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RISK FACTORS FOR DEVELOPMENT OF TYPE 2 DIABETES MELLITUS IN PATIENTS WITH OBESITY IN LATE POST-COVID PERIOD

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ФАКТОРЫ РИСКА РАЗВИТИЯ САХАРНОГО ДИАБЕТА 2-го ТИПА У ПАЦИЕНТОВ С ОЖИРЕНИЕМ В ОТДАЛЕННОМ ПОСТКОВИДНОМ ПЕРИОДЕ

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Objective. To study the risk factors for developing type 2 diabetes mellitus in patients with obesity after COVID-19.
Materials and methods. 61 case histories and outpatient card abstracts of patients with obesity, who suffered from moderate and severe forms of COVID-19 from 02.2021–04.2022 were analyzed. Demographic, laboratory and clinical parameters were studied during hospitalization and 12 months after discharge from the hospital. All patients initially were divided into 2 groups according to the glycated hemoglobin level. Group 1 consisted of 46 patients with prediabetes and group 2 included 15 patients without carbohydrate disorders.
Results. The median age of all patients was 64 (59–66) years. Median of HbAlc was 6,0 (5,6–6,2) %, BMI – 34 (33–35) kg/m^2. 24 patients from group 1, who took DPP-4-inhibitors in early post-COVID-19 period constituted subgroup 1A and 22 patients, who refused treatment with these drugs, constituted subgroup 1B. Currently, 2 patients from subgroup 1A and 10 patients from subgroup 1B (χ^2=8,2 p=0,004) have been diagnosed with DM2. In patients who developed DM2 in late post-COVID period the levels of HbAlc, fasting plasms glucose and BMI at the time of admission to the hospital were significantly higher (n=12) than in patients with persistent prediabetes (n=34), (p<0,05). Positive correlation between these parameters and the risk of developing DM2 (R=0,5, p<0,05; R=0,74, p<0,05; R=0,54, p<0,05, respectively) was determined. In group 2, DM2 is currently di-

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agnosed in 2 male patients with BMI over 40 kg/m^2. When comparing subgroup 1B and group 2, it was found out that DM2 in the post-COVID period occurs in every second patient with the previous initial carbohydrate disorders: in 10 people of 22 – in subgroup 1B (every 2nd patient) versus 2 patients from group 2 (every 7th patient), (χ^2=4,2, p=0,04). Online calculator from medstatistic. ru was used to determine relative risk (*RR*).
Conclusions. Thus, presence of impaired glucose tolerance increases the risk of development of DM2 in late post-COVID period. In patients with hyperlycemia on hospitalization for COVID-19, who did not receive incretin therapy (subgroup 1B) risk of DM2 was 3,4 times higher (*CI* 95 %=0,87–13,40). Patients, who received incretin (subgroup 1A) had risk of DM2 = 0,6 (*CI* 95 %=0,09–3,97). It should be assumed that incretin therapy prevents development of DM2 in patients with hyperglycemia/impaired glucose tolerance after COVID-19.
Keywords. Obesity, COVID-19, hyperglycemia, impaired glucose tolerance, type 2 diabetes mellitus, dipeptydilpeptidase-4-inhibitors.

