

# Non Alcoholic Wernicke's Encephalopathy with Cortical Involvement in a Patient of Active Peptic Ulcer Disease

Ghosh GC\*, Chatterjee K and Sharma B

PGIMER and Dr RML Hospital, New Delhi, India

## Abstract

We report a non-alcoholic man with Wernicke's encephalopathy with history of pain abdomen and vomiting. MRI showed features consistent with Wernicke's encephalopathy. Upper gastrointestinal endoscopy showed duodenal ulcer. We propose that peptic ulcer disease can cause thiamine malabsorption and Wernicke's encephalopathy. A diagnosis of peptic ulcer disease may merit prophylactic thiamine therapy.

**Keywords:** Wernicke's encephalopathy; Non-alcoholic; Peptic ulcer disease; Duodenal ulcer

## Introduction

Wernicke's encephalopathy is an acute neurological syndrome due to thiamine deficiency. Though classically described in relation to chronic malnutrition associated with alcoholism, it can also be precipitated in non-alcoholics due to various causes like gastrointestinal surgeries, recurrent vomiting, peptic ulcer disease, cancer chemotherapy and peritoneal or hemodialysis [1]. As the classical triad of confusion, ophthalmoplegia and ataxia is rarely present; a high index of clinical suspicion has to be maintained in non-alcoholics for the early diagnosis of Wernicke's encephalopathy. An early diagnosis and prompt institution of thiamine replacement leads to improvement in symptoms and prevents long term sequelae. We present this case to highlight peptic ulcer disease as a cause precipitating Wernicke's encephalopathy.

## Case Report

A 35 year old man, farmer by occupation presented with global confusion for 3 days with unsteadiness of gait. There was history of pain epigastrium and vomiting 3-4 episodes per day for past 15 days. There was no history of seizure, double vision or blurring of vision. There was no history of fever, headache, trauma, drug intake or exposure to toxins. There was no past history of jaundice, no history of diabetes mellitus or hypertension. Patient had no history of alcohol intake. On detailed clinical examination patient was drowsy and not oriented to time, place or person. (Glasgow coma scale score was E4V2M6). Signs of meningeal irritation were not present. Patient had nystagmus on bilateral horizontal gaze that increased on fixing the gaze otherwise cranial nerve examination was within normal limits. Motor and sensory system examination was within normal limits. Gait ataxia and truncal ataxia was present.

We evaluated the patient along the lines of acute encephalopathy. Random blood sugar was 105 mg/dl, SpO<sub>2</sub> was 98%, serum sodium was 145 meq/L, serum potassium was 3.8 meq/L, serum ammonia was 25 mcg/dL. Total leukocyte count was 5400/cu.mm, haemoglobin was 12.7 g/dl. Serum magnesium was 2.1 mg/dL. Serum thiamine levels were not done due to financial constraints. Serology for HIV was non-reactive, VDRL was non-reactive. Thyroid function tests profile was normal. Keeping in mind the presenting complaint of global confusion and ataxia with nystagmus in an afebrile patient, we made a provisional diagnosis of Wernicke's encephalopathy and empirically started intravenous thiamine 500 mg TDS. After 24 hours patient became conscious, oriented to time place and person, ataxia and nystagmus was still present. MRI brain (Flair T1W and fast spin echo T2W) done on the second day of admission revealed white matter hyper-intensity in

bilateral parietal region and left occipital region with additional hyper-intensity seen in periaqueductal white matter in brainstem, bilateral basal ganglia and thalami were normal. On the fifth day of admission patient had no residual ataxia but nystagmus was still present. An upper gastro-intestinal endoscopy was done which revealed duodenal ulcer (Figure 1).

## Discussion

Wernicke's Encephalopathy (WE) is an acute neuropsychiatric disorder which arises as the result of an inadequate supply of thiamine to the brain. It can occur in the context of inadequate dietary intake, and is also seen in a number of medical conditions associated with excessive loss of thiamine from the body, or impaired absorption of thiamine from the intestinal tract [2]. The present patient was diagnosed as a case of Wernicke's encephalopathy based on a compatible clinical presentation, neurological signs which rapidly improved after institution of thiamine therapy and typical MRI findings. Common clinical scenarios leading to Wernicke's encephalopathy like alcoholism, poor nutrition, gastrointestinal surgeries, cancer chemotherapy, total parenteral nutrition, dialysis therapy were ruled out from history.

