Spermatogenesis: How Sperm Are Produced, Disorders of Sperm Production, Treatment and the Role of Unani Medicine

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: "Doctor, How Are Sperm Actually Made?"

When a man comes to me because his semen report shows:

- low sperm count,
- no sperm,
- poor sperm motility,
- abnormal morphology,
- or another fertility problem,

one of the most useful things I can do is explain what normally happens inside the testis.

Patients often think sperm are produced quickly—almost like semen is made from today's food and appears a few days later.

The biology is much more complex.

Spermatogenesis is the highly organized process by which primitive male germ cells inside the testes develop into spermatozoa.

It involves:

- stem-cell renewal,

- mitotic cell division,
- meiosis,
- major structural transformation,
- DNA packaging,
- interaction with Sertoli cells,
- hormonal regulation,
- and finally release of sperm into the tubules.

After this, sperm undergo additional maturation in the:

epididymis

before acquiring their full motility and fertilizing potential.

A 2025 review describes spermatogenesis as a coordinated sequence of:

- proliferative stem-cell activity,
- meiosis,
- post-meiotic differentiation,
- and subsequent post-testicular maturation.

Understanding this process is extremely important because:

male infertility is not simply “weak sperm.”

The problem may occur at:

- the beginning of sperm production,
- during meiosis,
- during spermiogenesis,
- during hormonal stimulation,
- during epididymal maturation,
- or after perfectly normal sperm have already been produced.

Each situation requires different treatment.

At **Saira Health Care**, I therefore do not ask only:

“How can we increase sperm?”

I ask:

“At which stage of the reproductive process is the problem occurring?”

Spermatogenesis Is Not a Disease

This is the first terminology point I want to clarify.

Spermatogenesis itself is:

a normal physiological process.

It is not a disease.

The medical problems are conditions such as:

- impaired spermatogenesis,
- hypospermatogenesis,
- maturation arrest,
- Sertoli-cell-only syndrome,
- oligozoospermia,
- non-obstructive azoospermia,
- hormonal suppression of sperm production,
- genetic spermatogenic failure.

Therefore, the medically accurate subject is:

Spermatogenesis and Disorders of Sperm Production

rather than simply “treatment of spermatogenesis.”

Where Does Spermatogenesis Occur?

Sperm are produced inside the testes within microscopic coiled structures called:

Seminiferous Tubules

A normal adult testis contains a very large network of these tubules.

Inside them are:

- developing germ cells,
- Sertoli cells,
- specialized junctions,

- a carefully regulated biochemical environment.

Outside and between the seminiferous tubules lie:

Leydig Cells

which produce testosterone.

The entire system functions like an extremely sophisticated biological production unit.

The Important Cells Involved in Sperm Production

1. Spermatogonial Stem Cells

These are the foundation of continuous sperm production.

They have two important abilities:

Self-renewal

Some cells remain as stem cells so the sperm-producing system is not exhausted.

Differentiation

Other cells enter the pathway toward becoming mature sperm.

Modern research considers spermatogonial stem cells essential for lifelong spermatogenesis and potential future fertility-restoration technologies.

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2. Sertoli Cells – The "Nurse Cells" of Spermatogenesis

Sertoli cells are extremely important.

I often explain them to patients as:

the support or nursing cells for developing sperm.

They help:

- nourish developing germ cells,
- organize their movement through the seminiferous epithelium,
- create the blood-testis barrier,
- respond to FSH,
- respond indirectly to testosterone,
- regulate germ-cell survival,

- support meiosis,
- support spermiogenesis,
- remove residual cellular material.

Modern molecular research increasingly recognizes Sertoli cells as central coordinators of the testicular environment rather than passive support cells.

The Blood-Testis Barrier

Adjacent Sertoli cells create specialized tight junctions known collectively as the:

Blood-Testis Barrier

This barrier separates different stages of developing germ cells from the general circulation.

It creates a controlled environment necessary for:

- meiosis,
- germ-cell differentiation,
- protection from inappropriate immune attack.

Disruption of this barrier may impair spermatogenesis.

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3. Leydig Cells

Leydig cells lie between the seminiferous tubules.

Their major reproductive role is:

testosterone production.

They are stimulated by:

Luteinizing Hormone – LH

from the pituitary gland.

Testosterone concentrations inside the testis are essential for normal sperm production.

A 2025 review emphasizes that LH-driven Leydig-cell testosterone production is a central component of spermatogenic regulation.

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4. Peritubular Myoid Cells

These cells surround the seminiferous tubules.

They contribute to:

- structural support,
- the local cellular environment,
- tubular contractility,
- signaling between cells.

Modern research shows that spermatogenesis depends on communication between many cell types, not germ cells alone.

The Main Stages of Spermatogenesis

Sperm production can be understood through several major stages.

Stage 1: Spermatogonial Stem-Cell Renewal and Mitosis

The process begins with:

Spermatogonia

located near the basement membrane of the seminiferous tubule.

Some remain in the stem-cell pool.

Others differentiate and divide by:

mitosis

to produce cells committed to sperm development.

This stage ensures both:

- continuation of sperm production,
- production of sufficient germ cells.

Why Mitosis Matters

Ordinary body cells contain:

46 chromosomes

organized in 23 pairs.

Early male germ cells are also diploid.

Before a sperm can fertilize an egg, the chromosome number must eventually be reduced by half.

But before that happens, the germ-cell population must expand through mitosis.

Stage 2: Formation of Primary Spermatocytes

Differentiating spermatogonia give rise to:

Primary Spermatocytes

These cells enter the critical reproductive division called:

Meiosis

This stage is especially important because many severe forms of male infertility occur when germ cells fail to progress normally through meiosis.

Stage 3: Meiosis

Meiosis is different from ordinary cell division.

Its purpose includes:

- halving the chromosome number,
- recombining genetic material,
- producing genetically unique haploid cells.

There are two major meiotic divisions.

Meiosis I

The primary spermatocyte undergoes an elaborate sequence involving:

- chromosome pairing,
- genetic recombination,
- chromosome separation.

This produces:

secondary spermatocytes.

Meiosis II

Secondary spermatocytes divide again.

The result is:

haploid spermatids

containing 23 chromosomes.

This chromosome reduction is essential.

The egg also carries 23 chromosomes.

When sperm and egg combine:

23 + 23 = 46 chromosomes

in the embryo.

Why Meiosis Is Vulnerable

Meiosis requires extraordinarily accurate:

- chromosome pairing,
- DNA repair,
- recombination,
- separation.

Genetic defects involving these processes can cause:

maturation arrest

in which germ-cell development stops before mature sperm are formed.

This is one reason some men with azoospermia may have:

- early maturation arrest,
- late maturation arrest

on testicular histology.

Stage 4: Spermiogenesis

Once round spermatids are formed, they must undergo a dramatic transformation.

This process is called:

Spermiogenesis

Importantly:

spermiogenesis does not involve another cell division.

Instead, the round cell becomes a highly specialized sperm.

What Happens During Spermiogenesis?

The spermatid develops:

A Head

Containing highly condensed paternal DNA.

An Acrosome

A specialized cap-like structure important in sperm-egg interaction.

A Midpiece

Rich in mitochondria that participate in energy production.

A Tail or Flagellum

Required for movement.

At the same time:

- excess cytoplasm is removed,
- DNA packaging becomes extremely compact,
- the cell becomes streamlined.

