Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology

Wernicke Encephalopathy Associated with Cortical Laminar Necrosis: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i11.062 | **Received:** 17.09.2026 | **Accepted:** 14.11.2026 | **Published:** 17.11.2026

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Abstract | **Case Report**

Wernicke encephalopathy (WE) is an acute neurological disorder caused by thiamine deficiency, most commonly associated with chronic alcohol use. Although magnetic resonance imaging (MRI) is the preferred imaging modality for detecting characteristic lesions, computed tomography (CT) may reveal additional abnormalities in patients presenting with neurological deterioration. We report the case of a 73-year-old patient with a history of chronic alcohol use who initially presented with behavioral disturbances and underwent brain MRI, which supported the diagnosis of WE in conjunction with clinical and laboratory findings. The patient subsequently developed altered consciousness and was admitted to the emergency department. Brain CT revealed bilateral, symmetrical hypoattenuating abnormalities involving the posterior portions of the external capsules and extending to the cerebral peduncles. Additional findings included diffuse gyriform hyperattenuation of the supratentorial cortex and bilateral parieto-temporo-occipital subcortical white matter hypoattenuation. These findings raised the possibility of cortical laminar necrosis associated with posterior encephalomalacia. This case highlights the importance of comprehensive CT assessment in patients with WE who develop neurological deterioration. The coexistence of symmetrical deep cerebral abnormalities and diffuse cortical changes warrants careful interpretation and correlation with previous MRI findings, clinical data, and laboratory results. Alternative or concomitant mechanisms of cerebral injury should be considered before attributing all abnormalities to WE.

Keywords: Wernicke Encephalopathy, Computed Tomography, Magnetic Resonance Imaging, Cortical Laminar Necrosis, Chronic Alcohol Use.

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INTRODUCTION

Wernicke encephalopathy (WE) is an acute, potentially life-threatening neurological disorder resulting from thiamine (vitamin B1) deficiency. Although chronic alcohol use is a well-established risk factor, WE may also occur in other clinical settings associated with malnutrition or impaired thiamine absorption. Diagnosis can be challenging because of the variable clinical presentation and the frequent absence of the classical triad of altered mental status, ocular abnormalities, and gait ataxia. Early recognition and treatment are essential to prevent irreversible neurological sequelae [1, 2].

Neuroimaging plays an important role in supporting the diagnosis and characterizing the extent of cerebral involvement. MRI is the preferred imaging modality and typically demonstrates bilateral, symmetrical signal abnormalities involving the medial thalami, mammillary bodies, periaqueductal gray matter, and tectal plate. Atypical patterns, including cortical and other deep gray matter involvement, have also been reported [1–3]. By contrast, CT has limited sensitivity for detecting acute WE and may be normal or reveal nonspecific abnormalities. Nevertheless, CT remains valuable in the emergency setting, particularly when evaluating patients with altered consciousness or neurological deterioration [2-4].

We report the case of a 73-year-old patient with chronic alcohol use and an initial MRI-supported diagnosis of WE who subsequently developed altered consciousness. The subsequent CT examination demonstrated symmetrical deep cerebral hypoattenuation associated with diffuse gyriform cortical hyperattenuation and posterior subcortical white matter abnormalities. This case illustrates the diagnostic challenges posed by the coexistence of findings

suggestive of WE and additional patterns of cerebral injury.

Case Presentation

A 73-year-old patient with a history of chronic alcohol use initially presented with behavioral disturbances, prompting brain MRI. The MRI findings, considered alongside the clinical and laboratory data, supported the diagnosis of WE (Figure 1). The patient subsequently developed altered consciousness and was admitted to the emergency department for further evaluation.

Brain CT demonstrated bilateral, symmetrical hypoattenuating abnormalities involving the posterior portions of the external capsules, extending to the cerebral peduncles. Diffuse gyriform hyperattenuation of the supratentorial cortex was also observed, associated with bilateral parieto-temporo-occipital subcortical white matter hypoattenuation. Mild dilatation of the lateral ventricles and diffuse enlargement of the supratentorial subarachnoid spaces were additionally noted. The midline structures and fourth ventricle remained in their normal positions.

The CT findings were interpreted as compatible with the previously established diagnosis of WE, with additional abnormalities suggestive of diffuse supratentorial cortical laminar necrosis and posterior encephalomalacia (Figure 2).

