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Therapeutic Evaluation of COVID-19 Patients Complicated by Acute Kidney Injury

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ABSTRACT

During severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, the chance of suffering acute kidney injury (AKI) increases significantly. Therefore, the present study aimed to conduct a comprehensive pharmacotherapeutic evaluation of AKI in patients with coronavirus. From July to August 2021, a retrospective cohort study was conducted on coronavirus patients hospitalized at the Institute of Kidney Diseases, Hayatabad Medical Complex in Peshawar, Pakistan. Demographics, diagnosis, laboratory values, vital signs, and hospital care were all utilized to extract data. Parametric statistics, including regression analysis, one-way ANOVA, and Kruskal-Wallis, were used to examine the association between independent variables. Data from 595 coronavirus patients whose PCR results met predetermined criteria were collected. The most common symptoms among the patients were fever (n = 575 [96.6%]), lack of breathing (n = 570 [95.8%]), dry cough (n = 449 [75.5%]), and body pains (n = 129 [21.7%]). During their hospitalization, most patients were taking multiple medications. Overall, it was shown that coronavirus patients with stage III AKI had a significant reduction in most laboratory indicators. Compared with those without AKI, the mortality rate for AKI patients was 42% [0.418 [0.269–0.632], P < 0.001]. Intravenous dexamethasone was associated with a 96% reduction in mortality (1.968 [1.277–3.033], P = 0.002). To prevent the appearance of AKI in coronavirus patients, a clinician's top priority is to maintain appropriate oxygen saturation and refrain from using nephrotoxic medications while the patient is in the hospital.

Keywords: COVID-19, SARS-CoV-2, Kidney disease, AKI, Renal failure

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Introduction

The single-stranded RNA viruses that make up the coronavirus class of diseases are mostly found in humans and non-human animals [1]. These viruses are members of the Coronaviridae family, which is a subfamily of the gamma, delta, beta, and alpha coronaviruses in the order Nidovirales [2]. The novel COVID-19 was identified as SARS-CoV-2, which first appeared in late 2019, and spread COVID-19 without showing any specific preference [3]. According to a comparison assessment of coronavirus clinical characteristics, infected patients exhibit symptoms similar to pneumonia. However, cough, fever, and exhaustion are typical symptoms following the commencement of SARS-CoV-2 infection; other signs include diarrhea, dyspnea, hemoptysis, sputum production, and lymphopenia [4, 5]. While respiration signs are the primary clinical features of this illness, hospitalized

coronavirus patients have a greater probability of experiencing acute kidney damage (AKI); therefore, renal involvement throughout the illness is also a major concern [6].

Acute kidney damage (AKI) is much more likely to occur when SARS-CoV-2 is present [7]. Acute tubular necrosis during AKI is believed to be linked to multi-organ shock and failure, while the precise mechanism of action of coronavirus-associated AKI is yet to be understood [7]. Proteinuria, hematuria, high blood urea nitrogen (BUN), and baseline serum creatinine are a few signs of AKI when coronavirus is present [8]. There is a pressing requirement for renal replacement therapy (RRT) because the signs of AKI can occasionally get so bad during COVID-19 that conservative care is no longer effective [9]. The reduction of inflammatory cytokine levels has been achieved with great effectiveness with continuous renal replacement therapy (CRRT) [10].

According to several studies, acute renal damage has been documented in coronavirus patients when they are in the hospital, and it is more likely in individuals who are severely sick or who have other concomitant diseases. KDIGO (kidney disease improving global outcomes) level III AKI accounted for the majority of the 42.9% of AKI that occurred in 99 terminally ill coronavirus patients in a cohort investigation [11]. A retrospective analysis of hospitalized patients in New York City found that patients with coronavirus had a greater likelihood of AKI and needed advanced medical units, mechanical ventilation, and kidney transplant treatment than those lacking the virus [12]. AKI is mentioned as a separate risk element for death in clinical studies of coronavirus [13]. According to studies, severely sick AKI patients with coronavirus infection had an 8%–23% higher chance of dying [8, 14]. According to some investigations, coronavirus patients with level I, level II, and level III AKI had in-hospital death rates of 62%, 77%, and 80%, respectively [15].

