

Wernicke Encephalopathy Induced by Acute Cholecystitis A Case Report

Yucheng Fan

fan1993919@hotmail.com

Ningxia Medical University

Jizhou Ma

Ningxia Medical University

Yongjun Du

Ningxia Medical University

Yikun Cao

Ningxia Medical University

Xiaohua Wang

Ningxia Medical University

Peng Wu

Ningxia Medical University

Tao Ma

Ningxia Medical University

Li Jing

Ningxia Medical University

Hang Han

Ningxia Medical University

Feng Jing

The 942nd Hospital of PLA Joint Logistics Support Force

Fangjing Xu

The 942nd Hospital of PLA Joint Logistics Support Force

Case Report

Keywords: Acute Cholecystitis, Vitamin B1 deficiency, Wernicke encephalopathy

Posted Date: October 4th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11074076/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.
[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Introduction:

Wernicke's encephalopathy (WE) is an acute central nervous system disorder caused by thiamine deficiency, characterized by a classic triad of ophthalmoplegia, ataxia, and altered mental status. Chronic alcohol use is the most common etiology of WE, but the disease is often misdiagnosed clinically as only a minority of patients exhibit the typical symptoms clearly.

Case Report:

A patient with acute cholecystitis complicated by WE is reported, who presented with altered consciousness and dysarthria. Magnetic resonance imaging of the brain revealed high signal intensity on fluid-attenuated inversion recovery (FLAIR) sequences, affecting the dorsolateral brainstem, periaqueductal gray, bilateral thalami, and bilateral parietal lobes. Prior to the onset of neurological symptoms, the patient experienced abdominal pain and loss of appetite due to acute cholecystitis. Suspecting WE, high-dose thiamine treatment was initiated along with rehabilitative functional training. The clinical condition improved rapidly within two weeks.

Conclusion

In cases of acute cholecystitis accompanied by loss of appetite and fasting, endogenous thiamine reserves can be rapidly depleted. Early adequate parenteral vitamin B1 administration is critical. Additionally, early and tailored rehabilitative interventions for patients exhibiting significant cognitive and muscular impairments are essential for improving prognosis.

1. Introduction

Wernicke's Encephalopathy (WE) is a central nervous system metabolic disorder that is closely associated with thiamine (vitamin B1) deficiency. This condition often presents with insidious onset, exhibits a low incidence rate, and displays non-specific clinical symptoms. It is frequently observed in individuals with a history of chronic alcoholism, malnutrition, malignancies, gastrointestinal diseases, specific metabolic disorders, or prior surgical interventions. The underlying pathophysiological mechanism is primarily attributed to thiamine deficiency-induced energy metabolism disruption, resulting in dysfunctional and damaged neuronal cells^[1-2]. The hallmark clinical manifestations typically include ophthalmoplegia, ataxia, altered consciousness, or memory impairment, along with other neurological symptoms^[2-3]. WE is a relatively rare clinical entity, and definitive diagnosis lacks a gold standard. It commonly relies on a combination of patient history, clinical symptoms, and imaging studies. Magnetic Resonance Imaging (MRI) may reveal highly specific alterations, primarily localized in the bilateral thalami, mammillary bodies, midbrain tegmentum, and periaqueductal symmetrical regions;

however, atypical areas such as the frontal and parietal cortices, corpus callosum, and dentate nuclei of the cerebellum may also be involved ^[4–5]. Laboratory investigations can be employed to assess thiamine levels and exclude other potential diseases. Diagnosing and differentiating complicated Wernicke's encephalopathy or cases with multiple comorbidities can pose additional challenges.

In recent years, an increased understanding of Wernicke's encephalopathy has led to a focus on early diagnostic techniques, underlying etiological mechanisms, and clinical treatment strategies. This report describes the diagnostic and therapeutic management of a patient diagnosed with acute cholecystitis complicated by Wernicke's encephalopathy.

