Acute coronary syndrome: diagnosis, risk assessment and management

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Summary

This article focuses on the diagnosis, risk assessment and management of patients with acute coronary syndrome (ACS). It describes the pathophysiology, classification, epidemiology and natural history of ACS, the importance of early diagnosis and risk assessment, and the various management strategies used to reduce mortality and aid secondary prevention.

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Keywords

Coronary heart disease, ischaemia, myocardial infarction

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Aims and intended learning outcomes

This article provides an overview of the assessment and diagnosis of acute coronary syndrome (ACS). It describes how to manage patients with the condition using an evidence-based approach. After reading this article and completing the time out activities you should be able to:

- Understand the spectrum of ACS.
- Discuss how to assess, diagnose and risk-stratify patients presenting with the condition.
- Describe treatments and secondary prevention measures.
- Understand the nurse's role in caring for patients with ACS.

Introduction

Coronary heart disease (CHD) is the most common cause of death in the UK (British Heart Foundation (BHF) 2010). Around one in five men and one in eight women die from the disease (BHF 2010). A total of 2.7 million people are living with CHD and each year an estimated 124,000 people in the UK have a heart attack (BHF 2010). The clinical presentation of CHD includes silent myocardial ischaemia, stable angina, unstable angina, myocardial infarction (MI), heart failure and sudden death (Bassand *et al* 2007).

Time out 1

Describe your understanding of ACS. Reflect on the opportunities you have in your practice area to promote healthy lifestyles to reduce the risk of CHD. Consider how you can provide this information and effect change in patients who are at high risk.

In the acute phase, CHD is known as ACS. ACS describes a spectrum of clinical conditions, including ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI) and unstable angina (ACS without cardiac enzymes or troponin release) (Fox 2004). The rates of death, MI and re-admission of patients with ACS remain high despite advances in treatment (Bassand *et al* 2007).

Nurses in community and acute settings are pivotal in contributing to the safe and effective management of patients who present with acute

chest pain, the main symptom of ACS. They help to reduce the risk of adverse events and promote secondary prevention. An understanding of the pathophysiology, classification, epidemiology and natural history of ACS will help nurses appreciate the complexity of the condition. Up-to-date clinical knowledge and expertise will enable nurses to contribute to the accurate diagnosis, risk assessment and effective management of patients presenting with ACS.

Time out 2

Define artherosclerosis in your own words. What type of plaque is more prone to rupture? What factors are thought to contribute to plaque instability?

Pathophysiology

CHD is caused by atherosclerosis, which involves a gradual build up of plaque in the lumen of the coronary arteries. The development of atherosclerosis is influenced by risk factors such as smoking, hypertension, hyperlipidaemia and diabetes. ACS occurs as a result of disruption of an atherosclerotic coronary artery plaque, either through rupture or erosion. This causes thrombosis and possibly vasoconstriction, leading to a sudden and significant reduction in blood flow and myocardial oxygen supply (Bassand *et al* 2007).

Unstable plaques that are prone to rupture have a large lipid core, a low density of smooth muscle cells, a high concentration of inflammatory cells and a thin fibrous cap covering the lipid core (Libby 2002). Van de Werf *et al* (2008) suggested that inflammation, a combination of beta-adrenergic stimulation (increased vascular tone and blood pressure), hypercoagulability of the blood and hyper-reactivity of platelets, contribute to plaque instability.

Activities such as physical and emotional stress, which increase sympathetic stimulation and vasoconstriction, may trigger plaque disruption and coronary thrombosis (Stone 2004). ACS can occasionally have a non-atherosclerotic aetiology, such as cocaine use, trauma, thromboembolism, dissection, congenital abnormality, arteritis or complications of cardiac catheterisation (Bassand *et al* 2007). The underlying pathophysiology is the same across the ACS spectrum, but the degree of severity varies.

