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Kidney dysfunction and mortality risk in hospitalized Covid-19 patients: A large Covid-19 centre experience

Mamven H.M.[1], Kwaghe G.V.[1], Habib G.Z.[2], Galadima S.U.[2]

Abstract

Objective: Kidney dysfunction is common in patients infected with the coronavirus (COVID-19). The study's objective was to determine the relationship between glomerular filtration rate and mortality in COVID-19 patients.

Methods: This is a retrospective cohort study of patients admitted into the COVID-19 isolation center from March 2020 through December 2021. The serum creatinine at admission was used to estimate the glomerular filtration rate (eGFR) using the CKD equation method. The patients were categorized into 2 groups based on the eGFR (≥or < 60ml/minute). The outcome was in-hospital mortality. Kaplan Meier survival plots and cox proportional modelling were employed in the data analysis.

Results: A total of 623 patients were analysed. The mean age was 53.4±15.3 years, and 58.6% were male. An eGFR of < 60 ml/min was observed in 196 (31%) patients. A significantly higher number of deaths occurred among patients with eGFR <60ml/min (32% vs 10.5% (P<0.001). After adjusting for age, sex, disease severity, haemoglobin, ICU admission, and dialysis, the patients with reduced eGFR of (<60ml/min) were twice more likely to die than patients with eGFR ≥60mls/min (AHR 1.95, 95% CI 1.26-3.04, P = 0.003).

Conclusion: eGFR of < 60mls/min is associated with an increased risk of mortality in COVID-19 patients. This stresses the need for better recognition of renal dysfunction as a high-risk for mortality in COVID-19 infections.

Keywords- COVID-19, eGFR, Mortality

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Article Original Research Journal of Health Sciences

Dysfonctionnement rénal et risque de mortalité chez les patients hospitalisés Covid-19: une grande expérience de centre COVID-19

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Résumé

Objectif de l'étude: Le dysfonctionnement rénal est fréquent chez les patients infectés par le coronavirus (COVID-19). L'objectif de l'étude était de déterminer la relation entre le taux de filtration glomérulaire et la mortalité chez les patients COVID-19.

Méthode de l'étude : Il s'agit d'une étude de cohorte rétrospective de patients admis dans le centre d'isolement COVID-19 de mars 2020 à décembre 2021. La créatinine sérique à l'admission a été utilisée pour estimer le taux de filtration glomérulaire (eGFR) à l'aide de la méthode CKD EpI. Les patients ont été classés en 2 groupes en fonction du DFGe (≥ou < 60 ml/minute). Le critère de jugement était la mortalité hospitalière. Des diagrammes de survie de Kaplan Meier et une modélisation proportionnelle de Cox ont été utilisés dans l'analyse des données.

Résultat de l'étude: Au total, 623 patients ont été analysés. L'âge moyen était de 53,4 ± 15,3 ans et 58,6 % étaient des hommes. Un DFGe < 60 ml/min a été observé chez 196 (31 %) patients. Un nombre significativement plus élevé de décès est survenu chez les patients avec un DFGe<60 ml/min (32 % contre 10,5 % (P< 0,00 1). Après ajustement en fonction de l'âge, du sexe , de la gravité de la maladie , de l'hémoglobine, de l'admission en USI et de la dialyse, les patients avec un DFGe réduit de (<60 ml/min) étaient deux fois plus susceptibles de mourir que les patients avec un DFGe ≥60 ml/min (A HR 1,9 5 , IC à 95 % 1,2 6- 3,0 4 , P = 0,00 3).

Conclusion : un DFGe < 60 ml/min est associé à un risque accru de mortalité chez les patients COVID-19. Cela souligne la nécessité d'une meilleure reconnaissance de la dysfonction rénale en tant que risque élevé de mortalité dans le COVID-19 infections.

Titre du fonctionnement courant : Taux de filtration glomérulaire estimé et risque de mortalité.

