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Neurological Complications of Nutritional Disease

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Vitamins and minerals have essential functions in the body, from signal transduction to acting as cofactors for numerous enzymatic processes. Nutritional deficiencies and excess of certain vitamins and minerals can have profound effects on the central and peripheral nervous systems from early development into adulthood. This article summarizes the role of various nutritional factors in the nervous system and the neurological symptoms that can arise from deficiency or excess.
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Introduction

Malnutrition can have devastating effects on the developing and developed brain. Chronic protein and caloric deficiency, or marasmus, can lead to growth failure, mood changes, altered mental status, autonomic instability, muscle wasting, and eventually death. Kwashiorkor is a disease of chronic protein deficiency in the setting of sufficient caloric intake. Delayed cerebral myelination and diffuse cerebral atrophy have been described in both marasmus and kwashiorkor, and this cerebral atrophy appears to be reversible with improvement in nutrition[1] (Fig. 1). In addition to global nutritional deficiencies, deficiency or excess of specific nutrients can affect both the central and peripheral nervous systems. Most vitamins and essential minerals are not synthesized by the body and must be obtained through dietary intake. They are essential for many functions in the body, acting as cofactors or reducing agents in various enzymatic reactions. Disorders of absorption, inborn errors of metabolism, medication use, and removal of vitamins and minerals through dialysis can all lead to deficiencies. The remainder of this article summarizes the causes and neurological consequences of specific vitamin and mineral deficiencies and excesses.

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Vitamin A

Vitamin A, retinol, and retinoid derivatives are primarily used in the pigmentation of the retina. Different forms of vitamin A have specific physiological functions in the body, and they are found in fruits and vegetables. For example, 11-cis-retinal together with opsin forms rhodopsin, a photosensitive compound that initiates the retinal action potential through conformational changes when exposed to light. As early as embryogenesis and organogenesis, retinoids function in neural tube differentiation and patterning, neuronal apoptosis, neurogenesis, and plasticity.[2-4]

Hepatic and pancreatic disease, steatorrhea, chronic diarrhea, chronic infections, and hyperthyroidism can all result in vitamin A deficiency. Symptoms of deficiency are ocular disorders such as blindness due to retinal pigment abnormalities in addition to impaired immunity, abnormal organogenesis, and growth delay.[5] Clinical manifestations of night blindness, xerophthalmia, Bitot spots, corneal ulcerations, and congenital malformations of the eye can occur.[2,6,7]

Vitamin A intoxication is rare but can have deleterious effects. Elevated levels can occur acutely or more chronically from oral supplementation for treatment of acne or with diseases such as type I hyperlipidemia or alcoholic hepatic disease. Acute intoxication presents with symptoms of headache, vomiting, diplopia, papilledema, and idiopathic intracranial hypertension.[8] Chronic intoxication results in increased intracranial pressure, diplopia, hyperostosis of the skull and joints, hepatomegaly, and growth failure. During pregnancy, excess vitamin A supplementation can have toxic effects on the developing fetus. Teratogenic effects include hydrocephalus, microcephaly, optic nerve defects, and retinal defects. Diagnosis is made by measurement of plasma retinol concentration and by the presence of periosteal bone formation on X-ray. Treatment involves the removal of excess vitamin A from the diet.[2,8]