There is a dearth of information on AKI in coronavirus patients worldwide, and Pakistan is significantly below the other parts of the globe in reporting these cases [16]. Comprehending how the kidney functions when infected with SARS-CoV-2 and how this virus causes acute kidney damage is therefore critically required, particularly for the Pakistani population. We can comprehend the various clinical presentations of AKI in coronavirus patients by investigating such data from Pakistan in the form of a retrospective cohort. This will eventually assist us in handling coronavirus patients more effectively over time. The purpose of the present research is to do a thorough pharmacotherapeutic assessment for AKI in coronavirus patients in light of the aforementioned reality. Comparing the prevalence, risk variables, and consequences of AKI in normal and at-risk populations is the goal of the present research.

Materials and Methods

Study design and population

A retrospective cohort study was conducted from July to August 2021 among COVID-19 patients admitted at the Institute of Kidney Diseases, Hayatabad Medical Complex hospital in Peshawar, Pakistan. This medical complex was given the responsibility for the treatment of patients suffering from COVID-19. This medical complex was one of the single facilities in Peshawar that hosted the greatest number of COVID-19 patients daily at that time. Ethical approval for this study was obtained from the Institutional Review Board of the Hayatabad Medical Complex. A confirmed case of COVID-19 was defined by a positive RT-PCR assay of a specimen collected via nasopharyngeal swab. The inclusion criteria for this study were 1) all adult patients who tested positive by polymerase chain reaction (PCR) testing of a nasopharyngeal sample for SARS-CoV-2 infection, 2) only first hospitalization record was included for the patients who had multiple qualifying hospital admissions, and 3) only quantitative data from Institute of Kidney Disease, Hayatabad Medical Complex hospital were included. Whereas exclusion criteria for this study were 1) data of those patients were excluded if they were transferred to hospitals out of the health system from where it was impossible to obtain data, 2) data from those patients were excluded from whom the consent was not obtained, and finally, 3) no data outside of Institute of Kidney Disease, Hayatabad Medical Complex hospital was included.

Data collection

The data were extracted based on demographics, diagnosis, laboratory parameters, vital signs, and the treatment used during the hospitalization. Demographics include the gender and age of the participants. Diagnosis and classification of AKI were based on Kidney Disease Improving Global Outcomes (KDIGO) guidelines [17]. Serum creatinine of more than 0.3 mg/dl or increase to more than 1.5–1.9 times from the baseline serum creatinine level was categorized as Stage I AKI; a serum creatinine of more than 2–2.9 times from the baseline value was

categorized as Stage II AKI; whereas, serum creatinine of three times more than the baseline value or a level of more than 4 mg/dl was categorized as Stage III AKI. Laboratory parameters were collected from day one of the hospitalizations till the last day at the hospital. Vital signs were included along with laboratory parameters. A list of various drugs used for the management of COVID-19 and associated symptoms was also included and compared for various outcomes.

Statistical analysis

The collected data were processed using the Statistical Package for Social Science (SPSS) software program for Windows version 21.0 (SPSS Inc., Chicago, IL). The data were analyzed using appropriate descriptive statistics such as mean and standard deviation. Apart from this, the association of independent variables like gender, age, etc., with dependent variables like clinical symptoms, hospitalization, infection severity, co-morbidity, development of AKI, and mortality was explored using parametric statistics such as regression analysis, one-way ANOVA, and Kruskal-Wallis (only if the data were not normally distributed). The statistical significance level was 0.05 with a confidence interval of 95%.

Results and Discussion

Demographic details of the participants

From the nephrology ward of HMC Hospital, information was collected on 595 COVID-19 patients who had positive PCR testing according to predetermined criteria. The average age of the patients was 53 ± 13.55 years, while nearly all of them were sixty years of age or older. While the majority of patients (n = 423 [71.0%]) were either released or deemed dead during the first 10 days of hospitalization, the average length of stay for the patients was 6 ± 13.55 days. Males made up around 65.8% of the patients in this cohort research. The majority of patients had hypertension (n = 244 [41.0%]) and diabetes mellitus (n = 282 [47.3%]). Patients were classified into level I (n = 133 [22.3%]), level II (n = 22 [3.6%]), and level III (n = 42 [7.1%]) AKIs based on KDIGO criteria following the evaluation of laboratory data. **Table 1** provides further information on the demographic traits of coronavirus patients.

Table 1. Lists the demographic details of 595 COVID-19 patients.

