Acta Scientific Gastrointestinal Disorders(ISSN: 2582-1091)

Volume 2 Issue 9 November 2019

Case Report

Role of Hyperbaric Oxygen Therapy and Thiamine in Wernicke's Encephalopathy Secondary to Crohn's Disease: "Let there be Light"

Khalid N Abdelwali*, Iman Benelberhdadi, Anass Rahaoui, C Berhili, M Borahma, N Lagdali and Fatimazahraa Ajana

Department of Gastroenterology and Liver Diseases "C", CHU Ibn Sina, Morocco

***Corresponding Author:** Khalid N Abdelwali, Department of Gastroenterology and Liver Diseases "C", CHU Ibn Sina, Morocco.

Received: September 16, 2019; **Published:** October 16, 2019

DOI: 10.31080/ASGIS.2019.02.0091

Abstract

Wernicke's encephalopathy is an acute neurological problem resulting from thiamine deficiency and manifesting with mental confusion, oculomotor dysfunction, and ataxia. It is associated with alcohol dependence in adults. risk factors include hyperemesis gravidarum, prolonged diarrhea, prolonged parental nutrition without vitamin support, absorption disorders, cancer, and chemotherapy. Failure to consider the clinical findings and risk factors of this disease, can delay diagnosis. This report describes a case of Wernicke's encephalopathy developing in a 43 year-old patient with stenosing ileo-colonic and duodenal crohn's disease. The patient's clinical and cerebral magnetic resonance findings were compatible with Wernicke's encephalopathy. Although these are not widespread, typical ocular findings were present. Dramatic improvements were observed in clinical and cerebral magnetic resonance findings after exposure to 10 sessions of hyperbaric oxygen therapy.

Keywords: Encephalopathy; Crohn's Disease; Malabsorption

Introduction

The clinical triad in Wernicke's encephalopathy is mental confusion, oculomotor dysfunction, and ataxia. The condition was first described by Carl Wernicke in 1881. It is primarily associated with alcoholism in adults, and other preparatory factors include malnutrition, intestinal resection, hyperemesis gravidarum, cancer and chemotherapy, total parenteral nutrition without vitamin support, and hyperthyroidism. Thiamine deficiency leads to bilateral symmetrical signal changes in the medial thalamus, mammillary bodies, periaqueductal region, and fourth ventricle floor. Although symptoms may be reversible with early diagnosis and thiamine therapy, the mortality rate associated with delayed diagnosis is 10%. We describe the role of Hyperbaric oxygen in the ischemic and hypoxic brain injury complicating Wernick's encephalopathy in a 43 year-old woman suffering from iliocolic and duodenal crohn's disease. The use of HBOT enables the injured brain tissue to benefit from increased blood flow and oxygen delivery, which would act to metabolically or electrically reactivate the cells.

Case Report

43-year-old female patient with a history of ileocolic CD, benefited from right hemicolectomy and excision of 30-cm ileal segment. Inleximab therapy was started on October 2018.She was admitted to our department for sub-acute intestinal obstruction and epigastric pain.one week after hospitalization she experienced memory changes, progressive headache and drowsiness, oculomotor disturbances and ataxia with Glasgow coma score drop from 15 to 8. Brain CT angiography, lumber puncture and electroen cephalogram were without any significance, blood values were within the reference range, ENT and ocular examination showed benign paroxysmal positional vertigo (BPPV), and multidirectional nystagmus respectively. Brain MRI was compatible with Wernicke's encephalopathy (Figure 1,2). Thiamine level was measured in the blood and it was very low, parenteral 500 mg Thiamine t.i.d for 3 days and250 mg daily for the next 5 days given over 30 min diluted in 50–100 ml of normal saline then oral maintenance thiamine 100 mg p.o.t.i.d. was given as an outpatient treatment without clinical

improvement. We proposed a hyperbaric oxygen therapy (HBOT) for correction of the neural tissue hypoxia, brain ischemia, necrosis and edema. The patient was subjected to 10 sessions of HBOT with follow up after each session. One month later she showed a clinical improvement concerning her neurological manifestations. Creberal MRI was repeated after 3 months and showed a radiological amelioration with completer emission of the radiological signs of Wernicke's encephalopathy.

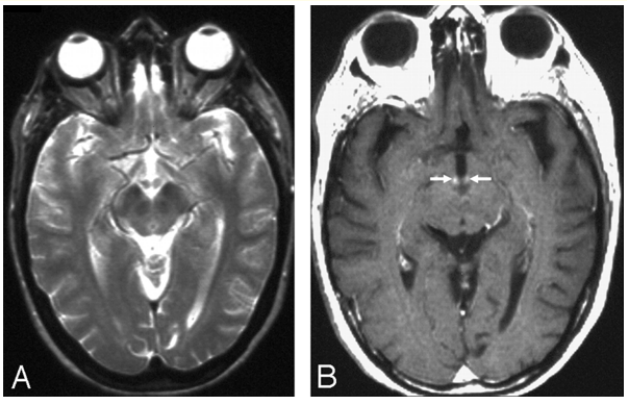

Figure 1: Hyperintense signal increases on T2 and fluid-attenuated inversion recovery images in both posteromedial portions of the thalamus, and in bilateral mammillary bodies.

