Male Hypogonadism and Infertility

Low Testosterone, Hormonal Causes of Male Infertility, Diagnosis, Fertility-Preserving Treatment and an 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

When a man comes to me with infertility and says, **"Doctor, my testosterone is low. Should I take a testosterone injection to increase my fertility?"**, I consider this one of the most important questions in male reproductive medicine.

The answer surprises many patients.

Testosterone is essential for sperm production—but taking testosterone from outside the body can actually suppress sperm production and sometimes cause severe oligozoospermia or azoospermia.

This apparent contradiction becomes easy to understand once we understand how the male hormonal system works.

Male hypogonadism is a clinical condition in which the testes do not produce enough testosterone, sperm, or both because of disease of the testes themselves or inadequate stimulation from the hypothalamus and pituitary gland. It can affect sexual desire, erections, energy, muscle and bone health, mood and fertility. The 2026 European Association of Urology guideline defines diagnosis through the combination of appropriate symptoms and consistently low testosterone, not through one isolated laboratory result.

Male hypogonadism and male infertility overlap, but they are **not identical conditions**.

A man can have low testosterone and still produce sperm. Another man can have normal serum testosterone but severe impairment of sperm production. A third may have low testosterone because of pituitary disease and have azoospermia that can sometimes be medically reversed.

This is why I believe the first goal should never be merely to "raise testosterone."

The real goal is to determine:

Why is testosterone low? Is fertility desired? Is sperm production preserved? And which treatment can improve hormonal health without damaging reproductive potential?

What Is Male Hypogonadism?

Male hypogonadism is a condition in which the male reproductive system is not functioning normally because testosterone production, sperm production, or both are impaired.

Testosterone is produced mainly by **Leydig cells inside the testes**.

But the testes do not operate independently.

They are controlled by a hormonal communication system called the:

Hypothalamic-Pituitary-Gonadal Axis — HPG Axis

The hypothalamus in the brain releases **gonadotropin-releasing hormone (GnRH)**.

GnRH stimulates the pituitary gland to release:

- **Luteinizing hormone (LH)**
- **Follicle-stimulating hormone (FSH)**

LH stimulates Leydig cells to produce testosterone.

FSH acts mainly through Sertoli cells and supports sperm development.

Together, FSH and very high concentrations of testosterone **inside the testes** are required for normal spermatogenesis.

If this system fails anywhere along the pathway, testosterone and fertility may be affected.

Male Hypogonadism and Infertility Are Related—but Different

This distinction is extremely important for patients.

Hypogonadism refers primarily to inadequate testicular endocrine/reproductive function.

Infertility refers clinically to failure to achieve pregnancy after an appropriate period of regular unprotected intercourse—WHO continues to use 12 months as the standard definition. Its first global infertility guideline was published in November 2025.

A man may therefore have hypogonadism without infertility if enough sperm remain.

Conversely, he may have severe infertility despite normal testosterone—for example because of a Y-chromosome microdeletion, maturation arrest, obstructive azoospermia or another spermatogenic problem.

Therefore:

Low testosterone does not automatically explain every abnormal semen report.

Why Testosterone Is Important for Sperm Production

Testosterone is essential for spermatogenesis.

But the testosterone concentration required inside the testes is much higher than the concentration measured in ordinary blood.

LH stimulates Leydig cells locally, producing high **intratesticular testosterone**, which supports germ-cell development.

This is why a man's blood testosterone can increase dramatically after an external testosterone injection while his sperm count falls.

The injected testosterone signals the brain:

“There is already enough testosterone.”

The hypothalamus and pituitary consequently reduce GnRH, LH and FSH.

The testes then lose the signals required for their own testosterone production and sperm development.

This can cause:

reduced sperm count → severe oligozoospermia → sometimes azoospermia.

The 2026 EAU guideline and the AUA/ASRM male-infertility guideline both strongly advise against testosterone therapy in men actively wishing to father children because exogenous testosterone suppresses gonadotropins and spermatogenesis.

The Most Important Warning for Men Trying to Conceive

Do Not Start Testosterone Replacement Therapy Simply Because Your Testosterone Is Low

This is one of the most important messages in this entire article.

Standard testosterone therapy can include:

- Testosterone injections
- Testosterone gels
- Testosterone patches
- Testosterone pellets
- Oral testosterone formulations
- Testosterone used for bodybuilding

These treatments can improve testosterone-deficiency symptoms in correctly selected men who are **not trying to preserve fertility**.

But in a man actively trying to become a father, conventional exogenous testosterone can suppress sperm production.

The Endocrine Society specifically recommends against starting testosterone therapy in men planning fertility in the near term.

The EAU's 2026 guideline goes further and lists active desire for children among the contraindications to testosterone therapy.

Types of Male Hypogonadism

Understanding the type of hypogonadism is essential because treatment differs completely.

The major classifications are:

1. Primary Hypogonadism

2. Secondary Hypogonadism

3. Functional Hypogonadism

Primary Hypogonadism

Primary hypogonadism means the main problem is **inside the testes**.

The pituitary gland may produce plenty of LH and FSH, but the testes do not respond adequately.

This is therefore also called:

Hypergonadotropic hypogonadism.

Typical laboratory pattern:

Low testosterone + high LH, often high FSH.

The EAU identifies Klinefelter syndrome as the most common genetic cause of primary hypogonadism and recognizes numerous acquired and developmental causes.

Causes of Primary Hypogonadism

Important causes include:

- Klinefelter syndrome
- Severe testicular damage
- Bilateral undescended testes
- Testicular torsion
- Testicular trauma
- Mumps orchitis
- Chemotherapy
- Testicular radiotherapy
- Some genetic disorders
- Testicular cancer or treatment
- Severe primary spermatogenic failure
- Age-related and chronic-disease-associated testicular dysfunction in selected men

In primary hypogonadism, the pituitary may already be stimulating the testes strongly.

This explains why simply giving more stimulation may have limited benefit if the testicular tissue itself has severe irreversible damage.

Klinefelter Syndrome

Klinefelter syndrome most commonly involves an additional X chromosome:

47,XXY

It is an important cause of primary hypogonadism and male infertility.

