Corellation Between Gaba/Glutamate Ratio Magnetic Resonance Spectroscopy and Electroencephalogram in Pediatric Epilepsy

Maryama Aisyah Pribadi[1], Rachmi Fauziah Rahayu[1], Fadhilah Tia Nur[2], Sulistyani Kusumaningrum[1], Evi Rokhayati[2]

[1] Department of Radiology, Faculty of Medicine, Sebelas Maret University/Dr. Moewardi General Hospital Surakarta

[2] Department of Child Health, Faculty of Medicine, Sebelas Maret University/Dr. Moewardi General Hospital, Surakarta

HOW TO CITE:

Maryama Aisyah Pribadi, Rachmi Fauziah Rahayu, Fadhilah Tia Nur, Sulistyani Kusumaningrum, Evi Rokhayati (2026). Corellation Between Gaba/Glutamate Ratio Magnetic Resonance Spectroscopy and Electroencephalogram in Pediatric Epilepsy

International Journal of Special Education, 41(21s), 310 - 319

ABSTRACT

To investigate the association between the gamma-aminobutyric acid (GABA)/glutamate ratio measured by magnetic resonance spectroscopy (MRS) and electroencephalographic (EEG) findings in children with epilepsy. This prospective cross-sectional study included 33 children with clinically diagnosed epilepsy at Dr. Moewardi Regional General Hospital, Surakarta, Indonesia, from July 2025 to January 2026. Participants underwent routine EEG, structural magnetic resonance imaging (MRI), and single-voxel MRS. The GABA/glutamate ratio was categorized using a reference range of 0.63-0.67. Associations among modalities were examined using contingency coefficients, and Cochran Q testing compared proportions of abnormal findings. MRI interobserver agreement was assessed using Cohen kappa. All participants had an abnormal GABA/glutamate ratio; 24 (72.7%) had a ratio <0.63 and 9 (27.3%) had a ratio >0.67. MRI abnormalities were identified in 28 participants (84.8%) and EEG abnormalities in 25 (75.8%). Abnormal finding proportions differed across modalities ($p=0.004$). The GABA/glutamate ratio was not significantly associated with EEG ($p=0.061$) or MRI findings ($p=0.200$). MRI and EEG were significantly associated (coefficient=0.612, $p=0.001$). MRI interobserver agreement was very high (kappa=0.901, $p<0.001$). The GABA/glutamate ratio was not significantly associated with EEG or structural MRI categories. The three modalities captured different dimensions of epilepsy and should be interpreted as complementary assessments.

Keywords: Epilepsy, Child, gamma-Aminobutyric Acid, Glutamic Acid; Magnetic Resonance Spectroscopy; Electroencephalography

1. Introduction

Epilepsy is a common chronic neurologic disorder in childhood and can have long-term consequences for development and quality of life. Reported incidence ranges from approximately 41 to 187 per 100 000

children per year, while the prevalence of active epilepsy is estimated at 5-10 per 1000 children (Beghi, 2020; Urbańska et al., 2024) at Dr. Moewardi Regional General Hospital, Surakarta, 399 pediatric patients with epilepsy accounted for 2966 outpatient visits in 2024, while 90 pediatric inpatients accounted for 131 admissions, indicating a substantial local clinical burden (RSUD Dr. Moewardi, 2024). Accurate characterization of pediatric epilepsy therefore remains important for diagnostic evaluation and treatment planning.

Electroencephalography (EEG) and magnetic resonance imaging (MRI) provide complementary information in the evaluation of epilepsy. EEG identifies abnormal electrical activity, including focal or generalized epileptiform patterns, but a routine recording may be normal despite a clinical diagnosis of epilepsy because epileptiform discharges can be intermittent or arise from regions that are difficult to detect with scalp electrodes (Baldini et al., 2020). Structural MRI can identify epileptogenic abnormalities such as hippocampal sclerosis, focal cortical dysplasia, vascular abnormalities, and developmental malformations. However, a proportion of patients with epilepsy have no visible lesion even when an epilepsy-focused MRI protocol is used (Jamalipour et al., 2024; Youssf et al., 2021). These limitations motivate investigation of additional markers that reflect mechanisms not directly captured by electrophysiologic or structural examinations.

