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Case Report Open Access

Anti-D and -C Produced by A Type DU Patient: A Case Report

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Case Presentation

The patient is a 31-year-old woman who was hospitalized in Qinghai province because of dizziness and fatigue without an apparent cause 10 years ago. She was treated with A type Rh-positive blood at that time, and subsequently had a multiple blood transfusion history. Until 2005, she was hospitalized again. Examination of the patient showed that the blood type was Rh-negative, but the blood type was ignored. She received Rh-positive blood in a number of other hospitals.

One week ago the patient felt dizzy and had increased symptoms of fatigue with palpitations and chest tightness. Therefore, she was hospitalized in our hospital for further treatment. The physical examination showed that patient had chronic disease and anemia with pallor. Emergency laboratory testing showed the following: WBC count, 4.3×10^9/L; RBC count, 0.38×10^9/L; Hb, 15 g/L; PLT count, 3.0×10^9/L; neutrophil count, 58.70% (2.530×10^9/L); and lymphocyte count 36.000% (1.550×10^9/L).

A bone marrow smear showed decreased bone marrow myeloid hyperplasia, megakaryocyte proliferation was inhibited, and loss of scattered platelets. Pathology showed that bone marrow hyperplasia was active. The megakaryocytes had mild mature barriers and the diagnosis was "aplastic anemia." When blood was prepared for the patient, laboratory examination from our department showed that her blood type was A type with type DU. Serum antibody screening was positive.

Moreover, anti-D and -C antibodies were detected in the serum. After a matched red cell suspension was infused, the symptoms of severe anemia were relieved. At the same time, symptomatic support treatment was performed. Repeat laboratory testing revealed the following: WBC count, 2.4×10^9/L; RBC count, 1.36×10^9/L; Hb: 45.0 g/L; and PLT count, 13.0×10^9/L. The patient's general condition was good.

Blood Type Serologic Examination

Reagents

The following products were used: micro-column gel anti-human globulin card (Johnson & Johnson Biotech Corp, New Jersey, USA ,USA; Diamed Biotech Corp, [creesier], Switzerland); Rh type reagents, anti-human globulin reagent, spectrum cell, and antibody screening cells (Shanghai Blood Bio Pharmaceutical Co., Ltd., Shanghai, China); import spectrum cell 5, 6 (Immucor Company, [New Jersey, USA]); monoclonal (IgM) anti-D (German Biotest Corporation, [Dreieich,Germany]; Shanghai Blood Bio Pharmaceutical Co., Ltd.); and human source anti-D (our laboratory).

Blood type serologic method

ABO-positive and -negative serotypes and Rh serotypes, cross matching blood testing of the -human globulin micro-column, indirect anti-human globulin test (IAT), irregular antibody screening, and antibody identification testing were performed in accordance with "national clinical laboratory operation procedures" [1].

Blood type serologic results

1. **Patient blood type:** Type A ccDuee
2. **Direct anti-human globulin test:** negative
3. **Antibody screening results:** antibody screening cells 1, 2, and 3 were positive.
4. **RhD^U confirmation test:** Monoclonal (IgM) anti-D serum from two different sources, human anti-D serum and red blood cells of patients was tested by water bath anti-globulin reaction at room temperature and 37.
5. **The room temperature brine method result:** Red blood cells of patients did not agglutinate with and No.1 and 3 anti-D, but agglutinated strongly with No. 2 anti-D. 37 water bath anti-globulin method results: Red blood cells of patients did not agglutinate with No.1 anti-D, but agglutinated strongly with No.2 anti-D and weakly with No.3 anti-D (Table 1).

Anti- D	No.1 anti-D	No.2 anti-D	No.3 anti-D
Patient red blood cells (brine method)	-	3+	-
Patient red blood cells (37 anti-globulin method	-	3+	±
Remarks: Serum of No.1 was from Biotest [New Jersey, USA]; Serum of No.2 was from Shanghai Blood Bio Pharmaceutical Co., Ltd.; Serum of No.3 was the human source of anti-D.			

Table 1: RhD^U confirmation test.

Spectral cell response

The antibody specificity was identified using the spectrum, and the response of a group of cells was calculated. The response of Nos.7 and 9 cells were negative. Agglutination of Nos.1, 3, 4, and 8 cells presented as 4+. Agglutination of Nos. 2, 5, 6, and 10 presented as 3+. The serum anti-D antibodies were determined using the negative exclusion method, and anti-C and anti-E may also exist (Table 2).

