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Influence of Covid-19 on the Course and Tactics of Therapy of Chronic Non-Communicable Diseases (On the Example of Arterial Hypertension and Type 2 Diabetes Mellitus).

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Annotation

The aim of the study was to evaluate the influence of COVID-19 on the course of arterial hypertension and type 2 diabetes mellitus and to determine the necessity of therapy correction. The study included 100 patients evenly divided into two groups.

After COVID-19, the majority of patients showed worsening of blood pressure and glycemic control. Hypertensives had increased mean BP values, and diabetics had increased HbA1c and fasting glucose levels. More than 20% of patients required intensification of therapy.

The results confirm the necessity of active surveillance and early adjustment of treatment after coronavirus infection.

Keywords: COVID-19, fasting syndrome, arterial hypertension, type 2 diabetes mellitus, chronic non-communicable diseases, blood pressure, glycemic control, therapy tactics, rehabilitation.

INTRODUCTION.

The COVID-19 pandemic caused by the SARS-CoV-2 virus has had an enormous impact on global health. The relationship of the new coronavirus infection with chronic non-communicable diseases such as arterial hypertension (AH) and type 2 diabetes mellitus (T2DM) has attracted particular attention of researchers. Already at the beginning of the pandemic it became clear that the presence of these diseases worsens the prognosis in acute COVID-19 infection: patients with AH and DM2 were more likely to carry COVID-19 in

severe form, they have a higher risk of complications (acute respiratory distress syndrome, thrombosis) and death.

Thus, according to a number of studies, hypertension and diabetes were among the most common comorbid conditions in hospitalized and deceased patients with COVID-19. In particular, Italian researchers noted that among those who died from COVID-19, 73-74% suffered from AH and about 34% had DM2. Such observations have led to an increased interest in these diseases in the context of COVID-19, as risk factors for an unfavorable course of infection [4].

The pathogenetic mechanisms underlying the mutual influence of COVID-19 and cardiovascular/metabolic disease are not fully understood, but it is known that the SARS-CoV-2 virus interacts with the angiotensin-converting enzyme 2 (ACE2) receptor on the cell surface. ACE2 is expressed in various tissues including lung, heart, vascular endothelium, and pancreas [8].

On the one hand, the presence of AH and DM2 may contribute to a more severe course of COVID-19 through chronic inflammation, endothelial dysfunction and impaired immune response.

On the other hand, coronavirus infection itself can cause damage to target organs and disturb homeostasis, aggravating the course of existing chronic diseases. During the acute period of infection, hypertension and diabetes require special attention: maintenance of haemodynamics, correction of hyperglycemia, and prevention of thromboembolism. There was also a discussion about the safety and tactics of some drugs in such patients.

For example, the question of continuing therapy with ACE inhibitors and angiotensin II receptor blockers (ARBs) in hypertensive patients on COVID-19 was discussed, as these drugs increase ACE2 receptor expression. Subsequently, however, data from a number of clinical trials showed that ACE inhibitors/BRAs should not be withdrawn: on the contrary, continuation of therapy with RAAS blockers was associated with a better prognosis in COVID-19 and reduced mortality. Thus, at the stage of acute COVID-19, the tactics of management of patients with AH and DM2 have been partially defined and reflected in the recommendations - for example, the need for careful BP and glycemic control, the use of low-molecular-weight heparins at high risk of thrombosis, continuation of baseline therapy of chronic diseases, etc.[6]

Less studied has been what happens to the course of arterial hypertension and diabetes mellitus after a patient has had COVID-19 and recovered. Long-COVID has proved to be a multiorgan condition: according to the current definition, it includes symptoms and abnormalities that persist for more than 12 weeks after the acute phase of infection and are not explained by other causes.

The most common manifestations of post-COVID syndrome are persistent asthenia (chronic fatigue), dyspnoea on exertion, tachycardia, cognitive impairment, sleep disturbances, depressive and anxiety disorders, and muscular-articular pain. However, in addition to subjective symptoms, increasing evidence points to objective metabolic and vascular abnormalities persisting in COVID-19 survivors. For example, there is evidence of an increased risk of new cases of diabetes mellitus after coronavirus infection and the possible development or worsening of arterial hypertension in survivors.