Variables	Groups	N	%
Age (in years) (Mean = 53 ± 13.55 years)	11-20	12	2.0
	21-30	42	7.0
	31-40	88	14.7
	41-50	119	20.0
	51-60	157	26.3
	> 60	177	29.7
Gender	Male	392	65.8
	Female	203	34.1
Number of days hospitalized (Mean = 6 ± 13.55 days, Range = 3-30 days)	Discharged on the same day	57	9.6
	1-10	423	71.0
	11-15	77	12.9
	16-20	25	4.2
	21-25	3	0.5
	26-30	10	1.6
Comorbidities	Hypertension	244	41.0
	Diabetes mellitus	282	47.3
	Asthma/COPD	38	6.3
	Rheumatoid/osteo arthritis	8	1.3
	Liver and biliary complications [HEP B, C, gallstones, biliary constriction]	31	5.2
	Other CVS disorders like PCI, CABG, CAD, etc.	99	16.7
	Tuberculosis	10	1.7
	Thrombosis/ PE/DVT	3	0.5

AKI classification on admission	No AKI	398	66.8
	Stage I	133	22.3
	Stage II	22	3.6
	Stage III	42	7.1

Clinical symptoms of the patients

Fever (n = 575 [96.6%]), dyspnea (n = 570 [95.8%]), dry cough (n = 449 [75.5%]), and body aches (n = 129 [21.7%]) were the most common signs among the individuals, as indicated in **Table 2**. It was shown that individuals with Stage I AKI had the majority of these signs. On the other hand, individuals with AKI-Stage I were shown to have a considerably greater shock rate. Of the patients on the ventilator, many of them (n = 12 [33.33%]) had undergone dialysis and were from the AKI Stage III ($P < 0.001$). The danger of mortality was shown to be greater for individuals with AKI ($P < 0.001$) than for those without the condition. While most of the patients (n = 305 [51.2%]) were released from the hospital in a stable state, a sizable portion of patients (n = 188 [31.6%]) were discharged with ended.

Table 2. Clinical characteristics and general outcomes of COVID-19 patients by AKI stage

Variables	No AKI (n = 429)	Stage I (n = 151)	Stage II (n = 30)	Stage III (n = 62)	Total	P-value
Symptoms						
Fever	366	132	24	54	576	0.727
SOB	359	133	27	51	570	0.454
Dry cough	283	98	22	46	449	0.478
Body aches	82	22	8	17	129	0.118
Sore throat	50	11	1	7	69	0.262
Anorexia	22	7	0	4	33	0.587
Shock	10	11	2	2	25	0.038*
Loose motion	22	6	3	1	32	0.319
Hemoptysis	1	0	0	0	1	0.904
Fits	1	0	0	0	1	0.908
Flu	15	9	0	1	25	0.251
Mechanical ventilator						
On ventilator	13	7	4	12	36	< 0.001*
Not on ventilator	377	126	17	39	559	
Overall outcome						
Discharged with creatinine above normal	0	26	7	12	45	< 0.001*
Discharged (recovered with hemodialysis done)	0	0	0	1	1	< 0.001*
Discharged stable	273	28	3	1	305	< 0.001*
Discharged and on hemodialysis	0	0	0	1	1	< 0.001*
Expired	84	55	15	34	188	< 0.001*
Still admitted	34	14	1	6	55	< 0.001*

Medication use in patients

During their hospital stays, the majority of the patients were taking several medications. The most frequently prescribed medications among patients were steroidal medicines (n = 544), multiple antibiotics (n = 514), low molecular weight heparin (n = 481), proton-pump inhibitors (n = 413), paracetamol (n = 350), numerous fluids (n = 257), insulin (n = 215), single antibiotic (n = 149), and multivitamins (n = 117). **Figure 1** displays general information on patient medication usage. The most often used antibiotics among patients were meropenem (n = 142), ceftriaxone (n = 322), azithromycin (n = 578), and cefoperazone (n = 156). **Figure 2** displays other antibiotics that patients have used.

