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Journal of
Clinical & Medical Surgery

Case Series

Thiamine deficiency: A never ending story from the pediatric age till adulthood, two cases and review of diagnosis and treatment

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Article Info

Received: Jul 18, 2022

Accepted: Aug 12, 2022

Published: Aug 19, 2022

Archived: www.jclinmedsurgery.com

Abstract

Thiamine is an important vitamin for energy metabolism, which plays a critical role in many pathways. Thiamine deficiency can lead to irreversible consequences, particularly at the neurological level, and increases mortality at all ages. Typical manifestation of thiamine deficiency is Wernicke Encephalopathy, with the classic clinical triad (ocular abnormalities with nistagmus, ataxia and confusion). We report one pediatric and one adult case of neurological symptoms due to thiamine depletion, in patients undergoing abdominal surgery, with vomiting, reduced oral intake and undergoing supportive parenteral nutrition.

Based on their medical history, although with partial clinical manifestations, incomplete acquisition of thiamine was suspected, although its plasma levels were within normal limits.

Rapid clinical response after thiamine administration and regression of lesions on brain Magnetic Resonance Imaging (MRI) made it possible to diagnose Wernicke's encephalopathy. Wernicke's encephalopathy should be suspected in all patients, not just alcoholics, who are at risk of inadequate thiamine intake. In order to prevent thiamine deficiency it is important to ensure adequate daily vitamin supplementation during artificial nutrition support or at risk of inadequate intake.

Abbreviations: CRP C: Reactive Protein; CT: Computed Tomography; EFNS; European Federation of Neurological Societies; ERCP: Post-Endoscopic Retrograde Cholangio-Pancreatography; IM: Intramuscularly; IV: Intravenously; MRI: Magnetic Resonance Imaging; PN: Parenteral Nutrition; RDA: Recommended Dietary Allowance; WE: Wernicke Encephalopathy.

Citation: Toniazzo S, Verlato G, Duci M, Francini Pesenti F, Spinella P. Thiamine deficiency: A never ending story from the pediatric age till adulthood, two cases and review of diagnosis and treatment. J Clin Med Surgery. 2022; 2(2): 1036.

Introduction

Thiamine, or vitamin B1, is an essential nutrient to carbohydrate metabolism [1,2]. It acts as a cofactor in various enzymatic reactions, mainly with mitochondrial localization. The brain is highly vulnerable to thiamine deficiency due to its heavy reliance on mitochondrial ATP production. Thiamine deficiency causes several clinical conditions, from mild neurological disorders to severe encephalopathy, such as Wernicke Encephalopathy (WE), and even death. WE is an acute neurological and medical emergency characterized by a clinical triad of ocular abnormalities with nistagmus, ataxia and confusion [3]. It has been reported in individuals with various conditions, such as alcohol dependence, severe malnutrition, intensive care unit stays, prolonged parenteral nutrition, cancer, following gastrointestinal or bariatric surgery [4,5]. However, the disorder is greatly under diagnosed both in adults as in children [6].

In this report, we describe 2 cases of WE, one in a girl with intestinal obstruction in polytrauma and one in a woman with abdominal pain, nausea, vomiting and Post-Endoscopic Retrograde Cholangio-Pancreatography (ERCP).

Case presentations

Pediatric case report

A 14-years-old girl with intestinal obstruction in polytrauma, was transferred from a peripheral hospital to undergo to surgical resolution of the duodenal volvulus. Episodes of biliary vomiting and reduced oral intake have been reported in the previous 16 days, so parenteral nutrition with an all-in-one, three-chamber bag (Olimel N4E, Baxter S.p.A), 80 ml/h was started although without multivitamin supplementation.

From the first postoperative day, the girl resumed feeding, but given the persistence of episodes of biliary vomiting, personalized parenteral nutrition support with a multivitamin-supplementation once daily containing 3.51 mg of thiamine/dose (Cernevit, Baxter S.p.A.) continued through a central venous catheter.

She then gradually tolerated oral feeding and Parenteral Nutrition (PN) was progressively reduced and stopped after 15 days.

Diplopia and nystagmus in the left eye occurred the day after PN discontinuation. Emergency brain magnetic resonance imaging (MRI) showed altered contrast in the medial thalamus and ruled out ischemic damage. This neuroimaging was compatible with thiamine deficiency encephalopathy.

The plasma level of thiamine, performed before the administration of the vitamin, was in the range of normal values (187 nmol/l, laboratory normal values: 66-200 nmol/l). Blood lactates were also within normal limits.

