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ORIGINAL RESEARCH **CORONA EXPERIENCE**

Survival of a cohort of COVID-19 patients on mechanical ventilation in the ICU of a Level III hospital in Bogotá, Colombia

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ABSTRACT

Background & Objective: The COVID-19 mortality varies from country to country. The pandemic has enriched our knowledge about the pathogenesis of the viral diseases in the human race. We aimed to establish survival curves based on a population of patients with confirmed diagnosis of COVID-19 who received mechanical ventilation in ICU of our level-III hospital.

Methodology: It is an observational study with a prospective cohort. All patients with a positive RT-PCR test for SARS-CoV-2 after the nasopharyngeal swab and who needed mechanical ventilation were identified. Kaplan–Meier survival estimates were calculated until the follow-up date of September 26, 2020.

Results: We included 62 patients in the study. The bivariate analysis found that congestive heart failure, kidney injury, dysnatremia, acid-base disorders, delta CO_2, and lactate levels were associated with higher mortality. When performing backward-type Cox regression, it was observed that the CO_2 delta [Hazard Ratio (HR) = 14.78, 95% CI: 2.1-100.5, P = 0.00] and lactate levels (HR = 6.15, 95% CI: 1.1-34.2, P = 0.038), were associated with an increase in mortality, for the admission variables. And only dysnatremia (HR = 5.28, 95% CI: 1.8-15.3, P = 0.00) and acid-base disorders (HR = 12.04: 95% CI: 4.1-35.3, P = 0.00) for the follow-up variables.

Conclusions: Among the factors associated with increased mortality in COVID-19 patients on mechanical ventilation in the first 24 h, parameters related to tissue hypoperfusion were identified. For the variables associated with mortality at 14 days, dysnatremia and acid-base disorders were identified.

Abbreviations: ng - Nanogram; FiO_2 - Inspired Oxygen Fraction, PEEP - Positive End-Expiratory Pressure; PAFI - Index of PaO_2/FiO_2; Delta CO_2 - Venous-to-Arterial Carbon Dioxide Difference; SIC - Sepsis-Induced Coagulopathy; SOFA - Sequential Organ Failure Assessment; NEW2 - National Early Warning Score 2

Key words: COVID-19; Critical Care; Mortality; Pulmonary Ventilation; Respiratory Distress Syndrome, Adult

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1. INTRODUCTION

Coronavirus disease 2019 (COVID-19) is the name given to the disease caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), which was first described in December 2019 in patients from Wuhan city, Hubei province, China.[1] From December 2019 to the present, the causative organism, SARS-CoV-2, has spread worldwide, infecting 548.990.094 people and causing 6.341.637 deaths, making this disease a public health problem at the global level.[2]

Epidemiological studies have shown that 6-10% of patients develop more severe form of COVID-19 and require admission to an intensive care unit (ICU) for acute hypoxemic respiratory failure. These patients also require mechanical ventilation, resulting in a prolonged hospital stay and an increased likelihood of mortality;[1] there are also reports of higher infection rates contributing to mortality.[3,4]

To date, few studies have been carried out in critically ill patients in Colombia; additionally, differences in demographics and population behavior may limit the generalizability of some findings. Therefore, it is imperative to evaluate the risk factors associated with mortality and survival of patients diagnosed with COVID-19 in a cohort of patients who were hospitalized and received mechanical ventilation in the ICU of a Level III hospital in the city of Bogotá, Colombia.

2. METHODOLOGY

An observational study was carried out with a prospective cohort of patients admitted to the ICU at a Level III institution in Bogotá, Colombia.

Patients' electronic medical records were reviewed and collected by trained intensive care physicians following a standardized protocol. Each researcher entered the data into an online data acquisition system explicitly predesigned for this purpose [CICERO (CovId-19 meChanical vEntilation Research grOup)].

All consecutive COVID-19 patients included in the dataset from March 1, 2020 to September 26, 2020, and who met the following inclusion criteria: ≥18 y of age, intubated and mechanically ventilated, confirmed SARS-CoV-2 infection by evidence-based RT–PCR analysis of a respiratory tract sample. The following patients were excluded: patients with unconfirmed SARS-CoV-2 disease, those without data at the beginning of the study, those without ventilator parameter information, or patients who were not intubated.

All the data were collected within the first 24 h of admission to the ICU. Data from days 3, 5, 7, and 14 were used to estimate a measure of central tendency relative to follow-up. The collected data included demographic characteristics, comorbidities, symptoms at the ICU admission, vital signs, paraclinical studies, ventilator parameters, complementary therapies, such as prone position and pharmacological treatments, and severity assessment scores.

Statistical analysis

The selected predictors of mortality were used as dichotomous variables, and the cutoff points were based on previous studies and included the following: creatinine (> 2 mg/dL), sodium (135-145 mEq/L; values outside this range indicated dysnatremia), potassium (3.5-5 mEq/L), pH (7.35-7.45; values outside this range indicated acid-base disorders), PaO_2 (< 65 mmHg), $PaCO_2$ (30-35 mmHg), bicarbonate in arterial blood (18-22 mEq/L), oxygen extraction (20-30%), arterial saturation (< 90%), venous saturation (70-80%), lactate levels (< 2 mmol/L) and sepsis-induced coagulopathy (SIC) score < 2).

