НИРКИ KIDNEYS

UDC 616.61.75-616.43.45 *DOI: https://doi.org/10.22141/2307-1257.14.1.2025.491*

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Tool for acute kidney injury prediction in hospitalised patients with COVID-19

For citation: Kidneys. 2025;14(1):24-31. doi: 10.22141/2307-1257.14.1.2025.491

Abstract. ***Background.*** *The study focuses on acute kidney injury (AKI) in hospitalised COVID-19 patients. AKI is a significant medical issue often linked to severe conditions like pneumonia and sepsis. Understanding the predictors of AKI in COVID-19 is crucial for improving patient outcomes.* ***Materials and methods.*** *In Kyiv, a retrospective, case-control study was conducted at the KAPITAL Ltd. (Medical Centre "Universal Clinic "Oberig"). The study included 129 patients hospitalised with moderate to severe COVID-19 from 2020 to 2021. They were divided into those who developed AKI (n = 19) and those who did not (n = 110). We used various statistical logistic regression models to predict AKI.* ***Results.*** *Significant predictors of AKI included age, C-reactive protein levels, absolute lymphocyte count, Padua Prediction Score, and respiratory insufficiency. No significant differences were observed in gender distribution, estimated glomerular filtration rate on admission, prevalence of hypertension, diabetes, or body mass index between the two groups. Multivariate logistic regression incorporating the Padua Prediction Score showed strong predictive performance with an AUC of 0.803.* ***Conclusions.*** *The study highlights the critical need for accurate predictive models tailored to specific populations. It aims to develop a novel, region-specific predictive model for AKI in COVID-19 patients by focusing on the Ukrainian population. The model leverages local data to improve risk predictions and patient outcomes, emphasising the importance of early identification and stratification of high-risk individuals.*

Keywords: *mortality; acute kidney injury; pneumonia; C-reactive protein; Padua Prediction Score; lymphocytes; respiratory insufficiency; public health; hospitalisation; outcomes; risk factors; modelling*

Introduction

Secondary outcomes of coronavirus disease 2019 (COVID-19) are relevant study directions. Acute kidney injury (AKI) is a significant medical problem, often associated with severe cases of pneumonia, sepsis, and other critical conditions that may result in a sudden loss of kidney function. In patients with COVID-19, AKI arises due to both direct and indirect impacts on renal function. AKI was associated with poor outcomes. Despite growing evidence, developing accurate prediction models for AKI in COVID-19 patients remains a topic of considerable debate. Recent studies propose using static predictive models [1] or artificial intelligence/machine learning [2]. However, we still lack a unique risk assessment application and additional decision-making tools in Ukraine.

Our study aims to analyse existing predictive models for acute kidney injury in hospitalised COVID-19 patients. We seek to evaluate their applicability and limitations while developing a novel predictive model explicitly tailored to the Ukrainian population. Using data from one of Kyiv hospitals, this model will incorporate region-specific clinical and demographic factors to enhance the accuracy of AKI risk prediction and improve patient outcomes. So, this study **aimed** to assess the risks of acute kidney injury and create a prediction model of acute kidney injury risk.

Materials and methods

We conducted a retrospective, case-control, single-center study to create an AKI risk-predicting model. The study was conducted at the KAPITAL Ltd. (Medical Cen-

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tre "Universal Clinic "Oberig"). A total of 914 patients with COVID-19, 503 (55 %) males, 411 (45 %) females with a median age of 61 years (interquartile range (IQR) 19.00–93.00), were hospitalized from September 29, 2020, to July 9, 2021. Three hundred and ninety-three patients were hospitalized in 2020 and 521 — in 2021. In 2020, 19 (4.83 %) patients died in COVID departments. We analysed hospitalised patients (Table 1) with severe and moderate COVID-19 cases from 2020 to 2021 (n = 129). They were divided into those who had acute kidney injury during hospitalisation (n = 19) and those who did not have AKI (n = 110). Our study intentionally included a higher proportion of elderly patients, reflecting the well-established association between age and the severity of COVID-19. This demographic focus was driven by their increased vulnerability to severe outcomes, including AKI and mortality. By emphasizing this group, we aimed to capture the clinical complexities most relevant to severe cases of COVID-19. However, this focus introduces a potential selection bias, as younger patients with milder forms of the disease were underrepresented. Future studies should incorporate a broader age range to ensure generalizability of the findings. All patients agreed to use personal data on the first day of hospitalisation. The Ethical Committee of the Bogomolets National Medical University proved the study (expert conclusion according to the Protocol of Ethical Committee No. 173 dated June 19, 2023).

