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Case Report

Wernicke encephalopathy with atypical imaging findings in a depressed patient: A case report☆,☆☆

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ABSTRACT

This case report highlights the rare occurrence of Wernicke encephalopathy caused by malnutrition in a depressed patient with atypical imaging findings. A 60-year-old female with depression developed a disturbance of consciousness owing to Wernicke encephalopathy. Magnetic resonance imaging showed abnormal signals in the thalamus and mammillary bodies around the third ventricle, cerebral aqueduct, and fourth ventricle. Abnormal signals were also present in the cerebral cortex around the central sulcus, and an intracranial hemorrhage from the thalamus was observed. Therefore, clinicians should consider Wernicke encephalopathy in the differential diagnosis of altered consciousness in depressed patients. Early assessment of nutritional status and prompt intervention are crucial in cases of prolonged depression-related malnutrition.

Introduction

Wernicke encephalopathy (WE) is an acute neuropsychiatric syndrome caused by vitamin B1 (thiamine) deficiency. Therefore, swift intervention is imperative in WE. Inadequate treatment may lead to the development of serious neurological disorders, such as Korsakoff psychosis, which in severe cases can result in death [1]. Its prevalence is highest among individuals with alcohol use disorder and malnutrition, with 30%-80% of alcoholics exhibiting signs or laboratory findings indicative of thiamine deficiency [2,3]. In depressed patients, malnutrition can occasionally lead to thiamine deficiency, independent of alcohol-related causes, although such instances are rare [4].

This case highlights the rare occurrence of WE caused by malnutrition in a depressed patient with atypical imaging findings. WE associated with cortical lesions and intracranial hemorrhage, as in our case, may indicate a poor prognosis [5]. Hence, early intervention is crucial to prevent the development of WE [6].

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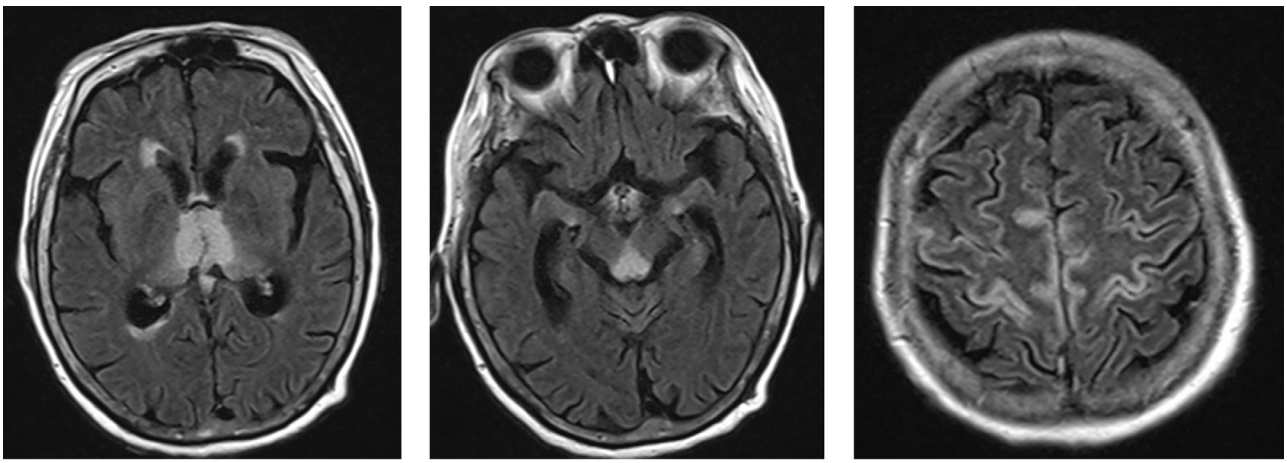

Fig. 1 – MRI of a 60-year-old woman with a history of depression for over 20 years who was admitted to the hospital complaining of altered consciousness. MRI identified fluid-attenuated inversion recovery (FLAIR) hyperintense signals in the thalamus (A), cerebral aqueduct (B), and cerebral cortex around the central sulcus (C).

