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PREVALENCE OF NEW-ONSET HYPERGLYCEMIA AND DIABETES IN HOSPITALIZED ADULTS WITH COVID-19

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COVID-19 infection may exacerbate glycemia and lead to metabolic problems in people with diabetes. Emerging data suggest that diabetes may also develop during coronavirus infection in individuals with no prior history of the disease. This study aimed to analyze the prevalence of new-onset hyperglycemia and diabetes mellitus (nDM) in COVID-19 patients. A retrospective study was conducted at AL-Kindy Teaching Hospital in Baghdad, Iraq, from August 2021 to January 2022, including a convenience sample of 150 non-diabetic COVID-19 patients. Data were extracted from medical records and included demographic information, disease severity, laboratory findings, presence of comorbidities, and disease outcomes. nDM was defined as a glucose level > 200 mg/dL on two occasions with no previous history of diabetes mellitus (DM). The mean age of participants was 54.81 ± 14.8 years, with a male-to-female ratio of 1.3:1. During hospitalization, 40 (26.7%) patients developed nDM; among them, 17.5% had moderate, 62.5% severe, and 20.0% critical COVID-19 infection (P = 0.370). nDM was associated with non-smoking status (35%, P = 0.026), hypertension (62.5%, P = 0.045), elevated D-dimer levels (3.261 ± 3.197 g/L, P = 0.036), and reduced lymphocyte counts (0.92 × 10^9 cells/L ± 0.98, P = 0.010). Non-smoking status and higher D-dimer levels were significant predictors of nDM, with odds ratios (95% CI) of 0.418 (0.175–0.997) and 1.2 (1.04–1.38), respectively, but nDM was not associated with worse outcomes. In conclusion, new-onset diabetes was observed in approximately one-fourth of hospitalized COVID-19 patients, but it did not predict adverse clinical outcomes.

Keywords: COVID-19, hyperglycemia, new-onset diabetes, outcome

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INTRODUCTION

It has been established that diabetes and COVID-19 are interconnected. Diabetes increases the risk of severe COVID-19 infection and has also been linked to COVID-19-induced severe acute respiratory syndrome (SARS-CoV-2) (1, 2). COVID-19 is a virus that can cause pneumonia and other respiratory complications. In infected individuals, the virus may affect pancreatic beta cells by exploiting the expression of angiotensin-converting enzyme 2 (ACE2) receptors. This could lead to a decrease in insulin secretion and, as a consequence, either an exacerbation of pre-existing diabetes or the development of new-onset diabetes mellitus (nDM) (3). Insulin resistance, which may be induced by elevated levels of interleukin-6 and tumor necrosis factor-alpha in COVID-19 patients, is likely another contributing factor to the development of diabetes (4, 5). Moreover, evidence suggests that COVID-19 patients with nDM experience worse outcomes compared to those with controlled glycemia or pre-existing diabetes (6, 7). Determining a direct causal relationship is challenging due to the presence of major confounding variables, which remains an unresolved question. Concerns have also been raised regarding glucocorticoid therapy, as it can trigger nDM and potentially exacerbate its negative impact on COVID-19 severity (7). Therefore, our study aimed to analyze the prevalence of new-onset hyperglycemia and diabetes in COVID-19 patients and to investigate its association with patient characteristics, disease severity, and clinical outcomes.

METHODS

This retrospective cohort study was conducted at Al Kindy General Teaching Hospital in Al-Rusafa Health Directorate, Baghdad, Iraq, from August 1, 2021, to January 31, 2022.
The study population included 150 COVID-19 patients admitted to an isolation ward at a tertiary care unit in Al Kindy Hospital in Baghdad between August and October 2021. Eligible participants were adults aged 18 years or older with a confirmed COVID-19 diagnosis by RT-PCR., while those with pre-existing diabetes and pregnant women were excluded from the study. The study was approved by the Ethics Committee of Medical Research at Al-Kindy College of Medicine, Baghdad University, under registration number 3676 on June 14, 2021.