Mechanisms of the impact of COVID-19 in chronic non-communicable diseases may include direct organ damage (e.g., pancreatic β-cells, cardiomyocytes, and kidneys), immunological and autoimmune processes, and a prolonged systemic inflammatory response [7].

In the acute phase of severe COVID-19, there is often a pronounced cytokine storm, hypercoagulability, and acute vascular endothelial damage. In part, these pathological changes may persist after recovery, setting the stage for decompensation of previously stable chronic conditions. For example, it is known that COVID-19 survivors show signs of endothelial dysfunction and increased arterial stiffness for several months, which negatively affects blood pressure.

In addition, tachycardia and autonomic dysregulation (autonomic nervous system dysfunction) persist in a significant proportion of patients after COVID-19, which may also contribute to BP fluctuations and elevation. With regard to carbohydrate metabolism - even after a relatively mild course of coronavirus infection - some patients demonstrate long-lasting hyperglycemia, decreased insulin sensitivity. The stress associated with the severe course of the disease, the use of glucocorticoids during COVID-19 treatment (in severe patients), decreased physical activity and increased body weight during the recovery period all potentially exacerbate glycemic control in individuals with pre-existing diabetes [9].

The relevance of this study stems from the need to understand the extent to which COVID-19 affects the long-term course of common diseases such as AH and DM2, and to determine the optimal management of these patients in the post-COVID period.

The data obtained will allow primary care physicians and subspecialists (cardiologists, endocrinologists) to timely adjust the plan of monitoring and treatment of patients who have undergone coronavirus infection in order to prevent deterioration of the condition and development of complications.

MATERIALS AND METHODS OF THE STUDY.

A single-centre retrospective observational study (cohort analysis of pre- and post-exposure data) was conducted. COVID-19 episode was considered as ‘exposure’, and the outcomes assessed were changes in chronic disease parameters (AH or DM2) and the need for therapy adjustment.

Patients aged 40-75 years, diagnosed with either essential arterial hypertension stage II-III or type 2 diabetes mellitus (moderate severity), who had been under observation at the polyclinic of our centre for at least 1 year before participation in the study. A mandatory condition for inclusion was documented COVID-19 disease between 2020 and 2023 (positive PCR test for SARS-CoV-2 or a characteristic clinical and radiological picture of viral pneumonia) and knowledge of the patient's pre-infection status. Participants must have fully recovered from acute COVID-19 (absence of fever and respiratory failure) and be in the recovalescence phase for at least 3 months after the acute phase by the time of the examination.

Patients with cancer, severe chronic renal failure (CRP <30 ml/min), decompensated hepatic failure, and those who had suffered a myocardial infarction, stroke, or other major cardiovascular events within the last year were excluded from the study (to exclude the influence of these events on the analyzed parameters). Patients with type 1 diabetes mellitus or

secondary (symptomatic) hypertension were not included. Pregnant women were also excluded.

A total of 100 patients were included in the study, of whom 50 had arterial hypertension and 50 had type 2 diabetes mellitus. Groups were formed based on the underlying chronic disease: patients with AH did not have DM2, and vice versa, to separately assess the effect of COVID-19 on each disease.

The main demographic and clinical characteristics of the participants are summarized in Table 1. The mean age was 58±9 years, and 45% of the subjects were male and 55% female. The mean duration of AH in the respective patient group was about 8±5 years, and the mean duration of DM2 was 10±6 years.

All patients had previously received baseline therapy of their chronic disease according to clinical guidelines: Hypertensives - various combinations of hypotensive agents (ACE inhibitors, BRAs, beta-blockers, calcium antagonists, diuretics), diabetics - diet and sugar-lowering drugs (metformin, sulfonylurea, etc., and 20% of them received basal-bolus insulin therapy before COVID-19).

The severity of COVID-19 in the sample ranged from mild to severe: 25 patients (25% of the cohort; 15 with AH and 10 with DM2) required hospitalization for coronavirus infection, of whom 10 had a severe course with viral pneumonia (requiring oxygen support, but no ventilator support and no deaths) and the rest had a moderate course. In 75% of COVID-19 survivors, the course was ambulatory, mild or moderate without respiratory failure. Specific antiviral therapy (remdesivir, etc.) was used in severe cases; dexamethasone was given to 12 patients (all from among those hospitalized with pneumonia).

