

Decreased Sperm Motility (Asthenozoospermia): Causes, Diagnosis, 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, My Sperm Count Is Normal but Motility Is Low. Can We Still Conceive?”

A very common question I receive from male-infertility patients is:

“Doctor, my sperm count is normal, but only a small percentage of sperm are moving properly. Can I still become a father?”

Another patient may bring a report showing:

Progressive motility – 18%

and immediately assume:

“My sperm are completely useless.”

That conclusion is not necessarily correct.

Sperm motility is certainly an important part of male fertility, but it is only **one component of the complete semen profile**.

The medical term for persistently reduced sperm movement is:

Asthenozoospermia

It is also commonly called:

- asthenospermia,
- poor sperm motility,



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- low sperm motility,
- sluggish sperm,
- or decreased progressive motility.

Sperm need the ability to move effectively because, after ejaculation into the female reproductive tract, they must progress toward the site where fertilization can occur.

However, I want patients to understand from the beginning:

Low motility does not automatically mean sterility.

A man may have:

- mildly reduced progressive motility,
- excellent sperm concentration,
- a large total sperm number,
- good morphology,
- and a fertile female partner,

and natural pregnancy may still occur.

Another man may have the same motility percentage but:

- very low sperm count,
- severe abnormal morphology,
- a clinically significant varicocele,
- or another reproductive problem.

His situation is completely different.

This is why my approach at **Saira Health Care** is never:

“Motility is low, therefore take one sperm-motility medicine.”

Instead, I ask:

Why is motility low?

Is the result genuine and persistent?

How many sperm are moving progressively?

How many sperm are alive?

Is the count also low?



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Is morphology abnormal?

Is there a varicocele?

Could infection, oxidative stress, fever, medication, obesity, smoking or a genetic flagellar disorder be contributing?

And finally:

What is the fertility situation of the couple as a whole?

That is the foundation of rational male-infertility treatment.

What Is Sperm Motility?

Sperm motility refers to the ability of spermatozoa to move.

But all movement is not equally useful.

A sperm that is simply vibrating or moving in small circles is different from a sperm that is making effective forward progress.

Modern semen analysis therefore evaluates the **quality and direction of movement**, not just whether sperm are moving.

According to current WHO methodology, sperm movement is broadly described as:

Progressive Motility – PR

The sperm moves actively forward.

This is generally the most relevant movement for natural fertility.

Non-Progressive Motility – NP

The sperm is moving, but there is little or no meaningful forward progression.

Immotile – IM

The sperm shows no movement.

The WHO Sixth Edition provides standardized laboratory procedures for assessing these semen characteristics. (who.int)

Current WHO Reference Values for Sperm Motility

Older websites and laboratory reports sometimes continue to use outdated reference values.



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Current WHO Sixth Edition values, which are also incorporated into the **March 2026 NICE fertility guideline**, include approximately:

- **Semen volume:** 1.4 mL or more
- **Sperm concentration:** 16 million/mL or more
- **Total sperm number:** 39 million per ejaculate or more
- **Total motility:** 42% or more
- **Progressive motility:** 30% or more
- **Vitality:** 54% or more live sperm
- **Normal morphology:** 4% or more

([nice.org.uk](https://www.nice.org.uk))

Therefore, as a practical contemporary reference:

Progressive motility around 30% or above

and

Total motility around 42% or above

fall within the current lower reference range.

However, these figures require careful interpretation.

They are **statistical reference values, not absolute boundaries between fertile and infertile men.**

Does Progressive Motility Below 30% Mean Infertility?

No.

This is one of the first misconceptions I want to correct.

A semen report showing:

Progressive motility – 27%

does not automatically mean natural pregnancy is impossible.

Likewise, a man with:

Progressive motility – 40%

is not automatically fertile.

Fertility depends on the complete reproductive picture.



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The important variables include:

- sperm concentration,
- total sperm count,
- progressive motility,
- total motility,
- morphology,
- vitality,
- total motile sperm number,
- semen volume,
- frequency and timing of intercourse,
- and female-partner fertility.

International male-infertility guidelines emphasize that no single semen parameter alone can determine whether an individual man is fertile or infertile. (auajournals.org)

Therefore:

Do not allow one motility percentage to define your entire fertility future.

What Is Asthenozoospermia?

Asthenozoospermia refers to an abnormally low proportion of progressively motile sperm.

In practical contemporary interpretation, significant reduction in progressive motility below the laboratory's WHO-based reference range may be described as asthenozoospermia.

Some European clinical classifications have historically used approximately **32% progressive motility** as a practical threshold, while current WHO Sixth Edition/NICE reference data use approximately **30% progressive motility**. (uroweb.org)

The exact percentage should therefore be interpreted using the methodology and reference standards of the laboratory.

More importantly:

Persistent abnormality is more meaningful than one isolated abnormal report.

Sperm Motility Is Different From Sperm Count



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This distinction is extremely important.

A patient's report may show:

Good count, poor motility

For example:

- sperm concentration: 60 million/mL
- progressive motility: 18%

This is primarily a motility problem.

Another patient may have:

Low count, good motility

For example:

- sperm concentration: 7 million/mL
- progressive motility: 45%

This is primarily an oligozoospermia problem.

Another patient may have:

Low count + poor motility

This is called:

Oligoasthenozoospermia

And another may simultaneously have:

- low sperm count,
- poor motility,
- abnormal morphology.

This is called:

Oligo-Asthen-Teratozoospermia – OAT

These different patterns should not receive identical treatment.

Sperm Motility Is Also Different From Sperm Morphology

Morphology means sperm shape.

Motility means sperm movement.



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A sperm may have:

- normal shape but poor movement,
- abnormal shape but good movement,
- or abnormalities in both.

Tail or midpiece defects can sometimes contribute to poor movement, but morphology and motility remain different semen parameters.

A complete semen analysis is therefore much more useful than concentrating on only one number.

Motility Is Different From Vitality

This is another extremely important concept.

A sperm that is not moving is **not necessarily dead**.

Some sperm are:

Alive but immotile.

Other sperm are:

Dead and therefore immotile.

The test used to differentiate these situations is called:

Sperm vitality testing.

The current lower WHO reference value for vitality is approximately:

54% live spermatozoa. ([nice.org.uk](https://www.nice.org.uk))

This distinction becomes particularly important when there is severe or complete sperm immotility.

What Is Necrozoospermia?

Necrozoospermia refers to an abnormally high proportion of dead sperm.

It is different from asthenozoospermia.

Asthenozoospermia

Sperm movement is reduced.

Necrozoospermia



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Sperm vitality or survival is reduced.

Some men may have both.

This distinction can become particularly important during assisted reproductive treatment because an embryologist may need to identify:

living but non-moving sperm

for ICSI.

Therefore, a report stating:

“0% motility”

requires more careful evaluation than simply concluding:

“all sperm are dead.”

Why Does Sperm Need to Move?

To understand asthenozoospermia, it helps to understand the biological journey of sperm.

After ejaculation, sperm must pass through a complex environment.

Successful sperm need to:

- survive after ejaculation,
- move through cervical mucus,
- pass into the uterus,
- progress toward the fallopian tube,
- undergo biochemical activation known as capacitation,
- develop specialized movement called hyperactivation,
- interact with the egg,
- and participate in fertilization.

The sperm tail—or **flagellum**—provides the mechanical movement required for propulsion.

A major 2025 review of human asthenozoospermia explains that normal sperm motility depends on highly coordinated flagellar structures, energy metabolism, ion channels and signalling pathways. (pubmed.ncbi.nlm.nih.gov)

Therefore, low motility is not caused by only one mechanism.



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How Does a Sperm Produce Energy for Movement?

Sperm movement requires energy in the form of ATP.

This energy is generated through pathways involving:

- mitochondria in the midpiece,
- glycolysis,
- and oxidative phosphorylation.

Sperm also rely on specialized:

- ion channels,
- signalling proteins,
- structural proteins,
- and flagellar motor proteins.

Modern genetic research has shown that abnormalities in any of these systems can produce asthenozoospermia. ([pmc.ncbi.nlm.nih.gov](https://pubmed.ncbi.nlm.nih.gov))

This is one reason why severe asthenozoospermia should not always be treated as simple “weak sperm.”

Symptoms of Asthenozoospermia

Asthenozoospermia usually causes:

No obvious physical symptom.

A man usually cannot feel that his sperm are moving slowly.

He may have completely normal:

- libido,
- erection,
- intercourse,
- orgasm,
- ejaculation,
- semen volume,
- semen appearance,
- testicular appearance,



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- and general health.

The most common way asthenozoospermia is discovered is during:

infertility investigation.

A couple may have been having regular unprotected intercourse but pregnancy has not occurred.

