Clinical History

A 66-year-old man is scheduled for an elective hip replacement surgery. A preadmission test sample (ethylenediaminetetraacetic acid [EDTA] anticoagulant) is submitted to the blood bank for type and screen along with an order for two units of red blood cells (RBCs). No transfusion history is provided.

ABO/Rh/Antibody Screen

ABO/Rh (tube method)				
Patient RBCs (forward typing)			Patient plasma (reverse typing)	
Anti-A	Anti-B	Anti-D	A_1 cells	B cells
4+	0	3+	1+	4+
Anti-A_1 lectin	Patient: 4+	A_1 cells: 4+	A_2 cells: 0	
Antibody screen (tube LISS method)				
	37 °C	AHG		
SC1	0	W+		
SC2	0	2+		

Reaction scale = 0 (no reaction) to 4 + (strong reaction); *W* weak

Tube Panel

		Rh-hr								Kell						Duffy		Kidd		Lewis		MNS				P	Lutheran		Test results: IAT/tube LISS			
Cell #	Rh-hr	D	C	E	c	e	f	C^w	V	K	k	Kp^a	Kp^b	Js^a	Js^b	Fy^a	Fy^b	Jk^a	Jk^b	Le^a	Le^b	M	N	S	s	P_1	Lu^a	Lu^b	IS	4 °C	37 °C	AHG
1	R_oR_1	+	+	0	0	+	0	+	0	0	+	0	+	0	+	0	+	+	+	0	+	0	+	+	+	+	0	+	2+	4+	0	1+
2	R_1R_1	+	+	0	0	+	0	0	0	+	+	0	+	0	+	+	+	+	0	0	+	+	+	+	+	0	0	+	2+	3+	0	2+
3	R_1R_2	+	0	+	+	0	0	0	0	0	+	0	+	0	+	0	+	+	0	0	0	+	0	+	+	$+^s$	0	+	2+	3+	0	W+
4	R_2r	+	0	0	+	+	+	0	+	0	+	0	+	0	+	0	0	+	0	0	0	+	+	+	+	+	0	+	1+	3+	0	W+
5	r'r	0	+	0	+	+	+	0	0	0	+	0	+	0	+	0	+	0	+	+	0	0	+	0	+	+	0	+	2+	3+	0	W+
6	r''r	0	0	+	+	+	+	0	0	0	+	0	+	0	+	+	0	0	+	0	+	+	+	+	+	$+^w$	0	+	2+	3+	0	1+
7	rr	0	0	0	+	+	+	0	0	+	+	0	+	0	+	0	+	+	+	0	0	+	0	+	+	$+^s$	0	+	2+	3+	0	2+
8	rr	0	0	0	+	+	+	0	0	0	+	0	+	0	+	+	+	0	+	+	0	+	+	+	0	$+^s$	0	+	2+	4+	0	1+
9	rr	0	0	0	+	+	+	0	0	0	+	+	+	0	+	0	+	+	0	0	+	0	+	0	+	0	0	+	2+	3+	0	W+
10	rr	0	0	0	+	+	+	0	0	0	+	0	+	0	+	+	0	+	0	0	+	+	+	+	+	$+^w$	0	+	1+	3+	0	W+
11	R_1R_1	+	+	0	0	+	0	0	0	0	+	0	+	0	+	0	+	+	0	0	+	+	0	+	0	+	0	+	2+	3+	0	W+
Patient cell																													2+	3+	0	W+

Reaction scale = 0 (no reaction) to 4+ (strong reaction); *S* strong, *W* weak

Pre-Warm Panel (37 °C)

		Rh-hr								Kell						Duffy		Kidd		Lewis		MNS				P	Lutheran		Test results: IAT/ tube LISS	
Cell #	Rh-hr	D	C	E	c	e	f	C^w	V	K	k	Kp^a	Kp^b	Js^a	Js^b	Fy^a	Fy^b	Jk^a	Jk^b	Le^a	Le^b	M	N	S	s	P_1	Lu^a	Lu^b	37 °C	AHG
1	$R_{zy}R_1$	+	+	0	0	+	0	+	0	0	+	0	+	0	+	0	+	+	+	0	+	0	+	+	+	+	0	+	0	0
2	R_1R_1	+	+	0	0	+	0	0	0	+	+	0	+	0	+	+	+	+	0	0	+	+	+	+	+	0	0	+	0	2+
3	R_2R_2	+	0	+	+	0	0	0	0	0	+	0	+	0	+	0	+	+	0	0	0	+	0	+	+	$+^S$	0	+	0	0
4	R_or	+	0	0	+	+	+	0	+	0	+	0	+	0	+	0	0	+	0	0	0	+	+	+	+	+	0	+	0	0
5	r'r	0	+	0	+	+	+	0	0	0	+	0	+	0	+	0	+	0	+	+	0	0	+	0	+	+	0	+	0	0
6	r''r	0	0	+	+	+	+	0	0	0	+	0	+	0	+	+	0	0	+	0	+	+	+	+	+	$+^W$	0	+	0	0
7	rr	0	0	0	+	+	+	0	0	+	+	0	+	0	+	0	+	+	+	0	0	+	0	+	+	$+^S$	0	+	0	2+
8	rr	0	0	0	+	+	+	0	0	0	+	0	+	0	+	+	+	0	+	+	0	+	+	+	0	$+^S$	0	+	0	0
9	rr	0	0	0	+	+	+	0	0	0	+	+	+	0	+	0	+	+	0	0	+	0	+	0	+	0	0	+	0	0
10	rr	0	0	0	+	+	+	0	0	0	+	0	+	0	+	+	0	+	0	0	+	+	+	+	+	$+^W$	0	+	0	0
11	R_1R_1	+	+	0	0	+	0	0	0	0	+	0	+	0	+	0	+	+	0	0	+	+	0	+	0	+	0	+	0	0
Patient cell																												0	0

