Introduction

EBV infection can occur in any part of the world and infection is common in early childhood and the second peak can occur during late adolescence, most of the EBV infections in infants and young children are asymptomatic. Whereas around 75% of infections in adolescents may present with infectious mononucleosis, common symptoms of this infection are sore throat, malaise, headache, abdominal pain and common signs are lymphadenopathy, fever, pharyngitis, hepatomegaly, splenomegaly, rash. Some adults (less than 0.5%) have been reported to have life-threatening hematological complications like subcapsular splenic hemorrhage or splenic rupture. Other hematological complications: Coombs test positive hemolytic anemia, severe thrombocytopenia, neutropenia, and aplastic anemia. Many cases of ITP turning to aplastic anemia have been found in the literature. IgM and IgG antibodies directed against the Epstein-Barr viral capsid antigen (VCA) are usually present at the onset of clinical illness because of the long viral incubation period. IgM levels wane approximately three months later; thus, they are a good marker of acute infection. IgG VCA antibodies persist for life and are a marker of EBV infection.

Case Report:

A seventeen year-old male presented with complaints of easy fatigability of 4 months duration, abdominal pain of 1 months duration and fever for 5 days. He had decreased energy associated with fatigue, decreased appetite. His sister also noticed pallor. The easy fatigability was progressive and was associated with palpitation on mild exertions because of which he had to take rest. His abdominal pain was mainly over epigastrium and left upper quadrant of abdomen and was dull aching in type associated with nauseating feeling and loss of appetite and occasional vomitings. The fever was intermittent not associated with chills and sweating, with maximum documented temperature being 102 degree F. He presented to our centre with those complaints. He had no significant past medical history. He was not taking any medications except some iron tablets for anemia from a nearby health facility and had no known allergies. Physical examination showed a febrile pale and ill-looking young male with normal other vital signs. Per-abdominal findings were suggestive of palpable nontender spleen around 2 cm below left subcostal margin along mid clavicular line, otherwise no other significant systemic findings were noted.

Peripheral smear showed macrocytic, microcytic and hypochromic occasional fragmented RBCs, decreased WBC with relative lymphocytosis, no blast, and reduced platelets without atypical cells and Chest X-ray was normal.

Abstract

EBV infection can occur worldwide. We managed a 17 year old male who developed transient pancytopenia followed by thrombocytosis concurrent with EBV infection. To the best of our knowledge we have not encountered any cases of thrombocytosis concurrent with EBV infection before. Our case was diagnosed as a case of EBV infection with the help of serology and managed conservatively. In our case, though it is uncommon to find thrombocytosis due to EBV infection, it is possible that following pancytopenia concurrent with the EBV infection, the patient might have developed secondary or reactive thrombocytosis due to elevated endogenous levels of thrombopoietin, interleukin-6, catecholamines.

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Thus, IL-6 may be a key mediator of the increased synthesis of thrombopoietin and the consequent reactive thrombocytosis.11

Conclusion
In our case, it is possible that following pancytopenia concurrent with the EBV infection, the patient might have developed thrombocytosis but it’s difficult to say whether it’s clonal (primary) or reactive type but the scenario may favour reactive type which in turn may be speculated to be caused by elevated endogenous levels of thrombopoietin, interleukin-6, other cytokines, or catecholamines which are not measured in this case. This could well be a tug of war between the myelosuppressive effects of EBV itself and the increasing levels of endogenous mediators during recovery phase of the infection.

References: