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Cardiovascular consequences of thiamine deficiency in the elderly: the literature review

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Summary

Vitamin B1 as an enzymatic cofactor plays a key role in many cellular processes during carbohydrate metabolism and energy production. The depletion of thiamine level, called beriberi syndrome, affects organs with the highest metabolic activity and has two primary forms: the dry one, affecting the neuromuscular system, and the wet one, affecting the cardiovascular system. In this review, we briefly summarize the consequences of thiamine deficiency in the cardiovascular system from a clinical perspective. Possible causes are highlighted. We discuss the prevalence, symptoms, and potential diagnostic and therapeutic strategies in cardiovascular beriberi disease, with a special insight into the geriatric population. *Geriatria 2022;16:189-194. doi: 10.53139/G.20221629*

Keywords: thiamine, beriberi, elderly

Introduction

Vitamin B1, also known as thiamine or aneurine, plays a key role in carbohydrate catabolism and pentose sugar biosynthesis [1]. Therefore, its importance is predominantly noticed in organs with the highest metabolic activity, including those in the cardiovascular, nervous and musculoskeletal systems [1]. A deficiency in vitamin B1 level is known as beriberi syndrome. The disease has two primary forms: the dry one, affecting the neuromuscular system, and the wet one, affecting the cardiovascular system. Rarely, a fulminant version, called Shoshin syndrome, may be seen [1]. However, these two forms often co-exist and heart failure (HF) along with peripheral neuropathy is observed.

The prevalence of thiamine deficiency in the geriatric population seems high, even though frequently remains underdiagnosed. In a systematic review of habitual dietary intakes of older adults (≥ 65 years) including 37 studies, subclinical deficiencies of this nutrient have been reported. The percentage of the population at risk for inadequate intake of vitamin B1 from food alone was greater than 30% [2]. In an observational pilot study of 75 elderly patients (aged ≥ 60 years) admitted to the emergency department in Utrecht, The Netherlands, 14% of them were thiamine-deficient [3]. In a cross-sectional study evaluating 309 elderly patients (aged ≥ 65 years) admitted to one rural hospital in the United States of America (USA), 28.5% had thiamine deficiency [4].

The current paper aims to briefly summarize the cardiovascular outcomes of thiamine deficiency, notably in older adults. We systematically searched the literature to provide the most recent information on the pathophysiology, prevalence, symptoms, and potential diagnostic and therapeutic strategies in thiamine deficiency disorders, with a special insight into the geriatric population.

Methods

We systematically searched PubMed for the literature, clinical trials, and databases from 1982 to November 2022. The keywords were ”thiamine deficiency”, ”wet beriberi”, ”cardiovascular beriberi”, or ”shoshin syndrome”, combined with ”elderly”, ”older adults” or „geriatric population”. Mostly case reports and case series were found. The final list of references was chosen subjectively for its applicability to the theme.

Physiology of Vitamin B1

Thiamine, in its activated form, thiamine pyrophosphate (TPP), acts as an enzymatic cofactor for several reactions during carbohydrate catabolism and pentose sugars biosynthesis. The most important aneurine-dependent biocatalysts are pyruvate dehydrogenase (PDH) and 2-oxoglutarate dehydrogenase (OGDH), which enable pyruvate, a product of glycolysis, to enter the citric acid cycle, and, therewith, promote aerobic metabolism and adenosine triphosphate (ATP) produc-

tion [1]. The reactions mentioned above are known as the Krebs cycle - the central metabolic pathway, which provides the cells with their energetic requirements. In the absence of thiamine, pyruvate cannot enter the Krebs cycle, and, consequently, is converted into lactate. A less efficient anaerobic respiration results in decreased ATP production, and, not meeting the organs' energetic requirements, leads to a decrease in their function. [1]

Like other animals, the human organism is not able to synthesize thiamine. The whole referenced daily intake needs to be supplemented by diet. The average requirement in healthy adults is 1.2 mg/day for men and 1.1 mg/day for women [1]. This, in its entirety, needs to be consumed regularly in order to meet the ongoing needs of the body, since thiamine has a short half-life time and only a small quantity (30mg) may be stored in the tissues [1]. The most common nutritional sources are whole grains, bread, and pulses, while fruits and vegetables are less useful. Moreover, several food products contain so-called anti-thiamine factors (e.g., thiaminases and thiamine antagonists) that inactivate or block the absorption of thiamine [1]. When heat-stable thiamine antagonists, including methylxanthines such as caffeine, theophylline, and theobromine (found in plant-origin products, i.e. blueberries, betel nuts, coffee, and tea) and phenolic compounds (especially with ortho-dihydroxy groups, e.g., tannins, caffeic acid, and chlorogenic acid) interact with aneurine, the non-absorbable thiamine disulfide is produced. On the other hand, heat-labile thiaminases, enzymes found in fish and shellfish, by cleaving the thiamine molecule, inactivate it [1]. Therefore, human dietary habits are of high significance, and, if unbalanced, can lead to thiamine deficiency and its consequences.