The data was processed using EZR. For analysing quantitative indicators, we used the mean value (M), standard error (± m), 95% confidence interval (CI) for the normal distribution; the median value (Me) and interquartile range (QI-QIII) were calculated for the non-normal distribution. We used prevalence (%) and 95% CI for qualitative indicators. The Mann-Whitney test was applied to compare mean values in two groups for quantitative measures, and the chi-square test (with Yates' correction) to compare qualitative measures. We used logistic regression models to estimate the association of factorial features with the outcomes risk assessment. We also assessed the odds ratio with 95% CI. The models were evaluated by the area under a receiver operating characteristic curve (AUC) and its 95% CI, with measurement of sensitivity and specificity. We performed all calculations for a critical significance level of 0.05.

There was no significant difference in gender distribution between the non-AKI (n = 110) and AKI group (n = 19) (p = 0.8). However, significant differences were observed in the median age of patients in the AKI group who were younger — 79 years (IQR 67.5–80). No significant difference was found in the estimated glomerular filtration rate (eGFR) on admission (p = 0.8). Additionally, there were no significant differences in the prevalence of hypertension, diabetes, or in body mass index (BMI) between the two groups.

Results and discussion

Univariate logistic regression analysis (Table 2) revealed no significant differences in factors such as baseline interleukin-6 (IL-6), albumin levels on admission, BMI, eGFR on admission, serum creatinine on admission, or glycated haemoglobin (HbA1c).

However, significant differences were observed in the following variables: age (negative influence), C-reactive protein (CRP) maximum level, minimum albumin levels, minimum eGFR, maximum creatinine levels, creatinine increase (times elevated), Padua Prediction Score, respiratory insufficiency (severity), severity of lung injury as assessed by multi-slice computed tomography.

These findings highlight key predictors associated with acute kidney injury in the studied population, suggesting their potential inclusion in future predictive modeling efforts. Using multivariate logistic regression, we developed a predictive model incorporating the Padua Prediction Score to assess the impact of various factors on the severity of kidney injury (Table 3). The model demonstrated strong performance, with the AUC of 0.803 (95% CI 0.698–0.908). The identified threshold for high risk was 0.167, with a sensitivity of 78.9 % and a specificity of 72.5 % (Fig. 1). These findings suggest the model's potential utility in predicting acute kidney injury in hospitalised COVID-19 patients.

In our previous studies, we suggested using the Padua Prediction Score, rise in serum creatinine, and proposed personal lethality risk index as additional tools for assessing hospitalisation criteria [3, 4].

We developed a model (Table 4) incorporating CRP levels and the Padua Prediction Score as predictive factors in a separate multivariate logistic regression analysis. This model

Table 1. Demographic and clinical characteristics of the study population

Characteristic	Measures	Both groups (n = 129)	Non-AKI group (n = 110)	AKI group (n = 19)	p
Gender	Females, N (%)	73 (56.6)	63 (57.3)	10 (52.6)	0.8
Age, years	Me (IQR)	79.1 (78–83)	81.0 (78–83.75)	79 (67.5–80)	0.001*
eGFR on admission, ml/min	M (SD)	60.1 (20.8)	60.31 (19.73)	59 (26.67)	0.8
Hypertension	N (%)	58 (45)	46 (41.8)	12 (63.2)	0.13
Diabetes mellitus	N (%)	29 (22.5)	25 (22.7)	4 (21.1)	1.0
BMI, kg/m^2	M (SD)	28.8 (5.4)	28.3 (5.58)	31.23 (3.88)	0.33

Note: * — if p is lower than 0.05, the difference between groups is significant.

demonstrated the AUC of 0.839 (95% CI 0.756–0.921), indicating substantial predictive accuracy (Fig. 2).

