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The relationship between vitamin D deficiency and erythrocyte sedimentation rate in patients with diabetes

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Background/aim: Vitamin D deficiency in diabetic patients has been shown to be associated with some inflammatory markers. However, its relationship with erythrocyte sedimentation rate (ESR) is still unknown. The aim of this study was to investigate the relationship between vitamin D deficiency and ESR in patients with type 2 diabetes mellitus (T2DM).

Materials and methods: This cross-sectional study was conducted with 294 consecutive patients with T2DM. Serum levels of 25-hydroxyvitamin D, glycemic parameters, lipids, ESR, and C-reactive protein were measured. Patients were evaluated according to 25-hydroxyvitamin D levels as having vitamin D deficiency, vitamin D insufficiency, and vitamin D sufficiency.

Results: ESR was found to be higher in patients with vitamin D deficiency than in patients who were vitamin D-sufficient ($P < 0.001$), and ESR was negatively correlated with 25-hydroxyvitamin D level ($r = -0.265$, $P < 0.001$). HbA1c and postprandial glucose levels were higher in patients with vitamin D deficiency than vitamin D-sufficient patients ($P = 0.005$ and $P = 0.019$, respectively). In receiver operating curve analysis, an ESR value of 14.5 mm/h had 70.1% sensitivity and 50.3% specificity for the prediction of vitamin D deficiency.

Conclusion: The present study revealed that ESR is higher in T2DM patients with vitamin D deficiency than patients with sufficient vitamin D. There was an inverse association between ESR and vitamin D levels. Furthermore, vitamin D deficiency was related to poor glycemic control.

Key words: Blood sugar, dyslipidemia, HbA1c protein, inflammation

1. Introduction

Type 2 diabetes mellitus (T2DM) may cause serious complications such as neuropathy, retinopathy, and nephropathy, and its prevalence is continuing to increase globally (1). The major points in the pathophysiology of this complex disease are impaired insulin secretion, insulin resistance, and systemic inflammation (2). Studies have shown that vitamin D may have an impact on all of these pathophysiological pathways (3,4). It has been found that vitamin D deficiency is related to increased risk of diabetes, impaired insulin secretion, and an increase in insulin resistance by affecting the glucose metabolism (5,6). Furthermore, in many studies of T2DM patients, serum 25-hydroxyvitamin D (25(OH)D) levels and glycosylated hemoglobin (HbA1c) values have been reported to be inversely proportional (7,8).

In the pathogenesis of T2DM, it has been demonstrated that both insulin resistance and beta cell insufficiency are related to inflammatory factors (9,10). On the other hand, it was found that there is alteration in levels of several systemic acute phase reactants in vitamin D deficiency, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor (TNF) (11,12). There are also data implicating vitamin D in the pathogenesis of diabetes through antiinflammatory effects by reducing cytokines (13,14). The erythrocyte sedimentation rate (ESR) is an inexpensive, easily measured, and widely used marker that demonstrates inflammation (15). In T2DM, in spite of a known association between 25(OH)D deficiency and some inflammatory markers, the association of vitamin D with ESR is unclear. The aim of the present study was to evaluate the possible association between vitamin D deficiency and ESR in patients with T2DM.

2. Materials and methods

This was a cross-sectional study conducted in the diabetic outpatient department of our university hospital. A total of 294 consecutive adult patients with T2DM were included between 1 April and 30 May in this study. Exclusion criteria were history of liver disease, chronic renal disease,

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inflammatory disease, parathyroid disease, acute and chronic infection, hypercortisolism, anemia, cancer, hematological diseases, malabsorption, alcoholism, current use of vitamin D or calcium preparations, and pregnancy or breastfeeding. Patients were also excluded if they were using heparin or oral contraceptives that may increase the ESR. T2DM was defined according to World Health Organization criteria (16).

The demographic and clinical features of the patients were recorded. Body mass index (BMI) was calculated by dividing the weight by the square of the height ((kg/m^2).

Blood samples were obtained after an overnight fast. Serum concentrations of 25(OH)D, CRP, glucose, creatinine, albumin, calcium, phosphate, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides were measured using standard laboratory procedures. The postprandial blood glucose concentration was measured 2 h after eating. HbA1c, complete blood count, and ESR of patients were measured as well. ESR (mm/h) was determined by the Westergren method, and 25(OH)D concentrations were measured using the enzyme-linked immunosorbent assay method.