A 2025 review describes this post-meiotic phase as involving profound DNA condensation and construction of specialized sperm structures.

Stage 5: Spermiation

At the end of spermiogenesis, mature-looking spermatozoa are released from Sertoli cells into the:

lumen of the seminiferous tubule.

This release process is called:

Spermiation

The sperm have now completed their main intratesticular developmental pathway.

But they are not yet fully mature in the functional sense.

Stage 6: Epididymal Maturation

This is an important point often omitted from simple descriptions.

Sperm leaving the testes are not immediately equivalent to fully functional ejaculated sperm.

They pass into the:

Epididymis

where they undergo additional biochemical and membrane changes.

During epididymal transit, sperm progressively acquire important characteristics including:

- forward motility,
- membrane modifications,
- greater fertilizing competence.

Human epididymal research confirms that acquisition of important motility and fertilization properties occurs during this post-testicular maturation period.

Therefore:

Spermatogenesis and sperm maturation are related but not exactly the same process.

Stage 7: Capacitation Occurs After Ejaculation

Even ejaculated sperm are not immediately capable of complete fertilization.

After entering the female reproductive tract, sperm undergo:

Capacitation

which involves further functional and membrane changes.

Later they develop vigorous:

hyperactivated motility

important for successful interaction with the egg.

Therefore sperm development is a biological journey extending from:

testicular stem cell → mature sperm → epididymal maturation → capacitation.

How Long Does Human Spermatogenesis Take?

Patients frequently ask:

“If I start treatment today, when will new sperm be produced?”

Human spermatogenesis takes much longer than a few days.

Historically, the complete intratesticular process was estimated at around:

64 days

but more modern analyses suggest a duration closer to approximately:

74 days

in humans.

Additional time is required for:

- epididymal maturation,
- transport.

Therefore, when treatment aims to improve sperm production, I generally explain:

Do not judge the biological effect after one or two weeks.

A meaningful semen reassessment often requires:

several months.

This is also why an illness or fever today may influence semen findings weeks later.

Is Sperm Production Continuous?

Yes.

After puberty, spermatogenesis normally continues continuously rather than all sperm starting and finishing at exactly the same time.

Different regions of seminiferous tubules contain germ cells at different developmental stages.

This arrangement allows ongoing production of sperm.

Hormonal Control of Spermatogenesis

Normal sperm production depends heavily on the:

Hypothalamic-Pituitary-Gonadal Axis

or:

HPG Axis

The pathway can be simplified as:

Hypothalamus

↓
GnRH
↓
Pituitary
↓
FSH + LH
↓
Testis

GnRH

The hypothalamus releases:

Gonadotropin-Releasing Hormone – GnRH

in pulses.

GnRH stimulates the pituitary gland.

FSH – Follicle-Stimulating Hormone

The pituitary releases:

FSH

In men, FSH acts mainly on:

Sertoli Cells

and supports:

- Sertoli-cell function,
- germ-cell proliferation,
- differentiation,
- sperm development.

Current EAU guidance summarizes that FSH promotes:

spermatogenesis and testicular growth.

LH – Luteinizing Hormone

LH acts mainly on:

Leydig Cells

stimulating production of:

testosterone.

Testosterone then acts locally within the testis and is indispensable for normal spermatogenesis.

Modern hormonal reviews emphasize the coordinated actions of:

- FSH on Sertoli cells,
- LH-driven testosterone from Leydig cells.

Why Intratesticular Testosterone Matters

The testosterone concentration required inside the testis for spermatogenesis is much higher than the testosterone level measured in ordinary blood.

This explains a seemingly paradoxical fact:

Taking testosterone from outside the body can reduce sperm production rather than improve it.

The Testosterone Paradox

A man may say:

“Doctor, testosterone is required for sperm, so why can't I take testosterone injections to make more sperm?”

Because external testosterone suppresses the brain's natural hormonal signals.

High external testosterone tells the hypothalamus and pituitary:

“There is enough testosterone.”

The body then reduces:

- GnRH,
- LH,

- FSH.

The testes receive less LH and FSH stimulation.

Intratesticular testosterone falls.

The result can be:

- severe oligozoospermia,
- or azoospermia.

Current AUA/ASRM guidance states:

Testosterone monotherapy should not be prescribed to men interested in current or future fertility.

Current EAU guidance similarly gives a strong recommendation:

Do not use testosterone therapy for treatment of male infertility.

This is one of the most important messages in modern male-fertility care.

Inhibin B

Sertoli cells also produce:

Inhibin B

which helps regulate FSH through negative feedback.

Inhibin B can provide information about Sertoli-cell and spermatogenic function in selected clinical or research contexts.

However, it is not a stand-alone fertility test.

Why Testicular Temperature Matters

Normal spermatogenesis functions best at a testicular temperature below core body temperature.

A 2025 review describes optimal testicular temperature as approximately:

2–4°C below core body temperature

and explains how excessive heat can contribute to:

- oxidative stress,
- germ-cell apoptosis,

- blood-testis-barrier disruption,
- impaired differentiation.

This helps explain why conditions such as:

- varicocele,
- repeated excessive heat exposure

can influence sperm production in susceptible men.

What Is Impaired Spermatogenesis?

Impaired spermatogenesis means that the testes are not producing mature sperm normally.

The problem may involve:

- reduced quantity,
- developmental arrest,
- absence of germ cells,
- poor maturation,
- severe focal failure.

Possible semen findings include:

- oligozoospermia,
- severe oligozoospermia,
- azoospermia,
- combined sperm abnormalities.

Important Histological Patterns of Spermatogenic Failure

When testicular tissue is examined microscopically in severe male infertility, several patterns may be seen.

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1. Hypospermatogenesis

In:

Hypospermatogenesis

all major stages of germ-cell development may be present, but sperm production is quantitatively reduced.

This often has a relatively better prognosis for sperm retrieval than some more severe histological patterns.

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2. Early Maturation Arrest

In:

Early Maturation Arrest

development stops relatively early, often around the:

- spermatogonial,
- or primary spermatocyte stage.

Mature sperm may be absent in affected tubules.

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3. Late Maturation Arrest

In:

Late Maturation Arrest

germ cells progress farther, often reaching:

- spermatids,

but fail to complete normal maturation into spermatozoa.

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4. Sertoli-Cell-Only Syndrome

In this pattern:

seminiferous tubules contain Sertoli cells but lack germ cells in the sampled areas.

This represents severe spermatogenic failure.

However, in non-obstructive azoospermia, sperm production can sometimes be:

focal

meaning small isolated areas elsewhere in the testis may still produce sperm.

This is one reason micro-TESE can occasionally retrieve sperm despite severe histology.

Normal FSH Does Not Guarantee Normal Spermatogenesis

This is an important clinical fact.

FSH often rises when germ-cell production is severely impaired.

However, current EAU guidance notes that when spermatogonia remain present but maturation arrest occurs at the:

- spermatocyte,
- or spermatid

stage, FSH may remain within the normal range.

Therefore:

A normal FSH does not rule out severe spermatogenic failure.

Similarly:

a high FSH does not prove that no sperm exist anywhere in the testes.

Men with non-obstructive azoospermia and high FSH may still have focal spermatogenesis detectable during micro-TESE.

Causes of Impaired Spermatogenesis

There are many possible causes.

