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The Association Between Vitamin D Levels and Glycemic Control Parameters in Elderly Populations

Shirly Gunawan[1*], Robert Kosasih[1], Bryan Anna Wijaya[2],Alexander Halim Santoso[3], Yohanes Firmansyah[4]

[1]Department of Pharmacology, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[2]Medical Profession Program, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[3]Department of Nutrition, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[4]Department of Physiology, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
*E-mail: shirlyg@fk.untar.ac.id

Abstract

Diabetes is common in the elderly, and routine monitoring of HbA1c and blood glucose is required to prevent complications. HOMA-IR assesses insulin resistance, while vitamin D may influence glucose metabolism by enhancing insulin sensitivity. This study examines the relationship between vitamin D levels and glycemic control, including HbA1c, fasting glucose, insulin, and HOMA-IR. This cross-sectional observational study included 91 elderly residents of Panti Wreda Bina Bhakti, excluding those with acute illness or chronic conditions affecting Vitamin D and diabetes parameters. All participants provided informed consent. The study measured serum Vitamin D-25(OH) and insulin using ELISA, while fasting blood glucose and HbA1c were assessed via blood chemistry analysis. HbA1c control was categorized as good (<6.5%), moderate (6.5–8.0%), or poor (>8%). HOMA-IR was calculated using a standard formula. Pearson correlation analysis determined the relationship between Vitamin D levels and glycemic parameters, with statistical significance at $p < 0.05$. The correlation analysis found a significant negative relationship between vitamin D levels and HbA1c (r: -0.211; p: 0.044). However, no significant correlation was found between vitamin D and fasting blood glucose levels (r: 0.057; p: 0.590), insulin (r: -0.083; p: 0.432), and HOMA-IR (r: -0.040; p: 0.704). Vitamin D levels show a significant negative correlation with HbA1c in the elderly, indicating its role in long-term glycemic control. Although no significant relationship was found with other glycemic parameters, these findings suggest that monitoring vitamin D status may be considered as part of a more comprehensive approach to diabetes management in the geriatric population.

Keywords: Diabetes, Elderly, Glycemic Control, Vitamin D

1. Introduction

Diabetes mellitus (DM) is a long-term metabolic disease marked by persistently elevated blood glucose levels and increased glycated hemoglobin (HbA1c) concentrations.[1] This condition begins with impaired insulin secretion by the pancreas, insulin resistance in target tissues, or a combination of both. The current global prevalence of DM stands at 537 million cases, projected to increase to 643 million by 2030 and 783 million by 2045, posing a significant social and economic challenge as the disease becomes epidemic and endemic.[2,3]

The elderly are individuals aged 60 years and above. The proportion of people aged 60 and over is rising globally, comprising 15% of the world's population and estimated to reach 7.5 billion. Approximately 20% of elderly individuals suffer from DM, with many cases remaining undiagnosed.[4,5] The prevalence of DM among the elderly ranges from 18% to 33%, and about 30% experience impaired glucose regulation classified as prediabetes. This indicates a potential increase in future DM cases. Diagnostic screening in the elderly shows that DM and its complications represent a significant health challenge. Diagnosis of diabetes mellitus is confirmed when fasting plasma glucose levels are ≥126 mg/dL (≥7.0 mmol/L), or when 2-hour postprandial/oral glucose tolerance test values reach ≥200 mg/dL (≥11.1 mmol/L).

