

Belly Fat, Low Testosterone and Male Sexual Health

Understanding Obesity-Related Secondary Hypogonadism, Hormonal Imbalance, Erectile Dysfunction, Low Libido and the Integrative Unani Approach

By Dr. Nizamuddin Qasmi

Founder & Chief Physician, Saira Health Care

Focused Practice in Sexual Disorders & Infertility

BUMS – Hamdard University, Delhi

MD, CGO

Certificate in Infertility – MGBIMS, Delhi

Certificate in Urology – London, UK

Masters in Male Infertility – MasterHealthPro (HealthPro)

Integrated Sexual and Reproductive Health – ISRH, UNFPA

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Introduction

Many men come to me with a combination of complaints rather than one isolated problem:

“Doctor, my belly has increased, my energy is low, sexual desire has reduced, erection is not as strong as before, and I feel that my testosterone may be low.”

These symptoms deserve careful assessment because there is a genuine relationship between **obesity, especially central or abdominal obesity, metabolic health, testosterone and male sexual function.**

However, the relationship is more complex than the popular statement that:

“Belly fat simply converts all testosterone into estrogen.”

Adipose tissue is not merely an inactive storage site for excess calories. It is biologically active tissue that influences inflammation, insulin sensitivity and hormone metabolism. Aromatase—the enzyme that converts androgens into estrogens—is present in adipose tissue, and obesity may alter aromatase activity. At the same time, obesity reduces sex hormone-binding globulin, affects hypothalamic and pituitary hormone signalling, promotes insulin resistance and inflammation, and is associated with sleep apnoea and other conditions capable of reducing testosterone.

This overall condition is often discussed clinically as **male obesity-related secondary hypogonadism**, sometimes abbreviated **MOSH**, or as a form of **functional secondary hypogonadism**.



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Current European Association of Urology guidance identifies central obesity, metabolic disorders and poor general health as major contributors to male hypogonadism and emphasizes that obesity-associated suppression of testosterone may be at least partly reversible.

I therefore explain to patients:

The increased waistline and the sexual problem may be connected, but we should diagnose the individual mechanism rather than treating a laboratory number alone.

What Is Male Hypogonadism?

Male hypogonadism means that the body does not produce adequate androgen activity—most importantly testosterone—to support normal physiological functions, and/or that sperm production is impaired because of problems involving the testes or the hormonal signals controlling them.

Testosterone contributes to:

sexual desire, spontaneous erections, muscle mass, bone health, body composition, mood, energy, red-blood-cell production and reproductive function.

Adult men with hypogonadism may experience low sexual desire, fewer morning erections, erectile difficulties, tiredness, changes in mood, reduced muscle mass, sleep disturbance, reduced body hair, breast enlargement and fertility difficulties. The EAU's 2026 patient information specifically notes that adult hypogonadism is more frequently seen in men who are overweight or have type 2 diabetes.

Importantly, these symptoms are **not specific to testosterone deficiency**.

Fatigue can result from poor sleep.

ED may result from diabetes or vascular disease.

Low libido can result from depression or relationship stress.

Therefore, a diagnosis requires symptoms **plus appropriately confirmed biochemical evidence**.

What Is Obesity-Related Secondary Hypogonadism?

In some men with obesity, the testes themselves may still be capable of producing testosterone, but signals from the hypothalamus and pituitary are suppressed or altered.

Normally:



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Hypothalamus → GnRH → Pituitary gland → LH/FSH → Testes → Testosterone and sperm production.

Luteinising hormone, or LH, stimulates Leydig cells in the testes to make testosterone.

Follicle-stimulating hormone, or FSH, contributes to sperm production.

In functional obesity-related secondary hypogonadism, testosterone may be low while LH and FSH are **low or inappropriately normal**, rather than markedly elevated.

This pattern differs from primary testicular failure, where the testes themselves are impaired and LH/FSH frequently increase in an attempt to compensate.

The distinction matters because treatment—particularly when fertility is important—is different.

Why Belly Fat Matters More Than Appearance

Abdominal obesity is not simply a cosmetic problem.

Visceral adipose tissue, the fat accumulated around the abdominal organs, is strongly associated with:

insulin resistance, type 2 diabetes, abnormal cholesterol, hypertension, chronic inflammation, fatty liver disease and cardiovascular disease.

All of these conditions can influence male sexual health.

Current EAU guidance states that low testosterone is common in men with obesity and that low testosterone is strongly associated with increased visceral adiposity. It also recognizes metabolic syndrome and type 2 diabetes as frequent companions of hypogonadism.

This can create a **two-way relationship**:

increasing obesity → declining testosterone in susceptible men → reduced muscle mass/activity and changes in body composition → greater tendency toward further adiposity.

This is sometimes called the **hypogonadal-obesity cycle**.

