

CASE REPORT

A Rare Case of Anti-Kp^a Antibody Identification in a Chinese Patient

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SUMMARY

Background: Here we present a rare case of anti-Kp^a alloantibody identified in a Chinese individual, along with a literature review.

Methods: An antibody identification test was conducted to clarify the antibody specificity. To further characterize this antibody, we assessed the reactivity of the patient's serum with relative antigen-positive red blood cells at different test phases. We also investigated the sensitivity of the antigen to DTT and papain. We separated the patient's own RBCs from the transfused ones using capillary centrifugation and performed direct antiglobulin test and Kell blood group antigen detection.

Results: The patient's Kell phenotype was Kp(a-b+), and an IgG-specific anti-Kp^a antibody was identified, resulting from the transfusion of Kp(a+b+) allogeneic red blood cells (RBCs). The Kp^a antigen on RBCs was susceptible to 0.2M DTT but not to papain.

Conclusions: This case highlights the presence of an anti-Kp^a antibody in the Chinese population, suggesting that a reassessment of the frequency of this antigen in this demographic is warranted.

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KEYWORDS

anti-Kp^a antibody, Kell blood group system; low-frequency antigens

INTRODUCTION

The Kp^a antigen (KEL3), a rare red blood cell antigen within the Kell system, is present in less than 2% European ethnicity and is not present in people of African or Japanese ethnicity [1]. It is resistant to the effects of enzyme treatment but is sensitive to treatment with dithiothreitol and acid. Anti-Kp^a, typically an IgG antibody, is acquired through exposure to Kp^a antigen-positive red blood cells during pregnancy or transfusion and has been implicated in both acute and delayed hemolytic transfusion reactions [2,3]. Here we present a rare case of anti-Kp^a alloantibody identified in a Chinese individual, along with a literature review.

Table 1. Antibody identification profile and identification results.

	Rh-hr		Kell						Duffy		Kidd		Lewis		P	MNS				Luther		Xg	IAT															
	C ^w	f	V	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	Xg ^b														
1	+	/	/	0	+	0	+	/	+	+	0	0	+	+	+	+	+	+	0	0	0	+	+	+	0	0												
2	0	/	/	0	+	0	+	/	+	0	+	0	+	+	0	+	0	+	0	+	0	0	+	/	0	0	0	0										
3	0	/	/	0	+	0	+	/	+	+	+	+	+	+	0	0	+	0	+	0	+	0	+	+	+	0	0	0	0									
4	0	/	/	0	+	0	+	/	+	+	+	+	+	+	0	+	0	+	0	+	0	+	+	/	0	0	0	0	0	0								
5	0	/	/	0	+	0	+	/	+	+	+	+	+	+	0	+	0	+	0	+	0	+	+	+	+	+	0	0	0	0								
6	0	/	/	0	+	0	+	/	+	+	+	+	+	+	0	+	0	+	0	+	0	+	+	+	+	+	0	0	0	0								
7	0	/	/	+	0	+	+	/	+	0	+	+	+	0	+	0	+	0	+	0	+	0	+	0	0	0	0	0	0	0								
8	0	/	/	0	+	+	+	0	+	+	+	0	+	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+	+							
9	0	/	/	+	+	0	+	0	+	+	+	0	+	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+	+							
10	0	/	/	+	+	0	+	0	+	+	+	0	+	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+	+							
11	0	/	/	+	+	0	+	/	+	+	+	+	+	0	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+							
12	0	/	/	0	+	+	+	/	+	+	0	+	+	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+	+							
13	0	/	/	+	+	0	+	/	+	+	+	+	+	0	+	0	+	0	+	0	+	0	+	0	0	0	0	0	0	0	0	0						
14	0	/	/	0	+	0	+	/	+	+	+	+	+	0	+	0	+	0	+	0	+	+	+	0	0	0	0	0	0	0	0	0						
15	0	/	/	+	+	0	+	/	+	+	+	+	+	0	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+	+						
16	0	/	/	0	+	0	+	/	+	+	+	+	+	0	+	0	+	0	+	0	+	0	+	+	+	+	+	+	+	+	+	+						
Auto-control																																				±H	±H	
DAT																																						

Table 1. Antibody identification profile and identification results (continued).

Rh-hr	e	+	+	0	+	+	0	+	+	+	+	+	0	+	+	+	+		
	c	0	0	+	+	0	+	+	+	+	+	0	+	0	+	0	+		
	E	0	0	+	0	0	+	0	0	0	0	+	+	0	+	0	0	Auto-control	DAT
	D	+	+	+	+	0	0	0	0	0	+	+	+	+	+	+	0		
	C	+	+	0	0	+	0	0	0	0	0	+	W	+	+	+	0		
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16			

0 - no agglutination, ± - weak agglutination, 2+ - agglutination strength of 2+, DAT - direct antiglobulin test. IAT - indirect antiglobulin test.

