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THE IMPACT OF THE KIDNEY FUNCTION ON THE PROGRESSION AND OUTCOMES OF CORONAVIRUS TREATMENT

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Summary

Introduction. Manifestations of kidney damage in coronavirus disease vary from asymptomatic proteinuria to acute kidney damage that requires renal replacement therapy. Decreased glomerular filtration rate (GFR) is associated with worse prognosis and increased in-hospital mortality.
Aim. To compare the clinical and laboratory characteristics of the course and the results of the coronavirus disease treatment in hospitalized patients depending on the GFR.
Materials and methods. The study included 243 hospitalized patients with moderate and severe coronavirus disease aged from 18 to 88 years, among them 110 females and 133 males. All patients were segregated into two groups depending on GFR: 1st group – 132 individuals with GFR > 60 mL/min per 1.73m^2, 2nd group – 111 individuals with GFR <60 mL/min per 1.73m^2. A general clinical examination and laboratory tests (PCR for the detection of SARS-CoV-2 virus, general blood test, glucose, liver and kidney markers, coagulation tests, d-dimer, interleukin-6, ferritin, procalcitonin, and determination of albumin and creatinine in urine with calculation of their ratio), instrumental studies (computed tomography or multi-positional radiography of the thoracic organs, pulse oximetry) were performed. The CKD-EPI (2021 update) formula was used to calculate GFR. Comparison of groups was performed by means of the Mann-Whitney U-test. Categorical data were presented as proportions and analyzed using the Chi-square test. The results were considered statistically reliable at $p<0.05$.
Results. Among the cohort of patients with diminished GFR, there was a notable prevalence of symptoms such as hemoptysis, hematuria, hypertension, and pronounced weakness. Additionally, higher levels of serum CRP, interleukin-6, and procalcitonin were significantly more frequent in this group. It was found that 7 patients (5.3 %) among the patients of the 1st group died, while 22 patients (19.8 %) died in the 2nd group, $p=0.0005$.
Conclusions. In patients with reduced GFR, such symptoms as hemoptysis, hematuria, hypertension and severe general weakness were significantly more often observed. Laboratory indicators included higher levels of CRP, interleukin-6, procalcitonin, and albumin/creatinine ratio, which indicated a more severe course and activity of the inflammatory process. The mortality rate in patients with reduced GFR was 19.8 % and was significantly higher than in the group with normal GFR – 5.3 %, $p=0.0005$.

Keywords: **COVID-19, creatinine, Glomerular Filtration Rate**

INTRODUCTION

The global coronavirus disease pandemic has presented a significant challenge for healthcare professionals worldwide. Given that the primary portals of entry for SARS-CoV-2 infection are the epithelium of the upper respiratory tract and, to a lesser extent, the stomach and intestines, the predominant clinical

manifestations of the disease often revolve around symptoms linked to respiratory and gastrointestinal tract damage. The manifestations of respiratory tract impairment ranged from anosmia to symptoms such as coughing and shortness of breath, indicative of acute respiratory failure. Nausea, vomiting or diarrhea were also observed quite often. Research has demonstrated that the SARS-CoV-2 virus exhibits a cytopathic effect on the kidneys, but it is practically impossible to establish the clinical manifestation of renal dysfunction, and the parameters of renal dysfunction were usually not used to make decisions regarding the tactics of patient management (hospitalization, transfer to the intensive care unit, prescription of antiviral drugs or immunosuppressants) [1, 2].

Renal disease in patients with COVID-19 may present as acute lesions, hematuria or proteinuria and is associated with an increased risk of mortality. In the study by Bowe B. et al., renal dysfunction was associated with the need for mechanical ventilation and with a longer length of hospital stay. About half of patients with acute kidney injury (AKI) failed to achieve full recovery of their function before discharge from hospital [3].

In the «eGFR-COV19 study» by the team of scientists in multivariate analysis, the value of GFR<60 ml/min/1.73m^2 was significantly associated with in-hospital mortality. GFR was not inferior in predicting in-hospital death compared to serum IL-6 levels [4].

Given that specific biomarkers of kidney dysfunction in coronavirus disease are lacking, earlier and more intensive strategies to monitor kidney function may be beneficial for those with COVID-19.

