

الكلية : الطب

القسم او الفرع :طب الاطفال وحديثي الولادة

المرحلة: الخامسة

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اسم المادة باللغة العربية : طب الاطفال وحديثي الولادة

اسم المادة باللغة الإنكليزية : طب الاطفال وحديثي الولادة

اسم المحاضرة الأولى باللغة العربية: سوء التغذية

اسم المحاضرة الأولى باللغة الإنكليزية : **Malnutrition**

Malnutrition

Malnutrition; is a general term for the medical condition caused by an improper or insufficient diet.

Undernutrition: Structural and functional changes due to inadequate intake of nutrients and energy sources.

#Primary Malnutrition: resulting from inadequate food intake.

Secondary Malnutrition: resulting from inadequate absorption, abnormal metabolism and increased nutrient losses.

Types of undernutrition

--**macronutrient malnutrition** ;(protein energy malnutrition PEM) ; deficiency of protein ,carbohydrates or fat .

--**micronutrient malnutrition**; deficiency of vitamins and minerals .

-wellcome classification

| Wt\age + , - | No edema | edema |
|-----------------|--------------------|--------------------|
| 60 -80 % | Underweight | kwashiorkor |
| Less than 60% | marasmus | Marasmic |

| | | |
|--|--|--------------------|
| | | kwashiorkor |
|--|--|--------------------|

-who classification

| | Normal | Mild | Moderate | Severe |
|--------------------------------|----------------------|--------------------|-------------------|---------------|
| Weight\height (wasting) | 120_90 % +2 _ -1 | 89_80% -1 _ -2 | 79_70% -2 _ -3 | <70 % < -3 |
| Height \age (stunting) | 110 _95 % +2 _ -1 | 94 _90% -1 _ -2 | 89 _85% -2 _-3 | <80 % < -3 |
| Edema | _ ve | _ ve | _ ve | + ve |

- **Wt\ht** ; every 10% of the standard wt\ht =1 Z score

Ht\age ;every 5% of the standard ht\age =1 Z score .

-**wt\age** ;first 10% of the standard wt\age =1 Z score , then every additional 15% of the standard wt\age =1 Z score .

Kwashiorkor

Deficiency of protein with relatively adequate energy intake. **60 -80 % of the expected wt\age with edema .**

* **Failure of growth** ,the muscles are wasted

* **edema**: pitting, bilateral including lower extremities.(may masks the failure to gain weight)

- **Hair:** becomes fine, straight and often sparse. Children with long straight black hair may show a pale band across the hair, the 'flag sign'.
- **Skin:** pigmentation, desquamation and ulceration. A severe case may look like an extensive burn. The legs, buttocks and perineum are most frequently involved, but any region may be affected.
- the **Liver may be enlarged** due to fatty liver .
- **Gastrointestinal system:** anorexia is usually present and sometimes vomiting.

diarrhea may be due to impaired secretion of digestive enzymes, to intestinal mucosal atrophy or to an intestinal infection.

- **Anaemia .**
- **Associated vitamin deficiencies.**

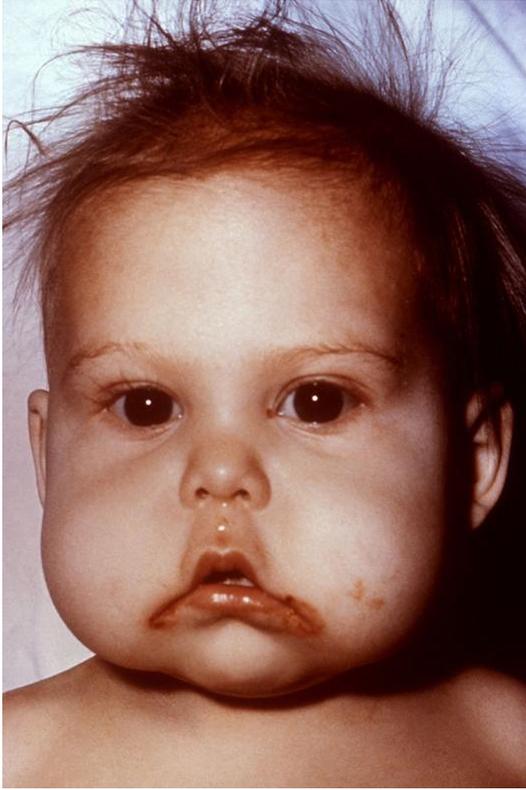
Causes ;

-insufficient intake /of protein of good biological value, poverty, starvation, inadequate intake

-impaired absorption / chronic diarrhea , celiac disease ,obstructive jaundice .

-abnormal protein loss /; nephrotic syndrome , protein losing enteropathy ,skin burn ,chronic bleeding .

