Evidence of Hepatitis in Patients Receiving Transfusions of Blood Components Containing Antibody to Hepatitis C


When hepatitis C virus antibody (anti-HCV) enzyme immunoassay (EIA1) testing became available in 1990, we tested samples from previously transfused blood units, traced the recipients of reactive units, and evaluated the recipients for HCV infection during the 12 months after transfusion. Ten of 42 recipients of EIA1-reactive blood were anti-HCV reactive on follow-up by EIA1 and 12 were reactive by a second-generation assay (EIA2). Reverse transcriptase-polymerase chain reaction (RT-PCR) detected HCV RNA in 5 seronegative recipients. In all, 17 of 42 recipients (40%) of EIA1-reactive blood had evidence of HCV infection. In comparison, 54 surgery patients, who received either no transfusion or autologous EIA1-nonreactive blood, remained EIA1 nonreactive and RT-PCR negative for 1 year; 1 patient (1.8%) became EIA2 reactive \( IP \leq .01 \). Of the recipients of anti-HVC reactive blood transfusions (reactive by both EIA1 and a supplemental 4-antigen strip immunoblot assay [RIBA2]), 14 (93%) of the recipients had evidence of HCV infection compared with only 3 of 27 recipients (11%) of EIA1-reactive, RIBA2-nonreactive blood \( (P \leq .01) \). Thus, blood components reactive for anti-HCV EIA1 may have transmitted HCV up to 40% of the time, but blood components found reactive by both EIA1 and RIBA2 may transmit HCV with a frequency of greater than 90%.

© 1993 by The American Society of Hematology.
RECIPIENTS OF BLOOD CONTAINING HCV ANTIBODIES

meeting the eligibility criteria were enrolled. The enrollment visit and follow-up testing of controls were the same as for the recipients of anti-HCV EIA1-reactive blood.

To permit adequate time for seroconversion, recipients were observed for 12 months after transfusion whenever possible. Transfusion recipients who were unavailable for testing 12 months after the transfusion and who did not already have a reactive test for anti-HCV before 12 months of follow-up were not eligible to be evaluated for antibody production.

The study was approved by the Institutional Review Board of the Sacramento Medical Foundation.

**Assays.** Anti-HCV was detected by EIA1 (Ortho Diagnostic Systems Inc, Raritan, NJ), an enzyme-linked immunosorbent assay, which uses a microtiter well coated with a recombinant HCV-derived nonstructural antigen, c100-3. Samples found to be initially reactive were tested again in duplicate; those with at least two reactive results (sample to cut off ratio of >1) were considered to be repeatedly reactive by EIA1. At a later date, samples were retested in a similar fashion using a second generation anti-HCV EIA (EIA2; Ortho Diagnostic Systems Inc). Besides the c100-3 antigen, EIA2 includes the additional HCV antigen c33-c, as part of a larger c200 antigen, and the core antigen, c22-3.

Samples found to be repeatedly reactive by either EIA1 or EIA2 were further tested with a strip immunoblot assay (RIBA2) (RIBA; Chiron Corp, Emeryville, CA). RIBA2 contains four separate HCV recombinant antigen bands: c100-3, a nonstructural protein; 5-1-1, a component of the c100 antigen; another nonstructural protein, c33c; and a core protein, c22-3. Because each HCV recombinant antigen is fused to superoxide dismutase (SOD), a separate control band of SOD is also included on each RIBA strip. Two gamma globulin standards representing 1 and 3 + "controls" are also present on each RIBA strip for comparison. A reactive RIBA2 test is a response of at least 1 + (scale of 1 + to 4+) against at least two HCV antigens and not against the SOD band. An indeterminate test result is either a response of ≥1 + to only one HCV antigen band in a pattern that does not meet the criteria for reactive or a response to the SOD band that renders the other responses questionable. A nonreactive test result is no reactivity of ≥1 + to any HCV antigen band.

RT-PCR to detect HCV RNA was performed under code on serum samples from transfusion recipients enrolled in the study along with several known positive and negative controls. When RT-PCR results were indeterminate or were discrepant with antibody results, RT-PCR testing was repeated with fresh aliquots of the samples again submitted under code. The RT-PCR assay used the RNAzol B extraction method, previously described primers, and contamination avoidance procedures. RT-PCR products were detected by liquid hybridization using a 32P-labeled probe. Specimens were run in duplicate through the reverse transcription, amplification, and detection steps. If bands were detected in the negative or water control lanes, the results were considered invalid and the test was repeated. Results were reported as HCV RNA detected (positive), RNA not detected (negative), or indeterminate. Specimens were restested in duplicate if replicate results did not match; final results were reported as indeterminate if they could not be resolved as positive or negative by retesting. Indeterminate RT-PCR results may represent low copy number of HCV or contamination.

