CSA and ACS

Prof. Mazin Zamel Alshibani

CSA:

Management: general measures:

- Careful assessment, identification and control of risk factors.
- Use measures to control symptoms
- Identification of high-risk patients for treatment to improve life expectancy.

Advice to patients with CSA:

- Don't smoke
- Aim for ideal body weight
- Take regular exercise (up to but not beyond the point of chest discomfort)
- Avoid severe unaccustomed exercise and vigorous exercise after heavy meal or in very cold weather
- Take sublingual nitrate before undertaking exercise that may induce angina

Treatment:

A. Antiplatelet therapy

* Low-dose (75 mg) aspirin reduces the risk of adverse events.

* Clopidogrel (75 mg daily) is an equally effective antiplatelet agent that can be prescribed if aspirin causes troubles.

B. Anti-anginal drug treatment

1. Nitrates:

* These drugs act directly on vascular smooth muscle to produce venous and arteriolar dilatation, their beneficial effects are due to a reduction in myocardial oxygen demand (lower preload and afterload) and an increase in myocardial oxygen supply (coronary vasodilatation). ** Side-effects include headache, symptomatic hypotension and, rarely, syncope.*

- GTN can be given transcutaneously as a patch (5–10 mg daily), or as a slow-release buccal tablet (1–5 mg 4 times daily).

- Other nitrates, such as isosorbide dinitrate (10–20 mg 3 times daily) and isosorbide mononitrate (20–60 mg once or twice daily), can be given by mouth.

- Continuous nitrate therapy can cause pharmacological tolerance. This can be avoided by a 6–8-hour nitrate-free period, best achieved at night when the patient is inactive.

- If nocturnal angina is a predominant symptom, long acting nitrates can be given at the end of the day.

Duration of action of some nitrate preparation		
Preparation	*Peak action*	*Duration of action*
Sublingual GTN	4-8 m	10-30 m
Buccal GTN	4-10 m	30-300 m
Transdermal GTN	1-3 hrs.	Up to 24 hrs.
Oral isosorbid dinitrate	45-120 m	2-6 hrs.
Oral isosorbid mononitrate	45-120 m	6-10 hrs

2. B-blockers

- These lower myocardial oxygen demand by reducing heart rate, BP and myocardial contractility, but they may provoke bronchospasm in patients with asthma.

- Non-selective β-blockers may aggravate coronary vasospasm and so a once-daily cardioselective preparation is used (e.g. slow-release metoprolol 50–200 mg daily, bisoprolol 5–15 mg daily).

** Beta-blockers should not be withdrawn abruptly as rebound effects may precipitate dangerous arrhythmias, worsening angina or MI (β-blocker withdrawal syndrome).

3. Calcium channel antagonists:

* These drugs inhibit the slow inward current caused by the entry of extracellular calcium through the cell membrane of excitable cells, particularly cardiac and arteriolar smooth muscle, and lower myocardial oxygen demand by reducing BP and myocardial contractility.

* A. Dihydropyridine calcium antagonists, such as nifedipine, amlodipin and nicardipine, often cause a reflex tachycardia. This may be counterproductive and it is best to use them in combination with a β-blocker.

B. Non-dihydropyridine, verapamil and diltiazem are particularly suitable for patients who are not receiving a β-blocker (e.g. those with airways obstruction) because they slow SA node firing, inhibit conduction through the AV node and tend to cause a bradycardia.

* Calcium channel antagonists reduce myocardial contractility and can aggravate or precipitate heart failure. Other unwanted effects include peripheral oedema, flushing, headache and dizziness.

4. Potassium channel activators

These have arterial and venous dilating properties but do not exhibit the tolerance seen with nitrates. Nicorandil (10–30 mg twice daily orally) is the only drug in this class currently available for clinical use.

5. I_f channel antagonist

- Ivabradine is the first drug of this class . It induces bradycardia by modulating iodine ionic channels in the SAN.
- In contrast to β-blockers and rate-limiting calcium antagonists, it does not have other cardiovascular effects.
- It appears to be safe to use in patients with heart failure.

C. Invasive therapy:

1. Percutaneous coronary intervention (PCI)

PCI by using special stent and balloon.

