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*Original Article*

# Risk factors for secondary epilepsy in children with viral encephalitis

Yun Cheng[1], Rui Wang[1], Mingnong Xia[1], Fan Zhang[1], Wenbo Li[1], Feng Li[2], Wu Yang[1]

*[1]Department of Pediatrics, Lu'an People's Hospital Affiliated to Anhui Medical University, Lu'an 237000, Anhui, China; [2]Department of Neurology, Lu'an People's Hospital Affiliated to Anhui Medical University, Lu'an 237000, Anhui, China*

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**Abstract:** Objective: To investigate the clinical manifestations of secondary epilepsy (EP) in children with viral encephalitis and to identify any associated risk factors. Methods: A retrospective analysis was conducted on 130 children with viral encephalitis treated at Lu'an People's Hospital Affiliated with Anhui Medical University between December 2021 and October 2024. Of these, 36 children who developed secondary EP were classified as the EP group, and 94 children without secondary EP were categorized as the non-EP group. The overall incidence of secondary EP, clinical symptoms, cerebrospinal fluid (CSF) indices, and electroencephalogram (EEG) findings were compared between the groups. Multivariate logistic regression analysis was employed to identify independent risk factors for the development of secondary EP. Results: Of the 130 children with viral encephalitis, 36 (27.69%) developed secondary EP. Among them, 10 children (27.78%) had self-limited generalized EP, and 26 children (72.22%) had self-limited focal EP. Status epilepticus occurred in 7/36 cases (19.44%), but not in the other 29/36 cases (80.56%). No notable differences were observed in fever, headache, drowsiness, and coma between the EP group and non-EP group ($P>0.05$). However, vomiting and coma were significantly more frequent in the EP group ($P<0.05$). Abnormal EEG findings were also more prevalent in the EP group compared to the non-EP group ($P<0.05$). Logistic regression analysis identified non-use of antiepileptic drugs ($P=0.039$; CI: 0.181-0.958), elevated white blood cell count in CSF ($P=0.006$; CI: 1.028-1.185), and moderate to severe abnormal EEG results ($P=0.041$; CI: 1.035-5.41) as independent risk factors for the occurrence of secondary EP in children with viral encephalitis. Conclusion: The incidence of secondary EP in children with viral encephalitis is relatively high. Non-use of antiepileptic drugs, elevated white blood cell count in the CSF, and moderate to severe abnormal EEG results were independent risk factors for the occurrence of secondary EP in children with viral encephalitis.

**Keywords:** Viral encephalitis, epilepsy, clinical manifestations, risk factors, EEG examination

## Introduction

Viral encephalitis (sporadic encephalitis), is a common central nervous system infection associated with variable clinical outcomes [1]. While mild cases may resolve spontaneously, severe viral encephalitis can progress rapidly, leading to persistent symptoms such as confusion, coma, and even death [2]. Viral encephalitis is characterized by rapid onset, intense clinical symptoms, and a high mortality rate [3].

Children have relatively immature immune function, making them more susceptible to viral encephalitis [4]. Viral encephalitis can cause significant damage to brain tissue and nerves, leading to various complications [5]. Epilepsy (EP) is a common complication [6], with significant implications for neurodevelopment and substantial socioeconomic burdens for affected families [7]. As the disease progresses, it gradually affects the cerebral cortex, causing abnormal neuronal discharges, and resulting in secondary EP. This exacerbates brain injury and, without effective seizure control, may progress to refractory epilepsy, further worsening long-term neurologic outcomes [8].

The risk of early-onset EP following viral encephalitis is often underestimated, possibly due to

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the highly heterogeneous presentation of seizures [9, 10]. Research has indicated that the clinical symptoms of secondary EP following viral encephalitis are associated with the type of viral pathogen involved [11]. Encephalitis is a major contributor to the development of secondary EP, with most cases evolving into refractory EP, requiring long-term management and associated with poor prognoses [12]. Early detection and treatment of secondary EP following viral encephalitis are critical for improving patient outcomes. Identifying the risk factors for secondary EP should enable prevention and targeted treatment. However, research on the treatment and prognosis of secondary EP following viral encephalitis in China remains limited.

Therefore, this study investigated the clinical manifestations of secondary EP in children with viral encephalitis and identified risk factors associated with its development using logistic regression analysis. The findings are intended to provide reference data for clinical management and improve prevention and treatment of secondary EP in affected children.

