EEG Oscillatory Patterns In Dementia And High-Risk Groups: A Quantum-Neuroscience Interpretation Of Neurophysiological Alterations

Dr. F. Anto Nazarene[1*], Dr. Viji Devanand[2], Dr. T. Lovie Beneta[3], Dr. S. Sathishkumar[4]

Abstract

Background:
Dementia is defined as the gradual worsening of the cognitive functions that remain accompanied by the detectable alterations in the neurophysiological functions. Electroencephalography (EEG) has also become a sensitive, non-invasive instrument to identify cortical slowing whereby the Mini-Mental State Examination (MMSE) is a standardized tool to screen the cognitive status of a person. Determining the connection between changes in EEG oscillations and cognitive functioning can contribute to the early diagnosis of the development of the disease.

Methods:
It was a cross-sectional study that involved dementia patients, people in high-risk groups and control people with normal cognition. The spectral power of the alpha, beta, theta and delta bands and alpha/theta ratio were examined. Pearson correlation coefficients were done to check the relation between EEG parameters and MMSE scores.

Results:
The scores of MMSE were clearly differentiated between groups with the minimum score being recorded in the dementia patients, with intermediate scores in high-risk individuals, and the highest scores in the controls who showed no impaired cognitive score. EEG data indicated significant decreases in alpha and beta activity and significant increases in theta and delta power in dementia patients and with the high-risk individuals less severe abnormalities were observed.

Conclusion:
MMSE and EEG when used together offer a good method of distinguishing the cognitive states in relation to various levels of dementia. The alpha/theta ratio as an EEG-based biomarker demonstrates a great potential of early detection and monitoring of cognitive impairment.

Keywords: Dementia, mini-mental state examination, electroencephalography oscillations, alpha/theta ratio, cognitive decline.

DOI Number: **NeuroQuantology 2025; 23(09):01-06**

1. Introduction

One of the most challenging neurological disorders in the world, dementia impairs not just general intellectual performance but also memory, attention, overall executive functioning, all of which deteriorate over time. It has become very common due to the increased life expectancy, and its impact has put enormous clinical, social, and economic

***Corresponding author:** Dr. F. Anto Nazarene
Address: [1*]MBBS, MD (Physiology) Medical Officer / Assistant Surgeon, Government Hospital, Pattiveeranpatti, Dindigul, Directorate of Medical and Rural Health Services (DMRHS), Department of Health and Family Welfare, Tamil Nadu Email ID: drantonazarene@gmail.com
[2]MBBS, MD (Physiology), Professor of Physiology, Department of Physiology, Sree Balaji Medical College & Hospital, Chrompet, Chennai, Tamil Nadu Email ID: devanandviji@gmail.com
[3]MBBS, MD (Physiology), Associate Professor of Physiology, Department of Physiology, Stanley Medical College, Chennai, Tamil Nadu Email ID: loviemerwin@gmail.com
[4]MBBS, MD (Physiology), Tutor of Physiology, Department of Physiology, Government Tiruppur Medical College, Tamil Nadu Email ID: marksathish2k6physio@gmail.com
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest

strains on the healthcare systems across the world [1]. Dementia has an underlying pathophysiology characterized by extensive neuronal dysfunction, loss of synapses, neuroinflammation, and impairment of intrinsic brain networks all leading to the objectively impaired cognitive performance [2]. Electroencephalography (EEG) has become a sensitive and non-invasive neurophysiological tool, which can be used to detect the abnormalities related to cognitive decline. EEG records the oscillatory brain activity and gives a clue about the functional integrity of cortical connectivity. Dementia and mild cognitive impaired individuals have a typical activation of a reduced alpha and beta activity with elevation of slower theta and delta activities, which indicate a lack of neural coordination and impaired cortical communication [3]. These oscillatory signals remain also increasingly known to be biomarkers of network-level degradation that provide valuable diagnostic data in the early stages of the disease progression [4].

As methods of quantitative EEG and signal processing have developed, EEG spectral analysis has come to the fore in the research of dementia. Changes in oscillatory rhythms have been found to be highly correlated with cognitive impairments and thus EEG is an index of usefulness in measuring functional brain impairment [5]. Especially spectral slowing, characterized by preeminence of low-frequency activity, has been reliably reported in Alzheimer disease and other related dementias [6]. This deceleration shows the decreased synaptic functioning, disrupted neuronal firing, and diminished arousal throughout the entire cortex, which is equivalent to the decline in clinical neuropsychological tests [7]. The EEG biomarkers remain also tested in a variety of neurodegenerative diseases such as Lewy body dementia, HIV-associated neurocognitive disorders, and subcortical vascular dementia, which shows their wide applicability in diagnosis [8]. Such results highlight the importance of EEG as a convenient neurophysiological outcome that can be used to measure diffuse cortical impairment that goes hand in hand with attenuated cognitive functions [9]. EEG dysfunctions indicate impairment of resting-state network connectivity, increased changes in synchronicity between areas, and modifications in the temporal dynamics- all of which, remain initial signs of neurodegeneration [10]. Network analysis studies have also shown that dementia is linked to a decrease in functional integrity and disturbance of information in intrinsic brain networks [11].

