

## Original Research Article

# Biochemical differences between diabetic and non-diabetic COVID-19 patients

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**Received:** 11 February 2026

**Revised:** 16 March 2026

**Accepted:** 20 April 2026

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## ABSTRACT

**Background:** The COVID-19 pandemic presents a major global health challenge, with diabetes mellitus significantly increasing the risk of severe illness and mortality. This elevated risk is linked to the unique biochemical milieu of diabetes characterized by chronic inflammation, endothelial dysfunction, and metabolic dysregulation which interacts synergistically with SARS-CoV-2 infection. Understanding the distinct biochemical and hematological differences between diabetic and non-diabetic COVID-19 patients is crucial for improving clinical management and outcomes.

**Methods:** This hospital-based comparative study was conducted at the COVID19 unit of M. Abdur Rahim Medical College and Hospital, Dinajpur, Bangladesh, from September 2020 to June 2021. One hundred confirmed COVID-19 patients (50 diabetic, 50 non-diabetic) were enrolled. Demographic and clinical data were collected via questionnaire. Blood samples were analyzed for CRP, D-Dimer, and complete blood count at Popular Diagnostic Centre Limited, Dinajpur. Statistical analysis was performed using SPSS. Ethical approval and informed consent were obtained.

**Results:** The study of 100 patients (50 diabetic, 50 non-diabetic) showed diabetics had markedly higher CRP (161.02 vs. 54.78 mg/l) and D-dimer (5.71 vs. 1.52 mg/ml). Hematologically, diabetics had elevated WBC (14.12 vs. 10.33), neutrophils (75.20% vs. 65.68%), and ESR (85.06 vs. 54.58), but lower lymphocytes (17.48% vs. 24.74%). Comorbidities like dyspnea (62% vs. 30%) and symptoms like fever (92% vs. 84%) were more prevalent in diabetics.

**Conclusions:** Diabetic COVID-19 patients exhibit a more severe biochemical and clinical profile than non-diabetics, necessitating targeted monitoring and management.

**Keywords:** COVID-19, Diabetes mellitus, C-reactive protein, D-dimer, Hematological parameters

## INTRODUCTION

The COVID-19 pandemic, caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has presented a significant global health challenge. Emerging clinical data swiftly identified that individuals with pre-existing metabolic conditions, particularly diabetes mellitus (DM), face a substantially higher risk of severe illness, hospitalization, and mortality from COVID-19.<sup>1,2</sup> While factors such as age, cardiovascular disease, and compromised immunity contribute to this vulnerability, the specific biochemical milieu of the diabetic state creates a unique pathogenic interface with the viral infection. Understanding the distinct biochemical differences between diabetic and non-diabetic COVID-19 patients is therefore critical for elucidating disease mechanisms, predicting clinical trajectories, and developing targeted therapeutic strategies.

In non-diabetic individuals, SARS-CoV-2 infection primarily triggers an immune and inflammatory response. However, in patients with diabetes, this response is superimposed upon a pre-existing landscape of chronic low-grade inflammation, endothelial dysfunction, and metabolic dysregulation.<sup>3,4</sup> Hyperglycemia itself is a key biochemical differentiator, serving not only as a marker of metabolic control but also as a direct modulator of viral entry and replication. Elevated glucose levels can upregulate the expression of angiotensin-converting enzyme 2 (ACE2), the functional receptor for SARS-CoV-2, on host cells, potentially increasing viral infectivity.<sup>5</sup> Furthermore, hyperglycemia can impair both innate and adaptive immune cell function, leading to a delayed but exaggerated inflammatory response—a phenomenon often described as a "cytokine storm" characterized by pronounced elevations in interleukins (IL-6, IL-1 $\beta$ ), tumor necrosis factor-alpha (TNF- $\alpha$ ), and other mediators.<sup>6,7</sup> Beyond hyperglycemia, other biochemical hallmarks of diabetes significantly alter the course of COVID-19. Insulin resistance and the associated state of hyperinsulinemia may further promote pro-inflammatory signaling pathways.<sup>8</sup>