The semen analysis then reveals reduced progressive motility.

Does Thin Semen Mean Poor Motility?

No.

Many patients tell me:

“Doctor, my semen looks watery. That must mean my sperm are weak.”

This is not medically reliable.

Semen consistency and sperm movement are different characteristics.

A man may have:

- relatively thin semen with excellent sperm motility,

or:

- thick semen with very poor sperm motility.

Only microscopic semen analysis can assess sperm movement.

Does Thick Semen Mean Strong Sperm?

Again, no.

Thick semen cannot prove:

- high sperm count,
- good motility,
- normal morphology,
- good DNA quality,
- or fertility.

Semen appearance should not replace laboratory evaluation.



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Does Asthenozoospermia Cause Erectile Dysfunction?

Not directly.

Asthenozoospermia is primarily a **fertility problem**.

Erectile dysfunction is primarily a **sexual-function problem**.

A man may have poor sperm motility but completely normal erections.

Likewise, a man may have excellent semen parameters but severe erectile dysfunction.

Because my focused practice includes both **sexual disorders and infertility**, I consider both areas during assessment, but they should not be confused.

Causes of Decreased Sperm Motility

Asthenozoospermia can result from many different factors.

Important causes and associations include:

- varicocele,
- oxidative stress,
- smoking,
- obesity,
- diabetes and metabolic problems,
- infection and inflammation,
- fever,
- genetic abnormalities,
- structural defects of the sperm flagellum,
- mitochondrial dysfunction,
- hormonal disorders,
- testicular disease,
- undescended testes,
- environmental toxins,
- radiation,



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- heat exposure,
- heavy alcohol intake,
- anabolic steroids,
- certain medications,
- and unexplained or idiopathic factors.

Let us understand these in detail.

1. Oxidative Stress: An Important Mechanism

One of the most actively studied mechanisms in asthenozoospermia is:

Oxidative stress.

Sperm naturally produce and encounter reactive oxygen species, commonly called ROS.

Small physiological amounts of ROS are actually necessary for normal sperm function, including:

- maturation,
- capacitation,
- acrosome reaction,
- and fertilization.

The problem occurs when ROS become excessive.

A 2025 review focusing specifically on asthenozoospermia describes how excessive oxidative stress can damage:

- sperm-cell membranes,
- mitochondrial function,
- proteins,
- enzymes,
- and DNA,

ultimately impairing motility. (pubmed.ncbi.nlm.nih.gov)

This is one reason antioxidants have attracted considerable interest in male-infertility treatment.



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However, as I explain later, understanding oxidative stress does **not** mean every patient should automatically take multiple antioxidant supplements.

Why Are Sperm So Sensitive to Oxidative Stress?

Sperm membranes contain large amounts of polyunsaturated fatty acids.

These are important for membrane flexibility but also make sperm particularly vulnerable to **lipid peroxidation**.

Excessive ROS can damage these membranes.

When the membrane and mitochondria are damaged:

- sperm may lose energy,
- tail movement becomes inefficient,
- motility falls,
- DNA damage may increase,
- and fertilizing ability can decrease.

Sperm also have comparatively limited repair mechanisms once mature.

This makes prevention of excessive oxidative damage especially important.

2. Varicocele

A varicocele is an enlargement of veins around the testis.

It is one of the more important potentially correctable male-fertility conditions.

Varicocele may impair sperm quality through several mechanisms, including:

- increased scrotal temperature,
- oxidative stress,
- inflammation,
- altered testicular blood flow,
- and impaired testicular function.

Recent research continues to identify oxidative stress and inflammation as important mechanisms linking varicocele with abnormal semen parameters.

(pubmed.ncbi.nlm.nih.gov)



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Varicocele can affect:

- sperm count,
- motility,
- morphology,
- and DNA integrity.

Does Every Varicocele Need Surgery?

No.

Current AUA/ASRM guidance recommends considering varicocele repair particularly in men who:

- are attempting conception,
- have infertility,
- have a **palpable clinical varicocele**,
- and have abnormal semen parameters.

The guideline specifically recommends **against routine surgery for a non-palpable varicocele detected only on imaging.** (auajournals.org)

Therefore:

Low motility + ultrasound varicocele

does not automatically mean:

surgery is required.

The varicocele must be evaluated clinically.

Can Varicocele Surgery Improve Motility?

In appropriately selected infertile men with clinical varicocele, semen parameters can improve following repair.

However:

- improvement is not guaranteed,
- treatment response takes time,
- and pregnancy depends on both partners.



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The decision should consider:

- severity of semen abnormalities,
 - female-partner age,
 - ovarian reserve,
 - duration of infertility,
 - and whether ART is already needed.
-

3. Smoking

Smoking is an important modifiable reproductive-health factor.

Tobacco smoke exposes sperm and reproductive tissues to:

- nicotine,
- cadmium,
- lead,
- and numerous oxidants.

A 2025 asthenozoospermia review highlights smoking as an important source of oxidative stress associated with reduced sperm motility. ([pmc.ncbi.nlm.nih.gov](https://pubmed.ncbi.nlm.nih.gov))

For a patient with poor motility who smokes regularly, one of my strongest recommendations is:

Stop smoking.

Giving fertility medicines while continuing substantial tobacco exposure is not a sensible long-term strategy.

4. Obesity

Obesity can affect male reproductive health through:

- hormonal changes,
- chronic inflammation,
- oxidative stress,
- increased scrotal temperature,
- insulin resistance,



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- sleep disturbance,
- and associated metabolic disease.

Weight reduction in an overweight or obese man may therefore form an important part of fertility management.

However, the goal should be:

healthy gradual weight management

rather than extreme dieting.

5. Diabetes and Metabolic Disease

Diabetes can affect:

- reproductive hormones,
- oxidative stress,
- nerve function,
- ejaculation,
- erections,
- and sperm quality.

A man with:

poor motility + uncontrolled diabetes

should not be treated simply with a reproductive tonic while glucose remains uncontrolled.

General health and fertility health are closely connected.

This is an area where modern metabolic care and the holistic principles of Unani medicine can work particularly well together.

6. Infection and Inflammation

Inflammation in the male reproductive tract can increase the production of reactive oxygen species.

Possible conditions include:

- prostatitis,
- epididymal inflammation,



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- and other reproductive-tract infections.

Activated white blood cells can produce large amounts of ROS, creating an environment that may impair sperm movement. ([pmc.ncbi.nlm.nih.gov](https://pubmed.ncbi.nlm.nih.gov))

However:

Low motility is not an indication for automatic antibiotics.

Antibiotic treatment is appropriate when a clinically meaningful infection has been identified.

Unnecessary antibiotics:

- do not treat idiopathic asthenozoospermia,
- can cause side effects,
- and contribute to antimicrobial resistance.

7. High Fever

Spermatogenesis is sensitive to heat.

A significant febrile illness may temporarily impair:

- sperm concentration,
- motility,
- and morphology.

Because sperm production takes several weeks, a high fever may affect semen analysis even after the patient has recovered clinically.

Whenever I see a newly abnormal semen report, I ask:

“Did you have high fever or a major illness during the last two to three months?”

A temporary post-febrile abnormality should not automatically be treated as permanent infertility.

8. Excessive Testicular Heat

The testes are anatomically located outside the abdomen partly because sperm production functions better at temperatures below core body temperature.

Repeated substantial heat exposure may affect spermatogenesis in susceptible men.



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Examples can include:

- occupational heat exposure,
- frequent prolonged hot tubs or very hot baths,
- or other chronic heat sources.

However, internet advice often exaggerates this issue.

Normal clothing or ordinary day-to-day heat does not automatically cause infertility.

The focus should be on **repeated significant exposure**, particularly when semen parameters are already abnormal.

9. Alcohol

Heavy chronic alcohol intake can adversely affect:

- hormonal health,
- liver function,
- body weight,
- sleep,
- sexual health,
- and potentially sperm quality.

Current male-fertility guidance recommends reducing high alcohol intake as part of fertility optimization.

Occasional alcohol exposure and severe chronic alcohol use should not be treated as though they have identical effects.

10. Anabolic Steroids and Testosterone

This is one of the most important warnings in male reproductive medicine.

A man may take:

- testosterone injections,
- bodybuilding steroids,
- testosterone gels,
- or other anabolic-androgenic drugs



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because he wants:

- larger muscles,
- greater strength,
- or improved sexual vitality.

These products can severely suppress sperm production.

External testosterone reduces the brain's production of:

- LH,
- FSH,

which are essential for normal intratesticular testosterone levels and spermatogenesis.

Sperm count may fall dramatically, sometimes to zero.

