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Original Article

Factors associated with cognitive impairment among adults with epilepsy: A cross-sectional study of clinical and electroencephalographic findings in Indonesia

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Abstract

The contributions of age at epilepsy onset, seizure burden, and electroencephalographic abnormalities to cognitive dysfunction remain incompletely understood, particularly among adults with epilepsy in resource-limited settings. The aim of this study was to evaluate the associations of age at epilepsy onset, seizure frequency, and electroencephalographic (EEG) findings with cognitive function in patients with epilepsy. A cross-sectional study was conducted among patients with epilepsy attending the Neurology Outpatient Clinic of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia, between November 2025 and January 2026. Cognitive function was assessed using the Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina), with scores <26 indicating cognitive impairment. Age at epilepsy onset, seizure frequency during the preceding 12 months, EEG findings, comorbidity status, and antiseizure medication regimen were obtained from medical records and structured interviews. Cognitive impairment was identified in 65.7% of patients, with a median MoCA-Ina score of 24 (range, 3–30). Patients with epilepsy onset at <10 or ≥40 years had a higher proportion of cognitive impairment than those with onset at 10–39 years (p=0.003); however, the logistic regression model failed to converge because of complete separation, precluding a valid odds-ratio (OR) estimate. Seizure frequency was not significantly associated with cognitive function (OR=0.991; p=0.306). In contrast, abnormal EEG findings were associated with lower odds of normal cognitive function (OR=0.260; p=0.031), while patients without comorbidities had higher odds of normal cognitive function than those with comorbidities (OR=5.40; p=0.004). Antiseizure medication regimen was not significantly associated with cognitive function (OR=1.53; p=0.405). Cognitive impairment was common among adults with epilepsy and was significantly associated with abnormal EEG findings and comorbidity status, while seizure frequency and antiseizure medication regimen were not significantly associated with cognitive function.

Keywords: Epilepsy, cognitive impairment, MoCA-Ina, seizure frequency, EEG

Introduction

Epilepsy is one of the most common chronic neurological disorders, affecting approximately 50 million individuals worldwide and contributing substantially to disability, premature mortality,

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and reduced quality of life [1]. According to the International League Against Epilepsy (ILAE), epilepsy is defined by the occurrence of at least two unprovoked seizures more than 24 hours apart, a single unprovoked seizure with a high probability of recurrence, or the diagnosis of a recognized epilepsy syndrome [2]. Despite advances in diagnostic and therapeutic strategies, epilepsy remains a major public health challenge, particularly in low- and middle-income countries where nearly 80% of affected individuals reside [3].

Beyond recurrent seizures, epilepsy is increasingly recognized as a disorder associated with a broad spectrum of neuropsychiatric and cognitive comorbidities [4]. Cognitive impairment represents one of the most prevalent and clinically significant complications, affecting up to 60–70% of patients with chronic epilepsy [4-8]. Deficits may involve multiple cognitive domains, including attention, memory, executive function, language, processing speed, and visuospatial abilities, often resulting in impaired academic achievement, occupational performance, social functioning, and overall quality of life [5].

The mechanisms underlying cognitive dysfunction in epilepsy are complex and multifactorial. Recurrent seizures can induce neuronal injury through excitotoxicity, oxidative stress, neuroinflammation, and dysregulated neuroplasticity, particularly within vulnerable structures such as the hippocampus and entorhinal cortex [4]. Experimental and clinical evidence suggests that repeated epileptic activity may disrupt neuronal networks involved in learning and memory, leading to cumulative cognitive decline over time [4-8]. Consequently, factors reflecting disease severity and chronicity, such as age at epilepsy onset and seizure frequency, have been proposed as important determinants of cognitive outcomes.

