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RESEARCH ARTICLE

Prognostic value of interictal epileptiform discharges on routine EEG in adults with epilepsy

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Abstract

Objective: To determine whether interictal epileptiform discharges (IEDs) on routine electroencephalography (EEG) predict seizure recurrence in adults with established epilepsy.

Methods: We conducted a retrospective survival analysis of consecutive adults with epilepsy undergoing routine EEG at a tertiary center between 2018 and 2019. Using multivariate Cox proportional hazards models guided by a directed acyclic graph and adjusted for confounders including past seizure frequency and duration of epilepsy, we estimated the association between the presence of IEDs and time to next seizure, stratified by epilepsy type.

Results: We included 488 consecutive routine EEG studies from 438 patients. Over a median follow-up of 124.5 weeks, seizures recurred in 50.4% of cases. The presence of IEDs was associated with increased seizure risk in both focal (adjusted hazard ratio [aHR] = 1.47, 95% confidence interval [CI]: 1.01–2.15, $p = .043$) and generalized epilepsy (aHR = 1.82, 95% CI: 1.08–3.06, $p = .024$).

Significance: IEDs on routine EEG independently predict increased seizure risk in adults with epilepsy, with a stronger effect in generalized epilepsies. This suggests that routine EEG may have prognostic value during epilepsy follow-up and warrant further investigations as a potential prognostic biomarker to help inform clinical decision-making.

KEYWORDS

electroencephalography, epilepsy prognosis, seizure recurrence, survival analysis, interictal epileptiforme discharge

1 | INTRODUCTION

Routine electroencephalography (EEG) is a fundamental tool in evaluating adults with suspected or ongoing epilepsy. In patients presenting with a first unprovoked seizure, the presence of interictal epileptiform discharges (IEDs) on EEG is associated with an approximately two-fold risk of seizure recurrence at 4 years.[1,2] In addition,

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EEG can help classify epilepsy and diagnose an epilepsy syndrome,[3,4] predict seizure recurrence after epilepsy surgery,[5] or predict the risk of failure of antiseizure medication (ASM) withdrawal.[6–8] However, its utility in monitoring patients already diagnosed with epilepsy and treated with ASM is less certain.[7,9]

Experts generally advise against escalating ASM treatment based solely on EEG findings in seizure-free patients, even if the EEG study shows IEDs.[7,9,10] In neurology training, this is emphasized by the common teaching principle: "do not treat the EEG." However, given that seizure reporting is often unreliable,[11,12] clinicians could benefit from additional markers to guide prognostication and medication adjustments. Prior work in both generalized[13–15] and focal[16–18] epilepsies has been limited by small sample sizes and inadequate control of important confounders such as epilepsy duration, age, and epilepsy risk factors. As such, despite its clinical relevance, the relationship between IEDs on routine EEG and future seizure risk remains poorly defined.

The goal of this study was to assess how the presence of IEDs on routine EEG affects future seizure risk in a cohort of consecutive adults with epilepsy. To achieve this, we performed a retrospective survival analysis, adjusted for key factors including baseline seizure frequency, epilepsy duration, type of epilepsy, and risk factors for epilepsy.

Key points

- Interictal epileptiform discharges (IEDs) on routine electroencephalography (EEG) independently predict increased seizure risk in a cohort of 438 adults with established epilepsy, with a stronger effect in generalized epilepsies.
- This association remains significant after controlling for past seizure frequency and other confounders selected using a directed acyclic graph analysis.
- Findings suggest an expanded role for routine EEG in prognostication during epilepsy follow-up.