Sampling method and data collection
Data were obtained from the documented records of 150 COVID-19 patients selected using convenience sampling. Information was collected using a questionnaire completed by the researcher during hospital visits. The dataset included demographics (age, gender, weight, and height), disease severity (moderate, severe, or critical), and laboratory results: white blood cell (WBC) count, lymphocyte, neutrophil, and platelet counts, lactate dehydrogenase (LDH), serum ferritin (s.ferritin) and D-dimer, comorbidities, including hypertension, obesity, and smoking, and disease outcomes (recovery or death).
All included patients were hospitalized with moderate, severe, or critical COVID-19 infection and received steroid therapy. Disease severity was classified according to the guidelines of the World Health Organization (WHO), which define the clinical spectrum as follows:
Mild disease - patients exhibit symptoms of COVID-19 without evidence of viral pneumonia or hypoxia;
Moderate disease (pneumonia): patients show signs of pneumonia, including fever, cough, dyspnea, and rapid breathing, but without severe pneumonia. Oxygen saturation (SpO_2) level is of 90% or higher on room air. Chest imaging (ultrasound, radiograph, CT scan) may aid in diagnosis and rule out or confirm pulmonary problems.
Severe disease (severe pneumonia): pneumonia symptoms, including high temperature, cough, shortness of breath, and rapid heart rate, in addition to one of the following – a respiratory rate exceeding 30 breaths per minute, acute respiratory distress, or a SpO_2 level below 90% on room air, contributed to a severe case of pneumonia.
Medical emergency (acute respiratory distress syndrome): onset occurs within seven days of new or worsening respiratory symptoms or a recognized clinical insult, such as pneumonia, and is confirmed by chest imaging (ultrasound, CT, or radiograph). Imaging of the chest (ultrasound, computed tomography, or radiograph) when nodules, lobar or lung collapse, or volume overload cannot adequately explain bilateral opacities.
Critical illness (septic shock): patients may experience sudden, potentially fatal organ failures. Indicators of organ dysfunction include laboratory findings such as coagulopathy, thrombocytopenia, acidosis, elevated lactate, or hyperbilirubinemia, as well as clinical signs including altered mental status, hypoxia, oliguria, weak pulse, and hypotension. Septic shock is a critical illness characterized by persistent low blood pressure (hypotension) despite adequate resuscitation of blood volume; the patient must be given vasopressors to maintain mean arterial pressure (MAP) at 65 mmHg or

higher and a serum lactate level of more than 2 mmol/L (8). New-onset diabetes mellitus (nDM): nDM is defined as two separate random blood sugar readings (RBS) (200 mg/dL or more) on different days during hospitalization, according to Davidson Principles & Practice of Medicine, edition 23 (9).

Statistical analysis

Data entry and statistical analysis were performed using SPSS software version 23 (Statistical Package for the Social Sciences). Continuous variables were expressed as mean (range), while categorical variables were presented as frequencies and percentages. The Mann–Whitney U test was used to compare continuous variables, and the Chi-square or Fisher's exact test was applied for categorical variables, as appropriate. Factors associated with mortality were predicted using multivariate logistic regression analysis, with results reported as odds ratios (ORs) and 95% confidence intervals. A p-value < 0.05 was considered statistically significant.

Data collection, analysis, and interpretation of results, along with drafting and preparing the manuscript, were conducted over an estimated period of six months.

RESULTS

This study included 150 COVID-19 patients with a mean age of 54.81 ± 14.8 years. Of these, 85 (56.7%) were male, and 65 (43.3%) were female. Patients were categorized into age groups as follows: 18–39 years (23, 15.3%), 40–59 years (64, 42.7%), and ≥ 60 years (63, 42.0%). There were 33 (22%) smokers, 73 (48.7%) hypertensive patients, and 79 (52.7%) obese patients (BMI ≥ 30), as shown in Table 1.

As presented in Table 2, 84 (56%) patients had severe disease, while 27 (18%) were critically ill. During hospitalization, 40 (26.7%) patients developed new-onset diabetes mellitus (nDM), defined as blood glucose levels > 200 mg/dL on two separate occasions. Recovery occurred in 102 (68%) patients, whereas 48 (32%) died.

