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Research Article

# RENAL FUNCTION ALTERATIONS IN PATIENTS WITH SARS COV2 INFECTION IN A MEXICAN POPULATION

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**ABSTRACT**

In the first days of 2020, the pathogen of a new disease, SARS-CoV-2, was isolated by August 7, 2020, there were 18,902,735 cases worldwide, while in Mexico there were 469,407 cases reported.
An observational, descriptive, cross-sectional and retrospective study carried out at the Dr. Enrique Cabrera General Hospital in Mexico City from April 1 to June 10, 2020, where patients with a confirmed infection by RT-PCR users of the service of emergencies that required hospitalization, paying specific attention to altered renal function.In our analysis we found that the mean age of the infected population was 52.53 years, being those of the oldest age who presented higher mortality, within the paraclinical tests performed Po2, lactate, leukocytes, lactic dehydrogenase, and C-reactive protein were parameters which were found to be elevated and associated with higher mortality. The values obtained for the glomerular filtration rate in patients, in general, were 85.84 ml/min / 1.73m2 by MDRD and 82.12 by CKD Epi, finding the greatest decrease in this in hypertensive patients with decreased GFR of 79.38 ml/min/1.73m2 per MDRD and 75.94 ml/min / 1.73m2 the findings observed in our population and the high differences in GFR in patients with COVID 19 are a variable highly associated with mortality after hospital admission, so it is necessary to pay more attention to this to modify its course.



## INTRODUCTION

At the end of December 2019, in one of the most populated provinces in China; Hubei, Wuhan; an outbreak of pneumonia cases of unknown origin was identified. This pneumonia shared some clinical characteristics similar to others of viral etiology since among infected patients it was found that all had visited or worked in a local Food market and exotic animals during the month before presenting symptoms (1-3).

On December 31 of this year, the Chinese health authorities notified the World Health Organization (WHO) about the presence of said outbreak, initiating health measures between countries bordering China such as South Korea, Macao, and Taiwan (1, 4).

In the first days of 2020, the causative pathogen of this new disease was isolated for the first time, and it was named 2019-novel coronavirus (2019-nCoV) (5,6). Subsequently, the complete sequencing of this new virus belonging to the Coronaviridae family was achieved, and it was found that it shared 79.5% of the genetic sequence with the Coronavirus causing Severe Acute Respiratory Syndrome (SARS CoV) (7), which is why it is renamed to such an agent as SARS CoV-2 (8).
As of August 7, 2020, 18,902,735 cases were recorded worldwide, of which 709,511 had died, while in Mexico there

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were 469,407 reported cases, with 51,311 deaths associated or confirmed by SARS CoV2.

Here we present the analysis of a Mexican population hospitalized in a second-level care reconversion center, paying specific attention to the clinical effects on kidney function in patients with COVID 19.

## METHODOLOGY

It is an observational, descriptive, cross-sectional, retrospective study. It was carried out at the Dr. Enrique Cabrera General Hospital in Mexico City, from April 1 to June 10, 2020, with prior authorization from the ethics and research committee of the Secretary of Health of Mexico City.

The inclusion criteria were patients with a confirmed infection by RT-PCR, users of the emergency services of the General Hospital Dr. Enrique Cabrera, and who required hospitalization. Clinical and epidemiological data were obtained from the medical records of the patients.

### *Statistic Analysis*

Descriptive statistical analysis of the different variables was carried out, categorical variables were reported as frequencies and proportions, while continuous variables were described using mean values, standard deviation, and interquartile range. Differences between the groups studied were evaluated by Student's T, Chi-square, Fisher's Exact, and Mann-Whitney U analysis, as necessary.

Statistical analysis was performed using the SPSS v.20 statistical package.

## RESULTS

A study was carried out at the Dr. Enrique Cabrera General Hospital in Mexico City, the analyzed population was divided into groups according to the most observed comorbidities (Systemic Arterial Hypertension, Type 2 Diabetes, Obesity) and in those who did not present any comorbidity. The analysis was performed between the recovered patients and those who died during their hospital stay.

In our analysis we found that the mean of the population infected by SARS CoV 2 was 52.53 years, finding important differences in the age of presentation of those with Systemic Arterial Hypertension with an average of 57.97 years, while the group with a more It was young in those who did not have any comorbidity at hospital admission (45.77 years). In the 4 groups, differences were observed between the recovered group versus the deceased, the deceased being those with older ages.

Various paraclinical analyzes were performed upon admission; arterial blood gas, hematic cytometry, blood chemistry, liver and kidney function tests, acute phase reactants; finding the following alterations, while the PO2 the average was 52.53 mmHg in general, while the patients with arterial hypertension presented the lowest levels with 52.67mmHg. In the obesity and arterial hypertension group, differences were found between the group of recovered patients versus those who died, the PO2 being lower in these, while in those with type 2 diabetes and without comorbidities it was found that those who died had higher PO2 levels than in those who recovered.

