CHAPTER 16:

PROVIDE DIET THERAPY 2

16.1 Introduction of the Unit of Learning / Unit of Competency

This unit addresses the unit of competency: provide diet therapy in diseases states involving CVDs; Atherosclerosis, stroke, hypertension, myocardial infarction, angina pectoris, and deep vein thrombosis. Renal disorders; glomerulonephritis, nephrotic syndrome, kidney stones AKD, CKD, kidney failure. Liver and gallbladder disorders; jaundice, liver encephalopathy, hepatitis, alcoholic liver disease, liver cirrhosis, gallbladder disease, disease of the pancreas. Metabolic disorders; diabetes mellitus, hyperthyroidism, hypothyroidism, hyperkalaemia, ketoacidosis. Mental and mood disorders; schizophrenia, bipolar, depression anxiety disorders, posttraumatic stress (PTSD) disorders and degenerative disorders.

16.2 Performance Standard

By the end of this unit of learning/competency, the trainee should be able to provide nutritional management of cardiovascular disorders in accordance with policies and guidelines and resource materials; provide nutritional management of renal disorders in line with resource materials, and policies and guidelines; provide nutritional management of metabolic disorders as per resource materials and policies and guidelines; provide nutritional management of mental and mood disorders in line with guidelines and policies and material resources; and provide nutritional management of degenerative disorders based on client's diagnosis, resource materials, and policies & guidelines.

16.3 Learning Outcomes

16.3.1 Learning Outcomes

1. Identify terminologies in diet therapy II
2. Demonstrate understanding in nutrition management of CVDs disorders
3. Demonstrate understanding in nutrition management of the renal disorders
4. Demonstrate understanding in nutritional management of liver and gallbladder disorders
5. Demonstrate understanding in nutritional management of metabolic disorders
6. Demonstrate understanding in nutritional management of mental and mood disorders
7. Demonstrate understanding in nutritional management of degenerative disorders

16.3.2 Learning Outcome 1: Identify terminologies in diet therapy II

16.3.2.2 Learning Activities

Learning Activities	Special instructions
1. Identify and describe terminologies under diet therapy II	• Use terminologies related to Diet Therapy 2
2. Illustrate the relationship between nutrition and disease and the roles of nutrition in disease management	• Consider the relationship between nutrition and disease • Consider how disease affects nutrition
3. Identify and describe the objectives of diet therapy II	• Formulate objectives of diet therapy

16.3.2.2 Information Sheet

Definitions

Diet Therapy: Practical application of nutrition in promotion of health and prevention of diseases

Malnutrition: A physiological state that results from inadequate, excess or imbalanced intake of nutrients.

Disease: A condition that impairs normal function of body or its parts , whch is manifested by signs and symptoms

Therapeutic diets: It is a diet that is modified from a normal diet to meet the requirements of the ill/ sick individual.

Normal diet: It consists of any and all foods eaten by the person in health. It satisfies the nutritional needs of most patients and serves as the basis for planning modified diets.

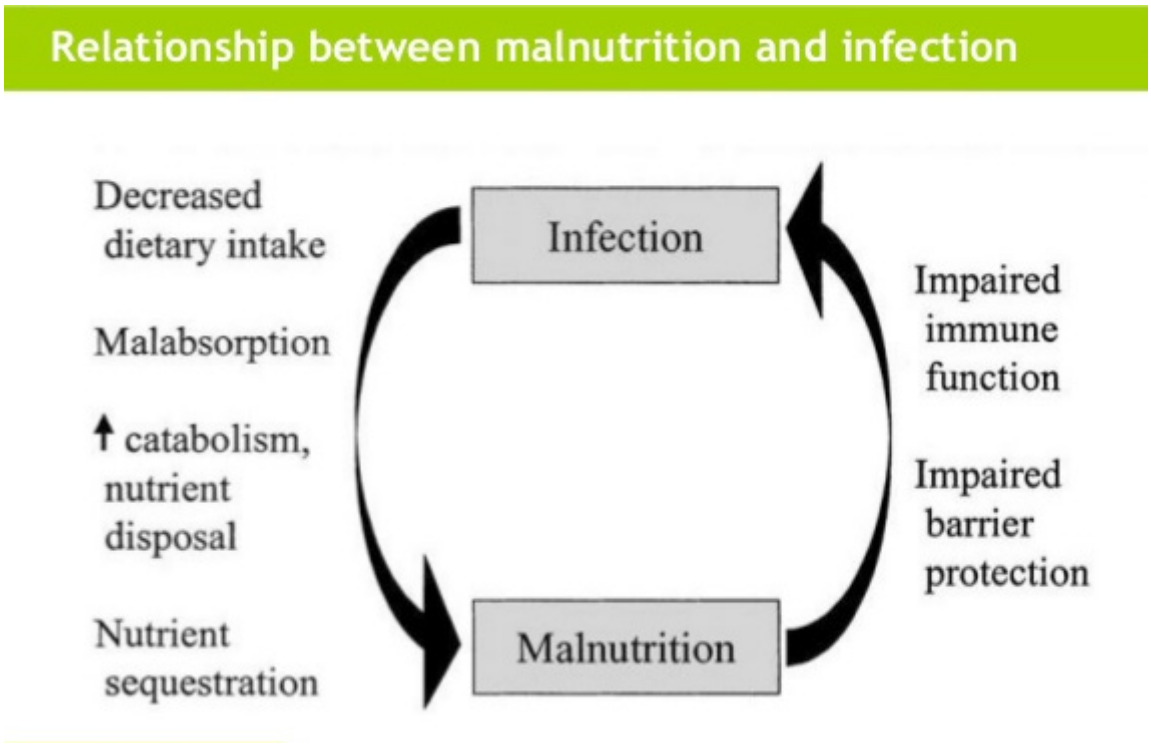
Diet modification: It refers to the action of adjusting a normal diet to change its consistency/ texture, flavor and nutrient contents.

Relationship between Nutrition and Disease

Food provides required raw materials for normal cellular activity. Nutrient deficiency impairs this activity and results to disease. Deficiency may be as a result of inadequate nutrient intake, malabsorption, increased requirement or increased excretion/nutrient loss.

Nutrients have nourishment and pharmacologic function and so they are directly involved with disease prevention. Examples:

- Zinc, Vitamin A, Vitamin B6 and folate support immune function
- Vitamin C, Vitamin E, selenium and carotenoids are antioxidants
- Iron, Vitamin A, zinc and Vitamin Care involved in tissue synthesis.



Relationship Between Malnutrition and Disease

The Goals of Nutrition in Disease Prevention

1. To optimize cellular activity and tissue/organ function:
    a) Provide sufficient amounts to satisfy daily demands of adequacy, balance and variety in food choices
    b) Maintain adequate reserves for intermittent increased demand through habitual diet and dietary patterns
2. To reduce the metabolic burden imposed on cardiac, pulmonary, renal, hepatic, musculoskeletal systems by environmental factors
    a) Minimize workload of organ systems by reducing stress on organs involved in transport, metabolism and elimination of nutrients and metabolic waste.
    b) Eliminate compensatory responses required to maintain normal function
3. To support cellular defenses that protects tissue integrity
    a) Maintain immune system competence
    b) Promote efficiency of detoxification systems by controlling levels of reactive chemical intermediates
4. Prevent oxidative damage that is involved in pathogenesis of most chronic diseases and reduction of efficiency of immune cells.

Role of Nutrition in Disease Management

Nutrition plays a key role in maintaining health and wellbeing. Adequate nutrition is needed in every step of the lifespan. Food is needed as a source of energy and various nutrients that support all body functions. Failure to meet nutrient and energy needs leads to nutrient deficiency disorders and increases susceptibility to disease.

Adequate nutrition is also needed in disease management. Disease affects nutrition in that it:

Ways Disease May Affect Nutrition:

- Affects food intake
- Affects digestion and absorption
- Affects nutrient utilization
- May increase nutrient excretion
- May affect nutrient storage
- Can increase nutrient and energy requirements
- Drug-nutrient interactio

Nutrients may also affect the disease progression in that food intake may affect drug absorption and utilization.

It is therefore imperative to plan a patient's diet with consideration of how the disease and nutrition interact. Nutrition should complement medical care to help enhance the recovery process.

16.3.2.3 Self-Assessment

1. Explain the goal of nutrition in disease prevention
2. Outline ways in which a disease may affect nutrition
3. Diet Therapy is:
    A. A condition that impairs normal function of body or its parts , whch is manifested by signs and symptoms
    B. A physiological state that results from inadequate, excess or imbalanced intake of nutrients.
    C. Practical application of nutrition in promotion of health and prevention of diseases
    D. The action of adjusting a normal diet to change its consistency/texture, flavor and nutrient contents.

4. A therapeutic diet is**:**
   A. A diet containing all nutrients in adequate amounts
   B. A diet that is modified from a normal diet to meet the requirements of the ill/ sick individual.
   C. Food prepared in hospital
   D. A diet rich in energy and proteins
5. The following nutrients are antioxidants except:
   A. Vitamin C
   B. Vitamin E
   C. Selenium
   D. Iron
6. Failure to meet nutrient and energy needs leads to nutrient :
   A. Weight gain
   B. Deficiency disorders and increases susceptibility to disease
   C. Growth and development
   D. Disease prevention

16.3.2.4 Tools, Equipment, Supplies and Materials

- Stationery
- WHO guidelines
- MOH
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise
- Computers with internet
- Library and resource centre

16.3.2.5 References

Anyang'Nyong'o, H. P. P., & EGH, M. Kenya National Clinical Nutrition And Dietetics Reference Manual First Edition.

Merritt, R., DeLegge, M. H., Holcombe, B., Mueller, C., Ochoa, J., & Smith, K. R. (2005). The ASPEN nutrition support practice manual.

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16.3.3 Learning Outcome 2: Demonstrate understanding in nutrition management of CVDs disorders

16.3.3.1 Learning Activities

Learning Activities	Special instructions
1. Identify and describe terminologies under CVDs disorders described	• Use terminologies under CVDs
2. Identify CVDs and discuss their pathophysiology	• Apply knowledge of common CVDs • Determine the nutritional factors associated with cardiovascular diseases • Consider the pathophysiology of CVDs
3. Identify and describe nutritional management of CVDs	• Determine the nutritional requirements in CVDs • Plan diet for persons suffering from CVDs • Consider nutrient interaction with drugs used in the management of common CVDs

16.3.3.2 Information Sheet

Definitions

Car**diovascular disease:** a class of diseases that affect the heart and blood vessels (arteries and veins). In the majority of cases, this is due to the progressive effects of atherosclerosis in the arteries.

Common examples of cardiovascular disease include:

- Coronary artery disease
- Stroke
- Congestive heart failure
- Hypertension
- Myocardial Infarction.

General risk factors for cardiovascular diseases

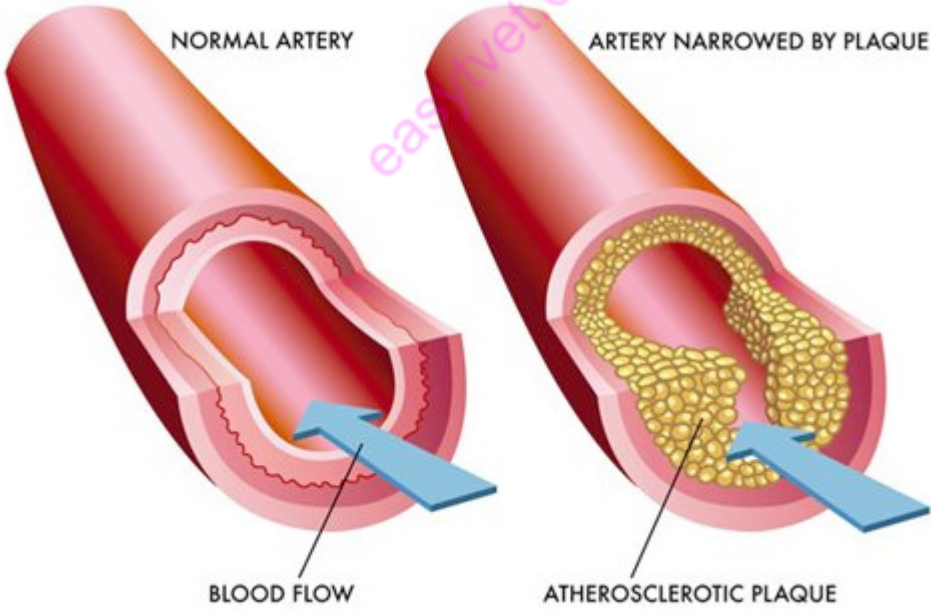
1. Non modifiable factors:
    - Age
    - Gender
    - Genetic factors
    - Race.

2. **Modifiable Risk Factors:**
    - High salt (sodium chloride) intake
    - High blood cholesterol
    - Too much fat, saturated fatty acid, Trans -unsaturated fatty acids
    - High levels of low density lipoprotein (LDL) cholesterol
    - Smoking cigarettes
    - Alcohol
    - Obesity
    - Reduced physical activity
    - Stress

Common Cardiovascular Diseases

1. ATHEROSCLEROSIS

This is a generative process that begins with the accumulation of soft fatty streaks along the inner arterial walls especially at the branch points. These streaks gradually enlarge and become hardened with minerals forming plaques.



