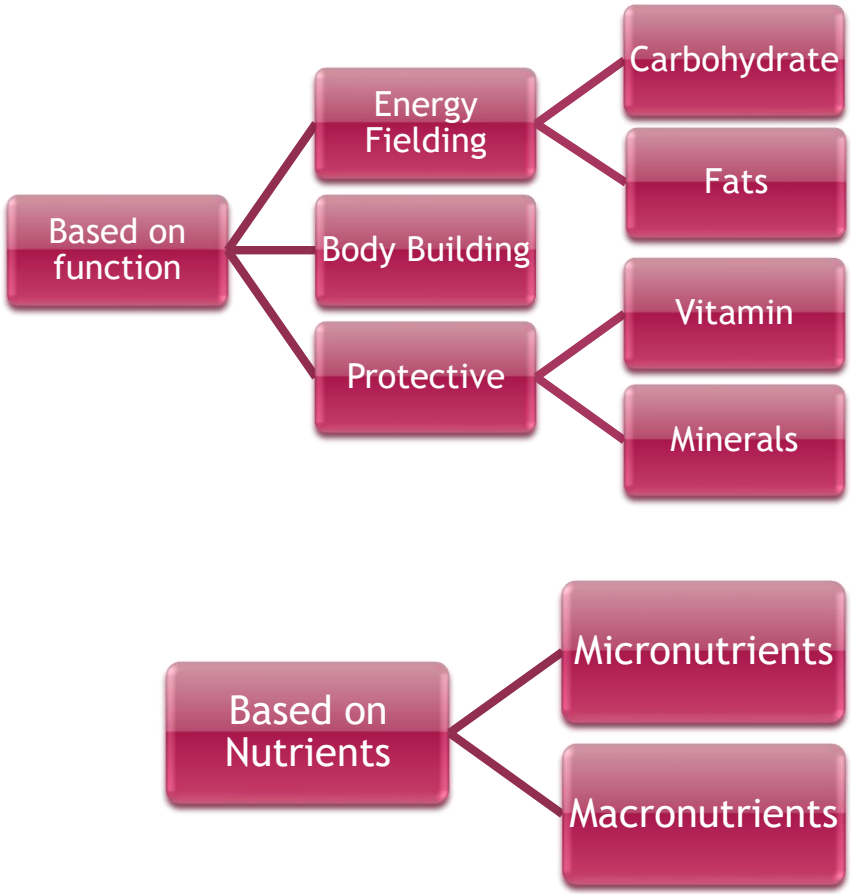
INTRODUCTION

- Food is one of the important and basic biological need of man. Food is the foundation for good health.
- It is essential for life growth and repair of human body, regulation of body mechanism and production of energy for work.
- Food can be achieved only through adequate nutrition that consist of essential nutrients.

CLASSIFICATION OF FOOD

- Food is classified into two catagories:



ENERGY FIELDING

- Energy fielding refers to the amount of energy that is gained from harvesting an energy sources.
- Eg.- cereals ,sugars , fat and oils etc.

BODY BUILDING

- The body building diet is designed to build muscles and reduce body fat
- Eg. Milk, Egg, Pulses, Poultry etc.

PROTECTIVE

- They help to keep the body healthy by regulating body process and helping the body to produce substances that fight disease causing agent.
- Eg. Vegetable, Fruit, Milk, Etc.

CLASSIFICATION OF NUTRITIONAL PROBLEM

- PROTEIN ENERGY MALNUTRITION
- MICRONUTRIENT DEFICIENCY
- MACRONUTRIENT DEFICIENCY

NUTRITIONAL DISORDER

- Nutritional disorders can be caused by insufficient intake of food or of certain nutrients, by an inability of the body to absorb & used nutrients, or by over consumption of certain food.
- Eg. Obesity – caused by excess energy intake

  Anemia – caused by insufficient intake of iron.

WHO IS AT RISK.?????

MALNUTRITION

- Definition :-

  Malnutrition is a condition that results from eating a diet in which nutrients are either not enough or too much such that the diet causes health problems. It may involve calories, proteins, etc..