## Patients and methods

### *Sample information*

This retrospective study was conducted on 155 children diagnosed with viral encephalitis and treated at our hospital between December 2021 and October 2024. The study was approved by the Ethics Committee of Lu'an People's Hospital Affiliated with Anhui Medical University.

Inclusion criteria: (1) Children who met the diagnostic criteria for viral encephalitis as outlined in the *Zhu Futang Practical Pediatrics* [13], including persistent fever, nausea, vomiting, positive meningeal signs, altered consciousness, changes in muscle tone, significant behavioral abnormalities, elevated white blood cell (WBC) count in cerebrospinal fluid (CSF), positive viral culture in CSF, evidence of brain parenchymal damage on head MRI or CT, and abnormal electroencephalogram (EEG) findings; (2) Children diagnosed with EP according to the definition of the International League Against Epilepsy (ILAE) [10]; (3) Patients with complete clinical data.

Exclusion criteria: (1) Children with non-viral encephalitis; (2) Children with autoimmune diseases; (3) Children with severe damage to vital organs such as the heart, brain, liver, or kidneys; (4) Children with a history of neurological diseases; (5) Children with seizures secondary to non-viral encephalitis.

Based on these criteria, 130 eligible patients were included in the study. Of them, 36 children with secondary EP following viral encephalitis were classified as the EP group, while the other 94 children without secondary EP were the non-EP group.

### *Diagnostic criteria*

The diagnosis of EP was based on the standards established by the ILAE in 2014 [14]. EP was diagnosed in children who experienced one or more non-provoked epileptic seizures following viral encephalitis, with EEG findings showing abnormal changes related to seizure symptoms.

### *Case data collection*

General information was collected through the hospital's electronic medical record system, including sex, age, time from symptom onset to hospital admission, history of head trauma, length of hospital stay, and family history of EP. Clinical examination data at admission included symptoms such as fever, headache, vomiting, drowsiness, and coma, as well as EEG findings. Laboratory data included CSF indices, such as pressure, protein concentration, and WBC count.

### *EEG examination*

All children underwent EEG examination on the day of admission using a Nihon Kohden Nilolet V32 digital EEG system. Scalp electrodes were placed according to the international 10-20 system. Depending on seizure occurrence and frequency, either routine or sleep-deprived EEG was performed. Monitoring durations ranged from 30 minutes to 24 hours, depending on the child's general condition.

### *Outcome measures*

*Primary outcome measures:* (1) Clinical symptoms were analyzed, including fever, headache, vomiting, drowsiness, and coma. (2) EEG findings were compared between the EP group and the non-EP group. EEG assessment was performed according to the criteria outlined in *Clinical Electroencephalography* [15]. Mild abnormality: Irregular alpha rhythm, absence or

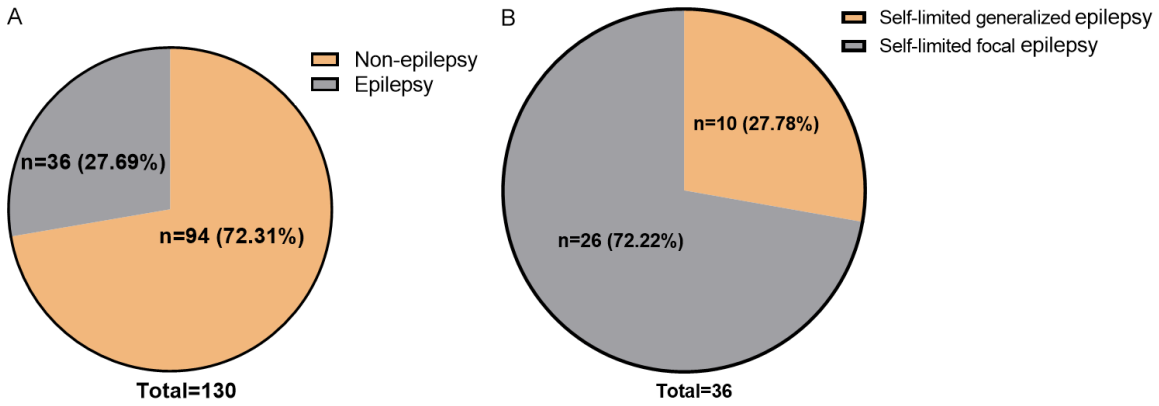


**Figure 1.** Overall occurrence of epilepsy in children with viral meningitis. A: The incidence of secondary epilepsy in children. B: The distribution of secondary epilepsy in children.

minimal suppression with eye-opening, presence of high-amplitude beta waves, increased Q-wave activity, appearance of high-amplitude Q waves after hyperventilation; Moderate abnormality: Disappearance or slowing of alpha rhythm, presence of episodic Q-wave activity, high-amplitude delta waves after hyperventilation; Severe abnormality: Disappearance or significant slowing of alpha rhythm, predominance of episodic Q-wave activity, high-voltage delta activity interspersed with slow waves, appearance of episodic delta waves, spontaneous or induced high-amplitude spike waves. (3) Multivariate logistic regression analysis was performed to identify independent risk factors affecting the occurrence of secondary EP in children with viral encephalitis.