Atherosclerosis

Risk Factors

- High calorie intake
- High saturated fat and cholesterol intake
- Increased serum LDL (harmful cholesterol) levels above 5mmol/liter

- Sedentary lifestyles
- Hypertension
- Diabetes.
- Stress
- Obesity

Implications

- Obstruction of normal blood flow
- Tissue damage
- Increased blood pressure

Aims of management

- To normalize blood lipids
- Control the modifiable risk factors and prevent complications.

Nutritional Management

- Reduce total fat intake(15-20%) -LDL–(saturated fat to 7% and dietary cholesterol 200mg/day)
- Reduce body weight for the overweight clients to the ideal body weight
- Avoid smoking and alcohol
- Physical activity is recommended.

2. MYOCARDIAL INFARCTION

This is sudden tissue death caused by interruption of blood flow in vessels that feed the heart muscle, also called heart attack/cardiac arrest. Risk factors include hypertension and arteriosclerosis. Other contributors include abnormal blood clotting, spasms of the coronary artery, rheumatic heart disease, infections of the membranes covering the heart and electrical disturbances that alter the hearty rate.

Implications

- Strained cardiac function

Aims of nutritional management

- To reduce the work load of the heart
- To relieve pain and stabilize the heart rhythm
- To treat infections and the underlying causes
- To regulate electrolyte balance

Nutritional Management

- When the patient is in shock, withhold food intake-nil per oral until shock resolves
- When shock resolves provide between 1000-1200 kcal that progresses from low sodium soft foods of moderate temperature in frequent and small feeding.
- After recovery adjust the diet to meet the individual needs and to deal with the underlying conditions such as hyper lipidemia, hypertension, and obesity
- Avoid caffeine as it stimulates metabolic rate and increase the workload of the heart
- A healthy heart requires minimal consumption of saturated fats and cholesterol, reduced use of salt and sugar, avoiding use of tobacco and too much coffee and regular exercise.

3. CONGESTIVE HEART FAILURE

This is a syndrome in which the heart can no longer adequately pump blood through the circulatory system. Risk factors include; uncontrolled atherosclerosis and hypertension.

Implications

- Pulmonary oedema
- Reduced blood flow to all organs
- Fluid retention hence stagnation of fluids in all organs
- Enlarged heart and rapid heart beat
- Malnutrition due to high energy needs

Aim of Management

- To reduce the workload of the heart
- To provide adequate nutrients
- To reduce weight for the overweight

Nutritional Management

- Restrict sodium, caffeine and fat intake
- Encourage gradual weight loss where necessary
- Use of liquid formula of high nutrient density as oral supplement or enteral or tube feeding to prevent or reverse malnutrition is recommended. In some cases total parenteral nutrition may be required
- Selection of enteral or parenteral formulas should be done carefully to ensure that energy ,fluid, sodium intake will not overload the body
- Adjust dietary fiber to avoid constipation but avoid amounts and types that produce gas and abdominal distention
- For overweight patient counsel on weight reduction
- Restrict cholesterol intake to 300 mg /day

- Reduce intake of saturated fats
- Encourage intake of unsaturated fats (oils)
- Increase intake of dietary fiber to control glucose/fat absorption
- Reduce alcohol intake and encourage the patient to avoid smoking to prevent development of atherosclerosis

4. HYPERTENSION

Hypertension is a cardiovascular disorder characterised by persistently elevated diastolic blood pressure (BP) of above 95mmHg. Uncontrolled hypertension can affect various body organs and can lead to impaired vision, kidney failure, stroke, paralysis, heart attack and brain damage. Risk factors include; diet, race, stress, age, diabetes, obesity, smoking, atherosclerosis and heredity among others.

Implications

- Strained cardiac and vascular function
- Cellular electrolyte imbalance
- Aneurysms (balloon out and busting of the arteries)
- Arterial lining injuries which accelerates the plaque formation.

Aims of nutritional management

- To control blood pressure within the normal ranges
- To achieve a gradual weight loss in overweight and obese individuals and maintain their weight slightly below the normal
- To reduce sodium intake based on severity
- To maintain adequate nutrition
- Regulate fat intake.

Nutritional management

- Provide low calorie diet if the patient is overweight until ideal body weight is achieved
- Reduce fat intake. Encourage intake of unsaturated fats (oils). The poly unsaturated and monounsaturated fatty acids lower BP, the level of triglycerides and LDL cholesterol and consequently lead to increase in HDL cholesterol that carries cholesterol in the blood back to the liver for recycling or disposal. Fats should be 20% of total kilo calorie
- Avoid alcohol intake
- Restrict sodium intake. To achieve this, encourage a selection of food low in sodium. Besides, reduce the amount of salt added to food, avoid using spices containing sodium and processed food.
- Abstain from stimulants such as spirits and caffeine
- Avoid from cigarette smoking that may result in atherosclerosis

- It might be necessary to restrict fluid intake in some cases
- Encourage those leading a sedentary lifestyle to engage in physical activity.

5. STROKE/TRANSIENT ISCHEMIC ATTACK

This is reduction in blood flow to the brain that causes temporal symptoms which depend on which part of the brain is affected.

Risk factors

- Atherosclerosis
- Hypertension or a combination of the two

Implications

- Light headedness
- Paralysis
- Numbness
- Visual disturbances
- Staggering
- Dysphasia (inability to coordinate swallowing appropriately).

Aim of management

- To treat the underlying risk factors

Management

- Restricted energy intake, total fat and sodium
- Tube feeding may be indicated initially until the client is safely able to chew and swallow
- Some patients may need assisted feeding

16.3.3.3 Self-Assessment

1. Outline the risk factors for atherosclerosis
2. Discuss the nutritional management of hypertension
3. Discuss the aim of nutritional management of congestive heart failure
4. Which of the following factors is not a risk factor associated with Atherosclerosis?
   A. Hypertension
   B. Obesity
   C. Low calorie intake
   D. Stress

5. Which of the following is a non-modifiable factor for cardiovascular diseases?
   A. Genetic factor
   B. High blood cholesterol
   C. Smoking
   D. Physical inactivity
6. The following statements are true about the nutritional management of congestive heart failure except:
   A. For overweight patient counsel on weight reduction
   B. Restrict cholesterol intake to 300 mg /day
   C. Reduce intake of saturated fats
   D. Sodium restriction is not necessary

7. Which of the following statement is true about hypertension?
   A. It only affects overweight people
   B. It is a reduction in blood flow to the brain
   C. It results to cellular electrolyte imbalance
   D. Patients with hypertension should be encouraged to reduce sodium intake
8. Which one of the following is an objective of nutritional management for myocardial infarction:
   A. To increase sodium intake
   B. To reduce the work load of the heart
   C. To stabilize blood pressure
   D. To maintain normal blood glucose

16.3.3.4 Tools, Equipment, Supplies and Materials

- Stationery
- Clinical guidelines
- WHO guidelines
- MOH
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise

- Computers with internet
- Library and resource centre

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Anyang'Nyong'o, H. P. P., & EGH, M. Kenya National Clinical Nutrition And Dietetics Reference Manual First Edition.

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16.3.4 Learning Outcome 3: Demonstrate understanding in nutrition management of the renal disorders

16.3.4.1 Learning Activities

Learning Activities	Special instructions
1. Identify and describe terminologies under renal disorders	• Use terminologies in renal disorders
2. Identify renal disorders and discuss their pathophysiology	• Determine how renal disorders affect nutrition
3. Identify and describe nutritional management of renal disorders	• Determine the nutritional requirements for renal disorders • Plan diet for persons suffering from renal diseases • Consider nutrient-drug interactions in the management of renal disorders

16.3.4.2 Information Sheet

Functions of Kidney

- Excretes dissolved unwanted substances, filtered out of the blood as urine.
- Filtration: a filter through which all dissolved substances pass & those to be retained are selectively re-absorbed.
- Maintenance of fluid, electrolyte and acid base balance
- Helps to regulate the blood pressure through excretion of sodium.
- Produces erythropoietin (hormone) which stimulates the maturation of red blood cells in the bone marrow.
- Converting vitamin D to its most active form calcitriol.

Causes of Kidney Diseases

1) **Inflammation and degeneration:** the membranes and small blood vessel in the nephrons may get inflamed for due to infections and thus lead to glomerulonephritis.
2) **Chemical damage:** Environmental agents such as pesticides, solvents etc. may cause kidney damage.
3) **Infections and obstructions**
4) **Other diseases that damage the kidney function**: any disorder in circulation which results in degeneration of small renal arteries disturbs the normal nephron function e.g. poorly controlled hypertension, uncontrolled type 2 diabetes.

1. GLOMERULONEPHRITIS

This is inflammation of the glomeruli. The inflammatory process affects the glomeruli which is a trough of blood capillaries in the head of the nephron. It is most common in its acute form in children between the age 4-10years and young adults under the age of 30years.

Causes

The most common cause is streptococci infection. In the past, acute glomerulonephritis commonly frequently followed infection with:

i) Beta-haemolytic streptococci in children

ii) Tonsillitis in young adults with respiratory tract infection or pneumonia.

Symptoms

Typical symptoms are:

- Hematuria
- Proteinuria.
- As much as 50% or more reduced renal blood flow and glomerular filtrate rate ,
- Urine volume falls to between 500-1000ml per day and sodium excretion is greatly reduced.
- Because the patient may continue to ingest the normal quantities of sodium and water, edema develops and blood pressure rises leading to complains of malaise, headache, swelling of the face and hands.
- Urea and creatinine concentration in the plasma rises in proportion to the fall in glomerular filtration rate (GFR).
- Patient is generally anorexic; nausea and vomiting occur which contributes to feeding problems.
- If the disease progresses to renal insufficiency, oliguria and anuria occur which is signal for the development of acute renal failure
- Mild acidosis and hyperkalemia are usually present

Objectives of Dietary Management

- To spare the diseased kidney
- To prevent uremia
- To prevent edema
- To maintain adequate nutrition

Dietary Recommendations

Energy: Requirement is the same as in good health. In the absence of fever, previous malnutrition, with bed rest, allowances can be reduced.

Proteins: Restriction of protein is only needed when the BUN is elevated and oliguria is present.

Diet should provide about 0.5g/kg of ideal body weight. When protein is restricted in quantity, it is important to provide these reduced quantities in the form of protein of high biological value for the efficient utilization.

Carbohydrates: In order to provide enough energy, carbohydrate needs to be given freely.

Fats: There is no need to restrict fat in the diet. Include emulsified and easily digestible fat in the diet. It reduces the bulk of the diet, provides non-protein kcal for energy needs and makes the diet palatable

Sodium: Restrict sodium in the diet in the presence of oliguria, edema and hypertension. Restriction of sodium depends on the extent or degree of the symptoms.

Potassium: The renal clearance of potassium is impaired when severe oliguria is a complication. It may lead to potassium intoxication and even require dialysis (hyperkalemia can result in cardiac arrest). Foods that are good sources of potassium should be avoided; it can lead to potassium intoxication especially if oliguria is present.

Water: Water and other fluids need to be restricted according to the ability of the kidney to excrete urine. When oliguria is present, restriction of fluid intake is imposed. The volume of fluid to be given is calculated from the volume of urine passed in the previous 24 hours plus estimated insensible water loss, usually 500ml daily.

2. NEPHROTIC SYNDROME/ NEPHROSIS

Nephrosis is a kidney disorder that causes the body to excrete too much protein in the urine. It is caused by a variety of diseases that all lead to decreased kidney ability to prevent leakage of macromolecules, particularly proteins, into the filtrate. Normally, only a small amount of proteins is filtrated through glomerular and these are completely reabsorbed in the tubules. Proteinuria develops when the leakage of protein from the glomerular exceed the reabsorption capacity of the renal tubules.

Causes:

It may be caused by progressive glomerulonephritis. Besides, it may be in association with several factors that include:

- Diabetes
- Drug reaction
- Exposure to heavy metals
- Reaction to toxic venom.

Symptoms

- It is characterized by heavy proteinuria, hypoalbuminemia and edema. Massive edema leads to ascites. Edema is due to excessive loss of protein in the urine; about 4-10g/day or more.
- Plasma protein is reduced greatly and the albumin fraction responsible for the maintaining the fluid balance between tissue fluids and circulating fluid is decreased

Objectives:

- Correct and control protein deficiency
- Correct and prevent edema.
- To maintain adequate nutrition
- To avoid unnecessary harm to the kidney.

Dietary Modification

Energy

Sufficient energy must be provided to ensure efficient use of protein for tissue synthesis. High daily intake of 50-60 kcal/kg body weight is essential.

Protein

Replacement of the prolonged protein loss is a most immediate and fundamental need. Plasma albumin levels may have been reduced to 20% or less of its normal value, a major cause of the development of nephritic ascites and edema. A high protein intake is associated with a positive nitrogen balance to replenish the depleted stores and enhance hepatic synthesis of albumin. High protein intake of 90-120g for adults and 2-3g/kg of body weight for children is recommended. Besides, intake of protein foods of high biological value such as milk and its products, eggs, meat etc. should be encouraged.

Unfortunately, these foods are also moderately rich source of sodium, and so their intake cannot indiscriminately increase.

Carbohydrates

Sufficient non- protein calories need to be provided for sufficient utilization of protein and for body protein synthesis. A high carbohydrate intake is recommended for protein sparing action.

Fats

Restriction is not necessary unless there are comorbidities that demand so.

Sodium

Sodium intake in the diet needs to be reduced to combat edema. Diuretics are also used to prevent further fluid accumulation for patients in whom the renal tubes are responsive to diuretics. For these patients, extreme degree of salt restriction is not required and they may be given about 1g of sodium per day.

Potassium

Potassium restriction is not necessary for these patients because oliguria and anuria are not present.

Calcium

In some patients with prolonged proteinuria, deficiency of calcium and potassium may occur resulting in bone rarefaction and hypokalemia. Both these defects are corrected by high protein diet with added calcium and potassium.

3. ACUTE RENAL FAILURE (ARF)

It is the sudden stop of renal function as a result of metabolic injury to the normal kidney.

The functioning of the kidney can be completely restored as long as the patient is kept alive during the period when the excretory and homeostasis functions are impaired.

ARF is often a life threatening illness with high mortality rates.

Causes of ARF

- Loss of blood due to any cause including; complications in pregnancy, trauma or gastrointestinal bleeding
- Loss of plasma due to tissue destruction as seen in burns and crush injuries
- Loss of fluid from the gut as seen in severe vomiting, diarrhea etc.
- Excess loss of fluid from the skin as seen in excessive sweating.
- Nephrotoxins: e.g drugs such as paracetamol, industrial chemicals and poisonous mushrooms.
- General anesthetics and surgery: reduces renal blood flow especially in pts with low blood volume.
- Infections especially septicemia caused by E-coli which results in reduced renal blood flow.

NOTE

In most of the conditions, prolonged hypotension with systolic blood pressure less than 90 for one hour or more is a common causative feature.

Whenever there is excessive loss of blood, plasma or fluid, resulting in peripheral circulation failure, the kidney becomes susceptible to ischemic damage.

Phases of ARF

- Initial /Onset phase
- Diuretic phase
- Oliguric/Anuric Phase
- Recovery Phase

Four phases of AKI

This chart describes the features and durations of the four phases of acute kidney injury (AKI).

Phase	Features	Duration
Onset phase	• Common triggering events: significant blood loss, burns, fluid loss, diabetes insipidus • Renal blood flow 25% of normal • Tissue oxygenation 25% of normal • Urine output below 0.5 mL/kg/hour	Hours to days
Oliguric (anuric) phase	• Urine output below 400 mL/day, possibly as low as 100 mL/day • Increases in blood urea nitrogen (BUN) and creatinine levels • Electrolyte disturbances, acidosis, and fluid overload (from kidney's inability to excrete water)	8 to 14 days or longer, depending on nature of AKI and dialysis initiation
Diuretic phase	• Occurs when cause of AKI is corrected • Renal tubule scarring and edema • Increased glomerular filtration rate (GFR) • Daily urine output above 400 mL • Possible electrolyte depletion from excretion of more water and osmotic effects of high BUN	7 to 14 days
Recovery phase	• Decreased edema • Normalization of fluid and electrolyte balance • Return of GFR to 70% or 80% of normal	Several months to 1 year

Phases of Acute Renal Failure

Treatment

The objectives of the initial nutrition therapy are to support overall medical management.

This includes:-

- Re-establishing fluid and electrolyte balance
- Maintenance of adequate nutritional status
- Providing an optimal environment for wound healing
- Preventing infection

Dietary management of the oliguric phase

Fluid intake

The fluid allowance is usually regulated in accordance with the urinary output and any additional losses from vomiting or diarrhea.

Its intake is usually restricted to a basic allowance of 500ml/day for an average adult with additions made for losses via other routes

Electrolyte

- Provided only to replace losses.
- Potassium allowance is individualized in accordance with serum levels.
- Since serum potassium levels are usually elevated due to massive tissue destruction, potassium administration should not be done.
- Sodium allowance is based on its concentration in serum and urine
- Restriction of sodium to 500-1000mg daily is usually necessary during the oliguric phase.

Protein

- During the oliguric and anuric phase, protein metabolism should be reduced to a minimum.
- Initially during the acute phase no protein should be given to the patient.
- As the condition improves only 20g protein daily should be given which is the minimum amount required to compensate for endogenous losses.
- The protein should be of high biological value

Energy

- Non protein sources of energy such as carbohydrate should be included
- A daily intake of 100-200g or more of sugar has a marked protein sparing effect.
- Additional energy may be obtained from fat also.

Dietary management of diuretic phase

This stage indicates a return of renal functions to normal.

Dietary treatment is still very important although the problems are reversed.

- Pt is passing a large volume of urine and so is at risk of excessive loss of water, sodium, potassium and calcium, bicarbonate, phosphate and magnesium.
- Proteins should continue to be restricted until blood urea nitrogen and serum creatinine levels return to normal.

- A normal diet with free fluid intake is prescribed and may even need to be supplemented with electrolytes.
- The fluid and electrolyte status of the patient needs to be monitored by daily weighing and blood and urine analysis.

4. CHRONIC RENAL FAILURE

Also called Chronic Kidney Disease (CKD)

Causes a progressive reduction in renal function that results in a reduced ability to control body water volume, acid-base balance, hormonal regulation, and electrolyte concentrations.

Causes

- Arises from any disease processes that compromises the renal blood perfusion
- Renal diseases; e.g. chronic glomerulonephritis, chronic urinary obstruction
- Systemic diseases, such as diabetes mellitus hypertension; infections;
- Medications or Environmental/ toxic agents- e.g. lead, mercury .

Pathophysiology

- There is decline of renal function as GFR falls.
- The serum levels of urea nitrogen & creatinine increases.
- Uremia develops and adversely affects every system in the body
- When GFR is less than 10– 20mls/min, the effect of uremic toxins on the body becomes evident.
- The greater the build up of waste products, the more severe the symptoms.

Stages of Chronic Renal Failure

Stage 1: Reduced renal reserve

Characterized by a 40% to 75% loss of nephron function.

There are No symptoms because the remaining nephrons are able to carry out the normal functions of the kidney.

Stage 2: Renal insufficiency

- Occurs when 75% to 90% of nephron function is lost.
- Increase in Serum creatinine and BUN
- Kidney loses its ability to concentrate urine

Stage 3: End-Stage Renal Disease (ESRD)

- The final stage of chronic renal failure,
- Occurs when there is less than 10% nephron function remaining.
- All of the normal regulatory, excretory, and hormonal functions of the kidney are severely impaired
- ESRD is evidenced by elevated creatinine and blood urea nitrogen levels as well as electrolyte imbalances.
- At this point, dialysis is indicated
- Many of the symptoms of uremia are reversible with dialysis

NOTE

The severity of the signs and symptoms depends on:-

- The degree of renal impairment
- Other underlying conditions
- The patient's age.

Signs & Symptoms

Cardiovascular manifestations

- Hypertension
- Heart failure and pulmonary edema (due to fluid overload)
- Pericarditis (due to irritation of the pericardial lining by uremic toxins)
- Pitting edema
- Hyperkalemia

Dermatologic symptoms

- Severe itching (pruritus) is common
- Thin, brittle nails; as nitrogen waste products build up
- Uremic frost, the deposit of urea crystals on the skin (uncommon today because of early and aggressive treatment of ESRD with dialysis)

Neurologic symptoms

- Weakness and fatigue
- Inability to concentrate;
- Tremors
- Restlessness
- Confusion
- Disorientation
- Seizures
- Behavior changes

Hematologic symptoms

- Anemia (diseased kidneys do not make enough erythropoietin)

Renal symptoms

- Anuria (output < 50 mL/day)

Psychological symptoms

- Depression
- Anxiety

Gastrointestinal symptoms

- Ammonia odor in breath
- Mouth ulcerations and bleeding,
- Nausea and vomiting
- Bleeding from gastrointestinal tract
- Metallic taste
- Anorexia
- Constipation or diarrhea

Musculoskeletal symptoms

- Muscle cramps
- Loss of muscle strength
- Renal osteodystrophy (alteration of bone morphology when kidneys fail to maintain proper levels of calcium and phosphorus in the blood)

Management

The goal of management is to maintain kidney function and homeostasis for as long as possible

Management is accomplished primarily with:

- Medication
- Dialysis
- Diet therapy
- Transplantation

Dialysis

Process of artificial removal of uremic waste products & excess water in the body.

Used to treat patients with:

- Edema
- Hypercalcemia
- Uremia &
- Hyperkalemia
- Hypertension
- Not responding to other treatments

Principles of dialysis

Diffusion

Toxins and wastes in the blood are removed by diffusion from an area of higher concentration in the blood to an area of lower concentration in the dialysate

Osmosis

Movement of a solvent such as water across a semi permeable membrane from areas of less solute to areas of high concentration of solute.

Ultrafiltration

- Movement of a fluid across a semi permeable membrane from a high pressure area to a low pressure area.
- It is more efficient in removal of water than osmosis.

Types of Dialysis

- Hemodialysis
- Peritoneal Dialysis
- Hemodialysis

Hemodialysis

A dialysis machine and a special filter called an artificial kidney, or a dialyzer, are used to clean the blood.

To get pt's blood into the dialyzer, the doctor needs to make an access, or entrance, into the blood vessels, done with minor surgery, usually to the arm.

The dialyzer, or filter, has two parts, one for pt's blood and one for a washing fluid called dialysate. A thin membrane separates these two parts.

Blood cells, protein and other important things remain in the blood because they are too big to pass through the membrane. Smaller waste products in the blood, such as urea, creatinine, potassium and extra fluid pass through the membrane and are washed away.

Complications of hemodialysis:

- Disturbance of lipid metabolism
- Hypertriglyceridemia
- Gastric ulcers & other GI problems - resulting from the physiologic stress of chronic illness & medication.
- Disturbed calcium metabolism –leads to bone pain & fractures (renal osteodystrophy).
- Sleep disturbance

- Painful muscle cramping due to rapid fluid shift from the extravascular space.
- Hypotension:-if too much fluid is eliminated
- Blood loss:-if blood lines separate or dialysis needles dislodge.
- Dialysis Disequilibrium Syndrome (rare):- due to rapid fluid shift from the cerebral fluid; characterized by headache, nausea, vomiting, restlessness, decreased level of consciousness & seizures.
- Arrhythmias (heart beats with an irregular or abnormal rhythm):-due to electrolyte & pH change.

Peritoneal dialysis (PD)

PD is performed by surgically placing a special, soft, catheter into the lower abdomen.

Dialysate is instilled into the peritoneal cavity and is left in for a designated period of time (dwell time) which will be determined by the nephrologist

The dialysate fluid absorbs the waste products and toxins through the peritoneum which acts as the semipermiable membrane

The fluid is then drained from the abdomen, measured, and discarded.

Indicated for patients who are unable or unwilling to undergo hemodialysis or kidney transplant

In peritoneal dialysis, there is no machine.

Instead of an artificial filter, the lining of the abdomen, the peritoneum is used as a natural filter.

The peritoneum has a lot of small vessels in it.

With peritoneal dialysis, it takes 36 – 48 hours to achieve what hemodialysis accomplishes in 6 –8 hours.

Peritoneal dialysis uses the principle of diffusion and osmosis

Types of peritoneal dialysis:

a) **Continuous Ambulatory Peritoneal Dialysis (CAPD):**

   The peritoneum is filled with dialysate, which remains there for a prescribed dwell time, then the fluid is drained. Gravity moves the fluid through the catheter and into, and out of the abdomen.

   Pt needs 3-5 exchanges during the day and one with a longer dwell time while asleep

   Pt. can do the exchanges at home, work or any clean place.

   Procedure allows the pt. reasonable freedom and control of daily activities while the dialysate dwells in their abdomen.

b) Continuous Cyclic Peritoneal Dialysis (CCPD):

Also known as Automated Peritoneal Dialysis (APD).

Method uses a machine (automated cycler) that performs multiple exchanges at night while pt. is asleep.

The cycler automatically fills the abdomen with dialysate, allows it to dwell there and then drains it to a sterile bag that emptied in the morning.

P.t must remain attached to the machine for 10 to 12 hours at night.P.t isn't connected to the machine during the day.

They might have a lower risk of peritonitis because they connect and disconnect to the dialysis equipment less frequently than with CAPD.

Nutrition concerns in peritoneal dialysis

- Patients who receive peritoneal dialysis may develop hypokalemia, since commercially available solutions do not contain potassium.
- Potassium can be liberalized in the diet or supplemented orally if needed.
- The peritoneal dialysate can provide a substantial amount of energy from glucose to the patient when hypertonic solutions are needed for increased fluid removal.
- Diabetic patients may have a greater risk for hyperglycemia, and all patients can develop hypertriglyceridemia.
- The nutritional intake of patients who receive peritoneal dialysis may be affected by bloating, abdominal fullness, and loss of appetite due to the indwelling dialysate .
- The protein needs of patients who receive peritoneal dialysis are increased, and it is important to encourage a high-protein diet to minimize the risks of malnutrition and infection.
- Some patients may require protein or protein-energy supplementation to meet their daily estimated protein needs of 1.2 to 1.3 g/kg.

