



A Case of Wernicke's Encephalopathy Responsive to Thiamine as Initial Presentation of HIV Seropositivity

Kamel E*, Yerokhin O and Ionete C

Department of Neurosurgery, University of Massachusetts Medical School, USA

Abstract

Wernicke's Encephalopathy (WE), classically seen in alcoholics has also been described in cases of AIDS with severe malnourishment. We would like to share our experience with an otherwise previously asymptomatic patient with no known WE risk factors who presented with acute truncal ataxia consistent with WE leading to the diagnosis of AIDS.

A 46-year-old woman presented for acute onset progressive gait unsteadiness and somnolence. She otherwise denied any disorientation, memory deficits, vertigo, weakness or numbness. She did not experience any weight loss or diarrhea and did not drink alcohol. Her only medical history was a chronic normocytic anemia and three episodes of left lower lobe pneumonia. She was not on any medications. Her full neurologic examination was unremarkable except for a pronounced truncal ataxia.

Neuroimaging did not show any acute abnormalities. As part of the workup for pancytopenia we performed HIV testing that was positive. The patient was empirically initiated on IV Thiamine from day 1 and steadily improved. She was safely discharged home after six days, requiring no assistance with ambulation.

Muri et al. evaluated the thiamine status in 55 HIV-positive patients without typical WE risk factors and found evidence of thiamine deficiency that did not correlate with the nutritional status or CD4 count. The pathophysiology of thiamine deficiency in asymptomatic HIV patients without any known WE risk factors remain unclear.

We would recommend keeping HIV infection in the differential in patients presenting with symptoms of Wernicke's encephalopathy even without any known risk factors.

Keywords: Wernicke's encephalopathy; HIV; Thiamine; Truncal ataxia

Introduction

Wernicke's encephalopathy (WE), classically described in alcoholics as a triad of encephalopathy, ophthalmopathy and truncal ataxia, has also been described in cases of severe malnourishment and malabsorption associated with chronic illness such as malignancy and acquired immunodeficiency syndrome. We would like to share our experience with an otherwise previously asymptomatic patient with no known nutritional deficiencies and no history of alcohol use who presented to our emergency department with an overall clinical picture consistent with Wernicke's encephalopathy with workup leading to the diagnosis of acquired immunodeficiency syndrome. We review whether this was mere coincidence or causality.

Case Presentation

A 46-year-old right-handed woman who emigrated from Ghana 27 years ago presented to our emergency department for acute onset progressive gait unsteadiness. The patient reports that symptoms started one day prior to admission when she noticed that she was excessively fatigued at the end of her work day in her office job. She was picked up by her husband around 5:00 p.m. and went home and napped, which she does not usually do after work and thereafter was noted to be stumbling while ambulating in the kitchen. When the patient's husband tried to help her up from a sitting position, she fell backwards and noted a feeling of lack of control of her position. She was able to make it to her bed and in the morning when she woke up at 6:30 a.m. was able to take a shower but needed to hold on to a railing for assistance. As the symptoms persisted, her husband drove her to the hospital for evaluation. She otherwise denied any disorientation, memory deficits, speech deficits, visual symptoms, vertigo, dysphagia, weakness, numbness, tingling, urinary or fecal

OPEN ACCESS

*Correspondence:

Edouard Kamel, University of Massachusetts Medical School, 55 Lake Avenue North Worcester MA 01655, USA,

E-mail: edouard.kamel@umassmemorial.org

Received Date: 25 Nov 2016

Accepted Date: 24 Jan 2017

Published Date: 26 Jan 2017

Citation:

Kamel E, Yerokhin O, Ionete C. A Case of Wernicke's Encephalopathy Responsive to Thiamine as Initial Presentation of HIV Seropositivity. *J Neurol Neurosurg Spine*. 2017; 2(1): 1008.

Copyright © 2017 Kamel E. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

incontinence or retention. There was no known history of recent infection, tick bite, recent travel abroad, or sick contacts. On further review of systems she would also deny any weight loss, night sweats, chills, rashes, headaches, cough, diarrhea, constipation, dysuria and hematuria. She reportedly does not drink alcohol, smoke, or use illicit drugs. The only known past medical history was chronic normocytic anemia and three episodes of left lower lobe pneumonia. She was not taking any medications and previously underwent two Cesarean section surgeries and tubal ligation.

On physical examination, she was anxious but fully oriented and alert; there were no memory or speech deficits, no cranial nerve abnormalities. She had full motor strength and no signs of appendicular ataxia. Her reflexes were symmetric without any spread or pathologic signs; there was no sign of any neuropathy or sensory abnormalities. She had pronounced truncal ataxia while sitting and standing that limited her ambulation to one or two steps even with assistance. We did not note any abnormalities on general examination including rash, lymphadenopathy, edema or organomegaly.

