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Original Research Article

Sleep quality as a predictor of sexual dysfunction in men with type 2 diabetes

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ABSTRACT

Background: Sexual dysfunction and hypogonadism are common yet under-recognized complications of type 2 diabetes mellitus (T2DM). Sleep disturbances—particularly clinical insomnia—may exacerbate neuroendocrine and metabolic dysregulation, but their role as predictors of sexual dysfunction and testosterone deficiency in diabetic men remains unclear.
Methods: In this cross-sectional study, 54 men with T2DM (age 35–60 years) attending our outpatient clinic were evaluated for insomnia (insomnia severity index, ISI), hypogonadal symptoms (androgen deficiency in ageing male, ADAM questionnaire), morning serum total testosterone (TT), and sexual dysfunction (self-reported clinical interview). Clinical insomnia was defined as ISI >15; biochemical hypogonadism as TT <3 ng/ml; confirmed hypogonadism as ADAM-positive plus TT <3 ng/ml. Participants were stratified by insomnia status (n=18 versus 36) and compared using t–tests and χ^2 tests. Logistic regression adjusted for age, body mass index (BMI), glycated haemoglobin (HbA1c), and diabetes duration identified independent predictors of confirmed hypogonadism.
Results: Clinical insomnia was present in 33.3% of the cohort. Overall, 37.0% of men reported sexual dysfunction. Insomniac participants had higher rates of sexual dysfunction (66.7% versus 22.2%; p<0.001), ADAM-positivity (83.3% versus 36.1%; p=0.001), biochemical hypogonadism (44.4% versus 11.1%; p=0.006), and confirmed hypogonadism (38.9% versus 8.3%; p=0.014) compared to non-insomniacs. In multivariable analysis, clinical insomnia remained the only independent predictor of confirmed hypogonadism (OR 12.14; 95% CI 1.16–126.60; p=0.037).
Conclusions: Clinical insomnia in men with T2DM is strongly associated with both sexual dysfunction and testosterone deficiency, and independently predicts confirmed hypogonadism. These findings support integrating sleep-quality assessment into diabetic care pathways and targeting insomnia as a modifiable factor to improve sexual and endocrine health.

Keywords: Type 2 diabetes mellitus, Clinical insomnia, Hypogonadism, Sexual dysfunction, Testosterone, Insomnia severity index, ADAM

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a multifaceted metabolic disorder that exerts profound effects on numerous organ systems, including the endocrine and reproductive axes. Among its myriad complications, sexual dysfunction and hypogonadism are increasingly recognized in men with T2DM, yet they often remain underdiagnosed and undertreated despite their substantial impact on quality of life.[1] Epidemiological studies report that hypogonadism affects a substantial portion of this patient population, often exceeding 30%.[2] In this population, hypogonadism is typically functional rather

than primary, arising from a complex interplay of disrupted hypothalamic–pituitary–gonadal (HPG) axis regulation, insulin resistance, and chronic inflammation.[3]

Concurrently, sleep disturbances are highly prevalent in individuals with T2DM. Poor sleep quality has been linked to impaired glucose tolerance, elevated evening cortisol, and systemic inflammation—factors that may further perturb testosterone secretion and sexual function. This pro-inflammatory milieu, characterized by elevated cytokines like IL-6 and TNF-α, is another factor implicated in HPG axis suppression.[4] This is particularly evident in conditions like obstructive sleep apnea (OSA), which is highly prevalent in T2DM and is an independent risk factor for both metabolic and gonadal dysfunction.[5] The bidirectional relationship between sleep disruption and metabolic health suggests that sleep quality might be a modifiable determinant in the development or progression of hypogonadal symptoms and sexual dysfunction among diabetic men.

