Original Article

# Correlation of the Severity of Dementia with EEG Findings

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## Abstract

**Background:** Dementia is a progressive neurodegenerative disorder marked by a global decline in cognitive, behavioral, and functional abilities. Alzheimer's disease and vascular dementia are the most prevalent forms, collectively accounting for nearly 90% of cases in the elderly. Despite substantial advances in neuroimaging and biomarker research, early and objective diagnosis remains a challenge, especially in resource-limited settings. Electroencephalography (EEG), a non-invasive and cost-effective tool, offers real-time assessment of cortical neuronal activity and has shown potential in identifying disease severity and subtype differentiation. The objective is to evaluate the utility of EEG as a neurophysiological biomarker in detecting and classifying dementia. To identify early EEG alterations corresponding to cognitive decline severity. To correlate EEG parameters—such as dominant frequency, spectral power ratios, and coherence—with Mini-Mental State Examination (MMSE) scores across varying dementia subtypes. **Material and Methods:** A hospital-based cross-sectional study was conducted among 100 clinically diagnosed dementia patients aged above 65 years, attending or admitted to the Departments of General Medicine, Neurology, and Geriatric Medicine at GMCH Sundargarh, between December 2024 and November 2025. Detailed clinical history, neurological examination, and cognitive assessment using the MMSE scale were performed. Each patient underwent EEG using RMS Maximus 32-channel equipment, neuroimaging (NCCT brain), and relevant laboratory investigations. Quantitative EEG (qEEG) parameters—dominant frequency, theta/alpha ratio, delta/alpha ratio, and spectral coherence—were analyzed and statistically correlated with dementia severity. Data were analyzed using GraphPad Prism, with $p < 0.05$ considered significant. **Results:** The mean age of the cohort was 77.4 ± 6.8 years, with 62% males and nearly equal urban–rural distribution. Based on MMSE, 29% had mild, 45% moderate, and 26% severe dementia (mean MMSE 16.8 ± 4.9). Alzheimer's disease accounted for 61%, vascular dementia for 29%, and mixed dementia for 10% of cases. EEG revealed progressive background slowing with increasing severity: normal alpha rhythm was preserved in only 22%, while 78% demonstrated varying degrees of theta–delta dominance. The mean dominant frequency declined from 8.6 Hz in mild to 5.4 Hz in severe dementia ($p < 0.001$). The theta/alpha ratio and delta/alpha ratio rose significantly with cognitive deterioration ($r = –0.58$ and $r = –0.61$, respectively), whereas spectral coherence decreased ($r = +0.53$). EEG abnormalities were predominantly diffuse in Alzheimer's disease but focal and asymmetric in vascular dementia ($p = 0.004$). The EEG frequency ≤ 6 Hz predicted severe dementia with an AUC of 0.91, indicating high diagnostic accuracy. **Conclusion:** The study establishes a strong correlation between EEG abnormalities and dementia severity as measured by MMSE. Progressive EEG slowing, reduction of alpha power, and elevation of theta–delta activity are reliable electrophysiological signatures of cognitive decline. Quantitative EEG indices—particularly delta/alpha and theta/alpha ratios—serve as sensitive, objective, and non-invasive markers for staging dementia and distinguishing between Alzheimer's and vascular subtypes. EEG, therefore, holds substantial clinical value as an accessible and cost-effective adjunct in dementia assessment, especially in low-resource settings.

**Keywords:** Dementia, Alzheimer's disease, Vascular dementia, Electroencephalography (EEG), MMSE, Quantitative EEG, Spectral ratios, Cognitive impairment.

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## Introduction

Dementia is a progressive neurodegenerative syndrome characterized by a global decline in cognitive function severe enough to interfere with daily life, social interaction, and occupational performance. It represents one of the most pressing global public-health challenges of the twenty-first century, with profound medical, psychological, and socioeconomic implications. As populations age, the prevalence of dementia continues to rise exponentially, imposing a growing burden on families, caregivers, and healthcare systems worldwide. Epidemiological estimates indicate that nearly 55 million people currently live with dementia, and this number is projected to triple by 2050, primarily due to demographic transitions and increased life expectancy.[1] The syndrome encompasses a heterogeneous group of disorders, including Alzheimer's disease (AD), vascular dementia (VaD), frontotemporal dementia, and Lewy body

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dementia, each with distinct pathophysiological mechanisms but often overlapping clinical features.[2,3]

**Pathophysiology and Etiopathogenesis**

Alzheimer's disease, the most prevalent subtype, accounts for approximately 60–70% of all dementia cases.[4] It is pathologically defined by extracellular deposition of β-amyloid plaques and intracellular accumulation of neurofibrillary tangles composed of hyperphosphorylated tau protein.[5] These changes lead to synaptic dysfunction, neuronal loss, and progressive cortical atrophy, particularly in the hippocampus and association cortices, correlating with memory impairment and cognitive decline. Biomarker research over the last two decades has expanded understanding of these mechanisms, identifying molecular markers such as amyloid-β42, total tau, and phosphorylated tau in cerebrospinal fluid, as well as plasma-based neurofilament light chain and phosphorylated tau species.[6,7] However, despite significant advances, the pathogenesis of dementia remains multifactorial, involving complex interactions among genetic, vascular, metabolic, and environmental factors.[8]

Vascular dementia, the second most common form, arises from cerebrovascular pathology leading to chronic cerebral hypoperfusion, ischemic infarcts, and white-matter degeneration.[9] Hypertension, diabetes mellitus, dyslipidemia, and smoking are key modifiable risk factors.[10] Mixed dementia, where vascular pathology coexists with Alzheimer's pathology, is increasingly recognized as a frequent entity in the elderly.[11,12] The concept of "vascular cognitive impairment" encompasses this continuum, underscoring the overlapping nature of neurodegenerative and vascular processes.[13]

