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Acute kidney injury in patients with severe and critical course of COVID-19

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Acute kidney injury (AKI), characterized by a sudden decline in kidney function, is typically identified by an increase in serum creatinine levels and/or a decrease in urine output. It is a common complication among critically ill COVID-19 patients, with studies reporting an incidence rate of 20–40% in individuals admitted to intensive care units (ICU). The development of AKI in COVID-19 is influenced by several factors, including systemic inflammatory response, sepsis, disruptions in renin-angiotensin-aldosterone system homeostasis, rhabdomyolysis, and potential direct viral injury to the kidneys. Early identification of kidney-related complications in COVID-19 patients is essential for reducing morbidity and mortality in hospitalized patients.
The objective: **to investigate the incidence, risk factors, clinical characteristics, and outcomes of AKI in patients with severe and critical COVID-19.**
Materials and methods**. A retrospective analysis of 1,311 hospitalized patients in Kyiv City Clinical Hospital No. 17 with COVID-19 (September 2020 – December 2021) identified 252 (19.2%) meeting severe or critical disease criteria. After applying exclusion criteria, 221 patients (87.7%) were included in the final analysis, comprising 113 (51.1%) women and 108 (48.9%) men. Among them, 78 (35.3%) developed AKI during hospitalization. AKI severity was classified according to the KDIGO (Kidney Disease: Improving Global Outcomes) criteria based on serum creatinine levels, with three stages indicating progressive renal dysfunction. According to the analysis of medical records, demographic and clinical data, course of the disease, and laboratory findings were analyzed.**
Results**. Among 221 analyzed patients, 176 (79.6%) were initially admitted to other departments before being transferred to the ICU, while 45 (20.4%) were directly hospitalized from the emergency department. AKI was present in 78 (35.3%) patients and was more common in older individuals (p = 0.048). While the prevalence of cardiovascular disease and diabetes mellitus did not significantly differ between AKI and non-AKI groups, chronic kidney disease (CKD) was markedly higher in AKI patients (20.5% vs 0.7%, p < 0.0001). Laboratory findings showed higher creatinine levels (105 vs 81 μmol/L, p < 0.0001), lower estimated glomerular filtration rate (70.5 vs 84.1 mL/min/1.73 m², p < 0.0001) and higher IL-6 (9.8 (5.2; 38.1) vs 8.4 (4.3; 36.1) pg/mL, p < 0.0001) in AKI patients, whereas hemoglobin, leukocytes, CRP, and ferritin levels did not significantly differ. Nephrotoxic medication use was significantly more common in AKI patients (65.4% vs 32.9%, p < 0.0001), while mechanical ventilation rates were similar (44.9% vs 46.2%, p = 0.854). Although ICU stays were longer for AKI patients (8.9 vs 6.3 days, p = 0.012), overall mortality did not significantly differ between AKI and non-AKI groups (39.7% vs 42.7%, p = 0.959). AKI most often developed upon ICU admission and Day 1, followed by a gradual decline, with a secondary peak after Day 7. The analysis of risk factors associated with AKI revealed that patients with CKD, with an odds ratio (OR) of 36.86 (95% confidence interval (CI) [4.78–284.1], p < 0.0001), the use of nephrotoxic medications was strongly associated with AKI (OR = 3.89; 95% CI [2.22–6.82], p < 0.0001). Older patients also had a significantly increased risk of AKI compared to younger age groups (OR = 2.56; 95% CI [1.32–4.97], p = 0.048).**
Conclusions**. AKI was a common complication, with older age and pre-existing CKD identified as key risk factors. The use of nephrotoxic medications also showed a strong association with AKI development. While AKI patients had longer ICU stays, overall mortality did not differ significantly between those with and without AKI. These findings emphasize the importance of early risk assessment and careful management of nephrotoxic exposures in critically ill patients.**
Keywords: *acute kidney injury, chronic kidney disease, nephrotoxic medications, risk factors, mortality.*

