Identification of Alloimmunized Patients: Use of Radiolabeled Allogeneic Platelet Kinetic Measurements and Platelet Antibody Tests

By William I. Bensinger, Jean Hadlock, and Sherrill J. Slichter

In a group of stable, nonthrombocytopenic leukemia patients awaiting bone marrow transplantation, results of paired allogeneic radiolabeled platelet kinetic measurements were correlated with the results of several different platelet and lymphocytotoxic antibody tests to determine which parameters could be used to identify patients who were alloimmunized to platelets. Seven patients with acute leukemia who had been transfused during induction therapy were used as the test group, and, as a control group, five untransfused patients with chronic myelogenous leukemia were also studied. Concurrent fibrinogen survival measurements were performed in all patients to assess whether hematicostatic factor consumption (ie, disseminated intravascular coagulation) was present. Allogeneic platelet survival measurements were markedly reduced to ≤2.1 days, compared with the 3.5- to 7.4-day platelet survival measurements found in the other eight patients. The disproportionately short platelet survivals compared with fibrinogen survival measurements in these four patients, combined with documented positive antibody tests to their donors’ platelets in the three patients with evaluable tests, suggested that these patients had become alloimmunized to platelets because of their prior transfusions. There was substantial concordance between the two radiolabeled allogeneic donor platelet survival measurements performed in each of these patients, suggesting that host rather than donor factors have a major influence on transfusion outcome (r = .93, P < .001). The platelet cross-match tests, using the radiolabeled protein Staph A assay combined with the IgG enzyme-linked immunosorbent assay test, had the best correlation with the posttransfusion recovery and survival of the donors’ platelets.

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PATIENTS UNDERGOING bone marrow transplantation often require large numbers of platelet transfusions during the course of the transplant procedure. Although it might be presumed that the intensive pretransplant chemotherapy and radiation therapy given to patients both to treat their underlying malignant process and to permit engraftment of an allogeneic transplant would prevent postgrafting platelet alloimmunization, such is not the case. Bone marrow transplant patients still have a very high rate of platelet alloimmunization requiring frequent administration of HLA-selected donor apheresis transfusions to provide adequate platelet support. Therefore, it is important to determine if there are effective techniques for preventing platelet alloimmunization in transplant patients. However, as most leukemia patients undergoing marrow transplantation have been previously transfused during their induction chemotherapy, it must be determined which patients have not been previously alloimmunized and, therefore, might benefit from a program to prevent platelet alloimmunization.

As a pilot study, a variety of measurements were performed pretransplant in previously transfused or nontransfused leukemia patients to determine those factors that would most likely predict whether or not a patient was or was not already alloimmunized. To document platelet alloimmunization, two criteria must be met: (1) the patient must demonstrate very poor responses to allogeneic platelets in the absence of other nonimmunologic causes of platelet refractoriness;2 and (2) there must be evidence that the patient has antibodies against allogeneic platelets. As thrombocytopenia per se will reduce platelet survival,4 as will a variety of clinical factors and medications,1,6 clinically stable patients in the pretransplant period with relatively normal platelet counts were selected for study. The purpose of this study was to determine the relationship between allogeneic platelet kinetic and antibody measurements in the diagnosis of platelet alloimmunization. To determine if platelet consumption might be a cause of reduced allogeneic platelet survival measurements in these leukemia patients,7 concurrent radiolabeled fibrinogen survival measurements were performed along with the platelet survival measurements.