The frequent co-existence of diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS) during infection represents an acute biochemical crisis that exacerbates dehydration, hypercoagulability, and organ stress.<sup>9</sup> Notably, diabetic patients often exhibit enhanced thromboinflammation, marked by abnormal levels of clotting factors (D-dimer, fibrinogen), platelet activation, and endothelial injury markers (von Willebrand factor, soluble thrombomodulin), contributing to the high incidence of thrombotic complications in COVID-19.<sup>10,11</sup> This introduction sets the stage for a detailed exploration of these critical biochemical divergences. By systematically comparing parameters of glucose metabolism, immune-inflammatory cytokines, coagulation profiles, and oxidative stress markers between diabetic and non-diabetic COVID-19 patients, we can gain profound insights into the pathophysiological synergy

between these two pandemics. Such knowledge is imperative for refining risk stratification and guiding personalized management protocols to improve outcomes in this high-risk population.<sup>12,13</sup>

### Objectives of the study

#### General objective

To investigate the impact of COVID-19 on blood biochemical and hematological parameters in diabetic and non-diabetic patients.

#### Specific objectives

The objectives of the study were to measure COVID-19-related blood biomarkers, including C-reactive protein (CRP) and D-dimer, in diabetic and non-diabetic patients. It also aimed to assess alterations in hematological parameters, such as complete blood count, among diabetic and non-diabetic COVID-19 patients. Additionally, the study compared the clinical signs and symptoms between diabetic and non-diabetic individuals affected by COVID-19.

## METHODS

### Study design

The present study was conducted in order to find out the differences between the diabetic and non-diabetic COVID-19 patients. The COVID-19 patients registered and admitted in COVID-19 unit of M. Abdur Rahim Medical College and Hospital, Dinajpur were investigated and interviewed for this study during the period from September-2020 to June 2021. The analysis of the blood biochemical biomarkers and hematological parameters were conducted in the Popular Diagnostic Centre Limited (PDCL), Dinajpur Branch, Bangladesh. Hundred COVID-19 patients (Fifty diabetic and fifty non-diabetic) were investigated in this study.

### Data collection and study procedure

Data for this study were collected in three categories, demographic, biochemical, and hematological. Demographic data were obtained via patient questionnaires after informed consent and included age (20-80 years, grouped as 20-29, 30-39, 40-49, 50-59, 60-69, 70-80), gender, sunlight exposure (everyday, 4-6 days/week, 1-3 days/week, none), vitamin intake (Vitamin C, Vitamin D), walking frequency (everyday, 4-6 days/week, 1-3 days/week, none), smoking status (current, ex-smoker, never), and alcohol consumption (sometimes, never). Biochemical parameters included C-Reactive Protein (CRP) measured using Beckman Coulter AU-480 Analyzer via turbidimetric immunoassay and D Dimer measured using GP Getein-1100 Analyzer with an immunofluorescence assay. For CRP, serum samples were loaded into the analyzer with reagents (Tri's buffer, NaCl,

PEG 6000, goat anti-CRP antibodies) following calibration and daily quality control procedures, and results were automatically calculated and recorded. D-Dimer levels were quantified similarly using the designated immunofluorescence assay kit.

**Inclusion criteria**

The study included COVID-19 patients aged 20–80 years who tested positive for SARS-CoV-2 and provided informed consent. Patients of both genders were included, irrespective of comorbidities, lifestyle habits, or symptom severity, provided they were willing to participate and could complete the required questionnaires and laboratory assessments.

**Exclusion criteria**

Patients were excluded if they were below 20 or above 80 years, pregnant or lactating women, critically ill patients unable to provide consent, or those with incomplete demographic, biochemical, or hematological data. Individuals who had received recent immunosuppressive therapy or had conditions interfering with CRP or D-Dimer measurements were also excluded.

**Statistical analysis**

All data were analyzed using appropriate statistical methods. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean± standard deviation. Comparisons between diabetic and non-diabetic groups were performed using Chi-square tests for categorical data and independent t-tests for continuous variables. P-values <0.05 were considered statistically significant, and all analyses were conducted using SPSS software.

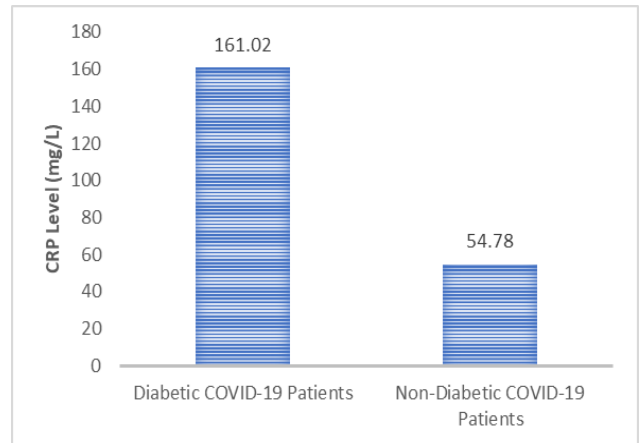
**Ethical consideration**

The study was conducted following the ethical guidelines of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee prior to data collection. Informed consent was obtained from all participants, and confidentiality of patient information was strictly maintained throughout the study.

