New Recurring Chromosomal Translocations in Childhood Acute Lymphoblastic Leukemia


We identified seven new recurring translocations among 483 cases of acute lymphoblastic leukemia (ALL) with adequate chromosome banding studies. Four were apparently balanced [t(1;3)(p34;q21), t(7;9)(p15;p23-p24), t(12;13)(p13; q14), t(17;19)(q22;p13)], while three were unbalanced with the formation of a dicentric chromosome [dic(7;9)(p13;p11), dic(7;12)(p11;p12), and dic(12;17)(p11;p11-p12)]. One translocation was observed in five cases, two in four cases, and the remaining four in two cases each. The modal chromosome numbers in these 21 cases were 45 (n = 11), 46 (n = 8), and 47 (n = 2). Eight of the 11 cases with a dicentric chromosome had a modal number of 45. Only a single translocation was found in 14 cases (67%), representing the sole structural abnormality in six cases. In three of the seven translocation subgroups, the blast cells were consistently of B lineage (pre-B, early pre-B, or both); in all others, they represented both the B and T lineages. The small size of these subgroups prevented definitive clinical correlations, although it may be important that two of the four cases with a t(17;19) and an early pre-B-cell immunophenotype had disseminated intravascular coagulation, an event usually observed in acute promyelocytic leukemia or T-cell ALL. These findings add substantially to the existing list of nonrandom chromosomal translocations in childhood ALL and may help to explain the genetic alterations leading to the loss of normal growth control mechanisms in this disease.

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assays relying on monoclonal antibodies (MoAbs) to lymphoid-associated antigens. Samples were analyzed with a flow cytometer (EPICS-C; Coulter Diagnostics) or fluorescence microscopy. Blast cells were also tested for surface (sIg) and cytoplasmic (cIg) Ig. Cells were classified as T (CD7+, CD5+, CD2+), B (sIg+), or early pre-B (cIg−, sIg−, HLA-DR+, CD19+, CALLA±) according to their reactivity with the MoAb panel.

RESULTS

Of the 242 ALL cases with one or more chromosomal translocations, a third comprised well-recognized recurring abnormalities: t(1;19)(q23;p13) or der(19)t(1;19) (n = 32), t(9;22)(q34;q11) (n = 19), t(4;11)(q21;q23) (n = 8), t(11;14)(p13;q11) (n = 5), t(11;14)(p15;q11) (n = 1), dic(9;12)(p11;p12) (n = 5), t(10;14)(q24;q11) (n = 4), t(11;19)(q23;p13) (n = 4), and t(8;14)(q24;q11) (n = 3). Most translocations in the remaining cases had unique chromosomal breakpoints, but only seven were frequent enough to warrant consideration as cytogenetic subgroups.

Details of the seven previously unreported rearrangements are shown in Table 1. One of these changes was the only structural abnormality. In four subgroups, the translocations involved different chromosomes, except for a t(7;9) that was observed in three cases (Nos. 9, 14, and 18). The t(7;9) was the sole chromosomal abnormality in two cases, and t(17;19) was the sole chromosomal abnormality in two additional cases. In one case, t(7;9) was the only abnormality. The remaining two cases had additional abnormalities.

