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IMMUNOLOGICAL ASPECTS OF KIDNEY DAMAGE IN POST-COVID PATIENTS

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Abstract

The article highlights considerations related to the emergence of COVID-19 and its spread in the world, challenges for medical workers, rapid diagnosis of infection, provision of specialized medical care, and prevention of complications. The article also reflects an analysis and consideration of modern approaches to the clinical and pathogenetic features of the epidemiology of the disease, complications, and diagnosis.

Keywords: COVID-19, type 2 diabetes mellitus, interleukin, chronic kidney disease.

Introduction

The COVID-19 pandemic (CoronaVirus Disease 2019) is characterized by high levels of morbidity and mortality. It has already affected more than five hundred million people in the world. Research results indicate that patients with diabetes are more prone to the new coronavirus infection. According to various studies, the prevalence of diabetes mellitus (DM) in patients with COVID-19 ranges from 5 to 36%; in Russia, it is up to 25% [1; 2].

Considering that diabetes is one of the most important comorbidities in patients with COVID-19, there has recently been a need to highlight all the epidemiological and pathophysiological aspects associated with these pathological conditions to offer useful tools for the most effective control.

One of the most severe manifestations of diabetes mellitus, regardless of its type, is diabetic nephropathy, which largely determines the level and severity of complications, mortality and disability of patients [2, 4, 8]. This severe complication of diabetes mellitus leads to the formation of end-stage renal failure

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in 30% of patients [9]. Diabetic nephropathy is considered a clinical syndrome characterized by microalbuminuria or proteinuria, a progressive decrease in glomerular filtration rate, and the development or worsening of arterial hypertension [3, 5].

More and more attention is being paid to the study of the state of the immune system in patients with diabetes mellitus [1, 6, 7]. It has been established that the immune system plays an important role in the occurrence of both the disease itself and its complications [4, 6]. The increase in the number of patients with diabetes mellitus, both type 1 and type 2 and the significant prevalence of diabetic nephropathy among them are largely associated with the disorder of the immune system in patients with diabetes mellitus.

Thus, many researchers associate the appearance and worsening of complications with the activation of immune factors [1, 7]. The development of microangiopathies in diabetes mellitus is associated with a chronic immunoinflammatory process and the formation of immune complexes.

Activated monocytes-macrophages, immunoglobulins, cytokines, adhesion molecules, and end-glycosylation products are directly involved in this. The role of pro-inflammatory cytokines in stimulating the initiation and progression of vascular complications in diabetes mellitus has been shown [7]. However, issues related to the state of cellular immunity in different types of diabetes mellitus have not been sufficiently studied.

The results show that long COVID patients experienced a decrease in eGFR of approximately 2.96 ml/min (3.39% decrease from baseline) one year after COVID-19 infection. Although everyone naturally loses some kidney function over time, a decrease of 1 ml/minute over this period is more typical for healthy people. The decline in kidney function was highest in patients hospitalized for COVID-19 at 6.72%, followed by patients with diabetes at 6.15%.

The purpose of the study is to study the characteristics of changes in humoral immunity in patients with type 2 diabetes mellitus and diabetic nephropathy. Identification of epidemiological and pathophysiological relationships between diabetes mellitus and COVID-19, and assessment of prognosis to ensure better treatment results in this category of patients.

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Material and Methods

In this study, in 2021-2022, patients aged 45 to 65 years (main group I) who had COVID-19, suffering from type 2 diabetes complicated by BCS (n-65), and group II type 2 diabetes complicated by BBS without COVID -19 (n-20), group III conditionally healthy patients with covid-19 (n-15). Laboratory data include: indicators of renal function (creatinine, urea, cystatin C, glomerular filtration rate), hemostasis parameters (D-dimer, reference value up to 500 ng/l), IgG COVID-19, Ig S-RBD COVID-19. Glomerular filtration rate (GFR) was calculated using the CKD-EPI Crea-Cys (Chronic Kidney Disease Epidemiology Collaboration) formula. Exclusion criteria from the study: patients previously on renal replacement therapy, hepatorenal syndrome, urolithiasis, kidney damage due to hypertension.

