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CORRELATION OF SERUM 25-HYDROXYVITAMIN D LEVELS WITH GLYCEMIC CONTROL IN TYPE 2 DIABETES MELLITUS PATIENTS

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is caused by reduced insulin sensitivity and secretion. Several recent investigations have found that vitamin D is connected with insulin secretion and sensitivity. Vitamin D insufficiency is linked to poor glucose regulation. The research aims to determine the correlation between serum 25-hydroxyvitamin D levels and glycemic control in T2DM patients.

Method: T2DM patients' serum levels of 25-Hydroxyvitamin D, Fasting Blood Glucose (FBG), 2-hours postprandial blood glucose (PPBG), and HbA1c were examined in this cross-sectional analytical observational study. Statistical analysis was also performed.

Result: From 49 samples included, the number of male subjects was almost the same as female, mean age was 58 years. There was a decrease in serum 25-Hydroxyvitamin D levels with a median of 27.87 ng/ml. Glycemic control was found to be poor, the median of FBG levels was 134 mg/dl. 2-h PBG levels were 208 mg/dl and HbA1c levels were 7.5%. Analysis using Spearman correlation between serum 25-Hydroxyvitamin D levels with levels of FBG. 2-h PPBG. and HbA1c showed values r=-0.538 and p=0.001; r=-0.354 and p=0.013; r=-0.501 and p=0.001.

Conclusion: There was a statistically significant negative correlation between serum 25-Hydroxyvitamin D levels and levels of FBP, PPBG, and HbA1c in T2DM patients.

Keywords: *25-Hydroxyvitamin D, FBG, PPBG, HbA1c.*

ABSTRAK

Latar Belakang: *Diabetes mellitus tipe 2 (DMT2) disebabkan oleh berkurangnya sensitivitas dan sekresi insulin. Beberapa penelitian baru-baru ini telah menemukan bahwa vitamin D terkait dengan sekresi dan sensitivitas insulin. Kekurangan vitamin D terkait dengan regulasi glukosa yang buruk. Tujuan penelitian adalah mengetahui korelasi antara kadar serum 25-hidroksivitamin D dan kontrol glikemik pada pasien T2DM.*

Metode: *Dilakukan analisis kadar serum pasien T2DM 25-Hydroxyvitamin D, gula darah puasa (GDP), 2 jam glukosa postprandial (GDPP), dan HbA1c diperiksa dalam studi observasional analitik cross-sectional..*

Hasil: *Dari 49 sampel yang dimasukkan, jumlah subjek laki-laki hampir sama dengan perempuan, usia rata-rata 58 tahun. Terjadi penurunan kadar serum 25-Hydroxyvitamin D dengan median 27,87 ng/ml. Kontrol glikemik ditemukan buruk, median kadar gula darah puasa (GDP) adalah 134 mg/dl. Kadar gula darah postprandial (GDPP) adalah 208 mg/dl dan kadar HbA1c adalah 7,5%. Analisis menggunakan korelasi Spearman antara kadar serum 25-Hydroxyvitamin D dengan kadar GDP. GDPP dan HbA1c menunjukkan nilai r=-0,538 dan p=0,001; r=-0,354 dan p=0,013; r=-0,501 dan p=0,001.*

Kesimpulan: *Ada korelasi negatif yang signifikan secara statistik antara kadar serum 25-Hydroxyvitamin D dan kadar GDP, GDPP dan HbA1c pada pasien T2DM.*