Mots-clés - COVID-19, eGFR, mortalité

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INTRODUCTION

Coronavirus disease (COVID-19) is a novel disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that is responsible for the global pandemic recently (1). Though respiratory involvement is the major presentation, other organ involvement is common, especially in severe cases. (2) The kidneys are one of the most commonly affected organs. Kidney abnormalities have been reported globally by several authors (3-6). The spectrum of renal involvement described in COVID-19, includes urinary abnormalities and changes in kidney function (reflected by decreased glomerular filtration rate (GFR) which might be present in up to 75%-80% of cases.(6) Acute kidney injury (AKI) at one end of the spectrum is a common complication and may be due to several causes such as sepsis, hypotension, or glomerulonephritis (7-9). Proteinuria and haematuria with or without the loss of kidney function are also common abnormalities encountered in these patients (7-10).

Several mechanisms for kidney damage have been proposed such as acute tubular injury from systemic hemodynamic changes, tissue inflammation, and local immune cell infiltration with endothelial injury and microvascular thrombi and possibly viral invasion in the kidneys. An impaired type I interferon response has also been reported in patients with severe COVID-19 (11). Kidney dysfunction in COVID-19 patients is associated with increased mortality (5,10,12). Other Significant independent predictors of mortality reported are older age, the presence of comorbidities such as diabetes mellitus, hypertension, and proteinuria (12-14).

During the early part of the COVID-19 pandemic, not much attention was paid to testing for kidney abnormalities routinely during hospitalization in many centres in Nigeria, unless there were obvious signs of kidney involvement, which might have been noticed too late. Our centre is a major referral centre that is well equipped to manage systemic complications such as kidney failure and has good laboratory support to run samples with a quick turnaround time of results. This significantly contributed to our success in managing COVID-19 infection. Few large studies have been conducted on kidney function and mortality outcomes in COVID-19 patients in Nigeria. The purpose of our study was to investigate the impact of eGFR (kidney function) on in-hospital mortality in hospitalized patients with COVID-19. We hypothesize that an estimated glomerular filtration rate (eGFR) of less than 60mls/min/1.73m^2 at admission is significantly associated with mortality among patients with COVID-19. The finding of this study will increase clinicians' awareness of kidney dysfunction in our hospitalized patients with COVID-19.

MATERIALS AND METHODS

Setting and design

The study was conducted at a major referral hospital in the North Central region of the Country. The hospital is a treatment centre for moderate to severe cases of COVID-19 in the Federal Capital Territory (FCT) in Nigeria.

The design was a retrospective cohort study involving all adult patients aged 18 years and above with at least one respiratory sample positive for SARS-CoV-2 by polymerase chain reaction (PCR), admitted to the isolation and treatment centre between the 21st of March 2020 and December 2021. We excluded patients with no measurements of creatinine at admission.

Sample size determination

For obtaining the minimum sample size, assuming that 18.4% of COVID-19 patients with eGFR > 60mls/min will die during the study,(14) and a prevalence of reduced eGFR in COVID-19 patients of 30% (14), with a power of 90% and alpha of 0.05, we would like our study to have adequate power to detect a relative risk of 2.0. A minimum sample size of 294 with N1=89 and N2=205 was required.

Data sources

The data were extracted from the patient medical records by a trained research assistant.

Variables and definitions

The primary exposure of interest was the eGFR. Using the creatinine obtained at admission, age and sex it was calculated using the CKD-EPI creatinine formula (15). Other exposures were age, sex, clinical features on admission such as oxygen saturation (SP02%), blood pressure, severity of COVID-19 disease, Elixhauser comorbidity index score (CIS), treatment received such as oxygen supplementation, renal replacement therapy use, use of medications and intensive care unit requirement. Laboratory data was full blood count, serum urea, creatinine, potassium, sodium, and bicarbonate obtained during admission.

For the purpose of this study, patients were categorised into two groups according to

their eGFR. Group one was made up of patients with eGFR ≥60ml/min/1.73 while group two was made up of those with eGFR <60ml/min/1.73 m^2 which (was termed low eGFR).(15)

The severity of respiratory COVID-19, cases was obtained from the patient's records and were categorized as mild, moderate, and severe. The comorbidity index score (CIS) was according to Elixhauser comorbidities and van wolverine scoring .(16,17) The neutrophil-lymphocyte ratio (NLR) at hospital admission was calculated as the ratio of neutrophils to lymphocytes and both were obtained from the blood sample collected.

