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Correlation Of Serum Calcium, Phosphorus And 25-Hydroxy Vitamin D With Glycemic Parameters In Patients With Type 2 Diabetes Mellitus: A Cross-Sectional Study

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a metabolic disorder associated with chronic hyperglycemia and insulin resistance. Mineral metabolism, particularly involving calcium, phosphorus, and vitamin D, may play a pivotal role in T2DM pathophysiology.

Aim and Objective: To study the correlation of serum calcium, phosphorus, and vitamin D in type 2 diabetes mellitus patients compared to healthy controls.

Materials & Methods: A prospective cross-sectional study was conducted on 160 individuals — 80 diagnosed T2DM patients (cases) and 80 age- and sex-matched healthy individuals (controls). Serum calcium, phosphorus, and 25-hydroxy vitamin D levels were measured using standardized biochemical methods. Data were analyzed using Pearson correlation and chi-square tests.

Results: Among 80 diabetic patients, 65% were male and 35% female. Vitamin D deficiency (<20 ng/ml) was present in 21.25% of diabetic patients, and serum calcium was below 8.4 mg/dL in 30% of them. Hypophosphatemia (<3.4 mg/dL) was observed in 72.5% of diabetic patients. All three parameters showed statistically significant differences compared to controls.

Conclusion: Diabetic patients exhibited lower levels of serum calcium, phosphorus, and vitamin D. These biochemical markers may serve as early indicators of metabolic dysfunction in T2DM and could have implications for clinical management

Keywords: Type 2 Diabetes Mellitus, Calcium, Phosphorus, Vitamin D, Mineral Metabolism.

1. INTRODUCTION

Diabetes mellitus (DM) is a complex metabolic disorder characterized by chronic hyperglycemia, which results from defects in insulin secretion, insulin action, or both. Among the two major types of diabetes, Type 2 Diabetes Mellitus (T2DM) is the most prevalent, accounting for over 90% of cases globally. The growing epidemic of T2DM has become a significant public health concern worldwide, especially in developing nations like India, where urbanization and lifestyle modifications have contributed to its rapid rise. According to the International Diabetes Federation (IDF), more than 463 million adults were living with diabetes in 2019, and this number is expected to rise to 700 million by 2045. India alone is estimated to have over

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101 million diabetic individuals by 2030 [1].

Type 2 Diabetes Mellitus (T2DM) is a chronic and progressive metabolic disorder that constitutes a major public health burden globally. Characterized by insulin resistance and impaired insulin secretion, T2DM affects the metabolism of carbohydrates, proteins, and fats, leading to chronic hyperglycemia and associated complications. According to the International Diabetes Federation, the global prevalence of diabetes was approximately 463 million in 2019 and is projected to rise to 700 million by 2045. India alone is expected to have over 101 million people with diabetes by 2030, earning it the title "Diabetes Capital of the World" [1,2]

T2DM is not only limited to derangements in glucose metabolism but also influences various physiological systems including bone and mineral metabolism. Numerous studies have reported that disturbances in calcium, phosphorus, and vitamin D homeostasis are commonly seen in diabetic patients [3,4]. The mechanisms behind these disturbances are multifactorial. Insulin resistance, hyperglycemia, and chronic inflammation are believed to affect the metabolism of these minerals. Moreover, vitamin D deficiency has been implicated in impaired pancreatic β-cell function and insulin resistance [5, 6].

Vitamin D, a fat-soluble prohormone, plays an important role in calcium and phosphorus homeostasis and also influences glucose metabolism. It has been shown to enhance insulin sensitivity and reduce systemic inflammation, potentially improving glycemic control[6]. Its receptors are found in pancreatic β -cells and various tissues involved in glucose and lipid metabolism, suggesting a wider role beyond bone health [7]. Several epidemiological studies have suggested that lower levels of serum 25-hydroxy vitamin D are associated with increased risk of developing T2D [8].

Calcium is essential for insulin secretion from β -cells and is involved in intracellular insulin signaling. A deficiency or imbalance in serum calcium may contribute to impaired insulin secretion and insulin resistance [9]. Studies have shown that low calcium levels may correlate with poor glycemic control in diabetic patients [10].

Phosphorus, another important mineral, also plays a role in energy metabolism through its involvement in ATP production. Though less studied than calcium and vitamin D in the context of diabetes, emerging evidence suggests that altered phosphorus levels may be associated with metabolic derangements in T2DM patients [11].

Previous studies have demonstrated variable findings with respect to serum calcium, phosphorus, and vitamin D levels in T2DM patients. Some studies reported significantly lower serum calcium and vitamin D levels in diabetic patients compared to non-diabetic controls, while others found no significant differences [12,13]. The inconsistency in findings could be due to differences in population characteristics, glycemic control, nutritional status, sun exposure, and study methodologies.

