

Prevalence & Determinants of Low Testosterone Levels in Men with Type 2 Diabetes Mellitus Presenting to a Tertiary Care Hospital

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HOW TO CITE:

Erigela Mallikarjuna Reddy, Suresh Kanna Subramaniam, Arvindraj R, Akita Gopinath, Gnanaprakash C (2026). Prevalence & Determinants of Low Testosterone Levels in Men with Type 2 Diabetes Mellitus Presenting to a Tertiary Care Hospital. International Journal of Special Education, 41(4s), 709-734.

ABSTRACT:

Introduction: The association between Type 2 Diabetes Mellitus (T2DM) and male hypogonadism is increasingly recognized. However, data regarding its prevalence and determinants remain limited in certain populations. Understanding this relationship is essential for improving comprehensive diabetic care and addressing associated complications such as erectile dysfunction and reduced quality of life.

Aim & Objective: The study aimed to assess the prevalence and determinants of low testosterone (LowT) in men with Type 2 Diabetes Mellitus (T2DM). It included evaluation of erectile dysfunction (ED) using the SHIM scale, identification of potential predictors such as body mass index (BMI) and lifestyle factors, and determination of the association between diabetic status and testosterone levels.

Materials & Methods: A cross-sectional study was conducted over 18 months at a tertiary care hospital in Chennai. A total of 329 patients with T2DM were recruited using purposive sampling. Data collection included demographic details, clinical history, and assessment of erectile dysfunction using the SHIM questionnaire. Laboratory investigations measured serum total, free, and bioavailable testosterone levels. Statistical analysis was performed using SPSS version 21.

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Results: The prevalence of LowT was 74.8%. Erectile dysfunction was observed in 94.2% of participants, with severity significantly associated with LowT ($p < 0.001$). No significant associations were found between LowT and age, occupation, smoking, alcohol consumption, physical activity, BMI, or glycemic parameters (fasting plasma glucose and HbA1c). However, strong associations were noted between LowT and psychological, somatic, and sexual symptom severity ($p < 0.001$). Waist circumference showed a trend toward association ($p = 0.068$).

Conclusion: There is a high prevalence of hypogonadism and erectile dysfunction among men with T2DM. Testosterone deficiency is strongly associated with symptom severity but not with conventional metabolic or lifestyle factors alone. Routine screening for hypogonadism should be incorporated into the management of diabetic men to improve overall quality of life.

Keywords: Hypogonadism, Type 2 Diabetes Mellitus, Testosterone, Erectile Dysfunction, SHIM Scale, Prevalence.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and progressive β -cell dysfunction. In the early stages, skeletal muscle, adipose tissue, and the liver become resistant to insulin, resulting in impaired glucose uptake and continued hepatic glucose production despite hyperglycemia. This leads to compensatory hyperinsulinemia, which over time causes β -cell stress, dysfunction, and eventual decline in insulin secretion capacity [1]. Persistent hyperglycemia, along with elevated free fatty acids, induces glucolipotoxicity, triggering oxidative stress, inflammation, and endoplasmic reticulum dysfunction, ultimately worsening β -cell failure and promoting diabetic complications through mechanisms such as advanced glycation end products (AGEs) and protein kinase C activation [2].

Genetic predisposition contributes to T2DM susceptibility, particularly genes regulating β -cell function and insulin processing. However, environmental factors—especially obesity, sedentary lifestyle, and high-calorie diets—play a dominant role in the rising global prevalence. Adipose tissue releases adipokines and proinflammatory cytokines that exacerbate insulin resistance and create a chronic low-grade

inflammatory state [3]. Visceral adiposity is particularly associated with metabolic dysfunction due to its increased lipolytic activity and portal circulation effects. Early lifestyle interventions can improve insulin sensitivity and delay disease progression [4].

T2DM has reached pandemic proportions, affecting over half a billion individuals worldwide, with projections nearing one billion by mid-century. The burden is rapidly increasing in low- and middle-income countries such as India, where over 10% of adults are affected, often at a younger age, increasing the lifetime risk of complications [5,6].

Testosterone plays a crucial role in male metabolic and sexual health. Produced by Leydig cells under luteinizing hormone stimulation, testosterone circulates as free, albumin-bound, and sex hormone-binding globulin (SHBG)-bound fractions, with the bioavailable portion exerting physiological effects [7]. It influences muscle mass, bone density, fat distribution, and metabolism. Testosterone enhances insulin sensitivity by promoting glucose uptake and modulating insulin signaling pathways. Low testosterone levels are associated with insulin resistance, metabolic syndrome, and increased cardiovascular risk [8,9]. Sexual function is closely linked to testosterone levels, with deficiency leading to reduced libido,

erectile dysfunction (ED), and impaired spermatogenesis. These effects are mediated through central and peripheral mechanisms involving androgen receptors and hypothalamic–pituitary–gonadal axis regulation [10]. Evaluation requires morning testosterone measurement along with SHBG assessment, and management includes lifestyle modification or testosterone replacement therapy in selected cases [11].

Hypogonadism is common in men with T2DM and is typically functional rather than primary. It is associated with suppression of the hypothalamic–pituitary–gonadal axis due to hyperinsulinemia, inflammation, leptin resistance, and increased aromatase activity in visceral fat, leading to conversion of testosterone to estradiol [12]. Comorbid conditions such as obesity, obstructive sleep apnea, and medication use further contribute. Approximately one-third of diabetic men exhibit low testosterone levels, with prevalence increasing with obesity and disease duration.

Clinically, hypogonadism worsens metabolic outcomes by increasing visceral adiposity, reducing muscle mass, and aggravating insulin resistance. It is strongly associated with erectile dysfunction, reduced bone density, fatigue, and poor quality of life. Furthermore, low testosterone has been linked to increased cardiovascular risk and disease progression in diabetic patients [13]. Identification and management of hypogonadism can therefore improve both metabolic and functional outcomes. Screening is recommended in symptomatic individuals, and addressing reversible causes may restore hormonal balance, while testosterone therapy may benefit selected patients [14].

T2DM is also associated with broader endocrine disturbances involving adipokines, cortisol, incretins, and sex hormones, creating a complex hormonal network that perpetuates metabolic dysfunction [15]. Evaluating these hormonal imbalances can guide targeted therapy, improve glycemic control, and reduce complications. Hormonal markers also aid in risk stratification and early intervention for cardiovascular and metabolic diseases [16,17]. Advances in understanding endocrine pathways have contributed to novel

therapeutic strategies, emphasizing a holistic approach to diabetes management [18].

This study aims to determine the prevalence of low testosterone and its determinants in men with T2DM. It also evaluates erectile dysfunction using the Sexual Health Inventory for Men (SHIM), examines predictors such as BMI and lifestyle factors, and assesses the relationship between glycemic status and testosterone levels to inform improved screening and management strategies.

MATERIALS & METHODS

This hospital-based cross-sectional analytical study was conducted over 18 months (July 2024–December 2025) in the Department of General Medicine at a tertiary care teaching hospital in Chennai, India, to determine the prevalence and determinants of low testosterone (LowT) among men with Type 2 Diabetes Mellitus (T2DM). Adult male patients (>18 years) diagnosed with T2DM as per WHO criteria (fasting plasma glucose ≥ 126 mg/dL, 2-hour plasma glucose ≥ 200 mg/dL, or HbA1c consistent with diabetes) were included, while those with conditions or treatments affecting testosterone levels (e.g., androgen therapy, steroids, chronic renal failure, prostate disease) were excluded. A sample size of 329 was achieved using purposive sampling. Data were collected using a structured proforma, including demographic and clinical details, and erectile dysfunction was assessed using the SHIM questionnaire (score <17 indicating ED). All eligible participants underwent laboratory evaluation including serum total, free, and bioavailable testosterone levels. Data were analyzed using SPSS version 21, with descriptive statistics (mean, standard deviation, frequencies) used to summarize variables. Associations between LowT and clinical or lifestyle factors were assessed using chi-square and independent t-tests, while multivariate regression analysis identified independent predictors after adjusting for confounders. A p-value <0.05 was considered statistically significant. Ethical approval was obtained, and informed consent was secured from all participants. Quality control was ensured through standardized measurement protocols, calibrated instruments, and laboratory testing in a NABL-accredited facility.

RESULT

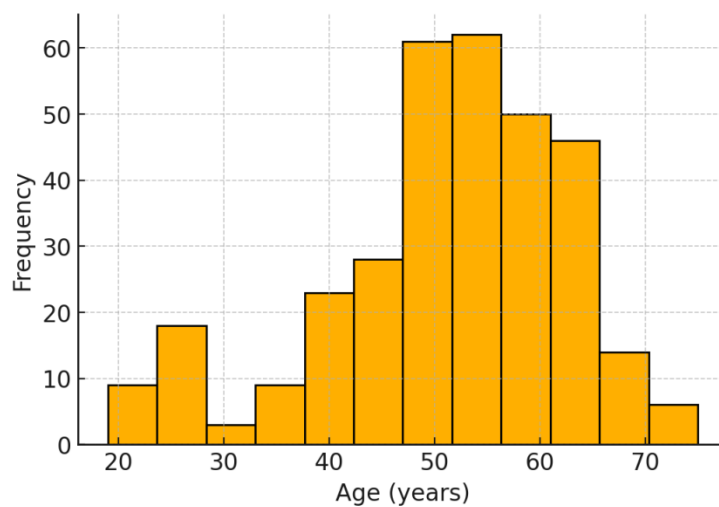


Figure 1: Distribution of Age Among T2DM Patients

Figure 1 included 329 patients with Type 2 Diabetes Mellitus aged 19–75 years, with a mean age of 51.10 ± 11.59 years, indicating a predominantly middle-aged population. The wide age range reflects

inclusion of both early- and late-onset cases, with moderate variability suggesting a heterogeneous age distribution.



