Update for nurse anesthetists

Transfusion-related acute lung injury

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Acute onset of dyspnea, fever, hypotension, and cough during or soon after a blood or blood-product transfusion has been described in the literature since the early 1950s. Between 2003 and 2005, transfusion-related acute lung injury (TRALI) surpassed ABO incompatibility as the number one cause of transfusion-related mortality in the United States, as reported to the Food and Drug Administration. Prompt recognition and appropriate intervention are required to reduce mortality. Prevention of TRALI represents a complex challenge. There is no single intervention that will prevent every case of TRALI, but several interventions have been proposed to reduce the risk.

Key words: Acute respiratory distress syndrome (ARDS), transfusion-associated circulatory overload (TACO), transfusion-related acute lung injury (TRALI).

Objectives
At the completion of this course, the reader should be able to:
1. Define transfusion-related acute lung injury (TRALI).
2. Differentiate TRALI from transfusion-associated circulatory overload, anaphylactic reaction to blood transfusion, or bacterially contaminated blood transfusion.
3. List the signs and symptoms of TRALI and the time frame of their onset.
4. State 2 current hypotheses for the cause of TRALI.
5. Discuss suggested interventions that may reduce the risk of TRALI.

Introduction
Transfusion-related acute lung injury (TRALI) is a little-known, frequently misdiagnosed complication that occurs with the transfusion of blood products. Because of the similarities of TRALI to other syndromes associated with blood-product transfusions, this syndrome is frequently confused with circulatory overload, and interventions may be directed toward alleviating the volume overload; however, volume overload is not the underlying problem. According to the number of cases that have been reported thus far, TRALI is associated with a mortality rate of 5% to 10%. Rapid diagnosis to allow for appropriate interventions is essential for alleviation of this syndrome rather than contributing to exacerbation. This AANA Journal course will begin with 3 hypothetical cases for anesthetists to consider. The 3 cases seem unrelated, but on closer inspection the common thread becomes more evident.

Case 1
A 48-year-old woman underwent a total abdominal hysterectomy. The preoperative diagnosis was uterine fibroids with abnormal uterine bleeding and anemia. The patient was 67 in (170.2 cm) tall and weighed 138 lb (62.1 kg). Preoperative laboratory data revealed the following: hemoglobin level, 9.3 g/dL; hematocrit, 32.4%; sodium level, 137 mEq/L; potassium level, 3.9 mEq/L; chloride level, 107 mEq/L; serum urea nitrogen level, 14 mg/dL; and creatinine level, 0.9 mg/dL. General anesthesia was induced with midazolam, 2.5 mg; fentanyl, 100 µg; lidocaine, 80 mg; and propofol, 130 mg. Tracheal intubation was facilitated with succinylcholine, 120 mg. General anesthesia was maintained by using sevoflurane, 2.5% to 3.7% in a 50% oxygen-air mixture. Muscle relaxation was accomplished with cis-atracurium, 30 µg/kg. Because of difficulty in ligating uterine vascular structures, intraoperative blood loss was estimated to be 650 mL. The total intravenous (IV) fluid volume given was 2,100 mL of lactated Ringer's solution. The
Intraoperative hemoglobin level was 7.8 g/dL. Transfusion of 1 U of packed red blood cells was ordered and administered intraoperatively. Neostigmine and glycopyrrolate were administered 75 minutes after completing the transfusion and at the end of surgery. On demonstration of adequate muscle strength and good spontaneous ventilatory efforts, the patient was extubated and transported to the PACU; she was receiving supplemental oxygen via simple mask.

On admission to the PACU, the patient complained of dyspnea with an unrelenting cough, and she had a fever (temperature, 38.4°C [101.1°F]) and a pulse oximeter reading of 88%. Oxygen delivery was changed to humidified oxygen at a fraction of inspired oxygen (FiO₂) of approximately 60%; however, the patient’s pulmonary function continued to decline. Arterial blood gas levels were obtained; the results were as follows: pH, 7.39; Po₂, 50 mm Hg; and PCO₂, 35 mm Hg. The patient was sedated with midazolam, 5 mg, and then tracheally intubated and mechanically ventilated. Crackles were auscultated in the lower lobes bilaterally, and pink, frothy fluid was observed in the endotracheal tube. The fluid was suctioned from the tube. A chest radiograph revealed diffuse alveolar infiltrates bilaterally.