Цель. Изучить факторы риска развития сахарного диабета 2-го типа (СД2) у лиц с ожирением, перенесших COVID-19.
Материалы и методы. Проанализирована 61 история болезни и выписки амбулаторных карт пациентов с ожирением, перенесших COVID-19 средней и тяжелой степени в период 02.2021–04.2022. Изучены демографические, лабораторные и клинические параметры в период госпитализации и спустя 12 месяцев с момента выписки из стационара. По уровню гликированного гемоглобина (HbAlc, %) больные исходно были разделены на две группы: группу 1 составили 46 пациентов с нарушением толерантности к глюкозе и группу 2 – 15 человек без углеводных нарушений. Количественные данные представлены в виде медианы.
Результаты. Медиана возраста 61 больного составила 64 (59–66) года. Медиана HbAlc 6,0 (5,6–6,2) %, индекса массы тела (ИМТ, кг/м^2) – 34 (33–35) кг/м^2. После выписки из стационара 24 пациента из 1-й группы, принимавшие ингибиторы дипептидилпептидазы 4-го типа (иДПП-4), сформировали подгруппу 1А, в подгруппу 1Б вошли 22 пациента, не принимавшие терапию по причине отказа. К настоящему времени СД2 диагностирован у 2 пациентов из подгруппы 1А и у 10 человек из подгруппы 1Б (χ^2 = 8,2; p = 0,004). Значения HbAlc, глюкозы плазмы натощак и ИМТ в период госпитализации по причине COVID-19 были достоверно выше у пациентов с развившимся в позднем постковидном периоде СД2 (n = 12) по сравнению с пациентами без СД2 (n = 34) (p < 0,05). Выявлена положительная корреляция между риском развития СД2 и HbAlc, глюкозой плазмы натощак, ИМТ (R = 0,5, p < 0,05; R = 0,74, p < 0,05; R = 0,54, p < 0,05, соответственно). В группе 2 в настоящее время диагноз СД2 установлен у 2 мужчин с ИМТ более 40 кг/м^2. При сравнении подгруппы 1Б и группы 2 выявлено, что СД2 в постковидном периоде встречается при наличии предшествующих углеводных нарушений у каждого второго пациента: у 10 человек из 22 в подгруппе 1Б против 2 человек из группы 2 (каждый 7-й пациент) (χ^2 = 4,2; p = 0,04). Для определения относительного риска (*RR*) взят online-калькулятор с medstatistic.ru.
Выводы. Таким образом, наличие гипергликемии (нарушение толерантности к глюкозе) увеличивает риск развития СД2 в отдаленном постковидном периоде. У пациентов с гипергликемией во время госпитализации с COVID-19, не получавших инкретиновую терапию (подгруппа 1Б), риск развития СД2 увеличивался в 3,4 раза (ДИ 95 % = 0,87–13,40). У пациентов, получавших инкретин (подгруппа 1А), риск развития СД2 увеличивался в 0,6 раза (ДИ 95 % = 0,09–3,97). Следует предположить, что инкретиновая терапия профилактирует развитие СД2 у пациентов с гипергликемией после перенесенного COVID-19.
Ключевые слова. Ожирение, COVID-19, гипергликемия, нарушение толерантности к глюкозе, сахарный диабет 2-го типа, ингибиторы дипептидилпептидазы 4-го типа.

INTRODUCTION

The current decade brought together two pandemics, obesity and COVID-19, which have aggravated pathogenetic mechanisms. Studies have shown that obese patients constituted the main cohort of patients hospitalized with COVID-19 [1; 2]. Notably, obesity leads to a more severe course of COVID-19 and increases the risk of poor outcomes of this viral infection [3]. In 2020, mortality from COVID-19 was 10

times higher in countries where majority of the adult population is overweight [4]. According to Chinese researchers, obesity occurs in 41.7 % of patients with COVID-19, second to arterial hypertension [5]. Owing to its pathogenetic commonality, obesity is often combined with carbohydrate disorders, including among patients with COVID-19 [6]. According to the Russian studies "ACTIV" and "ACTIV 2," hyperglycemia was detected in 28.9 % of COVID-19 patients [7]. In such patients, newly diagnosed hyperglycemia may indicate the presence of diabetes mellitus (DM), prediabetes, or transient steroid-induced hyperglycemia [8]. To verify the type of carbohydrate disorder, glycated hemoglobin was determined. Not all patients, especially those without an established DM diagnosis, have normal glycemic levels during the period of convalescence after COVID-19. The history of COVID-19 infection contributes to the deterioration of control of existing DM and progression of prediabetes to diabetes, which aggravates the course of postCOVID syndrome in these patients [9; 10]. Researchers have focused on new cases of type 2 DM (T2DM) following COVID-19. The risk of developing T2DM after COVID-19 is 4.9 % higher than the average in the population [11]. Special attention should be paid to patients with prediabetes, as they have a greater risk of developing T2DM. DM was found to aggravate the course of COVID-19, and therefore, the search for drugs that reduce hyperglycemia and improve the prognosis continued throughout the epidemic. The effects of the main groups of glucose-lowering drugs (GLDs) on the course of the acute period of COVID-19 in T2DM patients have been analyzed. Dipeptydilpeptidase-4 (DPP-4) inhibitors and sodium-glucose co-transporter 2 inhibitors are promising and priority groups of GLDs in the treatment of postCOVID hyperglycemia; however, no data was recorded on their use in patients with prediabetes (*of label* prescription) and hyperglycemia [10]. Studies that analyzed the outcomes of newly diagnosed hyperglycemia against COVID-19 in patients with prediabetes in the late postCOVID period are few.