2. Case presentation

A 59-year-old male was admitted to the hospital due to abdominal pain and altered consciousness. Upon admission, symptoms included confusion and dysarthria, characterized by difficulty in speech, pauses, and vague expressions, along with intermittent speech interruptions and incoherence. The patient exhibited motor weakness in the extremities, without loss of consciousness, incontinence, or convulsions. Vital signs at admission were as follows: temperature 37.9°C, pulse 98 beats per minute, blood pressure 119/84 mm Hg, and SpO2 96%. On physical examination, the Glasgow Coma Scale (GCS) score was assessed at 10, with the patient appearing drowsy, showing poor engagement but responding to call; speech was non-fluent, and both calculation and memory were impaired. Cooperation during the examination was limited, and the follow-through of verbal commands was poor. Ocular movements were sluggish, and neck stiffness was noted with some resistance. Both pupils were equal and round, measuring 2 mm, with a sluggish light reflex. Muscle strength in the upper extremities was rated at 3/5, while lower extremity strength was 4/5; muscle tone was normal bilaterally, and a pathological reflex was absent. The abdomen was soft but showed tenderness in the right upper quadrant, with positive rebound tenderness and Murphy's sign(+). Bowel sounds were normal, and coordination during finger-nose and finger-finger tests was slow and poorly coordinated. The heel-knee-shin test demonstrated poor stability, and superficial, deep, and composite sensations in the extremities were diminished.

The patient had a significant past medical history, having been diagnosed with schizophrenia for 30 years, and had a history of irregular medication adherence, including chlorpromazine and perphenazine. On the Brunnstrom scale, the left upper extremity and hand were at stage III, and the lower extremity at stage IV; the right upper extremity and hand were also at stage III, with the lower extremity at stage IV. Joint range of motion was normal, but sensory function was significantly impaired. The patient was assessed with a sitting balance of level 3, unable to maintain standing balance, and showed poor balance performance, resulting in a mobility score of 0, indicating total dependency in activities of daily living.

Auxiliary examinations revealed elevated laboratory parameters: WBC $13.8 \times 10^9/L \uparrow$, neutrophil percentage 86.3% $\uparrow$. Notably, CRP was elevated at 7.7 mg/L, procalcitonin at 0.32 ng/ml $\uparrow$, interleukin-6 at 7.6 pg/ml $\uparrow$, and liver function tests showed elevated alanine aminotransferase at 368.8 U/L $\uparrow$ and

aspartate aminotransferase at 77.7 U/L ↑, along with total bilirubin at 27.1 μmol/L ↑ and direct bilirubin at 10.9 μmol/L. Cerebrospinal fluid analysis revealed an elevated total cell count of $1.0 \times 10^9/L$, total protein of 0.6 g/L, and glucose of 4.54 mmol/L. Enhanced abdominal CT showed thickening of the gallbladder wall with no significant positive intraluminal stones, indicating cholecystitis. Cranial MRI demonstrated symmetrical abnormal signals in the dorsal brainstem, periaqueductal region, bilateral thalami, and bilateral parietal lobes (Fig. 1). Based on the clinical history, physical examination, and imaging, a preliminary diagnosis of non-alcoholic Wernicke's encephalopathy was considered. The patient was treated with intravenous thiamine hydrochloride (500 mg) dissolved in 100 mL of normal saline, administered every 8 hours for 3 days, in conjunction with oral vitamin B complex (three times daily) and mecobalamin (0.5 mg three times daily). The thiamine dosage was subsequently adjusted to 250 mg for 5 days, followed by 100 mg for another 5 days. Rehabilitation therapy was initiated, which included comprehensive training for the affected limbs, isokinetic muscle strength training, and low-frequency pulse therapy targeting both quadriceps and tibialis anterior muscle groups. This rehabilitation aimed to enhance the recovery of motor functions, increase movement independence of the affected limbs, improve postural transition capabilities, and promote walking balance. Electrical standing training was employed to aid lower limb muscle strength recovery and joint range of motion. After 14 days of integrated treatment, there was a marked improvement in the patient's consciousness, with relevant responses and nearly complete resolution of dysarthria. Significant relief in memory and orientation was observed, and motor function of the extremities showed substantial enhancement, with improved body and limb coordination. Follow-up MRI revealed a significant reduction in the previously noted abnormal signal areas, particularly in the dorsal brainstem, periaqueductal region, third ventricle, and bilateral thalami (Fig. 2). Following discharge, the patient adhered to a regimen of oral thiamine (100 mg three times daily), vitamin B complex (three times daily), and mecobalamin (0.5 mg three times daily). A two-month follow-up via telephone revealed no significant discomfort.

