total parenteral nutrition without zinc supplementation and in premature infants fed human milk without fortification. **Clinical manifestations** of mild zinc deficiency include anorexia, growth faltering, and immune impairment. Moderately severe manifestations include delayed sexual maturation, rough skin, and hepatosplenomegaly. The signs of severe deficiency include acral and periorificial erythematous, scaling dermatitis; growth and immune impairment; diarrhea; mood changes; alopecia; night blindness; and photophobia.

Diagnosis of zinc deficiency is challenging. Plasma zinc concentration is most commonly used, but levels are frequently normal in conditions of mild deficiency; levels in moderate to severe deficiency are typically less than 60 μg/dL. Acute infection also can result in depression of circulating zinc levels. The standard for the diagnosis of deficiency is response to a trial of supplementation, with outcomes such as improved linear growth or weight gain, improved appetite, and improved immune function. Because there is no pharmacologic effect of zinc on these functions, a positive response to supplementation is considered evidence of a preexisting deficiency. Clinically an empirical trial of zinc supplementation (1 μg/kg/day) is a safe and reasonable approach in situations in which deficiency is considered possible.

Acrodermatitis enteropathica is an autosomal recessive disorder that begins within 2 to 4 weeks after infants have been weaned from breast milk. It is characterized by an acute perioral and perianal dermatitis, alopecia, and failure to thrive. The disease is caused by severe zinc deficiency from a specific defect of intestinal zinc absorption. Plasma zinc levels are markedly reduced, and serum alkaline phosphatase activity is low. **Treatment** is with high-dose oral zinc supplements. A relatively uncommon condition associated with presentation of severe zinc deficiency is due to a defect in the secretion of zinc from the mammary gland, resulting in abnormally low milk zinc concentrations. Breastfed infants, especially those born prematurely, present with classic signs of zinc deficiency: growth failure, diarrhea, and dermatitis. Because there is no defect in the infant's ability to absorb zinc, treatment consists of supplementing the infant with zinc for the duration of breastfeeding, which can be successfully continued. Subsequent infants born to the mother will also need zinc supplementation if breastfed. Zinc is relatively nontoxic. Excess intake produces nausea, emesis, abdominal pain, headache, vertigo, and seizures.

Fluoride

Dental enamel is strengthened when fluoride is substituted for hydroxyl ions in the hydroxyapatite crystalline mineral matrix of the enamel. The resulting fluoroapatite is more resistant to chemical and physical damage. Fluoride is incorporated into the enamel during the mineralization stages of tooth formation and by surface interaction after the tooth has erupted. Fluoride is similarly incorporated into bone mineral and may protect against osteoporosis later in life.

Because of concern about the risk of **fluorosis**, infants should not receive fluoride supplements before 6 months of age. Commercial formulas are made with defluoridated water and contain small amounts of fluoride. The fluoride content of human milk is low and is not influenced significantly by maternal intake. Practitioners should evaluate all potential fluoride sources and conduct a caries risk assessment before prescribing fluoride supplementation.

Suggested Readings

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