JPPIPA 11(10) (2025)

Jurnal Penelitian Pendidikan IPA

http://jppipa.unram.ac.id/index.php/jppipa/index

Correlation between 25(OH)D and HbA1c with Diabetic Nephropathy

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Received: July 17, 2025
Revised: August 29, 2025
Accepted: October 25, 2025
Published: October 31, 2025

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DOI: 10.29303/jppipa.v11i10.12692

Abstract: Diabetes mellitus (DM) is a metabolic disorder characterized by hyperglycemia due to impaired insulin secretion or action. DM patients may develop diabetic nephropathy (ND), marked by decreased glomerular filtration rate (eGFR), also influenced by hypertension. Vitamin D in its active form, 25-hydroxyvitamin D (25(OH)D), regulates insulin secretion and supports pancreatic beta cell survival. The relationship between 25(OH)D, HbA1c, and ND needs evaluation to predict ND earlier. To analyze the correlation between 25(OH)D and HbA1c levels with ND measured by eGFR in DM patients at Diponegoro National Hospital, Semarang. A cross-sectional study of 82 DM patients. 25(OH)D levels were measured by Fluorescent Immunoassay (FIA) and HbA1c by High-Performance Liquid Chromatography (HPLC). eGFR was calculated from creatinine using the CKD-EPI formula. Data analysis used Pearson and regression tests ($p<0.25$). A weak positive correlation was found between HbA1c and eGFR ($p=0.002$, $r=0.307$) and a very weak negative correlation between 25(OH)D and eGFR ($p=0.147$, $r=-0.117$). The combined influence of HbA1c, 25(OH)D, and blood pressure yielded $R^2=0.21$. Higher HbA1c increases eGFR, while lower 25(OH)D also associates with higher eGFR. eGFR is influenced by HbA1c, 25(OH)D, and blood pressure by 21%, with other factors explaining the remainder.

Keywords: 25(OH)D; Diabetic nephropathy; eGFR; HbA1c

Introduction

Diabetes mellitus (DM) is a group of metabolic diseases with the characteristics of hyperglycemia that occurs due to abnormalities in insulin secretion, insulin action, or both. Insulin resistance in muscle and liver cells, as well as pancreatic beta cell failure, have been known to be involved in the central damage pathophysiology of type 2 DM. Other organs that are also involved in type 2 DM are adipose tissue (increased lipolysis), gastrointestinal (incretin deficiency), alpha pancreatic cells (hyperglucagonemia), kidneys (increased glucose absorption), and muscles (insulin resistance), which also play a role in causing impaired glucose tolerance (Soelistijo, 2019).

DM is the direct cause of death out of 1.5 million deaths in the world in 2019 and 48% of these deaths are due to a diagnosis of DM before the age of 70. The death rate from DM increased by 13% in low-income countries. (WHO, 2023)The report on the results of Basic Health Research (Riskesdas) in 2018 showed an increase in the prevalence of DM to 8.5% or around 20.4 million people in Indonesia diagnosed with DM. The increase is in line with the increase in obesity which is one of the risk factors for diabetes (Soelistijo, 2019).

DM complications are divided into microvascular and macrovascular complications. Microvascular complications include neuropathy, nephropathy, and retinopathy, while macrovascular complications include cardiovascular disease, stroke, and peripheral artery

How to Cite:
Hendrianingtyas, M., & Yuniarahmi, D. P. (2025). Correlation between 25(OH)D and HbA1c with Diabetic Nephropathy. *Jurnal Penelitian Pendidikan IPA*, *11*(10), 260–269. https://doi.org/10.29303/jppipa.v11i10.12692

disease (Papatheodorou et al., 2018). Research shows that about 30-40% of DM patients will develope diabetic nephropathy. The exact cause is not yet known for sure, but it is likely caused by insulin resistance, genetics, hyperglycemia, and autoimmune processes (Varghese, 2023).

Diabetic nephropathy is the most common cause of chronic kidney failure that requires kidney replacement therapy. Examinations that can be done to determine the presence of ND are by examining albuminuria and estimating the glomerular filtration rate (eGFR). Pathological changes in the kidneys are irreversible if albuminuria has occurred and eGFR decreased (<60 mL/min/1.73 m^2) which can later develop into chronic kidney failure. Chronic kidney failure in patients with DM is closely related to increased cardiovascular risk and poor prognosis (Powers, 2022).

