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Pernicious anemia: Autoimmunity and the vitamin B12 puzzle.

Fontenay Raphael*

Department of Medicine, Medical College of Wisconsin, USA

Correspondence to: Fontenay Raphael, Department of Medicine, Medical College of Wisconsin, USA, E-mail: fontenay@hotmail.com

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Introduction

Pernicious anemia is a complex and often misunderstood condition that sits at the intersection of nutrition, immunology, and hematology. While anemia is commonly associated with iron deficiency, pernicious anemia stems from a deficiency in vitamin B12—an essential nutrient for red blood cell formation and neurological function. What makes pernicious anemia particularly intriguing is its autoimmune origin, where the body sabotages its own ability to absorb this vital vitamin. Pernicious anemia is a type of megaloblastic anemia caused by impaired absorption of vitamin B12 due to the lack of intrinsic factor—a protein secreted by the stomach lining that binds to B12 and facilitates its absorption in the small intestine. Without intrinsic factor, even a diet rich in B12 cannot prevent deficiency [1].

The term “pernicious” reflects the condition’s historical severity before effective treatments were developed. Left untreated, it can lead to irreversible neurological damage, cognitive decline, and severe anemia. At the heart of pernicious anemia lies an autoimmune response. The immune system mistakenly attacks the stomach’s parietal cells, which produce intrinsic factor, or directly targets intrinsic factor itself. This leads to chronic atrophic gastritis and a cascade of malabsorption issues [2].

A rare congenital form of pernicious anemia can also occur in infants due to inherited defects in intrinsic factor production. The cornerstone of treatment is vitamin B12 supplementation, which bypasses the need for intrinsic factor: Typically given weekly at first, then monthly for maintenance. Effective in some cases, especially when intrinsic factor is partially functional. Alternative delivery methods for maintenance therapy. Treatment is lifelong, and regular monitoring is essential to prevent relapse and manage symptoms. Vitamin B12 is crucial for maintaining the myelin sheath that protects nerve fibers. These symptoms may persist even after B12 levels are corrected, especially if treatment is delayed. Early intervention is key to preventing permanent damage. Autoantibodies against intrinsic factor and parietal cells are detectable in many patients, making them useful diagnostic markers. This autoimmune attack is often associated with other autoimmune disorders such as type 1 diabetes, thyroid disease, and vitiligo.Vitamin B12 deficiency affects multiple systems, and symptoms can be subtle or severe: Fatigue and weakness, Pale or jaundiced skin [3].

Misdiagnosis or delayed diagnosis can lead to unnecessary procedures and prolonged suffering. Neurological symptoms may appear even before anemia is detected, making early diagnosis critical. Diagnosing pernicious anemia involves a combination of blood tests and clinical evaluation: Reveals macrocytic anemia with large red blood cells. Typically low, Confirms autoimmune origin, Supports diagnosis [4].

Socially, patients may struggle with fatigue, cognitive issues, and dietary restrictions. Awareness and education are vital to improving quality of life and reducing stigma. In some cases, a bone marrow biopsy may be performed to rule out other causes of megaloblastic anemia. Pernicious anemia primarily affects older adults, with peak incidence between ages 60 and 80. It is more common in individuals of Northern European descent and slightly more prevalent in women [5].

Conclusion

Understanding the autoimmune mechanisms may also shed light on related conditions and lead to more targeted therapies. Though treatable, pernicious anemia can impose significant costs due

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to frequent medical visits, diagnostic testing, and lifelong supplementation.

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