PATIENTS AND METHODOLOGY

Patient selection. Patients were selected for study from among patients with leukemia who were referred to the Fred Hutchinson Cancer Research Center (FHCRC), Seattle, WA, for allogeneic marrow transplantation from an HLA-matched sibling. Only patients with acute leukemia (AL) in remission or chronic myelogenous leukemia (CML) in chronic phase were studied. Criteria for determination of remission and chronic phase have been previously reported.89 Patients were required to have a platelet count greater than 90 × 10^9/L. Two patients with CML had palpable splenomegaly (unique patient numbers [UPNs] 3196 and 3754). Patients were clinically stable and had none of the usual causes of nonimmune platelet destruction. Specifically, they had no evidence of infection or fever, and they were generally not taking any medications. Studies were performed in five patients with CML and seven with AL (three acute myelogenous leukemia [AML], three acute lymphoblastic leukemia [ALL], and one acute progranulocytic leukemia [APL]). All of the patients with AL were
multiply transfused. One patient with CML (UPN 3196) had received a single red blood cell (RBC) transfusion 2 years before study, and the other CML patients (UPNs 3748, 3754, 3471, and 327) had never been transfused. This study was approved by the Human Subjects Review Board of the FHCRC.

Radio-labeled platelet and fibrinogen kinetic measurements. Platelet survival measurements were performed using platelets from two single donors randomly chosen from a group of seven normal volunteers. Patients received paired 51Cr and 111In-labeled platelets from two of the seven volunteers. Fresh donor platelets were harvested from 500 mL of whole blood when 51Cr was used as the label, or from 100 mL whole blood when labeling with 111In. Isotope labeling of platelets was performed according to established methods.20,21 Fibrinogen was prepared from fresh plasma obtained from a known hepatitis and human immunodeficiency virus (HIV)-negative donor and labeled with 111In. Labeled platelet and fibrinogen radioactivities were determined by centrifugation of whole blood samples to separate platelets and plasma, respectively.

Blood samples were drawn at 1 and 2 hours postinfusion on day 1, twice daily on days 2 and 3, and once on days 4 and 5. γ Counting was performed for measurement of indium activity, and the platelet buttons were then set aside for 4 weeks to allow for sufficient decay of the 111In to enable counting of 51Cr. Survival calculations were made using linear regression models.2 After extrapolating the survival curve to time zero (T0), platelet recovery was determined as follows:

\[
\text{Recovery (\%)} = \left( \frac{T_r \times \text{Radioactivity}}{\text{Platelet Radioactivity Injected}} \right) \times 100
\]

Total platelet viability was calculated as the area under the survival curve; ie, \( \frac{1}{2} \) recovery \( \times \) survival. To compare the viability of allogeneic platelets to the expected normal autologous platelet viability, a Viability Index (VI) was determined, ie:

\[
\text{VI} = \frac{\text{Allogeneic Platelet Viability}}{\text{Expected Autologous Platelet Viability}}
\]

For expected normal autologous platelet viability, a recovery value of 65% and a survival of 9.6 days were used. In aplastic thrombocytopenic patients and in normal volunteers, the recovery and survival of allogeneic platelets is the same as in autologous patients.20,21

Platelet and lymphocytotoxic antibody measurements. Platelet and lymphocytotoxic cross-match tests were performed using patient sera incubated with platelets and lymphocytes harvested from the same volunteers who were used for each patient’s platelet survival studies. Tests included a quantitative radioimmunoassay (RIA) for platelet-bound IgG22; three semi-quantitative tests for platelet-bound IgG using 125I-labeled Staph protein A (SPA),23 enzyme-linked anti-IgG (E-IgG),23 or fluorescein-linked anti-IgG (FACS)23; two release assays using H-serotonin and 4C-adenine (RELEASE);23 and a lymphocytic crossmatch test (LCT-AB).24 The SPA, E-IgG, and RELEASE tests were considered to be positive at a level of 1.3 times the control value (normal plasma tested with the same platelet source). This value was \( \geq 2 \) SD from the control value. The RIA mean value for 33 normal controls was 0.36 ± 0.14 fg IgG/platelet. A positive value was \( \geq 2 \) SD from the mean. FACs was considered to be positive when test platelets had a mean fluorescence 20% above control values. The lymphocytotoxic test was positive when greater than 20% of donor lymphocytes were killed.

Statistical methods. Statistical differences were determined by means of a Student’s t-test for paired or unpaired data as appropriate.