Kata kunci: *25-Hydroxyvitamin D, GDP, GDPP, HbA1c.*

1. Introduction

Diabetes Mellitus (DM) is a group of metabolic diseases characterized by hyperglycemia. This disease is caused by impaired insulin secretion impaired insulin sensitivity or both [1]. Managing T2DM is crucial for attaining glycemic control to avert its complications. Sure, please provide the text you would like me to paraphrase. A deficiency in vitamin D has emerged as a worldwide crisis, particularly among individuals with metabolic syndrome and obesity, even in regions that experience sufficient sunlight, and this could elevate the global disease burden. Due to the widespread occurrence of vitamin D deficiency [2]. The main biomarker for assessing vitamin D status currently is the serum level of 25(OH)D. This indicator reflects both vitamin D produced in the body and that obtained from food and supplements. A circulation half-life of twenty-five days for 25(OH)D in serum is quite lengthy. In contrast to 25(OH)D, circulating 1,25(OH)2D has a brief half-life (in hours), with parathyroid hormone, calcium, and phosphate tightly regulating its blood levels, rendering it an unreliable indicator of overall vitamin D status. There is a significant association between higher vitamin D levels and better glycemic control [3]. In the large-scale prospective cohort study, serum 25OHD concentrations were inversely associated with the risk of incident T2D in a dose-response fashion, independent of traditional risk factors. Several theories suggest that vitamin D improves pancreatic beta cell function. The active form of vitamin D can upregulate insulin receptor expression, and stimulate insulin secretion. Low levels of vitamin D are thought to be one of the causes of poor glycemic control [4]. Poor glycemic control increases the risk of type 2 diabetes complications, both micro and macrovascular complications. The study aimed to determine the correlation between serum 25-hydroxyvitamin D levels and glycemic control in T2DM patients in the Division of Endocrine Metabolic Diabetes, M. Djamil Padang Hospital.

2. Method

This research is an analytical observational study with a cross-sectional approach. The research was conducted at RSUP by Dr. M. Djamil Padang for 6 months. The research sample is a population that meets the inclusion and exclusion criteria. The sample collection method was consecutive sampling. Inclusion criteria are patients aged ≥ 30 years, patients willing to take part in the study, and patients who have been taking oral antidiabetic drugs combined with biguanide and sulfonylurea regularly for at least 3 months. Exclusion criteria are patients with renal failure and hemodialysis, liver dysfunction, acute infection, malignancy, anemia, cardiovascular disease, rickets, osteomalacia, patients with pregnancy, patients who received a blood transfusion in the last 3 months, and patients taking vitamin D. The samples were examined for levels of serum 25-hydroxyvitamin D, fasting blood glucose (FBG), 2 hours postprandial blood glucose (2-h PPG) and HbA1c. Serum 25-hydroxyvitamin D levels were examined using the enzyme-linked immunosorbent assay (ELISA) method using venous blood samples in ng/ml units. Normal values are 30-100 ng/ml. Descriptive analysis of basic data was carried out. Includes patient characteristics and laboratory tests. Numerical data is displayed in the form of a median. Numerical data was tested for normality using the Shapiro-Wilk test. Pearson correlation and Spearman correlation analyses were carried out. Data is processed and computerized using SPSS 26.

3. Result

In this study, the characteristics of 49 T2DM patients were obtained which can be seen in Table 1. Type 2 DM patients in this study were found to be almost equally male and female, namely 25 men (51%) and 24 women (49%). Based on age characteristics, the mean was 58.14 years with the largest age group of 45-65 years as many as 40 people (81.6%).

Table 1 Basic Characteristics

Characteristics	n (%)	Mean (SD)
Gender		
Male	25 (51)	
Female	24 (49)	
Age (year)		58.1 (9.0)
< 45	4 (8.2)	
45-65	40 (81.6)	
> 65	5 (10.2)	
Body Mass Index		
18.5-22.0	21 (42.9)	
23-24.9	11 (22.4)	
25-29.9	12 (26.5)	
≥ 30	4 (8.2)	
Duration of treatment (yr)		
< 5	40 (81.6)	
5-10	6 (12.2)	
> 10	3 (6.1)	
Sun exposure/day (minutes)		
< 60	15 (30.6)	
60-180	34 (69.4)	
> 180	0.0	

Based on the characteristics of body mass index (BMI), in this study, the samples with normal BMI of 18.5-22 kg/m2 were 21 people (42.9%). A total of 40 people (81.6%) underwent treatment duration of less than 5 years. Based on the basic characteristics of sun exposure, most samples received sun exposure of 60-180 minutes per day, namely 34 people (69.4%).
In this study, the results of serum 25-hydroxyvitamin D levels were not normally distributed. The median serum 25-hydroxyvitamin D level was 27.87 (12.626-94.367) ng/ml which can be seen in Table 2.

