

## Case Report

# Wernicke's Encephalopathy in a Patient with Nasopharyngeal Carcinoma: Magnetic Resonance Imaging Findings

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## Abstract

We report a case of Wernicke's encephalopathy in a patient with nasopharyngeal carcinoma with a 3-month history of poor oral intake related to nausea and vomiting due to chemotherapy. The patient later developed deep coma while receiving in-patient therapy. Magnetic resonance imaging of the brain revealed typical findings of Wernicke's encephalopathy. The patient was treated with thiamine injections, which resulted in subsequent partial recovery of neurological function. This paper stresses the importance of magnetic resonance imaging for prompt diagnosis of Wernicke's encephalopathy.

**Keywords:** adult, male, magnetic resonance imaging, neurology, thiamine deficiency, Wernicke's encephalopathy

## Introduction

Wernicke's encephalopathy (WE) is a neurological manifestation of thiamine deficiency. A history of alcohol abuse is present in 50% of patients with WE (1). In non-alcoholic patients, WE is due to malnutrition and malabsorption. WE is likely to be underdiagnosed and should be considered in patients with poor oral intake or nutrition, especially those with concurrent history of heavy alcohol intake (2). The most common presentation is altered consciousness ranging from disorientation to deep coma. The typical clinical triad of confusion, ataxia, and ophthalmoplegia is only present in 16%–38% of patients, making clinical diagnosis a challenge (1,3). Brain damage is related to osmotic injury and disruption of the blood–brain barrier at characteristic locations. This distribution gives rise to diagnostic findings on magnetic resonance imaging (MRI) of the brain. This case illustrates the importance of MRI in aiding the diagnosis of WE.

## Case Report

The patient was a 41-year-old male with nasopharyngeal carcinoma who had completed radiotherapy (70 Gy/38 times) and 6 cycles of weekly cisplatin (30 mg/m<sup>2</sup>, 60 mg) from

21 April to 10 June 2010. He was a social drinker and did not have any history of alcohol abuse. He presented to the oncology clinic complaining of a mouth ulcer, nausea, vomiting, and poor oral intake for 3 months since the commencement of therapy. Upon examination, the patient was orientated but was dehydrated and lethargic. His vital signs were stable, and the patient was afebrile. The patient's serum sodium level was 132 mmol/L, while the rest of the serum electrolytes and blood investigations were unremarkable.

The patient was admitted to the ward for hydration, antiemetic drug administration, and total parenteral nutrition. One week after admission, the patient was asymptomatic, but his serum sodium level was 111 mmol/L. Sodium replacement was commenced with slow bolus infusion of 3% normal saline given over 12 hours, followed by normal saline fluid maintenance. The serum sodium level was rechecked 24 hours later, and it was 126 mmol/L.

The patient began to experience diplopia and an unstable gait, which were attributed to hyponatraemia; therefore, sodium replacement was continued. The next day, he developed an episode of unresponsiveness but regained consciousness a few hours later. During this episode, a bolus of dextrose (50%) and mannitol each were given intravenously. His serum sodium level at this time was 129 mmol/L.