A central neurochemical feature of epileptogenesis is an imbalance between excitatory glutamatergic and inhibitory gamma-aminobutyric acid (GABA) neurotransmission. Altered glutamate receptor signaling, including N-methyl-D-aspartate and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid pathways, may increase neuronal excitability, while abnormalities in GABA receptors and transporters may weaken inhibitory control (Chen et al., 2023; Bryson et al., 2023). Studies of human epilepsy have therefore examined GABA and glutamate concentrations as potential markers of the excitation-inhibition balance (Sarlo & Holton, 2021). Magnetic resonance spectroscopy (MRS) offers a noninvasive method for characterizing brain metabolites and can provide information about neurochemical abnormalities that is not available from conventional structural MRI.

Previous pediatric epilepsy studies have primarily emphasized structural MRI findings and lesion detection (Raafat et al., 2024). Other work has investigated relationships among MRS, MRI, and EEG, but commonly using metabolite ratios such as N-acetylaspartate/creatine in temporal lobe epilepsy rather than specifically examining the GABA/glutamate balance in children (Jamalipour et al., 2024). Thus, evidence directly relating the GABA/glutamate ratio on MRS to EEG findings in pediatric epilepsy remains limited. This study addresses that gap by integrating neurochemical, structural, and electrophysiologic assessments in the same pediatric sample. We aimed to determine whether the GABA/glutamate ratio measured by MRS was associated with EEG findings in children with epilepsy and, secondarily, to describe relationships among MRS, MRI, and EEG abnormalities.

2. Methodology

Study Design and Setting

A prospective cross-sectional study was conducted at the Radiology Installation, EEG service, and Medical Records Department of Dr. Moewardi Regional

General Hospital, Surakarta, Indonesia, from July 2025 through January 2026. The study evaluated participants at a single period of clinical assessment and was designed to examine associations between the GABA/glutamate ratio on MRS and EEG findings rather than to establish temporal or causal relationships.

Participants

The study population comprised pediatric patients with epilepsy treated at Dr. Moewardi Regional General Hospital. Eligible participants had a diagnosis of epilepsy established by a pediatric specialist, were aged from 1 month to 17 years 11 months 29 days, agreed to undergo EEG, MRI, and MRS, and had parental or guardian informed consent. Exclusion criteria were a history of brain tumor or a contraindication to MRI, including cochlear implants, ferromagnetic metallic implants, cardiac pacemakers, or aneurysm clips. Participants meeting the criteria during the recruitment period were included using total purposive non-random sampling. The calculated minimum sample size was 30 participants based on a minimum meaningful correlation of 0.49, a two-sided alpha level of 0.05, and statistical power corresponding to Z beta=0.842. A total of 33 participants were analyzed.

EEG Assessment

Routine EEG reports were interpreted by a pediatric neurologist with more than 5 years of professional experience. EEG findings were categorized as focal when epileptiform activity involved one hemisphere, generalized when epileptiform activity involved both hemispheres, or normal when no epileptiform abnormality was identified in the routine recording. For the comparison of abnormal findings among modalities, focal and generalized EEG findings were combined as abnormal.

MRI Assessment

Structural MRI was performed using a 3.0-T Signa Pioneer system (General Electric) in 31 participants and a 1.5-T system in 2 participants. The imaging protocol included at least the sequences recommended for epilepsy-focused HARNESS-MRI: high-resolution three-dimensional T1-weighted imaging, high-resolution three-dimensional fluid-attenuated inversion recovery, and high in-plane resolution two-dimensional coronal T2-weighted imaging; double inversion recovery was also included in the source protocol. Children younger than 7 years received anesthesia support when required to reduce motion artifact. MRI examinations were independently interpreted in a blinded manner by two radiologists with more than 5 years of experience. Hippocampal measurements were performed in all participants. Structural findings were categorized as nonlesional, unilateral, or bilateral. Lesion types were additionally grouped as hippocampal sclerosis/atrophy, vascular/ischemic, developmental malformation, cyst/extra-axial cerebrospinal fluid lesion, infection/inflammation, or no lesion.