Thus, this study aimed to assess the significance of risk factors for the development of type 2 DM in obese individuals with a history of COVID-19.

MATERIALS AND METHODS

A retrospective analysis of 61 medical histories and outpatient records of obese patients who had moderate COVID-19 between February 2021 and April 2022 was performed. Demographic, laboratory, and clinical parameters during hospitalization and 12 months after discharge from the hospital were studied. Laboratory data include determination of levels of hemoglobin, ESR, blood plasma glucose, glycated hemoglobin (HbAlc, %), D-dimer, and procalcitonin, which were determined on hospitalization day two. Data were obtained from electronic health records. Inclusion criteria were age >50 years, COVID-19 diagnosis confirmed by chest CT and PCR, BMI >30 kg/m^2, and HbAlc level (%)

upon admission <6.5 %. All patients received glucocorticosteroid therapy during the acute phase of COVID-19. According to the level of HbAlc (%) determined upon hospital admission, patients were distributed into two groups: group 1, 46 patients with prediabetes (HbAlc < 6.5 %–> 5.8 %), and group 2, 15 people without carbohydrate disorders. Statistical processing of the results was performed using the Statistica 6 package. Quantitative characteristics are presented in the form of a median. The significance of differences between the groups was assessed using nonparametric comparison methods based on qualitative and quantitative criteria, namely, the Mann–Whitney U test and χ^2 criterion. Spearman's test (*R*) was used for correlation analysis. To determine the relative risk, an online calculator from medstatistic.ru was utilized.

RESULTS AND DISCUSSION

The median age of the 61 patients was 64 (59–66) years; of the total number of patients, 34 were men (56 %) and 27 (44 %) were women. The median glycated hemoglobin and BMI was 6.0 (5.6–6.2)% and 34 (33–35) kg/m^2, respectively. A comparative analysis of all parameters was performed between the groups of patients at baseline. The data is presented in Table 1.

After hospital discharge, patients in group 1 were recommended to have a combination of metformin and DPP-4-inhibitors for 3 months, with further referral to an endocrinologist.

The use of DPP-4 inhibitors in the group with impaired glucose tolerance (IGT) should be clarified. At the time of hospital discharge, all patients from group 1 (IGT) were diagnosed with T2DM, which was established based on additional glycemic tests performed during hospitalization, despite having an initial HbAlc level of <6.5 %. Low availability of endocrinologists on an outpatient basis at that time was considered, which required hospital endocrinologists to prescribe glucose-lowering therapy upon discharge. Therefore, the use

Table 1

Comparative characteristics of the main parameters in groups 1 and 2 (median)

Parameter	Group 1, *n* = 46	Group 2, *n* = 15	*P*
Age, years	64 (59–67)	63 (58–65)	n.a.
BMI, kg/m^2	34.5 (33.5–35.0)	35.0 (34.0–39.0)	n.a.
Hemoglobin, g/l	125 (120–128)	127 (120–138)	n.a.
ESR, mm/h	62 (55–67)	49.5 (32–60)	n.a.
Glucose, mmol/l	8.1 (7.9–9.1)	6.5 (5.9–7.3)	0.000
Glycated hemoglobin, %	6.0 (6.0–6.2)	5.3 (5.2–5.5)	0.000
Procalcitonin, ng/ml	1.2 (0.22–2.1)	0.8 (0.2–1.6)	n.a.
CRP, mg/l	53.5 (24–83.5)	43 (12–79)	n.a.

Note: n.a., not available.