Immediately, a bolus of 300 mg of thiamine was administered Intramuscularly (IM), with slow resolution of visual disorders and complete remission 10 days after administration of thiamine.

At the time of discharge, she tolerated oral feeding and had fully recovered with normal neurological examination.

Control brain MRI, 3 months after thiamine administration, was completely normal, with disappearance of thalamus lesions.

Adult case report

A 67-year-old woman presented abdominal pain, nausea, vomiting and weight loss 1 month after ERCP complicated by acute pancreatitis. When she was admitted neurologic examination was negative and laboratory tests showed increased C-Reactive Protein (CRP) (7.1 mg/l). Due to persistent gastrointestinal tract symptoms and poor oral intake, PN was started with an all-in-one, three-compartment bag (Olimel N4E, Baxter S.p.A.) 1000 ml/day, that was suspended after 10 days on patient's request. The subsequent oral refeeding attempt failed due to persistent vomiting episodes. At the same time, she presented a confusional status and bilateral nystagmus, with no evidence of organic lesions on brain computed tomography (CT). Thiamine deficiency was then suspected and vitamin supplementation was started at the dose of 100 mg IM (Benerva 100 mg/ml, Teofarma S.r.L.) for 8 days, followed by supportive PN (Olimel N4E 1000 ml/day) with a multivitamin preparation once daily containing 3.51 mg of thiamine/dose (Cernevit, Baxter S.p.A.).

The patient's blood thiamine level evaluated 12 h after vitamin supplementation was in the range of normal values (156 nmol/L, laboratory normal values: 66-200 nmol/L). Brain MRI performed 3 days after the beginning of the confusion status showed "a subtle hyperintensity in the dorsal medial thalamus", images consistent with the clinical suspicion of thiamine-deficient encephalopathy.

Following thiamine administration, there was a rapid improvement in the neurological status, with complete resolution of nystagmus. Fifteen days after the beginning of thiamine therapy, brain MRI showed a regression of the previously described lesions, confirming the diagnosis of WE.

She had recovered completely at the time of discharge and continued thiamine supplementation at home for an additional 10 days (300 mg 1 tablet/day).

Discussion

WE is a neurological disorder caused by thiamine deficiency mainly in alcoholics, but it is reported in other clinical conditions with altered nutritional status, including pediatric patients.

The exact prevalence and incidence of WE are unknown and it was estimated that about one third of patients, both pediatric and adult, were diagnosed during post mortem examination [1,7-9]. Some WE patients may present with the classic clinical triad [8], but more often they show atypical or incomplete clinical presentations, affecting various organs and systems, including heart, gastrointestinal tract, peripheral and central nervous systems [10,5].

In particular, nonalcoholic WE commonly presents changes in mental status with no other symptoms [4] and this makes diagnosis more difficult [11].

Our reported cases did not present with the complete classical triad on admission at the Hospital.

Only diplopia and unilateral nystagmus occurred in the pediatric patient, while confusion and bilateral nystagmus occurred in the woman.

In both our cases, the diagnosis of WE was based on these clinical manifestation, history, long-standing malnutrition with low vitaminic intake, but also the good response to thiamine treatment.

As thiamine is a water-soluble vitamin it is not stored in tissues in large amounts. A healthy diet provides sufficient thiamine stores for a few weeks under normal circumstances [12]. The human body can store about 30 mg of thiamine while its daily consumption is only about 2 mg [8,9,13].

A state of thiamine depletion can develop within 18-20 days in patients receiving a strict thiamine-free diet, like a PN without vitamin supplementation [8,9,13].

Additionally, thiamine requirement is related to caloric and carbohydrate intake [13] and about 0,5 mg for every 1000 Kcal consumed are recommended [8,9]. The recommended dietary allowance (RDA) for thiamine depends also on age such as illustrated on Table 1 [9].

Table 1: Recommended daily dietary intake of thiamine at various ages.

Age	Daily dietary intake of thiamine
1-3 years	0,5 mg/day
4-8 years	0,6 mg/day
9-13	0,9 mg/day
14-18	1-1,2 mg/day
Adult	1,1 mg/day
Pregnancy	1,2 mg/day
Lactation	1,4 mg/day

Despite the availability of dietary thiamine in wealthy countries, thiamine deficiency can be caused by inadequate dietary consumption, for example in patients receiving PN without vitamin supplementation, increased loss from the body, reduced intestinal absorption, increased metabolic demands (e.g. septic shock, acute or chronic disease, such as cancer and gastrointestinal disease) [8,12,14].