Figure 2: Occupational Distribution of the Study Population

Figure 2 evaluates the study included 329 participants, with farmers forming the largest group (45.6%), followed by unemployed individuals

(18.8%), traders (18.5%), and civil servants (17.0%). This distribution indicates a predominantly agrarian population with notable socioeconomic variation.

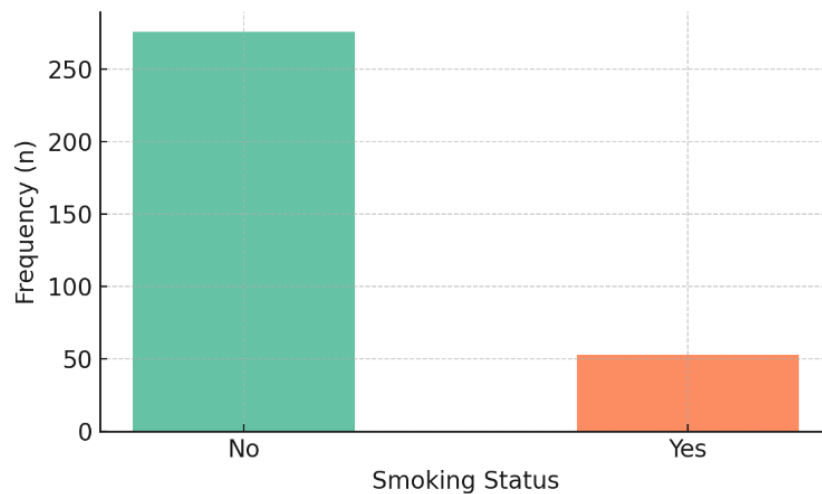


Figure 3: Smoking Status Among Study Participants

Figure 3 shows that 83.9% were non-smokers and 16.1% were smokers, indicating a low prevalence of smoking and a predominance of healthier lifestyle habits in the cohort.

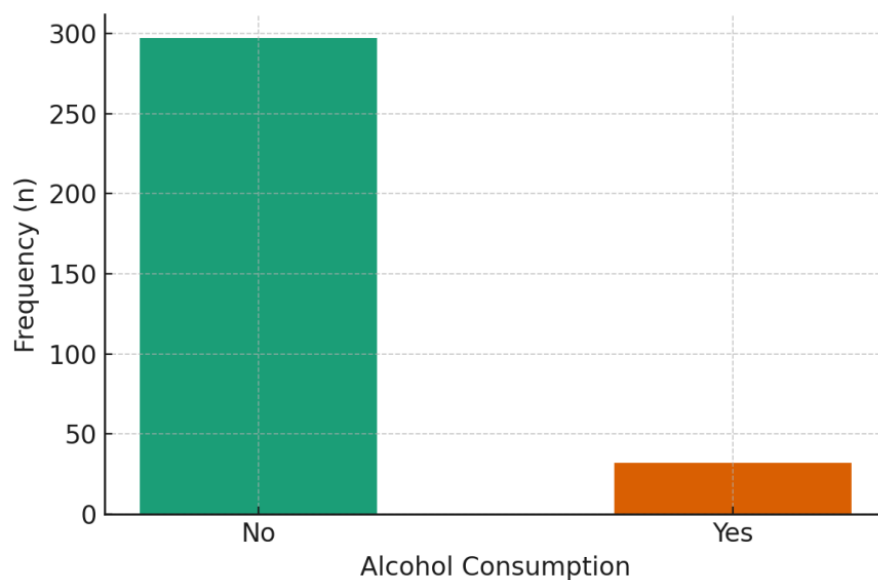


Figure 4: Alcohol Consumption Among Study Participants

Figure 4 shows that 78.4% were non-alcoholic while 21.6% consumed alcohol, indicating a predominantly abstinent population with a smaller subgroup that may influence metabolic and cardiovascular outcomes.

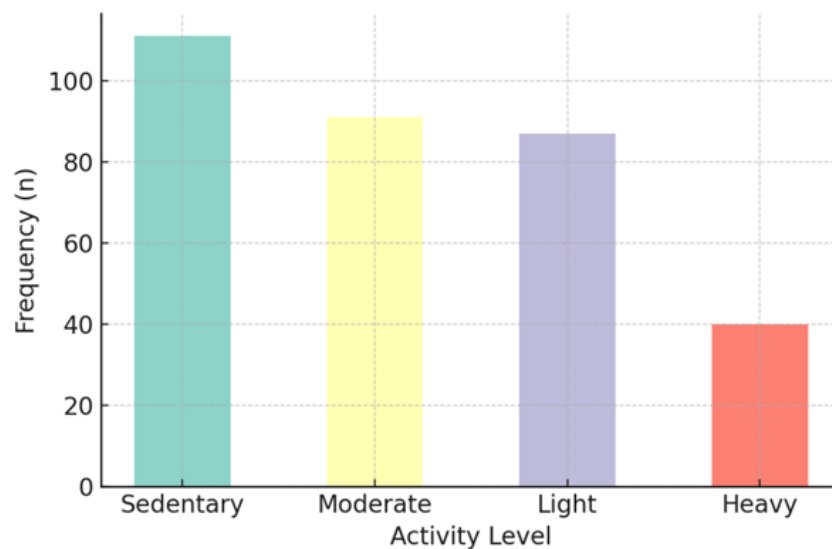


Figure 5: Activity Level Among Study Participants

Figure 5 Among 329 participants, sedentary activity was most common (33.7%), followed by moderate (27.7%), light (26.4%), and heavy activity (12.2%), indicating a predominantly sedentary lifestyle that may contribute to metabolic risk and diabetes-related complications.

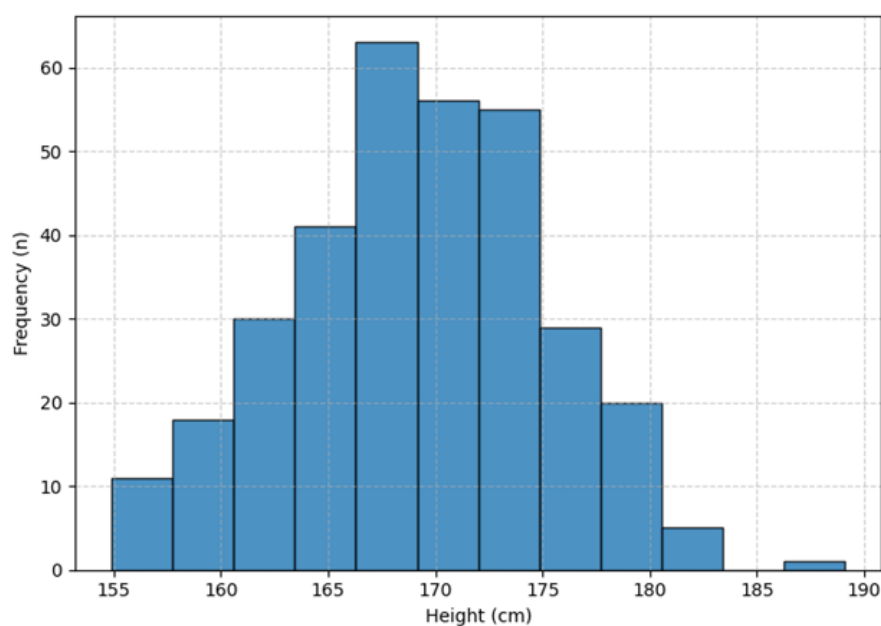


Figure 6: Distribution of Height Among Study Participants

Figure 6 shows that height among the 329 participants ranged from 154.9 cm to 189.1 cm, with most individuals clustered between 165–175 cm. The distribution was approximately normal, with a mean of 169.14 ± 5.91 cm and a median of 169.3 cm, indicating a symmetrical pattern with minimal skewness.

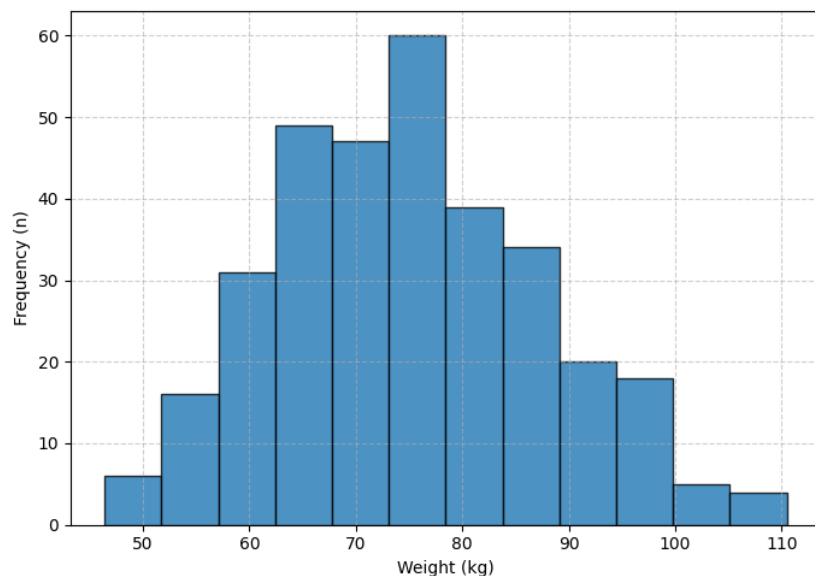


Figure 7: Distribution of Weight Among Study Participants

Figure 7 shows that body weight among the 329 participants ranged from 46.4 kg to 110.5 kg, with a mean of 74.98 ± 12.67 kg and a median of 74.4 kg. Most individuals weighed between 60–90 kg, with an approximately normal distribution, moderate variability, and minimal skewness.