RESULTS

Twenty-three platelet survival studies were performed in the 12 patients. Eleven patients had simultaneous, paired survival studies using platelets from two separate donors, and one patient had a single donor study performed. Demographic data, ABO and HLA types of donors and recipients, and platelet and fibrinogen kinetic measurements for study patients are given in Table 1.

Results of the platelet and fibrinogen survivals performed in the 12 patients are plotted in Fig 1. Comparable reductions in platelet and fibrinogen survival measurements are diagnostic of disseminated intravascular coagulation (DIC), while disparate results indicate either another or an additional factor that modifies the survival of one of these hemostatic components. By comparison with the historical results of autologous platelet survivals performed in normal volunteers, it is apparent that all of the patients had shortened allogeneic platelet survivals. Furthermore, only 2 of the 12 patients had fibrinogen survivals within the normal range. There were four patients whose platelet survivals were markedly shortened when compared with their corresponding fibrinogen survivals, suggesting platelet alloimmunization. The other eight patients had a more normal relationship between platelet and fibrinogen survivals. All four patients with extremely short allogeneic platelet survivals (UPNs 3252, 3781, 3598, and 3749) had AL and had been heavily transfused with more than 20 U of RBCs and platelets during their induction therapy.

Figure 2 plots the 111In-labeled platelet survival from one donor against the 51Cr-labeled platelet survival from the second donor in the 11 patients who had paired survivals. There is a remarkably good correlation between the survivals of platelets obtained from each pair of donors transfused to a given patient (correlation coefficient = .93, \( P < .001 \)). However, no such correlation was found between the paired platelet recovery measurements (Table 1).

When donor-recipient pairs were analyzed on the basis of ABO compatibility, a relationship between ABO compatibility and platelet recovery was found. There were eight blood group A donor transfusions given to seven group O recipients, seven incompatible combinations other than group A to group O, and eight ABO-compatible donor-recipient pairs (Table 2). The eight group A to group O transfusions had a significantly lower 1-hour platelet recovery compared with the 15 other ABO combinations (\( P < .05 \)). However, the donor platelet survivals were not significantly different. Furthermore, in the RIA platelet test, group A platelets incubated in group O recipient plasma bound significantly more antiplatelet IgG than any other combination of donor-recipient blood groups (\( P < .001 \)).

In six of the group O recipients, the results of group A compared with group O or group B donor transfusions were available for direct comparison. Table 3 compares antibody bound to donor platelets in the RIA cross-match assay and donor platelet survival and recovery measurements in these
Table 1. Patient and Donor Data