Affected men may have:

- Small testes
- Elevated FSH and LH
- Reduced testosterone
- Infertility
- Azoospermia
- Gynecomastia in some cases
- Reduced muscle mass or other androgen-deficiency features

The 2026 EAU guideline estimates Klinefelter syndrome at roughly 1 in 500–1,000 male births and notes that many affected individuals remain undiagnosed.

Some azoospermic men with Klinefelter syndrome still have isolated areas of sperm production and may be candidates for specialist sperm retrieval and ICSI.

Therefore, Klinefelter syndrome should not automatically be interpreted as absolute impossibility of biological fatherhood.

Secondary Hypogonadism

Secondary hypogonadism means the testes may be capable of working, but they are receiving inadequate stimulation from the **hypothalamus or pituitary gland**.

This is also called:

Hypogonadotropic hypogonadism.

The typical laboratory pattern is:

Low testosterone + low or inappropriately normal LH and FSH.

This distinction is extremely important for fertility.

The EAU states that, with appropriate treatment, both testosterone and fertility can potentially be restored in many men with secondary hypogonadism.

That makes secondary hypogonadism one of the most medically important causes of male infertility because, unlike many forms of severe primary testicular failure, it can sometimes respond very well to hormonal stimulation.

Causes of Secondary Hypogonadism

Important causes include:

- Congenital GnRH deficiency
- Kallmann syndrome
- Pituitary tumors
- Hyperprolactinemia
- Pituitary surgery
- Pituitary radiation
- Traumatic brain injury
- Hemochromatosis
- Severe systemic illness
- Certain medications
- Long-term opioid use
- Exogenous testosterone
- Anabolic steroids
- Severe obesity and metabolic dysfunction
- Other hypothalamic or pituitary disorders

The correct treatment depends entirely upon which of these mechanisms is present.

Congenital Hypogonadotropic Hypogonadism

Some males are born with inadequate GnRH or gonadotropin production.

Puberty may be delayed or incomplete.

Possible features include:

- Small testes
- Poor development of secondary sexual characteristics

- Reduced facial/body hair
- Small penis if deficiency began early
- Reduced libido
- Infertility

In **Kallmann syndrome**, impaired smell or absent sense of smell may accompany GnRH deficiency.

These patients are particularly important from a fertility perspective because the testes may be capable of producing sperm when appropriate hormonal stimulation is provided.

Functional Hypogonadism

Not every man with low testosterone has irreversible disease of the testes or pituitary gland.

The 2026 EAU guideline recognizes **functional hypogonadism**, in which testosterone suppression occurs in association with potentially modifiable conditions rather than a fixed organic lesion. Important contributors include obesity, metabolic disorders and chronic disease.

This is extremely relevant in modern male infertility practice.

A man may be:

- Significantly overweight
- Physically inactive
- Insulin resistant
- Sleeping poorly
- Using several medications
- Living with uncontrolled diabetes

and show reduced testosterone without having permanently failed testes.

In such cases, improving overall metabolic health may be an important part of treatment.

Obesity and Low Testosterone

Obesity and male hypogonadism can reinforce each other.

Excess adipose tissue increases aromatase activity, which converts more androgen to estrogen. Metabolic and inflammatory changes can also suppress the hypothalamic-pituitary-gonadal axis.

The EAU identifies obesity as a major cause of functional hypogonadism and recommends weight loss and lifestyle intervention as the first approach in overweight or obese men. Evidence shows that weight loss can increase testosterone and improve gonadotropin function, although the magnitude varies between individuals.

This is one area where modern endocrine care and the holistic lifestyle emphasis of Unani medicine can complement one another particularly well.

Diabetes and Metabolic Syndrome

Low testosterone is more frequently observed in men with:

- Type 2 diabetes
- Insulin resistance
- Central obesity
- Dyslipidemia
- Metabolic syndrome

The relationship works in both directions.

Metabolic ill health can suppress reproductive hormones, while hypogonadism can be associated with increased fat mass and reduced lean mass.

A patient with infertility, abdominal obesity and low testosterone therefore deserves metabolic evaluation rather than simply receiving a sexual-performance medicine.

Hyperprolactinemia

Prolactin is important in reproductive endocrinology.

When prolactin becomes excessively elevated, it can suppress GnRH and consequently reduce LH, FSH and testosterone.

Possible symptoms include:

- Reduced libido
- Erectile dysfunction

- Infertility
- Low testosterone
- Occasionally breast discharge
- Headache or visual symptoms if a large pituitary lesion is present

The EAU recommends prolactin measurement when secondary hypogonadism or low sexual desire raises suspicion and advises pituitary MRI when hyperprolactinemia or symptoms of a pituitary mass are present.

Treating hyperprolactinemia appropriately can sometimes restore the reproductive axis.

Opioids and Male Hypogonadism

Long-term opioid exposure can suppress the hypothalamic-pituitary-gonadal axis.

Men taking chronic opioid therapy may develop:

- Reduced testosterone
- Reduced libido
- Erectile dysfunction
- Fatigue
- Reduced fertility

Medication history is therefore essential.

A man should not abruptly stop necessary pain medicines, but potential opioid-induced hypogonadism should be discussed with the prescribing clinician.

Anabolic Steroids and "Gym Testosterone"

This has become an increasingly important fertility problem.

Some men use injectable or oral anabolic-androgenic steroids to increase muscle mass.

Initially they may feel sexually stronger because circulating androgen is high.

Meanwhile, LH and FSH are being suppressed.

The testes may become smaller.

Sperm production may fall dramatically.

Eventually semen analysis may show severe oligozoospermia or azoospermia.

Current EAU guidance recommends discontinuing anabolic steroids in infertile men and allowing approximately six to twelve months before considering selected fertility-restoring hormonal treatment, depending upon the clinical situation.

Recovery can take many months and sometimes longer.

Testosterone-Induced Infertility May Take Time to Recover

When exogenous testosterone is stopped, the HPG axis does not always recover immediately.