Previous studies have reported associations between earlier disease onset, higher seizure burden, longer disease duration, and poorer cognitive function in patients with epilepsy [7-9]. However, findings remain inconsistent across populations, and the contribution of objective neurophysiological markers has not been fully elucidated. Electroencephalography (EEG), a cornerstone investigation in epilepsy, provides valuable information regarding cortical dysfunction and epileptiform activity [10]. Abnormal EEG findings may reflect ongoing network disturbances that contribute not only to seizure generation but also to cognitive impairment [11]. Nevertheless, the association between EEG abnormalities and cognitive function remains incompletely understood, particularly when evaluated alongside clinical disease characteristics.

In Indonesia, where an estimated 1.1–1.8 million individuals are living with epilepsy [12], data regarding cognitive impairment and its associated factors remain limited. Moreover, few studies have simultaneously examined clinical variables and EEG findings within a unified analytical framework. Therefore, the aim of this study was to investigate the associations of age at epilepsy onset, seizure frequency, and EEG findings with cognitive function among patients with epilepsy.

Methods

Study design and setting

A cross-sectional analytical study was conducted at the neurology outpatient clinics of Dr. Zainoel Abidin Hospital, Banda Aceh, from November 2025 to January 2026. Adult patients with epilepsy aged 18–60 years were recruited using a total sampling approach. Demographic and clinical data, including age at epilepsy onset and seizure frequency during the preceding year, were obtained through medical record review and structured interviews. EEG findings were retrieved from the most recent interictal EEG recordings and cognitive function was assessed using the Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina).

Sampling method and eligibility criteria

As a total sampling strategy was applied, no formal sample size calculation was performed; the study size was determined by the number of eligible patients during the study period. Participants were eligible for inclusion if they (1) had a confirmed diagnosis of epilepsy, (2) were aged 18–60 years, and (3) provided written informed consent. Exclusion criteria were (1) a documented psychiatric disorder, including depression, anxiety, or psychosis; (2) intellectual disability; (3) inability to read and write; and (4) uncorrected visual or hearing impairment that could interfere with cognitive assessment.

Study variables and data collection

The dependent variable was cognitive function, assessed using MoCA-Ina. The primary independent variables were age at epilepsy onset, seizure frequency, and electroencephalographic (EEG) findings. Age at epilepsy onset was defined as the age at which the first unprovoked seizure occurred and was categorized as 0–9, 10–19, 20–39, or ≥40 years. Seizure frequency was defined as the number of seizures experienced during the preceding 12 months and was categorized as daily (>52 seizures/year), weekly (13–52 seizures/year), monthly (3–12 seizures/year), or yearly (1–2 seizures/year). EEG findings were classified as normal, abnormal non-epileptiform, or abnormal epileptiform. Comorbidity status and antiseizure medication regimen were included as clinical covariates and categorized as present or absent and monotherapy or polytherapy, respectively. Demographic characteristics included age, sex, educational level, and occupation. Educational level was classified as primary school or lower, junior high school, senior high school/vocational high school, or higher education, while occupation was categorized as unemployed/housewife/retired, student, or employed.

Cognitive function assessment

Cognitive function was assessed using MoCA-Ina, a validated screening instrument evaluating multiple cognitive domains, including visuospatial and executive function, naming, memory, attention, language, abstraction, and orientation. A total score <26 was classified as cognitive impairment, whereas a score ≥26 indicated normal cognitive function.

Electroencephalographic (EEG) assessment

EEG data were obtained from the most recent interictal EEG recordings documented in the medical records. EEG findings were interpreted by a board-certified neurologist and categorized as: (1) normal EEG; (2) abnormal non-epileptiform EEG, characterized by nonspecific slowing or background abnormalities without epileptiform activity; and (3) abnormal epileptiform EEG, characterized by the presence of epileptiform discharges, including spikes, sharp waves, spike-and-wave complexes, or polyspikes.