The difference in diabetic nephropathy that occurs in type 1 DM and type 2 DM is in albuminuria which can be found when the diagnosis of type 2 DM is established. This shows that long asymptomatic periods and hypertension often play a role in the occurrence of albuminuria and decreased glomerular filtration rate. Albuminuria in type 2 DM needs to be considered as it can occur due to secondary factors, including hypertension, congestive heart failure, prostate disease, or infection (Powers, 2022).

Vitamin D in active form 25-hydroxyvitamin D (25(OH)D) or calcidiol is a good indicator in knowing the status of vitamin D. There are two types of vitamin D, namely vitamin D3 (cholecalciferol) and Vitamin D2 (calciferol). Vitamin D3 undergoes two hydroxylation processes in the body for activation. First, the hydroxylation process occurs in the liver and converts vitamin D into 25-hydroxyvitamin D, often known as calcidiol is one of the active forms or metabolites of vitamin D. The second process occurs mainly in the kidney and forms an active physiological 1,25-dihydroxyvitamin D, also known as calcitriol (Molina, 2023).

Vitamin D can improve beta cell survival by way of inactivation of nuclear factor-κB (NF-κb) and the effects of cytokines. Vitamin D can also affect insulin resistance indirectly through the renin-angiotensin-aldosterone system (RAAS). Angiotensin II inhibits the action of insulin in vascular muscle and skeletal muscle tissue leading to impaired glucose uptake. Vitamin D suppresses the formation of local pancreatic renin and RAAS (Delrue et al., 2022; Huang et al., 2023).

Vitamin D deficiency is an independent risk factor for the incidence of DM. Vitamin D deficiency has been shown to have an association with impaired insulin secretion in rat pancreatic beta cells. In contrast, insulin secretion mediated by glucose improved after vitamin D supplementation (Huang et al., 2023).

A previous study by Pinky et al. (2023) found that there was a relationship between HbA1c levels of ≥ 7% and ureum and creatinine levels in DM patients which is a sign of diabetic nephropathy (Pinky, 2023). However, the research conducted by Tarawifa, et al. (2020) obtained the results that there was no significant relationship between HbA1c levels and the risk of diabetic nephropathy assessed with microalbuminuria (Tarawifa et al., 2020). Studies that analyze the relationship between HbA1c as glycemic control and vitamin D levels in diabetic nephropathy have not been widely conducted (Ali et al., 2019; Felício et al., 2021; Xiao et al., 2016). Examined the relationship between vitamin D status and albuminuria in type 2 DM patients with the results that there was no significant relationship between vitamin D deficiency and albuminuria (Indra et al., 2017). From the results of previous studies, the results were different from one another, so researchers were interested in assessing the relationship between 25(OH)D and HbA1c levels and diabetic nephropathy based on eGFR values.

Method

This study is analytical observational research using a cross-sectional approach. The research was conducted in February – April 2024 at the Diponegoro National Hospital Semarang. The inclusion criteria in this study were >18-year-old DM patients who perform routine tests at the hospital, willing to participate in the study, and signed the informed consent form. The exclusion criteria of this study are patients who have experienced kidney failure or taken vitamin D supplements before the study. The research subjects were taken by consecutive sampling with a total of 82 subjects. This research has received ethical clearance approval from the Health Research Ethics Commission, Faculty of Medicine, Diponegoro University with No: 122/EC/KEPK/FK-UNDIP/IV/2024.

The research was conducted in several stages. First, ethical clearance approval and administrative permits were obtained before subject recruitment. Second, subjects who met the inclusion and exclusion criteria were recruited consecutively and asked to sign the informed consent form. Third, data collection was performed, including patient characteristics, laboratory examinations of 25(OH)D, HbA1c, and creatinine levels for eGFR calculation. Finally, all collected data were processed and analyzed statistically.

The level of 25(OH)D was checked using the Fluorescence Immunoassay (FIA) method. HbA1c levels were measured using the NGSP (National Glycohemoglobin Standardization Program) High-Performance Liquid Chromatography (HPLC) method. The eGFR value was obtained by manual calculation of

creatinine levels using the CKD-Epi (Chronic Kidney Disease Epidemiology Collaboration Formula) formula.