A 2025 Nature Reviews Urology review emphasizes that sperm production often resumes after cessation of TRT, but the timeline is highly variable and may take **months or years**, particularly after prolonged or high-dose androgen exposure.

The AUA/ASRM guideline similarly notes that most men eventually recover ejaculated sperm after testosterone-associated suppression, but recovery can be prolonged and occasionally require years.

This is why fertility goals should be discussed **before** starting testosterone—not after azoospermia develops.

Symptoms of Male Hypogonadism

Symptoms depend on the severity, age of onset and underlying cause.

Common symptoms can include:

- Reduced sexual desire
- Fewer spontaneous or morning erections
- Erectile dysfunction
- Fatigue
- Reduced energy
- Lower mood
- Reduced motivation
- Loss of muscle mass
- Increased body fat

- Reduced shaving frequency or body hair
- Reduced testicular size
- Gynecomastia
- Reduced bone density
- Infertility
- Difficulty concentrating
- Sleep disturbance

The EAU's updated patient information lists low libido, erection problems, tiredness, mood changes, reduced muscle mass, loss of body hair, reduced bone strength and infertility among common adult manifestations.

Sexual symptoms are among the most specific symptoms associated with male hypogonadism.

Low Libido and Infertility Can Reinforce Each Other

A patient may have low testosterone and reduced libido.

The couple is told to have intercourse repeatedly around ovulation.

The man already feels less sexual interest.

Now intercourse becomes scheduled and performance-oriented.

Anxiety develops.

Erectile difficulty may appear.

The couple therefore faces two overlapping reproductive problems:

impaired hormonal fertility + reduced sexual frequency or performance.

For this reason, male infertility care should ask about sexual function rather than looking only at semen concentration.

How Male Hypogonadism Is Diagnosed

A diagnosis should not be based on one random testosterone result.

Current guidelines require both:

clinical symptoms or signs
and
consistent biochemical evidence of low testosterone.

The Endocrine Society similarly advises diagnosing hypogonadism only in men with compatible symptoms/signs and unequivocally and consistently low testosterone.

Testosterone Should Be Checked in the Morning

The 2026 EAU guideline recommends measuring total testosterone in the **fasting state between approximately 7:00 and 10:00 AM**, because testosterone follows a daily rhythm and food intake can lower the measured level.

A low result should be repeated before making a diagnosis or beginning testosterone treatment.

EAU recommends at least two measurements when total testosterone is below 12 nmol/L.

For night-shift workers, interpretation may need to take sleep pattern into account rather than relying mechanically on clock time.

What Testosterone Level Is Considered Low?

Different professional organizations and laboratories do not use completely identical cut-offs.

The **2026 EAU guideline uses 12 nmol/L—approximately 3.5 ng/mL or 350 ng/dL—as a practical threshold for symptomatic late-onset hypogonadism**, with greater treatment benefit generally seen at lower levels.

The Endocrine Society places more emphasis on **unequivocally and consistently low testosterone according to validated laboratory reference ranges together with symptoms**, rather than treating one universal number as the diagnosis.

This is why a result near the borderline should be interpreted clinically rather than labelled automatically.

Free Testosterone and SHBG

Most testosterone in blood is bound to proteins, especially **sex hormone-binding globulin (SHBG)**.

Conditions that change SHBG can make total testosterone misleading.

For example, SHBG can be affected by:

- Obesity
- Diabetes/metabolic syndrome
- Thyroid disease
- Liver disease
- Certain medications
- Ageing

The 2026 EAU guideline therefore recommends measuring SHBG and calculating free testosterone when indicated.

This is particularly useful in obese men, in whom low SHBG can make total testosterone appear lower than the biologically available testosterone really is.

LH and FSH: Two of the Most Important Tests

Once low testosterone is confirmed, I want to determine **where the failure is occurring**.

Low testosterone + high LH/FSH

This suggests **primary testicular hypogonadism**.

Low testosterone + low or inappropriately normal LH/FSH

This suggests **secondary hypogonadism** involving the hypothalamus or pituitary.

The EAU and Endocrine Society both recommend LH/FSH assessment to differentiate primary from secondary disease.

This distinction can completely change infertility treatment.

Semen Analysis Is Essential When Fertility Is the Concern

A testosterone result cannot tell us how many sperm are being produced.

A man interested in fertility requires semen analysis.

Depending on findings, semen evaluation examines parameters such as:

- Volume
- Sperm concentration

- Total sperm number
- Motility
- Morphology

A severely abnormal result generally requires appropriate repeat evaluation.

The WHO 2025 infertility guideline emphasizes systematic investigation of biological causes affecting both partners rather than basing treatment upon one isolated result.

Other Tests May Be Required

Depending upon the patient, evaluation can include:

- Prolactin
- Estradiol
- Thyroid tests
- HbA1c or glucose
- Lipid profile
- Liver and kidney function
- Iron studies where hemochromatosis is suspected
- Pituitary hormonal testing
- Pituitary MRI
- Scrotal ultrasound
- Genetic testing
- Sperm DNA fragmentation in selected infertility cases

Investigations should be targeted according to the history and findings rather than ordering everything for every patient.

When Is Pituitary MRI Needed?

A pituitary MRI becomes particularly important if secondary hypogonadism is associated with:

- High prolactin
- Headache

- Visual disturbance
- Other pituitary hormone abnormalities
- Findings suggesting a pituitary mass

The EAU strongly recommends MRI in secondary hypogonadism when hyperprolactinemia or specific symptoms of pituitary disease exist and suggests considering MRI in severe secondary hypogonadism when total testosterone is below approximately 6 nmol/L.

Genetic Evaluation

When hypogonadism coexists with **azoospermia or severe impairment of sperm production**, genetic assessment can become essential.

Depending upon the semen profile and clinical findings, testing may include:

- Karyotype
- Y-chromosome microdeletion analysis
- Other selected genetic tests

Klinefelter syndrome is a particularly important cause of primary testicular hypogonadism and infertility.

Genetic counselling is important because some abnormalities can affect both the man's prognosis and future children.

Treatment Must Begin With One Question

Does the Patient Want Children Now or in the Future?