Table 2 Serum 25-*hydroxyvitamin* D Levels in T2DM Patients

Variable	Median (Min-Max)
25-*hydroxyvitamin* D (ng/ml)	27.87 (12.6-94.3.4)

The distribution of serum 25-hydroxyvitamin D levels in Type 2 DM patients in this study can be seen in Table 3. Normal serum 25-hydroxyvitamin D levels were found in 18 patients (36.7%) and serum 25-hydroxyvitamin D levels below normal were found in 31 patients (63.3%) and there were no samples that had serum 25-hydroxyvitamin D levels above normal.

Table 3 Distribution of Serum 25-hydroxyvitamin D Levels in Patients with Type 2 DM

Serum 25-*hydroxyvitamin* D levels (ng/ml)	n (%)
< 30	31 (63.3)
30-100	18 (36.7)
>100	0

Serum 25- hydroxyvitamin D Level normal range: 30 – 100 ng/ml

The distribution of serum 25-hydroxyvitamin D levels in Type 2 DM patients divided based on their glycemic control can be seen in Table 4. Serum 25-hydroxyvitamin D levels below normal were found more in patients with poor glycemic control.

Table 4 Distribution of Serum 25- hydroxyvitamin D Level and Glycemic Control in Type 2 DM Patients

Glycemic control	Serum 25- hydroxyvitamin D Level (ng/ml)	n (%)
Controlled fasting blood glucose	< 30	7 (35)
	30-100	13 (65)
	>100	0
Uncontrolled fasting blood glucose	< 30	24 (82.7)
	30-100	5 (17.3)
	>100	0
Controlled 2-hour postprandial blood glucose	< 30	9 (50)
	30-100	9 (50)
	>100	0
Uncontrolled 2-hour postprandial blood glucose	< 30	22 (70.9)
	30-100	9 (29.1)
	>100	0
Controlled HbA1c	< 30	4 (22.2)
	30-100	14 (77.3)
	>100	0
Uncontrolled HbA1c	< 30	27 (87)
	30-100	4 (13)
	>100	0

Serum 25- hydroxyvitamin D Level normal range: 30 – 100 ng/ml

In this study, the results of fasting blood glucose, 2-hour postprandial blood glucose, and HbA1c levels were not normally distributed. The median levels of fasting blood glucose, 2-hour postprandial blood glucose, and HbA1c were 134 (61-339) mg/dl, 208 (93-488) mg/dl, and 7.5 (5.6-12.8)% which can be seen in Table 5.

Table 5 Fasting blood glucose, 2-hour postprandial blood glucose, HbA1c levels in type 2 DM patient

Variable (unit)	Median (Min-Max)
FBG (mg/dl)	134.0 (61.0-339.0)
2-h PPG (mg/dl)	208.0 (93.0-488.0)
HbA1c (%)	7.5 (5.6-12.8)

Based on Figure 1, it can be concluded that the higher the serum 25-hydroxyvitamin D level, the lower the fasting blood glucose level. The results of the analysis showed that there was a moderate correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose (r=-0.538) with a negative correlation direction that was statistically significant (p<0.005) and r^2 of 0.167 which means that serum 25-hydroxyvitamin D levels influenced fasting blood glucose by 16.7%.