The Mini-Mental State Examination (MMSE) is one of the most popular cognitive screening tools that is used in grading the level of cognitive impairment. It evaluates orientation, attention, memory, language and visuospatial abilities giving a global measure of cognitive functioning. Reductions in MMSE scores remain associated with clinical decline and can be linked to structural and functional alterations of the brain in dementia [12]. Given that both MMSE performance and EEG oscillatory activity remain indicators of underlying neurophysiological integrity, the analysis of the relationship between the two provides a promising method of studying the process of cognitive impairment progression [13]. Dementia is normally linked with significant losses in generation of alpha rhythm that is normally helpful in attention, memory encoding and incorporation of information. Meanwhile, elevation of theta and delta power is an indicator of more cortex deactivation and network inefficiency [14]. The marginal cases such as mild cognitive impairment or subjective cognitive decline tend to have milder so far significant deviations in these oscillatory patterns, and therefore, EEG may identify the early neurophysiological abnormalities before cognitive impairments develop to a clinically significant level [15]. Thus, it is possible to combine the results of MMSE with EEG data and define the range between the normal cognition and high-risk conditions and definite dementia.

The alpha/theta ratio especially has been found to be a robust measure of cortical slowness and deterioration of the network. Reduced alpha/theta ratio is always linked to greater cognitive dysfunction and worse results in memory and execution [16]. This measure has a high level of discriminating between healthy elderly individuals, patients with mild impairment, and those with dementia, and can be discussed as one of the most sensitive quantitative EEG biomarkers in the studies of dementia [17]. Based on these factors, a study of the parallel alterations in cognitive scores, and EEG oscillatory activity is of great importance in in what way dementia is progressed. Through a comparison of dementia patients, high risk subjects and healthy controls, a mapping of the gradient of neurophysiological changes that remain accompanied by the loss of cognitive function is possible. EEG abnormalities usually occur before clinical symptoms remain manifested and therefore they remain especially applicable in early detection and risk categorization [18]. Besides, the description of the correlation between MMSE performance and EEG oscillatory parameters can be used to determine the consistency with which a decline in cognitive ability is reflected in certain cortical slowing patterns [19]. Here, the current research is limited to cognitive performance assessment with MMSE, the EEG oscillatory qualities, and the relationship between these two complementary measures between dementia, high- risk, and normal groups. The study will examine an additional understanding of the neuronal dysfunction continuum and its measurable manifestations since both cognitive and

neurophysiological signatures of impairment remain being studied to gain a deeper understanding of the continuum using standardized methods. The selected methodology enables to better explain the neurophysiological nature of cognitive impairment, which is evidenced by the accumulating data indicating that the combination of EEG indices and cognitive functioning can be used to predict the underlying neural degeneration associated with dementia [20].

Objectives of the study

1. To study the neurophysiological changes with the help of EEG in patients with dementia and high-risk groups, and MMSE
2. To assess the cognitive functions in patients with dementia and high-risk groups
3. To correlate the EEG changes with the cognitive functions in patients with dementia and high-risk groups
4. To compare the neurophysiological changes and cognitive variations among dementia patients, high-risk groups and normal subjects

2. Materials and Methods

2.1 Study Design

The study was an analytical cross-sectional study designed to determine neurophysiological changes in dementia, high-risk and control groups. The design made possible the simultaneous measurement of EEG oscillatory patterns of any difference between the study populations. The individual participants were all observed in a single time frame and this enabled comparing of the cognitive status, electrophysiological rhythms and laboratory indicators in a systematic manner. The reason behind the choice of this method was to examine potential connections between cerebral waves and absent longitudinal variation. The design provided an effective model in the investigation that can be used in the deterioration of the mind.

2.2 Study Setting

The study was carried out in a clinical neurology environment where there remain EEG services. The participants used were chosen in an outpatient and an inpatient unit according to their physician referral or diagnosis test. The entire tests including cognitive testing, recording of EEG and collection of samples were within the same institution to facilitate procedural consistency. The environment facilitated the standardized data collection by the trained technicians and clinicians with calibrated equipment. EEG recording was done under environmental conditions that were controlled to reduce artefacts. To guarantee accuracy and consistency of tests, lab testing was carried out in certified diagnostic facilities.

2.3 Study Population

The total sample size was 50 participants: 16 dementia patients (Group A), 20 high-risk individuals (Group B), and 14 normal controls (Group C). The participants were recruited after qualification and grouped into three categories according to the clinical diagnosis. Group A was patients diagnosed with a dementia according to the existing clinical criteria. Group B had those that were categorized as high-risk according to subjective cognitive decline, or family history or initial symptoms without reaching the diagnosis criteria. Group C consisted of healthy controls that had no cognitive complaints or severe comorbid conditions in their medical histories. The procedures of the assessment were the same to all the participants to make them comparable. The idea of grouping was meant to embrace a continuum of cognitive vulnerability, which would allow the study of the serial neurophysiological and deviations of the stages of impairment of sampled population.

