ST-segment Elevation of Electrocardiogram in a Patient with Shoshin Beriberi

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Abstract

We report a case of a 58-year-old man with Shoshin beriberi who demonstrated ST-segment elevation and myocardial damage without coronary artery stenosis. The patient subsequently recovered with thiamine treatment. We conclude that it is important to consider Shoshin beriberi as part of the differential diagnosis in patients with shock and ST-segment elevation.
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Introduction

Beriberi is a disorder of thiamine deficiency that results in heart failure. Shoshin beriberi is the fulminant form of this disease and is characterized by hypotension, tachycardia, and lactic acidosis (1–6). While thiamine deficiency is also associated with ST-segment elevation in some animal models (7, 8), there has been no report of ST-segment elevation in humans with beriberi. We describe a case of Shoshin beriberi and ST-segment elevation, which was subsequently resolved with thiamine therapy.

Case Report

A 58-year-old man was admitted to our hospital in January 12, 1995 with symptoms of polyneuropathy. His past medical history consisted of an appendectomy and three operations for adhesive ileus. After undergoing these surgical procedures, the patient was not able to eat enough solid foods such as meat, fish, and vegetables except for rice.

On admission, he was not able to walk because of muscle weakness and atrophy of the bilateral lower extremities. He had hypesthesia (glove and stocking type) and hyporeflexia of the bilateral lower extremities. He also had mild pretibial edema. Brain computed tomography showed no abnormal finding, and examination of cerebrospinal fluid was normal. A needle electromyogram showed a neurogenic pattern. After admission, he had repeated vomition, his food intake decreased, and maintenance infusion without vitamin was started on January 24. Then, the patient experienced sudden onset of chest pain, hypotension, cyanosis, and dyspnea on January 28, 1995. Physical examination revealed blood pressure of 77/49 mmHg, and an electrocardiogram (ECG) showed sinus tachycardia, right axis deviation, a tendency to low voltage in limb leads, clockwise rotation (or poor R wave progression in precordial leads), and ST-segment elevation in leads I, $_aV_L$, and V_1 to V_6, and ST-segment depression in leads II, III, and $_aV_F$ (Fig. 1). A chest X-ray showed mild cardiomegaly and pulmonary congestion (Fig. 1), and an echocardiogram demonstrated left ventricular ejection fraction (LVEF) of 33% with diffuse hypokinesis of the left ventricle (LV), and mild pericardial effusion was seen. An emergency coronary angiogram (CAG) was performed, but the patient's coronary arteries were normal except for anomaly of a left single coronary artery (Fig. 2). Left ventriculogram (LVG) demonstrated diffuse hypokinesis and LVEF of 44%, despite the lack of coronary stenoses (Fig. 2). Right-sided and left-sided cardiac catheterization revealed a right atrial mean pressure (mRAP) of 22 mmHg, a right ventricular pressure (RVP) of 47/18 mmHg; the pulmonary arterial pressure (PAP) was 43/22 mmHg with a mean pressure of 30 mmHg; the pulmonary capillary wedge pressure (PCWP) was 35 mmHg; the left ventricular pressure (LVP) was 78/18 mmHg with an end-diastolic pressure of 23 mmHg, and the aortic pressure (AoP) was 82/44 mmHg. Cardiac output was 6.5 *l*/min (cardiac index; 3.8 *l*/min/m^2) by thermodilution catheter.