This question should ideally be asked **before any testosterone prescription is written**.

There are two fundamentally different treatment pathways.

If fertility is NOT currently desired

Conventional testosterone replacement may be appropriate in a correctly diagnosed symptomatic hypogonadal man after assessment of contraindications and risks.

If fertility IS desired

Treatment must attempt to improve testosterone **while protecting or restoring spermatogenesis**.

The two goals require different therapies.

Treatment of Secondary Hypogonadism When Fertility Is Desired

This is one of the areas where hormonal fertility treatment can be particularly successful.

If the testes remain functional but lack pituitary stimulation, we can replace the missing gonadotropin signals.

hCG Therapy

Human chorionic gonadotropin—**hCG**—acts similarly to LH.

It stimulates Leydig cells to produce testosterone inside the testes.

This is very different from injecting testosterone from outside the body.

With hCG, the testes themselves are stimulated.

FSH Therapy

FSH directly supports Sertoli cells and spermatogenesis.

In many men with hypogonadotropic hypogonadism, hCG is started and FSH is added when required; other protocols use combined treatment earlier.

The EAU considers gonadotropin treatment the standard fertility-preserving therapy for secondary hypogonadism and states that combined hCG/FSH treatment can produce better spermatogenic outcomes than hCG alone in appropriate patients.

How Long Does Gonadotropin Treatment Take?

Sperm production is slow.

A man should not expect the semen analysis to normalize within several days.

Treatment often requires months, and semen analysis is monitored periodically.

EAU male-infertility guidance advises regular semen assessment during gonadotropin treatment and addition or escalation of FSH when spermatogenesis is not adequately stimulated.

The final response depends upon factors including baseline testicular size, previous testosterone exposure and the underlying cause.

2026 Evidence on Gonadotropin Treatment

A systematic review and meta-analysis published in 2026 specifically evaluated gonadotropin therapy in azoospermic men with male hypogonadotropic hypogonadism, reflecting growing evidence that this is one of the relatively few severe male-infertility states in which medical treatment can restore spermatogenesis.

This is why accurately diagnosing secondary hypogonadism can make such a large difference.

A patient with treatable gonadotropin deficiency should not be managed in the same way as a patient whose testes have severe irreversible primary failure.

Pulsatile GnRH

When the primary defect lies in hypothalamic GnRH secretion and the pituitary remains functional, **pulsatile GnRH therapy** can theoretically restore gonadotropin secretion and induce spermatogenesis.

It can be effective but is logistically complicated and less commonly used than gonadotropin treatment.

A recent review on fertility-preserving hypogonadism treatment describes GnRH as physiologically effective but relatively impractical in routine care.

Treatment of Hyperprolactinemia

When prolactin is truly elevated, the underlying cause should be investigated.

For prolactin-producing pituitary disorders, dopamine agonists may lower prolactin and allow the reproductive hormonal axis to recover.

The EAU recommends dopamine-agonist treatment in men with proven hyperprolactinemia where appropriate.

A low-testosterone patient with hyperprolactinemia should therefore not simply be given testosterone before the pituitary problem is understood.

SERMs Such as Clomiphene

Selective estrogen receptor modulators (SERMs) such as clomiphene have been used off-label in selected hypogonadal men who wish to preserve fertility.

They work centrally by reducing estrogen-mediated negative feedback on the hypothalamic-pituitary system.

This can increase the body's own:

- LH
- FSH
- Testosterone

Because the testes remain stimulated, spermatogenesis may be better preserved than with exogenous testosterone.

The AUA/ASRM guideline states that clinicians may consider SERMs, hCG, aromatase inhibitors or combinations in infertile men with low serum testosterone.

However, these are not universal treatments.

Clomiphene Is Not Appropriate for Every Patient

Clomiphene requires a functional hypothalamic-pituitary-testicular axis.

If the testes have severe primary failure and LH is already very high, stimulating the pituitary further may accomplish little.

The 2026 EAU guideline specifically notes that SERMs and aromatase inhibitors require an intact HPG axis and do not work adequately in severe primary hypogonadism or organic destruction of the central axis. It also describes the overall evidence base as limited.

A 2026 systematic review comparing clomiphene with conventional testosterone therapy also found ongoing uncertainty and conflicting randomized evidence regarding symptom improvement, despite clomiphene's biological appeal for fertility preservation.

So I do not regard clomiphene as an automatic “fertility-friendly testosterone tablet.”

Patient selection matters.

Aromatase Inhibitors

Aromatase inhibitors reduce conversion of testosterone into estrogen.

They are sometimes considered off-label in selected men, particularly those with obesity or an unfavorable testosterone-to-estrogen hormonal environment.

By lowering estrogen feedback, LH and FSH may increase.

However, current EAU evidence remains limited, and long-term excessive estrogen suppression can adversely affect bone health.

These drugs therefore require specialist supervision rather than self-medication.

Primary Hypogonadism and Fertility

Primary hypogonadism is generally more challenging.

If the testes themselves are severely damaged, hCG cannot make non-functioning Leydig or germ cells normal simply by increasing stimulation.

The EAU makes this biological distinction clearly: in primary hypogonadism, fertility cannot generally be restored simply by replacing pituitary signals in the way that it can in many cases of secondary hypogonadism.

Treatment therefore depends on the remaining testicular function.

What If a Man With Primary Hypogonadism Still Produces Some Sperm?

If sperm are present in the semen—even at a very low concentration—fertility options depend on:

- Sperm number
- Motility
- Female partner's age
- Ovarian reserve
- Duration of infertility
- Other female factors

Options may include natural attempts, IUI in selected milder cases, or IVF/ICSI for more severe male factor.

If sperm production is unstable or extremely low, sperm cryopreservation may sometimes be considered.

What If Azoospermia Is Present?

In men with severe primary testicular failure and non-obstructive azoospermia, specialist assessment may consider whether focal sperm production could be found through testicular sperm retrieval such as **micro-TESE**.

If mature sperm are successfully retrieved, they may be used with ICSI.

But this is not the same as treating the hypogonadism.

