Creatinine levels on admission are main modulators of COVID-19 severity

Moni Nader
College of Medicine and Health Sciences, Khalifa University
Omar Zmerli
Saint George Hospital University Medical Center
Daniel E. Platt
IBM TJ Watson Research Centre
Hamdan Hamdan
College of Medicine and Health Sciences, Khalifa University
Salwa Hamdash
Haykel Hospital
Rami Abi Tayeh
Saint George Hospital University Medical Center
Jad Azar
Saint George Hospital University Medical Center
Diana Kadi
Saint George Hospital University Medical Center
Youssef Sultan
Saint George Hospital University Medical Center
Taha Bazarbachi
Haykel Hospital
Gilbert Karayakoupoglou
Haykel Hospital
Pierre Zalloua (pierre.zalloua@ku.ac.ae)
College of Medicine and Health Sciences, Khalifa University
Eid Azar
Saint George Hospital University Medical Center

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Abstract

Background: The COVID-19 pandemic claimed millions of lives worldwide without clear signs of abating. There have been tremendous interests in understanding the etiology of the disease particularly in what makes it fatal in certain patients.

Methods: This study investigated 819 COVID-19 patients admitted to the COVID-19 ward at a tertiary care center in Lebanon and evaluated their vital signs and biomarkers while probing for two main outcomes: intubation and fatality.

Results: Correlation analysis of various comorbidities revealed that hypertension, diabetes, being overweight, kidney disease, cardiovascular disease, autoimmune disease, and gender are independent risk factors for both intubation and fatality. Shortness of breath, age and being overweight correlated with intubation while fatality correlated with shortness of breath in our group of patients. Elevated level of serum creatinine was the highest correlating factor with fatality, while both white blood count and serum glutamic-oxaloacetic transaminase levels emerged as independent risk factors for intubation.

Conclusions: Collectively our data show that high creatinine levels are significantly associated with fatality in our COVID-19 study patients, underscoring the importance of kidney function as a main modulator of SARS-CoV-2 morbidity and favor a careful management of patients with elevated creatinine levels on admission.

Introduction

Prior to the current SARS-CoV-2 pandemic, two other coronavirus outbreaks were witnessed in the past two decades: the Severe Acute Respiratory Syndrome (SARS) in 2002– 2003, and the Middle East Respiratory Syndrome (MERS) in 2012 (1). The newly manifested SARS-CoV-2 pandemic has been initially associated with SARS (COVID-19). As the pandemic progressed, SARS-CoV-2 infections varied from asymptomatic to mild flu-like to severe symptoms leading to death in about 2% of cases (https://www.who.int/health-topics/coronavirus). In January 2020, The WHO declared the COVID-19 outbreak a public health emergency of international concern, and a global pandemic in March 2020. At the time of this writing, there were more than 410 million confirmed COVID-19 cases, with almost 5.8 million deaths Worldwide (https://covid19.who.int). The management of the disease varied from patient to patient (2) due to the paucity of information about the various symptoms at the onset of the pandemic, but more importantly from the lack of categorization of risk factors that were associated with high morbidity.

In some patients, during the course of infection, an imbalance of T helper-1 and T helper-2 cells in response to SARS-CoV2 invasion induced an inflammatory storm yielding an abundance of proinflammatory cytokines such as interleukin-1, interleukin-4, interleukin-6 and interleukin-10 (3). This cytokine storm, combined with chronic conditions such as cancer, diabetes, lung or kidney diseases and cardiac conditions, underscored critical and fatal COVID-19 outcomes. Other factors including obesity and gender have been shown to correlate with the severity of COVID-19 symptoms. The sickest COVID-19 patients, or those who were admitted to the intensive care unit (ICU) were often obese subjects irrespective of their age (4). While some comorbid factors for COVID-19 are established in certain populations, others have been shown to be population-specific (5, 6). For example, the fatality ratio in the majority of populations is about 3.5 male to female (7–9), except in India where COVID-19 fatality rate was shown to be higher in women than men (3.3% vs 2.9%) (10, 11).

The SARS-CoV-2 infection is not limited to the upper respiratory tract and lungs, but commonly affects multiple organs including the heart, liver, and kidneys (12–14). Moreover, multiorgan failure has been associated with many COVID-19 cases worldwide. Hospitalized COVID-19 patients often presented upon admission with acute respiratory distress syndrome, disseminated intravascular coagulation disease, venous thromboembolism, bone marrow failure, acute kidney injury, myocarditis, neurological manifestations, among others (15). Overall, a significant number of COVID-19 patients developed acute kidney injury (AKI) secondary to COVID-19. These variabilities in the clinical manifestations of COVID-19 call for a necessity to investigate various risk factors associated with fatality in COVID-19 patients and in different populations to account for inter-patient, clinical, and demographic variabilities.

According to the Ministry of Public Health in Lebanon, there were more than 1 million confirmed cases and more than 9,800 deaths at the time of this writing. Although 20% of the population was infected, and close to 2% of the confirmed infections were fatal, there is no systemic study that explored the risk factors associated with COVID-19 in Lebanon. Herein, we evaluated the clinical manifestations in a group of 819 COVID-19 patients with critical/fatal and non-critical illness from one of the largest tertiary care centers in Lebanon. We investigated and identified several risk factors for their association with severe COVID-19 and/or fatality. These factors can be used to build a comprehensive clinical platform for a better management of COVID-19 patients.

Materials And Methods

Patients

We investigated 819 COVID-19 patients who were admitted between January 2020 and April 2021 to the COVID-19 ward at a major tertiary care hospital in Lebanon. Patients were evaluated upon admission and were admitted to the COVID-19 ward if they tested positive for the SARS-CoV-2 by quantitative real-time reverse transcriptase-polymerase chain reaction (qRT-PCR) of nasopharyngeal swabs using LightMix Modular SARS-CoV-2 E, N, and RdRP-genes (TibMolbiol, Berlin, Germany) performed according to manufacturer's manual. COVID-19 diagnosis was made according to the World Health Organization guidance (https://www.who.int/publications/i/item/diagnostic-testing-for-sars-cov-2). Anonymized electronic medical records of all patients were retrieved and reviewed for epidemiological, demographic, clinical and laboratory data.

Clinical data on admission included clinical symptoms, biochemical markers on admission, previous medical conditions, oxygen requirement, the need for and the duration of intubation, and the length of hospital stay.

Statistical analysis

Data were managed using pandas (ver 1.1.2)(16). Multivariate logistic regressions were performed using statsmodels (ver 0.12.0) (17) and coxph in R (survival ver 3.2.1, survminer ver 0.4.9) (18). Categorical and quantitative variable definitions are defined in Table S1. The two main outcome variables tested were fatality or "severe" COVID-19 (defined as requiring intubation). The comparisons of the quantitative/categorical data were statistically evaluated using pandas.

Ethical Considerations. This study was approved by the Ethics Committee of University of Balamand/Saint George Hospital University Medical Center (IRB-REC/O1018-2011320). Consenting to participate in the study was not required, and all patient data was anonymized before it was transferred to the research team for analyses. This study was granted an exemption from requiring informed consent by the Ethics Committee of University of Balamand/Saint George Hospital University Medical Center (IRB-REC/O1018-2011320).

Results

In total, 819 hospital-admitted COVID-19 patients were enrolled in this study with 546 male and 273 female patients (66.77% M and 33.33% F). The comparison for each of the variables presented in Tables 1a and 1b showed no difference between male and female groups, with admission ratio being 1/3 female to male, suggesting that gender does not influence these variables in our population. Table 1b shows that there were more intubated males with shortness of breath than female (23.18 vs 16.42 %, and 72.48 vs 59.78 %, respectively). Males who presented with anosmia were almost double than females (3.30% vs 1.48%), albeit anosmia constituted a very small percentage of the hospitalized patients. Female patients were four times more likely to have autoimmune disease than males (8.21% vs 2.94%).

Overall, the intubation of COVID-19 subjects was highly associated with fatality (OR 71.20, CI[41.33-122.64], p <0.001) and with the oxygen consumption volume (OR, 3.18 CI[0.02-0.06], p<0.001) (Table 1c).

The fatality rate between male and female COVID-19 patients however was not significant (27% and 23% respectively, Figure 1) .

Applying a Cox regression of time to death on the time-constant covariates (age, WBC, asthma and overweight) revealed a significant implication of these factors as shows in the predictive survival proportion, at any given point, for the above-mentioned covariates shown in Figure 2.

Common comorbidities for intubation and fatality

We interrogated hypertension, diabetes, being overweight (BMI $\geq$ 25kg/m^2), kidney disease, CVD, autoimmune disease, sex, asthma, and age against the prevalence of intubation in COVID-19 patients. We found that diabetes (OR 1.57, CI[1.07-2.32], p=0.02), being overweight (OR 0.65, CI[0.46-0.94], p=0.02), kidney disease (OR 1.70, CI[1.01-2.84], p=0.04), and sex (OR 0.61, CI[0.41-0.91], p=0.016) are independent risk factors for intubation, with age conferring the most significant intubation risk (OR 2.03, CI[1.34-3.08], p=0.001) (Table 2a). When assessing for Fatality as an outcome variable (Table 2b), a similar trend was noticed, with age ($\geq$ 65 years) scoring the highest significance as an independent risk factor (OR 2.93, CI[1.97-4.34], p=0.000). However, being overweight did not show significance (p=0.07) in this analysis.

Correlation of symptoms at presentation with intubation and fatality

We further evaluated abdominal pain/diarrhea, fever/chills, fatigue, myalgia, headaches, cough, rhinorrhea, sore throat, and shortness of breath in our group of COVID-19 patients. Having shortness of breath (OR 2.97, CI[1.87-4.70], p=0.000), age (OR 2.60, CI[1.78-3.80], p=0.000), and gender (OR 0.64, CI[0.43-0.96], p=0.03) significantly correlated with intubation of these patients (Table 3a). Shortness of breath on admission was one of the most important risk factors driving patients into intubation. Further, when probing for fatality, data in table 3b showed that death of COVID-19 patients significantly correlated with shortness of breath (OR 2.63, CI [1.76-3.93], p=0.000).

Biomarkers of intubation and COVID-19 fatality

We investigated serum levels of platelets, ferritin, CRP, Hb, creatinine, WBC, SGOT and SGPT and analyzed these using intubation as an independent variable. CRP (OR 1.48, CI [1.21-1.81], p=0.000), WBC (OR 2.70, CI [1.68-4.32], p=0.000), SGOT (OR 3.08, CI [1.88-5.03], p=0.000), and SGPT (OR 0.44, CI [0.23-0.81], p=0.01) emerged as independent biomarkers for the intubation of these patients, while platelets, ferritin, Hb, and creatinine did not score significance. Out of these biomarkers WBC and SGOT (AST) were the most significantly correlated with intubation (Table 4a). These same biomarkers were interrogated against fatality as an independent variable. Platelets (OR 0.78, CI [0.61-0.97], p=0.029), CRP (OR 1.39, CI [1.14-1.69], p=0.001), creatinine (OR 2.02, CI [1.30-3.12], p=0.002), WBC (OR 3.25, CI [2.07-5.08], p=0.000), and SGOT (AST) (OR 2.18, CI [1.35-3.5], p=0.001) emerged as independent biomarkers of fatality in COVID-19 patients. WBC, platelets, and creatinine levels were among the most significant biomarkers correlating with fatality (Table 4b).

Data in Table 5 show that creatinine and/or diabetes are strongly correlated with hypertension in our group of patients. Similarly, diabetes and hypertension were associated with high creatinine levels, while hypertension strongly correlates with kidney disease. It is worth noting that diabetes and hypertension together did not show a significant association neither with kidney disease nor with high creatinine levels. Therefore, creatinine levels, hence, kidney injury, remain as the most significant risk factor for fatality namely in hypertensive subjects.

Discussion

The SARS-CoV-2 pandemic and its resulting COVID-19 illness have affected the lives of millions around the globe posing a severe worldwide public health challenge (2). This illness revealed deadly in patients with comorbidities namely cardiac conditions, diabetes, and kidney injury which materialized a pressing need to attenuate the viral spread (12, 13). While treatment protocols are now starting to converge, the identification of the numerous factors leading to COVID-

19 morbidity and fatality remains under extensive investigation. Herein we show that high levels of serum creatinine are major determinants of fatality among COVID-19 hospitalized patients who also tend to be hypertensive.

It has been established that males are more prone to hospitalization, intubation, and death than women (19). While we noticed a hospitalization ratio of 3:1 in males vs. female in our study population, the death rate post hospitalization between both sexes was similar, indicating that gender does not contribute to mortality in admitted COVID-19 patients.

The rate of autoimmune disease in the hospitalized COVID-19 population was substantially higher than those observed for common autoimmune diseases around the world

(https://nationalstemcellfoundation.org/glossary/autoimmune-disease/). While autoimmune diseases tend to be more prevalent in women more than men, being female tended to be protective in our dataset (20, 21). Our results suggest that autoimmune diseases may have promoted hospitalization, but the small numbers of patients with autoimmune diseases limited our ability to test significance.

Our patients showed increased WBC which was significantly correlated with both intubation and fatality of these patients. The levels of WBC in the settings of COVID-19 hospitalization remain debatable and the interaction between the immune system and SARS-CoV-2 is not yet fully understood. While studies report a decrease in WBC, others showed a correlation between WBC and fatality of COVID-19 patients (22–26).

We also note that ALT was significantly elevated among severe COVID-19 cases, but AST was not. This is typical for ischemic hepatic damage or skeletal/cardiac muscle (27) breakdown in contrast with toxicity induced damage (28), and would be expected among patients with severe COVID-19. This ALT-predominance further highlights the extrahepatic effect of the virus, possibly causing myositis. Our data is in accord with other studies showing that ALT levels are increased in severe cases of COVID-19 (29), and that high levels of ALT correlate with ICU admission in COVID-19 patients (30, 31).

Our finding that elevated creatinine, predominately in hypertensive patients, are highly associated with intubation and fatality of COVID-19 patients is of great importance since hypertension is rampant among adults in Lebanon (32). This is in accord with other reports showing a 2.5-fold increased risk of severity and mortality in hypertensive COVID-19 (33). The correlation between hypertension, elevated creatinine and COVID-19 severity maybe related to underlying kidney disease, hypovolemic kidney injury and or some failure of the renin-angiotensin system in response to SARS-CoV-2 (34). Yet, acute increase in blood pressure is also manifested in COVID-19 subjects (35) and whether hypertension is a risk factor for fatality of these patients, or whether it is a vital parameter indicative of severity remains to be determined.

These results point to a synergistic effect between kidney injury and hypertension that is modulating the progression of the COVID-19 illness. In fact, there was a strong correlation between increased levels of creatinine and kidney disease in our study group. A correlation that is in line with common medical practices adopting increased creatinine levels, chief among other factors as biomarker for kidney injury and the major component of eGFR (36). Elevated levels of creatinine scored the highest odds among all other risk factors in our study group, suggesting that kidney function upon admission maybe an important prognostic determinant of COVID-19 disease.

Our findings are in accord with others showing that acute kidney injury (AKI) is associated with high mortality rate in COVID-19 subjects (37). AKI is a clinical syndrome manifested by a decline in kidney function and is closely associated with morbidity and mortality (38). AKI was observed in 5–15% of SARS and MERS cases with a high mortality rate (60–90%). Some reports indicated that the incidence of AKI is inconsistent among patients with COVID-19 (39, 40). It was recently reported that COVID-19 patients with high levels of serum creatinine were more susceptible to develop AKI during hospitalization, an observation coherent with similar findings in SARS subjects (37, 41). The SARS-CoV-2 particles invade the kidneys and induce cellular damage leading to renal dysfunction (12). These viral particles were found in kidney tissues (autopsies) associated with podocytes injury leading to acute tubular necrosis protein leakage in Bowman's capsule, collapsing glomerulopathy, and mitochondrial impairment (42). The SARS-CoV-2 can also infiltrate into the renal tissues with inflammatory cells expressing CD147, thus leading to dysregulation of cell cycle and inflammatory response (43). Although it remains undiscernible whether AKI was caused by SARS-CoV-2/cytokine storm, hypovolemic status, or whether our patients have chronic kidney disease prior to their SARS-CoV-2 infection that exacerbated the condition. There is lack of autopsies in Lebanese COVID-19 subjects who died from the disease, and therefore the presence of SARS-CoV-2 in these kidneys remains uncertain. It is however evident that kidney function must be closely monitored in COVID-19 subjects. The strong association of increased creatinine levels and fatality of COVID-19 patients poses a high risk to subjects on hemodialysis who could possibly contract the SARS-CoV-2.

When measuring the interaction between hypertension, T2D and high creatinine levels, each of the variables (hypertension or T2D) highly correlated with increased creatinine levels when probing for intubation and fatality. T2D did not promote the infection by SARS-CoV-2 however it exacerbates the condition in COVID-19 patients (44). When investigating COVID-19 severity risk with hypertension and T2D in an interactive analysis, the increased creatinine levels failed to significantly correlate with intubation and fatality. Thus, our data suggest that diabetic patients who are not hypertensive and with elevated creatinine levels were less likely to get intubated or die from COVID-19 compared to hypertensive patients with elevated creatinine levels. Hypertension and obesity were also significantly correlated with the severity of the disease and with high mortality rate, possibly due to their higher frequency in the general population.

It is worth noting that all COVID-19 patients in this study were not vaccinated and therefore, the risk factors described in our population exerted their effects in unprimed individuals. Although, the disparities in the spread and infectivity of the different SARS-CoV-2 variants are highly related to mutations/modifications in the viral proteins involved in the interaction with the host cell (45), it remains unclear if the medical condition of the host subject is a risk factor for the COVID-19 ailment.

In conclusion, we found that together hypertension and increased creatinine levels are comorbid factors that cannot be ignored in COVID-19 subjects in view of their significant association with intubation and fatality. We therefore advocate for a complete appraisal of the biomarkers underlining kidney injury and hypertension to guide a therapeutic strategy for COVID-19. We also recommend a firm monitoring of these elements in every COVID-19 subject upon

admission to avoid deadly complications. Finally, special attention must be given to hypertensive subjects with reduced kidney function to reduce their mortality rate from SARS-CoV-2 infection.

Declarations

Conflict of interest statement: None declared.

Authors contributions: PZ, EA: study design and article revision; MN, OZ: drafting the article DEP: analysis and data interpretation; HH, SH, RAT, JA, DK, YS, TB, GK providing intellectual content of critical importance to the work described.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of University of Balamand/Saint George Hospital University Medical Center (IRB-REC/O1018-2011320). Consenting to participate in the study was not required, and all patient data was anonymized before it was transferred to the research team for analyses. This study was granted an exemption from requiring informed consent by the Ethics Committee of University of Balamand/Saint George Hospital University Medical Center (IRB-REC/O1018-2011320). All methods were carried out in accordance with relevant guidelines and regulations as stated in the ethical approval letter of committee.

Consent for publication

The data used in this study are entirely unidentifiable and there are no details on patients reported within the manuscript.

Availability of data and materials

The raw data set from which the analyses was carried are not publicly available due to

The raw dataset used to derive the tables and figures included in this the current study is available from the corresponding author on reasonable request due to the sensitivite nature of the information included.

Competing interests

Not applicable

Funding

Not applicable

Authors' contributions

PZ, EA: study design and article revision; MN, OZ: drafting the article DEP: analysis and data interpretation; HH, SH, RAT, JA, DK, YS, TB, GK providing intellectual content of critical importance to the work described.

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Authors' information

Provided above

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Tables

Table 1a. Descriptive statistics of the continuous variables in the COVID-19 population.

	Mean	Std Dev	F Mean	F Std Dev	M Mean	M StdDev
Age (years)	61.64	17.87	61.52	20.50	61.69	16.42
Weight (kg)	83.24	20.73	74.69	19.96	87.34	19.85
Height (m)	1.69	0.13	1.60	0.14	1.73	0.11
BMI (kg/m^2)	28.72	5.48	28.51	6.20	28.82	5.10
WBC (10^3/µl)	9.35	5.20	9.13	5.97	9.45	4.77
Hb (g/dL)	13.12	2.19	12.08	1.84	13.63	2.17
Platelets(10^3/µl)	238.09	106.50	247.46	118.90	233.42	99.55
D Dimer (ng/mL)	1.31	3.38	1.20	1.73	1.37	3.94
Creatinine(mg/dl)	1.25	1.08	1.19	1.16	1.27	1.04
SGOT (AST)	66.62	212.23	72.06	269.96	64.09	179.49
SGPT (ALT)	54.10	131.66	49.83	132.17	56.08	131.51
Ferritin (ng/ml)	906.96	661.67	657.25	634.16	1029.92	640.69
CRP (mg/l)	7.07	8.34	5.99	8.60	7.59	8.17

Table 1b. Descriptive statistics of the COVID-19 population stratified by medical condition.

	Count	Rel Freq %	F Count	F Rel Freq %	M Count	M Rel Freq %
Total	819.00	100.00	273.00	100.00	546.00	100.00
SexC	273.00	33.33	273.00	100.00	0.00	0.00
AgeC	390.00	47.62	136.00	49.82	254.00	46.52
FatalityC	213.00	26.01	64.00	23.44	149.00	27.29
Intubated	168.00	20.92	44.00	16.42	124.00	23.18
Asthma	29.00	3.55	12.00	4.43	17.00	3.11
COPD	37.00	4.53	11.00	4.06	26.00	4.76
Cancer	70.00	8.57	33.00	12.18	37.00	6.78
Kidney Disease	88.00	10.77	29.00	10.70	59.00	10.81
CVD	282.00	34.52	78.00	28.78	204.00	37.36
Diabetes	232.00	28.61	78.00	29.10	154.00	28.36
Hypertension	418.00	51.41	130.00	48.51	288.00	52.84
Autoimmune disease	38.00	4.67	22.00	8.21	16.00	2.94
Overweight	471.00	57.51	142.00	52.01	329.00	60.26
Obese	216.00	26.37	72.00	26.37	144.00	26.37
Abdominal pain/ diarrhea	155.00	19.00	59.00	21.77	96.00	17.61
Anosmia	22.00	2.70	4.00	1.48	18.00	3.30
fever/ Chills	512.00	62.82	149.00	54.98	363.00	66.73
Fatigue	261.00	32.18	78.00	28.89	183.00	33.83
Myalgia	268.00	32.88	79.00	29.15	189.00	34.74
Headaches	72.00	8.83	16.00	5.90	56.00	10.29
Cough	413.00	50.67	127.00	46.86	286.00	52.57
Sore throat	50.00	6.13	11.00	4.06	39.00	7.17
Shortness of breath	557.00	68.26	162.00	59.78	395.00	72.48

Table 1c. Binomial logistic regression odds ratios predicting fatality from oxygen volume requirement and intubation

	O_2 Volume				Intubation			
	OR	95CI-	95CI+	P-val	OR	95CI-	95CI+	P-val
Fatality	-	-	-	-	71.201	41.337	122.64	$2.393´10^{-62}$
Fatality	3.185	2.393	4.240	$2.039´10^{-15}$	-	-	-	-

Table 2.a Binomial logistic regression odds ratios of discrete variables predicting intubation from joint conditions as exposures.

Severity	OR	95CI-	95CI+	P-val
Hypertension	1.049	0.703	1.565	0.814
Diabetes	1.577	1.071	2.322	0.021
Overweight	0.653	0.455	0.937	0.021
Asthma	0.495	0.143	1.719	0.269
Kidney Disease	1.697	1.013	2.844	0.045
CVD	1.103	0.731	1.664	0.640
Autoimmune disease	0.555	0.189	1.630	0.284
SexC	0.609	0.407	0.911	0.016
AgeC	2.029	1.338	3.079	0.001

Table 2.b Binomial logistic regression coefficients of discrete variables predicting fatality from joint conditions as exposures.

Fatality	OR	95CI-	95CI+	P-val
Hypertension	1.111	0.762	1.619	0.586
Diabetes	1.471	1.019	2.124	0.040
Overweight	0.730	0.518	1.028	0.072
Asthma	0.338	0.097	1.182	0.089
Kidney Disease	2.253	1.378	3.684	0.001
CVD	1.373	0.938	2.008	0.103
Autoimmune disease	1.652	0.779	3.507	0.191
SexC	0.740	0.510	1.074	0.114
AgeC	2.927	1.972	4.344	0.000

Table 3.a Binomial logistic regression coefficients of various factors predicting intubation from joint symptoms as exposures.

Severity	OR	95CI-	95CI+	P-val
Abdominal pain/ diarrhea	1.324	0.838	2.091	0.229
fever/ Chills	0.753	0.511	1.111	0.153
Overweight	0.632	0.437	0.913	0.014
Fatigue	0.850	0.556	1.299	0.452
Myalgia	1.000	0.647	1.544	0.998
Headaches	0.490	0.205	1.172	0.109
Cough	1.110	0.764	1.613	0.583
Rhinorrhea	1.895	0.784	4.581	0.156
Sore throat	1.206	0.534	2.723	0.653
Shortness of breath	2.975	1.878	4.712	0.000
AgeC	2.608	1.786	3.807	0.000
SexC	0.637	0.424	0.957	0.030

Table 3.b Binomial logistic regression coefficients of various factors predicting fatality from joint symptoms as exposures.

Fatality	OR	95CI-	95CI+	P-val
Abdominal pain/ diarrhea	1.255	0.814	1.933	0.304
fever/ Chills	0.888	0.615	1.281	0.526
Overweight	0.713	0.506	1.006	0.054
Fatigue	0.870	0.588	1.287	0.485
Myalgia	1.151	0.770	1.721	0.493
Headaches	0.523	0.236	1.159	0.110
Cough	0.928	0.654	1.317	0.677
Rhinorrhea	1.504	0.623	3.630	0.364
Sore throat	0.885	0.392	1.998	0.769
Shortness of breath	2.628	1.756	3.932	0.000
AgeC	4.239	2.957	6.075	0.000
SexC	0.779	0.537	1.129	0.187

Table 4.a Binomial logistic regression coefficients of continuous variables predicting intubation from joint blood panels as exposures.

Severity	OR	95CI-	95CI+	P-val
PlateletsC	0.927	0.738	1.166	0.519
FerritinC	2.102	0.907	4.872	0.083
CRPC	1.484	1.214	1.813	0.000
HbC	0.915	0.725	1.156	0.457
CreatinineC	1.274	0.790	2.055	0.321
WBCC	2.701	1.689	4.320	0.000
SGOTC	3.077	1.880	5.038	0.000
SGPTC	0.440	0.236	0.819	0.010

Table 4.b Binomial logistic regression coefficients of continuous variables predicting fatality from joint blood panels as exposures.

Fatality	OR	95CI-	95CI+	P-val
PlateletsC	0.776	0.618	0.974	0.029
FerritinC	1.210	0.635	2.305	0.562
CRPC	1.390	1.142	1.691	0.001
HbC	0.833	0.672	1.032	0.095
CreatinineC	2.021	1.308	3.125	0.002
WBCC	3.246	2.073	5.081	0.000
SGOTC	2.178	1.355	3.501	0.001
SGPTC	0.588	0.325	1.062	0.078

Table 5. Interaction of the different risk factors of COVID-19 probed against fatality

	Diabetes				Hypertension				Diabetes & Hypertension				Creatinine			
	OR	95CI-	95CI+	P-val	OR	95CI-	95CI+	P-val	OR	95CI-	95CI+	P-val	OR	95CI-	95CI+	P-v
Kidney Disease	-	-	-	-	-	-	-	-	-	-	-	-	14.034	7.956	24.75	0.0
Kidney Disease	0.981	0.593	1.621	0.09	1.636	1.017	2.630	0.04	-	-	-	-	-	-	-	-
Kidney Disease	0.994	0.368	2.684	0.98	1.642	0.960	2.808	0.07	0.983	0.311	3.108	0.97	-	-	-	-
Creatinine	1.644	1.173	2.305	0.000	2.887	2.070	4.024	0.000	-	-	-	-	-	-	-	-
Creatinine	1.448	0.747	2.807	0.27	2.763	1.881	4.057	0.000	1.189	0.550	2.572	0.65	-	-	-	-
Hypertension	3.908	2.790	5.473	0.002	-	-	-	-	-	-	-	-	-	-	-	-
Hypertension	-	-	-	-	-	-	-	-	-	-	-	-	3.263	2.367	4.497	0.0
Hypertension	3.498	2.476	4.941	0.000	-	-	-	-	-	-	-	-	2.887	2.071	4.024	0.0

