



A study assess the correlation between microalbuminuria and 25-(OH) vitamin D levels in patients with type 2 DM at Index Medical College Hospital, Indore

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Introduction

VD is a steroid hormone that plays a significant role in the development of type 2 diabetes mellitus by regulating adipogenesis, protecting pancreatic B cells, as well as improving insulin resistance in muscle myocytes (myocytes). Impaired glucose tolerance is a common complication of VD deficiency, which is common in the population at large. It has been demonstrated in animal studies that a lack of 25-(OH) VD causes a reduction in insulin synthesis and secretion.

Ultrasound-detected microalbuminuria ranges from 31 to 299 mg/24 hours, while macroalbuminuria exceeds 300 mg/24 hours is classified as normoalbuminuric (UA), according to the American Society of Nephrology.

Patients with diabetes are more likely to develop end-stage renal disease as a result of diabetic nephropathy than those without the condition, and it is associated with increased mortality and morbidity. According to previous human studies, albuminuria rates rose as VD levels dropped. VD is known to inhibit renin biosynthesis, which contributes to the development of chronic renal failure. A severe 25-(OH) VD deficiency has been observed in patients with type 2 diabetes and renal failure. In a study of 4330 people with type 2 diabetes who participated in a 5-year follow-up study, low VD status was found to be a significant predictor of an increase in the urine albumin to creatinine ratio.

Microvascular complications in diabetic patients may be linked to VD levels, according to research. VD and microalbuminuria have not been studied in relation to each other in the literature. Diabetic micro complications and D vitamin levels have been linked in studies, but the results are conflicting. VD levels and microalbuminuria were examined in this study's primary goal.

Methodology

This one-year study was approved by the Index Medical College, Indore, Ethical Committee. Following written informed consent, we included in the study 200 patients aged 40–60 who had been diagnosed with type 2 diabetes mellitus for at least 10 years and were receiving treatment with oral anti-diabetic agents or insulin therapy and were admitted to our hospital's diabetes outpatient clinics between February and May of 2018.

The patients were subjected to a physical examination and measured to determine their BMI (kg/m²). After at least 8 hours of fasting, serum VD, fasting blood glucose, HbA1c, creatinine, total cholesterol, LDL-cholesterol, HDL-cholesterol, and triglycerides were measured. High-performance liquid chromatography (HPLC) was used to measure serum VD levels. It was determined using an enzymatic method and an HPLC-UV detector for HbA1c. Original measurement kits and an Abbott-Aeroset auto-analyzer were used to measure lipid levels. The Friedewald equation was used to estimate LDL cholesterol.

Two subsets of patients were subjected to the testing process. Patients in the first group had VD levels that were too low (10–30 ng/mL), while patients in the second group had levels that were too high (10 ng/mL). Both groups were tested for microalbuminuria levels. Continuous variables were described through the use of descriptive statistics (mean, standard deviation, minimum, median, and maximum). Mann-Whitney U and Student's t-test were used to compare two independent continuous variables with normal distribution and two independent variables without normal distribution, respectively, in the study. Chi-square or Fisher's exact tests were used to analyse the categorical variables, if necessary. Using the Spearman and Pearson correlation coefficients, correlations were evaluated. The presence of microalbuminuria was determined using multivariate linear regression analysis in relation to VD levels, HbA1c, and blood pressure measurements. The significance level was set at p .05. SPSS 21 was used to perform the

statistical analysis.

Results

A total of 200 patients took part in the research. Males comprised 33% of the patients, while females comprised 67%. The average person was 50.6 years old, with a standard deviation of 5.4 years. To put it another way, the average height was 165.4 cm, the average weight 85.1 kg, and the average waist circumference 102.4 cm. The average BMI was 34.7 kg/m². Diabetes lasted an average of 8.6 years. Average VD levels were 14.5 ng/mL and HbA1c levels were 8.3–8.5 percent in the study group. VD levels ranged from 11 to 30 ng/mL in 48.7% of these patients, with 56.1 percent having levels under 11 ng/mL.

There were 32 percent smokers and 19.4 percent ex-smokers in the study population. Diabetes mellitus was found in 56.2 percent of the patients, and coronary artery disease was found in 15 percent of the patients. Anti-diabetic treatments were not significantly different between the two groups. There was no significant difference in the use of insulin or oral anti-diabetics between the two groups. 28.5 percent of the study participants were taking anti-hypertensive medications, but those taking renin–angiotensin–aldosterone system anti-hypertensives were excluded. Only 58% of patients had stage 1 renal disease, which was determined by calculating the glomerular filtration rate (GFR) (Table 1). Males were more likely than females to have VD levels less than 10 ng/mL, but there was no significant difference between the two groups in terms of renal impairment. Patients with VD deficiency had significantly higher HbA1c and microalbuminuria levels ($p = .038$ and $p = .031$, respectively). Patients with VD deficiency had significantly higher SBP and DBP ($p = .011$ and $p = .012$) than healthy individuals.

Microalbuminuria and microalbuminuria/creatinine ratio were not statistically related to VD levels. In patients with VD insufficiency, there was no statistically significant correlation between VD levels and microalbuminuria levels.

Logistic regression analysis showed that the level of microalbuminuria was 2.1-fold higher in patients with a VD level of 10 ng/mL or lower, which was statistically significant using the chi-square test ($p = .014$).

Conclusion

Patients with VD deficiency had a higher incidence of microalbuminuria in this study. Blood pressure and glycemic control were significantly worse in this group of patients. Patients who are already devoid of the renoprotective effects of VD may have developed microalbuminuria as a result of impaired glycemic control and poor blood pressure regulation. Diabetic nephropathy is characterised by early renal damage, and the role of VD deficiency in this process has been shown to be important in preventing microvascular complications. There are a few flaws in this study that need to be taken into account. Cross-sectional, a single-center investigation, and no direct evidence of causality were found in this study. To confirm these findings, additional research is needed.

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