The statistical analysis was performed using IBM SPSS 22 software. The data were analyzed using the Pearson test and continued with the regression test. P value <0.25 was considered statistically significant.

Result and Discussion

Baseline Characteristics of Study Subjects

A total of 82 adult patients with diabetes mellitus (DM) were enrolled. Mean eGFR was 73.88 ± 22.15 ml/min/1.73 m² (median 76, range 32–129). Mean HbA1c was 8.17 ± 2.01% (median 7.95, range 5.0–14.1), indicating overall poor glycemic control in the sample. Mean systolic and diastolic blood pressures were 134.84 ± 17.83 mmHg and 83.41 ± 7.07 mmHg, respectively. Mean serum 25(OH)D level was 14.49 ± 7.96 ng/mL (median 13.34, range 4.54–36.46), consistent with vitamin D deficiency (<20 ng/mL).

Table 1. Baseline Subject Characteristic

Variable (n=82)	Mean (± SD)	Median (Min – Max.)
eGFR (ml/min/1.73 m2)	73.88 ± 22.15	76.00 (32 – 129)
HbA1c level (%)	8.17 ± 2.01	7.95 (5.0 – 14.1)
Systolic blood pressure (mmHg)	134.84 ± 17.83	131.5 (110 – 234)
Diastolic blood pressure (mmHg)	83.41± 7.07	80 (70 – 104)
25(OH)D level (ng/ml)	14.49 ± 7.96	13.34 (4.54 – 36.46)

Distribution of Biochemical Parameters

HbA1c: min 5.0% — max 14.1% (mean 8.17 ± 2.01%).
25(OH)D: mean 14.49 ± 7.96 ng/mL (deficient range).
eGFR (CKD-EPI): min 32 — max 129 ml/min/1.73 m² (mean 73.88 ± 22.15).

Correlation Analysis with eGFR

Pearson correlation results (n = 82):
HbA1c: r = 0.307, p = 0.002 (weak positive correlation).
Systolic BP: r = 0.220, p = 0.023 (weak positive correlation).
Diastolic BP: r = 0.146, p = 0.095 (weak positive, not statistically strong).
25(OH)D: r = -0.117, p = 0.147 (very weak negative correlation).

Table 2. Relationship between Biochemical and Clinical Parameters with eGFR

Variable (n = 82)	p	r
HbA1c	0.002	0.307
Systolic blood pressure	0.023	0.220
Diabolic blood pressure	0.095	0.146
Up to 25(OH)D	0.147	-0.117

p <0.25 is considered statistically significant

Multivariable Linear Regression

All variables with p < 0.25 entered into linear regression. The final model:

eGFR = -11.700 + 4,236 (HbA1c) + 0,301 (systole) + 0,237 (diastole) + (-0,647)(25(OH)D level)

Model R^2 = 0.21, indicating these variables explain 21% of the variance in eGFR; 79% remains unexplained by the model.

Subject Characteristics and Clinical Profile

This study involved 82 adult DM patients with an average eGFR of 73.88 ± 22.15 ml/min/1.73 m², reflecting mild-to-moderate decline in renal function. The mean HbA1c value of 8.17 ± 2.01% indicates poor glycemic control, consistent with the fact that many DM patients in clinical practice often fail to achieve optimal glycemic targets. The average vitamin D level was 14.49 ± 7.96 ng/mL, which is categorized as deficiency (<20 ng/mL), showing that vitamin D insufficiency is a common problem among DM patients. These findings align with previous studies by Li et al. (2020), who reported lower 25(OH)D levels in DM patients compared to non-DM individuals. The coexistence of poor glycemic control, hypertension, and vitamin D deficiency illustrates a complex interplay of metabolic and vascular disturbances that contribute to the progression of diabetic kidney disease (DKD).

Vitamin D deficiency is widely prevalent among patients with type 2 diabetes mellitus (T2DM), with studies reporting rates ranging from 63.5% to 91.1% worldwide(Anyanwu et al., 2020). A 2023 cross-sectional study in India found that 74.14% of T2DM patients were deficient, while a 2024 study in Saudi Arabia reported an even higher prevalence of 83.7%, both showing strong associations with obesity, poor glycemic control, and elevated HbA1c levels(Alzahrani et al., 2024; Vijay et al., 2023). A pooled global analysis further estimated the prevalence at around 64.2%, underscoring the global burden of vitamin D deficiency in T2DM (Taderegew et al., 2023). This global burden highlights the importance of evaluating vitamin D status alongside other metabolic factors, particularly glycemic control, which remains suboptimal in many populations.