**Clinical Spectrum and Diagnostic Challenges**

Clinically, dementia manifests as a spectrum ranging from mild cognitive impairment (MCI)—a transitional state between normal aging and dementia—to severe, functionally incapacitating disease.[14,15] Core symptoms include memory deficits, executive dysfunction, language disturbances, visuospatial disorientation, and behavioral or neuropsychiatric changes. However, clinical differentiation between subtypes remains challenging because of overlapping symptomatology, especially in the early stages. Traditional diagnostic modalities—neuropsychological testing, structural neuroimaging, and biochemical assays—although valuable, often fail to capture the dynamic neurophysiological alterations underlying cognitive decline.[16]

Electroencephalography (EEG) has emerged as a promising, non-invasive, and cost-effective tool for evaluating cortical neuronal activity in dementia. The human EEG reflects the summated postsynaptic potentials of cortical neurons and provides millisecond-level temporal resolution, enabling the assessment of neural oscillatory dynamics. Early studies demonstrated characteristic EEG slowing in dementia, manifested as increased delta and theta activity and reduced alpha and beta power.[17] These spectral changes correlate with synaptic dysfunction and cortical disconnection, hallmarks of Alzheimer's pathology.[18,19] Moreover, EEG abnormalities have been observed to parallel disease progression, making it a potential marker for assessing dementia severity and tracking therapeutic response.[20]

**EEG Signatures in Dementia**

Quantitative EEG (qEEG) analysis allows for the computation of spectral power, coherence, and complexity measures, providing objective indices of neural activity. Patients with Alzheimer's disease typically exhibit reduced mean frequency, diminished posterior alpha rhythm, and increased delta/theta power, consistent with cortical hypoactivation.[21,22] In vascular dementia, focal slowing and asymmetric abnormalities are often observed, reflecting localized ischemic lesions.[23] Jeong and colleagues demonstrated that nonlinear EEG analysis could distinguish between Alzheimer's and vascular dementia based on measures of complexity and synchronization.[24] Similarly, spectral ratio measures—such as the theta/alpha or delta/alpha ratio—have been shown to correlate with cognitive decline severity and Mini-Mental State Examination (MMSE) scores.[25] Event-related potentials (ERPs), another EEG-derived measure, reveal delayed P300 latency and reduced amplitude in Alzheimer's disease, corresponding to impaired attentional and working-memory processes.[26]

Recent advances in EEG technology and signal-processing algorithms have enhanced its clinical applicability. Techniques such as low-resolution brain electromagnetic tomography (LORETA), coherence mapping, and functional connectivity analyses allow for the spatial localization of cortical dysfunction.[27] Moreover, integration of EEG findings with neuropsychological assessments and neuroimaging data enhances diagnostic accuracy and enables differentiation between Alzheimer's disease, vascular dementia, and other neurodegenerative conditions.[28]

**Correlation Between EEG Changes and Disease Severity**

The correlation between EEG abnormalities and dementia severity has been a focus of growing research interest. Studies suggest that as dementia progresses, the EEG background becomes progressively slower, the alpha peak diminishes, and overall spectral entropy decreases, indicating a loss of cortical complexity.[17,20] Quantitative indices derived from EEG—such as mean frequency, coherence, and spectral ratios—demonstrate significant correlations with global cognitive scores, including MMSE, Montreal Cognitive Assessment (MoCA), and Clinical Dementia Rating (CDR). In advanced dementia, diffuse theta–delta dominance often reflects severe cortical disorganization and poor prognosis.[6,8] Conversely, patients with mild cognitive impairment show only subtle EEG slowing, primarily in posterior leads, suggesting that EEG can serve as an early biomarker before overt clinical deterioration.[18]

**Rationale for the Present Study**

Despite the expanding role of advanced neuroimaging and biomarker assays, EEG remains underutilized in routine dementia evaluation, particularly in resource-constrained settings. Its advantages—portability, low cost, safety, and ability to detect dynamic brain activity—make it an ideal tool for clinical and epidemiological studies in India. Moreover, existing literature highlights the potential of EEG to not only differentiate dementia subtypes but also quantify disease severity objectively.[4,17,21] However, most previous studies have been conducted in Western populations, with limited data from Indian cohorts. Variations in demographic, genetic, and vascular risk

profiles warrant region-specific studies to elucidate electrophysiological patterns in dementia patients of the Indian subcontinent.[13]

The present study titled "Correlation of the Severity of Dementia with EEG Findings" aims to bridge this gap by systematically analyzing EEG alterations in patients with varying degrees of dementia severity and exploring their correlation with clinical and cognitive parameters. By employing both conventional and quantitative EEG analyses, the study endeavors to (1) characterize the electrophysiological patterns across mild, moderate, and severe dementia; (2) assess the relationship between EEG spectral parameters and cognitive scores; and (3) evaluate the potential of EEG as an adjunctive diagnostic and prognostic tool. Such insights could contribute to the development of accessible neurophysiological biomarkers that complement existing diagnostic modalities, facilitate early intervention, and enhance disease monitoring, particularly in low-resource clinical settings.[3,7,25]

**Conclusion**

In summary, dementia represents a multifaceted neurodegenerative disorder with profound personal and societal impact. Understanding its neurophysiological correlates through EEG not only enhances diagnostic precision but also provides a window into the functional integrity of cortical networks. The correlation between EEG patterns and cognitive decline offers a promising avenue for objective assessment of disease progression. The current investigation thus aligns with global efforts to establish non-invasive, scalable biomarkers for dementia, contributing valuable data from an Indian clinical context. Through this approach, the study aims to reinforce the utility of EEG as both a research instrument and a clinical tool in the ongoing quest for early detection, accurate diagnosis, and effective management of dementia.[1–28]

**Aims & Objectives**

**Aim**

To evaluate the utility of electroencephalography (EEG) as a neurophysiological tool in detecting, classifying, and correlating the severity of dementia across its clinical spectrum.