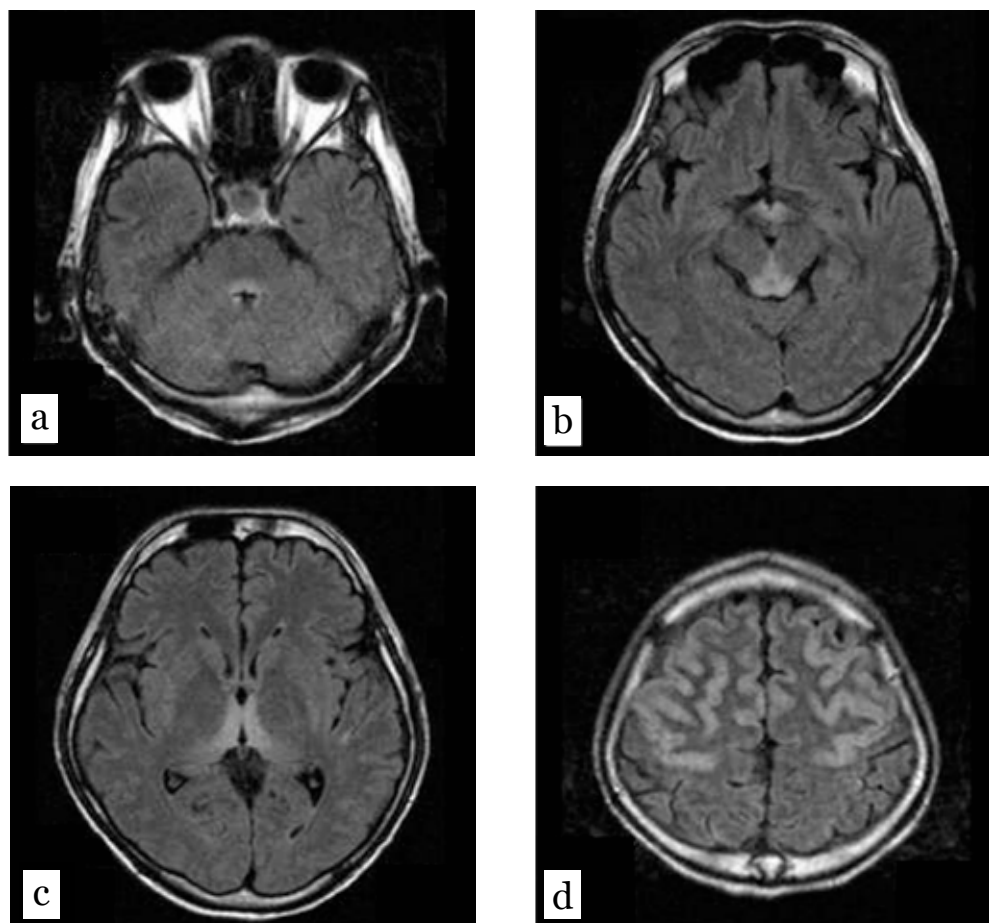
Subsequently, there was further deterioration of consciousness; the patient became deeply comatose after 2 days, with a Glasgow Coma Score (GCS) of 9. Computed tomography (CT) of the brain did not reveal any abnormality. A brain MRI was performed and showed symmetric hyperintensities in the medial thalami, periaqueductal grey, and mammillary bodies (Figure 1). Symmetric cortical hyperintensities at both frontal lobes were also present. These findings were consistent with WE.

There was a further reduction of the level of consciousness, with no response to pain stimuli and a GCS of 3. Pupillary reflexes were still present. Intubation was performed for airway protection. Intravenous thiamine replacement therapy was started immediately. There was a gradual improvement of the neurological function, and