Гостре ураження нирок у пацієнтів з тяжким та критичним перебігом COVID-19

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Гостре ураження нирок (ГУН) характеризується раптовим зниженням функції нирок, зазвичай відзначається підвищенням рівня креатиніну в сироватці крові та/або зниженням діурезу. Це поширене ускладнення серед тяжкохворих пацієнтів із COVID-19, при цьому дослідження повідомляють про частоту виникнення 20–40% в осіб, які були госпіталізовані до відділення інтенсивної терапії (ВІТ). Розвиток ГУН при COVID-19 залежить від багатьох факторів, зокрема наявності системної запальної реакції, сепсису, порушення гомеостазу ренін-ангіотензин-альдостеронової системи, рабдоміолізу й потенційного прямого вірусного пошкодження. Раннє виявлення ускладнень, пов'язаних із нирками, у пацієнтів із COVID-19 має важливе значення для зниження інвалідизації та смертності серед госпіталізованих хворих.
Мета дослідження: дослідження частоти виникнення, факторів ризику, клінічних характеристик і наслідків ГУН у пацієнтів із тяжким та критичним перебігом COVID-19.
Матеріали та методи. Проведено ретроспективний аналіз 1311 госпіталізованих пацієнтів із COVID-19 в Київській міській клінічній лікарні № 17 (вересень 2020 р. – грудень 2021 р.), з яких 252 (19,2%) відповідали критеріям тяжкого або критичного перебігу захворювання. Після застосування критеріїв виключення до остаточного аналізу відібрано 221 пацієнта (87,7%), серед яких – 113 (51,1%) жінок та 108 (48,9%) чоловіків. Серед них у 78 (35,3%) випадках під час госпіталізації розвинулося ГУН. Тяжкість ГУН класифікувалася відповідно до критеріїв KDIGO (Kidney Disease: Improving Global Outcomes) на основі

рівня креатиніну в сироватці крові, при цьому три стадії відповідали прогресивному порушенню ниркової функції. За результатами аналізу проаналізовано демографічні й клінічні характеристики, перебіг захворювання та лабораторні показники.
Результати. Серед 221 проаналізованого пацієнта 176 (79,6%) були первинно госпіталізовані до інших відділень із подальшим переведенням у ВІТ, тоді як 45 (20,4%) було госпіталізовано безпосередньо з приймального відділення. ГУН виявлено у 78 (35,3%) хворих, і воно частіше відзначалося серед осіб похилого віку (p = 0,048). Поширеність серцево-судинних захворювань і цукрового діабету істотно не відрізнялася між групами з ГУН і без нього, тоді як хронічна хвороба нирок (ХХН) значно частіше діагностувалася у пацієнтів із ГУН (20,5% проти 0,7%, p < 0,0001). Лабораторні показники продемонстрували вищий рівень креатиніну (105 проти 81 мкмоль/л, p < 0,0001), нижчу швидкість клубочкової фільтрації (70,5 проти 84,1 мл/хв/1,73 м^2, p < 0,0001) та вищий рівень IL-6 (9,8 (5,2; 38,1) проти 8,4 (4,3; 36,1) пг/мл, p < 0,0001) у пацієнтів із ГУН. При цьому рівні гемоглобіну, лейкоцитів, С-реактивного білка та феритину суттєво не відрізнялися. Застосування нефротоксичних препаратів було значно частішим у пацієнтів із ГУН (65,4% проти 32,9%, p < 0,0001), тоді як показники використання штучної вентиляції легень були подібними (44,9% проти 46,2%, p = 0,854). Хоча перебування у ВІТ було тривалішим у пацієнтів із ГУН (8,9 проти 6,3 дня, p = 0,012), показник загальної смертності істотно не відрізнявся між групами з ГУН та без ГУН (39,7% проти 42,7%, p = 0,959). ГУН найчастіше було вже наявним при надходженні до ВІТ або виникало у 1-й день госпіталізації, з поступовим зниженням частоти нових випадків і вторинним піком після 7-го дня. Аналіз факторів ризику, пов'язаних із розвитком ГУН, продемонстрував, що наявність ХХН (із відношенням шансів (ВІШ) = 36,86; 95% довірчий інтервал (ДІ) [4,78–284,1], p < 0,0001) та застосування нефротоксичних препаратів (ВІШ = 3,89; 95% ДІ [2,22–6,82], p < 0,0001) були тісно пов'язані з розвитком ГУН. У пацієнтів літнього віку також виявлено значно підвищений ризик виникнення ГУН порівняно з молодшими віковими групами (ВІШ = 2,56; 95% ДІ [1,32–4,97], p = 0,048).
Висновки. ГУН є поширеним ускладненням серед пацієнтів, причому літній вік і наявність ХХН є ключовими факторами ризику його розвитку. Застосування нефротоксичних препаратів також продемонструвало сильний зв'язок із розвитком ГУН. Хоча пацієнти з ГУН перебували у ВІТ довше, показник загальної смертності істотно не відрізнявся між пацієнтами з ГУН та без нього. Ці результати підкреслюють важливість ранньої оцінки ризиків і ретельного моніторингу нефротоксичних впливів у пацієнтів у критичному стані.
Ключові слова: *гостре ураження нирок, хронічна хвороба нирок, нефротоксичні препарати, фактори ризику, смертність.*