2.3.1 Group A: Dementia Patients

People diagnosed with dementia by the neurologists within the standardized clinical guidelines were included in Group A. These subjects exhibited cognitive dysfunction in various components and needed to be assisted in day-to-day activities. History, neurological examination and cognitive testing helped in the diagnosis. Patients were stable to be able to have EEG recording. The individuals who had acute infections, significant psychiatric disease, or a stroke were not included. The respondents of this category gave a clue to the advanced neurophysiological impairment related to known cognitive impairment. The significant comparators that were used in evaluating the deviations in the high-risk and control groups were their EEG profiles.

2.3.2 Group B: High-Risk Individuals

Group B was composed of the individuals who were known to be at high risk of dementia development. These subjects demonstrated onset of early cognitive symptoms or subjective memory impairment or high family predisposition but did not meet the diagnosis of dementia. They were examined and assessed as clinically stable, cooperative, and acute medical illness. Intermediate changes in EEG oscillations markers that might preclude the manifestation of evident cognitive impairment were explored due to the high-risk classification. This group was providing crucial information to comprehend the conditions of transitional neurophysiological impairment could serve as possible initial clues of dementia risk to the prevention and diagnosis of the disease.

2.3.3 Group C: Normal Controls

Group C consisted of cognitively intact persons who have no neurological disease, psychiatric conditions, or system diseases. These subjects denied having any memory complaints, and normal clinical examination, and were at age-appropriate MMSE. The control subjects were subjected to the same EEG testings as the other groups so that, methods were identical. The fact that they were included allowed the normative ranges of oscillatory rhythms to be established. Such a comparison at the baseline was crucial in determining any meaningful differences in the high-risk and dementia groups, which reinforced the explanations of the differences between the groups, and added to the diagnostic implications of the study.

2.4 Inclusion and Exclusion Criteria

The inclusion criterion was that the participants had to be adults and also be in a position to give consent and undergo all the procedures of the study. Dementia patients satisfied the diagnostic criteria, whereas high-risk persons exhibited the early cognitive symptoms without established diagnosis. The exclusion criterion used in all groups was the exclusion of major psychiatric illness, recent cerebrovascular damage, traumatic brain damage, active infections, acute liver or renal failure, and drugs that influence EEG rhythms. Those who had an uncooperative nature or could not undergo an EEG were not included. These conditions made sure that neurophysiological differences that were observed were more an indication of cognitive status than of confounding medical or neurological conditions.

2.5 Cognitive Evaluation Using MMSE

The MMSE was used to measure cognitive status, which is conducted by trained clinicians and under standardized conditions. The test assessed the orientation, attention, language, memory and the visuospatial abilities. Based on the scores, the participants were divided into dementia, high-risk, and control groups, which verified the clinical classification and justified the additional analyses. MMSE was also administered using validated procedures that guaranteed accuracy and comparison between people. EEG results were interpreted using results against cognitive performance. The MMSE was a valid measure of the, global cognition in the study and helped in establishing relationships on cognitive deficits and changes in biomarkers across study groups. MMSE scores were used as one of the primary outcome measures to fulfil the study objectives.

2.6 EEG Recording and Oscillatory Analysis

EEG was recorded with a standardized 10- 20 electrode arrangement in a controlled setting that is rather quiet. The interviewees were made to sit in a comfortable position and told to stay still with their eyes closed as they acquired. Established protocols were applied to filter, digitize and correct the signals artifacts. The power spectral analysis was to measure oscillatory activity on frequency bands. Emphasis was laid on the description of alpha, beta, theta, and delta rhythms, and alpha/ theta ratios. All the analyses were analyzed by use of validated software. The method provided objective evidence of the cortical activity through which comparison was made between the neurophysiological patterns of the dementia, the high-risk, and the control groups.

2.6.1 Alpha and Beta Frequency Bands

The activity of alpha and beta bands was assessed to identify the variation of fast cortical oscillations of attention, memory and executive processing. Spectral power values were derived after the correction of artifacts and mean band amplitudes of individual subjects were calculated. Intergroup comparison was applied in order to prove that this frequency rates remain frequently associated with cognitive deficiency. Validated techniques were used to make it reliable by having followed the analytical procedures. These bands were then results whose analysis would lead to the conclusion of in what way much cortical slowing and network desynchronization representative of dementia would occur which would be an element of an overall understanding of the neurophysiological changes of the study population.

2.6.2 Theta and Delta Frequency Bands

Theta and delta bands were examined to give the slow-wave abnormalities, which remain typical of dementia. These frequency to spectral power analyses gains were pointers of the impaired cortical functioning and deficit in cognitive integration. The measurements were acquired using standardized methods of spectral decomposition. Group comparisons were made to determine the scale of slow wave upsurge of the high risk and dementia participants in comparison with the controls. These parameters prejudiced the critical examination of the progressive neurophysiological loss of sight. The slow oscillatory activity analysis provided the required data regarding the malfunction of the neural network and evidenced in the identification of EEG predictors regarding the beginning and final stages of the decline of the mental process.

