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Autoimmune Hemolytic Anemia in Visceral Leishmaniasis

Pages with reference to book, From **100** To **101**

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A case of an 8-month old male child, who presented with lgG mediated Coomb's positive hemolytic anemia and visceral leishmaniasis is presented. The hemolytic anemia resolved following the treatment of leishmaniasis. Although various other mechanisms of anemia have been described, so far the association between IgG-mediated hemolytic anemia and visceral leishmaniasis has not been reported.

Case Report

A male child, aged 18 months presented with a three month history of pyrexia and abdominal distention and a non-productive cough for the past one month; purpuric spots also appeared all over the body in the preceding one week. The physical examination revealed pallor, tachycardia, tachypnoea and bilateral basal crepitation. Abdomen was distended and liver was palpably enlarged 3 cm below the subcostal margin in the midclavicular line and spleen was palpable 7 cm below the costal margin.

Laboratory investigations revealed pancytopenia with a Hb of 4.9 g/dl, WBCs $2.4x10^9$/L and platelets 11x109/L. The reticulocytes comprised 5% of the red blood cells. A direct Coomb's test was strongly positive and mono-specific Coomb's test revealed lgG-mediated activity. Peripheral blood film showed red cell agglutination, macrocytes, m icrospherocytes, polychromasia and occasional nucleated red blood cells. Reactivity to anti-complement sera was negative, anti nuclear antibody profile and cold agglutinin titer for mycolpiasma pneumonia were also negative. The liver function tests revealed the direct bilirubin to be 4.1 mg/dl, with an indirect component of 3.0 mg/dl. The rest of the serum chemistries were within normal limits. Radiological examination of the chest revealed bronchopneumonia.

The patient was transfused with minimally incompatible packed red cells and was started on intravenous antibiotics and oral Prednisolone. A provisional diagnosis of autoimmune hemolytic anemia, probably secondary to .a lymphoproliferative disorder was made. The bone marrow aspirate revealed a cellular marrow with erythroid hyperplasia, a myeloid: erythroid ratio of 1:1 and the presence of prominent Leishman-donovan bodies in the macrophages. These were also present extracellularfy (Figure).

