MEDICINE QUESTIONS AND ANSWERS

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Medicine I

Questions and Answers

Question 1

NGORA SCHOOL NURSING AND MIDWIFERY - NO.39

- Define the term leukemia.

- List the types of leukemia.

- Describe the management of a patient admitted with leukemia on your ward until discharge.

- Mention 5 complications of acute leukemia.

ANSWER: Examination Script

Leukemia, literally meaning "white blood," is a type of cancer that affects the blood and bone marrow. It is characterized by the abnormal and uncontrolled proliferation (rapid increase in numbers) of one particular type of white blood cell (leukocyte) or its precursors (immature forms). These abnormal cells (leukemic cells) crowd out normal blood cells (red blood cells, normal white blood cells, and platelets) in the bone marrow, leading to various complications. The defect originates in the hematopoietic stem cell, the myeloid stem cell, or the lymphoid stem cell.

1. Acute Myeloid Leukemia (AML): Affects myeloid cells (which form granulocytes, monocytes, red blood cells, platelets). Characterized by rapid proliferation of immature myeloid cells (blasts). Progresses quickly.

2. Chronic Myeloid Leukemia (CML): Affects myeloid cells but progresses more slowly than AML. Involves an overproduction of relatively mature but still abnormal myeloid cells. Often associated with a specific genetic mutation (Philadelphia chromosome).

3. Acute Lymphocytic Leukemia (ALL) / Acute Lymphoblastic Leukemia: Affects lymphoid cells (which form lymphocytes - B cells, T cells, NK cells). Characterized by rapid proliferation of immature lymphocytes (lymphoblasts). Most common type of childhood leukemia, but can also occur in adults.

4. Chronic Lymphocytic Leukemia (CLL): Affects lymphoid cells but progresses slowly. Involves an overproduction of mature-appearing but abnormal lymphocytes (usually B cells). Most common type of leukemia in older adults in Western countries. These cells often escape apoptosis (programmed cell death).

(Other less common types exist, e.g., Hairy Cell Leukemia, T-cell prolymphocytic leukemia).

Risk for Infection and Bleeding: Due to neutropenia (low neutrophils) and thrombocytopenia (low platelets) caused by leukemia itself or chemotherapy.

Risk for Impaired Skin Integrity: Due to chemotherapy side effects, malnutrition, immobility.

Impaired Gas Exchange: Due to anemia, lung infections, or leukemic infiltrates.

Impaired Mucous Membranes: Mucositis (mouth sores) from chemotherapy.

Imbalanced Nutrition: Less Than Body Requirements: Due to anorexia, nausea, vomiting, mucositis, hypermetabolic state.

Acute Pain and Discomfort: From mucositis, bone pain (due to marrow expansion), infections.

Hyperthermia: Due to infection or tumor lysis syndrome.

Fatigue and Activity Intolerance: Due to anemia, infection, disease process.

Self-Care Deficit.

Anxiety and Fear: Due to diagnosis, treatment, uncertain future.

Disturbed Body Image: Due to hair loss, weight changes, central lines.

1. Diagnosis Confirmation and Staging: Bone marrow aspiration and biopsy, blood tests (FBC, peripheral smear), cytogenetics, molecular studies.

2. Chemotherapy Administration (Primary Treatment): Specific regimens depend on leukemia type. Induction Phase: To achieve remission (kill most leukemic cells). Consolidation/Intensification Phase: To eliminate remaining leukemic cells. Maintenance Phase (for some types, e.g., ALL): Lower dose chemotherapy over a longer period to prevent relapse. Nursing: Safe handling and administration of cytotoxic drugs, managing side effects (nausea, vomiting, mucositis, hair loss, myelosuppression).

3. Infection Prevention and Management: Protective Isolation (Neutropenic Precautions): If severely neutropenic (e.g., single room, strict hand hygiene, no fresh flowers/fruits, mask for visitors/staff). Monitor for signs of infection (fever is a key sign, even without other symptoms). Prompt investigation (cultures) and empirical broad-spectrum antibiotics if febrile neutropenia. Oral hygiene to prevent mouth infections. Skin care. Prophylactic antimicrobials (antifungal, antiviral) may be used.

4. Bleeding Prevention and Management: Monitor for signs of bleeding (petechiae, bruising, epistaxis, gum bleeding, hematuria, melena). Avoid IM injections, rectal temperatures/medications if platelets are very low. Use soft toothbrush. Platelet transfusions if severe thrombocytopenia or active bleeding.

5. Blood Product Support: Packed red blood cell transfusions for anemia. Platelet transfusions for thrombocytopenia.

6. Management of Chemotherapy Side Effects: Nausea/Vomiting: Administer anti-emetics regularly. Mucositis/Stomatitis: Oral care with bland rinses, analgesics, soft diet. Diarrhea/Constipation: Monitor bowel function, manage appropriately. Fatigue: Plan rest periods, assist with ADLs.

7. Nutritional Support: High-calorie, high-protein diet if tolerated. Small, frequent meals. Nutritional supplements. Enteral or parenteral nutrition if severe malnutrition or inability to eat.

8. Pain Management: Assess and manage pain effectively (e.g., from bone pain, mucositis).

9. Psychological and Emotional Support: Allow patient and family to express feelings. Provide information and education about the disease and treatment. Offer counseling or refer to support groups/social worker.

10. Monitoring for Complications: E.g., Tumor Lysis Syndrome (metabolic emergency with rapid cell breakdown), leukostasis (very high WBC count causing sludging in blood vessels), organ infiltration by leukemic cells.

11. Discharge Planning: Education on home care, managing side effects, signs of infection/bleeding to report. Medication schedule and administration. Importance of follow-up appointments, blood tests. Central line care (if applicable). Support services in the community. Criteria for discharge: Medically stable, counts recovering (if expected), able to manage at home with support, follow-up arranged.

1. Severe Infections (Sepsis): Due to neutropenia (low number of functional neutrophils) caused by the leukemia itself or its treatment (chemotherapy). This makes patients highly susceptible to bacterial, fungal, and viral infections, which can rapidly become life-threatening.

2. Bleeding (Hemorrhage): Due to thrombocytopenia (low platelet count) caused by bone marrow infiltration by leukemic cells or chemotherapy. Can manifest as bruising, petechiae, nosebleeds, gum bleeding, or more serious internal bleeding (e.g., gastrointestinal, intracranial).

3. Anemia: Due to decreased production of red blood cells as leukemic cells crowd out normal marrow. Leads to fatigue, weakness, pallor, shortness of breath.

4. Tumor Lysis Syndrome (TLS): A metabolic emergency that can occur with rapid breakdown of large numbers of cancer cells (spontaneously or after starting chemotherapy). Release of intracellular contents (potassium, phosphate, uric acid) into the bloodstream can cause hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, and acute kidney injury.

5. Leukostasis: Occurs with very high white blood cell counts (especially blast cells in AML). The large number of leukemic cells can increase blood viscosity and "sludge" in small blood vessels, impairing blood flow to organs like the lungs (respiratory distress) and brain (neurological symptoms like headache, confusion, stroke). A medical emergency.

6. Organ Infiltration: Leukemic cells can infiltrate various organs, causing enlargement and dysfunction (e.g., hepatomegaly, splenomegaly, lymphadenopathy, skin infiltration (leukemia cutis), central nervous system (CNS) leukemia, testicular involvement).

7. Complications of Chemotherapy: Besides myelosuppression (infection, bleeding, anemia), includes mucositis, nausea/vomiting, hair loss, cardiotoxicity, neurotoxicity, infertility, risk of secondary cancers.

Question 2

IGANGA SCHOOL OF NURSING AND MIDWIFERY - NO.40

- List the cardinal signs of pulmonary TB.

- Outline the nursing intervention of a patient with pulmonary TB.

- List the complications of pulmonary TB.

ANSWER: Examination Script

1. Persistent Cough (usually >2-3 weeks): This is the most common symptom. The cough may be dry initially but can later become productive (producing sputum).

2. Sputum Production (sometimes with blood - Hemoptysis): Coughing up phlegm. The sputum may be mucoid, mucopurulent, or blood-streaked. Frank hemoptysis (coughing up significant amounts of blood) can occur in advanced disease.

3. Fever: Often low-grade, typically occurring in the late afternoon or evening. May be accompanied by chills.

4. Night Sweats: Profuse sweating during sleep, often drenching bedclothes, even when the room temperature is cool.

5. Unintentional Weight Loss / Loss of Appetite (Anorexia): Significant, unexplained weight loss and poor appetite are common systemic symptoms.

6. Chest Pain: May be pleuritic (sharp pain worse with breathing or coughing) if the pleura (lining of the lungs) is involved, or a dull ache.

7. Fatigue / Malaise / Weakness: A general feeling of tiredness, lack of energy, and being unwell.

8. Shortness of Breath (Dyspnea) - in more advanced disease: Difficulty breathing, especially with exertion, if lung damage is extensive.

