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To study correlation of serum 25(OH) vitamin D with insulin resistance in prediabetic patients

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Abstract

Background and Objectives: Prediabetes represents an intermediate state between normoglycemia and type 2 diabetes mellitus (T2DM), with high risk of progression to overt diabetes. Vitamin D deficiency has been linked to impaired insulin secretion, increased insulin resistance, and glucose intolerance. While the relationship between vitamin D status and insulin resistance in diabetic patients has been documented, evidence in prediabetic individuals remains limited. The present study aimed to evaluate the association between serum 25(OH) vitamin D and insulin resistance among prediabetic individuals.

Materials and Methods: This cross-sectional observational study was conducted at Mediciti Institute of Medical Sciences, Hyderabad, India. A total of 100 individuals aged 25-50 years were included, comprising 50 prediabetic patients (cases) and 50 age- and sex-matched healthy controls. Prediabetes was defined as fasting plasma glucose (FPG) 100-125 mg/dl and/or 2-hour post-OGTT plasma glucose 140-199 mg/dl. Serum 25(OH) vitamin D levels and fasting insulin were measured; insulin resistance was calculated using HOMA-IR. Data were analyzed using unpaired Student's t-test and Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

Results: The mean age, gender distribution, and BMI were comparable between groups. Hypovitaminosis D (<20 ng/ml) was observed in 66% of prediabetic individuals compared to 16% of controls. Prediabetic cases showed significantly higher FPG (100.24 ± 9.18 vs. 84.27 ± 7.44 mg/dl), 2-hour plasma glucose (153.87 ± 13.56 vs. 124.38 ± 11.46 mg/dl), insulin (18.26 ± 12.14 vs. 10.81 ± 8.29 µIU/ml), and HOMA-IR (5.32 ± 2.96 vs. 2.31 ± 2.01), and significantly lower serum 25(OH) vitamin D levels (14.88 ± 11.36 vs. 22.16 ± 8.47 ng/ml) compared to controls (all p<0.05). A significant inverse correlation was found between serum 25(OH) vitamin D and both HOMA-IR (r = -0.275, p=0.038) and fasting insulin (r = -0.268, p=0.029), while correlations with FPG and 2-hour plasma glucose were not significant.

Conclusion: Vitamin D deficiency is highly prevalent in prediabetic individuals and is significantly associated with insulin resistance. These findings suggest that hypovitaminosis D may contribute to the pathogenesis of insulin resistance in prediabetes, highlighting the potential role of vitamin D status as a modifiable factor in early prevention of diabetes.

Keywords: Prediabetes, Vitamin D deficiency, 25(OH) vitamin D, Insulin resistance, HOMA-IR

Introduction

Prediabetes is an intermediate state between normal glucose homeostasis and type 2 diabetes mellitus (T2DM), defined by impaired fasting glucose (100-125 mg/dl), impaired glucose tolerance (2-hour plasma glucose 140-199 mg/dl after oral glucose tolerance test), or both [1, 2]. Individuals with prediabetes are at substantially increased risk of progressing to overt T2DM, with reported annual conversion rates ranging from 2.5% to 18%. In India, this transition occurs more rapidly than in many other populations, contributing to the country's high burden of diabetes. According to the Indian Council of Medical Research (2011), an estimated 62.4 million individuals in India had diabetes, and 77.2 million had prediabetes, underscoring the urgent need for preventive strategies [3, 4].

Vitamin D has emerged as a potential modifiable factor influencing glucose metabolism. Beyond its classical role in calcium homeostasis and bone health, vitamin D is implicated in pancreatic β-cell function, insulin sensitivity, and regulation of inflammatory pathways. Deficiency of serum 25(OH) vitamin D may impair insulin secretion and enhance insulin resistance, thereby contributing to glucose intolerance. Conversely, supplementation with

vitamin D in diabetic patients has shown improvement in insulin sensitivity and metabolic control [5, 6].

Despite this evidence, the role of vitamin D deficiency in prediabetic individuals remains less clearly defined. Limited studies have explored whether hypovitaminosis D contributes to the pathophysiology of insulin resistance before the onset of diabetes. Understanding this association is important, as early identification and correction of vitamin D deficiency could potentially delay or prevent the progression from prediabetes to T2DM [7, 8].

The present study was designed to evaluate the relationship between serum 25(OH) vitamin D levels and insulin resistance in prediabetic individuals compared with normoglycemic controls.

Materials and Methods

This was a cross-sectional, observational study conducted in the Mediciti Institute of Medical Sciences, Hyderabad, India, between June 2024 and May 2025. Ethical clearance was obtained from the Institutional Ethics Committee prior to commencement of the study. Written informed consent was taken from all participants. A total of 100 individuals aged 25-50 years were enrolled. Fifty prediabetic subjects fulfilling the American Diabetes Association (ADA) criteria served as the case group, while 50 age- and sex-matched healthy individuals with normal glucose tolerance served as controls.