The patient was admitted to the ICU, and her respiratory status was maintained by mechanical ventilation. The patient did not, at any time, demonstrate overt signs or symptoms of volume overload, such as jugular vein distention. Approximately 48 hours after admission to ICU, the patient was extubated, demonstrated no further evidence of respiratory distress, and was transferred to a general inpatient ward. She was discharged home on postoperative day 5.

**Case 2**

A 67-year-old man with osteoarthritis was admitted for a right hip arthroplasty. The patient was 73 in (185.4 cm) tall and weighed 265 lb (119.3 kg). Preoperative laboratory data included the following: hemoglobin level, 12.5 g/dL; hematocrit, 38.7%; sodium level, 139 mEq/L; potassium level, 4.2 mEq/L; chloride level, 109 mEq/L; serum urea nitrogen level, 12 mg/dL, creatinine level, 1.1 mg/dL, and international normalized ratio, 1.7.

The patient's medical history was significant for chronic atrial fibrillation. Warfarin was discontinued 5 days before the date of surgery. Fresh frozen plasma (FFP), 300 mL, was infused 2 hours before surgery. The patient’s international normalized ratio, measured again 1 hour after completion of the FFP transfusion, was 1.5. The proposed surgical procedure went on as scheduled. A subarachnoid block was accomplished using 0.5% bupivacaine, 2 mL, with 1:200,000 epinephrine. The patient received IV midazolam, 2.5 mg, for sedation. A continuous propofol infusion was prepared and was about to be started; however, during placement of the surgical drapes, the patient complained of dyspnea, became tachycardic, and appeared to be in acute distress; a persistent cough developed. The patient became febrile, with a skin temperature of 38.6°C (101.5°F), and the pulse oximeter reading steadily declined from 98%, with oxygen via simple mask, to 87%. As the draping was completed, the copious amounts of pink, frothy sputum were produced. The patient's respiratory function continued to decline, and the case was canceled.

The patient was transferred to the PACU with oxygen via a nonrebreathing mask and was hypotensive, blood pressure, 84/42 mm Hg; and had a pulse oximeter reading of 84%, despite an FiO₂ of 100% via the nonrebreathing mask. Arterial blood gas levels were as follows: pH, 7.38; Po₂, 47 mm Hg, and PCO₂, 32 mm Hg. The patient was sedated with midazolam, 3.5 mg, tracheally intubated, and mechanically ventilated. The chest radiograph demonstrated lung fields “whited-out” with diffuse alveolar infiltrates. The patient was transferred to the ICU, mechanically ventilated, and sedated. On the fourth day in ICU, the patient was extubated, transferred to the orthopedic floor, and discharged home the following day.

**Case 3**

A 37-year-old woman who weighed 195 lb (87.8 kg) and was 63 in (160.0 cm) tall was admitted for exploratory laparotomy for severe abdominal pain with distention. The pain had become progressively worse during the previous 10 days. The patient had undergone a laparoscopic Roux-en-Y procedure 1 year earlier for morbid obesity.

The exploratory laparotomy revealed gas gangrene of a segment of small bowel that had incarcerated into an internal hernia. After a small bowel resection, her wound was packed open with plans for a return to the operating room (OR) for further irrigation and drainage after receiving antibiotic therapy for 2 to 3 days. The patient was transferred to the ICU and received vancomycin, ciprofloxacin, and metronidazole for the next 3 days. On the third postoperative day, the patient was brought back to the OR for reexploration. The pertinent laboratory data included the following: hemoglobin level, 8.5 g/dL; hematocrit, 28.4%; platelet count, 65 x 10⁹/µL cells per high-power field; sodium level, 134 mEq/L; potassium level, 3.3 mEq/L; chloride level, 105 mEq/L; serum urea nitrogen level, 18 mg/mL; and creatinine level, 1.8 mg/dL. The patient was intubated and mechanically ventilated on arrival in the OR. Desflurane, 4%
to 5.5%, in a 75% oxygen-air mix was used to establish and maintain general anesthesia, and muscle relaxation was achieved with atracurium. Because of the low platelet count, 6 U of platelets were ordered for transfusion. After receiving approximately 75 mL of the platelet transfusion, the patient became profoundly hypotensive and febrile, with her core temperature increasing from 36.2°C (97.1°F) to 39.1°C (102.4°F). Ventilatory pressures increased significantly, and pink, frothy fluid was observed in the endotracheal tube. The platelet infusion was immediately stopped and the IV tubing flushed. The irrigation and drainage of the patient’s abdomen was interrupted, and the patient was returned to the ICU. A follow-up chest radiograph in the ICU showed the classic white-out produced by diffuse alveolar infiltrates. Arterial blood gas results were as follows: pH, 7.45; P\textsubscript{O\textsubscript{2}}, 55 mm Hg; and P\textsubscript{CO\textsubscript{2}}, 38 mm Hg. The patient’s breath sounds were distant with audible crackles bilaterally over the bases. There were no overt signs of volume overload, even to the point of nominal values for the central venous and pulmonary artery occlusion pressures. After additional trips to the OR on subsequent days, the abdominal wound was eventually closed. The patient was able to be extubated 10 days after admission. Three weeks after the initial admission to the OR, the patient was discharged from the hospital.