5. TRANSPLANTATION

- A surgical procedure performed to replace a diseased kidney with a healthy kidney.
- It involves the surgical attachment of a functioning kidney, or graft, from a donor to a patient with end-stage renal disease (ESRD).

Sources of kidney

- Deceased-donor kidneys
- Living transplant /Live donor kidneys.

Goals for Dietary Management of CKD

- To minimize uremic toxicity
- To prevent wasting and malnutrition

Nutrition Management of CKD

Energy

The energy requirements of CKD patients who do not receive dialysis are similar to the requirements of healthy individuals and are influenced by age, sex, and physical activity level

When prescribing energy requirements for persons with CKD, the primary goals should be to provide an adequate amount of total energy to maintain or achieve a reasonable body weight and positive nitrogen balance.

Hemodialysis:

For patients 60 years and older with stage 3 disease who receive dialysis, an energy intake of 30 to 35 kcal/kg body weight. For younger patients, energy needs should be calculated at a minimum of 35 kcal/kg bd wt.

Peritoneal dialysis:

- In peritoneal dialysis, glucose is absorbed from the dialysate. Therefore, the dietary energy intake may need to be decreased to prevent excess weight gain and obesity
- Energy absorbed from the dialysate should be subtracted from the daily energy intake from the diet.

Protein

Protein intake is based on the patient's creatinine clearance, estimated GFR, and urinary protein losses.

Hemodialysis:

- For patients who receive hemodialysis three times per week : at least 1.2 g/kg bd wt/ day.
- A patient loses 10 to 13 g of amino acids and small peptides during a single hemodialysis treatment.
- Approximately 30% to 40% of the amino acids lost during hemodialysis are essential. Therefore, high–biological value protein should provide at least 50% of the total protein in the diet

Peritoneal dialysis:

- The protein recommendations is 1.2 to 1.3 g/kg of bd wt.
- Protein requirements may be even higher, depending on the patient's stress level or metabolic needs.

- When used for long-term management of CKD, peritoneal dialysis is associated with progressive wasting and malnutrition

Kidney transplant:

For adult kidney transplant recipients who have recovered from surgery and have an adequately functioning allograft, a daily protein intake of 0.8 to 1.0 g/kg of body weight is recommended.

Consider the medical status of each patient, addressing individual issues as needed.

Adequate but not excessive protein intake supports allograft survival and minimizes the impact on comorbid conditions.

Sodium and Fluid

Hemodialysis:

- The daily sodium allowance is 2 g/day with adjustments based on urine output.
- The more urine that the patient produces, the more sodium the patient may eliminate via the urine.
- If the patient is anuric, 1,000 to 1,500 mL/day of fluid is recommended.

Peritoneal dialysis:

The sodium intake for most patients should be 2 g/day.

The suggested fluid intake 2,000 mL/day

Potassium

Hemodialysis:

- A potassium intake of 40 mg/kg of standard body weight is recommended.
- Removes potassium; therefore, monitoring potassium levels and ensuring adequate intake is important .
- Adjustments in potassium intake (either from the diet or from the dialysate bath) can be made to achieve target potassium levels

Peritoneal dialysis:

- Patients who receive peritoneal dialysis may not need potassium restrictions; however, an assessment should be based on the patient's laboratory values.
- Peritoneal dialysis can increase the risk for hypokalemia, since most commercially available solutions do not contain potassium.
- Oral supplementation and/or dietary intake can be adjusted to compensate for low potassium levels. A target intake of 3 to 4 g/day of potassium is suggested.

16.3.4.3 Self-Assessment

1. Outline the causes of kidney diseases
2. Explain the objective of nutritional management in nephrotic syndrome
3. Discuss the phases of acute renal failure
4. Explain the stages of chronic renal failure
5. Outline the nutrition concerns in peritoneal dialysis
6. Uremia is:
   A. Reduced urine output
   B. Accumulation of urea in blood
   C. Fluid retention due to kidney failure
   D. Sudden failure of the kidneys due to injury
7. Which one the following statements is true about the diuretic phase of acute renal failure:
   A. Patient passes very little volume of urine
   B. BUN is usually high
   C. A normal diet with free fluid intake is prescribed and may even need to be supplemented with electrolytes.
   D. Creatinine level is abnormally high
8. Which one of the following is not a function of the kidney:
   A. Maintenance of fluid, electrolyte and acid base balance; carry out selective filtration.
   B. Helps to regulate the blood pressure through excretion of sodium.
   C. Produces erythropoietin (hormone) which stimulates the maturation of red blood cells in the bone marrow.
   D. Produces bile
9. Which of the following is a cardiovascular implication of chronic renal failure:
   A. Severe itching (pruritus) is common
   B. Thin, brittle nails; as nitrogen waste products build up
   C. Hypertension
   D. Confusion
10. The following statements are true about nutritional management in peritoneal dialysis except:
   A. Patients may develop hypokalemia
   B. Potassium intake should be restricted

C. The peritoneal dialysate can provide a substantial amount of energy from glucose to the patient when hypertonic solutions are needed for increased fluid removal.

D. Diabetic patients may have a greater risk for hyperglycemia, and all patients can develop hypertriglyceridemia.

16.3.4.4 Tools, Equipment, Supplies and Materials

- Stationery
- Reference materials
- Clinical guidelines
- WHO guidelines
- MOH guidelines
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise
- Computers with internet
- Library and resource centre

16.3.4.5 References

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16.3.5 Learning Outcome 4: Demonstrate understanding in nutritional management of liver and gallbladder disorders

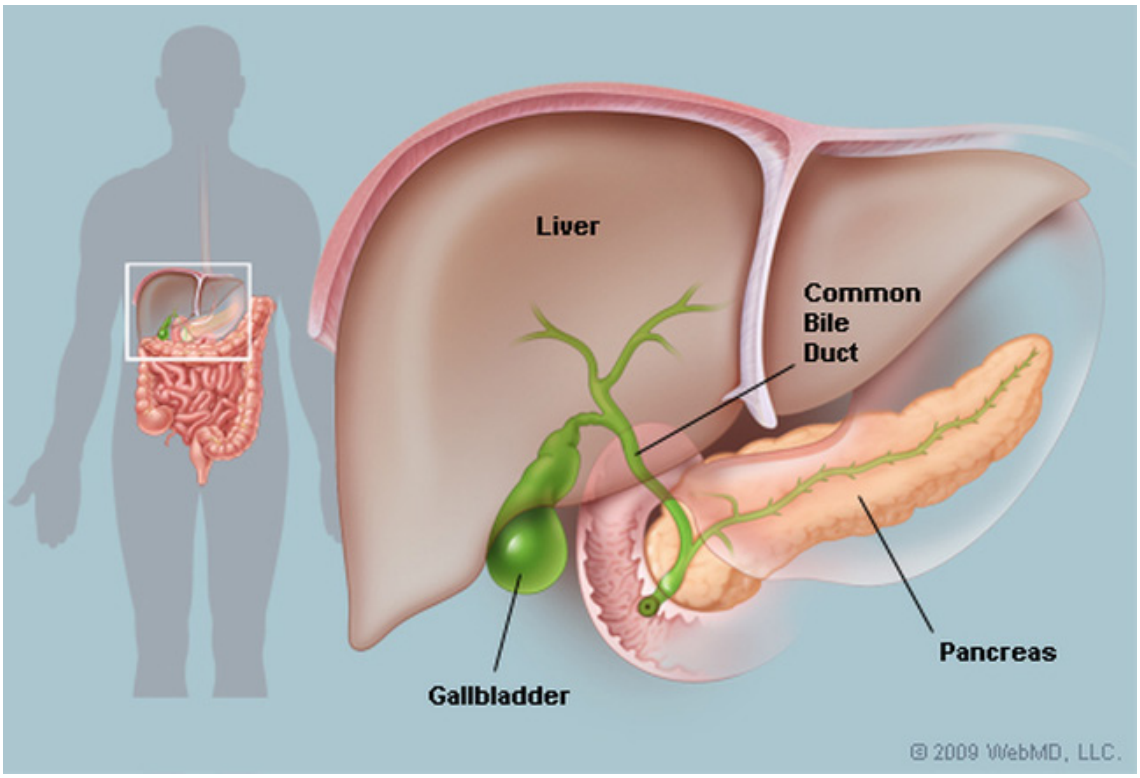
16.3.5.1 Learning Activities

Learning Activities	Special instructions
1. Identify and describe terminologies under liver and gallbladder disorders	• Use terminologies in diseases of the liver and gall bladder
2. Identify liver and gallbladder disorders and discuss their pathophysiology	• Consider how liver and gall bladder diseases affect nutrition
3. Identify and describe Nutritional management of liver and gallbladder disorders	• Plan diet for patients suffering from liver and gall bladder disorders • Determine nutritional requirements of patients suffering from liver and gall bladder disorders • Consider drug-nutrient interactions in management of diseases of the liver and the gall bladder • Apply guidelines in the management of diseases of the liver and the gall bladder

16.3.5.2 Information Sheet

Definitions

- **Atrophy** :the degeneration of hepatic cells
- **Fatty infiltration**: deposition of fat droplets in the hepatic cells.
- **Fibrosis**: formation of an abnormal amount of fibrous tissue as the result of inflammation, irritation, or healing; where the functioning hepatic cells are completely replaced by connective tissue cells.
- **Necrosis:** death of the hepatic cells.



The liver and gall bladder

Functions of the liver

- Fat metabolism.
- Vitamin and minerals storage.
- Bile formation
- Bilirubin excretion.
- Metabolism of glucose and regulation of blood glucose concentration. (Glycogenesis & Glycogenolysis)
- Converts metabolically generated ammonia into urea.
- Plays an important role in protein metabolism. Synthesizes almost all of the plasma protein e.g albumin, globulins e.t.c
- Activation of enzymes

1. HEPATITIS

An infectious disease characterized by inflammation and degeneration of liver cells.

Types of Hepatitis

Type	Virus	Mode of Transmission
Hepatitis A	hepatitis A virus (HAV).	Consuming food or water contaminated by feces from a person infected with hepatitis A.
Hepatitis B	Hepatitis B virus (HBV)	Contact with infectious body fluids, such as blood, vaginal secretions, or semen, containing the hepatitis B virus (HBV)
Hepatitis C	Hepatitis C (HCV)	Direct contact with infected body fluids, typically through injection drug use and sexual contact
Hepatitis D	Hepatitis D (HDV)	Direct contact with infected blood. A rare form of hepatitis that only occurs in conjunction with hepatitis B infection.; Can't multiply without the presence of hepatitis B.
Hepatitis E	Hepatitis E (HEV)	Waterborne disease. Mainly found in areas with poor sanitation and typically results from ingesting fecal matter that contaminates the water supply.

Symptoms of Hepatitis

Initially, non-specific symptoms occur :

- Anorexia,
- Nausea,
- Diarrhea,
- Abdominal discomfort
- Fatigue,
- Vomiting,
- Fever

Followed by :

- Jaundice
- Enlargement and tenderness of the liver.
- Patients may note that their urine darkens and their stools lighten in color.

Dietary Management

Objectives of dietary therapy are:

- To relieve symptoms
- To aid in the regeneration of liver tissue
- To prevent further liver damage.

Recommended Dietary Modifications

Energy

- A high energy intake to promote weight gain & ensure maximum protein utilization.
- Initially pt may not be able to eat such a large amounts of food due to anorexia and only 1500-2000 Kcal may be acceptable.
- Gradually, the energy intake may be increased to 20-30% more than the normal intake.
- If p.t is advised to have bed rest, the recommended intake of energy under normal conditions will suffice

Protein

Intake needs to be increased to:

- Overcome the negative nitrogen balance,
- Promote regeneration of the liver cells
- To prevent fatty infiltration of the liver.

However, the damaged liver may not be able to tolerate the high protein load because the conversion of ammonia to urea gets affected and there exist a danger of impending hepatic coma.

Therefore depending on the extent of liver damage; there is a need to adjust the protein intake accordingly.

In mild to moderate cases:1.5 to 2g/kg/bwt

On the other hand in acute cases with extensive liver damage, the protein intake may have to be decreased even below normal.

It is important to ensure protein of high biological value to ensure their maximum utilization, preferably supplemented with proteins of vegetable origin.

Carbohydrates

A high cbh diet is recommended to:

- Provide the bulk of energy,
- To build up glycogen stores in the liver
- For their protein sparing action.

A daily intake of 300 to 400 grams of cbh is recommended.

Fats

Decrease fat intake though not severely; digestion and absorption of fat is affected because of impaired bile secretion.

- Inclusion of moderate amounts of fat in the diet not only increases palatability of food but also promotes recovery.
- Tolerance of fat may vary from person to person.
- Mild to moderate cases: 40 to 50 grams total fat/day
- Severe cases accompanied by liver damage: restricted to 20-30grams per day.

Vitamins

The availability of fat soluble vitamins like vitamin A tends to be low due to the decreased intake and impaired fat absorption.

- For Vit.A, ensure inclusion of carotene rich foods like deep yellow and orange vegetables and fruits in the diet.
- Prothrombin time is invariably increased; may be corrected by giving vitamin K.
- B Vits: Needs are increased due to increased energy metabolism
- Higher amounts of vitamin C are needed for tissue healing.

