Severe Oligospermia

Severe Oligozoospermia: Causes, Diagnosis, Male Infertility, Treatment and an Integrative Unani Approach

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When a man receives a semen-analysis report showing a very low sperm count, the first reaction is often fear.

Many patients ask me:

“Doctor, does this mean I can never become a father?”

Another may ask:

“My sperm count is only 2 million. Is there any treatment?”

Some patients have already started taking multiple vitamins, herbal products, hormones or so-called “sperm boosters” without knowing **why their sperm concentration became severely low in the first place**.

My first message to such patients is:

Severe oligospermia is a serious male-fertility finding, but it is not the same as complete sterility.

Sperm are still present in the semen. Depending upon the underlying cause, some men improve after appropriate treatment, some may still achieve natural conception, and others can become fathers through assisted reproductive techniques such as IVF with ICSI.

However, a sperm concentration below approximately **5 million sperm per millilitre** deserves a much more detailed investigation than a mildly reduced sperm count because significant hormonal, genetic, testicular, varicocele-related or obstructive causes become more important.

Current European Association of Urology guidance defines oligozoospermia as a sperm concentration below **16 million/mL**, based on contemporary WHO reference data, and recognizes **less than 5 million/mL as severe oligozoospermia** in everyday clinical practice. Importantly, WHO reference limits are not absolute dividing lines between fertile and infertile men.

At **Saira Health Care**, my approach therefore begins with one question:

Why is the sperm count severely low?

Finding the cause is much more important than immediately prescribing a tonic.

What Is Oligospermia?

The more precise medical term is **oligozoospermia**.

It means that the concentration of spermatozoa in semen is lower than the expected reference range.

The current WHO sixth-edition reference data, incorporated into European urological guidance, give a lower reference limit of approximately:

- **16 million sperm/mL** for sperm concentration,
- **39 million sperm per ejaculate** for total sperm number,
- **30%** for progressive motility,
- **42%** for total motility,
- **4%** for normal morphology.

These values represent the lower fifth centile among men whose partners conceived naturally within twelve months. They are **reference values, not a diagnostic border between fertility and infertility**.

A man slightly below 16 million/mL may still father a child naturally.

Likewise, a man above 16 million/mL can still experience infertility if sperm movement, morphology, DNA quality, sexual function or his partner's reproductive factors are abnormal.

What Is Severe Oligospermia?

In contemporary andrology, sperm concentration below approximately:

5 million sperm/mL

is commonly described as **severe oligozoospermia**.

This is clinically important because men with counts below this level have a greater frequency of:

- impaired sperm production,
- genetic abnormalities,
- chromosomal disorders,

- Y-chromosome abnormalities,
- genital-tract obstruction,
- significant testicular dysfunction.

The current EAU guideline specifically notes that severe oligozoospermia below 5 million/mL is associated with an increased incidence of genital-tract obstruction and genetic abnormalities.

This is why I do not treat a sperm concentration of 2 million/mL as simply a more severe version of a count of 14 million/mL.

The investigation needs to become more detailed.

Oligospermia Is Not the Same as Low Semen Volume

This terminology is often confused.

Oligozoospermia means low sperm concentration.

Hypospermia means low semen volume.

A patient can have:

- normal semen volume with severe oligozoospermia,
- low semen volume with normal sperm concentration,
- both abnormalities together.

This distinction can provide diagnostic clues.

For example, very low semen volume combined with severe oligozoospermia may increase suspicion of an ejaculatory-duct problem or another abnormality of the seminal tract.

Severe Oligospermia Is Not Azoospermia

Another important distinction is between:

Severe oligozoospermia

A very small number of sperm remain in the ejaculate.

Azoospermia

No sperm are detected in the ejaculate after appropriate laboratory examination.

Cryptozoospermia

No sperm may be seen initially, but extremely small numbers are detected after the semen sample is centrifuged and the pellet examined carefully.

The WHO sixth-edition laboratory approach and current EAU guidance emphasize examining very low-count samples carefully because identifying even a few sperm may influence fertility treatment and potentially allow their use for ICSI.

One Semen Test Is Not Enough to Diagnose the Whole Problem

Semen parameters naturally fluctuate.

A man's sperm concentration can change because of:

- recent fever,
- incomplete sample collection,
- duration of abstinence,
- illness,
- laboratory variation,
- medication,
- temporary physiological changes.

Current EAU guidance recommends **at least two consecutive semen analyses when the baseline result is abnormal**. The AUA/ASRM guideline likewise emphasizes repeat semen analysis because sperm measurements can vary substantially between ejaculates.

Therefore, if a man receives one unexpectedly low report, I usually want the result appropriately confirmed before drawing major conclusions—unless there is an obvious urgent clinical reason not to wait.

How Semen Analysis Should Be Interpreted

I never look only at the sperm concentration.

A complete semen analysis provides information about several parameters.

Semen Volume

Low volume may provide clues about:

- incomplete sample collection,
- ejaculatory-duct obstruction,
- absence or abnormality of seminal vesicles or vas deferens,
- retrograde ejaculation,
- androgen deficiency.

Sperm Concentration

This is the number of sperm per millilitre.

Total Sperm Number

This represents the total number of sperm in the complete ejaculate.

Current WHO-based guidance considers total sperm output potentially more diagnostically useful than concentration alone, making accurate semen-volume measurement important.

Progressive Motility

This tells us how many sperm are moving forward effectively.

Total Motility

This includes progressive and non-progressive movement.

Morphology

This examines sperm shape.

Vitality

When many sperm are immotile, vitality testing helps determine whether those sperm are alive.

Oligoasthenoteratozoospermia — OAT

Severe oligospermia often does not occur alone.

A man's report may show:

- low sperm concentration,
- poor progressive motility,
- abnormal morphology.

When all three occur together, the pattern is commonly called:

oligo-astheno-teratozoospermia, or **OAT**.

This can have a greater impact on fertility than an isolated reduction in sperm concentration.

Current guidelines emphasize that the more semen abnormalities present simultaneously, the more clinically meaningful the fertility impairment tends to become.

Does Severe Oligospermia Mean Infertility?

Not automatically.

This point is very important.

Semen analysis measures the probability of fertility; it does not provide a simple yes-or-no answer in most men.