The identified threshold for high risk was 0.177, with a sensitivity of 78.9 % and a specificity of 80 %. These results underscore the potential of CRP and the Padua Prediction Score as valuable predictors for acute kidney injury in COVID-19 patients.

We found the positive influence of respiratory insufficiency and CRP on the AKI risk (Table 5).

At the threshold point of 0.322, sensitivity is 89.5 % and specificity is 87.8 % (Fig. 3).

For calculating AKI risk, we proposed the following formula:

$$pVSevAKI = 1/(1 + e^{-(-8.190447 + 0.862989 \cdot X1 + 0.010454 \cdot X2)}),$$

where X1 is respiratory insufficiency (1, 2 or 3), X2 is C-reactive protein, mg/L.

The formula could be implemented in R and Python as well. For example, patient A. had absolute lymphocyte count 0.06, respiratory insufficiency 3, CRP 185.06 mg/L, Padua Prediction Score 5. In R, it looks like a script, with

Table 2. Univariate logistic regression model for AKI risk

Factor feature	Model coefficient, b ± m	Significance, p*	Odds ratio (95% CI)	AUC (95% CI)
Age, years	–0.096 ± 0.030	0.00141	0.908 (0.856–0.963)	0.731 (0.612–0.85)
Absolute lymphocyte count, 10^9/l	–4.50 ± 1.41	0.0011	0.011 (0.0007–0.177)	0.799 (0.684–0.914)
CRP max, mg/l	0.014 ± 0.004	0.000184	1.02 (1.010–1.020)	0.815 (0.729–0.902)
IL-6	0.0027 ± 0.0027	0.327	1.00 (0.997–1.01)	0.66 (0.511–0.808)
Albumin baseline, g/l	–0.087 ± 0.077	0.262	–	0.618 (0.429–0.806)
Albumin min	–0.22 ± 0.08	0.00954	0.8 (0.684–0.949)	0.75 (0.607–0.893)
Alanine aminotransferase, U/l	0.02 ± 0.01	0.0477	1.02 (1.00–1.040)	0.641 (0.513–0.769)
Aspartate aminotransferase, U/l	0.016 ± 0.007	0.0352	1.02 (1.00–1.030)	0.64 (0.493–0.786)
BMI, kg/m^2	0.1 ± 0.1	0.323	–	0.725 (0.468–0.982)
eGFR baseline, ml/min	–0.003 ± 0.010	0.799	–	0.501 (0.336–0.667)
Creatinine baseline, μmol/l	0.009350 ± 0.004642	0.044	–	0.556 (0.402–0.71)
eGFR min, ml/min	–0.135 ± 0.030	0.00000896	0.873 (0.823–0.927)	0.941 (0.896–0.987)
Creatinine max, μmol/l	0.02000 ± 0.00468	0.0000181	1.02 (1.010–1.030)	0.949 (0.907–0.991)
Increase in creatinine, times	2.4900 ± 0.6488	0.00012	12.1 (3.38–43.1)	0.941 (0.874–1)
Ferritin	0.0003420 ± 0.0002500	0.175	–	0.774 (0.673–0.876)
HbA1C, %	0.43 ± 0.19	0.026	1.54 (1.05–2.25)	0.566 (0.4–0.733)
Padua Prediction Score	0.98 ± 0.36	0.00642	2.67 (1.32–5.41)	0.658 (0.53–0.786)
Respiratory insufficiency	2.40 ± 0.67	0.000406	11.1 (2.92–41.9)	0.845 (0.767–0.923)
CT, %	0.035 ± 0.012	0.00408	1.04 (1.01–1.060)	0.757 (0.637–0.877)
CT, % over 50 %	1.34 ± 0.52	0.00998	3.8 (1.38–10.6)	0.641 (0.52–0.762)

Notes (here and in Table 3–5): b — model coefficient; m — coefficient's error; * — if p is lower than 0.05, the model is significant.

