

ORIGINAL ARTICLE

Association between ABO Blood Groups and Transfusion-Transmitted Infections Among Blood Donors in Nantong, China

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SUMMARY

Background: ABO blood groups have certain connections with the occurrence of various infectious diseases. This article mainly explored the association between transfusion-transmitted infections (TTIs) and ABO blood groups and identified other possible influencing factors.

Methods: This study retrospectively analyzed the TTIs results of 257,117 blood donors and analyzed the relationship between age, gender, and blood groups and TTIs.

Results: The *treponema pallidum* (TP) infection rate in A and AB blood donors were significantly higher than that in B and O blood donors ($p = 0.023$), and the TP infection rate in women was significantly higher than that in men ($p = 0.007$). Age is an independent risk factor for HBsAg and TP infection [OR (95% CI): 1.4 (1.32 - 1.49), 1.44 (1.38 - 1.51)], and the infection rate gradually increases with age (p for trend 0.001). There may be a significant association between blood group A and TP infection risk [adj. OR (95% CI): 1.13 (1 - 1.28), $p = 0.045$], and the risk of blood group A increased by 13% compared with non-A group. Blood group A is an independent risk factor for TP infection. There is a significant association between blood group AB and hepatitis B virus (HBV) infection risk [adj. OR (95% CI): 0.71 (0.52 - 0.96), $p = 0.026$]; blood group AB has a 29% lower risk of infection compared with the non-AB group, so blood group AB is an independent protective factor against HBsAg infection.

Conclusions: The positive rate of TTIs among local blood donors is related to blood group and age, which has certain guiding significance for the recruitment of blood donor TTIs screening.

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KEYWORDS

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LIST OF ABBREVIATIONS

TTIs - transfusion-transmitted infections
TP - *treponema pallidum*
HBV - hepatitis B virus
HCV - hepatitis C virus
HIV - human immunodeficiency virus
WHO - World Health Organization
RBCs - red blood cells

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INTRODUCTION

Transfusion-transmitted infections (TTIs), including HBV, hepatitis C (HCV), human immunodeficiency virus (HIV) infection, and treponema pallidum (TP), are the most serious complications that can result from blood transfusions, posing a significant threat to patients' health [1]. According to global statistics, approximately 2 billion individuals are infected with HBV, 150 million with HCV, 33 million with HIV, and about 12 million annually with TP [2-5]. TTIs have a serious impact on the quality of life of the blood recipients and impose a considerable economic burden on society [6]. Unqualified blood products represent the primary cause of TTIs [7]. In the 1990s, the World Health Organization (WHO) recommended that blood products used for blood transfusion therapy should be screened for HBV, HCV, HIV, and TP to ensure the safety of blood products.

The surface of red blood cells (RBCs) contains various blood group antigens composed of polysaccharides and proteins. In addition to their expression on RBC, blood groups antigens are also expressed in tissues or cells such as sensory neurons, platelets, vascular endothelial cells, malignant cells, and so on [8,9]. Among the identified blood group antigens, the ABO system is of paramount importance within the human blood group system. Apart from its critical clinical significance in blood transfusion and transplantation, research has revealed associations between blood group antigens and numerous diseases, including cancer, cardiovascular disease, infection, hematologic disorders, cognitive impairment, circulatory diseases, metabolic conditions, and malaria [10]. There are numerous literature reports on the correlation between blood groups and TTIs, yet the results are not completely consistent [11-16]. In view of this, this paper collected relevant test results for blood donors in Nantong, China, to explore the distribution characteristics of HBV, HCV, HIV, and TP in blood donors in order to improve blood transfusion safety.

MATERIALS AND METHODS

General information

To ensure blood quality and safety, all donors completed a detailed donor health questionnaire prior to donating blood, which includes inquiries about family history, clinical conditions, and infection-related conditions. All donors in this study were consulted and evaluated according to standard operating procedures prior to donating blood. The inclusion criteria for blood donors were as follows: aged between 18 - 55 years old (those who donate multiple times may extend to 60 years); weight: male \geq 50 kg, female: \geq 45 kg; blood pressure within the range of 90 - 140/60 - 90 mm Hg; and a pulse pressure difference of \geq 30 mm Hg.

