Neurologic manifestations of malabsorption syndromes.

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BACKGROUND: Although malabsorption is generally considered to be a gastrointestinal problem, the effects of malabsorption extend far beyond the gastrointestinal tract and can include neurologic dysfunction. Malabsorption may occur by a variety of mechanisms, both genetic and acquired, that interfere with the absorption of basic nutrients, vitamins, minerals, and trace elements. Disorders that interfere with fat absorption can lead to neurologic dysfunction as a consequence of associated impairment of fat-soluble vitamin absorption.

FINDINGS: Thus, individuals with genetic vitamin E deficiency and the familial hypocholesterolemias may develop symptoms of peripheral neuropathy, cerebellar ataxia, and other neurologic signs and symptoms. Disease processes that damage the enteric mucosa and produce malabsorption can trigger neurologic dysfunction both by immune-related processes, as in celiac disease, and by impairing absorption of essential vitamins and other nutrients, as in tropical sprue. Deficiencies of water-soluble vitamins, such as thiamine and niacin, can also develop in the setting of malabsorption and lead to neurologic dysfunction. Neurologists are aware of the neurologic damage that copper excess can cause in Wilson's disease, but copper deficiency due to malabsorption can also produce neurologic dysfunction in the form of myelopathy.

CONCLUSIONS: It is vitally important for neurologists to be aware of the potential for malabsorptive processes to produce neurologic dysfunction, because effective treatment for such disorders is often available.

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