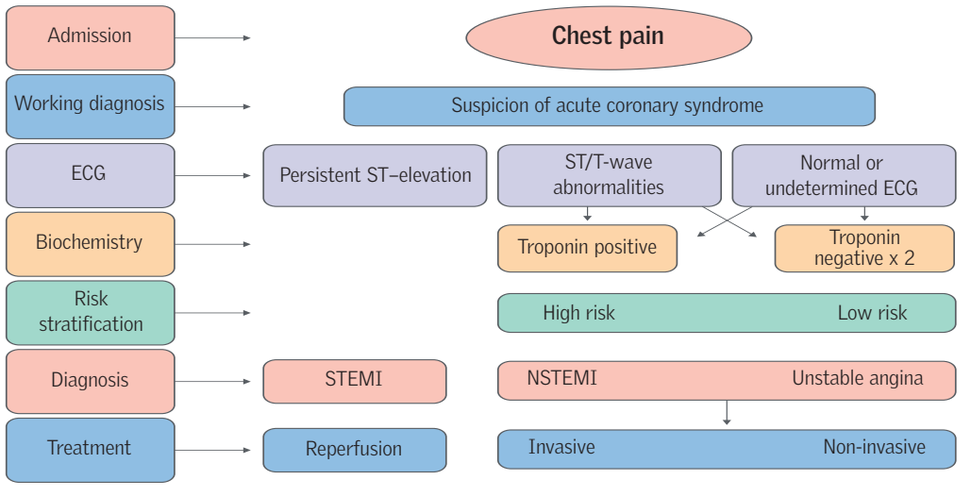
Classification

Chest pain is the main symptom associated with ACS. Depending on the results of an electrocardiogram (ECG), patients are categorised as having ST-elevation ACS or non-ST elevation ACS (Bassand *et al* 2007) (Figure 1).

ST-elevation ACS These patients will have experienced typical acute chest pain for more than 20 minutes and persistent ST-segment elevation (Bassand *et al* 2007). This condition usually occurs when a coronary artery supplying

FIGURE 1

Categorising patients with acute coronary syndrome



ECG = electrocardiogram, STEMI = ST-segment elevation myocardial infarction, NSTEMI = non-ST elevation myocardial infarction

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a large area of myocardium becomes totally occluded. Most of these patients will develop STEMI. In managing this group of patients, the aim is to achieve rapid reperfusion by primary angioplasty or fibrinolytic therapy.

Non-ST-elevation ACS These patients will have experienced acute chest pain without persistent ST-segment elevation. Based on the results of troponin tests, the initial working diagnosis of non-ST elevation ACS can be further categorised as NSTEMI and unstable angina. The aim of managing this group of patients is to correct ischaemia, relieve symptoms, monitor serial ECG recordings and repeat biochemical markers for myocardial necrosis.

An internationally accepted definition of MI was issued in a consensus document by the joint European Society of Cardiology (ESC)/American College of Cardiology committee for the redefinition of myocardial infarction (Alpert *et al* 2000). An updated version – the *Universal definition of myocardial infarction* – was issued by Thygesen *et al* (2007) (Box 1) and used by the National Institute for Health and Clinical Excellence (NICE) (2010a) in its guidelines on chest pain of recent onset.

BOX 1

Universal definition of acute myocardial infarction

Detection of rise and/or fall of cardiac biomarkers (preferably troponin); with at least one value above the 99th percentile of the upper reference limit; together with evidence of myocardial ischaemia with at least one of the following:

- Symptoms of ischaemia (for example, persistent pain in the chest and/or arm/back/jaw, breathlessness, nausea, vomiting, sweating, haemodynamic instability).
- Electrocardiogram (ECG) changes indicative of new ischaemia (new ST or T-wave changes or new left bundle branch block).
- Development of pathological Q waves in the ECG.
- Imaging evidence showing new loss of viable myocardium or new regional wall motion abnormality.

(Thygesen *et al* 2007)

Epidemiology and natural history of acute coronary syndrome

CHD causes about 88,000 deaths in the UK each year and is the most common cause of death among people aged under 75 years (BHF 2010). Tunstall-Pedoe *et al* (1999), who examined trends in survival and coronary event rates over ten years, found that 50% of patients with presumed MI or ACS died in the first month, with about half of deaths occurring within two hours.

Data from the Global Registry of Acute Coronary Events (GRACE Investigators 2001) and the myocardial infarction national audit project (MINAP) have shown that non-ST-elevation is more prevalent than ST-elevation ACS (Birkhead *et al* 2004). This may be because of the redefinition of MI and use of specific biochemical markers of cell necrosis (Alpert *et al* 2000), or changes in the management and prevention of CHD (Fox *et al* 2000, Furman *et al* 2001).

Terkelsen *et al* (2005) showed that death rates were higher among patients with non-ST-elevation ACS than ST-elevation ACS, with a twofold difference at four years. Nair *et al* (2010) demonstrated a significant difference in co-morbidities, revascularisation rates and three-year mortality among STEMI and NSTEMI patients. The NSTEMI patients were older, had more co-morbidities and experienced higher death rates than the STEMI patients. Nair *et al* (2010) found age to be the strongest independent predictor of death in both groups. Chronic renal failure had a negative effect on patient outcome.

Unal *et al* (2004) explored possible reasons for the decline in death rates from CHD in England and Wales between 1981 and 1990. Their findings suggested that 58% of the decline was the result of a reduction in major risk factors, particularly smoking and 42% was attributable to treatments, including secondary prevention. The results of this study emphasise the importance of managing patients who present with ACS effectively and the need to focus on long-term therapeutic strategies.

Time out 3

Reflect on your experience of caring for patients with ACS. How did you provide support and evidence-based information to meet their physical and emotional needs?