PROTEIN ENERGY MALNUTRITION

Introduction :-

- It is a group of body depletion disorder which include kwashiorkor, marasmus.
- PEM is also referred to as protein – caloric malnutrition.
- It is considered as the primary nutritional problem in India, also called as first national nutritional disorder.
- PEM is due to “food - gap” between the intake and requirement.

DEFINITION

- It is a vesting condition resulting from diet inadequate in either protein and energy both called PEM

ETIOLOGY

- Different combination of many eitiology factors can lead to PEM in children they are:
  - Social economical factors
  - Biological factors
  - Environmental factors
  - Age of the host

UNDER SOCIAL, ECONOMIC, BIOLOGICAL AND ENVIRONMENTAL FACTORS INCLUDES:-

I. Poverty
II. Infection
III. Ignorance
IV. Overcrowding and family
V. Illiteracy
VI. Lack of health education

TYPES OF PROTEIN ENERGY MALNUTRITION

KWASHIORKOR

MARASMUS

KWASHIORKOR
MARASMUS

KWASHIORKOR

Kwashiorkor is the most common and widespread nutritional disorder in developing countries. It is a form of malnutrition caused by not getting enough protein in the diet.

DEFINITION

Kwashiorkor also called protein malnutrition condition caused by severe protein deficiency, characterized by edema, irritability, anorexia.

SYMPTOMS

- Wasting
- Mental Change
- Hair Change
- Skin Change
- Diarrhea
- Moon Face

TREATMENT

- Generally disease can be treated by adding food energy & protein to the diet . It can have long term impact on child's physical and mental development & in severe cases may lead to death
- It is recommended to give 4/kg of body weight of protein and 120-140/kg/day.

PREVENTION & CONTROL

- By Including food rich in protein eg. Wheat, grain, peanut are recommended.
- Pregnant and lactating women are educated and provided supplementary diet.
- Encourage breastfeeding.

MARASMUS

DEFINATION

- The word “Marasmus” comes from a Greek word and it means “Starvation”
- Marasmus is generally known as the gradual wasting away of the body due to severe malnutrition or inadequate absorption of food.
- Marasmus is a form of severe protein deficiency and is one of the form of PEM.
- It is a severe form of malnutrition caused by inadequate intake of protein and calorie.

SYMPTOMS

- Severe growth retardation.
- Severe muscle wasting.
- The child looks apparently thin and limbs appear as skin and bones.
- Wrinkled skin.
- Associate vitamin deficiency.
- Failure to thrive.
- Frequent watery diarrhea and dehydration.
- Edema and fatty infiltration are absent.

Classification of Marasmus

TREATMENT

- It is necessary to treat not only the symptom but also the complications of the disorder, dehydration and circulation disorders, which are frequently lethal and lead to high mortality, if ignored.

PREVENTION AND CONTROL

- A protein rich diet such as a combination of wheat, gram, protein or a diet with animal protein like mutton, chicken and fish will help the patient to return back to health.

DIFFERENCES IN CLINICAL FEATURES BETWEEN MARASMUS AND KWASHIORKOR

s.no	Clinical Features	Marasmus	Kwashiorkor
1.	Muscle Wasting	Obvious	Sometimes hidden by Edema and Fat
2.	Fat Wasting	Severe loss of subcutaneous fat	Fat often retained but not firm
3.	Edema	None	Present in lower legs, face & arms
4.	Weight for height	Very low	May be marked by edema
5.	Mental change	Sometimes quite and apathetic	Irritable, moaning apathetic
6.	Appetite	Usually good	Poor
7.	Diarrhoea	Often	Often
8.	Skin changes	Usually none	Diffuse pigments
9.	Hair changes	Seldom	Sparse, silky, easily pulled on
10.	Hepatic enlargement	None	Due to accumulation of fat

MARASMIC- KWASHIORKOR

DEFINATION

- A severely malnourished child with features of both marasmus and kwashiorkor

FEATURES

- The features of kwashiorkor are severe edema of feet and legs and also hands, lower arms, abdomen and face. Also there is pale skin and hair and the child is unhappy.
- There are also signs of marasmus, wasting of muscle of the upper arms, shoulders and chest.