For each patient, data were collected for two time intervals, before COVID-19 and after COVID-19. The ‘before’ period was the last month prior to COVID-19 (from outpatient charts: blood pressure values, last blood glucose and glycated hemoglobin results, list of medications used and their doses, presence of complications of AH or DM2). The ‘after’ period covered the patient's examination performed at the time of inclusion in the study (3-6 months after COVID-19).

At the next stage, all patients underwent standard clinical and laboratory examination:

- repeated BP measurement (three times with an interval of 5 minutes with calculation of the mean value;

- 30% of patients also underwent daily BP monitoring to clarify the profile), venous blood collection to assess carbohydrate metabolism parameters (fasting glucose, glycated hemoglobin HbA1c), general blood count, biochemical analysis (with emphasis on inflammatory markers, liver and kidney function).

Information on changes in treatment was also collected: whether in the post-cohort period new drugs were prescribed or doses of previous drugs were increased, transfer to insulin, etc. New clinical events or complications that occurred after COVID-19 were recorded separately (hypertensive crises, cardiac rhythm disturbances, exacerbation of coronary disease, for diabetics - episodes of ketoacidosis, hypoglycemia, and occurrence of diabetic complications). Patients filled out a questionnaire about the presence of symptoms of postcovid syndrome (asthenia, dyspnoea, cognitive, emotional disorders, etc.).

Quantitative parameters are presented as mean and standard deviation (M±SD). Paired dependent samples (pre- and post-COVID-19 parameters in the same patients) were compared using paired Student's t-test for parameters with normal distribution or Wilcoxon test for related samples with non-normal distribution.

Categorical data (percentages of patients with certain features) were compared using McNemara's χ^2 criterion for dependent shares. The significance level was taken as 0.05.

To assess factors associated with the severity of changes, subgroup analysis was performed: patients with severe COVID-19 (with hospitalization) and those with outpatient COVID-19 were compared according to the difference in BP and HbA1c. Statistical processing was performed in SPSS 26.0.

THE RESULTS OF THE STUDY AND THEIR JUSTIFICATION.

Baseline characteristics. The study cohort included 100 patients (50 with AH, 50 with DM2) and the baseline characteristics are presented in Table 1. The mean age of patients with hypertension was 60 years, with diabetes - 55 years. In both groups, persons over 50 years of age predominated, sex distribution was comparable (men about 40-50%).

The duration of the underlying disease was significant: Hypertensives had an average of 8 years of AH, diabetics - about 10 years of DM2, indicating a formed comorbid background. Many patients had comorbidities: coronary heart disease in 30% of Hypertensives and 20% of diabetics, obesity (BMI>30) in 25% and 40%, respectively, and dyslipidaemia in the majority of patients in both groups. These concomitant risk factors could also influence the course of both COVID-19 and chronic diseases. The severity of COVID-19 experience differed slightly between the groups: among patients with AH, the proportion hospitalized (severe course) was 30%, whereas in diabetics it was 20%.

This is consistent with the general view that hypertension per se is associated with a higher risk of severe COVID-19. No patient in our sample required ICU treatment or invasive ventilation; all recovered. The mean duration of the acute phase of COVID-19 was ~2-3 weeks, and the follow-up period after recovery at the time of evaluation was 4.5 months (median).

Table 1. Clinical characteristics of patients who underwent COVID-19 (n=100)

Indicator	AH group (n=50)	DM2 group (n=50)
Age, years (M±SD)	60 ± 10	55 ± 8
Men, n (%)	25 (50%)	20 (40%)
Duration of underlying disease, years	8 ± 5	10 ± 6
Obesity (BMI > 30), n (%)	12 (24%)	20 (40%)
CHD, chronic heart failure	15 (30%)	10 (20%)
Chronic kidney disease (moderate)	5 (10%)	8 (16%)
Hospitalisation for COVID-19, n (%)	15 (30%)	10 (20%)
Severe COVID-19 (pneumonia), n (%)	8 (16%)	4 (8%)
Therapy of underlying disease before COVID-19	IAPPs/BPAs, BBs, ACs, diuretics	metformin, SM, DPP4 inhibitors;

	(average 1.8 drugs per patient)	insulin in 10 (20%) patients
Note: BMI, body mass index; CHD, coronary heart disease; iAPP, ACE inhibitor; BRA, angiotensin II receptor blocker; BB, beta-blocker; AC, calcium antagonist; SM, sulfonylurea derivatives.		

