



# **INDIRANI COLLEGE OF NURSING**

**ARIYUR,PUDUCHERRY.**

## **MEDICAL SURGICAL NURSING – I**

### **MYOCARDIAL INFARCTION**

**LEVEL OF STUDENTS: B.SC (N) II YEAR**

### **MYOCARDIAL INFARCTION**

#### **INTRODUCTION:**

- MI occurs when myocardial tissue is abruptly and severely deprived of oxygen. Ischemia can lead to necrosis of myocardial tissue if blood flow is not restored. Infarction does not occur instantly but evolves over several hours.
- Obvious physical changes do not occur in the heart until 6 hours after the infarction, when the infarcted area appears blue and swollen.
- After 48 hours, the infarct turns gray, with yellow streaks developing as neutrophils invade the tissue.
- By 8 to 10 days after infarction, granulation tissue forms.
- Over 2 to 3 months, the necrotic area develops into a scar; scar tissue permanently changes the size and shape of the entire left ventricle.
- Not all clients experience the classic symptoms of an MI.
- Women may experience atypical discomfort, shortness of breath, or fatigue and often present with non-ST-elevation myocardial infarction (NSTEMI) or T-wave inversion.

- An older client may experience shortness of breath, pulmonary edema, dizziness, altered mental status, or a dysrhythmia.

- NCLEX (772)

**DEFINITION:**

MI mostly affects the left ventricle. It usually results from sudden occlusion of coronary artery or one of its branches by thrombus over a preexisting atheromatous plaque.

Myocardial infraction is a diseased condition which is caused by reduced blood flow in a coronary artery due to atherosclerosis and occlusion of an artery by an embolus or thrombus.

-LEWIS (747)

**RISK FACTORS:**

1. Atherosclerosis
2. Coronary artery disease
3. Elevated cholesterol levels
4. Smoking
5. Hypertension
6. Obesity
7. Physical inactivity
8. Impaired glucose tolerance
9. Stress.

- NCLEX (PAGE NUMBER: 773)

**CAUSES:**

▶ **MODIFIABLE FACTORS**

▶ **NON- MODIFIABLE FACTORS**

**MODIFIABLE FACTORS**

- AGE
- SEX
- GENETIC PREDISPOSTION

**NON- MODIFIABLE FACTORS**

- ATHEROSCLEROSIS
- OBSEITY
- SMOKING
- DECRAESED SERUM HDL LEVELS
- STRESS
- SEDENTARY LIFE STYLE

### **SYMPTOMS:**

- Angina(Pain)- persistent and severe lasting over 30 minutes, radiating to arm, neck, jaw, shoulder and teeth.
- Associated nausea, vomiting,
- Up to one-third have no chest pain (particularly elderly, female and diabetics), they often present with LVF, collapse or syncope, confusion, or stroke. Many have upper epigastric discomfort only.
- Onset of new murmurs, gallop rhythm (S3) in chest examination.
- ECG changes.
- Diaphoresis
- Dyspnea
- Dysrhythmias
- Feelings of fear and anxiety
- Pallor, cyanosis, coolness of extremities

### **Diagnostic studies:**

1. Troponin level: Level rises within 3 hours and remains elevated for up to 7 to 10 days.
2. Total CK level: Level rises within 6 hours after the onset of chest pain and peaks within 18 hours after damage and death of cardiac tissue.
3. CK-MB isoenzyme: Peak elevation occurs 18 hours after the onset of chest pain and returns to normal 48 to 72 hours later.
4. Myoglobin: Level rises within 2 hours after cell death, with a rapid decline in the level after 7 hours.
5. White blood cell count: An elevated white blood cell count appears on the second day following the MI and lasts up to 1 week.

### **6. Electrocardiogram :**

- a. ECG shows either ST segment elevation MI (STEMI), T-wave inversion, or NSTEMI; an abnormal Q wave may also present.

b. Hours to days after the MI, ST- and T-wave changes will return to normal, but the Q-wave changes usually remain permanently.

### **HYPERACUTE PHASE**

- Increased ventricular activation time (i.e., start of R to peak of R > 0.45 sec); coving of ST segment, tall wide T waves.

### **EVOLVING PHASE**

- ST elevation; > 1mm in 2 limb leads and > 2 mm in 2 chest leads, reciprocal ST depression on opposite surface.
- Evolution of pathological Q waves, wide and > 25% height of following R waves.
- Symmetrical pointed deep T waves inversion.

### **CHRONIC PHASE:**

- Iso electric ST but persistent Q waves; normal T waves
- MI can involve RV but virtually never the atria. The part of myocardium affected can be implied from leads that show the changes.

<b>ECG Lead</b>	<b>Location of MI</b>
V1-3	Anteroseptal
V5-6, aVL	Anterolateral
V2-V4	Anterior
I,II, aVL, V6	Lateral
II, III, aVF	Inferior
V1, V4R	Right ventricle

Posterior MI is inferred from reciprocal changes in V1-V2 or typical ST changes in V7-V9. The reciprocal changes are wide R (reciprocal of Q) and concave ST depression (reciprocal of ST elevation) and wide tall upright T (reciprocal of T inversion). Posterior MI nearly always is accompanied with lateral or inferior wall MI.