*Secondary outcome measures:* (1) Baseline clinical data were analyzed, including age, gender, time from symptom onset to hospital admission, length of hospital stay, history of prior head trauma, family history of EP, use of antiepileptic drugs, and place of residence. (2) The overall incidence of secondary EP in the children was calculated. (3) Routine CSF indices were assessed, including WBC count, pressure, and protein content. Within 24 hours of admission, lumbar puncture was performed to collect 2 mL of CSF. The samples were centrifuged at 3,500 rpm for 10 minutes (centrifugal radius: 15 cm) to obtain the supernatant for analysis.

*Statistical analysis*

Statistical analyses were performed using SPSS 20.0 (IBM Corp, Armonk, NY, USA), and graph plotting was generated using GraphPad Prism 7 (GraphPad Software, San Diego, USA). Categorical variables were presented as [n (%)], and inter-group comparisons were conducted using the chi-square test. Continuous variables with normal distribution were expressed as mean ± standard deviation ($\bar{x}$±SD). Inter- and intra-group comparisons were performed using independent sample t-tests and paired t-tests, respectively. Logistic multivariate regression analysis was employed to identify independent risk factors associated with the development of secondary EP. Receiver Operating Characteristics (ROC) curves were generated to evaluate the predictive performance of the identified risk factors. A nomogram was subsequently constructed based on the logistic regression model using an online tool (https://shiny.medsta.cn/coxpre1/). A *P*-value <0.05 was considered significant.

## Results

*Overall occurrence of secondary EP in children with viral meningitis*

Among the 130 children with viral encephalitis, 36 cases (27.69%) developed secondary EP. Among them, 10 children (27.78%) had self-limited generalized EP, and 26 children (72.22%) had self-limited focal EP. Status epilepticus was observed in 7 cases (19.44%), while 29 cases (80.56%) did not develop status epilepticus (**Figure 1**).

*Comparison of baseline data between the two groups*

There were no significant differences between the EP group and the non-EP group in terms of

**Table 1.** Comparison of baseline data between the two groups

| Factor | EP group (n=36) | Non-EP group (n=94) | $\chi^2$/t | P |
|---|---|---|---|---|
| Age | 5.96±1.07 | 6.14±1.24 | 0.768 | 0.444 |
| Sex | | | 0.458 | 0.499 |
| Male | 17 | 58 | | |
| Female | 14 | 36 | | |
| Time from symptom onset to hospital admission (d) | 3.6±0.3 | 3.6±0.8 | 0.182 | 0.856 |
| Length of hospital stay (d) | 22.4±6.8 | 21.5±8.2 | 0.609 | 0.544 |
| History of head trauma | | | 0.410 | 0.522 |
| Yes | 3 | 5 | | |
| No | 33 | 89 | | |
| Family history of epilepsy | | | 4.327 | 0.038 |
| Yes | 6 | 5 | | |
| No | 30 | 89 | | |
| Use of antiepileptic drugs | | | 5.127 | 0.024 |
| Yes | 9 | 44 | | |
| No | 27 | 50 | | |
| Place of residence | | | 0.633 | 0.426 |
| Rural areas | 20 | 55 | | |
| Urban areas | 10 | 39 | | |

**Table 2.** Comparison of clinical symptoms between the two groups [n (%)]

| Clinical Feature | EP Group (n=36) | Non-EP Group (n=94) | $X^2$ | *P*-value |
|---|---|---|---|---|
| Fever | 30 (83.33%) | 73 (77.66%) | 0.509 | 0.476 |
| Headache | 25 (69.44%) | 69 (72.34%) | 0.204 | 0.652 |
| Vomiting | 29 (80.56%) | 54 (57.45%) | 6.022 | 0.014 |
| Drowsiness | 8 (22.22%) | 18 (19.15%) | 0.154 | 0.695 |
| Somnolence | 6 (16.67%) | 13 (18.83%) | 0.168 | 0.682 |
| Coma | 5 (13.89%) | 3 (3.19%) | 5.158 | 0.023 |

