ATYPICAL ANOREXIA NERVOSA- ENCEPHALOPATHY WERNICKE, NON ALCOHOLIC IN TEENAGE GIRL

CASE REPORT:

Z.BARAC-OTASEVIC, I. LJUTICA, S.KALAC

PSYCHIATRY CLINIC,
MEDICAL CLINICAL CENTRE MONTENEGRO, PODGORICA

A girl aged 14.6 years, 2 days before admission, started talking unrelated, was disoriented in space, disturbed, difficult to move, and her state of consciousness worsened aggressively, causing her to be admitted to the intensive care unit in Pediatric Clinic.

Intravenous hydration was initiated due to malnutrition, and a nasogastric probe was added. Upon examination of a psychiatrist at admission: neither verbal nor non-verbal communication was established, briefly disturbed, and wetted after re-wetting. Olanzapine 5,0 mg tablets in the evening with Sertraline 50 mg one tablet in the morning.

In a personal history: a girl has been treated for anorexia and depression since February 2017 since she lost over 30 pounds of body weight. She was treated psychiatric inpatient after the hospitalization was completed, she did not come for psychiatric control and did not take therapy.

Nutrition: is selective and generally takes what mom cooks.

The girl is usually a second child from another regular pregnancy and childbirth on time and regularly vaccinated for age. She has excelled at school, but in recent months she cannot attend school regularly. The first problems started in January 2017, with a whole headache that was intense and did not go for analgesics. Otherwise, she complained of headache in 2013 when brain MRI and MRA was performed and when an ectopic gray mass was observed in the parietal region in diameter. from 7mm.

At that time she had 62 kg . EEG findings as neat as a CT scan of the endocranium. Psychological testing of girl has high verbal abilities with a selective decline in the manipulative part of the scale. The personality profile was evaluated as conformist, in terms of potentiation of decency with suppression of aggression, which may lead to somatization. Recognized histrionic adjustment in stress. Individuation and separation conflict present. She begins to lose her appetite and from February 1st to April 23rd, 2017 she lost 15 kg.

Fear of weight gain was then described, a disorder of the body scheme then hospitalized at the Clinic for Neurology and Psychiatry for Children and Youth in Belgrade. Amenorrhea since february 2017, and had menarche since she was 13.

Her admission was in the intensive care unit. No severe general disturbance of consciousness disturbed verbal contact, no spontaneous movements of cachectic constitution, dry feathery pale petal skin and hematomas on the anterior upper chest, on the sacrum on the back of the left shoulder, and on the left anterior cristae and funnel decubitus. Body weight 30kg, body height 165cm. Head normocranial, pupil equal, circular, responsive to light. Extremities without edema and deformity. Neurological findings without spontaneous motions, no reflex, reflex Babine negative. Basic laboratory analyzes performed. MR examination of the endocranium corresponds to the reduction of supra and infratentorial white mass, Wernicke's non acute-phase encephalopathy and osmotic demyelination syndrome. Secretion in the right maxillary sinus.

Vitamin B1 administration begins. Left diaphragm paresis was registered and soon withdrawn, physical therapy was started. Neurological finding: Keeps the eyes open, does not blink, does not spontaneously cries at the light source, responds to painful stimuli with a crying, painful mimicry. There is an impression that the right rhyme is oculi somewhat cheesy. The limb muscle tone is elevated by a notch in the leg semiflexion in the extension of the contour of the hock. MTR is more pronounced on the lower extremities. Do not sit, stand static and supportive reactions absent.

At the beginning of 3 months of hospitalization, she begins to respond to the environment and smile. Neurological status: awake, accompanied by a glance of dropping voices. When crying, the face is symmetrical and unrestrained. The use of Benzodiazepine 2.0 mg twice a day with physical therapy is advised.

She starts walking with mid-5 month hospitalization and at the end of 5 month starts walking independently. Neurological finding: communicative orders understands and makes speech intelligible concludes a sentence. Extremity tone elevated, more pronounced to the right, in the course of circulation of the left leg circulation. Romberg negative.

The enteral nutrition is discontinued and the oral bolus nutrition begins. In the coming period, she gains in weight, which at discharge 170 days from the beginning of hospitalization is 47 kg, which is 17 kg more than on admission.

An EEG finding done that is neat. Give a tip for a hypercaloric nutrition. Continue therapy with Benzodiazepin 5 mg tablets twice a half tablet, Piracetam 400 mg twice a capsule, B vitamin complex 2 times one at a time. Endocranial control MR for 3 months

Wernicke encephalopathy (WE) is an acute neurological condition characterized by a clinical triad of ophthalmoparesis with nystagmus, ataxia, and confusion. This is a life-threatening illness caused by thiamine deficiency, which primarily affects the peripheral and central nervous systems. This should be differentiated from Korsakoff syndrome who is a neuropsychiatric disorder associated with memory disturbances in which there are significant deficits in anterograde and retrograde memory.