Case report

A 60-year-old female with a history of depression spanning over 20 years presented with altered consciousness. Over the past 6 months, the patient's depressive symptoms worsened, leading to decreased food intake. Additionally, the patient experienced a decline in activity and mobility, ultimately manifesting as altered consciousness. The initial suspicion of urinary tract infection prompted treatment; however, as the patient's consciousness deteriorated, she was transferred for further evaluation. Upon admission, the patient had a Japan Coma Scale score of 200, a Glasgow Coma Scale score of E1V1M1, dilated pupils, absent Doll 's eye reflex, absent eyelash reflex, and loss of limb tendon reflexes. The patient had a history of depression without any other medical conditions, family history of allergies, or a history of smoking or alcohol consumption. The patient was not on any regular medication and the clotting profile blood test was normal. The urine toxicology test was negative.

Biochemical screening of the blood revealed low serum albumin (2.3 mg/dL) and hemoglobin (10.9 g/dL) levels, without other significant abnormalities. Neuroimaging through magnetic resonance imaging (MRI) identified abnormal signals in the thalamus, mammillary bodies, around the third ventricle, cerebral aqueduct, and fourth ventricle. Abnormal signals were observed in the cerebral cortex around the central sulcus. Computed tomography (CT) and MRI revealed intracranial hemorrhage from the thalamus (Fig. 1, 2). Despite atypical imaging findings, WE was suspected. Abnormal MRI signals and decreased levels of vitamin B1 (6 ng/mL; normal value: 28-56 ng/mL) and folate (0.5 ng/mL; normal value: 3.6-12.9 ng/mL) raised suspicion of WE caused by malnutrition associated with depression. Although the differential diagnosis of the thalamic appearance includes hemorrhagic venous infarction, since cortical lesions were seen, we considered the lesions to be caused by WE. Vitamin supplementation therapy and nutritional improvement were promptly initiated. Despite improvement in MRI abnormalities and Vitamin B1 and folic acid levels after 2 weeks (Fig. 3), the altered consciousness persisted and poor improvement in neurological findings, leading to the patient's transfer to a chronic care facility 3 weeks later.

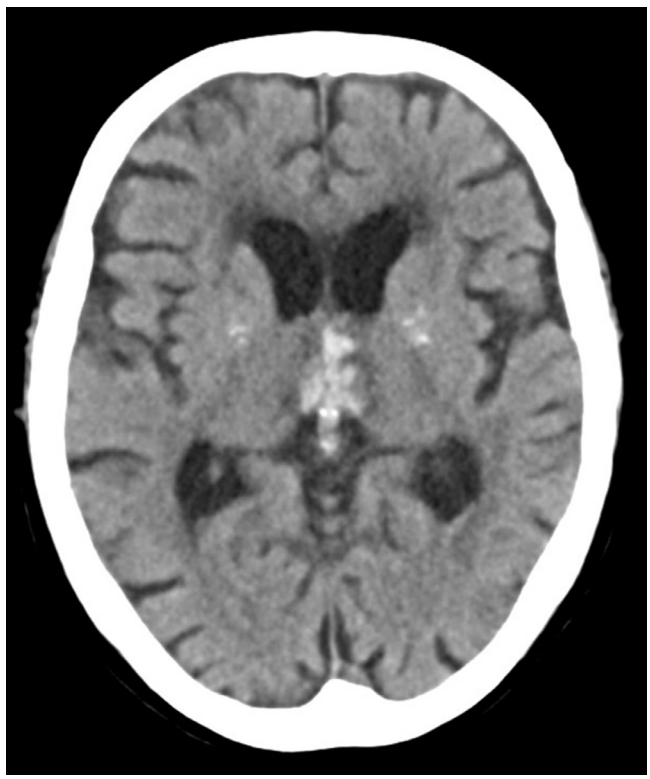

Fig. 2 – CT on the day of admission. Intracranial hemorrhage from the thalamus is observed on CT (A).