Table 2

Comparative characteristics of the main parameters in subgroups 1A and 1B (median)

Parameter	Subgroup 1A, $n = 24$	Subgroup 1B, $n = 22$	P
Age, years	63.5 (60.5–67)	62 (56–69)	n.a.
BMI, kg/m^2	33.0 (32.5–35.0)	34.0 (34.0–36.0)	n.a.
Hemoglobin, g/l	126 (121–128)	122 (120–135)	n.a.
ESR, mm/h	60 (57–61)	59.5 (42–63)	n.a.
Glucose, mmol/l	8.0 (7.7–8.8)	8.2 (7.9–8.9)	n.a.
Glycated hemoglobin, %	6.0 (5.9–6.2)	6.1 (5.8–6.3)	n.a.
Procalcitonin, ng/ml	1.4 (0.25–2.0)	0.9 (0.4–1.8)	n.a.
CRP, mg/l	43.5 (24–76.5)	48 (32–80)	n.a.

of DPP-4 inhibitors in this situation was indicated. Moreover, during that time, information about the beneficial effects of incretin therapy on the course and outcomes of DM in COVID-19 was provided [12; 13]. Thus, these patients had a diagnosis of T2DM; however, from the perspective of current concepts, we continued to interpret their condition as IGT.

Among 46 patients, 24, who made up the subgroup 1A, took these drugs for at least 3 months, and 22, who made up the subgroup 1B, refused treatment. No significant differences were noted between the main parameters in the subgroups during hospitalization (Table 2).

By the time of outpatient consultation with an endocrinologist, 4–6 months after hospitalization, the patients were not taking DPP-4 inhibitor therapy or metformin. After additional analysis, the patients' condition was reclassified. By this time, T2DM was diagnosed in two patients from subgroup 1A and 10 from subgroup 1B ($\chi^2 = 8.2$; $p = 0.004$). In patients with currently confirmed T2DM ($n = 12$), during hospitalization, the values of HbAlc, fasting plasma glucose, and BMI were significantly higher compared to the corresponding data for those who maintained the prediabetes state ($n = 34$; $p < 0.05$) (Figs. 1–3). In patients with persistent prediabetes, the median HbAlc values were 6.0 (5.8–6.1)%, fasting plasma glucose values were 8.0 (7.7–8.2) mmol/l, BMI were 34 (32–35) kg/m^2, versus 6.35 (6.2–6.4)%, 10.25 (9.4–11.1) mmol/l, and 35.5 (34.5–37) kg/m^2 ($p < 0.05$), respectively (Figs. 1–3).

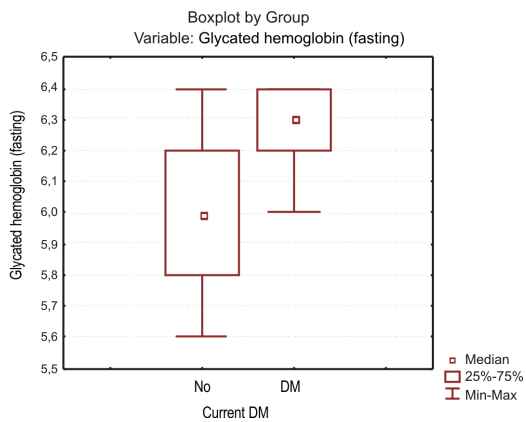

Fig. 1. HbAlc values during hospitalization in patients with currently established T2DM diagnosis and persistent prediabetes

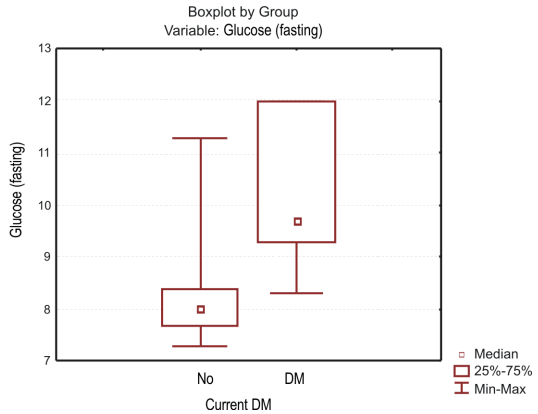

Fig. 2. Maximum plasma glucose values during hospitalization in patients with a current T2DM diagnosis and persistent prediabetes

