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Original Research Article

Study of new onset hyperglycemia and insulin resistance in COVID-19 patients

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ABSTRACT

Background: COVID-19 has been associated with new onset hyperglycemia and insulin resistance, even in patients without pre-existing diabetes. This study aimed to evaluate the prevalence and characteristics of new onset hyperglycemia and insulin resistance in COVID-19 patients.
Methods: This case-control study included 90 COVID-19 patients admitted to Era's Lucknow Medical College and Hospital. Patients were divided into a case group with new onset hyperglycemia (n=57) and a control group with normoglycemia (n=33). Fasting blood glucose, post-prandial glucose, HbA1c, fasting insulin, and HOMA-IR were measured. Disease severity and outcomes were also assessed.
Results: The prevalence of new onset hyperglycemia was 61.1% among COVID-19 patients. The case group had significantly higher mean fasting glucose (132.11 versus 90.43 mg/dl, p<0.001), post-prandial glucose (212.53 versus 157.91 mg/dl, p<0.001), HbA1c (5.86% versus 4.03%, p<0.001), fasting insulin (9.24 versus 7.67 µU/ml, p=0.004), and HOMA-IR (2.99 versus 1.72, p<0.001) compared to controls. Insulin resistance was present in 71.9% of cases versus 0% of controls (p<0.001). There was no significant difference in disease severity or mortality between groups.
Conclusions: New onset hyperglycemia and insulin resistance are common in COVID-19 patients, even without pre-existing diabetes. This metabolic dysregulation may contribute to worse outcomes and requires further study. Glucose monitoring and management should be considered in all hospitalized COVID-19 patients.

Keywords: COVID-19, Glucose metabolism, HOMA-IR, Insulin resistance, New onset hyperglycemia, SARS-CoV-2

INTRODUCTION

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), responsible for the coronavirus disease 2019 (COVID-19), was first identified in December 2019 in Wuhan, China. Since then, it has led to a global health crisis, resulting in over two million deaths worldwide and placing unprecedented strain on healthcare systems.[1] Although COVID-19 primarily affects the respiratory system, its systemic nature has become increasingly evident, especially in patients with underlying comorbidities.

Chronic conditions such as hypertension, cardiovascular disease, obesity, chronic kidney disease, and diabetes mellitus are frequently observed among individuals hospitalized with COVID-19.[2] While these comorbidities do not necessarily increase susceptibility to SARS-CoV-2 infection, they are consistently associated with more severe disease progression, higher rates of complications, and increased mortality.[2,3]

One of the emerging metabolic complications observed during the COVID-19 pandemic is new-onset hyperglycemia, which has been reported in patients both with and without a prior history of diabetes. Severe hyperglycemia is a common finding in critically ill patients and is recognized as a marker of systemic stress and disease severity.[4] However, SARS-CoV-2 infection appears to induce a unique glycometabolic disturbance. Several observational studies have highlighted the occurrence of new-onset diabetes or dysglycemia among hospitalized COVID-19 patients. For instance, a cohort study from Wuhan reported that among elderly inpatients, 20.8% were newly diagnosed with diabetes based on fasting glucose ≥7.0 mmol/l and/or HbA1c≥6.5%, while 28.4% exhibited intermediate hyperglycemia or dysglycemia.[5]

The mechanisms underlying this phenomenon are still being elucidated. Possible explanations include direct viral invasion of pancreatic β-cells via ACE2 receptors, heightened inflammatory cytokine responses leading to insulin resistance, and stress-induced hyperglycemia due to counter-regulatory hormone release.[6,7] Moreover, new-onset hyperglycemia has been associated with increased risk of complications, longer hospital stays, and higher mortality, even in individuals without previously diagnosed diabetes.[8]

Given these findings, it is critical to understand the glycemic alterations associated with COVID-19. This study aimed to investigate the prevalence of new-onset hyperglycemia and insulin resistance in COVID-19 patients, comparing glycemic markers, insulin levels, and HOMA-IR between normoglycemic and hyperglycemic individuals. Understanding these metabolic changes may aid in better risk stratification and clinical management of affected patients.

METHODS

Study design and participants

This case-control study was conducted at Era's Lucknow Medical College and Hospital, Lucknow, Uttar Pradesh, over a period of 24 months, from February 2021 to February 2023. A total of 90 adult patients who tested positive for COVID-19 by RT-PCR were enrolled after obtaining written informed consent. The study was approved by the institutional ethics committee.