The current EAU guideline explicitly states that semen analysis by itself cannot reliably distinguish fertile from infertile men.

Natural pregnancy depends upon many factors, including:

- sperm concentration,
- total sperm number,
- motility,
- morphology,
- sperm DNA integrity,
- ejaculation frequency,
- timing of intercourse,
- female partner's age,
- ovulation,
- fallopian-tube health,
- ovarian reserve,
- reproductive tract anatomy.

A concentration of 3 million/mL generally makes natural conception substantially more difficult than a concentration of 30 million/mL.

But it does not mathematically make conception impossible.

Couple-Based Assessment Is Essential

Male infertility should never be evaluated completely separately from the female partner.

Current EAU guidance recommends simultaneous evaluation of both partners when a couple seeks fertility care.

This becomes especially important in severe oligospermia because treatment decisions depend partly upon the female partner's:

- age,
- ovarian reserve,
- tubal status,
- reproductive history.

For example, repairing a clinically significant varicocele and waiting several months for sperm production to improve may be reasonable in one couple.

In another couple where the female partner has substantially reduced ovarian reserve, proceeding more rapidly toward IVF/ICSI may be appropriate.

The sperm report is therefore only one part of the couple's fertility plan.

What Causes Severe Oligospermia?

There are many causes.

I broadly think about them in several categories:

1. testicular sperm-production problems,
2. hormonal or endocrine disorders,
3. genetic causes,
4. varicocele,
5. partial obstruction,
6. previous testicular injury or disease,
7. medicines and hormones,
8. cancer treatment,

9. environmental and lifestyle factors,
10. unexplained or idiopathic infertility.

Often, more than one factor is present.

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1. Primary Testicular Failure or Impaired Spermatogenesis

Sperm are produced inside the seminiferous tubules of the testes.

Any condition that significantly damages this sperm-producing machinery can result in severe oligospermia.

When the testicles themselves are not producing sperm adequately, the pituitary gland often responds by producing more **FSH**.

Therefore, the pattern may include:

- severe oligospermia,
- small testes,
- elevated FSH,
- sometimes elevated LH,
- normal or reduced testosterone.

Current EAU guidance notes that FSH tends to rise as the number of functioning sperm-producing germ cells decreases, although a normal FSH does not completely exclude significant spermatogenic failure.

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2. Genetic Causes

Genetic assessment becomes especially important in severe oligospermia.

Possible causes include:

- chromosomal abnormalities,
- Klinefelter syndrome,
- Y-chromosome microdeletions,
- other less common inherited abnormalities.

The frequency of chromosomal abnormalities increases as sperm production becomes more severely impaired. European guideline data indicate that men with sperm

concentrations below 5 million/mL have a much higher frequency of chromosomal abnormalities than the general population.

Klinefelter Syndrome

Klinefelter syndrome most commonly occurs when a man has an additional X chromosome:

47,XXY

rather than the usual 46,XY chromosome complement.

Possible features include:

- very small firm testes,
- impaired sperm production,
- elevated FSH,
- reduced testosterone in some men,
- gynecomastia,
- reduced fertility.

The severity varies considerably.

Some men are diagnosed only during an infertility evaluation.

Y-Chromosome Microdeletions

Certain regions of the Y chromosome contain genes that are critical for sperm production.

Deletions in these regions can result in severe oligospermia or azoospermia.

This is important not only diagnostically but also for counselling before ICSI because some Y-chromosome abnormalities can be transmitted to male offspring.

Current European guidance recommends Y-chromosome microdeletion testing in men with sperm concentration **≤1 million/mL** and advises considering it below 5 million/mL.

The **2024 AUA/ASRM amendment** similarly lowered the routine Y-microdeletion threshold to **≤1 million/mL** in men with primary infertility when findings suggest impaired sperm production, such as elevated FSH or testicular atrophy.

This is an important modern update.

Older sources commonly recommended Y-microdeletion testing for every man below 5 million/mL.

Today, the threshold is more individualized.

When Is Karyotype Testing Considered?

Major guidelines are not completely identical in their thresholds.

The EAU recommends standard karyotype analysis and genetic counselling for men with azoospermia or sperm concentration below 5 million/mL.

The 2024 AUA/ASRM guideline uses a more phenotype-based approach, recommending karyotype testing in primary infertility with azoospermia or sperm concentration below 5 million/mL **when accompanied by elevated FSH, testicular atrophy or evidence of impaired sperm production**.

This difference is why genetic testing should be selected by an appropriately trained male-fertility specialist rather than simply ordered as an identical package for every patient.

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3. Varicocele

A **varicocele** is abnormal enlargement of veins around the testicle.

It is one of the most important potentially correctable findings in male infertility.

Current EAU data estimate that varicocele is found in about:

- 15% of the general male population,
- approximately 25% of men with abnormal semen analysis,
- around 35–40% of men presenting with infertility.

The mechanism probably involves combinations of:

- increased scrotal temperature,
- oxidative stress,
- hypoxia,
- altered testicular environment.

Does Every Varicocele Need Surgery?

No.

This is another important misconception.

A small varicocele visible only on ultrasound is not automatically responsible for infertility.

Current EAU guidance recommends against treating a **subclinical varicocele** or a varicocele in an infertile man whose semen parameters are normal.

However, treatment can be appropriate when there is:

- a clinically palpable varicocele,
- abnormal semen parameters,
- otherwise unexplained male-factor infertility,
- a couple for whom improving natural fertility is clinically worthwhile.

Varicocele repair may improve sperm concentration and pregnancy rates in appropriately selected patients.

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4. Hormonal Problems

Normal sperm production depends upon the hypothalamic-pituitary-testicular axis.

The brain produces hormonal signals that stimulate the testes.

Important hormones include:

- GnRH,
- LH,
- FSH,
- testosterone.

Abnormalities at any level can interfere with sperm production.

FSH

FSH stimulates Sertoli-cell function and spermatogenesis.

High FSH may suggest that the testes are struggling to produce sperm.

Low or inappropriately normal FSH in a man with low testosterone can suggest a hypothalamic or pituitary disorder.

LH

LH stimulates Leydig cells to produce testosterone.

It is especially useful when interpreting low testosterone and suspected hormonal infertility.

Testosterone

Testosterone is essential for spermatogenesis.

But an extremely important point is:

taking external testosterone can suppress sperm production.

