ORIGINAL ARTICLE

Blood type and outcomes in patients with COVID-19

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Abstract

This study aimed to determine if there is an association between ABO blood type and severity of COVID-19 defined by intubation or death as well as ascertain if there is variability in testing positive for COVID-19 between blood types. In a multi-institutional study, all adult patients who tested positive for COVID-19 across five hospitals were identified and included from March 6th to April 16th, 2020. Hospitalization, intubation, and death were evaluated for association with blood type. Univariate analysis was conducted using standard techniques and logistic regression was used to determine the independent effect of blood type on intubation and/or death and positive testing. During the study period, there were 7648 patients who received COVID-19 testing throughout the institutions. Of these, 1289 tested positive with a known blood type. A total of 484 (37.5%) were admitted to hospital, 123 (9.5%) were admitted to the ICU, 108 (8.4%) were intubated, 3 (0.2%) required ECMO, and 89 (6.9%) died. Of the 1289 patients who tested positive, 440 (34.2%) were blood type A, 201 (15.6%) were blood type B, 61 (4.7%) were blood type AB, and 587 (45.5%) were blood type O. On univariate analysis, there was no association between blood type and any of the peak inflammatory markers (peak WBC, $p = 0.25$; peak LDH, $p = 0.40$; peak ESR, $p = 0.16$; peak CRP, $p = 0.14$) nor between blood type and any of the clinical outcomes of severity (admission $p = 0.20$, ICU admission $p = 0.94$, intubation $p = 0.93$, proning while intubated $p = 0.58$, ECMO $p = 0.09$, and death $p = 0.49$). After multivariable analysis, blood type was not independently associated with risk of intubation or death (referent blood type A; blood type B: AOR: 0.72, 95% CI: 0.42–1.26, blood type AB: AOR: 0.78, CI: 0.33–1.87, blood type O: AOR: 0.77, CI: 0.51–1.16), rhesus factor positive (Rh+): AOR: 1.03, CI: 0.93–1.86. Blood type A had no correlation with positive testing (AOR: 1.00, CI: 0.88–1.13), blood type B was associated with higher odds of testing positive for disease (AOR: 1.28, CI: 1.08–1.52), AB was also associated with higher odds of testing positive (AOR: 1.37, CI: 1.02–1.83), and O was associated with a lower risk of testing positive (AOR: 0.84, CI: 0.75–0.95). Rh+ status was associated with higher odds of testing positive (AOR: 1.23, CI: 1.003–1.50). Blood type was not associated with risk of intubation or death in patients with COVID-19. Patients with blood types B and AB who received a test were more likely to test positive and blood type O was less likely to test positive. Rh+ patients were more likely to test positive.

Keywords COVID-19 · Blood type · SARS-CoV2 · Coronavirus

Introduction

COVID-19, the disease caused by the SARS-COV2 virus, has led to a global pandemic [1, 2]. The SARS-COV2 virus has

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had varying effects on the global population; those who are older and with comorbidities such as cardiovascular disease, diabetes, and pulmonary diseases have proven more vulnerable to severe disease [3–7]. Given the significant morbidity and mortality associated with COVID-19, there has been scientific interest in eliciting data that details characteristics that may render individuals more susceptible to COVID-19 infection and determining what risk factors may be associated with progression and severity of disease from the virus [8–11].

There have been numerous molecular level hypotheses raised for the variable susceptibility to disease and vulnerability to severe disease, such as the variable expression of ACE-2 expression in the airway epithelia [12]. Landsteiner's ABO

carbohydrate moieties are genetically inherited and previous reports have suggested a correlation between ABO blood type, cardiovascular disease, and cancers, as well as typing and susceptibility to certain infections, including SARS coronavirus [13–18]. In currently pre-printed data, Zhao et al. reported a possible association between blood type A and a higher risk for COVID-19 infection and mortality while blood group O was associated with a lower risk of infection and mortality [13]. Zietz and Tatonetti found that blood type A was correlated with a higher odds of testing positive for disease [19].

There is a paucity of data regarding the relationship between ABO blood typing and severity of COVID-19 disease. Using a large multi-institutional cohort of patients, this study aimed to determine if there is an association between ABO blood type and severity of COVID-19 disease as well as ascertain if there is variability in testing positive for COVID-19 between blood types.

Methods

Multi-institutional data were retrospectively reviewed on all patients with COVID-19 who presented to five major hospitals in the state of Massachusetts from March 6th to April 16th, 2020. The Partner's Healthcare System's Research Patient Data Registry (RPDR) was queried to document all patients who had a COVID-19 test performed in within the study period. Patients who tested positive for COVID-19 and had a blood type recorded in the health records were included in this study. Demographics, comorbidities, and laboratory markers of inflammation were reviewed. Patients less than 18 years of age were excluded. This study was reviewed and approved by the Massachusetts General Hospital institutional review board (IRB), and the requirement for individual informed consent was waived. All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (both institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008.

Definitions

Patient baseline comorbidities were determined by the International Classification of Diseases Codes, Ninth and Tenth Revision (ICD-9/10) which were assigned prior to the study period; all patients within the cohort had codes available prior to the study period. Comorbidities of interest were analyzed as binary variables and included hypertension, smoking (former or current), hyperlipidemia, chronic obstructive pulmonary disease (COPD), cardiovascular comorbidities (history of coronary artery

disease and stroke history), diabetes mellitus, a history of cancer, chronic kidney disease (CKD), end stage renal disease on hemodialysis (ESRD), cirrhosis, cardiac dysrhythmia, deep vein thrombosis (DVT), pulmonary embolism, and asthma. Patients were recorded as taking medications of interest if that medication was recorded on the medication list at an encounter within the year prior to a positive COVID-19 test. Medications of interest included aspirin, p2y12 inhibitor, anticoagulants, antihypertensives, and beta-blockers. Initial and peak lab values were recorded for creatinine, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), lactate dehydrogenase (LDH), and white blood cell count (WBC). Age and BMI were treated as continuous variables.

The primary outcome of interest was the composite of intubation or death. These occurrences, along with hospital admission, intensive care unit (ICU) admission, prone positioning while intubated, and initiation of extracorporeal membrane oxygenation (ECMO), were determined by manual chart review. Patient peak inflammatory marker values (WBC, LDH, CRP, and ESR) were also evaluated for correlation with blood type. Patients were followed until April 29th, 2020.

Proning referred only to proning while ventilated and not proning when not ventilated. Admission referred to admission in which the primary purpose for admission was for COVID-19 infection or any admission where treatment specifically related to COVID-19 took place. Death was defined as any mortality in which COVID-19 infection complications played a role in patient death.

Statistical analysis

Continuous variables were evaluated for normality as determined by skewness and kurtosis. The Chi-square test was utilized for categorical variables, and the ANOVA or Kruskal-Wallis was used, where appropriate, in comparing demographics, comorbidities, and medications across blood types. A univariate screen between blood type, demographics, comorbidities, and the outcomes of interest were performed using the Chi-square test for categorical variables and the Student's t test or the Wilcoxon rank-sum for continuous variables, as appropriate. Multivariable analysis was performed for the intubation and death composite variable using logistic regression; the model was built using a purposeful selection method with blood type and rhesus factor (Rh+) to be forced into the model at its completion, should they not already be selected for model inclusion. Purposeful selection inclusion criteria were P value < .20 on univariate analysis as the threshold for model inclusion; exit criteria was P > .10. Primary language, sex, age, and Rh+ were determined for adjustment as these covariates were thought to be potential confounders

Table 1 Demographics and patient comorbidities. *N* total = 1289. BMI: body mass index, COPD: chronic obstructive pulmonary disease, MI: myocardial infarction, ESRD: end-stage renal disease, CAD: coronary artery disease, CKD: chronic kidney disease, DVT: deep venous thrombosis, PE: pulmonary embolism, DOAC: direct oral anticoagulant. White refers to non-white, non-Hispanic

Factor	Level	Blood type A	Blood type B	Blood type AB	Blood type O	*P* value
N		440	201	61	587	
Age, mean (SD)		56.9 (18.6)	57.6 (18.1)	57.1 (19.9)	54.8 (18.1)	0.14
BMI, mean (SD)		30.8 (6.5)	30.6 (6.7)	29.4 (5.4)	32.0 (14.9)	0.32
Rhesus positive		392 (89.1%)	183 (91.0%)	53 (86.9%)	533 (90.8%)	0.63
Female sex		299 (68.0%)	136 (67.7%)	33 (54.1%)	404 (68.8%)	0.14
Language (primary)	English	328 (74.5%)	149 (74.1%)	54 (88.5%)	382 (65.1%)	<0.001
	Spanish	88 (20.0%)	36 (17.9%)	4 (6.6%)	180 (30.7%)	
	Other	24 (5.5%)	16 (8.0%)	3 (4.9%)	25 (4.3%)	
Race	White	221 (50.2%)	80 (39.8%)	28 (45.9%)	224 (38.2%)	0.008
	Black	84 (19.1%)	49 (24.4%)	15 (24.6%)	114 (19.4%)	
	Hispanic	52 (11.8%)	25 (12.4%)	4 (6.6%)	103 (17.5%)	
	Other	77 (17.5%)	41 (20.4%)	13 (21.3%)	128 (21.8%)	
	Not Reported	6 (1.4%)	6 (3.0%)	1 (1.6%)	18 (3.1%)	
Hypertension		256 (58.2%)	124 (61.7%)	42 (68.9%)	341 (58.1%)	0.34
Smoker		97 (22.0%)	39 (19.4%)	15 (24.6%)	117 (19.9%)	0.69
Hyperlipidemia		251 (57.0%)	105 (52.2%)	35 (57.4%)	323 (55.0%)	0.70
COPD		55 (12.5%)	26 (12.9%)	9 (14.8%)	72 (12.3%)	0.95
Diabetes mellitus		150 (34.1%)	66 (32.8%)	25 (41.0%)	197 (33.6%)	0.68
Cancer diagnosis		131 (29.8%)	57 (28.4%)	19 (31.1%)	161 (27.4%)	0.83
Cirrhosis		14 (3.2%)	9 (4.5%)	1 (1.6%)	21 (3.6%)	0.72
Asthma		124 (28.2%)	46 (22.9%)	14 (23.0%)	163 (27.8%)	0.44
History of stroke		50 (11.4%)	30 (14.9%)	10 (16.4%)	71 (12.1%)	0.47
ESRD		17 (3.9%)	13 (6.5%)	5 (8.2%)	30 (5.1%)	0.33
CAD		171 (38.9%)	71 (35.3%)	30 (49.2%)	231 (39.4%)	0.28
CKD		74 (16.8%)	41 (20.4%)	12 (19.7%)	111 (18.9%)	0.70
Dysrhythmia		199 (45.2%)	95 (47.3%)	34 (55.7%)	274 (46.7%)	0.49
Congestive heart failure		71 (16.1%)	41 (20.4%)	13 (21.3%)	107 (18.2%)	0.51
History DVT		39 (8.9%)	14 (7.0%)	6 (9.8%)	43 (7.3%)	0.71
History PE		26 (5.9%)	10 (5.0%)	4 (6.6%)	24 (4.1%)	0.55
Aspirin		66 (15.0%)	45 (22.4%)	16 (26.2%)	96 (16.4%)	0.029
Warfarin		14 (3.2%)	7 (3.5%)	1 (1.6%)	20 (3.4%)	0.90
Statin use		132 (30.0%)	64 (31.8%)	25 (41.0%)	141 (24.0%)	0.007
Calcium channel blocker		56 (12.7%)	29 (14.4%)	5 (8.2%)	76 (12.9%)	0.65
Thiazide diuretic		36 (8.2%)	14 (7.0%)	4 (6.6%)	46 (7.8%)	0.94
ACE inhibitor		59 (13.4%)	32 (15.9%)	7 (11.5%)	81 (13.8%)	0.78
ARB		32 (7.3%)	19 (9.5%)	6 (9.8%)	50 (8.5%)	0.76
Beta blocker		97 (22.0%)	51 (25.4%)	14 (23.0%)	124 (21.1%)	0.66
DOAC		31 (7.0%)	12 (6.0%)	6 (9.8%)	23 (3.9%)	0.071
p2y12 inhibitor		11 (2.5%)	11 (5.5%)	4 (6.6%)	9 (1.5%)	0.006

based on the primary model and these data were available for all patients who tested positive or negative. The model was run for each blood type against all others and with blood type as categorical to evaluate the independent effect of Rh+ status. Model diagnostics were performed using the Hosmer and Lemeshow Goodness of Fit test. An alpha level of ≤ .05 was utilized as the threshold for statistical significance. There were no missing data for any demographics, comorbidities, or medications. Statistical analysis for inflammatory marker outcomes included only patients who had such tests. All analyses were performed using Stata version 15.1 (StataCorp, College station, Texas, USA).

Table 2 Univariate analysis

Peak creatinine	1.8 (2.3)	1.9 (2.4)	1.5 (1.7)	1.7 (2.2)	0.64
Peak WBC, mean (SD)	9.9 (5.9)	10.2 (12.1)	10.9 (7.9)	8.9 (5.9)	0.25
Peak LDH, mean (SD)	484.8 (1180.4)	414.1 (198.0)	324.3 (141.3)	375.5 (165.0)	0.40
Peak ESR, mean (SD)	64.5 (37.9)	63.3 (36.4)	63.7 (37.9)	55.7 (33.4)	0.16
Peak CRP, mean (SD)	139.3 (110.3)	140.7 (97.8)	139.0 (116.5)	118.0 (95.3)	0.14
Admitted	158 (35.9%)	85 (42.3%)	28 (45.9%)	213 (36.3%)	0.20
ICU admission	41 (9.3%)	18 (9.0%)	7 (11.5%)	57 (9.7%)	0.94
Intubated	38 (8.6%)	15 (7.5%)	6 (9.8%)	49 (8.3%)	0.93
Required proning	18 (4.1%)	4 (2.0%)	2 (3.3%)	23 (3.9%)	0.58
ECMO	1 (0.2%)	2 (1.0%)	0 (0.0%)	0 (0.0%)	0.088
Dead	36 (8.2%)	14 (7.0%)	5 (8.2%)	34 (5.8%)	0.49
Intubation/death (ID)	63 (14.3)	23 (11.4)	8 (13.1)	68 (11.6)	0.57

WBC white blood count, *LDH* lactate dehydrogenase, *ESR* erythrocyte sediment rate, *CRP* C-reactive protein, *ICU* Intensive Care Unit, *ECMO* extracorporeal membrane oxygenation

Results

During the study period, there were 7648 symptomatic patients who received a COVID-19 test throughout the five institutions included. Of these, 1289 tests were positive and had their blood group documented hence included in the analysis; the demographics, comorbidities, and medications of this population are outlined in Table 1. Of these, 484 (37.5%) were admitted to hospital, 123 (9.5%) were admitted to the ICU, 108 (8.4%) were intubated, 3 (0.2%) required ECMO, and 89 (6.9%) died. Of the 1289 patients who tested positive, 440 (34.2%) were blood type A, 201 (15.6%) were blood type B, 61 (4.7%) were blood type AB, and 587 (45.5%) were blood type O. Six hundred and four COVID-19 positive patients had their WBC recorded (blood type A: 204, blood type B: 104, blood type AB: 35, blood type O: 261); 511 had their LDH evaluated (blood type A: 169, blood type B: 91, blood type AB: 29, blood type O: 222); 487 had their CRP evaluated (blood type A: 85, blood type B: 28, blood type AB: 212, blood type O: 487); and 393 had their ESR evaluated (blood type A: 130, blood type B: 71, blood type AB: 28, blood type O: 164). There was no association between blood type and any of the peak inflammatory markers (peak WBC, $p = 0.25$; peak LDH, $p = 0.40$; peak ESR, $p = 0.16$; peak CRP, $p = 0.14$). Moreover, there was no association between any of the clinical outcomes (admission $p = 0.20$, ICU admission $p = 0.94$, intubation $p = 0.93$, required proning while intubated $p = 0.58$, requiring ECMO $p = 0.09$, and death $p = 0.49$, Table 2).

In the multivariable analysis, blood type was not determined to be independently associated with COVID-19 disease severity (blood type A: ref., blood type B: AOR: 0.72, 95% CI: 0.42–1.26, blood type AB: AOR: 0.78, 95% CI: 0.33–1.87, blood type O: AOR: 0.77, 95% CI: 0.51–1.16), Rh+: AOR: 1.03, 95% CI: 0.93–1.86) (Table 3).

