

A Case report of Cardiac Beriberi: A Commonly Misdiagnosed Disease

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Abstract: Thiamine deficiency, or beriberi, is relatively rare in countries like Somalia, where alcohol consumption is uncommon and is usually related to alcohol abuse and chronic illnesses such as cancer. Despite that fact, it is necessary to ask the patients about their bad habits to unmask the etiology of the disease. A 30-year-old male who had been abusing alcohol and smoking heavily for the last six years presented with lower extremity edema, fatigue on exertion, and heart palpitations within a month. Previously there was no known chronic disease and immunosuppression drugs use, nor any documented family history of any heart disease. Upon inspection, He had a chronically ill-looking appearance but was oriented to time, place, and person. His vital signs were a blood pressure of 115/80 mm Hg, heart rate of 110 beats/min, and body temperature of 36.5°C. Peripheral oxygen saturation was around 97% without oxygen support, despite having subjective dyspneic

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symptoms. Transthoracic echocardiography showed LV global hypokinesia with severe reduction in LV systolic function (as demonstrated by an LV ejection fraction of 30%). Empirical oral Thiamine 200mg replacement was initiated emergently as a thiamine test was not available. Within ten days of thiamin supplement and heart failure medications, including furosemide 20mg, Aldactone 25mg, and carvedilol 6.25mg, the ejection fraction improved up to 45% also, the fatigues and dyspnea were also highly improved. We identified that heavy alcoholic drinking induced heart failure with reduced ejection fraction and that thiamine supplements improved these circumstances.

Keywords: Wet beri beri; Heart failure; Thiamine deficiency

Introduction

There are two main classifications of thiamine deficiency (beriberi): wet beriberi, which includes Wernicke-Korsakoff syndrome and lactic acidosis, and dry beriberi, which involves peripheral neuropathy (1).

This vitamin may be nutritionally deficient or result from alcohol consumption. A thiamine deficit (TD) may result in changes in cardiac metabolism because thiamine (vitamin B1) is a cofactor of important metabolic enzymes. But little is recognized about how TD affects the myocardium. Thiamine is regarded as having a clinically significant role in heart function, and it has been suggested that heart failure might result from its shortage (2).

Due to the body's limited capacity to store thiamine and its short half-life of 10 to 20 days, high-risk individuals might develop thiamine deficiency very quickly. The harmful effects of thiamine shortage on the myocardium are made worse by coexisting hypomagnesemia, which is probably caused by long-term alcohol addiction (3).

Due to the complex clinical presentation and lack of diagnostic tests, particularly in non-drinkers, thiamine deficiency is still going misdiagnosed (4).

We report a case of heart failure with a reduced ejection fraction that demonstrate some factors that may confound and clarify the presence of wet beriberi.

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Case report

A 30-year-old male who had been heavily abusing alcohol and smoking for the previous six years, presented lower extremity edema, fatigue on exertion, and heart palpitations within a month. On admission, the patient was on oral furosemide 40 mg and spironolactone 25 mg for the past two weeks as he visited a local health facility. The patient was diagnosed with alcoholic liver disease because he had abdominal ascites and a history of heavy alcohol consumption. The lower limb edema and abdominal distension improved, but the patient's fatigue and dyspepsia remained unchanged. There is no past medical history of chronic diseases like hypertension, diabetes, and immunosuppression disease; likewise, there is no documented family history of heart disease.

On examination, he appeared chronically ill-looking but alerted with mild hyperventilation. His vital signs were a blood pressure of 120/90 mm Hg, heart rate of 83 beats/min, and body temperature of 36.5°C. Peripheral oxygen saturation was approximately 97% without oxygen supplementation despite the subjective dyspneic complaint. Anemia and jaundice were not revealed on physical examination. The liver and spleen were not palpable. His jugular vein was not distended (the vertical distance between the sternal angle and the highest pulse point was 6 cm H₂O), and 2+ peripheral edema was noted. Vesicular breathing was heard in both lungs with normal heart sounds. His cognition and cerebellar examinations were normal. Laboratory tests revealed WBC 3.5 x1000/mm³, RBC 5.8 x10⁶/mm³ HGB 16.5g/dl, MCV 85.4fl, MCH 28.2 pg/cell, MCHC 33.4 g/dl. Aspartate aminotransferase and alanine aminotransferases were 81 IU/L (normal range: 0 IU/L to 35 IU/L) and 47 IU/L (normal range: 0 IU/L to 45 IU/L), respectively. Alpha-fetoprotein were 2.17 ng/ml (normal range: 0 ng/ml to 20 ng/ml), CA-19-9 7.8 u/ml (normal range: 0 u/ml to 37 u/ml), Serum albumin was 3.1 g/dL (normal range: 3.5 g/dL to 5.5 g/dL).