Minerals

Diet should provide all minerals, particularly iron in adequate amounts in view of the increased tissue catabolism.

2. LIVER CIRRHOSIS

It is a chronic disease of the liver in which fibrous connective tissue replaces the functioning hepatic cells.

It characterized by destruction of liver cells, distortion of normal lobules with growth of fibrous tissues and nodular regeneration of cell following fatty degeneration.

The cirrhotic liver is contracted and has lost most of its functions.

Causes

- **Chronic alcoholism:** Alcohol & its metabolic products disturb liver metabolism and damage liver cells directly.
- **Chronic alcoholism and malnutrition:** Chronic alcoholics have a long standing inadequate food intake leading to malnutrition and necrosis of liver cells and subsequently cirrhosis.
- **Gross dietary inadequacy, especially of protein**. When there is a nutritional deficiency, the liver is more vulnerable to injury from various toxic agents.
- **Various infective and toxic agents:** As seen in infectious hepatitis, destruction of liver cells. This may lead to fibrotic changes in the liver and finally cirrhosis.
- Obstructions in the bile duct.

- Idiopathic cirrhosis: Liver cirrhosis from undetermined causes is also sometimes observed.

Symptoms

Onset of the disease is gradual with initial symptoms of GI disturbances such as:

- Nausea
- Vomiting
- Anorexia
- Distension and epigastric pain.
- Jaundice appears, with increasing weakness, ascites, and tendencies of gastrointestinal bleeding, and iron deficiency anaemia.
- Steatorrhea is a common symptom too
- Impaired portal circulation with increasing venous pressure may lead to esophageal varices (enlarged veins) with danger of rupture and haemorrhage.
- Blood clotting mechanisms are impaired: factors such as prothrombin and fibrinogen are not adequately produced by the damaged liver.
- Negative nitrogen balance also seen. (Due to general tissue catabolism)

Treatment

- No treatment will cure cirrhosis or repair scarring in the liver that has already occurred.
- Treatment can sometimes prevent or delay further liver damage.
- Treatment involves lifestyle changes, medication, and regular doctor visits.
- Surgery may be used to treat complications from cirrhosis.

The basis of treatment is adequate rest along with dietary modification.

Dietary Management

The objectives of nutritional therapy are:

- To promote regeneration of liver cells
- To correct nutritional deficiencies if any

Energy

Energy requirements are increased to correct malnutrition and to promote regeneration of liver cells.

However since patient is on bed rest, their actual energy expenditure is reduced.

Normal recommended energy intake should be enough to meet the extra needs.

Protein

In the absence of impending coma, the protein intake should continue in the level suggested for hepatitis; is 1.0 to 1.5 grams per Kg actual body weight.

- Help to overcome malnutrition,
- Regenerate liver cells
- Replenish plasma proteins

If signs of impending coma appear, the protein intake is decreased to 0.3g/kg body weight depending on the individual tolerance

Inclusion of large amounts of animal protein may lead to hepatic encephalopathy. Use plant proteins too.

Carbohydrates

As in infective hepatitis, a high carbohydrate diet is recommended to provide energy and to protect the liver cells from further damage.

A daily intake of 300g of carbohydrate is advised.

Fats

Restrict fat: Many cirrhotic p.t suffer from malabsorption of fat due to impaired bile secretion.

Amount will vary depending on the individual's tolerance.

Inclusion of moderate amount of fat in the diet increases palatability of the diet and promotes recovery.

Emulsified fats and those containing medium chain triglycerides are better tolerated.

Vitamins

The availability of fat soluble vitamins like vitamin A is affected due to the decreased intake and impaired absorption of fat.

Supplements of some vitamins may have to be provided to replenish liver stores and repair tissue damage.

Minerals

Presence of ascites and oedema necessitates the restriction of sodium in the diet.

Supplementation is recommended when diagnosis of a specific deficiency may be more costly and would delay the provision of micronutrients

Supplementation with zinc may improve food intake for pt. with dysgeusia.

3. HEPATIC ENCEPHALOPATHY/HEPATIC COMA

A condition characterized by degenerative changes in the brain and neurological symptoms.

Generally attributed to the fact that the failing liver can no longer inactivate or detoxify certain substances or metabolize others.

Causes

Four main theories have been suggested as the causative factors:

- **The Ammonia Theory**

An important theory which attributes the pathological changes to elevated blood levels of ammonia.

Based on the cirrhotic changes in the liver which diminish portal circulation

Ammonia is not converted to urea for excretion, accumulates in the blood and subsequently affects the brain

- **The synergistic theory**

Suggests that the encephalopathy is due to synergistic effect of ammonia and other substances like short chain fatty acids.

However, the measurements of such effects are difficult and have not been substantiated.

- **The Amino Acid Neurotransmitter theory**

Suggests that the failing liver induces an amino acid imbalance, with an accumulation of animal source amino acids in the central nervous system.

These amino acids are neurotransmitter precursors and their imbalance leads to coma. This theory has lately developed much support.

- **The GABA theory**

This theory is being investigated.

Gamma aminobutyric acid (GABA) is an inhibitory neurotransmitter which has been found to be increased in the plasma of patients with hepatic coma.

One of the reasons suggested for this increase is decreased removal of circulating GABA by the liver

Symptoms

- Changes in consciousness, behavior and neurological status.
- Apathy, mental confusion, drowsiness, leading to coma.
- Speech may be slurred and monotonous with blank facial expression.
- Motor change involving flapping tremors in arms and legs. This is due to sustained contraction in a group of muscles
- The breath may have fecal odour

- Urine output decreases with advancement in coma. If the condition is not checked, it may even prove fatal.

Dietary management

Objectives of dietary treatment include:

- Reducing protein intake
- Minimizing tissue catabolism.

Modification in the diet

Energy

Adequate energy intake contributes to healing and building up glycogen stores in the liver.

About 1500-2000kcal mainly from carbohydrate is sufficient to prevent tissue breakdown.

Protein

Should be restricted according to the patient's condition.

15 to 20g of protein daily may be given initially and gradually increased according to tolerance.

Fats

Fat intake is lowered and small amounts may be included as tolerated.

Emulsified and medium chain triglycerides are better tolerated.

Vitamins

Parenteral administration of vitamin K, B complex and C may be done incase of deficiencies.

Minerals

Sodium restriction may be advised if ascites is observed in the patient.

Close attention is given to other mineral deficiencies like iron.

Fluid intake

It is closely monitored according to the output.

4. LIVER TRANPLANT

Used to treat life threatening end stage liver disease for which no other form of treatment is available.

The transplantation involves total removal of diseased liver and its replacement with a healthy liver in the same anatomic location.

A major surgery transferring a liver from the donor with healthy organs but who is brain dead is done.

The success of the liver transplant depends on the successful immunosuppression to reduce the incidence of rejection of the transplanted organ

- Liver transplant is not a routine procedure and may be accompanied by complications related to:
    - lengthy surgical procedure immunosuppressive therapy infections
    - technical difficulties encountered in reconstructing the blood vessels and biliary tract.

Nutrition Therapy

Goals for nutrition therapy are:

To correct malnutrition particularly:

- Muscle wasting
- Electrolyte imbalance
- Abnormal blood glucose and lipids.

Pre-plantation diet

- Recommend an intake of 30-35kcal/kg of energy.
- 1.0-1.5g/kg of protein is given as tolerated.
- Vitamins and minerals are given depending with the RDA.
- Adjust sodium and fluid as appropriate.

Post-plantation Diet

- TPN may be necessary if oral and enteral feeding cannot be initiated within 5 days after surgery.
- Feeding by mouth begins when post-operative ileus resolves (a disruption of the normal propulsive ability of the gastrointestinal tract following abdominal surgery)
- Reduce TPN as enteral nutrition is increased.
- 30% of the total calories should be provided as fats, 50-60% as carbohydrates.
- Give protein at 1.5-1.75g/kg body weight.

DISEASES OF THE GALL BLADDER

The gall bladder concentrates bile formed in the liver and stores it until needed for digestion of fat.

The entrance of fat in the duodenum stimulates the secretion of the hormone cholecystokinin by the intestinal mucosa. The hormone reaches the gall bladder via the blood and causes it to contract releasing bile. Interference with the flow of bile impairs fat digestion.

1. CHOLECYSTITIS

This involves the inflammation of the gall bladder usually due to infection. Such an infection affects the normal function of the gall bladder

Causes

- Infection
- High dietary fat intake over a long period of time can predispose to gallstone formation.

Symptoms

- Nausea and vomiting
- Sensitivity to fatty foods
- Flatulence
- Chills and fever
- Colicky pain

Treatment

Treatment may involve surgical removal of the gall bladder- cholecystectomy.

Surgery may have to be withheld till acute infection subsides.

Till then, patient is kept in bed and suitable analgesics and antibiotics are administered.

Dietary treatment

Main aim of dietary treatment is to reduce discomfort by providing a diet restricted in fat.

In acute cases, it is advisable to keep the gall bladder at rest and minimize contraction thus fat is excluded from the diet.

2. CHOLELITHIASIS

Formation of stones (hard, pebble-like deposits) in the gall bladder.

Stones are made up of cholesterol, bile acids, calcium and other inorganic salts and bilirubin.

Associated with:

- Obesity

- Use of oral contraceptives
- Hypercholesterolemia
- Cholecystitis.

Signs & Symptoms

- Most patients have no symptoms
- Discovered during routine medical procedures such as x-ray, abdominal surgery etc.
- Cramping pain in the center to the right upper abdomen when a large stone blocks either the cystic duct or common bile duct
- Fever
- Jaundice
- Nausea & vomiting

Treatment

Treatment involves a hospital stay to control the inflammation in the gall bladder.

Doctor may recommend surgery to remove the gallbladder,(cholecystectomy) since gallstones frequently recur.

Once the gallbladder is removed, bile flows directly from the liver into the small intestine, rather than being stored in the gallbladder.

Nutrition therapy

Unless fat induces symptoms, a low fat diet is not necessary.

In acute gallstone attack, use a low fat diet to decrease gall bladder contraction and lessen the pain.

If the gall bladder is sluggish, a moderate fat intake is desirable to stimulate its contraction and prevent stagnation of bile.

CASE STUDY 1:

56 year old Mrs. M is admitted at Outspan Hospital after undergoing laparoscopic cholecystectomy, following prolonged cholelithiasis. She is 153M tall, 92 kg. She is also anaemic and has poor appetite. Answer the following questions to explain her nutritional management

- What is laparoscopic cholecystectomy
- What are the risk factors in the development of cholelithiasis
- Discuss the dietary management of Mrs. M, citing specific nutrient requirements.

CASE STUDY 2:

Mr.Q is suffering from liver cirrhosis. He is a recovering alcohol addict, currently in a rehabilitation centre. You have been asked to provide nutritional management for Mr. Q 's condition. Upon assessment, you find; he is 163 M tall, 43 kg. His lab results show that he has low Hb, elevated AST & ALT levels and steatorrhea,

- Explain the lab results
- Formulate a nutrition diagnosis statement
- Discuss the nutritional management of Mr. Q's condition, citing specific nutrient requirement.

16.3.5.3 Self-Assessment

1. Identify the types of hepatitis
2. Outline the symptoms of hepatic encephalopathy
3. Distinguish between cholecystitis and cholelithiasis
4. Outline the causes of gall stones
5. Explain the causes of liver cirrhosis
6. Which one of the following is a function of the liver:

   A. Synthesis of Vitamin D
   B. Storage of Vitamin C
   C. Production of bile
   D. Production of insulin

7. Which of the following is an implication of protein deficiency in liver cirrhosis?

   A. Fluid retention
   B. Gastrointestinal bleeding
   C. Anorexia
   D. Nausea and vomiting

8. Which one of the following is not true about the gall bladder:

   A. It temporarily stores bile
   B. It is not an essential organ
   C. High dietary fat intake over a long period of time can predispose to gallstone formation
   D. We cannot live without the gall bladder

9. Which one of the following is an objective of the nutritional management of hepatitis?

   A. To correct electrolyte imbalance
   B. To increase fat intake
   C. To prevent further liver damage.
   D. To promote negative nitrogen balance

10. The _____ theory of hepatic encephalopathy attributes the condition to elevated blood levels of ammonia.

    A. GABA

    B. Ammonia

    C. Synergistic

    D. Amino Acid- Neurotransmitter

16.3.5.4 Tools, Equipment, Supplies and Materials

- Stationery
- Reference materials
- Clinical guidelines
- WHO guidelines
- MOH guidelines
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise
- Computers with internet
- Library and resource centre

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16.3.6 Learning Outcome 5: Demonstrate understanding in nutritional management of metabolic disorders

16.3.6.1 Learning Activities

Learning Activities	Special instructions
1. Identify and describe terminologies under metabolic disorders	• Use terminologies under metabolic disorders
2. Identify metabolic disorders and discuss their pathophysiology	• Consider how common metabolic disorders affect nutrition
3. Identify and describe nutritional management of metabolic disorders	• Consider the nutritional implications of metabolic disorders • Plan diet for metabolic disorders • Consider drug-nutrient interaction in the management of metabolic disorders • Apply guidelines for the management of metabolic disorders

16.3.6.2 Information Sheet

Metabolic Disorders

These physiological disorders either result from altered metabolism or affect metabolism. These includes; diabetes mellitus, gout, hyper/hypothyroidism.

