The patient was febrile (101°F, 38.3°C). There was also shifting dullness on abdominal examination suggestive of free fluid in the abdomen. There was no organomegaly or lymphadenopathy. The rest of the physical examination was normal.

Relevant investigations included hemoglobin of 12.2 g/dL, total leukocyte count of 4400/mm³ (48% neutrophils, 50% lymphocytes, and 2% eosinophils), platelet count of 56,000/mm³, and erythrocyte sedimentation rate of 90 mm during the first hour using the Westergren method. Peripheral blood smear was remarkable for paucity of platelets with normochromic and normocytic red blood cells (Figure 1). Mean corpuscular volume was 88 fL, and the corrected reticulocyte index was 1.7%. Bone marrow aspiration showed erythroid hyperplasia and megakaryocytosis cells without any granuloma and necrosis (Figure 2). Mantoux test was positive. Ultrasonography of the abdomen revealed moderate ascites. Ascitic fluid analysis revealed: total cells, 1000 cells/mL (85% lymphocytes); protein concentration, 4.1 gm%; and adenosine deaminase, 77 U/L. A qualitative multiplex polymerase chain reaction for tuberculosis (TB) using IS6110 and protein B was positive. The patient's liver and renal function tests, prothrombin time, activated partial thromboplastin time, lipid profile, iron studies including serum ferritin level, direct Coombs test, human immunodeficiency virus, antihepatitis C virus, and hepatitis B surface antigen reports were all negative. Chest x-ray was normal. Contrast-enhanced computed tomography scan of the abdomen and...
Abdominal Koch disease was diagnosed in our patient on the basis of a high adenosine deaminase value in the ascitic fluid, a positive multiplex polymerase chain reaction for TB, surrogatory contrast-enhanced computed tomography findings, and a treatment response to ATT. The patient reported no history of drug use in the recent past. Secondary causes of immune thrombocytopenia were also ruled out, and his bone marrow aspiration results were consistent with peripheral destruction of platelets.

CONCLUSION

For immune thrombocytopenia secondary to TB, a combination of steroid and ATT is generally preferred for rapid increase in platelet count. The role of intravenous immunoglobulin has also been described in the literature, but there are presently no definite guidelines for treatment of immune thrombocytopenia secondary to TB.

Because our patient was not symptomatic for thrombocytopenia, he was treated with ATT only. On follow-up after completing the full course of ATT, his platelet count and hemoglobin were normal, and he reported feeling fine.

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

References