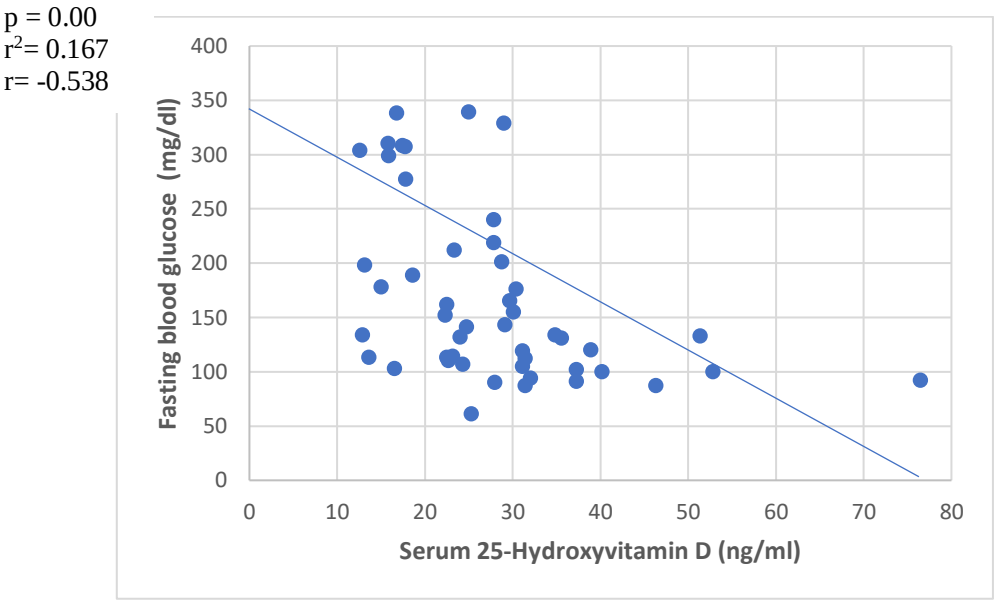

Figure 1 Correlation graph between serum 25-hydroxyvitamin D levels and fasting blood glucose levels in patients with T2DM

Based on Figure 5.2, it can be concluded that the higher the serum 25-hydroxyvitamin D level, the lower the 2-hour postprandial blood glucose level. The results of the analysis showed that there was a weak correlation between serum 25-hydroxyvitamin D levels and 2-hour postprandial blood glucose (r=-0.354) with a negative correlation direction that was statistically significant (p<0.005) and r^2 of 0.077 which means that serum 25-hydroxyvitamin D levels influenced 2-hour postprandial blood glucose by 7.7%.

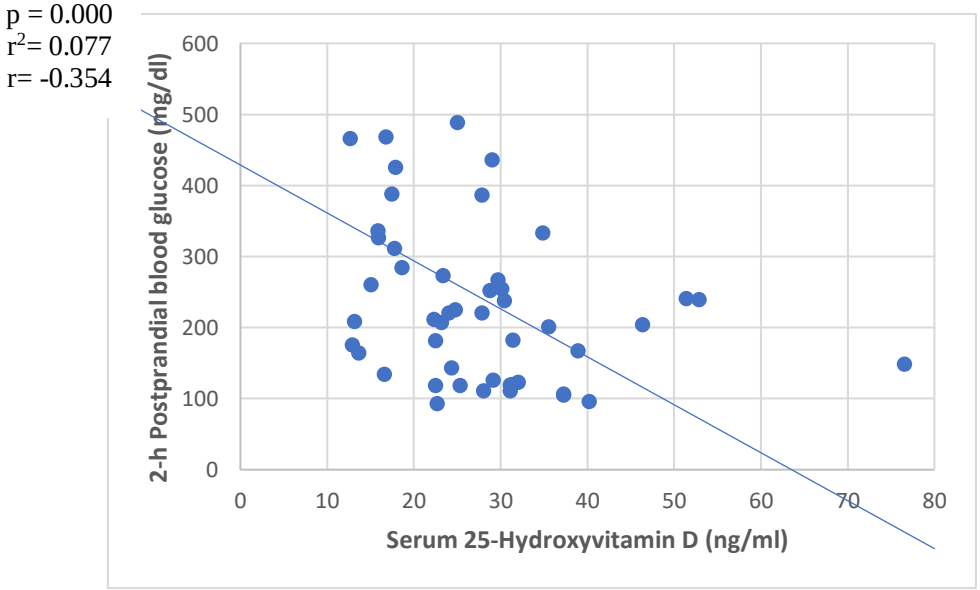

Figure 5.2 Correlation graph between serum 25-hydroxyvitamin D levels and blood glucose levels 2 hours postprandial in patients with T2DM

Based on Figure 5.3, it can be concluded that the higher the serum 25-hydroxyvitamin D level, the lower the HbA1c level. The results of the analysis showed that there was a moderate correlation between serum 25-hydroxyvitamin D levels and HbA1c (r=-0.501) with a negative correlation direction that was statistically significant (p<0.005) and r2 of 0.145, which means that serum 25-hydroxyvitamin D levels influenced HbA1c by 14.5%.

