CD7 Expression Does Not Predict for Poor Outcome in Acute Myeloblastic Leukemia

To the Editor:

Expression of the CD7 antigen, a marker of normal and neoplastic T-lymphocytes,1,4 on tumour cells in patients with acute myeloblastic leukemia (AML) has recently been reported as being a poor prognostic factor for achieving complete remission (CR), maintaining this status (CCR), and overall survival.5,6 Because this is controversial,7,8 we retrospectively analyzed 112 patients with de novo AML, presenting between October 1985 and November 1993, and classified them by the French-American-British (FAB) criteria. Peripheral blood or BM blast population was additionally characterized with conventional cytochemistry and a series of monoclonal antibodies using indirect immunofluorescent microscopy. These directed against CD7 were WT1 (courtesy of Dr W.I.M. Tax, St Radboud Hospital, Nijmegen, The Netherlands) and Leu-9 (Becton Dickinson, Mountainview, CA). Here, positivity was defined as there being more than 80% blasts in the Ficoll-Hypaque (Pharmacia, Uppsala, Sweden) centrifugation and more than 20% of these expressing the antigen, or 35% when a lesser number of blasts were recovered from the interface.
Table 1. Comparison of CD7+ and CD7- AML With Regard to Clinical and Laboratory Features

<table>
<thead>
<tr>
<th></th>
<th>CD7+</th>
<th>CD7-</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>12</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>Age Range</td>
<td>16-69</td>
<td>13-85</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>40.3</td>
<td>40.4</td>
<td>NS</td>
</tr>
<tr>
<td>Gender (M:F)</td>
<td>7:5</td>
<td>48:52</td>
<td></td>
</tr>
<tr>
<td>Hepatomegaly (%)</td>
<td>42</td>
<td>30</td>
<td>NS</td>
</tr>
<tr>
<td>Splenomegaly (%)</td>
<td>17</td>
<td>19</td>
<td>NS</td>
</tr>
<tr>
<td>Lymphadenopathy (%)</td>
<td>33</td>
<td>19</td>
<td>NS</td>
</tr>
<tr>
<td>CNS involvement (%)</td>
<td>0</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>Presenting WCC (x10⁹/L)</td>
<td>44.3</td>
<td>42.4</td>
<td>NS</td>
</tr>
<tr>
<td>Auer rods (%)</td>
<td>50</td>
<td>61</td>
<td>NS</td>
</tr>
<tr>
<td>Sudan Black (%)</td>
<td>47.2</td>
<td>58.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Myeloperoxidase</td>
<td>36.8</td>
<td>56.9</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Abbreviations: WCC, white blood cell count; NS, not significant.

Induction therapy consisted of conventional doses of cytosine arabinoside, daunorubicin, and etoposide. CR was followed by two consolidation courses of the same cytotoxic drugs. Those in CCR underwent allogeneic or autologous BM transplantation in 17 who were CD7- and 2 who were CD7+.

In a series of 112 patients, 12 (10.7%) met these criteria; none expressed T-cell markers, such as CD2, attesting to an absence of contamination with lymphocytes. There was no expression of CD14.

The two groups were well matched for mean age, gender, incidence of organomegaly, central nervous system involvement, presenting white count, and the presence of Auer rods (Table 1).

Those who were CD7+ had a greater percentage of leukemic cells staining with Sudan black or myeloperoxidase using light microscopy. Of note was the significant way in which these patients differed from those who were CD7+ with respect to FAB subtypes (Table 2). The CR rate was 70% whether the antigen was or was not expressed. Overall survival had a nonsignificant trend in the CD7+ group ($P = .06$) (Fig 1). This finding is consistent with that described by others, but at variance with earlier reports. The explanation for this may lie in methodological differences such as choice of antibodies and technique of immunophenotyping, inconsistency in criteria for defining CD7 positivity within the AML population or variation in patient selection and treatment such as BM transplantation.

Only 8 of 210 patients (2 CD7+ and 6 CD7-) reported underwent BM transplantation and were censored, but this aspect of treatment is not discussed by the other investigators. Of our CD7+ group, the only survivors are two patients who received BM transplants (1 allogeneic, 1 autologous).

Our experience is consistent with the viewpoint that this antigen is not an independent poor prognostic factor for any of the variables examined in patients with AML.

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REFERENCES

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