EMERGENT PERCUTANEOUS CORONARY INTERVENTION UNDER EXTRACORPOREAL MEMBRANE OXYGENATION SUPPORT FOR A PATIENT WITH INFERIOR ST ELEVATION MYOCARDIAL INFARCTION AND REFRACTORY SHOCK: A CASE REPORT AND LITERATURE REVIEW

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Abstract

Early emergency revascularization with intra-aortic balloon pump (IABP) support is mandatory for patients with acute myocardial infarction (AMI) and concomitant cardiogenic shock. We report our experience with a patient of cardiogenic shock caused by an AMI refractory to conventional therapies including IABP that impede subsequent revascularization procedures. We used extracorporeal membrane oxygenation (ECMO) to provide further hemodynamic support and performed successful revascularization. However, the patient failed to wean from ECMO and severe mitral regurgitation with suspected papillary muscle rupture was observed 5 days later. The patient later underwent mitral valve replacement, survived and was discharged from the hospital. The use of ECMO can support hemodynamics for revascularization and reverse shock-induced organ damage. The shock and heart failure caused by mitral regurgitation should be considered in patients with inferoposterior AMI after successful revascularization, who are still hemodynamically unstable. We also discuss the management regarding inferior AMI with mitral regurgitation and refractory shock.

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

Introduction

Cardiogenic shock develops in 7.5% of cases after acute myocardial infarction (AMI) and is the leading cause of death.1 Despite aggressive treatment strategies such as fibrinolysis and early revascularization, the mortality rates of cardiogenic shock are still high. We report our patient with inferior ST segment elevation AMI and profound cardiogenic shock unresponsive to high...
Case Presentation

A 57-year-old male patient with a previous history of hypertension suffered from dyspnea and chest tightness one hour prior to admission. He was brought to our emergency department due to dyspnea aggravated progressively. On arrival, the patient’s vital signs were as follows: pulse rate 176 beats/min, respiratory rate 27 breaths/min and blood pressure 96/69 mm Hg. The physical examination of the patient revealed inspiratory crackles of bilateral lung fields. The ECG showed ST segment elevation in leads II, III, and AVF, ST segment depression in leads V1-6 and sinus tachycardia (Fig. 1). The right-side ECG revealed no ST segment elevation in lead V4. The chest radiography demonstrated diffuse bilateral interstitial infiltration, compatible with pulmonary edema (Fig. 2). Blood tests showed normal electrolytes, creatine kinase (CK) 223 u/l with MB fraction 45 u/l, troponin-I 2.59 ng/dl, lactate 7.4 mmol/L, D-dimer 5702.83 ng/dl, and FDP 641.8 ng/dl. The arterial blood gas obtained with the patient placed on 100% oxygen therapy via a non-rebreathing mask showed PH 7.008, Pco2 66.6 mmHg, Po2 69.5 mmHg, HCO3 16.8 mmol/L, and O2 sat 81.8%. The patient was intubated and placed on ventilator support. The transthoracic echocardiography showed moderate mitral regurgitation (MR) and severe hypokinesis of inferoposterior wall of the left ventricle (LV) with estimated ejection fraction of 52%. The blood pressure of this patient went down to an unrecordable level after ICU admission. The intravascular fluid challenge, high doses of dopamine (>10mcg/kg/min) and norepinephrine (2mcg/kg/min) infusion and IABP support were given. In spite of aggressive resuscitation, this patient was still in shock status and had severe hypoxemia even 100% oxygen giving via a ventilator. We consulted cardiovascular surgeon (CVS) to set up ECMO. We used venoarterial (VA) ECMO with centrifugal pump through the

Fig. 1. The ECG demonstrated ST segment elevation in leads II, III, and AVF, ST segment depression in leads V1-6 and sinus tachycardia.
right femoral artery and vein. Then we performed coronary angiography (CAG) immediately. The CAG revealed total occlusion of the middle part of the right coronary artery (RCA) (Fig. 3A), patent left anterior descending artery (LAD) and 95% stenosis of the proximal left circumflex artery (LCX) (Fig. 3B). The complete revascularization was performed with stent deployment in the RCA (Figure 3C) and LCX lesions (Fig. 3D). The hemodynamics of our patient improved after the percutaneous coronary intervention (PCI) procedure. The peak cardiac enzyme (CK/CKMB: 718/103 u/l) was noted within the 12 hours after PCI. However, the persistence of pulmonary edema and difficulty in weaning from ECMO were both observed on the 5th post-procedure day. There was no evidence of recurrent infarction based on the serial ECG and cardiac enzymes follow-

Fig. 2. The chest radiography demonstrated severe bilateral pulmonary edema.

