Maturation Arrest in Males: Causes, Diagnosis, Treatment, Fertility Options and the Role of Unani Medicine

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Introduction: "Doctor, My Biopsy Says Maturation Arrest. Does That Mean My Testes Cannot Make Sperm?"

When a man comes to me with a report containing the words:

Maturation Arrest

he is usually very worried.

He may ask:

"Doctor, does this mean my sperm production has permanently stopped?"

Another patient may ask:

"Can medicine restart sperm production?"

A man with azoospermia may tell me:

"My semen contains no sperm, but the biopsy says germ cells are present. Can sperm still be found?"

Another patient may have been told:

"Take medicines for seven or eight months and the maturation will definitely complete."

These situations require careful explanation because **maturation arrest is not one simple disease and does not have one universal treatment**.

Maturation arrest—more precisely:

Spermatogenic Maturation Arrest

means that sperm-forming germ cells begin developing inside the testes but fail to progress normally beyond a particular stage.

In other words:

the sperm-production pathway starts, but development becomes arrested before fully mature spermatozoa are produced in the affected seminiferous tubules.

It is one of the histological patterns that can be found in men with:

Non-Obstructive Azoospermia – NOA

or, less commonly, very severe oligozoospermia.

Modern genetic research shows that maturation arrest is biologically heterogeneous. The problem can occur:

- before meiosis,
- during meiosis,
- or after meiosis,

and many different genes involved in:

- chromosome pairing,
- DNA repair,
- meiotic division,
- germ-cell differentiation

can be responsible.

The first thing I therefore explain to my patients is:

Maturation arrest does not automatically mean “no hope,” but neither can every case be restarted by medicines.

Treatment depends on:

- where maturation stops,
- whether arrest is focal or diffuse,
- the underlying cause,
- hormonal status,
- genetics,

- whether rare sperm are present elsewhere in the testis,
- and the fertility status of the female partner.

At **Saira Health Care**, I believe the correct question is not:

“Which medicine cures maturation arrest?”

The correct questions are:

“Why did sperm development arrest, is any part of the process reversible, are mature sperm present somewhere in the testes, and what is the safest realistic route to biological parenthood?”

What Is Normal Spermatogenesis?

To understand maturation arrest, we first need to understand normal sperm production.

Sperm are produced inside the:

Seminiferous Tubules of the Testes

through a complex biological process called:

Spermatogenesis

The basic developmental pathway can be simplified as:

Spermatogonia

↓
Primary Spermatocytes
↓
Secondary Spermatocytes
↓
Round Spermatids
↓
Elongating Spermatids
↓
Spermatozoa

This process involves three major biological events:

Mitosis

Early germ cells multiply.

Meiosis

Chromosome number is reduced from 46 to 23.

Spermiogenesis

Round spermatids transform structurally into spermatozoa.

Only after these stages are completed can mature sperm eventually:

- leave the testis,
- enter the epididymis,
- undergo additional maturation,
- and become capable of effective motility and fertilization.

What Exactly Does "Maturation Arrest" Mean?

Maturation arrest means that germ-cell development stops at one of these stages before mature sperm production is completed.

For example, a testicular tubule may contain:

- spermatogonia,
- primary spermatocytes,

but no later germ cells.

Or development may proceed further until:

- early spermatids,

but fail to form mature sperm.

Therefore:

maturation arrest is a developmental failure of spermatogenesis.

It is not simply:

- a low sperm count,
- low sperm motility,
- abnormal morphology,
- weak ejaculation,
- erectile dysfunction,
- or reduced libido.

Maturation Arrest Is Usually a Histological Diagnosis

This is extremely important.

A semen analysis can show:

azoospermia

but it cannot tell us whether the testes contain:

- Sertoli-cell-only syndrome,
- maturation arrest,
- hypospermatogenesis,
- or another microscopic pattern.

Maturation arrest is most accurately identified by examining:

testicular tissue microscopically.

In contemporary practice, this histological information may be obtained during:

- testicular sperm-retrieval procedures,
- particularly TESE or micro-TESE,

rather than performing a separate diagnostic biopsy in every patient.

AUA/ASRM advises that routine diagnostic testicular biopsy should **not** normally be performed simply to differentiate obstructive from non-obstructive azoospermia because clinical examination, semen findings and hormones usually provide substantial information.

Why a Small Biopsy Does Not Always Tell the Whole Story

Testicular sperm production can be:

heterogeneous.

One tiny biopsy may show maturation arrest while another region of the same testis may contain:

- more advanced spermatogenesis,
- or even rare mature sperm.

This concept is especially important in:

Non-Obstructive Azoospermia.

It is one reason micro-TESE is useful: rather than randomly taking one small tissue sample, the surgeon systematically examines seminiferous tubules under magnification searching for regions more likely to contain active sperm production.

Early Maturation Arrest

Maturation arrest can be classified according to the most advanced germ-cell stage identified.

In:

Early Maturation Arrest

sperm development stops relatively early.

A common histological definition is arrest around the:

primary spermatocyte stage

before successful completion of meiosis.

The testis may therefore contain:

- spermatogonia,
- primary spermatocytes,

but no mature spermatids or spermatozoa in affected tubules.

Late Maturation Arrest

In:

Late Maturation Arrest

development progresses further.

Germ cells may reach:

- spermatid stages

before failing to complete maturation into normal spermatozoa.

This distinction has practical fertility significance.

A large micro-TESE series of 211 men with maturation arrest reported sperm retrieval in:

- **40% of men with early maturation arrest**
- versus **78% of men with late maturation arrest.**

These figures came from one retrospective specialist-center series and **should not be presented as guaranteed individual success rates**, but they demonstrate why late maturation arrest can carry a better sperm-retrieval prognosis than early arrest.

Focal Maturation Arrest

In:

Focal Maturation Arrest

not every seminiferous tubule is affected identically.

Some regions may show arrest while others contain:

- more advanced germ cells,
- possibly mature sperm.

This is clinically important because micro-TESE may locate those isolated areas.

Diffuse or Complete Maturation Arrest

In:

Diffuse Maturation Arrest

the same developmental arrest is seen throughout essentially all sampled tubules.

This usually carries a poorer sperm-retrieval prognosis than focal disease.

In the same published micro-TESE series, sperm retrieval was approximately:

- **35% in diffuse maturation arrest**
- versus **57% in focal maturation arrest.**

Again, these are historical cohort results rather than guarantees for an individual patient.

Maturation Arrest vs Hypospermatogenesis

These terms are different.

Hypospermatogenesis

All stages of spermatogenesis may be present, including:

- mature sperm,

but the quantity is reduced.

Maturation Arrest

Development stops before mature sperm are consistently formed.

In general, hypospermatogenesis often offers a better sperm-retrieval prognosis because:

the biological machinery can complete the entire sperm-development pathway.

Maturation Arrest vs Sertoli-Cell-Only Syndrome

In:

Sertoli-Cell-Only Syndrome – SCOS

affected seminiferous tubules contain:

- Sertoli cells,

but lack germ cells.

In maturation arrest:

germ cells are present but fail to complete development.

This is a major biological difference.

Maturation Arrest vs Obstructive Azoospermia

Another essential distinction.

A man with:

Obstructive Azoospermia

may have completely normal spermatogenesis.

His testes produce mature sperm normally, but:

- epididymal,
- vasal,
- or ejaculatory-duct obstruction

prevents sperm from appearing in semen.

By contrast:

maturation arrest is a sperm-production problem.

