THE MEDICAL BULLETIN OF
SISLI ETFAL HOSPITAL

DOI: 10.14744/SEMB.2021.15045
Med Bull Sisli Etfal Hosp 2022;56(1):41–48

Original Research

Relationship Between ABO Blood Types and Coronavirus Disease 2019 Severity

Mufide Arzu Ozkarafakili,[1] Nesrin Gareayaghi,[2] Zeynep Mine Yalcinkaya Kara[3]

[1]Department of Chest Diseases, Sisli Hamidiye Etfal Training and Research Hospital, University of Health Sciences Turkey, Istanbul, Turkey
[2]Department of Microbiology and Clinical Microbiology, Sisli Hamidiye Etfal Training and Research Hospital, University of Health Sciences Turkey, Istanbul, Turkey
[3]Department of Biochemistry, Sisli Hamidiye Etfal Training and Research Hospital, University of Health Sciences Turkey, Istanbul, Turkey

Abstract

Objectives: Severe Acute Respiratory Syndrome Coronavirus-2 infection spreads rapidly around the world. The blood groups are recognized to influence susceptibility to certain viruses.

The aim of this research was to determine any potential role of the patients' ABO and Rh blood groups in both the acquisition and severity of coronavirus disease 2019 (COVID-19). As a growing global health problem, to find any marker for COVID-19 may help to identify high-risk individuals and ease the strain on health system.

Methods: The patients who were hospitalized between March and August 2020 with a diagnosis of COVID-19 and had a documented ABO blood type in medical database were examined retrospectively. Patients were grouped as survivors (followed up in pandemic wards /or intensive care unit [ICU]) and non-survivors. Their ABO blood types were correlated with general population's blood types. The labaratory findings of patients were evaluated according to the blood types.

Results: A total of 492 patients included, 233 (47.4%) were male. The mean age was 58.9±17.5. Data of ABO blood groups of 51966 individuals in general population was used as a control group; the number of the patients in Rh (-) blood type 0, were significantly lower than the control group (p=0.008). Among the whole patient group (survivors and non-survivors), Blood type A 210 (42%) was the most common and type AB 52 (10%) was the least common. However, no statistically significant difference was noted between survivors (pandemic wards/ICU) and non-survivors unlike the previous studies (p=0.514). No correlation was found between laboratory findings (Hemoglobin, red cell distribution width, platelet, white blood cell, lymphocyte, D-Dimer, C-reactive protein, ferritin) and ABO blood groups of COVID-19 patients (p>0.05).

Conclusion: There was no association found between the ABO blood type and COVID-19 infection rate or disease severity. No evidence was noted to support the use of ABO blood type as a marker for COVID-19. Further efforts are warranted to better predict outcomes of hospitalized COVID-19 patients.

Keywords: ABO blood types, COVID-19, severity

Please cite this article as "Ozkarafakili MA, Gareayaghi N, Yalcinkaya Kara ZM. Relationship Between ABO Blood Types and Coronavirus Disease 2019 Severity. Med Bull Sisli Etfal Hosp 2022;56(1):41–48".

Address for correspondence: Mufide Arzu Ozkarafakili, MD. Saglik Bilimleri Universitesi, Sisli Hamidiye Etfal Egitim ve Arastirma Hastanesi Gogus Hastaliklari Anabilim Dali, Istanbul, Turkey
Phone: +90 533 223 11 00 **E-mail:** aaarzup@yahoo.com

Submitted Date: June 04, 2021 **Accepted Date:** September 13, 2021 **Available Online Date:** March 28, 2022

Since December 2019, the world has been dealing with Coronavirus disease 2019 (COVID-19), which was declared as a global health emergency by the World Health Organization with 169 million patients and 3.5 million deaths, caused by Severe Acute Respiratory Syndrome Coronavirus2 (SARS-CoV-2).[1] While massive vaccination campaign is going on, the whole world is trying to cope with the waves of epidemic and the burden it puts on the health system.[2] COVID-19 can be seen in different forms; from asymptomatic patients to those with mild symptoms such as dry cough, fever, and weakness, to severe cases that result in acute respiratory distress syndrome and death. SARS-CoV-2 is transmitted by respiratory droplets.[3] The fact that asymptomatic cases can transmit the virus and the excess of critical patients who overburden the intensive care units (ICUs) bring along the search for a biomarker that can predict the risk of being infected with SARS-CoV-2. Individuals have A, B, AB, and O blood types, depending on whether antigen is present on the erythrocyte surface or not. These antigens are also found in the epithelium, endothelium, platelets, and exocrine glands that secrete mucin and may have a role on the onset of various diseases. Blood groups are also called positive or negative according to the presence of Rhesus (Rh) factor protein.[4] The Rh system is clinically the most important and complex protein-based system because Rh proteins are expressed only in the membranes of the red blood cells. Despite important geographical differences, the most common blood type in the world is the O blood group. In the (H1N1) epidemic caused by the influenza virus; it has been noted that people with A and B blood groups are more prone to infection than people with O and AB blood groups.[5]

