

SHORT COMMUNICATION

Prevalence of immunoglobulin A deficiency in Chinese blood donors and evaluation of anaphylactic transfusion reaction risk

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SUMMARY

Objectives/Aims: We investigated the incidence of immunoglobulin A (IgA) deficiency in Chinese population.

Background: The frequency of IgA deficiency, defined as a serum IgA level of $<0.05 \text{ mg dL}^{-1}$, has been broadly studied in different ethnic groups. Individuals with IgA deficiency may form specific antibodies against IgA, which can cause an anaphylactic response when the patient receives an IgA-containing blood transfusion.

Methods: A sandwich enzyme-linked immunosorbent assay was performed to screen for IgA deficiency and particle gel immunoassay used for confirmation. IgA antibodies were further detected by the DiaMed anti-IgA test in IgA-deficient blood donors.

Results: Of the total 22 609 healthy blood donors screened, only seven cases were confirmed as having IgA deficiency ($<0.05 \text{ mg dL}^{-1}$). Another seven cases displayed relative IgA deficiencies, with mean IgA concentrations ranging from 0.39 to 3.70 mg dL^{-1} . Anti-IgA was identified in 2 of the 14 IgA-deficient blood donors whose IgA levels were $<5 \text{ mg dL}^{-1}$. Estimation of the theoretical risk for IgA anaphylactic transfusion reaction was 0.009%.

Conclusion: The prevalence of IgA deficiency in Chinese is low. However, potential risks exist in performing blood transfusion to IgA-deficient persons, and measures should be taken to reduce IgA anaphylaxis.

Key words: anti-IgA, IgA deficiency, transfusion reaction.

Selective immunoglobulin A deficiency (SIgAD) is the most common primary immunodeficiency. A full 90% of patients with SIgAD are asymptomatic, although those who suffer clinical manifestations often also have various complications, such as recurrent respiratory infections, allergic disorders and autoimmune diseases (Cunningham-Rundles, 2001; Aghamohammadi *et al.*, 2009). Such patients may even progress to common variable immunodeficiency, which is an even more severe immunodeficiency (Hammarström *et al.*, 2000). SIgAD is also present in healthy individuals, with varying racial prevalence from as high as 1 : 328 in Americans (Clark *et al.*, 1983) to as low as 1 : 18 500 in Japanese (Kanoh *et al.*, 1986). Most laboratories define an immunoglobulin A (IgA) concentration of

$<5 \text{ mg dL}^{-1}$ as IgA deficiency and a concentration of $<0.05 \text{ mg dL}^{-1}$ as absolute IgA deficiency (Sandler, 2006).

Many absolute IgA-deficient patients develop anti-IgA antibodies, which can increase the risk of severe transfusion reaction to normal IgA-containing blood components (Laschinger *et al.*, 1984; Sandler *et al.*, 1995). Three approaches have been employed to transfuse IgA-deficient patients with anti-IgA: (i) autologous blood transfusion, (ii) transfusion with fully washed (up to five times) red blood cell (RBC) or platelet (PLT) components or (iii) transfusion components from IgA-poor blood donors (Foudoulaki-Papazis *et al.*, 2002). The former two methods can be considered for urgent transfusion needs or when IgA-deficient blood is unavailable. However, the safest alternative is the transfusion of absolute IgA-deficient blood components without anti-IgA antibody (Sandler, 2006). This is because autologous blood is not available for patients with excessive blood loss, and washing RBCs and PLTs is time-consuming, laborious and limits their activity. Many countries have developed large-scale detection methods

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for IgA deficiency, and registries of IgA-deficient donors have been established in Canada, the United States and in some European countries (Thibault *et al.*, 2006). These actions support the rare occurrence of IgA-negative blood transfusion to IgA-deficient persons, minimising the incidence rate of adverse transfusion reactions and enhancing blood safety.

Few studies have performed large-scale screening for IgA deficiency in healthy Chinese blood donors, and no specialised detection of IgA deficiency or anti-IgA is performed for routine plasma samples in national transfusion services. The purpose of this study was therefore to determine the frequency of IgA deficiency in the Chinese population. Healthy volunteer blood donations were screened for IgA deficiency, and the DiaMed particle gel immunoassays were used to detect anti-IgA in IgA-deficient individuals and to estimate the risk of anaphylactic transfusion reaction.