2 | METHODS

2.1 | Patient selection

We retrospectively recruited all consecutive patients with epilepsy who underwent routine EEG at the University of Montreal Hospital Center (CHUM) between January 2018 and June 2019. The diagnosis of epilepsy was based on the assessment by the treating neurologist at the last visit of follow-up, using all the data available during follow-up. We excluded EEG studies when there was no follow-up visit available before or after EEG, when the epilepsy diagnosis was uncertain at the end of the available follow-up, or when the EEG contained an electrical seizure (as per the EEG report). We reviewed the medical chart of each patient for clinical information. This included demographics (age, sex), reason for ordering the EEG, comorbidities at the time of the EEG, history of febrile seizures, first-degree relative history of seizures, duration of epilepsy, type of epilepsy, etiology of epilepsy, localization of epilepsy, presence of focal brain lesion on neuroimaging (when available), and number of ASMs. From the EEG report we extracted the type of recording (awake or sleep deprived), deepest sleep stage achieved, presence of IED(s), and presence of abnormal slowing. All clinical information was stored on a REDCap (Research Electronic Data Capture) database hosted on the CHUM Research Centre's secured servers. Ethics approval was granted by the CHUM Research Centre's Research Ethics Board (REB) (Montreal, Canada, project number: 19.334).

2.2 | Outcomes

The primary outcome was the time-to-seizure recurrence after EEG. For each EEG study, we collected the estimated date of first seizure after the EEG study based on the clinical note of the visit that followed EEG. When the date of first seizure after EEG was not explicitly provided in the clinical note, it was estimated by linear interpolation based on the reported seizure frequency at the visit following EEG (26% of cases).

The analysis was centered on three predictors: presence of IEDs, epilepsy type, and reason for ordering EEG. The EEG study was labeled as "presence of IED" when the conclusion of the report clearly stated that epileptiform discharges or spikes were present. If other terms were used ("irritative") or if the wording of the report cast doubts on the meaning of the anomalies ("potentially epileptiform discharges"), the presence of IED was labeled "uncertain." To maximize statistical power and maintain conservative estimates, uncertain IEDs were grouped with absence of IEDs for statistical analyses. Epilepsy type was labeled as "focal," "generalized," or "unknown" based on the clinical notes. To reduce the number of multiple comparisons, we pooled "unknown" type with "focal" epilepsy, based on the higher prevalence of focal epilepsies in the adult population.[19,20] Reasons for ordering EEG were divided into "follow-up," "characterization of epilepsy," "pre-ASM withdrawal," "post-operative," "new seizure

type," and "undiagnosed," based on current recommendations for the use of routine EEG in epilepsy.[7] "Follow-up" EEG studies were defined as routine EEG in patients with established epilepsy that did not meet other specific clinical indications.[7] These EEG studies were commonly ordered to monitor fluctuations in seizure frequency or to establish a baseline after transfer from another clinic.

2.3 | Statistical analyses

We performed a survival analysis to estimate the direct effect of the presence vs absence of IEDs on the risk of seizure recurrence through time. Observations were right-censored at the last available follow-up visit. Some patients contributed multiple EEG studies to the analysis, potentially with different IED status. To account for this within-patient correlation, we used a clustered variance estimator (Wei, Lin and Weissfeld's estimate[21]), which maintains valid statistical inference while allowing each EEG to contribute as a separate observation. In addition, we performed a sensitivity analysis including only the first EEG for each patient to confirm that our findings were not driven by repeat recordings.

The minimal set of covariates for the multivariate analysis was decided from a directed acyclic graph (DAG) (Figure S1). A DAG allows one to identify, under a given causal model, which covariables need to be controlled for in a multivariate analysis to properly estimate the relationship of interest (here, the effect of the presence of IEDs on seizure recurrence risk).[22] The DAG was built from domain knowledge according to the recommendations by Tennant et al.[22] The DAG was interpreted using the R library "dagitty." The minimal set of adjustment variables were: IEDs on EEG (presence, absence, uncertain), duration of epilepsy (log-transformed and mean-centered), type of epilepsy (generalized, focal, mixed, unknown), presence of focal lesion, sex, age (mean-centered), family history of seizures, history of febrile seizures, and active epilepsy. The number of ASMs was found to be a collider and thus was not included.[23] We performed an initial analysis to identify covariates with large effect size (Table S1); in the final iteration of the model, we allowed those terms to interact with the presence of IEDs. We performed a second analysis by including the effect of past seizure rate (log-transformed and mean centered), based on an alternative DAG in which the past seizure rate has a causal association with future seizure propensity.

