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Case Report

Growth Retardation and General Hypotonia Revealing Vitamin B12 Deficiency

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Abstract

Vitamin B 12 has important consequences for hematopoiesis and the central nervous system, its deficiency leads to clinical hematological, neurological and psychiatric manifestations.

We report the case of a ten months old male infant, exclusively breastfed, with a significant delay in the developmental and communicative reactions. Physical examination confirmed the pallor, the axial hypotonia and revealed that the child was below the 2nd percentile for weight and the 3rd percentile for length.

He had a hemoglobin level of 6.4 g/dL with an MCV of 100.2 fL, His serum vitamin B12 level was less than 50 pg/mL and serum folate level 20.4 ng/mL. No intrinsic factor antibodies were found. Hormonal and nutritional screening revealed no other deficiency.

Thalassemia screening resulted negative.

Maternal investigations showed anemia and maternal vitamin B12 deficiency. The research for anti-parietal antibodies of the stomach returns very positive.

Vitamin B12 supplementation permitted with weight gain and rapid improvement of interactions and muscle tone.

Introduction

Vitamin B 12 has important consequences for hematopoiesis and the central nervous system, its deficiency leads to clinical hematological, neurological and psychiatric manifestations.

However, most infants with B12 deficiency are born to women with low vitamin B12 levels and have been exclusively breastfed [1].

Patient and observation

We report the case of a ten months old male infant, was born after a normal full term (40 weeks) pregnancy with a weight of 2,850 g, length of 50 cm, and head circumference of 35 cm, who was hospitalized because of severe pallor and general hypotonia

The child was exclusively breastfed essentially because of anorexia

The parents reported a significant delay in the developmental milestones (he holds his head at the fourth month and acquires the sitting position at the seventh month) and communicative reactions (absence of social smile at the third month)

Physical examination upon admission confirmed the pallor, the axial hypotonia and revealed that the child was below the 2nd percentile for weight (7 kg), and the 3rd percentile for length (62 cm) head circumference was 42 cm,

He had a hemoglobin level of 6.4 g/dL with an MCV of 100.2 fL, a white blood cell count of 11,000/mm^3 and neutrophil count of 2,500/mm^3. His platelet count was 189,000/mm^3 and reticulocyte count was 140,000/ul.

His serum vitamin B12 level was less than 50 pg/mL (normal value 180-500 pg/mL) and serum folate level 20.4 ng/mL (normal value >3 ng/mL). Blood homocysteine was 8 μmol/L (normal value 4-15 μmol/L) and urinary methylmalonic acid 284 mmol/moL creatinine (normal value <5 mmol/moL creatinine).

No intrinsic factor antibodies were found. Hormonal and nutritional screening revealed no other deficiency.

Thalassemia screening resulted negative.

The myelogram shows an asynchronism of nucleocytoplasmic maturation of the erythroblastic line with megaloblastosis, which is in favor of a vitamin deficiency. In this context oral vitamin B12 supplementation is introduced. The development was rapidly favorable with weight gain, rapid improvement of interactions and muscle tone.

Maternal investigations showed a hemoglobin level of 9.4 g/dL with an MCV of 70.4 fL, serum ferritin level of 12ng/mL and serum vitamin B12 level of 101 pg/mL, the homocysteine increased to 42.3_ mol / L, confirming maternal vitamin B12 deficiency.

The research for anti-intrinsic factor antibodies was negative, and for anti-parietal antibodies of the stomach returns very positive

The examination of a gastric biopsy showed a fundal and interstitial atrophic gastritis. Helicobacter pylori research was negative.

Discussion

Vitamin B12 is only found in animal products such as meat, egg, fish and milk [2] except in newborns whose stock comes only from placental transfer [3].

The children of women with low vitamin B12 levels during pregnancy and lactation may have smaller stores of the vitamin at birth and its concentration in breast milk is likely to be low [4].

In babies exclusively fed with breast milk, the symptoms of vitamin B12 deficiency are observed in general between the 4th and the 12th months, although neonatal cases have also been reported, the clinical findings are nonspecific, The most common presentation symptoms are developmental retardation, pallor, weakness, vomiting and diarrhea and the most common clinical signs were irritability, apathy, hypotonia, motor retardation, decrease in deep tendon reflexes, encephalopathy, coma and microcephaly [5,6].

Young infants frequently have multiple deficiencies, particularly when they are exclusively breastfed; they develop hematological disease with anemia and pancytopenia in the most severe cases [7].

A recent review of childhood vitamin B12 deficiency found that the two-thirds of cases were due to an inadequate consumption of meat and other animal products or strict vegetarians and the quarter is due to maternal pernicious anemia [8].

Diverse recommendations exist for therapy of vitamin B12 deficiency in adults, and no clear guidelines are available for children, in our case, oral vitamin B12 supplementation was adopted, intramuscular replacement therapy is also possible [9], with an equivalent efficacy [10], it improved rapidly hematological values that return to normal, and the neurological signs progressively decrease.

However, in most of the severe cases described [8], some neurological problems may be permanent, What underlines the importance of maternal vitamin B12 supplementation during pregnancy especially, in the case of vegans, it should be greater than that usually given, and should be continued during lactation in order to avoid the development of persistent neurological problems in infancy.

Conclusion

In the infant, the association of neurological and haematological abnormalities, it is important to mention a vitamin B12 deficiency, especially if they are exclusively breastfed.

An early diagnosis and treatment may prevent the progression of neurological damage to irreversible deficits.

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