The outcome was in-hospital mortality and the time from admission to death. In-hospital mortality was all-cause deaths within 30 days of being admitted to the hospital. The time to event was the time from hospital admission to events which was all-cause death. Censored observations were those who had not yet had the event. Patients were censored on the day of discharge or 30 days from the day of admission if still alive and on 31st December 2021, the final date of follow-up for this study.

Statistical analysis

The normality of variables was assessed using visual inspection of histograms and confirmed by the Shapiro–Wilk test. Any variable with more than 10% missing was not used.

Patient characteristics were described for the overall cohort according to the two groups of eGFR. Categorical variables were summarized as proportions and percentages and continuous variables were expressed as the mean and standard deviation (SD) or median with interquartile range (IQR) for skewed measures. We compared characteristics between the eGFR groups using chi-square or Fisher exact tests for categorical variables. Two samples independent t-test or Wilcoxon rank-sum (Mann-Whitney U) test for skewed data was used to compare continuous variables between the two groups.

We explored the relationship between eGFR and 30-day mortality using a Kaplan–Meier survival curve with the log-rank test. Univariable and multivariable Cox proportional hazards regression models were estimated, to further explore the relationship between eGFR and in-hospital death while adjusting for any confounders. Results were reported as hazard ratios (HR) with a 95% confidence interval. The proportional hazard assumption was tested using graphical means.

Given the multiplicity of variables, We performed variable selection for the multivariable model building and used a stepwise selection of variables to select variables for the model. Other variables of known clinical relevance were added. Potential confounding variables were age, sex, comorbidities index, disease severity, haemoglobin concentration, and ICU admission. All tests were two-sided and the statistical significance was P <0.05 for all analyses.

Data were collected and managed using Excel and statistical analyses were performed using STATA software (16.1 StataCorp LLC, College Station, TX).

Ethical statement

The study was approved by the Health Research Ethics Committee of the institution with the number: UATH/HREC/PR/2022/003/006 on 21/03/2022 and was conducted in accordance with the National HREC code and with the Helsinki Declaration of 1975, as revised in 2000. A waiver of informed consent for patients was obtained as this was a review of the data collected. Strict confidentiality of data was maintained.

RESULTS

Out of the 750 patients hospitalised with COVID-19 during the period, 623 formed the primary sample for analysis. 196 (31%) had eGFR of < 60ml/min.

Characteristics of the primary sample are demonstrated in Table 1. A significant proportion of the lower eGFR group of < 60 ml/min was older and had a higher proportion of patients with diabetes, hypertension, anaemia, severe COVID-19, sepsis, and higher median CIS than those with eGFR ≥ 60 ml/min. Patients with eGFR < 60 ml/min also had lower levels of median oxygen saturation, lower mean haemoglobin, and lower bicarbonate levels. They also had higher mean WBC, and NLR. The treatment most frequently used was antibiotics (64.4%), dexamethasone (60.4%), and clexane (56.8%). The group with the lower eGFR was less frequently treated with antivirals (lopinavir/ritonavir) (6.6% vs 30.4%) and were more frequently treated with antibiotics (85% vs 54.9%), dexamethasone (79.5% vs 52%) and oxygen supplementation (55.6% vs 32.0%). A higher proportion of deaths occurred among patients with lower eGFR than in the higher eGFR group. (32 % vs 10.5%; P<0.001) (Table 1)

Figure 1 displays the Kaplan-Meier survival curves in the two eGFR groups.

Mortality was significantly higher in patients with eGFR < 60mls/min/1.73m^2 (global log-rank test P < 0.001). There were no violations of the Cox proportional hazards assumptions graphically.

Tables 2a and b demonstrate the distribution according to survival status in the patients. A significantly higher proportion of patients with COVID-19 who died were older. Those who died had more severe COVID-19 than mild disease, and higher median CIS than survivors. Table 2b shows the treatment of the patients. A significantly higher proportion of patients who were admitted into ICU died compared to those who were not. More of those treated with antibiotics (25.3 vs 3.7%), clexane (24 vs 9%) and with corticosteroids (25vs 7%) died compared to those who were not. A significantly higher proportion of patients who had dialysis did not survive compared to those who did not have (Table 2).

The Cox proportional analysis is displayed in Table 3, the unadjusted hazard ratio for eGFR
<60 ml/min on mortality was 3.3, while the adjusted hazard was 1.95 (95% CI, 1.26-3.04). The confounders, were age, sex, disease severity, haemoglobin, ICU admission, and dialysis. (Table 3). The Cox proportional hazards assumption after adjusting for confounders was met on graphical analysis.

DISCUSSION

In this study, we looked at the kidney function using the eGFR observed during admission and associated it with Mortality in patients afflicted with COVID-19. We observed that 31% of our patients had an eGFR of < 60 mL/min/1.73 m^2. This is consistent with several other authors globally that kidney dysfunction (low eGFR of <60 mL/min/1.73m^2) is not uncommon in confirmed cases of COVID-19 infection.(14, 18, 19) Uribarri, Mirijello and Cei reported similar values of about 30%,27.3% and 30% respectively in their patients.(14, 18, 19) On the other hand Cheng reported lower prevalences of elevated serum creatinine, blood urea nitrogen and eGFR under 60 ml/min/1.73m^2 of 14.4, 13.1 and 13.1%, respectively .(10) In SSA and specifically reporting on AKI as the dysfunction, Ibrahim et al. reported AKI occurring in 14.6% of their patients while Dolaamas reported higher in 32.6% .(20, 21) A report from a hospital-based registry in Ghana showed that 10% of patients admitted with COVID-19 had underlying CKD with AKI in nearly half of the cases.(22) This wide variation in prevalence of the kidney abnormalities is most likely due to heterogeneity in cohorts studied, from variations in definitions and components of kidney dysfunction to methods employed in diagnosis and reporting. While the studies reported on several renal abnormalities, our study reported low eGFR as the sole dysfunction.

Kidney disease in COVID-19 is associated with an enhanced risk of deterioration and mortality. In this study, we observed 32% mortality in our patients with kidney dysfunction and 10.5% in those with eGFR >60mls/min. The hazard of mortality in our patients wth dysfunction was 1.9, 95% CI: 1.26-3.04, p = 0.003 after adjusting for age, sex, disease severity, haemoglobin levels, ICU admission, and dialysis as confounders. Similarly, several other investigators worldwide reported high risk of mortality in their COVID-19 cohorts with renal dysfunction. In Africa, Dolaama reported death in 55.8% of their patients with kidney dysfunction and the factors associated with death were, KDIGO stage (p = 0.049), and invasive ventilation (p < 0.001). (21) In Ghana, In-hospital mortality of 43.5% was reported among those with CKD in an unpublished hospital-based report of COVID-19 patients admitted at the Komfo Anokye Teaching Hospital as of February 2021.(22)

In Italy, Cei, et al., showed that an eGFR value of <60 mL/min/1.73 m2 (OR 2.6,95% CI:1.7-4.8, p = 0.003); as well as age > 73 years (OR 4.3, 95% CI: 2–9, p < 0.001), lymphocyte count below 460 U/L (OR 3, 95% CI 1.4–6.4, p = 0.004) and platelet count below 177,000 (OR 2.2, 95% CI 1.2–4.2, p = 0.017) were significantly associated with in-hospital mortality (19), also in Italy Mirijello demonstrated a risk of 1.64 for low eGFR (AHR 1.64, 95% CI 1.02–2.63, P = 0.040).(18) In china Cheng et al., reported 16% of their patients died in-hospital and had a significantly higher risk for in-hospital death with an elevated creatinine (AHR:2.10, 95% CI: 1.36-3.26) (10). Pei et al. reported mortality in 11.2% of their patients with kidney involvement compared with (1.2%) of patients, without (7). Chan et al., reported higher mortality of 50% among their patients with AKI versus 8% among those without AKI (AOR, 9.2; 95%CI: 7.5 to 11.3) (4). A meta-analysis by Robbins-Juarez et al. reported higher mortality of 52% of patients with severe COVID-19 infection with AKI and a pooled odds ratio of 15.27; 95% CI 4.82-48.36) .(23) Contrary to the above studies, Bravi et al., reported that eGFR was not