Table 3. Multivariate logistic regression model for AKI risk as a calculator for evaluating Padua Prediction Score and absolute lymphocyte count

Factor feature	Model coefficient, b ± m	Significance, p*	Odds ratio (95% CI)
Intercept	–2.5610 ± 1.4226	0.07	0.077 (0.0047–1.26)
Padua Prediction Score	0.7298 ± 0.3774	0.05	2.07 (0.99–4.35)
Absolute lymphocyte count, 10^9/l	–4.1922 ± 1.4344	0.0035	0.015 (0.0009–0.251)

respiratory insufficiency (RI) of 3, CRP of 185.06 mg/L: «RI <- c(3); CRP << c(185.06); df = data.frame (RI, CRP); print(1/1+exp(-predict(GLM.1, df))))». The estimated risk could be thought as high. We presented calculations in Table 7.

The influence of CRP levels and the absolute lymphocyte count on acute kidney injury outcomes is significant, particularly in severe cases where the inflammatory process is critical. Protein levels, for example CRP, often rise rapidly during severe inflammation, contributing to multi-organ injury, including direct effects on kidney function.

While these effects may result from direct inflammatory damage, preclinical mechanisms like microvascular injury or immune-mediated pathways may also play a role. Recognising such patterns highlights the importance of using these markers as tools for decision-making in clinical practice.

Incorporating CRP levels and absolute lymphocyte count into predictive models offers an additional layer of risk stratification for COVID-19 patients, enabling earlier identification of those at higher risk of AKI. Such an approach can support timely interventions and improve patient care.

These results could also be applicable to other viral or bacterial types of pneumonia, as similar inflammatory mechanisms may contribute to acute kidney injury in these conditions. However, external validation must confirm the model's generalizability across different patient populations and settings.

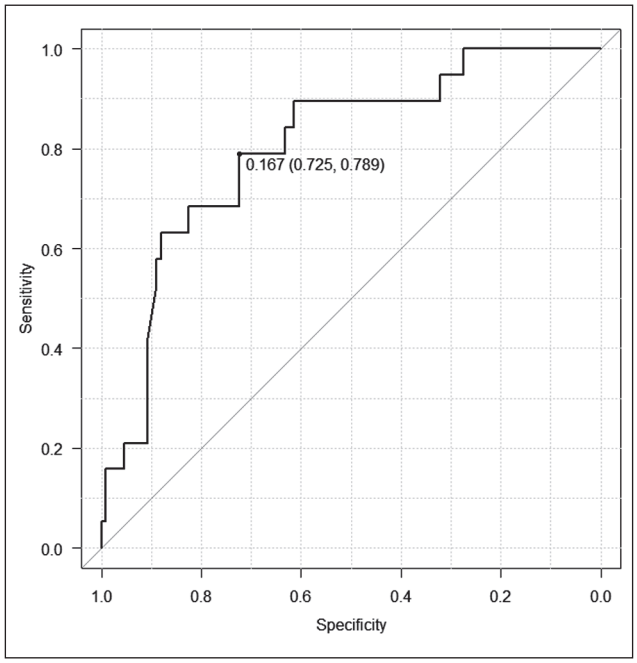

Figure 1. Logistic regression model for predicting AKI depending on Padua Prediction Score and absolute lymphocyte count with AUC 0.803 (95% CI 0.698–0.908)

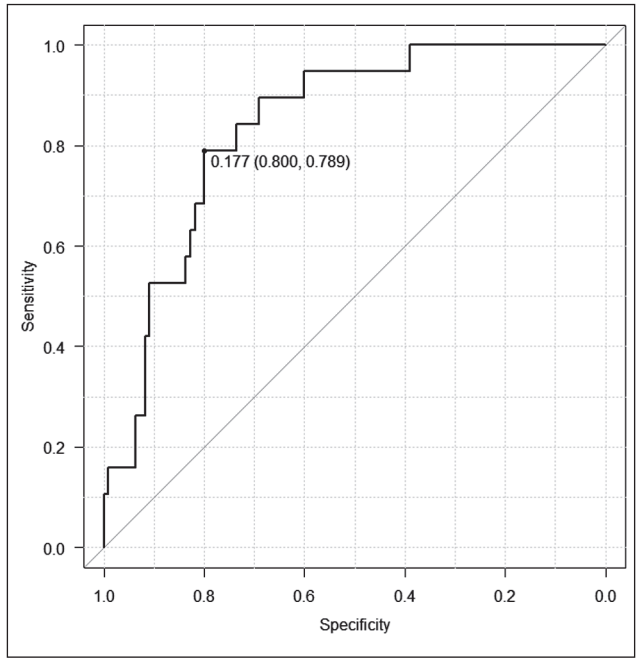

Figure 2. Logistic regression model for predicting AKI depending on Padua Prediction Score and CRP with AUC 0.839 (95% CI 0.756–0.921)