MRS Assessment

MRS was acquired using a single-voxel point-resolved spectroscopy technique. The typical voxel size was approximately 10 x 20 x 10 mm. When structural MRI did not identify a lesion, MRS sampling was performed in the temporal and frontal lobes. The principal MRS variable was the GABA/glutamate ratio. According to the reference range used in the source study, ratios from 0.63 to 0.67 were considered normal; values <0.63 or >0.67 were classified as abnormal. The ratio was also analyzed as two abnormal

categories, <0.63 and >0.67, for associations with MRI and EEG patterns.

Statistical Analysis

Participant characteristics and imaging findings were summarized using frequencies and percentages for categorical variables and mean, standard deviation, and range for continuous variables. Agreement between the two MRI readers was assessed using Cohen kappa. Associations among categorized MRS, MRI, and EEG findings were assessed using contingency coefficient analysis. Cochran Q testing was used to compare the within-participant proportions of abnormal findings across MRS, MRI, and EEG. Statistical significance was defined as $p<0.05$. Analyses were performed using IBM SPSS Statistics version 22.0.

Ethics Statement

The study protocol underwent ethical review and institutional research permission at Dr. Moewardi Regional General Hospital. Written informed consent was obtained from the parents or legal guardians of participants before study procedures. Participant identities were protected in the study records and reporting. The ethics approval number was not included in the source manuscript and should be inserted here before submission: 1.600/VII/HREC/2025.

3. Results

Participant Characteristics

Thirty-three children with epilepsy met the eligibility criteria. Twenty-two participants (66.7%) were male and 11 (33.3%) were female. Mean age was 7.32 years (standard deviation, 5.79; range, 0.5-17.5 years). Twelve participants (36.4%) were aged >10-18 years, 8 (24.2%) were infants aged 0-1 year, 7 (21.2%) were aged >1-5 years, and 6 (18.2%) were aged >5-10 years. Mean duration of illness was 8.21 months (standard deviation, 15.22; range, 1-85 months); 22 participants (66.7%) had been ill for 6 months or less. Participant characteristics and the primary examination findings are summarized in Table 1.

Table 1. **Characteristics of the 33 pediatric participants with epilepsy**

Variable	Value
Sex	
Male	22 (66.7)
Female	11 (33.3)
Age, y	7.32 +/- 5.79 (0.5-17.5)
0-1	8 (24.2)
>1-5	7 (21.2)
>5-10	6 (18.2)
>10-18	12 (36.4)
Illness duration, mo	8.21 +/- 15.22 (1-85)
<=6	22 (66.7)
>6	11 (33.3)
GABA/glutamate ratio	0.58 +/- 0.12 (0.40-0.85)
<0.63	24 (72.7)
>0.67	9 (27.3)
MRI findings	

Nonlesional	5 (15.2)
Unilateral lesion	15 (45.5)
Bilateral lesion	13 (39.4)
EEG findings	
Normal	8 (24.2)
Focal	16 (48.5)
Generalized	9 (27.3)

Values are presented as n (%) or mean +/- standard deviation (range). GABA, gamma-aminobutyric acid; MRI, magnetic resonance imaging; EEG, electroencephalography.

MRS, MRI, and EEG Findings

The mean GABA/glutamate ratio was 0.58 (standard deviation, 0.12; observed range, 0.40-0.85). None of the participants had a ratio within the prespecified normal interval of 0.63-0.67. Twenty-four participants (72.7%) had a ratio <0.63 and 9 (27.3%) had a ratio >0.67. Structural MRI identified a lesion in 28 participants (84.8%); 15 (45.5%) had unilateral lesions and 13 (39.4%) had bilateral lesions, whereas 5 (15.2%) were classified as nonlesional. Hippocampal sclerosis or atrophy was the most frequent lesion group (10/33, 30.3%), followed by vascular or ischemic lesions (7/33, 21.2%), developmental malformations (5/33, 15.2%), cystic or extra-axial cerebrospinal fluid lesions (4/33, 12.1%), and infection or inflammation (2/33, 6.0%) (Table 2). Routine EEG was abnormal in 25 participants (75.8%), including 16 (48.5%) with focal findings and 9 (27.3%) with generalized findings. Eight participants (24.2%) had a normal routine EEG.

