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An Overview of Beriberi

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Highlights of the Study

- Beriberi is rare in developed countries but remains prevalent in some regions.
- Diagnosis is based on symptoms and response to thiamine due to the cost of testing.
- Food fortification, education, and early action are key to preventing Beriberi.

Keywords
Thiamine · Beriberi · Clinical features · Diagnosis · Treatment

Abstract
Beriberi is a nutritional disorder caused by thiamine deficiency. Classically, Beriberi presents in two primary clinical forms: "wet" Beriberi, which features heart and circulatory system impairment, and "dry" Beriberi, which causes polyneuropathy. Although it is an easily treatable condition, it is often misdiagnosed and can be life-threatening if not promptly recognized and managed. The diagnosis of Beriberi is performed by the signs and symptoms of the disease and can be confirmed by thiamine deficiency identification or by therapeutic testing. However, considering the costs and the limitations of the assays to evaluate thiamine deficiency, the diagnosis based on the evaluation of clinical signs and symptoms and the therapeutic test could eliminate the need for measuring serum thiamine levels. Regarding treatment, immediate thiamine administration in the presence of clinical manifestations of the disease is recommended. Overall, 100–300 mg daily doses are enough to improve symptoms. In this review, we aim to (1) provide a clinical update about how to identify and treat the Beriberi and (2) describe the historical perspective, pathophysiological mechanisms, and other relevant aspects which may have applications in clinical management of Beriberi.

Introduction

Beriberi is a nutritional disorder caused by thiamine (vitamin B1) deficiency [1]. When left untreated or if the diagnosis is delayed, it can result in cardiovascular damage and motor changes, increasing the risk of mortality [2]. Classically, Beriberi presents two major

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clinical forms: "wet" Beriberi, which features heart and circulatory system impairment, and "dry" Beriberi, which causes damage to the nervous system, leading to polyneuropathy [3]. Long-term alcohol drinking is the most common cause of Beriberi. Still, etiologic factors are also related to food insecurity, increased demand for thiamine, medication use, and impaired absorption and metabolism of this vitamin [3, 4]. Beriberi is not a worldwide disease concern, occurring predominantly in outbreaks and isolated cases among susceptible groups [4].

From a worldwide perspective, thiamine deficiency is prevalent in many communities in Southeast Asia, South Asia, and West Africa. However, the assays' limitations in measuring thiamine levels, their high costs, and the limited number of clinical studies make it difficult to determine the global prevalence of thiamine deficiency [5–8]. Considering these limitations, the presence of Beriberi's signs and symptoms could eliminate the need for thiamine measurement to confirm its diagnosis and initiate treatment. In addition, there is still controversy regarding the need and dose of thiamine supplementation to improve clinical outcomes [9–11]. Although it is an easily treatable condition, it is often misdiagnosed and can be life-threatening if not promptly recognized and managed [12, 13]. In this review, we aim to (1) provide a clinical update about how to identify and treat the Beriberi and (2) describe the historical perspective, pathophysiological mechanisms, and relevant aspects that could have applications in the clinical management of Beriberi.

Historical Perspective

The etymology of the term beriberi is unclear, but it has been proposed that it is derived from the Sinhalese word "*Bharyee*," which means "extreme weakness." The earliest description of a condition supposed to be beriberi dates to the Huangdi Neijing, an ancient Chinese medical text compiled between the 5th and 3rd centuries BC [14, 15]. Later, a disease affecting the nervous system and causing symptoms such as edema, breathlessness, and unusual gait, was reported in Japan during the 17th century, named as *Kak'ke*, meaning "disease of the legs" [14]. In the late 19th century, the cause of Beriberi was initially attributed to an unknown infectious organism [15]. This theory was supported by the fact that the incidence of Beriberi seemed to be higher in specific populations, such as prisoners and soldiers, who were living in crowded and unsanitary conditions [15–17]. However, the physician Christiaan Eijkman's pioneering experiments in the Dutch East Indies (now Indonesia) demonstrated that chickens fed with polished rice experienced leg paralysis or "polyneuritis." In contrast, chickens given brown (unpolished) rice did not suffer from this condition. Concomitantly, in the early 20th century, a national epidemic of Beriberi in Japan coincided with the introduction of mechanical polishing of rice, establishing a clear connection between the rice brand and its specific dietary component against this condition known as the "anti-beriberi factor" [18, 19].