Reaction scale = 0 (no reaction) to 4+ (strong reaction); *S* strong, *W* weak

Additional Study: 4 °C Incubation (Cold Panel)

A_1 cells	1+
O cells	4+
O cord cells	0
O_i cells	0

1. **What antibodies did you identify?** Cold autoantibody and anti-K alloantibody are present.
2. **How does the pre-warm panel help you?** The pre-warm panel eliminates cold reactions allowing for identification of the warm anti-K antibody. Other methods to negate cold antibody reactions include cold autoadsorption and the use of rabbit erythrocyte stroma (RESt), both of which can remove the cold antibodies (typically immunoglobulin [Ig]M, anti-I, or anti-IH). RESt is an older technique not used often anymore and may remove anti-B antibodies as well.
3. **What is the significance of the anti-A_1 lectin result?** There is 1+ reactivity with A_1 cells in the patient's back type; this could be due to either a naturally occurring anti-A_1 antibody (if patient is of A_2 or other weak subtype of A) or interference from a cold-reacting antibody. The positive reaction with anti-A_1 lectin (*Dolichos biflorus*) indicates that the patient is A_1 type; thus, the reaction with A_1 cells in the reverse typing is not due to anti-A_1. In this case, it is due to cold autoantibody since the autocontrol is positive in the panel at cold temperatures.

4. **How do you interpret the results of the 4 °C incubation panel using A_1 cells, O cells, O cord cells, and O_i cells? Do the results of this cold panel have any clinical significance?** This identifies that the cold autoantibody is likely anti-IH (i.e., having reactivity with I and H antigens, I antigen is not expressed on cord cells and O_i cells, while H antigen is only weakly expressed on A_1 cells). The results of this panel, though, do not have any clinical significance. Cold-reacting antibodies are most commonly benign cold agglutinins; their significance lies in the potential for interference with ABO typing since this is generally performed at room temperature.
5. **How many donor RBC units need to be screened in order to find two compatible units as requested? (Refer to the Table of RBC Antigen Frequencies.)** K antigen, part of the Kell blood group antigen system, is present in only about 9% of the population (Caucasian frequency); thus, more than 90% of donor RBCs will lack the K antigen and will be compatible with the patient in this case. Dividing 2 by 0.9 equals 2.22; thus, only two units need to be screened in all probability to find two K-antigen-negative RBC units for the patient.
6. **What do the antibody findings suggest about this patient's past medical history?** The finding of anti-K antibody, a warm, immune alloantibody (i.e., requires prior exposure to the antigen for development), suggests that the patient received blood transfusion sometime in the past. Naturally occurring anti-K has been described, but in most cases is IgM, reacting best at room temperature and sometimes associated with infectious illness [1, 2]. Rare cases of autoanti-K mimicking an alloantibody have also been described [3]. If the patient denies any history of red cell transfusion, passive acquisition is possible (such as through recent plasma or platelet transfusion or, in the case of a woman, through exposure during pregnancy). Anti-IH is commonly seen in the serum of healthy A_1 individuals and does not imply any clinical history.

References

1. Marsh WL, Nichols ME, Oyen R, et al. Naturally occurring anti-Kell stimulated by E. coli enterocolitis in a 20-day-old child. Transfusion. 1978;18(2):149–54.
2. Judd WJ, Walter WJ, Steiner EA. Clinical and laboratory findings on two patients with naturally occurring anti-Kell agglutinins. Transfusion. 1981;21(2):184–8.
3. Viggiano E, Clary NL, Ballas SK. Autoanti-K antibody mimicking an alloantibody. Transfusion. 1982;22(4):329–32.