**Definition and signs and symptoms of TRALI**

The 3 cases involved the development of dyspnea, fever, hypotension, diffuse alveolar infiltrates, and pulmonary edema without demonstrable signs or symptoms of circulatory or volume overload. Each crisis was precipitated by the transfusion of a blood product, although each case involved a different blood product. Once circulatory overload and anaphylaxis were ruled out, the syndrome of TRALI emerged as the likely diagnosis.

The first description of fatal pulmonary edema associated with the transfusion of blood or blood products was in 1951, by Barnard.\(^1\) Six years later, Brittingham\(^2\) reported the development of fever, hypotension, dyspnea, bilateral diffuse alveolar infiltrates, and transient leukopenia in a healthy volunteer who received 50 mL of whole blood as a part of an investigation. In 1983, Popovsky et al\(^3\) described the initial defining criteria for the syndrome they termed TRALI. The TRALI syndrome has had various names (Table 1), although today, we recognize the definition by Popovsky et al\(^3\): a temporary, noncardiogenic pulmonary edema brought on by the transfusion of blood and/or blood products. All blood products have been implicated in the development of TRALI, particularly products containing higher volumes of plasma.

The onset of TRALI symptoms may occur in less than 30 minutes after the initiation of a blood-product transfusion, but most commonly, onset is within 1 or 2 hours, with the accepted latest time of onset being 6 hours after the transfusion. General symptoms of TRALI, initially defined by Popovsky et al\(^3\), include fever, dyspnea, cough, hypoxemia, and hypotension or hypertension. In addition to these defining symptoms, tachycardia, cyanosis, and acute hypoxemia accompany the onset of TRALI. The acute hypoxemia is characterized by an oxygen saturation of 90% or less while breathing room air, which corresponds to an approximate \(\text{PaO}_2\) value of 60 mm Hg or less. Generally, in a patient in whom TRALI develops, the \(\text{PaO}_2/\text{FiO}_2\) ratio is 60 mm Hg or less/0.21 or 300 mm Hg or less, which is in line with the \(\text{PaO}_2/\text{FiO}_2\) ratio criteria established for acute lung injury (ALI)\(^4\) (Table 2).

Cases of TRALI are frequently misdiagnosed, especially mild-to-moderate cases, which leads to underdiagnosis and underreporting of the syndrome. Misdiagnosis negates an opportunity to contribute to the prevention of future cases of TRALI.\(^5\) Transfusion-related acute lung injury is believed to be fatal in 5% to 10% of reported cases.\(^5,12\) By 2003, TRALI had surpassed ABO incompatibility reactions as the primary cause of transfusion-related mortality, as reported to the Food and Drug Administration. That same year, the National Heart, Lung, and Blood Institute produced a definition of TRALI (Table 3). The Institute’s working group built on the ALI definition that was established in 1994 by the American-European Consensus Committee.\(^5\) In an effort to reduce misdiagnosis of TRALI, a consensus conference was held in