2.6.3 Alpha/Theta Ratio Assessment

Low velocity swaying that is a delicate indicator of mind weakness. Mean spectral power values were also received and compared within groups in the form of ratios. A low ratio indicated a greater slowing of the cortex and it was expected to be more significant in dementia patients. This measure was an integrative measure of rhythmic activity and secondary measure of dysfunctional network

activity. Alpha/theta ratio has also been utilized to assist in differentiating between high-risk groups and the dementia groups and this analysis has enhanced the diagnostic applicability of EEG results to the study.

2.7 Correlation Analysis

In order to investigate in what way cognitive status is connected to neurophysiological activity correlation analysis was conducted between the major EEG parameter scores concerning alpha power, theta power and the alpha/theta ratio and MMSE scores. The strength of these associations and the direction were determined by Pearson correlation coefficients. This strategy enabled the analysis of whether a decrease in the cognitive performance was followed by stereotypical changes in EEG, especially, the elevation of the slow-wave activity and the decline of the alpha/theta ratio. The discussion was able to give an insight into the relationship between the progressive cortical slowing and deteriorating cognitive processes in both dementia and high-risk individuals, validating the EEG as an effective measure of cognitive impairment.

2.8 Statistical Analysis

Statistical software was used to compare EEG variables between groups of data. Descriptive statistics were used to summarize the characteristics of the participants whereas inferential tests, such as ANOVA and correlation tests, were used to determine the group differences and correlations. Conventional significance levels were used and normality and homogeneity assumptions tested. The findings had corresponding confidence intervals and p -values. This analytical approach allowed making sound interpretations of multimodal data, as well as it was possible to identify correlations between neurophysiological indicators. Validated statistical procedures were used and this made the study reliable and scientifically rigorous.

2.9 Ethical Considerations

Before commencing the studies, ethical approval was gotten by the institutional review board. Informed consent was given by all the participants following clear explanations of the procedures, risks, and confidentiality measures. Anonymity was maintained so that privacy was not violated. EEG records and cognitive measures were taken in observance of the ethics of conducting human research. The participants were not punished to discontinue participation at any point. The entire processes were made without compromising on the national and international ethical considerations in order to guarantee that the participants and scientific integrity were taken care of in the study.

3. Results

3.1 Demographic Characteristics

The study included a total of 50 participants consisting of 16 individuals diagnosed with dementia, 20 participants classified as high-risk for cognitive decline, and 14 cognitively normal controls. The overall sample represented older adults typically affected by neurodegenerative conditions, and all three groups were comparable in age distribution to ensure uniformity across cognitive assessments. Although gender distribution was not used as a primary variable, participants from both sexes were included across all groups. Clinical characteristics relevant to cognitive vulnerability-such as mild cognitive impairment, cardiovascular conditions, cerebrovascular events, metabolic disorders, and seizure history-were predominantly observed within the high-risk group, whereas dementia patients exhibited established cognitive and functional impairment. Controls had no significant neurological, psychiatric, or systemic illness. This demographic profile supports accurate comparison of cognitive and neurophysiological measures across the three groups.

3.2 Distribution of High-Risk Individuals

The high-risk group was composed of 20 persons with varied conditions which remain known to predispose to cognitive decline. The highest proportion was that of mild cognitive impairment, which had 10 participants (50%), which means that there was a significant subgroup of individuals who had early cognitive symptoms as shown in Table 1. 3 cases were found to have cerebrovascular accident (15%), and 2 cases were found to have ischemic heart disease and seizure disorders (10% each). A minor percentage (1 participant, 5 %) had a family history of dementia, uncontrolled diabetes mellitus, and hypercholesterolemia. This distribution has shown a non-homogeneous risk profile which represents various pathways that may lead to cognitive vulnerability.

Table 1. Distribution of High-Risk Group (Group B)

High-Risk Condition	Number of Subjects	Percentage (%)
Mild cognitive impairment (MCI)	10	50%
Cerebrovascular accident (CVA)	3	15%
Family history of dementia	1	5%
Ischemic heart disease (IHD)	2	10%
Seizure disorders	2	10%

