ST-ELEVATION MYOCARDIAL INFARCTION WITH CARDIOGENIC SHOCK RESCUED BY USE OF EXTRACORPOREAL MEMBRANE OXYGENATION: A CASE REPORT AND LITERATURE REVIEW

Li-Chin Sung¹, Yu-Chih Chen¹, Shen-Feng Chao², Ji-Hung Wang¹

Abstract

Cardiogenic shock is the leading cause of death in patients with acute myocardial infarction (AMI) despite aggressive treatment modalities such as fibrinolysis and percutaneous transluminal coronary angioplasty. We report our experience with a case of ST segment elevation AMI complicated with refractory cardiogenic shock and ventricular tachyarrhythmia even after successful revascularization using hemodynamic support with the extracorporeal membrane oxygenation (ECMO). This patient was weaned from ECMO, survived and discharged from hospital. The purpose of the ECMO is to restore the systemic perfusion while intra-aortic balloon pump failed and to avoid severe injury to the organs, and to allow time for intrinsic recovery of the heart, thus, improving the chance of survival.

Key words: Acute myocardial infarction, Cardiogenic shock, Extracorporeal membrane oxygenation

Introduction

Cardiogenic shock develops in 7-10% of cases after acute myocardial infarction (AMI) and is the most common cause of death.¹ Despite aggressive treatment modalities, the mortality rates of cardiogenic shock remain high. Extracorporeal membrane oxygenation (ECMO) for cardiogenic shock has been conducted since 1970s and has been proven to improve the survival rate of such patients.² We report our patient with AMI and profound cardiogenic shock refractory to high doses of inotropic agents and intra-aortic balloon pump (IABP) / emergent angioplasty who had been rescued by using ECMO hemodynamic support.

Case Presentation

A 50-year-old male patient presented with chest pain and was diagnosed as acute extensive anteroseptal myocardial infarction at emergency department. The patient had smoking, hypertension and dyslipidemia as risk factors for coronary artery disease. His blood pressure (BP) was 84/35 mmHg and the pulse rate was 153 beats/min.

Correspondence: Dr. Ji-Hung Wang
Division of Cardiology, Department of Internal Medicine, Buddhist Tzuchi General Hospital, Hualien, Taiwan; No. 707, Sec. 3, Chung-Yang Rd., Hualien City, Hualien County 970, Taiwan
Phone: 886-3-856-1825 ext. 2226; Fax: 886-3-846-6959; E-mail:jihung_wang@tzuchi.com.tw
¹Division of Cardiology, Department of Internal Medicine, and ²Division of Cardiovascular Surgery, Department of Surgery, Buddhist Tzuchi General Hospital, Hualien, Taiwan
The physical examination of the patient revealed inspiratory crackles of bilateral lower lung fields and irregular heart beat. The electrocardiogram (ECG) showed ST segment elevation in leads V1-5 with reciprocal ST segment depression in leads II, III, and AVF, and atrial fibrillation (Fig. 1). The emergent percutaneous coronary intervention (PCI) was scheduled for AMI with cardiogenic shock. Unfortunately, the patient had ventricular tachycardia/fibrillation (VT/VF) at catheterization room before coronary angiography and resuscitated by chest compression, defibrillation and epinephrine. After successful resuscitation, the systolic BP of this patient was restored to 70 mmHg under IABP support and high doses of inotropic agents administration. We began the PCI procedure immediately. The coronary angiography revealed total occlusion of the proximal part of left anterior descending artery and insignificant stenosis of the right coronary artery and left circumflex. We opened the occluded artery by deploying one stent. The total procedure time of PCI was about 10 minutes. Thereafter, he was sent to intensive care unit for further care. The mean BP was less than 65 mmHg with infusion of high doses of norepinephrine (2 mcg/kg/min) and dopamine (>10 mcg/kg/min). Besides, the patient had sustained VT/VF with undetectable BP even with multiple electrical cardioversions, IABP support and use of various anti-arrhythmic agents. Under the impression of cardiogenic shock and circulatory collapse due to sustained VT/VF refractory to conventional therapy, we consulted cardiovascular surgeon for ECMO support. We used venoarterial (VA) ECMO with centrifugal pump to support the patient through right femoral approach with vascular exploration by percutaneous Seldinger’s technique. One 17-French arterial cannula was inserted into the iliac artery via right femoral artery and one 19-French venous cannula was inserted through the right femoral vein and advanced into the inferior vena cava. The initial ECMO blood flow was 2500 cc/min. The VT/VF disappeared and patient’s hemodynamics became stable after ECMO was set up. The patient was weaned from ECMO after 62 hours of support. The IABP was also weaned off successfully 7 days later. The serial echocardiography showed his heart function did improve substantially with left ventricular ejection frac-