Current AUA/ASRM guidance explicitly states:

Testosterone monotherapy should not be prescribed to men interested in current or future fertility. (prod.asrm.org)

Therefore, always tell your physician if fatherhood is an immediate or future goal before beginning testosterone treatment.

11. Hormonal Disorders

Sperm production depends on the coordinated action of:

- hypothalamus,
- pituitary gland,
- testes.

Important hormones include:

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

Hormonal problems can impair the development and maturation of sperm.

However, isolated poor motility does not always require an extensive hormonal panel.

Hormonal testing is particularly important when poor motility occurs together with:



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- low sperm count,
- small testes,
- reduced libido,
- erectile problems,
- abnormal secondary sexual characteristics,
- or other endocrine clues.

12. Genetic Causes of Severe Asthenozoospermia

One of the most important advances in recent male-fertility research is our understanding of **genetic sperm-motility disorders**.

Sperm movement depends on the structure and function of the flagellum.

Dozens of proteins must work together correctly.

Modern studies have identified mutations involving:

- flagellar structural proteins,
- dynein motor proteins,
- ion transporters,
- mitochondrial proteins,
- cytoskeletal proteins,
- and sperm-activation pathways.

A major 2025 review summarizes many newly recognized genetic causes of human asthenozoospermia. (pubmed.ncbi.nlm.nih.gov)

This becomes particularly relevant when:

- motility is extremely low,
- nearly all sperm are immotile,
- tail morphology is severely abnormal,
- the condition is persistent,
- or there is a suggestive family or medical history.

Multiple Morphological Abnormalities of the Sperm Flagella – MMAF



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Some men have a severe condition known as:

Multiple Morphological Abnormalities of the Sperm Flagella
or **MMAF**.

Sperm tails may be:

- absent,
- short,
- coiled,
- bent,
- or irregular.

This can cause:

- severe asthenozoospermia,
- or asthenoteratozoospermia.

A 2025 study, for example, identified an **AK7 gene mutation** associated with characteristic MMAF and severe male infertility. (pubmed.ncbi.nlm.nih.gov)

These cases require much more than routine antioxidant treatment.

Genetic counselling and specialized assisted-reproduction assessment may be necessary.

Primary Ciliary Dyskinesia and Sperm Motility

Some genes involved in sperm-tail movement are also involved in the movement of cilia in the respiratory system.

Therefore, in rare patients, severe or complete sperm immotility may coexist with:

- chronic sinus problems,
- recurrent chest infections,
- bronchiectasis,
- or other ciliary disorders.

A detailed history can therefore provide an important clue.

Not every man with low motility has a genetic disease.

But when motility is extremely poor and persistent, the possibility deserves consideration.



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13. Mitochondrial Dysfunction

The sperm midpiece contains mitochondria.

These structures contribute to the energy required for movement.

Recent genetic and biological research has identified abnormalities in:

- mitochondrial structure,
- mitochondrial DNA,
- energy pathways,
- and mitochondrial proteins

among mechanisms contributing to asthenozoospermia. (pmc.ncbi.nlm.nih.gov)

This explains why sperm motility should not simply be described as:

“strength.”

It is an extremely complex cellular process.

14. Environmental Toxins

Occupational and environmental exposure may include:

- pesticides,
- solvents,
- heavy metals,
- industrial pollutants,
- radiation,
- and endocrine-disrupting chemicals.

Such exposures may increase oxidative stress or interfere with spermatogenesis.

The actual risk depends on:

- dose,
- duration,
- individual susceptibility,
- and protective measures.



I therefore include occupation and exposure history in male-fertility consultation where relevant.

15. Testicular Disorders

Conditions affecting testicular function may reduce overall semen quality.

Examples include:

- undescended testes,
- testicular injury,
- torsion,
- mumps orchitis,
- previous surgery,
- chemotherapy,
- radiation.

These conditions may affect count and morphology in addition to motility.

16. Age

Male fertility does not stop abruptly at a particular age.

However, increasing paternal age can be associated with changes in:

- semen quality,
- DNA fragmentation,
- reproductive hormones,
- and other fertility variables.

Age therefore forms part of the complete fertility assessment.

17. Idiopathic Asthenozoospermia

Sometimes no definite cause can be identified despite appropriate evaluation.

This is described as:

Idiopathic asthenozoospermia.



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This does not mean:

“Nothing is wrong.”

It means:

routine clinical testing has not identified the biological cause.

Research suggests that some idiopathic cases may involve:

- oxidative stress,
- subtle genetic factors,
- mitochondrial dysfunction,
- sperm-signalling abnormalities,
- or mechanisms not detected by routine semen testing.

Recent 2025 research continues to focus strongly on oxidative stress as one potential mechanism in idiopathic asthenozoospermia. (pubmed.ncbi.nlm.nih.gov)

Diagnosis of Asthenozoospermia

The foundation of diagnosis is:

A Proper Semen Analysis

The semen report should ideally include:

- volume,
- sperm concentration,
- total sperm number,
- progressive motility,
- total motility,
- morphology,
- vitality where indicated,
- and other relevant microscopic findings.

The WHO Sixth Edition provides standardized procedures intended to improve laboratory consistency. (who.int)

Why Laboratory Quality Matters



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Motility is time-sensitive.

Sperm movement can deteriorate if:

- the specimen becomes too cold,
- examination is substantially delayed,
- the sample is contaminated,
- collection is incomplete,
- or laboratory handling is poor.

Therefore, an apparently poor motility result is meaningful only if the sample was collected and processed appropriately.

This is why I pay attention to:

- laboratory quality,
- collection method,
- transport conditions,
- and time to analysis.

Should the Semen Analysis Be Repeated?

Yes, when the first result is abnormal, repeat testing is commonly appropriate.

The **March 2026 NICE fertility guideline** recommends a repeat confirmatory semen analysis after an abnormal initial result, ideally around three months later to allow the sperm-production cycle to be completed, although severe abnormalities may justify earlier confirmation. ([nice.org.uk](https://www.nice.org.uk))

This is important because sperm motility can change between samples.

One bad report after:

- fever,
- incomplete collection,
- major stress,
- or laboratory delay

should not automatically define a man's permanent fertility status.



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How Long Does Sperm Production Take?

Human sperm development takes approximately two and a half months, followed by additional maturation.

This is why I generally do not expect a biologically meaningful change in semen quality after only:

- five days,
- ten days,
- or two weeks.

When we correct a reversible cause today, the sperm tested months later may better reflect the improved environment.

This biological delay is important when assessing any:

- medical treatment,
- lifestyle treatment,
- or Unani programme.

Physical Examination

A male-fertility assessment may include evaluation of:

- testicular size,
- testicular consistency,
- epididymis,
- vas deferens,
- varicocele,
- secondary sexual characteristics,
- and relevant genital abnormalities.

A semen report cannot provide this information.

Current AUA/ASRM guidance recommends complete history and physical evaluation for men with abnormal semen parameters or presumed male infertility. (auajournals.org)

Hormonal Evaluation



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Depending on the semen profile and symptoms, tests may include:

- FSH,
- LH,
- morning testosterone,
- prolactin,
- and other hormones when indicated.

If only motility is mildly reduced while count and clinical examination are normal, extensive hormone testing may not always be necessary.

Investigation should be individualized.

Ultrasound

Scrotal ultrasound may be useful when:

- examination is unclear,
- testicular abnormalities are suspected,
- pain or swelling is present,
- or varicocele requires clarification.

However, an ultrasound finding should always be interpreted alongside clinical examination.

A tiny subclinical varicocele does not automatically explain poor sperm motility.

Vitality Testing in Severe Immotility

If motility is extremely poor, particularly when a large proportion of sperm are completely immotile, vitality testing becomes especially useful.

The key question is:

Are the sperm alive but unable to move, or are they dead?

This can directly influence:

- diagnosis,
- prognosis,
- and sperm-selection strategy during ART.



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Genetic Evaluation

Routine genetic testing is not necessary for every man with:

progressive motility 25%.

However, specialized genetic evaluation may be appropriate when there is:

- near-total sperm immotility,
- severe flagellar abnormalities,
- MMAF,
- suspected primary ciliary dyskinesia,
- severe asthenoteratozoospermia,
- or a suggestive family history.

A 2025 review describes rapidly expanding knowledge of genes associated with both structural and functional asthenozoospermia. (pubmed.ncbi.nlm.nih.gov)

Sperm DNA Fragmentation

Poor motility and DNA fragmentation can sometimes coexist, particularly in conditions associated with oxidative stress.

However:

Low motility does not automatically mean high DNA fragmentation.

Current male-infertility guidelines generally reserve sperm DNA-fragmentation testing for selected situations, such as:

- recurrent pregnancy loss,
- repeated ART failure,
- or certain unexplained infertility cases.