Poor glycemic control, reflected by elevated HbA1c levels, remains highly prevalent in low and middle-income countries (LMICs), with studies showing that more than half of T2DM patients fail to achieve HbA1c <7% (Adjei et al., 2025; Taderegew et al., 2023). Socioeconomic barriers such as limited healthcare access, low education, financial hardship, and treatment inertia are major contributors, with rural residence further exacerbating disparities (Adjei et al., 2025; Gomes et al., 2022; Mehrabbeik et al., 2024). Other

predictors include longer diabetes duration, inadequate disease knowledge, and complications, while mental health factors like depression play a less consistent role (Kamruzzaman et al., 2025; Nguyen et al., 2025). Together, persistent poor glycemic control and vitamin D deficiency contribute to a constellation of baseline risk factors that accelerate diabetic nephropathy progression.

Baseline characteristics play an important role in predicting the risk of diabetic nephropathy (DN) among patients with diabetes mellitus. Poor glycemic control, hypertension, longer diabetes duration, albuminuria, reduced baseline eGFR, and older age have consistently been reported as major predictors of DN progression (Baek et al., 2021; Natesan & Kim, 2021; Radcliffe et al., 2017; Yokoyama et al., 1998). Additional risk factors such as male sex, obesity, smoking, genetic predisposition, and the presence of other microvascular complications (e.g., retinopathy) further contribute to the likelihood of DN development (López-Revuelta et al., 2014; Natesan & Kim, 2021).

Association of HbA1c with eGFR

The present study found a weak positive relationship between HbA1c and eGFR (p=0.002; r=0.307). This suggests that higher HbA1c is correlated with relatively preserved eGFR in this cohort, which may appear paradoxical but has also been reported in previous studies. Akazawa et al. (2022) demonstrated a similar positive correlation, proposing that in the early stages of diabetic nephropathy, hyperfiltration occurs as a compensatory mechanism, thus elevating eGFR despite poor glycemic control. Over time, persistent hyperglycemia leads to glomerular damage, proteinuria, and subsequent decline in eGFR . Other studies, such as by Aniskurlillah (2019), also support the association between HbA1c and renal function in DM patients. Mechanistically, chronic hyperglycemia promotes the accumulation of advanced glycation end products (AGEs), oxidative stress, and activation of profibrotic pathways, which eventually impair renal hemodynamics and structure (Agarwal, 2021). This highlights the importance of understanding hyperfiltration as a transitional stage between early preserved eGFR and subsequent decline.

Hyperfiltration, an early renal abnormality in diabetic nephropathy, is strongly influenced by glycemic status. Elevated HbA1c has been shown to independently increase the risk of hyperfiltration, with acute hyperglycemia directly modulating renal hemodynamics, while early intensive glycemic control may normalize GFR and delay DN progression (Hu et al., 2015; MacIsaac et al., 2017; Rout P, 2025; Troya et al., 2016). Moreover, HbA1c variability even at acceptable mean levels emerges as an additional predictor of faster eGFR decline, underscoring the importance of both long-term control and glucose stability (Lee et al., 2020). These findings are consistent with evidence that HbA1c variability strongly predicts adverse renal outcomes.

Elevated HbA1c levels and greater HbA1c variability are strong predictors of accelerated kidney function decline and progression to ESRD in patients with diabetes (Arnold et al., 2024; C.-L. Lee et al., 2013, 2020; Xu et al., 2022). Notably, even in individuals achieving HbA1c <7%, fluctuations in HbA1c remain independently associated with faster renal deterioration, underscoring the need to target both average glycemic levels and stability for optimal renal outcomes (C.-L. Lee et al., 2020; M.-Y. Lee et al., 2018). This association has been consistently validated in longitudinal cohorts and meta-analyses.

Longitudinal studies consistently demonstrate that both elevated HbA1c and greater HbA1c variability predict accelerated decline in kidney function among patients with T2DM, independent of baseline eGFR (C.-L. Lee et al., 2013, 2020; Okawa et al., 2023). Meta-analyses further confirm that mean HbA1c and its fluctuations are robust predictors of earlier eGFR decline and progression to ESRD, underscoring the clinical importance of maintaining both optimal glycemic levels and stability (Habte-Asres et al., 2022; Y. Zhu et al., 2025).

