

**College of Medicine/ University of Diyala
Paediatrics division**

NUTRITION

by

Najdat S. Mahmood

Nutritional intakes of infants, children, and adolescents should provide for maintenance of current weight and support the normal growth and development. The infancy growth period is rapid, critical for Neuro-cognitive development, and has the highest energy and nutrient requirements relative to body size compared with other periods of growth (twice that of an adult).

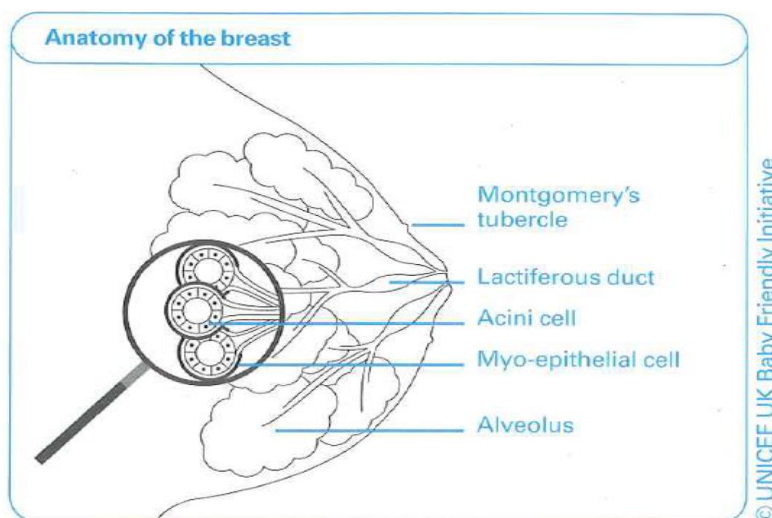
BREAST-FEEDING

Breastfeeding has documented short- and long-term medical and neuro-developmental advantages and rare contraindications. Thus the decision to breastfeed should be considered a public health issue and not only a lifestyle choice.

The AAP (American Academy of Pediatrics) and the WHO recommend that infants should be exclusively breastfed or given breast milk for 6 months. Breastfeeding should be continued with the introduction of complementary foods for 1 year or longer, as desired by mother and infant.

Anatomy of the breast

It is the largest exocrine gland, specialized for secretion of milk. Each breast consists of 16-18 lobes embedded in fat & connective tissue in the chest wall, each lobe is consisted of thousands of secreting units called alveoli.



Mechanism of milk production

Lactogenesis starts in the 5th mo of gestation under the effect of prolactin hormone, but full lactation during pregnancy is prevented by the elevated maternal progesterone level (which decreases after labor) that antagonizes the prolactin action.

Breast milk secretion in the alveoli is directed after birth by neurohormonal mechanism.

- milk production reflex: prolactin
- milk ejection or let down reflex: oxytocin

Factors affecting breast milk production

- Certain drugs, e.g., chlorpromazine & metaclopramide stimulate the prolactin secretion, while bromocriptin suppress its secretion & reduce milk production.
- Maternal status, e.g. tension, pain, fatigue & emotional distress, leads to failure of let down reflex & decrease of breast milk production.

Breast Milk Composition

It is isotonic with plasma & composed of protein, fat & lactose. During the 1st postpartum days, lactation started with colostrum, it is higher than the mature breast milk in sodium, protein, & cells & is lower in fat, lactose, & potassium. Then, during the 1st 2-3 wk of lactation, the protein continues to decrease, while the fat & lactose increase, so passing the transitional period to reach the mature milk.

Milk from the mother whose diet is sufficient and properly balanced will supply all the necessary nutrients, but the following to be recommended:

- If the water supply is not adequately **fluoridated** (≤ 0.3 ppm), the breast-fed infant should receive it.
- Begin daily oral vitamin D drops (400 IU) at hospital discharge post delivery.
- The **iron**:
- The **vitamin K**:

Establishing & Maintaining the Milk Supply

The most satisfactory stimulus to the secretion of human milk is early (during the 1st few days after birth), regular, and complete emptying of the breasts.

- Appropriate care for **sore nipples** should be instituted before severe pain from abrasions and cracking develops. Because if nipple is painful, the milk-ejection reflex may be delayed and this will makes the infant vigorously feeding, which further injures the nipple and areola area.

Treatment: Exposing the nipples to air; applying pure lanolin; avoiding soap & alcohol; changing disposable nursing pads frequently; feeding more frequently; manually expressing milk; & nursing in different positions are recommended. If

- **Retracted and/or inverted nipples** are not contraindications to breastfeeding, it usually benefits from daily manual breast-pump suction during the later weeks of pregnancy.

- To reduce **engorgement**, the breasts should be softened prior to infant feeding with a combination of hot compresses and expression of milk. Between feedings a supportive bra should be worn, cold compresses applied, and oral non-steroidal anti-inflammatory medications administered.

