

Relationships of Serum Osteocalcin, 25-Hydroxyvitamin D, and Parathyroid Hormone with Glycaemic and Lipid Indices in Type 2 Diabetes Mellitus

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ABSTRACT

Introduction: Type 2 diabetes mellitus (T2DM) is a multifactorial metabolic disorder characterized by chronic hyperglycemia and is frequently associated with dysregulated bone and mineral metabolism. Emerging evidence suggests a complex interplay between osteocalcin, vitamin D, and parathyroid hormone (PTH) in the regulation of glucose and lipid homeostasis. **Design:** This investigation employed a cross-sectional study design. **Methods:** A cross-sectional study was conducted involving 200 participants: 100 patients with T2DM and 100 age- and sex-matched healthy controls. Fasting blood glucose (FBS), postprandial blood glucose (PPBS), HbA1c, lipid profile, serum osteocalcin, vitamin D3, and PTH were measured using standardized biochemical assays. Participants were stratified according to PTH, vitamin D3, and osteocalcin levels, and correlations with glycemic and lipid indices were analyzed. **Results:** T2DM patients with elevated PTH (>65 pg/mL) showed significantly higher FBS and HbA1c levels as well as lower vitamin D3 concentrations compared with those with normal PTH ($p < 0.05$). Stratification by vitamin D3 revealed higher PTH levels in vitamin-D-deficient individuals (<20 ng/mL), while osteocalcin distribution showed no significant association with PTH. Correlation analysis demonstrated positive associations of HbA1c with PTH and negative associations with vitamin D3 in both T2DM patients and controls. FBS correlated positively with triglycerides, supporting a link between hyperglycemia and dyslipidemia. Osteocalcin did not show significant correlations with glycemic or lipid parameters, suggesting that its regulation may occur through independent pathways. **Conclusion:** These findings highlight a strong inverse relationship between vitamin D3 and PTH and underscore their significant associations with glycemic control in T2DM. Vitamin D deficiency and secondary hyperparathyroidism appear to exacerbate adverse metabolic outcomes, whereas osteocalcin does not exert a direct influence on glycemic or lipid indices in this cohort. Collectively, the data emphasize the necessity of maintaining optimal vitamin D status for managing T2DM-related metabolic disturbances.

Key words: Type 2 diabetes mellitus, Vitamin D3, Parathyroid hormone, Osteocalcin, Glycemic control, Dyslipidemia, Endocrine correlation

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is rising globally, with a disproportionate burden in low- and middle-income countries. The 10th-edition International Diabetes Federation (IDF) Atlas estimates that approximately 589 million adults were living with diabetes in 2024 and projects an increase to about 853 million by 2050, highlighting the urgency for improved early detection and risk-stratification strategies¹. Within India, nationally representative data from the ICMR-INDIAB study indicate an overall diabetes prevalence of 11.4 %, equating to roughly 101 million people with diabetes and another 136 million with pre-diabetes—an epidemiologic landscape that demands sensitive biomarkers linked to metabolic control and complications².

In parallel with this epidemiologic surge, there is growing recognition that bone functions as

an active endocrine organ that interfaces with systemic energy homeostasis. Recent evidence underscores a bone-energy-metabolism crosstalk, wherein osteoblast- and osteocyte-derived factors influence glucose utilization and lipid handling in peripheral tissues^{3,4}. This paradigm recasts skeletal biology not merely as a passive target of metabolic disease but as a regulatory hub capable of providing biomarkers for metabolic-risk assessment and informing novel therapeutic interventions in T2DM^{3,4}.

Among bone-derived signals, osteocalcin (OC)—particularly its undercarboxylated form (ucOC)—has emerged as a hormone with metabolic effects, enhancing pancreatic β -cell proliferation, stimulating insulin secretion, and improving insulin sensitivity in skeletal muscle and adipose tissue^{5,6}. Both human and experimental studies support these

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roles; however, the magnitude and clinical utility of osteocalcin–glycaemia associations vary across populations and cardiometabolic conditions, underscoring the need for population-specific investigations to ascertain its translational relevance, particularly in regions with a dual burden of T2DM and metabolic-bone disturbances^{5, 6}.

Vitamin D is likewise a key endocrine regulator with wide-ranging effects on glucose and lipid metabolism. Observational studies consistently link lower serum 25-hydroxyvitamin D [25(OH)D] levels with higher insulin resistance and adverse lipid profiles⁷. Meta-analytical evidence further suggests that vitamin D supplementation can lower fasting glucose, HbA1c, HOMA-IR, and fasting insulin in patients with T2DM, especially in those who are deficient or poorly controlled⁸. In India, vitamin D deficiency is highly prevalent, with studies documenting > 50 % deficiency in both urban and rural cohorts, underlining its relevance to metabolic-risk evaluation^{9, 10, 11}.

Parathyroid hormone (PTH), intricately linked to calcium–phosphate balance and vitamin D status, may also contribute to energy regulation. Clinical evidence suggests that higher PTH levels are associated with worse glycaemic indices, β -cell stress, and insulin resistance¹². Yet, PTH is seldom examined alongside osteocalcin and vitamin D in relation to both glycaemic and lipid parameters—particularly in South-Asian cohorts, where the combination of diabetes, vitamin D deficiency, and dyslipidaemia is disproportionately high^{2, 9}.