Likewise, a random plasma glucose of ≥200 mg/dL (≥11.1 mmol/L) accompanied by classic diabetic symptoms meets diagnostic criteria. Furthermore, an HbA1c level of ≥6.5% is also considered diagnostic for diabetes.[6,7]

Unhealthy and unbalanced nutrition patterns influence the development of DM and insulin resistance. Diets high in sugar and saturated fats contribute to metabolic dysfunction. Deficiencies in essential minerals, such as vitamin D, also reduce insulin sensitivity.[8,9] Vitamin D is crucial for maintaining pancreatic beta cells, enhancing insulin synthesis, and reducing insulin resistance. Vitamin D receptors (VDR) are expressed in pancreatic β-cells, where vitamin D influences intracellular calcium homeostasis essential for insulin secretion and enhances the synthesis and stability of this hormone. In addition, vitamin D improves insulin sensitivity in peripheral tissues by modulating insulin receptor expression and attenuating chronic low-grade inflammation that contributes to insulin resistance and β-cell dysfunction.[10,11]

A study conducted by Abubaker et al. (2022) also demonstrated that patients with poor glycemic control exhibited significantly lower vitamin D levels, further supporting the hypothesis that vitamin D deficiency may contribute to impaired glucose homeostasis and the progression of diabetes mellitus.[12] Based on this background, this study analyzes the correlation between vitamin D and several glycemic control parameters, namely HbA1c, fasting blood glucose, and insulin, in elderly patients to elucidate the interaction between vitamin D and glycemic parameters and their potential implications for DM management in the elderly.

2. Method

This observational study employed a cross-sectional design and was conducted among 91 elderly respondents residing at the Panti Wreda Bina Bhakti nursing home. Participants were recruited using predetermined eligibility criteria to ensure the internal validity of the findings. The inclusion criteria comprised elderly individuals aged 60 years or older, in good health, and capable of effective verbal communication. To minimize confounding, individuals who had received medical treatment or vitamin D supplementation within the past three months were excluded. Additional exclusion criteria encompassed the presence of acute conditions such as active infections or severe dehydration, as well as chronic diseases known to affect vitamin D or glucose metabolism, including hepatic, renal, or endocrine dysfunction, thyroid and parathyroid disorders, or a history of cancer and autoimmune diseases requiring immunosuppressive therapy. Finally, individuals unwilling or unable to provide informed consent were excluded from participation.

Each participant who fulfilled the inclusion criteria was provided with an explanation regarding the potential benefits and procedures. Informed consent was obtained by signing a written consent form before participation. Demographic characteristics and relevant health histories were collected through structured, brief interviews. Following this, laboratory investigations were performed, which included venous blood sampling to assess biochemical parameters. Serum Vitamin D-25(OH) levels were measured using the immunoassay method, while glycemic control was evaluated by determining fasting blood glucose and glycated hemoglobin (HbA1c) levels. This study was conducted per ethical standards and has received approval from the

Research Ethics Committee of Tarumanagara University (No. 013-UTHREC/UNTAR/VI/2024).

The independent variable in this study is the serum Vitamin D-25(OH) concentration. In contrast, the dependent variables comprise HbA1c, fasting blood glucose, insulin levels, and the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index. Vitamin D-25(OH) and insulin concentrations were quantified using the Enzyme-Linked Immunosorbent Assay (ELISA) technique, whereas fasting blood glucose and HbA1c levels were determined through standard biochemical analysis. A normal fasting blood glucose level was defined as <100 mg/dL, and HbA1c was categorized as good (<6.5%), moderate (6.5–8.0%), or poor (>8%). The degree of insulin resistance was evaluated by calculating the HOMA-IR index using the following formula. [13]

$$\text{HOMA–IR} = \frac{Insulin\ (\mu IU/mL) \times Glucose(mg/dL)}{405}$$

The Kolmogorov-Smirnov test evaluated the normality of data distribution. Subsequently, statistical analysis was performed with the Pearson correlation test to determine the association between serum vitamin D levels and glycemic control parameters, namely HbA1c, fasting blood glucose, insulin, and HOMA-IR. A p-value of less than 0.05 was considered to indicate statistical significance.