- **Mastitis** occurs (unilateral, localized warmth, tenderness, edema, and erythema with breast pain, myalgia, fever, fatigue, nausea, vomiting, and headache). Organisms include mainly *Staphylococcus aureus*. Oral antibiotics and analgesics with the promotion of breastfeeding or emptying of the affected breast.

The 1st 2 weeks after birth are important for establishing breastfeeding.

Determining the Adequacy of Breast Feeding

The milk supply is sufficient if the infant is:

- 1- Satisfied after each nursing.
- 2- Sleeps 2–4 hour between feedings especially in early infancy, unless awakened & crying due to other cause.
- 3- Gains weight adequately (start gaining weight by the end of the 2nd week).
- 4- Urinate a adequately.

Advantages of Breastfeeding vs Formula-Feeding.

- 1- Human milk is uniquely adapted to the infant's needs.
- 2- Breast milk is always available at the proper temperature & requires no preparation time, in addition to its economic benefits.
- 3- Breastfeeding is associated with fewer feeding difficulties incident to allergy and/or intolerance to bovine milk.
- 4- Decreasing incidence of infections, this due to many causes:
 - a- It is fresh and free of contaminating bacteria.
 - b- Human milk contains bacterial and viral antibodies, including secretory IgA.
 - c- Macrophages, lactoferrin, low pH, bile salt- stimulated lipase in human milk.
- 5- It reduces the liability of sudden infant death syndrome & later diabetes mellitus.
- 7- Psychological advantages of breastfeeding for both mother and infant.

For mothers, it enhances the involution of the uterus through the release of oxytocin, method of contraception, & decrease the incidence of breast cancer.

Disadvantages of Breast Milk

- 1- It is not possible to see how much the baby takes.
- 2- It needs a discrete place to expose the breast for feeding.
- 3- Women wishing to return to work will find it is difficult to fit with it.
- 4- Some times, breastfeeding may be impossible due to the medical illness of the mother.

Relative and absolute contraindications of breast feeding

1- Some inborn errors of metabolism of the baby: due to breast milk constituents, e.g. galactosemia.

2- Maternal Infections:

HIV: In the United States, breastfeeding is Contraindicated In other settings, health risks of not breastfeeding must be weighed against the risk of transmitting virus to the infant

Tuberculosis: Breastfeeding is contraindicated until completing approximately 2 wk of appropriate maternal therapy.

Varicella-zoster: Infant should not have direct contact to active lesions & infant should receive immune globulin

Herpes simplex: Breastfeeding is contraindicated with active herpetic lesions of the breast.

CMV: May be found in milk of mothers who are CMV seropositive Transmission through human milk causing symptomatic illness in term infants is uncommon

Hepatitis B: Infants routinely receive hepatitis B immune globulin and hepatitis B vaccine if mother is HbsAg positive. No delay in initiation of breastfeeding is required.

Hepatitis C: Breast-feeding is not contraindicated.

3- Chemotherapy and radiopharmaceuticals: Breastfeeding is generally contraindicated

FORMULA-FEEDING

Indications of Bottle Feeding

In healthy infants, bottle feeding can be used by one of the following 3 method:

a- Complementary feeding:

Where breast feeds are completed by bottle feeds. It is indicated when breast milk is *insufficient* for normal growth (scanty breast milk secretion). In this case, the breast milk should be given first, then the feed is completed by the bottle.

b- Supplementary feeding:

Where some breast feeds are replaced by bottle feeds. It is indicated in two conditions; *working mother* (where the mother is absent part of the day) & *twin delivery* (where the breast milk is not enough to feed both babies).

c- Substitutive feeding:

Where breastfeeding is completely replaced by bottle feeding. It is indicated in three conditions; *absent* breast milk secretion, *chronically sick mother* & mothers who are *unwilling* to breast feed their babies. In the last condition, the reason should be explored & the advantages of breast feeding should be explained. When she insists, encourage her on artificial feeding & do not let her to feel guilty.

Preparation of Milk

The dried milk should be properly reconstituted to provide the proper concentration. Small scoop of milk (4 gm) needs 1 oz (30 ml) of water, giving 20 kcal, a large scoop of milk (8 gm) needs 2 oz (60 ml) water giving 40 kcal.

Sterilization of Bottle

The bottle should be boiled with water for 10-15 minutes and the teat for 5 minutes only, after that it can be used or kept in a refrigerator to be used later on.

