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RESEARCH ARTICLE

The COVID-19 and Dysglycemia Connection: Unveiling the Truth

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Abstract

Background: It is well-established that COVID-19 is more prevalent among individuals with preexisting diabetes and can lead to worse outcomes. However, the question of whether COVID-19 contributes to the development of newly detected dysglycemia, including prediabetes and diabetes, remains uncertain.

Objectives: To see the frequency and association of newly detected dysglycemia in COVID-19 infection.

Materials and methods: This cross-sectional study was conducted in the Department of Endocrinology at BSMMU, spanned from March 2021 to September 2023. The research enrolled 177 participants, including 88 confirmed post COVID-19 patients and 89 individuals from a non-COVID-19 control group. Comprehensive sociodemographic, clinical, and laboratory data were collected, with a particular focus on oral glucose tolerance tests (OGTT) and HbA1c measurements.

Results: The analysis revealed that there was no significant difference in the prevalence of newly detected dysglycemia between the two groups (COVID-19 vs Control: 35.2% vs 31.5%, P= 0.353).A statistically significant association was observed between the severity of COVID-19 and the development of newly detected

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dysglycemia (OR 3.68, 95% CI 1.03-14.46, P= .04). Age (38.25 ± 9.38 vs 32.5 ± 8.77, P=0.005), oxygen therapy (16.1% vs 1.8%, P=0.019), and steroid therapy (16.1% vs 3.5%, P= 0.037) also showed significant associations with newly detected dysglycemia. However, they didn't show any significance after adjusting withlogistic regression. Only age remained an independent predictor of newly detected dysglycemia (OR 1.074, 95% CI 1.008-1.145, P=0.027). Conclusions: This study observed there was no significant association of developing new dysglycemia in COVID-19 infected patient. Older age is more vulnerable to develop new dysglycemia in COVID-19 infected patient.

Keywords: COVID-19, Dysglycemia, HbA1c.

1. Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), responsible for the global pandemic of coronavirus disease 2019 (COVID-19), has left an unforgettable mark on the world. As of May 2, 2023, the staggering figures stand at approximately 687 million confirmed global cases of COVID-19 and a mournful count of around 6.87 million lives lost [1]. This relentless virus has disproportionately impacted the elderly, particularly those with underlying health conditions such as diabetes mellitus (DM). A complex interplay has emerged between type 2 diabetes mellitus (T2DM) and COVID-19, revealing a bidirectional relationship. On one front, individuals with diabetes face an elevated risk of severe COVID-19 outcomes, contributing to increased mortality and morbidity rates [2]. On the other front, there is chance of development of new dysglycemia and diabetic complications in COVID-19 patient Notably, diabetes, along with cerebrovascular disease, has emerged as one of the most prevalent comorbidities, comprising 22% of COVID-19 cases [3]. When analyzed individually, studies have reported hypertension (24.7%), diabetes (21.2%), and coronary heart disease (8%) as the most frequent comorbidities among COVID-19 patients [4]. Alarming statistics from England reveal that 19% of those admitted to intensive care units with COVID-19 had diabetes, and one-third of them did not survive their hospitalization [5]. In the United Kingdom, individuals with diabetes face a 50% higher risk of serious complications and death from COVID-19 compared to non-diabetic individuals [6]. While there is ample evidence demonstrating the adverse impact of COVID-19 on preexisting diabetes patients, a notable gap in knowledge pertains to the incidence of new- onset diabetes following COVID-19 infection. A study by [7] conducted over a 6-month cohort study period in Italy reported a surprisingly low relationship between COVID-19 and fasting dysglycemia (3.31% in COVID cases vs. 2.91% in non- COVID cases) [7]. Similarly, a study in Bangladesh by Akter et al. incidentally discovered that 1.34% of post-COVID patients had developed new diabetes, although detailed findings were not provided in their published article [8]. The hypothesis of COVID-19 infection-related diabetes gains traction as studies point to the existence of virus-associated β-cell destruction [9]. SARS-CoV-2 exhibits the capability to infect both exocrine and endocrine pancreas cells, both in vitro and in vivo, owing to the expression of the viral entry receptor, angiotensin-converting enzyme 2 (ACE2), in human pancreatic β-cells and the microvasculature of the pancreas [10]. Yet, it remains unclear whether SARS-CoV-2's impact on the pancreas directly influences glucose homeostasis or serves as a trigger for diabetes mellitus [11]. This research seeks to bridge these knowledge gaps by investigating the association between SARS-CoV-2 infection and the emergence of newly detected dysglycemia, specifically focusing on individuals with no prior history of diabetes. Furthermore, we aim to identify any potential risk factors linked to the incidence of newly detected glucose abnormalities in COVID-19 patients. In doing so, we aim to deepen our understanding of how COVID-19 affects glucose metabolism and its clinical implication in the context of this unprecedented global health crisis.