AIM

The aim was to compare the clinical and laboratory characteristics of the course and the results of coronavirus disease treatment in hospitalized patients depending on the glomerular filtration rate.

MATERIALS AND METHODS

Therefore, a total of 243 patients with moderate to severe coronavirus disease, receiving inpatient care at the Clinical Municipal Communal Emergency Hospital in Lviv between 2020 and 2021, were included in the study. The diagnosis of coronavirus disease was confirmed based on the following criteria: 1) positive PCR testing for SARS-CoV-2 in a nasopharyngeal swab; 2) documented lung abnormalities; 3) detection of IgM antibodies against the coronavirus in patients suspected of SARS-CoV-2 infection according to the X-ray CORADS scale scoring 4-6 points; 4) positive epidemiological history.

The sampling was conducted according to the following inclusion criteria: verified diagnosis – coronavirus disease; documented lung damage; age from 18 to 90 years; written consent. Exclusion criteria comprised refusal to sign the informed consent or non-compliance with prescribed treatments, asymptomatic or mild course of coronavirus infection, chronic kidney disease stage 3, severe or decompensated heart failure; decompensated diabetes, oncological diseases in the active phase, mental diseases, pregnancy or lactation.

The gender structure of this cohort was presented as follows: women – 110 (45.3 %), men – 133 (54.7 %). The average patient age was 63.0 (52.0;71.0) years. The majority of patients comprised elderly individuals (aged 60-74 years), accounting for 111 patients (45.7 %).

All patients were divided into two groups depending on GFR: 1st group – 132 people with GFR > 60 mL/min per 1.73m^2, 2nd group – 111 people with GFR <60 mL/min per 1.73m^2.

Upon admission, patients underwent a comprehensive assessment including recording of complaints, medical history, past medical records, epidemiological history, evaluation of risk factors for severe COVID-19, prior treatments, standard physical examination, and blood sample collection. All patients received comprehensive treatment for coronavirus and associated diseases, as well as oxygen therapy if necessary.

To assess the level of oxygen saturation in blood, pulse oximetry was performed using a pulse oximeter Pro Zoneo Med 3.0 of the company «oMed» (USA).

Real-time PCR for the detection of the SARS-CoV-2 virus was performed using the test system «SARS-CoV-2-PCR» of the company «XEMA» (Ukraine) with a sensitivity of 5 copies per reaction on the thermocycler analyzer «q TOWER3» of the company «AnalytikJena®» (Germany). To determine the presence of IgM antibodies to the SARS-CoV-2 coronavirus, express tests «CitoTest Covid-19 Ag» of the «Pharmasco» company (Ukraine) were used.

Hematological, biochemical blood tests and coagulation tests were performed following standard protocols and generally accepted methods. D-dimer was determined on the automatic immunoenzyme analyzer «APE ELITE» of the company «Das S. R.L.» (Italy) using the «D-DimerAccuBind ELISA Microwells» kits of the «Monobind» company (USA). IL-6 was determined using the test system «IL-6 TestCassette, FI-IL6-402» of HangzhouAllTestBiotechCo., Ltd. (China) on the immunofluorescence analyzer «NovatrendTM Fluorescence Immunoassay Analyzer» of the company «Hangzhou All Test BiotechCo., Ltd.» (China). Ferritin was determined on an automatic immunochemoluminescence analyzer «Abbott ARCHITECT i1000SR, company «ABBOTT®» (USA) using «ARCHITECT FerritinReagentKit» kits, company «ABBOTT® IRELAND» (USA-Ireland).

To assess the functional state and establish a possible diagnosis of chronic kidney disease in patients with coronavirus disease, according to the 2012 KDIGO recommendations, the CKD-EPI (2021 update) formula was used [5]:

$$GFR = 141 * \min(Scr/\kappa,1)\ \alpha * \max(Scr/\kappa, 1)-1.209 * 0.993Age * 1.018\ [\text{if female}] * 1.159\ [\text{if black}].$$

Scr is serum creatinine (mg/dL), κ is 0.7 for females and 0.9 for males, α is –0.329 for females and –0.411 for males, min indicates the minimum of Scr/κ or 1, and max indicates the maximum of Scr/κ or 1.

A decrease in GFR <60 mL/min per 1.73m^2 was considered clinically significant.