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1. Genetic and Chromosomal Abnormalities

Spermatogenesis depends on hundreds of genes controlling:

- stem-cell maintenance,
- meiosis,
- chromosome pairing,
- DNA repair,
- flagellar development,
- germ-cell survival.

Genetic causes include:

- Klinefelter syndrome,
- Y-chromosome microdeletions,

- chromosomal translocations,
- specific gene mutations.

Modern genetic research continues to identify previously unexplained causes of spermatogenic failure.

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2. Y-Chromosome Microdeletions

Important Y-chromosome regions include:

- AZFa,
- AZFb,
- AZFc.

Complete AZFa and AZFb deletions have especially poor sperm-retrieval prognosis.

Current EAU guidance strongly recommends against sperm-retrieval surgery in complete AZFa and AZFb deletions because retrieval probability is essentially zero.

This shows why:

genetic diagnosis can prevent unnecessary treatment and surgery.

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3. Hormonal Deficiency

A particularly important treatable cause is:

Hypogonadotropic Hypogonadism

Here the testes may have the capacity to produce sperm, but they are not receiving adequate:

- LH,
- FSH

stimulation.

This is fundamentally different from primary testicular failure.

Treatment of Hypogonadotropic Hypogonadism

Current EAU guidance strongly recommends inducing spermatogenesis in men with congenital or acquired hypogonadotropic hypogonadism using fertility-directed therapies such as:

- hCG,
- hMG,
- recombinant FSH,
- highly purified FSH.

These men may respond significantly because the underlying problem is:

lack of hormonal stimulation

rather than complete loss of sperm-producing capacity.

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4. Hyperprolactinemia

High prolactin can suppress gonadotropin function and impair fertility.

When genuine hyperprolactinemia exists, treatment is directed at:

- the cause,
- and may involve dopamine agonists.

Current EAU guidance recommends appropriate treatment for proven hyperprolactinemia.

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5. Exogenous Testosterone and Anabolic Steroids

This has become a major fertility issue.

Men may use:

- bodybuilding steroids,
- testosterone injections,
- testosterone gels,
- anabolic-androgenic substances.

These may suppress spermatogenesis.

Current EAU guidance recommends withdrawal of anabolic steroids in infertile men and notes that sperm production often requires:

6–12 months

to improve after stopping, although recovery varies.

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6. Undescended Testes – Cryptorchidism

Normal testicular descent is important for:

- germ-cell development,
- appropriate temperature.

A history of:

- unilateral,
- or bilateral cryptorchidism

increases the risk of impaired adult fertility.

The risk is generally greater with:

- bilateral disease,
- delayed correction,
- abnormal testicular development.

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7. Varicocele

Varicocele is enlargement of veins around the testis.

Possible mechanisms impairing spermatogenesis include:

- increased temperature,
- oxidative stress,
- altered circulation,
- metabolic changes.

A clinical varicocele may be relevant in selected infertile men, but:

not every ultrasound-detected varicocele requires surgery.

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8. Testicular Torsion and Trauma

Severe torsion can interrupt blood supply to the testis.

Significant trauma can damage:

- germ cells,
- seminiferous tubules,
- circulation.

The extent of later fertility impairment depends on the severity and whether one or both testes are affected.

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9. Testicular Infection and Orchitis

Conditions such as:

- mumps orchitis,
- severe bacterial infection,
- epididymo-orchitis

may damage the sperm-producing tissue.

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10. Chemotherapy and Radiation

Cancer therapy may damage rapidly dividing germ cells.

The effect may be:

- temporary,
- prolonged,
- permanent.

Whenever possible, fertility preservation such as:

sperm cryopreservation

should be discussed before gonadotoxic treatment.

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11. Testicular Tumors and Surgery

Cancer itself and its treatment may impair:

- sperm production,
- testosterone function,
- fertility.

Men facing reproductive-organ treatment should receive fertility counselling when appropriate.

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12. Excessive Heat

Repeated or prolonged excessive testicular temperature may adversely affect:

- germ-cell development,
- sperm concentration,
- sperm morphology,
- motility.

Patients do not need extreme cooling measures.

Reasonable reduction of avoidable chronic heat exposure is sufficient.

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13. Smoking

Smoking is associated with:

- oxidative stress,
- sperm DNA injury,
- poorer semen quality.

WHO's first global infertility guideline, published in November 2025, specifically includes:

tobacco cessation

among recommended fertility-promoting lifestyle measures.

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14. Obesity, Diabetes and Metabolic Disease

Metabolic disease may influence:

- testosterone,
- inflammation,
- oxidative stress,
- sexual function,
- reproductive health.

Healthy metabolic management supports fertility but cannot correct every genetic or structural testicular disorder.

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15. Environmental Toxins

Potential exposures include:

- pesticides,
- heavy metals,
- solvents,
- industrial chemicals,
- ionizing radiation.

Environmental factors may contribute to impaired sperm production, although proving one specific exposure caused infertility can be difficult.

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16. Idiopathic Spermatogenic Failure

Sometimes extensive evaluation finds:

- no major genetic cause,
- no hormonal disorder,
- no obvious testicular disease,
- no major environmental cause,

yet sperm production remains abnormal.

This is often described as:

Idiopathic Male Infertility

or idiopathic spermatogenic dysfunction.

Current EAU guidance notes that historically **30–40%** of men with abnormal sperm parameters have had no identifiable male-associated cause using routine evaluation.

Sperm Production vs Sperm Transport: An Essential Difference

A man can have:

perfect spermatogenesis

and still have:

zero sperm in the semen.

How?

Because the sperm may be blocked.

This is:

Obstructive Azoospermia

Possible causes include:

- vasectomy,
- congenital absence of vas deferens,
- epididymal obstruction,
- infection,
- ejaculatory-duct obstruction.

Therefore:

Azoospermia does not automatically mean spermatogenesis has stopped.

This distinction is essential before treatment.

How Do We Evaluate Spermatogenesis Clinically?

We cannot watch sperm production directly without testicular tissue.

Instead, physicians infer its function through several methods.

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1. Semen Analysis

This is the fundamental laboratory assessment.

The WHO sixth-edition semen manual remains the international standard for semen examination.

Current lower fifth-percentile reference values include approximately:

Parameter	WHO 2021 lower reference
Semen volume	**1.4 mL**

Total sperm number	**39 million/ejaculate**
Sperm concentration	**16 million/mL**
Total motility	**42%**
Progressive motility	**30%**
Vitality	**54% live**
Normal morphology	**4%**

These values are:

reference distributions—not strict fertility boundaries.

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2. Repeat Abnormal Semen Testing

Semen varies biologically.

A temporary:

- fever,
- illness,
- medication,
- collection issue

can alter results.

Current EAU guidance states that if semen analysis is abnormal on at least two tests, further andrological investigation is indicated.

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3. Hormone Testing

Depending on the clinical picture, tests may include:

- FSH,
- LH,
- morning testosterone,
- prolactin.

These help distinguish:

- hormonal under-stimulation,
- from primary testicular failure.

How Do We Interpret FSH?

High FSH

Often suggests that the pituitary is trying harder to stimulate poorly functioning testes.

Low FSH + Low LH

May suggest:

- hypothalamic,
- or pituitary deficiency.

Normal FSH

Does not prove normal spermatogenesis because maturation arrest can exist despite normal FSH.