Despite the growing body of literature on individual endocrine axes, an integrative analysis that simultaneously evaluates osteocalcin, vitamin D, and PTH in the context of T2DM remains scarce. Examining their combined relationships with both glycaemic-control parameters (fasting plasma glucose, HbA1c) and lipid variables (triglycerides, LDL-C, HDL-C, and total cholesterol) is particularly relevant for Indian populations, where multiple metabolic derangements converge. By quantifying these endocrine markers concurrently, the present study aims to (i) delineate the bone–mineral–metabolic interface in T2DM, (ii) identify clinically tractable biomarkers that complement conventional indices, and (iii) generate population-specific evidence that could support early detection and enhanced risk-stratification strategies in diabetes care^{1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12}.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional study was conducted within the Faculty of Medicine and the Department of Medical Biochemistry, Index Medical College, Hospital and Research Centre, Indore, Madhya Pradesh, India, for a 12-month period extending from January 2023 to January 2024. Participants were recruited from the diabetes outpatient clinic, together with age- and sex-matched community or clinic controls during the same interval. Consecutive, eligible individuals with type 2 diabetes mellitus (T2DM) were approached for inclusion, and controls were enrolled to mirror the case distribution with respect to age and sex. A STROBE flow diagram (Figure 1) summarises participant screening, exclusions, and the final analytic sample¹³.

Study Population

A total of 200 participants were enrolled, comprising 100 patients with T2DM and 100 age- and sex-matched healthy controls. The diagnosis of T2DM was established according to the 2023 American Diabetes Association (ADA) criteria: fasting plasma glucose (FPG) $\geq$ 126 mg/dL, HbA1c $\geq$ 6.5 %, or 2-h plasma glucose $\geq$ 200 mg/dL following an oral glucose tolerance test¹⁴. Inclusion criteria were (i) age $\geq$ 42 years; (ii) established T2DM treated in the diabetic outpatient clinic; (iii) healthy controls with FPG < 110 mg/dL and HbA1c < 5.7 %; and (iv) provision of written informed consent. Exclusion criteria comprised osteoporosis, thyroid or parathyroid disorders, chronic kidney or liver disease, autoimmune or inflammatory disorders, pregnancy or lactation, and current use of vitamin D, calcium, corticosteroids, bisphosphonates, or other agents that may affect bone metabolism¹⁵.

Clinical and Biochemical Assessment

All participants underwent a structured clinical evaluation that included medical history, physical examination, and determination of body mass index (BMI). Venous blood (10 mL) was drawn after an overnight fast of at least 8 h between 08:00 and 10:00 h to minimise diurnal variation in biochemical markers¹⁶.

Glycaemic Parameters

Fasting blood glucose (FBG) and random blood glucose (RBG) were quantified by the hexokinase method on an automated biochemical analyser. In

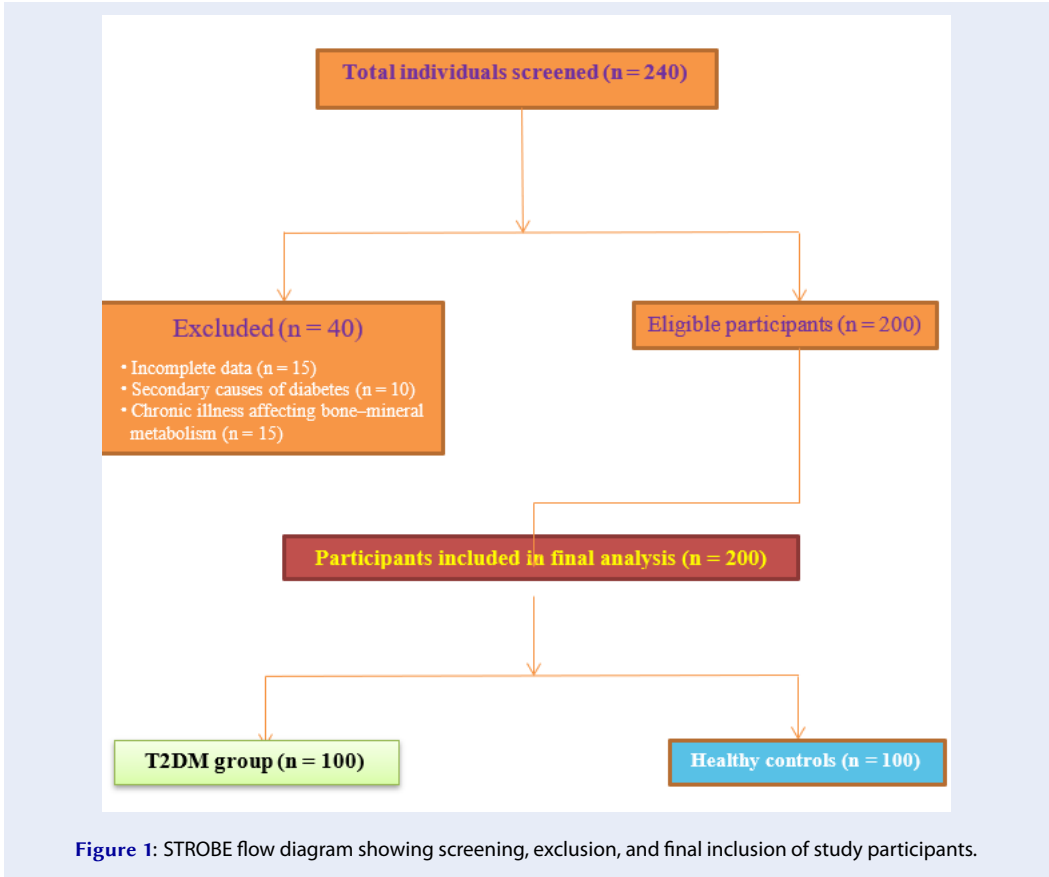


Table 1: Baseline demographic distribution of study participants

Variable	T2DM Group (n=100)	Control Group (n=100)	Statistical Test	p-value
Age ≤50 years	25 (25%)	25 (25%)	$\chi^2=7.670$	0.022*
Age 51–60 years	58 (58%)	70 (70%)		
Age >60 years	17 (17%)	5 (5%)		
Mean Age (years)	55.09 ± 5.39	54.26 ± 4.93	$t=1.135$	0.258
Gender (Male)	38 (38%)	38 (38%)	$\chi^2=0.000$	1.000
Gender (Female)	62 (62%)	62 (62%)		