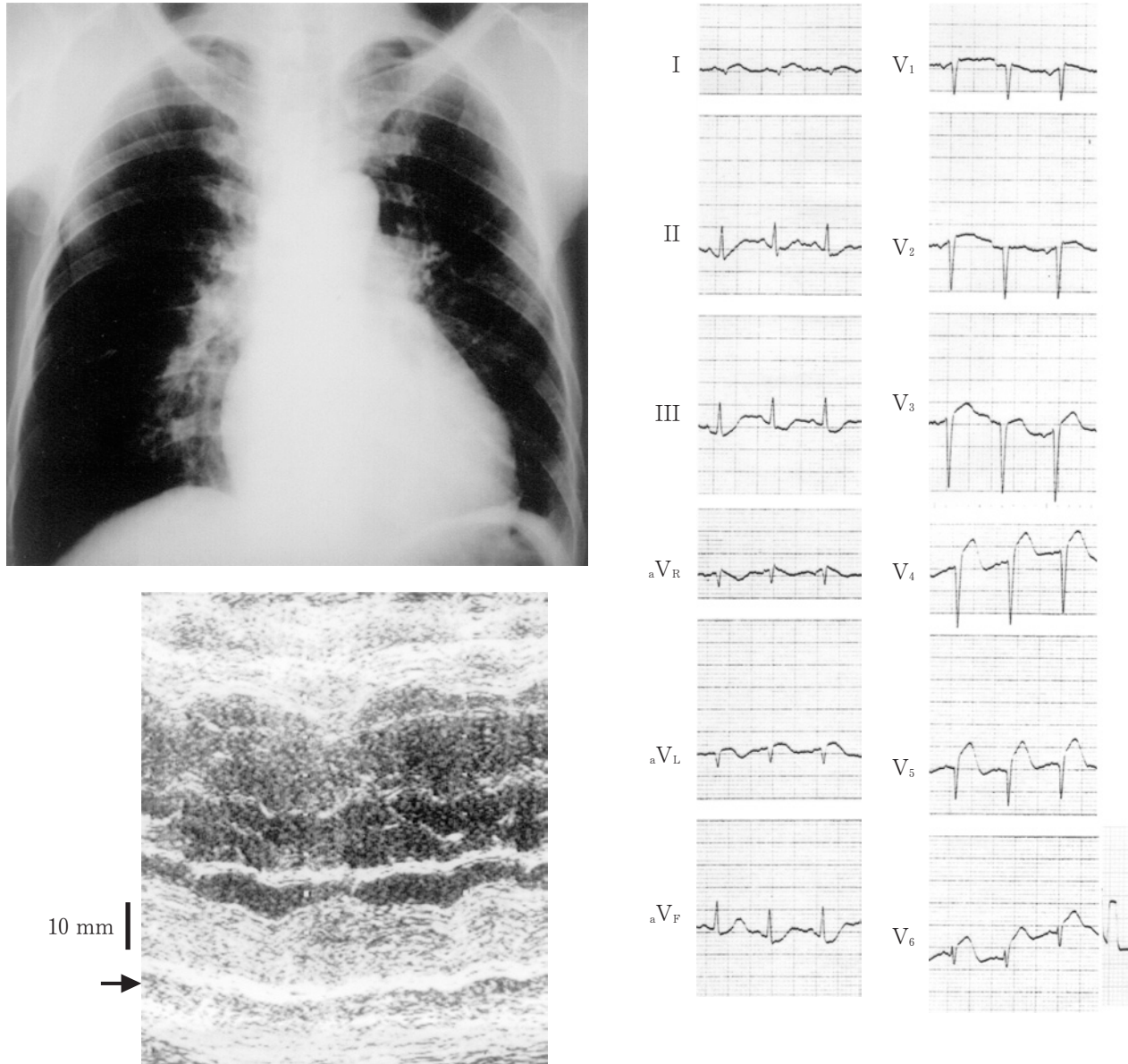The hemoglobin was 13.2 g/dl, hematcrit was 39.9%, and

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Figure 1. Chest X-ray, ECG, and echocardiogram on January 28, 1995. Chest rentogenogram shows cardiomegaly and lung congestion. ECG showed sinus tachycardia, right axis deviation, a tendency to low voltage in limb leads, clockwise rotation (or poor R wave progression), and ST-segment elevation in leads I, $_aV_L$, and V_1 to V_6, and ST-segment depression in leads II, III, and $_aV_F$. M-mode echocardiogram shows reduced left ventricular wall motion and mild pericardial effusion (arrow) (wall thickness of intraventricular thickness 10 mm, wall thickness of LV posterior wall 12 mm, LV end-diastolic diameter 56 mm, LV end-systolic diameter 47 mm, LV ejection fraction 33%).

