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ABO blood types as a risk factor for severe symptoms of COVID-19 infection

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ABSTRACT

Numerous studies have investigated whether the ABO blood group system influences susceptibility to SARS-CoV-2 infection and disease severity. The association between blood type and COVID-19 is thought to involve both genetic and environmental factors. The present study aimed to evaluate the relationship between ABO blood phenotypes and the severity of COVID-19 among Arab populations, with a particular focus on Iraqi patients.

To assess the association between ABO blood phenotypes and the occurrence and severity of SARS-CoV-2 infection.

A total of 731 patients with COVID-19 confirmed by a positive PCR test were included in the study. A control group consisted of healthy volunteers with negative PCR results and no clinical symptoms of COVID-19. In all participants, ABO blood phenotype was determined. In infected patients, disease severity and mortality were evaluated and analyzed in relation to blood group phenotype.

Patients with blood group A demonstrated a higher risk of COVID-19 infection compared with non-A blood groups. In contrast, individuals with blood group O showed a significantly lower incidence of COVID-19 infection and reduced disease severity (odds ratio [OR] = 0.686; 95% confidence interval [CI]: 0.521-0.902). No statistically significant differences were observed for other blood groups. Among critically ill patients, blood group A was associated with a significantly higher mortality rate compared with non-A blood groups (OR = 3.341; 95% CI: 1.416-8.158).

Blood group A is associated with an increased risk of severe COVID-19 manifestations, including pneumonia and mortality, whereas blood group O appears to confer a protective effect against severe disease.

INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a global viral pandemic associated primarily with severe acute respiratory illness that emerged in late 2019 [1]. According to the Centers for Disease Control and Prevention (CDC), more than 500 million confirmed cases have been reported worldwide. Although effective vaccines have been developed and a large proportion of the global population has received primary and booster doses, COVID-19 cases continue to occur, and infections were still being reported at the time this study was conducted. While the overall intensity of the pandemic has declined, the emergence of new viral variants continues to pose a risk of renewed transmission, morbidity, mortality, and the reintroduction of public health restrictions [2,3].

In Iraq, approximately 2.33 million COVID-19 cases have been reported, with an estimated 25,320 associated deaths. The pandemic caused substantial strain on healthcare systems worldwide; in some countries, health services collapsed under the burden, while others experienced severe economic disruption due to prolonged lockdowns and preventive measures [4,5].

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COVID-19 presents with a wide spectrum of clinical manifestations, ranging from asymptomatic infection and mild disease to moderate illness, such as viral pneumonia, and severe respiratory distress. Severe cases are characterized by symptoms including dyspnea, persistent cough, pulmonary infiltrates, hypoxemia, and hypercoagulability, which may result in critically reduced oxygen saturation and increased mortality. Although the pathogenesis of severe COVID-19 remains incompletely understood, several factors – including advanced age, male sex, and underlying comorbidities – have been consistently associated with worse clinical outcomes and increased mortality [6].

Currently, many countries have implemented effective preventive and control strategies, resulting in a reduction in disease burden. However, due to ongoing outbreaks in certain regions, vulnerable populations – including patients with chronic illnesses – remain at risk of SARS-CoV-2 infection. Vaccination remains one of the most important preventive strategies, yet questions persist regarding host-related risk factors that may influence susceptibility to infection and disease severity. These include chronic diseases such as cardiovascular disorders, diabetes mellitus, platelet dysfunction, and potentially genetic factors such as ABO blood group phenotype [6].

In recent years, increasing attention has been directed toward the possible association between ABO blood group antigens and susceptibility to COVID-19 infection, as well as disease severity [7]. The ABO blood group system has previously been linked to a variety of diseases, including oncological conditions (e.g., ovarian, gastric, and prostate cancers) and infectious diseases. Associations have been reported with parasitic infections caused by *Plasmodium falciparum* and *Plasmodium vivax*, bacterial infections such as those caused by *Escherichia coli* and *Helicobacter pylori*, and viral infections including parvovirus B19, hepatitis B virus, Chikungunya virus, and West Nile virus [6].