We excluded donors with various infectious diseases, chronic inflammation, history of blood transfusion,

pregnancy of less than six months, a history of blood transfusion within the past five years, a history of drug abuse, participation in high-risk activities, tumors, and other diseases deemed unsuitable for blood donation by the medical examination doctor. All donors had to provide written informed consent before donation. Throughout the donation process, donors were required to adhere to various pre-donation procedures to ensure safety and prevent any adverse events.

Methods

Peripheral venous blood (5 mL) was collected by blood donors, and the enzyme-linked immunosorbent assay was conducted for the detection of HBsAg, HCV-Ab, HIV-Ab, and TP-Ab (Beijing Wantai Biopharmaceutical Co., Ltd.). ABO blood groups of blood donors were identified by agglutination assay: Anti-A and anti-B standard serum were used to detect ABO antigen on the surface of red blood cells in blood donors, and A and B standard red blood cells were used to detect anti-A and B antibodies in the serum. The anti-A and anti-B blood groups typing reagents and the ABO blood groups anti-forming red blood cell kits were both from Shanghai Blood Biopharmaceutical Co., Ltd.

During blood collection and testing, all biosafety preventive measures and infection control protocols were followed. Data from 257,117 blood donors who donated blood at Nantong Blood Center from January 2019 through December 2023, including age, gender, ABO blood groups, and TTIs markers results, were collected from the Nantong Blood Center information system. All records were screened based on ID numbers, and repeat blood donors' records were retained with only the most recent sample test results.

Given that the frequency of Rh(D) negativity among the Han population was between 0.2% - 0.5%, the case data collected for this study consisted solely of Han individuals. Consequently, we did not undertake research on Rh(D) blood groups. Furthermore, although nucleic acid tests of TTIs were performed concurrently, a positive result in any of these tests classified blood donors as unqualified; as a result, nucleic acid testing was not conducted on the entire population, leading to a significant number of missing nucleic acid test results. Hence, these nucleic acid results were excluded from the present study.

This study was carried out in accordance with the declaration of Helsinki and was approved by the ethics committee of the Affiliated Hospital of Nantong University (2024-K200-01). Due to the retrospective nature of the study, written informed consent was exempted.

Statistical analysis

This study conducted statistical analysis on the data using SPSS 20.0. Continuous variables were normally distributed and expressed as $\bar{x} \pm s$, and the comparison between groups was conducted by *t*-test; categorical data is represented by *n* (%), and chi-squared test was used for comparison between groups. No missing vari-

ables were included in this study. $p < 0.05$ was considered statistically significant.

RESULTS

Distribution of the positive rates of TTIs antibodies in blood donors

A total of 257,117 samples were included in this study, with an average age of 37.8 ± 11.6 years, out of which 143,586 were male, accounting for 55.8%, and 113,531 were female, accounting for 44.2% (Table 1). From the perspective of blood group distribution, blood group O was the most common one, accounting for 31.5%, followed by group A (30.8%), group B (27.9%), and group AB (9.8%). The mean age of HBsAg- and TP-positive individuals was higher than that of -negative individuals ($p < 0.001$). The infection rate of TP was significantly correlated with gender and blood groups ($p = 0.007$, $p = 0.023$). Among the infected individuals, TP-Ab had the highest infection rate (0.50%), followed by HBsAg (0.20%), HCV-Ab (0.20%), and HIV-Ab (0.10%). The infection rates of TP in different blood groups were statistically significant. The infection rates of TP in group A and group AB were significantly higher than those in group B and O ($p = 0.023$), and the infection rates of TP in women were significantly higher than those in men ($p = 0.007$). There was no significant difference in the infection rate of HBsAg among the different genders and the four blood groups ($p = 0.126$, $p = 0.099$) and no difference in the infection rate of HCV and HIV in age, gender, and the four blood groups. (p -values were all > 0.05), as shown in Table 2.

