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Case Report

Pure Red Cell Aplasia in a Case of Severe Hookworm Infestation: Superadded Parvovirus B19 Infection Causing Diagnostic Confusion

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ABSTRACT
Anemia is a common manifestation of hookworm infestation. But clinically severe anemia in a child without any failure to thrive should warrant search for other etiologies. Here we report the case of a 11 years old boy with Ankylostoma duodenale infestation who presented with severe anemia with impending heart failure. A thorough search revealed concurrent Parvovirus B19 infection which caused pure red cell aplasia as proven by bone marrow study. Patient responded well to packed red blood cell transfusion, oral albendazole and corticosteroid therapy.
KEYWORDS: *Anemia; Hookworm; Parvovirus B19; Pure Red Cell Aplasia; Ankylostoma duodenale;*

INTRODUCTION

Anemia in children occurs due to red blood corpuscles (RBC) genesis or destruction or due to its loss. *Ankylostoma duodenale*, a hookworm, induce anemia in a host by causing loss of blood from the lumen of gastrointestinal tract. On the other hand, Parvovirus B19 causes aplastic crisis in patients with hemolytic anemias, prolonged red cell aplasia and chronic anemias in immunocompromised patients and transient aplasia in the normal hosts [1]. Isolated infection of both the organisms don't usually cause clinically severe anemia but in our case coinfection with both led to severe condition requiring repeated transfusions.

CASE REPORT

A 11-years-old boy, a habitant from rural West Bengal, born out of non-consanguineous marriage, was referred to our hospital with chief complaints of low grade off and on intermittent fever for last 2 months associated with severe weakness and fatigue. There was no history of contact with tuberculosis. There was nothing significant in past medical history. Neither there was any history of blood transfusion nor any family history of haematological disorders. History regarding bowel and bladder were normal.

On general examination, he was alert, conscious and co-operative. Patient appeared severely pale, temperature 100.4 degree F, heart rate was 124/min, regular rhythm, neck veins were not engorged. No lymphadenopathy was there. Height and weight were age appropriate. Systemic examination revealed an innocent ejection systolic murmur with audible third heart sound (S3). No organomegaly was noted. Chest was bilaterally clear. Neurological examination was within normal limits.

Routine investigations revealed: Hb-3.5 gm%, TLC- 21,800/cmm, Neutrophil-56%, Lymphocyte-20%, Eosinophil-22%; Platelet count-2.98 lac/cmm; MCV-91.6 fL, PCV-10.9%, Reticulocyte count- 1.9%; Iron studies revealed: serum Iron-69 microgram/dL, serum Ferritin- 235 ng/mL, serum TIBC- 312 microgram/dL, Transferrin saturation- 15%; Peripheral blood smear showed: Mild hypochromic microcytosis, anisocytosis, few

Poikilocytes, no immature WBC was seen, platelets were adequate. No malarial parasite was found. Malarial dual antigen test, Direct coomb's test, Widal test, blood and urine culture, sputum (microscopy and GeneXpert) for AFB, tuberculin test, chest X-ray, ultrasonogram of whole abdomen were normal.

Routine stool test came out to be positive for occult blood, few adult *Ankylostoma duodenale* and plenty of ova were seen. So provisionally he was diagnosed as a case of severe hookworm anemia. Antihelminthic treatment with oral Albendazole (400 mg) started promptly and he was transfused with one aliquot of packed red blood cells. After 24 hours of transfusion haemoglobin level was 5.2 gm% but again pallor was increasing clinically. Haemoglobin repeated after 4 days of transfusion showed 4.1 gm%.

So a bone marrow aspiration study was done and it showed features suggestive of Pure Red Cell Aplasia (PRCA): predominantly intermediate & early normoblasts; Myeloid/Erythroid ratio: 9/1; Predominant mature neutrophils, band forms; Eosinophilic series increased; No increase in myeloblasts; Mature & active megakaryocytes; Lymphoid series normal; No parasite/ infiltrative/neoplastic cells.

Diagnosis was modified to acquired pure red cell aplasia in a case of severe hookworm infestation. As repeat haemoglobin came as 3.6 gm% again transfusion was prescribed and before this second transfusion serological assay for IgM against Parvovirus B19 was sent which, fortunately, came out to be positive in moderately high titres despite receiving one unit of PRBC. Transfusion was given and due to unavailability of intravenous immunoglobulin G (IVIG) oral prednisolone was started at the dose of 2 mg/kg/day and gradually stopped after tapering over 4 weeks. Simultaneously, therapy with albendazole, iron and folate was continued and after 8 weeks stool became negative any adult/ova of hookworm, Hb was 10.2 gm%. Patient was continuously followed up monthly but within a span of 1 year post-discharge he didn't have any deterioration.

DISCUSSION

Hookworm infestation, a worldwide nuisance, affects approximately 600 million people all over the world; it is one of the most common parasitic infections of humans. The highest prevalence is reported in southeast Asia, sub-Saharan Africa, and the Indian subcontinent, especially in rural areas with low socioeconomic status, poor sanitary facilities, and indiscriminate defecation habits that allow larvae to develop in the soil. [2,3]

A heavy infestation may spread hookworms along the whole gastrointestinal tract, including the stomach, duodenum, jejunum, proximal ileum, and to the ascending and sigmoid colons.[4] Ankylostoma duodenale (adult male measuring 8–11 mm in length; adult females: 10–13 mm in length) consumes blood approximately at a rate of 0.26 mL/day per adult worm; this results in iron-deficiency anemia, which develops insidiously. [5] The chronic, slowly progressive blood loss activates compensatory mechanisms, which delay the clinical presentation until haemoglobin levels are very low. The diagnosis is primarily made by finding the characteristic eggs by fecal examination of patients with microcytic hypochromic anemia and eosinophilia.

Parvovirus B19 infection commonly results in erythema infectiosum (fifth disease) in childhood and may be associated with polyarthropathy in adults. [6] Parvovirus replicates exclusively in erythroid progenitor cells that have the P antigen receptor. [7] Patients with hemolytic anemias, including sickle cell anemia, thalassemia, pyruvate kinase deficiency, autoimmune hemolytic anemia, spherocytosis, pyropoikilocytosis, and elliptocytosis have all been reported to have erythroblastopenic crisis in conjunction with parvovirus infections. [8] In general, crisis caused by the virus is self-limiting and resolve in one to two weeks. [8] However, the anemia may be severe enough to result in symptoms of heart failure and require transfusion. [6,8]

PRCA can be easily diagnosed when isolated anaemia, in the presence of normal white cell and platelet counts, is associated with a marrow of normal cellularity in which there is an almost complete absence of erythroblasts but normal myeloid cells and megakaryocytes. [9] PRCA secondary to parvovirus B19 can be treated with intravenous immunoglobulin. [10] Corticosteroids (CS) were the first immunosuppressive drugs used in the treatment of PRCA and so far have been considered the treatment of first choice, especially in young adults. [9]

In our patient oral steroid therapy was successful and showed improvement as evidenced by rise in haemoglobin to almost normal range in 8 weeks. To our best knowledge, no report till date has shown such unique haematological feature in this type of coinfection.

DECLARATION

NO FUNDING was required for this case.

The authors do not have any **CONFLICT OF INTEREST**.

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