white blood cell count (WBC) was 6,400/mm³. The blood urea nitrogen was 26 mg/dl, creatinine 1.5 mg/dl, total protein 6.0 mg/dl, total bilirubin 1.1 mg/dl, aspartate aminotransferase (AST) 34 IU/*l*, alanine aminotransferase (ALT) 37 IU/*l*, lactate dehydrogenase (LDH) 465 IU/*l*, creatine kinase (CK) 261 IU/*l*, γ-glutamyltransferase 22 IU/*l*, glucose 103 mg/dl, total cholesterol 111 mg/dl, triglyceride 69 mg/dl, HDL-C 36 mg/dl, sodium 135 mEq/*l*, potassium 4.0 mEq/*l*,

and chloride 92 mEq/*l*. Thyroid function was normal. A blood gas analysis revealed an arterial pH of 7.099, pCO_2 of 23.2 mmHg, pO_2 of 107 mmHg, HCO_3 of 6.8 mEq/*l*, and base excess of –21.1 mEq/*l*. Severe acidosis was unaffected by administration of sodium bicarbonate, and blood pressure was not increased by administration of dopamine hydrochloride.

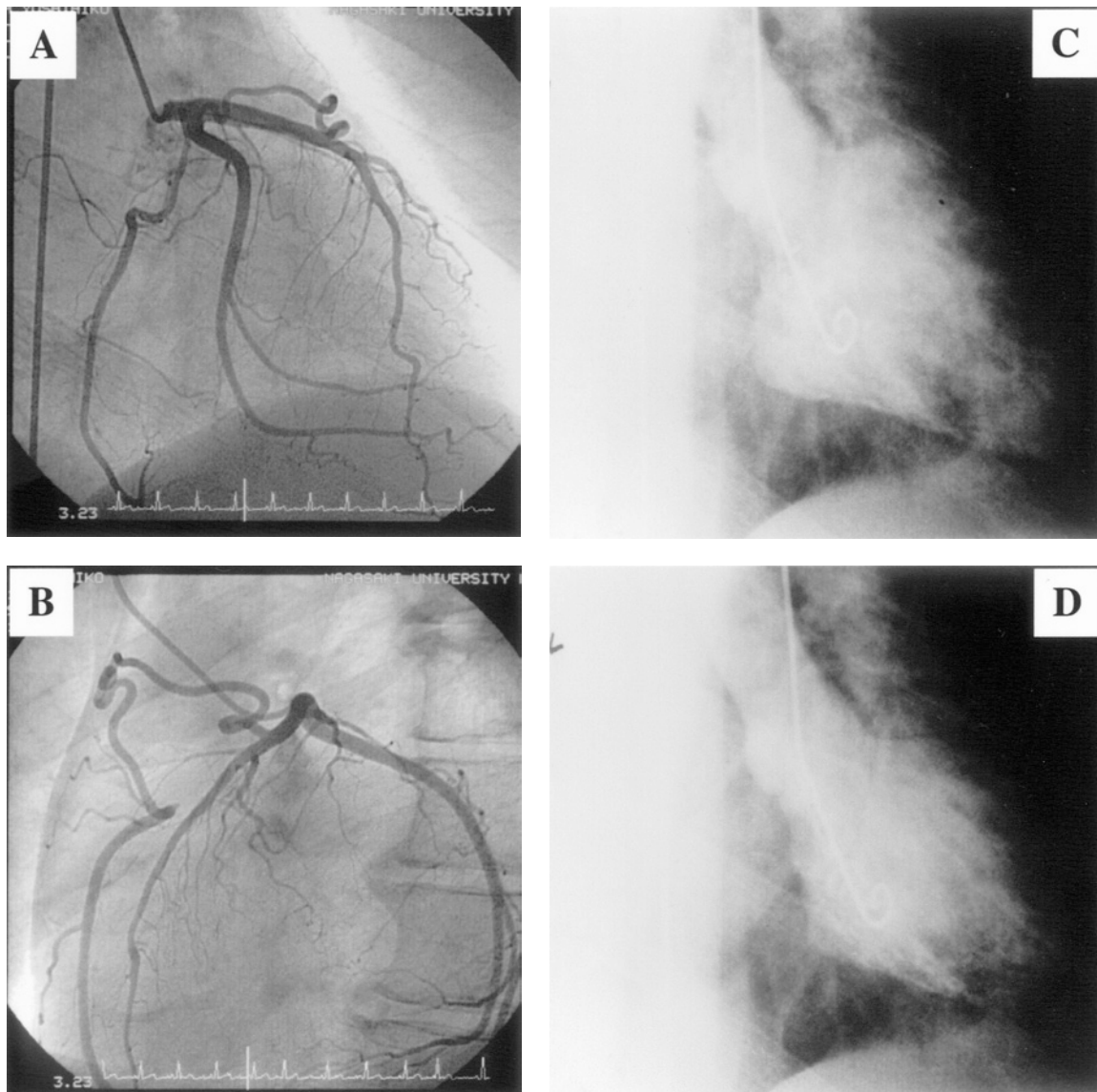Shoshin beriberi was suspected, and fursulthiamine (50