Association of Blood Pressure with eGFR

This study demonstrated a significant association between systolic blood pressure and eGFR (p=0.023; r=0.220), whereas the association with diastolic blood pressure was weaker and not statistically significant (p=0.095; r=0.146). This finding is consistent with (Truscello et al., 2023), who emphasized the stronger role of systolic compared to diastolic blood pressure in predicting renal function decline in diabetic kidney disease. Elevated systolic blood pressure contributes to glomerular hypertension, endothelial dysfunction, and activation of the renin-angiotensin-aldosterone system (RAAS). Angiotensin II further exacerbates renal injury through induction of fibrosis, oxidative stress, and pro-inflammatory cytokines (Qaz et al., 2022). The weaker role of diastolic pressure may reflect its less direct impact on glomerular hemodynamics compared to systolic load. Effective blood pressure control, particularly systolic pressure, is therefore crucial in slowing down renal decline in DM patients.

Systolic blood pressure (SBP) has been consistently identified as a dominant predictor of diabetic nephropathy (DN) progression, where persistent elevations above 125–130 mmHg markedly increase the risk of renal decline, ESRD, and mortality (Alwakeel et al., 2011; Bakris, 2003; Hata et al., 2023; Kitagawa et al., 2022; Leehey et al., 2005a; Stojceva-Taneva et al., 2007). Evidence from large cohort studies and clinical trials

further supports guideline recommendations to maintain SBP <130/80 mmHg, although excessively low targets (<120 mmHg) may pose cardiovascular risks, highlighting the need for individualized blood pressure management in DN (Grassi et al., 2016; Okada et al., 2013; Van Buren & Toto, 2011).

RAAS activation drives glomerular injury and fibrosis in diabetic nephropathy, while ACE inhibitors and ARBs remain the cornerstone of therapy, with emerging adjuncts such as SGLT2 inhibitors and GLP-1 receptor agonists showing synergistic renoprotection despite safety concerns (Alsalemi et al., 2022; Chawla, 2010; Elendu et al., 2023; Leoncini et al., 2020; Rout P, 2025; Ruggenenti et al., 2010; Zhao et al., 2025).

Blood pressure (BP) control is a cornerstone of managing diabetic nephropathy, with most guidelines recommending a target of <130/80 mmHg to reduce proteinuria, slow kidney disease progression, and lower cardiovascular risk (Sternlicht & Bakris, 2016; Tomlinson et al., 2003; Van Buren & Toto, 2011). Evidence from landmark trials such as IDNT and RENAAL supports this threshold, though excessively aggressive control (<120 mmHg) has been linked to adverse cardiovascular outcomes, indicating a U- or J-shaped relationship between BP and prognosis (Bakris, 2003; Leehey et al., 2005b; Van Buren & Toto, 2011). Recent reviews emphasize the need for individualized targets based on age, comorbidities, and hypotension risk, while highlighting the role of RAAS inhibitors and combination therapies in achieving optimal outcomes (Leehey et al., 2005; Park et al., 2024; Sternlicht & Bakris, 2016; Tomlinson et al., 2003).

Taken together, these findings highlight the pivotal role of systolic blood pressure in determining renal outcomes among patients with diabetes, suggesting that optimal management of hemodynamic load is as crucial as glycemic control in preserving kidney function. This interrelationship warrants further exploration alongside other metabolic and hormonal factors such as vitamin D status, which may exert additional influence on eGFR trajectories.

Association of 25(OH)D with eGFR

The analysis revealed a weak negative relationship between vitamin D levels and eGFR (p=0.147; r=-0.117), which contrasts with several previous studies. X. Zhu et al. (2021) reported a weak positive association between 25(OH)D and eGFR in Chinese DM patients, while (Li & Li, 2020) found that lower vitamin D levels correlated with decreased renal function. The discrepancy in this study may be due to the relatively small sample size, confounding factors such as sun exposure and dietary intake, or the possibility of reverse causality in patients with early nephropathy. Biologically, vitamin D plays a protective role in diabetic nephropathy through inhibition of pro-inflammatory cytokines (IL-1, IL-6, IL-18), regulation of pancreatic beta-cell function and insulin secretion, as well as suppression of RAAS activity (Huang et al., 2023; Xuan et al., 2023). Vitamin D deficiency in DM patients can also result from loss of vitamin D–binding protein (VDBP) due to proteinuria, leading to impaired reabsorption in the proximal tubules. Despite the negative correlation observed in this study, the deficiency status of 25(OH)D in almost all subjects highlights the clinical importance of monitoring and potentially correcting vitamin D levels in DM patients. Beyond the present findings, accumulating evidence has consistently demonstrated that vitamin D deficiency contributes to DN progression.

