Reciprocal Translocation Involving the Short Arms of Chromosomes 7 and 11, t(7p−;11p+), Associated With Myeloid Leukemia With Maturation

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A reciprocal translocation involving the short arms of chromosomes 7 and 11, t(7;11)(p15;p15), was found in nine patients including eight with acute myelogenous leukemia (AML) and one with Philadelphia (Ph+) chromosome-positive chronic myelogenous leukemia (CML) in blastic crisis. Although a similar chromosome rearrangement has previously been reported in five patients, including three with AML and two with CML, the 7p breakpoint in some of these cases was slightly different from that detected in our patients. Notable cytogenetic and clinicohematologic findings in our patients and those reported in the literature were as follows: (a) t(7;11) occurred in myeloid leukemia, predominantly AML with subtype M2, and occasionally in other AML subtypes and in CML with or without Ph+ chromosome; (b) t(7;11) frequently occurred as the sole chromosome abnormality; (c) most patients showed a low neutrophil alkaline phosphatase score; and (d) Auer rods were present in leukemic cells of most cases including Ph+positive CML. Our findings suggest that a t(7;11)-associated leukemia constitutes a subgroup of myeloid malignancy involving maturing leukemic cells.

IT IS NOW WELL ESTABLISHED that specific chromosome abnormalities correlate with a particular type of leukemia showing characteristic morphological and clinical features. The best known examples are Philadelphia (Ph+) translocation, t(9;22), in chronic myelogenous leukemia (CML)1 and t(8;21), t(15;17), and inv(16) in acute myelogenous leukemia (AML) with subtypes M2, M3, and M4 with abnormal eosinophils, respectively.2,3 These specific chromosome abnormalities have a diagnostic and sometimes a prognostic significance. Recently, a reciprocal translocation involving the short arms of chromosomes 7 and 11, t(7p−;11p+), was reported in several Japanese patients with leukemia.4,5 Although a possible association of t(7;11) with Ph−negative CML has been hypothesized,6,7 the clinicohematologic findings thus far obtained from the patients with this abnormality seem to be insufficient to warrant this association.

In the present investigation, we studied nine patients with t(7;11), including eight with AML and one with Ph+positive CML, and paid particular attention to their clinicohematologic characteristics.

MATERIALS AND METHODS

Patients. Nine patients including eight with AML and one with Ph+positive CML in blastic crisis were evaluated and treated at the following Japanese institutes: Asahikawa Red Cross Hospital, Hokkaido University Hospital, Jichi Medical School Hospital, Kinki Central Hospital, Hiroshima University Hospital, Medical College of Oita, and Naha Prefectural Hospital. Informed consent for chemotherapeutic procedures and necessary studies was obtained from all the patients. Diagnosis and classification of leukemias were based on morphological and cytochemical examinations of peripheral blood smears and bone marrow aspirates on admission. The eight AML patients included seven with subtype M2 and one with M5a according to classification by the French-American-British Cooperative Group criteria.8,9 Neutrophil alkaline phosphatase (NAP) score was counted according to the method described by Tomonaga et al.10 All eight AML patients were given intensive combination chemotherapy with cytosine arabinoside (Ara-C) or N4-behenoyl-1-β-D-arabinofuranosylcytosine (BH-AC; Asahi Chemical Industry, Osaka, Japan), daunorubicin (DNR) or aclacinomycin A, Yamanouchi Pharmaceutical Co. Ltd., Tokyo), 6-mercaptopurine (6-MP), and prednisolone (PSL). In some cases, either interferon-α (INF-α) or vincristine (VCR) was added as initial induction chemotherapy. The CML patient (patient no. 9) was treated with busulfan in the chronic phase and with intensive combination chemotherapy with BH-AC, DNR, VCR, and PSL in the blastic phase.

Cytogenetic study. Chromosome analysis with Q- or G-banding techniques was performed on bone marrow and peripheral blood cells obtained from each patient at the time of diagnosis. Patient nos. 1 and 5 were examined two or three times during the clinical course. One Ph+positive CML patient (patient no. 9) was examined both in the chronic and blastic phases. Karyotypes were described according to the International System for Human Cytogenetic Nomenclature.

