# CORRESPONDENCE

## ONCOGENES AND ACUTE LEUKEMIAS

*To the Editor:*

Modern cytogenetic techniques have placed a powerful new tool in the hands of clinicians and researchers.[1] Recently, Arthur and Bloomfield[2] reported del(16)(q22) as the sole chromosomal abnormality in five untreated acute nonlymphocytic leukemia (ANLL) patients. They believe this represents a new cytogenetic-clinical entity of ANLL. A similar association with a somewhat different interpretation has been made by Larson and coworkers,[3] who described an "internal inversion involving breaks in both the short and the long arms of chromosome 16"—inv(16)(p13q22). In both series,[2,3] significant marrow eosinophilia was noted in association with a myelomonocytic morphology (FAB M4). Often, the eosinophilic precursors contained large and mixed eosinophilic-basophilic granules. Within the past month, two adult acute leukemia patients with abnormal chromosome 16 have been studied in our laboratory. The first, age 34, had acute myelomonocytic leukemia (AMML) with 10% marrow eosinophilia. A clone with a del(16)(q13q22) and an inv(18)(p11q21) was found in 6 of the 25 metaphases analyzed. The patient entered a complete remission after therapy with daunomycin and Ara-C and has received consolidation treatment.

A second patient, a 55-yr-old female, presented with hepatosplenomegaly and morphology consistent with acute undifferentiated leukemia (peroxidase and esterase stains negative; CALLA negative; TdT negative; an initial white count of 40,000/dl). A clone with a del(16)(q13q24) was found in 4 of the 31 metaphases analyzed. She entered remission with daunomycin, prednisone, and vincristine.

Although patients with eosinophilia have an abnormal chromosome 16, which may be a result of a simple deletion at q22[2] or inv(16)(p13q22)[3] or del(16)(q13q22), as in our Patient 1, abnormalities involving chromosome 16 can also be seen in other types of acute leukemia without eosinophilia, as in our patient 2. In Patient 2, however, the deletion involved segment q13q24. Therefore, while chromosomal studies can help us in differentiating malignant from nonmalignant disease in some cases, at this time, the appropriateness of considering the abnormal chromosome 16 as having a unique morphologic representation appears to be premature.

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3. Larson RA, LeBeau MM, Bitter MA, Vardiman JW, Golomb HM, Rowley JD: Association of inv(16)(p13q22) with marrow eosinophilia in acute myelomonocytic leukemia (AMMOL). Blood 60(Suppl 1):131a, 1982 (abstr 453)

*To the Editor:*

We have read with interest the letter of Goh, Qazi, Owens, Herrmann, and Bennett. They report two patients with acute leukemia and partial deletion of the long arm of chromosome 16. The first appears to fit into the subgroup of patients with acute nonlymphocytic leukemia (ANLL) who have a structural abnormality of chromosome 16 and marrow eosinophilia that we and others have described.[1,2] Precise definition of the abnormality of chromosome 16 has been problematic. Both a del(16)(q22) and inv(16)(p13q22) have been reported, and this new case has a del(16)(q13q22). Because of technical difficulties in evaluating the abnormal 16, it remains unclear at present whether or not all of these patients have the same abnormality. If they are truly different, then the constant cytogenetic feature appears to be a break in band 16q22. Further studies will be important in clarifying this point.

The authors suggest that the abnormal 16 has been considered as having a unique morphological representation. We do not believe that such has been the case. What we have reported is an association of an abnormality of chromosome 16 with marrow eosinophilia among a subgroup of patients with newly diagnosed ANLL.[1,3] This does not imply that the abnormal 16 is specific for ANLL, nor that, in other diseases, the same abnormality of chromosome 16 will be accompanied by marrow eosinophilia. Indeed, we have found a partial deletion of the long arm of chromosome 16 as the sole karyotypic abnormality in two patients who did not have marrow eosinophilia. The first was a 13-yr-old male who had common ALL antigen positive acute lymphoblastic leukemia, and the second a 69-yr-old female with follicular, predominantly small cleaved cell lymphoma. Thus, it is not surprising that the second patient of Goh et al. with acute undifferentiated leukemia did not have eosinophilia. What we conclude from the data currently available is that the finding of a structural abnormality of chromosome 16 involving band q22 at diagnosis of ANLL appears to identify a specific subgroup of patients with abnormal marrow eosinophils and monocytoid blasts.

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