In 2003, the relationship between the SARS infection seen in Asia and blood groups was investigated; it has been found that those with blood type 0 are less affected by SARS-CoV than those with non- O blood group.[6] There are numerous studies with contradictory results about the relationship between ABO blood groups and COVID-19 infection.[7,8] We investigated the relationship with ABO blood group and the clinical characteristics, laboratory findings of 492 COVID-19 patients who were hospitalized and had a documented blood group in the medical database.

Methods

In this study, the electronic medical records of 492 patients who were diagnosed with COVID-19 and had a blood group record in the medical database and followed up in the hospital between March and August 2020 were retrospectively analyzed. Patients over 18 who were hospitalized and confirmed with real-time polymerase chain reaction test positive for SARS-CoV-2 in nasopharyngeal swab or who had epidemic history, symptoms, and Chest Computerized Tomography findings compatible with COVID-19 and couldn't be explained by other factors were included in the study. Those whose blood groups were not recorded in the medical database were excluded from the study. As the control group, the ABO blood group data of 51966 people of general population, obtained from the Blood Bank for 3 years of screening in the same region were used. The patients were categorized as survivors (followed up in the pandemic wards/ICU) and non-survivors. Data include demographics, comorbidities, laboratory findings. The non-survivors' data were collected 48 h before death. The survivors' data were collected at time of admission to pandemic wards or at the time to transfer to the ICU. Our study was approved by the Ethics Committee of our hospital (No: 2738) and was conducted in accordance with the Helsinki Declaration.

Statistical Analysis

Descriptive values of the obtained data were calculated as mean, standard deviation (SD), number and % frequencies depending on the variable type. The distribution of blood groups in the general population and the distribution of blood groups in COVID-19 patients in the study group were compared with the t-test for two independent proportions. Categorical characteristics of surviving patients (pandemic wards/ICU) and those who died were compared with the Fisher-Freeman-Halton exact test. In addition, the Kruskal-Wallis and Mann Whitney U test was used to compare these groups in terms of numerical type characteristics. Groups that differed significantly were determined using the post-hoc Dunn test. Relationships between numerical characteristics and blood groups, and the patient's current status (pandemic wards, ICU/death) were evaluated with analysis of variance and analysis of covariance (ANCOVA) models. In the ANCOVA model, these effects were eliminated by considering the effects of age and gender. Factors associated with patients' clinical outcome were evaluated using the multivariate logistic regression model to determine the independent effect. Statistical significance level was accepted as $p<0.05$ and SPSS ver. 23 (IBM, Turkey) program was used for calculation.

Results

We enrolled 492 COVID-19 patients. 233 (47.4%) were male. The mean age was 58.9±17.5 (between 18 and 95 years); 58.71±15.81 for male, 59.15±19.43 for female. Data retrieved from Blood Bank, 51,966 individuals in the same region were used as a control group (Table 1). The patients in the blood group 0 Rh (-) was significantly lower than

Table 1. Distribution of blood types in control and patient group

	Control Group		Patient Group		P*
	n	%	n	%	
ABO Blood Type					
0+	15600	30.020	142	28.862	0.573
0-	2556	4.919	12	2.439	0.008
A+	19843	38.185	187	38.008	0.936
A-	2690	5.176	23	4.675	0.601
B+	6630	12.758	71	14.431	0.293
B-	830	1.597	5	1.016	0.202
AB+	3335	6.418	44	8.943	0.126
AB-	482	0.928	8	1.626	0.222
Total	51966		492		

*t-test for two independent proportions.

the ones in the general population (p=0.008). The blood group distribution of COVID-19 patients were; type A 210 (42%), blood type 0 154 (31%), type B 76 (15%), type AB 52 (10%) respectively. Rh positivity was 444 (90%). Blood type A was found the most common and type AB was the least common. In terms of ABO blood type distribution, although there were percentile differences, this was not statistically significant when compared with the general population.