The testes require very high local concentrations of testosterone produced inside the testicle under LH stimulation.

Testosterone injections, gels or bodybuilding hormones suppress the pituitary hormones LH and FSH.

This can dramatically reduce or completely stop sperm production.

Never Treat Male Infertility With Testosterone Monotherapy

Current EAU guidance explicitly states:

testosterone therapy is contraindicated in infertile men who wish to conceive.

The AUA/ASRM guideline likewise advises that testosterone monotherapy should not be prescribed to men interested in current or future fertility.

This is one of the most important warnings for men with severe oligospermia.

A man may have low testosterone and low sperm count.

Giving him ordinary testosterone replacement can make the sperm problem worse.

The treatment of low testosterone in a man desiring fertility requires a completely different strategy.

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5. Hypogonadotropic Hypogonadism

In some men, the testes may be capable of producing sperm, but the pituitary or hypothalamus is not providing sufficient stimulation.

This is called **hypogonadotropic hypogonadism**.

Possible causes include:

- congenital disorders,
- pituitary disease,
- hypothalamic disease,
- severe systemic illness,
- certain medications.

The hormone pattern may include:

- low testosterone,
- low or inappropriately normal LH,
- low FSH.

This is a particularly important diagnosis because sperm production can often be stimulated using appropriate gonadotropin treatment.

Current EAU guidance strongly recommends hCG and FSH-based therapy for men with congenital or acquired hypogonadotropic hypogonadism who want fertility.

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6. High Prolactin

Excessive prolactin can suppress the reproductive hormonal axis.

A man may experience:

- low testosterone,
- low libido,
- erectile dysfunction,
- impaired sperm production.

Causes can include:

- pituitary adenoma,
- certain medicines,
- other endocrine abnormalities.

Current EAU guidance recommends treating confirmed hyperprolactinemia according to the underlying cause; dopamine-agonist therapy is commonly used when appropriate.

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7. Undescended Testes

A history of **cryptorchidism**, or undescended testis, is an important male-infertility risk factor.

The testes are designed to function at a temperature slightly below core body temperature.

If one or both remain outside the scrotum during childhood, sperm-producing tissue can be damaged.

Even after successful childhood surgery, some men have reduced adult fertility—particularly when both testes were affected.

Current EAU guidance specifically includes previous cryptorchidism in the reproductive history of infertile men.

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8. Testicular Torsion and Trauma

Previous twisting of the testicle, called **testicular torsion**, can damage sperm-producing tissue if blood supply was interrupted.

Severe testicular trauma can have similar consequences.

A history of childhood or adolescent testicular emergency is therefore clinically important even if the patient has had no symptoms for many years.

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9. Orchitis and Genital Infection

Certain infections can damage the testes or reproductive tract.

Examples include:

- severe epididymo-orchitis,
- some viral orchitis,
- sexually transmitted infections,
- tuberculosis in selected settings.

Infection may affect fertility by:

- damaging sperm-producing tissue,
- causing inflammation,
- producing partial obstruction.

However, infection should be diagnosed rather than assumed.

Repeated antibiotics should not be prescribed merely because sperm concentration is low.

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10. Partial Obstruction

Sperm may be produced but have difficulty travelling through the reproductive tract.

Possible locations include:

- epididymis,
- vas deferens,
- ejaculatory ducts.

Severe oligospermia associated with:

- unusually low semen volume,
- abnormal semen pH,
- dilated reproductive structures,
- previous genital surgery or infection

may increase suspicion of obstruction.

Current EAU guidance recommends transrectal ultrasound when distal obstruction is clinically suspected.

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11. Congenital Abnormalities of the Vas Deferens

Some men are born without one or both vas deferens.

This is more commonly associated with azoospermia than severe oligospermia but remains important in the differential diagnosis of severe male-factor infertility.

Abnormalities of the vas deferens can be associated with **CFTR gene mutations**.

Genetic counselling and appropriate partner testing may therefore be necessary in affected couples.

12. Anabolic Steroids and Bodybuilding Drugs

Anabolic steroid use is an increasingly important cause of male infertility.

These substances suppress the body's own LH and FSH production.

The testes receive less stimulation and sperm production may fall dramatically.

Current EAU guidance states that infertility caused by anabolic steroid use should initially be managed by stopping the steroid, with sperm production often improving over approximately **six to twelve months**, although recovery varies.

Some patients require specialist hormonal treatment if recovery remains inadequate.

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13. Cancer Treatment

Chemotherapy and radiation therapy can damage sperm-producing cells.

The degree of damage depends upon:

- drug used,
- cumulative dose,
- radiation field,
- baseline testicular function.

Men who may receive gonadotoxic cancer treatment should ideally discuss **sperm cryopreservation before treatment whenever possible**.

A cancer survivor with severe oligospermia deserves assessment of both fertility and hormonal testicular function.

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14. Testicular Cancer

Male infertility and testicular disease are interconnected.

Some men with testicular cancer already have impaired spermatogenesis before treatment.

Others develop reduced sperm production afterward.

A testicular lump or unexplained testicular abnormality should therefore never be dismissed simply because the man presented for infertility.

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15. Obesity and Metabolic Health

Obesity can influence male fertility through:

- hormonal changes,
- increased conversion of testosterone to estrogen,
- metabolic inflammation,
- higher scrotal temperature,
- associated diabetes and metabolic syndrome.

Current European guidance states that obesity, low physical activity, smoking and high alcohol intake are associated with reduced sperm quality and recommends improving these factors in infertile men.

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16. Smoking

Smoking has been associated with:

- poorer semen quality,
- increased oxidative stress,
- increased sperm DNA damage.

Stopping smoking is therefore one of the most rational lifestyle measures for an infertile man.

It does not guarantee that a sperm concentration of 2 million/mL will become normal.

But it removes an avoidable reproductive risk factor.

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17. Alcohol

Heavy alcohol intake may negatively affect:

- testosterone,
- semen parameters,
- general reproductive health.

Moderation or avoidance is appropriate in men being treated for significant infertility.

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18. Heat Exposure

The testes function best below normal body-core temperature.

Repeated excessive heat exposure may contribute to impaired sperm production in susceptible men.

Relevant exposures can include occupational heat and certain repeated high-temperature environments.

