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Aggressive Clinicopathological Course Of Myeloma With t(3;16)(q21;q22) Cytogenetic Abnormality

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Multiple myeloma is a heterogeneous disease and patients include a wide variety of cytogenetic anomalies reflecting the nature of the disease. [1] The aim of this paper is to report a **rare** karyotypic abnormality with an aggressive clinical course of MM.

A 56-year-old male patient was admitted to the neurosurgery clinic with dorsal shoulder pain and inability to walk in April 2011. He underwent thoracic and lumbar spinal MRI. Laminectomy was performed on the patient upon detecting masses at the levels of the first and seventh thoracic vertebrae. The patient was referred to our center when he was determined to have 'lymphoma' based on the first evaluation of his biopsy material. The specimen then was re-evaluated in our center. A high-grade hematopoietic neoplasia was detected. Immunophenotypic findings suggested the neoplasia with plasma cell origin. Immunohistochemically, neoplastic cells were positive for CD38, MUM-1 and kappa and

negative for lambda. The karyotype of the patient was identified as 44,X,-Y,del(1)(p13p35),+der(1),t(3;16)(q21;q22),-4,-13,-14, +mar[8]/46,XY[42] (fig.1).

Upon detection of the newly developed lesions in the tenth thoracic vertebra in the control imaging obtained after the radiotherapy of the patient, the treatment of vincristine-adriamycin-dexamethasone (VAD) was initiated. The paraprotein levels in the patient's serum decreased after four cycles of chemotherapy. However, the treatment was planned to continue with only velcade-dexamethasone due to severe infection after chemotherapy and the development of decubitus infection during the follow-up of the patient. After three cycles of the treatment, nodular lesions compatible with plasmacytomas appeared in his skin. The treatment strategy was changed since the patient was considered as the progression responded better to VAD chemotherapy. The patient was hospitalized due to pneumonia after chemotherapy and died due to severe sepsis.

The t(3;16)(q21;q22) anomaly reported in this case is a quite rare cytogenetic anomaly and according to the databases that we have investigated [2,3], it was reported only in three adults and one child to date [4]. One of these three adult cases was a male patient with the diagnosis of myelodysplastic syndrome (MDS). In this patient, the translocation of t(3;16)(q21;q22) was found in the complex karyotype as in our case [5]. The other two cases were female patients and they were diagnosed with acute myeloid leukemia (AML) developed after acute myelomonocytic leukemia and primary tumor Mantle cell lymphoma, respectively. None of the reported cases were diagnosed with MM. Therefore, we report the t(3;16)(q21;q22) anomaly which is a quite rare translocation in MM patient for the first time in this case. The prognostic effect of t(3;16)(q21;q22) is unknown. In the presented case, the anomaly of t(3;16)(q21;q22) coexisted with the anomalies of 1p deletion and monosomy 13 which are the markers of progressive disease and short survival, in other words, poor prognostic markers [6]. The clinical course of our case progressed in a manner that was consistent with poor prognosis. We believe that our case contributed to the literature since it is the first MM case with a very rarely encountered translocation.

Authors' Disclosures of Potential Conflicts of Interest

No potential conflicts of interest relevant to this article were reported.

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Image of the patient karyotype