Blood group antigens are organized into systems, collections, and low-incidence antigens (<1%). A defining feature of a blood group system is that it is encoded by a single genetic locus. The ABO blood group system is located on the long arm of chromosome 9 (9q34.2). ABO antigens are of particular biological importance because erythrocytes circulate throughout the body, and their antigens are also expressed on the surface of various tissues, including vascular endothelium, kidneys, heart, intestines, and pancreas. ABO antigens consist of carbohydrate structures attached to proteins or lipids and give rise to four genetically determined phenotypes: A, B, O, and AB. These phenotypes differ in both the expression and quantity of antigens on erythrocytes and in body secretions. In addition, the ABO system is characterized by the presence or absence of naturally occurring antibodies directed against A and/or B antigens (Figure 1) [8-10].

The figure illustrates a schematic representation of the four ABO blood group phenotypes, showing the erythrocyte membrane and the expression of ABO antigens: antigen A (blue star) and antigen B (green circle). The upper part of the image depicts the presence of naturally occurring ABO antibodies in the plasma corresponding to each blood phenotype [11].

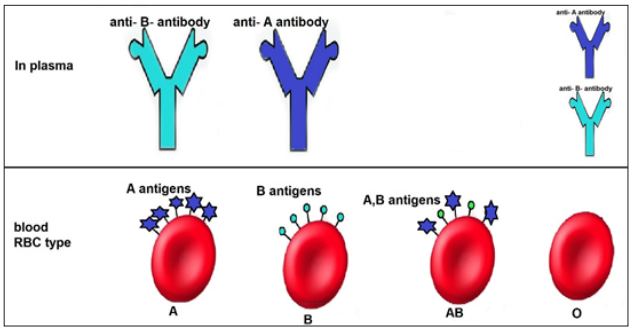

Figure 1. Diagram of the four ABO blood phenotypes, illustrating antigen expression on the erythrocyte membrane and corresponding plasma antibodies

Coronavirus vaccines

Pharmaceutical companies have competed to develop effective vaccines against coronavirus disease 2019 (COVID-19). Several vaccines, including those developed by Pfizer-BioNTech, AstraZeneca, and Sputnik V, have demonstrated protective efficacy against SARS-CoV-2 infection. However, the continuous emergence of viral mutations has led to the appearance of multiple SARS-CoV-2 variants, some of which exhibit increased transmissibility and altered pathogenicity. To date, several major variants have been identified, and additional variants may continue to emerge.

These viral mutations have contributed to sudden outbreaks in different regions of the world, including the resurgence observed in China in March 2022. COVID-19 is not expected to be the last global epidemic, as scientists anticipate the emergence of new viral strains or mutated forms even if SARS-CoV-2 transmission is eventually controlled. SARS-CoV-2 is characterized by a high mutation rate, particularly in viral structural proteins, which enhances its ability to adapt rapidly. Therefore, it is essential to study viral variants and their associated clinical manifestations to better understand disease progression and improve preventive and therapeutic strategies [12,13].

MATERIALS AND METHODS

A case-control study was conducted including 731 patients who met the operational definition of a confirmed COVID-19 case. All patients had SARS-CoV-2 infection confirmed by polymerase chain reaction (PCR) testing and received medical care at Alkarama Hospital between June 23 and July 23, 2021. To evaluate the association between COVID-19 infection and blood phenotypes, a control group of clinically healthy individuals was included. Controls consisted of voluntary blood donors who underwent standard blood phenotyping procedures at the hospital blood bank [14].

For the analysis of pneumonia and other clinical manifestations of COVID-19 in relation to blood phenotypes, participants were stratified according to disease severity into two groups: mild cases and severe cases, the latter defined clinically by the presence of acute respiratory distress syndrome (ARDS), in accordance with previous studies [13,14].