Although Wernicke encephalopathy mostly affects people who have a thiamine deficiency due to chronic alcoholism, various other causes include severe malnutrition, hyperemesis gravidarum, prolonged parenteral nutrition, malignancies, immunodeficiency syndromes, liver disease, hyperthyroidism, and severe anorexia nervosa.

Thiamine, one of the first B vitamins to be discovered also known as Vitamin B1, is a coenzyme that is essential for intricate organic pathways and plays a central role in cerebral metabolism. This vitamin acts as a cofactor for several enzymes in the Krebs cycle and the pentose phosphate pathway, including alpha-keto-glutamic acid oxidation and pyruvate decarboxylation. Thiamine-dependent enzymes function as a connection between glycolytic and citric acid cycles. Therefore, deficiency of thiamine will lead to decreased levels of alpha-keto-glutarate, acetate, citrate, acetylcholine and accumulation of lactate and pyruvate.

This deficiency can cause metabolic imbalances leading to neurologic complications including neuronal cell death. Neuronal death in the mammillary bodies and thalamus were implicated in multiple cases of Wernicke encephalopathy studied. Studies involving computed tomography (CT) and magnetic resonance imaging (MRI) of patients with Wernicke encephalopathy revealed lesions in the thalamus with dilated ventricles and volume loss in the mammillary bodies. The lesions are usually symmetrical in the midbrain, hypothalamus, and cerebellum.

Wernicke encephalopathy should be suspected in any patient with chronic alcohol abuse or any form of malnutrition and any of the following: acute altered mental status, ophthalmoplegia, ataxic gait, delirium, and hypotension. The classic triad of Wernicke encephalopathy is altered mental status, ataxic gait, and ophthalmoplegia.

The hallmark sign of Wernicke encephalopathy is ocular abnormalities especially nystagmus. Other oculomotor symptoms include cranial nerve involvement of oculomotor, abducens, and vestibular nuclei causing conjugate gaze palsies. Gait ataxia is also a significant finding in Wernicke encephalopathy where patients will present with a broad-based gait. Also, gait can worsen, and in many cases, patients are unable to walk. Physical examination may include a complete neurological exam with cerebellar testing. Disorientation and altered sensorium characterize encephalopathy.

Evaluation should include a thorough patient history with a focused physical exam and laboratory workup with appropriate imaging. There are no specific laboratory tests for diagnosing Wernicke encephalopathy as it is a clinical diagnosis with the above mentioned classic signs and symptoms.

The aim of treatment is prompt and quick correction of the thiamine deficiency in the brain. Wernicke encephalopathy is a medical emergency and considered a reversible condition, therefore, requiring immediate emergent attention although the onset of the disease may be acute or chronic. Parenteral administration of thiamine is most effective and provides for rapid administration, however, in some cases, there are persistent neurological deficits, and the acute condition can progress to chronic Korsakoff syndrome. All malnourished patient may need higher doses of thiamine. There is some evidence that thiamine treatment can improve the confusional state, quick resolution of ataxia, ophthalmoplegia, and nystagmus. Thiamine is generally administered before or together with glucose solutions because the glucose oxidation can decrease thiamine levels thereby exacerbating the neurological symptoms of Wernicke encephalopathy. WE is a serious medical disorder which carries an enormous morbidity and mortality. Even when the condition is managed with thiamine, the global confusion usually improves rapidly, but the ataxia and ophthalmoplegia may persist for some time. Patients who minimal or neurological signs have the best outcomes with thiamine supplement.

Instead conclusion:

The girl finished the first class of Secondary school. She goes to the psychotherapy once a week, and started swimming recreationally.

References

1. Oudman E, Wijnia JW, Oey MJ, van Dam MJ, Postma A. Preventing Wernicke's encephalopathy in anorexia nervosa: A systematic review. *Psychiatry Clin. Neurosci.* 2018 Oct;72(10):774-779. [\[PubMed\]](#)
2. Arts NJ, Walvoort SJ, Kessels RP. Korsakoff's syndrome: a critical review. *Neuropsychiatr Dis Treat.* 2017;13:2875-2890. [\[PMC free article\]](#) [\[PubMed\]](#)
3. Kim WJ, Kim MM. Wernicke's Encephalopathy Presenting with Bilateral Complete Horizontal and Downward Gaze Palsy in a Malnourished Patient. *Korean J Ophthalmol.* 2017 Aug;31(4):372-374. [\[PMC free article\]](#) [\[PubMed\]](#)