<table>
<thead>
<tr>
<th>UPN</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>ABO Type</th>
<th>Anti-A Titer*</th>
<th>HLA Type</th>
<th>Platelet Count (x10^3/µL)</th>
<th>Fibrinogen Survival (d)</th>
<th>ABO Type</th>
<th>HLA Type</th>
<th>Platelet Recovery (%)</th>
<th>Platelet Survival (d)</th>
<th>Vi</th>
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</thead>
<tbody>
<tr>
<td>3252</td>
<td>F</td>
<td>AML</td>
<td>0/256</td>
<td>A1,30/B37,51</td>
<td>209</td>
<td>3.1</td>
<td></td>
<td>A</td>
<td>2/88,65</td>
<td>38</td>
<td>0.1</td>
<td>0.01</td>
</tr>
<tr>
<td>3781</td>
<td>F</td>
<td>ALL</td>
<td>0/128,000</td>
<td>A3/B14,39</td>
<td>225</td>
<td>3.9</td>
<td></td>
<td>A</td>
<td>2/87,57</td>
<td>18</td>
<td>0.1</td>
<td>0.00</td>
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<tr>
<td>3598</td>
<td>F</td>
<td>ALL</td>
<td>B</td>
<td>A2,11/B7,44</td>
<td>90</td>
<td>5.0</td>
<td></td>
<td>O</td>
<td>2/87,65</td>
<td>80</td>
<td>0.3</td>
<td>0.04</td>
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<td>4.2</td>
<td></td>
<td>O</td>
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<td>250</td>
<td>4.3</td>
<td></td>
<td>A</td>
<td>2/87,57</td>
<td>25</td>
<td>1.4</td>
<td>0.06</td>
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<tr>
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<td>M</td>
<td>AML</td>
<td>0/256</td>
<td>A1,28/B8,35</td>
<td>96</td>
<td>4.1</td>
<td></td>
<td>B</td>
<td>2/88,65</td>
<td>66</td>
<td>3.9</td>
<td>0.42</td>
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<tr>
<td>3740</td>
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<td>0/128</td>
<td>A2,24/B7</td>
<td>315</td>
<td>2.9</td>
<td></td>
<td>O</td>
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<td>0.63</td>
</tr>
<tr>
<td>3754</td>
<td>M</td>
<td>CML</td>
<td>A</td>
<td>A2,31/B18</td>
<td>475</td>
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<td></td>
<td>A</td>
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<td>3471</td>
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<td>CML</td>
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<td>A1,2/B7,8</td>
<td>303</td>
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<td></td>
<td>O</td>
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<td>3.5</td>
<td>0.11</td>
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<tr>
<td>3196</td>
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<td>CML</td>
<td>0/256</td>
<td>A11,24/B7,57</td>
<td>900</td>
<td>3.5</td>
<td></td>
<td>O</td>
<td>2/811,25</td>
<td>47</td>
<td>5.1</td>
<td>0.39</td>
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<tr>
<td>3237</td>
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<td>A</td>
<td>A2/B44,62</td>
<td>380</td>
<td>4.0</td>
<td></td>
<td>A</td>
<td>2/88,65</td>
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<td>5.1</td>
<td>0.58</td>
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<tr>
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<td>APL</td>
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<td>A2,3/B35,65</td>
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<td>3.6</td>
<td></td>
<td>A</td>
<td>2/88,65</td>
<td>61</td>
<td>5.4</td>
<td>0.54</td>
</tr>
</tbody>
</table>

*By antiglobulin technique.

Fig 1. Platelet and fibrinogen kinetic measurements. Allogeneic platelet survival measurements are compared to simultaneously performed fibrinogen survivals in the 12 patients. Eight survival measurements performed in four patients (dotted oval) showed markedly reduced platelet survival compared with fibrinogen survival measurements. The one-to-one relationship between platelet and fibrinogen survival ±1 SD is shown in the hatched box. (C), Patients with CML; (O), patients with AL.

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PLATELET KINETICS IN STABLE LEUKEMIA PATIENTS

with fibrinogen survivals (in the hatched area from Fig 1) all had 0.08 hours, there was a very significant correlation between the paired survivals, correlation coefficient \( r = 0.93, \) \( P < 0.001 \). The four patients with disproportionately reduced platelet survivals compared with fibrinogen survivals (in the hatched area from Fig 1) all had platelet survivals of less than 2 days. The regression line for the relationship between platelet survivals is given by the formula: 

\[ y = 0.08 + 0.98x. \]

when all transfusions were considered (data not shown). When compared with the other cross-match tests, the E-IgG and SPA were clearly superior.

UPN 3252 had a positive lymphocytotoxic cross-match with both donors; UPN 3471 was not tested for lymphocytotoxicity; and all other patients had a negative lymphocytotoxic cross-match test with their transfused donors.

**DISCUSSION**

Despite selecting clinically stable, nonthrombocytopenic patients for study, none of the patients demonstrated a normal survival of transfused allogeneic platelets; this was true even for the four CML patients who had never been previously transfused or pregnant. Furthermore, 10 of 12 patients had evidence of decreased fibrinogen survivals. The reasons for the increased turnover of platelets and fibrinogen are not clear, but they are most likely related to the effects of the patients' underlying leukemia. Shortened fibrinogen and autologous platelet survival measurements in patients with AML and CML have been previously described.

The striking concordance of paired allogeneic platelet survival measurements in each patient was an unexpected finding and suggests that recipient characteristics may be more important than donor characteristics in determining platelet survival in these patients. This observation has not been previously made primarily because platelet survival studies using paired allogeneic platelet transfusions have not been done. Indeed, the concordance of the allogeneic platelet survival measurements in individual patients is in marked contrast to the highly variable posttransfusion platelet responses reported by others.