Uncontrolled diabetes mellitus	1	5%
Hypercholesterolemia	1	5%
Total	20	100%

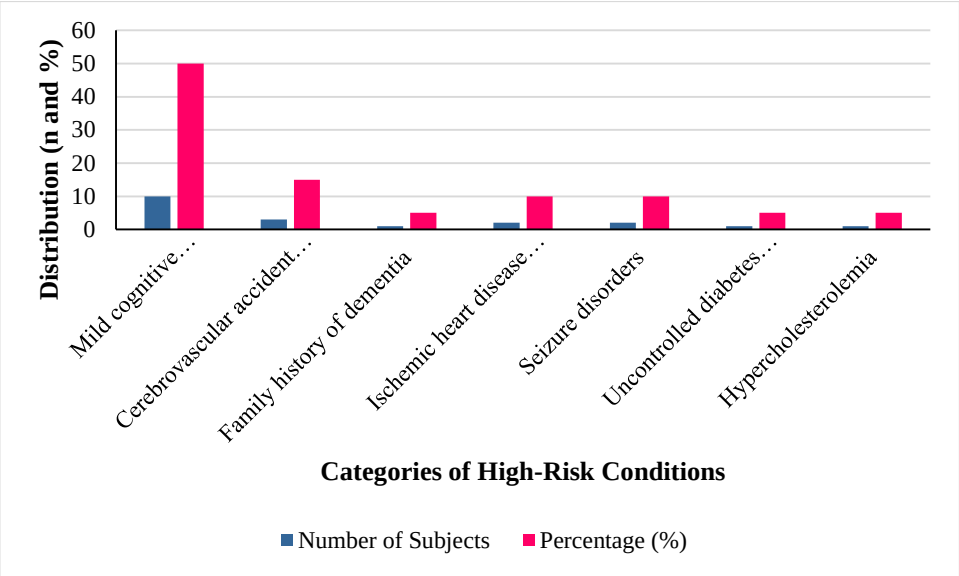

Figure 1. Distribution of High-Risk Conditions Among Study Participants

The spread of the high-risk disorders diagnosed among the people, who were characterized as having a high probability of cognitive drop, is visually represented in Figure 1. Figure 1 presents the distribution of high-risk conditions among the study participants. The bar chart will display the number and proportion of the participants in each category of the risk, and this will enable the bar chart to be visually compared across various contributing factors. The largest subgroup was mild cognitive impairment and then came cerebrovascular, metabolic, cardiovascular, and neurological conditions. Both the absolute and relative frequencies portray the heterogeneous character of risk contributors among this population. This type of distribution reflects the etiological complexity of cognitive vulnerability and is the argument of stratified assessment in the implementation of clinical research.

3.3 Cognitive Performance Across Groups (MMSE Scores)

The MMSE was used to assess cognitive performance and to show clear differences between the three study groups (n = 50). Table 2 indicates that there was a significant deficit in several areas of cognition and hence, the cases of dementia in Group A were impaired with a mean score of 14.13 ± 1.36. The intermediate mean MMSE score of 22.3 ± 1.92 found in Group B which consisted of high-risk people revealed mild-moderate cognitive impairment. On the contrary, normal subjects in Group C were found to have intact cognitive functions and the mean outturn was 26.29 ± 1.14. These results prove the progressive cognitive gradient between groups and confirm the clinical stratification of the study.

Table 2. MMSE Scores of the Study Population

Study Population (n) = 50		**Mean ± SD**	
	Group A (Dementia cases)	**Group B (High risk groups)**	**Group C (Normal subjects)**
MMSE Score (Out of 30)	14.13 ± 1.36	22.3 ± 1.92	26.29 ± 1.14

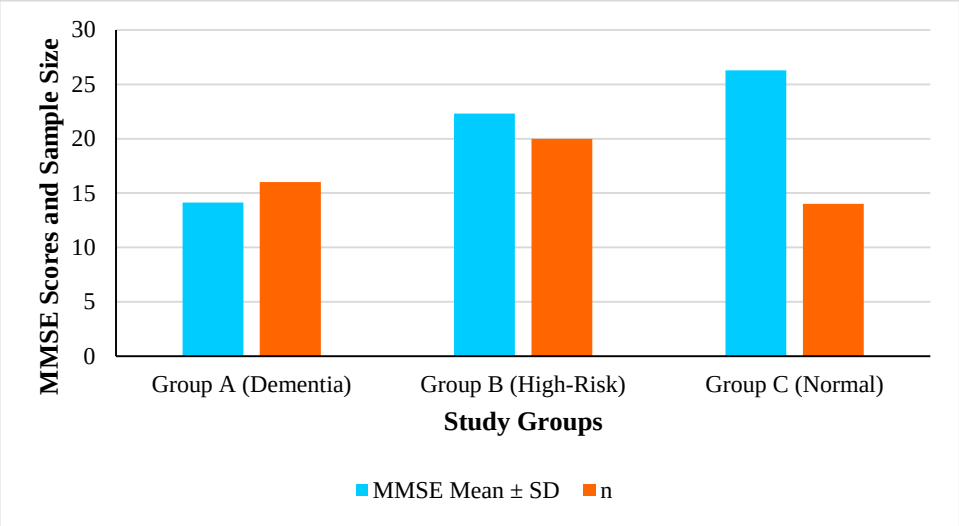

Figure 2. Comparison of Cognitive Performance Across Study Groups Using MMSE Scores

The cognitive performance of the three study groups as given by MMSE. The bar chart is used to compare the mean and the resultant sample sizes of cognitive scores to reveal the expected gradient of cognitive functioning as demonstrated by Figure 2. The cases of dementia have the lowest performance, which is considerably impaired, and high-risk individuals have intermediate scores in accordance with the initial or developing deficit. In normal subjects, cognitive functioning is intact and they perform better on MMSE. The overall display of the scores and the group size gives a visual comparison of the cognitive status which proves the validity of the use of groups stratification and validates the interpretation of the additional neurophysiological results.

