University of Mosul

College of Nursing

Department of Adult Nursing

Subject Name : Critical Care Nursing

Grade : 4th

Semester : 1st & 2nd / 2024 – 2025

Stroke

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Stroke

❖ **Definition :** Sudden onset of acute neurological deficit persisting for more 24 hours and caused by the interruption of blood flow to the brain.

❖ Classification of Stroke:

1. Ischemic
2. Hemorrhagic.
   A. Subarachnoid hemorrhage.
   B. Intracerebral hemorrhage.

Ischemic Stroke

1. It results from interruption of blood flow to brain and accounts 80 – 85 % of all strokes.
2. Results by thrombotic or embolic event.
3. Embolic source include the heart (cardioembolic strokes) and atherosclerotic plaques.

❖ Risk factor for ischemic stroke

1. Hypertension.
2. Dyslipidemia.
3. Diabetes mellitus.
4. Smoking.
5. Carotid atherosclerotic disease.

❖ Etiology (embolic stroke)

1. Atrial fibrillation.
2. Mitral stenosis.
3. Mechanical valves.
4. Atrial myxoma.
5. Endocarditis.
6. Recent myocardial infarction.

❖ Pathophysiology

When cerebral blood flow is reduced to a level insufficient to maintain neuronal viability, ischemic injury occurs. In focal stroke, an area of hypoperfused tissue, the ischemic penumbra, surrounds a core of ischemic cells. The ischemic penumbra can be salvaged with return of blood flow. Sustained anoxic insult initiates a chain of biochemical events leading to apoptosis, or cellular death.

Cerebral edema is sufficient to produce clinical deterioration develops in 10% to 20% of patients with ischemic stroke and can results in intracranial hypertension. The edema results from a loss of normal metabolic function of the cells and peaks at 4 days. This process is commonly the cause of death during the first week after stroke. Secondary hemorrhage at the site of the stroke lesion, known as hemorrhagic conversion, and seizures are the two other major acute neurological complication of the ischemic stroke.

❖ Assessment and Diagnosis

1. **Signs of ischemic stroke**. Sudden onset of focal neurological signs persisting for more than 24 hours.
2. **Neurological Abnormalities in Acute Ischemic Stroke:**

A. Left (Dominant) Hemisphere.

- Aphasia.
- Right hemiparesis.
- Right – sided sensory loss.
- Right visual field defect.
- Poor right conjugate gaze.
- Dysarthria.
- Difficulty in reading and writing or calculating.

B. Right (Nondominant) Hemisphere.

- Neglect of the left visual space.
- Left visual field defect.
- Left hemiparesis.
- Left – sided sensory loss.
- Poor left conjugate gaze.
- Extinction of left – sided stimuli.
- Dysarthria.
- Spatial disorientation.

C. Brainstem, Cerebellum, and posterior hemisphere.

- Motor or sensory loss in all four limbs.
- Crossed signs.
- Limb or gait ataxia.
- Dysarthria.
- Disconjugate gaze.
- Nystagmus
- Amnesia.
- Bilateral visual field defects.

D. Small Subcortical Hemisphere or Brainstem (Pure Motor Stroke).

- Weakness of face and limbs on one side of the body without abnormalities of higher brain function, sensation, or vision.

E. Small Subcortical Hemisphere or Brainstem (Pure Sensory Stroke).

- Decreased sensation of face and limbs on one side of the body without abnormalities of higher brain function, motor function, or vision.

3. **Assessment neurological impairment**: through use the National Institutes of Health Stroke Scale (NIHSS)which include the following:
   A. Level of consciousness.
   B. LOC Question.
   C. LOC Commands.
   D. Gaze.
   E. Visual fields.
   F. Face, arm, and leg strength.
   G. Sensation.
   H. Limb ataxia.
   I. Language function.
4. **Diagnostic Procedure include:**
   A. Non – contrast CT scanning. (Excluding intracranial hemorrhage, assist in identifying early neurological complication and the cause of insult).
   B. MRI. (Demonstrate infarction of cerebral tissue earlier than CT, useful in emergency differential diagnosis).
   C. ECG.
   D. Chest Radiography.

E. Continuous cardiac monitoring.

F. Echocardiography. (Identifying cardioembolic phenomenon).

G. Electroencephalogram (if seizures are suspected).

H. Lumber puncture.

5. **Laboratory Evaluation:**

A. Hematological function.

B. Electrolyte and glucose level.

C. Renal and hepatic function.

D. Arterial blood gas analysis. (Performed if hypoxia is suspected).

❖ Medical Management

1. Thrombolytic therapy with intravenous recombinant tissue – type plasminogen activator (rtPA) within 3 to 4.5 hours of onset of ischemic stroke.
2. Conformation of diagnosis with CT must be accomplished before rtPA administration.
3. The recommended dose of rtPA is 0.9 mg / kg up to a maximum dose of 90 mg.
4. 10 % of the total dose is administrated as initial intravenous bolus , and the remaining 90% is administered by intravenous infusion over 60 minutes.

5. Unlike thrombolytic protocols for acute myocardial infarction , subsequent therapy with anticoagulant or antiplatelet agent is not recommended after rtPA administration in ischemic stroke.
6. Airway protection and ventilatory assistance to maintain adequate tissue oxygenation.
7. Hypertension is not lowered for the patient received thrombolytic therapy, antihypertensive therapy is considered only if the diastolic blood pressure is greater than 120 mmHg or the systolic blood pressure is greater than 220 mmHg
8. Intravenous Labetalol or Nicardipine is used to achieve blood pressure control.
9. Nitroprusside, Hydralazine, or Enalaprilat are used when antihypertensive not effective.
10. Body temperature and glucose level also must be normalized.
11. Management of cerebral edema and seizure activity.
12. Deep vein thrombosis (DVT) prophylaxis to decreased the risk of pulmonary embolism.

Subarachnoid Hemorrhage (SAH)

1. Is bleeding into the subarachnoid space, which is usually caused by rupture of a cerebral aneurysm or arteriovenous malformation.
2. Aneurysmal (SAH) is associated with mortality rate of 25 to 50 % with most patients dying on the first day after the insult.

❖ Classification of Subarachnoid Hemorrhage

1. **Grade (I) :** asymptomatic or minimal headache and slight nuchal rigidity.
2. **Grade (II) :** Moderate to severe headache , nuchal rigidity, but no neurological deficit other than cranial nerve palsy.
3. **Grade (III) :** drowsiness, confusion , or mild focal deficit.
4. **Grade (IV) :** stupor , moderate to severe hemiparesis, possible early decerebrate rigidity, and vegetative disturbances.
5. **Grade (V) :** deep coma , decerebrate rigidity , moribund appearance.

❖ Etiology

1. Cerebral aneurysm accounts for approximately 85% of all cases.
2. Traumatic injury.
3. Infectious material.

❖ Pathophysiology

1. **Cerebral Aneurysm.**

As the individual with congenital cerebral aneurysm matures, blood pressure rises, and more stress is placed on poorly developed and thin vessel wall. Ballooning of the vessels occurs, giving the aneurysm a berry like appearance.

Aneurysms are usually small , 2 to 7 mm in diameter , and often occur at the base of the brain on the circle of Willis. The aneurysm becomes so thin that it ruptures , sending arterial blood at a high pressure into the subarachnoid space.

For a brief moment after the aneurysm rupture , ICP is thought to approach mean arterial pressure, and cerebral perfusion decreases. In other situation, the unruptured aneurysm expands and places pressure on surrounding structures. This is particularly true with posterior communicating artery aneurysms , because they put pressure on the oculomotor nerve (cranial nerve III) , causing ipsilateral pupil dilation and ptosis.

2. **Arteriovenous Malformation.**

One or more cerebral arteries tends to enlarge over time , increasing both the volume of blood shunted through the malformation and the overall mass effect . large dilated tortuous draining veins develop as a results of increasing arterial blood flow being flow being delivered at a higher than normal pressure .

❖ Assessment and Diagnosis

1. Signs of subarachnoid hemorrhage:

A. Abrupt onset of pain, described as the " worst headache of my life".

B. Brief loss of consciousness.

C. Nausea.

D. Vomiting.

E. Focal neurological deficits.(third cranial nerve palsy, develops before aneurysm rupture).

F. Stiff neck may accompany headache.

G. Photophobia.

H. Dizziness or syncope.

2. Diagnostic Procedure include:

A. CT. findings
B. Lumber puncture results. (CSF, appear bloody and has a red blood cells count greater than 1000 cells / mm^3
C. Non – contrast CT., 95% of the cases CT can demonstrate blood in the subarachnoid space if performed within 48 hours of the hemorrhage.
D. MRI. is not routinely used , but it may provide greater sensitivity for detecting area of subarachnoid hemorrhage clot and potential location of blood.
E. Cerebral angiography is necessary to identify the exact cause of the hemorrhage.

❖ Medical Management

1. Airway management and ventilatory assistance may be necessary.
2. A ventriculostomy is performed to control ICP if the LOC is depressed.
3. Surgical clipping of aneurysms.
4. Surgical Excision of ateriovenous malformation.

Intracerebral hemorrhage (ICH)

1. Is bleeding directly into cerebral tissue, (ICH)destroy cerebral tissue causes cerebral edema, and increase ICP.
2. The source of Intracerebral bleeding is usually a small artery, but it can result also from rupture of an ateriovenous malformation (AVM)or aneurysm.
3. Spontaneous ICH is caused by hypertension and at least 10% of all stroke admissions.
4. The mortality rate of hemorrhagic stroke is up to 50% within 1 month.

❖ Risk factor for Intracerebral hemorrhage (ICH)

1. Age – associated cerebral amyloid angiopathy.
2. Hypertension.

❖ Etiology

1. Long – standing history of hypertension.
2. Anticoagulant or thrombolytic therapy.
3. Coagulation disorder.
4. Drug abuse.
5. Cerebral infarct.
6. Brain tumors.
7. Patient who discontinued antihypertensive medication 2 – 3 week before the hemorrhage.

❖ Pathophysiology

Continued elevated blood pressure exerting force against smaller arterial vessels that have become damaged from arteriosclerotic changes. Eventually , these arteries break , and blood bursts from the vessels into the surrounding cerebral tissue, creating a hematoma. ICP rises precipitously in response to the increase in overall intracranial volume.

❖ Assessment and Diagnosis

1. History and signs may reveals:

A. Patient unconsciousness and needs ventilator.

B. Sudden onset of focal deficit.

C. Severe headache.

D. Nausea.

E. Vomiting.

F. Rapid neurological deterioration.

2. Physical Assessment :

A. Assessment of vital signs usually reveals a severely elevated blood pressure (200 / 100 to 250 / 150 mmHg).

B. Signs of increased ICP are often presented by the time the patient arrives in the emergency department.

3. Diagnostic Procedure include:

A. CT. findings

B. Angiography for patient considered surgical candidates without clear cause of hemorrhage.

❖ Medical Management

1. Initial management requires attention to airway, breathing , and circulation .
2. Intubation is usually necessary.
3. Blood pressure management must be based on individual factors.
4. Reduction in blood pressure is usually necessary to decrease ongoing bleeding, but lowering blood pressure too much or too rapidly may compromise cerebral perfusion pressure.
5. Mean arterial blood pressure should be below 130 mm Hg in patient with history of hypertension.
6. Vasopressor therapy after fluid replenishment is recommended if systolic blood pressure falls below 90 mm Hg.
7. Mannitol for management of ICP.
8. Sedation for management of hyperventilation , and neuromuscular blockade.
9. Steroid should be avoided.