Patients with a known history of diabetes mellitus, pregnancy, polycystic ovary syndrome (PCOS), or recent use of medications known to cause hyperglycemia (such as corticosteroids, thiazide diuretics, or antipsychotics) were excluded from the study to avoid confounding factors. All included patients had no prior diagnosis of impaired glucose metabolism.

The study population was divided into two groups. The case group consisted of 57 COVID-19 patients who developed new-onset hyperglycemia during hospitalization, whereas the control group comprised 33 COVID-19 patients who maintained normoglycemia throughout their hospital stay.

Data collection and laboratory measurements

A comprehensive clinical evaluation and history-taking were performed for each participant at the time of admission. Demographic data, vital signs, and comorbidities were recorded. Laboratory investigations included complete blood count (CBC), kidney function tests (KFT), liver function tests (LFT), and glycemic parameters such as fasting blood glucose, two-hour post-prandial blood glucose, and glycated hemoglobin (HbA1c).

Fasting serum insulin levels were measured to assess insulin sensitivity. Insulin resistance was evaluated using the homeostatic model assessment of insulin resistance (HOMA-IR), calculated with the formula:

HOMA-IR = (fasting glucose [mg/dl] × fasting insulin [μU/ml]) / 405

A HOMA-IR value greater than 2 was considered indicative of insulin resistance.

Inflammatory markers including interleukin-6 (IL-6), ferritin, D-dimer, and C-reactive protein (CRP) were also assessed to evaluate systemic inflammation. A chest x-ray was performed for radiological assessment of COVID-19 severity.

Classification of disease severity and outcomes

The severity of COVID-19 was categorized as mild, moderate, or severe based on clinical symptoms, oxygen requirement, and chest imaging findings in accordance with standard treatment guidelines. Patient outcomes including recovery, need for intensive care, and mortality were recorded.

Statistical analysis

All data were analyzed using SPSS version 21.0. Continuous variables were expressed as mean±standard deviation (SD) and compared between groups using the unpaired t-test. Categorical variables were presented as frequencies and percentages, and compared using the chi-square test. Pearson's correlation coefficient was used to evaluate the correlation between viral load [as inferred from cycle threshold (CT) values] and metabolic parameters. A p value of less than 0.05 was considered statistically significant.

RESULTS

The mean age of patients in the case group was 58.91±7.26 years, slightly higher than the control group,

which had a mean age of 57.06±7.81 years. However, this difference was not statistically significant (p=0.260). In terms of gender distribution, males constituted 61.4% of the case group and 45.5% of the control group, but again, this difference was not statistically significant (p=0.142). A separate unpaired t-test analysis of age indicated no significant difference between the groups (mean age 58.75±7.33 for cases versus 57.43±7.74 for controls; t=-0.81, p=0.418).

Table 1: Baseline characteristics of study participants.

Variable	Age in years		Unpaired t test	
	Mean	SD	t-value	P value
Control group	57.43	7.74		
Case group	58.75	7.33	-0.81	0.418
Overall	58.23	7.47		

Among the 90 COVID-19 patients studied, 55 patients (61.1%) were identified as having new-onset hyperglycemia, while 35 patients (38.9%) were normoglycemic, thus falling into the control group. This indicates that more than half of the COVID-19 patients developed hyperglycemia during the course of illness.

Table 2: Prevalence of new-onset hyperglycemia among COVID-19 patients.

Group	No.	%
Normoglycemic (control)	35	38.9
New onset hyperglycemia (case)	55	61.1
Total	90	100.0

The case group exhibited significantly elevated glycemic indices compared to the control group. Mean fasting blood glucose (FBS) in the case group was 132.11±28.08 mg/dl, markedly higher than the control group's 90.43±3.78 mg/dl (p<0.001). Post-prandial sugar (PPS) was also elevated in cases (212.53±46.87 mg/dl) compared to controls (157.91±13.24 mg/dl), with a statistically significant difference (p<0.001). Similarly, HbA1c levels were considerably higher in the case group (5.86±0.35%) versus controls (4.03±0.78%) with a very strong significance (p<0.001). Insulin resistance markers were also raised, as seen in higher fasting insulin levels in cases (9.24±2.73 µU/ml versus 7.67±1.82 µU/ml; p=0.004) and HOMA-IR scores (2.99±1.00 versus 1.72±0.54; p<0.001).

Table 3: Comparison of glycemic parameters between case and control groups.