Dyslipidemia was defined according to the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) (17) and patients who were taking lipid-lowering drugs were also accepted as having dyslipidemia. Patients were divided into three groups according to 25(OH)D levels as having vitamin D deficiency (25(OH)D < 20 ng/mL), vitamin D insufficiency (25(OH)D 20–30 ng/mL), and vitamin D sufficiency (25(OH)D ≥ 30 ng/mL) (18). The relationship between vitamin D deficiency and ESR was evaluated within the groups. The study protocol was approved by the institutional review board of our university (approval number: 71522473/050.01.04/102), and all participants provided informed consent before enrollment in the study.

Statistical analyses were performed with SPSS 17.0. Continuous data with normal distribution were expressed as mean and standard derivation (SD), whereas median and 25th–75th percentiles were used for nonnormally distributed variables. Comparison of each group of patients was performed using Kruskal–Wallis and Mann–Whitney U tests. The chi-square test was used to examine differences between categorical variables between groups. Spearman's correlation coefficients were computed to assess correlations between variables. Logistic regression analysis was performed to identify significant independent associates of vitamin D level. The receiver-operating characteristic (ROC) curve was used to determine the optimum cut-off value of ESR in the prediction of vitamin D deficiency. P < 0.05 was considered significant.

3. Results

Of the patients included in the study, 126 (42.1%) were male and the mean age was 56.8 ± 10.3 years. The demographic characteristics of the study population are shown in Table 1. The median value of duration of diabetes was 7 (3–12) years. The median levels of 25(OH)D and ESR were 20.6 (14.3–27.2) ng/mL and 17 (11–28) mm/h, respectively. When the patients were assessed according to 25(OH) D levels, 141 (47.9%) patients had vitamin D deficiency, 97 (32.9%) patients had vitamin D insufficiency, and 56 (19%) patients were vitamin D-sufficient (P < 0.001). ESR was significantly higher in patients with vitamin D deficiency than in vitamin D-sufficient patients (P < 0.001). Furthermore, in patients with vitamin D deficiency, the glycemic control was worse and the rate of dyslipidemia was higher. The characteristics of patients with and without vitamin D deficiency are presented in Table 2.

The correlations of 25(OH)D and ESR with clinical parameters are demonstrated in Table 3. 25(OH)D level was negatively correlated with ESR (r = –0.265, P < 0.001). 25(OH)D and glycemic parameters including fasting glucose, postprandial glucose, and HbA1c were negatively correlated (r = –0.133, P = 0.022; r = –0.146, P = 0.012; and

Table 1. Baseline characteristics of 294 type 2 diabetic patients.

Female, n (%)	168 (57.1)
Age, years	56.8 ± 10.3
Duration of diabetes, years	7 (3–12)
Hypertension, n (%)	175 (59.5)
Ischemic heart disease, n (%)	43 (14.6)
Dyslipidemia, n (%)	203 (69)
Smoking, n (%)	50 (17)
BMI, kg/m^2	31.7 ± 5.6
Weight, kg	82.2 ± 15.5
Height, m	161.4 ± 9.2
HbA1c, %	7.9 ± 2
Creatinine, mg/dL	0.8 ± 0.2
Hemoglobin, g/dL	14.1 ± 1.5
25-OH vitamin D, ng/mL	20.6 (14.3–27.2)
ESR, mm/h	17 (11–28)
CRP, mg/L	3.4 (3.4–5.7)

Values are presented as means ± SD or medians (25th–75th percentiles). BMI: Body mass index, HbA1c: glycated hemoglobin, ESR: erythrocyte sedimentation rate, CRP: C-reactive protein.

Table 2. Characteristics of patients with type 2 diabetes mellitus according to 25-OH vitamin D level.

