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CASE PRESENTATION

Wernicke-Kosarcoff syndrome. About a case

Síndrome de Wernicke-Kosarcoff. A propósito de un caso

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ABSTRACT

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Introduction: Wernicke-Korsakoff syndrome is an acute and reversible neurological disorder, consisting of two different brain disorders that often occur together: Wernicke's disease (or encephalopathy) and Korsakoff's psychosis (or syndrome). They are the result of brain damage that arises from the combination of chronic alcoholism and a deficiency of vitamin B1 (thiamine). The presented clinical case highlighted the necessity to recognize the condition, enabling the provision of treatment and the reintegration of the patient.

Objective: To describe Wernicke-Korsakoff syndrome as a severe brain disorder with reference to a case.

Case presentation: This is a 70-year-old male patient with a history of being an alcoholic and a chronic smoker. He came to our center because about 4 days ago he began experiencing a fever of 38-38.5 ◦C; in addition to the fever, he presented with vomiting and weakness. Upon receiving the case, the patient was found to be in a state of acute confusion, with involuntary movements that became more intense during his admission, accompanied by relaxation of the sphincters. It was decided to admit him to the intensive care unit for study and treatment. He was diagnosed at the Manuel Gonzales Díaz Hospitalization Services based on his clinical presentation, his history, and the additional tests performed. Following the general and pharmacological treatment applied subsequently, his progress was satisfactory.

Conclusions: The case presented highlights the importance of early diagnosis of this syndrome, as without treatment, the patient may become disabled, permanent memory loss may occur, and their life may be at risk.

Keywords: Thiamine Deficiency; Disliners; Encephalopathies; Korsakoff Syndrome; Thiamine

RESUMEN

Introducción: El síndrome de Wernicke-Korsakoff es un trastorno neurológico agudo y reversible, formado por dos trastornos cerebrales diferentes que a menudo ocurren juntos: la enfermedad (o encefalopatía) de Wernicke y la psicosis (o síndrome) de Korsakoff. Son el resultado del daño cerebral que surge de la relación del alcoholismo crónico combinado con la deficiencia de vitamina B1 (tiamina). El caso clínico presentado se evidenció la necesidad de reconocer el cuadro, lo que permite proporcionar tratamiento y reincorporar al paciente.

Objetivo: Describir el síndrome de Wernicke-korsakoff como trastorno cerebral grave a propósito de un caso.

Presentación del caso: Se trata de un paciente masculino de 70 años de edad, con antecedentes de ser alcohólico y fumador inveterado. Acudió a nuestro centro porque hace más menos 4 días comenzó con fiebre de 38-38,5°C, además de la fiebre presentó vómitos y decaimientos. Al recibimiento del caso se encontró; al paciente con estado confusional agudo, movimientos involuntarios que se volvieron más intensos durante su recepción, acompañados de relajación de esfínteres. Se decidió su ingreso en la sala de terapia intensiva para estudio y tratamiento. Fue diagnosticado en los Servicios de Hospitalización Manuel Gonzales Díaz a través de su cuadro clínico, sus antecedentes y los complementarios realizados. Mediante el tratamiento general y farmacológico aplicado posteriormente su evolución fue satisfactoria.

Conclusiones: El caso presentado resalta la importancia del diagnóstico temprano de este síndrome, pues sin tratamiento, el paciente puede quedar incapacitado, se puede producir pérdida permanente de la memoria y poner en peligro su vida.

Palabras clave: Deficiencia de Tiamina; Discinesias; Encefalopatías; Síndrome de Korsakoff; Tiamina

INTRODUCTION

Excessive alcohol consumption represents one of the main public health problems worldwide. This behavior acts as a risk factor, both direct and indirect, for the development of alcohol use disorders (AUDs). These, in turn, are frequently associated with malnutrition caused by vitamin deficiency, particularly thiamine (vitamin B1). [1,2]

Among the pathologies resulting from this deficiency, Wernicke-Korsakoff syndrome (WKS) stands out, a rare and severe neuropsychiatric disorder primarily caused by thiamine (vitamin B1) deficiency. This syndrome combines two closely related clinical entities: Wernicke's encephalopathy (WE), which has an acute onset, and Korsakoff's syndrome (KS), which has a chronic character, initially described in the 1880s by Carl Wernicke and Sergei Korsakoff, respectively. Subsequent research has proposed that both are different manifestations of the same pathophysiological process, with a common etiological basis. [3,4,5]

In alcoholic patients with thiamine deficiency, Wernicke's encephalopathy (WE)—the acute phase characterized by the classic triad of ataxia, ophthalmoplegia, and mental confusion—can progress to Korsakoff's syndrome (KS), a chronic phase dominated by amnesia and confabulation. This transition frequently occurs due to the lack of timely treatment. KS represents an irreversible neuropsychiatric manifestation, and its association with WE constitutes Wernicke-Korsakoff syndrome (WKS). [6]