It should not be ordered automatically for every asthenozoospermia patient.

Modern Treatment of Decreased Sperm Motility

There is no single medicine that is appropriate for every case.

Treatment should be divided into:

1. treating identifiable causes,



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2. reducing reversible reproductive risks,
 3. optimizing general health,
 4. using selected medical therapy when appropriate,
 5. considering ART according to the couple's circumstances.
-

1. Stop Smoking

For a smoker with impaired sperm motility, smoking cessation is one of the most important changes.

This can reduce exposure to:

- oxidants,
- heavy metals,
- nicotine,
- and other toxic substances.

The benefit extends far beyond fertility to:

- cardiovascular health,
 - cancer risk,
 - erectile function,
 - and general health.
-

2. Maintain a Healthy Body Weight

Obesity should be addressed using:

- a balanced diet,
- regular exercise,
- adequate sleep,
- and appropriate medical management.

Weight loss should not become extreme.

My goal is:

better metabolic health, not simply a lower number on the weighing scale.



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3. Improve Diabetes and Metabolic Control

If the patient has:

- diabetes,
- insulin resistance,
- dyslipidemia,
- or metabolic syndrome,

these conditions should be treated appropriately.

The reproductive system should not be treated as if it is separate from the rest of the body.

4. Avoid Testosterone and Anabolic Steroids

If fatherhood is desired, fertility-suppressing androgen use requires proper evaluation.

Some men recover sperm production after discontinuation, but recovery can take time.

Selected patients may require specialist hormonal treatment.

Do not attempt to reverse steroid-related infertility by adding random supplements while continuing anabolic steroids.

5. Treat Clinical Varicocele When Indicated

In an infertile man with:

- palpable varicocele,
- abnormal semen parameters,
- and appropriate couple-level circumstances,

varicocele repair may be considered.

Current AUA/ASRM guidance supports this approach. (auajournals.org)

But:

Subclinical ultrasound varicocele alone is not an indication for surgery.

6. Treat Proven Infection



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If infection is identified, cause-specific treatment should be given.

The goal is to treat the disease.

It is not appropriate to give repeated antibiotics simply because sperm are slow.

7. Treat Hormonal Disorders

When a genuine endocrine abnormality is responsible for impaired spermatogenesis, cause-specific hormonal treatment may be appropriate.

Examples can include selected treatment of:

- hypogonadotropic hypogonadism,
- hyperprolactinemia,
- or other diagnosed endocrine conditions.

Fertility-preserving hormonal treatment must be distinguished from testosterone replacement.

8. Lifestyle Optimization

I commonly advise:

- adequate sleep,
- regular moderate exercise,
- avoiding recreational drugs,
- limiting heavy alcohol consumption,
- avoiding chronic excessive heat,
- improving nutritional quality,
- reducing unnecessary environmental toxin exposure,
- and managing chronic stress.

These changes should not be advertised as guaranteed cures.

They create a healthier biological environment for sperm production.

Are Antioxidants Effective for Low Sperm Motility?

This is one of the most debated subjects in contemporary male-infertility care.



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The biological argument is strong.

Excessive oxidative stress can clearly impair sperm motility.

And many trials have reported improvements in semen parameters after antioxidant supplementation.

A large **2026 systematic review and meta-analysis involving 101 studies and 11,889 infertile men** found higher:

- progressive motility,
- total motility,
- sperm concentration,
- and morphology

among antioxidant-treated groups.

However, despite these improvements, the authors concluded that **no definitive recommendation could be made for antioxidants as male-infertility treatment** because of major differences between studies, formulations and methods. (pubmed.ncbi.nlm.nih.gov)

Another 2026 meta-analysis of 41 randomized trials found positive average effects on progressive and total motility but rated the certainty of evidence as very low, with substantial heterogeneity. (pubmed.ncbi.nlm.nih.gov)

A 2026 clinical review similarly concluded that selected antioxidants may be reasonable adjuncts in carefully selected men, but improvements in semen measurements do not consistently translate into higher pregnancy or live-birth rates. (pubmed.ncbi.nlm.nih.gov)

This is why current AUA/ASRM guidance states that the clinical benefit of fertility supplements such as antioxidants and vitamins remains uncertain and that evidence is inadequate to recommend one specific agent universally. (prod.asrm.org)

My approach is therefore:

Use supplements selectively—not blindly.

What About L-Carnitine?

L-carnitine and acetyl-L-carnitine are widely used in male-infertility treatment because sperm and epididymal tissues contain high carnitine concentrations and the compound plays a role in cellular energy metabolism.

Some meta-analyses have reported improvement in sperm motility.



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For example, a network meta-analysis of randomized trials reported favorable effects of L-carnitine-containing approaches on certain semen parameters. However, the same analysis did not demonstrate a clear improvement in pregnancy rates compared with placebo.

(pubmed.ncbi.nlm.nih.gov)

Therefore:

better motility on a semen report is encouraging, but pregnancy and live birth remain the outcomes that matter most.

Can IUI Help With Poor Sperm Motility?

Sometimes.

Intrauterine insemination—or **IUI**—involves processing the semen sample and concentrating motile sperm before placing them into the uterus around ovulation.

It may be considered in selected couples with:

- mild male-factor infertility,
- adequate total motile sperm,
- and suitable female-partner fertility.

However, when the number of motile sperm available after processing becomes very low, the chances of success decline.

The decision should therefore be based on:

- total motile sperm count,
 - female age,
 - ovarian reserve,
 - tubal status,
 - infertility duration,
 - and previous treatment.
-

IVF and ICSI

For more severe male-factor infertility, assisted reproduction may involve:

- IVF,
- or ICSI.



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With conventional IVF, sperm and eggs are placed together and fertilization is allowed to occur.

With:

Intracytoplasmic Sperm Injection – ICSI

an embryologist selects an individual sperm and injects it directly into the egg.

ICSI can be particularly valuable when:

- sperm motility is severely reduced,
- very few usable sperm are available,
- fertilization has previously failed,
- or another severe male-factor disorder is present.

However:

Every man with progressive motility below 30% does not automatically require ICSI.

The complete couple-level fertility situation matters.

What Happens When Sperm Are Completely Immotile?

When essentially no sperm are moving, the challenge is identifying which sperm are alive.

Special laboratory techniques may be used to select viable sperm for ICSI.

In selected severe genetic motility disorders, ICSI can sometimes bypass the inability of sperm to travel naturally through the female reproductive tract.

But outcomes depend on the underlying genetic defect and sperm viability.

This is another reason why:

0% motility

requires specialist evaluation rather than simple classification as:

“weak sperm.”

Asthenozoospermia and Natural Pregnancy

Natural conception may still occur with reduced motility.

The likelihood depends on:

- how low motility is,



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- total sperm count,
- total motile sperm number,
- morphology,
- semen volume,
- sexual timing,
- female age,
- ovulation,
- tubal health,
- and duration of infertility.

A mildly reduced motility result in a young couple with otherwise normal fertility factors is very different from severe asthenozoospermia combined with:

- low sperm count,
- abnormal morphology,
- and reduced ovarian reserve in the female partner.

Why the Female Partner Must Also Be Evaluated

Male fertility cannot be treated in isolation.

Current AUA/ASRM guidance recommends concurrent evaluation of male and female partners at the beginning of infertility assessment. (auajournals.org)

Imagine two men both have:

Progressive motility – 20%.

Couple A

The wife is:

- 25 years old,
- ovulating normally,
- with healthy tubes and good ovarian reserve.

Couple B

The wife is:

- 39 years old,



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- with reduced ovarian reserve.

The same semen number should not automatically produce the same treatment plan.

In Couple B, spending one or two years trying multiple supplements simply to raise motility may waste important reproductive time.

The correct treatment is always:

couple-centered.

Asthenozoospermia and Unani Medicine

As a physician trained in Unani medicine, I consider male reproductive health within the broader context of:

- Mizaj,
- general health,
- nutrition,
- digestion,
- physical activity,
- sleep,
- psychological well-being,
- metabolic health,
- reproductive-organ function,
- and environmental influences.

Classical Unani medicine developed long before sperm motility could be examined under the microscope.

Therefore, there is an important academic distinction.

Modern asthenozoospermia is a laboratory-defined condition.

Classical Unani medicine did not define:

“progressive motility below 30%.”

Traditional Unani physicians instead described disorders of:

- Mani,
- reproductive capacity,



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- testicular function,
- reproductive weakness,
- and general physiological balance.

Today, I believe the most responsible approach is:

Combine modern semen analysis with individualized Unani assessment.

This allows us to preserve the holistic strength of Unani medicine without ignoring modern reproductive biology.

Mizaj and Male Fertility

Mizaj, meaning temperament, is a foundational concept in Unani medicine.

