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Case Report
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IGH-MECOM (EVI1) Rearrangement in Therapy-Related Myeloid Neoplasm

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Abstract

Rearrangements involving EVI1 (MECOM) include in (3) (q21q26), t (3;3) (q21; q26), and other translocations involving 3q26, which result in overexpression of the EVI1 oncogene and are associated with aggressive myeloid neoplasms, including therapy-related Acute Myeloid Leukemia (AML) and Myelodysplastic Syndromes (MDS).

Keywords: Myeloid neoplasm; Myelodysplastic syndromes; Leukemia

Introduction

Rearrangements involving EVI1 (MECOM) include in (3) (q21q26), t (3;3) (q21; q26), and other translocations involving 3q26, which result in overexpression of the EVI1 oncogene and are associated with aggressive myeloid neoplasms, including therapy-related Acute Myeloid Leukemia (AML) and Myelodysplastic Syndromes (MDS) [1,2].

The immunoglobulin genes, including Immunoglobulin Heavy Chain Genes (IGH), light kappa (k) chain genes (IGK) and Light Lambda (Λ) Chain Genes (IGL), are frequently rearranged in B-cell tumors. These rearrangements result in a juxtaposition of IG enhancers to the vicinity of oncogenes such as MYC and BCL2, leading to their overexpression and activation [3]. We reported a case of Immunoglobulin Heavy Chain (IGH) and EVI1 rearrangement in therapy related myeloid neoplasm.

Case Presentation

A 75-year-old male presented with pancytopenia, a history of lung carcinoma three years ago and chemotherapy with cytotoxic agents. Bone marrow biopsy was performed (Figure 1). The blasts in the bone marrow aspirate are moderate to large in-sized with a high nuclear-to-cytoplasmic ratio and finely dispersed chromatin with prominent nucleoli (Figure 2). Prussian blue iron stain showed ringed side oblasts (Figure 3).

Bone marrow cytogenetics demonstrated monosomy 7, and a balanced translocation between chromosome 3 and 14 (45, XY, t (3;14) (q26; q32), -7) (Figure 4). Fluorescence In Situ Hybridization (FISH) study on the abnormal metaphases showed MECOM (EVI1) rearrangement (Figure 5). The overall findings on FISH and karyotype studies confirmed an IGH-MECOM (EVI1) rearrangement. The patient was diagnosed with therapy-related myeloid neoplasm, consistent with Therapy-Related Myelodysplastic Syndrome (t-MDS).

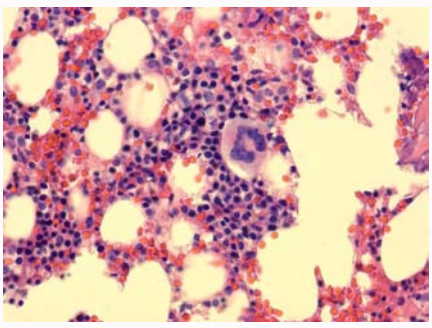

Figure 1: Bone marrow core biopsy of the patient.

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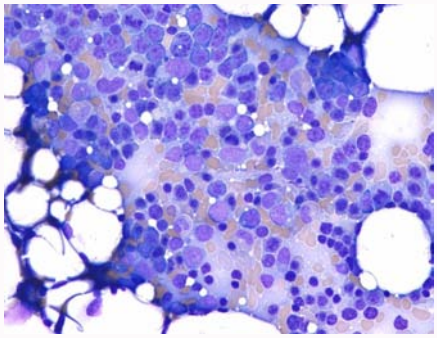

Figure 2: Bone marrow aspirate of the patient.

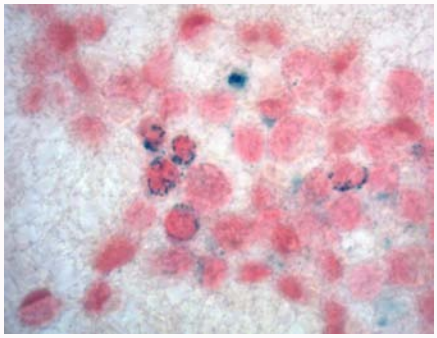

Figure 3: Prussian blue iron stain of the bone marrow aspirate.

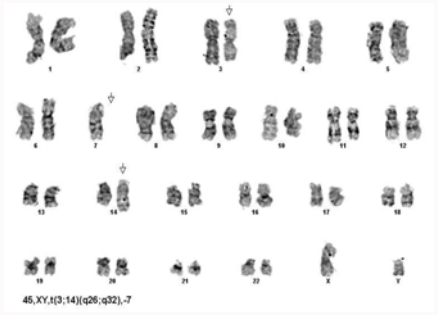

Figure 4: Cytogenetic karyotype study of the bone marrow aspirate.

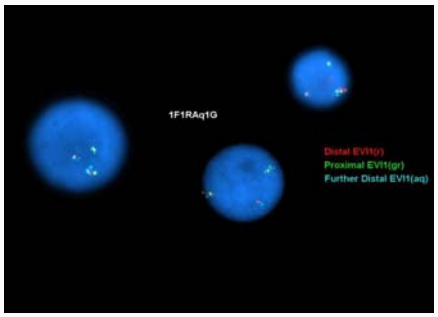

Figure 5: Fluorescence in situ hybridization (FISH) study of the bone marrow aspirate.

Discussion

We presented here a case report of therapy-related myeloid neoplasm with IGH-MECOM (EVI1) Rearrangement, which has not been reported previously. IGH rearrangement is one of the most common findings in mature B-cell lymphoma, which leads to a juxtaposition of the potent IGH enhancers near the silent oncogenes, leading to their expression and activation. IGH can also fuse with oncogenes such as MECOM in myeloid neoplasm. MECOM is a transcription factor that can induce upregulation of cell cycles and block granulocytic differentiation of hematopoietic stem cells.

Therapy-Related Myeloid Neoplasms (t-MNs) is a distinct category in the classification for patients who develop myeloid neoplasms following cytotoxic therapy. The t-MNs may be further subdivided as therapy-related MDS or AML (t-MDS or t-AML). Associated cytogenetic abnormality is important for determining therapy and prognosis. In myeloid malignancies such as acute myeloid leukemia, myelodysplastic syndromes as well as myeloproliferative disorders, involvement of 3q26 in balanced rearrangements is highly suggestive of MECOM (EVI1) rearrangement. The cytogenetic findings of our case report demonstrated a 3q26 rearrangement, an associated monosomy 7, and complex chromosomal aberrations.

In summary, MECOM (EVI1) rearrangement at 3q26 is a well-established adverse prognostic marker in therapy-related myeloid neoplasms. 3q26 rearrangements are frequently associated with monosomy 7 and complex chromosomal aberrations [3]. Our studies also provide evidence that B-cell tumor-specific oncogenic mechanism is also present in myeloid neoplasms.

References

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