Key Clinical Topics in Critical Care

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Acute coronary syndrome

Key points

- High-sensitivity troponin assay has a negative predictive value approaching 100% when repeated at 3 h
- All patients with acute coronary syndrome should receive aspirin, with intermediate and high risk patients also benefiting from the addition of an ADP-receptor blocker
- Percutaneous coronary intervention is preferred over fibrinolysis if performed within 120 min

Epidemiology

Cardiac chest pain is one of the most common reasons for emergency admission to hospital. The term acute coronary syndrome (ACS) encompasses a range of acute myocardial ischaemic states. ACS is divided into non-ST-elevation ACS (NSTE-ACS) which includes non-ST segment elevation myocardial infarction (NSTEMI) and unstable angina (UA) and ST segment elevation MI (STEMI). The mortality of acute myocardial infarction (MI) has been markedly reduced with early diagnosis and treatment.

Pathophysiology

ACS share common pathophysiology. Most commonly atherosclerotic plaque rupture exposes a highly thrombogenic core. This is followed by thrombus formation or distal embolization, which decreases myocardial blood flow. Less commonly, non-atherosclerotic processes such as dissection, spasm including from cocaine abuse, arteritis or trauma can cause ACS.

When ischaemia is severe enough to cause myocardial necrosis, detectable quantities of biomarkers (e.g. troponin) can be found in blood. Myocardial necrosis in the presence of ischaemia is myocardial infarction.

Clinical features

The typical presentation is with heavy chest pain radiating to the neck, left arm or jaw. This is often associated with nausea, diaphoresis and dyspnoea. Atypical presentations are more common in women, chronic renal failure, diabetics and the elderly.

Investigations

If ACS is suspected an urgent 12-lead-ECG is recorded and followed by serial records.

Elevated cardiac troponins reflect myocardial injury and are more sensitive and specific than creatinine kinase and its isoenzymes.

Important differential diagnoses (e.g. aortic dissection or pneumonia) can be detected or excluded with clinical examination, chest X-ray and echocardiography. Echocardiography should ideally be performed in all patients with MI but should not delay urgent management.

Prior to discharge, testing for residual ischaemia is recommended in stable patients whose condition was treated conservatively. This can be done by stress imaging or more commonly exercise stress testing.

Diagnosis

Diagnosis of ACS is based on history, physical examination, 12-lead-ECG, biomarkers and imaging. 12-lead ECG should be performed within 10 minutes of medical contact and separates patients into NSTE-ACS and STEMI.

Symptoms suggestive of ACS and ST elevation > 0.2 mV in two adjacent chest leads or > 0.1 mV in two or more adjacent limb leads, or new left bundle branch block is diagnostic of STEMI. Confirmatory laboratory results should not be waited for to diagnose STEMI.

A rise in troponin with ischaemic chest pain, ECG changes or a new wall motion abnormality diagnoses myocardial infarction. ST-segment depression, T wave inversion and/or transient ST-elevation can be seen in NSTE-ACS. Elevated biomarkers distinguish NSTEMI from unstable angina (UA). A fall in troponin may also indicate earlier ischaemia in people with no or non-specific symptoms presenting with a raised troponin level that

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subsequently decreases to their baseline level.

Treatment

STEMI requires urgent revascularisation.

Treatment of NSTE-ACS is guided by quantifying risk of short-term adverse cardiovascular events. Most centres have ACS pathways which include early risk stratification. Accepted scoring systems include the Global Registry of Acute Cardiac Events or Thrombolysis in Myocardial Infarction.

Immediate management

- Assess and stabilise airway, breathing and circulation
- Supplemental oxygen if SaO_2 <95%, dyspnoea or heart failure; attach continuous cardiac and SaO_2 monitoring
- 12-lead ECG: Repeat ECG if recurrent or ongoing pain (CXR later)
- Focused clinical exam
- Aspirin: 300 mg chewed then swallowed. Use clopidogrel if allergic to aspirin
- Blood tests: FBC, urea, Creatinine and electrolytes, glucose and troponin (high-sensitivity assay if available)
- Nitrates: Sublingual glyceryl trinitrate (GTN) 0.3–0.4 mg every 5 min up to three times can reduce ischaemic pain. Intravenous should be considered if ongoing pain
- Pain control: Morphine i.v. titrated to effect with an antiemetic
- Beta-blockade: Start orally within 24 h unless contraindicated (e.g. asthma, heart failure, bradycardia, first-degree or left bundle branch block). Use intravenous only in severe hypertension and tachycardia
- Disposition: Cardiac high dependency care area for bed rest, monitoring and ongoing management

Revascularisation

Early reperfusion strategies reduce myocardial damage and decrease mortality.

STEMI

Reperfusion improves outcomes when performed within 12 h of symptom onset. Timely primary percutaneous coronary intervention (PCI) can save up to 20 more lives per 1000 patients compared with thrombolysis. In general, give thrombolytics if PCI cannot be performed within 120 min of presentation.

