Severe Thrombocytopenia In Acute Brucellosis: A Case Report

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Citation


Abstract

Thrombocytopenic purpura associated with brucellosis has been rarely reported in the world literature. We describe a case of acute brucellosis in an 33-year old male, who presented with fever, purpuric skin lesions and epistaxis. Initial laboratory investigations revealed isolated thrombocytopenia with platelet count of 5,000/mm$^3$ and positive serology for Brucella. Thrombocytopenia resolved promptly with proper antibiotics on 8th day of treatment. Brucellosis should be included in the differential diagnosis of thrombocytopenic purpura in Brucella-endemic areas.

INTRODUCTION

Brucellosis is a zoonotic disease and is a common disorder in Turkey (1). Brucellosis is a multisystem disease with a broad spectrum of clinical manifestations. Haematological abnormalities including anaemia, leucopenia, thrombocytopenia and pancytopenia can be encountered during the course of the disease (2,3). Isolated thrombocytopenia was seen in 8 % of cases in a study report from Turkey (2). In the report, a case of brucellosis with severe thrombocytopenia was present.

CASE REPORT

A 33-year- old male patient was admitted to haematology service with fever and epistaxis. He had a history of weakness, night sweats and fever for 20 days. Vital signs were normal except for a temperature of 38° C. His physical examination was otherwise normal except for petechial and ecchymotic lesions in the lower extremities. Laboratory tests showed the following values: hemoglobin was 15.7g/dl, erythrocyte 5.51x10$^6$ / L, leukocyte 6770/mm$^3$ (neutrophile 33.9%, lymphocyte 54.8%, monocyte 9.15%), thrombocyte 0.5x10$^3$ / L sedimentation rate 20 mm/h, CRP 28 mg/dL. Liver function tests showed the following values: aspartate aminotransferase, 46 U/L (15-40); alanine amino transferase, 66 U/L (10-40); lactate dehydrogenase, 327 U/L (125-243); and total bilirubin, 0.53 mg/dL, (0.24 mg/dL direct). Haemostatic tests were in normal range. Serological tests for EBV, CMV, hepatitis B, rubella, toxoplasma Ig M were all negative. Direct antiglobulin test was negative. Serum ferritin, vitamin B$_{12}$ and folic acid levels were in normal range. Abdominal ultrasonography was normal. A blood smear examination showed predominant neutrophils and rarely large platelets. A bone marrow examination was normal except for an increased number of megakaryocytes. Transfusion of thrombocyte (3 units) was performed. While investigating the etiology of fever, positive agglutination test for brucellosis was documented at a titer 1/80 on day 2 hospitalization. Brucella was isolated from blood cultures taken on the day of fever which could not be serotyped. Appropriate treatment for brucellosis with rifampicin (600mg/d) and doxycycline (200 mg/d) was initiated; this brucellosis treatment was maintained for 6 weeks. In the first week of treatment his platelet count was 76 x 10$^9$/L. He was discharged from the hospital 13 days after admission good health on rifampicin and doxycycline therapy with a platelet count of 131x 10$^9$/L.

DISCUSSION

Brucellosis is an important medical problem in developing countries. In course of human brucellosis, seen commonly seen hematological abnormalities are anaemia, leucopenia, thrombocytopenia and pancytopenia. Thrombocytopenia is reported in 1-26 % of patients with brucellosis (1,2). In rare cases, thrombocytopenia can be severe and may cause bleeding into skin and from mucosal sites. (1,2). In our case the count of thrombocyte was near zero. Epistaxis and ecchymoses were occurred in patient. The mechanism thrombocytopenia in brucellosis is not
clearly understood. Among the proposed mechanisms are hypersplenism, intravascular coagulation, bone marrow suppression due to septicemia, hemophagocytosis, granulomas and peripheral immune destruction of platelets (4,5,6,7). The mechanism of thrombocytopenia in our patients is possibly related to brucellosis induced peripheral destruction of platelets since large platelets in the peripheral smear and megakaryocytic hyperplasia in the bone marrow was detected.

It is well documented that appropriate antimicrobial therapy improves the clinical and hematological status of patients with brucellosis. Platelet recovery usually occurs within 2-3 weeks of initiation of appropriate antimicrobial therapy (3). Our case presented with a fever, was immediately diagnosed as brucellosis and responded treatment. Thrombocytopenia resolved promptly with proper antibiotics on 8th day of treatment.

In conclusion, differential diagnosis of thrombocytopenia should include brucellosis, especially in regions where brucellosis is endemic.

References
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