Lactate in the general population was found to be elevated with 2,199 mmol / L, with the group with the highest elevation being the hypertensive (2,273 mmol / L), while those who did not have any comorbidity were lower than this value with 1.85 However, important differences in lactate value are found in all groups between those recovered concerning those who died during their hospital stay in all groups.

Leukocytosis was found in the general population (10.62 x103 x mm3), with this higher value being found in those who had a previous diagnosis of arterial hypertension. Although those patients without comorbidities presented less marked elevation, in all other groups upward differences were found between deceased patients than in those who recovered. An example of this is presented in the differential of the hematic cytometry, where neutrophilia was found with 9,066 x103 x mm3; while this was higher in those patients with arterial hypertension than in the other groups, the group with the lowest elevation of neutrophils was that of patients without added comorbidities. It is important to point out that in all the group's differences were found between the recovered and deceased groups, likewise in the lymphocyte count it was found that all the groups had lymphopenia with an average value of 0.944 x103 x mm3, and this was more marked in those patients with arterial hypertension with 0.9054 x103 x mm3, and there are also significant differences in the lymphocyte populations in those patients who died than in those recovered.

In the blood chemistry, we found an elevation of lactic dehydrogenase in general with 552.9 mg / dL, which was higher in those with type 2 diabetes with 583.7 mg / dL, meanwhile, the group without comorbidities presented an average value of 471.7 mg / dL, finding significant differences between those who died and those who did not, even with differences greater than 300 mg / dL (Diabetic and hypertensive). In the other blood chemistry values, no important alterations were found for this analysis. Regarding the acute phase reactants, we found a significant elevation of C-reactive protein, with a mean value of 21.46 mg / dL, having the most notable elevation in the group of diabetic patients with 22.08 mg/dl on average, while those who did not have any comorbidity had the lowest mean values (20.39 mg / dL), however, in all groups higher C-reactive protein values were found in those who died. D-dimer values were measured, finding an average elevation of 1454 ug / dL, with the highest elevation found in hypertensive patients (1645ug / dL), and the lowest in patients who did not have any comorbidity (1224ug / dL). As for fibrinogen, a mean value of 913.7 mg / dL was found, with the group of hypertensive patients (932 mg/dL) having the highest mean value, while the group of obese patients was the one with the lowest mean value (897.6 mg/dL).

Azoados and creatinine were measured, finding the following results: Concerning urea, an average of 51.78 mg / dL was found, with a more marked elevation in patients with type 2 diabetes (59.46 mg/dL), certainly the average value It was lower in those patients who did not have any comorbidity, however in this variable, a notable increase was found between deceased patients and those who achieved discharge due to improvement. Blood urea nitrogen, an average value of 28.9 mg/dL was found, and like the previous one, this was found with a higher elevation in those patients with diabetes (37.03 mg / dL), while the average creatinine values were 1.18 mg/dL for the general population, each group presented mean values higher than the normal range, for example, diabetics 1,326 mg / dL, hypertensive patients 1.22 mg / dL, and obese 1,146 mg /

dL. In all groups there were significant differences in those patients who discharged versus those who died (type 2 diabetes: 1,218 mg/dL vs 1,477 mg/dL; Systemic arterial hypertension: 1,067 mg/dL vs 1.42 mg/dL; Obesity: 1,051 mg/dL vs 1,302 mg/dL; No comorbidities 1,013mg/dL vs 1,221mg/dL).

With the serum creatinine value and the clinical characteristics of the patients, the glomerular filtration rate was calculated using the Modification of Diet in Renal Disease Study (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD Epi) formulas. The values obtained for the glomerular filtration rate by MDRD for the general population were 85.84 ml/min / 1.73m2, while CKD Epi the average was 82.12. Analyzing both formulas in the 4 groups and the general average as well as those recovered and the deceased, it is distinguished that for the group of diabetic patients the average, in general, was 85.37 ml/min / 1.73m2 for MDRD while for CKD Epi the value The mean was 79.72 ml/min / 1.73m2, finding that between the recovered and deceased groups there was a difference of 18.97 ml/min / 1.73m2 and 16.57 ml/min / 1.73m2 for MDRD and CKD Epi respectively.