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<th>Table 1. Former names for transfusion-related acute lung injury</th>
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<td>Pulmonary hypersensitivity reaction</td>
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<td>Allergic pulmonary edema</td>
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<td>Noncardiogenic pulmonary edema</td>
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<td>Pulmonary leukoagglutinin reaction</td>
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<th>Table 2. Acute lung injury criteria(^6)</th>
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<td>Acute onset</td>
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<td>Pulmonary artery occlusion pressure ≤ 18 mm Hg (if measured)</td>
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<td>No evidence of left atrial hypertension</td>
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<td>Bilateral infiltrates evident on chest radiograph</td>
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<td>Hypoxemic (\text{PaO}_2/\text{FiO}_2) ratio, ≤ 300 mm Hg</td>
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<td>Oxygen saturation ≤ 90% while breathing room air</td>
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Incidence
Because of misdiagnosis, the true incidence of TRALI is unclear. Older estimations generalize the incidence of TRALI at approximately 1:5,000 transfusions of blood products. Each blood product has been implicated in the development of TRALI at different rates. Newer estimates of the incidence further distinguish cases into fatal and nonfatal occurrences. In these newer estimates, fatal incidences of TRALI are estimated to occur in 1:1,300 component transfusions when all other ALI risk factors have been excluded and in 1:370 component transfusions when the other ALI risk factors are included. Nonfatal TRALI is estimated to occur in 1:2,400 component transfusions when other ALI risk factors are excluded and in 1:1,000 component transfusions when the

Toronto, Canada, in 2004. This conference was charged with setting forth a definition that allowed for broader patient inclusion, particularly patients with additional risk factors for developing TRALI and possible TRALI cases. The definitions set forth by the Toronto consensus conference are as follows:

...TRALI was defined as: 1) a new occurrence of acute onset ALI (with hypoxemia and bilateral infiltrates on chest radiograph but no evidence of left atrial hypertension), 2) not pre-existing, but 3) emerging during or within 6 hours of the end of the transfusion, and 4) having no temporal relationship to an alternative ALI risk factor. Possible TRALI included cases in which there was a temporal association with an alternative ALI risk factor.
other ALI risk factors are included.9,10 The highest incidences of TRALI involve components containing high-plasma volume (Table 5).11

**Differential diagnosis**

Accurate and rapid diagnosis of TRALI is crucial to the initiation of appropriate treatment. The differential diagnosis for TRALI includes ruling out transfusion-associated circulatory overload (TACO), anaphylactic transfusion reaction, and bacterial contamination of blood products12 (Table 6). All “suspects” in the differential diagnosis result in fever, dyspnea, cyanosis, hypoxemia, and tachycardia. Transfusion-associated circulatory overload, like TRALI, develops within minutes to hours after a transfusion and may also be characterized by tachypnea, hypertension, and pulmonary edema. In addition, TACO, like TRALI, has been documented in association with all types of blood products. Transfusion-associated circulatory overload does demonstrate overt signs of circulatory overload, such as jugular vein distention, dyspnea, rales, wheezing, or a Cheyne-Stokes respiratory pattern, which helps distinguish TACO from TRALI. Transfusion-associated circulatory overload rapidly responds to aggressive diuresis along with ventilatory support,13 which further distinguishes it from TRALI.

An anaphylactic transfusion reaction also may be characterized by bronchospasm, resulting in the following respiratory distress signs: tachypnea, wheezing, and cyanosis. The laryngeal and bronchial edema produced by anaphylactic reaction further exacerbate the respiratory distress instead of development of frank pulmonary edema, although pulmonary edema may develop if the laryngeal and bronchial edema progress unchecked. An anaphylactic reaction is also characterized by facial and truncal erythema and edema, as well as urticaria that involves the head, neck, and trunk, all of which help distinguish this condition from TRALI.

Bacterial sepsis, caused by transfusion of bacterially contaminated blood or blood products, also may result in hypotension, vascular collapse, and hemolysis. The key factor to distinguish between bacterial sepsis and TRALI is the occurrence of hemolysis, which is characteristic of bacterial sepsis. Hemolysis may be noted by hemoglobinuria and elevated serum urea nitrogen and creatinine levels.

**Cause and pathophysiologic factors**

There are at least 3 proposed mechanisms for the development of TRALI. Two of the currently proposed pathophysiologic mechanisms apply to immunocompetent patients, and the third relates to patients who are neutropenic (Figure 1).12

The first proposed mechanism suggests that TRALI is an antibody- or immune-mediated entity in which alloantibodies are activated against HLA or human neutrophil antigens found on leukocytes or in lung tissues.7 The antigen-antibody hypothesis is the oldest of the proposed mechanisms for TRALI development. The proposed antigen-antibody reaction leads to activation of granulocytes that adhere to and damage the pulmonary endothelium. The damage to the pulmonary endothelium results in increased endothelial permeability culminating in formation of pulmonary edema. In this pathogenic mechanism, the antibodies are most often found in the donated blood product. Transfusion of the blood product introduces the antibodies into the transfusion recipient and accounts for about 90% of the antibody-mediated cases of TRALI; the remaining 10% of cases seem to result from recipient antibodies reacting with donor neutrophils,14,15

