Case Report

Unusual Presentation of Amebic Liver Abscess with Thrombocytopenia and Splenomegaly

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Amebiasis is an important worldwide health problem. Liver abscess is one of the most common invasive complications of intestinal amebiasis. Abdominal pain and fever are the most common symptoms associated with acute amebic liver abscess (ALA). Although a few parasitic diseases, such as malaria and toxoplasmosis, are associated with thrombocytopenia, thrombocytopenia in the course of amebiasis or ALA has not previously been reported.

CASE REPORT

An 18-year-old male living in the Ankara region was admitted to Gaziantep University Hospital for fever. He was complaining of decreased appetite, nausea, vague right abdominal discomfort, and pleuritic chest pain. He passed watery stools 2 weeks prior to the onset of his symptoms. His axillary temperature was 38°C, and he had tachycardia with a heart rate of 120 beats per minute on physical examination. He also had diminished breath sounds in the right lower lung field and a tender hepatomegaly extending 8 cm below the right costal margin and 7 cm of splenomegaly below the left costal margin, without rebound tenderness on abdominal palpation. The rest of his examination was normal.

Chest radiograph showed an elevated right hemidiaphragm with a localized shadow at the lower base. Laboratory data showed mild leukocytosis of 11.8 × 10⁹/L, hemoglobin of 15.5 g/dL, and platelet count was 75 × 10⁹/L. Blood chemistry showed serum glutamate-oxaloacetic transaminase (SGOT) 107 IU/L, serum glutamate pyruvate transaminase (SGPT) 142 IU/L, alkaline phosphatase 113 IU/L, gamma-glutamyltransferase (GGT) 100 IU/L, lactate dehydrogenase (LDH) 1193 IU/L, albumin 2.5 g/L; other parameters, including prothrombin time (PT), activated partial thromboplastin time (aPTT), d-dimer and fibrin degradation products (FDP), were within normal limits. Ultrasound of his abdomen revealed a hypoechoic solitary cystic lesion in the right lobe of the liver measuring 12 × 10 cm, with normal portal dimensions and flow rate. Abdominal computed tomography (CT) revealed an enlarged liver and spleen and an abscess formation in the right lobe of the liver (Figure 1). The stool specimen showed a few trophozoites of Entamoeba histolytica. Serum indirect hemagglutination test for E. histolytica was positive in 1/256 titer (Cellagnost Amebiasis, Behring). All other tests, including viral markers for Epstein-Barr virus, cytomegalovirus, and hepatitis A, B, and C, hydatid cyst agglutinations, bone marrow aspiration, and alpha-fetoprotein levels, were within normal limits. Peripheral blood and bone marrow smears also were reviewed without any evidence of malaria. Metronidazole treatment 750 mg every 8 hours was commenced. The clinical condition improved, and the fever subsided within a few days. To evacuate the liver abscess, an ultrasound-guided percutaneous drainage catheter was inserted. A brownish discharge was obtained and a high titer of E. histolytica agglutination was detected in this fluid (1/1024), with a negative bacteriologic culture. After a week of antibiotic and drainage treatment, the patient appeared well without any symptoms, the chest radiograph was normal and CT of the abscess revealed a marked decrease in the diameter of the cyst (Figure 2).

Figure 1. CT scan of the abdomen showing a large abscess in the right lobe of the liver.
Figure 2. CT scan of the abdomen a week after percutaneous drainage and antibiotic treatment.

Splenomegaly and thrombocytopenia improved rapidly and were completely normal after the first week of the treatment was complete.

DISCUSSION

It has been estimated that 10% of the world’s population is infected with *E. histolytica*, with approximately 50 million of these individuals developing invasive amebiasis. Amoebic liver abscess is the most common extraintestinal form of amebiasis, most commonly presenting with fever and right abdominal pain in acute cases.

The presentation described in this young male (a history of fever, right upper quadrant pain, pleuritic chest pain, leukocytosis, abnormal serum transaminases and alkaline phosphatase, and elevated right hemidiaphragm) is typical for ALA, especially after the demonstration of amebic trophozoites in the stool specimen, and the presence of hypoechoic cysts, revealed by ultrasound and CT. The presence of splenomegaly at presentation and the rapid improvement of thrombocytopenia simultaneously with the disappearance of splenomegaly following treatment with metronidazole suggest increased sequestration of platelets in the enlarged spleen as a possible pathogenic mechanism. Increased portal tension could have triggered thrombocytopenia. However, neither clinical examination (ascites, varices) nor ultrasonographic examination (increased portal diameters or blood flow) suggested a diagnosis of portal hypertension.

Recently, endothelial cell activation and its possible pathogenic role in abscess formation was demonstrated by showing the expression of intracellular adhesion molecules 1 and 2 and of von Willebrand factor in hepatic endothelial cells of patients with ALA. The presence of inflammatory cells and the importance of this inflammatory process in the pathophysiology of tissue damage in ALA also have been demonstrated. Endothelial cell injury in the hepatic tissue, either directly or via the inflammatory process, leads to the initiation of coagulation with the expression of tissue factor, which in turn causes consumption of both the coagulation factors and the thrombocytes. Increased platelet consumption could be the cause of thrombocytopenia in the presented patient as well, with his large ALA.

Whereas splenomegaly is a frequent feature in certain parasitic infections, such as malaria, schistosomiasis, leishmaniasis, and trypanosomiasis, it is unusual in amebiasis. Sequestration of parasite-infected erythrocytes in the spleen is the cause of splenomegaly in malaria. Since, amebae do not infect erythrocytes, possibly the function of the spleen as a filter for microorganisms was the cause of splenomegaly in this patient. Though large masses in the liver, as in the patient described here, may cause increased portal pressure and splenomegaly, the patient did not present with signs of portal hypertension.

CONCLUSION

In the patient described in this case report, besides the classic findings of amebic abscess, splenomegaly and...
thrombocytopenia also were noted. As in the majority of cases with infection-associated thrombocytopenia, the cause of thrombocytopenia in this case remains to be elucidated. Either hypersplenism-induced or *E. histolytica*-induced increased platelet destruction via immune or nonimmune mechanisms may be the cause of thrombocytopenia. The filter function of the spleen is the probable cause of splenomegaly.

REFERENCES