TREATMENT

- Treatment strategy can be divided into three stages:
  - Resolving life threatening conditions
  - Resolving nutritional status
  - Ensuring nutritional rehabilitation

PREVENTION

- Promotion of breast feeding
- Development of low cost weaning
- Nutrition education and promotion of correct feeding practice
- Family planning & spacing of birth
- Immunization
- Early diagnosis and treatment

MICRONUTRIENTS AND MACRONUTRIENTS DEFICIENCY

MICRONUTRIENTS

- Micronutrients are the chemical element that is essential in minute amounts to the growth and health of living organism, for example

  - VITAMIN DEFICIENCY
    - Water soluble: vitamin C,B1,B2,B3,B5,B9,B6,B12, Cholin , Biotin
    - Fat soluble: Vitamin A,D, E, K, K1, K2, K3.

  - MINERAL DISORDER
    - Calcium, Copper, Iron, Magnesium, Phosphorous, Potassium, Sodium, Zinc.

VITAMIN DEFICIENCY

WATER SOLUBLE VITAMINS

Dissolves easily in water, readily excreted from the body

Vitamin C:

- It is also called as citric acid.
- It is Anti oxidant and helps protect cells and keep them healthy
- DEFICIENCY: SCURVY

SIGN AND SYMPTOMS OF SCURVY ARE:

- Bleeding gums
- Anemia weakness delayed wound healing
- Bleeding into the skin or joint
- Hematuria
- Melena

PREVENTION

- Providing vitamin c containing fresh food and encourage breast feeding.

MANAGEMENT

- Loading dose of vitamin c 500mg oral or parental , followed by daily dose of 100-300 mg for several week.

Vitamin B1 (Thiamine)

- They work as Co enzyme, they help in metabolism of carbohydrate, protein, fat
- DEFICIENCY: Beriberi

SIGN AND SYMPTOMS OF BERIBERI

- Irritability
- Headache
- Polyneuritis
- Emotional disturbance
- Reduce appetite
- Constipation
- Slow growth
- Vomiting
- cyanosis

PREVENTION

- Health education should be given on balance diet and thiamine rich foods.
- Adequate antenatal diet, treatment of prolonged illness and improvement of socioeconomic status.
- Breastfeeding mother should receive thiamine therapy.

Vitamin B2 (Riboflavin)

- It keeps skin eye and nervous system healthy as well as helping the body metabolism of carbohydrate protein and fats
- DEFICIENCY: Dermatitis, Skin Cracking.

❖ TREATMENT

- Promoting intake of riboflavin containing food.
- Administer riboflavin 3-10 mg orally or 2 mg im daily for 1 week followed by 10 mg orally for 3 weeks.

- Vitamin B5 (Niacin)
  - It helps produce energy from the food we eat as well as helping keeping the nervous and digestive system healthy.
  - DEFICIENCY: Pellagra (skin inflammation)
    - Dermatitis
    - Diarrhea
    - Dementia

PREVENTION

- Promotion of well balanced diet.
- Improvement of socioeconomic status and agriculture development will help to overcome the problem.
- Health education should be given

- Vitamin B6 (Pyridoxine)
  - It acts as a co-enzyme, which means it has chemical reaction take place and help to breakdown glycogen.
  - DEFICIENCY: Dermatitis, Convulsion, Depression, Epilepsy.

SIGN AND SYMPTOMS

- Nausea , Vomiting, Diarrhea , convulsion ,Irritability, Anemia And Failure To Thrive

TREATMENT

- Folic acid supplement
- Diet rich in folic acid.

- Vitamin B12 (CYANOCOBALAMIN)
  - It involves in making RBC and keeping the nervous system healthy and need for metabolism of fatty acid and amino acids and to synthesis the DNA in your cell
  - DEFICIENCY: Slow Growth , Numbness and tingling sensation.

PREVENTION MEASURES

- Adequate amount of animal food in daily balanced diet.
- Vitamin B12 supplementation to be done.

FAT SOLUBLE VITAMINS

- Vitamin A (Retinol)
  - Helps in maintaining eye sight
  - DEFICIENCY: Night Blindness, xerophthalmia, Bitots Spot ,keratomalacia.
  - NIGHT BLINDNESS: It Occurs Due To The Impairment Of Dark Adaptation. It Takes A Considerable Time In Adjusting Dim Light Or Darkness .
  - XEROPHTHALMIA : conjunctiva Becomes Dry, wrinkled With Dirty Brown Color.

