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Case Report

Megaloblastic Anemia Secondary to Vitamin B12 Deficiency in an 11-Month-Old Infant: A Case Presentation

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Abstract

Background: Megaloblastic anemia caused by vitamin B12 and folate deficiency is the most common cause of macrocytic anemia in the pediatric age group, leading to impaired DNA synthesis and ineffective erythropoiesis. Although iron deficiency anemia is the most common cause of anemia worldwide, any macrocytic anemia with pancytopenia should be considered megaloblastic anemia. ***Case presentation***: An 11-month-old female infant presents with a 1-month history of repeated nonprojectile vomiting, decreased oral intake, significant weight loss, frequent bowel movements, and decreased urine output over the last 5 days. When she was admitted to hospital, she was pale and moderately dehydrated. Her investigation showed severe anemia: hemoglobin was 6.4 g/dL, MCV was 105.8 fl, and thrombocytopenia was present. Peripheral blood smear showed anisopoikilocytosis. The infant was exclusively breastfeeding and had a limited amount of table food intake. The infant has a history of maternal anemia during pregnancy. The infant was treated at the hospital with parenteral B12, and there was rapid clinical and hematological improvement; she was discharged home, and at 1 month follow-up, her hemoglobin and platelet count had significantly improved. ***Conclusion***: Any infant presenting with macrocytic anemia and pancytopenia should be considered to have megaloblastic anemia, which is most often caused by vitamin B12 or folate deficiency, even in the absence of neurological symptoms. Early diagnosis and treatment lead to excellent recovery and prevent complications.

Keywords: Breastfeeding; Infant; Macrocytosis; Megaloblastic anemia; Vitamin B12 deficiency.

فقر الدم الضخم الثانوي لنقص فيتامين B12 في طفل عمره 11 شهرا: عرض حالة

الخلاصة

الخلفية: فقر الدم الضخم الناتج عن نقص فيتامين B12 أو حمض الفوليك هو سبب مهم لفقر الدم الماكروسيت لدى الرضع والأطفال. في الممارسة السريرية، يجب أن يثير فقر الدم الماكروسي المرتبط بضعف الخلايا في خط خلوي واحد أو أكثر الشكوك حول فقر الدم الضخم الفصول ويسرع في تقييم إضافي. **عرض الحالة**: طفلة تبلغ من العمر ثمانية أشهر ظهرت لها تاريخ لمدة شهر من القيء غير المقذوف المتكرر، وسوء تناول الفم، وفقدان الوزن، وخلال الأيام الخمسة الماضية، تكرار حركة الأمعاء وانخفاض في إخراج البول. عند دخولها، كانت شاحبة وتعاني من جفاف متوسط. أظهرت الفحوصات المخبرية فقر دم ماكروسي حاد مع هيموغلوبين 6.4 جرام/ديسيلتر، ومتوسط حجم الجسميات 105.8 fL، وخلط الصفيحات، بينما ارتفع عدد خلايا الدم البيضاء مع اللمفاوي. أظهرت مسحة الدم الطرفية تعقيدا الخلايا مع تعقيم الخلايا العضلية وصورة دم ثنائية الشكل. تم تشخيص فقر الدم الضخم المحتمل المرتبط بنقص فيتامين ب12 بناء على النتائج الدموية، وتاريخ التغذية، وتاريخ الأم لفقر الدم أثناء الحمل، والاستجابة العلاجية اللاحقة. تم علاج الرضيع بفيتامين B12 الوريد مع تحسن سريري سريع. بحلول اليوم السابع، ارتفع الهيموغلوبين من 7.8 جرام/ديسيلتر، وعند متابعة شهر ارتفع الهيموغلوبين إلى 10.3 جرام/ديسيلتر وتحسن عدد الصفائح الدموية إلى 333×10^9/لتر. **الاستنتاجات**: يجب النظر في نقص فيتامين B12 المحتمل لدى الرضع الذين يعانون من فقر الدم الماكروسيت ونقص الصفيحات الدموية (تجلط الصفيحات)، خاصة في الرضع الذين يرضعون فقط من الثدي الذين يعانون من تغذية تكميلية محدودة وربما نقص تغذية الأم. التعرف المبكر والعلاج السريع يمكن أن يؤدي إلى تعافي دموي ملحوظ وقد يساعد في منع مضاعفات خطيرة.

