Identifying and managing acute coronary syndrome

What is ACS?
The term ACS is credited to Fuster (1985), who identified a syndrome that includes a group of myocardial ischaemic events with different clinical presentations but common pathophysiology. The three presentations included are:

- Unstable angina (UA);
- Non-ST elevation myocardial infarction (nSTEMI);
- ST-elevation myocardial infarction (STEMI).

Although the term has been in clinical use for nearly two decades, ACS is not listed by the World Health Organization as a disease (WHO, 1992). Thus, patients who may be categorised as having ACS on admission will be discharged with a diagnosis of UA/nSTEMI/STEMI. This leads to confusing differences between clinical practice and clinically coded diagnoses, causing problems with primary care disease registers and financial reimbursement.

Pathophysiology
The diagnosis of ACS implies a change of symptom pattern and is usually a response to rupture of an atherosclerotic plaque within the coronary arteries. Plaque rupture triggers a chain of events (platelet activation, adhesion and aggregation) that may lead to thrombus formation in the damaged vessel. Traditionally, changes in ischaemic symptoms have been known as rest angina, new onset angina or increasing angina.

In UA and nSTEMI the thrombus partially occludes blood flow down a coronary artery, starving the heart muscle of oxygen and nutrients. This can result in symptoms with or without ECG changes and cardiac enzyme release. If the thrombus totally occludes the vessel a section of the myocardium will become starved of oxygen and nutrients, and will die, resulting in ST elevation and cardiac enzyme release.

The in-hospital mortality rate within six months of first presentation among patients admitted with ACS is seven per cent with STEMI, six per cent with nSTEMI and three per cent with UA. At three months 33 per cent of patients will have died, been readmitted or had a subsequent acute cardiac event (Fox et al, 2002; Collinson, 2000). The risk of death after four years rises to 25 per cent in both STEMI and nSTEMI presentations.

Signs and symptoms
Classically, patients suffering from myocardial infarction (nSTEMI or STEMI) present with precordial or retrosternal chest pain described as heavy, crushing or burning, with or without ECG changes and cardiac enzyme release. If the thrombus totally occludes the vessel a section of the myocardium will become starved of oxygen and nutrients, and will die, resulting in ST elevation and cardiac enzyme release.

Learning objectives
Each week Nursing Times publishes a guided learning article with reflection points to help you with your CPD. After reading the article you should be able to:

- Understand what acute coronary syndrome is;
- Identify the symptoms that ACS patients will present with;
- Know the clinical assessment and management for this condition;
- Recognise the possible new role for nurses in caring for these patients.
which may radiate to the neck, arms or jaw at rest. The pain may vary in intensity but normally persists for over 15 minutes. It may be accompanied by nausea, vomiting, sweating and dyspnoea. Unstable angina has a similar presentation but occurs at rest or with minimal exertion. It is normally shorter in duration and with less radiation, while symptoms of dyspnoea and nausea tend to be less severe (Joint European Society of Cardiology/American College of Cardiology Committee, 2000). The clinical presentation of UA and NSTE-MI may be indistinguishable from that of STEMI without an ECG.

It is important to note that women and older people with hypertension, diabetes or heart failure are more likely to have atypical symptoms. They may not experience chest pain and often have more nebulous symptoms that can be difficult to assess. For these patients, presentation at hospital is often delayed and they frequently go undiagnosed and do not receive evidence-based treatment. Delayed treatment is known to adversely affect long-term outcome (Avezum et al, 2005; Brieger et al, 2004).

**Clinical assessment**

Rapid and accurate assessment of patients with ACS is essential for successful management. This should include clinical history, physical examination, ECG, risk-factor profile, and cardiac enzymes. An appreciation of the patient’s predisposing risk should be combined with an evaluation of the current event to arrive at an overall assessment of immediate and predictable future clinical cardiovascular risk.

**Clinical history**

Core questions should include a description of the symptoms: Have you got chest discomfort? When did it start? What is it like? Did anything make it better/worse? Does it radiate anywhere? Are there any associated symptoms?

**Physical examination**

The physical examination is often unremarkable, with symptoms reflecting the amount of cardiac compromise. Physical examination may help rule out other potential diagnoses such as aortic dissection, pneumothorax, pericarditis and cardiac tamponade. Vital signs should be recorded, relevant pulses examined and the chest auscultated for gallop rhythms, valve abnormalities and pulmonary oedema.

The presence of heart failure in ACS can be categorised using the Killip classification (Killip and Kimball, 1967). In this simple system the higher the score the greater the prognostic risk to the patient in addition to that from their ECG (Table 1).

