Part VIII: Shock

Chapter 59: Cardiogenic Shock

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Although myocardial function may be impaired in any shocked patient, the term "cardiogenic shock" is reserved for patients with shock caused by heart disease. It is a clinical syndrome which is defined as:

1. A systolic arterial pressure less than 90 mmHg (12 kPa);

2. Evidence of reduced blood flow as shown by:
(a) Urine output less than 20 mL/h;
(b) Impaired mental function; and
(c) Peripheral vasoconstriction with a cold, clammy skin.

Hypotension resulting from pain, vasovagal reaction, serious arrhythmias, drug reaction or hypovolaemia is excluded. In patients with known hypertension, a reduction in the blood pressure of 30 mmHg (4 kPa) is used to define hypotension. Occasionally, urine output or cerebral function is preserved in cardiogenic shock.

Other changes seen in cardiogenic shock include reduced coronary blood flow, myocardial lactate production, abnormally high myocardial oxygen extraction, and an intense neurohumoral stress response. There are big increases in plasma concentrations of adrenaline and noradrenaline, glucose, free fatty acids, cortisol, renin, angiotensin II and glucagon, and decreased concentrations of insulin.

Aetiology (Table 1)

The most common cause of cardiogenic shock is myocardial infarction (MI). It affects up to 15% of those admitted to hospital with an acute infarct and is the commonest cause of hospital death. Factors associated with an increased risk of cardiogenic shock are age, diabetes, previous MI and anterior (rather than inferior) infarction. Myocardial dysfunction after cardiac surgery is considered in Chapter 15, Postoperative Intensive Care. Patients with severe cardiomyopathy suitable for heart transplant may need admission to an ICU for assessment and support while awaiting a donor heart.

Pathophysiology

Autopsy studies show that over 40% of functioning myocardium has been lost in patients who die of cardiogenic shock after MI. The effects of recent and old infarction are additive. Extension of the area of infarction during a single hospital admission is common in patients who develop cardiogenic shock. This may represent loss of viability of an ischaemic zone surrounding the initial infarct. The neurohumoral changes seen in cardiogenic shock, and to a lesser extent after aby infarct, cause an increase in heart rate, contractility and blood pressure.

Table 1. *Causes of Cardiogenic Shock*

Myocardial infarction
Myocardial dysfunction after cardiac surgery
Myocardial contusion by direct chest trauma
Reduced systemic flow from left ventricular contraction
- rupture of the interventricular septum
- acute mitral regurgitation
- acute aortic regurgitation
- left ventricular aneurysm

Obstruction to flow
- myxoma
- hypertrophic obstructive cardiomyopathy

Cardiomyopathy
Myocarditis
Heart transplant rejection

The clinical findings of cardiogenic shock occur when the cardiac index is less than 1.8 L/min/m^2. Heart rate and systemic vascular resistance (SVR) increase to try and maintain arterial pressure. The neurohumoral responses and these reflex changes increase myocardial oxygen demand at a time when myocardial perfusion is reduced by hypotension. The result is to further jeopardize ishaemic but potentially viable tissue and further impair left ventricular function, completing a vicious circle which ends with the patient's death.

In cardiogenic shock caused by septal perforation, ventricular aneurysm, or acute valvular regurgitation, there is a reduction of flow through the systemic circulation because a proportion of the blood ejected from the left ventricle is directed into the right ventricle, the aneurysm, the left atrium or back into the left ventricle.

Clinical Presentation

The clinical features are in keeping with the definition of cardiogenic shock above. Arrhythmias and the extremes of heart rate, cardiac tamponade, pulmonary embolism and other causes of shock must be excluded. Most patients with cardiogenic shock also have pulmonary oedema with raised left ventricular end diastolic pressure (LVEDP) and pulmonary capillary wedge pressure (PCWP). Pulmonary oedema may cause severe dyspnoea, central cyanosis and crepitations. The diagnosis of pulmonary oedema can be confirmed by an erect chest X-ray.

Less often, cardiogenic shock occurs without the PCWP being elevated. This group includes:

1. Patients with clinical and radiological evidence of pulmonary oedema, but normal or low PCWP. This can be a result of diuretic therapy or plasma volume depletion by the fluid lost into the lungs.

