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A Study to Assess the Role of Vitamin-D Deficit Among Diabetic Nephropathy Patients

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Introduction

Most of the body's supply of vitamin D (10–20%) comes from dietary sources, while the remaining portion is manufactured in the skin. It plays a traditional role in the regulation of calcium and phosphate homeostasis, but it also plays a significant part in the regulation of immunity, vascular function, cardiomyocyte health, inflammation, insulin resistance, and the reduction of urinary albumin concentration in patients with chronic kidney disease. It is possible for vitamin D to be hydroxylated in the liver to produce 25-hydroxyvitamin D (25[OH]D), which can then be converted to its active form through the process of 1-hydroxylation in the proximal tubules of the kidneys. Studies have shown that patients who have chronic kidney disease are more likely to have low serum 25(OH)D levels. Renal dysfunction is one factor that can have an effect on serum 25(OH)D levels. Several conditions characterised by impaired glucose metabolism, including diabetes mellitus, have been hypothesised to be linked to low levels of vitamin D. Diabetes mellitus can lead to a number of microvascular complications, the most common of which is diabetic nephropathy, which is the leading cause of end-stage renal disease across the globe. It has been demonstrated that oral paricalcitol can reduce albuminuria and inflammation in patients with chronic kidney disease. Furthermore, animal studies have demonstrated that supplementation with calcitriol can reduce albuminuria in mice with diabetic nephropathy.

Most patients who have been diagnosed with vitamin D deficiency have been found to suffer from either chronic kidney disease or diabetes mellitus in the presence of normal renal function. There have only been a few studies that have looked at the metabolic levels of vitamin D in diabetic nephropathy patients. In addition, the factors that put patients with diabetic nephropathy at risk for vitamin D deficiency are not well understood. The current study evaluated the prevalence of vitamin D deficiency in patients with diabetic nephropathy and determined their correlation with clinicopathological features and possible risk factors for vitamin D deficiency. Additionally, the study determined whether or not patients with diabetic nephropathy had an elevated risk of developing vitamin D deficiency.

Methodology

To eliminate the effect of season and geographical area, patients with various stages of diabetic nephropathy from the selected hospitals in Indore were sequentially enrolled between September 2021 and September2022. Retrospective data analysis was performed.All diabetic nephropathy patients who met the following inclusion and exclusion criteria were included. Patients with type 2 diabetes mellitus (including those with intermittent microalbuminuria, persistent urinary microalbumin excretion rate [AER] of 30-300 mg/24 h, or macroalbuminuria [AER > 300 mg/24 h]) were eligible. Exclusion criteria: patients under the age of 18; patients with end-stage renal disease not caused by diabetes mellitus who required renal replacement therapy; patients with an active infection, severe cardiovascular disease, cancer, liver disease, current pregnancy, gallbladder problem, or gastrointestinal disorder; patients who had used mineral oil products and daily multivitamins (i.e., vitamin D) for 4 weeks prior to the screening visit, or regularly used antacids (as these can reduce albuminuria). Healthy controls were recruited at random from the same area as the kidney injury patients. The control subjects were age and gender matched to the patients, as well as meeting the exclusion criteria. The American Diabetes Association guidelines for diabetic nephropathy were used to determine the stage: 12 GFR is significantly increased in Stage I; in Stage II, urinary albumin excretion rate is mostly normal but may increase intermittently (for example, after exercise or stress), and GFR may be slightly increased. Stage III: urinary albumin excretion rate is 20-200 g/min, GFR is normal or increased; Stage IV: urinary albumin excretion rate is >200 g/min, GFR is decreased; Stage V: urinary albumin excretion rate is decreased, but serum creatinine is increased. The Ethics Committee of All Selected Hospitals in Indore approved the study, and each participant provided written informed consent. Each participant had their body mass index (BMI), waist hip ratio (WHR), creatinine clearance rate (Ccr), and estimated glomerular filtration rate (eGFR) measured and calculated. A mercury sphygmomanometer was used to take the patient's blood pressure while they were seated and after they had rested for ten minutes. Each patient underwent the conventional ophthalmological examination in order to determine whether or not they had proliferative or nonproliferative diabetic retinopathy. An electromyogram was what helped determine that the patient had peripheral neuropathy. In this particular study, there were a total of 200 participants, including 100 healthy controls and 100 patients at varying stages of diabetic nephropathy.