Table 3 compares the demographic and clinical characteristics of patients who developed nDM during the course of the disease with those who did not. Two of the most noteworthy characteristics were the lower prevalence of smoking (35% vs 65%, P = 0.026) and the higher prevalence of hypertension (62.5 vs 37%, P = 0.045). nDM developed in 7 out of 39 patients with moderate illness (17.9%), 25 out of 80 patients (29.8%) with severe illness, and 8 out of 27 patients (29.6%) with critical illness (P = 0.370). Although mortality was higher among patients with nDM (42.5%) compared to those

Table 1. *Demographic features of patients*

Demographic features		N	%
Gender	Male	85	56.7%
	Female	65	43.3%
Age group	**18-39 years**	23	15.3%
	40-59 years	64	42.7%
	≥ 60 years	63	42.0%
Smoking	No	117	78.0%
	Yes	33	22.0%
Hypertension	No	77	51.3%
	Yes	73	48.7%
BMI	Normal (18.5-24.9)	39	26%
	Overweight (25-29.9)	32	21.3%
	Obese (≥ 30)	79	52.7%
Total		150	100 %

Table 2. *Clinical consequences of disease*

Clinical consequences of the COVID infection		N	%
		65	43.3%
Severity	**Moderate**	39	**26**
	Severe	84	**56**
	Critical	27	**18**
Newly diagnosed DM	**No**	110	**73.3**
	Yes	40	**26.7**
Outcome	Recovered	102	**68**
	Death	48	**32**
Total		150	100

Table 3. *Demographic and clinical distribution of patients according to the new-onset diabetes (two high readings of RBS ≥ 200 mg/dL on different days)*

Variables		New-onset diabetes				P
		No		Yes		
		N	%	N	%	
Age group	18-39 years	17	15.5%	6	15.0%	**0.446**
	40-59 years	50	45.5%	14	35.0%	
	≥ 60 years	43	39.1%	20	50.0%	
Gender	Female	47	42.7%	18	45.0%	**0.804**
	Male	63	57.3%	22	55.0%	
Smoking	No	91	82.7%	26	65.0%	**0.026**
	Yes	19	17.3%	14	35.0%	
Hyperten-sion	No	62	56.4%	15	37.5%	**0.045**
	Yes	48	43.6%	25	62.5%	
BMI	Normal	28	25.5%	11	27.5%	**0.665**
	Overweight	22	20.0%	10	25.0%	
	Obese	60	54.5%	19	47.5%	
Severity	Moderate	32	29.1%	7	17.5%	**0.370**
	Severe	59	53.6%	25	62.5%	
	Critical	19	17.3%	8	20.0%	
Disease outcome	Recovered	79	71.8%	23	57.5%	**0.115**
	Died	**31**	**28.2%**	**17**	**42.5%**	

without (28.2%), this difference was not statistically significant.

When laboratory findings were compared between the two groups (Table 4), higher D-dimer levels and lower lymphocyte counts were significantly associated with the development of nDM: p = 0.036 and 0.010, respectively. Multivariate analysis identified non-smoking status and an increase in D-dimer level as predictors of nDM development during the course of COVID-19 infection, with OR of 0.418 and 1.2, respectively.

Further details are presented in Table 5, while patient outcomes and their associations with demographic and clinical characteristics are summarized in Table 6. Recovered patients were significantly younger than those who died (51 ± 15 vs. 62 ± 13 years, p < 0.001). The mortality rate among patients with severe (29.8%) and critical (77.8%) illness was significantly higher compared to those with moderate disease (5.1%). Although the development of DM during the course of the disease was not significantly associated with patient outcome, deceased patients had higher RBS levels (196 ± 66) compared to recovered patients (172 ± 59; p = 0.007). Higher levels of D-dimer, LDH, ferritin, urea, creatinine, white blood cell (WBC), neutrophil, and lymphocyte counts, as well as low platelet count, were associated with poorer patient outcomes.

Among these variables, increased patient age, severe and critical disease, and low platelet count significantly predicted poor patient outcome with OR (95% CI) of 1.059 (1.01-1.112), 3.47 (0.597-20.26), 83.7 (9.48-739.54), and 0.993 (0.987-0.999), respectively. Developing hyperglycemia during the course of the disease did not predict poor patient outcome. Infection was also not a significant predictor, with ORs of 0.418 and 1.2, respectively.