Only in the 1920s, a substance that cured polyneuritis in pigeons was isolated and crystallized from rice polishings and named thiamine, based on its chemical structure, which includes a thiazole ring containing a sulfur atom (represented by "thia-") that is similar to the sulfur-containing amino acid cysteine (represented by "-amine") [18].

To date, reports of Beriberi are relatively rare due to the widespread availability of thiamine-enriched foods and supplements, particularly in high-risk regions. Although accurate estimates of its prevalence are unknown, thiamine deficiency remains a public health issue across South and Southeast Asia. It still happens because the population has a high consumption of polished white rice as a dietary staple, along with the frequent consumption of raw or fermented fish that contains antithiamine compounds, which inhibits thiamine bioavailability. Additionally, Asian cultural food restrictions during the perinatal period can lead to insufficient breast milk thiamine and, consequently, inadequate supply of thiamine to infantile [19, 20].

Also, many parts of Latin America and Africa still suffer from occasional outbreaks of Beriberi, related frequently to inadequate food consumption and preparation, poverty, malnutrition, and widely overlapping with other disease conditions, such as HIV/AIDS, malaria, and tuberculosis, which can increase the risk of thiamine deficiency [19, 21].

In the early 2000s, two sizeable outbreaks of Beriberi were recorded in Brazil. First, the Northeast region involved 1,207 cases and 40 deaths, potentially linked to alcohol abuse, mycotoxin contamination of rice, and extremely physically demanding [21, 22]. Soon after this, 10 cases and 3 deaths emerged within indigenous populations residing in a remote area of the Amazon Basin, a community subsisting mainly from fishing and daily consumption of *caxiri*, a local traditional alcoholic drink [21, 23]. Data obtained from the Ministry of Health through FormSUS revealed that between 2014

and 2020, 542 cases of Beriberi were identified in Brazil, resulting in 117 deaths. The majority of cases in the country are found within the indigenous community, primarily attributed to issues related to food insecurity and physically strenuous labor [24].

In the African continent, a large outbreak of Beriberi was reported in 2009 among 241 soldiers in Mogadishu, exposed to a severely restricted diet during the humanitarian crisis in Somalia. The clinical symptoms included the acute onset of peripheral edema, difficulty breathing, palpitations, and fever, evolving to heart failure and death in 4 patients [25].

Beyond social and cultural determinants, thiamine deficiency has also re-emerged in developed countries as a relevant complication following bariatric surgery, typically after procedures such as Roux-en-Y gastric bypass and sleeve gastrectomy. This procedure-related deficiency often arises within weeks due to limited body thiamine stores and reduced absorption, frequently manifesting as Wernicke's encephalopathy or wet beriberi if not promptly treated [26, 27]. In this sense, Beriberi is still an emerging disease today, especially in developing countries. Ongoing vigilance and intervention through public policies are necessary to address thiamine deficiency, particularly in vulnerable populations, and provide clinical updates on identifying and treating this disease.

Functions of Thiamine and Pathophysiology of Beriberi

Thiamine is naturally present in various foods, including most animal sources, yeast, wheat germ, and unrefined cereal grains [28]. The recommended daily intake of thiamine for children depends on age, ranging from 0.5 mg/day for 1- to 3-year-olds to 0.9 mg/day for 9- to 13-year-olds. Typically, thiamine requirements are around 1.1–1.2 mg/day for adults and older people. During pregnancy and lactation, the requirement increases to 1.4 and 1.5 mg/day, respectively. Nevertheless, the intake of this nutrient should be constant, as body reserves are relatively small and can only last about 18 days. Also, the use of medications, especially diuretics, is linked to increased excretion [3, 28, 29]. Thiamine is absorbed in the upper part of the small intestine through a saturable process mediated by two transporters: THTR2 on the luminal surface and THTR1 on the basal surface, releasing thiamine in its free form into the circulation. The biologically active form, thiamine pyrophosphate (TPP), also known as thiamine diphosphate, is generated in cells from free thiamine by the enzyme thiamine pyrophosphokinase [30]. TPP then acts as a cofactor for enzyme activity in several metabolic pathways, including glucose metabolism, energy production, and nucleic acid synthesis. Enzymes such as transketolase, α-ketoglutarate dehydrogenase, pyruvate dehydrogenase complex, and 2-hydroxyacyl-CoA lyase depend on TPP for proper functioning [28, 30]. In this sense, nutritional deficiency by thiamine can lead to the impairment of these enzymes, resulting in metabolic alterations that compromise adenosine triphosphate synthesis, which depends on TPP, as well as predisposing to metabolic acidosis, due to the high concentration of lactate [31].