Variables	Control		Case		Unpaired t test	
	Mean	SD	Mean	SD	t-value	P value
FBS	90.43	3.78	132.11	28.08	-8.71	<0.001
PPS	157.91	13.24	212.53	46.87	-6.71	<0.001
HbA1c	4.03	0.78	5.86	0.35	-15.05	<0.001
Homa IR	1.72	0.54	2.99	1.00	-6.90	<0.001

Table 4: Prevalence of insulin resistance (HOMA-IR >2) in study groups.

Insulin resistance	Control		Case		Total		Chi sq.	P value
	No.	%	No.	%	No.	%		
Absent	33	94.3	16	29.1	49	54.4		
Present	2	5.7	39	70.9	41	45.6	36.65	<0.001
Total	35	100.0	55	100.0	90	100.0		

Table 5: Severity of COVID-19 illness among case and control groups.

Severity	Control		Case		Total		Chi sq.	P value
	No.	%	No.	%	No.	%		
Severe	6	17.1	13	23.6	19	21.1		
Moderate	21	60.0	32	58.2	53	58.9	0.67	0.714
Mild	8	22.9	10	18.2	18	20.0		
Total	35	100.0	55	100.0	90	100.0		

A significantly higher prevalence of insulin resistance (defined as HOMA-IR>2) was found in the case group, where 71.9% (41 out of 57) patients were insulin resistant. In stark contrast, only 2 patients (5.7%) in the control group met the criteria for insulin resistance. The chi-square analysis confirmed this difference to be highly significant (χ^2=36.65, p<0.001), emphasizing the strong association between COVID-19-induced hyperglycemia and insulin resistance.

There was no statistically significant difference in the severity of disease between the case and control groups. Severe disease was observed in 23.6% of cases and 17.1% of controls (p=0.714), while the majority of patients in both groups had moderate disease (58.2% in cases versus 60.0% in controls). Mild cases accounted for 18.2% and 22.9% in the case and control groups, respectively. Regarding clinical outcomes, the mortality rate was higher among cases (36.4%) than controls (25.7%), but this difference was not statistically significant (p=0.292). Overall recovery rates were slightly better in controls (74.3%) compared to cases (63.6%).

Table 6: Clinical outcomes (recovery versus mortality) among case and control groups.

Outcome	Control		Case		Total		Chi sq.	P value
	No.	%	No.	%	No.	%		
Recovered	26	74.3	35	63.6	61	67.8		
Death	9	25.7	20	36.4	29	32.2	1.11	0.292
Total	35	100.0	55	100.0	90	100.0		

Table 7: Correlation between CT values of the E gene and various glycemic markers.

CT value on arrival	Control		Case		Unpaired t test	
	Mean	SD	Mean	SD	t-value	P value
E gene	25.43	2.80	24.19	3.10	1.92	0.058
N gene	25.92	3.05	24.98	2.83	1.50	0.138
S gene			28.00			

Table 6 compares the clinical outcomes of COVID-19 patients with new-onset hyperglycemia (case group) and normoglycemic patients (control group). In the control group, 74.3% (26 out of 35) recovered and 25.7% (9 out of 35) died, whereas in the case group, 63.6% (35 out of 55) recovered and 36.4% (20 out of 55) died. Although a higher mortality rate was observed in the hyperglycemic group, the difference was not statistically significant (chi-square =1.11, p=0.292), indicating that the presence of new-onset hyperglycemia was not conclusively associated with worse outcomes in terms of mortality in this cohort.

A significant negative correlation was found between CT values of the E gene and various glycemic markers. Specifically, lower CT values (indicating higher viral loads) were associated with elevated fasting glucose (r=-0.36, p<0.001), post-prandial glucose (r=-0.314, p=0.003), HbA1c (r=-0.283, p=0.007), and HOMA-IR (r=-0.297, p=0.004), suggesting that more severe viral replication may be linked with worsened glycemic control and insulin resistance. On arrival, mean CT values of the E gene were slightly lower in the case group (24.19±3.10) than in controls (25.43±2.80), approaching statistical significance (p=0.058). However, CT values for the N gene and S gene did not show statistically significant differences between the two groups

DISCUSSION

In this study, new-onset hyperglycemia was observed in 61.1% of COVID-19 patients, indicating a substantial prevalence of glycemic dysregulation during SARS-CoV-2 infection. Despite no significant difference in baseline age (case: 58.75±7.33 years, control: 57.43±7.74 years; p=0.418) or gender distribution (p=0.142), patients with new-onset hyperglycemia demonstrated significantly altered glycemic profiles and metabolic parameters compared to normoglycemic controls.

The hyperglycemic (case) group exhibited markedly higher fasting blood sugar (FBS) levels (132.11±28.08 versus 90.43±3.78 mg/dl), post-prandial sugar (PPS) (212.53±46.87 versus 157.91±13.24 mg/dl), and HbA1c (5.86±0.35% versus 4.03±0.78%), all with p<0.001. These findings are consistent with previous studies that have reported elevated glucose levels in hospitalized COVID-19 patients, even in those without prior diabetes.[9-12] For example, a large cohort from Wuhan reported that nearly 21% of hospitalized patients developed new-onset diabetes or dysglycemia during the course of illness.[13]

Furthermore, insulin resistance, assessed using HOMA-IR, was significantly more prevalent in the case group. The mean HOMA-IR value was nearly double in cases compared to controls (2.99±1.00 versus 1.72±0.54; p<0.001), and 71.9% of hyperglycemic patients exhibited insulin resistance compared to only 5.7% in the control group (p<0.001). These findings align with Montefusco et al who observed hyperinsulinemia in COVID-19 patients, suggesting that insulin resistance, rather than insulin deficiency, may underlie hyperglycemia in this population.[14] Supporting this, studies have reported elevated C-peptide levels in COVID-19-associated ARDS patients compared to non-COVID ARDS cases, reinforcing the notion of preserved insulin secretion amidst peripheral resistance.[15]

Our findings also contrast with earlier hypotheses that attributed COVID-19-induced hyperglycemia to pancreatic damage and insulin deficiency, based on elevated pancreatic enzymes and autopsy evidence of pancreatic necrosis.[16,17] However, the evidence of sustained insulin levels and the prevalence of insulin resistance in our cohort further supports the idea that COVID-19 primarily disrupts peripheral insulin signaling rather than insulin production, consistent with newer mechanistic insights.

Interestingly, CT values of the E gene, which inversely correlate with viral load, showed a significant negative correlation with key glycemic parameters: fasting glucose ($r=-0.36$, $p<0.001$), post-prandial glucose ($r=-0.314$, $p=0.003$), HbA1c ($r=-0.283$, $p=0.007$), and HOMA-IR ($r=-0.297$, $p=0.004$). This suggests that higher viral loads may exacerbate metabolic dysfunction, possibly through mechanisms such as inflammation-mediated insulin resistance, as suggested in other studies exploring the role of inflammatory mediators like MPO, apelin, and myostatin.[18-23]

Recent mechanistic studies have also revealed that SARS-CoV-2 may transcriptionally regulate metabolic factors via upregulation of the transcription factor REST, which modulates MPO, apelin, and myostatin expression-proteins involved in glucose and lipid metabolism. For instance, MPO has been shown to worsen insulin resistance, while apelin administration improves it, and myostatin exerts tissue-specific effects.[18-23] Additionally, alterations in lipid metabolites such as (±)5-HETE, (±)12-HETE, propionic acid, and isobutyric acid have been proposed as biomarkers of COVID-19-induced metabolic dysregulation, with both favorable and detrimental correlations to insulin sensitivity.[25-27]

However, in our study, disease severity and clinical outcomes did not differ significantly between groups. Severe cases were slightly more frequent in the hyperglycemic group (23.6%) compared to controls (17.1%; $p=0.714$), and mortality was higher among cases (36.4%) than controls (25.7%), though this difference also failed to reach statistical significance ($p=0.292$). While our findings do not show a direct link between hyperglycemia and short-term mortality, the metabolic complications may still have important long-term implications, as suggested by studies indicating the persistence of insulin resistance even after viral clearance.[14]

In summary, our findings underscore a strong association between COVID-19 and new-onset hyperglycemia with significant insulin resistance, even in individuals without pre-existing metabolic disease. These results are consistent with and build upon prior clinical and mechanistic studies. Although these changes did not significantly influence short-term outcomes in our cohort, they may have long-lasting metabolic consequences, highlighting the need for close glycemic monitoring and long-term follow-up in COVID-19 survivors.

CONCLUSION

New onset hyperglycemia and insulin resistance are common metabolic derangements in COVID-19, even in patients without pre-existing diabetes. This metabolic dysregulation may contribute to adverse outcomes and warrants close monitoring. Routine glucose screening and appropriate management should be considered for all hospitalized COVID-19 patients. Further research is needed to elucidate the underlying mechanisms and long-term implications of COVID-19-associated hyperglycemia.

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