Table 2. **Distribution of structural MRI lesion groups**

Lesion group	n	%
Hippocampal sclerosis/atrophy	10	30.3
Vascular/ischemic	7	21.2
Developmental malformation	5	15.2
No lesion	5	15.2
Cyst/extra-axial cerebrospinal fluid lesion	4	12.1
Infection/inflammation	2	6.0
Total	33	100.0

MRI, magnetic resonance imaging.

Comparison of Abnormal Findings Across Modalities

Because every participant had clinically diagnosed epilepsy, the analyses compared proportions of abnormal test findings rather than formal diagnostic accuracy. Abnormal findings were present in 33 of 33 participants (100%) on MRS, 28 of 33 (84.8%) on structural MRI, and 25 of 33 (75.8%) on EEG. Cochran Q testing showed that the proportions differed significantly among the three modalities (p=0.004) (Table 3). These results indicate that, in this selected epilepsy sample, neurochemical, structural, and electrophysiologic abnormalities were not identified with the same frequency.

Table 3. **Comparison of abnormal findings across MRS, MRI, and EEG (n=33)**

Examination	Normal, n (%)	Abnormal, n (%)
MRS (GABA/glutamate ratio)	0 (0.0)	33 (100.0)
MRI	5 (15.2)	28 (84.8)
EEG	8 (24.2)	25 (75.8)

Cochran Q test: p=0.004. MRS, magnetic resonance spectroscopy; GABA, gamma-aminobutyric acid; MRI, magnetic resonance imaging; EEG, electroencephalography.

Associations Among MRS, MRI, and EEG

The GABA/glutamate ratio category was not significantly associated with structural MRI category (contingency coefficient=0.298, p=0.200). Among participants with a ratio <0.63, 12 of 24 (50.0%) had unilateral lesions, 10 (41.7%) had bilateral lesions, and 2 (8.3%) were nonlesional. Among those with a ratio >0.67, 3 of 9 (33.3%) were represented in each MRI category. The association between the GABA/glutamate ratio and EEG category also did not reach statistical significance (contingency coefficient=0.380, p=0.061). Among participants with a ratio <0.63, 11 (45.8%) had focal EEG findings, 9 (37.5%) had generalized findings, and 4 (16.7%) had a normal EEG. Among participants with a ratio >0.67, 5 (55.6%) had focal findings and 4 (44.4%) had a normal EEG; no generalized EEG findings occurred in this group.

Table 4. **Associations among MRS, MRI, and EEG categories**

Comparison	Category	Group 1, n (%)	Group 2, n (%)	Coefficient	p-value
MRS vs MRI	Nonlesional	3 (33.3)	2 (8.3)	0.298	0.200
	Unilateral	3 (33.3)	12 (50.0)		
	Bilateral	3 (33.3)	10 (41.7)		
MRS vs EEG	Normal	4 (44.4)	4 (16.7)	0.380	0.061
	Focal	5 (55.6)	11 (45.8)		
	Generalized	0 (0.0)	9 (37.5)		
MRI vs EEG	EEG normal	4 (80.0)	1 (6.7) / 3 (23.1)	0.612	0.001
	EEG focal	1 (20.0)	12 (80.0) / 3 (23.1)		
	EEG generalized	0 (0.0)	2 (13.3) / 7 (53.8)		

For MRS vs MRI and MRS vs EEG, Group 1=>0.67 and Group 2=<0.63. For MRI vs EEG, Group 1=nonlesional MRI and Group 2=unilateral/bilateral MRI, respectively. Coefficients are contingency coefficients. MRS, magnetic resonance spectroscopy; MRI, magnetic resonance imaging; EEG, electroencephalography.

MRI-EEG Association and Interobserver Reliability

Structural MRI category was significantly associated with EEG category (contingency coefficient=0.612, p=0.001). Four of 5 participants (80.0%) with nonlesional MRI had a normal EEG. In contrast, 12 of 15 participants (80.0%) with unilateral MRI lesions had focal EEG findings, while 7 of 13 participants (53.8%) with bilateral lesions had generalized EEG findings (Table 4). Agreement between the two MRI readers was very high, with Cohen kappa=0.901 (p<0.001). The readers agreed on all nonlesional cases, and agreement remained high for unilateral and bilateral classifications.