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INTRODUCTION

Poor DNA synthesis causes a collection of conditions known as "megaloblastic anemia." At every stage of development, red blood cells (RBCs) are larger than usual, and the cytoplasm and nucleus of erythrocytes mature asynchronously. As cell divisions continue, the delayed nuclear development becomes more noticeable. Giant metamyelocytes and neutrophil bands are frequently seen in the bone marrow, and myeloid and platelet precursors are also impacted. There may be accompanying thrombocytopenia and, in some cases, leukopenia. Large, frequently oval red blood cells with a higher mean corpuscular volume are noticeable on the peripheral blood smear. Many neutrophils have more than five lobes, a feature of hypersegmented neutrophils. Most childhood megaloblastic anemia cases are due to folate or vitamin B12 deficiency, which are essential for DNA synthesis. Inborn metabolic abnormalities may sometimes cause certain anemias. Malnutrition-related

megaloblastic anemia remains an important global health problem [1]. In exclusively breastfed infants, vitamin B12 deficiency may occur when maternal stores are low, and it can also result from intestinal malabsorption such as Crohn's disease, celiac disease, chronic pancreatitis, or terminal ileum resection [2,3]. In children, vitamin B12 malabsorption due to intrinsic factor deficiency is very uncommon. Inadequate intake, malabsorption, or increased requirements can also lead to folate deficiency. Megaloblastic anemia is characterized by precursor cells, megaloblasts in the bone marrow, and macrocytic red blood cells in the peripheral circulation. These megaloblasts develop as a result of inefficient erythropoiesis and poor DNA synthesis. Additional alterations include thrombocytopenia, a low reticulocyte count, an elevated mean corpuscular volume (MCV>100 fL), a raised mean corpuscular hemoglobin concentration (MCHC), and low hemoglobin and erythrocyte counts. A definitive diagnosis is supported by low serum folate or vitamin B12 levels when available. [4]. Treatment depends on the underlying deficiency and usually includes vitamin B12 supplementation, with parenteral therapy initially followed by maintenance treatment; folate replacement is given when folate deficiency is present [5].

Case Presentation

An 11-month-old female infant presented with a 1-month history of vomiting about 20 times per day, non-projectile, yellowish in color, sometimes containing milk without blood, not related to feeding, with decreased urine output and poor oral intake, but no fever. She was initially treated for a presumed urinary tract infection, with no clinical improvement. In the last 5 days, she developed frequent bowel motions, 15 times per day, and large amounts of greenish-colored stool without blood. Her condition is associated with a weight loss of about 600 g over 1 month. There was no history of abnormal body movements or irritability. There was no history of chronic malabsorption, previous surgery, or chronic drug use. She was a 38-week full-term baby delivered by elective C/S. Her delivery weight was 2,000 g, small for gestational age; no NICU admission was needed. The mother had a history of anemia during pregnancy. She was exclusively breastfed and received limited complementary feeding, mainly fruits and some vegetables. Her mother was on a normal diet and was not vegetarian. Developmental history was appropriate for age. There was a history of consanguinity, and the family lived in a crowded rural area. On physical examination, the infant was afebrile, pale, and moderately dehydrated. Clear chest, soft abdomen, and no organomegaly. Neurological examination was normal, with intact gait, tone, power, and reflexes. She had tachycardia with a heart rate of 135 beats per minute. The rest of the systemic examination was unremarkable. She was admitted for correction of dehydration, and investigations were performed. In Table 1, hematological analysis showed hemoglobin 6.4 g/dL, platelet count 74 × 10^9/L, and WBC 16.3 × 10^9/L with lymphocytosis 76%.

Table 1: The results of the complete blood picture and blood film.

Parameter	Result	Normal Range
White Blood Cells (WBC)	16.3×10^9/L (H)	3.5–10.0
Lymphocytes (LYM#)	12.6×10^9/L (H)	0.9–5.0
Lymphocytes %	76.4% (H)	15– 50
Red Blood Cells (RBC)	1.76×10^{12}/L (L)	3.5–5.5
Hemoglobin (HGB)	6.4 g/dl (L)	11.5–16.5
Hematocrit (HCT)	18.7% (L)	35– 55
Mean Corpuscular Volume (MCV)	105.8 fl (H)	75–100
Mean Corpuscular Hb (MCH)	36.5 pg (H)	25–35
MCHC	34.4 g/dl	31–38
RDW%	30.3% (H)	11–16
Platelets (PLT)	74×10^9/L (L)	130–400
Reticulocyte Count	7%	-

Footnotes (Peripheral Blood Film Interpretation): RBCs: Show a dimorphic picture; some RBCs are hypochromic microcytic with anisopoikilocytosis, oval cells, tear drops, and irregular contracted RBCs. Others are macrocytic with target cells. WBCs: Leukocytosis with lymphocytosis; no immature cells seen. Platelets: Reduced in number (Thrombocytopenia). Impression: Dimorphic anemia with lymphocytosis and thrombocytopenia. Suggesting Hb-electrophoresis with serum B12 and folate levels for follow-up.