The relationship between the positive rate of TTIs antibodies in blood donors and age

As the infection rate of TTIs is significantly correlated with age, we grouped age by quintile and found that there were statistical differences in the infection rates of HBsAg, HCV, and TP among different age groups ($p < 0.001$, $p = 0.002$, $p < 0.001$). We witnessed an interesting phenomenon: the infection rates of HBsAg and TP gradually increased with age ($p < 0.001$), and the HCV infection rates of people aged 18 - 33 and 50 - 60 were significantly higher than those of people aged 34 - 49 ($p = 0.002$), as shown in Table 3.

We calculated the infection rates for each age group and used the logistic regression model to estimate the crude odds ratio (crude OR) and adjusted odds ratio (adj. OR) respectively to evaluate the impact of age on infection risk, as shown in Table 4. The crude OR did not consider other potential confounders, while the adj. OR included two variables of gender and blood group in the model to more accurately reflect the independent effects of age. As shown in this table, compared with group 1 (reference group), the risk of HBsAg infection in group 2 increased slightly when the confounding factors were not adjusted, but the difference was statistically nonsignificant [crude OR (95% CI): 1.18 (0.82 - 1.71), $p =$

0.373]. However, starting from group 3, the crude OR was significantly increased [crude OR (95% CI): 2.58 (1.85 - 3.58), $p < 0.001$]; the risk of infection in this group was about 1.58 times that of group 1, and the difference was statistically significant. The crude OR of group 4 and group 5 increased further, and the infection risk was 2.36 times and 2.69 times that of group 1 [crude OR (95% CI): 3.26 (2.37 - 4.47); crude OR (95% CI): 3.69 (2.7 - 5.05), p all < 0.001 , respectively]. After adjusting for gender and blood group, the adj. OR and crude OR of each group showed little change, and the infection rate of HBsAg still showed a significant increasing trend with age (p for trend < 0.001). It shows that age is an independent risk factor for HBV infection. Age had similar effects on the infection rates of TP. After adjusting for gender and blood group, the adj. ORs of each group were basically consistent with crude ORs, showing a significant increasing trend with age. In the calibration model, compared with group 1, the risk of TP infection in group 2 increased by 37% (95% CI: 1.04 - 1.8, $p = 0.023$), the risk of TP infection in group 3 increased by 135% (95% CI: 1.83 - 3.02), the risk of TP infection in group 4 increased by 251% (95% CI: 2.77 - 4.45), and the risk of TP infection in group 5 increased by 328% (95% CI: 3.39 - 5.4, all $p < 0.001$). Consequently, with the increase of age, the risk of TP infection increased significantly [p for trend < 0.001 , crude OR (95% CI): 1.45 (1.38 - 1.51); adj. OR (95% CI): 1.44 (1.38 - 1.51)], indicating that age is an independent risk factor for TP infection.

HCV infection rate fluctuated among different age groups. The infection rate was 0.2% in group 1 and 2, decreased to 0.1% in groups 3 and 4, but increased to 0.2% in group 5. The risk of infection in group 2 was lower than that in group 1, but the difference was statistically nonsignificant [crude OR (95% CI): 0.81 (0.61 - 1.08), $p = 0.150$]. The risk of HCV infection in group 3 was 32% lower than that in group 1, and the difference was statistically significant [crude OR (95% CI): 0.68 (0.5 - 0.93), $p = 0.014$].

The infection risk of group 4 was reduced by about 46% compared with group 1, and the difference was significant [crude OR (95% CI): 0.54, 0.39 - 0.74, $p < 0.001$]. The risk of infection in group 5 was 21% lower than that in group 1, and the difference was statistically nonsignificant [crude OR (95% CI): 0.79 (0.6 - 1.05), $p = 0.109$]. After adjusting the model, the adj. OR and crude OR of group 3, group 4, and group 5 were not significantly different [adj. OR (95% CI): 0.68 (0.50 - 0.93), $p = 0.014$; 0.53 (0.39 - 0.74), $p < 0.001$; 0.79 (0.59 - 1.05), $p = 0.105$], and the trend of infection risk reduction remained unchanged. The abovementioned results suggest that the infection rate of HCV fluctuates in different age groups, but with the increase of age, the risk of HCV infection gradually decreases with statistical significance [adj. OR (95% CI): 0.91 (0.85 - 0.98), p for trend = 0.011], indicating that age is a protective factor of HCV infection.