However, patients should not become excessively fearful of ordinary daily activities.

The important goal is to reduce substantial, repetitive and avoidable heat exposure.

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19. Environmental and Occupational Exposure

Current male-infertility guidelines recognize potential effects from:

- environmental toxins,
- pesticides,
- heavy metals,
- occupational exposures,
- radiation,
- gonadotoxic chemicals.

A detailed work history can therefore be valuable.

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20. Medicines

Some medicines can influence sperm production or sexual function.

Important examples may include:

- exogenous testosterone,
- anabolic steroids,
- certain chemotherapy drugs,
- some hormonal medicines.

Patients should provide a complete list of:

- prescription medicines,
- injections,

- gym products,
- supplements,
- herbal medicines.

Do not stop an essential prescription drug without discussing alternatives with the prescribing physician.

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21. Fever and Recent Illness

Sperm production is sensitive to systemic illness.

A significant febrile illness can temporarily lower sperm concentration or motility.

Because the process of sperm development takes several weeks, the effect may still be visible in semen testing after the patient has recovered.

This is one reason a previous report should be interpreted within the patient's recent medical history.

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22. Idiopathic Severe Oligospermia

Sometimes no single cause is identified despite appropriate investigation.

This is described as **idiopathic male infertility**.

Current EAU data indicate that a substantial proportion of male infertility remains idiopathic.

“Idiopathic” does not mean imaginary.

It means that currently available testing has not identified a definite cause.

How I Assess a Man With Severe Oligospermia

I do not begin simply by prescribing medicine for three months.

A severe sperm-count abnormality deserves structured evaluation.

Detailed Reproductive History

I ask:

- How long has the couple been trying?

- Has he caused a previous pregnancy?
- Has his partner ever conceived?
- Has the partner undergone fertility evaluation?
- How frequent is intercourse?
- Is ejaculation occurring normally?

Previous fertility can provide useful information.

Childhood History

Important questions include:

- Were the testes descended normally?
- Was there childhood testicular surgery?
- Was there hernia surgery?
- Was orchidopexy performed?
- Was there torsion?
- Was there significant trauma?

A problem that happened decades ago may explain today's semen result.

Puberty and Sexual Development

Delayed puberty or reduced secondary sexual characteristics can suggest endocrine or genetic disease.

I look at:

- body hair,
- beard development,
- muscle development,
- gynecomastia,
- sexual development.

Sexual History

Infertility can sometimes be partly caused by a sexual problem rather than sperm production alone.

I ask about:

- libido,
- erection,
- ejaculation,
- intercourse frequency,
- timing around ovulation.

Treating severe oligospermia while ignoring erectile or ejaculatory dysfunction would be incomplete fertility care.

Examination of the Testes

Testicular size and consistency provide important clues.

Small, firm testes may suggest significant primary testicular dysfunction.

Normal-sized testes in a man with severe sperm reduction can point toward a different set of possibilities.

Current EAU guidance specifically recommends assessing testicular size, consistency, epididymis, vas deferens and varicocele in infertile men.

Examination for Varicocele

I examine the spermatic veins in the standing position and, when appropriate, during the Valsalva manoeuvre.

A clinically palpable varicocele is more relevant to treatment decisions than a very small ultrasound-only finding.

Doppler ultrasound may be useful when the examination is unclear or for selected clinical questions.

Hormone Testing

For severe oligospermia, hormonal evaluation is important.

Current EAU recommendations include:

- total testosterone,
- FSH,
- LH

in men with oligozoospermia or azoospermia.

Additional testing may be selected according to the findings.

What Can FSH Tell Us?

High FSH + Small Testes

This pattern often raises concern for primary testicular spermatogenic failure.

Low FSH + Low Testosterone

This may suggest inadequate pituitary or hypothalamic stimulation.

Normal FSH

Normal FSH does not automatically prove that sperm production is normal.

Certain forms of maturation arrest can occur despite normal FSH.

Prolactin Testing

Prolactin testing becomes particularly relevant when there is:

- low libido,
- low testosterone with a central hormonal pattern,
- suspected pituitary disease.

It should not necessarily be ordered indiscriminately in every patient.

Genetic Tests

Depending upon sperm concentration, testicular findings, FSH and clinical history, genetic evaluation may include:

- karyotype,
- Y-chromosome microdeletion testing,
- CFTR testing in selected structural/obstructive conditions.

Genetic counselling becomes especially important before ART when an inheritable abnormality is detected.

Ultrasound

Ultrasound can be valuable in selected cases for assessing:

- testicular volume,
- testicular lesions,
- varicocele,
- epididymis,
- possible obstruction.

The need should be based on the patient's clinical findings.

Sperm DNA Fragmentation

Many patients now request a DNA fragmentation test immediately.

It is **not required as the initial test for every infertile man**.

Current European guidance particularly supports considering sperm DNA fragmentation in settings such as:

- unexplained infertility,
- recurrent pregnancy loss,
- repeated ART failure.

It may also be relevant in selected patients with varicocele or important lifestyle risk factors.

Can Severe Oligospermia Improve?

Yes, in some men.

The chance depends strongly on the cause.

For example:

- hypogonadotropic hypogonadism may respond substantially to gonadotropins,

- sperm suppression from anabolic steroids can improve after withdrawal,
- a clinically significant varicocele may respond to repair,
- some partial obstructions can be treated,
- lifestyle-related contributors can be corrected.

On the other hand, severe primary testicular failure caused by certain genetic abnormalities may not be reversible with medication.

This is why I avoid promising:

“Your sperm count will definitely become normal.”

A scientifically responsible physician treats the cause and discusses realistic reproductive options.

Treatment of Severe Oligospermia

Treatment falls into two broad categories:

Improving or correcting the underlying male condition

and

Helping the couple achieve pregnancy despite the remaining sperm abnormality.

Sometimes we can do both.

Treatment of Varicocele

Appropriately selected men with:

- clinical varicocele,
- abnormal semen parameters,
- infertility

may benefit from varicocele repair.

Current EAU evidence indicates that repair can improve sperm concentration and may improve pregnancy rates in selected infertile couples.

Improvement does not occur immediately because new sperm production takes time.

Hormonal Treatment

Hormonal therapy should be matched to the diagnosis.

A patient with hypogonadotropic hypogonadism may benefit significantly from:

- hCG,
- FSH or related gonadotropins.