Despite mounting evidence that general sleep disorders correlate with lower testosterone levels and poorer sexual function, a link substantiated by studies showing direct suppression of luteinizing hormone pulsatility after sleep restriction—there remains a paucity of data specifically examining clinical insomnia.[6] The impact of insomnia, as quantified by validated scales such as the insomnia severity index (ISI), on both symptomatic hypogonadism (evaluated with the androgen deficiency in ageing male, or ADAM, questionnaire) and biochemical testosterone deficiency in T2DM is not fully elucidated.[7,8]

Elucidating this relationship could enable clinicians to identify high-risk patients early, integrate sleep assessment into routine diabetic care, and implement targeted interventions aimed at improving both metabolic control and reproductive health. This study therefore evaluates this association and assesses whether insomnia can serve as an independent predictor for confirmed hypogonadism in adult men with T2DM.

Aim

Aim of the study was to evaluate whether clinical insomnia (ISI score >15) is associated with hypogonadism and sexual dysfunction in adult males with T2DM.

Objectives

Primary objective of the study was to investigate whether the presence of clinical insomnia (ISI >15) is associated with hypogonadal symptoms and sexual dysfunction among adult males with T2DM.

Secondary objectives of the study were: to assess the association between clinical insomnia and metabolic parameters such as body mass index (BMI), duration of diabetes, and glycated haemoglobin (HbA1c) levels, and to estimate the prevalence of clinical insomnia and hypogonadism in the study population.

METHODS

Study design and participants

This cross-sectional study was conducted at the outpatient department of the Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar, India. Participant enrollment occurred between 05 March 2025 and 05 May 2025.

Participant enrollment was guided by specific inclusion criteria and exclusion criteria. The inclusion criteria required participants to be men aged 35–60 years with a confirmed diagnosis of T2DM.

The exclusion criteria, designed to minimize confounding factors, comprised of prior diagnosis of hypogonadism or current testosterone replacement therapy; use of phosphodiesterase-5 inhibitors, antidepressants, or herbal medications; significant systemic conditions such as decompensated liver disease, malignancy, tuberculosis, AIDS, or major psychiatric illness; chronic kidney disease stage 3 or higher; congenital or acquired testicular disorders; an abnormal karyotype; or any acute illness known to affect testosterone levels.

Sample size

The required sample size was calculated based on prior research reporting a mean difference of 1.0 ng/ml (SD≈0.9 ng/ml) in testosterone between men with poor and good sleep quality (d≈1.1).[9,10] A minimum of 42 participants was needed to provide 80% power at α=0.05 using a two-sample t-test. Target enrollment was set at 54 to allow for a 20% dropout rate.

The final sample size comprised of 54 participants (Figure 1). This group was established from an initial pool of 64 men attending the T2DM outpatient clinic who were assessed for eligibility. Ten individuals were excluded for the following reasons: previously diagnosed hypogonadism (n=4), use of prohibited medications (PDE-5 inhibitors, n=2; antidepressants, n=1), significant comorbidity (chronic kidney disease stage ≥3, n=1), or declining to participate (n=2).

Study procedure

Upon enrollment, demographic data, clinical history (age, duration of diabetes), and anthropometric measurements (height, weight and BMI) were collected. BMI was categorized using Asia-Pacific guidelines (<18.5 kg/m² for underweight, 18.5–22.9 kg/m² for normal, 23.0–24.9 kg/m² for overweight, and >25.0 kg/m² for obese). Glycemic control was assessed by HbA1c, classified per American Diabetes Association criteria (<5.7% for non-diabetic, 5.7–6.4% for prediabetic, and ≥6.5% for diabetic).

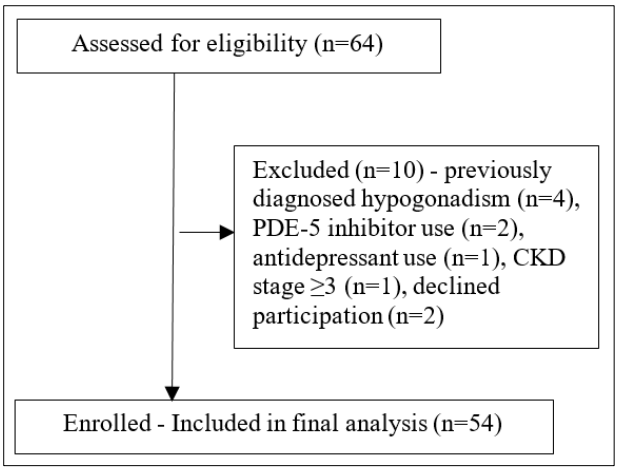

Figure 1: Patient selection.