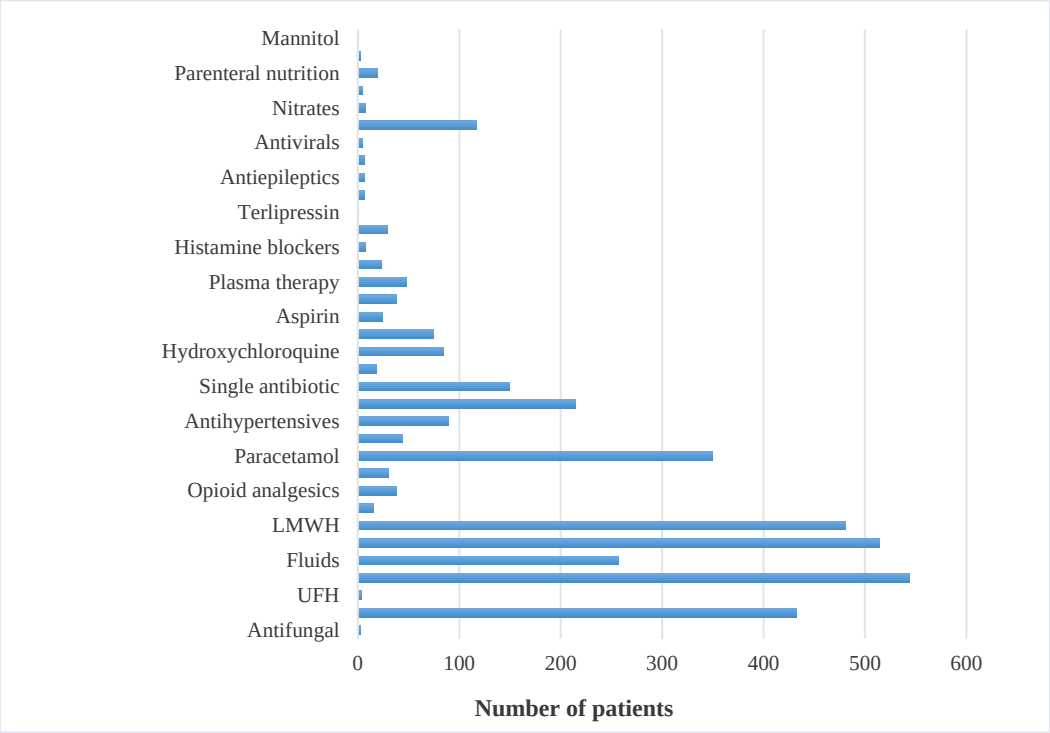

Figure 1. List of different drugs taken by coronavirus patients

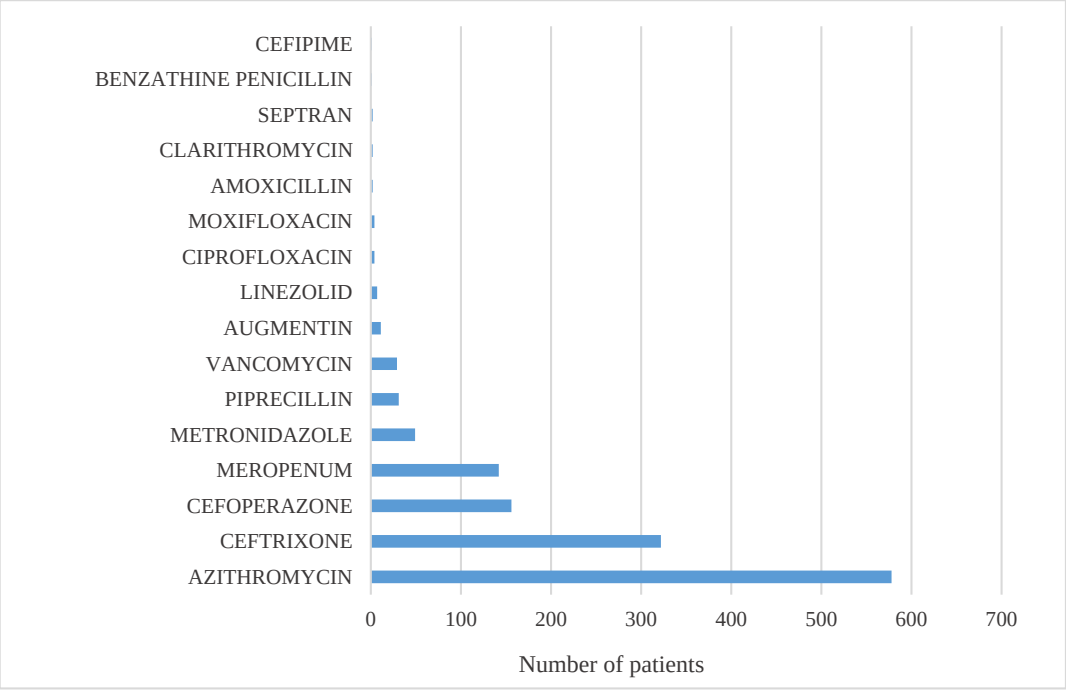

Figure 2. List of different antibiotics taken by coronavirus patients

Laboratory variables of patients in association with AKI

In general, it was shown that coronavirus patients with Stage III AKI had a substantial reduction in the majority of laboratory indicators. Both when the patients were in and when they were discharged, their creatinine levels were quite high. For the majority of patients, serum electrolytes (SE) were within the normal range; nevertheless, as the AKI stages progressed, the mean sodium concentration level progressively rose. As patients' AKI continued to worsen, so did their Complete Blood Count (CBC) readings. The results of the Liver Function Test (LFT) showed that during hospitalization, the levels of liver markers rose, suggesting that many patients had both AKI and liver failure. Furthermore, the Stage III groups were found to have considerably lower blood pressure, heart

rate, and oxygen saturation. **Table 3** provides further information on the laboratory parameters of coronavirus patients who were classified into different phases of AKI.

Table 3. Coronavirus patient laboratory variables

Lab test		N	Mean	Std. deviation	P-value
Creatinine at admission	No AKI	231	1.19	1.765	< 0.001*
	Stage I	96	1.39	0.305	
	Stage II	21	2.02	0.657	
	Stage III	44	3.19	3.012	
