CASE REPORT

Wernicke Encephalopathy in Anorexia Nervosa

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Abstract

Wernicke encephalopathy is an acute neuropsychiatric syndrome resulting from thiamine (vitamin B1) deficiency associated with significant morbidity and mortality. It has classically been described by the triad of altered mental status, ophthalmoplegia and ataxia. It has traditionally been associated with alcoholism, but it may also occur in patients with subacute or chronic conditions that increase metabolic demand or cause significant malnutrition. In this review, the authors report on a case of Wernicke encephalopathy in a 13-year-old girl with anorexia nervosa of the restrictive type. The diagnosis of Wernicke encephalopathy should be considered in anorexia nervosa whenever there is a mental status change, although the symptoms of the classic triad are not present. The authors emphasize the importance of early treatment with thiamine and recommended universal supplementation for patients with anorexia nervosa that meet the criteria for a high risk of refeeding syndrome.

Keywords: Adolescent; Anorexia Nervosa/complications; Portugal; Refeeding Syndrome; Thiamine Deficiency; Thiamine/therapeutic use; Wernicke Encephalopathy/diagnosis

Introduction

Anorexia nervosa is a psychiatric disease that often begins in adolescence[1] with a prevalence of approx. 0.5% in the general population.[2] It presents the highest mortality rate among psychiatric diseases[2,3] and can partly be justified by the multiple organic complications associated with severe states of malnutrition.[3] The therapeutic approach of anorexia nervosa involves nutritional strategies, in the most severe cases in a hospital ward, since refeeding syndrome can be associated.

Wernicke encephalopathy results from a thiamine (vitamin B1) deficiency and is characterized by the classic clinical triad of acute confusional state, ophthalmoparesis, and ataxia,[4,5-9] although these three symptoms are present concomitantly in only 16% of patients (Table 1).[4,8-10] Wernicke encephalopathy can lead to death or progress to the chronic and irreversible form of the disease, Korsakoff syndrome, characterized by mnesic changes and confabulation, which arises in 80% of surviving patients,[4,5,10] if there is no timely and appropriate therapeutic intervention.[7] Although traditionally associated with alcoholism, Wernicke encephalopathy has been associated with other conditions,[10] notably in the context of increased metabolism or significant malnutrition.[6,11] Despite this, Wernicke encephalopathy is still underdiagnosed in both the pediatric and adult age.[4]

The authors report on a case of Wernicke encephalopathy in a 13-year-old adolescent diagnosed with anorexia nervosa of the restrictive type.

Worldwide, the first case report with reference to a diagnosis of Wernicke encephalopathy in adolescents with anorexia nervosa was published in 2007.[6] Since that date, we have found two case reports in the literature, but none in Portugal,[11,12] and so this is the first published case in our country.

Case Report

A 13-year-old female adolescent went to the emergency room with abdominal skin lesions and marked weight loss. In the anamnesis, the existence of a weight loss of about 6 kg was observed in the previous two months, as a result of a restrictive diet and increased physical activity. She reported extreme sadness and the "desire to cry" (*sic*). There were no purgative behaviors. Although initially denying it, she would eventually confirm during hospitalization that she initiated weight loss because

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she considered herself "fat" (*sic*). She was described by her mother as "an exemplary girl, very adult for her age, well behaved, with a good school performance and winner of various school sports awards" (*sic*). She had no personal or family history of medical or psychiatric diseases. She had not yet experienced menarche.

At the physical examination, at admission, she was extremely underweight, with a weight of 30 kg, percentile (P) < 5 according to the weight curves for the age of the Center for Disease Control and Prevention, a height of 1.57 m, P 15-50 according to the height curves for the age of the World Health Organization (WHO), a body mass index (BMI) of 12.17 kg/m^2, P < 3, according to WHO BMI curves for age, a blood pressure of 103/67 mmHg, P < 90 for age, sex, and height, a heart rate of 38 bpm, respiratory rate of 40 cycles/minute, afebrile, with bleached and dehydrated mucous membranes, cheilitis and cold skin, hypertrophy of the salivary glands and a purpuric rash on the abdomen and thighs. Absence of lanugo. Filiform peripheral pulses. No other signs, namely edema, on examination. Tanner stage P5:M4.