1. Ineffective Airway Clearance (related to mucus impaction, inflammation): Monitor Vital Signs: Especially respiratory rate, rhythm, pattern, and oxygen saturation. Positioning: Encourage Fowler's or semi-Fowler's position (sitting up) to facilitate lung expansion and ease breathing. Encourage Deep Breathing and Coughing Exercises: To help mobilize and expectorate secretions. Teach effective coughing techniques. Increase Fluid Intake: Encourage plenty of oral fluids (unless contraindicated) to help liquefy secretions, making them easier to cough up. Steam Inhalation / Humidification: May help soothe airways and loosen mucus. Nebulizer therapy if prescribed. Chest Physiotherapy (if indicated and not contraindicated): Techniques like postural drainage and percussion to help mobilize secretions from lower airways. Oxygen Therapy: Administer as prescribed if hypoxic. Suctioning: If patient is unable to clear secretions effectively.

2. Risk for Infection Transmission / Adherence to Treatment: Administer Anti-TB Medications: Ensure correct drugs, dosages, and timing as prescribed. Understand the combination therapy. Directly Observed Therapy (DOTS): If part of the treatment plan, ensure patient takes medication under direct observation to promote adherence. Respiratory Hygiene Education: Teach patient to cover mouth and nose when coughing or sneezing (e.g., with a tissue or elbow), proper disposal of used tissues, and handwashing. Isolation Precautions (if infectious and hospitalized): Implement airborne precautions (e.g., negative pressure room if available, N95 respirator for staff). Educate patient and family on these measures. Educate Patient and Family: About the disease, mode of transmission, importance of completing the full course of treatment (typically 6 months or longer) to prevent relapse and drug resistance.

3. Imbalanced Nutrition: Less Than Body Requirements (related to anorexia, fatigue, increased metabolism): Assess Nutritional Status: Monitor weight regularly. Provide a Balanced, High-Calorie, High-Protein Diet: Small, frequent, appealing meals. Address food preferences. Manage Nausea/Vomiting: Administer anti-emetics if needed. Oral Hygiene: To improve taste and appetite. Nutritional Supplements: If intake is poor. Administer prescribed multivitamins.

4. Management of Symptoms (Pain, Fever): Pain: Assess and manage chest pain with analgesics as prescribed. Provide comfort measures. Fever: Administer antipyretics as prescribed. Tepid sponging. Encourage fluids.

5. Activity Intolerance / Fatigue: Encourage a balance of rest and activity. Plan care to conserve energy. Assist with activities of daily living (ADLs) as needed. Gradually increase activity as tolerated.

6. Psychosocial Support: Address anxiety, fear, and potential stigma associated with TB. Provide emotional support. Encourage verbalization of feelings. Involve family in care and support.

7. Monitoring for Complications and Drug Side Effects: Observe for signs of TB complications (see below) and common side effects of anti-TB drugs (e.g., liver toxicity, peripheral neuropathy, visual changes, orange discoloration of body fluids). Report to doctor.

8. Discharge Planning and Follow-up: Ensure patient understands home care, medication regimen, importance of adherence, follow-up appointments (for sputum checks, clinical review), and when to seek help. Contact tracing of close contacts if needed.

1. Hemoptysis (Coughing up Blood): Can range from blood-streaked sputum to massive, life-threatening bleeding due to erosion of blood vessels in lung cavities.

2. Pneumothorax: Air leakage into the space between the lung and chest wall, causing lung collapse. Can occur if a TB cavity ruptures into the pleural space.

3. Pleural Effusion: Accumulation of fluid in the pleural space, often due to inflammation of the pleura.

4. Empyema: Collection of pus in the pleural space.

5. Respiratory Failure: Extensive lung damage can lead to inability of the lungs to adequately oxygenate blood or remove carbon dioxide.

6. Bronchiectasis: Permanent abnormal widening of the bronchi (airways) due to chronic inflammation and infection, leading to chronic cough and sputum production.

7. Pulmonary Fibrosis / Scarring: Healing of TB lesions can lead to scar tissue formation in the lungs, reducing lung elasticity and function.

8. Aspergilloma: A fungal ball (usually Aspergillus fumigatus) that can grow in pre-existing lung cavities caused by TB. Can cause hemoptysis.

9. Spread of TB to Other Organs (Extrapulmonary TB): M. tuberculosis can spread from the lungs via bloodstream or lymphatics to other parts of the body, e.g.: TB Meningitis (brain/spinal cord membranes). TB of Bones and Joints (e.g., Pott's disease of the spine). Renal TB (kidneys). Miliary TB (widespread disseminated TB). TB Pericarditis (sac around the heart). Gastrointestinal TB.

10. Drug Resistance (MDR-TB, XDR-TB): If treatment is inadequate, incomplete, or incorrect, the TB bacteria can become resistant to standard anti-TB drugs, making treatment much more difficult, longer, and less successful.

11. Social Stigma and Isolation.

12. Malnutrition and Wasting.

Question 3

DOKOLO SCHOOL OF NURSING AND MIDWIFERY - NO.41

- Define rheumatic fever.

- List 6 causes of rheumatic fever.

- Describe the management of a patient admitted with rheumatic fever until discharge.

ANSWER: Examination Script

Acute Rheumatic Fever (ARF) is an autoimmune inflammatory disease that can develop as a delayed complication (sequela) of an inadequately treated Group A Streptococcus (GAS) throat infection (strep throat or streptococcal pharyngitis) or, less commonly, a GAS skin infection (impetigo). The body's immune system, in trying to fight the streptococcal infection, mistakenly attacks its own tissues, particularly affecting the heart, joints, brain, and skin. It primarily affects children and young adults (ages 5-15 years) in areas with poor socioeconomic conditions and limited access to healthcare.

1. Group A Streptococcus (GAS) Infection: A preceding infection with specific strains of Streptococcus pyogenes (Group A Strep), most commonly pharyngitis (strep throat). This is the essential trigger.

2. Inadequate or Delayed Treatment of Strep Throat: Failure to treat the initial GAS infection promptly and completely with appropriate antibiotics (like penicillin) significantly increases the risk of ARF developing 2-4 weeks later.

3. Host Susceptibility / Genetic Predisposition: Not everyone with a strep infection develops rheumatic fever. Certain individuals may have a genetic susceptibility that makes their immune system more likely to cross-react with their own tissues after a GAS infection.

4. Age: Most common in children and adolescents aged 5-15 years. It is rare before age 3 and after age 30.

5. Environmental and Socioeconomic Factors: Overcrowding: Living in crowded conditions (e.g., poor housing, schools, military barracks) facilitates the spread of GAS infections. Poor Sanitation and Hygiene: Can contribute to spread of infections. Limited Access to Healthcare: Leads to untreated or inadequately treated strep infections. Malnutrition: May impair immune response.

6. Recurrence: Individuals who have had a previous episode of rheumatic fever are at very high risk of developing recurrent attacks with subsequent GAS infections, and these recurrences often cause more severe heart damage.

7. Virulence of the Streptococcal Strain: Certain rheumatogenic strains of GAS are more likely to trigger the autoimmune response.

History: Recent sore throat or skin infection, symptoms like joint pain, fever, rash, jerky movements. Previous ARF.

Physical Examination: Check for major and minor Jones criteria Major: Carditis (inflammation of heart - new murmur, pericardial rub, heart failure signs), Polyarthritis (migratory, painful inflammation of large joints), Chorea (Sydenham's chorea - involuntary jerky movements), Erythema Marginatum (pink, ring-like rash), Subcutaneous Nodules (small, painless lumps under skin). Minor: Fever, Arthralgia (joint pain without swelling), Elevated ESR/CRP, Prolonged PR interval on ECG.

Investigations: Evidence of Recent GAS Infection: Throat swab culture for GAS, Rapid strep test, Antistreptolysin O (ASO) titre or other streptococcal antibody tests (Anti-DNase B). Inflammatory Markers: Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP) usually elevated. Electrocardiogram (ECG): To look for prolonged PR interval, arrhythmias, or signs of pericarditis. Echocardiogram: Crucial to assess for carditis, valvular damage (regurgitation or stenosis), pericardial effusion, and cardiac function. Chest X-ray: If carditis or heart failure suspected (cardiomegaly, pulmonary congestion).

1. Eradication of Group A Streptococcus: Administer a course of antibiotics to eliminate any residual GAS infection, even if throat culture is negative (as infection may have cleared but triggered immune response). Preferred: Benzathine Penicillin G intramuscularly (single dose). Alternative: Oral Penicillin V for 10 days, or Erythromycin/Azithromycin for penicillin-allergic patients.

2. Anti-inflammatory Treatment: To suppress inflammation and relieve symptoms. For Arthritis and Fever: Aspirin (salicylates) or NSAIDs (e.g., naproxen) are effective. Use with caution in children regarding Reye's syndrome if viral illness also present (aspirin). For Carditis (especially moderate to severe): Corticosteroids (e.g., Prednisolone orally) are used to reduce inflammation and potentially limit heart damage. Dose is tapered gradually.

3. Bed Rest and Activity Restriction: Strict bed rest during the acute phase, especially if carditis is present, to reduce workload on the heart. Activity is gradually increased as inflammation subsides (guided by clinical improvement and inflammatory markers).

4. Management of Specific Manifestations: Carditis / Heart Failure: If heart failure develops, manage with diuretics, ACE inhibitors, digoxin as indicated. Strict monitoring. Chorea: May require sedatives (e.g., diazepam) or other medications (e.g., haloperidol, sodium valproate) if severe and distressing. Provide a quiet, supportive environment. Arthritis: Analgesics, anti-inflammatories, comfortable positioning, joint support.