**ECG**

In UA/NSTEMI there may be no immediate ECG changes of note. Any changes in ST-segment morphology will influence outcome. Registry data suggests that one year after the initial event ST-segment depression has a risk of death/MI of eight per cent. The greater the degree of depression the higher the risk, with >0.5mm depression being associated with an incidence of death/MI of 16 per cent. The prognostic value of ST-segment depression is known to extend to four years post event (Hyde et al, 1999). ST-segment elevation in those admitted to hospital is associated with a nine per cent mortality at 30 days, and combined ST-segment elevation and depression is associated with a 12 per cent mortality at 30 days. Unfortunately, there may be times when the ECG may be indeterminable due to left bundle branch block. This may be associated with MI or other conditions such as left ventricular hypertrophy or paced rhythm. The presence of new left bundle branch block in MI doubles the mortality risk (Kaul et al, 2001).

**Risk**

There are several tools available to aid the evaluation of risk in ACS. Examples include the Thrombolysis in Myocardial Infarction (TIMI) Score and the Global Registry of Acute Coronary Events (GRACE) Score. Risk scores have become popular as they are simple to use and aid the identification of high-risk patients. They work by allocating a score to common predictors of outcome such as age, gender, prior manifestation of coronary heart disease, hypertension, diabetes and location of ECG change. The higher the score, the poorer the prognosis. In-hospital scores have a high predictive accuracy up to one year post event (De-Araujo et al, 2005; Antman et al, 2000).

**Table 1. Prognostic importance of heart failure on physical examination in acute coronary syndrome (Khot et al, 2003)**

<table>
<thead>
<tr>
<th>KILLIP CLASS</th>
<th>CLINICAL DESCRIPTION</th>
<th>MORTALITY</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>No heart failure</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>SBP &gt;90mmHg, rales one-third or less of lung fields</td>
<td>x3 30-day mortality</td>
</tr>
<tr>
<td>3</td>
<td>SBP &gt;90mmHg, rales one-third or more of lung fields</td>
<td>x5 30-day mortality</td>
</tr>
<tr>
<td>4</td>
<td>SBP &lt;90mmHg, cardiogenic shock, with any rales</td>
<td>x5 30-day mortality</td>
</tr>
</tbody>
</table>

**REFERENCES**


This article has been double-blind peer-reviewed.

For related articles on this subject and links to relevant websites see www.nursingtimes.net
Cardiac markers
Myocardial damage is defined by the detection of certain biochemical cardiac markers such as troponin or creatinine kinase-MB (CK-MB). There are two types of troponin (T and I) in regular diagnostic use. Both are skeletal muscle isoforms with high cardiac selectivity and are the preferred markers for myocardial damage because of this. Troponin starts to rise within six hours of some episodes of ACS and has a half-life of 24 hours. It is elevated for up to 10 days. Troponin is associated with up to a nine-fold increase in death/MI at 30 days. However, troponin elevation may occur in chronic renal failure, congestive cardiac failure, pulmonary embolism and myocarditis and other non-ACS conditions so it should not be used in isolation and the results should be interpreted with appropriate caution (European Society of Cardiology Task Force, 2002).

Management
The rapid and accurate diagnosis and treatment of ACS is a priority. Patients suspected of having ACS should initially be treated in an area with access to resuscitation facilities. They require an early 12-lead ECG (ideally 12-lead cardiac monitoring), an accurate history and risk review and a physical examination. Patients with UA/NSTEMI require oxygen, nitrates, analgesia (normally morphine or diamorphine), aspirin, low molecular-weight heparin, clopidogrel and beta-blockers (with caution in patients with acute heart failure).

Patients with STEMI also require thrombolytic therapy (clot-busting drugs) within 30 minutes or angioplasty within 90 minutes of arrival at hospital. Both groups may also require the addition of a GPIIb/IIIa inhibitor. Provocative cardiac testing such as myocardial perfusion scanning or exercise tolerance testing may be useful (NICE, 2004; DoH, 2000).

One proposal to improve the quality of care is the development of chest pain assessment nurses. These nurses are based in the cardiology or emergency department and are responsible for accurate triage and treatment initiation in patients with suspected ACS. However, a recent national survey indicated that fewer than 25 per cent of UK emergency departments employed nurses for this purpose (Goodacre, 2003). Alternatively, the US model of the chest pain assessment unit (CPAU) could be adopted.

At present UK evidence is limited but US data suggests that combining specialty staff and technological resources in an identified geographical location not only improves outcome but also reduces length of stay and health care costs (Clancy, 2002). Research is under way on both these modalities.

Conclusion
As previously discussed, ACS is a common and high-risk syndrome that can be difficult to diagnose, particularly in women, older people and those with diabetes. Early identification and appropriate management can significantly reduce the risk to the individual of early death, morbidity and subsequent hospital admission. Despite recent improvement in both diagnostic and therapeutic interventions, ACS management remains suboptimal.

Guided reflection
Use the following points to write a reflection for your PREP portfolio:

- Write about where you work and why you read this article;
- List any new information you learnt;
- Describe how you will be able to use this information in your practice;
- How will you follow up this learning?