

Antibody against compound antigen ce(f)

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Abstract

Antibodies against compound antigens occur infrequently. We report here a case of anti-ce (anti-f) in association with anti-Jk^a. The specificity was evident from the reaction pattern of the reagent panel cells such that adsorption and elution was not necessary.

Key words: Antibodies. Compound antigen. ce(f)

Case report

In the Rh blood group system, other than the existence of discrete antigens C,D,E, c and e, there are 4 other combination antigens, viz ce(f), Ce, cE and CE. They are commonly described as compound antigen¹, *cis* product antigen², or joint product³. The term is used to designate an antigen that results when the antigens are encoded by the same haplotype. (i.e. the genes are in *cis* position)

Antibodies against *cis* product antigens have been described as infrequent², though not rare. Such antibodies may be concealed by co-existing antibodies of the more obvious Rh specificities. e.g. co-existing anti-c and/or anti-e may mask the effect of anti-f. Its presence can only be demonstrated through adsorption and elution with red cells of selected phenotypes.

Our patient was an elderly Chinese female aged 64, admitted on 15.12.1997 because of gastrointestinal bleeding with haemoglobin level of 10.0 g/dL and platelet count of $2 \times 10^9/L$. Requests for crossmatch and platelet transfusion were received. Blood was not given although the antibody screening on the patient's serum was negative. Patient was given 2 units of packed red cells on 21.12.1997 after electronic crossmatch. The antibody screening was negative by the LISS-IAT technique (DiaMed Gel Test). Between these periods, patients had also received 16 units of platelet concentrate and she was subsequently diagnosed as having ITP.

On 22.4.1998, request for transfusion was again received. The result of the antibody screening on this occasion was positive with a score of 2+ (Grading of score: 0 - 4+) for all 3 reagent screening cells (Table 1). Antibody identification was

performed (Table 2). The 11 panel cells revealed a clear-cut reaction pattern of anti-Jk^a (anti-Le^a and anti-Mi to be excluded). Moreover 9 cells in the panel (Cell 1 and 4 to 11) gave inconsistent reaction score: Cell 1 with score of 2+ similar to the 3 screening cells while the eight other cells 4-11 showed a full score of 4+. This showed that there must be some other strong antibody in addition to anti-Jk^a. Review of these 8 panel cells showed that they all have c and e antigens in *cis* position. The two remaining Jk(a-) cells of R¹R¹ (Cell 2) and R²R² (Cell 3) showed negative reactions. Up to this point, it was very clear that the second antibody must be anti-f without admixture of anti-c or anti-e. As the pure specificity of anti-f was so evident from the panel cell reaction pattern, adsorption and elution for confirmation was not necessary.

The following additional tests were performed.

1. To exclude the possibility of co-existing anti-Le^a, Le(a+) Jk(a-) R²R² reagent cell from other panel showed negative reaction.
2. To exclude the possibility of co-existing anti-Mi, Jk(a-) R¹R² and Mi(+) cells showed negative reaction.
3. Four other reagent cells that were Jk(a-) and f+ from other panel showed positive reaction.
4. Two donor cells of R¹R¹ and Jk(a-) showed negative reaction.
5. Patient's cells were weakly positive for DAT: anti-IgG: weak, anti-C3d: negative
6. Patient's phenotypes: Jk(a-b+), CcDEe, MN, Le (a-b-).

Table 1: Result of antibody screening

	Screening cell	C	D	E	c	e	f	K	k	Fy ^a	Fy ^b	Jk ^a	Jk ^b	Le ^a	Le ^b	P ₁	M	N	S	s	Mi	Recation score
I	R ¹ R ¹	+	+	0	0	+	0	+	+	0	+	+	+	0	+	+	+	+	+	+	+	2+
II	R ² R ²	0	+	+	+	0	0	0	+	+	0	+	0	+	0	+	+	+	0	+	+	2+
III	R ¹ R ¹	+	+	0	0	+	0	0	+	+	0	+	+	0	+	0	+	+	0	+	+	2+

Table 2: Result of antibody identification by panel cells

	Panel cell	C	D	E	c	e	f	K	k	Fy ^a	Fy ^b	Jk ^a	Jk ^b	Le ^a	Le ^b	P ₁	M	N	S	s	Recation score
1	R ² R ¹	+	+	+	0	+	0	0	+	0	+	+	+	+	0	+	+	+	0	+	2+
2	R ¹ R ¹	+	+	0	0	+	0	0	+	+	+	0	+	0	+	+	0	+	0	+	0
3	R ² R ²	0	+	+	+	0	0	+	+	+	+	0	+	0	+	+	+	+	+	0	0
4	r ¹ r	+	0	0	+	+	+	0	+	+	+	0	+	0	+	+	+	0	+	0	4+
5	r ¹ r	0	0	+	+	+	+	0	+	0	+	+	0	+	0	0	+	0	+	+	4+
6	rr	0	0	0	+	+	+	+	+	0	+	+	0	+	0	+	0	+	0	+	4+
7	rr	0	0	0	+	+	+	0	+	+	0	+	+	0	0	+	0	+	0	+	4+
8	R ⁰ R ⁰	0	+	0	+	+	+	0	+	0	0	+	0	0	+	+	+	+	+	0	4+
9	rr	0	0	0	+	+	+	0	+	+	+	+	0	+	0	0	0	+	0	+	4+
10	R ¹ r	+	+	0	+	+	+	+	0	+	+	+	+	+	0	+	+	0	+	0	4+
11	R ² r	0	+	+	+	+	+	0	+	+	0	+	+	0	+	+	+	0	0	+	4+

Table 3: Characteristics of anti-f

	Reaction	
R ¹ R ¹	CDe/CDe	0
R ² R ²	cDE/cDE	0
R ¹ R ²	CDe/cDE	0
rr	cde/cde	+
R ⁰ R ⁰	cDe/cDe	+
CcDEe	CDe/cDE	0
CcDEe	CDE/cde	+

The reaction pattern of anti-f is peculiar in that it does not react with c or e singly or with combined c and e in *trans*. However reaction is seen only when c and e are in *cis*.

During the period 15.12.1997 - 5.1.1998, patient had been transfused with 2 units of packed red cells and 77 units of platelet concentrate. The 1st antibody screening was negative on 15.12.1997. On 22.4.1998, the 2nd antibody screening became positive. It is very probable that anti-Jk^a had been produced as a result of transfusion. However it was not known if anti-f was already present initially or formed only after blood transfusion.

Discussion

In routine antibody screening, anti-f cannot be detected because all reagent screening cells are usually R¹R¹ or R²R². It was not until the 2nd positive antibody screening that led us to perform the antibody identification which revealed the presence of anti-f in addition to anti-Jk^a. If the 2nd time antibody screening were negative, we might have missed the anti-f and the patient might have been transfused with incompatible blood. Every laboratory test has its limitation and the type & screen procedure is of no exception.

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Reference

1. Sally V. Rudmann. Textbook of Blood Banking and Transfusion Medicine. 1995:110.
2. Richard H. Walker. Technical Manual. American Association of Blood Banks. 1993:239.
3. P.L. Mollison, C.P. Engelfriet, Marcela Contreras. Blood Transfusion in Clinical Medicine. 1993:215