Mean	98.55
Standard Deviation (SD)	6.37
Minimum	81.4
Maximum	116.7
Variance	40.6
Range	35.3

Table 1: Descriptive Statistics Summary of Body Mass Index (BMI)

Statistic	Value
Count	329
Mean	26.18
Standard Deviation (SD)	4.01
Minimum	18
Maximum	37.6
Variance	16.09
Range	19.6

Table 1 shows that BMI among the 329 participants ranged from 18.0 to 37.6 kg/m², with a mean of 26.18 ± 4.01 kg/m² and a median of 26.1 kg/m². Most individuals had BMI between 22–30 kg/m², with an approximately normal distribution, moderate variability, and minimal skewness.

Table 2: Descriptive Statistics Summary of Waist Circumference (cm)

Statistic	Value
Count	329

Table 2 shows waist circumference ranging from 81.4 to 116.7 cm, with a mean of 98.55 ± 6.37 cm and a median of 98.5 cm. The distribution is approximately normal, with most values clustered between 92 and 105 cm, indicating moderate variability in central obesity among participants.

Table 3: Descriptive Statistics Summary Of T2DM Duration Yrs

Statistic	Value
Count	329
Mean	5.66
Standard Deviation (SD)	3.97
Minimum	0.3
Maximum	20
Median	5
Variance	15.78
Range	19.7

Table 3 shows that T2DM duration ranged from <1 to 20 years, with a mean of about 5 years. Most patients had diabetes for less than 7 years, indicating

early to mid-stage disease, with a right-skewed distribution and fewer long-standing cases (>10 years).

Table 4: Distribution of Treatment Type among Study Participants

Treatment Type	Frequency (n)	Percentage (%)
OHA only	181	55
OHA+Insulin	84	25.5
Insulin	50	15.2

only		
Lifestyle only	14	4.3
Total	329	100

Table 4 shows that OHA-only therapy was the most common treatment (55.0%), followed by OHA + insulin (25.5%), insulin only (15.2%), and lifestyle management (4.3%), reflecting predominant use of oral agents with a substantial need for combination or insulin therapy in this T2DM cohort.

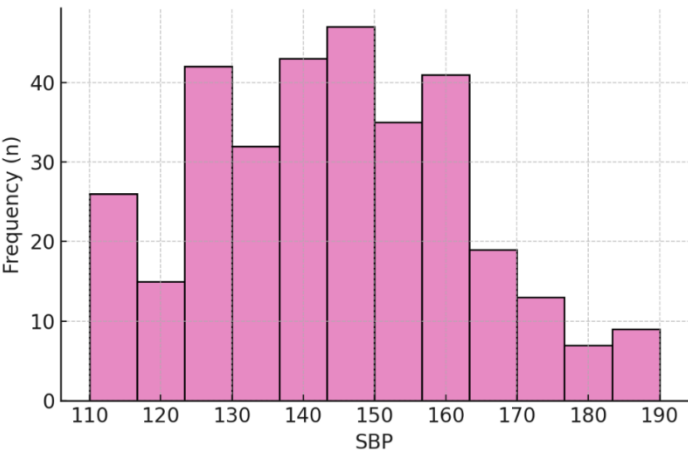


Figure 8: Distribution of Systolic Blood Pressure (SBP) Among T2DM Patients

Figure 8 shows systolic blood pressure ranging from 110 to 190 mmHg, with a mean of 144.26 ± 18.55 mmHg, indicating generally elevated and variably controlled blood pressure in the T2DM

cohort. The findings reflect a high burden of hypertension and the need for improved cardiovascular risk management.

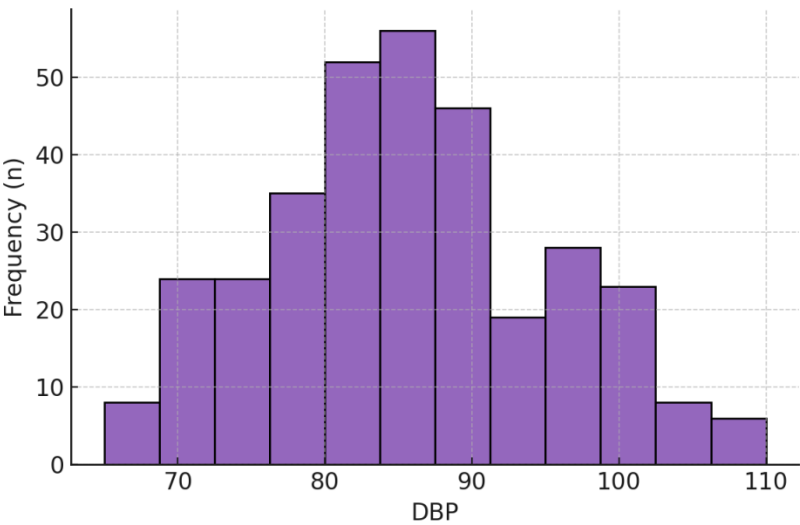


Figure 9: Distribution of DBP Among Study Participants

Figure 9 shows a near-normal distribution of diastolic blood pressure among 329 participants, ranging from ~65 to 110 mmHg, with most values clustered between 80–90 mmHg. The pattern

reflects generally borderline to mildly elevated DBP with moderate variability, including a small subgroup with poorly controlled hypertension.

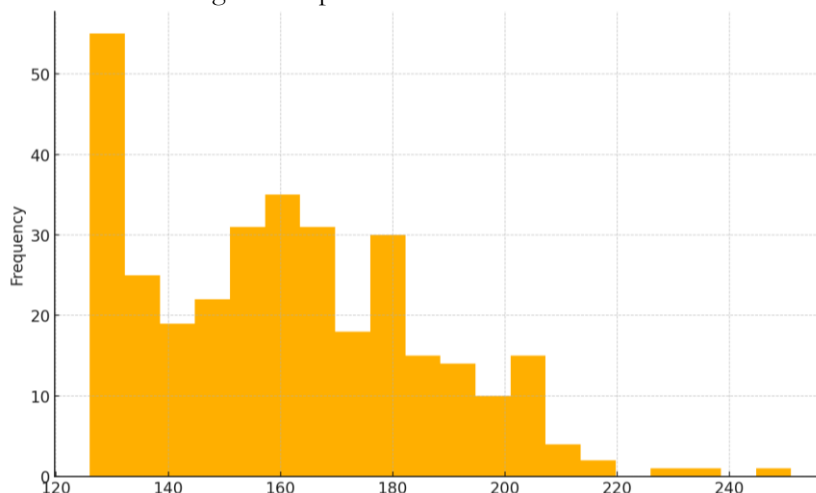


Figure 10: Distribution of FPG mg dL Among Study Participants

Figure 10 shows elevated fasting plasma glucose levels in 329 participants, with a mean of 160.38 ± 25.05 mg/dL and a range of 126–251 mg/dL. The

findings indicate overall poor glycemic control with considerable variability, reflecting heterogeneous diabetes management across the cohort.

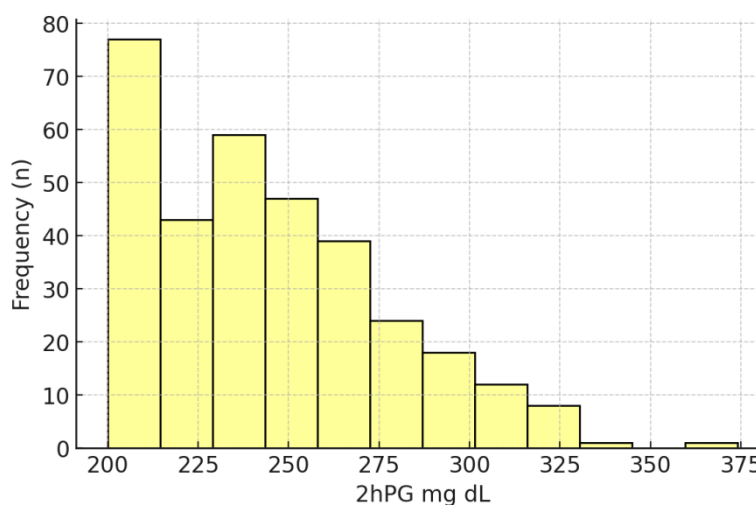


Figure 11: Distribution of 2hPG mg dL Among Study Participants

Figure 11 shows elevated 2-hour postprandial glucose levels in 329 T2DM patients, with a mean of 243.81 ± 33.58 mg/dL and a range of 200–374 mg/dL. The findings indicate poor post-meal

glycemic control and marked inter-individual variability, highlighting ongoing challenges in managing postprandial hyperglycemia.

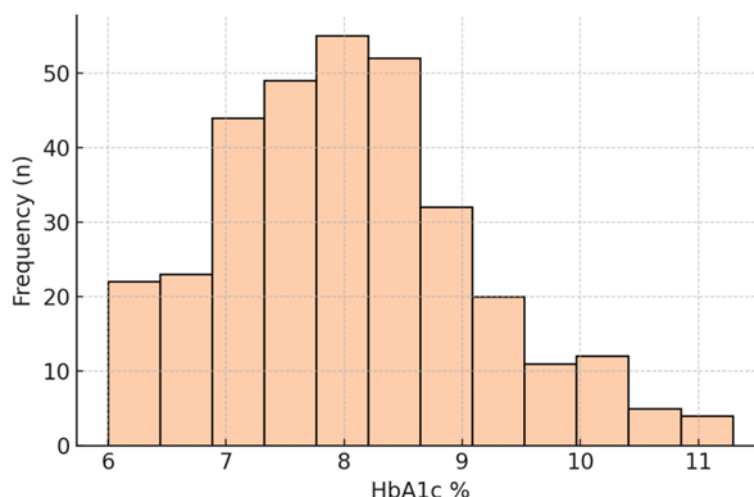


Figure 12: Distribution of HbA1c % Among Study Participants

Figure 12 shows a mean HbA1c of $8.03 \pm 1.09\%$, indicating overall poor glycemic control in the study population, with values ranging from 6.00% to 11.29%. The results reflect considerable inter-individual variation, with most participants exhibiting suboptimal diabetes control.