Thrombolytic therapy

Thrombolytic drugs given early can save up to 30 lives per 1000 STEMIs. Benefit is greatest if given within 3 h of the symptom onset, and can be given pre-hospital if delays to hospitalisation. Transfer all patients post thrombolysis to a PCI-capable centre.

- *Absolute contraindications.* Active haemorrhage. Recent major trauma/surgery/head injury. Any CNS haemorrhage/vascular lesion/malignancy. Aortic dissection. Recent CNS infarction or gastrointestinal haemorrhage. Non-compressible punctures within 24 h
- *Relative contraindications.* Coagulopathy. Pregnancy or <7 days post partum. Prolonged or traumatic resuscitation. Severe uncontrolled hypertension (systolic >180 mmHg or diastolic >130 mmHg)

Choice of thrombolytics

Fibrin-specific (alteplase, reteplase and tenecteplase) and non-fibrin specific drugs (streptokinase) improve outcomes in STEMI. Both classes increase conversion of plasminogen to plasmin, which promotes clot lysis. Streptokinase can cause systemic fibrinolysis and is antigenic. Fibrin-specific agents compared to streptokinase save an additional 10 lives per 1000 treated.

NSTE-ACS

Thrombolysis is not beneficial in NSTE-ACS. PCI evaluation followed by revascularisation improves outcome in the high risk patient.

Patients with refractory angina, arrhythmias, heart failure, new or worsening

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mitral regurgitation or haemodynamic instability should have urgent PCI. If troponin positive without any of the above features, angiography and/or revascularisation, in UK NICE guidelines, are recommended within 96 h, while other international guidelines recommend within 72 h.

Antiplatelet therapy

Thromboxane A_2 promotes the aggregation of platelets. Aspirin inhibits its synthesis, reduces the incidence of MI and improves the survival of patients with ACS. Given early enough, aspirin will save 20–30 lives per 1000 infarcts. Aspirin is continued indefinitely at 75 mg daily.

Glycoprotein IIb/IIIa receptor inhibitors (tirofiban and abciximab) and ADP-receptor blockers (e.g. clopidogrel, ticagrelor or prasugrel) inhibit platelet aggregation.

Ticagrelor (loading dose 180 mg) or prasugrel (loading dose 60 mg) over clopidogrel (loading dose 300—600 mg) are recommended in most patients with ACS. Clopidogrel remains the drug of choice post fibrinolysis.

Anticoagulants

Anticoagulants are used to decrease thrombin generation. All patients with ACS should receive anticoagulant therapy early after diagnosis. Choice is dependent on treatment strategy.

Low molecular weight heparin (LMWH) reduces mortality and MI events compared to unfractionated heparin (UFH) in STEMI. UFH is preferred when using fibrinolysis.

In NSTE-ACS receiving PCI, UFH, LMWH or bivalirudin are options. In no PCI, fondaparinux is preferred over LMWH over UFH. Once started, changing anticoagulant may increase bleeding risk.

Beta-blockade

Treatment can help relieve symptoms and decreases morbidity and mortality in all patients with ACS. Beta-blockers decrease the odds of death by 23% over 2 years in patients with MI.

ACE inhibitor/ARB

An angiotensin converting enzyme inhibitor (ACE-I) or receptor blocker (ARB) improve mortality in ACS. Start within 24 h of presentation. Patients with heart failure, LVEF <40%, diabetes or anterior infarction have most benefit.

Spironolactone

Spironolactone should be commenced prior to discharge in addition to ACE inhibitors for those patients with LVEF <40% (≤35% in NSTE-ACS), New York Heart Association (NYHA) grade 3 or 4 heart failure or diabetes.

Statins

Statin therapy should be commenced as soon as possible in patients with ACS. In the CARE trial pravastatin 40 mg showed an absolute risk reduction of 3% in coronary deaths and nonfatal MI over 5 years. Discontinuing a regular statin can worsen outcome.

Glycaemic control

Acute management should be insulin based and aim for a blood glucose level of less than 10 mmol/L. This includes patients with and without a previous diagnosis of diabetes. Following hospital discharge anti-hyperglycaemic therapy should aim for a haemoglobin A1C of less than 7%. Hypoglycaemia has been associated with poor outcomes and should be avoided.

Rehabilitation

Survivors should complete a cardiac rehabilitation programme and be given advice about their modifiable risk factors (family history, smoking, hyperlipidaemias, hypertension and diabetes mellitus).

Complications

Complications of myocardial infarction depend on the size and location of the infarct. They include arrhythmias, cardiac failure, mitral regurgitation, cardiac rupture and systemic emboli.

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Further reading

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Related topics of interest

- Cardiac pacing (p 88)
- Cardiopulmonary resuscitation (p 100)
- Post-resuscitation care (p 269)

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