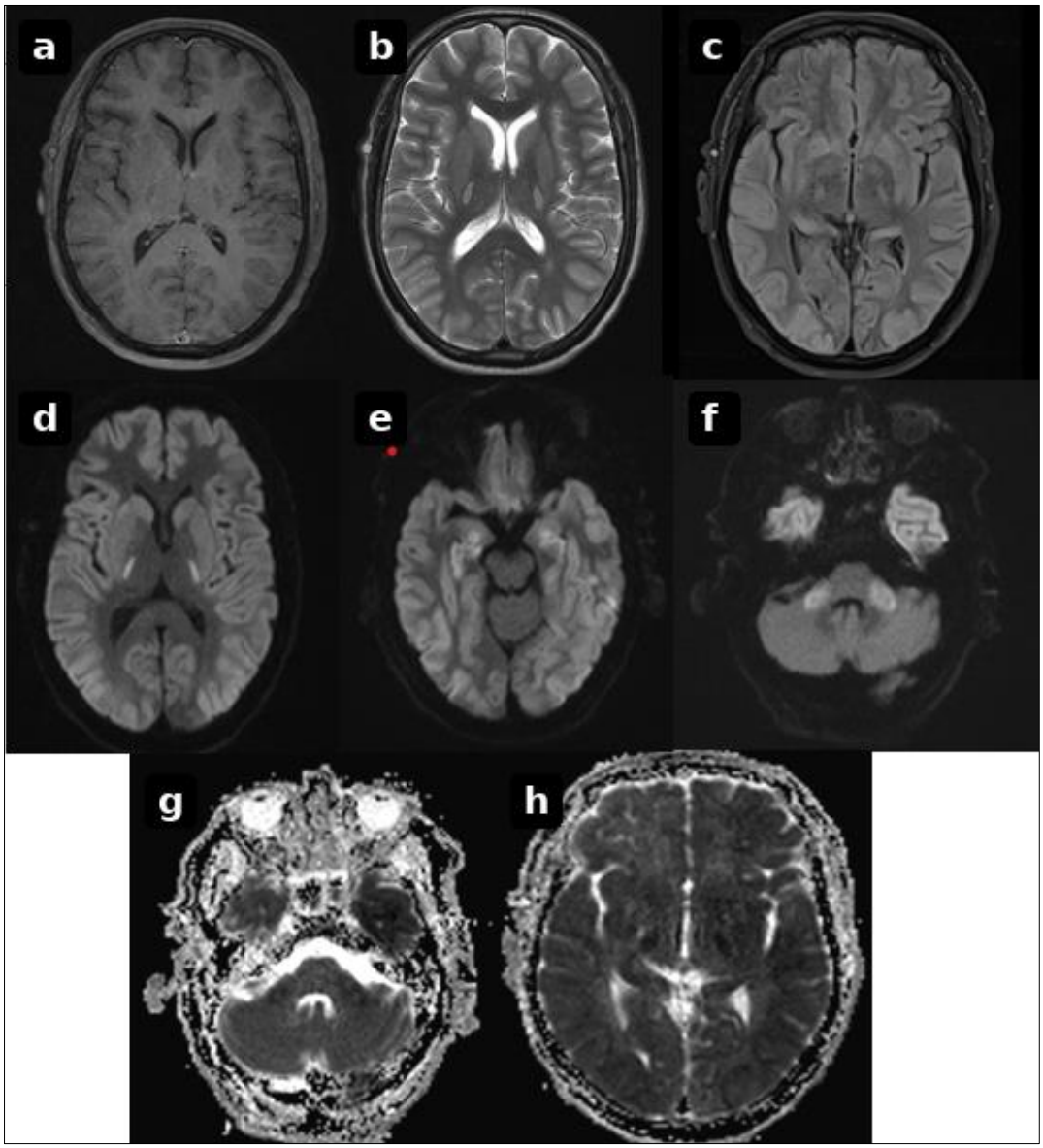


Figure 1: Axial T1-weighted (a), T2-weighted (b), FLAIR (c), diffusion-weighted (d, e, f), and ADC map (g, h) images demonstrating bilateral and relatively symmetrical signal abnormalities, particularly involving the cortical and subcortical regions, with increased signal intensity on diffusion-weighted images. These findings may represent atypical manifestations of Wernicke encephalopathy in the appropriate clinical context.

