Risk Factors and Outcomes of Acute Kidney Injury in Critically Ill Patients With Coronavirus Disease 2019

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Research

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Abstract

Background: Coronavirus disease 2019 (COVID-19) has emerged as a major global health threat with a great number of deaths worldwide. Acute kidney injury (AKI) is a common complication in patients admitted to the intensive care unit. We aimed to assess the incidence, risk factors and in-hospital outcomes of AKI in COVID-19 patients admitted to intensive care unit.

Methods: we conducted a retrospective observational study in intensive care unit of Tongji hospital, which was assigned responsibility for the treatments of severe COVID-19 patients by Wuhan government. The AKI was defined and staged based on Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Mild AKI was defined as stage 1, and severe AKI was defined as stage 2 or stage 3. We used logistic regression analysis to evaluate AKI risk factors and the association between AKI and in-hospital mortality.

Results: A total of 150 patients with COVID-19 were included in our study. The median age of patients was 70 (interquartile range, 60-80) years and 62.7% were male. 70 (46.7%) patients developed AKI during hospitalization, corresponding to the 17.3% in stage 1 and 9.3% in stage 2 and 20.0% in stage 3, respectively. Compared to patients without AKI, patients with AKI had higher proportion of mechanical ventilation mortality and higher in-hospital mortality. 95.5% patients with severe AKI received mechanical ventilation and in-hospital mortality was up to 79.5%. Severe AKI was independently associated with high in-hospital mortality (OR: 4.30; 95% CI: 1.83-10.10). Logistic regression analysis demonstrated that high serum interleukin-6 (OR: 2.54; 95%CI: 1.00-6.42) and interleukin-10 (OR: 3.02; 95%CI: 1.17-7.82) were risk factors for severe AKI development.

Conclusions: severe AKI was associated with high in-hospital mortality and inflammatory response may play a role in AKI development in critically ill patients with COVID-19.

Background

Coronavirus disease 2019 (COVID-19) is a respiratory tract infection caused by a newly emergent coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; previously known as 2019-nCoV) [1, 2]. The COVID-19 has swept into more than 100 countries and more than 2 million cases have been confirmed within a few months. While most people with COVID-19 develop mild or uncomplicated illness, it is reported that 14%~16% develop severe disease requiring hospitalization and 5% ~ 27% hospitalized patients require admission to an intensive care unit [3–6]. Thus, the COVID-19 pandemic significantly increased the burden of critical illness globally.

Studies showed that SARS-CoV-2 uses angiotensin converting enzyme II (ACE2) as cell entry receptor [7], which is highly expressed in lung tissues but also in human kidneys [8]. This suggests that lungs were not the only organs involved and the kidney would also be possible target in COVID-19. Acute kidney injury (AKI) is a common, serious complication and is associated with poor outcome in critically ill patients [9, 10]. Previous studies reported that AKI developed in 0.5–5.1% patients with COVID-19 [4, 11, 12]. However, details of the epidemiological characteristics and outcome of AKI in critically ill patients with COVID-19 have not yet been well described.

We conduct a retrospective study of COVID-19 patients admitted to intensive care unit to assess the incidence and risk factors of AKI and its impact on in-hospital mortality.

Results

Patients characteristics

A total of 150 patients with COVID-19 admitted to the intensive care unit were included in our study. Demographic and clinical characteristics were summarized in Table 1. The median age of patients was 70 (IQR, 60–80) years and most were male (62.7%). 58.7% patients had at least one comorbidity, the most common were hypertension (43.3%) and diabetes (24.7%). The median APACHE II score of all patients was 9 (IQR, 6–14), and the median of the SOFA score was 3 (IQR, 2–5).

Table 1
Demographic characteristics, laboratory data and outcomes of critically ill COVID-19 patients