ALT was measured by a kinetic enzyme spectrophotometric method (Pointe Scientific Inc, Lincoln Park, MI). The expected ALT range is 0 to 53 IU/L (53 IU/L is equal to two standard deviations above the log mean normal). Anti-HBc was measured on donor samples according to the manufacturer's directions (Corzyme; Abbott Laboratories, North Chicago, IL).

**Results.** Prevalence of reactive tests in the donor population are expressed as percents. Chi square or Fisher's exact test was used to evaluate prevalence of reactive tests in recipient and control groups; P < .05 was considered to be statistically significant.

**RESULTS**

**Donor blood anti-HCV test results.** Retrospective testing of frozen serum samples drawn at the same time as 20,446 homologous blood donations (transfused during the 6 months before the May 1990 licensure of the anti-HCV EIA1 test) detected 126 transfused units (0.6%) that were reactive for anti-HCV by EIA1. Testing of 8,921 refrigerated homologous donor serum samples, from units in inventory in May 1990, found 35 EIA1 reactive units (0.4%). Tracing of the total 161 EIA1-reactive homologous blood units found that they had yielded 214 blood components transfused at local hospitals just before the licensure of the EIA1 test. Testing of frozen samples from 699 autologous units donated in the same 6-month time period showed that 10 were EIA1 reactive (1.4%).

**Enrollment.** We attempted to contact all the recipients of the 214 anti-HCV EIA1-reactive homologous blood components. When they were located an average of 7 months after transfusion, 97 of 214 (45%) had expired, 7 (3%) had moved away, 41 (19%) declined to join the study, 24 (12%) never responded, 3 (1.4%) were mentally incompetent to give consent, and 42 (19.6%) were enrolled in the study (Table 1).

In contrast, of the first 331 anti-HCV EIA1-nonreactive autologous donors/patients eligible to be controls, 1 (0.3%) had expired, 10 (3%) had moved away, 86 (26%) declined participation, 171 (52%) never responded, 9 (2.7%) had also received homologous blood, and 54 (16%) were enrolled in the study. Of the 54 controls enrolled, 33 had received autologous blood only and 21 received no blood at all during hospitalization for surgery.

**Recipients of anti-HCV EIA1 blood components.** Follow-up testing, during the 1 year after the transfusion, was performed on serum samples from 42 recipients of 46 anti-HCV EIA1-reactive, homologous blood components. Reasons for transfusions were surgery or trauma (14), gastrointestinal bleeding (7), leukemia or cancer (13), and other hematologic problems (8). Anti-HCV status before the study was not known. Nine of 42 recipients (21%) did not complete the 1-year follow-up (6 died of underlying disease and 3 declined or were unable to continue participation). Several additional recipients were only available for testing.

<table>
<thead>
<tr>
<th>Study Patients</th>
<th>Subjects*</th>
<th>Controls†</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. enrolled</td>
<td>42</td>
<td>54</td>
</tr>
<tr>
<td>Median age (yr)</td>
<td>62.6</td>
<td>53.2</td>
</tr>
<tr>
<td>No. of women (%)</td>
<td>17 (40)</td>
<td>35 (65)</td>
</tr>
<tr>
<td>Transfused prior to study hospitalization/transfusion (%)</td>
<td>15 (36)</td>
<td>8 (15)</td>
</tr>
</tbody>
</table>

* Received anti-HCV EIA1 reactive blood.
† Received autologous anti-HCV EIA1-nonreactive blood or no blood.

From www.bloodjournal.org by guest on December 24, 2017. For personal use only.
at 9 or 12 months after transfusion. The results of the follow-up testing of the 42 recipients of the anti-HCV EIA1-reactive units are shown in Table 2. In all, 17 of 42 recipients (40%) of anti-HCV EIA1-reactive units had some marker of HCV infection. In contrast, 33 autologous donors/patients received their own EIA1-nonreactive blood at surgery and all remained nonreactive by both EIA1 and EIA2 and had ALT levels less than 53 IU/L at 6 and 12 months after surgery (P < .05).