2. Patients with relative hypovolaemia, ie, below the risk level of pulmonary oedema (18-20 mmHg, 2.4-2.7 kPa).

3. Patients with significant right ventricular infarction. Hypotension in patients with inferior infarction and the clinical signs of right heart failure should alert to this possibility, although the signs are more specific than sensitive. Right ventricular infarction can also mimic pericardial tamponade or pericardial constriction.

Patients presenting with these three syndromes are a minority of those with cardiogenic shock, but it is important to identify them. They may improve dramatically with plasma volume expansion and deteriorate if diuretics are given inappropriately.

A systolic murmur appearing after MI suggests mitral regurgitation or septal perforation. Clinical distinction between the two is difficult. Patients with infarction who respond to volume expansion or who have a mechanical lesion amenable to surgery, have potentially reversible cardiogenic shock. When shock is due solely to extensive left ventricular infarction it is usually irreversible. If cardiogenic shock fails to respond to treatment, multiple organ failure, metabolic acidosis, hypothermia and coma may precede death.

Management

The treatment of cardiogenic shock is difficult and the results often disappointing. Efforts have therefore been made to reduce the incidence of cardiogenic shock by techniques that will preserve myocardium during cardiopulmonary bypass and limit the amount damaged by MI. Studies in acute MI suggest improvement in global left ventricular function in patients treated with angioplasty and decreased mortality in patients given 1.5 MU of IV streptokinase. However, a consensus on the best management plan and the timing of its components has still to be reached. What is clear, is that for patients to benefit from active management of acute coronary occlusion, treatment must occur within the first few hours from the onset of symptoms.

If cardiogenic shock becomes established, management consists of general supportive measures, characterization of haemodynamic problems, mesures to optimize the myocardial oxygen supply:demand ratio and improve the haemodynamic disturbance, and specific treatment when possible.

1. General Supportive Measures

These include relief of pain, correction of severe acid base or electrolyte disturbances, and treatment of cardiac arrhythmias. Patients with bradyarrhythmias causes by atrioventricular block or junctional bradycardia may respond dramatically to sequential atrioventricular or atrial pacing. Oxygen is given to correct hypoxaemia. If hypoxaemia persists in patients with potentially reversible shock, continuous positive airway pressure (CPAP) or tracheal intubation and mechanical ventilation with positive end expiratory pressure (PEEP) may be used. The copious frothy sputum of severe pulmonary oedema is controlled by increasing PEEP rather than repeated endotracheal suction. Patients with cardiogenic shock may, however, become profoundly hypotensive when given sedation for intubation, and this can be exacerbated by the effects of positive pressure ventilation on the circulation.

2. Monitoring

The principle and methods for monitoring any critically ill patients apply to cardiogenic shock. These should include:

(a) Measurement of intra-arterial blood pressure.

(b) Assessment of venous pressures: direct measurement of central venous pressure is more accurate than clinical assessment of the jugular venous pressure, but only reflects the right ventricular filling pressure. Pulmonary oedema may be diagnosed by hearing wide spread crepitations and confirmed by the chest radiograph, but the most accurate assessment of left ventricular filling pressure is gained by measuring the PCWP.

(c) Assessment of cardiac output: while skin perfusion, hourly urine output and mental state provide clinical guides to cardiac output, changes in cardiac output with time or treatment are best measured directly bu the haemodilution method.

(d) Other investigations:

(i) Electrocardiogram and cardiac enzymes to confirm a diagnosis of MI.

(ii) Blood gases, pH, and plasma lactate.

(iii) Haemoglobin, electrolytes, urea, and creatinine.

Insertion of a pulmonary artery balloon flotation (Swan-Ganz) catheter helps to characterize the haemodynamic problems. When cardiogenic shock is caused by septal perforation, blood sampling during insertion of the catheter will show an increase in oxygen saturation between the right atrium and the right ventricle. Patients with severe mitral regurgitation usually have a large V-wave on the wedge pressure trace, but this is nonspecific; large V-waves are also seen in the absence of mitral regurgitation.

Echocardiography provides information about lesions of the valves, septum and overall ventricular function, and also helps to exclude rarer causes of shock such as an obstructing atrial myxoma.

3. Improvement of Haemodynamic Disturbance

The same principles can be applied in cardiogenic shock from all causes. The overall prognosis should be carefully considered before starting invasive monitoring and aggressive support, especially in patients with large myocardial infarcts without a correctable lesion and patients with severe cardiomyopathy.

