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Respiratory Distress Following Transfusion

M.S., a 48-year-old female with acute myelogenous leukemia (AML), received induction chemotherapy consisting of daunorubicin and cytosine arabinoside. On day 21 of therapy, her hemoglobin was 8 g/dl, her platelet count was 800 u/dL, and her white blood cell count was 100 mm^3. Her oncologist ordered two units of leukoreduced red blood cells and four units of leukoreduced platelets. She had no history of prior transfusion reactions; therefore, premedications were not ordered.

M.S.'s pretransfusion vital signs were temperature 98.6° F; pulse 90 and regular; respirations 14, regular, and symmetrical; and blood pressure 110/80 mm Hg. Her 24-hour intake was 2,800 ml and her output was 2,700 ml. Her heart rate and rhythm were regular, and her lungs were clear to auscultation. No peripheral edema was noted.

The RN initiated the first unit of red blood cells to run over three hours. Vital signs were assessed at the initiation of the transfusion, 15 minutes after initiation, and then hourly. Two hours after the transfusion was initiated, M.S. put her call light on and stated, "I am having problems breathing." The RN stopped the transfusion and started 0.9% normal saline at a keep-open rate. The patient's vital signs were temperature 102° F, pulse 120 and regular, respirations 30 and shallow, and blood pressure 90/70. Her oxygen saturation on room air was 85% by pulse oximetry. She had decreased breath sounds bilaterally. Her skin and mucous membranes were pale, and no peripheral edema was noted.

The RN notified the physician and blood bank. In addition to the blood reaction workup, the physician ordered

- 60% oxygen via a mask
- Chest x-ray
- Continuous IV of 0.9% normal saline at a rate to maintain the systolic blood pressure > 100 mm Hg and urine output > 100 ml/hour
- Diphenhydramine 50 mg via IV push
- Hydrocortisone 100 mg via IV piggyback injection.

The direct Coombs test was negative. The chest x-ray revealed bilateral pulmonary infiltrates without evidence of cardiac compromise or fluid overload. Based on the patient's symptoms and the negative Coombs test, the physician determined that the patient had experienced transfusion-related acute lung injury (TRALI). The patient continued to receive antihistamines, corticosteroids, and respiratory and blood pressure support until her condition was stabilized.

The solutions offered to the clinical problems posed in this column are the opinions of the authors and do not represent the opinions or recommendations of the Oncology Nursing Society, the Oncology Nursing Forum, *or the editorial staff.*

Clinical Problem Solving

Responding to this clinical challenge are Maureen Knippen, DNSc, RN, and Laurel Stark, RN, BSN. Dr. Knippen is from the Office of Compliance and Biologics Quality at the Center for Biologics Evaluation and Research of the U.S. Food and Drug Administration (FDA) in Rockville, MD. Ms. Stark is the chief operating officer of Hematology-Oncology Centers of the Northern Rockies, P.C., in Billings, MT.

How do the clinical findings of TRALI differentiate it from other transfusion reactions?

M. Knippen: The symptoms of TRALI may be indistinguishable from adult respiratory distress syndrome; however, TRALI typically manifests within one to two hours, but always within one to six hours, after transfusion of a plasma-containing blood component. The patient develops acute respiratory distress, hypotension, hypoxemia, noncardiac pulmonary edema, and fever. A chest x-ray often reveals a "white out" picture of the lungs. TRALI necessitates supplemental oxygen and, often, mechanical ventilation.

What laboratory tests can help establish the diagnosis of TRALI?

M. Knippen: Tests for TRALI should include measurement of antigranulocyte antibodies and antihuman leukocyte antibodies (HLA). TRALI has been associated with infusion of granulocyte or HLA class I antibodies, but a recent report cited a case of TRALI in which donor HLA class II antibodies were directed against the recipient's phenotype (Kopko et al., 2001). Multiparous women have a higher likelihood of HLA sensitization, which increases with each subsequent pregnancy. Donors who themselves have had multiple transfusions are at risk for developing anti-HLA or antigranulocyte antibodies.

Should TRALI be reported to anyone other than the physician and blood bank?

M. Knippen: TRALI cases should be reported to the blood center that supplied the blood component. The remaining product should be returned and tested for anti-HLA or antigranulocyte antibodies in the donor. Fatalities from TRALI should be reported to the FDA's Center for Biologics Evaluation and Research in accordance with 21 CFR 606.17(b). The FDA encourages voluntary reporting of TRALI as a serious adverse reaction to transfusions. Reports can be filed via MedWatch by phone (800-FDA-1088), fax (800-FDA-0178), U.S. Postal Service (MedWatch, HF-2, 5600 Fishers Lane, Rockville, MD 20852), or the World Wide Web (www.fda.gov/medwatch).

TRALI is a relatively unfamiliar blood reaction to many healthcare professionals. What can be done to increase awareness and educate healthcare professionals about this potentially fatal blood reaction?

L. Stark: The incidence of TRALI is unknown. This certainly is affected by the lack of knowledge regarding this syndrome. I have worked in the blood collection facility in our community and have seen the education, testing, and quality control that are required for collecting a unit of blood. I also have worked in a stem cell transplant center and administered many units of red blood cells, fresh frozen plasma, and single donor platelet products. Yet, even with this background, I was not aware of TRALI. When I consider that TRALI

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