# College of Medicine/ Diyala University
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## Failure to Thrive

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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, & pantathonic acid.

### 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 oxalluria.

***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, malena, orbital or subdural 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, 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 congestive heart failure & polyneuropathy: generalized weakness, ptosis, constipation, hoaresness of voice, ataxia, & signs of lower motor neuron lesion.

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 symmetric 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 $B_6$ (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 $B_6$ deficiency have been described in humans: vitamin $B_6$ dependence syndromes (including vitamin $B_6$-dependent convulsions with abnormal EEG, a vitamin $B_6$-responsive anemia) & others, e.g. cheilitis, glossitis, facial seborrhea. Microcytic anemia, Oxaluria, & infections

*Vitamin $B_6$ 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 $B_6$ deficiency or dependence. If more common causes of infantile seizures (e.g., hypocalcaemia, hypoglycemia, infection) are eliminated, 100 mg of pyridoxine should be injected. 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.

## 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, diphyllobotherium 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, Babenski 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 $B_{12}$ 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, Methotraxate).

**Clinical manifestations:**

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

**Diagnosis:**

Low serum & RBC Folate with normal vit B12.

**Treatment**

0.5 – 1 mg/ day folic acid orally or IM for 3-4 wks. 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 $B_{12}$ deficiency but may aggravate any associated neurologic abnormalities. Maintenance therapy with a multivitamin (containing 0.2 mg of folate) is adequate.

## FAT- SOLUBLE VITAMINS

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

## VITAMIN A

Important for synthesis of rhodopsin & iodeopsin, & 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, cloudness, 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. 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, dysartheria, nystigmus), posterior column dysfunction (loss of deep tendon reflex, decreased properioception and vibratory sensation), and retinal disease.

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

**Treatment:** vitamin E supplementation.