Figure 2. Coronary angiogram and left ventriculogram on January 28, 1995. An emergency coronary angiogram showed normal except for anomaly of a left single coronary artery. Left ventriculogram demonstrated diffuse hypokinesis and LVEF of 44%.

mg), a thiamine derivative, was administered intravenously. After the thiamine treatment, his blood pressure gradually increased, acidosis disappeared, and his condition improved dramatically (Fig. 3). Intravenous administration of fursulthiamine was continued for 11 days from January 28 to February 7, and fursulthiamine (150 mg/day) had been administered orally since February 8.

On January 29, 1995, cardiac output was 10.6 l/min (cardiac index; 6.1 l/min/m^2). In ECG, ST elevation in leads V$_4$ to V$_6$ disappeared, coved type ST-segment elevation in leads V$_1$ to V$_3$, and ST-segment depression in leads II, III, and $_a$V$_F$ (Fig. 4). Laboratory analysis was performed and showed elevations in WBC (15,000/mm^3), serum AST (165 IU/l),

LDH (1,148 IU/l), CK (629 IU/l), and CK-MB (32 IU/l), and peak CK was 1,134 IU/l (Fig. 5). Thiamine deficiency was confirmed by a blood test collected on January 27 (thiamine 8.7 ng/ml; normal 20–50 ng/ml).

The ST-segment elevation in V$_1$ to V$_3$ leads resolved, and inverted T waves developed in leads II, III, $_a$V$_F$, and V$_2$ to V$_3$, at five days after initiating thiamine treatment. These ECG changes normalized but T wave inversion was seen in leads I and $_a$V$_F$ on March 7, 1995 (Fig. 4). Cardiac function gradually improved and 6 weeks after the treatment, chest chest X-ray showed that the cardiothoracic ratio was normalized and lung congestion disappeared (Fig. 6). An echocardiogram showed normal cardiac function and no pericardial

BGA

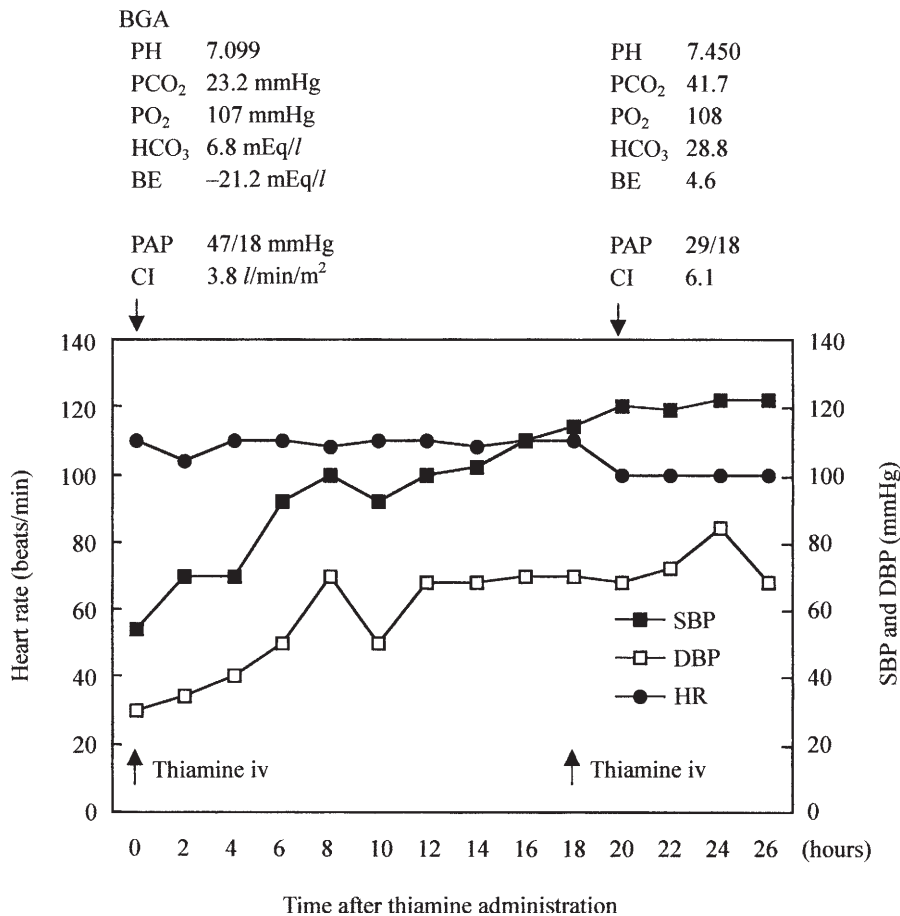|				
PH	7.099		PH	7.450
PCO_2	23.2 mmHg		PCO_2	41.7
PO_2	107 mmHg		PO_2	108
HCO_3	6.8 mEq/l		HCO_3	28.8
BE	−21.2 mEq/l		BE	4.6
PAP	47/18 mmHg		PAP	29/18
CI	3.8 l/min/m^2		CI	6.1