Patients were divided into two groups: Survivors (those followed in the pandemic wards or in the ICU) 458 93% and non-survivors 34 6.9%. The demographic characteristics, ABO blood groups, and comorbidities were examined in Table 2; the female patients followed up in the pandemic wards was found significantly higher (250.50%) and the male gender ratio was found to be significantly higher in patients who were transferred to the ICU (10.2%) and who died (26.5%) (p<0.01). Hypertension was the most common comorbidity 179.36%. No statistically significant difference was observed in terms of ABO blood types/Rh (Rh factor) between survivors and non-survivors.

The laboratory findings of the patients; (Red cell distribution width [RDW], D-Dimer, hemoglobin, red blood cell [RBC], lymphocyte, platelet, white blood cell [WBC]), ferritin, C-reactive protein [CRP]) were examined according to AB0 blood groups and no statistically significant difference was found between the groups (p>0.05) (Table 3).

In Table 4, according to the clinical outcomes of the patients, the descriptive statistics of age and laboratory findings are shown. The mean age of the survivors and the deceased patients was found to be similar. RDW, D-Dimer, and hemoglobin were found to be significantly lower in the patients followed in the pandemic wards than in the other two groups (p<0.01). The average of RBC, lymphocyte and platelet were found significantly lower (p=0.014, p=0,018, p=0.02) and WBC, ferritin, CRP were found to be significantly higher in non-survivors than the survivors (p<0.01).

In Tables 5 and 6, the demographic characteristics and the clinical outcomes of the patients are analyzed with multivariate logistic regression model. In the multivariable analysis, blood type was not determined to be independently associated with COVID-19 disease severity. No significant difference was found in terms of blood group distribution between the patients followed up in the pandemic wards and the ICU (95% CI. for OR, P=0.946) (Table 5).

Similarly, no significant difference was found in terms of blood groups distribution of patients who were hospitalized in the pandemic wards and who died with the multivariable analysis (95% CI. for OR, p=0.685) (Table 6).

Discussion

Several studies have been conducted to date on relationship between blood groups and COVID-19. Literature revealed that ABO blood group was associated not only with COVID-19 susceptibility but also with severe outcomes and death. In our retrospective analysis, no correlation between the blood groups and the acquisition of COVID-19 was found, also there was no association noted between ABO blood type and COVID-19 disease severity. Blood type AB had the lowest and blood type A had the highest frequency of the disease in our study. But no statistical significance was found. The prevalence between genders was equal, but the mortality rate was higher in men than women in our data. The study results

Table 2. Clinical and demographic characteristics, comorbidities, and ABO blood types of patients

	Survivors				Non-survivors		P*
	Pandemic wards		Intensive care unit				
	n	%	n	%	n	%	
Gender							
Male	197	84.5[a]	10	4.3[a]	26	11.2[a]	<0.001
Female	250	96.5[b]	1	0.4[b]	8	3.1[b]	
AB0 Rh							
0 Rh (+)	133	93.7	2	1.4	7	4.9	0.514
0 Rh (-)	11	91.7	1	8.3	0	0	
A Rh (+)	170	90.9	4	2.1	13	7	
A Rh (-)	19	82.6	1	4.3	3	13	
B Rh (+)	61	85.9	3	4.2	7	9.9	
B Rh (-)	5	100	0	0	0	0	
AB Rh (+)	41	93.2	0	0	3	6.8	
AB Rh (-)	7	87.5	0	0	1	12.5	
AB0							
Group 0	144	93.5	3	1.9	7	4.5	0.596
Group A	189	90	5	2.4	16	7.6	
Group B	66	86.8	3	3.9	7	9.2	
Group AB	48	92.3	0	0	4	7.7	
Hypertension							
No	285	91.1	7	2.2	21	6.7	0.966
Yes	162	90.5	4	2.2	13	7.3	
Diabetes							
No	339	91.4	10	2.7	22	5.9	0.200
Yes	108	89.3	1	0.8	12	9.9	
COPD/Asthma							
No	398	91.3	9	2.1	29	6.7	0.459
Yes	49	87.5	2	3.6	5	8.9	
Congestive heart failure							
No	426	90.8	11	2.3	32	6.8	0.802
Yes	21	91.3	0	0	2	8.7	
Chronic renal disease							
No	406	90.8	11	2.5	30	6.7	0.596
Yes	41	91.1	0	0	4	8.9	
Coronary artery disease							
No	392	90.7	10	2.3	30	6.9	0.947
Yes	55	91.7	1	1.7	4	6.7	
Malignity							
No	430	90.9	11	2.3	32	6.8	0.762
Yes	17	89.5	0	0	2	10.5	