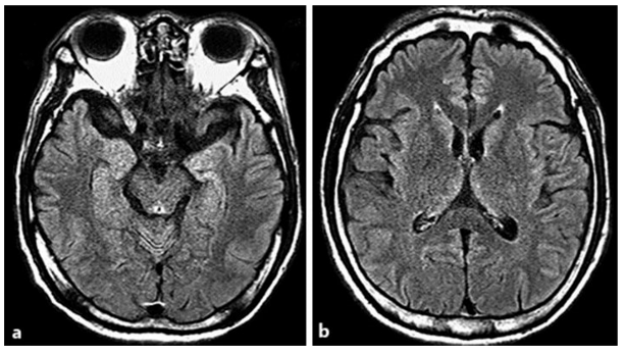

Figure 2: Complete remission and disappearance of radiological signs of Wernicke's encephalopathy.

Discussion and Conclusion

There is great interest in using hyperbaric oxygen (HBO) to treat neurological disease. The exquisite sensitivity of neural tissue to hypoxia makes increased oxygenation attractive as a therapy for disease processes that induce ischemia, edema, and, more recently, apoptosis.

Wernicke's encephalopathy is a picture of altered consciousness, ocular dysfunction, and ataxia resulting from thiamine deficiency. Thiamine pyrophosphate is an essential coenzyme required in the pentose phosphate pathway and the tricarboxylic acid cycle needed for the metabolism of carbohydrate and lipids. Thiamine deficiency thus leads to energy deficiency in the brain, metabolic acidosis, and cell death. The clinical triad of the disease is present in only 16% of patients. Patients may sometimes present with less specific findings, such as mild confusion, headache, and abdominal disorder. Initial findings may sometimes be only lethargy, loss of appetite, and concentration disorder. Progressive headache and dizziness were present in the early stages in our patient, later followed by impaired balance and visual problems and a disposition to somnolence. Brain MR imaging must be performed in all cases of suspected Wernicke encephalopathy. Symmetrical T2 signal increases are typically seen in the medial thalamus, mammillary bodies. Because delayed diagnosis is closely related to mortality, patients with suspected Wernicke's encephalopathy must be started on empiricthiamine therapy. HBOT could be introduced in association with thiamine replacement therapy. That the typical triad is initially not common must encourage physicians to be on their guard, especially with malnourished and anorexic patients undergoing intestinal surgery or having malabsorptive disease like our patient. Early diagnosis is important because findings can resolve with treatment. We observed complete radiological and clinical improvement in our case after thiamine replacement and HBOT [1-17].

Authors Contributions

- K.N.A: Case study design, formulation and clinical follow up
- I.B: Study revision
- A.R: Writing the paper draft
- F.A: Endoscopic assessment and final revision

Bibliography

1. British National Formulary (BNF 58) (2009).

2. Cook., *et al*. "B-vitamin deficiency and neuro-psychiatric syndromes in alcohol misuse". *Alcohol Alcohol* 33 (1998): 17-36.

3. Day E., *et al*. "Thiamine for Wernicke-Korsakoff syndrome in people at risk from alcohol abuse". *Cochrane Database System Review* (2009): CD004033

4. FlinkEB. "Therapy of magnesium deficiency". *Annals of the New York Academy of Sciences* 162 (1969): 901-905.

5. Kopelman MD., *et al*. "The Korsakoff syndrome: clinical aspects, psychology and treatment". *Alcohol Alcohol* 44 (2009): 148-154.

6. Lingford-Hughes AR., *et al*. "Evidence based guidelines for the pharmacological management of substance misuse, addiction and comorbidity: recommendations from the British Association for Psychopharmacology". *Journal of Psychopharmacology* 18 (2004): 293-335.

7. Victor M., *et al*. "The Wernicke-Korsakoff Syndrome and Related Disorders Due to Alcoholism and Malnutrition". Philadelphia: F.A. Davis Company (1989).

8. Martin PR., *et al*. "The role of thiamine deficiency in alcoholic brain disease". *Alcohol Research and Health* 27 (2003): 134-142.

9. Infante MT., *et al*. "Challenges in diagnosis and treatment of Wernicke encephalopathy: report of 2 cases". *Nutrition in Clinical Practice* 31 (2016): 186-190.

10. Zara G., *et al*. "Neurological complications in hyperemesis gravidarum". *Neurology Science* 33 (2012): 133-135.

11. Flabeau O., *et al*. "Hearing and seeing: unusual early signs of Wernicke encephalopathy". *Neurology* 71 (2008): 694.

12. Davies SB., *et al*. "Wernicke's encephalopathy in a non-alcoholic patient with a normal blood thiamine level". *Medical Journal of Australia* 194 (2011): 483-484.

13. Bohan PK., *et al*. "Wernicke encephalopathy after restrictive bariatric surgery". *The American Surgeon* 82 (2016): 73-75.

14. Caine D., *et al*. "Operational criteria for the classification of chronic alcoholics: identification of Wernicke's encephalopathy". *Journal of Neurology, Neurosurgery, and Psychiatry* 62 (1997): 51-60.

15. Galvin R., *et al*. "EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy". *European Journal of Neurology* 17 (2010): 1408-1418.

16. Scalzo SJ., *et al*. "Wernicke-Korsakoff syndrome not related to alcohol use: a systematic review". *Journal of Neurology, Neurosurgery, and Psychiatry* 86 (2015): 1362-1368.

17. Tallaksen CM., *et al*. "Concomitant determination of thiamin and its phosphate esters in human blood and serum by high-performance liquid chromatography". *Journal of Chromatography* 564 (1991): 127-136.

Volume 2 Issue 9 November 2019