Diagnosis and Management of Acute Myocardial Infarction

- Acute Myocardial Infarction (AMI) occurs as a result of prolonged myocardial ischemia.

- Atherosclerosis leads to endothelial rupture or erosion that leads to platelet aggregation at the site. This aggregation of blood cells occludes the culprit coronary artery.

- Blood flow through the artery is suspended leading to myocardial ischemia.

- The pain associated with AMI usually lasts longer than 30 minutes.
Typical Symptoms

- Pain – centre of chest radiating to the arms, back, neck, jaw and stomach
- Shortness of breath
- Diaphoresis – sweating
- Nausea
- Light-headedness
Atypical Symptoms

• Weakness
• Unusual Fatigue
• Hyperventilation
• Dizziness
Differences in Symptoms

Women, the elderly and patients with a history of stroke, heart failure, diabetes and hypertension experience more atypical symptoms during myocardial ischemia.
Pathological Evolution of Myocardial Infarction

- Ischemic Insult
- Coagulation Necrosis (lack of oxygen causes cell death)
- Healing
- Scarring
- Remodelling
• Ischemic insult phase lasts 4 hours and within this period the tissue in the infarct maybe saved from necrosis if reperfusion occurs.

• At 6 hours after occlusion the myocytes start to change.

• The phase of coagulation necrosis lasts from 4 to 48 hours after infarction.
• Healing begins after 48 hours after the occlusive event and lasts 7 days.
• The scarring phase begins in approximately one week after the infarction
• The scarring phase depends on the infarct size and may last from 2 weeks to months (Cunnigham 2000)
Diagnosis

• Based on history- chest discomfort which is severe and prolonged and may be described as crushing, constricting or oppressive.

• Serial ECGs

• Cardiac marker change – indicative of cardiac muscle necrosis
ECG

• Atrial contractions show up as the P wave.
• Ventricular contractions show as a series known as the QRS complex.
• The third and last common wave in an ECG is the T wave. This is the electrical activity produced when the ventricles are recharging for the next contraction (repolarizing).
ECG

- The twelve lead ECG is central to diagnosis of MI because ST segment elevation on the ECG is indicative of myocardial injury.
Cardiac Markers

- **Cardiac markers** are biomarkers measured to evaluate cardiac conditions.
- They are often discussed in the context of myocardial infarction but other conditions can lead to an elevation in cardiac marker level.
- Measuring cardiac biomarkers is part of the process toward making a diagnosis for myocardial infarction.
Troponins

Troponins are protein complexes found in cardiac and skeletal muscles.
Consist of three subunits—troponin C, troponin I, and troponin T
Troponin I is actually found exclusively in the myocardium and is 100% sensitive to MI - It is elevated in MI
CK(MB) - Creatine Kinase

• CK–MB levels, along with total CK, are tested in persons who have chest pain to diagnose whether they have had a myocardial infarction.

• Since a high total CK could indicate damage to either the heart or other muscles, CK–MB helps to distinguish between these two sources.
Immediate Nursing care

• Improve oxygenation – oxygen therapy if prescribed and monitoring
• Pain Control – IVI morphine + maxalon for nausea
• Cardiac monitoring
• BP half hourly (in the initial stages this may be more frequent)
• Urine output
Medical interventions - limitation of infarct size

• Restoration of blood flow to the myocardium prevents the extension of infarction or re-infarction

• Early reperfusion with thrombolytics or primary PCI (percutaneous coronary intervention)
Primary angioplasty and STENT

- Once diagnosis of a myocardial infarction is made, the patient can be taken straight to the cardiac suite where he is prepared for an angioplasty and STENT.
- This procedure makes a correct diagnosis and it also gets rid of the blockage which is causing the infarct.
Videos

• Video 1 – medical illustration

• Video 2  real life video showing the management of this medical emergency.
Nursing Care Following PCI
Nursing care following PCI

- **Chest pain** may occur in up to 50% of patients after PCI. Potential causes include benign stent sensation, acute stent thrombosis, abrupt vessel closure, transient coronary spasms. **All episodes of chest pain should be reported to a physician.**

- **Continuous ECG monitoring** is used to assess acute ischemic events. Monitoring of ST segments. Standard 12-lead ECGs are obtained after PCI procedures and whenever a patient has new cardiac signs or symptoms.

- **Cardiac markers** may be ordered as part of an institution’s protocol, although any patient experiencing persistent chest pain or after complicated PCI should have serial tests for cardiac markers. Measurements of troponins have a higher sensitivity and specificity for diagnosis of acute myocardial infarction than do measurements of creatine kinase MB.

- **Early sheath removal** decreases the incidence of vascular and bleeding complications.
• **Bed rest:** guidelines vary with institutional policy, the technique used for hemostasis, the size of the sheath. 3 – 6 hours bed rest according to unit protocol are required.

• **Standard assessment of the PCI access sites includes:**
  monitoring the arterial and venous puncture sites for bleeding and haematoma.

• **Assessment of peripheral circulation:** includes evaluation of bilateral pulses for comparison, warmth and color of the affected extremity, and capillary refill.

• **Urine output:** the dye used in this procedure can cause damage to the kidneys so the patient is advised to drink and urine output is monitored.
• Patients with brachial puncture sites are on bed or chair rest with the arm straight for a minimum of 2 hours.

• Using the affected arm for blood pressure checks should be avoided during the recovery period.

• The brachial site is monitored for bleeding, swelling, distal pulses, sensation, and motor function.

• Weak pulses or lack of pulses could be a sign of arterial spasm. Numbness and weakness may indicate medial nerve compromise or impaired circulation. The puncture sites should be checked to determine if the dressing is too tight or if a hematoma is compressing a nerve.
Thrombolytic therapy – intravenous infusion given to the patient to break down the blood clot
rt-PA

• Fibrin selective agents – rt-PA (recombinant tissue plasminogen activator) is a drug of choice
• Produced by vascular endothelial cells
• Given in intermittent doses over an hour and a half
• Has a short half life therefore systemic anticoagulation with continuous intravenous heparin is given
Contraindications

- Previous haemorrhagic or stroke
- Active internal bleeding (this does not include mensis)
- Suspected aortic dissection (tear in the intima of the aorta)
- Severe uncontrolled hypertension
- Recent trauma (within 2-4 weeks)
- Recent internal bleeding
- Pregnancy
- Active peptic ulcer
Nursing Care post thrombolysis

• Chest discomfort – always order ECG, read then act accordingly
• Monitor BP as a drop in BP can occur
• Bleeding
• Allergic ReactionsDuring thrombolysis watch out for hypotension and bleeding plus allergic reactions
• Monitor for abnormal heart rhythm
Patient management post MI

• Pharmacological management – medications prescribed. Aspirin, clopidogrel, nitrates, calcium channel blockers, ACE inhibitors and lipid lowering drugs

• Lifestyle modifications

• Follow up care
• Address risk factors: diet, smoking, weight
• Explain medications and their function – Aspirin, beta blockers, ACE inhibitors, nitrates, statins
• Gradually increase level of activity
• Discuss with cardiologist return to work – advice of cardiologist usually 2 months after infarct
• Driving – can be resumed usually after first outpatient visit
• Sexual activity best avoided 1 month post MI
• Travel abroad discouraged for first 2 months