

ABID CASE #27

(Case study by Jim Perkins, © 2009)



History: This patient was a 36 year-old woman with a stage 4 gastro-intestinal stromal tumor (GIST) who was taking an experimental drug. On a routine clinic visit a hemoglobin of 8.5 was noted, and a type-and-screen was ordered. She had received 36 units of RBCs over the previous 3 years including 21 units in the year in which the workup was done, most recently 45 days ago, at which time the antibody screen was negative.

ABO and Rh Typing

<A	<B	A1 cells	B cells	6% alb	<D	<D/AHG	CCC	Interp
0	0	4+	4+		4+			

Antibody Screen

	Gel
SCI	2+
SCII	2+

Direct Antiglobulin Test (tube method)

	Poly	IgG	<C3
AHG	0		
5' incub.	0		
CCC	2+		

Initial Plasma Panel

Lot# 8RA167	Rh system	Kell										Duffy		Kidd	Xg	Lewis		MNSs				P	Lutheran		Other Typings	Cell	Gel				
Cell	Rh	D	C	E	c	e	V	K	k	Kp ^a	Kp ^b	Js ^a	Js ^b	Fy ^a	Fy ^b	Jk ^a	Jk ^b	Xg ^a	Le ^a	Le ^b	S	s	M	N	P1	Lu ^a	Lu ^b				
1	R1wR1	+	+	0	0	+	0	0	+	0	+	0	+	+	0	+	0	0	0	+	0	+	0	+	+	+s	0	+	C ^w	1	0
2	R1R1	+	+	0	0	+	0	0	0	+	0	+	+	+	0	+	+	0	+	+	0	+	0	+	0	+	0	+		2	2+
3	R2R2	+	0	+	+	0	0	0	+	0	+	0	+	0	+	+	+	+	0	+	+	0	+	0	+	0	+		3	w+	
4	Ror	+	0	0	+	+	0	0	+	0	+	0	+	0	0	+	0	0	0	0	0	+	+	0	+s	0	+		4	2+	
5	r'r	0	+	0	+	+	0	0	+	0	+	0	+	0	+	0	+	+	+	0	0	+	0	+	+	0	+		5	0	
6	r ^w r	0	0	+	+	+	0	0	+	0	+	0	+	+	0	+	0	+	0	+	0	+	0	+	+	0	+		6	1+	
7	rr	0	0	0	+	+	0	0	+	+	+	0	+	+	+	+	+	+	0	+	0	+	+	+	0	+	+		7	2+	
8	rr	0	0	0	+	+	0	0	+	+	+	0	+	+	+	+	+	+	0	+	0	+	+	+	0	+	+		8	w+	
9	rr	0	0	0	+	+	0	0	+	0	+	0	+	+	0	0	+	+	+	0	0	+	0	+	+	0	+		9	2+	
10	rr	0	0	0	+	+	0	0	+	0	+	0	+	0	+	+	0	+	0	+	+	0	+	0	0	0	+		10	w+	
11	R1R1	+	+	0	0	+	0	+	+	0	+	0	+	0	+	+	+	+	0	+	0	+	0	+	0	0	+		11	w+	
Patient																													AC		

Extended Phenotype

	Rh system				Kell				Kidd		Duffy		Lewis		MNSs								
	C	E	c	e*	K	k	Kp ^a	Js ^a	Fy ^a	Fy ^b	Jk ^a	Jk ^b	Le ^a	Le ^b	S	s	M	N	P1	I	H	A ₁	
Patient	mf	3+	3+	3+	0				3+	3+	3+	3+	0	4+	3+	3+	4+	4+	4+				

*Patient RBCs are not phenotyped for e if the E antigen is not expressed.

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Questions:

2. Did the additional testing help? Can you form a new hypothesis that will lead to further investigation?

The technologist tested 3% (tube method) screening cells using LISS and PEG enhancement, and again after treating the screening cells with ficin and AET. They also tested dilutions of the plasma against screening cell II, which had the detectable reactions. (See reactions below)

Special tests (AET = 2-Aminoethylisothiuronium, a sulfhydryl reducing agent)

	LISS				PEG	Ficin treated RBCs			AET treated RBCs			
	IS	RT	37°, 30'	AHG	AHG	IS	37°, 30'	AHG	IS	37°, 30'	AHG	anti-k*
SCI	0	0	0	0 ^v	0 ^v	0	1+	0 ^v	1+	0	0 ^v	0 ^v
SCII	0	0	0	vw+	vw+	0	1+	0 ^v	1+	0	vw+	
Auto	0	0	0	0 ^v		0	1+	0 ^v				

*AET control

Titration

	2 drops diluted pt. plasma, 1 drop saline-susp. RBCs, read at AHG phase							
	1:1	1:2	1:4	1:8	1:16	1:32	1:64	Titre
SCII	w+	w+	vw+	vw+	vw+	vw+	0 ^v	2

Questions:

3. What do the new test results show? How was the AET control performed and what did it demonstrate? How might we proceed now?

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The technologist performed a plasma neutralization test with the following results:

Plasma neutralization test

Pt. plasma diluted as above and incubated 1:1 with pooled normal plasma, read at AHG								Titer
SCII	0 ^v	0 ^v	0 ^v	0 ^v	0 ^v	0 ^v	0 ^v	0
Dilution control (Saline substituted for pooled normal plasma)								
SCII	w+	w+	vw+	vw+	0 ^v	0 ^v	0 ^v	2

4. What does the plasma neutralization test demonstrate? What is the identity of this antibody? Why must a dilution control be done?

5. Would donor RBCs reactive with this antibody cause hemolytic transfusion reactions? Has any other type of reaction been associated with this antibody specificity? What do we know about the Ch/Rg antigen(s)?