

Immunohematology

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Immunoprophylaxis using intravenous Rh immune globulin should be standard practice when selected D-negative patients are transfused with D-positive random donor platelets

C.A. EWING, D.H. RUMSEY, A.F. LANGBERG, AND S.G. SANDLER

A 67-year-old female developed excessive bleeding and thrombocytopenia following cardiovascular surgery. Her blood type was group A, D-. The only platelet products available in the transfusion service were random donor platelet concentrates from D+ donors. She was transfused with a pool of 6 D+ random donor platelet concentrates. Anti-D undetected in her pretransfusion serum by solid-phase antibody screen was present 11 days later. Retrospectively, the patient provided a history of having two pregnancies more than 40 years ago, prior to the availability of immunoprophylaxis by Rh immune globulin (RhIG). Although studies have shown that as many as 19 percent of D- people may develop anti-D following transfusion of platelets from D+ donors, there is no specific standard requiring immunoprophylaxis with RhIG to prevent Rh alloimmunization after transfusion of random donor platelet concentrates from D+ donors. In contrast, vigorous efforts are routine for preventing Rh alloimmunization in D- patients requiring red cell transfusions or D- females during pregnancy or after delivery of D+ newborns. The absence of a comparable practice standard for platelet transfusions is based, in part, on concern that intramuscular injections of conventional RhIG may cause local hemorrhage in thrombocytopenic persons. The recent availability of a Food and Drug Administration-approved preparation of intravenous RhIG makes Rh immunoprophylaxis in thrombocytopenic patients safe and practical. We recommend that intravenous RhIG be considered if it is necessary to transfuse random donor platelet concentrates from D+ donors to D- recipients. As a minimal standard, intravenous RhIG should be administered to all D- females of childbearing age who are recipients of pools of random donor platelet concentrates from D+ donors. *Immunohematology* 1998;14:133-137.

Key Words: Rh alloimmunization, anti-D, RhIG, blood transfusion standards, platelet transfusion

Contemporary blood banking practice requires avoidance of Rh alloimmunization by transfusing only D- red blood cells (RBCs) to D- recipients. Transfusion services make vigorous efforts to ensure that highly sensitive and well-controlled serologic testing prevents accidental Rh

alloimmunization by RBC transfusions. Comparable standards have been promulgated to minimize the risk of Rh alloimmunization by fetomaternal hemorrhage by routine Rh immunoprophylaxis using Rh immune globulin (anti-D, RhIG) when a D- female is at risk for Rh alloimmunization as the result of pregnancy with a D+ fetus. In contrast, there is no specific standard that requires Rh immunoprophylaxis when D- people are at risk of Rh alloimmunization by contaminant D+ RBCs in platelet transfusions. Although the incidence of alloimmunization after transfusions of random donor platelet concentrates from D+ donors to D- recipients has been reported to be as high as 19 percent,¹ immunoprophylaxis using RhIG after such platelet transfusions is not a routine practice. Recently, we transfused a pool of 6 random donor platelet concentrates from D+ donors to a D- female, who subsequently developed anti-D. Although her anti-D may have been the result of a *secondary* immune response, the incident caused us to review our hospital's experience and practices for preventing *primary* Rh alloimmunization from platelet transfusions in D- recipients. In the course of our review of this event, we recognized that the recent availability of Federal Drug Administration (FDA)-approved RhIG for intravenous administration makes it now possible—for the first time in the United States—to perform Rh immunoprophylaxis in thrombocytopenic people without the risk of local hemorrhage from an intramuscular injection of RhIG.^{2,3} As a result, we have revised our hospital's policy, recommending a more aggressive approach to Rh immunoprophylaxis when D-

people—especially women of childbearing age—are transfused with random donor platelet concentrates from D+ donors. We present the following case report and discussion as a reminder that pools of random donor platelet concentrates may contain sufficient RBCs to induce an immune response. The option of preventing primary Rh alloimmunization in D- thrombocytopenic recipients of platelets from D+ donors by using intravenous RhIG should prompt a reevaluation of current practice.

