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Case Report
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A Case of Beriberi with Leg Edema, Pleural Effusion, and Anemia

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Abstract

Case: We describe a case of beriberi with leg edema, pleural effusion, and anemia. Echocardiography revealed that the inferior vena cava diameter was 18 mm, but respiratory changes were not observed. Pulmonary hypertension was observed, with a systolic pulmonary arterial pressure of 45 mmHg. Peripheral blood concentrations of vitamin B1 decreased significantly. Beriberi heart disease caused by vitamin B1 deficiency was considered to lead to systemic edema; continuous vitamin supplementation maintained cardiac movement, and the anemia did not progress.

Keywords: Vitamin B1; Beriberi heart disease

Introduction

Beriberi heart disease caused by vitamin B1 (thiamine) deficiency was considered rare; however, recently, it has emerged in elderly persons and heavy drinkers. Wet beriberi is characterized by symptoms of high output heart failure due to retention of sodium and water, peripheral vasodilation, and ventricular failure [1]. We report a case of beriberi with pleural effusion and leg edema. The patient's condition rapidly improved after vitamin B1 replacement.

Case Report

An 86-year-old man experienced severe leg edema and was hospitalized. Lower leg edema had developed 6 months ago, and he experienced difficulty walking. The edema spread to the upper leg region, and the patient experienced dyspnea in the decubitus position, which occurred 4 months ago. He visited a doctor and underwent medical examination 1 month ago. He was hospitalized because of pleural effusion and leg edema. This patient's oxygen saturation (SpO_2) was 92% at room air at his admission. SpO_2 was improved to 99% at discharge.

The patient had a history of congestive heart failure, atrial fibrillation, hepatitis C virus infection, and liver cirrhosis (Child-Pugh grade B). At the age of 83 years, he underwent low anterior resection for colon cancer. He had no allergy to food or drugs and consumed 80 g/day of alcohol for 60 years. He was a heavy smoker as he smoked 150 cigarettes per day for 43 years.

He was 162 cm in height and 59.3 kg in weight; on admission, his blood pressure and body temperature were 106/51 mmHg and 36.1°C, respectively. Heart and breath sounds were unremarkable. Bilateral pitting edema was observed. His deep tendon (patellar and Achilles tendons) reflex had weakened. No laterality was observed in the sensory nerve, limbs sense of touch and pain. Laboratory findings at hospitalization are listed in Table 1. An electrocardiogram showed atrial fibrillation (Figure 1). Chest radiography indicated cardiomegaly (cardiothoracic ratio=67.9%) and bilateral pleural effusion, markedly significant in left (Figure 2). Upper endoscopy revealed atrophic gastritis without gastric ulcers. Abdominal ultrasonography revealed liver cirrhosis, mild splenomegaly, and cysts in the right kidney. Echocardiography revealed that the inferior vena cava diameter was normal, that is, 18 mm, but respiratory changes were not observed. Pulmonary hypertension was observed, with a systolic pulmonary arterial pressure of 45 mmHg. The ejection fraction was 68%, and no left ventricular asynergy was observed.

A catheter was inserted into his right thoracic cavity after hospitalization, and pleural effusion was extracted from the left chest; hence, the effusion decreased. Pleural effusion was defined as transudates (pleural fluid-to-serum protein ratio=0.5). Anemia was identified macrocytic anemia; additionally, pancytopenia was observed. The patient was transfused 400 mL of red blood cells; however, subsequently the anemia progressed.

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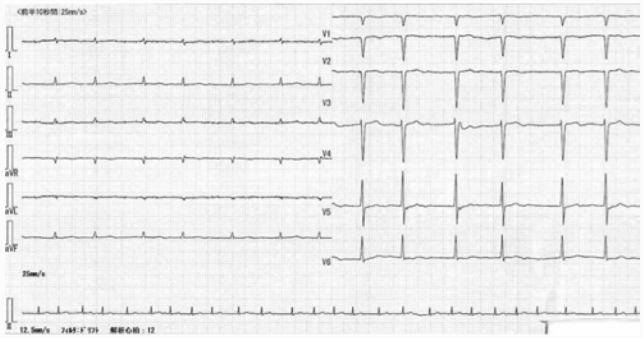

Figure 1: Electrocardiogram showed a trial fibrillation.

Table 1: Laboratory findings.