Table 4. Multivariate logistic regression model for AKI risk as a calculator for evaluating Padua Prediction Score and CRP

Factor feature	Model coefficient, b ± m	Significance, p*	Odds ratio (95% CI)
Intercept	–6.011678 ± 1.453929	0.0000355	0.002 (0.0001–0.04)
Padua Prediction Score	0.862989 ± 0.389262	0.026624	2.37 (1.11–5.08)
CRP, mg/l	0.013576 ± 0.003887	0.000478	1.01 (1.01–1.02)

Table 5. Multivariate logistic regression model for AKI risk as a calculator for evaluating respiratory insufficiency and CRP

Factor feature	Model coefficient, b ± m	Significance, p*	Odds ratio (95% CI)
Intercept	–8.190447 ± 2.074065	0.0000785	0.000277 (0.00000476–0.0162)
Respiratory insufficiency	2.227176 ± 0.693696	0.00132	9.270000 (2.38–36.1000)
CRP	0.010454 ± 0.004735	0.02725	1.0 (1.0–1.02)

Further research is essential to explore the utility of this approach in broader contexts. Future studies should focus on validating these findings in larger, more diverse cohorts and assessing the potential for integrating these predictive tools into routine clinical practice.

The current literature highlights gaps in the systematic evaluation of available data. Many findings are derived from isolated case-control studies, and few comprehensive studies specifically address the AKI prediction issue. Notably, Krzanowska et al. conducted a nationwide prospective study in Poland in 2024, focusing on risk factors for AKI among COVID-19 patients treated in Polish hospitals. Their work examined 5,806 patients with moderate to severe COVID-19, identifying a subgroup of 4,630 patients for detailed analysis. The study provided valuable insights into the role of some conditions and laboratory tests, including chronic kidney disease, a history of previous AKI episodes, kidney transplantation, need for oxygen therapy, hypertension, circulatory failure, procalcitonin, neutrophil count, and myoglobin concentration [1]. The research summarised key treatment outcomes by leveraging their predictive model and underscored the importance of stratifying patients based on AKI risk profile. The findings highlighted the need for further validation of predictive tools across diverse populations to improve AKI management in COVID-19 cases.

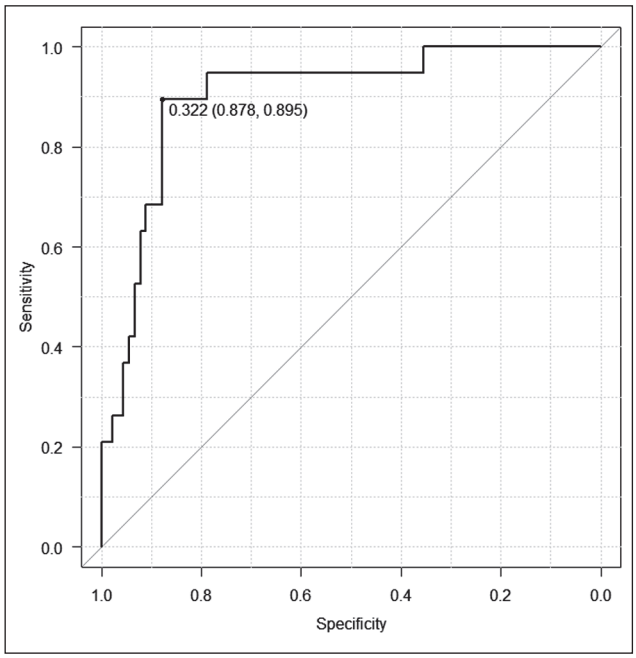

Figure 3. Logistic regression model for predicting AKI depending on respiratory insufficiency and CRP with AUC 0.901 (95% CI 0.826–0.975)

Several studies have reported similar findings in predicting AKI in COVID-19 patients. A study conducted by Li et al. in the Boston area [5] developed risk scores for AKI

