

CASE 3

Short case number: 3_2_3

Category: Cardiovascular

Discipline: Medicine

Setting: Hospital_Urban

Topic: Coronary artery disease – chronic stable angina, pharmacology

Case

Ham Wong, aged 62 years present concerned about recurrent chest pain. The episodes started about three months ago. They were always mild and came on when he played tennis. He felt mild chest discomfort that only in hindsight he feels must have been heart pain. The discomfort always resolved after resting. However, recently the episodes have been more severe and take longer to go away after resting. He was prescribed GTN by his general practitioner, and has been urgently referred to you today for a full cardiac workup.

Questions

1. Outline the types of angina and how they are differentiated?
2. In dot format summarise the pathogenesis of ischaemic heart disease
3. What further history and examination is required?
4. What features would suggest the pain was cardiac in nature?
5. Using a table list the following drugs and their mechanism of action - nitrates, beta blockers, thrombolytics, antiplatelet agents?
6. What further investigations would you order?
7. What are the key principles of management?

Suggested reading:

- Colledge NR, Walker BR, Ralston SH, Penman ID, editors. Davidson's Principles and Practice of Medicine. 22nd edition. Edinburgh: Churchill Livingstone; 2014. Chapter 18.

Advanced Reading

- Kereiakes DJ. Teirstein PS. Sarembock IJ. Holmes DR Jr. Krucoff MW. O'Neill WW. Waksman R. Williams DO. Popma JJ. Buchbinder M. Mehran R. Meredith IT. Moses JW. Stone GW. The truth and consequences of the COURAGE trial. *Journal of the American College of Cardiology*. 50(16):1598-603, 2007 Oct 16.
<http://www.sciencedirect.com/science/article/pii/S0735109707026629>

ANSWERS

1. Outline the types of angina and how they are differentiated?

Stable angina

Symptom complex caused by transient myocardial ischaemia;
It occurs when there is an imbalance between myocardial O₂ supply and demand, usually due to coronary atheroma.

Unstable angina

Clinical syndrome characterised by new onset or rapidly worsening angina with minimal exertion or at rest;

Part of the spectrum of the acute coronary syndrome, which represents ischaemia with no myocardial damage, ischaemia with minimal myocardial damage or ischaemia with partial thickness myocardial damage or full thickness myocardial damage.

2. In dot format summarise the pathogenesis of ischaemic heart disease

- Ischaemia heart disease (AKA coronary artery disease) occurs as the result of numerous risk factors. The major factors are smoking, diabetes, hypercholesterolemia, hypertension and a family history of premature coronary artery disease (female <65, male<55)
- The clinical presentations follow development of atherosomatous plaques:
 - I. Activated endothelial cells express adhesion molecules and recruit inflammatory cells, especially monocytes
 - II. Monocytes migrate into the intima of the artery wall and differentiate into macrophages and ingest lipid into foam cells
 - III. Cytokines and growth factors produced by the activated macrophages induce smooth muscle cell migration into the intima; the migrating smooth muscle cells change from their contractile properties to properties that allow them to repair the damaged intima
 - IV. If the cells are successful, the lipid core is covered by cells to form a stable plaque of atherosclerosis
 - V. If the cells are not successful, inflammation predominates and the plaque becomes unstable and the fibrous cap thins
 - VI. The plaque becomes susceptible to mechanical stress and the irregular surface becomes a focus for platelet aggregation and thrombus formation
 - VII. The formation of thrombous leads to partial or complete obstruction of the artery lumen.
- Once obstruction to the lumen has occurred, there is deficient blood supply to the myocardium.

3. What further history and examination is required?

Further history must try and ascertain whether this could be a presentation of a myocardial infarction. This includes type and nature of the pain, radiation of the pain, whether there was a change in the pain with the application of the nitrate patch, associated physical symptoms such as breathlessness, light-headedness, nausea, vomiting, sweating, change in the pain with the application of the nitrate patch or a sense of fear or impending doom?

Exam for signs of sympathetic activation i.e. pallor, sweating and tachycardia, or vagal activation i.e. bradycardia and vomiting.

Check for pallor and cool peripheries, pulse (character and rate), blood pressure (hypotension), JVP, character of the apex beat, osculate for heart sounds (quiet S1, S3) and osculate the chest for crackles.

4. What features would suggest the pain was cardiac in nature?

Characteristics of cardiac chest pain include:

- Central chest pain
- Radiation to the neck, jaw, upper or lower arms
- Dull constricting, choking heavy pain that is sometimes described as breathlessness, squeezing, crushing, burning or aching
- Cardiac pain is often provoked or worsened by activity, emotion or a heavy meal.
- Often associated with breathlessness and autonomic disturbances such as pallor, sweating, nausea and vomiting.

5. Using a table list the following drugs and their mechanism of action - nitrates, beta blockers, thrombolytics, antiplatelet agents?

Medication	Mechanism of action
nitrates	Vascular smooth muscle relaxation to produce venous and arteriolar dilatation leading to reduced preload and increase myocardial O ₂ supply
β-blockers	Reduce heart rate, blood pressure and myocardial contractility therefore lowering myocardial demand for O ₂
thrombolytics	Dissolve blood clots by activating plasminogen thereby restoring coronary patency
anti-platelet agents aspirin IIb/IIIa platelet receptor inhibitors	Inhibit platelet aggregation

Note: lipid lowering therapy (e.g. statin) should also be considered.

6. What further investigations would you order?

Blood tests (FBC, EUC, LFT, coagulation screen, ESR, C-reactive protein, serial levels of troponins or if this was not available, CK, CKMB)

Serial ECG

Chest x-ray

Fasting blood Glucose, fasting lipid profiles

Echocardiography

7. What are the key principles of management?

The most important concern is that this patient is at risk of death or acute myocardial ischemia and therefore requires admission to hospital.

The initial management includes bed rest, antiplatelet medications, heparin, and a β-blocker.

Once the pain has stabilised, further investigations must be undertaken with a stress test.

If the pain fails to stabilize or there are ECG changes or there have been elevations in the serum markers of myocardial damage or the pre-hospital angina was very severe, an angiogram is indicated.