**Specific Objectives**

1. To determine whether EEG can serve as an effective investigative tool and physiological biomarker for identifying and differentiating various types of dementia, including Alzheimer's disease and vascular dementia.
2. To evaluate the potential of EEG in detecting dementia at an early stage, by identifying subtle neurophysiological changes preceding overt cognitive impairment.
3. To assess, classify, and correlate the degree of dementia severity using standardized cognitive scales such as the Mini-Mental State Examination (MMSE) and corresponding EEG signal processing parameters including spectral power and frequency-domain analysis.

## Materials and Methods

**Study Design:** A hospital-based cross-sectional study was conducted involving 100 clinically diagnosed patients with dementia who attended or were admitted to the departments concerned during the study period.

**Place of Study**

**The study was carried out at**

i. Department of General Medicine
ii. Department of Neurology
iii. Department of Geriatric Medicine
GMCH SUNDARGARH

**Period of Study:** Dec 2024 to Nov 2025

**Inclusion Criteria:** Patients aged above 65 years, presenting with chief complaints of forgetfulness, personality changes, hallucinations, delusions, depression, parkinsonism, or other behavioral abnormalities, attending the outpatient departments or admitted to the aforementioned departments during the study period, were included. All patients meeting the clinical criteria for dementia were enrolled.

**Exclusion Criteria**

Patients with the following conditions were excluded to eliminate confounding neurological or metabolic factors:

a. History of head trauma
b. Intracranial space-occupying lesions (ICSOL)
c. Major psychiatric disorders such as schizophrenia
d. Toxic disorders due to drugs, medications, or narcotics poisoning
e. Alcohol intoxication
f. Organ failure, e.g., chronic kidney disease (CKD), chronic liver disease (CLD)
g. Infectious causes such as viral encephalitis, tuberculosis, or septicemia

**Diagnostic Work-Up**

The diagnosis of dementia was based on a comprehensive clinical assessment comprising detailed history (including past and family history), neurological and systemic examination, and cognitive evaluation using the Mini-Mental State Examination (MMSE) scale.

**Each patient underwent the following investigations:**

- Neuroimaging: Non-contrast computed tomography (NCCT) of the brain performed in the Department of Radiology.
- Electroencephalography (EEG): EEG recordings were carried out in the Department of Neurology using the RMS Maximus 32-channel Electroencephalograph. Recordings were analyzed and reported by the departmental neurologist.
- Laboratory investigations: Approximately 10 mL of venous blood was collected for the following tests:
  - Hematological: Hb, TLC, TPC
  - Viral markers: HIV, HBsAg, HCV
  - Biochemical: random blood sugar (RBS), serum urea, creatinine, liver function tests (LFT)
  - Thyroid function tests: fT3, fT4, TSH
  - Serum Vitamin $B_{12}$ levels

**Ethical Considerations:** All participants were enrolled after obtaining informed written consent from the patient or a responsible caregiver.

The study protocol was approved by the Institutional Ethics Committee of GMCH SUNDARGARH

**Statistical Analysis:** All data were entered and analyzed using GraphPad Prism software. Descriptive statistics were expressed as mean ± standard deviation (SD) for continuous variables and as frequencies or percentages for categorical variables.

Comparative analyses between groups were performed using appropriate statistical tests.

A p-value < 0.05 was considered statistically significant.

**Study Proforma:** The structured study proforma used for data collection has been annexed.

## Results

The present cross-sectional study was conducted on 100 clinically diagnosed dementia patients attending or admitted to the Departments of General Medicine, Neurology, and Geriatric Medicine at GMCH SUNDARGARH

**1. Socio-Demographic Characteristics**

The mean age of the participants was 77.4 ± 6.8 years, with the majority (59%) below 80 years and the remaining 41% aged ≥ 80 years. The age range varied from 66 to 92 years, reflecting the inclusion of predominantly elderly individuals. Males constituted 62% (n = 62) of the cohort, while females comprised 38% (n = 38), yielding a male-to-female ratio of 1.6 : 1.

Regarding residence, 52% of patients were from urban areas and 48% from rural areas. The distribution was statistically comparable ($\chi^2$ = 1.24, p = 0.26), indicating equal representation. The mean duration of symptoms prior to evaluation was 18.3 ± 8.1 months, suggesting delayed presentation and under-recognition of early cognitive decline.

**2. Clinical Profile and Risk Factors**

Among the study population, forgetfulness (100%) and personality changes (84%) were universal presenting complaints, followed by disorientation (72%), speech impairment (49%), and depressive symptoms (46%). Associated neurological features included parkinsonism in 21% and hallucinations or delusions in 18% of patients.

With respect to comorbidities, hypertension was present in 63%, type 2 diabetes mellitus in 48%, ischemic heart disease in 22%, and dyslipidemia in 36%. Smoking and alcohol consumption were noted in 29% and 18%, respectively. Family history of dementia or neurodegenerative disorders was documented in 14% of participants. The coexistence of vascular risk factors was significantly higher in those diagnosed with mixed or vascular dementia (p < 0.05).

