ABC of interventional cardiology

Percutaneous coronary intervention: cardiogenic shock

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Cardiogenic shock is the commonest cause of death after acute myocardial infarction. It occurs in 7% of patients with ST segment elevation myocardial infarction and 3% with non-ST segment elevation myocardial infarction.

Cardiogenic shock is a progressive state of hypotension (systolic blood pressure <90 mm Hg) lasting at least 30 minutes, despite adequate preload and heart rate, which leads to systemic hypoperfusion. It is usually caused by left ventricular systolic dysfunction. A patient requiring drug or mechanical support to maintain a systolic blood pressure over 90 mm Hg can also be considered as manifesting cardiogenic shock. As cardiac output and blood pressure fall, there is an increase in sympathetic tone, with subsequent cardiac and systemic effects—such as altered mental state, cold extremities, peripheral cyanosis, and urine output <30 ml/hour.

Effects of cardiogenic shock

Cardiac effects

In an attempt to maintain cardiac output, the remaining non-ischaemic myocardium becomes hypercontractile, and its oxygen consumption increases. The effectiveness of this response depends on the extent of current and previous left ventricular damage, the severity of coexisting coronary artery disease, and the presence of other cardiac pathology such as valve disease.

Three possible outcomes may occur:

- Compensation—which restores normal blood pressure and myocardial perfusion pressure
- Partial compensation—which results in a pre-shock state with mildly depressed cardiac output and blood pressure, as well as an elevated heart rate and left ventricular filling pressure
- Shock—which develops rapidly and leads to profound hypotension and worsening global myocardial ischaemia. Without immediate reperfusion, patients in this group have little potential for myocardial salvage or survival.

Systemic effects

The falling blood pressure increases catecholamine levels, leading to systemic arterial and venous constriction. In time, activation of the renin-aldosterone-angiotensin axis causes further vasoconstriction, with subsequent sodium and water retention. These responses have the effect of increasing left ventricular filling pressure and volume. Although this partly compensates for the decline in left ventricular function, a high left ventricular filling pressure leads to pulmonary oedema, which impairs gas exchange. The ensuing respiratory acidosis exacerbates cardiac ischaemia, left ventricular dysfunction, and intravascular thrombosis.

Time course of cardiogenic shock

The onset of cardiogenic shock is variable. In the GUSTO-I study, of patients with acute myocardial infarction, 7% developed cardiogenic shock—11% on admission and 89% in the subsequent two weeks. Almost all of those who developed cardiogenic shock did so by 48 hours after the onset of symptoms, and their overall 30 day mortality was 57%, compared with an overall study group mortality of just 7%.

A 65 year old man with a 3–4 hour history of acute anterior myocardial infarction had cardiogenic shock and acute pulmonary oedema, requiring mechanical ventilation and inotropic support. He underwent emergency angiography (top), which showed a totally occluded proximal left anterior descending artery (arrow). A soft tipped guidewire was passed across the occlusive thrombotic lesion, which was successfully stented (middle). Restoration of brisk antegrade flow down this artery (bottom) followed by insertion of an intra-aortic balloon pump markedly improved blood pressure and organ perfusion. The next day he was extubated and weaned off all inotropic drugs, and the intra-aortic balloon pump was removed.

Cardiac compensatory response to falling cardiac output after acute myocardial infarction.

Differential diagnosis

Hypotension can complicate acute myocardial infarction in other settings.

Right coronary artery occlusion

An occluded right coronary artery (which usually supplies a smaller proportion of the left ventricular muscle than the left coronary artery) may lead to hypotension in various ways: cardiac output can fall due to vagally mediated reflex venodilatation and bradycardia, and right ventricular dilation may displace the intraventricular septum towards the left ventricular cavity, preventing proper filling.

In addition, the right coronary artery occasionally supplies a sizeable portion of left ventricular myocardium. In this case right ventricular myocardial infarction produces a unique set of physical findings, haemodynamic characteristics, and ST segment elevation in lead V₄R. When this occurs aggressive treatment is indicated as the mortality exceeds 30%.

Ventricular septal defect, mitral regurgitation, or myocardial rupture

In 10% of patients with cardiogenic shock, hypotension arises from a ventricular septal defect induced by myocardial infarction or severe mitral regurgitation after papillary muscle rupture. Such a condition should be suspected if a patient develops a new systolic murmur, and is readily confirmed by echocardiography—which should be urgently requested. Such patients have high mortality, and urgent referral for surgery may be needed. Even with surgery, the survival rate can be low.

Myocardial rupture of the free wall may cause low cardiac output as a result of cardiac compression due to tamponade. It is more difficult to diagnose clinically (raised venous pressure, pulsus paradoxus), but the presence of haemopericardium can be readily confirmed by echocardiography. Pericardial aspiration often leads to rapid increase in cardiac output, and surgery may be necessary.

Management

The left ventricular filling volume should be optimised, and in the absence of pulmonary congestion a saline fluid challenge of at least 250 ml should be administered over 10 minutes. Adequate oxygenation is crucial, and intubation or ventilation should be used early if gas exchange abnormalities are present. Ongoing hypotension induces respiratory muscle failure, and this is prevented with mechanical ventilation. Antithrombotic treatment (aspirin and intravenous heparin) is appropriate.

Supporting systemic blood pressure

Blood pressure support maintains perfusion of vital organs and slows or reverses the metabolic effects of organ hypoperfusion. Inotropes stimulate myocardial function and increase vascular tone, allowing perfusion pressures to increase. Intra-aortic balloon pump counterpulsation often has a dramatic effect on systemic blood pressure. Inflation occurs at end diastole, greatly increasing aortic diastolic pressure to levels above aortic systolic pressure. In addition, balloon deflation during the start of systole reduces the aortic pressure, thereby decreasing myocardial oxygen demand and forward resistance (afterload).