MATERIALS AND METHODS

Subjects studied and serum sample collection

Blood samples from 22 609 healthy volunteer blood donors already located at Shanghai Blood Center were taken according to routine procedure and tested within 1 week from collection, excluding haemolysed and clotted samples. These donors were recruited from Shanghai and surrounding areas and all of them were Han Chinese population. Written informed consent was provided in recruiting blood donors, and the work was approved by the ethics committee of the service. Serum from each sample was diluted in 0.9% NaCl (1 : 10 000 dilutions).

ELISA for screening IgA-deficient serum samples

As described previously (Sanz *et al.*, 1999; Thibault *et al.*, 2006), the prepared test samples, positive and negative controls were added to the commercial 96-well microplate coated with goat anti-human IgA antibody (Bethyl Laboratories, Montgomery, TX, USA). After incubation and washes, 100 µL of 1 : 50 000 horseradish peroxidase-conjugated goat anti-human IgA antibody (Bethyl Laboratories) was then added and incubated for 1 h. The enzyme substrate reaction was performed by adding 100 µL of 3, 3', 5, 5' - tetramethylbenzidine liquid substrate and then stopped with 50 µL of 2N H₂SO₄ (BioFX Laboratories Inc., Owings Mills, MD, USA). The plate was read at 450 nm using a SpectraMax Absorbance microplate reader (Molecular Devices, Sunnyvale, CA, USA).

To standardise the assay, human reference serum (Bethyl Laboratories) was diluted by a serial two-fold dilution resulting in final IgA concentrations ranging from 500 to 7.8 ng mL⁻¹. Samples with the optical

density (OD) values below the final concentration in the standards solution were considered to be IgA deficiency and retested as above. Samples that repeatedly tested as IgA deficient by enzyme-linked immunosorbent assay (ELISA) were stored frozen at -20 °C in 0.1% NaN₃ for further confirmation.

DiaMed ID-PaGIA IgA deficiency test for confirmation and anti-IgA test

The test samples to be confirmed were thawed and centrifuged. According to the manufacturer's instructions for the IgA-deficiency test (DiaMed GmbH, Cressier FR, Switzerland), 10 µL of each sample or control was added to identified reaction chambers of the gel card, followed by 50 µL of the particle suspension. After a 5-min incubation at room temperature, the gel card was centrifuged for 10 min (ID-Centrifuge 12 SII, DiaMed). The results were read and recorded as indicated the manufacturer's instructions. The anti-IgA test was performed similarly to that for IgA deficiency detection.

Statistical analysis

The OD value of each well, mean concentration, standard deviation (SD) and coefficient of variation (CV) percent of IgA was detected and analysed with SoftMax Pro software 4.8 (Molecular Devices). All these data were obtained using computer program Microsoft Office Excel for tabulated statistics and management.

RESULTS

ELISA standard curve and assay assessment

Using the microplate reader, averages were obtained from duplicate readings of each standard, blank control and sample. A standard curve relating the IgA concentration to the mean OD value was created using computer software and a four-parameter logistic curve fit (Fig. 1). A standard curve was generated for each sample batch. The OD value of the highest standard concentration was approximately 2.0, that of the lowest standard concentration was above the background and the curve displayed a good fit ($r^2 = 1$). A CV range of 0.4–2.8% for IgA concentrations of 500–7.813 ng mL⁻¹ revealed an ideal precision. The assay sensitivity was high, as indicated by an approximately 100 fold difference between the OD values of the lowest IgA concentration and the IgA-deficient samples (or blank control).