Patient-centred care

Patients with ACS require information at various stages: before and on arrival at hospital; before undergoing investigations or procedures; on receiving their test results; when discussing treatment options; and pre and post-discharge. Nurses and other members of the multidisciplinary team need effective communication skills to help alleviate patients' anxieties, give accurate information and correct any misconceptions. They also require up-to-date, evidence-based knowledge to help patients reach informed decisions about their care and management.

Patients' understanding needs to be assessed regularly. Verbal and written information should be provided (NICE 2010a). Nurses may also need to give information to family members and/or carers.

Diagnosis and management

It is vital that nurses have evidence-based knowledge of how patients should be assessed, risk-stratified and managed. Patients' experience and outcomes will be enhanced if nurses know and understand the tools used in the early diagnosis of ACS, such as history-taking, physical examination, ECG recordings and biochemical markers.

Once diagnosis is established, nurses need to be aware of the treatment options available to alleviate the patient's symptoms and improve outcomes. Nurses can use clinical guidelines 48 (NICE 2007), 94 (NICE 2010b) and 95 (NICE 2010a), and the ESC guidelines (Bassand *et al* 2007, Van de Werf *et al* 2008), to make informed decisions about the diagnosis and management of ACS.

History-taking Patients presenting with acute chest pain of suspected cardiac origin account for approximately 6% of all visits to emergency departments in the UK (Goodacre *et al* 2005) and up to 30% of emergency hospital admissions (Murphy *et al* 2004). Identifying patients with ACS as opposed to non-cardiac chest pain presents a major challenge. There is a range of conditions, from fairly benign (muscular skeletal problems and gastric oesophageal reflux) to life-threatening (aortic dissection, pulmonary embolism), that can present as acute chest pain.

Typically, ACS pain is retrosternal, severe and prolonged (>20 minutes); it may radiate to the left arm, jaw or neck (Bassand *et al*, 2007). The use of glycerine trinitrate (GTN) may not relieve symptoms. The patient may also experience symptoms of diaphoresis, abdominal pain, nausea, dyspnoea or syncope (Bassand *et al* 2007).

BOX 2

SOCRATES mnemonic for assessing patients with chest pain

- **S**ite – where is the pain?
- **O**nset – when did the pain start?
- **C**haracteristics – what is the pain like (aching, tight, stabbing)?
- **R**adiation – does the pain spread anywhere else?
- **A**ssociated symptoms – are there any other signs or symptoms associated with pain?
- **T**iming – how long does it last? How often does it occur?
- **E**xacerbating or relieving factors – does anything make it worse or better?
- **S**everity – how bad is the pain?

(Kernicki 1993, Briggs 2010)

Not all patients present with typical chest pain. Older patients and those with diabetes are particularly prone to atypical presentation, such as fatigue, shortness of breath, pre-syncope or syncope. Atypical symptoms may include pain in the epigastric region or back, rather than the centre of the chest. Patients may also display signs of autonomic nervous system activation, such as pallor and hypotension. Other characteristics may include irregular heart rate, bradycardia, tachycardia, third heart sound and basal rales (Van de Werf *et al* 2008).

It is important to follow a systematic approach when assessing patients presenting with chest pain. Nurses may find the mnemonic SOCRATES helpful (Box 2).

NICE (2010a) guidelines on chest pain of recent onset stress the importance of assessing:

- The characteristics of pain.
- Associated symptoms, including nausea, vomiting, sweating and shortness of breath.
- The patient's family history of cardiovascular disease.
- Cardiovascular risk factors.
- Previous investigation and treatments for chest pain.

The guidelines state that GTN is not helpful as a diagnostic tool to differentiate between cardiac and non-cardiac pain.

Physical assessment

Patients presenting with ACS may appear pale, anxious and distressed. A physical assessment is usually unremarkable, but is an important tool to establish baseline information. It provides information on haemodynamic status and can identify signs of complications such as pulmonary oedema or cardiogenic shock. In patients with complications following an acute STEMI, the physical examination may reveal crackles in the lung bases, added heart sounds and raised jugular venous pressure indicating evidence of left ventricular dysfunction.

The presence of a systolic murmur may indicate mitral valve regurgitation secondary to papillary muscle dysfunction (Del Bene and Vaughan 2000). In right ventricular infarction, there may be signs of systemic venous congestion and poor systemic perfusion.

Physical examination is also important to help exclude non-cardiac and non-ischaemic cardiac disorders such as pulmonary embolism, aortic dissection, pericarditis and valvular heart disease (Bassand *et al* 2007).