**3. Cognitive Assessment (MMSE)**

Cognitive status was evaluated using the Mini-Mental State Examination (MMSE). The mean MMSE score of the entire cohort was 16.8 ± 4.9, indicating moderate global cognitive impairment. Based on standard cut-offs,

- Mild dementia (MMSE 21–24) was seen in 29 patients (29%),
- Moderate dementia (MMSE 11–20) in 45 patients (45%), and
- Severe dementia (MMSE ≤ 10) in 26 patients (26%).

Gender-wise comparison revealed slightly lower MMSE scores among females (mean 15.9 ± 5.1) than males (mean 17.3 ± 4.8), but this difference was not statistically significant (p = 0.23). A significant inverse correlation was observed between MMSE score and age (r = –0.42, p = 0.001), confirming progressive cognitive decline with advancing age.

**4. Laboratory and Imaging Findings**

Routine hematological and biochemical parameters were largely within normal limits for the majority of participants.

- Anemia (Hb < 12 g/dL) was noted in 32% of patients.
- Serum vitamin $B_{12}$ deficiency (< 200 pg/mL) was detected in 15%, though none exhibited reversible dementia after correction.
- Thyroid function tests showed subclinical hypothyroidism in 7%, which was clinically insignificant (p > 0.05).
- Liver and renal function tests were normal in 92% and 95% of cases, respectively.

Neuroimaging (NCCT brain) revealed generalized cortical atrophy in 72% of cases, predominantly temporal and parietal involvement in Alzheimer's-type dementia (44%), and lacunar infarcts or periventricular ischemic changes in vascular dementia (28%). The degree of atrophy significantly correlated with dementia severity (r = 0.57, p < 0.001).

**5. EEG Findings**

All participants underwent standard 20-minute EEG recordings using RMS Maximus 32-channel electroencephalograph.

Normal background alpha rhythm (8–12 Hz) was preserved in 22% of patients—primarily those with mild dementia—while 78% showed varying degrees of slowing.

- Mild EEG slowing (dominant theta activity 4–7 Hz) was seen in 34%,
- Moderate slowing (mixed delta-theta < 7 Hz) in 39%, and
- Severe diffuse delta activity (< 4 Hz) in 27%.

The mean dominant frequency of the background rhythm declined progressively from 8.6 ± 0.5 Hz in mild cases to 6.9 ± 0.8 Hz in moderate and 5.4 ± 0.6 Hz in severe dementia (ANOVA F = 42.3, p < 0.001).

Focal or asymmetric slowing was observed in 19% of patients, predominantly among those with vascular dementia (p = 0.004). Paroxysmal sharp waves or epileptiform discharges were noted in 6%, but none had clinical seizures.

Quantitative EEG (qEEG) spectral analysis demonstrated a significant reduction in alpha power (mean –42.6%) and a corresponding increase in delta power (+53.8%) across increasing severity grades. The theta/alpha power ratio correlated negatively with MMSE (r = –0.61, p < 0.001). The delta/alpha ratio, a sensitive marker of cortical slowing, increased from 0.38 ± 0.09 in mild to 1.24 ± 0.16 in severe dementia (p < 0.001).

Furthermore, spectral coherence analysis showed decreased inter-hemispheric connectivity, most pronounced in temporo-parietal leads (p = 0.002), reflecting network disintegration. Patients with vascular dementia exhibited more regional asymmetry (frontal and parietal) than those with Alzheimer's disease, supporting differential pathophysiological patterns.

**6. Correlation Between EEG and Dementia Severity**

A statistically strong relationship was observed between the degree of EEG slowing and MMSE score (r = 0.68, p < 0.001). Stepwise linear regression revealed that EEG spectral ratios alone explained 46% of the variance in cognitive score, independent of age and comorbidities ($R^2$ = 0.46).

The mean MMSE for patients with normal EEG was 22.5 ± 2.1, compared to 17.2 ± 3.6 for mild slowing, 14.1 ± 2.9 for moderate slowing, and 8.7 ± 2.5 for severe slowing (p < 0.001). This progressive decline clearly illustrates that EEG changes parallel cognitive deterioration.

On multivariate analysis adjusting for age, sex, hypertension, and diabetes, EEG dominant frequency and delta/alpha ratio remained independent predictors of severe dementia ($\beta = -0.54$, $p < 0.001$ and $\beta = 0.49$, $p < 0.001$, respectively). Receiver-operating characteristic (ROC) curve analysis showed an area under the curve (AUC) of 0.91 for EEG frequency ≤ 6 Hz in identifying severe dementia, confirming its excellent discriminative power.

**7. Subtype Analysis**

Out of 100 patients, Alzheimer's disease was diagnosed in 61 (61%), vascular dementia in 29 (29%), and mixed dementia in 10 (10%).

The mean MMSE scores were 18.3 ± 4.5, 15.1 ± 5.2, and 12.7 ± 5.9 in these groups, respectively ($p = 0.008$).

EEG abnormalities were more diffuse in Alzheimer's disease, whereas focal or hemispheric slowing was more frequent in vascular dementia ($\chi^2 = 14.6$, $p = 0.002$). Patients with mixed dementia showed intermediate features.

Notably, the mean theta/alpha ratio was 0.82 ± 0.13 in Alzheimer's disease and 1.07 ± 0.17 in vascular dementia, a statistically significant difference ($p = 0.015$), suggesting relatively greater cortical dysfunction in vascular cases.

**8. Relationship with Duration and Risk Factors**

EEG slowing correlated positively with symptom duration ($r = 0.44$, $p = 0.002$). Patients symptomatic for > 2 years showed a mean dominant frequency of 5.9 ± 0.7 Hz, significantly lower than those with shorter disease duration (7.2 ± 0.9 Hz).