Complementary foods & weaning

Complementary foods (weaning foods) should be introduced in a stepwise fashion to both breastfed and formula-fed infants, beginning about the time the infant is able to sit, usually at 6 mo of age. Avoid starting with foods with high allergenic potential (cow's milk, eggs, fish, nuts, soybeans), Cereals, a good source of iron, is usually introduced 1st, followed by vegetables and fruits, then meats, and finally, eggs. Only 1 new food should be introduced every 3–4 days. Weaning from breast feeding can be initiated when mutually desired by the mother and infant by substituting formula or bovine milk by bottle or cup for part and, then, for all of a breastfeeding. These changes should be made gradually and should be a pleasant experience, not a conflict, for both the mother and the infant.

Constitutes of Breast Milk and standard Cow Milk- Based Formula

Water: relatively the same.

Calories: may vary slightly, but in average is 20 kcal/oz and approximately 67 kcal/dL

Protein: in breast milk is about 1.5 gm/100 kcal, in formula milk is about 1.8 to 3 g/100 kcal. Its type in breast milk casein: whey protein is 25:75, while casein (may be associated with cow milk allergy) is usually more in formula milk (whey : casein ratio varies from 20 : 80 to 60 : 40; one manufacturer markets a formula that is 100% whey). The predominant whey protein is β -globulin in cow milk and α -lactalbumin in human milk. This and other differences between human milk and cow milk– based formulas result in different plasma amino acid profiles in infants on different feeding patterns, but a clinical significance of these differences has not been demonstrated.

CHO: Lactose is the major carbohydrate in breast milk and in standard cow milk-based formulas. Formulas for term infants may also contain modified starch or other complex carbohydrates. Carbohydrates comprise 69-75g/L of cow milk-based formula.

Fat: is about 3.5 % in all. Plant or a mixture of plant and animal oils are the source of fat in infant formulas; fat provides 40-50% of the energy in cow milk-based formulas. All infant formulas are supplemented with long-chain polyunsaturated fatty acids PUFAs, docosahexaenoic acid (DHA), and arachidonic acid (ARA) at varying concentrations. ARA and DHA are found at varying concentrations in human milk and vary by geographic region and maternal diet. No studies in term infants have found a negative effect of DHA and ARA supplementation, and some studies have demonstrated positive effects on visual acuity and neurocognitive development.

Iron: it is lower in breast milk than the other, but it has more biological value by better absorption.

Calcium: is more in both cow & formula milk than breast milk, but the incidence of hypocalcaemia is more with the former due to high phosphorus.

Sodium, chloride, & potassium: they are more in cow milk based formula.

Types of formula milk

Soy Formulas (e.g. Isomil)

Soy protein-based formulas are all free of cow milk-based protein and lactose, it contains sucrose, corn syrup solids, and/or maltodextrin instead of lactose and the protein is a soy isolate supplemented with l-methionine, l-carnitine, and taurine. The fat is the same as in standard formula

Indications for soy formula include galactosemia and hereditary lactase deficiency, and situations in which a vegetarian diet is preferred, it also may be indicated when documented secondary lactose intolerance occurs. The routine use of soy protein-based formula has no proven value in the prevention or management of infantile colic, fussiness, or atopic disease. Infants with documented cow protein-induced enteropathy or enterocolitis are often sensitive to soy protein.

Protein Hydrolysate Formula

These formulas contain proteins that have been broken down to small segment, so that all less likely to induce an allergic reactions. They may be *partially hydrolyzed* (Dovamil®, Iptomil HA®) consisting from oligopeptides or *extensively hydrolyzed* (e.g. Alimentum®, Nutramigen®, Modilac riz®) which consist of peptides.

The fat blends similar to cow milk-based formulas, and carbohydrates are supplied by corn maltodextrin or corn syrup solids. Because the protein is not extensively hydrolyzed in

partially hydrolyzed proteins, these formulas should not be fed to infants who are allergic to cow protein.

Extensively hydrolyzed formulas are recommended for infants intolerant to cow milk or soy proteins. These formulas are lactose free and can include medium-chain triglycerides, making them useful in infants with gastrointestinal malabsorption as a consequence of cystic fibrosis, short gut syndrome, prolonged diarrhea, and hepatobiliary disease.

In studies, there was modest evidence that childhood atopic dermatitis at high risk infants may be delayed or prevented by using of extensively or partially hydrolyzed formulas.

Amino Acid Formulas (e.g. Neocate® or EleCare®)

Amino acid formulas are peptide-free formulas that contain mixtures of essential and nonessential amino acids. They are designed for infants with dairy protein allergy who failed to thrive on extensively hydrolyzed protein formulas.

MALNUTRITION

It is one of the leading causes of morbidity & mortality in childhood. The greatest risk of undernutrition occurs from conception to 24 mo of age, and this early damage to growth and development can have adverse consequences in later life on health, intellectual ability, school achievement, work productivity, and earnings; therefore they advised to focus interventions on this critical window of opportunity.

It may be *primary* (due to inadequate dietary intake, more in developing countries) or *secondary* (due to inadequate absorption, increased metabolism, or an abnormal loss, more in developed countries).