Table 6. Evaluation of diagnostic test performance: sensitivity, specificity, and predictive values

Characteristic	Estimation (lower-upper CI)
Apparent prevalence	0.257 (0.178–0.349)
True prevalence	0.174 (0.108–0.259)
Sensitivity	0.895 (0.669–0.987)
Specificity	0.878 (0.792–0.937)
Positive predictive value	0.607 (0.406–0.785)
Negative predictive value	0.975 (0.914–0.997)
Diagnostic accuracy	0.881 (0.805–0.935)
Likelihood ratio of a positive test	7.321 (4.120–13.006)
Likelihood ratio of a negative test	0.120 (0.032–0.446)

Table 7. Calculator for AKI risk prognosis in COVID-19

Intercept	**Absolute lymphocyte count, 10^9/L**	**Padua Prediction Score**	**Calculation**	**Result**	**Critical value**	0.167	**Sensitivity, %**	78.9
N/a	0.06	5					**Specificity, %**	72.5
–2.561	–4.1922	0.7298	1.0161269	**High risk of acute kidney injury**				
Intercept	**CRP, mg/L**	**Respiratory insufficiency (1, 2, 3 or absent)**	**Calculation**	**Result**	**Critical value**	0.322	**Sensitivity, %**	89.5
N/a	185.06	3					**Specificity, %**	87.8
–8.190447	0.010454	2.227176	1.6533135	**High risk of acute kidney injury**				
Intercept	**CRP, mg/L**	**Padua Prediction Score**	**Calculation**	**Result**	**Critical value**	0.177	**Sensitivity, %**	78.9
N/a	185.06	5					**Specificity, %**	80.0
–6.011678	0.013576	0.862989	1.4423554	**High risk of acute kidney injury**				

and death using demographic factors and biomarkers. Their model achieved an AUC of 0.785 for predicting AKI and utilised factors like age, CRP, and D-dimer levels. Other research in Switzerland [6] also highlighted the incidence of AKI in hospitalised COVID-19 patients. This study focused on factors like age, chronic kidney disease history, CRP levels and found that AKI was common in patients with severe COVID-19. It highlights that AKI is associated with high in-hospital mortality [6]. A comprehensive review critically appraised various predictive models for diagnosing and prognosing COVID-19. It emphasised the necessity for validated models to improve clinical decision-making and patient outcomes [7]. These studies highlight the importance and ongoing efforts to improve predictive models for AKI in COVID-19 patients.

The BIRCOV trial provides valuable insights into the role of renin-angiotensin system (RAS) inhibitors in COVID-19 patients, demonstrating increased risks of hypotension and acute kidney injury with angiotensin-converting-enzyme inhibitors compared to angiotensin II receptor blockers or direct renin inhibitors. These findings align with our observations regarding the challenges in managing blood pressure in patients with COVID-19 pneumonia and coexisting hypertensive conditions [8]. While our retrospective study could not fully capture the use and duration of RAS inhibitors, the BIRCOV study results highlight the importance of considering such medications' impact on kidney function and blood pressure control. It is important to note that our model does not account for the use or duration of renin-angiotensin system inhibitors, given the retrospective design and incomplete data from external intensive care unit transfers. Future prospective studies should aim to include this variable for a more comprehensive risk assessment. The patients in our study presented with diverse clinical backgrounds, including varying stages of COVID-19 pneumonia. Some were admitted from home, while others were transferred from other intensive care units, often with advanced conditions and prior interventions. Some patients experienced acute respiratory distress syndrome that is why it was difficult to achieve complete information on their medical history. This population also had high rates of complications, including bacterial superinfection and sepsis, which likely contributed to AKI and other organ injuries.

Although hypertension was a common comorbidity in the medical histories of many patients, our study did not focus on real-time blood pressure measurements during hospitalization. This decision was made because blood pressure variability in critically ill COVID-19 patients is often influenced by the use of vasopressors such as noradrenaline or other adrenomimetic agents rather than underlying hypertensive pathology. Given the complexity of managing blood pressure in critically ill patients, particularly those requiring hemodynamic support, the direct relationship between baseline hypertension and AKI risk could not be reliably assessed in our cohort. Comprehensive monitoring and intervention strategies in elderly patients, given their heightened vulnerability to severe COVID-19 outcomes, including AKI and multi-organ dysfunction, are essential. The study inclusion criteria focused on hospitalised patients with moderate or severe COVID-19, and we did not analyse the impact of hospitalisation duration or timing in this cohort.