To obtain the hazard ratio (HR) of the presence vs absence of IEDs depending on the type of epilepsy, we performed a multivariate survival analysis using the Cox proportional hazards model. We fit the model with the two sets of adjustment covariates described previously (minimal set and minimal set + past seizure rate). From both models we extracted the marginal effect that the presence of IEDs has on the risk of seizure recurrence, averaged over all other covariates. We repeated the analysis to calculate the HR of the presence vs absence of IEDs depending on the EEG reasons. This was done under a different model, where the presence of IEDs had an interaction with the reason for ordering the EEG study. In both cases, *p*-values for the HR were Tukey-adjusted for multiple comparisons. The marginal effects were computed using the R library "emmeans."[24]

2.4 | Sample size estimation and power analysis

Power analysis was performed using the R library "powerSurvEpi," according to Schmoor et al.[25] We estimated a range of sample sizes for a Cox proportional hazard model with a binary predictor and an interaction term. We assumed a rate of events of .5 and an insignificant correlation between the predictor and interaction term. We use a significance level of .05 and target power of .8. For an HR of 1.75, 2.0, 2.25, and 2.5, the sample sizes are 803, 523, 383, and 300 per group, respectively. Therefore, with an average of 1200 EEG studies per year, half of them from people with epilepsy, and an exclusion rate of .1, we estimated that all consecutive routine EEG studies from 1.5 years (included EEG studies ≈810) would provide us with sufficient statistical power. We also performed a retrospective power analysis to test these assumptions.

3 | RESULTS

3.1 | Patient characteristics

We screened 502 records for eligibility; 488 individual EEG recordings were included, from 438 patients (Table 1). Reasons for exclusion were uncertain diagnosis at the end of follow-up for seven EEG studies, unavailable follow-up after EEG for three studies, and electrical seizures on EEG for four EEG studies (three temporal lobe seizures and one generalized seizures; seizure duration range: 30 s to 6 min). Median age was 42 years (interquartile range [IQR]: 29–59 years), and median age of onset was 21.0 years (IQR: 13.0–39.5). Median follow-up after EEG was 124.5 weeks (IQR: 86.0–152.0 weeks). In 136 EEG studies, IEDs were detected by the appointed neurologist (27.9%); 308 studies did not show IEDs (63.1%), and 44 EEG studies had potentially epileptiform transient(s) ("uncertain IEDs," 9.0%). There were 348 EEG studies from patients with focal epilepsy (71.3%), 121 for generalized epilepsy (24.8%), and 19

TABLE 1 Description of the population studied.

	Overall population
No. of EEG studies (no. of patients)	488 (438)
Age. median [IQR]	42.00 [29.00, 59.00]
sex = female (%)	249 (51.0)
Interictal epileptiform discharges, *n* (%)	
Absence	308 (63.1)
Presence	136 (27.9)
Uncertain	44 (9.0)
Seizure recurrence after EEG, *n* (%)	246 (50.4)
Total follow-up after EEG in weeks, median [IQR]	124.50 [86.00, 152.00]
N epilepsy risk factors, median [IQR]	2.00 [1.00, 4.00]
Febrile convulsions, *n* (%)	18 (3.7)
Family history, *n* (%)	54 (11.1)
Age at epilepsy onset, median [IQR]	21.00 [13.00, 39.50]
Epilepsy type, *n* (%)	
Focal	348 (71.3)
Generalized	121 (24.8)
Unknown	19 (3.9)
No. of days since last seizure, median [IQR]	247.00 [55.50, 1162.00]
No. of ASMs, *n* (%)	
0	51 (10.5)
1	262 (53.7)
2	118 (24.2)
3	46 (9.4)
4	10 (2.0)
5	1 (0.2)
Focal lesion on brain imaging, *n* (%)	209 (42.8)
Sleep-deprived EEG, *n* (%)	58 (11.9)
Abnormal slowing on EEG, *n* (%)	192 (39.3)
Focal slowing on EEG, *n* (%)	145 (29.7%)
Deepest sleep achieved on EEG, *n* (%)	
Awake	196 (41.6)
N1	192 (40.8)
N2	75 (15.9)
N3	5 (1.1)
N4	2 (0.4)
REM	1 (0.2)

Abbreviations: EEG, electroencephalography; IQR, interquartile range; No, number; REM, rapid eye movement.

