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Short Communication

Unusual Presentation of Vitamin B1 Deficiency Mimickig Heolytic Anemia

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Abstract:

Vitamin B12 deficiency is most often associated with megaloblastic anemia, glossitis, and neurological manifestations. However, in rare presentations, it may mimic hemolytic anemia due to ineffective erythropoiesis and intramedullary destruction of red blood cells. We present a short communication on an adult patient who exhibited pallor, jaundice, elevated lactate dehydrogenase (LDH), indirect hyperbilirubinemia, and reticulocytosis—features initially suggestive of hemolytic anemia. Peripheral smear revealed macrocytosis with hypersegmented neutrophils, and serum vitamin B12 levels were markedly reduced. Following parenteral vitamin B12 supplementation, rapid hematological recovery and normalization of hemolysis parameters were observed. This case highlights the importance of considering vitamin B12 deficiency in the differential diagnosis of hemolytic anemia–like presentations, particularly in populations at risk of nutritional deficiency. Early recognition can prevent unnecessary investigations, avert neurological sequelae, and ensure prompt, cost-effective treatment.

Key words: vitamin b12 deficiency, hemolytic anemia, megaloblastic anemia & diagnostic confusion

Introduction

Vitamin B12 (cobalamin) deficiency is a common cause of macrocytic anemia, primarily seen in older adults and individuals with malabsorption syndromes or strict vegetarian diets. Classical features include fatigue, pallor, glossitis, and neurological deficits such as paresthesia and ataxia. However, in rare instances, B12 deficiency may present with signs suggestive of hemolysis, including elevated lactate dehydrogenase (LDH), indirect hyperbilirubinemia, and low haptoglobin.

We present a case of a 45-year-old female who was referred to the hematology department for evaluation of suspected hemolytic anemia. The patient reported increasing fatigue, mild jaundice, and dark urine over the past three weeks. Physical examination revealed pallor, mild icterus, and glossitis, but no hepatosplenomegaly.

Laboratory investigations revealed the following: hemoglobin 7.6 g/dL, MCV 108 fL, reticulocyte count 3.2%, LDH 950 IU/L, total bilirubin 3.1 mg/dL (indirect 2.4 mg/dL), and haptoglobin <10 mg/dL. Peripheral smear showed anisocytosis, poikilocytosis, and hypersegmented neutrophils. Direct antiglobulin (Coombs) test was negative.

Further evaluation showed a markedly low serum vitamin B12 level at 85 pg/mL (normal >200 pg/mL). Folate levels were normal. The patient denied alcohol use, had no history of gastrointestinal surgery, and followed a vegetarian diet. Intrinsic factor antibodies were positive, supporting a diagnosis of pernicious anemia.

The patient was started on intramuscular vitamin B12 injections (1000 mcg weekly), with oral folic acid supplementation. Within four weeks, her hemoglobin levels improved to 10.5 g/dL, LDH normalized, and bilirubin levels decreased significantly. Her symptoms resolved entirely within two months.

This case highlights an uncommon but clinically relevant presentation of B12 deficiency that may mimic hemolytic anemia. The pseudo-hemolytic features are attributed to ineffective erythropoiesis and intramedullary destruction of red blood cell precursors, rather than true immune-mediated hemolysis.

Prompt recognition is crucial because misdiagnosis may lead to inappropriate therapies, such as corticosteroids or immunosuppressants, which carry unnecessary risks. Vitamin B12 deficiency is treatable and reversible, making awareness of its atypical presentations important for practicing clinicians.

Conclusion

Clinicians should consider vitamin B12 deficiency in the differential diagnosis of hemolytic-like anemia, especially in the presence of

macrocytosis and hypersegmented neutrophils. Early diagnosis and treatment can prevent complications and ensure full recovery.

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