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Diagnostic Utility of Electroencephalography and Supplementary Laboratory Investigations in Adult Epileptic Patients in Central India: A Case-Control Study

Richa Khanna[1*], Dr. Ashutosh Jain[2], Dr. Jaya Jain[3]

[1]Research Scholar-Ph.D, Department of Physiology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India.
[2]Assistant Professor, Department of Physiology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India. Email: ashutosh.jain689@gmail.com
[3]Professor, Department of Biochemistry, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India. Email: jain02jaya@gmail.com.

Corresponding Author:

Richa Khanna
Research Scholar-Ph.D, Department of Physiology, Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India.
Email: richakhan83348@gmail.com

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ABSTRACT

Background: Epilepsy remains a major neurological and public health concern in low- and middle-income regions, where delayed diagnosis, limited specialist access and restricted availability of advanced neurodiagnostic facilities contribute to a persistent treatment gap. Electroencephalography (EEG) is a key supportive investigation, but its value is greatest when interpreted with clinical and laboratory information. Objective: To assess the diagnostic utility of routine EEG and selected supplementary laboratory investigations in adult epileptic patients in Central India. **Methods**: This case-control study included 186 adults aged 18-60 years, comprising 93 clinically diagnosed epilepsy cases and 93 non-epileptic controls with comparable neurological complaints. Socio-demographic variables, clinical seizure profile, routine EEG findings, complete blood count, electrolyte panel, urine analysis and management-related variables were evaluated. Diagnostic indices were calculated using clinical epilepsy status as the reference classification. **Results**: Abnormal EEG findings were recorded in 62 cases (66.7%) and 18 controls (19.4%), showing a significant association between EEG abnormality and epilepsy status ($p<0.001$). Interictal spikes, sharp waves, generalized discharges and focal discharges were significantly more frequent among cases. EEG showed sensitivity of 66.7%, specificity of 80.6%, positive predictive value of 77.5%, negative predictive value of 70.8% and overall accuracy of 73.7%. Laboratory evaluation showed higher frequencies of anemia, sodium, calcium and magnesium imbalance among cases. Suspected metabolic triggers were observed in 37.6% of cases versus 18.3% of controls ($p=0.005$). Treatment modification was required in 73.1% of cases compared with 9.7% of controls. **Conclusion**: Routine EEG is a clinically useful diagnostic support tool for adult epilepsy in Central India, especially when combined with structured clinical assessment and low-cost laboratory investigations. A multi-diagnostic pathway may improve diagnostic confidence and identify reversible contributors to seizure occurrence. However, a normal routine EEG cannot exclude epilepsy and all EEG findings require clinical correlation.

Keywords: *epilepsy; electroencephalography; EEG; case-control study; diagnostic accuracy; Central India; metabolic triggers; seizure evaluation.*

INTRODUCTION

Epilepsy is a chronic disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, along with neurobiological, cognitive, psychological and social consequences [1]. It is not simply a history of abnormal

movement or transient loss of consciousness. The diagnosis implies either recurrent unprovoked seizures, a high probability of recurrence after a single unprovoked event, or a recognized epilepsy syndrome. This distinction is clinically important because acute symptomatic seizures due to fever, metabolic derangement, intoxication, infection or brain injury require a different diagnostic and therapeutic approach from chronic epilepsy [1,13].

The global burden of epilepsy remains substantial. The World Health Organization estimates that around 50 million people live with epilepsy, with most cases occurring in low- and middle-income countries [2,3]. The condition is associated with physical injuries, stigma, educational disruption, unemployment, marital difficulties, reduced quality of life and premature mortality. Despite this burden, a large proportion of people with epilepsy can become seizure-free with appropriate diagnosis and treatment [2,3]. The central problem in many low-resource settings is therefore not only therapeutic efficacy but also timely recognition, reliable classification and sustained access to care.

India contributes a large share of the global epilepsy burden. Indian reviews have shown that epilepsy care is affected by delayed presentation, stigma, poor awareness, shortage of specialists, irregular availability of diagnostic services and treatment discontinuity [4,5]. The epilepsy treatment gap has been repeatedly highlighted in global and Indian literature, particularly among rural and socio-economically disadvantaged populations [6,14-18]. Central India includes rural, semi-urban and urban communities with diverse educational and socio-economic profiles. In such settings, the diagnostic pathway must be clinically sound but also feasible, affordable and accessible.

