Case Report

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High Serum Iron in Human Parvovirus-Induced Aplastic Crisis in Iron Deficiency Anemia

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Key Words

Erythroblastopenia
Iron deficiency anemia
Parvovirus

Abstract

Recent study has shown that the human parvovirus (HPV) is a causative agent of aplastic crisis in hemolytic anemias. In subjects without shortened red blood cell life span, HPV is not associated with symptomatic anemia. A case of iron deficiency anemia revealed by HPV linked to erythroblastopenia is described in a child. On admission, the serum iron was not decreased and other blood chemistry tests were also not consistent with the diagnosis of iron deficiency anemia. Abrupt cessation of erythropoiesis induced by HPV infection appeared to prolong plasma iron clearance, which elevated serum iron concentration.

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Human parvovirus (HPV) infection can induce transient and intense erythroblastopenia in patients with hemolytic anemia such as congenital spherocytosis, sickle cell anemia and homozygous β -thalassemia, but the reports of erythroblastopenia associated with HPV infection are very few in patients with other anemias, including iron deficiency anemia (IDA) [1–3]. In this report we present the clinical findings of a patient with severe IDA revealed by human parvovirus linked to erythroblastopenia.

Case Report

A 13-year-old boy was admitted to the Japanese Red Cross Nagoya First Hospital on October 21, 1986, because of fatigue, anemia and fever of 4 days duration. There was no past history of anemia. He had neither respiratory symptoms nor skin rash. Physical examination revealed a thin, pale boy in no acute distress. There were no enlarged lymph nodes or hepatosplenomegaly. The hemoglobin level was 6.8 g/dl, hematocrit 24.4% and reticulocyte count 0%. The anemia was microcytic (mean corpuscular volume 52.5 μ m³), hypochromic (mean corpuscular hemoglobin 15.1 pg), and with anisocytosis and poikilocytosis. The white blood cell count was 2.6×10^9 /l with a differential of 21 % segmented neutrophils, 10% bands, 7% monocytes, 61% lymphocytes and 1% eosinophils.

The platelet count was 280×10^9 /l. A bone marrow smear revealed marked erythroblastopenia (0.8%) with normal granulocytic maturation (54%), megakaryocytes and scattered giant

pronormo-blasts. The serum iron was 179 µg/dl, total iron binding capacity 355 µg/dl and serum ferritin was 88.6 µg/l.

The reticulocytopenia and leukopenia rapidly resolved over the next week. On October 28, the reticulocyte count was 4.8%, but his anemia did not improve after the recovery of erythropoiesis. The transient red blood cell aplasia and normal level of serum iron prompted the investigation for the possible underlying hemolytic anemia and viral infection. The tests performed in this regard were for osmotic fragility, isopropanol precipitation, heat stability, G-6-PD screen, haptoglobin screen, and hemoglobin electrophoresis. All the results were normal. The red blood cell life span measured by the use of a radioisotopic technique was also normal (half-life span: 31.6 days). He had no evidence of underlying hemolytic anemia.

However reinvestigations performed on November 9 revealed the cause of the underlying anemia. At that time, his laboratory findings were typical of IDA. The serum iron was 10 µg/dl, total iron binding capacity 427 µg/dl and serum ferritin was below 10 µg/dl. After the administration of iron, his anemia improved rapidly. Viral studies revealed recent HPV infection by the presence of anti-HPV IgM. A serum sample drawn 2 months after his hospitalization was positive for anti-HPV IgG.

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Results

The patient presented here provides two points for consideration. Firstly, because red blood cell aplasia linked to HPV infection had been recognized mostly in patients with chronic hemolytic anemia with shortened red cell life span, it was considered that the very active marrow found in these circumstances might be necessary for the replication of HPV [4]. The report showed that erythroid progenitors in a patient with IDA who had normal red blood cell life span could be a target of HPV infection.

The second point is that, although on admission, his peripheral blood smear was typical of IDA, the blood chemistry tests were contrary to the diagnosis. In conditions of impaired erythropoiesis such as aplastic anemia, plasma iron clearance is slow, plasma iron turnover is decreased and the serum iron concentration is elevated [5]. In our patient, probably abrupt cessation of erythropoiesis caused by HPV infection prolonged plasma iron clearance, which elevated serum iron concentration. If we had noticed this fact, we could have avoided the unnecessary tests performed to investigate the possibility of an underlying hemolytic anemia.

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