


CASE REPORT

A case of cardiopulmonary arrest due to left ventricular free wall rupture successfully treated with sutureless repair supported by venoarterial extracorporeal membrane oxygenation

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Abstract

Extracorporeal membrane oxygenation for cardiopulmonary arrest due to left ventricular free wall rupture is considered effective, because it enables rapid cardiopulmonary support and introduction of targeted temperature management.

KEYWORDS

ECMO, left ventricular free wall rupture (LVFWR), targeted temperature management (TTM)

1 | INTRODUCTION

We successfully treated a case of cardiopulmonary arrest caused by left ventricular free wall rupture (LVFWR) by immediately introducing venoarterial extracorporeal membrane oxygenation and targeted temperature management, followed by sutureless surgical repair of the LVFWR.

Left ventricular free wall rupture often occurs within 24 hours after myocardial infarction, and is a serious condition that causes cardiac arrest due to cardiac tamponade.¹ As emergency measures, relief of cardiac tamponade by pericardiocentesis and initiating venoarterial extracorporeal membrane oxygenation (VA ECMO) are considered. However, in cases of cardiac arrest, it is difficult to perform pericardiocentesis during chest compressions, and emergency left

thoracotomy or initiating ECMO may be considered.²⁻⁴ We encountered a case of cardiopulmonary arrest (CPA) due to LVFWR that was treated with VA ECMO and immediate induction of targeted temperature management (TTM).

2 | CASE REPORT

A 73-year-old man was lost consciousness and was transferred to our hospital. The patient had a history of testicular cancer treated with chemotherapy 2 years earlier and was in remission. He was barely able to communicate, and the complaint was unknown. Furthermore, he was in shock with cold extremities. His blood pressure was unmeasurably low, and neither the radial nor the common carotid artery was palpable.

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However, his limbs were still moving. His Glasgow Coma Scale result was E1V2M4 and his mental status was agitated state. Peripheral intravenous infusion was initiated and transthoracic echocardiography revealed pericardial effusion and almost no systole. Pericardial fluid was hyperechoic, which could signify hematoma formation. Pericardiocentesis was duly considered, but if a hematoma was present, it could have led to insufficient drainage. We decided to initiate ECMO in the emergency ward based on a judgment of obstructive shock or cardiogenic shock. Seven minutes after arrival, cardiopulmonary resuscitation (CPR) was started because of CPA (his breathing and limbs stopped moving), before beginning ECMO. We also considered emergency left thoracotomy, but ECMO was prioritized. We inserted an ECMO catheter under ultrasound guidance. ECMO circulation was started 17 minutes after arrival and 10 minutes after CPA. Twelve-lead electrocardiography (ECG) revealed a QS pattern and ST elevation in leads V1-V5 (Figure 1). We did not drain the pericardial fluid, but ECMO flow was stabilized using rapid infusion. The patient was immediately cooled with ECMO and reached a body temperature of 34°C 23 minutes after CPA. He was then transferred for computed tomography (CT)

for diagnosis. Contrast-enhanced CT showed no acute aortic dissection (AAD), but the anterior wall myocardium showed poor contrast; hence, we diagnosed LVFWR. According to his family, he had never complained of chest pain. We assumed that the myocardial infarction had occurred 12 to 24 hours before his arrival. No leakage of contrast agent was apparent (Figure 2). Blood tests revealed a troponin-I concentration of 13.4 ng/mL (Table 1); additional blood test results are shown in the Table 1. Emergency coronary angiography (CAG) was performed to evaluate coronary artery lesions and to determine surgical procedures. In the CAG images, the #7 branch of the left anterior descending coronary artery (LAD) was completely occluded, and the #4 branch of the right coronary artery provided collateral circulation to the LAD (Figure 3). There was no other significant stenosis, and no rupture point could be found on left ventricular angiography. An intra-aortic balloon pump (IABP) was inserted under fluoroscopy, and the patient was immediately transferred to the operating room for surgical hemostasis.