Survival analysis was performed using the Kaplan–Meier method; the onset of symptoms was considered the time point when monitoring began. The dates were available in the database, and the following was used to create time intervals for the survival analysis: time (T1) from the beginning of symptoms to death/end of follow-up (discharge from the ICU) in days (d). There were no losses to follow-up, and competitive risks were ignored when assuming that all the deaths were influenced by COVID-19 infection within their causal complex; thus, the only censor in patients who did not present the event was the cutoff date of the study. Considering that the exact moment of the day at which each patient enrolled in or left the investigation is unknown, the days of follow-up were counted in a non-inclusive way; that is,

Table 1: Demographic characteristics and variables of the cohort at admission (N = 62)

Variable	N (%)	HR	95% CI
Age > 60 y	34 (54.8)	1.26	0.6−2.5
Body mass index ≥ 30 (kg/m^2)	10 (16.1)	1.42	0.5−3.7
Symptoms at admission			
• Fever	30 (48.4)	2.0	0.4−8.5
• Difficulty breathing	43 (69.4)	1.37	0.5−3.1
• Diarrhea	4 (6.5)	0.9	0.2−2.9
• Cough	39 (62.9)	1.01	0.5−2.0
• Myalgia	13 (21)	1.50	0.6−3.6
Cause of admission			
• ARDS	14 (22.6)	1.11	0.5−2.3
• COPD	8 (12.9)	0.84	0.3−2.0
• Pneumonia	51 (82.3)	1.89	0.8−4.0
• Congestive heart failure	7 (11.3)	2.90	1.1−7.1
Comorbidities			
• Hypertension	24 (38.7)	1.84	0.8−3.7
• Coronary heart disease	4 (4.8)	1.72	0.5−5.7
• Renal disease	2 (3.2)	1.18	0.1−8.7
• Diabetes	8 (12.9)	1.02	0.3−2.9
• Obesity	12 (19.9)	2.78	0.8−9.0
• Malignancy	2 (3.2)	2.63	0.6−11.1
• Temperature >38.3°	4 (6.5)	2.00	0.4−8.5
• SBP < 90 mmHg	8 (12.9)	1.12	0.4−2.9
• Heart rate > 100 BPM	20 (32.3)	1.87	0.9−3.7
• Respiratory rate > 30 /min	7 (11.3)	1.24	0.4−3.6

ARDS = Adult Respiratory Distress Syndrome; COPD = Chronic Obstructive Pulmonary Disease; BPM = Beats per minute; HR = Hazards Ratio; SBP = Systolic blood pressure

one day was added to the final count to account for this uncertainty.

A survival function estimation was performed using the global Kaplan–Meier method, and subsequently, a bivariate analysis was performed with each of the variables mentioned above. The difference between these curves was evaluated using a log-rank test. As an estimator of association, an HR (hazard ratio) was used with a backward-type Cox regression method; this analysis included the statistically significant variables in the bivariate analysis, and variables such as age and comorbidities were added to the model. Collinearity between the variables was previously analyzed (In regression analysis, collinearity of two variables means that strong correlation exists between them, making it difficult or impossible to estimate their individual regression coefficients reliably-www.statistics.com). Additionally, the McNemar test

was performed to identify the modifications of the categorical variables over time.

All analyses were performed in the SPSS program (IBM® SPSS® Statistics 24.0). An $\alpha \leq 0.05$ was used for all cases, with a one-tailed P for the log-rank test and a two-tailed P for the Wald test.

Ethical aspects

This study received ethical approval (Ethics Committee Letter No. 200-057; Dated October 6, 2020) from the Ethics Committee of Integrated Subnetwork of Health Services Central East of the Santa Clara Hospital, Bogotá, Colombia. This research followed the international ethical standards for biomedical research with human beings established in the Declaration of Helsinki,[5] and the regulations of the Ministry of Social Protection of Colombia, Resolution 8430 of 1993.[6]

3. RESULTS

In total, 62 patients with COVID-19 were analyzed. The mean age was 62.7 ± 11.1 y, with an average age of 65.4 vs. 58.5 y for patients who died versus patients who survived. At admission, the most frequent comorbidity was hypertension (38.7%). The most common symptom at admission to the ICU was respiratory distress, which was observed in 43(69.4%) of the patients (Table 1).

The laboratory abnormalities observed in this cohort were (in order of frequency) lymphopenia (62.9%), elevated lactate dehydrogenase levels (46.8%), and elevated ferritin levels (29%). Regarding the relevant findings by ventilator monitoring, we found compromised dynamic compliance (67.7%), increased resistance in the airway (51.6%), the use of a high PEEP (46.8%), severe pulmonary dysfunction according to the Berlin classification (45.2%), and compromised static compliance (38.7%). Concerning the parameters related to protective ventilation (plateau pressure, conduction pressure, and tidal volume), we found a low frequency of changes in these parameters from admission to follow-up (Table 2).

