Diagnosis and Management of Acute Myocardial Infarction

- Acute MI occurs as a result of prolonged myocardial ischemia.
- The pain associated with MI usually lasts longer than 30 minutes.
- Atherosclerosis leads to endothelial rupture or erosion that leads to platelet aggregation at the site.
- A thrombus forms and vasoconstrictors are released.
Pathological Evolution of Myocardial Infarction

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

The endocardium increases in thickness.
Classification of MI’s

- Based on the location of the infarction and the layers of the heart involved
- Anterior MI’s occur with the occlusion of the left anterior descending artery (LAD) a major supplier of the anterior wall of the left ventricle
- Complications: Severe LV dysfunction and cardiogenic shock.
- Changes in heart rhythm – tachycardia and AV block (the LAD supplies most of the intraventricular septum with blood)
Inferior/Posterior infarction

- Result from the occlusion of the right coronary artery which supplies 80% to 90% of the patients.
- In the remaining 10% to 20% these MI result from the occlusion of the left circumflex artery.
- Complications include various degrees of heart block.
Diagnosis

• Based on history- chest discomfort which is sever and prolonged and may be described and crushing, constricting or oppressive. Pain may radiate in the neck. Jaw and left arm

• Serial ECGs

• Serum enzyme change – indicative of cardiac muscle necrosis
Common Physical Presentation

- General
  - Alert, anxious and often fatigued
- Skin
  - Cool, clammy diaphoretic
- Lungs
  - Dyspnoea +/- crackles
- Gastrointestinal
  - Nausea and vomiting
ECG

• The twelve lead ECG is central to diagnosis of MI because patient with ST segment elevation or new bundle branch block should receive immediate therapy.

• Diagnosis becomes difficult when there are pre-existing ECG abnormalities
Anterior AMI
Acute Inferior AMI
Intervention

• Thrombolytic therapy – recommended in patients without contraindications to restore coronary blood flow

• Primary coronary angioplasty – if performed in a timely manner by experienced cardiologists
Limitation of infarct size

• Early reperfusion with thrombolytics or primary PTCA

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

• Fall in two categories:

• Fibrin selective agents – rt-PA (recombinant tissue plasminogen activator)

• Non-selective agents - Streptonkinase
Streptokinase

- A product of haemolytic streptococci
- SK combines with circulating plasminogen and this creates a systemic lytic state with dissolution of recent thrombi
- Given in a dose of 1.5 million units infused over 30-60 minutes IV
- SK may trigger an antigenic response because of its foreign protein origin
- SK should not be used with patients with a known recent streptococcal infection and who received SK within the last 5 days to 6 months
rt-PA

- 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
• Severe uncontrolled hypertension
• Recent trauma (within 2-4 weeks)
• Recent internal bleeding
• Pregnancy
• Active peptic ulcer
Nursing

• Monitor for signs of reperfusion arrhythmias
• Common arrhythmias: Ventricular tachycardia, premature ventricular ectopics, heart blocks and sinus bradycardia
• Chest discomfort – always order ECG, read then act accordingly
• Monitor BP as a drop in BP can occur
• Bleeding
• Allergic Reactions
Immediate Nursing care

- Improve oxygenation – oxygen therapy 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
- During thrombolysis watch out for hypotension and bleeding plus allergic reactions (especially with SK)
Nursing care

Daily ECG s for the first three days – this will show the evolution of the infarct

Cardiac markers
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
Myoglobin

- Found in myocardium and skeletal muscles
- Releases into the circulation after damage to the heart or skeletal muscle
- High myoglobin levels are seen in skeletal muscle injury, severe burns and renal failure when this is not excreted
CKMB

• In acute MI inadequate oxygen delivery to the myocardium causes cell injury. CK diffused out from the cell into the blood

• CK MB is released from the damaged myocardial tissue
Cardiac Enzymes
• Respond to Pain – analgesia and ECG
• Observations – vital signs
• O₂ therapy
• Colour and respirations
• 4 hourly temperature
• Fluid intake and output
• Bed rest – 24 hours, bed to chair 48 hours then gentle mobilization
- Reassure about condition
- Allow visits as necessary
- Explain procedures and equipment
- Keep next of kin informed
Patient management post MI

• Pharmacological management – medications prescribed. Aspirin, 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
Post Myocardial Infarction Rehabilitation programme

• Usually starts 3 weeks after discharge and is of 12 weeks duration
• Ask patient to bring a relative with them
• On their first visit assess:
  o BP HR and symptoms of angina and breathlessness
  o Emphasis drug compliance and identify side effects
  o Try to define their psychological status eg. depression or anxiety
  o Establish smoking status ie stopped or considering stopping or re-started smoking
  o Weigh if necessary
Multidisciplinary education programme

- Diet and heart disease – dietician
- Cholesterol and heart disease – clinical biochemist
- Exercise and the heart – physiotherapist
- Drugs for the heart – pharmacist
- Blood pressure – cardiologist or physician
- Stress – rehabilitation nurse/ hospital psychologist
- Risk factors – rehabilitation nurse