Under general anesthesia, a skin incision was made from the sternal notch to the xiphoid process, and the pericardium was exposed by a median sternotomy. Leakage of bloody

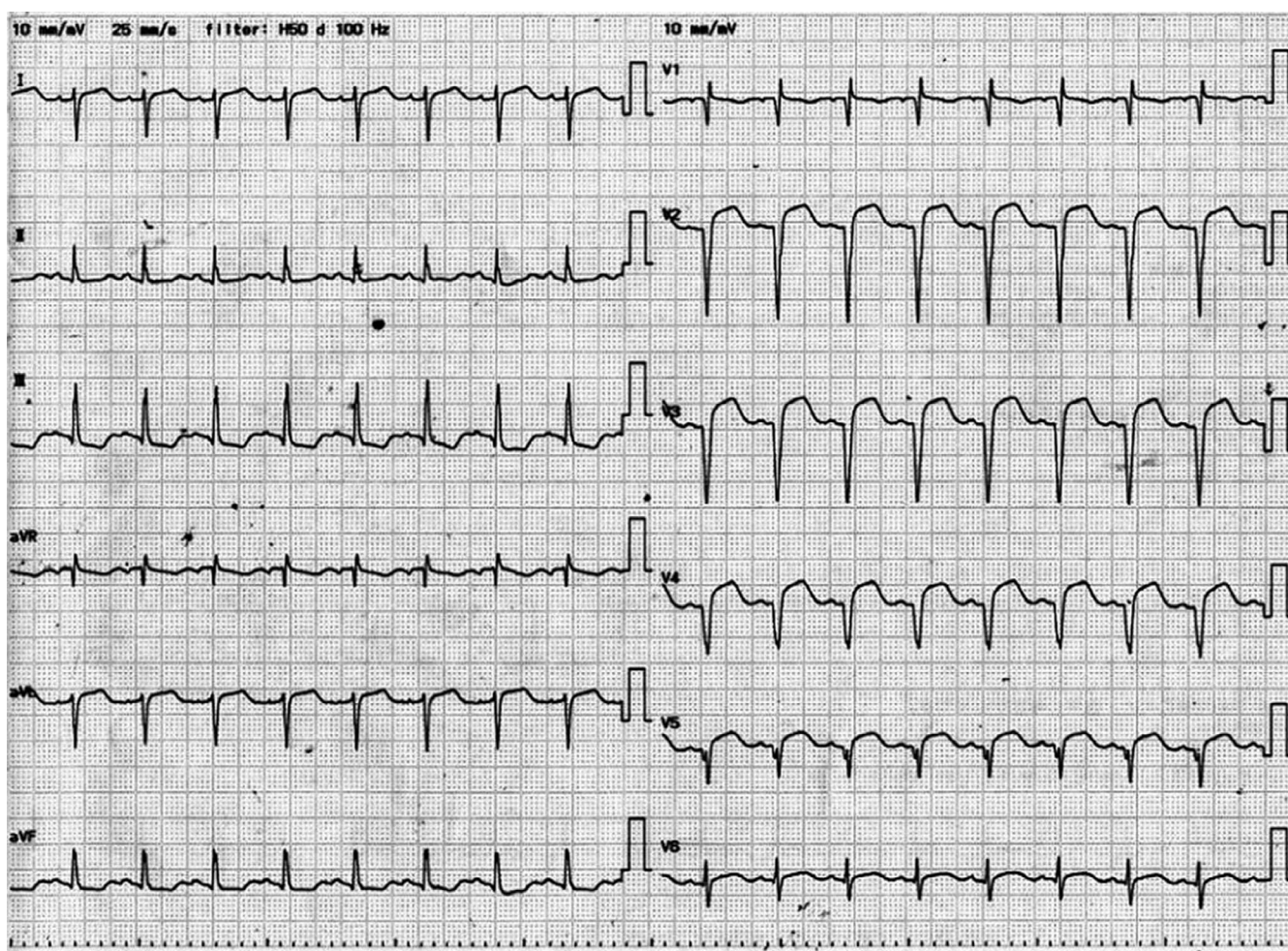


FIGURE 1 The 12-lead electrocardiogram on arrival. A QS pattern and ST elevation were found in leads V1-V5