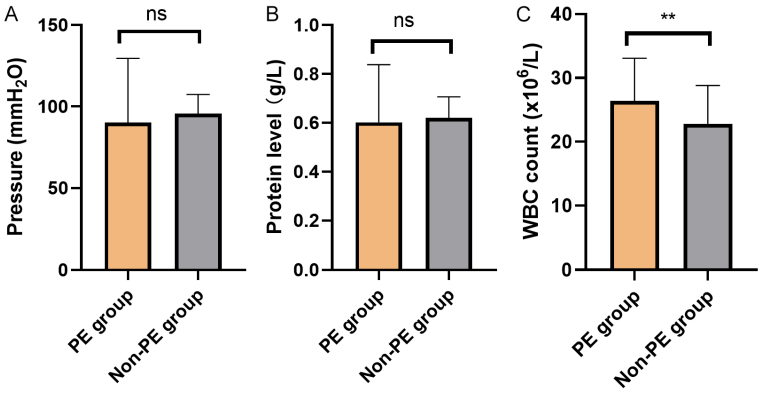


**Figure 2.** Comparison of routine CSF indices between the two groups. A: Comparison of CSF pressure between the two groups of children; B: Comparison of CSF protein levels between the two groups of children; C: Comparison of CSF WBC counts between the two groups of children. Note: ns: Non-significant; ***P*<0.01. EP: Epilepsy; WBC: White blood cell; CSF: Cerebrospinal fluid.

age, gender, time from symptom onset to hospital admission, length of hospital stay, history of head trauma, or place of residence (*P*>0.05). However, the EP group had a significantly lower rate of antiepileptic drug use (25.0% *vs.* 46.8%, *P*=0.024) and a higher prevalence of family history of epilepsy (16.7% *vs.* 5.3%, *P*= 0.038) compared to the non-EP group (*P*<0.05, **Table 1**).

*Comparison of clinical symptoms between the two groups*

In the EP group, the incidences of clinical symptoms were

**Table 3.** Comparison of EEG abnormalities between the two groups [n (%)]

| | Mild Abnormalities | Moderate Abnormalities | Severe Abnormalities |
|---|---|---|---|
| EP group (n=36) | 16 (44.44%) | 9 (25.00%) | 11 (30.56%) |
| Non-EP group (n=94) | 60 (63.83%) | 22 (23.40%) | 12 (12.77%) |
| $\chi^2$ | | 6.357 | |
| P | | 0.042 | |

Notes: EP: Epilepsy; EEG: electroencephalogram.

