

Initial Management of a Child with Suspected Acute Encephalitis in a Community Hospital: A Case Report

Takayuki Hamada^{1*}, Toru Oshibuchi¹, Takumi Yamada², Mio Kishino³, Yoshikazu Otsubo³

¹Department of Surgery, National Health Insurance Hirado Municipal Hospital, Hirado, Japan

²Department of Internal Medicine, National Health Insurance Hirado Municipal Hospital, Hirado, Japan

³Department of Pediatrics, Sasebo City General Hospital, Sasebo, Japan

Email: *takahamada@hotmail.co.jp

How to cite this paper: Hamada, T., Oshibuchi, T., Yamada, T., Kishino, M. and Otsubo, Y. (2026) Initial Management of a Child with Suspected Acute Encephalitis in a Community Hospital: A Case Report. *Case Reports in Clinical Medicine*, 15, 454-462.

<https://doi.org/10.4236/crcm.2026.1510057>

Received: September 10, 2026

Accepted: October 18, 2026

Published: October 21, 2026

Copyright © 2026 by author(s) and Scientific Research Publishing Inc. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

A previously healthy, fully immunized 13-year-old boy presented to a community hospital with fever, generalized seizures, impaired consciousness, and respiratory failure. He required intubation, mechanical ventilation, sedation, and correction of metabolic acidosis. Despite persistent seizures, he was stabilized and transferred to a tertiary intensive care unit for suspected acute encephalitis and, he rapidly improved. Magnetic resonance imaging and electroencephalography findings were normal. Cerebrospinal fluid findings were consistent with central nervous system inflammation, and viral meningitis was considered; however, no specific viral etiology was identified. He recovered without neurological deficits and was discharged on day 6. This case highlights the importance of prompt initial management of suspected pediatric acute encephalitis in a resource-limited community hospital.

Keywords

Acute Encephalitis, Children, Initial Management, Community, Case Report

1. Introduction

In community healthcare, providing initial treatment for a broad range of conditions affecting individuals across all age groups, including suspected pediatric acute encephalitis, is essential. Encephalitis is a syndrome characterized by neurological dysfunction resulting from inflammation of the brain parenchyma [1]. In children, acute encephalitis presents with a variety of clinical manifestations, including fever, impaired consciousness, seizures, and respiratory failure. As this

condition often progresses rapidly, leading to death or severe neurological sequelae, initial management is critical, especially in community hospitals located far from advanced medical facilities. In Japan, acute encephalitis/encephalopathy annually affects 1,000 children aged < 15 years, with a mortality rate of 5% - 10% and neurological sequelae observed in 20% - 30% of cases [2]. This report describes a case of suspected acute encephalitis in a child presenting with seizures and respiratory failure in a remote area of Japan and highlights the importance of early intervention in suspected pediatric acute encephalitis.

2. Case Presentation

A previously healthy, fully immunized 13-year-old boy was transported to our hospital by emergency medical services (EMS) because of seizures and respiratory failure. Upon arrival, he was unconscious with shallow breathing and an oxygen saturation (SpO₂) of 60% on room air. Oral suctioning and assisted ventilation with oxygen were initiated using a manual resuscitator, during which he experienced a seizure lasting approximately 1 minute. He was brought to our hospital 32 minutes after the onset of symptoms. No sick contact was confirmed; however, he had been experiencing upper respiratory symptoms for several days prior to the event. He had no prior travel history and had no contact with or been bitten by any animals; he also had no exposure to mosquitoes or ticks. No family history of epilepsy was found. Previous herpes simplex virus (HSV) and varicella zoster virus (VZV) infections were not confirmed. His vital signs were as follows: body temperature, 39.0°C; pulse, 153 beats/minute; blood pressure, 190/93 mmHg; and SpO₂, 98% under assisted ventilation with oxygen administration using a manual resuscitator. Physical examination revealed a Glasgow Coma Scale score of 10 (eye response +4, verbal response +2, motor response +4), generalized seizures, and severe respiratory distress. Intubation was attempted immediately under intravenous diazepam sedation (total doses: 10 mg); however, it was unsuccessful due to recurrent generalized seizures. The patient was subsequently sedated with intravenous midazolam (total doses: 4.5 mg) and successfully intubated 28 minutes after arrival at the hospital. No signs of meningeal irritation were observed during sedation and intubation. Blood gas analysis following intubation and mechanical ventilation (**Table 1**) revealed significant acidosis (pH, 7.106; partial pressure of carbon dioxide [PaCO₂], 50.3 mmHg; partial pressure of oxygen [PaO₂], 209.0 mmHg; bicarbonate [HCO₃⁻], 15.1 mmol/L; base excess, -14.8 mmol/L; blood glucose, 251 mg/dL; lactic acid, 14.9 mmol/L), prompting the immediate administration of sodium bicarbonate. However, computed tomography (CT) of the brain revealed no abnormalities (**Figure 1**). Laboratory studies (**Table 2**) revealed a white blood cell count of $31.6 \times 10^3/\mu\text{L}$ with a normal differential; platelet count of $416 \times 10^3/\mu\text{L}$; hemoglobin level of 14.5 g/dL; C-reactive protein of 0.05 mg/dL; blood urea nitrogen of 20.4 mg/dL; creatinine of 1.48 mg/dL; and ammonia of 147 $\mu\text{g/dL}$. Hepatobiliary enzymes, the remaining complete metabolic panel, and coagulation studies were within normal ranges. Blood gas analysis after 1 hour (**Table 3**) demonstrated improvement (pH, 7.428; PaCO₂, 45.9 mmHg; PaO₂,