5. Supportive Nursing Care: Monitor vital signs, signs of carditis (new murmurs, tachycardia, gallop rhythm, signs of heart failure), joint inflammation, chorea, rash. Ensure comfort, good nutrition, and hydration. Provide psychological support to child and family; explain the illness and treatment. Educate on importance of medication adherence.

6. Secondary Prophylaxis (Prevention of Recurrences): This is CRITICAL. Start before discharge. Regular, long-term antibiotic prophylaxis is given to prevent further GAS infections and recurrent attacks of ARF, which can cause cumulative heart damage (Rheumatic Heart Disease - RHD). Preferred: Benzathine Penicillin G IM every 3-4 weeks. Alternative: Oral Penicillin V twice daily, or Erythromycin/Sulfadiazine for penicillin-allergic. Duration of prophylaxis depends on severity of carditis and age, can be for many years, sometimes lifelong.

Education: Thorough education of patient and family about ARF, the risk of recurrence and RHD, the absolute necessity of long-term antibiotic prophylaxis (adherence is key), and signs of recurrence or worsening carditis.

Medications: Ensure understanding of any ongoing medications and the schedule for prophylactic antibiotics.

Activity Limitations: Advise on gradual return to normal activity, any restrictions if carditis was severe.

Dental Hygiene: Good oral hygiene is important as dental procedures can be a source of bacteremia. Antibiotic prophylaxis may be needed before some dental procedures if significant RHD.

Follow-up: Schedule regular follow-up appointments with a pediatrician and cardiologist (if carditis present) for monitoring, echocardiograms, and continued prophylactic antibiotic administration.

Question 4

KIBULI MUSLIM HOSPITAL TRAINING SCHOOL - NO.42

- Outline 5 (five) cardinal signs of diabetes mellitus.

- Prepare a diabetic plate.

- Explain the risk factors of diabetes mellitus.

ANSWER: Examination Script

1. Polyuria (Frequent Urination): When blood glucose levels are high (e.g., >180 mg/dL or 10 mmol/L), the kidneys cannot reabsorb all the glucose, so excess glucose spills into the urine. Glucose in the urine draws extra water with it (osmotic diuresis), leading to increased urine volume and frequency.

2. Polydipsia (Excessive Thirst): The excessive urination (polyuria) leads to dehydration and increased loss of body fluids, triggering an intense thirst mechanism as the body tries to replace the lost water.

3. Polyphagia (Excessive Hunger / Increased Appetite): Even though blood glucose levels are high, insulin deficiency or resistance prevents glucose from entering cells effectively to be used for energy. Cells "starve," sending signals that stimulate hunger. This is more common in Type 1 Diabetes.

4. Unexplained Weight Loss: Despite increased appetite (polyphagia), weight loss can occur, especially in Type 1 Diabetes. The body cannot use glucose for energy, so it starts breaking down muscle tissue and fat stores for fuel. Calories are also lost through glucose in the urine.

5. Fatigue / Weakness / Lethargy: Cells are deprived of glucose (their main energy source) due to insulin problems, leading to a lack of energy, persistent tiredness, and weakness.

(Additional common signs) 6. Blurred Vision: High blood glucose levels can cause fluid to be pulled from the lenses of the eyes, affecting their ability to focus. This is usually temporary and improves when blood sugar is controlled.

(Additional common signs) 7. Slow-Healing Sores or Frequent Infections: High blood sugar can impair immune function and circulation, making it harder for the body to fight infections and heal wounds. Recurrent skin infections, UTIs, or yeast infections can occur.

(Additional common signs) 8. Presence of Ketones in Urine (Ketonuria - more common in Type 1 DM): When the body breaks down fat for energy due to lack of insulin, ketones are produced as byproducts. High levels can lead to diabetic ketoacidosis (DKA).

1. Half ( ) of the Plate: Non-Starchy Vegetables Fill half your plate with low-carbohydrate, high-fiber vegetables. Examples: Leafy greens (spinach, sukuma wiki, cabbage), broccoli, cauliflower, carrots, green beans, tomatoes, cucumbers, peppers, mushrooms, onions. Rationale: These are low in calories and carbohydrates, high in vitamins, minerals, and fiber, which helps with satiety and blood sugar control.

2. One Quarter ( ) of the Plate: Lean Protein Foods Fill one quarter of your plate with lean sources of protein. Examples: Skinless chicken or turkey, fish (especially oily fish like salmon, mackerel), beans, lentils, chickpeas, tofu, lean beef or goat, eggs. Rationale: Protein helps with satiety and has less impact on blood sugar than carbohydrates. Choose lean options to manage fat intake.

3. One Quarter ( ) of the Plate: Whole Grains or Starchy Foods (Carbohydrates) Fill the remaining quarter with carbohydrate foods, prioritizing whole grains and complex carbohydrates. Examples: Brown rice, whole-wheat ugali/posho, whole-grain bread or chapati, quinoa, oats, sweet potatoes, yams, cassava, green bananas (matoke), peas, corn. Rationale: Carbohydrates raise blood sugar, so portion control is key. Whole grains have more fiber, which slows down glucose absorption.

4. Add a Serving of Fruit and/or Dairy (on the side or as part of the meal): Fruit: A small piece of whole fruit (e.g., apple, orange, mango slice) or about cup of berries or cubed fruit. Dairy: A glass of low-fat milk or a small pot of plain low-fat yogurt. Rationale: These provide important nutrients but also contain carbohydrates (natural sugars), so portions should be controlled.

5. Choose Healthy Fats in Small Amounts: Use healthy fats for cooking or as additions, e.g., avocado, nuts, seeds, olive oil, canola oil. Limit saturated and trans fats.

6. Drink Water or Low-Calorie Drinks: Choose water, unsweetened tea, or coffee instead of sugary drinks, juices, or sodas.

Risk Factors for Type 2 Diabetes (Most Common Type): Overweight or Obesity: Excess body fat, especially abdominal fat, increases insulin resistance (cells don't respond well to insulin). This is a major risk factor. Physical Inactivity / Sedentary Lifestyle: Lack of regular exercise contributes to weight gain and insulin resistance. Family History of Diabetes: Having a parent or sibling with diabetes increases risk, suggesting a genetic predisposition. Age: Risk increases with age, particularly after 45 years, possibly due to decreased muscle mass, increased body fat, and reduced physical activity. However, Type 2 diabetes is increasingly seen in children and adolescents. Race or Ethnicity: Certain ethnic groups have a higher prevalence (e.g., people of African, Hispanic, Asian, Native American, Pacific Islander descent). History of Gestational Diabetes: Women who had diabetes during pregnancy are at higher risk of developing Type 2 diabetes later in life. Also, giving birth to a baby weighing over 9 pounds (4 kg). Pre-diabetes: Having blood glucose levels higher than normal but not yet high enough to be diagnosed as diabetes (impaired glucose tolerance or impaired fasting glucose). High Blood Pressure (Hypertension): Often coexists with insulin resistance and increases diabetes risk. BP >140/90 mmHg is a risk. Abnormal Cholesterol and Triglyceride Levels: Low HDL ("good") cholesterol and high triglycerides are associated with increased risk. Polycystic Ovary Syndrome (PCOS): Women with PCOS often have insulin resistance and are at higher risk. History of Cardiovascular Disease. Diet: Diets high in processed foods, sugary drinks, red and processed meats, and unhealthy fats, and low in fiber, fruits, and vegetables, contribute to risk.

Risk Factors for Type 1 Diabetes (Autoimmune Disease): Family History: Having a parent or sibling with Type 1 diabetes slightly increases risk. Genetics: Certain genes (HLA types) are associated with increased susceptibility. Autoimmune Factors: The body's immune system mistakenly attacks and destroys insulin-producing beta cells in the pancreas. Environmental Triggers (less clear): Viral infections or other environmental factors may trigger the autoimmune process in genetically susceptible individuals. Geography: Incidence tends to increase as you travel away from the equator.

Risk Factors for Gestational Diabetes (Diabetes During Pregnancy): Overweight or Obesity before pregnancy. Previous history of gestational diabetes. Family history of Type 2 diabetes. Having previously delivered a large baby (>4 kg or 9 lbs). Older maternal age (>25-30 years). Certain ethnicities. Polycystic Ovary Syndrome (PCOS).

Question 5

MASAKA SCHOOL OF COMPREHENSIVE NURSING - NO.43

- Define the term glomerulonephritis.

- Explain 5 (five) risk factors for glomerulonephritis.

- Describe the nursing concern of a patient with glomerulonephritis.

ANSWER: Examination Script

Glomerulonephritis is a type of kidney disease characterized by inflammation of the glomeruli, which are tiny clusters of capillaries (small blood vessels) in the kidneys responsible for filtering waste products and excess fluid from the blood to produce urine. When the glomeruli are inflamed, their ability to filter properly is impaired, leading to leakage of red blood cells and protein into the urine, and potentially a buildup of waste products in the blood and reduced urine output. It can be acute (sudden onset) or chronic (developing gradually). The PDF note specifies: "Acute glomerulonephritis (GN) comprises a specific set of renal diseases in which an immunologic mechanism triggers inflammation and proliferation of glomerular tissue that can result in damage to the basement membrane, mesangium, or capillary endothelium alteration in renal function due to injury characterized by the classic symptoms of gross hematuria, mild proteinuria, edema (usually periorbital), hypertension, and oliguria. The glomerula capillaries inflame causing AGN."