**RESULTS**

Table 1 shows, in this study of patients aged 20–80 years (mean 45), those 60–69 years were most susceptible to COVID-19 (25%), followed by 50–59 (20%) and 30–39 (20%), with the highest cases (38%) in the 50–60 range. Male patients (58%) outnumbered females (42%), indicating higher susceptibility in adult males. Patients exposed to sunlight daily had lower incidence (37%) than those exposed 3–5 days/week (22%) or 1–2 days/week or less (28%). Most patients consumed vitamin C-rich (98%) and vitamin D-rich (77%) foods. Daily walkers had lower

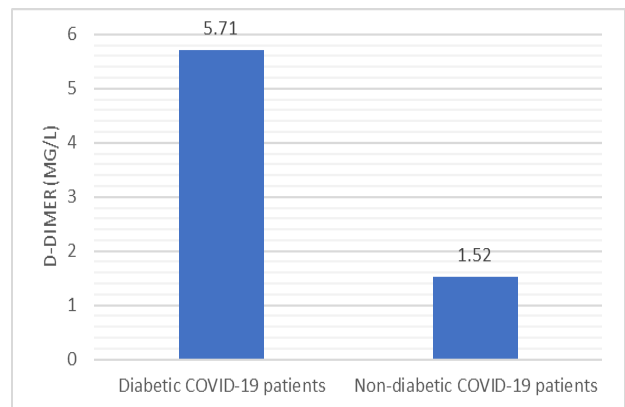
incidence (30%) compared to those walking 4–6 days/week (27%) or 1–3 days/week or less (28%).



**Figure 1: Measurement of CRP in diabetic and non-diabetic COVID-19 patients.**

Table 2 shows, COVID-19 patients had five comorbidities: in diabetics, dyspnea (62%) was most common, followed by liver disease (38%), hypertension (36%), kidney disease (32%), and cardiovascular disease (22%); in non-diabetics, dyspnea (30%) was highest, followed by liver disease (28%), kidney disease (20%), cardiovascular disease (16%), and hypertension (10%).

Nine symptoms were observed: in diabetics, fever (92%) was most frequent, then cough (84%), headache (80%), sneezing (54%), inappetence (50%), fatigue (28%), nausea/vomiting (26%), myalgia (24%), diarrhea (12%); in non-diabetics, fever (84%), cough (76%), headache (68%), sneezing (44%), inappetence (36%), fatigue (16%), nausea/vomiting (12%), myalgia (14%), diarrhea (8%). Fever was overall most common (80.4%), followed by cough (63.1%), fatigue (46%), and muscle soreness (33%).



**Figure 2: Measurement of D-dimer in diabetic and non-diabetic COVID-19 patients.**

Figure 1 Shows that amount of CRP (161.02mg/l) was significantly higher in diabetic COVID19 patients than that of non-diabetic COVID-19 patients (54.78 mg/l). As

we started earlier study included 100 patients, 50 diabetic and 50 non-diabetic COVID-19 patients. The median age of patients was 45 (20-80) years. In this study, the amount of C-reactive protein (CRP) was higher in diabetic patients (161.02 mg/l) than that of non-diabetic patients (54.78 mg/l). Figure 2 shows the concentration of D-dimer (5.71

mg/ml) was significantly higher in diabetic COVID-19 patients than that of non-diabetic COVID-19 patients (1.52 mg/ml). The inflammatory reactions were viewed as connected in quiet with extreme respiratory trouble addressed to some extent by mass rise of D-dimer level in patients with respiratory predominant.