Fig. 3. (A) Coronary angiography revealed total occlusion of the middle part of the RCA and (B) 95% stenosis of the LCX. (C) Repeat coronary angiography following successful PCI of the RCA and (D) the LCX. (RCA: right coronary artery; PCI: percutaneous coronary intervention; LCX: left circumflex artery)
up. The pulmonary artery occlusion pressure was 22 mm Hg and its waveform tracing showed prominent regurgitant V wave. The transesophageal echocardiography showed severe MR with suspected papillary muscle rupture (Fig. 4). We consulted CVS for surgical intervention. During the operation, severe anterior mitral valve prolapse (MVP) without papillary muscle rupture was detected. Then mechanical mitral valve replacement (MVR) was done. ECMO was removed successfully after operation with total support duration of 120 hours. The IABP was removed on the post-MVR day 5. The patient was extubated on the post-MVR day 18. The patient recovered well and was discharged 28 days after admission. During the 18-month follow-up period, the patient has remained free of symptoms with no cardiovascular event.

Discussion

ST segment elevation AMI with cardiogenic shock is a medical emergency and the mortality was about 60 to 80% in previous reports. Early successful revascularization including PCI or coronary artery bypass graft (CABG) has been shown to increase the survival rate. AMI with cardiogenic shock leads to decrease cardiac performance, blood pressure, coronary perfusion and further damage to myocardium. These events induce compensatory response, which include activation of the sympathetic and renin-angiotensin systems, leading to progressive worsening ischemia, impairment of cardiac output, and worsening shock. The initial therapy for cardiogenic shock is to maintain adequate organ perfusion and achieve target vessel revascularization. Usually, we can raise blood pressure by using vasopressors/IABP and perform angioplasty safely. However, it is nearly impossible to perform PCI in patients with refractory shock.

The use of IABP is essential in patients with AMI complicated with shock. The major limitations of the IABP are the requirement of some level of LV function and the lack of active cardiovascular support. The hemodynamic support from IABP is usually insufficient to revert circulatory collapse in patients with massive AMI.

![Fig. 4. The transesophageal echocardiography showed severe mitral regurgitation with suspected papillary muscle rupture.](image-url)
ECMO for cardiogenic shock has been used since 1970s and has been proven to improve the survival rate. There are two types of ECMO: Venovenous ECMO provides respiratory support. VA ECMO provides both respiratory and hemodynamic support. Our patient used VA ECMO for circulatory shock. The ECMO has several advantages as described below: (1) It provides more effective hemodynamic support than IABP. (2) It is relatively less costly than other forms of mechanical circulatory support. (3) The percutaneous approach is technically simple, rapid and can be done at bedside. ECMO setup can be achieved within one hour in our hospital. The use of ECMO can effectively restore systemic circulation, reduce cardiac workload, eliminate the doses of inotropic agents and prevent shock-induced organ damage. However, ECMO support has several disadvantages include the significant risk of stroke, need for anticoagulant, limited duration of support and distal limbs ischemia. The ventricular assist device (VAD) is another form of mechanical circulatory support. Recently, a newly developed percutaneous left VAD may offer effective treatment for these patients. This device diverts oxygenated blood from left atrium to the femoral artery to increase systemic perfusion and reduce cardiac workload of the LV. The VAD significant improves hemodynamics compared to IABP and provides a means for long-term support. The VAD was not available in our institution and there were several reasons against its use. The implantation of VAD was more time-consuming and costly procedure than ECMO. The long-term support for this patient was not anticipated due to treatable illness and the characteristics of our patient, which included young (≤ 75 years of age) age, first AMI episode, no multiple co-morbidities in the past and shock-induced organ damage was not prolong before ECMO support.

The cardiac catheterization can be safely performed on patients with failing circulation under ECMO support in one retrospective review. Although there is little literature about the PCI for ST segment elevation AMI under ECMO support, it seems to be feasible according to the result of our patient. The use of ECMO also provides a time for functional recovery of myocardial stunning. The effectiveness of ECMO is unclear in patients with non-ST segment elevation AMI and shock because these patients are older, and have higher rates of prior myocardial infarction, three-vessel coronary disease and multiple co-morbidities.