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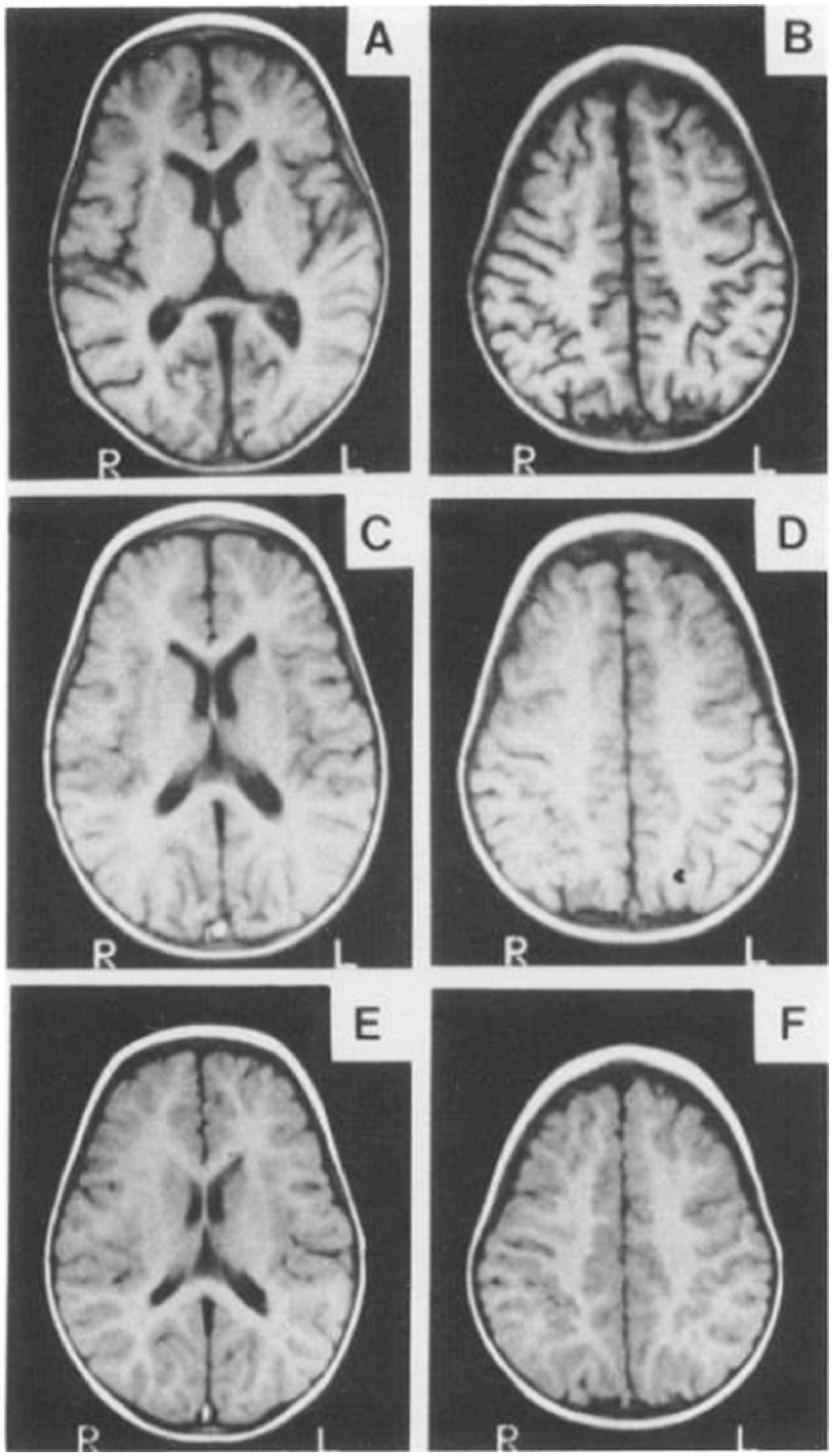

Figure 1 (A and B) The typical MRI changes of cerebral atrophy as seen on admission; (C and D) the improvement by day 30; and (E and F) day 90 scans, illustrating the rapid resolution of the signs of cerebral atrophy with refeeding (patient 4). A, C, and E are mid-axial views and B, D, and F are high axial views of the brain.[1]

Vitamin B1

Vitamin B1, thiamine, is involved in many physiological processes in the body. It is a cofactor in oxidative decarboxylation and has an important function in the metabolism of carbohydrates, lipids, and amino acids, and in the synthesis of nucleotides, fatty acids, ATP, and neurotransmitters. It is also involved in myelin maintenance, neuronal membrane transport, and axonal conduction.[2]

Dietary sources of thiamine include meats, grains, and vegetables, but only a fraction is absorbed by the small intestine. It is transported across the blood-brain barrier by a