Complications associated with myocardial infarction

MYOCARDIAL infarction (MI) is usually the result of thrombosis in a coronary artery, triggered by fissuring of an atheromatous plaque. Platelets and fibrin are deposited on the damaged plaque resulting in the formation of a clot and the occlusion of the artery. This article is an overview of the most common complications associated with MI.

Sudden death
Fatality from MI remains formidable high, with 50 per cent of patients who die after an acute coronary occlusion doing so within the first hour after the onset of symptoms (Rawles, 1997). Death is commonly due to the dysrhythmia, ventricular fibrillation.

The risk of sudden death is highest at the onset of symptoms and declines progressively over a number of hours (Resuscitation Council UK, 2000). This is why individuals need early access to defibrillation in the event of cardiac arrest (Department of Health, 2000).

Disturbance of rate, rhythm and conduction
Dysrhythmias are experienced more frequently than any other MI complication, with the incidence of some type of disturbance at virtually 100 per cent. Although these may be life-threatening, many patients experience only self-limiting dysrhythmias of minimal haemodynamic consequence. Patients with an MI are usually admitted to a coronary care unit where therapy for dysrhythmias producing or likely to produce haemodynamic problems, or any precursors to cardiac arrest, can be commenced.

Traditionally dysrhythmias are classified as early or late in relation to the cardiac event. Early dysrhythmias, occurring within the first 24–48 hours are due to myocardial ischaemia and bear no relation to the size of the MI. The dysrhythmia is unlikely to recur and if it does it does not imply an adverse prognosis.

Late dysrhythmias occur after 48 hours and are a reflection of the extent of ventricular damage. They are likely to recur and indicate an adverse prognosis. From a nursing perspective attention needs to be directed towards identifying and alleviating the cause of the dysrhythmia, such as pain, fear, hypoxia, acidosis or electrolyte imbalance. Dysrhythmias that occur after the MI has fully evolved are more likely to be secondary to other complications such as ventricular aneurysm or heart failure, which are discussed below.

Cardiogenic shock
The term cardiogenic shock is used to describe a complex syndrome associated with inadequate perfusion of vital organs – most significantly the brain, kidneys and heart. It occurs in 15 per cent of MI patients and of these, 90 per cent will die in spite of recent advances in therapy. Patients with an anterior MI or who have lost more than 40 per cent of functional myocardium are at greatest risk. Most deaths occur within the first 24 hours although a small number of patients may die more than seven days later.

Cardiogenic shock is caused by massive irreversible damage to the myocardium, so early treatment of dysrhythmias may prevent its development. Measures to reduce the size of MI, such as early thrombolysis and beta-blockers, may be useful (DoH, 2000).

Nursing management includes reducing the oxygen demands of the already compromised myocardium, preventing infarct extension and maintaining perfusion to the vital organs. The patient with cardiogenic shock presents as cold, sweaty and cyanosed with rapid, shallow respiration, hypotension and tachycardia. Changes in the patient’s mental state are usually present and reflect poor cerebral perfusion – these changes include irritability and restlessness and may lead to coma. As well as physical care, the nurse needs to offer psychological support. Some patients will realise that they are unlikely to recover, and careful and sensitive nursing management needs to ensure that patients’ final hours are spent in comfort with their families.

Cardiac rupture
After arrhythmias and cardiogenic shock, the commonest cause of death after acute MI is rupture. Cardiac rupture complicates 10 per cent of acute MIs and occurs in the healing stages at around five to nine days. However, since the introduction of thrombolytic therapy the maximum risk seems to have moved towards the first 24 hours (Jowett and Thompson, 1995). Attempts have been made to reduce the risk by early treatment with beta-blockers (DoH, 2000). The risk appears to be higher with hypertension or an extensive MI, and it is four times more common in women than men. The commonest site is through the left ventricular wall, manifesting as chest pain, hypotension and dyspnoea. Death is rapid due to haemopericardium and the resultant cardiac tamponade leading to a pulseless electrical activity cardiac arrest.
For some patients the symptoms of pericarditis may be alone. Nursing care involves keeping the patient comfortable and relieving pain, usually with non-steroidal anti-inflammatory agents such as ibuprofen (Swanton, 1994). For some patients the symptoms of pericarditis may be worse than the symptoms of MI. It is, therefore, important that nurses relieve patient anxiety and emphasise that this is a temporary setback to a full recovery.

**Ventricular septal defect**

This structural complication occurs in two per cent of cases following extensive destruction of cardiac muscle, and its replacement by scar tissue. During ventricular systole the aneurysm bulges outwards and reduces the ejection fraction by absorbing the force of myocardial contraction. In effect it steals some of the left ventricular stroke volume.

The aneurysm may act as a focus for abnormal electrical activity and also as a site for thrombi formation. If death occurs this is either due to arrhythmia or emboli rather than cardiac rupture. Patients with ventricular aneurysm are often identified because of refractory left ventricular failure or recurrent angina. Treatment is surgical by left ventricular aneurysectomy (Swanton, 1994).

**Ruptured papillary muscles**

This rare complication affects one per cent of patients, but 70 per cent of these die within the first 24 hours. Rupture of the papillary muscles occurs in the healing stages usually complicating an inferior or anteroseptal MI. There is a sudden onset of mitral insufficiency and heart failure. Treatment is surgical and involves an urgent valve replacement.

**Dressler’s syndrome**

Some texts describe this as post MI syndrome. It presents as pleuropericarditis occurring within the first 12 weeks following an acute MI. The pericarditis is secondary to the MI and is induced by an abnormal autoimmune mechanism. This syndrome is rarely serious but may be distressing and frightening to the patient still recovering from an acute MI. It is treated with anti-inflammatories in the first instance and steroids as a last resort.

**Shoulder hand syndrome**

Left shoulder pain and stiffness is felt two to eight weeks after MI and there may be pain and swelling of the hand. With early mobilisation of the patient it has become a rare complication. It is treated with physiotherapy and is usually resolved after two years.

**Psychological problems and depression**

Up to one-third of MI patients may present with anxiety, depression and over-dependence. Early mobilisation and exercise programmes help prevent this.