Electroencephalography is a non-invasive recording of cortical electrical activity and remains a central supportive investigation in epilepsy. EEG may detect interictal epileptiform discharges, focal or generalized abnormalities, background rhythm changes, generalized spike-wave discharges and other abnormalities that support seizure classification [10,11,19,20]. EEG can help distinguish focal and generalized epilepsy and may guide antiseizure medication selection. However, EEG is not a replacement for clinical diagnosis. A normal routine EEG does not exclude epilepsy, and an abnormal EEG must be interpreted in the context of seizure semiology, neurological examination and differential diagnosis [10,20,24,29].

Technical quality is an important determinant of EEG yield. Minimum recording standards and the 10-20 electrode placement system improve comparability and reduce technical error [11,19]. Overinterpretation of benign variants, artifacts or nonspecific slowing may lead to misdiagnosis and unnecessary treatment [22,23]. Conversely, a single short routine EEG may fail to capture interictal epileptiform abnormalities, causing false reassurance in patients with clinically convincing epilepsy [20,21]. Therefore, the clinical value of EEG lies in careful integration rather than isolated interpretation.

Additional investigations are also important because seizures may be precipitated or worsened by systemic or metabolic derangements. Electrolyte abnormalities, including sodium, calcium and magnesium disturbances, may lower seizure threshold and produce acute symptomatic seizures [25,26]. Complete blood count can identify anemia or infection, while urine analysis may reveal dehydration, glycosuria, ketonuria or urinary abnormalities that contribute to clinical interpretation. These tests do not diagnose epilepsy directly, but they identify reversible contributors and help avoid the misclassification of acute symptomatic seizures as chronic epilepsy.

The present study was undertaken to evaluate the role of routine EEG and selected additional diagnostic investigations in adult epileptic patients in Central India. The study compared clinically diagnosed epilepsy cases with non-epileptic controls presenting with comparable neurological complaints. The primary objective was to assess the frequency and pattern of EEG abnormalities and estimate diagnostic indices. Secondary objectives were to evaluate selected laboratory abnormalities, socio-demographic characteristics and management-related implications of an integrated diagnostic approach.

MATERIALS AND METHODS

Study design and setting: This was a hospital-based case-control study conducted in a tertiary care setting in Central India. The study focused on adult participants attending the Department of Physiology and related clinical or laboratory services of Index Medical College, Hospital and Research Centre, Indore, Madhya Pradesh. The design was selected because it allows direct comparison of EEG abnormalities and supplementary laboratory findings between clinically diagnosed epilepsy cases and non-epileptic controls.

Participants: A total of 186 adult participants were included, with 93 cases and 93 controls. Cases were patients aged 18-60 years with a clinical diagnosis of epilepsy who consented to EEG and study participation. Controls were adults aged 18-60 years who presented with neurological complaints such as headache, dizziness, syncope-like episode, sleep disturbance, anxiety-related symptoms or non-epileptic events under evaluation, but did not have a clinical diagnosis of epilepsy. Controls were selected to be comparable to cases by age and gender where possible.

Eligibility criteria: Cases were included when they were clinically diagnosed with epilepsy and were willing to provide informed consent. Controls were included when they had neurological complaints without clinical epilepsy. Participants with other major neurological disorders such as brain tumor or stroke likely to confound EEG interpretation, history of drug or alcohol abuse, or prior neurosurgery were excluded. The age range was restricted to 18-60 years to maintain adult clinical comparability.

Data collection: A structured case record form was used to document socio-demographic variables, clinical history, seizure characteristics, treatment details, EEG findings and laboratory parameters. Socio-demographic variables included age, gender, residence, education, occupation, socio-economic status and marital status. Clinical variables among cases included seizure type, seizure frequency, duration of epilepsy, age at first seizure, family history, antiseizure medication use and adherence.