WE predominantly manifests with the aforementioned symptomatic triad, with mental confusion being the most prevalent sign (82 % of cases). Other common findings include nystagmus, ocular motor disturbances (e.g., conjugate gaze palsy), and, in some cases, hallucinations or agitation. These atypical features often lead to diagnostic confusion, especially with conditions such as delirium tremens, psychosis, or metabolic disorders. [7,8]

This syndrome predominantly affects people with disorders that impair duodenal thiamine absorption, such as chronic alcoholism, its main etiology. It is also associated with gastrointestinal diseases, hyperemesis gravidarum, malignant tumors, bariatric surgery, and medical treatments such as chemotherapy. Its estimated prevalence ranges between 0,4 % and 2,8 %. [9,10]

Wernicke-Korsakoff syndrome (WKS) represents a neurological emergency that requires immediate diagnosis and treatment to prevent irreversible consequences. Although the classic triad (ataxia, ophthalmoplegia, and mental confusion) is highly suggestive of the condition—and early intervention can achieve complete reversal—its atypical presentation (seen in >50 % of cases) and symptomatic variability make its clinical recognition difficult. Neuropathological studies indicate that up to 80 % of cases are not diagnosed ante mortem, particularly in at-risk populations such as patients with alcohol use disorders. [11,12]

The limited scientific evidence and the paucity of epidemiological data on Wernicke-Korsakoff syndrome in Cuba prompted the presentation of the following case, with the aim of contributing to its knowledge in the field of medical sciences and encouraging future research in this area.

CASE PRESENTATION

A 70-year-old male patient, white, urban, living alone, with a history of chronic alcoholism and a smoker for over 30 years, and with no relevant family history of illness. He came to our emergency department at the Manuel González Díaz Polyclinic with Inpatient Services in Bahía Honda, Artemisa, because

MedEst. 2025 Vol.5; e341
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approximately four days ago he developed a fever of 38°C that occurred throughout the day without a specific time, accompanied by chills. He did not respond readily to antipyretics, and upon physical examination, he revealed nystagmus and truncal ataxia.

He was referred for further evaluation and treatment to the General Comandante Pinares Hospital in Artemisa Province, San Cristóbal Municipality. He did not complete the referral satisfactorily, and returned to our department a few days later, where he was then seen with involuntary movements, relaxation of the bladder sphincter, and an acute confusional state.

Physical examination of the respiratory system: patent airway, no tachypnea, no visible indrawing, no use of accessory muscles or paradoxical breathing, audible breath sounds, abundant transmitted sounds, respiratory rate: 22 bpm, partial oxygen saturation: 93 %, fraction of breath: 30 %. Cardiovascular system: no jugular vein engorgement, capillary refill time: less than three seconds, rhythmic heart sounds with good pitch and intensity, and no murmur. Heart rate: 80 bpm, blood pressure: 130/80 mmHg.

On physical examination of the nervous system, the patient was alert, presenting an acute confusional state that had been developing for several hours. He had a Glasgow coma score of 11 points, ocular response: 4 points, verbal response: 1 point, and motor response: 6 points. Anisocoric pupils: 2 mm on the right and 3 mm on the left, both reactive to light. Expressive aphasia, generalized rigidity, no myoclonus, no motor deficit, and no signs of meningeal irritation were present.

The abdominal examination revealed bowel sounds, tympany, a soft, depressible abdomen that was not tender on superficial and deep palpation, no masses, no retractions, and no relevant findings; hypocolored mucosa, no peripheral edema, and no Godet syndrome.

Laboratory tests were performed upon admission; These showed: hematocrit 0,44 (0,40-0,50), hemoglobin 12 g/dL (12-16 g/dL), uric acid 220 mmol/L (208-428 mmol/L), urea 6.3 mmol/L (1,7-8,1 mmol/L), creatinine 80 mmol/L (40-128 mmol/L), total protein 63 g/L (60-80 g/L). One blood glucose test was performed upon admission, and two consecutive fasting blood glucose tests were performed in the ward, which were around 6 mmol/L (4,2-6,1 mmol/L). All parameters showed values within or close to their normal ranges according to the reference values of the Polyclinic Laboratory Service.

Glucose monitoring showed persistent values around 6 mmol/L (VR 4,2-6,1) in three measurements (one upon admission and two fasting measurements), confirming that all parameters were within the normal ranges established by the institution's clinical laboratory.

In addition, three electrocardiograms were performed: one upon admission, which showed no signs or information relevant to the case other than an apparent enlarged left ventricle, and two subsequent ECGs in the operating room showed similar conditions.

The patient was given a semi-Fowler position at 30°, humidified oxygen by face mask at a rate of 9 liters per minute, continuous monitoring, a soft diet, intravenous parenteral hydration of 0,9 % physiological saline at a rate of 500 ml every 6 hours, ceftriaxone (bbo 1 g) at a rate of 1 intravenous bulb every 12 hours, hydrochlorothiazide (bbo 15 ml / 500 mg) 1 bulb every 12 hours, magnesium sulfate (1 g / 10 ml ampoule) 1 intravenous ampoule per day, Vitamin B12 (1000 mg) 1 intramuscular ampoule daily, Thiamine (500 mg - 5 ml) 1 cc intramuscular or intravenous, salbutamol aerosol every 6 hours plus an electrocardiogram and glycemia every 24 hours. The treatment administered confirmed the complete resolution of the condition

DISCUSSION

According to García-Maldonado et al. [13], vitamin deficiency can produce a wide range of alterations in the human body. Among these, B vitamins are generally capable of producing alterations in the central nervous system, temporarily or permanently affecting various areas of it. Thiamine deficiency has similar consequences; Wernicke-Korsarcoff syndrome (WK) is one of the best-known, a contradiction given how underdiagnosed it can be.