2. Materials and Methods

2.1 Study Design

Cross sectional observational study.

2.2 Place of Study

Department of Endocrinology, Bangabandhu Sheikh Mujib Medical University.

2.3 Period of Study

Initiated in March'2021, Started activity in January'2022 and Completed by September'2023.

2.4 Sample Size

Total study participants were 177 of which 88 had COVID-19 and another 89 had a non COVID-19 control group.

2.5 Inclusion Criteria

- All confirmed COVID-19 patients who were aged ≥ 18 years and diagnosed within six months period following infection.

2.6 Exclusion Criteria

- Known cases of prediabetes and diabetes mellitus before the COVID-19 infection
- Pregnancy and lactating mother.

2.7 Sample Selection Technique

Patients who fulfilled the inclusion criteria and, in the absence of exclusion criteria, were included in the study as a COVID-19 group. Subjects had been collected from the post COVID-19 OPD at Bangabandhu Sheikh Mujib Medical University (BSMMU). The control group had taken from all non-diabetic participants who were ≥18 years old and who had no history of confirmed and had come for other problems to non-post COVID-19 OPD. Patients were recruited after a complete explanation of the steps and purpose of the study and informed written consent from the patient himself and/or a responsible family member. Recruitment of study subjects had been continued until the desired number of post- COVID-19 participants and controls were enlisted in this study. Data had been collected in the questionnaire after the completion of a history and physical examination, including measurements of height, weight, blood pressure, and BMI. Then samples were collected for the oral glucose tolerance test (OGTT) and HbA1c. The assay for measurement of OGTT and HbA1c was done in the laboratory of the Biochemistry Department at BSMMU.

2.8 Comparator Group

Equal numbers of age and sex matched non diabetic asymptomatic participants who had no clinical history suggestive of COVID-19 and no documentation of confirmed or provable COVID-19.

2.9 Statistical Methods

All data were processed using SPSS version 25.0. Data were expressed as frequencies or percentages for qualitative values and mean (±SD) for quantitative values with normal distribution (waist circumference, biochemical values). Assessment of the normality of the data was done by the Shapiro-Wilk test. Quantitative values with a skewed distribution were presented as the median and interquartile range (25th and 75th percentiles) (age, BMI, blood pressure, etc.). Between the COVID-19 group and the control group, subgroups between dyglycemia and NGT in post-COVID-19 participants were compared by a chi-square test (for qualitative) and an unpaired t-test (for quantitative data) for normally distributed variables as applicable. For continuous variables with a skewed distribution, the Mann-Whitney U test was used to estimate differences between the groups. To assess the association among COVID-related and diabetes related risk factors with dysglycemia, binary logistic regression was applied. All tests were two sided and statistical significance was set at a P-value of ≤0.05.