RESULTS

Hematologic and clinical findings. The hematologic and clinical findings in the nine patients examined at the time of diagnosis or at the onset of blastic crisis are summarized in Table 1. The ages of these patients, who included five males and four females, ranged from 16 to 59 years old (median, 40). None of the patients except for the CML...
Table 1. Clinical, Hematologic, and Cytochemical Data of the Patients With t(7;11)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>WBC (x 10^3/L)</th>
<th>Blasts (%)</th>
<th>Hb (g/dL)</th>
<th>Platelet (x 10^3/L)</th>
<th>NAP* Score</th>
<th>NCC (x 10^3/L)</th>
<th>Blasts (%)</th>
<th>Prom (%)</th>
<th>Mature Granulo (%)</th>
<th>Auer Rods</th>
<th>AS-Dch</th>
<th>α-NB</th>
<th>Therapy</th>
<th>RM (mo)</th>
<th>Survival† (mo)</th>
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<tr>
<td>1</td>
<td>36/F</td>
<td>AML(M2)</td>
<td>48.5</td>
<td>12</td>
<td>5.9</td>
<td>3.6</td>
<td>33</td>
<td>66.0</td>
<td>42.0</td>
<td>17.6</td>
<td>23.8</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td></td>
<td>A</td>
<td>31</td>
</tr>
<tr>
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<td>AML(M2)</td>
<td>69.7</td>
<td>91</td>
<td>11.7</td>
<td>5.6</td>
<td>ND</td>
<td>12.6</td>
<td>79.4</td>
<td>5.2</td>
<td>0.2</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td></td>
<td>B</td>
<td>No 6</td>
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<tr>
<td>3</td>
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<td>13</td>
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<td>2.1</td>
<td>14.5</td>
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<td>+</td>
<td>+</td>
<td>INF-a.</td>
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<td>40/F</td>
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<td>13</td>
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<td>dec</td>
<td>16.7</td>
<td>52.2</td>
<td>8.4</td>
<td>7.4</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td></td>
<td>B</td>
<td>Yes 5</td>
</tr>
<tr>
<td>5</td>
<td>42/M</td>
<td>AML(M2)</td>
<td>35.2</td>
<td>22</td>
<td>10.5</td>
<td>12.1</td>
<td>dec</td>
<td>87.0</td>
<td>40.8</td>
<td>9.0</td>
<td>32.0</td>
<td>+</td>
<td>+</td>
<td>ND</td>
<td></td>
<td>B, VCR</td>
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</tr>
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<td>6</td>
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<td>8.2</td>
<td>45</td>
<td>7.7</td>
<td>5.1</td>
<td>82</td>
<td>15.0</td>
<td>54.6</td>
<td>4.5</td>
<td>19.5</td>
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<td>+</td>
<td>-</td>
<td></td>
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<td>51/M</td>
<td>AML(M2)</td>
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<td>3</td>
<td>9.3</td>
<td>3.0</td>
<td>95</td>
<td>4.8</td>
<td>37.0</td>
<td>7.9</td>
<td>10.8</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td></td>
<td>B</td>
<td>Yes 13</td>
</tr>
<tr>
<td>8</td>
<td>28/M</td>
<td>AML(5b)</td>
<td>54.2</td>
<td>20</td>
<td>8.6</td>
<td>7.2</td>
<td>ND</td>
<td>Hyper</td>
<td>13.6</td>
<td>-</td>
<td>5.2</td>
<td>+</td>
<td>+</td>
<td>ND</td>
<td>B</td>
<td>Yes 17</td>
<td></td>
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<tr>
<td>9</td>
<td>55/F</td>
<td>Ph&quot; (+)CML-BC51.2</td>
<td>8</td>
<td>10.4</td>
<td>54.4</td>
<td>51</td>
<td>Hyper</td>
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<td>3.0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>C</td>
<td>No 56 (4)</td>
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Abbreviations: NCC, nucleated cell count; Prom, promyelocytes; Granulo, granulocytes; POX, peroxidase; AS-Dch, naphthol-AS-D-chloroacetate esterase; α-NB, α-naphthyl butyrate esterase; RM, remission; A, BH-AC, ACM, 6-MP, and PSL; ND, not done; B, BH-AC, DNR, 6-MP, and PSL; dec., deceased; Hyper, hypercellularity; C, BH-AC, DNR, VCR, and PSL; BC, blastic crisis.