The COVID-19 pandemic, caused by the novel coronavirus SARS-CoV-2, emerged as a global health crisis in late 2019 and has since had profound impacts on societies, economies, and healthcare systems worldwide. The first cases of the disease were reported in Wuhan, Hubei Province, China, in December 2019, with the virus likely originating from a zoonotic transmission, possibly linked to a seafood market where live animals were also sold [1]. On January 30, 2020, the World Health Organization declared the outbreak a Public Health Emergency of International Concern, and by March 11, 2020, it was characterized as a pandemic [2].

Common initial COVID-19 symptoms include fever, cough, fatigue, and loss of taste or smell, though severe cases can lead to pneumonia, acute respiratory distress syndrome, and death, particularly among older adults and those with underlying health conditions [3, 4].

The scientific community responded rapidly to the pandemic. By December 2020, multiple vaccines had been developed and authorized for emergency use, marking a historic achievement in vaccine development. Vaccines from Pfizer-BioNTech, Moderna, AstraZeneca made a huge impact on controlling COVID-19 pandemic [5].

In a large cohort of patients with COVID-19 symptoms described at the beginning of the pandemic, 81% had a mild course, 14% had a severe course, and 5% had a critical course with multiple organ failure with the mortality rate near 50% [6]. Nowadays, after worldwide vaccination, the level of severe and critical course decreased substantially, severe forms of COVID-19 can still lead to acute cardiovascular failure, acute kidney injury (AKI), cardiac arrhythmias, coagulopathy, ischemic or hemorrhagic stroke, and the optimal treatment strategy needs to be clarified [7, 8].

AKI, which is defined by a sudden decline in kidney function, which is typically identified by a rise in serum creatinine and/or a reduction in urine output, is frequently observed in critically ill COVID-19 patients. Studies indicate that it occurs in 20–40% of individuals admitted to intensive care units (ICU) [9]. AKI has few basic contributors in COVID-19: among them systemic inflammatory response, sepsis, disturbances in renin-angiotensin-aldosterone homeostasis, rhabdomyolysis, and even direct viral kidney injury [10].

Identification of kidney-related complications in COVID-19 patients, along with the implementation of therapeutic strategies to prevent AKI or decrease its progression to more severe stages, is crucial for reducing morbidity and mortality in hospitalized patients.

The objective: to investigate the incidence, risk factors, clinical characteristics, and outcomes of AKI in patients with severe and critical COVID-19.

MATERIALS AND METHODS

A retrospective analysis of the treatment outcomes of 1,311 patients in Kyiv City Clinical Hospital No. 17 with City Clinical Hospital the treatment out test, who were hospitalized from September 2020 till December 2021, was conducted.