Recipients of anti-HCV EIA2-reactive units. When the second-generation anti-HCV test, EIA2, became available, the donor unit samples in the study were retested. Only 22 of the 46 (48%) EIA1-reactive homologous blood components transfused in the study were also EIA2-reactive. In all, there were 20 recipients of 22 units reactive by both anti-HCV EIA1 and anti-HCV EIA2 (Table 2). After receiving these units, 11 of the 17 recipients (65%), observed long enough after transfusion to be evaluated, were found to be anti-HCV EIA2-reactive. In 2 of these recipients, use of the second-generation test detected anti-HCV seroconversion earlier than did EIA1. Of the 20 recipients of EIA2-reactive units, 13 (65%) were positive for HCV RNA (Table 2).

Recipients of RIBA2-nonreactive units. Blood donations reactive for anti-HCV EIA1 or EIA2 were further evaluated by the RIBA2 assay when it became available. Follow-up of transfusion recipients of EIA1-reactive, but RIBA2-nonreactive blood components, showed that only 3 of 27 had markers of HCV infection (Table 2). Those 3 may have been infected with HCV at some time before the transfusions involved in this study. One recipient was anti-HCV EIA1-reactive, EIA2-reactive, and RIBA2-reactive, but had no detectable HCV RNA by RT-PCR. He had a 9-year history of chronic hepatitis B infection with continued presence of hepatitis B surface antigen (HBsAg). In this study, he had received 1 unit of EIA1-reactive, RIBA2-nonreactive, RT-PCR-negative, fresh frozen plasma; the recipient of the packed red blood cells from this same unit remained anti-HCV- and RT-PCR-negative during the 12-month follow-up period. Two other recipients of EIA1-reactive, RIBA2-nonreactive, RT-PCR-negative components did not have antibody detectable by either EIA1 or EIA2, but HCV RNA was detected by RT-PCR. Follow-up was only 6 months for one recipient, but the other completed the 12-month follow-up period. These two patients were immunocompromised and had received, respectively, 121 and 38 blood components (untested for anti-HCV) before this study.

Recipients of RIBA2-reactive units. RIBA2 reactivity of the donor blood transfused correlated well with the presence of markers of HCV infection (anti-HCV by EIA1 or EIA2, elevated ALT, and HCV RNA) in the recipients (Table 2). Acute posttransfusion hepatitis (as defined by an ALT level of at least twice the upper limit of normal) was seen in 3 of 6 patients who were tested 6 to 12 weeks after receiving a blood component reactive by EIA1, EIA2, and RIBA2 (patients no. 2, 4, and 5 in Table 3). ALT levels were 465, 836, and 106 IU/L, respectively, and jaundice, fatigue, and loss of appetite occurred in patients no. 2 and 4. Patient no. 5 was asymptomatic.

Fourteen of 15 recipients (93%) of RIBA2-reactive units had some marker of HCV infection (Table 3). In contrast, only 3 of 27 recipients (11%) of EIA1-reactive, RIBA2-nonreactive blood were judged to be HCV infected by these measurements (P < .01).

RT-PCR for HCV RNA. Serum samples obtained at enrollment (the earliest serum sample available after transfusion) from 40 of the recipients of EIA1-reactive units were evaluated for HCV RNA by RT-PCR. HCV RNA was detected in 14 of 40 recipients' samples (35%). Thirteen of these 14 had at least one elevated ALT level during the 12 months of follow-up (Table 2). Eight of 14 RT-PCR-positive recipients were anti-HCV reactive by both EIA1 and EIA2; an additional individual was anti-HCV reactive only by EIA2. Five RT-PCR-positive recipients did not have any antibody detected by EIA1 or EIA2 at 6 months and 3 of these were able to be retested at 12 months. All 5 recipients were immunosuppressed and undergoing treatment for cancer, leukemia, or acquired immunodeficiency syndrome (AIDS). Three recipients had received RIBA2-reactive units; two recipients had received RIBA2-nonreactive units.