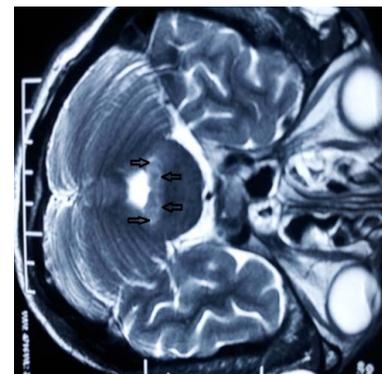


Figure 1: Upper gastro-intestinal endoscopy.

\*Corresponding author: Gopal Chandra Ghosh, PGIMER and Dr RML Hospital, New Delhi, India, Tel: 011 2336 5525; E-mail: [gcgghosh86@gmail.com](mailto:gcgghosh86@gmail.com)

Received May 08, 2014; Accepted June 26, 2014; Published June 28, 2014

**Citation:** Ghosh GC, Chatterjee K, Sharma B (2014) Non Alcoholic Wernicke's Encephalopathy with Cortical Involvement in a Patient of Active Peptic Ulcer Disease. J Clin Case Rep 4: 379. doi:10.4172/2165-7920.1000379

**Copyright:** © 2014 Ghosh GC, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Patient had history of multiple episodes of vomiting prior to onset of neurological symptoms. While recurrent vomiting associated with hyperemesis gravidarum and other causes has been implicated as an etiology of Wernicke's encephalopathy, the intensity and duration of vomiting in the present patient was not significant enough to precipitate Wernicke's encephalopathy. Upper gastrointestinal endoscopy showed the patient to be suffering from a duodenal ulcer.

Thiamine taken orally is known to be absorbed primarily from the duodenum, gastric absorption is also hypothesized to play a role. Proton pump inhibitors and antacids taken by peptic ulcer disease patients also decreases thiamine absorption by causing hypo magnesemia [3,4] and decreasing luminal acidity [5].

MRI is the radiological investigation of choice to support a clinical diagnosis of Wernicke's encephalopathy. Symmetric alterations in the thalami, mamillary bodies, tectal plate, and periaqueductal area are lesions typical of Wernicke's encephalopathy. Atypical MRI features like symmetric alterations of the cerebellum, vermis of cerebellum, red nuclei, dentate nuclei, caudate nuclei, cranial nerve nuclei, splenium, and cerebral cortex are commonly seen in non-alcoholic Wernicke's encephalopathy [6]. In this patient it is probable that peptic ulcer disease lead to thiamine mal absorption which precipitated Wernicke's encephalopathy when oral intake was further decreased by vomiting. Due to the early institution of thiamine therapy based on clinical

diagnosis this patient had a good neurological recovery without residual sequelae. Physicians should be alert to the possibility of Wernicke's encephalopathy occurring in non-alcoholics without major co-morbidities. A case may also be made for institution of prophylactic thiamine therapy in patients diagnosed with peptic ulcer disease [7].

#### References

1. Fei GQ, Zhong C, Jin L, Wang J, Zhang Y, et al. (2008) Clinical characteristics and MR imaging features of nonalcoholic Wernicke encephalopathy. *AJNR Am J Neuroradiol* 29: 164-169.
2. Cook CC, Hallwood PM, Thomson AD (1998) B Vitamin deficiency and neuropsychiatric syndromes in alcohol misuse. *Alcohol Alcohol* 33: 317-336.
3. Epstein M, McGrath S, Law F (2006) Proton-pump inhibitors and hypomagnesemichypoparathyroidism. *N Engl J Med* 355: 1834-1836.
4. McLean J, Manchip S (1999) Wernicke's encephalopathy induced by magnesium depletion. *Lancet* 353: 1768.
5. Sechi G, Serra A (2007) Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. *Lancet Neurol* 6: 442-455.
6. Zuccoli G, Pipitone N (2009) Neuroimaging findings in acute Wernicke's encephalopathy: review of the literature. *AJR Am J Roentgenol* 192: 501-508.
7. Thomson AD, Cook CC, Touquet R, Henry JA; Royal College of Physicians, London (2002) The Royal College of Physicians report on alcohol: guidelines for managing Wernicke's encephalopathy in the accident and Emergency Department. *Alcohol Alcohol* 37: 513-521.