*Fisher-Freeman-Halton exact test, n: Data set, COPD: Chronic obstructive pulmonary disease.

of Zhao et al. corresponded a significantly increased risk of blood type A and decreased risk of blood type 0 for COVID-19 and similar distribution pattern for mortality also.[7] Ellinghaus et al. study on genetics data suggested blood type A is associated with higher risk of acquiring COVID-19 than non-A blood groups.[9] Latz et al. and Zietz et al. showed Rh (-) patients had lower risk of infection by SARS-CoV-2.[10,11] After multivariable analysis, blood type was not independently associated with risk of surviving or death (95% CI. for OR). No association between ABO subtype (Rh factor) and severe disease was found in our study. Our data are different from the experiences of Zhao et al., Ellinghaus et al., Latz et al., and Zietz and Tatonetti. Despite these prior studies, Jeffrey et al. had shown no association between ABO blood type and COVID-19 predisposition or severity.[8] Some hypothesis raised for the vari-

Table 3. Distribution of laboratory findings according to blood types

	AGE	RDW	RBC	LYM	WBC	FERRITIN	CRP	DDIMER	HGB	PLT
Blood Type										
0 RH (-)										
Mean	50	13.87	4.15	1.34	9.23	237.70	95.08	1083.08	124.92	231.25
SD	18	1.61	0.59	0.64	5.32	253.39	104.36	1115.05	22.84	111.01
0 RH (+)										
Mean	59	14.54	4.32	1.47	7.97	223.35	59.94	1296.85	122.87	218.99
SD	18	2.26	0.83	1.07	4.01	222.97	78.59	1598.24	27.72	92.02
A RH (-)										
Mean	61	14.58	4.41	7.11	15.23	233.37	75.53	1061.74	126.17	207.09
SD	16	2.11	0.70	28.47	30.11	233.12	98.79	1050.06	24.77	92.06
A RH (+)										
Mean	59	14.39	4.25	1.48	7.60	228.95	60.56	1983.81	121.18	215.15
SD	17	2.13	0.88	0.97	4.93	242.75	73.08	7940.34	25.50	89.86
AB RH (-)										
Mean	58	15.39	4.43	2.66	14.39	256.90	10.88	850.00	118.75	240.63
SD	19	2.16	1.33	1.37	15.61	200.02	12.38	1198.77	29.42	77.04
AB RH (+)										
Mean	61	14.23	4.57	1.55	8.47	190.84	60.54	1046.64	127.73	220.59
SD	16	1.46	1.60	0.87	3.90	197.03	71.46	855.28	20.64	98.94
B RH (-)										
Mean	49	13.58	4.78	2.23	13.94	220.38	56.66	366.00	141.60	235.60
SD	9	0.75	0.34	0.96	10.61	203.37	78.06	205.64	12.34	16.21
B RH (+)										
Mean	59	14.32	4.47	2.77	11.93	182.94	64.95	2332.59	124.11	237.87
SD	18	1.88	0.80	11.49	23.63	172.50	77.30	9642.40	24.52	88.59
P-value*	0.742	0.742	0.191	0.070	0.019	0.882	0.335	0.896	0.330	0.727

*Corrected differences between blood groups by eliminating the impact of gender and age differences, RDW: Red cell distribution width, RBC: Red blood cell, LYM: Lymphocyte, WBC: White blood cell, CRP: C-reactive protein, HGB: Hemoglobin, PLT: Platelet.