In the group of hypertensive patients, the lowest glomerular filtration rate value was found, with 79.38 ml/min / 1.73m2 for MDRD and 75.94 ml/min / 1.73m2 for CKD Epi. Likewise, in this group of patients, there is a significant decrease in the glomerular filtration rate between those recovered and those who died, with differences of 19.32 ml/min / 1.73m2 and 16.68 ml/min / 1.73m2 for MDRD and CKD Epi respectively. In the group of obese patients, the mean value of the glomerular filtration rate was 87.58 ml/min / 1.73m2 by MDRD and 83.82 ml/min / 1.73m2 by CKD Epi, in the analysis between deceased and recovered patients there is an also decreased glomerular filtration rate between these groups, with a difference of 17.96 ml/min / 1.73m2 for MDRD and 15.24 ml/min / 1.73m2 for CKD Epi. In the group of patients without comorbidities, although we found the most conserved glomerular filtration rate with 91.31 ml/min / 1.73m2 by MDRD and 90.43 by CKD Epi, it is in these patients that the most marked decrease is observed among the recovered patients and those who died, finding that the values for recovered patients were 97.86 ml/min / 1.73m2 for MDRD and 90.43 ml/min / 1.73m2 for CKD Epi, while glomerular filtration rates of 76.48 ml were found for deceased patients /min/1.73m2 and 75.64 ml/min / 1.73m2 by MDRD and CKD Epi respectively, with differences of more than 20 ml/min / 1.73m2 between both groups. All this results see in the table 1.

**Table 1** Bioquimical characteristics of patins hospitlized.

| Variable | | Total Population | | Type 2 Diabetes | | | | Hypertension | | | | Obesity | | | | Without Comorbidities | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | General | p | General | Recovered | Dead | p | General | Recovered | Dead | p | General | Recovered | Dead | p | General | Recovered | Dead | p |
| **Age** | | 52.53 | 0.009 | 53 | 51.75 | 54.74 | 0.269 | 57.97 | 57.09 | 59.14 | 0.407 | 51.67 | 50.7 | 53.23 | 0.155 | 45.77 | 42.6 | 52.95 | 0.011 |
| **PO2 (mmHg)** | Mean | 54.18 | 0.829 | 54.77 | 53.2 | 56.95 | 0.394 | 52.67 | 53.79 | 51.18 | 0.56 | 53.43 | 54.05 | 52.43 | 0.6 | 58.26 | 56.23 | 62.84 | 0.393 |
| | S.D | 22.74 | | 21.91 | 20.24 | 24.13 | | 22.24 | 20.75 | 24.23 | | 21.73 | 19.27 | 25.34 | | 27.8 | 20.29 | 40.34 | |
| | Minimum | 14 | | 14 | 14 | 24 | | 17 | 18 | 17 | | 16 | 16 | 17 | | 17 | 17 | 25 | |
| | Maximum | 158 | | 125 | 116 | 125 | | 131 | 131 | 125 | | 148 | 116 | 148 | | 158 | 109 | 158 | |
| **Lactate (mmol/L)** | Mean | 2.199 | < 0.001 | 2.252 | 1.792 | 2.895 | 0.004 | 2.273 | 1.975 | 2.666 | 0.036 | 2.24 | 1.951 | 2.716 | <0.001 | 1.852 | 1.547 | 2.542 | <0.001 |
| | S.D | 1.629 | | 1.919 | 0.7305 | 2.733 | | 1.658 | 1.184 | 2.078 | | 1.608 | 1.342 | 1.885 | | 0.9993 | 0.6235 | 1.323 | |
| | Minimum | 0.5 | | 0.6 | 0.6 | 0.8 | | 0.6 | 0.6 | 0.8 | | 0.5 | 0.5 | 0.8 | | 0.8 | 0.8 | 1 | |
| | Maximum | 16 | | 16 | 3.7 | 16 | | 10.4 | 8.3 | 10.4 | | 11.7 | 11.7 | 10.4 | | 6.1 | 4 | 6.1 | |
| **White Blood Cells (10^3 / mm^3)** | Mean | 10.62 | < 0.001 | 10.49 | 9.17 | 12.33 | 0.003 | 10.98 | 9.405 | 13.06 | 0.002 | 10.79 | 9.583 | 12.77 | <0.001 | 9.608 | 8.772 | 11.5 | 0.034 |
| | S.D | 5.284 | | 5.442 | 3.395 | 7.055 | | 5.875 | 4.224 | 7.045 | | 5.067 | 4.168 | 5.763 | | 4.691 | 3.978 | 5.675 | |
| | Minimum | 1.8 | | 3.8 | 3.8 | 4.2 | | 3.2 | 3.2 | 4.2 | | 3.7 | 3.7 | 3.7 | | 1.8 | 2 | 1.8 | |
| | Maximum | 35 | | 35 | 18.8 | 35 | | 35 | 24.4 | 35 | | 35 | 26.8 | 35 | | 22.7 | 17.7 | 22.7 | |
| **Neutrophils (10^3 / mm^3)** | Mean | 9.066 | < 0.001 | 8.874 | 7.561 | 10.7 | 0.002 | 9.408 | 7.787 | 11.55 | <0.001 | 9.266 | 8.05 | 11.25 | <0.001 | 8.137 | 7.026 | 10.65 | 0.003 |
| | S.D | 5.132 | | 5.212 | 3.329 | 6.671 | | 5.757 | 4.244 | 6.763 | | 4.942 | 4.202 | 5.422 | | 4.515 | 3.891 | 4.911 | |
| | Minimum | 0.1 | | 2.6 | 2.6 | 3.3 | | 1.5 | 1.5 | 3.3 | | 0.1 | 0.1 | 2.9 | | 1.1 | 1.5 | 1.1 | |
| | Maximum | 33.1 | | 33.1 | 17.2 | 33.1 | | 33.1 | 22.2 | 33.1 | | 33.1 | 26.1 | 33.1 | | 21.5 | 16.4 | 21.5 | |
| **Lymphocytes (10^3 / mm^3)** | Mean | 0.944 | 0.674 | 0.9343 | 0.9138 | 0.9628 | 0.617 | 0.9054 | 0.9147 | 0.8932 | 0.847 | 0.9664 | 0.9496 | 0.9938 | 0.661 | 0.9465 | 1.013 | 0.7947 | 0.129 |
| | S.D | 0.6116 | | 0.4861 | 0.3962 | 0.5932 | | 0.5523 | 0.5437 | 0.5695 | | 0.7118 | 0.5244 | 0.944 | | 0.5211 | 0.5229 | 0.4972 | |
| | Minimum | 0 | | 0.2 | 0.2 | 0.3 | | 0.3 | 0.3 | 0.3 | | 0.1 | 0.1 | 0.3 | | 0 | 0 | 0.2 | |
| | Maximum | 7.6 | | 3.4 | 1.9 | 3.4 | | 3.5 | 3.5 | 3.4 | | 7.6 | 3.5 | 7.6 | | 2.5 | 2.5 | 2 | |
| **Creatinine (mg/dL)** | Mean | 1.181 | 0.004 | 1.326 | 1.218 | 1.477 | 0.298 | 1.22 | 1.067 | 1.42 | 0.049 | 1.146 | 1.051 | 1.302 | 0.057 | 0.99 | 0.8909 | 1.221 | 0.002 |
| | S.D | 0.9563 | | 1.237 | 1.335 | 1.082 | | 0.89 | 0.7848 | 1.002 | | 0.9375 | 0.962 | 0.8798 | | 0.3953 | 0.2855 | 0.5094 | |
| | Minimum | 0.4 | | 0.4 | 0.4 | 0.5 | | 0.5 | 0.5 | 0.6 | | 0.4 | 0.4 | 0.5 | | 0.6 | 0.6 | 0.6 | |
| | Maximum | 9.2 | | 9.2 | 9.2 | 5.1 | | 5.9 | 5.9 | 5.1 | | 9.2 | 9.2 | 5.1 | | 2.3 | 2.2 | 2.3 | |
| **Urea (mg/dL)** | Mean | 51.78 | < 0.001 | 59.46 | 50.93 | 71.35 | 0.086 | 54.8 | 45.24 | 67.42 | 0.008 | 48.91 | 42.97 | 58.59 | 0.012 | 43.94 | 33.44 | 67.69 | <0.001 |
| | S.D | 46.11 | | 59.6 | 62.1 | 54.42 | | 42.11 | 25.92 | 54.67 | | 43.98 | 44.18 | 42.15 | | 29.84 | 11.45 | 43.05 | |
| | Minimum | 11.9 | | 11.9 | 11.9 | 17.2 | | 16.2 | 16.2 | 16.4 | | 11.9 | 11.9 | 16.4 | | 16.8 | 16.87 | 16.9 | |
| | Maximum | 484.7 | | 984.7 | 484.7 | 260.5 | | 260.5 | 160.4 | 260.5 | | 484.7 | 484.7 | 260.5 | | 150.5 | 72.8 | 150.5 | |
| **Blood Ureic Nitrogen (mg/dL)** | Mean | 28.9 | 0.969 | 37.03 | 38.31 | 35.24 | 0.862 | 26.56 | 22.31 | 32.15 | 0.011 | 28.38 | 28.72 | 27.84 | 0.928 | 21 | 16.3 | 31.64 | <0.001 |
| | S.D | 61.63 | | 87.76 | 113 | 27.86 | | 19.42 | 12 | 25.27 | | 67.74 | 84.81 | 19.5 | | 13.77 | 5.302 | 20.12 | |
| | Minimum | 5.58 | | 5.58 | 5.58 | 8.06 | | 6.86 | 7.56 | 6.86 | | 5.58 | 5.58 | 6.86 | | 7.88 | 8.87 | 7.88 | |
| | Maximum | 963 | | 870 | 870 | 121.7 | | 121.7 | 74.96 | 121.7 | | 963 | 963 | 121.7 | | 70.34 | 34.03 | 70.34 | |
| **Albumine (g/dL)** | Mean | 3.267 | 0.002 | 3.166 | 3.314 | 2.967 | 0.002 | 3.24 | 3.309 | 3.152 | 0.121 | 3.299 | 3.365 | 3.192 | 0.029 | 3.37 | 3.442 | 3.205 | 0.181 |
| | S.D | 0.5424 | | 0.5539 | 0.563 | 0.4794 | | 0.5013 | 0.5292 | 0.4542 | | 0.5191 | 0.4948 | 0.5423 | | 0.6398 | 0.6921 | 0.4778 | |
| | Minimum | 0.7 | | 1.9 | 2.1 | 1.9 | | 1.9 | 2.1 | 1.9 | | 1.9 | 2.1 | 1.9 | | 0.7 | 0.7 | 2.3 | |
| | Maximum | 4.96 | | 4.9 | 4.9 | 3.8 | | 4.9 | 4.9 | 4 | | 4.9 | 4.9 | 4.6 | | 4.96 | 4.96 | 4 | |
| **Lactic dehydrogenase (U/L)** | Mean | 552.9 | 0.009 | 583.7 | 430.5 | 797.5 | 0.055 | 559.2 | 521.1 | 609.3 | 0.332 | 558.5 | 458.1 | 722.1 | 0.006 | 471.7 | 449.4 | 522.3 | 0.227 |
| | S.D | 676.5 | | 960 | 171.5 | 1455 | | 452.4 | 575.3 | 193.7 | | 690.6 | 219.4 | 1068 | | 217.4 | 200.9 | 249.3 | |
| | Minimum | 56 | | 139.9 | 139.9 | 309.5 | | 139.9 | 139.9 | 313.2 | | 56 | 56 | 258.5 | | 59.92 | 162.1 | 59.92 | |
| | Maximum | 1.003 | | 1.003 | 1039 | 1.003 | | 4570 | 4570 | 955.4 | | 1.003 | 1369 | 1.003 | | 1030 | 973.2 | 1030 | |
| **Procalcitonine (ng/mL)** | Mean | 0.9354 | 0.057 | 1 | 0.975 | 1.035 | 0.873 | 0.9804 | 0.9397 | 1.034 | 0.802 | 0.938 | 0.7992 | 1.074 | 0.258 | 0.8629 | 0.5349 | 1.605 | 0.021 |
| | S.D | 1.775 | | 1.863 | 1.75 | 2.031 | | 1.867 | 1.77 | 2.007 | | 1.717 | 1.449 | 2.08 | | 1.704 | 0.2287 | 2.979 | |
| | Minimum | 0.5 | | 0.5 | 0.5 | 0.5 | | 0.5 | 0.5 | 0.5 | | 0.5 | 0.5 | 0.5 | | 0.5 | 0.5 | 0.5 | |
| | Maximum | 10 | | 10 | 10 | 10 | | 10 | 10 | 10 | | 10 | 10 | 10 | | 10 | 2 | 10 | |
| **C-Reactive Protein (mg/dL)** | Mean | 21.46 | < 0.001 | 22.08 | 21.91 | 22.32 | 0.816 | 21.33 | 18.61 | 24.9 | 0.004 | 21.53 | 19.82 | 24.32 | 0.001 | 20.39 | 16.65 | 28.84 | <0.001 |
| | S.D | 10.05 | | 8.796 | 8.142 | 9.73 | | 11.13 | 8.638 | 13.01 | | 9.832 | 9.478 | 9.82 | | 10.96 | 8.867 | 10.7 | |
| | Minimum | 0.5 | | 4.57 | 4.57 | 5.18 | | 1.87 | 1.87 | 8.29 | | 0.5 | 0.5 | 2.7 | | 1.18 | 1.18 | 12.23 | |
| | Maximum | 84.4 | | 47.54 | 43.22 | 47.54 | | 84.4 | 43.22 | 84.4 | | 53.6 | 43.22 | 53.6 | | 48.45 | 44.4 | 48.45 | |
| **D-Dimer (µg/L)** | Mean | 1454 | 0.012 | 1491 | 1311 | 1741 | 0.123 | 1645 | 1509 | 1823 | 0.276 | 1413 | 1194 | 1769 | 0.002 | 1224 | 1214 | 246 | 0.92 |
| | S.D | 1336 | | 1392 | 1341 | 1438 | | 1433 | 1456 | 1397 | | 1314 | 1189 | 1432 | | 1123 | 1217 | 903.6 | |
| | Minimum | 0.5 | | 0.5 | 0.5 | 179 | | 100 | 100 | 314 | | 0.5 | 0.5 | 137 | | 100 | 100 | 294 | |
| | Maximum | 5000 | | 5000 | 5000 | 5000 | | 5000 | 5000 | 5000 | | 5000 | 5000 | 5000 | | 5000 | 5000 | 4500 | |
| **Fibrinogen (mg/dL)** | Mean | 913.7 | 0.089 | 931.2 | 950.6 | 904.3 | 0.54 | 932 | 899.1 | 975.5 | 0.302 | 897.6 | 371.5 | 347.9 | 0.463 | 909.4 | 833.9 | 1080 | 0.065 |
| | S.D | 383.4 | | 375.2 | 389.9 | 356.3 | | 368.1 | 392.3 | 333 | | 362.3 | 371.5 | 347.9 | | 485.9 | 440.6 | 550.2 | |
| | Minimum | 1.19 | | 222 | 222 | 222 | | 108 | 108 | 432 | | 108 | 108 | 222 | | 1.19 | 1.19 | 193.4 | |
| | Maximum | 1972 | | 1811 | 1811 | 1560 | | 1738 | 1738 | 1611 | | 1972 | 1972 | 1890 | | 1892 | 1868 | 1892 | |
| **Glomerular Filtration** | Mean | 85.84 | < 0.001 | 85.37 | 93.29 | 74.32 | 0.033 | 79.38 | 87.71 | 68.39 | 0.005 | 87.58 | 94.41 | 76.45 | <0.001 | 91.31 | 97.86 | 76.48 | 0.007 |
| | S.D | 37.82 | | 44.69 | 44.69 | 42.77 | | 35.19 | 33.8 | 34.3 | | 37.44 | 36.78 | 36.01 | | 29.36 | 25.43 | 32.83 | |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Rate by MDRD Glomerular Filtration Rate by CKD Epi** | Minimum | 6 | < 0.001 | 6 | 6 | 12.1 | 0.019 | 10.2 | 10.2 | 12.1 | 0.005 | 6 | 6 | 12.1 | <0.001 | 29.7 | 37.1 | 29.7 | 0.002 |
| | Maximum | 216.3 | | 216.3 | 216.3 | 170.8 | | 171.4 | 171.4 | 155.6 | | 216.3 | 216.3 | 167 | | 154.2 | 154.2 | 149.9 | |
| | Mean | 82.12 | | 79.72 | 86.64 | 70.07 | | 75.94 | 83.14 | 66.46 | | 83.82 | 89.61 | 74.37 | | 90.43 | 96.97 | 75.64 | |
| | S.D | 30.96 | | 35.66 | 33.73 | 36.42 | | 30.02 | 27.01 | 31.44 | | 29.91 | 27.35 | 31.62 | | 25.41 | 19.54 | 31.06 | |
| | Minimum | 5.151 | | 5.151 | 5.151 | 10.96 | | 9.065 | 9.065 | 10.96 | | 5.151 | 5.151 | 10.96 | | 27.15 | 38.48 | 27.15 | |
| | Maximum | 150 | | 150 | 150 | 141.4 | | 132.4 | 132.4 | 122.6 | | 150 | 150 | 134.3 | | 134.3 | 131.5 | 134.3 | |