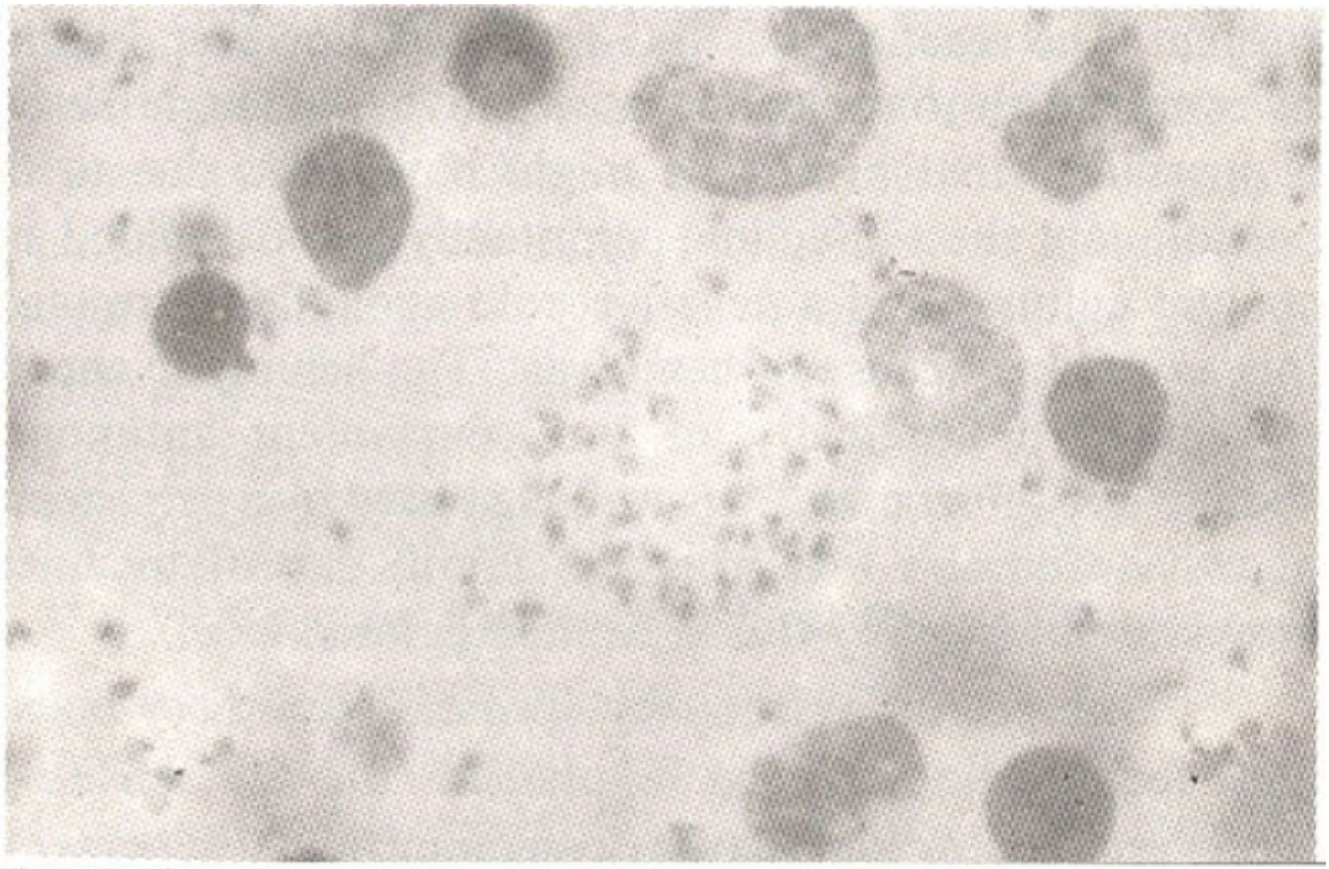

Figure. Leishman-Donovan bodies seen both within the macrophage and extracellularly (X 100).

On the third day of admission, the patient was started on parental pentavalent Antimony preparation (Inj Stibogluconate 20 mg/kg/day) and steroids were tapered off and stopped.
afebrile, Hb increased to 6.7 g/dl, WBCs to $4.5x10^9$/L and platelets increased to $70xl0^9$/L. Direct Coomb's test became negative and pneumonia resolved completely. Patient was discharged and was continued on daily Stibogluconate injections for another 21 days, along with folic acid 5 mg oraly. After the completion of treatment, Coomb's test remained negative and the full blood count revealed a Hb 9.0 g/dl, WBCs $8.8x10^9$/L, platelets $301x10^9$/L. The liver and spleen were not palpable anyore. The peripheral blood film did not reveal any abnormality.

Discussion

One of the major presenting features in untreated and visceral Leishmaniasis is anemia, the cause of which has been a focus for many researchers. A variety of hemolytic and non-hemolytic mechanisms have been discribed[1]. Subsequent to phagocytosis of Leishmania, the macrophages rupture and the parasite is carried to the reticu lo-endothel i al system, where progressive hyperplasia of macrophages and lymphocytes result in enlargement of the liver, spleen and lymph nodes[2]. The severity of pancytopenia may be related to the size of spleen. The role of spleen and hypersplenisrn has been emphasized by a report of near normal hematology in a patient who had been splenectomized prior to infection with leishmania[2]. The ^{51}Cr half life of both parasitized and normal red blood cells has been shown to be shortened[3]. Another postulated mechanism of hemolysis is the fixation of complement (C3) onto red cells. However, it has been clearly demonstrated that complement fixation leads to hemolysis in leishmaniasis[4]. Other hemolytic mechanisms suggested, include an increased sensitivity

to complement, inhibition of certain erythrocyte enzymes, and the direct production of hemolysins by Ieishmania[5].

In this case hemolysis was found to be immune-mediated as detected by a strong positivity to Coomb's reagent. The reaction was lgG-mediated, however, binding of complement to red cells was negative. Although there is a possibility that hemolysis might have been due to the concomitant chest infection, persistently negative blood and sputum cultures and a negative cold agglutinin titre made this an unlikely cause of hemolysis. The hemolytic anemia responded dramatically to the pentavalent antimonaal sodium suboluoonate. This was suggesed by a persistently negative Coomb's test from the fourth day of treatment

Our patient was: pancytopenic. Thrombocytopenia and leueopenia can be explained on the basis of sequestratrnn or mcreased consumption. such as in latter probability was explored and was excluded on the basis of PT, APT!' and D - Dimer assay. Both thromobocytompenia and leucopenia resolved over time along with the regression of spleen.

We conclude that lgG-mediated autoimmune mechanism is the most likely explanation of hemolysis in our patient. Visceral leishmaniasis should be considered as a possible cause of autoimmune hemolytic anemia.

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