	All patients N= 150	Non-AKI N= 80	Mild AKI N= 26	Severe AKI N = 44
Clinical characteristics				
Age, years	70 (60–80)	68 (55–81)	70 (58–84)	72 (67–78)
Male patients, %	94/150 (62.7)	47/80 (58.8)	17/26 (65.4)	30/44 (68.2)
Any comorbidity, %	88/150 (58.7)	41/80 (51.3)	19/26 (73.1)	28/44 (63.6)
Chronic kidney disease, %	2/150 (1.3)	0/80 (0)	2/26 (7.7)	0/44 (0)
Chronic lung disease, %	12/150 (8.0)	4/80 (5.0)	4/26 (15.4)	4/44 (9.1)
Diabetes, %	37/150 (24.7)	17/80 (21.3)	7/26 (26.9)	13/44 (29.5)
Hypertension, %	65/150 (43.3)	30/80 (37.5)	13/26 (50.0)	22/44 (50)
Tumor, %	8/150 (5.3)	5/80 (6.3)	1/26 (3.8)	2/44 (4.5)
APACHE II score	9 (6–14)	10 (7–16)	11 (7–16)	7 (6–18)
SOFA score	3 (2–5)	3 (2–4)	4 (2–7)	3 (2–5)
Laboratory data				
Leukocyte count, × 10 ⁹ /L	9 (6–13)	10 (6–14)	11 (5–16)	8 (7–14)
Lymphocyte count, × 10 ⁹ /L	0.7 (0.5–0.9)	0.7 (0.5–0.9)	0.5 (0.4–0.9)	0.6 (0.4–0.8)
Platelet count, × 10 ⁹ /L	161 (120–250)	169 (132–281)	137 (98–157)	166 (122–250)
Total bilirubin, μmol/L	12 (8–19)	12 (8–19)	13 (7–22)	12 (8–20)
D-dimer, mg/L	2.9 (1.3–14.9)	2.7 (1.3–12.6)	4.6 (1.4–21.0)	3.9 (1.3–15.0)
Procalcitonin, ng/mL	0.2 (0.1–0.4)	0.2 (0.1–0.5)	0.2 (0.1–0.3)	0.2 (0.1–0.4)
hs-CRP, mg/L	78 (41–142)	76 (19–154)	56 (16–106)	93 (63–142)
Lactose dehydrogenase, U/L	460 (315–602)	430 (307–512)	496 (259–802)	479 (382–745)
Serum creatinine, μmol/L	76 (58–94)	71 (52–94)	87 (57–126)	76 (63–106)
eGFR, ml/min/1.73 m ²	79 (63–96)	83 (73–98)	78 (44–97)	79 (55–93)
Interleukin-6, pg/ml	44 (19–118)	40 (17–115)	26 (12–75)	75 (32–138)
Interleukin-8, pg/ml	24 (15–52)	26 (15–49)	20 (15–62)	42 (21–74)
Interleukin-10, pg/ml	9 (5–15)	9 (5–13)	9 (5–24)	12 (7–16)
Interleukin-2 receptor, U/ml	1049 (674–1287)	995 (601–1476)	918 (527–1075)	1095 (919–1266)
Tumor necrosis factor α, pg/ml	10 (7–13)	10 (7–13)	9 (7–15)	11 (8–16)
Outcomes				
Mechanical ventilation, %	130/150 (86.7)	65/80 (81.3)	23/26 (88.5)	42/44 (95.5)
Non-invasive, %	114/150 (76.0)	57/80 (71.3)	21/26 (80.8)	36/44 (81.8)
Invasive, %	81/150 (54.0)	37/80 (46.3)	15/26 (57.7)	29/44 (65.9)
Hospital length of stay, days	19 (12–38)	20 (10–37)	20 (16–44)	38 (18–56)
14-day mortality, %	49/150 (32.7)	24/80 (30.0)	6/26 (23.1)	19/44 (43.2)
28-day mortality, %	76/150 (50.7)	33/80 (41.3)	14/26 (53.8)	29/44 (65.9)
In-hospital mortality, %	88/150 (58.7)	38/80 (47.5)	15/26 (57.7)	35/44 (79.5)
AKI, acute kidney injury; COVID-19, coronavirus disease 2019; APACHE, acute physiologic and chronic health evaluation; SOFA, sequential organ failure assessment; hs-CRP, high-sensitivity c-reactive protein; eGFR, estimated glomerular filtration rate.				

Aki And In-hospital Outcomes

During hospitalization, AKI developed in 70 (46.7%) patients, corresponding to the 17.3% mild (stage 1) and 28.1% severe AKI (9.3% in stage 2 and 20.0% in stage 3, respectively). The median duration from admission to AKI occurrence was 11 days (IQR, 6–19).