Figure 3. Time course of blood pressure, heart rate, arterial blood gas analytic data, pulmonary artery pressure, and cardiac index. BGA: arterial blood gas analysis, BE: base excess, PAP: pulmonary artery pressure, CI: cardiac index, Thiamine iv: thiamine intravenous infusion.

effusion (Fig. 6). CAG, LVG and cardiac catheterization were performed again, and we tried a spasm provocation test on March 15. However, coronary artery spasm was not induced. LVG showed normal LV contraction (LVEF; 66%) (Fig. 6), right-sided and left-sided cardiac catheterization data were normal (mRAP of 4 mmHg, RVP of 23/3 mmHg; the PAP was 25/7 mmHg with a mean pressure of 14 mmHg; PCWP was 6 mmHg; LVP was 104/0 mmHg with a end-diastolic pressure of 7 mmHg, and AoP 104/60 was mmHg), and cardiac output was 5.6 l/min (cardiac index; 3.3 l/min/m^2).

Discussion

Thiamine deficiency can induce high-output cardiac failure due to the accumulation of pyruvate and lactate, leading to intensive vasodilatation (1). Hypokinesia and/or dilatation of the left ventricle (due to thiamine deficiency) are sometimes noted in Shoshin beriberi (9). The present case had cardiac failure although his cardiac output was within the normal range at shock state. Clinical improvement was rapid after intravenous infusion of thiamine and we confirmed thiamine deficiency by a blood test. Thus, we diagnosed the patient as Shoshin beriberi.

While some studies have failed to demonstrate any characteristic ECG changes in patients with beriberi (10), others have reported the presence of biphasic, or inverted T-waves, low-voltage ventricular complexes, prolongation of the Q-T interval, abnormal PR interval (long and short), and sinus tachycardia (11, 12). In most of these reports, ECG abnormalities normalized after thiamine administration (10, 13, 14).

This is the first case report to demonstrate ST-segment elevation and myocardial damage without coronary artery stenosis in a case of human Shoshin beriberi. Read et al (8) reported that three of eleven dogs with thiamine deficiency experienced sudden death. Further, one of these animals was noted to have severe ECG abnormalities, including ST-segment elevation and tall or deeply inverted T waves, prior to death. These observations, combined with the present