Milder degrees over a prolonged period result in *failure to thrive* with *growth retardation*, whereas severe deficiencies cause protein-energy malnutrition.

Types of malnutrition, according to the deficient nutrient,

- Macronutrient malnutrition: Def. of CHO, protein, or fat, also called Protein Energy Malnutrition (PEM).
- Micronutrient malnutrition: Def. of vitamins or minerals.

Assessment of Nutritional State

Clinical assessment of nutritional status should be an essential step in examination of every infant or child. Severe disturbances are readily apparent, but mild one may be overlooked, it needs careful evaluation.

Careful dietary history: With good *nutritional history*, an alert physician can detect nutritional disorders very early at the stage of dietetic errors.

- Physical examination: for Anthropometric measures, include:
 - . Wt/ Ht (wasting): indicate acute malnutrition.
 - . Ht/ age (length/ age for children <2 yr) (stunting): indicate chronic malnutrition.
 - . Wt/age (underweight): indicate combined acute & chronic malnutrition.
 - . OFC: affected only in severe cases especially during the 1st 2 years of life.
 - . Skin fold thickness: for subcutaneous fat.
 - . Mid arm circumference : for muscle mass.
 - . BMI (body mass index): for overweight & obesity.
- Regional examination: full body examination from above downward for signs of macro- & micro-nutrient malnutrition.
- Biochemical tests: albumin, hemoglobin, electrolyte, vitamins, & amino acids.
- Radiological examination: for signs of vitamin C & D deficiencies.

Classifications of Malnutrition

WHO classification

It uses standard deviation (SD, Z score) of displacement from the median.

- Every 5 % decrease from the standard Ht /age = 1 z score (1 SD).
- Every 15 % decrease from the standard Wt /age =1 z score (1SD).
- Every 10 % decrease from the standard Wt /Ht =1 z score (1SD).

So, - If the reading was 2 SD or more below median> moderate malnutrition.

- If it was 3 SD or more below median.....> severe malnutrition.

- If the child has malnutrition with edema.....> severe malnutrition.

Wellcome classification

Concentrate on weight for age & presence of edema, here malnutrition divided into:

- 1- underweight: weight is 60-80% of median wt/age with no edema.
- 2- Kwashiorkor: weight is 60-80 % of median wt/age with edema.
- 3- Marasmus: weight is less than 60 % of the median wt/age with no edema.
- 4- Marasmic- kwashiorkor: weight is less than 60% of the median wt/age with edema.

Note: the median for certain age and sex is the reading at the 50th percentile on the growth chart, roughly we can calculate approximated median from the following equations:

- Median body weight:

(from 2 mo - 12 mo) = (age in month + 9)/ 2

(above 1 yr)= age in year × 2 + 8

- Medians height= age in year × 6 +77

PROTEIN-ENERGY MALNUTRITION (PEM)

It includes both marasmus & kwashiorkor & a third disorder, marasmic kwashiorkor, which has features of both disorders.

NON- EDEMATOUS PEM (MARASMUS)

Most common forms of PEM

Mainly due to caloric depletion.

More common in young infant.

Less common in breastfeeding infants.

It occurs due to insufficient diet, also severe impairment of any body system may lead to marasmus.

Clinical manifestations

-It is characterized by failure to gain weight followed by weight loss until emaciation results, making the weight below 60 % with no edema

- At first, the infant may be fretful with increased appetite, but later the appetite diminishes

- The subcutaneous fat disappears making the skin loses its turgor and becomes wrinkled and loose especially over the thigh, buttock, & shoulders, loss of fat from the sucking pads of the cheeks often occurs late in the course of the disease; thus, the infant's face may retain a relatively normal appearance compared with the rest of the body, but this, too, eventually becomes affected.

- Infants are often constipated, but may have starvation diarrhea, with frequent, small, greenish mucoid stools.

- The abdomen is flat, but may be distended, with the intestinal pattern readily visible.

- There are muscle atrophy and resultant hypotonia.



- The basal metabolic rate tends to be reduced, as the condition progresses, the temperature usually becomes subnormal and the pulse slows.

- Marasmus is divided into 3 clinical grades:

Grade 1 : loss of subcutaneous fat over the abdominal wall.

Grade 2 : loss of subcutaneous fat over the buttocks & thighs.

Grade 3 : loss of subcutaneous fat over the face (senile face).

EDEMATOUS PEM (KWASHIORKOR)

Mainly due to protein deficiency, less common than Marasmus. It may become evident from early infancy to about 5yr old, usually after weaning from breastfeeding, so called kwashiorkor (means deposed child).

It occurs due to insufficient intake of protein of good biological value, this may be accompanied by abnormal absorption, synthesis, or loss of protein.