Inclusion criteria

- Fasting plasma glucose between 100-125 mg/dl, and/or
- 2-hour post-oral glucose tolerance test plasma glucose between 140-199 mg/dl.

Exclusion criteria

- History of diabetes mellitus or current use of oral antidiabetic drugs/insulin.
- Chronic illnesses such as chronic kidney disease, liver disease, malignancy.
- History of long-term drug use known to interfere with vitamin D metabolism.
- Prior calcium or vitamin D supplementation within the last year.

Data collection

Demographic details including age, sex, and anthropometric measurements (weight, height, body mass index [BMI]) were recorded. Routine laboratory investigations (complete blood count, renal and liver function tests, serum calcium and phosphate) were performed in all participants.

Biochemical measurements

- Fasting plasma glucose and 2-hour plasma glucose were measured using the standard glucose oxidase-peroxidase method.
- Serum insulin levels were determined using a commercially available Insulin IRMA kit (immunoradiometric assay).
- Insulin resistance was calculated using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR):\

$$\text{HOMA-IR} = \frac{\text{Fasting insulin } (\mu\text{IU/mL}) \times \text{Fasting glucose (mmol/L)}}{22.5}$$

- **Serum 25(OH) vitamin D levels** were measured using a commercial radioimmunoassay kit (DiaSorin 25(OH) Vitamin D 125I RIA kit, Stillwater, Minnesota, USA). Values were expressed in ng/mL and categorized as: deficiency (<20 ng/mL), insufficiency (20-29.9 ng/mL), and sufficiency (≥30 ng/mL).

Statistical analysis

Data were analyzed using SPSS software version 20. Continuous variables were expressed as mean ± standard deviation (SD). Comparisons between prediabetic cases and controls were made using the unpaired Student's *t*-test. Correlations between serum 25(OH) vitamin D and glycemic/insulin resistance parameters were assessed using Pearson's correlation coefficient. A *p*-value <0.05 was considered statistically significant.

Results

A total of 100 participants were included in the study, comprising 50 prediabetic individuals (cases) and 50 healthy normoglycemic individuals (controls). The two groups were comparable in terms of age, gender distribution, and body mass index (BMI) (Table 1).

Vitamin D status

Among prediabetic individuals, 66% had hypovitaminosis D (<20 ng/ml) compared to only 16% among controls. Vitamin D sufficiency (≥30 ng/ml) was observed in 14% of cases versus 46% of controls, indicating a significantly higher prevalence of vitamin D deficiency in the prediabetic group.

Table 1: Characteristics of prediabetic cases and controls

Variable	Cases (n=50)	Controls (n=50)	Total (n=100)
Age 25-40 years	19 (38%)	21 (42%)	40
Age 41-50 years	31 (62%)	29 (58%)	60
Male	32 (64%)	30 (60%)	62
Female	18 (36%)	20 (40%)	38
BMI ≤ 25 kg/m²	20 (40%)	28 (56%)	48
BMI > 25 kg/m²	30 (60%)	22 (44%)	52
Vitamin D < 20 ng/ml	33 (66%)	8 (16%)	41
Vitamin D 20-29.9 ng/ml	10 (20%)	19 (38%)	29
Vitamin D ≥ 30 ng/ml	7 (14%)	23 (46%)	30

Glycemic and biochemical parameters

Prediabetic cases demonstrated significantly higher fasting plasma glucose, 2-hour plasma glucose, fasting insulin, and HOMA-IR values, along with significantly lower serum 25(OH) vitamin D levels compared to controls (all $p < 0.05$) (Table 2).

Table 2: Comparison of glycemic parameters, insulin levels, HOMA-IR, and vitamin D levels

Variable	Cases (n=50)	Controls (n=50)	*p*-value	Significance
Fasting plasma glucose (mg/dl)	100.24 ± 9.18	84.27 ± 7.44	<0.001	Significant
2-hour plasma glucose (mg/dl)	153.87 ± 13.56	124.38 ± 11.46	<0.001	Significant
Fasting insulin (µIU/ml)	18.26 ± 12.14	10.81 ± 8.29	<0.001	Significant
HOMA-IR	5.32 ± 2.96	2.31 ± 2.01	<0.001	Significant
25(OH) vitamin D (ng/ml)	14.88 ± 11.36	22.16 ± 8.47	0.032	Significant

Correlation analysis

Pearson's correlation analysis showed a significant inverse correlation of serum 25(OH) vitamin D with HOMA-IR (r = -0.275, p = 0.038) and fasting insulin levels (r = -0.268, p = 0.029). No significant correlation was found between serum vitamin D and fasting plasma glucose or 2-hour plasma glucose (Table 3).