Creatinine at discharge	No AKI	286	0.92	0.178	< 0.001*
	Stage I	74	1.28	0.353	
	Stage II	12	1.73	0.723	
	Stage III	16	4.10	3.289	
Urea	No AKI	404	42.12	32.714	< 0.001*
	Stage I	141	63.32	26.292	
	Stage II	30	91.80	52.007	
	Stage III	62	125.03	94.659	
Sodium	No AKI	347	136.77	6.207	0.031*
	Stage I	141	136.43	8.400	
	Stage II	27	138.37	11.287	
	Stage III	61	139.46	9.204	
Potassium	No AKI	346	4.22	0.716	< 0.001*
	Stage I	141	4.50	0.809	
	Stage II	27	4.56	1.226	
	Stage III	61	4.71	0.949	
Chloride	No AKI	346	98.36	8.320	0.024*
	Stage I	141	98.53	7.677	
	Stage II	27	102.24	10.241	
	Stage III	61	100.93	10.104	
RBC	No AKI	201	199.24	107.503	0.258
	Stage I	79	231.85	175.837	
	Stage II	17	211.94	117.363	
	Stage III	41	221.80	118.570	
Hemoglobin	No AKI	347	13.56	7.338	0.664
	Stage I	127	13.18	2.018	
	Stage II	30	12.96	1.902	
	Stage III	61	12.65	2.472	
WBC	No AKI	347	12.00	5.820	0.017*
	Stage I	127	12.86	5.453	
	Stage II	30	15.13	5.383	
	Stage III	61	27.95	1956.464	
Platelets	No AKI	347	245.60	101.251	0.565
	Stage I	127	239.14	111.329	
	Stage II	30	270.00	167.124	
	Stage III	61	241.62	126.158	
Neutrophils	No AKI	334	81.18	44.335	0.876
	Stage I	137	79.58	13.901	
	Stage II	29	83.95	11.029	
	Stage III	61	83.37	10.697	
Lymphocytes	No AKI	354	13.83	11.083	0.030*
	Stage I	127	13.95	12.801	
	Stage II	29	9.37	5.187	
	Stage III	61	8.99	9.240	
PT	No AKI	213	14.49	5.860	0.460
	Stage I	92	15.55	7.013	
	Stage II	21	16.70	5.420	
	Stage III	43	17.23	8.611	
APTT	No AKI	196	30.54	8.468	0.050