The 14-day mortality, 28-day mortality and in-hospital mortality of critically ill patients with COVID-19 were 32.7%, 50.7% and 58.7%, respectively. The median of hospital length of stay was 19 days (IQR, 12–38). Compared to patients without AKI, patients with AKI had higher proportion of mechanical ventilation mortality, higher in-hospital mortality and longer hospital length of stay. Notably, 95.5% patients with severe AKI received mechanical ventilation and a in-hospital mortality was up to 79.5%. The Kaplan-Meier curve showed a diversion of the survival rates between severe AKI and non-AKI, while no significant difference was observed in mild AKI and non-AKI (Fig. 1). Likewise, logistic regression analysis demonstrated that only severe AKI was associated with high 28-day mortality (OR: 2.75; 95% CI: 1.28–5.92) and in-hospital mortality (OR: 4.30; 95% CI: 1.83–10.10), and the association remained significant after adjustment for age, sex, SOFA score, lymphocyte count and D-dimer (Table 2).

Table 2
Association of AKI and in-hospital mortality in critically ill COVID-19 patients

	Unadjusted		Adjusted	
	OR (95% CI)	P value	OR (95% CI)	P value
14-days mortality				
Non-AKI	1.00 (reference)		1.00 (reference)	
Mild AKI	0.7 (0.25, 1.96)	0.497	0.58 (0.18, 1.81)	0.347
Severe AKI	1.77 (0.83, 3.81)	0.142	1.65 (0.69, 3.96)	0.258
28-days mortality				
Non-AKI	1.00 (reference)		1.00 (reference)	
Mild AKI	1.66 (0.68, 4.05)	0.264	1.8 (0.69, 4.72)	0.229
Severe AKI	2.75 (1.28, 5.92)	0.010	2.57 (1.11, 5.93)	0.027
In-hospital mortality				
Non-AKI	1.00 (reference)		1.00 (reference)	
Mild AKI	1.51 (0.62, 3.68)	0.368	1.58 (0.59, 4.24)	0.36
Severe AKI	4.30 (1.83, 10.10)	< 0.001	4.34 (1.68, 11.26)	0.003
AKI, acute kidney injury; COVID-19, coronavirus disease 2019; OR, odds ratio; 95% CI, 95% confidence interval; ORs were obtained in multivariate logistic regression models after adjustment for age, sex, sequential organ failure assessment (SOFA) score, lymphocyte count and D-dimer.				

Risk Factors For Aki

Compared to patients without AKI, patients with AKI had a higher proportion of comorbidities, including diabetes and hypertension. In addition, patients with AKI showed lower lymphocyte count, higher D-dimer, high-sensitivity c-reactive protein and lactose dehydrogenase (Table 1). We further analyzed the level of serum cytokines, which was available in 101 patients in our study, moreover, nearly all clinical characteristics, laboratory data and in-hospital mortality were similar in 101 patients with available data on cytokines and the 49 patients with missing data on cytokines (Supplementary table 1). We found that the cytokines on admission were similar between mild AKI and non-AKI, while interleukin (IL)-6, IL8, IL10 and IL2R on admission was notably higher in patients with severe AKI.

Given that the in-hospital mortality was remarkably increased in patients with severe AKI but not in mild AKI, we further to explore the risk factors for severe AKI in patients with COVID-19 and admitted to the intensive care unit. Among the clinical characteristics, common laboratory data and cytokines, we found that only higher IL6 (OR: 2.54; 95%CI: 1.00-6.42) and IL10 (OR: 3.02; 95%CI: 1.17–7.82) were associated with severe AKI development in univariate logistic regression (Table 3). In addition, we described the trends of cytokines in patients with AKI. We found that cytokines were markedly increased, and patients with severe AKI tended to have a higher level of IL6 and IL10 than patients with mild AKI when AKI occurred.

Table 3
Univariable logistic analysis of risk factors for severe AKI in critically ill COVID-19 patients