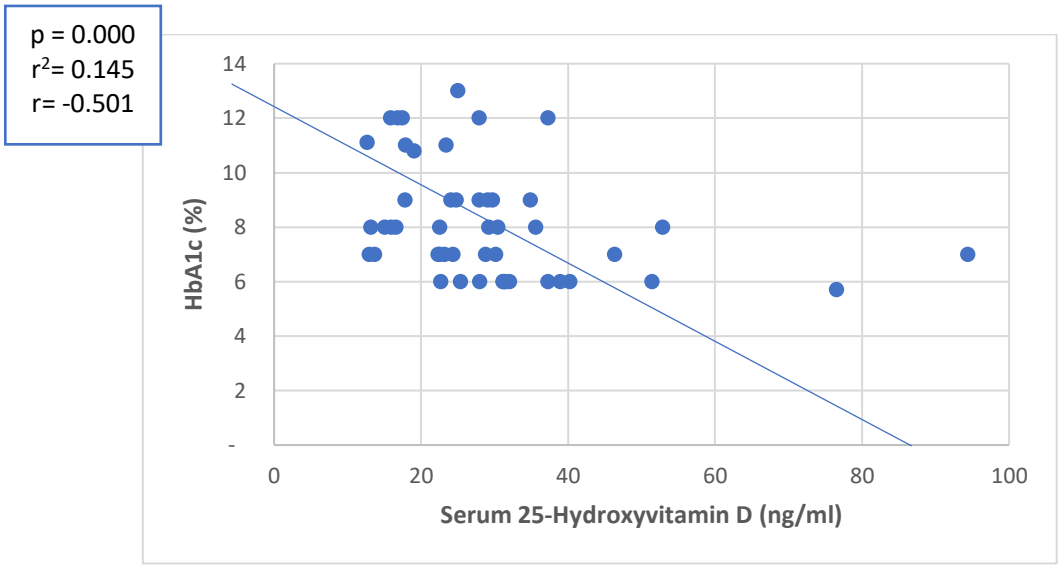

Figure 5.3 Correlation graph between serum 25-hydroxyvitamin D levels and HbA1c levels in patients with T2DM

4. Discussion

The analysis results indicate a moderate correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose (r=-0.538) with a statistically significant negative correlation (p<0.05) and an r² of 0.167, meaning that serum 25-hydroxyvitamin D levels influence fasting blood glucose by 16.7%, while the remaining percentage is influenced by other factors. Ehrampoush et al. (2021) in their research found results that align with a negative correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose. Fasting blood glucose was found to be significantly higher in patients with moderate to severe vitamin D deficiency (p<0.05). Similar research was found in the study by Yang et al. (2020), which reported a negative correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose (p<0.05). Meanwhile, the study by Alkhatatbeh et al. (2018) also found a negative correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose (r=-0.17, p<0.01). Another study by Valladares et al. (2019) indicated that low serum 25-hydroxyvitamin D levels, below 30 ng/dl, were associated with an increase in fasting blood glucose above 100 mg/dl (p<0.006). Additionally, the research by Huu et al. (2021) showed a negative correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose (r=-0.229, p=0.016). [5][6][7][8][9]

The analysis results show a weak correlation between serum 25-hydroxyvitamin D levels and blood glucose levels 2 hours postprandial (r=-0.354). There is a statistically significant negative correlation (p<0.005) and a r² of 0.077%, indicating that serum 25-hydroxyvitamin D levels influence blood glucose levels 2 hours postprandial by 7.7%, while the remaining percentage is influenced by other factors. The results of this study are in line with the research by Ehrampoush et al. (2021), which found a significant negative correlation between serum 25-hydroxyvitamin D levels and blood glucose levels 2 hours postprandial. The lower the levels of 25-hydroxyvitamin D, particularly in cases of moderate to severe vitamin D deficiency, the higher the blood glucose levels 2 hours postprandial were observed (p<0.05) [6].