3. Results

Total study participants were 177 of which 88 had COVID-19 and another 89 had a non COVID-19 control group. All the sociodemographic variables were statistically similar between the groups. The majority were of middle age (30 to 50 years) and male. And most of them were nonsmokers, service holders, vaccinated and from an urban area (Table-1). Clinical and laboratory parameters were also similar in both groups. The median BMI in the COVID-19 group was 24.25(22.62, 27.35) Kg/m2 and in the control group it was 23.5(21.4, 26.0) Kg/m2 and there was no significant difference (p=0.171). In both group most of them were overweight (68.2% vs. 60.7%). Other clinical variables like blood pressure, family history of diabetes, and hypertension had no significant difference. Their liver function, renal function and hemoglobin level were normal and similar in both groups (Table-2).

Table 1. *Sociodemographic characteristics of the participants (N=177)*

Variables	COVID-19 group(n=88)	Control group(n=89)	P
Age (Years)*	34.53 ± 9.34	33.55 ± 10.23	0.158†
Age Group			
<30 Years	30 (34.1)	42 (47.2)	0.199‡
30-50 Years	47 (53.4)	37 (41.6)	
>50 years	11 (12.5)	10 (11.2)	
Gender			
Male	58 (65.9)	55(61.80)	0.561‡
Residence			

Urban	78 (88.6)	77 (86.52)	0.669‡
Rural	10 (11.4)	12 (13.48)	
Smoking			
Smoker	13 (14.8)	17 (19.10)	0.092‡
Non-smoker	57 (64.8)	64 (71.91)	
Ex-smoker	18 (20.5)	8(8.99)	
Profession			
Service holder	55 (62.5)	53(59.55)	0.196‡
Business	12 (13.6)	15 (16.85)	
Day laborer Student	4 (4.5)	3 (3.37)	
Others	4 (4.5)	7(7.87)	
Business	13 (14.7)	11 (12.36)	
COVID-19 Vaccinated	78 (88.6)	84(94.4)	0.234†

(Within parenthesis values are percentages over column total)

**Mean ± SD*

†calculated using independent sample t-test

‡calculated using Chi-square test

Table 2. *Clinical and laboratory characteristics of participants (N=177)*

Variables	COVID-19 group(n=88)	Control group (n=89)	P
BMI(Kg/m2)*	24.25 (22.62,27.35)	23.5 (21.4,26.0)	0.171†
BMI Category			
Underweight	4 (4.5)	7 (7.9)	0.488‡
Normal	24 (27.3)	28 (31.5)	
Overweight/ obese	60 (68.2)	54 (60.7)	
SBP(mm of Hg)*	120 (110,130)	120(110,120)	0.207†
DBP(mm of Hg)*	80(70,82)	80(70,80)	0.211†
F. History DM			
Present	24(27.3)	30 (33.7)	0.353‡
F. History HTN			
Present	25 (28.4)	26 (29.2)	0.960‡
Comorbidity			
Present	30 (34.1)	24 (27)	0.231‡
SGPT(IU/L)*	30.0(18,46.75)	26.0(1.5,44.0)	0.210 †
Creatinine(mg/l)§	0.833± 0.18	0.825 ±0.2	0.541‖
Hb%(gm/dl)§	13.60 ± 1.63	13.56 ± 1.72	0.647‖

(Within parenthesis vslues are percentages over column total)

** Median (interquartile range)*

† values were calculated using Mann-Whitney U test

‡ values were calculated using Chi-square test

§ Mean ± SD and

Pvalues were calculated using Independent T- test

BMI=Body Mass Index

SBP = Systolic blood pressure DBP = Diastolic blood pressure

F. History = Family History

SGPT = Serum glutamic pyruvic transaminase Hb% = Hemoglobin(gm/dl)

3.1 Frequency and Level of Dysglycemia in Covid-19 Group

Among the 88 participants in the post COVID-19 group, 35.2% had newly detected dysglycemia, whereas in the control group 31.5% had dysglycemia. There was no significant difference between the two groups (p = 0.595) (Figure 1). 26.1% (23 out of 88) had prediabetes 9.1% (8 out of 88) had DM, and there was no significant difference from the control group (p =0.886] (Figure-2). There was no significant differenece among their plasma glucose level in dysglymic participant between post COVID-19 and control group (Table-3).