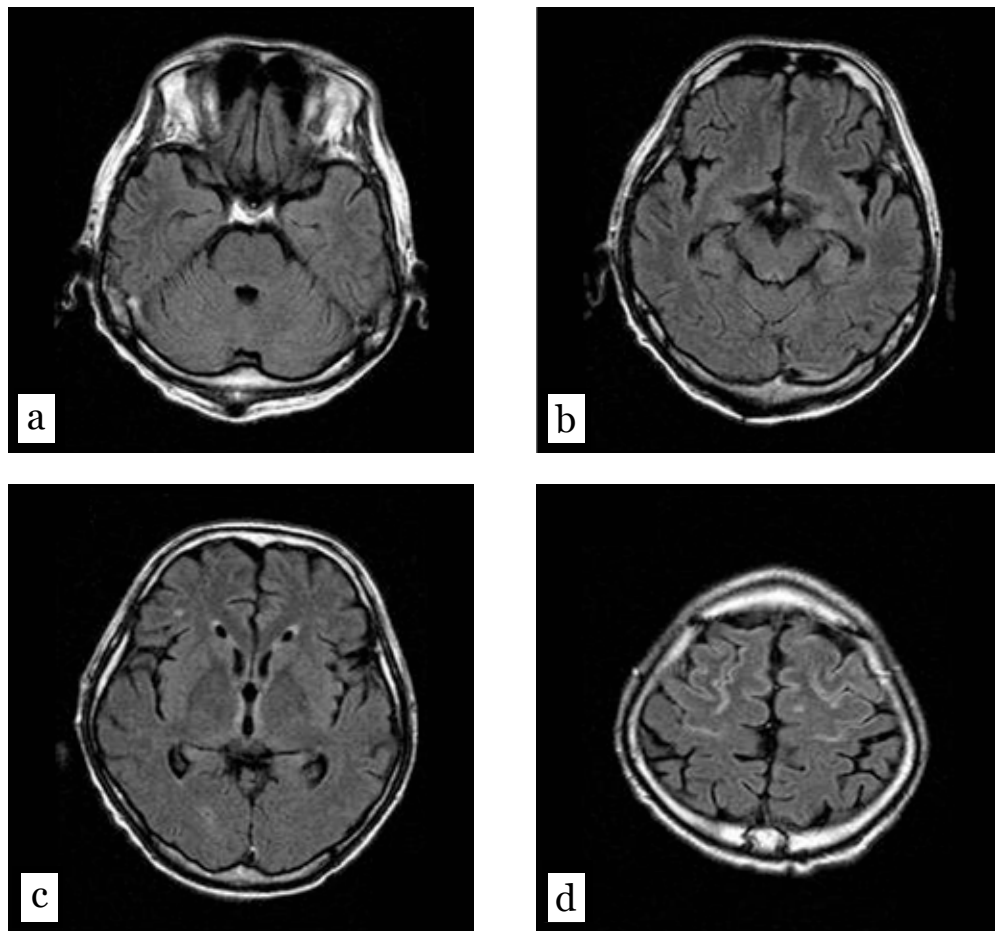
the patient was able to demonstrate spontaneous eye opening and appropriate responses to pain stimuli after 1 week. His progress was complicated by aspiration pneumonia, which was treated with intravenous antibiotics and chest physiotherapy. A follow-up MRI of this patient 1 month later showed a reduction in the signal abnormalities seen previously (Figure 2), corresponding with the clinical improvement. Following this slight improvement, his condition remained the same with no further improvement.

## Discussion

This case highlights the crucial role of brain MRI in the diagnosis of WE in non-alcoholic patients who can present in a variety of clinical settings. In the literature, non-alcoholic WE is



**Figure 1:** Magnetic resonance imaging of brain performed several days after acute neurological symptoms revealed bilateral symmetric T2 fluid-attenuated inversion recovery hyperintensities in a) the floor of the fourth ventricle, b) the periaqueductal grey and the mammillary bodies, c) the medial thalami, and d) the bifrontal lobes cortices. The patient was deeply comatose during this period.



**Figure 2:** Magnetic resonance imaging of brain performed 1 month after the first imaging revealed a reduction and resolution of the high signal intensity on T2 fluid-attenuated inversion recovery in a) the floor of the fourth ventricle, b) the periaqueductal grey and the mammillary bodies, c) the medial thalami, and d) the bifrontal lobes cortices. There was a marked neurological improvement, with the patient being able to demonstrate spontaneous eye opening and appropriate responses to pain stimuli.

most likely to occur after prolonged therapeutic fasting, parenteral nutrition, intravenous infusion of glucose, and post-bariatric surgery (4,5). Less commonly, it is also reported in other varied circumstances, including hyperemesis gravidarum, chemotherapy, and incorrect feeding formula. In our patient, who was on chemotherapy with a poor oral intake due to poor appetite, mucositis predisposed him to the thiamine deficiency.

WE is an uncommon neurological disorder in the non-alcoholic population. Traditionally, WE is a clinical diagnosis in an alcoholic who presents with the triad of global confusion, ataxia, and ophthalmoplegia. Therefore, it is

not surprising that in clinical practice, WE can be missed or underdiagnosed in non-alcoholic patients for a myriad of reasons from lack of awareness to its less typical course and other compounding factors. In this case, the co-existing hyponatraemia, underlying primary malignancy, and chemotherapy contributed to the difficulty in making a clinical diagnosis. Although the clinical triad of diplopia, unsteady gait, and confusion was present in this case, the diagnosis of WE was made only after a brain MRI showed the typical findings of WE. The administration of 50% dextrose during the initial episode of loss of consciousness in this patient may have precipitated the WE, because the patient subsequently went into a deep coma.

