ECRONICON OPEN ACCESS

EC CANCER
Case Report

Atypical Wernicke Encephalopathy with Hemorrhagic Pontine Involvement and Extensive Cortical Lesions: A Fatal Case Report

Basma Dghoughi[1]*, Kaoutar Maslouhi[1], Basma Beqqali[1], Eric Michel Kessi[1], Zaynab Iraqi Houssaini[1], Ola Messaoud[1], Omar El Aoufir[1], Laila Jroundi[1], Larbi Aberouch[2], Salim Chajai[2], Jawad Tadili[2], Mohamed Ali Echcherif El Kettani[2] and Mamoun Faroudy[2]

[1]*Emergency Radiology Department, Ibn Sina University Hospital, University Mohamed V Souissi, Rabat, Morocco*

[2]*Intensive Care Unit, Ibn Sina University Hospital, University Mohamed V Souissi, Rabat, Morocco*

***Corresponding Author:** Basma Dghoughi, Emergency Radiology Department, Ibn Sina University Hospital, University Mohamed V Souissi, Rabat, Morocco.

Received: October 17, 2025; **Published:** October 30, 2025

Abstract

Wernicke encephalopathy (WE) is a neurologic emergency resulting from thiamine deficiency, typically associated with chronic alcoholism. Standard MRI findings include lesions in the thalami, mammillary bodies, and periaqueductal gray matter; however, atypical manifestations such as cortical and hemorrhagic lesions are uncommon and underrecognized. We report a 55-year-old male with chronic alcohol use disorder admitted to ICU following traumatic brain injury. Initial CT was unremarkable, but persistent altered mental status prompted MRI, revealing diffusion and FLAIR hyperintensities in the mammillary bodies, periaqueductal region, cortex, and pons. Susceptibility-weighted imaging indicated hemorrhagic transformation in the pons. Markedly reduced thiamine levels confirmed WE. Despite intravenous thiamine administration, the patient rapidly deteriorated and died within 48 hours. This case highlights a rare presentation of WE with diffuse cortical involvement and pontine hemorrhage, signaling advanced disease and poor prognosis. Early identification of atypical imaging features is crucial to prevent treatment delays and reduce morbidity and mortality.

Keywords: *Wernicke Encephalopathy; Thiamine Deficiency; MRI; Cortical Involvement; Hemorrhagic Pons; SWI; Alcohol Use Disorder*

Introduction

Wernicke encephalopathy (WE) is a potentially reversible but often underdiagnosed neurologic emergency resulting from thiamine (vitamin B1) deficiency. Although classically associated with chronic alcoholism, it can also arise in various clinical settings, including malnutrition, hyperemesis gravidarum, malignancy, and postoperative states. The classic clinical triad-ophthalmoplegia, ataxia, and altered mental status-is present in fewer than 20% of cases, contributing to the diagnostic challenge [1].

Neuroimaging, particularly magnetic resonance imaging (MRI), plays a pivotal role in the diagnosis of WE, often revealing symmetrical hyperintensities on FLAIR and diffusion-weighted imaging in characteristic regions such as the medial thalami, mammillary bodies, and periaqueductal region. However, atypical imaging findings may occur, including cortical involvement, cerebellar abnormalities, and in rare

cases, hemorrhagic lesions. Such atypical presentations can lead to diagnostic delay, especially when the clinical context is confounded by other acute pathologies such as trauma.

We report a rare and fatal case of Wernicke encephalopathy in a 55-year-old male with chronic alcohol use disorder, presenting with altered consciousness following head trauma. MRI revealed not only the classic signs of WE but also extensive bilateral cortical involvement and a hemorrhagic pontine lesion—an unusual and severe manifestation of the disease. This case highlights the importance of recognizing atypical imaging features of WE, particularly in critically ill patients, and underscores the role of MRI in guiding timely diagnosis and treatment.

Case Presentation

A 55-year-old male with a history of chronic alcohol use disorder and social vulnerability (homelessness) was admitted to the emergency department following a road traffic accident. On initial assessment, he was found to be malnourished, with a body mass index (BMI) of approximately 17 kg/m^2 and exhibited an altered level of consciousness with a Glasgow Coma Scale (GCS) score of 10 (E3V2M5). There were no focal neurological deficits or signs of hepatic encephalopathy. Initial non-contrast head computed tomography (CT) demonstrated bilateral hygromas without associated mass effect or parenchymal lesions in the supratentorial or infratentorial compartments (Figure 1).