It is a reproductive strategy for obtaining sperm despite severe testicular dysfunction.

Testosterone Replacement Therapy When Fertility Is NOT Desired

For men with confirmed symptomatic hypogonadism who are not seeking fertility, testosterone therapy can be effective.

The 2026 EAU guideline reports that testosterone therapy can improve several outcomes in appropriately diagnosed hypogonadal men, particularly:

- Sexual desire
- Mild erectile dysfunction
- Sexual activity
- Body composition
- Bone mineral density
- Some hypogonadal symptoms

The benefit is generally greater in men with genuinely low baseline testosterone than in men whose testosterone is already normal.

Testosterone should **not** be prescribed to eugonadal men simply as a general sexual-performance or fertility medicine.

Testosterone Therapy Requires Monitoring

For men appropriately receiving testosterone therapy, monitoring can include:

- Symptoms
- Testosterone level
- Hematocrit
- Blood pressure
- PSA according to age/risk and relevant guidelines

- Metabolic health
- Adverse effects

The EAU recommends early reassessment after starting therapy and continued monitoring, particularly because testosterone can raise hematocrit.

However, in an infertility patient, the most important point remains that standard testosterone replacement is generally **not the treatment of choice when active fatherhood is desired**.

Fertility Preservation Before Testosterone Therapy

A young hypogonadal man may need long-term androgen treatment but not currently be ready to have children.

This creates an important counselling opportunity.

If sperm are present, **semen analysis and sperm cryopreservation may be worth discussing before long-term testosterone treatment**, particularly when future fertility is uncertain.

The Endocrine Society specifically notes that men who are uncertain about future conception may wish to bank sperm before testosterone suppresses spermatogenesis.

This conversation is often much easier before treatment than after years of gonadotropin suppression.

Recovery After Testosterone-Induced Azoospermia

If infertility develops after exogenous testosterone or anabolic-steroid use, treatment commonly begins by stopping the suppressive androgen under appropriate medical supervision.

Some men recover spontaneously.

Others may need fertility-directed therapy such as:

- hCG
- FSH
- Selected SERMs
- Selected combinations

A recent review confirms that recovery varies according to duration, dose and preparation and that specialized hormonal treatment may be useful when spontaneous recovery is slow.

It is important not to guarantee a particular recovery time.

Lifestyle Treatment in Functional Hypogonadism

When the problem is largely functional—for example obesity-associated secondary suppression—lifestyle treatment is medically meaningful.

I focus on:

- Gradual weight reduction
- Regular physical activity
- Better sleep
- Diabetes control
- Reduction of excessive alcohol
- Smoking cessation
- Adequate diet
- Treatment of associated metabolic disease

EAU 2026 strongly recommends lifestyle improvement, weight reduction, management of comorbidities and withdrawal of contributing medications when possible before moving directly to testosterone therapy.

WHO's 2025 infertility guideline similarly recommends healthy diet, physical activity and tobacco cessation for people planning or attempting pregnancy.

Sleep and Testosterone

Sleep deserves more attention than it often receives.

Poor sleep, shift work, obesity and sleep-disordered breathing can coexist with reduced testosterone and poor metabolic health.

Good sleep alone cannot correct Klinefelter syndrome or pituitary disease.

But in functional hypogonadism, improving sleep and overall health can be an important part of reversing suppressive lifestyle factors.

Male Hypogonadism and Erectile Dysfunction

Low testosterone can contribute to erectile dysfunction, particularly when accompanied by reduced libido and other androgen-deficiency symptoms.

However, erectile dysfunction has many other causes:

- Diabetes
- Vascular disease
- Hypertension
- Smoking
- Obesity
- Psychological stress
- Medication effects

Therefore, giving testosterone to every man with ED is inappropriate.

The EAU states that testosterone can improve sexual function in genuinely hypogonadal men but provides no demonstrated benefit for sexual dysfunction in men with normal testosterone levels.

Male Hypogonadism and Low Sexual Desire

Low libido is one of the more specific sexual manifestations of androgen deficiency.

But low desire can also occur with:

- Depression
- Relationship problems
- Chronic stress
- Poor sleep
- Medication use
- Hyperprolactinemia
- Chronic illness

This is why low libido plus one low testosterone result should not automatically lead to TRT.

A complete assessment is more appropriate.

Male Hypogonadism and Bone Health

Severe longstanding testosterone deficiency can reduce bone mineral density.

Men may develop osteopenia or osteoporosis.

The EAU notes a stronger relationship between severe hypogonadism and bone loss than between mild testosterone reduction and osteoporosis. Testosterone replacement can improve bone mineral density in appropriately treated men, although fracture-risk evidence is less certain.

This illustrates why male hypogonadism is not merely a sexual problem.

Male Hypogonadism and Mental Health

Low testosterone can coexist with:

- Low mood
- Reduced motivation
- Fatigue
- Lower quality of life

However, testosterone is not a general antidepressant.

The EAU notes relatively small improvements in mild depressive symptoms in appropriately diagnosed hypogonadal men and recommends conventional psychiatric treatment for severe depression.

Mental-health symptoms deserve independent assessment.

Couple-Based Infertility Assessment

When hypogonadism is discovered in the male partner, I still consider it essential that the female partner be evaluated at the same time.

Suppose a man with secondary hypogonadism may require several months of gonadotropin treatment.

If his partner is 26 with strong ovarian reserve, the couple may have more time.

If his partner is older with markedly reduced ovarian reserve, waiting a long period without parallel fertility planning may reduce overall reproductive opportunity.

WHO's 2025 infertility guideline emphasizes person-centred and couple-oriented assessment and progressive treatment based upon clinical findings and patient preferences.

Male treatment should therefore not occur in isolation.

When Assisted Reproduction May Be Needed

Even after successful hormonal treatment, semen quality may remain inadequate for natural conception.

Depending on the couple, fertility treatment may involve:

- Timed intercourse
- Intrauterine insemination
- IVF
- ICSI
- Surgical sperm retrieval in selected severe male-factor cases

The appropriate option depends upon the post-treatment semen profile and the female partner's reproductive status.