Clinical manifestations

- Initially, it may present as vague manifestations include lethargy, apathy, or irritability with loss of appetite. When advanced, there is a failure to gain weight, then weight loss, edema (usually develops early and may mask the failure to gain weight, it is often present in internal organs before it is recognized in the face and limbs), muscle wasting, hypotonia, flabby subcutaneous tissues.

- Abdominal distention & Liver enlargement may occur early or late in the course of disease due to fatty infiltration.

- Dermatitis is common, it usually occurs in irritated areas, not in areas exposed to sunlight (in contrast to pellagra). Dyspigmentation (darkening) may occur after desquamation in these areas, or it may be generalized.

- The hair is sparse and thin, easily extractable, and in dark-haired children, it may become red or gray, in chronic cases become coarse in texture. A flag sign may be present.

- Starvation diarrhea.

Complications of kwashiorkor

- Increased susceptibility to infections: measles (may be fatal), T. B., parasitic infections, HIV.

- In severe cases, mental & physical retardation.

- Eventually, if untreated, stupor, coma, and death.



TREATMENT OF MALNUTRITION

1st phase: Stabilization phase: (24- 48 hours -7 days)

The child with malnutrition is liable for life threatening sequelae that must be considered immediately, those include:

1- *Hypoglycemia* (<3 mmol/L): prevent hypoglycemia by feeding every 3 hours day & night.

* If it is developed & the patient is conscious, treat him by giving 50 ml of 10 % GW orally or 1 teaspoon sugar under the tongue, then start feeding.

** If the child is unconscious, give 5 ml /kg of 10 % GW intravenously & start feeding orally or by nasogastric tube.

2- *Hypothermia* (axillary temp. less than 35 °C): treated by warming the child by skin by skin contact with carer “kangaroo technique”, or use hot water bottle or warm incubator.

3- *Infection*: it is often silent, give broad spectrum antibiotic even without signs of infection

4- *Anemia*: if severe or associated with respiratory distress, give a blood transfusion slowly with furosemide.

5- *Dehydration & electrolyte disturbances*: Because of the difficulty of estimating hydration, oral rehydration solution is better than IV fluid. Malnourished child has low body potassium & high sodium; therefore give ReSoMal solution instead of ORS (half Na & double K content), if ReSoMal solution is not available, give WHO ORS diluted in 1.5 liter. Give IV fluid if the child is in shock.

** in this phase feeding started with frequent small amounts of F75 (especial formulated low-lactose milk with 75 kcal and 0.9 g protein per100 mL to which potassium, magnesium, and micronutrients are added) orally or by nasogastric tube.

2nd phase: Nutritional rehabilitation: (2nd week- 6th week)

- Replace F75 with an equal volume of F100 (100 kcal and 3 g protein per 100 ml) for 2 days and then increase each successive feed by 10 ml until reaching 150-220 kcal/kg/day. If breastfed, encourage continued breastfeeding.
- In general, feedings are initiated with higher frequency and smaller volumes; over time, the frequency is reduced and the amount is increased.
- Monitoring is needed for electrolyte imbalances, poor cardiac function, edema, or feeding intolerance, if any of these developed, further increase is stopped until stabilization.
- If diarrhea starts or fails to resolve and lactose intolerance is suspected, a non-lactose-containing formula should be substituted. If milk protein intolerance is suspected, a soy protein hydrolysate formula can be used.
- Children with severe malnutrition have developmental delays, so social care during and after treatment are essential to aid recovery of brain function.
- Early iron therapy usually is not started until this phase of treatment to prevent binding of iron to transferrin and this decreasing protein's host defense mechanisms, also free iron may exacerbate the oxidant damage, precipitating for kwashiorkor in malnourished child.

Follow up : to prevent recurrence.

Failure to Thrive

It refers to failure to gain adequate weight over a period of time where serial measurements are needed. Traditionally, Inadequate weight for age, weight for height, and body mass index less than 2 SD (or <3 or 5 percentile) for age and gender are diagnostic of FTT; others use weight for age which crossing 2 major percentiles on the growth curve (e.g. from above the 50th percentile to below the 25th) in a short time.

2 types:

1) **Non-organic or psychosocial FTT** occurs in a child who is usually <5 yr old; more in developing countries, may be due to poverty, errors in food preparation, child/parent interaction problems, food refusal, child neglect.

2) **Organic FTT** is marked by an underlying medical condition; more in developed countries.

- The cause of FTT has been often multi-factorial; organic and non-organic etiologic factors are usually coexisting, e.g. in neglected child, difficult premature infants, or child infected with HIV.

Organic FTT: all systems are involved;

1- **CNS:** e.g. CP.

2- **Renal:** e.g. UTI, RTA.

3-**Endocrine:** e.g. Diabetes insipidus, Adrenal insufficiency, hyperthyroidism.