Reperfusion

Although inotropic drugs and mechanical support increase systemic blood pressure, these measures are temporary and have no effect on long term survival unless they are combined with coronary artery recanalisation and myocardial reperfusion.

Hallmarks of right ventricular infarction

- Rising jugular venous pressure, Kussmaul sign, pulsus paradoxus
- Low output with little pulmonary congestion
- Right atrial pressure > 10 mm Hg and > 80% of pulmonary capillary wedge pressure
- Right atrial prominent Y descent
- Right ventricle shows dip and plateau pattern of pressure
- Profound hypoxia with right to left shunt through a patent foramen ovale
- ST segment elevation in lead V₄R

Main indications and contraindications for intra-aortic balloon pump counterpulsation

Indications

- Cardiogenic shock
- Unstable and refractory angina
- Cardiac support for high risk percutaneous intervention
- Hypoperfusion after coronary artery bypass graft surgery
- Septic shock
- Enhancement of coronary flow after successful recanalisation by percutaneous intervention
- Ventricular septal defect and papillary muscle rupture after myocardial infarction
- Intractable ischaemic ventricular tachycardia

Contraindications

- Severe aortic regurgitation
- Abdominal or aortic aneurysm
- Severe aorto-iliac disease or peripheral vascular disease

Diagram of intra-aortic balloon pump (left) and its position in the aorta (right)

Effects of intra-aortic balloon pump during systole and diastole

Thrombolysis is currently the commonest form of treatment for myocardial infarction. However, successful fibrinolysis probably depends on drug delivery to the clot, and as blood pressure falls, so reperfusion becomes less likely. One study (GISSI) showed that, in patients with cardiogenic shock, streptokinase conferred no benefit compared with placebo.

The GUSTO-I investigators examined data on 2200 patients who either presented with cardiogenic shock or who developed it after enrolment and survived for at least an hour after its onset. Thirty day mortality was considerably less in those undergoing early angiography (38%) than in patients with late or no angiography (62%). Further analysis suggested that early angiography was independently associated with a 43% reduction in 30 day mortality.

In the SHOCK trial, patients with cardiogenic shock were treated aggressively with inotropic drugs, intra-aortic balloon pump counterpulsation, and thrombolytic drugs. Patients were also randomised to either coronary angiography plus percutaneous intervention or bypass surgery within six hours, or medical stabilisation (with revascularisation only permitted after 54 hours). Although the 30 day primary end point did not achieve statistical significance, the death rates progressively diverged, and by 12 months the early revascularisation group showed a significant mortality benefit (55%) compared with the medical stabilisation group (70%). The greatest benefit was seen in those aged <75 years and those treated early (<6 hours). Given an absolute risk reduction of 15% at 12 months, one life would be saved for only seven patients treated by aggressive, early revascularisation.

Support and reperfusion: impact on survival

Over the past 10 years, specific measures to improve blood pressure and restore arterial perfusion have been instituted. Mortality data collected since the 1970s show a significant fall in mortality in the 1990s corresponding with increased use of combinations of thrombolytic drugs, the intra-aortic balloon pump, and coronary angiography with revascularisation by either percutaneous intervention bypass surgery. Before these measures, death rates of 80% were consistently observed.

Cardiogenic shock is the commonest cause of death in acute myocardial infarction. Although thrombolysis can be attempted with inotropic support or augmentation of blood pressure with the intra-aortic balloon pump, the greatest mortality benefit is seen after urgent coronary angiography and revascularisation. Cardiogenic shock is a catheter laboratory emergency.

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The diagram of patient mortality after myocardial infarction is adapted with permission from Goldberg RJ et al, *N Engl J Med* 1999;340:1162-8.

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Further reading

- Hochman JS, Sleeper LA, Webb JG, Sanborn TA, White HD, Talley JD, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med* 1999;341:625-34
- Berger PB, Holmes DR Jr, Stebbins AL, Bates ER, Califf RM, Topol EJ. Impact of an aggressive invasive catheterization and revascularization strategy on mortality in patients with cardiogenic shock in the global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries (GUSTO-I) trial. *Circulation* 1997;96:122-7

Diagram of electrocardiogram and aortic pressure wave showing timing of intra-aortic balloon pump and its effects of diastolic augmentation (D) and reduced aortic end diastolic pressure

Aortic pressure wave recording before (left) and during (right) intra-aortic balloon pump counterpulsation in a patient with cardiogenic shock after myocardial infarction. Note marked augmentation in diastolic pressure (arrow A) and reduction in end diastolic pressures (arrow B). (AO=aortic pressure)

Mortality after myocardial infarction with or without cardiogenic shock (1975 to 1997). Mortality of patients in shock fell from roughly 80% to 60% in the 1990s

Names of trials

- GISSI—Gruppo Italiano per lo studio della sopravvivenza nell'infarto miocardico
- GUSTO—global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries
- SHOCK—should we emergently revascularize occluded coronaries for cardiogenic shock

- Golberg RJ, Samad NA, Yarzebski J, Gurwitz J, Bigelow C, Gore JM. Temporal trends in cardiogenic shock complicating acute myocardial infarction. *N Engl J Med* 1999;340:1162-8
- Hasdai D, Topol EJ, Califf RM, Berger PB, Holmes DR. Cardiogenic shock complicating acute coronary syndromes. *Lancet* 2000;356:749-56
- White HD. Cardiogenic shock: a more aggressive approach is now warranted. *Eur Heart J* 2000;21:1897-901