ISSN 0973-2063 (online) 0973-8894 (print)
Bioinformation 22(10): 6211-6216 (2026)
©Biomedical Informatics (2026)

BIOINFORMATION
Discovery at the interface of physical and biological sciences
BIOMEDICAL INFORMATICS
www.bioinformation.net
Volume 22(10)
Research Article

Received October 1, 2026; Revised October 31, 2026; Accepted October 31, 2026, Published October 31, 2026
DOI: 10.6026/973206300226211

SJIF 2026 (Scientific Journal Impact Factor for 2026) = 8.478
2022 Impact Factor (2023 Clarivate Inc. release) is 1.9

**Declaration on Publication Ethics:**
The author's state that they adhere with COPE guidelines on publishing ethics as described elsewhere at https://publicationethics.org/. The authors also undertake that they are not associated with any other third party (governmental or non-governmental agencies) linking with any form of unethical issues connecting to this publication. The authors also declare that they are not withholding any information that is misleading to the publisher in regard to this article.

**Declaration on official E-mail:**
The corresponding author declares that lifetime official e-mail from their institution is not available for all authors

**License statement**:
This is an Open Access article which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly credited. This is distributed under the terms of the Creative Commons Attribution License

**Comments from readers:**
Articles published in BIOINFORMATION are open for relevant post publication comments and criticisms, which will be published immediately linking to the original article without open access charges. Comments should be concise, coherent and critical in less than 1000 words.

**Disclaimer:**
Bioinformation provides a platform for scholarly communication of data and information to create knowledge in the Biological/ Biomedical domain after adequate peer/editorial reviews and editing entertaining revisions where required. The views and opinions expressed are those of the author(s) and do not reflect the views or opinions of Bioinformation and (or) its publisher Biomedical Informatics. Biomedical Informatics remains neutral and allows authors to specify their address and affiliation details including territory where required.

**Edited by Vini Mehta**
**E-mail: vmehta@statsense.in**
**Citation**: Baburaj *et al.* Bioinformation 22(10): 6211-6216 (2026)

# Vitamin D and glycemic control for type 2 diabetes: A cross-sectional study in Datia region India

**A.B. Akash Baburaj*, Praveen Kumar Tagore & Jitendra Kanjolia**

Department of General Medicine, Government Medical College, Datia, Madhya Pradesh, India; *Corresponding author

**Affiliation URL:**
https://datiamedicalcollege.edu.in

**Author contact:**
A.B. Akash Baburaj - E-mail: akash4absam@gmail.com; Phone: +91 7749996507
Praveen Kumar Tagore - E-mail: tagore.praveen@gmail.com
Jitendra Kanjolia - E-mail: jitendrakanjolia@gmail.com

ISSN 0973-2063 (online) 0973-8894 (print)
Bioinformation 22(10): 6211-6216 (2026)
©Biomedical Informatics (2026)

**Abstract:**
Vitamin D deficiency has been implicated in impaired glucose metabolism, yet its relationship with glycemic control in individuals with Type 2 Diabetes Mellitus remains incompletely understood. Therefore, it is of interest to evaluate the association between serum vitamin D and glycated hemoglobin levels in 250 patients aged 30–75 years with Type 2 Diabetes Mellitus using biochemical and immunoassay-based investigations. The mean serum vitamin D level was 21.95 ± 9.74 nanograms per milliliter, with 45.6% of participants' deficient, 37.2% insufficient and 75.6% exhibiting poor glycemic control, while serum vitamin D was measured by chemiluminescent immunoassay and glycated hemoglobin by high-performance liquid chromatography. Serum vitamin D showed significant negative correlations with fasting blood sugar ($r = -0.620$), post-prandial blood sugar ($r = -0.632$) and glycated hemoglobin ($r = -0.701$), with a significant association between vitamin D status and glycemic control (chi-square = 53.744; $p < 0.001$). Thus, data shows the evidence linking vitamin D deficiency with poor glycemic control and support the incorporation of vitamin D assessment into the comprehensive management of Type 2 Diabetes Mellitus.