ABO blood group phenotypes and Rh status were determined using an automated Wadiana (Grifols) system based

on column agglutination methodology with gel cards (DG Gel) [15,16]. Demographic variables, including age and sex, as well as clinical severity indicators such as the need for intubation, admission to the intensive care unit (ICU), and mortality, were recorded and analyzed.

Data were expressed as percentages of COVID-19 patients. Comparisons among COVID-19 patients were performed to assess differences in ABO phenotypic frequencies in relation to disease severity, intubation, ICU admission, and mortality [17]. Cross-tabulation analyses were conducted using the chi-square test. Risk was assessed using odds ratios (ORs) with corresponding 95% confidence intervals (95% CIs). A p value of <0.05 was considered statistically significant.

Statistical analysis was performed using SPSS software version 24.0 (IBM Corp., USA), licensed by the University of Baghdad. This study was classified as risk-free research in accordance with the regulations of the General Law on Health Research.

RESULTS

The demographic characteristics of COVID-19 patients and control subjects are summarized in Table 1.

Table 1. COVID-19 data and ABO blood groups in patients with coronavirus disease 2019

	All n = 1250 (%)	COVID-19 n = 731 (%)	Control n = 519 (%)	χ^2	p
Age					
Mean (DS) (min-max)	35.70 ± 14.60 (18-70)	43.750 ± 13.27 (23-71)	24.460 ± 7.040 (18-61)	–7,97*	<0.0010
Sex					
Male	1030 (82.4%)	600 (82.2%)	430 (82.7%)	0.005	0.9420
Female	220 (17.6%)	130 (17.8%)	90 (17.3%)		
Type of blood					
A	230 (18.4%)	180 (24.7%)	50 (9.6%)	5,525	0.0630
B	70 (5.6%)	50 (6.8%)	20 (3.8%)		
O	950 (76.0%)	500 (68.5%)	450 (86.5%)		
Study groups					
A	230 (18.4%)	180 (24.7%)	50 (9.6%)	4,576	0.0320
not-A	1020 (81.6%)	550 (75.3%)	470 (90.4%)		
B	70 (5.6%)	50 (6.8%)	20 (3.8%)	0.518	0.4720
not-B	1180 (94.3%)	680 (93.2%)	500 (96.2%)		
O	950 (76.0%)	500 (68.51%)	450 (86.51%)	5,4220	0.020
not-O	300 (24%)	230 (31.5%)	70 (13.5%)		
RH					
Positive	1230 (98.4%)	710 (97.3%)	520 (100%)	2.19	0.139
Negative	2 (1.6)	2 (2.7%)	0 (0%)		

DS - standard deviation. * - Mann-Whitney U test

When comparing age and sex distributions between COVID-19 patients and the control group, no significant difference was observed with respect to sex, although a slightly higher proportion of males was noted among infected patients. In contrast, a significant difference was observed for age: patients with confirmed COVID-19 were older (43.75 ± 13.27 years) than control subjects (24.46 ± 7.04 years) (Figure 2).

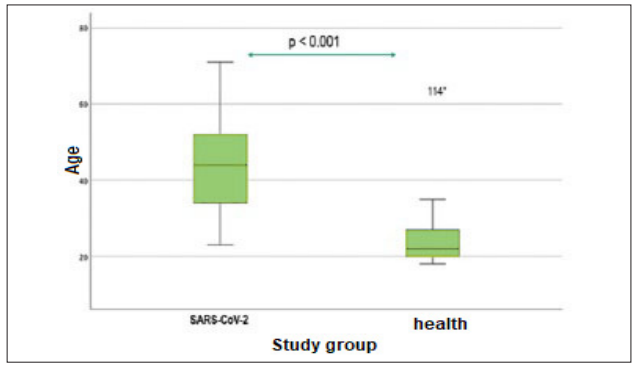

Figure 2. Distribution of age among healthy individuals and infected patients

This box-and-whisker plot illustrates age differences between the two study groups. Among patients infected with SARS-CoV-2, ages ranged from 23 to 71 years, whereas in the control group ages ranged from 18 to 62 years (as indicated by the whiskers). The median age was 54 years in COVID-19 patients and 23 years in controls (horizontal line within the box). The Mann-Whitney U test was applied. An outlier value of 114 years was identified in the control group and is indicated accordingly [18,19].