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4. Testicular Examination

I assess:

- testicular volume,
- consistency,
- varicocele,
- epididymis,
- vas deferens,
- secondary sexual characteristics.

Small firm testes may suggest certain forms of primary testicular failure.

Normal-sized testes do not guarantee normal sperm production.

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5. Genetic Testing

Genetic evaluation becomes especially important in:

- azoospermia,
- severe oligozoospermia,
- testicular atrophy,
- evidence of primary sperm-production failure.

Depending on the case, testing may include:

- karyotype,
- Y-chromosome microdeletion testing,
- selected additional genetic testing.

The AUA/ASRM guideline was amended in 2024 to refine these genetic-testing indications.

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6. Scrotal Ultrasound

Ultrasound may help evaluate:

- testicular size,
- masses,
- structural abnormalities,
- varicocele.

But:

ultrasound cannot directly tell us whether meiosis is occurring normally.

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7. Testicular Histology

Microscopic examination of testicular tissue can reveal patterns such as:

- hypospermatogenesis,
- maturation arrest,
- Sertoli-cell-only pattern.

However, current practice does not recommend routine diagnostic biopsy simply to evaluate every infertile man.

The test should be used only when clinically appropriate.

Can Spermatogenesis Be "Boosted"?

This is one of the most common questions patients ask.

The answer depends entirely on:

why sperm production is reduced.

There is no universal treatment that increases spermatogenesis in every man.

When Spermatogenesis Can Be Restored Medically

One of the best examples is:

Hypogonadotropic Hypogonadism

If a man lacks appropriate FSH/LH stimulation but testicular tissue remains responsive, fertility-directed hormonal therapy can induce sperm production.

Current EAU guidance gives a strong recommendation for gonadotropin treatment in this condition.

When Hormones Are Less Useful

In:

classical primary testicular failure

current EAU guidance states there is no substantial evidence that gonadotropin therapy improves spermatogenesis.

This distinction is critical.

Giving FSH simply because sperm count is low is not the same as replacing a genuine FSH deficiency.

FSH in Idiopathic Oligozoospermia

Current EAU guidance allows FSH treatment in selected men with:

- idiopathic oligozoospermia,
- normal-range FSH

but rates this as a:

weak recommendation.

It also advises against routine high-dose FSH treatment.

Therefore this is a selective specialist option, not universal treatment.

Hormonal Treatment Before Micro-TESE

Some men with non-obstructive azoospermia are given:

- hCG,
- FSH,
- clomiphene,
- aromatase inhibitors

before sperm retrieval.

However, current EAU guidance states:

Do not routinely start hormonal stimulation before testicular sperm extraction in NOA outside clinical trials.

Evidence remains insufficient to promise improved sperm retrieval.

Lifestyle Measures That Support Spermatogenesis

The WHO's November 2025 infertility guideline recommends fertility-promoting lifestyle measures such as:

- healthy diet,
- physical activity,
- tobacco cessation.

For men, I commonly advise:

- stop smoking,
- avoid anabolic steroids,
- avoid recreational drugs,
- reduce heavy alcohol,
- maintain healthy metabolic status,
- treat diabetes,
- sleep adequately,

- exercise appropriately,
- avoid excessive chronic testicular heat,
- eat a balanced nutritious diet.

These measures improve the:

environment in which spermatogenesis occurs.

But they do not repair every genetic or structural defect.

Antioxidants and Fertility Supplements

A large fertility-supplement industry promises to:

- boost sperm production,
- increase motility,
- repair DNA,
- improve fertility.

Some studies report improvement in semen parameters with specific agents.

However, current AUA/ASRM guidance states that the clinical utility of supplements such as:

- antioxidants,
- vitamins

is questionable and that evidence is insufficient to recommend specific universal agents.

Therefore:

supplements should never replace diagnosis.

Treatment of Varicocele

A clinically significant varicocele may adversely affect spermatogenesis in selected men.

Appropriate treatment may include:

- microsurgical varicocelectomy,
- radiological embolization.

But treatment is directed to selected patients based on:

- physical examination,

- semen findings,
- infertility,
- couple factors.

Not every ultrasound-detected vein needs repair.

Treatment of Obstruction

If spermatogenesis is normal but sperm are blocked, treatment should target the obstruction.

Options can include:

- microsurgical reconstruction,
- epididymal sperm retrieval,
- testicular sperm retrieval,
- IVF/ICSI.

Giving sperm-production medicine alone cannot open a completely blocked vas deferens.

Micro-TESE in Non-Obstructive Azoospermia

If sperm production is severely impaired and no sperm appear in the semen, some men may still have:

small focal areas of spermatogenesis.

For selected men with NOA, current EAU guidance recommends:

microdissection Testicular Sperm Extraction – micro-TESE

as the treatment of choice for sperm retrieval.

Under an operating microscope, the surgeon searches for seminiferous tubules most likely to contain sperm.

Why High FSH Does Not Automatically Rule Out Micro-TESE

Current EAU guidance specifically notes that men with:

- NOA,
- high FSH

may still contain focal areas of spermatogenesis.

Preoperative hormone values do not reliably guarantee the result of sperm retrieval.

Therefore, I do not tell a patient:

“Your FSH is high, so there is definitely no sperm.”

That statement is too absolute.

IVF and ICSI

If usable sperm are available but natural conception is unlikely, fertility options may include:

IVF

or:

ICSI

During ICSI:

- one viable sperm is injected directly into an egg.

This has transformed fertility treatment for men with:

- severe oligozoospermia,
- poor motility,
- poor morphology,
- surgically retrieved sperm.

Sperm Freezing

Cryopreservation is important in several situations.

It may be considered:

- before chemotherapy,
- before radiotherapy,
- when sperm numbers are dangerously low,
- when rare sperm appear intermittently,
- after successful surgical sperm retrieval.

Preserving available sperm can prevent loss of future reproductive options.

Unani Understanding of Male Reproductive Function

The Unani system has historically placed considerable emphasis on:

- reproductive strength,
- semen production,
- nutrition,
- general vitality,
- temperament,
- digestion,
- lifestyle.

Classical concepts include:

- **Mizaj**
- **Akhlat**
- reproductive-organ function,
- general body strength.

However, a scientifically responsible distinction is important.

Classical Unani scholars did not have:

- electron microscopy,
- semen counting chambers,
- FSH assays,
- chromosome testing,
- testicular histopathology.

Therefore:

classical Unani concepts should not be described as if they identified spermatogonia, meiosis, spermatids or genetic maturation arrest exactly as modern histology does.

Qillat-i-Mani in Unani Medicine

CCRUM's Standard Unani Treatment Guidelines discuss:

Qillat-i-Mani

in the context of reduced seminal production and male reproductive weakness.

Traditional causes described include:

- general debility,
- inadequate nutrition,
- altered functional state of semen-producing organs.

Traditional treatment principles include:

- nutritional support,
- improving digestion,
- individualized treatment according to the patient's condition.

However:

Qillat-i-Mani is not automatically identical to every modern case of impaired spermatogenesis.

Traditional Unani Therapeutic Modalities

CCRUM identifies four broad treatment modes:

Ilaj-bil-Ghiza

Dietotherapy

Ilaj-bit-Tadbir

Regimenal Therapy

Ilaj-bil-Dawa

Pharmacotherapy

Ilaj-bil-Yad

Surgery

This is extremely relevant in infertility care.