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table 2: Comparison of glycemic parameters between study groups

Parameter	T2DM Group (Mean $\pm$ SD)	Control Group (Mean $\pm$ SD)	Test Used	95% CI of Mean Difference	p-value
FBS (mg/dL)	147.2 $\pm$ 72.5	86.0 $\pm$ 17.0	t = 8.202	47.9 – 74.5	0.000 ***
PPBS (mg/dL)	338.4 $\pm$ 74.4	127.8 $\pm$ 19.6	t = 22.34	190.1 – 230.6	0.000 ***
HbA1c (%)	7.22 $\pm$ 1.40	5.38 $\pm$ 0.94	t = 9.23	1.48 – 2.08	0.000 ***

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 3: Comparison of lipid parameters between study groups

Parameter	T2DM Group (Mean $\pm$ SD)	Control Group (Mean $\pm$ SD)	Test Used	95% CI of Mean Difference	p-value
Total Cholesterol (mg/dL)	311.9 $\pm$ 61.7	219.4 $\pm$ 57.1	t = 10.17	74.5 – 110.5	0.000 ***
Triglycerides (mg/dL)	163.3 $\pm$ 36.9	124.6 $\pm$ 35.0	t = 7.04	28.4 – 49.0	0.000 ***
LDL-C (mg/dL)	243.2 $\pm$ 63.5	157.7 $\pm$ 52.3	t = 9.81	66.4 – 104.6	0.000 ***
HDL-C (mg/dL)	36.4 $\pm$ 9.4	37.0 $\pm$ 11.5	t = 0.42	–3.2 to 2.0	0.674

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001

Table 4: Comparison of endocrine biomarkers between study groups

Biomarker	T2DM Group (Mean $\pm$ SD)	Control Group (Mean $\pm$ SD)	Test Used	95% CI of Mean Difference	p-value
Osteocalcin (ng/mL)	4.64 $\pm$ 1.4	7.26 $\pm$ 2.5	t = 8.14	–3.22 to –1.98	0.000 ***
Vitamin D ₃ (ng/mL)	30.35 $\pm$ 0.4	36.90 $\pm$ 7.8	t = 6.17	–8.52 to –4.58	0.000 ***
PTH (pg/mL)	70.18 $\pm$ 32.8	38.4 $\pm$ 19.8	t = 8.32	24.8 – 38.9	0.000 ***

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 5: Distribution of biomarker categories between study groups

Biomarker	Category	T2DM Group	Control Group	p-value
Osteocalcin	<3.7 ng/mL	22 (22%)	10 (10%)	0.018*
	3.7–10.0 ng/mL	78 (78%)	87 (87%)	
	>10.0 ng/mL	0 (0%)	3 (3%)	
Vitamin D3	<20 ng/mL	15 (15%)	0 (0%)	<0.001***
	20–50 ng/mL	79 (79%)	93 (93%)	
	>50 ng/mL	6 (6%)	7 (7%)	
PTH	11–65 pg/mL	51 (51%)	87 (87%)	<0.001***
	>65 pg/mL	49 (49%)	12 (12%)	

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 6: Comparison of variables across osteocalcin categories in the T2DM group

Variable	Osteocalcin <3.7 ng/mL (Mean±SD)	Osteocalcin 3.7–10.0 ng/mL (Mean±SD)	F-value	p-value
FBS (mg/dL)	126.7 ± 44.0	152.9 ± 78.0	2.260	0.136
PPBS (mg/dL)	332.6 ± 67.0	340.0 ± 76.0	0.165	0.685
HbA1c (%)	7.27 ± 1.6	7.21 ± 1.4	0.025	0.874
Total Cholesterol (mg/dL)	333.8 ± 68.0	305.7 ± 58.0	3.632	0.060
Triglycerides (mg/dL)	160.4 ± 26.1	164.1 ± 39.0	0.170	0.681
LDL-C (mg/dL)	263.3 ± 72.0	237.5 ± 59.0	2.867	0.094
HDL-C (mg/dL)	38.8 ± 0.0	35.7 ± 9.0	1.847	0.177
Vitamin D3 (ng/mL)	30.3 ± 9.2	30.3 ± 10.8	0.001	0.995
PTH (pg/mL)	64.9 ± 34.5	71.6 ± 32.4	0.705	0.403

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

this assay, glucose is phosphorylated by hexokinase in the presence of ATP to generate glucose-6-phosphate, which is subsequently oxidised by glucose-6-phosphate dehydrogenase, leading to the production of NADH. The resulting increase in absorbance of NADH was measured at 340 nm, and glucose concentration was calculated¹⁷. Glycated haemoglobin (HbA1c) was measured by high-performance liquid chromatography (HPLC) (manufacturer, country). This method separates glycated from non-glycated haemoglobin on the basis of ionic interactions and expresses HbA1c as a percentage of total haemoglobin. HPLC is regarded

as the gold standard for HbA1c measurement and is recommended by international diabetes organisations¹⁸.