Traditional assessment considers the individual's:

- physical constitution,
- functional characteristics,
- nutritional condition,
- psychological state,
- and other clinical features.

The Ministry of AYUSH describes Unani medicine as an individualized system in which Mizaj is important to health assessment and treatment planning. (ayush.gov.in)

This individualized philosophy is particularly useful in male infertility.

Two men may both show:

progressive motility 20%.

But one may have:

- varicocele.

Another may have:

- obesity, smoking and uncontrolled diabetes.

Another may have:

- severe genetic flagellar disease.

The semen number is identical.

The disease is not.



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Akhlat and the Traditional Unani Framework

Classical Unani medicine traditionally describes four humors:

- **Dam**
- **Balgham**
- **Safra**
- **Sauda**

and considers physiological balance important for normal organ function.

However, I explain to patients that:

Unani humoral terminology is a traditional medical framework.

It should not be confused with modern measurable factors such as:

- FSH,
- LH,
- testosterone,
- sperm motility,
- sperm DNA,
- chromosomes,
- or oxidative stress biomarkers.

Respecting traditional medicine does not require us to pretend that classical concepts and modern laboratory variables are identical.

Asbab-e-Sitta Zarooriya and Sperm Motility

One of the Unani concepts that fits particularly well with modern reproductive-health care is:

Asbab-e-Sitta Zarooriya

or the six essential factors.

These traditionally include:

1. air and environment,
2. food and drink,



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3. physical activity and rest,
4. psychological activity and rest,
5. sleep and wakefulness,
6. retention and elimination.

Official AYUSH material continues to identify these factors as important principles of health preservation in Unani medicine.

Several have obvious relevance to modern male-fertility care.

For example:

Air and Environment

Environmental toxins, occupational chemicals and smoking can affect reproductive health.

Food and Drink

Nutrition, obesity, alcohol intake and metabolic health can affect sperm quality.

Physical Activity and Rest

Appropriate exercise supports metabolic and cardiovascular health.

Psychological Activity and Rest

Infertility itself creates substantial emotional stress.

Sleep and Wakefulness

Poor sleep can affect hormonal and metabolic health.

These traditional lifestyle principles therefore provide a useful framework for comprehensive reproductive care.

Four Major Modes of Unani Treatment

Unani medicine traditionally describes four major therapeutic approaches:

Ilaj-bil-Ghiza

Dietotherapy

Ilaj-bit-Tadbir

Regimenal therapy

Ilaj-bid-Dawa

Pharmacotherapy



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Ilaj-bil-Yad

Surgical treatment

These approaches can be integrated according to the actual cause of the patient's infertility.

Ilaj-bil-Ghiza: Dietary Management for Poor Motility

My dietary approach aims to create a healthy environment for sperm production and general reproductive function.

I may evaluate:

- body weight,
- protein intake,
- vegetable intake,
- fruit intake,
- healthy fats,
- micronutrient adequacy,
- diabetes,
- digestive health,
- processed foods,
- hydration,
- alcohol,
- smoking,
- and metabolic health.

I do **not** tell patients:

“Eat one particular food and sperm motility will become 60%.”

No diet can guarantee a specific motility result.

The goal is broader:

support reproductive physiology and general health.

Ilaj-bit-Tadbir: Regimenal and Lifestyle Management



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Depending on the patient, Unani regimenal care may emphasize:

- appropriate exercise,
- healthy sleep,
- rest,
- stress reduction,
- weight management,
- and other individualized lifestyle modifications.

These measures fit particularly well into modern management when:

- obesity,
- inactivity,
- chronic stress,
- sleep disturbance,
- or metabolic problems

are present.

What About Hijama or Cupping?

Hijama has an established historical place within Unani regimenal therapy for selected conditions.

However:

I do not consider Hijama a scientifically proven stand-alone cure for asthenozoospermia.

At present, high-quality evidence showing that Hijama reliably raises progressive sperm motility or improves live-birth outcomes is insufficient.

If used in selected patients, it should be regarded as:

- supportive regimenal care,

not a replacement for:

- semen analysis,
- varicocele treatment,
- hormonal management,
- genetic evaluation,



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- IVF,
- or ICSI.

Ilaj-bid-Dawa: Individualized Unani Pharmacotherapy

This is an important area of male-infertility management at Saira Health Care.

Unani medicines may traditionally be selected according to:

- Mizaj,
- reproductive symptoms,
- semen profile,
- general vitality,
- nutrition,
- digestive state,
- metabolic health,
- sexual function,
- and associated conditions.

I do not support a rigid idea that:

“Every man with motility below 30% should take exactly the same medicine.”

That is not individualized medicine.

Scientific Research on Unani Medicine and Sperm Motility

There is published Unani clinical literature evaluating traditional formulations in men with male infertility.

A CCRUM-associated publication reported an open clinical study in oligospermic men in which the test formulation was associated with improvement in:

- sperm count,
- and motility.

The investigators also acknowledged important limitations and called for larger and longer studies. (ccrum.res.in)

I consider this encouraging.



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It suggests that traditional Unani male-fertility formulations deserve further serious clinical research.

However, it does **not** prove that every Unani formulation cures isolated asthenozoospermia.

And it does not establish a guaranteed pregnancy or live-birth rate.

The correct conclusion is:

Unani reproductive medicine has promising clinical observations, but stronger modern randomized research is still needed.

Traditional Herbal Medicine and Motility: What Broader Research Shows

There is also research from other traditional systems examining herbal formulations for asthenozoospermia.

Some randomized studies have reported improvement in progressive motility.

However, these should not be automatically presented as evidence for Unani formulations because:

Traditional Chinese Medicine is not the same as Unani Medicine.

Likewise, evidence for one specific standardized herbal formulation cannot automatically be transferred to another product.

This is an important scientific distinction.

Spermogenic Powder in Saira Health Care's Male-Fertility Practice

Saira Health Care has traditionally used and published information about **Spermogenic Powder** as part of its male reproductive-health programme.

Saira Health Care's public material describes Spermogenic as a traditional fertility-support formulation used particularly for semen-quality concerns, including sperm motility.

(sairahealthcare.com)

Within my clinical approach, such a formulation should never be prescribed merely because a report shows:

“Motility low.”

I consider the entire patient's condition.

When Spermogenic is used, I regard it as part of an individualized Unani supportive programme addressing:



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- semen quality,
- reproductive nutrition,
- general vitality,
- and male reproductive health.

I do not believe it is medically appropriate to promise:

“Spermogenic will cure every case of asthenozoospermia.”

There is currently insufficient product-specific controlled clinical evidence to make such a universal claim.

This distinction protects patients and strengthens the credibility of responsible Unani practice.

Dr. Qasmi's Nuskha No. 129

Dr. Qasmi's Nuskha No. 129 is another formulation used within Saira Health Care's male reproductive-health practice.

Saira Health Care's existing published material describes Nuskha No. 129 as a traditional formulation used to support:

- semen quality,
- sperm production,
- and broader male reproductive health. (sairahealthcare.com)

In clinical practice, I consider it within the context of:

- semen findings,
- Mizaj,
- reproductive history,
- associated sexual symptoms,
- lifestyle,
- and general health.

It should not be interpreted as:

a guaranteed substitute for treatment of a varicocele, genetic flagellar defect or serious hormonal disease.



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How I Use Spermogenic and Nuskha No. 129 Responsibly

When such formulations are appropriate, they may be integrated with:

- Ilaj-bil-Ghiza,
- lifestyle correction,
- smoking cessation,
- weight management,
- control of diabetes,
- avoidance of testosterone or steroids,
- stress management,
- and appropriate medical investigation.

The most important point is:

The formulation must fit the patient—not the patient fit the formulation.

“Natural” Does Not Automatically Mean Risk-Free

A herbal product can contain pharmacologically active compounds.

Patients may simultaneously be taking:

- diabetes medicines,
- blood-pressure medicines,
- anticoagulants,
- thyroid medicines,
- psychiatric medicines,
- fertility medicines,
- or other supplements.

Therefore, all herbal and conventional medicines should be disclosed to the treating clinician.

Self-prescribing multiple fertility products can create:

- unnecessary expense,



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- excessive doses,
- drug interactions,
- and confusion about what is actually helping.

Dr. Nizamuddin Qasmi's Special Individualized Approach to Asthenozoospermia

At **Saira Health Care**, my approach can be understood step by step.

Step 1: Confirm the Semen Abnormality

I first review:

- laboratory quality,
- semen volume,
- concentration,
- total count,
- progressive motility,
- total motility,
- morphology,
- vitality,
- and whether the entire semen sample was collected.

If appropriate, the semen analysis is repeated.