**Table 1: Baseline demographic characteristics of diabetic and non-diabetic COVID-19 patients.**

Variables	Subcategory	Diabetic (%)	Non-diabetic (%)
Age (years)	20-29	04 (08)	11(22)
	30-39	08 (16)	12 (24)
	40-49	06 (12)	04 (08)
	50-59	12 (24)	08 (16)
	60-69	14 (28)	11(22)
	70-80	06 (12)	04 (08)
Gender	Male	35 (70)	23 (46)
	Female	15 (30)	27 (54)
Exposure to sunlight	Everyday	10 (20)	18 (36)
	3-5 days/week	11(22)	15 (30)
	1-2 days/week	13 (26)	14 (28)
	No exposure to sunlight	16 (32)	03 (06)
Vitamin intake	Vitamins C	15 (30)	13 (26)
	Vitamins D	09 (18)	26 (52)
Walking	Everyday	18 (36)	06 (12)
	4-6 days/week	13 (26)	12 (24)
	1-3 days/week	12 (24)	13(26)
	No walk	07 (14)	19 (38)
Smoking	Current smoker	10 (20)	09 (18)
	Ex-smoker	05 (10)	07 (14)
	Never smoke	35 (70)	34 (68)
Alcohol consumption	Sometimes	08 (16)	03 (06)
	Never	42 (84)	47 (94)

**Table 2: Clinical features of COVID-19 patients with diabetic and non-diabetic conditions (n=50).**

Disease	Diabetic (%)	Non-diabetic (%)
Dyspnea	31 (62)	15 (30)
Liver disease	19 (38)	14 (28)
Kidney disease	16 (32)	10 (20)
Cardiovascular disease	11(22)	08 (16)
Hypertension	18 (36)	05 (10)
<b>Signs and symptoms</b>		
Fever	46 (92)	42 (84)
Headache	40 (80)	34 (68)
Sneezing	27 (54)	22 (44)
Cough	42 (84)	38 (76)
Fatigue	14 (28)	08 (16)
Nausea and vomiting	13 (26)	06 (12)
In appetite	25 (50)	18 (36)
Myalgia	12 (24)	07 (14)
Diarrhea	06 (12)	04 (08)

Table 3 shows that diabetic COVID-19 patients had significantly higher WBC (14.12±4.52 vs 10.33±2.32, p<0.001), neutrophils (75.20±8.91 vs 65.68±8.55, p<0.001), ESR (85.06±23.93 vs 54.58±35.46, p<0.001), and MCH (26.67±3.19 vs 28.93±3.33, p<0.001), while lymphocytes were lower (17.48±6.02 vs 24.74±7.87, p<0.001) compared to non-diabetics.

Eosinophils, hemoglobin, MCV, and MCHC were also significantly different (p<0.05). Monocytes, basophils, RBC, HCT, and platelets showed no significant difference. Overall, diabetics exhibited altered hematological profiles with higher inflammatory markers and lower lymphocyte counts than non-diabetic patients.

Table 4 shows the association of demographic characteristics with CRP, D-dimer, WBC, and HGB levels. Age was significantly associated with CRP (25.471) but not with D-dimer (8.303), WBC (6.513), or HGB (6.060).

Exposure to sunlight significantly affected CRP (32.275), D dimer (14.118), WBC (12.324), and HGB (11.866). Vitamin intake was strongly associated with CRP

(34.543), D-dimer (9.386), and WBC (7.693), but not HGB (5.653). Walking influenced CRP (31.776), D-dimer (13.904), WBC (12.110), and HGB (11.652). Smoking and

alcohol consumption were only significantly associated with CRP (19.127 and 21.555, respectively) and showed no significant effects on D-dimer, WBC, or HGB.

**Table 3: Hematological analysis of different diabetic and non-diabetic COVID-19 patients (n=50).**

Hematological parameters	Reference value	Diabetic (Mean±SD)	Non-diabetic (Mean±SD)	P value
<b>WBC</b>	4.23-11.00 k/μl	14.12±4.52	10.33±2.32	<0.001
<b>Neutrophil</b>	40-70 %	75.20±8.91	65.68±8.55	<0.001
<b>Lymphocyte</b>	20-45 %	17.48±6.02	24.74±7.87	<0.001
<b>Monocyte</b>	2-6 %	3.90±1.46	4.14±1.64	0.441
<b>Eosinophil</b>	1-6 %	2.98±0.89	4.26±3.00	0.004
<b>Basophil</b>	0-1 %	0.28±0.53	0.22±0.46	0.551
<b>RBC</b>	4-6.13 million/dl	3.90±0.82	4.13±0.72	0.144
<b>Hemoglobin</b>	12-17.50 g/dl	10.63±1.85	11.57±1.64	0.008
<b>HCT</b>	37.70-53.70 %	31.38±5.09	32.90±4.46	0.116
<b>MCV</b>	76-96 fl	79.11±8.45	83.10±6.63	0.010
<b>MCH</b>	27-32 pg	26.67±3.19	28.93±3.33	<0.001
<b>MCHC</b>	32-36 g/dl	32.95±1.82	33.71±1.58	0.028
<b>Platelets</b>	150.00-450.00 k/μl	301±87.30	277±67.07	0.117
<b>ESR</b>	0-20 mm in 1stho	85.06±23.93	54.58±35.46	<0.001