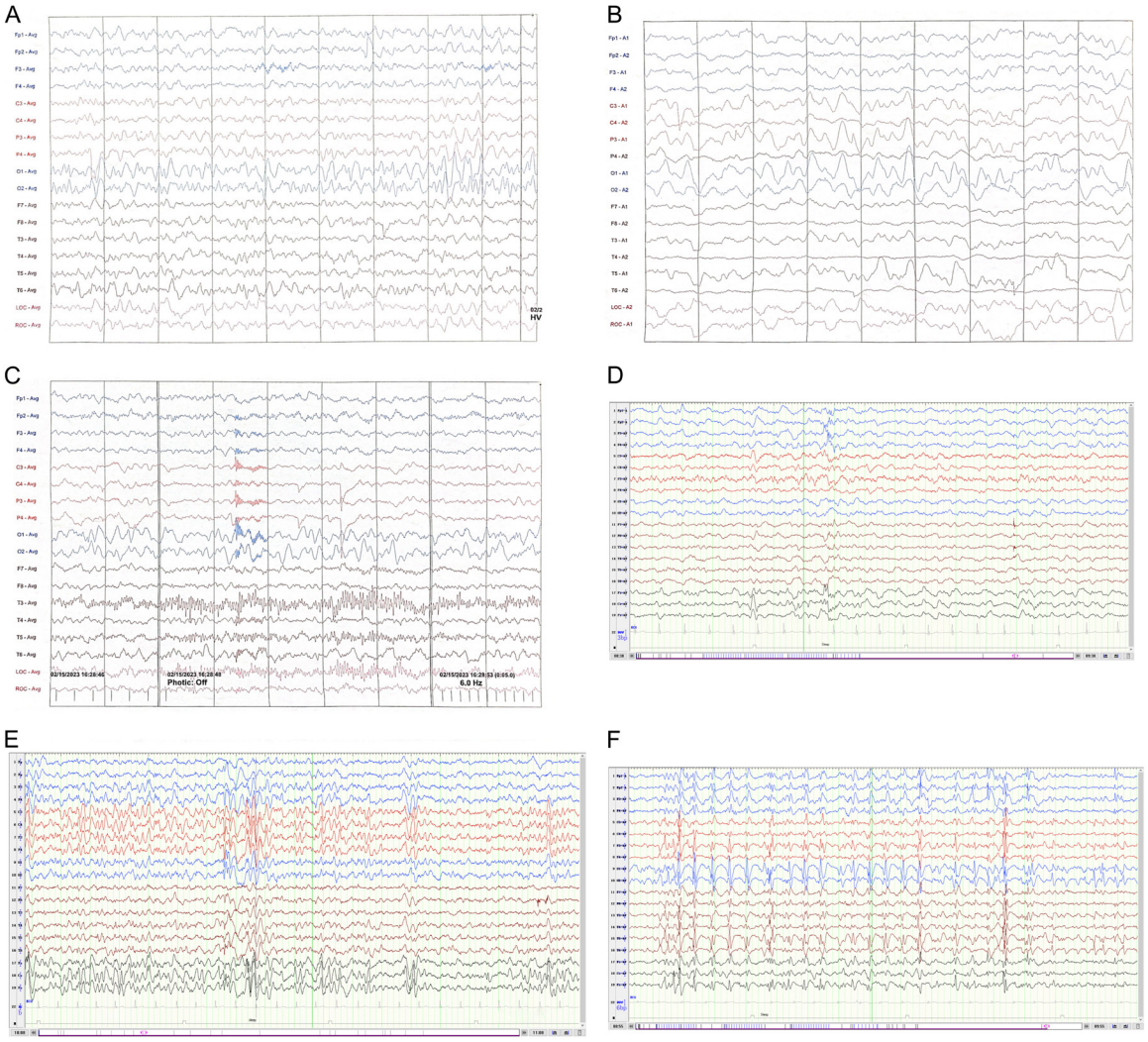


**Figure 3.** Representative EEG findings. A: Mild abnormality in the non-EP group. B: Moderate abnormality in the non-EP group. C: Severe abnormality in the non-EP group. D: Mild abnormality in the EP group. E: Moderate abnormality in the EP group. F: Severe abnormality in the EP group. Notes: EP: Epilepsy; EEG: electroencephalogram.

as follows: fever in 83.33% (30 cases), headache in 69.44% (25 cases), vomiting in 80.56% (29 cases), drowsiness in 22.22% (8 cases), somnolence in 16.67% (6 cases), and coma in 13.89% (5 cases). In the non-EP group, the corresponding rates were: fever in 77.66% (73 cases), headache in 72.34% (69 cases), vomiting in 57.45% (54 cases), drowsiness in 19.15% (18 cases), somnolence in 18.83% (13 cases), and coma in 3.19% (3 cases). There

**Table 4.** Univariate analysis of secondary EP in children with viral encephalitis

| Factor | EP group (n=36) | Non-EP group (n=94) | $\chi^2$/t | P |
|---|---|---|---|---|
| Family history of EP | | | 4.327 | 0.038 |
| Yes | 6 | 5 | | |
| No | 30 | 89 | | |
| Use of antiepileptic drugs | | | 5.127 | 0.024 |
| Yes | 9 | 44 | | |
| No | 27 | 50 | | |
| Vomiting | | | 6.022 | 0.014 |
| Yes | 29 | 54 | | |
| No | 7 | 40 | | |
| Coma | | | 5.158 | 0.023 |
| Yes | 5 | 3 | | |
| No | 31 | 91 | | |
| Cerebrospinal fluid white blood cell count ($10^6$/L) | 26.38±6.71 | 22.80±6.02 | 2.938 | 0.004 |
| Abnormal EEG examination | | | 6.357 | 0.042 |
| Mild Abnormalities | 16 | 60 | | |
| Moderate Abnormalities | 9 | 22 | | |
| Severe Abnormalities | 22 | 12 | | |

Notes: EP: Epilepsy; EEG: electroencephalogram.

**Table 5.** Assignment table

| Factor | Assignment | |
|---|---|---|
| | 1 | 0 |
| Family history of epilepsy | Yes | No |
| Use of antiepileptic drugs | No | Yes |
| Vomiting | Yes | No |
| Coma | Yes | No |
| Abnormal EEG examination | Moderate-severe abnormalities | Mild abnormalities |
| Secondary epilepsy | Yes | No |

Note: EEG: electroencephalogram.

