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Impact of 25(OH)D Deficiency in Clients With Diabetic Nephropathy : A Cross Sectional Study

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Introduction

Only around 5-10% of the vitamin D you need may be made in your skin, while the other 15-25% must come from diet. In addition to its more obvious role in regulating calcium and phosphate levels, it also regulates immune function, vascular health, cardiomyocyte health, inflammation, insulin resistance, and albumin loss in the urine of persons with chronic kidney disease. The liver may hydroxylate vitamin D to produce 25-hydroxyvitamin, which can then be further converted to active vitamin D by 1-hydroxylation in the proximal tubules of the kidneys. The serum 25(OH)D levels of patients with chronic renal disease have been shown to be lower. Renal impairment is one of the conditions that might affect serum 25(OH)D levels. Scientists have speculated that low vitamin D levels contribute to diabetes mellitus and other disorders with impaired glucose metabolism. One of the numerous microvascular complications of diabetes mellitus is diabetic nephropathy, which is the leading cause of end-stage renal disease globally. Chronic renal disease patients who take paricalcitol orally have a reduction in albuminuria and inflammation. Studies in animals with diabetic nephropathy suggest that calcitriol treatment may reduce albuminuria.

Even in patients with good kidney function, those with vitamin D deficiency often also suffer from either chronic renal disease or diabetes mellitus. However, there is a lack of data about the metabolic levels of vitamin D in people with diabetic nephropathy. Diabetic nephropathy patients often have a hard time pinpointing the causes of their vitamin D deficiency. Vitamin D status was determined through a survey of patients with diabetic nephropathy, and associations between vitamin D deficiency and other clinicopathological features and risk factors for vitamin D deficiency were identified. Vitamin D deficiency in patients with diabetes was also evaluated.

Methodology

Individuates with varied stages of diabetic nephropathy were researched at Index Medical College in Indore, India. A cross-sectional method was used to analyse the data. In addition to the inclusion criteria, patients with diabetic nephropathy who did not fit any of the aforementioned exclusions were also included. There was no distinction made between patients with microalbuminuria (which may be either intermittent or chronic) and those with macroalbuminuria who also had type 2 diabetes mellitus. Patients under the age of 18; those on dialysis for a condition other than diabetes mellitus; those with an active infection; those with severe cardiovascular disease, cancer, liver disease, pregnancy, gallbladder problem, or gastrointestinal disorder; patients who used mineral oil products and daily multivitamins (i.e. vitamin D) for 5 weeks prior to the screening visit; and patients who regularly used antacids were excluded (as these can reduce albuminuria). Individuals without kidney impairment were randomly selected from the same location as those with the injury. Similarities between the patients and the healthy volunteers included age, gender, and exclusion criteria. The American Diabetes Association's guidelines were used to categorise the severity of diabetic nephropathy. Stage I causes a roughly 2-fold increase in GFR, Stage II causes a 3-fold increase, and Stage III causes a generally normal but perhaps elevated urine albumin excretion rate (for instance, after exercise or stress). Urinary albumin excretion rate between 30 and 200 g/min; normal or improving glomerular filtration rate (GFR); Reduced GFR and a urine albumin excretion rate of more than 200 g/min characterise Stage IV, whereas decreasing GFR and elevated serum creatinine define Stage V. The study was approved by the Ethics Committee of All Selected Hospitals in Indore, and all participants signed a consent form indicating that they had been adequately informed of the risks and benefits of taking part. Body mass index, water holding capacity, ccr, and estimated glomerular filtration rate were all measured and calculated for each individual. After having the patient sit for 10 minutes and relax, the blood pressure was measured using a mercury sphygmomanometer. To determine if a patient had proliferative or nonproliferative diabetic retinopathy, standard ophthalmological testing was conducted. An electromyogram helped doctors determine that the patient had peripheral neuropathy. Totaling 200 participants participated in the study, with another 200 serving as healthy controls and 200 having been diagnosed with diabetic nephropathy at varying stages.

Results

Patients who suffer from diabetic nephropathy have their pre-diabetic clinical features compared to those of healthy control subjects. Patients who were diagnosed with stage V diabetic nephropathy were much older than the controls and had a considerably longer history of being diabetic. Patients whose

diabetic nephropathy had progressed to stages III and IV were included in the study as a control group. Patients who had reached stages IV and V of diabetic nephropathy had a higher body mass index than controls and patients who had reached stages I and II of the disease. The systolic blood pressure (SBP) of those with diabetic nephropathy was consistently greater than that of those in the control group throughout all stages of the disease. Patients with DNP stage IV had an AER that was statistically distinct from that of controls and patients with DNP stages I/II or III; however, the AER of patients with DNP stage V had an AER that was considerably higher than that of all other groups. The levels of HbA1c and fasting blood glucose were higher in all of the diabetic nephropathy groups when compared to the values seen in the controls. On the other hand, people with stage IV diabetic nephropathy did not show an increase in their blood glucose levels when they were fasting. Patients with diabetic nephropathy in stages IV and V had substantially lower haemoglobin levels compared to healthy controls. Patients with diabetic nephropathy in stage V had significantly lower haemoglobin levels than patients with diabetic nephropathy in stage IV. There was a correlation between the severity of diabetic nephropathy and a decrease in serum albumin levels, as well as a rise in the rate of creatinine clearance and glomerular filtration. Those with diabetic nephropathy at stage V exhibited significantly greater levels of blood urea nitrogen (BUN), uric acid, and cystatin-C than those with diabetic nephropathy at stages I/II and III, in addition to those who served as controls. Patients who had diabetic nephropathy in stage V were shown to have considerably higher levels of total cholesterol, triglyceride, and low-density lipoprotein (LDL) cholesterol compared to patients in stages I and II, as well as to healthy controls (all P 0.05). Blood calcium levels were substantially lower in patients who had advanced stages of diabetic nephropathy compared to those in the other groups (all P values were less than 0.05). In comparison to the healthy-control group, the serum 25(OH)D levels of all four groups with diabetic nephropathy exhibited significant differences. When compared to Group I of diabetic nephropathy, both Group IV and Group V of diabetic nephropathy exhibited significantly lower amounts of 25(OH)D in their blood. In a univariate conditional logistic regression analysis, it was discovered that age, sex, a history of diabetes, body mass index, systolic blood pressure, acute kidney injury, estimated glomerular filtration rate, fasting blood glucose, haemoglobin A1c, hematocrit, creatinine, urea nitrogen, and proliferative diabetic retinopathy were all significantly related to serum 25(OH)D levels. People who were older, female, or had a higher body mass index also had lower levels of serum 25(OH)D. Low levels of serum albumin, moderate anaemia, reduced eGFR, and Ccr were all associated with low levels of 25(OH)D. On the other hand, high levels of AER and glucose were associated with low levels of 25(OH)D in the opposite direction. It was shown that low levels of both of these were connected to low levels of 25(OH)D. Other indicators, such as a history of diabetes in the patient's family, systolic blood pressure (SBP), serum creatinine (SCr), blood urea nitrogen (BUN), smoking and alcohol consumption, and diabetic neuropathy with consequences were also taken into consideration as potential confounding variables.

Conclusion

Non-dialysis-treated patients with diabetic nephropathy often have low 25(OH)D levels, although these levels tend to rise as the illness improves. Serum 25(OH)D appears to be a good inverse predicator of the development of diabetic nephropathy, and age, obesity, glucose level, and renal function are all independent risk factors for 25(OH)D insufficiency. However, serum 25(OH)D seems to be a useful inverse predictor of the onset of diabetic nephropathy.

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