The analysis results indicate a moderate correlation between serum levels of 25-hydroxyvitamin D and HbA1c (r=-0.501) with a statistically significant negative correlation (p<0.005) and an r² of 0.145, meaning that serum levels of 25-hydroxyvitamin D influence HbA1c by 14.5%, while the remaining is influenced by other factors. Similar research was conducted by Zhao et al. (2020), which found a significant negative correlation between serum 25-hydroxyvitamin D levels and HbA1c levels. The lower the serum 25-hydroxyvitamin D levels, the higher the HbA1c results (p<0.05). A similar finding was reported in the study by Alkhatatbeh et al. (2018), where HbA1c was found to be higher in participants with lower serum 25-hydroxyvitamin D levels. Another study by Swamy et al (2016) and Hossain et al (20210, showed a significant negative correlation between

vitamin D and HbA1c. There is a negative correlation between serum 25-hydroxyvitamin D levels and HbA1c. ($r=-0.23$, $p<0.01$). [7][10][11][12]

There is a difference in the degree of correlation in this study's results compared to other studies, particularly the correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose levels as well as HbA1c levels, which may be influenced by various factors affecting these levels. The inclusion and exclusion criteria in sample selection will also impact the examination results. For example, in this study, the sample selection was carried out by including inclusion criteria, namely patients who were only consuming oral antidiabetic medications, specifically a combination of biguanides and sulfonylureas. Meanwhile, in the research by Alkhatatbeh et al. (2018) and Huu et al. (2021), samples were taken from patients with T2DM who were undergoing various types of treatment for Type 2 Diabetes Mellitus. The differences in the types of treatment for T2DM will affect the glycemic control of the patients and will influence the degree of correlation. [7][9]

Vitamin D is believed to have effects that can weaken the pathophysiological mechanisms of pancreatic beta cell dysfunction and enhance pancreatic efficiency. Yaribeygi et al. (2020) state that vitamin D can improve glucose homeostasis by enhancing the function of pancreatic beta cells through the repair of molecular mechanisms. Vitamin D can enhance peripheral insulin sensitivity through three distinct pathways, including reducing oxidative damage, suppressing inflammatory responses, and increasing the expression and activity of insulin signaling transduction. The upregulation of insulin signaling transduction elements and the enhancement of its function can occur through the PI3K/Akt pathway and IRS-1, as well as the expression or localization of GLUT-4 and the induction of the PPAR-δ pathway. [13]

The correlation degree was also found to be different in this study. The correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose levels was found to be moderate, while the correlation between serum 25-hydroxyvitamin D levels and blood glucose levels two hours postprandial was found to be weak. The difference in the degree of correlation may be caused by factors that influence each level of fasting blood glucose, two-hour postprandial blood glucose, and HbA1c. The increase in blood glucose levels can generally be caused by eleven egregious factors: decreased insulin secretion by pancreatic beta cells, increased glucagon secretion by pancreatic alpha cells, increased lipolysis in fat cells, decreased glucose utilization in muscles, increased glucose production in the liver, neurotransmitter dysfunction in the brain, abnormal microbiota in the colon, increased glucose absorption in the small intestine and kidneys, accelerated gastric emptying, and dysregulation of the immune system related to inflammation. [6]

Osmani et al. (2020) state that vitamin D contributes to normalizing extracellular calcium, which is beneficial for calcium channels functioning in cell membranes. Low levels of vitamin D impact the reduction of calcium that operates on cell membranes regulating insulin secretion. Additionally, another mechanism related to vitamin D and T2DM is the enhancement of insulin action through increased regulation of insulin receptors, improved insulin response in insulin transport, indirect effects through calcium's impact on insulin secretion, and the improvement of systemic inflammation. [14]

Research conducted by Elsheikh (2024) shows that vitamin D status is strongly correlated with the prevalence of Type 2 diabetes. The increase in the number of patients with diabetes in the vitamin D deficiency group strengthens the hypothesis regarding the role of vitamin D in glucose metabolism. This mechanism is linked to insulin secretion, insulin sensitivity, and inflammation. Vitamin D stimulates the expression of insulin receptors and regulates calcium transport, which triggers insulin secretion. The signaling pathway of vitamin D receptors with pancreatic beta cells is also key in stimulating the biosynthesis and release of insulin. [15]