FIGURE 2 Contrast-enhanced CT on arrival. Pericardial fluid was observed in the images. The white arrow represents the extracorporeal membrane oxygenation cannula

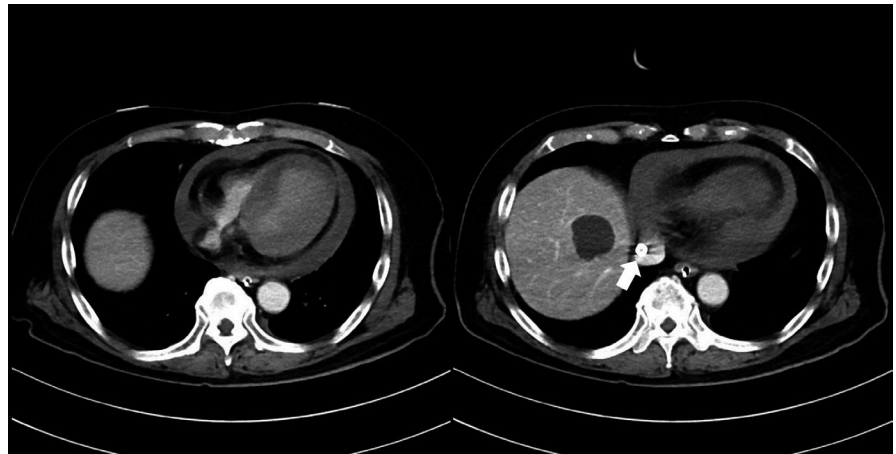


TABLE 1 The patient's blood test data

CRP (mg/dL)	6.8	WBC ($\times 10^3/\mu\text{L}$)	8.2
Na (mEq/L)	145	Hb (g/dL)	11.3
K (mEq/L)	3.3	PLT ($\times 10^3/\mu\text{L}$)	188
Cl (mEq/L)	107		
Alb (mg/dL)	3.3	PT (seconds)	9.1
T-Bil (mg/dL)	0.5	PT-INR	0.8
BUN (mg/dL)	19	APTT (seconds)	24.7
Cr (mg/dL)	1.32	FDP ($\mu\text{g/mL}$)	15.1
AST (U/L)	99	Fibrinogen (mg/dL)	692
ALT (U/L)	169		
LDH (U/L)	499		
CK (U/L)	143		
CK-MB (U/L)	22		
Troponin-I (ng/mL)	13.4		
Lactate (mmol/L)	14.9		

Abbreviations: Alb, albumin; ALT, alanine aminotransferase; APTT, activated partial thromboplastin time; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CK, creatine kinase; CK-MB, creatine kinase MB isoenzyme; Cl, chloride; Cr, creatinine; CRP, C-reactive protein; FDP, fibrin degradation product; Hb, hemoglobin; INR, international normalized ratio; K, potassium; LDH, lactate dehydrogenase; Na, sodium; PLT, platelet; PT, prothrombin time; T-Bil, total bilirubin; WBC, white blood cell.

pericardial fluid was observed when the pericardium was incised. The pericardial fluid was aspirated to remove the blood clots in the pericardium, and oozing blood was observed near the cardiac apex (Figure 4). The myocardium around the bleeding point had a poor color tone, but no myocardial necrosis was observed in other areas. A total of five pieces of TachoSil[®] (Takeda Pharmaceutical Company Limited) were attached, focusing on the bleeding site, and hemostasis was confirmed (Figure 4). We did not perform a coronary artery bypass graft (CABG) for the affected LAD because we believed that it would promote intramyocardial bleeding, which could lead to the development of blowout rupture and perforation of the interventricular septum. A towel was inserted

into the pericardium to cover the bleeding area. Next, substernal and intrapericardial drains were inserted, and a 20-cc syringe was modified and used as a sternal bridge to prevent bony union. Four pieces of gauze were also inserted into the subcutaneous space. The skin was not sutured, but instead was covered with an Esmarch tourniquet and sutured, and a sterile drape was applied. Negative pressure was applied to the drain, and the chest was temporarily closed with a vacuum pack. Surgery was performed consistently with only ECMO support and without cardiopulmonary bypass. The intraoperative blood pressure averaged 100/60 mm Hg, and the pulse was approximately 60-90 beats/minute. The lowest blood pressure recorded was 80/45 mm Hg. The total blood transfusion volume included 280 mL red blood cells and 600 mL platelets; fresh frozen plasma was not used.