**Keywords:** Type 2 diabetes mellitus (T2DM), vitamin D, Glycated hemoglobin (HbA1c), glycemic control, hypovitaminosis D, insulin resistance, diabetes mellitus (DM)

**Background:**
Type 2 Diabetes Mellitus (T2DM) is one of the most prevalent non-communicable diseases worldwide and represents a major public health challenge. According to the International Diabetes Federation, more than 530 million adults are currently living with diabetes globally and this number is expected to rise substantially in the coming decades. India is recognized as the diabetes capital of the world, with an estimated burden exceeding 100 million individuals affected by diabetes, posing significant socioeconomic and healthcare challenges. The increasing prevalence is attributed to urbanization, sedentary lifestyles, obesity, dietary transitions and population aging **[1, 2].** The pathophysiology of T2DM involves a complex interaction between insulin resistance, pancreatic β-cell dysfunction, genetic susceptibility and environmental influences. Chronic hyperglycemia leads to microvascular and macrovascular complications including nephropathy, retinopathy, neuropathy, cardiovascular disease and increased mortality. Glycated hemoglobin (HbA1c) remains the gold standard marker for long-term glycemic control because it reflects average blood glucose levels over the preceding two to three months. Elevated HbA1c levels are strongly associated with diabetes-related complications and poorer clinical outcomes **[3, 4].** Vitamin D is a fat-soluble secosteroid hormone traditionally known for its role in calcium homeostasis and skeletal health. However, recent research has identified numerous extra-skeletal functions of vitamin D, including modulation of immune responses, inflammation, cardiovascular health and metabolic regulation. Vitamin D receptors (VDRs) are widely distributed in pancreatic β-cells, skeletal muscle, adipose tissue and various organs involved in glucose metabolism, suggesting a broader physiological role beyond bone health **[5, 6].** Several biological mechanisms have been proposed to explain the relationship between vitamin D and glucose metabolism. Vitamin D may enhance insulin secretion by regulating intracellular calcium concentrations within pancreatic β-cells. It may also improve insulin sensitivity by increasing insulin receptor expression and reducing systemic inflammation. Furthermore, vitamin D deficiency has been associated with increased production of pro-inflammatory cytokines, oxidative stress and impaired glucose utilization, all of which contribute to insulin resistance **[7, 8].** Hypovitaminosis D has emerged as a common nutritional deficiency worldwide, affecting individuals across different age groups and geographic regions. Despite abundant sunlight exposure, vitamin D deficiency remains highly prevalent in India due to factors such as skin pigmentation, limited outdoor activities, dietary inadequacies and cultural practices. Several epidemiological studies have showed a high prevalence of vitamin D deficiency among patients with T2DM and have suggested an inverse relationship between serum vitamin D levels and HbA1c. However, findings remain inconsistent across populations and geographical regions **[9–11].** A recent cross-sectional study from eastern India similarly reported a high prevalence of vitamin D deficiency and a strong inverse correlation between serum vitamin D and HbA1c, although findings remain inconsistent across populations and geographical regions **[12].** Recent systematic reviews and observational studies have reported that diabetic individuals with lower vitamin D concentrations tend to exhibit poorer glycemic control and higher HbA1c levels. Recent evidence from India has similarly shown that vitamin D-deficient patients with T2DM had significantly higher HbA1c, fasting blood glucose and post-prandial blood glucose levels than vitamin D-sufficient patients, further supporting an association between vitamin D status and glycemic control. Some interventional studies have also suggested that vitamin D supplementation may improve insulin sensitivity and glycemic parameters, although the magnitude of benefit remains controversial. Consequently, understanding the association between vitamin D status and diabetes control may have important implications for prevention and management strategies **[5, 13 and 14].** Therefore, it is of interest to evaluate serum vitamin D levels and determine their correlation with glycated hemoglobin levels in patients with Type 2 Diabetes Mellitus.

**Materials and Methodology:**
**Study design:**
A hospital-based cross-sectional observational study was conducted to evaluate the correlation between serum vitamin D levels and glycated hemoglobin (HbA1c) levels among patients diagnosed with Type 2 Diabetes Mellitus.

**Study setting:**
The study was carried out in the Department of General Medicine, Government Medical College, Datia, Madhya Pradesh.

**Study duration:**
The study was conducted between 2023 and 2026 after obtaining approval from the Institutional Ethics Committee.

**Study population:**
The study population consisted of patients diagnosed with Type 2 Diabetes Mellitus attending the Outpatient Department (OPD) and Inpatient Department (IPD) of the Department of General Medicine.

**Sample size:**
A total of 250 patients fulfilling the eligibility criteria were included in the study using simple random sampling techniques.

