

Hormonal Profile and Sexual Health in Adult Males

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Abstract

Original Research Article

Background: Male sexual health is closely linked to hormonal balance and cardiometabolic status. Testosterone and other endocrine factors play a critical role in libido, erectile function, and overall sexual performance. **Objective:** To evaluate the hormonal profile and its association with sexual health status in adult males. **Methods:** This cross-sectional analytical study was conducted among 200 adult males aged 18–60 years at Satkhira Medical College Hospital, Satkhira, Bangladesh. Demographic and clinical data were recorded, and sexual health was assessed using the IIEF-5 along with evaluation of libido and orgasmic function. Morning fasting blood samples were analyzed for total and free testosterone, LH, FSH, prolactin, TSH, and SHBG. Data were analyzed using SPSS version 22, and $p < 0.05$ was considered statistically significant. **Results:** The mean age of participants was 38.6 ± 9.4 years. Erectile dysfunction was present in 59% of subjects, with 32% having moderate to severe ED. Reduced libido was reported in 40% and impaired orgasmic function in 27%. Low total testosterone (< 300 ng/dL) was significantly associated with moderate to severe ED ($p < 0.001$). A moderate positive correlation was observed between total testosterone levels and IIEF-5 scores ($r = 0.46$, $p < 0.001$). **Conclusion:** Sexual dysfunction is common among adult males and is significantly associated with hormonal status, particularly serum testosterone. Comprehensive hormonal and metabolic evaluation is essential in men presenting with sexual health complaints.

Keywords: Hormonal profile; Erectile dysfunction; Testosterone; Male sexual health; Cardiometabolic risk.

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INTRODUCTION

Male sexual health covering libido, erectile function, orgasmic function, mood/energy, and fertility is tightly linked to endocrine balance, vascular integrity, neurologic pathways, and psychosocial factors. Contemporary guidelines therefore treat sexual symptoms as an important “window” into broader cardiometabolic and endocrine health, not only a quality-of-life issue [1].

At the center of the hormonal contribution is the hypothalamic–pituitary–gonadal (HPG) axis. Gonadotropin-releasing hormone drives pituitary luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which regulate testicular testosterone production and spermatogenesis. Testosterone supports sexual desire and contributes to erectile physiology through central (brain) and peripheral (penile tissue) mechanisms, while also influencing body composition, bone health, and metabolic regulation. Because

symptoms such as low libido, reduced morning erections, fatigue, and depressed mood can overlap with many conditions, expert guidance emphasizes that testosterone deficiency should be diagnosed only when consistent symptoms accompany unequivocally low serum testosterone, confirmed on repeat morning testing [2-3].

A practical “hormonal profile” for adult men presenting with sexual complaints typically begins with total testosterone (morning, fasting when feasible), and when results are borderline or conditions affecting binding proteins exist, includes free testosterone or an estimate derived from sex hormone-binding globulin (SHBG). This is especially relevant because obesity and insulin resistance can lower SHBG, reducing measured total testosterone without necessarily indicating primary testicular failure sometimes described as a “pseudo-hypogonadism of obesity [4].” Measuring LH/FSH helps distinguish primary (testicular) from secondary

(pituitary/hypothalamic) hypogonadism and guides further evaluation [5].

Beyond testosterone, several hormones commonly intersect with male sexual function. Prolactin excess can reduce libido and impair erectile function directly and indirectly via suppression of the HPG axis; thus, prolactin testing is often recommended when hypogonadotropic patterns or suggestive symptoms are present [6]. Thyroid dysfunction (both hypo- and hyperthyroidism) is also linked with higher rates of male sexual dysfunction, and growing synthesis evidence suggests a substantial global burden of sexual symptoms among men with thyroid disorders supporting thyroid screening in selected patients [7]. Depending on clinical context, clinicians may also assess estradiol (particularly in obesity or gynecomastia), morning cortisol (if systemic endocrine disease is suspected), and metabolic markers that strongly modulate sexual health.