During follow-up, the significantly altered variables were lymphocyte count, tidal volume, LDH level,

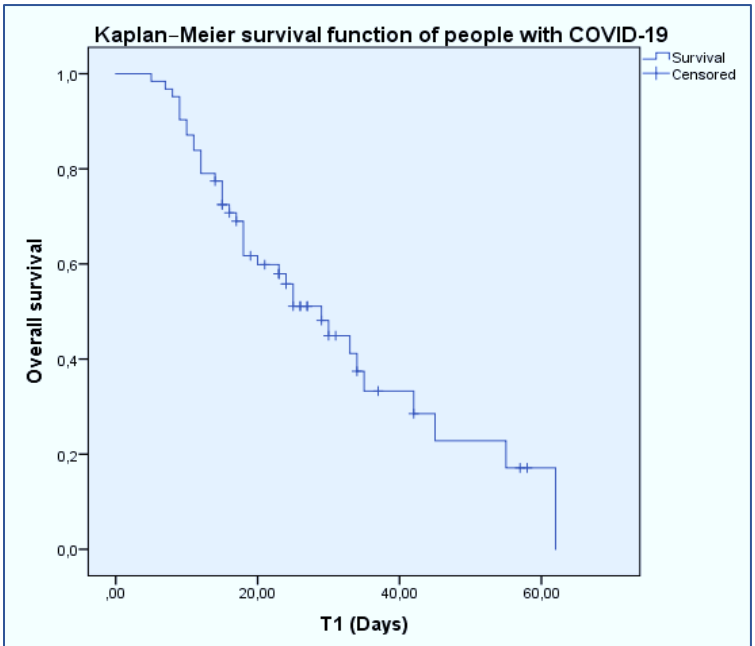

Figure 1: Overall survival curve

creatinine level, acid-base status, sodium level, potassium level, $PaCO_2$ level, and PaO_2 level (Table 2).

During the follow-up period 37(59.7%) patients died. A progressive decrease was observed in the overall survival function, reaching a survival of 50% on Day 25. Generally, the highest mortality rate occurred between Days 0 and 30 of follow-up; after that, the mortality rate decreased to zero beginning on Day 62. When evaluating the Kaplan–Meier survival function, a 99.9% probability of survival was observed on the first day, a 68% probability was observed on the tenth day, and a 78% probability was observed on the twelfth day. As of Day-30, of the course of the disease, the survival rate was approximately 69% (Figure 1).

Survival associated with risk factors

With the LogRank tests, it was possible to identify the differential survival associated with the presence or absence of selected factors, such as concurrence with decompensated heart failure at admission, elevated creatinine level, dysnatremia, acid-base disorders (diagnosed by a change in pH), increased delta CO_2 and increased lactate level. The associations were statistically significant when each factor was evaluated in the first 24 h after admission (Figure 2). By averaging the values within the first 14 days of the hospital stay, the log-rank tests identified differential survival associated with the presence or absence of selected factors, such as dysnatremia, acid-base disorders, and elevated levels of lactate (Figure 3). For the other variables, no statistically significant differences were identified (Tables 1 and 2).

Cox regression

Under the backward-type Cox regression, it can be

observed that after controlling for the effect of the other factors, the HR of increased lactate and delta CO_2 levels remained statistically significant (Table 3). Similarly, a backward-type Cox regression was carried out for the follow-up variables. It was observed that after controlling for the effect of the other factors, the HR for dysnatremia and acid-base disorders remained statistically significant (Table 3).

4. DISCUSSION

To date, numerous studies have identified risk factors associated with mortality from COVID-19; this is a relevant document because it allows us to determine the outcomes of a cohort of patients from a Level III hospital in Bogotá, Colombia, and to estimate COVID-19 survival in a critically ill and vulnerable Colombian population. Thus, this study provides an essential insight into the reality of the Latin American people.

In this cohort of patients, the mean age was 63 ± 11 y, the SOFA score was 6 ± 3.5, and the NEW2 score was 8 (IQR 7-11). Additionally, 51.6% of the patients required vasopressor support, and 48.4% required prone therapy. The median hospital stay was 38 vs. 17 days, the median ICU stay was 25 vs. 17 days, and the median mechanical ventilation duration was 9 vs. 8.5 days among survivors vs. non-survivors. These patients were treated with high ventilator parameters (a median FiO_2 of 80% (IQR 50-100) and a PEEP of 13 (IQR 10-14)) and received protective mechanical ventilation (a median tidal volume 6.9 mL/kg (IQR 6.5-7.4), a driving pressure of 10 (IQR 6-14), and a plateau pressure of 21 (IQR 17-24).

Patients severely affected by COVID-19 usually present with the first phase of illness characterized by acute viremia (fever, cough, and myalgia); the second phase is then characterized by a pulmonary injury caused mainly by severe inflammation due to a massive cytokine response, also called "cytokine storm", which can lead to elevated serum lactate levels and could be mediated by tissue hypoperfusion.[7,8] However, Giuseppe Nardi et al. found that high delta lactate levels may be an early marker of biological damage mediated by virus-related inflammation rather than low output.[9] In contrast, Thirumalaisamy P et al. found that serum lactate levels

