Case Report

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Vitamin B12 deficiency in sickle cell patients at the Bamako sickle cell research and control center: About two cases

Abstract

Vitamin B12 deficiency, also known as hypocobalamemia refers to low blood levels of vitamin B12.

The authors report two cases of vitamin B12 deficiency in two sickle cell patients followed at the Bamako sickle cell research center.

The objective of this work is to describe this pathology in sickle cell patients and to provide a treatment plan.

Keywords: sickle cell anemia, vitamin B12 deficiency, center for reseach and fight against sickle cell disease

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Introduction

Sickle cell disease is a hereditary genetic disease characterized by the presence of sickle-shaped red blood cells and chronic anemia linked to the presence of its deformed red blood cells.[1]

In Mali the prevalence of healthy hemoglobin S carriage is on average 12% with rates varying from one region to another ranging from 6% for the northern regions and up to 20% for certain southern regions.[2]

Sickle cell disease in its homozygous form is characterized by the presence of chronic anemia. Acute anemia in sickle cell patients is also linked to malaria; bacterial infections and splenic sequestration. This anemia is the leading cause of death in sickle cell patients in several studies.[1,2]

The prevention of this anemia is based on the use of folic acid which would also have a role in the manufacture of DNA molecules and prevention against certain cancers.[3]

Additionally, folic acid supplements may mask vitamin B12 deficiencies, which can cause serious hematological, digestive and neurological problems.[4,5]

Biermer anemia, also called pernicious anemia, is a macrocytic anemia that does not respond to treatments used for other anemias. We report here two observations of anemia due to vitamin B12 deficiency or pernicious anemia in two women with sickle cell disease.

Observation 1

A young woman aged 22, female, followed for heterozygous sickle cell disease SC.

The current symptomatology is marked by acute anemia with asthenia, dizziness and headache following a vaso-occlusive crisis. She was transfused three times. The first blood count showed a hemoglobin level of 2.5 g/dl; the mean corpuscular volume at 108.3 f l; platelets: 550,000; reticulocyte levels 19.54%; white blood cells 881000.

Faced with macrocytosis without the notion of taking hydroxy-carbamide, a vitamin B12 dosage was requested which returned to 145.60 pg for normal values between (200-1100 pg).

Additional studies revealed that the patient has a folate level (B9) of 24.00 ng/ml, a normal creatinine level of 78 umol/L, à glomerular filtration rate estimated at 108 ml/min/1.73m², the reticulocyte rate was at 29.41%

The treatment was based on vitamin B12 in high doses up to 2 g per day.

Observation 2

Young woman of 29 years homozygous SS followed, female gender, the young woman presented in the postpartum period, episodes of headache fever and diffuse osteoarticular pain. After initial treatment with analgesics and rehydration, the evolution was marked by intense fatigue and mucocutaneous pallor on physical examination.

The blood count revealed that the young woman had a hemoglobin level of 3.6g/dl, MCV of 120.2 fl, white blood cells of 82,000/m³

After several transfusions for acute anemia, a vitamin B12 dosage came back with a level of 12.50 pg/ml. Folates: 12.91 for normal values included (5.21-20)

Discussion

Pathophysiology and clinical

In a young patient with sickle cell disease, the appearance of persistent anemia led to the diagnosis of vitamin B12 deficiency given the unfavorable clinical course despite repeated transfusions.

These two observations show that vitamin B deficiency can mimic the signs of hemolytic anemia. Vitamin B12 deficiency is common in people with increased demand, inadequate supply, and reduced absorption of vitamin B12.[6]

In sickle cell disease there is systematic folic acid supplementation and chronic hemolysis, which leads to a functional deficiency of vitamin B 12. In addition folic acid, from the treatment of sickle cell disease, if administered particularly in high doses, can mask vitamin B12 deficiency by completely correcting hematological abnormalities. In cases of vitamin B12 Folic acid may result in complete resolution of characteristic megaloblastic anemia, while allowing progression of potentially irreversible neurological damage (due to continued inactivity of methylmalonyl mutase). Thus, vitamin B12 status should be determined before folic acid is administered as monotherapy.[7]

Also repeated sickling in gastric blood vessels, leading to vascular microinfarctions, leads to a reduction in the functioning of gastric parietal cells leading to reduced production of intrinsic factor essential for the proper absorption of vitamin B12.[8]

It is difficult to suspect vitamin B12 deficiency in patients with sickle cell anemia because they usually present with microcytosis. Anemia is usually explained by the sickle cell process itself or by other associated factors or phenomenon such as folate deficiency. Chronic pain and vague neurological symptoms are common in both conditions.

According to several studies, vitamin B levels are low in sickle cell patients.[9,10] These subjects with sickle cell disease may be at higher risk of vitamin B deficiency.[9]

In these studies the proportion of sickle cell patients with unexplained low cobalamin levels was higher than in patients without sickle cell syndrome. Patients with sickle cell disease who had low cobalamin levels are younger and more likely to be male.[9]

The average age is respectively 28.6 and according to previous studies[9] which is what was observed in our two clinical cases.

His two clinical cases also note that none of the sickle cell patients had neurological manifestations as described in the literature.[9] Anemia, macrocytosis, thrombocytopenia and low white blood cell counts are most often found in the blood counts of patients with vitamin B12 deficiency.[9] In our two patients there was thrombocytosis associated with hyperleukocytosis probably linked to a coexisting infection.

Therapeutic aspects

The parenteral route is preferred for the treatment of vitamin B12 deficiency in symptomatic patients and high-risk situations. The generally proposed regimen is 1 mg/day of cyano-cobalamin for 1 week, then 1 mg/week for 1 month, then 1mg/month for a duration to be defined on a case-by-case basis.

The oral route seems to be an effective alternative in other situations (1-2 mg/day); it avoids the discomfort, inconvenience and costs of injections.[9]

Conclusion

Systematic screening for vitamin B12 deficiency seems necessary in the face of any anemia resistant to transfusion and folic acid in sickle cell patients.

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Contribution of the authors

All authors contributed in one way or another to the development of this article from start to finish.

Conflicts of interest

The authors declare no conflict of interest.

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