One abnormal report does not always justify prolonged treatment.

Step 2: Determine Whether This Is Isolated Asthenozoospermia

I ask:

Is only motility abnormal?

Or does the patient also have:

- low sperm count,
- abnormal morphology,
- low vitality?



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This helps distinguish:

Isolated asthenozoospermia

from

Oligoasthenozoospermia

or

OAT syndrome.

The severity and prognosis are different.

Step 3: Differentiate Poor Motility From Poor Vitality

If many sperm are immotile, I want to know:

Are they alive?

Vitality testing may therefore become important.

This prevents living but non-moving sperm from being incorrectly interpreted as dead sperm.

Step 4: Look for a Varicocele

I perform or arrange appropriate evaluation for clinical varicocele.

If a palpable varicocele is present together with:

- infertility,
- abnormal semen parameters,

a reproductive-urology opinion regarding treatment may be appropriate.

Step 5: Review Lifestyle

I ask specifically about:

- smoking,
- alcohol,
- weight,
- exercise,



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- diet,
- sleep,
- heat exposure,
- occupation,
- recreational drugs,
- testosterone,
- and bodybuilding steroids.

Sometimes the most important treatment clue comes from this history.

Step 6: Review Medical Conditions

I consider:

- diabetes,
 - fever,
 - infection,
 - testicular disease,
 - childhood undescended testis,
 - previous surgery,
 - mumps orchitis,
 - chemotherapy,
 - radiation,
 - and other health conditions.
-

Step 7: Assess Hormonal Health When Indicated

When count is also low or endocrine symptoms are present, I may advise tests such as:

- FSH,
- LH,
- testosterone,
- prolactin,



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or other investigations according to the clinical picture.

Step 8: Recognize Severe Genetic Patterns

If there is:

- near-total immotility,
- severe tail abnormality,
- persistent severe asthenoteratozoospermia,
- or possible ciliary disease,

I consider whether specialized genetic evaluation is necessary.

These cases should not be managed indefinitely with empirical tonics alone.

Step 9: Assess Sexual Health

As a physician with focused practice in **sexual disorders and infertility**, I also evaluate:

- erection,
- ejaculation,
- libido,
- intercourse frequency,
- timing of intercourse,
- and any sexual difficulty interfering with conception.

Sometimes sperm motility is not the only obstacle.

For example, a man may have moderate asthenozoospermia and also severe erectile dysfunction preventing regular intercourse.

Both problems need attention.

Step 10: Evaluate the Female Partner

The woman's:

- age,
- ovarian reserve,



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- ovulation,
- fallopian-tube status,
- reproductive history,
- and duration of infertility

can completely change the treatment strategy.

This is why infertility should be treated at the level of the couple.

Step 11: Correct Reversible Causes

Where relevant, treatment may involve:

- smoking cessation,
 - steroid cessation,
 - weight management,
 - diabetes control,
 - improved sleep,
 - nutritional improvement,
 - treatment of infection,
 - management of hormonal disease,
 - and treatment of clinical varicocele.
-

Step 12: Incorporate Individualized Unani Care

Where appropriate, I may integrate:

Ilaj-bil-Ghiza

for nutritional and metabolic optimization.

Ilaj-bit-Tadbir

for lifestyle and regimenal correction.

Ilaj-bid-Dawa

through selected Unani medicines or fertility-support formulations according to the patient's overall condition.



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This is where Spermogenic Powder, Nuskha No. 129 or other selected medicines may sometimes form part of the individualized treatment programme.

Step 13: Reassess After an Appropriate Interval

I generally judge biological response after sufficient time has passed for newly developing sperm to reflect treatment.

At follow-up, I assess:

- progressive motility,
- total motility,
- concentration,
- total motile sperm number,
- morphology,
- vitality,
- and the couple's actual fertility progress.

The goal is not simply:

“Make motility 50%.”

The goal is:

improve the realistic possibility of conception.

Step 14: Do Not Delay ART When It Is Needed

This is one of the most important principles of responsible integrative fertility treatment.

If a couple has:

- severe male-factor infertility,
- prolonged infertility,
- significantly reduced female ovarian reserve,
- advanced female reproductive age,
- tubal disease,
- or repeated treatment failure,



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I do not believe we should spend years trying supplements simply to obtain a better semen report.

Appropriate referral for:

- IUI,
- IVF,
- or ICSI

should be discussed at the right time.

Can Sperm Motility Improve?

Yes, in many patients it can improve.

Improvement is more likely when there is a reversible factor, such as:

- smoking,
- obesity,
- uncontrolled diabetes,
- fever,
- oxidative stress,
- certain lifestyle problems,
- hormonal abnormalities,
- or an appropriately treatable varicocele.

However, not every patient's motility will normalize.

A severe genetic flagellar disorder cannot be treated the same way as smoking-associated oxidative stress.

Therefore:

Individual prognosis matters more than universal promises.

How Long Does Improvement Take?

Treatment response is commonly evaluated over approximately one spermatogenic cycle.

That usually means thinking in terms of:

months, not days.



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A medicine claiming to completely rebuild sperm quality within:

- three days,
- seven days,
- or ten days

should be approached with caution.

Sperm biology does not work that quickly.

Does Better Motility Guarantee Pregnancy?

No.

A motility increase from:

18% → 36%

is encouraging.

But pregnancy still depends on:

- sperm count,
- morphology,
- DNA integrity,
- ovulation,
- egg quality,
- tubes,
- intercourse timing,
- and many other factors.

Likewise, if the couple becomes pregnant while motility remains at 25%, that is a successful reproductive outcome regardless of whether the report crossed a particular reference threshold.

What Does Successful Treatment Mean?

For me, success has several possible forms.

Patient 1



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A smoker with low motility stops smoking, improves lifestyle and experiences improved semen parameters.

Patient 2

A man with obesity and diabetes improves his metabolic health and sperm quality.

Patient 3

A man with clinically significant varicocele receives appropriate treatment and semen parameters improve.

Patient 4

A man using testosterone discovers that his fertility treatment itself is suppressing spermatogenesis; the problem is managed appropriately.

Patient 5

A man with severe genetic sperm-tail disease receives the correct diagnosis and timely ICSI counselling instead of years of unnecessary medicines.

Patient 6

A couple with mild asthenozoospermia achieves natural pregnancy.

Patient 7

A couple with persistent male-factor infertility successfully moves to IUI or IVF/ICSI when that becomes the most appropriate option.

These are all examples of responsible fertility care.

Why I Avoid Fixed “Success Rate” Claims

Patients often ask:

“Doctor, what percentage of motility patients become completely normal?”

There is no honest universal percentage.

A patient with:

- progressive motility 27%,
- normal count,
- smoking,

is very different from a patient with:



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- progressive motility 2%,
- severe genetic flagellar abnormalities.

Likewise, improving the semen report does not guarantee:

- fertilization,
- implantation,
- pregnancy,
- or live birth.

For this reason, I prefer:

individualized prognosis over marketing statistics.

Myths About Low Sperm Motility

Myth 1: Poor motility means all sperm are dead.

Fact: Some sperm may be alive but immotile. Vitality testing can help distinguish the two.

Myth 2: Progressive motility below 30% means permanent infertility.

Fact: It is below the current WHO lower reference range, but natural conception may still occur depending on the complete fertility picture. ([nice.org.uk](https://www.nice.org.uk))

Myth 3: Thick semen means strong sperm.

Fact: Semen consistency does not determine microscopic sperm motility.

Myth 4: Thin semen means sperm cannot swim.

Fact: Sperm motility must be assessed microscopically.

Myth 5: Low motility means sexual weakness.

Fact: Fertility and erectile function are different. A man can have normal sexual performance and poor sperm motility.

Myth 6: Masturbation permanently reduces sperm motility.



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Fact: Normal masturbation does not cause chronic asthenozoospermia.

Myth 7: Frequent sex destroys motility.

Fact: Regular intercourse does not permanently damage sperm movement.

Myth 8: Testosterone increases sperm strength.

Fact: External testosterone can suppress spermatogenesis and should not be prescribed as fertility treatment to a man seeking current or future fatherhood. (prod.asrm.org)

Myth 9: Every varicocele causes low motility.

Fact: Varicocele is common, including among fertile men. Treatment is generally considered for clinically palpable varicocele in the appropriate infertility setting.

Myth 10: Every ultrasound varicocele should be operated upon.

Fact: Current AUA/ASRM guidance advises against routine varicolectomy for non-palpable varicocele detected only by imaging. (auajournals.org)

Myth 11: One antioxidant cures asthenozoospermia.

Fact: Some studies show improved motility, but current evidence does not establish one universally effective antioxidant treatment or consistent improvement in pregnancy/live birth. (pubmed.ncbi.nlm.nih.gov)

Myth 12: Herbal medicine has no side effects.