Cases of PN-induced WE occurring 4-40 days after initiation of PN have been reported in the literature [9].

Both of our patients had episodes of vomiting and reduced nutritional intake in the previous weeks or months, as well as having received a support PN without vitamin supplementation in the previous 10-15 days.

Although only 17% of WE cases have the classical clinical triad [8], WE diagnosis is clinical.

Diagnosis of alcohol-related WE is based on the clinical criteria of the 2010 European Federation of Neurological Societies (EFNS) guidelines (at least 2 of: nutritional deficiency and a history of an alcohol use disorder, or any other deficiency state, oculomotor abnormalities, balance disorders and altered mental status or mild memory impairment) and it is reasonable to apply the same criteria to non-alcoholic patients [1].

Nonalcoholic patients with WE often present symptoms and radiological imaging findings different than patients with alcoholism, which further complicates the diagnosis of WE [1].

As reported in the literature, several conditions associated with thiamine deficiency, in particular malnutrition, prolonged parenteral feeding, malignancy, hyperemesis gravidarum, chronic inflammatory bowel disease, immunodeficiency, malabsorption, dialysis, hyperthyroidism, sepsis, are also due to increased metabolism request [1,15].

Post-abdominal surgery can trigger a thiamine deficiency due to malabsorption and vomiting. Prolonged intravenously (IV) infusion of solutions containing glucose/dextrose or PN without adequate vitamin intake can rapidly precipitate thiamine deficiency in these patients whose thiamine reserves are depleted [15].

Both of our patients underwent abdominal surgery, vomited in the weeks or months prior to the onset of neurological symptoms, and although supportive PN was initiated, thiamine intake was not adequate. All these factors could therefore have contributed to precipitate thiamine deficiency.

About neuroimaging studies, brain MRI has low sensitivity of only 53% but high specificity of 93% for the diagnosis of WE. The absence of MRI signal-intensity alterations, however, does not exclude the diagnosis of WE [1]. Atypical MRI features include involvement of the cerebellum, cranial nerve nuclei, red nuclei, dentate nuclei head, splenium, fornix and cerebral cortex [7,16].

Although symmetrical lesions of the medial thalamine and periventricular region of the third ventricle are the most common (80%), Zuccoli et al demonstrated that lesions may be present in other sites such as the periaqueductal area (59%), the mammillary bodies (45%), the tectal plate (36%), the nuclei of the cranial nerves (18%), the periventricular gray matter located anterior to the fourth ventricle (7%), the cerebellum (5%), the worm (4%), the dentate nuclei (1.8%) [7]. Cerebella involvement on imaging is rare but autopsy studies have demonstrated that the superior vermis is affected in one-third of patients with WE [1,16].

In accordance with scientific data, in our pediatric case, MRI shows bilateral altered contrast in the medial thalamus, and hyperintensity in the medial dorsal thalamus we found also in our adult patient. Although neuroimaging studies are not recommended as a diagnostic tool, they could be used to rule out the alternative diagnosis [8]. A CT scan is not recommended for routine diagnosis of WE [8], but in our adult patient this examination excluded organic cause.

WE cannot be diagnosed on the basis of thiamine concentration, as there is no specific critical level below which all individuals develop the condition [8]. Our patients plasma levels of thiamine were in normal range.

WE is a medical emergency and patients require immediate administration of thiamine IM or IV to prevent further progression, permanent neurological impairment or death [8]. The prognosis of WE depends on the time of initiation of thiamine supplementation [7] and empiric thiamine replacement should be initiated as soon as WE is suspected [3]. However, there are no guidelines for the management and treatment of thiamine deficiency [8,17].

Acute thiamine deficiency should be treated with high dose of thiamine. The doses used in the treatment of WE patients range from 50 mg IM for several days to 500 mg IV several times a day [18].

EFNS guidelines recommend 200 mg of thiamine diluted with 100 ml of normal saline or 5% dextrose infused IV, given over 30 min [19]. The half-life of thiamine is 96 min, and therefore the ideal regimen would involve administration of the drug twice or thrice daily [8]. In alcoholic WE, thiamine 500 mg IM three times daily for 2 to 3 days and 250 mg IV daily for the next 3 to 5 days are recommended as immediate treatment and its daily administration should be continued for several days, until all symptoms have disappeared [3,18].