**Table 6.** Multivariate analysis of secondary EP in children with viral encephalitis

| Factor | B | S.E. | Wals | df | Sig. | Exp (B) | 95% C.I. For EXP (B) | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower limit | Upper limit |
| Family history of epilepsy | 0.786 | 0.742 | 1.121 | 1 | 0.290 | 2.195 | 0.512 | 9.404 |
| Use of antiepileptic drugs | -0.875 | 0.425 | 4.250 | 1 | 0.039 | 0.417 | 0.181 | 0.958 |
| Vomiting | 0.452 | 0.457 | 0.980 | 1 | 0.322 | 1.572 | 0.642 | 3.846 |
| Coma | 0.823 | 1.110 | 0.850 | 1 | 0.357 | 0.360 | 0.041 | 3.164 |
| White blood cell count | 0.099 | 0.036 | 7.484 | 1 | 0.006 | 1.104 | 1.028 | 1.185 |
| Abnormal EEG examination | 0.861 | 0.422 | 4.167 | 1 | 0.041 | 2.367 | 1.035 | 5.411 |

Notes: EP: Epilepsy; EEG: electroencephalogram.

were no significant differences between the groups regarding the incidences of fever, headache, drowsiness, and somnolence ($P$>0.05). However, the EP group had significantly higher rates of vomiting and coma compared to the non-EP group ($P$<0.05, **Table 2**).

*Comparison of routine CSF indices between the two groups*

In the EP group, the mean CSF pressure, protein level, and WBC count were (90.14±39.36) $mmH_2O$, (0.60±0.23) g/L, and (26.38±6.71)

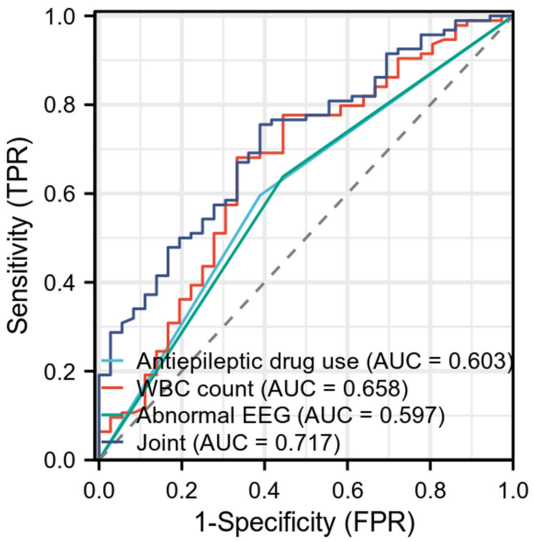


**Figure 4.** Predictive performance of independent risk factors and their combination for the occurrence of secondary EP in children with viral encephalitis. Notes: EP: Epilepsy.

× $10^6$/L, respectively. In the non-EP group, the corresponding values were (95.56±11.89) mm$H_2$0, (0.62±0.08) g/L, and (22.80±6.02) × $10^6$/L, respectively. The two groups had no significant differences in CSF pressure or protein content ($P$>0.05). However, the EP group had a significantly higher WBC count compared to the non-EP group ($P$<0.05, **Figure 2**).

*Comparison of EEG findings between the two groups*

In the EP group, mild, moderate, and severe EEG abnormalities were observed in 44.44% (16 cases), 25.00% (9 cases), and 30.56% (11 cases) of children, respectively. In the non-EP group, the proportions were 63.83% (60 cases), 23.40% (22 cases), and 12.77% (12 cases), respectively. The EP group had a higher incidence of moderate to severe EEG abnormalities compared to the non-EP group ($P$<0.05, **Table 3**). Representative EEG findings are shown in **Figure 3**.

*Univariate analysis of secondary EP in children with viral encephalitis*

Univariate analysis revealed significant differences between the EP and non-EP groups in terms of family history of EP, use of antiepileptic drugs, vomiting, coma, CSF WBC count, and EEG abnormalities (all P<0.05, **Table 4**). Therefore, these factors were identified as possible risk factors for the occurrence of secondary EP in children with viral encephalitis.

*Multivariate logistic regression analysis of secondary EP in children with viral encephalitis*

For multivariate analysis, variables showing significant differences in the univariate analysis were included and assigned with values (**Table 5**). Secondary EP was taken as the dependent variable, and family history of EP, use of antiepileptic drugs, vomiting, coma, CSF WBC count, and EEG abnormalities were used as independent variables. Logistic regression analysis identified non-use of antiepileptic drugs ($P$=0.039; CI: 0.181-0.958), elevated WBC count in CSF ($P$=0.006; CI: 1.028-1.185), and moderate to severe EEG abnormalities ($P$=0.041; CI: 1.035-5.41) as independent risk factors for secondary EP (**Table 6**).

