

Beriberi Disease: Forgotten but Not Gone!

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Abstract

Case Report

Beriberi is a Sinhalese phrase that means “I cannot, I cannot,” in reference to the profound weakness that may accompany the disorder, it is currently considered as an uncommon disease [1], caused by thiamine (vitamin B1) deficiency, this may result in under recognition of potentially treatable cases. Beriberi has two major sub-types or possible manifestation: Wet beriberi, when the predominant symptoms are related to the cardio-circulatory system leading to a high-output heart failure and even cardiogenic shock, and Dry beriberi in which the peripheral or central nervous systems are affected, but it is more likely to not see a full classic example of wet or dry beriberi, but rather a combination of symptoms that may lead in the absence of adequate treatment to the full picture of thiamine deficiency. One of the most important elements of diagnosis is the medical history, to look for factors that may cause or participate in the disorder (diet, alcohol consumption, pregnancy, hyperthyroidism, liver disease..) and eventually help treating the underlying causes. We report a case of an atypical beriberi disease associated with ST segment elevation with no obstructive coronary artery disease.

Keywords: Beriberi, vitamin B1, ST segment elevation, pregnancy, Wernicke–Korsakoff syndrome.

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INTRODUCTION

Thiamine is a soluble vitamin essential for carbohydrate metabolism, production of energy and neurotransmission, despite the crucial importance, the storage is limited [2], leading to its deficiency in some particular situations that will be discussed later. Cardiac beriberi occurs as a result of impaired metabolism leading to a decreased cardiac function. The diagnosis represents a true challenge owing to the absence of pathognomonic manifestation and requires a thorough investigation, exclusion of other etiologies and even a positive therapeutic test response.

The prevalence of thiamine deficiency was found in up to 30% hospitalized for acute heart failure [3], we also have small studies that showed functional improvement after supplementation regardless of the etiology of the cardiomyopathy [4-6]. Still, ST segment elevated acute coronary syndrome is a rare presentation of the deficiency, and deserves a particular consideration since it can be easily overlooked.

OBSERVATION

We received a 33 years old woman, at 12 weeks of pregnancy, with no known cardio-vascular

risk factors, she was under treatment for a recently discovered hyperthyroidism, and presented a severe form of hyperemesis.

She consulted our emergency department for retrosternal chest pain, associated with an extreme fatigue and anorexia.

Clinical examination found no significant signs, EKG showed a normal sinus rhythm at 75 Bpm, ST segment elevation in the inferior leads concomitant with an ST depression in the anterior territory.

Routine lab tests objectified a low potassium level and reduced prothrombin, successfully corrected with the injection of intravenous vit K.

Tran's thoracic echocardiography showed an inferior hypokinesia with a preserved ejection fraction at 50%.

After confirming the absence of contraindications, we proceeded to thrombolysis using Metalyse, 2 hours later the EKG showed a regression of the ST segment elevation.

The day after, the patient presented a ventricular tachycardia reduced successfully with an external cardiac electric choc, coronary angiography was preformed, the images showed no signs of atherothrombotic lesion.

Cardiac MRI was also done in the light of exploring what it seemed to be a myocardial infarction with no coronary injury, it revealed signs of localized myocarditis, patient was already on double anti-aggregation, Beta-blockers and ACE inhibitors.

During her hospitalization, the patient presented numerous tonico-clonic seizures, with no detected anomaly is the head CT.

The association of cardio-neuro manifestations and the patient profile (anorexia, pregnancy, hyperthyroidism, vomiting) vit B1 deficiency was suspected and later confirmed with lab tests.

The treatment consisted of injecting 500 mg of Vit B1 per day, quick amelioration of symptoms was noticed.

The follow-up at 36 weeks of pregnancy was good, transthoracic echocardiography showed a completely normal cardiac function, no signs of heart failure, the patient delivered her full-term baby with no incident.