	Vit-D deficiency (<20 ng/mL)	Vit-D insufficiency (20-30 ng/mL)	Vit-D sufficient (≥30 ng/mL)	P-value
Patients, n (%)	141 (47.9)	97 (32.9)	56 (19)	<0.001
Age, years	57.8 ± 10	56.1 ± 11	55.5 ± 9.8	0.425
Female, n (%)	94 (66.7)	37 (38.1)	37 (66.1)	0.937
Duration of diabetes, years	7 (3–14)	7 (3–13.4)	6 (3–11)	0.557
Dyslipidemia, n (%)	106 (75.1)	63 (64.9)	34 (60.7)	0.044
Hypertension, n (%)	91 (64.5)	53 (54.6)	31 (55.3)	0.232
BMI, kg/m^2	32.5 ± 5.5	30.5 ± 5.8	32.2 ± 5	0.787
HbA1c, %	8.4 ± 2.2	7.6 ± 2	7.4 ± 1.4	0.005
Fasting glucose, mg/dL	170 (133–233)	151 (129–200)	150 (121–195)	0.060
Postprandial glucose, mg/dL	222 (170–300)	207 (170–256)	194 (161–245)	0.019
Total cholesterol, mg/dL	211 ± 45	198 ± 48	188 ± 32	0.001
HDL cholesterol, mg/dL	41 ± 8.4	41.4 ± 10.9	42.2 ± 7.7	0.440
LDL cholesterol, mg/dL	119 ± 33	115 ± 33	106 ± 25.5	0.027
Triglyceride, mg/dL	163 (120–238)	157 (116–220)	127 (94–194)	0,007
25 (OH) vitamin D, ng/mL	14 ± 3.8	24.2 ± 2.6	40.5 ± 10.9	<0.001
Calcium, mg/dL	9.3 ± 0.4	9.2 ± 0.6	9.3 ± 0.5	0.381
Albumin, g/dL	4.3 ± 0.2	4.3 ± 0.3	4.3 ± 0.2	0.657
WBC count, ×10^3/μL	7.75 ± 1.99	7.49 ± 2.14	7.82 ± 1.87	0.114
CRP, mg/L	3.4 (3.4–6.5)	3.4 (3.4–5)	3.4 (3.4–5.7)	0.467
ESR, mm/h	22 (13.5–32.5)	14 (10–23)	15 (10–21)	<0.001

Values are presented as means ± SD or medians (25th–75th percentiles). Vit: Vitamin, BMI: body mass index, HbA1c: glycated hemoglobin, HDL: high-density lipoprotein, LDL: low-density lipoprotein, WBC: white blood cell, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate.

Table 3. Correlation of 25-OH vitamin D and erythrocyte sedimentation rate with clinical and laboratory parameters in patients with type 2 diabetes mellitus.

Variables	25-OH vitamin D (ng/mL)		ESR (mm/h)	
	r	P-value	R	P-value
Age, years	–0.086	0.141	0.266	<0.001
Duration of diabetes, years	–0.079	0.177	0.174	0.003
BMI, kg/m^2	–0.137	0.018	0.262	<0.001
Fasting glucose, mg/dL	–0.133	0.022	0.017	0.775
Postprandial glucose, mg/dL	–0.146	0.012	0.000	0.993
HbA1c, %	–0.166	0.004	0.057	0.337
Total cholesterol, mg/dL	–0.192	0.001	0.075	0.202
LDL, mg/dL	–0.137	0.019	0.036	0.548
Triglyceride, mg/dL	–0.123	0.035	0.102	0.085
25-OH vitamin D, ng/mL	-	-	–0.265	<0.001
ESR, mm/h	–0.265	<0.001	-	-
CRP, mg/L	–0.002	0.969	0.324	<0.001
WBC count, ×10^3/μL	0.030	0.610	0.122	0.037

ESR: Erythrocyte sedimentation rate, BMI: body mass index, HbA1c: glycated hemoglobin, LDL: low-density lipoprotein, CRP: C-reactive protein, WBC: white blood cell.

r = –0.166, P = 0.004, respectively). Moreover, there was a negative correlation between 25(OH)D and lipid parameters such as total cholesterol, LDL cholesterol, and triglyceride (r = –0.192, P = 0.001; r = –0.137, P = 0.019; r = –0.123, P = 0.035, respectively). Multivariate logistic regression analysis of clinical and laboratory predictors for vitamin D level revealed that ESR and HbA1c were significant independent predictors (OR = –0.205, 95% CI = –0.282 to –0.076, P = 0.001 and OR = –0.131, 95% CI = –1.380 to –0.085, P = 0.027, respectively). ROC curve analysis suggested that the optimum ESR cut-off point for the development of vitamin D deficiency was 14.5 mm/h, with a sensitivity and specificity of 70.1% and 50.3%, respectively (area under the curve = 0.659, P < 0.001) (Figure).

