MYOCARDIAL INFARCTION
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Coronary artery disease is mostly due to atherosclerosis and can lead to a number of clinical status such as angina pectoris. Myocardial infarction, cardiac arrhythmias, heart failure and sudden death.

An acute M.I. or heart attack occurs when a portion of the cardiac muscle is deprived of an adequate supply of arterial blood, with it oxygen and nutrients for a long time, leading to death of tissues in that area. The most common site for M.I. is anterior wall of the left ventricle near the apex, resulting from thrombosis of descending branch of left coronary artery, the other sites are posterior wall of the left ventricle near the base and behind the posterior cusps of mitral valve, inferior wall M.I. is due to occlusion or right coronary artery.

DEFINITION
Acute M.I. otherwise known as coronary artery occlusion and heart attack is a life-threatening situation of localized necrotic areas within the myocardium.

ETIOLOGY
- Atherosclerosis – The most common cause. The precipitating causes are: - Thrombus / Emboli; Fatigue; Spasm of coronary artery; Sudden exertion; Emotional stress; Heavy meal etc.

RISK FACTORS:
(a) Non Modifiable Risk Factor
Age - above 40 years of age
Sex - Men are more prone than women
Family History - There is a familial tendency or genetic pre-disposition towards CAD.

(b) Modification Risk Factors:
Elevated lipid level in blood, Diet high in fat; Hypertension; Cigarette smoking; Obesity; D.M.; Psychological tension; Oral contraceptives

PATHO-PHYSIOLOGY:
When oxygen supply to the myocardium is diminished glycolysis takes place to meet the increased demand for oxygen. This causes lactic acid production, this lactic acid builds up in the body causing metabolic acidosis. Myocardium is sensitive to changes in P.H. and becomes less functional. As the myocardium dies with necrosis, cellular enzymes are thrown into the bloodstream which are detected by lab tests like CKMB, SGOT, LDH etc. The main changes that are noticed in ECG after M.I. are ST elevation/ depression, T Inversion and appearance of prominent Q Wave.

ZONES OF M.I.
(1) Ischemia: Here the resting of the myocardium is affected. There will be impaired blood supply to the myocardium but the myocardial cells can return back to normal state with treatment. ECG-Shows T wave inversion.
(2) Injury: There is a considerable loss of blood supply to the heart. If adequate treatment is given it can be brought back, otherwise it will lead to infarction.
(3) Infarction: The blood and oxygen supply is completely cut off for 20 mths. Myocardial death occurs and is irreversible. ECG show prominent Q wave, T wave inversion, ST elevation.

Pain: Crushing, severe, prolonged, unrelieved by rest or NGT, often radiating to one or both arms. Neck and back.

Shock: Systolic BP below 80 mm of Hg, grey facial colour, lethargy, cold, diaphoresis, cyanosis; tachycardia or bradycardia.

Oliguria: Urine flow less than 30 ml/hr.
Fever: Temperature rises within 24 hours and lasts for 3 to 7 days.
Apprehension: Great fear of death
Indigestion: Nausea and vomiting
Acute pulmonary edema: Sense of suffocation, dyspnea, orthopnea.

MANAGEMENT:
a) Medical Management: Major goals, of care for clients with acute M.I are
(1) Successful treatment of the acute attack and prompt alleviation of manifestation.
(2) Prevention of complications and further attack.
(3) Rehabilitation and education of the client and significant others.

The client who suffers an acute MI needs immediate admission to a hospital with coronary care unit. The medical management includes:
1. Pain relief is the priority need. Pain stimulates the autonomic nervous system and increases the myocardial demand for oxygen.
2. Vasodilators must be given to increase the blood flow to the myocardium.
3. Oxygen is administered to treat tissue hypoxia.
5. Anticoagulants are given to prevent the risk of embolism.
6. Stool softeners to reduce the straining at stool.
7. In case of shock vasopressors such as dopamine and dobutamine can be given to raise the B.P.
8. Thromboembolysis therapy by using streptokinase, urokinase or tissue plasminogen activator. It must be given within 3-6 hours after the onset of chest pain. After thrombolysis I/V heparin therapy is continued for 5-7 days.