Table 1. Distribution of TTIs rates by age, gender, and blood groups.

Variables	Total (n = 257,117)	HCV-Ab		HIV-Ab		TP-Ab		Statis- tic
		Negative (n = 260,066)	Positive (n = 418)	Statis- tic	Positive (n = 285)	Statis- tic	Positive (n = 1,206)	
Age, mean \pm SD	37.8 \pm 11.6	37.8 \pm 11.6	36.7 \pm 12.2	0.056	3.666	37.8 \pm 11.6	38.0 \pm 11.3	0.735
Gender, n (%)				0.476	0.508			0.161
Female	113,531 (44.2)	113,341 (44.2)	190 (45.9)			113,419 (44.2)	112 (40)	
Male	143,586 (55.8)	143,362 (55.8)	224 (54.1)			143,418 (55.8)	168 (60)	
Blood group, n (%)				0.708	1.389			0.075
A	79,250 (30.8)	79,123 (30.8)	127 (30.7)			79,176 (30.8)	74 (26.4)	
B	71,740 (27.9)	71,618 (27.9)	122 (29.5)			71,669 (27.9)	71 (25.4)	
O	81,029 (31.5)	80,898 (31.5)	131 (31.6)			80,931 (31.5)	98 (35)	
AB	25,098 (9.8)	25,064 (9.8)	34 (8.2)			25,061 (9.8)	37 (13.2)	

Table 1. Distribution of TTIs rates by age, gender, and blood groups (continued).

Variables	HBsAg		p	Statistic
	Total (n = 257,117)	Negative (n = 256,485)		
Age, mean \pm SD	37.8 \pm 11.6	37.8 \pm 11.6	42.8 \pm 10.2	< 0.001
Gender, n (%)				120.71
Female	113,531 (44.2)	113,271 (44.2)	0.126	2.369
Male	143,586 (55.8)	143,214 (55.8)	372 (58.9)	
Blood group, n (%)				0.099
A	79,250 (30.8)	79,048 (30.8)	202 (32)	6.284
B	71,740 (27.9)	71,570 (27.9)	170 (26.9)	
O	81,029 (31.5)	80,814 (31.5)	215 (34)	
AB	25,098 (9.8)	25,053 (9.8)	45 (7.1)	

Table 2. Vertically-arranged distribution by blood groups and gender.

Variables	Total (n = 257,117)	Blood groups				p	Statistic	Gender		p
		A (n = 79,250)	B (n = 71,740)	O (n = 81,029)	AB (n = 25,098)			Female (n = 113,531)	Male (n = 143,586)	
HBsAg, n (%)						0.099	6.284			0.126
Negative	256,485 (99.8)	79,048 (99.7)	71,570 (99.8)	80,814 (99.7)	25,053 (99.8)			113,271 (99.8)	143,214 (99.7)	
Positive	632 (0.20)	202 (0.30)	170 (0.20)	215 (0.30)	45 (0.20)			260 (0.20)	372 (0.3)	
HCV-Ab, n (%)						0.708	1.389			0.476
Negative	256,703 (99.8)	79,123 (99.8)	71,618 (99.8)	80,898 (99.8)	25,064 (99.9)			113,341 (99.8)	143,362 (99.8)	
Positive	414 (0.20)	127 (0.2)	122 (0.2)	131 (0.20)	34 (0.10)			190 (0.20)	224 (0.2)	
HIV-Ab, n (%)						0.075	6.911			0.161
Negative	256,837 (99.9)	79,176 (99.9)	71,669 (99.9)	80,931 (99.9)	25,061 (99.9)			113,419 (99.9)	143,418 (99.9)	
Positive	280 (0.10)	74 (0.1)	71 (0.10)	98 (0.10)	37 (0.10)			112 (0.10)	168 (0.10)	
TP-Ab, n (%)						0.023	9.499			0.007
Negative	255,926 (99.5)	78,850 (99.5)	71,437 (99.6)	80,675 (99.6)	24,964 (99.5)			112,959 (99.5)	142,967 (99.6)	
Positive	1,191 (0.50)	400 (0.50)	303 (0.40)	354 (0.40)	134 (0.50)			572 (0.50)	619 (0.40)	

Table 3. Distribution of TTIs rates in different age groups.

