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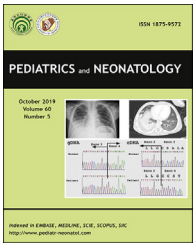

Letter to the Editor

A 2-year-old twin boy with pancytopenia caused due to nutritional cobalamin deficiency

Dear Editor,

Although nutritional vitamin B_{12} deficiency has become an extremely rare condition in developed countries,[1] its wide spectrum of neurological or hematological symptoms can sometimes be challenging.[2]

We report a case of a 2-year-old twin A boy suffering from pallor and decreased activity for 1 month and low-grade fever for 1 week. Physical examination revealed delay in language and motor development, hypotonia, and body weight of 9.5 kg (<3rd%). There were no glossitis or skin changes, no surgical history or other chronic conditions, and no family history of genetic disorders. Initial hemogram showed pancytopenia and macrocytic anemia with the following results: hemoglobin level 3.6 g/dL, red cell distribution width 27.7% (normal, 11.5%–14.5%), white blood cell count 4.05 $\times$ 10^9/L (segmented neutrophil, 22%; lymphocytes, 69%; monocytes, 6%), platelet count 48 $\times$ 10^9/L, and mean corpuscular volume 104 fL. Neutrophils in the peripheral blood smear demonstrated hypersegmentation of the nuclei with bizarre shapes (Fig. 1A). Other laboratory parameters were as follows: aspartate transaminase level 69 U/L (normal, 15–37 U/L), alanine transaminase level 39 U/L (normal, 16–63 U/L), and direct/total bilirubin level 0.22/1.56 mg/dL (normal, 0–0.3/0–1.0 mg/dL). Due to a high lactate dehydrogenase level of 3295 IU/L (normal, 85–227 IU/L) and pancytopenia, bone marrow studies were performed that revealed hypercellularity with erythroid hyperplasia and dysplasia (Fig. 1B–D); cytogenetic study showed a normal karyotype. Vitamin B_{12} level was 90 pg/mL (normal, 206–678 pg/mL), whereas that of folic acid was 7.9 ng/mL (normal, 1.5–16.9 ng/mL). Serum levels of methylmalonic acid (MMA) and homocysteine were high, with values of 1.4 μM (normal, <1 μM) and 15.83 μM (normal, 5.08–15.39 μM), respectively. The antiparietal cell antibody test showed negative results.

The patient was administered 1 mg of vitamin B_{12} injection every day for 7 days, and 19 days after the initiation of treatment, the muscle power and hemogram results showed significant improvement.

The patient and his fraternal twin B brother were breastfed until the age of 14 months. Due to misinformation perceived by the parents, the twin's diet did not include animal protein derived from meat, eggs, or formulas. His twin B brother also had a low vitamin B_{12} level of 89 pg/mL but presented with developmental delay with a normal hemogram and no macrocytosis. Tandem mass tests of both children in the neonatal period showed normal results. The nutritional concept of the parents was reinforced to include animal-source foods for their twins.

Both these fraternal twins presented with hypotonia and delays in language and motor development; however, only twin A developed severe pancytopenia, emphasizing the heterogeneity of vitamin B_{12} deficiency.

An earlier study reported that among 41 children with cobalamin deficiency (mean age, 12 months), the most common clinical manifestations included motor retardation, hypotonia, pallor, skin hyperpigmentation, apathy, and anorexia.[2] Another study reported that approximately 10% of patients with vitamin B_{12} deficiency had pancytopenia or other hematological disorders.[3] In contrast, 28% of patients with a neuropsychiatric abnormality caused due to cobalamin deficiency were reported to have no anemia or macrocytosis.[4] Thus, measuring the accumulation of MMA and homocysteine levels can help diagnose vitamin B_{12} deficiency.[4]

In conclusion, it is necessary to evaluate the levels of vitamin B_{12}, MMA, and homocysteine in children with a history of inadequate animal food source, global