EEG assessment: Routine EEG was recorded using standardized scalp electrode placement based on the international 10-20 system. EEG variables included normal or abnormal EEG status, background rhythm, interictal spikes, sharp waves, slow-wave activity, generalized discharges, focal discharges, background rhythm abnormality and seizure activity recorded during EEG. EEG interpretation was treated as abnormal when epileptiform discharges, focal or generalized abnormalities or other clinically relevant abnormalities were documented.

Supplementary investigations: Complete blood count, serum electrolyte panel and urine analysis were included as supplementary diagnostic investigations. CBC variables included hemoglobin, white blood cell count and platelet count. Electrolytes included serum sodium, potassium, calcium and magnesium. Urine analysis included specific gravity, protein, glucose and ketones. A suspected metabolic trigger was recorded when laboratory findings suggested a reversible systemic or metabolic contributor to seizure occurrence.

Statistical analysis: Data were summarized using frequency, percentage, mean and standard deviation. Cases and controls were compared using chi-square test or Fisher exact test for categorical variables and t-test for continuous variables. Diagnostic accuracy indices for EEG were calculated using clinical epilepsy status as the reference classification. Sensitivity, specificity, positive predictive value, negative predictive value and overall accuracy were reported. Odds ratios with 95% confidence intervals were calculated for selected factors associated with epilepsy status. A p value less than 0.05 was considered statistically significant. Diagnostic accuracy reporting was conceptually aligned with standard diagnostic study principles [30,31].

Ethical considerations: The thesis protocol included written informed consent. Before journal submission, the authors should insert the institutional ethics committee approval number, approval date and consent details exactly as recorded in the approved research file. The final publication version should conform to the Declaration of Helsinki and the target journal's ethics policy [32].

RESULTS

The study included 186 participants, equally divided into 93 cases and 93 controls. The mean age was comparable between cases and controls, with cases having a mean age of 38.00 ± 7.65 years and controls having a mean age of 38.37 ± 8.54 years. The age difference was not statistically significant (p=0.759), indicating that age distribution was balanced between the two groups. Gender distribution was identical, with 54 males and 39 females in each group. Rural residence was slightly more common among cases, but the difference was not statistically significant.

Among epilepsy cases, generalized tonic-clonic seizures accounted for 41 participants (44.1%), while focal aware or impaired awareness seizures were documented in 40 participants (43.0%). Absence, myoclonic or other seizure types were less frequent. Most cases experienced recurrent monthly episodes, with 52.7% reporting 3-5 seizures per month and 17.2% reporting more than five seizures per month. The duration of epilepsy exceeded 10 years in 37.6% of cases, suggesting that a considerable proportion had long-standing disease.

Table 1. Baseline socio-demographic characteristics of cases and controls

Variable	Case n (%) / Mean ± SD	Control n (%) / Mean ± SD	p value
Sample size	93 (50.0)	93 (50.0)	-
Age (years)	38.00 ± 7.65	38.37 ± 8.54	0.759
Male gender	54 (58.1)	54 (58.1)	-
Female gender	39 (41.9)	39 (41.9)	-
Rural residence	58 (62.4)	53 (57.0)	0.550
Urban residence	35 (37.6)	40 (43.0)	-
Middle socioeconomic status	49 (52.7)	48 (51.6)	-

Graduate and above education	36 (38.7)	29 (31.2)	-

Note. The two groups were comparable for major demographic variables, supporting the internal validity of case-control comparisons.

Table 2. Clinical profile of epilepsy cases

Clinical variable	Category	Frequency	Percentage
Seizure type	Generalized tonic-clonic	41	44.1
Seizure type	Focal aware/impaired awareness	40	43.0
Seizure type	Absence/myoclonic/other	5	5.4
Seizure type	Unclassified	7	7.5
Seizure frequency	1-2/month	28	30.1
Seizure frequency	3-5/month	49	52.7
Seizure frequency	>5/month	16	17.2
Duration of epilepsy	<5 years	33	35.5
Duration of epilepsy	5-10 years	25	26.9
Duration of epilepsy	>10 years	35	37.6
Family history of epilepsy	Yes	26	28.0
Current AED use	Yes	71	76.3

Note. AED: antiepileptic drug/antiseizure medication as recorded in the thesis dataset.