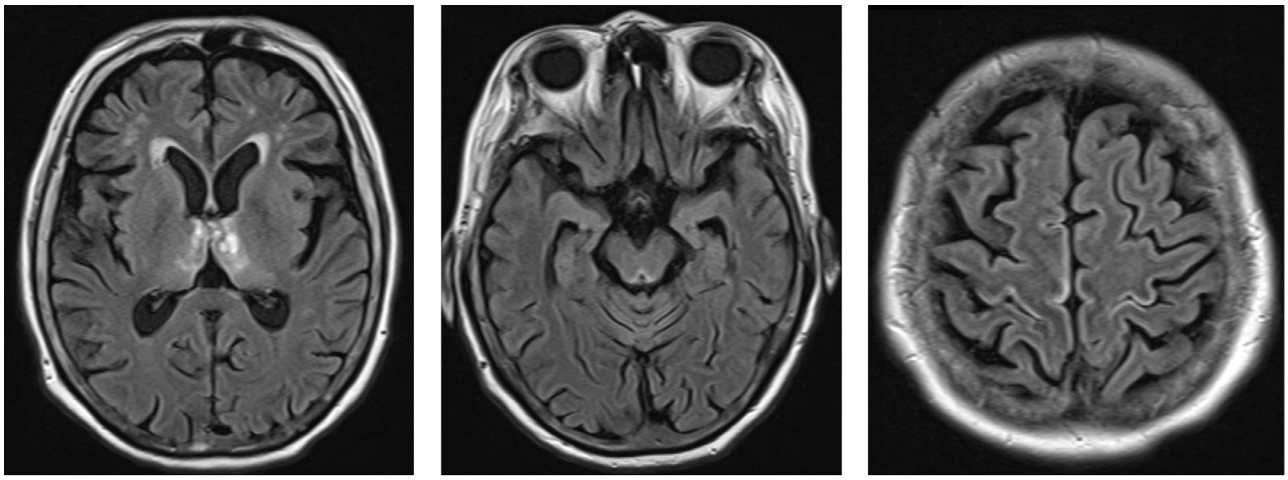

Fig. 3 – MRI of 2 weeks after starting treatment. The MRI abnormalities in the thalamus (A), cerebral aqueduct (B), and cerebral cortex (C) improved. However, altered consciousness persisted.

Table 1 – Literature review of Wernicke encephalopathy (WE) complicated by cortical lesions and intracranial hemorrhage.

Author	Age	Sex	Symptom	Cause of WE	Location of bleeding	Prognosis (activities of daily living when last followed up)
DePolo D [18]	31	male	extremity weakness, gait instability, confusion	poor oral intake for pharyngitis	thalamus	good
Pereira DB [19]	27	female	aphasia, diplopia seizures, confusion	Crohn disease, total colectomy	cortex of the frontal lobes	good
Present case	60	female	altered consciousness, dilated pupils, absent doll's eye reflex, absent eyelash reflex, loss of limb tendon reflexes	poor oral intake for depression	thalamus	poor

Discussion

WE emerges as a neurological condition triggered by a lack of thiamine. It typically manifests as a trio of ocular dysfunction, changes in mentation, and ataxia, although only approximately 10%-33% of individuals exhibit the complete triad [7]. Although it is frequently linked to prolonged excessive alcohol consumption, it is crucial to consider this condition in individuals with acute neurological impairments and a background of malnutrition.

Depression is a mental health condition that occurs most often among individuals facing substantial health challenges [8]. In depression, there is often a decrease in food consumption, and the role of nutrition becomes pivotal in both the initiation and intensity of depressive symptoms [9]. Owing to the lack of studies in this area, it remains relatively unknown whether malnutrition in depression can lead to WE [10]. This may be owing to WE being overlooked in depressed patients. The outcome in our case may have been different if WE had been suspected earlier.

WE is characterized by distinct MRI findings, including T2-weighted imaging/fluid-attenuated inversion recovery (FLAIR) hyperintense signals in the thalami, mammillary bodies, tectal plates, and periaqueductal area [9]. These findings are crucial for the early diagnosis and timely initiation of vitamin B1 supplementation, which can reverse the clinical features of the disease [11].

Involvement of the cerebral cortex in WE is rare. In addition, WE with cortical damage has been reported in patients without alcoholism [12]. Cortical abnormalities have been reported in the frontal and parietal lobes, particularly around the central sulcus [13]. These cortical lesions can be reversed with vitamin B1 supplementation but may persist and result in long-term impairment [14]. Cortical involvement indicates irreversible damage and a poor prognosis [12]. Linear ribbon-like cortical lesions have also been noted, although their prognostic significance is unclear [15].

