EXCELLENCIA
INTERNATIONAL MULTI-DISCIPLINARY JOURNAL OF EDUCATION
Volume 02, Issue 08, 2024 ISSN (E): 2994-9521

Features of the Flow COVID-19in Patients with Type 2 Diabetes Mellitus

Juraeva M. Sh , Ashurova N.G

Bukhara State Medical University

Abstract:

Concomitant diabetes mellitus (DM) negatively affects the prognosis of patients with COVID-19, being associated with high rates of mortality, complications and the need for intensive care [1–3].

The pathophysiological causes of severe SARS-CoV-2 viral infection in diabetes are being actively studied and include:

1. the negative impact of hyperglycemia on the course of any infection [6, 7];
2. damage to β-cells during COVID-19, with a further increase in hyperglycemia and additional activation of pro-inflammatory mechanisms [8, 9];
3. mutual overlap and aggravation of systemic inflammatory changes characteristic of diabetes and COVID-19, including disturbances in the production of cytokines and the elimination of microorganisms [10–14];
4. The influence of the SARS-CoV-2 virus on the renin-angiotensin-aldosterone system, with an imbalance of angiotensins and an increase in insulin resistance [15].

Goal of the work— to study the course of COVID-19 with concomitant diabetes mellitus, to assess the impact of carbohydrate metabolism disorders and systemic inflammation on the severity of the concomitant pathology.

MATERIALS AND METHODS

An open comparative prospective study was conducted, during which 109 patients with COVID-19 who were admitted to the infectious diseases hospital of the Bukhara region from 2021-2023 were observed.

Inclusion criteria for the study were:

A positive test for COVID-19 based on a swab from the nasopharynx and oropharynx and 2) viral pneumonitis based on computed tomography (CT) results. All patients were divided into two groups: the main group (57 patients with COVID-19 with concomitant diabetes) and the control group (52 patients without diabetes). The criteria for inclusion of patients in the main group included a history of diabetes and/or a level of glycated hemoglobin (HbA1c) above the upper limit of normal upon admission.

When comparing baseline demographic indicators, patients without diabetes were expectedly characterized by lower age (53.90±15.52 vs. 60.20±11.75 years, p≤0.05) and body mass index BMI (29.60±4.81 vs. 31.70±5.58 kg/m2, p≤0.05).

There were no differences between the control and main groups in gender composition (women 52 (63.4±5.3%) and men 39 (68.4±6.2%), respectively) and in the duration of COVID-19 until the day of hospitalization (10, 00±3.65 vs. 10.40±8.44 days, p≥0.05).

Table 1. Comorbid pathology in observation groups

Disease	Group 1 (without diabetes), n=52,p±σp, %	Group 2 (with diabetes), n=57,p±σp, %
Obesity	30.5±5.1	59.6±6.5fi
Arterial hypertension	50.0±5.5	75.4±5.7fi
Cardiac ischemia	22.0±4.6	33.3±6.2
Chr. heart failure	2.4±1.7	3.5±2.4
Chr. obstructive diseaselungs	4.9±2.4	5.3±3.0
Bronchial asthma	3.7±2.1	7.0±3.4
Rheumatological diseases	1.2±1.2	3.5±2.4
Diseases kidney	7.3±2.9	12.3±4.4
Oncological pathology	6.1±2.6	1.8±1.7
Diseases Gastrointestinal tract	28.0±4.9	15.8±4.8

Note: statistical significance of differences between the main and control groups, * - p≤0.01; ** — p≤0.001.

As for comorbid pathology in the observation groups (Table 1), among patients with COVID-19 with concomitant diabetes, obesity and arterial hypertension were naturally more often detected, and there were no differences in the prevalence of coronary heart disease (CHD), chronic heart failure, chronic obstructive pulmonary disease , bronchial asthma, rheumatological diseases, diseases of the gastrointestinal tract and other pathologies.

Table 2. Nosological peculiarities SD among sick COVID-19

SD (type; length of service)	Total,p±σp, % ofthe number of all pain-new COVID-19	Gender male/female, abs. number	Averageage, years
SD,Total	41.0±4.2	18/39	60.20±11.75
Type 2 diabetes:– first identified SD-2 ≤5 years –SD-2 >5 years	39.6±4.1 20.8±3.4 6.6±2.1 12.2±2.8	18/37 12/17 4/5 2/15	59.60±11.63 59.80±10.68 59.90±12.77 62.70±12.97
Type 1 diabetes	1.4±1.0	0/2	.00±11.31

RESULTS AND DISCUSSION

Upon admission, the group of patients with diabetes was characterized (Table 3) by significantly higher clinical risk scores on the SMRT-CO scale, volumes of lung damage on CT, the proportion of patients with oxygen saturation less than 95 (with a sevenfold greater percentage of patients with SpO2 less than 90%), and also by a decrease in the initial level of SpO2 relative to the control.