Fig. 1. ECG demonstrates ST segment elevation in leads V1-5 with reciprocal ST segment depression in leads II, III, and AVF, and atrial fibrillation.
tion measured from 35% to 75%. The echocardiography also demonstrated mild hypokinesis of anteroseptal wall of LV on the 5th day after PCI. Two weeks later, the patient was discharged without any symptoms of congestive heart failure. During the 3 months of follow up, the patient has remained free of symptoms with no cardiovascular event.

Discussion

Cadiogenic shock is the most common cause of death in patients with AMI. The mortality of AMI complicated with cardiogenic shock was 60-80%. Early diagnosis and revascularization for ST segment elevation AMI including PCI or coronary bypass graft (CABG) has been shown to increase the survival of these patients, consistent with the guidelines. A strategy of early revascularization resulted in a 13.2% absolute and a 67% relative improvement in 6-year survival compared with isolated medical treatment. There was no significant difference in survival between PCI or CABG group for early revascularization. AMI complicated with cardiogenic shock leads to further damage to myocardium, and decrease cardiac output, arterial pressure and coronary perfusion. These events trigger compensatory response, which include activation of the rennin-angiotensin and sympathetic systems, leading to fluid retention, systemic vasoconstriction, and tachycardia. These mechanisms contribute to further reduction in cardiac output and worsen myocardial ischemia. However, recent evidence from analysis of results from the SHOCK trial suggests that about one fifth of patients had systemic inflammatory response syndrome (SIRS) in the presentation of cardiogenic shock. The clinical presentation of SIRS include fever, leukocytosis and low systemic vascular resistance. The vasodilatation appears to be the end result of high concentration of nitric oxide produced by nitric oxide synthase (NOS) during AMI.

The American College of Cardiology/American Heart Association has given emergent coronary artery revascularization in combination with IABP mechanical support a class I recommendation for use in AMI with pharmacologically resistant cardiogenic shock. Mortality rate of cardiogenic shock remains at high level with aggressive treatment modalities. Myocardial stunning with severe left ventricular dysfunction may require several days to recovery after successful revascularization. The use of mechanical assistance such as IABP or ECMO provides a time window for PCI and recovery of myocardial contractility.

The major limitations of the IABP are the lack of active cardiac support and the requirement of a certain level of left ventricular function. In patients with severe left ventricular dysfunction or tachyarrhythmia, left ventricular unloading and hemodynamic support from IABP is insufficient to reverse cardiogenic shock. In this situation, the ECMO can restore systemic circulation, increase diastolic perfusion of myocardium, reduce cardiac workload, eliminate the dosage of inotropic agents and prevent further injury of organ by SIRS reaction.

ECMO for cardiogenic shock has been conducted since 1970s and has been proven to improve the survival rate of such patients. There are two types of ECMO- Venovenous (VV) and VA. Both provide respiratory support, but only VA ECMO provides hemodynamic support. Our patient was placed on VA ECMO in order to decrease the cardiac workload and for hemodynamic stabilization. The complications of ECMO include bleeding, heparin-induced thrombocytopenia, thromboembolism, vessel perforation and limbs ischemia. Complications specific to VA ECMO include arterial thrombosis or cerebral hypoxemia. There were no severe complications and the bleeding was minimal in our patient. However, the routine use of ECMO is limited by their availability, technique expertise and surgeon with specialized training required to successfully implant the device.