1. DIABETES MELLITUS

Diabetes mellitus is a chronic metabolic disorder that occurs when the pancreas does not produce enough insulin or when the body cannot effectively utilize the insulin it produces. It is characterized by decreased ability or complete inability of the tissue to utilize carbohydrates accompanied by a change in metabolism of fats, protein, water and electrolytes. This results in elevated blood sugar (hyperglycemia) which over time leads to multiple organ damage. It is associated with acute complications such as ketoacidosis and hypoglycemia, as well as long-term complications affecting the eyes, kidneys, feet, nerves, brain, heart and blood vessels.

There are three types of Diabetes mellitus.

- Type I: Results from the body's failure to produce insulin
- Type II: Results from Insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with relative insulin deficiency

- Gestational Diabetes: Pregnant women who have never had diabetes before but who have high blood sugar (glucose) levels during pregnancy are said to have gestational diabetes. Gestational diabetes affects about 4% of all pregnant women worldwide. It may precede development of type 2 (or rarely type 1)

Risk factors for diabetes mellitus

These include: heredity, age, sex, obesity, dietary factors, physical inactivity and infections

Common symptoms include:

- Increased thirst (polydipsia)
- Increased urination (polyuria)
- Increased hunger (polyphagia)
- Weight loss in Type 1 diabetes
- Over weight/Obesity in type 2 diabetes
- Sugar in the urine (glycosuria)
- Elevated blood sugar or glucose (hyperglycemia)
- Skin irritation or infection
- Weakness/general loss of strength

Management Plan

The aim of management in diabetes is to control blood sugar and prevent development of disease complications.

Objectives

- Attain and maintain blood glucose levels as close to normal as possible
- Prevent hypo- and hyperglycaemia
- Attain optimum blood lipids and blood pressure control and so reduce the risk of macro vascular disease
- To promote physical, social and psychological well being
- To prevent, delay or minimize the onset of chronic degenerative complications e.g. hypertension and renal diseases
- To achieve and maintain optimal metabolic and physiologic outcomes
- To provide relief from symptoms

Components of management plan

- Medical therapy
- Medical nutrition therapy
- Exercise and physical activity

Medical Therapy

Medical therapy includes clinical diagnosis, drug prescription and administration. Insulin administration is important in the nutrition therapy. Insulin doses need to be adjusted to balance with nutritionally adequate food and physical activity. The quantity of food at each meal should be consistent and at regular times every day and harmonized with drug intake.

The table below shows types of insulin, onset peak and duration of action and the consequences resulting when the drugs administration and diet intake is not harmonized in reference to drug action.

Type of insulin	Onset of action	Peak of action	Duration of action	Common pitfalls
Insulin lispro (Humalog)	5 to 15 minutes	1 to 2 hours	4 to 5 hours	Hypoglycemia occurs if the lag time is too long or the patient exercises within one hour of administration; with high-fat meals, the dose should be adjusted downward.
Regular insulin (Humulin R)	30 to 60 minutes	2 to 4 hours	6 to 8 hours	Lag time is not used appropriately; the insulin should be given 20 to 30 minutes before the patient eats.
NPH insulin (Humulin N)	1 to 3 hours	5 to 7 hours	13 to 18 hours	In many patients, breakfast injection does not last until the evening meal; administration with the evening meal does not meet insulin needs on awakening.
Lente insulin (Humulin L)	1 to 3 hours	4 to 8 hours	13 to 20 hours	Zinc suspension binds with regular insulin, which loses its effect if it is left in the syringe for more than a few minutes.
Ultralente insulin (Humulin U)	2 to 4 hours	8 to 14 hours	20 to 24 hours	Same as for lente insulin; in addition, peak of action is erratic in some patients.

Medical Nutrition Therapy

Medical nutrition therapy is an integral component of diabetes management. It has both short and long term benefits for diabetes outcomes. Dietary modification is one of the cornerstones of diabetes management, and is based on the principle of healthy eating in the context of social, cultural and psychological influences of food choices. Dietary modification and increasing levels of physical activity should be the first step in the management of diabetes mellitus that have to be maintained. The nutrition care process should be followed when managing diabetic patients.

Diabetes nutrition therapy aims to enable people with diabetes to make appropriate changes to their lifestyle in order to reduce the risks of both micro- and macro vascular complications and control blood sugar. Nutritional therapy should be individualized to accommodate age, nutritional needs, religion, culture, preferences and lifestyle. Nutrition therapy involves modifying both diet and patterns of physical activity.

Positive outcomes of the therapy include:

- Improved metabolic control
- Decreased risk of micro- and macro vascular complications
- Quality of life and life expectancy similar to that of the general population

Aims of the Nutrition Therapy

Diet therapy aims at tailoring the diet care plan in accordance with the prevailing clinical situation. Diet therapy is not only concerned with the prevention and management of micro and macro vascular complications but also chronic complications of diabetes.

The objectives of nutrition therapy are to:

- Attain and maintain blood glucose levels as close to normal as possible
- Prevent hypo- and hyperglycaemia
- Attain optimum blood lipids and blood pressure control and reduce the risk of macro vascular disease
- Assess energy intake to achieve optimum body weight (this can mean taking action to either increase or decrease body weight).
- Promote physical, social and psychological well being
- Prevent, delay or minimize the onset of chronic degenerative complications e.g. hypertension and renal diseases
- Achieve and maintain optimal metabolic and physiologic outcomes
- Provide relief from symptoms

Individualize meal plan according to a person's lifestyle and based on usual dietary intake

Essential considerations in planning the diet

- Determine energy requirements

  Calculate the energy requirement for each diabetic patient individually

  Type 1: base the energy requirement on needs for normal growth and development, physical activity and maintenance of desirable body weight

  Type 2: majority is overweight and obese therefore energy requirement is meant for weight loss

  Distribute energy in terms of carbohydrates, proteins and fats

- **Protein** allowance is essentially as that for normal individuals and should not exceed 1gm/kg. Protein should provide 15-20% of total energy in the diet.
- Energy from carbohydrates should contribute 45% to 60% of total calories. An amount of less than 100gms carbohydrates per day is not advisable as it leads to ketosis, on the other hand more than 300g per day may overburden the metabolic capacity. The

distribution and amount of carbohydrates between meals is extremely important to synchronize with the action of insulin and drugs.

- **Fats** should provide < 30% of energy

  A lower fat intake of up to 20% or less of the daily energy in case of obese adult diabetics

- Determine the type of carbohydrates and type of preparations

Give more of carbohydrate as complex starches e.g. whole grain cereals, roots and stem tubers, whole grain bread, rather than simple sugars because they breakdown more slowly to release glucose.

In case of hypoglycaemia provide some glucose

Nutritional Education and Counseling

It is important to educate all diabetic patients on the disease and its' management i.e.

- Causes, signs and symptoms
- Basic information about nutrition
- Nutrient requirements
- Healthy eating guidelines
- How to make healthy food choices
- Relationship between diabetes and diet
- Drug-nutrient interactions
- Acute and chronic complications and their management
- Encouraging self monitoring of blood glucose at home (SMBG) or at the nearest facility
- Self management training using food pyramid, plate model, signal system
- Administration of insulin
- Healthy lifestyle- i.e. importance of exercise and maintenance of ideal body weight
- Preparing structured meal plan using menus, food exchange lists, counting calories, counting carbohydrates, glycemic index
- How to deal with special situations-eating out, travelling, exercise, sickness, lifestyle
- Individual counselling
- Discuss the outcome of assessment with the diabetic client/patient
- Explain how the diagnosis has been arrived at
- Involve the patient in the formulation of the diet
- Discuss other factors that may affect the disease e.g. stress
- Fill in knowledge gap identified in the assessment

Exercise and Physical Activity

Exercise and physical activity is an important component in diabetes management. All patients should have individualized exercise and physical activity plan. This helps to:

- Improve insulin resistance and lipid profile
- Lower blood pressure
- Reduce mortality in Type I diabetes and can reduce HbA1c by 0.7% in Type II diabetes
- Protect against the development of Type II diabetes
- Maintain appropriate body weight

NB: It is recommended that every patient should have at least 30 minutes of exercise per day.

Diabetes Complications

a) Hypoglycemia

This is a metabolic disorder caused by a drop in the blood glucose level to below the normal minimum essential for normal (80mg/100ml) brain functioning.

Causes

- Uncontrolled diabetes
- Strenuous physical activity
- Inadequate food intake
- Excessive insulin administration
- Skipped meals and delayed meals
- Severe vomiting or diarrhea

Symptoms

Weakness, hunger, nervousness, dizziness, sweating, palpitation, disorientation, slurred speech, headache, shakiness

Advanced hypoglycemia symptoms are related to neuroglycopenia and include headaches, confusion and lack of coordination, blurred vision, anger, seizures and coma.

Aim of management

Increase the glucose level to normal.

Management

In acute state administer one sweet or one tea spoon of sugar to raise the blood glucose followed by a meal

For long term management advise the client to adhere to the diet recommendations as stipulated

b) Hyperglycemia

This is a condition that is characterized by elevated blood glucose.

Causes

Include insufficient insulin, ineffective insulin and untreated diabetes.

Symptoms and implications

Drawing of water from tissues into the blood leading to severe dehydration

Glycosuria (when blood glucose exceeds 180mg/100ml), polydepsia, polyuria, blurred vision, weight loss, fatigue, acetone breath, labored breathing.

Management

- Adjust dosage of regular insulin
- Enhance physical activity
- Reduce amount of CHO
- Space meals based on insulin activity time span

c) Metabolic Acidosis

This disorder results from a lowered blood and extracellular fluid PH of < 7.4

Risk factors/causes

Heavily meat based diet; uncontrolled diabetes; renal failure and prolonged fasting.

Implications

Acetone breath; dehydration; severe acidosis can result to fatal coma.

Aim of management

To control acidosis by increasing the blood PH

Management

- Treat underlying cause
- Withhold acidic foods especially meat
- Use of plant based foods that are alkaline in nature .e.g. potatoes is recommend
- Take safe drinking water based on tolerance.

2. GOUT

A disorder of purine metabolism in which abnormal levels of uric acid accumulate in the blood and result to deposition of uric acid at the joints. It is a manifestation of inflammation and sharp joint pain because of crystallized deposits of uric acid.

Main **Risk factors/causes**

- Excessive intake of red meat and fish which result to elevated uric acid in the blood
- Excessive intake of alcohol, as it blocks the elimination of uric acid from the body
- Excessive consumption of stimulant beverages as caffeine if part of the chemical family of purine. It transforms into uric acid in the body
- Hormonal factor
- Obesity.

Symptoms/ implications

Inflammation and pain of the joints especially the meta tarsal pharyngeal (the base of big toe)

- A risk factor to chronic arthritis
- Aim of nutritional management
- Prevent excessive accumulation of uric acid

Management

- Use of low purine diet by restricting consumption red meat, fish, alcohol, stimulants, and high protein foods to avoid exogenous addition of purines to the existing high uric acid load is recommended
- Encourage consumption of alkalizing foods e.g. lemons, tomatoes, green beans, fruits milk and milk products
- Intake of fluids about 3lts/day to enhance excretion of uric acid based on assessment is recommended
- Moderate protein intake (0.8g/kg/day)
- Maintain adequate CHO intake to prevent ketosis
- Limit fat intake
- Avoid large and heavy meals late in the evening
- Encourage consumption of whole grains.

3. HYPERTHYROIDISM

This is condition due to overactive thyroid gland. This may be initiated by hormonal imbalances **or tumors.**

Causes

- Hormonal imbalances
- Tumors

Symptoms and implications

- Increased metabolic rate
- Excessive production of the thyroid hormones
- Increased energy expenditure and weight loss
- Nervous excitation due to excessive hormone product
- Tachycardia (high heart rate)
- Increased perspiration and heat sensitivity

Aims of management

- To prevent/control weight loss-through provision of high calorie diet.
- Reduce workload

Management

- Treat the underlying cause
- Use of high calorie diet to meet the extra energy needs is recommended
- Refer to high calorie diet

4. HYPOTHYROIDISM

This is state resulting from reduced activity of the thyroid gland. The gland does not produce sufficient levels of thyroxine hormone.

Causes

Inadequate iodine intake and selenium deficiency

Symptoms/implications

- Enlargement of thyroid gland as the cells enlarge to trap as much iodine as possible
- Sluggishness and weight gain
- In pregnancy it can result to impaired fetal development

Aim of management

To control iodine deficiency

Management

- Recommend iodine rich foods e.g. sea foods or iodine fortified foods
- Recommend suitable exercise program

16.3.6.3 Self-Assessment

1. Outline the causes of hypoglycemia
2. Explain the common symptoms of diabetes
3. Discuss the nutritional management of diabetes
4. List the causes of gout
5. Explain the signs and symptoms of hyperthyroidism
6. Hypoglycemia is:
   A. Abnormally high blood glucose
   B. Low blood glucose
   C. The amount of glucose in a carbohydrate food
   D. Blood glucose level after a meal
7. Which one of the following is not true about diabetes:
   A. All obese people develop diabetes
   B. Diabetic people cannot participate in sports
   C. Type 2 diabetes results from insulin resistance
   D. Diabetics can consume more simple carbohydrates than complex carbohydrates
8. The following are risk factors of gout except:
   A. Excessive intake of red meat and fish
   B. Excessive intake of alcohol
   C. Excessive consumption of stimulant beverages as caffeine
   D. Weight loss
9. The following are symptoms of hyperthyroidism except:
   A. Increased metabolic rate
   B. Decreased energy expenditure and weight loss
   C. Tachycardia
   D. Increased perspiration and heat sensitivity
10. The aim of nutritional management in diabetes is:
    A. To control blood pressure
    B. To increase bmi
    C. To control blood sugar and prevent development of disease complications
    D. To reduce physical activity.

