
**Acute Myeloid Leukemia**

**Tetraploidy or near-tetraploidy clones with double 8;21 translocation: a non-random additional anomaly of acute myeloid leukemia with t(8;21)(q22;22)**

We report on 6 patients with tetraploidy or near-tetraploidy acute myeloid leukemia (AML) with double t(8;21)(q22;q22) and review the literature on cases with the same cytogenetic abnormalities. Some common features were revealed by this analysis.

The cytogenetic abnormality of tetraploidy or near-tetraploidy is a rare finding in acute myeloid leukemia (AML). Lemez et al. divided near-tetraploidy AML into 2 categories: primary and secondary according to its origin. It is known that patients with primary near-tetraploidy AML manifest some common features: (i) near-tetraploidy karyotypes in most of the bone marrow metaphases examined at diagnosis of AML; (ii) the presence of giant myeloid blasts and dysplastic morphology in erythroid and/or megakaryocytic lineages in the bone marrow, pointing to the origin from pluripotent myeloid progenitor cells; (iii) expression of CD34 antigen; (iv) low yields of granulocyte- macrophage colony-forming units (GM-CFU) in culture; (v) a preceding preleukemic phase before the onset of the disease, and (vi) a poor prognosis. So far secondary tetraploidy or near-tetraploidy AML has been associated with multiple structural chromosomal aberrations. There have been seven cases of AML with clearly secondary tetraploidy or near-tetraploidy metaphases with duplication of t(8;21)(q22;q22) at diagnosis or during the course of leukemia reported in the literature2-6 (Table 1). We report here another six such cases.

Between January 1990 and December 2003, 216 cases of t(8;21)(q22;q22) AML were diagnosed in our institute, and all of them were AML-M2 subtype according to the FAB criteria. The patients’ ages ranged 3–65 years with a median of 28 years; 125 were male and 91 female. Seventy-three patients(33.8%) presented with coexistence of normal and abnormal karyotypes, an additional chromosome aberration occurred in 146/216 (67.6%) of the patients and 53 (24.5%) had a complex karyotype (≥3 chromosomal abnormalities). The most frequent additional abnormalities were loss of a sex chromosome (41.2%), partial deletion of the long arm of chromosome 9(23 cases, 10.6%) and less often, deletion or partial deletion of the long arm of chromosome 7 (6 cases, 2.85%). Other additional structural abnormalities included

Table 1. Clinical and genetic features of the 13 cases of tetraploidy or near-tetraploidy AML with double t(8;21)(q22;q22).

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex/age(years)</th>
<th>Karyotype</th>
<th>Survival (months)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/28</td>
<td>90,XX,Y-Y, t(8;21)(q22;q22) 7[25]/46,XY, t(8;21)(q22;q22), +der(1), t(1;?) (p36;?) 2[2]/46,XY[4]</td>
<td>32/46,XY[2]</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M/53</td>
<td>46,XY/90,XX,-Y2,-11, t(8;21)(q22;q22) 7, +mar[92.5%] 12+</td>
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</tr>
<tr>
<td>3</td>
<td>M/31</td>
<td>46,XY, t(8;21)(q22;q22)[2]/46,XY,add(7)(q31) 7, t(8;21)(q22;q22)[7]/46,XY[10] 4</td>
<td></td>
<td></td>
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<tr>
<td>4</td>
<td>F/8</td>
<td>46,XX, t(8;21)(q22;q22)[13]/46,XX, +4 t(8;21)(q22;q22) 45/92,XXYY, t(8;21)(q22;q22) 2[45]/46,XX[4]</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>F/10</td>
<td>46,XX, t(8;21)(q22;q22)[2]/46,XX, add(7)(q31) 7, t(8;21)(q22;q22) 2[72]/46,XX[7] 8</td>
<td></td>
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</tr>
<tr>
<td>6</td>
<td>M/7</td>
<td>45,XY, t(8;21)(q22;q22)[6]/90,XX, 46,XY, t(8;21)(q22;q22) 2[32]/46,XY[2] 20+</td>
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</tr>
<tr>
<td>7</td>
<td>F/61</td>
<td>45,XY, t(8;21)(q22;q22) 7[21]/90,XX-Y, t(8;21)(q22;q22) 2[7]/46,XX[12] 3</td>
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<tr>
<td>8</td>
<td>F/6</td>
<td>46,XX, t(8;21)(q22;q22) 7[13]/46,XX, t(8;21)(q22;q22) 2[2] 12</td>
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<td></td>
</tr>
<tr>
<td>9</td>
<td>M/48</td>
<td>41-44,X,Y, t(8;21)(q22;q22) 2[9]/92,XXYY, t(8;21)(q22;q22) 2[7]/46,XX[12] 9</td>
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</tr>
<tr>
<td>10</td>
<td>F/9</td>
<td>46,XX, t(8;21)(q22;q22) 2[7]/92,XXYY, t(8;21)(q22;q22) 2[8] 11</td>
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<td></td>
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<td>11</td>
<td>M/35</td>
<td>46,XX, t(8;21)(q22;q22) 2[10]/45,XY, t(8;21)(q22;q22) 2[3]/46,XX, t(8;21)(q22;q22) 2[10]/46,XX[12] 3</td>
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<td></td>
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<tr>
<td>12</td>
<td>F/11</td>
<td>46,XX, t(8;21)(q22;q22) 2[7]/46,XX[2] 4</td>
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<td>13</td>
<td>F/12</td>
<td>46,XX, t(8;21)(q22;q22) 2[7]/46,XX[2] 6</td>
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</table>
has been followed in some tetraploid or near-tetraploid AML patients with double t(8;21) (q22;q22). In these patients the initial chromosome changes were only t(8;21) cells without double t(8;21) (q22;q22); the latter emerged during the course of the disease, suggesting that the tetraploidy or near-tetraploidy clone was the consequence of a clonal evolution.

In conclusion, a tetraploidy or near-tetraploidy clone with double 8;21 translocation is a non-random additional anomaly in some cases of t(8;21)(q22;q22) AML and predicts a poor prognosis.

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Reference

Acute Lymphoblastic Leukemia

The t(12:21) is underrepresented in childhood B-lineage acute lymphoblastic leukemia in Kerala, Southern India

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