The coronary lesions in patients with ST segment elevation AMI and shock are usually left main, severe triple-vessel or proximal LAD stenosis. In patients with inferior ST segment elevation AMI and shock, the concomitant right ventricular infarction or mechanical complications such as MR or ventricular septal defect should be evaluated. The right side ECG, chest X-ray, echocardiography and physical examination are enough to make differential diagnosis. The right heart catheterization is usually unnecessary except the cause of shock is unknown. The presence of large V wave is usually a non-specific finding indicating decrease LV compliance and not necessarily acute MR. The causes of MR following AMI are mostly due to LV dilatation, papillary muscle dysfunction, mitral annular dilatation and inferoposterior asynergy. The ischemic MR is usually without valvular damage. We performed PCI in order to improve inferoposterior wall of LV hypokinesis, papillary muscle dysfunction and degree of MR. In previous publications, the acute MR related to papillary muscle rupture occurs at a median of 1 day following AMI, and the subacute and chronic MR related to papillary muscles dysfunction due to scar, hibernation or repeated ischemia occurs at a median of 7 days after AMI. The presence of any degree MR carries an adverse prognosis in patients undergoing PCI for AMI and aggressive medical or surgical therapies may improve the prognosis. Serial echocardiography was suggested to detect MR in post-AMI patients and thus pave the way for further treatment. Our patient had severe MR in the subacute phase of AMI maybe due to papillary muscle dysfunction. The MVR for this patient was decided because
of difficulty in weaning from ECMO and suspecting papillary muscle rupture before operation. This patient could be weaned from ECMO quickly after MVR as an indirect evidence of hemodynamic compromise due to severe MR.

There were two important decisions that made this patient survival possible. Failing circulation under ECMO support providing the chance for PCI was the first critical point. The secondary critical point was the surgical therapy for severe MR after AMI. The acute MR may be the main cause of cardiogenic shock because of pulmonary edema without severe LV dysfunction in our patient. However, the question arises: Why don’t we suggest conjunct CABG and MVR initially. There were several reasons for this: firstly, the patient had no history of diabetes mellitus and the CAG revealed double-vessel disease without LAD involvement; secondly, we can shorten more cardiac ischemic time by performing PCI than surgery; thirdly, the improvement in ischemic MR after PCI was anticipated; fourthly, the hospital mortality of MVR was high (40%) under the condition of shock-induced multiple organ failure; lastly, patients not surviving the PCI therapy under ECMO support would not have survived surgical therapy. However, the PCI may be abandoned if coronary lesions are at high risk for complications and anatomically unsuitable. The surgical repair or PCI for shock due to MR complicating AMI is still equivocal. The favorable response of acute severe MR to PCI has been reported. Tcheng reported the mortality rate was higher in patients with acute ischemic MR treated with PCI than in those treated medically or with surgery. However, patients with better LV function and smaller infarctions into the surgical group may lead to a selection bias and the use of ECMO was not mentioned in this observational study. They advocated earlier surgery (before shock) of severe MR complicating AMI may improve prognosis. But the proper therapy for patients with acute ischemic MR and refractory shock is still unclear due to no randomized trial.

In conclusion, ST segment elevation AMI complicated with shock is challenge to every cardiologist. Early mechanical revascularization may improve the survival rate. However, there is little chance to perform PCI in patients with extreme unstable hemodynamics. The asystole/ventricular tachyarrhythmia requiring cardiopulmonary resuscitation may occur when ischemic burden adds to the failing heart during CAG, PCI or contrast injection. Besides, shock-induced irreversible microvascular collapse was considered the main cause of death among patients with AMI and shock despite successful revascularization. The use of ECMO can solve above problems. From this case, we learn that early application of ECMO is essential to safe life in AMI patients with short-term refractory shock. If the inferior AMI patients survive the initial period of revascularization, the MR should be followed up closely. The hemodynamically significant MR should be suspected when these patients are difficult to wean from ECMO after successful PCI.

References

4. Sung LC, Chen YC, Chao SF, Wang JH. ST-Elevation Myocardial Infarction with Cardiogenic Shock Rescued by Use of Extracorporeal Membrane Oxygenation:


在體外膜氧合支援下的緊急經皮冠狀動脈介入治療下壁 ST 段升高心肌梗塞合併類性休克的病人：一案例報告及文獻回顧

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摘要

主動脈內氣球幫浦支持下的早期緊急血管再通術對於急性心肌梗塞合併心因性休克的病人是必要的。我們報告一個急性心肌梗塞導致心因性休克病人的經驗，其對於傳統的治療，包括主動脈內氣球幫浦無效且無法進行後續的血管再通術。我們使用體外膜氧合來當做進一步血行動力支持，並且施行成功的血管再通術。然而這病人術後並無法脫離體外膜氧合，五天後我們觀察到疑似乳頭肌斷裂所引起的重度二尖瓣膜逆流。這個病人後來接受二尖瓣膜置換而成功地存活且出院。故使用體外膜氧合可做血行動力支持及利血管再通及回復休克所引起的器官傷害。當這類已接受成功血管再通術的下壁心肌梗塞病人，其血行動力仍不穩定時，我們必須想到是二尖瓣膜逆流所引起的休克及心衰竭。我們另外討論急性下壁心肌梗塞合併二尖瓣膜逆流及類性休克的處理。

關鍵詞：急性心肌梗塞，心因性休克，體外膜氧合，重度二尖瓣膜逆流

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