Adipose Tissue Is an Endocrine Organ

One of the most important developments in modern metabolic medicine has been recognition that fat tissue is metabolically active.

Adipose tissue releases or influences many signalling molecules, including:



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leptin, adiponectin, inflammatory cytokines and factors involved in insulin sensitivity.

As obesity progresses, enlarged adipocytes may develop altered oxygen supply, inflammatory signalling and changes in immune-cell activity.

This chronic low-grade inflammatory environment is one mechanism that may interfere with normal endocrine and vascular health.

The detailed material supplied for this article describes expansion of adipose tissue, macrophage-related inflammation and changes in metabolic signalling as part of obesity-associated endocrine dysfunction.

Aromatase: How Testosterone Can Be Converted Into Estradiol

The enzyme **aromatase**, encoded by the CYP19A1 gene, converts certain androgens into estrogens.

In men:

testosterone can be converted into estradiol, while androstenedione can be converted into estrone.

Estradiol is not a “female hormone that men should eliminate.”

Men require appropriate estrogen signalling for normal:

bone health, brain function, sexual physiology and other metabolic processes.

The problem is therefore not that men have estrogen.

The important issue is **hormonal balance and the functioning of the entire hypothalamic-pituitary-gonadal axis.**

A 2025 study in *The Journal of Clinical Endocrinology & Metabolism* found increased aromatase expression in adipose tissue from men with obesity, associated with adiposity, hyperglycaemia and insulin resistance. Interestingly, increased adipose aromatase did **not** directly correlate with circulating estradiol levels in that study, reminding us that the hormonal relationship is more complicated than “more fat always equals high blood estrogen.”

That nuance is important.

Belly Fat Does Not Simply “Turn Masculinity Into Estrogen”

The supplied background material places major emphasis on aromatization.



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Aromatization is biologically relevant, but modern understanding of obesity-related hypogonadism includes several mechanisms simultaneously:

reduced SHBG, insulin resistance, chronic inflammation, altered leptin signalling, sleep disturbance, metabolic disease, changes in gonadotropin secretion, medication effects and sometimes increased estrogenic feedback.

Therefore, I would not tell a patient:

“Your belly fat is destroying all your testosterone.”

I would say:

“Excess abdominal fat can disturb several hormonal and metabolic systems that help regulate testosterone. Losing excess fat can often improve this environment.”

That statement is both more accurate and more useful.

Why SHBG Is Important in Obesity

One reason testosterone interpretation becomes difficult in obesity is **sex hormone-binding globulin**, or SHBG.

Most testosterone circulating in blood is bound to proteins.

SHBG is one of the most important of these.

Obesity, insulin resistance, type 2 diabetes and metabolic syndrome commonly **reduce SHBG**.

When SHBG falls, total testosterone can also fall.

This means that an obese man may have a low total testosterone result without the degree of biologically active testosterone deficiency that the number initially appears to suggest.

In other patients, both total and free testosterone are genuinely reduced.

This is why testosterone should not be interpreted from one random blood test.

Obesity, Insulin Resistance and Testosterone

Insulin resistance is central to many cases of abdominal obesity.

The body produces insulin, but tissues respond inadequately.

This can lead to:



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higher insulin levels, type 2 diabetes, abnormal lipids, fatty liver and systemic metabolic dysfunction.

Low testosterone and insulin resistance are often found together, but one should be careful about claiming that one always directly causes the other.

Current evidence supports a **bidirectional association**.

Obesity can suppress testosterone.

Low testosterone may contribute to less favourable body composition.

Type 2 diabetes further increases the likelihood of hypogonadism and erectile dysfunction.

The result may be a metabolic-sexual cycle that requires treatment of the whole patient rather than simply testosterone replacement.

Inflammation and the Testes

Obesity is associated with chronic low-grade inflammation.

Inflammatory mediators such as TNF-alpha and interleukin-6 have been investigated for their effects on the hypothalamic-pituitary-gonadal axis and testicular steroid production.

Experimental research suggests that inflammation may interfere with Leydig-cell steroidogenesis.

The source material describes possible effects on the steroidogenic acute regulatory protein and other pathways necessary for testosterone synthesis.

This mechanism is biologically plausible, but patients should understand that routine clinical practice does not measure these cytokines to diagnose obesity-related hypogonadism.

The practical clinical assessment remains centred on symptoms, testosterone testing, gonadotropins and metabolic health.

Leptin and Male Hormonal Health

Leptin is produced by adipose tissue and helps communicate energy availability to the brain.

In obesity, leptin levels are often high, but the brain may become relatively resistant to its effects.

This phenomenon is called **leptin resistance**.

Leptin signalling interacts with reproductive pathways, and obesity-associated leptin dysfunction has been proposed as another contributor to functional hypogonadism.



