Acute Medicine

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Acute cardiovascular medicine

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Cardiovascular disease accounts for up to 40% of all adult general medical admissions in the UK.[1] Patients often present acutely unwell and unstable. Rapid assessment, diagnosis and intervention in this situation can be life-saving.

Acute myocardial infarction

Pain in acute myocardial infarction (AMI) is typically retrosternal, crushing and severe, lasting longer than 20 minutes, often radiating to the arms, neck, jaw or back. However, it may be minimal or even absent, especially in diabetics.

Management of ST elevation acute myocardial infarction

Patients with suspected AMI should be triaged urgently and undergo an immediate ECG. Blood tests, including cardiac enzymes, should be taken but reperfusion therapy should not be delayed while awaiting the results. Similarly, reperfusion should not await a chest X-ray (CXR). If an aortic dissection is considered in the differential diagnosis, immediate imaging of the aorta should be performed with computed tomography/magnetic resonance imaging. A CXR is not helpful in this context.

General treatments

Cardiac monitoring is essential and high-flow oxygen and opiate analgesia should be given. Intravenous (iv) nitrates may be given to aid ischaemic discomfort, control hypertension and pulmonary congestion.

Prognostic treatments

Non-enteric-coated aspirin 300 mg should be chewed, followed by 75 mg per day indefinitely,[2] substituted by clopidogrel 300–600 mg in patients with true aspirin allergy. In the absence of contraindications, oral beta-blockers should be given early in AMI and continued for up to two years.[3] There appears to be no mortality benefit from their iv usage in the revascularisation stage.[3] Hyperglycaemia should be treated with insulin, given on a sliding scale, if the plasma glucose is above 11 mM.[4]

Reperfusion therapy. In the absence of contraindications, reperfusion therapy should be instituted without delay in the presence of ST elevation, new left bundle branch block or posterior infarction (ST depression in leads V1–V4 with prominent R waves and/or ST elevation in leads V7–V9 placed horizontally beneath the left scapula at the level of V4–V6). Thrombolysis or primary percutaneous coronary intervention (PPCI)[5,6] are both acceptable alternatives, although the aphorism 'time is myocardium' holds true. The door-to-needle time for thrombolysis should be within 30 min[7] and the door-to-balloon time for PPCI within 90 min.[8]

Advantages of PPCI include:[9]

- a higher patency rate of the infarct related artery
- reduced short- and long-term mortality, stroke and re-infarction rates
- shorter hospital stay
- the likelihood that it will be more cost-effective.

PPCI outcome appears better even if patients require transfer to another hospital with PPCI facilities within two hours of original presentation.[10] Longer delays may be deleterious, thus the early availability of interventional cardiology

Key Points

Patients presenting with possible acute myocardial infarction (AMI) require urgent triage, urgent ECG and, if confirmatory, immediate reperfusion therapy

In AMI, primary angioplasty reduces mortality, stroke and reinfarction rates versus thrombolysis and is more cost-effective

Early coronary intervention improves prognosis in moderate and high-risk patients presenting with unstable angina or non-ST elevation AMI

In atrial fibrillation of more than 48 hours or of unknown duration, acute cardioversion should be attempted only as an emergency or if trans-oesophageal echocardiography has ruled out left atrial thrombus

Broad complex tachycardias are almost always ventricular tachycardia; they should be treated as such, especially in patients with known structural or ischaemic heart disease

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facilities is a key determinant of whether PPCI should be provided. The National Infarct Angioplasty Project is currently testing its feasibility in the UK. Clopidogrel, 300–600 mg initially followed by 75 mg od, is essential prior to PPCI and is also likely to improve prognosis with thrombolysis therapy.[11]

Management of acute coronary syndromes

Subjects presenting with acute ischaemic symptoms but no ST elevation are said to have a non-ST elevation myocardial infarction (NSTEMI) if cardiac enzymes are raised later and unstable angina if enzymes remain normal. The ECG may be normal or show ST depression and/or T wave flattening or inversion. Autonomic symptoms frequently accompany a significant event whether due to cardiac ischaemia or other intrathoracic catastrophe; chest pain with these symptoms should not be lightly dismissed.

