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Acute coronary syndromes

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Key points

- Coronary artery disease is a common condition and anaesthetists frequently encounter patients who have suffered or are at risk of suffering from acute coronary syndrome (ACS). A diagnosis of new or recent ACS carries important implications for anaesthetic and critical care management.
- ACS is categorized into two broad groups according to ECG appearance and evidence of myocardial necrosis by biochemical markers. This categorization determines immediate treatment priorities.
- Myocardial infarction and ischaemia can be difficult to diagnose in the perioperative period; most present without chest pain, and the pathophysiology of perioperative myocardial ischaemia is complex.
- Timing of elective surgery in patients with recent coronary stents is complex and requires a multidisciplinary assessment of risks of in-stent thrombosis *vs* risks of perioperative bleeding.
- Starting beta-blockers prophylactically in the immediate preoperative period may increase mortality.

Coronary heart disease is the biggest killer in the UK, resulting in more than 80 000 deaths in 2010.[1] Major non-cardiac surgery is associated with an incidence of perioperative cardiac death of 0.5–1.5%, and of major cardiovascular complications (e.g. myocardial infarction, heart failure and stroke) of 2–3.5%.[2] This article provides a brief synopsis of the diagnosis and management of acute coronary syndrome (ACS). The recognition and implications of perioperative myocardial infarction are discussed, and the perioperative care of patients on anti-platelet therapy with coronary stents *in situ* is summarized.

Definitions

ACS refers to a spectrum of conditions of varying myocardial ischaemic states. It can be broadly categorized into two groups, depending on evidence of myocardial ischaemia on an electrocardiogram (ECG) and evidence of myocardial necrosis from serum levels of cardiac biochemical markers (e.g. troponin):

- ST-elevation myocardial infarction (STEMI)
- Non-ST elevation ACS (NSTE-ACS):
  - Non-ST-elevation myocardial infarction [NSTEMI] (associated troponin rise).
  - Unstable angina (no myocardial cell necrosis/troponin increase).

In general, STEMI is associated with more prolonged duration of ischaemia and a larger territory of myocardial necrosis (e.g. after a complete artery coronary occlusion) than NSTEMI. The short-term mortality from STEMI is marginally higher than that from NSTEMI, but the 4-yr mortality rate for patients with NSTEMI is twice that of STEMI patients.[3] The incidence of STEMI has declined over the past 20 yr[4] whereas the incidence of NSTEMI remains unchanged or has even increased.[3] Improved quality and timeliness of treatment for ACS has resulted in a decrease in hospital mortality from around 20% to nearer 5% over the past 30 yr.[4]

Risk factors

Non-controllable risk factors include: advancing age, male gender, family history of premature coronary artery disease (males <55 yr and females <65 yr), premature menopause and ethnicity (e.g. higher in those from the Indian subcontinent). Modifiable risk factors include smoking, diabetes mellitus, hypertension,

obesity, sedentary lifestyle, and high cholesterol—specifically a high ratio of low- to high-density lipoprotein.

Presentation

Patients usually present with central chest pain of >20 min duration, often radiating to the neck, left arm, or jaw. There may be associated dyspnoea, sweating, palpitations, dizziness, nausea, and vomiting. The character of the pain may be atypical, or chest pain may occasionally be absent. Silent myocardial ischaemia is more common in certain patient populations (e.g. in the perioperative and ITU setting)[3] and in certain conditions (e.g. diabetes mellitus). The relief of chest pain by nitrates is unreliable as a diagnostic tool.

Investigations

12-Lead ECG

ST-segment elevation >1 mm in two consecutive limb leads, 2 mm in two consecutive chest leads (Fig. 1), or new onset left bundle branch block (LBBB) is indicative of STEMI, and should be promptly managed (see below). NSTE-ACS usually presents as ST-segment depression or T-wave inversion (Fig. 2). The ECG may be normal, especially if the pain has resolved.

