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N. E. ELIMAM, L. V. PANTELEIENKO

Bogomolets National Medical University, Kyiv

Wernicke encephalopathy after chronic pancreatitis exacerbation. Case report

Wernicke encephalopathy (WE), is an emergency acute neuropsychiatric condition, which develops secondary to thiamine deficiency. According to data of multiple investigations, this disorder is greatly underdiagnosed. WE classically connected to chronic alcohol abuse, but also diagnosed in non-alcoholic patients with different risk factors including severe vomiting, chronic malnutrition, systemic infections, malignancies, chemotherapy, HIV/AIDS, gastrointestinal surgeries or disease, and renal problems.

We present a case of a 33-year-old woman with history of chronic pancreatitis exacerbation with prolonged vomiting, present to the neurological department with neurological symptoms consistent with WE. WE was suspected according to triad of symptoms, such as altered mental state, oculomotor disturbances and ataxia. Brain magnetic resonance imaging (MRI) and thiamine blood level aid us to confirm WE diagnosis. The patient was given thiamine, at the dosage of 200 mg 3 times per day intravenously for seven days, followed by oral thiamine. Gaze paresis disappeared at day 10, truncal ataxia noticeably improved after two weeks. The patient was left with some signs of anterograde amnesia, gaze-provoked nystagmus at the two month follow-up.

Physicians should be familiar with clinical presentation of WE in non-alcoholic patients with appropriate history and risk factors, like prolonged emesis, unbalanced diet, malignancies, etc. Presence of classic triad of clinical symptoms and pathological findings on brain MRI are the most important diagnostic tool, while the blood thiamine level may be within the normal range. Early diagnosis and therapy onset are crucial for patients to prevent further irreversible complications or death.

Keywords: Wernicke encephalopathy, thiamine deficiency, pancreatitis.

Wernicke encephalopathy (WE) is an acute, life-threatening reversible neuropsychiatric condition, which develops secondary to thiamine deficiency. Thiamine is a water-soluble vitamin, which acts as a coenzyme in glucose metabolism and neuronal activity, not being endogenously synthesized. Thiamine-dependent enzymes are involved in energy metabolism and biosynthesis of nucleic acids, and brain, highly dependent on ATP, is so vulnerable to thiamine deficiency [2]. The pathophysiology of thiamine-related disease is due to impaired glucose breakdown which lead to lactic acid production and affection of highly aerobic tissue, including the brain [3].

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Chronic alcoholism is the most common cause for WE, other risk factors include severe vomiting, chronic malnutrition, systemic infections, malignancies, chemotherapy, HIV/AIDS, gastrointestinal surgeries or disease, and renal problems [7]. We present a case of non-alcoholic patients with WE manifestations.

Case presentation

A 33-year-old white woman presented with a one week history of severely unsteady gait, inability to walk unassisted, and confusion for 2 days.

She had a one month previous history of loss of appetite, vomiting, upper abdominal pain, 6 kg weight loss, and generalized weakness because of

Пантелеєнко Лариса Василівна,
к. мед. н., доцент кафедри неврології
http://orcid.org/0000-0002-7342-2488. E-mail: panteleenko@hotmail.com

pancreatitis due to which she was undergoing inpatient treatment and was discharged 2 weeks ago.

Her other past medical history was unremarkable; she works as accountant, and had no significant family history. She denied use of alcohol, tobacco, or recreational drugs, and she was not taking any medications or vitamin supplements on admission. The patient has no known history of drug allergies or sexually transmitted diseases.

On physical examination, she was afebrile and there were no palpable lymph nodes, her blood pressure was 110/70 mm Hg and pulse rate 74/min. Neurological examination revealed slight confusion, disorientation to time and place, and short term memory loss, she showed horizontal nystagmus and bilateral ocular movement restriction to the lateral side, slight limb ataxia was found, but truncal ataxia was extremely profound, the patient couldn't stay and even sit without help. She had no focal weakness, numbness, reflex pathology or pelvic organ disturbances.

