

Nutritional Deficiencies

Index	
Introduction.....	1
Iron Deficiency.....	1
Vitamin D Deficiency.....	2
Calcium Deficiency.....	2
Deficiencies of Cobalamin and Folate.....	2
Deficiencies of Other Water-Soluble Vitamins.....	3
Deficiencies of Other Fat Soluble Vitamins.....	3
Other Mineral Deficiencies.....	3
Conclusion.....	4
References.....	4
Acknowledgements.....	4

Introduction

Nutritional deficiencies can be very significant to the overall health of infants and children because growth and development can be seriously hindered by shortages in essential vitamins or nutrients. The two most common are iron and vitamin D deficiency.

Iron Deficiency

In general, iron deficiency presents as microcytic anemia in a well-nourished infant who is otherwise healthy and asymptomatic. Less common presentations include pallor, lethargy, irritability, poor feeding, and cardiomegaly. Although iron deficiency is often caused by deficient iron in the diet, it can also be caused by intestinal blood loss (e.g. due to early introduction of cow's milk), celiac disease, *Helicobacter pylori* infections, and anemia of chronic disease. Risk factors for iron deficiency include children in poverty, premature and low-birth-weight infants, toddlers of African or Hispanic ancestry, infants who are exclusively fed with non-iron fortified formulas, obese children aged 1-3, and immigrant children.

Infants and children can be screened for dietary iron deficiency by hemoglobin levels, but this is generally not very sensitive or specific. The most important screening tool is a careful dietary history. Iron deficiency can be suspected in children who consume less than five servings of meat, grains, and fruits and vegetables per week, drink more than 480 mL of milk or soda per day, or have a daily intake of snacks high in fat or sugar. Iron deficiency can be diagnosed by low hemoglobin levels with low ferritin levels. A more complete iron workup could also be indicated. Once nutritional iron deficiency has been diagnosed, the condition can be treated with supplemental oral iron. Close follow-up of the patient is indicated.

Vitamin D Deficiency

Vitamin D is essential for the absorption of calcium in the gastrointestinal tract. Deficiencies in vitamin D can lead to hypocalcemia and hypophosphatemia, leading to rickets in children. Pediatric patients with vitamin D deficiency are generally asymptomatic, but can present with secondary hyperparathyroidism and changes in growth plates. Risk factors for vitamin D deficiency include prolonged breast-feeding without vitamin D supplementation, breast-feeding mothers who are dark-skinned, breast-feeding mothers who are vitamin D deficient, and low sun exposure.

Additionally, there has been evidence that suggests that vitamin D deficiency has a role in immune function independent from the calcium metabolism pathway. This implies that vitamin D deficiency could be involved in the development of allergies and atopic diseases, tuberculosis and other respiratory infections, and autoimmune diseases such as type I diabetes mellitus, asthma, and multiple sclerosis.

The characteristic laboratory finding of rickets due to vitamin D deficiency is elevated serum parathyroid hormone concentrations. There can also be radiologic findings in severe rickets. Treatment involves daily vitamin D supplementation at doses ranging from 1000-5000 IU depending on the age of the child. Once there is radiographic evidence of bone healing, this dose of vitamin D can be reduced to 400 IU. Calcium supplementation should be concomitant at 1000 mg per day. Patients can be followed-up with a test for urinary calcium, which should become detectable after three months of treatment. Other tests used to follow-up these patients include serum calcium, phosphorus, alkaline phosphatase, and urinary calcium/creatinine ratio.

Calcium Deficiency

Rickets secondary to nutritional causes can be due to either vitamin D deficiency, calcium deficiency, or both. Particularly in regions of the world where there is ample sunlight, rickets may be due to calcium deficiency rather than vitamin D deficiency. In these cases, it would be best to treat with supplemental calcium with vitamin D rather than with supplemental vitamin D alone.

Deficiencies of Cobalamin and Folate

Deficiencies of cobalamin (vitamin B12) and folate can cause megaloblastic anemia, which is diagnosed by red blood cell histology. Macrocytosis with hypersegmentation of neutrophils is pathognomonic for megaloblastic anemia. There may also be other hematologic abnormalities, such as decreased reticulocyte count, increased serum iron, thrombocytopenia, and neutropenia.

