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Prevalence and specificity of some rare red cell pan-reactive alloantibodies against high frequency red cell antigens among hospitalized Saudi patients

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ABSTRACT

Some patients´ sera react with all available donors´ red cells and a compatible donor is difficult or impossible to be found. These may either be due to a complex mixture of antibodies or the presence of alloantibodies against high-frequency antigens (HFAs). The aim of this study is to identify the prevalence and characteristics of antibodies to HFAs in Saudi Arabian patients. A total of 23 out of 172 000 patients who received blood transfusions had rare alloantibodies to HFAs at an incidence of 0.013%. Twenty-three patients suspected with pan-reactive alloantibodies against HFAs had their red cells tested using antisera to HFAs, while their plasma was tested against a selected panel of red blood cells with rare phenotypes. Anti-Ge2 antibody was found in the highest number of patients (56.5%), whereas anti-U, anti JK3, anti H, anti-RH 29, anti-hr^s, anti-Kn^a, anti-Ch, anti-Rg, anti-Yt^a, and anti-Cr^a antibodies were found in the remaining patients (43.5%). This study suggests that although antibodies such as anti-Ge2, anti-Kn^a, anti-Ch, anti-Rg, anti-Yt^a , and anti-Cr^a are not clinically significant, they cause a delay in the provision of compatible blood. Whereas, anti-U, anti JK3, anti H, anti-RH29 and anti-hr^s are clinically significant antibodies. An understanding of antibody characteristics to HFAs and the widespread use of the extended red cell phenotype and antibody identification panel will both be helpful for the diagnosis of these HFAs.

Keywords: pan-reactive alloantibody, high-frequency antigen, blood transfusion

INTRODUCTION

High-frequency antigens (HFAs) occur at frequencies of over 99 percent and vary according to population. For example Rh D antigen is an HFA among Chinese (99.71%) and South East Asian populations[1]. Alloantibodies to HFAs arise in the patient´s blood in isolated cases only after exposure to foreign red cells by pregnancy, transfusion, or transplantation. The provision of compatible blood for the patients with alloantibodies against HFAs is a real challenge for safe transfusions[2]. The Rare Donor Working Party of the International Society for Blood Transfusion and several other national working parties collaborate in

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procuring the supply of RBC units for patients with antibodies to HFAs[3]. Since *in vivo* red cell survival studies and macrophage monolayer assays (MMAs)[4] are not utilized by most transfusion services, the identification of red cell phenotypes and rare alloantibodies is essential to avoid hemolytic transfusion reactions. In this study, we identified different rare antibodies to HFAs and their characteristics in Saudi Arabian patients to help minimize sensitization by the provision of phenotypically correct blood in emergency situations.

MATERIALS AND METHODS

Patients

This retrospective study was done in the Blood Bank Laboratory of King Fahad Armed Forces Hospital, Jeddah. Patients´ data was taken from the blood transfusion laboratory information management system (LIMS) and was analyzed for positive antibodies for patients admitted between 1st January 2000 and 31st December 2013. A total of 23 out of 172 000 patients who were diagnosed with suspected antibodies to HFAs by pre-transfusion testing were selected in this study[5].

Methods

The pre-transfusion tests consisted of identifying recipient ABO and Rh D types and antibody screening. These were compared with the patients´ previous records of cross-matching[5].

The antibody screening was done by Indirect Antiglobulin Technique (IAT) and Enzyme IAT testing using three-cell screens (ID-DiaCell Ⅰ - Ⅱ - Ⅲ, ID-DiaCell Ⅰ P- Ⅱ P- Ⅲ P, Diamed IgG cards supplied by BioRad, Switzerland). The antibody specificity was investigated using both an 11 cell IAT panel (ID-DiaPanel, DiaMed), as well as enzyme-treated Papain panel cells (ID-DiaPanel P). Red blood cell phenotype detection for common antigens was performed for all 23 patients using ID-Antigen Profile Ⅰ, Ⅱ, Ⅲ (BioRad).