EEG abnormalities were substantially more frequent among epilepsy cases than controls. Normal EEG was found in 31 cases (33.3%) and 75 controls (80.6%), while abnormal EEG was documented in 62 cases (66.7%) and 18 controls (19.4%). The association between abnormal EEG and case status was statistically significant ($p<0.001$). Interictal spikes, sharp waves, generalized discharges and focal discharges were significantly more common in cases than controls. Seizure activity was recorded during EEG in 10 cases and in none of the controls.

Table 3. EEG findings and diagnostic accuracy indices

Parameter	Cases	Controls	Interpretation
Normal EEG	31 (33.3%)	75 (80.6%)	Normal routine EEG was common enough to avoid using EEG as an exclusion test.
Abnormal EEG	62 (66.7%)	18 (19.4%)	Significantly higher among cases ($p<0.001$).
Interictal spikes	30 (32.3%)	0 (0.0%)	Strong epileptiform marker in clinically diagnosed cases.
Sharp waves	28 (30.1%)	6 (6.5%)	More frequent among cases ($p<0.001$).
Generalized discharge	23 (24.7%)	0 (0.0%)	Supported generalized epileptiform tendency.
Focal discharge	21 (22.6%)	2 (2.2%)	Supported focal epileptiform involvement.
Sensitivity	66.7%	-	Moderate sensitivity.
Specificity	80.6%	-	Good supportive value for clinical epilepsy.
Positive predictive value	77.5%	-	Abnormal EEG frequently corresponded to case status.
Negative predictive value	70.8%	-	Normal EEG reduced but did not remove clinical suspicion.
Overall accuracy	73.7%	-	Useful as part of a multi-diagnostic pathway.

Note. Diagnostic indices were calculated using clinical epilepsy status as the reference classification.

Laboratory differences supported the clinical value of supplementary investigations. Cases had lower mean hemoglobin and higher mean white blood cell count than controls. Anemia was present in 41.9% of cases compared with 25.8% of controls ($p=0.030$). Sodium imbalance, calcium imbalance and magnesium imbalance were significantly more frequent

among cases. Suspected metabolic triggers were recorded in 35 cases (37.6%) and 17 controls (18.3%), showing a significant difference (p=0.005). These findings suggest that affordable laboratory tests can identify treatable contributors that may modify seizure threshold, treatment planning or differential diagnosis.

Table 4. Supplementary laboratory findings

Variable	Cases	Controls	p value
Hemoglobin (g/dL), mean ± SD	12.70 ± 1.31	13.20 ± 1.31	0.010
WBC count (10^3/uL), mean ± SD	8.14 ± 2.17	7.13 ± 1.53	<0.001
Platelet count (lakh/uL), mean ± SD	2.37 ± 0.58	2.57 ± 0.50	0.015
Anemia	39 (41.9%)	24 (25.8%)	0.030
Sodium imbalance	29 (31.2%)	14 (15.1%)	0.015
Calcium imbalance	12 (12.9%)	3 (3.2%)	0.031
Magnesium imbalance	15 (16.1%)	5 (5.4%)	0.033
Suspected metabolic trigger	35 (37.6%)	17 (18.3%)	0.005

Note. Laboratory abnormalities were interpreted as supportive and not as stand-alone diagnostic criteria for epilepsy.

In the association analysis, abnormal EEG showed the strongest relationship with epilepsy status, with an odds ratio of 8.33 (95% CI: 4.26-16.31; p<0.001). Suspected metabolic trigger, anemia, sodium imbalance, calcium imbalance and magnesium imbalance were also significantly associated with epilepsy status in unadjusted analysis. Rural residence was not statistically significant. Treatment modification was required in 68 cases (73.1%) compared with 9 controls (9.7%), indicating that integrated clinical, EEG and laboratory assessment influenced management planning. Among cases followed for outcome, 24.7% achieved a 50% or greater reduction in seizure frequency, while 37.6% had a 25-49% reduction.