Statistical analysis

Association between categorical variables was evaluated using the chi-square test, and the strength of association was quantified using the contingency coefficient. Due to the small sample size, epilepsy onset age was categorized as <10 years or ≥40 years and 10–39 years to facilitate analysis by combining the extreme onset age groups, whereas EEG findings were classified as normal or abnormal. Logistic regression analysis was subsequently performed to evaluate the association between each independent variable and cognitive function. Model fit was assessed using the omnibus chi-square test, −2 log likelihood, Cox and Snell R^2, and Nagelkerke R^2. Comorbidity (present or absent) and antiseizure medication regimen (monotherapy or polytherapy) were analyzed as dichotomous independent variables. Separate univariable logistic regression analyses were performed to evaluate the association of each variable with cognitive function. All statistical analyses were performed using SPSS Version 25.0 (IBM Corp., Armonk, NY, USA), with statistical significance defined as two-tailed $p<0.05$.

Results

Baseline characteristics of patients

A total of 70 patients were included in the study and their characteristics are presented in **Table 1**. Among 70 patients, 40 (57.1%) were male and the majority were aged 20–39 years (71.4%, n=50). Most participants had completed senior high school or vocational high school (70.0%), and 45.7% were unemployed, housewives, or retired. Comorbid conditions were absent in 54.3% of patients, and 51.4% were receiving antiseizure medication monotherapy. The most common age at epilepsy onset was 10–19 years (42.9%), followed by 20–39 years (37.1%). Regarding seizure frequency, 42.9% of patients experienced 1–2 seizures per year, whereas 30.0% reported 3–12 seizures annually. EEG findings were predominantly normal (65.7%), followed by non-epileptiform abnormalities (25.7%) and epileptiform abnormalities (8.6%). Overall, 46

patients (65.7%) had cognitive impairment based on a MoCA-Ina score <26, whereas 24 (34.3%) had normal cognitive function. The median MoCA-Ina score was 24 (range, 3–30) (**Table 1**).

Table 1. Characteristics of the included patients (n=70)

Variable	Frequency (%)
Age (years)	
10–19	10 (14.3)
20–39	50 (71.4)
40–59	10 (14.3)
Sex	
Male	40 (57.1)
Female	30 (42.9)
Educational level	
Primary school or lower	3 (4.3)
Junior high school	3 (4.3)
Senior high school/vocational high school	49 (70.0)
Higher education	15 (21.4)
Occupation	
Unemployed/housewife/retired	32 (45.7)
Student	10 (14.3)
Employed	28 (40.0)
Comorbidity	
Present	32 (45.7)
Absent	38 (54.3)
Antiseizure medication regimen	
Polytherapy	34 (48.6)
Monotherapy	36 (51.4)
Age at epilepsy onset (years)	
0–9	9 (12.9)
10–19	30 (42.9)
20–39	26 (37.1)
≥40	5 (7.1)
Seizure frequency	
Daily (>52 seizures/year)	8 (11.4)
Weekly (13–52 seizures/year)	11 (15.7)
Monthly (3–12 seizures/year)	21 (30.0)
Yearly (1–2 seizures/year)	30 (42.9)
Electroencephalographic (EEG) findings	
Epileptiform abnormality	6 (8.6)
Non-epileptiform abnormality	18 (25.7)
Normal	46 (65.7)
Cognitive function (MoCA-Ina)	
Cognitive impairment (score 0–25)	46 (65.7)
Normal cognitive function (score 26–30)	24 (34.3)

Cognitive function according to age at epilepsy onset, seizure frequency, and EEG findings

Cognitive function according to age at epilepsy onset, seizure frequency, and EEG findings is presented in **Table 2**. The highest mean score on MoCA-Ina was observed among patients with epilepsy onset between 10 and 19 years of age (24.93±3.68), whereas the lowest score was found in those with onset between 0 and 9 years (16.00±8.33). A trend toward higher cognitive function was observed with decreasing seizure frequency, with mean MoCA-Ina scores increasing from 20.62±6.65 among patients experiencing daily seizures to 23.50±5.88 among those experiencing 1–2 seizures per year. Similarly, patients with normal EEG findings demonstrated higher mean MoCA-Ina scores (24.32±4.23) than patients with non-epileptiform EEG abnormalities (20.11±7.38) or epileptiform EEG abnormalities (19.00±9.16) (**Table 2**).