**Inclusion criteria:**
**[1]** Known cases of Type 2 Diabetes Mellitus.
**[2]** Age between 30 and 75 years.
**[3]** Patients willing to provide informed written consent.

**Exclusion criteria:**
**[1]** Diabetes mellitus with renal complications.
**[2]** Patients receiving vitamin D supplementation.
**[3]** Patients receiving calcium supplementation.
**[4]** Critically ill patients.
**[5]** Pregnant women.
**[6]** Individuals unwilling to participate in the study.

**Data collection procedure:**
All eligible participants were screened according to the inclusion and exclusion criteria. Written informed consent was obtained before enrollment. Demographic information including age, sex, residence, body mass index, duration of diabetes and relevant clinical history was recorded using a predesigned structured proforma. Venous blood samples were collected under aseptic precautions after overnight fasting. Laboratory investigations included:

**[1]** Fasting Blood Sugar (FBS)
**[2]** Post-Prandial Blood Sugar (PPBS)
**[3]** Glycated Hemoglobin (HbA1c)
**[4]** Serum Vitamin D (25(OH)D)

HbA1c estimation was performed using High-Performance Liquid Chromatography (HPLC). Serum vitamin D concentrations were measured by Chemiluminescent Immunoassay (CLIA).

**Classification of vitamin D status:**
Participants were categorized according to serum vitamin D levels as follows:
**[1]** Deficient: <20 ng/mL
**[2]** Insufficient: 20–30 ng/mL
**[3]** Sufficient: >30 ng/mL

**Assessment of glycemic control:**
Glycemic control was categorized based on HbA1c levels:
**[1]** Good Glycemic Control: HbA1c <7%
**[2]** Poor Glycemic Control: HbA1c ≥7%

**Outcome measures:**
**Primary outcome:**
Correlation between serum vitamin D levels and HbA1c levels.

**Secondary outcomes:**
**[1]** Correlation between serum vitamin D levels and fasting blood sugar.
**[2]** Correlation between serum vitamin D levels and post-prandial blood sugar.
**[3]** Prevalence of vitamin D deficiency, insufficiency and sufficiency.
**[4]** Association between vitamin D status and demographic or clinical variables.

**Statistical analysis:**
Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software version 26.0 (IBM Corp., Armonk, New York, USA). Quantitative variables were expressed as mean ± standard deviation. Categorical variables were expressed as frequency and percentage. Pearson correlation coefficient was used to assess the relationship between serum vitamin D levels and glycemic parameters. Chi-square test was used to evaluate associations between categorical variables, while one-way ANOVA was applied where appropriate. A p-value <0.05 was considered statistically significant.

**Ethical considerations:**
The study was approved by the Institutional Ethics Committee of Government Medical College, Datia, Madhya Pradesh (Approval No. 1376/Pharma/IECBMHR/2024 dated 08/04/2024). Written informed consent was obtained from all participants before enrollment. Confidentiality of patient information was maintained throughout the study.

**Results:**
A total of 250 patients with Type 2 Diabetes Mellitus were included in the present study to evaluate the correlation between serum vitamin D levels and glycated hemoglobin (HbA1c). The analysis included assessment of sociodemographic characteristics, anthropometric parameters, glycemic profile, vitamin D status and their associations. The study population predominantly consisted of middle-aged males with urban residence. Obesity was highly prevalent, affecting 60% of participants, indicating a substantial burden of excess body weight among diabetic patients **(Table 1).** Most participants had poor glycemic control with a mean HbA1c of 8.19%. Vitamin D deficiency or insufficiency was present in 82.8% of patients, highlighting the high prevalence of hypovitaminosis D among individuals with Type 2 Diabetes Mellitus **(Table 2).** No

statistically significant association was observed between vitamin D status and residence, BMI category, or duration of diabetes (p>0.05), indicating that vitamin D deficiency was common across all participant subgroups **(Table 3).** A highly significant association was observed between vitamin D status and glycemic control (p<0.001). Patients with poor glycemic control had a markedly higher prevalence of vitamin D deficiency. Serum vitamin D levels showed significant inverse correlations with fasting blood sugar, post-prandial blood sugar and HbA1c, with the strongest correlation observed for HbA1c (r = −0.701), indicating that lower vitamin D levels were associated with poorer long-term glycemic control **(Table 4)**.