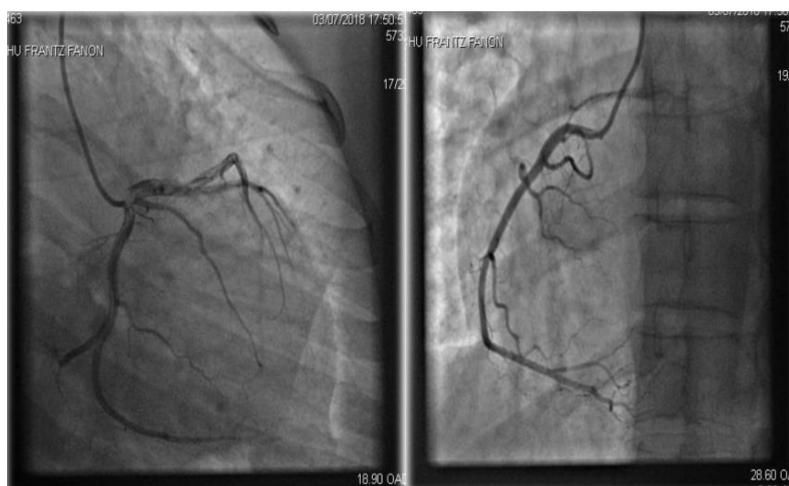


Figure1: Angiography of the right and left coronaries

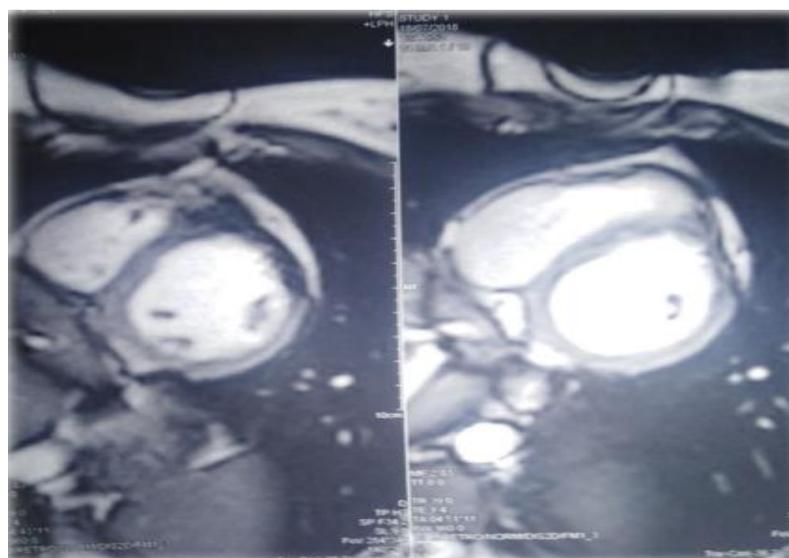


Figure 2: MRI image of the same patient

DISCUSSION

Thiamine or vitamin B1, serves as a cofactor for several enzymes involved in energy metabolism.

Thiamine deficiency is now very rare in developed countries, but still common in South East

Asia specially in developing countries. It is an important public health problem with potentially fatal consequences.

Primary thiamine deficiency is caused by inadequate intake of thiamine, most commonly in under

developed countries. The great outbreaks of thiamine deficiency in Southeast Asia at the beginning of the 20th century followed the large-scale production of milled rice and its extensive distribution.

Secondary thiamine deficiency is caused by increased requirement, as in hyperthyroidism, pregnancy, lactation and fever. It is also associated with impaired absorption, as in prolonged diarrheas and impaired utilization, as in severe liver disease.

There are 2 major manifestations of thiamine deficiency [7]: cardiovascular disease (wet beriberi) and nervous system disease (dry beriberi and Wernicke–Korsakoff syndrome).

- Wet beriberi occurs in thiamine deficiency when myocardial disease is prominent. This causes a high cardiac output with peripheral vasodilation due to the accumulation of pyruvate and lactate. Before heart failure occurs, tachycardia, a wide pulse pressure, sweating, warm skin, and lactic acidosis develop, leading to sodium and water retention by the kidneys. The resulting fluid overload leads to edema of the dependent extremities. A more rapid form of wet beriberi has been termed acute fulminant cardiovascular beriberi with lactic acidosis or Shoshin beriberi, in which vasodilation continues, resulting in shock in a patient with heart failure. While thiamine deficiency is also associated with ST-segment elevation in some animal models [9], there has been no report of ST-segment elevation in humans with beriberi [1, 8].
- Symptoms of dry beriberi are bilateral and symmetric, predominantly involving the lower extremities and beginning with paresthesia of the toes, burning of the feet (particularly severe at night), muscle cramps in the calves, and pain in the legs. Calf muscle tenderness, difficulty in rising from a squatting position, a decrease in the vibratory sensation in the toes, and plantar dysesthesia are early signs. A diagnosis of mild peripheral neuropathy can be made when ankle jerks are absent. Continued deficiency causes loss of knee jerk, loss of vibratory and position sensation in the toes, atrophy of the calf and thigh muscles and finally foot drop and toe drop. Arms can be affected after leg signs are well established.
- In Wernicke–Korsakoff syndrome-manifestations consist of nystagmus, ophthalmoplegia and ataxia evolving into confusion, retrograde amnesia, cognitive impairment and confabulation

We reported a case of a 33-year-old woman with beriberi disease who demonstrated ST-segment elevation and myocardial damage without coronary artery stenosis. The clinical presentation was atypical as

it associated incomplete form of dry beriberi, with only fatigue, muscle weakness and tonico-clonic seizures that can be rare but possible especially if B6 deficiency is present.

The patient subsequently recovered with thiamine treatment. We conclude that it is important to consider beriberi as part of the differential diagnosis in patients with ST-segment elevation with no coronary stenosis in the presence of an adequate context of deficiency, especially if associated with neurological signs.

CONCLUSION

Thiamine deficiency is nowadays a rare disease in near eradication in some parts of the globe, but it remains, in the presence of a favoring context, a serious complex syndrome responsible for dramatic manifestation resulting in death, but yet totally reversible under adequate treatment.

REFERENCES

1. Ito, M., Tanabe, Y., Suzuki, K., Kumakura, M., & Aizawa, Y. (2002). Shoshin Beriberi With Vasospastic Angina Pectoris Possible Mechanism of Mid-Ventricular Obstruction. *Circulation journal*, 66(11), 1070-1072.
2. Donnino, M. W., Carney, E., Cocchi, M. N., Barbash, I., Chase, M., Joyce, N., ... & Ngo, L. (2010). Thiamine deficiency in critically ill patients with sepsis. *Journal of critical care*, 25(4), 576-581.
3. Hanninen, S. A., Darling, P. B., Sole, M. J., Barr, A., & Keith, M. E. (2006). The prevalence of thiamin deficiency in hospitalized patients with congestive heart failure. *Journal of the American College of Cardiology*, 47(2), 354-361.
4. Schoenenberger, A. W., Schoenenberger-Berzins, R., Der Maur, C. A., Suter, P. M., Vergopoulos, A., & Erne, P. (2012). Thiamine supplementation in symptomatic chronic heart failure: a randomized, double-blind, placebo-controlled, cross-over pilot study. *Clinical research in cardiology*, 101, 159-164.
5. Shimon, H., Almog, S., Vered, Z., Seligmann, H., Shefi, M., Peleg, E., ... & Ezra, D. (1995). Improved left ventricular function after thiamine supplementation in patients with congestive heart failure receiving long-term furosemide therapy. *The American journal of medicine*, 98(5), 485-490.
6. Seligmann, H., Halkin, H., Rauchfleisch, S., Kaufmann, N., Tal, R., Motro, M., ... & Ezra, D. (1991). Thiamine deficiency in patients with congestive heart failure receiving long-term furosemide therapy: a pilot study. *The American journal of medicine*, 91(2), 151-155.
7. Hassan, M., Rahman, H., Yasmeen, B. N., Mukti, A., Haque, H., Khan, M., ... & Kabir, A. L. (2018).

Thiamine deficiency-Beriberi—A forgotten disease. *Northern International Medical College Journal*, 10(1), 351-354.

8. Kawano, H., Koide, Y., Toda, G., & Yano, K. (2005). ST-segment elevation of electrocardiogram

in a patient with Shoshin beriberi. *Internal medicine*, 44(6), 578-585.

9. Read, D. H., & Harrington, D. D. (1981). Experimentally induced thiamine deficiency in beagle dogs: clinical observations. *American journal of veterinary research*, 42(6), 984-991.