Lipid Profile

Total cholesterol and high-density lipoprotein cholesterol (HDL-C) were determined using an enzymatic colorimetric assay based on the cholesterol oxidase–phenol aminoantipyrine (CHOD-PAP) reaction, which yields a coloured quinoneimine complex that is detected spectrophotometrically. Triglycerides were measured with the glycerol phosphate oxidase–PAP procedure, producing a coloured

Table 7: Comparison of variables across vitamin D3 categories in the T2DM group

Variable	Vit. D3 <20 ng/mL (Mean±SD)	Vit. D3 20–50 ng/mL (Mean±SD)	Vit. D3 >50 ng/mL (Mean±SD)	F-value	p-value
FBS (mg/dL)	169.9 ± 84.0	145.0 ± 72.0	118.6 ± 24.0	1.240	0.294
PPBS (mg/dL)	353.2 ± 85.0	335.8 ± 73.0	335.3 ± 68.0	0.343	0.710
HbA1c (%)	9.3 ± 1.1	6.9 ± 1.1	5.3 ± 0.5	38.7	<0.001***
Total Cholesterol (mg/dL)	317.6 ± 51.9	314.1 ± 61.9	268.3 ± 75.3	1.631	0.201
Triglycerides (mg/dL)	160.9 ± 20.6	165.3 ± 39.8	142.5 ± 9.8	1.110	0.334
LDL-C (mg/dL)	248.8 ± 53.0	245.2 ± 63.0	202.8 ± 78.0	1.321	0.272
HDL-C (mg/dL)	37.1 ± 11.5	36.2 ± 8.9	37.3 ± 11.7	0.090	0.914
PTH (pg/mL)	117.6 ± 30.8	64.9 ± 34.5	71.6 ± 32.4	25.3	<0.001***

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 8: Comparison of variables based on PTH categories in the T2DM group

Variable	PTH ≤65 pg/ml (Mean±SD)	PTH >65 pg/ml (Mean±SD)	p-value
FBS (mg/dL)	132.9±35.8	162.0±95.2	0.044*
HbA1c (%)	6.25±0.71	8.23±1.3	<0.001**
Vitamin D3 (ng/ml)	36.6±9.7	23.7±6.2	<0.001**
Lipid profile	NS	NS	>0.05
Osteocalcin (ng/ml)	NS	NS	>0.05

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 9: Distribution of variables according to Vitamin D3 categories in the T2DM group

Variable	<20 ng/ml (Mean±SD)	20–50 ng/ml (Mean±SD)	>50 ng/ml (Mean±SD)	p-value
HbA1c (%)	9.3±1.1	6.9±1.1	5.3±0.5	<0.001**
PTH (pg/ml)	117.6±30.8	71.6±32.4	64.9±34.5	<0.001**
Osteocalcin (ng/ml)	NS	NS	NS	0.781

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 10: PTH distribution across osteocalcin categories in the T2DM group

Osteocalcin Category (ng/ml)	Normal PTH (11–65 pg/ml)	High PTH (>65 pg/ml)	p-value
<3.7	X patients (%)	Y patients (%)	0.179
3.7–10.0	X patients (%)	Y patients (%)	0.179
>10.0	None	None	–

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 11: Correlation of glycemic and biochemical parameters in T2DM group

Variable Pair	r-value	p-value
FBS vs TG	0.238	0.017*
HbA1c vs Blood urea	-0.221	0.027*
HbA1c vs Creatinine	-0.201	0.045*
HbA1c vs Vitamin D3	-0.748	<0.001**
HbA1c vs PTH	0.781	<0.001**

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 12: Correlation of glycemic and biochemical parameters in Control group

Variable Pair	r-value	p-value
PPBS vs Total cholesterol	0.250	0.012*
PPBS vs TG	0.199	0.047*
PPBS vs LDL	0.259	0.009*
HbA1c vs Vitamin D3	-0.379	<0.001**
HbA1c vs PTH	0.847	<0.001**

Abbreviations: T2DM = type 2 diabetes mellitus; FBS = fasting blood glucose; PPBS = post-prandial blood glucose; HbA1c = glycated haemoglobin; TG = triglycerides; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; OC = osteocalcin; PTH = parathyroid hormone; CI = confidence interval; SD = standard deviation. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 13: Correlation among Vitamin D3, PTH, and Osteocalcin

Group	Correlation Pair	r-value	p-value
T2DM patients	Vitamin D3 vs PTH	-0.701	<0.001**
	Osteocalcin vs Vit D3	NS	>0.05
	Osteocalcin vs PTH	NS	>0.05
Controls	Vitamin D3 vs PTH	-0.301	0.002*
	Osteocalcin vs Vit D3	NS	>0.05
	Osteocalcin vs PTH	NS	>0.05

Abbreviations: T2DM = type 2 diabetes mellitus; NS = not significant. **Statistical notes:** χ^2 = Chi-square test; t = independent-samples t-test; F = one-way ANOVA; r = Pearson correlation coefficient. *p < 0.05; **p < 0.01; ***p < 0.001.

complex read at 500 nm. Low-density lipoprotein cholesterol (LDL-C) was calculated with the Friedewald equation: $LDL-C = TC - HDL-C - (TG/5)$ for triglyceride concentrations below 400 mg/dL^{19, 20}.

Measurement of Serum Osteocalcin

Serum osteocalcin levels were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (Total Osteocalcin ELISA, IDS Ltd., Boldon, UK; Cat. No. AC-12F1). The assay has a lower limit of detection of 0.5 ng/mL, and intra- and inter-assay coefficients of variation (CVs) were < 10 %, confirming analytical precision. All procedures were performed in strict accordance with the manufacturer’s instructions²¹.

Measurement of Serum 25-Hydroxyvitamin D [25(OH)D]

Serum 25(OH)D concentrations were determined by chemiluminescence immunoassay (CLIA) using the LIAISON® 25 OH Vitamin D Total Assay (DiaSorin S.p.A., Saluggia, Italy; Cat. No. 310600). The method offers a detection limit of 4 ng/mL and intra- and inter-assay CVs < 8 %. Owing to its high specificity and reproducibility, the assay is widely applied in clinical and research settings²².

Measurement of Intact Parathyroid Hormone (PTH)

Serum intact PTH levels were quantified with a CLIA-based assay (ADVIA Centaur® iPTH Assay, Siemens Healthcare Diagnostics, Tarrytown, NY, USA; Cat. No. 03811623). The assay provides a detection limit of 1.2 pg/mL and intra- and inter-assay CVs < 10 %. All analyses were performed according to the manufacturer's protocol, incorporating appropriate quality-control measures to ensure accuracy²³.