**Table 1:** Sociodemographic and Anthropometric Profile of Study Participants (N = 250)

| Variable | n | (%) |
|---|---|---|
| **Age Distribution** | | |
| **30–40 years** | 54 | 21.6 |
| **41–50 years** | 59 | 23.6 |
| **51–60 years** | 57 | 22.8 |
| **61–70 years** | 48 | 19.2 |
| **71–75 years** | 32 | 12.8 |
| **Mean Age (years)** | 52.94 ± 13.35 | |
| **Gender** | | |
| **Male** | 148 | 59.2 |
| **Female** | 102 | 40.8 |
| **Residence** | | |
| **Urban** | 153 | 61.2 |
| **Rural** | 97 | 38.8 |
| **BMI Category** | | |
| **Normal (<23 kg/m²)** | 37 | 14.8 |
| **Overweight (23–27.5 kg/m²)** | 63 | 25.2 |
| **Obese (>27.5 kg/m²)** | 150 | 60.0 |

**Table 2**: Diabetes duration, glycemic profile and serum vitamin D status (N = 250)

| Parameter | Value |
|---|---|
| **Mean Duration of Diabetes (years)** | 7.70 ± 4.51 |
| **HbA1c <7%** | 61 (24.4%) |
| **HbA1c ≥7%** | 189 (75.6%) |
| **Mean Fasting Blood Sugar (mg/dL)** | 170.41 ± 29.44 |
| **Mean Post-Prandial Blood Sugar (mg/dL)** | 241.24 ± 42.11 |
| **Mean HbA1c (%)** | 8.19 ± 1.30 |
| **Mean Serum Vitamin D (ng/mL)** | 21.95 ± 9.74 |
| **Vitamin D Deficiency (<20 ng/mL)** | 114 (45.6%) |
| **Vitamin D Insufficiency (20–30 ng/mL)** | 93 (37.2%) |
| **Vitamin D Sufficiency (>30 ng/mL)** | 43 (17.2%) |
| **Deficiency + Insufficiency** | 207 (82.8%) |

**Table 3:** Association of serum vitamin D category with sociodemographic and clinical variables (N = 250)

| Variable | Deficient <20 ng/mL n (%) | Insufficient 20–30 ng/mL n (%) | Sufficient >30 ng/mL n (%) | Statistical Test |
|---|---|---|---|---|
| **Residence** | | | | |
| **Urban (n=153)** | 64 (41.8) | 60 (39.2) | 29 (19.0) | $\chi^2$=2.365, p=0.306 |
| **Rural (n=97)** | 50 (51.5) | 33 (34.0) | 14 (14.4) | |
| **BMI Category** | | | | |
| **Normal (n=37)** | 17 (45.9) | 14 (37.8) | 6 (16.2) | $\chi^2$=0.240, p=0.993 |
| **Overweight (n=63)** | 30 (47.6) | 23 (36.5) | 10 (15.9) | |
| **Obese (n=150)** | 67 (44.7) | 56 (37.3) | 27 (18.0) | |
| **Duration of Diabetes (years)** | 7.83 ± 4.42 | 7.26 ± 4.31 | 8.33 ± 4.96 | F=0.909, p=0.404 |

**Table 4**: Association of HbA1c category with vitamin D status and correlation of serum vitamin D with glycemic parameters (N = 250)

| HbA1c Category | Deficient <20 ng/mL n (%) | Insufficient 20–30 ng/mL n (%) | Sufficient >30 ng/mL n (%) | Statistical Test |
|---|---|---|---|---|
| **HbA1c <7% (Good control) (n=61)** | 10 (16.4%) | 23 (37.7%) | 28 (45.9%) | |
| **HbA1c ≥7% (Poor control) (n=189)** | 104 (55.0%) | 70 (37.0%) | 15 (7.9%) | $\chi^2$ = 53.744, df = 2, p <0.001* |