No.	Rh-hr					Kidd		MNSs					Duffy		Diego		Kell		Lewis		P	DO		Yt			
	D	C	E	c	e	JkA	Jkb	M	N	S	s	Mur	Fya	Fyb	Dia	Dib	K	k	Lea	Leb	P1	DOA	DOB	Yta	Ytb	BR	AG
1	+	+	0	0	+	+	+	+	+	0	+	0	+	0	0	+	0	+	0	+	+	0	+	+	0	-	4+
2	+	0	+	+	0	+	0	0	+	0	+	0	+	0	0	+	0	+	0	+	+	0	+	+	0	-	3+
3	+	+	0	+	+	+	+	+	+	+	+	0	+	0	+	+	0	+	0	+	+	0	+	+	0	-	4+
4	+	+	+	+	+	+	0	+	0	0	+	0	+	0	0	+	0	+	+	+	+	0	+	+	0	-	4+
5	+	0	+	+	+	0	+	+	+	0	+	0	+	0	0	+	0	+	0	+	+	0	+	+	0	-	3+
6	+	0	0	+	+	0	+	0	+	0	+	0	+	0	0	+	0	+	0	+	0	0	+	+	0	-	2+
7	0	0	0	+	+	+	+	+	+	0	+	+	+	0	0	+	0	+	+	0	+	0	+	+	0	-	-
8	+	+	+	0	0	+	+	+	+	0	+	0	+	0	0	/	0	+	0	0	+	/	/	/	/	-	4+
9	0	0	0	+	+	+	+	0	+	0	+	0	+	0	0	+	0	+	+	0	0	0	+	+	0	-	-
10	+	0	+	+	+	+	0	+	+	+	+	0	+	0	0	/	+	+	/	+	0	/	/	/	/	-	3+

Table 2: Results of spectrum of cell responses by serum and antibody identification.

Nos. 5 and 6 spectral cell produced by Immucor Company [New Jersey, USA] reacted with patient serum: Agglutination of patient serum and No.5 cell (Ccdee) presented as 4+, but agglutination of patient serum and No.6 cell (ccdEe) presented as negative, which confirmed the identification that there was anti-C in patient serum, but not anti-E (Table 3).

Spectral cell	No.5 cell (C c d e e)	No.6 cell (c c d E e)
Serum (brine method)	4+	-
Serum (anti-globulin method)	4+	-

Table 3: Response results of patient serum and Nos.5 and 6 spectral cells.

The serum antibody titer was determined (anti-D=512, and anti-C=256).

Transfusion therapy

Patients were treated with A or type O blood with ccdee because of the relatively high frequency of Rh blood type ccdee antigen. At the same time, E antigen was also avoided by blood transfusion in patients, so that immunity produced anti-E in the patient.

Discussion

Most of the Rh antibody is IgG antibody, produced by the immune pathway, such as blood transfusion and pregnancy. The patient failed to identify as Rh (D)-negative blood group during hospitalization in 1996 and Rh-positive blood was repeatedly infused. Because clinical data cannot always be obtained, and blood transfusion is often were without hospitalization records, the actual conditions could not be determined. Until August 2009, the patients were treated in our hospital.

The diagnosis was confirmed by the detection of the blood type in the reference department ($CcD^{U}ee$ type A blood type with anti-C and -D antibody. After treatment with matched ccdee type A or C blood, the patient's condition improved.

The Rh blood type system is one of the most polymorphic blood type systems known. Based on the D antigen of red blood cells, the Rh blood type system can be classified as Rh-positive and -negative. The quality and changes of the D antigen in the red blood cell may result in a weak D phenotype and the variation of the partial D phenotype.

There is no difference between a weak D phenotype and D antigen. With the lack of normal D antigen expression, partial D phenotype can also produce anti-D antibody through the immune response of D antigen-positive red blood cells [2]. This patient had anti-D antibody, which may be derived from the latter reason and needs further investigation.

In clinical blood transfusion treatment, the Rh-blood type system can cause a hemolytic transfusion reaction, neonatal hemolytic disease, and medical autoimmune hemolytic anemia [3]. Therefore, for the blood transfusion involving rare blood type patients with anti-D and other irregular antibodies caused by iso-immunization, matched blood or washed red blood cells must be used to prevent the occurrence of immune transfusion reactions [4,5].

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