4. Discussion

This study examined the relationship between the GABA/glutamate ratio on MRS and routine EEG findings in children with epilepsy while also characterizing structural

MRI findings in the same participants. Three findings are central. First, all participants had a GABA/glutamate ratio outside the prespecified reference interval, whereas structural MRI and routine EEG were abnormal in 84.8% and 75.8%, respectively. Second, the GABA/glutamate ratio category was not significantly associated with either EEG or MRI category. Third, structural MRI and EEG findings were strongly associated, with nonlesional MRI tending to accompany normal EEG, unilateral lesions tending to accompany focal EEG abnormalities, and bilateral lesions tending to accompany generalized EEG abnormalities. Together, these findings suggest that the three examinations describe partly distinct aspects of the epileptic condition.

The predominance of reduced GABA/glutamate ratios is biologically plausible in the context of epilepsy. GABA is the principal inhibitory neurotransmitter in the central nervous system, whereas glutamate is the major excitatory neurotransmitter. Disruption of the balance between these systems can facilitate neuronal hyperexcitability and epileptiform activity (Chen et al., 2023; Bryson et al., 2023; Sarlo & Holton, 2021). In the present study, 72.7% of participants had a ratio <0.63, consistent with a relative shift toward reduced inhibition or increased excitation under the reference framework used in the study. However, the finding that 27.3% had ratios >0.67 also underscores that an abnormal ratio is not unidirectional in every patient. Because antiepileptic drug exposure was not systematically controlled and MRS sampling used a limited number of voxels, the observed ratio should be interpreted as a regional neurochemical measure rather than a comprehensive indicator of whole-brain neurotransmission.

The lack of a statistically significant association between MRS and EEG ($p=0.061$) may reflect fundamental differences in what the two modalities measure. MRS provides a neurochemical snapshot within selected brain regions, whereas scalp EEG records electrical activity during a limited observation period. Routine EEG can miss interictal epileptiform discharges when abnormalities are intermittent, deep, spatially restricted, or absent during the recording period (Baldini et al., 2020; Wander et al., 2024). In our sample, 8 children had a normal routine EEG despite a clinical epilepsy diagnosis, while all had an abnormal GABA/glutamate ratio by the study definition. This pattern supports the view that a normal routine EEG does not necessarily imply an absence of neurobiologic abnormality. Nevertheless, the cross-sectional design does not establish that MRS abnormalities precede EEG abnormalities, and the observed difference should not be interpreted as proof that MRS is diagnostically superior.

Similarly, the GABA/glutamate ratio was not significantly associated with structural MRI category ($p=0.200$). Structural MRI depicts macroscopic anatomy, whereas MRS characterizes local biochemical composition. A visible lesion may therefore coexist with a variable neurochemical profile, while neurochemical abnormalities may occur in regions without a conspicuous structural lesion. Five participants in this study had nonlesional MRI. Studies of epilepsy imaging have emphasized that lesion detection depends on lesion size, location, imaging protocol, scanner characteristics, and reader expertise (Bernasconi et al., 2019). The use of HARNESS-oriented MRI sequences and experienced readers in this study likely strengthened structural assessment, as also suggested by the high interobserver kappa.

Even so, a nonlesional MRI classification should not be equated with absence of epileptogenic pathology.

Hippocampal sclerosis or atrophy was the most frequent structural lesion category, accounting for 30.3% of participants. Hippocampal sclerosis is closely associated with mesial temporal lobe epilepsy and is characterized by neuronal loss, gliosis, and hippocampal volume reduction, often accompanied by T2 or fluid-attenuated inversion recovery signal abnormalities (Lee et al., 2021; Deleu et al., 2026). Focal EEG abnormalities were also the most common EEG pattern in this sample, and the temporal region was frequently identified in the source clinical interpretation. Prior pediatric focal epilepsy data similarly describe temporal involvement as a common localization (Gauci et al., 2025). These parallel structural and electrophysiologic patterns provide a clinical context for the significant MRI-EEG association observed here.