Authentic Unani medicine does not require us to reject:

- surgery,
- ART,

- sperm retrieval

when these are appropriate.

How Unani Medicine Can Support Spermatogenesis

I consider the role of Unani treatment strongest in creating a healthier biological environment for sperm production.

This may involve:

- individualized nutrition,
- lifestyle modification,
- improving general health,
- correcting unhealthy habits,
- treating associated weakness,
- supervised traditional pharmacotherapy.

It may be particularly useful as supportive treatment in selected men with:

- idiopathic oligozoospermia,
- mild or moderate semen abnormalities,
- poor general health,
- nutritional deficiency,
- metabolic problems,
- lifestyle-related reproductive stress.

Mizaj and Individualization

In the Unani system:

Mizaj

provides a traditional framework for individualized treatment.

In my clinical practice, I do not use Mizaj instead of semen testing.

I use it alongside:

- medical history,

- semen analysis,
- hormone findings,
- physical examination,
- fertility diagnosis.

This allows us to consider:

- constitution,
- digestion,
- appetite,
- sleep,
- physical activity,
- general weakness,
- reproductive health.

The important principle is:

Two men with the same sperm count may not have the same disease.

Ilaj-bil-Ghiza for Spermatogenic Support

Diet cannot change:

- a complete AZFa deletion,
- absent germ cells,
- a completely blocked vas.

But appropriate nutrition can support:

- protein availability,
- micronutrient status,
- metabolic health,
- normal hormone function,
- overall testicular health.

I commonly recommend a diet containing:

- vegetables,

- fruits,
- pulses,
- whole grains,
- nuts,
- seeds,
- adequate protein,
- appropriate healthy fats.

I advise reducing:

- smoking,
- excessive alcohol,
- sugary drinks,
- heavily processed foods,
- repeated deep-fried foods.

Ilaj-bit-Tadbir

Regimenal therapy may include individualized attention to:

- physical activity,
- adequate rest,
- good sleep,
- stress management,
- weight control,
- reducing excessive testicular heat.

These measures are supportive rather than guaranteed spermatogenic treatments.

Ilaj-bid-Dawa and Male-Fertility Formulations

Traditional medicines may be selected according to:

- the cause,
- semen findings,

- Mizaj,
- general condition,
- associated sexual or reproductive symptoms.

At Saira Health Care, individualized male-fertility programmes may include selected traditional formulations such as:

- Dr. Qasmi's Spermogenic,
- Dr. Qasmi's Nuskha No. 129,

where clinically appropriate.

Dr. Qasmi's Nuskha No. 129 – Vitasem Max

The current Saira Health Care Pharmacy listing describes **Dr. Qasmi's Nuskha No. 129 – Vitasem Max** as a:

Unani preparation

for male health, general weakness and semen-quality support.

The published ingredient list includes several traditional herbal and mineral ingredients.

The pharmacy appropriately warns that:

- self-medication is not advised,
- overdose may cause adverse effects,
- the preparation should be used for a limited period under physician direction.

This is especially important for formulations containing mineral preparations.

Spermogenic

Another Dr. Qasmi formulation used within Saira Health Care's male-fertility practice is:

Spermogenic

which is used for several semen-related reproductive concerns.

In an individualized programme, the intention is to support:

- male reproductive health,
- semen parameters,
- general spermatogenic function

where appropriate.

However:

I do not identify a high-quality independent randomized trial proving that Spermogenic restores normal spermatogenesis in every cause of testicular failure.

It should therefore be presented as:

individualized traditional fertility-support therapy

rather than as a universal biological cure.

An Important Product-Classification Correction

The supplied material describes:

- Semen Gold Plus,
- Spermzoa,
- Sperm Plus

as Unani medicines.

Current Saira Health Care Pharmacy listings describe them differently.

Semen Gold Plus Capsule

The current Saira Health Care Pharmacy page describes:

Semen Gold Plus as an Ayurvedic supplement

and lists its brand as:

Himcure Pharmaceuticals Pvt. Ltd.

Therefore it should not be described on the website as a classical Unani formulation.

Spermzoa Capsule

The current pharmacy page explicitly describes:

Spermzoa Capsule as an Ayurvedic formulation

from:

Swastic Ayurveda.

It is marketed for:

- sperm count,
- motility,
- male reproductive support.

It can be part of an integrative treatment plan if clinically selected, but should be classified accurately.

Sperm Plus Capsule

The current Saira Health Care Pharmacy page describes:

Sperm Plus as an Ayurvedic herbal product

focused mainly on:

- sexual vitality,
- stamina,
- libido,
- reproductive support.

Therefore, it should also not be called a classical Unani medicine.

Why Correct Product Classification Matters

For a professional medical website:

Unani, Ayurvedic, herbal and modern medical therapies should be identified accurately.

Patients deserve to know:

- which system the medicine belongs to,
- what its intended role is,
- what evidence supports it,
- what it cannot guarantee.

A clinic can use an:

integrative programme

without mislabelling every traditional product as Unani.

What Does Published Unani Research Show About Male Fertility?

There is some clinical research supporting further investigation of Unani fertility formulations.

CCRUM Observational Study

A CCRUM-associated observational study at the National Institute of Unani Medicine evaluated:

30 infertile men with oligospermia

using the compound Unani formulation:

Sufoof-e-Muallif

for 60 days.

The study assessed:

- semen parameters,
- testosterone,
- FSH,
- LH,
- general laboratory safety measures.

This is encouraging as preliminary clinical research.

However, it was:

- open-label,
- uncontrolled,
- small.

It cannot establish a universal treatment effect.

126-Patient Retrospective Study

A CCRUM-published comparative retrospective analysis reviewed:

126 patients with idiopathic oligospermia

across four Unani treatment groups.

The authors reported varying improvements in:

- semen volume,
- sperm count,
- motility

depending on the treatment group.

Again, this supports continued scientific study of Unani male-fertility therapy.

But:

oligospermia research is not proof that every maturation arrest, genetic azoospermia or Sertoli-cell-only condition can be reversed by Unani medicine.

Why Scientific Precision Strengthens Unani Medicine

I believe Unani medicine is best represented when we say clearly:

What traditional care may support

- nutrition,
- general reproductive health,
- lifestyle,
- selected semen abnormalities.

What modern diagnosis must identify

- genetic defects,
- hormonal deficiency,
- obstruction,
- testicular failure,
- maturation arrest.

What modern procedures may be necessary for

- varicocele,
- obstruction,
- sperm retrieval,
- IVF/ICSI.

This is much stronger than promising that every sperm-production problem can be cured with one herbal formulation.

Dr. Nizamuddin Qasmi's Individualized Approach to Impaired Spermatogenesis

When a patient comes to Saira Health Care asking:

"Doctor, how can I increase sperm production?"

I prefer a structured pathway.

Step 1: Confirm Whether Sperm Production Is Actually Low

I begin with standardized semen analysis.

I ask:

- Is sperm concentration low?
- Is total sperm number low?
- Is sperm completely absent?
- Is only motility abnormal?
- Is morphology the main problem?

Poor motility is not necessarily poor spermatogenesis.

Step 2: Repeat Abnormal Semen Testing

One report may be influenced by:

- fever,
- illness,
- collection problems,
- biological variation.

Persistent abnormalities deserve further evaluation.