However, prior studies have relied only on early posttransfusion corrected count increments measured at 10 minutes, 1 hour, or 24 hours to evaluate transfusion responses. Such data points correspond most closely to our platelet recovery determinations, which were also extremely variable in individual patients. As platelet recovery measurements did not correlate with platelet survival measurements, it is apparent that neither platelet recovery nor early corrected count increments can accurately predict the subsequent survival of allogeneic platelets. Therefore, both platelet recovery and survival measurements in thrombocytopenic patients, by either radiolabeling techniques or by serial platelet counts at a minimum of 1 and every 24 hours until the next transfusion is given, are required to fully determine the value of a specific transfusion.

Most of the observed variability in platelet recoveries was related to donor-recipient ABO incompatibility. This is not a surprising finding as ABO antigens are expressed on platelets and anti-A and anti-B antibodies are naturally occurring.

Group A donors given to group O recipients demonstrated reduced platelet recoveries and increased platelet-associated IgG in the platelet cross-match tests. In contrast, the subsequent survival of group A platelets given to group O recipients was not significantly different from other ABO combinations. Most other studies investigating ABO incompatibility in patients have similarly shown reduced platelet recoveries but not survivals, suggesting that only a portion of the transfused incompatible platelets
Table 4. Donor Platelet VI Correlates With E-IgG and SPA Platelet Cross-Match Tests

<table>
<thead>
<tr>
<th>Donor Platelet Kinetics</th>
<th>Platelet Cross-Match</th>
<th>Donor Patient Kinetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>UPN Recovery (%)</td>
<td>Survival (d) VI</td>
<td>E-IgG SPA (%)</td>
</tr>
<tr>
<td>3252</td>
<td>38†</td>
<td>0.1</td>
</tr>
<tr>
<td>3781</td>
<td>18†</td>
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</tr>
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<td>16†</td>
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<tr>
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<td>3754</td>
<td>20</td>
<td>3.5</td>
</tr>
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<td>3196</td>
<td>ND†</td>
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<tr>
<td>3237</td>
<td>20</td>
<td>7.4</td>
</tr>
<tr>
<td>3527</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mean ± 1 SD
29 ± 13 2.9 ± 2.4 0.09 ± 0.07 56 ± 21 4.5 ± 1.7 0.39 ± 0.22

Abbreviations: ND, not done; Neg, negative cross-match result.

*If either the E-IgG or SPA cross-match test results were positive (ie, > 1.3 × control), the cross-match was considered positive.
†Blood group A donor transfusion into a blood group O recipient.

are immediately destroyed.22–25 This may indicate either variable expression of ABO antigens on platelets,26 variable adsorption of group A substance by group A donors' platelets, or variable elution of group A substance after a group A donor transfusion is given to a non-group A recipient.

As previously stated, the value of a platelet transfusion to a patient is a combination of recovery and survival. Thus, calculation of a platelet VI (the area under the platelet survival curve in the patient compared with expected normal values) can be used as a representative measure of an "incompatible" versus a "compatible" transfusion. Of six different platelet cross-match tests, only the E-IgG and SPA were able to reliably predict transfusion outcome; ie, a VI of ≤ 20% was selected as an "incompatible" transfusion, and greater than 20% as "compatible." Although a "compatible" transfusion that gives a result of only 20% of normal may appear to be a poor transfusion outcome, this result represents at least 30% of the highest allogeneic platelet VI in the study; ie, VI of 0.63 or 63% of normal. Thus, in the context of patients who receive allogeneic platelet transfusions, this criterion represented a good transfusion result.

In view of the uniformly reduced allogeneic platelet survival measurements found in these patients, it clearly will be important in future studies to correlate allogeneic platelet viability measurements to concurrent autologous platelet viability measurements. Only by comparing the allogeneic VI to the autologous VI can the effects of disease-related platelet consumption that would be reflected by the autologous platelet survival measurement be separated from other causes of platelet destruction that would be unique to the allogeneic platelets.