ability of contagiousness of SARS-CoV-2, infection rates and the severity of the disease. Angiotensin-converting enzyme 2 (ACE2) is the primary receptor and major way for SARS-CoV-2 entering into host cells. During the SARS outbreak in 2003, the scientists reported higher risk for SARS-CoV-1 infection for blood type A and explained it by, spike protein/ACE2 dependent adhesion to ACE2 cell lines was inhibited by monoclonal or natural human anti-A antibodies. In the individuals with blood type 0 and B, these anti-A antibodies may protect from SARS-CoV-2 infection by blocking the interaction between coronavirus and ACE2.[12] Cheng et al. had shown that blood type 0 was found to be less common in SARS-CoV-1 infection.[6] As the geographical dependence of the blood type distribution could affect the regional infection rates, Mattio et al.'s present paper help us understand the importance of some other population-dependent antigens' role in COVID-19.[13] Some reports reveal that the ABO blood group antigen alters the inflammatory response. Non-0 blood types have been previously shown to effect hemostasis by increasing Von Willebrand Factor and Factor VIII, that can lead to thrombotic events, which is the cornerstone for COVID-19.[14] Inflammation is a condition known as related to COVID-19 disease state and severe outcomes.[15] The cytokine storm leads to T cell dysfunction and peripheral lymphopenia in most hospitalized patients and building up to severe forms of the disease.[16] No difference was demonstrated for inflammatory markers (WBC, CRP) according to ABO blood type in our study. Furthermore, WBC, CRP, ferritin were found to be significantly higher in non-survivors than the survivors ($p<0.01$), and RBC, lymphocyte and platelet were found significantly lower in non-survivors than the survivors in our study ($p=0.014$, $p=0,018$, $p=0.02$).

We had some limitations; the sample size was small, and the number of blood type O Rh (-) patients were significantly lower than the general population which might lead to bias in the results. Only the patients who had the

Table 4. Descriptive features of numerical characteristics of patients according to clinical outcome

	n	Mean	SD	Percentiles			P*
				25	Median	75	
Age							
Pandemic wards	447	58.38	17.79	47	59	72	0.136
Intensive care unit	11	62.55	13.52	48	61	74	
Non-survivors	34	64.59	12.64	54	64	76.25	
Red cell distribution width							
Pandemic wards	447	14.31	2.05	13	13.80[a]	15	<0.001
Intensive care unit	11	15.47	2.09	13.5	15.60[b]	16.3	
Non-survivors	34	15.37	1.9	13.85	15.25[b]	16.23	
Red blood cell							
Pandemic wards	447	4.38	0.91	3.93	4.64[a]	4.9	0.014
Intensive care unit	11	4.16	0.9	3.13	4.57[a]	4.91	
Non-survivors	34	3.92	1.1	3.25	4.01[b]	4.52	
Lymphocyte							
Pandemic wards	445	1.5	0.94	0.93	1.29[a]	1.85	0.018
Intensive care unit	11	1.23	0.52	0.79	1.01a[b]	1.67	
Non-survivors	34	1.57	1.92	0.54	.90[b]	1.58	
White blood cell							
Pandemic wards	447	7.98	7.73	5.15	6.65[a]	9.63	<0.001
Intensive care unit	11	11.08	7.53	5.59	9.43[ab]	13.67	
Non-survivors	34	21.43	34.1	7.85	11.40[b]	18.9	
Hemoglobin							
Pandemic wards	447	125.15	24.7	110	129.00[a]	142	<0.001
Intensive care unit	11	104.91	25.49	81	108.00[b]	130	
Non-survivors	34	103.12	25.67	87.75	101.00[b]	119.5	
Platelet							
Pandemic wards	447	222.53	84.06	166	207.00[a]	263	0.002
Intensive care unit	11	285.55	147.54	187	271.00[a]	421	
Non-survivors	34	175.04	132.54	79	168.00[b]	242.75	
D-Dimer							
Pandemic wards	444	1095.13	1491.66	379	622.00[a]	1257.5	<0.001
Intensive care unit	11	1640.45	1006.19	776	1330.00[b]	2400	
Non-survivors	34	8940.52	21867.25	906	1915.00[b]	4475	
Ferritin							
Pandemic wards	445	299.02	571.06	55.8	136.60[a]	333.95	<0.001
Intensive care unit	11	329.45	279.39	111.4	176.00[a]	577.5	
Non-survivors	34	1951.11	3318.51	296.77	594.35[b]	1714.5	
C-reactive protein							
Pandemic wards	445	51.7	63.62	8.5	26.60[a]	74	<0.001
Intensive care unit	11	112.86	103.07	15.1	87.50[b]	220.7	
Non-survivors	34	174.69	120.23	68.75	174.25[b]	272.98	

*Kruskal Wallis test and post-hoc Dunn test.

documented ABO blood type were included in the study, so these results might not truly reflect the correct number of COVID-19 cases.

