CASE REPORT
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Died immediately after corrective surgery for right ventricular acute myocardial infarction and ventricular septal rupture

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Postinfarction ventricular septal rupture (VSR) is a rare but fatal complication of acute myocardial infarction. In many cases, postinfarction VSR leads to hemodynamic instability and urgent surgical treatment is necessary. Here we describe a case of a patient with right ventricular (RV) dysfunction caused by acute RV infarction and with cardiogenic shock, whose condition improved after development of postinfarction VSR, but the patient died after corrective surgery.

Keywords: Myocardial infarction; Ventricular septal rupture; Right ventricular dysfunction

INTRODUCTION

Postinfarction ventricular septal rupture (VSR) is a rare but fatal complication of acute myocardial infarction (AMI). Because of the poor outcome of medical treatment [1] and the possibility of hemodynamic instability, most postinfarction VSR cases are treated surgically [2]. In patients with right ventricular (RV) dysfunction, this condition is relevant to postoperative mortality [3-5]. Here we describe a rare case of a patient with cardiogenic shock caused by RV infarction, whose condition improved after development of postinfarction VSR, but the patient died after corrective surgery.

CASE

A 70-year-old man without any known underlying disease visited the local medical center's emergency department because of chest pain for 2 hours' duration. An initial electrocardiogram (ECG) showed normal sinus rhythm with ST elevation as well as Q wave in leads II, III, and aVF (Fig. 1A). A reverse ECG showed ST elevation in leads V1R to V4R (Fig. 1B). Soon afterward, the patient lost consciousness and ECG showed ventricular fibrillation. Defibrillation and cardio-

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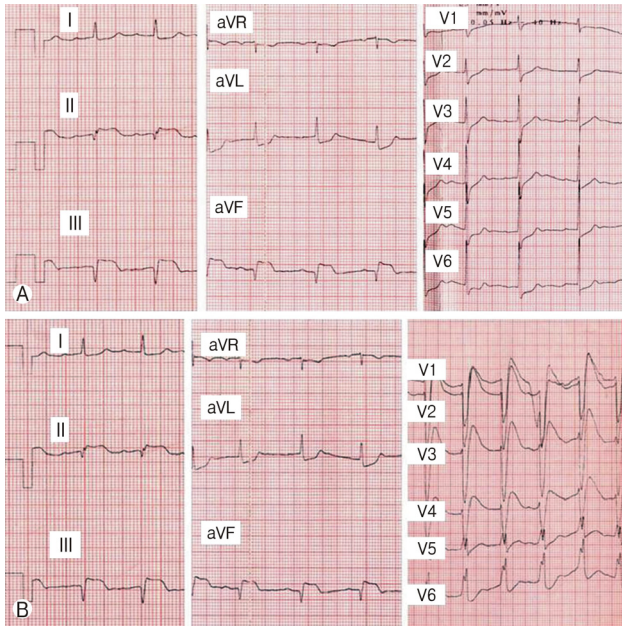

Fig. 1. Initial electrocardiogram (ECG) at a local medical center. (A) ECG showed ST elevation as well as Q wave in leads II, III, aVF. (B) Reverse ECG showed ST elevation in leads V1R to V4R.

pulmonary resuscitation were performed, and the patient's cardiac rhythm returned to normal sinus rhythm. However, the patient went into shock. He was intubated and then transferred to our emergency department.

The patient was in a drowsy mental state, and his blood pressure (BP) was 80/60 mm Hg. Heart sounds was regular, and cardiac murmur was not audible. Neck vein distension was observed. Cardiac troponin I level was 0.35 ng/mL (reference range<0.4 ng/dL), and white blood cell count was 11,400/μL. Arterial blood gas analysis showed metabolic acidosis (pH 7.235) with hypoxemia (pO_2 67.0 mm Hg). There were no significant differences of ST elevation and Q wave at leads II, III, and aVF on ECG. Acute right coronary artery (RCA) territory myocardial infarction was suspected, and primary percutaneous intervention was planned. However, because of the patient's age, drowsy mental state, and poor economic status, his family declined coronary intervention, and he was transferred to the local medical center.

Soon after the patient's arrival to the local hospital, his condition improved dramatically without specific treatment or intervention. His mental state became clear, and vital signs were stabilized. The patient was transferred to our hospital again one day afterward for evaluation and further treatment of myocardial infarction. Physical examination revealed a BP of 110/80 mm Hg with administration of dopamin 10 μg/kg/min. Neck vein distension was less than it was during the previous examination. In contrast to the previous examination, grade 3 pansystolic murmur was checked at the left sternal border. Echocardiogram revealed preserved left ventricular (LV) function (LV ejection fraction 50%) with regional wall motion abnormality at the base to the mid inferior wall of the left ventricle. The right ventricle was enlarged with severe global hypokinesia of the RV free wall, suggestive of RV infarction. A large VSR (1.7 cm) with continuous shunt flow from the left ventricle to the right ventricle directed toward the RV outflow tract was observed at the basal portion of the interventricular septum (Fig. 2). Qp/Qs ratio was estimated at 2.7. Moderate pulmonary hypertension (RV systolic pressure 58 mm Hg) with severe tricuspid regurgitation was noted.

Coronary angiography and cardiac catheterization were performed 7 days after admission. Discrete concentric stenosis with thrombolysis in myocardial infarction grade 3 distal flow in the middle portion of the RCA was noted, and balloon angioplasty with stent insertion was performed (Fig. 3). Oxygen step-up was observed at the right ventricle, and the Qp/Qs ratio was estimated at 3.5. Surgical closure was planned because of a large VSR with a hemodynamically significant left-to-right shunt after the patient's condition was stabilized.

