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Babesiosis-associated immune thrombocytopenia

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Abstract: Thrombocytopenia is a common feature of babesiosis. The mechanism for thrombocytopenia in babesiosis remains elusive. We report a case of babesiosis with severe new onset immune thrombocytopenia (ITP). In addition to antibiotics treatment for babesiosis, ITP therapy was administered. ITP in the present case was most likely triggered by the babesia infection. The severity of ITP in this case was not proportional to the severity of parasitemia. The neoantigen triggering the autoimmune response in babesiosis requires further characterization.

Keywords: Babesiosis; tick-borne disease; thrombocytopenia; immune thrombocytopenia (ITP)

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Introduction

Babesiosis is a common tick borne zoonosis with a diverse spectrum of severity. Patients most often present with fevers, headaches, severe myalgias and nausea. Severe babesiosis can be a life threatening condition causing acute respiratory distress syndrome, renal failure, cytopenias and disseminated intravascular coagulation (DIC) (1,2). The parasite level and the severity of the disease do not always correlate. Persistent parasitemia has been described in both immunocompetent patients with and without treatment and more frequently in immunocompromised population (3). Common laboratory findings include hemolytic anemia, thrombocytopenia and elevated liver enzymes (1,2). However, babesia triggering new onset severe immune thrombocytopenia (ITP) has not been reported in the literature. Here we present a case of a patient with babesiosis and new onset severe ITP despite low parasitemia.

Case presentation

A septuagenarian woman with past medical history of asthma was transferred to our institution from an outside hospital for management of babesiosis and thrombocytopenia. A week before admission, she developed flu-like symptoms followed by progressive decrease in

exercise tolerance and dyspnea on exertion. Diffuse rash on both her legs prompted her to seek medical evaluation. She also reported excessive bruises and ecchymosis following blood draws. Review of systems was negative for fever, chills, chest pain, weight changes, nausea, abdominal pain or vomiting. Her last blood count obtained one year earlier was normal. She denied any significant personal or family history of bleeding/clotting disorders. In the outside hospital babesia organisms were found in the peripheral blood smear, with estimated load of 0.5%. Upon admission to our hospital the parasite load was 0.3%. She was found to have platelet count of 1,000/mcL, LDH 243 IU/L, total bilirubin 1.1 mg/dL and haptoglobin 92 mg/dL. On physical examination splenomegaly, diffuse petechial rash and ecchymoses were present. Peripheral blood smear revealed no evidence of thrombotic microangiopathy. Platelets auto-antibody was reported as being strongly positive. Ehrlichia and Lyme titers were negative. Cytomegalovirus and Epstein-Barr virus antibodies were detected in IgG class, but not IgM. She also had negative dengue and human immunodeficiency virus serology. ADAMTS 13 activity was within normal limits. Abdominal ultrasonography revealed hepatosplenomegaly. She was initially given intravenous clindamycin and quinine, which were later changed to oral atovaquone and azithromycin to complete a course of antibiotic therapy for babesiosis.

The patient received intravenous immunoglobulin (IVIG) for babesiosis-associated ITP. Serial blood smears showed clearance of parasitemia. *Babesia microti* DNA PCR was not detected prior to discharge. The patient was discharged with platelet count of 104,000/mcL with sustained trend upwards. Following discharge the platelet count further increased to 169,000/mcL within three days and to 309,000/mcL within 10 days.

Discussion

Babesia species are intra-erythrocytic parasites. Effective cellular response and spleen play a major role in host defense against babesiosis. Splenectomy and advancing age related decline in cellular immunity are major risk factors for severe babesiosis (2,4). Thrombocytopenia is a common hematologic finding in tick borne diseases (5). Splenomegaly with peripheral destruction of platelets, direct consumption and injury to the platelets and marrow suppression are a few mechanisms of thrombocytopenia described in the literature. Dumler *et al.* studied bone marrow findings of a few patients with human ehrlichiosis and reported findings ranging from megakaryocytosis and myeloid hyperplasia to pancellular hypoplasia (6). Non-immune mediated platelet consumption and destruction leading to diffuse intravascular coagulation and thrombotic thrombocytopenia purpura have been described in tick borne illnesses like ehrlichiosis, babesiosis and rocky mountain spotted fever. Autoimmune antibodies were reported in babesiosis and ehrlichiosis. Auerbach *et al.* described hemophagocytosis in a case of severe babesiosis which cleared after appropriate antibiotic therapy (7). Autoimmune phenomena have also been found to be contributory factors for thrombocytopenia in tick borne illnesses. Wong *et al.* used flow cytometry to demonstrate presence of anti-platelet antibodies in patients with human granulomatous ehrlichiosis (8,9). However, in the review of the literature to date, we have not found a report of babesiosis causing new onset ITP in an otherwise healthy individual. Animal models have previously demonstrated immune phenomena against platelets in the setting of *Babesia* infection. Lewis *et al.* demonstrated the presence of anti-platelet antibodies in the serum of dogs with babesiosis and ehrlichiosis (10,11). Orinda *et al.* reported auto ITP caused by autoantibodies to phosphatidyl-serine in cattle infected by *Babesia bovis* (12). ITP was also described in a patient with history of Hodgkin lymphoma with splenectomy who presented with malaria and was diagnosed with *Babesia divergens* infection (13,14).

It remains unclear whether in this case babesia infection triggered the ITP. In the reported by us case, the patient was a previously healthy individual. We conclude that ITP was most likely triggered by the babesia infection. The severity of ITP in this case did not correlate with parasitemia which was only 0.3%. Multiple mechanisms of ITP have been previously described in the literature (15-18). The pathogenetic mechanisms of ITP associated with babesiosis remain elusive and require further characterization.

Conclusions

Thrombocytopenia is a common feature of babesiosis. ITP could be a probable cause and should be considered in patients with new onset severe thrombocytopenia. In addition to antibiotics for treatment of babesiosis, ITP treatment should be initiated in severe cases. The neoantigen triggering the autoimmune response in babesiosis requires further characterization.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Informed Consent: Written informed consent was obtained from the patient for publication of this manuscript and any accompanying images.

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