Use of a ventricular assist device (VAD) might be another alternative to improve the survival. Recently, a newly developed percutaneous left VAD may offer effective treatment for these
 ECMO for STEMI with cardiogenic shock

patients\textsuperscript{13,15} without the need for extracorporeal oxygenation and surgical procedure. The VAD has two catheter: venous inflow catheter was inserted into left atrium by trans-septal puncture method via femoral vein and arterial perfusion catheter was inserted into femoral artery. This device diverting oxygenated blood from left atrium to the femoral artery, which will result in reducing filling pressure in the left ventricle, cardiac workload and oxygen demand. The VAD significantly improves hemodynamics and urine output compared to IABP in one study.\textsuperscript{15} However, this device can not be used in patient with right ventricle failure or severe peripheral vascular disease,\textsuperscript{16} and its effect on long-term prognosis deserve further investigation.

After revascularization, the VT/VF occurring in this patient was refractory to IABP, defibrillation and anti-arrhythmic agents. The sustained VT/VF may be secondary to myocardial ischemia, infarction, stunning, reperfusion or prolong shock with metabolic derangements. The patient’s condition stabilized after ECMO was set up, thus, the sustained cardiogenic shock with/without VT/VF after revascularization may be an indication for ECMO support. The Sepsis-related Organ Failure Assessment (SOFA) score can be applied in an ECMO situation to predict outcomes in one original study.\textsuperscript{17} Our patient’s maximal SOFA score was below the cut-off value of 17.8 ± 2.8 and correlated with better prognosis. The characteristics of our patient included younger age (< 75 years old), first AMI episode, no other systemic co-morbidity and anticipated reversible myocardial dysfunction due to early successful PCI for single coronary artery lesion.

In conclusion, AMI complicated with refractory shock is a life-threatening emergency. Early revascularization such as PCI, thrombolysis or CABG may improve the survival rate. The basic approach is rapid reperfusion of myocardium and support of hemodynamic system. The latter includes adequate fluid volume, maintaining suitable cardiac output with drugs or mechanical assistance and providing adequate oxygenation. The hemodynamic failure is the most frequent cause of in-hospital mortality, so the complete hemodynamic support is mandatory. The ECMO is essential to support organ function by prompt reversal of hypoperfusion, both during myocardial recovery after treatment and to stabilize the patient for definitive percutaneous or surgical intervention. In patients with AMI with cardiogenic shock/or sustained VT/VF, who are unresponsive to conventional treatment including IABP, the early ECMO support may be considered to shorten the duration of shock and save life.

References

8. Hochman JS. Cardiogenic shock complicating acute myocardial infarction: expanding the paradigm.


使用體外膜氧合來援救 ST 段升高心肌梗塞合併心因性休克：一病例報告及文獻回顧

宋立勤¹，陳郁志¹，趙盛豐²，王志鴻¹

摘要

雖然在血栓溶解劑或經皮腔內冠狀動脈成形術的積極治療下，急性心肌梗塞的病人若合併有心因性休克，仍是造成死亡主要的原因。我們報告我們的經驗在一個 ST 段升高急性心肌梗塞的病人接受成功的血管再通術後，依然有顚因性心因性休克及心室頻脈心律不整，使用體外膜氧合來當做血行動力支持。這個病人成功地脫離體外膜氧合並存活而出院。而使用體外膜氧合的主要目的在於當主動脈內氣球幫浦失敗時可再增加系統灌流、避免器官嚴重的損傷，並提供時間讓心臟得以恢復功能來增加病人的存活率。

關鍵詞：急性心肌梗塞，心因性休克，體外膜氧合

聯絡人：王志鴻醫師
970 花蓮市中央路三段 707 號 花蓮慈濟醫院內科部心臟內科
電話：03-856-1825 轉 2226；傳真：03-846-6959；E-mail:jihung_wang@tzuchi.com.tw
花蓮慈濟醫院外科部心臟外科²

209