Thiamine deficiency is still often an under-recognized condition; however, the presence of signs and symptoms of Beriberi indicates thiamine deficiency without the need for laboratory confirmation [32]. Beriberi is categorized as either dry or wet [2].

Dry Beriberi is distinguished by psychomotor alterations stemming from disruptions in the neurotransmission pathways of the glutamatergic and GABAergic systems, leading to neurotoxicity. Additionally, a reduction in myelin sheath formation occurs due to pentose phosphate pathway disturbances [2]. The other clinical form is wet Beriberi, which includes cardiovascular alterations through the stimulation of peripheral vasodilation [33, 34]. This vasodilation triggers compensatory mechanisms, including activating the sympathetic and renin-angiotensin systems. Consequently, this leads to increased renal sodium and water absorption. The reduction in afterload, in conjunction with hypervolemia, culminates in a clinical presentation characteristic of heart failure, characterized by elevated cardiac output aimed at sustaining blood pressure. However, in the most severe cases, where acute and often fatal manifestations occur, it is referred to as "Shoshin beriberi" [35, 36]. In the context of "Shoshin Beriberi," individuals commonly present with cardiogenic shock [35]. Thiamine deficiency also contributes to increased lactate production and subsequent metabolic acidosis. In turn, the subsequent accumulation of hydrogen ions results in depression of myocardial contractility and additional vasodilation, exacerbating systemic hypotension [10, 35, 37]. However, the pathophysiological manifestation of Beriberi is not completely understood, as it is still unclear whether genetic and epigenetic factors influence its development. Furthermore, even following the confirmation of thiamine deficiency, some individuals

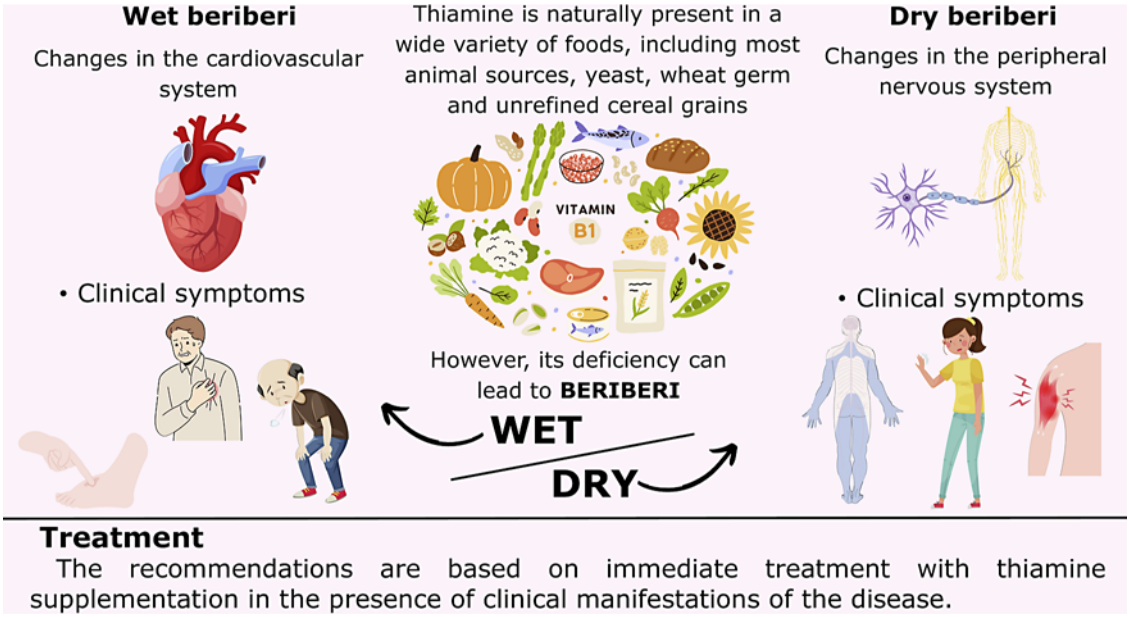

Fig. 1. Clinical features of Beriberi.