Table 2: Variables of the cohort at admission and follow-up

Variable	First 24 h				Follow-up			
	N (%)	HR	95% CI	P	N (%)	HR	95% CI	P *
Laboratory results								
D-dimer (>1 mcg/ml)	15 (24.2)	1.22	0.3−4.4	0.754	33 (53.2)	1.51	0.6−3.3	1,000
Troponin (> 0.05 ng/dl)	27 (43.5)	1.18	0.5−2.6	0.687	30 (48.4)	1.23	0.5−2.5	1,000
Lymphocyte count (<1000 mm^3)	39 (62.9)	1.36	0.6−2.6	0.372	53 (85.5)	1.02	0.4−2.5	0.010
LDH (> 350 UI/L)	29 (46.8)	0.45	0.2−1.0	0.058	43 (69.4)	0.32	0.1−0.8	0.060
Ferritin (>1000 mcg/L)	18 (29)	1.50	0.5−3.8	0.390	26 (41.9)	0.84	0.3−1.8	1,000
Creatinine	19 (30.6)	2.30	1.1−4.5	0.014	35 (56.5)	1.30	0.6−2.6	0.001
Sodium	23 (37.1)	2.01	1.02−3.9	0.043	12 (19.4)	5.13	2.4−10.7	0.027
Potassium	16 (25.8)	1.08	0.5−2.2	0.828	5 (8.1)	1.53	0.4−5.1	0.006
Ventilatory parameters								
Tidal volume (> 6 ml/kg)	7 (11.3)	1.24	0.4−3.6	0.686	23 (37.1)	2.02	0.8−4.9	0.004
FiO_2 > 60%	37 (59.7)	1.25	0.6−2.5	0.525	26 (41.9)	1.30	0.6−2.5	0.170
PEEP > 12	29 (46.8)	1.18	0.5−2.3	0.636	28 (45.2)	1.01	0.4−2.0	1,000
Plateau pressure > 30	3.2 (2)	1.7	0.2−13.1	0.585	2 (3.2)	1.97	0.2−14.9	1,000
Driving pressure > 15	7 (11.3)	1.06	0.3−3.1	0.909	4 (6.5)	1.20	0.3−4.0	0.125
Peak Pressure >35	15 (24.2)	2.58	0.9−7.1	0.069	22 (35.5)	1.28	0.6−2.7	0.688
Dynamic compliance ml/cmH_2O < 40	42 (67.7)	1.51	0.1−11.5	0.687	3 (4.8)	1.32	0.3−5.7	−
Static compliance ml/cmH_2O < 40	24 (38.7)	1.83	0.7−4.2	0.162	15 (24.2)	1.88	0.8−4.3	−
Ventilatory Ratio > 2	4 (6.5)	1.22	0.2−5.5	0.791	2 (3.2)	1.04	0.1−7.9	0.500
PAFI <100	28 (45.2)	1.36	0.7−2.6	0.355	4 (6.5)	1.22	0.3−4.0	0.000
Gasometric parameters								
pH	32 (51.6)	3.19	1.5−6.7	0.002	22 (35.5)	12.27	5.0−29.7	0.08
PaO_2	24 (38.7)	1.03	0.5−2.0	0.914	55 (88.7)	1.15	0.4−3.0	0.000
$PaCO_2$	42 (67.7)	2.00	0.9−4.0	0.053	59 (95.2)	3.20	0.7−13.7	0.000
Bicarbonate in blood	32 (51.6)	1.17	0.5−2.4	0.660	39 (62.9)	1.41	0.6−3.0	0.238
Venous saturation	17 (27.4)	1.19	0.3−3.5	0.716	27 (43.5)	1.53	0.7−3.2	1,000
Tissue oxygen extraction	15 (24.2)	1.19	0.3−3.5	0.098	22 (35.5)	1.06	0.4−2.8	1,000
Delta CO_2	7 (11.3)	10.99	2.6−45.7	0.001	14 (22.6)	1.54	0.6−3.8	0.625
Arterial saturation	13 (21.6)	1.13	0.4−2.6	0.772	8 (12.9)	1.68	0.6−3.8	0.227
Lactate	15 (24.2)	3.75	1.8−7.8	0.000	9 (14.5)	3.32	1.4−7.5	0.039
Complementary therapies								
Prone position	30 (48.4)	1.40	0.7−2.7	0.313	−	−	−	−
Vasopressors	32 (51.6)	1.08	0.5−2.0	0.801	−	−	−	−
Images								
Chest X-ray (4 quadrant infiltrates)	35 (56.5)	1.41	0.7−2.7	0.312	12 (19.4)	1.22	0.5−2.6	0.000
Scales								
NEWS2 > 7	35 (56.5)	1.01	0.3−2.7	0.970	30 (48.4)	1.11	0.5−2.5	0.125
SOFA score > 4	29 (46.8)	1.14	0.3−3.4	0.812	36 (58.1)	1.13	0.3−8.3	1,000
SIC	7 (11.3)	2.55	0.6−9.5	0.162	9 (14.5)	1.38	0.4−3.8	1,000

* = McNemar statistic; **Abbreviations:** *ng = nanogram; FIO_2 = Inspired oxygen fraction; PAFI = index of PaO_2/FiO_2; Delta CO_2 = Venous-to-arterial carbon dioxide difference; SIC = Sepsis-induced coagulopathy; SOFA Sequential Organ Failure Assessment; NEW2 = National Early Warning Score 2; HR = Hazards Ratio*

