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Letter to Editor

Internal Medicine Section

Hepatosplenomegaly and Pernicious Anaemia

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Sir,

Pernicious anaemia (PA) is the end stage of atrophic gastritis which results in the loss of parietal cells in the fundus and body of the stomach. Loss of parietal cells is associated with the failure of intrinsic factor production and results in vitamin B12 deficiency and megaloblastic anaemia. Only two cases of splenomegaly coexisting with PA have been reported, but both splenomegaly and hepatomegaly has not been reported yet [1,2]. In this report, we aimed to describe a patient with hepatosplenomegaly due to PA. To the best of our knowledge this is the first documented case of hepatosplenomegaly due to PA and totally resolved by vitamin B12 therapy.

A 20-year-old male patient was admitted to our hospital with the complaints of fatigue, weakness, anorexia and palpitation. His complaints had been continued for the past one month. He had no history of another disease or medication. Pallor, subikterus, 3/6 systolic murmur in all valves, palpable splenomegaly at 4 cm below the left costal margin and palpable hepatomegaly 2 cm below the right costal margin were detected on the physical examination. Laboratory examinations revealed the following: LDH: 8440 U/L, AST: 91 U/L, ALT: 45 U/L, total bilirubin: 2.8 mg/dL, indirect bilirubin mg/dL: 2.14, Hgb: 3.9 g/dL, Hct: 12.3%, Wbc: 1400 /uL, Plt: 22000 /uL, MCV: 96 fL, corrected retikulocyte count: 0.25%, direct coombs (-), indirect coombs (-).Hepatitis markers were normal. Hepatosplenomegaly was confirmed using abdominal ultrasonography; the longitudinal spleen size was 182 mm and the craniocaudal liver size was 160 mm, and were found to be increased. The peripheral blood smear contained oval macrocytes, hypersegmented neutrophils, trombocytopenia, leucopenia and low reticulocyte count. Leukemia blasts or atypical cells were not seen on marrow aspiration and biopsy, thus hemolytic anaemia and leukemia were excluded from the differential diagnosis. Bone marrow biopsy was compatible with megaloblastic anaemia. To find the cause of megaloblastic anaemia, the level of vitamin B12 was found 30 pg/mL (197-886 pg/mL), and folic acid level was found in normal ranges. Atrophic gastritis was revealed by gastroscopy and verified in pathological examination. Anti parietal cell antibody was detected positive in blood test. There was no other endocrinopathy accompanying to B12 deficiency; TSH, cortisol, antiadrenal antibody and antithyroid peroxidase levels were found to be normal. Patient was diagnosed as pernicious anaemia due to atrophic gastritis and was given parenteral vitamin B12 treatment. Reticulocyte crisis was seen on the 5th day and leukocyte, hemoglobin, platelet levels were increased on the 3rd week of the treatment without transfusion. In the second month of the treatment there was no hepatosplenomegaly at ultrasonographic scan. The patient has no complaints during the one year follow-up while being monitored from the outpatient clinic.

Even though severely anaemic patient with PA may have splenomegaly, hepatosplenomegaly co-existing with pernicious anaemia is not considered to be a characteristic feature of PA, [1,2]. PA should be kept in mind in the differential diagnosis of hepatosplenomegaly like in our patient. According to the literature the relationship between PA and splenomegaly has been well established, but hepatomegaly of this association has not been mentioned. In our opinion, it might be resulted from extramedullary hematopoiesis, because hepatomegaly was regressed after the treatment of severe anaemia by vitamin B12. Plasma B12 levels, gastroscopy, peripheral blood smear, bone marrow biopsy should be considered, especially in pancytopenic patients with hepatosplenomegaly.

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