Risk stratification

Patients with a suspected acute coronary syndrome should be risk stratified using a formal scoring system such as the Thrombolysis in Myocardial Infarction (TIMI) risk score (Table 1). Patients with an initial TIMI score of 3 or more benefit from an early invasive strategy and should be considered for early angiography (ideally within 24–48 hours), with a view to percutaneous coronary intervention (PCI) or bypass surgery. One-fifth of medically treated patients with an elevated troponin marker, ST segment changes or haemodynamic instability will suffer death, MI or further angina in the 30 days after the event. All such patients should also undergo early angiography.[12–14]

General treatments

Bed rest, ECG monitoring, high-flow oxygen, opiate analgesia, iv nitrate infusion and beta-blockade are all useful in the relief of ischaemia.

Prognostic treatments

Aspirin and clopidogrel 300 mg, both continued at 75 mg od,[15,16] and low-molecular weight heparin are all useful.[17]

Activation of the glycoprotein (GP) IIb/IIIa receptor is the final common pathway for platelet aggregation. A number of agents have been developed to inhibit this receptor (abciximab, eptifibatide, tirofiban), producing complete inhibition of platelet activation. Their role in NSTEMI is still debated, especially in patients not referred for coronary interventions.[18–20] Currently, the small molecule GPIIb/IIIa receptor antagonists are recommended prior to early coronary intervention in intermediate- or high-risk patients (TIMI risk score ≥3) with ECG changes and/or raised troponin levels. However, many units reserve this treatment for their most unstable patients when angiography/ intervention is not immediately available. Abciximab is predominantly used in conjunction with PCI.

Table 1. Thrombolysis in Myocardial Infarction (TIMI) risk score for unstable angina/non-ST elevation myocardial infarction: stratifying prognostic risk.

Risk factor	TIMI score
Age ≥65 years	1
≥3 coronary artery disease risk factors (family history, hypertension, hypercholesterolaemia, diabetes, current smoker)	1
Known significant coronary artery stenoses (≥50%)	1
ST deviation on the ECG	1
Severe anginal symptoms (eg ≥2 anginal events in past 24 hours)	1
Use of aspirin in past 7 days	1
Elevated serum cardiac markers (CKMB fraction and/or cardiac specific troponin levels)	1
Total possible risk factors	*7*

Risk	TIMI score
Low	0–2
Intermediate	3–4
High	≥5

Troponin levels should be measured 8–12 hours after the episode of pain.

CKMB = creatine kinase containing MB isoenzyme.

Complications of acute myocardial infarction

Cardiogenic shock occurs in 5–10% of patients with AMI, with high in-hospital mortality. In such cases, early PCI improves prognosis over medical therapy.[21] Other potentially reversible causes of cardiogenic shock include right ventricular infarction, acute mitral regurgitation and ventricular septal rupture. If the cause is not clear, urgent echocardiography with or without pulmonary artery catheterisation may be required.

Treatment for pump failure includes inotropic support, intra-aortic balloon counterpulsation and early coronary intervention. Treatment for right ventricular infarction includes fluid loading to increase right ventricular filling pressures and avoidance of vasodilator drugs. If a surgically correctable problem is confirmed on echocardiography, an urgent cardiothoracic referral is required.

Acute pericarditis

Acute pericarditis typically presents with pleuritic central chest pain, worse lying flat and relieved sitting forwards, with or without a recent prodromal phase of fever, malaise and myalgia. A pericardial friction rub is often heard on auscultation, loudest in the left sternal edge leaning forwards. The ECG classically shows widespread concave, saddle-shaped ST elevation, although it may be normal. The ST elevation settles with time and T wave inversion may occur before the ECG normalises. The ST segment elevation is typically more wide-

spread than in STEMI, involving more than one coronary territory. Furthermore, atrial repolarisation is affected, unlike for STEMI, leading to PR elevation in lead aVR (in association with ST depression) and PR depression elsewhere (in association with ST elevation). Unlike AMI, Q waves do not develop and no reciprocal ST depression is seen. Underlying aetiologies are shown in Table 2. Management is symptomatic, with regular high-dose non-steroidal anti-inflammatory medication and bed rest. Anticoagulants should be discontinued and any underlying cause treated. Symptoms usually settle, but relapses occur in 15–40% of cases for which colchicine or steroids may be given.