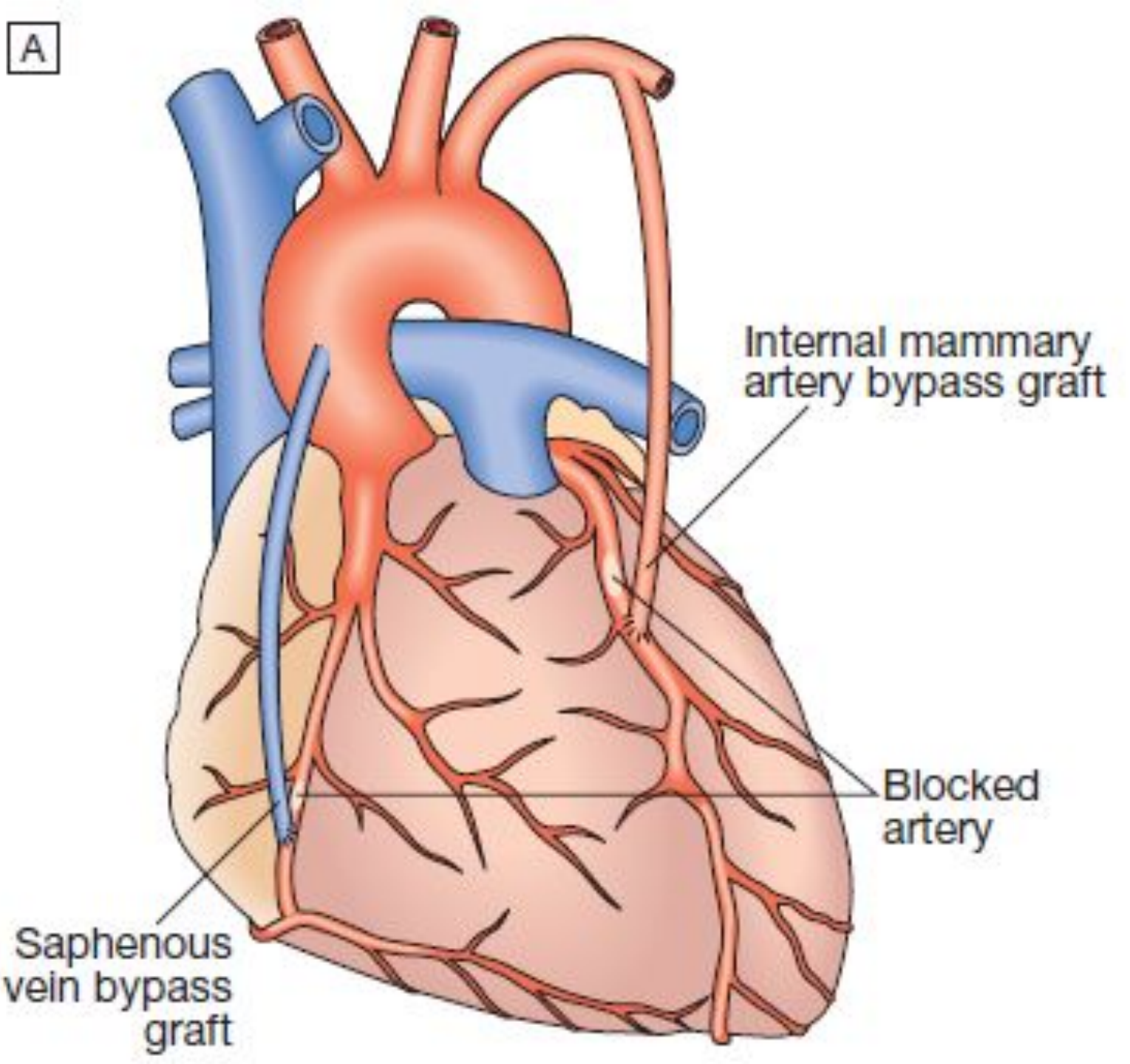
It is mainly used in single- or two-vessel disease.

2. Coronary artery bypass grafting (CABG)

Coronary surgery is usually the preferred option in patients with three-vessel or left main stem disease.

The internal mammary arteries, radial arteries or reversed segments of the patient's own saphenous vein can be used to bypass coronary artery stenosis.

Comparison of PCI and CABG		
	PCI	CABG
Death	< 0.5%	< 1.5 %
MI	2%	10%
Hospital stay	12-36 hrs.	5-8 days
Return to work	2-5 days	6-12 wks
Recurrent angina	15-20 % at 2 ys	10 % at 1y
Repeat revascularization	10-20 % at 2 ys	25% at 2 ys
Neurological complication	Rare	Common
Other complication	Emergency CABG, vascular damage related to access site	Diffuse myocardial damage, infection (chest, wound), wound pain


A
Internal mammary artery bypass graft
Blocked artery
Saphenous vein bypass graft

Prognosis

- Symptoms are a poor guide to prognosis; the 5-year mortality of patients with severe angina (NYHA class III or IV) is nearly double that of patients with mild symptoms.