*Predictive efficacy of independent risk factors and their combination for secondary EP*

ROC curves were generated to evaluate the predictive performance of the independent risk factors. The AUC of combined detection was 0.771, higher than that of any single factor (**Figure 4** and **Table 7**). Based on the logistic regression analysis results, a nomogram prediction model incorporating these independent risk factors was constructed to predict the occurrence of secondary EP in children with viral encephalitis (**Figure 5**). The calibration curve (**Figure 6**) demonstrated good agreement between predicted and observed probabilities (Hosmer-Lemeshow test, $P$=0.511). The bootstrap-corrected calibration curve (orange) closely aligned with the ideal reference line, indicating minimal overfitting (**Figure 6**).

## Discussion

Viral encephalitis is a severe neurological disorder characterized by cerebral inflammation, which substantially compromises quality of life for pediatric patients and imposes considerable burdens on caregivers [16, 17]. Secondary EP is a common complication of viral encephalitis, primarily resulting from cortical involvement that leads to abnormal neuronal discharges. Clinically, patients may present with episodic confusion, tonic-clonic seizures, limb rigidity, and urinary and fecal incontinence [18, 19].

**Table 7.** ROC values of independent risk factors and their combination for predicting the occurrence of secondary EP in children with viral encephalitis

| | Sensitivity | Specificity | Accuracy |
|---|---|---|---|
| Antiepileptic drug use | 59.57% | 61.11% | 60.00% |
| WBC count | 68.09% | 66.67% | 67.69% |
| Abnormal EEG | 63.83% | 55.56% | 61.54% |
| Joint | 75.53% | 61.11% | 71.54% |

Notes: ROC: Receiver Operating Characteristics; EP: Epilepsy; WBC: White blood cell; EEG: electroencephalogram.

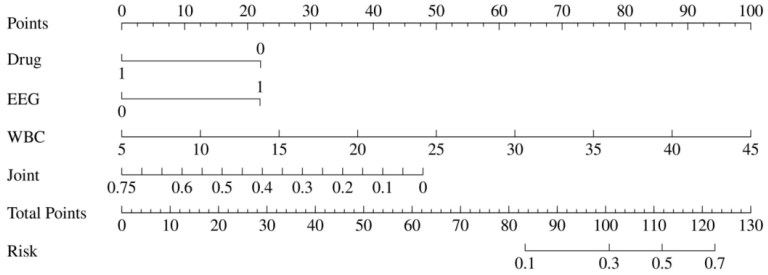


**Figure 5.** Nomogram for predicting the occurrence of secondary EP in children with viral encephalitis. Note: EP: Epilepsy.

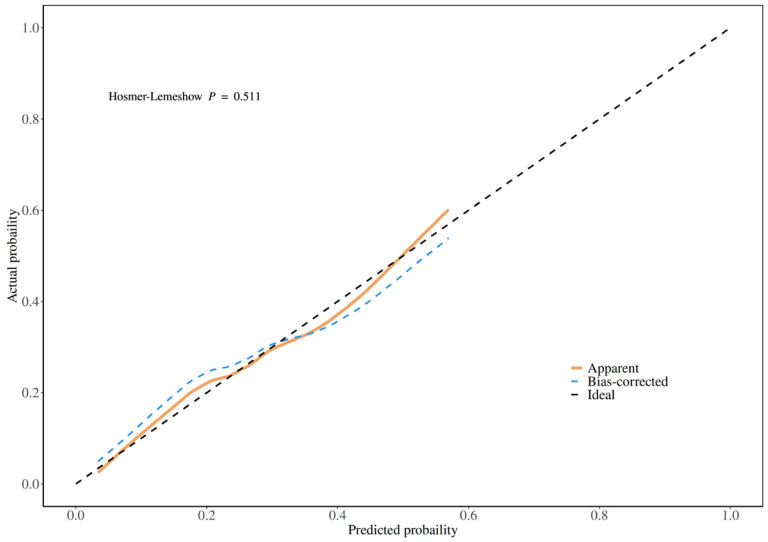


**Figure 6.** Calibration curve of the nomogram for predicting secondary epilepsy. Notes: The apparent (blue) and bias-corrected (orange) curves represent the original and bootstrap-adjusted predictions, respectively. The gray diagonal line indicates perfect calibration. The Hosmer-Lemeshow test shows good calibration without significant deviation (P=0.511).

