

Transient Acquired Myelodysplasia in an Infant with P.VIVAX Infection

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ABSTRACT

BACKGROUND

Myelodysplastic bone marrow changes are reported with nutritional deficiencies, toxin exposures, aplastic anemia, inherited disorders (e.g., congenital sideroblastic anemia), other myeloid neoplasms, and few viruses including CMV, HHV 6, HIV. Plasmodium vivax (P. vivax) has not been reported as a cause of myelodysplasia previously.

CASE SUMMARY

A 4-month-old well thriving male child presented with 10-day history of high fever and feed intolerance. Past history was non contributory. Physical examination revealed partial cleft lip, marked pallor and hepatosplenomegaly; with no lymphadenopathy, skin rash or other congenital abnormality.(fig 1)



Initial investigations showed anemia (hemoglobin 5.9 g/dL), thrombocytopenia (platelets 55×10^9 /L), leucocyte count of 11.50×10^9 /L (P25%, L72%, M2%, E1%), normal liver enzymes, negative blood and urine cultures and negative peripheral smear for malarial parasite. Peripheral smear showed dimorphic picture with anisopoikilocytosis and polychromasia. Rapid antigen test showed P.vivax positivity. Fig- 2



His hemolytic workup including direct Coombs' test, reticulocyte count, hemoglobin electrophoresis (HbF 18.8%, HbA2 2.5%) were normal. Further bone marrow aspiration was done which revealed dysplastic changes in myeloid and erythroid series, with monocytoid forms including giant metamyelocytes and reduced number of megakaryocytes precursors. Fig- 3

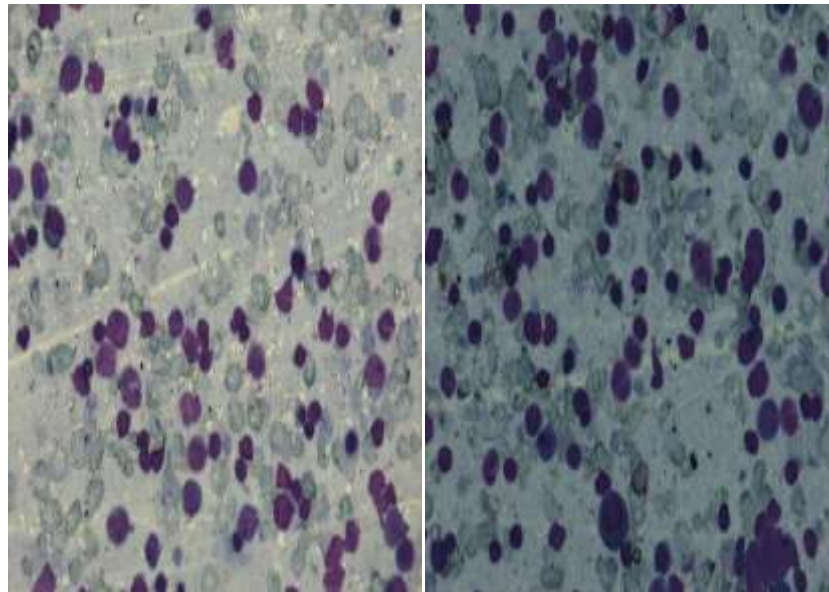


Fig- 3 bone marrow findings showing dysplastic changes

Further workup revealed normal karyotyping (46 XY) and negative HIV, hepatitis B surface antigen and TORCH serology. He had normal iron studies, serum ferritin 93 ng/dL, Serum triglycerides >500mg/dL. Child responded within three days of starting oral antimalarials. Other laboratory abnormalities improved gradually over next few weeks. He completed six months of follow up now and is clinically and neurologically well.