Peripheral blood	
WBC	2800 μL
Blast	0
Promyelo	0
Myelo	1
Meta	0
Stab	3
Seg	71
Eosino	1
Baso	1
Mono	1
Lympho	12
RBC	$209x10^4$ μL
Hb	6.8 g/dL
Ht	19.80%
Plt	$11.4x10^3$
Coagulation study	
PT	78%
APTT	31.9s
HPT	87%
Fibrinogen	221 mg/dL
FDP	5 μg/mL
D-dimer	2.1μg/mL
Urinary	
Protein	(-)
Glucose	(-)
Keton	(-)
Urobilinogen	(-)
Occult	(-)
Bone Marrow	
NCC	$240x10^3$/uL
Blast	1.2
Promyelo	1.6
Myelo	11.8
Meta	3.2
Stab	13.4
Seg	11.4
Eosino	1.6
Baso	0.2
Mono	1
Lympho	12
Chemistory	
AST	28 U/L
ALT	13 U/L
LDH	209 U/L
ChE	95 U/L
T-Bil	0.8 mg/dL
ALP	155 U/L
γ-GTP	56 U/L
TP	5.1 g/dL
α1-g	4.10%
α2-g	8.20%
β-g	11.40%
γ-g	20.5%
TG	109 mg/dL
LDL-C	55 mg/dL
HDL-C	35 mg/dL
CK	41 mg/dL
BUN	25 mg/dL
Cr	0.91 mg/dL
eGFRcre	60 ml/min/1.73m^2
BNP	240.7
CRP	0.1 mg/dL
HBs-Ag	0
HCV-Ab	4.53
CEA	6.5 ng/mL
AFP	2 ng/mL
CA19-9	30 U/mL
Vitamin B1	12 ng/mL
Vitamin B2	43.6 ng/mL
Vitamin B12	430 pg/mL
Folic acid	5.6 ng/mL
Fe	148 μg/dL
UIBC	87μg/dL
Ferritin	570 ng/mL
T-SPOT	(-)
Plueral effusion	
hyaluronic acid	5340 ng/mL
ADA	42.8 U/L
CEA	2.1 ng/mL
Protein	2.7 g/dL
Rivaluta	(-)

Peripheral blood concentrations of vitamins B1 and B2 decreased significantly (Table 1), and leg edema was treated via intravenous administration of thiamine (100mg i.v.). The patient was administered azosemide and trichlormethiazide (Figure 2 and 3). In addition, hypokalemia was treated with oral administration of potassium. We suspected that the patient had myelodysplastic syndrome (MDS) or lymphoma on the basis of the splenomegaly and low levels of IgM and conducted a bone marrow examination. Hyperplasia was observed, and megaloblasts were detected in the bone marrow cells. There was no formal abnormality in the bone marrow cells. MDS was examined via chromosomal examination of 46 XY, inv (3) (q21q26.2) and translocation using karyotyping of the bone marrow cells (7/20 cells). Pleural effusion decreased in the left thorax on day 23 (Figure 2). Systemic edema did not develop. Beriberi heart disease caused by vitamin B1 deficiency was considered to lead to systemic edema; continuous vitamin supplementation maintained cardiac movement, and the anemia did not progress.

Discussion

Vitamin B1 is a water-soluble vitamin that is necessary as a cofactor for pyruvate dehydrogenase complex, 2-oxoglutarate dehydrogenase (associated with the tricarboxylic acid cycle), and branched-chain ketoacid dehydrogenase. Vitamin B1 is also required

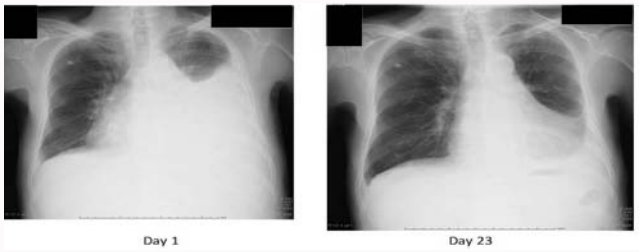

Figure 2: Chest radiography.