The r^2 value between serum 25-hydroxyvitamin D levels and fasting blood glucose control is 16.7%, meaning that serum 25-hydroxyvitamin D levels influence fasting blood glucose by 16.7%. The remaining factors that affect glycemic control are due to other influences. Other factors that can affect fasting blood glucose include increased glucagon that triggers gluconeogenesis and glycogenolysis, the intake of foods high in carbohydrates, and the inadequacy of diabetes medications being consumed. The r2 value between serum 25-hydroxyvitamin D levels and blood glucose control 2 hours postprandial is 7.7%, meaning that serum 25-hydroxyvitamin D levels influence fasting blood glucose by 7.7%. The remaining factors that affect blood glucose 2 hours postprandial are due to other influences. Postprandial blood glucose is greatly influenced by the food intake consumed. In patients with Type 2 diabetes, blood glucose levels postprandial will peak 2 hours after consuming food. Postprandial blood glucose is also influenced by other factors such as activity, insulin sensitivity, average gastric emptying, and the composition of food intake. High-carbohydrate foods have a significant contribution to the increase in postprandial blood glucose levels. The r^2 value between serum 25-hydroxyvitamin D levels and HbA1c glycemic control is 14.5%, meaning that serum 25-hydroxyvitamin D levels influence HbA1c by 14.5%. The remaining factors affecting HbA1c are other influences. HbA1c levels are greatly affected by blood glucose levels, both fasting blood glucose and postprandial blood glucose. In addition, HbA1c levels are also influenced by conditions such as hypertriglyceridemia, hyperbilirubinemia,

and the use of medications such as salicylates and opioids, vitamin E, ribavirin, interferon A, cephalosporins, levofloxacin, penicillin, anti-inflammatory drugs, and quinine. Blood disorders that affect the lifespan of erythrocytes will also impact HbA1c levels. [16] [17].

Bin Rakhis et al. (2022) discuss factors that can influence glycemic control, such as patient-related factors including education level, gender, body mass index, and obesity. In addition, there are clinical factors such as the duration of suffering from Type 2 diabetes, fasting blood glucose levels, and hypertension that also impact glycemic control. Other factors related to treatment, such as the number of antidiabetic medications consumed, the duration of treatment, and the diabetes treatment regimen, as well as lifestyle factors, have an impact on glycemic control, such as adherence to medication and exercise. According to Yahaya et al. (2023), glycemic control is influenced by education, medication adherence, alcohol consumption, duration of treatment, and physical activity [18][19].

One of the key strengths of this study is patients in this study received the same diabetes therapy. Inclusion criteria are patients who have been taking oral antidiabetic drugs combined with biguanide and sulfonylurea regularly for at least 3 months. These criteria are expected to reduce bias towards glycemic control that can be influenced by various therapies. In another study, the research sample included all Type 2 DM patients with various types of Type 2 DM management. The research sample of Abubaker et al (2022) was all Type 2 DM patients, whether using insulin or not, while in the research of Salih et al (2021), the sample used in their study was Type 2 DM patients who were treated with diet alone and those combined with oral antidiabetics. This will affect the glycemic control studied because several factors play a role in achieving glycemic control [3][18][20].

The limitations of this study are that it did not assess other factors that may affect serum 25-hydroxyvitamin D levels such as vitamin D receptors, skin surface area exposed to sunlight per day, and intake of foods containing vitamin D as well as other factors that may affect fasting blood glucose levels, two-hour postprandial blood glucose levels and HbA1c levels such as genetic factors, composition and amount of calorie intake, physical activity and medication compliance.

5. Conclusion

There is a moderate negative correlation between serum 25-hydroxyvitamin D levels and fasting blood glucose, a weak negative correlation between serum 25-hydroxyvitamin D levels and blood glucose levels 2 hours postprandial, and a moderate negative correlation between serum 25-hydroxyvitamin D levels and HbA1c in patients with Type 2 Diabetes Mellitus. There is a lower median level of serum 25-hydroxyvitamin D in patients with Type 2 Diabetes Mellitus.

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Conflict interest

The authors declare no conflict of interest

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