*Tomonaga's method; normal value, 177 to 350.
†The number in parenthesis shows the survival time from blastic crisis.
‡In this patient, the percentage of monocytoid elements was 48.7%. See the text for the details.

Patient (patient no. 9) had a previous history of chemotherapy or mutagen exposure. Organomegaly was found in three patients, one (patient no. 6) with hepatosplenomegaly and two (patients nos. 3 and 9) with hepatomegaly.

In peripheral blood, the WBC count ranged from 2.4 to 69.7 x 10^3/L (median, 35.2 x 10^3/L), the hemoglobin level from 5.9 to 11.7 g/dL (median 9.3 g/dL), and the platelet count from 3% to 91% (median, 13%). Bone marrow aspirates showed a various number of myeloblasts, promyelocytes, and maturing granulocytic elements (Fig 1A). The proportion of myeloblasts in patient nos. 1 to 7 ranged from 37.0% to 78.4%. In patient no. 8, the proportion of myeloblasts was 13.6%, but that of monocytoid elements showing α-naphthyl butyrate esterase activity with NaF inhibition was 48.7%. In all patients, the myeloblasts exhibited peroxidase activity (Fig 1B), and in two, a fraction of myeloblasts showed naphthol-AS-D-chloroacetate esterase activity (patient nos. 3 and 9). No eosinophilia was observed in the bone marrow (below 1.0%) of all patients, although abnormal eosinophils with basophilic granules were found in two patient (patient nos. 1 and 2). Large myelocytes and metamyelocytes, and giant mature polymorphs were found in some patients (patient nos. 1 to 4). Auer rods were found in the cytoplasm of a fraction of the blasts from all patients including the patient with Ph"-positive CML (patient no. 9); they were usually single and occasionally two or three in a leukemic blast (Fig 1C), but were not in mature polymorphs of any patient. NAP score (normal value, 177 to 350) was decreased in seven of the patients, including the CML patient in blastic crisis. Patient no. 6 was diagnosed as having M2 when first admitted, but at relapse 13 months later, his leukemic blasts exhibited myelomonocytoid features. The details of this case will be published elsewhere.16

Complete remission was achieved in seven of the eight AML patients, among whom patient no. 6 was still alive in the third remission at the time of the latest follow up (March 1987). Their survival times ranged from 5 to 31 months, with a median of 12.8 months. Patient no. 9 did not achieve

![Fig 1](http://www.bloodjournal.org)
remission and died 4 months after the onset of blastic crisis; her total survival time from initial diagnosis was 56 months.

**Cytogenetic findings.** The results of the chromosome analyses of the nine patients are summarized in Table 2. The t(7;11) translocation was detected in all of the patients. The breakpoints on chromosomes 7 and 11 were common to all patients and occurred at 7p15 and 11p15, respectively (Fig 2). At the time of diagnosis, five of the eight AML patients exhibited the t(7;11) as the sole chromosome abnormality. Patient nos. 7 and 8 showed others in addition to t(7;11), the former showing 6q- and the latter showing 7q-. Karyotypically normal cells were observed in four of the eight AML patients, including patient no. 1 who exhibited complex additional abnormalities during the later clinical course. Patient no. 9 showed t(7;11) together with trisomy 8 and an extra Ph' chromosome in blastic crisis in addition to the Ph' translocation, which was the only chromosomal abnormality observed in the chronic phase.

**DISCUSSION**

Since the first description of the t(7p-;11p+) translocation in the blastic crisis of a patient with Ph'-positive CML,7 five patients with this abnormality have been reported1-2 (Table 3). A review of 14 cases including the present nine indicates that this abnormality frequently occurs in AML patients (11/14 cases), particularly those with subtype M2 (7/14) and occasionally in M4 and M5, rather than in CML patients with or without the Ph' translocation (3/14). These findings contradict the previous report that suggested an association of t(7;11) with Ph'-negative CML.11 The occurrence of t(7;11) may be analogous to that of the t(8;21) abnormality because the latter has been reported to occur predominantly in subtype M2 and occasionally in M1 and M4.17,18

Our patients commonly showed decreased NAP levels, peroxidase-positive leukemic blasts, and Auer rods in a fraction of leukemic blasts (Table 1). These features are usually observed in patients with t(8;21).18-20 Although large myelocytes and metamyelocytes, giant mature granulocytes, and abnormal eosinophils with basophilic granules were found in some of our patients, marrow eosinophilia was not observed in our patients. The presence of these abnormal bone marrow cells and marrow eosinophilia is another morphological feature of an M2 patient with t(8;21).18,20 Auer rods, occurring in about 30% of AML patients,21 are thought to be a distinctive feature of myeloid malignancies, although they are quite rare in myeloblastic crisis of CML. In this context, special attention should be paid to patient no. 9 who showed Auer rods in a small fraction of blasts and low NAP levels.

