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Acquired severe aplastic anemia in a 1-year-old child after ingesting vitamin-containing cocoa paste

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Dear Editor

Acquired aplastic anemia is a life-threatening bone marrow failure.The annual incidence is reported to be 2 per million(2). It is characterized by damage to the bone marrow by stimulation of the immune system and release of cytokines due to an unknown antigenic stimulus such as viral infections, drugs, and chemical exposures. In this letter, we would like to draw your attention to a case of acquired aplastic anemia in a pediatric patient that may have been caused by the consumption of vitamin-enriched cocoa paste. A 15-month-old male patient presented to the pediatric emergency department with bruising on his arms. He had no medical history other than atopic dermatitis. His parents were unrelated and he had no siblings.Physical examination revealed no developmental malformations, lymphadenopathy, and hepatosplenomegaly with diffuse petechiae and oral mucosal bleeding. Blood count revealed Wbc:7760u/L, hemoglobin:8.6gr/dL, MCV:76fL, platelets:1000/uL new:190/uL; reticulocytes: 0.18%. Renal function tests were normal, liver function tests were high (ALT:250).The patient's vitamin B12, folic acid levels, and serologies for hepatitis A, B C, human immunodeficiency virus, parvovirus B-19, Epstein-Barr virus, cytomegalovirus, and herpes virus, which were checked to exclude viral causes, were negative. Further investigation of the patient's history revealed that she had consumed three boxes of cocoa paste containing flower honey, pollen, royal jelly, locust bean molasses, propolis, L-ornithine, histidine, colostrum, calcium gluconate, L-tryptophan, zinc gluconate, ascorbic acid, and various over-the-counter vitamin supplements purchased from the Internet, which were quite inexpensive compared to their equivalents. No blasts were detected in the flow cell analysis of the bone marrow aspirate. The pathology result of the bone marrow biopsy was reported as "hypocellular bone marrow, erythroid and granulocytic lineages were markedly decreased, megakaryocytic lineages were not observed, 80% adipose tissue" and the granulocyte count was <200 µL, platelet count was <20,000/µL, and reticulocyte count was <20,000/µL in the blood smear.Intrafamilial donor screening for bone marrow transplantation (BMT) was initiated. Diepoxibutane test and Fanconi subgroup gene test, paroxysmal nocturnal hemoglobinuria (PNH) panel, myelodysplastic syndrome (MDS) and other gene analyses showed no positive findings. During two months of clinical follow-up, the patient, who had oral mucosal bleeding and a platelet count <10,000/uL, received platelet suspension every two days for the first two weeks, then twice weekly on average, and erythrocyte suspension support every three weeks on average. ALT levels, which were high during the first 7 days of hospitalization, gradually decreased and returned to normal. Immunosuppressive therapy (IST) was initiated in the patient for whom a fully matched family donor could not be found. Horse-derived antithymocyte globulin (ATG) was administered and cyclosporine A (CSA) was started. Eltrombopag could not be added to the regimen because the patient was younger than 2 years. In the 5th month of IST treatment, the patient's blood counts returned to normal.

BMT is the first-line treatment for aplastic anemia in patients with an HLA-matched family donor, and overall survival rates have exceeded 90% (2). In children without a fully matched family donor, response rates to IST range from 75-85% at 3-6 months (3). The combination of equine ATG and CSA in combination with L-thrombopaq has shown the highest treatment success in IST.However, there is a risk (10-40%) of clonal hematopoietic disorders such as MDS, AML and PNH in IST (4). For patients who have failed multiple cycles of IST, an incompletely matched unrelated donor should be considered as salvage therapy.In our case, the response to IST treatment was successful (5,6). Nevertheless, it is thought-provoking that a paste purchased on the Internet with the intention of a healthy diet can cause aplastic anemia.With this letter to the editor, we wanted to draw attention to products taken as vitamin supplements during childhood that have not been adequately studied and whose effects are not well known.

Keywords

Severe aplastic anemia, vitamin, acquired aplastic anemia, immunosuppressive therapy, bone marrow transplantation.

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