Table 1. Clinical features of Wernicke encephalopathy

COMMON SYMPTOMS OR SIGNS AT PRESENTATION*
Ocular abnormalities (are one of the signs that can be found, **e.g.** nystagmus, conjugate gaze palsies, papillary abnormalities, retinal hemorrhages, ptosis, scotoma, diplopia, blurred vision, and photophobia).
Mental status changes (acute confusional state, apathy, indifference, incoherence in speech, indifference, disorder of memory, drowsiness, and coma).
Ataxia (is an example of various cerebellar signs that can occur – unsteadiness of gait, dysdiadochokinesis, impaired heel-shin testing, and dysarthria).
UNCOMMON SIGNS AND SYMPTOMS AT PRESENTATION
Stupor
Hypotension and tachycardia
Hypothermia
Bilateral visual disturbances and papilledema
Epileptic seizures
Hearing loss
Hallucinations and behavioral disturbances
LATE STAGE SYMPTOMS
Hyperthermia
Increased muscular tone and spastic paresis
Choreic dyskinesias
Coma

* 82% of patients have mental status changes,[4,9] 29% of patients present ocular abnormalities,[4,9] 23% of patients have ataxia,[4,9] only 16% of patients present the classic triad,[4,8-10] 19% of patients have none of the symptoms of the classic triad at the presentation of Wernicke encephalopathy.[4,9,10]

Adapted from Sechi G, Serra A. Wernicke's encephalopathy: New clinical settings and recent advances in diagnosis and management. Lancet Neurol 2007;6:442-55[4] and Isenberg-Grzeda E, Kutner HE, Nicolson SE. Wernicke-Korsakoff-syndrome: Under-recognized and under-treated. Psychosomatics 2012;53:507-16.[9]

In the diagnostic exams, she presented hypophosphatemia (2.8 mg/dL), with a normal potassium and magnesium value and a slight decrease in free thyroxine (T4) (0.77 ng/dL, reference 0.9-1.8 ng/dL), with a normal thyroid-stimulating hormone (TSH). Blood count and coagulation tests were normal. The electrocardiogram confirmed a 35 bpm sinus bradycardia with a prolonged PQ interval (397 ms), without other relevant abnormalities.

Once the hypothesis of anorexia nervosa with cardiac impairment was confirmed, the patient was hospitalized in the adolescent medicine unit, with a follow-up by a multidisciplinary team consisting of a pediatrician with differentiation in adolescent medicine, child and adolescent psychiatrist, and a nutritionist.

At the adolescent medicine unit, a feeding plan was initiated with a total of 1,050 kcal/day, oral supplementation with multivitamin (phytin 400 mg + glutamine 400 mg + thiamine 20 mg), administered twice/day, with the correction of hypophosphatemia on the sixth day of hospitalization. Caloric intake was progressively increased on the fifth, eighth, and tenth day of hospitalization, until 1,620 kcal/day.

Echocardiogram was performed and was normal.

On the 11th day of hospitalization, the patient initiated emotional instability that progressed slowly over the subsequent five days to a confusional state with behavioral changes, allopsychic disorientation, psychomotor lentification, impaired attention, concentration and recent memory, incoherent, sometimes repetitive speech, visual and auditory hallucinations, changes in the content of thought (delusional ideas of persecutory theme) and inversion of the sleep/wake cycle. This clinical picture presented a floating course. It did not show other neurological signs (including at the ophthalmologic and at the coordination/gait examination).

In the suspicion of an acute central nervous system (CNS) infection and/or hydroelectrolytic imbalance, a lumbar puncture was performed with cytochemical analysis of cerebrospinal fluid within the parameters of normality, negative virus and bacteriological examination. Analytical control, including B complex vitamin dosages the results of which were only available weeks later, showed no changes. Imaging tests - head computed tomography (CT) and head magnetic resonance imaging (MRI) - revealed a diffuse and significant enlargement of the grooves at the supra and infra-tentorial level. The electroencephalogram (EEG) showed no alterations.

Caloric intake was progressively increased to 1,830 kcal/day and 2,060 kcal/day, on the 12th and 15th days, respectively. On the 18th day of hospitalization, empirical treatment with intravenous (IV) thiamine, 500 mg/day,

risperidone, 0.25 mg twice/day *per os,* and IV acyclovir, 300 mg three times/day, was initiated.