DISCUSSION

Previous studies have recognized the role of diabetes mellitus in the inflammatory response and progression of COVID-19. Recent studies have also reported elevated blood glucose levels in patients with COVID-19. Hyperglycemia in COVID-19, whether due to insulin resistance or pre-existing diabetes mellitus, has been associated with adverse effects on both disease course and outcomes. Recent literature suggests that COVID-19 may induce inflammation of pancreatic β-cells, potentially leading to new-onset diabetes mellitus (nDM) (10). This study highlights the prevalence of new-onset hyperglycemia and diabetes in adults admitted to the hospital due to COVID-19.

Disease severity

In the current study, more than half (56%) of COVID-19 cases were classified as severe. These findings are consistent with those of a study conducted in Kirkuk (11), in which 59.2% of cases were also classified as severe. However, a study from Turkey (12) reported a lower proportion (30.3%) of severe COVID-19 cases. The higher proportion of severe cases observed in our study may be attributed to differences in the study population, as only

Table 4. *Mean distribution of laboratory findings according to the new-onset diabetes (two readings of RBS > 200 mg/dL on different days)*

Lab test	nDM				p value
	No		Yes		
	Mean	SD	Mean	SD	
D-dimer (g/L)	2.078	2.341	3.261	3.197	**0.036**
LDH (U/L)	572	204	611	233	**0.386**
S. ferritin (µ/L)	526.1	220.6	524.7	169.8	**0.810**
Blood urea (mg/dL)	58.1	32.9	63.5	47.9	**0.954**
S. creatinine (mg/ dL)	0.95	0.63	1.03	1.14	**0.217**
WBC count (x 10^9 cell/L)	13.65	6.02	14.29	4.83	**0.249**
Neutrophil (x 10^9 cell/L)	9.97	4.68	10.44	4.29	**0.201**
Lymphocyte (x 10^9 cell/L)	1.19	1.05	0.92	.98	**0.010**
Platelet count (x 10^9 cell/L)	**273.1**	**92.3**	**263.8**	**125.6**	**0.362**

Table 5. *Multivariate analysis of new-onset diabetes in COVID-19 patients*

		OR	95% CI		p value
Smoking	No	Reference			**0.049**
	Yes	0.418	0.175	0.997	
Hypertension	No	Reference			**0.090**
	Yes	1.96	0.899	4.290	
D dimer (g/L)		1.197	1.041	1.376	**0.012**
Lymphocytes count (x 10^9 cell/L)		**0.753**	**0.472**	**1.201**	**0.233**

hospitalized patients were included, and the majority of participants were elderly, with a high prevalence of underlying comorbidities such as obesity and hypertension.

The prevalence of hyperglycemia among the studied COVID-19 patients was considerable, with 26.7% of patients developing hyperglycemia. This finding aligns with the results reported by Li H et al. (7), which showed that 20.75% of patients developed nDM. Similarly, a nationwide retrospective cohort study involving 12,817 non-diabetic patients reported the prevalence of 14% (13). In another study conducted by Zhang W et al. (14) in China, 12.5% of non-diabetic COVID-19 patients exhibited hyperglycemia. Similarly, a nationwide retrospective cohort study involving 12,817 non-diabetic patients reported a prevalence of 14% (13). In another study conducted by Zhang W et al. in China, 12.5% of non-diabetic COVID-19 patients exhibited hyperglycemia (14). Discrepancies in reported prevalence across studies may be attributed to differences in selection criteria and patient populations.

Hyperglycemia in COVID-19 may result from systemic inflammatory response and severe sepsis. In these conditions, elevated cytokine levels constitute an initial response and are associated with hyperglycemia (15, 16). The systemic inflammatory response syndrome has also been described as metabolic stress, which can induce glycogen breakdown, adrenocorticotropic hormone and catecholamine production, insulin resistance, and increased glucagon synthesis, all contributing to hyperglycemia (17, 18). Some studies have proposed that

Table 6. *Distribution of patients' outcomes according to demographic, clinical, and lab findings with multivariate analysis of significant variables*

Variables		Disease outcome				P value	OR (95% CI)	P value
		Recovered		Death				
		N	%	N	%			
Age	Mean ± SD)	51	±15	62	±13	< 0.001	1.059 (1.01-1.112)	0.023
Gender	Female	44	43.1%	21	43.8%	1		
	Male	58	56.9%	27	56.3%			
Smoking	No	79	77.5%	38	79.2%	0.837		
	Yes	23	22.5%	10	20.8%			
Hypertension	No	57	55.9%	20	41.7%	0.117		
	Yes	45	44.1%	28	58.3%			
BMI	Normal	31	30.4%	8	16.7%	0.159		
	Overweight	19	18.6%	13	27.1%			
	Obese	52	51.0%	27	56.3%			
Severity	Moderate	37	36.3%	2	4.2%	< 0.001	Reference	
	Severe	59	57.8	25	52.1%		3.47 (0.597-20.26)	0.166
	Critical	6	5.9	21	43.8%		83.7 (9.48-739.54)	0.001
Newly diagnosed DM	No	42	41.2%	22	45.8%	0.115		
	Yes	37	36.3%	26	54.2%			
RBS (mg/dL)	Mean (±SD)	172	±59	196	±66	0.007	1.007 (0.997-1.016)	0.160
D. dimer (g/L)	Mean (±SD)	131	±27	138	±30	< 0.001	0.96 (0.763-1.204)	0.713
LDH (U/L)	Mean (±SD)	172	±59	196	±66	< 0.001	1.003 (1.000-1.007)	0.051
S.ferritin (µ/L)	Mean (±SD)	1.949	±2.610	3.338	±2.466	< 0.001	1.000 (0.997-1.003)	0.910
B. urea (mg/dL)	Mean (±SD)	539	±200	676	±209	< 0.001	1.027 (1.006-1.047)	0.010
S. creatine (mg/dL)	Mean (±SD)	492.6	±228.0	596.2	±132.8	0.237		
WBC (x 10^9 cell/L)	Mean (±SD)	49.5	19.0	81.0	54.4	< 0.001	1.084 (0.953-1.233)	0.219.
Neutrophil (x 10^9 cell/L)	Mean (±SD)	.80	.27	1.33	1.28	< 0.001	0.985 (0.831-1.167)	0.859
Lymphocyte (x 10^9 cell/L)	Mean (±SD)	12.62	5.06	16.38	6.23	0.040	1.156 (0.648-2.063)	0.623
Platelet (x 10^9 cell/L)	Mean (±SD)	9.08	3.30	12.26	5.97	< 0.001	0.993(0.987-0.999)	0.018

SARS-CoV-2 may directly infect pancreatic β-cells, reducing insulin synthesis and secretion (19, 20). Additionally, excessive cytokine production in COVID-19 contributes to insulin resistance (19), suggesting that hyperglycemia may arise from a combination of impaired insulin secretion and resistance (19, 20).

Stress hyperglycemia, characterized by elevated blood glucose levels, is mediated by elevated levels of cytokines, particularly tumor necrosis factor and interleukin-1, and counter-regulatory hormones (21).

COVID-19 outcome: In this study, approximately two-thirds of patients achieved full recovery, whereas nearly one-third died (68% and 32%, respectively). These rates are higher than those reported in Yemen and Syria, where mortality rates were 19.8% and 7.2%, respectively (22). Discrepancies in mortality rates between these studies may reflect variations in COVID-19 severity, age distribution, and comorbidities in the included cohorts.

The current study showed a negative correlation between smoking and blood sugar level during COVID-19, with non-smokers being significantly more hyperglycemic. Although the relation between smoking and the risk of severe COVID-19 respiratory syndrome has been previously investigated (23, 24), most studies were methodologically limited. Smoking has not been shown to protect against severe COVID-19. To our knowledge, the effect of smoking on the risk of nDM in COVID-19 has not been previously investigated. This makes the current study the first of its kind to investigate the potential impact of smoking on nDM development, despite smoking being a well-known risk factor for diabetes in the general population.