- KERATOMALACIA : It Consists Of Softening, Necrosis And Ulceration Of Cornea.

- Vitamin A Deficiency Can Occur Due To Various Reasons:
- Diet Deficient In Vitamin.
- Decreased Absorption Due To Chronic Intestinal Disorders.
- Reduced Storage In The Liver In Liver Disease.

TREATMENT

- Encourage Diet Rice In Vitamin A.
- Children Of 6-11 Months Should Receive 100,000 IU Of Oral Vitamin A And Children Of 1-5 Years Should Receive 200,000 IU Of Vitamin A Every Six Months In The Target Areas.

- Vitamin D
  - It helps in bone development.
  - DEFICIENCY: Osteomalacia , Rickets.

TREATMENT

- Expose children to sun light.
- vitamin D supplements orally.

- Vitamin E
  - Helps in immune system and reproduction.
  - DEFICIENCY: Muscle dystrophy (weakness)

PREVENTIVE MEASURES

- Dietary improvement of mother and child.
- Creating awareness about the function of vitamin E and its deficiency conditions.

- Vitamin K
  - Helps in blood clotting.
  - DEFICIENCY: Hemophilia

MINERAL'S DISORDER

- Mineral metabolism disorders are abnormal level of minerals – either to much or too little in the blood.

- They are essential for the proper function of cell, tissue and organs.

DISORDERS

- CALCIUM DEFICIENCY : rickets, hypocalcemic tetanic with muscle cramps , numbness, tingling sensation of limbs.
- PHOSPHORUS DEFICIENCY : rickets in growing children.
- SODIUM DEFICIENCY : dehydration, weakness, dizziness, hypotension, anorexia.
- POTASSIUM DEFICIENCY : hypokalaemia (weakness, abdominal distension, diarrhoea, cardiac arrest, abnormal cardiac rhythm).

- IODINE DEFICIENCY : goitre, hypothyroidism, cretinism, dwarfism, impaired physical and mental growth.
- IRON DEFICIENCY : nutritional anaemia.

ANAEMIA

- Anemia is the deficiency in the no. of erythrocytes (red blood cells),the quantity of HB and the volume of packed RBCs (hemocrit).
- Anemia is the condition in which the HB concentration is lower than normal , reflects the presence of fewer than normal erythrocytes within the circulation. As a result ,the amount of O2 delivered to body tissue is also diminished.

ETIOLOGY:-

- -decreased erythrocyte production
- -decreased haemoglobin synthesis
  - iron deficiency
  - Thalassemias (decreased globin synthesis)

- Defective DNA synthesis
  - Cobalamin (vit-B12) deficiency
  - Folic acid deficiency

- Decreased number of erythrocyte precursors:

-aplastic anemia

-anemia of myeloproliferative disease(e.g.:-leukemia)

- Chemotherapy
- Blood loss

-acute

-trauma

-blood vessel rupture

- Chronic

-gastritis

-menstrual flow

-hemorrhoids

- Increased erythrocyte destruction
- Intrinsic

-abnormal haemoglobin (HB s-sickle cell anemia)

-enzyme deficiency

-membrane abnormalities(hereditary spherocytosis

TYPES OF ANEMIA

1. ANEMIA CAUSED BY DECREASED ERYTHROCYTE PRODUCTION:-

-Iron deficiency anemia

-thalassemia

-megaloblastic anemias

-Anemia of chronic disease

-Aplastic anemia

2-ANEMIA CAUSED BY BLOOD LOSS

3-ANEMIA CAUSED BY INCREASED ERYTHROCYTE DESTRUCTION

-Sickle cell anemia

IRON DEFICIENCY ANEMIA

- Iron deficiency anemia typically results when the intake of dietary iron is inadequate for HB synthesis.

ETIOLOGY:-

- Inadequate dietary intake
- Malabsorption
- Blood loss or hemolysis
- Menorrhagia (excessive menstrual bleeding)
- Inadequate iron supplement during pregnancy
- Gastrointestinal surgery and malabsorption
- GI bleeding (common causes of GI blood loss are peptic ulcer ,gastritis ,esophagitis ,hemorroids.