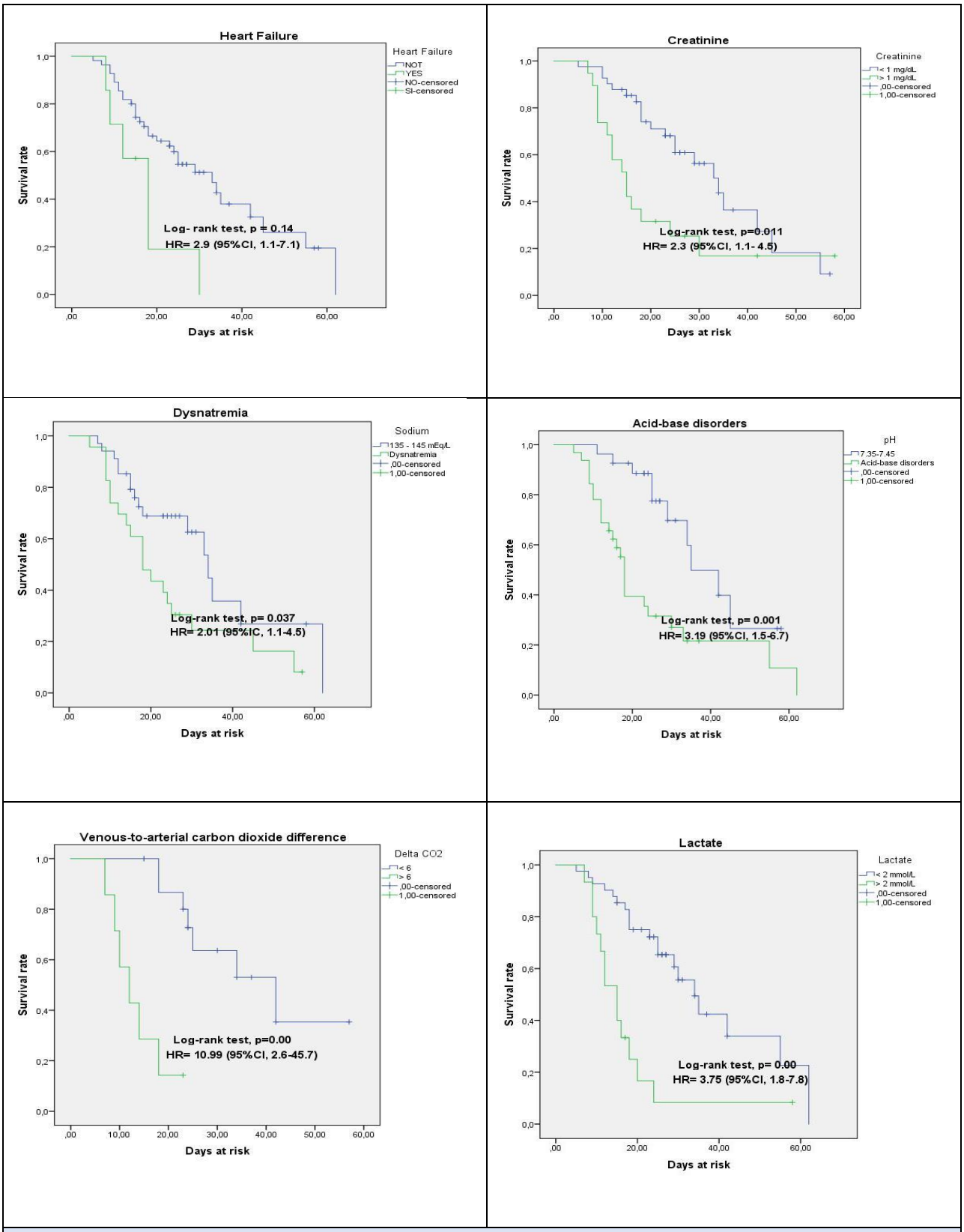

Figure 2: Survival at admission

an increase in mortality, regardless of whether it corresponds to lactic or non-lactic acidosis[13] or alkalosis (mediated by increased aldosterone levels).[14] In this

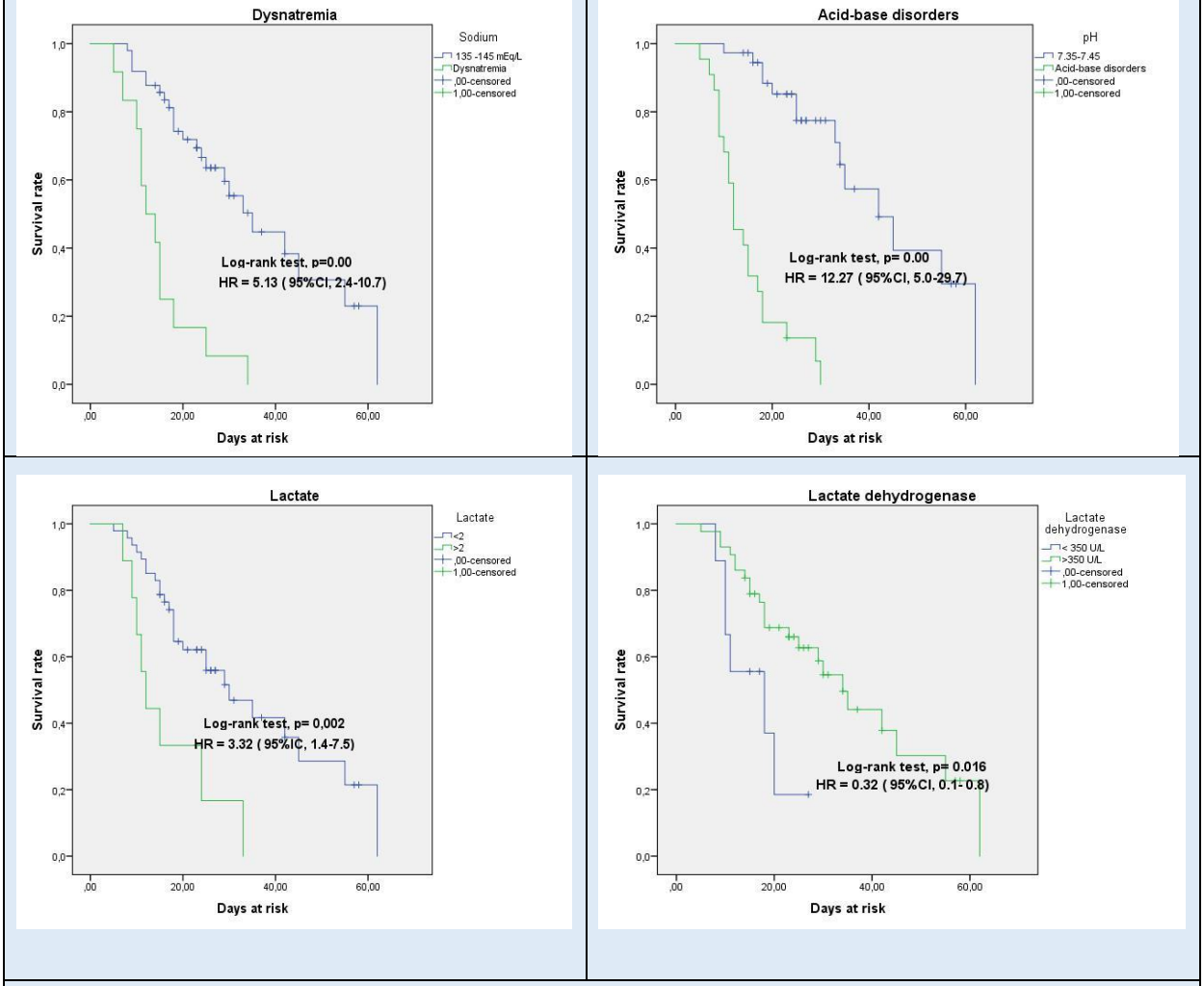

Figure 3: Survival at follow-up

can be helpful in early risk stratification,[10] and are most likely associated with tissue hypoperfusion. The data from this cohort of patients support this last theory, given that hyperlactatemia was observed (HR = 6.15, 95% CI: 1.1-34.2, P = 0.038), which was accompanied by increased delta CO_2, suggesting tissue hypoperfusion as an essential mediator of these findings (HR = 14.78, 95% CI: 2.1-100.5, P = 0.00).[11,12]

On the other hand, numerous investigations have been carried out during the pandemic on serum biomarkers associated with COVID-19-related mortality, but few studies have investigated the acid-base status. It is known that acid-base alterations are frequently observed in the ICU. These alterations have been associated with cohort, it was found that acid-base alterations (HR = 12.04, 95% CI: 4.1-35.3, P = 0.00) were associated with an increase in mortality. However, further studies with physicochemical approaches are required to characterize the disorder better.

Dysnatremia is a common disorder in critically ill patients.[15] It is often acquired in the hospital, which explains why this disorder corresponds to findings in the late stage of the disease. Multiple mechanisms have been postulated to explain these findings; for example, the inappropriate secretion of antidiuretic hormones, associated with drugs. These findings are consistent with the results from this cohort, where higher mortality was observed to be associated with sodium disorders (HR =

5.28, 95% CI: 1.8-15.3, P = 0.00); it should be noted that these appear to be relevant in later stages of the disease, and the causal mechanism is not completely clear thus far.

The obesity and BMI variables were not statistically significant either for the antecedent (taken as a dichotomous variable) or for the BMI calculation at admission. These findings differ from similar studies that showed a clear association.[16-18] Among the mechanisms proposed to explain this association is the alteration of mechanical properties, such as reduced lung compliance, reduced chest wall compliance, reduced residual functional capacity, and reduced expiratory reserve volume.[19] Another of the proposed mechanisms is chronic inflammation caused by excessive amounts of adipose tissue in people with obesity; in these patients, COVID -19 may further exacerbate inflammation, as obese patients are exposed to higher concentrations of circulating inflammatory molecules than non-obese patients.[20]

In this study, age was not associated with an increase in mortality; however, other studies have found a link between them.[1,21] For example, Sousa et al. reported a 3.6-fold mortality rate associated with this age group.[22] Zhou et al. also observed similar findings (OR = 1.10, 95% CI: 1.03–1.17; P = 0.0043).[23]

The mortality rate in this disease is variable since it could range from 20% to 60%, depending on the type of study.[21,24-28] In this cohort of patients, the mortality rate was 59.7%, suggesting that a high mortality rate is expected, given that the study included critically ill

patients who received mechanical ventilation and vasopressor support and had a high rate of multiorgan dysfunction. Myocardial dysfunction, measured by increased troponin levels, was observed in a significantly high percentage of patients at admission and follow-up (43.5% and 48.4%, respectively). The same was true for renal dysfunction (30.6% and 56.6%) and hematological dysfunction (24.2% and 53.2%). When the follow-up variables were analyzed, it was found that in this cohort of patients, kidney injury, electrolyte alterations, lactate levels, PaO_2 levels, $PaCO_2$ levels, and radiological findings significantly worsened during follow-up. The above explains why these mortality rates were similar to those reported in the literature for this type of condition.[24,29–31] On the other hand, a progressive decrease in overall survival was observed, reaching a survival of 50% on Day 28; generally, the highest mortality rate was observed between Days 0 and 35 of follow-up, with a median survival of 29 days (95% CI: 21–36). These data suggest the need for early identification and timely treatment of critical patients, and these approaches should be considered crucial for reducing the number of deaths and increasing survival.

5. LIMITATIONS

The current study has some limitations. First, in some cases, there was incomplete documentation of antecedents, symptoms, or laboratory findings in the database, even after efforts to find and collect these data. Some diagnoses of disease coexistence were based on the self-reports of patients or relatives at admission, leading to a recall bias. Second, with the limited number of patients, it is difficult to assess the risk factors associated with mortality with adjusted multivariate methods; the preceding explains the wide confidence intervals described in this study. It could be said that this is a cohort with a modest number of patients admitted to the hospital, with a collection of standardized data. Nevertheless, larger cohorts would help to define better the clinical presentation, natural history, and risk factors of such patients. More studies on patients hospitalized in ICUs are needed to obtain a complete picture of the spectrum of the clinical severity of the disease in Colombia and Latin America. Additionally, the statistical findings should be interpreted with caution, and nonsignificant P values do not necessarily rule out the absence of an association.

6. CONCLUSION

Patients admitted to a Level III hospital with a diagnosis of COVID-19 and who received mechanical ventilation had a mortality rate of 59.7%. Among the factors associated with increased mortality in the first 24 hours, parameters related to tissue hypoperfusion, such as increased lactate and CO_2 delta levels, were identified. For variables associated with mortality at 14 days, dysnatremia and acid-base disorders were identified. For the most part, these results are consistent with what have been observed by other researchers in other countries. However, studies with a larger sample size are possibly needed to confirm these findings. This study is one of the first to estimate the survival of critically ill Colombian patients diagnosed with COVID-19.

7. Data availability

The numerical data generated during this research is available with the authors.

8. Acknowledgement

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9. Conflict of interest

The study utilized the hospital resources only, and no external or industry funding was involved.

10. Authors' contribution

GO: Conceptualization, Validation, Writing - Review & Editing

MG: Writing - Review & Editing, Supervision

AL: Data acquisition, revision for important intellectual content
ELL, JB: Methodology, Formal analysis, Writing - Original Draft, Review & Editing
JM, DG, CC, JM, MM: Acquisition of data, analysis, interpretation of data for the work and revision for important intellectual content

