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Comparative analysis of the glomerular filtration rate effect on the course of COVID-19 in patients with coronary heart disease with and without concomitant coronavirus disease

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Abstract. The research relevance is determined by the COVID-19 pandemic, which has led to serious medical consequences, including high levels of infectiousness, development of diseases accompanied by complications of kidney and cardiovascular system function, and increased mortality. Therefore, the research aims to study and compare the impact of glomerular filtration rate on the course of COVID-19 in patients with and without coronary heart disease. A retrospective analysis of 410 patients with coronavirus was conducted, who were divided into 2 groups: those with chronic coronary heart disease and those without this disease. During the hospital period (14.7 ± 5.3 days), the composite endpoint of all-cause and cardiovascular deaths in combination with major adverse cardiovascular events was assessed. The thresholds for glomerular filtration rate associated with an increase in the incidence of the composite endpoint were determined: for patients with COVID-19, less than 35 mL/min×1.73 m^2 ($p < 0.01$); for patients with coronary heart disease and COVID-19, less than 60 mL/min×1.73 m^2 ($p<0.01$). Independent predictors of decreased renal filtration capacity in patients in group 1 were: age over 65 years, type 2 diabetes mellitus, high cholesterol, D-dimer, C-reactive protein, and ferritin. Patients in group 2 were adversely affected by type 2 diabetes mellitus, arterial hypertension, and high levels of D-dimer and C-reactive protein ($p < 0.05$). The difference was explained by the influence of the applied therapy on the anticoagulant and renin-angiotensin systems. This study will allow to stratify patients with coronavirus in terms of renal impairment and risk factors, as well as to identify effective strategies for their management depending on the glomerular filtration rate

Keywords: arterial hypertension; risk factors; comorbidity; mortality

Introduction

The COVID-19 pandemic made a significant impact on Ukraine, given its influence on the healthcare system and public health. The increase in the number of cases has led to an excessive burden on the healthcare system, including a shortage of beds, intensive care, medical equipment, and staff [1]. As reported by I. Seriakova *et al.* [2], the pandemic has also led to the postponement or cancellation of necessary medical procedures and surgical interventions, which has led to an increase in cardiovascular and renal pathologies. The increased mortality rate among patients with pre-existing cardiovascular and renal diseases indicates a serious impact of the pandemic on these body systems. In addition, A. Shishkin *et al.* [3] found that the pandemic has increased the risk of severe COVID-19 complications for the target group of patients with cardiovascular and renal diseases, such as pneumonia and thrombosis, and is accompanied by increased levels of stress and psychological problems, which can affect the general condition of these systems. Thus, research and monitoring are critical to understanding and mitigating the negative impact of the COVID-19 pandemic on the cardiovascular and renal health of the Ukrainian population.

As of 22 July 2023, about 5.5 million people were infected in Ukraine, of whom 112.5 thousand died, which

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accounts for a 2% fatality rate [4]. D. Chumachenko & T. Chumachenko [5] point out that due to Russia's full-scale armed invasion of Ukraine, the epidemiological situation with coronavirus infection has only worsened – the destruction of infrastructure and human resources has made it difficult to diagnose, identify cases, impossible to hospitalise, and lack of funds for adequate treatment and prevention. Therefore, even though the incidence of COVID-19 is on a downward trend and has lost its status as a global emergency, Ukraine still faces risks associated with this infection in both the short and long term.

The consequences of COVID-19 are pathophysiologically associated with the development of a cytokine storm, which causes endothelial dysfunction and endotheliitis, which in turn leads to the development of microvascular thrombi, ischaemia, and multiple organ failure, which determines the multisystemic nature of the lesion [6]. Several studies confirm the fact that patients with cardiovascular disease and COVID-19 are prone to severe disease and have a higher risk of death [7-9]. Similar mechanisms are responsible for direct and indirect renal dysfunction, characterised by a decrease in renal filtration capacity and organic damage [10]. V.P. Chavda *et al.* [11] point out that COVID-19 in combination with other therapeutic diseases (obesity, type 2 diabetes mellitus, coronary heart disease, renal failure, fatty liver, etc.) is more prone to severe course of all associated diseases, so comorbidity is an important predictor of complications. Considering the relationship between coronary heart disease (CHD) and chronic kidney disease (CKD), the conference Kidney Disease: Improving Global Outcomes (KDIGO) conference in 2020, presents data showing that the progression of cardiovascular damage increases with the progression of renal dysfunction, despite the correction of traditional cardiovascular risk factors [12].