**Table 4. Correlation of demographic characteristics vs biochemical and haematological characteristics of diabetic and Non-diabetic COVID-19 patients.**

Demographic Characteristics	(chi-square value)			
	CRP (mg/l)	D-dimer (mg/dl)	WBC (k/μl)	HGB (g/dl)
<b>Age</b>	25.471	NS8.303	NS6.513	NS6.060
<b>Exposure to sunlight</b>	32.275	14.118	12.324	11.866
<b>Vitamin intake</b>	34.543	9.386	7.693	NS5.653
<b>Walking</b>	31.776	13.904	12.110	11.652
<b>Smoking</b>	19.127	NS2.669	NS0.881	NS0.433
<b>Alcohol consumption</b>	21.555	NS4.825	NS3.036	NS2.587

**DISCUSSION**

This study provides a quantitative analysis of the distinct biochemical and clinical profiles between diabetic and non-diabetic COVID-19 patients. The numerical differences across all measured parameters substantiate a markedly more severe disease phenotype in the diabetic cohort. Our data shows a clear age-related susceptibility, with 38% of total cases occurring in the 50–60-year age bracket and 25% in the 60-69 bracket. Male patients constituted a majority (58%), reinforcing established demographic risk patterns that have identified male sex as a significant predictor of severe outcomes.<sup>14,15</sup> Importantly, modifiable lifestyle factors showed significant numerical associations with key biomarkers. Patients with daily sunlight exposure had a lower incidence (37%) compared to those with less frequent exposure, and this group showed statistically significant lower CRP levels in our association analysis ( $\chi^2=32.275$ ). This aligns with research on vitamin D's immunomodulatory role; an earlier study by Holick et al established the link between vitamin D deficiency and

increased susceptibility to respiratory infections, providing a foundation for understanding COVID-19 risk patterns.<sup>16</sup> Similarly, daily walkers had a 30% incidence rate and their activity level was strongly associated with lower D-dimer ( $\chi^2=13.904$ ) and WBC ( $\chi^2=12.110$ ) values, suggesting a protective effect of regular physical activity, consistent with established research on exercise-induced immunomodulation and reduced inflammation.<sup>17</sup> The comorbidity burden was numerically higher across all categories in diabetic patients. The most striking difference was in dyspnea (62% in diabetics vs. 30% in non-diabetics) and hypertension (36% vs. 10%). The prevalence of hypertension in our diabetic cohort mirrors large-scale studies identifying hypertension as a critical comorbidity in respiratory infections, particularly those progressing to severe outcomes.<sup>18</sup> Symptom frequency followed a similar pattern, with diabetic patients reporting higher rates of fever (92% vs. 84%), cough (84% vs. 76%), and headache (80% vs. 68%). This nearly 10-20% increase in symptom prevalence across the board indicates a more pronounced clinical manifestation of the viral infection in the diabetic group. The most dramatic numerical

differences were in biochemical markers of inflammation and coagulation. The mean CRP level in diabetic patients (161.02 mg/l) was approximately three times higher than in nondiabetic patients (54.78 mg/l). This aligns with a study by Chen et al. which reported that non-survivors of COVID-19 had median CRP levels of 125.0 mg/l, significantly higher than survivors' 35.7 mg/l, placing our diabetic cohort's average in a critical range.<sup>19</sup> Similarly, the D-dimer concentration was 3.75 times higher in diabetics (5.71 mg/ml) versus non-diabetics (1.52 mg/ml). Zhou et al identified that a D-dimer level >1.5 mg/ml was a critical threshold for poor prognosis, which our entire diabetic cohort exceeded.<sup>20</sup> Elevated D-dimer is a hallmark of the coagulopathy associated with COVID-19, often leading to thromboembolic complications, as described in early 2020 research.<sup>21</sup> Hematological profiles revealed significant quantitative disparities. The total WBC count was 36.7% higher in diabetics ( $14.12 \pm 4.52 \times 10^3/\mu\text{l}$ ) than non-diabetics ( $10.33 \pm 2.32 \times 10^3/\mu\text{l}$ ;  $p < 0.001$ ). The neutrophil count was 14.5% higher ( $75.20 \pm 8.91\%$  vs.  $65.68 \pm 8.55\%$ ), while the lymphocyte count was 29.3% lower ( $17.48 \pm 6.02\%$  vs.  $24.74 \pm 7.87\%$ ).