In this study, a higher incidence of blood phenotype A was observed among COVID-19 patients compared with other blood groups. In contrast, blood group O was associated with a lower incidence of SARS-CoV-2 infection and reduced disease severity.

To assess whether susceptibility to SARS-CoV-2 infection was associated with ABO blood group antigens, phenotype frequencies were compared between individuals carrying a given antigen and those without it. Accordingly, three comparisons were performed: A versus non-A, B versus non-B, and O versus non-O (Table 2).

Table 2. Risk analysis of ABO blood group phenotypes in COVID-19 patients versus controls

Comparison groups	COVID-19 OR	(95% CI)	χ^2	p
An against. not-A	1,452	(1,060-1,920)	4,577	0.0320
B against not-B	1,238	(0.756-2.030)	0.517	0.4720
O against not-O	0.687	(0.523-0.902)	5,423	0.020

OR - odds ratio CI - confidence interval

When comparing COVID-19 patients according to disease severity, a statistically significant difference was observed only for age. Patients with severe disease were older than those with mild disease (51.38 ± 11.67 vs. 34.52 ± 8.35 years; p < 0.001) (Figure 3). No statistically significant differences were found with respect to sex, ABO blood phenotype, or Rh factor (Table 3).

This box-and-whisker plot illustrates the age distribution in the two study groups. Among patients with mild SARS-CoV-2 disease, ages ranged from 23 to 48 years, whereas in patients with severe disease the age range was 26 to 71 years (whiskers). The median age was 36 years in the mild group and 51 years in the severe group.

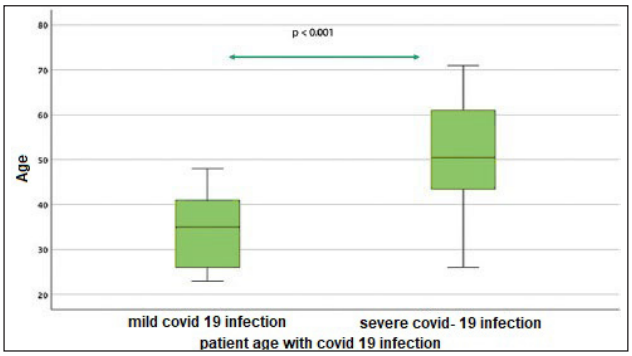

Figure 3. Age distribution among patients with mild and severe COVID-19

Table 3. Demographic appearances and ABO blood group in affected role with coronavirus disease 2019

	All n = 731 (100%)	Mild COVID-19 n = 330 (100.0%)	Severe COVID-19 n = 401 (100.0%)	χ^2	p
Age					
Mean SD (min.-max. value)	43.751 ±13.270 (23-71)	34.522 ±8.351 (23-48)	51.381 ±11.671 (26-71)	5,398*	<0.001
Sex					
Male	600 (82.20%)	290 (87.9%)	310 (77.52%)	1,331	0.249
female	131 (17.8%)	40 (12.1%)	90 (22.5%)		
Blood phenotype					
A	180 (24.7%)	90 (27.3%)	110 (27.5%)	0.502	0.778
B	50 (6.8%)	20 (6.1%)	30 (7.5%)		
O	500 (68.5%)	240 (72.7%)	260 (65%)		
Rh					
Positive	710 (97.31%)	320 (97.0%)	390 (97.5%)	1,0191	0.890
Negative	20 (2.70%)	10 (3.0%)	10 (2.50%)		

SD= standard deviation. *Mann-Whitney U test

The frequencies of blood phenotypes were analyzed in relation to the need for intubation, ICU admission, and mortality. A significant difference was observed in the distribution of blood phenotypes among deceased patients, with a higher proportion of blood group A compared with the other blood groups (46.7% *vs*. 15.2%; p = 0.017). No significant associations were found between blood phenotype and the need for intubation or ICU admission (Table 4).