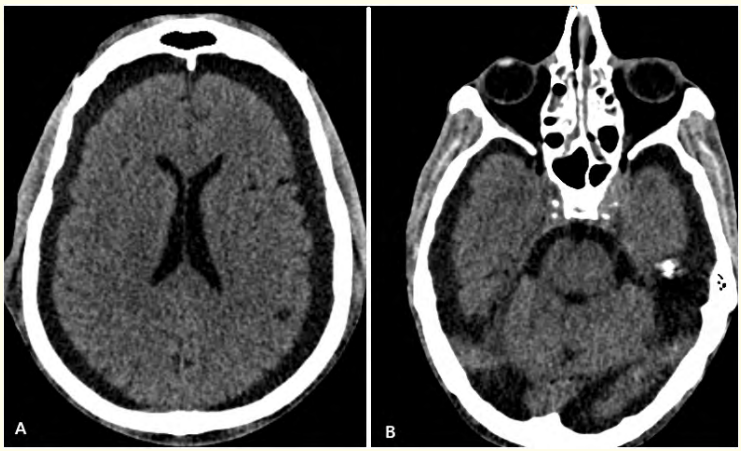

Figure 1: *Initial CT scan of the patient showing bilateral hygroma without any cortical involvement (A) nor pontine lesions (B).*

The patient was admitted to the intensive care unit (ICU) for neurological monitoring. Due to progressive worsening of consciousness (GCS dropped to 8), he was intubated and sedated. Over the next three days, despite stable hemodynamics and resolution of sedation, his consciousness did not improve. Given the persistent encephalopathy, a brain MRI was obtained one week post-admission to evaluate for possible traumatic vascular injury specifically arterial dissection or post-traumatic infarction.

MRI was performed on a 1.5 Tesla system using axial T1-weighted with fat suppression as typical arterial dissection sequence, coronal T2-weighted, axial FLAIR, diffusion-weighted imaging (DWI), susceptibility-weighted imaging (SWI), 3D time-of-flight (TOF)

MR angiography sequences and 3D T1 MPRAGE with contrast injection. Due to the onset of patient agitation during the examination, certain sequences were acquired using echo-planar imaging to reduce acquisition time. The imaging revealed bilateral DWI and FLAIR hyperintensities in the mammillary bodies, periaqueductal gray matter, and diffuse symmetrical involvement of the supratentorial cortex (Figure 2). The pontine involvement appeared as a diffuse hyperintensity on T2 and DWI, hypointensity on T1 with associated hypointensity on SWI with slight enlargement, suggestive of hemorrhagic transformation in its super acute phase (0-6 hours) (Figure 3).

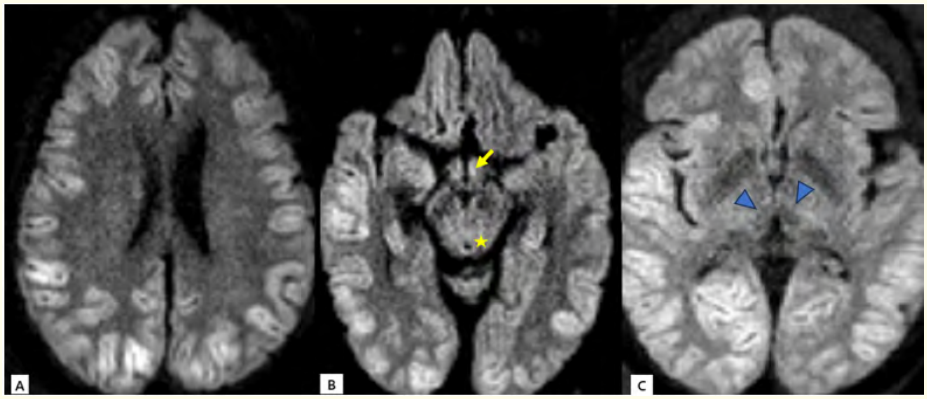

Figure 2: *Axial DWI images showing hyperintensity in the cortex (A), mamillary bodies (arrow in B) and periaqueductal region (star in B), without signal abnormalities in the thalamic region (C).*

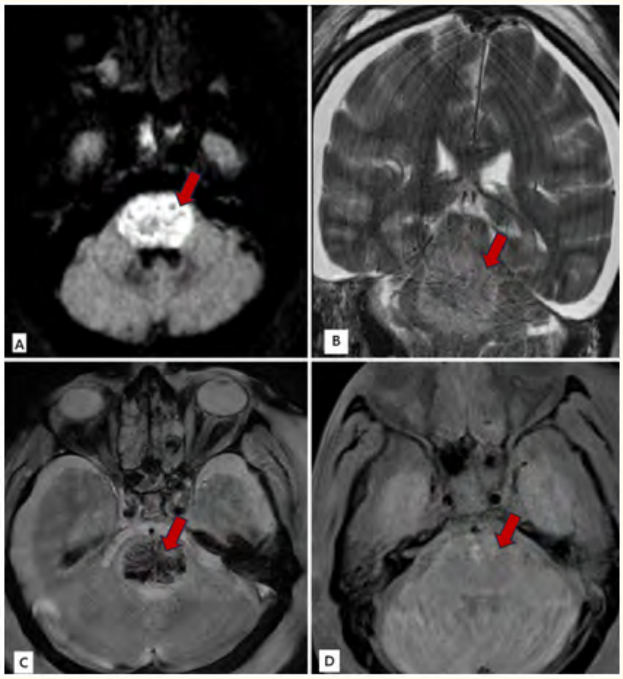

Figure 3: *MRI imaging of the pontine involvement showing high signal on DWI (A) and T2 (B), and low signal on SWI (C) and T1 (D).*

