Shoshin Beriberi With Vasospastic Angina Pectoris
—— Possible Mechanism of Mid-Ventricular Obstruction ——

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A 73-year-old heavy drinker was admitted to hospital in a state of shock. He had been suffering from frequent angina at rest, causing him to drink more heavily in an effort to overcome his anginal chest pain. He had been drinking hard each day and had not eaten for 4 weeks. His hemodynamic state on admission showed high-output heart failure. Echocardiography revealed hyperkinesis of the left ventricle and mid-ventricular obstruction with peak intraventricular gradients of 30 mmHg. Although no improvement was seen despite administering the maximal dose in catecholamine therapy, his condition improved rapidly after vitamin B1 was administered. Cardiac catheterization revealed mid-ventricular obstruction with an apical aneurysm. Coronary artery spasm was induced by injecting acetylcholine in the distal site of the left anterior descending artery, which perfused the area of the apical aneurysm. In the present case, both left ventricular hyperkinesis caused by shoshin beriberi and apical myocardial infarction caused by frequent coronary spasms produced mid-ventricular obstruction with an apical aneurysm. (Circ J 2002; 66: 1070–1072)

Key Words: Mid-ventricular obstruction; Shoshin beriberi; Vasospastic angina pectoris

Beriberi heart is a very rare disease today and is caused by thiamin deficiency. Beriberi heart is hemodynamically characterized by high cardiac output failure associated with arteriolar vasodilatation. Shoshin beriberi is a fulminating form of beriberi heart and is characterized by hypotension, tachycardia, and lactic acidosis. Initially, mid-ventricular obstruction had been reported as a special form of hypertrophic cardiomyopathy, but it can be found in other conditions such as myocardial infarction, hypertensive heart disease, and even in normal subjects with alteration of loading conditions. We report a case of shoshin beriberi and mid-ventricular obstruction of the left ventricle with an apical aneurysm. The patient’s left ventricular wall thickness was not hypertrophic, and the aneurysmal site was perfused by the left anterior descending artery, in which spasm was provoked using acetylcholine.

Case Report

A 73-year-old man was admitted to hospital suffering from chest pain and adynamia of the limbs. He had a history of inferior myocardial infarction, which was followed by conventional therapy when he was aged 65 years. Thereafter, he frequently experienced angina at rest, especially at night. He was a heavy drinker from his young days and he drank to overcome his anginal chest pain instead of taking medication. His alcohol intake had increased gradually, and he had stopped eating completely and been drinking all day for 1 month prior to admission. His blood pressure on admission was 68/30 mmHg and he was in a state of shock. His first and second heart sounds were normal and, although hepatomegaly was present, bilateral pitting edema was not. The power of his inferior limbs was very weak and his deep tendon reflex had disappeared.

An electrocardiogram showed sinus tachycardia with a heart rate of 120 beats/min, and ST depression in leads I, II, III, aVF, and V1–V6, as well as negative T waves in leads II, III, and V1–V3. Echocardiography demonstrated hyperkinesis of the left ventricle and mid-ventricular obstruction with a peak intraventricular pressure gradient of 30 mmHg, but his left ventricular wall thickness was not hypertrophic. Laboratory data were normal except for a slight increase in creatine kinase (CK) (223 IU/L). After administering 3 L/min of oxygen, arterial blood gases revealed a pH of 7.27, pO2: 105 mmHg, and pCO2: 17 mmHg.

Despite massive intravenous administration of catecholamine (dopamine 20 lg·min–1·kg–1, dobutamine 20 lg·min–1·kg–1, and noradrenaline 0.2 lg·min–1·kg–1), his blood pressure did not increase. Despite his state of shock, cardiac output was increased markedly and systemic vascular resistance was extraordinarily low. From his hemodynamic data (Table 1), shoshin beriberi was suspected and 100 mg of thiamin was administered intravenously, resulting in the patient’s condition improving quickly (Table 1); however, peripheral blood concentration of vitamin B1, which was obtained after intravenous administration of 10 mg of thiamin, was still low (16 ng/ml). From day 3, the intraventricular pressure gradient could not be detected by echocardiography.