Increasing testosterone is not itself the final fertility outcome.

The real objective is a safe path toward pregnancy and live birth.

Male Hypogonadism and the Unani System of Medicine

As a physician trained in the **Unani System of Medicine**, I believe male hypogonadism is an excellent example of why traditional medicine should be applied through **cause-oriented, individualized clinical reasoning** rather than through one universal "strength medicine."

Official CCRUM material describes a foundational Unani principle called:

Izala-i-Sabab — removal or correction of the causative factor

CCRUM also describes treatment through:

- **Ilaj-bil-Ghiza — Dietotherapy**

- **Ilaj-bil-Tadbir — Regimental therapy**
- **Ilaj-bil-Dawa — Pharmacotherapy**
- **Ilaj-bil-Yad — Surgical/procedural treatment where appropriate**

and emphasizes a holistic assessment of the patient's constitution, lifestyle, emotional state and organ function.

I find this cause-oriented philosophy particularly relevant to hypogonadism.

How I Apply Izala-i-Sabab to Hypogonadism

Consider five different patients with low testosterone.

Patient One

He is severely obese with diabetes and functional suppression of the HPG axis.

For him, weight and metabolic management are fundamental.

Patient Two

He has hyperprolactinemia caused by pituitary disease.

The prolactin disorder needs treatment.

Patient Three

He is injecting bodybuilding testosterone.

The exogenous androgen is suppressing his fertility.

Patient Four

He has congenital hypogonadotropic hypogonadism.

He may need gonadotropins to induce spermatogenesis.

Patient Five

He has primary testicular failure from Klinefelter syndrome.

Simply giving a general reproductive tonic cannot reverse the chromosome abnormality.

All five men have “low testosterone,” but their treatment is completely different.

This is exactly why **Izala-i-Sabab—identify the cause before treating the symptom—remains a useful clinical principle**.

Ilaj-bil-Ghiza — Dietotherapy

Diet has particular relevance in men with functional hypogonadism associated with:

- Obesity
- Diabetes
- Metabolic syndrome
- Poor nutrition

An individualized dietary plan can support:

- Weight reduction
- Better insulin sensitivity
- Cardiovascular health
- General energy
- Reproductive health

This is not merely traditional reasoning.

Modern EAU guidance also recommends weight reduction and lifestyle treatment as first-line management for obesity-associated functional hypogonadism.

Therefore, **Ilaj-bil-Ghiza can form a meaningful supportive bridge between Unani lifestyle principles and contemporary endocrine care**.

Is There a Special "Testosterone-Boosting Diet"?

No particular food has been proven to cure genuine male hypogonadism.

A diet can improve nutritional and metabolic conditions that contribute to functional hormone suppression, but it cannot:

- Correct Klinefelter syndrome
- Remove a pituitary tumor
- Replace absent LH/FSH in severe congenital deficiency
- Regenerate severely damaged testes

Therefore, I avoid promising that any single food, seed, herb or tonic will normalize testosterone and fertility in every patient.

Ilaj-bil-Tadbir — Regimental Therapy and Lifestyle

Within Unani medicine, Ilaj-bil-Tadbir involves non-drug regimental approaches and broader lifestyle management.

For a patient with hypogonadism and infertility, a modern individualized version may pay attention to:

- Exercise
- Body weight
- Sleep
- Stress management
- Daily routine
- Smoking
- Alcohol
- Metabolic disease

These are particularly useful in functional hypogonadism.

They can also improve general health in men whose underlying hypogonadism is not fully reversible.

Ilaj-bil-Dawa — Unani Pharmacotherapy

Unani medicine contains numerous single and compound medicines historically used for male vitality and reproductive concerns.

A responsible contemporary approach requires a clear distinction between **traditional use** and **scientifically demonstrated fertility treatment**.

At present, high-quality evidence does **not** establish a specific Unani formulation as a substitute for hCG/FSH in true hypogonadotropic hypogonadism, as a cure for Klinefelter syndrome, or as a reliable treatment for severe primary testicular failure.

Therefore, I use the concept of Unani pharmacotherapy most appropriately as an **individualized supportive component**, selected after establishing the endocrine and reproductive diagnosis.

It should not delay evidence-based treatment of a pituitary disorder, genetic disease or severe male-factor infertility.

Where Unani Treatment May Be Particularly Useful

The potential supportive role becomes more clinically meaningful when low testosterone coexists with modifiable problems such as:

- Obesity
- Poor diet
- Inactivity
- Stress
- Sleep disturbance
- Metabolic dysfunction
- General health deterioration

These factors fit naturally into a holistic Unani assessment and are also recognized by modern guidelines.

This is one of the areas where integration can be genuine rather than merely combining medicines from two systems.

Unani Medicine Should Not Be Used to Hide a Correctable Endocrine Disease

If prolactin is high because of a pituitary tumor, the patient needs appropriate endocrine evaluation.

If LH and FSH are absent because of congenital hypogonadotropic hypogonadism, fertility-directed gonadotropin treatment may be necessary.

If exogenous testosterone is suppressing sperm, the hormonal plan needs to be changed.

If severe primary testicular failure is present, prognosis and assisted reproduction must be discussed realistically.

Using a tonic for months while these conditions remain undiagnosed wastes reproductive time.

The Saira Health Care Approach to Male Hypogonadism and Infertility

At **Saira Health Care**, my approach begins with the principle that low testosterone and male infertility must be investigated together—but not confused with one another.

When a patient arrives with low testosterone, I want to answer several questions before recommending treatment.

1. Is the Testosterone Truly Low?

Was it tested in the morning?

Was the patient fasting?

Was the result repeated?

Are symptoms present?

Could obesity or abnormal SHBG be distorting the total testosterone result?

Current EAU 2026 recommendations emphasize exactly these diagnostic safeguards.

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2. Is the Problem Primary or Secondary?

LH and FSH help answer this.

High gonadotropins suggest primary testicular failure.

Low or inappropriate gonadotropins suggest secondary disease.

This distinction directly changes fertility treatment.

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3. Does the Patient Want Children?

This may be the most important treatment question.

