

Abstract

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Copper deficiency myelopathy (human swayback).

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BACKGROUND: The hematologic manifestations of copper deficiency are well known and include anemia and neutropenia. In the past few years, the neurological manifestations of acquired copper deficiency in humans has been recognized, the most common being a myelopathy presenting with a spastic gait and prominent sensory ataxia. The known causes of acquired copper deficiency include prior gastric surgery, excessive zinc ingestion, and malabsorption; however, often the cause is unclear. Hyperzincemia may be present even in the absence of exogenous zinc ingestion. The clinical features and neuroimaging findings are similar to the subacute combined degeneration seen in patients with vitamin B12 deficiency. Copper and vitamin B12 deficiency may coexist. The neurological syndrome may be present without the hematologic manifestations.

CONCLUSION: Copper supplementation resolves the anemia and neutropenia promptly and completely and may prevent the neurological deterioration. Improvement, when it occurs, is often subjective and preferentially involves sensory symptoms. This article describes patients with copper deficiency myelopathy seen at the Mayo Clinic in Rochester, Minn, and reviews the literature on neurological manifestations of acquired copper deficiency in humans.

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