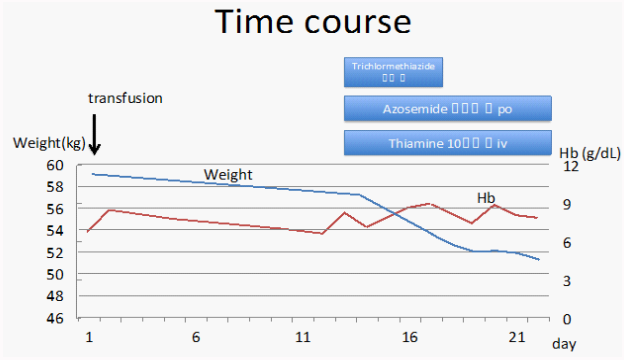

Figure 3: Time course graph of beriberi heart disease.

as a cofactor for the transketolase catalyzed reactions of the pentose phosphate pathway [2]. Vitamin B1 deficiency is known to cause beriberi heart disease and Wernicke encephalopathy [3]. The onset of this disease is more frequent in summers. Vitamin B1 deficiency occurs mainly in patients with inadequate nutrition and chronic excessive alcohol intake [4]. In addition, postgastrectomy absorption disorder and diuretic dosage have been reported as other etiological factors for developing beriberi [5]. Vitamin B1 administration can reverse enzyme activity and metabolism immediately.

A patient with beriberi heart disease shows high output cardiac failure due to abnormal decrease in peripheral artery resistance. Vitamin B1 deficiency induces the exhaustion of intracellular ATP and release of endogenous ATP outside the cells, and finally induces peripheral artery expansion. An increase in pulmonary arterial pressure and right heart failure are observed in the early stage of the disease. High output heart failure and metabolic acidosis are identified in the later stages. The most fulminant form is shoshin beriberi, which is characterized by cardiovascular collapse, and a patient might die within hours. Vitamin B1 treatment is recommended without waiting for the results of measurement of the blood vitamin B1 levels when a patient is suspected to have beriberi heart disease [6].

Massive leg edema and pleural effusion were observed in the patient, along with heart failure (including atrial fibrillation), liver cirrhosis, chronic kidney failure (CKD stage 3), excessive salt intake, or hypo-alimentation state.

This patient had macrocytic anemia, and pancytopenia was observed. Owing to anemia, the patient's condition due to high output heart failure and massive edema worsened. Differential diagnoses of pancytopenia included MDS, aplastic anemia, vitamin deficiency, collagen disease, liver cirrhosis, and hyper splenic disease. This patient underwent bone marrow aspiration examination. The bone marrow examination indicated hyperplasia, and no formal abnormality was observed; however, the patient demonstrated a chromosomal abnormality (46 XY, inv (3) (q21q26.2)) in the bone marrow cells. We hypothesized that this patient had MDS as an underlying disease and vitamin B1 deficiency-induced macrocytic anemia. We assumed that anemia was induced by both hear failure and thiamine deficiency. Blood transfusion improved hemodynamic status of this patient, but anemia was gradually worse. We estimated that thiamine deficiency was main cause of sever anemia. Thiamine-responsive megaloblastic anemia syndrome is a rare condition characterized by hearing loss, diabetes, and a blood disorder called megaloblastic anemia. The symptoms of this disorder include decreased appetite, energy loss, headaches, pale skin, diarrhea, and tingling or numbness in the hands and feet. Individuals with thiamine-responsive megaloblastic anemia syndrome show symptoms of megaloblastic anemia between infancy and adolescence [7]. On the basis of the patient's symptoms and age, thiamine-responsive megaloblastic anemia syndrome was ruled out. We regularly followed up the patient to observe the course of anemia.

Conclusion

We report a case of beriberi heart disease. In cases of high output heart failure, beriberi should be taken into consideration if a patient's lifestyle is asocial, and there is a risk of nutritional deficiency.

References

1. Blacket RB, Palmer AJ. Haemodynamic studies in high output beri-beri. Br Heart J. 1960; 22: 483-501.
2. Frank RA, Titman CM, Pratap JV, Luisi BF, Perham RN. A molecular switch and proton wire synchronize the active sites in thiamine enzymes. Science. 2004; 306: 872-876.
3. Blass JP, Gibson GE. Abnormality of a thiamine-requiring enzyme in patients with Wernicke-Korsakoff syndrome. N Engl J Med. 1977; 297: 1367-1370.
4. Grossman W, Braunwald E. Beriberi heart disease. Brounwald heart disease: A textbook of cardiovascular medicine, 4th edition, MS Saunders company, 1992; 461-462.
5. Ayzenberg O, Silber MH, Bortz D. Beriberi heart disease. A case report describing the haemodynamic features. S Afr Med J. 1985; 68: 263-265.
6. Dabar G, Harmouche C, Habr B, Riachi M, Jaber B. Shoshin Beriberi in Critically-Ill patients: case series. Nutr J. 2015; 14: 51.
7. Viana MB, Carvalho RI. Thiamine-responsive megaloblastic anemia, sensorineural deafness, and diabetes mellitus: a new syndrome?. J Pediatr. 1978; 93: 235-238.