3. Discussion

A typical case of WE has been reported. The patient initially presented with altered consciousness and limb weakness, accompanied by hyperintense lesions observed on brain MRI (FLAIR). The emergence of additional symptoms, along with characteristic neuroimaging findings, ultimately led to a diagnosis of WE. Treatment involved intravenous administration of thiamine, and both clinical and radiographic abnormalities were reversed following cognitive and strength rehabilitation exercises.

The pathogenesis of WE is primarily associated with thiamine (vitamin B1) deficiency and its crucial role in energy metabolism. Thiamine is a water-soluble vitamin that cannot be synthesized by the human body and is predominantly obtained from food. It is absorbed in the duodenum and stored in the liver, with an in-body half-life of approximately 18 days. The recommended daily intake of thiamine for a healthy adult is 1.4 mg, or 0.5 mg for every 1000 kcal consumed. The body's thiamine reserves typically range from 30 to 50 mg, and without dietary intake, these stores can be depleted within 4 to 6 weeks^[4, 6-7]. Thiamine functions as an essential cofactor in the citric acid cycle and the pentose phosphate

pathway; therefore, deficiency results in decreased production of pyruvate and α -ketoglutarate, leading to the accumulation of lactate and alanine within cells and intracellular accumulation of glutamate^[8]. This accumulation restricts ATP-dependent cellular pump functions, causing decreased intracellular pH, electrolyte imbalances, and subsequently results in toxic edema, functional impairment, and cell injury^[9–12]. Consequently, thiamine deficiency typically affects regions of the brain with high metabolic activity. The optimal functioning of the brain critically depends on the maintenance of glucose metabolism and thiamine levels, and damage is initially reversible following thiamine supplementation. However, delayed thiamine administration may result in structural and irreversible brain lesions, potentially accompanied by permanent neurological deficits or fatal outcomes^[12]. This process not only affects the central nervous system but may also exacerbate various metabolic-related symptoms, leading to multi-organ damage. Therefore, ensuring adequate thiamine intake is vital for the prevention and treatment of WE.

WE is generally localized to brain structures that are most vulnerable to thiamine deficiency, including the medial thalamus, mammillary bodies, periaqueductal gray matter, and paraventricular areas, which display higher concentrations of vitamin B1. The clinical manifestations of WE are thus associated with these susceptible brain regions, leading to highly variable and nonspecific presentations^[3,13]. Only 16% of patients exhibit the classic triad of symptoms (altered mental status, ataxia, and ophthalmoplegia). The most common symptom is altered mental status, occurring in 76–80% of cases^[2, 14, 15]. Although the clinical features of WE are relatively well-defined, its incidence varies widely across different populations and geographic regions. Particularly in atypical cases, the clinical diagnosis of WE can be challenging due to the absence of biomarkers to confirm the diagnosis or quantify the severity of the illness. Furthermore, the relationship between plasma thiamine levels associated with the etiology and the diagnosis, severity, and prognosis of WE remains unclear, resulting in lower rates of definitive diagnosis. Currently, a high degree of clinical suspicion and recognition of predisposing factors are critical for diagnosing WE. In 1997, Caine proposed diagnostic criteria that include: 1) inadequate food intake; 2) abnormalities of ocular movements; 3) ataxia; and 4) changes in mental status or mild memory impairment^[16]. The presence of two or more of these criteria warrants consideration of WE. Additionally, neuroimaging often reveals characteristic symmetric abnormal signal changes, a favorable response to thiamine treatment, and differentiation from clinical manifestations induced by central nervous system injuries such as stroke or brain tumors is necessary^[16–18].