The strong association between MRI and EEG was the most statistically robust cross-modal relationship. Most participants with unilateral structural lesions had focal EEG findings, while generalized EEG findings were more common among participants with bilateral lesions. This pattern is consistent with the concept that a focal structural abnormality can provide an anatomic substrate for localized epileptiform activity, whereas more extensive bilateral abnormalities may be associated with broader electrophysiologic involvement. Previous work combining neuroimaging and EEG in temporal lobe epilepsy has likewise reported correspondence between structural abnormalities and electrophysiologic localization (Rentería-Palomo et al., 2021). However, the association observed in the present study is categorical and does not demonstrate that a specific MRI lesion caused the recorded EEG pattern.

Several limitations should be considered. The sample was small and was drawn from a single tertiary hospital, limiting generalizability. The study included pediatric epilepsy of heterogeneous etiologies, syndromes, ages, and illness durations rather than a narrowly defined epilepsy subtype. The cross-sectional design precludes temporal or causal inference. EEG was a routine recording rather than prolonged ambulatory or video EEG, creating the possibility of missed interictal epileptiform activity (Wander et al., 2024). MRS used single-voxel acquisition in selected regions, so the GABA/glutamate ratio could not represent neurochemical variation across the entire epileptic network. In addition, antiepileptic medication exposure was not systematically controlled even though some agents can affect GABAergic or glutamatergic pathways. Finally, because all participants had clinically diagnosed epilepsy and no non-epilepsy comparison group was included, the study cannot estimate specificity or overall diagnostic accuracy of MRS, MRI, or EEG.

In conclusion, the GABA/glutamate ratio measured by MRS was not significantly associated with EEG or structural MRI categories in this sample of children with epilepsy. The different frequencies and patterns of abnormalities across MRS, MRI, and EEG indicate that neurochemical, structural, and electrophysiologic assessments provide non-equivalent information. The significant MRI-EEG association supports concordance between structural and electrophysiologic abnormalities in a subset of patients, while abnormal MRS findings in patients with normal routine EEG or nonlesional MRI warrant further investigation. Larger studies

using well-defined epilepsy subgroups, standardized medication assessment, multivoxel or advanced MRS protocols, and prolonged EEG are needed to clarify the clinical role of GABA/glutamate measurements.

5. Conclusion

This study found that all pediatric epilepsy patients had an abnormal GABA/glutamate ratio, with the majority showing ratios below the reference range. However, the GABA/glutamate ratio was not significantly associated with EEG or structural MRI findings. In contrast, EEG and MRI demonstrated a significant association, with high interobserver agreement for MRI interpretation. These findings indicate that GABA/glutamate MRS, EEG, and structural MRI capture different aspects of pediatric epilepsy and should therefore be considered complementary diagnostic modalities rather than interchangeable assessments.

6. References

Baldini, S., Pittau, F., Birot, G., Rochas, V., Tomescu, M. I., Vulliémoz, S., & Seeck, M. (2020). Detection of epileptic activity in presumably normal EEG. *Brain communications*, *2*(2), fcaa104. doi:10.1093/braincomms/fcaa104.

Beghi, E. (2020). The epidemiology of epilepsy. *Neuroepidemiology*, *54*(2), 185-191. doi:10.1159/000503831.

Bernasconi, A., Cendes, F., Theodore, W. H., Gill, R. S., Koepp, M. J., Hogan, R. E., ... & Bernasconi, N. (2019). Recommendations for the use of structural magnetic resonance imaging in the care of patients with epilepsy: a consensus report from the International League Against Epilepsy Neuroimaging Task Force. *Epilepsia*, *60*(6), 1054-1068. doi:10.1111/epi.15612.

Bryson, A., Reid, C., & Petrou, S. (2023). Fundamental neurochemistry review: GABAA receptor neurotransmission and epilepsy: principles, disease mechanisms and pharmacotherapy. *Journal of neurochemistry*, *165*(1), 6-28. doi:10.1111/jnc.15769.

Chen, T. S., Huang, T. H., Lai, M. C., & Huang, C. W. (2023). The role of glutamate receptors in epilepsy. *Biomedicines*, *11*(3), 783. doi:10.3390/biomedicines11030783.