PAWP < 18 mmH (2.4 kPa)

Plasma volume expansion is the initial treatment. Although a left ventricular filling pressure of 18 mmHg (2.4 kPa) is greater than normal, this is usually the optimum for ventricular performance in patients with recent myocardial infarcts and anything less is

consistent with hypovolaemia. Blood should be used to correct anaemia, otherwise repeated fluid challenges with 100-200 mL of colloid should be used.

In patients with predominant right ventricular infarction, fluid loading may produce a CVP of 30 mmHg (4 kPa) or more. Conversely, drugs conventionally used in the treatment of acute MI which reduce preload, exacerbate hypoperfusion in these patients.

PAWP > 18 mmHg (2.4 kPa)

This group contains most patients with cardiogenic shock, including those who initially have low PCWPs and are still hypotensive after fluid loading. The treatments available include drugs and mechanical circulatory assistance.

(a) *Drug Therapy*

(i) *Catecholamines*, including adrenaline, noradrenaline, isoprenaline, dopamine and dobutamine. The aim of these drugs is to increase arterial pressure and improve coronary perfusion. They do this by varying degrees of increased cardiac contractility and rate (beta-adrenergic stimulation) and peripheral vasoconstriction (alpha-adrenergic effect). The increased arterial pressure is, however, at the expense of increased myocardial oxygen consumption which may endanger additional areas of myocardium. Other side effects include potentiation of arrhythmias by beta stimulation and a reduction in renal blood flow by alpha-mediated vasoconstriction.

Dopamine may have advantages over other catecholamines. At low infusion rates of 0.5-5 microg/kg/min the main effect is renal and mesenteric vasodilation mediated by dopaminergic receptors. Infusion rates of 5-20 microg/kg/min cause increased beta-adrenergic stimulation, and above 20 microg/kg/min alpha-adrenergic stimulation and vasoconstriction become increasingly prominent.

Dobutamine acts mainly on beta-1-adrenergic receptors. In the majority of patients, it has little effect on SVR or heart rate in lower doses. Reduction of venous pressures and SVR may, however, cause hypotension and tachycardia in some patients with shock. The range of infusion rates is 2-40 microg/kg/min. Comparisons in patients with a low cardiac output between dopamine and dobutamine have shown greater haemodynamic benefit from dobutamine, but there is considerable variation between patients. When low cardiac output complicates recovery from cardiac surgery, there can be considerable changes in the response to an inotropic drug with time.

(ii) *Digoxin* has been used to improve myocardial contractility but its relatively slow onset of action, long half-life and low therapeutic ratio make it less suitable as an inotropic in cardiogenic shock.

(iii) *Vasodilators* used in cardiogenic shock include sodium nitroprusside, nitroglycerin, isosorbide dinitrate, salbutamol and angiotensin converting enzyme (ACE) inhibitors. The potential benefits are: a reduction in myocardial work and oxygen demand; pooling of blood on the venous side of the circulation with a reduction in atrial pressures to promote clearing

of pulmonary oedema; and possibly a redistribution of blood flow within organs and tissues to improve the supply of oxygen and metabolites at cellular level. The greatest danger is a precipitous fall in arterial pressure which reduces coronary perfusion. Invasive haemodynamic monitoring is therefore essential. Vasodilators also tend to increase hypoxaemia by increasing intrapulmonary shunting.

Sodium nitroprusside infusion consistently increases cardiac output in patients with left ventricular failure and shock after MI. An initial dose of 10 microg/min should be increased slowly to a maximum of 500 microg/min above which cyanide toxicity becomes a hazard of prolonged infusion. Patients with cardiogenic shock are usually quite sensitive to its effect.

Nitroglycerine is predominantly a venodilator when given by intravenous infusion. The rapid onset of action is useful in the management of pulmonary oedema. The acute haemodynamic effects of intravenous isosorbide dinitrate in MI are very similar.

Salbutamol is a relatively specific beta-2-adrenergic receptor agonist. Its main effect is arteriolar dilation. In patients with cardiogenic shock after infarction, infusions of 10-40 microg/min increased cardiac output without affecting PCWP.

Impressive results were reported in a small group of patients with cardiogenic shock given the ACE inhibitor captopril. More often, these drugs are used to treat residual cardiac failure in patients being weaned off intravenous drugs.

(iv) *Other drugs:* steroids, glucagon and glucose-insulin-potassium infusions have been used in patients with cardiogenic shock. They are generally considered ineffective. The role of newer vasoactive drugs such as amrinone, enoximone and dopexamine in cardiogenic shock has still to be defined.