The patient refused to take oral risperidone because she believed she was "being poisoned" (*sic*). Acyclovir was suspended on the fifth day of treatment after a negative result of herpes polymerase chain reaction test in cerebrospinal fluid. She took 500 mg/day of thiamine for 14 days (IV) (up to the 31st day of hospitalization), with progressive dose reduction (250 mg IV from 32nd to 37th days and 100 mg *per os* from the 37th day to the date of discharge).

The patient's clinical condition significantly improved on the third day of thiamine treatment, and psychomotor lentification was the symptom that persisted longer, remitting on the 12th day of treatment (29th day of hospitalization). During hospitalization, the patient presented progressive weight increase, having been discharged with a BMI of 15.3 kg/m^2 (P < 3 according to the WHO BMI curves for age). Skin purpuric lesions disappeared without any specific measure. Bradycardia and blood analysis alterations reversed with the therapeutic measures instituted and with weight gain. After discharge, she maintained follow-ups in the child and adolescent psychiatry, pediatrics-medicine of adolescents and nutrition clinics. There were no *sequelae* observed, namely psychological or cognitive, presenting a favorable evolution, with a clinical discharge about two years after the episode of hospitalization.

Discussion

Refeeding syndrome refers to a set of metabolic and hydroelectrolytic changes that may occur as a result of increased nutritional intake (oral, enteral, or parenteral) in markedly malnourished patients,[13-15] as one of the most severe complications during refeeding in patients with *anorexia nervosa.* Some criteria are identified for a high risk of this syndrome (Table 2).[16] It is characterized by electrolyte changes (hypophosphatemia, hypomagnesaemia, and hypokalemia) due to the movement of these ions into the intracellular space and/or changes in plasma volume (sodium and water retention) mediated by increased enteric glucose with a consequent increase in the production of insulin.[13-15] Refeeding syndrome can also occur as a result of acute thiamine deficiency.[14] Clinical manifestations vary depending on the changes involved - hydroelectrolytic imbalance and/or thiamine deficiency - and may trigger severe cardiac and/or neurological dysfunction and eventual sudden death (Fig. 1).[14,15]

Thiamine is a water-soluble vitamin, which in its metabolically active form, thiamine pyrophosphate, is an essential coenzyme for the action of numerous enzymes,[6,7,9,14] which act as intermediates in various metabolic pathways, whose final product is, among others, adenosine triphosphate, an essential energy source for all cellular metabolism.[7,9] The role of magnesium as a thiamine cofactor should be emphasized, playing a crucial role in the catalytic action of various enzymes.[4,9,10,17]

Thiamine is found primarily in metabolically active tissues, such as the heart, kidney, brain,[4,6,9,18] skeletal muscle system, and liver.[9,18]

Faced with a deficiency of this vitamin at the neuronal and glial cells level, various metabolic changes are expected with marked and progressive cellular consequences (Fig. 2).[4]