The inclusion criteria for the study were: a diagnosis of "coronavirus disease" confirmed by a PCR (Polymerase Chain Reaction) test for COVID-19; patients' age over 18 years; severe (in the presence of the following factors: respiratory rate ≥ 30 breaths/min; blood oxygen saturation $SaO_2 \leq 92\%$; $PaO_2/FiO_2 < 300$ mmHg; pulmonary infiltrates > 50% with their progression within 24–48 hours) or critical course (severe multi-organ failure, decreased level of consciousness according to the Glasgow Coma Scale) coronavirus disease [5]. 252 (19.2%) patients have meet inclusion criteria. Exclusion criteria comprised: (1) refusal to participate by the patient or their legal representative, (2) in-hospital mortality within 24 hours of admission (n = 30), (3) active pulmonary tuberculosis (n = 1),

and (4) transfer to other healthcare facilities. Taking into account the exclusion criteria, the observation group consisted of 221 (87.7%) patients (113 (51.1%) women and 108 (48.9%) men). Among them, 78 (35.3%) patients had AKI during hospitalization. AKI was classified according to the Kidney Disease: Improving Global Outcomes (KDIGO) on 3 stages based on serum creatinine (SCr) levels:

- stage 1: a rise in SCr by 0.3 mg/dL within 48 hours or a 1.5- to 1.9-fold increase from baseline within 7 days;
- stage 2: a 2.0- to 2.9-fold increase in SCr from baseline within 7 days;
- stage 3: a 3-fold or greater increase in SCr from baseline within 7 days or the need for renal replacement therapy (RRT) [11].

The estimated glomerular filtration rate (eGFR) was determined using the Chronic Kidney Disease (CKD) Epidemiology Collaboration creatinine equation.

Analysis of medical records, demographic, clinical and laboratory data was performed. Prescription of nephrotoxic agents at least one time during hospitalization (non-steroidal anti-inflammatory drugs, antibiotics (vancomycin, aminoglycosides, piperacillin-tazobactam, cephalosporins, colistin/polymyxin B), proton pump inhibitors, iodinated contrast) were evaluated. The diagnosis of CKD was identified based on: documented medical history (prior diagnosis of CKD by nephrologists or hospital records) and laboratory criteria (persistent eGFR < 60 mL/min/1.73 m^2 for ≥ 3 months or proteinuria > 30 mg/g creatinine in patients past recordings). CKD stages 3–5 (eGFR < 60 mL/min/1.73 m^2) were considered as risk factors and were analyzed in risk factors evaluation of AKI in COVID-19.

Additionally, the need for mechanical ventilation, treatment strategy and vasopressor support of the disease was studied.

The requirement for consent was waived by the Ethics Committee of Bogomolets National Medical University (Protocol No. 12, 24.04.2024) due to the retrospective analysis of anonymized patient records.

Statistical analysis

Normal distribution of variables was assessed using the Shapiro–Wilk test. Categorical variables were summarized as the total count and percentage for each category, while continuous variables were expressed as either the mean ± standard deviation or the median and range. Since most of the data ware categorical variables, the χ^2 (chi-square) test was used to assess the statistical significance, and for small sample sizes (less than 5), the Fisher exact test. Comparison of continuous variables in independent samples was done using the Mann–Whitney U test. The initial database was formed in Microsoft Excel, and statistical processing of the results was performed on a personal computer using the program Statistica 6.1 (StatSoft Inc., USA).

RESULTS AND DISCUSSION

Among 221 patients who underwent retrospective analysis, 176 (79.6%) patients were initially hospitalized to the other departments, and were subsequently transferred to ICU, 45 (20.4%) patients were hospitalized directly to the ICU from emergency department. 127 (57.5%) patients were discharged from the hospital (54 (35.4%) men and 73 (64.6%) women (age 61.00 ± 9.41 years)) and 94 (42.5%) patients died (54 (57.5%) men, 40 (42.5%) were women, age 71.20 ± 10.29 years).

In our cohort, 78 (35.3%) patients had AKI. Demographic and clinical data of the patients with AKI is presented in Table 1.

P-value < 0.05 was considered statistically significant between patients the patients with AKI and those without (non-AKI) (Table 2) reviled that proportion of male patients was similar between groups (47.4% vs 49.7%, p = 0.752). Age distribution showed that patients with AKI were frequently older (p = 0.048). Comorbidities such as cardiovascular disease (CVD) and diabetes mellitus (DM) showed no significant difference, but CKD was markedly higher in AKI patients (20.5% vs 0.7%, p < 0.0001).