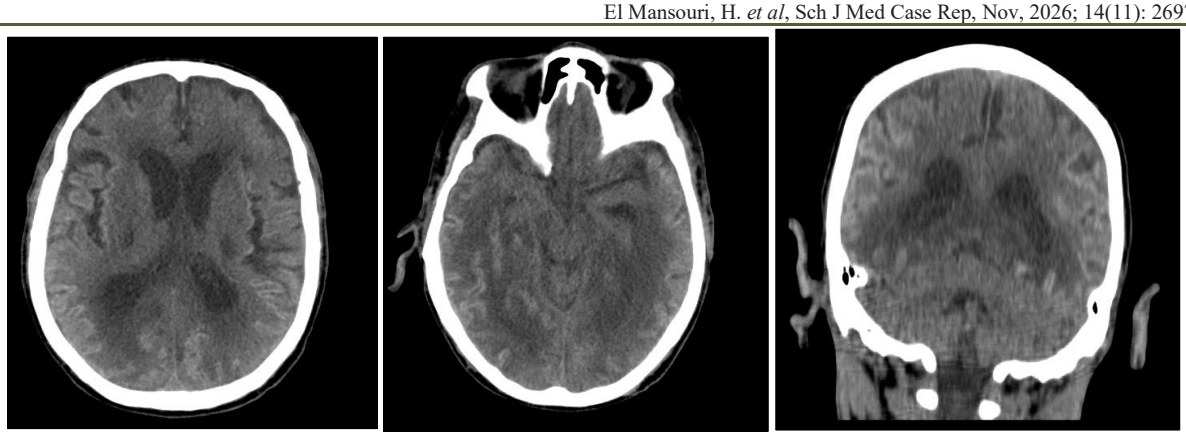


Figure 2: Brain CT images in axial (A, B) and coronal (C) planes showing bilateral, symmetrical hypoattenuating lesions involving the posterior portions of the external capsules and cerebral peduncles, consistent with Wernicke encephalopathy. Diffuse supratentorial gyriform cortical hyperattenuation, suggestive of cortical laminar necrosis, is associated with posterior encephalomalacia.

DISCUSSION

WE is a neurological emergency caused by thiamine deficiency, which impairs cerebral energy metabolism and may result in reversible edema or irreversible neuronal injury. Chronic alcohol use is a major risk factor because of its association with inadequate nutritional intake and impaired thiamine absorption and utilization [1, 2]. The clinical presentation is variable, and the classical triad of altered mental status, ocular abnormalities, and ataxia is frequently incomplete. In our patient, behavioral disturbances prompted the initial MRI examination, which supported the diagnosis of WE. Subsequent deterioration, characterized by altered consciousness, led to emergency assessment and further brain imaging.

MRI is the preferred imaging modality for suspected WE. Typical abnormalities consist of bilateral, symmetrical T2-weighted and fluid-attenuated inversion recovery (FLAIR) hyperintensities involving the medial thalami, mammillary bodies, periaqueductal region, and tectal plate. Atypical involvement of the cerebral cortex, basal ganglia, cerebellum, brainstem, and corpus callosum has also been described [1–3]. In their comparative study, Antunez *et al*., reported an MRI sensitivity of 53% and specificity of 93%. CT demonstrated paraventricular thalamic hypoattenuation in only 13% of patients with acute WE, underscoring its limited sensitivity for the diagnosis [4]. Nevertheless, CT can provide useful information about additional abnormalities and alternative causes of neurological deterioration.

In the present case, the subsequent CT examination revealed bilateral, symmetrical hypoattenuating abnormalities involving the posterior portions of the external capsules and extending to the cerebral peduncles. These findings were interpreted in the context of the previously established diagnosis of WE. However, the precise anatomical localization should be confirmed by reviewing the original images to ensure accurate radiological terminology.

A particularly notable finding was the diffuse gyriform hyperattenuation of the supratentorial cortex, associated with bilateral parieto-temporo-occipital subcortical white matter hypoattenuation. The cortical appearance raised the possibility of cortical laminar necrosis, a pattern of selective cortical injury that may occur following hypoxic-ischemic insults, prolonged seizures, and severe metabolic disturbances [5]. Although cortical laminar necrosis is commonly characterized on MRI by gyriform T1-weighted hyperintensity, the appearance on CT may vary according to the stage and underlying mechanism of injury. Consequently, the CT findings should be interpreted in conjunction with the clinical context and MRI appearances rather than considered independently diagnostic.

Cortical involvement is an uncommon but recognized imaging manifestation of WE. Wu *et al*., reviewed 24 cases of WE associated with cortical injury, including two cases reported by the authors, demonstrating that cortical abnormalities may occur in both alcoholic and nonalcoholic patients [6]. Ababou *et al*., also described an association between WE and diffuse cortical laminar necrosis in a chronic alcohol user presenting with profound impairment of consciousness [7]. These reports provide relevant comparisons with the present observation, although similarities in imaging appearance do not establish an identical underlying mechanism.