Variables	Total (n = 257,117)	Group 1	Group 2	Group 3	Group 4	Group 5	p
		18 - 24 years (n = 47,220)	25 - 33 years (n = 53,480)	34 - 40 years (n = 49,098)	41 - 49 years (n = 53,679)	50 - 60 years (n = 53,640)	
HBsAg, n (%)							< 0.001
Negative	256,485 (99.8)	47,171 (99.9)	53,414 (99.9)	48,967 (99.7)	53,498 (99.7)	53,435 (99.6)	
Positive	632 (0.2)	49 (0.1)	66 (0.1)	131 (0.3)	181 (0.3)	205 (0.4)	
HCV-Ab, n (%)							0.002
Negative	256,703 (99.8)	47,120 (99.8)	53,388 (99.8)	49,027 (99.9)	53,618 (99.9)	53,550 (99.8)	
Positive	414 (0.2)	100 (0.2)	92 (0.2)	71 (0.1)	61 (0.1)	90 (0.2)	
HIVAb, n (%)							0.181
Negative	256,837 (99.9)	47,180 (99.9)	53,408 (99.9)	49,045 (99.9)	53,625 (99.9)	53,579 (99.9)	
Positive	280 (0.1)	40 (0.1)	72 (0.1)	53 (0.1)	54 (0.1)	61 (0.1)	
TP-Ab, n (%)							< 0.001
Negative	255,926 (99.5)	47,134 (99.8)	53,347 (99.8)	48,888 (99.6)	53,335 (99.4)	53,222 (99.2)	
Positive	1,191 (0.5)	86 (0.2)	133 (0.2)	210 (0.4)	344 (0.6)	418 (0.8)	

The relationship between the positive rate of TTIs antibodies and blood groups in blood donors

We tried to group blood groups according to A, non-A, B, non-B, O, non-O, AB, and non-AB. We found that compared with group non-A, the TP-Ab positive rate of group A donors was higher, and the difference was statistically significant ($p = 0.038$); compared with non-AB group, the positive rate of HBsAg for group AB donors was lower, and the difference was statistically significant ($p = 0.025$), as shown in Table 5. We further used the logistic regression analysis method. After adjusting for the two potential confounding factors of age and gender, we calculated the adj. OR and 95% CI of each group compared with others to evaluate the independent impact of blood groups on the risk of infection, as shown in Table 6. The infection risk of group A donors increased by about 13% compared with non-A blood donors, (adj. OR: 1.13, 95% CI: 1 - 1.28, $p = 0.045$). This indicates that group A is an independent risk factor for TP infection. The infection risk of group AB was reduced by about 29% compared with non-AB group (adj. OR: 0.71, 95% CI: 0.52 - 0.96, $p = 0.026$). Group AB was an independent protective factor for HBsAg infection. This result means that there is a significant correlation between blood groups and the risk of HBV and TP infection.

DISCUSSION

Since the discovery of the human ABO blood groups system, researchers have conducted extensive discussions on the correlation between blood groups and various diseases. However, the results have been inconsistent, and the mechanism behind the relationships remains a mystery. In this study, we screened 257,117 blood donors and analyzed the relationship between age, gender, and blood groups and the positivity rate of TTIs antibodies.

We observed that the number of male donors exceeded the number of female donors. The most prevalent blood groups among donors were O and A, followed by B and AB, which aligns with the findings of other studies [6, 11,17]. Interestingly, Sana's findings indicated that group A (28.947%) was the most frequent, followed by group O (22.267%) [18], whereas other studies have typically found group B to be most common, followed by blood group O [19]. This discrepancy may stem from the varying distributions of blood group systems in different races and regions.

A study on the demographic characteristics of TTIs among blood donors from 14 different blood banks in China revealed that donors over 36 years of age had a higher risk of TP infection compared to other TTIs.

Table 4. The relationship between each age group and HBsAg, HCV Ab, and TP-Ab.