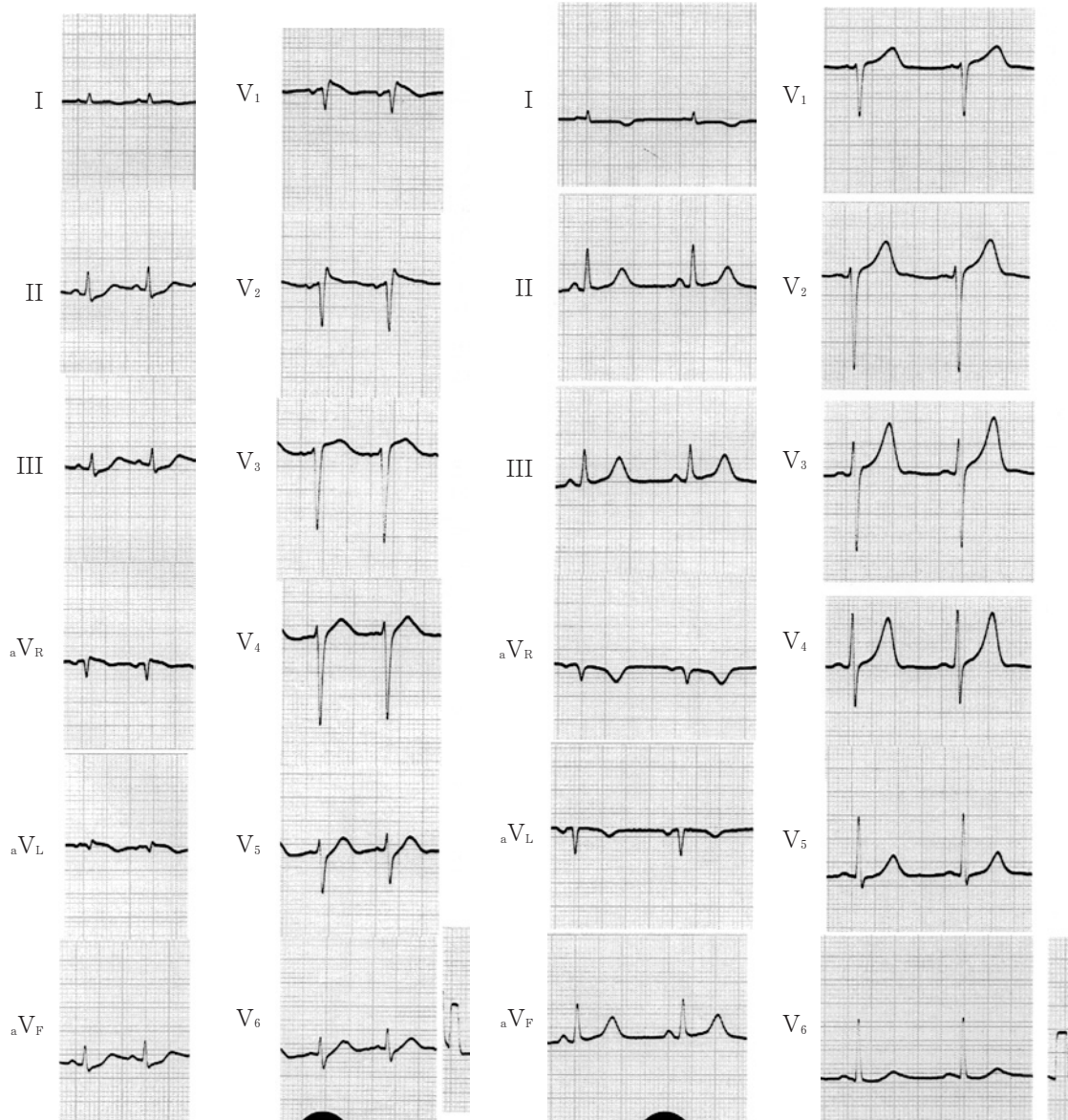1995. 1. 29   1995. 3. 7

Figure 4. Time course of ECG changes. In ECG, ST elevation in leads V₄ to V₆ disappeared, coved type ST-segment elevation in leads V₁ and V₂, and ST-segment depression in leads II, III, and ₐV_F on January 29, 1995. The ST-segment elevation in V₁ to V₃ leads resolved, and inverted T waves developed in I and ₐV_F on March 7, 1995.

case, suggest that ST-segment elevation may occur with severe forms of beriberi heart disease. However, its mechanism is unknown.

