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Association between Blood Glucose Levels and Biochemical Variables in COVID-19 Patients - A Cross-Sectional Study

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ABSTRACT

Objectives: To identify the association between blood glucose concentrations and biochemical variables that lead to high blood glucose levels in patients with coronavirus disease 2019 (COVID-19).

Methods: This study was conducted on 100 plasma samples obtained from individuals with confirmed COVID-19, diagnosed using polymerase chain reaction. Samples were collected from the Barakuli Isolation Centre, Respiratory Clinic, and Sebha Medical Center in Sebha City, Libya. Concentrations of glucose, insulin, cortisol, triglycerides, cholesterol, C-reactive protein, and liver enzyme activities were measured.

Results: Sixty percent of patients had elevated blood glucose concentrations, whereas 40% had normal concentrations. The levels of insulin, cortisol, CRP, triglycerides, and liver enzyme activity were compared between the two groups. The results showed an increase in the mean concentrations of cortisol, triglycerides, CRP, and liver enzymes in patients with elevated glucose concentrations compared with those in patients with normal glucose concentrations. Statistical analysis using the t-test showed significant differences between the means ($p = 0.000$). Conversely, the mean insulin concentration was low in patients with elevated glucose levels. The results also showed a positive correlation between glucose and cortisol, triglyceride, and CRP concentrations, as well as GPT and GOT activity.

Conclusion: Patients with COVID-19 exhibited elevated blood glucose concentrations associated with decreased insulin levels. Additionally, high concentrations of biochemical variables contributing to hyperglycemia were observed in patients with COVID-19.

العلاقة بين مستوى جلوكوزالدم والمتغيرات البيوكيميائية لدى مرضى كوفيد 19– دراسة مقطعية

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الكلمات المفتاحية

جلكوز الدم
كوفيد 19
الأنسولين
المرضى

الملخص

الهدف : تهدف هذه الدراسة إلى تحليل العلاقة بين تركيز الجلوكوز في الدم والمتغيرات الكيميائية الحيوية لدى مرضى كوفيد-19.

الطرق: شملت الدراسة 100عينة ذكور وإناث (من مرضى تم تأكيد إصابتهم بكوفيد 19-بواسطة تفاعل البوليميراز التسلسلي PCR، تم قياس تركيز الجلوكوز، الأنسولين، الكورتيزول، الدهون الثلاثية، الكوليسترول، البروتين التفاعلي C، وإنزيمات الكبد، في عينات البلازما لجميع المرضى. كما تم حساب مقاومة الأنسولين ووظيفة خلايا بيتا لجميع المرضى.

النتائج: أظهرت النتائج أن 60% من المرضى لديهم ارتفاع في جلوكوز الدم، مع انخفاض معنوي في مستوى الأنسولين ترافق مع إنخفاض في وظيفة خلايا بيتا في البنكرياس، كما ارتبط ارتفاع الجلوكوز لديهم مع تركيز الكورتيزول، الدهون الثلاثية، CRP، وإنزيمات الكبد (GPT, GOT).

الاستنتاجات: تشير الدراسة إلى أن كوفيد -19قد يؤثر على التحكم في جلوكوز الدم عبر آليات متعددة، بما في ذلك إفراز الأنسولين من خلايا بيتا في البنكرياس والمساهمة في ارتفاع هرمونات الإجهاد وحدوث الالتهاب.

Background

Coronavirus disease 2019 (COVID-19), also known as acute respiratory disease caused by the novel coronavirus, affects humans following exposure to coronaviruses of the subfamily Orthocoronavirinae, which are single-stranded RNA viruses that infect mammals and birds [1]. Since the onset of the COVID-19 pandemic, studies have shown that diabetes is one of the main comorbidities associated with increased severity and mortality of COVID-19 symptoms [2]. Adults with diabetes are at greater risk for acute respiratory distress syndrome, pneumonia, and exaggerated inflammatory and hypercoagulable responses [3]. Recently, a new hypothesis has emerged proposing a bidirectional relation between diabetes and COVID-19 — not only does preexisting diabetes increase the risk of complications from COVID-19,

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but the virus itself may contribute to the development of diabetes [4]. Locally, there are many studies that have addressed diabetes and COVID-19 as two separate diseases, but the relation between them has not been studied (to the authors' knowledge) [5-12]. Worldwide, many studies have investigated the relation between COVID-19 and diabetes. However, do all patients with COVID-19 develop diabetes? Was the observed hyperglycemia caused by the virus itself, or were patients who exhibited elevated blood glucose levels already diabetic prior to infection but only diagnosed after contracting the virus? What are the underlying causes of hyperglycemia in these cases?