30.4% of the population required mechanical ventilation at some point during their hospital stay, the group of patients that most required ventilatory support with invasive mechanical ventilation was obese patients, followed by hypertensive patients.

Regarding mortality, 39% of our patients had a fatal outcome, being higher in males than in females. 43.1% of the patients with arterial hypertension died, this being the group that presented the highest mortality in our analysis.

## DISCUSSION

The pandemic caused by SARS CoV2 has severely hit most of the nations of the world, with a high mortality rate mainly among the elderly and in those with various comorbidities such as Diabetes, Arterial Hypertension, Heart, Pulmonary and Kidney Diseases. In the analyzed population, we found that the main comorbidities found were: Arterial Hypertension, Diabetes and Obesity, associating the coexistence of these comorbidities with admission to intensive care units and worse prognoses (9, 10). Various series have reported that the most affected population is those older than 60 years (11), besides, it has been reported in the literature that advanced ages worsen the clinical outcome and are even related to an increase in mortality in these groups (11) However, in our population we find that the average age is 52 years, which some authors have proposed is directly related to the high levels of prevalence and incidence of obesity in our population (12), the same relationship that has been reported by other authors (13,14).

In our analysis, biochemical variables related to oxygenation, blood chemistry, and acute phase reactants were included, as well as the evaluation of the procoagulant state that has a high impact on the pathophysiology of COVID 19. In the same way, within the Imaging resources, in many cases these are limited and various imaging techniques must be used, such as simple chest radiography, chest computed tomography, and pulmonary ultrasound to assess lung involvement in these patients (15).

We initially found that PaO2 was being reported with a value lower than the established parameters. Some authors have described that the decrease in PaO2 is presented as one of the main variables related to the need for hospital admission in the same way with the increase in mortality (16). A phenomenon called “happy hypoxia” has been described in patients with SARS CoV2 infection, in which values less than 40 mmHg have been found without presenting any symptoms (17). In our population, a significant difference in this factor was found between the groups with a fatal clinical outcome and those recovered after infection, which is why it is considered one of the main predictors of mortality. Additionally, we found an elevation of lactate in all groups, despite this, there is a marked difference between deceased and recovered patients. Some authors have suggested that there is a pathological process mediated by the toxicity of the virus on the beta cells of the pancreas, which is directly related to the elevation of lactate in patients (18), considering that this variable by itself can predict mortality in patients. Patients with SARS CoV2 infection (10, 19, 20).

Among the alterations of blood cells, various alterations have been found in the population infected by SARS CoV2, and alterations have even been described among various population groups (diabetics, hypertensive, obese, etc.). One of the main alterations found is in the leukocyte count, which has been reported that values greater than 8 x 103 x mm3 are related to more severe stages of the disease (21,22), while values less than 4 x103 x mm3 are associated with a better clinical outcome and hospital discharge (23). Some authors have found that leukocytosis is associated with a worse prognosis during the disease (24). Increased neutrophils have also been described in these patients, which is directly related to the chemotaxis produced by the release of cytokines during infection, and many authors have even described marked differences between discharged patients and those who died during their hospital stay, which has been found that the neutrophil count is higher in deceased patients (24, 25). The lymphocyte count in patients has been found with a tendency to lymphopenia (24-26), this related to lymphocytosis processes caused by direct toxicity of the virus; this lymphocyte depletion has been more marked in diabetic patients (24-26), is that in our population the value was lower in hypertensive patients. Lymphopenia has been related as a severity marker in patients with SARS CoV2 infection (21,24), which, in addition to the comorbidities presented in our population, increases the risk of presenting more severe conditions in those mentioned above.

In our analysis, differences in serum albumin concentration were found between recovered and deceased patients, this being consistent with that reported by other authors, who have found that critically ill patients present a more marked decrease in serum albumin values than in those recovered (22,24).

In multiple reports, altered values of lactic dehydrogenase have been found, as in our analysis. These elevations were more marked in those who died and those who recovered (10, 22, 23, 27, 28) and some authors have even suggested that this value can be considered as a predictor of severity and death in these patients (26). In the analysis of the D-dimer parameter, it has been reported that there are alterations in this value at admission in various series. In previous research, it has been described that elevations greater than 500 mcg /dL are highly related to a worse prognosis (23,27), while a result greater than 2000 ng/ml is considered a predictor of mortality in patients. With COVID 19.

One of the variables analyzed with the greatest significance was the elevation of C-reactive protein. This relationship has been described in this pathology, and various authors have even associated this elevation with worse prognoses concerning complications and death (21,26-29). Likewise, fibrinogen elevations have been reported in other series (30,31), which is directly related to the development of coagulopathy associated with COVID 19, which has been considered a worse prognostic factor in this disease (32, 33).

***Kidney Damage Induced by SARS CoV 2***

It is known that the virus uses the angiotensin-converting enzyme 2 (ACE2) as a receptor to enter the cell, however, the transmembrane serine protease type 2 (TMPRSS2) is also essential for this process, which facilitates the fusion of the virus through of the cell membrane thus achieving its invasion. The expression of these molecules in podocytes and cells of the renal proximal tubule make the kidney a target for the infection and replication of SARS CoV-2, a theory that has been reinforced by detecting viral antigens in postmortem samples of renal tissue and urinary samples from patients with acute kidney injury and COVID 19 (34).

Various theories have been proposed through which an attempt is made to explain the pathophysiological mechanism of kidney damage associated with COVID 19, within these the following mechanisms are proposed: a) association with cytokine storm, b) direct damage of the virus on kidney cells, c) Renin-angiotensin-aldosterone system imbalance and d) Thrombosis.

***Renin Angiotensin Aldosterone System Imbalance***

SARS-CoV-2 binds to ACE2, decreasing its expression in the membrane, thus indirectly promoting the accumulation of angiotensin II by reducing its degradation into angiotensin 1-7. Therefore, the accumulation of angiotensin II-mediated by COVID-19 may promote an unbalanced activation of the RAAS leading to inflammation, fibrosis, and vasoconstriction of the renal afferent artery (34-36).