Multi-position radiography of the thoracic organs was performed on a Shimadzu Flexavision F3 digital X-ray machine, manufactured by Shimadzu Corp. (Japan).

CT of the patients' lungs was performed on a multispiral tomograph «Philips Ingenuity CT 64» of the Philips company (Netherlands). To classify patients with suspected COVID-19, the CO-RADS scale, proposed by the COVID working group of the Dutch Radiological Society, was used [6].

The results are given as mean values with statistical error. The values with normal distribution are presented as confidence interval (95 %); and the values, where distribution significantly differs from normal, are presented as intervals of 25 % and 75 % percentiles. Comparison of groups was performed by means of the Mann-Whitney U-test. Categorical data were presented as proportions and analyzed using the Chi-square test. The results were considered statistically reliable at $p<0.05$.

RESULTS

Most patients were admitted to hospital within 8 to 14 days from the onset of the disease – 143 patients (58.8 %), while 96 patients (39.5 %) were hospitalized within 1-7 days, and only 4 patients (1.6 %) in 15-21 days. Before hospitalization, patients commonly underwent outpatient treatment, which typically involved the administration of nonsteroidal anti-inflammatory drugs, antibiotics, anticoagulants and antiplatelet agents, antivirals, glucocorticosteroids, vitamins, and decongestants. In addition, the patients were administered medications for the management of pre-existing conditions, particularly antihypertensive, antianginal, hypoglycemic agents, and diuretics.

As presented in table 1, upon admission, patients from both groups reported experiencing fever, shortness of breath, cough, chest pain, nasal congestion and anosmia and other non-specific symptoms with the same frequency. The following symptoms were statistically different in frequency: hemoptysis, hematuria, hypertension and extreme weakness.

Table 1

Symptoms in patients with coronavirus disease depending on GFR

	1st group, n=132	2nd group, n=111	P
Dyspnea	105 (79,5 %)	93 (83,8 %)	0,39
Cough	119 (90,2 %)	91 (82,0 %)	0,64
Fever	102 (77,3 %)	89 (80,2 %)	0,58
Hypertension	93 (70,5 %)	95 (85,6 %)	0,005
Chest pain	87 (65,9 %)	76 (68,5 %)	0,67
Hemoptysis	7 (5,3 %)	27 (24,3 %)	0,00002
Hematuria	2 (1,5 %)	8 (7,2 %)	0,03
Nausea/vomiting	15 (11,4 %)	10 (9,0 %)	0,55
Diarrhea	8 (6,1 %)	14 (12,6 %)	0,07
Myalgia/arthralgia	66 (48,5 %)	58 (52,3 %)	0,56
Anosmia	46 (34,8 %)	52 (46,8 %)	0,06
Nazal congestion	88 (66,7 %)	80 (72,0 %)	0,36
Sore throat	51 (38,6 %)	42 (37,8 %)	0,90
Lymphadenopathy	6 (4,5 %)	8 (7,2 %)	0,38
Rash	1 (0,8 %)	3 (2,7 %)	0,24
Fatigue	99 (7,5 %)	103 (92,8 %)	0,0002

Regarding laboratory parameters, patients with reduced GFR, in addition to the expected significant difference in creatinine, urea, and urinary albumin-creatinine ratio, had significantly higher levels of serum CRP, interleukin-6, and procalcitonin, which are predictors of a more severe course and worse prognosis (table 2).

The effectiveness of treatment for hospitalized patients was further assessed by comparing the mortality rates between the groups. Thus, it was revealed that among the patients of the 1st group, 7 patients died (5.3 %), while in the 2nd group, 22 patients died (19.8 %), p=0.0005 (fig. 1).