1. Infections: Certain infections can trigger an immune response that leads to glomerular inflammation. Post-Streptococcal Glomerulonephritis (PSGN): Develops 1-2 weeks after an untreated throat infection (strep throat) or skin infection (impetigo) with specific strains of Group A Streptococcus bacteria. This is a common cause, especially in children. Bacterial Endocarditis: Infection of the heart valves can lead to immune complex deposition in the glomeruli. Viral Infections: Such as Hepatitis B, Hepatitis C, HIV. These can cause GN through various immune mechanisms. Other infections: Malaria, syphilis, some fungal infections.

2. Autoimmune Diseases (Immune System Disorders): Conditions where the body's immune system mistakenly attacks its own tissues, including the glomeruli. Systemic Lupus Erythematosus (SLE / Lupus Nephritis): Lupus is a chronic autoimmune disease that can affect many organs, including the kidneys. IgA Nephropathy (Berger's Disease): An autoimmune condition where IgA antibody deposits build up in the glomeruli, causing inflammation. Often triggered by respiratory or GI infections. Goodpasture Syndrome: A rare autoimmune disease where antibodies attack the basement membranes of the glomeruli and lungs. Vasculitis: Inflammation of blood vessels (e.g., ANCA-associated vasculitis like Wegener's granulomatosis or microscopic polyangiitis) can affect glomerular capillaries.

3. Genetic Factors / Family History of Kidney Disease: Some types of glomerulonephritis may have a genetic predisposition or run in families (e.g., Alport syndrome, some forms of IgA nephropathy). A family history of kidney disease in general can be a risk factor.

4. Certain Cancers: Some cancers, such as lung cancer, colon cancer, or blood cancers like lymphoma and multiple myeloma, can sometimes be associated with specific types of glomerulonephritis (paraneoplastic GN).

5. Exposure to Certain Medications or Toxins (Less Common): Prolonged or heavy use of certain drugs, like Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), or exposure to certain toxins (e.g., some solvents) may rarely trigger or worsen glomerular damage in susceptible individuals. Some antibiotics (like penicillamine) have also been implicated.

6. Chronic Inflammatory Conditions: Conditions with chronic inflammation, like diabetes or chronic Hepatitis C, can indirectly lead to glomerular damage over time. (Note: Diabetes typically causes diabetic nephropathy, a distinct form of kidney disease, but the chronic inflammation can be a factor).

7. Past or Current Kidney Problems: Having a history of kidney disease can make one more susceptible to further glomerular issues.

1. Managing Fluid Volume Excess: This is a primary concern due to decreased kidney function leading to salt and water retention. Assessment: Monitor for edema (periorbital, dependent), weight gain, crackles in lungs (pulmonary edema), shortness of breath, increased blood pressure, distended neck veins. Interventions: Strict intake and output monitoring, daily weights, fluid restriction as ordered, administration of diuretics (e.g., furosemide) as prescribed, low-sodium diet.

2. Monitoring and Maintaining Renal Function: Assessment: Observe urine characteristics (color - hematuria, amount - oliguria, specific gravity). Monitor laboratory results (BUN, creatinine, GFR, electrolytes). Interventions: Ensure adequate renal perfusion (manage BP), administer medications that protect kidneys if prescribed, avoid nephrotoxic drugs.

3. Blood Pressure Control: Hypertension is common and can worsen kidney damage. Assessment: Frequent BP monitoring. Interventions: Administer antihypertensive medications as prescribed, educate on low-sodium diet, promote rest.

4. Preventing and Managing Infections: Patients may be susceptible to infections, or an infection might be the cause (e.g., PSGN). Assessment: Monitor for signs of infection (fever, increased WBC). Interventions: Administer antibiotics if prescribed (e.g., for PSGN), maintain good hygiene, aseptic technique for procedures.

5. Ensuring Adequate Nutrition: Dietary modifications are often needed. Interventions: Educate on and provide appropriate diet often low sodium, sometimes low potassium or low protein (depending on severity of renal impairment and type of GN). Ensure adequate calorie intake to prevent catabolism. Encourage foods high in carbohydrates and fats if protein is restricted (as mentioned in PDF for acute phase).

6. Promoting Rest and Managing Activity Intolerance: Fatigue and weakness are common due to anemia and illness. Interventions: Encourage bed rest during acute phase, plan care to allow for rest periods, assist with ADLs as needed, gradually increase activity as tolerated.

7. Patient and Family Education and Support: Educate about the disease process, treatment plan, dietary and fluid restrictions, medications (purpose, side effects), signs of complications to report. Provide emotional support and address anxiety. Emphasize importance of long-term follow-up, including BP monitoring and urine tests.

8. Monitoring for Complications: Such as hypertensive encephalopathy, pulmonary edema, acute kidney injury progressing to chronic kidney disease, electrolyte imbalances (e.g., hyperkalemia).

Question 6

KABOONG COLLEGE OF HEALTH SCIENCES - NO.61

- An adult male patient has been diagnosed with nephrotic syndrome. List five signs and symptoms of nephrotic syndrome.

- Explain how you would manage the patient from the time of admission up to discharge.

- Outline five complications of nephrotic syndrome.

ANSWER: Examination Script

Definition of Nephrotic Syndrome (from PDF): Nephrotic syndrome is a collection of symptoms caused by various diseases that damage the kidney's filtering units (glomeruli). This damage leads to a severe and prolonged loss of protein (especially albumin) in the urine, decreased levels of protein in the blood, retention of excess salt and water (causing edema), and high levels of fats (lipids) in the blood. Alternatively: A condition of the urinary system where there is damage to the kidney glomeruli resulting in the escape of 3-3.5 grams of protein or more in urine daily.

1. Massive Proteinuria (Heavy Protein in Urine): This is the hallmark sign, defined as excretion of >3.0-3.5 grams of protein per 24 hours (or equivalent on a spot urine protein/creatinine ratio). Urine may appear frothy or foamy.

2. Hypoalbuminemia (Low Blood Albumin): Low levels of albumin (a major protein) in the blood due to its excessive loss in the urine and the liver's inability to compensate fully by producing more.

3. Edema (Generalized Swelling): Significant swelling due to fluid retention. Typically starts around the eyes (periorbital edema, especially in the morning) and ankles, but can become generalized (anasarca), affecting legs, abdomen (ascites), and lungs (pleural effusion). Caused by low plasma oncotic pressure (due to hypoalbuminemia) and sodium/water retention by the kidneys.

4. Hyperlipidemia (High Blood Lipids/Fats): Elevated levels of cholesterol, triglycerides, and other lipids in the blood. This is thought to be due to increased liver production of lipoproteins in response to low plasma oncotic pressure and loss of proteins that regulate lipid metabolism.

5. Lipiduria (Lipids in Urine): Fat bodies (oval fat bodies, fatty casts) may be present in the urine, giving it a milky appearance sometimes.

(Additional common signs) 6. Fatigue and Malaise: Feeling tired and generally unwell.

(Additional common signs) 7. Increased Susceptibility to Infections: Due to loss of immunoglobulins (antibodies) in the urine and impaired immune function.

(Additional common signs) 8. Thromboembolic Risk: Increased risk of blood clots (e.g., DVT, PE) due to loss of anticoagulant proteins in urine and increased production of procoagulant factors by the liver.

Admission: Admit to a medical ward, ensure a well-ventilated room.

Assessment: History Taking (Data Collection): Objective Data (From caretaker/relative, past medical records) and Subjective Data (From the patient himself - symptoms, concerns). Focus on onset of edema, any known kidney disease, diabetes, infections, medications. Physical Examination: Vital signs, weight, height (for BMI), assess extent of edema, skin integrity, respiratory status (for pleural effusion), cardiovascular status. Investigations: Urinalysis (For proteinuria, hematuria, casts, lipiduria). Blood Tests (FBC, UEC/RFTs, LFTs, serum albumin, lipid profile, blood glucose, HbA1c). Serological Tests (Hepatitis B/C, HIV, ANA, complement levels). Renal Ultrasound (To assess kidney size and structure). Kidney Biopsy (Often essential to determine the specific type of glomerular disease causing nephrotic syndrome and guide treatment, especially in adults).

1. Management of Edema: Dietary Sodium Restriction: Limit salt intake (e.g., Fluid Restriction: May be necessary if severe edema or hyponatremia (low sodium). Based on urine output and insensible losses. Diuretics: Loop diuretics (e.g., Furosemide) are often used, sometimes in combination with thiazide diuretics or potassium-sparing diuretics (like spironolactone, especially if hypoalbuminemia is severe, to counteract aldosterone effects). Monitor response and electrolytes. Elevation of Edematous Limbs. Skin care to prevent breakdown. IV Albumin Infusion: Sometimes used in severe hypoalbuminemia with resistant edema to temporarily increase oncotic pressure and improve diuretic response, but its routine use is controversial.

2. Reduction of Proteinuria: ACE Inhibitors (e.g., Enalapril, Lisinopril) or Angiotensin II Receptor Blockers (ARBs, e.g., Losartan, Valsartan): These drugs help reduce proteinuria and can slow progression of kidney disease, also help control blood pressure. Dietary Protein: Moderate protein intake is usually recommended (not high, not severely restricted unless advanced renal failure).

