CASE REPORT

Wernicke's encephalopathy in a patient with gastric carcinoma: a diagnosis not to miss

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Accepted 10 February 2014

SUMMARY

We describe a case of a patient who presented with a 20-day history of vomiting, generalised weakness and loss of appetite and a 2-day history of altered sensorium. On examination, he was grossly emaciated and there were no palpable lymph nodes. Central nervous system examination revealed nystagmus with bilateral lateral recti palsy and abdominal examination showed mild hepatomegaly. MRI of the brain showed bilateral and symmetrical hypertense signal changes in T2-weighted and fluid-attenuated inversion recovery sequences with diffusion restriction in the paramedian ventromedial thalamus. These findings were compatible with Wernicke's encephalopathy. He was started on thiamine supplementation with which neurological signs improved. An ultrasound of the abdomen showed mild hepatomegaly with multiple hyperechoic lesions and wall thickening of the pyloric antrum. Upper gastroduodenoscopy showed ulcerative lesions involving the antrum, pylorus and duodenum. Biopsy revealed moderately differentiated adenocarcinoma. The patient underwent palliative gastrojejunostomy and was clinically better at discharge. It is important to consider Wernicke encephalopathy in patients with gastric cancer who have acute neurological symptoms.

BACKGROUND

Wernicke's encephalopathy (WE) is an often unrecognised disease of nutritional deficiency, and can lead to death if not treated. Although WE usually results from chronic alcoholism, non-alcoholic causes, such as gastrointestinal tract surgery, AIDS, chronic malnutrition, prolonged parenteral nutrition, hyperemesis gravidarum and rarely in malignancy, are reported in 20–50% of cases.[1 2] It is often underdiagnosed because clinicians may be less likely to recognise this condition in non-alcoholic patients, hence we present this report.

CASE PRESENTATION

A 35-year-old man presented with a 20-day history of vomiting, generalised weakness and severe loss of appetite. There was a history of swaying to either side while walking with slurring of speech for 10 days and altered sensorium for 2 days. On reviewing the history with the relatives, he was also found to have low-grade fever for the past 10 days. He was a non-smoker and a non-alcoholic. On examination, he was grossly emaciated, afebrile and there were no palpable lymph nodes. Oral thrush was noted. His blood pressure was 100/60 mm Hg and pulse rate 94/min. Neurological examination revealed disorientation to time and place, horizontal nystagmus and bilateral lateral recti palsy. Abdominal examination showed mild hepatomegaly. Other systems were unremarkable. The initial diagnosis was acute disseminated encephalomyelitis (ADEM).

INVESTIGATIONS

Blood investigations including complete blood count, serum glucose and renal and liver function tests were within normal limits. Thyroid study was normal. HIV serology by ELISA was negative. Blood culture was sterile and test for malarial parasites was negative. The chest X-ray was normal.

A CT of the brain and cerebrospinal fluid analysis did not reveal any abnormality. MRI of the brain showed symmetrical non-enhancing T2 and fluid-attenuated inversion recovery (FLAIR) hyperintensities with diffusion restriction in the paramedian ventromedial thalamus (figure 1). Hyperintensities were also seen along the dorsal portion of upper medulla and periaqueductal region, suggestive of WE.

An ultrasound of the abdomen showed mild hepatomegaly with multiple hyperechoic lesions and wall thickening of pyloric antrum. A CT of the abdomen showed short-segment circumferential thickening involving pyloric canal causing partial gastric outlet obstruction with liver metastases.

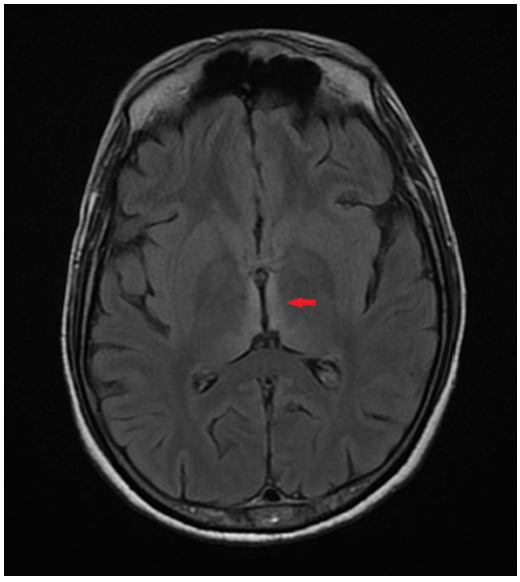

Figure 1 Symmetrical non-enhancing T2-weighted and fluid-attenuated inversion recovery hyperintensity with diffusion restriction seen in the paramedian ventromedial thalami.