**Discussion:**

Type 2 Diabetes Mellitus (T2DM) continues to represent a major global health challenge because of its increasing prevalence and associated complications. The present cross-sectional study evaluated the relationship between serum vitamin D levels and glycemic control among patients with T2DM in the Datia region and showed a high prevalence of hypovitaminosis D along with a significant inverse association between vitamin D levels and glycemic parameters. These findings support the growing evidence that vitamin D may play an important role in glucose metabolism and diabetes management. The study population predominantly comprised middle-aged and elderly individuals with a mean age of 52.94 ± 13.35 years and males accounted for 59.2% of participants. This demographic profile is comparable with national and international diabetes statistics, which indicate that the burden of T2DM is greatest among middle-aged adults owing to cumulative exposure to insulin resistance, obesity, sedentary behavior and metabolic risk factors **[1, 2].** The predominance of urban residents observed in the present study further reflects the influence of urbanization, dietary changes and reduced physical activity on diabetes prevalence. Obesity was highly prevalent among study participants, with 60% classified as obese. Obesity is recognized as a major contributor to insulin resistance and chronic inflammation, both of which are central to the pathogenesis of T2DM. Moreover, obesity has also been associated with reduced bioavailability of vitamin D due to sequestration of the vitamin within adipose tissue **[3].** However, despite the high prevalence of obesity, the present study did not show a significant association between BMI category and vitamin D status (p=0.993). Similar findings have been reported by Ghavam *et al.* and Zhu *et al.*, who observed that vitamin D deficiency may occur across all BMI categories, suggesting that factors beyond adiposity contribute to hypovitaminosis D in diabetic populations **[3, 10].** One of the most important findings of the present study was the high prevalence of vitamin D deficiency and insufficiency. The mean serum vitamin D concentration was 21.95 ± 9.74 ng/mL, while 82.8% of participants exhibited either deficiency or insufficiency. These findings are consistent with previous studies showing widespread vitamin D deficiency among patients with T2DM despite residence in regions with abundant sunlight exposure **[4, 5].** Factors such as limited outdoor activity, increased indoor

occupations, skin pigmentation, inadequate dietary intake and obesity have been proposed to explain the persistence of vitamin D deficiency in developing countries including India **[13, 15]**. The present study demonstrated that only 24.4% of patients achieved good glycemic control, whereas 75.6% had HbA1c values ≥7%, indicating poor diabetes control. Furthermore, a highly significant association was observed between vitamin D status and HbA1c category ($\chi^2$ = 53.744, p<0.001). Among patients with good glycemic control, nearly half had sufficient vitamin D levels, whereas vitamin D deficiency predominated among patients with poor glycemic control. Similar associations have been reported by Haq and Khattak, Ahi *et al.* and Ghavam *et al.*, who showed significantly lower vitamin D levels among patients with elevated HbA1c values **[9–11]**. These observations suggest that vitamin D status may influence long-term glycemic control and could serve as a useful biomarker of metabolic health. The biological mechanisms underlying this relationship are multifactorial. Vitamin D receptors are expressed in pancreatic β-cells, skeletal muscle, adipose tissue and immune cells involved in glucose homeostasis. Vitamin D contributes to insulin secretion by regulating calcium-dependent intracellular signaling pathways and may enhance insulin sensitivity through modulation of insulin receptor expression **[6, 13 and 16].** Additionally, vitamin D exerts anti-inflammatory and immunomodulatory effects that may reduce chronic low-grade inflammation, an established contributor to insulin resistance and progression of T2DM **[15, 16].** These mechanisms provide biological plausibility for the observed association between vitamin D deficiency and poor glycemic control. Correlation analysis in the present study revealed significant negative associations between serum vitamin D levels and fasting blood sugar (r = −0.620), post-prandial blood sugar (r = −0.632) and HbA1c (r = −0.701), all of which were highly significant (p<0.001). The strongest correlation was observed with HbA1c, suggesting that vitamin D status is more closely related to long-term glycemic control than to single-point glucose measurements. Similar inverse correlations have been reported by several observational studies and systematic reviews evaluating vitamin D and diabetes outcomes **[4, 8].** Similarly, Mughal *et al.* reported that vitamin D deficiency was prevalent among patients with T2DM and that individuals with poor glycemic control had significantly lower serum vitamin D levels than those with good glycemic control; an inverse correlation was also observed between vitamin D and HbA1c **[17]**. Xu *et al.*, in their bibliometric analysis, identified vitamin D and glycemic regulation as one of the most actively investigated areas of diabetes research over the last two decades, reflecting increasing scientific interest in this relationship **[18].** Recent meta- analyses and systematic reviews have further strengthened the evidence supporting a beneficial role of vitamin D in glucose metabolism. Farahmand *et al.* reported that vitamin D supplementation resulted in modest but significant improvements in HbA1c and fasting blood glucose among patients with T2DM **[4]**. Similar findings were reported by Afraie *et al.*, Sim *et al.* and Cheng *et al.*, who observed improvements in glycemic indices and insulin resistance following vitamin D supplementation, particularly among individuals with baseline vitamin D deficiency **[5, 7 and 8].** Nevertheless, evidence from intervention studies remains inconsistent. Danasekaran *et al.*, in a double-blind randomized controlled trial among vitamin D-deficient patients with T2DM, found no significant difference in glycemic control between vitamin D supplementation and placebo groups at 3 and 6 months **[19].** Such discrepancies may be attributed to differences in supplementation regimens, baseline vitamin D status, study duration, ethnicity and glycemic control at enrollment **[6, 13].** The present study has certain limitations. The cross-sectional design precludes establishment of causality between vitamin D deficiency and poor glycemic control. Information regarding dietary habits, physical activity, sun exposure and seasonal variation in vitamin D levels was not assessed. Additionally, being a single-center study, the findings may not be generalizable to all populations. Despite these limitations, the study benefits from a relatively large sample size and standardized biochemical measurements, enhancing the reliability of the findings. Overall, the present study shows a high prevalence of hypovitaminosis D among patients with T2DM and identifies a strong inverse relationship between vitamin D levels and glycemic control. These findings support the potential role of vitamin D assessment as part of comprehensive diabetes care and highlight the need for future longitudinal and interventional studies to determine whether correction of vitamin D deficiency can improve long-term metabolic outcomes.