3. Management of Hyperlipidemia: Dietary modifications: Low saturated fat, low cholesterol diet. Lipid-lowering medications (e.g., Statins like Atorvastatin, Simvastatin) if lifestyle changes are insufficient and cardiovascular risk is high.

4. Treatment of Underlying Cause (if identified from biopsy/investigations): Immunosuppressive Therapy: For many primary glomerular diseases like Minimal Change Disease, Focal Segmental Glomerulosclerosis (FSGS), Membranous Nephropathy, or lupus nephritis. Drugs include corticosteroids (e.g., Prednisolone), cyclophosphamide, cyclosporine, tacrolimus, mycophenolate mofetil, rituximab. Treatment of infections if they are the trigger.

5. Prevention and Management of Complications: Infections: Vigilant monitoring for signs of infection. Prompt antibiotic treatment. Pneumococcal and influenza vaccinations recommended. Thromboembolism: Prophylactic anticoagulation (e.g., low molecular weight heparin or warfarin) may be considered for high-risk patients (e.g., very low albumin, history of clots, certain types of GN). Encourage mobilization. Acute Kidney Injury: Monitor renal function closely, manage fluid balance carefully.

6. Blood Pressure Control: Use antihypertensives (ACE inhibitors/ARBs are often first-line) to achieve target BP (e.g., <130 /80 mmHg or lower if proteinuria significant).

7. Nutritional Support: Ensure adequate calorie intake. Refer to a dietitian for personalized advice on protein, sodium, fluid, and fat intake.

8. Monitoring: Daily weights, strict fluid balance, regular vital signs (especially BP), monitoring urine for protein, regular blood tests (UEC, albumin, lipids), monitoring for side effects of medications (especially immunosuppressants).

9. Patient Education and Psychological Support: Explain the condition, treatment plan, importance of adherence, dietary/fluid restrictions, and signs of complications. Address anxiety and concerns.

Stable Condition: Edema controlled, proteinuria reducing (if treatment effective), BP managed, no acute complications.

Medication Management: Clear instructions on all discharge medications (purpose, dose, frequency, side effects).

Dietary and Fluid Instructions: Reinforce any ongoing restrictions.

Self-Monitoring: Teach how to monitor weight, BP (if home monitor available), urine (for frothiness), and signs of worsening edema or infection.

Follow-up: Schedule regular follow-up appointments with nephrologist/physician for ongoing monitoring of kidney function, proteinuria, BP, and adjustment of treatment. Long-term follow-up is essential.

Lifestyle Advice: E.g., avoiding nephrotoxic drugs, managing other comorbidities.

1. Infections: Increased susceptibility to bacterial infections (e.g., peritonitis - especially Spontaneous Bacterial Peritonitis, cellulitis, pneumonia, sepsis) due to loss of immunoglobulins (antibodies) and complement components in the urine, and impaired T-cell function.

2. Thromboembolism (Blood Clots): Increased risk of venous thromboembolism (VTE), including Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE), as well as renal vein thrombosis. This is due to urinary loss of anticoagulant proteins (like antithrombin III, Protein S), increased liver synthesis of procoagulant factors (like fibrinogen), increased platelet aggregation, and hyperlipidemia.

3. Hyperlipidemia and Atherosclerosis: Persistently high levels of cholesterol and triglycerides can accelerate the development of atherosclerosis (hardening and narrowing of arteries), increasing long-term risk of cardiovascular disease (heart attack, stroke).

4. Acute Kidney Injury (AKI) / Renal Failure: Can occur due to severe hypovolemia (intravascular volume depletion despite edema, sometimes called "hypovolemic crisis"), renal vein thrombosis, interstitial edema within the kidneys, or progression of the underlying glomerular disease. Chronic kidney disease (CKD) and end-stage renal disease (ESRD) are long-term risks.

5. Malnutrition / Protein-Energy Wasting: Significant ongoing protein loss in urine can lead to muscle wasting and poor nutritional status if not adequately compensated by dietary intake or if appetite is poor. Vitamin D deficiency and bone disease can also occur.

6. Complications of Edema: Severe edema can cause skin breakdown, cellulitis, discomfort, reduced mobility, pleural effusions (difficulty breathing), and ascites.

7. Complications of Treatment: Side effects from medications used to treat nephrotic syndrome, such as corticosteroids (e.g., infections, hyperglycemia, osteoporosis, Cushingoid features) or other immunosuppressants (e.g., increased infection risk, bone marrow suppression).

8. Hypovolemic Crisis (mentioned in PDF): Occurs when severe hypoalbuminemia leads to a significant shift of plasma water into the interstitial space, causing a decrease in circulating blood volume despite visible edema, potentially leading to shock.

Question 7

LACOR HOSPITAL TRAING SCHOOL - NO.63

- Outline 3 types of meningitis.

- List the clinical features of Cryptococcal meningitis.

- Describe the management of a child admitted to the ward with a diagnosis of meningococcal meningitis.

ANSWER: Examination Script

Meningitis is an acute or chronic inflammation of the meninges, which are the protective membranes covering the brain and spinal cord (pia mater, arachnoid mater, dura mater).

1. Bacterial Meningitis (Septic Meningitis): Caused by bacterial infection. This is a medical emergency and can be life-threatening if not treated promptly with antibiotics. Common causative bacteria include Streptococcus pneumoniae (pneumococcus), Neisseria meningitidis (meningococcus), Haemophilus influenzae type b (Hib - less common now due to vaccination), Listeria monocytogenes, and Group B Streptococcus (in newborns).

2. Viral Meningitis (Aseptic Meningitis): Caused by viral infection. It is usually less severe than bacterial meningitis and often resolves on its own without specific antiviral treatment (supportive care is given). Common causative viruses include enteroviruses, herpes simplex virus (HSV), varicella-zoster virus (VZV), mumps virus, and HIV.

3. Fungal Meningitis: Caused by fungal infection. It is relatively rare and typically occurs in individuals with weakened immune systems (immunocompromised), such as those with HIV/AIDS, cancer, or on immunosuppressive drugs. Cryptococcus neoformans is a common cause (Cryptococcal meningitis). Treatment involves long courses of antifungal medications.

(Additional Types often mentioned): Tuberculous (TB) Meningitis: Caused by Mycobacterium tuberculosis. Develops more slowly (subacute or chronic) and requires long-term anti-TB treatment. Parasitic Meningitis: Rare, caused by parasites like Naegleria fowleri (primary amoebic meningoencephalitis) or Angiostrongylus cantonensis (eosinophilic meningitis). Non-Infectious Meningitis: Can be caused by certain drugs, cancers, or inflammatory conditions like lupus, but this is distinct from infectious meningitis. Neonatal Meningitis: Meningitis occurring in the first month of life, often caused by GBS, E. coli, Listeria. (PDF point)

Headache: Often the most common and persistent symptom. Can be severe, throbbing, and gradually worsening.

Fever: May be present, but can also be low-grade or absent, especially in severely immunocompromised patients.

Malaise / Fatigue: General feeling of being unwell, tired, and lacking energy. Often develops over one or two weeks (PDF point).

Nausea and Vomiting: Can occur due to increased intracranial pressure or direct irritation of meninges.

Altered Mental Status / Confusion: Ranging from mild confusion, lethargy, or personality changes to drowsiness, stupor, or coma in severe cases.

Neck Stiffness (Nuchal Rigidity): Pain and resistance when trying to flex the neck forward. May be less pronounced than in bacterial meningitis, especially in HIV patients.

Photophobia (Intolerance to Light): Light sensitivity causing eye discomfort.

Visual Disturbances: Blurred vision, double vision (diplopia), or vision loss can occur due to increased intracranial pressure or direct involvement of optic nerves.

Cranial Nerve Palsies: Weakness or paralysis of muscles controlled by cranial nerves (e.g., facial droop, eye movement problems).

Seizures: Can occur, especially if there is significant brain inflammation or increased intracranial pressure.

Increased Intracranial Pressure (ICP) Signs: Besides headache and vomiting, may include papilledema (swelling of optic disc on fundoscopy), sixth nerve palsy. Lumbar puncture often reveals very high opening pressure.

Behavioral Changes / Personality Changes.

Gait Ataxia (Unsteady Walk).

Hearing Loss.

Symptoms of Underlying Immunocompromise: E.g., signs of advanced HIV/AIDS. (PDF point: "The patient is immune suppressed").

CSF Findings (from PDF point about CSF): CSF when examined (from lumbar puncture) may show presence of yeast (Cryptococcus seen with India ink stain) and may appear cloudy or have elevated protein and low glucose, with pleocytosis (increased white cells, often lymphocytes).

1. Isolation and Infection Control: Implement droplet precautions immediately (e.g., surgical mask for patient if tolerated, staff wear masks when close) for at least 24 hours after starting effective antibiotic therapy to prevent spread. Single room if possible.

2. ABCs (Airway, Breathing, Circulation): Ensure patent airway. Position child appropriately. Administer high-flow oxygen. Assist ventilation if needed. Establish IV access quickly with large-bore cannula. If signs of shock (hypotension, tachycardia, poor perfusion), give rapid IV fluid bolus (e.g., 20ml/kg normal saline or Ringer's lactate) and vasopressors if needed.