472.4 mmHg; HCO_3^- , 29.7 mmol/L; base excess, +5.0 mmol/L, lactic acid, 6.6 mmol/L). Nasopharyngeal exudate testing was negative for severe acute respiratory syndrome coronavirus 2 and influenza virus antigens. Despite intubation, mechanical ventilation, and repeated midazolam injection, the patient continued to experience generalized seizures and involuntary movements. He was administered midazolam for continuous sedation and transferred to a specialized medical facility for further care on suspicion of acute encephalitis 2 hours after arrival at our hospital. During the two-hour stay at our hospital, the patient experienced eight seizures lasting 3 - 5 minutes each, and two more seizures during the one-hour transport, and the patient's consciousness did not recover between seizures, which fulfilled the criteria for status epilepticus. After transfer, the patient was admitted to the intensive care unit and received mechanical ventilation with sedation using midazolam and fentanyl. No seizures were observed, and metabolic acidosis and hyperammonemia improved. Magnetic resonance imaging (MRI) of the brain performed on the day revealed no evidence of hemorrhagic lesions, encephalopathy, or encephalitis. Additionally, electroencephalography revealed no abnormalities. The patient's respiratory condition rapidly improved, and purposeful movement was noted even under sedation, prompting extubation the following day. Cerebrospinal fluid (CSF) studies (**Table 4**) performed on 15 hours after the onset of symptoms indicated a high protein level of 97.8 mg/dL, increased cell counts of 51/ μL with 94.1% monocytes, and normal glucose and chloride levels. Serum blood glucose level was 111 mg/dL. Gram staining and bacterial culture of the CSF did not reveal any bacterial growth, and blood culture yielded no bacterial growth. The CSF polymerase chain reaction (PCR) test did not identify any pathogens (**Table 5**). Because the patient's condition improved rapidly, repeated PCR tests for herpes simplex virus were not performed. Based on the clinical course and a series of test results, the possibility of meningitis caused by some other virus was considered. After sedation was discontinued, the patient regained full consciousness and had no difficulty with daily activities. The altered mental status was not observed. The patient was discharged on day 6 and exhibited no neurological deficits at the 6-month follow up.



Figure 1. Brain computed tomography displaying preserved gray-white matter differentiation with no evidence of intracranial hemorrhage, infarction, or structural abnormalities.

Table 1. Arterial blood gas analysis obtained after intubation and initiation of mechanical ventilation.

Parameter	Value	Reference range
pH	7.106	7.34 - 7.45
PaCO ₂ (mmHg)	50.3	32 - 45
PaO ₂ (mmHg)	209	69 - 116
HCO ₃ ⁻ (mmol/L)	15.1	20 - 26
Base excess (mmol/L)	-14.8	-3.3 - +2.3
Lactic acid (mmol/L)	14.9	0.5 - 1.6
Glucose (mg/dL)	261	73 - 109

PaCO₂, partial pressure of carbon dioxide; PaO₂, partial pressure of oxygen; HCO₃⁻, bicarbonate.