Table 4. Frequencies of blood phenotypes according to disease severity characteristics (outcome, intubation, and admission to the adult intensive care unit [ICU])

	Blood phenotype					
	Whole	A n = 18	B n = 5	O n = 44	χ^2	p
Outcome (n =731)						
Alive	520 (78.80%)	80 (53.3%)	**50 (100%)**	390 (84.8%)	8,150	0.017
Passed away	140 (21.2%)	70 (46.7%)	0 (0%)	70 (15.2%)		
Intubated (n = 73)						
Yes	420 (57.5%)	80 (44.4%)	20 (40%)	320 (64%)		
Not	310 (42.5%)	100 (55.6%)	30 (60%)	180 (36%)	2,747	0.253
Admission to UTIA admission (n = 73)						
Yes	90 (45%)	90 (50%)	30 (60%)	160 (32%)	2,877	0.237
Not	110 (55%)	90 (50%)	20 (40%)	340 (68%)		

To assess the association between clinical outcome (alive vs. deceased) and ABO blood group antigens, differences were analyzed between individuals carrying a given antigen and those without it (A vs. non-A, B vs. non-B, and O *vs*. non-O). The results of these comparisons are presented in Table 5 [20].

Table 5. Mortality risk associated with ABO blood group phenotypes in COVID-19 patients

Comparison groups	COVID-19 OR	95% CI	χ2	p
An against. not-A	3.34	1,417-8,159	7,526	0.006
B against not-B	-------	-------	-------	-------
O against not-O	0.435	0.176-1.077	3,263	0.0710

COVID-19 - SARs Cov2; OR - odds ratio; CI - confidence interval

DISCUSSION

Differences in the expression of blood group antigens may increase susceptibility to many pathogens. Some blood groups are related to receptors for different organisms, including viruses, and certain blood types influence innate and adaptive immunity in host defense.

The antigens of the ABO system are a group of glycolipids and cellular glycopeptides that are secreted and distributed throughout the body. The ABO blood system has been associated with susceptibility to other coronaviruses. During the epidemic caused by SARS-CoV-1 (2002), an association was demonstrated between disease transmission and the ABO system in Hong Kong. In addition, individuals with blood type O were found to be more resistant to infection (OR = 0.180; 95% CI: 0.040-0.810; p = 0.003) [20,21].

Lehrer *et al.* performed a genome-wide association analysis to identify possible hereditary factors involved in the progression of COVID-19 and demonstrated an association with a group of genes located on chromosomes 3 and 9 (3p21.3 and 9q34.2), which coincides with the ABO blood group locus [19]. In their study, they concluded that patients with blood phenotype A had a higher risk of acquiring SARS-CoV-2 infection compared with other blood types (odds ratio = 1.450; 95% CI: 1.21-1.74; $p = 1.47 \times 10^{-4}$), whereas blood type O showed a protective effect against viral infection compared with other blood groups (odds ratio = 0.650; 95% CI: 0.530-0.790; $p = 1.06 \times 10^{-5}$). Their genetic findings support the observation that blood type A is associated with an increased risk of disease [22-24].

The present study shows that the risk of developing SARS-CoV-2 infection increases by 45.1% (odds ratio = 1.451; 95% confidence interval: 1.061-1.921; p = 0.032) in individuals with blood group A compared with those with other blood groups. These results are consistent with those reported by Badedi *et al.* [25], who found a 54% increase in risk (odds ratio = 1.54; 95% CI: 1.222-2.104; p = 0.006) in patients with blood group A compared with non-A individuals. Other studies, such as that by Liu *et al.* [22], also reported an increased risk for this blood group, with a twofold increase in odds ratio (odds ratio = 2.10; 95% CI: 1.50-2.90; p < 0.001) [26].