Large-scale screening for IgA deficiency in healthy blood donors

A quantitative screening for IgA deficiency of 22 609 healthy blood donors was initially performed using a

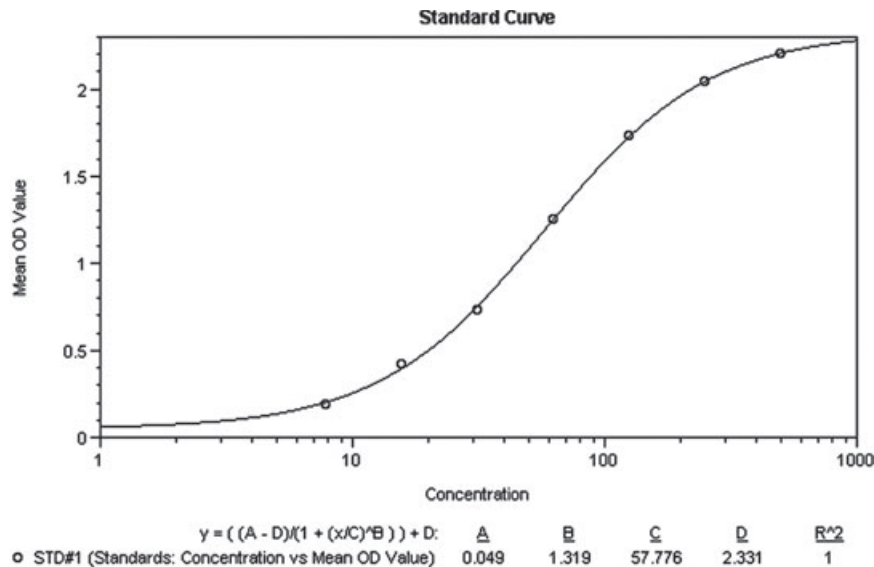


Fig. 1. IgA standard curve. Each point represents a mean OD value $\pm$ SD (at 450 nm) at each dilution.

simple in-house sandwich ELISA. The average IgA level in non-IgA-deficient blood donors was $216.60 \pm 198.95 \text{ mg dL}^{-1}$ (mean $\pm$ SD). Fourteen IgA-deficient samples, in which the OD values were between those of the lowest IgA concentration in standards and the blank control, were identified by ELISA. According to the date screened, the deficient samples were identified as SBC001, SBC002...SBC014. Seven of the samples were confirmed as absolute IgA deficient by the DiaMed. The remaining seven samples, which displayed concentrations ranging from 0.39 to 3.70 mg dL^{-1} , were defined as relative IgA deficient according to previously published definitions (Sandler, 2006) (Table 1). One sample (SBC004, Table 1) that displayed an IgA level of 0.037 mg dL^{-1} by ELISA failed to be classified as absolute IgA deficient by the DiaMed assay. The reason for this discrepancy may be that the sample value was close to the cut-off or some unknown reason. The frequency of IgA deficiency in the screened blood donors is shown in Table 2.

Anti-IgA antibodies in IgA-deficient blood samples

The 14 absolute and relative IgA-deficient serum samples were tested for anti-IgA antibodies by the DiaMed assay. Of the 14 IgA-deficient samples, 2 (14.3%) were weakly positive for anti-IgA, both of which were from relative IgA-deficient donors. None of absolute IgA-deficient donors had detectable levels of anti-IgA.

Evaluation of theoretical risk of IgA anaphylactic transfusion reaction

On the basis of the data obtained, the frequency of IgA-deficient persons with detectable anti-IgA among

Table 1. IgA level in relative IgA-deficient blood donors by ELISA¹

Sample	Mean concentration (ng mL ⁻¹) ²	Adjusted concentration (ng mL ⁻¹) ³	CV (%)
SBC001	89.618 ± 10.6	8961.753	12.0
SBC004	3.687 ± 0.283	368.737	7.7
SBC006	47.807 ± 3.042	4780.721	6.4
SBC009	112.27 ± 3.733	11 226.953	3.3
SBC011	38.576 ± 1.929	3857.579	5.0
SBC013	41.373 ± 1.993	4137.276	4.8
SBC014	369.579 ± 25.123	36 957.945	6.8

¹One microplate. IgA levels in relative IgA deficiency range 0.05 – 5 mg dL^{-1} (500 – $50\,000 \text{ ng mL}^{-1}$).

²Mean $\pm$ SD of three wells.

³Final concentration of IgA in relative IgA-deficient donors. Before testing, samples were diluted 1 : 100; thus, all data were in the linear range.