Table 2. Initial laboratory findings on admission.

Parameter	Value	Reference range
Complete Blood Count		
White blood cells (×10 ³ /μL)	31.6	3.3 - 8.6
Hemoglobin (g/dL)	14.5	13.7 - 16.8
Hematocrit (%)	46	40.7 - 50.1
Platelets (×10 ³ /μL)	416	158 - 348
Neutrophils (%)	52.1	
Lymphocytes (%)	43.5	
Eosinophils (%)	1	
Basophils (%)	0.5	
Coagulation Studies		
Activated partial thromboplastin time (seconds)	21.6	24.0 - 38.0
Prothrombin time (seconds)	10.9	10.0 - 12.0
International normalized ratio	0.98	0.84 - 1.14
Complete Metabolic Pannel		
Sodium (mmol/L)	139	138 - 145
Potassium (mmol/L)	4.8	3.6 - 4.8
Chloride (mmol/L)	95	101 - 108
Calcium (mg/dL)	9.9	8.8 - 10.1
Phosphate (mg/dL)	10.6	2.7 - 4.6
Blood urea nitrogen (mg/dL)	20.4	8.0 - 20
Creatinine (mg/dL)	1.48	0.65 - 1.07
Amylase (U/L)	263	44 - 132
Creatinine phosphokinase (U/L)	157	59 - 248

Continued

Ammonia (mg/dL)	147	40 - 80
Total protein (g/dL)	7.9	6.6 - 8.1
Albumin (g/dL)	4.5	4.1 - 5.1
Aspartate aminotransferase (U/L)	39	13 - 30
Alanine aminotransferase (U/L)	43	10 - 42
Lactate dehydrogenase (U/L)	342	124 - 222
Alkaline phosphatase (U/L)	320	38 - 113
γ -Glutamyl transpeptidase (U/L)	63	13 - 64
Total bilirubin (mg/dL)	0.3	0.4 - 1.5

Table 3. Arterial blood gas analysis performed 1 hour after hospital arrival.

Parameter	Value	Reference range
pH	7.428	7.34 - 7.45
PaCO ₂ (mmHg)	45.9	32 - 45
PaO ₂ (mmHg)	472.4	69 - 116
HCO ₃ ⁻ (mmol/L)	29.7	20 - 26
Base excess (mmol/L)	5.0	-3.3 - +2.3
Lactic acid (mmol/L)	6.6	0.5 - 1.6

PaCO₂, partial pressure of carbon dioxide; PaO₂, partial pressure of oxygen; HCO₃⁻, bicarbonate.

Table 4. Cerebrospinal fluid analysis performed at the referral hospital.

Parameter	Value	Reference Range
Appearance	clear and colorless	
Xanthochromia	negative	
Bloody	negative	
pH	7.6	7.31 - 7.34
Specific gravity	1.006	1.005 - 1.007
Cell counts (/μL)	51	0 - 5
Polymorphonuclear cells (%)	0.7	
Monocytes (%)	94.1	
Large mononuclear cells (%)	5.1	
Red blood cells (/μL)	1.0	
Protein (mg/dL)	97.8	8 - 43
Glucose (mg/dL)	49	50 - 75
Chloride (mmol/L)	125	120 - 130
Lactate dehydrogenase (U/L)	23	8 - 50

Table 5. PCR test performed at the referral hospital on 15 hours after the onset of symptoms.

Parameter	Result
<i>Escherichia coli</i>	negative
<i>Haemophilus influenzae</i>	negative
<i>Listeria monocytogenes</i>	negative
<i>Neisseria meningitidis</i>	negative
Group B streptococcus	negative
<i>Streptococcus pneumoniae</i>	negative
Cytomegalovirus	negative
Enterovirus	negative
Herpes simplex virus-1	negative
Herpes simplex virus-2	negative
Human herpes virus-6	negative
Human parecho virus	negative
Varicella zoster virus	negative

PCR; polymerase chain reaction.

3. Discussion

We report the case of a pediatric patient with suspected acute encephalitis presenting with respiratory failure and generalized seizures in a remote area of Japan. Although acute encephalitis in children is relatively rare, the condition can progress rapidly, often leading to death or severe neurological sequelae. Clinical outcomes depend on prompt identification of the cause and initiation of appropriate treatment [3]. Therefore, physicians in community hospitals play a crucial role in the initial management of children with suspected acute encephalitis.