Therefore, simply having:

zero sperm in semen

does not tell us which condition is present.

Symptoms of Maturation Arrest

Most men with maturation arrest have:

no specific physical symptoms.

The usual presentation is:

infertility.

Many affected men may have:

- normal sexual desire,
- normal erection,
- normal ejaculation,
- normal orgasm,
- normal-looking semen.

This is another reason semen appearance and sexual performance cannot diagnose sperm-production disorders.

Does Maturation Arrest Cause Erectile Dysfunction?

Not necessarily.

A man may have:

complete azoospermia due to maturation arrest

while still having completely normal:

- testosterone,
- libido,
- erection,
- ejaculation.

Sexual performance and sperm production are separate biological functions.

Because my focused clinical practice includes both:

I consider it important to separate these two issues clearly.

Does Maturation Arrest Cause Low Testosterone?

Sometimes, but not always.

Maturation arrest can exist in men with:

- normal serum testosterone,
- normal testicular volume,
- and even normal FSH.

Current EAU guidance specifically notes that when spermatogonia are present but maturation arrests at the:

- spermatocyte,
- or spermatid

stage, FSH may remain within the normal range.

Therefore:

normal hormones do not exclude maturation arrest.

Can FSH Be High?

Yes.

Other men with severe spermatogenic impairment have:

- elevated FSH,
- small testes,
- evidence of primary testicular failure.

But an important clinical principle is:

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

EAU notes that men with NOA and high FSH may still have:

focal areas of spermatogenesis

that can potentially be found during TESE or micro-TESE.

Why Does Maturation Arrest Occur?

Maturation arrest has many possible causes.

They can broadly be divided into:

1. Genetic causes
2. Chromosomal causes
3. Hormonal causes
4. Acquired testicular injury
5. Environmental or toxic causes
6. Medication-related causes
7. Idiopathic causes

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1. Genetic Causes

Modern genetics is transforming our understanding of maturation arrest.

Sperm production depends on hundreds of genes controlling:

- germ-cell division,
- DNA repair,
- chromosome pairing,
- meiotic recombination,
- chromosomal separation,
- germ-cell differentiation.

Pathogenic variants can prevent germ cells from progressing through meiosis.

Published genetic research has identified maturation-arrest-associated variants involving genes such as:

- **TEX11**
- **SYCE1**
- **MEIOB**
- **MEI1**

- **STAG3**
- **TEX14**
- **DMRT1**
- **ADAD2**
- **TERB1**
- **SHOC1**
- **MSH4**
- **RAD21L1**

among others.

A 2025 review of the genetic and epigenetic landscape of NOA also highlights genes such as:

- **SYCP1**
- **SYCE1**
- **HORMAD1**

in spermatogenic arrest and meiotic failure.

This is one reason a man with genetically determined maturation arrest may not respond to an ordinary:

- vitamin,
- antioxidant,
- herbal fertility medicine.

The underlying developmental instructions themselves may be abnormal.

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2. Y-Chromosome Microdeletions

The Y chromosome contains regions important for spermatogenesis known as:

- **AZFa**
- **AZFb**
- **AZFc**

Certain deletions are strongly associated with severe spermatogenic failure.

The 2024 AUA/ASRM guideline recommends Y-chromosome microdeletion testing particularly in men with:

- primary infertility,
- azoospermia,
- or sperm concentration **≤1 million/mL**

when accompanied by evidence of impaired sperm production such as:

- high FSH,
- testicular atrophy.

This represents an important update from older recommendations that used a broader ≤5 million/mL threshold.

Complete AZFa and AZFb Deletions

This is one of the most important prognostic genetic findings.

Current AUA/ASRM evidence reports that sperm have not been successfully retrieved by micro-TESE in men with:

- complete AZFa,
- complete AZFb,
- AZFab,
- or AZFabc deletions.

Current EAU recommendations therefore advise:

against sperm-retrieval surgery in complete AZFa and AZFb deletion because the chance of sperm retrieval is essentially zero.

This illustrates why genetic testing should sometimes occur **before surgery**.

AZFc Deletion

AZFc behaves differently.

Men may have:

- severe oligozoospermia,
- or azoospermia.

In azoospermic men with isolated AZFc deletion, the 2024 AUA/ASRM guideline reports sperm retrieval with micro-TESE in approximately:

50%

of cases in available studies.

However, male offspring can inherit the same Y-chromosome deletion.

Therefore:

genetic counselling is essential.

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3. Chromosomal Abnormalities

A karyotype can identify abnormalities involving:

- chromosome number,
- chromosome structure.

Examples include:

- Klinefelter syndrome,
- translocations,
- inversions.

The 2024 AUA/ASRM guideline recommends karyotype testing for men with:

- primary infertility,
- azoospermia,
- or sperm concentration **<5 million/mL**

when accompanied by:

- elevated FSH,
- testicular atrophy,
- or diagnosed impaired sperm production.

Certain chromosomal rearrangements may disrupt normal meiosis and produce:

meiotic maturation arrest.

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4. Hypogonadotropic Hypogonadism

This is extremely important because it represents a very different—and often treatable—problem.

In:

Hypogonadotropic Hypogonadism

the testes do not receive sufficient stimulation from:

- LH,
- FSH.

This may result from problems involving:

- hypothalamus,
- pituitary gland.

Without adequate stimulation:

- Leydig cells do not produce sufficient intratesticular testosterone,
- Sertoli-cell support is reduced,
- spermatogenesis may fail.

Unlike a fixed genetic meiotic defect:

spermatogenesis can often be induced in appropriately diagnosed hypogonadotropic hypogonadism.

Current EAU guidance recommends fertility-directed therapy using:

- hCG,
- FSH/hMG,
- recombinant or purified gonadotropins

in men with congenital or acquired hypogonadotropic hypogonadism who desire fertility.

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5. Exogenous Testosterone

This deserves special attention.

Men sometimes take:

- testosterone injections,
- testosterone gels,
- bodybuilding hormones

thinking:

"Testosterone is necessary for sperm, so taking more testosterone should improve sperm production."

The opposite can happen.

External testosterone suppresses:

- GnRH,
- LH,
- FSH.

This reduces intratesticular testosterone and can produce:

- severe oligozoospermia,
- or azoospermia.

AUA/ASRM states clearly:

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

EAU similarly considers testosterone therapy contraindicated as treatment for male infertility.

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6. Anabolic Steroids

Bodybuilding steroids can suppress the same hormonal axis.

EAU recommends withdrawal of anabolic steroids in infertile men and notes that sperm production often improves over approximately:

6–12 months after cessation

although recovery varies between individuals.

Therefore, a man with steroid-induced azoospermia should not automatically be labelled as having irreversible genetic maturation arrest.

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7. Testicular Heat

The testes function optimally at a temperature slightly below core body temperature.

Excessive or chronic heat may interfere with:

- germ-cell survival,

- meiosis,
- sperm development.

Possible sources include:

- severe varicocele,
- prolonged occupational heat,
- repeated extreme sauna or hot-tub exposure.

Heat alone should not be assumed to explain biopsy-confirmed maturation arrest without appropriate evaluation.

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8. Varicocele

Varicocele can create an abnormal testicular environment through:

- increased temperature,
- oxidative stress,
- altered venous circulation.

Varicocele treatment may improve fertility in appropriately selected men with:

- clinical palpable varicocele,
- abnormal semen parameters.

However, maturation arrest often occurs in the context of:

NOA

where the evidence for varicocele repair is much less certain.

