Discussion -- Transfusion Reaction

DISCUSSION:

The onset of signs and symptoms of a transfusion reaction can be quite similar for various types/causes and must be assessed quickly and efficiently in order to rule out the most serious reactions first. Additional work up can then be performed to rule out other causes and to determine if additional precautionary steps are required for future transfusions.

The initial steps to be followed in the event of any transfusion reactions are the same.

1. Stop the transfusion and maintain IV access.
2. Perform clerical check of labels, forms and patient identification.
3. Report reaction to physician and blood bank personnel.
4. Draw a post-transfusion sample and urine sample and send it along with the unused product to the blood bank.

These four steps result in the following actions: First, preventing further infusion of a potentially harmful product and maintaining IV access that may be necessary for intervention. Second, confirming that the correct patient is receiving the correct product and allowing others to be alerted if a switch has occurred. Third, alerting the physician and the blood bank personnel of a potential problem. The physician can initiate the appropriate therapy and the blood bank can quarantine or recall associated products if warranted. Fourth, allowing laboratory evaluation patient for hemolysis.

The most serious reaction to be excluded is an acute hemolytic transfusion reaction (AHTR). This occurs is about 1 in 25,000 transfusions and is most frequently due to ABO incompatible blood infusion secondary to clerical error (McClatchey). Clinical presentation typically includes fever and chills, chest constriction, dyspnea, lumbar pain and hypotension. Examination of a post-transfusion blood sample typically reveals evidence of hemolysis and urine sample will show hemoglobinuria. Most commonly a clerical error in sample collection or patient identification during blood administration is found. The physiology of this reaction is the presence of naturally occurring antibodies in the recipient causing intravascular lysis of the donor red cells through complement activation resulting in release of cytokines and activation of the coagulation cascade and other inflammatory mediators (Cayon Transfusion 1995).

The presence of fever with dyspnea in a patient receiving a transfusion raises several important differentials to be investigated: febrile non-hemolytic transfusion reaction (FNHTR), septic transfusion reactions, transfusion related acute lung injury (TRALI) and volume overload.

FNHTRs are non life threatening reactions which typically present with fever and chills towards the end of the transfusion. Other signs and symptoms include nausea, vomiting, dyspnea, and hyper or hypotension. These reactions occur in about 1-2% of all transfusions and are more common in patients who have received multiple transfusions in the past or have had multiple pregnancies(Technical manual). It is a cytokine mediated reaction due to antibodies in the patient's plasma against antigens present on donor leukocytes. FNHTR may also be caused by infusion of cytokines released from white cells during storage of the component. Typically, the fever responds to antipyretics such as acetaminophen and rigors if present will respond to meperidine. Leukocyte reducing the blood product using filters can prevent most of these reactions , but is usually reserved for patients with recurrent FNHTRs.

Septic transfusion reactions secondary to infusion of bacterial contaminated products is a rare, potentially life-threatening event. The onset of signs and symptoms are typically rapid, occurring within 30 minutes of completion of the transfusion (Harmening). The clinical presentation includes fever, hypotension, chills, abdominal cramps, vomiting and diarrhea, shock, renal failure and DIC. The reaction is most often associated with skin flora but can be due to bacterial endotoxins of psychrophilic organisms such as Pseudomonas spp., Escherichia coli, and Yersinia enterocolitica (Harmening). The remaining product should be promptly returned to the blood bank and then forwarded on to the microbiology laboratory for visual inspection, Gram's stain and culture of residual product. The patient should also be cultured and antibiotics initiated as appropriate.

TRALI (non cardiogenic pulmonary edema) presents with fever, marked dyspnea, cyanosis, cough and hypotension (McClatchey). Chest x-ray shows patchy pulmonary infiltrates. Characteristically, the patients have normal right sided cardiac pressures and do not have physical signs of volume overload. The primary mechanism of this reaction involves donor leukocyte antibodies and recipient leukocytes producing white cell aggregates which become trapped in the pulmonary circulation. These aggregates cause changes in the vascular permeability allowing fluid to leak into the alveoli with an adult respiratory distress syndrome (ARDS)-like picture. The leukoagglutinates activate complement and generate anaphlylatoxins C3a and C5a causing release of serotonin and histamine from platelets and tissue basophils which results in aggregation of white cells and causes leukoemboli (Henry, Technical manual). A second less common mechanism can occur when the patient has leukoagglutinating antibodies directed at leukocytes in the blood component. The signs and symptoms resolve within 24 - 48 hours (unlike ARDS) and should be treated with supportive therapy as needed. TRALI typically results in hypotension rather than hypertension and will not respond to diuresis.

The most likely cause of the transfusion reaction in our patient is volume overload. Patients with volume overload usually present with dyspnea, cyanosis and increased systolic blood pressure. A chest x-ray will reveal pulmonary infiltrates due to the presence of hydrostatic or cardiogenic pulmonary edema. Although fever may be present, it is not a necessary component in volume overload. Patients with compromised cardiac or pulmonary status and/or chronic anemia with an expanded plasma volume are at risk for the complication of transfusion. Symptoms may resolve with stopping the infusion or may require diuresis and oxygen.

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