-failure of protein synthesis / chronic liver disease.



Nutritional marasmus

is the most common form of PEM, Weight for age < 60% expected with No edema

#. Retardation of growth , Wasting of muscles & loss of subcutaneous fat.

Appetite is usually preserved or enhanced & the liver is usually normal, with no fatty infiltration.

Episodes of hypothermia and fasting hypoglycemia are common.

Emaciated thin, flaccid skin ,fat and muscle tissue grossly reduced, prominence spine and ribs.

Behavior: alert and irritable.

Normal hair.

#. Pot belly: the child's abdomen protruded, because the muscles of the abdominal wall are wasted and weak

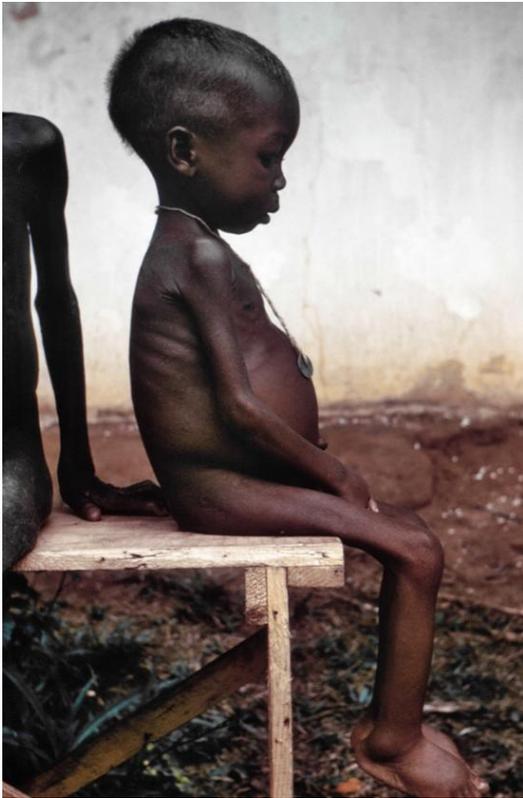
Causes ;

starvation , disturbed maternal child relationship .

#chronic diarrhea and vomiting.

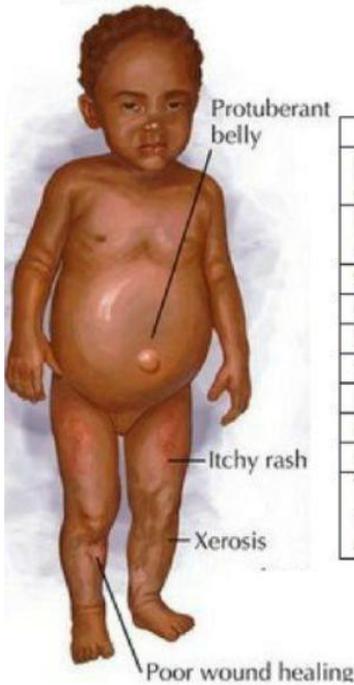
#metabolic abnormalities.

#congenital G.I. tract anomalies ; pyloric stenosis ,esophageal atresia



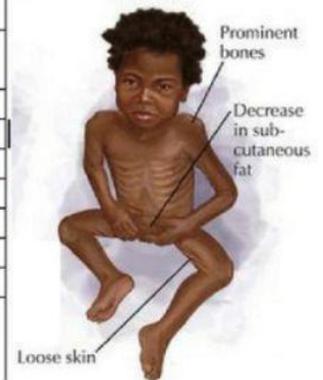
10 Differences between Kwashiorkor and Marasmus

Kwashiorkor



| Kwashiorkor | Marasmus |
|---|--|
| It develops in children whose diets are deficient of protein. | It is due to deficiency of proteins and calories. |
| It occurs in children between 6 months and 3 years of age. | It is common in infants under 1 year of age. |
| Subcutaneous fat is preserved. | Subcutaneous fat is not preserved. |
| Oedema is present. | Oedema is absent |
| Enlarged fatty liver. | No fatty liver. |
| Ribs are not very prominent. | Ribs become very prominent. |
| Lethargic | Alert and irritable. |
| Muscle wasting mild or absent. | Severe muscle wasting |
| Poor appetite. | Voracious feeder. |
| The person suffering from Kwashiorkor needs adequate amounts of proteins. | The person suffering from Marasmus needs adequate amount of protein, fats and carbohydrates. |

Marasmus



Kwashiorkor vs Marasmus

TREATMENT ;

Phases of Management of severe malnutrition is best divided into three phases.