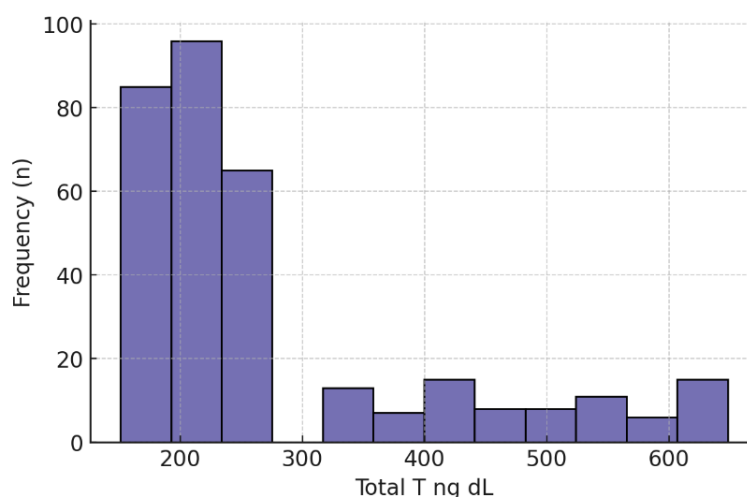


Figure 13: Distribution of Total T ng dL Among Study Participants

Figure 13 shows a mean total testosterone level of 277.58 ± 132.63 ng/dL, indicating overall reduced levels with wide variability (151–648 ng/dL). The findings suggest a general trend toward lower testosterone concentrations in this cohort, with possible clinical relevance in Type 2 Diabetes Mellitus.

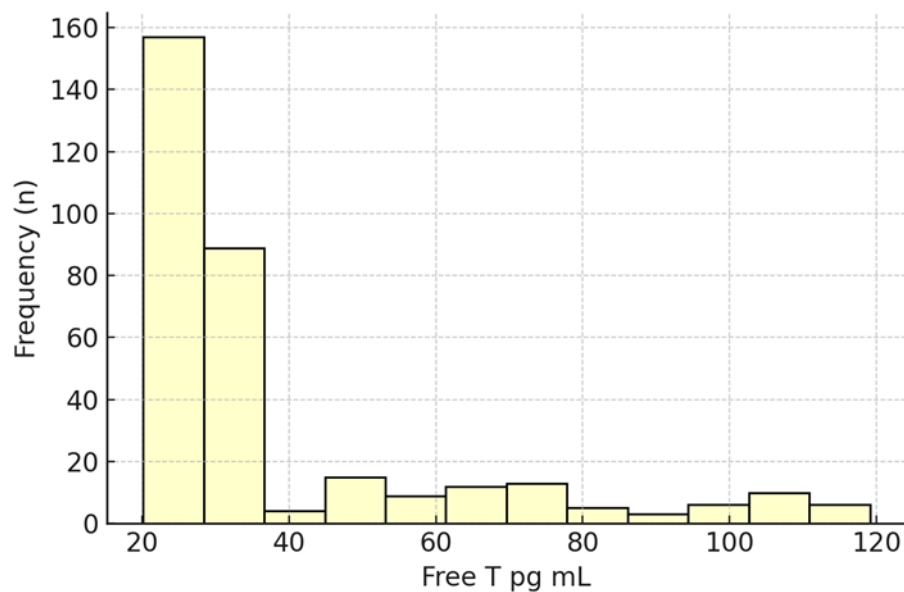


Figure 14: Distribution of Free T pg mL Among Study Participants

Figure 14 shows a mean free testosterone level of 38.92 ± 24.05 pg/mL, indicating relatively low levels with wide inter-individual variability (20.1–119.1 pg/mL). The results suggest a substantial

proportion of participants had reduced free testosterone, potentially reflecting hypogonadism in Type 2 Diabetes Mellitus.

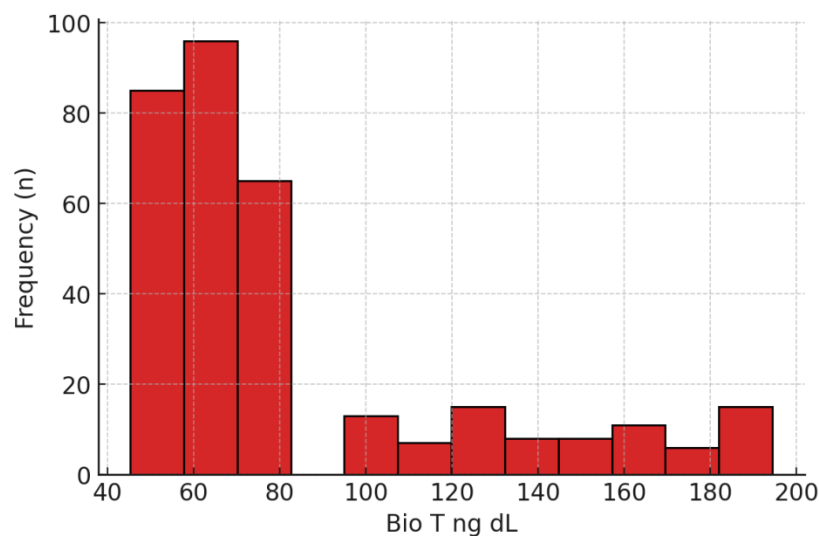


Figure 15: Distribution of Bio T ng dL Among Study Participants

Figure 15 shows a mean bioavailable testosterone level of 83.28 ± 39.79 ng/dL, indicating relatively low levels with wide variability (range 45.3–194.4 ng/dL). The findings suggest substantial inter-individual variation and a notable proportion of participants with reduced bioavailable testosterone.

Table 5: Distribution of LowT Among Study Participants

LowT	Frequency (n)	Percentage (%)
TRUE	246	74.8
FALSE	83	25.2
Total	329	100

Table 5 shows that 74.8% of participants had low testosterone, while 25.2% had normal levels, indicating a high prevalence of hypogonadism in Type 2 Diabetes Mellitus and highlighting the need for routine screening in diabetic men.

Table 6: Descriptive Statistics Summary of FSH
mIU/mL

Statistic	Value
Count	329
Mean	6.94
Standard Deviation (SD)	2.29
Minimum	1
Maximum	13.5
Variance	5.23
Range	12.5

Table 6 shows a mean FSH level of 6.94 ± 2.29 mIU/mL, within the normal physiological range, with values ranging from 1.00 to 13.50 mIU/mL. The results indicate generally preserved gonadotropic function with moderate inter-individual variability across the study population.

Table 7: Descriptive Statistics Summary of LH
mIU/mL

Statistic	Value
Count	329
Mean	5.97
Standard Deviation (SD)	1.93
Minimum	1
Maximum	10.3
Variance	3.74
Range	9.3

Table 7 shows a mean LH level of 5.97 ± 1.93 mIU/mL, within the normal physiological range, with values spanning 1.00 to 10.30 mIU/mL. The findings indicate generally preserved pituitary gonadotropic function with only mild variability in LH levels among participants.

Table 8: Descriptive Statistics Summary of SHIM
Score

Statistic	Value
Count	329
Mean	12.29

Standard Deviation (SD)	4.88
Minimum	5
Maximum	25
Variance	23.79
Range	20

Table 8 shows a mean SHIM score of 12.29 ± 4.88 , indicating overall moderate erectile dysfunction among participants, with scores ranging from 5 to 25. The wide variability reflects significant differences in sexual function, highlighting a high burden of erectile dysfunction in Type 2 Diabetes Mellitus.

Table 9: Distribution of ED Category Among
Study Participants

ED Category	Frequency (n)	Percentage (%)
Mild-to-moderate ED	132	40.1
Moderate ED	82	24.9
Severe ED	63	19.1
Mild ED	33	10
No ED	19	5.8
Total	329	100

Table 9 shows a high burden of erectile dysfunction among participants, with mild-to-moderate ED being most common (40.1%), followed by moderate (24.9%), severe (19.1%), and mild (10.0%), while only 5.8% had no ED, reflecting significant sexual dysfunction in Type 2 Diabetes Mellitus.

Table 10: Distribution of ED Present Among
Study Participants

ED Present	Frequency (n)	Percentage (%)
TRUE	310	94.2
FALSE	19	5.8
Total	329	100

Table 10 shows a very high prevalence of erectile dysfunction, with 94.2% of participants affected and only 5.8% without ED, highlighting a strong association between Type 2 Diabetes Mellitus and

erectile dysfunction and emphasizing the need for routine sexual health assessment in diabetes care.

