**CONCISE REPORT**

**Human Immunodeficiency Virus Infection in Patients With Leukemia**

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Eighteen human immunodeficiency virus (HIV)-seropositive patients were found among 211 previously treated adult patients with a variety of leukemias who had been multiply transfused before April 1985. Patients known to be homosexual or intravenous drug users were excluded from this study. The spouse of one HIV-seropositive patient became HIV infected and subsequently developed the acquired immune deficiency syndrome. Patients with leukemia who were multiply transfused before the availability of screening of blood products for HIV antibody should be counseled regarding their individual risks of HIV infection and the risk to sexual contacts.

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**MATERIALS AND METHODS**

The leukemia patients tested for antibody to HIV included 182 deceased patients who were previously treated and transfused and for whom sera were available, 29 living patients who were previously treated and transfused, and 26 patients who were newly diagnosed and untransfused. Informed consent was obtained from the living patients before testing.

Sera were stored at -70°C or -20°C. The sera for the deceased patients had been submitted for the serodiagnosis of viral, parasitic, bacterial, and fungal infections. The most recent serum specimens available were selected for testing. Sera from patients known to be homosexual/bisexual or intravenous drug users were not included in this study. The leukemia represented among these patients included acute myelogenous leukemia (45%), acute lymphocytic leukemia (20%), chronic myelogenous leukemia (13%), chronic lymphocytic leukemia (4%), myelodysplastic syndromes (6%), and other or unclassified leukemias (12%). The sera were screened by enzyme-linked immunosorbent assay; all positive results were confirmed by Western blot analysis.

**RESULTS**

The results are summarized in Table 1. HIV antibody was present in sera dating from 1981 onward. The type of leukemia did not correlate with HIV infection. Males and females were equally affected. All patients who were positive received blood products before the availability of HIV antibody testing of donated blood. Of the 182 deceased patients tested, 12 were HIV-seropositive. Of the 29 living patients in complete remission who were tested, six tested positively for HIV antibody. Testing of long-term survivors was nonrandom and prompted in part by the presence of symptoms compatible with HIV infection. Therefore, the percentage of positive samples cannot be considered a true prevalence in this population. A review of the records of deceased patients testing positively did not reveal signs, symptoms, or opportunistic infections compatible with HIV infection that were not attributable to the leukemia or its treatment. Five of the six seropositive survivors had at least one of the following signs or symptoms: fever (1/6), weight loss (3/6), diarrhea (1/6), lymphadenopathy (2/6), oral candidiasis (1/6), and unexplained thrombocytopenia or pancytopenia (3/6); the sixth was asymptomatic. None of these six patients had infections or neoplasms that would meet the criteria for the definition of AIDS as defined by the Centers for Disease Control. Seven of eight sexual contacts of HIV-seropositive patients tested were seronegative. However, the widow of one of the HIV-seropositive patients has developed AIDS. She was found to be seropositive after she developed persistent unexplained fever, night sweats, and lymphopenia, and she subsequently developed *Pneumocystis carinii* pneumonia. She had no risk factor other than sexual contact with her HIV-infected spouse.

Seroconversion could be demonstrated for five of nine patients for whom earlier sera were available, thereby confirming that HIV infection occurred after transfusions, although not necessarily because of them. In the remaining nine seropositive patients, earlier sera were not available; it is...
Table 1. Presence of HIV Antibody in Patients Treated for Leukemia

<table>
<thead>
<tr>
<th>Year of Serum Sample</th>
<th>Total Number of Patients</th>
<th>Number of Patients Testing Positive</th>
<th>Percentage Testing Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>21 (11 M, 10 F)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1979</td>
<td>31 (22 M, 9 F)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1980</td>
<td>34 (22 M, 12 F)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1981</td>
<td>32 (16 M, 16 F)</td>
<td>3 (2 M, 1 F)</td>
<td>9</td>
</tr>
<tr>
<td>1982</td>
<td>20 (10 M, 10 F)</td>
<td>3 (1 M, 2 F)</td>
<td>15</td>
</tr>
<tr>
<td>1983</td>
<td>26 (13 M, 13 F)</td>
<td>4 (3 M, 1 F)</td>
<td>15</td>
</tr>
<tr>
<td>1984</td>
<td>13 (6 M, 7 F)</td>
<td>1 (1 M)</td>
<td>8</td>
</tr>
<tr>
<td>1985</td>
<td>5 (4 M, 1 F)</td>
<td>1 (1 M)</td>
<td>20</td>
</tr>
<tr>
<td>1986</td>
<td>27 (14 M, 13 F)</td>
<td>5 (5 F)</td>
<td>18</td>
</tr>
<tr>
<td>1987</td>
<td>2 (1 M, 1 F)</td>
<td>1 (1 M)</td>
<td>50</td>
</tr>
<tr>
<td>Total</td>
<td>211 (119 M, 92 F)</td>
<td>18 (9 M, 9 F)</td>
<td>9</td>
</tr>
</tbody>
</table>