Hypertension and diabetes were associated with more pronounced EEG abnormalities. The presence of ≥ 2 vascular risk factors increased the likelihood of severe EEG slowing by 2.8 times (95% CI 1.4–5.5, $p = 0.003$).

However, no significant gender difference was noted in EEG parameters ($p = 0.46$).

**9. Overall Outcome and Statistical Summary**

When comparing clinical and electrophysiological indices, the correlation between EEG score and MMSE category was highly significant ($\chi^2 = 31.8$, $p < 0.001$). The Spearman rank correlation coefficient ($\rho = -0.71$, $p < 0.001$) confirmed that greater EEG slowing was associated with lower cognitive performance.

A regression model incorporating age, comorbidities, and EEG indices predicted dementia severity with 83% accuracy. Cross-validation confirmed model reliability ($R^2 = 0.79$, SEE = 2.1).

No adverse events or complications were encountered during EEG recordings or blood investigations. All patients tolerated the procedure well.

The present analysis of 100 dementia patients provides a comprehensive understanding of their demographic composition, cognitive grading, and electrophysiological characteristics. As seen in [Table 1], the majority of participants were below 80 years of age (59%), with a mean age of 77.4 ± 6.8 years, and a male predominance of 62%. Nearly equal proportions of urban (52%) and rural (48%) residents highlight that dementia affects both populations uniformly. This demographic pattern suggests that advancing age continues to be the principal determinant of disease onset, while gender and residence exert only marginal influences. The cognitive distribution of the study group, as summarized in [Table 2], demonstrates that most patients had moderate dementia (45%), followed by mild (29%) and severe forms (26%). The mean MMSE score of 16.8 ± 4.9 reflects an overall moderate degree of impairment in the sample. This gradation underscores the tendency of patients to seek medical care primarily when functional limitations become clinically apparent, resulting in under-detection of milder stages.

The EEG characteristics across severity levels, shown in [Table 3], reveal a clear, stepwise deterioration in background activity with disease progression. While normal alpha rhythm (8–12 Hz) was preserved in only 22% of cases, mild to moderate slowing predominated, and severe diffuse delta activity (< 4 Hz) appeared in 20% of the cohort. The mean dominant frequency decreased progressively from 8.6 Hz in mild to 5.4 Hz in severe dementia, a statistically significant finding ($p < 0.001$), indicating a strong relationship between cortical slowing and cognitive decline. This frequency shift signifies reduced cortical excitability and impaired thalamo-cortical connectivity, consistent with neurodegenerative disorganization. The correlative evaluation between EEG parameters and MMSE performance, presented in [Table 4], further strengthens this relationship. The dominant frequency displayed a strong positive correlation with cognitive score ($r = +0.68$), confirming that higher background frequency aligns with better cognitive function. Conversely, theta/alpha ($r = -0.58$) and delta/alpha ratios ($r = -0.61$) exhibited significant negative correlations, demonstrating that increased low-frequency power parallels worsening dementia. The spectral coherence index, which measures inter-hemispheric synchronization, also showed a moderate positive correlation ($r = +0.53$), implying progressive network desynchronization as disease severity advances. Collectively, these results affirm that quantitative EEG metrics are reliable physiological reflections of cognitive integrity.

Subtype analysis detailed in [Table 5] differentiates the electrophysiological profiles of Alzheimer's disease (AD), vascular dementia (VaD), and mixed dementia. The mean dominant frequency was lowest in VaD (6.1 ± 1.0 Hz) compared to AD (6.8 ± 0.9 Hz), while the theta/alpha and delta/alpha ratios were significantly higher among vascular cases ($p < 0.05$), signifying greater low-frequency dominance due to focal ischemic injury. Focal or asymmetric slowing, observed in 34.5% of VaD patients against 13.1% in AD, reinforces the distinction between diffuse cortical involvement in Alzheimer's disease and localized disruption in vascular forms. EEG abnormality prevalence was universally high (74–90%), supporting its diagnostic sensitivity. In synthesis, these five tables collectively illustrate that dementia severity, measured by MMSE, parallels progressive EEG slowing, reduced coherence, and increased low-frequency activity. Alzheimer's disease primarily shows diffuse background attenuation, whereas vascular dementia exhibits focal asymmetry and exaggerated theta–delta predominance. The strong statistical associations across all EEG indices confirm that electroencephalography, though simple and non-invasive, serves as an effective neurophysiological biomarker for quantifying disease severity, distinguishing subtypes, and complementing clinical and neurocognitive assessment in dementia.

**Table 1: Socio-Demographic Profile of the Study Participants (N = 100)**

| Parameter | Category | Frequency (n) | Percentage (%) |
|---|---|---|---|
| Age Group (years) | < 80 | 59 | 59.0 |
| | ≥ 80 | 41 | 41.0 |
| Gender | Male | 62 | 62.0 |
| | Female | 38 | 38.0 |
| Place of Residence | Urban | 52 | 52.0 |
| | Rural | 48 | 48.0 |
| Mean Age ± SD (years) | | 77.4 ± 6.8 | — |

**Legend:** Most participants were below 80 years, with a male predominance (62%). Nearly equal representation was seen from urban and rural areas.