- Spontaneous symptomatic improvement due to the development of collateral vessels is common.

- TMT and other forms of stress testing are much more powerful predictors of mortality

** The prognosis of CAD is related to the number of diseased vessels and the degree of left ventricular dysfunction.

** Patient with single-vessel disease and good left ventricular function has an excellent outlook, whereas a patient with severe left ventricular dysfunction and extensive 3VD has a poor prognosis without revascularization.

Population advice to prevent CAD:

* Don't smoke
* Take regular exercise (minimum of 20 ms, three times / week)
* Maintain ideal body weight
* Eat a mixed diet rich in fresh fruit and vegetables
* Aim to get no more than 10 % of energy intake from saturated fat

Acute coronary syndrome (ACS):

- Consistent with prolonged, severe cardiac chest pain and may be due to unstable angina (UA) or acute MI (STEMI, full thickness, trans-mural and NSTEMI, partial thickness, sub-endocardial).

- Signs of haemodynamic instability (hypotension, pulmonary oedema), ECG changes (ST segment elevation or depression) and elevated troponin I or T, are powerful indicators of short-term risk.

- Troponin concentrations should be measured at presentation and, if normal, repeated 6–12 hours after the onset of symptoms or hospital admission. New ECG changes or an elevated plasma troponin concentration confirm the diagnosis of an ACS.
- If the pain has not recurred, troponin concentrations are not elevated and there are no new ECG changes, the patient may be discharged from hospital.

- UA characterized by new-onset or rapidly worsening angina (crescendo angina), angina on minimal exertion or angina at rest.

- In UA, the culprit lesion is usually a complex ulcerated or fissured atheromatous plaque with adherent platelet-rich thrombus and local coronary artery spasm. The degree of obstruction may either increase, leading to complete vessel occlusion, or regress due to the effects of platelet disaggregation and endogenous fibrinolysis.

- In acute MI, occlusive thrombus is almost always present at the site of rupture or erosion of an atheromatous plaque. The thrombus may undergo spontaneous lysis over the course of the next few days, although, by this time, irreversible myocardial damage has occurred.

Braunwald classification of unstable angina:

Class	Characteristics
I	Exertional angina New onset, severe, or accelerated Angina < 2 mo duration More frequent angina Angina precipitated by less exertion No rest angina in the last 2 mo
II	Rest angina, subacute Rest angina within the last month but non within 48 h of presentation
III	Rest angina, acute Rest angina within 48 h of presentation
Clinical circumstances	
A	Secondary unstable angina Caused by non-cardiac condition such as anemia, infection, thyrotoxicosis or hypoxemia
B	Primary unstable angina
C	Post infarcted unstable angina Within 2 wks of documented MI

Clinical classification of different types of MI:

Type 1 spontaneous MI: related to ischemia from a coronary plaque rupture or dissection

Type 2 MI: due to ischemia resulting from increased oxygen demand or decreased supply

Type 3 MI: Sudden cardiac death with symptoms of ischemia, new ST elevation, or LBBB or coronary thrombus

Type 4a MI: associated with PCI

Type 4b MI: associated with stent thrombosis

Type 5 MI: associated with CABG

Definition of AMI: Evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia. Any one of the following meets the criteria for Dx of AMI:

- ✔ Detection of rise or fall in cardiac biomarker with at least one of the following:

1. Symptom of ischemia
2. New or presumably new significant ST-T changes or new LBBB
3. Development of pathological Q-wave
4. Imaging of evidence of new loss of viable myocardium
5. Identification of intracoronary thrombus by angiography

- ✔ Cardiac death with symptoms of myocardial ischemia and presumed new ischemic ECG changes or new LBBB

- ✔ PCI-related MI define by elevation of Tr in addition to suggestive symptom of ischemia or new ischemic ECG changes or angiographic finding consistent with procedure related complication
- ✔ Stent thrombosis associated with MI
- ✔ CABG related MI: elevation of biomarker in patients with normal baseline Tr values, plus new pathological Q waves or new LBBB, or angiographic documented new graft or new native coronary A occlusion or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Criteria for prior MI: any one of the following:

** Pathological Q-wave with or without symptoms in the absence of non-ischemic causes

Imaging evidence of a region of loss of viable myocardium that ** .thinned and fail to contract, in the absence of non-ischemic cause

Clinical features of ACS:

Symptoms:

- Prolonged periodic pain, which is the cardinal feature: chest, throat, arm, epigasterium, or back. The pain occurs in the same sites as angina but is usually more severe and lasts longer; it is often described as a tightness, heaviness or constriction in the chest.
- MI may pass unrecognized. Painless or 'silent' MI is particularly common in elderly patients, patient with autonomic neuropathy, those with diabetes mellitus or anemia.
- Anxiety and fear of impending death
- Nausea and vomiting , may aggravated by opiate giving for pain relief
- Breathlessness
- Collapse/ syncope (usually due to an arrhythmia or profound hypotension)

Physical signs:

- Signs of sympathetic activation: pallor, sweating, tachycardia
- Signs of vagal activation; vomiting, bradycardia (particularly in patient with inferior MI)
- Sign of impaired myocardial function: (hypotension, oliguria, cold extremities, narrow pulse pressure, raised JVP, S3, quite S1, diffuse apical impulse, lung crepitation)
- Signs of complications: MR, pericarditis.
- Sudden death, from ventricular fibrillation or asystole, may occur immediately and often within the first hour.
- Sometimes infarction occurs in the absence of physical signs.

Diagnosis and risk stratification

- The differential diagnosis is wide and includes most causes of central chest pain or collapse.
- The assessment of acute chest pain depends heavily on an analysis of the character of the pain and its associated features, evaluation of the ECG, and serial measurements of biochemical markers of cardiac damage, such as troponin I and T.

- A 12-lead ECG is mandatory. Patients with ST-segment elevation or new bundle branch block require emergency reperfusion therapy (thrombolytic therapy or primary PCI).

- In patients with ACS without ST-segment elevation, the ECG may show transient or persistent ST-T wave changes, including ST depression and T-wave inversion (UA).

- Approximately 12% of patients will die within 1 month and 20% within 6 months of the event.

- The risk markers of adverse prognosis include: recurrent ischaemia, extensive ECG changes at rest or during pain, the release of biochemical markers (creatine kinase or troponin), arrhythmias, and haemodynamic complications (e.g. hypotension, MR) during episodes of ischaemia.

Differential diagnosis of ST-segment elevation MI

Comorbid ischemia	**ST-elevation but no ischemia**	**Chest pain but no ischemia**
Aortic dissection	Earle repolarization	Aortic dissection
System arterial embolism	LVH	Myopericarditis
Hypertensive crisis	LBBB	Pleuritis
AS	Hyperkalemia	Pumonary embolism
Cocaine use	Brugada syndrome	Costochondritis
Arteritis		GIT disorders

ECG criteria for Dx of AMI in presence of LBBB (Sgarbbosa criteria)

Criteria	Score
ST-segment elevation ≥ 1mm concordant with QRS	5
ST-segment depression ≥ 1mm in leads V1, V2, or V3	3
ST-segment elevation ≥ 5 mm discordant with QRS	2
Total point score of 3 yield 90% of specificity and 80% positive predictive value	

Investigations

1. *ECG:* may be difficult to interpret if there is bundle branch block or previous MI. The initial ECG may be normal or non-diagnostic in 1/3 of cases.

* Repeated ECGs are important if diagnosis is uncertain or the patient has recurrent or persistent symptoms. The earliest change is usually ST-segment deviation.

* With proximal occlusion of a major coronary artery, ST-segment elevation (or new bundle branch block) is seen initially, with diminution in the size of the R-wave and, T wave becomes inverted, and development of a Q wave in transmural, full thickness MI (STEMI).

In NSTEMI, there is partial occlusion of a major vessel or * complete occlusion of a minor vessel, causing unstable angina or partial-thickness (subendocardial) MI. This is usually associated with ST-segment depression and T-wave changes.

* The ECG changes are best seen in the leads that ‘face’ the ischaemic or infarcted area. In anteroseptal infarction, abnormalities are found in one or more leads from V1 to V4, while anterolateral infarction produces changes from V4 to V6, in aVL and in lead I. Inferior infarction is best shown in leads II, III and aVF, (at the same time) leads I, aVL and the anterior chest leads may show ‘reciprocal’ changes of ST depression.

