Complications of Acute Myocardial Infarction
—Diagnosis and Treatment—
JMAJ 45(4): 149–154, 2002

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Abstract: For the past 20 years, the in-hospital mortality rate of acute myocardial infarction has significantly decreased to less than 10%. This reduction can be attributed mainly to the development of acute-phase treatment such as reperfusion therapy. However, cardiogenic shock and cardiac rupture dominate more than 70% of the causes of death. CCUs have made efforts to treat these problems whose mortality rate is still high. For cardiogenic shock, maintenance of circulation followed by reperfusion therapy is necessary before it causes irreversible impairment of organ function. For cardiac rupture, a critical care system should be established to implement emergency surgical intervention following the early detection of cardiac tamponade, in addition to the emphasis on prevention in high-risk populations for cardiac rupture.

Key words: Acute myocardial infarction; Cardiogenic shock; Cardiac rupture; Mortality; Complication

Introduction

The mortality rate of acute myocardial infarction in our CCUs has decreased from 12% in the early period (before the era of reperfusion therapy, 1977–1986), through 8% in the middle period (thrombolytic treatment widely adopted, 1987–1992), to 5% in the late period [PTCA (percutaneous transluminal coronary angioplasty) adopted as the main reperfusion therapy, 1993–1998]. The improved prognosis can be attributed to the progress of treatment of complications of acute myocardial infarction. Pump failure and cardiac rupture (including ventricular septal rupture) dominate approximately 70% of the causes of death in the acute period. Prognosis has been improved, but the mortality rate is still high. Therefore, CCUs are making efforts to improve treatment effectiveness.1

The diagnosis and treatments against the two main causes of death will be discussed along with the major complications in the acute period.
Cardiogenic Shock

1. Definitions (Table 3)

Pump failure is defined as the condition in which cardiac output is insufficient to perfuse various body organs because of acute left ventricular contractile dysfunction caused by myocardial infarction. Cardiogenic shock is defined as myocardogenic shock, except for when it is caused by underlying diseases before the onset of infarction, mechanical complications, and arrhythmia. Shock includes not only a low arterial blood pressure, but also severe, prolonged rate would not improve without reperfusion even when using assisted circulation such as IABP (intra-aortic balloon pumping).

Evaluation of the Severity

The classic Killip classification, one of the available methods for evaluating the severity of acute myocardial infarction, is still helpful for the estimation of prognosis. As shown in Table 2, the severity can be easily judged from physical findings according to the Killip classification. Killip et al. reported that the mortality rate increased with the rise of the type of the disease, and that mortality rate in class IV (cardiogenic shock) was as high as 81%. In our facility, the mortality rate was the same as that of Killip-class IV before the era of reperfusion therapy (Fig. 2), suggesting that the mortality rate would not improve without reperfusion even when using assisted circulation such as IABP (intra-aortic balloon pumping).

Table 1 Major Complications of Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Complication</th>
<th>Year</th>
<th>Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pump failure (cardiogenic shock, heart failure)</td>
<td>1977</td>
<td>12%</td>
</tr>
<tr>
<td>Cardiac rupture (free wall rupture, ventricular septal rupture, papillary muscle rupture)</td>
<td>1987</td>
<td>8%</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>1993</td>
<td>5%</td>
</tr>
<tr>
<td>Post-infarction angina</td>
<td></td>
<td></td>
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<tr>
<td>Right ventricular infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pericarditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular thrombus and complicated embolism</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Severity Classification of Acute Myocardial Infarction (Killip Classification)

<table>
<thead>
<tr>
<th>Killip class</th>
<th>Mortality rate in the original article</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I: no sign of CHF (no rales in lung)</td>
<td>6%</td>
</tr>
<tr>
<td>Type II: pulmonary congestion limited to basal lung segments (bi basilar rales)</td>
<td>17%</td>
</tr>
<tr>
<td>Type III: acute pulmonary edema (rales in more than 50% of lung)</td>
<td>38%</td>
</tr>
<tr>
<td>Type IV: cardiogenic shock</td>
<td>81%</td>
</tr>
</tbody>
</table>