an independent predictor of poor outcomes including death, however, their population was composed of younger patients with a higher prevalence of normal eGFR and fewer co-morbidities (24). There is no doubt that mortality risk is increased in COVID-19 patients with kidney dysfunction. Reasons for the high mortality rate observed in our cohort are proffered. Mortality occurred more in the older patients as they tend to have higher prevalence of kidney diseases, more comorbidities and so poorer prognoses. The higher mortality observed in older patients with comorbidities may be related to a reduction in their renal functional reserve, an impaired capacity of their kidney to increase GFR in response to stress, and reduced functioning nephron mass (25). Chronic kidney disease alone, or with other comorbidities such as diabetes mellitus, hypertension and obesity may be present in some of our patients and is linked to increased mortality in patients with COVID-19.(10) There was a higher occurrence of these comorbidities in our patients with eGFR < 60mls/min/1.73m2. Our findings show that sex, severity of Covid-19, eGFR <60mls/min, WBC count, and use of antibiotics were significantly associated with mortality in the relationship. (table 3)

This study has some limitations even as it represents a real-life setting in a tertiary health centre in the tropics. It was a retrospective design with some missing data. only those with creatinine values taken at admission were included in the study. There was no baseline information on kidney function (serum creatinine) in some patients and almost all of the patients had creatinine done only on admission and once so we could not differentiate between AKI or if pre-existing CKD was the case. During the early phase of the COVID-19 pandemic, available resources were focused more on COVID-19 infection, so kidney function was assessed only in patients who had symptoms, signs, and known risk factors of kidney disease. This may have led to an underestimation of kidney dysfunction or distorted associations between COVID-19 infection and kidney outcomes. We did not include urine analysis because it was not done for most of the patients. complicating the reliability of the diagnosis. Nonetheless, this study was performed in a tertiary-level hospital and a dedicated referral center for COVID-19 especially for severe infections in the Federal Capital Territory and beyond, therefore, the result can be generalisable locally.

CONCLUSION

In conclusion, kidney dysfunction in our hospitalized patients with COVID-19 was high. Patients with eGFR, < 60mls/min/1.73m2 were 2 times more likely to die than those with eGFR $\geq$ 60mls/min/1.73m^2. Findings from our study underscore the importance of looking out for kidney abnormalities in COVID-19 patients. Even though the infection and the pandemic seem to have subsided, it is still pertinent for clinicians and hospitals in Nigeria to be ready and prepared for future occurrences or epidemics of COVID-19.(26, 27) Clinicians should be aware of kidney diseases in COVID-19 infections and monitor closely patients who are at risk of kidney involvement this may reduce mortality in these patients.

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Author's Contribution: MM and VK developed the concepts, the definition of intellectual content and manuscripts preparation while both the design of the study and the statistical analysis of the acquired data was done by MM. The literature search was carried out by MM, ZH and UG. VK, ZH and UG did the data acquisition. The manuscript editing and review was done by MM, VK, ZH and UG.

