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Case report

Identification of anti-c, E alloantibodies with autoantibodies mimicking anti-c, E alloantibodies

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ABSTRACT

Objectives: Autoantibodies mimicking alloantibodies (referred to as mimicking antibodies) are a type of specific antibody that reacts with all red blood cells, but exhibits a stronger reaction with red blood cells expressing the target antigens. This study aimed to explore immunohematologic methods for identifying mimicking antibodies, autoantibodies and alloantibodies, and to formulate safe transfusion strategies based on the results.

Methods: ABO, Rh blood types and direct antiglobulin test were determined using the tube saline method. Antibody detection and identification were performed using tube saline and anti-human globulin microcolumn gel method with two sets of panel red blood cells. The specificity of antibodies was confirmed through absorption elution test, antibody dilution and titer integral method.

Results: The study identified the presence of both anti-c and anti-E alloantibodies as well as anti-c and anti-E mimicking antibodies in patient samples. Based on these findings, suitable A-type CCDee donors can be selected for the patient, while avoiding both mimicking antibodies and alloantibodies.

Conclusion: When mimicking antibodies, autoantibodies and alloantibodies are present in patients, either separately or simultaneously, antibody identification can be challenging. Accurate determination of the type and specificity of antibodies is essential for developing safe transfusion strategies in clinical practice.

1. Introduction

To improve the efficiency of clinical blood transfusion and avoid hemolytic transfusion reactions, screening for unexpected red blood cell (RBC) antibodies has become a routine pre-transfusion test. These antibodies generally be divided into autoantibodies and alloantibodies [1]. Alloantibodies are directed against RBC antigens absent in the individual and often develop following exposure to allogeneic RBCs through blood transfusion or pregnancy. In contrast, RBC autoantibodies arise from disruption of self-immune tolerance, triggered by factors such as drugs, tumors, infections or autoimmune diseases [2,3]. Autoantibodies frequently display panreactivity against all RBCs including self-RBC due to their lack of antigen specificity.

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In addition, there is a type of autoantibodies mimicking alloantibodies (referred to as mimicking antibodies) between autoantibodies and alloantibodies, reacting with all RBCs, but exhibiting a stronger reaction with RBCs expressing the target antigens. These antibodies may develop due to antigen exposure or breakdown of immune tolerance, regardless of itself presence of the target antigens [4–6]. Paul et al. discovered that a patient with the phenotype (CDe/CDe) had eluates from his RBCs that contained anti-c, E antibodies [4]. Jeremy W et al. identified anti-Jk^{a} and anti-f mimicking antibodies in different patients [6]. Y Feng et al. summarized 32 global articles published between 1977 and 2019 on mimicking antibodies, and found that antibodies against the Rh system were the most common [5]. Identifying such mimicking antibodies poses challenges in immunohematologic evaluations, particularly in cases where individuals simultaneously have both alloantibodies and mimicking antibodies targeting the same antigens. In this study, we present a rare case of a patient producing both anti-c, E alloantibodies and anti-c, E mimicking antibodies.

2. Materials and methods

2.1. Patient information

A 72-year-old male patient presented with unexplained fatigue on February 29, 2024, and was subsequently diagnosed with gastric malignancy at a local hospital. He has no known family history of autoimmune conditions or other significant medical conditions. On March 2, 2024, the patient received a transfusion of 3 units of leukocyte-depleted RBCs (RhCcEe phenotype unknown). Following this, on March 25, 2024, he underwent one cycle of comprehensive chemotherapy consisting of a SOX regimen (oxaliplatin on Day 1 + tegafur from Day 1 to Day 14 orally) combined with cetuximab at a provincial tertiary hospital. On May 20, 2024, due to the need for blood preparation prior to planned gastric cancer surgery, an antibody screening test was performed, which returned a positive result. After obtaining informed consent from the patient, blood samples were collected by the clinical hospital and sent to our laboratory for antibody identification.