Sleep quality was evaluated with the insomnia severity index (ISI), with clinical insomnia defined as a total score >15. Symptoms of hypogonadism were assessed using the androgen deficiency in the aging male (ADAM) questionnaire. ADAM-positivity was defined as a "yes" response to question 1 (decreased libido) or 7 (erectile dysfunction), or to any three other items. A "yes" response to question 7 served as the study's proxy for erectile dysfunction.

Fasting venous blood samples were collected between 08:00 and 10:00 hours. Serum was separated by centrifugation at 3,000 rpm for 10 minutes (Eppendorf 5702, Germany) after a 30-minute clotting period and was stored at –20 °C until analysis. Total testosterone (TT) was measured via immunoassay (Beckman Coulter DxI 800) with a measurement range of 0.1–15 ng/ml, an intra-assay CV of <8%, and an inter-assay CV of <10%. Biochemical hypogonadism was defined as TT <3 ng/ml. The primary outcome, confirmed hypogonadism, was defined as the presence of both biochemical hypogonadism and ADAM-positivity.

Ethical approval

The study protocol was approved by the Institutional Ethics Committee of Kalinga Institute of Medical Sciences (Ref. no. KIIT/KIMS/IEC/1979/2025). All participants provided written informed consent before any study-related procedures were performed. Data and biological samples were managed under IEC guidelines to ensure confidentiality.

Statistical analysis

Analyses were conducted using IBM statistical package for the social sciences (SPSS) statistics v20.0. Continuous variables were tested for normality with the Shapiro–Wilk test and are reported as mean±SD, while categorical variables are reported as count (percentage). Group differences based on insomnia status (ISI >15 versus ≤15) were compared using independent-samples t-tests or Chi-square tests, as appropriate. A multivariable logistic regression model was used to assess predictors (age, BMI, HbA1c, diabetes duration, and insomnia status) of confirmed hypogonadism. Participants with missing data for BMI (n=1) or diabetes duration (n=2) were excluded from analyses involving those variables (complete-case analysis). Statistical significance was set at a two-sided $p<0.05$.

RESULTS

Participant characteristics

Fifty-four adult men with T2DM were included. The mean age was 49.5±6.2 years (range 38–60), mean BMI 25.8±2.6 kg/m² (range 21.9–32.5), mean duration of T2DM 8.5±3.3 years (range 3–15), mean HbA1c 8.12±0.71% (range 6.9–9.5), and mean morning serum TT 3.85±0.90 ng/ml (range 1.7–5.3). Clinical insomnia (ISI >15) was present in 18 participants (33.3%).

Baseline characteristics by insomnia status

Participants were stratified into those with (n=18) and without (n=36) clinical insomnia (Table 1 and Figure 2).

Age

Insomniacs were 50.1±7.0 years versus 48.9±6.7 years in non-insomniacs (p=0.60).

BMI

Participants with insomnia had higher BMI (27.4±3.8 kg/m²) than those without (25.6±2.9 kg/m²; p=0.046).

Duration of T2DM

A non-significant trend toward longer diabetes duration was observed in the insomnia group (9.6±3.8 versus 7.7±4.0 years; p=0.14).

HbA1c

Glycemic control was worse among insomniacs (8.77±1.11% versus 7.89±1.10%; p=0.005).

Morning TT

Insomniacs had significantly lower testosterone levels (3.10±0.82 ng/ml) compared to non-insomniacs (4.18±0.86 ng/ml; p<0.001).

Hypogonadism-related outcomes by insomnia status

Rates of hypogonadal symptoms and biochemical/ confirmed hypogonadism were substantially higher in the insomnia group (Table 2 and Figure 3).

Table 1: Baseline characteristics by insomnia status.