Table 2. Frequency of SIgAD in Chinese blood donors by ELISA

	Number of samples	Frequency
Total study samples	22 609	–
IgA deficient by ELISA	14	1 : 1615
Absolute IgA deficient (<0.05 mg dL ⁻¹)	7	1 : 3230
Relative IgA deficient (0.05–5 mg dL ⁻¹)	7	1 : 3230

22 609 blood donors was 1 : 11 305. The theoretical incidence of IgA anaphylactic transfusion reaction in IgA-deficient recipients with anti-IgA after transfusion

of IgA-containing blood components was approximately 0.009% (the frequency of anti-IgA in donors multiplied by percentage of non-IgA deficiency).

DISCUSSION

The prevalence of SIgAD varies significantly in different areas and among races (Table 3); however, this incidence has not been previously quantified in the healthy Chinese blood population. In this study, a large-scale screening of healthy blood donors for IgA deficiency was performed. The frequency of IgA deficiency ($<5 \text{ mg dL}^{-1}$) in Chinese healthy blood donors was 1 : 1615, and that of absolute IgA deficiency was 1 : 3230. Feng *et al.* (1992) reported an SIgAD (IgA $<5 \text{ mg dL}^{-1}$) prevalence of 1 : 4100 in an epidemiological study of six nationalities in China, with differences among the different subpopulations (e.g. prevalence in the Hans was 1 : 2600). The results here are comparable with those of this previous report. Although a strict criteria for IgA deficiency and healthy blood donors as object of study were employed in this study, as well as relation to safe blood transfusion. IgA-deficient patients we found were adults rather than children reported in Feng's study. The results of this work are also consistent with the observation that IgA deficiency is rare in Asians compared with Caucasians. However, the frequency in the Chinese is higher than that in other studied Asian countries. These

results strongly suggest a genetic influence on disease development (Mestecky & Hammarström, 2008). Differences in the methods and criteria used may also be responsible for the diverse data observed in different races, as also shown in Table 3.

Anaphylaxis is an important immune transfusion adverse reaction that is mainly caused by immunodeficiency, particularly IgA defects (Horn *et al.*, 2007). In many studies, the occurrence of transfusion adverse reactions is estimated by the presence of detectable anti-IgA in IgA-deficient patients (IgA $< 0.05 \text{ mg dL}^{-1}$). To diagnose and prevent suspected IgA anaphylactic transfusion reactions, identification of IgA deficiency and subsequent determination of anti-IgA levels in these persons is an efficient and safe alternative. Passive haemagglutination assay (PHA) is the traditional strategy for detecting the presence of anti-IgA. Unfortunately, the PHA test requires the use of specialised laboratories and costly instruments. Furthermore, Sandler *et al.* (1994) suggested that the use of PHA to detect anti-IgA overestimates the risk of anaphylactic transfusion reaction. The novel DiaMed particle immunoassay was recently evaluated by comparison with the standard PHA method (Brown *et al.*, 2008). We used the method to determine anti-IgA and obtained the frequency (14.3%) of anti-IgA in IgA-deficient (IgA $< 5 \text{ mg dL}^{-1}$) blood donors. This is lower than other reports in south-west England (Holt *et al.*, 1977), Australia (Wells *et al.*, 1980), the United

Table 3. Prevalence of IgA deficiency among healthy blood donors in different ethnic groups from 1977 to date

Country	Sample size	Method	Prevalence	Criteria (mg dL^{-1})	Anti-IgA
South-west England (Holt <i>et al.</i> , 1977)	29 745	Double diffusion Haemagglutination inhibition assay	1 : 522 1 : 875	400 5	29.0% ¹
Australia (Wells <i>et al.</i> , 1980)	6191	Double diffusion	1 : 442	5	27.0% ¹
France (Ropars <i>et al.</i> , 1982)	108 000	Passive haemagglutination inhibition assay	1 : 1300	0.02	5.0% ¹
United States (Clark <i>et al.</i> , 1983)	6240	Double diffusion	1 : 328	5	36.8% ¹
Japan (Kano <i>et al.</i> , 1986)	222 597	Double diffusion Single radial immunodiffusion	1 : 18 500 1 : 31 800	5 1	25.0% ¹
Spain (Sanz <i>et al.</i> , 1999)	20 000	ELISA	1 : 625	0.05	NT
Greek (Foudoulaki-Papazios <i>et al.</i> , 2002)	5000	Nephelometry	1 : 455	0.1	28.0% ²
Canada (Thibault <i>et al.</i> , 2006)	38 759	ELISA	1 : 531	0.05	53.0% ¹
India (Chaudhary <i>et al.</i> , 2005)	3818	ELISA	0	5	NT
Portugal (Duran <i>et al.</i> , 2008)	4906	Nephelometry	1 : 327	0.02	NT
Iran (Rezvan <i>et al.</i> , 2009)	8852	Immunodiffusion	1 : 983	5	NT
China (this study)	22 609	ELISA DiaMed ID-PaGIA	1 : 1615 1 : 3230	5 0.05	14.3% ²