AUA/ASRM states that men with:

- NOA,
- clinical varicocele

should be informed that definitive evidence supporting varicocele repair before ART is lacking.

Therefore:

varicocele repair should not be advertised as a guaranteed treatment for maturation arrest.

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9. Chemotherapy and Radiation

Cancer treatments can damage:

- dividing germ cells,
- Sertoli cells,
- testicular tissue.

This may produce:

- temporary,
- prolonged,
- or permanent spermatogenic failure.

Whenever possible, men facing gonadotoxic therapy should discuss:

sperm cryopreservation before treatment.

AUA/ASRM recommends fertility-preservation counselling before chemotherapy or radiation likely to affect sperm production.

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10. Severe Testicular Infection

Conditions such as:

- mumps orchitis,
- severe epididymo-orchitis

can damage testicular tissue.

The effect depends on:

- severity,
- whether one or both testes were involved,
- degree of tissue injury.

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11. Undescended Testes

A history of:

Cryptorchidism

is an important risk factor for impaired spermatogenesis.

Testicular exposure to higher abdominal or inguinal temperature during development may damage:

- germ-cell number,
- maturation.

The risk is greater with:

- bilateral disease,
- delayed correction.

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12. Testicular Torsion or Trauma

Severe loss of testicular blood supply or tissue injury can impair:

- germ-cell survival,
- seminiferous-tubule function.

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13. Environmental and Occupational Toxins

Potential concerns include:

- pesticides,
- heavy metals,
- solvents,
- radiation,
- endocrine-disrupting chemicals.

Proving that one particular exposure caused maturation arrest in an individual patient is difficult.

A detailed occupational and medication history remains important.

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14. Idiopathic Maturation Arrest

Even after:

- hormonal testing,
- karyotype,
- Y-chromosome testing,

- medical history,

some men have no identifiable cause.

These cases are described as:

Idiopathic Maturation Arrest.

Modern genomic research suggests that a proportion of apparently idiopathic cases probably contain:

- currently unrecognized single-gene,
- regulatory,
- epigenetic abnormalities.

How Is Maturation Arrest Diagnosed?

Diagnosis requires a structured male-infertility evaluation.

Step 1: Confirm Infertility and Evaluate Both Partners

Infertility is a couple's condition.

AUA/ASRM recommends concurrent evaluation of:

- male,
- female partner.

This is particularly important before spending months trying to improve sperm production.

If the female partner has:

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

the couple may not have unlimited time.

Step 2: Semen Analysis

A standardized semen analysis is the starting laboratory investigation.

If sperm are completely absent:

azoospermia must be confirmed properly.

Current EAU guidance recommends confirming NOA on:

two consecutive semen analyses

when no sperm are found after centrifugation.

The WHO's first global infertility guideline, published on **28 November 2025**, suggests repeating semen analysis after at least:

11 weeks

when one or more semen parameters are outside the WHO reference ranges.

Clinical urgency can justify earlier reassessment in selected situations.

Step 3: Search the Centrifuged Pellet Carefully

Before declaring complete azoospermia, the laboratory should examine a:

centrifuged concentrated semen pellet

for rare sperm.

Some men initially labelled azoospermic actually have:

cryptozoospermia

with very rare sperm detectable only after extensive search.

If rare sperm are identified:

cryopreservation should be considered.

Step 4: Physical Examination

I assess:

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

This helps distinguish:

- impaired sperm production,
- from obstruction.

Step 5: Hormonal Evaluation

Important tests may include:

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

according to the clinical picture.

EAU emphasizes that FSH is useful but imperfect.

A man with maturation arrest may have:

normal FSH and normal testicular volume.

Step 6: Genetic Testing

Depending on the semen pattern and examination, current genetic evaluation may include:

Karyotype

particularly in azoospermia or sperm concentration <5 million/mL with evidence of impaired production.

Y-Chromosome Microdeletion Testing

particularly in azoospermia or sperm concentration ≤1 million/mL with evidence of testicular production failure under the 2024 AUA/ASRM recommendations.

Further gene-panel or exome testing may be considered in selected specialized settings.

Step 7: Scrotal Ultrasound

Ultrasound may help assess:

- testicular size,
- structural abnormalities,

- varicocele.

But:

ultrasound cannot diagnose maturation arrest.

It cannot see whether a germ cell stopped at:

- primary spermatocyte,
- spermatid stage.

That requires tissue-level assessment.

Step 8: Histopathology

Histology may identify:

- hypospermatogenesis,
- early maturation arrest,
- late maturation arrest,
- Sertoli-cell-only syndrome,
- mixed patterns.

However, routine biopsy purely for diagnosis is generally avoided when the result would not change management.

Step 9: Micro-TESE in Appropriate NOA Patients

When a man with NOA seeks biological parenthood and sperm retrieval is appropriate:

micro-TESE

is the principal modern surgical strategy.

AUA/ASRM recommends micro-TESE for men with NOA undergoing sperm retrieval.

EAU likewise recommends surgical sperm retrieval in appropriate NOA candidates for ICSI and recognizes micro-TESE as the preferred approach.

What Is Micro-TESE?

Micro-TESE means:

Microdissection Testicular Sperm Extraction

During the procedure:

1. testicular tissue is exposed,
2. the surgeon uses operating-microscope magnification,
3. seminiferous tubules that appear more promising are identified,
4. small targeted samples are removed,
5. an embryology laboratory searches for sperm.

The objective is to find:

focal spermatogenesis

while limiting unnecessary testicular tissue removal.

Can Micro-TESE Find Sperm in Maturation Arrest?

Yes, sometimes.

The best-known large maturation-arrest series included 211 men and reported:

- overall sperm retrieval: **52%**
- early arrest: **40%**
- late arrest: **78%**
- diffuse arrest: **35%**
- focal arrest: **57%**.

However, several cautions are essential.

These percentages:

- come from a retrospective tertiary-center study,
- do not apply identically to every patient,
- do not equal pregnancy rates,
- do not equal live-birth rates.

A more recent multicenter NOA study also found that histological maturation arrest was associated with lower overall sperm-retrieval probability compared with more favorable histological patterns.

Therefore:

micro-TESE offers a possibility—not a guarantee.

Why Late Maturation Arrest Has a Better Prognosis

In late maturation arrest:

the spermatogenic process has already progressed much closer to mature sperm production.

This suggests that some neighboring tubules may successfully complete development.

In early arrest, the interruption occurs farther upstream.

That helps explain why published sperm-retrieval rates are often better in late than early maturation arrest.

Can High FSH Predict Micro-TESE Failure?

No.

FSH provides useful information but cannot reliably tell us whether:

one tiny focus of mature sperm exists.

EAU specifically states that FSH does not accurately predict the presence of spermatogenesis at TESE, and high-FSH NOA patients may still have focal sperm production.

I therefore discourage statements such as:

“Your FSH is 25, so there is absolutely no sperm.”

That conclusion is too strong.

Can Normal FSH Guarantee Micro-TESE Success?

No.

Normal FSH can be seen with:

- maturation arrest.

Therefore:

normal FSH is not proof that sperm will be retrieved.

Hormones are part of the assessment, not a substitute for complete diagnosis.

Treatment of Maturation Arrest

This is the central question.

There is currently:

no one universally established medicine that reverses all maturation arrest.

Treatment depends on the cause.

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1. Treat True Hormonal Deficiency

If spermatogenesis is arrested or absent because of:

hypogonadotropic hypogonadism

this is one of the clearest situations in which medical treatment may restore sperm production.