CLINICAL MANIFESTATION:-

- Pallor
- Glossitis (inflammation of the tongue)
- Cheilitis (inflammation of the lips)
- Brittle and ridge nails
- Angular cheilosis (ulceration of the corner of the mouth)
- Headache
- Parenthesis
- Burning sensation of tongue

DIAGNOSTIC FINDING:-

- History collection
- Physical examination
- Blood test
- Stool guaiac test(to see or detect the presence of blood in the stool)
- Endoscopy and colonoscopy to detect GI bleeding
- Bone marrow biopsy

MANAGEMENT:-

- The main goal of collaborative care of iron deficiency anemia is to treat the underlying disease that is causing reduced intake (e.g.:- malnutrition)or absorption of iron.

- Foods are good source of irons

- Orally or occasionally parentral iron supplement are used.

- The daily dosage should provide 150-200mg,three or four daily doses.

- Iron is absorbed best from the duodenum and proximal jejunum. Therefore enteric coated or sustained release capsules ,which release iron further down in the GI tract , the counter productive and expensive.

- Iron is the best absorbed as ferrous sulfate in an acidic environment. Iron should be taken about an hour before meals.
- GI side effects of iron administration are constipation ,heartburn and diarrhea ,so started with stool softner and laxative.

THALASSEMIA:-

- The thalassemia are a group of hereditary anemias characterised by hypochromia (an abnormal decrease in the hemoglobin content of erythrocytes) ,extreme microcytosis (smaller then normal erythrocytes),destruction of blood elements (hemolysis) and variable degree of anemia.

ETIOLOGY:-

- Defective synthesis of the hemoglobin chain the production of one or more globulin chains within the hemoglobin molecule is reduced.

- It is due to an absent or reduced in alpha-thalassemia and beta globin chain are absent or reduced in beta thalassemia.

CLIINICAL MANIFESTATION

- Pale
- Tachycardia
- Glossitis(other symptoms like anemias)
- The symptoms develop in childhood by 2yrs of age and can cause growth and development deficit.
- Splenomegaly
- Hapatomegaly
- Jaundice

DIAGNOSTIC FINDING:-

- History collection
- Physical examination
- CBC COUNT
- Hb test:-measure the types of Hb in blood sample. People who have thalessemia have problem with the alpha or beta globin protein chain of Hb.

MANAGEMENT:-

- No specific drugs or diet therapies are effective in treating thalassemia.
- Thalassemia minor requires no treatment because the body adopts to the reduction of normal Hb.
- Zinc supplement may be needed (it is reduced with the chelation therapy.
- Blood transfusion or exchange transfusion in conjunctive with I/V deferoxamine (a chelating agents that binds to iron) to reduce iron overloading that occurs with chronic transfusion therapy.

MEGALOBLASTIC ANEMIA

- Are a group of disorders caused by impaired DNA synthesis and characterized by the presence of large RBCs.
- The RBCs are large (macrocytic) and abnormal and referred to as “MEGABLASTS”.
- Macrocytic RBCs are easily destroyed because they have fragile cell membranes.

TWO COMMON FORMS OF MEGALOBLASTIC ANEMIA ARE:-

- -COBALAMIN DEICIENCY(VIT-B12)
- -FOLIC ACID DEFICIENCY(VIT-B9)

ETIOLOGY:-

- GI surgery such as gastrostomy
- Small bowel resection involving the ileum
- ileitis
- Chronic atrophic gastritis(mucosal atrophy)

CLINICAL MANIFESTATION:-

- Hypoxia
- Sore tongue
- Anorexia
- Nausea , vomiting
- Abdominal pain

GI MANIFESTATION

- Weakness
- Paresthesia of the feet and hand
- Ataxia
- Muscle weakness
- Impaired thought process ranging from confusion to dementia

neuro muscular manifestation

DIAGNOSTIC FINDING:-

- History collection
- Physical examination
- RBC count , including morphology
- Schilling test:- in which the patient receives a small dose of radioactive vitamin-B12,followed in a few hours by a large , nonradioactive vit-b12 parentral dose (this aids in renal excretion of the radioactive dose). If the dose (oral 0vitamin is absorbed, more than 8% will be excreted in the urine within 24 hours, therefore ,if no radioactivity is present in the urine (i.e. the radioactive vit –B12 stays within the GI tract), the cause is GI Malabsorption of the vit-B12.