3. Prompt Administration of Antibiotics: This is critical and should not be delayed by investigations if meningitis is strongly suspected. Preferred: Intravenous (IV) Ceftriaxone or Cefotaxime (third-generation cephalosporins) are usually first-line. Penicillin G can also be used if strain is known to be sensitive. Give the first dose as soon as possible, ideally within 30 minutes of suspicion.

4. Lumbar Puncture (LP) for Cerebrospinal Fluid (CSF) Analysis: Perform LP as soon as possible (if no contraindications like severe shock, bleeding disorder, or signs of very high ICP with focal neurological signs/papilledema CT scan may be needed first in some cases) to confirm diagnosis, identify organism, and guide antibiotic choice. Send CSF for Gram stain, culture, cell count, protein, glucose. (PDF point: "Proper identification of organisms by gram staining").

5. Corticosteroids (e.g., Dexamethasone): May be considered, especially in suspected bacterial meningitis in children >6 weeks old, given just before or with the first dose of antibiotics. It may reduce inflammation and risk of neurological complications like hearing loss. Benefit is most clear for Hib meningitis, less so for meningococcal.

6. Monitoring Vital Signs and Neurological Status: Frequent monitoring of temperature, pulse, respirations, blood pressure, oxygen saturation, level of consciousness (e.g., using GCS or AVPU), pupil size/reaction, signs of increased ICP (headache, vomiting, bulging fontanelle in infants, altered consciousness), and seizure activity.

7. Fluid and Electrolyte Management: Maintain IV fluids carefully to ensure adequate hydration and renal perfusion, but avoid fluid overload, especially if there's risk of cerebral edema or SIADH (Syndrome of Inappropriate Antidiuretic Hormone secretion). Monitor urine output, electrolytes.

8. Management of Seizures: If seizures occur, manage with IV anticonvulsants (e.g., Diazepam, Lorazepam, Phenobarbital, Phenytoin) as per protocol. Ensure airway safety during seizures.

9. Management of Increased Intracranial Pressure (ICP): If signs of raised ICP, nurse with head of bed elevated (30 degrees), maintain head in midline position, avoid fluid overload, consider osmotic diuretics (e.g., Mannitol) or hypertonic saline if severe, under specialist guidance.

10. Nutritional Support: Enteral feeding (NG tube) or parenteral nutrition if child is unable to take orally for a prolonged period. (PDF point: "NGT is passed for feeding and to aspirate the gastric content" - context from Lacor PDF seems to have general meningitis management points).

11. Supportive Care: Pain relief (e.g., paracetamol). Antipyretics for fever. (PDF point: "Tepid sponging... antipyretic also given e.g. diclofenac" - Diclofenac use in children needs caution). Maintain quiet, calm environment. Minimize handling if irritable. Care of skin, mouth, eyes if unconscious or very ill. (PDF point: "Do oral care and personal hygiene to the patient").

12. Chemoprophylaxis for Close Contacts: Identify close contacts (household members, daycare attendees, those with direct saliva exposure) and provide antibiotic prophylaxis (e.g., Rifampicin, Ciprofloxacin, or Ceftriaxone) to prevent secondary cases. Public health notification.

13. Monitoring for and Managing Complications: Such as hearing loss (audiology assessment needed), seizures, hydrocephalus, subdural effusion, cranial nerve palsies, learning difficulties, skin necrosis (with meningococcemia).

14. Psychological Support for Child and Family: Provide information, reassurance, and support. Allow family presence as appropriate. (PDF point: "Alley anxiety from both patient and attendants").

Completion of Antibiotic Course: Ensure full course of IV antibiotics (typically 7-14 days for meningococcal) is completed or a plan for transition to oral antibiotics if appropriate.

Clinical Stability: Afebrile, neurologically stable, feeding well.

Education: Teach parents about signs of complications or relapse, importance of follow-up.

Follow-up: Arrange for audiology testing, neurodevelopmental assessment, and any other necessary specialist follow-up. Ensure routine immunizations are up to date (including meningococcal vaccine if appropriate for age/type and not already given).

Question 8

NSYAMBYA SCHOOL OF NURSING AND MIDWIFERY - NO.60

A known diabetic patient has been brought to OPD in hyperglycemic coma.

- List five signs and symptoms he is likely to present with.

- Describe his management from admission to discharge.

- The patient is prone to diabetic foot. Explain 10 preventive measures of diabetic foot.

ANSWER: Examination Script

1. Altered Level of Consciousness / Coma: Ranging from drowsiness, confusion, lethargy to profound unresponsiveness (coma). This is due to severe dehydration, electrolyte imbalances, hyperosmolarity, and direct effects of ketoacidosis (in DKA) on the brain.

2. Severe Dehydration: Dry mucous membranes (mouth, tongue). Poor skin turgor (skin slow to return when pinched). Sunken eyes. Tachycardia (rapid heart rate). Hypotension (low blood pressure, especially orthostatic).

3. Polyuria and Polydipsia (Initially, leading to Dehydration): Before coma develops, the patient likely experienced excessive urination (due to osmotic diuresis from high blood glucose) and extreme thirst as the body tried to compensate for fluid loss.

4. Specific Breathing Patterns: Kussmaul Respirations (in DKA): Deep, rapid, sighing respirations as the body tries to blow off excess CO2 to compensate for metabolic acidosis. Tachypnea (Rapid Breathing): Can also be present in HHS due to dehydration or associated conditions.

5. Fruity Odor on Breath (Acetone Breath - in DKA): A sweet, fruity smell on the breath due to the presence of ketones, which are produced when the body breaks down fat for energy in the absence of sufficient insulin. Not typically present in HHS.

(Additional common signs) 6. Nausea, Vomiting, Abdominal Pain: Common, especially in DKA, can worsen dehydration.

(Additional common signs) 7. Very High Blood Glucose Levels: Confirmed by blood glucose testing (often >13.9 mmol/L or 250 mg/dL in DKA, and often much higher, e.g., >33.3 mmol/L or 600 mg/dL, in HHS).

(Additional common signs) 8. Ketones in Urine and Blood (in DKA).

(Additional common signs) 9. Signs of Precipitating Illness: Fever, cough, etc., if an infection triggered the hyperglycemic crisis.

ABCs: Ensure Airway, Breathing (oxygen if hypoxic), Circulation (IV access).

Confirm Diagnosis: Rapid blood glucose test, urine ketones, blood ketones (if available), arterial/venous blood gas (for pH, bicarbonate - key for DKA).

Fluid Resuscitation: Aggressive IV fluid replacement is critical. Initially with isotonic saline (0.9% NaCl), e.g., 1-2 liters in the first 1-2 hours for an adult, then adjusted based on hydration status, electrolytes, and urine output. Once blood glucose falls to around 12-14 mmol/L (200-250 mg/dL), change IV fluids to include dextrose (e.g., 5% Dextrose in 0.45% Saline) to prevent hypoglycemia and cerebral edema.

Insulin Therapy: Start with a continuous IV infusion of regular (short-acting) insulin (e.g., 0.1 units/kg/hour). Aim for a gradual fall in blood glucose (e.g., 3-4 mmol/L or 50-75 mg/dL per hour). Avoid rapid drops. Do NOT start insulin until potassium levels are known and are >3.3 mmol/L (insulin drives potassium into cells, worsening hypokalemia). Correct hypokalemia first.

Electrolyte Correction: Potassium: Patients are often total-body potassium depleted even if initial serum levels are normal or high (due to acidosis shifting K+ out of cells). Add potassium to IV fluids once urine output is established and serum K+ starts to fall with insulin and fluid therapy. Monitor K+ levels very closely. Bicarbonate (for DKA): Routine use is controversial. May be considered in severe acidosis (e.g., pH Phosphate: May need replacement if severely low.

Monitoring: Hourly blood glucose, electrolytes (K+, Na+, bicarbonate), BUN, creatinine, blood gases (for DKA). Frequent vital signs, level of consciousness, fluid balance.

Identify and Treat Precipitating Cause: E.g., infection (antibiotics), non-compliance with insulin, myocardial infarction.

Continue IV insulin until DKA/HHS resolves: (ketones clear, acidosis corrects for DKA; osmolality normalizes and mental status improves for HHS) and patient can eat.

Transition to Subcutaneous Insulin: Once stable and eating, switch to a subcutaneous insulin regimen. Overlap IV and SC insulin for 1-2 hours to prevent rebound hyperglycemia.

Patient Education: Crucial to prevent recurrence. Teach about diabetes, blood glucose monitoring, insulin administration, sick day rules, signs of hypo/hyperglycemia, foot care, diet, exercise.

Nutritional Management: Referral to a dietitian.

Address Psychosocial Issues.

Criteria: Medically stable, blood glucose controlled on SC insulin, eating normally, underlying cause treated, patient/family educated and competent in self-management.

Follow-up: Arrange close follow-up with diabetes clinic/specialist.

1. Daily Foot Inspection: Inspect all surfaces of both feet daily (including between toes) for any cuts, blisters, redness, swelling, calluses, or changes in skin color or temperature. Use a mirror if difficult to see the bottom of the feet, or ask a family member for help. (Reason: Early detection of minor problems allows for prompt treatment, preventing them from becoming serious ulcers or infections. Neuropathy can mask pain, so visual inspection is crucial.)

2. Good Foot Hygiene: Wash feet daily with lukewarm (not hot) water and mild soap. Dry thoroughly, especially between the toes. (Reason: Keeps skin clean, reduces risk of fungal and bacterial infections. Prevents maceration between toes.)

