Thiamine Deficiency as a Cause for Acute Circulatory Failure: An Overlooked Association in Western Countries

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ABSTRACT
A 42-year-old patient presented with circulatory failure and lactic acidosis. Clinical features, later coupled with biological tests, led to the diagnosis of wet beriberi syndrome and scurvy. Echocardiography showed a pattern of thiamine deficiency with high cardiac output and low vascular resistance. The patient’s condition and biological parameters immediately improved after treatment injections of thiamine. Wet BeriBeri is often overlooked in western countries and is a diagnosis that must be considered based on history, and clinical and echocardiographical findings.

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Case
A 42-year-old Caucasian man presented to the emergency department with a 1-week history of chest pain and progressive dyspnea. On admission, he had minimal peripheral edema and circulatory failure features with low blood pressure of 90/50 mm Hg, tachypnea at 40 per minute, and tachycardia of 130 beats per minute. Capillary refill time was higher than 3 seconds. A grade 2/6 systolic ejection murmur was found, as well as bilateral pulmonary basal crackles. There was no fever, no ascites, or any stigmata of cirrhosis. Periodontal disease (with bleeding gums) and features of scurvy were also observed. He admitted to chronic alcohol consumption of 20 g/day and a diet restricted to rapidly absorbed refined carbohydrates. Past medical history was otherwise unremarkable. He had no history of toxic ingestions, which was later confirmed with a toxicology screen.

Electrocardiogram showed regular sinus rhythm with a negative T wave in lead augmented vector left. Initial blood tests (Table 1) indicated high levels of N-terminal pro-B-type natriuretic peptide (NT-proBNP) (7527 ng/L), elevated troponin T (high-sensitivity cardiac troponin T [hs-cTnT]; 125 ng/L), and lactic acidosis (6.3 mmol/L). A transthoracic echocardiogram revealed preserved left-ventricular ejection fraction (LVEF 50%) with inferior hypokinesia and non-dilated left cavities (left ventricle diastolic diameter 55 mm; left atrial surface 18 cm²). The right-ventricle ejection fraction was also preserved with a tricuspid annular plane systolic excursion of 20 mm. Estimated systolic pulmonary artery pressure was 50 mm Hg. A 2-mm pericardial effusion was found anterior to the right atrium and ventricle. Hemodynamic echocardiographic profile assessment indicated high cardiac output at 9.1 L/min (indexed cardiac output of 4.9 L/min per m²), with velocity time integral at 22 cm and heart rate at 110
Thiamine deficiency is a well known, yet unfrequent cause of circulatory shock and lactic acidosis in western countries and remains a health hazard, especially as a result of malnutrition and alcohol consumption.

Wet beriberi must be considered based on history, and clinical and echocardiographical findings, without waiting for thiamine testing in patients with dietary decompensation.

A small drop in thiamine blood levels may be sufficient to explain lactic acidosis and hemodynamic instability.

Scurvy can occur in conjunction with wet beriberi in patients with altered nutritional status.

Discussion

Thiamine deficiency is an unusual cause of circulatory failure and remains a health hazard, especially due to malnutrition. Treatment with thiamine is safe and effective, highlighting the importance of suspecting the diagnosis early, particularly in an emergency situation. Its prevalence has never been evaluated in western countries, and the literature is limited to a few case reports. To the best of our knowledge, there are no published data describing thiamine concentration at clinical presentation. In a few case reports, blood levels were found not to be severely depressed. To highlight this finding, Martin et al. discussed that not all people are equally sensitive to thiamine deficiency, with consequences depending on genetic predisposition, alcoholic and chronic nutritional state, arguing for the existence of thiamine-using enzyme variants. The capacity for thiamine uptake into the cells may also play a role. However, to clarify these points, genetic studies would be required.

Beriberi is a well known etiology of high-output cardiac failure. Several mechanisms have been proposed to explain the observed hemodynamic abnormalities.

Our case describes a patient with cardiogenic shock due to thiamine deficiency and scurvy. Right heart failure has been previously described in patients with scurvy, mainly as a result of severe pulmonary hypertension. Vitamin C has vasodilatory properties, increasing availability of endothelial nitric oxide. In our case, the data and the complete recovery after thiamine administration suggest the exclusive accountability of thiamine deficiency in the presentation. Diagnosing wet beriberi requires a high clinical level of suspicion based on medical history and physical examination. The only way to establish the diagnosis is to consider it, and monitor improvement after thiamine administration, as blood tests are not rapidly available (they take weeks to be processed).

Funding Sources

The authors have no funding sources to declare.
Disclosures
The authors have no conflicts of interest to disclose.

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Figure 1. Acute clinical evolution and biological findings kinetics, for a patient with circulatory shock due to wet beriberi and scurvy. Thiamine injections were started upon admission. bpm, beats per minute; CO, indexed cardiac output; H, hour; HR, heart rate; MBP, mean blood pressure; nT-pro BNP, N-terminal pro-B-type natriuretic peptide; SBP, systolic blood pressure; SVR, systemic vascular resistance.