Table 1 - Characteristics of patients by group (n=100)

Groups		Gender		Age	Duration of Type 2 Diabetes (years)
		Male	Female		
Group I (n =65)		30	35	62.9±11.1	9.9±1.8
	%	41.7	58.3		
Group II (n=20)		13	7	56.4±6.8	9.1±1.9
	%	56.7	46.3		
Group III (n =15)		7	8	44.1±7.3	
	%	45	55		

Inclusion criteria for the study: age 45 years and older. Gender: men and women. Patients with a history of pneumonia of viral etiology. All patients from whom IgG COVID-19 and Ig S-RBD COVID-19 were taken from venous blood.

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Table 2 - The characteristics and clinical data of the post-COVID-19 patients

	Variables	1st group (DM2T,CKD, COVID-19) n=65	2nd group (DM2T, CKD without COVID-19) n=20	3rd group (DM2T and COVID-19) n=20	p Value
1	Gender Male(%) Female (%)	 29(44.61) 36(55.38)	 11(55.00) 9(45.00)	 8 (40.00) 7 (60.00)	<0.001
2	Age (years), median (IQR)	59 (61, 77)	60 (65.25)	60(65.25)	<0.001
3	BMI/(kg/m2) median (IQR)	24.79 (22.91, 26.83)	23.76 (21.96, 26.98)	22.67(20.89, 25.87)	0.043
4	Hypertension (%)	47 (72.30)	17 (85.00)	12 (60.00)	0.187
5	cardiovascular disease (%)	35 (53.84)	8 (40.00)	6 (30.00)	0.881
6	Diabetic retinopathy (%)	49 (75.38)	6 (30.00)	5 (25.00)	0.043
7	HbA1C(%), median (IQR)	7.75 (6.60, 9.10)	7.10 (6.40, 8.05)	7.15 (6.55, 8.10)	0.042
9	TG/(mmol/L), median (IQR)	2.41 (1.73, 3.74)	1.32 (1.02, 1.91)	1.12 (0.92, 1.73)	<0.001
10	ApoB/(mmol/L), median (IQR)	1.42 (1.23, 1.67)	0.81 (0.70, 0.94)	1,03 (0.97, 1.12)	<0.001
11	Fasting blood glucose /(mmol/L), median (IQR)	5.94 (4.78, 7.92)	5.96 (4.64, 7.43)	6.13(5.73, 7.24)	0.492
12	Creatinine/ (μmol/L), median (IQR)	152.15 (128.25, 152.15)	159.50 (122.00, 252.58)	85.15 (59.5, 77.5)	0.898
13	CysC/(mg/L), median (IQR)	1.98 (1.57, 2.60)	1.95 (1.55, 2.86)	0.79 (0.67, 0,1)	<0.001
14	eGFR/[mL/(min·1.73m2)], median (IQR)	40.24 (25.62, 51.38)	42.04 (28.22, 50.46)	87.69(90.5, 63.5)	<0.001
15	D-dimer(μg/ml) median (IQR)	1.77(0.3, 34.00)	0.77(1.5, 46.5)	1.15(2.00, 56.00)	0.492
16	IL 17A (pg/ml) median (IQR)	45.79(41.5, 81.00)	38.41(41.00, 81.00)	1.80(2.00 78.00)	<0.001
17	IL11 (pg/ml) median (IQR)	809.83(19.00, 94.00)	793.83(416.94, 178.00)	7.25(8.00, 72.00)	<0.001
18	TGFβ1 (ng/ml) median (IQR)	5.27(12.5, 178.00)	5.24(12.5, 175.00)	2.94(10.00, 75.00)	<0.001
19	COVID-19 IgG S-RBD (BAU/ml) median (IQR)	927.50 (234.00, 449.00)	6.08(6.00, 5.00)	621.47(129.00, 1165.00)	<0.001
20	SARS-COV-2 (COVID-19) IgG antibodies (U/mL) median (IQR)	971.74 (129.00, 1165.00)	4.73(5.00, 5.00)	1971.74(92.00, 371.00)	<0.001