A patient with hyperprolactinemia may require treatment directed toward the prolactin abnormality.

A man with classical primary testicular failure usually does not respond in the same way to gonadotropin treatment.

Current EAU guidance specifically states that there is no substantial evidence that gonadotropins improve spermatogenesis in classical primary testicular failure.

Clomiphene, Aromatase Inhibitors and Other Hormonal Medicines

Medicines such as:

- clomiphene,
- letrozole,
- anastrozole,
- hCG

are sometimes used in carefully selected infertile men.

These treatments work by modifying the patient's own reproductive hormone axis rather than simply supplying external testosterone.

However, they are not universal sperm-count medicines.

Current EAU guidance states that evidence for routine SERMs or aromatase inhibitors in **idiopathic infertility** remains inconclusive, while the AUA/ASRM guideline allows selected hormonal therapy particularly in infertile men with low endogenous testosterone.

Therefore, hormone therapy should be based upon the hormonal pattern rather than prescribed automatically.

Antioxidants and Male Infertility

Antioxidant products are heavily marketed to infertile men.

Some individual trials have reported improvements in semen parameters, including a 2023 randomized Indian trial of an antioxidant combination.

However, guideline-level evidence remains less certain.

Current EAU guidance states that no conclusive data establish a clinically meaningful benefit of routine antioxidant treatment in idiopathic male infertility and advises against routine use as a universal treatment.

The AUA/ASRM guideline similarly advises patients that supplements and antioxidants have **questionable clinical utility** and that available evidence is insufficient to recommend specific products universally.

Therefore:

supplements should not replace investigation of severe oligospermia.

Lifestyle Treatment

Lifestyle changes are most useful when they address real risk factors.

I advise patients to focus on:

- stopping smoking,
- avoiding anabolic steroids,
- reducing heavy alcohol intake,
- improving obesity and metabolic health,
- maintaining regular physical activity,
- avoiding significant occupational toxins where possible,
- obtaining adequate sleep,
- managing chronic medical conditions.

Current EAU guidance supports lifestyle improvement in infertile men because obesity, inactivity, smoking and high alcohol intake are associated with poorer sperm quality.

Diet

There is no single food that transforms a sperm count of 2 million into 40 million.

A generally nutritious diet can support overall metabolic and reproductive health, but severe oligospermia requires medical investigation.

I advise patients to be cautious with claims such as:

“Eat this food for seven days and your sperm count will become normal.”

Spermatogenesis is biologically more complex than that.

Natural Pregnancy With Severe Oligospermia

Natural pregnancy is still possible in some couples.

The probability depends upon:

- actual total motile sperm number,
- repeat semen results,
- sperm motility,
- morphology,
- female partner's age,
- ovarian reserve,
- duration of infertility,
- frequency and timing of intercourse.

A man should therefore not be told he is “sterile” solely because his concentration is under 5 million/mL.

But the couple should also understand that the chance of natural pregnancy is generally lower, and time matters—particularly when the female partner is older.

IUI and Severe Oligospermia

Intrauterine insemination, or IUI, involves preparing sperm in the laboratory and placing selected motile sperm inside the uterus around ovulation.

IUI is less effective when the number of usable motile sperm is extremely low.

AUA/ASRM guidance states that when the **total motile sperm count after processing is below approximately 5 million**, the chance of pregnancy with IUI becomes limited, and IVF/ICSI may be considered.

This does not mean 5 million represents a magical absolute cutoff.

It means success becomes increasingly limited as the usable motile sperm number falls.

IVF and ICSI

For severe male-factor infertility, one of the most important reproductive technologies is:

ICSI — Intracytoplasmic Sperm Injection.

In ICSI, an embryologist selects a sperm and injects it directly into an egg.

ICSI was originally developed to overcome severe male-factor infertility and remains a major treatment in couples where sperm concentration or function is severely impaired. The 2026 ASRM committee opinion continues to describe ICSI as an established technique for couples with clear semen abnormalities.

Why ICSI Can Help in Severe Oligospermia

Conventional fertilization requires large numbers of sperm to reach and penetrate the egg.

ICSI dramatically reduces this numerical requirement because one viable sperm is selected for each mature egg.

AUA/ASRM guidance notes that ICSI can largely overcome adverse effects of sperm concentration, motility and morphology when sufficient viable sperm are available for injection.

This is why men with very low sperm counts should not assume biological fatherhood is impossible.

Genetic Counselling Before ICSI

ICSI can allow sperm with certain underlying genetic abnormalities to achieve fertilization.

This is beneficial, but it also means genetic counselling becomes important when the severe sperm problem itself has a hereditary basis.

For example, some Y-chromosome microdeletions can be transmitted to male offspring.

Current EAU guidance specifically recommends genetic counselling for couples when a genetic abnormality is identified.

Should Sperm Be Frozen in Severe Oligospermia?

In selected men with extremely low or fluctuating sperm counts, sperm cryopreservation may be discussed.

This can be particularly useful if there is concern that the patient may progress toward azoospermia or if future treatment could further impair spermatogenesis.

The decision should be individualized by the fertility team.

Severe Oligospermia and Sexual Function

Low sperm count itself usually does not automatically cause:

- erectile dysfunction,
- premature ejaculation,
- low libido.

However, the underlying hormonal disease might.

For example, a man with significant hypogonadism may have:

- low sperm production,
- reduced libido,
- poor erections.

Likewise, the psychological stress of infertility can reduce sexual confidence and make intercourse feel like a fertility "task."

This deserves attention.

Severe Oligospermia and Masculinity

One of the most harmful misconceptions is:

"Low sperm count means I am less of a man."

It does not.

Sperm concentration is a reproductive biological parameter.

It does not determine:

- masculinity,
- personality,

- sexual worth,
- ability to satisfy a partner.

A man with 2 million sperm/mL may have completely normal erections, desire and testosterone.

Another man with normal sperm concentration may have erectile dysfunction.

These are separate aspects of male sexual health.

Stress and Infertility

Severe infertility can be emotionally difficult.

Patients may experience:

- guilt,
- anxiety,
- depression,
- fear of losing a relationship,
- pressure from family,
- repeated focus on semen reports.

The male partner often receives less emotional support than the female partner despite experiencing significant distress.