MANAGEMENT:-

- Regardless of how much is ingested ,the patient is not able to absorb cobalamin if intrinsic factor is lacking or if there is impaired absorption in the ileum

- So ,the parentral (cyanocobalamin or hydroxocobalamin) or intranasal (nascobal) administration of cobalamin IM daily for 2 weeks then weekly until hemocrit is normal.

- High dose oral coalmine and sublingual coalmine are also available.
- Without coalmine administration ,these individuals will die in 1 to 3 yrs.

FOLIC ACID DEFICIENCY:-

- Folic acid deficiency also causes megaloblastic anemia.

ETIOLOGY:-

- Poor nutrition
- Mal absorption syndrome(small bowel disorders)
- Alcohol abuse and anorexia
- Drugs that impede the absorption like antiseizure drugs(e.g.:0phenobarbital)
- Hemodialysis patients because folic acid is lost during dialysis.

CLINICAL MANIFESTATION:-

- GI disturbance includes:-

-dyspepsia(accompanied
by bloating ,belching ,nausea heartburn)

-Smooth beefy red tongue

-absence of neurologic problems is an important diagnostic findings

-this lack of neurologic involvement differentiates folic acid deficiency from cobalamin deficiency.

DIAGNOSTIC FINDING:-

- HISTORY COLLECTION
- PHYSICAL EXAMINATION
- RBC count ,including morphology
- Serum folate level (normal 3-25mg/dl)

MANAGEMENT:-

- Folic acid deficiency is treated by replacement therapy.
- The usual dose is 1mg per day by mouth.
- In malabsorption states up to 5mg per day may be required.
- The patient should be encouraged to eat food containing large amount of folic acid.

ANEMIA OF CHRONIC DISEASE:-

- The term “anemia of chronic disease “ is a misnomer in that only the chronic disease of inflammation , infection and malignancy cause this type of anemia.
- anemia of chronic disease is associated with an underproduction of RBCs and mild shortening of RBC survival.

ETIOLOGY:-

- RENAL DISEASE
- RADIATION AND CHEMOTHERAPY
- HIV
- HEPATITIS
- MALARIA AND BLEEDING EPISODES

MANAGEMENT:-

- Anemia of chronic disease must be recognized and differentiated from anemia.
- The best treatment of anemia of chronic disease is correction of the underlying disorder.
- If anemia severe ,blood transfusion may be indicated ,but not recommended for long term treatment.
- Intravenous iron should administered.

APLASTIC ANEMIA:-

- Is the disease in which the patient has peripheral blood pancylopenia (decrease of all blood cell types-RBCs , white blood cells, platelets and hypocellular bone marrow.

or

Aplastic anemia is a rare disease caused by a decrease in or damage to marrow stem cells, damage to the microenvironment of the marrow with fat.

ETIOLOGY:-

- The etiological classification for aplastic anemia is divided into two groups:-

- 1-CONGENITAL :-Caused by chromosomal alteration ,approximately 30% of the aplastic anemia that appear in childhood are inherited.

- 2-ACQUIRED APASTIC ANEMIA:-Results from exposure to ionizing radiation ,chemical agent (e.g:-benzene,DDT,alcohol),viral or bacterial infection, prescribed medication(e.g.:-antiseizure agents).Approximately 70% of the acquired aplastic anemia are idiopathic.

CLINICAL MANIFESTATION

- Fatigue , dyspnea
- Tachycardia ,increased pulse rate
- Systolic murmur
- Angina
- The patient with neutrophil is susceptible to infection.
- Thrombocytopenia is manifested

by a predisposition to bleeding

(e.g.:-petechiae, ecchymosis ,

epistaxis)

DIAGNOSTIC FINDING:-

- History collection
- Physical examination
- CBC, including morphology
- Serum iron and total iron binding capacity
- Bone marrow biopsy :-finding are especially important in aplastic anemia because the marrow is hypocellular with increased yellow marrow (fat content).