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Again, this is part of a network rather than a single cause.

Sleep Apnoea: An Often-Missed Connection

One issue that deserves more attention than it receives is obstructive sleep apnoea.

Men with central obesity commonly have:

loud snoring, interrupted sleep, daytime sleepiness and episodes of breathing interruption.

Poor sleep can disrupt normal testosterone rhythms and contribute to:

fatigue, reduced sexual desire, hypertension, insulin resistance and erectile dysfunction.

Therefore, when an overweight patient tells me:

“My testosterone is low and I am tired all day,”

I also ask about sleep.

Treating sleep apnoea can be just as important as discussing hormones.

Why Low Testosterone Can Affect Sexual Desire

Testosterone plays an important role in libido.

A genuinely hypogonadal man may notice:

reduced sexual thoughts, reduced spontaneous desire, fewer morning erections and less interest in initiating sexual activity.

The EAU lists reduced libido, erectile dysfunction and decreased spontaneous or morning erections among the more specific sexual symptoms of adult hypogonadism.

However, low libido is multifactorial.

Depression, anxiety, relationship conflict, medications, chronic illness, sleep deprivation and sexual dissatisfaction can produce similar symptoms.

Does Low Testosterone Cause Erectile Dysfunction?

Testosterone contributes to sexual health, but a normal erection depends heavily on **blood vessels and nerves**.

Therefore, low testosterone and ED are related but not identical.

An obese man may have ED because of:



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vascular disease, diabetes, hypertension, endothelial dysfunction, smoking, performance anxiety or a combination of factors—even when his testosterone is adequate.

Conversely, a man with low testosterone may mainly complain of reduced libido rather than severe mechanical ED.

The 2026 EAU guideline notes that testosterone therapy can improve sexual function in genuinely hypogonadal men, but metabolic conditions such as obesity and diabetes tend to reduce the magnitude of improvement. Severe ED may require additional established ED therapy.

Obesity and Vascular Erectile Dysfunction

Obesity affects sexual health through more than hormones.

Erection requires healthy endothelial function and adequate nitric oxide activity.

Central obesity is associated with:

hypertension, dyslipidaemia, diabetes, chronic inflammation and atherosclerosis.

All can damage endothelial health.

The source material therefore correctly emphasizes that obesity can influence sexual function through both **endocrine and vascular pathways**.

This explains why simply normalising testosterone does not guarantee that severe ED will disappear.

Obesity and Male Fertility

Another important area is fertility.

Obesity has been associated in research with changes in:

testosterone regulation, semen quality, oxidative stress and reproductive hormones.

But a man's body weight alone cannot diagnose infertility.

If a couple has difficulty conceiving, evaluation may include semen analysis and appropriate hormonal assessment according to the clinical situation.

Most importantly:

Testosterone therapy and fertility treatment are not the same thing.

External testosterone can suppress LH and FSH and therefore suppress sperm production.



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The 2026 EAU guideline gives a strong recommendation **not to use testosterone therapy in men who wish to father a child**, while gonadotropin treatment may be used in appropriate men with true secondary hypogonadism who desire fertility.

This is a critical issue in my infertility practice.

Symptoms That May Suggest Obesity-Associated Testosterone Deficiency

A patient should consider evaluation when excess weight is accompanied by persistent symptoms such as reduced sexual desire, fewer morning erections, erectile dysfunction, unexplained loss of muscle strength, marked fatigue, low mood, reduced body hair, breast enlargement or fertility difficulties.

However, symptoms should guide testing rather than obesity alone.

Major endocrine guidelines do **not** recommend giving testosterone simply because a man has abdominal obesity.

The diagnosis requires an appropriate clinical picture and confirmed low testosterone.

How Testosterone Should Be Tested Properly

This is an area where many patients receive misleading information.

A testosterone test taken:

late in the afternoon, after eating, during acute illness or after poor sleep

may not accurately reflect baseline testosterone physiology.

The current 2026 EAU guideline recommends measuring total testosterone:

in the morning, between approximately 7:00 and 10:00 AM, in the fasting state, using a reliable assay.

If testosterone is low, it should be repeated on a separate occasion before treatment is started.

The EAU uses **12 nmol/L, approximately 3.5 ng/mL or 346 ng/dL**, as a useful threshold when considered together with relevant symptoms. Men below 8 nmol/L tend to have more marked biochemical deficiency and may derive greater symptomatic benefit from appropriate treatment.

Different professional organizations and laboratories may use somewhat different cut-offs, so the number must always be interpreted clinically.



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One Testosterone Test Is Not Enough

I strongly discourage patients from receiving a diagnosis like:

“Your testosterone was 280 once, therefore you need lifelong testosterone injections.”

Testosterone changes naturally according to:

time of day, food intake, sleep, acute illness, medications and laboratory methodology.