Given the pattern of signal abnormalities, particularly the involvement of the mammillary bodies and periaqueductal gray matter, a metabolic encephalopathy was suspected, with Wernicke encephalopathy (WE) considered the leading hypothesis despite the unusual distribution and severity and the absence of thalamic involvement. Differential diagnoses such as hypoglycemic encephalopathy, hepatic encephalopathy, and post-anoxic injury were considered but deemed less likely based on the imaging and clinical context.

Liver and renal function tests, electrolytes, glucose, and complete blood count were within normal limits. Critically, laboratory workup revealed a severely decreased serum thiamine level of 9 µg/L (reference range: 28-85 µg/L), confirming profound thiamine deficiency.

Empirical intravenous thiamine replacement was immediately initiated upon receiving the laboratory results. The patient received high-dose parenteral thiamine, administered as 500 mg IV three times daily, in accordance with established clinical guidelines. Adjunctive magnesium and multivitamin supplementation were also provided to optimize thiamine utilization. Despite treatment, the extent of cerebral injury, particularly the hemorrhagic brainstem involvement and diffuse cortical damage, was deemed irreversible. The patient exhibited no neurological improvement and passed away 48 hours after the initiation of thiamine therapy.

Discussion

Wernicke encephalopathy (WE) is a serious but potentially reversible neurological disorder caused by thiamine (vitamin B1) deficiency. While alcoholism remains the most well-known risk factor, WE can also occur in non-alcoholic individuals due to malnutrition, gastrointestinal surgery, hyperemesis gravidarum, or prolonged vomiting or fasting [1-3]. The classic clinical triad of encephalopathy, oculomotor abnormalities, and ataxia is observed in only 16-33% of patients, leading to frequent underdiagnosis, particularly in complex clinical settings such as trauma or critical illness [4-6].

Magnetic resonance imaging (MRI) is a key diagnostic tool, typically revealing symmetrical hyperintensities in the mammillary bodies, medial thalami, periaqueductal gray matter, and floor of the fourth ventricle on T2-weighted and FLAIR sequences [7]. However, atypical MRI findings have been increasingly recognized, including involvement of the cerebellum, cranial nerve nuclei, basal ganglia, and cerebral cortex [8,9]. Such atypical presentations are more frequently reported in non-alcoholic patients and are associated with more severe disease and poorer outcomes [10].

Our case is notable for two rare and clinically significant features: cortical involvement and pontine hemorrhage. Cortical signal abnormalities, particularly in the fronto-parieto-occipital regions, are uncommon in WE and are often associated with cytotoxic edema and a high risk of irreversible damage such as cortical laminar necrosis [11]. In the case described by Pereira., *et al*. cortical laminar necrosis and SWI-detected hemorrhagic deposits were seen in a non-alcoholic WE patient, correlating with poor neurological recovery [11].

Hemorrhagic changes in WE are rare but have been described in a limited number of reports. They are believed to result from severe disruption of the blood-brain barrier and endothelial injury secondary to oxidative stress, mitochondrial dysfunction, and impaired energy metabolism [12]. SWI imaging is highly sensitive for detecting such microhemorrhages, as demonstrated in our case by the hypointense signal in the pons. A similar presentation with pontine and cortical hemorrhagic features has been reported by Ouchen., *et al*. is associated with a fatal outcome [6].

Biochemical confirmation of thiamine deficiency is often pursued to support the diagnosis, though testing presents several limitations. Plasma thiamine levels or thiamine pyrophosphate (TPP) in whole blood are the most commonly used biomarkers. However, these tests are not universally available, may be subject to delayed turnaround times, and are not always reflective of tissue-level thiamine stores [13,14]. Moreover, normal thiamine levels do not exclude the diagnosis, as illustrated in the case by El Berhoumi., *et al.* where WE was diagnosed based on imaging and clinical context despite normal thiamine levels [8].

Thiamine replacement therapy is the cornerstone of treatment for WE and should be initiated immediately and empirically upon clinical suspicion-ideally before neuroimaging or laboratory confirmation. High-dose parenteral thiamine (500 mg IV TID for 2 - 3 days, followed by 250 mg daily for 3 - 5 days) is currently the recommended regimen [5,15-17]. Oral supplementation is inadequate in acute cases due to impaired absorption. Concomitant magnesium should also be administered, as it is a necessary cofactor in thiamine-dependent enzymatic activity [5].