Other studies have reported that patients with COVID-19 and hyperglycemia — whether previously diagnosed with diabetes or newly developing it — experienced more severe disease outcomes compared with those of patients without diabetes or hyperglycemia [13, 14]. Following the emergence of these complications, and as researchers confirmed elevated blood glucose levels in patients with COVID-19, investigations began to identify the causes of post-infection hyperglycemia. What are the contributing factors? Can all patients develop diabetes upon infection? Does the condition persist after recovery from COVID-19? These questions have led to several hypotheses regarding the mechanisms of virus-induced diabetes. One such hypothesis suggests that COVID-19 targets multiple organs, including the pancreas [14]. Previous studies have identified viral receptors in the endocrine tissues of the pancreas, leading to damage to the insulin-producing islet cells of Langerhans, thereby disrupting glucose metabolism and impairing insulin secretion. This may result in hyperglycemia and diabetic ketoacidosis — potential triggers for type 1 diabetes [15]. Notably, researchers have reported that most cases of diabetes emerging during hospitalization resolved within 3 years.

Some prevalent treatments for COVID-19 include the use of glucocorticoids, a class of steroid hormones that affect carbohydrate metabolism, and insulin hormone antagonists, which can cause significant variations in blood glucose levels, potentially leading to type II diabetes [16]. The relation between COVID-19 and diabetes may be related to inflammation, as previous studies have indicated a relation between diabetes and the occurrence of inflammation in the body, leading to an immune response that carries out a series of reactions resulting in insulin resistance, which may lead to type II diabetes [17]. High body mass index, overweight, and obesity were also among the hypotheses developed to explain the cause of high blood glucose and the new diabetes caused by COVID-19, as obesity complicates the body's glucose metabolism [18], and high blood glucose is closely related to obesity, which plays an important role in disease severity and mortality [19]. Some studies conducted on patients with COVID-19 have recorded cases of obesity in patient profiles associated with high glucose levels during their period of infection with the virus, and a reference study that included numerous studies around the world indicated that obesity may also increase the risk of infection with COVID-19. The relation between obesity and COVID-19 is bilateral and mutual: obesity is a cause of high glucose in patients with COVID-19, and conversely, obesity is one of the factors that increase the risk of infection with COVID-19 [20]. Reports issued by the World Health Organization have indicated that during the quarantine period — a measure to prevent infection with the virus — there was a decrease in the physical activity of people, along with unhealthy eating habits; all these factors led to weight gain and changes in insulin resistance, increasing the likelihood of the onset of diabetes [21]. This study aimed to determine the relation between blood glucose concentration and biochemical variables that lead to high blood glucose levels in patients with COVID-19.

Methods

Study design and sample

One hundred samples of people infected with COVID-19 were collected from isolation centers in the city of Sebha (Libya), including the Barakuli Isolation Center, Respiratory Clinic, and Sebha Medical Center. Data collection was conducted from August 31 to October 20, 2020, where the mean age of the infected was 57.07 ± 20.87 years, divided into 36 women and 64 men. Their diagnosis of COVID-19 was confirmed using polymerase chain reaction, where the test result was positive for all patients from whom samples were collected. These samples were collected in tubes containing lithium heparin anticoagulant, suitable for conducting all biochemical tests without interference or false results.

Procedures

The measurement was performed using a Cobas Integra 400 Plus device, which is a fully automatic and computerized chemical analyzer. Hormones were measured using a Cobas e 411, a fully automated analyzer using patented electrochemiluminescence immunoassay technology.

Homeostatic model assessment of insulin resistance (HOMA-IR) and beta cell function (HOMA-β)

HOMA-IR and HOMA-β constitute a method for assessing beta cell function and insulin resistance based on glucose and insulin concentrations. To calculate the values of insulin resistance and β cell function, the following equations were used:

HOMA-IR = [fasting insulin (μU/mL) × fasting plasma glucose (mmol/L)] / 22.5

HOMA-β% = [fasting insulin (μU/mL) × 20] / [fasting plasma glucose (mmol/L) – 3.5]

Statistical analysis

The data were statistically analyzed using SPSS Statistics, Version 26.0 (Armonk, NY: IBM Corp). We used a two-sample t-test and confidence interval to analyze variance between groups and Pearson correlation between variables at a significance level of ≤ 0.05.