The 23 cases were tested positive for antibodies by screening, while the negative auto control obtained a positive reaction with all 1–11 panel cells. The patients´ sera were incompatible with all tested donors´ RBC units in the blood bank and have positive reactions with reagent red cells that matched the patient´s phenotype, which excluded the presence of a complex mixture of antibodies. The patient´s blood samples were sent to IBGRL, Bristol (UK), to determine the specificity of antibodies. The patients´ red cells were tested with antisera to HFAs or with frozen antisera of previously identified antibodies to HFAs, which was confirmed by cross-matching the pa-tients´ plasma with selected rare phenotype and null cells, based on characteristics observed in the initial panel. An eluate was made from group O antigen, cross-matched with patient´s plasma and tested for ABO blood group to eliminate ABO incompatibility issues. A neutralization test for the CH/RG blood group system was done for 2 cases and DNA extraction and sequencing for all exons of the *RHAG* gene and for all 10 exons of *RhCE* for patients with anti Rh 29 and anti-hr^s respectively. The research protocol was approved by the local hospital´s Ethics Committee.

Statistical analysis

The data was analyzed using SPSS statistical software, version 17.0 (SPSS Inc., Chicago, IL, USA). Cross tabulation and χ^2 tests were used to detect the significant differences in sex distribution between the 2 groups of patients, one with anti-Ge2 and the other with alloantibodies. The $\bar{x}\pm s$, student t-test was used to compare age between the 2 groups. *P* values of less than 0.05 were considered statistically significant.

RESULTS

Age and sex distribution in the studied group

From 2000 to 2013, a total of 23 out of 172 000 patients who received blood transfusions had rare alloantibodies to HFAs at an incidence of 0.013%. ***Table 1*** described the age and sex distribution of the studied group. There were 8 males and 15 females with a mean age 50.7±20.6, which indicated that antibodies to HFAs occurred at middle age or older or after repeated pregnancy or blood transfusion. The female group was more than the male which may be due to histories of pregnancy, but this was not statistically significant. The most frequently identified alloantibodies to HFAs were anti-Ge2, which were present in 13/23 (56.5%) while the remaining others represented 10/23 (43.5%).

The reaction characteristics of different panreactive alloantibodies against HFAs

Table 2 showed that there were 11 different panreactive antibodies identified against HFAs. They were divided into 5 antibodies according to the intensity of agglutination, which varied in intensity (anti-Yt^a-, anti-CH, anti-Rg, anti-Cr^a, anti-Kn^a), while there were 6 of the same intensity(anti-Ge2, anti-U, anti-JK3, anti-Hr^s, anti-RH29, anti-H). According to the effect of papain treatment, 7 patients were sensitive to papain treated cells (anti-Ge2, anti-U, anti-Yt^a, anti-CH, anti-Rg, Cr^a,

Table 1 **Age and sex distribution in the studied group**

	Sex[*n*(%)]			Age (year, $\bar{x}\pm s$)		
	Total	Male	Female	Total	Male	Female
Total	23(100.0)	8(34.8)	15(65.2)	50.7±20.6	56.3±27.5	47.7±16.1
Anti-Ge2	13(56.5)	6(46.2)	7(53.8)	60.1±22.6	60.1±30.5	59.4±15.5
Others	10(43.5)	2(20.0)	8(80.0)	38.5±7.9	42.5±10.6	37.5±7.6
P		0.192			0.228	

anti-Kn^a), and 4 enhanced or not affected by papain treatment (anti-JK3, anti-hr^s, anti-RH29 and anti-H). This difference may be helpful in the identification of some antibodies to HFAs.