The 2026 EAU guideline recommends at least **two morning fasting measurements** when total testosterone is below the diagnostic threshold.

Symptoms must also be present.

What About Free Testosterone?

Free testosterone can be particularly useful in obesity because SHBG is frequently altered.

The EAU recommends measuring SHBG and calculating free testosterone when appropriate, especially in conditions known to alter SHBG such as:

obesity, insulin resistance, type 2 diabetes and metabolic syndrome.

Direct commercial free-testosterone assays are not all equally reliable.

Calculation using total testosterone, SHBG and albumin or specialized methods may provide better information in selected patients.

LH and FSH Help Identify the Cause

If testosterone is genuinely low, I want to know **why**.

LH and FSH help distinguish:

Primary hypogonadism

The testes are failing.

Testosterone is low and LH/FSH are usually elevated.

Secondary hypogonadism

The hypothalamus or pituitary is not providing enough stimulation.

Testosterone is low while LH and/or FSH are low or inappropriately normal.

Obesity-associated functional hypogonadism generally falls into the second category after other causes have been excluded.



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The EAU strongly recommends LH and FSH measurement to classify hypogonadism.

Should Estradiol Be Tested in Every Obese Man?

Not necessarily.

This is another place where the supplied material goes beyond standard clinical practice by presenting fixed testosterone-to-estradiol ratios and universal estradiol targets as central diagnostic tools.

Current major guidelines do **not** make a testosterone-to-estradiol ratio below 10 a standard diagnostic criterion for obesity-related hypogonadism.

Estradiol measurement can be helpful in selected situations—for example:

significant gynaecomastia, suspected estrogen excess, selected infertility evaluations or before considering particular off-label therapies.

But it should not replace proper testosterone, LH, FSH and SHBG assessment.

Prolactin, Thyroid and Other Investigations

Additional tests depend on the clinical picture.

Prolactin may be appropriate where low libido and secondary hypogonadism are present.

Thyroid testing may be considered when symptoms suggest thyroid disease.

Pituitary imaging is reserved for selected patients, particularly severe secondary hypogonadism, markedly abnormal prolactin or symptoms suggesting a pituitary lesion.

The EAU recommends pituitary MRI in appropriate secondary hypogonadism rather than routinely scanning everyone with obesity and low testosterone.

The Most Important Treatment: Weight Reduction

For obesity-related functional hypogonadism, **weight reduction is the foundation of treatment.**

The latest 2026 EAU guideline states that weight loss and lifestyle changes should be the **first approach for overweight or obese men with functional hypogonadism.**

This recommendation matters because weight loss addresses the metabolic cause rather than only replacing the hormone.

Weight reduction can improve:



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insulin sensitivity, cardiovascular health, inflammatory burden, sleep apnoea, physical fitness, body confidence and sometimes testosterone levels.

The improvement in testosterone from lifestyle intervention alone can be modest, but greater weight loss is generally associated with greater hormonal improvement.

Diet: Not a “Testosterone Food,” but a Metabolic Strategy

Patients frequently ask:

“Which food increases testosterone?”

There is no single food that reliably reverses obesity-related hypogonadism.

The objective is sustainable reduction of excess adiposity while preserving nutrition and muscle.

A suitable dietary plan should emphasize adequate protein, vegetables, whole foods, fibre and appropriate caloric intake while limiting excessive refined carbohydrates, sugar-rich drinks and highly processed foods.

For a patient with diabetes, fatty liver or kidney disease, dietary planning must also take those conditions into account.

The Unani system's traditional emphasis on **Ilaj bil Ghiza—dietotherapy—** fits particularly well here when interpreted responsibly and aligned with modern metabolic needs.

Exercise: Both Aerobic and Resistance Activity Matter

Exercise can help break the obesity-testosterone cycle.

Aerobic activity supports:

cardiovascular fitness, insulin sensitivity, weight loss and vascular health.

Resistance training helps:

preserve or increase muscle mass and improve metabolic health during weight reduction.

The EAU guideline reports that physical activity and weight loss are associated with improvement in testosterone and sexual health, though the average hormonal increase from lifestyle intervention may be modest.

The objective should not be extreme exercise.

Consistency matters more than punishment.



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Sleep and Stress Management

Weight reduction is much harder when a patient sleeps poorly.

Poor sleep can increase appetite, worsen metabolic health and reduce energy for physical activity.

If snoring, daytime sleepiness or witnessed breathing pauses are present, obstructive sleep apnoea should be considered.

Stress and depression should also be addressed because low mood itself can reduce sexual desire and contribute to ED.

A complete sexual-health programme therefore includes mental health, not just hormones.

What About GLP-1 Medicines and Testosterone?

This is an important area of recent research.

Medicines in the GLP-1 receptor agonist family are used for appropriate patients with obesity and/or type 2 diabetes.