Importantly, sexual symptoms in adult men frequently reflect cardiometabolic comorbidity. Obesity is consistently associated with higher erectile dysfunction (ED) risk, and population analyses show increased odds of moderate/severe ED among men with obesity [8]. Metabolic syndrome is likewise associated with higher ED risk in large datasets [9]. Diabetes markedly amplifies ED prevalence and severity through vascular, neurologic, and endothelial mechanisms, making glycemic status and cardiovascular risk assessment integral to the evaluation [10]. Sleep disorders add another layer: obstructive sleep apnea has been associated with lower testosterone and worse sexual outcomes, supporting screening where symptoms or risk factors exist [11].

Because male sexual health is multifactorial, modern best practice integrates (1) symptom-anchored hormonal testing, (2) identification of reversible contributors (weight, sleep, medications, mental health), and (3) evidence-based sexual-medicine management pathways for ED and related conditions [12]. Framing sexual symptoms alongside a structured hormonal and metabolic work-up can improve diagnostic precision, guide targeted therapy, and uncover broader health risks at a stage when intervention may yield substantial benefits. Therefore, this study aims to assess the hormonal profile of adult males and its association with sexual health outcomes.

Objectives

The main objective was to evaluate the hormonal profile and its association with sexual health outcome in adult males.

METHODOLOGY & MATERIALS

The cross-sectional analytical study was conducted in the Department of Urology, Satkhira Medical College Hospital, Satkhira, Bangladesh. The

study was conducted over a 2 years period from 1 January 2024 to 31 December 2025.

Inclusion Criteria:

- Adult males aged 18–60 years
- Married and sexually active individuals
- Patients willing to participate and provide informed consent

Exclusion Criteria

- Known primary testicular failure or pituitary disorders
- History of androgen replacement therapy within the last 6 months
- Chronic liver or renal failure
- Severe psychiatric illness
- Acute systemic illness at the time of evaluation

A total of 200 patients were included in the study. Data were collected using a structured questionnaire and clinical data collection form. Demographic variables such as age, body mass index (BMI), residence, occupation, and lifestyle factors including smoking status, alcohol intake, and physical activity were recorded. Information regarding comorbidities such as diabetes mellitus, hypertension, obesity, and dyslipidemia was also documented. Sexual health status was assessed using the International Index of Erectile Function (IIEF-5) questionnaire along with additional questions evaluating libido, orgasmic function, and overall sexual satisfaction.

For hormonal evaluation, venous blood samples were collected between 8:00 AM and 10:00 AM after overnight fasting to minimize diurnal variation. Serum levels of total testosterone, free testosterone (calculated or measured), luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, thyroid-stimulating hormone (TSH), and sex hormone-binding globulin (SHBG) were measured using standardized chemiluminescent immunoassay methods in the hospital laboratory. Ethical approval was obtained from the Institutional Review Board, and confidentiality of all participants was strictly maintained throughout the study.

Statistical Analysis:

Data were entered and analyzed using SPSS version 22. Quantitative variables such as hormonal levels were expressed as mean \pm standard deviation, while qualitative variables including sexual health categories were presented as frequency and percentage. Associations between hormonal profile and sexual health outcomes were assessed using appropriate statistical tests (t-test, ANOVA, and chi-square test). A p-value <0.05 was considered statistically significant. Confidentiality was strictly maintained.

RESULT

Table 1: Distribution of Patients by Demographic Characteristics (n = 200)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	18–30	52	26
	31–40	68	34
	41–50	50	25
	51–60	30	15
Mean ± SD	38.6 ± 9.4		
BMI (kg/m ²)	Normal	72	36
	Overweight	84	42
	Obese	44	22
Diabetes Mellitus	Yes	60	30
	No	140	70
Hypertension	Yes	54	27
	No	146	73

Table 1 shows that the mean age of the participants was 38.6 ± 9.4 years, with the majority (34%) belonging to the 31–40 years age group. Most

participants were overweight (42%) or obese (22%). Diabetes mellitus was present in 30% of patients, and 27% had hypertension.