Electrocardiogram monitoring

Patients presenting with acute chest pain need to have a resting ECG recorded and interpreted as soon as possible by a suitably qualified professional (NICE 2010a). It has been shown that the door-to-treatment interval can be reduced if hospital staff receive the results of an ECG undergone by the patient before admission (Morrison *et al* 2006). This practice is recommended by NICE (2010a) provided it does not delay transfer to hospital.

A diagnosis of acute STEMI is likely if the ECG shows ST-segment elevation (usually defined as 1mm in at least two contiguous limb leads or 2mm in two contiguous chest leads) (Figure 2) or presumed new left bundle branch block (Figure 3).

Immediate action must be taken to initiate reperfusion therapy to reduce myocardial damage and improve the patient's outcome. ST elevation reflects myocardial injury resulting from ischaemia that has lasted for more than a few minutes. The presence of Q waves indicates infarcted tissue that extends at least halfway through the myocardial wall. Q waves can occur within hours of STEMI, but their presence does not indicate when the event occurred (Del Bene and Vaughan 2000).

If the ECG is equivocal in the early stages of acute chest pain, it is vital to repeat the ECG recordings and, if possible, compare the findings with previous ECGs. It is also important to compare the results of ECGs undertaken when the patient is symptomatic and asymptomatic (Bassand *et al* 2007). Changes on the ECG may occur over minutes or hours; it is imperative to detect these changes early so that prompt appropriate treatment can be given. ECG changes reflecting myocardial ischaemia in NSTEMI (two or more contiguous leads) (Figure 4) can include ST depression, T-wave inversion or transient ST-segment elevation.

Cannon *et al* (1997) found evidence that patients with ≥1mm ST depression are at increased risk of death, with a rate of 11% at one year. Deep symmetrical inversion of T waves in the anterior leads is often related to significant stenosis in the proximal left anterior descending coronary artery or in the left main stem (de Zwaan *et al* 1989). It may be necessary to carry out additional recordings to establish a true posterior infarction (V_7-electrode placement in posterior axillary line and V_8-electrode placement in the posterior scapular line), or right ventricular infarction (rV_4-electrode placed in the fifth intercostals space at the right mid-clavicular line).

A normal ECG does not exclude a diagnosis of ACS. Kellett *et al* (2004) suggested that initial ECGs are non-diagnostic in 20-50% of patients with acute MI.

FIGURE 2

ST-segment elevation myocardial infarction

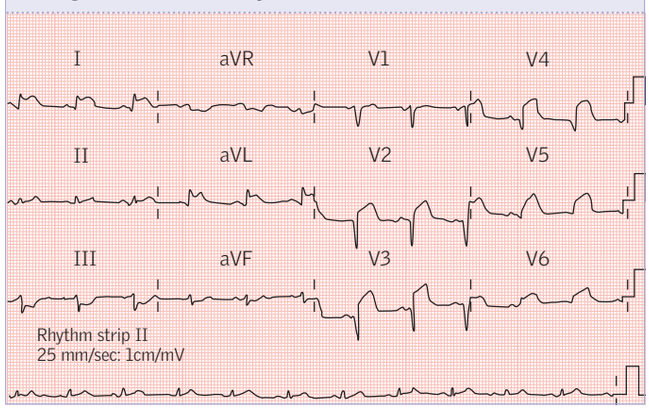


FIGURE 3

Left bundle branch block

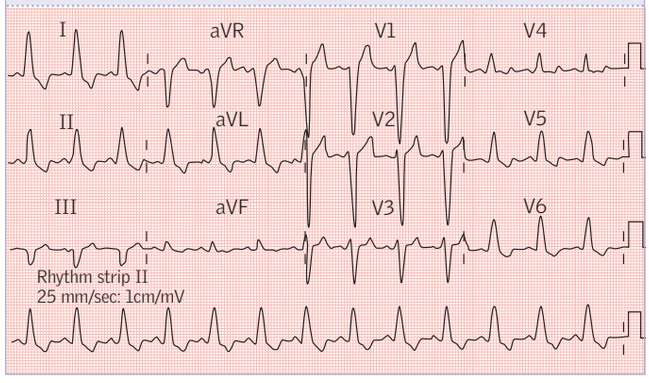
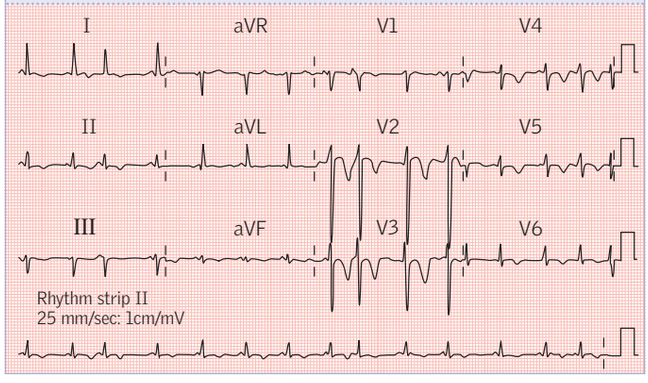


FIGURE 4

Non-ST-segment elevation myocardial infarction in a patient with chest pain and raised levels of troponin



When ECGs are recorded it is essential to ensure the documentation includes the patient's details, the time of the recording and whether or not the patient is experiencing acute chest pain. Nurses caring for patients with acute chest pain need to be competent in ECG recording and may find the

articles by Crawford and Doherty (2008, 2009) useful. Nurses may also find the regular 'ECG of the month' article in the *British Journal of Cardiac Nursing* a useful resource to help improve ECG interpretation.