Complete blood count, serum glucose, renal and liver function tests were within normal limits. Thyroid study was normal. HIV serology was negative. The chest X-ray was normal.

Presence of altered mental state (AMS) and memory impairment, truncal ataxia, and oculomotor dysfunction was consistent with the triad of WE.

We performed a brain MRI which showed symmetrical high intensity in bilateral paramedian thalamic nuclei (Fig. 1), and periaqueductal areas (Fig. 2) on non-enhancing T2 and fluid-attenuated inversion recovery (FLAIR) sequence images, suggestive of WE.

Her thiamine blood level was 70 nmol/L (normal range: 82—239 nmol/L).

Thiamine, at the dosage of 200 mg 3 times a day, was supplied intravenously for seven days, followed by oral thiamine.

Lateral gaze paresis disappeared at day 10, truncal ataxia noticeably improved after two weeks. The patient was left with some signs of anterograde amnesia, gaze-provoked nystagmus at the two month follow-up.

Discussion

WE has an acute onset characterized by AMS, ophthalmoplegia/nystagmus and ataxia. The main etiology is thiamine deficiency; thiamine pyrophosphate (TPP) plays an important role as co-factor for several dehydrogenase enzyme reactions some of which are involved in energy metabolism, as the brain use ATP in abundance, brain is easily damaged in thiamine deficiency [3].

Our patient had been treated for exacerbation of chronic pancreatitis nearly a month before admission to the neurology department, during which she had multiple vomiting episode; she was stabilized with fluids and electrolytes, as well as glucose, but not with vitamins. After obtaining the medical history and performing necessary investigations, imaging was done. MRI is one of the most important diagnostic tool for early diagnosis and prognosis, the sensitivity and specificity of MRI has been reported at 53 % and 93 % with a positive predictive value of 89 %; in other words, MRI is better at confirming the diagnosis of WE than ruling it out [4]. Determining blood thiamine concentrations also helps in WE diagnosis confirmation.

Diagnosis was done according to European Federation of Neurological Societies (EFNS) guidelines [5].

Differential diagnosis of exclusion: brain encephalitis, including viral (we considered lumbar puncture, but after MRI high suspicion was for WE); toxic and other metabolic encephalopathy (no history, regular blood biochemistry and toxic screen were normal); brain stem stroke (not consistent with MRI findings); Miller-Fisher syndrome, excluded after MRI findings; hereditary cerebellar ataxias (in patients' case ataxia is mostly truncal, with subacute, not chronic development and MRI didn't show any cerebellar dysplasia).

WE after prolonged vomiting is reported in many cases including hyperemesis gravidarum [1], acute pancreatitis [6] and other vomiting related disorders.

Conclusions

This case present one of the potentially different medical background for WE. Physicians should be

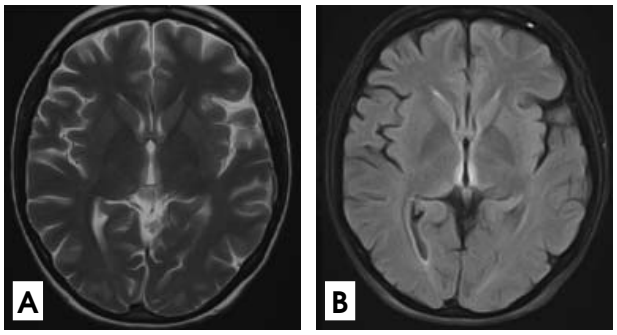

Fig. 1. Symmetrical high intensity in bilateral paramedian thalamic nuclei on non-enhancing T2 (A) and fluid-attenuated inversion recovery (FLAIR) (B) sequence images