This study analyzed the clinical characteristics of secondary EP and the risk factors associated with its development in children with viral encephalitis.

In this study, 36 patients (27.69%) developed secondary epilepsy, significantly higher than the 10.99% reported by Hu et al. [20]. This discrepancy may be due to the relatively small sample size in our study, possibly introducing selection bias. Further research with larger sample sizes is needed to validate these findings. No significant differences were observed between the EP and the non-EP groups in terms of fever, headache, or drowsiness, indicating that these symptoms lack specificity for distinguishing between children at high or low risk of developing secondary EP. However, the incidences of vomiting and coma were higher in the EP group compared to the non-EP group. Subsequent univariate analysis confirmed vomiting and coma as influencing factors for secondary EP. Yang et al. [6] and Chen et al. [9] also reported coma as an independent risk factor for secondary EP. This may be explained by the association between coma and extensive brain cell damage due to viral invasion, leading to cerebral ischemia, hypoxia, and brain cell edema, which in turn increases the likelihood of epileptic seizures [21, 22]. Additionally, a family history of EP and non-use of antiepileptic drugs were identified as risk factors for the development of secondary EP in children with viral encephalitis. Genetic predisposition in children with a family history of epilepsy likely contributes to increased susceptibility to seizures [23]. Moreover, non-use of antiepileptic drugs was identified as an independent risk factor for secondary EP. The use of antiepileptic drugs can prevent seizure occurrence by stabilizing neuronal excitability and maintaining neuronal function, thereby reducing the risk of secondary EP and mitigating neurological damage [24, 25].

Furthermore, this study found no significant differences in CSF pressure or protein concentration between the two groups. However, the EP group exhibited a significantly higher CSF WBC count compared to the non-EP group. Moreover, logistic regression analysis confirmed that elevated WBC count was an independent risk factor for the development of secondary EP in children with viral encephalitis. This finding may be explained by the fact that an increase in WBC count in the CSF reflects an intensified inflammatory response, which can lead to abnormal neuronal excitability, altered synaptic transmission, and neuronal damage, thereby facilitating the occurrence and progression of EP [26]. Moderate to severe abnormalities in the EEG were also identified as independent risk factors for secondary EP. Research has reported that EEG has certain advantages in diagnosing EP [27]. The present findings suggest that moderate to severe EEG abnormalities are independent risk factors for secondary EP in children with viral encephalitis. This is likely due to acute brain inflammation caused by viral invasion of the central nervous system, which induces abnormal neuronal discharges and subsequent epileptic events [28].

In the final stage of the study, ROC curve analysis was performed to evaluate the predictive performance of combined detection (non-use of antiepileptic drugs, elevated CSF WBC count, and moderate to severe EEG abnormalities) for secondary EP. The combined model demonstrated moderate discriminative capacity, with an AUC of 0.771, outperforming any single predictor and significantly improving classification accuracy. Identifying and assessing risk factors can assist clinicians in making more accurate diagnoses and developing personalized preventive strategies for children at risk of secondary EP following viral encephalitis.

This study still has several limitations. First, it is a retrospective study based on existing data and statistical modeling, which may introduce selection bias and limit the ability to establish causal relationships. Second, the relatively small sample size may have reduced the degree of statistical power and generalizability of the findings. Third, model evaluation was restricted to ROC analysis without assessment of clinical utility using decision curve analysis (DCA) or probability calibration. Therefore, further prospective multicenter studies with larger cohorts are still needed to validate these results and explore the underlying mechanisms in greater depth.

## Conclusion

Our study demonstrated three key findings. First, the incidence of secondary epilepsy (27.69%) in pediatric patients with viral encephalitis underscores the importance of vigilant neurological monitoring in this population. Second, non-use of antiepileptic drugs, elevated CSF WBC count, and moderate to severe EEG abnormalities were identified as independent risk factors for the development of secondary EP in children with viral encephalitis. Third, the combined predictive model (AUC= 0.771) outperformed individual predictors, providing clinicians with a practical tool for risk stratification. These findings suggest that early EEG monitoring and consideration of prophylactic antiepileptic therapy may be warranted in high-risk cases, though prospective studies are needed to validate these recommendations.

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## Disclosure of conflict of interest

None.

**Address correspondence to:** Wu Yang, Department of Pediatrics, Lu'an People's Hospital Affiliated to Anhui Medical University, Lu'an 237000, Anhui, China. E-mail: ruijing9516@163.com

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