Given the increasing prevalence of T2DM and its multisystem complications, it becomes imperative to identify biochemical markers that may serve as early indicators of metabolic dysfunction. Assessing serum calcium, phosphorus, and vitamin D levels in diabetic patients may not only provide insights into their metabolic status but may also help in timely intervention to prevent long-term complications such as osteoporosis, cardiovascular diseases, and nephropathy [14,15].

This study aims to investigate the correlation between serum calcium, phosphorus, and vitamin D levels in T2DM patients compared to healthy controls in a tertiary care hospital setting. By evaluating these parameters, we intend to explore their potential role as biochemical markers in the clinical management of type 2 diabetes mellitus.

2. MATERIAL AND METHODS

A hospital-based prospective cross-sectional observational study was conducted at the Departments of Biochemistry and Department of Physiology, Tertiary Care Centre, over a period of 12 months i.e, March 2024 to March 2025. A total of 160 participants were enrolled, comprising 80 patients with confirmed diagnosis of Type 2 Diabetes Mellitus (T2DM) and 80 age- and sex-matched healthy individuals as controls.

A Total of 160 patients were included in this study and divided into two groups.

Group1: (CASE GROUP) consist of 80 patients with type -2 Diabetes mellitus.

Group 2: (CONTROL GROUP) consists of 80 healthy individuals.

Inclusion Criteria:

Diagnosed cases of T2DM aged between 20 and 80 years.

Patients attending the outpatient or inpatient departments.

Voluntary consent obtained.

Exclusion Criteria:

Patients with chronic liver or kidney diseases.

Individuals on vitamin D or calcium supplementation.

Pregnant women and heavy smokers.

Patients on medications like isoniazid or rifampicin.

Sample Collection

After informed consent, 5 ml of venous blood was collected under aseptic conditions following an overnight fast. Serum was separated and stored at -20°C until analysis. Calcium was estimated using the Arsenazo III method, phosphorus by the Molybdate UV method, and 25-hydroxy vitamin D by ELISA. Fasting and postprandial glucose levels were measured using the GOD-POD method.

Statistical Analysis:

Data were analyzed using Microsoft Excel and SPSS software (version 22.0).

3. RESULTS

In the present study prospective cross-sectional study was conducted on 160 individuals — 80 diagnosed T2DM patients (cases) and 80 age- and sex-matched healthy individuals (controls). Serum calcium, phosphorus, and 25-hydroxy vitamin D levels were measured using standardized biochemical methods. Data were analyzed using Pearson correlation and chi-square tests.

In the control group, males constituted the majority (65%), whereas in the diabetic group, females were predominant (80%). This indicates a significantly higher number of female diabetic patients compared to males in this cohort, suggesting possible gender-specific vulnerability or healthcare-seeking patterns in this population.

Table 1: Comparison of Gender between healthy individuals and diabetic patients.

GENDER	CONTROL GROUP(n=80)	DIABETIC PATIENTS(n=80)
MALES	52 (65%)	16 (20%)
FEMALES	28 (35%)	64 (80%)

The age distribution indicates that the highest proportion of diabetic patients was in the 61–70 years range (28.75%), followed by 51–60 years (25%). The control group had a relatively even distribution, but the majority were aged 41–50 years (25%). This supports the known association between increasing age and risk of T2DM.

Table 2: Comparison of Age between control group and diabetic patients.

AGE	DIABETIC PATIENTS(n=80)	CONTROL GROUP(n=80)
20-30	5 (6.25%)	10(12.5%)
31-40	6 (7.5%)	10 (12.5%)
41-50	14 (17.5%)	20 (25%)
51-60	20 (25%)	15 (18.75%)
61-70	23 (28.75%)	18 (22.5%)
>71	12 (15%)	7 (8.75%)

Table 3: Comparison of Serum Phosphorous level between Diabetic patients and in control groups.

SERUM LEVEL	PHOSPHOROUS	DIABETIC PATIENTS(n=80)	CONTROL GROUP(n=80)	P Value
<3.4mg/dl		58 (72.5%)	8(10%)	
3.4- 4.5mg/dl		18 (22.5%)	68(85%)	0.001
>4.5mg/dl		4 (5%)	4(5%)	

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A significant majority (72.5%) of diabetic patients had hypophosphatemia (<3.4 mg/dL), whereas only 10% of controls had low phosphorus levels. The difference is statistically significant (p = 0.001), suggesting an association between diabetes and altered phosphorus metabolism, potentially due to insulin resistance affecting renal phosphate handling

Table 4: Comparison of Serum calcium level between diabetic patients and control group.