The patient exhibited the classic triad of clinical symptoms, leading to a presumptive clinical diagnosis of WE based on specific imaging findings. Timely thiamine supplementation constitutes the primary treatment approach for WE. Parenteral administration via intravenous or intramuscular routes is considered most effective, while oral administration is not recommended. For outpatient treatment, intramuscular injection is preferred, whereas intravenous administration is advised for hospitalized patients. A suggested thiamine treatment regimen includes intravenous infusion of 500 mg three times daily for 3 to 5 days, followed by 250 mg once daily for an additional 3 to 5 days, until clinical improvement is fully achieved^[3,12,19]. Preventive administration of thiamine is recommended for individuals with alcohol withdrawal, malnutrition, or signs of nutritional deficiency^[3,12,20]. Thiamine

supplementation can improve symptoms in most patients; however, early personalized rehabilitation interventions should be initiated for those with significant cognitive and strength impairments, focusing primarily on cognitive deficits, gait, and balance. Enhanced rehabilitation programs, monitored through neuropsychological assessment pre- and post-rehabilitation, are expected to boost patients' confidence in daily activities. Outcome measures from cognitive assessments, memory tests, and functional independence scales have demonstrated significant improvement post-treatment, underscoring the effectiveness of implemented rehabilitation strategies^[21]. Tailored rehabilitation strategies for the patient, addressing specific deficits, included cognitive exercises, gait and balance training, comprehensive training for hemiplegic limbs, isokinetic strength training, low-frequency pulse therapy, and addressing emotional concerns, which facilitated the recovery of motor functions and enhanced limb mobility. Balance training improved postural transfer abilities and promoted walking stability. Electrical standing training aided in the restoration of lower limb strength and expanded the range of joint motion.

The patient's acute cholecystitis, combined with fasting and poor appetite, led to insufficient thiamine intake. Additionally, the infection from cholecystitis resulted in an immunocompromised state, causing an inflammatory condition that depleted body thiamine reserves and produced nystagmus, altered mental status, and emotional instability. Early manifestations included altered consciousness, speech confusion, and gait instability, with cranial MRI revealing hyperintense changes in the parietal lobes, bilateral thalami, periaqueductal gray, and dorsal aspect of the brainstem, culminating in a diagnosis of Wernicke's Encephalopathy. In terms of treatment, thiamine replacement therapy is the cornerstone for patients with WE, initiated with intravenous thiamine infusion for rapid replenishment of the deficient vitamin, followed by a transition to oral supplementation. Concurrent with pharmacological treatment, rehabilitation exercise should not be overlooked. Rehabilitation aims to enhance the patient's capacity for daily living and maximize the restoration of compromised motor and cognitive functions. Appropriate rehabilitative interventions can significantly improve the patient's quality of life, accelerate recovery, and reduce the risk of long-term disability. After comprehensive treatment, the patient achieved a full recovery of symptoms, with follow-up MRI indicating resolution of lesions, confirming the effectiveness of the treatment prior to discharge.

4. Conclusions

A case of WE caused by thiamine deficiency secondary to cholecystitis has been reported. The clinical manifestations of WE are complex, and diagnosis is often challenging and delayed. Therefore, it is essential for clinicians to be familiar with the predisposing factors and maintain a high level of clinical suspicion. Empirical thiamine supplementation should also be administered to malnourished patients to prevent the occurrence of Wernicke's Encephalopathy. The management of Wernicke's Encephalopathy should include a comprehensive, individualized treatment plan that incorporates both pharmacological therapy and functional rehabilitation, as these are critical components for improving patient outcomes and quality of life.