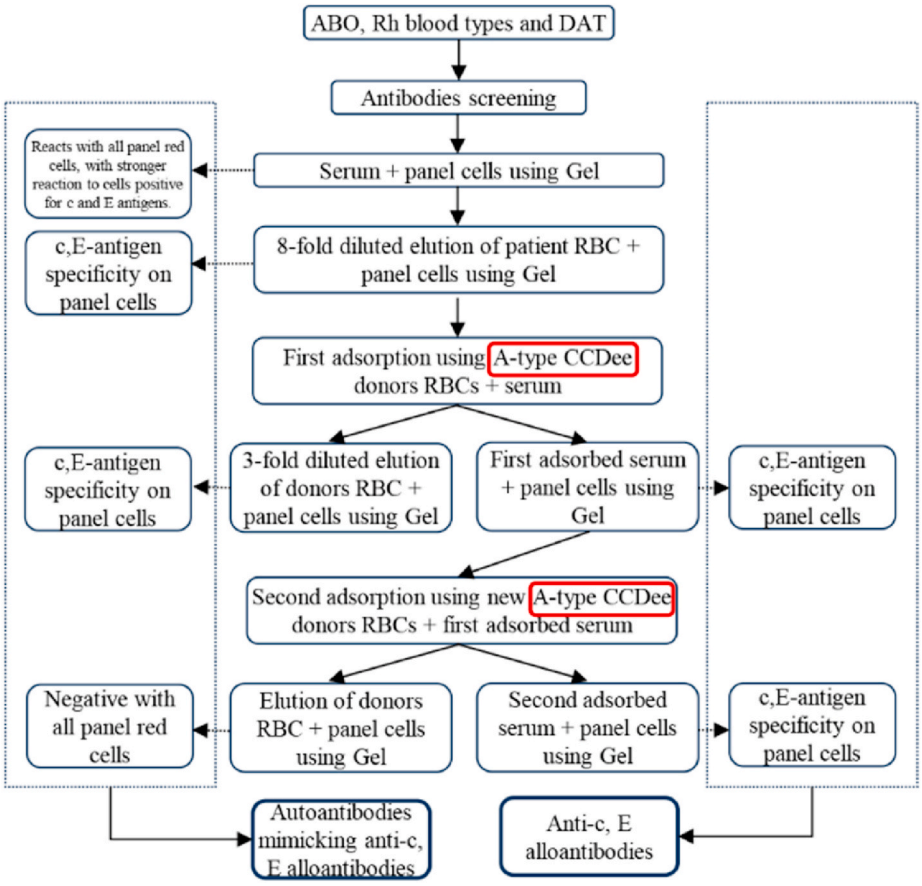

Fig. 1. Antibody identification flow chart.
Note: DAT direct antiglobulin test, Gel anti-human globulin microcolumn gel.

2.2. Reagents and Instruments

Immunoglobulin M (IgM) monoclonal anti-A/B, anti-D, anti-C, anti-c, anti-E, anti-e, A1 and B reagent RBCs, anti-human globulin (AHG), anti-IgG, anti-C3d, acid elution reagent, antibody screening cells (Shanghai Blood Biopharmaceutical Co., Ltd., batch numbers: 20230301, 20231803, 20233001, 20223102, 20233201, 20233301, 20245317, 20225001, 20225101, 20235202, 20232101, 20247016); panel cells (Reagens/Sanquin, batch numbers: 732405, 8000460525); low ionic anti-human globulin card, ABO blood type forward/reverse typing card (DiaMed, batch numbers: 50531.96.38, 50093.34.22); Rh blood type antigen detection card (Jiangsu Zhongji Wantai Biopharmaceutical Co., Ltd., batch number: 103231101). Instruments utilized included the Heraeus-Labofuge 200 blood typing centrifuge; Julabo MB-19 water bath; DiaMed-ID Incubator 37 SI blood typing and crossmatching centrifugal incubator; DiaMed-ID Centrifuge 12 SII dedicated centrifuge.

