DUAL ETIOLOGY OF CHRONIC HYPERCOBALAMINEMIA: MACRO-VITAMIN B12 INTERFERENCE AND INFLAMMATORY PROTEIN OVEREXPRESSION IN A PATIENT WITH SJÖGREN'S SYNDROME

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Persistent hypercobalaminemia often triggers extensive investigations to rule out malignancies. However, elevated levels can result from analytical interference or increased cobalamin-binding proteins. We report a 73-year-old female with vitamin B12 levels exceeding 2000 nanograms per liter for over a decade. Her history included Sjögren syndrome with antinuclear antibodies and lupus anticoagulant positivity, fibromyalgia, and hypertension treated with olmesartan medoxomil. Laboratory showed diluted vitamin B12 of 4911 nanograms per liter, normal homocysteine (11.4 micromoles per liter), and normal methylmalonic acid (16.0 micrograms per liter). Normal functional markers confirmed absence of true deficiency and supported pseudohypercobalaminemia. To investigate the suspected presence of macro-vitamin B12, polyethylene glycol precipitation was performed. Post-polyethylene glycol vitamin B12 was 1672 nanograms per liter, showing 66% reduction. A reduction exceeding 50% is diagnostic for macro-vitamin B12, an artifact caused by immunocomplexes between vitamin B12 and immunoglobulins. However, residual value remained elevated compared to reference range (165 to 695 nanograms per liter). This suggests dual etiology: interferential component from macro-vitamin B12 and true increase in cobalamin-binding proteins specifically haptocorrin or transcobalamin. The chronic inflammatory state in Sjögren syndrome, with B-cell hyperactivity and interferon pathway activation, likely stimulates production of these transport proteins. This case highlights the importance of polyethylene glycol precipitation to identify artifacts, while recognizing that residual elevated values may reflect underlying inflammatory processes. In autoimmune diseases with persistent hypercobalaminemia, clinicians should consider both analytical interference and inflammation-driven increases in binding proteins.