Remaining tests like renal function test, electrolytes, HBsAg, Anti HCV, and Anti-HIV were seen in normal ranges. An abdominal ultrasound revealed normal abdominal ultrasound. A chest x-ray was unremarkable on the patient's admission (Figure 1). Transthoracic echocardiography showed LV global hypokinesia with severe reduction in LV systolic function (as demonstrated by an LV ejection fraction of 30%) and normal LV size (end-diastolic dimensions: 49 mm and end-systolic dimensions: 44mm). The patient was given oral thiamin 200mg as a thiamine test was not available then. Within ten days of thiamine supplement and heart failure medications like furosemide 20 mg orally once daily, spironolactone 25 mg orally once daily, and carvedilol 6.25mg oral twice daily, the ejection fraction

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improved up to 45% also, the fatigues and dyspnea were also highly improved. After three months, the patient showed up for a follow-up with no cardiac complaint.



Figure 1. As shown in this PA view chest X-ray, there was no obvious lung pathology.

Discussion

Thiamine is a water-soluble B vitamin that functions as a cofactor in the metabolism of carbohydrates and energy synthesis. Additionally, the synthesis of neurotransmitters requires thiamine (5). A severe thiamine deficiency may result in cognitive decline (Wernicke's encephalopathy), peripheral neuropathy (dry beriberi), or heart failure (cardiac, or "wet beriberi"). Cardiac beriberi develops due to decreased cellular metabolism, which lowers cardiac function. Adenosine triphosphate (ATP) generation is impaired by thiamine deficiency, which results in adenosine accumulation. Thiamine deficiency impairs the production of adenosine triphosphate (ATP), leading to the accumulation of adenosine. This increase causes a reduction in systemic vascular resistance via direct vasomotor depression, leading to a compensatory high-output state with increased blood volume (6). Eventually, myocardial weakness develops, leading to systolic dysfunction and a low-output state. Ultimately, patients develop hypotension and complete cardiovascular collapse unless thiamine is provided (7). Shoshin beriberi, a severe form of cardiac beriberi, can potentially cause more sudden cardiogenic shock and death. Patients with cardiac beriberi typically have upper-body cachexia but significant lower-extremity edema due to heart failure. However, patients with calorie-rich but nutritionally deficient diets or those whose diets have recently changed

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may not appear malnourished. Echocardiography may show a decreased ejection fraction similar to other dilated cardiomyopathies. Some studies recommend using cardiac magnetic resonance imaging to make a diagnosis based on high T2 signal intensity caused by myocardial edema; however, further investigations discover that myocardial edema may not always be present.

The patient's medical history is very important for a wet beriberi diagnosis. Long-term drinking can lead to decreased vitamin B1 absorption and storage dysfunction and can increase damage; long-term drinking is the most common cause of wet beriberi. According to whether the patient has a history of long-term drinking or not, beriberi can be divided into alcohol-related beriberi and non-alcohol-related beriberi (8). Similarly, our case has long-term alcohol drinking, which strongly supports the possibility of heart failure secondary to thiamine deficiency.

Furosemide administration is related to thiamine deprivation, as it causes increased urinary thiamine excretion and is thus frequently associated with low thiamine intake levels (12, 13). In our case, there is no history of furosemide use before the illness. Acute renal failure was the most common wet beriberi complication, with some patients requiring CRRT (9, 10).

However, in our case, the renal function test was normal, and the patient did not mention a previous history of renal failure. Watson et al. reported that 39.4% (13/33) of wet beriberi patients had acute renal failure with high levels of blood lactate and pyruvate, which produced peripheral arteriovenous shunts, renal vascular contraction and blood flow reduction, resulting in a decreased glomerular filtration rate.

Treatment with vitamin B1 is helpful for the diagnosis and treatment of wet beriberi. It is generally accepted that suspected patients should be given a therapeutic administration of thiamine. Thiamine therapy is thought to be safe, even though measuring blood thiamine content is challenging, complex, and unusual. As a result, data frequently come late and lack specificity. When the blood level of thiamine was very high, there were no recorded signs of toxicity (8). Authors differ in how they administer thiamine for wet beriberi. Since alcohol can prevent the absorption of vitamin B1 and the phosphorylation of its active form (TPP), patients with beriberi caused by alcohol often received a larger dose of thiamine than those with non-alcohol-related beriberi (11, 12). Currently, daily intravenous therapy of 100 to 200mg of thiamine is the most common treatment for those with alcohol-related wet beriberi. In our case, the condition significantly improved after thiamine administration. Although the patient was previously on

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some heart medications, this makes the case more interesting and strongly indicates thiamine deficiency was the underlying cause of the heart failure.

Conclusion

In conclusion, cardiac beriberi related to thiamine deficiency is difficult to diagnose due to its nonspecific symptoms, signs and rarity, particularly, in countries like Somalia, where alcohol consumption is uncommon. However, cardiologists should still consider cardiac beriberi a possible diagnosis in young patients with unexplained LV systolic dysfunction and heart failure.

Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. On request, a copy of the written consent is available for review by the editor-in-chief of this journal.

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