	Stage I	82	30.20	6.802	
	Stage II	19	35.89	8.743	
	Stage III	42	34.01	10.235	
INR	No AKI	213	1.29	1.205	0.864
	Stage I	93	1.27	0.598	
	Stage II	21	1.32	0.362	
	Stage III	43	1.42	0.748	
LDH	No AKI	245	617.61	374.315	< 0.001*
	Stage I	79	815.28	503.551	
	Stage II	17	879.94	582.704	
	Stage III	32	868.84	322.295	
ALT	No AKI	360	60.70	66.194	0.020*
	Stage I	137	65.66	69.936	
	Stage II	28	110.25	144.444	
	Stage III	56	117.79	308.278	
ALP	No AKI	354	99.92	66.128	0.050
	Stage I	136	107.86	54.923	
	Stage II	29	117.24	56.319	
	Stage III	56	148.52	238.589	
Total bilirubin	No AKI	354	0.72	1.849	0.334
	Stage I	136	0.78	0.907	
	Stage II	29	1.07	1.353	
	Stage III	56	1.11	2.593	
CRP	No AKI	286	21.77	81.214	0.034*
	Stage I	103	14.47	11.883	
	Stage II	19	13.67	9.064	
	Stage III	39	84.91	398.888	
Ferritin	No AKI	237	1388.28	1765.571	0.842
	Stage I	79	1461.11	1626.054	
	Stage II	14	1481.79	1171.368	
	Stage III	33	1660.50	848.622	
D-DIMER	No AKI	196	10.23	64.287	0.375
	Stage I	90	32.41	179.079	
	Stage II	19	3.60	2.689	
	Stage III	36	13.10	20.053	
Oxygen saturation	No AKI	344	83.0443	12.88103	< 0.001*
	Stage I	138	79.4570	13.32053	
	Stage II	30	72.6667	17.44416	
	Stage III	62	79.2581	14.02683	
Pulse rate in BPM	No AKI	359	94.6425	16.98658	0.001*
	Stage I	138	94.7667	19.81709	
	Stage II	30	103.9000	23.88962	
	Stage III	59	109.1613	74.08064	
Systolic blood pressure	No AKI	361	151.5164	578.40775	0.882
	Stage I	142	122.0867	21.25806	
	Stage II	28	117.6667	20.95699	
	Stage III	62	120.8065	29.16142	
Temperature	No AKI	361	99.0925	4.97653	0.944
	Stage I	142	99.2220	1.30805	
	Stage II	30	99.4000	1.22051	
	Stage III	62	99.3435	1.43705	
Diastolic blood pressure	No AKI	361	77.5316	11.03306	0.163
	Stage I	138	77.9333	15.41361	
	Stage II	32	72.6667	17.20732	
	Stage III	60	75.6935	17.45213	

One-way ANOVA has applied; * P-value < 0.05 is statistically significant

Association of mortality with different variables

The moment COVID-19 patients were 50 years of age or above, the probability of death was 56%, based on numerous linear regression analysis findings (0.566 [0.364–0.873], P = 0.008). In addition, the death rate for AKI patients was 42% [0.418 [0.269–0.632], P < 0.001] compared to those without AKI. Mechanical ventilation also substantially raised the death risk by 9% [0.095 [0.043–0.219], P < 0.001]. A high oxygen saturation level (over 90%) may help lower the risk of death [2.446 [1.550–3.803], P < 0.001]. It was also mentioned that a lower level of lymphocytes and improved diastolic blood pressure control might potentially greatly lower the risk of death.

In comparison to all other drugs, the usage of meropenem increased mortality by 62% (0.625 [0.396–0.982] P = 0.041), intravenous hydrocortisone by 58% (0.582 [0.358–0.943] P = 0.028), hydroxychloroquine by 42% (0.423 [0.245–0.727] P = 0.002), and oral steroids by 16% (0.161 [0.092–0.277] P < 0.001), according to the analysis of the medications used in hospitalized patients. When intravenous dexamethasone was used, nevertheless, the mortality rate was significantly reduced by 96% (1.968 [1.277–3.033], P = 0.002). **Table 4** highlights additional information on the factors that estimate death in COVID-19 patients.