Initial Phase

Following clinical evaluation, the Initial Phase (days 1-7)

The principle tasks are:

- To treat or prevent hypoglycemia and hypothermia;
- To treat or prevent dehydration and restore electrolyte balance;
- To start feeding to the child
- To treat infection;
- To identify and treat any other problems, including vitamin deficiency, severe anemia and heart failure

* Severely malnourished patients do not tolerate the usual amounts of dietary protein, fat, and sodium, and require a diet low in these component and in osmolality, **but high in carbohydrate. Start with 80 – 100 kcal/kg/day of actual weight and increase gradually .start with formula 75 (75 kcal/100 ml) .** At each feed the food should be offered by mouth, after which the remainder is given by NG tube.

*avoid giving iron because will increase the liberation of free radicals.

Rehabilitation Phase ;

The second Phase (weeks 2-6)

_ To encourage the child to eat as much as possible. Or (re-initiate breastfeeding) , and to stimulate emotional and physical development;

Started when the appetite and general conditions improved , so , **shift to formula 100(100kcal\100ml)** .give the other vitamins and add iron .stop the F100 and shift to normal diet when the child catches up 90% of his weight \height .

Follow-Up Phase ;

to prevent the recurrence of malnutrition .

Refeeding syndrome

It occur if high-energy feeding is started too soon or vigorously, and it may lead to sudden death with signs of heart failure.The refeeding syndrome onset is usually 24-48 hr after the start of high energy feeding and is characterized by breathlessness, rapid pulse, increased venous pressure, rapid enlargement of the liver, and watery diarrhea

Also there is Hypophosphatemia, hypokalemia,hypomagnesemia, hypernatremia withThiamin deficiency.

Note / **give broad spectrum antibiotics for 10 days even if the child has no signs of infection

Note / **malnourished child has low body potassium and high sodium . So if mildly or moderately dehydrated give **resomal solution** (contain lower NA and higher k) .
if severely dehydrated , give ivf .

B/ VITAMIN AND MINERAL DEFICIENCIES

Micronutrients include vitamins and trace elements

WATER- SOLUBLE VITAMINS;

Ascorbic Acid (VIT C)

. The disease may occur if infants are fed unsupplemented cow's milk in the first year of life or if the diet is devoid of fruits and vegetables .

Infantile scurvy is manifested by **irritability, bone tenderness with swelling.. Sub periosteal hemorrhage, bleeding gums and loosening of teeth , petechiae , bleeding of skin and mucous membranes and Anemia (secondary to ;1- bleeding,2- decreased iron absorption, or 3-abnormal folate metabolism is also seen in chronic scurvy).**

X ray shows thin cortex with generalized osteopenia , white metaphysical line (frankel line) with area of bone resorption under that line , sub periosteal hemorrhage then calcification

Treatment ;100-200 mg\day vit c orally or iv , give vit c rich food



B Vitamins; most of B vitamins must be replaced daily since any excess is excreted in the urine.

Thiamine (Vitamin B1)

Deficiency causes (beriberi)

Functions as a coenzyme in biochemical reactions related to carbohydrate metabolism. Thiamine is lost during milk pasteurization and sterilization.

Infantile beriberi occurs between 1 and 4 months of age in breastfed infants whose mothers have a thiamine deficiency,

Affects mainly the CVS And CNS. Anorexia, apathy, vomiting, restlessness, and pallor progress to dyspnea, cyanosis, and death from heart failure. Infants with beriberi have a characteristic aphonic cry (they appear to be crying, but no sound is uttered). Other signs include peripheral neuropathy and paresthesias.

Treatment ;10mg im thiamin \day IM for children ,50 mg \day for adults. continue on thiamin rich diet .and treat with anti failure if heart failure is present .

Riboflavin Vitamin B2 ;

deficiency of riboflavin affects glucose, fatty acid, and amino acid metabolism. Riboflavin is heat stable but destroyed by exposure to light . **Ariboflavinosis is characterized by an angular stomatitis; glossitis; cheilosis; seborrheic dermatitis around the nose and mouth; and eye changes that include reduced tearing, photophobia, and cataracts.**

Treatment;3-10 mg\day orally +well balanced diet .

Niacin; B3(niacinamide)

Important for the synthesis of (NAD) and (NADP) used for glycolysis and electron transfer .

Pellagra, or niacin deficiency (Diarrhea , Dysphagia , Dermatitis ,Dementia)

Treatment ;50-300 mg\day niacin orally .if severe 100 mg \day IM .and avoid sunlight .

Vitamin B6

The metabolic functions of vitamin B6 include conversion of tryptophan to niacin and serotonin, metabolic reactions in the brain, carbohydrate metabolism, immune development, and the biosynthesis of heme and prostaglandins. Goat's milk is deficient in vitamin B6.

Deficiency of vitamin B6 in children results in ; anemia ,vomiting, diarrhea, dermatitis , peripheral neuritis, and seizures.

