VITAMIN B12 DEFICIENCY

Autoimmune and Other Causes of Cobalamin Deficiency

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Vitamin B12 deficiency can cause a variety of symptoms, including dementia. Autoimmune causes of this deficiency include pernicious anemia and autoimmune gastritis.

Vitamin B12

Vitamin B12, which is the common name for cobalamin, is an essential vitamin obtained from dietary sources. Vitamin B12 deficiency is present in 1 to 3 percent of the population, although the incidence of vitamin B12 deficiency rises dramatically with age. Among people older than 60 years, the incidence of cobalamin deficiency is as high as 10 to 30 percent. This incidence is expected to increase in the next 20 years. Cobalamin deficiency is caused by diets low in vitamin B12, malabsorption syndromes, infection, and the autoimmune disorders autoimmune gastritis and pernicious anemia. Vitamin B12 deficiency is treated with either oral or intramuscular injections of vitamin B12.

Symptoms and Signs of Vitamin B12 Deficiency

A number of unrelated symptoms can occur in vitamin B12 deficiency. Clinical manifestations of vitamin B12 deficiency include:

* Hematological changes, such as megaloblastic anemia (characterized by large red blood cells), and a low production of other blood cell types, which is known as pancytopenia. Types of pancytopenia seen in vitamin B12 deficiency include leucopenia (low white blood cell levels), thrombocytopenia (low platelet count), and anemia.

* Neurologic changes, including paresthesias, peripheral neuropathy and combined systems disease such as demyelination (loss of protective outer sheath) of the dorsal columns of the nerve cells in the spine.

* Psychiatric changes, including irritability, personality change, mild memory impairment, dementia, depression, and psychosis.

* Cardiovascular symptoms, including a possible increased risk of heart attack and stroke.

During its absorption vitamin B12 binds to the protein haptocorin found in saliva. In the duodenum, vitamin B12 is released from haptocorin. The free cobalamin molecules are
then linked to a substance known as intrinsic factor in the proximal ileum of the intestines. In this form vitamin B12 enters the mucosal cells that line the intestines. Here, cobalamin is released. In its free form it binds to the protein transcobalamin, and in this form vitamin B12 is released into the blood circulation. Within the body’s cells vitamin B12 is freed from transcobalamin protein and it acts as a coenzyme for the synthesis for various enzymes needed for DNA synthesis and energy production.

**Pernicious Anemia**

Pernicious anemia is an autoimmune disorder characterized by atrophy of the gastric mucosa, selective loss of parietal and chief cells from the gastric mucosa, and an infiltration of lymphocytes into the submucosa. Immunologically, pernicious anemia is characterized by autoantibodies to gastric parietal cells (AGPA), proton pump (H⁺K⁺ATPase), and to the cobalamin-absorbing protein, intrinsic factor. Intrinsic factor is a 60 kD glycoprotein produced by the parietal cells of the stomach lining that enables the absorption of vitamin B12. Two types of intrinsic factor antibodies can occur. Type I antibodies block the binding of vitamin B12 to intrinsic factor, thereby preventing the uptake of vitamin B12. Type II intrinsic factor antibodies bind to intrinsic factor and prevent the attachment of intrinsic factor-cobalamin complex to receptors in the ileum. Both types of antibodies prevent absorption of cobalamin.

**Autoimmune Gastritis**

Autoimmune gastritis is also called type 1 chronic gastritis. Chronic gastritis type 2 is a similar disease caused by Helicobacter pylori infection. Over type patients with chronic H pylori infection can develop autoimmune gastritis. In autoimmune gastritis, the mucosal cells of the intestines are destroyed by autoantibodies to gastric parietal cells. Unlike pernicious anemia, autoantibodies to intrinsic factor are not presenting autoimmune gastritis. However, chronic autoimmune gastritis may progress to pernicious anemia. This process may take 20 to 30 years, which suggests that autoimmune gastritis is an early phase of pernicious anemia.

AGPA antibodies occur in about 90 percent of patents with pernicious anemia, 30 percent of first-degree relatives of patents with pernicious anemia, and they are also seen in up to 50 percent of adults and 18 percent of children with Helicobacter pylori infection. In addition, AGPA antibodies are seen in patients with various autoimmune endocrinopathies, including autoimmune thyroid disease. 2-8 percent of normal elderly subjects may also have AGPA antibodies, and the incidence of atrophic gastritis increases with age.

**Resources:**

Vjay Kumar, Pernicious Anemia, Medical Laboratory Observer, February 2007: 28-30.

autoimmunedisease.suite101.com/article.cfm/autoimmune_atrophic_gastritis
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