tioned with Bu-Cy; we do not know whether more gut-toxic conditioning regimens (e.g. TBI) are equally safe in this group of patients.

Marco Picardi, Carmine Selleri, Gennaro De Rosa, Catello Califano, Andrea Camera, Bruno Rotoli

Division of Hematology, Federico II University Medical School, Naples, Italy

Key words

Acute myeloid leukemia, life-threatening abdominal infections, bone marrow transplantation

Correspondence

Prof. Bruno Rotoli, Divisione di Ematologia, Nuovo Policlinico, via S. Pansini 5, 80131, Naples, Italy. Phone: international +39-081-7462068 – Fax: international +39-081-7462165.

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Splenic inflammatory pseudotumor mimicking primary splenic malignancy

Sir,

We report the case of a patient with splenic inflammatory pseudotumor (IPT). Recognition of this rare entity is important because the clinical manifestations and radiographic features may be indistinguishable from a malignant lymphoproliferative disorder.

A 52-year old woman was admitted to hospital for evaluation of a 6-week history of fever, chills and night sweats. Physical examination was remarkable only for palpable non-tender splenomegaly. The pertinent laboratory tests were: ESR 100 mm/hour, microcytic hypochromic anemia (hemoglobin 8.5-9.5 g/dL) and persistent leukocytosis (25,500/mm^3). Various serologic tests were all negative. Abdominal CT demonstrated splenomegaly with a non-homogenous focal lesion in the upper pole (7.5×6.6 cm) with septa and coarse calcifications. ^{67}Gallium scan demonstrated a pathologic uptake in this region. CT guided fine needle aspirate (FNA) yielded 3 mL of sterile fluid. Cytologic examination of the aspirated fluid showed abundant lymphocytes, histiocytes and granulocytes. The patient was treated with intravenous antibiotics but failed to respond to therapy and a splenectomy was performed. On gross pathologic examination the spleen weighed 430 g. Cross-section of the spleen revealed a firm single yellow-gray, circumscribed mass in the upper pole, containing focal areas of calcifications and an area of necrosis. Histologic examination revealed spindle-shaped cells which were stained with smooth muscle actin and vimentin surrounded by large numbers of lymphocytes. After 3 years of follow-up, the patient is asymptomatic, with a normal ESR and leukocyte count.

IPT is a lesion of disputed etiology characterized by proliferation of myofibroblasts accompanied by a prominent inflammatory component. Although the lung is the best known and most common site, IPT occurs in diverse extra pulmonary locations including the spleen.[1,2] IPT of the spleen is extremely rare and occurs in adults, with a propensity in middle-aged individuals.[3] Microscopically, the lesions are composed of a variable mixture of inflammatory cells within spindle cell proliferation. Coagulative necrosis is located centrally in most patients, neutrophilic leukocytes dominating in the presence of necrosis. Our patient presented with a 6-week history of fever and chills, night sweats, splenomegaly, high ESR, anemia, and persistent leukocytosis. These are the most frequent symptoms and signs observed in patients with splenic abscess.[4,5] CT guided FNA performed in the patient yielded 3 mL of sterile fluid, but the cytologic findings of abundant lymphocytes, histiocytes and granulocytes were consistent with abscess. The patient was immunocompetent without evidence of a predisposing condition or bacterial infection. Despite this fact, we erroneously diagnosed and treated the patient as having a splenic abscess. Combined therapy with antibiotics and percutaneous drainage of splenic abscesses have been demonstrated to be an effective and safe procedure.[6,7] After 3 weeks a splenectomy was performed and histopathologic examination showed IPT. The

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diagnosis of IPT can be established only by histopathologic examination of the splenic lesion, since neither clinical and imaging findings, nor cytologic findings are pathognomonic.

Clinically and radiologically, IPT may mimic a malignant process such as malignant splenic lymphoma. The origin of hematopoietic inflammatory myofibroblastic proliferation is unknown. Recently, a study by Arber *et al.*[8] suggested that EBV plays a role in at least a subset of IPT, since EBV RNA was detected in 41.2% of cases.

The prognosis of IPT is excellent, since there is no recurrence or subsequent development of malignant lymphoma. IPT of the spleen should be included in the differential diagnosis of prolonged fever with clinical, radiologic and cytologic findings suggesting splenic abscess or malignancy.

Yeouda Edoute, Ariel Roguin,* Zahava Gallimidi,° Ofer Ben-Izhak,# Pradeep Nagachandran,* Haim Ben-Ami**

**Department of Internal Medicine C; °Diagnostic Radiology and #Pathology, Rambam Medical Center and The Bruce Rappaport Faculty of Medicine, Technion - Israel Institute of Technology, Haifa, Israel*

Key words

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Correspondence

Haim Ben-Ami M.D., Department of Internal Medicine C, Rambam Medical Center, P.O.B. 9602, Haifa-31096, Israel. Phone: international +972-4-852 5473 – Fax: international +972-4-8542260 – E-mail: mdhaim@tx.technion.ac.il

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Concomitant chronic lymphocytic leukemia and acute myeloid leukemia diagnosed by two color flow cytometric analysis

Sir,

Chronic lymphocytic leukemia (CLL) is the most common form of leukemia in the Western hemisphere, accounting for about 25-30% of all leukemias. CLL increases in incidence exponentially with age; by age 80 the incidence rate is 20 cases per 100,000 persons per year. Acute myeloid leukemia (AML) is the second most common leukemia type in adults also with an increasing incidence with age.[3] An increased incidence of leukemia has been reported in patients receiving alkylating agents for hematologic and non-hematologic malignancies including CLL, but the concomitant presentation of AML and CLL is extremely rare, fifteen cases having been reported so far.[1,2,4-6] We have diagnosed a patient who presented simultaneously with these two distinct forms of leukemia by using flow cytometry.

A 82-year-old male was admitted to the hospital with weakness, dizziness and headache. The blood cell count was: WBC 37×10^9/L, hemoglobin 8 g/dL, hematocrit 24.8%, MCV 108 fL, platelets 26×10^9/L. Pallor and petechial skin lesions were the only findings on his physical examination, without hepatomegaly and splenomegaly. Two main distinct populations of leukocytes were seen in peripheral blood smear. While 65% of them were blast cells with round nuclei, one or two nucleoli, few azurophilic granules and narrow rim of cytoplasm, 35% of them were relatively mature appearing lymphocytes. Peroxidase activity was found to be positive by cytochemical peroxidase staining of the blast cells. We diagnosed the AML-M1 with light microscopy and cytochemical peroxidase staining. We performed two-color flow cytometric analysis that revealed two distinct cell populations with immunophenotyping patterns consistent with CLL ($CD5^+/CD19^+$) and AML ($CD33^+$) (Figure 1). The diagnosis of CLL was supported by the presence of lymphocytosis (12.9×10^9/L) and mature B-cell markers with CD5 and CD19 dual positivity (28%) in the population of peripheral blood mononuclear cells. AML was documented by the presence of circulating blast cells with cytochemical peroxidase positivity and 51% of the cells expressing CD33 as a myeloid marker in the peripheral blood. CD33 was the only antigen expressed in more than 50% of the cells. We performed CD33 and HLA-DR dual staining and HLA-DR expression was found to be positive in 28% of the cells without expression of CD33. This result confirmed that most of the cells detected with HLA-DR expression were B-CLL lymphocytes which had also expressed CD19, CD20 and CD22 antigens with similar percentages (28%, 30% and 26% respectively).

We could not explore B cell clonality for CLL diag-

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