Table 2. The reactivity profiles of the antibody with screening cells treated with different reagents or in different reaction media.

	Kell				IAT (untreated serum)	IS	Polybrene test	IAT (0.01M DTT-treated serum)
	K	k	Kp ^a	Kp ^b				
1	0	+	0	+	0	0	0	0
2	0	+	0	+	0	0	0	0
8	0	+	+	+	2+	0	0	2+
12	0	+	+	+	2+	0	0	2+
1*	0	+	0	+	0			
2*	0	+	0	+	0			
8*	0	+	+	+	2+			
12*	0	+	+	+	2+			
1 [#]	0	+	0	+	0			
2 [#]	0	+	0	+	0			
8 [#]	0	+	+	+	0			
12 [#]	0	+	+	+	0			

* - treated with papain, [#] - treated with 0.2M dithiothreitol (DTT), IS - immediate spin, 0 - no agglutination, 2+ - agglutination strength of 2+.

Table 3. The results of DAT and Kell antigen phenotype of proximal and distal RBCs.

	Kell				DAT
	K	k	Kp ^a	Kp ^b	
Proximal RBCs	0	+	0	+	0
Distal RBCs	0	+	+	+	+

0 - negative, + - positive.

CASE PRESENTATION

A 69-year-old male patient was admitted for gastrointestinal bleeding. He underwent left atrial appendage

closure surgery and received a transfusion of 4 units of red blood cells (RBCs) at another hospital 8 weeks ago. Initial laboratory tests did not show significant hemolysis. Pre-transfusion testing revealed O RhD positive

blood type, with a positive antibody screen result, and the direct antiglobulin test (DAT) showed weak agglutination. To determine the antibody specificity, antibody identification tests were conducted, identifying an anti-Kp^a antibody (as detailed in Table 1). To further characterize this antibody, we assessed the reactivity of the patient's serum with Kp^a antigen-positive red blood cells at different test phases (e.g., immediate spin, indirect antiglobulin test), with DTT used for further confirmation. We also investigated the sensitivity of the Kp^a antigen to DTT and papain.

RESULTS

As shown in Table 1, the patient's serum was found to contain an unexpected anti-Kp^a antibody by an antibody identification test with IAT method.

As shown in Table 2, the anti-Kp^a antibody in the patient's serum was identified as an IgG antibody: detectable by IAT, but not by immediate spin saline method and polybrene test; the antibody could not be destroyed by 0.01M DTT. Furthermore, the Kp^a antigen on RBCs was susceptible to 0.2M DTT, but not to papain.

Considering the patient's recent red blood cell transfusion, we separated the patient's own RBCs from the transfused ones using capillary centrifugation and performed direct antiglobulin test and Kell blood group antigen detection. As shown in Table 3, proximal (autologous red blood cells) DAT was negative, and the Kell antigen phenotype was Kp(a-b+), while the distal (transfused red blood cells) DAT was positive, and the Kell antigen phenotype was Kp(a+b+).

DISCUSSION

This case report describes the identification of an anti-Kp^a antibody in a Chinese patient presenting with moderate anemia without overt hemolysis. The antibody was verified through antibody identification and red blood cell antigen phenotype testing, and its characteristics were further studied using DTT and enzymatic treatment, etc. Finally, based on capillary centrifugation method, we determined that the cause of the patient's anti-Kp^a antibody was due to the transfusion of Kp^a antigen-positive RBCs. Anti-Kp^a antibody, a low-frequency antigen antibody within the Kell blood group system, is typically an allogeneic antibody resulting from exposure to Kp^a antigen-positive RBCs through blood transfusion or pregnancy [2-4], and can also occur as a result of cross-reactivity following severe infection [5].

A systematic search of the Pubmed database revealed no prior publications by Chinese scholars on anti-Kp^a, likely due to its low incidence and the high risk of missed detection. The majority of common screening cell reagents on the domestic market do not include Kp^a antigen-positive red blood cells, contributing to the high risk of missed detection and potentially affecting the ac-

curacy of data on the distribution of Kell blood group antigens in the Chinese population. This highlights a significant deficiency in current domestic screening cell reagents and provides a basis for establishing requirements for transfusion laboratory testing reagents. The missed detection of low-frequency antigen antibody poses significant safety risks, particularly for medical institutions conducting electronic crossmatch, with reports of acute extravascular hemolytic transfusion reactions due to anti-Kp^a antibody missed by electronic crossmatch [6]. Therefore, it is imperative to improve the detection rate of several clinically significant low-frequency antigen antibodies by designing a comprehensive screening cell spectrum to ensure patient transfusion safety. As the first case of anti-Kp^a in the Chinese population published in an international journal, this report offers a new perspective for evaluating the distribution frequency of Kell blood group antigens in the Chinese population, providing a theoretical foundation for refining electronic crossmatch strategies and enhancing patient transfusion safety.

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Declaration of Interest:

None.

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