Perspective

iMedPub Journals
www.imedpub.com

Journal of Autoimmune Disorders
ISSN 2471-8513

2022
Vol.8 No.1:009

Autoimmune Dermatitis disease associations and hepatobiliary disorders

Ralph Pfeiffer*

Department of Internal Medicine, Naval Hospital of Crete, Crete, Greece

***Corresponding author:** Nathan Levitsky, Department of Medicine, University of Rochester Medical Center, Rochester, New York, Email: ralpfeiffer@hotmail.com

Received date: December 04, 2021, Manuscript No: IPADO-22-13072; **Editor assigned date:** December 09, 2021, PreQC No. IPADO-22-13072 (PQ); **Reviewed date:** December 23, 2021, QC No. IPADO-22-13072; **Revised date:** December 28, 2021, Manuscript No. IPADO-22-13072 (R); **Published date:** January 03, 2022, DOI: 10.36648/2471-8513.8.1.009

Citation: Pfeiffer R (2022) Autoimmune Dermatitis disease associations and hepatobiliary disorders. J Autoimmune Disord Vol.8 No.1: 009

Description

The role of vitamin B 12 in human immunity is still obscure. There are few studies reporting changes observed in immune parameters after vitamin B 12 administration in patients with pernicious anemia. However, some of these studies were case reports or inadequately designed clinical trials. The most important function of vitamin B 12 is in DNA synthesis where it is necessary for cell replication. In this process vitamin B 12 acts with folic acid. It is believed that vitamin B 12, which has a role in cell division, also acts as a modulator of human immunity; it facilitates the production of T lymphocytes recruited in cellular immunity, restores abnormally increased CD4/CD8 ratio and maintains the count of lymphocyte subgroups in the normal range. An in vitro experimental study demonstrated that vitamin B 12 was implicated in concanavalin Adependent T cell production and pokeweed mitogendependent immunoglobulin synthesis in B cells.

Absolute numbers of CD3 and CD19 significantly increased after cyanocobalamin administration, but CD10 and CD7 were slightly elevated. The levels of serum IgG, IgA, and IgM were significantly elevated. These observations are consistent with the proposition that vitamin B 12 favors humoral and cellular immunity. Lymphocyte apoptosis can be prevented by vitamin B 12 administration and may thus improve immunologic abnormalities observed in pernicious anemia.

Previous studies suggested that ineffective hematopoiesis caused by vitamin B 12 deficiency concerned primarily CD8+ cells. In our patients with pernicious anemia, decline in absolute numbers of lymphocytes and CD8+ cells was partially restored by vitamin B 12 administration. However, the role of CD8+ in this regard warrants further studies. The cyanocobalamin-mediated recovery from depressed NK cell function in our vitamin B 12 patients are in accordance with another study where lymphocyte and NK cell functions were completely restored and serum levels of vitamin B 12 returned to normal after 1 year of vitamin B 12 treatment reported that increase in absolute numbers of CD3–, CD16+, and CD57+ cells (these cells have strong NK cell activity) after vitamin B 12 treatment had augmented the antitumor activity.

Autosomal Dominant Retinitis Pigmentosa

Rounding out the list of causes of B12 deficiency is food-B12 malabsorption, which is the leading cause of B12 malabsorption —especially in elderly patients. In our own studies in which we have followed more than 300 patients with a documented B12 deficiency, food-B12 malabsorption accounts for about 60% to 70% of the cases of B12 deficiency in elderly patients, whereas pernicious anemia accounts for only 15% to 25%. In our study of 172 hospitalized patients with B12 deficiency (median age, 70), 53% had food-B12 malabsorption.

Food-B12 malabsorption is a syndrome characterized by the inability to release B12 from food or intestinal transport proteins, particularly in the presence of hypochlorhydria, in which the absorption of “unbound” B12 is normal. As various studies have this syndrome is defined by B12 deficiency in the presence of sufficient food-B12 intake and normal Schilling test results, which rules out pernicious anemia. In theory, indisputable evidence of food-B12 malabsorption comes from using a modified Schilling test, which uses radioactive B12 bound to animal proteins (eg, salmon, trout) and reveals malabsorption when the results of a standard Schilling test are normal. Some authors have speculated about the significance of B12 deficiency related to food-cobalamin malabsorption, because many patients have only mild clinical or hematological features. Several of our patients, however, have had significant features classically associated with pernicious anemia, including polyneuropathy, confusion, dementia, medullar-combined sclerosis, anemia, and pancytopenia. Nevertheless, the partial nature of this form of malabsorption might produce a more slowly progressive depletion of B12 than does the more complete malabsorption engendered by disruption of intrinsic factor–mediated absorption. The slower progression of depletion probably explains why mild, preclinical deficiency is associated with food-B12 malabsorption more often than with pernicious anemia.

Multiple Autoimmune Syndromes

Pernicious anemia is the most common cause of vitamin B12 deficiency. Vitamin B12 deficiency has many causes; the term

"pernicious anemia" applies only to the condition associated with chronic atrophic gastritis. A recent population survey revealed that 1.9 percent of persons more than 60 years old have undiagnosed pernicious anemia. Earlier studies suggested that pernicious anemia is restricted to Northern Europeans.2 However, subsequent studies have reported the disease in black and Latin-American subjects, with an earlier age of onset in black women. Although the disease is silent until the end stage, the underlying gastric lesion can be predicted many years before anemia develops. Pernicious anemia was first described by Thomas Addison in 1849. The anemia was linked to the stomach by Austin Flint in 1860 and named pernicious anemia soon thereafter. Successful treatment of the anemia with cooked liver suggested that it was caused by the lack of an extrinsic factor that was found in liver (later identified as vitamin B12) and an intrinsic factor in gastric juice. Although pernicious when first discovered, the disease is now controlled by treatment with vitamin B12. The discovery of a serum inhibitor of intrinsic factor (later found to be an autoantibody to intrinsic factor) and of autoantibodies to parietal cells laid the foundation for the immunologic explanation of the underlying gastritis that causes pernicious anemia.

Only serum vitamin B12 level was measured in the study to assess cobalamin status. The cutoff points of cobalamin deficiency were 220 ng/L with the initial fluorometric method, and 180 ng/L with the later chemiluminescent immunoassay. Many studies in the last decade have shown that serum cobalamin is sensitive in symptomatic patients, but is less reliable among asymptomatic patients. Levels of 2 serum cobalamin-dependent metabolites, methylmalonic acid and homocysteine, are more sensitive indicators of cobalamin deficiency. The levels of these metabolites rise early in the course, often preceding symptoms, and progress as deficiency worsens. These measurements are now widely used in western countries to identify patients, especially those presenting with atypical or neuropsychiatric features in the absence of hematologic changes and with serum cobalamin levels in the low-normal range.