In dynamics, the slow and incomplete recovery of lung tissue in patients with diabetes was confirmed by a large proportion of patients with SpO2 less than 95% after one and two weeks of treatment, an increase in the time of normalization of SpO2 (approximately doubling), a large percentage of people with residual lung damage ≥50% in CT upon discharge.

As a result, with concomitant diabetes, COVID-19 patients required longer inpatient treatment (in terms of bed days).

Another consequence of the greater severity of pulmonary disorders in combined pathology was the need to intensify therapy for COVID-19

Table 3. Indicators clinical currents COVID-19 V groups observations

Index	Group 1 (without diabetes), n=52	Group 2 (DM), n=57
Proportions of persons with different amounts of lung damage according to CT (p±σp, %) On admission:		
• before 50%	69.5±5.1	56.1±6.6
• ≥50%	28.5±5.1	43.9±6.6
Upon discharge (if done):		
• before 50%	84.1±4.0	70.1±6.9fi
• >50%	12.2±3.6	26.3±5.8fi
Percent damagelungs		
Upon admissionUpon dischargeRdin.	42.10±19.13 32.00±15.93 ≤0.0000	48.40±16.84fi 42.20±20.07fifi ≤0.0001
SMRT-CO scale (points)	2.00±1.14	3.0 [1.0; 3.0]fi
Decrease in SpO2 (p±σp, %)		
On admission:		
SpO2 ≥95	81.7±4.3	52.7±
SpO2 92–94	15.8±4.0 2.5±1.7	6.5fifi 29.8±
SpO2<90	90.2±3.3 9.8±3.3 0	6.1fi 17.5±5.0fifi 59.6±6.6fifi
After 1 week: *SpO2* ≥95	95.1±2.4 4.9±2.4	28.1±5.9fifi 12.3±4.4
SpO2 92–94	0	75.4±5 .7fififi 24.6 ±5.7f
SpO2<90		ififi 0
In 2 weeks: *SpO2* ≥95		
SpO2 92–94		
SpO2<90		
SpO2 upon admission	93.60±3.71	91.80±4.92fi
Day normalization **SpO2**	4.80±4.57	9.50±6.77fififi
Bed day	14.8±5.1	16.40± 4.87fi
Adverse Exodus (p±σp, %)	14.6±3.9	26.3±5.8
Treatment in the ICU	14.6±3.9	21.1±5.4
LethalExodus	7.3±2.9	8.8±3.7

Notes: statistical significance of differences between the main and control groups: * - p≤0.05; ** — p≤0.01; *** — p≤0.001; Rdin - statistical significance of differences in indicators over time (from admission to discharge).

(Table 4). Against the background of diabetes, modern immunosuppressants were significantly more often prescribed (3.2 times), combined antiviral drugs (6.2 times), as well as intravenous antibacterial drugs (including levofloxacin and amoxicillin clavulanate).

To identify mechanisms and factors associated with the severity of COVID-19, correlation analysis was used: for all patients (n=109), significant relationships between SMRT-CO risk values and demographic and clinical indicators were examined. Of the clinical indicators, only the presence of concomitant diseases such as diabetes (ρ=0.17, p≤0.05), kidney disease (including diabetic nephropathy, ρ=0.23, p≤0) was associated with high risk according to the SMRT-CO scale .01), as well as, at the trend level, IHD (ρ=0.16, p=0.053). It can be concluded that SD refers to the most significant concomitant diseases that aggravate COVID-19.

The maintenance of hyperglycemia in the combination of COV ID-19 and diabetes may be facilitated by a complex of factors associated both with the consequences of the SARS-CoV-2 viral infection itself and with the therapy (for example, corticosteroids, which were prescribed to 43.9% of people with COVID-19 and SD).

Table 5. Laboratory indicators in patients with COVID-19 against the backgrounddiabetes mellitus

Indicators	Group 1 (without SD)	Group 2 (with SD)
Glucose,mmol/lOn admissionUpon discharge Rdin	4.90±0.83 4.70±0.52 ≥0.05	7.50±2.75fi 5.90±1.69 ≤0.05
SRB, mg/lOn admissionDay 3–5 Atdischarge Rdin Normalization Day	37.5 [19.0; 103.0] 12.0 [5.0;22.0] 5.0 [3.0; 6.0] 9.10±5.22 ≤0.001	47.0 [25.0; 118.0] 34.5 [17.0; 77.5]fifi 5.0 [4.0; 9.2] 12.2±6.05fi ≤0.001
ESR,mm/hOn admissionUpon discharge from Rdin	27.40±19.26 16.40±12.58 ≤0.001	27.0 [14.0; 52.0] 19.0 [10.0; 31.0]fi ≤0.001
Leukocytes,x10^9/lUpon	5.50 [4.55; 7.34]	6.70 ± 3.78

admission Upon discharge Rdin	5.50 [4.64; 7.13] ≥0.05	5.80 [4.69; 7.54] ≥0.05
Hemoglo bin,g/lOn admissio nUpon discharge from Rdin	139.50±15.8 1 126.90±17.2 6 ≤0.001	139.60±15 .10 126.30±17 .94 ≤0.001

Note: statistical significance of differences between the main and control groups, * - p≤0.01; ** — p≤0.001; Rdin - statistical significance of differences in indicators over time (from admission to discharge).