Table 2 shows creatinine, urea, cystatin C, glomerular filtration rate), D-dimer, IgG COVID-19, IgG S-RBD COVID-19, IL11, IL17A, and TGF-β1 concentrations were measured in blood/serum samples of control subjects and

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three groups of COVID-19 patients. A statistically significant difference in Creatinine between the 1st group, and the group 3rd of COVID-19 patients (152.15±45.6 vs 85.15±34.2, $p<0.001$) are shown, while the difference between 1st group and 2nd group of COVID-19 patients was not significant (152.15±45.6 vs 159.50±86.0,$p=0.091$).

Also, statistically significant differences in serum levels of CysC between the 1st group and group 3 as well as between the 2nd and 3rd group of COVID-19 patients were shown. In contrast, the difference between 1st group and group 2 patients was not significant, it shows that CKD impacts to level of Cystatin C.

Analyzing the difference between serum levels of D-dimer among the groups shows a significant difference between the levels of the 1st and 2nd group, as there's a dramatic decrease in the levels of the 2nd group. While 1st (the patients with COVID-19) and 3rd group (patients without COVID-19) had minimal difference it also shows that CKD affects the level of D-dimer.

On measuring the concentration of eGFR, minimal elevation of eGFR can be seen in the 2nd group, as compared to the 1st. But again, there's a significant difference between the levels of 1st and 3rd group. On the other hand, while analyzing the levels of IgG S-RBD, significant differences in levels can be seen in all three groups. Levels of 3rd group which includes patients without COVID-19 have the lowest level among the three.

Serum levels of IgG antibodies are significantly higher in group 3rd and group 1st. While group 3rd hit the peak, group 2nd had the lowest IgG serum levels, showing the severe impact of COVID-19 on other groups.

Results

The average age was 62 years, 58% of them were women. The level of D-dimer was determined in all patients from the entire study population: 34 (70%) of them showed an elevated level associated with a high risk of thromboembolic complications. In 12 cases (35%) the d-dimer level exceeded 1.5 times, in 11 cases (32.5%) 2 times, and in the remaining 11 cases (32.5%) 3 times or more. The majority of the studied patients had chronic concomitant diseases: arterial hypertension, coronary heart disease, diabetes mellitus, and chronic lung

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diseases, 65%, 49%, 37%, and 6%, respectively. Among them, patients with one concomitant disease are 19 (19%), comorbid - 44 (43%), and multimorbid - 31 (30%). There were no concomitant diseases in 8 (8%) patients. To identify correlations between the stage of CKD and the degree of lung damage, we divided patients with kidney damage into three groups according to GFR.

Conclusion

In conclusion, it should be noted that the new coronavirus infection can be considered a systemic disease since it is not limited to damage to one organ. The presence of diabetes mellitus in patients with COVID-19 aggravates the severity of the disease and is accompanied by an increased risk of adverse outcomes, including increased mortality. Some specific pathogenetic mechanisms have been identified that likely predispose to this. Factors that cause such adverse events are of practical interest as potential targets for effective therapy. Inflammation has a significant influence on the pathogenesis of DN, and anti-inflammatory processes may have a significant impact on the protective effect on the kidneys. Tissue damage activates innate immunity through the recognition of DAMPs by various PRRs during hyperglycemia, causing several inflammatory reactions. Persistent inflammation caused by long-term kidney damage can lead to the development of DN. Various proinflammatory pathways are involved in the progression of DN. Several studies focusing on innate immune pathways in DN have shown promising results. Given the critical role of inflammatory pathways, a combination treatment approach including both anti-inflammatory and antidiabetic drugs may provide better protection against DN. However, clinical trials of anti-inflammatory drugs are just beginning. Patients may become more susceptible to infection in the absence of specific anti-inflammatory treatment. In addition, TLR suppression in patients with DN may increase the risk of tumorigenesis and malignancy recurrence. Therefore, the regulation of innate immune pathways and the development of more specific and less toxic treatments for DN require further study in the future.

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