<table>
<thead>
<tr>
<th>Transfused Blood</th>
<th>Anti-HCV Results</th>
<th>Transfusion Recipients' Test Results*</th>
<th>No. of Recipients</th>
<th>Anti-HCV</th>
<th>HCV RNA</th>
<th>ALT &gt;53 IU/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EIA1</td>
<td>EIA2</td>
<td>RIBA2</td>
<td></td>
<td>EIA1</td>
<td>EIA2</td>
</tr>
<tr>
<td>R</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>22</td>
<td>1/18</td>
<td>1/17</td>
</tr>
<tr>
<td>R</td>
<td>R</td>
<td>R</td>
<td>N</td>
<td>5</td>
<td>0/3</td>
<td>0/3</td>
</tr>
<tr>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>15</td>
<td>9/13</td>
<td>11/14</td>
</tr>
<tr>
<td>N</td>
<td>N</td>
<td>N</td>
<td>–</td>
<td>33t</td>
<td>0/33</td>
<td>1/33</td>
</tr>
<tr>
<td>NBT</td>
<td>NBT</td>
<td>NBT</td>
<td>NBT</td>
<td>215</td>
<td>0/21</td>
<td>0/21</td>
</tr>
</tbody>
</table>

Abbreviations: R, reactive; N, nonreactive; –, not tested; NBT, no blood transfusion.
* Expressed as recipients with abnormal results/"evaluable" recipients. For anti-HCV assays, recipients were "evaluable" if tested 12 months after transfusion or found reactive before 12 months after transfusion. For other assays, all recipients tested were considered "evaluable." RT-PCR was performed only on the earliest sample after transfusion.
† RIBA2 performed only on samples reactive by EIA1 and/or EIA2.
‡ An additional recipient was RIBA2 indeterminate.
§ These two recipient groups made up the control population.
Of the 3 RT-PCR–positive recipients persistently nonreactive by both EIA1 and EIA2 at 12 months after transfusion, one had received 1 unit reactive by EIA1 but not by EIA2 or RIBA2; the other 2 patients had received units that were reactive for anti-HCV by EIA1, EIA2, and RIBA2 (patients no. 14 and 15 in Table 3).

Conversely, on review of the 12 recipients found to have anti-HCV as shown by EIA1 or EIA2, 9 also had HCV RNA detected by RT-PCR; 8 of the 9 had an elevated ALT level on at least one posttransfusion sample. Of the 3 recipients with HCV antibodies but no detectable HCV RNA, 1 was the chronic hepatitis B patient whose results are described above and the other two received units that were RIBA2-reactive and are shown as patients no. 10 and 11 in Table 3.

RT-PCR testing was also used to investigate the single case of a recipient who remained negative for all HCV markers for 1 year after transfusion despite receiving a presumed infectious blood component (patient no. 1, Table 3). The component transfused was a 17-day-old unit of whole blood that was reactive by EIA1, EIA2, and RIBA2 and contained HCV RNA detected by RT-PCR. In association with coronary surgery, the patient also received 31 EIA1-reactive blood components on the same day as the reactive blood component. The recipient’s serum remained nonreactive for anti-HCV by EIA1 and EIA2 and was negative for HCV RNA by RT-PCR on three occasions during the 12-month follow-up period.

Recipients of no blood (controls). Twenty-one EIA1-nonreactive autologous donors/patients had surgery but did not require blood transfusion. EIA1 and EIA2 testing of these controls was entirely nonreactive before and after surgery, except for a single donor/patient who became EIA2-reactive (RIBA2 indeterminate) by 6 months after hysterectomy despite receiving no blood transfusions. She continues to be EIA2-reactive, but EIA1-nonreactive, 18 months after surgery and has been entirely asymptomatic with no ALT elevation before or after surgery. Her three autologous blood donations before surgery and all samples after surgery were negative for HCV RNA by RT-PCR.

DISCUSSION

The prevalence of anti-HCV by EIA1 in volunteer blood donations reported here (0.5%) is similar to that given by other US reports from that time period and somewhat less than that given in reports of blood donations that were not screened for anti-HBc and ALT level. Although the anti-HCV EIA1 has high specificity in patients with established non-A, non-B hepatitis, the specificity of the assay is quite low when applied to a blood donor population. In this study, use of a strip immunoblot assay (RIBA2) as a supplemental test to validate the apparent detection of anti-HCV by EIA1 suggested that 60% of EIA1-reactive samples were false-positives.