Cardiovascular (wet) beriberi

As cardiomyocytes are characterized by high metabolic activity and strictly depend on aerobic respiration, with thiamine depletion, its energetic requirements are not met and therefore loss of cells' function is observed, resulting in HF [1]. The symptoms usually include those of high-output HF with cardiomegaly, peripheral edema, elevated venous pressure, due to capillary leak, and low systemic vascular resistance caused by peripheral vasodilatation [1,5]. However, low-output HF may be also observed [5]. Additionally, unspecific changes in chest x-ray, electro-, and echocardiography can be noted [5]. However, frequently both versions of beriberi disease (wet and dry) overlap, and additional peripheral neuropathy or pellagra may appear [5]. The causes are numerous and include, i.e, inadequate dietary intake, malabsorption, increased loss, or increased demand [1]. Table I summarizes the most frequent pathogenic factors of vitamin B1 deficiency reported in the literature.

Table I. Most common causes of beriberi syndrome in the elderly reported in the literature.

Inadequeate dietary intake [23-25]: – diet high in carbohydrates (polished rice/ processed grains) – total parenteral nutrition – chronic alcoholism
Malabsorbtion due to [17,18 29-33]: – gastrectomy – bariatric surgery, including vertical sleeve gastrectomy – celiac disease
Increased loss [9-16, 34]: – diuretics use – renal replacement therapy
Increased demand [20, 22, 24-28, 35]: – high carbohydrates intake – sepsis – neoplasms

The issue of thiamine depletion is of high significance. With the aging of society, vitamin B1 deficiency in the elderly will become crucial, as the prevalence is high, especially among the ones with critical conditions such as neoplasms. In such patients, the symptoms, are usually unambiguous and may be overlooked due to various comorbidities, such as anemia, diabetes mellitus, or hypertension. Also, dementia or depression, frequently observed in the elderly, lead to changed perception and possible misdiagnosis of the condition. Thus, the diagnosis of beriberi requires a high index of suspicion, so clinicians should always keep it in mind and suspect thiamine deficiency in elderly patients with unexplained heart failure. Collecting detailed medical history seems crucial, as laboratory blood tests lack specificity and are not used routinely [5-7]. Detailed diagnostic methods are discussed below, in an appropriate chapter.

Thiamine, heart failure and the use of diuretics

Heart failure (HF) is an important global problem affecting more than 64.3 million individuals worldwide

with the prevalence steadily increasing and being the highest among the elderly [8]. Patients with HF struggle with numerous consequences, and thiamine deficiency is not uncommon among them [5]. Risk factors for developing aneurine depletion in HF include, i.e., malnutrition, advanced age, the severity of HF, and the use of diuretics [5]. These medications, with furosemide being the most frequently prescribed agent, are commonly used to reduce dyspnea and peripheral edema. However, despite the symptom alleviation, such therapy may lead to electrolyte and micro-element depletion [9-15]. Here, thiamine deficiency is believed to be caused predominantly due to increased urine volume and urinary flow rate [12]. Some evidence points out that furosemide may additionally directly inhibit thiamine uptake at the cellular level [12].

Many studies have looked at the effect of diuretics on the development of thiamine deficiency in patients with heart failure. However, the association between thiamine deficiency and diuretic use remains somewhat controversial. In a prospective analysis of thiamine status in 33 patients with congestive HF treated with furosemide, severe deficiency was found in 98% of the individuals receiving at least 80 mg/day of furosemide and in 57% of individuals taking 40 mg of furosemide daily. [9] In another study, between 118 patients admitted to the geriatric ward, the prevalence of thiamine deficiency was estimated at 40%. Among them, heart failure was commonly reported and furosemide was more frequently taken by thiamine-deficient patients [10] Suter et al. reported an association between diuretic use and thiamin deficiency in 149 patients hospitalized for HF [11]. In an uncontrolled intervention study conducted on healthy volunteers, furosemide treatment caused an increased urinary loss of thiamine in a dose-dependent manner [12]. However, in a randomized trial studying thiamine status in 35 elderly hospitalized patients with HF, including the effect of supplementation, the authors failed to find any difference in the prevalence of thiamine deficiency in patients receiving furosemide compared to patients not receiving it [13]. Hanninen et al. also failed to find a significant relationship between the use of furosemide and the development of thiamine deficiency in a large cross-sectional study involving 100 hospitalized patients with HF [14]. Despite the variability in thiamine status in patients with HF, it is still worthwhile to consider monitoring thiamine status in patients on chronic diuretic therapy and supplement as needed.