Table 4. Death estimates for coronavirus patients

Variables	β (CI)	Std. error	P-value
Age	0.566 [0.364 – 0.873]	0.213	0.008*
Gender	1.227 [0.791 – 1.903]	0.224	0.361
AKI	0.418 [0.269 – 0.632]	0.204	< 0.001*
On ventilator	0.095 [0.043 – 0.219]	0.408	< 0.001*
Oxygen sat	2.446 [1.550 – 3.803]	0.227	< 0.001*
CPR	0.484 [0.318 – 0.734]	0.213	0.001*
Lymph	1.863 [1.237 -2.807]	0.209	0.003*
Systolic BP	1.195 [0.792- 1.805]	0.210	0.396
Diastolic BP	3.350 [1.906- 5.888]	0.288	< 0.001*
LDH	0.983 [0.663 – 1.457]	0.201	0.930
Oral steroids	0.161 [0.092 – 0.277]	0.277	< 0.001*
Intravenous hydrocortisone	0.582 [0.358 – 0.943]	0.246	0.028*
Intravenous dexamethasone	1.968 [1.277 – 3.033]	0.220	0.002*
Intravenous methylprednisolone	1.118 [0.637 – 1.962]	0.287	0.697
LMWH	1.340 [0.883 – 2.033]	0.213	0.170
Hydroxy Chloroquine	0.423 [0.245 – 0.727]	0.276	0.002*
Azithromycin	0.780 [0.451 – 1.348]	0.279	0.373
Ceftriaxone	1.052 [0.723 – 1.530]	0.191	0.792
Meropenem	0.625 [0.396 – 0.982]	0.231	0.041*

*P-value < 0.05 was deemed of statistical significance when using linear regression; β = standardized beta, CI = confidence interval

In addition to offering a thorough baseline clinical characteristic of the patients, this study had a sufficient sample size. In-depth details about the patient's co-morbidities, COVID-19-related clinical symptoms, hospitalization treatment, and laboratory results following serum electrolyte (SE), complete blood count (CBC), liver function test (LFT), renal function test (RFT), etc., were provided. Using these clinical data, AKI was categorized according to KDIGO standards. To gain a deeper comprehension of the various clinical presentations of coronavirus, several connections were investigated. To document the course of this illness in an infected patient, follow-up was guaranteed.

Among coronavirus patients, fever, dyspnea, and dry cough were among the prevalent signs. The signs were more prevalent in individuals without AKI than in patients with AKI, particularly those in the third stage of the disease, based on the classification of signs based on AKI stages. The findings of the current research are nearly identical to those of different research conducted on a sizable population in China that showed a high frequency of cough, fever, and different signs in a comparable percentage [18]. In our investigation, diabetes mellitus and hypertension were present in the majority of the patients. The reason is due to the high prevalence of infectious illnesses and non-communicable diseases (NCDs) in Pakistan [19]. The results indicate that comorbidities are strongly linked to insufficient results and the severity of coronavirus infection [20]. An investigation with a large sample size found that diabetes is strongly linked with COVID-19 mortality and severity, even though there is very little evidence linking diabetes to disease severity and subsequent death [21]. Although the investigation we conducted

did not examine the connection between diabetes and COVID-19, the high number of diabetic individuals is concerning. Likewise, there is no clinical proof that hypertension and COVID-19 clinical signs are related, but some research has shown that hypertensive individuals' signs have become severe [22].

When comparing individuals with type 3 AKI to those without AKI, several laboratory measures were discovered to be significantly aberrant. Both when the patients were in and when they were discharged, their creatinine levels were quite high. The buildup of COVID-19 in the kidney, which results in renal cell destruction and necrosis, is the mechanism linked to the rise in SARS-CoV-2-related creatinine [8]. Nearly all of the participants in this research had lymphopenia. According to several investigations, the typical feature of a SARS-CoV-2 infection is lymphopenia [23]. In individuals with coronavirus, raised levels of IL-6 and elevated concentrations of tumor necrotic factor-alpha (TNF-alpha) cause lymphocytes to undergo apoptosis [24]. Highly aberrant liver indicators were discovered in our investigation, demonstrating that the coronavirus also damages the host's liver. Several studies have linked elevated levels of alanine transaminase (ALT) and aspartate transaminase (AST) to the indirect effect of COVID-19 causing liver dysfunction [23]. It is necessary to determine whether a patient has multiple organ harm since a significant amount of alkaline phosphatase (ALP) is indicative of an immune system that is overpowered [25].

The patient's oxygen saturation was extremely low, particularly for those in the third stage of AKI. The respiratory system failure, commonly referred to as "silent hypoxemia," is the initial symptom of this illness [26]. Decreased oxygenation is the result of this compromised respiratory system. Within 8–14 days, this frequently results in respiratory failure. Reduced pulmonary diffusion is indicated by a drop in oxygen saturation and an increase in breathing rates. Diffuse alveolar injury is frequently found in histopathological examinations of these individuals [27]. Additionally, this study discovered that individuals with AKI had a progressive decline in oxygen saturation. Furthermore, it was determined that greater than 90% oxygen saturation might considerably lower the risk of death in coronavirus patients after evaluating the relationship between oxygen saturation and mortality. The results of this research are comparable to those of an additional investigation that was carried out on coronavirus patients in China. In that research, it was discovered that when patients got oxygen supplementation at a cut-off value of higher than 90%, high oxygenation significantly reduced the chance of patient mortality [28].