Vitamin D deficiency has been consistently linked to the progression of diabetic nephropathy (DN), with lower serum levels observed in patients as the disease advances and meta-analyses confirming an increased risk of nephropathy (OR 1.80) (Dean et al., 2023; Derakhshanian et al., 2015). Mechanistically, deficiency exacerbates proteinuria and renal injury through activation of the renin–angiotensin system, inflammation, oxidative stress, and fibrosis (Delrue et al., 2022b; Derakhshanian et al., 2019; Momeni et al., 2016; Souza et al., 2023). While several trials indicate that vitamin D supplementation may reduce proteinuria and slow renal decline, results remain inconsistent, underscoring the need for larger and long-term studies to establish therapeutic efficacy (de Oliveira e Silva Ullmann et al., 2023; Momeni et al., 2016). These protective effects are largely mediated through suppression of the RAAS.

Vitamin D exerts renoprotective effects partly through suppression of the RAAS, where activation of the vitamin D receptor inhibits renin transcription and reduces angiotensin II and aldosterone levels, thereby limiting vasoconstriction, fibrosis, and hypertension (Ajabshir et al., 2014; Forman et al., 2010; Jia et al., 2022; Santoro et al., 2015). Experimental and epidemiological studies consistently demonstrate that vitamin D deficiency promotes RAAS overactivation, leading to glomerular injury, inflammation, and cardiovascular complications (Ajabshir et al., 2014; Jia et al., 2022; Santoro et al., 2015). By modulating RAAS activity, vitamin D may protect against renal inflammation and fibrosis, highlighting its therapeutic potential in diabetic nephropathy and hypertensive kidney disease (Koroshi & Idrizi, 2011).

Elevated urinary vitamin D-binding protein (VDBP) is strongly correlated with albuminuria and tubular injury in diabetic nephropathy (DN) (Fawzy & Abu AlSel, 2018; Maghbooli et al., 2022; Tian et al., 2014), while reduced serum VDBP, partly due to urinary loss, is linked to microalbuminuria, eGFR decline, and diabetic retinopathy (Blanton et al., 2011; Maghbooli et

al., 2022). Emerging evidence suggests urinary VDBP may serve as a sensitive early biomarker for DN progression (Chen et al., 2023; Fawzy & Abu AlSel, 2018), and its loss may impair vitamin D transport, thereby exacerbating RAAS activation and inflammatory pathways in kidney damage (Emini Sadiku, 2025; Maghbooli et al., 2022). In addition, VDBP clearance ratios provide valuable information for detecting subclinical tubular injury before overt renal function decline (Semnani-Azad et al., 2024).

Implications and Limitations

The present findings suggest that HbA1c, systolic blood pressure, and vitamin D status are interrelated factors influencing renal function in DM patients. Although the regression model explained 21% of the variation in eGFR, the remaining 79% indicates that other determinants such as duration of DM, proteinuria, lipid abnormalities, and genetic predisposition also play crucial roles. The study has several limitations: it used a cross-sectional design, included a relatively small single-center sample (n=82), and lacked adjustment for confounding factors such as diet, sun exposure, and physical activity, which affect vitamin D status. Furthermore, renal function was assessed only by eGFR without additional biomarkers (e.g., albuminuria, VDBP), and HbA1c was measured at a single time point, not accounting for glycemic variability. Given these limitations, future multicenter longitudinal studies with larger sample sizes and broader biomarker assessment are warranted to validate these associations and evaluate the potential benefit of vitamin D supplementation in preventing diabetic nephropathy progression.

Conclusion

An increase in the HbA1c value results as an increase in the eGFR value, but the lower the 25(OH)D level, the higher the eGFR value will be. The eGFR value was influenced by the value of HbA1c, 25(OH)D levels, systolic and diastolic blood pressure of 21%, while the rest was influenced by other variables.

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