Biochemical markers

Cardiac biochemical markers are proteins which are released into the bloodstream as a result of damage to the myocyte cell membranes caused by myocardial ischaemia. Over recent years, assays have been developed that are highly sensitive and specific for detection of myocardial necrosis, including troponin I, troponin T and myoglobin. These newer markers have mainly replaced the use of less sensitive and specific enzyme release markers such as creatine kinase (CK-MB) and lactate dehydrogenase (LDH), which are used for the retrospective detection of myocardial necrosis.

Thygesen *et al* (2007) recommended the use of troponin I or troponin T as the preferred biochemical markers because of their high sensitivity of almost 100% for diagnosing acute MI. Troponin I and troponin T levels peak six to 12 hours after onset of acute MI; troponin I may be detected in the bloodstream for seven to ten days, whereas troponin T can last seven to 14 days (NICE 2010a). NICE (2010a) recommend a baseline troponin measurement on patient admission and a second ten to 12 hours after the onset of symptoms. It is important to bear in mind that troponin levels can be raised in a range of other conditions, such as heart failure, renal failure and myocarditis.

Myoglobin is a protein found in cardiac and skeletal muscle. It is released quickly after muscle injury and can be detected in the bloodstream within one hour. It is not cardiac selective, but a diagnosis of MI is unlikely if myoglobin is not raised within three to four hours of symptom onset.

Other diagnostic tools

The ESC recommends the use of echocardiogram in the diagnosis of chest pain (Bassand *et al* 2007, Van de Werf *et al* 2008). An echocardiogram can detect regional wall motion abnormalities, which can occur within seconds after coronary occlusion (Van de Werf *et al* 2008). It can also help to rule out differential diagnosis. A chest X-ray can be used to exclude complications of ACS, such as pulmonary oedema, or to establish a differential diagnosis, such as pneumothorax or pneumonia.

Time out 4

Identify the risk scoring tool recommended by NICE (2010b) to determine mortality at six months and risk of future cardiovascular events. List the variables included in this risk assessment tool.

Early risk-stratification

Assessing the extent to which patients are at risk of an adverse event such as MI, stroke or death, is an important stage in the early management of patients with ACS. Risk will vary between patients. It is important that risk is assessed on an individual basis so that the most appropriate treatment strategy can be adopted. Lee *et al* (2008) found that patients may receive sub-optimal treatment if their risk is not assessed accurately. They found that cardiac catheterisation is not optimally used in patients with non-ST-elevation ACS because doctors do not always risk-stratify patients appropriately.

The use of one variable, such as troponin, is not a reliable assessment of risk when compared to a validated risk scoring system with multiple risk components (NICE 2010b). Two of the most commonly used tools are the thrombolysis in myocardial infarction (TIMI) risk score (Antman *et al* 2000) and the GRACE (Global Registry of Acute Coronary Events) risk score (Granger *et al* 2003).

TIMI was developed to predict the risk of mortality, MI and urgent revascularisation at 14 days. TIMI scoring has been used to analyse the effectiveness of treatment in various risk groups, however it is less accurate in predicting cardiac events (Bassand *et al* 2007). TIMI risk variables include: ≥ 65 years; at least three risk factors for CHD; previous coronary artery stenosis ≥ 50%; ST-segment deviation on ECG at presentation; at least two anginal events in the previous 24 hours; use of aspirin in the previous seven days; and elevated serum cardiac markers. The TIMI risk score is widely accepted and easy to use in clinical practice.

The GRACE risk score originated from an international registry of a large unselected population across the spectrum of ACS. It is recommended as the risk tool of choice by NICE (2010b) guidelines for unstable angina and NSTEMI to estimate the probability of myocardial infarction or death in hospital and at six months. GRACE score variables are shown in Box 3. A disadvantage of the GRACE tool is the need for a computer programme to estimate risk at the bedside, although this can be overcome by the use of a hand-held device such as a personal

digital assistant. Figure 5 identifies the different risk categories and recommended management of unstable angina and non-STEMI (NICE 2010b).

Time out 5

Reflect on the immediate management of patients presenting with acute chest pain. List immediate management strategies based on NICE (2010a) guidance.