Children receiving isoniazid or penicillamine may require additional vitamin B6 because the drug binds to the vitamin .

Note/ Newborns of mothers with history of taking large doses of B6 during pregnancy must receive 10 mg\day orally after birth for several weeks , to prevent convulsions . Convulsions weather B6 dependent or deficient ,must be treated by 100 mg pyridoxine IM .& keep on well-balanced diet to prevent recurrence .

Folic acid

*Food sources include/ vegetables, liver, whole grains;

*** Folate deficiency, characterized by ; hyper-segmented neutrophils, macrocytic anemia, neutropenia, thrombocytopenia and glossitis.**

Risk factors for Deficiency;

* Low dietary intake, malabsorption, or drug interactions.

* Infants require high amounts but have scant stores of folate in the newborn period.

* Heat-sterilizing home-prepared formula can decrease the folate content by half.

*goat's milk are low in folate.

*Patients with chronic hemolysis (sickle cell anemia, thalassemia) may require extra folate to avoid deficiency because of the relatively high requirement of the vitamin to support erythropoiesis.

Because closure of the neural tube occurs before usual recognition of pregnancy, all women of reproductive age are recommended to have a folate intake of at least 400 µg/day as prophylaxis.

Vitamin B12(cobalamin)

*Vitamin B12 is essential for protein biosynthesis and DNA synthesis .

*The cobalamin is efficiently absorbed from the distal ileum.

*Dietary sources of the vitamin are animal products only (meat ,egg , milk) ,so ,Strict vegetarians should take a vitamin B12 supplement.

* Pernicious anemia ,Gastric or intestinal resection and small bowel bacterial overgrowth also cause vitamin B12 deficiency. *Exclusively breastfed infants ingest adequate vitamin B12 (unless the mother is a strict vegetarian without supplementation).

* **Deficiency of B12, presented as hyper-segmented neutrophils and and megaloblastic anemia. also causes neurologic manifestations, including depression, peripheral neuropathy, posterior spinal column signs, dementia, and eventual coma**

FAT-SOLUBLE VITAMINS

Vitamin A

The clinical manifestations of vitamin A deficiency in humans appear as **xerophthalmia. The earliest symptom is night blindness, which is followed by xerosis of the conjunctiva and cornea ,bitot spots (silver gray spots on the bulbar sclera)**.

* Clinical and subclinical vitamin A deficiencies are associated with increased risk of infection.

Treatment ;

for latent deficiency ; 1500 ug \day for xerophthalmia ; 1500 ug \day orally for 5 days then daily IM injection of 7500 ug\day till recovery .

*Hypervitaminosis A also has serious sequelae, including headaches, pseudotumor cerebri, and hepatotoxicity.

Vitamin E

Acts as anti oxidant , and for nucleic acid metabolism .

Deficiency Caused by; malabsorption , high unsaturated fatty acid diet ,prematurity .

*** progressive sensory and motor neuropathy develops; anemia cerebellar ataxia , the first sign of deficiency is loss of deep tendon reflexes.**

Deficient preterm infants at 1 to 2 months of age have hemolytic anemia , retinopathy ,increased platelet count and adhesiveness so prone for intravascular thrombosis .

Treatment ;for premature 15 -25 iu\day vit E orally .

For children 100 -200 Iu \day .

Vitamin K

Plasma factors II (prothrombin), VII, IX, and X in the cascade of blood coagulation factors depend on vitamin K.

Other vitamin K–dependent proteins include proteins C, S .

___ **Vitamin K deficiency has been observed in subjects with ;** impaired fat absorption caused by obstructive jaundice, pancreatic

insufficiency, celiac disease; prolonged use of antibiotics that change intestinal flora ,more common among breastfed infants .

, occurs in the first few weeks of life(rare in infants who receive prophylactic intramuscular vitamin K on the first day of life).

Hemorrhagic disease of the newborn usually is marked by generalized ecchymoses, gastrointestinal hemorrhage, or bleeding from a circumcision or umbilical stump; intracranial hemorrhage can occur .

Prophylaxis ;parenteral vitamin K (1 mg) is recommended to be given to all newborns shortly after birth.

Treatment ; 5mg \day IM .

IF bleeding is severe or the patient has liver disease , give fresh blood or fresh frozen plasma .

Vitamin D;

Group of sterols supplied to the body either from the diet (d3,cholecalciferol ,from animals) and (d2 , ergocalciferol) or from the skin by direct uv light exposure .

Regulate absorption of calcium and phosphorous from the intestine , and their deposition in bones .

Sources ; margarine , fish liver oil and by the skin through the sun light exposure to uv light type B .

Vitamin d deficiency and the interference with its metabolism will cause rickets.

Daily requirement; 400 iu/day (10 ug) for infant and children.