Scores

	Frontal Lobe							Central Area			Parietal Lobe			Temporal Lobe				Occipital Lobe	
	Fp1	Fp2	Fz	F3	F4	F7	F8	Cz	C3	C4	Pz	P3	P4	T3	T4	T5	T6	O1	O2
Delta1	.808	.368	.570	.570	.808	.048	.214	.683	.368	.368	.570	.683	.683	.933	.933	1.00	.808	.933	.683
Delta	.570	1.00	.368	.933	.214	.461	.154	.570	.570	.808	.283	.214	.154	.933	.808	.933	.683	.808	.283
Theta	.368	.461	.683	.570	.933	.109	1.00	.214	.154	.154	.160	.073	.214	.461	.570	.109	1.00	.808	1.00
Alpha	.933	1.00	1.00	.570	.808	.933	.808	.808	.461	.808	1.00	.570	.461	.683	.808	.368	.461	.570	1.00
Beta	.283	.368	.808	.808	.808	.283	.368	.683	1.00	.808	.683	.683	.808	.933	.368	.808	.368	.368	.570
Sum	1.00	.808	.808	.683	.570	1.00	1.00	.808	.368	1.00	.283	.808	.683	.808	1.00	.283	1.00	.933	1.00
Beta2	.214	.400	.570	.808	.808	.933	.683	.368	1.00	.368	.109	.933	.808	.283	.214	1.00	.461	.073	.280
Gamma	.368	.933	.368	.808	.214	1.00	.683	.933	.283	.808	1.00	.570	1.00	1.00	.368	.570	1.00	.570	.214
Alpha1	.933	.933	.933	.933	.933	.808	.570	1.00	.683	.808	.808	.368	.461	.808	.683	.214	.461	.461	.933
Alpha2	.808	.808	1.00	.461	.570	1.00	1.00	1.00	.214	.808	.283	.808	.368	1.00	.570	.808	.368	.368	.808

In a study conducted by Li et al. (2017), P300 was collected during a working memory task in individuals with amnestic mild NCD and healthy subjects, and it was found that subjects with mild NCD exhibited larger P300 amplitudes in the left frontal region. They interpretated this, according to previous literature, as an explanation that those individuals with mild NCD recruit more neural resources than individuals without impairment in various cognitive tasks, including memory and attention, to attempt to compensate for their cognitive deficits; hence, larger amplitudes of this component were observed.

The concept of using cordance in this pilot study stems from the well-established fact that there are changes in perfusion in patients with cognitive impairment (Alexopoulos et al., 2012). Given that cordance is a measure highly correlated with perfusion (Leuchter et al., 1994; Leuchter et al., 1999), it would not be surprising to uncover relevant findings in this regard. To the best of our knowledge, there have been few or no studies to date that have focused on the use of cordance measurement for the identification of markers of cognitive decline. In this regard, our research is pioneering. However, we did not uncover any significant findings related to cordance. One reason for the lack of findings in this metric could lie in the intermediate state (between normal cognitive functioning and established mild NCD diagnosis) in which the mild NCD group was situated. Although some studies (Sierra-Marcos, 2017) have found alterations in perfusion in patients with mild NCD compared to healthy controls, others have shown that there are no differences between individuals with and without subjective memory complaints (Funaki et al., 2019). Further research in intermediate states, between normal cognitive functioning and established cognitive impairment, is likely necessary to clarify this aspect.

No differences were found between the groups in sLORETA. Despite a significant difference being found in F7, we did not find anything relevant in the three-dimensional analysis. The structures represented by the sLORETA projector are created based on voxels (units of 5 mm^3). One possible explanation for this is that only part of some structures may exhibit significant elevations, but the averaging effect of the activity recorded in those voxels could dilute the elevation. In other words, one or several nearby structures may show elevations in a group of voxels, but the remaining portion of them may not show such elevations and therefore fail to generate sufficient deviating data. In one of our studies (Pérez-Elvira & Jiménez Gómez, 2020), we found that some voxels within a structure could exhibit elevations expressed in *z*-scores, while the remaining voxels of the structure could fall within the norm.

In summary, our work has presented as main innovations the testing of normalized data, transformed into *z*-scores, and concordance metrics as potential markers that differentiate cognitively healthy individuals from those at risk of cognitive decline. According to our results, and contrary to comparing healthy subjects with patients with Alzheimer's or with established cognitive impairment, raw qEEG values would not be informative. However, qEEG data transformed into *z*-scores through normative databases could reveal differences that assist in discriminating subjects at risk of cognitive decline. In this regard, it appears that changes in the frontal region, specifically the left frontal area, is a relevant location for detecting the risk of cognitive decline.

It should be noted that this study had significant limitations, being the sample size with the primary one. On the other hand, voxel-by-voxel data calculations were also not conducted in the case of sLORETA, which could have provided more information. It would be interesting for future research to increase the sample size and record information on a voxel-by-voxel basis.

Author Disclosure

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