Immediate management of acute chest pain

It is important to manage patients effectively when they first present with acute chest pain to reduce the risk of further adverse events. Early administration of antiplatelet therapy, pain relief, and oxygen therapy, if required, will help to reduce this risk.

Aspirin There is strong evidence that aspirin is beneficial in patients with ACS. In a cohort study of patients with acute MI, Barbash *et al* (2002) demonstrated that patients to whom aspirin was administered before hospital admission had lower mortality at seven and 30 days than patients receiving aspirin at admission or during their hospital stay. NICE (2010a, 2010b) recommends a single loading dose of 300mg of aspirin at the earliest opportunity for patients with acute chest pain of suspected cardiac origin, assuming they are not intolerant of, or allergic to, aspirin.

Pain relief Prompt and effective pain relief is a priority when managing patients with chest pain of suspected cardiac origin. NICE (2010a) recommends that patients should be treated so that they are completely pain free. GTN is effective in some patients, but additional pain relief with opioids will be required by others (NICE 2010a).

BOX 3

Global Registry of Acute Coronary Events Risk Score

The variables used to calculate the risk of mortality in hospital or at six months following acute coronary syndrome are:

- Age.
- Heart rate.
- Systolic blood pressure.
- Creatinine levels.
- Killip class (congestive heart failure).
- Cardiac arrest on admission.
- ST-segment deviation.
- Elevated cardiac enzymes or markers.

(Granger *et al* 2003)

Oxygen NICE (2010a) recommends that supplementary oxygen should not be given routinely to patients with acute chest pain of suspected cardiac origin. NICE (2010a) clinical guideline 95 states that oxygen saturation should be monitored and that the levels recorded should be used to guide administration; supplementary oxygen is not beneficial and may be harmful. The British Thoracic Society (2008) states that most patients with ACS are not hypoxaemic and that it is not known whether oxygen therapy is beneficial or harmful.

NICE (2010a) recommends oxygen saturations of 94-98% in patients without hypercapnic respiratory failure (such as those with chronic obstructive pulmonary disorder) and 88-92% in those with the condition. For patients with hypoxaemia, NICE (2010a) recommends 2-6 L/min via a nasal cannula or 5-10 L/min via a simple face mask. The use of a reservoir mask is recommended for patients with oxygen saturation of <85% or those at risk from hypercapnia.

Time out 6

What would make you think that the patient you are caring for has STEMI? What is the recommended management strategy for this patient?