Variable	n. total	HCV-Ab				TP-Ab					
		n. event %	Crude OR (95% CI)	Crude p	adj. OR (95% CI)	adj. p	n. event %	Crude OR (95% CI)	Crude p	adj. OR (95% CI)	adj. p
Group 1	47,220	100 (0.2)	1 (ref)		1 (ref)		86 (0.2)	1 (ref)		1 (ref)	
Group 2	53,480	92 (0.2)	0.81 (0.61 - 1.08)	0.150	0.81 (0.61 - 1.08)	0.157	133 (0.2)	1.37 (1.04 - 1.79)	0.024	1.37 (1.04 - 1.8)	0.023
Group 3	49,098	71 (0.1)	0.68 (0.5 - 0.93)	0.014	0.68 (0.50 - 0.93)	0.014	210 (0.4)	2.35 (1.83 - 3.03)	< 0.001	2.35 (1.83 - 3.02)	< 0.001
Group 4	53,679	61 (0.1)	0.54 (0.39 - 0.74)	< 0.001	0.53 (0.39 - 0.74)	< 0.001	344 (0.6)	3.53 (2.79 - 4.48)	< 0.001	3.51 (2.77 - 4.45)	< 0.001
Group 5	53,640	90 (0.2)	0.79 (0.6 - 1.05)	0.109	0.79 (0.59 - 1.05)	0.105	418 (0.8)	4.3 (3.41 - 5.43)	< 0.001	4.28 (3.39 - 5.4)	< 0.001
Trend. test	25,7117	414 (0.2)	0.92 (0.86 - 0.98)	0.012	0.91 (0.85 - 0.98)	0.011	1,191 (0.5)	1.45 (1.38 - 1.51)	< 0.001	1.44 (1.38 - 1.51)	< 0.001

Table 4. The relationship between each age group and HBsAg, HCV Ab, and TP-Ab (continued).

Variable	n. total	HBsAg			
		n. event %	Crude OR (95% CI)	Crude p	adj. OR (95% CI)
Group 1	47,220	49 (0.1)	1.19 (0.82 - 1.72)	0.358	1.18 (0.82 - 1.71)
Group 2	53,480	66 (0.1)	2.58 (1.85 - 3.58)	< 0.001	2.58 (1.86 - 3.58)
Group 3	49,098	131 (0.3)	3.26 (2.37 - 4.47)	< 0.001	3.28 (2.39 - 4.5)
Group 4	53,679	181 (0.3)	3.69 (2.7 - 5.05)	< 0.001	3.72 (2.72 - 5.08)
Group 5	53,640	205 (0.4)	1.4 (1.31 - 1.48)	< 0.001	1.4 (1.32 - 1.49)
Trend. test	25,7117				

Crude means unadjusted model, adjusted model is adjusted for gender and blood groups.

Within this group, the infection rate of TP in women was found to be higher than that in men, and gender was identified as an independent predictor of TP infection ($p < 0.05$) [20]. This study bears a striking resemblance to our own findings. In our study, TP emerged as the disease with the highest infection rate, which also increased progressively with age. Notably, the infection rate among individuals over 34 years old and women experienced a significantly rise. Regrettably, this study did not incorporate blood groups analysis, and the sample size was limited to 1,976 participants. Our study indicates that the positivity rate of HBsAg also escalates with age, aligning with the outcomes of Behal's study [21].

We found that the TP infection rate among group A and AB donors were significantly higher than that among group B and O donors. Furthermore, we determined that the risk of TP infection in group A increased by approximately 13% compared with non-A, and that group A is an independent risk factor for TP infection. Conversely, the risk of HBsAg infection in group AB is reduced by

about 29% compared with non-AB groups, and blood group AB is an independent protective factor against HBsAg infection. Similar to our findings, a meta-analysis showed that group B was associated with a lower risk of HBV infection [22]; Hassan reported the lowest incidence of HBV among Iraqi blood donors [23]. However, there are also some divergent research outcomes. Thakur's study found that the prevalence of HBV is not related to ABO blood group antigen [24].

