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Case Report

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Psychogenic anorexia and non-alcoholic Wernicke's encephalopathy: Complete clinicoradiological recovery with thiamine

Anirban Ghosal[1]✉, Kajari Bhattacharya[2], A Shobhana[3]

[1]*Department of Neurology, Institute of Neurosciences, Kolkata, India*

[2]*Department of Neuroradiology, Institute of Neurosciences, Kolkata, India*

[3]*Department of Neurology & Stroke Specialist, Institute of Neurosciences, Kolkata, India*

ABSTRACT

Rationale: Prolonged undernutrition may arise out of depression and lead to Wernicke's encephalopathy if timely diagnosis and intervention are missed. Wernicke's encephalopathy is potentially treatable, and appropriate treatment may revert clinical depression and cognitive dysfunction to some extent.

Patient's concern: A 69-year-old female who had been taking escitalopram for one year developed tremor, ophthalmoplegia, ataxia, progressive cognitive decline, and convulsions.

Diagnosis: Non-alcoholic Wernicke's encephalopathy and hypomagnesemia due to psychogenic anorexia.

Interventions: High dose intravenous thiamine and magnesium were supplemented.

Outcomes: The patient showed remarkable improvement in neurological complications and even in depressive features.

Lessons: Wernicke's encephalopathy should not be ignored in the treatment of depression.

KEYWORDS: Wernicke's encephalopathy; Non-alcoholic; Treatment-resistant depression; Thiamine

1. Introduction

Wernicke's encephalopathy (WE) is characterized by the clinical triad of mental status changes, ocular dysfunction, and ataxia caused by thiamine deficiency. Most of the WE cases were found in alcoholics, thus the non-alcoholic form is mostly misdiagnosed due to lack of clinical symptoms[1,2]. Here we report a patient with non-responsive depression ultimately being diagnosed as WE with developing seizure and abnormal movements.

2. Case report

The study was approved by the Ethical Committee of Institute of Neurosciences, Kolkata, and informed consent has been obtained from the patient.

A 69-year-old lady referred to the emergency department with progressive confusional state, imbalance, trembling of hands and legs, and visual blurring for two weeks. Medical history showed that she was non-alcoholic and had no recent complaint of vomiting, abdominal pain, jaundice, or gastrointestinal surgery. However, she had severe apathy towards food for six months, lost 15 kg body weight, and now her weight was 45 kg. On the day of admission, she had two episodes of convulsion, and had significant word-finding difficulty, confusion, bilateral lateral rectus weakness, horizontal gaze nystagmus, intention tremor in upper limbs, and cerebellar ataxia. It is worth noting that she had been suffering from depression for one year since the death of her husband, therefore, she was on the treatment of 20 mg daily dose of escitalopram before the admission. Besides, She was normotensive, non-diabetic, afebrile, and had no meningeal sign, and the imaging of immediate brain CT was normal. Along with levetiracetam to control seizures, we started an intravenous magnesium supplement for the treatment of hypomagnesemia that was the only evident initial blood