2.3. Blood serology testing

2.3.1. Overall process

The overall process of blood serology testing is depicted in Fig. 1.

2.3.2. Blood typing and direct antiglobulin test

A 3 % red blood cell suspension was prepared from the patient and subjected to ABO and Rh blood typing using the tube saline method. Additionally, a direct antiglobulin test (DAT) was performed.

2.3.3. Serum and eluate antibody identification

Sanquin panel cells were used with the globulin microcolumn gel method, and Reagens panel cells were used with the tube saline method to identify free antibodies in the patient's serum. Eluate from saline-washed patient's RBCs was performed, with serial dilutions used to determine antibody titers against mixed screening cells [7,8], guiding subsequent antibody specificity identification [9]. The Sanquin panel and Reagents panel both consist of O-type red blood cells, from 16 to 11 individuals respectively, each representing various antigen components from different blood group systems. The antibody identification is based on the pattern of reactivity. Negative exclusion: if the test result for a given cell is negative, any antibody corresponding to a positive antigen on that cell can be excluded. Positive possibility: if the test result for a given cell is positive, the antibody corresponding to any positive antigen on that cell is a potential match.

2.3.4. Adsorption-elution test

Packed RBCs from A-type CCDee blood donors were used for the adsorption-elution test. Briefly, donors' RBCs were incubated with patient serum at a ratio of 3:1, adsorbed at 37 °C for 1 hour, followed by acid elution of adsorbed RBCs [10]. The eluate was collected for antibody titer determination and identification via the anti-human globulin microcolumn gel method. The adsorbed serum was further subjected to a second adsorption using a new aliquot of packed RBCs from A-type CCDee blood donors. The second absorption serum was identified antibody specificity by anti-human globulin microcolumn gel method.

2.3.5. Antibody titer determination and scoring method

Using A-type ccDEE cells, antibody titers were determined from the patient's original serum and serum post first and second adsorptions using anti-human globulin microcolumn gel method [11]. The scoring standards corresponding to strength of agglutination are: 4+ (score 9), 3+ (score 7), 2+ (score 5), 1+ (score 2), w+ (score 1), and 0 (score 0) [8].

3. Results

3.1. Blood typing and DAT

The patient's blood type was A Rh-CCDee with a positive DAT, showing positivity for both anti-IgG and anti-IgG + C3d, but negativity for anti-C3d.

3.2. Serum antibody screening

Original serum antibody screening indicated the presence of saline antibodies and autoimmune antibodies, with alloantibodies not being excluded (Table 1).

Table 1
Result of patient serum reaction with screening cells (Shanghai, 20247016).

Ps	1	2	3	Ctl
IS	1+	0	1+	0
Gel	4+	2+	4+	2+

Note: IS immediate spin, Gel anti-human globulin microcolumn gel, Ctl patient RBCs.

3.3. Identification of autoantibodies mimicking anti-c, E alloantibodies

The patient's serum exhibited full agglutination with panel red cells, but showed a stronger reaction with RBCs expressing the c and E antigens (Table 2), suggesting the presence of anti-c, E alloantibodies alongside non-specific autoantibodies or potentially only mimicking anti-c, E antibodies. After dilution of the patient's RBC eluate 8-fold, the reaction pattern matched the characteristics of anti-c, E antibodies (Table 2). Given the patient's Rh phenotype of CCDee, this confirmed the presence of anti-c, E mimicking antibodies bound to the patient's RBCs. Absorption and elution tests using A-type CCDee donor RBCs were performed on the patient's serum. After dilution of the donor RBC eluate 3-fold, the reaction pattern matched the characteristics of anti-c, E antibodies too (Table 2), further confirming the presence of anti-c, E mimicking antibodies in the patient.