Table 1. Karyotypes of the Seven Translocation Subgroups in Childhood ALL

<table>
<thead>
<tr>
<th>Subgroup of Translocations</th>
<th>Patient No.</th>
<th>Karyotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>t(1;19)</td>
<td>1</td>
<td>45,XY,t(17;19)(p22;p13)</td>
</tr>
<tr>
<td>t(9;22)</td>
<td>2</td>
<td>46,XY,t(17;19)(q22;p13)</td>
</tr>
<tr>
<td>t(11;14)</td>
<td>3</td>
<td>46,XY,t(17;19)(p22;p13),+der(11)(t1;?)p36; ?,+der(22)(t22;7)(q13;?),dic(12;16)p11; p13,t(11;12)q22,p13,t(17;19)q22;p13</td>
</tr>
<tr>
<td>dic(7;9)</td>
<td>4</td>
<td>46,XY,t(17;19)(p22;p13),+der(11)(t1;?)p36; ?,+der(22)(t22;7)(q13;?),dic(12;16)p11; p13,t(11;12)q22,p13,t(17;19)q22;p13</td>
</tr>
<tr>
<td>dic(7;12)</td>
<td>5</td>
<td>45,XY,dic(7;9)(p13;p11),dup(1)(p373→p38)/ 45,XY,same,del(8)(p22)/ 46,XY,dup(1)p373→p38</td>
</tr>
<tr>
<td>dic(7;12)</td>
<td>6</td>
<td>45,XX,dic(7;9)(p13;p11),−20,+mar</td>
</tr>
<tr>
<td>dic(7;12)</td>
<td>7</td>
<td>45,XY,dic(7;9)(p13;p11)</td>
</tr>
<tr>
<td>dic(7;12)</td>
<td>8</td>
<td>45,XX,dic(7;9)(p13;p11)</td>
</tr>
<tr>
<td>t(1;3)</td>
<td>9</td>
<td>45,XY,dic(7;12)(p11;p12),del(6)(q14q23)/ 46,XY,dic(7;12)(p11; p12),del(6)(q14q23),+mar</td>
</tr>
<tr>
<td>t(1;3)</td>
<td>10</td>
<td>46,XY,+21,dic(7;12)(p11;p12),del(1)(q32)</td>
</tr>
<tr>
<td>t(1;3)</td>
<td>11</td>
<td>45,XY,dic(7;12)(p11;p12),46,XY,+718,dic(7; 12)(p11;p12);90XXX,del(7)(d12;72)</td>
</tr>
<tr>
<td>t(12;13)</td>
<td>12</td>
<td>47,XX,+20,dic(7;12)(p11;p12),+dic(7; 12)t(11;15)(q23;q15→q21)</td>
</tr>
<tr>
<td>t(12;13)</td>
<td>13</td>
<td>45,XX,dic(7;12)(p11;p12),del(9)(p21)</td>
</tr>
<tr>
<td>t(1;3)</td>
<td>14</td>
<td>45,XY,−X,del(6)(q13c21),(t1;3)p34;p21</td>
</tr>
<tr>
<td>t(1;3)</td>
<td>15</td>
<td>46,XY,t(1;3)p34;p21/ 46,XY,−X,+der(13)(t13;7)(q34;?),t1;3p374→p21</td>
</tr>
<tr>
<td>t(7;9)</td>
<td>16</td>
<td>47,XY,−X,del(13)(q14),(t7;9)p15;p23→p24</td>
</tr>
<tr>
<td>t(7;9)</td>
<td>17</td>
<td>45,XX,−Y,−1,del(12)(p21),del(3)(q11),i(17q), inv(12)(p11q13),t(12;14)(p11;q11),t(7;9) (p15;p23→p24),+mar(200)</td>
</tr>
<tr>
<td>t(12;13)</td>
<td>18</td>
<td>46,XY,−1,−der(11)(t1;?)p36;?,del(2)(p22), del(6)(q14q22),del(9)(q1q22),−11, +der(11)(t1;?)p34;?,del(11)(q23→ q23),t(12;13)(p13;p14)92,XXXY, same,del(8)(p21)</td>
</tr>
<tr>
<td>dic(12;17)</td>
<td>19</td>
<td>46,XY,−Y,+der(19)(t19;7)(q13;7),t1;12 (13p13;34)</td>
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<tr>
<td>dic(12;17)</td>
<td>20</td>
<td>45,XX,dic(12;17)(p11;p11),inv(5) (p13q14),del(10)(q22),del(11)(q22→q23)</td>
</tr>
<tr>
<td>dic(7;9)</td>
<td>21</td>
<td>46,XX,+10,dic(12;17)(p11;p11→ p12),dup(5)q32→q35</td>
</tr>
</tbody>
</table>

Table 2 summarizes the presenting features of patients with one of the seven new chromosomal translocations. Blast cell surface antigens were characteristic of B-lymphoid differentiation (n = 16) or T-lymphoid derivation (n = 4), or were unclassifiable (n = 1). Within the B-cell lineage, nine cases were subclassified as early pre-B (cIg−) and seven as pre-B (cIg+). Two of the four T-cell cases (Nos. 11 and 18) and one of the early pre-B cases (No. 21) also expressed a myeloid-associated antigen (CD33).

