Wernicke's Encephalopathy in a Non-Alcoholic Patient with Pancreatic Pseudocyst: A Case Report

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ABSTRACT

A young male, with no known comorbids, with a history of vomiting and significant weight loss for five months due to subacute pancreatitis and pancreatic pseudocyst, presented with altered mentation and inability to walk.

He was initially managed on differentials of septic *vs.* metabolic encephalopathy but was later diagnosed and managed on the lines of Wernicke's encephalopathy (WE). Over time with thiamine replacement, his Glasgow coma scale (GCS) improved and right lateral nystagmus was completely resolved in a week. He was then managed for vomiting due to gastric outlet obstruction caused by a pancreatic pseudocyst over the course of the hospital stay and started tolerating orally over months in follow-up visits. This is the first case report of Wernicke's encephalopathy in a non-alcoholic patient with chronic malnutrition secondary to gastrointestinal obstruction caused by pancreatic pseudocyst from Pakistan.

Key Words: *Nutrition, Pancreatic pseudocyst, Thiamine, Wernicke's encephalopathy.*

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INTRODUCTION

Wernicke's encephalopathy (WE) is an acute neurological condition characterised by a classical triad of ataxia, ophthalmoplegia, and encephalopathy requiring urgent recognition and treatment initiation to prevent further neurological progression.[1,2] It occurs secondary to thiamine (vitamin B1) deficiency. Traditionally linked to alcohol misuse, other causes of WE include severe malnutrition which may be secondary to vomiting with underlying hyperemesis gravidarum or a gastrointestinal pathology, starvation, refeeding syndrome, anorexia nervosa, gastrointestinal surgery, or even dialysis.[3] The diverse symptoms of WE make it challenging to diagnose early, often leading to a late or misdiagnosis.[4]

CASE REPORT

A young male, with a history of exploratory laparotomy and ileostomy three years back (secondary to ileal perforation due to complicated enteric fever), status post-reversal of ileostomy after seven months, presented with a history of vomiting for five months. The vomiting was non-bilious, contained food particles, and occurred five to six times per day.

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He was initially admitted for a week at a government hospital, where he was managed conservatively along the lines of subacute pancreatitis due to pancreatic pseudocyst. An endoscopy was planned but was not performed due to the unavailability of the facility. He was then advised to follow-up on a clinical basis for further management.

The patient presented to us 15 days later with a history of persistent vomiting and gradual weakness for 15 days, inability to walk for five days, and altered mentation for two days. He had not passed any stool in 10 days but was passing flatus. He usually ate a soft diet involving rice and pulses and preferred liquids due to on / off vomiting, which were 2-3 episodes / day.

He had a history of betel nut addiction and undocumented weight loss in the past three months. He had never drunk any alcohol. On arrival at the emergency, the blood pressure (BP) was 92 / 56 mmHg and the pulse was 150 beats/min. He was afebrile and maintaining saturation on room air. General physical examination was remarkable for sunken eyes, cold peripheries, and increased capillary refill time (CRT). Systemic examination was unremarkable except for a previous midline abdominal scar, clear gut sounds, and a Glasgow coma scale (GCS) of 12 / 15 with E4 V3 M5. He was oriented to person, but not time and place. Cranial nerves were grossly intact, and speech was clear but irrelevant and fluent. The ocular examination could not be performed due to the disorientation of the patient, though the pupils were bilaterally reactive to light. Reflexes were equal and normal in all four limbs but the patient was unable to walk due to ataxia and followed commands intermittently.

The patient was managed with a differential diagnosis of gastric

outlet obstruction, subacute pancreatitis, electrolyte imbalance, and possible toxicity *vs*. brain disorders (septic *vs*. metabolic encephalopathy). Laboratory work-up showed a normal complete blood count, low sodium (132 mmol/L, Normal: 136-145 mmol/L), potassium (2.8 mmol/L, Normal: 3.5-5.1 mmol/L), and chloride (91 mmol/L, Normal: 98-107 mmol/L), which were replaced by IV fluids and potassium supplementation, the calcium was mildly raised (10.9 mg/dl, Normal:10.2 mg/dl) and magnesium was normal. The amylase was raised (142 U/L, Normal: <51 U/L) with normal liver function tests. The urine analysis and chest x-ray were normal. Serum ammonia was also normal. General surgery and gastroenterology were consulted for gastric outlet obstruction secondary to pancreatic pseudocyst. CT abdomen with pancreatic protocol showed findings of subacute pancreatitis with pseudocyst formation. The patient was kept nil by mouth and continued on IV hydration and was admitted to the Internal Medicine Department for further diagnosis and management.

On the floor, he was commenced on IV thiamine for possible WE. As serum thiamine level tests were unavailable, neurology was consulted, and MRI Brain + MRA + MRV was done which showed abnormal signals in the dorsomedial thalamus (Figure 1), periaqueductal area and mammillary bodies (Figure 2), consistent with radiological diagnosis of WE.