Table 2. MoCA-Ina scores according to age at epilepsy onset, seizure frequency, and electroencephalographic (EEG) findings

Variable	MoCA-Ina score, mean±SD
Age at epilepsy onset	
0–9 years (n=9)	16.00±8.33
10–19 years (n=30)	24.93±3.68
20–39 years (n=26)	23.50±5.38
≥40 years (n=5)	18.40±5.94

Variable	MoCA-Ina score, mean±SD
Seizure frequency	
Daily (n=8)	20.62±6.65
Weekly (n=11)	22.45±4.98
Monthly (n=21)	22.76±6.55
Yearly (n=30)	23.50±5.88
EEG findings	
Epileptiform abnormality (n=6)	19.00±9.16
Non-epileptiform abnormality (n=18)	20.11±7.38
Normal (n=46)	24.32±4.23

Association between age at epilepsy onset and cognitive function

Cross-tabulation showed that all participants with epilepsy onset before 10 years of age or at ≥40 years had cognitive impairment (100.0%) (**Table 3**). In contrast, among those with onset between 10 and 39 years, 32 (57.1%) had cognitive impairment and 24 (42.9%) had normal cognitive function. The contingency coefficient indicated a significant weak-to-moderate association between epilepsy onset age group and cognitive function (r=0.340, p=0.003), indicating a higher proportion of cognitive impairment in patients with extreme onset ages.

Table 3. Association between age at epilepsy onset and cognitive function

Age at epilepsy onset	Cognitive impairment	Normal cognitive function	Total	r	p-value[a]
	n (%)	n (%)	n (%)		
<10 years and ≥40 years	14 (100)	0 (0)	14 (100)	0.340	0.003
10–39 years	32 (57.1)	24 (42.9)	56 (100)		

[a] Analyzed using chi-square test; association strength was quantified using the contingency coefficient

Univariable logistic regression yielded a significant overall model (χ^2=13.522, p<0.001; Nagelkerke R^2=0.243) (**Table 4**). However, the model failed to converge due to complete separation, resulting in unstable coefficient estimates (B=−20.915, SE=1.074×10⁴, p=0.998). Therefore, the association between epilepsy onset age and cognitive function should be interpreted primarily from the bivariate analysis, which consistently demonstrated cognitive impairment in all participants with onset before 10 years or at ≥40 years.

Table 4. Univariable logistic regression analysis of the association between epilepsy onset age group and cognitive function

Variable	B	Standard error	Wald	df	Odds ratio	p-value[a]
Age at epilepsy onset	-20.915	1.074×10⁴	0.000	1	0.000	0.998
Constant	-0.288	0.270	1.135	1	0.750	0.287

Model statistics: omnibus χ^2=13.522, p<0.001; −2 log likelihood=76.486; Cox & Snell R^2=0.176; Nagelkerke R^2=0.243.
[a] Analyzed using logistic regression

Association between seizure frequency and cognitive function

Patients with cognitive impairment (n=46) had a higher median seizure frequency than those with normal cognitive function, with median frequencies of 5.5 (range, 0–240) and 3.0 (range, 0–144) episodes per year, respectively (**Table 5**). However, binary logistic regression analysis demonstrated no significant association between seizure frequency and cognitive function (B=-0.009; OR=0.991; p=0.306).

Table 5. Association between seizure frequency and cognitive function

Cognitive function	Seizure frequency (episodes/year), median (min–max)	B	Odds ratio	p-value[a]
Cognitive impairment (n=46)	5.5 (0–240)	-0.009	0.991	0.306
Normal cognitive function (n=24)	3.0 (0–144)			

[a] Analyzed using logistic regression

Association between electroencephalographic findings and cognitive function

Among patients with abnormal EEG findings, 20 (83.3%) had cognitive impairment and 4 (16.7%) had normal cognitive function (**Table 6**). In comparison, 26 (56.5%) of those with normal EEG findings had cognitive impairment and 20 (43.5%) had normal cognitive function.

A significant weak association was observed between EEG findings and cognitive function (r=0.268, p=0.025).