EEG studies from patients with epilepsy of unknown type (3.9%).

Among patients with generalized epilepsy ($n = 106$), 67 (63%) had idiopathic generalized epilepsy (as defined by the International League Against Epilepsy [ILAE] in[4]), whereas 33 (31%) did not have a well-characterized syndrome; two additional patients had epilepsy with eyelid myoclonia and three had epileptic encephalopathies (Table 2). A sensitivity analysis excluding patients with well-characterized syndromes showed results similar to our main analysis (Table S3).

TABLE 2 Epilepsy syndromes in patients with generalized epilepsy.

	Number of patients (%)
Generalized tonic–clonic seizures alone	31 (29%)
Juvenile myoclonic epilepsy	19 (18%)
Juvenile absence epilepsy	15 (14%)
Childhood absence epilepsy	2 (2%)
Epilepsy with eyelid myoclonia	2 (2%)
Other	3 (3%)
None	33 (31%)

3.2 | Survival analysis

Overall, seizure recurrence after EEG occurred in 246 EEG studies (50.4%). Median time-to-event was 651 days (95% confidence interval [CI]: 459–949). At 1 year, there were 238 patients at risk and 201 events, for an estimated 1-year seizure-free survival of 57% (0.52–0.61). The hazard function for seizure-free survival in the presence vs absence of IEDs on index EEG is presented in Figure 1.

The unadjusted HR for seizure recurrence in the presence vs absence of IEDs was 1.65 (1.27–2.15, $p < .001$): on average, during the study period, patients with IEDs on routine EEG were 1.65 times more at risk of seizure recurrence than patients with no IEDs.

We evaluated the effect of IEDs depending on the type of epilepsy using a multivariate Cox proportional hazards model (Table 3). In patients with focal epilepsy, the presence of IEDs on EEG conferred a 1.68-fold increase (1.17–2.39, $p = .0044$) in the HR of seizure recurrence compared with the absence of IEDs when controlling for age, sex, presence of focal lesion, history of febrile convulsions, history of seizures in first-degree relatives, and time from epilepsy onset to EEG. In patients with generalized epilepsy, the HR for seizure recurrence was 1.89 [1.14–3.13, $p = .0141$].

When considering the effect of past seizure rate, the marginal adjusted HR (aHR) for presence vs absence of IEDs in focal epilepsy was 1.47 (1.01–2.15, $p = .0427$) and in generalized epilepsy, 1.82 (1.08–2.15, $p = .0241$). The survival function for each epilepsy type is presented in Figure 2. The estimates for the full set of covariates for both models are provided as Table S2.

3.3 | Impact of the reason for ordering EEG

We estimated the effect of the presence of IEDs depending on the reason that EEG was ordered. The distribution of reasons is displayed in Table 4. Most EEG studies (265) were ordered during epilepsy follow-up, that is, an EEG study that should not, in itself, impact the management of epilepsy: this includes a change in seizure frequency or "baseline" EEG after being transferred from another clinic. The second most frequent reason (74 EEG studies) was to characterize epilepsy in a patient with newly diagnosed epilepsy (differentiate focal vs generalized epilepsy, localize an epileptic focus, or diagnose an epilepsy syndrome). In 79 distinct cases, the diagnosis of epilepsy was not clear at the time of EEG but was established during follow-up, with no overlap with other EEG indications.