Moreover, Limited reports suggest that in some cases thiamine supplementation can improve cardiac function by increasing left ventricular ejection fraction, improving biomarker outcomes, and relieving HF symptoms, i.e. disappearance of the loud third heart sound, improvement of exercise tolerance, or peripheral edema withdrawal [5, 10, 16]. Additionally, it leads to a decrease in HF indicators (N-terminal pro-B-type natriuretic peptide) [16]. However, to date, routine thiamine administration is not recommended. More large-scale randomized controlled trials are required in the future for further determination of the thiamine replacement therapy effects in patients with HF. Clinicians should be aware that diuretics may cause thiamine deficiency and a potential worsening of preexistent HF. Thiamine therapy can be beneficial in such cases, particularly in elderly patients.

Beriberi as a reversible cause of pulmonary hypertension

Numerous systemic diseases can manifest as acute pulmonary hypertension (PH) and thiamine deficiency is reported to be one of its reversible causes [17,18]. Cardiac beriberi, which is characterized as high-output HF with systemic vascular dilatation, sometimes presents as PH and right-sided HF. Even though the majority of the reports indicate its higher prevalence in younger patients, PH may also occur in the elderly [17,18]. The symptoms include progressive dyspnoea and generalized edema, sometimes leading to right--sided HF. However, urgent thiamine therapy usually results in a quick and favorable clinical response with symptom alleviation, LVEF improvement, and mean pulmonary systolic pressure normalization [17,18]. Thus, it should be always kept in physicians' minds, as the condition, if left untreated, may be fatal.

Shoshin beriberi

Shoshin syndrome is a rare, life-threatening version of beriberi disease, characterized by hemodynamic collapse with severe cardiac dysfunction and lactic acidosis [19-21]. In developed countries nowadays, aside from patients with excessive alcohol intake, the condition is considered rare. However, there have been numerous cases described, notably in patients in intensive care units, from which the majority are older adults [20]. Available data indicate that thiamine deficiency is associated with an almost 50% increase

in mortality [22]. The potential risk factors include malnutrition and total parenteral nutrition [20]. Due to low awareness of this condition among physicians, unrevealing symptoms involving multiple systems, and lack of specific, widely obtainable laboratory assays, the fulminant beriberi disease may be easily misdiagnosed. However, despite being rare, Shoshin syndrome should always be included in the differential diagnosis of critically-ill patients, notably presenting with unexplained heart failure, polyneuropathy, refractory cardio-circulatory collapse, or inexplicable lactic acidosis. Under-recognition of this disease may entail serious consequences since fatal cases are known [21]. With prompt diagnosis and urgent thiamine administration patients quickly regain cardiac function, restore hemodynamic and metabolic parameters and improve neurological symptoms [19,20]. Sometimes mechanical circulatory support is required, but optimal medical management with thiamine replacement therapy results in improved long-term outcomes [19,20].

Wet alcoholic beriberi

Excessive alcohol intake is a common cause of thiamine deficiency due to reduced intestinal absorption. Moreover, the patients usually follow an unbalanced, high-level-carbohydrate diet, which leads to a further decrease in vitamin B1 levels. Even though most frequently the patients are middle-aged men, presenting with dry beriberi disease, with Wernicki-Korsakoff syndrome being its most severe form, wet beriberi in elderly alcoholics is also seen [23,24]. The condition most commonly compromises vasodilatation and, consequently, high-output heart failure (HF) with fluid retention [23]. However, left-sided HF with a decrease in LVEF or biventricular failure may be observed [24,25]. The symptoms are not specific and may include nonspecific ST-T segment changes in ECG, pulmonary congestion, pleural effusion, or elevation in the cardiac troponin or liver enzyme levels [24,25]. Thus, clinicians should be cautious, as even though rarely, wet beriberi in the geriatric population may be seen and, if misdiagnosed, may imply severe consequences.

