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CASE REPORT

A cause of bicytopenia in infancy: CMV

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Abstract

Bicytopenia is frequently observed in the pediatric population as a result of transient bone marrow suppression, which may occur following vaccination or as a consequence of viral infections, including influenza, Epstein-Barr virus (EBV), and cytomegalovirus (CMV). Symptoms of CMV infection, one of these agents, vary according to age and host immune competence. Although it is usually asymptomatic in healthy children, it can cause various infectious clinical presentations. In immunocompromised patients, it may cause pneumonia, retinitis, hepatitis, fever, thrombocytopenia, and leukopenia. Here, we present a four-month-old girl who was investigated for bicytopenia and subsequently diagnosed with CMV infection.

Keywords: cytomegalovirus; bicytopenia; neutropenia; thrombocytopenia

Introduction

Cytomegalovirus (CMV) belongs to the Herpes virus family and is also known as Human Herpes Virus-5 (HHV-5). The virus settles in endothelial cells, fibroblasts, myocytes, macrophage cells and remains latent. Transmission is possible through all body fluids such as respiratory droplets, urine, tears, breast milk and transplacentally [1]. Although the incubation period is four-six weeks on average, it can last up to four months. It may remain latent after viremia and show periodic reactivation especially when the immune system is suppressed [2].

Bicytopenia may occur due to benign and malignant causes and may be seen as a permanent or transient condition depending on the etiology. When studies in the field of pediatric bicytopenia are examined, etiologic causes including hematologic malignancies, aplastic anemias, secondary bone marrow suppression and megaloblastic anemia have been found with different frequencies in different studies [3–5].

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Case Report

A four-month-old girl presented with a rash on both feet and legs. It was learned that the rash started about 10 days ago, was rare on the dorsum of the feet in the first week, then progressed to the legs, there was no other symptom, the rash became more prominent and progressed to the upper parts of the legs, and the patient was referred to the hospital.

It was learned that our patient was born at term, had no hospitalization during or after the neonatal period, no prolonged jaundice, was breastfed only, used vitamin D drops, and received routine childhood vaccinations as well as the first doses of rotavirus and meningitis vaccines in accordance with her age.

Systemic examination of the patient revealed that pale red petechial eruptions on bilateral lower extremities and dorsum of the feet which did not fade with pressure. Complete blood count resulted as hemoglobin (Hb) 11,9 g/dL, white blood cell count (WBC) 8960/mm^3, absolute neutrophil count (ANC) 920/mm^3, platelet count (PLT) 82000/ mm^3 and peripheral blood smear, biochemistry, C-reactive protein (CRP), iron parameters, vitamin B12, thyroid function tests (TFT), Epstein-Barr virus (EBV) and CMV were then sent for etiologic investigation. No atypical cells were seen in the peripheral blood smear. Aspartate aminotransferase (AST) 78 U/L, alanine aminotransferase (ALT) 53 U/L, CRP negative, ferritin-vitamin B12-TFT were in normal range, EBV negative and CMV IgM 1.58, CMV IgG 21.94 were detected. CMV Polymerase Chain Reaction (PCR) was analyzed from serum due to CMV IgM positivity. 223 copies/ml was detected. Complete blood count taken at the 5th week after the onset of complaints showed Hb 11,5 g/dL, WBC 7560/mm^3, ANC 620/mm^3, PLT 93000/mm^3, AST 55 U/L, ALT 41 U/L. Although biochemistry parameters normalized, bicytopenia still persists. We followed up our patient with weekly complete blood count and bicytopenia was etiologically attributed to CMV.

Discussion

Peripheral cytopenia is defined as a decrease in blood cells (erythrocytes, leukocytes or platelets). Although it varies according to age, on average, Hb <11 g/dL, ANC <1000-1500/mm^3 and platelet count <150 000/ mm^3 is considered low. Bicytopenia is a decrease in two different cell series. Its etiology shows a wide distribution. It may be due to bone marrow suppression especially as a result of viral infection or may develop as a result of bone marrow suppression due to malignancy, drugs, vaccines, chemotherapy or radiotherapy [3,4,6]. Viral infections including influenza, varicella, measles, rubella, hepatitis A and B, CMV, EBV, parvovirus B19, adenovirus, and coxsackie may cause neutropenia and thrombocytopenia by decreasing production and increasing destruction [7,8].

Though acquired CMV usually proceeds with asymptomatic viremia in healthy people, it may progress critically in newborns, premature infants, adolescents, and children with immunosuppression. In immunosuppressed patients, it may progress with findings such as fever, leukopenia, thrombocytopenia, increased liver enzymes and atypical lymphocytosis [1]. Congenital CMV may progress with findings like sepsis, pneumonia, prolonged jaundice, hepatosplenomegaly, petechiae-purpura, periventricular calcification, chorioretinitis, sensorineural hearing loss and cytopenia in preterm infants. We didn't consider late presentation of congenital CMV in our patient because he had no history of prolonged jaundice or hospitalization in the neonatal period, no hepatosplenomegaly, normal complete blood count in the neonatal period, passed hearing test, no microcephaly and normal neurodevelopment.

Acquired CMV infection in healthy individuals doesn't require antiviral therapy, but only symptomatic supportive treatment and follow-up is sufficient. However, in congenital CMV infection, antiviral agents including valganciclovir, ganciclovir, foscarnet, and cidofovir are used in case of immunosuppression and cranial system effects including chorioretinitis and periventricular calcification [1]. Our patient was in the group who did not require treatment because we didn't think of congenital CMV or because she did not have immunosuppression.

The onset of our patient's symptoms shortly after the fourth-month vaccination initially suggested vaccination-related bone marrow suppression, however the positive CMV PCR results shifted the diagnosis towards bone marrow suppression secondary to CMV infection. The PCR copy number was not high enough, but we attribute this to the fact that one month had passed since the initial complaints and the viral load had decreased when the sample was taken for PCR.

As a result, CMV is a highly asymptomatic agent in healthy children unlike other viral infections, we recommend screening for CMV in cases of bicytopenia even if the clinical findings are inconsistent.

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