In infants, cobalamin deficiency is usually due to cobalamin deficiencies of breast-feeding mothers who follow strict vegan or moderate vegetarian diets. Other causes include breast-feeding mothers who have had gastric bypass, malabsorptive syndromes, and pernicious anemia. Infants often present with growth, movement, developmental, and hematologic problems, but if identified and treated early with supplementation, children can avoid long term consequences of this nutritional deficiency.

Folate deficiency is usually caused by dietary folate deficiency. Medications such as pyrimethamine, methotrexate, and phenytoin can also cause folate deficiency.

Deficiencies of Other Water-Soluble Vitamins

Thiamine (vitamin B₁) deficiency is associated with beriberi, Wernicke-Korsakoff syndrome, and Leigh syndrome. Foods with more thiamine include yeast, legumes, pork, rice, and cereals; milk products, fruits, and vegetables are not good sources of thiamine. Beriberi in infants can present as cardiomegaly, tachycardia, a loud piercing cry, cyanosis, dyspnea, and vomiting, all culminating in fulminant cardiac syndrome. These patients can also develop aseptic meningitis.

Riboflavin (vitamin B₂) deficiency presents with relatively non-specific signs, such as sore throat, hyperemia and edema of mucous membrane, cheilitis, stomatitis, glossitis, normocytic anemia, and seborrheic dermatitis. Foods rich in niacin include meats, fish, eggs, milk, green vegetables, and yeast.

Niacin (vitamin B₃) deficiency presents with hyperpigmentation in sun-exposed areas, a red tongue, diarrhea, vomiting, and neurologic symptoms, such as insomnia, anxiety, delusions, and dementia. Dietary niacin deficiency is known as pellagra.

Pyridoxine (vitamin B₆) deficiency is not commonly encountered as an isolated nutritional deficiency. If encountered, it is often associated with isoniazid treatment or in infants older than six months who are exclusively breast-fed.

Biotin deficiency in children is generally due to a genetic disorder known as biotinidase deficiency, which causes hypotonia, ataxia, hearing loss, optic atrophy, skin rash, and alopecia. Supplementation with biotin resolves these symptoms.

Rates of vitamin C deficiency are quite low, but when patients' diets are deficient in this vitamin, they present with symptoms such as bleeding gums, ecchymoses, petechiae, coiled hairs, hyperkeratosis, arthralgias, and impaired wound healing. This is because vitamin C is involved in collagen synthesis. Supplementation with vitamin C resolves these symptoms.

Deficiencies of Other Fat Soluble Vitamins

Vitamin A deficiency typically presents as drying of the conjunctiva, presence of Bitot spots (keratin debris on the conjunctiva), drying of the cornea, and night blindness. This can eventually progress to permanent blindness. Vitamin A deficiency is often subclinical and renders the pediatric patient susceptible to infection. Physical growth is also reduced.

Vitamin E deficiency is not common, but can be due to patients who have difficulty absorbing fat, as well as in patients with disorders such as abetalipoproteinemia and cystic fibrosis. Clinical presentation of vitamin E deficiency includes myopathy, ataxia, pigmented retinopathy, and vision loss.

Vitamin K deficiency causes hypocoagulation and can be diagnosed with an elevated prothrombin time and international normalized ratio. Vitamin K deficiency in infants can be due to mothers taking medications that inhibit vitamin K (e.g. warfarin), insufficient nutrition, and exclusive breast-feeding up to six months of age. Administration of vitamin K within 24 hours of delivery can prevent vitamin K deficiency at birth.

Other Mineral Deficiencies

Zinc deficiency can cause growth failure, primary hypogonadism, and impaired immune system

functioning. This could be due to diets high in phytates, which bind and inhibit the absorption of zinc.

Iodine deficiency causes deficiencies in thyroid hormones, which can lead to goiter, hypothyroidism, mental retardation, and cretinism, although iodine deficiency in children may be subclinical.

Selenium deficiency can present in infants and children with diet restrictions or in those who are on long term parenteral nutrition.

Copper deficiency in children can result from the X-linked Menkes syndrome, which is associated with growth retardation, peculiar hair, and focal degeneration of the cerebrum and cerebellum.

Conclusion

Even though nutritional deficiencies can have profound impacts on the health of children, timely identification and management of these imbalances can reduce their deleterious effects. Physicians would be advised to elicit a complete history, perform a physical examination, and order relevant laboratory tests if suspicions of a nutritional deficiency are raised. If a nutritional deficiency is confirmed, appropriate counseling regarding adjustments in diet and supplementation would then be indicated.

References

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