10. Body temperature is maintained at less than 38.5º C through use of acetaminophen or cooling blankets.
11. Euglycemia , a blood glucose level less than 140 mg / dL, is maintained with insulin therapy, but hyperglycemia should be avoided.
12. Use of short – acting benzodiazepines or propofol is recommended to treat agitation or hyperactivity.
13. Pneumatic compression devices are used to decrease risk of pulmonary embolism.
14. Anticonvulsant therapy is initiated if the patient experience seizures.
15. Surgical evacuation of the clot is recommended for the patients with cerebral hemorrhage greater than 3 cm with neurological deterioration or hydrocephalus with brainstem compression.

Nursing Management for Patient with Stroke

❖ Nursing priorities

1. Monitoring for changes in neurological and hemodynamic status.
   A. Monitor for neurological signs and vital signs
   B. Blood pressure cuff.
   C. Pulse oximeter.
   D. Seizure activity identified and treated.

2. Maintaining Surveillance for complications.

A. Monitor for signs of bleeding.

- Sudden onset of or an increase in headache and nausea and vomiting , increased blood pressure , and change in respiration herald the onset of rebleeding and indicate a second (SAH).

B. Precautions that prevent SAH, and precipitate rebleeding.

- Blood pressure control.
- Bed rest.
- Dark quiet environment.
- Use of stool softeners.
- Short – acting analgesic and sedative to relive pain and anxiety.
- Patient must be clam
- Limb restraining cause straining and must be avoided.
- The head of the bed should be elevated to 35 – 45 degrees at all time.

C. Indication of vasospasm.

- New focal or global neurological deficits.

D. Monitor for signs of increased ICP.

- Change in the level of consciousness.
- Other signs include : unequal pupil size, decreased pupillary response to light, headache, projectile vomiting, altered

breathing patterns, Cushing's triad (bradycardia , systolic hypertension, and bradypnea), diminished brainstem reflexes, papilledema, and abnormal extension (decerebrate posturing) or flexion (decorticate posturing).

E. Impaired Swallowing.

- Observed signs of dysphagia which include :
  - Drooling.
  - difficulty handling oral secretions.
  - absence of gag reflex.
  - absence of cough reflex.
  - absence of swallowing reflex.
  - Moist gurgling voice quality.
  - Decreased mouth and tongue movement
  - Presence of dysarthria.

F. Nursing measures applied to prevent stroke complication which include: Aspiration, Malnutrition, Pneumonia, DVT, Pulmonary embolism, Pressure ulcer, Contractures, and joint abnormalities.

3. Providing comfort and emotional support.

4. Educating the patient and family.

A. Patient and family must be taught about stroke , its causes, and its treatment.

B. Teach and focus on the interventions necessary for preventing the recurrence of the event and on maximizing the patient's rehabilitation potential.

C. Teach the patient's family how to feed, dress, and bath .

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Pulmonary Embolism

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Pulmonary Embolism

❖ **Definition:** A pulmonary embolism (PE) occurs when a clot (thrombotic embolus) or other matter (nonthrombotic embolus) lodges in the pulmonary arterial system, disrupting the blood flow to a region of the lungs.

❖ Origin of Thrombotic Emboli

The majority of thrombotic emboli arise from:

1. The deep leg veins, particularly the iliac, femoral, and popliteal veins.
2. Other sources include the right ventricle, the upper extremities, and the pelvic veins.

Nonthrombotic emboli arise from:

1. Fat.
2. Tumors.
3. Amniotic fluid.
4. Air.
5. Foreign bodies.

❖ Risk Factors for Pulmonary Thromboembolism

A. Predisposing Factors

I. Venous stasis
   1. Atrial fibrillation
   2. Decreased cardiac output (CO)
   3. Immobility

II. Injury to vascular endothelium
   1. Local vessel injury
   2. Infection
   3. Incision
   4. Atherosclerosis

III. Hypercoagulability
   1. Polycythemia

B. Precipitating Conditions

I. Previous pulmonary embolus

II. Cardiovascular disease
   1. Heart failure
   2. Right ventricular infarction
   3. Cardiomyopathy
   4. Cor pulmonale

III. Surgery
   1. Orthopedic
   2. Vascular
   3. Abdominal

IV. Cancer
   1. Ovarian
   2. Pancreatic
   3. Stomach
   4. Extrahepatic bile duct system

V. Trauma (injury or burns)
   1. Lower extremities
   2. Pelvis
   3. Hips

VI. Gynecologic status
   1. Pregnancy
   2. Postpartum
   3. Birth control pills
   4. Estrogen replacement therapy

❖ Pathophysiology

1. A massive PE occurs with the blockage of a lobar or larger artery, resulting in occlusion of more than 40% of the pulmonary vascular bed.
2. Blockage of the pulmonary arterial system has both pulmonary and hemodynamic consequences.
3. The effects on the pulmonary system are increased alveolar dead space, bronchoconstriction, and compensatory shunting.
4. The hemodynamic effects include an increase in pulmonary vascular resistance and right ventricular workload.

Increased Dead Space

An increase in alveolar dead space occurs because an area of the lung is receiving ventilation without being perfused. The ventilation to this area is known as *wasted ventilation*, because it does not participate in gas exchange. This effect leads to alveolar dead space ventilation and an increase in the work of breathing. To limit the amount of dead space ventilation, localized bronchoconstriction occurs.

Bronchoconstriction

Bronchoconstriction develops as a result of alveolar hypocarbia, hypoxia, and the release of mediators.

Alveolar hypocarbia occurs as a consequence of decreased carbon dioxide in the affected area and leads to constriction of the local airways, increased airway resistance, and redistribution of ventilation to perfused areas of the lungs. A variety of mediators are released from the site of the injury, either from the clot or the surrounding lung tissue, which further causes constriction of the airways. Bronchoconstriction promotes the development of atelectasis.

Compensatory Shunting

Compensatory shunting occurs as a result of the unaffected areas of the lungs having to accommodate the entire CO. This creates a situation in which perfusion exceeds ventilation and blood is returned to the left side of the heart without participating in gas exchange. This leads to the development of hypoxemia.

Hemodynamic Consequences

The major hemodynamic consequence of a PE is the development of pulmonary hypertension, which is part of the effect of a mechanical obstruction when more than 50% of the vascular bed is occluded. In addition, the mediators released at the injury site and the development of hypoxia cause pulmonary vasoconstriction, which further exacerbates pulmonary hypertension.

As the pulmonary vascular resistance increases, so does the workload of the right ventricle as reflected by a rise in pulmonary artery (PA) pressures. Consequently, right ventricular failure occurs, which can lead to decreases in left ventricular preload, CO, and blood pressure, and shock.

❖ Assessment and Diagnosis

- **Signs and symptoms**
  1. Tachycardia.
  2. Tachypnea.
  3. Dyspnea.
  4. Apprehension.
  5. Increased pulmonic component of the second heart sound (P1).
  6. Fever.
  7. Crackles.
  8. Pleuritic chest pain.
  9. Cough.
  10. Evidence of deep vein thrombosis (DVT).
  11. hemoptysis.
  12. Syncope and hemodynamic instability can occur as a result of right ventricular failure.
- **Initial laboratory studies and diagnostic procedures**
  1. ABG analysis: may show a low Pao2, indicating hypoxemia; a low Paco2, indicating hypocarbia; and a high pH, indicating a respiratory

alkalosis. The hypocarbia with resulting respiratory alkalosis is caused by tachypnea.

2. d-dimer: An elevated d-dimer will occur with a PE and a number of other disorders. A normal d-dimer will not occur with a PE and thus can be used to rule out a PE as the diagnosis.
3. Electrocardiogram (ECG): The most frequent ECG finding seen in the patient with a PE is sinus tachycardia. The classic ECG pattern associated with a PE—S wave in lead I, and Q wave with inverted T wave in lead III—is seen in fewer than 20% of patients. Other ECG findings associated with a PE include right bundle branch block, new-onset atrial fibrillation, T-wave inversion in the anterior or inferior leads, and ST segment changes.
4. chest radiography: Chest x-ray findings vary from normal to abnormal and are of little value in confirming the presence of a PE. Abnormal findings include cardiomegaly, pleural effusion, elevated hemidiaphragm, enlargement of the right descending pulmonary artery (Palla's sign), a wedge-shaped density above the diaphragm (Hampton's hump), and the presence of atelectasis.

5. echocardiography (ECHO). An ECHO, either transthoracic or transesophageal, is also useful in the identification of a PE, because it can provide visualization of any emboli in the central pulmonary

arteries. In addition, it can be used for assessing the hemodynamic consequences of the PE on the right side of the heart.

❖ Medical Management

A. Prevention strategies include

1. The use of prophylactic anticoagulation with low – dose or adjusted-dose heparin, LMWH, or oral anticoagulants .
2. The use of pneumatic compression has also been demonstrated as effective methods of prophylaxis in low-risk patients.

B. Treatment strategies include

1. Preventing the recurrence of a PE.
2. Facilitating clot dissolution, reversing the effects of pulmonary hypertension.
3. Promoting gas exchange, and preventing complications.
4. Medical interventions to promote gas exchange include supplemental oxygen administration, intubation, and mechanical ventilation.

Prevention of Recurrence

Interventions to prevent the recurrence of a PE include:

1. The administration of unfractionated or LMWH and warfarin (Coumadin).

2. Heparin is administered to prevent further clots from forming and has no effect on the existing clot. The heparin should be adjusted to maintain the activated partial thromboplastin time (aPTT) in the range of 2 to 3 times of upper normal.
3. Warfarin should be started at the same time, and when the international normalized ratio (INR) reaches 3.0, the heparin should be discontinued.
4. The INR should be maintained between 2.0 and 3.0. The patient should remain on warfarin for 3 to 12 months, depending on his or her risk for thromboembolic disease.
5. Interruption of the inferior vena cava is reserved for patients in whom anticoagulation is contraindicated. The procedure involves placement of a percutaneous venous filter (e.g., Greenfield filter) into the vena cava, usually below the renal arteries. The filter prevents further thrombotic emboli from migrating into the lungs.

Clot Dissolution

1. The administration of fibrinolytic agents in the treatment of PE has had limited success. Currently, fibrinolytic therapy is reserved for the patient with a massive PE and concomitant hemodynamic

instability. Either recombinant tissue-type plasminogen activator (rt-PA) or streptokinase may be used.

2. The therapeutic window for using fibrinolytic therapy is up to 14 days though the most benefit is usually obtained when given within 48 hours.
3. If the fibrinolytic therapy is contraindicated, a pulmonary embolectomy may be performed to remove the clot.
4. Generally it is performed as an open procedure while the patient is on cardiopulmonary bypass.
5. An emerging alternative to surgical embolectomy is catheter embolectomy. It appears to be particularly useful if surgical embolectomy is not available or is contraindicated.
6. It appears to be most successful when performed within 5 days of the occurrence of the PE.

Reversal of Pulmonary Hypertension

1. The administration of inotropic agents and fluid.
2. Fluids should be administered to increase right ventricular preload, which would stretch the right ventricle and increase contractility, thus overcoming the elevated pulmonary arterial pressures.
3. Inotropic agents also can be used to increase contractility to facilitate an increase in CO

❖ Nursing Management

1. Prevention of PE should be a major nursing focus, because the majority of critically ill patients are at risk for this disorder.
2. Nursing actions are aimed at preventing the development of DVT, which is a major complication of immobility and a leading cause of PE.

   These measures include the use of pneumatic compression devices, active/passive range-of-motion exercises involving foot extension, adequate hydration, and progressive ambulation.
3. Nursing interventions to optimize oxygenation and ventilation include positioning, preventing desaturation, and promoting secretion clearance.
4. The patient receiving anticoagulant or fibrinolytic therapy should be observed for signs of bleeding. The patient's gums, skin, urine, stool, and emesis should be screened for signs of overt or covert bleeding. In addition, monitoring the patient's INR or aPTT is critical to managing the anticoagulation therapy.
5. Early in the patient's hospital stay, the patient and family should be taught about pulmonary embolus, its etiologies, and its treatment . As the patient moves toward discharge, teaching should focus on:
   A. The interventions necessary for preventing the reoccurrence of DVT and subsequent emboli.
   B. Signs and symptoms of DVT and anticoagulant complications.
   C. Measures to prevent bleeding.