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Nursing management:
The nursing priorities are
1. To relieve pain, anxiety.
2. To reduce myocardial workload
3. To prevent and assist in treatment of life threatening disarrhythmias.
4. To promote self care.

NURSING DIAGNOSIS, INTERVENTIONS AND RATIONALE

Pain related to tissue ischemia secondary to coronary occlusion manifested by complaints of chest pain, facial grimacing.

Intervention: Obtain full description of pain from patient including location, intensity, duration, quality and radiation.
Rationale: Pain is a subjective symptom and must be described by the patient.
Intervention: Instruct patient to report pain immediately.
Rationale: Delay in reporting pain hinders pain relief.
Intervention: Provide calm and quiet environment and other comfort measures.
Rationale: Decreased external stimuli may aggravate anxiety.
Intervention: Administer supplemental oxygen.
Rationale: Increases the oxygen supply to myocardium thereby relieving discomfort.

II. Anxiety/fear related to change in health and socio economic status manifested by apprehension, increased tension, restlessness, uncertainty etc.

Intervention: Note presence of hostility withdrawal or denial.
Rationale: Ongoing anxiety may be present manifested by depression.
Intervention: Encourage patient to communicate with one another; sharing questions.
Rationale: Sharing information may relieve tension of unexpressed worries.

Intervention: Answer all questions honestly.
Rationale: To win the confidence of the patient.

III. Altered tissue perfusion related to reduction of blood flow due to vasoconstriction manifested by thromboembolic formation.

Interventions: Inspect for pallor, cyanosis, cold and clammy skin.
Rationale: Systemic vasoconstriction resulting from decreased cardiac output may be evidenced by decreased skin perfusion.
Intervention: Assess for human’s sign, erythema and edema.
Rationale: Indicator for deep vein thrombosis.
Intervention: Monitor laboratory details eg: ABG’S, BUN.
Rationale: Indicators of organ perfusion.
Intervention: Prepare patient for thromboembolic therapy.
Rationale: To dissolve the clot and to restore perfusion of myocardium.

IV. Excess fluid volume related to increased sodium and water retention manifested by dependent edema.

Interventions: Measure intake output to detect whether there is decrease in output.
Rationale: Decreased cardiac output results in impaired kidney perfusion. Na/H2o retention and reduced urine output.
Intervention: Weigh daily
Rationale: Sudden changes in weight indicate fluid imbalance.
Intervention: Provide low sodium diet.
Rationale: Sodium will enhance fluid retention.

V. Knowledge deficit related to lack of factual information regarding implications of heart disease and future health status manifested by anxiety, worries, gloomy face etc.

Interventions: Assess patient’s level of knowledge and ability to learn.
Rationale: It is necessary to create individual instruction plan.
Intervention: Educate the patient about basic informations regarding M.I., its cause, prevention and management.
Rationale: Patient will gain adequate knowledge about his disease and will try to avoid a second attack.
Intervention: Emphasize on the importance of avoiding the risk factors of M.I.
Rationale: Modifiable risk factors can be prevented if we take adequate precautions.

OTHER NURSING MANAGEMENT:

Provide semi Fowler’s positions to promote chest expansion and comfort. Oxygen administration to treat tissue hypoxia. Check vital signs every 15 minutes. Watch for PVC (Premature Ventricular Contractions) in the ECG. Assess the L.O.C. Morphine is the drug of choice to relieve chest pain. Strict I/O chart. Bed rest. Sedation and hypnotics to relieve unnecessary anxiety. Clear liquid diet for 48 hours and thereafter soft bland diet. Cardiac enzymes should be repeated. Educate the patient to control diet high in fats and cholesterol.

REFERENCES
1. Medical Surgical Nursing, Luckman and Sorensen. IVth edition, Pg No. 1150-1164