

**Prolonged Erythroblastopenia
in Thalassemia Intermedia**

Transient aplastic crisis (TAC) usually lasts for 7-10 days in chronic hereditary or acquired hemolytic anemias of varied etiologies in children(1). Human parvovirus B-19 (HPV) infection, which is cytotoxic to erythropoietic progenitor cells has been associated with development of transient aplastic crisis. We have observed unusually prolonged transient aplastic phase in a 8-years-old adapted girl who presented with severe anemia. The present episode of anemia was preceded by low grade fever lasting for about 3 days. There was no history of blood loss, hemolysis, exposure to chemicals or drugs. Examination revealed afebrile child having marked pallor with features of mild congestive cardiac failure. There was no icterus, cyanosis, bony tenderness or lymphadenopathy. Liver and spleen were palpable 2 and 2.5 cm, respectively. Other systemic examination was within normal limits. Investigations showed Hb 2.4 g/dl, reticulocyte count of 0.01%, with normal platelets, total and differential count. Urine for hemosiderin was negative. Peripheral blood showed hypochromic, microcytic red cells with significant aniso and poikilocytosis and target cells. Bone marrow demonstrated paucity of erythroid precursors with adequate myeloid and megakaryocytes with normoblastic maturation. Liver function tests were within normal limits. Blood, urine, throat swab and stool for bacterial and fungal cultures were negative. Coomb's test, Ham's acidified serum

test, sucrose water lysis test were negative. HBsAg and HTV tests were negative. Fetal hemoglobin (HbF) after blood transfusion was 0.8%, however, repeat HbF was 69.4% after 12 weeks of her last transfusion. The G-6-PD screening tests were negative after 16 weeks of her last blood transfusion. The child needed 17 units of packed red cell transfusions to maintain her hemoglobin (4-6 g/dl) during her period of aplastic crisis. The first sign of marrow response with elevation in reticulocyte count was seen on 25th day of admission. Subsequently, she maintained her hemoglobin between 8-9 g/dl without any transfusion for follow up period of 18 months. Reticulocyte count during this period varied between 5-8%.

Transient aplastic phase in hemolytic anemia has been described in congenital and acquired hemolytic conditions such as hereditary spherocytosis, sickle cell anemia, thalassemia, pyruvate ktnase deficiency, autoimmune hemolytic anemia and congenital dyserythropoietic anemia type II. It may follow the development of non-specific viral illnesses such as HPV B-19 infection which predominantly occurs in children between 4 to 10 years of age(2). However, aplastic phase in hemolytic anemia has been described in adults. The presence of anti-HPV of IgM class denotes a recent infection and these antibodies may be detected in some patients even upto 10-12 months after the infection(3). Virus primarily affects the erythroblasts but may involve other cell lines resulting in leucopenia and/or thrombocytopenia(4). Other viruses such as mumps, non A-non-B, HIV, EB virus and infections such as mycoplasma, salmonella, streptococcus have been implicated in the causation of aplastic crisis.

The impact of infection is more apparent in conditions of ineffective erythropoiesis(5). In healthy persons erythroblastopenic effect of infection passes off without its recognition whereas in hemolytic anemia the effect of any of these infections leading to erythroblastopenia of marrow becomes evident by development of severe anemia. It is of interest that our case initially presented with erythroblastopenia lasting for 25 days and diagnosis of thalassemia intermedia was unmasked. Supportive treatment with packed red cell is the mainstay of treatment. The possibility of the immunization against HPV in children with chronic hemolytic anemia will be of interest as children with hemolytic anemia will not develop this complication following an effective immunization.

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Extravasation Injuries Prevention, the Best Policy

I read with interest the recent article by Tomaraei and Marwaha(1). As the authors themselves agree, the most important approach should be preventive. In most hospitals in India where the rush is so high and no close monitoring by nursing staff nor sophisticated infusion pumps are available,

it is highly important that extravasation injuries be prevented to the utmost and early intervention attempted. The important guidelines will be:

(i) All hypertonic solutions including antibiotics should be diluted and given as a slow intravenous push; (ii) Before giving any hypertonic solution, like calcium or sodium bicarbonate, it must be confirmed that the cannula is in the vein and the intravenous line is patent and that there is no redness or induration at the needle tip. If there is any doubt, it is always better to