For the analysis evaluating for correlation of blood type with a positive test, blood type A had 440 (16.6%) positive tests, blood type B had 201 (19.4%) positive tests, blood type AB had 61 (19.8%) positive tests, and blood type O had 587 (16.1%) positive tests ($p = 0.036$). After multivariable

Table 3 Multivariable analysis: blood type versus intubation/death. Referent is blood type A. Also adjusted for sex, primary language, aspirin use, calcium channel blocker use, diagnoses of chronic kidney disease, coronary artery disease, prior stroke and diabetes mellitus, race not reported (referent: white), sex and presence of rhesus factor. Rh +: Rhesus factor positive. Hosmer and Lemeshow goodness of fit $p = 0.98$

Blood type	AOR	95% CI	*P* value
A	Ref		
B	0.72	0.42–1.26	0.25
AB	0.78	0.33–1.87	0.58
O	0.77	0.51–1.16	0.21
Rh+	1.03	0.93–1.86	0.10

Table 4 Rate of positive test by blood type. Overall *P* value 0.036 derived by Chi-squared testing. Adjusted odds ratio (AOR) with adjustment for sex, primary language, age, and rhesus factor with each blood type compared to all others. Rh+ model included blood type as a categorical covariate. Rh+: Rhesus factor positive. Hosmer and Lemeshow goodness of fit $p > .5$ for all models

Blood type	Total *N*	*N* positive (%)	AOR (95% CI)	*P* value
A	2649	440 (16.6)	1.00 (0.88–1.13)	0.96
B	1035	201 (19.4)	1.28 (1.08–1.52)	0.004
AB	308	61 (19.8)	1.37 (1.02–1.83)	0.035
O	3656	587 (16.1)	0.84 (0.75–0.95)	0.007
Rh+	6707	1161 (17.3)	1.22 (1.003–1.50)	0.047
All	7648	1289 (16.9)		

analysis, blood type A had no correlation with positive testing (AOR: 1.00, 95% CI: 0.88–1.13), blood type B was associated with higher odds of testing positive for disease (AOR: 1.28, 95% CI: 1.08–1.52), AB was also associated with higher odds of testing positive (AOR: 1.37, 95% CI: 1.02–1.83), O was associated with lower odds of testing positive (AOR: 0.84, 95% CI: 0.75–0.95), and Rh+ blood was associated with a higher odds of testing positive (AOR: 1.22 (1.003–1.50)) (Table 4).

Discussion

In this large, multi-institutional, retrospective review, there was no association noted between ABO blood type and COVID-19 disease severity defined as intubation or death. These data are different from that of Zhao et al. in the Wuhan experience who evaluated the association between blood type and mortality [13]. While there is data that ABO blood typing plays a role in disease acquisition and severity in other diseases, this was not the case in these data for COVID-19 [17–19]. The primary outcome of interest, composite intubation, or death yielded no association with blood type; furthermore, there were no significant associations with blood type and need for hospitalization, proning requirement while intubated or for any of the inflammatory markers reviewed in this study on univariate analysis. Given the lack of association between ABO subtype and severe disease found in these data as well as the preliminary data from Zietz and Tatonetti, ABO blood typing should not currently be considered prognostic in those who acquire the disease.

Blood type O had the lowest frequency of disease positivity, similar to Zhao et al., but blood type A had a lower frequency than blood types B and AB [13]. Both Zhao et al. and Zietz and Tatonetti, in their preliminary data, show correlation between blood type A and likelihood of positive testing and blood type O and likelihood of negative testing [13, 14]. The finding related to blood type O appears to correlate across our study and that of both Zhao et al. and Zietz and Tatonetti, but the blood type A correlation was not found in our study [13, 14]. This association of blood type O being less common in infection is the same as that found for SARS-CoV-1 [17]. The Rh+ association with disease positivity appears to be a novel finding and warrants further investigation. Given the relative rarity of rhesus negative blood types, these could not be stratified out by blood type given our patient numbers in this study.

A final element worthy of discussion is that there is certainly a racial element to ABO blood typing [20]. We were able to account for confounding factors of race and primary language through our multivariable models, likely isolating the effect of ABO blood typing, independent of ethnicity. However, the full effects of ethnicity on COVID-19 susceptibility and severity warrant further investigation.

Limitations

Our sample sizes are relatively small, with 483 hospitalized patients available for review and 1289 total positive patients that had blood type documented. It is possible that there may be a lead-time bias as some of the positive patients were early in their course and will go on to require hospitalization, require intubation, or die secondary to their disease. Additionally, this is an observational study, and while attempts were made to control for confounding, where such controls were possible, there is always the possibility that unmeasured confounding is driving the results.

Conclusions

Blood type is not associated with risk of progression to severe disease requiring intubation or causing death, nor is it associated with higher peak levels of inflammatory markers. Patients with blood types B and AB who received a test were more likely to test positive as were those who are Rh+ positive, and blood type O was less likely to test positive.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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