Fact: Herbal products contain biologically active compounds and should be selected responsibly.

Myth 13: ICSI is needed whenever motility is below 30%.

Fact: ART decisions are based on the complete semen profile and the couple's reproductive situation—not one number.



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Frequently Asked Questions

What sperm motility is considered normal in 2026?

Current WHO Sixth Edition/NICE reference values include approximately:

- **30% or more progressive motility**
- **42% or more total motility**

([nice.org.uk](https://www.nice.org.uk))

These are reference values rather than absolute fertility boundaries.

Is 25% progressive motility very low?

It is below the current lower reference value.

However, its significance depends on:

- sperm concentration,
 - total sperm number,
 - morphology,
 - total motile sperm,
 - and female-partner factors.
-

Can pregnancy occur with 20% motility?

Yes.

Natural conception may still occur, particularly when:

- sperm numbers are high,
- morphology is reasonable,
- the female partner has good fertility,
- and intercourse is appropriately timed.

The probability may be reduced but not necessarily eliminated.

What if progressive motility is 5%?

This represents a much more severe motility problem.



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It deserves:

- repeat confirmation,
- proper male-fertility evaluation,
- vitality testing where appropriate,
- and assessment of the underlying cause.

Depending on the complete fertility profile, ART may become more relevant.

What does 0% sperm motility mean?

It means no sperm movement was observed in that sample.

It does **not automatically mean all sperm are dead**.

Vitality testing can determine whether viable but immotile sperm remain.

Persistent complete immotility may also justify specialized investigation for structural or genetic flagellar disorders.

Is motility the same as vitality?

No.

Motility measures movement.

Vitality measures whether sperm are alive.

Can varicocele reduce sperm motility?

Yes.

Varicocele can be associated with oxidative stress and impaired semen parameters, including motility. (pubmed.ncbi.nlm.nih.gov)

Can varicocele treatment improve motility?

It may in appropriately selected infertile men with a clinically significant varicocele.

Improvement is not guaranteed.

Can smoking decrease motility?



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Yes.

Smoking is associated with oxidative stress and poorer sperm quality, including impaired motility. ([pmc.ncbi.nlm.nih.gov](https://pubmed.ncbi.nlm.nih.gov/))

Can diabetes affect sperm motility?

Poor metabolic health can contribute to impaired male reproductive health and should be appropriately managed.

Can high fever temporarily reduce motility?

Yes.

A significant recent fever can temporarily impair semen parameters.

Recovery should be assessed after an appropriate biological interval.

Can testosterone injections reduce sperm motility?

External testosterone can suppress overall sperm production and may lead to severe oligozoospermia or azoospermia.

It should not be used as fertility treatment in men trying to conceive. ([prod.asrm.org](https://procedures.asrm.org/))

Can sperm motility improve naturally?

It can improve in some men after correcting reversible factors such as:

- smoking,
- obesity,
- poor metabolic health,
- steroid use,
- recent illness,
- or other lifestyle problems.

However, severe structural or genetic causes may not respond to lifestyle treatment alone.

Can antioxidants improve motility?

Some studies and meta-analyses report improvement in motility measurements.

However, evidence remains heterogeneous, and current major guidelines do not recommend one antioxidant regimen universally. (pubmed.ncbi.nlm.nih.gov)

Can L-carnitine improve motility?

Some research suggests possible improvement in sperm motility.

However, evidence for improved pregnancy outcomes is much less certain.
(pubmed.ncbi.nlm.nih.gov)

Can Unani medicine help poor sperm motility?

Unani medicine can contribute meaningfully to an individualized treatment plan through:

- Ilaj-bil-Ghiza,
- Ilaj-bit-Tadbir,
- Ilaj-bid-Dawa,
- nutritional management,
- metabolic-health support,
- sleep and lifestyle management,
- stress reduction,
- and selected fertility-support formulations.

Published Unani research includes clinical observations of improvements in sperm count and motility, although stronger modern randomized studies are still required. (ccrum.res.in)

Is Spermogenic proven to cure asthenozoospermia?

Spermogenic is used within Saira Health Care's traditional male-fertility programme and is described by the clinic as a fertility-support formulation for semen-quality concerns.
(sairahealthcare.com)

However, I do not claim that product-specific high-quality controlled trials prove that it universally cures asthenozoospermia.

It should be used as part of an individualized programme after appropriate evaluation.



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What is the role of Nuskha No. 129?

Nuskha No. 129 is used within Saira Health Care's traditional male reproductive-health practice as part of individualized Unani management. (sairahealthcare.com)

It should complement—not replace—treatment of identifiable causes such as:

- clinical varicocele,
- infection,
- hormonal disease,
- steroid suppression,
- or severe genetic sperm disorders.

Can Hijama improve motility?

At present, high-quality evidence is insufficient to claim that Hijama reliably corrects asthenozoospermia.

It may be used as supportive regimenal therapy in selected Unani treatment plans but should not be advertised as a proven stand-alone fertility cure.

Is IUI possible with poor motility?

Yes, in selected patients.

The important parameter is often the number of **motile sperm available after semen processing**, along with the female partner's fertility.

Is ICSI useful for severe asthenozoospermia?

Yes.

ICSI can bypass the need for sperm to travel naturally to the egg by directly injecting a selected sperm into an oocyte.

It can be especially useful in severe male-factor infertility.

Does ICSI guarantee pregnancy?

No.



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ICSI assists fertilization.

Pregnancy still depends on:

- sperm viability,
- egg quality,
- embryo development,
- female age,
- uterine factors,
- and implantation.

When Should a Man Seek Specialist Evaluation?

I recommend proper male-fertility evaluation when:

- progressive motility remains below reference values on repeated semen analysis,
- motility is severely reduced,
- nearly all sperm are immotile,
- vitality is also low,
- sperm count is reduced,
- morphology is also abnormal,
- a palpable varicocele is present,
- testes are unusually small,
- testicular pain or swelling occurs,
- testosterone or anabolic steroids have been used,
- childhood undescended testes were present,
- there is a history of testicular injury,
- recurrent genital infection is suspected,
- chemotherapy or radiation has been received,
- there are recurrent miscarriages,
- repeated ART attempts have failed,
- or the couple has been unable to conceive.



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Severe persistent asthenozoospermia deserves diagnosis—not endless empirical treatment.

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 listed for this clinical work 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 current public physician profile describes my focused practice in sexual disorders and infertility and specifically lists **Asthenospermia**, along with:

- azoospermia,
- oligospermia,
- teratospermia,
- necrospermia,
- varicocele,
- epididymal cyst,
- hormonal abnormalities,
- and other reproductive-health concerns. (sairahealthcare.com)

Saira Health Care's current professional educational material also lists the **Masters in Male Infertility – MasterHealthPro (HealthPro)** and **Integrated Sexual and Reproductive Health – ISRH, UNFPA** in the professional profile used for its sexual- and reproductive-health content. (sairahealthcare.com)

This combined focus is particularly useful in asthenozoospermia because male infertility is not simply a laboratory problem.



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It can involve:

- semen quality,
- reproductive hormones,
- testicular health,
- sexual function,
- varicocele,
- lifestyle,
- metabolic health,
- genetics,
- psychological stress,
- and the female partner's fertility.

Saira Health Care's Contribution to Sexual Disorders and Infertility

One of the major objectives of Saira Health Care has been to improve understanding of sexual and reproductive disorders.

Many men reach us after being told:

“Your sperm are slow, so you cannot become a father.”

Others have been told:

“Take one medicine and sperm will become 100% active.”

Both statements can be misleading.

Saira Health Care's publicly available clinical material identifies male infertility—including abnormal:

- count,
- motility,
- morphology,
- varicocele,
- hormonal problems

—as an important area of its work. (sairahealthcare.com)

Our broader contribution should therefore not be measured only by:



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how many medicines are prescribed.

I believe it should be measured by our ability to:

- interpret semen reports correctly,
- identify potentially reversible causes,
- correct myths,
- evaluate sexual health,
- integrate Unani treatment responsibly,
- identify when modern medical or surgical treatment is necessary,
- evaluate both partners,
- and refer patients for assisted reproduction when that offers the better option.

The Saira Health Care Integrative Treatment Philosophy

For me, integrative medicine means selecting the treatment according to the patient's actual condition.

Patient A

Has progressive motility 27%, excellent count, good morphology and no major clinical problem.

He may primarily require:

- reassurance,
- lifestyle optimization,
- appropriate follow-up,
- and couple-level assessment.

Patient B

Has progressive motility 15%, obesity, smoking and uncontrolled diabetes.

