

lar pattern (Figure 2). The final diagnosis was pulmonary relapse of lymphoma, since no nodal involvement was assessed.

After bone marrow transplantation, pulmonary complications occur in 40% to 60% of patients.^{1,2} Most of these have nonspecific radiologic features, requiring additional diagnostic procedures. Among those occurring in the late post-transplant period, idiopathic interstitial pneumonia, cryptogenic organizing pneumonia, restrictive and obstructive diseases as well as infectious complications usually have vague radiologic features in common which do not permit a diagnosis. Although CT may be able to provide more specific diagnoses with greater confidence, these complications can not be easily characterized by x-ray imaging.³ Nodular low-density, ill-defined opacities have been described in lung involvement of lymphomatous relapse, but several other patterns have been found: alveolar infiltrates, interstitial infiltrates and combinations of these; thus, no specific pattern can be considered as diagnostic.⁴ On radiological grounds, diffuse alveolar hemorrhage (DAH) and lymphomatous relapse are indistinguishable. Clinical signs and symptoms are nonspecific, but DAH is usually found in the immediate post-transplant period, with a rapidly fatal clinical course,⁵ while relapse usually progresses more slowly, depending on the growth rate of the tumour. On the other hand, BAL from patients with lymphoma may yield bloody returns with hemosiderin-laden macrophages, which has been considered the hallmark of DAH. Thus, radiologic evolution with growing nodular lesions was an important clue leading to the diagnosis, although in order to make the diagnosis an open lung biopsy was required. The possibility of relapse must be borne in mind when approaching a differential diagnosis of pulmonary complications after bone marrow transplantation.

Key words

Non-Hodgkin's lymphoma, pulmonary relapse

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Pathologic rupture of the spleen as the initial manifestation in acute lymphoblastic leukemia

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Pathologic splenic rupture is a rare and life-threatening complication of acute leukemia. It is even more uncommon as the initial manifestation, and only a few cases has been reported in the literature. Early recognition of this complication is vital because the prognosis is fatal without immediate treatment by splenectomy. We report the case of a spontaneous spleen rupture irreversibly complicating the onset of acute lymphoblastic leukemia in a 19-year-old man, in spite of splenectomy. In our case abdominal ultrasound was a good, non-invasive diagnostic test. Therefore, we believe that the course of the underlying disease and the physical condition of the patient dramatically influenced the disease evolution.

Spontaneous rupture of the spleen has been reported in many diseases associated with splenomegaly, e.g. infectious diseases, inflammatory diseases, and hematological malignancies.¹⁻⁴ Non traumatic rupture of the spleen is a rare and life-threatening complication of acute leukemia.¹⁻¹⁰ However, the splenic rupture as the initial symptom of acute leukemia is extremely unusual and only a few cases are reported in the literature.⁵⁻¹⁰

We report the fatal course of a patient with acute lymphoblastic leukemia (ALL) in which pathologic rupture of spleen was the initial manifestation of the disease.

A 19-year-old man was admitted to hospital with a two week history of weakness, nausea, vomiting and epigastric pain. There was no history of fever or bleeding diathesis. On admission, he had a petechial rash and severe pain in the left upper quadrant of the abdomen radiating up to the left scapula. Physical examination showed an acutely ill patient with a petechial rash on his legs, thorax and abdomen, without lymphadenopathy and with painful abdominal distension. He was pale, tachycardic and had a blood pressure of 60/40 mm Hg. The peripheral blood count showed hemoglobin 71 g/L, leukocyte count $640 \times 10^9/L$ with 100% lymphoblastic cells and platelet count $68 \times 10^9/L$, fibrinogen 103 mg/dL, prothrombin time 25%, partial thromboplastin time 50". Additional results of laboratory tests were AST 692

U/L, ALT 163 U/L, serum amylase 1,402 U/L, uric acid 15 mg/dL, LDH 9,030 U/L, blood urea and creatinine concentrations within the normal limits. An abdominal ultrasound revealed free peritoneal fluid and an enlarged spleen. An emergency laparotomy and splenectomy were performed and the patient was given supportive therapy with blood, platelets, fresh frozen plasma and fibrinogen. Operative findings were two and a half liters of intraperitoneal blood, with marked splenomegaly (size 17×16×8 cm, weight 841 g). Four lacerations were found with an active bleeding. Microscopic examination of the spleen revealed diffuse infiltration with leukemic cells and multiple small hemorrhagic foci in the parenchyma with a subcapsular hematoma.

On the basis of the morphologic characteristics of the peripheral blood and a bone marrow aspirate, cytochemical staining and immunophenotyping of the blast cells, the diagnosis of T-cell ALL was established. The immunophenotype revealed that the blasts were positive for CD1, CD7, CD2, CD5, CD8, CD34, CD38 and TdT, and negative for CD3, CD4, CD19, CD20, CD10, DR, CD14, CD13 and CD33. Immediately after the splenectomy a cytoreductor treatment was initiated with prednisone, vincristine and daunorubicin with intensive prophylaxis of lysis tumour syndrome.

The patient continued to be hemodynamically unstable, in renal failure and have hemorrhagic episodes with disseminated intravascular coagulation and hyperfibrinolysis refractory to supportive therapy. He died 48 hours after arriving at hospital. Permission to carry out a *post-mortem* examination was denied.

The diagnosis of splenic rupture must be considered in all patients with hematologic malignancies and a new abdominal pain, acute or subacute, hypotension and sudden anemia, even more so if there is not previous history of trauma. Diagnosis is based on clinical signs (abdominal pain, splenomegaly, hypotension, tachycardia, etc.) and confirmatory diagnostic tests. Although some authors have reported paracentesis to be the most effective diagnostic procedure,^{4,9} we have found that abdominal ultrasound can be a good, non-invasive technique without risk to patients who are hemodynamically unstable. In our case, the abdominal ultrasound was diagnostic and the splenectomy was performed immediately.

The prognosis in splenic rupture is poor; in the non-operative cases reviewed the mortality was 100%. The survival of patients following splenectomy is probably well correlated with the course of the underlying disease. Aggressive management with early surgical intervention and appropriate hemoderivative support is important.

Key words

Spleen, pathological rupture, ALL, initial manifestation

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Interferon- α 2b is not effective in the treatment of refractory immune thrombocytopenic purpura

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About 25-30% of patients with immune thrombocytopenic purpura (ITP) are refractory to corticosteroids, splenectomy and other treatments. It has been suggested that interferon- α 2b (IFN- α 2b) may be useful in the treatment of chronic refractory ITP patients. We treated 9 chronic refractory ITP patients with IFN- α 2b: the results were poor.

Immune thrombocytopenic purpura (ITP) is an autoimmune disease mediated by antiplatelet antibodies. Corticosteroids and splenectomy are effective treatment in the majority of patients. However, 25-30% of patients are refractory to these treatments, thus, morbidity increases, and the mortality rate rises to about 16%.¹ It has been suggested that inter-