Intracranial hemorrhage in WE is rare, and its cause is unclear; however, 2 factors have been considered in the past [16,17]. The primary factor is bleeding diathesis that is not intrinsic to WE itself [16,17]. The secondary factor, intrinsic to WE itself, influences both the localization and severity of lesions observed in the disease [16,17]. Hemorrhage may be a component of these lesions, even if it is only petechial or microscopic. Thiamine deficiency is believed to be a crucial factor in the development of these lesions; however, the mechanism by which morphological changes occur and the reasons for the

selective vulnerability of specific brain areas remain unclear. One of the most compelling mechanical theories suggests that when cell apoptosis and necrosis occur in WE, vascular structures are concurrently damaged [16,17]. Although there have been no reports of WE in depressed patients complicated by intracranial hemorrhage, this may indicate a poor prognosis. The cause of cerebral hemorrhage in our case is unknown, but long-term thiamine deficiency may have caused blood vessels to become fragile and rupture.

WE, characterized by atypical imaging findings such as cortical lesions and intracranial hemorrhage, as observed in our case, is a rare occurrence. We searched PubMed and Google Scholar to review the available literature (published until December 2023) on WE complicated by cortical lesions and intracranial hemorrhage using the key terms "cortical," "intracranial hemorrhage," and "MRI." Previous reports have described 3 cases of WE complicated by cortical lesions and intracranial hemorrhage, including our case (Table 1). All patients were nonalcoholic, and no episodes of hypertension or other coagulopathies were present as alternative causes of intracranial hemorrhage. Early high-dose thiamine treatment may lead to significant improvement and favorable clinical outcomes, even in the presence of these imaging findings [18,19]. Only our case had a poor prognosis (activities of daily living when last followed up). Therefore, the relationship between these atypical features and the prognosis of WE requires further research, but in any case, early intervention is necessary. Even if a coagulation profile is normal, cerebral hemorrhage may occur, it requires close attention during treatment.

There are no reports of WE with poor prognosis associated with atypical imaging findings in depressed patients, which may be owing to long-term vitamin B1 deficiency, as seen in our case.

In conclusion, this case report highlights the rare occurrence of WE caused by malnutrition in a depressed patient. MRI abnormalities, intracranial bleeding, and cortical signals were observed, which contributed to a challenging prognosis. Therefore, clinicians should consider WE in the differential diagnosis of altered consciousness in depressed patients. Early assessment of nutritional status and prompt intervention are crucial in cases of prolonged depression-related malnutrition. This report emphasizes the importance of early diagnosis, comprehensive care, and consideration of a poor prognosis without appropriate treatment, even in WE with atypical MRI findings.

Technical information about the MR and CT scans

Brain MRI were performed at 3T (Siemens Magnetom Prisma, Erlangen, Germany) using a 64-channel receive head/neck coil.axial T1-weighted spin-echo (430/10 [repetition time (TR) msec/echo time (TE) msec]),axial T2-weighted fast spin-echo (3400/100 [effective echo time]), and axial FLAIR (10000/400/2200 [inversion time]). The parameters were a 256 × 192 matrix, a 23-cm field of view, and a 5 mm/2 mm slice thickness/intersection gap. Single-shot, spin-echo, echo-planar DWI sequences, maps of the apparent diffusion coefficient (ADC) were generated using the b-values of 0 s/mm^2 and 1000 s/mm^2.

A multidetector helical CT scanner (GE Lightspeed; GE Medical Systems, Milwaukee, WI) was performed: non-contrast-enhanced transaxial CT of the brain (contiguous, 3.75-mm-thick sections, 120 kV, 170 mAs).

Author contributions

Contributed equally to this work with: H.T. and R.I.; Conceptualization, H.T., R.I., A.T. and Y.T.; Methodology, H.T.; Investigation, H.T.; Resources, H.T.; Data curation, H.T. and A.T.; Writing—original draft preparation, H.T.; Writing review and editing, R.I., K.Y., Y.S., Y.A., Ke.K, T.N., K.I., Ko.K., T.Y., H.Y., T.O., A.T. and Y.T.; Visualization, H.T.; supervision, T.Y., H.Y., T.O., A.T. and Y.T., All authors have read and agreed to the published version of the manuscript.

Institutional review board statement

Not applicable.

Data availability statement

The data presented in the present study are available on request from the correspondent author.

Patient consent

Written informed consent was obtained from the patient's legal guardians for publication of this case report and any accompanying images. A case report is not required institutional review board (IRB) in our institution.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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