Thus, it is important to study the relationship between cardiovascular risks, renal damage and coronavirus infection. The research aims to study and compare the effect of glomerular filtration rate on the course and development of COVID-19 complications in patients with CHD and COVID-19 without CHD.

Materials and Methods

The study retrospectively analysed data from 410 patients with coronavirus disease, treated at Kyiv City Clinical Hospital No. 18 and the private medical centre Medbud from 2 March 2020 to 31 December 2022. The diagnosis of COVID-19 was confirmed by detecting the ribonucleic acid (RNA) of the SARS-CoV-2 pathogen in samples from the upper respiratory tract using polymerase chain reaction (PCR). Multidetector computed tomography (MDCT) was used to assess the presence and extent of COVID-19-related lung damage. Verification of the diagnosis of CHD was performed according to current guidelines [13]. COVID-19 and its complications were treated following the guidelines of the Ministry of Health of Ukraine dated 2 April 2020, No. 762 [14]. All patients were informed about their participation in the study and signed an informed consent to the processing of personal data.

Exclusion criteria for the study include the following conditions: acute coronary syndrome, previously documented acute cerebrovascular accident, type 1 diabetes mellitus, severe and/or decompensated major comorbidities (including malignancies), history of heart valve replacement, vaccination against COVID-19, and lack of informed consent.

Among the 210 patients with COVID-19 without CHD (group 1), there were 85 women (40%) with a mean age of 52 ± 21.6 years and 125 men (59%) with a mean age of 49 ± 21 years. There were 200 patients with CHD in combination with COVID-19 (group 2), including 124 men (62%) with a mean age of 62.4 ± 12.6 years and 76 women (38%) with a mean age of 62.2 ± 9.5 years. The control group consisted of 35 healthy volunteers, and 90 patients with coronary heart disease without COVID-19 who matched the study sample in terms of age distribution and gender ratio were examined as a comparison.

The standard examination of all patients included general clinical examination of blood and urine, biochemical blood tests (lipid spectrum, blood glucose, creatinine), cardiac ultrasound, and electrocardiography. The glomerular filtration rate (GFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula (1) using an online calculator:

$$GFR = 141 \times \min\left(\frac{\text{Scr}}{\kappa}, 1\right)^{\alpha} \times \max\left(\frac{\text{Scr}}{\kappa}, 1\right)^{-1.209} \times \\ \times 0.993^{Age} \times 1.018^{[\text{if female}]} \times 1.159^{[\text{if black}]}, \quad (1)$$

where Scr – serum creatinine (mg/dl), κ – 0.7 for women and 0.9 for men, α – -0.329 for women and -0.411 for men, min indicates the minimum of Scr/κ or 1, and max indicates the maximum of Scr/κ or 1.

The development of cardiovascular events was assessed during the hospital period, which averaged 14.7 ± 5.3 days. The date of hospitalisation was considered the start of observation. The composite endpoint (CEP) was all-cause death (including cardiovascular) and major adverse cardiovascular events (non-fatal myocardial infarction (MI), acute cerebrovascular accident, pulmonary embolism, and acute left ventricular failure > Killip class I).

Statistical processing was performed using Statistica v.7.0 and SPSS Statistics v.27.0. Continuous variables are expressed as mean (M) and standard deviation (σ), and their differences were assessed using analysis of variance or unpaired t-test. Pearson's $\chi 2$ test was used for categorical variables. The Cox model was used to estimate the odds ratio (OR) and confidence interval (CI) within the framework of multivariate regression analysis. The log-rank test and the Kaplan-Meier method were used to analyse the significance of differences in survival. The difference in the values at $p < 0.05$ was considered statistically significant.

All procedures performed in studies involving human subjects complied with the ethical standards of the institutional and national research committee, as well as the Declaration of Helsinki [15]. The study was approved by the National Ethics Committee of the Bogomolets National Medical University.