CT of the brain is not sufficiently sensitive to diagnose WE. The value of MRI is in the prompt diagnosis of acute WE to facilitate early treatment and to prevent irreversible brain damage (1,2). There is a recognisable pattern in WE, which, if present, is generally regarded as pathognomonic of WE. Findings on MRI include a typical distribution of high signal intensities on T2 and fluid-attenuated inversion recovery sequences at the medial thalami, head of the caudate nuclei, mammillary bodies, areas surrounding the aqueduct, third ventricle, and floor of the fourth ventricle. These changes are characteristically bilateral and symmetrical (1,2).

In addition to these typical lesions, our case also had bilateral and symmetrical lesions in the cortex of both frontoparietal regions. Cortical lesions surrounding the central sulcus are increasingly recognised as part of the WE spectrum. Other atypical findings that have been reported include the involvement of the dorsal medulla, red nuclei, and dentate nuclei (1,7). A lack of typical imaging features on an MRI does not exclude the diagnosis. In these cases, contrast-enhanced MRI and diffusion weighted imaging may help to visualise early changes (8).

MRI is also helpful in predicting disease progression or prognosis (2). Cortical involvement is associated with deep coma and poorer prognosis, as was seen in our patient (2,6). Reduction in these signal abnormalities on follow-up imaging corresponds to clinical improvement, while progressive brain atrophy is associated with a poor neurological outcome (2). Follow-up MRI in this patient 1 month later showed a reduction of the signal abnormalities seen previously, corresponding with the clinical improvement.

Empirical treatment should be started as soon as WE is clinically suspected (3). Ocular symptoms will resolve within hours, with subsequent improvement of ataxia within days, and improvement of confusion within weeks after the start of treatment (3). We found that 67% of patients had full neurological recovery over a variable length of time, from 2 weeks to 1 year after thiamine therapy. Patients whose cases have been reported in the literature were administered 100 mg of thiamine intravenously or intramuscularly for 5 to 15 days after initial presentations (2).

Non-alcoholic WE is underdiagnosed. MRI facilitates early diagnosis by virtue of its typical findings. WE is a fatal but potentially treatable condition that can be resolved by thiamine administration. Prompt treatment administered prior to established brain damage ensures a

good outcome for patients. This case highlights the important practical role of MRI in aiding diagnosis as well as its prognostic value.

## Authors' Contributions

Conception and design, critical revision and final approval of the article: ST

Provision of patient, collection and assembly of the data: RS

Analysis and interpretation of the data, drafting of the article: HLL

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## References

1. Zuccoli G, Gallucci M, Capellades J, Regnicolo L, Tumiatì B, Giadas TC, et al. Wernicke encephalopathy: MR findings at clinical presentation in twenty-six alcoholic and nonalcoholic patients. *AJNR Am J Neuroradiol*. 2007;**28**(7):1328–1331.
2. Zhong CJ, Jin LR, Fei GJ. MR imaging of nonalcoholic Wernicke encephalopathy: A follow-up study. *Am J Neuroradiol*. 2005;**26**(9):2301–2305.
3. Sechi G, Serra A. Wernicke's encephalopathy: New clinical settings and recent advances in diagnosis and management. *Lancet Neurol*. 2007;**6**(5):442–455.
4. Fei GQ, Zhong C, Jin L, Wang J, Zhang Y, Zheng X, et al. Clinical characteristics and MR imaging features of nonalcoholic Wernicke encephalopathy. *Am J Neuroradiol*. 2008;**29**(1):164–169.
5. Singh S, Kumar A. Wernicke encephalopathy after obesity surgery: A systematic review. *Neurology*. 2007;**68**(1):807–811.
6. Sakurai K, Sasaki S, Hara M, Yamawaki T, Shibamoto Y. Wernicke's encephalopathy with cortical abnormalities: Clinicoradiological features: Report of 3 new cases and review of the literature. *Eur Neurol*. 2009;**62**(5):274–280.
7. Bae SJ, Lee HK, Lee JH, Choi CG, Suh DC. Wernicke's encephalopathy: Atypical manifestation at MR imaging. *Am J Neuroradiol*. 2001;**22**(8):1480–1482.
8. Osborn AG. Alcoholic encephalopathy. In: Osborn AG, Blaser SI, Salzman KL, Katzman GL, Provenzale J, Castillo M, et al. *Diagnostic imaging: Brain*. 1st ed. Canada: Amrisys; 2004. 1-10:20–23.