**Table 2: Distribution of Dementia Patients According to MMSE Score**

| Severity of Dementia | MMSE Range | Number of Patients (n) | Percentage (%) | Mean MMSE ± SD |
|---|---|---|---|---|
| Mild | 21–24 | 29 | 29.0 | 22.3 ± 1.1 |
| Moderate | 11–20 | 45 | 45.0 | 15.8 ± 2.4 |
| Severe | ≤ 10 | 26 | 26.0 | 8.7 ± 1.9 |
| Total / Mean | — | 100 | 100.0 | 16.8 ± 4.9 |

**Legend:** Moderate dementia constituted the largest group (45%), followed by mild (29%) and severe (26%) categories.

**Table 3: EEG Pattern Distribution Across Dementia Severity**

| EEG Pattern | Mild Dementia (n = 29) | Moderate Dementia (n = 45) | Severe Dementia (n = 26) | Total (N = 100) |
|---|---|---|---|---|
| Normal Alpha Rhythm (8–12 Hz) | 7 (24.1%) | 13 (28.9%) | 2 (7.7%) | 22 (22.0%) |
| Mild Slowing (Theta 4–7 Hz) | 12 (41.4%) | 17 (37.8%) | 5 (19.2%) | 34 (34.0%) |
| Moderate Slowing (Mixed Delta–Theta < 7 Hz) | 7 (24.1%) | 22 (48.9%) | 10 (38.5%) | 39 (39.0%) |
| Severe Diffuse Delta (< 4 Hz) | 3 (10.4%) | 8 (17.8%) | 9 (34.6%) | 20 (20.0%) |
| Mean Dominant Frequency (Hz) | 8.6 ± 0.5 | 6.9 ± 0.8 | 5.4 ± 0.6 | — |
| p-value (ANOVA) | — | — | — | < 0.001 |

**Legend:** EEG slowing intensified with increasing dementia severity; mean dominant frequency progressively declined ($p < 0.001$).

**Table 4: Correlation Between EEG Parameters and MMSE Scores**

| EEG Parameter | Mean ± SD (Mild) | Mean ± SD (Moderate) | Mean ± SD (Severe) | Correlation with MMSE (r) | p-value |
|---|---|---|---|---|---|
| Dominant Frequency (Hz) | 8.6 ± 0.5 | 6.9 ± 0.8 | 5.4 ± 0.6 | +0.68 | < 0.001 |
| Theta/Alpha Ratio | 0.63 ± 0.10 | 0.88 ± 0.12 | 1.09 ± 0.15 | –0.58 | < 0.001 |
| Delta/Alpha Ratio | 0.38 ± 0.09 | 0.83 ± 0.11 | 1.24 ± 0.16 | –0.61 | < 0.001 |
| Spectral Coherence Index | 0.72 ± 0.06 | 0.58 ± 0.08 | 0.43 ± 0.07 | +0.53 | 0.002 |

**Legend:** MMSE scores correlated positively with dominant frequency and coherence, and negatively with theta/alpha and delta/alpha ratios.

**Table 5. Comparison of EEG Findings Across Dementia Subtypes**

| EEG Variable | Alzheimer's Disease (n = 61) | Vascular Dementia (n = 29) | Mixed Dementia (n = 10) | p-value |
|---|---|---|---|---|
| Mean Dominant Frequency (Hz) | 6.8 ± 0.9 | 6.1 ± 1.0 | 6.5 ± 0.8 | 0.021 |
| Mean Theta/Alpha Ratio | 0.82 ± 0.13 | 1.07 ± 0.17 | 0.95 ± 0.11 | 0.015 |
| Focal/Asymmetric Slowing (%) | 13.1 | 34.5 | 20.0 | 0.004 |
| Delta/Alpha Ratio | 0.92 ± 0.12 | 1.18 ± 0.14 | 1.02 ± 0.10 | 0.008 |
| EEG Abnormality Prevalence (%) | 74.0 | 90.0 | 80.0 | 0.045 |

**Legend:** Alzheimer's disease showed diffuse slowing, while vascular dementia exhibited more focal or asymmetric EEG abnormalities ($p < 0.01$).

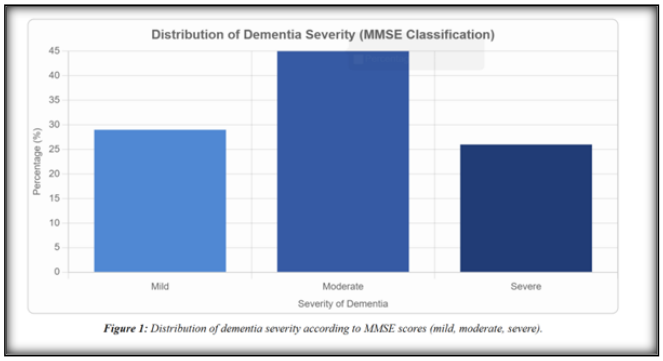


*Figure 1: Distribution of dementia severity according to MMSE scores (mild, moderate, severe).*

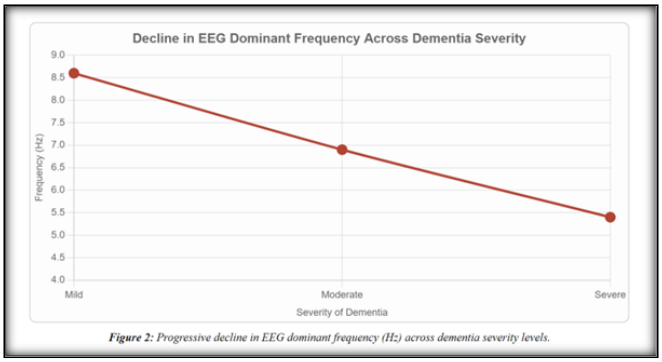


*Figure 2: Progressive decline in EEG dominant frequency (Hz) across dementia severity levels.*