Results

The clinical and anamnestic characteristics of the study patients in group 1 (200 patients with CHD+COVID-19) and group 2 (210 patients with COVID-19) are shown in Table 1. The majority of patients were male (60.7%) and over 60 years of age, and since the subjects were selected from the usual contingent treated during the COVID-19 pandemic, this preliminarily indicates a greater vulnerability of men in the older age group. A retrospective analysis of the medical records revealed that more than half of the patients had hypertension (64%) and chronic heart failure in 42% of cases, with most of them having preserved ejection fraction. CKD was observed in 11.2% of patients, with only one-third (31.0%) of patients having normal GFR and 42.2% with slightly reduced GFR. Given the fact that the study was based on a retrospective analysis, the stage of CKD was determined solely by GFR, and the level of albuminuria could not be considered. The body mass index revealed that obesity was present in almost half of the cases (42.0%) and dyslipidaemia in 24%, although there were 48.8% of patients with CHD in the study cohort, which may indicate the effectiveness of the therapeutic intervention.

Table 1. Clinical and anamnestic characteristics of patients with COVID-19

Indicators	Indicator value
Age, years (M±SD)	61.2 ± 10.7
Men, n (%)	249 (60.7%)
Women, n (%)	161 (39.3%)
Body mass index, kg/m^2	25.9 ± 7.3
Smokers, n (%)	86 (21%)
Arterial hypertension, n (%)	262 (64%)
Type 2 diabetes, n (%)	114 (28%)
History of myocardial infarction, n (%)	64 (15.6%)
History of left ventricular anterior wall myocardial infarction, n (%)	45 (11%)
Coronary arteriography, n (%)	190 (46%)
Glucose, mmoL/L	5.8 ± 2.3
Dyslipidaemia, n (%)	98 (24%)
Creatinine μmoL/L	112.7 ± 9.5
GFR ≥ 90 mL/min×1.73 m^2, n (%)	127 (31%)
GFR = 60-90 mL/min×1.73 m^2, n (%)	173 (42%)
GFR = 30-60 mL/min×1.73 m^2, n (%)	89 (21.7%)
GFR < 30 mL/min×1.73 m^2, n (%)	21 (5.3%)
Chronic heart failure, n (%)	172 (42%)
Release fraction, %.	43.5 ± 12.7
Duration of CHD, years (M ± SD)	7.5 ± 3.9
Angina pectoris class II-IV, n (%)	69 (17%)
Low molecular weight heparins (enoxaparin)	265 (64.6%)
Treatment before hospitalisation with a diagnosis of COVID-19:	
Acetylsalicylic acid	114 (27.8%)
Clopidogrel	86 (35.2%)
Angiotensin-converting enzyme inhibitors	112 (27.3%)
Beta-blockers	103 (42.2%)
Diuretics	62 (15%)
Calcium channel blockers	37 (9%)
Angiotensin-II receptor blockers	88 (21.5%)
Nitrates	74 (18%)
Statins	170 (41.4%)

Notes: M – mean value of the parameter; SD – standard deviation of the mean
Source: compiled by the author

Considering medications, it is worth noting that antithrombotic therapy with enoxiparin was performed in 98.7% of patients, and 92.3% required antibiotic therapy (ceftriaxone, levofloxacin mofloxacin) for the prevention and treatment of bacterial complications, and 66.5% of patients were prescribed glucocorticosteroids (dexamethasone, hydrocortisone, methylprednisolone), considering the pathogenetic features of the infectious process. Patients were prescribed antiviral drugs and an interleukin-6 inhibitor in circumstances related to their coronavirus infection. In cases where patients were infected with COVID-19, antiviral drugs such as remdesivir and favipiravir were prescribed in 29% of cases. In addition, an interleukin-6 inhibitor such as tocilizumab was used in 18% of patients to modulate the immune response and treat the inflammation that occurs with coronavirus. These measures were aimed at improving the clinical condition and treatment outcomes of these patients, especially in the context of the pandemic.

All patients with chronic coronary syndrome were taking antiplatelet, antianginal and statin therapy (acetylsalicylic acid, clopidogrel, nitrates and statins). It should be noted that the incidence of bleeding has not been studied. Angiotensin-converting enzyme inhibitors (ACEIs), angiotensin-II receptor blockers (ARBs) and β-blockers (BABs) were prescribed in 80.2% of patients, respectively, to correct chronic heart failure and hypertension. It is important to note that by the time of hospitalisation and treatment adjustment, less than half of the patients were taking the medications prescribed for their chronic comorbidities, and none of the patients with diagnosed type 2 diabetes (28%) were on glucose-lowering therapy.

A preliminary assessment of the possible association between the occurrence of the critical endpoint and the value of GFR is shown in Figure 1. In patients of both groups, the frequency of adverse outcomes of COVID-19 had an inverse correlation with the kidney function indicator. However, it was found that in patients with COVID-19 with a GFR of less than 60 mL/min×1.73 m^2, the incidence of CEP was significantly higher than in patients with CHD+COVID-19. Thus, among patients in group 2 with a GFR of 45-59 mL/min×1.73 m^2, CEP occurred in 15.1% versus 10.2% in patients in group 2, in patients with a GFR of 31-44 mL/min×1.73 m^2 – in 17.2 and 14.6%, and patients with a GFR ≤ 30 mL/min×1.73 m^2 – in 27.5 and 21.6%, respectively ($p < 0.05$). This difference in favour of CHD seemed unclear, as a history of cardiovascular disease would increase the percentage of patients with the primary endpoint. When re-analysing (30 days later) the clinical and anamnestic characteristics of patients, it was suggested that ACEIs and ARBs had a positive effect on renal function and cardiovascular risk reduction.

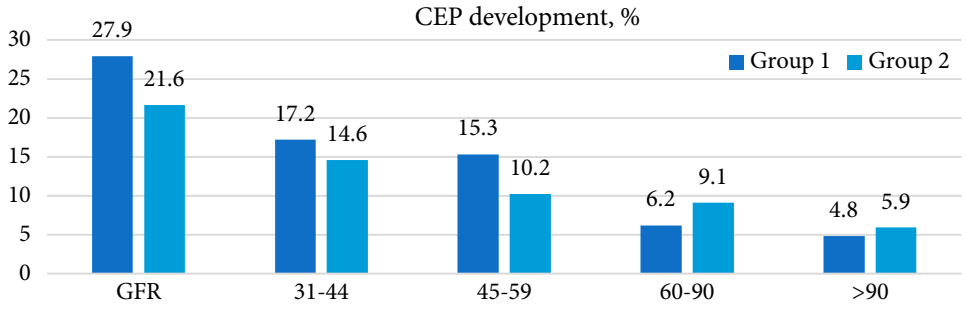

Figure 1. Incidence of the composite endpoint depending on GFR in patients in groups 1 (COVID-19) and 2 (CHD+COVID-19)

Source: compiled by the author

In order to determine the probability of developing the combined endpoint in patients with COVID-19 and CHD+COVID-19, depending on the value of GFR, cut-off values were sought separately for each group. After calculating the odds ratio for the onset of CEP at stepwise values of GFR, the following thresholds were established that were associated with an unfavourable course of COVID-19:

- For group 1, this value was GFR ≤ 35 mL/min×1.73 m^2 (OR 3.7; 95% CI 1.8-7.6, $p < 0.01$);
- For group 2 – ≤60 mL/min×1.73 m^2 (OR 4.1; 95% CI 2.1-9.8, $p < 0.01$).

When constructing Kaplan-Meier curves in patients with COVID-19 19 without CHD with a GFR ≤ 34 mL/min×1.73 m^2 compared with patients with a GFR > 34 mL/min×1.73 m^2, a significantly lower survival rate with the development of a combined cardiovascular endpoint throughout the hospital period was found ($p = 0.042$), as shown in Figure 2(a). At the same time, a significantly higher proportion of patients with CHD+COVID-19 with the development of a combined cardiovascular event according to the logarithmic criterion was detected in patients with a GFR ≤ 52 mL/min×1.73 m^2 compared with those with a GFR > 52 mL/min×1.73 m^2 ($p = 0.04$) (Fig. 2(b)). Multivariate regression analysis revealed independent decrease predictors of GFR ≤ 34 mL/min×1.73 m^2 in group 1 and ≤52 mL/min×1.73 m^2 in group 2. These data can be interpreted in such a way that chronic renal hypoperfusion occurs in concomitant chronic coronary syndrome, and, accordingly, in concomitant infectious disease, compensatory mechanisms fail at a higher level than without concomitant cardiovascular disease, and a decision on renal replacement therapy should be made as soon as possible in such patients.