Conclusion

Despite the early reports suggesting that blood type A might be more susceptible while blood type O might be less susceptible to infect COVID-19; we found no evidence for association between ABO blood groups and COVID-19, also there was no correlation noted for disease severity and mortality throughout our analysis. The conflicting data in the literature necessitates further higher-quality studies to identify the role of ABO blood groups in SARS-CoV-2 infection.

Table 5. Multivariate logistic regression model of patients in intensive care unit and the pandemic wards.

	P	OR	95% CI. for OR	
			Lower	Upper
Blood Types	0.946			
Type (A/0)	0.543	1.754	0.288	10.688
Type (AB/0)	0.997	0.000	0.000	--
Type (B/0)	0.749	1.416	0.168	11.959
Age	0.354	0.974	0.920	1.030
Gender (Female/Male)	0.012	0.031	0.002	0.460
RDW	0.578	0.889	0.587	1.347
RBC	0.019	5.338	1.321	21.569
LYM	0.282	0.499	0.141	1.770
WBC	0.840	1.020	0.838	1.243
Ferritin	0.536	0.999	0.995	1.003
CRP	0.039	1.013	1.001	1.026
D-Dimer	0.536	1.000	1.000	1.001
HGB	0.003	0.917	0.865	0.971
PLT	0.194	1.005	0.997	1.013
CVD (Yes/No)	0.998	0.000	0.000	--
COPD (Yes/No)	0.220	3.832	0.447	32.841
CHF (Yes/No)	0.997	0.000	0.000	--
CAD (Yes/No)	0.280	0.238	0.018	3.213
HT (Yes/No)	0.764	1.308	0.227	7.526
DM (Yes/No)	0.224	0.209	0.017	2.609
Constant	0.741	6.558		

OR: Odd ratio, CI: Confident interval, CVD: Cerebrovascular disease, COPD: Chronic obstructive pulmonary disease, CHF: Congestive heart failure, CAD: Coronary artery disease, HT: Hypertension, DM: Diabetes mellitus, RDW: Red cell distribution width, RBC: Red blood cell, LYM: Lymphocyte, WBC: White blood cell, CRP: C-reactive protein, HGB: Hemoglobin, PLT: Platelet.

Table 6. Multivariate logistic regression model of patients in the pandemic wards and the non-survivors

	P	OR	95% CI. for OR	
			Lower	Upper
Blood Types	0.685			
Type (A/0)	0.352	2.380	0.383	14.771
Type (AB/0)	0.288	3.701	0.331	41.372
Type (B/0)	0.320	3.112	0.331	29.232
Age	0.727	0.991	0.939	1.045
Gender (Female/Male)	0.162	0.319	0.064	1.585
RDW	0.229	1.205	0.889	1.634
RBC	0.763	0.898	0.447	1.805
LYM	0.234	0.566	0.222	1.444
WBC	0.003	1.175	1.057	1.305
Ferritin	0.460	1.001	0.998	1.004
CRP	0.015	1.009	1.002	1.016
D-Dimer	0.576	1.000	0.999	1.000
HGB	0.546	0.987	0.948	1.029
PLT	0.102	0.994	0.986	1.001
CVD (Yes/No)	0.999	0.000	0.000	--
COPD (Yes/No)	0.998	0.000	0.000	--
CHF (Yes/No)	0.328	3.467	0.288	41.800
CAD (Yes/No)	0.416	0.356	0.030	4.284
HT (Yes/No)	0.282	2.319	0.501	10.731
DM (Yes/No)	0.725	1.320	0.280	6.222
Constant	0.285	0.007		

OR: Odd ratio, CI: Confident interval, CVD: Cerebrovascular disease, COPD: Chronic obstructive pulmonary disease, CHF: Congestive heart failure, CAD: Coronary artery disease, HT: Hypertension, DM: Diabetes mellitus, RDW: Red cell distribution width, RBC: Red blood cell, LYM: Lymphocyte, WBC: White blood cell, CRP: C-reactive protein, HGB: Hemoglobin, PLT: Platelet.

