The Journal of Emergency Medicine, Vol. 41, No. 4, pp. e73–e77, 2011

0736-4679/$–see front matter

ELSEVIER

doi:10.1016/j.jemermed.2008.03.040

Clinical Communications: Adults

SHOSHIN BERIBERI MIMICKING A HIGH-RISK NON-ST-SEGMENT ELEVATION ACUTE CORONARY SYNDROME WITH CARDIOGENIC SHOCK: WHEN THE ARTERIES ARE NOT GUILTY

Pablo Loma-Osorio, MD,* Pablo Peñafiel, MD,* Ada Doltra, MD,* Alessandro Sionis, MD,* and Xavier Bosch, MD, PHD, FESC*†‡

*Unidad Coronaria, Servicio de Cardiología, Hospital Clínic, Barcelona, Spain, †Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain, and ‡Department of Medicine, University of Barcelona, Barcelona, Spain
Reprint Address: Xavier Bosch, MD, PHD, FESC, Unidad Coronaria, Servicio de Cardiología, Hospital Clínic, Villarroel 170, 08036 Barcelona, Spain; e-mail: xbosch@clinic.ub.es

□ **Abstract—Background: Cardiac acute beriberi (Shoshin syndrome) is a rare disease that may lead to a fatal outcome if not treated specifically. Objectives: We report a case of Shoshin syndrome with an unusual presentation of cardiogenic shock and an electrocardiographic pattern of severe myocardial ischemia suggesting left main coronary artery obstruction. Case Report: A 35-year-old man presented with chest discomfort, diffuse ST-segment depression in the 12-lead electrocardiogram (ECG) with ST-segment elevation in aVR, and rapidly evolving congestive heart failure leading to cardiogenic shock. Intensive support was required, including mechanical ventilation, high doses of inotropics and vasopressors, intra-aortic balloon counterpulsation, and continuous renal replacement therapy. An emergency coronary angiogram was performed that showed normal coronary arteries. Right heart catheterization showed a high-output state with elevated filling pressures suggesting high-output heart failure. The echocardiography confirmed normal left and right ventricular contraction. Thiamine deficiency was suspected as the cause of the high-output heart failure. After a single dose of intravenous thiamine (100 mg), the patient's hemodynamic status improved dramatically within minutes, allowing a rapid discontinuation of hemodynamic support. Subsequent ECGs showed complete resolution of ST-segment abnormalities. Serial lactate measurements, red blood cell transketolase activity, and the thiamine pyrophosphate response test were concordant with a thiamine deficiency state. Conclusion: Shoshin syndrome may present as cardiogenic shock with an ECG mimicking severe myocardial ischemia, and if suspected, can be rapidly and effectively treated. © 2011 Elsevier Inc.**

□ **Keywords—cardiogenic shock; Shoshin syndrome; cardiac beriberi; thiamine deficiency; electrocardiography; heart failure**

INTRODUCTION

Thiamine (vitamin B1) is a coenzyme for decarboxilation reactions, present in a wide range of foods, with the exception of fats, oils, and refined sugars. Because it is needed for glucydic metabolism, its requirements are increased in carbohydrate-rich diets. Severe chronic alcoholism may lead to an abnormal absorption and storage of thiamine, as well as an increase in its destruction, thus making it the main cause of thiamine deficiency in Western countries (1). Two distinct clinical pictures have been described related to severe thiamine deficiency, depending on clinical presentation. If the main symptoms are neurological (peripheral neuropathy), it is called "dry beriberi," in contrast to "wet beriberi," whose main symptoms involve the cardiovascular system. Shoshin beriberi is a potentially fatal form of wet beriberi, pre-

RECEIVED: 5 November 2007; FINAL SUBMISSION RECEIVED: 18 January 2008;
ACCEPTED: 17 March 2008