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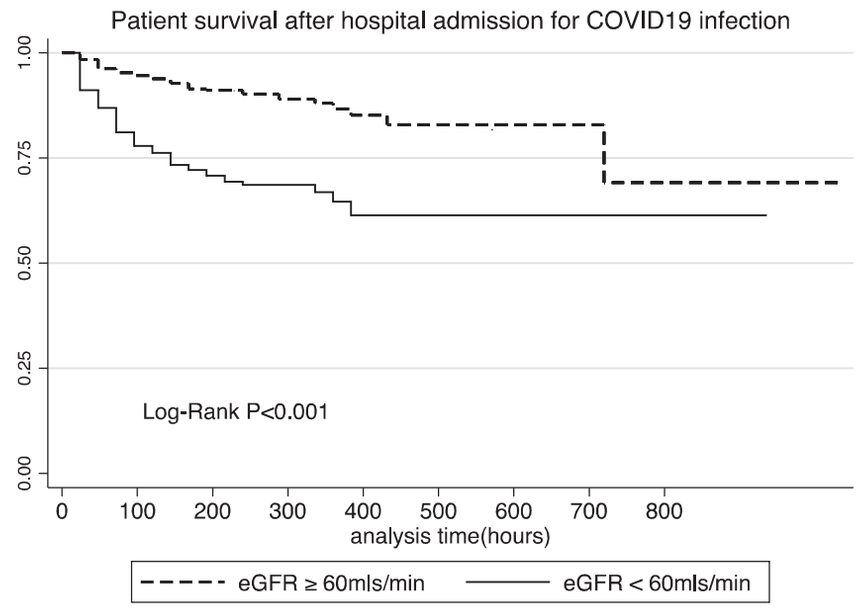
Table 1: Clinico-demographic, laboratory and treatment profiles of COVID-19 patients during hospitalization

Parameters	All patients	eGFR =60mls/min	eGFR<60mls/min
Total number (%)	623	427(68.5%)	196(31.4%)
Mean eGFR mls/min	76.7±39.0	98.5± 22.6	29.1±20.0
Mean age(years)	53.4±15.3	50.9±15.5)	58.9±13.4
Sex Male (%)	386(61.9)	271(63.4)	115 (58.6)
Female	237(38)	156(36.5)	81 (41.3)
Mean temp ^{0}C	36.6±0.6	36.48±0.5	36.7± 0.8
Temp >37.2^{0}C (%)	62(9.9)	27(6.3)	35(18)
Mean Spo2.	93.1±9.7	94.5± 7.9	90.1±12.3
MAP	100.9±14.7	101.4± 14.6	99.8± 14.9
Severity of COVID			
Mild	196(32.0)	177(42.2)	19 (9.8)
Moderate	169(27.6)	104(24.8)	65 (33.7)
Severe	247(40.3)	138(32.9)	109 (56.5)
Diabetes (%)	197(31.7)	121(28.40)	76 (38.8)
Hypertension (%)	322(51.6)	194(45.4)	128 (65.3)
Sepsis (%)	25 (4.0)	12 (2.8)	13 (6.6)
Median CIS	0(0,3)	0(0,0)	0(0,5)
Laboratory			
Mean WBC	10.4± 6.5	9.4±5.9	12.4±7.4
Median NLR ratio	2.3 (1.3,4.3)	2.06(1.2,3.8)	2.95(1.8,5.5)
Mean Hemoglobin, g/l	12.4±2.7	12.9±2.3	11.43±3.04
Mean HGB = 10(%)	497(84)	359(88.9)	138 (73.8)
Mean HGB <10	94(15.9)	45(11.1)	49 (26.20)
Median urea mmol/l	5.8±(4.1,9.8)	4.8 (3.7,6.5)	14.7(8.4,23.2)
Med Creatinine umol/l	82(64, 117)	69(52, 82)	208.5(125.5,450.5)
Mean Sodium mmol/l	138.5± 3.9	138.7± 3.3	138.1± 4.9
Mean Potassium mmol/	4.0± 0.5	4.0± 0.5	4.1±0.6
Mean HCO3	22.8±3.3	23.2± 2.8	21.8±3.9
Treatments (%)			
Antibiotics	395(64.4)	229(54.9)	166(84.7)
Diuretics	24(3.9)	9(2.2)	15 (7.7)
Hydroxychloroquine	118(18.9)	92 (21.6)	26 (13.3)
Lopinavir-ritonavir	143(22.9)	130(30.4)	13 (6.6)
Remdesevir	151(24.3)	94(22.1)	57 (29.1)
Dexamethasone	376(60.7)	220(52.0)	156 (79.6)
Clexane	354(56.8)	207(48.5)	147 (75)
Zinc	443(71.2)	323(75.8)	120 (61.2)
Oxygen supplementation	246(39.5)	137(32.1)	109 (55.6)
Dialysis	23(3.7)	0(0)	23/196(11.7)
ICU admission	25(4)	7 (1.64)	18 (9.18)
Died	108 (17.4)	45(10.5)	63 (32.1)
Median LOS	9(6,13)	9(7,13)	9(4,13)

