—Case Reports—

Transient Thrombocytopenia after Incompatible Red Blood Transfusion in an Infant Girl with Autoimmune Hemolytic Anemia

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Abstract

We report on 5-month-old girl with severe autoimmune hemolytic anemia (hemoglobin 2.9 g/dl, positive direct Coombs test) in whom thrombocytopenia developed after red blood cell transfusion. The platelet count was $62.1 \times 10^4/mm^3$ on admission and rapidly fell to $6.0 \times 10^4/mm^3$ rapidly after transfusion of incompatible red blood cells with intravenous predonisolone administration. No increased hemolysis or alloimmunization was observed after transfusion. The platelet count and hemoglobin levels gradually improved over 8 weeks with corticosteroid therapy. We suspect that the thrombocytopenia in our patient was transfusion-induced.
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Key words: infant, autoimmune hemolytic anemia, thrombocytopenia, incompatible blood transfusion, Evans' syndrome

Introduction

Autoimmune hemolytic anemia is rare in children[1]. Evans' syndrome, which is characterized by autoimmune hemolytic anemia and thrombocytopenia ($<10 \times 10^4/mm^3$) of unknown caused etiology, is also rare condition in children; the prognosis is variable[2,3]. Here, we report on a 5-month-old girl with autoimmune hemolytic anemia in whom thrombocytopenia developed after incompatible red blood cell transfusion.

Patient

A 5-month-old girl was referred to our hospital because of facial pallor for 3 days before admission. We noted that her face was extremely pale and slight jaundiced. Because hematological examination revealed severe anemia and jaundice, she was admitted (**Table 1**). The family history did not show any abnormalities, including hemolytic anemia and thrombocytopenia. After an uneventful delivery at full term in a gravida 2, para 2 mother, she was a healthy baby wihtout infections during the previous 3 months. She had not been received any medications before admission. We diagnosed hemolytic anemia. We could not transfuse red blood cells immediately because of an incompatible result on cross-matching with a positive finding on direct Coombs' test against IgG. After admission, her condition worsened within 3 hours, and respiratory failure occurred despite the use of oral oxygen inhalation. After we explained her severe illness and the risk of transfusion of incompatible blood to her parents, they consented to the transfusion; we finally

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Table 1 Laboratory data on admission

Red blood cells	99×10^4 /mm^3	Total protein	6.0 /d*l*
White blood cells	17,250 /mm^3	Albumin	4.2 g/d*l*
stab	4 %	Total Bilirubin	6.0 g/d*l*
segment	71 %	Direct Bilirubin	0.8 g/d*l*
lymphocytes	24 %	Blood Urea Nitrogen	23.8 mg/d*l*
monocytes	1 %	Creatinine	0.34 mg/d*l*
Hemoglobin	2.9 g/d*l*	Aspartate amino transferase	46 IU/*l*
Hematocrit	9.0 %	Alanine amino transferase	14 IU/*l*
Platelet	62.1×10^4 /mm^3	LDH	1,530 IU/*l*
Reticulocyte	1.6 %	Creatine phoshpokinase	122 IU/*l*
C-Reactive Protein	0.7 mg/d*l*	Amylase	5 IU/*l*
Na	133 mEq/*l*		
K	5.2 mEq/*l*	Direct Coombs	Positive
Cl	97 mEq/*l*	Indirect Coombs	Negative

Table 2 Changes platelet mean value and LDH isozyme during admission

Date	Day 1	Day 2	Day 7	Day 14	Day 60
Red blood cell (10^4/μ*l*)	99				422
Hemoglobin (g/d*l*)	2.9	5.3	4.0	6.9	12.3
Reticulocyte (%)	1.6	4.8	11.5	8.8	0.6
White blood cells (/μ*l*)	17,250				7,420
Haptoglobin (mg/d*l*)	NA	2	3	4	11
D-Coombs	Positive	NA	Negative	Negative	Negative
Platelet ($\times10^4$/mm^3)	62.1	6.0	4.2	63.0	56.6
MPV (fL)	8.0	10.4	10.4	8.0	NA
Total Bilirubin (g/d*l*)	6.0	2.7	1.0	0.3	0.2
Indirect Bilirubin (g/d*l*)	5.2	2.0	0.9	0.2	0.1
LDH (IU/L) (normal: 220 ～ 430)	1,530	5,190	3,002	860	646
LDH1	NA	1,038	NA	361	NA
LDH2	NA	1,402 *	NA	353	NA
LDH3	NA	1,453 *	NA	103	NA
LDH4	NA	934	NA	26	NA
LDH5	NA	363	NA	17	NA
Treatment	Steroid 20 mg+ Blood transfusion	20 mg	20 mg	15 mg	none