Declarations

Funding

This work was funded by the Ningxia Natural Science Foundation (grant numbers 2025AAC031002, 2026AAC030439 and 2026AAC030617), the 2024 Autonomous Region Youth Science and Technology Talent Training Project [Ningxia Association for Science and Technology (2025) No. 2] and Shizuishan City Research Team for Tumor Molecular Pathological Mechanisms and Translational Innovation [Notice on the Identification of Municipal Scientific and Technological Innovation Teams for 2026 (Shike Fa [2026] No.15)].

Author contribution statement

Li Jing, Fangjing Xu, Yucheng Fan and Yongjun Du were actively involved in the conception and design of the study, the acquisition of data, and the analysis and interpretation of the results. F.J., Y.C., H.H., P.W., X.W., T.M. and J.M. contributed to the drafting of the manuscript and participated in the critical revision of the manuscript, ensuring its intellectual rigor. All authors had full access to the study data, collaborated throughout the research process, approved the final version of the manuscript for publication, and assume responsibility for the accuracy and integrity of the work.

Conflicts of interest statement

The authors declare no competing interests.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The patient was informed that anonymization could not be fully guaranteed and that the material may be publicly available online. A copy of the signed consent form is retained by the corresponding author and is available for review by the journal editorial team upon request.

Clinical trial number

Not applicable.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics approval and consent to participate

This case report was reviewed and approved by the Ethics Committee of the 942nd Hospital of PLA Joint Logistics Support Force and the Ethics Committee of the First People's Hospital of Shizuishan, affiliated to Ningxia Medical University (approval number: 2025AAC031002 and 2026AAC030439). All

procedures performed were in accordance with the ethical standards of the institutional research committees and with the 1964 Declaration of Helsinki and its later amendments. Written informed consent for the publication of potentially identifying information and images was freely given by the patient prior to submission.

References

1. Xu J, Li F, Xue Y. Acute hearing and visual loss caused by thiamine deficiency. *BMC Neurol.* 2023;23(1):285. PMID: 37525097; PMCID: PMC10388443.
2. Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. *J Neurol Neurosurg Psychiatry.* 1986;49(4):341–5. PMID: 3701343; PMCID: PMC1028756.
3. Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. *Lancet Neurol.* 2007;6(5):442–55. PMID: 17434099.
4. Zuccoli G, Pipitone N. Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. *AJR Am J Roentgenol.* 2009;192(2):501-8. PMID: 19155417.
5. Tsao WC, Ro LS, Chen CM, Chang HS, Kuo HC. Correction to: Non-alcoholic Wernicke's encephalopathy with cortical involvement and polyneuropathy following gastrectomy. *Metab Brain Dis.* 2017;32(6):2163. Erratum for: *Metab Brain Dis.* 2017;32(5):1649–1657. PMID: 29052074.
6. Kim TE, Lee EJ, Young JB, Shin DJ, Kim JH. Wernicke encephalopathy and ethanol-related syndromes. *Semin Ultrasound CT MR.* 2014;35(2):85–96. PMID: 24745886.
7. Gangolf M, Czerniecki J, Radermecker M, Detry O, Nisolle M, Jouan C, Martin D, Chantraine F, Lakaye B, Wins P, Grisar T, Bettendorff L. Thiamine status in humans and content of phosphorylated thiamine derivatives in biopsies and cultured cells. *PLoS ONE.* 2010;5(10):e13616. PMID: 21049048; PMCID: PMC2963613.
8. Abdul-Muneer PM, Alikunju S, Schuetz H, Szlachetka AM, Ma X, Haorah J. Impairment of Thiamine Transport at the GUT-BBB-AXIS Contributes to Wernicke's Encephalopathy. *Mol Neurobiol.* 2018;55(7):5937–50. Epub 2017 Nov 11. PMID: 29128903; PMCID: PMC9420083.
9. Abdou E, Hazell AS. Thiamine deficiency: an update of pathophysiologic mechanisms and future therapeutic considerations. *Neurochem Res.* 2015;40(2):353–61. Epub 2014 Oct 9. PMID: 25297573.
10. Chandrakumar A, Bhardwaj A, 't Jong GW. Review of thiamine deficiency disorders: Wernicke encephalopathy and Korsakoff psychosis. *J Basic Clin Physiol Pharmacol.* 2018;30(2):153–62. PMID: 30281514.
11. Sinha S, Kataria A, Kolla BP, Thusius N, Loukianova LL. Wernicke Encephalopathy-Clinical Pearls. *Mayo Clin Proc.* 2019;94(6):1065–1072. PMID: 31171116.
12. Tao X, Qiao R, Liu C, Guo L, Li J, Kang Y, Wei Y. Wernicke encephalopathy induced by glucose infusion: A case report and literature review. *Heliyon.* 2024;10(8):e29419. PMID: 38681648; PMCID:

PMC11053267.

13. Unlu E, Cakir B, Asil T. MRI findings of Wernicke encephalopathy revisited due to hunger strike. *Eur J Radiol.* 2006;57(1):43–53. Epub 2005 Aug 8. PMID: 16085381.
14. Zahr NM, Kaufman KL, Harper CG. Clinical and pathological features of alcohol-related brain damage. *Nat Rev Neurol.* 2011;7(5):284–94. Epub 2011 Apr 12. PMID: 21487421; PMCID: PMC8121189.
15. Zahr NM, Kaufman KL, Harper CG. Clinical and pathological features of alcohol-related brain damage. *Nat Rev Neurol.* 2011;7(5):284–94. Epub 2011 Apr 12. PMID: 21487421; PMCID: PMC8121189.
16. Caine D, Halliday GM, Kril JJ, Harper CG. Operational criteria for the classification of chronic alcoholics: identification of Wernicke's encephalopathy. *J Neurol Neurosurg Psychiatry.* 1997;62(1):51–60. PMID: 9010400; PMCID: PMC486695.
17. Cerase A, Rubenni E, Rufa A, Vallone I, Galluzzi P, Coratti G, Franchi F, Giannini F, Venturi C. CT and MRI of Wernicke's encephalopathy. *Radiol Med.* 2011;116(2):319–33. English, Italian. Epub 2011 Jan 12. PMID: 21225366.
18. Infante MT, Fancellu R, Murialdo A, Barletta L, Castellan L, Serrati C. Challenges in Diagnosis and Treatment of Wernicke Encephalopathy: Report of 2 Cases. *Nutr Clin Pract.* 2016;31(2):186 – 90. Epub 2016 Feb 11. PMID: 26869612.
19. Boulanger AS, Paquette I, Létourneau G, Richard-Devantoy S. Thiamine et encéphalopathie de Gayet-Wernicke: quelles règles de prescription ? [Wernicke encephalopathy: Guiding thiamine prescription]. *Encephale.* 2017;43(3):259–67. French. Epub 2016 Sep 9. PMID: 27623120.
20. Ota Y, Capizzano AA, Moritani T, Naganawa S, Kurokawa R, Srinivasan A. Comprehensive review of Wernicke encephalopathy: pathophysiology, clinical symptoms and imaging findings. *Jpn J Radiol.* 2020;38(9):809–20. Epub 2020 May 10. PMID: 32390125.
21. Burile GC, Arya N, Seth NH, Fating T. Rehabilitation Strategies for Wernicke-Korsakoff Syndrome: Physiotherapy Interventions and Management Approaches. *Cureus.* 2024;16(2):e53948. PMID: 38469004; PMCID: PMC10926318.