Dynamics of blood pressure after COVID-19. Analysis of data from patients with arterial hypertension showed a statistically significant increase in blood pressure levels in the post COVID period compared to the baseline pre-infection level. The mean office systolic BP (CAD) in the AH group before COVID-19 was 135±10 mmHg, whereas ~4 months after COVID-19 it was 142±12 mmHg (Figure 1).

Similarly, diastolic BP (DBP) increased from 85±7 to 90±8 mmHg. The increase in both SBP and DA was modest in absolute terms (about +7 mm Hg), but sufficient to affect the quality of hypertension control: the proportion of patients achieving target BP values (<140/90 mm Hg according to most guidelines) decreased significantly.

If before COVID-19 40 out of 50 patients (80%) had BP in the target range on therapy, after COVID-19 only 32 patients (64%) managed to maintain the target BP. Accordingly, the proportion of patients with uncontrolled hypertension increased from 20% to 36% ($p<0.05$). Of these, some patients had episodes of pressure rise (hypertensive reactions) in the first months after the disease, although no severe hypertensive crises requiring emergency treatment were recorded.

The deterioration of the BP profile was also confirmed by the data of diurnal monitoring in those 15 patients who underwent it: the average daily SBP increased by 5-6 mmHg during the post-coital period, the diurnal rhythm was disturbed in 4 patients (appearance of a shallow ‘night slump’ or ‘non-dipper’ phenomenon).

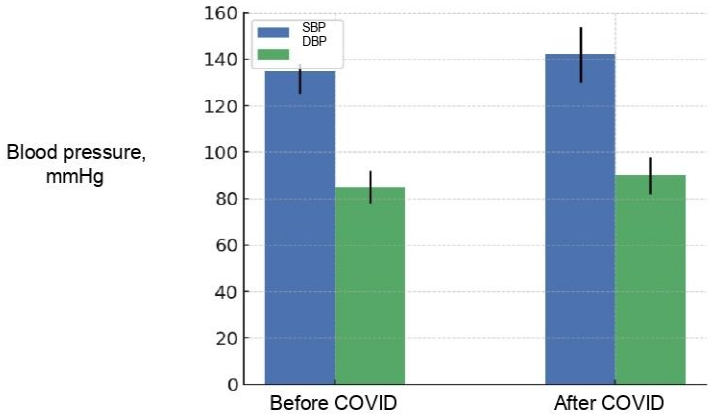

Figure 1. Mean systolic (CAD) and diastolic (DBP) blood pressure values before and ~4 months after COVID-19 in patients with arterial hypertension.

Changes in BP were accompanied by the need to adjust therapy. In 10 patients with AH (20% of the group), the treating physician intensified hypotensive therapy in the post-cohort period: either the doses of existing drugs were increased or a new antihypertensive drug was added to the regimen. In particular, the addition of a diuretic or calcium antagonist to control blood pressure was more often required, and less often an ACE inhibitor/BPA inhibitor dose increase was required.

The remaining 40 patients were able to compensate for small increases in BP with non-medication measures (increased salt control, regimen, and physical activity) or temporary correction, after which BP stabilized. Nevertheless, even those who did not change therapy tended to have higher working BP values.

Table 2 summarizes the main parameters characterizing the course of hypertension before and after COVID-19.

Table 2. Blood pressure and therapy parameters in patients with AH (n=50) before and after COVID-19

Parameter	Before COVID-19.	After COVID-19.
Systolic BP, mm Hg (M±SD)	135 ± 10	142 ± 12 *
Diastolic BP, mm Hg (M±SD)	85 ± 7	90 ± 8 *
Uncontrolled hypertension, n (%)	10 (20%)	18 (36%)
Number of hypotensive drugs, mean	1,5	1,8
Increased therapy (new drug or dose ↑), n (%)	–	10 (20%)
Note: * statistically significant differences ($p < 0.05$) compared with pre-COVID-19.		

