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Case Report

Don't Worry It's Just a Vitamin B12 Deficiency!!

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Abstract

A thrombotic microangiopathy can be induced by vitamin B12 deficiency, known as pseudothrombotic microangiopathy which is manifested by hemolytic anemia, schistocytosis and thrombocytopenia. We report here an unusual observation concerning vitamin B12 deficiency revealed by a table of pseudothrombotic microangiopathy. The aim of this clinical case is to raise awareness of clinicians toward this differential diagnosis might spare patients from unnecessary and invasive therapies.

Keywords: Thrombotic microangiopathy; Pseudothrombotic microangiopathy; Schistocytosis; Vitamin B12 deficiency

Introduction

Pseudothrombotic microangiopathy is a life-threatening hematological condition that has been reported in very few cases of vitamin B12 deficiency. Until now, it is a rare clinical situation, not well documented in medical practice, and most often it is misdiagnosed with other diseases, especially true thrombotic microangiopathy [1]. Vitamin B12 deficiency is often unrecognized or not investigated because the clinical manifestations are 'subtle' and various like bone marrow failure, demyelinating nervous system disease, and neuropsychiatric manifestations [2]. Here we report a case of pseudothrombotic microangiopathy caused by vitamin B12 deficiency that is challenging because of the presentation with pancytopenia, macrocytosis, schistocytosis, thus mimicking an authentic thrombotic microangiopathy. Distinguishing between the two allows avoidance of unnecessary aggressive treatments that are burdened by morbidity and mortality.

Case Presentation

A 51-year-old man with no medical and surgical history, he did not take any medication prior to hospital admission, presented to the emergency department with severe asthenia and progressive dyspnea on minimal exertion of one-week duration. He denied fever, headache, abdominal pain, blurred vision, bleeding, and changes in bowel habits. On admission, the patient was afebrile, hemodynamically stable and eupneic with an oxygen saturation of 96% on room air, conscious, oriented, and cooperative but lack of energy was noted. At physical examination, he appeared pale with noninjected conjunctiva, and no ecchymosis, mucosal lesions, lymphadenopathy, purpura, or hepatosplenomegaly were detected. Neurologic examination showed no sensitive and motor abnormalities. Initial laboratory investigations showed a pancytopenia with hemoglobin 5.2 g/dL, Mean Corpuscular Volume (MCV) 110 fl, hematocrit 19.5%, White Blood Cell (WBC) 2,200/mm^3 (neutropenia 1000/ mm^3, lymphopenia 900/ mm^3), platelet count 100,000/mm^3, and corrected reticulocyte count reflected an inadequate reticulocyte response (1.6%) for the degree of anemia present. Peripheral smear revealed 12% of schistocytes, anisocytosis, and hypersegmented neutrophils. Direct coombs test was negative and haptoglobin was less than 0.08 g/L, lactate dehydrogenase (LDH) increase (more than 1995 U/L), and high bilirubinemia (21 μmol/L). Thrombotic microangiopathic anemia was suspected. The patient was kept under close surveillance. The renal function, coagulation studies, D-dimer, markers of inflammation, and liver function were normal. Vitamin B12 dosage was 82.6 pg/mL, folic acid 8.3 ng/mL, and TSH was normal. Then, bone marrow aspirate revealed hypercellular marrow with erythroid hyperplasia, and the erythroid precursors presented megaloblastosis. Our investigation was completed by serum parietal cell antibody and intrinsic factor antibody tests which proved to be positive. Esophagogastroduodenoscopy was performed and gastric biopsies were taken which showed evidence of atrophic gastritis. At the end of this biological assessment we concluded to a deficiency of vitamin B12 due to pernicious anemia with thrombotic microangiopathy as a result.

A week after beginning the vitamin B12 supplement with 1000 μg in intramuscular injections on a daily basis we observed a dramatic hematological and clinical improvement, the patient was discharged to home with weekly parenteral vitamin B12 for one month, then once a month with regular monitoring.

Discussion

Thrombotic microangiopathies, are defined by clinical and pathological characteristics. The clinical features include microangiopathic hemolytic anemia, thrombocytopenia, or even pancytopenia and organ injury while the pathologic characteristic is vascular damage that is manifested by arteriolar and capillary thrombosis [3]. Discriminating between this entity and pseudothrombotic microangiopathy, due to vitamin B12 deficiency, is of crucial importance, as the treatments are significantly different. Vitamin B12 deficiency is frequent in the general population and can affect up to 15% of the elderly [4,5]. Moreover, pseudothrombotic microangiopathy represents 2.5% of the hematological disorders related to vitamin B12 deficiency [6], although few cases have been reported. The pathogenesis of vitamin B12 deficiency induced thrombotic microangiopathy is not clearly known but many studies suggest that hyperhomocysteinemia may be implicated leading to clot activation and endothelial dysfunction, which results in fragmentation of erythrocytes to schistocytes [7,8]. Moreover, vitamin B12 deficiency increases red blood cell membrane rigidity that results in intramedullary hemolysis and entrapment in the microcirculation

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[9,10]. This intramedullary hemolysis is also the cause of the low reticulocyte count (ineffective erythropoiesis) [11]. In our case there is a low macrocytosis seen the depth of vitamin B12 deficiency, This could possibly be explained by the presence of abundant schistocytes as the small size of schistocytes decreases the mean corpuscular volume and increases the red cell distribution width [12]. LDH elevation may be explained by their release during intramedullary destruction of erythroblasts, Furthermore, erythroid progenitors produce little hemoglobin and only low levels of free bilirubin can be released [13]. In our case the combination of hemolytic anemia and the presence of schizocytes were in favor of thrombotic microangiopathy, but the low reticulocyte rate as well as the low macrocytosis, allowed us to correct the diagnosis and ask for the dosage of vitamin B12, thus allowing to conclude a pseudothrombotic microangiopathy secondary to vitamin B12 deficiency whose etiological investigations had led to pernicious anemia, thereby avoiding a heavy and unnecessary treatment.

Conclusion

The tip to remember in this clinical case is to maintain a high level of vigilance in front of atypical clinical manifestations of vitamin B12 deficiency associated with schizocytes and microangiopathic hemolytic anemia, because it may lead to confusion with another more urgent and critical diagnoses such as thrombotic thrombocytopenic purpura. It is essential for physicians to be aware of the various clinical features of pseudothrombotic microangiopathy and to assess the severity of this entity promptly and accurately, enabling simple, adequate and effective treatment.

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