All patients with diabetes in our study continued to receive their outpatient therapy upon admission, wherever clinically appropriate. However, for patients experiencing decompensation of diabetes, particularly those influenced by corticosteroid therapy, we employed a uniform strategy of insulin therapy using short-acting insulin to manage hyperglycemia effectively. This approach aligns with standard practices for managing hyperglycemia in critically ill patients. The introduction of corticosteroids often exacerbated glycemic instability, necessitating careful monitoring and frequent insulin titration to prevent hyperglycemia and its associated complications. We excluded detailed information about the use of non-insulin medications for diabetes such as metformin, sitagliptin, or dapagliflozin, as the exposure to these agents was inconsistent among patients. The variability in their usage and potential discontinuation upon hospital admission made it challenging to assess their influence on outcomes like AKI or mortality. Instead, our analysis focused on therapies recommended in clinical guidelines for COVID-19 management in intensive care settings, ensuring consistency across the cohort. In our cohort, patients received glucocorticoids and anticoagulants as part of their COVID-19 management, in line with clinical guidelines. Low molecular weight heparins were administered in therapeutic dosages to prevent or manage thrombotic complications commonly observed in severe COVID-19 cases. These interventions were guided by regular monitoring of coagulation parameters such as D-dimer levels, platelet count, and fibrinogen. The use of glucocorticoids was particularly critical in managing hyperinflammatory states, though it necessitated close monitoring of glucose levels and hemodynamics to mitigate potential side effects like hyperglycemia or fluid retention. We emphasized the importance of systematically monitoring laboratory and clinical parameters to guide treatment decisions. For assessing hemodynamic and coagulation status, the PaO_2/FiO_2 ratio was a key metric for respiratory function and oxygenation, while coagulation markers informed adjustments to anticoagulant therapy. The Padua Prediction Score was utilized to stratify thrombotic risk and guide the dosing of anticoagulants, ensuring that patients at higher risk of thrombosis received adequate prophylaxis or therapeutic management. Our study collected detailed data on oxygen therapy methods used for patients, including nasal cannula, high-flow oxygen therapy, continuous positive airway pressure therapy, non-invasive ventilation, and mechanical ventilation. These interventions were implemented based on the severity of respiratory distress and clinical judgment during treatment. Despite the detailed data on oxygen supply types, our analysis did not identify a statistically significant influence of these interventions on the predictive outcomes of our model. This may reflect the heterogeneity in patient responses to oxygen therapy and the multifactorial nature of AKI in severe COVID-19. The lack of significance could also be attributed to the advanced disease stages of our cohort, where oxygen therapy might act as a supportive measure but not directly influence key predictors such as creatinine levels, ferritin, or

respiratory insufficiency scores. Although the type of oxygen supply did not impact our model, its importance in patient management remains indisputable. Escalation of oxygen therapy, particularly to non-invasive or mechanical ventilation, often reflects severe disease progression and is an essential component of supportive care in COVID-19.

Conclusions

Our study underscores the significant impact of acute kidney injury in hospitalised COVID-19 patients and highlights the critical need for accurate predictive models. The analysis of existing models reveals gaps and limitations, particularly in COVID-19. By focusing on the Ukrainian population, our study aims to develop a novel, region-specific predictive model for AKI, considering unique clinical and demographic factors.

Significant predictors like CRP, absolute lymphocyte count, the Padua Prediction Score, and respiratory insufficiency were identified as critical in assessing AKI risk. It is obligatory to calculate eGFR and compare it with previous available results. In case of a rapid rise in creatinine or a decrease in urine output, the decision on hospitalisation should be highly recommended.

Our multivariate logistic regression model incorporating the Padua Prediction Score demonstrated strong performance with an AUC of 0.803, indicating reliable predictive accuracy.

By leveraging data from Kyiv, our model achieves tailored risk predictions that account for local healthcare dynamics and patient characteristics.