Thiamine deficiency and sepsis

The overall prevalence of sepsis is increasing, which creates a significant burden. More than 60% of the cases are diagnosed in patients aged ≥ 65 years and the mortality rate of patients with sepsis-induced acute kidney injury (AKI) is high, particularly in the elderly [26,27]. The pathogenesis is complex, however, one of the contributing factors to AKI may be mitochondrial dysfunction, where cells, despite normal oxygen supply, are unable for proper oxygen extraction [26,28].

Thiamine deficiency, as previously mentioned, may cause high-output heart failure with vasodilatory shock and inadequate cellular oxygen extraction [1], which has previously been associated with renal failure [26]. Moreover, vitamin B1 requirement considerably increases in metabolically active cells, i.e. during fever or sepsis, since a 1°C rise in body core temperature will result in a 10% increase in the basal metabolic rate [28]. Thus thiamine supplementation has emerged as a possible pharmacological treatment for improvement in mitochondrial function and renal protection in sepsis [26,28]. In one secondary analysis of a randomized, controlled trial comparing thiamine to placebo in patients aged 68 ± 16 with septic shock, the ones who received thiamine had lower serum creatinine levels and a lower rate of progression to renal replacement therapy than patients randomized to placebo [26]. However, until now, despite an excellent safety profile, good biologic rationale, and promising results of clinical trials, routine vitamin B1 supplementation to improve outcomes in sepsis is not recommended [28]. The design of available studies varies noticeably, regarding the dosage and timing of thiamine administration, sample sizes, and different septic phenotypes [28]. Future, prospective trials are necessary to clearly define the function of thiamine in sepsis-related renal dysfunction [26].

Gastric surgery beriberi

Gastric surgery is frequently associated with numerous complications such as pernicious anemia, malabsorption, or malnutrition. Increasing evidence points to a rising incidence of vitamin B1 deficiency in the postoperative period and elderly people are at a high risk of its development [29-31]. The observed symptoms include generalized edema and exertional dyspnea along with numbness and weakness in the extremities [29]. In a Japanese study, decreased serum vitamin B1 levels were recognized in 12 out of 87 gastrecomized patients for gastric cancer or colorectal cancer (radical surgery). The median age was 59 ±13 years. Postoperative serum vitamin B1 levels were significantly lower 6 months after the operation [30]

Noteworthy, also bariatric surgery carries the risk of thiamine depletion. In a retrospective study of 147

bariatric patients between the age of 18- and 65 years old, who underwent vertical sleeve gastrectomy, 25.7% had thiamine deficiency. The majority were female [31]. Thus, as morbid obesity is becoming a global problem and the number of surgeries is increasing, physicians should keep in mind the possible thiamine deficiency in gastrectomized patients and monitor its blood level in the post-operative period. When thiamine stores are depleted in the organism, which happens approximately 3-4 weeks after the inappropriate intake, symptoms, like HF, start to emerge and may have severe consequences if thiamine therapy is not implemented in time [1,21].

Diagnosis of thiamine deficiency

Laboratory blood testing (red blood cell -RBC- transketolase activity, pyrophosphatase effect assessment, and plasma and intraerythrocytic thiamine levels) can be helpful when suspecting vitamin B1 deficiency [36]. The thiamine pyrophosphatase effect is considered particularly effective during the beriberi diagnostic pathway. The physiological background consists of the fact that when thiamine pyrophosphatase activity, which depends on the RBC transketolase enzyme, is low, an increase is observed after thiamine injection in vitro [36]. However, any of these tests can be performed routinely. The measurement of serum thiamine concentration is difficult, complicated, and uncommon from a technical point of view. Moreover, obtaining the results frequently requires a lot of time and lacks specificity [6,7]. Therefore the tests are performed only in highly-specialized centers. Routinely, empirical thiamine therapy is thought to be both safe and effective for the detection of disorders caused by vitamin B1 insufficiency [37].

Management

The early recognition and rapid administration of parenteral thiamine in suspected beriberi are crucial to prevent fatal consequences of the disease [21]. The treatment is met with favorable clinical response and withdrawal of symptoms [37,38]. The cause should be identified and further targeted treatment, i.e. sepsis or oncological management, should be implemented along with thiamine replacement.

Conclusions

Thiamine deficiency is of high prevalence in elderly people even though frequently remains underdiagnosed. The causes are numerous and diagnosis is challenging due to unspecific symptoms, frequently overlapping with those of comorbidities, and a lack of widely-obtainable diagnostic tests. However, treatment with vitamin B1 leads to quick and effective clinical response. Future tailored studies are necessary for better prognosis assessment.

Conflict of interest
None

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