Disclosures

Ethics Committee Approval: Our study was approved by the Ethics Committee of our hospital (No: 2738) and was conducted in accordance with the Helsinki Declaration.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Concept – M.A.O.; Design – M.A.O.; Supervision – M.A.O.; Data collection &/or processing – N.G.; Analysis and/or interpretation Z.M.Y.K.; Literature search – M.A.O.; Writing – M.A.O.; Critical review – M.A.O., Z.M.Y.K., N.G.

References

1. World Health Organization WHO Coronavirus Disease (COVID-19) Dashboard. Available at: https://covid19.who.int/. Accessed Jan 31, 2022.
2. Besirli A, Erden SC, Atilgan M, Varlihan A, Habaci MF, Yeniceri T, et al. The relationship between anxiety and depression levels with perceived stress and coping strategies in health care workers during the COVID-19 pandemic. Sisli Etfal Hastan Tip Bul 2021;55:1–11.
3. Rothan HA, Byrareddy SN. The epidemiology and pathogenesis of coronavirus disease (COVID-19) outbreak. J Autoimmun 2020;109:102433. [CrossRef]
4. Huang CH, Liu PZ, Cheng JG. Molecular biology and genetics of the Rh blood group system. Semin Hematol 2000;37:150–65.
5. Aho K, Pyhälä R, Visakorpi R. ABO associated genetic determinant in H1N1 influenza. Tissue Antigens 1980;16:310–3. [CrossRef]
6. Cheng Y, Cheng G, Chui CH, Lau FY, Chan PK, Ng MH, et al. ABO blood group and susceptibility to severe acute respiratory syndrome. JAMA 2005;293:1450–1. [CrossRef]
7. Zhao Q, Meng M, Kumar R, Wu Y, Huang J, Lian N, et al. The impact of COPD and smoking history on the severity of COVID-19: A systemic review and meta-analysis. J Med Virol 2020;92:1915–21.
8. Anderson JL, May HT, Knight S, Bair TL, Muhlestein JB, Knowlton KU, et al. Association of sociodemographic factors and blood group type with risk of COVID-19 in a US population. JAMA Netw Open 2021;4:e217429. [CrossRef]
9. Ellinghaus D, Degenhardt F, Bujanda L, Buti M, Albillos A, Invernizzi P et al. The ABO blood group locus and a chromosome

3 gene cluster associate with SARS-CoV-2 respiratory failure in an Italian-Spanish genome-wide association analysis. medRxiv 2020 Jun 02 2020, doi: 10.1101/2020.05.31.20114991. [CrossRef]
10. Latz CA, DeCarlo C, Boitano L, Png CYM, Patell R, Conrad MF, et al. Blood type and outcomes in patients with COVID-19. Ann Hematol 2020;99:2113–8. [CrossRef]
11. Zietz M, Tatonetti N. Testing the association between blood type and COVID-19 infection, intubation and death. medRxiv April 11 2020, doi: 10.1101/2020040820058073. [CrossRef]
12. Guillon P, Clément M, Sébille V, Rivain JG, Chou CF, Ruvoën-Clouet N, et al. Inhibition of the interaction between the SARS-CoV spike protein and its cellular receptor by anti-histo-blood group antibodies. Glycobiology 2008;18:1085–93. [CrossRef]
13. Miotto M, Di Rienzo L, Gosti G, Milanetti E, Ruocco G. Does blood type affect the COVID-19 infection pattern? PLoS One 2021;16:e0251535. [CrossRef]
14. Iba T, Levy JH, Raj A, Warkentin TE. Advance in the management of sepsis-induced coagulopathy and disseminated intravascular coagulation. J Clin Med 2019;8:728. [CrossRef]
15. Meftahi GH, Jangravi Z, Sahraei H, Bahari Z. The possible pathophysiology mechanism of cytokine storm in elderly adults with COVID-19 infection: the contribution of "inflame-aging". Inflamm Res 2020;69:825–39. [CrossRef]
16. Tay MZ, Poh CM, Rénia L, MacAry PA, Ng LFP. The trinity of COVID-19: immunity, inflammation and intervention. Nat Rev Immunol 2020;20:363–74. [CrossRef]