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; in particular, patients may be considered no longer eligible for bone marrow transplantation (BMT) owing to a high risk of suffering infection reactivation.

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; probably dosage and modality of cytarabine administration are relevant for the development of such complications. 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 or growth factors. We confirm that these complications appear often more after induction than after consolidation and that they can be successfully treated with vigorous conservative medical management, if an early diagnosis is made. It may be relevant that all patients in our series were conditioned...
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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References

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 manifesta-tions 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/mm³). 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. 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...