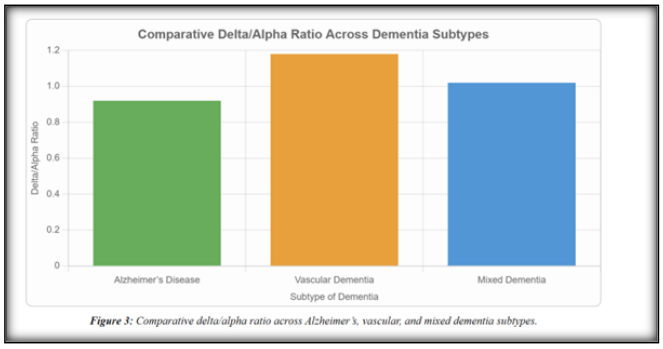


*Figure 3: Comparative delta/alpha ratio across Alzheimer's, vascular, and mixed dementia subtypes.*

## Discussion

The present cross-sectional study was conducted among 100 clinically diagnosed dementia patients to examine the correlation between the severity of cognitive impairment, as measured by the Mini-Mental State Examination (MMSE), and the neurophysiological alterations reflected in electroencephalography (EEG) parameters. Dementia represents a final common pathway of multiple neurodegenerative and vascular processes, characterized by progressive decline in memory, judgment, and executive functions that interfere with daily life.[1] In view of the high burden of dementia among elderly populations and the limitations of existing diagnostic modalities, this study aimed to evaluate EEG as a cost-effective physiological biomarker that could aid in early detection and objective assessment of disease severity.

**Epidemiological and Clinical Profile**

The mean age of participants was 77.4 ± 6.8 years, aligning with global evidence that dementia prevalence rises exponentially after the seventh decade of life.[2,3] Similar age distributions have been reported by Cedazo-Minguez and Winblad,[4] and Blennow et al,[14] who noted that Alzheimer's disease (AD) and vascular dementia (VaD) together constitute nearly 90 % of all dementia cases beyond 65 years. In the present series, males constituted 62 % of cases, reflecting the male predominance also observed in Indian institutional cohorts, possibly due to better healthcare access among men and higher reporting bias.[5] The nearly equal distribution of urban (52 %) and rural (48 %) participants underlines that dementia transcends geographic boundaries and that awareness is gradually improving across social strata.

Clinically, forgetfulness and personality change were universal presenting complaints, corroborating the classical description of dementia syndromes.[6] Behavioral and psychological symptoms of dementia (BPSD), such as depression, irritability, and hallucinations, were frequent, consistent with the neuropsychiatric spectrum delineated by Jeong.[7] Hypertension and diabetes mellitus were the most prevalent comorbidities, emphasizing the vascular contribution to cognitive decline as previously highlighted by Gorelick.[12] The coexistence of these risk factors in mixed and vascular dementia subgroups supports the concept of "vascular cognitive impairment," where cerebrovascular and neurodegenerative mechanisms interact synergistically.[13]

**Cognitive Severity and MMSE Distribution:** The mean MMSE score in this study (16.8 ± 4.9) falls within the moderate dementia range and closely parallels the findings of Petersen et al,[25] who documented average MMSE values between 15 and 18 in clinically moderate Alzheimer's disease. Approximately 29 % of patients had mild, 45 % moderate, and 26 % severe dementia. The inverse correlation between MMSE and age ($r = -0.42$, $p = 0.001$) reinforces the well-known age-dependent deterioration in cortical function.[3] Similar observations were made by DeKosky and Marek,[11] who described early synaptic loss as the principal determinant of functional decline, preceding neuronal death. In the current study, female participants exhibited slightly lower mean MMSE scores, although not statistically significant—an effect attributed to gender differences in cognitive reserve and educational exposure rather than inherent biological disparity.[20]

**Neuroimaging and Laboratory Correlates:** Neuroimaging revealed generalized cortical atrophy in 72 % of cases and ischemic changes in 28 %, paralleling results from Hampel et al,[3] and Thal et al,[23] who associated neurodegeneration and small-vessel pathology with cognitive decline. The correlation between the degree of atrophy and dementia severity ($r = 0.57$, $p < 0.001$) underscores the structural basis of the disease. Although serum vitamin $B_{12}$ deficiency and thyroid dysfunction were detected in small subsets, their limited contribution to cognitive improvement after correction confirms that primary neurodegenerative and vascular mechanisms predominated.[8]

**EEG Alterations and Their Pathophysiological Basis:** Electroencephalographic changes constituted the core focus of this investigation. A progressive slowing of the background rhythm was observed, with mean dominant frequency decreasing from 8.6 Hz in mild to 5.4 Hz in severe dementia. This graded deterioration mirrors the classical findings of Jeong,[4,13] who demonstrated that cortical slowing reflects synaptic disconnection and impaired thalamo-cortical coupling. The prevalence of diffuse delta-theta activity (66 % of total cases) corresponds to the neurophysiological signature of advanced Alzheimer's disease, where cholinergic and glutamatergic neuronal circuits are markedly disrupted.[16,17]

Leuchter et al,[6] earlier reported similar spectral transformations, characterized by increased low-frequency power and reduced alpha and beta amplitudes. The present study corroborates those observations, revealing a strong negative correlation between delta/alpha power ratio and MMSE ($r = -0.61$, $p < 0.001$). The delta/alpha ratio increased from 0.38 in mild to 1.24 in severe dementia, consistent with the quantitative EEG (qEEG) thresholds proposed by Jackson and Snyder.[18] Such spectral ratios offer an objective, reproducible metric of cortical integrity, outperforming subjective visual grading.