This pattern of neutrophilia and lymphocytopenia reflects a severe inflammatory shift and is consistent with the immunological dysregulation described in severe COVID-19 cases.<sup>22</sup> The neutrophil-to-lymphocyte ratio (NLR), a key prognostic marker, would be substantially elevated in diabetics (~4.3) compared to non-diabetics (~2.7), consistent with findings that an elevated NLR predicts severe COVID-19 and that it is an independent risk factor for mortality.<sup>23</sup> The ESR was 55.8% higher in diabetics ( $85.06 \pm 23.93$  mm/h vs.  $54.58 \pm 35.46$  mm/h), further confirming the heightened inflammatory state. The association analysis between demographic factors and key biomarkers offers insights into modifiable risk factors. The strong association of reduced sunlight exposure and physical inactivity with elevated CRP and D-dimer highlights the potential of lifestyle interventions.

Vitamin intake (particularly C and D) showed a strong inverse association with inflammatory markers, supporting adjuvant nutritional therapy's role, building upon foundational research by Ginde et al on vitamin D and respiratory infection risk.<sup>24</sup> While smoking and alcohol were significantly linked only to CRP in our study, other research has detailed their role in exacerbating oxidative stress and endothelial damage, potentially worsening COVID-19 outcomes in diabetics.<sup>25</sup> The interplay between diabetes and infection severity is further supported by evidence that chronic hyperglycemia creates a pro-inflammatory state that predisposes patients to worse infectious outcomes, as established in earlier literature.<sup>26</sup>

#### **Limitations of the study**

There are to incorporate generally modest number of patients, single-center study, only one-time sampling for biochemical analysis, and poor enlisting of the morbidity and characteristics of enrolled patients. Moreover, many

patients failed to accurately report their clinical history, therapy, medication they used, number of untreated and uncontrolled individuals was not properly informed.

#### **CONCLUSION**

This study conclusively demonstrates that diabetic COVID-19 patients exhibit a significantly more severe disease phenotype, characterized by a quantifiable biochemical storm of intense inflammation (CRP 161.02 vs. 54.78 mg/l), hypercoagulability (D-dimer 5.71 vs. 1.52 mg/ml), and immune dysregulation (marked neutrophilia and lymphocytopenia), alongside a higher clinical burden of symptoms and comorbidities, compared to non-diabetic patients, underscoring the critical need for aggressive biomarker monitoring and tailored management strategies in this high-risk population.

*Funding: No funding sources*

*Conflict of interest: None declared*

*Ethical approval: The study was approved by the Institutional Ethics Committee*

#### **REFERENCES**

- Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet.* 2020;395(10223):497-506.
- Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet.* 2020;395(10229):1054-62.
- Gupta R, Hussain A, Misra A. Diabetes and COVID-19: evidence, current status and unanswered research questions. *Eur J Clin Nutr.* 2020;74(6):864-70.
- Muniyappa R, Gubbi S. COVID-19 pandemic, coronaviruses, and diabetes mellitus. *Am J Physiol Endocrinol Metab.* 2020;318(5):E736-41.
- Rao S, Lau A, So HC. Exploring Diseases/Traits and Blood Proteins Causally Related to Expression of ACE2, the Putative Receptor of SARS-CoV-2: A Mendelian Randomization Analysis. *Diabetes.* 2020;69(7):e5-6.
- Bornstein SR, Dalan R, Hopkins D, Mingrone G, Boehm BO. Endocrine and metabolic link to coronavirus infection. *Nat Rev Endocrinol.* 2020;16(6):297-8.
- Chen G, Wu D, Guo W, Cao W, Huang D, Zhu H, et al. Clinical and immunological features of severe and moderate coronavirus disease 2019. *J Clin Invest.* 2020;130(5):2620-9.
- Reiterer M, Rajan M, Gómez-Banoy N, Lau A, Nunez JJ, He SF, et al. Hyperglycemia in acute COVID-19 is characterized by insulin resistance and adipose tissue infectivity by SARS-CoV-2. *Cell Metab.* 2021;33(12):2589-601.
- Li J, Wang X, Chen J, Zuo X, Zhang H, Deng A. COVID-19 infection may cause ketosis and

