An Unusual Case of Acute Myeloid Leukemia with t(8:21) Presenting with Hypereosinophilia Showing Dysplastic Features

Abstract
Acute myeloid leukemia (AML) with specific genetic abnormalities is a clinically, biologically, and prognostically distinct category with some of the entities in it displaying characteristic morphology. AML with t(8;21) is one such subtype carrying favorable prognosis with specific blast morphology. Eosinophilia, characterized by increased peripheral eosinophil counts, has been described till date in AML with inv (16); however, hypereosinophilia with prominent dysplastic features has yet not been seen with any AML subtype. We report the case of an 8-year-old child presenting with massive splenomegaly, hypereosinophilia, and low marrow blast percentage. The initial clinical and hematological impression was that of a chronic myeloproliferative neoplasm, which was later diagnosed as AML with t(8;21) with the help of cytogenetic studies. The case report highlights the unusual and extremely rare presentation of this AML subtype and the importance of cytogenetic studies in definite categorization, especially in cases with overlapping morphological and immunophenotypic findings.

Keywords: Acute myeloid leukemia, cytogenetics, hypereosinophilia, splenomegaly

Introduction
Acute myeloid leukemia (AML) results from clonal proliferation of undifferentiated myeloid precursors leading to bone marrow failure. Various subsets of AML have been defined depending on the characteristic cytogenetic abnormality, which have prognostic implications. One such subset is AML with t(8;21) (q22;q22), which has a favorable prognosis and distinct biological characteristics.[1] It was the first cytogenetic abnormality detected in AML.[1] The translocation involves RUNX1 gene present on the chromosome 21 and RUNX1T1 gene on chromosome 8. This abnormality is found in 5%–10% of all AML cases and is most commonly seen in younger patients.[1] Morphologically, this category has characteristic blasts and increased numbers of neutrophils, eosinophils, and their precursors to an extent that the picture sometimes resembles that of a chronic myeloproliferative neoplasm, especially when the blast counts are low.[1] However, AML with t(8;21) presenting initially with massive splenomegaly and hypereosinophilia with many dysplastic eosinophils and their precursors is extremely rare. Hypereosinophilia in itself enlists many differential diagnoses and is an alarming finding in rapid turnover hematological states. It can be found either as an associated finding in acute leukemia or as a clonal proliferation in chronic eosinophilic leukemia. The dilemma might not always be entirely solved on morphology alone.

Case Report
An 8-year-old girl presented with fever for 3 months, pain in the abdomen, and progressive abdominal distension for 1 month. Fever was intermittent, high grade, and was not associated with vomiting. There was a history of weight loss, loss of appetite, and generalized weakness. There was no breathing difficulty, bleeding from orifices, or loose stools. On general examination, her general condition was poor. She was febrile and pale. Few petechial spots could be identified over the abdomen. There was no lymphadenopathy or icterus. On local examination, there was massive splenomegaly (10 cm below the costal margin) and mild hepatomegaly (1 cm below costal margin).

Hemogram showed hemoglobin level of 8.4 g/dl and a total leukocyte count of 120,000/ cubic mm with eosinophils accounting for 52%. Peripheral blood...
AML with t(8:21) with hypereosinophilia showing dysplastic eosinophils and simulating chronic myeloproliferative neoplasm

Figure 1: (a) Peripheral blood smear shows eosinophils with dysplastic features such as nuclear hyperlobation and coarse granules. Inset shows the presence of blasts (Giemsa, ×1000). (b and c) Diffuse replacement of bone marrow by eosinophils and their precursors (H and E ×1000, 100 respectively). (d) Bone marrow blasts showing positivity for CD34 (Immunohistochemistry, ×1000)
features is extremely rare and is uncommon in AML with t(8;21). This case report highlights the still another type of morphology that can be seen with this karyotypic abnormality. The exact pathogenesis behind eosinophilia observed in these cases is still unknown. However, a few published reports have shown that these atypical eosinophils might be derived from the leukemic clone itself by increased expression of interleukin-5 receptors as demonstrated by in vitro studies.[5]

Due to a lack of much data on this, how well these patients respond to the conventional antileukemic therapy is unknown. AML with t(8;21) in general has a favorable prognosis; however, hypereosinophilia in itself can cause a lot of parenchymal damage to the lungs, heart, and gastrointestinal tract due to the liberated cytokines, leading to organ fibrosis and increased morbidity.[5]

Our patient received both steroids and antileukemic therapy with daunorubicin and cytarabine. Repeat PBF after the completion of the first cycle showed marked reduction in the total leukocyte counts including the absolute eosinophil numbers. The patient is also recovering and is afebrile with moderate decrease in the spleen size.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

References
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