A combination of catecholaminae and vasodilator is often found to produce the best haemodynamic response.

(b) *Mechanical Circulatory Assistance*

This should be considered for patients who remain hypotensive and shocked after a short trial of aggressive medical treatment. Factors favouring its use are a potentially reversible cause of cardiogenic shock and shock after cardiac surgery or chest trauma. Intra-aortic counterpulsation with a balloon pump (IABP) is the method of choice. The IABP is contra-indicated in patients with aortic regurgitation. Percutaneous insertion of the IABP has generally superseded surgical arteriotomy. Using the Seldinger technique to place the introducer in the femoral artery and inserting the balloon over a flexible tip J-wire guide under fluoroscopy, an IABP can be inserted quickly and comparatively atraumatically in nearly all patients.

Potential hazards include limb ischaemia from occlusion of the femoral artery by the balloon catheter or thrombus, aortic dissection, infection and embolization. The IABP is synchronized with the patient's ECG. Inflation and deflation are adjusted by inspection of the ECG and arterial pressure wave form. The balloon is inflated during early diastole (on the dicrotic notch of the arterial wave form), so increasing diastolic pressure and coronary

perfusion. The balloon is deflated immediately before ventricular systole, decreasing aortic pressure and so reducing ventricular afterload. The net effect is to increase cardiac output and coronary perfusion, while decreasing myocardial work and oxygen demand.

Patients with cardiogenic shock after surgery or trauma frequently improve with time and can be weaned off IABP. In those with cardiogenic shock after MI, the IABP is usually a prelude to coronary angiography and ventriculography to demonstrate lesions correctable by surgery. The IABP is also used in patients suitable for cardiac transplant.

Various types of mechanical ventricular assist devices (VAD) have been used in patients with cardiogenic shock which is unresponsive to drugs and IABP. Most of the patients treated with VAD have had recent cardiac surgery but other indications have included shock after MI, myocarditis and support while awaiting cardiac transplantation.

4. Specific Treatment

In some centres, surgical revascularization has been used in patients with MI and cardiogenic shock. The results have been variable and uncontrolled reports make it difficult to assess the effect of patient selection on the results obtained. Systemic blood flow can be improved in patients with acute valvular regurgitation or a ventricular septal defect by afterload reduction with vasodilators and the IABP. Definitive treatment, however, is surgical repair. Early surgery improves the overal outcome. The definitive treatment of an obstructing atrial myxoma is also surgical.

Hypoperfusion in patients with hypertrophic obstructive cardiomyopathy will usually be exacerbated by the measures used to treat other forms of cardiogenic shock, ie, inotropic drugs and afterload reduction. Plasma volume expansion and intravenous titration of beta-adrenergic blocking drugs reduce ventricular outflow obstruction and improve cardiac output.

Cardiogenic shock caused by poor compliance with immunosuppressive treatment in patients with a cardiac transplant can respond rapidly to high dose intravenous steroids.

Prognosis

The prognosis of cardiogenic shock associated with myocardial infarction is poor. In the absence of a surgically correctable lesion, only about one-third of the patients treated actively survive the initial episode, and many of the survivors have continuing angina, congestive heart failure and decreased exercise tolerance. Approximately half of the patients with a surgically correctable lesion should survive to leave hospital. Some of the best results, but from an uncontrolled study, of surgical revascularization in patients with cardiogenic shock but no other surgically correctable lesion, show a 70% thirty day survival and 48% survival at 2 years.

The mortality in patients who need maximal therapy and the IABP to separate from cardiopulmonary bypass, is about 50%. Survival in patients who have required VAD in addition to inotropic drugs and the IABP has been 35-45%. The functional prognosis for these survivors seems relatively good.

Patients remaining dependent on inotropic drugs or mechanical assistance may be considered for heart transplantation. Criteria for this vary between centres, but patients with dilated cardiomyopathy are particularly suitable candidates because they are usually young adults who are otherwise healthy and over 50% die within 2 years of diagnosis. It has been stated that contra-indications to heart transplantation are age over 55 years, widespread vascular disease, high pulmonary vascular resistance, diabetes mellitus, recent pulmonary infarction, active infection and neoplastic disease. The prognosis after cardiac transplantation is relatively good with 70-80% surviving 2-4 years.