Variables	OR	95% CI	P value
Age (≥ 65 vs < 65)	1.68	0.78–3.63	0.186
Sex (male vs female)	1.41	0.67–2.96	0.369
Chronic lung disease (yes vs no)	1.22	0.35–4.30	0.751
Diabetes (yes vs no)	1.43	0.65–3.16	0.373
Hypertension (yes vs no)	1.47	0.72–2.97	0.290
APACHE II score	0.97	0.92–1.03	0.325
SOFA score	1.02	0.90–1.15	0.802
Leukocyte count $\times 10^9/L$ (> 10 vs ≤ 10)	0.98	0.48–2.02	0.963
Lymphocyte count $\times 10^9/L$ (< 1.0 vs ≥ 1.0)	1.01	0.40–2.51	0.990
Platelet count $\times 10^9/L$ (< 100 vs ≥ 100)	0.85	0.33–2.19	0.730
Total bilirubin, $\mu\text{mol/L}$ (> 20 vs ≤ 20)	1.04	0.43–2.50	0.929
D-dimer, mg/L (≥ 0.5 vs < 0.5)	1.17	0.23–6.05	0.853
Procalcitonin, ng/mL (≥ 0.5 vs < 0.5)	1.15	0.43–3.10	0.782
Lactose dehydrogenase, U/L (> 245 vs ≤ 245)	2.05	0.65–6.43	0.221
Interleukin-6, pg/mL (> 44 vs ≤ 44)	2.54	1.00–6.42	0.049
Interleukin-8, pg/mL (> 24 vs ≤ 24)	1.83	0.74–4.55	0.194
Interleukin-10, pg/mL (> 9 vs ≤ 9)	3.02	1.17–7.82	0.022
Interleukin-2 receptor, U/mL (> 1049 vs ≤ 1049)	1.93	0.78–4.80	0.158
Tumor necrosis factor α , pg/mL (> 10 vs ≤ 10)	1.73	0.70–4.31	0.237
AKI, acute kidney injury; COVID-19, coronavirus disease 2019; APACHE, acute physiologic and chronic health evaluation; SOFA, sequential organ failure assessment.			

Medications

We summarized the medications during hospitalization in Table 4. Most critically ill patients with COVID-19 received treatment with antibiotics (93.3%), antiviral (74.0%) and glucocorticoid (73.3%). The proportion of treatment with anti-diabetes and diuretics was higher in patients with AKI. In addition, few patients (6.0%) received angiotensin-converting-enzyme inhibitor or angiotensin receptor blocker.

Table 4
Treatment during hospitalization in critically ill COVID-19 patients with COVID-19

	All patients N = 150	Non-AKI N = 80	Mild AKI N = 26	Severe AKI N = 44
ACEI or ARB, %	9/150 (6.0)	5/80 (6.3)	3/26 (11.5)	1/44 (2.3)
Antibiotics, %	140/150 (93.3)	73/80 (91.3)	25/26 (96.2)	42/44 (95.5)
Antiviral, %	111/150 (74.0)	61/80 (76.3)	19/26 (73.1)	31/44 (70.5)
Antidiabetic, %	50/150 (33.3)	22/80 (27.5)	9/26 (34.6)	19/44 (43.2)
Diuretic, %	96/150 (64.0)	43/80 (53.8)	20/26 (76.9)	33/44 (75.0)
Glucocorticoid, %	110/150 (73.3)	59/80 (73.8)	15/26 (57.7)	36/44 (81.8)
AKI, acute kidney injury; COVID-19, coronavirus disease 2019; ACEI, angiotensin-converting-enzyme inhibitor; ARB, angiotensin receptor blocker.				

Discussion

In this cohort of 150 critically ill patients with COVID-19, about half (46.7%) patients developed AKI during hospitalization, and most of whom (62.9%) were severe AKI. Patients with severe AKI had extremely high in-hospital mortality (79.5%). After adjustment for confounders, severe AKI was

independently associated with in-hospital mortality. In addition, patients with high a level of serum cytokines were more likely to develop AKI during hospitalization.

AKI occurred frequently in critically ill patients with COVID-19. However, previous studies reported that the incidence varies from 8.9% ~ 28.8% [12–14], which was much lower than 46.7% reported in our study. This variation probably results from differences in the case number, illness severity, and coexisting conditions. Another major reason was that we retrospectively recorded the AKI occurrence during the entire hospitalization, and the previous studies were cross-sectional [14] or had short follow-up duration [12, 13]. Our result demonstrated that the median of hospital length of stay was 19 days and the median duration from admission to AKI occurrence was 11 days, which suggested that some AKI cases were not able to be detected in the early stage of hospitalization. In addition, it is reported that the incidence of AKI in critically ill patients was 58.0% in MERS [15] and 61% in septic shock patients [16]. Hence, the actual incidence might be higher considering that some AKI might be missed in our study. In accordance with the recent reports on the outcomes of COVID-19 patients, 5 ~ 26% patients will be admitted to the intensive care unit during hospitalization [4–6]. Given that the number of COVID-19 cases is enormous and growing rapidly, there would be a substantial burden of medical care from AKI during the treatment of COVID-19. Therefore, plans should be made at local and regional levels for how to prepare and manage the potential surge in the need for critical care resources for AKI, including renal replacement treatment.