The aHR between the presence and absence of IEDs in EEG studies that were ordered as part of follow-up was 2.27 (1.55–3.32, $p < .0001$). In other reasons, the aHR was non-significant between the presence and absence of IEDs (Table 5).

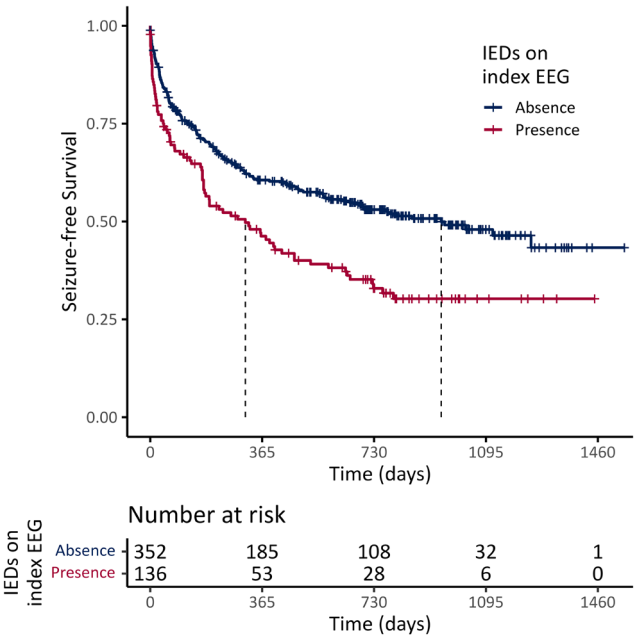

FIGURE 1 Seizure-free survival after routine EEG in patients with epilepsy, stratified by the presence vs absence of IEDs on EEG. EEG, electroencephalography; IEDs, interictal epileptiform discharges.

3.4 | Post hoc power analysis

We performed a post hoc power analysis to calculate the power of the Cox proportional hazards model using the data that were included in the study. Using the conservative formula proposed by Schmoor et al., which decreases the power in the presence of interaction term,[25] the power was above .80 for HR estimates larger than 1.5. For HRs of 1.15, 1.25, and 1.40, the power was .16, .34, and .65, respectively.

3.5 | Sensitivity analyses

We performed sensitivity analyses to evaluate the robustness of the Cox proportional hazards model to the following subgroups: patients with IGE and patients with repeated EEG studies. In both cases, the aHR for IEDs remained significant (Table S3). In addition, we noted that IEDs frequently co-occurred with focal slowing (56.1% of EEG studies with focal slowing had IEDs vs 29.2% without, $p = .01$). Adding focal slowing as a covariable to the Cox proportional hazards model showed that our estimates were robust to this potential confounder (Table S3).

4 | DISCUSSION

In this retrospective study, the presence of IEDs on routine EEG independently predicted increased seizure risk

TABLE 3 Adjusted hazard ratios between presence vs absence of IEDs on seizure-free survival after routine EEG dependent on the type of epilepsy.

	aHR (minimal sets of covariates only)	*p*-value[a]	aHR (+ adjusted for past seizure rate)	*p*-value[a]
Focal epilepsy				
Presence vs absence of IEDs	1.68 [1.17–2.39]	.0044	1.47 [1.01–2.15]	.0427
Generalized epilepsy				
Presence vs absence of IEDs	1.89 [1.14–3.13]	.0141	1.82 [1.08–3.06]	.0241

Abbreviations: aHR, adjusted hazard ratio; EEG, electroencephalography; IEDs, interictal epileptiform discharges.

[a]Tukey-adjusted for multiple comparisons.