The results suggest that COVID-19 was associated with some degree of worsening of the course of arterial hypertension. The increase in BP may reflect both direct effects of the infection and indirect factors.

Possible mechanisms include persistent endothelial dysfunction and increased arterial stiffness after COVID-19. Our findings are in agreement with other studies, where it has been observed that increased vascular wall stiffness and endothelial dysfunction persist in patients several months after coronavirus infection. This leads to increased peripheral vascular resistance and, consequently, BP levels.

In addition, cardiovascular symptoms were widely reported in our patients in the post-coital period: 50% complained of intermittent tachycardia, heart palpitations or palpitations, and 20% had chest pain of an intermittent nature. According to the literature, up to 86% of COVID-19 survivors have some form of cardiovascular symptoms associated with autonomic dysfunction and residual myocardial inflammation. Tachycardia and an imbalance of the autonomic nervous system (with a predominance of sympathetic tone) may cause a tendency to increase blood pressure, especially in predisposed individuals with hypertension.

Interestingly, we observed the most pronounced deterioration in BP control in patients who had undergone COVID-19 in a more severe form. Among the 15 hospitalized hypertensive patients, the post-illness CAD elevation averaged 10 mm Hg, and 7 of them (≈47%) required

intensified therapy. In contrast, patients who underwent COVID-19 on an outpatient basis had an average increase in CAD of 4 to 5 mm Hg, and only 3 (≈8%) required the addition of medication. Although statistical significance of differences between subgroups was not formally achieved (possibly because of the relatively small group size), a clear trend emerged: a more severe course of COVID-19 was associated with more significant subsequent AH decompensation. This may be explained by a greater degree of systemic inflammation and vascular damage in severe COVID-19. In addition, these patients were more likely to use corticosteroids and other aggressive therapies potentially affecting BP.

Our results confirm the data of other studies indicating an increased risk of cardiovascular events in patients with coronavirus infection. For example, one large cohort study (Sweden) showed a several-fold increased risk of myocardial infarction and ischemic stroke in the first months after COVID-19 (reported by the authors to be about 7-fold higher than in comparable individuals without COVID-19). This emphasizes that the post-COVID period is a critical time for patients with cardiovascular risk factors and requires increased surveillance.

In the context of arterial hypertension, this implies the need for tighter control of blood pressure in re-hypertensive patients. Our observation of an increase in the number of hypertensive patients requiring intensified treatment is in line with the results of Italian colleagues who reported that about 20% of patients after COVID-19 experienced deterioration in BP control requiring initiation or intensification of hypotensive therapy.

It is important to note that persisting asthenisation and psychoemotional disturbances after COVID-19 may indirectly complicate the treatment of hypertension. In our study, about half of the patients showed symptoms of anxiety, depression, cognitive complaints (difficulty in concentration, attention, memory) - that is, elements of post-CoVID-19 neurological syndrome. The presence of even one pronounced psychological or neurological symptom, according to experts, can hinder patient adherence to treatment, complicate the control of diet and medication [10].

Professor O.D. Ostroumova in a review on post-COVID syndrome in hypertensive patients drew attention to the fact that the presence of asthenia and other long-term persistent symptoms significantly complicates the treatment of cardiovascular diseases, provoking their exacerbation or debut after COVID-19. Our data confirm it: in many patients decompensation of AH occurred against the background of pronounced asthenia and insomnia, which required a complex approach - not only increasing pills, but also symptomatic therapy (sedatives, metabolic drugs), rehabilitation measures.

From the practical point of view, the obtained results dictate the need to adjust the tactics of management of patients with AH after COVID-19[1].

First of all, more frequent BP monitoring in the first months after the disease is recommended for such patients. It is desirable to ensure home BP control (self-monitoring) and timely visits to the doctor to assess the need for changes in therapy.

Secondly, attention should be paid to factors that exacerbate hypertension: restoration of physical activity (taking into account the patient's tolerance), reduction of body weight in patients with weight gain during illness/immobilization, dietary correction (salt restriction, stimulation of intake of foods rich in potassium and magnesium).