Twenty-five days after admission, bovine pericardial patch closure of the VSR through the right ventricle and De-Vega tricuspid valve annuloplasty were performed. During surgery, a 1.7 cm VSR was identified at the inferior septum, and the right atrium and the right ventricle were dilated. After the patient was weaned from cardiopulmonary bypass, his systolic BP was maintained at 90-100 mm Hg, urine output was 60-80 mL/h, and central venous pressure (CVP) was 14 mm Hg. Intraoperative transesophageal echocardiography showed no interventricular shunt flow, and therefore surgery was co mpleted. However, a hour after surgery, the patient's BP decreased to 80/60 mm Hg, CVP was elevated up to 25 mm Hg, and urine output decreased. On physical examination, cardiac murmur was not identified, but neck vein distension was more intense than that during the pre-operative period. A non invasive cardiac output (NICO) monitor was applied, and cardiac output decreased to 2.4 L/min (reference range 4.0-8.0 L/min). A bedside echocardiogram was obtained, and there was no shunt flow, pericardial effusion, or significant

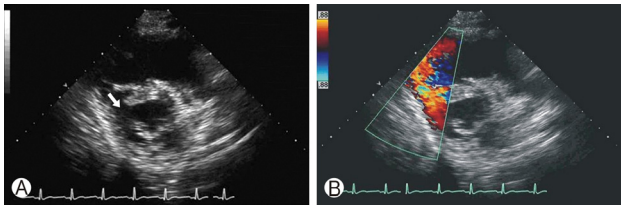

Fig. 2. Two-dimensional echocardiography. (A) Ventricular septal rupture is observed in a parasternal short axis view (white arrow). (B) Color doppler echocardiography in a parasternal short axis view reveals blood flow through a ventricular septal rupture.

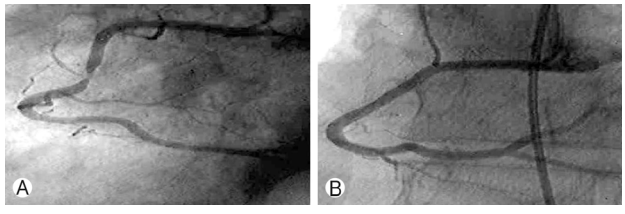

Fig. 3. Coronary angiography. (A) Discrete concentric stenosis with thrombolysis in myocardial infarction grade 3 distal flow is observed in the middle portion of the right coronary artery. (B) Balloon angioplasty with stent insertion was performed.

valvular dysfunction. However, RV dysfunction and inferior vena cava plethora were observed. Despite the use of inotropic agents, the patient's systolic BP remained at 60 mm Hg and metabolic acidosis was aggravated. Emergent surgical reopening of the VSR was planned, but the patient did not recover from shock and eventually died.

DISCUSSION

Postinfarction VSR is a rare complication of AMI. Despite thrombolysis and percutaneous coronary intervention, the incidence of postinfarction VSR ranges from 0.2% to 0.5%, and 30-day mortality is approximately 50% [1,6-8]. According to several studies, poor outcome of postinfarction VSR is related to preoperative shock and RV dysfunction [5,9-11]. When a large VSR develops, cardiac output decreases because a large amount of blood shifts from the left ventricle to the right ventricle. In the presence of RV dysfunction, overloaded RV blood cannot be delivered to the left atrium, therefore, RV dysfunction is more aggravated. Furthermore, shunt flow can cause sustained work overload of the left ventricle and result in secondary LV failure. Finally, cardiac output further decreases and shock can occur [9,12]. Therefore, urgent surgery is recommended.

In the present case, the patient's condition worsened immediately after surgical closure of VSR, and cardiac output decreased. We initially thought that surgical complications caused this, but echocardiogram revealed no interventricular shunt, pericardial effusion, and significant valvular dysfunction. Interestingly, the patient's vital signs became stable without any intervention, but he died immediately after surgical closure of VSR. Therefore, we made the careful assumption that AMI caused RV infarction and dysfunction, which led to cardiogenic shock during the initial hospital visit. VSR occurred in this patient after 2 days of medical treatment without intervention. Because of VSR with preserved LV systolic function, a large amount of left-to-right shunt occurred, and the left ventricle possibly took charge of the pump function of the right ventricle, resulting in blood flow through the pulmonary artery and blood return to the left-side chambers of the heart. As a result, the patient's BP increased, and weaning from inotropic agents became possible. In other words, the left ventricle might have functioned as a right ventricular assist device (RVAD). Although the patient's condition remained stable after RCA intervention, we thought it was necessary to perform surgical VSR closure because of the large amount of left-to-right shunt that we believed would eventually result in secondary LV failure. However, after closure of the VSR, the RVAD function of the left ventricle disappeared and the patient died from decompensated RV failure.

The current treatment of postinfarction VSR is surgery [2,13]. However, the timing of surgery is a topic of debate. Some studies have shown that delayed surgery is associated with survival, because the myocardium is too fragile immediately after the myocardial infarction [4,14]. However, delayed surgery is possible only when the patient is hemodynamically stable. In addition, the surgery should be carried out expeditiously when the patient is hemodynamically unstable [3,6,8,13,15].

In the present case, we assumed that postinfarction VSR might have functioned as a RVAD and brought hemodynamic stability. Unfortunately, we did not obtain an echocardiogram during the preoperative period. Soon after surgery for VSR closure, the patient's condition deteriorated. Based on NICO and echocardiogram findings, we assumed that decompensated RV failure led to the patient's death. As seen in this case, when delayed surgery was performed in a patient with hemodynamically stable postinfarction VSR, assessment of preoperative cardiac function can help decide the timing of surgery.

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