Variables	Insomnia mean±SD	No insomnia mean±SD	P value
Age, years	50.06±6.98	48.93±6.73	0.6
BMI, kg/m²	27.40±3.83	25.56±2.93	0.046
Duration of T2DM, years	9.56±3.81	7.69±3.96	0.14
HbA1c, %	8.77±1.11	7.89±1.10	0.005
Morning TT, ng/ml	3.10±0.82	4.18±0.86	<0.001

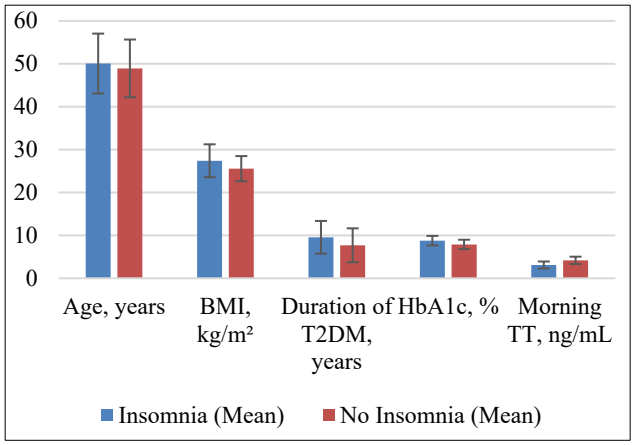

Figure 2: Continuous variables by insomnia status.

ADAM-positivity

83.3% of insomniacs screened positive on the ADAM questionnaire versus 36.1% of non-insomniacs (p=0.001).

Biochemical hypogonadism

Defined as TT <3 ng/ml, this was observed in 44.4% of insomniacs versus 11.1% of the comparison group (p=0.006).

Confirmed hypogonadism

Both symptomatic (ADAM+) and biochemical criteria were met in 38.9% of insomniacs compared to 8.3% of non-insomniacs (p=0.014).

Table 2: Hypogonadism-related outcomes by insomnia status.

Outcome	Insomnia N (%)	No insomnia N (%)	P value
ADAM-positive	15 (83.3)	13 (36.1)	0.001
Biochemical hypogonadism	8 (44.4)	4 (11.1)	0.006
Confirmed hypogonadism	7 (38.9)	3 (8.3)	0.014

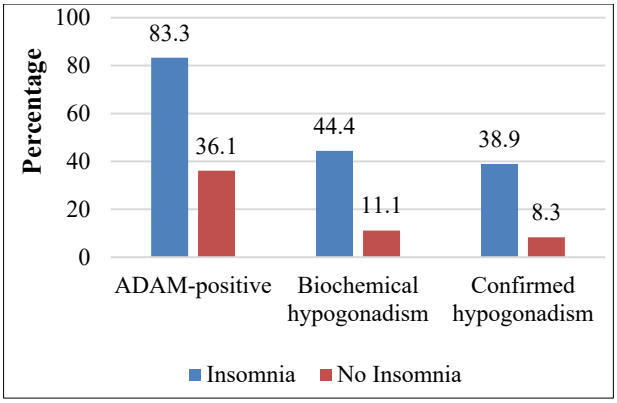

Figure 3: Hypogonadism-related outcomes by insomnia status.

Multivariable analysis

We fitted a logistic regression for confirmed hypogonadism, adjusting for age, BMI, HbA1c, and diabetes duration (Table 3).

Clinical insomnia emerged as a powerful independent predictor (OR 12.14; 95% CI 1.16–126.60; p=0.037).

Age, BMI, HbA1c, and duration of diabetes did not retain statistical significance in the adjusted model (all p>0.28).

Table 3: Multivariable predictors of confirmed hypogonadism.