Table 1

Baseline characteristics of COVID-19 patients with and without AKI

Characteristics	AKI (n = 78)		Non-AKI (n = 143)		p-value	Total (n = 221)	
	n	%	n	%		n	%
Male	37	47.4	71	49.7	0.752	108	48.9
Age groups, years							
18–44	8	10.3	17	11.9	0.048*	25	11.3
45–59	16	20.5	33	23.1		49	22.2
60–74	31	39.7	73	51.0		104	47.1
75–95	23	29.5	20	14.0		43	19.5
Comorbidities							
CVD	31	39.7	74	51.7	0.087	105	47.5
DM	19	24.4	22	15.4	0.101	41	18.6
CKD	16	20.5	1	0.7	< 0.0001*	17	7.7
Cancer	3	3.8	10	7.0	0.342	13	5.9
CORD	2	2.6	8	5.6	0.301	10	4.5
BMI, kg/m^2	30.4 (26.3; 38.3)		29.4 (25.2; 40.1)		0.345	29.1 (24.5; 38.3)	

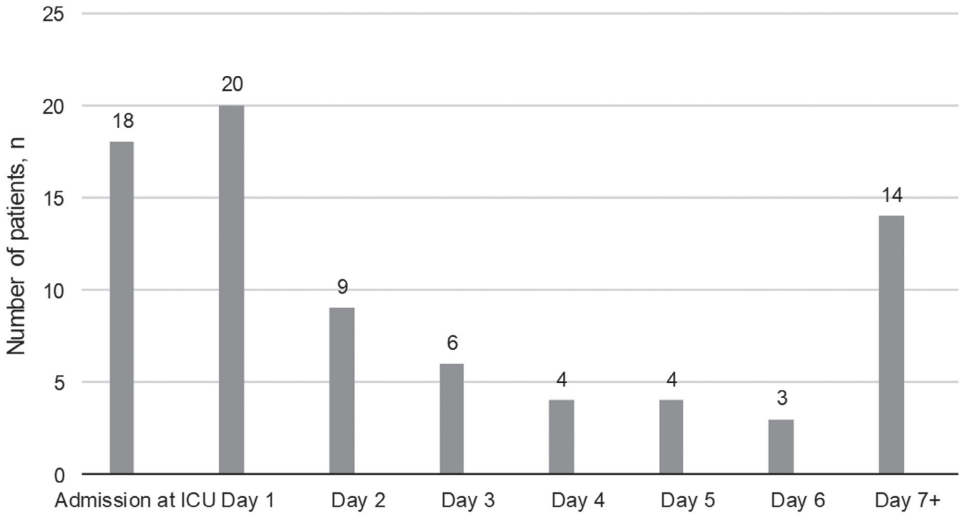
Notes: * – p-value < 0.05 was considered statistically significant; AKI – Acute Kidney Injury; CVD – Cardiovascular Disease; DM – Diabetes Mellitus; CKD – Chronic Kidney Disease; CORD – Chronic Obstructive Respiratory Disease; BMI – Body Mass Index.