Current EAU guidance supports:

- hCG,
- FSH/hMG,
- related gonadotropin therapy

to induce spermatogenesis in appropriately diagnosed men.

AUA/ASRM also states that spermatogenesis and pregnancies can be achieved in many men with idiopathic hypogonadotropic hypogonadism using:

- gonadotropins,
- or pulsatile GnRH.

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2. Do Not Give Testosterone as Fertility Treatment

I repeat this because it is extremely important.

External testosterone may make a patient feel:

- energetic,
- stronger,
- sexually better,

while simultaneously suppressing:

sperm production.

Men wishing current or future fertility should not receive testosterone monotherapy as infertility treatment.

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3. Stop Anabolic Steroids

If spermatogenesis is suppressed by:

- bodybuilding steroids,
- non-medical testosterone,

withdrawal is fundamental.

Recovery may require:

- months,
- sometimes longer.

EAU describes sperm improvement commonly occurring over about **6–12 months** after cessation in steroid-related suppression.

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4. Treat Hyperprolactinemia Where Present

High prolactin can impair the reproductive hormonal axis.

When genuinely elevated:

- the cause should be identified,
- appropriate dopamine-agonist therapy may improve reproductive function.

This is cause-specific treatment—not general maturation-arrest therapy.

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5. Varicocele Management

If a man has:

- a palpable clinical varicocele,
- infertility,
- abnormal testicular function,

repair may be considered in appropriate situations.

However, for:

NOA with maturation arrest

the evidence that varicocelectomy reliably restores sperm to the ejaculate is limited.

AUA/ASRM advises counselling NOA couples about the lack of definitive evidence for varicocele repair before ART.

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6. Lifestyle Optimization

Lifestyle does not repair:

- a complete Y deletion,
- a pathogenic meiotic gene defect.

However, reproductive-health optimization remains worthwhile.

I commonly recommend:

- stop smoking,
- avoid excessive alcohol,
- avoid recreational drugs,
- avoid anabolic steroids,
- control diabetes,
- achieve reasonable metabolic health,
- maintain regular physical activity,
- sleep adequately,
- reduce unnecessary excessive testicular heat,
- follow a balanced diet.

WHO's 2025 infertility guideline specifically emphasizes:

- healthy diet,
- physical activity,
- tobacco cessation

as part of fertility care.

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7. Antioxidants and Supplements

Patients with maturation arrest are often given many supplements:

- CoQ10,
- zinc,
- selenium,
- vitamins,
- carnitine,
- antioxidant mixtures.

Current AUA/ASRM guidance states that the benefits of antioxidant/vitamin supplements in male infertility are of:

questionable clinical utility

and evidence is insufficient to recommend specific universal agents.

This is particularly important for maturation arrest.

A supplement that slightly improves:

- motility in ejaculated sperm

is not evidence that it can restart:

genetically blocked meiosis.

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8. Empirical Hormonal Treatment Before Micro-TESE

Some clinics give:

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

to almost every NOA patient before micro-TESE.

Current evidence does not support this as universal practice.

EAU states:

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

AUA/ASRM similarly advises patients that available evidence supporting:

- SERMs,

- aromatase inhibitors,
- gonadotropins

before surgery in NOA is limited.

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9. Micro-TESE + ICSI

For a man with:

- persistent NOA,
- maturation arrest,
- no correctable hormonal cause,

the principal fertility option may be:

Micro-TESE followed by ICSI

if viable mature sperm can be retrieved.

What Is ICSI?

ICSI stands for:

Intracytoplasmic Sperm Injection

An embryologist injects:

one viable sperm directly into one mature egg.

This avoids the need for sperm to:

- travel naturally,
- penetrate cervical mucus,
- penetrate the egg independently.

However:

ICSI requires a usable sperm.

It cannot manufacture mature sperm where none exist.

Does Sperm Retrieval Mean Pregnancy Is Guaranteed?

No.

There are several stages:

1. sperm must be found,
2. eggs must be obtained,
3. ICSI must achieve fertilization,
4. embryos must develop,
5. implantation must occur,
6. pregnancy must continue,
7. live birth must occur.

Therefore:

sperm retrieval rate and pregnancy rate are not the same thing.

This distinction is particularly important when success percentages are advertised.

What If Micro-TESE Finds No Sperm?

When no sperm are identified after an appropriately performed procedure, the couple may need counselling about:

- future reassessment in selected circumstances,
- clinical trials,
- donor sperm,
- adoption,
- other family-building choices.

Repeated surgery should not automatically be performed without considering:

- histology,
- genetics,
- prior operative findings,
- testicular reserve,
- risks.

Can Stem Cells Treat Maturation Arrest?

This is an area of active research.

Scientists are investigating:

- spermatogonial stem cells,
- testicular organoids,
- in-vitro spermatogenesis,
- germ-cell differentiation.

However:

stem-cell therapy is not an established standard cure for maturation arrest in 2026.

Patients should be cautious of advertisements promising:

“Guaranteed sperm production using stem cells.”

Modern NOA research is progressing rapidly, but routine human clinical restoration of mature sperm production through stem-cell therapy remains experimental.

Can Gene Therapy Cure Genetic Maturation Arrest?

Not currently in routine clinical practice.

As genetic causes become better characterized, future treatment may become increasingly precise.

But altering germ-cell genes raises major issues involving:

- safety,
- inheritance,
- ethics.

At present, genetic diagnosis is much more established than genetic correction.

Maturation Arrest in the Unani System of Medicine

The Unani system of medicine has a longstanding tradition of addressing:

- male infertility,
- reproductive weakness,
- semen disorders,
- nutrition,

- general vitality.

However, an important scientific distinction must be made.

Classical Unani medicine developed long before:

- testicular histology,
- meiosis staining,
- Y-chromosome testing,
- gene sequencing,
- micro-TESE.

Therefore:

there is no classical Unani diagnosis that should be claimed to be an exact equivalent of biopsy-confirmed early or late maturation arrest.

This distinction should be stated clearly on a professional medical website.

Qillat-e-Haiwaniyat-Manwiya and Related Traditional Concepts

Unani literature discusses male reproductive disorders involving:

- reduced sperm/semen production,
- reproductive weakness,
- altered reproductive function.

Traditional therapeutic concepts often focus on:

- general strength,
- nutrition,
- digestion,
- Mizaj,
- reproductive-organ function.

These concepts can be useful in holistic patient assessment.

But:

Qillat-e-Haiwaniyat-Manwiya or Qillat-i-Mani should not automatically be equated with meiotic maturation arrest.

A patient with oligozoospermia still has mature sperm in the ejaculate.

A patient with complete maturation arrest may have:

no mature sperm at all.

They are biologically different conditions.

Mizaj and Individualized Care

One of the valuable principles of Unani medicine is:

Mizaj-based individualization.

In a man with infertility, I may assess:

- body constitution,
- nutrition,
- digestive health,
- sleep,
- physical activity,
- psychological health,
- sexual function,
- metabolic condition.

I combine this traditional assessment with modern information such as:

- semen analysis,
- FSH,
- LH,
- testosterone,
- genetics,
- ultrasound,
- histology.

I believe this combination gives a more complete view of the patient.

Akhlat and Modern Spermatogenesis Are Different Frameworks

Classical Unani medicine uses the traditional humoral framework of:

- Dam,
- Balgham,
- Safra,
- Sauda.