Pediatric acute encephalitis has diverse etiologies, including viral, autoimmune, and demyelinating causes, with initial clinical presentations and laboratory findings often being nonspecific. In immunocompetent individuals in Europe and the United States, viral encephalitis is most commonly caused by HSV types 1 and 2, VZV, and enterovirus [4] [5]. In cases of suspected encephalitis at the initial visit, hypoglycemia, electrolyte imbalances, and head trauma must be considered in the differential diagnosis. In the present case, these conditions were ruled out based on laboratory data and brain CT findings.

MRI of the brain is critical for diagnosing suspected encephalitis, as the modality allows for morphological and functional studies that can identify lesions in the cerebral parenchyma, even during the hyperacute phase [6]. CSF analysis is also essential for suspected encephalitis cases. Although both examinations are valuable for diagnosis, avoiding delays in definitive diagnostic confirmation is vital in community hospitals in remote areas. If acute encephalitis is strongly suspected, priorities in a remote hospital are life support and brain function protection before transfer for further investigation at a higher-level medical institution. Therefore, pre-

transfer care includes three keys: first, respiratory and circulatory management; second, suppression of seizures and increased intracranial pressure; and third, correction of metabolic disturbances, including blood gas and electrolyte abnormalities.

However, exceptions exist, particularly when HSV encephalitis cannot be ruled out. The mortality rate associated with untreated HSV encephalitis is approximately 70%, and even with antiviral therapy, the rate remains over 30%. Furthermore, nearly 60% of survivors develop neurological sequelae [7]. Prompt diagnosis and initiation of antiviral therapy are crucial for favorable outcomes in HSV encephalitis [8]. Even if initial CSF or imaging findings are normal, acyclovir should be administered within 6 hours of symptom onset if HSV encephalitis is clinically suspected [1]. As brain CT scan has a 50% detection rate for acute changes in HSV encephalitis [9], this condition could not be excluded in the present case before transfer to an advanced medical institution. Although the patient had mild renal impairment, acyclovir should have been administered with adequate fluid replacement before transfer.

Moreover, bacterial meningitis is a crucial differential diagnosis for suspected acute encephalitis. Although seizures and respiratory distress are uncommon early symptoms, bacterial meningitis necessitates urgent initial treatment similar to that for HSV encephalitis. Given the high case-fatality rate of up to 30% and the development of neurological complications in up to 66.1% of survivors, the condition should be empirically treated before a specific pathogen is identified [10]–[12]. Delays in antibiotic administration exceeding 2 hours are associated with doubled mortality [13]; thus, antibiotics should be administered as soon as bacterial meningitis is suspected, ideally within 2 hours [14]. In this case, the absence of meningeal sign and nonspecific blood laboratory findings cannot reliably exclude bacterial meningitis, particularly in a sedated and intubated condition. Empiric antibiotic administration should have been initiated before transfer to an advanced medical institution.

4. Conclusion

In suspected pediatric acute encephalitis cases in remote areas, initial management is critical. Priorities include providing ventilatory and circulatory support and protecting brain function. Only essential imaging and blood chemistry tests should be performed, and any metabolic disturbances should be corrected before the patient is promptly transferred to an advanced medical facility. However, if HSV encephalitis or bacterial meningitis cannot be ruled out, initial treatment should be administered at the community hospital prior to transfer. Physicians treating children in remote areas must understand the management of suspected encephalitis, as this can facilitate earlier diagnosis and treatment, ultimately improving outcomes.

Consent for Publication

Written informed consent for publication was obtained from the patient's parents.

Author Contributions

Conceptualization, T.H. and T.Y.; Investigation, T.H., T.O., T.Y., M.K., Y.O.; Writing-original draft preparation, T.H.; Writing-review and editing, T.H.; Supervision, T.O. and Y.O. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare they have no competing interests.