The current theory is that the antibodies most frequently develop in multiparous women, particularly women who have had 2 or more pregnancies.8 Recipient-generated antibodies may be generated by previous transfusions. Patients who have received multiple transfusions have a particularly high risk of having recipient-generated antibodies. Multiple transfusions are defined as follows: (1) transfusion of more than 10 U of red blood cells or whole blood in 12 hours or less,16 (2) 15 U of blood or more in 24 hours or less,17 or (3) 8 U of red blood cells or more in 24 hours or less.18

The second proposed mechanism occurs in 2 stages, the so-called 2-hit model. The development of TRALI requires a “first hit,” which is dependent on the patient's overall health condition when the transfusion is initiated. The first hit primes or activates the neutrophils and sequesters them in the lungs. Examples of predisposing conditions include sepsis, surgery during the preceding 48 hours, massive transfusion, and trauma. These predisposing conditions seem to activate the pulmonary endothelium and further contribute to the sequestration of neutrophils in the pulmonary tissues.12 The “second hit” in this pathogenic model occurs with the transfusion of the blood product. The antibodies found in the blood product attack the previously primed neutrophils and/or compounds that modify biological responses, such as lysophosphatidylcholine compounds, tumor necrosis factor α, interferon-γ, or interleukin-18, which cause activation of the primed and adherent neutrophils.7,8,12 This activation of the neutrophils produces damage to the pulmonary endothelium, leakage from the pulmonary capillaries, and pulmonary edema—thus, TRALI. These biological response–modifying compounds are believed to accumulate over time during the storage of donated blood and blood products.

The occurrence of TRALI is rare in neutropenic patients. Transfusion-related acute lung injury has
fusion. It may be necessary to remove and replace the IV tubing to avoid further transfusion of the blood product.

Milder manifestations of TRALI—those without pulmonary edema—may be amenable to the administration of supplemental oxygen. Patients with fulminant pulmonary edema should be sedated, tracheally intubated, and mechanically ventilated. Consideration should be given to using lung protective ventilation using smaller tidal volumes with an appropriate increase in rate. On intubation, an undiluted sample of the pulmonary edema fluid should be obtained and sent to the laboratory, along with a sample of plasma obtained simultaneously, for total protein concentration determination, which will help confirm the diagnosis of TRALI. A protein ratio (edema fluid/plasma) of 0.6 or more is suggestive of pulmonary edema produced by increased endothelial permeability rather than the hydrostatic form that is associated with TACO.

In actual and suspected cases of TRALI, IV fluids should be given relatively liberally in an effort to counter the associated hypotension that frequently occurs, even though the hypotension may not respond very quickly. For TRALI and suspected fusion. It may be necessary to remove and replace the IV tubing to avoid further transfusion of the blood product.

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Figure 2. Potential algorithm for the hematologic investigation of TRALI

**TRALI Diagnostic Algorithm**

Suspected TRALI case (ALI developing during transfusion or within 6h of completion)

- Rule out cardiogenic pulmonary edema and volume overload
  - Clinical exam and chest radiograph
  - If needed, echocardiography +/- pulmonary arterial catheterization
  - If present, obtain undiluted pulmonary edema fluid from the trachea and a matched plasma sample for protein analysis

TRALI still suspected (cardiogenic pulmonary edema and volume overload ruled out)

- Keep recently transfused blood bags and send to blood bank
- Draw blood from patient and send to blood bank

Blood Bank Protocol

- Investigate blood products transfused within 4-6 hours of reaction
  - Check female donors for granulocyte and HLA antibodies
  - If female donors negative, check male donors
  - Determine HLA and neutrophil antigens of patient
  - Antibody–antigen crossmatch

TRALI cases, diuretics should never be administered because diuresis will exacerbate the severity of this manifestation of noncardiogenic pulmonary edema. In addition, as with other forms of noncardiogenic pulmonary edema, administration of corticosteroids has not demonstrated any definitive benefits.

In addition to the blood sample obtained for total protein concentration determination, a sample for complete blood cell count with differential should be sent to the laboratory. It is noteworthy that in patients in whom TRALI develops, leukopenia will be present after the syndrome develops. But, the leukopenia is transient and is seen if the blood sample is obtained very quickly after the onset of signs and symptoms, usually within 15 minutes. This transient leukopenia is a diagnostic confirmation for TRALI.