Table 1. Signs and symptoms of Beriberi

Wet Beriberi
High-output heart failure
Tachycardia
Pulmonary hypertension
Predominantly right-sided heart failure
Lower limb edema
Dyspnea
Liver and spleen congestion
"Shoshin beriberi"
Cardiogenic shock
Increased serum lactate
Metabolic acidosis
Generalized fatigue
Loss of appetite
Abdominal pain
Heart failure
Reduced cardiac output
Dry Beriberi
Peripheral polyneuropathy
Muscle weakness

do not progress to the clinical presentation of the disease. In addition, no conclusive evidence is available to discern which individuals are more predisposed to cardiac as opposed to psychomotor complications. Additional studies are imperative to elucidate and address these knowledge gaps.

Clinical Features

Diagnosis

The diagnosis of Beriberi is performed by the signs and symptoms of the disease and can be confirmed by identification of thiamine deficiency or by therapeutic testing. The assessment of serum thiamine level is commonly used for this purpose.

However, it only provides an inadequate evaluation of the body's thiamine status due to the short-term thiamine intake representation [38]. Alternative methods used for thiamine measurement include urine thiamine levels, which is not considered a reliable indicator of overall body thiamine store, and the thiamine pyrophosphate (TPP) analysis, which is characterized considered highly sensitivity and specific [39]. However, small studies suggest that TPP levels alone may not effectively distinguish clinical beriberi from subclinical deficiency. In populations with chronically low thiamine intake, both symptomatic and asymptomatic individuals often present relatively low TPP concentrations [40–42].

The gold standard method is the test for activating erythrocyte transketolase by TPP since it can indirectly measure the biologically active form of thiamine. Nevertheless, this method also remains less accessible in clinical practice due to its high cost [43]. Considering the costs and limitations of the assays to evaluate thiamine deficiency, a diagnosis based on the evaluation of clinical signs and

Table 2. Recommendations for thiamine administration for Beriberi treatment

Condition	Recommendation
Wet Beriberi	Orally, 100–300 mg daily for at least 6 weeks, accompanied by hemodynamic support [26]
"Shoshin beriberi"	100–300 mg per day, parenterally, for at least 6 weeks, accompanied by hemodynamic support [26]
Dry Beriberi	25–100 mg per day, orally, for at least 2 weeks, accompanied by psychomotor therapy [19]

symptoms and the therapeutic test could eliminate the need for measuring serum thiamine levels [28].

The main clinical manifestations of wet Beriberi are linked to changes in the cardiovascular system, mainly high-output heart failure. The impairment of the vascular endothelium in response to reduced nitric oxide synthesis that is thiamine-dependent and could also lead to pulmonary hypertension and right-sided heart failure [33, 34]. These alterations are detected by symptoms such as lower limb edema, dyspnea, tachycardia, and hepatic and splenic congestion (Figure 1) [13, 34]. However, in more severe cases of wet Beriberi, the clinical manifestations may resemble cardiogenic shock, i.e., ventricular failure, metabolic acidosis, changes in cardiac output, and vascular changes. As previously mentioned, this particular condition is referred to as "Shoshin beriberi," which frequently presents with indications of generalized fatigue, loss of appetite, and abdominal pain. However, when left undiagnosed in its early stages, it can lead to a fatal outcome [36, 44]. On the other hand, dry Beriberi is based on signs and symptoms related to peripheral polyneuropathy and the presence of muscle weakness [2].

Diagnosing Beriberi can be difficult due to diverse clinical manifestations and similar symptomatology with other illnesses, which may mask its diagnosis. Vulnerable populations such as malnourished individuals, alcoholics, infants, the indigenous community, and individuals with malabsorptive syndromes, are at a higher risk of thiamine deficiency and, subsequently, beriberi development. Signs and symptoms of Beriberi were also observed in individuals following bariatric and gastrointestinal surgery, as well as in patients with cancer and pancreatitis [14, 45]. Table 1 shows the signs and symptoms of Beriberi.

