Aggressive management of acute myocardial infarction: successful outcome in an older patient with cardiogenic shock

Tsao Y T, Wu C J, Lin S L, Liu C P, Tak T

ABSTRACT
A 71-year-old man was referred to our emergency department presenting with acute inferior and right ventricular myocardial infarction with cardiogenic shock. He developed ventricular fibrillation 80 minutes after arrival. Immediate defibrillation, mechanical ventilatory support with oxygenation, and inotropic agents were instituted. Despite restoration of sinus rhythm, his hypotension persisted. He promptly received intra-aortic balloon pump (IABP) counterpulsation and cardiac catheterisation. Coronary angiography revealed a subtotal occlusion of the left anterior descending coronary artery and complete occlusion of the right coronary artery. Since the right coronary artery was considered to be the infarct-related coronary artery, percutaneous coronary intervention (PCI) was carried out to the right coronary artery only. The patient was extubated and IABP was removed on the second and third admission day, respectively. He was discharged from the hospital eight days later. A second PCI to the left anterior descending coronary artery was performed successfully three weeks later. This case illustrates that in patients with acute myocardial infarction and cardiogenic shock, prompt application of IABP and PCI of the infarct-related coronary artery may be beneficial in reducing the catastrophic morbidity and mortality, especially in older patients.

Keywords: acute myocardial infarction, cardiogenic shock, intra-aortic balloon pump, percutaneous coronary intervention, ventricular fibrillation

INTRODUCTION
Acute myocardial infarction complicated by cardiogenic shock is a dramatic clinical condition with a high mortality rate. (1-3) Rapidly re-establishing blood flow of the infarct-related artery is essential in the management of patients with shock. (4) Although thrombolysis can be attempted and inotropic support instituted to augment the blood pressure with intra-aortic balloon pump (IABP), the greatest benefit is seen after urgent coronary angiography and revascularisation. (5) Immediate percutaneous coronary intervention (PCI) may reduce mortality in patients with cardiogenic shock after acute myocardial infarction. (6) Higher reperfusion rates can be achieved with direct PCI than with thrombolysis, and this should result in greater myocardial salvage and improved ventricular function. Early PCI also improved long-term outcomes in patients with cardiogenic shock after acute myocardial infarction. (7) We present a case of successful resuscitation with primary PCI and IABP in the setting of ventricular fibrillation and cardiogenic shock resulting from acute inferior wall and right ventricular myocardial infarction.

CASE REPORT
A 71-year-old man with a past history of intermittent exertional chest pain presented with a sudden onset of substernal squeezing pain with radiation to the lower jaw and shoulders. He developed diaphoresis, shortness of breath, and palpitation concomitantly with chest pain. A 12-lead electrocardiogram showed evidence of acute inferior myocardial infarction associated with right ventricular infarction. The patient was then referred to our emergency department. Vital signs showed a body temperature of 36.2°C, a pulse rate of 86 beats/minute, a respiratory rate of 24 breaths/minute, and a blood pressure of 98/58 mmHg. Pertinent physical findings in the emergency room included engorged jugular veins and S4 gallop. A 12-lead electrocardiogram showed evidence of acute inferior myocardial infarction associated with right ventricular infarction. The patient was then referred to our emergency department. Vital signs showed a body temperature of 36.2°C, a pulse rate of 86 beats/minute, a respiratory rate of 24 breaths/minute, and a blood pressure of 98/58 mmHg. Pertinent physical findings in the emergency room included engorged jugular veins and S4 gallop. A 12-lead electrocardiogram showed evidence of acute inferior myocardial infarction associated with right ventricular infarction. The patient was then referred to our emergency department. Vital signs showed a body temperature of 36.2°C, a pulse rate of 86 beats/minute, a respiratory rate of 24 breaths/minute, and a blood pressure of 98/58 mmHg.
Right-sided electrocardiogram revealed significant ST-segment elevation at V4. An acute inferior wall and right ventricular myocardial infarction together with old anteroseptal infarction were diagnosed. Electrocardiographical monitor showed intermittent high degree atrioventricular block, which responded to intravenous atropine administration temporarily. Chest radiograph revealed pulmonary congestion with redistribution and borderline cardiomegaly.

The patient received aspirin, heparin, and glycoprotein IIb/IIIa inhibitor (Tirofiban). About 80 minutes after arriving at our emergency department, the patient suddenly became unresponsive and in the meantime, electrocardiographical monitor showed ventricular fibrillation. Cardiac defibrillation was performed immediately, resulting in the restoration of a sinus rhythm. Endotracheal intubation and mechanical ventilation were applied, following successful defibrillation. His systolic blood pressure was 60 mmHg. Intravenous normal saline repletion and inotropic agents were initiated. The patient was rapidly transferred to the cardiac catheterisation room, where IABP and intravenous temporary pacing wire were instituted for maintaining proper haemodynamics. Coronary angiography revealed a total occlusion of the right coronary artery at the middle segment without collateral circulation (Fig. 2a) and a subtotal occlusion of left anterior descending coronary artery at middle segments with TIMI grade I-II flow (Fig. 3a). Left ventriculography showed marked hypokinesia of the mid- to apical inferior wall and mild hypokinesia of apical anterior segments presented with an ejection fraction of 28% and left ventricular end-diastolic pressure of 24 mmHg.

Primary PCI of the right coronary artery, which was considered to be the infarct-related artery, was undertaken. 2.5 mm × 20 mm and 3.5 mm × 20 mm Maverick balloon catheters (Boston Scientific, Maple Grove, MN, USA) were used to dilate the occluded lesion, and the angiographical result was optimal, with improved distal coronary flow to TIMI grade III (Fig. 2b). He regained consciousness soon after the PCI procedure. The blood pressure increased to 100/68 mmHg. The electrocardiographical monitor showed a sinus rhythm with a rate of 85 beats/minute, and he was sent to the intensive care unit for further management.