A man who does not desire fertility and has confirmed symptomatic hypogonadism may be suitable for testosterone therapy.

A man actively seeking pregnancy should usually **not be placed on conventional testosterone monotherapy**.

Instead, fertility-preserving strategies need to be considered.

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4. What Does the Semen Analysis Show?

Low testosterone alone does not tell me whether sperm are normal.

A fertility evaluation therefore includes semen analysis and appropriate repeat testing.

The treatment of a man with normal sperm differs from someone with severe oligozoospermia or azoospermia.

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5. Is There a Reversible Cause?

I look for:

- Obesity
- Metabolic syndrome
- Hyperprolactinemia
- Pituitary disease
- Exogenous testosterone
- Anabolic steroids
- Opioids
- Other medication effects
- Thyroid or systemic disease

Correcting a cause may be more effective than simply treating the laboratory number.

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6. Does the Patient Need Genetic Evaluation?

Severe sperm deficiency combined with primary hypogonadism can indicate an underlying genetic disorder.

The investigation should be appropriate to the semen profile and clinical findings.

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7. Can Fertility Be Restored Medically?

In secondary hypogonadism, often yes.

hCG and FSH can stimulate testicular testosterone production and spermatogenesis in appropriate men.

This is very different from conventional TRT.

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8. Is Assisted Reproduction Needed?

If adequate sperm cannot be restored, reproductive options may include IVF/ICSI or sperm retrieval depending upon the underlying testicular condition.

The female partner's reproductive status is evaluated simultaneously.

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9. Where Can Unani Support Add Value?

I then consider diet, weight, sleep, activity, stress and other individualized health factors through an integrative Unani framework.

The objective is not to replace the endocrine diagnosis but to strengthen the patient's overall health while the actual reproductive problem is managed appropriately.

Saira Health Care's Contribution in Sexual Disorders and Infertility

At **Saira Health Care**, focused work in sexual disorders and infertility is particularly relevant because hypogonadism can affect several systems at the same time.

A patient may present with:

- Infertility
- Low libido
- Erectile dysfunction
- Reduced energy
- Abnormal sperm production
- Psychological distress
- Relationship difficulties

These complaints should not be treated independently without asking whether a hormonal disorder connects them.

Likewise, not every infertility patient should receive hormone treatment.

The contribution of a focused sexual and infertility clinic is in **differentiating the mechanism and coordinating the correct pathway**.

Common Mistakes I Encourage Patients to Avoid

Taking testosterone because sperm count is low

Testosterone can worsen sperm production.

Taking "testosterone boosters" before establishing a diagnosis

Low testosterone has many causes.

Testing testosterone randomly in the afternoon and acting on one result

Diagnosis generally requires properly timed repeat testing.

Ignoring LH and FSH

These hormones tell us where the problem is located.

Treating only the male partner for months

Female fertility should be evaluated in parallel.

Assuming every low testosterone level needs TRT

Functional causes such as obesity may respond to lifestyle and metabolic treatment.

Assuming every fertility-preserving drug works in primary testicular failure

Clomiphene, hCG and similar therapies depend upon the underlying physiology.

Believing normal testosterone means normal fertility

Severe sperm disorders can occur despite normal testosterone.

When Should a Man Seek Medical Evaluation?

Professional evaluation is particularly appropriate when a man has:

- Infertility
- Low libido
- Persistent erectile difficulty
- Very low morning testosterone
- Reduced testicular size
- Delayed or incomplete puberty
- Gynecomastia
- Loss of body hair
- Unexplained fatigue with other androgen-deficiency symptoms
- Azoospermia or severe oligozoospermia
- Previous chemotherapy
- Previous undescended testes
- Use of testosterone or anabolic steroids
- Symptoms of pituitary disease

Headache plus visual disturbance and severe secondary hypogonadism deserves particular attention because pituitary pathology may be present.

Can Male Hypogonadism Be Cured?

There is no single answer.

Some forms can be substantially corrected.

Examples include:

- Weight-associated functional hypogonadism
- Hyperprolactinemia
- Testosterone-induced suppression
- Some medication-related cases
- Hypogonadotropic hypogonadism

Other forms are usually permanent.

Examples include:

- Severe primary testicular failure
- Some genetic conditions
- Major irreversible testicular injury

Even when the hormonal condition cannot be cured, symptoms and fertility options can often still be managed.

The key is accurate classification.

Frequently Asked Questions

Does low testosterone always cause infertility?

No. Low testosterone can contribute to infertility, but some hypogonadal men still produce sperm. Conversely, some men with normal testosterone have severe sperm-production disorders.

Can I take testosterone injections while trying for pregnancy?

Conventional exogenous testosterone should generally not be used in men actively seeking fertility because it suppresses LH, FSH and spermatogenesis.

Why does testosterone reduce sperm if testosterone is necessary for sperm production?

Because externally administered testosterone suppresses pituitary LH and FSH, reducing the extremely high **intratesticular testosterone** needed for spermatogenesis.

Is low testosterone diagnosed from one blood test?

No. Current guidelines recommend compatible symptoms plus consistently low testosterone, generally confirmed with properly timed repeat morning measurements.

What is the EAU threshold for low testosterone?

The 2026 EAU guideline uses **12 nmol/L, approximately 3.5 ng/mL or 350 ng/dL**, as a practical threshold for symptomatic late-onset hypogonadism. Interpretation still needs symptoms, repeat testing and appropriate assessment.

What do LH and FSH tell us?

High LH/FSH with low testosterone generally suggests primary testicular failure. Low or inappropriately normal LH/FSH suggests secondary hypothalamic-pituitary hypogonadism.

Can secondary hypogonadism cause azoospermia?

Yes. Severe gonadotropin deficiency can cause azoospermia. Importantly, some of these patients can restore spermatogenesis with hCG/FSH therapy.

Can hCG improve fertility?

Yes, particularly in appropriately diagnosed secondary or hypogonadotropic hypogonadism. FSH may also be required.

Can clomiphene preserve fertility?