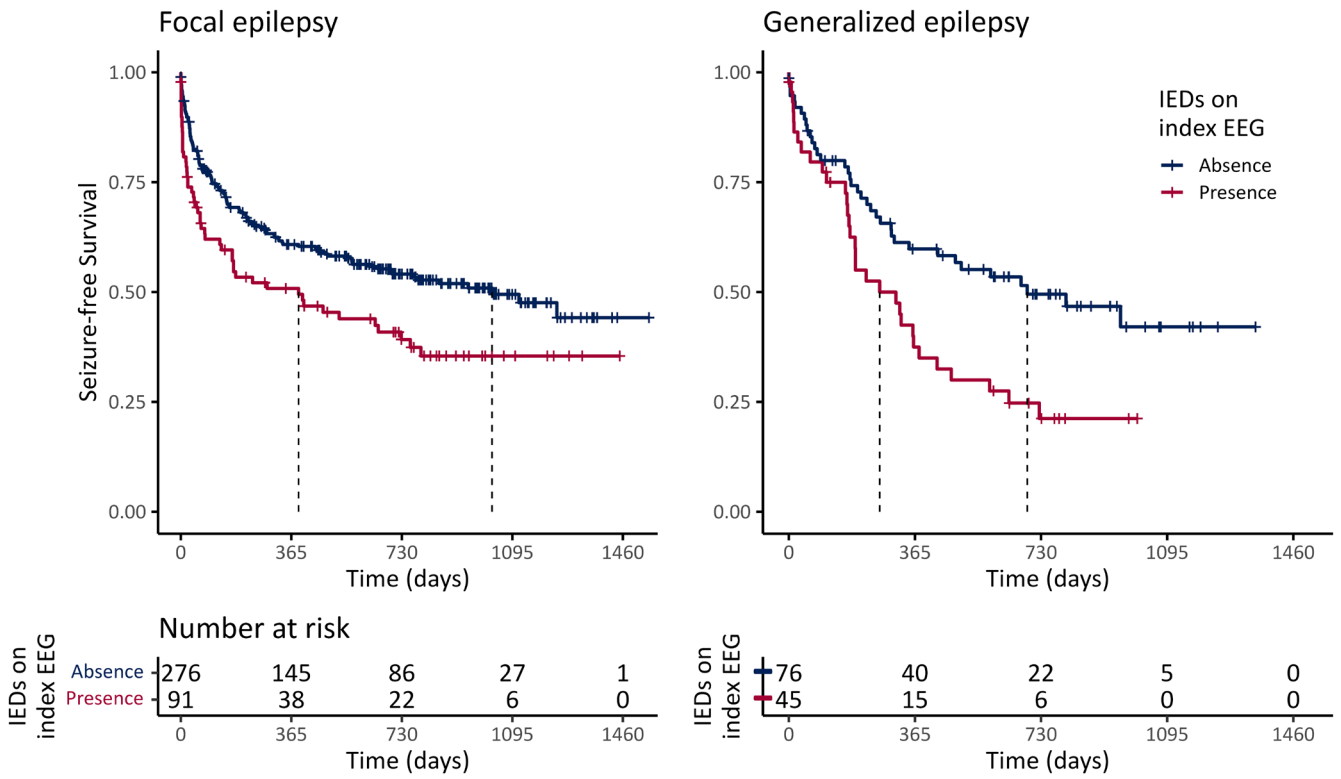

FIGURE 2 Seizure-free survival after routine EEG in patients with focal and generalized epilepsy, stratified by the presence vs absence of IEDs on EEG. EEG, electroencephalography; IEDs, interictal epileptiform discharges.

TABLE 4 Reasons for ordering index EEG.

	Number of EEG studies
Follow-up EEG	265
Characterization of epilepsy	74
Pre-withdrawal	42
Post epilepsy surgery	21
New seizure type	5
Undiagnosed	
Transient neurological deficit	31
Altered mental status	19
Single unprovoked seizure	16
Abnormal movements	12
Syncope	1
Other	2

Abbreviation: EEG, electroencephalography.

