

Wernicke's Encephalopathy in a Nigerian with Schizophrenia

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Abstract

While Wernicke's encephalopathy (WE) is a well-characterized syndrome in alcoholism and malnutrition, little is written of its prevalence or presentation in patients with psychiatric illness. We present a case of a 37-year-old Nigerian male with schizophrenia and malnutrition who presented with delirium and ophthalmoplegia. The clinical diagnosis was supported by dramatic reversal of the symptoms and signs following the administration of intravenous thiamine. Owing to the high rate of mortality and morbidity, WE should be considered in the evaluation of any patient with unexplained nystagmus, gaze palsies, gait ataxia, or confusion, especially if a condition associated with malnutrition is present. This is particularly important in psychiatric patients where the clinical history and syndrome may be obscured and treatment delayed.

Keywords: *Wernicke's encephalopathy, schizophrenia*

Résumé

Bien que l'encéphalopathie de Wernicke (WE) est un syndrome bien caractéristique de l'alcoolisme et de la sous-alimentation. Il y a peu d'écrits à propos de sa fréquence ou présentation chez des patients atteints de la maladie psychiatrique. Nous présentons un cas d'un homme nigérian âgé de 37 ans atteint de la schizophrénie et de la sous-alimentation qui s'est présenté avec delirium et ophtalmologie. Le diagnostic clinique a été appuyé par le renversement spectaculaire de symptômes et signes à la suite de l'administration intraveineuse de la thiamine. En raison de taux élevé de mortalité et de morbidité, le WE devraient être considérée dans l'évaluation de tous les patients avec une nystagmus inexplicée, regard palsie, giat ataxie, ou la confusion surtout si une condition liée à la sous alimentation est présent. Cela est particulièrement important chez des patients psychiatriques où l'histoire clinique et syndrome peuvent être obscur et traitement retardé.

Mots- cles: - *Encéphalopathie de Wernick, schizophrénie*

Introduction

Wernicke's encephalopathy (WE) is most commonly associated with heavy alcohol consumption,¹ but is also seen in other clinical settings such as starvation and hyperemesis gravidarum where malnutrition and vitamin deficiencies occur.¹⁻⁵ WE is a brain disorder involving loss of specific brain functions caused by thiamine deficiency and is characterized by the triad of ophthalmoplegia, ataxia and mental confusion. Thiamine deficiency damages regions of the brain, particularly the thalamus and the mammillary bodies. The mechanism by which thiamine deficiency leads to damage in these in these specific areas is not fully understood. Proposed mechanisms include altered cerebral energy metabolism resulting from decreases

in transketolase, pyruvate and acetylcholine; diminished nerve-impulse transmission at synapses; and impaired DNA synthesis.⁶⁻⁹ Variations in clinical presentations and the fact that not all patients with thiamine deficiency develop WE has raised the possibility that a genetic predisposition may exist in some patients. Data from autopsy series note the prevalence of non-alcoholic WE to range from 0.8% to 2.8%.^{10,11} In one report, 20% of cases were diagnosed before death.² Patients with psychiatric disorders often have poor dietary habits, malnutrition, and high prevalence of alcoholism, predisposing them to WE.¹² Wernicke's encephalopathy may be overlooked or obscured by psychiatric illness. We present a case of non-alcohol associated WE in a patient with chronic schizophrenia.

Case report

A 37-year-old Nigerian male with chronic schizophrenia of 9-year duration who refuses his medication of chlorpromazine for four months, had delusion that he was dying from a tumour and withdrew to his bed with neglect of his nutrition and subsequently loss of weight. He was admitted to hospital in a mute unresponsive state. He had a significant history of alcohol use as an adolescent but stopped drinking 10 years ago. He had no history of delirium tremens, seizures, tobacco or illicit drug use. Examination revealed normal temperature and blood pressure, blood pressure of 112/82mmHg, heart rate of 104/min and respiration 22/min. He was disorientated, agitated and emaciated. Pupils were equal, round and reactive to light, with marked limitation of bilateral vertical and horizontal gaze with no nystagmus. He had hypertonia but deep tendon reflexes could not be elicited and plantar respond was flexor because of mild polyneuropathy. He moves all four extremities but he was unable to stand or ambulate.