16.3.6.4 Tools, Equipment, Supplies and Materials

- Stationery
- Reference materials
- Clinical guidelines
- WHO guidelines
- MOH
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise
- Computers with internet
- Library and resource centre

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16.3.7 Learning Outcome 6: Demonstrate understanding in nutritional management of mental and mood disorders

16.3.7.1 Learning Activities

Learning activities	Special instructions
1. Identify and describe terminologies under mental and mood disorders	• Use terminologies under mood disorders
2. Identify mental and mood disorders and discuss their pathophysiology	• Consider how mental and mood disorders affect nutrition
3. Identify and describe nutritional management of mental and mood	• Determine the nutritional requirements in metabolic disorders • Plan diet for mental and mood disorders • Apply guidelines provided for the management of mental and mood disorders

16.3.7.2 Information Sheet

Definition

Mental health; Refers to emotional, psychological and social well-being of an individual.

Mental health conditions can cause individuals disability and even cause death through suicides. Medical care for mental conditions only provides partial benefit. Nutrition intervention supports medical treatment by enhancing function of the nervous system and dealing with nutritional problems caused –by the mental conditions.

Diet therapy also helps in management of the side effects of medication, such as poor appetite.

Interventions provided by Registered Dietitians to individuals with mental health conditions and their care providers can lead to reduced nutrition-related side effects of psychiatric medications, improved cognition, better self-management of concurrent and comorbid conditions, and improved overall occupational, social, and psychological functioning.

1. AUTISM SPECTRUM DISORDERS

Autism spectrum disorders (ASD) are pervasive developmental disorders with the onset usually before 3 years of age. Individuals with ASDs usually have communication, social, and behavioural characteristics in common, with individual differences in levels of functioning

ASD may impact appetite, with increased needs being common.

Nutritional Implications and Recommendations:

The diets of children with ASD may lack dairy, fibre, calcium, iron, and vitamins D and E.

Some may respond to increased intake of omega-3 fats, especially docosahexaenoic acid (DHA), ranging from 1 g to 3 g per day.

Glutenfree, casein-free diets are often advocated for ASD, but the current evidence is limited.

Autistic person exhibit some difficult feeding behaviours may affect food intake. Examples:

- Limited diet
- Dysfunctional feeding behaviour
- Sensory sensitivities.

2. ATTENTION DEFICIT HYPERACTIVITY DISORDER

Attention deficit/hyperactivity disorder (ADHD) includes inattention (e.g., distractibility), hyperactivity, and/or impulsivity (e.g., fidgeting, excessive running, interrupting others). This condition can affect children and adults — up to 60% of those with ADHD are adults. There is a high overlap of ADHD with other conditions, including dyslexia (reading problems), dyspraxia (motor skill problems), and autism spectrum disorders. A challenge of working with people with ADHD is that they may have impaired ability to retain and use new information after counselling. Individuals with ADHD tend to have deficiencies of polyunsaturated fatty acids, zinc, magnesium, and iron. Serum ferritin and zinc levels may be low; supplementation of iron and zinc helps symptoms if there is deficiency

- If the child is food sensitive, an additive-free diet (no food colours or preservatives) may improve symptoms but needs to be supervised by a Registered Dietitian to ensure adequacy.
- Though sugar is thought to cause hyperactivity, research suggests removal of this ingredient from the diet will not improve symptoms.
- The individual with ADHD should be checked for celiac disease and, if present, a gluten-free diet can improve behaviour.
- The ketogenic diet has been suggested for ADHD, but the available evidence is only based on animal experiments.
- Supplementation with magnesium and iron therapies may help reduce ADHD severity.
- Some studies show lower levels of docosahexaenoic acid (DHA) and arachidonic acid (ARA) in children with hyperactivity.
- For the person who is hyperactive during meals, behavioural management programs may be effective.
- Children with ADHD are often prescribed stimulants (e.g., methylphenidate or Ritalin) to improve the ability to concentrate. These medications have been shown to reduce growth in children . Height and weight should be monitored (measured at least twice a year) and dietary advice that focuses on consumption of adequate calories from a healthy balanced diet provided. Altering the times and dosages of stimulant medication and taking breaks from their use (e.g., during summer holidays) may help reduce effects on growth

3. BIPOLAR AND RELATED DISORDERS

Bipolar disorders include a history of manic, mixed, or hypomanic episodes, usually with concurrent or previous history of one or more major depressive episodes. Mania is an abnormally elated mental state, typically characterized by feelings of euphoria, lack of inhibitions, racing thoughts, and diminished need for sleep, talkativeness, risk taking, and irritability. In extreme cases, mania can induce hallucinations and other psychotic symptoms. Bipolar disorders may be classified as bipolar I, bipolar II, or cyclothymia, depending on the severity of symptoms.

Nutritional concerns and recommendations

- The cyclical nature of bipolar disorder presents unique challenges for nutritional care. During mania, large amounts of sugar, caffeine, and food may be consumed or there may be periods of not eating. If the individual is in a controlled environment, measures can be put into place to ensure healthy foods are available in order to prevent weight gain from overeating.
- With mood instability, contact with health care providers may be infrequent, leading to increased risk of developing a chronic condition.
- Depressive episodes can lead to increased risk of cardiovascular disease through the effects of a sedentary lifestyle.
- Compared with those without a mental health condition, people with bipolar disorder are more likely to report poor exercise habits and suboptimal eating behaviours such as having fewer than two daily meals and having difficulty obtaining or cooking food.
- Antipsychotic medications are often prescribed as treatment for this condition, which contributes to weight gain and metabolic disturbance as detailed in the previous section on the schizophrenia spectrum and other psychotic disorders.
- Celiac disease, which is associated with increased prevalence of depressive and disruptive behaviours, should be tested for.
- If the individual is taking lithium, caffeine-containing drinks such as tea and coffee should be minimized as they can reduce lithium levels.
- Selenium, folic acid (folate), omega-3 fatty acids, and tryptophan have all been implicated in keeping moods stable. A diet rich in these nutrients should be tried before considering supplements.
- Supplementation with 1 g to 3 g of omega-3 fatty acids daily may help with depressive episodes.
- If folate supplementation is warranted, it may mask a deficiency of vitamin B12; therefore, supplementation with vitamin B12 should also occur.

4. DEPRESSIVE DISORDERS

Within the group of depressive disorders are chronic depressive (dysthymia), disruptive mood dysregulation, major depressive, and premenstrual dysphoric disorders.

Depression is manifested by a combination of symptoms that interfere with the ability to work, study, sleep, eat, and enjoy pleasurable activities.

Disabling episodes of depression commonly occur several times in a lifetime.

Dysthymia involves longterm (two years or longer) less severe symptoms that keep one from functioning normally or from feeling good. Some forms of depressive disorder exhibit slightly different characteristics or they may develop under unique circumstances and include psychotic

depression (depression accompanied by psychosis), postpartum depression *(*new mother develops a major depressive episode within one month of delivery), and seasonal affective disorder (onset of depression occurs during the winter months when there is less

natural sunlight).

Almost all chronic health conditions are associated with major depression, particularly those characterized by inflammation and pain.

- Depression often leads to weight changes as appetite may increase or decrease. For some, overeating or comfort eating may occur and lead to weight gain. The tendency in this population to carry excess weight may be exacerbated by a preference for higher-calorie liquids and/or convenience foods as well as a sedentary lifestyle.
- Other individuals with depressive disorders may undereat due to feelings such as not being worthy enough to eat, lacking motivation or energy to prepare foods, or somatic delusions of not being able to eat. Reduced food intake leads to nutrient inadequacies and weight loss.
- Tube-feedings may be needed for those who refuse food. Total parenteral nutrition (TPN) is usually not recommended as the TPN line may be used to inflict sepsis or other harm (e.g., suicide attempt).
- A well balanced diet with protein/calorie supplementation as needed and structuring eating for mood stability throughout the day may help.
- Poor food hygiene presents food safety risks so advice may be given to care providers to assist the person with keeping food safe.
- Because celiac disease is associated with an increased prevalence of depressive disorders, it is recommended that testing be done to rule it out.
- Depressive disorders may coexist with an eating disorder, thereby requiring behavioural interventions to normalize eating.

16.3.7.3 Self-Assessment

1. Explain how depressions affects nutrition
2. Explain three difficult feeding behaviour common in autistic children
3. Outline the nutrition concerns in bipolar disorder

4. Which of the following statements is not true about Attention Deficit Hyperactivity Disorder:

   A. Children with sensitivities should consume food free of additives

   B. Removal of sugar from the diet improves symptoms

   C. For patients with celiac disease, a gluten-free diet is recommended

   D. Supplementation with magnesium and iron therapies may help reduce ADHD severity.

5. _____is an abnormally elated mental state, characterized by feelings of euphoria, lack inhibitions, racing thoughts, and insomnia, talkativeness, risk taking, and of irritability.

   A. ADHD

   B. Autism

   C. Mania

   D. Bipolar disorder

6. The following nutrients help in mood stabilization in bipolar disorder except:

   A. Selenium

   B. Folic acid

   C. Omega-3 fatty acids

   D. Methionine

7. _____ refers to emotional, psychological and social well-being of an individual.

   A. ADHD

   B. Bipolar disorder

   C. Mental health

   D. Psychiatry

8. The following conditions affect the health and nutrition of autistic individuals except:

   A. Limited diet

   B. Dysfunctional feeding behaviour

   C. Gluten-free diet

   D. Sensory sensitivities

16.3.7.4 Tools, Equipment, Supplies and Materials

- Stationery
- Reference materials
- Clinical guidelines
- WHO guidelines
- MOH guidelines
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise
- Computers with internet
- Library and resource centre

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16.3.8 Learning Outcome 7: Demonstrate understanding in nutritional management of degenerative disorders

16.3.8.1 Learning Activities

Learning Activities	Special instructions
1. Identify and describe terminologies under degenerative disorders	• Use terminologies under degenerative disorders
2. Identify degenerative disorders and describe their pathophysiology	• Consider how degenerative disorders affect nutrition
3. Identify and describe nutritional management of degenerative disorders	• Determine the nutritional requirements in degenerative disorders • Plan diet for degenerative disorders • Apply guidelines provided for the management of degenerative disorders

16.3.8.2 Information Sheet

1. Amyotrophic Lateral Sclerosis (ALS)

Amyotrophic lateral sclerosis (ALS) is the most common of the MNDs, defined by the progressive degeneration of upper and lower motor neurons, and causes atrophy, fasciculation, weakness and spasticity .

The natural course of ALS can be defined as death from respiratory failure. The prevalence rate of ALS currently is 2.7-7.4 per 100,000 inhabitants; the incidence is 1.9 per 100,000 inhabitants.

The aetiology of ALS is not fully clarified; however, excitotoxicity from glutamate neurotransmitter, changes in immunity, deficiency of neurotrophic factors, physical traumas, persistent viral infections and even environmental factors has been suggested as possible causes of the disease.

During the course of ALS, nutritional status (NS) declines, and is often inadequately treated in clinical practice, Although various studies confirm the correlation between decrease in body weight and body mass index as negative predictors of survival.

Some factors are inherent to the amendments to the NS and the reduction of food intake in patients with it, such as: loss of appetite, dysphagia, dyspnea, depression and hyper-metabolism. Hyper-metabolism origin and development in ALS have not been thoroughly elucidated; 50% of these patients, however, present in hypermetabolic state causing the increased nutritional needs make the nutritional treatment even more complex.