Recent studies suggest that men who lose weight and improve metabolic health while taking these medicines may also experience improvements in reproductive hormones.

A 2025 systematic review and meta-analysis found an increase in bioavailable testosterone with GLP-1 receptor agonists, although effects on free testosterone and SHBG remained uncertain and the evidence base was still small.

Another 2025 meta-analysis involving seven studies reported increased total testosterone together with reductions in weight, BMI, waist circumference and HbA1c. The researchers cautioned that available evidence does not establish whether the effect is a direct testicular action or is mainly related to weight and metabolic improvement.

A newer 2026 meta-analysis also reported improvements in testosterone and some sexual outcomes among overweight and obese patients receiving GLP-1 receptor agonists, but this remains an evolving field.

These medicines should therefore be prescribed for appropriate metabolic indications—not casually as “testosterone boosters.”

Aromatase Inhibitors: Should Estrogen Simply Be Blocked?

Because aromatase converts testosterone into estrogen, it may appear logical to simply block aromatase.



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Medicines such as anastrozole and letrozole can increase testosterone and reduce estradiol in some men.

A meta-analysis found that aromatase inhibitors can improve testosterone levels in selected adult men with obesity- or age-related hypogonadism.

However, the 2026 EAU guideline makes an important point:

aromatase inhibitors and SERMs remain off-label treatments for functional secondary hypogonadism, and the quality of evidence is limited. Long-term excessive estrogen suppression can affect bone density, while some therapies may have other risks.

Therefore, aromatase inhibitors should not become an internet self-treatment for belly fat.

Estrogen is physiologically necessary in men.

SERMs Such as Clomiphene

Selective estrogen receptor modulators such as clomiphene can increase endogenous gonadotropin and testosterone production in some men with an intact hypothalamic-pituitary-testicular axis.

They are sometimes considered by specialists when preservation of fertility is important.

But their use in male hypogonadism is generally **off-label**, and current evidence is less robust than for established testosterone therapy.

Treatment requires professional monitoring.

Testosterone Replacement Therapy

If a man has:

persistent compatible symptoms, repeatedly low appropriately measured testosterone and no correctable or contraindicating cause,

testosterone replacement therapy may be considered after discussion of benefits and risks.

According to the EAU, testosterone therapy can improve sexual desire and other symptoms in appropriately diagnosed hypogonadal men. It should **not** be used in men with normal testosterone merely to reduce body weight or enhance metabolic status.

That distinction is essential.

Testosterone is not an anti-obesity supplement.



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Testosterone and Erectile Dysfunction

If low testosterone is truly contributing to ED, normalization can improve sexual function—particularly in men with more severe deficiency.

But men with obesity and diabetes may have vascular ED as well.

In those patients, testosterone alone may produce only partial improvement, and standard ED treatments may also be required.

This is why I treat **the patient, not just the hormone report.**

Testosterone and Fertility: A Critical Warning

A man who wants children should never casually start testosterone injections because:

external testosterone suppresses pituitary LH and FSH → intratesticular testosterone falls → sperm production can decline severely or stop.

The 2026 EAU guideline specifically states that testosterone therapy is contraindicated in men actively seeking fertility.

At Saira Health Care, where infertility is an important part of my practice, this question is always relevant:

“Do you want children now or in the future?”

The answer can completely change the treatment plan.

The Unani Perspective on Obesity and Male Sexual Health

The Unani system of medicine provides a useful traditional framework for looking at the patient as a whole.

Unani medicine originated from the Greco-Arabic medical tradition and was developed through the work of physicians such as Hippocrates, Galen, Al-Razi and Ibn Sina before becoming deeply established and further developed in the Indian subcontinent. CCRUM describes this historical development and emphasizes the continuing scientific standardization of Unani medicines.

In classical Unani thought, health is not considered merely an isolated abnormal laboratory value.

Assessment considers:

Mizaj – temperament,

Akhlat – the classical humours,



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nutrition and digestion,
physical activity,
sleep,
mental state,
and overall functioning of the body.

These traditional concepts should be distinguished from modern biochemical concepts such as testosterone, insulin and estradiol.

I believe both can be discussed honestly without pretending they are identical.

Obesity in Contemporary Unani Guidelines

CCRUM's recent **Standard Treatment Guidelines in Unani on Metabolic Disorders** recognizes obesity as multifactorial and specifically notes that endocrine disorders and obesity can influence each other. Hypogonadism in men is included among endocrine conditions associated with obesity.

This is an important development because it supports an approach in which a Unani practitioner does not simply prescribe a sexual tonic.

The underlying metabolic condition must be assessed.

Asbab-e-Sitta Zarooriya: The Six Essential Factors

One of the most useful Unani concepts for metabolic and sexual health is **Asbab-e-Sitta Zarooriya**, the six essential factors involved in health maintenance.

