Crude incidence of MDS varies between 3.5 and 12.6/100,000 patients/year in different studies, while, in the over 70-year old population an incidence rate of 15-50/ 100,000 patients/years has been reported:[5] the case of MDS seen in a patient over 70 could, therefore, represent an increased frequency of MDS in ET treated with cytotoxic drugs.

Our study provides no evidence that HU is leukemogenic when used alone: none of the 12 patients treated with HU alone, despite a follow-up longer than 10 years, developed AL.[3,6] However, HU seems to increase the leukemic risk of other cytoreductive agents.[7,8] Low doses of ASA are not associated with increased rate of hemorrhages[9] and do not induce leukemic transformation but are able to prevent thrombotic complications totally in all patients (previous published data).

Maria Luigia Randi, Fabrizio Fabris, Antonio Girolami*

*Department of Medical and Surgical Science, II Chair of Internal Medicine and *Chair of Semeiotics and Clinical Methodology, University of Padua Medical School, Padua, Italy*

Key words

Essential thrombocythemia, myelotoxic agents, leukemia, myelodysplasia

Correspondence

Maria Luigia Randi, M.D., Dipartimento di Scienze Mediche e Chirurgiche, via Ospedale 105, 35128 Padua, Italy. Phone: international +39-049-8212668 – Fax: international +39-049-8212661.

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Ph⁺ acute lymphoblastic leukemia after iodine-131 treatment for thyroid cancer

Sir,

The incidence of leukemia after iodine-131 (^{131}I) treatment was not found to be increased in large retrospective observational studies and the risk did not seem to vary according to sex, age or time from exposure.[1,2] Leukemia after ^{131}I therapy has been reported in the medical literature to be possible following cumulative dosages of more than 800 mCi,[2] but more recently after a total dose of less than 100 mCi.[3] We report here the first case of Ph⁺ acute lymphoid leukemia following low dose radioiodine therapy. In January 1986 a 48-year old Caucasian woman with a papillary thyroid cancer following a total thyroidectomy received 95mCi of ^{131}I and two doses of 100 mCi after 6 and 12 months. In October 1998 she developed fever, diffuse bone pain, severe hyperleukocytosis, anemia, and thrombocytopenia. The bone marrow aspirate and immunophenotype were consistent with a diagnosis of acute lymphoblastic leukemia L1. Cytogenetic analysis showed an abnormal karyotype: 46XX (t9,22)(q34 q11) and the presence of the BCR/ABL (p190) rearrangement. The patient was treated with induction chemotherapy but she never achieved complete remission. She was submitted to HLA identical allogeneic transplantation. The patient died of graft-versus-host disease 24 days after transplantation.

Although ^{131}I is known to induce chromosome aberrations, large epidemiologic studies did not find a significant increase in cancer incidence.[1,2] In the last decade there have been at least 8 reports describing the occurrence of acute and chronic myeloid leukemia after ^{131}I.[1,3-9] The latency from exposure and the occurrence of acute or chronic leukemia were, respectively, 1-3 years and 5-11 years.[8] Most of the cases occurred after a cumulative dosage higher than 800 mCi; there are at least 4 reports in the last 2 years concerning acute and particularly CML after small dose of ^{131}I.[3,8,9] The involvement of lymphoid lineage seems extremely rare after ^{131}I with only 2 cases having been reported so far.[10] Interestingly the small dose of ^{131}I used and the latency observed in our patient are similar to those in patients developing CML after radioiodine. The presence of the p190 transcript is highly specific for Ph⁺ ALL and progression to a blastic crisis of CML in this patient is unlikely. Although we cannot exclude a

coincidence, our hypothesis is that the low dose of ^{131}I used for thyroid disease could generate a chromosomal aberration, particularly (t 9,22) (q34 q11) developing, with a long latency, CML or in this case Ph$^+$ ALL. Although the risk of leukemia after ^{131}I exposure cannot be considered a contraindication to ^{131}I therapy, strict hematologic follow-up of patients submitted to ^{131}I treatment is recommended. Retrospective analysis of ^{131}I exposure in large leukemia registries may also be worthwhile.

Nicola Piccirillo, Federica Sorà, Luca Laurenti, Simona Sica, Patrizia Chiusolo, Giuseppe Leone

Divisione di Ematologia, Istituto di Semeiotica Medica, Università Cattolica Sacro Cuore, Rome, Italy

Key words

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Correspondence

Simona Sica, MD, Divisione Ematologia, Istituto di Semeiotica Medica, Policlinico A. Gemelli, Università Cattolica Sacro Cuore, largo A. Gemelli, 8, 00168 Roma, Italy.

Phone: international +39-06-35503953 – Fax: international +39-06-3017319 – E-mail: emacat@rm.unicatt.it

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Two cases of fatal transfusion-associated bacterial sepsis provoked by *Providencia rettgeri*

Sir,

Bacterial contamination of blood components is an exceptional, but probably underestimated, adverse effect of transfusion and has a high mortality rate.[1] We describe two patients who died from sepsis secondary to the transfusion of blood components produced from the same donor.

A 51-year old male with ulcerative colitis who underwent a total colectomy with conservation of the rectum 20 years before was admitted for removal of the rectal stump. In the post-operative period, he developed chills without fever coinciding with the transfusion of a non-leukocyte-reduced red cell unit. The transfusion was stopped and studies of blood samples from both the patient and the bag excluded an acute hemolytic transfusion reaction. The unit, which was twenty days old, had been correctly stored at 2-6°C until just before being taken to the bedside. Half an hour later, the patient became pyrexial and shocked, with bleeding and evidence of disseminated intravascular coagulation. Suspecting septic shock, blood cultures from the patient and the bag were made, using Bactec Plus® aerobic and anaerobic bottles, which were processed in a Becton Dickinson 9240 R device. Since the patient did not improve, a laparotomy was carried out 48 hours later. Diffuse abdominal bleeding and a pelvic hematoma were found. At that moment, blood cultures from both the patient and the transfused unit grew *Providencia rettgeri* sensitive to all antibiotics administered. The patient died 24 h after the second surgical operation.

The Regional Transfusion Center that had provided the unit was informed. It located the other components produced from the same donation. Multiple cultures of the stored fresh frozen plasma were negative. The platelet concentrate had been transfused on the third day of its useful life in another hospital, using a leukocyte adsorption filter, to a patient with acute leukemia. This patient developed sepsis provoked by *P. rettgeri* and died, but the platelet concentrate was not cultured. Laboratory data concerning the isolates involved in both cases were reviewed and demonstrated the same biochemical identification and sensitivity to antibiotics. These facts strongly suggest that the bacteria provoking the two septic episodes were of the same strain. The Regional Transfusion Center located the donor, who could not attend a medical review, but by phone stated that he was healthy in the days prior, during and after the donation.

To our knowledge, this is the first description of two probably linked episodes of bacterial contami-