**Conclusion:**

Vitamin D deficiency or insufficiency was present in 82.8% of patients and was significantly associated with poor glycemic control in individuals with Type 2 Diabetes Mellitus. Lower serum vitamin D levels showed significant inverse correlations with fasting blood sugar, post-prandial blood sugar and glycated hemoglobin, indicating worsening metabolic control. These findings support routine vitamin D assessment as an adjunct to diabetes management, while larger prospective studies are needed to establish therapeutic benefits.

**References:**

**[1]** https://www.who.int/news-room/fact-sheets/detail/diabetes
**[2]** https://idf.org/about-diabetes/facts-figures/
**[3]** Zhu A *et al. Nutrients.* 2022 **14**:3282. [PMID: 36014788]
**[4]** Farahmand MA *et al. BMC Endocr Disord.* 2023 **23**:15. [PMID: 36647067]
**[5]** Cheng L *et al. Front Nutr.* 2024 **11**:1462535. [PMID: 39525505]
**[6]** Haroon NN *et al. J Diabetes Metab Disord.* 2015 **14**:3. [PMID: 25722966]
**[7]** Afraie M *et al. J Diabetes Res.* 2024 **2024**:9960656. [PMID: 39290798]
**[8]** Sim G *et al. Exp Ther Med.* 2024 **28**:451. [PMID: 39421597]
**[9]** Ahi S *et al. PLoS One.* 2025 **20**:e0324729. [PMID: 40435133]
**[10]** Ghavam S *et al. J Family Med Prim Care.* 2018 **7**:1314. [PMID: 30613518]

**[11]** Haq AU & Bilal Khattak M. *Cureus.* 2025 **17**:e99160. [PMID: 41541973]
**[12]** Dahal T *et al. South Asian J Health Prof.* 2026 **9**:66. [DOI: 10.18231/j.sajhp.67348.1784367453]
**[13]** Saadon HB. *Bull Natl Res Cent.* 2025 **49**:82. [DOI: 10.1186/s42269-025-01377-5]
**[14]** Neeraj RK *et al. Eur J Cardiovasc Med.* 2025 **15**:71. [DOI: 10.61336/ejcm/25-10-14]
**[15]** Młynarska E *et al. Pharmaceuticals.* 2025 **18**:1467. [DOI: 10.3390/ph18101467]
**[16]** Fuentes-Barría H *et al. Int J Mol Sci.* 2025 **26**:2153. [PMID: 40076782]
**[17]** Mughal HMF *et al. Cureus.* 2025 **17**:e92179. [PMID: 41098282]
**[18]** Xu R *et al. Front Endocrinol.* 2024 **15**:1421953. [PMID: 39193371]
**[19]** Danasekaran R *et al. Natl J Community Med.* 2024 **15**:519. [DOI: 10.55489/njcm.150720244069]

*Caveat Emptor is applicable among the literate community where required and possible. The publisher, its journal, editors and the internal/external reviewers take adequate steps to check, evaluate, correct, edit, revise and improve content where possible and required.*