D. Encourage patient to stop smoking and be referred to a smoking cessation program.

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Status Asthmaticus

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Status Asthmaticus

- **Definition:** Asthma is a COPD that is characterized by partially reversible airflow obstruction, airway inflammation, and hyperresponsiveness to a variety of stimuli.
- **Definition:** Status asthmaticus is a severe asthma attack that fails to respond to conventional therapy with bronchodilators, which may result in ALF.

Etiology

1. The precipitating cause of the attack is usually an upper respiratory infection, allergen exposure, or a decrease in antiinflammatory medications.
2. Overreliance on bronchodilators, environmental pollutants, lack of access to health care, failure to identify worsening airflow obstruction, and noncompliance with the health care regimen.

Pathophysiology

1. An asthma attack is initiated when exposure to an irritant or trigger occurs, resulting in the initiation of the inflammatory immune response in the airways.
2. Bronchospasm occurs along with increased vascular permeability and increased mucus production.

3. Mucosal edema and thick, tenacious mucus further increase airway responsiveness.
4. The combination of bronchospasm, airway inflammation, and hyperresponsiveness results in narrowing of the airways and airflow obstruction.

Pulmonary Effects

As the diameter of the airways decreases, airway resistance increases, resulting in increased residual volume, hyperinflation of the lungs, increased work of breathing, and abnormal distribution of ventilation. V/Q mismatching occurs, which results in hypoxemia. Alveolar dead space also increases as hypoxic vasoconstriction occurs, resulting in hypercapnia.

Cardiovascular Effects

Inspiratory muscle force also increases in an attempt to ventilate the hyperinflated lungs. This results in a significant increase in negative intrapleural pressure, leading to an increase in venous return and pooling of blood in the right ventricle. The stretched right ventricle causes the intraventricular septum to shift, thereby impinging on the left ventricle. In addition, the left ventricle has to work harder to pump blood from the markedly negative pressure in the thorax to elevated pressure in systemic circulation. This leads to a decrease in CO and a fall in systolic blood pressure on inspiration (pulsus paradoxus).

❖ Assessment and Diagnosis

- Signs and Symptoms
  1. Cough.
  2. Wheezing.
  3. dyspnea.
  4. As the attack continues, the patient develops tachypnea, tachycardia, diaphoresis, increased accessory muscle use, and pulsus paradoxus greater than 25 mm Hg.
  5. Decreased level of consciousness, inability to speak, significantly diminished or absent breath sounds, and inability to lie supine herald the onset of ALF (Acute Lung Failure).
- Investigation
  1. Initial ABGs indicate hypocapnia and respiratory alkalosis caused by hyperventilation. Lactic acidosis also may occur as a result of lactate overproduction by the respiratory muscles. The end result is the development of respiratory and metabolic acidosis.
  2. A peak expiratory flow rate (PEFR) less than 40% of predicted or forced expiratory volume in 1 second (FEV1; maximum volume of gas that the patient can exhale in 1 second) less than 20% of predicted indicates severe airflow obstruction, and the need for intubation with mechanical ventilation may be imminent.

❖ Medical Management

A. Bronchodilators

1. Inhaled beta 2–agonists and anticholinergics are the bronchodilators of choice for status asthmaticus. Beta2-agonists promote bronchodilation and can be administered by nebulizer or metered-dose inhaler (MDI). Usually larger and more frequent doses are given, and the medication is titrated to the patient's response.
2. Anticholinergics in conjunction with beta2-agonists, they have a synergistic effect and produce a greater improvement in airflow.
3. The routine use of xanthines is not recommended in the treatment of status asthmaticus because they have been shown to have no therapeutic benefit.
4. A number of other studies are evaluating the effects of leukotriene inhibitors such as zafirlukast, montelukast, and zileuton in the treatment of status asthmaticus.

B. Systemic Corticosteroids

1. Intravenous or oral corticosteroids also are used in the treatment of status asthmaticus. Their anti-inflammatory effects limit mucosal edema, decrease mucus production, and potentiate beta2-agonists.
2. It usually takes 6 to 8 hours for the effects of the corticosteroids to become evident.
3. The use of inhaled corticosteroids for the treatment of status asthmaticus remains undecided at this time.

C. Oxygen Therapy

1. Initial treatment of hypoxemia is with supplemental oxygen.
2. High-flow oxygen therapy is administered to keep the patient's Spo2 greater than 92%.
3. Another therapy currently under investigation is the use of heliox. A mixture of helium and oxygen, heliox has a lower density and higher viscosity than an oxygen and air mixture. Heliox is believed to reduce the work of breathing and improve gas exchange because it flows more easily through constricted areas.

D. Intubation and Mechanical Ventilation

1. Indications for mechanical ventilation include cardiac or respiratory arrest, disorientation, failure to respond to bronchodilator therapy, and exhaustion.
2. A large endotracheal tube (8 mm) should be used to decrease airway resistance and to facilitate suctioning of secretions.
3. Ventilating the patient with status asthmaticus can be very difficult.
4. High inflation pressures should be avoided because they can result in barotrauma.
5. The use of PEEP should be monitored closely because the patient is prone to developing air trapping. Patient–ventilator asynchrony also can be a major problem.
6. Sedation and neuromuscular paralysis may be necessary to allow for adequate ventilation of the patient.

❖ Nursing Management

A. Optimizing Oxygenation and Ventilation

1. Positioning.
2. Preventing desaturation.
3. Promoting secretion clearance.

B. Educating the Patient and Family

1. As the patient moves toward discharge, teaching should focus on the interventions necessary for preventing the recurrence of status asthmaticus, early warning signs of worsening airflow obstruction, correct use of an inhaler and a peak flowmeter, measures to prevent pulmonary infections, and signs and symptoms of a pulmonary infection.
2. If the patient smokes, he or she should be encouraged to stop smoking and be referred to a smoking cessation program.

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Pain and Pain Management

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Pain and Pain Management

❖ Definition of pain

Pain is described as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage

❖ Components of Pain

The experience of pain includes:

1. Sensory: (perception of many characteristics)
   A. Intensity.
   B. Location.
   C. Quality.
2. Affective. (negative emotions)
   A. Unpleasantness.
   B. Anxiety.
   C. Fear.
   D. anticipation
3. Cognitive. Refers to the interpretation or the meaning of pain by the person who is experiencing it.
4. Behavioral: includes the strategies used by the person to express, avoid, or control pain.
5. Physiologic components: refers to nociception and the stress response.

❖ Types of Pain

1. Acute Pain

Acute pain has a short duration, and it usually corresponds to the healing process (30 days), but should not exceed 6 months. It implies tissue damage that is usually from an identifiable cause.

2. **Chronic Pain**

Chronic pain persists for more than 6 months after the healing process from the original injury, and it may or may not be associated with an illness. It develops when the healing process is incomplete or, as described earlier, when acute pain is poorly managed.

❖ **Type of Pain Origin**

1. Nociceptive Pain

Nociceptive pain arises from activation of nociceptors, and it can be somatic or visceral. Somatic pain involves superficial tissues, such as the skin, muscles, joints, and bones. Its location is well defined. Visceral pain involves organs such as the heart, stomach, and liver. Its location is diffuse, and it can be referred to a different location in the body.

2. Neuropathic Pain

Neuropathic pain arises from a lesion or disease affecting the somatosensory system.1The origin of neuropathic pain may be peripheral or central. Neuralgia and neuropathy are examples related to peripheral neuropathic pain, which implies a damage of the peripheral somatosensory system. Central neuropathic pain involves the central somatosensory cortex and can be experienced by patients after a cerebral stroke.

Neuropathic pain can be difficult to manage and frequently requires a multimodal approach (i.e., the combinations of several pharmacologic and/ or nonpharmacologic treatments).

Pain Assessment

Pain assessment is an integral part of nursing care. It is a prerequisite for adequate pain control and relief.

Pain assessment has two major components:

1. Nonobservable or subjective.
2. Observable or objective.

The Subjective Component

Refers to the patient's self-report about his or her sensorial, affective, and cognitive experience of pain. Because it is considered the most valid measure of pain, the patient's self-report must be obtained whenever possible.

A simple yes or no (presence versus absence of pain) is a valid self-report.

The mnemonic PQRSTU in Pain Assessment :

P: provocative and palliative or aggravating factors

Q: quality

R: region or location, radiation

S: severity and other symptoms

T: timing

U: understanding

P: Provocative and Palliative or Aggravating Factors

Deep breathing intensifying chest pain in the case of pericarditis is an illustration of an aggravating factor.

Q: Quality

pain as dull, aching, sharp, burning, or stabbing. This information provides the nurse with data regarding the type of pain the patient is experiencing (e.g., somatic or visceral). The differentiation between types of pain may contribute to the determination of cause and management.

A patient who has had open-heart surgery may complain of chest pain that is shooting or burning. This information can lead the nurse to investigate for cutaneous or bone injuries as a result of a sternotomy. Another patient may describe a sharp thoracic pain that may lead the nurse to consider visceral pain as a result of pulmonary embolism. A verbal description of pain is important because it provides a baseline account, allowing the critical care nurse to monitor changes in the type of pain, which may indicate a change in the underlying pathology.

R: Region or Location, Radiation

usually is easy for the patient to identify, although visceral pain is more difficult for the patient to localize. If the patient has difficulty naming the location or is mechanically ventilated, ask the patient to point to the location on himself or herself or on a simple anatomic drawing.

S: Severity and Other Symptoms

the severity or intensity of pain, is a measurement that has undergone much investigation. Many pain intensity scales are available, including the descriptive and numeric pain rating scales that are often used in the critical care environment. Many critical care units use a specific pain intensity scale.

Asking the patient to grade his or her pain on a scale of 0 to 10 is a consistent method and aids the nurse in objectifying the subjective nature of the patient’s pain.

T: Timing

refers to documenting the onset, duration, and frequency of pain. This information can help to determine whether the origin of the pain is acute or chronic.

Duration of pain can indicate the severity of the problem. For instance, chest pain of less than 15 minutes' duration may be angina, and pain lasting more than 15 minutes may indicate a myocardial infarction.

U: Understanding

is the patient's perception of the problem or cognitive experience of pain. Patients with known cardiac problems can tell the nurse whether their pain is the same as they had during myocardial infarction. Patients with a cerebral hemorrhage often describe experiencing the worst headache they have ever had.

Pain Assessment: The Observable or Objective Component

When the patient's self-report is impossible to obtain, nurses can rely on the observation of behavioral indicators, which are strongly emphasized in clinical recommendations and guidelines for pain management in nonverbal patients.

Behavioral Pain Scale

The BPS was tested mostly in nonverbal mechanically ventilated patients with altered levels of consciousness.

Its validity was supported with significantly higher BPS scores during nociceptive procedures (e.g., turning, endotracheal suctioning, peripheral venous cannulation) compared with rest or nonnociceptive procedures (e.g., arterial catheter dressing change, compression stocking applications, eye care).

Patient Barriers to Pain Assessment and Management

A. Communication

1. The patient who is mechanically ventilated cannot verbalize a description of the pain.
2. patients unable to self-report, the nurse relies on behavioral indicators to assess the presence of pain.

B. Altered Level of Consciousness and Unconsciousness

The patient either unconscious or with an altered level of consciousness presents a dilemma for all clinicians.

C. Older Adult Patients

1. Many older adult patients do not complain much about pain.
2. older adult patients with mild to moderate cognitive impairments and even some with severe impairment are able to use pain intensity scales.