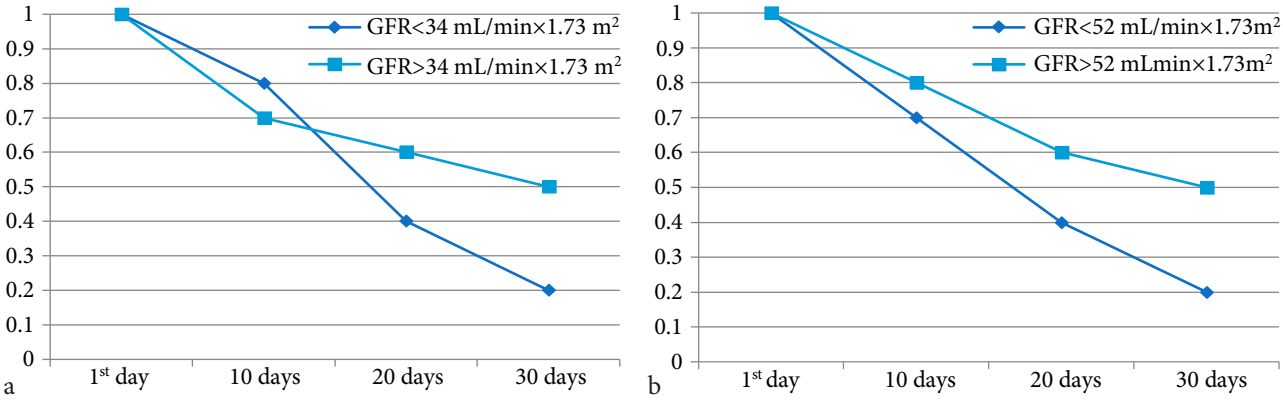

Figure 2. Comparison of survival in patients with COVID-19 in patients with threshold
Notes: a) GFR = 34 mL/min×1.73 m²; b) GFR = 52 mL/min×1.73 m². After the hospital period, patient monitoring was continued, so the time interval in the Figure comprises 30 days
Source: compiled by the author

For patients with COVID-19, important prognostic factors were age >60 years (OR 1.12; 95% CI 1.01-1.60), the presence of hypertension (OR 2.50; 95% CI 1.17-4.85), high D-dimer levels (OR 4.0; 2.02-5.20), type 2 diabetes mellitus (OR 2.75; 2.01-4.79), and high C-reactive protein levels (OR 1.94; 95% CI 0.89-3.81) (Fig. 3). For patients with the concomitant chronic coronary syndrome, age >60 years (OR 2.61; 95% CI 1.63-6.41), hypertension (OR 2.69; 95% CI 1.81-3.15), high D-dimer levels (OR 4.95; 3.15-6.80), type 2 diabetes mellitus (OR 2.15; 1.67-2.99), high ferritin (OR 3.85; 1.54-6.91) and high C-reactive protein (OR 3.01; 2.06-7.33) significantly influenced the decrease in GFR ($p < 0.05$) (Fig. 3). There was no significant effect of cholesterol level and gender on GFR in both groups. It is worth noting that elevated ferritin levels reflect the risk of renal dysfunction to a greater extent than elevated C-reactive protein levels, which is in favour of the former as a more representative laboratory prognostic marker. D-dimer, which is a fibrin degradation product, is indicative of hypercoagulability, and this indicator, as well as age over 60, was more pronounced in patients with concomitant coronary heart disease, which may be associated with chronic inflammation in the vessels and their atherosclerotic lesions.