Figures

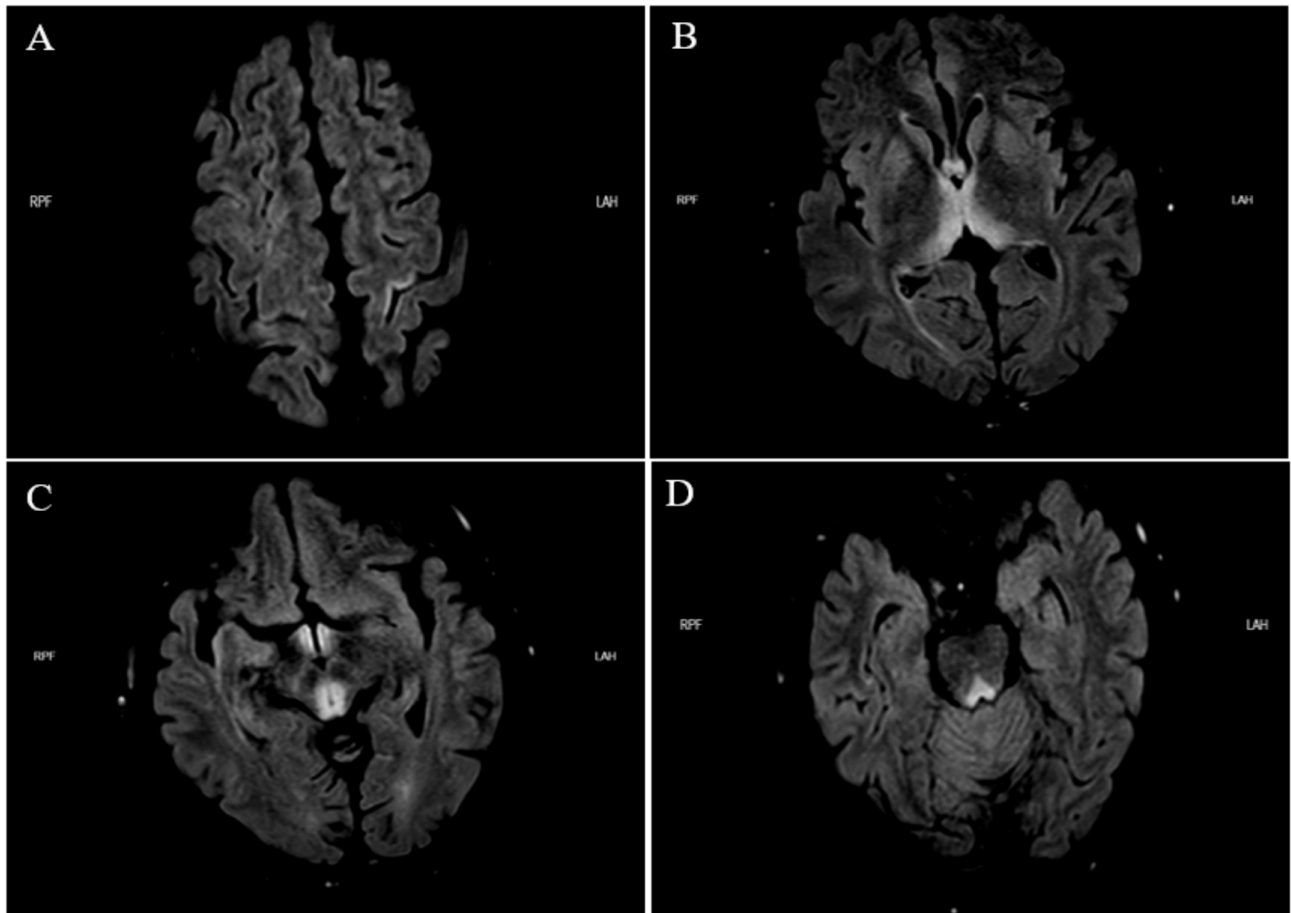


Figure 1

Axial images from brain magnetic resonance imaging (MRI) using the T2 Fluid-Attenuated Inversion Recovery (FLAIR) sequence. (A) The T2 FLAIR axial image demonstrates symmetrical hyperintensity in the bilateral parietal lobes. (B) The T2 FLAIR axial image shows symmetrical hyperintensity surrounding the bilateral dorsomedial thalami. (C) The T2 FLAIR axial image reveals symmetrical hyperintensity around the cerebral aqueduct. (D) The T2 FLAIR axial image presents symmetrical hyperintensity in the dorsal aspect of the brainstem.