TABLE 5 Adjusted hazard ratios between presence vs absence of IEDs on seizure-free survival after routine EEG dependent on the reason for ordering the EEG.

	aHR (adjusted for past seizure rate)	*p*-value[a]
Follow-up		
Presence vs absence of IEDs	2.27 [1.55–3.32]	<.0001
Characterization of epilepsy		
Presence vs absence of IEDs	1.12 [0.55–2.30]	.753
Pre-ASM withdrawal		
Presence vs absence of IEDs	0.99 [0.28–3.53]	.997
Undiagnosed epilepsy		
Presence vs absence of IEDs	1.07 [0.55–2.05]	.857

Abbreviations: aHR, adjusted hazard ratio; ASM, antiseizure medication; EEG, electroencephalography; IEDs, interictal epileptiform discharges.
[a]Tukey-adjusted for multiple comparisons.

in adults with epilepsy, with aHRs of 1.48 in focal epilepsy and 1.87 in generalized epilepsy. These findings suggest that the presence of IEDs on routine EEG carries a prognostic value during follow-up of patients with epilepsy, regardless of epilepsy type, which could extend its use beyond the currently recommended indications (i.e., before ASM withdrawal, with new seizure types, or after epilepsy surgery).

The clinical significance of IEDs has evolved since the early days of EEG, when any EEG abnormality prompted treatment.[26] Evidence that IEDs can occur in individuals without epilepsy[27] and are often overinterpreted[28] has led

to a more nuanced approach emphasizing clinical correlation. Existing evidence supports the prognostic value of IEDs in specific contexts: after a first unprovoked seizure,[1,2,29] before ASM withdrawal,[6–8,30] and after epilepsy surgery.[5] Our findings shift focus to a different, understudied population: patients already diagnosed and treated for epilepsy. Despite representing the bulk of epilepsy practice, these patients lack reliable biomarkers capable of capturing the risk of seizures through time.[31] This information could have significant clinical impact: optimization of ASM regimens, increased suspicion for unreported seizures, refining safety recommendations for driving and other activities, and earlier identification of patients who might require alternative treatments such as surgery or neuromodulation.[31,32]

The association between IEDs and seizure risk is more pronounced in patients with generalized epilepsy. In this group, the presence of IEDs nearly doubles the risk of seizures, independent of past seizure frequency. The interictal hallmark of generalized epilepsy is bilateral spike–wave activity, typically manifesting as 2–5 Hz bursts, reflecting the hijacking of the cortico-thalamo-cortical networks by epileptic discharges.[4,13,33–35] The rhythmic and sometimes prolonged nature of this activity can blur the boundary between interictal and ictal activity, which likely explains clinicians' lower threshold for adjusting ASMs based on IEDs in this subgroup. Still, previous research on the prognostic values of interictal activity on EEG in generalized epilepsy has been inconclusive. A 2012 review of 20 studies revealed conflicting results,[4] with most studies predominantly including children and focusing on epilepsy remission—an outcome achieved less commonly in adult-onset generalized epilepsy.[36–39] A 2019 retrospective cohort study also did not find an association between long-term seizure outcomes and epileptiform anomalies on EEG studies at ASM initiation (specifically: prolonged interictal discharges and generalized polyspike trains).[40] In addition, a 2023 systematic review studying the effect of ASM on epileptiform discharges found that although ASM tended to reduce epileptiform discharges on 24 h ambulatory EEG, the association between epileptiform discharge burden and seizure control was unclear.[41] Our study, comprising exclusively adults, adds to this body of literature by providing robust evidence supporting the prognostic value of IEDs in generalized epilepsy, and could reinforce the sentiment that epileptiform anomalies should optimally be suppressed in generalized epilepsies.[13,41] However, we did not distinguish based on the morphology, frequency, or abundance of the discharges, and further studies are needed to explore whether these characteristics carry specific prognostic implications.

In focal epilepsies, IEDs are associated with an ~1.5-fold risk of seizures, an association more modest than for generalized epilepsies but still significant. Again, our findings contrast with previous research: a cohort study of 129 consecutive patients undergoing routine EEG found no statistically significant association between the presence of IEDs and recent seizure frequency,[16] whereas a systematic review of seizure outcomes following ASM initiation found mixed results.[42] Several factors explain these discrepancies. Previous studies were underpowered, with sample sizes ranging from 64 to 154 patients—insufficient to detect our study's modest effect size.[43–45] In addition, these studies failed to control for important confounders that influence both IED prevalence and pharmacoresistance, such as duration of epilepsy, localization of the epileptic focus, and presence of a lesion.[43–45] Our study controlled for these predictors, which were identified using a DAG. Given the critical understudied nature of this association and the potential clinical implications, further investigation of the prognostic value of IEDs in focal epilepsy is warranted.