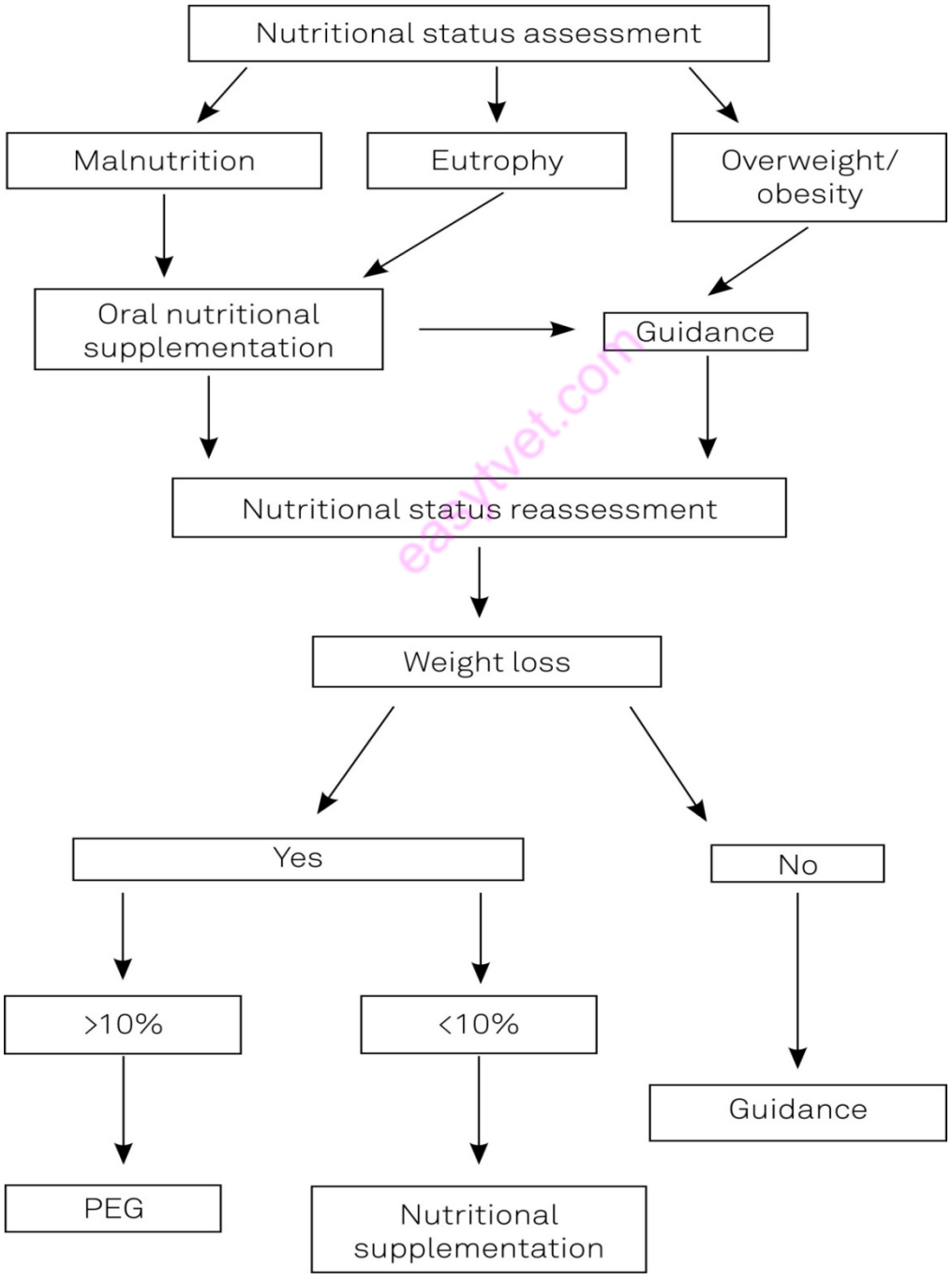
The disease affects bulbar muscles of patients first, from 25% to 30%, resulting in progressive dysphagia and leading to decrease in food and hydric intake. The need for modifying diet consistency contributes to reduction of high-value food-protein energy. In analysis of food intake, found that only one of sixteen patients studied presented appropriate recommendations for front intake energy.

Malnutrition, common with progression of disease, muscle strength and breathing capacity due to weakening as well as increase the relative risk of death .

Meet the energy requirements of macro and micronutrients, adjust degree of dysphagia brought and indicate enteral nutritional therapy at right time have been the subject. Within this context, this present review aims to present nutritional treatment strategies for the maintenance of NS of ALS patients.

Nutrition intervention

The identification of nutritional status through diagnostic methods leads to the formulation of a plan to deal with each situation individually.



Nutritional treatment protocol

Dietary Recommendation

Nutritional therapy applied to ALS aims to supply the nutritional needs for all stages of disease progression, minimise protein catabolism, ensure oral feeding and indicate early nutritional support.

Energy needs

Estimate energy requirements used with the current weight while considering factors such as activity and injury.

Protein

The calculation of protein requirements is based on the recommendations of the Brazilian Society of Food and Nutrition , being offered 1.0 g–1.2 g protein/kg in current weight/ day. Food intake, source of high biological value protein, should be highlighted, comprising roughly 70% in total protein intake per day.

Carbohydrates

The diet is oriented normoglycidic (50-60% of total energy intake) and modified to hypoglycemic, specifically for patients with abnormal oxygen saturation. For these cases, the energy supply must be maintained at the expense of lipids, with high-fat diets (>35% of total energy intake).

Lipids

It is recommended ingesting 25-35% of total energy intake, considering ≤7% saturated fatty acids, ≤10% polyunsaturated fatty acids and ≤20% monounsaturated fatty acids

Fibres

The fibre intake gradually restricts itself in the course of the disease, worsening dysphagia. Cooking food is necessary for a suitable consistency limitation, ensuring that its consumption is not excluded from diet.

Adding cereal flour to porridge, orange juice with papaya and dried pitted prunes, liquefied food, boiled and mashed vegetables and legumes cooked well are preparations easy to swallow and help intestinal transit.

When adequate intake of fibres becomes impossible, supplemental fibre with module containing industrialized mix of soluble and insoluble fibres is necessary.

According to the age and sex of the individual, 25-38 g of dietary fibre intake per day is **recommended.**

Water

At times, when dysphagia signals occur, liquid must be thickened to reduce the aspiration risk and offer the proper water requirement to patients. The use of industrialised thickeners grants a more suitable consistency to liquid, however, excessive consumption can worsen constipation. Gelatin, with or without flavour, can also be used as a means to modify food consistency.

Food with high liquid content, e.g. fruit and vegetable puree, fruit juice and smoothies assist in hydration and reaching the estimated water requirement of 30-40 mL/kg/day

Micronutrients

The micronutrient prescription follows the recommendations of dietary intake recommended according to age and sex of patients.

2. PARKINSON'S DISEASE

It's a degenerative disorder of the central nervous system.

It's the second most common neural degenerative disorder and the most common movement disorder.

It's characterized by progressive loss of muscle control which leads to trembling of the limbs and head at rest, stiffness, slowness and impaired balance.

As symptoms worsen it may become difficult to take and complete simple tasks.

The progression of Parkinson's disease and degree of impairment vary from individual to individual and many people with Parkinson's leave long productive life whereas others become disabled much more quickly.

Premature death is usually due to complications such as falling related injuries or pneumonia

Causes

The neurotransmitter dopamine acts as a messenger between two brain areas to control movement. They are :

- Substantia nigra
- Corpus stratium

Most of the movement related symptoms of Parkinson's diseases are caused by lack of dopamine due to the loss of dopamine producing cells in the substantia nigra.

When the amount of dopamine is too low communication between the substantia nigra and the corpus stratium becomes defectivesand movement becomes impaired.

The greater loss of dopamine the worst the movement related symptoms.

Risk Factors

- Age –is the largest risk factor for the development and progression of prisons disease and most people who develop Parkinson disease are older than 60 years of age
- Sex or gender –men are affected about 1.5 -2 times more often than women
- Genetics- a small number of individuals are at a n increased risk because of a family history of the disorder
- Head trauma illness or exposure to environmental toxins such as pesticides and herbicides maybe at a risk.

Symptoms

- The primary symptoms of Parkinson's disease are all related to voluntary and involuntary motor function and usually starts on one side of the body
- Symptoms are usually mild at first and they will progress overtime
- Tremors –trembling in fingers, arms, feet ,legs ,jaw or head they most often occur while the individual is resting they may worsen when an individual is exited, tired or stressed
- Rigidity-stiffness of the limbs which may increase during movement and they may produce muscle aches and pain loss of fine movement may lead to crammed handwriting and may make eating difficult
- Bradykinesia –it's the slowness of voluntary movement bradykinesa together with stiffness affects the facial muscles and therefore resulting to an expressionless appearance
- Postural instability-impaired or lost reflex can make it difficult to adjust posture to maintain balance
- Other symptoms: anxiety confusion constipation depression difficulty in swallowing diminished sense of smell male erectile dysfunction urinary frequency slowed quieter speech and excessive salivation.

Medical Treatment

- There is currently no treatment to cure Parkinson's disease.
- Several therapies are available to delay the onset of motor symptoms and to decrease the motor symptoms.
- All this therapies are designed to increase dopamine movement in the brain
- Either by replacing dopamine or prolonging the effect of dopamine inhibiting its symptoms.
- The main drug levodopa or combination with carbidopa.

Nutrition Therapy

Parkinson's disease shows mild to moderate nutritional depletion with weight loss less of subcutaneous fat varying degrees of swallowing difficult.

Dysphagia becomes a significant feeding programme in the final stages of the disease while rigidity is the permanent feature.

The nutritionist should focus on the dysphagia difficult in self feeding and the nutrients drug interactions.

- If patient is taking levodopa.a high level of protein interferes with this drug.
- Restrict protein to 0.8g/kg/day
- Patients responding poorly to levodopa may benefit from the intake of low protein meals for breakfast and lunch, followed by an evening meal that provides the balance of 0.8g/kg/day allowance for protein.

- Give proteins of high biologic value, modifying the consistency according to the chewing and swallowing abilities of the patient.
- Serve the protein rich foods mainly in the evening meal.
- This may reduce tremors and allow some normal functioning during the day.
- However, consumption of a larger protein meal in the evening can result in suboptimal levodopa effect and increased rigidity.
- Tyrosine (a precursor of dopamine) maybe beneficial.
- Limit the intake of pyridoxine to less that 5mg/day to make levodopa move effective.
- Avoid constipation and encourage the intake of fluids.
- Promote independence with self feeding. Get the patient in the best possible for feeding and use adaptive feeding devices such as cups with double handles and timed dishes.
- Serve small frequent meals and use semisolid foods rather than fluids if swallowing is a problem.

3. ALZHEIMER'S DISEASE

It's a form of dementia characterized by a group of symptoms that include loss of memory thinking and reasoning power disorientation confusion and sometimes speech disturbances.

The exact causes of Alzheimer disease depends on its characterized symptoms.

A victim gradually looses memory and reasoning, the ability to communicate, physical capabilities and eventually life itself.

Nerve cells in the brain die and the communication between the cells breaks down

Other symptoms include early depression found in 30% of the patient's mild anemia repetition of words and sounds

NB: Early onset is usually mild and middle and then there is late onset.

This disease progresses to death due to infection or malnutrition.

Food intake is affected and weight loss is common and the following are typical feeding problem seen during the three stage of the disease.

Stages of Alzheimer's Disease

Stage 1: difficult in shopping and cooking unusual food choices decreased appetite changes in taste and smell and forgetting to eat.

Stage 2- : loosing ability to use utensils eating with hands holding food in the mouth forgetting to swallow and failing to chew food before swallowing.

Stage 3-: no recognition of food refusing to eat or opening the mouth and dysphagia.

Causes

- Genetics
- Cardiovascular disease i.e. high blood pressure and diabetes maybe related to the development of Alzheimer's disease

Medical Treatment

Medical treatment involves providing care to clients and support to their families.

Drugs are use to improve or at least slow the loss of short term memory and cognition but they do not treat the disease.

Other drugs maybe used to control depression anxiety and behavior problems.

Nutrition Therapy

- Feeding must be individualized and one should be able to recognize and assess feeding problems associated at each stage of the disease
- Encourage and promote as much independence in eating by selecting appropriate food consistency providing adequate time t eat using the appropriate feeding equipment and technique and by providing the proper dining environment
- Give small frequent meals of nutrient dense foods
- Supplemental vitamins and minerals maybe necessary monitor body weight and guard against dehydration and aspiration of food
- Delay tube feeding unless absolutely necessary

16.3.8.3 Self-Assessment

1. Discuss the dietary management of Parkinson's disease.
2. Discuss the stages of Alzeihmer's disease.
3. _____is a progressive loss of muscle control which leads to trembling of the limbs and head at rest, stiffness, slowness and impaired balance.

   A. Alzheimer's Disease

   B. Parkinson's Disease

   C. ALS

   D. Dysphagia
4. Nutritional care in ALS aims to do the following except:

   A. To supply the nutritional needs for all stages of disease progression

   B. Minimise protein catabolism

   C. Ensure oral feeding

   D. Reduction of high-value food-protein energy.

5. Which of the following is a risk factor for Parkinson's Disease?

   A. Gluten-free diet

   B. Head trauma

   C. Obesity

   D. High fat diet

6. _____ is a form of dementia characterized by a group of symptoms that include loss of memory thinking and reasoning power disorientation confusion and sometimes speech disturbances

   A. Depression

   B. ADHD

   C. Alzeihmers

   D. Parkinson's Disease

7. Which of the following is true about the nutritional management of Parkinson's disease:

   A. Tyrosine improves symptoms

   B. Intake of pyridoxine should be increased

   C. Protein foods should not be taken in the evening as they increase tremors

   D. High level of protein enhances drug utilization

16.3.8.4 Tools, Equipment, Supplies and Materials

- Stationery
- Reference materials
- Clinical guidelines
- WHO guidelines
- MOH guidelines
- Ministry of Education
- Skills lab
- Use of LCDs, video clips, charts and other teaching aids
- Invitation of competent expertise
- Computers with internet
- Library and resource centre

16.3.8.5 References

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