4-**GIT:** e.g. Malabsorption, Celiac disease, Milk intolerance, cystic fibrosis.

5-**Cardiac:** e.g. CHD.

6-**Respiratory:** e.g. bronchiectasis, Bronchopulmonary dysplasia.

7-**Infection:** e.g. TORCH, TB, HIV.

8-**Genetic/ Chromosomal/ Metabolic:** e.g. Inborn errors of metabolism.

9-**Miscellaneous:** e.g. Malignancy.

According to pathophysiology:

- 1- **Inadequate nutrient intake:** e.g. poverty, Oromotor dysfunction, neurologic disease, Anorexia from systemic causes
- 2- **Malabsorption or increased losses:** e.g. celiac disease, chronic diarrhea.
- 3- **Increased metabolic demands or ineffective utilization:** e.g. Hyperthyroidism, malignancy, inborn errors of metabolism.

Clinical Manifestations

The clinical presentation of FTT ranges from failure to meet expected age norms for weight and height to loss of subcutaneous fat, reduced muscle mass, dermatitis, alopecia, recurrent infections, marasmus, kwashiorkor. The degree of FTT is usually calculated as for malnutrition, by standard deviation.

The physical examination should focus on identifying chronic illnesses, recognizing syndromes that may alter growth, and documenting the effects of malnutrition.

Diagnosis & treatment

- The **history, physical examination, and observation** of the parent-child interaction usually suggest the diagnosis. Regardless of the cause, an appropriate feeding environment at home is important.

- The **laboratory evaluation** is often not helpful and, therefore, should be used judiciously. A CBC and urinalysis represent a reasonable initial screen. Other tests should be performed if indicated.

- If the history, clinical examination, & investigations fail to find the cause;

1) Give a full caloric diet: the mealtimes should be approximately 20-30 min, solid foods should be offered before liquids, environmental distractions should be minimized, and children

should eat with other people and not be forced, the intake of water, juice, and low-calorie beverages should be limited. High-calorie foods, such as peanut butter, whole milk, cheese, dried fruits, & formulas containing more than 20 calories per ounce (PediaSure) are sometimes necessary.

2) Give multivitamins, iron, zinc, vitamin D.

3) Observe the daily weight, if it is 30- 60 gm/day in response to adequate caloric feedings usually establishes the diagnosis of psychosocial FTT, so continue the nutritional supplements.

4) Therapy for the psychosocial factors, parent education about feeding and temperament, as well as learning the infant cues for hunger, satiety, and sleep.

If after 1 wk no satisfactory increase of weight, or if the child has severe malnutrition, so admit the child to hospital & give full caloric diet (as above) & observe the daily increase in weight, if there is a satisfactory gain in weight, so non- organic FTT.

If the child failed to gain weight on admission & full diet, so organic FTT is highly possible, start more specific investigations for diagnosis.

MICRONUTRIENT DEFICIENCY

VITAMINS

They are organic substances, required in minute amount. 2 types:

- Water soluble: C, B- complex, folic acid. Toxicity is not common.
- Fat soluble: A, K, E, D. toxicity is common esp. A & D.

Vitamins which are produced from the intestinal flora: vit K, biotin, & pantoic acid (B5).

VITAMIN C

It has many functions, e.g. increasing the absorption of iron, but the major role is the formation of normal collagen of mesenchymal structures especially bone, cartilage, dentine, and blood vs.

Sources are fruits, tomatoes, & green vegetables. Breast milk produced by a vitamin C-sufficient mother contains adequate vitamin C, as do all infant formulas.

Deficiency causes scurvy, while excess causes oxaluria.

Scurvy :

The defective formation of collagen causes fragile blood vs. & defective tooth dentin, Common between 6- 24 mo, leads to:

- Easy bleeding: skin & mucous membrane hge, hematuria, melena, orbital or subdural hemorrhages, and the characteristic perifollicular hemorrhages

- Subperiosteal hg (bone tenderness, irritability, pseudo paralysis, frog like posture, edematous swelling of extremities).
- Gum changes: bluish purple, spongy swellings of the mucous membrane, hypertrophy & bleeding in advanced cases.
- Anemia.
- A "rosary" at the costochondral junctions and depression of the sternum.
- Slow wound and fracture healing.

Diagnosis

X ray: occur at the distal ends of the long bones and are particularly common at the knees. The shafts have a ground-glass appearance because of trabecular atrophy. The cortex is thin and dense with *white line of Frankel* (an irregular but thickened white line at the metaphysis, represents the zone of well-calcified cartilage).

Low serum & WBC ascorbic acid.

Treatment: Vitamin C orally.

B- COMPLEX VITAMINS

They share in the same sources and related reactions, so the def. of one is usually associated with def. of the others.