Laboratory studies revealed serum sodium of 130mmol/L, serum chloride of 92mmol/L, serum bicarbonate of 18mmol/L and serum creatinine of 152µmol/L. His mean corpuscular volume (MCV) was 92 fl, prothrombin time was 12 seconds, electrocardiogram and chest radiograph were normal. Serum aspartate transaminase (AST) was 15 U/L, alanine transaminase (ALT) 22 U/L, serum albumin 30 g/L and total protein 68 g/L. He was treated with intravenous 0.9% saline with 5% dextrose, thiamine and multivitamins.

He responded rapidly within four to six hours after the administration of 100mg of intravenous thiamine, with improvement in his mental status, dyskinesia, rigidity and ophthalmoplegia and a balanced diet was resumed as soon as possible. Flupentixol decanoate was initiated for his schizophrenia. The thiamine dose was maintained at 100mg/d throughout hospitalization and multivitamin tablets continued upon discharge.

Discussion

Wernicke's encephalopathy (originally called *polioencephalitis haemorrhagica superioris*) was first recognized by a German neurologist and psychiatrist Karl Wernicke in 1881 as a morbid neurological condition associated with thiamine deficiency and characterized by nystagmus, abducens nerve and conjugate gaze palsies, ataxia of gait, and mental confusion, and a predictable response to thiamine.¹³ It is a medical emergency and if not recognized and treated early is associated with progression to irreversible Korsakoff psychosis, with confabulation and anterograde memory deficits, and with 17% mortality.⁹

Clinically, we most often associate Wernicke's encephalopathy with alcohol consumption.¹⁻⁵ Wernicke's encephalopathy

producing an altered state may occur in malnourished psychiatric patients even in the absence of alcohol abuse.¹⁻⁵ While it is always difficult to completely exclude recent alcohol abuse as a contributor to his presentation, the observations of his family and coworker, and the absence of macrocytosis and normal values of serum transaminases make it less likely. The most commonly used biological markers of excessive alcohol consumption are mean cell volume (MCV) and gamma glutamyl transpeptidase (GGT). Of these, GGT is a better predictor than MCV it could not be assayed in our center. Our patient presented with decompensated chronic schizophrenia, acute delirium, ophthalmoplegia, malnutrition without evidence of trauma, or other medication toxicity and he had stopped drinking alcohol over a decade ago. Given the course of our patient's presentation of ophthalmoplegia, and mental confusion which all improved rapidly with IV thiamine, we feel this is most supportive of a diagnosis of non-alcohol associated WE.

Wernicke's encephalopathy is diagnosed when patients seek medical attention and have the classic trio of signs: mental confusion, eye movement disorder, and ataxia. Patients with schizophrenia are at greater risk for WE due to minimal self-care and homelessness, predisposing them to poor dietary habits, and malnutrition.¹⁴ Additionally, as the diagnosis of WE is primarily clinical it could be supported by neuroimaging or autopsy findings showing degeneration of the thalamus and mammillary bodies and loss of brain volume in the area surrounding the fourth ventricle-a fluid filled cavity near the brainstem. No test is diagnostic of WE and computed tomography is only 13% sensitive,¹⁵ it was not carried out on our patient. Data from autopsy series note the prevalence of non-alcoholic Wernicke's encephalopathy to range from 0.8% to 2.8%.^{6,7}

Owing to the high rate of mortality and morbidity, Wernicke's encephalopathy should be considered in the evaluation of any patient with unexplained nystagmus, gaze palsies, gait ataxia or confusion, especially if a condition associated with malnutrition is present. While this case of Wernicke's encephalopathy appeared in the setting of a patient with active psychiatric disease, we believe it highlights the importance of recognizing non-alcoholic populations at risk for thiamine deficiency and Wernicke's encephalopathy and carrying out a detailed neurological examination in such patients.

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