Previous occurrence of life-threatening abdominal infection is not a contraindication to bone marrow transplantation

Sir,

Protracted severe infections during the post-induction aplastic phase in leukemic patients may cause delay and/or modification of the planned treatment strategy;[1] in particular, patients may be considered no longer eligible for bone marrow transplantation (BMT) owing to a high risk of suffering infection reactivation.[2,3]

We report on seven patients admitted to our Unit because of acute myeloid leukemia, who received additional intensive chemotherapy followed by allogeneic or autologous BMT, in spite of a recent episode of life-threatening clinically documented abdominal infection (CDAI) (Table 1). These infections appeared during the aplastic phase after induction treatment given according to the EORTC-GIMEMA AML 10 Protocol. All patients had high fever, severe abdominal symptoms (diffuse pain and tenderness, vomiting, diarrhea and/or melena); they had abnormal ultrasound (US) findings, characterized by terminal ileal loop overdistension and wall thickening (> 6 mm in patients #1 and 2; 5 mm in patient #3), by an intrasplenic spherical hypoechoic lesion (3.5 cm) in patient #4 and by dilated fluid-filled ileal loops in patients #5, 6 and 7. All patients received conservative medical management with bowel rest (naso-gastric suction and total parenteral nutrition), antimicrobial coverage (including anti-anaerobic and antimycotic drugs) and G-CSF. They all recovered from the infection and received the subsequent consolidation treatment (containing either idarubicin or mitoxantrone or daunorubicin in combination with cytarabine) with a minor delay. Then, according to the protocol, all patients were transplanted, using the same BU-Cy conditioning regimen and a standard antimicrobial prophylaxis (ciprofloxacin, fluconazole and acyclovir). None had pre-emptive parenteral nutrition or glutamine supplementation or growth factors; the allografted patients received CsA plus a short course of MTX as acute GvHD prophylaxis. The median interval from diagnosis of CDAI to BMT was 3.5 months (range 2-6).

All patients achieved complete hematologic engraftment with a median duration of severe neutropenia of 13 days (range 12-15). No transplant-related mortality was observed; no early or late abdominal complications occurred in any patient. In the post-transplant period, three patients developed an episode of fever of unknown origin, which rapidly responded to broad-spectrum antibiotic treatment. In patient #4, in whom the clinical resolution of a splenic abscess was associated with a persistently abnormal US spleen scan, the residual hypoechoic lesion continued to improve after BMT. Patients #2 and 4 died from leukemia relapse six and four months post-BMT, respectively; the others are alive and healthy, in complete hematologic remission at 8, 4, 32, 45 and 36 months post-BMT.

Severe abdominal infections may occur in about one third of acute leukemia patients undergoing intensive chemotherapy according to the EORTC-GIMEMA-AML 10 Protocol;[4,5] probably dosage and modality of cytarabine administration are relevant for the development of such complications.[4,6] Our small series shows that patients who have recovered from a life-threatening abdominal infection can safely receive additional chemotherapy courses followed by allogeneic or autologous BMT, without major delay or modification of the planned treatment strategy; there is no need for secondary broad-spectrum antimicrobial prophylaxis or pre-emptive nutritional support[8] or growth factors.[2] We confirm that these complications appear more often after induction than after consolidation[4,5] and that they can be successfully treated with vigorous conservative medical management,[5,7] if an early diagnosis is made.[5] It may be relevant that all patients in our series were condi-

Table 1. Characteristics of patients who received BMT in spite of a prior life-threatening abdominal infection.

Pt.	*Sex, age (years)*	*Type of infection*	*Interval from infection to BMT (months)*	*Type of BMT*	*BMT-related complications*	*Outcome*
1	M, 61	ileocecitis	6	autologous	none	alive
2	F, 26	ileocecitis	3	allogeneic	none	died from leukemia relapse
3	F, 25	borderline ileocecitis	3	autologous	none	alive
4	M, 49	spleen abscess	2.5	allogeneic	none	died from leukemia relapse
5	F, 53	gut-syndrome	4	autologous	1 episode of FUO	alive
6	F, 25	gut-syndrome	3	allogeneic	1 episode of FUO	alive
7	M, 18	gut-syndrome	2	autologous	1 episode of FUO	alive

FUO= fever of unknown origin, BMT= bone marrow transplantation, gut-syndrome= syndrome characterized by high fever, severe abdominal pain and tenderness, vomiting, diarrhea and/or melena, without ultrasound finding of ileal wall thickening.

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.

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Key words

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

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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