Description of Subgroups

t(17;19)(q22;p13). Four cases had identical 17;19 translocations with breakpoints in the q arm of chromosome 17 at q22 and in the p arm of chromosome 19 at p13 (Fig 1A). The t(17;19) was the sole chromosomal abnormality in two of these cases (Nos. 1 and 2) but was associated with additional abnormalities in the remaining two. In case 3 the rearrangements were highly complex. Case 4 showed a derivative (19)t(17;19) with loss of one chromosome 17 and no evidence of a der(17); an independent line containing a t(13;14)(q32;q11) as the only abnormality was also present. This case was unclassifiable by immunophenotype; it expressed CD7 and CD10 but not TdT, sIg, cIg, CD2, CD5, CD13, CD22, or CD33. Patients 1 and 2 had remarkably similar immunophenotypes (CD19+, CD10+, CD21+, cIg−) and one case (No. 2) expressed Mo-1. Both patients presented with disseminated intravascular coagulation that improved during antileukemic therapy.

dic(7;9)(p13;p11). In four cases, the leukemic blast cells
had a dicentric chromosome, dic(7;9), that contained the long arms of chromosomes 7 and 9, with partial loss of 7p and 9p (Fig 1B). In each case, the modal chromosome number was 45; in two cases, the dicentric chromosome was the only abnormality. Consistent presenting characteristics of these patients were an L1 FAB subtype, age less than 6 years old with low leukocyte counts who are currently in complete clinical remission after completing all chemotherapy.

dic(7;12)(p11;p12). Five patients had a dicentric chromosome resulting from a break and subsequent loss of the p arms of chromosomes 7 and 12 (Fig 1C). The modal number was 45 in three cases (Nos. 9, 11, and 13), 46 in one (No. 10) due to a +21, and 47 in the other (No. 12), which had a +20 and a duplication of the dicentric chromosome. One case (No. 11) had an additional abnormal line containing an exact duplication of the stemline with 45 chromosomes; thus, the dic(7;12) was duplicated while two copies each of chromosomes 7 and 12 were missing. These patients did not have similar presenting features.

t(1;3)(p34;p21). The assignment of the breakpoint in chromosome 1 in two cases with this abnormality was difficult because the definition of the 1p band was not optimal in the metaphase preparations (Fig 1D). Both patients were more than 11 years old and had an L1 FAB morphologic classification.

t(7;9)(p15;p23;p24). The two patients with this abnormality had leukocyte counts less than $5 \times 10^9/L$. At diagnosis, patient 17 was evaluated elsewhere and was initially studied cytogenetically at our institution during his first hematologic relapse. After a second relapse, this patient’s blast cell population had a nonlymphoid leukemia phenotype and morphology, although the karyotype retained the t(7;9) (Fig 1E).

t(12;13)(p13;q14). The breakpoint on chromosome 12 in these cases was clearly defined as p13; the breakpoint at 13q was more difficult to assign (Fig 1F). One of these karyotypes (in case 18) had multiple chromosomal abnormalities. Both translocations occurred in boys less than 6 years old with low leukocyte counts who are currently in remission after completing all chemotherapy.

dic(12;17)(p11;p11;p12). Two of these cases had leukemic blasts with a dicentric chromosome containing the long arms of chromosomes 12 and 17 (Fig 1G). Thus, the malignant cells were deficient for both p arms. This was the sole translocation in both cases.