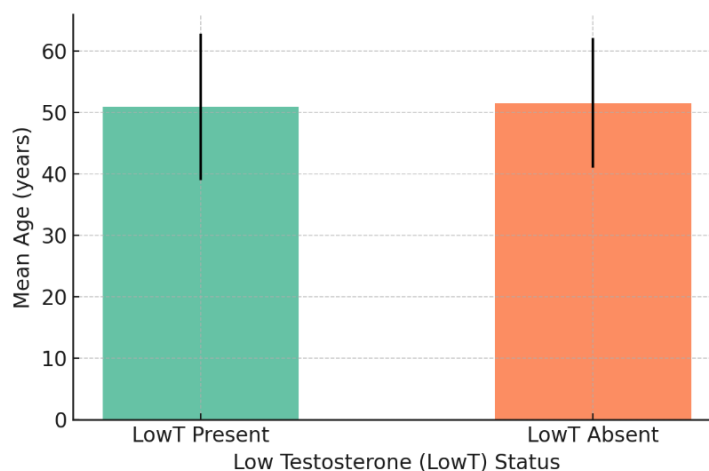


Figure 16: Comparison of Mean Age Between LowT and Non-LowT Groups

Figure 16 shows comparable mean age between LowT and normal groups (50.88 ± 11.62 vs 51.67 ± 11.47 years; $p = 0.63$), indicating that age was not significantly associated with testosterone status in this cohort.

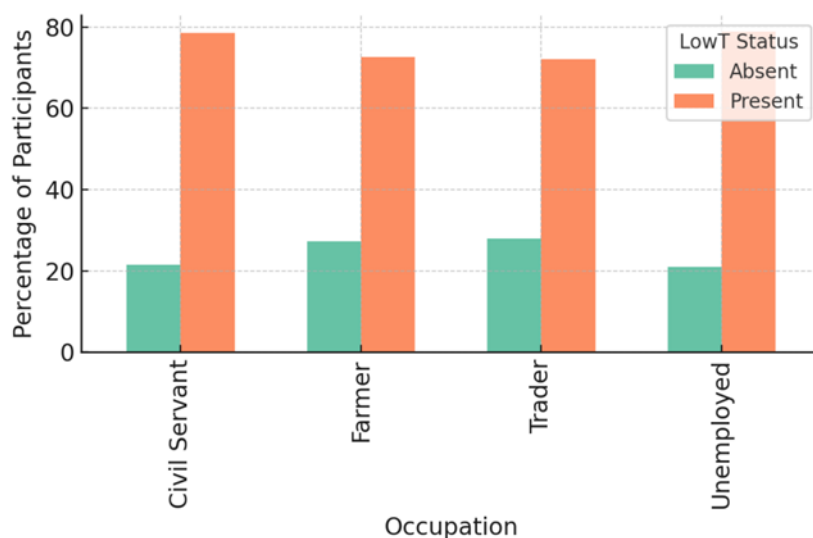


Figure 17: Association Between Occupation and Low Testosterone (LowT) Status

Figure 17 shows no significant association between occupation and testosterone status ($\chi^2 = 1.60$, $df = 3$, $p = 0.66$), indicating that occupational category does not influence LowT prevalence in this cohort.

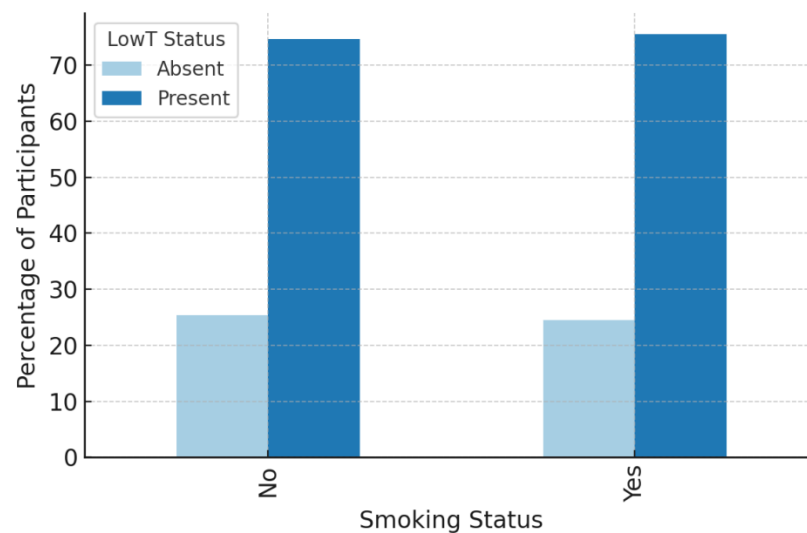


Figure 18: Association Between Smoking Status and Low Testosterone (LowT) Status

Figure 18 shows no difference in LowT prevalence between smokers and non-smokers, with no significant association observed ($\chi^2 = 0.00$, $df = 1$, $p = 1.00$), indicating that smoking status did not influence testosterone levels in this cohort.

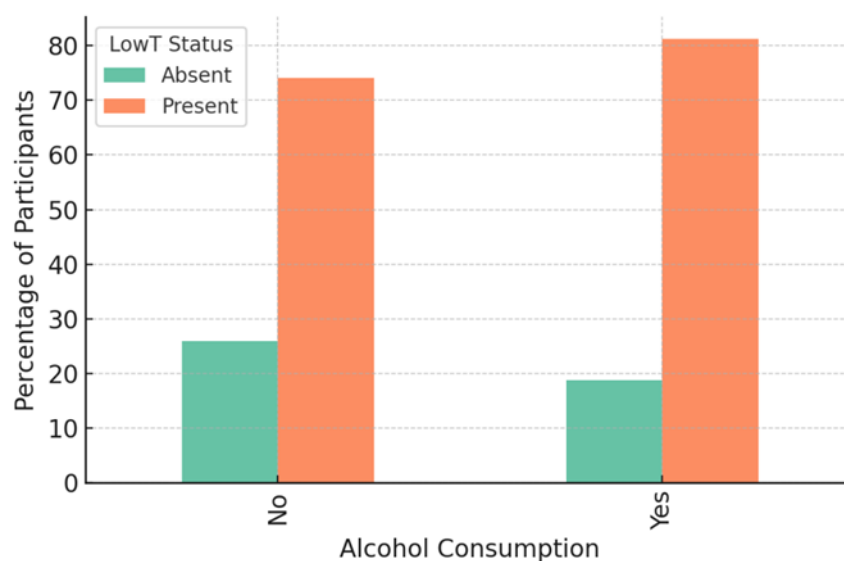


Figure 19: Comparison of Low Testosterone (LowT) by Alcohol Consumption

Figure 19 shows comparable LowT prevalence between alcohol users and non-users, with no significant association observed ($\chi^2 = 0.45$, $df = 1$, $p = 0.50$), indicating that alcohol consumption was not a determinant of testosterone deficiency in this cohort.

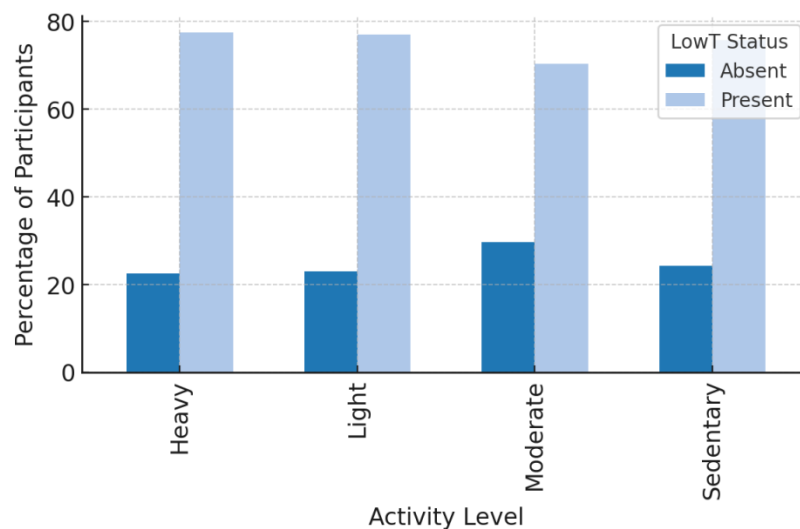


Figure 20: Comparison of Low Testosterone (LowT) Across Activity Levels

Figure 20 shows no significant association between physical activity level and LowT status ($\chi^2 = 1.39$, $df = 3$, $p = 0.71$), indicating that testosterone deficiency was independent of activity level in this study population.

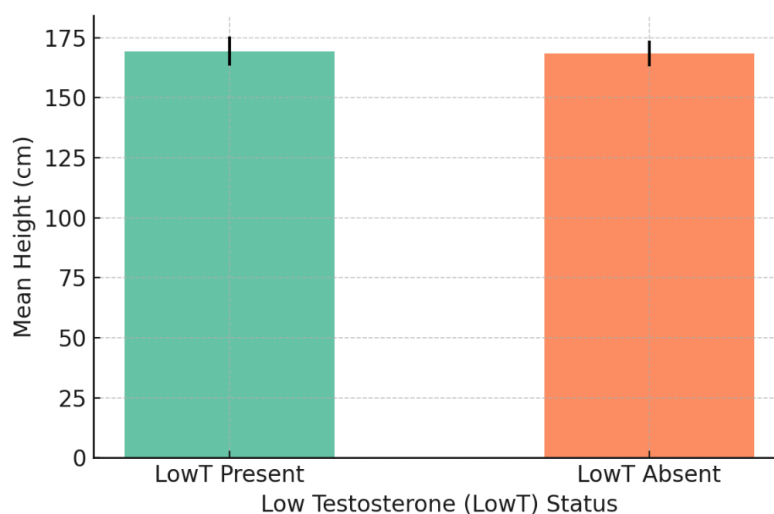


Figure 21: Comparison of Mean Height Between LowT and Non-LowT Groups

Figure 21 shows a slightly higher mean height in the LowT group, but the difference was not significant ($p = 0.22$), indicating no association between height and testosterone status in this population.