A study of the physical examination population from 31 provinces in China indicated that individuals with blood group O had a higher risk of HBV infection (adj. OR: 1.22, 95% CI: 1.20 - 1.25) [25]; Additionally, a survey of maternal women at confinement centers revealed that those with blood group AB were associated with an increased risk of HBV infection (adj. OR: 1.134, 95% CI: 1.024 - 1.256, $p = 0.016$) [26]. A study conducted in Saudi Arabia revealed an association between ABO blood group and TTIs, particularly noting that blood donors with blood groups O blood have a higher risk of TTIs [12]. Conversely, Biruk's research in Ethiopia

Table 5. Pairwise comparisons of blood groups based on presence and absence of TTIs.

Variables	Total (n = 257,117)	Non-A (n = 177,867)	A (n = 79,250)	Non-B (n = 185,377)	B (n = 71,740)	Sta- tistic	Non-O (n = 176,088)	O (n = 81,029)	Sta- tistic	Non-AB (n = 232,019)	AB (n = 25,058)	p	Sta- tistic
HBsAg, n (%)			0.535	0.386		0.574	0.317		0.175	1.841		0.025	5.017
Negative	256,485 (99.8)	177,437 (99.8)	79,048 (99.7)	184,915 (99.8)	71,570 (99.8)		175,671 (99.8)	80,814 (99.7)		231,432 (99.7)	25,053 (99.8)		
Positive	632 (0.2)	430 (0.2)	202 (0.3)		462 (0.2)	170 (0.2)		417 (0.2)	215 (0.3)		587 (0.3)	45 (0.2)	
HCV-Ab, n (%)			0.949	0.004		0.477	0.506		0.955	0.003		0.288	1.129
Negative	256,703 (99.8)	177,580 (99.8)	79,123 (99.8)	185,085 (99.8)	716,18 (99.8)		175,805 (99.8)	80,898 (99.8)		231,639 (99.8)	25,064 (99.9)		
Positive	414 (0.2)	287 (0.2)	127 (0.2)		292 (0.2)	122 (0.2)		283 (0.2)	131 (0.2)		380 (0.2)	34 (0.1)	
HIV-Ab, n (%)			0.111	2.538		0.342	0.902		0.209	1.578		0.051	3.794
Negative	256,837 (99.9)	177,661 (99.9)	79,176 (99.9)	185,168 (99.9)	71,669 (99.9)		175,906 (99.9)	80,931 (99.9)		231,776 (99.9)	25,061 (99.9)		
Positive	280 (0.1)	206 (0.1)	74 (0.1)		209 (0.1)	71 (0.1)		182 (0.1)	98 (0.1)		243 (0.1)	37 (0.1)	
TP-Ab, n (%)			0.038	4.283		0.058	3.602		0.182	1.779		0.083	3.015
Negative	255,926 (99.5)	177,076 (99.6)	78,850 (99.5)	1,84489 (99.5)	71,437 (99.6)		175,251 (99.5)	80,675 (99.6)		230,962 (99.5)	24,964 (99.5)		
Positive	1,191 (0.5)	791 (0.4)	400 (0.5)		888 (0.5)	303 (0.4)		837 (0.5)	354 (0.4)		1,057 (0.5)	134 (0.5)	

Table 6. Influence of ABO blood groups on TTIs markers.

Vari- able	n. total	HBsAg				HCV				HIV				TP			
		n. event %	adj. OR (95% CI)	adj. p- value	n.event %	adj. OR (95% CI)	adj. p- value	n. event %	adj. OR (95% CI)	adj. p- value	n. event %	adj. OR (95% CI)	adj. p- value	n. event %	adj. OR (95% CI)	adj. p- value	
Non- A	177,867	430 (0.2)	1 (ref)		287 (0.2)	1 (ref)		206 (0.1)	1 (ref)		791 (0.4)	1 (ref)					
A	79,250	202 (0.3)	1.05 (0.89 - 1.24)	0.574	127 (0.2)	0.99 (0.81 - 1.23)	0.958	74 (0.1)	0.81 (0.62 - 1.05)	0.111	400 (0.5)	1.13 (1 - 1.28)	0.045				
Non- B	185,377	462 (0.2)	1 (ref)		292 (0.2)	1 (ref)		209 (0.1)	1 (ref)		888 (0.5)	1 (ref)					
B	71,740	170 (0.2)	0.96 (0.8 - 1.14)	0.620	122 (0.2)	1.08 (0.87 - 1.33)	0.484	71 (0.1)	0.88 (0.67 - 1.15)	0.343	303 (0.4)	0.89 (0.78 - 1.01)	0.072				
Non- O	176,088	417 (0.2)	1 (ref)		283 (0.2)	1 (ref)		182 (0.1)	1 (ref)		837 (0.5)	1 (ref)					
O	81,029	215 (0.3)	1.12 (0.95 - 1.32)	0.179	131 (0.2)	1.01 (0.82 - 1.24)	0.955	98 (0.1)	1.17 (0.92 - 1.5)	0.209	354 (0.4)	0.92 (0.81 - 1.04)	0.172				
Non- AB	232,019	587 (0.3)	1 (ref)		380 (0.2)	1 (ref)		243 (0.1)	1 (ref)		1,057 (0.5)	1 (ref)					
AB	25,098	45 (0.2)	0.71 (0.52 - 0.96)	0.026	34 (0.1)	0.83 (0.58 - 1.17)	0.288	37 (0.1)	1.41 (1 - 1.99)	0.052	134 (0.5)	1.17 (0.98 - 1.41)	0.082				

