Delayed Repair of Ventricular Septal Rupture
Following Preoperative Awake Extracorporeal Membrane Oxygenation Support

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Outcomes of ventricular septal rupture (VSR) as a complication of acute myocardial infarction are extremely poor, with an in-hospital mortality rate of 45% in surgically treated patients and 90% in patients managed with medication. Delaying surgery for VSR is a strategy for reducing mortality. However, hemodynamic instability is the main problem with this strategy. In the present case, venoarterial extracorporeal membrane oxygenation (ECMO) was used to provide stable hemodynamic support before the delayed surgery. Awake ECMO was also used to avoiding the complications of sedatives and mechanical ventilation. Here, we describe a successful operation using awake ECMO as a bridge to surgery.

Key words: 1. Ventricular septal rupture
2. Extracorporeal membrane oxygenation
3. Awake extracorporeal membrane oxygenation
4. Myocardial infarction

Case report

A 68-year-old woman was admitted to Hallym University Sacred Heart Hospital for chest pain persisting for 2 days. An electrocardiogram showed ST-segment elevation in all the precordial leads and Q waves in leads II, III, avF, and V1–V6. Initial laboratory results showed increased levels of troponin I, creatinine kinase-myocardial band, and brain natriuretic peptide. An emergency coronary angiogram showed chronic total occlusion of the mid-right coronary artery (RCA) with collaterals and total occlusion of the mid-left anterior descending (LAD) artery. Percutaneous coronary intervention (PCI) was performed by inserting a Resolute Onyx stent (Medtronic Inc., Santa Rosa, CA, USA) into the LAD. The occluded LAD was successfully reperfused.

Three hours after PCI, the patient experienced sudden dizziness with hypotension and tachycardia. A transthoracic echocardiogram (TTE) showed a 1.78-cm ventricular septal rupture (VSR) at the lower muscular part of the ventricular septum, extensive ischemic insult in the LAD territory, and severe right and left ventricular dysfunction with an ejection fraction (EF) of 19% (Fig. 1). Cardiogenic shock was sustained despite incremental infusions of norepinephrine and intra-aortic balloon pump (IABP) support. Therefore, after 3 hours and 5 minutes of hemodynamic instability, venoarterial extracorporeal membrane oxygenation (ECMO) was administered through right
femoral vessel cannulation instead of the IABP. This was promptly followed by mechanical ventilator care and continuous renal replacement therapy. On a plain chest radiograph obtained initially after ECMO, pulmonary edema was not prominent, although the pulmonary vascular parameters increased (Fig. 2). The mean arterial pressure was maintained at more than 70 mm Hg with a continuous infusion of nor-epinephrine at a dose of 3 μg/kg/min, which was gradually decreased. Sedation was induced with a controlled dose of fentanyl (0.6 μg/kg/hr) and dexmedetomidine HCl (0.2 μg/kg/hr). After 4 days of ECMO support, extubation was performed since the patient was cooperative and had minimal airway secretions. Thereafter, awake ECMO was maintained with the addition of quetiapine (25 mg orally) before bedtime, and oral feeding was started the next day.

On the ninth day of ECMO support, the EF recovered to 27% on a repeat TTE. Therefore, surgery for post-myocardial infarction (MI) VSR was planned. The VSR was found anteriorly and 2.5 cm away from the apex. It had a rough, irregular margin with adjacent necrotic debris and muscular discoloration; it was closed by interrupted 3-0 prolene sutures using a single-layered Gore-Tex patch through left ventriculotomy, which was performed laterally along the course of the LAD. The distal RCA was bypassed using a greater saphenous vein graft. The left ventricle was repaired with 2-0 prolene sutures buttressed with Teflon felts. Cardiopulmonary bypass (CPB) was weaned according to the patient’s recovery status, and this was followed by the removal of ECMO support and the repair of femoral vessels. The duration of CPB and aortic cross-clamping was 174 minutes and 115 minutes, respectively.

The patient recovered uneventfully and was ex-
tubated on the day after the operation. Percutaneous catheter drainage (PCD) was performed on postoperative day 2 because of pleural effusion. Despite the delay due to PCD, she was discharged from the hospital on postoperative day 18 without additional complications. She has been doing well for 6 months without residual or recurrent VSR.

**Discussion**

VSR as a complication of acute myocardial infarction (AMI) is uncommon, with an incidence of 1% to 2% among patients with AMI. It typically occurs in the first week after infarction, with a mean time of 3 to 5 days from symptom onset. Previous investigations have found that age and female sex are risk factors for the development of VSR; such patients also commonly have no prior angina or MI. Angiographically, patients with VSR have been noted to have total occlusion of the infarct-related artery with minimal collaterals. Outcomes after the development of VSR are extremely poor, with an in-hospital mortality rate of 45% in surgically treated patients and 90% in patients managed with medication. Poor prognostic factors in this patient population include the development of cardiogenic shock, right ventricular dysfunction, advanced age, and an inferior infarct location [1].

Labrousse et al. [2] reported that a relatively short interval between septum perforation and surgical repair was an important risk factor, with no deaths occurring in patients who were operated on 15 days or more after the perforation. This result can be explained by the fact that a more chronic VSR is easier to repair because the septum is well scarred and the patch can be securely sutured. However, the delay in surgery is directly dependent on the patient’s condition. Frequently, patients with similar conditions undergo an urgent or emergency operation if they have a hemodynamically unstable condition [2,3].

To delay surgery, various strategies such as adequate fluid therapy, high-dose inotropic drugs, and IABP and mechanical ventilator support have been used. Recently, ECMO has been widely administered to hemodynamically compromised patients, with the advent of a long-running oxygenator, heparin-coated tubing, a new centrifugal blood pump and generator, and percutaneous catheters. Nevertheless, conventional ECMO requires general anesthesia and mechanical ventilation, which are associated with risks such as pulmonary barotrauma, respiratory infections, and hemodynamic collapse [4]. The concept of awake ECMO has been introduced to avoid these complications. Awake ECMO prevents critically ill patients from losing muscle power due to long-term mechanical ventilation [5]. In addition, a recent study has reported the benefit of active physical therapy, including ambulation for patients on veno-arterial ECMO [6].

The indications for ECMO in patients with post-MI VSR are crucial. Patients can be placed on ECMO if their cardiac function can be resolved with primary repair or transplantation, the medical means of stabilization are unsuccessful, and emergency surgery is considered to have a prohibitive risk.

In the present case, we used awake ECMO to avoid the complications of mechanical ventilation and to delay surgery in order to improve the patient’s likelihood of survival. Awake ECMO may be a good alternative as a bridge to surgery in hemodynamically compromised patients with post-MI VSR. However, ECMO can cause complications such as hemolysis, bleeding, systemic inflammation and infection, thromboembolism, neurological sequelae, and vascular problems associated with the cannulation sites. Therefore, excessive use of ECMO can be dangerous. Thus, careful consideration is required to determine the optimal timing of surgical interventions in patients with post-MI VSR with ECMO support.

**Conflict of interest**

No potential conflict of interest relevant to this article was reported.

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