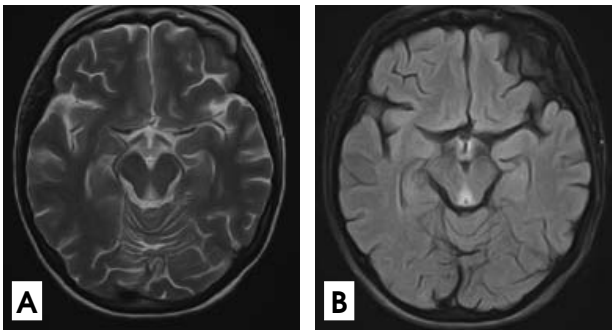

Fig. 2. Symmetrical high intensity in periaqueductal areas on non-enhancing T2 (A), and fluid-attenuated inversion recovery (FLAIR) (B) sequence images

able to notice the symptoms of WE in a non-alcoholic patient with appropriate history and risk factors, like prolonged emesis, unbalanced diet, malignancies, ets. Brain MRI and thiamine blood level help in confirming the diagnosis. Treatment delay may lead to irreversible brain damage or death.

Conflicts of interest: none.

Authorship contributions: conception and design, critical revision of the article, acquisition of data — L. P.; analysis and interpretation of data, drafting the article — N. E.

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Н. Е. ЕЛІМАМ, Л. В. ПАНТЕЛЕЄНКО
Національний медичний університет імені О. О. Богомольця, Київ

Енцефалопатія Верніке після загострення хронічного панкреатиту. Клінічний випадок

Енцефалопатія Верніке (ЕВ) — невідкладний гострий нейропсихіатричний стан, що виникає на тлі дефіциту тіаміну. За даними численних досліджень, цей розлад є недостатньо діагностованим. Зазвичай вона розвивається на тлі хронічного зловживання алкоголем, але також може виникнути у пацієнтів без алкогольного анамнезу, але з різноманітними чинниками ризику (тривале багаторазове блювання, хронічне недоїдання, системні інфекції, пухлини, хіміотерапія, вірус імунодефіциту людини/синдром набутого імунодефіциту, шлунково-кишкові захворювання та операції на шлунково-кишковому тракті, захворювання нирок).

Описано клінічний випадок 33-річної пацієнтки із загостренням хронічного панкреатиту з тривалим блюванням в анамнезі, яка була госпіталізована у неврологічне відділення з клінічними ознаками ЕВ. Останню запідозрили на підставі наявності тріади симптомів — когнітивні та окорухові порушення, атаксія. Магнітно-резонансна томографія і визначення рівня тіаміну в крові підтвердили діагноз. Пацієнтці призначили тіамін у дозі 200 мг тричі на добу внутрішньовенно протягом семи днів з подальшим пероральним прийомом. У результаті проведеного лікування окорухові порушення зникли на 10-й день, атаксія значно поліпшилася протягом 2 тиж. Під час контрольного огляду через 2 міс у хворої спостерігали незначні ознаки антероградної амнезії та горизонтальний ністагм при погляді в обидва боки.

Лікарі мають бути обізнаними з клінічними симптомами ЕВ у хворих, які не зловживають алкоголем, з відповідним анамнезом та чинниками ризику, такими як тривале блювання, незбалансована дієта, пухлини тощо. Наявність клінічної тріади симптомів та патологічні зміни на МРТ головного мозку є найбільш важливими діагностичними інструментами, тоді як рівень тіаміну в крові може бути в межах норми. Ранні діагностика і початок лікування ЕВ є надзвичайно важливими для запобігання незворотним ускладненням та смерті.

Ключові слова: енцефалопатія Верніке, дефіцит тіаміну, панкреатит.

ДЛЯ ЦИТУВАННЯ

Elimam N. E., Panteleienko L. V. Wernicke encephalopathy after chronic pancreatitis exacerbation. Case report // Український неврологічний журнал. — 2022. — № 1—2. — С. 60—62. http://doi.org/10.30978/UNJ2022-1-60.

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