Abbreviations: eGFR estimated glomerular filtration rate, MAP mean arterial pressure, Spo2 oxygen saturation, CIS comorbidity index score, HGB haemoglobin, ICU intensive care unit, WBC white blood cells, NLR neutrophil lymphocyte ratio, HCO3 bicarbonate, LOS length of stay, *Mann-Whitney U



Number at risk

Time(hrs)	0	100	200	300	400	500	600	700
eGFR=60	427	379	246	119	47	22	10	6
eGFR<60	191	141	99	49	16	8	2	2

Figure 1: Kaplan-Meier curve for patient survival after hospital admission for COVID-19 infection according to eGFR.
The median survival time was not defined because less than half of the patients have experienced an event by day 30.

Table 2a: Distribution according to survival status in patients with COVID-19

Parameters	Numbers (%)	Survivors N=515 (82.7%)	Non survivors N=108 (17.3%)	P-value
eGFR = 60mls/min(%)	427	382(89.5)	45 (10.5)	<0.001
eGFR <60mls/min	196	133(67.9)	63(32.1)	
Age: <60 years	392(63)	339(86.5)	53(13.5)	<0.001
= 60 years	231(37)	176(76.2)	55(23.8)	
Sex male (%)	386(62.0)	326(84.5)	60(15.5)	0.132
Females	237(38.0)	189(79.7)	48(20.3)	
Mean temperature^{0}C	36.6±0.6	36.5± 0.6	36.7± 0.8	0.002
Temperature = 37.2	62(10.0)	47(75.8)	15(24.2)	0.133
Temperature <37.2	561(90)	468(83.4)	93(16.5)	
Mean Spo2	93.1±9.7)	94.7±7.4	85.9±14.8	<0.001
MAP	100.9±14.7	100.8±14.1	101.2±17.4	0.808
Severity of COVID (%)				
Mild	196(31.5)	191(97.1)	5(2.5)	<0.001
Moderate	168(27.0)	149(88.7)	19(11.3)	
Severe	248(39.8)	164(66.1)	84(33.9)	
Diabetes:yes (%)	197(31.7)	152(77.2)	45(22.8)	0.011
No	425	363(85.4)	62(14.6)	
Hypertension (%)	322(51.7)	251(77.9)	71(22.1)	0.001
No	301	264(87.7)	37 (12.29)	
Sepsis (%)	25(4.01)	17	8(32)	0.048
No	598	498(83.3)	100(16.7)	
Median CIS	0(0,3)	0(0,3)	0(0,5)	0.006*
Laboratory				
Mean WBC count,	10.4±6.5	9.6±6.1	14.2±7.2	<0.001
Median NLR	2.3 (1.3,4.3)	2.1(1.2,3.6)	3.9 (2.3,7.6)	<0.001*
Median urea mmol/l	5.8 ±(4.1,9.8)	5.5(3.9,8.6)	9(5.0,17.9)	<0.001*
Median creatinine umol/l	82(64,117)	77(62.3,108)	110(82, 352.5)	<0.001*
Mean Hemoglobin g/l	12.4± 2.7	12.53± 2.6	11.91± 2.9	0.032
HGB = 10 (%)	497(84.1)	418(84.1)	79(15.9)	0.024
HGB <10 (%)	94(15.1)	70(74.47)	24(25.5)	
Mean Sodium, mmol/l	138.5± 3.9	138.6± 3.6	138.1± 4.9	0.245
Mean Potassium, mmol/l	4.05±0.6	4.03±0.5	4.16± 0.7	0.034
Mean HCO3	22.8±3.3	22.9±3.2	22.3±3.7	0.149

Abbreviations: eGFR estimated glomerular filtration rate, MAP mean arterial pressure, Spo2 oxygen saturation, CIS comorbidity index score, HGB haemoglobin, ICU intensive care unit, WBC white blood cells, NLR neutrophil-lymphocyte ratio, HCO3 bicarbonate, LOS length of stay, *Mann-Whitney U