Table 2: Distribution of Sexual Health Status Among Participants (n = 200)

Sexual Health Parameter	Category	Frequency (n)	Percentage (%)
Erectile Function (IIEF-5)	No ED	82	41
	Mild ED	54	27
	Moderate ED	40	20
	Severe ED	24	12
Libido	Normal	120	60
	Reduced	80	40
Orgasmic Function	Normal	146	73
	Impaired	54	27

Table 2 demonstrates that 59% of participants had some degree of erectile dysfunction, including 20% with moderate and 12% with severe ED. Reduced libido

was reported in 40% of participants, while impaired orgasmic function was observed in 27%.

Table 3: Hormonal Profile of the Study Participants (n = 200)

Hormonal Parameter	Mean ± SD	Reference Range
Total Testosterone (ng/dL)	412.5 ± 128.4	300–1000
Free Testosterone (pg/mL)	11.8 ± 3.9	9–30
LH (IU/L)	5.6 ± 2.1	1.5–9.3
FSH (IU/L)	6.3 ± 2.7	1.4–18.1
Prolactin (ng/mL)	14.5 ± 6.2	4–15
TSH (mIU/L)	2.7 ± 1.1	0.4–4.0
SHBG (nmol/L)	34.2 ± 12.5	10–57

Table 3 shows that the mean total testosterone level was 412.5 ± 128.4 ng/dL. The mean values of free

testosterone, LH, FSH, prolactin, TSH, and SHBG were within normal reference ranges.

Table 4: Association Between Total Testosterone Level and Erectile Dysfunction (n = 200)

Testosterone Level	No/Mild ED (n=136)	Moderate/Severe ED (n=64)	p-value
≥300 ng/dL	118 (86.8%)	32 (50.0%)	<0.001*
<300 ng/dL	18 (13.2%)	32 (50.0%)	

Chi-square test applied

Table 4 indicates that low testosterone levels (<300 ng/dL) were significantly more common among

patients with moderate to severe erectile dysfunction compared to those with no or mild ED ($p < 0.001$).

Table 5: Correlation Between Total Testosterone and IIEF-5 score

Variable	r-value	p-value
Total Testosterone vs IIEF-5 score	0.46	<0.001*

Pearson correlation test applied

Table 5 reveals a moderate positive correlation between total testosterone levels and IIEF-5 score ($r = 0.46$, $p < 0.001$), suggesting that lower testosterone levels are associated with poorer sexual function.

DISCUSSION

In this study of 200 adult males, 59% had some degree of erectile dysfunction (ED) and 32% had moderate–severe ED. This prevalence is comparable to several recent population/clinic-based studies reporting ED in the range of ~50–56%. For example, a 2025 population-based assessment using IIEF-5 methods reported substantial ED burden in men, highlighting how prevalence varies by definition and sampling frame [13]. A 2025 study also reported ED prevalence of 56.3% among participants assessed by IIEF-5 [14]. Differences across studies are expected because ED prevalence is strongly influenced by age distribution, comorbidities (diabetes, hypertension), cultural reporting, and whether the sample is community-based or clinic-attending [15].

We observed reduced libido in 40% and impaired orgasmic function in 27%. These findings align with evidence that male sexual health is multidimensional and that sexual symptoms frequently cluster, especially when endocrine or metabolic disturbances are present. Large syntheses in endocrine conditions (e.g., thyroid disorders) demonstrate high pooled prevalence of sexual dysfunction among affected men, supporting the concept that libido and orgasmic complaints often co-exist with erectile problems in hormonally vulnerable groups [16].