MANAGEMENT:-

- In severe case hematopoietic stem cell transplant (HSCT)and immunosuppressive therapy with antithymocyte globulin (ATG) and cyclosporine or high dose cyclophosphamide have improved outcomes significantly.

- ATG is a horse serum that contains polyclonal antibodies against human t-cells.

- The treatment of choice for adults less than 45 yrs of age who do not respond to the immunosuppressive therapy and who have a human leukocyte antigen (HLA)-matched donor is HSCT. The best results occur in a younger patient who has not previous blood transfusion.

ANEMIA CAUSED BY BLOOD LOSS:-

- Anemia resulting from blood loss may be caused by either acute or chronic blood loss.

1-ACUTE BLOOD LOSS:-

It occur as a result of sudden hemorrhage.

ETIOLOGY:-

- Trauma
- Complication of surgery
- Condition or disease that disrupt vascular integrity

CHRONIC BLOOD LOSS:-

- The source of chronic blood loss are similar to those of iron deficiency anemia.

ETIOLOGY:-

-bleeding ulcer

-hemorroids

-menstrual or
postmenstrual blood
loss

-esophagitis

-diverticuli

-the effect of chronic blood loss are usually related to the depletion of iron store and are usually considered as iron deficiency anemia

DIAGNOSTIC FINDING:-

- History collection
- Physical examination
- CBC
- When blood volume loss is sudden ,plasma volume has yet had a chance to increase , the loss of RBCs is not reflected in lab data and values may seen normal or high for 2-3 days.
- Once the plasma replaced by endogenous and exogenous means ,the RBC mass is less concentrated .At this time RBC , Hb and hematocrit levels are low.

MANAGEMENT:-

- Replacing blood volume to prevent shock.
- Identifying the source of the hemorrhage and stopping the blood loss.
- I/V fluids used in emergencies include dextran,RL,albumin.
- Once volume replacement is established, attention can be directed to correcting the RBC loss.
- Oral or parentral iron

preparations are administratered.

SICKLE CELL ANEMIA:-

- Is a group of inherited ,autosomal recessive disorders characterized by the presence of an abnormal form of Hb in the erythrocyte.
- This abnormal Hb , hemoglobin s cause the erythrocyte to stiffen and elongate taking on a sickle shape in response to low O2 levels.

ETIOLOGY:-

- Autosomal recessive disorder
- Mutation in beta-globin gene located on chromosome 11

INCIDENCE:-

- Affects people of American ,Caribbean , Arabian and INDIAN
- Affects 8 of every 1,00,000 people.

CLINICAL MANIFESTATION:-

- Pallor of mucous membrane
- Fatigue
- Decreased exercise tolerance

- Sickle cell crisis –is a severe pain because of ischemia of tissue

- SICKLING EPISODES:-are most commonly triggered by low O2 tension in the blood. Hypoxia or deoxygenation of the RBCs can be caused by viral or bacterial infection,surgery,physical stress accompanied by objective clinical signs such as fever,swelling, tenderness,tachypnea,hypertension, nausea

DIAGNOSTIC FINDINGS:-

- History collection
- Physical examination
- Electrophoresis of hemoglobin and sickling screening test are more commonly used.
- DNA testing is available but costly.
- Skeletal x-rays :-to check bone and joint deformity
- MRI:-To diagnose a stroke caused by blocked cerebral vessels from sickle cells.
- Doppler studies:- to assess the deep vein thrombosis.

MANAGEMENT:-

- There is no specific treatment for this disease.
- The patient should be taught to avoid high attitudes, maintain adequate intake and treat infection.
- Sickle cell crises may requires hospitalization.
- O2 may be administered to treat hypoxia and control sickling.
- Fluid and electrolytes are administered to reduce blood viscosity and maintain renal function.

- Non-steroidal anti-inflammatory agent ,local anesthetic ,nerve blocks, acupuncture may be used.
- Transfusion therapy is indicated when an aplastic crises occur.
- Transplantation of (HSCT) hematopoietic stem cell transplantation.