Myocardial perfusion imaging with thallium-201 demonstrated that both the inferior wall and apical areas were damaged. Cardiac catheterization on day 33 revealed a total occlusion in the distal segment of the left circumflex artery with good collateral flow from the right coronary artery. Left ventriculography demonstrated mid-ventricular...
Shoshin beriberi has been reported previously, 1,4,14,15 and cardia, and lactic acidosis.1,4 Left ventricular function of heart disease and is characterized by hypotension, tachycardia, and by his marked improvement after thiamin administration because of the patient's decreased blood thiamin concentration.

Holism. Beriberi heart was diagnosed with certainty on the basis of the high-output heart failure, low systemic vascular resistance, lactic acidosis, and a history of alcoholism. The classic beriberi heart shows high-output heart failure and is characterized hemodynamically by low vascular resistance and a hyperkinetic circulatory state.1,2 In the present case, left ventricular wall thickness was not hyperkinetic. In the present case, left ventricular hypertrophy, but also physiologic changes such as left ventricular hypertrophy, and this phenomenon may occur in patients undergoing dobutamine stress echocardiography and even in subjects with normal left ventricles with loading conditions manipulated by the Valsalva maneuver after the administration of amyl nitrate.11,16 These findings suggest that there are several mechanisms that underlie each case of mid-ventricular obstruction; not only pathologic changes such as left ventricular hypertrophy, but also physiologic changes such as the hyperdynamic state and reduction both in preload and afterload.

It is known that an apical aneurysm sometimes coexists with mid-ventricular obstruction as a special form of hypertrophic cardiomyopathy, and this phenomenon may be explained by 2 theories:10 (1) that mid-ventricular obstruction is a result of apical infarction in patients with hypertrophic cardiomyopathy, and (2) that mid-ventricular obstruction with high left ventricular apical pressures leads to infarction and apical aneurysm. In the present case, the patient's left ventricular wall thickness was not hypertrophic but his heart showed a hyperdynamic state, which must have been caused by shoshin beriberi. Coronary artery spasms occurred frequently in the distal site of the left anterior descending artery and these events led to infarction of the apical myocardium. In the present case, the extremely rare combination of a hyperkinetic left ventricular obstruction with an apical aneurysm (Fig.1). Pressure gradient in the left ventricle could not be evaluated because of the inability to insert a catheter without frequent ventricular premature beats. A spasm provocation test was performed with an intracoronary injection of acetylcholine to determine the cause of his chest pain. After injecting acetylcholine into the left coronary artery, the patient still complained of the same pain as he had previously felt frequently, and ST elevation was observed in leads V2–V4. Coronary angiography showed a total occlusion at the distal site of the left anterior descending artery (Fig 2). The perfused area of the occluded coronary artery coincided with the area of the apical aneurysm.

**Discussion**

The classic beriberi heart shows high-output heart failure and is characterized hemodynamically by low vascular resistance and a hyperkinetic circulatory state.1,2 In the present case, the diagnosis of beriberi heart was suspected on the basis of the high-output heart failure, low systemic vascular resistance, lactic acidosis, and a history of alcoholism. Beriberi heart was diagnosed with certainty because of the patient's decreased blood thiamin concentration and by his marked improvement after thiamin administration. Shoshin beriberi is the fulminant form of beriberi heart disease and is characterized by hypotension, tachycardia, and lactic acidosis.1,4 Left ventricular function of shoshin beriberi has been reported previously1,4,14,15 and most reports have shown that left ventricular wall motion becomes hyperkinetic. In the present case, left ventricular wall motion was hyperkinetic upon admission.

Mid-ventricular obstruction was first reported by Falicov et al as a rare manifestation of hypertrophic cardiomyopathy...
tricle caused by beriberi and an apical infarction caused by frequent coronary spasms probably produced the shape of a mid-ventricular obstruction with an apical aneurysm without any cardiac hypertrophy. Because an intraventricular pressure gradient could be detected only at the time of admission, this finding suggests that the degree of obstruction had reduced by those physiologic changes that occurred with the improvement in the patient's hyperdynamic state.

In summary, the present report describes a patient with shoshin beriberi and mid-ventricular obstruction with an apical aneurysm of the left ventricle, in which the spasm site coincided with the apical aneurysm. In the present case, hyperkinetic wall motion of the left ventricle and frequent coronary artery spasms may be the etiology of a mid-ventricular obstruction with an apical aneurysm.

References