When comparing patients by disease severity and the need for intubation, no significant differences were found between blood phenotype and disease severity (mild versus severe),

intubation, or admission to the ICU. These findings are similar to those reported by Liu *et al.* [22] and Almadhi *et al.* [23], who did not observe a clear association between erythrocyte antigens and intubation or ICU admission.

Regarding mortality, it was observed that among patients with COVID-19, the risk of death was approximately threefold higher (odds ratio = 3.4; 95% CI: 1.417-8.159; p = 0.006) in individuals with blood group A. These results differ from those reported by Zietz and Tatonetti, who, in a cohort of 1,559 patients from three geographic regions (New York, Wuhan, and Shenzhen), did not identify statistically significant differences between deceased and surviving patients. It is important to note that in their study, blood phenotype information was obtained from electronic medical records, which may be subject to information bias [24,27].

This study confirms that the ABO system is involved in susceptibility to pneumonia, severe symptoms, and death in patients with COVID-19, although the underlying mechanisms remain unclear. Two possible biological mechanisms may explain this association. The first is related to the natural antibodies of the ABO system. In this context, the protective effect observed in individuals with blood phenotype O may be explained by the presence of anti-A antibodies. Conversely, individuals with blood phenotype A, who lack these antibodies, may be more vulnerable to infection. This mechanism has been described based on the similarity between the saccharide structures on the membranes of some microorganisms and the saccharides of antigens A and B. Therefore, it would not be surprising if a similar phenomenon occurs in SARS-CoV-2 infection.

In studies involving other coronaviruses, such as SARS-CoV-1, viral adhesion mediated by the spike (S) protein to target cells expressing the angiotensin-converting enzyme 2 (ACE2) receptor was specifically inhibited by anti-A monoclonal antibodies. This finding demonstrates that such antibodies can prevent viral interaction with the receptor and thus provide protection [28]. If this hypothesis is correct, it may also explain the increased susceptibility observed in older patients (Figures 2 and 3), since titers of anti-A and anti-B antibodies decline with age. Nevertheless, the specific role of antigen A and its variants in SARS-CoV-2 infection remains unknown, and the observed association may also be influenced by interactions with other blood group systems; therefore, additional factors should be investigated [25,26,28].

Do mutations have any significance?

Many of the known SARS-CoV-2 mutations affect viral structure, particularly the spike protein. These mutations can lead to significant changes in viral behavior, including transmission efficiency, host cell entry, viral genome transcription, and potential resistance to vaccines. Such changes may also result from alterations in viral surface proteins and their interaction with the ACE2 receptor. Recently, new mutations have been reported that affect interactions between red blood cells and the SARS-CoV-2 spike protein, potentially influencing viral entry and disease severity. Moreover, several in silico studies have investigated potent drug-receptor interactions regardless of mutations in the target protein, highlighting the importance of drug design strategies focused on ACE2 modulation and inhibition of spike protein binding [28-31].

CONCLUSION

Blood phenotype A is associated with an increased risk of COVID-19. In this investigation, which included patients with mild symptoms compared with those with severe COVID-19, a weak association was observed between ABO erythrocyte antigens and severe disease manifestations, as well as the probability of intubation or admission to the ICU; however, a significant association was observed with COVID-19-related mortality.

It is suggested that individuals with blood group A strengthen protective measures to reduce the risk of SARS-CoV-2 infection. In hospital settings, identifying patients with this blood group may help ensure closer medical surveillance.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

FUNDING

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ETHICAL APPROVAL AND LEGAL RESPONSIBILITIES

This study received ethical approval from the Iraqi Medical Research Center (Approval No. 11990/2019) and was conducted in accordance with national regulations governing health research.

DATA AVAILABILITY

All data generated or analyzed during this study are included in the manuscript.

PRIVACY AND INFORMED CONSENT

The authors confirm that no identifiable patient data are included in this article; therefore, informed consent was not required.

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