If the patient's surgical procedure and actual and anticipated blood volume loss are such that transfusion is deemed unavoidable, a second IV line should be inserted. The anesthetist must call the blood bank to report the suspected TRALI reaction and request blood products from a different donor.

On suspicion of TRALI, the blood bank should be notified immediately (Figure 2). Any further blood or blood products from the suspected donor(s) must, of necessity, be quarantined. If a sufficient supply of the suspect blood product remains, the sample should be tested for the presence of antibodies or biological response–modifying compounds to determine whether the donor should be permanently deferred as a blood donor.

Finally, if TRALI is suspected, the anesthetist must follow the institution's policies with regard to transfusion reaction. This includes obtaining a blood sample from the patient and retrieving all bags of transfused blood products, if possible, and copies of all documents pertinent to the patient's blood product exposure.

**Prevention**

The only way to absolutely avoid TRALI is to avoid blood-product transfusion. Short of this impossible prescription, the American Association of Blood Banks, in 2006, recommended strategies intended to reduce the risk of TRALI:

1. Facilities where blood is collected should implement interventions to minimize the preparation of high-volume components from donors known to be leukocyte-alloimmunized or at risk of leukocyte alloimmunization;
2. Blood transfusion facilities should work toward implementing appropriate, evidence-based hemotherapy practices in order to minimize unnecessary transfusion(s);
3. The transfusion of blood components from donors known to be leukocyte-alloimmunized or at risk of leukocyte alloimmunization should be deferred from future blood donations.

Prevention of the American Association of Blood Banks measures for plasma and whole blood have been recommended to be complete by November 2007, and measures for platelet components should be in place by November 2008. Ideally, the availability of a single laboratory test on blood products to prevent their use would significantly contribute to the prevention of future TRALI cases. Such a test has yet to be developed.

Several interventions can be undertaken to reduce the risk of TRALI. First, donors whose blood has been implicated in cases of TRALI and have demonstrated the presence of alloantibodies should likely be permanently deferred from future blood donations. Second, because of a significant correlation of blood products received from multiparous women in the development of TRALI cases, deferral of multiparous women has been suggested. The negative argument on this intervention is the large and dramatic reduction in the number of units of blood that would occur, which is estimated in the hundreds of thousands. Third, rather than permanently or completely deferring multiparous women from future blood donations, investigators have suggested blood donation from this subset of donors should not be used for whole blood, FFP, or single-donor apheresis platelets. A similar recommendation was implemented in the United Kingdom in 2003 as part of the Serious Hazards of Transfusion Scheme. Since that time, more than 90% of FFP in the United Kingdom is now derived from male donors.
Fourth, screening of donors for anti–granulocyte and anti–HLA antibodies has been suggested for identification of donors with these TRALI-implicated compounds to completely defer their donations or to direct their donation to the preparation of blood products containing minimal plasma or for products that are fractionated.23 Fifth, use of leukocyte-reduced blood products has been suggested as a measure to reduce the risk of TRALI24; leukocyte reduction is suggested to prevent complement-mediated hemolysis and complement activation. Sixth, because of the accumulation of biological response–mediating compounds in stored blood over time, “younger” or newer blood products could potentially have significantly lesser amounts—hopefully nil—of these compounds, thus reducing the risk of TRALI. Seventh, the theory has been proposed that solvent detergent–treated plasma use rather than FFP has a lesser risk for initiating TRALI because of the greatly reduced titer, if not complete removal, of causative antibodies.25,26

Finally, perhaps the simplest and most logical intervention to reduce the risk of TRALI is to use blood products only when clinically indicated, thus reducing unnecessary transfusions. Although generally not viewed as such, blood-product transfusions are essentially tissue or organ transplants with all the potential hazards incumbent in such an undertaking.

Conclusion

The lifesaving ability of blood and blood-product transfusions is undeniable. Even though the safety of these products is at the highest in history, transfusions are still risky, having potentially life-threatening consequences. Since 2003, the primary cause of transfusion-related deaths has been TRALI. Rapid recognition is essential to the initiation of appropriate treatment measures. Unfortunately, at present, prevention of this syndrome cannot be guaranteed, but there are measures that can be instituted to reduce the risk of TRALI.

REFERENCES


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