3.4 EEG Oscillatory Characteristics

EEG showed significant changes in cortex rhythms in dementia group. There was reduced alpha (15/16 (93.75%)) and beta (11/16 (68.75%)) and increased theta (15/16 (93.75%)) and delta (10/16 (62.5)) as represented in Table 3. Mean frequency and the a/th ratio were also reduced in all the cases of dementia, and the mean was 0.6 ± 0.19. The intermediate abnormalities were shown in high-risk people: reduction of alpha in 13/20 (65%), increase of theta in 11/20 (55%), and a/th ratio of 0.85 ± 0.18. Neurophysiology was preserved with controls of a/th ratio of 1.31 ± 0.05.

The EEG characteristics of the study population are shown in Table 3 below.

Table 3. EEG Characteristics of the Study Population

Features	Group A (Dementia cases)	Group B (High-risk group)	Group C (Normal subjects)
Decreased Alpha (α)	15/16 (93.75%)	13/20 (65%)	1/14 (7.14%)
Decreased Beta (β)	11/16 (68.75%)	7/20 (35%)	Normal
Increased Theta (θ)	15/16 (93.75%)	11/20 (55%)	Normal
Increased Delta (δ)	10/16 (62.5%)	6/20 (30%)	Normal
Decreased Mean frequency	16/16 (100%)	14/20 (70%)	Normal
Decreased α/θ ratio (<1) (O1 electrode)	16/16 (100%)	16/20 (80%)	Normal
Mean ± SD (α/θ ratio) (O1 electrode)	0.6 ± 0.19	0.85 ± 0.18	1.31 ± 0.05

3.4 Correlation Between MMSE and EEG Parameters

The correlation analysis revealed significant relationships among age, MMSE scores, α/θ EEG ratio, and serum ANA levels. Age showed a strong negative correlation with MMSE ($r = -0.82$; $p < 0.0001$**) and a moderate negative correlation with the α/θ ratio ($r = -0.66$; $p < 0.0001$**), indicating progressive cognitive and electrophysiological decline with ageing. MMSE demonstrated a strong positive association with the α/θ ratio ($r = +0.85$; $p < 0.0001$**), confirming its reliability as a cognitive biomarker. Serum ANA levels displayed a mild positive correlation with age ($r = +0.32$; $p = 0.0117$*) and moderate negative correlations with both MMSE ($r = -0.41$; $p = 0.0016$*) and α/θ ratio ($r = -0.38$; $p = 0.0032$*), suggesting an autoimmune influence on neurophysiological impairment.

Table 4. Correlation (Pearson's correlation coefficient R values) among age, MMSE score and α/θ ratio (EEG) of the study population

	Age	MMSE	α/θ ratio (EEG)
Age	-	- 0.82($p < 0.0001$**)	- 0.66 ($p < 0.0001$**)
MMSE	-	-	+ 0.85($p < 0.0001$**)

Note: *Statistically significant ($p < 0.05$), **Statistically very significant ($p < 0.0001$)

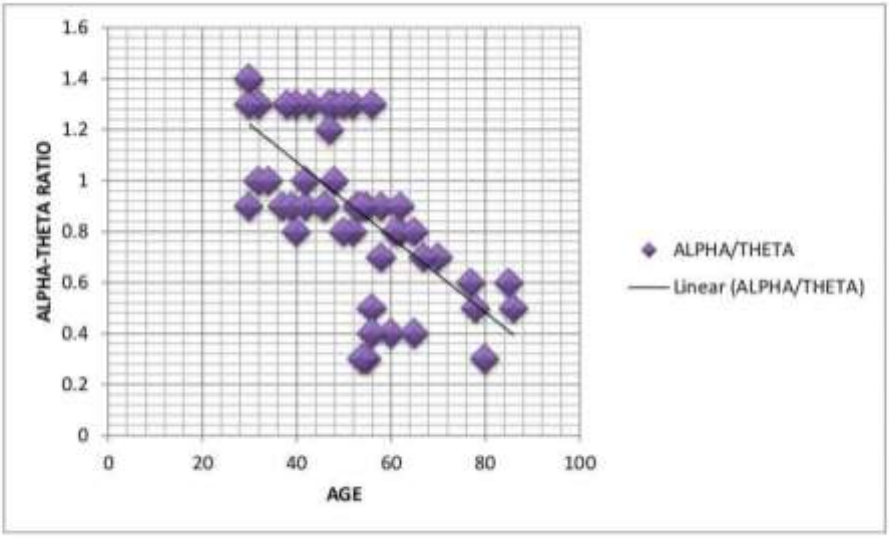

Figure 3: Correlation between Age and α/θ ratio (EEG)

Figure 3 illustrates the correlation between age and alpha/theta ratio in EEG.