The bilateral parieto-temporo-occipital subcortical white matter hypoattenuation represents an additional component of the imaging pattern. This finding is nonspecific and may reflect edema or established parenchymal injury. The interpretation of posterior encephalomalacia suggests tissue damage, but its chronicity and precise nature cannot be determined from the CT description alone. Direct comparison with

the initial MRI is therefore important to establish whether the cortical and subcortical abnormalities were already present at the time of the initial diagnosis.

The temporal sequence also requires cautious interpretation. Although the cortical abnormalities were identified on the subsequent CT, their actual onset cannot be established without direct review and comparison of the initial MRI and subsequent CT. They should therefore be described as identified on the subsequent CT rather than definitively newly developed. Furthermore, the available information does not establish whether hypoxic-ischemic injury, prolonged seizures, or another metabolic disturbance contributed to the cortical changes. Such mechanisms should be considered according to the clinical course and laboratory findings [5–7].

MRI, including T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging, and apparent diffusion coefficient maps, may help characterize the cortical and subcortical abnormalities and assess the extent of tissue injury. Susceptibility-sensitive sequences may also be useful if hemorrhagic components are suspected. However, the interpretation of these sequences must take into account the timing of imaging and the clinical context. In particular, no single imaging feature should be used in isolation to establish the cause of cortical injury [1–5].

Mild dilatation of the lateral ventricles and diffuse enlargement of the supratentorial subarachnoid spaces were also observed. These findings may be associated with cerebral volume loss but are nonspecific and should be interpreted in relation to the patient's age and the overall imaging appearance.

The principal interest of this case lies in the complementary assessment provided by sequential MRI and CT in a patient with WE who initially presented with behavioral disturbances and subsequently developed altered consciousness. The later CT demonstrated deep cerebral abnormalities together with diffuse gyriform cortical hyperattenuation suggestive of cortical laminar necrosis and posterior parenchymal abnormalities interpreted as encephalomalacia. This combination emphasizes the importance of reassessing the entire brain parenchyma when neurological status deteriorates. It also highlights the need to consider associated or alternative mechanisms of cerebral injury rather than automatically attributing all abnormalities to WE.

Conclusion

This case illustrates the complex imaging findings that may be encountered in a patient with Wernicke encephalopathy who develops neurological deterioration. The coexistence of symmetrical deep cerebral abnormalities, diffuse gyriform cortical hyperattenuation, and posterior subcortical white matter changes highlights the importance of a comprehensive radiological assessment. Direct comparison of sequential imaging examinations, together with clinical and laboratory correlation, is essential to characterize the extent of cerebral injury and avoid premature attribution of all findings to a single mechanism.

References

1. 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. doi:10.1007/s11604-020-00989-3.
2. Manzo G, De Gennaro A, Cozzolino A, Serino A, Fenza G, Manto A. MR imaging findings in alcoholic and nonalcoholic acute Wernicke's encephalopathy: a review. Biomed Res Int. 2014;2014:503596. doi:10.1155/2014/503596.
3. Zuccoli G, Santa Cruz D, Bertolini M, Rovira A, Gallucci M, Carollo C, et al. MR imaging findings in 56 patients with Wernicke encephalopathy: nonalcoholics may differ from alcoholics. AJNR Am J Neuroradiol. 2009;30(1):171-6. doi:10.3174/ajnr.A1280.
4. Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A. Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy. AJR Am J Roentgenol. 1998;171(4):1131-7. doi:10.2214/ajr.171.4.9763009.
5. Laksiri N, Kaphan E, Pellissier JF, Ali Chérif A. Nécrose laminaire corticale : aspects en imagerie par résonance magnétique et étude anatomoclinique. Rev Neurol (Paris). 2007;163(3):365-8. doi:10.1016/S0035-3787(07)90410-5.
6. Wu L, Jin D, Sun X, Liang L, Huang D, Dong Z, et al. Cortical damage in Wernicke's encephalopathy with good prognosis: a report of two cases and literature review. Metab Brain Dis. 2017;32(2):377-84. doi:10.1007/s11011-016-9920-0.
7. Ababou M, Kartite N, Boubekri A, Bennani H, Houba A, Doghmi N, et al. Nécrose corticale laminaire associée à une encéphalopathie de Gayet-Wernicke révélée par un trouble de conscience profond chez un alcoolique chronique. PAMJ Clin Med. 2020;4:3. doi:10.11604/pamj-cm.2020.4.3.23633.