DISCUSSION

The diversity of chromosomal translocations in childhood ALL is impressive, with the majority involving unique breakpoints that affect the arms of virtually every chromosome. We have identified seven new nonrandom translocations in 21 cases of ALL. Most of these abnormalities had at least one breakpoint in regions commonly affected by cytogenetic changes in this disease (9p11-p24 [n = 6], 12p11-p13 [n = 9], 19p13 [n = 4]); the others occurred within less frequently involved regions (1p34 [n = 2], 3p21 [n = 2], 7p11-p15 [n = 11], 13q14 [n = 2], 17p11-p12 [n = 2], and 17q22 [n = 4]).

Chromosome 12p12 is the region most often involved in nonrandom abnormalities in childhood ALL (10% of all cases). In our original report of 23 cases with 12p12 abnormalities, 18 were classified as “common,” three as

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**Table 2. Clinical and Laboratory Features of Patients With Specific Translocations**

<table>
<thead>
<tr>
<th>Translocation Subgroup</th>
<th>Patient Age (y)</th>
<th>Race/Sex</th>
<th>Leukocyte Count (x10^9/L)</th>
<th>Hemoglobin Level (g/dL)</th>
<th>Platelet Count (x10^9/L)</th>
<th>Liver/Spleen (cm)</th>
<th>FAB Subtype</th>
<th>Immunophenotype</th>
<th>Clinical Status</th>
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<tbody>
<tr>
<td>t(1;3)</td>
<td>16.9 W/M</td>
<td>11.8</td>
<td>9.4</td>
<td>24</td>
<td>2/5</td>
<td>L1 Early pre-B</td>
<td>Died in CCR</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>15.5 W/F</td>
<td>4.3</td>
<td>6.4</td>
<td>48</td>
<td>4/7</td>
<td>L2 Early pre-B</td>
<td>CCR on therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.2 W/M</td>
<td>16.1</td>
<td>6.7</td>
<td>15</td>
<td>1/2</td>
<td>L1 Early pre-B</td>
<td>CCR on therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>11.1 W/M</td>
<td>71.9</td>
<td>9.3</td>
<td>29</td>
<td>5/11</td>
<td>L1 Unclassified</td>
<td>Failed induction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dic(7;9)</td>
<td>5.5 W/M</td>
<td>11.1</td>
<td>9.6</td>
<td>192</td>
<td>3/0</td>
<td>L1 Pre-B</td>
<td>CCR off therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.5 W/F</td>
<td>64.0</td>
<td>8.4</td>
<td>34</td>
<td>6/8</td>
<td>L1 Pre-B</td>
<td>CCR on therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.0 W/M</td>
<td>88.6</td>
<td>11.4</td>
<td>56</td>
<td>5/3</td>
<td>L1 Pre-B</td>
<td>CCR on therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.0 W/F</td>
<td>6.0</td>
<td>4.3</td>
<td>362</td>
<td>1/1</td>
<td>L1 Early pre-B</td>
<td>CCR on therapy</td>
<td></td>
<td></td>
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<tr>
<td>dic(7;12)</td>
<td>2.3 W/M</td>
<td>30.0</td>
<td>12.6</td>
<td>23</td>
<td>7/10</td>
<td>L1 Early pre-B</td>
<td>Died</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.5 W/M</td>
<td>107.2</td>
<td>7.8</td>
<td>9</td>
<td>4/4</td>
<td>L1 Early pre-B</td>
<td>CCR off therapy</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>10.8 B/M</td>
<td>68.5</td>
<td>13.6</td>
<td>80</td>
<td>4/5</td>
<td>L2 T-cell</td>
<td>CCR off therapy</td>
<td></td>
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<tr>
<td></td>
<td>8.7 W/F</td>
<td>4.0</td>
<td>7.0</td>
<td>380</td>
<td>0/0</td>
<td>L2 Pre-B</td>
<td>CCR off therapy</td>
<td></td>
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<tr>
<td></td>
<td>13.2 W/F</td>
<td>65.2</td>
<td>9.8</td>
<td>42</td>
<td>5/3</td>
<td>L1 Pre-B</td>
<td>CCR off therapy</td>
<td></td>
<td></td>
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<tr>
<td>t(7;9)</td>
<td>11.1 W/F</td>
<td>3.0</td>
<td>11.7</td>
<td>220</td>
<td>1/1</td>
<td>L1 Early pre-B</td>
<td>CCR off therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>16.1 W/M</td>
<td>163.0</td>
<td>16.1</td>
<td>131</td>
<td>8/9</td>
<td>L1 T-cell</td>
<td>CNS relapse still on therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>17.0 W/M</td>
<td>2.0</td>
<td>5.7</td>
<td>300</td>
<td>—</td>
<td>L1 Pre-B-AML</td>
<td>Died</td>
<td></td>
<td></td>
</tr>
<tr>
<td>t(12;13)</td>
<td>14.7 W/F</td>
<td>85.7</td>
<td>12.1</td>
<td>310</td>
<td>0/0</td>
<td>L2 T-cell</td>
<td>Died</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.0 B/F</td>
<td>53.4</td>
<td>4.3</td>
<td>23</td>
<td>0/0</td>
<td>L1 Early pre-B</td>
<td>CCR off therapy</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CCR, complete clinical remission; AML, acute myeloid leukemia.