Case Report

A 67-year-old, group A, D- female developed thrombocytopenia and excessive bleeding following cardiovascular surgery. A request for platelet transfusion could not be filled in the blood bank by using D- random or single-donor (pheresis) platelet concentrates. A pool of 6 units of random donor platelet concentrates, consisting of 3 group A, D+ and 3 group O, D+ units, was issued using platelet concentrates supplied by the American Red Cross regional blood center. A pretransfusion antibody screen by a solid-phase method (Capture-R Ready Screen, Immucor, Inc., Norcross, GA) was negative for the presence of alloantibodies, but a routine postoperative sample collected on day 11 revealed the presence of anti-D. Serial blood samples collected after the platelet transfusion documented an anti-D immune response (Table 1). Retrospective testing of the pretransfusion serum confirmed the absence of anti-D by enzyme (ficin/anti-human globulin [AHG]) and polyethylene glycol/AHG methods. An interview with the patient revealed that her two pregnancies occurred prior to the availability of RhIG. She did not recall previous transfusions. No adverse clinical events resulted from the patient's platelet transfusion. She was discharged and informed that if she needed an RBC transfusion in the future, she would require D-blood.

Table 1. Anti-D titers before and following transfusion of D+ random donor platelet concentrates

Test method	Pretransfusion*	Posttransfusion				
		Day 11	Day 12	Day 13	Day 14	Day 17
Instant spin, room temperature	0	<1	<1	<1	<1	<1
37°C	0	1	1	1	2	2
Solid phase (Capture-R)	0					
Saline/indirect antiglobulin	0	64	128	128	256	256

*Retrospective testing also confirmed the absence of anti-D by ficin/anti-human globulin (AHG), and polyethylene glycol/AHG methods

Materials and Methods

Random donor platelets

Random donor platelets were prepared by the American Red Cross according to standard operating procedures. These procedures monitor the extent of contamination with RBCs by visual inspection of each concentrate. Units that are grossly contaminated by visual inspection are not released. The procedure does not include an RBC count on individual units of platelet concentrates.

Estimating red cell contamination of random donor platelet concentrates

To estimate the quantity of contaminating RBCs that were transfused to this patient (or any other recipient of random donor platelet concentrates), we conducted a simple experiment by injecting a standard 50mL outdated random donor platelet concentrate with increasing increments of concentrated RBCs. We recorded the color changes that occurred in the bag after adding each 0.1 mL increment, until the color of the platelet concentrate was burgundy from RBC contamination.

Platelet utilization

To determine how often our blood bank issued platelet concentrates from D+ donors to D- recipients, we examined platelet administration records for the previous 1-year period. We confirmed by interviews with blood bank personnel that random donor platelet concentrates from D+ donors were issued to D- recipients only when D- platelets were not available. Using the blood bank's computerized records, we searched for the results of blood group antibody screens that had been performed on D- recipients of platelets from D+ donors.

Results

Red cell contamination of platelet concentrates

The first color change that could be detected was a pink tinge after 0.1 mL of RBCs was added to the 50 mL volume of platelet concentrate (Table 2). This color change was obvious to all observers in the context of the study. However, the color change was sufficiently subtle for us to anticipate that it would not be detected by all observers under conditions of routine blood bank operations. After 0.3 mL of RBCs were added, the color of the platelet concentrate was overtly pink. The color changed to *rosé* after 0.5 mL and then to burgundy after 0.8 mL of RBCs were added. Because overtly pink random donor

platelet concentrates (0.3 mL of RBCs) are the most contaminated units that might be issued by our blood bank, we estimated that the quantity of contaminating RBCs in the transfused pool of 6 units of random donor platelet concentrates was 1.8 mL or less.

Table 2. Color changes observed after adding red blood cells (RBCs) to a random donor platelet concentrate

Volume of RBCs (mL)	Color
0.0	Yellow, slightly turbid
0.1	First detectable pink tinge
0.2	Pink tinge
0.3	Pink
0.4	Pink
0.5	Rosé
0.6	Rosé
0.7	Rosé
0.8	Burgundy
0.9	Burgundy
1.0	Burgundy

Platelet utilization

Of 3587 units of platelets issued by the hospital's blood bank during the previous year, 1327 units were random donor platelet concentrates and 2260 units were single-donor (pheresis) units. These platelets were transfused to 400 D+ and 60 D- recipients.

