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Aplastic Anemia Following Epstein Barr Virus Infection In A Child: A Case Report

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Abstract
A 4 year old girl presented with aplastic anemia secondary to Epstein-Barr virus (EBV) infection. Severe aplastic anemia has been reported in children following EBV infection. Review of literature, suggests that steroid therapy is indicated in such patients. Some studies have shown acyclovir as a treatment modality. However, our patient, improved without any antivirals or steroids, just with supportive therapy.

Keywords: Aplastic anemia, Epstein - Barr virus

Introduction

Majority of cases of primary EBV infection in infants and young children are clinically silent. Very few patients with EBV infection, develop hematological complications such as severe anemia, thrombocytopenia, neutropenia and occasionally aplastic anemia (1, 2, and 3). There are few cases reported in literature of EBV infection induced aplastic anemia in children. Considering the severity of presentation in such cases, we must be aware of the occurrence of aplastic anemia following EBV infection.

Case Report

A 4 year old girl from Western India presented with high grade, continuous fever since 1 month, history of frequent falls with swelling over trauma sites and vomiting since 15 days and cough since 10 days. She had decreased appetite and fatigue, with no significant past history. She was intermittently treated by multiple physicians for fever since the past 1 month before presenting to us.

On general physical she was febrile (102◦F), pale and sick looking, with palpable lymph nodes in bilateral cervical and inguinal regions which were firm, non-matted and non-tender. She had a tender swelling over the sternum, with ecchymosis of the overlying skin. Her pulses were bounding with a rate of 140/min, respiratory rate of 50/min without distress and blood pressure was 96/54 mm Hg. There were no dysmorphic features, skeletal abnormalities or skin pigmentations. On Anthropometry, weight/age was between -1 SD and Median and weight/height was between -1SD and Median. Liver and spleen were not palpable, air entry was equal in both lung fields without any adventitious sounds. Child was conscious and oriented.

Laboratory results on admission, showed pancytopenia, with hemoglobin of 2.8 gm/dl (hematocrit 7.4 %), total WBC counts of 500/mm3 (neutrophils 60%, lymphocytes 38%, monocytes 1%, eosinophils1 %) and platelet count of 19,000/mm3. Peripheral blood smear showed normochromic, normocytic picture with few microcytes and ovalocytes, mild hypochromasia, no blast cells or any other abnormal cells. Reticulocyte count was 0.5 % (corrected Retic count of 0.03%). Sickling test was negative, with no evidence of hemoglobinopathy on HPLC. C-reactive Protein (CRP) was 102 mg/dl. Liver and renal function tests were within normal limits. Serum/Blood LDH was 346 U/l (reference

range: 230-460 U/l). Fine needle aspiration of cervical lymph node showed reactive lymphoid hyperplasia. Bone marrow biopsy showed hypocellular marrow with bony trabeculae, marrow spaces and adipocytes. Marrow spaces mainly showed fibrosis and spindle shaped cells, fibroblasts, histiocytes and scanty hematopoietic cell lineage consisting of immature erythroid, lymphoid cells and few megakaryocytes.

Chest X-ray was normal and without any mediastinal mass. The patient was started on IV piperacillin-tazobactum, vancomycin and fluconazole, as per the protocol for febrile neutropenia, after sending 3 site blood cultures .The child was put on oxygen by nasal prongs at 4 liters/min with dobutamine infusion and given multiple packed red cell transfusions over 3 to 4 days. Investigations for tuberculosis were negative. Blood & urine cultures were sterile. Ultrasonography of the chest swelling showed localized hematoma.

In the ward, fever spikes reduced gradually and child was afebrile by 12th day of hospital stay. Child did not have any bleeding episodes in the ward. Repeat blood counts done at 2 weeks showed improvement, with a hemoglobin of 9 gm/dl, total WBC counts of 6810/mm3 (neutrophils 54%, lymphocytes 31%, monocytes 6%, eosinophils 3%, basophils 3%) and platelet count of 2,51,000/mm3. Child improved clinically with a betterment of appetite.

Parvovirus B19 IgM and IgG were negative. EBV IgM to viral capsid antigen was negative. EBV IgG antibody to viral capsid was positive (>750 U/ml). Genetic studies for Fanconi anemia, dyskeratosis congenita and congenital amegakaryocytic thrombocytopenia could not be done due to financial issues with the patient.

Child was discharged after 3 weeks of hospital stay.

Discussion

The case described here is a case of aplastic anemia which was seen secondary to Epstein-Barr virus infection. Several studies have shown aggressive disease following Epstein-Barr virus infection as was seen in a case report by Khan et al 2013(4).In our study the child had lymphadenopathy and fever which assisted in diagnosis of Epstein-Barr virus infection, but literature shows that some patients did not show any signs of EBV infection .(3)

Literature shows that EBV infection has been treated with antivirals like acyclovir (3, 4).Also, ATG and steroids have been used for severe cases (3,4). In our study, however, the child improved without antivirals and ATG.

The pathophysiology of acquired aplastic anemia is an immune mediated process that involves cytotoxic T lymphocytes which target bone marrow and cause hematopoietic dysfunction (5). Studies show that EBV is implicated in this process of inducing the proliferation of virus-specific cytotoxic T lymphocytes and decreases the CD4+/CD08+T cell ratio (5).

Hence, it is understood that EBV induced aplastic anemia may be much more common than literature shows mainly because most patients are asymptomatic for EBV infection or does not have stereotypical symptoms. Viral causes of aplastic anemia should be considered in young, previously healthy patients.

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