Considering the possibility of recurrent cardiac tamponade, the patient was admitted to the intensive care unit (ICU) with temporary chest closure. There was marked hemorrhagic drainage from the substernal and pericardial drains, and blood products were supplemented to stabilize the hemodynamics. We continued to use 34°C TTM after surgery. If hemostasis could not be obtained, we would switch to 36°C TTM in consideration of the effect on coagulation ability. We maintained his body temperature at 34°C for 24 hours, and then rewarmed to 36°C over the next 48 hours. From the start of rewarming, cardiac function and wall movement, other than the anterior wall, gradually improved. The ECMO flow was weaned to 1 L/minute as the cardiac function improved. Under these conditions, the blood pressure was maintained at 130/80 mm Hg and the pulse rate at 80/minute, and ECMO was subsequently withdrawn after the clamp test. After which, ECMO was discontinued, but the consciousness disorder persisted. On the sixth day of hospitalization, the patient was able to communicate. It was judged that the left ventricular assisting effect of the IABP had diminished, and we removed the device. On the same day, the amount of urine started to increase and diuresis began. Because edema of the mediastinal tissue and myocardium was controlled, we closed the chest incision on the 9th day of hospitalization. The patient

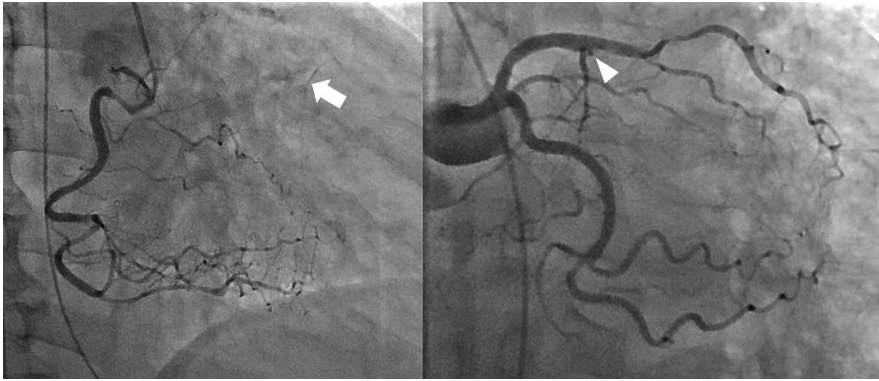


FIGURE 3 Coronary artery angiography images on arrival. The #7 branch of the left anterior descending coronary artery (LAD) was chronically totally occluded (white arrowhead), and the #4 branch of the right coronary artery provided collateral circulation to the LAD (white arrow)

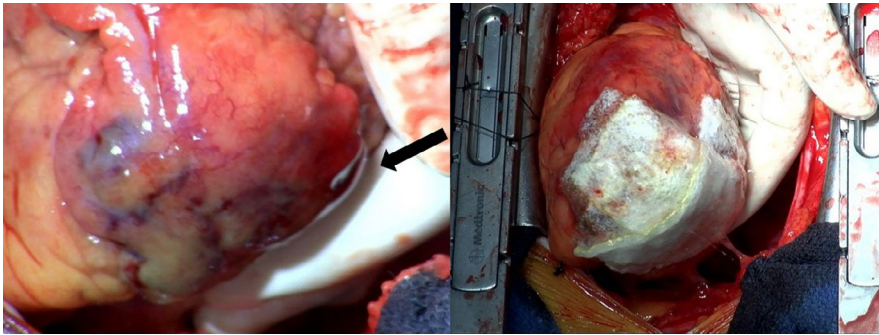


FIGURE 4 Intraoperative images. Oozing bleeding is visible near the cardiac apex (arrow). Chest closure was performed using a sutureless procedure

was extubated on the 14th day of hospitalization, and on the 15th day of hospitalization, he was transferred to the general ward without neurological deficits. He is currently receiving rehabilitation.