4. Discussion

The study compared the cognitive and neurophysiological features of the dementia, high and normal groups by the performance of MMSE and EEG oscillatory activity. The explanation of high-risk condition distribution Table 1 and Figure 1 showed a high level of heterogeneity, which reveals the wide range of etiological routes that precondition people to cognitive vulnerability. This heterogeneity justifies the presence of neurophysiological markers that have the capacity to screen early dysfunction in diverse risk groups. The outcomes of MMSE showed the presence of the evident progressive gradient Table 2 and Figure 2, the worst impairment was presented by dementia patients, and the most high-risk individuals demonstrated the intermediate impairment. The results support the clinical stratification adopted in the study and establish the validity of MMSE as a cognitive discriminant in neurodegenerative diseases. EEG results provided evident cortical slowing in patients with dementia; the slowing of alpha and beta and an increase in theta and delta activity Table 3. These spectral frequency abnormalities remain well known indicators of disturbed cortical regularities and defective brain neuron interaction. The high-risk group showed midlevel abnormal oscillatory patterns indicating that neurophysiological disturbances were experienced even when severe cognitive impairment has not reached severe levels. A decrease in alpha activity, an increase in theta

power and a decrease in alpha/theta ratios of high-risk individuals is a sign of developing dysconnectivity, which can be a precursory event to cognitive deterioration. The correlation analysis in Table 4 revealed significant relationships among age, MMSE scores and α/θ EEG ratio. The higher were the MMSE scores, the more significant were alpha power and alpha/theta ratio, and the lower were the theta dominance. These results validate the fact that cognitive deterioration is similar to progressive cortical slowing and functional deterioration in neural efficiency. Alpha/theta ratio proved to be the most powerful predictor, as per its already established potential to help identify normal aging, mild impairment, and dementia.

The EEG abnormalities observed in the current study can be compared with the previous neurophysiological studies that revealed that dementia is correlated with extensive network dysconnectivity and spectral slowing. Previous evidence indicates that the neurodegeneration perturbs the white matter microstructure and spontaneous activity despite being a disease even at an early stage of progression [21]. These early disturbances remain reflected in the intermediate EEG abnormalities in the high-risk people, and this indicates that the cortical slowing starts before the clinical symptoms escalate. The gradual transition of the fast- to slow-frequency rhythms in dementia is congruent with the evidence of the pathophysiological validity of neural-surface antibodies in changing neuronal signaling and functional connectivity during neurodegenerative disorders [22)]. Even though the present research does not investigate immunological biomarkers, the mental and EEG activities remain the outcomes of the functional implications of cortical vulnerability as described by such literature. Additionally, the previous quantitative EEG research has highlighted the fact that the oscillatory connectivity and spectral measures and especially theta elevation and alpha suppression remain valid diagnostic indicators of Alzheimer disease as well as dementias [23]. This is supported by the current results, especially the high level of discriminative ability of the alpha/theta ratio which has been reported several times as a high-accuracy biomarker in quantitative EEG studies [24]. Also, spectral slowing measures have remained important in studies that remain developing robust EEG-based predictive models of the Alzheimer disease [25]. The existing findings add to this amount of evidence by showing that such spectral deviations remain common among cognitively impaired individuals and confirm the tests of EEG markers as sensitive to cognitive deterioration.

The results highlight the clinical viability of co-combining EEG indicators with the MMSE functioning to detect the initial cognitive alterations in persons who remain at risk of developing dementia. The cortical slowing generated can be detected using EEG, which means it is sensitive to minor neurophysiological deficits in the brain before the cognitive symptoms take over. EEG as a tool is inexpensive and available to many people, which can be improved to practice early screening, clinical decision-making, and early identification of high-risk individuals that might need early intervention or constant monitoring. The longitudinal research designs ought to be embraced in future studies to establish whether the changes in EEG spectrals foretell the subsequent deterioration in high-risk individuals. The sample size could be increased and more quantitative EEG measures could be added (coherence, connectivity, and microstate analysis) to enhance the accuracy of diagnostic procedures. EEG would be helped by digital cognitive testing or machine learning models to improve predictive ability. The multi-site and cross-cultural research will also enhance the applicability of EEG markers in the early detection of dementia.

5. Conclusion

The combination of cognitive testing with the Mini-Mental State Examination (MMSE) and electrophysiological testing with electroencephalography (EEG) can present a sound system of cognitive decline in dementia. The steady trend of low MMSE scores of normal people to high-risk groups and eventually to dementia patients show the existence of a specific pattern of cognitive decline. In tandem with this, results of EEG revealed a significant change of fast-frequency activity to the slow-wave activity, where dementia patients demonstrated significant decreases in alpha and beta activity and dramatic increases on theta and delta power activity. The large correlations obtained between the MMSE scores and EEG parameters, especially the alpha/theta ratio, highlight the high level of correlation between cortical slowing and cognitive dysfunction. Notably, the alpha/theta ratio turned out to be a consistent group-centered discriminator, which supports its usefulness as a possible clinical biomarker to detect and stratify the risks at an earlier stage. Such results demonstrate the clinical significance of the performance of MMSE in association with EEG-based measurements to enhance the detection of people at risk developing a progressive decline in cognitive functions. EEG has significant potential to aid in screening and monitoring cognitive health and in particular due to its low cost, non-invasive nature and wide accessibility in settings where high-tech neuroimaging is not feasible. Longitudinal research should be conducted in the future to determine if these electrophysiological markers can be used to predict and provide timely intervention.