Table 2

Laboratory parameters in patients with coronavirus disease depending on GFR

	1st group, n=132	2nd group, n=111	P
RBC, 10*12/l	4,7 (4,3;5,2)[2]	4,7 (4,2;5,1)[2]	0,49
WBC, 10*9/l	7,1 (4,7;9,6)[2]	7,8 (5,5;11,4)[2]	0,12
Platelets, 10*9/l	217,0 (173,0;279,0)[2]	202,0 (164,0;263,0)[2]	0,89
Haemoglobin, g/l	141,0 (127,0;152,0)[2]	136,5 (126,0;148,0)[2]	0,24
ESR, mm/h	28,4 (25,5;31,3)[1]	27,0 (14,0;41,3)[2]	0,57
Glucose, mmol/l	5,3 (4,4; 6,8)[2]	6,0 (4,4;8,0)[2]	0,20
ALT, U/l	28,2 (17,9;45,0)[2]	25,1 (14,3;45,2)[2]	0,89
AST, U/l	28,1 (21,0;34,3)[2]	28,2 (19,6;42,9)[2]	0,43
Bilirubin, mcmol/l	9,7 (6,9;13,8)[2]	9,6 (7,4;14,1)[2]	0,48
Creatinine, mcmol/l	92,9 (90,1;95,7)[1]	123,5 (107,0;152,0)[2]	<0,0001
Urea, mmol/l	6,5 (6,1;6,9)[1]	8,9 (7,3;13,4)[2]	<0,0001
CRP, mg/l	36,0 (21,0;72,0)[2]	48,0 (33,0;76,0)[2]	0,04
Fibrinogen, g/l	5,5 (5,1;5,9)[1]	5,2(4,8;5,6)[1]	0,24
D-dimer, ng/ml	420,0 (190,0;1156,4)[2]	447,0 (190,0;1968,2)[2]	0,33
Interleukin-6, ng/ml	7,8 (6,0;11,3)[2]	8,3 (4,6;14,9)[2]	0,01
Ferritin, ng/ml	418,9 (340,3;497,5)[1]	435,6 (351,1;520,1)[1]	0,85
Procalcitonin, ng/ml	0,1 (0,02;0,2)[2]	0,14 (0,02;0,8)[2]	0,02
Albumin/creatinine ratio, mg/mmol	2,0 (1,1;4,5)[2]	12,5 (3,9;34,1)[2]	<0,0001

[1] – values with normal distribution, M (M-CI; M+CI).
[2] – values, where distribution significantly differs from normal, Me (25 %;75 %).

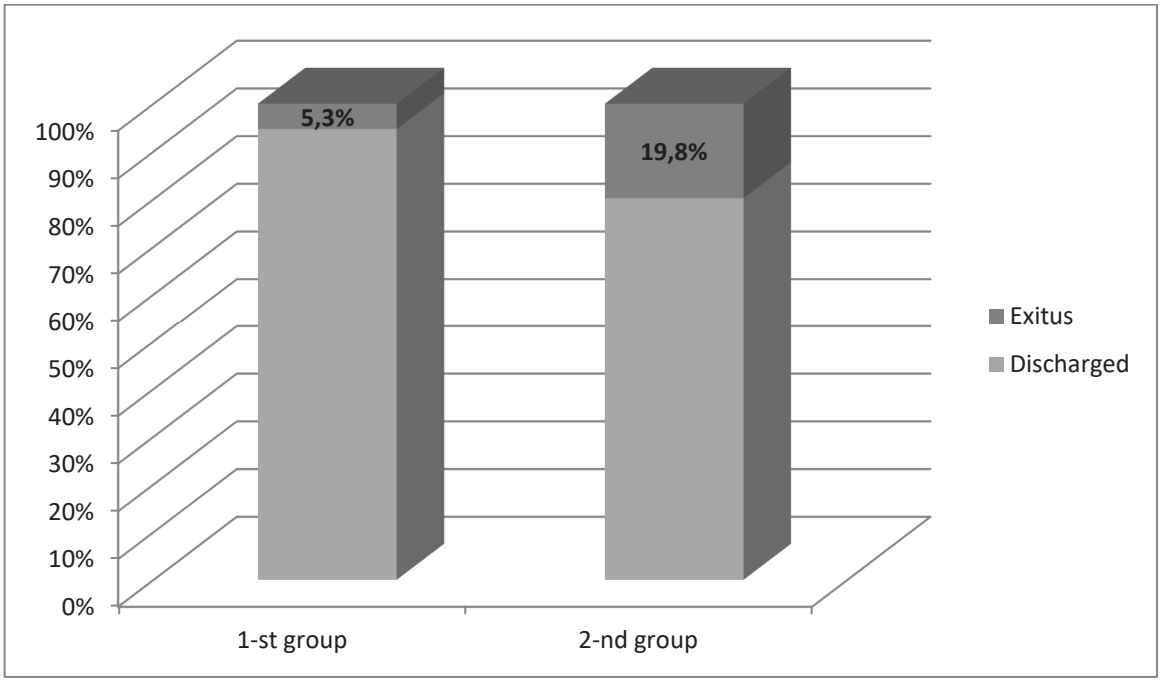

Figure 1. Comparison of mortality rates in hospital, p =0.0005.