Table 2

Laboratory findings in COVID-19 patients with and without AKI

Characteristics	AKI (n = 78)		Non-AKI (n = 143)		p-value	Total (n = 221)	
	n	%	n	%		n	%
Laboratory findings							
Erythrocyte count, × 10^{12}/L	4.0 (3.7; 4.3)		4.2 (3.8; 4.4)		0.076	4.1 (3.8; 4.5)	
Hemoglobin, g/L	103 (91; 114)		105 (92; 118)		0.453	104 (92; 119)	
Leukocytes count, × 10^9/L	11.9 (8.9; 16.5)		11.4 (8.7; 15.5)		0.098	11.5 (8.8; 15.9)	
CPR, mg/L	89.1 (53.5; 145.9)		87.4 (51.3; 138.9)		0.067	88.4 (52.3; 141.2)	
Ferritin, ng/mL	395.5 (192.3; 419.8)		390.1 (190.3; 412.8)		0.19	394.2 (194.3; 420.8)	
Creatinine, μmol/L	105 (98.5; 140.7)		81 (72.3; 122.7)		<0.0001*	88 (78.3; 125.7)	
IL-6, pg/mL	9.8 (5.2; 38.1)		7.1 (4.2; 27.1)		< 0.0001*	8.4 (4.3; 36.1)	
eGFR, mL/min/1.73 m^2	70.5 ± 23.1		84.1 ± 21.7		< 0.0001*	75.1 ± 22.0	
KDIGO stage							
1	46	59.0	–	–	–	46	20.8
2	17	21.8	–	–	–	17	7.7
3	15	19.2	–	–	–	15	6.8
RRT indication	7	9.0	–	–	–	7	3.2
Clinical findings							
Nephrotoxic medications	51	65.4	47	32.9	< 0.0001*	98	44.3
Mechanical ventilation	35	44.9	66	46.2	0.854	101	45.7
Length of stay in the ICU	8.9 ± 7.1		6.3 ± 5.1		0.012*	7.2 ± 1.2	
Mortality	31	39.7	61	42.7	0.959	94	42.5

Notes: * – p-value < 0.05 was considered statistically significant; AKI – Acute Kidney Injury; CRP – C-Reactive Protein; IL-6 – Interleukin-6; eGFR – Estimated Glomerular Filtration Rate; KDIGO – Kidney Disease: Improving Global Outcomes; RRT – Renal Replacement Therapy; ICU – Intensive Care Unit.



Time to onset of AKI in patients with severe and critical course of COVID-19 according to the length of stay in the ICU, days

Comparison of laboratory parameters between patients with AKI and those without (non-AKI) indicate higher creatinine levels in AKI patients (105 vs 81 μmol/L), lower eGFR (70.5 vs 84.1 mL/min/1.73 m^2, $p < 0.0001$) and higher IL-6 (9.8 (5.2; 38.1) vs 8.4 (4.3; 36.1) pg/mL, $p < 0.0001$), while hemoglobin, leukocytes, CRP, and ferritin levels showed no significant differences. AKI severity according KDIGO staging reveled 59.0% of patients had stage 1, 21.8% – stage 2, and 19.2% – stage 3. Clinical findings show that nephrotoxic medication use was significantly more common in AKI patients (65.4% vs 32.9%, $p < 0.0001$), while mechanical ventilation rates are similar (44.9% vs 46.2%, $p = 0.854$). Despite ICU stay was longer for AKI patients (8.9 vs 6.3 days, $p = 0.012$), but overall mortality did not significantly differ (39.7% vs 42.7%, $p = 0.959$).

Most often AKI occurred on ICU admission (18 patients) and on Day 1 (20 patients), followed by a gradual decline over the next days, with a secondary increase on Day 7+ (14 patients) (Figure).

Table 3

OR and 95% CI for Risk Factors Associated with AKI

Characteristics	OR	95% CI	p-value
Male gender	0.915	[0.527–1.587]	0.752
Age groups, years			
18–44	0.847	[0.34–2.11]	0.720
45–59	0.857	[0.43–1.71]	0.660
60–74	0.634	[0.36–1.12]	0.120
75–95	2.56	[1.32–4.97]	0.048
CVD	0.62	[0.35–1.09]	0.087
DM	1.78	[0.89–3.56]	0.101
CKD	36.86	[4.78–284.1]	< 0.0001
Cancer	0.52	[0.14–1.95]	0.342
CORD	0.45	[0.09–2.18]	0.301
Nephrotoxic medications	3.89	[2.22–6.82]	< 0.0001
Mechanical ventilation	0.95	[0.54–1.67]	0.854

Notes: AKI – Acute Kidney Injury; CVD – Cardiovascular Disease; DM – Diabetes Mellitus; CKD – Chronic Kidney Disease; CORD – Chronic Obstructive Respiratory Disease; OR – Odds Ratio; CI – Confidence Interval.

Using our cohort data, we computed odds ratios (OR) with 95% confidence intervals (CI) to identify risk factors associated with AKI development in ICU-admitted patients with severe/critical COVID-19 (Table 3).

