ANTIANGINAL DRUGS

- Angina pectoris: a characteristic sudden severe pressing chest pain radiating to the neck, jaw, back and arms
- Caused by coronary blood flow insufficient to meet the oxygen demands of the myocardium, leading to ischemia
- Angina is caused by varying combinations of increased myocardial demand and decreased myocardial perfusion
- The imbalance between oxygen delivery and demand may result :
  - During exertion
  - From a spasm of the vascular smooth muscle
  - From obstruction of blood vessels by atherosclerotic lesions

Myocardial Ischemia and Angina

- Myocardial ischemia: Insufficient blood flow through coronary arteries to heart leading to imbalance between oxygen supply and demand
- Angina Pectoris: Choking and squeezing pain in the chest produced by ischemia
- Myocardial Infarction (MI): Extreme form of ischemia leading to significant cardiac tissue damage and cell death

Angina pectoris

- The transient episodes of myocardial ischemia do not cause cellular death as in MI
- Chronic ischemia may lead to deterioration in cardiac function causing:
  - Heart failure
  - Arrhythmias
  - Sudden death
- Life style modifications especially cessation of smoking are important in treatment of angina

Angina

- Sometimes surgery might be needed like
  - Angioplasty: A procedure used to for widening a narrowed or blocked coronary heart artery
  - Coronary artery bypass grafting (CABG) surgery which creates a new path for blood to flow to the heart and avoid the occluded are of the coronary artery

Types of angina

1. Effort induced angina, classis or stable angina

   The pattern of chest pains and the amount of effort needed to trigger the chest pain do no change

2. Unstable angina

   Chest pain occurs with increased frequency, duration and intensity and is caused by progressively less effort

   (The most common cause of MI)

3. Prinzmetal, variant vasospastic or rest angina

   Uncommon, occurs at rest. Is due to sudden and unpredictable coronary artery spasm which decreases blood flow to the heart.

4. Mixed forms of angina

   Patients with advanced coronary artery disease may have angina episodes during effort and during rest

Acute coronary syndrome

- An emergency situation
- Commonly results from rupture of an atherosclerotic plaque and partial or complete thrombosis of a coronary artery
- If thrombosis occludes most of the blood vessel and if the occlusion is untreated, necrosis of the cardiac muscle may occur (myocardial infarction)

Antianginal drugs

1. Organic nitrates
2. β -Blockers
3. Calcium channel blockers

- These drugs lower oxygen demand of the heart by affecting
  - Blood pressure
  - Venous return
  - Heart rate
  - Contractility

Organic nitrates

- Isosorbide dinitrate
- Isosorbide mononitrate
- Nitroglycerin

Organic nitrates

- Cause a rapid reduction in myocardial oxygen demand
- Effective in stable and unstable and variant angina
- Mechanism of action
  - Inhibit coronary vasoconstriction or spasm increasing perfusion of the myocardium and relieving the angina
  - Relax the veins (vasodilation) reducing the preload and myocardial oxygen consumption

Organic nitrates

- For an angina attack caused by exercise or emotional stress sublingual or spray nitroglycerin is the drug of choice

- Nitroglycerin is destroyed by first pass effect and so is given sublingually, as spray or patches

- Adverse effects
  - Headache
  - High dose can cause postural hypotension, facial flushing, and tachycardia

β-Blockers

- β adrenergic blockers decrease the oxygen demand of the myocardium by lowering the rate and force of contraction of the heart
- β-Blockers suppress the activation of the heart by blocking β1 receptors
- They reduce the work of the heart by decreasing heart rate, contractility, cardiac output and blood pressure
- The demand for oxygen by the myocardium is reduced during exertion or rest
- They reduce the frequency and severity of angina attacks

β-Blockers

- Atenolol (β1 blocker)
- Metoprolol (β1-blocker)
- Propranolol (non-cardio selective, contraindicated in asthma)
- Selective β1-blockers are preferred
- β-Blockers can be used with nitrates to increase exercise duration and tolerance
- They should not be discontinued abruptly, to avoid rebound angina, myocardial infarction and hypertension

Calcium channel blockers

- Calcium channel blockers protect the tissue by inhibiting calcium entry into cardiac and smooth muscle cells of coronary and systemic arteries
- Cause vasodilation, reducing vascular resistance
- They decrease the myocardium oxygen consumption by reducing afterload
- They may worsen heart failure due to their negative inotropic effect
- Examples
  - Nifedipine
  - Verapamil
  - Diltiazm

MYOCARDIAL INFARCTION

STEMI

- ST-elevation MI
  - Complete interruption of regional myocardial blood flow
  - Elevation of ST segment
  - Diagnosis
    - Chest pain
    - ECG changes
    - High serum levels of troponin CK
    - Sweating
    - Weakness
    - 20% asymptomatic

Management of STEMI

- Restoring the cardiac oxygen supply in balance with oxygen demand
- The first hours are most critical
  - Risk of ventricular dysrrhythmias, heart failure and cardiogenic shock

Management of STEMI

- Oxygen
- Aspirin/Fibrinolytic therapy
- Morphine
- Beta blockers (Atenolol, metoprolol)
- Nitroglycerin
- Thrombolytics
- Percutaneous coronary intervention (PCI)
  Reperfusion therapy

- Adjuncts to reperfusion therapy
  - Anticoagluants (ex heparin)
  - Antiplatelets
  - ACE inhibitors or ARBs

Post-MI

All post-MI patients should take four drugs

- Beta Blocker
- ACE-I or ARB
- Anticoagulant or antiplatelet
- Statin