Modern medicine describes maturation arrest through:

- germ cells,
- meiosis,
- chromosomes,
- Sertoli cells,
- DNA-repair proteins,
- gene variants.

These should not be artificially equated.

For example:

a humoral imbalance is not scientifically the same thing as an MSH4 or TEX11 mutation.

Respecting both traditions requires intellectual clarity.

Asbab-e-Sitta Zarooriya and Male Fertility

Unani preventive medicine places emphasis on essential lifestyle determinants such as:

- food and drink,
- physical activity and rest,
- sleep and wakefulness,
- psychological state,
- environmental exposure,
- elimination and retention.

These principles can be relevant to general male reproductive health.

For example:

Food

supports nutritional and metabolic health.

Activity

supports weight and insulin sensitivity.

Sleep

supports endocrine health.

Environment

relates to:

- smoking,
- toxins,
- excessive heat.

Therefore, Unani lifestyle medicine can play a useful supportive role even when it cannot correct a fixed genetic maturation defect.

Ilaj-bil-Ghiza – Dietotherapy

A healthy reproductive diet may provide:

- adequate protein,
- micronutrients,
- antioxidants,
- healthy fats,
- metabolic support.

I commonly advise:

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

- appropriate hydration.

I advise reducing:

- smoking,
- excessive alcohol,
- ultra-processed food,
- heavy added sugar,
- repeated fried foods.

However:

diet cannot correct a complete genetic meiotic block.

This must be communicated honestly.

Ilaj-bit-Tadbir – Regimenal Therapy

Individualized regimenal care may include:

- exercise,
- sleep,
- weight management,
- reducing excessive heat,
- stress management,
- general health optimization.

These can help create a healthier reproductive environment.

Ilaj-bid-Dawa – Unani Pharmacotherapy

Traditional medicines may be considered according to:

- general reproductive health,
- associated semen abnormalities,
- Mizaj,
- nutrition,
- individual medical findings.

At Saira Health Care, selected male-fertility programmes may include:

Dr. Qasmi's Spermogenic Powder

and:

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

where appropriate.

Dr. Qasmi's Spermogenic Powder

Saira Health Care Pharmacy currently lists:

Spermogenic

as a Dr. Qasmi herbal formulation used for several male reproductive concerns including:

- low sperm count,
- reduced sperm motility,
- broader semen-quality problems.

The formulation includes multiple traditional herbal ingredients used in male reproductive-health care.

In a patient with:

- mild spermatogenic weakness,
- idiopathic semen abnormalities,
- poor general reproductive health,

such supportive traditional treatment may be considered as part of individualized care.

However:

the current product listing is not evidence that Spermogenic reverses biopsy-confirmed maturation arrest.

I have not identified a high-quality independent controlled study demonstrating that Spermogenic:

- restarts meiosis in maturation arrest,
- consistently converts NOA into ejaculated sperm,
- or improves live-birth rates specifically in maturation-arrest patients.

Therefore its role should be presented as:

individualized supportive male-fertility therapy—not a guaranteed maturation-arrest cure.

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

Saira Health Care Pharmacy currently describes:

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

as a Unani preparation used for:

- male problems,
- general weakness,
- vitality,
- semen-quality support.

Its public ingredient list includes:

- herbal,
- mineral

components and the pharmacy appropriately advises:

- avoiding self-medication,
- using precise physician-directed doses,
- avoiding overdose.

In my clinical framework, Nuskha No. 129 may be considered for selected patients as part of:

general reproductive and constitutional support.

However:

no current high-quality clinical evidence demonstrates that it corrects a specific genetic or histological maturation arrest.

Semen Gold Plus, Spermzoa, Sperm Plus and Ativeerya

The supplied material also refers to these formulations.

For website accuracy, their current classification should be clear.

Semen Gold Plus

Saira Health Care Pharmacy currently classifies it as an:

Ayurvedic supplement

Spermzoa Capsule

is currently described as an:

Ayurvedic formulation from Swastic Ayurveda.

Sperm Plus Capsule

is currently marketed as an:

Ayurvedic/herbal product by UA Herbal Health Care.

Ativeerya Capsule and Pouch Kit

is sold under an Ayurvedic brand category and contains multiple Ayurvedic herbs and mineral preparations.

These products may form part of an:

integrative male-fertility programme

when individually selected.

But they should not be described as classical Unani medicines or as scientifically proven treatments for histological maturation arrest.

What Does Published Unani Research Actually Show?

This question is very important.

A CCRUM-associated study evaluated:

Sufoof-e-Muallif

in **30 men with oligospermia**.

The study was:

- observational,
- open-label,
- uncontrolled,
- conducted over 60 days.

It reported improvement in:

- sperm count,
- motility,

- morphology.

This is encouraging preliminary evidence for further scientific research.

However, the study specifically excluded patients with:

organic disease of the testes and related organs of spermatogenesis.

That point is crucial.

Therefore:

this study cannot be used as evidence that Unani medicine reverses biopsy-confirmed maturation arrest.

Other Unani Oligospermia Research

CCRUM has also published retrospective literature involving men with:

idiopathic oligospermia

and traditional Unani formulations, with reported changes in semen parameters.

Again:

oligospermia is not maturation arrest.

A man with oligospermia already produces mature sperm.

A man with maturation arrest may fail to produce mature sperm in large or all areas of the testis.

This distinction must be maintained in a professional article.

Where Unani Medicine May Be Most Useful

I consider Unani supportive treatment most reasonable when the patient also has factors such as:

- nutritional weakness,
- unhealthy lifestyle,
- obesity or metabolic imbalance,
- inadequate sleep,
- associated mild semen abnormalities,
- idiopathic reproductive weakness,

- sexual-health problems.

It may also help patients improve their:

general reproductive environment

before:

- repeat semen analysis,
- ART,
- surgical sperm retrieval.

But Unani treatment should not delay:

- genetic counselling,
- micro-TESE,
- IVF/ICSI

when these are clearly indicated.

Is a 7–8 Month Treatment Required?

The supplied description mentions approximately:

7–8 months of treatment.

I would not present this as a universal medical rule.

Maturation arrest does not have one standard treatment duration.

Treatment time depends on:

- the cause,
- hormone status,
- female partner's age,
- ovarian reserve,
- whether sperm are present,
- whether ART is planned.

Because one spermatogenic cycle takes several weeks, supportive therapy is commonly assessed over:

several months

rather than days.

However:

there is no evidence that every maturation-arrest patient will complete sperm maturation after exactly seven or eight months of medicine.

Some reversible hormonal causes may respond over months.

A fixed genetic meiotic defect may not respond at all.

Dr. Nizamuddin Qasmi's Individualized Approach to Maturation Arrest

At Saira Health Care, I prefer a structured approach.

Step 1: Confirm That the Diagnosis Is Really Maturation Arrest

I ask:

- Was this diagnosis made from a testicular biopsy?
- Was it found during micro-TESE?
- Was it simply assumed because semen showed azoospermia?

Azoospermia alone does not equal maturation arrest.

Step 2: Review the Histology Carefully

I want to know:

- early arrest?
- late arrest?
- focal?
- diffuse?
- mixed with hypospermatogenesis?
- mixed with Sertoli-cell-only pattern?

These distinctions can affect prognosis.

Step 3: Reconfirm Azoospermia

I review:

- standardized semen analysis,
- centrifuged pellet examination,
- repeat testing where needed.