D. Neonates and Infants

The emphasis in pain assessment should be on behavioral indicators. Physiologic indicators should be interpreted with caution because they are also affected by disease, medications, and physiologic status.

E. Cultural Influences.

patient speaks a language other than that of the health team members.

F. Lack of Knowledge

Many patients and their families are frightened by the risk of addiction to pain medication. They fear that addiction will occur if the patient is medicated frequently or with sufficient amounts of opioids necessary to relieve the pain.

Health Professional Barriers to Pain Assessment and Management

A. Addiction and Tolerance

- Addiction is defined by a pattern of compulsive drug use that is characterized by an incessant longing for an opioid and the need to use it for effects other than pain relief.
- Tolerance is defined as a diminution of opioid effects over time. Physical dependence and tolerance to opioids may develop if the medication is given over a long period. Physical dependence is manifested by withdrawal symptoms when the opioid is abruptly stopped. If this is an anticipated

problem, withdrawal may be avoided by weaning the patient from the opioid slowly to allow the brain to reestablish neurochemical balance in the absence of the opioid.

B. Respiratory Depression

the fear that aggressive management of pain with opioids will cause critical respiratory depression. Opioids can cause respiratory depression, but in the critically ill, this is a rare phenomenon.

PAIN MANAGEMENT

1. Pharmacologic Control of Pain

Pharmacologic management of pain has infinite variety in the critical care unit.

Pain pharmacology is divided into three categories of action:

1. Opioid agonists. The opioids most commonly used and recommended as firstline analgesics are the agonists.
   - **A. Morphine.** Morphine is the most commonly prescribed opioid in the critical care unit. Because of its water solubility, morphine has a slower onset of action and a longer duration compared with the lipid-soluble opioids (e.g., fentanyl). A more serious side effect requiring diligent monitoring is the respiratory depressant effect.
   - **B. Fentanyl** is a synthetic opioid preferred for critically ill patients with hemodynamic instability or morphine allergy.
2. Nonopioids.
   - **A. Acetaminophen** is an analgesic used to treat mild to moderate pain. It inhibits the synthesis of neurotransmitter prostaglandins in the CNS, and this is why it has no antiinflammatory properties.

B. Nonsteroidal Antiinflammatory Drugs. The use of NSAIDs in combination with opioids is indicated in the patient with acute musculoskeletal and soft tissue inflammation.

2. Nonpharmacologic Methods of Pain Management

A. Cold Application

Ice therapy was found to be helpful to reduce procedural pain in critically ill patients.

B. Massage

The effect of massage on pain relief

3. Cognitive-Behavioral Techniques

A. Relaxation

Relaxation is a well-documented method for reducing the distress associated with pain. Although not a substitute for pharmacology, relaxation is an excellent adjunct for controlling pain.

Relaxation decreases oxygen consumption and muscle tone, and it can decrease heart rate and blood pressure. Relaxation gives the patient a sense of control over the pain and reduces muscle tension and anxiety. Not all patients are interested in relaxation therapy. For those patients, deep-breathing exercises may be helpful.

B. Music Therapy

Music therapy is a commonly used intervention for relaxation. Music that is pleasing to the patient may have soothing effects, but its effects on reducing pain are controversial.

University of Mosul

College of Nursing

Department of Adult Nursing

Subject Name : Critical Care Nursing

Grade : 4th

Semester : 1st & 2nd / 2024 – 2025

Coma

Dr. Saad Hussein Murad Al – Shammary

Coma

Normal consciousness = Awareness and arousal

- Awareness = cognition (mental and intellectual) + affect of (mood) .
- Alteration of consciousness result of deficit in awareness, arousal or both.
- Disorder of consciousness (four discrete disorder)
  1. Coma : absence of both wakefulness and awareness.
  2. Vegetative state: presence of wakefulness with absence of awareness.
  3. Minimally conscious state: presence of wakefulness and severe diminished of awareness but not absent.
  4. Locked – in syndrome: presence of wakefulness and awareness

- **Definition of coma :** is the deepest state of unconscious ; arousal and awareness are lacking. It's a symptom rather than disease, and it occurs as results of some underlying process.

- **Etiology of coma**

  divided into 2 general categories

  I. Structural or Surgical.
     1. Ischemia stroke, Intracerebral hemorrhage (ICH), Brain Tumors, Epidural hematoma, Subdural hematoma, Brain contusion, Subarachnoid hemorrhage, Posterior fossa hemorrhage , Supratentorial hemorrhage, Hydrocephalus
     2. Trauma.
     3. Diffuse axonal injury.

II. Metabolic or Medical.

1. Drug over dose, Opioid overdose, Alcohol
2. Infectious disease.
3. Endocrine disorder.
4. Poisoning.
5. Meningitis, Encephalitis, Metabolic encephalopathy
6. Metabolic conditions.
7. Hypoglycemia, Hyperglycemia, Hyponatremia, Hypercalcemia
8. Hyperosmolar states.
9. Uremia.
10. Hepatic encephalopathy.
11. Hypertensive encephalopathy.
12. Hypoxic encephalopathy.
13. Myxedema.
14. Intoxication.
15. Psychogenic causes.

❖ Pathophysiology

Ascending fibers of the reticular activating system (ARAS) in the pons, thalamus maintain arousal as an autonomic function. Neurons in the cerebral cortex are responsible for awareness. Diffuse dysfunction of both cerebral hemispheres and diffuse or focal dysfunction of the reticular activating system can produce coma. Structural causes usually produce compression or dysfunction in the area of the ARAS, whereas most medical causes lead to general dysfunction of both cerebral hemispheres

❖ Assessment and Diagnosis

1. Assessment of level of consciousness.

2. Medical history.
3. Physical examination.
4. Neurological examination.

A. Assessment of pupillary size and reaction to light (normal , sluggish, or fixed).

B. Assessment of extraocular eye movements (normal , asymmetrical , or absent).

Abnormal Pupillary response

- **Damage in midbrain region =** Pupils slightly enlarged and unresponsive to light.
- **Lesion compress third nerve =** fixed and dilated pupil on the same side as the neurological insult.

C. Assessment of motor response to pain (normal , decorticate, decerebrate, or flaccid).

Abnormal Posture Findings

- Decorticate posturing (abnormal flexion) = damage to the diencephalon.
- Decerebrate posturing (abnormal extension)= damage to the midbrain and pons.
- Flaccid posturing = damage to the medulla .

D. Assessment of breathing pattern (Cause structural or metabolic).

Abnormal breathing patterns

- Cheyne – Stokes respiration = cerebral hemispheric dysfunction or metabolic suppression.
- Central neurogenic hyperventilation or Kussmaul breathing = metabolic acidosis or damage to the midbrain and upper pons.
- Apneustic breathing= damage to the pons, hypoglycemia, and anoxia.
- Ataxic breathing= damage to the medulla.
- Agonal breathing = failure of the respiratory center in the medulla.

- Diagnostic procedure:
  1. Computed Tomography (CT).
  2. Magnetic Resonance Imaging (MRI).
- Medical Management
  1. Emergency measures to support vital functions and prevent further neurological deterioration.
  2. Protection of the airway and ventilator assistance are often needed.
  3. Administration of thiamine (at least 100mg) , glucose, and opioid antagonists is suggested when cause is not immediately known.
  4. Thiamine is administered before glucose , because the coma produced by thiamine deficiency , Wernike's encephalopathy, can be precipitated by a glucose load.
  5. Supportive measures to maintain physiological body functions and prevent complications.
  6. Intubation for continued airway protection and nutritional support are essential .
  7. Fluid and electrolyte management is often complex because of alteration in the neurological system.
  8. Anticonvulsant therapy may be necessary to prevent further ischemic damage to the brain.
- Nursing Diagnosis Priorities.
  1. Ineffective Airway Clearance related to excessive secretions or abnormal viscosity of mucus.
  2. Imbalanced Nutrition: Less than Body Requirements related to lack of exogenous nutrients or increased metabolic demand.
  3. Risk for Aspiration.

4. Risk for infection.
5. Compromised Family Coping related to critically ill family member.

❖ Nursing Management

1. Monitoring for changes in neurological status and clues to the origin of the coma.
2. Supporting all body functions.
   A. Promoting pulmonary hygiene.
   B. Maintaining skin integrity.
   C. Initiating range – of – motion exercise.
   D. Managing bowel and bladder function.
   E. Ensuring adequate nutritional support.
3. Maintaining surveillance for complications.
4. Providing comfort and emotional support
5. Initiating rehabilitation measures.

▪ EYE CARE

1. The eyelid may be flaccid and may depend on body positioning to remain in a closed position, and edema may prevent complete closure.
2. Loss of these protective mechanisms results in drying and ulceration of the cornea, which can lead to permanent scarring and blindness.
3. Protect the eyes are instilling saline every to 2 hours, or methylcellulose lubricating drops and taping the eyelids in the shut position.
4. A polyethylene film is taped over the eyes, extending beyond the orbits and eyebrows. The film creates a moistening chamber around the cornea and assists in keeping the eyes moist and the closed position.

❖ Collaborative Management

1. Identify and treat the underlying cause.

2. Protect the airway.
3. Provide ventilator assistance, as required.
4. Support circulation, as required.
5. Initiate nutritional support.
6. Provide eye care.
7. Protect skin integrity.
8. Initiate range – of – motion exercise.
9. Maintain surveillance for complications:
    A. Infection.
    B. Metabolic alterations.
    C. Cardiac dysrhythmias.
    D. Temperature alterations.
10. Provide comfort and emotional support.
11. Plan for the rehabilitation program.

University of Mosul

College of Nursing

Department of Adult Nursing

Subject Name : Critical Care Nursing

Grade : 4th

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Guillain-Barré Syndrome (GBS)

Dr. Saad Hussein Murad Al – Shammary

Guillain-Barré Syndrome (GBS)

❖ **Description :** inflammatory peripheral neuropathy, is a combination of clinical features with various forms of presentation and multiple pathologic processes.

- involves a rapidly progressive, ascending peripheral nerve dysfunction, which leads to paralysis that may produce respiratory failure.
- The annual incidence of GBS is 1.8 cases per 100,000 persons.
- It occurs more often in males and is the most commonly acquired demyelinating neuropathy.
- Clusters of cases are reported, as occurred following the 1977 swine flu vaccinations.

❖ Etiology

1. The precise cause of GBS remains unknown, but the syndrome involves an immune-mediated response involving cell-mediated immunity and development of immunoglobulin G (IgG) antibodies.
2. Most patients report a viral infection 1 to 3 weeks before the onset of clinical manifestations, usually involving the upper respiratory tract.
3. Viral infections (e.g., influenza virus; cytomegalovirus; hepatitis A, B, or C virus; Epstein- Barr virus; human immunodeficiency virus), bacterial infections (e.g., gastrointestinal *Campylobacter jejuni*, *Mycoplasma pneumoniae*), vaccines (e.g., rabies, tetanus, influenza), lymphoma, surgery, and trauma.

❖ Pathophysiology

1. GBS affects the motor and sensory pathways of the peripheral nervous system as well as the autonomic nervous system functions of the cranial nerves.

2. The major finding in AIDP-type GBS is a segmental demyelination process of the peripheral nerves.
3. GBS is thought to be an autoimmune response to antibodies formed in response to a recent physiologic event. T cells migrate to the peripheral nerves, resulting in edema and inflammation.
4. Macrophages then invade the area and break down the myelin.
5. Inflammation around this demyelinated area causes further dysfunction. Some axonal damage also occurs.