With regard to the HbA1c level, which was determined in individuals with COVID-19 and diabetes, there were no significant correlations with indicators of disease severity, but there was a relationship with initial blood glucose levels (ρ=0.37, p≤0.05).

According to correlation analysis, the activity of systemic inflammation was directly related to the severity of the disease according to the SMRT-CO index (for CRP on admission ρ=0.42, p≤0.001, for ESR on admission ρ=0.29, p≤0.001).

Conclusions:

Diabetes mellitus is one of the most significant comorbid diseases that aggravate the SARS-CoV-2 viral infection. The course of COV ID-19 against the background of diabetes mellitus is characterized by a greater severity of viral pneumonitis, the severity and persistence of oxygen saturation disorders, the prevalence, as well as delayed and incomplete elimination of pulmonary lesions on CT, an increase in the need of patients for modern immunosuppressive, glucocorticoid and combination antiviral therapy, an extension of the period hospitalization.

Hyperglycemia in patients with SARS-CoV-2 infection and diabetes mellitus is associated with greater severity of viral pneumonitis, respiratory failure and systemic inflammatory changes. Elevated levels of glycated hemoglobin are less closely related to the severity of concomitant pathology.

LITERATURE/REFERENCES

1. Onder G., Rezza G., Brusaferro S. Case-fatality rate and characteristics of patients dying in relation to COVID-19 in Italy. JAMA 2020; 323(18): 1775–1776, https://doi.org/10.1001/jama.2020.4683.
2. Guan WJ, Liang WH, Zhao Y., Liang HR, Chen ZS, Li YM,
3. Liu XQ, Chen RC, Tang CL, Wang T., Ou CQ, Li L., Chen PY,
4. Sang L., Wang W., Li JF, Li CC, Ou LM, Cheng B., Xiong S., Ni ZY,
5. Xiang J., Hu Y., Liu L., Shan H., Lei CL, Peng YX, Wei L., Liu Y., Hu YH,
6. Peng P., Wang JM, Liu JY, Chen Z., Li G., Zheng ZJ, Qiu SQ, Luo J.,
7. Ye CJ, Zhu SY, Cheng LL, Ye F., Li SY, Zheng JP, Zhang NF,

8. Zhong NS, He JX; China Medical Treatment Expert Group for CO VID-19. Comorbidity and its impact on 1590 patients with COVID-19 in China: a national analysis. Eur Respir J 2020; 55(5): 2000547, https://doi.org/10.1183/13993003.00547–2020.

9. Wu Z., McGoogan JM Characteristics of and important lessons from the coronavirus disease 2019 (COVID-19) outbreak in China: summary of a report of 72314 cases from the Chinese Center for Disease Control and Prevention. JAMA 2020; 323(13): 1239,https://doi.org/10.1001/jama.2020.2648.

10. Glybochko P.V., Fomin V.V., Avdeev S.N., Moiseev S.V., Yavo-Rovsky A. G., Brovko M. Yu., Umbetova K. T., Aliev V. A., Bulanova E. L., Bondarenko I. B., Volkova O. S., Gainitdinova V. V., Gneusheva T. Yu., Dubrovin K. V., Kapustina V. A., Kraeva V. V., Merzhoeva Z. M., Nuralieva G. S., Nogtev P. V., Panasyuk V. V., Politov M. E., Popov A.M., Popova E.N., Raspopina N.A., Royuk V.V., Sorokin Yu.D., Trushenko N.V., Khalikova E.Yu., Tsareva N. . A., Chikina S. Yu., Chichkova N. V., Akulkina L. A., Bulanov N. M., Ermolova L. A., Zykova A. S., Kitbalyan A. A., Moise- ev A. S., Potapov P. P., Tao E. A., Sholomova V. I., Shchepalina A. A., Yakovleva A. A. Clinical characteristics of 1007 patients with severe SARS-COV-2 pneumonia who needed in respiratory support. Clinical Pharmacology and Therapeutics 2020; 29(2): 21–29, https://doi.org/10.32756/0869–5490–2020–2–21–29.