They broadly concern:

air and environment, food and drink, movement and rest, psychological activity and repose, sleep and wakefulness, and retention/elimination.

In modern practice these concepts create a structured way of discussing:

diet, obesity, physical activity, sleep, mental stress and daily routine.

These factors are particularly relevant because lifestyle intervention is also the first-line modern recommendation for obesity-related functional hypogonadism.

This is one area where Unani preventive principles and contemporary metabolic medicine can complement each other naturally.

Ilaj bil Ghiza – Dietotherapy



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For the obese patient with sexual and hormonal concerns, **Ilaj bil Ghiza** can be one of the most useful components of Unani care.

But it must be applied intelligently.

Traditional “strengthening” diets containing large quantities of sugar, honey, sweet Majun, dates or calorie-dense preparations may be unsuitable for a patient with:

central obesity, diabetes, fatty liver or metabolic syndrome.

The objective should be:

reduction of excess calories, adequate nutrition, control of metabolic risk and preservation of muscle.

A modern Unani treatment programme should adapt dietotherapy to the patient's actual metabolic condition.

Ilaj bit Tadbir – Regimenal Therapy

Unani regimenal therapy can support:

appropriate physical movement, daily routine, stress management and healthy sleep.

For a sedentary man with obesity, gradual regular exercise can have meaningful effects on both general and sexual health.

CCRUM has also used integrated Unani lifestyle clinics within programmes targeting diabetes, cardiovascular disease and other chronic conditions, incorporating diet therapy, regimenal therapy and health education.

This preventive orientation can be particularly valuable in obesity-related sexual dysfunction.

Ilaj bil Dawa – Unani Pharmacotherapy

CCRUM recognizes **Ilaj bil Dawa**, or pharmacotherapy, as one of the established modes of Unani treatment alongside dietotherapy and regimenal therapy.

CCRUM's sexual-debility guideline, under **Zuf-i-Bah**, includes traditional considerations such as reduced sexual desire, penile flaccidity and psychological factors, and also recommends investigations including blood glucose, lipid profile, testosterone, gonadotropins, liver function and kidney function.

This is significant.

It demonstrates that contemporary responsible Unani practice can integrate laboratory assessment rather than depending entirely on traditional symptom interpretation.



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Can Unani Medicines Increase Testosterone?

This question should be answered carefully.

Traditional Unani pharmacology includes a number of herbs and compound preparations historically used for general vitality or sexual health.

Some individual plants have laboratory, animal or limited human research suggesting effects on stress, reproductive parameters or sexual wellbeing.

But evidence for a **specific Unani formulation reversing obesity-related secondary hypogonadism** remains limited compared with evidence supporting weight reduction and established endocrine treatment.

Therefore, I would not promise:

“This Unani medicine will raise your testosterone by a fixed amount.”

Instead, where pharmacotherapy is appropriate, I would consider it as part of a broader individualized programme addressing:

weight, diet, sleep, stress, metabolic disease and sexual symptoms.

Why “Natural” Does Not Mean “Unlimited”

Men sometimes buy multiple products because the label says:

“natural,” “herbal,” or “testosterone booster.”

That does not guarantee effectiveness or safety.

Some preparations may:

interact with medicines, contain excessive sugars, affect the liver or kidneys, or contain poorly standardized ingredients.

CCRUM itself maintains drug-standardization programmes evaluating identity, purity, heavy metals, microbial contamination, aflatoxins and pesticide residues in Unani medicines.

Quality control matters.

Special Approach of Dr. Nizamuddin Qasmi at Saira Health Care

At **Saira Health Care**, when a patient comes with belly fat, low sexual desire, erectile difficulty or suspected low testosterone, I do not start by prescribing hormones or sexual tonics.



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I first try to determine which problem is actually present.

A typical assessment may consider whether the patient has central obesity, diabetes, insulin resistance, hypertension, abnormal cholesterol, sleep apnoea, confirmed hypogonadism, erectile dysfunction, psychological stress or infertility.

I also ask:

Is sexual desire reduced?

Are morning erections reduced?

Was testosterone measured correctly?

Was it repeated?

What are LH and FSH showing?

Is SHBG low because of obesity?

Does the patient want children?

Is there diabetes or cardiovascular risk?

Is ED mainly vascular, hormonal, psychological—or mixed?

Only then should an individualized treatment plan be developed.

Why Saira Health Care's Sexual-Health and Infertility Focus Matters

Obesity can affect several areas simultaneously:

sexual desire, erection, testosterone, confidence, metabolic health and reproductive function.

These problems are often treated separately.

A patient may visit one doctor for obesity, another for ED and another for infertility without anyone putting the complete picture together.