NA: not available, * LDH2 < LDH3 indicates platelet consumption

transfused the least-incompatible red blood cells together with 2 mg/kg of predonisolone. The blood type of her mother, patient, and transfused blood was all A and Rh (+).

After the transfusion, her condition gradually improved. However, the next day, the platelet count fell from 62.1 to 6.0 × 10^4/mm^3. Thrombocytopenia (range, 6～9 × 10^4/mm^3) with increased mean platelet volume (MPV; 10.4 fL) continued until day 7 of admission (**Table 2**). The lactate dehydrogenase (LDH) isozymes also showed a LDH2<LDH3 pattern, suggesting platelet consumption (**Table 2**). However, she did exhibit any bleeding tendency, such as purpura. Bone marrow examination on day 7 after admission revealed moderate hypercellularity with increased numbers of erythroblasts and normal to small sized megakaryocytes without abnormal cells (**Fig. 1**). On day 7, although direct Coombs' test was negative, the platelet count remained low (**Table 2**). Anti-DNA antibody and anti-nuclear antibody were negative and values of C3, C4, and CH50 were normal. After 9 days of predonisolone therapy, the platelet count was increased with decreases in MPV to 8.0 fL along with increased hemoglobin levels.

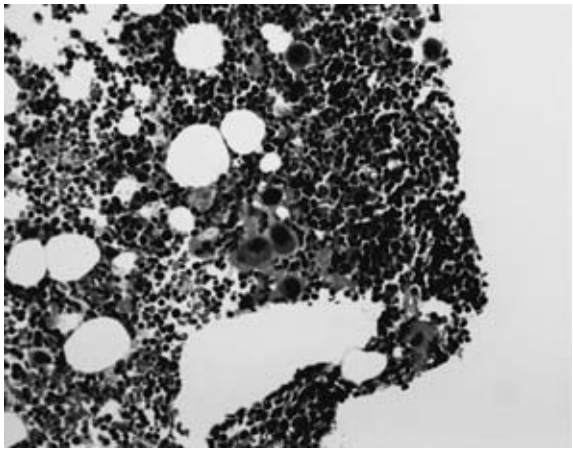

Fig. 1 Bone marrow examination: Moderate hypercellularity with increased numbers of erthroblasts and megakaryocytes was noted

During 8 weeks of corticosteroid therapy, the hemolytic and thrombocytopenic status improved and direct Coombs' test continued to be negative. At 1 year after discontinuation of predonisolone, the clinical course, including hematological status, remains uneventful.

Discussion

Transfusing blood cells to patients with autoimmune hemolytic anemia is generally inadvisable[4–11]. The reason for this contraindication is the high risk of increased hemolysis and alloimmunization after transfusion in autoimmune hemolytic anemia[12,13]. The present patient was a 5-month-old girl with direct Coombs'-positive autoimmune hemolytic anemia and, subsequently, thrombocytopenia after transfusion of red blood cells. The thrombocytopenia was accompanied by increases in MPV, the LDH3/LDH2 ratio, the LDH level **(Table 2)**, and the number of megakaryocytes. These results strongly suggest that the thrombocytopenia in our patient was caused by platelet consumption[14,15].

Complications, such as massive hemolysis, are also rare of the transfusion of incompatible blood in patients with autoimmune hemolytic anemia[6,11,12]. Hemolysis might depend on the number of antibodies on the surfaces of red blood cells. If the same phenomenon occurred in our patient's platelets, it is reasonable to conclude that the amount of autoantibody on the platelets was a factor in consumption thrombocytopenia induced by incompatible blood transfusion. However, we could not find any report on the relationship between platelet consumption and blood transfusion in autoimmune hemolytic anemia[4–13,16]. In this report, we must, therefore, consider thrombocytopenia as an additional complications of incompatible blood transfusion in patients with autoimmune hemolytic anemia.

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