❖ Assessment and Diagnosis

A. Signs & Symptoms

1. Motor weakness.
2. Paresthesias and other sensory changes.
3. Cranial nerve dysfunction (especially oculomotor, facial, glossopharyngeal, vagal, spinal accessory, and hypoglossal), and some autonomic dysfunction.
4. The usual course of GBS begins with an abrupt onset of lower extremity weakness that progresses to flaccidity and ascends over a period of hours to days.
5. Motor loss usually is symmetric, bilateral, and ascending.
6. In the most severe cases, complete flaccidity of all peripheral nerves, including spinal and cranial nerves, occurs.
7. The patient is admitted to the hospital when lower extremity weakness prevents mobility.

B. Physical Examination

1. Frequent assessment of the respiratory system(ventilatory parameters such as inspiratory force and tidal volume).

2. Frequent assessment of neurologic deterioration is continued until the patient reaches the peak of the disease, and a plateau occurs.

C. Diagnosis

1. CSF analysis and nerve conduction studies.
2. The diagnostic finding is elevated CSF protein with normal cell count.
3. The increased protein count usually occurs after the first week but does not occur in approximately 10% of all cases.
4. Nerve conduction studies that test the velocity at which nerve impulses are conducted show significant reduction, as the demyelinating process of the disease suggests.

❖ Medical Management

1. With no curative treatment available, the medical management of GBS is limited.
2. The disease must run its course, which is characterized by ascending paralysis that advances over 1 to 3 weeks and then remains at a plateau for 2 to 4 weeks.
3. The plateau stage is followed by descending paralysis and return to normal or near-normal function.
4. The main focus of medical management is the support of bodily functions and the prevention of complications.
5. Plasmapheresis and intravenous immune globulin (IVIG) are used to treat GBS. Plasmapheresis involves the removal of venous blood through a catheter, separation of plasma from blood cells, and reinfusion of the cells plus autologous plasma or another replacement solution. Although the number of exchanges may vary, four to six exchanges usually are

performed over a 5- to 8-day period. IVIG has emerged as the preferred therapy because of convenience and availability. The usual dose is 0.4 mg/kg for 5 days.

❖ **Nursing Management**

Goal of nursing management

- support all normal body functions until the patient can do so on his or her own. the condition is reversible, the patient with GBS requires extensive long-term care because recovery can be a long process.
- Focus on maintaining surveillance for complications, initiating rehabilitation, facilitating nutritional support, providing comfort and emotional support, and educating the patient and family.

❖ **Nursing Intervention**

A. Maintaining Surveillance for Complications

1. Continuous assessment of the progressive paralysis associated with GBS is essential to timely intervention and the prevention of respiratory arrest and further neurologic insult.
2. After the patient is intubated and placed on mechanical ventilation, close observation for pulmonary complications such as atelectasis, pneumonia, and pneumothorax is necessary.
3. Autonomic dysfunction (dysautonomia) in the GBS patient can produce variations in heart rate and blood pressure that can reach extreme values.
4. Hypertension and tachycardia may require beta-blocker therapy.

B. Initiating Rehabilitation

1. immobility may last for months. The usual course of GBS involves an average of 10 days of symptom progression and 10 days of maximal level of dysfunction, followed by 2 to 48 weeks of recovery.
2. The patient will require physical and occupational rehabilitation because of the problems of longterm immobility. Rehabilitation starts in the critical care area, with a multidisciplinary team designing and implementing an individualized plan for maximizing the patient's potential for rehabilitation.

C. Facilitating Nutritional Support

1. Nutritional support is implemented early in the course of the disease.
2. Nutritional support usually is accomplished through the use of enteral feeding.

D. Providing Comfort and Emotional Support

1. Pain control is another important component in the care of the patient with GBS.
2. Because of the length of this illness, a safe, effective, long-term solution to pain management must be identified.
3. patients also require extensive psychologic support.
4. Patient interaction and communication are essential elements of the nursing management plan.

E. Educating the Patient and Family

1. Early in the patient's hospital stay, the patient and family must be taught about GBS and its different treatments .
2. As the patient moves toward discharge, teaching focuses on the interventions to maximize the patient's rehabilitation potential.
3. The patient's family must be encouraged to participate in the patient's care and to learn some basic rehabilitation techniques.
4. The importance of participating in a neurologic rehabilitation program (if necessary) must be stressed.

University of Mosul

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Subject Name : Critical Care Nursing

Grade : 4th

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Pneumothorax

Lecturer : Ali Mohammed Fathi

❖ Background

- Partial or complete collapse of the lung due to positive pressure in the pleural space
- A pneumothorax occurs when free air accumulates in the pleural cavity between the visceral and parietal areas, and causes a portion or the complete lung to collapse.
- Pressure in the pleural space is normally less than that of atmospheric pressure but following a penetration injury, air can enter the cavity from the outside changing the pressure within the lung cavity and causing it to collapse.
- Air can also migrate to the area when the esophagus is perforated or a bronchus ruptures, leaking air into the mediastinum (pneumomediastinum).
- Barotrauma related to mechanical ventilatory support using high levels of **PEEP** leads to alveoli rupture and collapse.
- Gas formation from gas forming organisms can also result in pneumothorax.
- Pneumothorax may occur spontaneously in cases where a sub-pleural bleb or emphysematous bulla ruptures due to chronic obstructive pulmonary disease, tuberculosis, cancer, **or** infection and this is the most common reason in otherwise healthy individuals.
- A tension pneumothorax is a life-threatening emergency and occurs when air is permitted into the pleural cavity but not allowed to escape, resulting in increased intrathoracic pressure and complete collapse of the lung.

❖ Incidence

Spontaneous pneumothorax affects about 9,000 persons each year in the U.S. who have no history of lung disease. This type of pneumothorax is most common in men between the ages of 20 and 40, particularly in tall, thin men. Smoking has been shown to increase the risk for spontaneous pneumothorax

❖ Types of Pneumothorax

1. **Simple Pneumothorax**
   - A simple, or spontaneous, pneumothorax occurs when air enters the pleural space through a breach of either the parietal or visceral pleura.
   - Most commonly this occurs as air enters the pleural space through the rupture of a bleb or a bronchopleural fistula.
2. **Traumatic Pneumothorax**
   - A traumatic pneumothorax occurs when air escapes from a laceration in the lung itself and enters the pleural space or enters the pleural space through a wound in the chest wall.
   - **It may result from:**

A. Blunt trauma (e.g., rib fractures).

B. penetrating chest or abdominal trauma (e.g., stab wounds or gunshot wounds).or,

C. diaphragmatic tears.

D. It may occur during invasive thoracic procedures (i.e., thoracentesis, trans bronchial lung biopsy, insertion of a subclavian line) in which the pleura is inadvertently punctured, or with barotrauma from mechanical ventilation.

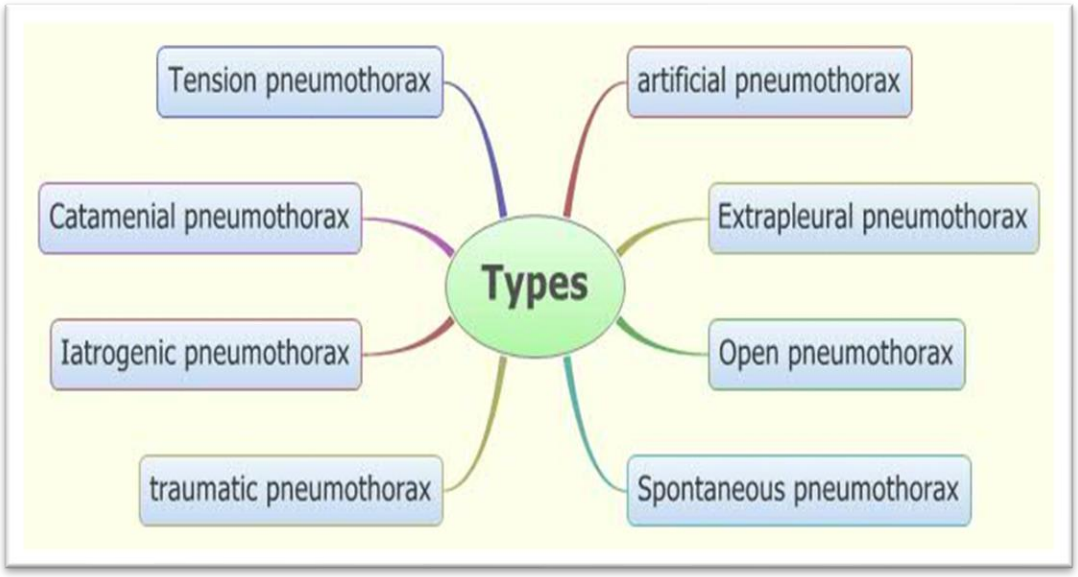
3. **Open pneumothorax**

- Is one form of traumatic pneumothorax. It occurs when a wound in the chest wall is large enough to allow air to pass freely in and out of the thoracic cavity with each attempted respiration.
- Pneumothorax in which the pleural cavity is exposed to the atmosphere through an open wound in the chest wall.

4. **Tension Pneumothorax**
    - A tension pneumothorax occurs when air is drawn into the pleural space from a lacerated lung or through a small opening or wound in the chest wall.
    - It may be a complication of other types of pneumothorax.
    - In contrast to open pneumothorax, the air that enters the chest cavity with each inspiration is trapped; it cannot be expelled during expiration through the air passages or the opening in the chest wall.
5. **Artificial pneumothorax**
    - an pneumothorax induced intentionally by artificial means.
6. **Iatrogenic pneumothorax**

This type occur as a complication of some medical procedures ,such as:

- Central venous catheter insertion
- Thoracentesis
- Transbronchial and transthoracic lung biopsy .



❖ Clinical Presentation

1. Sudden onset chest pain sharp in nature .
2. Shortness of breath
3. Tachypnea
4. Tachycardia
5. Cyanosis
6. Decrease or abscent breath sounds

❖ Treatment

Varies according to type and amount of lung collapse

A: traumatic

- Chest tube to closed water seal .
- chest drainage for lung expansion .
- surgery .
- bed rest

B: Spontaneous

If no sign of increased pleural pressure ,less than 15% lung collapse , and no dyspnea or other indication of physiological compromise.

Thoracostomy tube

- If no fluid present (Second intercostal space) .
- If fluid present(Fourth, fifth or sixth intercostal space)

❖ Diagnostic procedure

A. Laboratory: hemoglobin and hematocrit may be decreased with blood loss.

B. Chest x-ray findings

- Increased translucency
- Mediastinal shift to unaffected side in tension pneumothorax
- Depressed diaphragm
- Lung collapse
- Atelectasis

C. Arterial blood gases: vary depending on the severity of the pneumothorax; oxygen saturation usually decreases, Pa02 is usually normal or decreased, and PaCO2 is occasionally increased.

D. Chest tube: placement required to facilitate re-expansion of the collapsed lung and to permit drainage of fluid from lung.

E. Thoracentesis: needle thoracentesis is required for removing the accumulation air in the pleural cavity.

❖ Patient Care

1. Vital signs
2. Chest expansion
3. Pulse Oxemetry
4. blood gasses
5. Purpose for placing a chest tube explained to the pt.

Nursing Diagnosis

- Acute Pain related to recent injury ,coughing .and deep breathing .
- Fear Related to threat to own well-being and difficulty breathing
- Impaired Gas exchange related to ventilation perfusion imbalance.
- Risk for injury related to possible complications associated with closed chest drainage system .

Nursing Interventions

1. Reduce anxiety.
2. foster cooperation with the procedure.
3. Semi Fowlers position
4. Encourage deep coughing exercise.
5. Incentive spirometry .
6. Encourage early ambulation.
7. Administered analgesics as order.

if chest tube is accidentally dislodged , occlusive dressing *(petroleum gauze) , to prevent lung collapse

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Subject Name : Critical Care Nursing

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Cardiogenic Shock

Lecturer : Ali Mohammed Fathi

❖ **Definition :** Cardiogenic shock is a clinical condition of inadequate tissue (end-organ) perfusion due to cardiac dysfunction.