In the present case, there was no significant stenosis in the coronary artery during ST segment elevation. Acute myocardial infarction in patients with normal coronary arteries may occur via transient occlusion of coronary vessel(s) by spasm and/or thrombus (15). Indeed, a previous case report described a case of vasospastic angina in a patient with Shoshin beriberi (16). In the present case, spasm of coronary arteries

was not induced by provocation test. Thus, myocardial damage in the present case was not likely the result of coronary artery disease. However, hypotension and secondary global coronary hypoperfusion may have played a role in the subsequent cardiac dysfunction and myocardial damage, and thiamine therapy may improve them and result in the normalization of ECG changes.

There has been no report on the direct effect of thiamine deficiency on myocardial change related to ST elevation. Myocardial energy depletion may induce myocardial damage
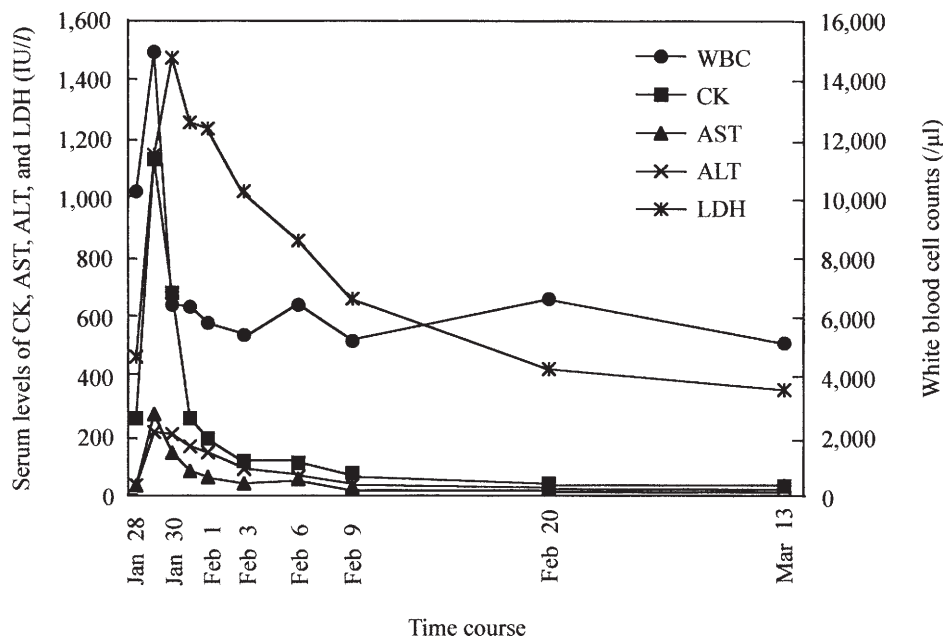
Figure 5. Time course of laboratory data.

with ST elevation because thiamine deficiency impairs myocardial energy metabolism. Moreover, various studies have suggested that activation of sarcolemmal adenosine triphosphate (ATP)-sensitive potassium (KATP) channels by ischemic ATP depletion may result in ST-segment elevation (17–19). Thiamine deficiency also induces ATP depletion (20). Thus, activation of sarcolemmal KATP channels by ATP depletion induced by thiamine deficiency may contribute to ST-segment elevation in severe beriberi.

The present case had another interesting ECG change. It is a coved type ST segment elevation in V$_1$ and V$_2$ with incomplete right bundle branch block pattern during the time course of ECG change. The ECG change is similar to that which can be detected in Brugada syndrome. Wilde et al (21) mentioned that thiamine deficiency can lead to ST-segment elevation in the right precordial leads. Previous studies have demonstrated that various central and autonomic nervous system abnormalities can result in ST-segment elevation with or without CK elevation (22–27). The autonomic nervous function also participates in the formation of Brugada-type ECG (28,29), and unbalanced autonomic nerve function has been thought to play an important role in inducing Brugada-type ECG (30). The present case also experienced symptoms of severe neuropathy secondary to thiamine deficiency. Thus, another potential explanation for the ST-segment elevation is dysfunction of the autonomic nervous system. However, regression of R wave in precordial leads or clockwise rotation was also seen in ECG at the acute phase in the present case. This finding is different from the typical ECG change in Brudaga syndrome, and it may indicate transient myocardial damage due to thiamine deficiency

as well as right ventricular dilatation.