3.4. Identification of anti-c, E alloantibodies

The tube saline method for antibody identification revealed IgM anti-E alloantibodies in the patient's serum (Table 3). The patient's serum was adsorbed twice with A-type CCDee donor RBCs. The second absorption RBC eluate tested negative for antibodies, indicating that the mimicking antibodies had been completely adsorbed. Post-second absorption, the antibody pattern in the serum was consistent with anti-c, E (Table 2), suggesting the presence of anti-c, E alloantibodies that were unadsorbed by CCDee donor RBCs (Fig. 1).

3.5. Antibody titer score identification

To verify the presence of anti-c, E alloantibodies in the patient's serum using another method, the serum was adsorbed twice with A-type CCDee donor RBCs, with a known anti-D serving as a control. The titer of anti-c, E in the serum pre- and post-each absorption, and anti-D as well as in the control, were measured using A-type ccDEE cells. Before absorption, the titer of anti-c, E was 128 with a score of 66. Post-second absorption, it decreased to 16 with a score of 33 (Fig. 2), but the anti-D positive control titer decreased from 256 to 0, with its score decreasing from 74 to 0. The slight decrease in the patient's titer and score post-absorption could be attributed to dilution during absorption and elution. This test again confirmed the presence of anti-c, E alloantibodies in the patient's serum which were not adsorbed by CCDee RBCs.

3.6. Blood transfusion and follow-up results

The patient underwent radical gastrectomy for gastric cancer on the afternoon of May 21. Preoperative hemoglobin level was 104 g/L. The blood transfusion department prepared 2 units of A + RhCCDee red blood cells. The total intraoperative blood loss was 50 ml, and no allogeneic red blood cells were transfused. The patient was discharged on May 29, and no blood transfusions were administered during the hospital stay. Unfortunately, follow-up information on the patient could not be obtained thereafter. If blood transfusion is required in the future, it is recommended to transfuse A + RhCCDee red blood cells. Close monitoring of the transfusion process is necessary, and biochemical indicators should be tested post-transfusion to assess for hemolysis. Additionally, changes in hemoglobin levels should be monitored to evaluate transfusion effectiveness. It is advised that the patient regularly test for anti-c, E mimicking antibodies titers to monitor the persistence of these antibodies.

4. Discussion

The production of alloantibodies is mainly due to previous exposure to exogenous RBC antigens, which may be generated through processes such as previous blood transfusions, pregnancies or transplants. The production of autoantibodies may occur because pathogenic antigens (bacteria, viruses, etc.) share certain molecular structures with self-components, leading to an immune response that cross-reacts with self-antigens. Alternatively, it may be that certain infectious can expose hidden self-antigens, prompting the immune system to produce autoantibodies against these newly exposed antigens [12,13]. This "traditional sense" of autoantibodies is often detected first as specific antibodies, followed by the discovery that patients also express the corresponding antigens. Antibodies that possess characteristics of both alloantibodies and autoantibodies are called autoantibodies mimicking alloantibodies, which not only target antigens on RBCs in a manner akin alloantibodies but also exhibit properties of autoantibodies targeting all RBC, including self-RBC antigens [4,14]. Identifying these mimicking antibodies often requires multiple serological tests. Absorption-elution tests are powerful tools for distinguishing between alloantibodies and mimicking antibodies, and multiple absorption-elution processes can differentiate between multiple mixed antibodies [10,15,16].

In this case, the patient has a recent history of blood transfusion, suggesting probable alloantibody production. However, due to a lack of post-transfusion follow-up, it's uncertain if a hemolytic transfusion reaction occurred [17]. The saline-reactive anti-E antibody identified in the serum is related to the recent transfusion of the alloimmunization. The patient's serum showed reactivity with all panel red cells but exhibited an anti-c, E pattern, suggesting the presence of anti-c, E alloantibodies alongside non-specific autoantibodies or potentially only mimicking anti-c, E antibodies. Furthermore, the antibody characteristics identified through the patient RBCs elution test were anti-c, E specificity, and the patient's Rh typing was CCDee, confirming the adsorption of anti-c, E mimicking antibodies on the patient RBCs. Using A-type CCDee donor RBCs for absorption-elution tests with the patient's serum revealed the absorption of anti-c, E antibodies, further confirming the presence of anti-c, E mimicking antibodies in patient's serum. Absorbing the patient's serum twice with A-type CCDee donor RBCs still showed the presence of anti-c, E specific antibodies, with only a slight

Table 2
Results of patient serum and eluate reaction with spectral cells (Sanquin,8000460525).