Results

The fundamental clinical characteristics of patients diagnosed with diabetic nephropathy as well as normal controls are discussed. Patients who had diabetic nephropathy in stage V were considerably older than the control group and had a longer history of diabetes (P 0.05). This was in comparison to patients who had diabetic nephropathy in stages III or IV. Patients with diabetic nephropathy stages IV and V exhibited greater body mass indexes compared to both controls and patients with diabetic nephropathy stages I and II (P 0.05). Every diabetic nephropathy group, including the control group, had a higher systolic blood pressure (SBP) than the diabetic nephropathy groups, and the SBP exhibited a trend to increase as the diabetic nephropathy advanced. AER was substantially different in patients with diabetic nephropathy stage IV compared to controls and patients with diabetic nephropathy stage I/II or stage III (P 0.05), while AER was significantly greater in patients with diabetic nephropathy stage V compared to all other groups (all P 0.05). Both the HbA1c and the fasting blood glucose levels were considerably higher in all diabetic nephropathy groups compared to the controls. However, the increase in fasting blood glucose levels appeared to level off in patients with diabetic nephropathy stage IV. Patients with diabetic nephropathy stages IV and V had considerably lower levels of haemoglobin compared to controls, while patients with diabetic nephropathy stage V had significantly lower levels of haemoglobin compared to patients with diabetic nephropathy stage IV (all P 0.05). Albumin in the serum tended to decrease as diabetic nephropathy progressed, as did creatinine clearance and estimated glomerular filtration rate. Patients with diabetic nephropathy stage V had considerably higher levels of blood urea nitrogen (BUN), uric acid, and cystatin-C than patients with diabetic nephropathy stages I/II and III, as well as controls. Patients with diabetic nephropathy stage V had significantly higher levels of total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol when compared to patients with diabetic nephropathy stages I/II and controls (all P 0.05). Patients who had diabetic nephropathy stage V had significantly lower serum calcium levels compared to the other groups (all P values were less than 0.05). There was a statistically significant difference between the serum 25(OH)D levels of the four diabetic nephropathy groups and those of the healthy controls (P 0.05). In addition to this, the levels of serum 25(OH)D in diabetic nephropathy groups IV and V were considerably lower than the levels in diabetic nephropathy group I/II (P 0.05). An analysis using univariate conditional logistic regression found that serum 25(OH)D levels were significantly associated with age, sex, diabetes history, BMI, SBP, AER, eGFR, fasting blood glucose, HbA1c, haemoglobin, serum albumin, Ccr, BUN, and proliferative diabetic retinopathy. In addition, older participants, female participants, and obese participants had lower levels of serum 25(OH)D. Significantly more patients with low serum albumin, mild anaemia, and decreased eGFR and Ccr had low 25(OH)D levels (18 ng/ml), and significantly more patients with high AER and high glucose levels had low 25(OH)D levels (18 ng/ml). Both of these associations were found to be significantly associated with low 25(OH)D levels. Other indicators such a family history of diabetes, blood pressure (SBP), serum creatinine, blood urea nitrogen (BUN), smoking, drinking, and diabetic neuropathy with complications were regarded to be confounding factors.

Conclusion

Deficiency in 25(OH)D is common among patients with diabetic nephropathy who are not on dialysis, and it has a tendency to improve as the diabetic nephropathy stage progresses. The presence of serum 25(OH)D appears to be a beneficial inverse predicator of diabetic nephropathy progression, and age, obesity, glucose level, and renal function are all independent risk factors for 25(OH)D insufficiency.

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