3. Result

This study involved 91 elderly respondents, the majority female (72 people; 79.1%) and males numbering 19 people (20.9%). The mean age of participants was 74.04 years (SD = 7.95). Based on glycemic control parameters, the mean HbA1c level was recorded at 7.63% (SD = 1.41), with glycemic control category distribution: Normal (16.5%), Prediabetic (57.1%), and Diabetic (26.4%). Meanwhile, the mean fasting blood glucose was 86.51 mg/dL (SD = 16.12), with the majority of respondents in the normal category (90.1%) and a small portion showing hyperglycemia (9.9%). Additionally, the mean fasting insulin level of respondents was recorded at 4.77 µIU/mL (SD = 1.7), HOMA-IR value of 1.01 (SD = 0.40), and mean serum vitamin D level of 9.86 ng/mL (SD = 4.77).

Table 1. Respondent Characteristics

Parameter	Category	N	%	*Mean*	SD
Gender					
	Male	19	20.9		
	Female	72	79.1		
Age (years)				74.04	7.95
HbA1c (%)				7.63	1.41
	Normal	15	16.5		
	Prediabetic	52	57.1		
	Diabetic	24	26.4		
Fasting Glucose (mg/dL)				86.51	16.12
	Normal	82	90.1		
	Hyperglycemia	9	9.9		
Insulin (µIU/mL)				4.77	1.7
HOMA-IR				1.01	0.40
Vitamin D (ng/mL)				9.86	4.77

The Pearson correlation analysis revealed a significant inverse relationship between vitamin D levels and HbA1c ($r = -0.211$, $p = 0.044$), suggesting that higher serum vitamin D concentrations are associated with lower HbA1c values. In contrast, no significant correlation was observed between vitamin D and fasting blood glucose, insulin, or HOMA-IR. Further correlation analysis among glycemic parameters demonstrated that fasting blood glucose showed a strong positive association with both HbA1c and HOMA-IR. At the same time, insulin levels exhibited a strong correlation with HOMA-IR ($r = 0.424$, $p < 0.001$) (Table 2, Figure 1).

The Vitamin D-25(OH) levels among elderly respondents aged 60 to 100 exhibited a wide range, from approximately two ng/mL to over 25 ng/mL. Most respondents were female (indicated by red dots) with a relatively heterogeneous distribution of vitamin D levels, spanning from very low values to above 20 ng/mL. Despite fewer subjects in the male group (blue dots), the distribution of vitamin D levels was similar to that of females, with several extreme cases exceeding 20 ng/mL observed, particularly among men aged 70–80 years (Figure 2).

Table 2. Pearson Correlation Analysis between Vitamin D and Glycemic Control Parameters

	HbA1c (r, *p*)	Fasting Glucose (r, *p*)	Insulin (r, *p*)	HOMA-IR (r, *p*)
Vitamin D	–0.211, 0.044*	0.057, 0.590	–0.083, 0.432	–0.040, 0.704

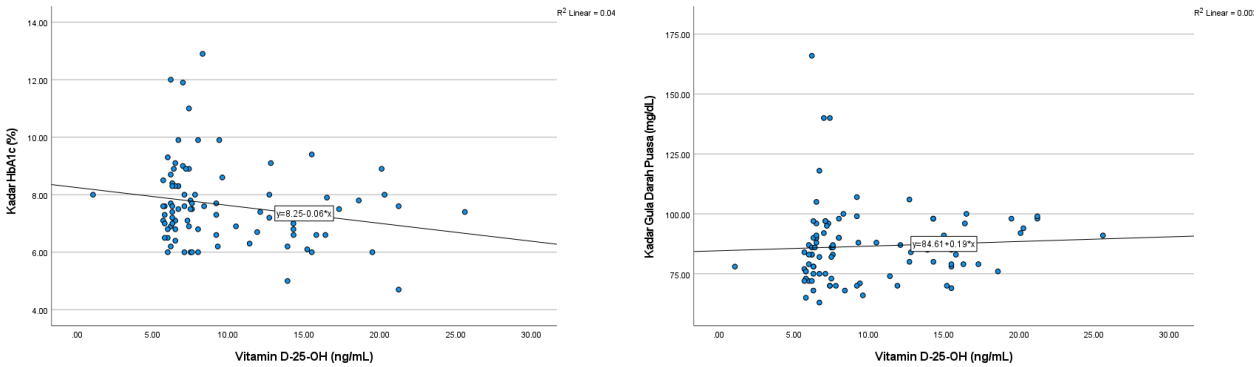

Figure 1. Correlation Between Vitamin D-25(OH) with Fasting Glucose and Glycated Hemoglobin (HbA1c)