MACRONUTRIENTS DEFICIENCY

A chemical element that is essential in relatively large amount to the growth and healthy of a living organism. Micronutrients includes:

- Carbohydrates: It is important dietary nutrients and source of caloric energy.
  - Deficiency: Ketoacidosis
  - Source: Carrot ,Oats ,Brown Rice, Apple.
- Fats: Fatty acid deficiency is rare, occurring most in infants fed diet deficiency.
  - Deficiency: dermatitis, Osteopenia.
- Protein: Protein are broken down to there consistent amino acid which are used to form enzyme, hormones and cell protein.
  - Deficiency: Kwashiorkor, Muscle wasting, Edema.

NUTRITIONAL ASSESSMENT

- RISK FACTOR
  - There are numerous risk factors for poor nutritional status, including major, burns, sepsis, GI disorders . Additional information learned through a careful medical history, can also suggest possible risk factors for malnutrition.
    - Age <18yrs or > 65 yrs (increased risk age > 75yrs)
    - Homelessness, limited access to food.
    - Limited capacity for oral intake (dysphasia, stomatitis)
    - Increased metabolic demands: extensive burns, trauma, infection.

DIET HISTORY

- A detailed diet history provides insight into a patient baseline nutritional status and may defect subclinical nutrient deficiencies.
- Assessment includes regarding chewing or swallowing problems, avoidance of eating related to abdominal pain, changes in appetite.

MEDICAL HISTORY

- A review of past medical history includes indentifying existence of condition resulting in increased metabolic need, altered gastro-intestinal function and absorptive capacity
- Chronic disease states and level of physical activity.

NATIONAL NUTRITIONAL PROGRAMMER IN INDIA

- Integrated child development services scheme. (ICDS)
- Balwaldi nutrition programme.
- Special nutrition programme.
- National nutritional anemia prophylaxis programme.
- National prophylaxis programme for prevention of blindness due to vitamin A deficiency.
- National iodine deficiency disorder control programme.
- Mid-day meal programme.

BALWADI NUTRITION PROGRAMME

- This was started in 1970 under the department of social welfare.
- Beneficiary group: pre school children 3-5 years of age.

SPECIAL NUTRITION PROGRAMME

- Started in 1970 by ministry of school welfare.
- Operation in urban slums, tribal and backward rural areas.
- Beneficiary group: Preschool children, Pregnant and Lactating mother

NATIONAL PROPHYLAXIS PROGRAMME AGAINST NUTRITIONAL BLINDNESS DUE TO VITAMIN A DEFICIENCY

- Launched in 1970 as a centrally sponsored scheme by ministry of health and family welfare.
- Target group = all children 1-3 years of age.

NATIONAL IODINE DEFICIENCY DISORDER CONTROL PROGRAMME

- The beginning- kanga valley study (1956-72)
- National Goiter control programme launched in 1962 at the end of 2nd 5 year plan by ministry of H & FW G.O.I
- Focus on use of Iodized salt.

INTEGRATED CHILD DEVELOPMENT SERVICES (I.C.D.S) SCHEME

- Initiated October 2 1975, in 33 CD block under 5th year plan
- Worlds largest programme for early childhood development.
- Services:
  - Supplementary nutrition
  - Non-formal pre school education
  - Immunization and health check up
  - Referral services
  - Nutrition and health education
- BENEFICIARY:
  - Children < 6yrs
  - Pregnant and lactating women
  - Women in reproductive age group

NATIONAL NUTRITIONAL ANEMIA PROPHYLAXIS PROGRAMME

- Launched during 4th 5th plan in 1970 by ministry of health and family welfare.
- Prevention of nutritional anemia in mothers and children.

MID-DAY MEAL PROGRAMME

- First started in Tamil Nadu.
- Also known as school lunch programme.
- Programme in operation since 1961 under ministry of education.
- Aim: to provide at least one nourishing meal to school going children per day.

CONCLUSION

- Malnutrition is an barrier to development and its presence indicates that basic physiological need have not been met. What is observed on malnutrition is not only the result of insufficient food, but also a consequence of other condition such as poor water supply and sanitation and a high prevalence of disease.


THANK YOU


Any questions?