Clinical Characteristics and Treatment Outcome of Childhood Acute Lymphoblastic Leukemia with the t(4;11)(q21;q23): A Collaborative Study of 40 Cases


The t(4;11)(q21;q23) chromosomal abnormality was identified in 40 (2%) of 1,986 children with newly diagnosed acute lymphoblastic leukemia (ALL). This translocation was associated with female sex (63%), age less than 1 year (60%), hyperleukocytosis (median leukocyte count, 156.5 x 10^9/L), CD10+/CD19- B precursor cell immunophenotype, and myeloid-associated antigen (CD15) expression (63%). Nearly all cases had at least some CD24+ blast cells. The CD10+/CD19-/+/CD24- phenotype was found in 20 of the 32 (t4;11) cases tested. None of the 40 cases had the cytogenetic finding of hyperdiploidy >50, which is a favorable prognostic feature. For clinical comparison, the (t4;11) cases were divided into three groups according to age at diagnosis: less than 1 year (n = 24), 1 to 9 years (n = 8), and ≥10 years (n = 8). Compared with older patients, infants were more likely to have initial central nervous system leukemia (P = .05) and less likely to have pre-B-cell ALL (P = .05). Complete continuous remission has been maintained in only 7 of 24 infants and 2 of 8 patients aged ≥10 years, in contrast to 7 of 8 children in the intermediate age group (P = .048). These findings suggest that the t(4;11) is an adverse prognostic feature in these two age groups.

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Materials and Methods

Leukemia cells from a total of 1,986 children with ALL were successfully studied for their karyotype. Of these patients, 515 were admitted to Total Therapy Studies X through XII at St Jude Children’s Research Hospital (SJCRH) and 1,471 were enrolled in Pediatric Oncology Group (POG) studies 8493 and 8602 from 1986 to 1989. The 1,986 successfully karyotyped cases represent 73% of all children enrolled in POG studies. Bone marrow specimens from patients enrolled in the POG studies were shipped to the University of Alabama at Birmingham (UAB) for chromosome analysis. Informed consent was obtained in each instance in accord with institutional guidelines.

Morphologic studies and immunophenotyping. Cases were classified according to French-American-British (FAB) criteria, based on bone marrow cell morphology and cytochemical staining characteristics. Bone marrow cells were separated on a Ficoll-Hypaque gradient. Immunophenotyping was performed at SJCRH and at Duke University Medical Center (Durham, NC), using slightly different panels of monoclonal antibodies to lymphoid-associated antigens (CD2, CD5, CD7, CD19, CD20, CD21, CD22, and CD24), myeloid-associated antigens (CD13, CD15, and CD33), and lineagespecific antigens (CD10, CD34, CD45, and HLA-DR). Cells were also tested for surface (sIg) and cytoplasmic Ig (cIg). Cell surface antigens were detected by a standard indirect immunofluorescence assay using flow cytometry and were considered positive if they were expressed in 20% or more of the blasts cells. Significant cIg expression was defined by the presence of Ig in the cytoplasm of 10% or more of blasts. Based on their pattern of reactivity, lymphoblasts were classified as early pre-B (CD19+, CD22+, CD24+, CD5-, CD7-, cIg-, sIg-), pre-B (cIg+), B cell (sIg+), or T cell (CD5+ plus CD7+).

Chromosome analysis. Bone marrow samples from the patients studied at SJCRH were processed immediately after collection by the direct method. Samples from POG institutions other than SJCRH were placed in RPMI 1640 supplemented with 15% fetal calf serum and shipped overnight to the UAB cytogenetic laboratory. On arrival, samples were placed in short-term (24-hour) culture. Routine methods were used for culture harvest, slide preparation, and GTG-banding. Chromosomal abnormalities were classified according to the International System for Human Cytogenetic Nomenclature.

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Statistical analysis. Differences in the distribution of clinical and biologic features between subgroups of cases were tested by the exact conditional chi-square for categorical variables or the Kruskal-Wallis test for continuous variables. Life tables for event-free survival were constructed by the method of Kaplan and Meier, with differences compared by the logrank test.

RESULTS

Of the 1,986 cases studied, 40 (2%) were found to have a leukemic cell line with the t(4;11)(q21;q23). These children (25 girls and 15 boys) ranged in age from 1 month to 14 years 8 months at diagnosis (median, 9 months) (Table 1). None of these patients had a mediastinal mass at diagnosis. Central nervous system (CNS) leukemia was present at diagnosis in eight infants. Leukocyte counts ranged from 1.6 to 832 × 10^9/L (median, 156.5 × 10^9/L), platelet counts from 5 to 345 × 10^9/L (median, 44 × 10^9/L), and hemoglobin levels from 3.1 to 12.9 g/dL (median, 6.6 g/dL). Leukemic cell L2 morphology was found in 5 of 38 cases tested.