Step 3: Take a Detailed Reproductive History

I ask about:

- previous pregnancies,
- duration of infertility,
- childhood undescended testes,
- mumps,
- surgery,
- testicular injury,
- chemotherapy,
- radiation,
- testosterone,
- steroids,
- medications,
- smoking,
- occupational exposure.

Step 4: Examine the Testes and Reproductive Tract

I assess:

- testicular size,
- consistency,
- epididymis,
- vas deferens,
- varicocele,
- secondary sexual characteristics.

This often provides information that semen analysis cannot.

Step 5: Evaluate Hormones

Depending on the case, I assess:

- FSH,
- LH,

- morning testosterone,
- prolactin.

This helps identify:

- under-stimulation,
- primary testicular failure,
- other endocrine disorders.

Step 6: Do Not Mistake High FSH for "No Hope"

High FSH suggests testicular stress or failure in many patients.

But it does not prove:

zero focal spermatogenesis.

Current EAU guidance explicitly cautions that FSH does not accurately predict sperm retrieval in every man.

Step 7: Consider Genetics When Indicated

In:

- azoospermia,
- severe oligozoospermia,
- testicular failure,

I consider appropriate:

- karyotype,
- Y-microdeletion testing,
- genetic counselling.

This step can prevent:

- useless empirical treatment,
- unnecessary surgery.

Step 8: Identify Reversible Causes

I look for:

- hormonal deficiency,
- anabolic steroids,
- clinical varicocele,
- infection,
- metabolic disease,
- harmful lifestyle factors.

Step 9: Stop Fertility-Suppressing Testosterone

If a patient is using:

- testosterone,
- anabolic steroids

and wants fertility, this must be addressed.

External testosterone should not be used as fertility treatment.

Step 10: Improve the Reproductive Environment

I advise:

- stop smoking,
- healthy diet,
- physical activity,
- good sleep,
- reasonable weight,
- diabetes control,
- avoiding unnecessary heat,
- avoiding harmful drugs.

Step 11: Use Unani Treatment Individually

Where appropriate, I may use:

- Ilaj-bil-Ghiza,
- Ilaj-bit-Tadbir,
- selected Ilaj-bid-Dawa,
- Spermogenic,
- Nuskha No. 129

according to:

- semen profile,
- diagnosis,
- overall health,
- Mizaj.

Step 12: Monitor With Objective Testing

I do not consider treatment successful merely because the patient says:

“My semen looks thicker.”

Improvement should be assessed through:

- sperm concentration,
- total sperm number,
- motility,
- morphology,
- hormones where relevant,
- pregnancy outcomes.

Step 13: Preserve Rare Sperm

If rare sperm are detected in a patient previously considered azoospermic or severely oligospermic:

cryopreservation should be discussed promptly.

Severe sperm production can fluctuate.

Step 14: Refer for Micro-TESE When Appropriate

For persistent NOA in selected men seeking biological fatherhood, current EAU guidance recommends:

micro-TESE

as the sperm-retrieval technique of choice.

Step 15: Do Not Delay IVF/ICSI When Time Matters

If the female partner has:

- advanced reproductive age,
- low ovarian reserve,
- tubal disease,

it may be harmful to spend years waiting for a perfect sperm count.

The objective is:

successful family building for the couple

rather than merely improving a laboratory report.

How Long Should Treatment Be Tried?

There is no universal answer.

Because spermatogenesis takes roughly:

two to three months

before newly developing sperm reach the later stages, most interventions aimed at sperm production should not be judged after only a few days.

However, treatment duration should depend on:

- diagnosis,
- female partner's age,
- severity,
- ART planning.

A fixed:

“six months for everyone”

or:

“eight months for everyone”

is not medically appropriate.

What If Spermatogenesis Cannot Be Restored?

This is an important and sometimes difficult conversation.

Some causes may not currently be reversible.

Examples include:

- complete AZFa deletion,
- complete AZFb deletion,
- severe germ-cell loss.

In such situations, the goal changes from endlessly trying to “boost” spermatogenesis to discussing realistic options.

These may include:

- micro-TESE when biologically appropriate,
- donor sperm,
- adoption,
- other family-building choices.

Future of Spermatogenesis Treatment

Research is advancing quickly.

Several areas are being investigated.

Spermatogonial Stem-Cell Culture

Scientists are trying to:

- isolate,
- expand,
- preserve,

- differentiate

human spermatogonial stem cells.

A 2025 review concludes that current culture systems still cannot reliably recreate the complex human testicular niche sufficiently for routine clinical fertility restoration.

In-Vitro Spermatogenesis

The long-term objective is to produce functional sperm outside the human body from:

- immature germ cells,
- testicular tissue,
- stem-cell populations.

Modern experimental systems include:

- organ culture,
- 3D organoids,
- testis-on-chip models,
- engineered tissue platforms.

However, a 2025 review notes that generation of fully functional mature human sperm in vitro has **not yet become an established clinically validated treatment**.

Stem-Cell Therapy

Stem-cell research is promising, and early experimental human studies exist.

However:

stem-cell injection into the testes is not established standard treatment for male infertility in 2026.

A 2025 Fertility and Sterility review emphasizes that although stem-cell approaches have shown important progress—particularly in animal models—substantial:

- scientific,
- ethical,
- regulatory

barriers remain before routine clinical application.

Therefore patients should be cautious of advertisements claiming:

“Guaranteed sperm production with stem cells.”

Gene-Based Future Treatments

As more genetic causes of:

- meiotic failure,
- maturation arrest,
- flagellar defects

are discovered, future treatment may become increasingly precise.

However, correcting infertility-causing germline mutations raises major:

- safety,
- ethical,
- inheritance

questions.

This remains research rather than routine treatment.

Common Myths About Spermatogenesis

Myth 1: Sperm are made in a few days.

Fact: Human spermatogenesis requires approximately several months from early germ-cell development to clinically assessable new sperm.

Myth 2: Semen and sperm are the same thing.

Fact: Semen is the fluid ejaculate; sperm are cells contained within it.

Myth 3: Thick semen means spermatogenesis is excellent.

Fact: Semen appearance cannot determine sperm concentration or testicular histology.

Myth 4: Low motility means the testes are not producing sperm.

Fact: Sperm may be produced normally but fail to acquire normal motility.

Myth 5: Azoospermia always means sperm production has completely stopped.

Fact: Obstructive azoospermia may occur despite normal spermatogenesis.

Myth 6: High FSH means there is definitely no sperm in the testes.

Fact: Men with high FSH and NOA may still have focal sperm production.

Myth 7: Normal FSH proves normal sperm production.

Fact: Maturation arrest may occur with normal FSH.

Myth 8: Testosterone injections increase spermatogenesis.

Fact: External testosterone can suppress FSH/LH and cause severe oligozoospermia or azoospermia.

Myth 9: Every infertile man should receive FSH injections.

Fact: FSH treatment is strongly established for selected hormonal deficiency and only weakly recommended in selected idiopathic oligozoospermia.

Myth 10: Every supplement scientifically increases sperm production.

Fact: Current AUA/ASRM guidance considers the clinical usefulness of most antioxidant/vitamin supplements uncertain.

Myth 11: One Unani formulation can reverse every form of spermatogenic failure.

Fact: Genetic, hormonal, structural and testicular causes require different approaches.

Myth 12: Semen Gold Plus, Spermzoa and Sperm Plus are classical Unani medicines.