Pertinent diagnostic workup

BMI 22.14 Albumin 2.8 g/dl Total Serum proteins 8.1 g/dl AST 22 IU/L ALT 9 IU/L Alkaline phosphatase 23 IU/L total Bilirubin 0.3 mg/dl. Sodium 135 mmol/l Potassium 4.1 mmol/l Bicarbonate 22 mmol/l Creatinine 0.58 mmol/l BUN 9 mmol/l. Wbc 4.8 th/mm³ Hb 9.9 th/mm³ Platelet count 97 th/mm³. ESR 109 mm/hr.

Due to the concern for a neurovascular event she underwent a workup including a CT of the head which suggested subtle areas of hypodensity in the right pons, right thalamus and possible left thalamus, unclear if artifact versus small infarct. A follow-up CTA of the head and neck showed no abnormalities of her intracranial and cervical vessels. MRI of the brain with and without gadolinium the next day did not show any abnormalities. A repeat MRI that was done one month later did not show any change. The patient's initial CBC showed pancytopenia. As part of the workup, an HIV viral load was sent that showed 254,812 RNA copies per mL. Her absolute CD4 count was 86cells/microliter.

Therapeutics

The patient was empirically initiated on IV Thiamine 500mg on the day of presentation due to the finding of truncal ataxia and reports by the husband that the patient had poor appetite although significant weight loss was not reported.

Clinical course

The patient's ataxia steadily improved and by the fourth day after presentation she was ambulating independently without any deficits. She presented with very severe ataxia and was safely discharged home after six days, requiring no assistance with ambulation. The patient was kept on IV thiamine 500 mg three times daily throughout the duration of her hospital stay and was discharged with 100 mg oral thiamine daily.

Discussion

The clinical presentation of acute, rapidly progressive truncal ataxia with complete reversal of symptoms after several days of IV thiamine as the only therapy suggest a clinical diagnosis of Wernicke's

encephalopathy presenting primarily as truncal ataxia. Two brain MRIs, both on admission and one month after presentation, revealed no abnormalities and helped exclude any obvious alternative diagnosis. Other etiologies of acute cerebellar diseases such as infectious, post infectious or autoantibody associated ataxias would not typically resolve after only four days, especially without any therapeutics. The patient was not exposed to any chemotherapy or medications that could be responsible for her presentation. She did not have any additional features of heavy metal poisoning such as tremors, Parkinsonism or disorientation often described in heavy metals causing cerebellar ataxia such as mercury or manganese; nor was she at obvious risk for it. It is not unusual that Wernicke's encephalopathy presents with an incomplete picture and in fact the classical clinical triad is uncommon. Our patient does not use alcohol to the best of our knowledge, and although her husband attested to her poor appetite, she denied weight loss, her BMI was within normal limits (and did not fluctuate more than one point within that same year) and she did not have any signs or symptoms of malabsorption. It is worth noting that low albumin is a common finding in patients with inflammation being a negative phase reactant. She did test positive for HIV that was ordered for the workup of pancytopenia. WE has been previously described in the setting of AIDS, although typically involving cachectic and malnourished patients [1,2] and to our knowledge it has not been described in asymptomatic and undiagnosed patients. Of note, Muri et al. [3] evaluated the thiamine status in 55 HIV-positive patients in the outpatient setting by measuring erythrocyte transketolase activity and thiamine pyrophosphate. Eliminating patients with typical WE risk factors such as alcoholism, chronic diarrhea and vomiting, they nevertheless found evidence of thiamine deficiency that did not correlate with the patient's nutritional status or CD4 count. In light of these findings and with the knowledge that our patient did not have any classic risk factors for WE, we believe that the patient's HIV infection was the most likely cause of her symptoms. Unfortunately, we did not have a level of the serum thiamine at the time of presentation. Nevertheless, the pathophysiology of thiamine deficiency in asymptomatic HIV patients without any known malnutrition, alcoholism or malabsorption remains unclear.

There is not enough evidence to recommend testing every patient for HIV that presents with Wernicke's and no clear risk factors as there are no studies that address the epidemiology of HIV seropositivity in these patients in addition to the unclear cost effectiveness. Even so, we would recommend keeping HIV infection in the differential in patients presenting with symptoms of Wernicke's encephalopathy even without any known risk factors.

References

1. Larsen TR, Dragu D, Williams M. Wernicke's Encephalopathy: An Unusual Consequence of the Acquired Immune Deficiency Syndrome- Case Report and Literature Review. *Case Rep Med.* 2013; 2013: 709474.
2. Schwenk J, Gosztonyi G, Thierauf P, Iglesias J, Langer E. Wernicke's encephalopathy in two patients with acquired immunodeficiency syndrome. *J Neurol.* 1990; 237: 445-447.
3. Müri RM, Von Overbeck J, Furrer J, Ballmer PE. Thiamin deficiency in HIV-positive patients: evaluation by erythrocyte transketolase activity and thiamin pyrophosphate effect. *Clin Nutr.* 1999; 18: 375-378.