Leukemic cell immunophenotyping was performed in 35 patients, all of whom were found to have B-precursor-cell ALL (Table 2). Of the 33 cases that could be subclassified, 26 had early pre-B and 7 had pre-B-ALL. Only one case (pre-B) expressed CD10 (common ALL antigen; CALLA), whereas HLA-DR and CD45 were each found in all cases tested (n = 34 and n = 27, respectively). Weak expression of CD7, with only a slight shift of the fluorescence histo-

### Table 1. Clinical and Laboratory Findings in the 40 Cases With t(4;11)(q21;q23)

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<th>FAB</th>
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Abbreviations: B, black; W, white; H, Hispanic; O, Oriental; ND, not done; BM, bone marrow relapse; T, testicular relapse; IF, induction failure.

*Some clinical features and early treatment outcome have been reported previously.\textsuperscript{53,55,59,60}
gram compared with the control, was found in 5 of 33 cases tested (nos. 4, 11, 15, 22, and 38).

CD24 was expressed in 13 (46%) of 28 cases tested and CD19 in all of the 32 cases tested. The percentage of blast cells positive for CD19 was greater than that for CD24 in all cases except one, which was positive for CD10 (Table 2). Examination of the fluorescence histogram in these cases showed that expression was not simply due to low levels of expression of CD24, but reflected the presence of a distinct CD24- subpopulation. Two-color immunofluorescence experiments in cases positive for both CD19 and CD24 antigens confirmed the presence of a distinct CD24- subpopulation (Fig 1). Of the 32 cases analyzed for myeloid-associated antigens, significant CD15 expression was found in 20, two of which also expressed CD33. In four additional cases, CD15 antigen expression was found in 10% to 20% of cells.

The modal chromosome number of the leukemic cell lines was 46 in 36 cases, 47 in two cases, and 48 and 82 in one case each. Structural or numerical chromosomal abnormalities in addition to the t(4;11) were found in 11 cases (Table 3). Three of these cases (nos. 25, 26, and 33) had two or three leukemic lines. The near-tetraploid line in case 40 appears to have arisen from endoreduplication, as the t(4;11) was found in two copies.

For comparison of clinical and laboratory features, the 40 cases were divided into three subgroups by age: less than 1 year (cases 1 through 24), 1 to 9 years (cases 25 through 32), and ≥10 years (cases 33 through 40) (Tables 1 and 4). There were no significant differences among the three groups with respect to sex, race, leukocyte count, platelet count, hemoglobin level, FAB classification, or myeloid-associated antigen expression. However, infants were more likely than those in the other age groups to have CNS leukemia at diagnosis (8 of 24 v 0 of 7 v 0 of 8, P = .05), and were less likely to have the pre-B immunophenotype (1 of 19 v 3 of 7 v 3 of 7, P = .05).

Four patients failed to achieve a complete remission and 20 have relapsed or died (Table 1). CNS relapse has occurred in four infants but in none of the older patients. Perhaps more important is the finding of a significant difference in event-free survival favoring patients 1 to 9 of these groups.
CHILDHOOD ALL WITH THE t(4;11)

years old compared with older patients and infants: 7 of 8 children aged 1 to 9 years versus 7 of 24 infants and 2 of 8 patients aged ≥10 years remain in continuous complete remission (P = .048, logrank test; Fig 2). Of the 16 patients who remain in remission, eight have been followed for less than 1 year. When this comparison was limited to patients treated after 1984 with contemporary treatment programs, a trend (P = .12) toward a better outcome in patients aged 1 to 9 years was still present (6 of 7 v 7 of 22 infants v 2 of 7 >10 years remain in remission).

DISCUSSION

The reported frequency of the t(4;11) in patients with ALL has ranged from 1.6% to 11% for children. In our study of almost 2,000 cases of childhood ALL, this chromosomal abnormality occurred in 2%. Thus, the t(4;11) is one of the most common specific chromosomal translocations in childhood ALL, along with the t(1;19), found in 5% to 6% of cases, and the t(9;22), seen in 2.3% to 5%.

In this study and in prior reports of small series (summarized in Table 4), females and infants are disproportionately represented among cases of acute leukemia with the t(4;11). Whereas infants account for only 2% to 3% of all children with ALL, this age group comprised 60% of our sample and 47% of other reported cases in children. Hyperleukocytosis is a frequent presenting feature of t(4;11) ALL in all age groups. A leukocyte count greater than 100 x 10⁹/L was found in more than half of the children we studied (14 of 24 infants, 6 of 8 patients aged ≥10 years, and 4 of 8 patients aged 1 to 9 years). By contrast, in large reported series of children with ALL, hyperleukocytosis is present at diagnosis in 23% to 50% of infants, 18% of adolescents, and 10% of patients in the intermediate age group. In fact, the median leukocyte count of our patients with the t(4;11), 156.5 x 10⁹/L, is higher than that reported for any other phenotypic or karyotypic subgroup, including T-cell ALL (100 x 10⁹/L), pre-B ALL with the t(1;19), and Philadelphia chromosome-positive ALL (33 x 10⁹/L). Among t(4;11) cases, infants had a significantly higher frequency of CNS leukemia than older children. This finding is not surprising in that leukemias in infancy are typically associated with a high frequency of CNS leukemia.