WHO 2025 recommends repeat semen testing after a minimum of approximately 11 weeks when one or more semen parameters are outside WHO reference ranges.

Step 4: Search for Rare Sperm

If even a few mature sperm are detected in semen:

cryopreservation may be valuable.

A patient should not assume:

“There will always be sperm in the next sample.”

Severe spermatogenesis can fluctuate.

Step 5: Assess FSH, LH and Testosterone

I distinguish:

- primary testicular failure,
- hormonal under-stimulation.

The treatment implications are completely different.

Step 6: Never Interpret FSH Alone

Normal FSH:

does not exclude maturation arrest.

High FSH:

does not prove micro-TESE will fail.

Step 7: Review Testosterone and Steroid Use

I specifically ask:

- testosterone injections?
- gym steroids?
- testosterone gel?
- bodybuilding hormones?

If present, fertility suppression may be potentially reversible.

Step 8: Perform Genetic Evaluation When Indicated

This may include:

- karyotype,
- Y-chromosome microdeletion analysis,
- specialist genetic testing.

This is particularly important before:

micro-TESE

because some genetic findings dramatically change the retrieval prognosis.

Step 9: Look for Other Reversible Problems

I assess:

- clinical varicocele,
- endocrine disease,
- metabolic health,
- medication effects,
- severe testicular heat exposure.

Step 10: Evaluate the Female Partner at the Same Time

This is essential.

If the female partner is:

- 38 years old,
- has low ovarian reserve,

I would not advise waiting indefinitely for a man's semen to improve.

The fertility plan belongs to:

the couple.

Step 11: Introduce Individualized Unani Support Where Appropriate

Depending on the case, supportive treatment may include:

- Ilaj-bil-Ghiza,
- Ilaj-bit-Tadbir,
- selected Unani pharmacotherapy,
- Spermogenic,
- Nuskha No. 129.

The aim is:

to optimize the patient's reproductive health and reversible factors

rather than promising to overcome every genetic meiotic block.

Step 12: Monitor Objective Findings

Treatment response should be judged using:

- semen analysis,
- hormones where relevant,
- clinical findings,
- appearance of sperm in ejaculate,
- sperm retrieval,
- reproductive outcome.

I do not consider statements such as:

“The patient feels stronger”

sufficient proof that:

maturation arrest has reversed.

Step 13: Consider Micro-TESE at the Appropriate Time

If:

- azoospermia persists,
- no medically reversible cause exists,
- the couple wishes biological parenthood,

micro-TESE should be discussed with an experienced reproductive-urology/ART team.

Step 14: Do Not Delay ICSI When Viable Sperm Are Found

If sperm are successfully retrieved:

- fresh,
- or cryopreserved

sperm may be used for ICSI according to the fertility centre's plan.

AUA/ASRM allows either fresh or cryopreserved retrieved sperm for ICSI where adequate viable sperm remain.

Step 15: Provide Realistic Counselling

This is one of the most important parts of treatment.

I explain:

- success cannot be guaranteed,
- sperm retrieval does not equal pregnancy,
- treatment outcome depends on both partners.

Hope should be:

realistic rather than artificial.

Success Stories: How They Should Be Presented Responsibly

Patients understandably want to read:

success stories.

Stories can provide encouragement.

But in a professional fertility article, I do not believe anonymous statements such as:

"Hundreds of maturation-arrest patients were cured."

should be presented without:

- diagnostic evidence,
- histology,
- semen results,
- treatment details,
- documented outcomes.

A genuine maturation-arrest success story should ideally record:

1. confirmation of azoospermia,
2. hormone profile,
3. genetic evaluation where appropriate,
4. histological diagnosis,
5. early/late and focal/diffuse pattern,
6. treatment received,
7. post-treatment semen analysis,
8. sperm retrieval findings,
9. IVF/ICSI outcome,
10. pregnancy/live birth where available,
11. patient consent for publication.

What Published Success Data Tell Us

The best-known maturation-arrest micro-TESE series demonstrates something important:

maturation arrest is not automatically synonymous with zero sperm-retrieval potential.

Among 211 men:

- sperm were retrieved in about half overall,
- late arrest had better retrieval than early arrest,
- focal arrest had better retrieval than diffuse arrest.

These scientific outcomes can give patients genuine hope without inventing treatment claims.

What I Would Call a Successful Outcome

Success does not always mean:

“Medicine produced sperm naturally.”

For one patient, success may mean:

A hormonal cause was discovered and treated.

For another:

Testosterone injections were stopped and sperm returned.

For another:

Genetic testing identified complete AZFa deletion and prevented unnecessary micro-TESE.

For another:

Micro-TESE identified viable sperm despite azoospermia.

For another:

One retrieved sperm enabled ICSI and pregnancy.

For another:

Accurate counselling prevented years of ineffective treatment.

All of these represent:

meaningful fertility care.

Saira Health Care's Contribution to Male Infertility and Maturation Disorders

At **Saira Health Care**, my focused work is in:

Sexual Disorders & Infertility.

Saira Health Care's current professional profile identifies me as:

Dr. Nizamuddin Qasmi, Founder & Chief Physician

with a focused clinical practice in sexual disorders and infertility, and publicly lists the requested professional education and training including:

- 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.

Why Additional Male-Infertility Training Is Relevant

Maturation arrest sits at the intersection of:

- andrology,
- reproductive endocrinology,
- genetics,
- urology,
- ART,
- traditional reproductive medicine.

A clinician dealing with these patients must understand that:

“zero sperm” is not one diagnosis.

It may represent:

- obstruction,
- hormonal suppression,
- genetic failure,
- maturation arrest,
- Sertoli-cell-only syndrome,
- focal spermatogenesis.

This is why focused male-infertility training is particularly relevant.

Saira Health Care's Integrative Philosophy

My philosophy at Saira Health Care is:

diagnosis first, individualized treatment second.

Modern medicine helps identify:

- azoospermia,
- hormones,
- genetics,
- histology,
- focal sperm production.

Unani medicine can contribute through:

- diet,
- lifestyle,
- individualized constitutional assessment,
- selected supportive pharmacotherapy.

Reproductive surgery can provide:

- micro-TESE,
- varicocele management where appropriate.

ART can provide:

- IVF,
- ICSI.

These approaches should work together rather than competing unnecessarily.

Why I Do Not Promise a Fixed "Cure Rate"

Maturation arrest caused by:

hormonal suppression

is biologically different from maturation arrest caused by:

complete AZFb deletion.

A focal late maturation arrest is different from:

diffuse early maturation arrest.

It would therefore be scientifically inappropriate to promise one fixed success percentage for:

- every patient,
- every medicine,
- every treatment system.

Psychological Impact of Maturation Arrest

The emotional burden can be significant.

A man may feel:

- inadequate,
- ashamed,
- frightened,
- guilty.

I want to tell patients clearly:

Maturation arrest does not define masculinity.

A microscopic testicular developmental problem says nothing about:

- sexual worth,
- strength,
- character,
- ability to be a supportive partner.

WHO's current infertility guidance recognizes the psychological burden of infertility and emphasizes the importance of appropriate counselling and psychosocial support.

Common Myths About Maturation Arrest

Myth 1: Maturation arrest means every sperm in the body is dead.

Fact: It means germ-cell development has stopped at a stage in affected tubules. Dead sperm and maturation arrest are different conditions.

Myth 2: Low sperm motility means maturation arrest.

Fact: Motility is mainly a functional property of mature sperm. Maturation arrest is a testicular developmental problem.