Of the D- recipients, 33 received 151 units of single-donor (pheresis) platelets; 81 units were D+ and 70 units were D-. Twenty-one of these patients received D+ units. Of the 60 D- patients, 38 received 69 units of pooled random donor platelets; 34 units were D+, and 35 units were D-. Twenty-two of these patients received D+ units. Two D- patients received both D+ single-donor and D+ pooled random donor concentrates.

The retrospective review of laboratory records revealed that postplatelet transfusion antibody screens were performed in 26 of the 41 D- patients who had received platelets from D+ donors. Of these patients, two had antibody screens that were positive for anti-D. One of these patients had anti-D prior to transfusion, and the other had received a routine postpartum injection of RhIG prior to the antibody screen. The mean time interval between transfusion and follow-up antibody screens was 43 days (range: 9-240 days); the median time interval was 20 days.

Discussion

In our hospital, as in most other hospitals in the United States, there is a vigorous effort to prevent Rh alloimmunization when transfusing RBCs to D- patients. Comparable vigilance and standards apply for managing pregnancies and deliveries in D- females at risk for Rh alloimmunization by fetomaternal hemorrhage. In con-

trast, as illustrated by this case report, hospital practice with regard to D- recipients of platelet transfusions is not as rigorous. The American Association of Blood Banks' *Standard for Blood Banks and Transfusion Services* (16th edition, 1994) states that "treatment with Rh Immune Globulin for prevention of immunization to D may be appropriate for a D-negative patient who has received D-positive red blood cells, including those transfused with red cells..."⁴ The current *Standards* (18th edition, 1996) states that "each blood bank and transfusion service must have written criteria for Immune Globulin prophylaxis for Rh-negative patients who receive blood components containing Rh-positive red blood cells."⁵ The only change proposed for the next edition of *Standards* (19th edition, 1999) is the addition of the following nonbinding statement: "Rh-negative patients who have been exposed to Rh-positive red cells shall be candidates for Rh Immune Globulin treatment" (*News Briefs*, American Association of Blood Banks, July 1998, pg. 15). In 1998, the American Society of Clinical Pathologists (ASCP) published a practice parameter on the use of Rh immune globulin.⁶ This practice parameter is also permissive with regard to platelet transfusions in D- women of childbearing potential: "Depending on the volume of Rh-positive RBCs transfused, the childbearing potential of the recipient, and the clinical situation, RhIG prophylaxis should be considered for Rh-negative patients who receive Rh-positive blood components."⁶ The ASCP document makes no specific recommendation for Rh prophylaxis in D- women who have been transfused with D+ platelets, in contrast with specific recommendations for pre- and postpartum Rh prophylaxis. Also, there is no reference to prophylaxis with RhIG for D- women exposed to D+ RBCs in platelet transfusions in the 1997 final consensus statement of the Royal College of Physicians of Edinburgh/Royal College of Obstetricians and Gynecologists.⁷ Thus, there is no standard of practice in the United States or in the United Kingdom that requires Rh immunoprophylaxis with RhIG if a D-woman of childbearing age receives a transfusion of pooled random donor platelet concentrates from D+ donors. Also, there is no standard for the maximum number of RBCs that is acceptable in a random donor platelet concentrate.

The case we report represents an uncommon situation in our hospital, and we assume elsewhere, when a bleeding D- patient requires a platelet transfusion and random donor platelet concentrates from D+ random donors are the only platelets available. Our hospital records reveal that 52 percent of all D- patients requiring platelet trans-

fusions were able to be managed using D- or D+ single-donor (pheresis) platelets exclusively. These products contain a negligible number of RBCs and are unlikely to sensitize recipients to blood group antigens. Thirty-seven percent (22/60) of D- recipients of platelet transfusions required at least one pool of random donor platelets from D+ donors. These 22 recipients represent 4.7 percent of all recipients of platelet transfusions in our hospital during the 1 year surveyed. We have no reason to believe that this experience is exceptional, given the prompt response expected for supplying platelets and the difficulty in managing an inventory of D- platelet products. As a result of our internal review of this event, we have a heightened awareness that for those uncommon events, there is now an effective and safe means of avoiding the risk of Rh alloimmunization by using intravenous RhIG.

