Delayed hemolytic transfusion reaction by a Kidd antibody after heart surgery: Case report and review of the literature

Vassiliki Kazakou, MD, Alexandra Kousoulakou, MD, and Euthemia Melissari, MD, Athens, Greece

Delayed hemolytic transfusion reactions (DHTRs) usually occur between 3 and 14 days after transfusion as a result of a secondary immune response, with a drop in hemoglobin level, fever, jaundice, or hemoglobinuria. There is no sure means of avoiding DHTR completely, even with the application of the most sophisticated methods.

Kidd system antibodies are difficult to detect because they often show a great deal of serologic variability and weak in vitro expression. They have been reported to cause severe immediate or delayed hemolytic transfusion reactions.

We present a case of DHTR resulting from anti-Jka after cardiac surgery, and we report the observed changes in the pattern of occurrence of DHTRs.

Clinical Summary

A 63-year-old woman was admitted to our hospital for mitral valve replacement. The patient had a history of one pregnancy and no previous history of transfusion. Her red blood cells (RBCs) typed as group A, D+. Antibodies were not detected during preoperative routine screening procedures (Gel test; DiaMed Inc, Hellas, Greece). During the operation, the patient received 2 units of compatible concentrated RBCs and 2 units of fresh frozen plasma. The postoperative period was uncomplicated. The patient was discharged on day 6 with a hemoglobin level of 12.7 g/dL.

On day 13, the patient was urgently readmitted with fatigue and hemoglobinuria. Laboratory data showed a drop of the hemoglobin level to 10.5 g/dL and a rise of the reticulocyte level to $56 \times 10^9/L$. Indirect bilirubin and lactic dehydrogenase levels increased to 2.05 mg/dL and 3857 IU/L, respectively. Serum haptoglobin value was normal. We suspected DHTR and screened for anti-RBC antibodies. Serum antibody identification procedures (Gel test; commercial panel cells) showed the presence of anti-Jka. The patient's RBCs exhibited a negative direct antiglobulin test (DAT; Gel test). The patient's pretransfusion serum was retrospectively retested: anti-Jka was not detected. The patient's RBC phenotype was Jk (a-b+), and her husband's typed as Jk (a+b+). Their son was not available for assessment. Kidd phenotyping of transfused red cells revealed Jka antigen positivity. The hemolytic reaction gradually subsided, despite the presence of antibodies in vitro.

Discussion

Individuals exposed to RBC alloantigens through transfusion, pregnancy, or transplantation may produce antibodies. Kidd antibodies are notorious for involvement in severe DHTRs. In many cases, retesting the patient's pretransfusion serum confirms that the antibody was, indeed, undetectable in the initial tests. This highlights the importance of consulting previous transfusion reports before selecting blood for transfusion.¹

Our patient had a DHTR, which was diagnosed 13 days after the transfusion of the causative RBCs. The antibody production probably occurred during pregnancy, 32 years earlier.

Mechanical hemolysis caused by extracorporeal circulation can be confused with DHTR, so physicians should be aware of distinguishing the two conditions. Typically, the DAT of patients with DHTR is positive. It is often one of the important laboratory findings supporting

From the Department of Haematology and Blood Transfusion, Onassis Cardiac Surgery Center, Athens, Greece.

Received for publication Oct 24, 2006; accepted for publication Nov 16, 2006.

Address for reprints: Alexandra Kousoulakou, MD, Department of Haematology and Blood Transfusion, 356 Sygrou Ave, 17674 Kallithea, Athens, Greece (E-mail: alexkousoulakou@yahoo.com).

J Thorac Cardiovasc Surg 2007;133:1364-5
0022-5223/\$32.00

Copyright © 2007 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2006.11.022

an immune-mediated hemolytic reaction. In our case, it was not clear why the DAT was negative. Perhaps almost all of the sensitized RBCs were rapidly cleared from the patient's circulation during the hemolytic episode and decreased to undetectable levels.

To our knowledge, this is the third reported case of DHTR resulting from anti-Jka after cardiac surgery, but it is the first case in which the development of this antibody occurred during pregnancy.

