Risk stratification in stable angina.


Other forms of stress testing:

**Myocardial perfusion scanning:** useful when exercise test is not diagnostic, or patient can not exercise, its accuracy is higher than exercise test.

The technique involve obtaining scintiscans of the myocardium at rest and during stress after administration of an i.v radioactive isotope e.g thallium201, it is taken up by viable myocardium.

A perfusion defect present during stress but not rest indicates reversible myocardial ischemia, whereas persistent perfusion defect indicates previous M.I

**Stress echocardiography:**

It is alternative to perfusion scanning with similar accuracy.

Uses transthoracic echo to identify ischemic segments of myocardium & area of infarction, the latter do not contract at rest or during stress.

**Coronary arteriography:**

Shows the extent & nature of coronary artery disease, it may be indicated when other investigations fail to diagnose the cause of atypical chest pain.

Management

- Assessment of the extent & severity of arterial disease.
- Identification & control of significant risk factors
- Measures to control symptoms.
- Identification of high risk patients & application of treatment to improve life expectancy.

**Antiplatelet therapy:**

Aspirin 75-150 mg reduce the risk of MI, Clopidogrel 75 mg daily equally effective but more expensive can be used if the patient has dyspepsia.

**Anti-anginal drugs:**

- Nitrates: produces venous & arteriolar dilatation
- Decrease myocardial oxygen demand (lower preload & afterload) & increase myocardial oxygen supply.
- Sublingual glyceryl trinitrate (GTN), as aerosol 400 microgm or tablet 300-500 microgm sublingually usually relieve angina in 2-3 minutes, side- effects include headache, symptomatic hypotension & syncope, the tablet should be replaced 8 weeks after
the bottle has been opened.

- Nitrates can be used prophylactically before exercise.
- GTN is subject to extensive first pass metabolism in the liver, its ineffective when swallowed.
- Nitrate free period of 6-8 hr. every day to avoid tolerance.

<table>
<thead>
<tr>
<th>Preparation</th>
<th>Peak action</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sublingual GTN</td>
<td>4-8 mins</td>
<td>10-30 mins</td>
</tr>
<tr>
<td>Buccal GTN</td>
<td>4-10 mins</td>
<td>30-300 mins</td>
</tr>
<tr>
<td>Transdermal GTN</td>
<td>1-3 hrs</td>
<td>up to 24 hrs</td>
</tr>
<tr>
<td>Oral isosorbide dinitrate</td>
<td>45-120 mins</td>
<td>2-6 hrs</td>
</tr>
<tr>
<td>Oral isosorbide mononitrate</td>
<td>45-120 mins</td>
<td>6-10 hrs</td>
</tr>
</tbody>
</table>

**Beta-blockers**

- Reduce myocardial oxygen demand by reducing heart rate, BP, and myocardial contractility.
- Non-selective BB may exacerbate coronary spasm by blocking Beta 2 coronary adrenoceptors.
- Give once daily cardioselective preparation, atenolol 50-100 mg daily, slow release metoprolol 200 mg daily, bisoprolol 5-10 mg daily.
- BB should not be withdrawn suddenly as this may cause arrhythmia, more angina or MI. (BB withdrawal syndrome).

**Calcium antagonists:**

- lower myocardial oxygen demand by reducing blood pressure and myocardial contractility.
- Dihydropyridine calcium antagonists, such as nifedipine and nicardipine, often cause a reflex tachycardia; it is often best to use these drugs in combination with a β-blocker.
- In contrast, verapamil and diltiazem are particularly suitable for patients who are not receiving a β-blocker because they inhibit conduction through the AV node and tend to cause a bradycardia or even atrioventricular block in susceptible individuals.
- The calcium antagonists may reduce myocardial contractility and can aggravate or precipitate heart failure. Other unwanted effects include peripheral oedema, flushing, headache and dizziness.

**Potassium channel activators:**

This class of drug has arterial and venous dilating properties but does not exhibit the tolerance seen with nitrates. Nicorandil (10-30 mg 12-hourly orally) is the only drug in this class currently available for clinical use.

it is conventional to start therapy with low-dose aspirin, sublingual GTN and a β-blocker, and then add a calcium channel antagonist or a long-acting nitrate later, if necessary.
The goal is the control of angina with minimum side-effects and the simplest possible drug regimen. There is little or no evidence that prescribing multiple anti-anginal drugs is of benefit, and revascularisation should be considered if an appropriate combination of two drugs fails to achieve a symptomatic response.