In the specific example of our case, we assume that the detection of anti-D only 11 days after exposure to D+ RBCs reflects a secondary immune response resulting from sensitization during previous pregnancies. Although the estimated volume of RBCs in her pool of platelet concentrates (1.8 mL) was adequate for inducing a secondary immune reaction, this dose is also adequate for inducing a primary immune response to the D antigen.⁸ Studies of D- recipients of platelet transfusions from D+ donors document an incidence of 0-19 percent Rh alloimmunization.^{1,9-12} Most of these recipients were immunocompromised because they were transfused following chemotherapy for malignant diseases; therefore, it is reasonable to expect the incidence of Rh alloimmunization to be higher in immunocompetent recipients of platelet transfusions.

In our hospital, the absence of a requirement to prevent Rh alloimmunization by routine injections of RhIG following transfusions of D+ platelets to D- recipients is based on (a) the concern that intramuscular injections in thrombocytopenic recipients may cause a deep intramuscular hemorrhage or bleeding at the injection site, (b) the perception of a low risk of Rh alloimmunization in platelet recipients, and (c) the rarity of the situation. This case prompted a timely review of this practice, because recent FDA approval of an intravenous anti-D immunoglobulin preparation (WinRho SDF™, Nabi, Boca Raton, FL) presents an opportunity to update our practice. In addition to approval for suppression of Rh alloimmunization in nonsensitized D- women with an Rh-incompatible pregnancy, WinRho is also FDA-approved for the suppression of Rh alloimmunization in D- premenopausal women transfused with D+ blood or blood components containing RBCs.³ In the case of transfusion of blood

components containing D+ RBCs to a D- individual, the manufacturer's package insert recommends 18 micrograms (90 International Units) per milliliter of RBCs for adequate prophylaxis. Therefore, an intravenous injection of 64.8 micrograms (or 1.3 mL using the 120 microgram/vial preparation) should be adequate for preventing Rh alloimmunization with a 100 percent margin of safety (double dose) following transfusion of a pool of 6 units of random donor platelet concentrates containing approximately 1.8 mL of RBCs. The total dose could be given as a single injection because it is less than the manufacturer's maximum recommended single dose of 600 micrograms (3000 International Units). Because a standard 300 microgram dose of intramuscular RhIG persists as detectable anti-D in most women for several months, we anticipate that a 64.8 microgram dose of intravenous anti-D (WinRho SDF™) also will persist in the circulation for several months. The need for a follow-up dose could be determined by performing an antibody screen for anti-D, if additional D+ platelet transfusions are required.

In summary, we recommend that Rh immunoprophylaxis with intravenous RhIG be considered whenever it is necessary to transfuse D- recipients with random donor platelet concentrates from D+ donors. Intramuscular RhIG may be a suitable alternative, if injected in the immediate postplatelet transfusion period and if there is evidence that the transfused platelets will be clinically effective for at least several hours to prevent thrombocytopenic bleeding at the injection site. Immunoprophylaxis with intravenous RhIG should be a minimum standard for all D- women of childbearing age who are recipients of transfusions of random donor platelet concentrates from D+ donors.

References

1. McLeod BC, Piehl MR, Sasseti RJ. Alloimmunization to RhD by platelet transfusions in autologous bone marrow transplant recipients. *Vox Sang* 1990; 59:185-9.
2. Hong F, Ruiz R, Price H, et al. Safety profile of WinRho anti-D. *Semin Hematol* 1998;35(Suppl):9-13.
3. Package insert: Rho (D) Immune Globulin Intravenous (Human)/WinRho SDF™ Cangene Corporation, Winnipeg, Canada. Distributed by Nabi, Boca Raton, FL.
4. Standards for Blood Banks and Transfusion Services. 16th ed. Bethesda, MD: American Association of Blood Banks, 1994.
5. Standards for Blood Banks and Transfusion Services. 18th ed. Bethesda, MD: American Association of Blood