Thirdly, at the first signs of worsening BP control, it is necessary to promptly intensify drug therapy without waiting for the development of complications.

Our experience shows that the addition of a thiazide diuretic or calcium antagonist has been effective in most cases to stabilize blood pressure in patients after COVID-19. At the same time, baseline therapy (e.g., ACE inhibitors) should not be cancelled - on the contrary, their continuation, as mentioned, may have a protective effect.

Finally, the increased pro-coagulation status after COVID-19 should be taken into account: if indicated (e.g., in patients with concomitant CHD, atrial fibrillation), it is worth considering the prescription or continuation of antithrombotic therapy (aspirin, anticoagulants) for the prevention of cardiovascular events, together with optimization of BP control[4].

Dynamics of carbohydrate metabolism and diabetes mellitus parameters. The patients with DM2 included in the study also showed a significant deterioration of parameters characterizing the course of diabetes in the post-cohort period. The key findings are summarized in Table 3.

The mean glycated hemoglobin (HbA1c) level, reflecting long-term glycemic control, before COVID-19 was 7.5±0.8%. This indicates that diabetes was relatively satisfactorily compensated in the whole group (the target HbA1c level for most patients with DM2 is usually <7.0-7.5%, although higher values are acceptable for the elderly). Several months after COVID-19, mean HbA1c increased to 8.2±1.0%, which was statistically significant (Figure 2, left panel). This change of +0.7 percentage points indicates the group's transition into the zone of diabetes decompensation (HbA1c >7.5% can be conventionally considered a sign of inadequate control).

HbA1c increased in 80% of patients, and in 30% of patients - by more than 1%. Similar changes were observed in fasting plasma glucose levels: the mean concentration increased from 7.0±1.0 mmol/L to 8.0±1.2 mmol/L (Figure 2, right panel). In comparison, fasting normoglycemia is <6.1 mmol/L, the target level for many diabetics is <7 mmol/L. Before COVID-19, 60% of patients managed to maintain fasting glucose <7.2 mmol/L, whereas after the disease only 40% of patients did so. The proportion of patients with severe fasting hyperglycemia (>8-9 mmol/l) increased from 10% to 28%. Thus, coronavirus infection was associated with a worsening of the glycemic profile in a significant proportion of patients with DM2.

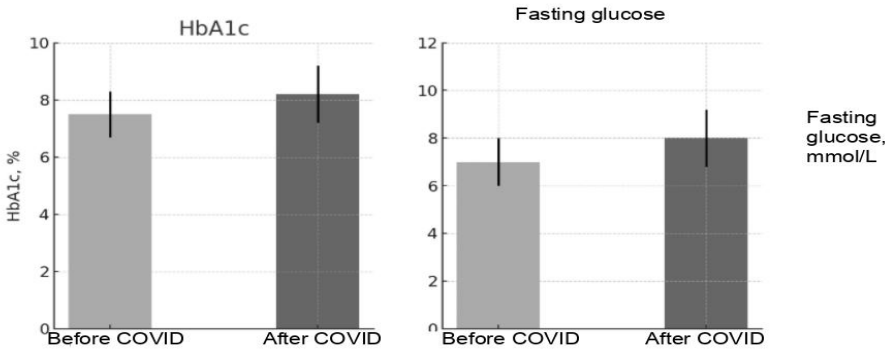

Figure 2. Carbohydrate metabolism parameters before and after COVID-19 in patients with type 2 diabetes: (left) mean glycated hemoglobin (HbA1c); (right) mean fasting blood glucose concentration. Error bars indicate standard deviation.

An important consequence of the deterioration of glycemic control was the need to correct antidiabetic therapy. Prior to COVID-19, 40 patients (80%) received oral hypoglycemic drugs (metformin in combination with other drugs), 10 patients (20%) were on insulin therapy (in combination or monotherapy).

After COVID-19 and repeated assessment of the condition, the number of patients requiring insulin increased to 15 (30%). In other words, an endocrinologist was forced to prescribe insulin after an episode of COVID-19 to 5 of those patients who had previously been controlled with tablets due to deterioration in glycemic parameters.