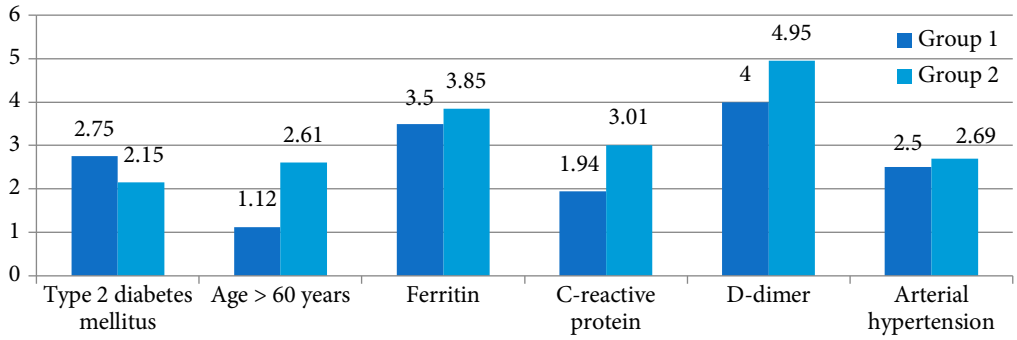

Figure 3. Odds ratio of GFR ≤ 34 mL/min×1.73 m² decrease in group 1 patients and ≤52 mL/min×1.73 m² in group 2 patients with each independent predictor)
Source: compiled by the author

At the same time, antithrombotic therapy in patients with COVID-19 without CHD had a favourable effect on the GFR (OR 0.58; 95% CI 0.31-0.97) ($p < 0.05$), but in the group with concomitant cardiovascular comorbidity, no such association was found. Thus, the clinical significance of the study is to identify the association between a decrease in glomerular filtration rate and an unfavourable prognosis of COVID-19 in patients with coronary heart disease, as well as to establish thresholds for GFR for each group, which can be used to separate patients at high risk of adverse cardiovascular events and death from all causes and cardiovascular causes. A special risk category is represented by patients with COVID-19 in combination with coronary heart disease and moderately reduced GFR ≤ 51 mL/min×1.73 m². It is necessary to consider the level of D-dimer, C-reactive protein, and ferritin to provide these patients with oxygen, low-molecular-weight heparins, glucocorticosteroids, high-dose statin therapy and timely administration of artificial lung ventilation and extracorporeal membrane oxygenation to eliminate hypoxia and thrombosis. Evaluation of the various factors that influence the improvement or deterioration of COVID-19 (including

medications) and maximising vaccination of people worldwide can play a vital role in controlling the spread of the infectious process and reducing the number of deaths.

Discussion

V. Mahalingasivam *et al.* [16] in their review study presented data from the United Kingdom that patients with a GFR of 30-60 mL/min×1.73 m^2 had a 1.3-fold higher risk of death associated with COVID-19, and in patients with a GFR <30 mL/min×1.73 m^2, this risk was 2.5 times higher than in patients with normal renal function. In this experiment, the incidence of CEP among patients in group 1 with a GFR <35 mL/min×1.73 m^2 was 3.7 times higher, which considers not only the onset of death but also the development of non-fatal cardiovascular complications, but this figure is still relatively higher. The authors noted that the lack of baseline data on renal function in the experiments creates problems in the classification of chronic kidney disease and acute kidney injury, which potentially impairs the generalisability of the results, which would be important to consider in this study.

In an experiment conducted by X. Han *et al.* in New York [17], 7.9% of hospitalised patients out of 5700 people with COVID-19 had a history of chronic kidney disease, and 9.7% of cases were fatal, which is significantly lower than the results obtained in this study. Further, the researchers cite the results of studies from China and draw attention to the fact that serum creatinine, acute kidney injury, proteinuria and haematuria were independent risk factors for mortality in COVID-19, which indicates that patients with kidney disease require increased attention and monitoring, especially for patients on haemodialysis. A meta-analysis by V. Liakopoulos *et al.* [18] demonstrated that the prevalence of CKD among patients with COVID-19 is 5.2% and is associated with a 3-fold increase in the severity of the infectious disease and a 2-fold increase in mortality.

Concerning acute kidney injury in COVID-19, according to a review study by K. Amann *et al.* [19], 20% of patients had acute renal symptoms, and 13.1% had a GFR <60 mL/min×1.73 m^2, which is also significantly less than in this study. Further, the authors, referring to the Australian registry, presented data on high overall mortality among patients with CKD and COVID-19 – 27.9% of infected dialysis patients and 6% of kidney transplant patients, which means a 1.28-fold increase in mortality compared to the control group. According to L. Yang *et al.* [20], the incidence of acute kidney injury among patients with COVID-19 is 27.17%, which increases the risk of mortality by 5.24 times and the development of severe conditions by almost 15 times. At the same time, the prevalence of CKD among patients with COVID-19 was 5.7%, which increases the risk of mortality by more than 2 times and the development of severe conditions by 1.87 times.