In our study, patients with AKI demonstrated an extremely poor prognosis. Of note, the mortality of severe AKI was 79.5%, which was much higher than those who developed ARDS [17]. Likewise, the reported mortality of AKI in patients with SARS was up to 91.7% [18]. One possible explanation is the development of AKI may indicate the involvement of multiple organs dysfunction. In addition, AKI may worsen lung injury via several mechanisms including impaired fluid excretion, direct capillary endothelial injury, and exacerbating inflammatory response [19]. Hence, in order to detect the AKI timely and reduce the deaths, closer monitoring of serum creatinine and urine output is needed in the management of critically ill patients with COVID-19. Moreover, our findings showed that only severe AKI but not mild AKI was associated with in-hospital mortality. Therefore, a clear escalation plan on individualized treatment should be made depending on the severity of AKI.

The increased cytokines might be a predictor of AKI development in critically ill patients with COVID-19. Accumulating evidence suggests that cytokine release syndrome plays a role in severe COVID-19 [20]. It is reported that an inflammation-related cytokine profile, characterized by IL2, IL2R, IL-8, IL-10 and tumor necrosis factor- α (TNF α), were associated with COVID-19 disease severity [21, 22]. Our study was the first to confirm the high level of IL6 and IL10 were associated with high risk of AKI development in critically ill patients with COVID-19. In addition, we found that cytokines were increased remarkably when AKI occurred. Although kidney tissues from postmortems showed that AKI might be attributed to reduced renal perfusion and widespread tubular necrosis [23], our results suggested more complex and subtle mechanisms of immune-mediated microvascular and tubular dysfunction was involved in AKI in critically ill patients with COVID-19. Moreover, timely inhibiting the excessive inflammatory response in its early stage through such means as immunomodulators and cytokine antagonists, as well as continuous renal replacement therapy, might be breakthroughs in the treatment of critically ill patients with COVID-19.

Some conventional risk factors for AKI in critically ill patients, including age and severity of disease assessed by SOFA score, were not identified in our study. One possible explanation is that Tongji Hospital was one of the designated hospitals for COVID-19 patients and the study population was relatively homogeneous with respect to age and disease severity. Most patients included in our study were the elderly, with median age of 70. And almost all patients were admitted to intensive care unit due to the respiratory failure, thus, the SOFA score on admission was lower than studies included patients with different causes and multiple organs dysfunction [24]. In addition, our study was performed in single center and the generalizability of the study is limited by the small sample size. Further study in a larger cohort is needed to gain a better understanding of risk factors for AKI in critically ill COVID-19 patients.

We also summarized the medications during the hospitalization in critically ill patients with COVID-19. Our result showed that most patients have been treated with antibiotics and antivirus, which might induce and worsen AKI. Thus, the appropriate dosage of antibiotics and antivirus should be considered on basis of kidney function in clinical practice. In addition, we found that patients with AKI had high proportion of treatment with diuretics, which are often frequently applied to control hypervolemia and prevent pulmonary edema in critically ill patients [25]. Previous studies showed the use of diuretics in critically ill patients with acute kidney injury was associated with an increased risk of death and nonrecovery of kidney function [26]. Therefore, diuretics should be used with caution in management of critically ill patients with COVID-19. However, due to the small number of patients and the bias in different therapy of COVID-19 patients, causal relationship between medications and AKI in critically ill COVID-19 patients remains undetermined.

Our study has several limitations. First, an accurate baseline serum creatinine and urine output was not available, which may have led to an underestimation of AKI or erroneous associations. Second, our study has a retrospective observational design with its inherent biases and potential for unmeasured confounding variables. Finally, this investigation was conducted in a medical intensive care unit of a tertiary referral center, potentially limiting the generalizability of our study results.

Conclusions

In conclusion, our study involving critically ill patients with COVID-19 showed that AKI is common and carries extremely high in-hospital mortality. Inflammatory response may play a role in AKI development. Clinicians should increase their awareness of AKI in critically ill patients with COVID-19.

Methods

Participants

This retrospective observational study was done at Tongji hospital, which located in Wuhan, Hubei Province, the major endemic area of COVID-19. Tongji hospital was assigned responsibility for the treatments of severe COVID-19 patients by Wuhan government on January 31, 2020. We retrospectively analyzed patients from Jan 28 to March 29, 2020, who had been diagnosed with COVID-19 according to the guidance provided by the Chinese National Health Commission (version 7.0) [27], and who were admitted to intensive care unit.