❖ **Pathophysiology:**

The heart muscle loses its contractile power

resulting in a marked reduction in SV and CO

The decreased CO in turn reduces arterial blood pressure and tissue perfusion in the vital organs (heart, brain, lung, kidneys).

Flow to the coronary arteries is reduced

decreased oxygen supply to the myocardium, which increases ischemia and further reduces the heart's ability to pump

Inadequate emptying of the ventricle

leads to increased pulmonary pressures, pulmonary congestion, and pulmonary edema, exacerbating the hypoxia, causing ischemia of vital organs, and setting a vicious cycle in motion

❖ Etiology:

1. Coronary artery disease
2. Cardiac arrhythmias
3. Systolic dysfunction
4. Diastolic dysfunction
5. Valvular dysfunction
6. Mechanical complications

❖ Clinical Manifestations

The classic signs of cardiogenic shock are tissue hypoperfusion manifested as cerebral hypoxia (restlessness, confusion, agitation), low blood pressure, rapid and weak pulse, cold and clammy skin, increased respiratory crackles, hypoactive bowel sounds, and decreased urinary output. Initially, arterial blood gas analysis may show respiratory alkalosis. Dysrhythmias are common and result from a decrease in oxygen to the myocardium

❖ Physical examination

1. Skin is usually ashen or cyanotic and cool; extremities are mottled.
2. Peripheral pulses are rapid and faint and may be irregular if arrhythmias are present.
3. Jugular venous distention and crackles in the lungs are usually (but not always) present; peripheral edema also may be present.
4. Heart sounds are usually distant, and third and fourth heart sounds may be present.
5. The pulse pressure may be low, and patients are usually tachycardiac.

6. Patients show signs of hypo perfusion, such as altered mental status and decreased urine output.
7. Ultimately, patients develop systemic hypotension (i.e., systolic blood pressure below 90 mm Hg or a decrease in mean blood pressure by 30 mm Hg).

❖ Diagnostic tests

1. Laboratory studies

- Blood urea nitrogen (BUN)
- Creatinine
- Urine specific gravity
- LDH
- CPK
- Arterial Blood Gas analysis (ABGs).
- Serum electrolyte.

2. Imaging studies

- Echocardiography should be performed early to establish the cause of cardiogenic shock.
- Chest radiographic findings are useful for excluding other causes of shock or chest pain (e.g., aortic dissection, tension pneumothorax, pneumomediastinum) .
- Coronary angiography is urgently indicated in patients with myocardial ischemia or MI who also develop cardiogenic shock.

3. **Electrocardiography.**
4. catheterization is very useful for helping exclude other causes and types of shock (e.g., volume depletion, obstructive shock) **.**

❖ Management

1. Fluid resuscitation to correct hyporvolemia and hypotension, unless pulmonary edema is present.
2. pharmacologic therapy to maintain blood pressure and cardiac output
3. Early restoration of coronary blood flow
4. Correction of electrolyte and acid-base abnormalities (e.g., hypokalemia, hypomagnesaemia, acidosis).
5. provide vascular access for multiple infusions, and allow invasive monitoring of central venous pressure .
6. An arterial line may be placed to provide continuous blood pressure monitoring.
7. An intra-aortic balloon pump may be placed as a bridge to percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) .

❖ Complications of cardiogenic shock

1. Complications of the etiologic problem.
2. Complications of shock (generally end- organ destruction).
   A. **Brain**. Ischemia, leaving neurologic deficits.
   B. **Kidney**. Acute tubular necrosis.
   C. **Lungs**. Adult respiratory distress syndrome.

complications associated with IABP include, insertion injuries, malposition of balloon, ischaemia of the leg in which the balloon is inserted, aortic embolism, aortic thrombus, aorto-iliac dissection, false femoral aneurysm, infection, thrombocytopenia, balloon rupture or leak with gas embolism and hemorrhage.

❖ Nursing Care Plan for patients with cardiogenic shock

Assessment

Appearance

- Restlessness progressing to unresponsiveness.
- Chest pain.
- Dysrhythmias.

Vital signs

- HR: >100 beats/min.
- BP: <80 mm Hg.
- RR: > 20 breaths/min.

Neurologic

- Agitation.
- Restlessness progressing to unresponsiveness, and changes in level of consciousness.

Cardiovascular

- Weak thready pulses.
- Rhythm may be irregular.

Pulmonary

- Orthopnea
- Crackles
- Cough with increased secretions.

Nursing Diagnosis:

Impaired gas exchange related to increased left ventricular diastolic pressure (LVEDP) and pulmonary edema associated with severe left ventricular (LV) dysfunction.

Nursing Interventions

1. Continuously monitor oxygenation status with pulse oximetry.

2. Monitor for desaturation in response to nursing intervention.
3. Monitor fluid volume status.
4. Obtain HR, RR, and BP every 15 minutes to evaluate the patient's response to therapy and detect cardiopulmonary deterioration.
5. Assess the patient's respiratory status. The use of accessory muscles and inability to speak suggest worsening pulmonary congestion.
6. Review ABGs for decreasing trend in Pao2 (hypoxemia) or pH (acidosis). These conditions can adversely affect myocardial contractility.
7. Provide supplemental oxygen as ordered. If the patient develops respiratory distress, be prepared for intubation and mechanical ventilation.
8. Administer low-dose morphine sulfate as ordered to reduce preload in an attempt to decrease pulmonary congestion.
9. Minimize oxygen demand by maintaining bed rest and decreasing anxiety, fever, and pain.
10. Position the patient for maximum chest excursion and comfort

University of Mosul

College of Nursing

Department of Adult Nursing

Subject Name : Critical Care Nursing

Grade : 4th

Semester : 1st & 2nd / 2024 – 2025

Acute Kidney Injury

Lecturer : Ali Mohammed Fathi

❖ **Definition :** Sudden and almost complete loss of kidney functions.

Acute kidney injury (AKI) is a relatively new term used to describe the spectrum of acute-onset kidney disorders that can range from mild impairment of kidney function through acute kidney failure that requires renal replacement therapy.

❖ Causes

1. **Pre-renal causes**
   - Hypovolaemia: haemorrhage, dehydration, GI losses, burns
   - Decreased cardiac output: arrhythmia, heart failure, MI, cardiogenic shock
   - Decreased vascular resistance: anaphylaxis, neuro injury, septic shock
   - Decreased renovascular blood flow: embolism, renal artery thrombosis
2. **Intra -renal causes**
   - Prolonged renal ischemia.
   - Hemoglobinuria (transfusion reaction, hemolytic anemia, crush injury, burns, massive tissue injury).
   - Exposure to nephrotoxic agents (Aminoglycoside antibiotics, heavy metals)
   - Acute glomerulonephritis.
   - Acute pyelonephritis.
3. **Post- renal causes**
   - Benign prostatic hyperplasia
   - Bladder cancer
   - Calculi formation (kidney stones)
   - Trauma
   - Prostate cancer

❖ Signs and Symptoms

1. Lethargy
2. Persistent nausea, vomiting , and diarrhea.
3. Dry skin and mucus membranes.
4. The breath may have the odor of urine.
5. Drowsiness, headache , muscle twitching and convulsion.
6. Scanty urinary output, with low specific gravity.
7. Daily rise in the serum creatinine value.
8. Severe hyperkalemia which may lead to dysrhythmias and cardiac arrest.
9. Progressive acidosis.
10. Decrease in serum calcium level.
11. Anemia due to uremic ,gastrointestinal lesions, reduced red cells life span and reduced erythropoietin production.

❖ Complications

Potential complications of acute kidney failure include:

1. Fluid buildup. Acute kidney failure may lead to a buildup of fluid in lungs, which can cause shortness of breath.
2. Chest pain. If the lining that covers heart (pericardium) becomes inflamed, may experience chest pain.
3. Muscle weakness. When the body's fluids and electrolytes - body's blood chemistry — are out of balance, muscle weakness can result.
4. Permanent kidney damage. Occasionally, acute kidney failure causes permanent loss of kidney function, or end-stage renal disease. People with end-stage renal disease require either permanent dialysis — a mechanical

filtration process used to remove toxins and wastes from the body — or a kidney transplant to survive.

5. Death. Acute kidney failure can lead to loss of kidney function and, ultimately, death.

❖ Management

The goals of management are;

- To restore normal chemical balance.
- To prevent complications.

The treatment modalities include the followings:

1. Dialysis , hemodialysis, peritoneal dialysis.
2. Reduce potassium level by:
   a. Monitoring serum electrolytes level.
   b. ECG.
   c. Administering ion exchange resins (**kayexalate**) orals, or by retention enema and eliminate external sources of potassium.
3. Management of fluid balance by:
   a. Daily body weight.
   b. Measurement of serum and urine concentrations.
   c. Intake and output recording.
   d. Check blood pressure.
   e. Check for any edema, or distension of jagular veins.
   f. Dietary protein are limited to approximately 1 g/1 kgBW during oliguric phase and the high protein diet after the diuretic phase.

g. High carbohydrates, restricts foods and fluids containing potassium and phosphorus such as banana, juice, and coffee.
h. Bed rest to reduce patient's metabolic rate.
i. Skin care to avoid dryness, breakdown, and itching

❖ Nursing Considerations

1. Fluid replacement therapy
2. Daily weight of patient
3. Monitor for hypervolaemia in oliguric phase and hypovolaemia in diuretic phase
4. Monitor potassium levels and ECG
5. Monitor GSC as waste product accumulation can affect mental status
6. Restrict potassium and sodium
7. Calcium supplements
8. Adequate protein intake
9. monitor closely for signs of infection as this is a common cause of death with AKI
10. Encourage respiratory exercises

General Rules to Keep Kidneys as Healthy as Possible

1. Work with doctor & nurse to manage diabetes and high blood pressure.
2. Live healthy! Eat a diet low in salt and fat, exercise for 30 minutes at least five days per week, limit alcohol and take all prescription medicines as doctor tells to it.
3. If take over-the-counter pain medicines, such as aspirin or ibuprofen, do not take more than is recommended on the package. Taking too much of these medicines can hurt kidneys and can cause AKI.

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Myocardial Infarction

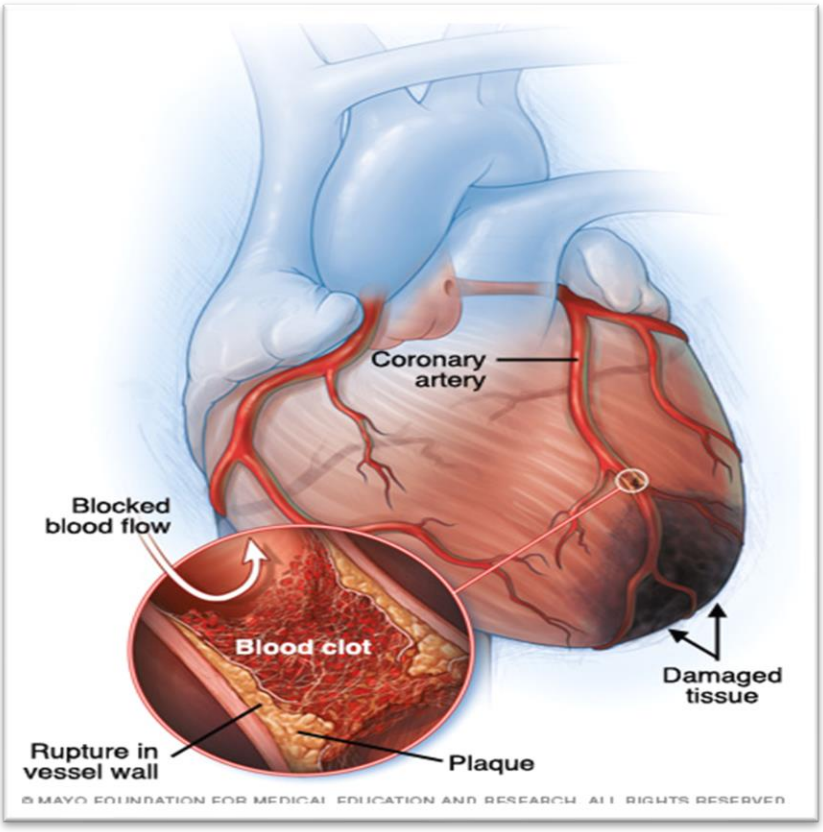
Lecturer : Ali Mohammed Fathi

Heart attack

❖ Overview

A heart attack occurs when the flow of blood to the heart is blocked, most often by a build-up of fat, cholesterol and other substances, which form a plaque in the arteries that feed the heart (coronary arteries). The interrupted blood flow can damage or destroy part of the heart muscle.