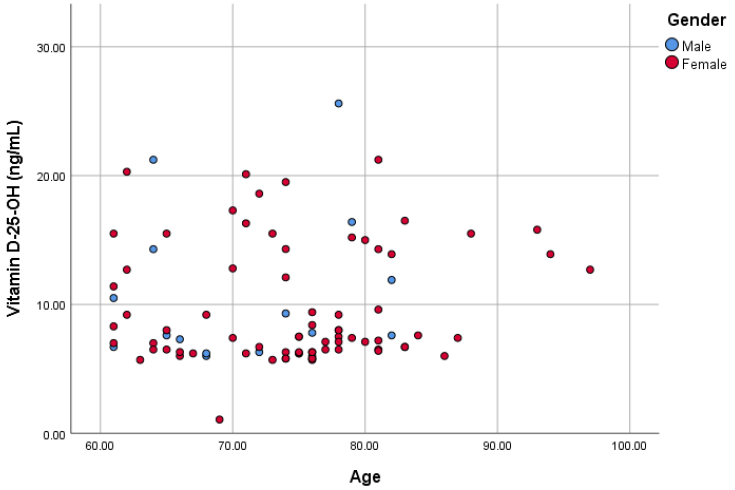

Figure 2. Distribution of Vitamin D Levels by Age and Gender

4. Discussion

This study observed a significant negative correlation between serum vitamin D levels and HbA1c ($r = –0.211$; $p = 0.044$). This finding indicates that higher vitamin D concentrations are associated with lower HbA1c values, reflecting better glycemic control. Physiologically, vitamin D is known to suppress renin expression, an enzyme that catalyzes the formation of angiotensin I, the precursor of angiotensin II, a potent vasoconstrictor that can impair blood flow to metabolically active tissues, such as the liver and skeletal muscle. Excessive activation of the renin–angiotensin–aldosterone system (RAAS) restricts tissue perfusion, thereby limiting oxygen and substrate delivery, ultimately stimulating hepatic gluconeogenesis as a compensatory response to tissue hypoxia. Vitamin D improves tissue perfusion by inhibiting RAAS, reduces excessive gluconeogenic drive, and enhances peripheral glucose utilization. These mechanisms collectively contribute to reducing chronic hyperglycemia, as reflected in lower HbA1c levels.[14,15,16]

These findings are supported by Zhao et al. (2023), who specifically investigated the relationship between Vitamin D-25(OH) concentrations, short-term glucose variability, and HbA1c in 325 patients with type 2 diabetes mellitus. Using continuous glucose monitoring (CGM), the study assessed parameters of glycemic variability, including mean amplitude of glycemic excursions (MAGE), coefficient of variation (CV), glucose sensor standard deviation (SD), and time in range (TIR). This methodological approach enabled a comprehensive evaluation of both long-term glycemic control (HbA1c) and short-term glucose fluctuations, the latter being increasingly recognized as a predictor of diabetic complications. Higher vitamin D levels were significantly associated with lower HbA1c, MAGE, CV, and SD values, alongside significantly higher TIR. Thus, patients with adequate vitamin D status not only demonstrated improved HbA1c but also exhibited more stable daily glucose profiles.[17] Similarly, Felício et al. (2021), in a cohort of 1,576 patients with diabetes, reported that individuals with HbA1c <7% had significantly higher Vitamin D-25(OH) concentrations compared with those with HbA1c ≥7%.[18]