SERUM CALCIUM LEVEL	DIABETIC PATIENTS(n=80)	CONTROL GROUP(n=80)	P Value
<8.4mg/dl	24 (30%)	0	
8.5- 10.5mg/dl	50 (62.5%)	80(100%)	0.005
>10.5mg/dl	6 (7.5%)	0	

Among diabetic patients, 30% had hypocalcemia (<8.4 mg/dL), compared to none in the control group. Most controls had normal calcium levels. The significant p-value (0.005) supports that altered calcium levels are common in T2DM and may contribute to its pathophysiology via impaired insulin release and signaling.

Table 5: Comparison of Serum 25-hydroxy Vitamin-D level between diabetic patients and control group.

SERUM 25-hydroxy Vitamin-D level	DIABETIC PATIENTS(n=80)	CONTROL GROUP(n=80)	P value
<20ng/ml	17 (21.25%)	0	
20-40ng/ml	63 (78.75%)	80(100%)	0.001

Vitamin D deficiency was noted in 21.25% of diabetic patients, whereas all control subjects had sufficient levels. This significant difference (p = 0.001) reinforces the growing body of evidence linking vitamin D deficiency with impaired glucose metabolism and increased T2DM risk.

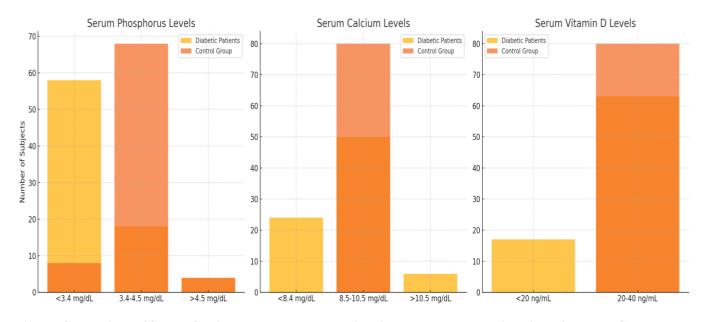


Figure: Comparison of Serum Calcium, Phosphorus, and Vitamin D levels between Diabetic Patients and Controls.

4. DISCUSSION

This study examined the correlation of serum calcium, phosphorus, and vitamin D levels in individuals with type 2 diabetes mellitus (T2DM) compared to healthy controls. Significant differences were observed between the two groups, indicating potential implications in the management and early detection of T2DM

.The finding of lower serum calcium in diabetic patients aligns with previous studies, such as Qadri et al. [16], which noted significantly reduced calcium levels in T2DM compared to prediabetics. Similarly, Kanchana et al. [17] demonstrated a negative correlation between serum calcium and fasting plasma glucose. The mechanism may involve impaired insulin secretion and resistance due to calcium dysregulation. Phosphorus deficiency was also prevalent in diabetic patients in our study (72.5%), consistent with Nigah et al. [18], who showed statistically lower phosphorus levels in diabetics. Mancini et al. [19] found that high dietary phosphorus paradoxically increases diabetes risk, highlighting complexity in phosphorus metabolism. Vitamin D deficiency was evident in 21.25% of diabetic patients. Although not significantly correlated in our results, previous reports—such as those by Bayani et al. [20], Arif et al. [21], and Pittas et al. [22]—have established a strong link between vitamin D deficiency and impaired glycemic control. Teegarden and Donkin [23] proposed vitamin D's role in improving insulin sensitivity and reducing inflammation. In contrast, Marwa et al. [24] found a higher prevalence of T2DM in females, while our study noted higher prevalence in males. These demographic variances could be due to regional, genetic, or lifestyle factors. Likewise, while some studies showed no significant vitamin D differences, others such as Forouhi et al. [25] demonstrated predictive relationships between baseline 25(OH)D and future glucose status. Therefore, this study reinforces the importance of evaluating mineral and vitamin levels in diabetic patients. These biomarkers may aid in early metabolic assessment, guiding interventions like supplementation or lifestyle modification.

Our findings confirm a consistent pattern of reduced vitamin D levels in T2DM, accompanied by modest inverse associations with glycemic control. Calcium and phosphorus remained relatively stable, suggesting their homeostasis is less disrupted. Screening for and treating vitamin D deficiency may complement glycemic management strategies in T2DM

5. CONCLUSION

This study concludes that serum levels of calcium, phosphorus, and vitamin D are significantly altered in T2DM patients compared to healthy individuals. Monitoring these markers can assist in the early identification of metabolic disturbances and potentially improve glycemic control. Further longitudinal studies are needed to elucidate causal mechanisms and optimize clinical interventions.

Limitations

The cross-sectional design prevents inference of causality.

Confounders such as sun exposure, dietary calcium/vitamin D intake, and body mass index were not controlled.

Vitamin D status was based solely on serum 25(OH)D without account of seasonal or environmental factors.

DECLARATIONS

Conflicts of interest: There is no any conflict of interest associated with this study

Consent to participate: We have consent to participate.

Consent for publication: We have consent for the publication of this paper.

Authors' contributions: All the authors equally contributed the work..

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