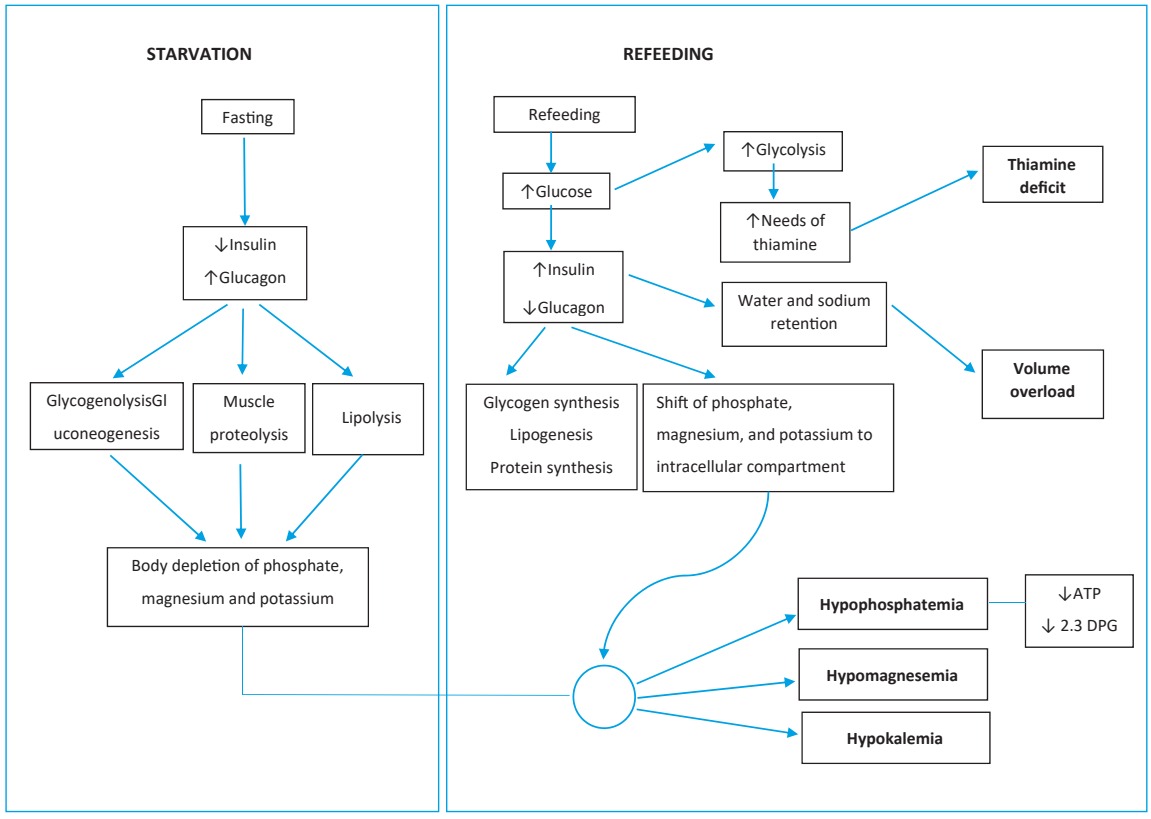
In the present clinical case, there might be low underlying thiamine reserves due to the chronic decrease in food intake because of anorexia nervosa. In addition, despite the gradual onset of nutritional intake, there has been an increase in carbohydrate intake, which has conditioned the increase in thiamine requirements. The patient presented acute confusional symptoms on the 11th day of hospitalization without any hydroelectrolytic changes that could justify the confusional state (hypophosphatemia observed at the beginning of the hospitalization was corrected since the sixth day of hospitalization by phosphorus supplementation). A complementary study was performed with EEG, head CT, head MRI, and lumbar puncture that excluded other etiological causes, namely acute CNS infections.

Although two of the clinical changes that are part of the classic Wernicke encephalopathy triad were not present, acute confusional symptomatology in a patient

Table 2. National Institute for Clinical Excellence (NICE) criteria for determining people at high risk of developing refeeding problems

Anorexia nervosa - people at **high risk** of developing refeeding problems
A. Patient has **one** or **more** of the following:
Body mass index less than 16 kg/m^2
Unintentional weight loss greater than 15% within the last 3-6 months
Little or no nutritional intake for more than 10 days
Low levels of potassium, phosphate, or magnesium prior to feeding
B. Or patient has **two** or **more** of the following:
Body mass index less than 18.5 kg/m^2
Unintentional weight loss greater than 10% within the last 3-6 months
Little or no nutritional intake for more than five days
A history of alcohol abuse or drugs including insulin, chemotherapy, antacids, or diuretics

Adapted from National Collaborating Centre for Acute Care. Nutrition support for adults: Oral nutrition support, enteral tube feeding and parenteral nutrition. London: NCCAC; 2006.[16]



REFEEDING SYNDROME

	Cardiovascular manifestations	Neurological manifestations	Others
Thiamine deficiency	Wet *beriberi*	Wernicke´s encephalopathy: ophthalmoplegia, confusion, ataxia and nystagmus.	
		Korsakoff´s syndrome, anterograde and retrograde amnesia	
		Dry *beriberi*	

Fluid overload	Cardiac failure		Edema
	Tachycardia		Acute lung edema
			Decreased hematocrit
			Low serum levels of albumin

Hypophosphatemia	Left ventricle dysfunction	Weakness	Rhabdomyolysis
	Cardiac failure	Paresthesia	Breathing muscles weakness
	Ventricular arrythmia	Lethargy	Hemolysis
		Confusion	Increased infection susceptibility
		Coma	
		Death	Tissue hypoxia

Hypomagnesemia	Electrocardiographic changes	Hyperreflexia	Hypocalcemia
	Cardiac arrhythmia	Tetany	
		Trousseau, Chvostek	
		Tremor	
		Fasciculations	
		Seizures, ataxia	
		Nystagmus	
		Vertigo	
		Abnormal movements	
		Muscle weakness	
		Apathy	
		Depression	
		Irritability	
		Delirium	
		Psychosis	

Hypokalemia	Electrocardiographic changes	Weakness	Breathing muscles weakness
	Cardiac arrhythmia		Constipation
			Paralytic ileus
			Fatigue
			Rhabdomyolysis

Signs and symptoms

Adapted from Martínez JJ, Moreno IH, Romero FB, López AH. Etiology and complications of refeeding syndrome in the UCI. New York: Springer; 2015.14
ATP - adenosine triphosphate; 2,3 DPG - 2,3 diphosphoglycerate.

