

Vitamin B12 Deficiency in an Exclusively Breastfed 7-Month-Old Infant Born to a Vegan Mother

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Abstract

Dietary vitamin B12 deficiency in infancy is rare, and most reported cases are breast fed infants of mothers who themselves are deficient in vitamin B12 as a result of strict vegetarian diet. Here we describe a case, a 7 month old male infant, presented with noisy breathing who was born to a vegan mother and was diagnosed as megaloblastic anemia and treated with intramuscular vitamin B12 injections. A few days after the start of therapy, his hemoglobin levels improved, and a clinical improvement was observed within few weeks.

Keywords: Megaloblastic anemia, Vegan mother, Vitamin B12 deficiency

INTRODUCTION

Vitamin B12 is a water soluble vitamin and plays a major role in human metabolic reactions. Humans are totally dependent on dietary vitamin B12. Microorganisms are the ultimate origin of cobalamin in the food chain and strictly vegetarian or macrobiotic diets do not provide adequate amounts of this essential nutrient. Vitamin B12 functions as a cofactor for isomerization of methylmalonyl-CoA to succinyl-CoA, an essential reaction in lipid and carbohydrate metabolism and to ensure the activity of methionine synthase, an enzyme that catalyses the methylation of homocysteine to form the essential amino acid methionine, which is important for protein and nucleic acid biosynthesis.¹ Dietary sources of vitamin B12 are almost exclusively from animal foods. Organ meats, muscle meats, sea food, poultry, and egg yolk are rich sources. Fortified ready-to-eat cereals and milk and their products are the important sources of the vitamin for vegetarians. Vitamin B12 deficiency leads to the accumulation of methylmalonic acid and homocysteine in blood and urine, and the onset of clinical hematological and neurological manifestations. Vitamin B12 deficiency in infancy may be due to an inborn error of absorption and metabolism, but most reported cases are breast fed infants of mothers who themselves are deficient in vitamin B12 as a result of

strict vegetarian diet.^{2,3} Here we describe a case, a 7 month old male infant, born to a strict vegan mother, with megaloblastic anemia due to deficiency of vitamin B12.

CASE PRESENTATION

This 7 month old male infant was born after a full term (40 weeks) cesarean delivery with birth weight 2.9 kg and was exclusively breast fed till 7 months of age. The infant presented with noisy breathing since 1 week and was hospitalized in view of pneumonia. On admission, he was found to be pale, with a weight of 6500 g (3rd-10th percentile), length of 69 cm (25th-50th percentile) and head circumference 41 cm (10th-25th percentile), blackish knuckle pigmentation of fingers and toes were seen (Figure 1). There was no lymphadenopathy and no organomegaly. Neurodevelopmental assessment was appropriate for age and mother dietary history was normal comprising of vegan diet.

He had a hemoglobin level of 7.8 mg/dl, white blood cell count of 6200 cells/cmm, hematocrit of 21%, ESR of 40, MCV of 100fl and Reticulocyte count of 0.3%. His peripheral smear showed macrocytosis, severe anisocytosis, poikilocytosis and hypersegmented neutrophils (Figure 2).

Occasional fragmented cells and tear drop cells were seen. His serum vitamin B12 level was 101 pg/ml (normal value: 200–800 pg/ml) and serum folate level was 24 ng/ml (normal value: 5–21 ng/ml). On the basis of these data, child was diagnosed as having megaloblastic anemia due to vitamin B12 deficiency and was treated with intramuscular injections of vitamin B12 at a dose of 1000 µg/day for 2 weeks and followed by once a month.

Few days after the start of therapy, his hemoglobin levels improved to 10 mg/dl, and a clinical improvement was observed after a few weeks (Figure 3). Hematological improvement was seen after 3 months (Figure 4).

DISCUSSION

Vitamin B12 deficiency usually occurs in infants born to vegan mothers and this is important as it is a preventable cause of neurodevelopmental delay. The average daily requirement for an infant is 0.5–0.6 µg/day. Vitamin B12 is freed from binding proteins in food through the action of pepsin in the stomach and binds to salivary proteins called cobalophilins, or R-binders. In the duodenum,