4. Discussion

This study demonstrates that patients with T2DM who were vitamin D-deficient had higher ESR, poor glycemic control, and a greater prevalence of dyslipidemia. Additionally, in the present study, we found a negative correlation between 25(OH)D level and both ESR and glycemic parameters, i.e. fasting glucose, postprandial glucose, and HbA1c.

The pathophysiology of T2DM is complex and several different interrelated mechanisms are involved, which are still not precisely understood (2). Accruing evidence has shown that vitamin D deficiency and inflammation play important roles in this chronic process through several pathways (19,20). Some previous studies have demonstrated that vitamin D deficiency is related to insulin resistance, impaired insulin secretion, and T2DM development (3,5,7). Furthermore, similar to our results, earlier studies reported that there is an inverse correlation between plasma 25(OH)D level and blood glucose in T2DM patients (7,8).

There are various data regarding how vitamin D affects glucose metabolism in patients with diabetes (3,21). In some studies, it has been demonstrated that there are

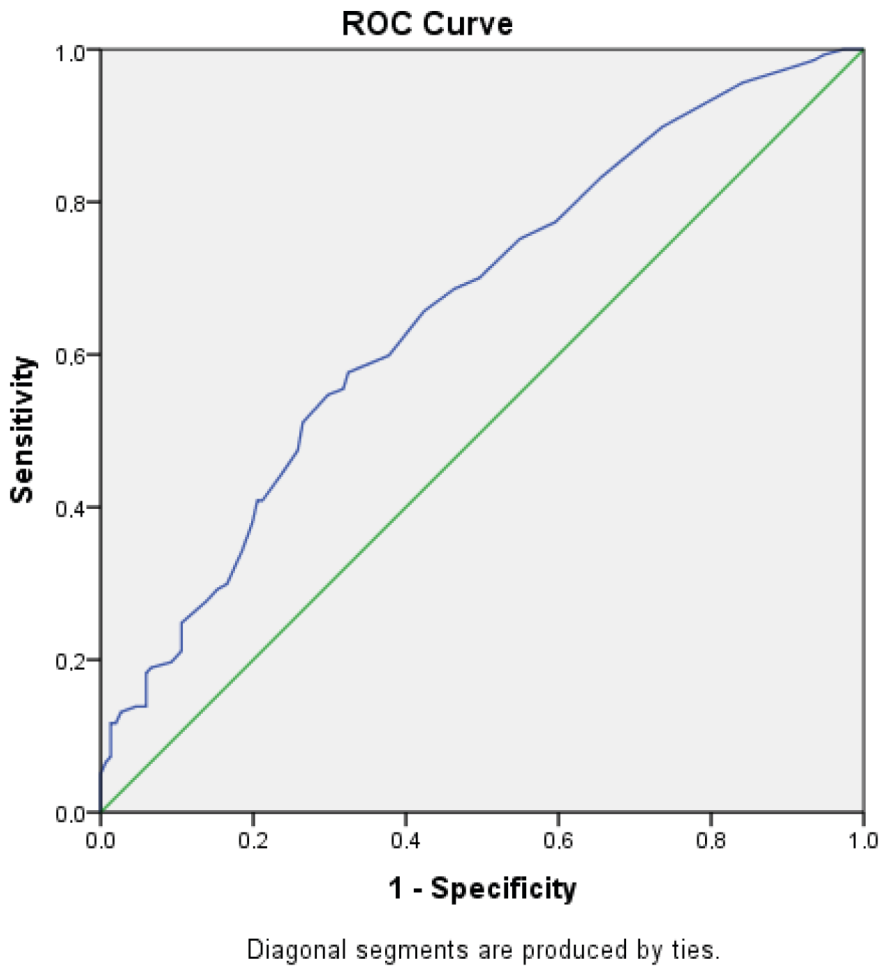

Figure. Receiver operator characteristic curve analysis of erythrocyte sedimentation rate for vitamin D deficiency.