Figure 1. Physiopathology and clinical manifestations of refeeding syndrome.

with a high risk of refeeding syndrome and the absence of hydroelectrolytic or other changes in the various diagnostic exams, it led to a high level of suspicion of Wernicke encephalopathy and, therefore, empirical treatment with thiamine was started.

There are no specific routine laboratory tests, changes in cerebrospinal fluid, EEG, or evoked potentials that are specific to the diagnosis of Wernicke encephalopathy, which remains a clinical diagnosis, dependent on a high index of suspicion.[4,7,9,10]

A head MRI may be useful if Wernicke encephalopathy is clinically suspected, as it is the most reliable diagnostic exam and can support the diagnosis.[4,10] Head MRI imaging studies in non-alcoholic Wernicke encephalopathy patients typically show bilateral and symmetrical changes,[4,10,19] in the mammillary bodies and periventricular encephalic regions (medial thalamus, periaqueductal region, and fourth ventricle floor).[4,10] Sensitivity is low (53%), specificity 93%,[4,7,8,10,20] with a positive predictive value of 89%.[8,20] The absence of typical lesions in a head MRI does not exclude the diagnosis[19] but their presence is highly suggestive, although not pathognomonic of this pathology.[7]

The presumptive diagnosis can be confirmed by the determination of a serum thiamine concentration or transketolase activity levels in erythrocytes.[4,7,10] However, in addition to the lack of these analyses in most hospitals, its sensitivity and specificity are low.

The diagnostic confirmation was initially supported by clinical improvement on the third day after parenteral thiamine administration was initiated and later, with continued treatment, by the complete resolution of symptomatology. Note that psychotic symptomatology is refractory to traditional psychopharmacological treatment and does not improve until thiamine deficiency is corrected.[12] In the present clinical case, the patient refused to take the oral antipsychotic, presenting the regression of delusional symptomatology with thiamine administration.

The failure to timely diagnose or properly treat Wernicke encephalopathy can lead to death (mortality rate of approx. 17%-20%)[4,5,7,10] or the progression of the pathological process to the chronic form, Korsakoff syndrome, marked by anterograde amnesia and confabulation.[5,7,10] Wernicke encephalopathy is, therefore, a medical emergency[4,10] and, if clinically suspected, the parenteral administration of thiamine should be initiated immediately as changes at the cellular level may be initially reversible.

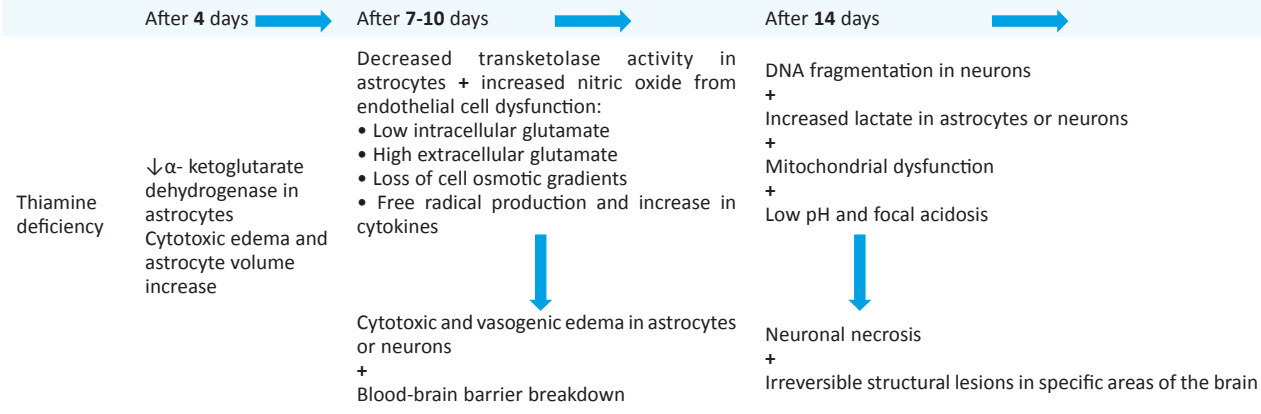
There is no consensus on the frequency, duration, or optimal dose of thiamine that should be given for both treatment and prophylaxis of Wernicke encephalopathy.[4,5,8,9,20] Several studies indicate, and appear to be unanimous, that replacement should be performed parenterally,[4-6,9,10,12,14,20-22] which enables a rapid increase of the thiamine levels in CNS by active uptake and mainly by passive diffusion through the blood-brain barrier,[4,6] safely[9,20] and associated with a low risk of side effects.[20,23]