### Table 2. Cytogenetic Findings of Nine Patients With t(7;11)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Date</th>
<th>Specimen</th>
<th>Total No. of Metaphases</th>
<th>No. of Cells (%)</th>
<th>Karyotype</th>
</tr>
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<tr>
<td>1</td>
<td>May 1985</td>
<td>PB</td>
<td>5</td>
<td>4 (80.0)</td>
<td>46,XX,t(7;11)(p15;p15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 (20.0)</td>
<td>46,XX</td>
</tr>
<tr>
<td>2</td>
<td>March 1985</td>
<td>BM</td>
<td>20</td>
<td>20 (100.0)</td>
<td>46,XY,t(7;11)(p15;p15)</td>
</tr>
<tr>
<td>3</td>
<td>March 1986</td>
<td>BM</td>
<td>18</td>
<td>8 (44.4)</td>
<td>46,XX,t(7;11)(p15;p15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10 (55.6)</td>
<td>46,XX</td>
</tr>
<tr>
<td>4</td>
<td>August 1985</td>
<td>BM</td>
<td>20</td>
<td>20 (100.0)</td>
<td>46,XX,t(7;11)(p15;p15)</td>
</tr>
<tr>
<td>5</td>
<td>March 1985</td>
<td>BM</td>
<td>20</td>
<td>20 (100.0)</td>
<td>46,XY,t(7;11)(p15;p15)</td>
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<tr>
<td>6</td>
<td>April 1985</td>
<td>BM</td>
<td>7</td>
<td>7</td>
<td>46,XY,t(7;11)(p15;p15)</td>
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<tr>
<td>7</td>
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<td>46,XY,t(7;11)(p15;p15)</td>
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<td>8</td>
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<td>93</td>
<td>65 (69.8)</td>
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<td>28 (30.2)</td>
<td>46,XY</td>
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<tr>
<td>9</td>
<td>December 1985</td>
<td>BM</td>
<td>32</td>
<td>25 (78.1)</td>
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<td></td>
<td></td>
<td>7 (21.9)</td>
<td>46,XY</td>
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<tr>
<td>9</td>
<td>July 1982</td>
<td>BM</td>
<td>10</td>
<td>10 (100.0)</td>
<td>46,XX,t(9;22)(q34;q11)</td>
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<tr>
<td></td>
<td>November 1986</td>
<td>BM</td>
<td>60</td>
<td>37</td>
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<tr>
<td>(M-BC)</td>
<td></td>
<td></td>
<td></td>
<td>14</td>
<td>47,XX,t(9;22)(q34;q11),t(7;11)(p15;p15),+Ph'</td>
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<td>47,XX,t(9;22)(q34;q11),t(7;11)(p15;p15),+Ph'</td>
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<td>4</td>
<td>46,XX,t(9;22)(q34;q11),t(7;11)(p15;p15)</td>
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Abbreviations: M-BC, myeloid blastic crisis; PB, periperal blood; BM, bone marrow.
levels in the blast crisis. Moreover, over 60% of positive-peroxidase reaction in this patient's blasts was unusual because the peroxidase reaction in CML blasts is usually negative or weakly positive in a small fraction, even in the myeloid crisis. Thus, cytochemical and hematologic findings of our patients and reported cases indicate that t(7;11)-associated leukemia may involve precursors with myeloid maturation, although other morphological features that distinguish this type of leukemia from others were not observed.

In the present study, AML patients exhibited a fairly good response to chemotherapy, although the CML patient failed to enter remission, as did two other CML patients reported in the literature (Table 3). However, the remission rate in our AML patients (7/8 cases, 87.5%) as well as the overall rate obtained when those reported in the literature were included (10/11 cases, 90.9%) is not significantly higher than that of M2 patients without t(7;11) at our institutes.