To cite: Udyavara Kudru C, Kaniyoor Nagiri S, Rao S. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2013-203511

Upper gastroduodenoscopy showed ulcerative lesions involving the antrum, pylorus and duodenum. Biopsy revealed moderately differentiated adenocarcinoma.

DIFFERENTIAL DIAGNOSIS

Fever, vomiting, gait ataxia, altered sensorium and oculomotor deficits on examination led to a strong suspicion of ADEM.

TREATMENT

The patient was started on thiamine intravenously 500 mg in 0.9% normal saline thrice daily for two consecutive days followed by 500 mg intravenously once daily for an additional 5 days.

OUTCOME AND FOLLOW-UP

Confusion, ocular motor abnormalities and gait ataxia improved after few days following thiamine supplementation. The patient underwent palliative gastrojejunostomy after few days and was clinically better at discharge.

DISCUSSION

WE is a medical emergency caused by thiamine deficiency. Thiamine is a cofactor for several enzymes in the Krebs cycle and the pentose phosphate pathway, including α-ketoglutarate dehydrogenase, pyruvate dehydrogenase and transketolase, and thus plays a vital role in carbohydrate metabolism. A decrease in their activity may lead to increased buildup of toxic intermediates. The buildup of intermediates that are unable to be metabolised may induce damage in areas of the brain where excess accumulation occurs.[3]

WE has an acute onset characterised by nystagmus, ophthalmoplegia, mental status changes and unsteadiness of stance and gait, although this triad is seen only in 16% of patients.[4 5] About 82% of patients have mental status changes according to autopsy-based series.[5] These changes occur due to involvement of thalamic or mammillary bodies and range from acute confusional state to mental sluggishness, apathy and inability to concentrate, and, if not treated, it leads to coma and death. Ocular abnormalities occurring in about 29% of patients include nystagmus, bilateral lateral recti palsy and conjugate-gaze palsies.[4]

In addition to the aforementioned predisposing factors, several kinds of cancer have been associated with WE including gastric carcinoma, malignant lymphoma, acute leukaemia and breast cancer. Increased thiamine consumption due to the rapid growth of cancer cells and inadequate nutrition due to nausea and anorexia from chemotherapy or malabsorption syndrome have been suggested as possible reasons for cancer-associated WE.[6 7]

The initial diagnosis of WE remains clinical because there is no specific routine laboratory tests available. The presumptive diagnosis of WE can be confirmed by determining blood thiamine concentrations or by measuring the erythrocyte transketolase activity.[8]

MRI is currently considered the most valuable method to confirm a diagnosis of WE. MRI shows symmetric involvement of the mammillary bodies, the tectal plate, the periaqueductal grey matter and the periventricular region of the third ventricle including the paramedian thalamic nuclei. Signal hyperintensities on T2-weighted sequences, FLAIR and diffusion-weighted images within the posteromedial thalami and surrounding the third ventricle are the most common abnormality described in patients with WE.[9 10] In the present case, the diagnosis of ADEM was initially considered based on the history of fever, vomiting, altered sensorium, gait ataxia and oculomotor deficits. MRI of the brain led us to reconsider the initial diagnosis, and ADEM was ruled out. MRI results also revealed that the lesions in ADEM are typically bilateral involving the deep and subcortical white matter; however, periventricular lesions are less common.[11]

Patients with suspected WE require immediate parenteral administration of thiamine. A recommended regimen is 500 mg of thiamine intravenously, infused over 30 min, three times daily for two consecutive days and 500 mg intravenously once daily for an additional 5 days, in combination with other B vitamins.[12]

To conclude, acute WE continues to be a rare but life-threatening condition often overlooked in the non-alcoholic population, resulting in the further progression of an easily treatable condition. Treatment should be initiated at the earliest possible time to avoid persistent brain damage. The prognosis of WE is favorable if diagnosed and treated early, as shown in our patient who was treated with thiamine and made a remarkable recovery.

Learning points

- Although most prevalent in alcoholics, Wernicke's encephalopathy (WE) may occur in anyone who develops a nutritional deficiency of thiamine.
- It is important to consider WE in patients with gastrointestinal tract cancer presenting with acute neurological symptoms such as ataxia, altered sensorium and oculomotor abnormalities and who are in a state of malnutrition.
- Early diagnosis and timely administration of thiamine will help prevent the devastating consequences of a treatable disease.

Contributors CUK prepared the manuscript. CUK, SKN and SR were involved in the diagnostic workup and management of the patient.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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