* Infarction of the posterior wall of the LV does not cause ST elevation or Q waves in the standard leads, but can be diagnosed by the presence of reciprocal changes (ST depression and a tall R wave .in leads V1–V4)

* Some infarctions (especially inferior) also involve the RV. This may be identified by recording from additional leads placed over the right precordium (ST elevation in RV4-5).

2. Cardiac biomarkers

- In unstable angina, there is no detectable rise in cardiac biomarkers or enzymes, and the initial diagnosis is made from the clinical history and ECG only.

- In MI, there is rise in concentration of CK, a more sensitive and specific is (CK-MB), and the cardio-specific proteins, troponins T and I. CK starts to rise at 4–6 hours, peaks at about 12 hours and falls to normal within 48–72 hours. (Three types of troponins exist—troponin I, troponin T, and troponin C. Each subunit has a unique function: Troponin T binds the troponin components to tropomyosin, troponin I inhibits the interaction of myosin with actin, and troponin C contains the binding sites for Ca^{2+} that helps initiate contraction.)

- The most sensitive markers of myocardial cell damage are troponins T and I, which are released within 4–6 hours and remain elevated for up to 2 weeks.

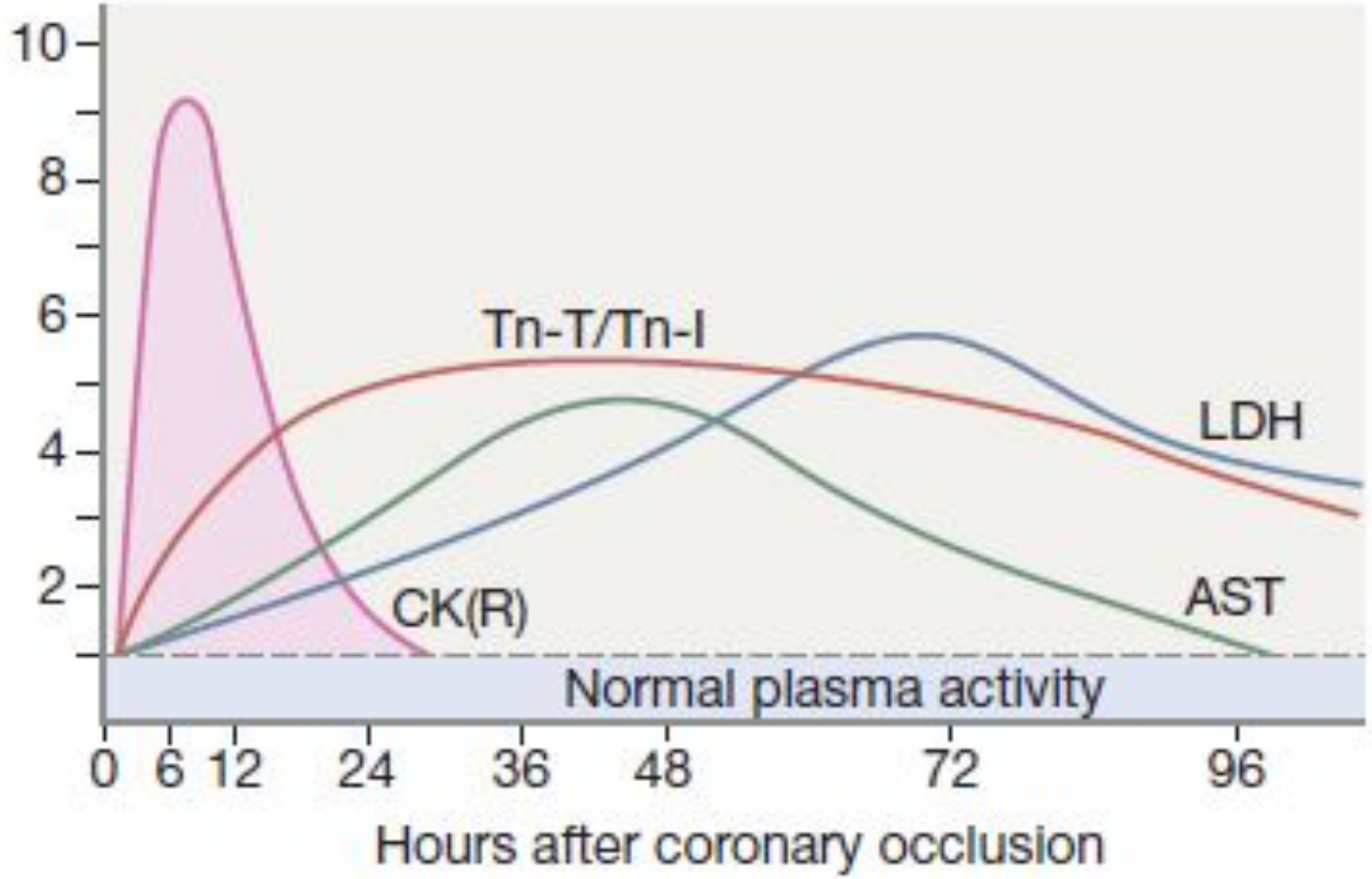
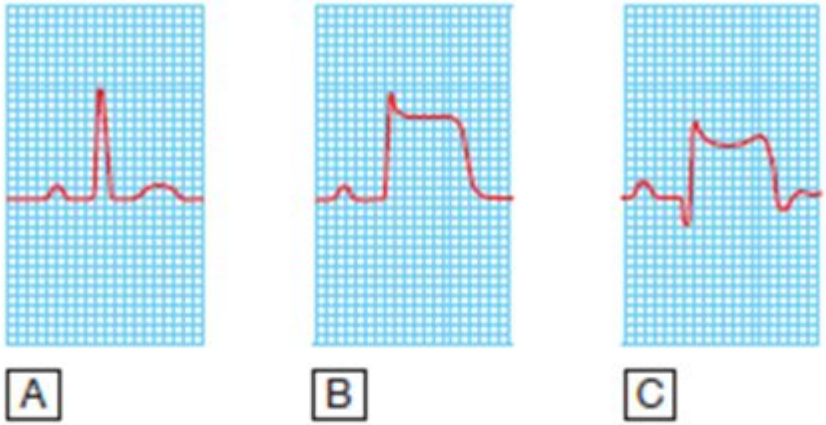