*Palpable below the costal margins.

†CD33 positive.
pre-B, and two as T-cell ALL. None of the 23 cases had the prognostically favorable feature of hyperdiploidy greater than 50 chromosomes, and the median leukocyte count (30 \times 10^9/L) was higher than that of cases lacking this abnormality. At that time, we described a t(7;12)(q11;p12); with additional cases and better definition of chromosome morphology, the breakpoint in chromosome 7 has been reassigned to 7p11, and the derivative chromosome has been reclassified as a dicentric. In this study, we also identified two additional nonrandom translocations involving chromosome 12p, a balanced t(12;13) and a dicentric 12;17. None of these nine patients with 12p abnormalities described in this report had hyperdiploidy greater than 50 chromosomes; six had B-lineage ALL (two pre-B) and three T-cell ALL; five had leukocyte counts greater than 50 \times 10^9/L; and seven remain in complete clinical remis-
sion. These findings are similar to those in other 12p12 cases.

Cases of 12;13 chromosomal translocations similar to those in our report have been described by others. Descriptions of the t(12;13) published by Keene et al. correspond to findings in the present study, except that the breakpoints are interpreted differently and their patients had eosinophilic leukemia. The 13q14 band is the site of the 

breakpoints are interpreted differently and their patients had eosinophilic leukemia. The 13q14 band is the site of the 

point. The breakpoints of the t(1;3) include a putative T-cell leukemia/lymphoma gene, TCL5 or TAL, and a stem cell leukemia gene (SCL), both located at the 13p13 region. It may be of interest that one of our cases with the t(1;3) had a T-cell immunophenotype. Also, the human HF10 finger gene was recently mapped to 3p21-p22, a region frequently altered in human cancer.

In the past, dicentrics were seldom observed in leukemic cells, but as the quality of metaphase preparations has improved, such chromosomes are being identified more often.

Dicentrics are known to have a limited life-span because of frequent bridge formation during division. It appears that the dicentrics (7;9)(p13;p11), (7;12)(p11;p12) and (12;17)(p11;p12) may remain stable through multiple cell divisions because the two centromeres are close enough to prevent the formation of anaphase bridges. During the process of dicentric formation in our cases, most of the DNA from the p arms of chromosomes 7, 9, 12, and 17 is lost.

With improved karyotyping and optimal sampling techniques, we are now able to identify chromosomal translocations in 50% of cases of ALL. Although a third of the translocations identified in this study were shown to be nonrandom, it is likely that additional studies will reveal more new clusters. The identification of recurring translocations in ALL is important because such changes implicate specific genes whose alteration may contribute to the clinical and biologic properties of the disease. As more is learned about the molecular pathology underlying these rearrangements, it may be possible to devise new therapeutic approaches that are specifically targeted to interfere with aberrant gene products expressed by the leukemic cells.

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