✉To whom correspondence may be addressed. E-mail: dranirban.ghosal@gmail.com

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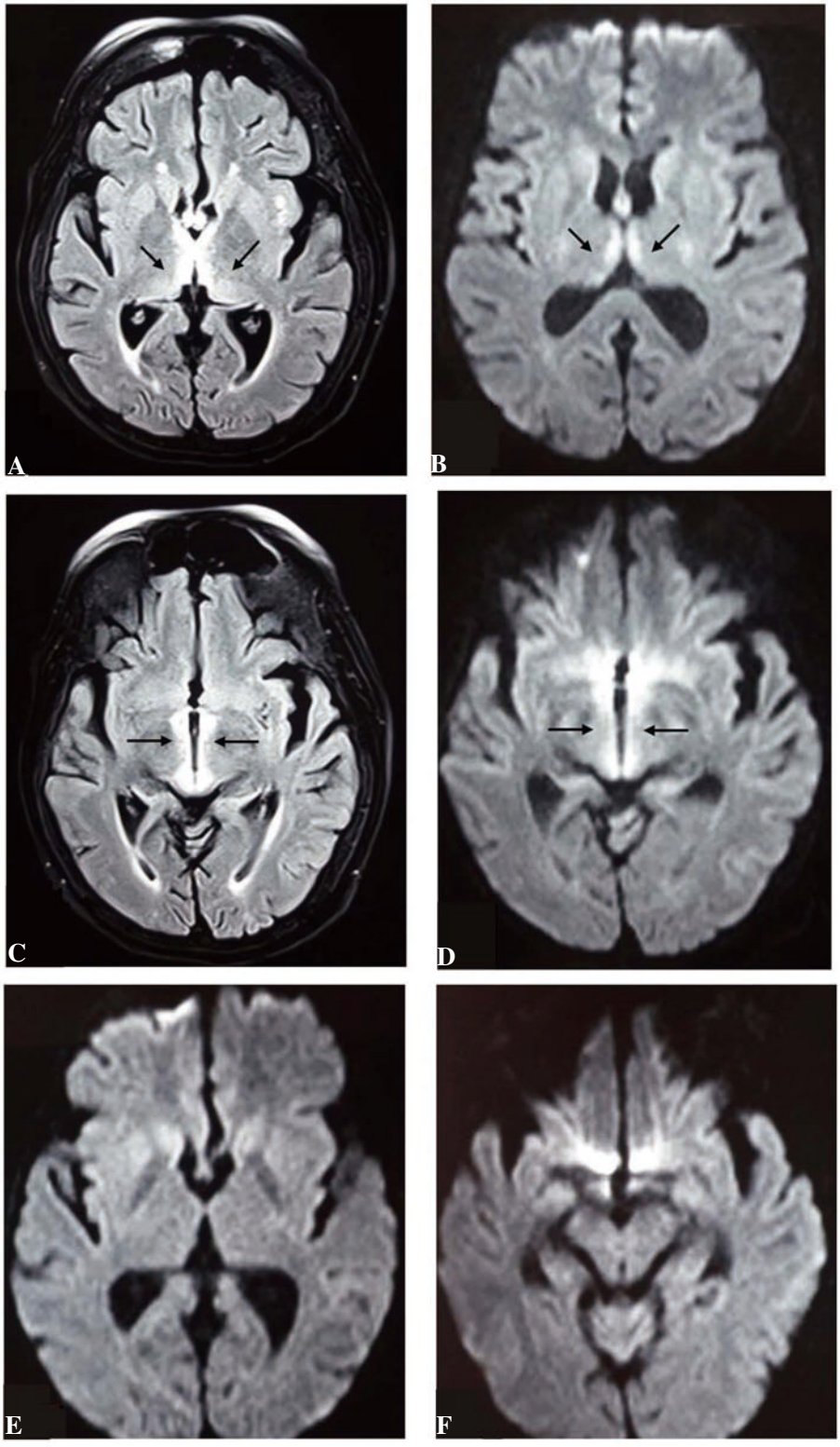

Figure 1. Pre-treatment axial MRI (FLAIR and DWI) of the brain. A and B: bilateral symmetrical hyperintensities and diffusion restriction in the medial thalami (arrows); C and D: periaqueductal grey matter (arrows); E and F: post-treatment axial DWI of the brain shows resolution of signal changes previously seen.

biochemical abnormality. Brain MRI revealed symmetric T2/FLAIR hyper intensities in periaqueductal grey matter, dorsomedial thalami, mammillary bodies, and lateral walls of 3rd ventricle suggestive of Wernicke's encephalopathy (Figure 1). High dose intravenous infusion of thiamine 200 mg diluted with 100 mL of normal saline, was given over 30 min 3 times daily. After 3 d of parenteral thiamine treatment, her blood test showed low haemoglobin, normal reticulocyte count, microcytic hypochromic red blood cells, low serum iron and ferritin, and high total iron-binding capacity suggestive of iron deficiency anemia. Her renal, liver function tests, and thyroid profile were normal. Serum alpha-fetoprotein, carcinoembryonic antigen, chest, and abdomen CT to screen for malignancy were all

normal. Nystagmus and lateral rectus weakness subsided, word-finding and comprehension was improved, and then the oral dose of thiamine was readjusted to 100 mg 3 times daily. Besides, oral iron and folate supplements were added. To further improve the patient's status, the dietician's help was sought. Seven days after the treatment, her cognition was better, and then psychotherapy sessions were started. After two months of physiotherapy and balance retraining, her gait was normal, and she could even do a tandem walk. By this time her dose of escitalopram was reduced to 10 mg per day as she was recovering from depression and apathy. Her appetite was improved, and her weight increased to 49 kg. A repeat brain MRI showed complete resolution of previous abnormalities.