vitamin D receptors in pancreatic beta cells, liver, skeletal muscles, and adipose tissue, and that vitamin D affects insulin synthesis, secretion, and sensitivity through these receptors (3,22,23). In support of this relationship, Bland et al. noted that pancreatic beta cells also possess the 1-alpha-hydroxylase enzyme that converts 25(OH)D to its active form (24). It has been suggested that vitamin D can be effective on insulin metabolism by stimulating the insulin receptors in peripheral tissues, which is important for insulin resistance and adjusting intracellular cytosolic calcium (3,19). Calcium is needed for insulin to act on target tissues (3,19). Calcium depletion in these tissues secondary to vitamin D deficiency can lead to peripheral insulin resistance (19). On the other hand, it has been reported in some studies that the decrease in insulin sensitivity in vitamin D deficiency can be due to increased parathyroid hormone secretion (3,19).

The available data demonstrate that in the pathophysiology of T2DM systemic inflammation is one of the important factors of beta cell dysfunction, beta cell apoptosis, and development of insulin resistance in peripheral tissues (9,10). There are conflicting results about whether there is a positive relationship between vitamin D deficiency and some inflammatory markers or not (11–14). In some studies, an increase in some inflammation markers such as TNF, CRP, IL-6, and IL-8 has been observed in T2DM (9,10). In our study, we demonstrated for the first time that ESR is higher in patients with vitamin D deficiency. With regard to vitamin D and inflammation interaction, it has been postulated that vitamin D may increase insulin resistance by decreasing some inflammatory cytokines and stimulating the glucose transport (3,19,21). Moreover, Giulietti et al. reported a decrease in some inflammatory cytokines such as TNF-alpha, IL-1, IL-6, and IL-8 after administration of vitamin D (25).

In the present study we found higher levels of total cholesterol, LDL cholesterol, and triglycerides in patients with vitamin D deficiency. In addition, there was an inverse correlation between serum 25(OH)D and lipid parameters and BMI. Dyslipidemia is one of the most important factors for cardiovascular disease and a common cause of morbidity and mortality in diabetic patients (26). Hyperglycemia and dyslipidemia may affect each other causally (27). Furthermore, similar to our study, low 25(OH)D levels were associated with obesity and dyslipidemia in previous studies (27,28). There are data demonstrating that vitamin D deficiency may influence dyslipidemia through various mechanisms such as insulin secretion, lipotoxicity, gastrointestinal calcium absorption, inflammation, and increase in the parathyroid hormone level (27). However, the pathophysiology of diabetic dyslipidemia is yet to be fully understood (27).

The present study revealed that ESR was positively correlated with age, duration of diabetes, and BMI. In addition, ESR was positively correlated with inflammation markers such as CRP and white blood cell count. ESR is a simple and cheap laboratory test used as a nonspecific indicator of acute phase response (15). It has been demonstrated that ESR increases with age every 5 years by 0.85 mm/h; however, the reason for this increase is unclear (15). At the same time, an increase in ESR with obesity has been reported in previous studies (15). Similar to our study, Colombet et al. reported that there is a positive relationship between simultaneously measured ESR and CRP (29). Analyses of the First National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (NHANES I) data have demonstrated that ESR is a predictor for the risk of coronary heart disease and stroke incidence among a subset of adults (30,31). However, there are sparse data about the relationship of ESR with diabetes and vitamin D level.

Our study has some limitations. First, our research included a relatively small sample size and study patients were from only our country. Second, the research design of current study was cross-sectional. An interventional research study including the comparison of pretreatment and posttreatment vitamin D could be designed.

In conclusion, our study revealed that ESR is higher in vitamin D-deficient T2DM patients than those with sufficient vitamin D levels, and there is an inverse correlation between ESR and vitamin D levels. Furthermore, in the present study, vitamin D-deficient patients had poor glycemic control and higher levels of total cholesterol, LDL cholesterol, and triglycerides. Further studies may expand our understanding of how inflammation, vitamin D, glycemic control, and dyslipidemia interact in patients with T2DM.

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