

*Case observed***A new chromosome abnormality in acute nonlymphocytic leukemia****M. J. J. Vekemans and D. W. Esseltine**

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Chromosome banding techniques have markedly increased our understanding of the type and frequency of chromosome abnormalities associated with discrete subgroups of acute nonlymphocytic leukemia (ANLL) in adults (First International Workshop on Chromosomes in Leukemia 1978; Second International Workshop on Chromosomes in Leukemia 1980). In childhood, however, ANLL is uncommon and no clear correlation exists yet between the ANLL subgroup and the cytogenetic findings (Benedict et al. 1979; Hagemeyer et al. 1979; Morse et al. 1979; Brodeur et al. 1983). We report a case of childhood ANLL with a heretofore undescribed cytogenetic abnormality.

A 2½-year-old girl, with a family history of colon cancer, neurosensory deafness, and histiocytosis X, presented with a 3-day history of fever, cough, and generalized malaise. On physical examination, she was febrile, in mild respiratory distress, with a palpable spleen tip. A chest X-ray revealed a right middle lobe pneumonia. The leukocyte count was 6600/mm³ with 22% blasts, many of which contained Auer rods. The coagulation profile and CSF were normal. The hemoglobin was 11.9 g/dl and the platelet count 70,000/mm³. A bone marrow examination established the diagnosis of M2 type AML (Bennet et al. 1976). Cytochemistry of the blast granules (PAS and alpha naphthylesterase: negative, chloroacetate esterase and peroxidase: positive) confirmed the diagnosis. A cytogenetic analysis was performed on preparations from both a peripheral blood and a bone marrow sample. After analyzing the bone marrow preparations with trypsin-Giemsa banding, the chromosome complement was 46,XX,t(6;9)(q13;p24) (ISCN 1978). The constitutional chromosome complement was normal. After one course of induction therapy with cytosine arabinoside and daunorubicin, the patient remained in remission on maintenance therapy for 10½ months. She relapsed and subsequently died, 14 months after diagnosis, of progressive leukemia while being prepared for bone marrow transplantation.

To our knowledge, this is the first report of an M2 type AML with a chromosomal rearrangement involving the long arm of chromosome 6 (q13) and the short arm of chromosome 9 (p24). This rearrangement also affects the chromosomal segment on the long arm of chromosome 6 (q13) which has been associated with several lympho- and myeloproliferative disorders (Lawler et al. 1975; Rowley et al. 1977; Alimena et al.

1977). This suggests that this site is involved with tumor progression rather than with the first step of malignant transformation of myeloid cells. However, the significance of this form of (6;9) translocation awaits further study in terms of prognostic value and we would be very interested to hear of similar observations from other investigations.

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