A heart attack, also called a myocardial infarction, can be fatal.



❖ Symptoms

Common heart attack signs and symptoms include:

1. Pressure, tightness, pain, or a squeezing or aching sensation in chest or arms that may spread to neck, jaw or back
2. Nausea, indigestion, heartburn or abdominal pain
3. Shortness of breath
4. Cold sweat
5. Fatigue
6. Lightheadedness or sudden dizziness

❖ Causes

A heart attack occurs when one or more of **coronary arteries become blocked**. Over time, a coronary artery can narrow from the build up of various substances, including cholesterol (atherosclerosis). This condition, known as coronary artery disease, causes most heart attacks.

During a heart attack, one of these plaques can rupture and spill cholesterol and other substances into the bloodstream. A blood clot forms at the site of the rupture. If large enough, the clot can completely block the flow of blood through the coronary artery. Another cause of a heart attack is a **spasm of a coronary artery** that shuts down blood flow to part of the heart muscle. Use of tobacco and of illicit drugs, such as cocaine. A heart attack can also occur due to a **tear in the heart artery** (spontaneous coronary artery dissection)

❖ Risk factors

Heart attack risk factors include:

1. **Age**. Men age 45 or older and women age 55 or older are more likely to have a heart attack than are younger men and women.
2. **Tobacco**. Smoking and long-term exposure to smoke increase the risk of a heart attack.
3. **High blood pressure**. Over time, high blood pressure can damage arteries that feed heart by accelerating atherosclerosis. High blood pressure that occurs with obesity, smoking, high cholesterol or diabetes increases risk even more.
4. **High blood cholesterol or triglyceride levels**. A high level of low-density lipoprotein (LDL) cholesterol (the "bad" cholesterol) is most likely to narrow arteries. A high level of triglycerides, a type of blood fat related to diet, also ups risk of heart attack. However, a high level of high-density lipoprotein (HDL) cholesterol (the "good" cholesterol) lowers risk of heart attack.
5. **Diabetes**. not producing enough insulin or not responding to insulin properly — causes body's blood sugar levels to rise. Diabetes, especially uncontrolled, increases risk of a heart attack.
6. **Family history of heart attack**.
7. **Lack of physical activity**. An inactive lifestyle contributes to high blood cholesterol levels and obesity.
8. **Obesity**. Obesity is associated with high blood cholesterol levels, high triglyceride levels, high blood pressure and diabetes.
9. **Stress**. respond to stress in ways that can increase risk of a heart attack.
10. **Illegal drug use**. Using stimulant drugs, such as cocaine, can trigger a spasm of coronary arteries that can cause a heart attack.
11. **A history of preeclampsia**. This condition causes high blood pressure during pregnancy and increases the lifetime risk of heart disease.

❖ Complications

Complications are often related to the damage done to heart during an attack. Damage can lead to:

1. **Abnormal heart rhythms (arrhythmias)**. Electrical "short circuits" can develop, resulting in abnormal heart rhythms.
2. **Heart failure**. An attack may damage so much heart tissue that the remaining heart muscle can't adequately pump blood out of heart
3. **Heart rupture**. Areas of heart muscle weakened by a heart attack can rupture, leaving a hole in part of the heart. This rupture is often fatal.
4. **Valve problems**. Heart valves damaged during a heart attack may develop severe leakage problems.

❖ Prevention

Here are ways to prevent a heart attack.

1. **Medications.** Taking medications can reduce risk of a subsequent heart attack and help damaged heart function better.
2. **Lifestyle factors**.

❖ Diagnosis

1. **Electrocardiogram (ECG)**. This first test done to diagnose a heart attack records the electrical activity of heart via electrodes attached to skin.
2. **Blood tests**. Certain heart enzymes slowly leak out into blood if the heart has been damaged by a heart attack.
3. **Chest X-ray**. An X-ray image of chest allows doctor to check the size of heart and its blood vessels and to look for fluid in lungs.
4. **Echocardiogram**. During this test, sound waves directed at heart from a wandlike device (transducer) held on chest bounce off heart and are processed electronically to provide video images of heart. An echocardiogram can help

identify whether an area of heart has been damaged by a heart attack and isn't pumping normally or at peak capacity.

5. **Coronary catheterization (angiogram)**.
6. **Exercise stress test**. In the days or weeks after heart attack, may also undergo a stress test. Stress tests measure how heart and blood vessels respond to exertion. may walk on a treadmill or pedal a stationary bike while attached to an ECG machine. Or may receive a drug intravenously that stimulates heart similar to exercise.
7. **Cardiac computerized tomography (CT)**
8. **Magnetic resonance imaging (MRI)**.

❖ Management

The main goals in treating myocardial infarction are to increase blood flow to the coronary arteries which lead to:

1. Decrease infarction size.
2. Increase oxygen supply .
3. Decrease oxygen demand to prevent myocardial death or injury.
4. Control or correct dysrhythmias .

Heart attack treatment at a hospital

With each passing minute after a heart attack, more heart tissue loses **oxygen** and deteriorates or dies. The main way to prevent heart damage is to restore blood flow quickly.

Medications

Medications given to treat a heart attack include:

1. **First Step Give Oxygen Therapy.**

2. **Aspirin**. emergency medical personnel may give aspirin immediately. Aspirin reduces blood clotting, thus helping maintain blood flow through a narrowed artery.
3. **Thrombolytics**. These drugs, also called clotbusters, help dissolve a blood clot that's blocking blood flow to heart. The earlier receive a thrombolytic drug after a heart attack, the greater the chance survive and with less heart damage.
4. **Antiplatelet agents**. Emergency room doctors may give other drugs to help prevent new clots and keep existing clots from getting larger. These include medications, such as clopidogrel (Plavix) and others, called platelet aggregation inhibitors.
5. **Other blood-thinning medications**. likely be given other medications, such as heparin, to make blood less "sticky" and less likely to form clots. Heparin is given intravenously or by an injection under skin.
6. **Pain relievers**. may receive a pain reliever, such as morphine, to ease discomfort.
7. **Nitroglycerin**. This medication, used to treat chest pain (angina), can help improve blood flow to the heart by widening (dilating) the blood vessels.
8. **Beta blockers**. These medications help relax heart muscle, slow heartbeat and decrease blood pressure, making heart's job easier. Beta blockers can limit the amount of heart muscle damage and prevent future heart attacks.
9. **ACE inhibitors**. These drugs lower blood pressure and reduce stress on the heart.

Surgical and other procedures

In addition to medications, may undergo one of the following procedures to treat heart attack:

1. **Coronary angioplasty and stenting**. Doctors insert a long, thin tube (catheter) that's passed through an artery, usually in leg or groin, to a blocked artery in heart.

If had a heart attack, this procedure is often done immediately after a cardiac catheterization, a procedure used to locate blockages.

2. **Coronary artery bypass surgery**. In some cases, doctors may perform emergency bypass surgery at the time of a heart attack. If possible, doctor may suggest that have bypass surgery after heart has had time — about three to seven days — to recover from heart attack.

Lifestyle and home remedies

lifestyle affects heart health. The following steps can help not only prevent but also recover from a heart attack:

1. **Avoid smoke**. The most important thing can do to improve heart health is to not smoke. Also, avoid being around secondhand smoke.
2. **Control blood pressure and cholesterol levels**.
3. **Get regular medical checkups**. Some of the major risk factors for heart attack — high blood cholesterol, high blood pressure and diabetes.
4. **Exercise regularly**. Regular exercise helps improve heart muscle function after a heart attack and helps prevent a heart attack by helping to control weight, diabetes, cholesterol and blood pressure.
5. **Maintain a healthy weight**.
6. **Eat a heart-healthy diet**. Saturated fat, trans fats and cholesterol in diet can narrow arteries to heart, and too much salt can raise blood pressure. Eat a heart-healthy diet that includes lean proteins, such as fish and beans, plenty of fruits and vegetables and whole grains.
7. **Manage diabetes**.
8. **Control stress**.
9. **No drink alcohol, do so in moderation**.

❖ Nursing Management

The nursing management involved in MI is critical and systematic, and efficiency is needed to implement the care for a patient with MI.

Nursing Assessment

One of the most important aspects of care of the patient with MI is the assessment.

1. Assess for chest pain not relieved by rest or medications.
2. Monitor vital signs, especially the blood pressure and pulse rate.
3. Assess for presence of shortness of breath, dyspnea, tachypnea, and crackles.
4. Assess for nausea and vomiting.
5. Assess for decreased urinary output.
6. Assess for the history of illnesses.
7. Perform a precise and complete physical assessment to detect complications and changes in the patient's status.
8. Assess IV sites frequently.

Diagnosis

Based on the clinical manifestations, history, and diagnostic assessment data, major nursing diagnoses may include.

1. Ineffective cardiac tissue perfusion related to reduced coronary blood flow.
2. Risk for ineffective peripheral tissue perfusion related to decreased cardiac output from left ventricular dysfunction.
3. Deficient knowledge related to post-MI self-care.

Planning & Goals

To establish a plan of care, the focus should be on the following:

1. Relief of pain or ischemic signs and symptoms.
2. Prevention of myocardial damage.
3. Absence of respiratory dysfunction.

4. Maintenance or attainment of adequate tissue perfusion.
5. Reduced anxiety.
6. Absence or early detection of complications.
7. Chest pain absent/controlled.
8. Heart rate/rhythm sufficient to sustain adequate cardiac output/tissue perfusion.
9. Achievement of activity level sufficient for basic self-care.
10. Anxiety reduced/managed.
11. Disease process, treatment plan, and prognosis understood.
12. Plan in place to meet needs after discharge.

Nursing Priorities

1. Relieve pain, anxiety.
2. Reduce myocardial workload.
3. Prevent/detect and assist in treatment of life-threatening dysrhythmias or complications.
4. Promote cardiac health, self-care.

Nursing Interventions

Nursing interventions should be anchored on the goals in the nursing care plan.

1. Administer oxygen along with medication therapy to assist with relief of symptoms.
2. Encourage bed rest with the back rest elevated to help decrease chest discomfort and dyspnea.
3. Encourage changing of positions frequently to help keep fluid from pooling in the bases of the lungs.
4. Check skin temperature and peripheral pulses frequently to monitor tissue perfusion.

5. Provide information in an honest and supportive manner.
6. Monitor the patient closely for changes in cardiac rate and rhythm, heart sounds, blood pressure, chest pain, respiratory status, urinary output, changes in skin color, and laboratory values.

Evaluation

After the implementation of the interventions within the time specified, the nurse should check if:

1. There is an absence of pain or ischemic signs and symptoms.
2. Myocardial damage is prevented.
3. Absence of respiratory dysfunction.
4. Adequate tissue perfusion maintained.
5. Anxiety is reduced.