Table 2 **The summary of the reaction characteristics of different pan-reactive alloantibodies against HFAs in the DiaCell Ⅰ-Ⅱ-Ⅲ and DiaCell Ⅰ P-Ⅱ P-Ⅲ P**

Antibodies	Case	DiaCell Ⅰ-Ⅱ-Ⅲ	Intensity	DiaCell Ⅰ P-Ⅱ P-Ⅲ P
Anti-Ge2	1–13	2+	U	–
Anti-U	14	2+	U	–
Anti-JK3	15	2+	U	2+
Anti-hr^s	16	3+	U	3+
Anti-RH29	17	2+	U	3+
Anti-H	18	4+	U	4+
Anti-Yt^a	19	2+	V	–
Anti-CH	20	2+	V	–
Anti-Rg	21	2+	V	–
Anti-Cr^a	22	2+	V	–
Anti-Kn^a	23	2+	V	–

U: uniform with the same strength of agglutinations in all test cells; V: variable intensity.

Antibody screening and identification in different patients with pan-reactive alloantibodies

Table 3 showed that the cases from 1–13 had anti-G2 with the rare Ge:–2,3,4 phenotype (Yus type Ge-negative). The patient´s serum reacted with IAT untreated cells, but not with papain treated cells. Several Ge:–2 cells were compatible. Case 14 had anti-U in his serum, which reacted with IAT untreated cells but not with papain treated cells and was compatible with U-cells. Case 15 had anti-JK3, which was further confirmed by negative urea lysis test and its compatibility with JK(a–b–) cells. Case 16 had anti-hr^s and his cells were hr^s-antigen. The patient´s plasma was found to be compatible with Rh_{null}, –D–/–D–, Dce^{AR}/Dce^{AR}, but incompatible with hrs and Hr cells. The DNA sequencing was done on all ten exons of the RHCE gene and five mutations were detected in exon 5, and one mutation in exon 6, with the expected phenotype hr^s–, VS–, V+^{WK}. Case 17 had strong anti Rh29 and the very rare Rh_{null} phenotype, so was typed as blood group O Rh_{null} (D–, C–, E–, c–, e–), Cw–, K–k+. The

Table 3 **The characteristics of antibody screening and identification in patients with pan-reactive alloantibodies to HFAs**

Case	Allo AB Type	Red cell phenotype	LISS /Coombs	Auto control	Screening cells			Panel red cells										
					1	11	111	1	2	3	4	5	6	7	8	9	10	11
1–13	Anti-Ge2	Ge:–2,3,4	LISS/C	–	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
14	Anti-U	M+N–S–s–	LISS/C	–	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
15	Anti-JK3	JK(a–b–) JK:–3	LISS/C	–	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+
			Enz	–	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+
16	Anti-hr^s	hr^s –	LISS/C	–	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+
			Enz	–	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+
17	Anti-RH29	O Rh null (D–, C–,E–,c–,e–)	LISS/C	–	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+
			Enz	–	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+
18	Anti-H	Oh	LISS/C	–	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+
			Enz	–	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+
19	Anti-Yt^a	Yt(a–b+)	LISS/C	–	1+	2+	2+	2+	2+	2+	2+	2+	2+	2+	1+	2+	2+	1+
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
20	Anti-CH	Ch–Rg+	LISS/C	–	w	1+	1+	1+	1+	1+	1+	1+	1+	1+	1+	1+	1+	1+
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
21	Anti-Rg	h+Rg–	LISS/C	–	w	1+	1+	1+	1+	w	1+	1+	2+	1+	1+	2+	1+	w
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
22	Anti-Cr^a	Cr(a–)	LISS/C	–	1+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+	2+
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
23	Anti-Kn^a	Kn(a–)	LISS/C	–	2+	2+	2+	2+	W	2+	2+	2+	2+	2+	W	W	2+	2+
			Enz	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