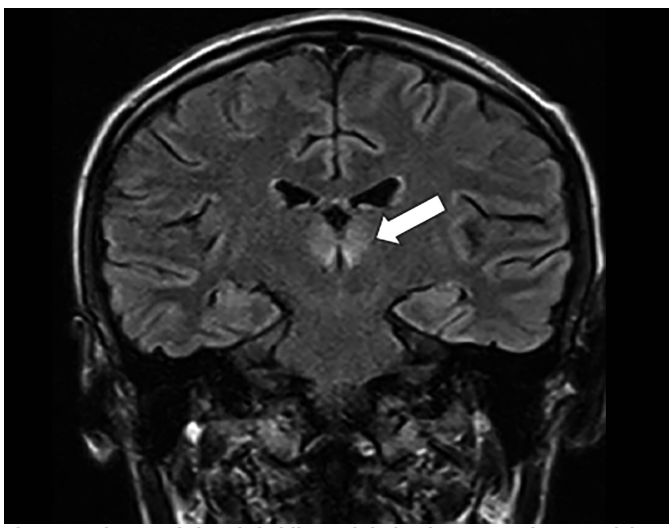

Figure 1: Abnormal signals in bilateral thalami representing Wernicke's encephalopathy (arrow).

After diagnosis, he was continued on thiamine replacement and screened negative for beriberi cardiomyopathy on echocardiography. The patient regained his baseline mental status within two days with the persistence of right lateral gaze nystagmus on ocular examination.

He was started on parental nutrition after a dietitian's review due to his inability to tolerate it orally. Upper gastrointestinal endoscopy revealed a distended stomach with significant residue and an external bulge was noted just proximal to the pyloric channel with luminal compression and endoscopy--guided nasojejunal (NJ) tubing was successfully inserted. NJ liquid feed was started which progressed to semi-solids. He was ambulated out of bed. WE resolved completely by the eighth day of admission along with the resolution of the lateral nystagmus.

He remained clinically stable and was discharged on NJ feed with follow-up in the clinic with a repeated CT scan of the abdomen to monitor the resolution of pancreatic pseudocyst. On subsequent follow-ups, he gradually started tolerating solid feeds. He is currently on follow-up every three months for further observation.

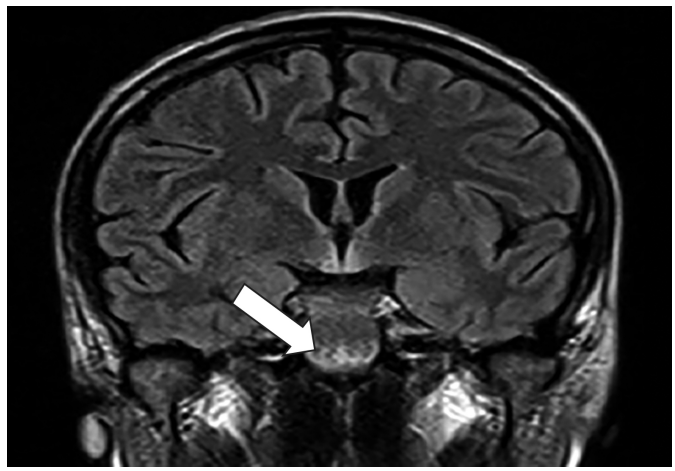

Figure 2: Abnormal signals in mammillary bodies representing Wernicke's encephalopathy (arrow).

DISCUSSION

WE is mainly a clinical diagnosis. Patients may not present with the classic triad which highlights why it may not be recognised at the early stages of presentation and may even remain undiagnosed till postmortem brain biopsy is done.[5]

A study done by Harper *et al*. showed that confusion is the predominant initial symptom, presenting in roughly 82% of the cases, followed by oculomotor abnormalities in 29%, gait ataxia in 23%, and polyneuropathy in 11%.[6] This is in agreement with the Caine criteria,[7] which is frequently used to identify WE. The presence of two or more of the four components confirms diagnosis including dietary deficiency or other risk factors like alcoholism or malnourishment, ocular signs, including abnormal eye movements, such as nystagmus (rapid, involuntary eye movements), or gaze palsies (inability to move the eyes in certain directions), cerebellar dysfunction, manifesting as ataxia (uncoordinated movements), unsteady gait, or tremors and fourth is either altered mental status or memory impairment.[8] The presented patient was a classic case of WE and he fulfilled all the four components of the Caine criteria.

MRI is a key radiological investigation used for the confirmation in situations where a diagnostic dilemma is present. Images will show hyperintense signals on T2 and FLAIR images with symmetrical involvement of the thalami, mammillary bodies, tectal plate, and periaqueductal areas. Atypical sites of involvement include the cerebellum, cranial nerve nuclei, and cerebral cortex.[9]

WE has been traditionally linked with alcohol intake but over the years, case reports have been published linking it to various causes including a complication of gastrointestinal surgeries,[10] gastric outlet obstruction secondary to gastric malignancies,[11] acute pancreatitis,[12] and occasionally, with a combination of the above risk factors due to poor parenteral nutrition intake, malnutrition and sepsis.[13] In the case of our patient, although he had experienced persistent vomiting for an extended period, his neurological symptoms were of relatively recent onset. He responded rapidly to intravenous thiamine replacement with complete resolution of neurological manifestations by the time of discharge.

In conclusion, there should be a low threshold of suspicion of WE in any chronically malnourished patient who presents with acute neurological symptoms, especially in low-middle-income countries (LMICs). Caine criteria of WE can be used along with an MRI brain to aid in the diagnosis of this rare disease entity. Prompt and timely treatment with high-dose thiamine therapy is crucial in the resolution of symptoms along with preventing its progression to Korsakoff syndrome.

PATIENT'S CONSENT:

Written informed consent was obtained from the patient for the publication of this case report and accompanying images after explaining the specific details of the data that will be shared and ensuring anonymity.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

RJ: Manuscript writing and literature review
AF: Drafted the discussion and literature review.
MAKP: Supervised and reviewed the final manuscript.
All authors approved the final version of the manuscript to be published.

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