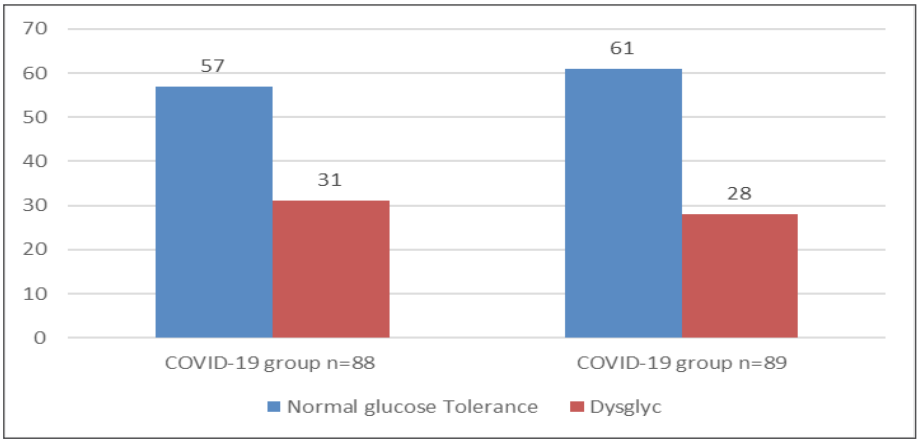

Figure 1. *Frequency of dysglycemia in COVID-19 vs control group. p-values were calculated using Chi-square test*

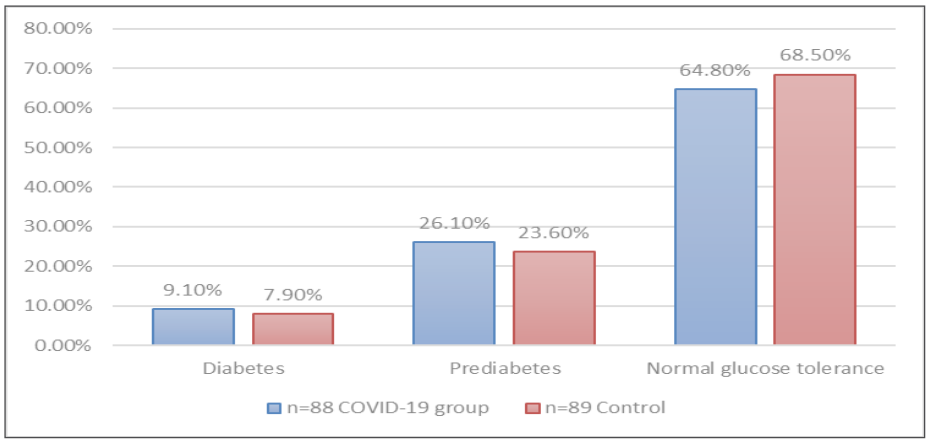

Figure 2. *Different type of glycemia in COVID-19 and control group. P values were calculated using Chi-square test*

Table 3. *Plasma glucose level of dysglecemic participants in the post COVID-19 group vs control group.*

Variables	COVID-19 group (n=31)	Control group (n=28)	P
FBS (mmol/l)*	5.7(5.3,6.3)	5.3(5.1,6.1)	0.59†
2Hours after 75gm glucose(mmol/l)*	8.5(6.6,10.8)	8.3(6.9,10.8)	0.47†
HbA1c(%)*	5.9(5.7,6.6)	5.9(5.6,6.35)	0.66†

**Median (interquartile range)*

† Values were calculated using Mann-Whitney U test

3.2 Association of Severity of Covid-19 and Dysglycemia in Covid-19 Group

12.5% (11) had severe COVID-19 among the 88 participants in the COVID-19 group. The majority (63.63%) of severe COVID-19 patients had dysglycemia (7 out of 11). Dysglycemia was associated with the severity of the COVID infection and those with severe COVID-19 had a 3.68 times greater risk of developing dysglycemia than mild-to- moderately affected patients. (95 CI,1.03-14.88) (Table-4). In the severe COVID-19 group 7 participants had a high fever, 9 had breathlessness, 6 got oxygen therapy, 4 had more than 3 symptoms, 6 had long COVID syndrome and 2 got steriod therapy (Figure 6). Their average BMI, FBS, 2 hours after 75gm glucose, HbA1 were 24.53±2.49, 5.6 mmol/l, 8.33 mmol/l, 5.8% respectively.

Table 4. *Types of dysglecemia in different severity of COVID-19 infection (N=88)*

	Dysglycemia(n=31)	NGT (n=57)	Total (N=88)	OR	95% CI LowerUpeer		P
Severity ofCOVI-19	24 (31.17)	53(68.83)	77(100)	3.68	1.033	14.46	0.04*
Mild to Moderate Severe	7 (63.63)	4(36.37)	11(100)				

(Within parenthesis values are percentages over colum total)

Values were calculated using Chi-square test

OR = Odd Ratio 95% CI = 95% confidence interval NGT = Normal Glucose Tolerance

3.3 Association Between Risk Factors of Diabetes and Dysglycemia in Covid-19 Group

Among participants with COVID 19, those with dysglycemia were significantly older than those with NGT (38.25 ± 9.38 vs 32.5 ± 8.77, P=0.005). And also dysglycemic participants in the COVID-19 group had higher rates of steroid use (P=0.037) and oxygen use (P=0.019). All the other risk factors such as BMI, family history of diabetes, comorbidity, type & number of symptoms, hospitalization, and high flow nasal canula use were similar between the two groups. (Table-5 and Table-6).

Table 5. *Sociodemographic characteristics of COVID-19 group. (N= 88)*

Variable	Dysglycemia(n=31)	NGT (n=57)	P
Time length from diagnosis to testing(days)*	155 (56,177)	133 (56,153)	0.665†
Gender			
Male	19 (61.3)	39 (68.4)	0.50‡
Female	12 (38.7)	18 (31.6)	
Age (years)§	38.25 ± 9.38	32.5 ± 8.77	0.005‖
BMI*	24.3 (22.9,27.7)	24.1 (21.8,26.75)	0.189†
F.H. HTN	6 (19.4)	16 (28.1%)	0.165‡
F.H.DM	8 (25.8)	19 (33.3)	0.820‡
Residence			
Urban	26 (83.9)	52 (91.2)	0.299‡
Rural	5 (16.1)	5 (8.8)	
Profession			
Service	21 (47.7)	34 (59.6)	0.172‡
Business	2 (6.5)	10 (17.5)	
Day labourer	1 (3.2)	3 (5.3)	
Student	0 (00)	4 (7)	
Others	7 (22.6)	5 (10.6)	
Smoker	6 (19.4)	7 (12.3)	0.572‡
Comorbidity	13 (41.9)	17 (29.8)	0.347‡

(Within parenthesis are percentages over column total)

Median (interquartile range)

Independent T- test

BMI=Body Mass Index

F. H. DM = Family history of diabetes mellitus

F. H. HTN = Family history of hypertension

SGPT = Serum glutamic pyruvic transaminase

Table 6. *Clinical and laboratory characteristics of COVID-19 group. (N= 88)*

Variable	Dysglycemia(n=31)	NGT (n=57)	P
Symptoms of COVID-19			
High Fever	12 (38.7)	21 (36.8)	0.863*
Anosmia	9 (29.0)	26 (45.6)	0.465*
Sob	8 (25.8)	19 (33.3)	0.129*
Cough	19(61.3)	38 (66.7)	0.614*

Sore throat	14 (45.2)	21(36.8)	0.446*
Total symptoms			
2symptoms	10 (32.3)	14 (24.6)	0.498*
3symptoms	12 (38.7)	20 (24.6)	
4symptoms	5 (16.1)	15 (35.1)	
5Symptoms	1 (3.2)	4 (24.3)	
Steroid	5 (16.1)	2 (3.5)	0.037*
O2 treatment	5 (16.1)	1(1.8)	0.019*
HFNC	2 (6.5)	0(00)	0.121*
Severe COVID-19/ Hospitalize	7 (22.6)	4 (7)	0.172*
Long COVID-19	13 (41.9)	23 (40.4)	0.839*
SGPT†	39 (23,53)	34.1±26.69	0.072‡
Creatinine§	0.83±0.21	0.84(0.70,0.95) †	0.662‡
Hb%#§	13.68 ± 1.45	13.56 ± 1.72	0.889‖

(Within parenthesis values are percentages over column total)

HFNC= High flow nasal canula Hb% = Hemoglobin(gm/dl)

3.4 Binary Logistic Regression Model Predicting Dysglycemia in Covid-19

To assess the impact of several factors on development of new dysglycemia after COVID- 19 infection, binary logistic model (enter method) was used. The model contained ten independent variables - age, BMI, comorbidity , family history of DM, severity of COVID- 19 infection, hospitalization history, oxygen and steroid treatment , symptoms (fever severity , anosmia) . The model containing all predictors was statistically significant, $\chi 2(10, N = 88) = 65.1, p < .006$, indicating that the model was able to distinguish between respondents who had dysglycemia and those who did not. The model as a whole explained between 19.1% (Cox and Snell R square) and 27.3% (Nagelkerke R squared) of the variance in glycemic status after COVID-19 infection and correctly classified 75 % of cases. Only age was an independent predictor of dysglyacaemia in post COVID 19 patients, after adjusting for all the risk factors. A unit (1 year) increase of age increased the risk of developing dysglycaemia by 7.4% (95CI, 00%- 14.5%) (Table-7).

Table 7. *Binary logistic regression model predicting dysglycemia in post COVID-19 (N =88)*

Variables	P*	OR	95% CI. Lower	for OR Upper
Age	0.027	1.074	1.008	1.145
BMI	0.870	0.996	0.948	1.046
Oxygen therapy	0.131	16.705	0.433	645.167
Steroid therapy	0.650	0.475	0.019	11.875
F. history of DM	0.743	1.213	0.382	3.849
Long COVID-19 syndrome	0.946	1.040	0.334	3.238
Severe COVID-19	0.786	1.389	0.130	14.856
Comorbidity	0.294	1.811	0.597	5.493
High fever	0.782	1.178	0.370	3.754
Anosmia	0.095	0.370	0.115	1.191

** P- values were calculated using wald type test*

95% CI= 95% confidence interval

F. history= Family history OR=Odd ratio

HFNC= High flow nasal canula Hb% = Hemoglobin(gm/dl)

Model Summary:

$\chi 2(10, N = 88) = 65.1, p < .006$

Omnubus test, P= 0.04

Hosmer and Lemshgow test X2=0.422 Varience (Cox & snell= 19, Nagelkare=27%) Over all Classable =75%