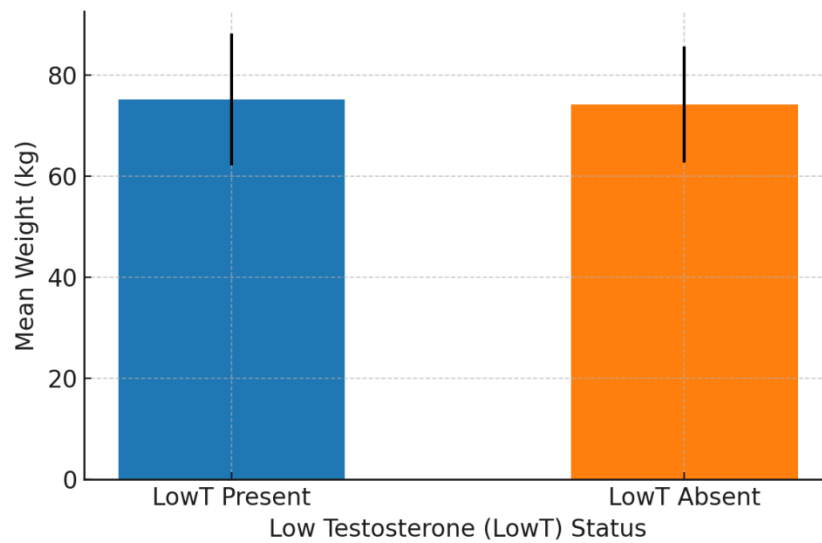


Figure 22: Comparison of Mean Weight Between LowT and Non-LowT Groups

Figure 22 shows a slightly higher mean body weight in the LowT group, but the difference was not significant ($p = 0.50$), indicating that body weight

alone does not predict testosterone status in men with Type 2 Diabetes Mellitus.

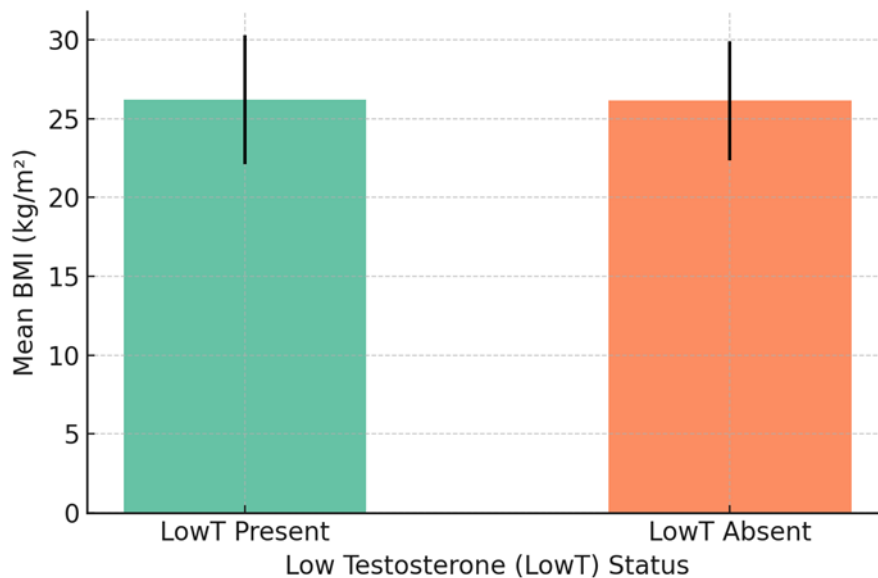


Figure 23: Comparison of Mean BMI Between LowT and Non-LowT Groups

Figure 23 shows no significant difference in BMI between LowT and normal groups (26.47 vs 26.39 kg/m²; $p = 0.90$), indicating that overall BMI was

not associated with testosterone status in this cohort.

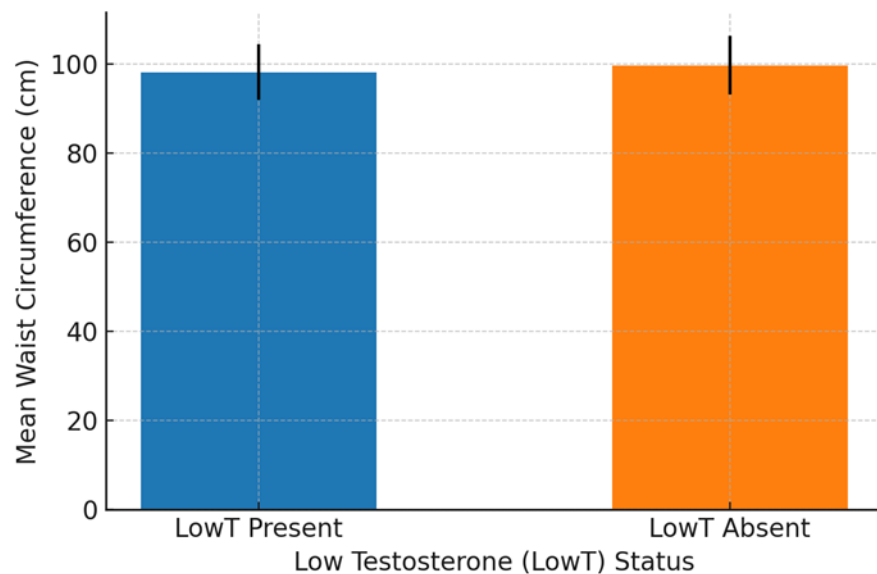


Figure 24: Comparison of Mean Waist Circumference Between LowT and Non-LowT Groups

Figure 24 shows a higher mean waist circumference in the LowT group, but the difference was not statistically significant ($p = 0.0677$). However, it suggests a trend toward greater central obesity in individuals with LowT, consistent with its association with insulin resistance and Type 2 diabetes.

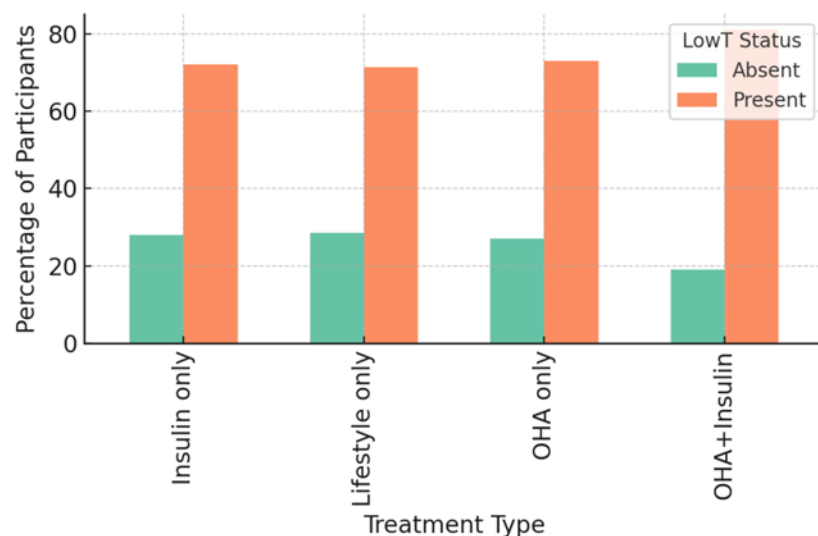


Figure 25: Comparison of Low Testosterone (LowT) Across Different Treatment Types

Figure 25 shows no significant association between diabetes treatment modality and LowT status ($\chi^2 = 2.31$, $df = 3$, $p = 0.51$), indicating that testosterone deficiency prevalence is independent of the type of diabetes management.

Table 11: Comparison of Duration of Diabetes Between Low Testosterone (LowT) Groups

LowT	N	Mean Duration (years)	SD	Minimum	Maximum
FALSE	83	5.42	3.81	0.5	18
TRUE	246	5.74	4.03	0.3	20
t test value= -0.00, p value= 0.9971					

Table 11 shows no significant difference in duration of diabetes between LowT and normal groups ($p = 0.997$), indicating that disease duration does not influence testosterone deficiency in this population.

Table 12: Comparison of Mean DBP Value Between Low Testosterone (LowT) Groups

LowT	N	Mean DBP Value	SD	Minimum	Maximum
FALSE	83	86.25	10.76	65	110
TRUE	246	85.41	9.27	65	110
t test value= -0.64, p value= 0.5249					

Table 12 shows no significant difference in diastolic blood pressure between LowT and normal groups (85.41 ± 9.27 vs 86.25 ± 10.76 mmHg; $p = 0.525$), indicating comparable DBP irrespective of testosterone status.

Table 13: Comparison of Mean FPG_mg_dL Value Between Low Testosterone (LowT) Groups

LowT	N	Mean FPG_mg_dL Value	SD	Minimum	Maximum
FALSE	83	161.98	26.72	126	251
TRUE	246	159.84	24.5	126	238
t test value= -0.64, p value= 0.5209					

Table 13 shows no significant difference in fasting plasma glucose between LowT and normal groups (159.84 ± 24.50 vs 161.98 ± 26.72 mg/dL; $p = 0.521$), indicating similar fasting glycemic levels regardless of testosterone status.

Table 14: Comparison of Mean 2hPG Between Low Testosterone (LowT) Groups

LowT	N	Mean 2hPG_mg_dL Value	SD	Minimum	Maximum
FALSE	83	243.1	35.4	200	374
TRUE	246	244.05	33.02	200	344
t = 0.22, p = 0.8290					

Table 14 shows no significant difference in 2-hour plasma glucose between LowT and normal groups (244.05 ± 33.02 vs 243.10 ± 35.40 mg/dL; $p = 0.829$), indicating comparable postprandial glucose levels irrespective of testosterone status.

Table 15: Comparison of Mean HbA1c Between Low Testosterone (LowT) Groups

LowT	N	Mean HbA1c_% Value	SD	Minimum	Maximum
FALSE	83	8.02	1.17	6	11.1
TRUE	246	8.03	1.06	6	11.29
t = 0.10, p = 0.9182					

Table 15 shows no significant difference in HbA1c vs $8.02 \pm 1.17\%$; $p = 0.918$), indicating similar between LowT and normal groups ($8.03 \pm 1.06\%$ glycemic control regardless of testosterone status.

