Pediatric Neurology Part III: Chapter 184. Vitamin-responsive disorders: cobalamin, folate, biotin, vitamins B1 and E (Handbook of Clinical Neurology)

Matthias R. Baumgartner

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The catalytic properties of many enzymes depend on the participation of vitamins as obligatory cofactors. Vitamin B12 (cobalamin) and folic acid (folate) deficiencies in infants and children classically present with megaloblastic anemia and are often accompanied by neurological signs. A number of rare inborn errors of cobalamin and folate absorption, transport, cellular uptake, and intracellular metabolism have been delineated and identification of disease-causing mutations has improved our ability to diagnose and treat many of these conditions. Two inherited defects in biotin metabolism are known, holocarboxylase synthetase and biotinidase deficiency. Both lead to multiple carboxylase deficiency manifesting with metabolic acidosis, neurological abnormalities, and skin rash. Thiamine-responsive megaloblastic anemia is characterized by megaloblastic anemia, non-type I diabetes, and sensorineural deafness that responds to pharmacological doses of thiamine (vitamin B1). Individuals affected with inherited vitamin E deficiencies including ataxia with isolated vitamin E deficiency and abetalipoproteinemia present with a spinocerebellar syndrome similar to patients with Friedreich's ataxia. If started early, treatment of these defects by oral or parenteral administration of the relevant vitamin often results in correction of the metabolic defect and reversal of the signs of disease, stressing the importance of early and correct diagnosis in these treatable conditions.

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