3. Moisturize Dry Skin: Apply a thin coat of moisturizing lotion or cream to the tops and bottoms of feet to prevent dry, cracked skin, but AVOID applying between the toes (can cause maceration). (Reason: Dry skin is prone to cracking, which can create entry points for infection.)

4. Proper Nail Care: Cut toenails straight across and file edges smooth. Avoid cutting too short or digging into corners. If vision is poor or nails are thick/difficult to cut, seek help from a podiatrist or trained healthcare provider. (Reason: Prevents ingrown toenails and accidental injury.)

5. Wear Appropriate Footwear (Shoes and Socks): Shoes: Wear well-fitting, comfortable, supportive shoes with a wide toe box that do not rub or cause pressure. Avoid tight shoes, high heels, pointed toes, or open-toed shoes/sandals (which offer less protection). Check inside shoes for foreign objects before wearing. Socks: Wear clean, soft, well-fitting socks (preferably cotton or wool, or special diabetic socks that are seamless and non-constricting). Change socks daily. Avoid socks with tight elastic bands. Reason: Protects feet from injury, reduces pressure points, and helps keep feet dry.

6. Avoid Walking Barefoot (Indoors and Outdoors): Always wear shoes or slippers to protect feet from cuts, punctures, burns, or other injuries that may go unnoticed due to neuropathy. (Reason: Prevents accidental injury to feet that may not be felt.)

7. Protect Feet from Extreme Temperatures: Avoid exposing feet to very hot water, heating pads, or hot surfaces (can cause burns if sensation is reduced). Also, protect feet from extreme cold (can worsen circulation). Test water temperature with elbow or thermometer before bathing. (Reason: Reduced sensation increases risk of burns or frostbite.)

8. Promote Good Blood Circulation to Feet: Avoid crossing legs for long periods. Wiggle toes and move ankles frequently throughout the day. Elevate feet when sitting if possible. Avoid tight socks or garters that can restrict circulation. (Reason: Improves blood flow, which is essential for tissue health and healing.)

9. Regular Professional Foot Examinations: Have feet checked by a doctor, nurse, or podiatrist at least once a year (more often if high risk or existing problems). They can assess sensation, circulation, identify early problems, and provide specialized care. (Reason: Professional assessment can detect problems the patient might miss and provide expert advice and treatment.)

10. Good Blood Glucose Control: Maintain blood sugar levels as close to target range as possible through diet, exercise, and medication/insulin as prescribed. (Reason: Poor glucose control is a major contributor to neuropathy and vascular disease, which are the underlying causes of most diabetic foot problems. Good control slows progression of these complications.)

11. Promptly Treat Minor Foot Problems: Clean minor cuts or sores with mild soap and water, apply an antiseptic, and cover with a sterile dressing. Seek medical attention immediately for any foot injury that doesn't heal, shows signs of infection (redness, swelling, pus, warmth), or if a new ulcer develops. Do not self-treat corns or calluses with sharp instruments or harsh chemicals.

12. Quit Smoking: Smoking damages blood vessels and reduces circulation to the feet, significantly increasing the risk of foot problems and impairing healing.

Question 9

ACCESS SCHOOL OF NURSING AND MIDWIFERY - NO.62

An outbreak of measles has been identified in Kampala suburb.

- Mention ten signs and symptom of measles.

- Explain the preventive measures of measles in your community.

- Explain how you would manage 5yrs old child diagnosed with measles up to discharge using a nursing process.

ANSWER: Examination Script

1. Fever: Usually high fever (can reach 39-40.5 degC or 103-105 degF), often one of the first symptoms.

2. Cough: A persistent, dry, hacking cough is characteristic.

3. Coryza (Runny Nose): Similar to a common cold, with clear nasal discharge.

4. Conjunctivitis (Red, Watery Eyes): Inflammation of the conjunctiva, leading to redness, watering, and often photophobia (sensitivity to light). Often referred to as the "3 Cs" - Cough, Coryza, Conjunctivitis.

5. Koplik's Spots: Small, white or bluish-white spots with a red base found on the inside of the cheeks (buccal mucosa) opposite the molars. They are pathognomonic (specifically characteristic) of measles and appear 1-2 days before the skin rash.

6. Maculopapular Rash: The characteristic measles rash appears 3-5 days after initial symptoms. Appearance: Starts as flat red spots (macules) which then become raised bumps (papules), often merging together (confluent). Distribution: Typically begins on the face (behind the ears and along the hairline) and then spreads downwards over the neck, trunk, arms, and legs, usually reaching the feet in 2-3 days. Progression: The rash may become blotchy and can sometimes be itchy. It usually lasts 5-6 days and then fades in the same order it appeared, often leaving a brownish discoloration and fine desquamation (skin peeling).

7. Malaise / Fatigue: General feeling of being unwell, tired, and lacking energy.

8. Anorexia (Loss of Appetite).

9. Sore Throat.

10. Lymphadenopathy (Swollen Lymph Nodes): Especially in the neck.

11. Diarrhea (more common in young children and malnourished individuals).

12. Photophobia (Sensitivity to Light) - due to conjunctivitis.

1. Vaccination (Immunization): This is the most effective preventive measure. The measles vaccine (usually given as part of MMR - Measles, Mumps, Rubella, or MR - Measles, Rubella vaccine) is safe and highly effective. Routine Immunization: Ensure all children receive two doses of measles-containing vaccine according to the national immunization schedule (e.g., first dose around 9 or 12-15 months, second dose later, e.g., 15-18 months or 4-6 years). Supplementary Immunization Activities (SIAs) / Campaigns: Conduct mass vaccination campaigns, especially in areas with low routine coverage or during outbreaks, to reach unimmunized or under-immunized children. Catch-up Vaccination: For older children, adolescents, or adults who missed doses.

2. Isolation of Cases: Individuals diagnosed with measles should be isolated (e.g., stay home from school, work, public places) for at least 4 days after the onset of the rash to prevent spreading the virus to others. Healthcare workers caring for measles patients should use airborne precautions.

3. Post-Exposure Prophylaxis (PEP) for Susceptible Contacts: Vaccination: Susceptible (unimmunized or inadequately immunized) contacts exposed to measles can be given the measles vaccine within 72 hours of exposure to potentially prevent or modify the illness. Immunoglobulin (IG): For high-risk susceptible contacts (e.g., infants

4. Health Education and Community Awareness: Educate the community about how measles is spread, its signs and symptoms, the importance and safety of vaccination, and when to seek medical care. Dispel myths and misinformation about vaccines.

5. Promoting Good Personal Hygiene and Respiratory Etiquette: Encourage handwashing, covering coughs and sneezes, and proper disposal of tissues, although vaccination is the primary control.

6. Early Case Detection and Reporting (Surveillance): Promptly identify and report suspected measles cases to public health authorities to allow for rapid investigation, contact tracing, and implementation of control measures to prevent further spread.

7. Improving Nutritional Status: Good nutrition, especially adequate Vitamin A, can reduce the severity and complications of measles. Vitamin A supplementation is part of measles treatment.

8. Maintaining High Herd Immunity: When a large percentage of the population (e.g., >95%) is immunized, it protects even those who are not vaccinated by reducing the overall circulation of the virus (herd immunity).

1. Assessment: History: Onset and nature of symptoms (fever, cough, coryza, conjunctivitis, rash), exposure history, immunization status, nutritional status, any underlying conditions. Physical Examination: Vital signs (temperature, pulse, respirations, SpO2 if available). Inspect for Koplik's spots, rash characteristics and distribution. Assess hydration status (skin turgor, mucous membranes, urine output). Respiratory assessment (rate, effort, breath sounds for pneumonia). Neurological status. Look for signs of complications (e.g., earache, severe diarrhea, dehydration, convulsions, clouding of cornea).

2. Nursing Diagnoses (Examples): Hyperthermia related to viral infection as evidenced by elevated temperature. Risk for Deficient Fluid Volume related to fever, anorexia, vomiting, or diarrhea. Impaired Skin Integrity related to rash and potential itching. Risk for Imbalanced Nutrition: Less than Body Requirements related to anorexia, sore throat, or oral lesions. Risk for Infection (secondary bacterial infection, e.g., pneumonia, otitis media) related to compromised immune response from measles. Acute Pain/Discomfort related to rash, fever, conjunctivitis, cough, sore throat. Knowledge Deficit (Parental) related to measles, home care, and prevention of spread.

3. Planning (Goals): Child will maintain normal body temperature. Child will maintain adequate hydration and nutrition. Child's skin integrity will remain intact or improve. Child will be free from or have early detection and management of complications. Child will experience relief from discomfort. Parents will demonstrate understanding of measles, home care, and preventive measures.