NT, not tested.

¹Detected by PHA.

²Detected by DiaMed.

States (Clark *et al.*, 1983) and Japan (Kano *et al.*, 1986) at an equivalent definition of 5 mg dL⁻¹ for IgA deficiency (Table 3). Different populations and the method we used may be responsible for the low proportion of anti-IgA in this study. Although the DiaMed assay cannot quantitate the level of IgA antibodies, it is readily available providing an effective and simple strategy for diagnosis of IgA anaphylaxis combined with DiaMed IgA deficiency test.

The mechanism of anti-IgA production remains unknown. Some studies have shown that IgA-deficient patients with anti-IgA antibodies had a history of pregnancy, transfusion or Ig administration, although those who have never been exposed to IgA can also develop anti-IgA antibodies (Sanz *et al.*, 1999; Thibault *et al.*, 2006). IgA-deficient persons without detectable anti-IgA may form antibodies against IgA with sensitisation, and will be at risk of IgA anaphylactic reaction when received multiple transfusions. What level of anti-IgA are clinically significant remains unpredictable (Shan & Goldfinger, 2008). In this study, 2 in 22 609 (0.009%) blood donors were IgA deficient and also had anti-IgA determined by DiaMed assay. We hereby assume that the anti-IgA is clinically significant and hence expect that of the order of 0.009% cases will be at potential risk for the IgA anaphylactic transfusion reactions. The reactions occur in IgA-deficient patients with anti-IgA after they receive normal IgA-containing blood components transfusion. Thus, we estimate that the theoretical incidence of IgA anaphylaxis may be calculated from frequency of IgA deficiency with anti-IgA (0.009%) multiplied by the frequency of non-IgA deficiency (99.9%). Persons with normal IgA level as recipients transfused with IgA-deficient blood containing anti-IgA are not taken into consideration for estimation of the risk because of recipient plasma dilution of the antibodies (Winters *et al.*, 2004).

In conclusion, we provide the first report of the prevalence of IgA deficiency in a Chinese population by

screening healthy blood donors, and estimate the risk of transfusion reaction induced by anti-IgA. These results showed a low frequency rate of IgA deficiency in China, and the incidence of IgA deficiency (<5 mg dL⁻¹) with anti-IgA was 1 : 11 305 and without anti-IgA was 1 : 1884 using DiaMed assay. No anti-IgA has been detectable in absolute IgA-deficient blood donors. However, IgA-deficient patients readily develop anti-IgA after multiple transfusions, which may induce the occurrence of adverse reactions (even shock and death). Anti-IgA-mediated anaphylactic reaction accounts for 3.1% of transfusion-related deaths (Brown *et al.*, 2008). Three cases of transfusion reactions in IgA-deficient patients have been clinically reported in China (Wei *et al.*, 1996; Wang *et al.*, 2001; Xiao, 2004). It showed a potential risk exists in performing blood transfusion to Chinese IgA-deficient persons. Strategies are needed to prevent and reduce the risk of anaphylaxis. For the purpose of safe and effective blood transfusion, we suggest that transfusion services in China should actively develop the screening for IgA-deficient blood donors in case of need for IgA-poor blood products in the future. If urticaria or anaphylaxis suggests IgA deficiency, clinicians are advised to measure IgA levels and anti-IgA in IgA-deficient recipients before transfusion.

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CONFLICT OF INTEREST

The authors have no competing interests.

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