Table 6. Association between electroencephalographic (EEG) findings and cognitive function

Electroencephalographic (EEG) findings	Cognitive impairment	Normal cognitive function	Total	r	p-value[a]
	n (%)	n (%)	n (%)		
Abnormal	20 (83.3)	4 (16.7)	24 (100)	0.268	0.025
Normal	26 (56.5)	20 (43.5)	46 (100)		

[a] Analyzed using chi-square test; association strength was quantified using the contingency coefficient

The univariable logistic regression model was statistically significant (χ^2=5.396, p=0.020; Nagelkerke R^2=0.103) and converged successfully (**Table 7**). Abnormal EEG findings were independently associated with lower odds of normal cognitive function (B=−1.347, SE=0.623, OR=0.260, p=0.031), indicating that patients with abnormal EEG findings were less likely to have preserved cognitive function and had a higher likelihood of cognitive impairment.

Table 7. Univariable logistic regression analysis of the association between electroencephalographic (EEG) findings and cognitive function

Variable	B	Standard error	Wald	df	Odds ratio	p-value[a]
Abnormal EEG findings	-1.347	0.623	4.671	1	0.260	0.031
Constant	-0.262	0.297	0.778	1	0.769	0.378

Model statistics: omnibus χ^2=5.396, p=0.020; −2 log likelihood=84.612; Cox & Snell R^2=0.074; Nagelkerke R^2=0.103

[a] Analyzed using logistic regression

Association of comorbidity and antiseizure medication regimen with cognitive function

Univariable logistic regression demonstrated that comorbidity was significantly associated with cognitive function (**Table 8**). The overall model was statistically significant (omnibus χ^2=9.591, p=0.002), with Cox and Snell R^2=0.128 and Nagelkerke R^2=0.177, indicating that comorbidity explained approximately 13–18% of the variance in cognitive function. The presence of comorbidity was significantly associated with cognitive status (B=1.686, OR=5.400, p=0.004), indicating that patients without comorbidities had 5.4-fold higher odds of having normal cognitive function than those with comorbidities.

In contrast, the antiseizure medication regimen was not significantly associated with cognitive function (**Table 8**). The logistic regression model was not statistically significant (omnibus χ^2=0.700, p=0.403), with Cox and Snell R^2=0.010 and Nagelkerke R^2=0.014. Although patients receiving monotherapy had higher odds of normal cognitive function than those receiving polytherapy (B=0.423, OR=1.530, p=0.405), this association did not reach statistical significance.

Table 8. Univariable logistic regression analysis of the associations of comorbidity and antiseizure medication regimen with cognitive function

Variable	Comparison	B	Odds ratio	p-value[a]
Comorbidity	No comorbidity vs comorbidity	1.686	5.400	0.004
Antiseizure medication regimen	Monotherapy vs polytherapy	0.423	1.530	0.405

Comorbidity model: omnibus χ^2=9.591, p=0.002; Cox & Snell R^2=0.128; Nagelkerke R^2=0.177.
Antiseizure medication regimen model: omnibus χ^2=0.700, p=0.403; Cox & Snell R^2=0.010; Nagelkerke R^2=0.014.

[a] Analyzed using logistic regression

Discussion

The present study demonstrated that epilepsy onset age was significantly associated with cognitive function. Participants with epilepsy onset before 10 years of age or at ≥40 years uniformly had cognitive impairment, whereas a substantially lower proportion of cognitive impairment was observed among those with onset between 10 and 39 years. These findings suggest that both early- and late-onset epilepsy may represent periods of increased vulnerability

to cognitive dysfunction. Early-onset epilepsy was associated with lower scores across nearly all cognitive domains, particularly memory, language, and abstraction. This observation is biologically plausible because recurrent epileptic activity during critical periods of neurodevelopment may disrupt synaptic maturation, myelination, and frontotemporal network organization, leading to long-lasting cognitive consequences [13,14]. Previous studies have demonstrated that epilepsy beginning in childhood is associated with impaired working memory, attention, executive function, and reduced intellectual performance, particularly when onset occurs during early brain development [15,16]. Some cohorts have demonstrated that epilepsy beginning during childhood is associated with lower IQ, poorer academic achievement, and greater impairment across multiple cognitive domains [17,18]. Early seizure exposure may interfere with normal neurodevelopment by disrupting synaptic pruning, cortical maturation, and the establishment of large-scale cognitive networks [19]. Structural neuroimaging studies have further shown that early-onset epilepsy is associated with more extensive hippocampal atrophy, white matter disruption, and widespread cortical thinning [20,21].