4. Discussion

To address the issue of an association between COVID-19 and dysglycemia in individuals both with previously unknown diabetes [12], we designed a cross-sectional study involving a total of 177 subjects, including 88 who had recovered from COVID-19 and 89 from a non-COVID-19 control group. Our findings indicate that there is no significant association between COVID-19 and the newly detected of dysglycemia within six months of infection. Our study primarily demonstrates the absence of an association between COVID-19 and the newly detected of dysglycemia. Most of our study findings align with previous research. For example, a study from the early days of the pandemic reported that six months after COVID-19 infection, only a small percentage (4.3%) of patients who had experienced severe COVID-19 pneumonia exhibited fasting glucose abnormalities [7]. However, our study differs from Molinari et al. [7] in that we assessed fasting and 2-hour post- 75g glucose levels as well as HbA1c. Similar to Molinari's study, our study observed a slightly higher incidence of dysglycemia in the COVID-19 group compared to the control group (35.2% vs. 31.5%), although this difference was not statistically significant (p = 0.595). The ADA guidelines in 2023 also noted conflicting reports regarding newly detected diabetes, with publications from various countries. It remains unclear whether newly detected diabetes following COVID-19 is likely to be permanent or aggressive. However, many recent studies have reported an association between newly detected diabetes and COVID-19. For instance, Lu JY et al [13] found that the incidence of newly detected type-2 diabetes was higher in patients with COVID-19 compared to influenza. They concluded that the increased risk of diabetes associated with COVID- 19 is mediated through disease severity, which plays a dominant role in the development of post-acute infection sequelae. It's worth noting that their study compared COVID-19 with influenza, whereas our study compared COVID-19 patients with a control group matched for similar characteristics, and this difference in study design may account for the varying results. Furthermore, Lu JY's study included more participants who had experienced severe COVID-19 and were included after hospital discharge, whereas only a small proportion of our participants were hospitalized (12.5%). However, it's essential to recognize that even asymptomatic COVID-19 cases can be associated with newly detected diabetes [14]. Although the frequency of newly detected dysglycemia in the COVID-19 group did not significantly differ from that in the non-COVID-19 control group (35.2% vs. 31.5%), its prevalence and glycemic levels align with findings from studies conducted in other parts of the world. For example, Keerthi BY et al. [15] observed their cohort study three months after COVID-19 infection and found that 42% had newly detected prediabetes, and 10.3% had newly detected diabetes. In our study, we found 26.1% prediabetes and 9.1% diabetes. Keerthi BY et al. [15] investigated participants three to six months after infection, a time frame similar to our median of 155 days. Although our study was not a cohort study like Molinari C, et al. [7] and Keerthi BY, et al. [15] we used HbA1c to exclude previously unknown diabetes onset for at least the last three months. The non- significant difference in median HbA1c between the COVID-19 and control groups (5.5 vs. 5.5) suggests that previous onset was unlikely. Ahmed A. Metwally et al. [16] also found that 35% of participants remained hyperglycemic six months after COVID-19 recovery, while an additional 2% were diagnosed with type 2 diabetes, indicating that newly detected hyperglycemia can predispose individuals to long-term glycemic abnormalities Metwally et al. [16]. This result is consistent with our findings and the additional 1.2% (9.1% - 7.9%) prevalence is slightly lower than their additional prevalence (2%) but not statistically different from the control group. Given that we found no significant differences in terms of frequency, type, and level of dysglycemia between the post-COVID-19 and control groups, we explored the association between dysglycemia and the severity of COVID-19 infection. In our study, we found that participants with severe COVID-19 were at a significantly higher risk of developing dysglycemia (63.63% vs. 31.37%) compared to those with mild to moderate COVID-19, with a statistically significant odds ratio (OR) of 3.68 (95% CI 1.03-14.46, P = 0.04). This finding aligns with research by Yang J-K et al. [17], which reported a 53.85% prevalence of newly detected diabetes in critically ill patients. It's worth noting that Yang J-K et al. [17] excluded patients receiving glucocorticoid treatment or with a history of diabetes, myocardial infarction, heart failure, dialysis, renal transplant, or cirrhosis and patients lacking basic medical information. The higher prevalence in our study compared to Yang J-K et al. may be due to our inclusion of steroid-treated participants and those with other severe