We excluded patients without serum creatinine on admission. Pediatric patients and patients with a history of maintenance dialysis or renal transplantation were also excluded from the study. Clinical outcomes were monitored up to April 23, 2020, the final date of follow-up.

Data Collection

The demographic characteristics, laboratory data and outcome were extracted from electronic medical records. The admission data of these patients were collected. Laboratory data consisted of complete blood count, liver and renal function, D-dimer, high-sensitivity C-reactive protein, procalcitonin, lactate dehydrogenase and cytokines. The data were reviewed by a trained team of physicians. eGFR was calculated with the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [28]. The Sequential Organ Failure Assessment (SOFA) score [29] and Acute Physiologic and Chronic Health Evaluation (APACHE) II score [30] were calculated on admission.

Definition

AKI was defined as an increase in serum creatinine by 0.3 mg/dL within 48 hours or a 50% increase in serum creatinine from baseline within 7 days according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria [31]. Urine output criteria were not used to define AKI. Baseline serum creatinine was defined as the serum creatinine value on admission. The date of AKI onset was defined as the earliest day of a serum creatinine change meeting KDIGO criteria. The maximum stage of AKI was determined using the peak serum creatinine level after AKI detection, with increases of 1.5–1.9, 2.0–2.9 and ≥ 3 times baseline being defined as AKI stage 1, 2 and 3, respectively. Mild AKI was defined as stage 1, and severe AKI was defined as stage 2 or stage 3.

Statistical Analysis

Categorical variables were summarized as number (%), and continuous variables were expressed as median with interquartile range (IQR). Mann Whitney U test was used for continuous variables, and Chi-square test or Fisher's exact test for categorical variables as appropriate. Kaplan–Meier survival curves and log-rank tests were used to describe the effect of the stage of acute kidney injury on in-hospital mortality. The association of AKI and in-hospital mortality was assessed with logistic regression analysis. Adjusted variables were chosen on the basis of previous findings. Age, lymphocyte count, SOFA score and D-dimer were reported to be associated with in-hospital mortality [6, 17, 32], in addition, previous studies have shown that the mortality was higher in male patients [33]. To explore the risk factors associated with severe AKI, univariable logistic regression models were used. We excluded variables if the number of events was too small to estimate odds ratios. No imputation was made for missing data. Categorization was performed for continuous variables, as it is easier to interpret and also the simplicity of reporting results. For common laboratory values, we used the cut-off points which were widely recognized and adopted in clinical practice. The median was used as cut-off points for inflammation-related cytokines. Statistical analyses were performed using SPSS, version 22.0, with statistical significance set at 2-sided $P < 0.05$.

Abbreviations

COVID19

coronavirus disease 2019; AKI:acute kidney injury; SARS-CoV-2:severe acute respiratory syndrome coronavirus 2; ACE2 angiotensin converting enzyme II; CKD-EPI:Chronic Kidney Disease Epidemiology Collaboration; APACHE:Acute Physiologic and Chronic Health Evaluation; SOFA:Sequential Organ Failure Assessment; KDIGO:Kidney Disease:Improving Global Outcomes; IQR:interquartile range; IL:interleukin; TNF α :tumor necrosis factor- α .

Declarations

Ethics approval and consent to participate:

The study protocol and waived written informed consent was approved by the Medical Ethics Committee of Tongji Hospital (No. TJ-C20200132).

Consent for publication:

Not applicable.

Availability of data and material :

The data used and analyzed during the current study are available from the corresponding author on reasonable request

Competing interests:

Authors have disclosed no conflicts of interest

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Authors' contributions

G.X., S.G. designed the study. Y.C., N.Z., R.L., M.Z., Z.W., L.D., J.L. R.Z. and Y.Y. collected the data, prepared the figures and tables. Y.C. and S.G. contributed analytical tools. Y.C. and S.G. wrote the paper. S.G. and G.X. conceived the project and supervised and coordinated all the work.

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Figures

Figure 1

Survival curves according to severity of acute kidney injury in critically ill COVID-19 patients

Figure 2

Trends of serum cytokines in critically ill COVID-19 patients with acute kidney injury. Data was expressed as median with interquartile range.