LISS/C : LISS/Coombs; Enz: Enzyme

patients´ cells were negative for all Rh antigens tested for, including Rh17 and Rh 29. DNA sequencing of all exons of the *RHAG* gene detected a homozygous C>A mutation in exon 3 at n353, resulting in an alanine to glutamic acid change at position 118 of the RhAg protein (A118E), which is considered a novel mutation. Case 18 had the rare Oh phenotype and strong anti H, which reacted with all cells by all techniques. Several examples of Oh cells were compatible. Case 19 had anti-Yt^a and several examples of Yt(a-) were compatible with the patient´s serum. Case 20 and 21 were diagnosed with anti-Ch/anti-Rg respectively, which detected an antigen on C4 that is absorbed onto red cells *in vivo*. Their serum reacted to IAT with variable intensity with untreated cells, but not with papain treated cells, and was compatible with Ch–Rg+/Ch+Rg- cells respectively. This was confirmed by a neutralization test, which gave a strongly positive reaction with cells coated with C4 in LISS (coated with a large amount of Ch/Rg antigen). However, this reaction was inhibited by Ch+Rg-/Ch-Rg+ patient´s serum respectively. Case 22 had anti-Cr^a and his cells were negative for high incidence Cr^a antigen within the Cromer blood group system. Case 23 had an extremely weak anti-Kn^a(CR1) and the patient´s serum reacted with untreated cells at a different intensity by IAT, and being very weak in some cells explained why it gave negative results by tube method.

DISCUSSION

Pan-reactive alloantibodies due to HFAs detected during pre-transfusion testing is challenging to cross-matching and transfusion laboratories especially in emergency situations. The aim of this study was to estimate the prevalence and characteristics of these rare red cell alloantibodies and HFAs among Saudi Arabian patients. This may be the first step for starting a national panel for rare donors in Saudi Arabia, as there are no previous reports about rare red cell phenotypes among the Saudi Arabian population.

The antibodies to HFAs were very rare in our hospital, with 23/172 000 (0.013%) of all transfused patients having rare red blood cell phenotype. Anti G2 Gerbich antibodies were observed with the highest frequency of 13/23 (56.5%), which was higher than anti-U, anti JK3, anti Hrs, anti H, anti RH 29, anti-Kn^a, anti-Ch, anti-Rg, anti-Yt^a, and anti-Cr^a, which formed 10/23 (43.5%) and 10/172 000 of total transfusions. This result was similar to a study by Axel Seltsam *et al.*[6], who diagnosed a total of 52 hospitalized patients with antibodies to HFAs during their 20-month study in Austria, Germany, and Switzerland with an incidence of 0.04/100 000 in habitants per year. There were 8 cases of anti-Yt^a, 3 cases of anti-H and one case of anti-Rh17, but no cases of anti G2 antibodies.

There are three rare phenotypes for the Gerbich blood group, in which red cells lack one or more of the three high-frequency Gerbich antigens, Ge: –2, 3, 4 (Yus phenotype), Ge: –2, –3, 4 (Gerbich phenotype) and Ge: –2, –3, –4 (Leach phenotype)[7]. In our study 13/13 (100%) had the Ge: –2, 3, 4 phenotype. However, in the Daniel study[8], after 28 331 blood samples were taken from a Caucasian population of English, Danish, New Zealander, and Californian patients and screened with anti-Ge-2 antisera, 100% were found positive for the Ge-2 antigen. On the other hand, Booth *et al.*[9] studied 3 110 Melanesian Papua New Guinean blood samples, and findings of the 700 (22.5%) were negative for Ge-2 antigen, indicating that Papua New Guinea had the highest Ge-2 antigen-negative rate yet known.

In our study, due to emergency open heart surgeries, 4 patients with suspected anti-Ge2 received blood transfusions, in which 3 patients received from their relatives´ compatible cross-matched blood, while one received incompatible blood without any transfusion reaction either in clinical or laboratory tests. This in agreement with Woodfield[10] who reported that the anti-Ge2 was not generally considered to be clinically significant, although anti-Ge3 caused mild to moderate hemolytic transfusion reactions and also caused HDFN, which can be easily differentiated by resistance to papain treatment, whereas Ge2 and Ge4 are sensitive.