M. Brogan & M.J. Ross [21] note that up to 50% of patients requiring renal substitution therapy are asymptomatic, and only 47% have fever (compared to 90% in the general population), which requires increased vigilance in terms of COVID-19 infection, as well as monitoring the functional state of the kidneys. The study's author provides similar statistics, stating that the mortality rate in such patients increases by 20-30%. J.B. Wetmore [22] presented information that about 4.0% of patients on renal replacement therapy who are hospitalised for a cardiovascular event die in the hospital and another 4.5% of discharged patients die within the next 30 days. It has also been proven that not all patients with COVID-19 can be hospitalised and, therefore, are not included in trials, so the mortality rate is likely to be even higher. The scientist's study coincides with the results of this research, emphasising the fact that patients with COVID-19 in combination with various comorbidities, including coronary heart disease, have a worse tolerance of the lesions, which negatively affects the quality of kidney function.

Zh. Zheng *et al.* [23] concluded in their meta-analysis that in men over 65 years of age, hypertension, diabetes mellitus, and concomitant cardiovascular and respiratory diseases can significantly worsen the prognosis of COVID-19. The authors also noted qualitative laboratory prognostic markers, namely white blood cell count, aspartate aminotransferase, creatinine, procalcitonin, lactate dehydrogenase, high-sensitivity troponin I and D-dimer. The data obtained in this study are fully consistent with the analysed information from other sources, and it is possible to expand the range of the diagnostic programme in the future.

S. Shah & M.A. Sparks [24] also noted the role of hypertension and old age as risk factors for severe disease, and pathogenetically this is since SARS-CoV-2 penetrates host cells through the angiotensin-converting enzyme-2 receptors of the renin-angiotensin system, which in turn leads to the question of the effect of ACEIs and ARBs, which was also recorded in the present study, but was not subjected to further analysis, as it requires a separate study.

M.A. Podestà *et al.* [25] note that cardiovascular damage in patients with CKD and COVID-19 occurs in approximately 20-25% of cases, especially concerning thrombotic events, as the infectious disease is associated with a state of hypercoagulation. The authors present data with a wide range of risk rates – from 4.7 to 31%, but these cases also had a relationship with the D-dimer. P. Theofilis *et al.* [26] in their study confirm that, in addition to pneumonia and thromboembolism, renal dysfunction is a common and poor prognostic indicator associated with increased disease severity and mortality. The authors note that for the timely detection of renal damage, it is necessary to use inflammatory biomarkers not only in blood but also in urine and that urinary SARS-CoV-2 virus load may also be an early prognostic sign. However, similar findings have not been reported in other studies, which requires further discussion and study.

E.Y.M. Chung *et al.* [27] demonstrated the mortality rate among patients with CKD and COVID-19 was 32 deaths per 1000 person-weeks in their meta-analysis, which is an interesting unit of measurement that could unify patients by time of hospitalisation in subsequent studies.

According to this system, the risk of death in such patients increases 10-fold compared to people with CKD but no concomitant infection. According to R.M. May *et al.* [28], the incidence of diabetes mellitus and hypertension as clinical comorbidities was increased in patients with COVID-19 (40.4% and 72.5%, respectively), which differs from the data obtained in this study (28% and 64%, respectively).

J. Smolander & A. Bruchfeld [29] emphasise the importance of assessing the patient on admission with urine and creatinine diagnostic test strips, and subsequently, urine sedimentation and quantification of albuminuria if the results of previous tests are positive. The authors emphasise the need to continue treatment with ACEIs and ARBs, if possible.

It is also worth paying attention to the issue of post-COVID syndrome (the presence and/or persistence of symptoms not associated with any other disease 8-12 weeks after the onset of COVID-19), as the review by S. Copur *et al.* [30] notes that more than 30% of patients have symptoms of this syndrome, more than 15% require re-hospitalisation, and mortality reaches more than 6%, with cardiovascular risks and symptoms playing a key role, and this is especially true for patients with impaired renal function. Thus, the results of this study are consistent with the findings of other researchers, but COVID-19 requires a range of new qualitative studies that could highlight the issues of comorbidity, pharmacotherapy, and effective management.