Fig. 16.69 Changes in plasma cardiac biomarker concentrations after myocardial infarction. Creatine kinase (CK) and troponins T (Tn-T) and I (Tn-I) are the first to rise, followed by aspartate aminotransferase (AST) and then lactate (hydroxybutyrate) dehydrogenase (LDH). In patients treated with reperfusion therapy, a rapid rise in plasma creatine kinase (curve CK(R)) occurs, due to a washout effect.

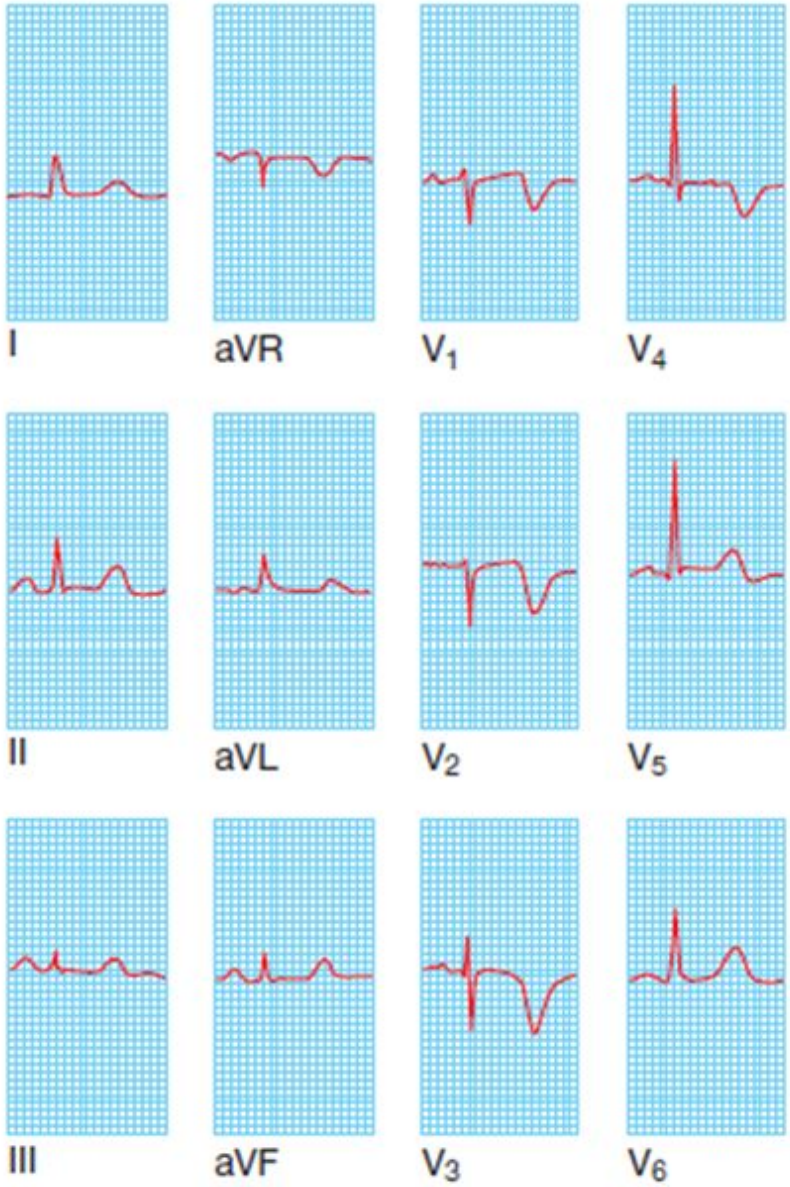
3. Other blood tests (WBC, ESR, & CRP may elevated)