At Saira Health Care, my aim is to look at the interaction between these areas and determine where investigation or referral is required.

My professional training for this work includes:

BUMS – Hamdard University, Delhi

MD

CGO

Certificate in Infertility – MGBIMS, Delhi

Certificate in Urology – London, UK



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Masters in Male Infertility – MasterHealthPro (HealthPro)

Integrated Sexual and Reproductive Health – ISRH, UNFPA

My focused clinical interest is in sexual disorders and infertility, with an integrative approach that incorporates responsible Unani principles while using appropriate modern investigations.

A Practical Treatment Strategy

For most patients with obesity-related sexual and hormonal symptoms, I use a staged approach rather than chasing one testosterone number.

First, we confirm whether genuine testosterone deficiency exists.

Second, we assess obesity and metabolic risk—including diabetes, cholesterol and blood pressure where appropriate.

Third, we identify the sexual complaint separately: is it reduced desire, ED, premature ejaculation or fertility difficulty?

Fourth, we address reversible contributors such as obesity, inactivity, poor sleep, smoking and metabolic disease.

Fifth, we introduce an individualized diet and exercise strategy, which can incorporate the preventive and lifestyle principles of Unani medicine.

Sixth, when clinically appropriate, we consider Unani pharmacotherapy as supportive care with attention to formulation quality, sugar content and possible interactions.

Seventh, established endocrine or sexual-medicine treatments are used or referred for when indicated.

Finally, we reassess symptoms rather than treating laboratory values indefinitely.

How Much Weight Loss Is Needed?

There is no single percentage that guarantees testosterone normalization in every man.

Even modest weight reduction improves metabolic health.

Greater sustained weight loss tends to have a larger hormonal effect.

The EAU specifically notes that testosterone improvement correlates with the degree of weight reduction, although average improvement from conventional diet and exercise alone may be modest.

Therefore, the aim should not be:



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“Lose exactly 5 kg and testosterone will become normal.”

It should be:

“Reduce excess visceral fat sustainably and monitor how the metabolic and hormonal system responds.”

Can Losing Belly Fat Restore Testosterone Naturally?

In some men, yes.

Obesity-related functional hypogonadism can be partly reversible.

Current EAU guidance specifically describes suppression of the HPG axis in functional hypogonadism as potentially reversible and recommends lifestyle improvement and weight reduction before testosterone treatment where appropriate.

But recovery is not guaranteed.

If another cause of hypogonadism exists, weight loss may not fully correct testosterone.

Should Every Overweight Man Check Testosterone?

No.

Routine hormone screening simply because a man is overweight is not universally recommended.

Testing becomes particularly relevant when symptoms such as:

low libido, fewer morning erections, ED, infertility or other signs of androgen deficiency are present.

The European Society of Endocrinology similarly recommends testing obese men when clinical features suggest hypogonadism rather than screening every man solely because of obesity.

Frequently Asked Questions

Does belly fat really reduce testosterone?

It can.

Obesity, particularly central obesity, is strongly associated with reduced testosterone in many men.



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The mechanisms involve altered SHBG, metabolic disease, inflammation, hormonal signalling and possibly increased aromatization.

Does belly fat convert testosterone into estrogen?

Adipose tissue contains aromatase, which converts testosterone into estradiol.

Obesity can increase adipose aromatase activity, but it is inaccurate to say that all—or even most—testosterone simply gets converted into estrogen. A recent 2025 study found greater adipose aromatase expression in obese men but no direct relationship between that expression and circulating estradiol.

Is estrogen harmful in men?

No.

Men need estrogen for normal bone and other physiological functions.

Both excessive and excessively suppressed estrogen can create problems.

The goal is physiological balance, not eliminating estrogen.

Does low testosterone always cause erectile dysfunction?

No.

ED can result from vascular disease, diabetes, neurological illness, medicines, psychological factors and many other causes.

Testosterone is only one part of normal erectile physiology.

Can obesity cause low sexual desire?

Yes, particularly where obesity is associated with hypogonadism, fatigue, depression, diabetes or poor sleep.

However, libido should be evaluated individually rather than assuming testosterone is always responsible.

What is the best test for low testosterone?



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A properly performed **fasting morning total testosterone measurement**, repeated on another day when abnormal, is the foundation.

SHBG and calculated free testosterone may be particularly useful in obesity. LH and FSH help determine whether the problem is primary or secondary.

Is a testosterone-to-estrogen ratio necessary?

It is not currently a standard diagnostic requirement for obesity-related hypogonadism.

A fixed T:E ratio below 10 should not be used alone to diagnose or treat a patient.

Can exercise increase testosterone?

Weight loss and regular physical activity can produce a modest improvement in testosterone in obese men and provide much broader metabolic and cardiovascular benefits.

Can GLP-1 medicines improve testosterone?