11. REFERENCES

1. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020;395(10223):497–506. [PubMed] DOI: 10.1016/S0140-6736(20)30183-5
2. WHO. WHO Coronavirus (COVID-19) Dashboard; 2021 [cited 2021 Dec 1]. Available from: https://covid19.who.int/
3. Wu Z, McGoogan JM. Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72 314 Cases From the Chinese Center for Disease Control and Prevention. JAMA. 2020 Apr 7;323(13):1239-1242. [PubMed] DOI: 10.1001/jama.2020.2648
4. Oliveira E, Parikh A, Lopez-Ruiz A, Carrilo M, Goldberg J, Cearras M, et al. ICU outcomes and survival in patients with severe COVID-19 in the largest health care system in central Florida. PLoS One. 2021 Mar 25;16(3):e0249038. [PubMed] DOI: 10.1371/journal.pone.0249038
5. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013 Nov 27;310(20):2191-4. [PubMed] DOI: 10.1001/jama.2013.281053
6. De Salud M. Resolucion numero 8430 DE 1993. 1993.
7. Tisoncik JR, Korth MJ, Simmons CP, Farrar J, Martin TR, Katze MG. Into the eye of the cytokine storm. Microbiol Mol Biol Rev. 2012 Mar;76(1):16-32. [PubMed] DOI: 10.1128/MMBR.05015-11
8. Tang X, Du RH, Wang R, Cao TZ, Guan LL, Yang CQ, et al. Comparison of Hospitalized Patients with ARDS Caused by COVID-19 and H1N1. Chest. 2020 Jul;158(1):195-205. [PubMed] DOI: 10.1016/j.chest.2020.03.032
9. Nardi G, Sanson G, Tassinari L, Guiotto G, Potalivo A, Montomoli J, et al. Lactate Arterial-Central Venous Gradient among COVID-19 Patients in ICU: A Potential Tool in the Clinical Practice. Crit Care Res Pract. 2020;2020:4743904. [PubMed] DOI: 10.1155/2020/4743904
10. Velavan TP, Kieu Linh LT, Kreidenweiss A, Gabor J, Krishna S, Kremsner PG. Longitudinal Monitoring of Lactate in Hospitalized and Ambulatory COVID-19 Patients. Am J Trop Med Hyg. 2021 Jan 11;104(3):1041-1044. [PubMed] DOI: 10.4269/ajtmh.20-1282
11. Shoar S, Hosseini F, Naderan M, Mehta JL. Meta-analysis of Cardiovascular Events and Related Biomarkers Comparing Survivors Versus Non-survivors in Patients With COVID-19. Am J Cardiol. 2020 Nov 15;135:50-61. [PubMed] DOI: 10.1016/j.amjcard.2020.08.044
12. Lamia B, Monnet X, Teboul JL. Meaning of arterio-venous PCO2 difference in circulatory shock. Minerva Anestesiol. 2006 Jun;72(6):597-604. [PubMed]
13. Gunnerson KJ, Saul M, He S, Kellum JA. Lactate versus non-lactate metabolic acidosis: a retrospective outcome evaluation of critically ill patients. Crit Care. 2006 Feb;10(1):R22. [PubMed] DOI: 10.1186/cc3987
14. Bezuidenhout MC, Wiese OJ, Moodley D, Maasdorp E, Davids MR, Koegelenberg CF, et al. Correlating arterial blood gas, acid-base and blood pressure abnormalities with outcomes in COVID-19 intensive care patients. Ann Clin Biochem. 2021 Mar;58(2):95-101. [PubMed] DOI: 10.1177/0004563220972539
15. Pokaharel M, Block CA. Dysnatremia in the ICU. Curr Opin Crit Care. 2011 Dec;17(6):581-93. [PubMed] DOI: 10.1097/MCC.0b013e32834cd388
16. Caussy C, Wallet F, Laville M, Disse E. Obesity is Associated with Severe Forms of COVID-19. Obesity (Silver Spring). 2020 Jul;28(7):1175. [PubMed] DOI: 10.1002/oby.22842
17. Simonnet A, Chetboun M, Poissy J, Raverdy V, Noulette J, Duhamel A, et al. High Prevalence of Obesity in Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) Requiring Invasive Mechanical Ventilation. Obesity (Silver Spring). 2020 Jul;28(7):1195-1199. [PubMed] DOI: 10.1002/oby.22831
18. Lighter J, Phillips M, Hochman S, Sterling S, Johnson D, Francois F, et al. Obesity in Patients Younger Than 60 Years Is a Risk Factor for COVID-19 Hospital Admission. Clin Infect Dis. 2020 Jul 28;71(15):896-897. [PubMed] DOI: 10.1093/cid/ciaa415
19. Dixon AE, Peters U. The effect of obesity on lung function. Expert Rev Respir Med. 2018 Sep;12(9):755-767. [PubMed] DOI: 10.1080/17476348.2018.1506331
20. Muscogiuri G, Pugliese G, Barrea L, Savastano S, Colao A. Commentary: Obesity: The "Achilles heel" for COVID-19? Metabolism. 2020 Jul;108:154251. [PubMed] DOI: 10.1016/j.metabol.2020.154251
21. Fu L, Wang B, Yuan T, Chen X, Ao Y, Fitzpatrick T, et al. Clinical characteristics of coronavirus disease 2019 (COVID-19) in China: A systematic review and meta-analysis. J Infect. 2020 Jun;80(6):656-665. [PubMed] DOI: 10.1016/j.jinf.2020.03.041
22. Sousa GJB, Garces TS, Cestari VRF, Florêncio RS, Moreira TMM, Pereira MLD. Mortality and survival of COVID-19. Epidemiol Infect. 2020 Jun 25;148:e123. [PubMed] DOI: 10.1017/S0950268820001405
23. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet. 2020 Mar 28;395(10229):1054-1062. [PubMed] DOI: 10.1016/S0140-6736(20)30566-3
24. Dan S, Pant M, Upadhyay SK. The Case Fatality Rate in COVID-19 Patients With Cardiovascular Disease: Global Health Challenge and Paradigm in the Current Pandemic. Curr Pharmacol Rep. 2020;6(6):315-324. [PubMed] DOI: 10.1007/s40495-020-00239-0
25. Petrilli CM, Jones SA, Yang J, Rajagopalan H, O'Donnell L, Chernyak Y, et al. Factors associated with hospitalization and

critical illness among 4,103 patients with Covid-19 disease in New York City. medRxiv. 2020;2020.04.08.20057794. DOI: 10.1101/2020.04.08.20057794

26. Hu Y, Sun J, Dai Z, Deng H, Li X, Huang Q, et al Prevalence and severity of corona virus disease 2019 (COVID-19): A systematic review and meta-analysis. J Clin Virol. 2020 Jun;127:104371. [PubMed] DOI: 10.1016/j.jcv.2020.104371

27. Rossi PG, Ferroni E, Alegiani SS, Pitter G, Leoni O, Cereda D, et al. Survival of hospitalized COVID-19 patients in Northern Italy: A population-based cohort study by the ITA-COVID19 Network. medRxiv. 2020;2020.05.15.20103119. DOI: 10.1101/2020.05.15.20103119

28. Wang K, Zhang Z, Yu M, Tao Y, Xie M. 15-day mortality and associated risk factors for hospitalized patients with COVID-19 in Wuhan, China: an ambispective observational cohort study. Intensive Care Med. 2020 Jul;46(7):1472-1474. [PubMed] DOI: 10.1007/s00134-020-06047-w

29. Yang X, Yu Y, Xu J, Shu H, Xia J, Liu H, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. Lancet Respir Med. 2020 May;8(5):475-481. [PubMed] DOI: 10.1016/S2213-2600(20)30079-5

30. Bhatraju PK, Ghassemieh BJ, Nichols M, Kim R, Jerome KR, Nalla AK, et al. Covid-19 in Critically Ill Patients in the Seattle Region - Case Series. N Engl J Med. 2020 May 21;382(21):2012-2022. [PubMed] DOI: 10.1056/NEJMoa2004500

31. Arentz M, Yim E, Klaff L, Lokhandwala S, Riedo FX, Chong M, et al. Characteristics and Outcomes of 21 Critically Ill Patients With COVID-19 in Washington State. JAMA. 2020 Apr 28;323(16):1612-1614. [PubMed] DOI: 10.1001/jama.2020.4326