CHF: congestive heart failure

listed in Table 1.
tissue hypoperfusion.3)

2. Treatment strategies4)

Because impairment of organ function caused by shock rapidly develops and becomes irreversible within a short period, the most suitable treatment should be adopted as early as possible. Prolonged tissue hypoperfusion makes prognosis worse, leading to death from multiple organ failure even if hemodynamics is improved. Immediately after admission, respiratory control should be implemented for significant hypoxemia with the administration of catecholamines. If 5–20 μg/kg/min of dopamine cannot achieve pressor effects, 5–20 μg/kg/min of dobutamine (DOB), or 0.05–0.2 μg/kg/min of noradrenaline (NAd) should be administered.

At the same time, the femoral artery and vein should be secured with a 5 Fr sheath to enable use of assisted device at any time. Puncturing is often difficult if the femoral artery is not palpable. In principle, the internal jugular or subclavian vein should not be punctured in case of shock. The reason is that there are cases in which even one or two mispunctures can lead to fatal bleeding because of thrombolytic therapy or heparinization. If systolic pressure does not rise to more than 80 mmHg within 15 minutes, assisted device should be implemented.

Treatment requiring a combination of reperfusion therapy and maintenance of peripheral circulation is continued while in transit from the emergency room (or CCU) to the catheterization room. The reperfusion method of choice is PTCA. Prolonged shock with unavailability of a catheterization room should be treated with intravenous thrombolysis.

3. Assisted devices

(1) **Intra-aortic balloon pumping (IABP):**

The results of IABP treatment without reperfusion therapy for cardiogenic shock in acute myocardial infarction support IABP: intra-aortic balloon pumping, PCPS: percutaneous cardiopulmonary system, PTCA: percutaneous transluminal coronary angioplasty.
myocardial infarction have not been encouraging. However, IABP is still an indispensable assisted device method when combined with reperfusion therapy. If systolic pressure is less than 80 mmHg, and cardiac index is less than 2.2 l/min/m² even with catecholamines, IABP should be used. IABP serves as a pressure-support device, supporting only 15% of cardiac output. Therefore, if cardiac output by own beat cannot be expected, the following PCPS should be used.

(2) Percutaneous cardiopulmonary support system (PCPS): PCPS is a method in which an artificial cardiopulmonary device enabling assisted circulation can be made available by inserting a cannula percutaneously. Perfusion is maintained by delivering oxygen to venous blood derived from right atrium by means of an artificial lung, and by delivering the blood retrogradually into the femoral artery (Fig. 4). It is possible to deliver blood at 2–4 l/min. If systolic pressure is less than 50 mmHg, or repeated ventricular fibrillation or tachycardia requiring cardioversion is observed, PCPS should be used, rather than IABP. To support coronary perfusion, it should be combined with IABP.

Because PCPS should be implemented quickly, our CCU is always equipped with PCPS with a circuit organized in sanitary conditions. In order to enable a few night staff members to deal with it, every nurse has routine training to complete circuit filling within 5 minutes as a part of lessons on cardio-pulmonary resuscitation.

If these types of assisted circulation and reperfusion are insufficient, the use of a left ventricular assist device to take care of part of the cardiac pumping function should be considered. Because reduced cardiac function will be prolonged even after successful reperfusion, assisted circulation should be continued. Intensive care is required to maintain assisted circulation for a long period, and to prevent complications such as infection, bleeding, and lower limb ischemia.

4. Efficacy of treatments for Cardiogenic shock

In cardiogenic shock, tissue hypoperfusion and acute myocardial infarction should be treated at the same time. Failing in either treatment would be lethal. Reperfusion of occluded coronary artery is essential to improve prognosis. Moreover, it is important to achieve reperfusion within a short period from the development of shock. If reperfusion cannot be achieved, the mortality rate is as high as 80% even with
assisted device (Fig. 2). If a major vessel causing occlusion is reperfused irrespective of the type of reperfusion therapy, the in-hospital mortality rate is less than 40%, suggesting the importance of successful reperfusion.