Fact: Current Saira Health Care Pharmacy listings classify these products as Ayurvedic.

Myth 13: Stem-cell treatment can routinely make sperm in humans today.

Fact: Stem-cell and in-vitro spermatogenesis remain experimental fields.

Frequently Asked Questions

What is spermatogenesis?

It is the biological process in which male germ cells inside the testes develop into spermatozoa.

Where does spermatogenesis occur?

Primarily in the:

seminiferous tubules of the testes.

What are the main stages?

In simplified form:

Spermatogonial stem cells

↓

Spermatogonia

↓

Primary spermatocytes

↓

Secondary spermatocytes

↓

Spermatids

↓

Spermatozoa

followed by:

epididymal maturation.

What are the three classical processes?

They are:

- mitosis,
- meiosis,
- spermiogenesis.

Spermiation then releases sperm into the tubular lumen.

How long does spermatogenesis take?

Modern literature commonly estimates the intratesticular process at roughly:

74 days

although historical estimates and methodology vary.

Additional maturation occurs in the epididymis.

What hormone makes sperm?

No single hormone does this alone.

Spermatogenesis requires coordinated action of:

- FSH,
- LH,
- intratesticular testosterone,
- Sertoli cells,
- Leydig cells.

What does FSH do?

FSH acts mainly on Sertoli cells and supports sperm development.

What does LH do?

LH stimulates Leydig cells to produce testosterone.

Does high testosterone mean high sperm count?

No.

External testosterone can actually suppress sperm production.

Can testosterone injections cause azoospermia?

Yes.

This is a well-established fertility risk.

What is hypospermatogenesis?

It means all stages of sperm development may be present but overall sperm production is reduced.

What is maturation arrest?

It means germ-cell development stops before mature sperm production is completed.

It can be:

- early,
- or late.

What is Sertoli-cell-only syndrome?

It is a severe histological pattern in which sampled seminiferous tubules contain Sertoli cells but no germ cells.

Can high FSH be reduced to restore sperm?

The goal is not simply to lower the laboratory FSH number.

High FSH often represents the body's response to testicular impairment.

Treatment should address the underlying condition.

Can FSH injections improve sperm?

They are clearly useful in appropriately diagnosed hypogonadotropic hypogonadism.

They may also be considered selectively in idiopathic oligozoospermia, but the evidence is weaker.

Can Unani medicine improve spermatogenesis?

Unani medicine may contribute through:

- individualized nutrition,
- regimenal care,
- lifestyle correction,
- reproductive-health pharmacotherapy.

Published CCRUM studies in oligospermia have reported improvements in semen parameters, but evidence remains limited and does not prove reversal of every form of severe spermatogenic failure.

Is Nuskha No. 129 a Unani medicine?

Yes.

The current Saira Health Care Pharmacy describes **Dr. Qasmi's Nuskha No. 129 – Vitasem Max** as a Unani preparation for male reproductive and general vitality support.

Is Semen Gold Plus Unani?

The current pharmacy page classifies it as:

Ayurvedic.

Is Spermzoa Unani?

The current pharmacy page classifies it as:

Ayurvedic.

Is Sperm Plus Unani?

The current Saira pharmacy description presents it as an:

Ayurvedic herbal formulation.

Can micro-TESE find sperm when semen shows zero sperm?

Yes, in selected men with non-obstructive azoospermia.

Current EAU guidance recommends micro-TESE as the preferred retrieval procedure in appropriate NOA patients.

Can micro-TESE work in complete AZFa or AZFb deletion?

Current EAU guidance advises against sperm retrieval because the chance is essentially zero.

Can diet cure genetic infertility?

No.

Diet supports health but cannot correct a complete genetic deletion.

Can smoking affect spermatogenesis?

Yes.

Smoking is associated with oxidative stress and poorer reproductive health.

WHO recommends tobacco cessation for people planning pregnancy.

Does fever affect sperm production?

A significant fever can temporarily impair spermatogenesis and may influence semen results weeks later.

Can sperm production recover after anabolic steroids?

Often it can improve after withdrawal, but recovery varies.

Current EAU guidance describes improvement commonly occurring over approximately:

6–12 months

after cessation.

Latest Scientific Perspective: 2025–2026

Modern spermatogenesis research is moving far beyond conventional sperm counting.

WHO's First Global Infertility Guideline

On **28 November 2025**, WHO published its first global guideline dedicated to:

- prevention,
- diagnosis,
- and treatment of infertility.

It emphasizes:

- evidence-based investigation,
- lifestyle measures,
- appropriate fertility treatment,
- progression to ART where necessary.

Molecular Understanding Is Rapidly Expanding

Recent research increasingly maps spermatogenesis using:

- single-cell sequencing,
- transcriptomics,
- epigenetics,
- proteomics.

A 2024 scoping review describes major advances in mapping the molecular development of human germ cells and understanding spermatogenic failure.

Temperature Biology Is Better Understood

A 2025 review further details how abnormal temperature can affect:

- germ-cell apoptosis,
- oxidative stress,
- DNA damage,
- blood-testis-barrier integrity.

This adds modern molecular detail to the longstanding clinical understanding that the testes require a specialized thermal environment.

Stem-Cell Science Is Promising but Experimental

Research into:

- spermatogonial stem cells,
- organ culture,
- 3D testicular models,
- artificial gametogenesis

may one day expand fertility options for men currently lacking sperm.

However, routine clinically validated restoration of human spermatogenesis with these techniques has not yet been achieved.

Dr. Nizamuddin Qasmi and Saira Health Care

I am **Dr. Nizamuddin Qasmi**, Founder and Chief Physician of **Saira Health Care**, with focused clinical practice in:

Sexual Disorders & Infertility

My professional education and additional training include:

- **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**

Saira Health Care's public physician profile lists:

- BUMS,
- MD,
- CGO,

- Certificate in Infertility,
- Certificate in Urology – London, UK,

and describes my focused clinical work in:

- sexual disorders,
- azoospermia,
- oligospermia,
- asthenospermia,
- abnormal sperm morphology,
- necrospermia,
- varicocele,
- and other infertility conditions.

Saira Health Care's current educational profile additionally lists:

- **Masters in Male Infertility – MasterHealthPro (HealthPro)**
- and **Integrated Sexual and Reproductive Health – ISRH, UNFPA**.

Saira Health Care's Contribution to Spermatogenesis and Male Infertility Care

One of the most important contributions I believe Saira Health Care can make is:

helping patients understand what their sperm problem actually represents biologically.

A man with:

low sperm count

is different from a man with:

maturation arrest.

A man with:

obstructive azoospermia

is completely different from a man with:

Sertoli-cell-only syndrome.

A man taking:

testosterone injections

is different from a man with:

a complete Y-chromosome deletion.

And these patients should not all receive the same treatment.

Our Integrative Philosophy

Saira Health Care describes its approach as patient-centered and combining:

- traditional knowledge,
- modern research,
- individualized treatment,
- lifestyle guidance.

For spermatogenic disorders, I believe this means:

Use modern medicine to diagnose:

- semen abnormalities,
- hormones,
- genetics,
- obstruction,
- histological failure.

Use Unani medicine to support:

- nutrition,
- lifestyle,
- general reproductive health,
- selected male-fertility disorders.

Use surgery when required for:

- varicocele,
- obstruction,
- sperm retrieval.