AKI was shown to be strongly linked with death in coronavirus individuals ($P < 0.05$), with a 42% probability of mortality owing to comorbidities. The risk of death was found to be much greater (56%) in elderly adults aged 50 or older. It might be linked to the gradual changes in the morphology of the elderly's lungs, along with muscle atrophy, which causes physiological alterations in pulmonary functions like diminished lung reserve, decreased airway clearance, and impaired defensive barrier-related activities [29-31]. C-reactive protein levels were also much higher in the elderly than in the younger or middle-aged individuals [32]. Computed tomography investigations revealed that multilobe lesions were substantially greater in senior coronavirus patients than in younger individuals [33]. The quickest and most straightforward way to detect lung damage from SARS-CoV-2 and gauge the disease's severity is by computerized tomography. In this investigation, oral steroids were shown to be strongly linked to COVID-19 patient death. The increased patient fatality rate may have been caused by the fact that most patients were using oral steroids. Furthermore, it is well known that steroids impair immunological function, which may serve as a haven for SARS-CoV-2 replication and the ensuing seriousness of the illness [34]. On the other hand, it was discovered that patients who took dexamethasone had a lower chance of dying. The use of dexamethasone in conjunction with normal treatment techniques considerably boosted ventilator-free days and decreased patient mortality, according to a clinical trial done on coronavirus patients [35].

In patients with coronavirus, the virus causes kidney impairment to varying degrees. Blood urea nitrogen (BUN) and creatinine levels, as well as other structural alterations such as localized fibrosis, epithelial cell necrosis with interstitial hyperemia, and renal parenchymal edema or inflammation, can be used to assess this [36, 37]. COVID-19-related acute kidney damage is linked to increased production of pro-inflammatory substances due to the viral infection [38]. Particularly in older individuals or those with concomitant illnesses like diabetes, cancer, or cardiovascular disease, norepinephrine and other medications, such as antiviral medicines, antibiotics, and NSAIDs, can also result in acute renal damage [36]. The identification of viral infection is linked to the diagnosis of AKI in coronavirus patients [39]. CRRT is typically employed to treat COVID-19-associated AKI in conjunction with other supportive medications that combat viral infection. Additionally, CRRT is significant because it may lessen inflammatory cytokine excess and, if utilized early in AKI, may lower the death rate in patients who are severely sick [40]. In our research, the death rate for coronavirus patients with AKI was greater than that of those without AKI, and those who are released from the hospital do not fully recover their kidneys.

AKI in coronavirus patients should be diagnosed as soon as possible since it can raise death rates and cause chronic renal disease in patients who are released from the hospital [41].

There is currently no known cure for AKI, although early diagnosis and treatment of underlying diseases linked to AKI may lower the risk of AKI in coronavirus patients. Avoiding different nephrotoxic medications and treating hypoxemic situations are two examples of these treatment strategies. For a high-risk patient population, early hemodynamic and blood volume optimization is necessary to guarantee appropriate and efficient renal perfusion pressures. On the other hand, the utilization of Continuous Renal Replacement Therapy is the next sensible course of action if the patient does not react to the traditional therapy options. According to the data that this investigation provides, physicians and other decision-makers will be able to make wise clinical judgments. It will aid in comprehending the final development of this epidemic. Further research on this subject and the exploration of other results are also made possible by this investigation. It will act as a focal point around which further research might expand its purview.

Conclusion

Given that hospitalized coronavirus patients are more likely to experience acute kidney damage (AKI), which can worsen the disease and potentially cause mortality, renal involvement throughout the illness is concerning. According to our research, when COVID-19 patients go through AKI phases, the clinical signs become severe. The mortality threat facing COVID-19-afflicted AKI patients may be influenced by several variables. Although there is no specific treatment for AKI, a clinician's main objective is to prevent the use of nephrotoxic medications while a patient is in the hospital and to keep appropriate oxygen saturation levels to prevent the establishment of AKI or the exacerbation of AKI that has already occurred in coronavirus patients. For patients who do not respond to conventional treatment of AKI, CRRT may be the final resort.

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