Furthermore, late-onset epilepsy may predispose patients to cognitive impairment through both the underlying brain pathology and persistent epileptic activity [22]. In older adults, seizures frequently arise secondary to cerebrovascular disease, structural brain lesions, or neurodegenerative disorders, all of which independently contribute to cognitive decline [23]. Moreover, cognitive deficits may precede seizure onset, suggesting that epilepsy and cognitive impairment often represent parallel manifestations of a common underlying disease rather than a simple cause-and-effect relationship [24]. Ongoing epileptiform activity may further accelerate cognitive deterioration by disrupting neuronal networks, particularly in patients with temporal lobe hyperexcitability or drug-resistant epilepsy [25].

Similarly, patients with lower seizure frequency tended to show better cognitive performance, whereas those with daily seizures showed lower scores across most domains. Although statistical significance was not achieved, the observed trend is consistent with evidence indicating that recurrent seizures adversely affect hippocampal-dependent memory processes [5,26,27]. Experimental studies have demonstrated that repeated seizures impair long-term potentiation within the hippocampus, disrupt synaptic plasticity, and accelerate neuronal loss in temporal lobe structures, ultimately compromising memory formation and learning [27–30]. Memory and abstraction were the domains most vulnerable to increasing seizure burden in the present cohort, whereas orientation remained relatively preserved [5,26,27].

Seizure frequency was not significantly associated with cognitive impairment in the present study, although patients with cognitive impairment exhibited a higher median seizure frequency than cognitively normal patients. Each additional seizure per year was associated with only a minimal reduction in the odds of normal cognitive function, and the association did not reach statistical significance. This result differs from several previous studies that reported significant relationships between seizure burden and cognitive decline [15,31]. A clinical study has demonstrated that frequent seizures are associated with impairments in memory, attention, executive function, and overall cognitive performance [15]. In elderly patients with epilepsy, higher annual seizure frequency has been associated with substantially increased odds of cognitive impairment. Similar findings have been reported in both pediatric and adult populations [29].

The absence of a significant association in the present study may reflect the heterogeneous nature of epilepsy. Seizure frequency alone may not adequately capture cumulative disease burden. Factors such as seizure type, duration of epilepsy, underlying etiology, history of status epilepticus, and treatment response may exert stronger effects on cognition than the absolute number of seizures experienced within a single year. For example, generalized tonic–clonic seizures and prolonged status epilepticus are generally associated with greater neuronal injury than focal seizures without impaired awareness. Furthermore, seizure frequency measured over the preceding year may not accurately reflect lifetime seizure burden [32]. Cognitive impairment likely develops through cumulative exposure to recurrent epileptic activity over many years [5]. Consequently, short-term seizure counts may underestimate the long-term impact of epilepsy on cognitive trajectories.

The present study demonstrated that abnormal EEG findings were significantly associated with cognitive impairment. Patients with abnormal EEG findings had a higher prevalence of cognitive impairment, and univariable logistic regression showed that abnormal EEG was independently associated with lower odds of normal cognitive function. These findings align with previous studies showing that interictal epileptiform activity may interfere with cognitive processing by disrupting temporal and frontoparietal networks involved in memory encoding, attention, and executive function [5,27,33,34]. Notably, orientation remained relatively intact across all EEG categories, suggesting that distributed cortical networks supporting basic orientation may be more resilient to epileptic network dysfunction than hippocampal and executive systems [35].