Our study focuses on routine EEG recordings, which remain the mainstay in the electrophysiological evaluation of epilepsy. In our center, routine EEG studies are still favored over ambulatory EEG, and most studies are ordered by five fellowship-trained epileptologists. However, ambulatory EEG recordings are increasingly used and provide higher diagnostic yield (equivalent to three to four routine EEG studies).[46,47] Given their heightened sensitivity for detecting IEDs, ambulatory EEG recordings could offer even greater prognostic value for seizure risk prediction, although this has yet to be investigated. Ultimately, practice patterns may vary significantly between centers based on local expertise, resources, and health care systems. As such, our findings may not be generalizable to centers with vastly different practice patterns.

Although our findings suggest the prognostic value of routine EEG in epilepsy follow-up, they should be interpreted cautiously. EEG results must always be contextualized within the overall clinical picture, and should not be interpreted as direct evidence supporting EEG-guided treatment escalation. Increasing ASM doses or adding agents carries significant risks of side effects, which can substantially impact quality of life.[48–50] The optimal balance between seizure control and treatment burden, and the real impact of EEG-guided management, can be determined only through rigorous prospective trials comparing EEG-guided vs standard treatment approaches. Such studies should evaluate outcomes beyond seizure control, such as quality-of-life measures and adverse effects of more aggressive ASM regimens.

The study has limitations. Our study focused on the presence vs absence of IEDs, but did not evaluate other features such as the abundance or morphology of the discharges, which could impact the association with seizure

risk. The inherent indication bias limits our analysis to patients referred for EEG; we attempted to mitigate this bias by stratifying by referral reasons. A treatment bias also exists, wherein IEDs could have prompted for ASM adjustments, thereby potentially decreasing subsequent seizure risk. However, being conservative, this bias would only decrease the observable effect size. Other factors that could decrease the observable effect size include the inherent unreliability of seizure reporting, our binary classification of IEDs without consideration of their morphology or abundance, and the large number of EEG recordings for which the exact date of next seizure was unavailable (26%). Finally, there is insufficient power for some secondary analyses, particularly in ASM withdrawal, post-surgical, and undiagnosed subgroups.

5 | CONCLUSION

In this retrospective cohort study, we found that IEDs on routine EEG independently predict increased seizure risk in both focal and generalized epilepsies. This association persists after controlling for past seizure frequency and other confounders, with a stronger effect in generalized epilepsies. Our findings suggest that routine EEG carries prognostic value beyond its currently recommended uses, potentially informing clinical decision-making in medication adjustments, safety recommendations, and the selection of patients for alternative treatments. Prospective studies are needed to determine the impact of EEG-guided management on seizure outcomes.

AUTHOR CONTRIBUTIONS

E.L., M.J., D.K.N., F.L., and E.B.A. conceived and planned the experiments. E.L., A.Q.X., M.J., and J.D.T. collected the data. E.L., A.Q.X., M.J., J.D.T., and E.B.A. had direct access and verified the underlying data. E.L. and M.J. performed the analyses. E.L., M.J., D.K.N., F.L., and E.B.A. contributed to the interpretation of the results. E.L. wrote the first draft of the manuscript. All authors provided critical feedback and reviewed the manuscript.

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CONFLICT OF INTEREST STATEMENT

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DATA AVAILABILITY STATEMENT

Anonymized data in the form of clinical variables will be provided upon reasonable request to the corresponding author and is conditional to the approval by our ethics research board.

ETHICS APPROVAL STATEMENT

The study's protocol was approved by the local Ethics Review Board (project number: 19.334). We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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