Serial laboratory analysis showed that the peak creatine kinase/CK-MB isoenzyme was 9427/575 U/L and troponin I was 368 ng/ml. His haemodynamics improved rapidly after intervention and therefore the Swan-Ganz catheter was not inserted. The endotracheal tube was removed on the second admission day and the IABP was removed on the third day. There was no evidence of ensuing shortness of breath, palpitation,
orthopnoea, or chest pain. He was transferred to the ordinary ward on the seventh day and was discharged on the eighth day. Three weeks later, he underwent elective PCI with successful stent deployment by using a 2.75 mm × 18 mm S660 stent and 3.0 mm × 24 mm S7 stent to the distal and middle segments of the left anterior descending coronary artery, respectively, which rebuilt the TIMI Grade III flow (Fig. 3b). He was then followed-up regularly at our hospital and remains event-free to date.

**DISCUSSION**

The incidence of cardiogenic shock in community studies has not decreased significantly over time. However, the extent of myocardial salvage from reperfusion treatment strategy decreases exponentially with time. Unfortunately, the harvest of reducing time from clinical presentation to hospital intervention delineates little progress over the past decade, and this perhaps accounts for the stagnant incidence of cardiogenic shock in community studies. The onset of cardiogenic shock in a patient following ST-segment elevation myocardial infarction heralds a dismal in-hospital prognosis. In the GUSTO-I trial, 7.2% of patients developed shock, which accounted for 58% of overall deaths in 30 days. Even with early revascularisation, almost 50% of patients die in 30 days.

The current American College of Cardiology/American Heart Association guidelines recommend the adoption of an early revascularisation strategy for patients less than 75 years old with cardiogenic shock. This improved survival has occurred in the setting of increased use of coronary revascularisation for patients with cardiogenic shock. The results of the SHOCK trial showed an overall benefit for patients with shock treated by a strategy of early revascularisation.

The major contribution to the success and popularity of PCI include stent utilisation with reduction of restenosis and potent antiplatelet therapies (aspirin and glycoprotein IIb/IIIa receptor inhibitors), which reduce procedure-related and long-term morbidity and mortality. Prevention should include early recognition and identification of the pre-shock state followed by treatment aimed at preventing deterioration into cardiogenic shock, i.e., relief of ischaemia, control of arrhythmias, optimisation of haemodynamic variables by inotropic support, and the administration of glycoprotein IIb/IIIa receptor inhibitors to improve angiographical outcomes of patients undergoing primary PCI. This patient received glycoprotein IIb/IIIa receptor inhibitors shortly after arrival at our emergency department. This facilitated regimen may be helpful for reperfusion in patients undergoing PCI treatment.

Application of IABP creates unloading of the pressure generated by the heart in systole and augmentation of diastolic pressure between cardiac contractions. Systolic unloading promotes left ventricular emptying and reduces wall tension by lowering both systolic pressure and volume. This decreased wall tension results in decreased myocardial oxygen consumption, increased stroke volume and cardiac output, and lowered left ventricular filling pressure and pulmonary capillary wedge pressure. Alleviation of left ventricular intramyocardial pressure decreases coronary arteriolar “closing pressure” and enhances myocardial perfusion. Diastolic augmentation induces
a retrograde flow, which may cause an increase in the coronary flow and cerebral flow. The improvement in coronary blood flow and decrease in myocardial oxygen consumption can reverse ischaemic left ventricular dysfunction, which can further improve systemic arterial pressure and cardiac output while lowering the pulmonary wedge pressure to help reverse pulmonary oedema. Several clinical studies have suggested an improved outcome for patients with acute myocardial infarction complicated by cardiogenic shock when they were treated with the combination of IABP and thrombolytic therapy. Augmentation of blood pressure with an IABP in this situation could facilitate thrombolysis by increasing coronary perfusion pressure. Patients with cardiogenic shock often manifest haemodynamic improvement with IABP; however, it is unclear if this translates into reduced morbidity or mortality in the absence of concomitant revascularisation. Consensus on optimal treatment continues to be hindered by a lack of randomised controlled trials. This patient received intervention of IABP and primary PCI of infarct-related coronary artery as soon as possible, leading to early restoration of coronary blood flow. The PCI procedure can increase the coronary artery perfusion, which is beneficial for rescue from the cardiogenic shock status. The recovery from cardiogenic shock in this patient demonstrated that aggressive intervention strategy, including the ventricular pacing, primary PCI, IABP, and glycoprotein IIb/IIIa receptor inhibitors infusion, may play a central role for the treatment of this kind of patients.

The coronary angiogram of our patient demonstrated multivessel disease. A recent study disclosed that in patients with multivessel disease and acute myocardial infarction, multivessel PCI is technically feasible, but is associated with higher rates of re-infarction, need for revascularisation and major adverse cardiac events, and offers no incremental benefits in mortality rate. In patients with multivessel disease and acute myocardial infarction, initial percutaneous revascularisation strategies should focus on the infarct-related artery. However, in patients with persistent cardiogenic shock, despite initial PCI of the infarct-related coronary artery, the PCI of the non-culprit but critically-stenosed artery should also be considered. Our patient’s clinical status was stabilised with the IABP and initial PCI of the culprit vessel. Elective PCI of the left anterior descending artery was performed later after treatment of the cardiogenic shock. This strategy may have prevented further complications resulting from acute closure of this vessel. In conclusion, primary PCI strategy associated with IABP support may be life-saving. More studies focusing on early diagnostic methods, pharmacological and mechanical haemodynamic supports, and refinement of revascularisation techniques are crucial for the management of patients with cardiogenic shock.

REFERENCES