At the transient level, interictal epileptiform discharges (IEDs) occurring during memory encoding or retrieval phases have been shown to reduce memory accuracy and prolong reaction time [36]. Intrahippocampal studies further demonstrate that IEDs arising within the critical 500–2,000 ms window surrounding encoding or recall decrease the probability of successful memory retrieval by 15–52% per event while increasing reaction time [37]. Similarly, IEDs involving the medial temporal and parietal cortices impair free recall when they occur during encoding or retrieval but have no significant effect during distractor periods [38]. These findings suggest that epileptiform activity, even in the absence of clinical seizures, can directly disrupt memory processing in a phase-specific manner.

In addition, the present study demonstrated that comorbidity was significantly associated with cognitive function, whereas the antiseizure medication regimen was not. Patients without comorbidities had more than fivefold higher odds of normal cognitive function than those with comorbidities. This finding is consistent with previous studies showing that cognitive impairment in epilepsy is multifactorial and often reflects the combined effects of epilepsy and underlying medical comorbidities rather than seizures alone [27,39]. Vascular and metabolic disorders, including hypertension, diabetes, dyslipidemia, and cardiovascular disease, may exacerbate cognitive decline through chronic inflammation, oxidative stress, endothelial dysfunction, and cerebrovascular injury, leading to structural and functional brain damage [27]. Furthermore, previous studies suggest that cognitive impairment may arise from the same underlying pathology responsible for epilepsy, highlighting the importance of identifying and managing comorbid conditions early in the disease course [4].

In contrast, no significant association was observed between the antiseizure medication regimen and cognitive function. Although patients receiving monotherapy tended to have higher odds of normal cognitive function than those receiving polytherapy, the association did not reach statistical significance. This finding is consistent with evidence indicating that the cognitive effects of antiseizure medications are heterogeneous and depend more on the specific drug, cumulative drug exposure, and patient characteristics than on the treatment regimen alone [40,41]. Certain antiseizure medications, particularly topiramate, valproate, and carbamazepine, have been associated with greater cognitive burden, whereas others appear to have minimal or neutral cognitive effects [40]. Therefore, the absence of a significant association between monotherapy and polytherapy in the present study should not be interpreted as evidence that antiseizure medications do not influence cognition [41]. Instead, future studies should evaluate the effects of individual medications, cumulative dosage, and treatment duration on cognitive outcomes.

This study has several limitations. The cross-sectional design precludes determination of causal relationships between clinical factors and cognitive function. Seizure frequency was assessed within a limited time frame and may not accurately reflect cumulative lifetime seizure burden, disease duration, or history of status epilepticus. EEG findings were categorized broadly as normal and abnormal, without quantification of interictal epileptiform discharge burden, sleep-related activation, or regional distribution, while routine EEG may have failed to capture intermittent epileptiform activity. In addition, potentially important confounders, including epilepsy syndrome, underlying etiology, disease duration, and antiseizure medication burden, were not evaluated in multivariable analyses. Future longitudinal studies incorporating cumulative seizure burden, quantitative prolonged EEG monitoring, neuroimaging biomarkers,

and comprehensive adjustment for clinical covariates are needed to better identify the determinants of cognitive impairment in epilepsy.

Conclusion

Cognitive impairment was common among adults with epilepsy in this study. Abnormal EEG findings and the presence of comorbidities were significantly associated with poorer cognitive status, whereas seizure frequency and antiseizure medication regimen were not significantly associated with cognitive function. Extreme ages at epilepsy onset were associated with cognitive impairment in the bivariate analysis; however, a valid regression estimate could not be obtained because of complete separation. These findings support the consideration of EEG abnormalities and comorbid conditions when assessing cognitive function in patients with epilepsy.

Ethics approval

The protocol of the present study was reviewed and approved by the Ethical Committee for Health Research, Dr. Zainoel Abidin Hospital, Banda Aceh (Approval number: 367/ETIK-RSUDZA/2025).

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Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

Artificial intelligence (AI) tool, ChatGPT, was used for language refinement (improving grammar, sentence structure, and readability of the manuscript). We confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the manuscript. The final decisions and interpretations presented in this article were solely made by the authors.

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