The specificity of the first anti-HCV test, EIA1, was evaluated primarily by testing for evidence of HCV in the recipients of blood components. When EIA1 was the only HCV marker evaluated in patients after transfusion, we found that anti-HCV occurred in no more than 32% of the recipients of an anti-HCV EIA1-reactive blood component. (The exact proportion may be less because the pretransfusion anti-HCV status of these recipients is unknown.) Van der Poel et al reported that only 20% of recipients of anti-HCV EIA1-reactive blood components developed antibody to HCV. When we evaluated recipients of units reactive by both EIA1 and supplemental anti-HCV testing (RIBA2), we found that 69% of recipients were anti-HCV EIA1-reactive.

**Table 3. Detailed Results of Follow-up Testing of 15 Patients Who Received at Least One Blood Component Found to be Reactive by All Three Anti-HCV Assays (EIA1, EIA2, and RIBA2)**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Reactive Component Received</th>
<th>Follow-up Testing of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EIA1</td>
<td>EIA2</td>
</tr>
<tr>
<td>1</td>
<td>Whole</td>
<td>N</td>
</tr>
<tr>
<td>2</td>
<td>Whole</td>
<td>N</td>
</tr>
<tr>
<td>3</td>
<td>RBC</td>
<td>N</td>
</tr>
<tr>
<td>4</td>
<td>RBC</td>
<td>R</td>
</tr>
<tr>
<td>5</td>
<td>Platelets</td>
<td>R</td>
</tr>
<tr>
<td>6</td>
<td>Platelets</td>
<td>R</td>
</tr>
<tr>
<td>7</td>
<td>Cryo</td>
<td>R</td>
</tr>
<tr>
<td>8</td>
<td>Platelets</td>
<td>R</td>
</tr>
<tr>
<td>9</td>
<td>RBC</td>
<td>R</td>
</tr>
<tr>
<td>10</td>
<td>RBC</td>
<td>R</td>
</tr>
<tr>
<td>11</td>
<td>RBC</td>
<td>N</td>
</tr>
<tr>
<td>12</td>
<td>RBC</td>
<td>N</td>
</tr>
<tr>
<td>13</td>
<td>RBC</td>
<td>N</td>
</tr>
<tr>
<td>14</td>
<td>RBC</td>
<td>N</td>
</tr>
<tr>
<td>15</td>
<td>RBC</td>
<td>N</td>
</tr>
</tbody>
</table>

Blank spaces indicate that no sample was available.

Abbreviations: Whole, whole blood; RBC, packed red blood cells; Cryo, cryoprecipitate; ALT, ALT expressed as IU/L (normal range is 0 to 53 IU/L); R, reactive; N, nonreactive; I, indeterminate; POS, HCV RNA detected; NEG, no HCV RNA detected.

* Performed only if the sample was reactive by EIA.
on follow-up. Other studies have shown that up to 88% of recipients of anti-HCV EIA1-reactive blood developed anti-HCV detectable by EIA. However, these other studies in which HCV transmission appeared to occur at a higher rate included transfusion of units with abnormal results on "surrogate" tests (anti-HBc and ALT). In contrast, our study highlights the fact that blood reactive for anti-HCV may be infectious for HCV despite having a normal ALT level and a nonreactive anti-HBc test.

The EIA2 assay for anti-HCV does appear to have increased sensitivity. Our finding of two transfusion recipients who were EIA2-reactive, but EIA1-nonreactive, agrees with the report of Aach et al stating that EIA2 detects additional HCV seroconversions posttransfusion.

In general, blood units with anti-HCV by EIA1 verified as reactive by RIBA2 appear to contain, and be capable of transmitting, HCV; units reactive by EIA1 but not by RIBA2 do not appear to be carrying HCV. In our study, evidence of HCV infection was found in 14 of 15 recipients (93%) of RIBA2-positive blood who completed follow-up, whereas evidence of HCV infection was found in only 3 of 27 recipients (11%) of EIA-reactive, RIBA2-negative blood (P < .01). Each of the 3 recipients had been previously extensively transfused and, thus, may have been infected before the study presented here. Similar infectivity data for RIBA-reactive blood components were reported by Esteban et al.

The results presented here raise the question of "serologically silent" HCV infections in the three multiply transfused, immunosuppressed patients who completed the full 12-month follow-up period. They had no detectable antibodies to HCV up to 12 months after transfusion, but HCV RNA was detected in their serum by RT-PCR. Although RT-PCR techniques in current use may have a 5% false-positive rate, that does not seem to have been the case here as separate aliquots sent for RT-PCR testing on different occasions (under code) yielded the same results. The lack of detectable HCV antibodies could be due to the failure of current tests to detect antibodies that were very weak or directed against unrepresented antigens or against other strains of HCV. However, it is most likely that these immunosuppressed patients are infected with HCV and are unable to mount an immune response.