A significant positive correlation was observed between hypertension and nDM development. The relationship between hypertension and diabetes has been well documented in previous studies (25). Hypertension may increase the risk of developing diabetes, particularly type 2 diabetes. Hypertension may increase the risk of developing type 2 diabetes due to shared risk factors such as obesity, insulin resistance, and an unhealthy lifestyle (26). Individuals with hypertension often exhibit insulin resistance, which can lead to elevated blood glucose and diabetes (27). COVID-19 is also known to trigger insulin resistance (28), and the presence of hypertension may further increase the risk of developing nDM in these patients.

Disease outcomes were generally more favorable among non-DMD COVID-19 patients, although the difference was not statistically significant. This is consistent with a study from Sudan, which reported a 91% recovery rate among normoglycemic patients (29). Poor prognosis in COVID-19 has been linked to diabetes, whether newly diagnosed or previously established. Patients with SARS or COVID-19 who also have hyperglycemia or diabetes are at increased risk of severe illness or death (30). Multi-organ failure in severe COVID-19 is associated with a cytokine storm, characterized by markedly elevated inflammatory cytokines (31).

The current study found that D-dimer levels were significantly higher in hyperglycemic patients compared to normoglycemic patients. This is consistent with a study from Wuhan (32), in which D-dimer levels in COVID-19 patients were higher in patients with hyperglycemia than in those with normoglycemia. Other studies have also documented elevated D-dimer levels in patients with both diabetes and COVID-19 (33–36). Several reports indicate that higher COVID-19 severity is associated with higher D-dimer levels (32–35), and severe disease is more common among hyperglycemic and diabetic patients (29–31, 33–35).

Lymphocyte count has shown a significant negative association with nDM development. Previous studies have shown that lymphopenia is associated with severe COVID-19 infection (37, 38) and can serve as a useful predictor of disease severity (39).

Elevated serum urea and low platelet counts were significant predictors of poor disease outcomes, likely reflecting multi-organ failure characteristic of severe COVID-19.

Interpretation of our findings regarding new-onset hyperglycemia should consider pharmacological management. In our cohort, all patients received glucocorticoid therapy, a standard treatment for mitigating inflammatory lung injury in moderate-to-critical COVID-19. Glucocorticoids are known to induce hyperglycemia by promoting hepatic gluconeogenesis and peripheral insulin resistance. Therefore, distinguishing hyperglycemia caused by direct pancreatic β-cell injury from steroid-induced effects is challenging. The observed hyperglycemia likely reflects a combination of steroid-induced insulin resistance and subclinical β-cell dysfunction triggered by SARS-CoV-2. Future studies incorporating control groups not receiving steroids, or closely monitoring steroid dosing, are needed to clarify causation. Furthermore, investigation into the pharmacological management of hyperglycemia, such as insulin therapy, and its impact on patient outcomes, remains an important area for future research.

In conclusion, the present study demonstrates that COVID-19 itself is a risk factor for hyperglycemia and new-onset diabetes mellitus (nDM), especially in patients with severe disease. Clinical and laboratory predictors of severe COVID-19—such as hypertension, elevated D-dimer, lymphopenia, and low platelet count—also appear to contribute to the development of hyperglycemia and/or nDM. Although smoking was associated with a lower risk of nDM in this cohort, this observation requires further investigation.

Study limitations

As the follow-up required a lot of time, we were unable to use prospective studies. There were no patients who did not get steroids for comparison. It was impossible to determine whether the patient had diabetes prior to COVID-19 using a blood test such as the HbA1c.

As a result of the coronavirus pandemic and the loss of patient files' personal information, laboratory results, and follow-up reports, some patient files were missing, which had an impact on the sample size.

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Authors' Contributions

Conceptualization, methodology, investigation, formal analysis, writing – original draft, review and editing: S.M.Y. and Z.A. Both authors have read and approved the published version of the manuscript.

Statement of Ethics

The study was reviewed and approved by the Ethics Committee of Medical Research at Al-Kindy College of Medicine, Baghdad University, approval number 3676, issued on June 14, 2021.

Statement of Competing Interest

The authors declare no relevant conflicts of interest.

Statement of Data Availability

Not applicable.

Statement of Generative AI Technologies Use

No generative AI was used.

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