Treatment

Early diagnosis of Beriberi is essential for therapeutic success. Thus, the recommendations are based on immediate treatment with thiamine administration in the presence of clinical manifestations of the disease [29]. The thiamine is usually administered for a minimum period of 6 weeks and may be provided orally. Overall, 100–300 mg daily doses are enough to improve the symptoms [3, 29].

After diagnosis, the thiamine administration regimen is based on the type of Beriberi. In cases where the manifestations align with wet Beriberi, the recommendations suggest thiamine supplementation orally, along with hemodynamic support. In more severe cases, where the clinical presentation indicates "Shoshin beriberi," some authors recommend parenteral administration of higher thiamine doses until the signs and symptoms subside. The most commonly employed dose ranges between 100 and 300 mg per day [28, 29]. For dry Beriberi, thiamine recommendations are generally lower, with variations in administration time, depending on psychomotor impairment. Also, in cases of moderate to severe neuropathy, physical rehabilitation is recommended [39, 46].

The recommendations for thiamine administration (Table 2) reinforce that when the intravenous method is chosen; the dilution should be performed using a saline solution without adding glucose. This approach is recommended as carbohydrate metabolism can elevate the thiamine requirement, potentially exacerbating the clinical condition and increasing mortality risk [2]. To summarize, the recommendations are based on thiamine administration for an extended period until the risk factor for thiamine deficiency is removed. In addition, it is important to assess and replace other micronutrients besides thiamine that may be deficient [47].

Health Education Activities to Prevent Beriberi

Beriberi is considered a prevalent disease in developing countries [21]. Thus, global cooperation strategies and knowledge translation are essential to implementing scientific evidence [48]. In Brazil, where the disease persists, particularly within indigenous lands, the Ministry of Health, in collaboration with the Pan American Health Organization (PAHO) and the World Health

Organization (WHO), has established a partnership under the "Country Cooperation Strategy 2022–2027" program. This collaboration aims to strengthen the commitment to addressing both communicable and non-communicable diseases that continue to affect Brazil and to guarantee control and eradication within the realm of public health. Beriberi stands as one of the priority diseases to be targeted in this endeavor [49]. On a worldwide scale, since 1999, the WHO has put forth global strategies regarding thiamine deficiency that can be implemented, such as the development of food products fortified with thiamine, food and nutrition education for individuals and communities, continuing education for health professionals, and easier methods of diagnosing nutritional deficiencies [50]. This body of information highlights the need for short-term, medium-term, and long-term solutions to prevent thiamine deficiency and address potential repercussions stemming from thiamine deficiency and Beriberi.

Conclusion

Beriberi is a clinical condition that still exists. Although it is a readily treatable disease, the challenges associated with diagnosis make it potentially fatal, particularly within vulnerable populations. The certainty of beriberi symptoms renders measurement of serum thiamine levels unnecessary, considering the limitations and costs associated with these methods. Refined epidemiological screening can contribute to the timely identification of at-risk populations, fostering proactive intervention strategies, and improving patient outcomes and effective public health management.

Statement of Ethics

This article is a narrative review and did not involve any studies with human or animal subjects performed by any of the authors. Therefore, ethical approval was not required.

Conflict of Interest Statement

None of the authors have any conflict of interest to declare.

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Author Contributions

Marcos Ferreira Minicucci and Leonardo Antônio Mamede Zornoff: study conception and critical review for intellectual improvement. Amanda Gomes Pereira, Nara Aline Costa, and Letycia Netto de Paula Cunha: study design; acquisition, analysis, interpretation of data for the work, and drafting. Paula Schmidt Azevedo, Bertha Furlan Polegato, and Sergio Alberto Rupp Paiva: critical review for intellectual improvement. All authors critically revised the article for important intellectual content and approved the final version of the manuscript.