Regarding hormonal status, the mean total testosterone (412.5 ± 128.4 ng/dL) was within reference range overall; however, low testosterone (<300 ng/dL) was significantly more common in men with moderate–severe ED (50%) than in those with no/mild ED (13.2%), and testosterone correlated positively with IIEF-5 score ($r=0.46$). This pattern is consistent with clinical studies showing that lower testosterone is associated with worse erectile function scores. A 2025 tertiary-care study found a significant positive relationship between serum testosterone and IIEF-5, supporting the endocrine contribution to ED severity [17]. Similarly, a 2025 analysis examining hormones and ED severity emphasized the value of considering both total and free testosterone and binding proteins (e.g., SHBG) when interpreting sexual symptoms [18]. Not all research finds testosterone alone explains ED in men with “normal-range” testosterone, and some evidence suggests other hormones (e.g., DHEA-S, estradiol) may be independently associated with ED, indicating heterogeneous mechanisms across populations [19].

A key contextual finding in our cohort was the high burden of cardiometabolic risk (64% overweight/obese; diabetes 30%; hypertension 27%), which likely contributed to the observed ED prevalence and severity. Recent evidence strongly supports this link: metabolic syndrome is associated with higher ED risk in systematic review/meta-analysis and large datasets, reflecting shared pathways of endothelial dysfunction, inflammation, and insulin resistance [20]. Obesity-related studies also continue to report higher ED severity with increasing adiposity and related lifestyle factors such as smoking [21]. Diabetes is another major driver of ED, with contemporary studies in diabetic populations showing high ED prevalence and identifying factors such as age, glycemic control, and comorbidity burden [22]. These findings support an integrated interpretation of ED as both a sexual health condition and a potential marker of broader cardiometabolic disease.

Although our mean prolactin and thyroid parameters were largely within reference ranges, the literature supports targeted evaluation of prolactin and thyroid dysfunction in selected men with sexual complaints particularly when hypogonadotropic patterns, reduced libido, or refractory ED are present. Recent work has strengthened the link between hyperprolactinemia and male sexual dysfunction, and improvement in sexual function has been observed when prolactin normalizes with dopamine agonist therapy [23]. Mechanistic and clinical evidence also suggests prolactin can impair erectile function via pathways beyond testosterone alone [24]. For thyroid disease, systematic review/meta-analysis indicates a high prevalence of sexual dysfunction among men with thyroid disorders, and population-level research has reported increased ED risk in hyperthyroidism, supporting thyroid screening when clinically indicated [25].

Sleep-related factors may also influence hormonal balance and sexual function. Emerging evidence suggests that obstructive sleep apnea can contribute to reduced testosterone levels and erectile dysfunction through intermittent hypoxia and metabolic dysregulation. Although sleep parameters were not directly evaluated in the present study, the high prevalence of sexual dysfunction and cardiometabolic risk factors in our cohort highlights the importance of considering sleep disorders as a potential contributing factor [26]. Overall, our findings support the growing evidence that erectile dysfunction is multifactorial and closely linked to hormonal status and metabolic health, emphasizing the need for a comprehensive hormonal and

systemic evaluation in adult males presenting with sexual complaints.

Limitations of the study

It was conducted in a single center with a limited sample size, which may affect generalizability. Sexual health assessment was based on self-reported questionnaires, introducing potential reporting bias. Additionally, hormonal levels were measured from a single morning sample, and some influencing factors such as psychological and sleep-related variables were not fully evaluated.

CONCLUSION

The present study demonstrates a high prevalence of erectile dysfunction and other sexual health disturbances among adult males. Serum testosterone levels were significantly associated with erectile function, and lower testosterone was linked to greater severity of ED. Additionally, a substantial burden of cardiometabolic risk factors was observed, highlighting the multifactorial nature of male sexual dysfunction. These findings emphasize the importance of comprehensive hormonal and metabolic evaluation in adult males presenting with sexual health complaints to enable early identification and targeted management.

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Ethical approval: The study was approved by the Institutional Ethics Committee.

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