About the t(8;13)(p11;q12) Clinico-Pathologic Entity

To the Editor:

We read the interesting report by Inhorn et al describing a clinico-pathologic entity characterized by T-cell lymphoblastic lymphoma, eosinophilia, myeloid hyperplasia/malignancy, and t(8;13)(p11;q12) presenting both in bone marrow and in lymph node cells and involving a 1.5-mb region of chromosome 13q12. We wish to report our recent observation on a 53-year-old Caucasian woman who presented with fever, dyspnoea, hemoptysis, and gastrointestinal hemorrhage. Hepatosplenomegaly without lymphadenopathy or mediastinal mass was observed.

The blood count was as follows: white blood cells, 128 × 10^9/L (69% blasts, 8% promyelocytes, 20% segmented neutrophils, 1% eosinophils, and 2% lymphocytes); red blood cells, 6.2 × 10^12/L; hemoglobin, 21.1 g/dL; hematocrit, 62% (these values were confirmed); and platelets 45 × 10^11/L. The bone marrow aspirate showed hypercellularity and invasion by 9S% of blasts, which strongly expressed HLA-DR, CDIO, and CD24, moderately expressed CD34 and CD19, and weakly expressed Tdt. No surface Ig could be detected. The more mature B-cell markers CD22 and FMC7 were also negative. The T-cell markers CD2, CD3, CD4, CD5, CD7, and CD8 were all negative, as were the myelomonocytic markers CD13, CD14, and CD33. A common B-cell acute lymphoblastic leukemia (B-ALL), classified as L2 according to the French-American-British criteria, was diagnosed. In addition, a slightly increased proportion of bone marrow eosinophils (5%) and basophils (with clusters of mast cells) was observed. These findings, along with the high red blood cell count in the peripheral blood, suggest involvement of the myeloid lineage in this malignancy. Cytogenetic analysis of the bone marrow showed a t(8;13)(p11;q12) in 6 of 8 fully karyotyped metaphases (Fig 1). Chemotherapy associating vincristine, daunorubicine, asparaginase, prednisone, cyclophosphamide, cytosine arabinoside, 6-mercaptopurine, and methotrexate was started. A complete clinical remission was achieved at day 28 and the patient received a classical consolidation regimen and prophylactic cranial irradiation, followed by maintenance therapy with methotrexate and 6-mercaptopurine. During the following 36 months, a borderline proportion of bone marrow blasts (ie, 5% 36 months after diagnosis) and eosinophils, as well as a persistent t(8;13)(p11;q12) in 10% to 75% of the cells were repeatedly recorded. Because of psychologic problems, the patient was then lost to effective follow-up, but is alive 54 months after diagnosis.

The present case clinically as well as cytogenetically obviously belongs to the recently defined clinico-pathologic entity associating T-cell lymphoblastic lymphoma, eosinophilia, myeloid hyperplasia/malignancy, and a translocation t(8;13). However, in our case, as well as in one published case, no T-cell lymphoblastic lymphoma was observed. Instead, our patient had a common B-ALL, suggesting that T-cell lymphoblastic lymphoma is not the only way the lymphoid malignancy may present in this multilineage disorder.

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Fig 1. Partial karyotype showing t(8;13)(p11;q12).
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