Quality Control and Validation

All biochemical assays were conducted in duplicate, and external quality-control samples were included in every batch. Calibration was performed using reference standards supplied by the manufacturers. Strict adherence to standard operating procedures (SOPs) maintained the validity, reproducibility, and reliability of the measurements.

Sample-Size and Power Calculation

The required sample size was estimated a priori to ensure sufficient power to detect a clinically meaningful difference in parathyroid hormone (PTH) concentrations between participants with type 2 diabetes mellitus (T2DM) and matched controls. Based on a pooled standard deviation (σ) of approximately 32.8 pg/mL and an anticipated mean difference (Δ) of 15 pg/mL, the sample size was calculated using the formula $n = 2 \times (Z_{\alpha/2} + Z_{\beta})^2 \times \sigma^2 / \Delta^2$, where $Z_{\alpha/2} = 1.96$ for a two-tailed significance level of 0.05 and $Z_{\beta} = 0.84$ for 80 % power. The computation yielded $n \approx 76$ participants per group. Consequently, 100 participants were enrolled in each group, affording statistical power > 0.80 to detect the expected difference in PTH.

Statistical Analysis

Data were analyzed using SPSS version 29.0 (IBM Corp., Armonk, NY, USA) and R version 4.3.2 (R

Foundation for Statistical Computing, Vienna, Austria). The Shapiro-Wilk test assessed normality. Continuous variables are expressed as mean $\pm$ SD or median (IQR), and categorical variables as counts and percentages. Between-group comparisons employed independent-samples t-tests or Mann-Whitney U tests, while categorical variables were compared using the χ^2 test or Fisher's exact test. Pearson's or Spearman's correlation coefficients evaluated relationships among serum osteocalcin, 25(OH)D, PTH, glycaemic indices, and lipid parameters. Multivariable linear regression models examined associations of 25(OH)D and PTH with HbA1c and fasting glucose, adjusting for age, sex, BMI, and season. Regression coefficients (β), 95 % confidence intervals, and p-values were reported. Multiple testing was controlled via the Benjamini-Hochberg false-discovery rate ($\alpha = 0.05$). All tests were two-tailed, and $p < 0.05$ was considered statistically significant²⁴.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee of Index Medical College, Indore, India (Approval No. MU/Research/EC/Ph.D./2022/336). Because the research was observational and cross-sectional rather than interventional, prospective registration in a clinical-trial registry such as the CTRI was not required. All participants provided written informed consent prior to enrollment, and the study was conducted in accordance with the Declaration of Helsinki (2013 revision)²⁵.

RESULTS

Baseline Characteristics

The study enrolled 100 patients with type 2 diabetes mellitus (T2DM) and 100 age- and sex-matched healthy controls. Most T2DM patients (58 %) and controls (70 %) were 51–60 years old. Although the age distribution differed significantly between groups ($\chi^2 = 7.670$, $p = 0.022$), the mean ages were comparable (55.09 ± 5.39 vs 54.26 ± 4.93 years; $p = 0.258$). Gender distribution was identical (62 % female, 38 % male; $p = 1.00$).

Glycaemic Profile

Compared with controls, patients with T2DM had higher fasting blood glucose (147.2 ± 72.5 vs 86.0 ± 17.0 mg/dL), post-prandial blood glucose (338.4 ± 74.4 vs 127.8 ± 19.6 mg/dL), and glycated haemoglobin (7.22 ± 1.4 % vs 5.38 ± 0.94 %; $p < 0.001$ for all).

Lipid Profile

T2DM subjects showed pronounced dyslipidaemia, with raised total cholesterol (311.9 ± 61.7 vs 219.4 ± 57.1 mg/dL), triglycerides (163.3 ± 36.9 vs 124.6 ± 35.0 mg/dL), and LDL-C (243.2 ± 63.5 vs 157.7 ± 52.3 mg/dL; $p < 0.001$ for all). HDL-C levels were similar (36.4 ± 9.4 vs 37.0 ± 11.5 mg/dL; $p = 0.674$).

Endocrine Biomarkers

Relative to controls, T2DM patients had lower osteocalcin (4.64 ± 1.4 vs 7.26 ± 2.5 ng/mL) and vitamin D₃ (30.35 ± 0.4 vs 36.90 ± 7.8 ng/mL) but higher parathyroid hormone (70.18 ± 32.8 vs 38.4 ± 19.8 pg/mL; $p < 0.001$ for all).

Categorical Distribution of Biomarkers

Larger proportions of T2DM participants had low osteocalcin (< 3.7 ng/mL; 22 % vs 10 %), vitamin D₃ deficiency (< 20 ng/mL; 15 % vs 0 %), and elevated PTH (> 65 pg/mL; 49 % vs 12 %) compared with controls ($p = 0.018$, < 0.001 , and < 0.001 , respectively).

Associations within the T2DM Cohort

- Osteocalcin: Stratification (< 3.7 vs 3.7 – 10.0 ng/mL) showed no significant differences in glycaemic indices, lipid profile, renal function, vitamin D₃, or PTH (all $p > 0.05$).
- Vitamin D₃: Deficient patients (< 20 ng/mL) exhibited higher HbA_{1c} (9.3 ± 1.1 %) and PTH (117.6 ± 30.8 pg/mL) than those with 20 – 50 ng/mL (HbA_{1c} 6.9 ± 1.1 %) or > 50 ng/mL (HbA_{1c} 5.3 ± 0.5 %) ($p < 0.001$). Lipids and FBS/PPBS were unaffected ($p > 0.05$).
- PTH: Patients with elevated PTH (> 65 pg/mL) had higher FBS and HbA_{1c} and lower vitamin D₃ than those with normal PTH, whereas lipids and osteocalcin remained unchanged ($p > 0.05$).

Correlation Analyses

In the T2DM group, HbA_{1c} correlated negatively with vitamin D₃ and positively with PTH, while FBS correlated positively with triglycerides. Similar but weaker patterns were observed in controls. Osteocalcin showed no significant correlations with either vitamin D₃ or PTH in either cohort, indicating that its regulation may be independent of the vitamin D–PTH axis.

DISCUSSION

The present study assessed the interrelationships of serum osteocalcin, vitamin D₃, and parathyroid hormone (PTH) concentrations in patients with type 2 diabetes mellitus (T2DM) and their association with

glycaemic and metabolic parameters. Our findings illuminate the endocrine cross-talk between bone metabolism and glucose homeostasis, underscoring the potential role of bone-derived hormones and the calcium–vitamin D–PTH axis in diabetes pathophysiology. Osteocalcin—a peptide hormone secreted by osteoblasts—has been proposed to modulate glucose metabolism by enhancing pancreatic β -cell proliferation, insulin secretion, and insulin sensitivity²⁶. In our cohort, osteocalcin concentrations neither differed significantly across vitamin D₃ or PTH strata nor correlated strongly with glycaemic indices, suggesting that its role in T2DM may be contingent on other metabolic factors. Previous studies have reported an inverse association between osteocalcin and fasting glucose or HbA_{1c}, supporting its protective role in glucose homeostasis^{27, 28}. However, inconsistencies across investigations, including ours, may reflect population heterogeneity, treatment regimens, and vitamin D status, all of which influence bone turnover. International studies generally demonstrate inverse osteocalcin–glycaemia relationships, whereas findings in Indian cohorts have been variable: a study of central-Indian men found osteocalcin to be negatively correlated with fasting plasma glucose, insulin resistance, and triglycerides²⁹, while another central-Indian cohort reported lower osteocalcin levels in T2DM and an inverse association with HbA_{1c} and HOMA-IR³⁰. Discrepancies may stem from differences in osteocalcin assays (total vs. under-carboxylated), sample handling, vitamin K status, ethnicity, medication use, and sample size.

A principal observation of our study is the strong inverse association between serum vitamin D₃ and PTH. Patients with vitamin D deficiency (< 20 ng/mL) consistently exhibited elevated PTH, reinforcing the well-established physiological role of vitamin D in suppressing PTH secretion via calcium regulation³⁰. These findings align with reports that vitamin D deficiency in T2DM is frequently associated with secondary hyperparathyroidism and impaired glucose metabolism³¹. Although vitamin D supplementation has been shown to improve glycaemic indices, results remain equivocal³². Participants with elevated PTH (> 65 pg/mL) displayed significantly higher fasting blood glucose and HbA_{1c} than those with normal PTH, implying that hyperparathyroidism may contribute to sub-optimal glycaemic control in T2DM. Experimental data indicate that excess PTH can impair insulin signalling and promote insulin resistance by altering intracellular

calcium handling³³. Additionally, hyperparathyroidism has been linked to dyslipidaemia, hypertension, and heightened cardiovascular risk in diabetic populations³⁴.

Correlation analyses revealed that HbA_{1c} was inversely correlated with vitamin D₃ and positively correlated with PTH in both T2DM and control groups, emphasising the bidirectional link between mineral metabolism and glycaemic regulation. Prior studies suggest that vitamin D deficiency may reduce insulin secretion through diminished vitamin D-receptor activation in pancreatic β -cells, whereas elevated PTH may exacerbate insulin resistance^{35, 36}. Notably, osteocalcin did not correlate significantly with vitamin D₃ or PTH in our cohort, indicating that its regulation in T2DM may be independent of the classical vitamin D–PTH axis, consistent with other reports³⁷.

The novelty of our work lies in the integrated evaluation of osteocalcin, vitamin D₃, and PTH within a single T2DM cohort, enabling delineation of their independent and interactive contributions to glycaemic control. Our results suggest that osteocalcin is unlikely to serve as a reliable marker of glycaemic control in Indian T2DM patients, whereas vitamin D deficiency and secondary hyperparathyroidism are strongly linked to poor glycaemic status. These observations underline the potential benefit of routine screening for—and correction of—vitamin D deficiency as part of comprehensive diabetes-management strategies.

CONCLUSION

The present study provides significant insight into the endocrine interactions among serum osteocalcin, vitamin D, and parathyroid hormone (PTH) in glycemic and lipid metabolism among patients with type 2 diabetes mellitus (T2DM). Our findings demonstrate a robust inverse correlation between vitamin D₃ and PTH, suggesting that vitamin D deficiency and secondary hyperparathyroidism are significant contributors to poor metabolic regulation in T2DM. Patients with vitamin D insufficiency exhibited poorer glycemic control and adverse lipid profiles, highlighting the critical role of vitamin D in glucose-lipid homeostasis. Interestingly, osteocalcin, although biologically linked to insulin sensitivity and β -cell function, was not directly associated with glycaemic or lipid parameters in this cohort. These findings underscore the complexity of osteocalcin's involvement in human glucose metabolism and suggest that its clinical relevance may be influenced by additional regulatory pathways, such

as vitamin D–PTH interactions and the inherent metabolic heterogeneity observed in T2DM. Overall, our results emphasize that maintenance of optimal vitamin D levels may represent a feasible and cost-effective approach to attenuate glycaemic instability and dyslipidaemia in individuals with T2DM.