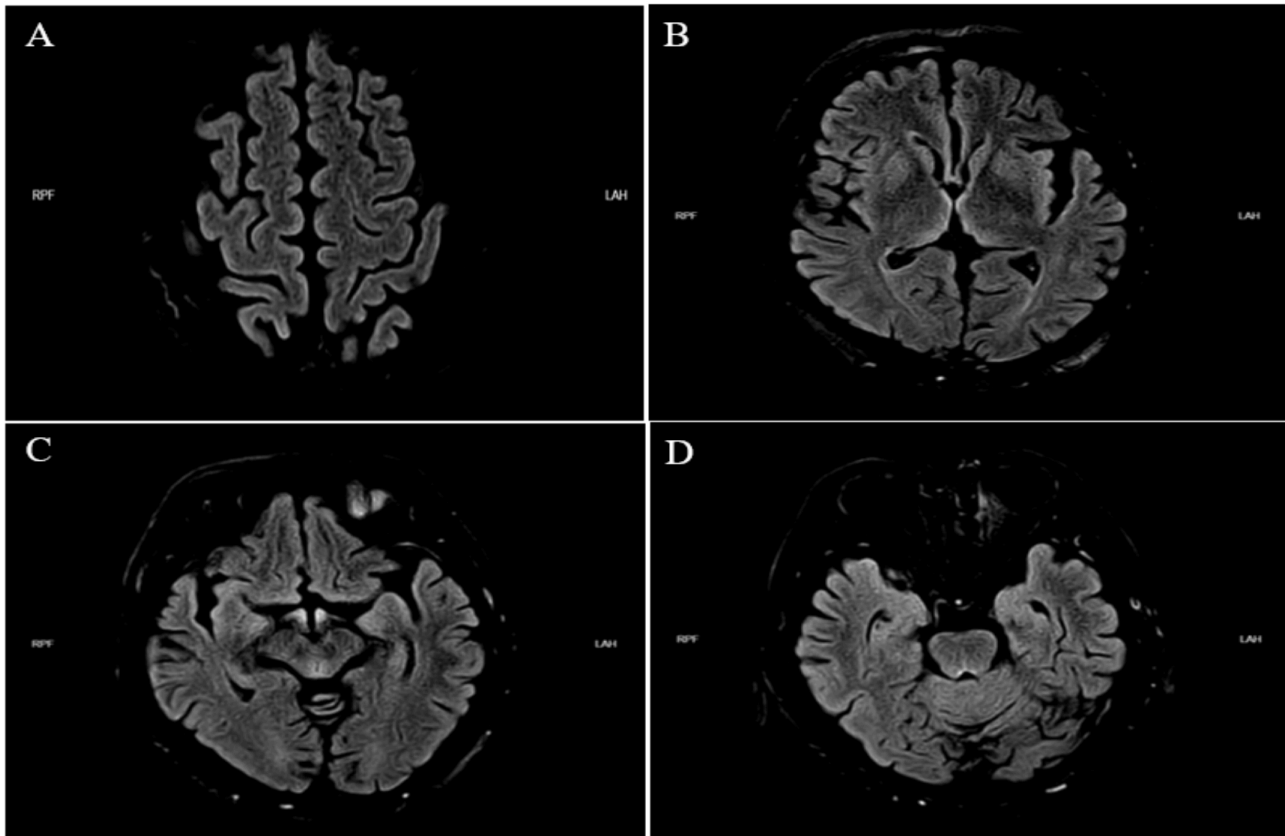


Figure 2

Axial images from brain magnetic resonance imaging (MRI) using the T2 Fluid-Attenuated Inversion Recovery (FLAIR) sequence, obtained two weeks after comprehensive treatment. (A) The T2 FLAIR axial image demonstrates symmetrical hyperintensity in the bilateral parietal lobes. (B) The T2 FLAIR axial image reveals symmetrical hyperintensity surrounding the bilateral dorsomedial thalami. © The T2 FLAIR axial image shows symmetrical hyperintensity around the cerebral aqueduct. (D) The T2 FLAIR axial image presents symmetrical hyperintensity in the dorsal aspect of the brainstem.