Table 16: Comparison of Total_T_ng_dL Between Low Testosterone (LowT) Groups

LowT	N	Mean Total_T_ng_dL Value	SD	Minimum	Maximum
FALSE	83	482.27	103.75	324	648
TRUE	246	208.52	31.09	151	260
t = -23.68, p < 0.001					

Table 16 shows a highly significant difference in total testosterone, with markedly lower levels in the LowT group (208.52 ± 31.09 vs 482.27 ± 103.75 ng/dL; $p < 0.001$), confirming robust group classification and hypogonadal status.

Table 17: Comparison of Mean Free_T_pg_mL Value Between Low Testosterone (LowT) Groups

LowT	N	Mean Free_T_pg_mL Value	SD	Minimum	Maximum
FALSE	83	74.66	23.15	43	119.1
TRUE	246	26.85	4.03	20.1	33.9
t = -18.72, p < 0.001					

Table 17 shows a highly significant reduction in free testosterone in the LowT group compared to normal (26.85 ± 4.03 vs 74.66 ± 23.15 pg/mL; $p < 0.001$), confirming clear group differentiation and consistency with hypogonadism.

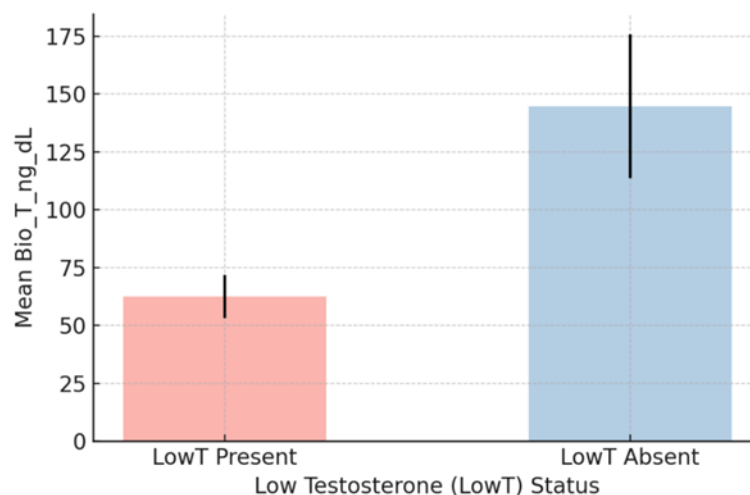


Figure 26: Comparison of Mean Bio_T_ng_dL Value Between LowT and Non-LowT Groups

Figure 26 shows a highly significant difference in bioavailable testosterone between groups, with markedly lower levels in LowT participants (62.56 ± 9.33 vs 144.68 ± 31.13 ng/dL; $p < 0.001$), confirming the validity of LowT classification and pronounced hormonal deficiency.

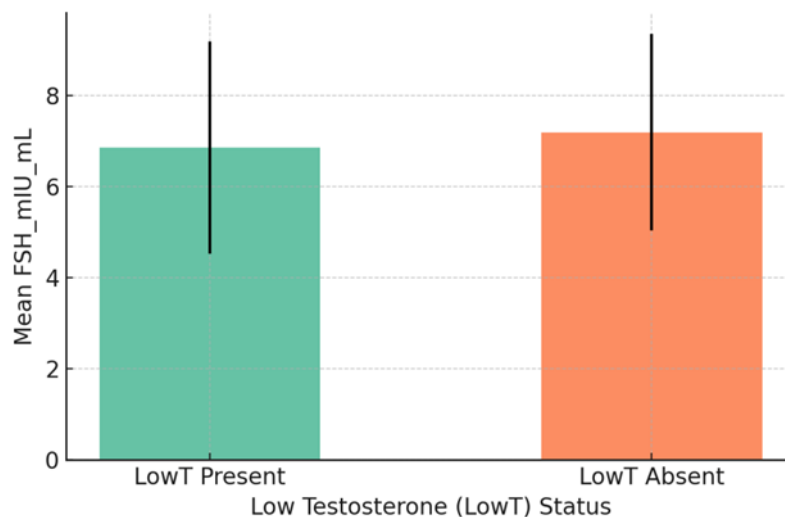


Figure 27: Comparison of Mean FSH_mIU_mL Between LowT and Non LowT Groups

Figure 27 shows no significant difference in mean FSH levels between LowT and normal groups (6.85 ± 2.33 vs 7.19 ± 2.16 mIU/mL; $p = 0.231$). This indicates comparable FSH levels, suggesting low testosterone is likely of secondary (hypogonadotropic) rather than primary origin.

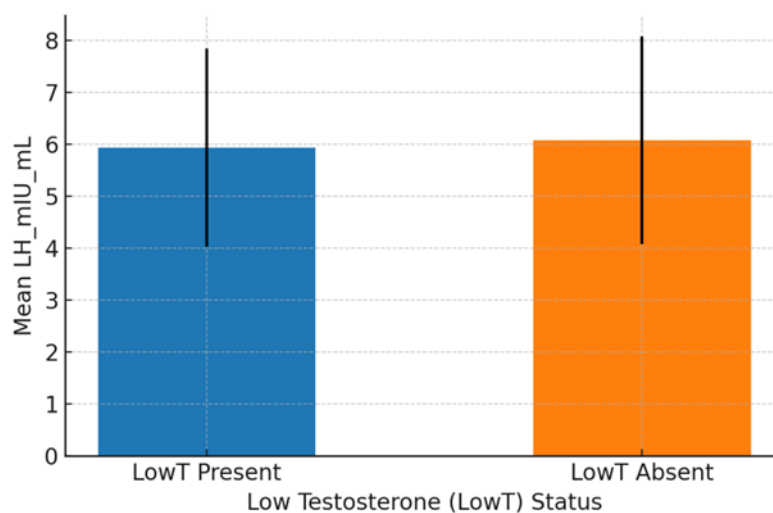


Figure 28: Comparison of Mean LH_mIU_mL Between LowT and Non-LowT Groups

Figure 28 shows no significant difference in mean LH levels between LowT and normal groups (5.94 ± 1.91 vs 6.08 ± 2.00 mIU/mL; $p = 0.578$). This suggests similar LH secretion in both groups, indicating absence of a compensatory LH rise and pointing toward a central (hypogonadotropic) mechanism of testosterone deficiency.

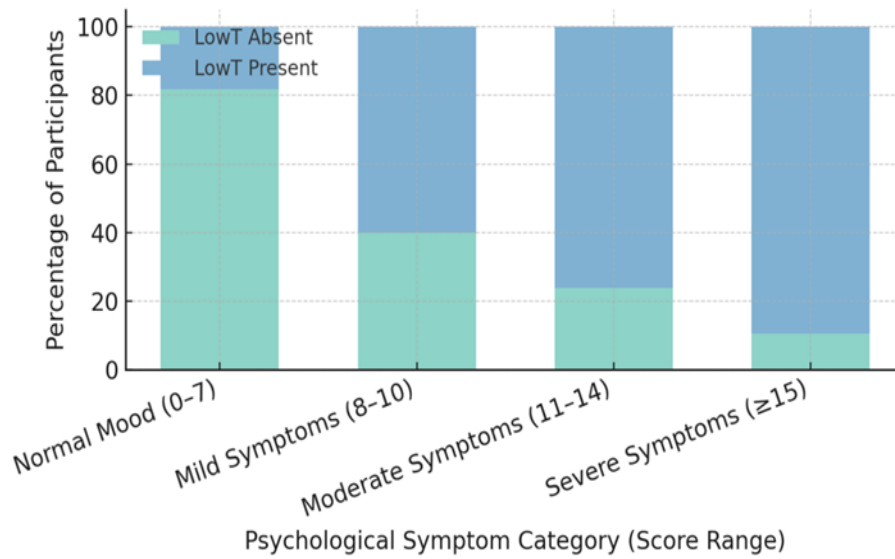


Figure 29: Distribution of Low Testosterone (LowT) Across Psychological Symptoms category

Figure 29 shows a highly significant association between psychological symptom severity (SHIM score) and testosterone status. LowT prevalence increased with worsening mood from 18.1% in

normal to 89.5% in severe cases. Overall, 61.7% had LowT, with a significant chi-square association ($p < 0.001$).

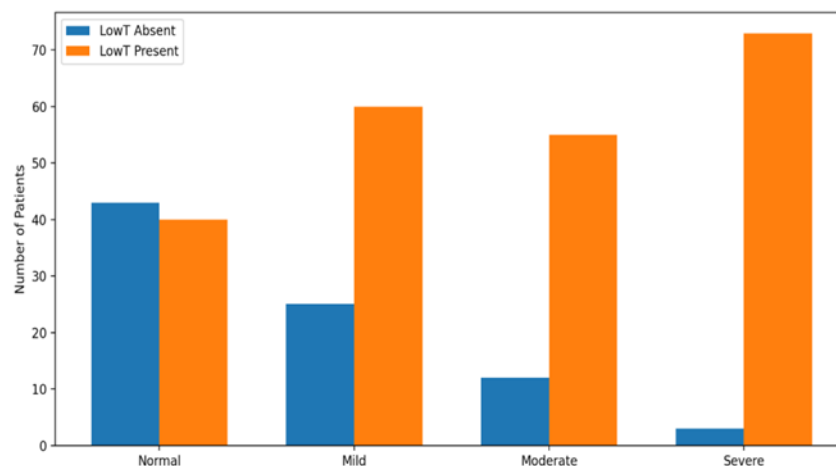


Figure 30: Distribution of Low Testosterone (LowT) Across Somatic Symptom Severity Categories

Figure 30 shows a significant association between somatic symptom severity and LowT status. LowT prevalence increased from 48.2% in the normal

group to 96.1% in the severe group, indicating a strong link between higher somatic symptom burden and testosterone deficiency.