Conclusions

This study found that the adverse effect of kidney damage on the course of coronavirus disease depends on the glomerular filtration rate, and it is more pronounced in patients with concomitant coronary heart disease than in patients with COVID-19 without chronic coronary syndrome. This effect is detected as early as at a GFR <59 mL/min×1.73 m^2 and is manifested in a decrease in patient survival and an increase in the number of major adverse cardiovascular events (development of acute cerebrovascular accident, non-fatal myocardial infarction, pulmonary embolism, and acute left ventricular failure > Killip class I). The survival rate of patients with CHD with and without COVID-19 during the hospital period decreases at glomerular filtration rates <52 mL/min×1.73 m^2 and <34 mL/min×1.73 m^2, respectively. The concomitant chronic coronary syndrome provokes the development of chronic renal hypoperfusion, and, accordingly, in the event of an acute infectious trigger, compensatory mechanisms fail, and among patients with an unfavourable cardiometabolic background, a decision should be made as soon as possible to conduct therapeutic interventions aimed at preserving the functional capacity of the kidneys. The independent predictors of the decrease in velocity were: type 2 diabetes mellitus, age over 60, hypertension, high levels of D-dimer, C-reactive protein and ferritin, which are consistent with studies in other countries, and it is the assessment of these factors that affect the improvement (such as low-molecular-weight heparin therapy) or worsening of COVID-19, and timely vaccination of people can play an important role in the qualitative stratification of patients into risk groups and the subsequent control of the spread of the infectious process and reduction of infectious mortality and the percentage of non-infectious complications. However, COVID-19 requires further research that could highlight the problems of comorbidities, drug interactions pathogenetic treatment, and effective management in a multidisciplinary team. One of the fundamental prospects for research on this topic may be the possibility of further analysing this sample of patients.

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None.

Conflict of Interest

The author declares no conflict of interest.

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Порівняльний аналіз впливу швидкості клубочкової фільтрації на перебіг COVID-19 у хворих на ішемічну хворобу серця при супутній коронавірусній хворобі та без неї

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Анотація. Актуальність даної проблематики полягає в тому, що пандемія COVID-19 призвела до серйозних медичних наслідків, включаючи високий рівень контагіозності, розвитку хвороб, що супроводжуються ускладненням роботи нирок та серцево-судинної системи, а також підвищену смертність. Тому мета даної роботи полягала у вивченні та порівнянні впливу швидкості клубочкової фільтрації на перебіг COVID-19 у хворих на ішемічну хворобу серця та без неї. Було проведено ретроспективний аналіз 410 хворих на коронавірус, які були розподілені на 2 групи: ті, у яких була наявна хронічна ішемічна хвороба серця, і ті, у яких не було цього захворювання. Впродовж госпітального періоду (14,7 ± 5,3 діб) оцінювали комбіновану кінцеву точку – смерть від усіх причин та від серцево-судинних причин у поєднанні з основними несприятливими серцево-судинними подіями. Було визначено порогові значення швидкості клубочкової фільтрації, які асоціювалися зі зростанням частоти виникнення комбінованої кінцевої точки: для хворих на COVID-19 менше 35 мл/хв×1,73 м^2 ($p < 0,01$); для пацієнтів із ішемічною хворобою серця та COVID-19 – менше 60 мл/хв×1,73 м^2 ($p < 0,01$). Незалежними предикторами зниження фільтраційної здатності нирок у хворих 1 групи були: вік понад 65 років, наявність цукрового діабету 2 типу, високий рівень холестерину, Д-димеру, С-реактивного білка та феритину. На хворих 2 групи несприятливо впливали цукровий діабет 2 типу, артеріальна гіпертензія, високий рівень Д-димеру та С-реактивного білка ($p < 0,05$). Таку різницю було пояснено впливом проведеної терапії із точкою прикладання на антикоагулянтну та ренін-ангіотензинову систему. Це дослідження дозволить стратифікувати пацієнтів з коронавірусом в аспекті порушення ниркової функції та факторів ризику, а також визначити ефективні стратегії їх ведення в залежності від швидкості клубочкової фільтрації

Ключові слова: артеріальна гіпертензія; фактори ризику; коморбідність; летальність