Case 14 had anti-U, which is very rare in Saudi Arabia and has been found exclusively in sub-Saharan Africans. It is a clinically significant antibody, which can cause hemolytic disease of the newborn and hemolytic transfusion reactions. Ringressi *et al.*[11] reported that less than 1% of Saharan Africans were U-negative and were capable of producing anti-U. There are 24 cases of anti-U immunization reported in the literature, all of which occurred in pregnant women originating from Sub-Saharan Africa[12]. Case 15 had anti-JK3 with a null phenotype Jk(a-b-), which is a very rare antibody that reacts with all red cells except those of the Jk(a-b-) phenotype. It can cause immediate and delayed HTRs and Jk(a-b-) blood must be selected. Rabeya *et al.*[13] reported the prevalence of Jk null phenotype: Jk(a-b-) was less than 1% among the white population, 0.58% among the Indian population, and 0.025% among Thai blood donors. Case 16 had the anti-hr^s antibody with hr^s-antigen and ideally antigen-negative (e-) blood must be considered for selection in these cases. The e antigen consists of several epitopes and is an HFA, which is found in more than

98% of the general population. It is called "partial e-antigen" when it lacks one or more of its epitopes, a situation seen more often in African Americans than in Caucasians[14]. Case 17 had anti-Rh29 antibody with Rh_{null} phenotype. It is a rare blood group with a reported frequency of approximately 1 in 6 million individuals[15]. This is a clinically significant antibody and only Rh_{null} should be used for transfusion. The patient required valve repair, and we obtained 3 units from her brother who had the same phenotype. Case 18 had high-titer anti-H antibody with the Bombay phenotype, which is a rare autosomal recessive phenotype within the ABO blood group. The estimated prevalence is 1 in 10 000 in India and 1 in 1 000 0000 outside of India[16]. A person with the Bombay Blood Group should receive only Bombay phenotype blood[17]. The patient was a 23 year-old female, who had been sensitized due to a previous pregnancy, however, she did not require a blood transfusion. Case 19 had anti-Yt^a with Yt(a−b+) phenotype. The patient could receive serologically least incompatible red cells, nevertheless, antigen-negative red cells for strong antibody as rare cases of *in vivo* hemolysis have been reported. This suggests that clinical significance should be interpreted on a case-by-case basis[18]. Cases 20 and 21 had anti-Chido and Rodgers antibodies respectively, which are generally considered clinically non-significant and serologically least incompatible red cells can be selected for transfusion. Anti-Ch and-Rg have very rarely been implicated in severe anaphylactic reactions following the infusion of a plasma product[19]. The patient in this study with anti-Rodgers had SCD, and after transfusion of 17 units of PRBCs he developed anti Rodgers antibodies and was known to have received many serologically least incompatible red cell transfusions without any complication. Case 22 had an anti-Cr^a antibody and his cells were negative for high incidence Cra antigen within the Cromer blood group system[20]. Case 23 had anti-Kn^a antibody directed against antigens in the Knops blood group system. Both cases were difficult to identify due to variable, weak reactivity with most red cells tested. Although anti-Kn^a is generally thought to be clinically insignificant, these antibodies need to be identified correctly because they can mimic or mask the presence of other clinically significant antibodies and/or alloantibodies.

In summary, extended red cell phenotype will be helpful for the diagnosis of some of HFA. Patients with the following red cell phenotypes (M+S+s+), Lu(a-b+), Kp(a-b+), Fy(a+b-), and the presence of anti-U, anti-Lub, anti-Kpb and anti-Fy3 are excluded respectively while Jk(a-b-) phenotype is suggesting the presence of anti-Jk3. Finding cross-matched and compatible blood can sometimes be solved by screening family members or by using an international rare donor panel, which facilitates the rapid location and exchange of rare blood among countries. The consolidation of a national panel for rare donors and a national bank of frozen red cells in Saudi Arabia are needed. More studies in other hospitals in Saudi Arabia for HFAs will give more accurate data regarding rare red blood cell phenotypes and the identification of rare donors from family members.

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