comorbidities[17]. To investigate the association of other factors with dysglycemia, apart from the severity of COVID-19, we compared all sociodemographic and clinical variables between dysglycemic (n=31) and normal glucose tolerance (NGT) (n=57) groups among COVID- 19 participants. However, we did not find any significant differences between the dysglycemic and NGT groups, except for age (dysglycemia vs. NGT: 38.25 ± 9.38 vs. 32.5 ± 8.77, P = 0.005), steroid use, and oxygen therapy. Sixteen percent of dysglycemic participants received steroids and oxygen therapy, which significantly differed from the NGT group (P = 0.037 and P = 0.019, respectively). Though findings are significant, its merely act as predictors of dysglycema as only 6 got oxygen thrapy and 2 got steroid therapy. No significant differences were observed in other common risk factors for diabetes, such as BMI, family history of diabetes, or the type and number of symptoms. Dysglycemia was detected a median of 155 days after disease diagnosis, while normoglycemic patients were assessed earlier, although this time difference was not significant. In contrast, Dambal et al. [18] found that common risk factors associated with the occurrence of newly detected diabetes were those on high doses of steroids (P=0.0001), a family history of diabetes mellitus (DM) (P=0.001), overweight and obesity (P=0.0001), fungal infections (P=0.0001), and the need for oxygen and intensive care unit (ICU) requirement (P=0.0001) Dambal et al. [18]. Additionally, patients with increased laboratory markers of inflammation, such as ferritin, neutrophil leukocyte ratio, lactate dehydrogenase, and C-reactive protein, had a strong association with the occurrence of newly detected diabetes (P=0.0001). It's essential to note that these risk factors differed from our study, primarily due to the younger age (38 vs. 48) and lower BMI (24.3 vs. 26.2) of participants in our study. Additionally, various laboratory markers were not recorded in our study, as the pilot study did not provide previous laboratory investigation reports, except for RT-PCR and antigen tests for COVID-19. Keerthi BY et al. [15] found associations with a family history of DM (P < 0.001), severity at admission (P < 0.006), diabetic ketoacidosis (P < 0.0275), and persistent symptoms, all significantly associated with newly detected DM. Individuals with newly detected DM had significantly higher BMI, O2 duration, and steroid duration (P < 0.001 for all). In comparison, Keerthi BY et al. [15] could not establish associations with persistent symptoms and BMI. This discrepancy may be attributed to most cases in our study being mild and not requiring hospitalization. Additionally, our study participants were younger and had lower BMIs than those in Keerthi BY et al.'s [15] study. Lastly, a binary logistic regression analysis was performed to adjust for potential confounding factors. This analysis highlighted age as the sole risk factor significantly associated with dysglycemia following COVID-19 infection. Older individuals were found to have a 7% increased risk of developing dysglycemia after SARS-CoV-2 infection, a finding that aligns with the well-established association between age and diabetes [19]. In summary, the prevalence of dysglycemia in the COVID-19 group was slightly higher than in the control group, but this difference was not statistically significant. However, the severity of COVID-19 emerged as a significant risk factor for dysglycemia, with older age being the sole risk factor in logistic regression analysis. Furthermore, while the influence of steroid therapy and oxygen use on dysglycemia did not achieve statistical significance in logistic regression, their clinical relevance should not be underestimated, and vigilant monitoring is needed for individuals who received these treatments during COVID-19 infection.

Limitations of The Study

1. Previous diabetes was excluded based on patient-provided information rather than documented medical records.
2. The control group was selected based on clinical history suggestive of COVID-19, without confirmatory antibody testing to exclude previous infection.
3. There were few patients in the severe COVID category.
4. The fact that everyday intraassay and interassay CVs were not checked but instead the standard coefficient of variation of the kit was considered may introduce potential variability in the measurements.

5. Conclusions

This study doesn't find any association between COVID-19 infection and dysglycemia compared to the control group. Dysglycemia is more frequent in severe COVID-19. There is a preponderance of dysglycemia with the advancement of age, O2 and steroid therapy.

Recommendations

Healthcare providers should remain vigilant in

monitoring the glycemic status of individuals recovering from COVID-19, especially those who experienced severe disease and aged. To determine the true newly detected dysglycemia rate, a large cohort study with severely affected COVID-19 participants is required.There is a scope of study to see new development of T1DM in COVID-19 patient. A follow-up study can be conducted to examine the long-term glycemic profiles of COVID-19 patients.

6. References

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