The control group included 21 autologous donors/patients who had surgery but did not require any transfusions. This subgroup was included in the study to evaluate the existence of "hospitalization hepatitis." A large posttransfusion study reported that 3.3% of hospitalized patients acquired hepatitis (as defined by elevated liver enzymes during the 6 months after hospitalization) even if they did not receive blood transfusions. In our control group of 54 surgical patients who did not receive any homologous transfusions, 1 patient (1.9%) developed antibody to HCV. She became anti-HCV reactive by both EIA2 and RIBA2, but not by EIA1. This patient did not receive any transfusions while hospitalized for hysterectomy and denied other risk factors. RT-PCR was negative on multiple presurgery and postsurgery samples and ALT was consistently less than 53 IU/L. This patient may have had a transient HCV infection, an anamnestic antibody response, or a false-positive reaction to HCV antigens coincident with surgery and hospitalization. Overall, there was no established HCV infection in any of the patients who did not receive homologous (allo- genic) blood.

Our study encountered many difficulties inherent in studying transfusion-transmitted infections. First, despite testing more than 29,000 units of blood, the recipient group of interest (patients who received a unit with a reactive anti-HCV EIA1 test) is small due to the low prevalence of HCV in a "healthy" volunteer donor population. Second, patients ill enough to require homologous blood transfusion have a 50% 1-year mortality rate from their primary illness. In this study, more than 45% of 214 transfusion recipients had expired by 6 months posttransfusion. Of those enrolled, 9 of 42 (21%) died or became too ill to continue in the study at some point between 6 and 12 months after the transfusion. Third, despite being told they had received a unit of blood that might transmit HCV, many patients declined our offer of convenient, free follow-up testing. Fourth, because before anti-HCV licensing no ongoing study was in place to obtain pretransfusion samples from area patients before they received transfusions, pretransfusion HCV status was unknown. Despite these difficulties, this study did not permit the capture of data not available through the study of collections of linked donor and recipient sera from past eras when blood bank testing procedures and donor eligibility criteria were markedly different. The actual effect of using a new infectious disease assay to screen donor blood can only be adequately assessed at the time the change in testing is implemented and only if all types of patients receiving transfusions are included.

Infectious units identified by this study were not detected by blood screening tests in place before the availability of anti-HCV testing. The donors of these infectious blood components were asymptomatic, had no history of hepatitis, and their blood donations met all the requirements for transfusion in early 1990, including normal ALT levels and lack of anti-HBc. As an exercise, using the data from this study, one can approximate the reduction in posttransfusion hepatitis that occurred in the United States due to the implementation of the anti-HCV EIA1 test. Only 0.5% of 29,367 transfused units were reactive for anti-HCV by EIA1 and, considering all markers for HCV, 40% of recipients of an anti-HCV EIA1-reactive unit had some evidence of HCV infection during a 1-year follow-up period. Assuming that none of the recipients had HCV infection before the study, crude calculation suggests that implementation of anti-HCV EIA1 testing prevented up to 2 HCV infections per 1,000 units transfused. The only other US transfusion study conducted during the same time period, an ongoing study in cardiac surgery patients observed for 6 months posttransfusion, estimated that 1.6 HCV infections per 1,000 units transfused were prevented by the implementation of anti-HCV EIA1 testing.

After our study was completed, anti-HCV EIA2 was licensed by the FDA and implemented by blood centers. It has been estimated that EIA2 testing of blood for transfusion further reduces the rate of HCV transmission by another 1 per 1,000 units of blood transfused. The current...
residual risk for non-A, non-B hepatitis per unit transfused in the United States has been estimated at 0.03% to 0.05%. 18,19

ACKNOWLEDGMENT
Ortho Diagnostic Systems provided test kits for anti-HCV EIA first- and second-generation testing (Ortho Diagnostic Systems Inc. Ortho also provided the Riba strip immunoblot assay (Chiron Corp).

REFERENCES
Evidence of hepatitis in patients receiving transfusions of blood components containing antibody to hepatitis C

SK Aoki, PV Holland, LP Fernando, IK Kuramoto, S Anderson, K Sazama, V Schoening, P Johnson, JC Wilber and L Fenner