LETTERS TO THE EDITOR

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Anahtar Sözcükler: İzlem, Uluslararası normalleştirilmiş oran, Hemostaz

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Iron Overload in Hematopoietic Stem Cell Transplantation

Hematopoetik Kök Hücre Transplantasyonunda Aşırı Demir Yüklenmesi

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To the Editor,

We read the publication entitled "Current Review of Iron Overload and Related Complications in Hematopoietic Stem Cell Transplantation" with great interest [1]. As summarized by Atilla et al. [1], "Organ dysfunction due to iron overload may cause high mortality rates and therefore a sufficient iron chelation therapy is recommended". We would like to share the experience from our settings where there is a very high prevalence of thalassemia and transplantation is the only curative treatment.

Iron overload is common among transfusion-dependent thalassemia patients and transfusion during transplantation might increase the risk of the complication of iron overload. However, in clinical practice, the problem is not common and improvement of the patients after transplantation is reported. According to the recent report by Inati et al. [2], with standard chelation therapy, the outcome of thalassemic patients undergoing stem cell transplantation is usually favorable. The use of the standard dosage of deferoxamine, with or without phlebotomy, accompanied with close iron status monitoring can be effective [2,3]. It can be seen that stem cell transplantation can be problematic despite there being a need of hypertransfusion

during the process even though the patient might have an underlying severe iron overload condition such as thalassemia.

Keywords: Iron, Overload, Hematopoietic stem cell, Transplantation

Anahtar Sözcükler: Demir, Aşırı yüklenme, Hematopoietik kök hücre, Transplantasyon

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Reply

Dear Sora Yasri,

Thank you very much for your valuable comments and sharing your experience. We agree for your contribution. In thalassemia patients, several transplantation centers categorised risk factors prior to allogenic hematopoietic stem cell transplantation. Pesaro classification assigned patients to three arms according to the absence or presence of one, two or three risk factors: hepatomegaly > 2 cm, portal fibrosis, and irregular chelation history [1]. It should be kept in mind that in a study by Ghavamzadeh et al., liver iron overload did not change after transplant (p=0.61) but hepatic fibrosis progressed (p=0.01) [2]. Allogeneic stem cell transplantation did not reduce liver iron overload and in fact liver fibrosis increased. Also steps for reducing iron overload should be taken in the post transplant setting [3]. Iron overload is still an essential issue in both pre

and post transplant settings. Survival in transfusion-dependent thalassemia patients can be improved with proper understanding of the pathophysiology of thalassemia and iron toxicity.

Regards,

Erden Atilla, Selami K. Toprak, Taner Demirer

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Sole Infrequent Karyotypic Aberration Trisomy 6 in a Patient with **Acute Myeloid Leukemia and Breast Cancer in Remission**

Akut Miyeloid Lösemi ve Remisyonda Meme Kanserli Hastada Nadir İzole Karyotipik Bozukluk

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To the Editor,

Cytogenetic abnormalities play important roles in the diagnosis and prognosis of leukemias [1]. Trisomy 6 as the sole karyotypic aberration is infrequent in leukemias [1,2]. A 50-year-old female patient presented with fatigue. She had been treated by mastectomy and given chemotherapy (no further information available) for breast cancer 3 years ago. She had been using tamoxifen for 3 years. Her breast cancer was in remission. Physical examination was consistent with a pale appearance. Hemoglobin, neutrophils, and platelet count were 8.5 g/dL, 900/ μL, and 11,000/μL, respectively, on admission. In the peripheral blood smear, there were dysplastic features in monocytes and a few blasts were reported. In flow cytometry, CD13, CD33, CD34, CD45, CD117 (c-kit), HLA-DR, and MPO were positive. Bone marrow aspiration and biopsy revealed hypercellularity with

dysplastic and megaloblastic features in erythroid series, grade 1/3 reticulin fibrosis, and 24% blasts without ring sideroblasts, which in turn with cytometry findings were accepted as evidence of acute myeloid leukemia (AML). Bone marrow cytogenetic analysis revealed trisomy 6 (47,XX, +6 [20]) in all the metaphases (Figure 1). The patient was not in remission after the first induction treatment and she passed away due to septic shock during the second induction treatment.

Chromosome aberrations detected in therapy-related AML (t-AML) and de novo AML cases are identical but their frequencies may differ [3]. In a series at the University of Chicago, normal karyotypes were seen in 9.6% of t-AML cases [4]. In the report of Godley and Larson, among 306 patients with t-AML, 32 had solid breast cancer as the primary diagnosis [5]. Alkylating exposures and topoisomerase II inhibitors are associated with