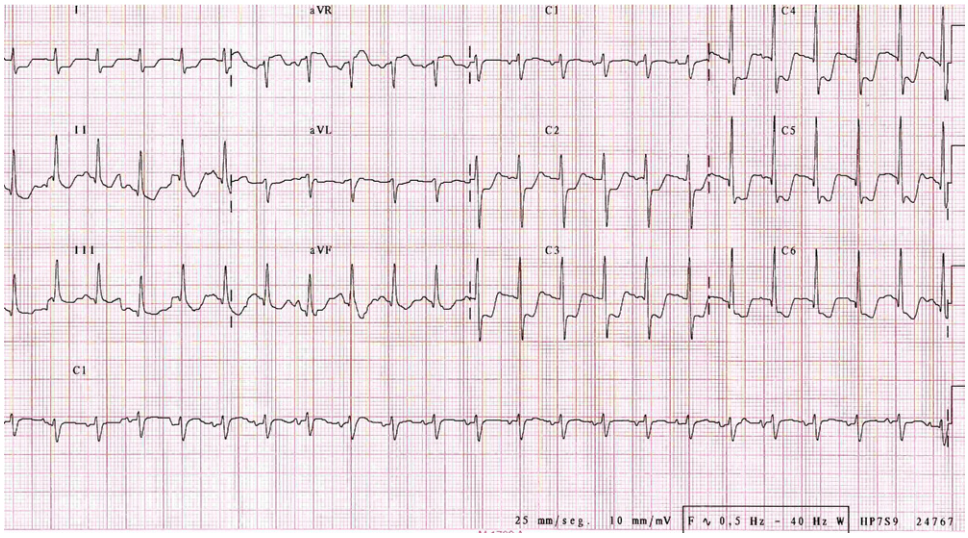

Figure 1. Electrocardiogram on admission. Diffuse ST-segment descent involving 9 leads. Note the ST-segment elevation in aVR.

senting with hypotension, tachycardia, and lactic acidosis, rapidly progressing to death if left untreated (2). Although relatively common in Asia, it is rare and often under-diagnosed in Western countries (3).

Patients with acute coronary syndromes should be rapidly diagnosed, risk-stratified, and treated. A rapid diagnosis is critical to guiding early intervention and appropriate management in these patients. High-risk patients, characterized by their clinical, electrocardiographic, and hemodynamic characteristics, should have early coronary angiography and revascularization (4).

We present a rare case of Shoshin syndrome mimicking a high-risk non-ST-segment elevation acute coronary syndrome (NSTEACS) with cardiogenic shock. To our knowledge, this association has not been previously reported and may have important clinical implications.

CASE REPORT

A 35-year-old obese man was admitted to the Cardiac Intensive Care Unit of our institution with the diagnosis of NSTEACS and cardiogenic shock. He previously presented to the Emergency Department (ED) of a primary care center complaining of chest discomfort and progressive dyspnea. On admission, his main physical findings were a temperature of 36.5°C, regular tachycardia (heart rate 135 beats/min), hypotension (blood pressure 69/35 mm Hg), high jugular venous pressure, wet crackles in both lungs, and tachypnea (respiratory rate 35 breaths/min).

An electrocardiogram (ECG) was recorded, showing sinus tachycardia, ST-segment depression in leads I, II, III, aVF, and V2–V6, and ST-segment elevation in aVR (Figure 1). Due to severe respiratory distress, intubation and mechanical ventilation were required. Despite the administration of high-dose vasoactive drugs (dopamine 25 μg/kg/min, dobutamine 20 μg/kg/min, and norepinephrine 0.4 μg/kg/min), hypotension and hypoperfusion persisted. With the clinical suspicion of cardiogenic shock secondary to a high-risk NSTEACS with left main coronary artery obstruction, the patient was transferred to our institution for emergent coronary angiography.

On admission, marked hemodynamic instability, anuria, and persistent manifestations of heart failure were noted. The laboratory investigation showed a pronounced lactic acidosis with pH 7.18, pCO_2 27 mm Hg, lactate 9.71 mmol/L (normal 0.63–2.44 mmol/L), base excess −20 mmol/L, an anion gap of 26 mmol/L, and hyperamylasemia (1849 UI/dL). Troponin I was 0.7 ng/dL (normal $<$ 0.04). The patient was directly transferred to the catheterization laboratory, where an intra-aortic balloon pump was inserted. An urgent coronary angiogram was performed, which showed no significant lesions in the epicardial coronary arteries. The left ventricular end-diastolic pressure was 40 mm Hg. A left ventriculogram was performed, showing no wall motion abnormalities and a preserved left ventricular ejection fraction.

The patient was transferred to the Cardiac Intensive Care Unit and continuous veno-venous hemofiltration was started. Transthoracic and transesophageal echocardiography were performed, ruling out mechanical complications and showing hyperdynamic motility of both ventricles. Heart valves were normal. Right heart catheterization showed a pulmonary artery pressure of 58/30