Sources: animal products followed by grains & vegetables.

THIAMIN (B1)

It is a co- enzyme for CHO metabolism & acetylcholine synthesis in CNS and deficiency results in impaired nerve conduction.

Breast milk (from a vitamin B-sufficient mother) and bovine milk are good sources of thiamine

Def. cause beriberi

Beriberi

Affect mainly CVS & CNS, presented with either congestive heart failure (wet type) or neurological (dry type) manifestations including generalized weakness, ptosis, constipation, hoarseness of voice, ataxia, & signs of lower motor neuron lesion. Mixed type may also presented

Treatment

Vitamin B1 orally + anti- failure measures.

If a breast-fed infant develops beriberi, both the mother and child should be treated with thiamine.

RIBOFLAVIN (B2)

Important for fat, CHO, & protein metabolism & retinal pigmentation for light adaptation.

Deficiency causes cheilosis, glossitis, keratitis, conjunctivitis, photophobia, lacrimation, corneal vascularization, and seborrheic dermatitis.

Normochromic, normocytic anemia may also be seen because of the impaired erythropoiesis.

Diagnosis

Most often, the diagnosis is based on the clinical features of angular cheilosis in a malnourished child, which responds promptly to riboflavin supplementation.

Treatment:

Vitamin B2

NIACIN (NICOTINAMIDE)

Niacin forms part of two cofactors, NAD & NADP.

Niacin deficiency cause pellagra

Pellagra

The early symptoms of pellagra are vague: anorexia, lassitude, weakness. After a long period of deficiency, the classic triad (3D) of dermatitis, diarrhea, and dementia appears. Dermatitis, the most characteristic manifestation of pellagra, may be elicited by intense sunlight. The lesions first appear as symmetrical areas of erythema on exposed surfaces, resembling sunburn, then it progress to vesicles, crusts, & desquamation. The lesions are usually sharply demarcated from the healthy skin around them, on the hands often have the appearance of a glove & on the foot and leg (pellagrous boot).

It occurs chiefly in countries where corn (maize) is a basic foodstuff.

Treatment

50–300 mg/ day of Niacin. Sun exposure should be avoided during the active phase of pellagra, and the skin lesions may be covered with soothing applications.

VITAMIN B₆ (PYRIDOXINE)

It is a co-enzyme for protein, fat, & CHO metabolism, important for CNS function (serotonin & GABA formation), and in the synthesis of heme. It has anti-emetic properties.

The pyridoxine content of human milk and infant formulas is adequate. Pyridoxine antagonists (e.g., isoniazid, penicillamine, corticosteroids, phenytoin, carbamazepine). High protein intake & pregnancy increase the requirements for pyridoxine.

Many clinical disturbances caused by vitamin B₆ deficiency have been described in humans: vitamin B₆ dependence syndromes (including vitamin B₆-dependent convulsions with abnormal EEG, a vitamin B₆-responsive anemia, homocystineuria, others) & others, e.g. cheilitis, glossitis, facial seborrhea. Microcytic anemia, Oxaluria, & infections

Vitamin B₆ dependence syndromes result from errors in enzyme structure or function, they respond to very large amounts of pyridoxine.

The use of high doses during pregnancy has been implicated in some cases of transient vitamin B₆ dependent syndrome in infants, so for those infant & patients on large doses for long periods of time, withdrawal of vitamin B₆ should be gradual.

Treatment

All infants with seizures should be suspected of having vitamin B₆ deficiency or dependence if more common causes of infantile seizures (e.g., hypocalcaemia, hypoglycemia, infection) are eliminated and 100 mg of pyridoxine should be injected. In general, it is enough to stop convulsion with good dietary follow.

Newborn of a mother took large amount of pyridoxine during pregnancy must receive 10 mg/day orally after birth for several wks to prevent convulsions. Dependency syndromes respond to daily 10 mg pyridoxine.

VITAMIN B12

Important for DNA synthesis.

It needs intrinsic factor (IF), which is secreted from the stomach, for its absorption at the terminal ileum, then carried in the serum by transcobalamin to the liver.

Sources: animal product & low in vegetable.

Body store is sufficient for 3-5 yr.

Causes of deficiency

Extreme vegans (vegetarian), juvenile pernicious anemia (absent IF), problems of terminal ileum (surgical resection, tuberculous & regional enteritis, diphyllobothrium latum infestation, malabsorption), & congenital transcobalamin deficiency.

Clinical manifestations

General: Irritability, anorexia, diarrhea, FTT.

Hematological: megaloblastic anemia, thrombocytopenia, & neutropenia with polysegmented nucleus.

Neurological: ataxia, parasthesia, hypotonia, Babinski sign, clonus, & coma.

Diagnosis

Low serum B₁₂, normal Folate, +ve schilling test.

