Infectious Diseases & Therapy

Busuttil, J Infect Dis Ther 2015, 3:1
http://dx.doi.org/10.4172/2332-0877.1000195

Letter to the Editor Open Access

The Paradoxical Beneficial Effect on Thrombocytopenia in Refractory Immune Thrombocytopaenic Purpura by Leishmaniasis

David Paul Busuttil*

Department of Haematology, Mater Dei Hospital, Malta

***Corresponding author**: David Paul Busuttil, Department of Haematology, Mater Dei Hospital, Malta, Tel: 21313692; E-mail: david.p.busuttil@gov.mt

Rec date: Aug 05, 2014; **Acc date:** Jan 02, 2015; **Pub date:** Jan 07, 2015

Keywords: Thrombocytopenia; Immune Thrombocytopenic purpura; Leishmaniasis

Letter to the Editor

The phenomenon of platelet recovery in untreated patients with severe chronic refractory immune thrombocytopenic purpura (ITP) has never been described. A 44 year old lady presented with pyrexia. The blood count was Hb 9.8 g/l WBC 6.5 × 10^9/l Platelets 125 × 10^9/l. A bone marrow aspirate revealed macrophage hyperplasia with heavy infiltration by *Leishman Donovani*. The patient had a twenty year history of severe ITP responsive only to high doses of prednisolone. She had undergone two splenectomies and failed to respond to the sequential administration of dapsone, azathiaprine and cyclosporine. The minimum daily dose of prednisolone required to maintain the platelet count was 30 mg and she was on this maintenance dose for most of the duration of the disease. Intensive treatment with liposomal amphotericin and sodium stibogluconate failed to clear the parasites and for the rest of her life (14 months) she remained a carrier of leishmania requiring maintainance monthly doses of Leishmaniacides. During this period the platelet count remained normal even though the steroids were tapered to zero. The normalisation of the platelet count without steroids was unexpected and had never been observed before in her case. It was noted that she maintained a normal platelet count spontaneously even though she had developed transfusion dependent anaemia. Moreover, by then she had developed severe neutropenia partially corrected with granulocyte colony stimulating factor. Both cytopenic effects were a consequence of the ongoing sepsis. The patient eventually succumbed to renal failure secondary to a diabetic nephropathy. Kala-azar in normal subjects characteristically causes thrombocytopenia partly because of hypersplenism and partly because of consumption by the diffuse macrophage hyperplasia within the reticuloendothelial system. Also, in HIV infection where ITP is common, visceral leishmaniasis has been shown to worsen the thrombocytopenia. The main reason for thrombocytopenia in ITP is macrophage mediated phagocytosis of the antibody coated platelets. Two highly effective therapeutic agents are directed against this macrophage activity. Intravenous immunoglobulin saturates the macrophage receptor sites, thereby sparing the sensitised platelets by competitive inhibition [1]. Anti–D transiently diverts macrophages from destruction of platelets by focusing their activity against the sensitised erythrocytes [2]. Using the similar paradigm of these immunemodulatory agents, I postulate that the continual saturation of macrophages with the Leishmania parasites likewise had a platelet sparing effect. This observation may be of interest for pharmaceutical companies in their drug development research.

References

1. Newland AC (1989) The use and mechanisms of action of intravenous immunoglobulin. An update. Br J. Haematol 72: 301.
2. Bussel JB, Graziano JN, Kimberley RP (1991) Intravenous anti-D treatment of immune thrombocytopenic purpura: an analysis of efficacy, toxicity and mechanism of effect. Blood 77: 1884-1893.