Acute arrhythmias

Narrow complex tachycardias

Irregular

Atrial fibrillation presents as an irregular narrow complex tachycardia without P waves. Patients with atrial fibrillation, fast ventricular response and angina, heart failure, hypotension (systolic blood pressure <90 mmHg) or impaired conscious level should undergo urgent synchronised DC cardioversion. The following recommendations are made in patients who tolerate atrial fibrillation:

- duration less than 48 hours: chemical cardioversion with flecainide (if no structural or ischaemic heart disease (IHD)), amiodarone or sotalol is preferable to DC cardioversion
- duration longer than 48 hours or unknown duration:
  - anticoagulation followed by rate control with digoxin and/or rate controlling calcium antagonists or beta-blockers prior to staged electrical cardioversion
  - acute cardioversion should be attempted only if trans-oesophageal echocardiography has ruled out left atrial thrombus.

Recommendations are similar for atrial flutter. Carotid sinus massage or adenosine may transiently slow atrioventricular (AV) conduction, making the flutter waves more obvious and thus aiding diagnosis. Atrial flutter ablation, a definitive cure, is now available and should be considered at an early stage in patients with recurrent atrial flutter.

If delta-waves are seen or the ventricular rate is very fast (200–300 bpm), a rapidly conducting accessory pathway is likely. In such cases iv flecainide and/or DC cardioversion is recommended. AV node blockers (eg adenosine, digoxin, verapamil, diltiazem) are contraindicated because they potentially further accelerate the ventricular response.

Regular

In narrow complex regular tachycardias, vagal manoeuvres (carotid sinus massage, Valsalva manoeuvre) or adenosine given rapidly into a large vein may terminate the arrhythmia by transiently blocking the AV node. Serial boluses of adenosine in increasing doses (3 mg then 6 mg then 12 mg) may be required for successful termination. Adenosine should be used with caution in chronic obstructive pulmonary disease and should not be used in asthma. Verapamil 5 mg iv over one minute, repeated once if necessary, is an alternative regimen.

Broad complex tachycardias

Broad complex tachycardias are almost always ventricular tachycardia (VT), especially in patients with known structural disease or IHD. Other suggestive markers include QRS duration above 140 ms, severe axis deviation and ventricular concordance. Confirmatory findings include capture or fusion beats and AV dissociation.

If well tolerated, iv amiodarone or lignocaine may terminate the arrhythmia. Urgent DC cardioversion is required in cardiogenic shock. Torsades de pointes VT should be distinguished from polymorphic VT with a normal QT interval. The latter can be treated as standard VT. The use of QT prolonging anti-arrhythmics (class 1A, 1C and 3) is contraindicated in

Table 2. Causes of pericarditis.

Infections	Bacterial, viral, fungal
Neoplasia	Primary: mesothelioma, angiosarcoma Secondary: lung, breast, bone, lymphoma, melanoma
Connective tissue disorders	Rheumatoid arthritis, systemic lupus erythematosus, mixed connective tissue disorder, Wegener's granulomatosis, Takayasu's arthritis
Drugs	Doxorubicin, hydralazine, methyldopa, minoxidil, penicillin, phenytoin, procainamide
Myocardial injury	Cardiac surgery, trauma, Dressler's syndrome
Miscellaneous	Hypothyroidism, uraemia, sarcoidosis, radiation, idiopathic

Table 3. Rarer broad complex tachycardias.

Tachycardia	QRS complex shape	Axis	Terminated by:
Right ventricular outflow tract	Left bundle branch block	Right axis deviation	Adenosine Verapamil (only under senior cardiology advice)
Posterior fascicular	Right bundle branch block (duration ca 120 ms)	Left axis deviation	Verapamil (only under senior cardiology advice)
Anterior fascicular	Right bundle branch block (duration ca 120 ms)	Right axis deviation	Verapamil (only under senior cardiology advice)

torsades; iv magnesium, overdrive ventricular pacing and beta-blockade may be required. In incessant VT or 'VT storm', the potassium should be corrected; iv amiodarone, magnesium and/or overdrive pacing may be required. Pacing at 100 b/min reduces ectopics likely to initiate VT and alters the conducting characteristics of the VT circuit. If VT continues, an urgent cardiology opinion is required leading to intra-aortic balloon counterpulsation/angiography/VT ablation.