References

1. WHO. Dementia Fact Sheet. 2019.
2. Cunningham EL, McGuinness B, Herron B, Passmore AP. Dementia. Ulster Med J. 2015.
3. World Alzheimer Report. Alzheimer's Disease International. 2015.
4. Ferri CP, Prince M, Brayne C, Brodaty H, Fratiglioni L, Ganguli M, Hall K, Hasegawa K, Hendrie H, Huang Y, Jorm A, Mathers C, Menezes PR, Rimmer E, Scazufca M; Alzheimer's Disease International. Global prevalence of dementia: a Delphi consensus study. Lancet. 2005 Dec 17;366(9503):2112-7. doi: 10.1016/S0140-6736(05)67889-0. PMID: 16360788; PMCID: PMC2850264.
5. Niedermeyer E, da Silva FL, editors. Electroencephalography: basic principles, clinical applications, and related fields. Lippincott Williams & Wilkins; 2005.
6. AM, Husain & Benbadis, Selim & Kaplan, Peter. (2007). Handbook of EEG interpretation.
7. Vespa PM, Nenov V, Nuwer MR. Continuous EEG monitoring in the intensive care unit: early findings and clinical efficacy. Journal of Clinical Neurophysiology. 1999 Jan 1;16(1):1-3.
8. Burns A, Robert P. The national dementia strategy in England. Bmj. 2009 Mar 10;338.
9. Iadecola C. The pathobiology of vascular dementia. Neuron. 2013 Nov 20;80(4):844-66.
10. Kuźma E, Lourida I, Moore SF, Levine DA, Ukoumunne OC, Llewellyn DJ. Stroke and dementia risk: A systematic review and meta-analysis. Alzheimer's & Dementia. 2018 Nov 1;14(11):1416-26.
11. Finger EC. Frontotemporal dementias. Continuum: Lifelong Learning in Neurology. 2016 Apr 1;22(2):464-89.
12. Snowden JS, Neary D, Mann DM. Frontotemporal dementia. Br J Psychiatry. 2002.
13. Neary D, Snowden J, Mann D. Frontotemporal dementia. The Lancet Neurology. 2005 Nov 1;4(11):771-80.
14. Pangman VC, Sloan J, Guse L. An examination of psychometric properties of the mini-mental state examination and the standardized mini-mental state examination: implications for clinical practice. Appl Nurs Res. 2000 Nov;13(4):209-13. doi: 10.1053/apnr.2000.9231. PMID: 11078787.
15. Tombaugh TN, McIntyre NJ. The mini-mental state examination: a comprehensive review. Journal of the American Geriatrics Society. 1992 Sep;40(9):922-35.
16. Soininen H, Partanen JV, Puranen M, Riekkinen PJ. EEG and computed tomography in the investigation of patients with senile dementia. Journal of Neurology, Neurosurgery & Psychiatry. 1982 Aug 1;45(8):711-4.
17. Moretti DV, Paternicò D, Binetti G, Zanetti O, Frisoni GB. EEG markers are associated to gray matter changes in thalamus and basal ganglia in subjects with mild cognitive impairment. Neuroimage. 2012 Mar 1;60(1):489-96.
18. Moretti DV, Frisoni GB, Fracassi C, Pievani M, Geroldi C, Binetti G, Rossini PM, Zanetti O. MCI patients' EEGs show group differences between those who progress and those who do not progress to AD. Neurobiology of aging. 2011 Apr 1;32(4):563-71.
19. Jeong J. EEG dynamics in patients with Alzheimer's disease. Clin Neurophysiol. 2004 Jul;115(7):1490-505. doi: 10.1016/j.clinph.2004.01.001. PMID: 15203050.
20. De Waal H, Stam CJ, Blankenstein MA, Pijnenburg YA, Scheltens P, van der Flier WM. EEG abnormalities in early and late onset Alzheimer's disease: understanding heterogeneity. Journal of Neurology, Neurosurgery & Psychiatry. 2011 Jan 1;82(1):67-71.
21. Gouw AA, Stam CJ. Electroencephalography in the differential diagnosis of dementia. Epileptologie. 2016;33:173-82.
22. Cibils D. Dementia and qEEG (Alzheimer's disease). InSupplements to Clinical Neurophysiology 2002 Jan 1 (Vol. 54, pp. 289-294). Elsevier.
23. Lizio R, Vecchio F, Frisoni GB, Ferri R, Rodriguez G, Babiloni C. Electroencephalographic rhythms in Alzheimer's disease. Int J Alzheimers Dis. 2011;2011:927573. doi: 10.4061/2011/927573. Epub 2011 May 12. PMID: 21629714; PMCID: PMC3100729.
24. Fahimi G, Tabatabaei SM, Fahimi E, Rajebi H. Index of theta/alpha ratio of the quantitative electroencephalogram in Alzheimer's disease: a case-control study. Acta Medica Iranica. 2017 Oct 7:502-6.
25. Lopez OL, Rabin BS, Huff FJ, Rezek D, Reinmuth OM. Serum autoantibodies in patients with Alzheimer's disease and vascular dementia and in nondemented control subjects. Stroke. 1992 Aug;23(8):1078-83. doi: 10.1161/01.str.23.8.1078. PMID: 1636181.