- Banks, 1996.
6. Hartwell EA. Use of Rh immune globulin/ASCP practice parameter. *Am J Clin Pathol* 1998;110:281-92.
 7. Urbaniak SJ. Consensus conference on anti-D prophylaxis, April 7 & 8, 1997: final consensus statement, Royal College of Physicians of Edinburgh/Royal College of Obstetricians and Gynecologists. *Transfusion* 1998;38:97-9.
 8. Mollison PL, Engelfriet CP, Contreras M. Blood transfusion in clinical medicine. 10th ed. Oxford, England: Blackwell Science, 1997:175.
 9. McGinniss MH, Bronson WR, Freireich E, Kliman A. Formation of red cell antibodies in response to platelet transfusions. *Transfusion* 1963;3:426.
 10. Goldfinger D, McGinniss MH. Rh-incompatible platelet transfusions—risks and consequences of sensitizing immunosuppressed patients. *N Engl J Med* 1971;284:942-4.
 11. Lichtiger B, Surgeon J, Rhorer S. Rh-incompatible platelet transfusion therapy in cancer patients. *Vox Sang* 1983;45:139-43.
 12. Lichtiger B, Hester JP. Transfusion of Rh-incompatible blood components to cancer patients. *Haematologia* 1986;19:81-8.

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Detection of anti-D following antepartum injections of Rh immune globulin

M.S. KENNEDY, J. MCNANIE, AND A. WAHEED

Antepartum prophylaxis using Rh immune globulin (RhIG) at 28 weeks of gestation is routine in unsensitized Rh-negative women. As various sources state that anti-D may be detected up to 6 months after administration, we reviewed the medical and laboratory records of all Rh-negative women who delivered at our institution during 1995. For 385 evaluable women, only 137 (35.6%) had anti-D demonstrable in their sera at delivery; 97.8 percent of these delivered within 75 days after administration of RhIG. Of 248 women (64.4%) who delivered in < 76 days after administration of RhIG, 134 (54%) had demonstrable anti-D. For 123 women who delivered between 76 to 95 days after RhIG, only 3 (2.4%) had demonstrable anti-D. Of 14 women who delivered more than 96 days after RhIG, none had anti-D at delivery. These data show that the 300- μ g dose used in the United States may not be adequate for antepartum protection and that the detection of anti-D more than 100 days after the administration of RhIG should be viewed with suspicion. *Immunohematology* 1998;14:138-140.

Rapid screening of platelet donors for PI^{A1} (HPA-1a) alloantigen using a solid-phase microplate immunoassay

J.L. PROCTER, F. VIGUE, E. ALEGRE, J. HONDA, K. MATSUO, AND D. REID

PI^{A1} and PI^{A2} are alternative platelet-specific alloantigens in the PI^A system. Sensitization to PI^{A1} underlies most cases of neonatal alloimmune thrombocytopenia (NAIT) and posttransfusion purpura (PTP) in white populations. A rapid and simple method for large-scale platelet phenotyping is desirable for identifying expectant mothers at risk of allosensitization and for identifying PI^{A1} -negative donors when transfusions are indicated for treatment of NAIT or PTP. We investigated the effectiveness of a solid-phase microplate immunoassay for this purpose. Platelet-rich donor plasmas were tested using the Capture-P[®] kit (Immucor, Norcross, GA). Platelet monolayers in microtiter wells were incubated with anti- PI^{A1} , washed, and exposed to red blood cells (RBCs) precoated with anti-human IgG. Adherence of RBCs in a diffuse pattern across the well surface indicated the attachment of anti- PI^{A1} to PI^{A1} -positive platelets whereas sedimentation of unattached RBCs into a central pellet indicated the platelets were PI^{A1} -negative. Of 520 donors, 15 (2.88%) tested PI^{A1} -negative, which correlates well with the reported $PI^{A2,2}$ frequency in whites of 2.25 percent. Results were confirmed by DNA genotyping and/or immunoblotting. This screening technique permits phenotyping donors for PI^{A1} alloantigen with minimal specialized equipment. Confirmatory testing for PI^{A2} alloantigen can be reserved for donors that test negative for PI^{A1} . *Immunohematology* 1998;14:141-145.

Evaluation of column technology for direct antiglobulin testing

J.M. MOULDS, L. DIEKMAN, AND T.D. WELLS

Preliminary reports have suggested that microcolumn technology might be too sensitive for direct antiglobulin testing (DAT). We studied 228 samples from patients with autoimmune diseases and 30 samples from healthy controls to determine the sensitivity of column techniques. Both Sephadex[®] gel and protein A/G columns were compared with manual methods using rabbit or murine polyspecific reagents. Of the 187 samples that were negative by both manual methods, an additional 29 (15%) and 42 (22%) samples gave weakly positive reactions with the Sephadex[®] and protein A/G bead columns, respectively. Subsequently, there was poor correlation between manual and column techniques ($r = 0.40-0.61$). Acid eluates from these samples were negative. We concluded that the column technology may detect too many weakly positive DATs that are clinically insignificant. *Immunohematology* 1998;14:146-148.