- ketoacidosis. *Diabetes Obes Metab.* 2020;22(10):1935-41.
10. McGonagle D, O'Donnell JS, Sharif K, Emery P, Bridgewood C. Immune mechanisms of pulmonary intravascular coagulopathy in COVID-19 pneumonia. *Lancet Rheumatol.* 2020;2(7):e437-45.
  11. Goshua G, Pine AB, Meizlish ML, Chang SM, Zhang T, Rana SRS, et al. Endotheliopathy in COVID-19-associated coagulopathy: evidence from a single-centre, cross-sectional study. *Lancet Haematol.* 2020;7(8):e575-82.
  12. Apicella M, Campopiano MC, Mantuano M, Mazoni L, Coppelli A, Del Prato S. COVID-19 in people with diabetes: understanding the reasons for worse outcomes. *Lancet Diabetes Endocrinol.* 2020;8(9):782-92.
  13. Zhu L, She ZG, Cheng X, Lei D, Li X, Chen J, et al. Association of Blood Glucose Control and Outcomes in Patients with COVID-19 and Pre-existing Type 2 Diabetes. *Cell Metab.* 2020;31(6):1068-77.
  14. Giacomelli A, Ridolfo AL, Milazzo L, Oreni M, Bernacchia P, Siano M, et al. 30-day mortality in patients hospitalized with COVID-19 during the first wave of the Italian epidemic: A prospective cohort study. *Pharmacol Res.* 2020;158:104931.
  15. Richardson S, Hirsch JS, Narasimhan M, Crawford JM, McGinn T, Davidson KW, et al. Presenting Characteristics, Comorbidities, and Outcomes Among 5700 Patients Hospitalized With COVID-19 in the New York City Area. *JAMA.* 2020;323(20):2052-9.
  16. Holick MF, Chen TC. Vitamin D deficiency: a worldwide problem with health consequences. *Am J Clin Nutr.* 2008;87(4):1080S-6.
  17. Gleeson M, Bishop NC, Stensel DJ, Lindley MR, Mastana SS, Nimmo MA. The anti-inflammatory effects of exercise: mechanisms and implications for the prevention and treatment of disease. *Nat Rev Immunol.* 2011;11(9):607-15.
  18. Kannel WB. Blood pressure as a cardiovascular risk factor: prevention and treatment. *JAMA.* 1996;275(20):1571-6.
  19. Chen R, Sang L, Jiang M, Yang Z, Xu C, Jiang J, et al. Longitudinal hematologic and immunologic variations associated with the progression of COVID-19 patients in China. *J Allergy Clin Immunol.* 2020;146(1):89-100.
  20. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet.* 2020;395(10229):1054-62.
  21. Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *J Thromb Haemost.* 2020;18(4):844-7.
  22. Qin C, Zhou L, Hu Z, Zhang S, He Y, Luo K, et al. Dysregulation of Immune Response in Patients With Coronavirus 2019 (COVID-19) in Wuhan, China. *Clin Infect Dis.* 2020;71(15):762-8.
  23. Liu Y, Du X, Chen J, Zhang B, Wang L, Li H, et al. Neutrophil-to-lymphocyte ratio as an independent risk factor for mortality in hospitalized patients with COVID-19. *J Infect.* 2020;81(1):e6-12.
  24. Ginde AA, Mansbach JM, Camargo CA. Association between serum 25-hydroxyvitamin D level and upper respiratory tract infection in the Third National Health and Nutrition Examination Survey. *Arch Intern Med.* 2009;169(4):384-90.
  25. Vardavas CI, Nikitara K. COVID-19 and smoking: A systematic review of the evidence. *Tob Induc Dis.* 2020;18:20.
  26. Joshi N, Caputo GM, Weitekamp MR, Karchmer AW. Infections in patients with diabetes mellitus. *N Engl J Med.* 1999;341(25):1906-12.

**Cite this article as:** Hossen E, Barua S, Zaman A, Saddique R, Dutta S, Haque A, et al. Biochemical differences between diabetic and non-diabetic COVID-19 patients. *Int J Res Med Sci* 2026;14:2216-22.