3. Discussion

WE is characterized by the classical triad of mental status changes, ocular dysfunction, and gait apraxia caused by thiamine deficiency. Alcoholism is the commonest etiology, however, a non-alcoholic type may occur in nutritional deficiency states such as hyperemesis gravidarum, intestinal obstruction, gastrointestinal surgical procedures, starvation, magnesium deficiency, cancer, and chemotherapeutic treatments. Complete clinical triad is seen in only 10% of cases, leading to under-diagnosis of this potentially treatable entity. Our case had the complete clinical features triad. Notably, other symptoms include apathy, inattention, memory problems, loss of consciousness, seizures, and even coma can occur. Nystagmus and lateral rectus weakness are common ocular manifestations[1].

Thiamine deficiency may lead to swelling of intracellular space and local disruption of the blood-brain barrier. Brain regions with high thiamine turnover are more affected[2]. Shah *et al.* in a retrospective analysis of 50 nonalcoholic patients from northern India noted that recurrent vomiting was the commonest and initial symptom in the majority of the cases. Vomiting is believed to be both a cause and a manifestation of thiamine deficiency[2]. However, our case did not have any gastrointestinal symptoms including nausea or vomiting.

MRI revealed that typical sites of involvement are medial thalamic region, periaqueductal grey matter, mamillary body, tectum, and periventricular region seen as bilateral and symmetrical high signal intensity on T2 weighted sequences. The involvement of atypical sites like caudate head, cerebellum, cortical, and the subcortical region is more frequently seen in non-alcoholic WE cases. Otherwise, the involvement of atypical sites may indicate the progression of the disease, for example, the presence of cortical damage may be related to deep coma[2,3]. Our case did not have any atypical MRI feature of WE but had a favorable outcome.

Despite the treatment with high dose parenteral thiamine as per the European Federation of Neurological Societies guideline recommendation, most patients are left with residual gait disturbances and neurologic deficits including the persistence of memory problems[1]. However, our case had a complete recovery in all symptoms in two months. Magnesium supplementation in hypomagnesemia is important because magnesium serves as a cofactor for thiamine activity. It has a crucial role in the catalytic action of many enzymes, including thiamine pyrophosphokinase in the conversion of thiamine in its biologically active form, thiamine pyrophosphate[4].

Unusual cases of WE linked to prolonged deliberate religious starvation, anorexia nervosa, diet pill use, and an unbalanced diet have been reported[5,6]. Our case had prolonged undernutrition arising out of depression leading to WE. Consequently, her apathy worsened from WE leading to a further decrease in desire to take food, and it was a vicious cycle unless the proper diagnosis was established with brain MRI. Till then she was treated as a case of depression only.

In conclusion, this unique case emphasizes the need to keep WE as an underlying cause in the treatment of depression. As the knowledge of WE has progressed it is clear that many cases may even be MRI negative, and a therapeutic trial of thiamine is worth recommendation.

Conflict of interest statement

The authors report no conflict of interest.

Authors' contributions

A.G.: Clinical diagnosis, management of the case, article concept and authorship; K.B.: Imaging diagnosis of the case, and authorship; A.S.: Clinical diagnosis, management of the case and article concept.

References

[1] Sinha S, Kataria A, Kolla BP, Thusius N, Loukianova LL. Wernicke Encephalopathy-Clinical Pearls. *Mayo Clinic Proc* 2019; **94**(6): 1065-1072.

[2] Shah IA, Asimi RP, Kawoos Y, Wani M, Saleem T, Baba WN. Nonalcoholic Wernicke's encephalopathy: A retrospective study from a tertiary care center in northern India. *J Neurosci Rural Pract* 2017; **8**(3): 401-406.

[3] Santos Andrade C, Tavares Lucato L, da Graça Morais Martin M, Joaquina Marques-Dias M, Antonio Pezzi Portela L, Scarabôtolo Gattás G, et al. Non-alcoholic Wernicke's encephalopathy: broadening the clinicoradiological spectrum. *Brit J Radiol* 2010; **83**(989): 437-446.

[4] Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. Lancet Neurol 2007; **6**(5): 442-455.

[5] Panjwani S, Humayun M, Pirzada N. Curious case of starvation induced Wernicke's Encephalopathy (P6.315). *Neurology* 2017; **88**(16 Supplement): P6.315.

[6] Tóth A, Aradi G, Várallyay G, Arányi Z, Bereczki D, Vastagh I. Wernicke's encephalopathy induced by the use of diet pills and unbalanced diet. *Orv Hetil* 2014; **155**(12): 469-474.