Successful Management of a Patient with Refractory Ventricular Fibrillation (VF) due to Acute Myocardial Infarction (AMI) and Lung Injury by Transition from Percutaneous Cardiopulmonary Support (PCPS) to Veno-Venous Extracorporeal Membrane Oxygenation (ECMO)

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Abstract

A 69-year-old man was admitted to our hospital with cardiopulmonary arrest. Percutaneous cardiopulmonary support (PCPS) using the right femoral artery and vein was initiated, because ventricular fibrillation continued. Although we succeeded in defibrillation after percutaneous coronary intervention (PCI), a chest radiograph indicated a pneumothorax in the right lung and a pulmonic contusion in the left lung caused by cardiopulmonary resuscitation. Two days after PCI, partial pressure of arterial oxygen (PaO₂) from the right radial artery suddenly decreased, and his cardiac function showed improvement on an echocardiogram. To avoid additional brain damage, we converted the treatment to veno-venous extracorporeal membrane oxygenation by changing the blood returning site of PCPS from the right femoral artery to the right jugular vein. Thereafter, the patient’s PaO₂ level gradually improved.

Key words: acute myocardial infarction, cardiopulmonary resuscitation, lung injury, PCPS, ECMO


Introduction

Cardiopulmonary resuscitation (CPR) is a necessary procedure to rescue cardiopulmonary arrest patients. However, CPR has several complications, which include rib fracture, sternal fracture, mediastinal hemorrhage, hemothorax, pneumothorax, and lung contusion (1). We herein describe the successful management of an acute myocardial infarction (AMI) patient with lung injury due to CPR by transition from percutaneous cardiopulmonary support (PCPS) to a veno-venous extracorporeal membrane oxygenation (ECMO).

Case Report

A 69-year-old man was admitted to our hospital presenting with cardiopulmonary arrest. In the emergency room, an electrocardiogram (ECG) showed ventricular fibrillation. We performed CPR and defibrillation several times with the injection of adrenaline and amiodarone, however, ventricular fibrillation persisted. He was immediately intubated and transferred to the catheterization laboratory. PCPS was rapidly inserted through the right femoral artery and vein, and intra-aortic balloon pumping (IABP) was inserted through the left femoral artery. Then, we performed coronary angiography (CAG), which revealed total occlusion of the proximal right coronary artery (RCA) (Fig. 1, left). After aspiration, we implanted a bare metal stent (Integrity 3.5×18 mm).
Figure 1. Angiography of the right coronary artery (RCA). Emergency coronary angiography (CAG) revealed total occlusion of the proximal RCA (left). Final CAG showed that the RCA flow improved to TIMI 3 (right).

Figure 2. Serial images of chest Xp. The left chest Xp shows a pneumothorax in the right lung and a pulmonary contusion in the left lung on the first hospital day. The middle chest Xp reveals recovery of the pneumothorax in the right lung and improvement of the pulmonary contusion in the left lung on the second hospital day. The right chest Xp demonstrates recovery of lung injury on the 9th hospital day.

and CAG showed that the RCA improved to TIMI 3 (Fig. 1, right). Finally, we successfully performed defibrillation and his vital signs thereafter became stable.

After intervention, a chest radiograph indicated a pneumothorax in the right lung and a pulmonary contusion in the left lung, which had been caused by CPR (Fig. 2, left). We subsequently inserted a chest tube in the right thoracic cavity. Two days after percutaneous coronary intervention (PCI), partial pressure of arterial oxygen (PaO₂) from the right radial artery suddenly decreased (Fig. 3), and a blood gas analysis from the right radial artery under the ventilator (tidal volume 370 mL, FiO₂ 1.0 and positive end-expiratory pressure (PEEP) 10 cm H₂O) and PCPS (FiO₂ 1.0 and 3.5 L/min) showed a pH 7.374 of partial pressure of oxygen (pO₂) 43.3 mmHg, and carbon dioxide partial pressure (pCO₂) 45.4 mmHg. However, an echocardiogram at this time revealed that his cardiac function significantly improved [left ventricular ejection fraction (LVEF) 0%→32%]. We speculated that the blood flow from his heart showed low oxygenation due to his lung injury (Fig. 2, middle), and the mixing point was moved into the peripheral side from his aortic arch. To avoid additional brain damage due to hypoxia, we converted PCPS to veno-venous ECMO by changing the blood returning site of PCPS from the right femoral artery to the right jugular vein. To prevent recirculation in veno-venous ECMO, we separated the area between the sending line and the removal line pulling down into the inferior vena cava and decreased the pump flow. After conversion from PCPS to veno-venous ECMO, the systemic blood pressure (84/48 mmHg→102/58 mmHg) and LVEF (32%→54%) rapidly improved. Furthermore, PaO₂ gradually improved (Fig. 3), and both veno-venous ECMO and IABP were successfully removed due to complete recovery of the injured lung 5 days after PCI. After extubation on the 9th hospital day (Fig. 2, right), the patient underwent rehabilitation. Finally, he was discharged without any sequelae on the 21st hospital day.
Figure 3. Clinical course in the coronary care unit. Two days after PCI, PaO₂ from the right radial artery suddenly decreased, and we converted the treatment to veno-venous ECMO by changing the blood returning site of PCPS from the right femoral artery to the right jugular vein. On the 3rd hospital day, PaO₂ of the right radial artery gradually improved. The patient’s pneumothorax and pulmonary contusion healed, and veno-venous ECMO was successfully removed on the 4th hospital day. Finally, he was extubated on the 9th hospital day.

Discussion
CPR is a necessary procedure to rescue cardiopulmonary arrest patients. However, CPR has several complications, which include rib fracture, sternal fracture, mediastinal hemorrhage, hemothorax, pneumothorax, and lung contusion. A recent report demonstrated that pneumothorax and lung contusion as CPR complications develop in 8.4% and 4.2%, respectively, of all patients (1). Our patient had a pneumothorax in the right lung and a pulmonic contusion in the left lung, which were caused by CPR. The lung injury affected low oxygenation of the blood from his heart at 2 days after PCI. His cardiac function greatly improved, and the flow of the low oxygenated blood from his heart overcame the flow of the high oxygenated blood from PCPS near the aortic arch. Indeed, a blood gas analysis from his right radial artery under the ventilator (tidal volume 370 mL, FiO₂ 1.0 and PEEP 10 cm H₂O) and PCPS (FiO₂ 1.0 and 3.5 L/min) showed a pH 7.374 of pO₂-43.3 mmHg, and pCO₂-45.4 mmHg. Furthermore, the blood flow from PCPS results in left ventricular afterload. To avoid additional brain damage due to hypoxia and afterload for the infarcted left ventricle, we converted to veno-venous ECMO by changing the blood returning site of PCPS from the right femoral artery to the right jugular vein. We speculate that the transition from PCPS to veno-venous ECMO is not difficult, because it is possible to use the circuit and pump of PCPS previously placed in the patient. Veno-venous ECMO improved uniform oxygenation of all organs, and facilitated recovery of the respiratory function by providing high oxygenated blood to the alveolus of his lung and changing the ventilation with a lower tidal volume and lower concentration of oxygen [referred as to “lung rest” (2)]. The adaptation of veno-venous ECMO is a respiratory disorder with a preserved cardiac function. We therefore believed that the condition of our patient after the second hospital day should be good indication for veno-venous ECMO.

This is the first report to describe the successful management of an AMI patient with lung injury due to CPR using the transition from PCPS to veno-venous ECMO.

The authors state that they have no Conflict of Interest (COI).

References