Fertility treatment should recognize this.

The Unani Perspective on Severe Oligospermia

As a physician trained in Unani medicine and working with sexual disorders and infertility, I consider male fertility within the broader condition of the reproductive system and the patient's general health.

Unani literature has traditionally discussed reduced sperm quantity and related semen abnormalities under concepts including **Qillat-e-Huwaniya**.

Traditional assessment may consider:

- reproductive capacity,
- semen characteristics,

- constitutional temperament or Mizaj,
- general physical strength,
- nutrition,
- digestive function,
- sleep,
- physical activity,
- associated sexual complaints,
- systemic health.

This whole-person perspective can be useful, particularly in men whose fertility problem coexists with metabolic, digestive, lifestyle or sexual-health concerns.

However, severe oligospermia requires an important modern distinction:

A sperm concentration below 5 million/mL should not be treated solely according to traditional symptom patterns before genetic, hormonal, testicular and obstructive causes have been appropriately considered.

Unani Medicine and Male Fertility

Various Unani pharmacopoeial herbs and compound formulations have traditionally been used in male reproductive disorders.

The intended therapeutic approach may include supporting:

- reproductive health,
- general vitality,
- nutritional status,
- selected semen parameters,
- associated sexual-health concerns.

At Saira Health Care, I prefer to use these principles selectively and according to the patient's complete clinical picture rather than give the same formulation to every man whose laboratory report says “low sperm count.”

What Does Research Say About Unani Treatment of Oligospermia?

There is some published Unani research in oligospermia.

A CCRUM-associated publication from researchers at A.K. Tibbiya College, Aligarh Muslim University reviewed several earlier clinical studies involving **126 patients with idiopathic oligospermia** and reported improvements in different semen parameters with different Unani formulations.

This research is relevant because it demonstrates longstanding clinical investigation of Unani treatment in male infertility.

However, several limitations should be clearly understood.

The analysis was retrospective, combined older studies, involved different formulations, and used older definitions of oligospermia. It therefore cannot establish that one Unani treatment reliably reverses modern, properly investigated **severe oligozoospermia below 5 million/mL**, nor does it establish live-birth benefit.

This distinction is important.

Why Older Unani Studies Need Modern Re-Evaluation

Older studies frequently defined oligospermia using sperm-count thresholds such as 20 million/mL.

Current WHO-based reference data use **16 million/mL**, while severe oligozoospermia is clinically considered below approximately 5 million/mL.

Laboratory techniques, genetic testing and infertility treatment have also changed substantially.

Therefore, traditional formulations that appear promising should ideally be evaluated again using:

- current WHO semen-analysis standards,
- randomized controlled methodology,
- clearly defined severe oligozoospermia subgroups,
- hormonal and genetic classification,
- pregnancy outcomes,
- live-birth outcomes.

That would provide much stronger evidence.

Unani Treatment Should Be Cause-Aware

I believe this is particularly important.

Consider five men who each have sperm concentration of 3 million/mL.

The first has a clinically significant varicocele.

The second has a Y-chromosome microdeletion.

The third is taking testosterone injections.

The fourth has hypogonadotropic hypogonadism.

The fifth has no identifiable cause.

It would not be scientifically reasonable to give all five exactly the same treatment and expect the same outcome.

Their diagnoses are fundamentally different.

This is why I combine Unani principles with modern male-infertility assessment.

Where Unani Support May Be Most Reasonable

An integrative approach may be particularly appropriate in selected men with:

- idiopathic oligospermia after serious causes have been assessed,
- potentially modifiable lifestyle contributors,
- nutritional problems,
- associated general-health concerns,
- sexual-health complaints requiring broader management.

Even in these situations, semen improvement should be demonstrated by repeat objective testing rather than assumed from subjective wellbeing.

Herbal Treatment Is Not a Substitute for Genetic Testing

This deserves special emphasis.

If a man has:

- extremely low sperm concentration,
- small testes,
- high FSH,

I would not delay indicated genetic assessment while repeatedly changing herbal medicines.

Similarly, a patient with suspected obstruction or significant varicocele needs appropriate anatomical evaluation.

Integrative care means **adding rational supportive therapy to correct diagnosis**, not replacing diagnosis.

My Clinical Approach at Saira Health Care

At **Saira Health Care**, when I evaluate severe oligospermia, I try to answer several questions systematically.

First:

Is the sperm count genuinely and persistently low?

This requires reliable semen testing.

Second:

Is sperm production impaired, or is sperm transport affected?

Third:

Is there a correctable cause?

For example:

- hormonal disorder,
- varicocele,
- drug or steroid use,
- infection,
- obstruction.

Fourth:

Is genetic testing indicated?

Fifth:

How urgent is pregnancy for this couple?

Female age and ovarian reserve matter.

Sixth:

Can natural fertility reasonably be improved, or should ART be discussed early?

Only after answering these questions do I consider the most rational treatment plan.

Special Treatment Planning by Dr. Nizamuddin Qasmi

My focused clinical work in **Sexual Disorders & Infertility** allows severe oligospermia to be approached as more than a number on a semen report.

Depending upon the patient, my evaluation may incorporate:

- detailed reproductive history,
- sexual history,
- repeat semen analysis,
- sperm concentration and total count,
- motility and morphology,
- hormonal profile,
- testicular examination,
- varicocele assessment,
- review of medicines and anabolic steroids,
- relevant metabolic and lifestyle factors,
- investigation for infection or obstruction when indicated,
- genetic evaluation or referral when appropriate,
- review of the female partner's fertility situation.

Where clinically suitable, individualized Unani supportive management can be integrated alongside evidence-based male-fertility care.

What I Do Not Promise

I do not believe it is responsible to tell every patient:

“Your sperm count will become normal in three months.”

Severe oligospermia has many causes.

Some are highly treatable.

Some improve partially.

Some do not substantially improve despite good medical care.

Successful fertility care therefore means more than increasing a laboratory number.

The real objectives are:

- identify the cause,
- protect future testicular function,
- improve sperm production when medically possible,
- maximize the couple's chance of pregnancy,
- decide when ART should not be unnecessarily delayed.

Saira Health Care's Contribution to Sexual Disorders and Infertility

At **Saira Health Care**, male infertility is evaluated within the broader framework of sexual and reproductive health.