Table 3: Correlation between vitamin D status and glycemic/insulin resistance parameters in prediabetic individuals

Parameters compared	Correlation coefficient (r)	*p*-value	Significance
25(OH)D vs. HOMA-IR	-0.275	0.038	Significant
25(OH)D vs. Fasting insulin	-0.268	0.029	Significant
25(OH)D vs. Fasting glucose	-0.092	0.071	Not significant
25(OH)D vs. 2-hour glucose	-0.132	0.075	Not significant

These results demonstrate that hypovitaminosis D is highly prevalent in prediabetes and is significantly associated with higher insulin resistance and fasting insulin levels, but not directly with glucose levels.

Discussion

The present study evaluated the association between serum 25(OH) vitamin D levels and insulin resistance in prediabetic individuals compared with normoglycemic controls. We observed that vitamin D deficiency was significantly more prevalent in the prediabetic group (66%) than in healthy controls (16%). Furthermore, serum 25(OH) vitamin D levels showed a significant inverse correlation with HOMA-IR and fasting insulin, suggesting that hypovitaminosis D contributes to increased insulin resistance even before the onset of overt type 2 diabetes mellitus (T2DM) [9-11].

These findings are consistent with several Indian and international studies. Dutta *et al*. reported that prediabetic individuals with severe vitamin D deficiency (<10 ng/ml) had markedly higher insulin resistance compared to those with higher vitamin D levels, supporting the inverse relationship between vitamin D status and insulin resistance. Similarly, Manju *et al*. demonstrated a strong negative correlation between serum vitamin D levels and HOMA-IR in prediabetic and diabetic states. Studies in Western populations have also documented an association of low serum 25(OH) vitamin D with impaired glucose tolerance, metabolic syndrome, and increased risk of progression to diabetes [12-14].

The biological plausibility of this association is supported by several mechanisms. Vitamin D receptors are expressed in pancreatic β-cells and insulin-sensitive tissues, where vitamin D regulates insulin secretion, enhances insulin sensitivity, and modulates inflammatory pathways. Vitamin D deficiency may lead to increased intracellular calcium in adipocytes, promoting lipogenesis and insulin resistance. It may also impair β-cell function through decreased insulin synthesis and secretion. Thus, hypovitaminosis D may act both as a marker and mediator of insulin resistance in prediabetes [15, 16].

Our study also demonstrated that although vitamin D levels correlated significantly with HOMA-IR and fasting insulin, they did not correlate significantly with fasting plasma glucose or 2-hour post-OGTT glucose levels. This suggests that vitamin D status may influence insulin sensitivity more directly than glycemic values in the prediabetic stage. Glycemic abnormalities may manifest later as insulin resistance progresses, further emphasizing the role of vitamin D deficiency as an early metabolic risk factor [16, 17].

The prevalence of hypovitaminosis D in prediabetes in our study (66%) is comparable to other Indian reports, highlighting the widespread vitamin D deficiency in this population, likely related to limited sunlight exposure, urban lifestyles, and inadequate dietary intake. This raises the possibility that correcting vitamin D deficiency in prediabetic individuals may improve insulin sensitivity and potentially delay progression to diabetes. Indeed, interventional studies have shown mixed results: Pittas *et al*. demonstrated improvement in HOMA-IR and glycemic indices with vitamin D and calcium supplementation, while some meta-analyses found no significant effect on insulin resistance but reported modest reductions in fasting plasma glucose and HbA1c. These inconsistencies suggest that the benefit of supplementation may depend on baseline vitamin D status, degree of deficiency, and study population [17, 18].

Strengths of this study include strict application of inclusion/exclusion criteria, measurement of both insulin resistance indices and vitamin D status, and comparison with a well-matched control group. Limitations include the relatively small sample size, single-center cross-sectional design, and absence of long-term follow-up or intervention to assess the effect of vitamin D supplementation. Furthermore, seasonal variation in vitamin D synthesis and dietary intake was not evaluated, which may have influenced results [18, 19].

Despite these limitations, our findings add to the growing body of evidence linking hypovitaminosis D with insulin resistance in prediabetes. This suggests that serum 25(OH) vitamin D could serve as a simple biomarker for early risk stratification. Future large-scale prospective studies are warranted to clarify whether vitamin D supplementation can improve insulin sensitivity and prevent progression from prediabetes to T2DM [19, 20].

Conclusion

The present study demonstrates that vitamin D deficiency is highly prevalent among individuals with prediabetes and is

significantly associated with increased insulin resistance. These findings suggest that hypovitaminosis D may contribute to the pathophysiology of prediabetes and highlight the potential role of vitamin D status as a modifiable risk factor. Screening and correction of vitamin D deficiency in prediabetic individuals may help improve insulin sensitivity and could delay progression to type 2 diabetes mellitus.

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None.

Conflict of Interest
None.

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