Table 2b: Treatment profile according to survival status in patients with COVID-19

Parameters	Numbers	Survivors	Non survivors	P-value
		N=515 (82.7%)	N=108 (17.3%)	
Treatments (%)				
Antibiotics	395(64.4)	295(74.7)	100(25.3)	<0.001
No	218	210(96.3)	8(3.7)	
Diuretics	24(3.9)	17(71)	7(29)	0.127
No	591	490(83)	101(17)	
Hydroxychloroquine	118(19)	105 (88.9)	13(11.9)	0.044
No	505	410(91.2)	95(18.8)	
Lopinavir- ritonavir	143(23)	136(95.1)	7(4.9)	<0.001
No	480	379(79)	101(21.04)	
Remdesevir	151	120 (79.5)	31(20.5)	0.242
No	470	393(83.62)	77(16.38)	
Dexamethasone	376(61)	284(75.5)	92(24.5)	<0.001
No	243	227(93.4)	16 (6.6)	
Clexane	354	270 (76.3)	84(23.7)	<0.001
No	269	245 (91.0)	24 (9.0)	
Zinc	443(71.2)	384 (86.7)	59 (13.3)	<0.001
No	179	130 (72.6)	49 (27.4)	
O_2 supplementation	246(39.5)	166 (67.5)	80(32.5)	<0.001
No		349(92.6)	28(7.4)	
Dialysis	23(3.7)	16(69.5)	7(30.4)	<0.001
No		499(83.2)	101(16.8)	
ICU admission	25(4.0)	12(48)	13(52)	<0.001
No	598	503(84.11)	951(5.89)	
Median LOS	9(6,13)	10 (7,13)	3(1.5,6)	<0.001

Abbreviations: eGFR estimated glomerular filtration rate, MAP mean arterial pressure, Spo2 oxygen saturation, CIS comorbidity index score, HGB haemoglobin, ICU intensive care unit, WBC white blood cells, NLR neutrophil-lymphocyte ratio, HCO3 bicarbonate, LOS length of stay, *Mann-Whitney U

Table 3: Cox proportional analysis of the relationship between eGFR and mortality in COVID-19 patients.

	Univariate analysis			Multivariable analysis		
Covariate	Crude-HR	95% CI	P-value	Adj-HR	95% CI	P-value
eGFR =60ml/min	ref			ref		
eGFR<60mls/min	3.3	2.24-4.86	<0.001	1.95	1.26-3.04	0.003
Age <60 years	ref			ref		
Age = 60 years	1.76	1.20-2.57	0.004	1.15	0.76-1.72	0.500
Sex Male%	0.69	0.47-1.02	0.063	0.61	0.39-0.92	0.019
Severity of disease						
Mild	ref			ref		
Moderate	4.83	1.81-12.96	0.002	1.84	0.65-5.25	0.253
Severe	15.17	6.15-37.45	<0.001	4.73	1.79-12.55	0.002
Sepsis	1.95	0.94-4.01	0.071	1.11	0.52-2.38	0.786
HGB =12g/l	ref					
HGB 10-11.9	1.04	0.62-1.76	0.873	0.62	0.36-1.07	0.088
HGB <10	1.64	1.01- 2.64	0.043	0.85	0.50-1.45	0.557
WBC	1.06	1.04-1.08	<0.001	1.04	1.01-1.06	0.001
CIS	1.07	1.02-1.12	0.009	1.03	0.98-1.09	0.180
Temperature >37.2	1.35	0.77-2.37	0.296	0.83	0.45-1.51	0.543
ICU admission	3.79	2.08-6.92	<0.001	1.25	0.64-2.43	0.515
Antibiotics	7.16	3.48-14.72	<0.001	3.46	1.52-7.88	0.003
Dialysis	1.87	0.87-4.03	0.111	0.74	0.31-1.78	0.497

Abbreviations: eGFR estimated glomerular filtration rate, CIS comorbidity index score, HGB haemoglobin, ICU intensive care unit, WBC white blood cells.