For him:

- smoking cessation,
- metabolic treatment,
- weight management,
- nutritional correction,



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- and individualized Unani supportive care

may be particularly important.

Patient C

Has poor motility and a clinically significant palpable varicocele.

He may need:

- reproductive-urology assessment

rather than simply more fertility supplements.

Patient D

Has 0% motility but good sperm vitality.

This raises the possibility of:

- living but immotile sperm,
- including a possible structural or genetic motility disorder.

Specialized reproductive assessment becomes essential.

Patient E

Has poor motility after anabolic steroid or testosterone use.

The primary problem may be:

- suppression of reproductive hormones.

Patient F

Has moderate asthenozoospermia, but his wife has significantly diminished ovarian reserve.

We should not waste years simply trying to increase his motility percentage.

Timely ART discussion may be more appropriate.

This is what I mean by:

individualized treatment.

Latest Scientific Perspective: 2025–2026

Recent research has considerably improved our understanding of asthenozoospermia.

WHO and 2026 NICE Semen Standards

Current reference values include approximately:



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Progressive motility $\geq 30\%$

and

Total motility $\geq 42\%$. ([nice.org.uk](https://www.nice.org.uk))

These values should not be interpreted as absolute fertility cutoffs.

WHO Infertility Guideline – November 2025

WHO published its first global evidence-based guideline for infertility in November 2025, emphasizing standardized diagnosis and evidence-based treatment of both male and female factors. ([who.int](https://www.who.int))

This reinforces the need to evaluate infertility at the couple level rather than concentrating on one semen result.

Advances in Genetics

A major 2025 review describes rapidly expanding knowledge of genetic causes of asthenozoospermia involving:

- sperm flagella,
- ion channels,
- energy pathways,
- mitochondrial function,
- and ciliary proteins. (pubmed.ncbi.nlm.nih.gov)

These discoveries are particularly important in severe persistent sperm immotility.

Oxidative Stress Research

A major 2025 review describes oxidative stress and mitochondrial damage as important biological mechanisms in asthenozoospermia. (pubmed.ncbi.nlm.nih.gov)

However, oxidative stress should be understood as a mechanism—not used as an excuse to prescribe unlimited antioxidants to every patient.

Antioxidant Evidence in 2026



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A 2026 meta-analysis involving 11,889 infertile men found improvements in several semen parameters, including progressive and total motility, but concluded that the evidence remains too heterogeneous for a definitive universal antioxidant recommendation.

(pubmed.ncbi.nlm.nih.gov)

Another 2026 systematic review found very-low-certainty evidence for improvements in semen parameters and emphasized the need for stronger studies measuring pregnancy and live-birth outcomes. (pubmed.ncbi.nlm.nih.gov)

Therefore, the modern treatment direction is moving toward:

cause-targeted rather than purely empirical treatment.

My Final Message to Patients

If your report says:

“Sperm motility is low”

please do not immediately think:

“I can never become a father.”

Instead, ask:

What is my progressive motility?

What is my total motility?

How many sperm are present in total?

How many motile sperm are actually available?

How is my morphology?

Are the immotile sperm alive?

Is my vitality normal?

Do I have a varicocele?

Do I smoke?

Do I have diabetes or obesity?

Did I recently have a high fever?

Am I taking testosterone or steroids?

Could there be infection or inflammation?



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Is this mild acquired asthenozoospermia or a severe genetic sperm-tail disorder?

How is my wife's fertility?

These questions are more useful than simply asking:

“Which medicine makes sperm move fast?”

At Saira Health Care, my philosophy is to provide:

hope without false promises and treatment without guesswork.

Conclusion

Decreased sperm motility, medically known as **asthenozoospermia**, is a condition in which a lower-than-reference proportion of sperm demonstrate effective forward movement.

Under current WHO Sixth Edition standards incorporated into the 2026 NICE fertility guideline, the lower reference values are approximately:

- **30% progressive motility**
- **42% total motility**
- **54% vitality**

([nice.org.uk](https://www.nice.org.uk))

These values are statistical reference points rather than absolute fertile-versus-infertile boundaries.

Asthenozoospermia usually causes no physical symptoms and is most often detected through semen analysis during infertility evaluation.

Potential causes and contributing factors include:

- oxidative stress,
- clinical varicocele,
- smoking,
- obesity,
- diabetes,
- genital inflammation,
- fever,
- excessive heat,



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- heavy alcohol use,
- environmental toxins,
- anabolic steroids,
- testosterone therapy,
- hormonal disease,
- testicular disorders,
- mitochondrial dysfunction,
- genetic flagellar abnormalities,
- and unexplained factors.

Modern treatment depends on the cause.

It may involve:

- smoking cessation,
- weight and metabolic management,
- avoidance of anabolic steroids,
- treatment of hormonal disorders,
- treatment of proven infection,
- treatment of clinically significant varicocele,
- selective use of nutritional or antioxidant support,
- IUI,
- IVF,
- or ICSI.

Current evidence suggests that antioxidants may improve semen parameters in some men, but the evidence remains heterogeneous and does not justify a universal antioxidant prescription or guaranteed pregnancy claim. (pubmed.ncbi.nlm.nih.gov)

The **Unani system of medicine** can make a valuable contribution through its individualized whole-person approach involving:

- **Mizaj**
- **Asbab-e-Sitta Zarooriya**
- **Ilaj-bil-Ghiza**



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- **Ilaj-bit-Tadbir**
- **Ilaj-bid-Dawa**
- nutritional support,
- lifestyle optimization,
- metabolic-health improvement,
- stress and sleep management,
- and individually selected fertility-support formulations.

Unani clinical literature contains promising observations of improvement in sperm count and motility with selected traditional formulations, although stronger modern controlled research is required. (ccrum.res.in)

At **Saira Health Care**, Spermogenic Powder and Dr. Qasmi's Nuskha No. 129 may be incorporated into selected individualized male-fertility programmes based on the patient's overall condition. They should be understood as **traditional supportive formulations**, not as guaranteed substitutes for appropriate medical, surgical, genetic or assisted-reproductive treatment. (sairahealthcare.com)

My approach can therefore be summarized as:

Confirm the motility abnormality.

Look at the complete semen report.

Differentiate motility from vitality.

Find the underlying cause whenever possible.

Look for varicocele and reversible lifestyle factors.

Avoid fertility-suppressing testosterone and steroids.

Recognize severe genetic motility disorders.

Use Unani medicine rationally and individually.

Monitor improvement over a biologically meaningful period.

Evaluate both partners.

And use IUI, IVF or ICSI without unnecessary delay when the couple's situation requires it.

For every patient who comes to me worried about “slow sperm,” my central message is:

Poor sperm motility is an important fertility finding—but it is not automatically a final verdict on fatherhood.



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The most important next step is:

accurate diagnosis, identification of the cause and individualized treatment.

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

Dr. Nizamuddin Qasmi's current public profile at Saira Health Care describes his focused clinical work in sexual disorders and infertility and includes **asthenospermia/decreased sperm motility**, oligospermia, azospermia, teratospermia, necrospermia, varicocele and other male- and female-infertility conditions among the reproductive-health concerns addressed in his practice. (sairahealthcare.com)

His clinical approach combines the individualized principles of Unani medicine with contemporary semen analysis, reproductive-health assessment, lifestyle guidance, patient education and appropriate modern infertility referral.

Medical Disclaimer

This article is intended for patient education and general reproductive-health awareness.

It is not a substitute for an individual medical consultation, semen-analysis interpretation, physical examination, diagnosis or personalized fertility-treatment plan.

A single low-motility result does not by itself establish permanent male infertility.

Abnormal semen findings should be interpreted together with:



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- sperm count,
- total sperm number,
- morphology,
- vitality,
- medical history,
- sexual function,
- and female-partner fertility.

Repeat semen analysis may be appropriate after an abnormal initial test.

Do not independently start or stop:

- testosterone,
- anabolic steroids,
- hormonal medicines,
- antibiotics,
- fertility medicines,
- antioxidant supplements,
- or Unani/herbal medicines

without appropriate professional guidance.

Men interested in current or future fatherhood should specifically inform their healthcare professional before beginning testosterone therapy because external testosterone can suppress spermatogenesis.

Unani medicines should be prescribed under appropriately qualified supervision.

Spermogenic Powder, Nuskha No. 129 or any other fertility formulation should not be interpreted as a guaranteed cure for asthenozoospermia or a guaranteed method of achieving pregnancy.

Patients with severe or complete sperm immotility, low vitality, major flagellar abnormalities, recurrent pregnancy loss or repeated ART failure may require specialist investigation.

Where reproductive urology, endocrinology, genetic counselling, varicocele surgery, IUI, IVF or ICSI is required, timely referral should form part of responsible infertility care.



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