Use ART when required:

- IUI,

- IVF,
- ICSI.

That is a more complete treatment philosophy than forcing every patient into one system.

What I Do Not Promise

I do not believe patients should be told:

"This medicine will make every testis produce sperm."

That is not medically realistic.

A complete AZFa deletion cannot be corrected with a fertility supplement.

A blocked vas deferens is not opened by a spermatogenic powder.

A genuine hormonal deficiency should not be ignored while giving only herbs.

A man with persistent NOA should not lose years if micro-TESE and ICSI are appropriate.

Likewise:

patients with mild idiopathic semen abnormalities should not automatically be pushed toward invasive treatment when careful medical, lifestyle and individualized traditional care may reasonably be attempted first.

Good fertility medicine requires balance.

My Final Message to Patients

If your doctor tells you:

"Your sperm production is weak"

do not stop at that statement.

Ask:

What exactly is abnormal?

Is my sperm count low?

Is sperm completely absent?

Is the problem production or obstruction?

What is my FSH?

What is my LH?

What is my testosterone?

Do I have a genuine hormone deficiency?

Is there a clinical varicocele?

Did I have undescended testes?

Have I used testosterone or steroids?

Do I need genetic testing?

Could I have maturation arrest?

Could my sperm production be focal?

Would Unani lifestyle and pharmacotherapy be useful in my particular case?

Do I need hormonal treatment?

Do I need surgery?

Would micro-TESE be appropriate?

How is my wife's fertility and ovarian reserve?

These questions lead to real treatment.

Conclusion

Spermatogenesis is the complex biological process through which male germ cells develop into spermatozoa inside the seminiferous tubules of the testes.

It depends on coordinated interaction between:

- spermatogonial stem cells,
- developing germ cells,
- Sertoli cells,
- Leydig cells,
- peritubular cells,
- FSH,
- LH,
- testosterone,
- genetics,

- temperature,
- and the specialized testicular environment.

The major cellular stages include:

mitosis → meiosis → spermiogenesis → spermiation

followed by:

epididymal maturation.

Modern literature commonly estimates complete human intratesticular spermatogenesis at approximately:

74 days

with further post-testicular maturation afterward.

Disorders of spermatogenesis may manifest as:

- oligozoospermia,
- severe oligozoospermia,
- non-obstructive azoospermia,
- hypospermatogenesis,
- early maturation arrest,
- late maturation arrest,
- Sertoli-cell-only syndrome.

Potential causes include:

- genetic abnormalities,
- hormonal deficiencies,
- testosterone and anabolic steroids,
- cryptorchidism,
- varicocele,
- testicular infection,
- trauma,
- torsion,
- chemotherapy,

- radiation,
- metabolic disease,
- excessive heat,
- environmental exposure.

Diagnosis may require:

- standardized semen analysis,
- repeat semen testing,
- physical examination,
- FSH,
- LH,
- testosterone,
- genetic testing,
- selective ultrasound,
- and histology in appropriate situations.

Treatment must match the cause.

For men with:

hypogonadotropic hypogonadism

fertility-directed hCG/FSH therapy can successfully induce spermatogenesis and is strongly supported by current EAU guidance.

In contrast, routine hormone therapy is not proven to restore classical primary testicular failure.

External:

testosterone should not be used as male-infertility treatment

because it can suppress spermatogenesis.

For appropriately selected men with persistent:

non-obstructive azoospermia

micro-TESE remains the preferred sperm-retrieval procedure.

The **Unani system of medicine** may contribute meaningfully through:

- Mizaj-based individualization,

- Ilaj-bil-Ghiza,
- Ilaj-bit-Tadbir,
- Ilaj-bid-Dawa,
- nutrition,
- lifestyle correction,
- selected traditional reproductive medicines.

CCRUM has published preliminary clinical research showing improvement in selected semen parameters in men with oligospermia receiving Unani formulations, though stronger controlled studies are still required.

At **Saira Health Care**, **Dr. Qasmi's Nuskha No. 129 – Vitasem Max** may be used within selected individualized Unani male-fertility programmes.

Other products mentioned in the supplied material—**Semen Gold Plus, Spermzoa and Sperm Plus**—are currently classified by Saira Health Care Pharmacy as **Ayurvedic**, not classical Unani formulations, and should be described accurately.

My approach at Saira Health Care can therefore be summarized as:

Understand how sperm production is failing.

Do not treat every low sperm count the same way.

Differentiate production failure from obstruction.

Assess hormones correctly.

Never use external testosterone as fertility treatment.

Investigate genetics when indicated.

Correct smoking, metabolic disease, heat and harmful exposures.

Treat clinically significant varicocele appropriately.

Use individualized Unani dietotherapy, regimenal care and pharmacotherapy where suitable.

Monitor sperm objectively.

Preserve rare sperm when necessary.

Use micro-TESE, IVF or ICSI when appropriate.

And never promise that one medicine can reverse every biological cause of impaired spermatogenesis.

My central message to patients is:

Spermatogenesis is a complex biological process, and impaired sperm production does not have one universal cause or one universal treatment. The best fertility outcomes come from finding where the process is failing and then choosing an individualized treatment that combines appropriate modern diagnosis with responsible supportive Unani care.

About the Author

Dr. Nizamuddin Qasmi

Founder & Chief Physician, Saira Health Care
Focused Practice in Sexual Disorders & Infertility

Professional Education & 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**

Saira Health Care identifies Dr. Nizamuddin Qasmi as Founder and Chief Physician with focused clinical work involving sexual disorders and infertility, including major male-factor conditions affecting sperm production and quality.

His approach combines:

- Unani medicine,
- semen-analysis interpretation,
- male reproductive endocrinology,
- fertility evaluation,
- sexual-health assessment,
- lifestyle correction,
- and timely referral for reproductive surgery or assisted reproduction.

Medical Disclaimer

This article is intended for:

- general education,
- fertility awareness,
- and patient understanding.

It is not a substitute for:

- individual medical consultation,
- semen analysis,
- physical examination,
- hormone testing,
- genetic testing,
- scrotal imaging,
- testicular histology,
- reproductive-urology assessment,
- ART consultation.

Do not independently start or stop:

- testosterone,
- anabolic steroids,
- hCG,
- FSH,
- clomiphene,
- aromatase inhibitors,
- dopamine agonists,
- antibiotics,
- Unani medicines,
- Ayurvedic medicines,
- mineral preparations,
- fertility supplements

without appropriate professional supervision.

Spermogenic, Dr. Qasmi's Nuskha No. 129, Semen Gold Plus, Spermzoa, Sperm Plus or any other fertility formulation should not be interpreted as a guaranteed method of restoring spermatogenesis in every patient.

Men with:

- azoospermia,
- severe oligozoospermia,
- very high FSH,
- small testes,
- maturation arrest,
- suspected genetic disease,
- history of undescended testes,
- chemotherapy,
- radiotherapy,
- or prolonged infertility

should receive appropriate specialist evaluation.

Stem-cell therapy and complete in-vitro production of clinically usable human sperm remain experimental and should not currently be advertised as established cures for spermatogenic failure.

Where:

- hormonal induction,
- varicocele repair,
- reconstructive surgery,
- micro-TESE,
- IUI,
- IVF,
- or ICSI

is medically indicated, timely treatment should form part of responsible integrative fertility care.