Despite prompt initiation of thiamine in our patient following MRI and biochemical confirmation, the disease course progressed rapidly, leading to death within 48 hours. This outcome underscores the poor prognosis associated with extensive cortical involvement and hemorrhagic changes, and highlights the necessity of early diagnosis and empirical treatment in at-risk populations [6,10,11].

Conclusion

This case highlights a rare and severe manifestation of Wernicke encephalopathy, characterized by extensive bilateral cortical involvement and hemorrhagic changes within the pons without thalamic abnormality. These atypical MRI features, while uncommon, are critical for radiologists and clinicians to recognize, particularly in patients with chronic alcohol use, malnutrition, or unexplained encephalopathy. Prompt diagnosis is essential, as delayed treatment is associated with irreversible neurological injury and high mortality. Empirical initiation of high-dose parenteral thiamine remains the cornerstone of management. This report underscores the importance of maintaining a high index of suspicion for Wernicke encephalopathy in critically ill or neurologically deteriorating patients, even in the presence of competing diagnoses such as trauma.

Acknowledgements

I would like to express my gratitude for all my professors and colleagues for their help in the accomplishment of this work.

Conflict of Interest

The authors declare no conflict of interest.

Funding Support

The authors received no fundings for this work.

Ethical Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Guarantor

Dr Basma Dghoughi.

Conflict of Interest

The authors declare having no competing interest.

Consent

A written consent was obtained from the patient's family for the anonymous use of this data.

Bibliography

1. Ota Y., *et al*. "Comprehensive review of Wernicke encephalopathy: pathophysiology, clinical symptoms and imaging findings". *Japanese Journal of Radiology* 38.9 (2020): 809-820.

2. Habas E., *et al*. "Wernicke encephalopathy: an updated narrative review". *Saudi Journal of Medicine and Medical Sciences* 11.3 (2023): 193-200.

3. Kohnke S and Meek CL. "Don't seek, don't find: The diagnostic challenge of Wernicke's encephalopathy". *Annals of Clinical Biochemistry* 58.1 (2020): 38-46.

4. Isen DR and Kline LB. "Neuro-ophthalmic manifestations of Wernicke encephalopathy". *Eye and Brain* 12 (2020): 49-60.

5. Chandrakumar A., *et al*. "Review of thiamine deficiency disorders: Wernicke encephalopathy and Korsakoff psychosis". *Journal of Basic and Clinical Physiology and Pharmacology* 30.2 (2018): 153-162.

6. Ouchen K., *et al*. "Intracerebral hematoma secondary to Wernicke's encephalopathy: A case report with a review of literature". *Cureus* 17.2 (2025): e78330.

7. Zuccoli G and Pipitone N. "Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature". *American Journal of Roentgenology* 192.2 (2009): 501-508.

8. Berhoumi AE., *et al*. "[Gayet-Wernicke encephalopathy with normal thiamine level: a case report]". *Pan African Medical Journal* 38 (2021): 16.

9. Bouchal S., *et al*. "L'encéphalopathie de Gayet Wernicke: aspects Cliniques et anomalies radiologiques". *Pan African Medical Journal* 36 (2020): 259.

10. Benzalim M., *et al*. "Gayet Wernicke's encephalopathy with cortical damage following a subtotal gastrectomy: An uncommon association". *Radiology Case Reports* 16.1 (2020): 94-97.

11. Pereira DB., *et al*. "Nonalcoholic Wernicke encephalopathy with extensive cortical involvement: cortical laminar necrosis and hemorrhage demonstrated with susceptibility-weighted MR phase images". *American Journal of Neuroradiology* 32.2 (2011): E37-E38.

12. Zuccoli G., *et al*. "Wernicke encephalopathy: MR findings at clinical presentation in twenty-six alcoholic and nonalcoholic patients". *American Journal of Neuroradiology* 28.7 (2007): 1328-1331.

13. Attaluri P., *et al*. "Thiamine deficiency: An important consideration in critically ill patients". *American Journal of the Medical Sciences* 356.4 (2018): 382-390.

14. Whitfield KC., *et al*. "Thiamine deficiency disorders: diagnosis, prevalence, and a roadmap for global control programs". *Annals of the New York Academy of Sciences* 1430.1 (2018): 3-43.

15. Sechi G and Serra A. "Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management". *Lancet Neurology* 6.5 (2007): 442-455.

16. Oudman E., *et al*. "Preventing Wernicke's encephalopathy in anorexia nervosa: A systematic review". *Psychiatry and Clinical Neurosciences* 72.10 (2018): 774-779.

17. Galvin R., *et al*. "EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy". *European Journal of Neurology* 17.12 (2010): 1408-1418.

Volume 3 Issue 1 January 2025