DISCUSSION

Currently, numerous scientific studies primarily focus on exploring acute kidney injury (AKI) in COVID-19 patients, both with or without previously diagnosed chronic kidney disease (CKD), as well as COVID-19 in CKD patients undergoing hemodialysis treatment [7, 8, 9].

Clinically, renal injury associated with COVID-19 is a heterogeneous syndrome. Kidney damage in patients with COVID-19 is common and can range from proteinuria, hematuria to acute kidney injury requiring renal replacement therapy. Microalbuminuria or hematuria may be signs of subclinical AKI, even in the absence of a concomitant increase in serum creatinine or a decrease in GFR. AKI cannot always be recognized, as it can be asymptomatic. Severe AKI is usually an independent risk factor for death because it can cause systemic effects on other vital organs, including the lungs, heart, liver, brain, and immune system [10].

The lack of prehospital serum creatinine measurements often complicated the ability to identify existing CKD and created challenges for reliable detection and staging of AKI. It was extremely difficult to differentiate AKI that arose de novo and AKI superimposed on already existing CKD in patients hospitalized for coronavirus disease. Thus, in our study, we divided patients into groups considering a clinically significant decrease in GFR <60 mL/min per 1.73m^2.

In one of the largest ongoing studies on AKI associated with COVID-19, known as the ISARIC WHO CCP-UK Study, researchers have identified risk factors for AKI: older age, combination of several chronic diseases, male gender, tachypnea, hypoxemia, proteinuria, higher pro-inflammatory markers, artificial lung ventilation, the dosage of vasopressors and nephrotoxic drugs [11].

In a study by Portolés J. et al. in Spain, 21 % of patients with COVID-19 had elevated creatinine on admission, of which 43.5 % had pre-existing CKD. The in-hospital mortality rate was higher in patients with elevated creatinine (32.4 %), in patients with a history of CKD (41.1 %), and in those who developed AKI during hospitalization (15.9 %) compared to patients with a normal level of creatinine (5.8 %). Kaplan-Meier analysis found higher in-hospital mortality in patients with elevated creatinine on admission, in patients with hematuria, and in those who develop AKI, but not in individuals with proteinuria or leukocyturia [12].

An observational study by Russo E. and co-authors conducted in Italy revealed that among 777 patients hospitalized for coronavirus disease, AKI developed in 176 (22.6 %), of which 79 (45 %) already had CKD, and 21 (12 %) individuals required renal replacement therapy. Among patients with AKI, 111 died (63 %), and its occurrence increased the risk of death by 60 % (HR 1.60 [95 % IC 1.21-2.49] p = 0.002) independently of other factors [13].

In our investigation, we opted not to categorize patients by nosology but instead concentrated on the actual glomerular filtration rate (GFR) determined by creatinine levels. Our findings revealed that the mortality rate among patients with GFR of less than 60 mL/min per 1.73m^2 stood at 19.8 %, a figure nearly fourfold higher than the 5.3 % observed in the group with normal GFR. These results align with previous studies conducted during the initial phase of the coronavirus disease outbreak.

Although there has been a decline in the incidence of acute renal failure cases and the necessity for renal replacement therapy following the initial surge of coronavirus disease [14, 15], the issue of renal dysfunction in the context of coronavirus disease and strategies to avert irreversible kidney damage continue to be pertinent.

CONCLUSIONS

In individuals with diminished kidney function, symptoms such as hemoptysis, hematuria, hypertension, and marked general weakness were notably more prevalent. Regarding laboratory parameters, elevated levels of PSA, interleukin-6, procalcitonin, and albumin/creatinine ratio were observed. The mortality rate among patients with reduced GFR stood at 19.8 %, significantly surpassing the 5.3 % observed in the normal GFR group (p=0.0005).