The analysis of risk factors associated with AKI revealed that patients with CKD had a markedly higher risk of developing AKI, with an OR of 36.86; 95% CI [4.78–284.1], $p < 0.0001$. Similarly, the use of nephrotoxic medications was strongly associated with AKI (OR = 3.89; 95% CI [2.22–6.82], $p < 0.0001$). Older patients aged 75–95 years also had a significantly increased risk of AKI compared to younger age groups (OR = 2.56; 95% CI [1.32–4.97], $p = 0.048$). Other factors, such as gender, CVD, DM, cancer, chronic obstructive pulmonary disease, and mechanical ventilation, did not show statistically significant associations with AKI in our study.

Although COVID-19 primarily affects the respiratory system, kidney involvement is frequently observed, ranging from mild proteinuria to severe AKI and need for RRT. The mechanism underlying AKI in COVID-19 patients is multifactorial and includes virus-mediated injury, cytokine storm, activation of the angiotensin II pathway, hypercoagulation, etc. [12].

Evidence suggests that SARS-CoV-2 directly infects the kidneys by binding to ACE2 (Angiotensin-Converting Enzyme 2) receptors, leading to renal cell damage. Studies using ACE2 staining have demonstrated that the kidneys and bladder are highly enriched with ACE2, making them particularly vulnerable to viral invasion [13]. ACE2 proteins are predominantly expressed on the proximal tubular apical membranes and, to a lesser extent, in podocytes. It is hypothesized that the virus initially infects glomerular endothelial cells through the arteriole and glomerular capillaries, subsequently spreading to podocytes and binding to receptors in the proximal tubules. This process can result in acute tubular necrosis, protein leakage into Bowman's capsule, collapsing glomerulopathy, and mitochondrial dysfunction [14].

Studies showed that, inflammatory infiltration of the renal interstitium is primarily composed of lymphocytes and plasma cells, with occasional eosinophils [6]. Activated lymphocytes migrate to kidney tissue to eliminate infected cells and release inflammatory cytokines, causing local inflammation and tissue damage. Furthermore, cytotoxic particles and proinflammatory cytokines, which are highly expressed in lymphocytes, exacerbate kidney injury [15].

Among the cytokines, IL-6 plays a central role by stimulating renal endothelial cells to release pro-inflammatory chemokines and cytokines, and we reviled its higher level among patients with AKI [16].

One of the first studies in China reporting a lower rate of AKI among 1,099 hospitalized patients, which was only found in 6 patients (0.5%) [17]. However, Yang et al. reported a 29% incidence of AKI among 52 critically ill COVID-19 patients, which was associated with the severity of pneumonia [18]. Overall, the incidence of AKI ranged from 3.5% in moderate disease patients to 42.9% in critically ill patients [19]. Our data reveled 35.3% among patients with severe and critical course of COVID-19, and most patients presented with AKI either at admission or within the first two days.

Data showed that AKI was independently associated with higher mortality [20–22], however we didn't find higher mortality in our cohort. Probably, the explanation can be high percentage of nephrotoxic drugs in our patients with AKI, that was 65.4%, especially antibiotics, which could cause additional laboratory AKI, but with good outcome in case of its discontinuation. Data also showed a link between rapid kidney function recovery and improved short-term survival [23], and only 7 patients required RRT.

We also concluded, that patients with CKD had a substantially higher risk of developing AKI. Furthermore, older patients aged 75–95 years were at a significantly increased risk of AKI compared to younger age

groups. These findings underscore the importance of closely monitoring patients with CKD, those receiving nephrotoxic medications, and elderly individuals, as they are at a heightened risk of AKI. Early identification and targeted interventions for these high-risk groups are crucial to reducing the incidence and severity of AKI, ultimately improving patient outcomes. Further research is warranted to explore additional risk factors and refine preventive strategies.

CONCLUSIONS

AKI was a common complication, with older age and pre-existing CKD identified as key risk factors. The use of nephrotoxic medications also showed a strong association with AKI development. While AKI patients had longer ICU stays, overall mortality did not differ significantly between those with and without AKI. These findings emphasize the importance of early risk assessment and careful management of nephrotoxic exposures in critically ill patients.

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