As shown in Tables 2 and 3, the t(7;11) translocation mostly occurred as the sole chromosome abnormality in the patients concerned herein. In the previously reported five patients with t(7;11), the breakpoint of chromosome 11, 11p15, concurred with that observed in our nine patients, whereas somewhat variable breakpoints (p13 to p15) were observed for chromosome 7. Recently, we have corrected the breakpoint of 7p14 to 7p15 in a previously reported CML case (patient no. 14) (Tomiyasu et al, unpublished). In addition, the 7p13 breakpoint in another reported case (patient no. 12), was revised to 7p15 (Ishihara et al, unpublished). Thus, 13 of the 14 patients thus far reported show the same 7p and 11p breakpoints in the t(7;11) translocation. A complex translocation involving chromosomes 7 and 11 at these breakpoints has recently been reported as the sole abnormality in chronic phase of a Ph'-negative CML patient in whom a low NAP level and peroxidase positivity were also observed. This patient may therefore bear a variant type of the standard t(7;11) translocation.

Because the band 11p15 includes the c-Ha-ras-1 oncogene, it will be interesting to assess the involvement of this oncogene in the t(7;11) translocation. Our preliminary results from the in situ chromosome hybridization study showed a different localization for the c-Ha-ras-1 gene in the t(7;11) between one AML patient (patient no. 6) and one CML patient (patient no. 9) of the current series; the translocation of c-Ha-ras-1 was demonstrated in patient no. 6 but not in patient no. 9. This may indicate a difference in the breakpoint within 11p15 at a molecular level between these two cases, as has been shown recently for the molecular breakpoints of 22q11 in Ph' translocation between CML and acute lymphocytic leukemia cases. Further studies should be performed on more patients with t(7;11) to determine both its c-Ha-ras-1 localization and the involvement of this oncogene in the leukemic process.

Table 3. Clinical, Hematologic, and Cytogenetic Findings of the Patients With t(7;11) Reported in the Literature

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>Auer Rods</th>
<th>NAP level</th>
<th>POX</th>
<th>AS-D ch</th>
<th>α-NB</th>
<th>Karyotype</th>
<th>RM</th>
<th>Survival*</th>
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<tr>
<td>10</td>
<td>25/F</td>
<td>AML + MF</td>
<td>+</td>
<td>276</td>
<td>+</td>
<td>-</td>
<td>-</td>
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<td>Yes</td>
<td>17 mo</td>
<td>10</td>
</tr>
<tr>
<td>11</td>
<td>72/F</td>
<td>AML (M4)</td>
<td>-</td>
<td>ND</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>46,XX,t(7;11)(p14;p15)</td>
<td>Yes</td>
<td>8 mo</td>
<td>9</td>
</tr>
<tr>
<td>12</td>
<td>18/F</td>
<td>AML (M4)</td>
<td>-</td>
<td>ND</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>46,XX,t(7;11)(p13;p15)</td>
<td>Yes</td>
<td>20 mo</td>
<td>12</td>
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<td>6/F</td>
<td>Ph'(-) CML-BC</td>
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*The number in the parenthesis shows the survival time from blast crisis.
†The 7p breakpoint in these cases recently has been revised to be 7p15 (see the text).

Fig 2. Q- and R-banded partial karyotype showing reciprocal translocation involving the short arms of chromosomes 7 and 11, t(7;11)(p15;p15), from patient no. 6 (A) and a schematic diagram of the t(7;11) translocation (B).

Table 3. Clinical, Hematologic, and Cytogenetic Findings of the Patients With t(7;11) Reported in the Literature
In conclusion, the present study indicates that a t(7;11)-
associated leukemia represents a subtype of myeloid malign-
nancy showing characteristic clinicohematologic features as
described earlier. Considering that all of the 14 patients with
t(7;11) thus far reported are Japanese, its occurrence might
show an uneven geographic distribution, as has already been
found for the t(8;21) and t(15;17) translocations, both of
which showed a much higher incidence in Japan than in
other countries.6,26 Nevertheless, careful observation may
help to detect leukemia patients with t(7;11) in other coun-
tries, particularly because two non-Japanese patients with
myeloid malignancy have recently been reported to have the
t(7;11) abnormality.27

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