Table 5. Predictors and management-related findings

Factor/outcome	Value	95% CI / Group comparison	p value
Abnormal EEG	OR 8.33	4.26-16.31	<0.001
Suspected metabolic trigger	OR 2.70	1.38-5.29	0.005
Anemia	OR 2.08	1.12-3.86	0.030
Sodium imbalance	OR 2.56	1.25-5.24	0.015
Calcium imbalance	OR 4.44	1.21-16.31	0.031
Magnesium imbalance	OR 3.38	1.18-9.74	0.033
Treatment modification required	73.1% cases vs 9.7% controls	77/186 overall	<0.001
>=50% seizure reduction at follow-up	23 cases	24.7% of cases	-

Note. OR: odds ratio; CI: confidence interval.

DISCUSSION

This case-control study demonstrates that routine EEG provides meaningful diagnostic support in adult epilepsy evaluation in Central India. Abnormal EEG was detected in two-thirds of clinically diagnosed epilepsy cases and in less than one-fifth of non-epileptic controls. The magnitude of difference and the odds ratio of 8.33 indicate a strong association between abnormal EEG and clinical epilepsy status. This finding is consistent with previous literature describing EEG as a key supportive tool for epilepsy diagnosis and classification [10,20,24,29]. It also agrees with guidance emphasizing EEG as most useful when the history suggests an epileptic seizure and the clinician needs support for seizure type or syndrome classification [10,12].

The diagnostic profile observed in the present study is clinically realistic. EEG sensitivity was 66.7%, while specificity was 80.6%. This supports the view that routine EEG has useful but incomplete diagnostic power. A normal EEG was observed in one-third of cases, confirming that a single routine EEG cannot exclude epilepsy. Previous studies and reviews have similarly noted that interictal EEG yield depends on timing, sleep state, repeated recordings, activation procedures, duration of recording and epilepsy type [20,21,24,29]. Therefore, normal EEG findings should prompt clinical review, consideration of repeat EEG or sleep-deprived EEG where indicated, and continued attention to seizure semiology rather than immediate dismissal of epilepsy.

The pattern of abnormalities also supports clinical usefulness. Interictal spikes, sharp waves, generalized discharges and focal discharges were more common among cases. These abnormalities have established relevance for epilepsy diagnosis

and classification, particularly when they correspond to clinical seizure features [7,8,20,22]. Focal discharges may suggest focal epilepsy or a focal epileptogenic network, while generalized discharges may support a generalized epilepsy pattern. This classification value is important because antiseizure medication selection and counselling differ according to seizure type and epilepsy type [7,8,27].

The presence of abnormal EEG findings in some controls highlights a major interpretive issue. Controls in this study were not completely healthy individuals; they presented with neurological complaints such as headache, dizziness or syncope-like episodes. Nonspecific slowing or background abnormalities can occur in non-epileptic contexts and may be misinterpreted if clinical correlation is weak. Literature on EEG overinterpretation warns that false diagnosis of epilepsy can occur when benign variants or nonspecific findings are reported as epileptiform abnormalities [22,23]. Thus, the present findings support both the value and caution required in routine EEG interpretation.

Supplementary laboratory testing added clinically useful information. Cases had significantly higher frequencies of anemia, sodium imbalance, calcium imbalance and magnesium imbalance. Electrolyte disturbances are recognized contributors to acute symptomatic seizures and may lower seizure threshold in vulnerable individuals [25,26]. Hyponatremia, hypernatremia, hypocalcemia and hypomagnesemia can alter neuronal excitability and trigger seizures. The higher proportion of suspected metabolic triggers among cases suggests that routine laboratory screening may identify reversible contributors and guide immediate clinical management. This finding is particularly relevant in resource-limited settings where advanced testing may not be immediately available.

The multi-diagnostic approach appears more clinically valuable than EEG alone. EEG identifies neurophysiological abnormalities, while CBC, electrolyte panel and urine analysis help detect systemic or metabolic contributors. In the present study, treatment modification was required in nearly three-fourths of cases. This suggests that integrated evaluation influenced practical management planning, although causal impact cannot be proven from the case-control design. The finding aligns with the clinical principle that epilepsy management requires accurate diagnosis, classification, comorbidity assessment and patient-centered treatment planning [10,12,27].

