

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 Kanseri 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/ μ L, and 11,000/ μ L, 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

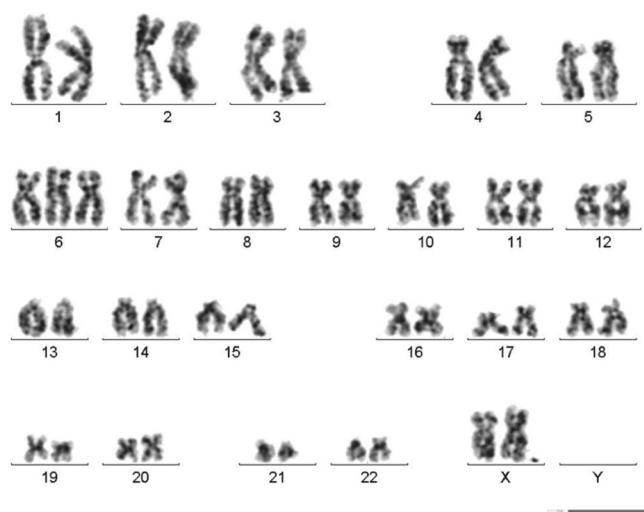


Figure 1. Bone marrow cytogenetic analysis revealed trisomy 6 (47,XX,+6 [20]) in all the metaphases.

t-AML [3,6]. Godley and Larson mentioned granulocyte colony-stimulating factor usage as a risk factor in t-AML after breast cancer [5]. Unfortunately, we do not know which agents were given for our patient's breast cancer.

Autosomal trisomies have been described in several hematologic malignancy cases. The first case of sole trisomy 6 was reported in aplastic anemia. Other reports showed that trisomy 6 was associated with hypoplastic bone marrow, dyserythropoiesis, and AML [7]. Mohamed et al. reviewed 7 patients with trisomy 6. Patients presenting with overt AML had hyperplastic marrows [8]. Our patient had hypercellular marrow, as well. Mohamed et al. also reviewed the literature and found 4 MDS cases among 22 patients with trisomy 6 [8]. The marrow examination of this case revealed secondary dysplastic leukemia. The patient of Gupta et al. had de novo AML and did not respond to the first remission induction treatment [1].

Yu et al. reviewed ten reports in PubMed describing 18 cases of AML presenting with trisomy 6 as the sole karyotypic abnormality along with 3 cases of their own [7]. They concluded

that there were no direct correlations between the number of blasts and the percentage of abnormal metaphases. They could not identify any correlation between morphology or prognosis and trisomy 6 [7].

Under these circumstances, as in our case, we lack information on the impact of trisomy 6 on prognosis in secondary AML patients.

Keywords: Trisomy 6, acute myeloid leukemia, Breast cancer

Anahtar Sözcükler: Trizomi 6, Akut miyeloid lösemi, Meme kanseri

Conflict of Interest: The authors of this paper have no conflicts of interest, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

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