These insights emphasise the importance of such models in improving patient outcomes through early identification and stratification of high-risk individuals. Furthermore, our findings advocate for continued research and model validation across diverse populations to enhance the management of AKI in COVID-19 and other critical illnesses.

Practical recommendations. Based on the findings of our study and existing literature, we propose the following recommendations for clinical practice:

1. Close monitoring of renal function. Regular assessment of creatinine and eGFR levels in COVID-19 patients, particularly those with preexisting hypertension, chronic kidney disease, or exposure to RAS inhibitors.

2. Individualized blood pressure management. For patients with severe COVID-19, adjust or withdraw RAS inhibitors as necessary to prevent hypotension and exacerbation of organ injury. Substitute with alternative antihypertensive regimens when clinically indicated.

3. Early detection and management of superinfection. Vigilance for bacterial infections and sepsis is critical, as these may exacerbate AKI and other organ injuries. Consider prompt antibiotic therapy in suspected cases.

4. Utilization of predictive models. Incorporating AKI risk prediction tools can help identify high-risk patients early, allowing for tailored interventions to mitigate renal injury.

Ethical issues. On the first day of hospitalisation, all patients agreed to use personal data, and the Bogomolets National Medical University's Ethical Committee proved the study (expert conclusion according to the Protocol of Ethical Committee No. 173 dated June 19, 2023).

Acknowledgement. We are grateful to the staff of the KAPITAL Ltd. (Medical Centre "Universal Clinic "Oberig") for clinical activity during pandemics and for the quality of medical care, which could be comparable with results in prominent European medical centres.

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Received 19.12.2024
Revised 23.01.2025
Accepted 31.01.2025

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Conflicts of interests. Author declares the absence of any conflicts of interests and own financial interest that might be construed to influence the results or interpretation of the manuscript.
Information about funding. This study hadn't obtained external funding.

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Інструмент для прогнозування гострого ураження нирок у госпіталізованих пацієнтів із COVID-19

Резюме. Актуальність. Дослідження зосереджене на гострому пошкодженні нирок (ГПН) у госпіталізованих пацієнтів із COVID-19. ГПН є серйозною медичною проблемою, що часто пов'язана з тяжкими станами, як-от пневмонія та сепсис. Розуміння предикторів ГПН у пацієнтів із COVID-19 має вирішальне значення для поліпшення результатів лікування. **Матеріали та методи.** У Києві було проведено ретроспективне дослідження типу «випадок — контроль» на базі ТОВ «КАПИТАЛ» (Медичний центр «Універсальна клініка «Оберіг»). У ньому взяли участь 129 пацієнтів, госпіталізованих із помірним та тяжким перебігом COVID-19 з 2020 по 2021 рік. Вони були розділені на 2 групи: тих, у кого розвинулося ГПН (n = 19), і осіб без ГПН (n = 110). Для прогнозування ГПН використано різні моделі статистичної логістичної регресії. **Результати.** Значущими предикторами ГПН були вік, рівень С-реактивного білка, абсолютна кількість лімфоцитів, оцінка за прогностичною шкалою Падуї та дихальна недостатність. Не спостерігалося суттєвих відмінностей у розподілі за статтю, розрахунковій швидкості клубочкової фільтрації при госпіталізації, артеріальній гіпертензії, цукровому діабеті або індексі маси тіла між двома групами. Багатофакторна логістична регресія, що включає оцінку за шкалою Падуї, продемонструвала високу прогностичну ефективність з AUC 0,803. **Висновки.** У статті підкреслено критичну потребу в точних прогностичних моделях розвитку ГПН, адаптованих до конкретних популяцій. Це дослідження спрямоване на розробку нової регіональної прогностичної моделі для пацієнтів із COVID-19, із зосередженням на популяції України. У моделі використовуються дані для поліпшення прогнозування ризиків і результатів лікування пацієнтів, з акцентом на важливості раннього виявлення й стратифікації осіб із високим ризиком.

Ключові слова: смертність; гостре пошкодження нирок; пневмонія; С-реактивний білок; прогностична шкала Падуї; лімфоцити; дихальна недостатність; громадське здоров'я; госпіталізація; результати лікування; фактори ризику; моделювання