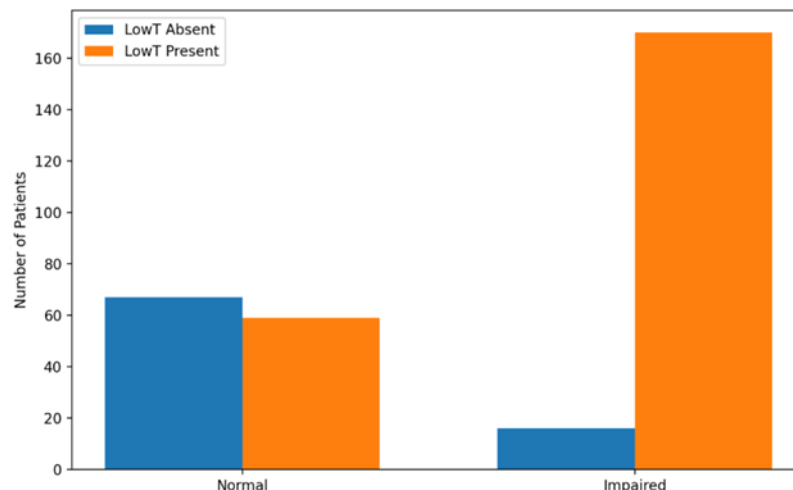


Figure 31: Distribution of Low Testosterone (LowT) Across Sexual Symptom Categories

Figure 31 demonstrates a strong association between sexual symptom severity and LowT status. LowT was present in 91.4% of patients with impaired symptoms versus 46.8% with normal scores, indicating a significant link between sexual dysfunction severity and testosterone deficiency.

4. DISCUSSION

Age is an important determinant in the clinical course of Type 2 Diabetes Mellitus (T2DM). Sattar N et al. (2019) demonstrated that earlier onset of diabetes, particularly before 40 years of age, is associated with significantly higher risks of cardiovascular events and mortality, with risks diminishing at older ages [19]. In contrast, the present study population had a mean age of 51.1 ± 11.59 years, representing a predominantly middle-aged cohort. This may explain the absence of extreme age-related risk gradients observed in large registry-based studies. Nevertheless, both studies reinforce the clinical relevance of age in T2DM progression and outcomes.

A high prevalence of hypogonadism was observed in the present study (74.8%), consistent with findings by Serwaa D et al. (2021), who reported similarly elevated rates of low testosterone among men with T2DM [20]. However, unlike their findings, which identified smoking, sedentary behavior, and BMI as significant predictors, the current study did not demonstrate significant associations between low testosterone and lifestyle factors such as smoking, alcohol intake, or physical

activity. This discrepancy may be attributed to differences in population characteristics, as the present cohort had a predominance of non-smokers and low alcohol consumption. These findings suggest that hypogonadism in T2DM may be driven more by intrinsic metabolic and endocrine mechanisms rather than lifestyle factors alone in certain populations.

Similarly, although BMI has been identified as a predictor of testosterone deficiency in several studies, including Serwaa et al. [20], the present study did not find a significant association between BMI and low testosterone. This indicates that BMI alone may not be a consistent determinant across populations, and factors such as visceral adiposity, insulin resistance, and hormonal dysregulation may play a more critical role. Supporting this, Hu G et al. (2007) emphasized the role of central adiposity, particularly waist circumference, in cardiometabolic risk [21]. In the current study, waist circumference showed a trend toward association with low testosterone, suggesting that central obesity may be more relevant than overall body mass.

The duration of diabetes is another key factor influencing outcomes. Low Wang CC et al. (2016) highlighted that cardiovascular risk increases with longer disease duration [22]. In the present study, the mean duration of diabetes was relatively short (5.66 years), which may explain the lack of marked cardiovascular differentiation within the cohort. However, a subset of patients with longer disease

duration remains at increased risk, underscoring the importance of early intervention and risk factor management.

Treatment patterns also influence glycemic control and long-term outcomes. Nichols GA et al. (2007) reported delayed insulin initiation in many patients, leading to prolonged hyperglycemia [23]. In contrast, the current study demonstrated a more heterogeneous treatment approach, with a significant proportion of patients receiving insulin therapy, suggesting improved recognition of disease progression. However, the continued reliance on oral therapy in many patients highlights ongoing challenges in timely treatment escalation.

Hypertension is a common comorbidity in T2DM. Studies such as Basil B et al. (2025) have demonstrated a high prevalence of hypertension and its role in cardiovascular risk [24]. The present study similarly showed elevated systolic blood pressure levels, reinforcing the need for strict blood pressure control. Evidence also suggests a U-shaped relationship between blood pressure and cardiovascular outcomes, with optimal diastolic levels between 80–90 mmHg [25]. The current findings align with this, as most participants had diastolic values within this range.

Despite advances in management, glycemic control remains suboptimal in many patients. Rosenfeld RM et al. (2025) emphasized the importance of lifestyle interventions in improving glycemic outcomes [26]. However, the present study demonstrated elevated fasting and postprandial glucose levels, indicating inadequate control. Hiyoshi T et al. (2017) highlighted the role of postprandial hyperglycemia in cardiovascular risk [27], and the high postprandial glucose levels observed in this study further support this association. Similarly, Whyte MB et al. (2019) reported persistent poor glycemic control influenced by systemic factors [28], consistent with the variability observed in HbA1c levels in the present cohort.

Testosterone deficiency is well recognized in men with T2DM. Cheung KK et al. (2015) reported a high prevalence of low testosterone associated with metabolic dysfunction [29], which aligns with the

findings of this study. Interventional studies have suggested potential metabolic benefits of testosterone therapy, including improved glycemic control and lipid profiles [30,31]. However, the present study did not assess therapeutic outcomes, emphasizing the need for further research in this area.

The relationship between obesity, insulin resistance, and hypogonadism has been described as a bidirectional cycle. Saboor Aftab SA et al. (2013) proposed male obesity-associated secondary hypogonadism as a distinct entity [32]. The high prevalence of low testosterone in the current study supports this concept. Additionally, hormonal evaluation revealed that gonadotropin levels (LH and FSH) remained largely within normal ranges, suggesting that hypogonadism in this population is predominantly functional or secondary rather than due to primary testicular failure [33,34].

Sexual dysfunction was highly prevalent in the present study, with over 94% of participants exhibiting erectile dysfunction. This is consistent with findings by Bjerggaard M et al. (2015) and Gupta M et al. (2022), who reported a high burden of sexual dysfunction in men with T2DM [35,36]. Furthermore, Nguyen Ngoc Dang H et al. (2024) demonstrated that erectile dysfunction is closely associated with increased cardiovascular risk [37]. These findings highlight the importance of routine sexual health assessment as part of diabetes management.

The present study also found no significant association between testosterone levels and variables such as occupation, smoking, alcohol consumption, and physical activity, consistent with findings by Vinnikov D et al. (2021) and Pan W et al. (2025) [38,39]. This suggests that lifestyle factors may have a limited independent effect on testosterone levels in the presence of established metabolic disease.

Interestingly, no significant associations were observed between testosterone levels and glycemic parameters such as fasting glucose, postprandial glucose, or HbA1c, contrasting with studies that report inverse relationships between these variables [40–42]. This may indicate that testosterone

deficiency reflects broader metabolic dysregulation rather than direct glycaemic control alone.

Biochemical assessment confirmed significantly lower total, free, and bioavailable testosterone levels in the hypogonadal group, consistent with established diagnostic criteria [43–45]. The importance of free testosterone as a more accurate marker of androgen status has been emphasized in previous studies [46]. Additionally, normal LH and FSH levels in most participants suggest a central or functional origin of hypogonadism [47,48].

Psychological, somatic, and sexual symptoms were strongly associated with low testosterone levels in the present study. Similar associations have been reported in previous studies linking testosterone deficiency with mood disturbances and reduced quality of life [49,50]. Sexual dysfunction, in particular, showed a strong correlation with low testosterone, supporting findings by Corona G et al. (2017), who demonstrated significant improvement in sexual function with testosterone therapy [50].

Overall, the findings of this study highlight the high prevalence of hypogonadism and erectile dysfunction among men with T2DM. While lifestyle and anthropometric factors showed limited association, hormonal deficiency was strongly linked to symptom severity, emphasizing the multifactorial nature of hypogonadism in diabetes.

These results underscore the importance of routine screening for testosterone deficiency and comprehensive management strategies that address both metabolic and hormonal aspects of the disease.

CONCLUSION

This study demonstrates that hypogonadism is a highly prevalent comorbidity among Indian men with Type 2 Diabetes Mellitus, affecting nearly three-fourths of patients, and is strongly associated with significant clinical manifestations, including erectile dysfunction, fatigue, and psychological distress. The pattern of normal or low gonadotropin levels indicates that hypogonadism in this population is predominantly secondary, likely resulting from metabolic disturbances, such as visceral adiposity and insulin resistance, that affect the hypothalamic–pituitary–gonadal axis. The absence of association with glycaemic parameters or treatment modalities suggests that testosterone deficiency is intrinsically linked to the underlying metabolic dysfunction of T2DM rather than hyperglycemia alone. These findings highlight the need to consider all men with T2DM, particularly those with central obesity or suggestive symptoms, as high-risk for hypogonadism, and support the integration of routine testosterone screening into diabetes management to improve quality of life and potentially influence metabolic outcomes.

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