11. Glybochko PV, Fomin VV, Avdeev SN, Moiseev SV, Yavorovskiy AG, Brovko M. Yu., Umbetova KT, Aliev VA, Bulanova EL, Bondarenko IB, Volkova OS, Gaynitdinova VV, Gneusheva T. Yu., Dubrovin KV, Kapustina VA, Kraeva VV, Merzhoeva ZM, Nuralieva GS, Nogtev PV, Panasyuk VV, Politov ME, Popov AM, Popova EN, Raspopina NA, Royuk VV, Sorokin Yu.D., Trushenko NV, Khalikova E. Yu ., Tsareva NA, Chikina S. Yu., Chichkova NV, Akulkina LA, Bulanov NM, Ermolova LA, Zykova AS, Kitbalyan AA, Moiseev AS, Potapov PP, Tao EA, Sholomova VI, Shchepalina AA, Yakovleva AA Clinical characteristics of 1007 intensive care unit patients with SARS-COV-2 pneumonia. Klinicheskaya farmakologia i terapia 2020; 29(2): 21–29, https://doi.org/10.32756/0869–5490–2020–2–21–29.

12. Shestakova M. V., Vikulova OK., Isakov M.A., De-

13. Dov I. I. Diabetes mellitus and COVID-19: analysis of clinical outcomes according to the diabetes registry of the Russian Federation. Problems of endocrinology 2020; 66(1): 35–46, https://doi.org/10.14341/probl12458. Shestakova M.V., Vikulova OK, Isakov M.A., Dedov II Diabetes and COVID-19: analysis of the clinical outcomes according to the data of the Russian diabetes registry. Problemy endokrinologii 2020; 66(1): 35–46, https://doi. org/10.14341/probl12458.

14. Li S., Wang J., Zhang B., Li X., Liu Y. Diabetes mellitus and cause-specific mortality: a population-based study. Diabetes Metab J 2019; 43(3): 319–341, https://doi.org/10.4093/dmj.2018.0060.

15. Pearson-Stuttard J., Blundell S., Harris T., Cook DG, Critch-Ley J. Diabetes and infection: assessing the association with glycemic control in population-based studies. Lancet Diabetes Endocrinol 2016; 4(2): 148–158, https://doi.org/10.1016/s2213–8587(15)00379–4.

16. Wang A., Zhao W., Xu Z., Gu J. Timely blood glucose management for the outbreak of 2019 novel coronavirus disease (COV ID-19) is urgently needed. Diabetes Res Clin Pract 2020; 162: 108118, https://doi.org/10.1016/j.diabres.2020.108118.

17. Iqbal A., Prince L.R., Novodvorsky P., Bernjak A., Thom-as MR, Birch L., Lambert D., Kay LJ, Wright FJ, Macdonald IA, Jacques RM, Storey RF, McCrimmon RJ, Francis S., Heller SR, Sabroe I. Effect of hypoglycemia on inflammatory responses and the response to low -dose

endotoxemia in humans. J Clin Endocrinol Metab 2019; 104(4): 1187–1199, https://doi.org/10.1210/ jc.2018–01168.

18. Drucker DJ Coronavirus infections and type 2 diabetes shared pathways with therapeutic implications. Endocr Rev 2020; 41(3): 457–470, https://doi.org/10.1210/endrev/bnaa011.
19. Mehta P., McAuley D.F., Brown M., Sanchez E., Tattersall R.S.
20. Manson JJ; HLH Across Specialty Collaboration, UK. COVID-19: consider cytokine storm syndromes and immunosuppression. Lancet 2020; 395(10229): 1033–1034, https://doi.org/10.1016/s0140–6736(20)30628–0.
21. Hodgson K., Morris J., Bridson T., Govan B., Rush C., Keth-eesan N. Immunological mechanisms contributing to the double burden of diabetes and intracellular bacterial infections. Immunol- ogy 2015; 144(2): 171–185, https://doi.org/10.1111/imm.12394.
22. Kulcsar KA, Coleman CM, Beck SE, Frieman MB Co-morbid diabetes results in immune dysregulation and enhanced disease severity following MERS-CoV infection. JCI Insight 2019; 4(20): e131774, https://doi.org/10.1172/jci.insight.131774.
23. Chen X., HuW., Ling J., Mo P., Zhang Y., Jiang Q., Ma Z., Cao Q., Deng L., Song S., Zheng R., Gao S., Ke H., Gui X., Lundkvist Å., Li J., Lin-Dahl JF, Xiong Y. Hypertension and diabetes delay the viral clear- ance in COVID-19 patients. medRxiv 2020: 2020.03.22.20040774, https://doi.org/10.1101/2020.03.22.20040774.
24. Šestan M., Marinović S., Kavazović I., Cekinović Đ., Wueest S.,
25. Turk Wensveen T., Brizić I., Jonjić S., Konrad D., Wensveen FM, Polić B. Virus-induced interferon-γ causes insulin resistance in skeletal muscle and derails glycemic control in obesity. Immunity 2018; 49(1): 164–177.e6, https://doi.org/10.1016/j.immuni.2018.05.005.