Invasive treatment The most widely used invasive options for the treatment of ischaemic heart disease include percutaneous coronary intervention (PCI; including percutaneous transluminal coronary angioplasty, PTCA) and coronary artery bypass graft (CABG) surgery.

**UNSTABLE ANGINA**

is a clinical syndrome that is characterised by new-onset or rapidly worsening angina (crescendo angina), angina on minimal exertion or angina at rest.

- The condition shares common pathophysiological mechanisms with acute myocardial infarction,
- the term 'acute coronary syndrome' is used to describe these disorders collectively.
- These entities comprise a spectrum of disease that encompasses ischaemia with no myocardial damage, ischaemia with minimal myocardial damage, partial thickness (non-Q wave) myocardial infarction, and full thickness (Q wave) myocardial infarction.
- An acute coronary syndrome may present as a new phenomenon or against a background of chronic stable angina.
The culprit lesion is usually a complex ulcerated or fissured atheromatous plaque with adherent platelet-rich thrombus and local coronary artery spasm.

**Diagnosis and risk stratification**

- 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 and is the most useful method of initial triage.
- Evolving transmural infarction is characterised by persistent ST elevation, new Q waves or new left bundle branch block.
- In patients with unstable angina or partial thickness (non-Q wave or non-ST elevation) myocardial infarction, the ECG may show ST/T wave changes including ST depression, transient ST elevation and T-wave inversion; the T-wave changes are sometimes prolonged.
- Approximately 12% of patients with well-characterised unstable angina or non-ST segment elevation myocardial infarction progress to acute infarction or death, and almost one-third will suffer a recurrence of severe ischaemic pain, within 6 months of the index event.

**The risk markers that are indicative of an 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, mitral regurgitation) during episodes of ischaemia
- those who experience unstable angina following acute myocardial infarction are also at increased risk.

**Risk stratification** is important because it guides the use of more complex pharmacological and interventional treatment.

**High risk:**

**Clinical:**
- Post-infarct angina
- Recurrent pain at rest
- Heart failure

**ECG:**
- Arrhythmia
- ST depression
- Transient ST elevation
- Persistent deep T-wave inversion

**Biochemistry:**
- Troponin T > 0.1 μg/l
**Low risk:**

Clinical
- No history of MI
- Rapid resolution of symptoms

ECG:
- Minor or no ECG changes

Biochemistry:
- Troponin T < 0.1 microgm/l

**The initial treatment should include**

- bed rest,
- antiplatelet therapy (aspirin 300 mg followed by 75-325 mg daily long-term and clopidogrel 300 mg followed by 75 mg daily for 12 months,
- anticoagulant therapy (e.g. unfractionated or fractionated heparin)
- β-blocker (e.g. atenolol 50-100 mg daily or metoprolol 50-100 mg 12-hourly
- A dihydropyridine calcium antagonist (e.g. nifedipine or amlodipine) can be added to the β-blocker, but may cause an unwanted tachycardia if used alone; verapamil or diltiazem is therefore the calcium antagonist of choice if a β-blocker is contraindicated
- An intravenous infusion of unfractionated heparin (with dose adjusted according to the activated partial thromboplastin time) or weight-adjusted subcutaneous low molecular weight heparin (e.g. enoxaparin 1 mg/kg 12-hourly) should be given
- If pain persists or recurs, infusions of intravenous nitrates (e.g. GTN 0.6-1.2 mg/hr or isosorbide dinitrate 1-2 mg/hr) or buccal nitrates may help, but such patients should also be considered for early revascularisation.
- Refractory cases or those with haemodynamic compromise should be considered for a glycoprotein IIb/IIIa receptor antagonist (e.g. abciximab, tirofiban or eptifibatide), intra-aortic balloon pump or emergency coronary angiography.
- Most low-risk patients stabilise with aspirin, clopidogrel, heparin and anti-anginal therapy, and can be gradually mobilised. If there are no contraindications,
- exercise testing may be performed prior to or shortly following discharge.
- Coronary angiography should be considered with a view to revascularisation in all patients at moderate or high risk, including those who fail to settle on medical therapy, those with extensive ECG changes, those with an elevated plasma troponin and those with severe pre-existing stable angina.
- This often reveals disease that is amenable to PCI; however, if the lesions are not suitable for PCI the patient should be considered for urgent CABG.