A guideline of the ACC/AHA (American College of Cardiology/American Heart Association) strongly recommends the use of PTCA. If PTCA is not available, or if it takes over 30 minutes to start it, intravenous infusion of tissue plasminogen activator (t-PA) should be performed. However, because the mortality rate of cardiogenic shock is still high, new therapies such as myocardial protection methods should be reviewed in addition to reperfusion therapy.

Cardiac Rupture

Cardiac rupture occurs through an area of transmural neurosis and expansion in the acute phase of myocardial infarction. It is classified as left ventricular free wall rupture, ventricular septal perforation or papillary muscle rupture, depending on the site of the lesions. The incidence of rupture shows two peaks: within 24 hours after the onset of infarction, and one week later. The mortality rate of free wall rupture is high. It is one of the concerns for CCUs in parallel with death from pump failure. The frequency is low, but it does have a noticeable incidence. The frequency of the oozing type has been increasing compared with the sudden rupture type, which is lethal and requires preventive measures.

1. Free wall rupture

In cases of successful reperfusion, the second peak of cardiac rupture is reported to decrease, while the possibility of increased cardiac rupture caused by thrombolytic therapy is pointed out. Therefore, for patients over the age of 70 complicated with hypertension, PTCA is often the reperfusion method used.

Attention should be paid to suppress systolic pressure under 120mmHg in the acute phase of myocardial infarction, and under 20mmHg increase in the rehabilitation. Because patients showing significant retention of pericardial effusion in echocardiogram over time and patients with rapidly thinning of the infarction wall are highly susceptible to cardiac rupture, they are carefully monitored and the rehabilitation process is delayed.

An oozing type of rupture with gradual retention of bloody pericardial effusion leading to cardiac tamponade can be diagnosed by echocardiography before the patient goes into shock. Cardiac tamponade can be treated by performing a surgical repair after drainage of pericardial cavity. A sudden rupture, which immediately leads to electro-mechanical dissociation, is lethal. It is treated by PCPS immediately after the rupture to secure general circulation before stepping up, but the prognosis for sudden rupture is unfavorable.

2. Ventricular septal perforation

Like free wall rupture, the peak incidence of septal rupture appears to be within a week after infarction. When a new, harsh, loud holosystolic murmur is heard between the third and the fourth intercostal of the left parasternum, ventricular septal perforation is strongly suspected. It can be diagnosed by confirming the existence of a left-to-right interventricular shunt using two-dimensional echocardiography, and by confirming a significant increase in oxygen saturation from the right atrium to pulmonary artery. The frequency of ventricular septal perforation in our facility is approximately 1% of all myocardial infarction. Out of 33 patients, 18 underwent an operation, and 12 of them survived and were discharged from the hospital. Only 2 out of 15 patients who did not undergo surgery survived and were discharged from the hospital. Therefore, the operation should be performed whenever possible. Especially for shock, early surgical intervention is essential.

3. Papillary muscle rupture

Severe mitral regurgitation occurs after acute myocardial infarction in papillary muscle rup-
ture, rapidly progressing to shock from left-sided heart failure. As cardiac murmur associated with mitral regurgitation is brief or even completely absent in patients with reduced cardiac function, echocardiography is an effective diagnostic tool. Because significant mitral regurgitation is an indication to perform early surgical correction, valve replacement or repair should be performed.

**Conclusion**

The prognosis for acute myocardial infarction after admission has improved for the past 20 years because of the progress in treatments such as reperfusion therapy. However, as mentioned in the present paper, the mortality rate of cardiogenic shock and cardiac rupture is still high, and further efforts should be made to improve treatment effectiveness. The mortality rate before admission is higher, and 30–50% is estimated to die outside the hospital. Establishment of a cardiovascular critical care system is urgently required to reduce the general mortality of acute myocardial infarction.

**REFERENCE**