All patients included in this table were transfused before screening of blood products began on April 18, 1985.

possible that they were HIV-seropositive before receiving any transfusions despite the fact that risk factors for HIV infection could not be identified. Sera from these patients at the time of the initial diagnosis were not available. Twenty-six patients with a similar spectrum of newly diagnosed leukemias who were not yet treated or transfused were also screened for HIV antibody. All were negative.

Transfusion records were available on the 17 HIV antibody-positive patients treated at our hospital; the 18th seropositive patient was transfused elsewhere. Before the dates of the sera tested, these patients received a mean of 23 units of RBCs (SD, 20 units; range, 2 to 73 units) and 110 units of platelets (SD, 101 units; range, 10 to 355 units) through a mean of 88 separate platelet donations. Transfusion records on 100 consecutive, deceased, HIV-seronegative, treated leukemic patients showed that these patients received a mean of 23 units of RBCs (SD, 18 units; range, 0 to 73 units) and 100 units of platelets (SD, 109 units; range 0 to 648 units). The transfusion histories of these two groups of patients were not significantly different: $P = .95$ and .70 for RBC units and platelet units, respectively ($t$ test).

DISCUSSION

These findings demonstrate that patients treated for leukemia and transfused before the availability of routine screening of blood products for HIV antibody are at significant risk for transfusion-acquired HIV infection. It is of interest whether the rate of HIV infection in this population is unusually high or is expected on the basis of the number of donations of blood products. Using a range of average prevalence rates during the period between 1978 and 1985 of 0.0197% to 0.11% for HIV infections in blood donors during this time, we calculated between 5.11 and 28.55 expected cases among our patients. We observed 17 cases in the leukemic patients transfused in our hospital. These calculations reflect the unavailability of data on the actual prevalence of HIV infection in blood donors during this period. In addition, our data do not represent a prevalence study and may overestimate or underestimate the true prevalence of HIV infection in multiply transfused leukemia patients for several reasons: patients were tested only if sera were available, some patients were tested because of symptoms suggesting HIV infection, later specimens if available on some seronegative patients might have tested positively for the antibody, and the immune suppression associated with leukemia and its treatment may have impaired the antibody response in some patients.

The large number of platelet transfusions, unique to this patient population, may confound comparison of this group of patients with other heavily transfused patients. There is no evidence to suggest that patients with hematologic malignancies are intrinsically more susceptible to HIV infection. Because of their larger transfusion requirements, however, they are probably at higher risk for HIV infection than are other patients. There was no evidence that a single donor was responsible for multiple cases, as in the recently described cluster of HIV infections among 25 patients with a variety of neoplasms including solid tumors who had received 1 to 2 units of blood products from the same HIV-seropositive donor.

The routine screening of blood products for evidence of HIV infection since April 1985 should markedly reduce the risk of transfusion-acquired HIV infection in patients transfused since then; however, the HIV infection of patients transfused before that time may have a major impact on their prognoses, as illustrated by those patients in remission of leukemia who were symptomatic for HIV infection. Furthermore, the sexual contacts of these patients are at risk for developing HIV infection.

Screening for HIV infection in multiply transfused leukemia patients, especially those who received transfusions before April 1985, should be initiated, with adequate counseling, because of the risks of HIV infection in these patients and their sexual contacts. Studies of the effect of HIV infection on the outcome of leukemia and other neoplasms and their treatment are needed. In addition, all persons transfused between 1978 and April 1985 who are planning to have children or who are sexually active and their sexual contacts should be advised of the risks of HIV infection in terms of time, place, and number of transfusions. Counseling and HIV antibody testing for these patients and their sexual contacts should be available to help control the spread of infection.

REFERENCES


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Human immunodeficiency virus infection in patients with leukemia

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