Vitamin B1 (thiamine) is an essential water-soluble micronutrient that acts as a key cofactor in multiple metabolic pathways, particularly in carbohydrate catabolism and the metabolism of branched-chain amino acids (BCAAs). Since the human body cannot synthesize vitamin B1 endogenously, it must be obtained exogenously through the diet. [14,15,16]

Alcohol consumption affects millions of people worldwide and is recognized as a source of potential neuropsychiatric complications. Excessive consumption is considered one of the main risk factors for depleting the body's thiamine reserves, as its storage lasts approximately 18 days to 6 weeks. WKD can also be caused by eating disorders due to strict or poorly prepared diets, chronic

MedEst. 2025 Vol.5; e341
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diseases such as cancer and its associated cancer treatments, prolonged vomiting such as bulimia, and organ transplants. (16,17)

Contemporary medical literature documents numerous atypical presentations of Wernicke-Korsakoff syndrome (WKS) associated with diverse etiologies beyond chronic alcohol consumption. These clinical variants, although they share the classic symptomatic triad (confusion, ataxia, and ophthalmoplegia), often manifest with different patterns of onset and severity depending on their underlying cause. However, all share a favorable prognosis when thiamine treatment is initiated early. (18)

Among the most relevant cases is one associated with hyperemesis gravidarum, where a patient presented with persistent nausea, uncontrollable vomiting, and peripheral vertigo that did not respond to conventional therapy. The diagnosis was confirmed by magnetic resonance imaging, which revealed characteristic thalamic lesions, allowing immediate initiation of parenteral thiamine administration, followed by complete recovery. This case underscores the importance of considering SWK in pregnant women with neurological symptoms associated with persistent vomiting.

Another paradigmatic case is a 23-year-old patient undergoing gastric bypass who developed an acute episode of exhaustible horizontal nystagmus toward the left hemifield 48 hours postoperatively, accompanied by lateralization of eye movements. Rapid initiation of intravenous thiamine therapy resulted in complete remission of symptoms within 72 hours. This example illustrates the risk of thiamine deficiency in the postoperative period of bariatric surgery and the need for prophylaxis in these patients. (18,19)

These clinical cases demonstrate that, regardless of the underlying etiology, the common denominator of SWK is the time-dependent nature of diagnosis and treatment. Magnetic resonance imaging is emerging as an essential diagnostic tool, particularly in atypical presentations, while immediate administration of thiamine remains the cornerstone of therapeutic management. These findings reinforce the importance of maintaining a high level of clinical suspicion in contexts of diverse nutritional risk beyond chronic alcoholism. (18,19,20)

The clinical picture is caused by the association of two entities: Wernicke's encephalopathy and Korsakoff's syndrome. Wernicke's encephalopathy is characterized by the triad of ataxia, ocular involvement, and confusion. Korsakoff's syndrome, on the other hand, involves disproportionate memory

impairment and states of mental confusion that can become irreversible. The link between the two entities is the level of impairment in thiamine levels. The condition usually begins with periods of memory loss and cognitive impairment. Over time, mitochondrial energy production becomes limited due to the thiamine shortage, continuing with an inadequate decarboxylation process, generating a chain reaction that affects Wernicke's area, the hippocampus, and the thalamus. (19,20)

The diagnosis of this condition is clinical, hence the importance of proper clinical method and correct medical procedure, although the definitive diagnosis is made by Nuclear Magnetic Resonance Imaging. Treatment within the first 48-72 hours is essential. Thiamine should be administered before glucose solution administration, as doing so could worsen the condition. The low cost and low adverse reactions justify its administration in patients with suspected alcoholism without a confirmed diagnosis. These conclusions were drawn by Sillero-Castillo et al. (20)

Alcoholic patients are at risk, for whom a comprehensive therapy should be designed, combining pharmacological treatment and rehabilitation. They should be referred to specialists who can support them in eliminating the habit and maintain their intake of multivitamins along the way.

CONCLUSIONS

This often underdiagnosed condition requires clinical evaluation and assessment. The potential irreversible complications of this disease, which affect the nervous system, require prompt and effective action in outpatient clinics and emergency departments. Community-based health education is essential, as is primary care, promoting healthy nutrition complete with vitamins and minerals to reduce the potential for these cases. Educating at-risk patients is essential.

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STATEMENT OF AUTHORSHIP

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JAGS: Conceptualization. Research. Formal analysis. Methodology. Writing the original draft and writing, review, and editing.

TPL: Research. Formal analysis. Funding acquisition. Research. Methodology.

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CONFLICT OF INTERESTS

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