As a rule, these were patients who had suffered from a moderate or severe coronavirus infection, against which they were given high doses of glucocorticoids in the hospital, which caused a temporary diabetic effect, as well as receiving parenteral nutrition. Some of them already had severe hyperglycemia during COVID-19, requiring insulin, and after discharge, the need for it persisted. Other patients (with relatively mild COVID-19) retained their previous therapy to a greater extent, however, 3 of them had increased metformin dosages or added sulfonylureas to improve glucose control [3].

Table 3. Indicators of glycemic control and therapy in patients with DM2 (n=50) before and after COVID-19

Indicator	Before COVID-19	After COVID-19
HbA1c, % (M±SD)	7,5 ± 0,8	8,2 ± 1,0 *
Fasting plasma glucose, mmol/L (M±SD)	7,0 ± 1,0	8,0 ± 1,2 *
HbA1c > 7%, n (%) (unsatisfactory control)	20 (40%)	30 (60%)
Receive insulin therapy, n (%)	10 (20%)	15 (30%)
Episodes of severe hyperglycemia (>15 mmol/l) for the last 3 months, n	0	3 (6%)
Note: * $p < 0.05$ compared to the period before COVID-19.		

In addition to the objective figures of glycemia, there was a clinical increase in cases of decompensation in diabetics in the post-covid period. Thus, 3 patients (6%) suffered episodes of severe hyperglycemia (glucose level > 15 mmol/L) with mild ketoacidosis within 3 months after COVID-19, requiring short-term hospitalization and infusion therapy.

For comparison, a year before COVID-19, no cases of diabetic ketoacidosis were noted in this group – in all patients, T2DM was compensated without such serious acute complications. Also, 5 patients (10%) complained of increased symptoms of diabetes after the disease: polyuria, thirst, weight loss. These symptoms correlated with an increase in blood glucose levels. It was noted that almost all cases of severe worsening of diabetes occurred in patients who had been severely ill with COVID-19 and were receiving corticosteroids. This suggests the determining role of acute period factors (stress effects on metabolism) in subsequent decompensation.

However, even in patients with mild COVID-19, there was a slight increase in HbA1c (average +0.3% among outpatients versus +0.9% in hospitalized patients). Consequently, the tendency to deterioration of metabolic control is common to most patients with T2DM, although the degree of this decompensation varies depending on the severity of the infection and, probably, the individual characteristics of the patient.

The rationale for the data obtained should be sought in the totality of the path physiological effects caused by the new coronavirus infection [6].

First, there is a direct viral attack on beta cells of the pancreas. Some studies have found that SARS-CoV-2 can infect cells of the islets of Langerhans (where the ACE 2 receptor is also expressed) and cause their dysfunction or death. This leads to a decrease in insulin production and, as a result, to hyperglycemia.

Secondly, systemic inflammation and the cytokine cascade in COVID-19 induce insulin resistance in peripheral tissues. It is known that pro-inflammatory cytokines (for example, TNF-α, IL-6) can disrupt signal transmission from insulin, leading to a decrease in glucose uptake by cells. The level of these cytokines is elevated in severe COVID-19 and may remain moderately elevated for some time after recovery, maintaining insulin resistance.

Thirdly, steroid therapy and other medications used in the treatment of COVID-19 negatively affect carbohydrate metabolism.

As already mentioned, in some patients, the appointment of dexamethasone in the hospital was necessary for the treatment of viral pneumonia; however, glucocorticoids cause an increase in blood glucose and can provoke steroid diabetes. Although the situation usually improves after steroid withdrawal, some patients seem to have experienced a “breakdown” in diabetes compensation, which is difficult to overcome without increased therapy.

In our study, all the patients were already diabetics before COVID-19, but the fact that their condition worsened indirectly confirms the ability of coronavirus infection to exacerbate carbohydrate metabolism disorders. It can be assumed that in predisposed individuals, COVID-19 accelerates the transition from prediabetes to manifest diabetes, and in existing diabetics, it brings the development of complications and the need for more intensive treatment closer.

In the context of practical management of patients with DM2 after COVID-19, our results highlight several important points. First, it is necessary to monitor glucose and HbA1c levels in all diabetics who have had COVID-19 immediately after recovery and over time.