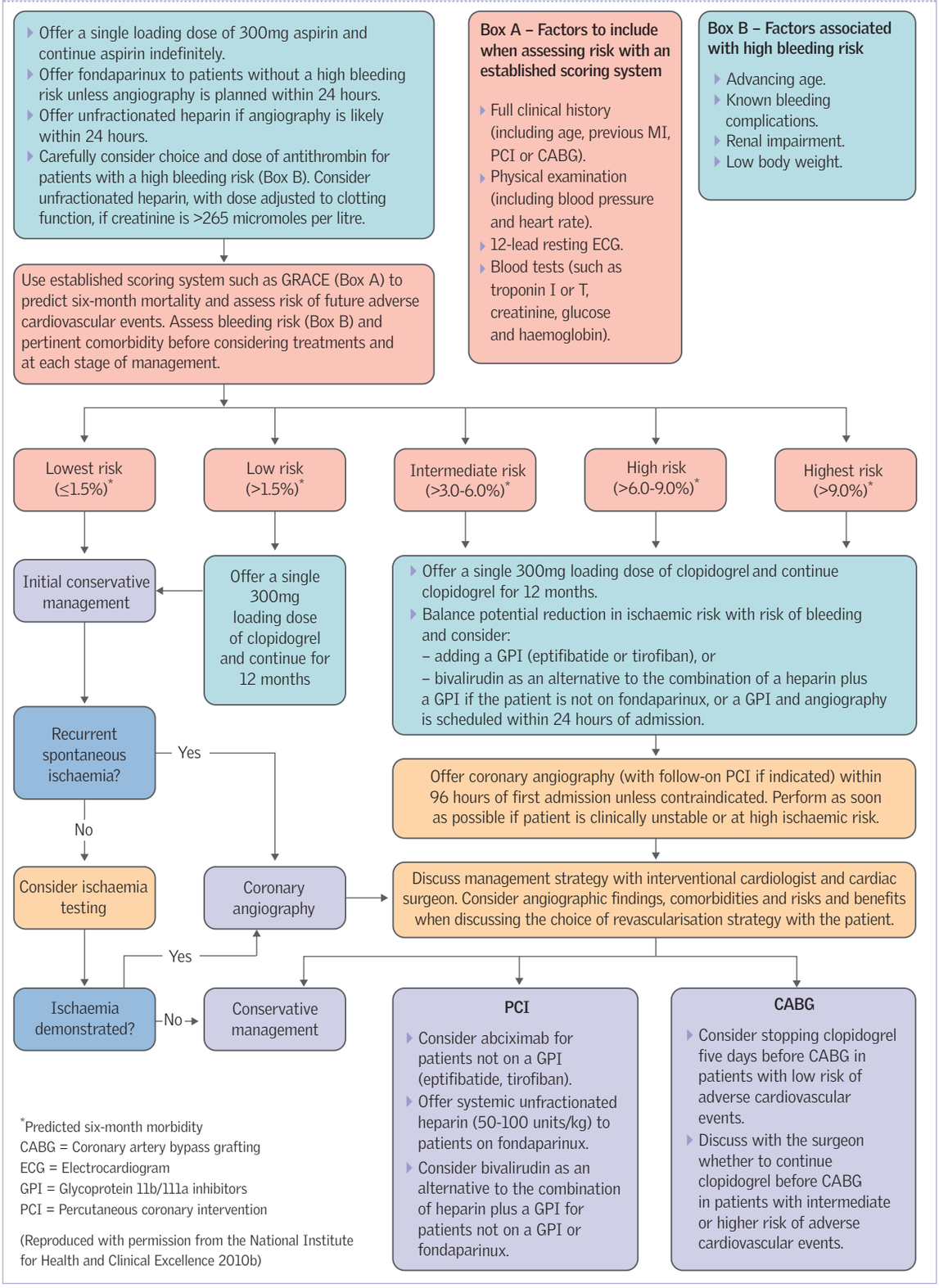
Early management of ST-segment MI

The ESC guidance on the management of patients presenting with STEMI is outlined in Figure 6 (Van de Werf *et al* 2008).

Primary percutaneous coronary intervention In recent years, there has been a move away from the use of thrombolysis in favour of primary percutaneous coronary intervention (PPCI). This is based on evidence that having PPCI available 24 hours a day, seven days a week produces better outcomes than thrombolysis. The National Infarct Angioplasty Project (Department of Health (DH) 2008) demonstrated that PPCI is clinically and cost effective when delivered within 120 minutes of a patient's call for help. The project showed that the longer the delay between the patient's arrival at hospital and receipt of a PPCI, the higher mortality rates in hospital, at 30 days and at one year (DH 2008). The Myocardial Ischaemia National Audit Project demonstrated lower mortality rates at six months among patients receiving PPCI than those receiving thrombolysis (Birkhead *et al* 2009). When thrombolysis is used as a first-line therapy, the ESC guidelines for STEMI recommend that coronary angiography is undertaken within 24 hours (Van de Werf *et al* 2008).

FIGURE 5

Early management of unstable angina and non-ST-elevation myocardial infarction (MI)

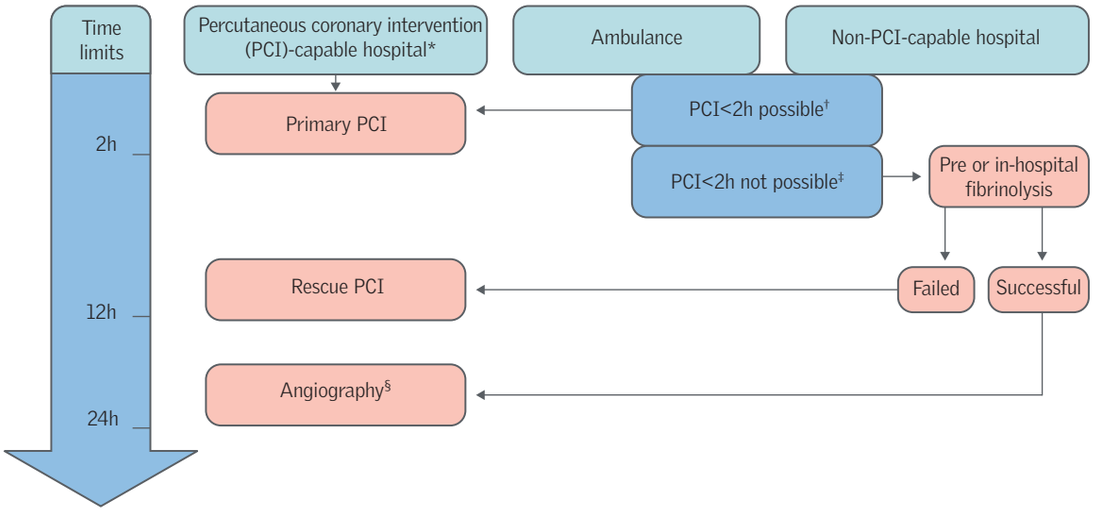


*Predicted six-month morbidity
CABG = Coronary artery bypass grafting
ECG = Electrocardiogram
GPI = Glycoprotein 11b/111a inhibitors
PCI = Percutaneous coronary intervention

(Reproduced with permission from the National Institute for Health and Clinical Excellence 2010b)

FIGURE 6

Recommended time frame for the management of patients presenting with ST-segment elevation myocardial infarction



First medical contact (FMC)

*24-hour service, 7 days a week.