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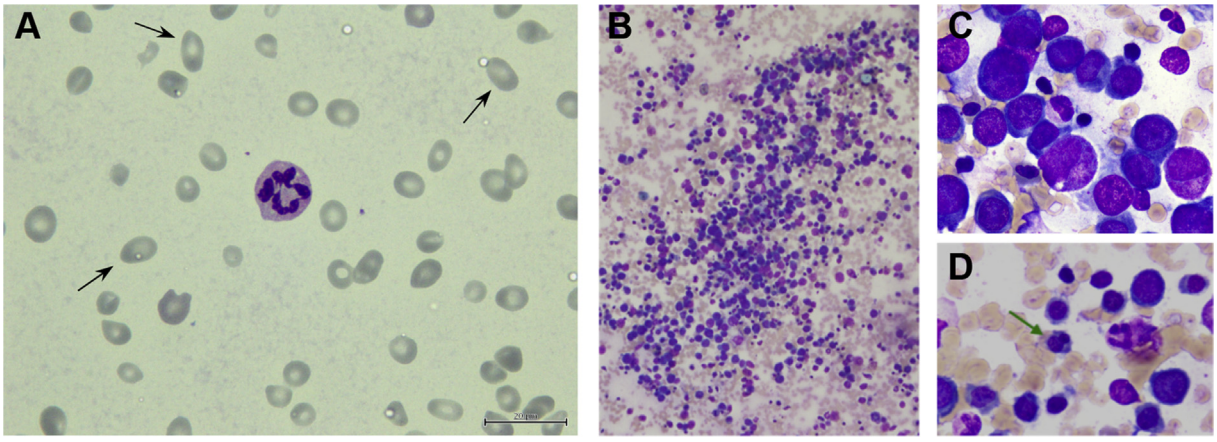

Figure 1 Hematological findings of vitamin B_{12} deficiency in twin A. **(A)** Peripheral blood smear (Liu stain; 100×) revealed hypersegmentation of neutrophils, marked anisocytosis, poikilocytosis, and large oval erythrocytes (arrows). **(B—D)** Bone marrow smear revealed hypercellularity **(B)** (Liu stain; 100×) with megaloblastosis **(C)** and occasional erythroid dysplasia (green arrow) **(D)** (Liu stain; 1000×).

developmental delay, with or without macrocytic anemia, and/or other cytopenias. Megaloblastic anemia caused due to vitamin B_{12} deficiency must not be mistaken for myelodysplastic syndrome or even acute erythroid leukemia.[5]

Conflicts of interest

None declared.

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Shu-Huey Chen
Department of Pediatrics, Shuang Ho Hospital, Ministry of Health and Welfare, Taipei Medical University, New Taipei, Taiwan
Department of Pediatrics, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan
Taipei Cancer Center, Taipei Medical University, Taipei, Taiwan

Yung-Ting Kuo
Department of Pediatrics, Shuang Ho Hospital, Ministry of Health and Welfare, Taipei Medical University, New Taipei, Taiwan
Department of Pediatrics, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan

Li-Ping Tsai
Department of Pediatrics, Taipei Tzu Chi Hospital, Xindian, New Taipei, Taiwan
Department of Pediatrics, School of Medicine, College of Medicine, Tzu Chi University, Hualien, Taiwan

Yen-Lin Liu*
Department of Pediatrics, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan
Taipei Cancer Center, Taipei Medical University, Taipei, Taiwan
Department of Pediatrics, Taipei Medical University Hospital, Taipei, Taiwan

James S. Miser
Taipei Cancer Center, Taipei Medical University, Taipei, Taiwan
College of Medical Sciences and Technology, Taipei Medical University, Taipei, Taiwan

*Corresponding author. Department of Pediatrics, Taipei Medical University Hospital, 252 Wuxing St, Taipei, 11031, Taiwan. Fax: +886 2 6639 7181.
E-mail address: ylliu@tmu.edu.tw (Y.-L. Liu)

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