4. Chest X-ray may demonstrate pulmonary oedema that is not evident on clinical examination. The heart size is often normal but there may be cardiomegaly due to pre-existing myocardial damage.

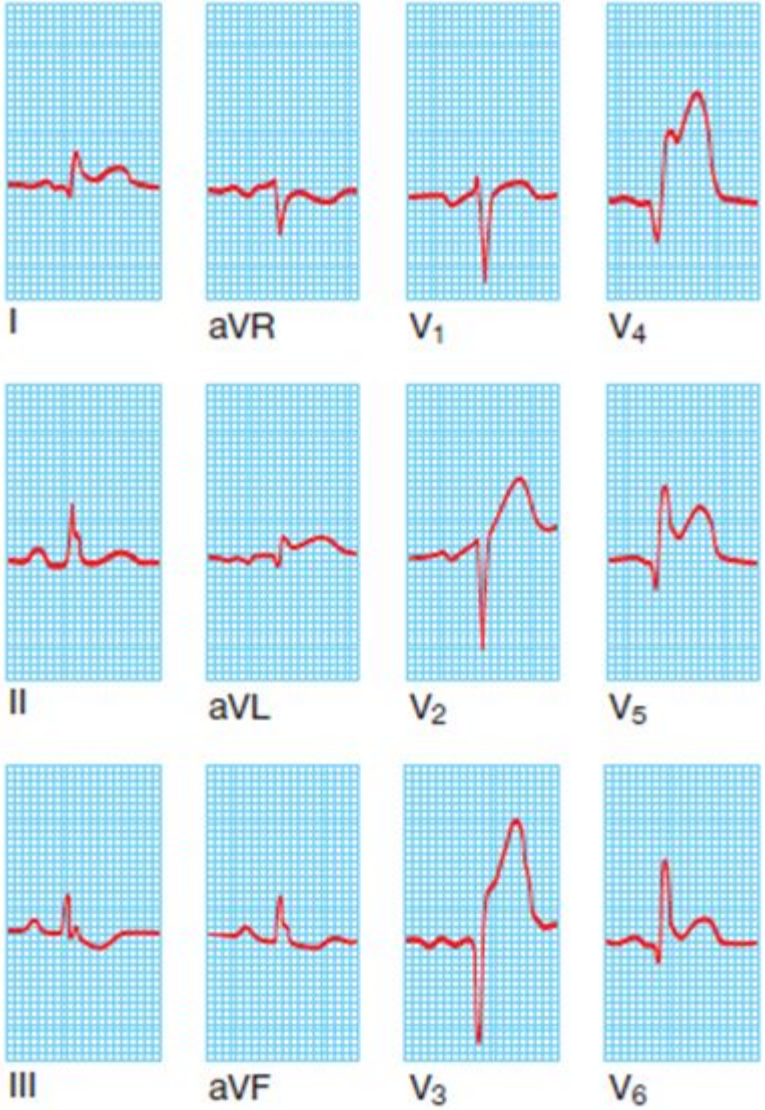
5. Echo for assessing ventricular function and for detecting important complications, such as mural thrombus, cardiac rupture, ventricular septal defect, mitral regurgitation and pericardial effusion