During the past 2 decades, a trend toward a decrease in the incidence of DHTRs occurred according to the Mayo Clinic's data.² A number of factors could influence the incidence of DHTRs, including changes in the average length of stay for inpatients, the average number of RBC transfusions per inpatient, and the adoption of new antibody detection systems.^{2,3} Cumulative data from 8 years (1996–2004) of Serious Hazards of Transfusion (SHOT) show that the incidence of DHTRs remains stable, accounting for 10.2% of all transfusion-related reactions. Responsible antibodies were consistent with those reported in the literature, with Kidd remaining the major cause of DHTRs (42%).⁴

Development of new screening techniques to improve the detection of extremely low levels of Kidd antibodies is mandatory, especially for patients undergoing cardiac surgery, where the transfusion requirements are increased. Consideration should be given to issuing antibody cards or similar information to all patients with clinically significant red cell antibodies.

References

1. Blood groups: In: Brecher ME, editor. Technical manual, 14th ed. Bethesda: American Association of Blood Banks; 2002. p. 327.
2. Pineda AA, Vamvakas CE, Gorden DL, Winters LJ, Moore BS. Trends in the incidence of delayed hemolytic and delayed serologic transfusion reactions. *Transfusion*. 1999;39:1097-103.
3. Schonewille H, van de Watering LGM, Loomans ED, Brand A. Red blood cell alloantibodies after transfusion: factors influencing incidence and specificity. *Transfusion*. 2006;46:250-6.
4. Serious Hazards of Transfusion annual report 2004. Manchester: Serious Hazards of Transfusion Office; 2005.

Sternal tuberculosis after sternotomy for coronary artery bypass surgery: A case report and review of the literature

Kirun Gopal, MS,^{a,c} Austin Raj, DNB,^a M. R. Rajesh, MBBS,^a S. K. Prabhu, FRCS,^a and Jayasree Geothe, MD,^b Kerala, India

Clinical Summary

A 72-year-old man presented with upper back pain for 1 month and swelling over the sternum of 4 days' duration. He had no constitutional symptoms. One year earlier he had undergone coronary artery bypass surgery with an uneventful postoperative recovery. Clinical examination revealed a fluctuant swelling over the body of the sternum. There was mild tenderness but no erythema. Radiography of the chest and thoracic spine showed normal results (Figure 1). He had a total white blood cell count of 10,000/mm³,³ with a differential count of 68% neutrophils and 30% lymphocytes, and an erythrocyte sedimentation rate of 105 mm/hour. Drainage of sternal abscess and removal of underlying possibly infected sternal wires were performed. Intraoperatively, a

large abscess cavity (3.5 × 3 cm) was detected in the body of the sternum unrelated to the sternal wires. The cavity was lined with infected granulation tissue extending to the inner table of the sternum. The granulation tissue was curetted until healthy bleeding bony surface was observed. The pectoralis major muscle was mobilized to cover the defect, and the wound was closed in layers over a suction drain. The patient had an uneventful postoperative recovery and was relieved of back pain. The curetted tissue was sent for histopathology and bacterial culture. Histology revealed a caseating granulomatous lesion with sequestrum formation consistent with tuberculous osteomyelitis (Figure 2); the routine culture was negative, whereas BACTEC culture revealed mycobacterium tuberculosis (TB). On a 4-drug antituberculous regimen, the patient is doing well at 6 months.

Dr Gopal

Discussion

TB of the sternum has rarely been reported in the literature. Primary sternal TB is relatively more common compared with sternal TB secondary to surgery. A Medline search for cases of

From the Departments of Cardiovascular and Thoracic Surgery^a and Pathology,^b SUT Hospital, Trivandrum, Kerala, India.

Received for publication Dec 14, 2006; accepted for publication Jan 8, 2007.

Address for reprints: Kirun Gopal, Add: Chathayam, Ancheril Gardens, Gandhinagar PO, Kottayam 686008, Kerala, India (E-mail: kirungopal@yahoo.com).

J Thorac Cardiovasc Surg 2007;133:1365-6

0022-5223/\$32.00

Copyright © 2007 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2007.01.022