	Rh-hr	Rh-hr						Kell						Duffy		Kidd		Lewis		P	MNSs				Luther		Xg	Ps	Pel*8	Del*3	2Ab-Ps
		C	D	E	c	e	C^w	K	k	Kp^a	Kp^b	Js^a	Js^b	Fy^a	Fy^b	Jk^a	Jk^b	Le^a	Le^b	P_1	M	N	S	s	Lu^a	Lu^b	Xg^a	Gel Ll	Gel	Gel	Gel
1	$R_1\ ^wR_1$	+	+	0	0	+	+	0	+	0	+	/	+	+	0	0	+	0	+	+	+	0	+	+	0	+	0	1+	0	0	/
2	R_1R_1	+	+	0	0	+	0	+	0	0	+	0	+	+	0	+	0	0	+	0	0	+	0	+	0	+	0	1+	0	0	0
3	$R_2\ R_2$	0	+	+	+	0	0	0	+	0	+	/	+	0	+	+	+	+	0	+	+	+	+	0	0	+	+	4+	2+	2+	/
4	R_0	0	+	0	+	+	0	0	+	0	+	/	+	+	0	+	0	0	+	+	+	+	0	+	+	+	/	3+	1+	$1+^s$	2+
5	r ' r '	+	0	0	0	+	0	0	+	0	+	/	+	0	+	+	+	0	+	+	+	0	+	+	0	+	+	1+	0	0	0
6	r '' r ''	0	0	+	+	0	0	0	+	0	+	/	+	+	+	+	0	0	0	+	+	0	+	+	0	+	+	4+	3+	3+	/
7	rr	0	0	0	+	+	0	0	+	0	+	/	+	0	+	+	0	+	0	+	+	0	+	+	0	+	0	3+	1+	2+	2+
8	rr	0	0	0	+	+	0	+	0	0	+	/	+	0	+	0	+	0	+	+	0	+	0	+	0	+	0	3+	$1+^w$	2+	/
9	rr	0	0	0	+	+	0	+	+	0	+	/	+	+	0	+	+	0	0	+	+	+	+	0	0	+	0	3+	1+	2+	/
10	rr	0	0	0	+	+	0	0	+	0	+	/	+	+	+	+	0	0	0	+	0	+	0	+	+	+	+	3+	$1+^s$	2+	/
11	R_zR_1	+	+	+	0	+	0	0	+	0	+	/	+	0	+	0	+	+	0	+	+	0	+	+	0	+	+	4+	1+	2+	$2+^w$
12	R_zR_2	w	+	+	+	0	0	0	+	0	+	/	+	+	0	+	+	0	+	0	+	0	+	+	0	+	/	4+	2+	$2+^s$	/
13	r $'^w$ r '	+	0	0	+	+	+	0	+	0	+	/	+	+	+	+	0	+	0	+	+	+	+	+	0	+	0	3+	±	±	/
14	$R_2\ R_2$	0	+	+	+	0	0	0	+	0	+	/	+	0	+	+	0	0	+	0	0	+	0	+	0	+	+	4+	2+	3+	/
15	R_1R_1	+	+	0	0	+	0	0	+	+	+	0	+	+	+	0	+	+	0	+	+	0	+	0	0	+	+	2+	0	0	/
16	rr	0	0	0	+	+	0	0	+	0	+	/	+	+	0	0	+	+	0	+	+	+	0	+	0	+	+	3+	$2+^w$	2+	/
Ctl																												2+	3+	3+	/