2.1 | Ethical approval

All subjects enrolled in this research have given their informed consent, which has been approved by the institutional committee on human and/or animal research, and this protocol has been found acceptable by them.

3 | DISCUSSION

In the present case, the keys to the patient's survival without neurological deficits were, first, the rapid initiation of ECMO for the CPA due to LVFWR, and second, we performed immediate TTM utilizing ECMO.

We would like to expand on the first point. Currently, there are three possible emergency treatments for CPA due to LVFWR, and the first is pericardiocentesis and drainage.¹⁻⁴ However, this procedure may be difficult to perform while continuing effective chest compressions. The second method involves pericardial drainage, primary hemostasis, and direct cardiac massage with left intercostal thoracotomy.⁴ This method may be the quickest way to achieve drainage for a trained doctor. However, it may be difficult to identify the

bleeding point, and if the point cannot be identified quickly, further bleeding may be promoted, and the patient may die. In addition, even if spontaneous circulation returns, this approach is highly invasive and may increase the risk of infection.⁵ Moreover, when spontaneous circulation returns, ECMO may be necessary if cardiac function is severely deteriorated due to myocardial infarction. The third approach is to use ECMO.^{2,3} We were able to establish ECMO as early as 10 minutes after CPA, as skilled doctors and clinical engineers were available continuously at our university hospital. As in the present case, when CPA occurs before a definitive diagnosis, AAD may be a cause of pericardial effusion,⁶ and lifesaving is expected to be difficult with left thoracotomy. Even if ST changes are detected on the ECG, these changes may occur with coronary artery dissociation, so definitive diagnosis before CT is difficult. Pericardiocentesis can treat circulatory collapse for oozing-type myocardial ruptures, but hematoma formation may result in insufficient drainage of the pericardial fluid. In addition, in case of blowout type cardiac rupture, successful resuscitation becomes difficult without mechanical support. The type of cardiac rupture is also difficult to differentiate by examination or echography alone. In addition, even in case of pericardial effusion due to AAD, if there is likelihood of cardiac arrest, we believe that priority should be given to the establishment of ECMO considering the possibility that sufficient drainage of pericardial fluid cannot occur due to hematoma formation. ECMO flow may be insufficient for cardiac tamponade, but there is a possibility of survival with concomitant pericardiocentesis.⁶ Even

with CPA due to LVFWR, initiating ECMO is considered effective with skilled staff. Furthermore, introducing ECMO permits CAG. If there was stenosis in the remaining branch in our patient, CABG may have been added.

Regarding the second key point, in the present case, rapid ECMO enabled early introduction of TTM by rapid blood cooling. The patient's body temperature reached 34°C 30 minutes after arrival and 23 minutes after CPA, suggesting the usefulness of ECMO from the viewpoint of brain protection. If resuscitation is performed with left thoracotomy, even if TTM is performed, an intravascular cooling device or body surface cooling is necessary,⁷ and it is difficult to introduce TTM promptly. Delayed brain protection can lead to post-cardiac arrest encephalopathy. In the present case, we introduced TTM at 34°C. If hemostasis could not be obtained, we planned to switch the TTM to 36°C in consideration of the effect of body temperature on coagulation ability. However, there is no evidence that TTM in ECMO should be performed at 34°C, so it may have been better to target 36°C for safety reasons. This needs further consideration.

In conclusion, ECMO for CPA due to LVFWR is considered an effective resuscitation method because it enables rapid and minimally invasive cardiopulmonary support and rapid introduction of TTM.

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CONFLICT OF INTEREST

None declared.

AUTHORS CONTRIBUTIONS

GS: wrote and drafted the manuscript. GS, RI, SY, HS, YN, MW and MH: helped draft the manuscript. All authors read and approved the final manuscript.

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