Rarer types of broad complex tachycardias include right ventricular outflow tract tachycardia and fascicular tachycardia which may need to be dealt with differently (Table 3).

Table 4(a). Major Duke criteria for infective endocarditis (IE). Reprinted with permission from Elsevier Inc.[22]

Criteria	
Positive blood culture for IE	Typical micro-organism consistent with IE from 2 separate blood cultures, ie: • viridans streptococci, *Streptococcus bovis* or HACEK group or • community-acquired *Staphylococcus aureus* or enterococci in the absence of a primary focus *or* Micro-organisms consistent with IE from persistently positive blood cultures defined as: • 2 positive cultures of blood samples drawn >12 hours apart or • all of 3 or majority of 4 separate blood cultures (first and last sample drawn 1 hour apart)
Evidence of endocardial involvement	Positive echocardiography for IE defined as: • oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets or on implanted material in the absence of an alternative anatomic explanation or • abscess or • new partial dehiscence of prosthetic valve *or* New valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)

HACEK = *Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikinella*, *Kingella*.

Table 4(b). Minor Duke criteria for infective endocarditis (IE). Reprinted with permission from Elsevier Inc.[22]

Criteria	
Predisposition	Predisposing heart condition or intravenous drug use
Fever	Temperature >38.0°C
Vascular phenomena	Major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial haemorrhage, conjunctival haemorrhages and Janeway lesions
Immunological phenomena	Glomerulonephritis, Osler's nodes, Roth spots and rheumatoid factor
Microbiological evidence	Positive blood culture not meeting a major criterion* or serological evidence of active infection with organism consistent with IE
Echocardiographic findings	Consistent with IE but do not meet a major criterion

*Excludes single positive cultures for coagulase-negative staphylococci, diphtheroids and organisms that do not commonly cause endocarditis.
IE is diagnosed if two major *or* one major and three minor *or* five minor criteria are met.

Bradycardias

The various causes of symptomatic bardycardia are:

- acute ischaemia of the sinus node
- metabolic disturbances (hypothermia, hypothyroidism, rheumatic fever)
- drug therapy (beta-blockers, digoxin, verapamil, diltiazem)
- chronic degenerative conduction disease
- Lyme disease.

In addition to investigating and treating any underlying cause, acute therapy includes iv atropine, repeated as necessary, isoprenaline infusion, external temporary pacing and internal temporary pacing.

Infective endocarditis

Approximately half of all cases of infective endocarditis (IE) occur on normal valves, the rest on rheumatic or congenitally abnormal valves or other cardiac structures. Symptoms include fever, rigors, night sweats, malaise and weight loss. Signs include:

- finger clubbing
- splinter haemorrhages
- Osler's nodes
- Janeway lesions
- a new cardiac murmur or change in prior murmur
- splenomegaly
- fever.

Tables 4(a) and (b) depict the Duke criteria for diagnosing infective IE.[22] IE is diagnosed if two major or one major and three minor or five minor criteria are met. When IE is suspected, three sets of blood cultures should be taken in the first 24 hours. In patients who are very unwell, three sets should be taken within two hours before starting empiric therapy (Table 5).[23] An early cardiology opinion should be sought in all patients with IE.

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Table 5. Current recommended empiric antibiotic regimens in suspected infective endocarditis. Reproduced with permission of the British Society for Antimicrobial Chemotherapy.[23]

	Antibiotic regimen
Acute presentation	Flucloxacillin 8–12 g iv in 4–6 divided doses + Gentamicin 1 mg/kg iv 8 hourly modified by renal function
Indolent presentation	Benzyl penicillin 7.2 g iv in 6 divided doses or Ampicillin/amoxicillin 2 g iv 6 hourly + Gentamicin 1 mg/kg iv 8 hourly modified by renal function
Penicillin allergy or Intracardiac prosthesis or Suspected MRSA	Vancomycin 1 g 12 hourly iv modified by renal function + Rifampicin 300–600 mg 12 hourly po + Gentamicin 1 mg/kg iv 8 hourly modified by renal function

iv = intravenous; MRSA = methacillin-resistant *Staphylococcus aureus*; po = by mouth.

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