Results

Comparison of the concentration of biochemical variables in patients based on blood glucose concentration

The patients were divided into two groups based on their blood glucose levels: a group with high glucose levels consisting of 60 individuals, and a group with normal glucose levels consisting of 40 individuals. The mean concentrations of insulin, cortisol, C-reactive protein (CRP), triglycerides, cholesterol, and liver enzyme activities were analyzed in both groups. The results indicated that the mean cortisol concentration in the group of patients with high glucose levels was significantly higher than that in patients with normal glucose levels ($p = 0.000$). Furthermore, the mean insulin concentration was significantly lower in the group of patients with high glucose levels than in the group with normal glucose levels ($p = 0.000$). Similarly, the mean concentrations of CRP, triglycerides, and cholesterol were significantly higher in the group of patients with high glucose levels than in the group with normal glucose levels ($p = 0.000$). In addition, the results showed that the mean activity of liver enzymes was higher in the group of patients with

high glucose concentrations than in that of patients with normal glucose concentrations, with significant differences in these means between the two groups, except for ALP, where statistical analysis did not indicate significant differences between the two groups. The detailed comparison is shown in Table 1.

Table 1: Comparison of biochemical variables in patients with high versus normal blood glucose

Variables	High Glucose (n=60)	Normal Glucose (n=40)	P-value
Glucose	11.2 ± 4.4	4.9 ± 0.7	0.000*
Insulin (µU/mL)	3.4 ± 1.11	11.7 ± 5.8	0.000*
Cortisol (nmol/L)	592 ± 222	255.1 ± 70.2	0.000*
CRP (mmol/L)	66.6 ± 30.2	229 ± 121	0.000*
Triglycerides (mmol/L)	2.90 ± 1.47	1.52 ± 0.45	0.000*
Cholesterol (mmol/L)	8.26 ± 2.04	3.39 ± 1.02	0.000*
GOT (U/L)	106.5 ± 79.9	30.7 ± 12.1	0.000*
GPT (U/L)	145 ± 30.7	39.8 ± 15.4	0.000*
GGT (U/L)	54.6 ± 16.4	21.5 ± 11.9	0.000*
ALP (U/L)	171 ± 18.2	144.8 ± 20.6	0.113

Values are presented as mean ± standard deviation (SD), CRP: C reactive protein, TG: Triglyceride, CHOL: Cholesterol, GOT: Glutamate Oxaloacetate Transaminase, GPT: Glutamate Pyruvate Transaminase, GGT: Gamma-Glutamyl Transferase, ALP: Alkaline Phosphatase. *Significant ($p < 0.05$).

Correlation between glucose and other variables in patients with high glucose

When examining the correlation coefficient between glucose concentration and other variables in patients with hyperglycemia, the results indicated a significantly strong negative correlation with insulin concentration ($r = -0.63$, $p = 0.000$) and a significantly strong positive correlation with cortisol concentration ($r = 0.61$, $p = 0.000$). In addition, there was a significantly positive correlation with CRP concentration ($r = 0.43$, $p = 0.000$) and a weak positive correlation with both cholesterol and triglyceride concentrations ($r = 0.29$, $p = 0.002$; $r = 0.39$, $p = 0.002$). Regarding liver enzymes, the correlation analysis showed a weak positive correlation between glucose concentration and GOT and GPT enzyme activities ($r = 0.30$, $p = 0.02$; $r = 0.35$, $p = 0.006$), whereas there was no significant correlation between glucose concentration and GGT or ALP activities ($p > 0.05$). When testing the correlation between glucose concentration and other variables in the group of patients with normal glucose levels, no significant association was found ($p > 0.05$) (Table 2).

Table 2: The correlation coefficient between glucose and other variables in the group of patients with high glucose concentration

	Glucose (mmol/L)	
Variables	**r**	***P- value***
Insulin (µU/mL)	-0.63	0.000*
Cortisol (nmol/L)	0.61	0.000*
CRP (mmol/L)	0.34	0.000*
TG (mmol/L)	0.29	0.002*
CHOL (mmol/L)	0.39	0.002*
GOT(U\L)	0.29	0.02*
GPT(U\L)	0.35	0.006*
GGT(U\L)	0.18	0.15
ALP(U\L)	0.2	0.19

CRP: C reactive protein, TG: Triglyceride, CHOL: Cholesterol, GOT: Glutamate Oxaloacetate Transaminase, GPT: Glutamate Pyruvate Transaminase, GGT: Gamma-Glutamyl Transferase, ALP: Alkaline Phosphatase. *Significant ($p < 0.05$).

