CASE REPORT

Complete recovery from severe myocardial dysfunction in a patient with anorexia nervosa

Takeru Ono (MD), Shunji Kasaoka (MD, PhD, FJCC), Motoki Fujita (MD)*, Susumu Yamashita (MD, PhD), Kazumi Kumagai (MD), Kotaro Kaneda (MD), Ryosuke Tsuruta (MD, PhD), Tsuyoshi Maekawa (MD, PhD)

Advanced Medical Emergency and Critical Care Center, Yamaguchi University Hospital, Japan

Received 2 December 2008; received in revised form 9 February 2009; accepted 24 February 2009
Available online 9 April 2009

KEYWORDS
Anorexia nervosa;
Hypoglycemia;
Cardiogenic shock;
Percutaneous cardiopulmonary support

Summary
This report describes a patient who experienced cardiopulmonary arrest caused by severe hypoglycemia and malnutrition, which was successfully treated with percutaneous cardiopulmonary support (PCPS) and intra-aortic balloon pumping (IABP).

A 33-year-old female with anorexia nervosa (AN) was transferred to the emergency center because of a loss of consciousness. On admission, she was extremely emaciated, hypotensive, and hypoglycemic (10 mg/dl). A chest X-ray showed butterfly shadow. Echocardiography showed severe hypokinesis of left ventricular wall motion. On the 3rd hospital day, she experienced cardiac arrest. Myocardial dysfunction caused by malnutrition was suspected, and therefore both PCPS and IABP were administered for circulatory support and myocardial protection. Thereafter, cardiac function gradually recovered and she was later weaned from PCPS and IABP on the 9th and the 10th hospital day, respectively. She was discharged from the intensive care unit on the 43rd hospital day with normal cardiac function. Her neurological outcome after 6 months as evaluated by the Glasgow Outcome Scale was considered to be good recovery.

Cardiomyopathy in AN patients is reversible ventricular dysfunction, and circulation assisting devices are considered for the treatment of cardiogenic shock.

Introduction

Anorexia nervosa (AN) is an eating disorder with a significant risk of sudden death due to severe cardiac complications [1,2]. In AN patients, car-
diovascular abnormalities such as bradycardia, hypotension, mitral valve prolapse, and arrhythmias due to hypokalemia or hypomagnesaemia are often found [3]. However, the left ventricular function is generally normal [4].

This report describes a case of a temporary and reversible myocardial dysfunction with cardiopulmonary arrest (CPA) caused by severe hypoglycemia and malnutrition, which was successfully treated using percutaneous cardiopulmonary support (PCPS) and intra-aortic balloon pumping (IABP).

Case report

A 33-year-old female, who had been suffering from AN for several years, was transferred to our emergency center because of a loss of consciousness. On admission, her vital signs were Glasgow Coma Scale 5 (E1 V1 M3), systolic blood pressure of 70 mmHg, heart rate of 70 beats/min, body temperature of 33.8°C, and respiratory rate of 20 breaths/min. Her height, body weight, and body mass index were 150 cm, 20 kg, and 8.9, respectively. Laboratory data showed hypoglycemia (blood glucose, 10 mg/dl), hypoproteinemia (total protein, 5.0 g/dl; albumin, 2.9 g/dl), an elevation of hepatic enzymes (aspartate aminotransferase, 1875 IU/l; alanine aminotransferase, 3063 IU/l; lactate dehydrogenase, 782 IU/l), and bleeding tendency (prothrombin time-international normalized ratio, 2.88). Creatine kinase was elevated mildly (156 IU/l), but troponin T-test was negative. Free triiodothyronine level was 1.3 pg/ml and free thyroxine level was 1.3 ng/dl. Chest X-ray (Fig. 1) showed butterfly shadow. An electrocardiogram (ECG) demonstrated abnormal Q waves in V3, V4, and V5 (Fig. 2A). An echocardiogram showed severe left ventricular dysfunction with an ejection fraction (EF) of 10% (Fig. 2C). The head computed tomography (CT) scan showed no abnormal lesion such as hemorrhage or stroke.

She was diagnosed to have cardiogenic shock and was therefore intubated and mechanically ventilated, because she did not regain consciousness in spite of treatment for hypoglycemia. Low-dose dobutamine (2 μg/(kg min)) was administered to improve the low EF. To prevent refeeding syndrome, potassium phosphate was administered and her calorie intake was a maximum of 480 kcal/day until the 5th hospital day and thereafter it was increased gradually.