Biomarkers of myocardial necrosis

The cardiac troponins (cTn) T and I are the most commonly used biomarkers of myocardial damage. They become elevated around 3–6 h, peak at around 12–24 h and can remain elevated for up to 2 weeks, dependent upon renal function. Abnormal levels of cTn are defined as those above the 99th percentile of the normal population or upper reference limit for that laboratory assay, and most local cardiology services will issue a 'positive' result range. If troponin levels are elevated, myocardial necrosis is likely, although there are many other non-ACS causes of an elevated cTn.[5] These include severe congestive cardiac failure, dysrhythmia, myocarditis, pulmonary embolus, acute subarachnoid haemorrhage, severe sepsis, burns, rhabdomyolysis, and renal failure. Elevated levels of cTn must therefore be interpreted allowing for the clinical context. Serial cTn levels may aid diagnosis (e.g. a rapid increase within 24 h, peak levels >50× upper reference

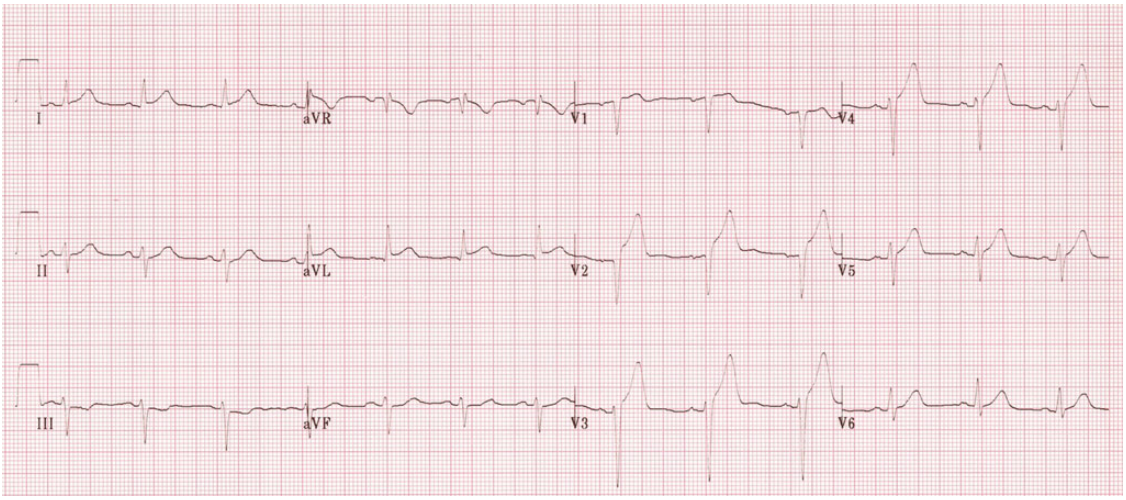

Fig 1 Anterioseptal MI: ST elevation is maximal in the anteroseptal leads (V1–4). Q waves are present in the septal leads (V1–2). Less obvious ST elevation in I, aVL, and V5, with reciprocal ST depression in lead III.

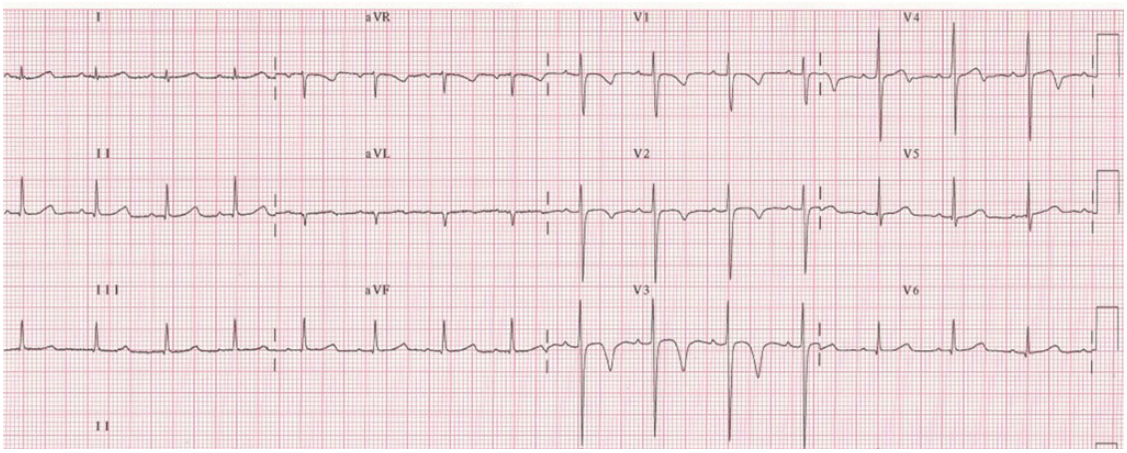

Fig 2 ECG changes possible in NSTEMI: T-wave inversion in the anterioseptal chest leads.