❖ Discharge and Home Care Guidelines

The most effective way to increase the probability that the patient will implement a self-care regimen after discharge is to identify the patient's priorities.

1. Education. This is one of the priorities that the nurse must teach the patient about heart-healthy living.
2. Home care. The home care nurse assists the patient with scheduling and keeping up with the follow-up appointments and with adhering to the prescribed cardiac rehabilitation management.
3. Follow-up monitoring. The patient may need reminders about follow-up monitoring including periodic laboratory testing and ECGs, as well as general health screening.
4. Adherence. The nurse should also monitor the patient's adherence to dietary restrictions and prescribed medications.

❖ Documentation Guidelines

To ensure that every action documented is an action done, documentation must be secured. The following should be documented:

1. Individual findings.
2. Vital signs, cardiac rhythm, presence of dysrhythmias.
3. Plan of care and those involved in planning.
4. Teaching plan.
5. Response to interventions, teaching, and actions performed.
6. Attainment or progress towards desired outcomes.
7. Modifications to plan of care.

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Angina Pectoris

Lecturer : Ali Mohammed Fathi

❖ **Fast facts on angina**

- Angina is not a disease in itself, but a symptom of heart disease .
- Attacks are caused by reduced oxygen in the blood reaching the heart.
- Symptoms include tightness and difficulty breathing.
- Treatment options can range from lifestyle changes to medications.

❖ **Definition:** Angina is not a disease in its own right but a probable symptom of coronary artery disease. It is a tightness, pain, or discomfort in the chest that occurs when an area of the heart muscle receives less blood oxygen than usual. It is not a life-threatening condition when experienced on its own. However, if a person is experiencing angina, it is a strong indicator that they have a type of heart disease.

❖ Types

There are several types of angina.

1. **Stable or chronic angina**

   Stable angina occurs when the heart is working harder than usual, for instance, during exercise. It has a regular pattern and can be predicted to happen over months or even years. Rest or medication relieves symptoms.

2. **Unstable angina**

   Unstable angina does not follow a regular pattern. It can occur when at rest and is considered less common and more serious because rest and medication do not relieve it. This version can signal a future heart attack within a short time - hours or weeks.

3. **Variant and microvascular angina**

   Variant or Prinzmetal's angina This type of angina, or chest pain, may be a symptom of coronary microvascular disease (MVD). Coronary MVD is heart disease that affects the heart's smallest coronary artery blood vessels.

❖ Symptoms

Angina is usually felt in the chest region as:

1. squeezing
2. pressure
3. heaviness

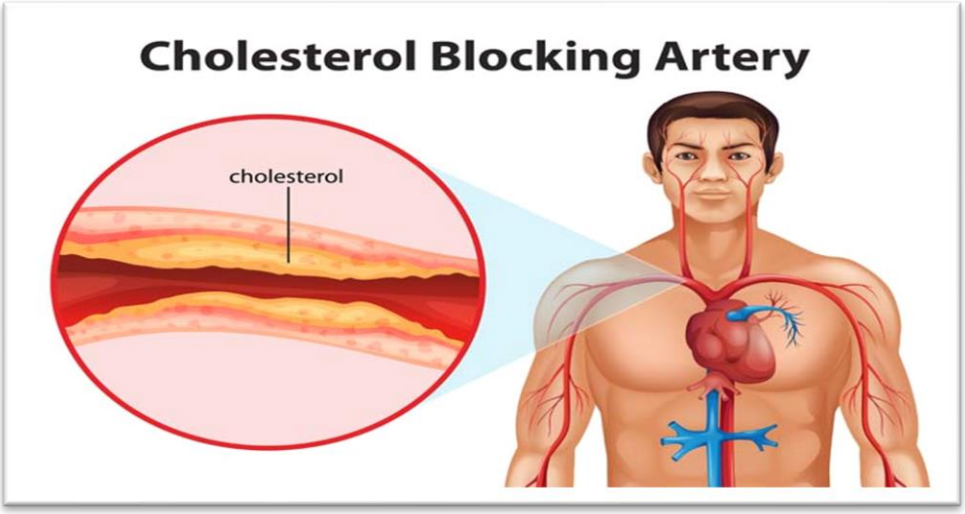
4. tightening
5. burning or aching across the chest, usually starting behind the breastbone This pain often spreads to the neck, jaw, arms, shoulders, throat, back, or even the teeth.

Patients may also complain of symptoms including:

1. Indigestion
2. Heartburn
3. Weakness
4. Sweating
5. Nausea
6. Cramping
7. Shortness of breath

Stable angina usually lasts a short period and may feel like gas or indigestion. Unstable angina occurs at rest, is surprising, lasts longer, and may worsen over time. Variant angina occurs at rest and is usually severe.

❖ Causes



Atherosclerosis - a buildup of plaque around the artery wall - is the most common cause of angina.

Angina is most frequently the result of underlying coronary artery disease. The coronary arteries supply the heart with oxygen-rich blood. When cholesterol aggregates on the artery wall and hard plaques form, the artery narrows.

This means:

1. It is increasingly difficult for oxygen-rich blood to reach the heart muscle as the arteries become too narrow.
2. Damage to the arteries from other factors, such as smoking and high levels of fat or sugar in the blood, can cause plaques to build up where the arteries are damaged.
3. These plaques narrow the arteries or may break off and form blood clots that block the arteries.

The actual angina attacks are the result of this reduced oxygen supply to the heart. Common triggers include:

1. physical exertion
2. severe emotional stress
3. a heavy meal
4. exposure to extreme temperatures
5. smoking may trigger angina attacks

Unstable angina is often caused by blood clots that partially or completely block an artery. Larger blockages may lead to heart attacks. As blood clots form, dissolve, and form again, angina can occur with each blockage.

Variant angina occurs when an artery experiences a spasm that causes it to tighten and narrow, disrupting the blood supply to the heart. This can be triggered by exposure to cold, stress, medicines, smoking, or cocaine use.

❖ Diagnosis

A correct diagnosis is important because it can predict the likelihood of having a heart attack. The process will start with a physical exam as well as a discussion of symptoms, risk factors, and family medical history.

A physician who is suspicious of angina will order one or more of the following tests:

1. **Electrocardiogram (EKG):** Records electrical activity of the heart and can detect when the heart is starved of oxygen.
2. **Stress test:** Blood pressure readings and an EKG while the patient is increasing physical activity.
3. **Chest X-ray:** This enables the doctor to see structures inside the chest.
4. **Coronary angiography:** Dye and special X-rays to show the inside of coronary arteries.
5. **Blood tests:** These check fat, cholesterol, sugar, and protein levels.

❖ Risk factors

Those at an increased risk of coronary artery disease are also at an increased risk of angina. Risk factors include:

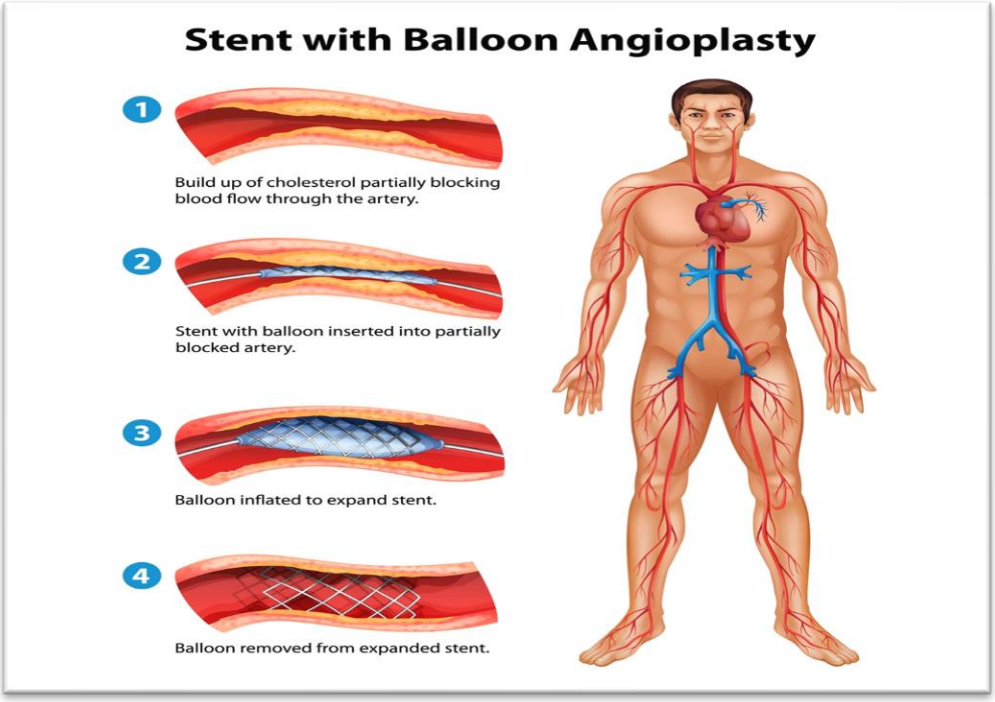
1. unhealthy cholesterol levels
2. high blood pressure
3. tobacco smoking
4. diabetes

5. being overweight or obese
6. metabolic syndrome
7. sedentary lifestyle
8. being over 45 years of age for men and over 55 years of age for women
9. a family history of early heart disease

Angina can be prevented by changing lifestyle factors and by treating related conditions that exacerbate or contribute to angina symptoms. To prevent or delay angina, eat healthfully, quit smoking, be physically active, and learn how to handle stress.

Also, make sure to receive proper treatment for high blood cholesterol, high blood pressure, diabetes, and obesity.

❖ Treatment



Angina treatments aim to reduce pain, prevent symptoms, and prevent or lower the risk of heart attack. Medicines, lifestyle changes, and medical procedures may all be employed.

Lifestyle changes recommended to treat angina include:

1. Stopping smoking
2. Controlling weight
3. Regularly checking cholesterol levels
4. Resting and slowing down
5. Avoiding large meals
6. Learning how to handle or avoid stress
7. Eating fruits, vegetables, whole grains, low-fat or no-fat dairy products, and lean meat and fish
8. Nitrates, such as nitroglycerin, are most often prescribed for angina. Nitrates prevent or reduce the intensity of angina attacks by relaxing and widening blood vessels.

Other medicines may be used such as:

1. Beta blockers
2. Calcium channel blockers
3. Angiotensin – covering enzyme (ACE) inhibitors
4. Oral anti – platelet medicines
5. anticoagulants

High blood pressure medications may also be prescribed to treat angina. These medicines are designed to lower blood pressure and cholesterol levels, slow the heart rate, relax blood vessels, reduce strain on the heart, and prevent blood clots from forming.

In some cases, surgical procedures are necessary to treat angina. A heart specialist may recommend angioplasty. Coronary artery bypass grafting is another standard procedure. This is surgery where the narrowed arteries in the heart are bypassed using a healthy artery or vein from another part of the body.

References

American Association of Critical-Care Nurses. AACN Essentials of Critical Care Nursing. 5th ed. St. Louis, MO: Elsevier; 2023.

2. Brunner LS, Suddarth DS. Bruner & Suddarth's Textbook of Medical-Surgical Nursing. 15th ed. Philadelphia, PA: Wolters Kluwer; 2023.

3. American Association of Critical-Care Nurses. AACN Essentials of Critical Care Nursing. 4th ed. St. Louis, MO: Elsevier; 2020

4. Titler MG, Kleiber C, Steelman VM, et al. Critical Care Nursing: A Holistic Approach. 9th ed. St. Louis, MO: Elsevier; 2020**.**

5. Drain CB. Inhalation anesthesia. In: Odom-Forren J, ed. *Drain's Perianesthesia Nursing: A Critical Care Approach*. 6th ed. St. Louis: Elsevier; 2013.