†Time of FMC to first balloon inflation must be shorter than 90 minutes in patients presenting early (<2 hours after symptom onset), with large amount of viable myocardium and low risk of bleeding.

‡If PCI is not possible in <2 hours of FMC, start fibrinolytic therapy as soon as possible.

§Not earlier than 3 hours after start of fibrinolysis.

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Time out 7

What would make you think that the patient you are caring for has unstable angina or NSTEMI? What is the recommended management strategy for this patient?

Early management of unstable angina and non-ST-segment elevation MI

The NICE (2010b) recommendation on the early management of patients with unstable angina and NSTEMI is outlined in Figure 5.

Drug therapy There is evidence from numerous clinical trials and meta-analyses that angiotensin-converting enzyme (ACE) inhibitors, beta blockers, statins and aspirin are beneficial for early and long-term use in patients with CHD.

Clinical trials suggest that ACE inhibitors have an anti-atherogenic effect in patients with risk factors for coronary artery disease or established disease (Yusuf *et al* 2000, Fox 2003). ACE inhibitors are indicated for long-term use in patients with left ventricular ejection fraction ≤ 40%, in patients with diabetes, hypertension or chronic kidney disease, unless contraindicated. They are considered for all other patients to prevent recurrence of ischaemic events (Bassand *et al* 2007). ACE inhibitors should be started early after presentation and titrated to a maximum tolerated or target dose (NICE 2007).

Beta blockers should be offered as soon as the patient is clinically stable, titrated to the maximum tolerated dose and continued indefinitely (NICE 2007). Calcium channel blockers can be used if beta blockers are contraindicated.

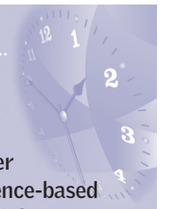
Statin therapy has been shown to reduce cardiovascular risk (Heart Protection Study Collaborative Group 2002, LaRosa *et al* 2005). Intensive lipid lowering in ACS is recommended; it is thought statins may stabilise plaque, have an anti-inflammatory effect and restore endothelial function (Bassand *et al* 2007). They should be offered as soon as possible after the acute event (NICE 2007). Other lipid-lowering drugs should be considered for patients who are intolerant of statins (NICE 2007).

Antiplatelet agents are important in the acute and long-term management of CHD. Aspirin should be continued indefinitely after the acute

event. Clopidogrel is recommended in patients who are intolerant of aspirin (NICE 2007). However, clopidogrel in combination with low dose aspirin is not recommended for longer than 12 months after ACS and four weeks in patients with STEMI, unless there are other indications for continuing dual antiplatelet therapy (NICE 2007).

An aldosterone antagonist may be given to patients with signs of heart failure and left ventricular systolic dysfunction within three to 14 days of acute MI (NICE 2007). For patients not consuming at least 7g of omega 3 fatty acids per week from two to four portions of oily fish, NICE (2007) recommends at least 1g daily of omega-3-acid ethyl esters within three months of acute MI for up to four years.

Time out 8



Consider the long-term management and secondary prevention of patients with coronary artery disease. Consider your role in the delivery of evidence-based practice for this group of patients?

Rehabilitation and discharge planning

NICE (2010b) guidelines recommend that before discharge patients are offered information and advice on:

- Their diagnosis and follow-up arrangements.
- Cardiac rehabilitation.
- Management of cardiovascular risk factors.
- Drug therapy for secondary prevention.

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- Lifestyle changes.

Nurses are well placed to give advice on symptom management to patients before discharge. Patients should be advised to dial 999 and ask for an ambulance should chest pain last longer than 15 minutes despite rest or the use of GTN spray. It is also good practice to give patients a copy of their latest ECG so that they can present it to staff in the emergency department should they be re-admitted.

Conclusion

Patients with ACS usually experience chest pain that is constant and unprovoked. They may first present in primary or secondary care. These patients have a high mortality risk in the early stages of the acute event, so it is imperative that they are managed effectively to reduce or eliminate risk through emergency treatment.

Nurses are in a prime position to identify patients presenting with, or at risk of, developing ACS. A high level of clinical knowledge and skills will enable nurses to contribute to the early diagnosis, management and care of this vulnerable patient group. Effective communication skills will enable nurses to provide valuable information, support and advice for patients and their families throughout their journey from acute care to secondary prevention **NS**

Time out 9

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