Table 2. **Ps** shows weak to moderate reactivity (1+ to 2+) with cells 1, 2, 5, and 15 (negative c and E antigens), and strong reactivity (3+ to 4+) with the remaining cells (positive c and/or E antigens). **Pel*8** and **Del*3** show identical reactivity patterns, with no reaction to cells 1, 2, 5, and 15 (negative c and E antigens) and reactivity with the remaining cells (positive c and/or E antigens). The reaction is weaker for single-positive c or E antigens and stronger for double-positive c and E antigens. **2Ab-Ps** does not react with cells 2 and 5 (negative c and E antigens), but reacts with cells 4 and 7 (positive c antigen) and cell 11 (positive E antigen). According to the negative exclusion and positive possibility methods, no other antigens were found to match the above conditions. **Note**: Ps patient serum, Pel*8 dilution of the patient RBC eluate with 8-fold, Del*3 dilution of the donor RBC Absorption with patient serum eluate with 3-fold, 2Ab-Ps patient serum after the second absorption, Ctl patient RBCs.

Table 3
Result of patient serum reaction with spectral cells (Reagents, 732405).

	Rh-hr	D	C	E	c	e	C^W	K	k	M	N	S	s	P_1	Le^a	Le^b	Fy^a	Fy^b	Jk^a	Jk^b	Di^a	Kp^a	Kp^b	Lu^a	Lu^b	IS
1	R_1R_1K+	+	+	0	0	+	0	+	0	+	+	0	+	+	0	0	+	+	+	+	0	0	+	0	+	0
2	$R_2\ R_2$	+	0	+	+	0	0	0	+	+	0	0	+	0	+	0	+	+	+	0	0	0	+	0	+	1+
3	r ' r *	0	+	0	+	+	0	0	+	0	+	0	+	+	0	+	0	+	+	0	0	0	+	0	+	0
4	r '' r	0	0	+	+	+	0	0	+	0	+	+	+	0	0	+	0	+	+	+	0	0	+	0	+	1+
5	rrK+	0	0	0	+	+	0	+	+	+	+	0	+	+	0	+	+	0	+	0	0	0	+	+	+	0
6	R_0r	+	0	0	+	+	0	0	+	+	0	+	+	+	0	+	+	0	+	+	0	0	+	0	+	0
7	$R_1R_1^W$	+	+	0	0	+	+	0	+	+	0	+	0	+	0	+	+	+	+	+	0	0	+	0	+	0
8	Rr	0	0	0	+	+	0	0	+	+	+	+	0	0	+	0	+	0	0	+	0	0	+	0	+	0
9	R_1R_2	+	+	+	+	+	0	0	+	+	+	0	+	+	0	+	+	+	0	+	+	0	+	0	+	1+
10	R_1R_1	+	+	0	0	+	0	0	+	+	0	+	+	+	0	+	0	+	0	+	0	+	+	0	+	0
11	rr	0	0	0	+	+	0	0	+	0	+	+	0	0	+	0	0	+	+	+	0	0	+	0	+	0
Ctl																										0

Table 3. **The serum** reacts with cells 2, 4, and 9 (positive E antigen), but does not react with the remaining cells (negative E antigen). According to the negative exclusion and positive possibility methods, no other antigens were found to match the above conditions. **Note:** IS immediate spin.

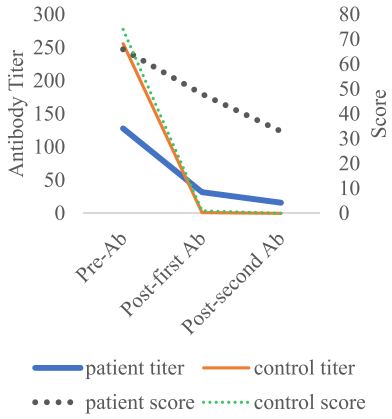

Fig. 2. The titer and integral of anti-c, E before and after 2nd absorption.

decrease in anti-c, E titers, simultaneously proving the existence of anti-c, E alloantibodies. A limitation of this study is the inability to use absorption-elution tests with rare CCEE cells to investigate potential complex anti-cE antibodies in the patient's serum.