Deleu, B., Jeuris, J., & Van Paesschen, W. (2026). The interacting etiologies of hippocampal sclerosis in epilepsy: A scoping review. *Epilepsia*, *67*(2), 527-541. doi:10.1111/epi.18676.

Gauci, M. C., Gauci, R., Ruggieri, M., Polizzi, A., & Praticò, A. D. (2025). Clinical, genetic, EEG, neuroimaging insights and conservative treatment in pediatric focal epilepsy: a retrospective observational study. *Journal of Clinical Medicine*, *14*(7), 2234. doi:10.3390/jcm14072234.

Jamalipour Soufi, G., Hekmat Nia, A., Hajalikhani, P., Mehvari-Habibabadi, J., & Chit Saz, N. (2024). Correlation of magnetic resonance spectroscopy and magnetic resonance imaging with findings of electroencephalography in patients with temporal lobe epilepsy. *Journal of medical radiation sciences*, *71*(1), 51-56. doi:10.1002/jmrs.718.

Lee, S. B., Cho, Y. J., Jeon, K., Park, S., Lee, S., Choi, Y. H., ... & Kim, W. S. (2021). Diagnosis of hippocampal sclerosis in children: comparison of automated brain MRI volumetry and readers of varying experience. *American Journal of Roentgenology*, *217*(1), 223-234. doi:10.2214/AJR.20.23990.

Raafat, B. M., Alotaibi, A., Alfaqih, W., Alotaibi, Y., Alotaibi, J., Alghoraibi, F., ... & Fagiry, M. A. (2024). The effectiveness of MRI in the management of pediatric epilepsy at a tertiary care hospital in Taif, Saudi Arabia. *Journal of Radiation Research and Applied Sciences*, *17*(2), 100916. doi:10.1016/j.jrras.2024.100916.

Rentería-Palomo, A. A., Montes-Ochoa, J. L., Martinez-Mayorga, A., Reyes-Vaca, J. G., & Rodríguez-Leyva, I. (2021). Determination of the Initial Abnormal Discharge Site in Temporal Lobe Epilepsy Through Combined EEG and Neuroimaging. What Is Next?. *Frontiers in Neurology*, *12*, 798228.doi:10.3389/fneur.2021.798228.

RSUD Dr. Moewardi. (2024). Rekam Medis Pasien Rawat Jalan dan Rawat Inap.

Sarlo, G. L., & Holton, K. F. (2021). Brain concentrations of glutamate and GABA in human epilepsy: A review. *Seizure*, *91*, 213-227. doi:10.1016/j.seizure.2021.06.028.

Singh, R. P., Ranjan, R. K., Rai, N., Xalxo, A. R., Toppo, S. K., & Ram, P. N. (2023). Role Of Mri In Evaluation Of Epilepsy In Pediatric Age Group In A Tertiary Care Centre Of Jharkhand, India-A Prospective Study. *Student's Journal of Health Research Africa*, *4*(9), 7-7. doi:10.51168/sjhrafrica.v4i9.673.

Urbańska, S. M., Leśniewski, M., Welian-Polus, I., Witas, A., Szukała, K., & Chrościńska-Krawczyk, M. (2024). Epilepsy diagnosis and treatment in children–new hopes and challenges–literature review. *Journal of Pre-Clinical and Clinical Research*, *18*(1), 40-49. doi:10.26444/jpccr/185469.

Youssf, A. F., Shwaky, K. M., El Ghaiaty, H. A., & Soliman, F. I. (2021). Role of MRI in the diagnosis of epilepsy in children. *Benha Journal of Applied Sciences*, *6*(6), 155-159. doi:10.21608/bjas.2021.214404.

Wander, A., Chakrabarty, B., Gulati, S., Jauhari, P., Pandey, R. M., & Upadhyay, A. (2024). Optimal duration for recording EEG in children and adolescents-a prospective interventional study. *Seizure: European Journal of Epilepsy*, *115*, 14-19. doi:10.1016/j.seizure.2023.12.012.

7. Acknowledgements

The authors would like to express their gratitude to all parties who provided support and assistance throughout the research and preparation of this manuscript.

8. Author Declaration

- The authors have no conflicts of interest.
- Funding information was not provided in the source manuscript.