Myth 3: Low sperm count automatically means maturation arrest.

Fact: Many causes of oligozoospermia exist without histological arrest.

Myth 4: Azoospermia automatically means maturation arrest.

Fact: Azoospermia can be obstructive or caused by many different NOA patterns.

Myth 5: Normal FSH rules out maturation arrest.

Fact: FSH may remain normal when arrest occurs at spermatocyte or spermatid stages.

Myth 6: High FSH means micro-TESE is useless.

Fact: High-FSH NOA patients may still have focal sperm production.

Myth 7: Testosterone injections improve sperm maturation.

Fact: Exogenous testosterone can suppress spermatogenesis and cause azoospermia.

Myth 8: Every maturation arrest can be treated with FSH injections.

Fact: Gonadotropins are strongly useful in true hypogonadotropic hypogonadism. Their benefit in idiopathic NOA/maturation arrest is much less certain.

Myth 9: Every varicocele causes maturation arrest.

Fact: Varicocele can impair testicular function in some men, but it is not the universal cause of maturation arrest.

Myth 10: Every maturation arrest improves after seven or eight months.

Fact: Treatment duration and response depend on the underlying cause. No universal seven- or eight-month cure can be promised.

Myth 11: One herbal medicine can restart meiosis in every patient.

Fact: Genetic meiotic arrest cannot currently be assumed to respond to any universal herbal formulation.

Myth 12: Published Unani oligospermia studies prove maturation-arrest cure.

Fact: The CCRUM study commonly cited involved oligospermia and excluded organic testicular disease.

Myth 13: Micro-TESE always finds sperm in late maturation arrest.

Fact: Published retrieval rates are better in late than early arrest, but no individual procedure can be guaranteed.

Myth 14: Sperm retrieval guarantees pregnancy.

Fact: Retrieval is only one step before ICSI, embryo development, implantation and live birth.

Frequently Asked Questions

What is maturation arrest?

It is a pattern of impaired spermatogenesis where germ-cell development stops before mature sperm production is completed.

Is maturation arrest a semen-analysis diagnosis?

No.

Semen analysis may show:

- azoospermia,
- severe oligozoospermia,

but true maturation arrest is fundamentally a:

testicular histological diagnosis.

What is early maturation arrest?

It commonly refers to arrest at an earlier germ-cell stage such as:

primary spermatocytes

before successful completion of meiosis.

What is late maturation arrest?

Development progresses further, often to:

spermatid stages

before mature sperm production fails.

Which is better: early or late maturation arrest?

Published micro-TESE series generally show a better sperm-retrieval prognosis with:

late maturation arrest.

One large series reported 40% retrieval in early versus 78% in late arrest, but these numbers should not be used as personal guarantees.

What is focal maturation arrest?

Some testicular regions show arrest while others may have more advanced sperm development.

What is diffuse maturation arrest?

Maturation arrest is present throughout essentially all sampled tubules.

Can FSH be normal?

Yes.

This is particularly possible with spermatocyte/spermatid-stage arrest.

Can testosterone be normal?

Yes.

Many men with maturation arrest have normal serum testosterone and normal sexual function.

Can a man with maturation arrest have normal erection?

Absolutely.

Erection and sperm production are different processes.

Can sperm return naturally?

Sometimes, especially if the underlying issue is:

- hormonal suppression,
- medication/steroid related,
- transient acquired dysfunction.

But fixed genetic maturation arrest may not be reversible.

Can hCG and FSH help?

They are especially appropriate when the cause is:

hypogonadotropic hypogonadism.

They are not universally proven to reverse idiopathic or genetically determined maturation arrest.

Should I take testosterone for low testosterone and infertility?

Do not start testosterone monotherapy when trying to conceive without specialist guidance.

External testosterone can suppress sperm production.

Does varicocele surgery cure maturation arrest?

Not reliably.

NOA patients with clinical varicocele should be counselled that definitive evidence for repair before ART remains limited.

Can micro-TESE find sperm?

Yes, in selected men.

The probability depends on:

- histology,
- genetics,
- focal sperm production,
- individual biology.

What was the published retrieval rate in maturation arrest?

One major retrospective series reported:

- overall: **52%**
- early MA: **40%**
- late MA: **78%**
- diffuse MA: **35%**
- focal MA: **57%**.

These are study data, not guaranteed individual outcomes.

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

Current guidelines advise against retrieval surgery because successful sperm retrieval is essentially not expected.

Can AZFc deletion still have sperm?

Yes.

Current AUA/ASRM evidence indicates sperm may be found with micro-TESE in approximately half of azoospermic men with isolated AZFc deletion.

Is genetic testing necessary?

It is particularly important in selected men with:

- azoospermia,
- severe oligozoospermia,
- signs of impaired testicular sperm production.

Can Unani medicine help maturation arrest?

Unani medicine may provide useful supportive care through:

- individualized nutrition,
- Ilaj-bil-Ghiza,
- Ilaj-bit-Tadbir,
- lifestyle optimization,
- selected reproductive pharmacotherapy.

However, current evidence does not demonstrate that Unani medicine reverses all biopsy-confirmed maturation arrest.

What is the role of Spermogenic?

Spermogenic is a Dr. Qasmi herbal formulation currently used within Saira Health Care's male-fertility programmes for several semen-quality concerns.

Its role in maturation arrest should be considered:

supportive and individualized

rather than a scientifically proven universal cure.

What is Nuskha No. 129?

Dr. Qasmi's Nuskha No. 129 – Vitasem Max is currently described by Saira Health Care Pharmacy as a Unani preparation for:

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

Is Semen Gold Plus Unani?

The current Saira Health Care Pharmacy listing classifies it as:

Ayurvedic.

Is Spermzoa Unani?

The current pharmacy listing classifies it as an:

Ayurvedic formulation.

Is Sperm Plus Unani?

The current listing describes it as an:

Ayurvedic/herbal formulation.

Does CCRUM research prove Unani treatment cures maturation arrest?

No.

The available study was in men with:

oligospermia

and excluded organic testicular disease related to spermatogenesis.

Can stem cells cure maturation arrest?

Not as established standard treatment in 2026.

Stem-cell and in-vitro spermatogenesis research remain experimental.

Latest Scientific Perspective: 2025–2026

Modern understanding of maturation arrest is changing rapidly.

WHO's First Global Infertility Guideline

WHO published its first comprehensive global infertility guideline on:

28 November 2025

covering:

- prevention,
- diagnosis,
- male and female infertility,
- ART,
- psychosocial care.

For abnormal semen findings, WHO now recommends repeat testing after at least:

11 weeks

in most appropriate cases.

Genetics Is Becoming Central

A 2025 review emphasizes that NOA is not one disorder but a genetically heterogeneous group involving:

- chromosomal defects,
- single-gene mutations,
- epigenetic abnormalities.

Genes affecting:

- meiosis,
- DNA repair,
- chromosome structure

are increasingly being connected with maturation arrest.

This means future male-infertility care will become more:

genotype-specific.

Modern Genetic Testing Can Change Treatment Decisions

A man with:

complete AZFa deletion

should not undergo years of empirical therapy followed by futile micro-TESE.

A man with:

isolated AZFc deletion

has a very different sperm-retrieval prognosis.

This is personalized infertility medicine.

Better Sperm-Retrieval Prediction Is Being Studied

A 2025 multicentre study of 333 men with idiopathic NOA evaluated:

- FSH,
- testicular volume,
- histology

to predict micro-TESE outcomes and found maturation arrest and Sertoli-cell-only histology to be important negative predictors compared with more favorable patterns.