***Direct virus injury to the kidney cell***

In various studies it has been reported that RNA was isolated in urine samples, also, the presence of vacuoles has been observed by electron microscopy, which contains multiple spherical particles in the cytoplasm of podocytes that could correspond to viral inclusion bodies of the SARS-CoV-2 which corroborates its renal tropism, while multiple renal biopsies reported the presence of severe focal segmental glomerulosclerosis and acute tubular necrosis that could be related to direct viral toxicity in tubular cells (35,37).

***Endothelial damage and thromboinflammation***

Microscopic studies have found evidence of the presence of SARS-CoV-2 viral particles in renal endothelial cells mediating an endothelial inflammatory response with a marked presence of activated neutrophils and macrophages that can trigger excessive thrombin production, inhibit fibrinolysis and activate complement pathways initiating thromboinflammation and, ultimately, causing microthrombus deposits and microvascular dysfunction (34,37).

***Dysregulation of the immune response***

There is multiple evidence that patients with severe COVID 19 present high levels of inflammatory cytokines such as interleukin 1-beta, IL-1RA, IL-7, IL-8, IL-9, IL-10, fibroblast growth factor, factor granulocyte, and macrophage colony growth stimulant, gamma interferon, IP10 protein, monocyte attractant protein, platelet-derived growth factor, and tumor necrosis factor-alpha. The conjunction of all these factors has been associated with endothelial and tubular dysfunction at the renal level. It has been proposed that tumor necrosis factor can bind directly to tubular cell receptors by triggering the apoptotic death receptor pathway. Other studies have also found the elevation of IL-6 significantly in critically ill patients. IL-6 induces an increase in renal vascular permeability, also inducing the secretion of pro-inflammatory cytosines by renal endothelial cells (IL-6, IL-8, and monocyte chemoattractant protein) (34,35).

The factors already described have also been associated with the cytokine release syndrome, closely related to the overactivation of innate immunity characteristically observed in patients with COVID 19; especially in severe patients.
Previous preclinical and human studies with pathogenic human coronaviruses have shown rapid viral replication, antagonism of interferon signaling and activation of neutrophils and monocyte-macrophages as mediators of hyper inflammation as well as the elevation of inflammatory markers such as C-reactive protein, ferritin, erythrocyte sedimentation rate, D-dimer, fibrinogen, and lactate dehydrogenase, which corroborate the presence of an altered immune response as well as a severe inflammatory state in response to the presence of SARS-CoV-2 in the body (36).

In the population analyzed, we found various alterations in renal function such as elevated Blood urea nitrogen and urea, as well as creatinine, are directly related to the decrease in the glomerular filtration rate in the various groups analyzed, the latter being the one most likely associated with the mechanisms previously described.

It has been reported by other authors that even about 30% of patients have evidence of elevated serum creatinine upon admission (38, 39), associating this with higher in-hospital mortality (38-40). In our population, we must recognize two major factors involved in this process: the first is that the coexisting comorbidities among patients can cause a decrease in the glomerular filtration rate in the long term, the second being the association of kidney damage induced by SARS CoV2 infection (40,41).

In the first factor, we must seek information that gives us an approximation of the glomerular filtration rate before admission and thus can differentiate between chronic and acute damage, and thus also contribute to the recognition of the pattern of acute kidney injury that occurs. presents on admission, and design better interventions that can influence the limitation of damage by COVID 19.

To address the second factor, renal function alterations are facts that we must pay particular attention to in clinical practice, as this can be an early manifestation of hyperinflammatory states in patients, particularly those who have not yet developed acute respiratory distress syndrome. (38, 40). Likewise, it is important to assess kidney function at admission, since a large number of drugs that have been proposed for the treatment of COVID 19 can also contribute to the perpetuation of this damage, and worsen the prognosis of these patients.

One of the main limitations of our study is that we do not have a follow-up of patients, and we cannot follow-up of the renal function of these patients, however, we consider that we have an open window to continue investigating on the subject and thus be able to create interventions that help improve the prognosis of these patients.

## CONCLUSIONS

The findings observed in our population and the high differences in glomerular filtration rate in patients with

COVID 19 are variables highly associated with increased mortality after hospital admission.

We must emphasize this point since although there is kidney damage during infection, other factors; as nephrotoxic drugs, comorbidities, or water management; also influence the persistence and increase of this problem. Our study has several limitations, and that is why we must continue researching this problem to more accurately predict fatal clinical outcomes or complications. For this reason, more studies are required to continue the study of kidney function in patients with COVID 19.

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