In contrast, no significant correlation was identified between vitamin D levels and fasting plasma glucose ($r = 0.057$; $p = 0.59$). This suggests that variations in vitamin D status among study participants were not directly associated with fasting glucose levels. This finding aligns with a large-scale epidemiological analysis by Hwang et al. (2022), which evaluated 32,943 Korean adults from the Korea National Health and Nutrition Examination Survey (KNHANES). The authors reported no significant association between vitamin D status and fasting glucose in diabetic and non-diabetic populations.[19] Conversely, Taloyan et al. (2023), in a Swedish cohort of 510 individuals of Middle Eastern and Swedish descent without diabetes, identified a weak but significant positive correlation between Vitamin D-25(OH) and fasting glucose in the Middle Eastern subgroup ($\rho = 0.20$; $p = 0.002$), whereas no significant association was observed among those of Swedish ancestry.[20]

From a mechanistic perspective, vitamin D may influence glucose homeostasis through several pathways. First, it enhances peripheral tissue sensitivity to insulin, facilitating more efficient glucose uptake. Second, it supports pancreatic β-cell function, particularly insulin biosynthesis and secretion, essential for maintaining fasting glucose stability. Third, vitamin D exerts

immunomodulatory effects by attenuating systemic inflammation, a process known to impair insulin signaling and worsen insulin resistance. Consequently, sufficient vitamin D levels may protect against dysregulated fasting glucose while maintaining overall metabolic balance.[21]

Furthermore, no significant correlations were identified between vitamin D levels and serum insulin concentrations ($r = –0.083$; $p = 0.432$), nor between vitamin D and insulin resistance assessed by HOMA-IR ($r = –0.040$; $p = 0.704$). These results suggest that, in elderly populations, vitamin D status does not directly influence insulin secretion or insulin sensitivity as reflected by HOMA-IR indices. While vitamin D physiologically contributes to β-cell regulation via the VDR–PDX-1 axis, our findings imply that in elderly individuals, other dominant factors, such as age-related β-cell dysfunction, oxidative stress accumulation, chronic low-grade inflammation, and genetic or environmental determinants, may outweigh the contribution of vitamin D. Additionally. However, β-cells can locally convert vitamin D into its active form; this capacity may be insufficient to counterbalance systemic deficiency or age-related pathophysiological changes. Thus, the relationship between vitamin D and insulin dynamics in elderly populations appears complex and may not be readily captured through serum measurements of vitamin D, insulin, or HOMA-IR. These results are consistent with previous studies reporting heterogeneous vitamin D and insulin secretion findings in aging cohorts.[22-25]

Several limitations of this study warrant consideration. First, the cross-sectional design precludes establishing causal inferences. Although a correlation between vitamin D and HbA1c was observed, it remains unclear whether vitamin D deficiency contributes to impaired glycemic control, poor glycemic control reduces vitamin D status, or unmeasured confounders influence both. Second, the relatively small sample size (91 participants) and gender imbalance (79.1% female) may limit generalizability. Moreover, the study sample was drawn from a single nursing home, which may not represent community-dwelling elderly or other populations. Third, no adjustment was made for key potential confounders such as body mass index, physical activity, sunlight exposure, or nutritional intake, all of which are highly relevant to both vitamin D status and glucose metabolism. These limitations suggest that the findings should be interpreted cautiously and validated in larger, more diverse cohorts with comprehensive control of confounding variables.

5. Conclusion

Vitamin D levels were inversely correlated with HbA1c, but not fasting glucose, insulin, or HOMA-IR. These findings suggest that vitamin D may contribute to improving long-term glycemic control. From a clinical perspective, monitoring vitamin D status in elderly individuals with diabetes or prediabetes may be considered part of a comprehensive approach to glycemic management. Vitamin D supplementation could benefit those with a deficiency, particularly in the context of HbA1c control. However, given the complexity of metabolic interactions in the aging population, such interventions should be individualized and accompanied by regular monitoring of both glycemic parameters and vitamin D status. Further prospective and interventional studies are warranted to elucidate the impact of vitamin D supplementation on glycemic control and clinical outcomes in this population.

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