It may increase endogenous LH, FSH and testosterone in selected men with an intact HPG axis and is used off-label. Evidence is less robust than for established gonadotropin treatment in true hypogonadotropic hypogonadism.

Can obesity lower testosterone?

Yes. Obesity is an important contributor to functional secondary hypogonadism, and weight reduction can improve testosterone in some men.

Can bodybuilding steroids cause infertility?

Yes. Anabolic steroids suppress LH and FSH and can severely reduce sperm production.

How long does fertility take to return after stopping testosterone?

Recovery varies greatly. Many men recover over months, while some take substantially longer—occasionally years—especially after prolonged or high-dose exposure.

Can Klinefelter syndrome cause hypogonadism and infertility?

Yes. It is an important genetic cause of primary hypogonadism and severe male infertility.

Can Unani medicine raise testosterone?

An individualized Unani approach may support general health, nutrition, weight, sleep and metabolic factors that influence functional hormone status. However, there is not strong evidence that a specific Unani medicine can reliably cure organic primary hypogonadism, pituitary disease or genetic testicular failure.

A Message to My Patients

When a patient tells me:

“Doctor, my testosterone is low, so please give me testosterone because we want a baby,”

I explain that fertility treatment cannot be based on the testosterone number alone.

We need to know:

Why is it low?

If your pituitary gland is not stimulating your testes, we may need to stimulate the testes rather than replace testosterone from outside.

If obesity and metabolic problems are suppressing your hormones, treating those causes may be important.

If testosterone injections themselves caused the sperm count to fall, we need a fertility-recovery plan.

And if your testes have severe primary failure, simply increasing the blood testosterone level will not necessarily restore sperm production.

That is why the diagnosis matters more than the name of the medicine.

I also tell my patients:

Low testosterone does not make you less masculine.

And an abnormal semen analysis does not measure your value as a husband, partner or man.

These are medical conditions involving hormones and reproductive biology.

They should be treated scientifically and without shame.

About Dr. Nizamuddin Qasmi

Dr. Nizamuddin Qasmi
Founder & Chief Physician, Saira Health Care
Focused Practice in Sexual Disorders & Infertility

Professional Qualifications and Training

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 focused clinical approach to sexual disorders and infertility includes assessment of male reproductive hormones, sexual function, semen abnormalities, fertility-related counselling and individualized Unani supportive care.

In a condition such as male hypogonadism with infertility, the objective is not merely to raise serum testosterone. It is to identify whether the problem lies in the testes, pituitary-hypothalamic system, medication exposure, metabolic health or another cause—and then select a treatment that respects the patient's fertility goals.

When endocrinology, genetics, advanced andrology, pituitary evaluation, surgical sperm retrieval or assisted reproduction is required, appropriate specialist collaboration should form part of comprehensive care.

Conclusion

Male Hypogonadism and Infertility represent a complex interaction between hormones, testicular function, sexual health and reproductive potential.

The most important distinction is between **primary hypogonadism**, in which the testes themselves are failing, and **secondary hypogonadism**, in which inadequate hypothalamic or pituitary stimulation prevents normal testicular function. Modern guidelines recommend using symptoms, repeated morning testosterone measurements, LH, FSH and targeted additional testing to establish this distinction.

The 2026 EAU update emphasizes fasting morning testosterone testing, repeat confirmation, consideration of SHBG/free testosterone when appropriate, and investigation of associated diseases and medications before treatment.

For infertile men, the central therapeutic principle is particularly important:

Standard exogenous testosterone should not be used as fertility treatment.

It may improve hypogonadal symptoms, but it suppresses gonadotropins and sperm production.

In contrast, men with **secondary hypogonadism** who desire fatherhood may benefit substantially from fertility-preserving treatment with **hCG and FSH/gonadotropins**, because these therapies stimulate the testes rather than shutting down the reproductive axis.

SERMs and aromatase inhibitors may have roles in selected men with an intact hormonal axis, but they are largely off-label and supported by less robust evidence.

Functional hypogonadism associated with obesity and metabolic disease deserves equally serious attention. Weight reduction, physical activity and treatment of metabolic abnormalities can improve testosterone in some men and are strongly recommended as part of care.

From the **Unani perspective**, the classical principles of **Izala-i-Sabab, Ilaj-bil-Ghiza, Ilaj-bil-Tadbir and individualized Ilaj-bil-Dawa** offer a useful whole-person framework, particularly for diet, weight, lifestyle, sleep, stress and general health. Official CCRUM material specifically emphasizes identifying the causative factor and treating the patient holistically.

However, responsible integrative care also means understanding the limits of every treatment. A herbal preparation cannot correct Klinefelter syndrome, replace missing pituitary hormones in severe hypogonadotropic hypogonadism or regenerate severely damaged testicular tissue with guaranteed success.

At **Saira Health Care**, my preferred approach is therefore:

confirm the hormonal diagnosis, identify the cause, assess semen quality, understand the couple's reproductive goals, correct reversible factors, preserve fertility, integrate appropriate Unani supportive care and use modern hormonal or reproductive treatment when it offers the patient the best opportunity.

The question should never simply be:

“How can we raise testosterone?”

For a man who wishes to become a father, the more important question is:

“How can we improve his hormonal health while protecting—or restoring—his ability to produce sperm?”

That distinction can completely change the outcome of treatment.

Saira Health Care:
https://www.sairahealthcare.com/

Appointment & Consultation:
https://www.sairahealthcare.com/Appointment.aspx

Saira Health Care Pharmacy:
https://pharmacy.sairahealthcare.com/

Medical Disclaimer: This article is intended for medical education and patient awareness and does not replace individual examination or treatment. Male hypogonadism should be diagnosed using symptoms together with appropriately repeated hormonal testing. Men currently trying to conceive or wishing to preserve future fertility should not begin testosterone replacement, anabolic steroids, fertility hormones, supplements, herbal medicines or Unani formulations without professional assessment. Testosterone therapy, gonadotropins, SERMs and aromatase inhibitors have substantially different effects on fertility and should not be used interchangeably. Genetic, pituitary or severe testicular disorders may require specialist endocrinology, andrology/urology, genetics or reproductive-medicine care.