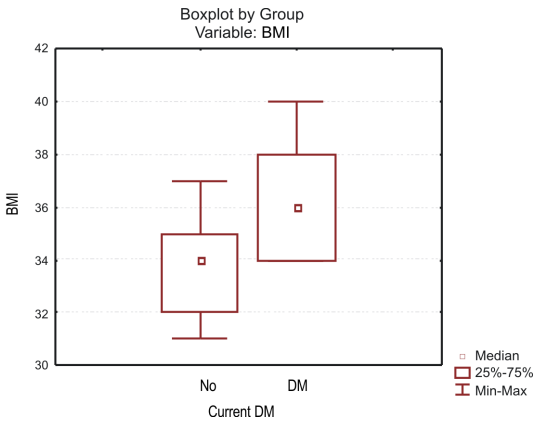

Fig. 3. BMI values during hospitalization in patients with currently established T2DM diagnosis and persistent prediabetes

A positive correlation was revealed between the maximum values of fasting blood plasma glycemia, glycated hemoglobin, BMI, and the risk of developing T2DM ($R = 0.5$, $p < 0.05$; $R = 0.74$, $p < 0.05$; $R = 0.54$, $p < 0.05$, respectively). No significant differences were noted in age, levels of acute phase proteins, and the degree of lung tissue damage during hospitalization between the groups of patients with new-onset T2DM and persistent prediabetes.

In group 2, T2DM was diagnosed in two male patients with morbid obesity (BMI > 40 kg/m^2). When comparing data between subgroup 1B and group 2, it was revealed that T2DM in the postCOVID period occurs in the presence of previous carbohydrate disorders in every second patient who did not use treatment, that is, in 10 of 22 patients in subgroup 1B versus 2 patients from group 2 (every seventh patient) ($\chi^2 = 4.2$; $p = 0.04$). When comparing data from subgroup 1A and group 2, it was determined that T2DM in the postCOVID period occurs in every 11th patient with hyperglycemia who received DPP-4 inhibitors and in every 6th–7th patient without carbohydrate disorders at hospitalization ($p > 0.05$). In patients who did not receive incretin therapy (subgroup 1B), the risk of T2DM occurrence increased by 3.4 times (95 % CI = 0.87–13.40). In patients receiving incretin (subgroup 1A), the risk of T2DM increased by only 0.6 times (95 % CI = 0.09–3.97). Thus, patients with hyperglycemia who received DPP-4 inhibitors in the early postCOVID period and those without carbohydrate disorders have similar incidence of T2DM in the long-term period. It should be assumed that DPP-4 inhibitors reduce the risks of T2DM in patients with hyperglycemia after COVID-19. The protective role of DPP-4 inhibitors in COVID-19 is presented in several studies and justifies the use of this group of drugs in T2DM patients with a history of COVID infection. Studies have reported that patients taking DPP-4 inhibi-

tors had a milder course of this viral infection [14; 15]. The mechanism by which DPP-4 inhibitors protect the body from severe COVID-19 has been studied and is due to inhibition of the DPP-4 enzyme. Moreover, a high level of this enzyme increases the body's susceptibility to the SARS-CoV-2 virus, reduces the effect of incretins and thus causing hyperglycemia, and increases the production of proinflammatory factors [16]. T2DM and obese patients have shown increased DPP-4 activity, which affects the course of the acute phase of COVID-19 and postCOVID period and the risk of T2DM occurrence.

Conclusions

Previous carbohydrate disorders (prediabetes) and high BMI values are crucial in the development of T2DM in obese patients who have had moderate and severe COVID-19. The presence of hyperglycemia (IGT) increases the risk of developing T2DM in the late postCOVID period. Therapy with metformin and DPP-4 inhibitors may play a protective role in this process. The absence of incretin therapy in patients with hyperglycemia after COVID-19 leads to the development of T2DM 3.5 times more often than in patients taking DPP-4 inhibitors. It should be assumed that incretin therapy prevents T2DM development in patients with hyperglycemia (IGT) after COVID-19, and the possibility of prescribing this group of drugs to all patients with obesity and hyperglycemia should be considered. In obese patients without carbohydrate disorders, weight loss should be recommended to reduce the risk of developing T2DM.

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