Socio-demographic variables were largely comparable between cases and controls, and most were not statistically significant predictors in the analysis. This may reflect the matched case-control design rather than absence of social influence. Global and Indian studies consistently show that rural residence, lower income, poor education, stigma and limited specialist access contribute to delayed epilepsy care and treatment gaps [4-6,15,18]. Therefore, socio-demographic factors remain clinically and public-health relevant even when not statistically significant in a balanced hospital-based dataset.

The present study has several strengths. It used a case-control design, equal group sizes, clinically relevant controls and a practical diagnostic protocol suitable for a tertiary care setting in Central India. It also evaluated EEG alongside low-cost laboratory investigations rather than treating EEG as an isolated test. However, important limitations must be acknowledged. The study was hospital-based and may not represent community epilepsy patterns. Routine EEG was used, and prolonged video-EEG or repeated EEG was not mandatory. Some laboratory abnormalities may have been associated with acute illness rather than epilepsy itself. Diagnostic accuracy was calculated using clinical case status as the reference classification, which is pragmatic but not equivalent to long-term electroclinical confirmation. Finally, all results must be verified against original patient data before publication.

Overall, the findings support a structured diagnostic pathway for epilepsy evaluation in resource-constrained clinical settings. Routine EEG should be available, technically standardized and interpreted by trained personnel. Basic laboratory screening should be incorporated to identify treatable metabolic or systemic contributors. Patients and families should be counselled that EEG supports diagnosis but does not independently confirm or exclude epilepsy. Such an approach may reduce misdiagnosis, improve treatment selection and support more rational use of specialist referral pathways.

Clinical Implications

The clinical implication of the present work is that EEG should be positioned as a decision-support investigation within a larger epilepsy evaluation pathway. In many resource-limited hospitals, patients are first assessed by general physicians, emergency staff or non-specialist clinicians before referral to neurology services. A standardized approach that begins with careful seizure history, eyewitness description, clinical examination, routine EEG and basic laboratory evaluation can reduce both underdiagnosis and overdiagnosis. In the present study, the high specificity of EEG suggests that abnormal epileptiform findings provide strong supportive evidence when clinical features are compatible with epilepsy.

The moderate sensitivity has equal practical importance. One-third of clinically diagnosed cases had normal routine EEG. If normal EEG is misunderstood as proof of absence of epilepsy, patients may remain untreated or inadequately counselled. Therefore, reports should be accompanied by clear interpretation, and clinicians should be trained to communicate that EEG is supportive, not absolute. Where clinical suspicion remains high, repeat EEG, sleep-deprived EEG, prolonged recording or video-EEG should be considered according to availability and affordability.

The laboratory findings emphasize the need to search for reversible contributors. Electrolyte imbalance, anemia or systemic illness may worsen seizure control or mimic epilepsy. Screening for sodium, calcium and magnesium disturbances can guide urgent correction and may prevent recurrent acute symptomatic seizures. This is particularly relevant in Central India, where patients may present after dehydration, febrile illness, irregular food intake, medication non-adherence or delayed consultation. Incorporating laboratory assessment into the EEG pathway can therefore make care more clinically beneficial and not merely diagnostic.

Patient counselling is another major implication. The results suggest that many cases required treatment modification, and a meaningful subgroup reported improvement during follow-up. Counselling should include adherence to antiseizure medication, avoidance of sleep deprivation, reduction of alcohol or substance exposure, safety during bathing, cooking and driving, and the importance of regular follow-up. Family members should be educated to record seizure episodes, describe events accurately and seek medical care promptly. Such counselling may reduce injuries, stigma and treatment discontinuation.

Strengths and Limitations

The main strength of this study is the use of a case-control design with equal numbers of epilepsy cases and non-epileptic controls. The control group included participants with neurological complaints rather than only healthy volunteers, which makes the comparison more clinically realistic. This design helps evaluate whether EEG abnormalities truly discriminate epilepsy from common neurological presentations such as dizziness, headache, syncope-like episodes and non-epileptic events. The study also included demographic, clinical, EEG and laboratory variables, allowing a more integrated interpretation than an EEG-only analysis.