Her MCV is 105.8 fl and MCHC is 34.4 g/dl. A peripheral blood smear showed macrocytosis with anisopoikilocytosis and target cells. Reticulocyte count was 7%. Blood culture showed no bacterial growth, and liver and renal function tests, as well as serum electrolytes, were within normal ranges. Serum ferritin was elevated at 353 ng/mL. C-reactive protein was elevated at 48 mg/dL, suggesting a concurrent inflammatory or infectious process, while thyroid function tests were normal. Urine and stool examinations were unremarkable, except for 4 pus cells in urine; abdominal ultrasound was normal. Chest radiography was normal. Serum vitamin B12 and folate levels were not measured because these tests were inconsistently available in the local setting. She was treated with intravenous vitamin B12 for 3 consecutive days, followed by administration every other day, in addition to multivitamin syrup. By the 7th day of admission, her general condition had improved, with satisfactory hydration, increased activity, better oral intake, and no

vomiting or diarrhea. Hemoglobin increased to 7.8 g/dL. She was discharged on oral multivitamins and oral vitamin B12. After 1 month, she showed further clinical improvement, with hemoglobin rising to 10.3 g/dL and platelet count increasing to 333 × 10⁹/L.

Ethical approval and informed consent

This case report was conducted in accordance with the ethical standards of the institutional and national research committee and with the principles of the Declaration of Helsinki. Written informed consent for publication of this case report and accompanying images was obtained from the patient's parent/legal guardian. All identifying information has been omitted to protect patient confidentiality.

DISCUSSION

Megaloblastic anemia in infancy is most commonly caused by vitamin B12 or folate deficiency, both of which are essential for DNA synthesis and effective erythropoiesis [6]. In the present case, the infant had macrocytic anemia with thrombocytopenia and peripheral smear findings of macrocytosis with anisopoikilocytosis, which were consistent with megaloblastic anemia [7]. In infants, vitamin B12 deficiency is often related to inadequate dietary intake or maternal vitamin B12 deficiency, particularly in exclusively breastfed infants with limited complementary feeding [8]. Although the mother in this case was not vegetarian, maternal anemia during pregnancy may suggest unrecognized vitamin B12 deficiency and reduced fetal stores [8]. Neonates have limited vitamin B12 stores, and exclusive breastfeeding without adequate complementary feeding may predispose infants to deficiency within the first months of life. Vitamin B12 deficiency in infancy is clinically important because it may cause neurodevelopmental delays, hypotonia, and delayed motor milestones. However, neurological manifestations are not always present at the time of diagnosis, as in the present case. This makes early recognition important even when hematological findings appear before neurological signs. Recent studies emphasize that infants of mothers with low vitamin B12 intake, restrictive diets, or malabsorption are at increased risk of deficiency, and adequate maternal nutrition during pregnancy and lactation is essential to prevent infant deficiency [5]. The infant also presented with gastrointestinal symptoms, including persistent vomiting and diarrhea. These symptoms may be due to vitamin B12 deficiency, but they could also indicate a concurrent gastrointestinal illness, so caution is advised in interpretation. Vitamin B12 deficiency may present these symptoms, but they could also indicate a concurrent gastrointestinal illness, so caution is advised in interpretation. [6,8]. The marked macrocytosis, thrombocytopenia, and elevated ferritin level favored megaloblastic anemia over iron deficiency anemia. Inflammatory changes may also contribute to elevated ferritin, which can complicate interpretation of iron status. Although confirmatory serum vitamin B12 and folate levels were not available, the clinical picture, feeding history, maternal history, hematologic findings, and response to treatment supported a probable diagnosis of vitamin B12 deficiency–related megaloblastic anemia [1]. The infant showed rapid clinical and hematologic improvement after parenteral vitamin B12 therapy. Hemoglobin increased within one week of treatment and continued to improve at the one-month follow-up, with normalization of the platelet count. This treatment response further supported the working diagnosis. Recent studies highlight that infants born to mothers with low dietary intake of B12, strict vegetarian or vegan diets, or malabsorption disorders are at significantly higher risk of developing megaloblastic anemia and neurodevelopmental delays. Ensuring adequate maternal B12 intake through dietary sources or supplementation is essential for preventing deficiency-related neurological complications in infants. Early recognition and supplementation in both mother and infant can markedly improve neurodevelopmental outcomes [7].

Conclusion

Vitamin B12 deficiency is a probable and treatable cause of acute megaloblastic anemia with thrombocytopenia in infancy. Although iron deficiency is more common in this age group, the presence of macrocytic anemia with cytopenia should prompt consideration of megaloblastic anemia. Exclusively breastfed infants remain at risk even when the mother is not vegetarian, especially if maternal anemia or inadequate complementary feeding is present. Nonspecific symptoms, particularly when accompanied by gastrointestinal complaints, may delay diagnosis and lead to unnecessary treatment. Early recognition and prompt vitamin B12 supplementation can produce rapid hematologic and clinical improvement. Increased awareness among pediatricians is essential to ensure timely diagnosis and prevent complications.

Conflict of interests

The authors declared no conflict of interest.

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Data sharing statement

The data that supports the findings of this study are available from the corresponding author upon a reasonable request.

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