Crude means unadjusted model, adjusted model is adjusted for gender and age.

found no significant correlation between blood group and TTIs markers among blood donors [27]. In Pakistan, however, group A is notably linked to HIV and HBV infection, whereas group O does not appear to be significantly associated with TTIs and may even offer some protective benefits [16]. Mohammadali discovered that HBV and HIV infection were significantly associated with blood groups in Iranian blood donors, with a higher proportion of HIV Ag/Ab in group A blood donors and a lower proportion in group O blood donors [2]. Additionally, a study among South African blood donors found no association between HIV infection and ABO blood groups, aligning with our findings [28]. The aforementioned studies on the correlation between blood groups and TTIs markers originate from various countries and regions, and the reasons for the stated discrepancies may be attributed to racial differences.

The correlation between blood groups and certain specific infectious diseases has also been reported. Studies have shown that individuals with group O are associated with an increased incidence of cholera, plague, tuberculosis infection, and mumps, while group A is associated with an increased incidence of smallpox and *Pseudomonas aeruginosa* infection [10]. Group B is associated with increased incidence of gonorrhea, tuberculosis, *Streptococcus pneumoniae*, *Escherichia coli*, and *Salmonella* infections. Group AB is associated with the increased incidence of smallpox, *Escherichia coli*, and *Salmonella* infections. These studies show that there is a certain correlation between blood groups and infectious diseases, potentially due to multiple underlying mechanisms. Blood groups can act as receptors and/or auxiliary receptors of the virus and play a direct role in infection. Furthermore, blood group antigen promotes intracellular uptake, signal transduction, or adhesion through the tissue membrane microdomains, thereby influencing viral susceptibility [29]. For instance, norovirus can recognize and bind to B antigens, facilitating the virus's entry into host cells and making individuals with group B more susceptible to norovirus infection. This binding not only impacts the virus's infection efficiency but may also be related to the severity of the disease [30]. Blood group antigens can also influence the virus internalization process. Some studies suggested that blood group antigens may alter the internalization pathway or efficiency of viral particles by interacting with viral surface proteins, thus affecting the number and speed of viruses entering host cells. For example, anti-A antibodies may inhibit the binding of HIV virus and ACE2 on the surface of human cells, which may affect the spread of the virus [31]. The distribution and density of blood group antigens on the cell membrane may affect the formation of membrane micro-regions required for viral replication, thus providing an appropriate microenvironment for viral replication [29,32]. It appears that blood group antigens can influence various aspects of the virus, including recognition, replication, infection, and transmission, through various channels and methods.

There are some limitations in this study. Given that it is

based on observational studies, while blood groups are associated with TTIs, it cannot be concluded that blood groups are the direct cause of changes in disease risk. Secondly, due to the limited data collected, we only adjusted for age, gender, and blood groups, and did not account for other potential confounders. Thirdly, since our research population is based on the data from local blood donors, it lacks universality. The possible mechanism linking blood groups with TTIs will be the subject of further study.

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Declaration of Interest:

The authors have no competing interests.

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