Figures

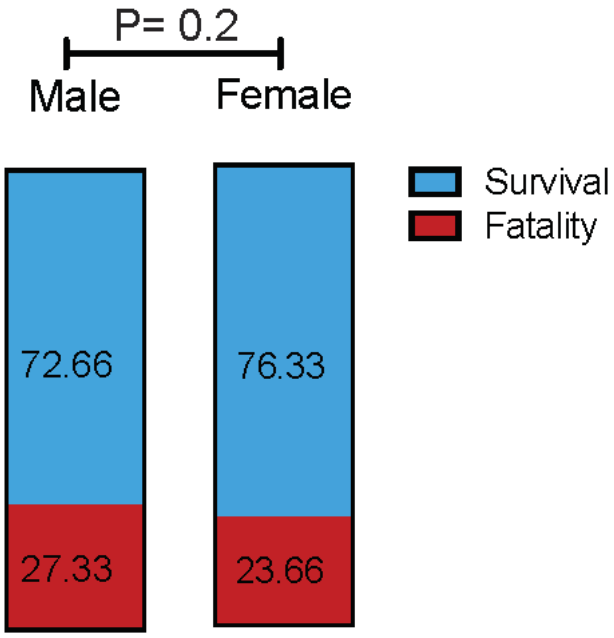

Figure 1

Histograms showing the percentage of fatality in male and female admitted to the ICU. P <0.02 was determined used the Chi-Square test.

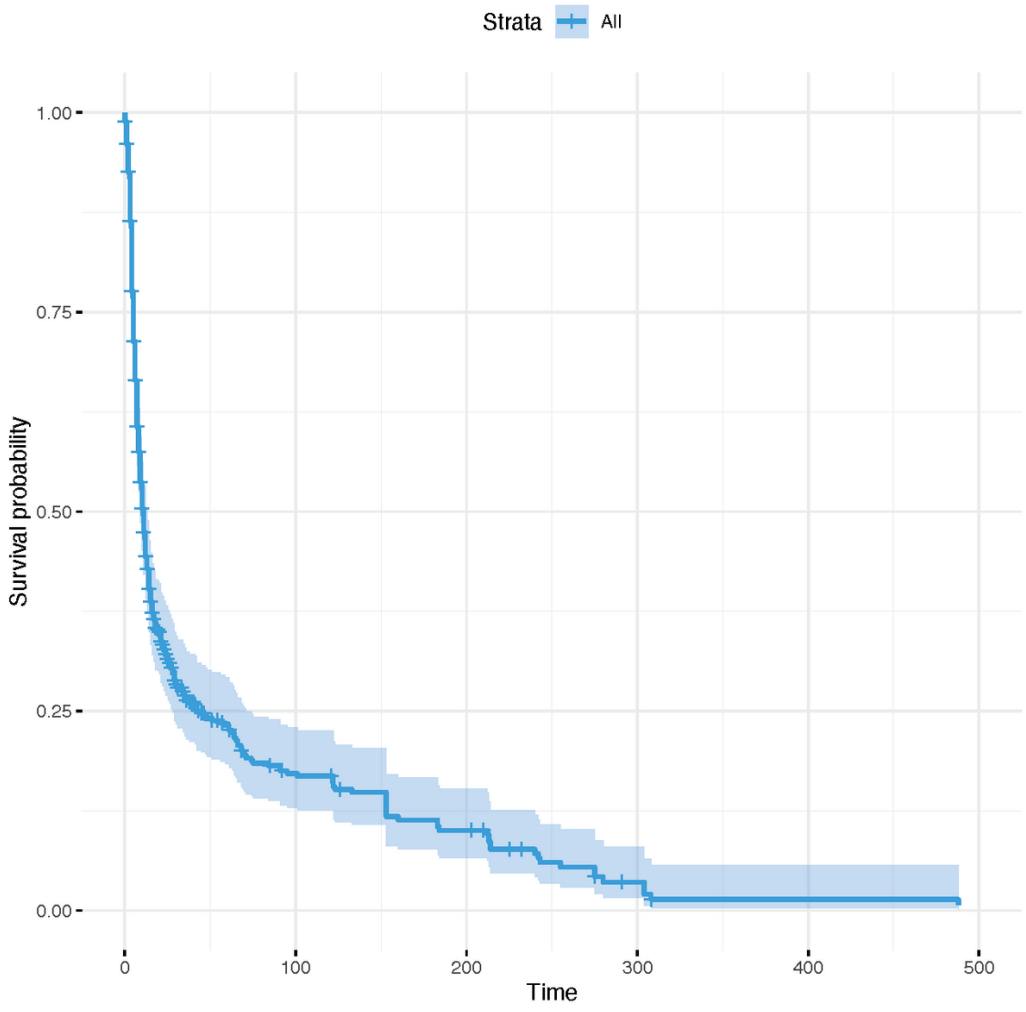

Figure 2

Cox regression predicting the hazard ratio from overweight, asthma, and high white blood count.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- TableS1.doc