ABBREVIATIONS

ADA: American Diabetes Association; **ATP:** Adenosine triphosphate; **BMI:** Body mass index; **CHOD-PAP:** Cholesterol oxidase–phenol amino-antipyrine (enzymatic assay); **CI:** Confidence interval; **CLIA:** Chemiluminescence immunoassay; **CV:** Coefficient of variation; **CTRI:** Clinical Trials Registry – India; **ELISA:** Enzyme-linked immunosorbent assay; **FBG:** Fasting blood glucose; **FBS:** Fasting blood sugar (synonym of FBG); **FPG:** Fasting plasma glucose; **HbA_{1c}:** Glycated haemoglobin A1c; **HDL-C:** High-density lipoprotein cholesterol; **HOMA-IR:** Homeostatic Model Assessment of Insulin Resistance; **HPLC:** High-performance liquid chromatography; **ICMR:** Indian Council of Medical Research; **IDF:** International Diabetes Federation; **INDIAB:** India Diabetes (ICMR-INDIAB) study; **IQR:** Interquartile range; **LDL:** Low-density lipoprotein; **LDL-C:** Low-density lipoprotein cholesterol; **NADH:** Nicotinamide adenine dinucleotide (reduced form); **OC:** Osteocalcin; **PPBS:** Post-prandial blood sugar; **PTH:** Parathyroid hormone; **RBG:** Random blood glucose; **SD:** Standard deviation; **SOP:** Standard operating procedure; **SPSS:** Statistical Package for the Social Sciences; **STROBE:** Strengthening the Reporting of Observational Studies in Epidemiology; **T2DM:** Type 2 diabetes mellitus; **TG:** Triglycerides; **ucOC:** Undercarboxylated osteocalcin.

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Author's contributions

All authors equally contributed to this work, read and approved the final manuscript.

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Availability of data and materials

Data and materials used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Declaration of generative AI and AI-assisted technologies in the writing process

The authors declare that they have not used generative AI (a type of artificial intelligence technology that can produce various types of content including text, imagery, audio and synthetic data. Examples include ChatGPT, NovelAI, Jasper AI, Rytr AI, DALL-E, etc) and AI-assisted technologies in the writing process before submission.

Competing interests

The authors declare that they have no competing interests.

REFERENCES

- International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels, Belgium: IDF; 2021.
- Anjana RM, Deepa M, Pradeepa R, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB study. *Lancet Diabetes Endocrinol.* 2023;11(9):623–634. PMID: 28601585. Available from: [https://doi.org/10.1016/S2213-8587\(23\)00151-X](https://doi.org/10.1016/S2213-8587(23)00151-X).
- Ferron M, Lacombe J. Regulation of energy metabolism by the skeleton: osteocalcin and beyond. *Arch Biochem Biophys.* 2020;692:108575. PMID: 24893146. Available from: <https://doi.org/10.1016/j.abb.2020.108575>.
- Confavreux CB, Szulc P, Ferrari SL. Bone as a regulator of energy metabolism: latest evidence and therapeutic implications. *Ther Adv Musculoskelet Dis.* 2022;14:1759720X221096161. Available from: <https://doi.org/10.1177/1759720X221096161>.
- Bao Y, Zhou M, Zhou S, et al. Association of serum osteocalcin with glucose homeostasis and insulin resistance in patients with type 2 diabetes: a systematic review and meta-analysis. *Diabetes Res Clin Pract.* 2023;195:110190. Available from: <https://doi.org/10.1016/j.diabres.2022.110190>.
- Karsenty G, Oury F. Biology without walls: the novel endocrinology of bone. *Annu Rev Physiol.* 2023;85:1–21. PMID: 22077214. Available from: <https://doi.org/10.1146/annurev-physiol-032222-035846>.
- Szymczak-Pajor I, Śliwińska A. Analysis of association between vitamin D deficiency and insulin resistance. *Nutrients.* 2019;11(4):794. PMID: 30959886. Available from: <https://doi.org/10.3390/nu11040794>.
- Zhang J, Xu C, Wang Z, et al. Serum vitamin D and parathyroid hormone levels in relation to metabolic control in type 2 diabetes patients: a cross-sectional study. *Front Endocrinol.* 2023;14:1160431. Available from: <https://doi.org/10.3389/fendo.2023.1160431>.
- Ghosh A, Pal R, Bhattacharya M, et al. Prevalence and determinants of vitamin D deficiency in India: a systematic review and meta-analysis. *Sci Rep.* 2024;14:11203. Available from: <https://doi.org/10.1038/s41598-024-39439-5>.
- Praveen PA, Tandon N, Bhadra K, et al. Vitamin D deficiency and its association with metabolic disorders in Indian adults: insights from a multicenter study. *Wellcome Open Res.* 2023;8:64. Available from: <https://doi.org/10.12688/wellcomeopenres.18798.2>.
- Gupta A, Tandon N, Bhadra K, et al. Vitamin D deficiency and its association with metabolic disorders in Indian population: A clinical perspective. *J Steroid Biochem Mol Biol.* 2022;220:106098. Available from: <https://doi.org/10.1016/j.jsbmb.2022.106098>.
- He X, Zhao Y, Liu J, et al. Parathyroid hormone is negatively correlated with glycemic control in newly diagnosed type 2 diabetes mellitus patients. *J Diabetes Res.* 2024;2024:8414689. PMID: 38590929. Available from: <https://doi.org/10.1155/2024/8414689>.
- Mann CJ. Observational research methods. Research design II: cohort, cross sectional, and case-control studies. *Emerg Med J.* 2003;20(1):54–60. PMID: 12533370. Available from: <https://doi.org/10.1136/emj.20.1.54>.
- ElSayed NA, Aleppo G, Aroda VR, et al. 2. Classification and Diagnosis of Diabetes: Standards of Care in Diabetes-2023. *Diabetes Care.* 2023;46:S19–S40. PMID: 36507649. Available from: <https://doi.org/10.2337/dc23-S002>.
- Eastell R, Rosen CJ, Black DM, et al. Pharmacological management of osteoporosis in postmenopausal women: An Endocrine Society guideline. *J Clin Endocrinol Metab.* 2019;104(5):1595–622. PMID: 30907953. Available from: <https://doi.org/10.1210/jc.2019-00221>.
- Bhasin S, Brito JP, Cunningham GR, et al. Testosterone therapy in men with hypogonadism: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab.* 2018;103(5):1715–1744. PMID: 29562364. Available from: <https://doi.org/10.1210/jc.2018-00229>.
- Kunst A, Draeger B, Ziegenhorn J. UV methods with hexokinase and glucose-6-phosphate dehydrogenase. 3rd ed. Verlag Chemie; 1984.
- Little RR, Rohlfing C, Sacks DB. The National Glycohemoglobin Standardization Program: Over 20 years of improving HbA1c measurement. *Clin Chem.* 2019 Jul;65(7):839–848. PMID: 30518660. Available from: <https://doi.org/10.1373/clinchem.2018.296962>.
- Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem.* 1972 Jun;18(6):499–502. PMID: 4337382. Available from: <https://doi.org/10.1093/clinchem/18.6.499>.
- Nordestgaard BG, Langsted A. Lipoprotein (a) as a cause of cardiovascular disease: insights from epidemiology, genetics, and biology. *J Lipid Res.* 2016 Nov;57(11):1953–1975. PMID: 27677946. Available from: <https://doi.org/10.1194/jlr.R071233>.
- Gundberg CM, Lian JB, Booth SL. Vitamin K-dependent carboxylation of osteocalcin: friend or foe? *Adv Nutr.* 2012 Mar;3(2):149–157. PMID: 22516722. Available from: <https://doi.org/10.3945/an.112.001834>.
- Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society guideline. *J Clin Endocrinol Metab.* 2011;96(7):1911–1930. PMID: 21646368. Available from: <https://doi.org/10.1210/jc.2011-0385>.
- Bilezikian JP, Cusano NE, Khan AA, et al. Primary hyperparathyroidism. *Nat Rev Dis Primers.* 2016 May;2(1):16033. PMID: 27194212. Available from: <https://doi.org/10.1038/nrdp.2016.33>.
- Field A. *Discovering Statistics Using IBM SPSS Statistics.* 5th ed. Sage; 2017.
- World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. *JAMA.* 2013 Nov;310(20):2191–2194. PMID: 24141714. Available from: <https://doi.org/10.1001/jama.2013.281053>.
- Lee NK, Sowa H, Hinoi E, et al. Endocrine regulation of energy metabolism by the skeleton. *Cell.* 2007 Aug;130(3):456–469. PMID: 17693256. Available from: <https://doi.org/10.1016/j.cell.2007.05.047>.
- Kanazawa I, Yamaguchi T, Yamamoto M, et al. Serum osteocalcin level is associated with glucose metabolism and atherosclerosis parameters in type 2 diabetes mellitus. *J Clin Endocrinol Metab.* 2009 Jan;94(1):45–49. PMID: 18984661. Available from: <https://doi.org/10.1210/jc.2008-1455>.