The purpose of distinguishing between alloantibodies and mimicking antibodies is for transfusion safety. For patients with mimicking antibodies, various factors should be considered when transfusing blood, whether to choose blood type-matched with the patient or to avoid mimicking antibodies remains a topic of debate [18]. In this case, subsequent blood transfusions for the patient are relatively straightforward because both alloantibodies and mimicking antibodies target the same c, E antigens, allowing for the choosing of CCDee blood for transfusion, as the patient Rh typing is CCDee, eliminating concerns about introducing new antigens. If mimicking antibodies target self-antigens, transfusions should steer clear of these antigens to prevent immune reactions. However, this approach, while necessary, risks triggering the production of new alloantibodies, such as a patient whose Rh type is ccDEE but has anti-E mimicking antibody [6], suggesting the need to use ccee RBCs to avoid the potential immune response caused by anti- E mimicking antibody. Nevertheless, this strategy also poses the risk of stimulating the production of anti-e alloantibodies [5], presenting a dilemma that complicates clinical decisions.

Some researchers suggest that anemic patients with mimicking antibodies should avoid blood transfusions if possible. When transfusions are unavoidable, temporarily avoiding blood transfusions with mimicking antibodies specificity may be considered. If finding RBCs negative for mimicking antibodies proves too difficult, ignoring mimicking antibodies and adopting a minimal incompatibility transfusion strategy may be an option, although the risk of delayed hemolytic reactions should be considered [19]. When autoantibodies are present in the patient, the use of immunosuppressive agents can be considered to suppress the antibodies' destruction of the patient's RBCs. Additionally, while adopting a minimal incompatibility transfusion strategy, the use of immunosuppressive agents can also be considered to prevent the occurrence of hemolytic transfusion reactions.

There are several different theories regarding the causes of mimicking antibodies production, including nonspecific adsorption of alloantibodies, simultaneous occurrence of autoimmunity and alloimmunity, B cell regulatory imbalance, infection-related factors and drug-induced mechanisms. Drug-induced factors, comprising about 48 % of cases, involve medications like anti-tumor agents, antibiotics, antivirals, and anti-inflammatory drugs, possibly due to drug-related immunological mechanisms and biochemical modifications [5]. Some scholars propose that mimicking antibodies are transition of the production of specific alloantibodies in response to external antigen stimulation [20–22]. Notably, these mimicking antibodies often emerge within two weeks following a blood transfusion, during the initial immune response to RBC alloimmunity. In this case, the patient received a transfusion of 3 units of leukocyte-depleted RBCs (RhCcEe phenotype unknown) three months prior, which may have introduced foreign RBC antigens, potentially provoking the production of alloantibodies. The patient subsequently developed an immune response, as evidenced by the presence of both alloantibodies and mimicking antibodies against the same c and E antigens. Continuous follow-up of the patient will help monitor changes in the patient's mimicking antibodies over time [11]. Therefore, non-ABO phenotyping, particularly for RhCcEe and other clinically significant antigens (such as Kell, Kidd, and Duffy), plays a crucial role in preventing alloantibody production and ensuring compatibility for patients with complex transfusion needs. By addressing this issue, we aim to enhance transfusion safety and clinical practice.

CRediT authorship contribution statement

Kangle Feng: Project administration. **Xinyu Huang:** Formal analysis. **Xianguo Xu:** Validation. **Xiaodi Shi:** Supervision.

Ethics approval and consent to participate

This study was reviewed and approved by [Ethics Committee of Shaoxing Central Hospital] with the approval number: [2024-024], dated [2024.7.15].

Consent for publication

The manuscript has not been previously published and is not being concurrently submitted elsewhere. All authors have agreed on the contents of the manuscript.

Competing interests

The authors declare that they have no competing interests.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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