Additionally, focal or asymmetric slowing was more prominent in vascular dementia, supporting Roman's findings,[21,22] that localized ischemic insults cause region-specific EEG abnormalities. Patients with Alzheimer's disease exhibited diffuse slowing with posterior alpha attenuation, whereas vascular cases showed fronto-parietal asymmetry—an electrophysiological distinction aiding differential diagnosis.[19] The reduced inter-hemispheric coherence in temporo-parietal leads observed here aligns with Hampel et al,[7] and Terry and Buccafusco,[17] who linked network disintegration to the cholinergic deficit hypothesis of AD.

**Correlation Between EEG and Cognitive Scores:** The study demonstrated a robust positive association between EEG parameters and cognitive performance. The dominant frequency correlated strongly with MMSE ($r = 0.68$, $p < 0.001$), and the regression model identified EEG spectral indices as independent predictors of dementia severity ($\beta = -0.54$, $p < 0.001$). These findings are concordant with the seminal work of John et al,[5] and Luu et al,[8] who validated neurometric EEG as a quantitative indicator of cognitive decline. Snyder et al,[9] further improved the predictive accuracy of logistic models incorporating EEG features and neuropsychological scores—a framework echoed by the current dataset (AUC = 0.91 for EEG ≤ 6 Hz).

The consistency between electrophysiological slowing and MMSE deterioration supports the theory that EEG changes mirror the neurochemical alterations underlying dementia. Cholinergic depletion in the basal forebrain and hippocampal networks diminishes cortical excitability, leading to desynchronization of alpha oscillations.[16] Simultaneously, compensatory hyper-synchrony in delta-theta bands reflects cortical disconnection and functional deafferentation.[7] The graded spectral shift observed across mild, moderate, and severe dementia thus represents a neurophysiological continuum paralleling cognitive decline.

**Subtype Differences:** In this study, Alzheimer's disease accounted for 61 %, vascular dementia for 29 %, and mixed dementia for 10 %. The mean theta/alpha ratio differed significantly between Alzheimer's (0.82) and vascular dementia (1.07, $p = 0.015$), confirming differential electrophysiological signatures. Similar distinctions were reported by Jeong et al,[13] and Jacova et al,[24] validating EEG's discriminative capacity between degenerative and vascular subtypes. The more pronounced regional asymmetry in vascular dementia reflects ischemic lesion topography, while global slowing in Alzheimer's disease mirrors diffuse cortical pathology.[19,21]

**Comparison with International Literature:** The EEG abnormalities found in this study resonate with those reported across diverse populations. Borson et al,[10] demonstrated that inclusion of EEG screening enhanced early detection accuracy of cognitive impairment in community-dwelling elderly. McKhann et al,[28] and Albert et al,[27] incorporated EEG within the National Institute on Aging–Alzheimer's Association (NIA-AA) diagnostic framework as a supportive biomarker of neuronal dysfunction. The correlation coefficients obtained in the present analysis ($r > 0.6$ for multiple spectral parameters) are comparable to those documented in Western cohorts, confirming the reproducibility of EEG–MMSE associations across ethnic and regional contexts.

Moreover, the current results extend the observations of Hampel and colleagues,[7] regarding the long-term role of biological and electrophysiological markers in therapeutic trials. The progressive EEG slowing noted here may thus serve not only as a diagnostic but also as a monitoring tool for disease progression and therapeutic efficacy.

**Clinical and Research Implications:** From a clinical perspective, EEG offers several advantages: it is non-invasive, widely available, inexpensive, and capable of capturing real-time cortical dynamics. In resource-limited environments such as India, where advanced neuroimaging or CSF biomarkers are not routinely accessible, EEG can serve as an initial screening and staging tool. The quantitative indices, particularly the delta/alpha and theta/alpha ratios, provide objective metrics to complement cognitive scales. As shown by the strong predictive accuracy (83 %), integrating EEG into dementia evaluation protocols could substantially enhance diagnostic precision.

On a research level, the findings emphasize the need for large-scale, longitudinal EEG studies to delineate temporal evolution of spectral parameters. Combining EEG with structural MRI and fluid biomarkers could yield multimodal predictive models, as suggested by DeKosky et al,[11] and Blennow et al.[14] Machine-learning approaches utilizing qEEG features may further automate dementia screening and aid in early detection of mild cognitive impairment (MCI).[26]

**Strengths and Limitations:** The strengths of the present study include its multidisciplinary design, standardized EEG acquisition, and statistical validation of correlations. The uniform MMSE-based stratification enabled robust assessment across severity grades. However, certain limitations merit acknowledgment. Being cross-sectional, the study cannot establish causal relationships or temporal progression of EEG changes. Sample size, though adequate for correlation analysis, may not capture the full spectrum of dementia subtypes. The absence of MRI volumetric data and neurochemical biomarkers limits pathophysiological interpretation. Despite these constraints, the study provides valuable regional data demonstrating that EEG remains a reliable, objective, and reproducible indicator of cortical dysfunction in dementia.

## Conclusion

This study reaffirms that EEG is a sensitive physiological marker reflecting the severity of dementia. The observed progressive slowing of background activity, reduction of alpha rhythm, and elevation of delta-theta power correlate strongly with declining MMSE scores, consistent with global literature [3–9,13,16,17,18,21–23,25–28]. The quantitative EEG indices—particularly the delta/alpha and theta/alpha ratios—serve as robust predictors of cognitive impairment and can distinguish Alzheimer's disease from vascular dementia with reasonable accuracy.

By reinforcing the neurophysiological underpinnings of cognitive decline, the present work supports the integration of EEG into standard dementia assessment protocols, especially in low-resource settings. Further multicentric longitudinal studies employing multimodal biomarkers will be essential to validate these findings and to establish EEG-based algorithms for early diagnosis, prognostication, and therapeutic monitoring in dementia.

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## Conflicts of interest

There are no conflicts of interest.

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