It is not surprising that individual patients had varying patterns of cross-match-positive and -negative results with their random donors' platelets. However, even some of the

Table 5. Correlation of Platelet Cross-Match Tests With Successful Versus Unsuccessful Allogeneic Platelet Transfusion Results

<table>
<thead>
<tr>
<th>LCT AB Release</th>
<th>FACS RIA</th>
<th>E-IgG SPA</th>
<th>Combined E-IgG and SPA</th>
</tr>
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<tbody>
<tr>
<td>Sensitivity*</td>
<td>2/2+11</td>
<td>4/4+9 (31%)</td>
<td>4/4+10 (29%)</td>
</tr>
<tr>
<td>Specificity†</td>
<td>6/6+0</td>
<td>5/5+1 (83%)</td>
<td>8/8+0 (100%)</td>
</tr>
<tr>
<td>Predictability‡</td>
<td>2/2+6</td>
<td>4/4 (43%)</td>
<td>4/8 (62%)</td>
</tr>
</tbody>
</table>

Platelet VI of > 0.2 was used as the criterion for a successful transfusion result and < 0.2 as an unsuccessful transfusion.

*Sensitivity = true positives/(true positives + false negatives).
†Specificity = true negatives/(true negatives + false positives).
‡Predictability = (true positives + true negatives)/all test results.
male, nontransfused patients with CML had evidence of antibodies to random donor platelets that adversely affected the transfusion results. In three of these patients (UPNs 3748, 3754, and 3196), the antibody-positive transfusion was from an ABO-incompatible donor. However, in one case (UPN 3237) an antibody-related poor transfusion result occurred even with an ABO-compatible transfusion. Thus, some of these antibodies may represent disease-related “autoantibodies.” We have previously identified nontransfused CML patients who were refractory to their first transfusion of pooled random donor platelets. Although some of these patients had an enlarged spleen, as did two of our patients, the present data indicate that at least one of the two patients (UPN 3196) had a normal recovery of transfused platelets from one of his two donors, suggesting that hypersplenism cannot account for every poor platelet recovery measurement in CML patients.

Concerning the primary question to be addressed by these studies, can allogeneic platelet kinetics contribute to the determination of alloimmunization and, if so, in conjunction with which antibody tests, the following conclusions can be drawn: (1) In clinically stable, nonthrombocytopenic patients, allogeneic platelet survivals of less than 2.1 days reliably indicate alloimmunization rather than consumptive platelet destruction. (2) The majority of leukemia patients, even those with chronic disease or those in remission, have evidence of some degree of platelet consumption (reduced fibrinogen survivals in 10 of 12 patients) producing homologous platelet survivals that ranged between 3.5 and 7.4 days. Normal platelet survivals are 9.6 ± 0.4 days. (3) Even in the patients with alloimmune destruction of allogeneic platelets, there is a striking concordance between the survival of transfused platelets from two randomly selected allogeneic donors. This suggests either a similar effect of the antibody on all donor platelets or other host factors that significantly influence allogeneic survivals. (4) Platelet cross-match tests (SPA and E-IgG) can usually detect anti-A antibodies in group O recipients that will markedly reduce the recovery but not the survival of transfused group A donor platelets (six of seven donor-recipient combinations). (5) Finally, the platelet cross-match tests (SPA and E-IgG) can reliably predict substantial decreases in posttransfusion platelet recovery measurements (five of six recipients with posttransfusion platelet recovery values of ≤21% were antibody-positive), or a combination of these two parameters (9 of 11 recipients with VI of ≤20% were antibody-positive).

Future studies should correlate autologous versus allogeneic platelet survival measurements in conjunction with platelet autoantibody and alloantibody studies in an attempt to further clarify antibody/platelet kinetic measurements in leukemia patients. In addition, ABO-compatible allogeneic platelets should be used as the test transfusion to avoid effects of ABO-incompatibility on platelet increments.

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