Treatment

Administration of a minidose (1–5µg/day) may be used as a therapeutic test when the diagnosis of vitamin B₁₂ deficiency is in doubt. If there is evidence of neurologic involvement, 1 mg should be injected intramuscularly daily for at least 2 wk.

FOLIC ACID

Folate coenzymes for synthesis of DNA and purine. Maternal folic acid status is known to be protective against neural tube defects

Limited body store, so depleted within 2-3 mo.

Causes of deficiency:

Deficient diet (goat milk)

Increased requirement (hemolytic anemia, prematurity, pregnancy, & infection)

Malabsorption syndrome.

Drug interaction (anticonvulsants, Methotrexate).

Clinical manifestations:

Irritability, anorexia, FTT, diarrhea, megaloblastic anemia, thrombocytopenia, & neutropenia with polysegmented nucleus.

Neurological manifestation is due to hereditary malabsorption of folic acid which respond to folic acid.

Diagnosis:

Low serum & RBC Folate with normal vit B₁₂.

Treatment

0.5 – 1 mg/ day folic acid orally or IM for 3-4 wks with maintenance therapy with a multivitamin (containing 0.2 mg of folate) is adequate.

If the specific diagnosis is in doubt, smaller doses (0.1 mg/day) may be used for 1 week as a diagnostic test, because a hematologic response can be expected within 72 hr. Doses of folate >0.1 mg can correct the anemia of vitamin B₁₂ deficiency but may aggravate any associated neurologic abnormalities.

FAT- SOLUBLE VITAMINS

In general, they are deficient in cases of fat malabsorption.

VITAMIN A

Important for synthesis of rhodopsin & iodopsin, & for skin & mucous membrane integrity.

Sources: yellow & green vegetables, fruits, eggs, butter, liver.

Overdose may lead to toxicity (pseudotumour cerebri: benign increase of intracranial pressure) &, in pregnancy, to congenital anomalies.

Deficiency result in:

Eye: Ocular lesions of vitamin A deficiency develop insidiously and rarely occur before 2-3 yr of age.

At early stage: Delayed dark adaptation, then night blindness. Photophobia is common. corneal keratinization, cloudiness, then xerophthalmia (dry, scaly layers of cells). The conjunctiva keratinizes and develops plaques (**Bitot spots**).

In later stages: infection occurs, lymphocytes infiltrate, and the cornea becomes wrinkled; it degenerates irreversibly (keratomalacia and corneal ulceration), resulting in blindness.

Skin: follicular hyperkeratosis with dry scaly skin.

Mucous membrane: epithelial changes lead to:

Bronchial obstruction with recurrent chest infection.

Infection of urinary tract & salivary gland.

Brain: Increased intracranial pressure with wide separation of cranial bones at the sutures.

Treatment

For latent deficiency: 1500 µg / day vit. A.

For xerophthalmia or other major complications: 1500 µg / kg/ day orally for 5 days , then daily IM injection of 7500 µg till recovery, with eye care by ophthalmologist.

Morbidity and mortality rates from viral infections such as measles may be lower in non- deficient children who are given daily doses of 1,500-3,000 µg of vitamin A.

VITAMIN K

Required for production of factors (10, 9, 7, 2) for blood clotting, protein Z & M (stimulate platelet activity), for protein C & S (anticoagulant).

it presents in natural form (K1 from food), K2 from intestinal flora, & in synthetic form (Large doses of may cause hyperbilirubinemia & kernicterus in neonate & in patient with G6PD def).

Deficiency

Causes:

Breast feeding; prolong use of AB that kills the bacterial flora, fat malabsorption, & chronic diarrhea.

Diseases of the liver may limit synthesis of prothrombin. Hypoprothrombinemia from this cause usually does not respond to administration of vitamin K.

It will leads to hemorrhagic disease of newborn & bleeding tendency at any age.

Treatment

For mild def.: 1- 2 mg orally every 24 hr should be given.

If bleeding occurs, 5 mg IM every 24 hr should be given.

If bleeding is severe, or the patient has liver diseases give fresh blood or fresh frozen plasma.

VITAMIN E

It acts antioxidant.

Sources: seeds, nut, green leafy vegetables.

Deficiency:

Causes: fat malabsorption, high polyunsaturated fatty acid diet (because it is oxidant agent, so more anti- oxidant is needed). Premature infants are particularly susceptible to vitamin E deficiency, because there is a significant transfer of vitamin E during the last trimester of pregnancy.

Leads to:

Cerebellar disease (ataxia, dysarthria, nystigmus), posterior column dysfunction (loss of deep tendon reflex, decreased proprioception and vibratory sensation), and retinal disease.

In premature infant (during the 2nd month): hemolytic anemia, edema, & thrombocytosis.

Treatment: vitamin E supplementation.