Predictor	OR	95 % CI	P value
Clinical insomnia	12.14	1.16–126.60	0.037
Age (per year)	1	0.86–1.16	0.998
BMI (per kg/m²)	0.97	0.68–1.38	0.861
HbA1c (per %)	0.62	0.11–3.53	0.588
Duration (years)	1.1	0.92–1.32	0.284

DISCUSSION

In this cohort of 54 men with T2DM, we observed that clinical insomnia was not only associated with higher body mass index (BMI) and poorer glycemic control but also with markedly lower TT levels. Insomniac participants exhibited three- to four-fold higher rates of symptomatic (ADAM-positive) and biochemical hypogonadism compared to their well-rested counterparts. Crucially, clinical insomnia emerged as the only independent predictor of confirmed hypogonadism in multivariable analysis, highlighting the intimate link between sleep disturbances and the integrity of the HPG axis in men with metabolic disease.

Our results align with and extend prior research. For instance, Smith et al reported a significant inverse relationship between sleep fragmentation and TT in middle-aged men, while Rodriguez et al found that acute

sleep deprivation suppresses morning testosterone secretion.[9,10] Our data also resonate with broader findings in metabolic syndrome, where central obesity and insulin resistance are established drivers of reduced testosterone, independent of sleep.[11] Unlike these earlier studies—which primarily focused on sleep duration or fragmentation—our work specifically identifies insomnia severity (ISI >15) as a predictor of both symptomatic and biochemical hypogonadism in a T2DM population.

This suggests that subjective sleep quality warrants attention alongside standard metabolic parameters. Our findings are consistent with the European Male Ageing Study by Camacho et al, which demonstrated that reduced sleep duration is independently associated with lower TT.[12] In men with T2DM specifically, Corona et al reported a biochemical hypogonadism prevalence of over 30% and a strong link to erectile dysfunction, a finding supported by other large-scale studies that detail the complex pathophysiology, including endothelial dysfunction and neuropathy, underlying this connection.[13,14] Our observed rates of hypogonadism among insomniac diabetics mirror these reports.

Insomnia's effect on elevating evening cortisol is well-documented and represents a classic stress response that can directly inhibit the HPG axis at the level of the hypothalamus.[15] At the same time, fragmented or insufficient sleep exacerbates insulin resistance—a hallmark of T2DM—which may directly interfere with testicular steroidogenesis by impairing Leydig cell function.[16] These converging influences may create a vicious cycle whereby hyperglycemia and hyperinsulinemia further disrupt sleep architecture, perpetuating hypogonadal states.

Clinically, our findings suggest that routine assessment of insomnia symptoms should be integrated into the hormonal evaluation of men with T2DM. Given the high prevalence of both sleep disturbances and hypogonadism in this population, targeted interventions such as cognitive behavioral therapy for insomnia (CBT-I), the gold-standard non-pharmacologic treatment, are highly recommended. Future randomized trials are needed to determine whether treating insomnia can reverse hypogonadal features or enhance response to testosterone replacement therapy in men with diabetes.

Strengths

The primary strength of this study lies in its comprehensive approach to defining hypogonadism, which integrated both validated subjective symptom assessment (ADAM questionnaire) and objective biochemical evidence (serum total testosterone). Furthermore, the use of the Insomnia Severity Index provided a standardized, validated measure of clinical insomnia. The application of multivariable logistic regression allowed for the statistical adjustment of key metabolic and demographic confounders, thereby strengthening the evidence for insomnia as an independent predictor.

Limitations

This investigation possesses several limitations. The cross-sectional nature of the study design establishes a significant association but precludes the establishment of causality between clinical insomnia and hypogonadism. Recruitment from a single tertiary care center may limit the generalizability of our findings to other populations or healthcare settings. Additionally, the use of a single morning total testosterone measurement, while standard practice, does not account for the hormone's diurnal rhythm or potential intra-individual variability.

CONCLUSION

In men with T2DM, clinical insomnia is strongly and independently associated with confirmed hypogonadism. This study advances current knowledge by identifying a specific, modifiable sleep disorder, as defined by a validated instrument, as a significant predictor of both symptomatic and biochemical testosterone deficiency, independent of established metabolic risk factors. These findings suggest that the clinical assessment of sleep quality should be considered an integral component of the diagnostic workup for hypogonadism in this patient population and that insomnia may represent a novel therapeutic target for improving endocrine and sexual health in men with T2DM.

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