Her clinical course in the intensive care unit (ICU) is shown in Fig. 3. On the 3rd hospital day, she abruptly developed CPA. Bradycardia (heart rate, 45 beats/min) was shown on the ECG monitor. Cardiopulmonary resuscitation with the use of epinephrine was immediately performed, but spontaneous circulation could not be established. An echocardiogram revealed almost total akinesis of the whole left ventricular wall. Therefore, PCPS and IABP were induced and maintained. The continuous administration of dobutamine was discontinued for myocardial protection. To protect the patient’s neurological function, her deep body temperature was maintained at less than 37.0°C. After the start of PCPS and IABP, her hemodynamics gradually improved and the systolic blood pressure exceeded more than 90 mmHg. Her cardiac function thereafter recovered gradually, and the EF recovered to 25% on the 9th hospital day (Fig. 3). She was thereafter weaned from PCPS on the 9th hospital day and weaned from IABP on the 10th hospital day. She was extubated on the 15th hospital day, because she regained consciousness. She was discharged from the ICU on the 43rd hospital day with normalized ECG (Fig. 2B) and an improved EF (Fig. 2D). After discharge from the ICU, she had no cardiac events and was discharged from the hospital on the 78th hospital day. Her neurological outcome after 6 months, as evaluated by the Glasgow Outcome Scale, was good recovery.

Discussion

AN is a psychiatric disease with a high mortality and it is often associated with cardiovascular
abnormalities including sudden death [1,2]. The sudden death is often caused by arrhythmias and acute heart failure [1,2]. Malnutrition causes cardiac abnormalities on a cellular level. This leads to diminished protein synthesis, the activation of calcium-dependent proteinases, mitochondrial swelling, a decreased glycogen content, interstitial edema, and myofibrillar atrophy [5,6]. These
Complete recovery from severe myocardial dysfunction

Figure 3  Clinical course of the patient. The changes in the systolic blood pressure (sBP) and left ventricular ejection fraction (LVEF) are shown. On admission, her LVEF was 10%. On the 3rd hospital day, cardiac arrest suddenly occurred, and percutaneous cardiopulmonary support (PCPS) and intra-aortic balloon pumping (IABP) were induced. Thereafter, cardiac function gradually recovered and PCPS and IABP were weaned off on the 9th and the 10th hospital days, respectively. DOB, dobutamine.

conditions cause a decrease of the contractile force of the ventricle and diastolic compliance [7].

Hypoglycemic state induces a high blood level of catecholamine, because the state stimulates the central sympathetic nervous system. An excessive release of catecholamines may cause myocardial damage. A catecholamine surge might cause reversible ventricular dysfunction in patients with subarachnoid hemorrhage, endocrine disorder, and hypoglycemic stress [8–10]. Catecholamine-mediated myocardial injury is thought to be caused by an elevation of intracellular calcium and possibly oxygen free radicals [11]. Histologically, the myocardium shows contraction band necrosis and myocytolysis [11]. Ohwada et al. reported a case of ampulla cardiomyopathy in three young female patients with AN [10]. They suggested that excess catecholamine release, possibly induced by hypoglycemia, was the cause of ampulla cardiomyopathy. In the present case, excessive catecholamine release due to persistent hypoglycemia might have caused severe myocardial dysfunction, although ampulla cardiomyopathy could not be diagnosed because coronary angiography was not performed.

In the present case, an echocardiogram on admission showed cardiac wall thinning (Fig. 2C), that might have been caused by AN. It was therefore suspected that her heart might be sensitive to catecholamine. Excessive catecholamine released by hypoglycemia might not improve contractile force of the left ventricle but exacerbate cardiac function by increase of myocardial oxygen consumption or vasoconstriction of coronary artery. Thereafter, severe myocardial dysfunction occurred in the present case. Furthermore, the administration of dobutamine (2 \( \mu g/(kg\ min) \)) might accelerate the myocardial dysfunction and thereby induce cardiac arrest.

We considered her myocardial state caused by a catecholamine surge to be temporary and reversible, and a full recovery of the cardiac function was expected following myocardial protective treatment. In addition, myocardial viability was expected because mild elevation of cardiac enzymes was observed including creatinine kinase and negative troponin T. Therefore, when the patient went into cardiac arrest, PCPS and IABP were immediately started. Eventually, her heart recovered and thereafter demonstrated a normal function as expected.

The complete recovery of cardiac function suggested that severe myocardial dysfunction with AN might be reversible, so the use of PCPS should be considered to maintain the systemic circulation.

References