Comparison of affinity column technology and LISS tube tests

K. CHAMPAGNE, P. SPRUELL, J. CHEN, L. VOLL, G. SCHLANSER, AND M. MOULDS

Proteins G and A coated on agarose have been extensively used in affinity chromatography. Protein G will bind to all four subclasses of human IgG and protein A to the subclasses IgG1, IgG2, and IgG4. This IgG binding ability of protein G and protein A has been used in a red cell affinity column technology developed for the detection and identification of IgG red cell antibodies. When serum or plasma is incubated in a microcolumn with red blood cells (RBCs) that express the appropriate antigens, the antibodies become attached to the RBC surface. When the microcolumns are centrifuged, the RBCs pass through a viscous barrier into an active matrix containing proteins G and A. Positive tests adhere at the top of the gel and negative tests pass through, settling to the bottom. This study was undertaken to compare affinity column technology with low-ionic saline solution (LISS) tube tests in a reference laboratory setting. Over a 1-year period, 314 samples were tested in parallel by affinity column technology and by LISS tube technique. Both methods detected antibodies directed at common RBC antigens, high-incidence and low-incidence RBC antigens, and warm-reacting autoantibodies. IgM antibodies were not detected by affinity column technology. Affinity column technology compares favorably with the LISS tube technique for IgG antibody detection and identification. *Immunohematology* 1998; 14:149-151.

Comparison of gel technology and red cell affinity column technology in antibody detection

S.I. CHANFONG AND S. HILL

Both column (gel) agglutination technology and red cell affinity column technology (ReACT™) have been approved by the Food and Drug Administration for antibody detection and identification. Parallel studies using these two methods were performed on 100 samples to evaluate their sensitivity, advantages, and disadvantages. Sixteen significant antibodies, anti-D(2), -C(1), -E(1), -c(1), -C,D(1), -K(4), -S(1), -Fy^a(3), -Jk^a(1), and -Jk^b(1), were found during the study. MTS-Gel detected one anti-D due to Rh immune globulin but missed one anti-Jk^a. ReACT missed one anti-D and one anti-Jk^b. MTS-Gel detected one anti-I and one anti-H whereas ReACT detected two anti-H but not anti-I. No false positive reactions were found by either method. Sensitivity based on this study for MTS-Gel is 93.3% and ReACT is 86.7%. Advantages for MTS-Gel included the small volume needed for testing, and the reaction was stable for 48 hours; for ReACT, there was less spin time and no special pipette was needed. Disadvantages for MTS-Gel included the need for a special pipette and manual preparation of 0.8% RBC suspensions, and the disadvantages for ReACT included the small column for reaction reading and the reaction was stable for only 24 hours. *Immunohematology* 1998;14:152-154.

A comprehensive IgA service provided by a blood transfusion center

R. MUNKS, J.R. BOOTH, AND R.J. SOKOL

IgA is best known in transfusion practice for its deficiency when anti-IgA antibodies cause severe anaphylactic reactions. Following the realization that IgA deficient products were needed on demand, blood donors were routinely screened, initially by latex agglutination inhibition and subsequently by hemagglutination inhibition using an Olympus PK-7200™ blood grouping machine. IgA deficiency (<.0016 g/L) was found in 357 (with anti-IgA in 28%) of 301,310 donors, an incidence of 1 in 844. By screening new donors and directed call-up, group O, D-red blood cell (RBC) units are always in stock. During 1 year, the center supplied 79 units of RBCs and 64 units of fresh frozen plasma to a variety of patients with IgA deficiency, including three undergoing liver transplantation. The center also provides a reference service for IgA/anti-IgA status. The technique used (hemagglutination inhibition) has a sensitivity well below the threshold of standard quantitation methods. Samples were most commonly referred from departments investigating possible immunodeficiency and suspected transfusion reactions. Of 247 patients investigated, 122 had IgA deficiency, 43 with anti-IgA (of whom 5 had suffered a transfusion reaction). Donors and patients with anti-IgA were issued blood group cards warning that they should only receive IgA deficient products. *Immunohematology* 1998; 14:155-160.