No available prediction model is accurate enough to guarantee the result before surgery.

New Biomarkers Are Experimental

Recent 2025 studies are also investigating potential biomarkers such as:

- seminal angiotensin II

to predict spermatogenic activity and micro-TESE success.

One prospective study reported higher levels in men with late maturation arrest than in Sertoli-cell-only syndrome.

This remains research—not routine clinical testing.

The Future

Future treatment may involve:

- better genetic panels,
- molecular profiling,
- spermatogonial stem-cell technology,
- in-vitro spermatogenesis,
- more accurate sperm-retrieval prediction.

For now:

accurate diagnosis, genetic counselling, cause-specific treatment, micro-TESE and ICSI remain the clinically relevant tools.

About the Author

Dr. Nizamuddin Qasmi

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

Professional Education & Training

- **BUMS – Hamdard University, Delhi**
- **MD**
- **CGO**
- **Certificate in Infertility – MGBIMS, Delhi**
- **Certificate in Urology – London, UK**
- **Masters in Male Infertility – MasterHealthPro (HealthPro)**
- **Integrated Sexual and Reproductive Health – ISRH, UNFPA**

Saira Health Care's current published professional profile identifies Dr. Nizamuddin Qasmi as Founder and Chief Physician with a focused clinical practice in **sexual disorders and infertility** and lists the above infertility, urology and reproductive-health education used in its professional articles.

My approach combines:

- Unani medical principles,
- male-infertility assessment,
- semen-analysis interpretation,
- reproductive hormonal evaluation,
- genetics awareness,
- sexual-health care,
- lifestyle optimization,
- and timely referral for assisted reproduction or reproductive surgery.

My Final Message to Patients With Maturation Arrest

If you have been diagnosed with:

Maturation Arrest

please do not conclude immediately:

“I can never become a biological father.”

But do not believe anyone who says:

“One medicine will definitely restart every maturation arrest.”

Ask instead:

Was the diagnosis confirmed by histology?

Is it early or late maturation arrest?

Is it focal or diffuse?

Has azoospermia been confirmed properly?

Were rare sperm searched for in the centrifuged semen pellet?

What is my FSH?

What is my LH?

What is my testosterone?

Am I using testosterone or anabolic steroids?

Do I need a karyotype?

Do I need Y-chromosome microdeletion testing?

Could the problem be hormonal and reversible?

Is there a clinical varicocele?

Could micro-TESE find focal sperm?

What is the fertility status and age of my wife?

Can individualized Unani supportive treatment improve my overall reproductive health?

When should we proceed to micro-TESE and ICSI?

These are the questions that lead to scientifically responsible treatment.

Conclusion

Male maturation arrest is a significant form of:

spermatogenic failure

in which developing germ cells fail to progress normally into mature spermatozoa.

It is typically a:

histological pattern

found particularly in men with:

non-obstructive azoospermia.

Maturation arrest can be:

- early,
- late,
- focal,
- diffuse.

Early arrest commonly occurs around the:

- primary-spermatocyte stage.

Late arrest occurs after germ cells have progressed further, often to:

- spermatid stages.

The underlying causes may include:

- genetic abnormalities,
- chromosomal disorders,
- Y-chromosome microdeletions,
- hormonal disease,
- exogenous testosterone,
- anabolic steroids,
- testicular injury,
- chemotherapy,
- radiation,
- varicocele,
- environmental factors,

- unexplained causes.

Diagnosis may require:

- repeated standardized semen analysis,
- hormone testing,
- reproductive examination,
- appropriate genetic testing,
- and testicular histology obtained in selected clinical circumstances.

Normal FSH does not rule out maturation arrest.

High FSH does not prove that no focal sperm production exists.

Cause-specific treatment is essential.

For:

hypogonadotropic hypogonadism

gonadotropin therapy can induce spermatogenesis and is supported by current guidelines.

External testosterone should not be used as fertility treatment because it can suppress sperm production.

For persistent NOA where biological fatherhood is desired:

micro-TESE followed by ICSI

is a major established fertility option.

In a large published maturation-arrest series, sperm retrieval was better in:

- late than early arrest,
- focal than diffuse arrest,

but individual success cannot be guaranteed.

The **Unani system of medicine** can offer valuable supportive care through:

- Mizaj-based individualization,
- Ilaj-bil-Ghiza,
- Ilaj-bit-Tadbir,
- nutritional optimization,
- lifestyle correction,

- supervised pharmacotherapy.

At **Saira Health Care**, formulations including:

- **Dr. Qasmi's Spermogenic Powder**
- **Dr. Qasmi's Nuskha No. 129 – Vitasem Max**

may be used within selected individualized male reproductive-health programmes.

However:

current evidence does not establish these formulations as proven cures for biopsy-confirmed maturation arrest.

Published CCRUM Unani evidence is mainly related to:

oligospermia

and one commonly cited study specifically excluded organic testicular disorders of spermatogenesis.

Therefore, I believe the most scientifically responsible integrative approach is:

Confirm the diagnosis.

Differentiate early from late arrest.

Understand focal versus diffuse disease.

Check for reversible hormonal suppression.

Stop testosterone and anabolic steroids where relevant.

Investigate genetic causes appropriately.

Do not interpret FSH alone as the prognosis.

Improve diet, lifestyle and general reproductive health.

Use Unani medicine rationally as individualized supportive treatment.

Do not delay micro-TESE/ICSI when it is medically appropriate.

Evaluate the female partner at the same time.

And never guarantee a fixed duration, sperm-retrieval rate, pregnancy rate or cure from one medicine.

For every patient who asks me:

“Doctor, sperm maturation has stopped. Is there still a chance?”

my answer is:

There may still be meaningful options. In some men the underlying cause is treatable; in others, focal mature sperm may still be found through micro-TESE even when no sperm appear in the semen. The key is to determine exactly where and why sperm development has stopped, and then choose the treatment according to that diagnosis rather than relying on a generic fertility prescription.

Selected Medical References

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Medical Disclaimer

This article is intended for:

- patient education,
- medical awareness,
- reproductive-health information.

It is not a substitute for:

- individualized consultation,
- semen analysis,
- hormonal evaluation,
- genetic counselling,
- urological examination,
- testicular histopathology,
- micro-TESE consultation,
- IVF/ICSI assessment.

Maturation arrest should not be self-diagnosed from:

- low sperm count,
- low motility,
- abnormal morphology,
- or one semen report.

Do not independently start:

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

without appropriate professional guidance.

Dr. Qasmi's Spermogenic, Nuskha No. 129, Semen Gold Plus, Spermzoa, Sperm Plus, Ativeerya or other fertility formulations should not be interpreted as guaranteed treatments for biopsy-confirmed maturation arrest.

There is no scientifically established universal:

- seven-month,
- eight-month,
- or other fixed-duration cure

for maturation arrest.

Men with:

- azoospermia,
- early or late maturation arrest,
- testicular atrophy,
- abnormal FSH,
- suspected genetic disease,
- Y-chromosome abnormality,
- previous failed sperm retrieval

should receive specialist male-fertility evaluation.

Where:

- gonadotropin therapy,
- genetic counselling,
- micro-TESE,
- IVF,
- or ICSI

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

No medical, Unani, surgical or ART treatment can guarantee:

- sperm production,
- sperm retrieval,
- fertilization,

- pregnancy,
- live birth.

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