References

1 Lonsdale D. Thiamin. Adv Food Nutr Res. 2018;83:1–56. https://doi.org/10.1016/bs.afnr.2017.11.001
2 Smith TJ, Johnson CR, Koshy R, Hess SY, Qureshi UA, Mynak ML, et al. Thiamine deficiency disorders: a clinical perspective. Ann NY Acad Sci. 2021;1498(1):9–28. https://doi.org/10.1111/nyas.14536
3 Frank LL. Thiamin in clinical practice. JPEN J Parenter Enteral Nutr. 2015;39(5):503–20. https://doi.org/10.1177/0148607114565245
4 Sycks K, Simerlink S, McKnight L, Trovato V. A case of dry beriberi from alcohol use disorder and disordered eating. Subst Abus. 2022;43(1):204–5. https://doi.org/10.1080/08897077.2021.1915917
5 Barennes H, Sengkhamyong K, René JP, Phimmasane M. Beriberi (thiamine deficiency) and high infant mortality in Northern Laos. PLoS Negl Trop Dis. 2015;9(3):e0003581. https://doi.org/10.1371/journal.pntd.0003581
6 Porter SG, Coats D, Fischer PR, Ou K, Frank EL, Sreang P, et al. Thiamine deficiency and cardiac dysfunction in Cambodian infants. J Pediatr. 2014;164(6):1456–61. https://doi.org/10.1016/j.jpeds.2014.01.049
7 Qureshi UA, Sami A, Altaf U, Ahmad K, Iqbal J, Wani NA, et al. Thiamine responsive acute life threatening metabolic acidosis in exclusively breast-fed infants. Nutrition. 2016;32(2):213–6. https://doi.org/10.1016/j.nut.2015.08.007
8 Nosten F. Beriberi in Cambodia. Paediatr Int Child Health. 2015;35(4):283–4. https://doi.org/10.1080/20469047.2015.1109246
9 Costa NA, Gut AL, de Souza Dorna M, Pimentel JAC, Cozzolino SMF, Azevedo PS, et al. Serum thiamine concentration and oxidative stress as predictors of mortality in patients with septic shock. J Crit Care. 2014;29(2):249–52. https://doi.org/10.1016/j.jcrc.2013.12.004
10 Donnino MW, Andersen LW, Chase M, Berg KM, Tidswell M, Giberson T, et al. Randomized, double-blind, placebo-controlled trial of thiamine as a metabolic resuscitator in septic shock: a pilot study. Crit Care Med. 2016;44(2):360–7. https://doi.org/10.1097/CCM.0000000000001572
11 Pereira AG, Costa NA, Amancio SCP, Okoshi MP, Zornoff LAM, Azevedo PS, et al. Effect of thiamine on clinical outcomes in septic shock patients: a randomized, double-blinded pilot study. Am J Respir Crit Care Med. 2023;208(5):616–8. https://doi.org/10.1164/rccm.202208-1583LE
12 Wooley JA. Characteristics of thiamin and its relevance to the management of heart failure. Nutr Clin Pract. 2008;23(5):487–93. https://doi.org/10.1177/0884533608323430

