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TRANSIENT ERYTHROBLASTOPENIA AND PARVOVIRUS B19 INFECTION IN A HEALTHY NEWBORN

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ABSTRACT

A newborn baby with severe anemia and reticulocytopenia in whom parvovirus B19 antibodies were detected is described. Anemia was treated with a single transfusion of red blood cells and it did not recur. She is now 15 months old with normal hematologic data. We would like to emphasize via the present patient that parvovirus B19 infection has to be considered in newborns presenting with severe anemia especially if reticulocytopenia is also present. The diagnosis is of importance in evaluating the patient and in treatment planning.

Key Words: Anemia, Newborn, Reticulocytopenia, Human Parvovirus B19.

SAĞLIKLI BİR YENİDOĞANDA GEÇİCİ ERİTROBLASTOPENİ VE PARVOVİRÜS B19 ENFEKSİYONU

ÖZ

Bu yazıda, ağır anemi ve retikülositopeni bulunan, parvovirüs B19 enfeksiyonu saptanan sağlıklı bir yenidoğan olgusu sunuldu. Anemi tek transfüzyonla düzeldi ve tekrarlamadı. Hasta şu anda 15 aylık olup normal hematolojik bulgulara sahiptir.

Bu olgu nedeniyle ağır anemi ile başvuran yenidoğanlarda retikülositopeni varsa parvovirüs B19 enfeksiyonunun düşünülmesi gerektiğini vurgulamak istiyoruz. Tanı konulması, hastanın değerlendirilmesi ve tedavisinin planlanması açısından önemlidir.

Anahtar Kelimeler: Anemi, Yenidoğan, Retikülositopeni, Parvovirüs B19.

INTRODUCTION

According to current knowledge, transient erythroblastopenia of childhood (TEC) is an acquired and benign disorder occurring in children.¹ It is characterized by a transient suppression of erythropoiesis in marrow, resulting in reticulocytopenia in blood. Treatment is usually supportive and most patients recover completely. The etiology of TEC is still being debated. However, recent laboratory methods have demonstrated the causative role of human parvovirus B19 (PV B19).² PV B19 is a DNA virus and its target of infection is erythroid progenitors.^{3,4} It usually induces an erythroblastopenia crisis in patients suffering from chronic hemolytic disorders.⁵⁻⁸ There are occasional cases of hematological abnormalities caused by the virus in healthy subjects.^{9,10} The virus can also be transmitted vertically from pregnant woman to fetus and may cause various perinatal complications.^{11,12} In this report TEC caused by transplacentally transmitted PV B19 in a healthy newborn is presented.

CASE REPORT

The patient was delivered via an uneventful pregnancy to a 33-year-old woman. Her birth weight was 2910 gram. She was found to be pale on the 4th day after delivery on routine examination. The rest of the physical examination was unremarkable. The laboratory evaluation revealed severe anemia with a hemoglobin value of 10.1 g/dl. Count and differentiation of leukocytes, and count and morphology of platelets were normal. No hemolysis was seen on the peripheral smear. Reticulocyte count was 0.04%. There was no ABO or Rh incompatibility and the direct antiglobulin test was negative. Abdominal and transfontanel ultrasonography disclosed no abnormalities. Further evaluations for fetomaternal hemorrhage, placental hemorrhage, or intrauterine infections in terms of TORCH (toxoplasma, rubella, cytomegalovirus, and herpes simplex) were negative according to clinical and laboratory analyses. Serologic tests of IgM and IgG against PV B19 were positive in both patient and mother. It was discovered from the medical history that at the 37th week of pregnancy the mother had had mild to moderate cold-like symptoms.

On consecutive controls hemoglobin level decreased from 10.1 to 6.4 g/dl and the patient required transfusion of red blood cells once on the 8th day in hospital. The hemoglobin level remained stable thereafter. Reticulocyte count start-

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There were no sources of support

ed to increase beginning from the 14th day. No therapy was required in the mother. IgG antibodies for PV B19 remained detectable in the patient on day 450 after hospital admission. During the follow-up period of 15 months the anemia did not recur. The patient is now in good health with normal hematologic data.

DISCUSSION

Transient erythroblastopenia of childhood is an acute form of anemia characterized by a transient red cell aplasia in marrow.¹ Marrow aplasia results in reticulocytopenia in blood. The occurrence of normochromic normocytic anemia accompanied with reticulocytopenia should prompt consideration of TEC. There are strong data to suggest that PV B19 plays an important role in the etiology.² Hence, PV B19 should be included in the evaluation of patients with TEC. This virus selectively infects the erythroid progenitor cells^{3,4} and induces an erythroblastopenia crisis usually in patients suffering from chronic hemolytic disorders such as hereditary spherocytosis, paroxysmal nocturnal hemoglobinuria, and sickle cell anemia.⁵⁻⁸ There are only a few reports of PV B19 infection in subjects who do not have an underlying hematological disease.^{9,10} In addition, the virus can be transmitted transplacentally to the fetus and may cause perinatal complications, primarily nonimmune fetal hydrops, severe fetal anemia, cardiomegaly, intrauterine growth retardation, and erythroblastopenia during the neonatal period.^{11,12} In the present case, there was no underlying hematological disease. The virus is thought to have reached the fetus via the transplacental route and caused characteristic anemia observed during the early neonatal period. The cold-like symptoms in the mother during the third trimester were probably related to the virus. What is more, the fact that viral serology was found to be positive in both the mother and the newborn baby points to the strong possibility of transplacental transmission. Finally, the presence of positivity of viral serology simultaneously with TEC stage again indicated its causal relation to the development of transient erythroblastopenia.

The laboratory diagnosis of the virus can be made by serologic tests or via PCR of viral genome. Viral DNA testing is recommended for patients who are immunocompromised. Moreover, PCR analysis is not available in every medical center. For immunocompetent patients, like ours, serum IgM testing, as also applied here, is sufficient for laboratory diagnosis of the virus.⁷

Severe anemia in a newborn constitutes a hematological emergency. It calls for rapid and detailed scrutiny. However, reticulocytopenia is noteworthy in TEC. In such cases, considering PV B19 infection early on will help avoid overinvestiga-

tion since it can be diagnosed by a simple test, namely serology. Obviously, overinvestigation occurred in our case, but we will take into consideration that point in subsequent patients.

Human parvovirus B19 can demonstrate a wide spectrum of clinical presentations.⁷ There are cases free of symptoms, as well as cases requiring complicated therapies, i.e. immunoglobulins, erythropoietin, or plasmapheresis.³ However, the disease is usually self-limited and in 90% of patients a single transfusion is sufficient.¹ In the present case, only one transfusion was needed. During the follow-up period of 15 months, hemoglobin never went below the level achieved after transfusion. Moreover, reticulocytosis was observed after the 14th day and thereafter hemoglobin started to go up gradually.

Absence of congenital abnormalities and rapid resolution of anemia in this case excluded the possibility of the chronic form of isolated erythroid hypoplasia described by Diamond and Blackfan.

In brief, we report here an observation of TEC with a classical clinical course. The patient is presented because she is one of the few cases of TEC caused by PV B19 infection. Moreover, transient anemia attributable to transplacentally transmitted virus in an immunocompetent patient is even rarer. However, the principal aim of this presentation is to emphasize that, beside the other reasons, PV B19 infection should be considered in severe anemia during the neonatal period, especially if reticulocytopenia is also present. The diagnosis is of importance in evaluating the patient and will help the clinician in treatment planning.

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