For non-alcoholic Wernicke encephalopathy patients, 200 mg of thiamine (diluted in 100 mL 0.9% saline) is suggested as a 30-minute infusion at a frequency of three times/day for 2-3 days.[5,8,22]

If there is no clinical improvement, thiamine supplementation should be discontinued after 2-3 days.[4,22] If there is clinical improvement, treatment with 250 mg/day (intramuscular or IV) should be continued for 3-5 days or until clinical stabilization.[4]

Magnesium administration should also be considered as its severe deficiency may lead to a refractory response to thiamine administration.[4,9,10,22]

Due to the severity and challenge of a diagnosis of Wernicke encephalopathy due to thiamine deficiency in patients with anorexia nervosa in the context of refeeding syndrome, the authors propose a review



Adapted from Sechi G, Serra A. Wernicke's encephalopathy: New clinical settings and recent advances in diagnosis and management. Lancet Neurol 2007;6:442-55.4
DNA - deoxyribonucleic acid.

Figure 2. Proposed sequence of metabolic and morphological changes during thiamine deficiency.

of treatment recommendations for anorexia nervosa patients meeting the criteria for high refeeding syndrome risk (Table 2), considering the universal indication for prophylactic thiamine supplementation (*per os,* 100-250 mg/day, depending on the nutritional status and risk of Wernicke encephalopathy).

WHAT THIS CASE REPORT ADDS

- Thiamine deficiency should be considered in all patients at risk of malnutrition including those with psychiatric illness.
- Wernicke encephalopathy is not a rare entity, but it is underdiagnosed and the presence of all three symptoms of the classic triad in early clinical manifestations of the disease is uncommon.
- Clinical suspicion is critical for diagnosis as there is no specific diagnostic test.
- The importance of having knowledge of the risk factors is emphasized in order to be able to make an early clinical diagnosis, which is essential for a favorable prognosis.
- The authors propose a review of treatment recommendations for anorexia nervosa patients meeting the criteria for high refeeding syndrome risk (Table 2), considering the universal indication for thiamine supplementation.

Conflicts of Interest

The authors declare that there were no conflicts of interest in conducting this work.

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Consent for publication

Consent for publication was obtained.

Confidentiality of data

The authors declare that they have followed the protocols of their work center on the publication of patient data.

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Um Caso Clínico de Encefalopaia de Wernicke na Anorexia Nervosa

Resumo:
A encefalopatia de Wernicke é uma síndrome neuropsiquiátrica aguda que resulta do défice de tiamina (vitamina B1) e que está associada a morbilidade e mortalidade significativas. Caracteriza-se pela tríade clínica clássica de estado confusional agudo, oftalmoparésia e ataxia. Encontra-se tradicionalmente associada ao alcoolismo mas pode também ocorrer em doentes com patologias subagudas ou crónicas que aumentem o metabolismo ou que causem malnutrição significativa. Os autores descrevem um caso clínico de encefalopatia de Wernicke numa adolescente de 13 anos com uma anorexia nervosa tipo restritivo. O diagnóstico de encefalopatia de Wernicke deve ser considerado na anorexia nervosa sempre que se verifique um estado confusional agudo, ainda que os sintomas da tríade clássica não estejam presentes. Os autores realçam a importância do tratamento precoce com tiamina e consideram a suplementação universal a todos os doentes com anorexia nervosa que preencham critérios para alto risco de síndrome de realimentação.

Palavras-Chave: Adolescente; Anorexia Nervosa/complicações; Deficiência de Tiamina; Encefalopatia de Wernicke/diagnóstico; Portugal; Síndrome da Realimentação; Tiamina/uso terapêutico