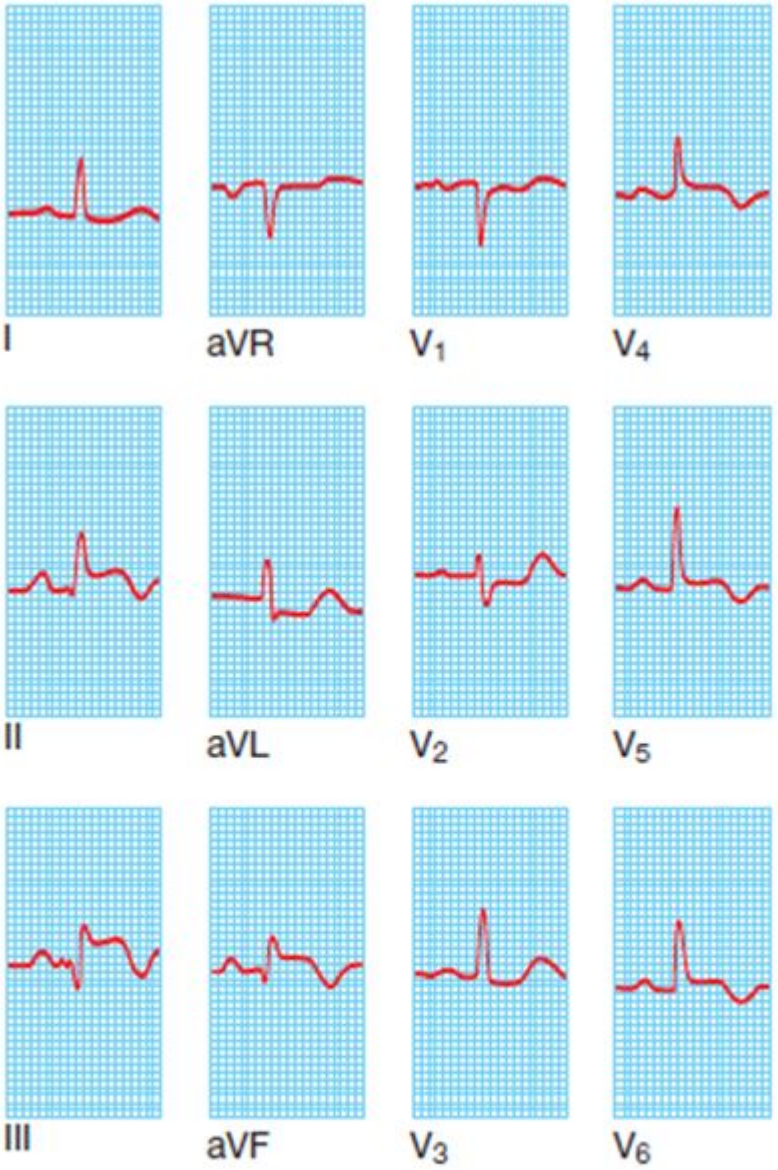
The serial evolution of ECG changes in transmural myocardial infarction. A Normal ECG complex. B Acute ST elevation ('the current of injury'). C Progressive loss of the R wave, developing Q wave, resolution of the ST elevation and terminal T-wave inversion. D Deep Q wave and T-wave inversion. E Old or established infarct pattern; the Q wave tends to persist but the T-wave changes become less marked. The rate of evolution is very variable but, in general, stage B appears within minutes, stage C within hours, stage D within days and stage E after several weeks or months.



Recent anterior non-ST elevation (subendocardial) myocardial infarction. This ECG demonstrates deep symmetrical T-wave inversion, together with a reduction in the height of the R wave in leads V_1, V_2, V_3 and V_4.



Acute transmural anterior myocardial infarction. This ECG was recorded from a patient who had developed severe chest pain 6 hours earlier. There is ST elevation in leads I, aVL, V_2, V_3, V_4, V_5 and V_6, and there are Q waves in leads V_3, V_4 and V_5. Anterior infarcts with prominent changes in leads V_2, V_3 and V_4 are sometimes called 'anteroseptal' infarcts, as opposed to 'anterolateral' infarcts, in which the ECG changes are predominantly found in V_4, V_5 and V_6.



Acute transmural inferolateral myocardial infarction. This ECG was recorded from a patient who had developed severe chest pain 4 hours earlier. There is ST elevation in inferior leads II, III and aVF and lateral leads V_4, V_5 and V_6. There is also 'reciprocal' ST depression in leads aVL and V_2.


Thank you
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