Recent evidence suggests that testosterone may improve in some overweight or obese men treated with GLP-1 receptor agonists, particularly alongside weight and metabolic improvements. The evidence is still evolving, and these drugs should be used for appropriate obesity or diabetes indications rather than solely as testosterone therapy.

Should an obese man take an aromatase inhibitor?

Not routinely.

Aromatase inhibitors are off-label in male functional hypogonadism and evidence remains limited. Excessive estrogen suppression can also adversely affect bone.

Can testosterone help weight loss?

Testosterone therapy should not be prescribed as a weight-loss medicine.

Current EAU guidance specifically advises against using testosterone simply to reduce weight or enhance cardiometabolic health.

Can testosterone treatment reduce sperm count?



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Yes.

External testosterone suppresses gonadotropins and can substantially reduce or stop sperm production.

Men wishing to father children should tell their clinician before starting treatment.

Can Unani medicine help obesity-related sexual problems?

Unani medicine can be particularly useful as an **integrative lifestyle-oriented system** through dietotherapy, regimenal therapy, sleep and stress management, individualized assessment and appropriately selected traditional pharmacotherapy.

CCRUM's metabolic and sexual-debility guidance recognizes both metabolic disease and sexual health and includes modern investigations such as glucose, lipid profile and reproductive hormones.

However, no Unani formulation should be described as scientifically proven to permanently reverse every case of obesity-related testosterone deficiency.

When to Seek Medical Evaluation

A man with abdominal obesity should seek professional evaluation when low sexual desire, erection difficulties, reduced morning erections, unexplained fatigue, breast enlargement, infertility or other signs suggestive of testosterone deficiency persist.

Prompt assessment is especially important when ED appears alongside:

diabetes, high blood pressure, chest discomfort, severe breathlessness or other cardiovascular symptoms.

Likewise, severe headache or visual disturbance associated with markedly low testosterone can sometimes require assessment for pituitary disease.

A Message From Dr. Nizamuddin Qasmi

When a man tells me:

“My belly is increasing and my sexual strength is decreasing,”

I do not dismiss the connection.

There is a genuine relationship between metabolic health and male sexual health.

But I also do not reduce the entire problem to the statement:



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“Fat converts testosterone into estrogen.”

Your sexual function depends on much more than one hormone.

It depends on:

your metabolic health, circulation, nerves, sleep, psychological state, testosterone regulation, relationship and general health.

If your testosterone is genuinely low, I want to know why it is low.

If your erection is weak, I want to know whether the primary problem is hormonal, vascular, psychological or mixed.

If you want children, that changes the treatment completely.

And if obesity is one of the driving factors, reducing that metabolic burden is usually more meaningful than simply trying to increase a testosterone report.

I believe this is also where the holistic philosophy of Unani medicine can make an important contribution.

Through **Ilaj bil Ghiza, Ilaj bit Tadbir, attention to sleep, exercise, mental wellbeing and carefully selected therapy**, we can work on the patient's whole health rather than looking at only one number.

But Unani care should work alongside appropriate modern investigation—not instead of it.

A patient with true hypogonadism deserves proper hormonal evaluation.

A patient with diabetes deserves good diabetic care.

A man with infertility should not be given testosterone blindly.

And a patient with significant erectile dysfunction should receive a complete sexual and cardiovascular assessment.

My objective is not merely to raise testosterone. It is to improve the patient's metabolic health, sexual confidence, reproductive health and overall quality of life in a medically responsible way.

About the Author

Dr. Nizamuddin Qasmi

Founder & Chief Physician, Saira Health Care

Focused Practice in Sexual Disorders & Infertility



+91-9452580944



+91-5248-359480



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BUMS – Hamdard University, Delhi

MD

CGO

Certificate in Infertility – MGBIMS, Delhi

Certificate in Urology – London, UK

Masters in Male Infertility – MasterHealthPro (HealthPro)

Integrated Sexual and Reproductive Health – ISRH, UNFPA

At Saira Health Care, the clinical focus includes individualized assessment and integrative management of sexual disorders, male reproductive-health concerns and infertility, including conditions where metabolic and hormonal health may contribute to sexual dysfunction.

Medical Disclaimer

This article is intended for **health education and general patient awareness**. It does not replace an individual medical consultation, physical examination, laboratory assessment or treatment plan.

A diagnosis of testosterone deficiency should not be made from symptoms alone or from one random laboratory test. Testosterone therapy, aromatase inhibitors, SERMs, gonadotropins and obesity medications all require appropriate professional assessment.

Men who are planning fertility should tell their clinician before any hormonal treatment because external testosterone can suppress sperm production.

Unani medicines should also be used under qualified supervision, particularly in patients with diabetes, cardiovascular disease, liver or kidney problems or those already taking prescription medicines.



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