A man presenting with severe oligospermia may simultaneously have:

- erectile dysfunction,
- premature ejaculation,
- low libido,
- painful ejaculation,
- varicocele,
- hormonal symptoms,
- metabolic problems,
- fertility-related anxiety.

Treating one sperm number while ignoring the rest of the patient is incomplete.

Our objective is therefore a **whole-patient, couple-oriented, evidence-aware approach**.

Where necessary, patients can be guided toward appropriate:

- urological evaluation,
- genetic counselling,
- reproductive endocrinology,
- IVF/ICSI treatment.

Unani medicine can form part of this pathway where clinically appropriate, but it should not isolate the patient from investigations or fertility technology that may be important.

How Long Does Sperm Treatment Take?

Patients frequently ask for improvement within a few days.

Sperm production does not work that quickly.

Spermatogenesis is a biological process requiring weeks, followed by additional maturation within the reproductive tract.

Therefore, when a treatment genuinely influences sperm production, meaningful semen changes are generally assessed over a period of months rather than days.

This is one reason fertility therapies are commonly evaluated with repeat semen analysis after an appropriate interval.

Can a Sperm Count Fall Further?

Yes.

Severe oligospermia can sometimes fluctuate or progress.

This may occur with:

- continuing testicular disease,
- ongoing anabolic steroid use,
- gonadotoxic treatment,
- progressive testicular failure,
- certain genetic conditions.

This is why patients with extremely low counts may sometimes benefit from discussion of sperm cryopreservation.

Can Severe Oligospermia Become Azoospermia?

In selected patients, yes.

But it is not inevitable.

Whether progression occurs depends upon the cause.

A man with temporary steroid-induced suppression may improve dramatically after stopping steroids.

A man with progressive testicular failure may move in the opposite direction.

Diagnosis determines prognosis.

Frequently Asked Questions

What sperm count is considered severe oligospermia?

Severe oligozoospermia is generally used clinically for sperm concentration **below 5 million sperm/mL**. Current WHO-based lower reference data place the general lower reference level for sperm concentration around 16 million/mL.

Is 5 million sperm enough for pregnancy?

Pregnancy may still occur.

But the probability is influenced by motility, total sperm number, morphology, female fertility and other factors.

A count below 5 million/mL warrants detailed evaluation rather than being interpreted alone.

Is a sperm count of 1 million permanent?

Not necessarily.

It depends upon the cause.

Steroid suppression or some hormonal disorders may improve substantially.

Certain genetic or severe testicular disorders may be more difficult to reverse.

Can I become a father with severe oligospermia?

Yes, many men can.

Possible pathways include:

- natural conception in selected couples,
- treatment of a correctable male factor,

- IUI in appropriately selected circumstances,
- IVF/ICSI.

The best approach depends upon both partners.

Does severe oligospermia mean I am impotent?

No.

Infertility and erectile dysfunction are different conditions.

A man can have severe oligospermia with completely normal erections.

Does masturbation cause severe oligospermia?

Ordinary masturbation does not cause permanent severe oligospermia.

Very frequent ejaculation can temporarily influence semen measurements, which is why semen laboratories specify an abstinence interval before testing, but it does not ordinarily destroy sperm production.

Does having sex every day reduce sperm permanently?

No.

Frequent ejaculation can temporarily change sperm concentration in an individual sample, but it does not normally produce permanent severe oligospermia.

Can varicocele cause severe oligospermia?

Yes, varicocele can contribute to reduced sperm quality and concentration in some men.

Not every varicocele requires treatment.

Clinical examination and the couple's fertility situation determine whether repair is appropriate.

Will varicocele surgery definitely normalize my sperm count?

No.

Varicocele repair can improve semen parameters and fertility outcomes in appropriately selected patients, but it does not guarantee normalization.

Can testosterone increase my sperm count?

Ordinary testosterone replacement can do the opposite.

External testosterone suppresses LH and FSH and may reduce or completely stop sperm production.

Current guidelines advise against testosterone monotherapy in men seeking fertility.

I use gym steroids. Can they reduce sperm?

Yes.

Anabolic steroids can cause severe oligospermia or azoospermia.

Stopping them is a central part of treatment, and recovery may take many months.

Which hormones should be tested?

FSH, LH and total testosterone are particularly important in men with significant oligozoospermia.

Additional tests such as prolactin may be selected according to the clinical and hormonal picture.

Do I need genetic testing?

Possibly.

Severe oligospermia increases the likelihood of genetic abnormalities.

The need for karyotype and Y-chromosome microdeletion analysis depends upon sperm concentration, FSH, testicular size and evidence of impaired spermatogenesis. Current AUA/ASRM and EAU recommendations differ slightly in their precise thresholds.

Why is Y-chromosome testing particularly considered below 1 million?

Modern evidence indicates that clinically important complete Y-chromosome microdeletions become uncommon above approximately 1 million sperm/mL.

The 2024 AUA/ASRM amendment therefore recommends testing particularly at **≤1 million/mL** when findings indicate impaired sperm production.

Can diet increase sperm from 2 million to normal?

A healthy diet supports general reproductive health, but no specific diet can be guaranteed to normalize severe oligospermia.

A count this low should first be investigated for hormonal, genetic, anatomical and testicular causes.

Should I take antioxidants?

Some studies report improvement in semen parameters, but current major guidelines find the overall clinical evidence insufficient to recommend routine antioxidant treatment for every man with idiopathic infertility.

Do I need a DNA fragmentation test?

Not automatically.

It is not a routine first test for every case of severe oligospermia.

It is more useful in selected clinical situations such as unexplained infertility, recurrent pregnancy loss or repeated ART failure.

Is IUI possible with severe oligospermia?

Sometimes, but success depends particularly upon the total number of motile sperm remaining after laboratory preparation.

AUA/ASRM guidance notes that pregnancy chances with IUI become limited when fewer than approximately 5 million motile sperm remain after processing.

Is ICSI better for very low sperm counts?

ICSI is a major fertility technique for severe male-factor infertility because a viable sperm is injected directly into an egg.

It can overcome the numerical disadvantage of very low sperm concentration when usable sperm are available.

Can ICSI guarantee pregnancy?

No.

ICSI helps fertilization but cannot guarantee:

- embryo development,
- implantation,
- pregnancy,
- live birth.