4. Implementation (Nursing Interventions): Isolation: Implement airborne precautions if hospitalized (or advise on home isolation keep child away from others, especially unimmunized individuals, for 4 days after rash onset). Fever Management: Administer antipyretics (e.g., paracetamol) as prescribed. Encourage fluid intake. Tepid sponging if very high fever. Avoid over-bundling. Hydration and Nutrition: Encourage frequent small amounts of oral fluids (water, juice, ORS if diarrhea). Offer soft, appealing, nutritious foods. Continue breastfeeding if applicable. Monitor intake/output. IV fluids if severe dehydration or unable to take orally. Eye Care: Clean eyes gently with normal saline if discharge present. Dim lights if photophobic. Skin Care: Keep skin clean. Tepid baths or calamine lotion may soothe itching (if present). Keep fingernails short to prevent scratching. Respiratory Support: Encourage rest. Humidified air may soothe cough. Monitor for signs of pneumonia (fast/difficult breathing). Vitamin A Supplementation: Administer Vitamin A as per WHO guidelines (two doses on consecutive days, age-appropriate dose) to all children with measles, as it reduces severity and mortality. Monitor for Complications: Regularly assess for signs of pneumonia, otitis media, diarrhea, dehydration, encephalitis, eye complications. Report promptly. Antibiotics: Only if a secondary bacterial infection (e.g., pneumonia, otitis media) is diagnosed or strongly suspected. Measles itself is viral and not treated by antibiotics. Health Education to Parents: Explain the illness, its contagious nature, importance of completing Vitamin A, home care (fluids, nutrition, fever control), signs of complications requiring immediate return, and importance of immunization for future prevention for other children/contacts. Comfort and Rest: Provide a quiet, comfortable environment. Encourage rest.

5. Evaluation (and leading to Discharge): Assess if goals are met: Temperature normalized, child is well-hydrated and eating/drinking adequately, rash fading, no signs of complications. Evaluate parents' understanding of discharge instructions and home care. Child is afebrile for at least 24 hours without antipyretics, clinically stable, and appetite has returned. Discharge planning includes advice on when the child can return to school (usually after infectious period), follow-up if needed, and reinforcing immunization for household contacts if not up-to-date.

Question 10

MOYO SCHOOL OF NURSING AND MIDWIFERY - NO.64

- Outline 5 clinical features of PUD. (Peptic Ulcer Disease)

- Outline 10 nursing intervention for the patient admitted with PUD.

- List the 5 complications of the above condition.

ANSWER: Examination Script

1. Epigastric Pain (Abdominal Pain): This is the most common symptom. Often described as a burning, gnawing, aching, or hunger-like pain in the upper abdomen (epigastrium), between the breastbone and navel. Duodenal Ulcer Pain: Typically occurs 2-3 hours after meals, or at night (may wake patient). Often relieved by eating food or taking antacids (as food buffers the acid). Pain may be periodic (comes and goes for days or weeks). Gastric Ulcer Pain: Often occurs shortly after eating (food may worsen the pain, or sometimes relieve it initially then worsen). Less predictable pattern than duodenal ulcer pain.

2. Dyspepsia (Indigestion): A general term for discomfort in the upper abdomen, which can include bloating, belching (burping), feeling of fullness, and early satiety (feeling full quickly after eating little).

3. Nausea and/or Vomiting: Some patients may experience nausea, and less commonly, vomiting. Vomiting may relieve pain in some cases, especially with gastric outlet obstruction (a complication).

4. Loss of Appetite and Unintentional Weight Loss: Pain associated with eating (especially with gastric ulcers) or fear of pain can lead to reduced food intake and subsequent weight loss.

5. Heartburn (Acid Reflux): A burning sensation in the chest, often rising from the stomach towards the throat, due to stomach acid backing up into the esophagus. Can occur with PUD, though also a primary symptom of GERD.

(Signs of Complications can also be presenting features): Melena (Black, Tarry Stools): Indicates upper gastrointestinal bleeding (digested blood). Hematemesis (Vomiting Blood): Can be bright red blood or dark "coffee-ground" like material, indicating upper GI bleeding. Sudden, Severe Abdominal Pain: May indicate perforation (ulcer has eroded through the stomach/duodenal wall). Symptoms of Anemia (from chronic blood loss): Fatigue, weakness, pallor, shortness of breath.

1. Pain Assessment and Management: Assess pain regularly (location, character, severity, timing in relation to meals). Administer prescribed medications to reduce acid and protect mucosa: Proton Pump Inhibitors (PPIs, e.g., Omeprazole, Pantoprazole) or H2 Receptor Antagonists (H2RAs, e.g., Ranitidine - though less used now, Famotidine), Antacids (e.g., Magnesium Hydroxide/Aluminum Hydroxide) for symptomatic relief, Mucosal protectants (e.g., Sucralfate). Advise on avoiding foods/drinks that aggravate pain.

2. Monitor for Signs of GI Bleeding: Observe for hematemesis, melena, or coffee-ground vomitus. Monitor vital signs for tachycardia, hypotension (signs of shock). Check hemoglobin/hematocrit. Test stool/vomitus for occult blood if ordered.

3. Administer Medications to Eradicate H. pylori (if present): If H. pylori infection is confirmed, ensure patient takes the prescribed combination therapy (usually two antibiotics + a PPI) correctly and completes the full course.

4. Provide Dietary Counseling and Support: Advise avoiding irritant foods/drinks: Spicy foods, acidic foods (citrus, tomatoes), caffeine, alcohol, carbonated beverages. Encourage small, frequent meals rather than large ones if this helps comfort. Advise against eating late at night or close to bedtime if reflux is an issue. Emphasize importance of not skipping meals if pain is relieved by food (duodenal ulcer pattern).

5. Educate on Lifestyle Modifications: Smoking Cessation: Smoking impairs ulcer healing and increases recurrence. Provide resources. Alcohol Limitation/Avoidance: Alcohol can irritate the stomach lining. Stress Management: Teach relaxation techniques, as stress can exacerbate symptoms. NSAID Avoidance: Advise to avoid or use NSAIDs (aspirin, ibuprofen) very cautiously and with gastric protection if absolutely necessary. Discuss alternatives with doctor.

6. Maintain Fluid and Electrolyte Balance (especially if vomiting or bleeding): Monitor intake/output. Administer IV fluids if NPO or dehydrated.

7. Prepare for and Provide Post-Procedure Care (if endoscopy or surgery needed): If endoscopy is done for diagnosis or treatment of bleeding, provide pre- and post-procedure care. If surgery is required for complications, provide appropriate surgical nursing care.

8. Monitor for Complications of PUD: Be alert for signs of perforation (sudden severe abdominal pain, rigid abdomen), gastric outlet obstruction (persistent vomiting, abdominal distension), or worsening bleeding. Report immediately.

9. Provide Psychological Support: Address anxiety related to pain, diagnosis, or potential complications. Allow patient to verbalize concerns.

10. Discharge Planning and Education for Self-Management: Ensure understanding of medication regimen (purpose, dose, side effects, importance of adherence). Reinforce dietary and lifestyle modifications. Teach signs and symptoms of recurrence or complications that require medical attention. Importance of follow-up appointments.

11. If Bleeding is Active (Admission for GI Bleed): NPO status, IV access, vital signs, oxygen, NG tube for lavage (controversial), blood transfusion readiness, preparation for endoscopy.

1. Hemorrhage (Bleeding): This is the most common complication. The ulcer erodes into an underlying blood vessel, causing bleeding into the GI tract. Can be acute (sudden, severe) or chronic (slow, leading to anemia). Manifests as hematemesis or melena.

2. Perforation: The ulcer erodes completely through the wall of the stomach or duodenum, creating a hole that allows gastric or duodenal contents to leak into the peritoneal cavity. This causes sudden, severe abdominal pain, a rigid (board-like) abdomen, and peritonitis. A surgical emergency.

3. Gastric Outlet Obstruction (Pyloric Stenosis in adults): Swelling (edema), inflammation, spasm, or scarring from an ulcer near the pylorus (the opening from the stomach into the duodenum) can narrow or block this outlet. This prevents food from leaving the stomach, leading to persistent vomiting (often of undigested food), abdominal distension, early satiety, and weight loss.

4. Penetration: The ulcer erodes into an adjacent organ, such as the pancreas, liver, or biliary tract, without free perforation into the peritoneal cavity. This can cause intense, persistent pain that may radiate to the back (if pancreas involved).

5. Increased Risk of Gastric Cancer (for H. pylori-associated gastric ulcers): Chronic H. pylori infection, a major cause of PUD, is also a significant risk factor for developing certain types of stomach cancer (gastric adenocarcinoma and MALT lymphoma) over a long period. Duodenal ulcers are not typically associated with an increased cancer risk.

6. Anemia: Chronic, slow blood loss from an ulcer can lead to iron-deficiency anemia.

High-Yield Clinical Summary: Medicine Questions and Answers

Clinical Section / Focus | Key Features & Exam Points | Nursing Management & Rationale

NGORA SCHOOL NURSING AND MIDWIFERY - NO.39 | Define the term leukemia. | List the types of leukemia.

IGANGA SCHOOL OF NURSING AND MIDWIFERY - NO.40 | List the cardinal signs of pulmonary TB. | Outline the nursing intervention of a patient with pulmonary TB.

DOKOLO SCHOOL OF NURSING AND MIDWIFERY - NO.41 | Define rheumatic fever. | List 6 causes of rheumatic fever.

KIBULI MUSLIM HOSPITAL TRAINING SCHOOL - NO.42 | Outline 5 (five) cardinal signs of diabetes mellitus. | Prepare a diabetic plate.

MASAKA SCHOOL OF COMPREHENSIVE NURSING - NO.43 | Define the term glomerulonephritis. | Explain 5 (five) risk factors for glomerulonephritis.