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Wernicke's encephalopathy in a young psychiatric patient with delirium: a diagnostic challenge

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Summary

Wernicke's encephalopathy (WE) is an acute neurological disorder caused by depletion of thiamine and characterized by the clinical triad of ataxia, confusion and ophthalmoplegia. Thiamine deficiency is characteristically associated with severe alcohol use disorder. We present a rare case of WE in a non-drinker patient affected by obsessive compulsive disorder who presented a dramatic worsening of the clinical situation after the administration of clomipramine, underlining how brain Magnetic Resonance Imaging (MRI) scan can play a key role for the correct diagnosis and clinical management.

Key words: Wernicke's encephalopathy, brain MRI, delirium, obsessive compulsive disorder, thiamine deficit

Introduction

Wernicke's encephalopathy is a neurological disorder induced by depletion of thiamine due to different causes including alcoholism, gastrointestinal surgery, prolonged vomiting and dietary imbalance.

Thiamine plays an essential role in carbohydrate metabolism in the brain; periventricular structures are particularly affected as the blood-brain barrier is physiologically less tight and there is a high rate of thiamine-related glucose and oxidative metabolism. This leads to the typical clinical presentation: confusion, cerebellar ataxia and ophthalmoplegia.

If unrecognized, Korsakoff Syndrome or death may ensue [1].

Clinical history

A 27 year-old man with a diagnosis of Obsessive Compulsive Disorder (OCD) was admitted at our psychiatric first care service due to an acute anxiety episode in OCD.

Since two months he was having severe food restriction behaviors associated to an anxiety status based on intrusive thoughts. These restrictions brought to compromise his oral drug therapy for OCD (clomipramine) and the specific therapy for comorbidity (ulcerative colitis and hiccup).

At admission, blood tests showed abnormal values of RBC 4.10 x10^6/mL and low potassium 3.30 mmol/L. For the anxiety episode and considering the clinical picture, recovery of nutrition and intravenous therapy were started with clomipramine in glucose solution and chlorpromazine in physiologic solution. Potassium chloride (1,200 mg/day) was also administered.

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Conflict of interest
The Authors declare no conflict of interest.

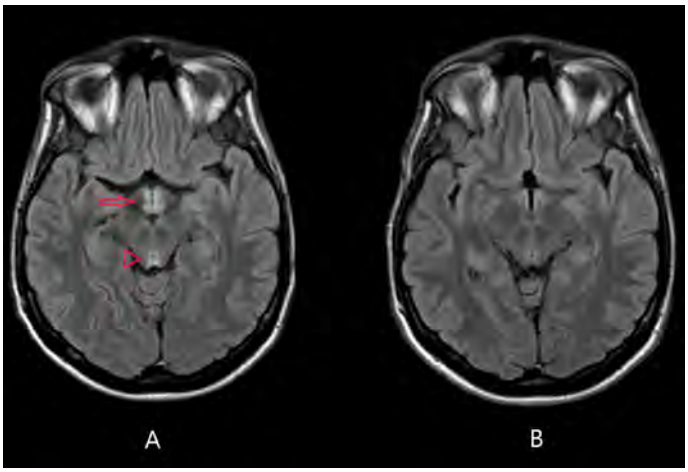

Figure 1. A) FLAIR weighted axial MRI images showed bilateral and symmetric hyperintensity at fornices and mammillary bodies (red arrowhead) and periaqueductal gray (red arrow); the same signal features were present also in the postero-medial portion of both thalami (not showed in the Figure); **B)** Image B: after 45 days of treatment brain MRI scan showed normal signal in those areas.

Suddenly he developed an acute delirium with disorientation, confusion and visual hallucinations (meeting criteria of DSM 5).
Hypotheses of an infectious process or central anticholinergic syndrome due to antipsychotics, were considered.
Pharmacological treatment was suspended, and brain MRI, EEG and urgent neurological examination were requested. Neurological exam and EEG were not specific. MRI showed a mild hyperintensity in long TR sequences involving symmetrically the postero-medial portion of the thalami, fornices, mammillary bodies and periaqueductal gray; a corresponding mild restriction of the molecular diffusivity in Diffusion-weighted imaging sequences was found. MRI findings were consistent with diagnosis of Wernicke encephalopathy (Fig. 1). Subsequent neurological control revealed ophthalmoparesis and ataxic gait supporting the diagnosis.
Administration of thiamine hydrochloride 100 mg/ml was set two times/day and cyanocobalamin 1,000 mcg 1fl/ weekly intramuscularly.
During the following 48 hours since the start of the new therapy, an improvement in the acute situation was observed with a clinical residual of slowing down and disorientation.

Discussion

WE is an acute neuropsychiatric syndrome characterized by the classic triad of ataxia, eye movement disorders, and mental status change. The incidence of WE is 0.6% of the population. The majority of patients that develop WE have a history of chronic alcoholism and accompanying malnutrition. In non-alcoholics patients WE onset is generally acute, rarely with ocular and cerebellar signs. Vomiting and a restrictive diet are the most frequent causes [1,2].
Intravenous administration of clomipramine (infusion in physiological solution, glucose and isotonic) is indicated in cases of severe obsessive-compulsive disorder when several therapeutic attempts do not lead to adequate results. The management of hiccups and vomiting through the correction of hypokalemia and intravenous administration of chlorpromazine is also indicated [3].
In our patient, immediately treated for the delirium in OCD with chlorpromazine and potassium, MRI made it possible to diagnose WE and set a correct therapy as is known the high sensibility of brain MRI for a correct and early diagnosis [4].
Reviewing the medical history of our patient and the first clinical setting it is possible to recognize several risk factors for WE development. He had a restrictive diet with predominant use of carbohydrate causing a relevant loss of weight in association with a possible malabsorption of nutrients, due to the chronic inflammatory disease of the patient; finally the administration of clomipramine with glucosal solution could have worsened the clinical setting as it is reported in literature in WE cases of pregnant women [5].
Immediate therapy with thiamine led to a functional recovery with a parallel improvement of the visible damage on MRI.

Conclusions

In conclusion, in our clinical practice, we have to know that WE could present without the typical clinical triad but just with delirium. Such an unusual clinical set must be considered in psychiatric patients as a diagnostic challenge.

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