It is advisable to check glycated hemoglobin 3 months after the infection, without waiting for an annual routine checkup, since a significant proportion of patients need to adjust treatment earlier.

Secondly, doctors should be prepared for an accelerated transition to insulin therapy in those patients who are difficult to control with pills. COVID-19 could reduce the reserve of beta cells, so what previously could be compensated by diet and metformin may now require the addition of basal insulin. In our small study, 10% of patients received newly prescribed insulin, which is quite a lot in a short period of time, and at the population level may mean a surge in insulin demand after a pandemic.

Thirdly, attention should be paid to rehabilitation and lifestyle of diabetics after COVID-19. We noticed that many patients had significantly decreased physical activity: due to weakness, shortness of breath, people moved less; some gained 3-5 kg of weight during

recovery. This, in turn, worsens insulin sensitivity and glycemia. Therefore, rehabilitation programs, including metered-dose physical therapy, cardio respiratory exercises, are very useful for improving overall health and metabolic control. It is also necessary to evaluate the function of the thyroid gland and adrenal glands in patients with postcovid syndrome, since hormonal imbalance (for example, subclinical hypothyroidism, adrenal hypofunction) can indirectly affect carbohydrate metabolism.

Finally, it is important to actively work to increase adherence to therapy in patients who have suffered from COVID-19. As our survey showed, some patients attributed the deterioration in diabetes control not only to objective reasons, but also to the fact that during and after their illness they "gave up" in terms of dieting and taking pills on time. Psychological support, the inclusion of these patients in diabetes schools, and self–control training remain relevant, perhaps even more so than before, given the general negative impact of the pandemic on the psyche.

Other effects of COVID-19 on chronic diseases. Although the focus of our study is hypertension and DM2, it is worth noting that coronavirus infection can also affect other chronic non-communicable diseases. For example, the literature describes cases of exacerbation or onset of autoimmune endocrinopathies (thyroiditis, type 1 diabetes) after COVID-19, worsening asthma control, and accelerated progression of chronic kidney disease.

Our data indirectly confirm the overall negative impact of COVID-19 on the comorbid background: we observed an increase in angina episodes in patients with coronary heart disease, an exacerbation of polyneuropathy in diabetic patients, and a slight (on average 5-7 units) increase in cholesterol and triglyceride levels after infection.

The latter coincides with publications indicating new cases of dyslipidaemia in those who have been ill (including those who previously had a normal lipid profile). Thus, we can talk about the syndrome of polysystemic metabolic exacerbation after COVID-19 – affecting blood pressure, sugar, lipids and general inflammation. This requires a comprehensive approach: such patients should be monitored jointly by a therapist, a cardiologist, an endocrinologist, and, if necessary, a neurologist and a psychotherapist (to correct cognitive and emotional disorders).

CONCLUSIONS.

The transmitted new coronavirus infection COVID-19 has a distinctly negative effect on the course of chronic non-communicable diseases, in particular on arterial hypertension and type 2 diabetes mellitus.

In patients with hypertension, there is an increase in blood pressure and a deterioration in its daily profile in the post-covid period, which leads to a decrease in the proportion of patients with controlled hypertension. In patients with DM2 after COVID-19, glycemic control worsens: HbA1c and blood glucose levels increase, and episodes of hyperglycemia become more frequent. These changes dictate the need to adjust therapy tactics – approximately one in five hypertensive patients and one in ten diabetics required increased medication in the first months after suffering from COVID-19.

Thus, doctors should plan more careful monitoring and proactive therapy for patients who have suffered from COVID-19 and have chronic diseases.

It is recommended to monitor key indicators (blood pressure, glucose and glycated hemoglobin levels) more often, to enhance therapy in time (add drugs or switch to insulin for diabetes), as well as to carry out rehabilitation measures aimed at restoring the function of the cardiovascular system and metabolism.

A comprehensive interdisciplinary approach to the management of such patients – with the participation of internists, cardiologists, endocrinologists, and rehabilitologists – will help mitigate the effects of post-covid syndrome, prevent the development of complications (hypertensive crises, strokes, heart attacks, and diabetic comas), and improve the prognosis for patients with chronic non-communicable diseases in the context of the ongoing COVID-19 pandemic and its long-term consequences.

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