13 Lei Y, Zheng MH, Huang W, Zhang J, Lu Y. Wet beriberi with multiple organ failure remarkably reversed by thiamine administration: a case report and literature review. Medicine. 2018;97(9):e0010. https://doi.org/10.1097/MD.0000000000010010
14 Wilson RB. Pathophysiology, prevention, and treatment of beriberi after gastric surgery. Nutr Rev. 2020;78(12):1015–29. https://doi.org/10.1093/nutrit/nuaa004
15 Hawk A. The great disease enemy, Kak'ke (beriberi) and the imperial Japanese army. Mil Med. 2006;171(4):333–9. https://doi.org/10.7205/milmed.171.4.333
16 Ijaz S, Thorley H, Porter K, Fleming C, Jones T, Kesten J, et al. Interventions for preventing or treating malnutrition in homeless problem-drinkers: a systematic review. Int J Equity Health. 2018;17(1):8. https://doi.org/10.1186/s12939-018-0722-3
17 Banna J, Bersamin A. Community involvement in design, implementation and evaluation of nutrition interventions to reduce chronic diseases in indigenous populations in the U.S.: a systematic review. Int J Equity Health. 2018;17(1):116. https://doi.org/10.1186/s12939-018-0829-6
18 Carpenter KJ. The discovery of thiamin. Ann Nutr Metab. 2012;61(3):219–23. https://doi.org/10.1159/000343109
19 Johnson CR, Fischer PR, Thacher TD, Topazian MD, Bourassa MW, Combs GF. Thiamin deficiency in low- and middle-income countries: disorders, prevalences, previous interventions and current recommendations. Nutr Health. 2019;25(2):127–51. https://doi.org/10.1177/0260106019830847
20 Smith TJ, Hess SY. Infantile thiamine deficiency in South and Southeast Asia: an age-old problem needing new solutions. Nutr Bull. 2021;46(1):12–25. https://doi.org/10.1111/nbu.12481
21 Assunção AKM, Branco MRFC, Santos TS, Costa SSB, Júnior JJD, Soeiro VMS, et al. Beriberi in Brazil: a disease that affects Indigenous people. Food Nutr Bull. 2021;42(3):427–36. https://doi.org/10.1177/03795721211002057
22 Padilha EM, Fujimori E, Borges ALV, Sato APS, Gomes MN, Branco MRFC, et al. Perfil epidemiológico do beribéri notificado de 2006 a 2008 no Estado do Maranhão, Brasil. Cad Saude Publica. 2011;27(3):449–59. https://doi.org/10.1590/s0102-311x2011000300006
23 Cerroni MP, Barrado JCS, Nobrega AA, Lins ABM, da Silva IP, Mangueira RR, et al. Outbreak of beriberi in an Indian Population of the upper Amazon region, Roraima State, Brazil, 2008. Am J Trop Med Hyg. 2010;83(5):1093–7. https://doi.org/10.4269/ajtmh.2010.10-0345
24 Maximino FDS, Branco MRFC. Análise espacial de casos e óbitos por beribéri e distribuição de tiamina, Brasil, 2014–2020. REAS. 2023;23(5):e12032. https://doi.org/10.25248/reas.e12032.2023
25 Watson JT, El Bushra H, Lebo EJ, Bwire G, Kiyengo J, Emukule G, et al. Outbreak of beriberi among African Union troops in Mogadishu, Somalia. PLoS One. 2011;6(12):e28345. https://doi.org/10.1371/journal.pone.0028345
26 Oudman E, Wijnia JW, van Dam M, Biter LU, Postma A. Wernicke's encephalopathy after bariatric surgery: a systematic review. Obes Surg. 2018;28(7):2060–8. https://doi.org/10.1007/s11695-018-3262-4
27 Oudman E, Wijnia JW, van Dam M, Biter LU, Postma A. Wernicke's encephalopathy in bariatric surgery patients due to thiamine deficiency: a systematic review. Clin Nutr. 2018;37(3):744–53.
28 Polegato BF, Pereira AG, Azevedo PS, Costa NA, Zornoff LAM, Paiva SAR, et al. Role of thiamin in health and disease. Nutr Clin Pract. 2019;34(4):558–64. https://doi.org/10.1002/ncp.10234
29 Berger MM, Shenkin A, Schweinlin A, Amrein K, Augsburger M, Biesalski HK, et al. ESPEN micronutrient guideline. Clin Nutr. 2022;41(6):1357–424. https://doi.org/10.1016/j.clnu.2022.02.015
30 Brown G. Defects of thiamine transport and metabolism. J Inherit Metab Dis. 2014;37(4):577–85. https://doi.org/10.1007/s10545-014-9712-9
31 Fattal-Valevski A. Thiamine (vitamin B1). J Evid-Based Complement Altern Med. 2011;16(1):12–20. https://doi.org/10.1177/1533210110392941