Although there are no references for critically ill pediatric patients [9], 100 mg of replacement thiamine per day is routinely recommended [20]. The literature suggests that pediatric patients at risk of thiamine deficiency must be supplemented with 0.35-0.5 mg/kg/day of oral thiamine to prevent the development of encephalopathy and dosage of 50-100 mg daily or 1,8 mg thiamine per 1000 Kcal has been suggested [21]. Unfortunately, there is no agreement on the therapeutic dosage and route of administration of thiamine even in childhood and adolescence, as evidenced by literature. Table 2 shows pediatric cases over the past 10 years, specifying the dosage and route of thiamine administration (Table 2).

Table 2: Pediatric thiamine deficiency case studies, over the past 10 years, specifying the thiamine's dosage and route of administration.

Case report	Age	Thiamine treatment
Han et al 2012 [22] Darlington et al 2015 [23] Forno et al 2020 [24]	12 5 14	100 mg daily for 7 days
Cefalo MG et al, 2013 [25]	6	20 mg/kg oral daily for 75 days
Cefalo MG et al, 2013 [25]	12	500 mg IV for 5 days, 100 mg IM for next 20 days, 100 mg for 3 times a week
Benidir et al 2014 [26] Long et al 2014 [27]	8 17	100 mg IV daily
Arslan et al 2014 [28]	13	100 mg IV daily for 1 week
Gliebus G et al, 2014 [29]	1	50 mg IM daily for 14 days
Park et al 2014 [21]	13	100 mg IV daily for 5 days + 500 mg thrice daily
Simalti AK et al, 2015 [30]	0	5 mg/kg IV twice daily
Han MJ et al, 2016 [31]	2	50 mg daily for 3 weeks
Kizilocak et al 2017 [32]	13	200 mg IM thrice daily for 3 days + 100 mg IM twice daily for next 3 days + 100 mg daily for last 3 days
Lerner RK et al, 2017 [33]	4 10	10 mg IV daily
Teagarden AM. et al, 2017 [34]	0	50 mg IV daily for 2 weeks
Kamasak T et al., 2018 [35]	11	100 mg IM daily for 5 days
Elias IM et al, 2019 [36]	14	50 mg daily for 3 days
Roilides et al 2019 [12]	3	25 mg IM daily for 17 days
Forno et al 2020 [24]	7	250 mg IV daily for 3 days + 100 mg daily for next 41 days
Dilber B et al, 2020 [37]	12	200 mg twice daily for 5 days
Zhang Y et al, 2020 [38]	0	100 mg IV daily for 3 days, 20 mg oral thrice daily for next 5 days
Cornalius et al 2021 [20]	2	100 mg IV twice daily for 3 days + 100 mg IV daily for next 4 days
Derespina KR et al, 2021 [39]	1	25 mg IV daily + 10 mg IV daily for next month
Suryakanthi C et al, 2022 [40]	0	100 mg oral daily for 4 days
Suryakanthi C et al, 2022 [40]	0	100 mg IV daily for 1-3 days
Suryakanthi C et al, 2022 [40]	0	100 mg IV thrice daily for 1 day

IM: Intramuscular; IV: Intravenous

Apparently, 100 mg of replacement thiamine per day has not been documented in pediatric patients, but this mentioned adult dosage may be useful in the treatment of adolescents, particularly those approaching adult weight [5].

The use of the IM route for thiamine repletion should be limited to patients without IV access in emergency situations as only a few studies have examined the therapeutic benefits of IM administration of thiamine [9]. Francini-Pesenti et al, reported a series of WE cases treated with supplementation of thiamine IV (100 mg per day) with complete resolution of the neurological presentation within a few days when administered early [13], like in our patients. Fortunately, diplopia and nystagmus completely disappeared after a few days of thiamine administration in our pediatric patient. At the same way, neurological symptoms improved rapidly in the adult patient following thiamine administration. The rapid thiamine administration allowed a complete regression of neurological symptoms and avoided permanent damage in both our patients, as confirmed by the neurological follow-up.

Conclusion

In conclusion, WE is a treatable but under diagnosed condition with potential for complete reversal of neurological manifestations after prompt diagnosis and treatment in both adults

and the pediatric population.

Medical staff and caregivers should be aware of the importance of vitamin supplementation including thiamine, especially in patients during prolonged and inadequate enteral intake, during hydration or PN, to prevent the development of iatrogenic WE. Furthermore, it is essential to initiate thiamine supplementation in patients at risk or with suggestive symptoms, as early supplementation can allow for complete recovery. However more data is needed for specific treatment recommendation, especially in children.

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