RV Infraction: Occurs more often than appreciated and is accompanied by inferior MI. ST Elevation is seen in V1 greater than that of V2, V3, V4. R shows typical MI changes.

7. Cardiac catheterization to determine the extent and location of obstructions of the coronary arteries.

8. ECHO.

9. TMT.

### **MANAGEMENT**

- Sit up the patient in comfortable position
- Give O<sub>2</sub> by face mask, attach to cardiac monitor.
- Obtain IV access, draw blood for CBC, cardiac enzymes, glucose.
- Aspirin 300 mg PO.
- IV opioid analgesics titrated to effect, along with metoclopramide 10 mg IV.

### **ADMINISTER THROMBOLYSIS:**

- Indications – ST elevation > 1mm with typical chest pain;
- Contraindications – severe hypertension (>200/120 mm Hg) recent GI bleed, major surgery within weeks, pregnancy, recent major trauma, esophageal varices, severe liver disease, appearance of Q waves.

### **CHOICE OF THROMBOLYTIC AGENTS:**

- Streptokinase 1.5 MU IV infusion in 1 hour, with hydrocortisone 100 mg and chlorpheniramine 10 mg.
- Tenectapase – 30 mg for < 60 kg, 35 mg for 60-69 kg, 40 mg for 70-79 kg given IV over 10 seconds
- Give LMWH e.g. enoxaparin 1 mg/kg IV start or heparin through separate IV line.
- If symptoms persist, ECG shows extensions of infarction, myocardial dysfunction unrelieved refer for angiography and planning for angioplasty/CABG.
- Give aetanol 5 mg IV.
- For persistent pain give nitroglycerine IV infusion
- Give ACE inhibitor e.g., lisinopril 2.5 mg within 24 hours of MI if there is CHF or myocardial dysfunction in Echo.

### **SUBSEQUENT MANAGEMENT:**

- Daily examination of heart, lungs, legs (DVT).
- Daily ECG, cardiac enzymes, urea, creatine, electrolytes for 3 days
- Heparin prophylaxis 5000 units SC/ 12 hr till fully ambulatory

- Oral betablocker (50 mg metoprolol) to decrease heart rate to around 60/min, continued for atleast 1 year (reduces subsequent cardiac mortality by 25 %).
- Continue ACE inhibitor, start statin for 1 year (for parevention/plaque stabilization).
- Continue aspirin 75-150 mg daily indefinitely.
- Address modifiable risk factors.
- Risk satisfication by limited exercise ECG at 4 weeks.
- If uncomplicated MI discharge after 7 days of hospital stay with restriction on activity.

### **COMPLICATION OF MI:**

- Cardiac arrest, cardiogenic shock.
- Tachyarrhythmas
- Bradycardias and heart block
- LVF
- Pericarditis
- DVT and pulmonary embolism, systemic embolism
- Cardiac tamponade due to cardiac rupture
- VSD, MR (papillary muscle infarction)
- LV aneurysm (risk of clot with SE, recurrent VT)
- Dressler's syndrome (recurrent pleuro pericarditis, fever).

### **KEY NOTES**

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### **NCLEX QUESTIONS:**

1. A client with myocardial infarction suddenly becomes tachycardia, shows signs of air hunger, and begins coughing frothy, pink-tinged sputum. Which finding would the nurse anticipate when auscultating the client's breath sounds?

1. Stridor
2. Crackles
3. Scattered rhonchi
4. Diminished breath sounds



**Rationale:** Pulmonary edema is characterized by extreme breathlessness, dyspnea, air hunger, and the production of frothy, pink-tinged sputum. Auscultation of the lungs reveals crackles. Rhonchi and diminished breath sounds are not associated with pulmonary edema. Stridor is a crowing sound associated with laryngospasm or edema of the upper airway.

Test-Taking Strategy: Focus on the subject, breath sounds characteristic of pulmonary edema. Recalling that fluid produces sounds that are called crackles will assist you in eliminating the incorrect options.

2. A client with myocardial infarction is developing cardiogenic shock. Because of the risk of myocardial ischemia, what condition should the nurse carefully assess the client for?

1. Bradycardia
2. Ventricular dysrhythmias
3. Rising diastolic blood pressure
4. Falling central venous pressure.

**Rationale:** Classic signs of cardiogenic shock as they relate to myocardial ischemia include low blood pressure and tachycardia. The central venous pressure would rise as the backward effects of the severe left ventricular failure became apparent. Dysrhythmias commonly occur as a result of decreased oxygenation and severe damage to greater than 40% of the myocardium.

Test-Taking Strategy: Focus on the subject, cardiogenic shock, and note the words myocardial ischemia. Recall that ischemia makes the myocardium irritable, producing dysrhythmias. Also, knowledge of the classic signs of shock helps to eliminate the incorrect options.

**REFERANCE:**

- Lewis, Medical Surgical Nursing; 9<sup>th</sup> Edition; Elsevier,page number- 747-750.
- Linda anne, NCLEX RN - EXAMINATION; 7<sup>th</sup> Edition; Elsevier,page number- 772-790.