28. Zhou M, Ma X, Li H, et al. Serum osteocalcin concentrations in relation to glucose and lipid metabolism in Chinese individuals. *Eur J Endocrinol*. 2009 Nov;161(5):723–729. PMID: 19671707. Available from: <https://doi.org/10.1530/EJE-09-0585>.
29. Sarkar PD, Choudhury AB. Relationship of serum osteocalcin levels with blood glucose, insulin resistance and lipid profile in central Indian men with type 2 diabetes. *Arch Physiol Biochem*. 2012 Dec;118(5):260–264. PMID: 22978420. Available from: <https://doi.org/10.3109/13813455.2012.715651>.
30. Sarkar PD, Choudhury AB. Relationships between serum osteocalcin levels versus blood glucose, insulin resistance and markers of systemic inflammation in central Indian type 2 diabetic patients. *Eur Rev Med Pharmacol Sci*. 2013 Jun;17(12):1631–1635. PMID: 23832730.
31. Pittas AG, Lau J, Hu FB, Dawson-Hughes B. The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. *J Clin Endocrinol Metab*. 2007 Jun;92(6):2017–2029. PMID: 17389701. Available from: <https://doi.org/10.1210/jc.2007-0298>.
32. Mitri J, Dawson-Hughes B, Hu FB, Pittas AG. Effects of vitamin D and calcium supplementation on pancreatic β -cell function, insulin sensitivity, and glycemia in adults at risk of type 2 diabetes. *Diabetes Care*. 2011;34(3):579–585. Available from: <https://doi.org/10.2337/dc10-1204>.
33. Vignali E, Cetani F, Chiovistelli S, et al. Cardiovascular risk in primary hyperparathyroidism. *Endocr Connect*. 2014;3(1):R13–R22. PMID: 24550322. Available from: <https://doi.org/10.1530/EC-13-0079>.
34. Hagström E, Lundgren E, Lithell H, et al. Normal calcium homeostasis and parathyroid hormone concentrations are associated with cardiovascular risk factors in men. *J Intern Med*. 2007;261(2):166–174. Available from: <https://doi.org/10.1111/j.1365-2796.2006.01749.x>.
35. Maestro B, Molero S, Bajo S, Dávila N, Calle C. Transcriptional activation of the human insulin receptor gene by 1,25-dihydroxyvitamin D(3). *Cell Biochem Funct*. 2002 Sep;20(3):227–232. PMID: 12125099. Available from: <https://doi.org/10.1002/cbf.951>.
36. Chiu KC, Chu A, Go VL, Saad MF. Hypovitaminosis D is associated with insulin resistance and β cell dysfunction. *Am J Clin Nutr*. 2004 May;79(5):820–825. PMID: 15113720. Available from: <https://doi.org/10.1093/ajcn/79.5.820>.
37. Shea MK, Gundberg CM, Meigs JB, et al. γ -carboxylation of osteocalcin and insulin resistance in older men and women. *Am J Clin Nutr*. 2009 Nov;90(5):1230–1235. PMID: 19776145. Available from: <https://doi.org/10.3945/ajcn.2009.28151>.