HOMA-IR calculation

The HOMA-IR value was calculated for the group of patients whose glucose concentration was normal, and the results showed that their mean insulin resistance value was 2.54 ± 1.27, which is high compared to the reference range, indicating that this group had high insulin resistance (Table3). When testing the correlation between HOMA-IR and insulin concentration, and between glucose and insulin concentration, there was a significantly strong positive correlation ($r = 0.90$, $p = 0.000$) and a significantly strong negative correlation ($r = -0.30$, $p = 0.04$), respectively.

HOMA-β calculation

The results showed that the mean HOMA-β level in patients with high glucose concentration (accompanied by low insulin concentration) was below the normal level, and patients with normal glucose concentration (accompanied by high insulin concentration) had a higher mean HOMA-β compared with that of the high glucose group ($p = 0.000$) (Table 3). When measuring the correlation between HOMA-β and insulin concentration in both groups, we found a strong positive correlation ($r = 0.96$, $p = 0.000$; $r = 0.51$, $p = 0.002$, respectively).

Discussion

One of the reasons that contributed to mortality during the emergence of COVID-19 was high blood sugar, and opinions differed regarding the reason for the increase in diabetes in infected patients. Numerous studies have reported that the frequent occurrence of high blood sugar levels in individuals is associated with numerous deaths, confirming its status as a critical and severe complication of the disease [22]. Patients with diabetes are particularly susceptible to respiratory issues, breathing difficulties, and weakened immune responses, as evidenced by data from Canada in September 2020, which is consistent with findings from other nations. Studies have highlighted the increased risk of severe COVID-19 in individuals with diabetes [23], resulting in a higher mortality rate compared with that of patients without diabetes. Moreover, it is noteworthy that even patients without diabetes presented with elevated blood sugar levels [24].

Table 3: Comparison of HOMA-IR and HOMA-β values in both groups

Variables	Patients with high glucose (n=60)	Patients with normal glucose (n=40)	*P value*
HOMA-IR	2.55 ± 1.27	1.25 ± 1.15	0.000*
HOMA-β	352.46 ± 124	18.26 ± 17.55	0.000*

Values are presented as mean ± standard deviation (SD), *= Significant; HOMA-IR: The homeostasis model assessment of insulin resistance, and HOMA-β: The homeostasis model assessment of beta cell function

The impact of COVID-19 on blood sugar levels raises questions regarding whether the virus induces dysregulation of glucose levels by compromising insulin secretion, potentially leading to the long-term development of diabetes. Another aspect to consider is whether irregularities occur because of infection-related factors that contribute to increased blood glucose levels. Several theories have been proposed to explain the relation between COVID-19 and elevated blood glucose levels. One theory suggests that the virus damages pancreatic beta cells responsible for insulin production, resulting in high blood glucose levels [25]. In a study involving patients with COVID-19 confirmed by polymerase chain reaction testing and placed in isolation, 60% of the patients exhibited elevated blood glucose levels, whereas 40% did not. These results indicate that more than half of infected individuals experience an increase in glucose levels [26]. Additionally, patients with elevated glucose levels had lower insulin levels than those of patients with normal glucose levels, supporting the theory that COVID-19 may lead to the destruction of pancreatic beta cells and subsequently decrease insulin secretion. These findings align with the conclusion that the virus diminishes insulin secretion by directly affecting pancreatic beta cells [26].

Earlier studies have indicated that elevated levels of cortisol in the blood can directly decrease insulin secretion by affecting the secretion rate of beta cells in the pancreas. This has been observed in laboratory experiments involving cultured beta cells and in experiments with genetically modified mice. These studies have revealed that high cortisol levels reduce insulin secretion by inhibiting hormone release. Moreover, research has shown that patients with significantly elevated cortisol levels were treated with dexamethasone (4 mg twice daily) along with antibiotics, such as azithromycin and amantadine, at the beginning of the pandemic. Dexamethasone, a synthetic corticosteroid used to alleviate inflammation, was administered to patients with respiratory distress who required oxygen therapy. However, this medication can result in various complications, such as increased levels of triglycerides, cholesterol, and blood glucose [27]. Therefore, patients taking this synthetic hormone, in addition to the natural cortisol produced in their bodies (which escalates in response to stress, tension, and anxiety — hence its label as the “stress hormone”), may experience changes that contribute to heightened blood glucose levels, known as stress hyperglycemia [27].