In summary, the present report describes ST-segment elevation and myocardial damage in a patient with Shoshin beriberi although the precise mechanism is not known. The possibility of Shoshin beriberi should be considered as part of a differential diagnosis in patients with shock and ST-segment elevation.

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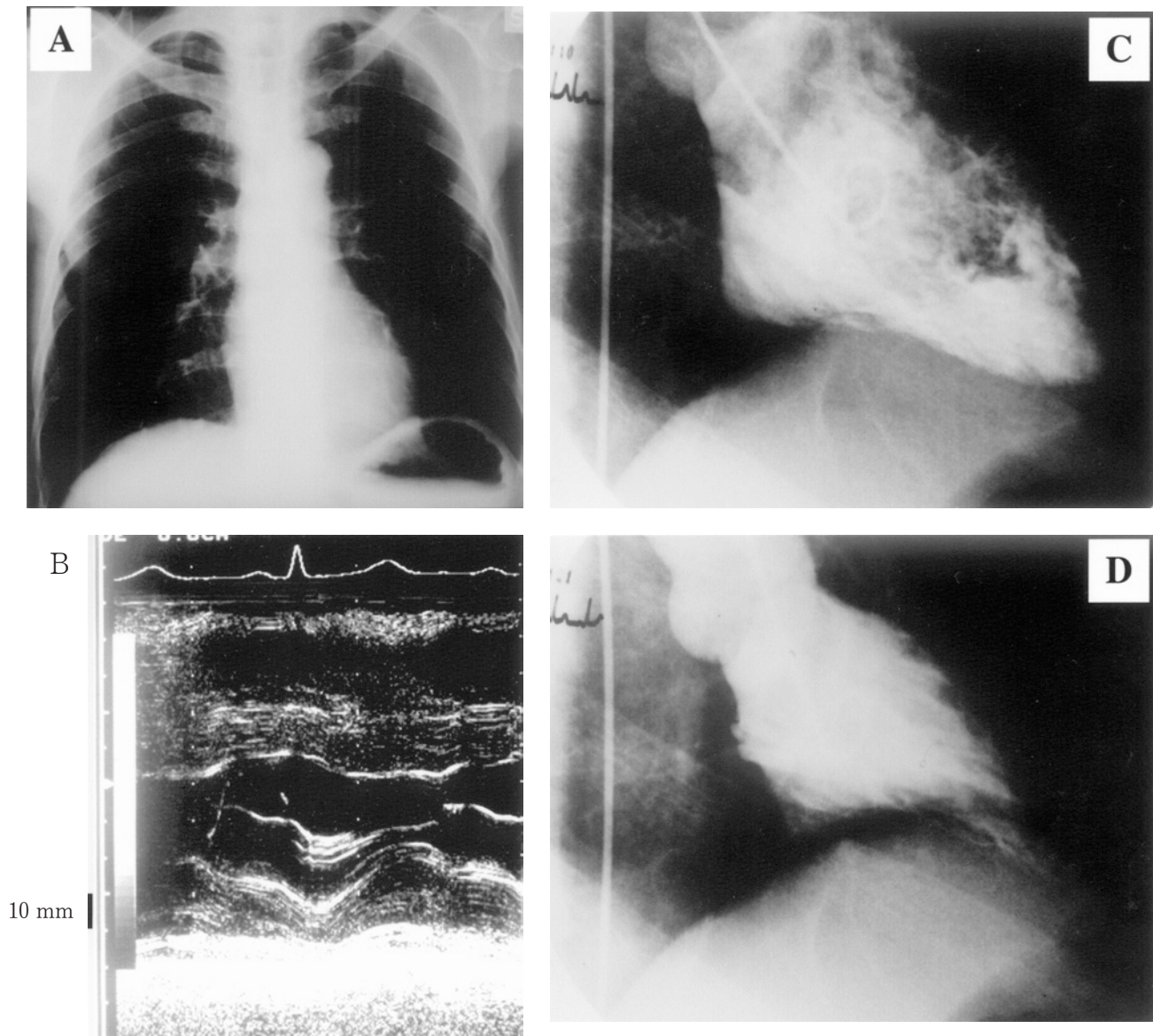
Figure 6. Chest X-ray, echocardiogram, and left ventriculogram on March 15, 1995.

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