32 Whitfield KC, Bourassa MW, Adamolekun B, Bergeron G, Bettendorff L, Brown KH, et al. Thiamine deficiency disorders: diagnosis, prevalence, and a roadmap for global control programs. Ann NY Acad Sci. 2018;1430(1):3–43. https://doi.org/10.1111/nyas.13919
33 Gioda CR, Capettini LSA, Cruz JS, Lemos VS. Thiamine deficiency leads to reduced nitric oxide production and vascular dysfunction in rats. Nutr Metab Cardiovasc Dis. 2014;24(2):183–8. https://doi.org/10.1016/j.numecd.2013.06.010
34 Jain A, Mehta R, Al-Ani M, Hill JA, Winchester DE. Determining the role of thiamine deficiency in systolic heart failure: a meta-analysis and systematic review. J Card Fail. 2015;21(12):1000–7. https://doi.org/10.1016/j.cardfail.2015.10.005
35 Cottini M, Ranucci M, Facciolo C, Satriano A, Kandill H, Bettini F, et al. An unusual case of cardiogenic shock in which thiamine administration led to reversal of lactic acidosis and heart function recovery: Shoshin beriberi in an adolescent. Int J Cardiol. 2016;222:401–3. https://doi.org/10.1016/j.ijcard.2016.07.248
36 Tanné C, Nguyen J, Blondé R. Shoshin beriberi and thiamine-responsive right heart failure: a case report in Mayotte recognition and management of infant Shoshin beriberi. Arch Pediatr. 2022;29(8):624–5. https://doi.org/10.1016/j.arcped.2022.08.017
37 Costa NA, Azevedo PS, Polegato BF, Zornoff LAM, Paiva SAR, Minicucci MF. Thiamine as a metabolic resuscitator in septic shock: one size does not fit all. J Thorac Dis. 2016;8(6):E471–2. https://doi.org/10.21037/jtd.2016.04.29
38 Marrs C, Lonsdale D. Hiding in plain sight: modern thiamine deficiency. Cells. 2021;10(10):2595. https://doi.org/10.3390/cells10102595
39 DiNicolantonio JJ, Liu J, O'Keefe JH. Thiamine and cardiovascular disease: a literature review. Prog Cardiovasc Dis. 2018;61(1):27–32. https://doi.org/10.1016/j.pcad.2018.01.009
40 Whitfield KC, Karakochuk CD, Liu Y, McCann A, Talukder A, deSouza S, et al. Poor thiamine and riboflavin status is common among lactating women in Prey Veng, Cambodia. J Nutr. 2018;148(5):868–76.
41 Khounnorath S, Chamberlain K, Taylor AM, Soukkhaseum V, Mayxay M, Lee SJ, et al. Clinically unapparent thiamine deficiency in Laos: a pilot study of the usefulness of erythrocyte transketolase activity. Am J Clin Nutr. 2011;94(4):789–95.
42 Coats D, Shelton-Dodge K, Ou K, Khun V, Seab S, Sok K, et al. Thiamine deficiency in Cambodian infants with and without beriberi. J Pediatr. 2012;161(5):843–7. https://doi.org/10.1016/j.jpeds.2012.05.006
43 Leite HP, de Lima LFP, Taddei JAAC, Paes ÂT. Effect of blood thiamine concentrations on mortality: influence of nutritional status. Nutrition. 2018;48:105–10. https://doi.org/10.1016/j.nut.2017.11.020
44 Dabar G, Harmouche C, Habr B, Riachi M, Jaber B. Shoshin Beriberi in Critically-Ill patients: case series. Nutr J. 2015;14:51. https://doi.org/10.1186/s12937-015-0039-7
45 Huertas-González N, Hernando-Requejo V, Luciano-García Z, Cervera-Rodilla JL. Wernicke's encephalopathy, Wet Beriberi, and polyneuropathy in a patient with folate and thiamine deficiency related to Gastric Phytobezoar. Case Rep Neurol Med. 2015;2015:624807.
46 Shible AA, Ramadurai D, Gergen D, Reynolds PM. Dry beriberi due to thiamine deficiency associated with peripheral neuropathy and Wernicke's encephalopathy mimicking Guillain-Barré syndrome: a case report and review of the literature. Am J Case Rep. 2019;20:330–4. https://doi.org/10.12659/AJCR.914051
47 Costa NA, Pereira AG, Sugizaki CSA, Vieira NM, Garcia LR, de Paiva SAR, et al. Insights into thiamine supplementation in patients with septic shock. Front Med. 2021;8:805199. https://doi.org/10.3389/fmed.2021.805199
48 Straus SE, Tetroe J, Graham I. Defining knowledge translation. CMAJ Can Med Assoc J. 2009;181(3–4):165–8. https://doi.org/10.1503/cmaj.081229
49 Pan American Health Organization. Risk country cooperation strategy 2022–2027 Brazil. Revised version. Brasilia: PAHO; 2023.
50 World Health Organization. Thiamine deficiency and its prevention and control in major emergencies. Geneva: World Health Organization; 1999.