Perspectives for further research. Explore early, distinctive markers of kidney dysfunction in coronavirus disease and strategies to avert irreversible kidney damage. Develop a predictive model for identifying severe disease progression and in-hospital mortality in coronavirus disease based on indicators of kidney function involvement.

COMPLIANCE WITH ETHICAL REQUIREMENTS

This research is a prospective cohort study. Clinical trial was conducted in accordance with the Declaration of Helsinki, The Convention for the Protection of Human Rights and Biomedicine, Legislation of Ukraine and agreed by commission on ethics of research, experimental development and scientific works of Danylo Halytsky Lviv National Medical University: No. 10 of December 20, 2021. All patients signed an informed consent before the study.

CONFLICT OF INTEREST STATEMENT

This article is a part of the complex research topic of the Department of Therapy No. 1, Medical Diagnostics and Hematology and Transfusiology of the Faculty of postgraduate education «Characteristics of pathogenesis, diagnosis, and treatment of cardiovascular, digestive, endocrine, and respiratory system disorders in both clinical settings and experimental contexts» (state registration number 0120U002142).

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Резюме

ВПЛИВ ФУНКЦІОНАЛЬНОГО СТАНУ НИРОК НА ПЕРЕБІГ І РЕЗУЛЬТАТИ ЛІКУВАННЯ КОРОНАВІРУСНОЇ ХВОРОБИ

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Вступ. Прояви ураження нирок при коронавірусній хворобі варіюють від безсимптомної протеїнурії до гострого пошкодження нирок, яке потребує замісної ниркової терапії. Зниження швидкості клубочкової фільтрації (ШКФ) асоціюється з гіршим пронозом та підвищеним рівнем госпітальної смертності.
Мета. Порівняти клініко-лабораторні особливості перебігу та результати лікування коронавірусної хвороби у госпіталізованих пацієнтів залежно від ШКФ.
Матеріали та методи. У дослідження було залучено 243 пацієнти госпіталізовані з приводу коронавірусної хвороби середнього та важкого ступеня віком від 18 до 88 років. Серед них було 110 жінок та 133 чоловіків. Усі пацієнти були поділені на 2 групи залежно від ШКФ: 1 група – 132 особи з ШКФ > 60 мл/хв/1.73м^2, 2 група – 111 осіб з ШКФ <60 мл/хв/1.73м^2. Проводили загально-клінічні та лабораторні обстеження (ПЛР на виявлення вірусу SARS-CoV-2, загальний аналіз крові, глюкоза, печінкові та ниркові маркери, коагулограма, д-димер, інтерлейкін-6, феритин, прокальцитонін та визначення альбуміну та креатиніну в сечі з розрахунком їхнього співвідношення), інструментальні дослідження (комп'ютерна томографія або поліпозиційна рентгенографія органів грудної клітки, пульсоксиметрія). Для розрахунку ШКФ використовували формулу CKD-EPI (2021). Для порівняння двох груп використовували критерій Манн-Уїтні. Відносні величини представлено у відсотках, групи порівнювали за критерієм χ^2 Пірсона. Результати вважалися статистично достовірними при p<0,05.
Результати. У групі пацієнтів зі зниженою ШКФ достовірно частіше спостерігали кровохаркання, гематурію, гіпертензію, виражену слабкість та вищі рівні сироваткових СРП, інтерлейкіну-6 та прокальцитоніну. Було виявлено, що серед пацієнтів 1 групи померло 7 хворих (5,3 %), тоді як у 2-ій групі померло 22 хворих (19,8 %), p=0,0005.
Висновки. У пацієнтів зі зниженою ШКФ достовірно частіше спостерігалися такі симптоми як кровохаркання, гематурія, гіпертензія та виражена загальна слабкість. Серед лабораторних показників були вищі рівні сироваткових СРП, інтерлейкіну-6, прокальцитоніну та альбумін-креатинінове співвідношення в сечі, що свідчило про важчий перебіг та активність запального процесу. Рівень летальності у пацієнтів зі зниженою ШКФ становив 19,8 % і був достовірно вищим, ніж у групі з нормальною ШКФ – 5,3 %, p=0.0005.

Ключові слова: **COVID-19, креатинін, швидкість клубочкової фільтрації**