Female age, egg quality, embryo quality and other factors remain important.

Can Unani medicine increase sperm count?

Some Unani clinical literature has reported improvements in semen parameters in men with idiopathic oligospermia. However, much of the evidence is older or methodologically limited, and direct high-quality evidence specifically for severe oligozoospermia below 5 million/mL remains insufficient.

I therefore use Unani principles most responsibly as part of an individualized, diagnosis-based fertility plan rather than as a substitute for genetic, hormonal or anatomical evaluation.

How long should Unani or medical treatment be tried before repeating semen analysis?

Because sperm production requires time, changes are usually evaluated over months rather than a few days.

The exact follow-up interval should depend upon the diagnosis and treatment being used.

A Message From Dr. Nizamuddin Qasmi

When a patient comes to me with a report showing:

Sperm concentration: 2 million/mL

I do not immediately tell him:

“Your sperm are weak.”

That statement does not explain anything.

I want to know:

Why are there only 2 million?

Is there a varicocele?

Are the testes small?

Is FSH high?

Is testosterone low?

Is the patient using gym steroids?

Did he have undescended testes as a child?

Was there previous testicular surgery?

Is there a partial obstruction?

Could there be a genetic cause?

Has the count been confirmed on another sample?

And equally important:

How old is his wife, and what is her fertility status?

These questions determine treatment far more intelligently than simply changing one sperm supplement after another.

My Advice to Patients

Do not panic after one abnormal report.

But do not ignore severe oligospermia either.

A sperm concentration below 5 million/mL deserves a serious and structured evaluation.

Do not begin testosterone injections for fertility without specialist advice.

Do not assume every low sperm count is caused by “heat” or “weakness.”

Do not spend months taking supplements while a significant varicocele, hormonal disorder or genetic abnormality remains unidentified.

And do not assume that biological fatherhood is impossible merely because the sperm count is severely low.

Modern reproductive medicine offers many possibilities.

Latest Medical Understanding

Current male-infertility care is increasingly moving away from treating the semen report alone.

Three developments are particularly important.

First: severe oligospermia is a signal for deeper investigation.

Current EAU guidance recognizes sperm concentration below 5 million/mL as severe and associates it with increased risk of obstruction and genetic abnormalities.

Second: genetic testing is becoming more targeted.

The 2024 AUA/ASRM amendment now recommends Y-chromosome microdeletion testing primarily in azoospermia or sperm concentration **≤1 million/mL** when impaired sperm production is suspected, rather than automatically using the older 5-million threshold for everyone.

Third: semen numbers should be interpreted in the context of the couple.

The WHO sixth-edition reference values are not absolute fertility cutoffs, and both partners' reproductive potential must be considered when choosing between medical treatment, surgery, IUI or IVF/ICSI.

Conclusion

Severe oligospermia, or severe oligozoospermia, generally refers to sperm concentration below approximately 5 million sperm/mL.

It is a significant male-fertility finding but does **not automatically mean permanent sterility**.

Possible causes include:

- primary testicular sperm-production failure,
- genetic abnormalities,
- Y-chromosome microdeletions,
- Klinefelter syndrome,

- varicocele,
- hormonal disorders,
- hypogonadotropic hypogonadism,
- hyperprolactinemia,
- undescended testes,
- testicular torsion or trauma,
- infection,
- partial reproductive-tract obstruction,
- anabolic steroid or testosterone use,
- cancer treatment,
- environmental factors,
- idiopathic male infertility.

A proper investigation generally includes:

- repeat high-quality semen analysis,
- reproductive and medical history,
- physical examination,
- assessment of testicular size,
- evaluation for varicocele,
- FSH, LH and testosterone,
- selected genetic testing,
- imaging where clinically indicated.

Current guidelines recommend more detailed genetic assessment as sperm concentration becomes extremely low.

Treatment should be **cause-specific**.

Correctable causes such as:

- varicocele,
- hormonal deficiency,
- anabolic steroid use,

- certain obstruction

may sometimes be treated directly.

When severe sperm reduction remains, assisted reproductive technologies—especially **IVF with ICSI**—can allow many affected men to achieve biological fatherhood.

From the Unani perspective, male infertility and reduced sperm parameters can be approached within the patient's broader reproductive, constitutional, nutritional and lifestyle health. Published Unani clinical literature has reported improvements in semen parameters in some patients with idiopathic oligospermia, but the available evidence is not sufficiently strong to claim that Unani therapy reliably reverses all causes of severe oligozoospermia.

For this reason, my approach at **Saira Health Care** is integrative but diagnosis-led:

identify why sperm production is severely reduced, correct reversible causes, evaluate genetic and hormonal factors, improve general reproductive health, use individualized Unani supportive management where appropriate, and guide the couple toward natural conception or assisted reproduction according to their realistic fertility potential.

The most important message I give my patients is:

Do not treat severe oligospermia simply as "low sperm count." Treat the man, identify the cause, and plan fertility for the couple.

About the Author

Dr. Nizamuddin Qasmi

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Qualifications & Professional Training

BUMS — Hamdard University, Delhi
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Dr. Nizamuddin Qasmi's clinical work at **Saira Health Care** focuses particularly on sexual disorders, male reproductive health and infertility.

His approach to severe oligospermia emphasizes detailed evaluation of male reproductive function rather than treatment based solely upon a single sperm-count value.

Where clinically appropriate, principles of Unani medicine are integrated with contemporary semen analysis, hormonal assessment, genetic evaluation, urological assessment and modern fertility treatment planning.

Medical Disclaimer

This article is intended for health education and general public awareness. It does not establish an individual diagnosis and does not replace examination or treatment by an appropriately qualified male-infertility specialist, urologist, reproductive physician or other healthcare professional.

Severe oligospermia can occasionally indicate an important hormonal, genetic, anatomical or testicular disorder.

Men with sperm concentration below approximately 5 million/mL should obtain appropriate medical evaluation rather than relying solely on over-the-counter fertility supplements.

Do not take testosterone, anabolic steroids, fertility hormones, prescription medicines or herbal/Unani formulations for severe oligospermia without appropriate professional assessment.

Couples should remember that successful fertility treatment depends upon the reproductive health of **both partners**, and female age and ovarian reserve may significantly influence the timing and choice of treatment.