Several studies have suggested a positive relation between CRP levels and prediabetes, including hyperglycemia and metabolic syndrome. However, these studies have not definitively confirmed whether high blood glucose levels lead to inflammation and consequently stimulate CRP secretion, or whether high CRP levels lead to an increase in blood glucose through its impact on insulin [28]. Additionally, acute inflammation of liver cells, such as in hepatitis, can cause insulin resistance and subsequent high blood glucose, contributing to the onset of type II diabetes [29]. While some studies have demonstrated acute inflammation in liver cells, indicated by a clear increase in the liver enzymes GOT and GPT, there was no evidence of insulin resistance as insulin levels were low, thus excluding hepatitis as the cause of high blood glucose levels. Consequently, the elevation of liver enzymes can be attributed to an inflammatory condition in the body resulting from viral infection.

Comparing the findings of this study with existing theories attempting to explain the relation between COVID-19 and high blood sugar, it is evident that factors leading to elevated blood glucose levels were present in the patients studied. Therefore, it can be inferred that these factors collectively contribute to the increase in blood glucose levels. These factors are likely to produce similar effects in patients with diabetes before contracting COVID-19, possibly leading to a significant increase in blood glucose levels and even fatalities. Moreover, some studies have indicated that several patients develop diabetes after recovering from COVID-19, implying a direct impact of the virus on pancreatic beta cells, resulting in reduced insulin secretion, as supported by the findings of this study.

In this study, insulin secretion was assessed using HOMA-β as a model to evaluate pancreatic β-cell function. The results demonstrated a statistically significant difference in HOMA-β values between individuals with high blood glucose and those with normal blood glucose. The findings indicated that a decline in β-cell function is associated with low HOMA-β values in individuals with high blood glucose.

The study results also indicated that the HOMA-IR value was elevated in individuals with normal blood glucose concentrations. High HOMA-IR values are indicative of insulin resistance, suggesting that these individuals were in the prediabetes stage. In this group, the HOMA-β value was higher compared with that of the group with high glucose levels. Elevated HOMA-β and HOMA-IR values suggest that pancreatic β-cells were exerting increased effort to secrete more insulin because of insulin resistance. This compensatory process occurs in the prediabetes stage, confirming that this group is at heightened risk of developing type 2 diabetes if the underlying causes are not addressed [30]. These results are consistent with findings from other studies reporting that COVID-19 leads to the destruction of pancreatic β-cells [14], resulting in a reduction in insulin concentration and a corresponding increase in blood glucose concentration.

This study had some limitations. First, the relatively small sample size and its restriction to one geographic region (Sebha, Libya) may limit the statistical power and external validity of the results. Second, the cross-sectional design prevents any inference of causality between elevated glucose and biochemical variables. Third, due to the absence of prior medical history, including known diabetes status or medication use, some findings may be confounded. Lastly, the sampling approach might have introduced selection bias. Additionally, the study findings may not be directly generalizable to broader populations due to geographic and demographic limitations of the sample. Further research involving larger, more diverse cohorts from different regions is necessary to validate and expand upon these results.

Conclusion

Blood glucose levels increase in some patients infected with COVID-19, accompanied by a decrease in insulin concentration. Our findings also revealed an increase in variables associated with elevated blood glucose concentration — namely, cortisol and fat — accompanied by a strong positive correlation with these variables.

Declarations

Ethics approval and consent to participate: Ethical approval for this study was obtained from the Research Ethics Committee of the authors’ university, and all procedures were carried out in accordance with relevant institutional and international guidelines. The study was conducted during the COVID-19 pandemic using blood

samples collected from patients at the Respiratory Clinic of Sabha Medical Center and the Barakuli Isolation Centre. These samples were originally obtained for routine diagnostic purposes at the request of treating physicians and were subsequently used for research analysis. No additional clinical interventions or drug trials were performed on patients, and the researchers had no direct contact with patients. Because only de-identified residual diagnostic specimens were used, the requirement for individual informed consent was waived by the ethics committee.

Consent for publication: Not applicable. The manuscript contains no identifiable individual person's data or images, and therefore consent for publication is not required. All authors have read and approved the final version of the manuscript and consent to its publication, acknowledging its scientific significance.

Authors' contributions: Bisher: Conceptualized the study, developed the research design, performed the data analysis, and interpreted the results. **Thamer**: Wrote the first draft of the manuscript. **Shahata** and **Thamer**: Data collection and measuring variables during the experimental phase. All authors critically reviewed the manuscript and approved the final version for publication..

Data Availability: The datasets generated and/or analysed during the current study contain sensitive patient information and are not publicly available due to confidentiality and ethical restrictions. De-identified data relating to test results are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests. None of the authors have any financial or personal relationships that could inappropriately influence this work. The authors also confirm that no conflicts of interest exist related to this study.

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