bound vitamin B12 is released by the action of pancreatic proteases. The released vitamin B12 binds to intrinsic factor produced by gastric parietal cells and is transported to the distal ileum. Within ileal cells, vitamin B12 associates with a major carrier protein, transcobalamin II, and is secreted into the plasma. Transcobalamin II delivers vitamin B12 to the liver and other cells of the body, including rapidly proliferating cells in the bone marrow and the gastrointestinal tract. In the absence of intrinsic factor, cobalamin is absorbed only very inefficiently by passive diffusion.⁴ Megaloblastic anemia due to cobalamin or folate deficiency is due to ineffective erythropoiesis. Vitamin B12 is necessary for DNA synthesis and its deficiency prevents cell division in the marrow. Due to deficiency of folate or vitamin B12, red blood cells become large with nuclear or cytoplasmic asynchrony, a characteristic of all megaloblastic anemias. Non specific manifestations of megaloblastic anemia include weakness, fatigue, failure to thrive and irritability. Other features seen are pallor, glossitis, vomiting and diarrhea. Neurologic symptoms include hypotonia, developmental delay, seizures, psychiatric changes and subacute combined degeneration of spinal cord.³ In peripheral smear, macrocytic red cells, hypersegmented



Figure 1: Photography showing pallor and blackish knuckle pigmentation in fingers



Figure 3: Photograph after treatment showing the disappearance of pallor and blackish knuckle discoloration in fingers

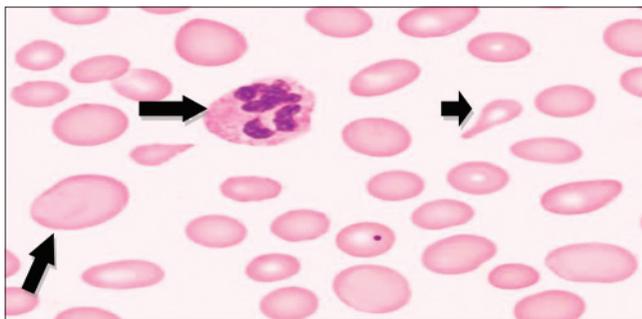


Figure 2: Peripheral smear showing Macrocytosis, Anisocytosis, Poikilocytosis and Hypersegmented neutrophils

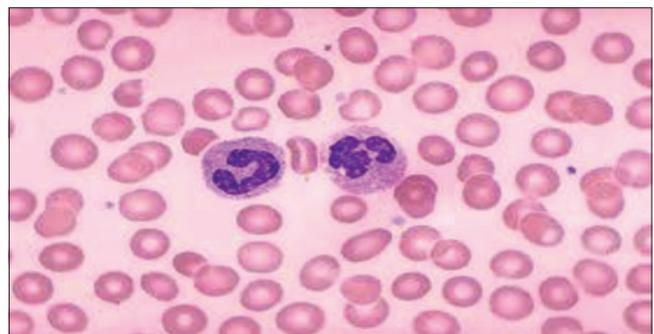


Figure 4: Peripheral smear after treatment showing normal neutrophils and RBCs

neutrophils, anisocytosis and poikilocytosis are seen. Reticulocyte count is low, elevated homocysteine and LDH levels in blood are seen.

In our case, child clinically had pallor and blackish knuckle pigmentation of fingers and toes. Smear showed macrocytosis, severe anisocytosis, poikilocytosis and hypersegmented neutrophils with occasional fragmented cells and tear drop cells. Reticulocyte count was low. Serum vitamin B12 levels were low with normal folate and ferritin levels.

In India, where people tend to be vegetarians, vitamin B12 deficiency during pregnancy is common,⁵ and the infants of vitamin B12 deficient mothers can suffer from mild developmental delay and skin pigmentation. Vitamin B12 supplementation in pregnant and lactating women, and the use of complementary vitamin B12-rich foods in infants aged >6 months are useful in preventing megaloblastic anemia but in a developing country like India, economic problems may profoundly impact the consumption of meat and other animal products. Unlike infants, even if the serum levels of vitamin B12 are low, pregnant women generally show no related signs or symptoms because they usually consume large amounts of vegetables containing high folate concentrations that masks the hematological effects of vitamin B12 deficiency.⁶

If vitamin B12 deficiency in infants is not treated early, it leads to developmental delay, developmental regression and convulsions. Cognitive and developmental delay may persist despite of adequate therapy even though the hematological Problems may disappear completely.⁷

CONCLUSION

This case shows the importance of vitamin B12 supplementation in pregnancy and lactation especially in case of vegans, whose infants are more likely to be affected than other babies. In infants diagnosed with anemia, it is important to rule out megaloblastic anemia, as it is a preventable cause of developmental delay. In a developing country like India, more measures should be taken to diagnose vitamin B12 deficiency and prevent vitamin B12 deficiency in pregnancy by supplementations. Early detection of megaloblastic anemia in infants is important for early intervention.

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