References

- [1] Kneen, R., Michael, B.D., Menson, E., Mehta, B., Easton, A., Hemingway, C., *et al.* (2012) Management of Suspected Viral Encephalitis in Children—Association of British Neurologists and British Paediatric Allergy, Immunology and Infection Group National Guidelines. *Journal of Infection*, **64**, 449-477. <https://doi.org/10.1016/j.jinf.2011.11.013>
- [2] Morishima, T. (2009) Infantile Viral Encephalitis and Encephalopathy in Japan. *Uirusu*, **59**, 59-66. <https://doi.org/10.2222/jsv.59.59>
- [3] Huong, N.H.T., Toan, N.D., Quy, D.T., Khanh, T.H., Thinh, L.Q., Nhan, L.N.T., *et al.* (2022) Study Protocol: The Clinical Features, Epidemiology, and Causes of Paediatric Encephalitis in Southern Vietnam. *Wellcome Open Research*, **6**, Article No. 133. <https://doi.org/10.12688/wellcomeopenres.16770.3>
- [4] Glaser, C.A., Gilliam, S., Schnurr, D., Forghani, B., Honarmand, S., Khetsuriani, N., *et al.* (2003) In Search of Encephalitis Etiologies: Diagnostic Challenges in the California Encephalitis Project, 1998-2000. *Clinical Infectious Diseases*, **36**, 731-742. <https://doi.org/10.1086/367841>
- [5] Donoso Mantke, O., Vaheri, A., Ambrose, H., Koopmans, M., de Ory, F., Zeller, H., *et al.* (2008) Analysis of the Surveillance Situation for Viral Encephalitis and Meningitis in Europe. *Eurosurveillance*, **13**, pii=8017. <https://doi.org/10.2807/es.e.13.03.08017-en>
- [6] Urso, L., Distefano, M.G., Cambula, G., Colomba, A.I., Nuzzo, D., Picone, P., *et al.* (2022) The Case of Encephalitis in a COVID-19 Pediatric Patient. *Neurological Sciences*, **43**, 105-112. <https://doi.org/10.1007/s10072-021-05670-9>
- [7] Piret, J. and Boivin, G. (2015) Innate Immune Response during Herpes Simplex Virus Encephalitis and Development of Immunomodulatory Strategies. *Reviews in Medical Virology*, **25**, 300-319. <https://doi.org/10.1002/rmv.1848>
- [8] Erdem, H., Cag, Y., Ozturk-Engin, D., Defres, S., Kaya, S., Larsen, L., *et al.* (2015) Results of a Multinational Study Suggest the Need for Rapid Diagnosis and Early Antiviral Treatment at the Onset of Herpetic Meningoencephalitis. *Antimicrobial Agents and Chemotherapy*, **59**, 3084-3089. <https://doi.org/10.1128/aac.05016-14>
- [9] Whitley, R.J. (2006) Herpes Simplex Encephalitis: Adolescents and Adults. *Antiviral Research*, **71**, 141-148. <https://doi.org/10.1016/j.antiviral.2006.04.002>
- [10] van de Beek, D., de Gans, J., Spanjaard, L., Weisfelt, M., Reitsma, J.B. and Vermeulen, M. (2004) Clinical Features and Prognostic Factors in Adults with Bacterial Meningitis. *New England Journal of Medicine*, **351**, 1849-1859. <https://doi.org/10.1056/nejmoa040845>
- [11] Araújo, R.D., Diniz, L.M.O., Teixeira, D.C., Viana, A.C.V.F., Franco, L.P., Carvalho, S.A.d.S., *et al.* (2026) Long-Term Outcomes Following Bacterial Meningitis in Child-

- hood: A Systematic Review and Meta-Analysis. *Journal of Paediatrics and Child Health*, **62**, 1335-1345. <https://doi.org/10.1111/jpc.70469>
- [12] Schiess, N., Groce, N.E. and Dua, T. (2021) The Impact and Burden of Neurological Sequelae Following Bacterial Meningitis: A Narrative Review. *Microorganisms*, **9**, Article No. 900. <https://doi.org/10.3390/microorganisms9050900>
- [13] Eisen, D.P., Hamilton, E., Bodilsen, J., Køster-Rasmussen, R., Stockdale, A.J., Miner, J., *et al.* (2022) Longer than 2 Hours to Antibiotics Is Associated with Doubling of Mortality in a Multinational Community-Acquired Bacterial Meningitis Cohort. *Scientific Reports*, **12**, Article No. 672. <https://doi.org/10.1038/s41598-021-04349-7>
- [14] Carter, E. and McGill, F. (2022) The Management of Acute Meningitis: An Update. *Clinical Medicine*, **22**, 396-400. <https://doi.org/10.7861/clinmed.2022-cme-meningitis>