Another strength is the focus on a feasible diagnostic package. Advanced tools such as video-EEG, high-density EEG, epilepsy-protocol MRI and genetic testing are valuable but may not be accessible to many patients in low-resource regions. Routine EEG, CBC, electrolyte panel and urine analysis are more widely available and can be implemented in tertiary and district-level systems with appropriate training. This makes the findings relevant for practical clinical pathways in Central India and similar settings.

The study has limitations. It was hospital-based and therefore may not represent the community prevalence or untreated epilepsy burden. Clinical diagnosis was used as the reference classification for diagnostic accuracy, which reflects real-world practice but may not equal long-term expert electroclinical confirmation. Routine EEG was used, so abnormalities that appear only during sleep, prolonged recording or ictal events may have been missed. The study did not include advanced neuroimaging data as a core variable. Some laboratory abnormalities may be contributors, consequences or coincidental findings rather than direct causes of seizures. Finally, outcome assessment was short-term and not designed to establish long-term seizure remission.

Before publication, the dataset must be validated against original clinical records. Patient consent, ethics approval details, missing data handling and statistical outputs should be checked carefully. A Scopus-indexed journal will usually require exact ethics committee approval number, date of approval, informed consent statement, data availability statement and conflict-of-interest declaration. These elements should be completed by the investigators and not inferred from the thesis text.

Future Research

Future research should use a prospective diagnostic accuracy design with longer follow-up and stronger reference standards. Repeated EEG, sleep-deprived EEG, ambulatory EEG or video-EEG can be added where feasible to determine incremental diagnostic yield. Future studies should also evaluate the effect of timing of EEG after seizure, sleep state during recording, antiseizure medication status and activation procedures on detection of epileptiform abnormalities. These variables can help optimize routine EEG scheduling in resource-limited services.

Further work is also needed to understand the interaction between laboratory abnormalities and seizure recurrence. Prospective correction of sodium, calcium, magnesium or anemia-related abnormalities followed by seizure outcome assessment may clarify whether these findings act as direct triggers, risk modifiers or markers of general health. Larger multicentric studies from Central India can improve generalizability and support regional epilepsy care planning. Research on cost-effectiveness may also help policymakers decide whether wider access to EEG and basic laboratory screening reduces referral delay, unnecessary medication and preventable morbidity.

Emerging technologies such as portable EEG, automated artifact detection and artificial intelligence-assisted EEG interpretation may eventually improve diagnostic access. However, these tools should be validated locally against standard clinical EEG interpretation and outcome data. Technology should support, not replace, clinical judgement. The findings of the present study provide a practical baseline for such future service innovations.

CONCLUSION

Routine EEG is a clinically useful diagnostic support tool in adult epilepsy evaluation in Central India. In this case-control study, abnormal EEG findings were significantly more common among epilepsy cases than non-epileptic controls, and EEG demonstrated good specificity with moderate sensitivity. The findings confirm that abnormal EEG provides strong supportive evidence for epilepsy when correlated with clinical history, but a normal routine EEG cannot exclude the disorder. Supplementary investigations, especially CBC and electrolyte analysis, identified anemia and metabolic disturbances that may contribute to seizure occurrence or influence management. A practical multi-diagnostic pathway combining clinical assessment, standardized EEG and low-cost laboratory testing may improve diagnostic confidence and management planning in resource-limited settings.

Recommendations

- Routine EEG should be used as a supportive investigation in suspected epilepsy and interpreted with clinical seizure semiology.
- EEG laboratories should follow standardized electrode placement, recording duration, activation and reporting protocols.
- CBC, electrolyte panel and urine analysis should be included in initial seizure evaluation, especially in resource-limited settings.
- A normal routine EEG should not be used to exclude epilepsy when the clinical history is convincing.
- Patients and families should receive counselling on medication adherence, sleep hygiene, seizure safety and follow-up.
- Future studies should use prospective follow-up, repeated EEG or video-EEG where feasible, and verified real-world outcome data.

Declarations

Ethics approval and consent to participate: Written informed consent was described in the thesis protocol. The final manuscript must include the institutional ethics committee approval number, date and exact approval authority before journal submission.

Consent for publication: Not applicable for anonymized aggregate data; to be confirmed by authors according to journal policy.

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