Despite comparably high leukocyte counts, our patients aged 1 to 9 years had a significantly better treatment outcome than infants and older patients with the t(4;11). Seven of eight patients in this age group, compared with 9 of the 32 other patients, remain in remission. Thus, the adverse prognosis of the t(4;11) may differ at the molecular level among the age subgroups. In this regard, different breakpoints on chromosome 19 have been reported for cases with the t(11;19)(q23;p13). Although the independent significance of the t(4;11) is not yet known, it is noteworthy that, with contemporary treatment, long-term event-free survival can be achieved in approximately one third of infants and one half of adolescent ALL cases, whereas less than 10% of infants or patients aged ≥10 years with the t(4;11) ALL in this study are expected to be long-term survivors.

Ultrastructural, immunophenotypic, and in vitro culture studies have demonstrated marked lineage heterogeneity in cases with the t(4;11). Although many reported cases have had myelomonocytic characteristics, most have been classified as B-precursor-cell ALL (CD19+, HLA-DR+). Only rarely have T-cell or B-cell phenotypes been reported in patients with this translocation. Indeed, all 35 evaluable cases in this study had B-precursor-cell ALL. During the study period, we have not encountered any case of acute myeloid leukemia with the t(4;11).

CD10 is expressed in 94% of all childhood ALL cases,
and the lack of its expression is associated with a poorer treatment outcome.68 This antigen was present in only 1 of our 34 cases tested, and 7 of 69 previously described t(4;11) cases.7,11,16,23,24,29 Interestingly, the one CD10+ case in our series was also the only infant with the pre-B cell phenotype. Other unusual phenotypic features of the t(4;11) cases included the presence of CD15+ and CD24- blast cells. CD24, a B-lineage marker present in more than 95% of cases of ALL, is present in less than 5% of cases of the t(4;11).14 In contrast, CD15 expression was demonstrated in 20 of our 32 t(4;11) cases and in 8 of 16 previously reported cases.72

For leukemic cell immunophenotype, the CD10+/CD19+/CD24- phenotype was found in only 13 non-t(4;11) cases. Eight of these 13 cases were CD10+/CD15+/CD19+/CD24-; of these, four had successful cytogenetic studies that showed 11q23 abnormalities in three. In the remaining case, the t(4;11) was found in the institutional laboratory but chromosome study in the reference laboratory was unsuccessful. The other five cases with the CD10-/CD19+/CD24- phenotype ALL had a clonal karyotypic abnormality other than the t(4;11), with involvement of 11q23 in two. Of our 32 t(4;11) cases tested, all but one had the CD10+/CD19+/CD24- phenotype. Among the approximately 2,000 ALL cases we studied, the CD10+/CD19+/CD24- phenotype was found in only 13 non-t(4;11) cases. Eight of these 13 cases were CD10+/CD15+/CD19+/CD24-; of these, four had successful cytogenetic studies that showed 11q23 abnormalities in three. In the remaining case, the t(4;11) was found in the institutional laboratory but chromosome study in the reference laboratory was unsuccessful. The other five cases with the CD10+/CD19+/CD24- phenotype ALL had a clonal karyotypic abnormality other than the t(4;11), with involvement of 11q23 in two. Of our 32 t(4;11) cases tested, all but one had the CD10+/CD19+/CD24- phenotype. Among the cases with complete immunophenotyping and satisfactory chromosomal studies, 11q23 abnormalities were detected in all 24 with the CD10+/CD15+/CD19+/CD24- phenotype [21 of which were the t(4;11)], and in 37 of 40 with the CD10+/CD19+/CD24- phenotype. It is not known whether the CD10+/CD15+/CD19+/CD24- phenotype is present in normal cells during an early stage of differentiation. If this phenotype is unique to leukemic cells, it may provide a means of monitoring these patients for residual leukemia.

That five of our cases showed expression of the T-cell-associated antigen CD7 is not surprising, given this antigen’s broad spectrum of activity. CD7 antigen expression is present in many cases of acute myeloid leukemia and may be a marker of an early multipotent progenitor.73 None of our patients with the t(4;11) and only two previous cases80 had blast cell hyperdiploidy >50, a favorable cytogenetic feature found in one fourth of all childhood ALLs.80 One of our cases was classified as near-tetraploid, a subgroup that differs clinically and biologically from the hyperdiploid >50 group.77 Finally, the t(7;9) noted in case 39 is a relatively common structural abnormality in t(4;11) cases.71,11,13,15,16,23,29

In summary, the t(4;11) is one of the most common nonrandom translocations in childhood ALL, accounting
for 2% of newly diagnosed cases. Cases with the (t(4;11)) are associated with female sex, very young age at presentation, hyperleukocytosis, the CD10<sup>+</sup>/CD15<sup>-</sup>/CD19<sup>-</sup>/CD24<sup>-</sup>/ immunophenotype, and poor treatment outcome for infants and patients aged ≥ 10 years. Additional studies are clearly merited to determine the independent prognostic significance of the (t(4;11)) and to assess possible molecular differences in this translocation for different age groups.

ACKNOWLEDGMENT

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CHILDHOOD ALL WITH THE t(4;11)

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CH Pui, LS Frankel, AJ Carroll, SC Raimondi, JJ Shuster, DR Head, WM Crist, VJ Land, DJ Pullen and CP Steuber

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