Klinefelter Syndrome and Male Infertility

Understanding 47,XXY, Azoospermia, Low Testosterone, Sperm Retrieval, ICSI and an Integrative Unani Approach

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When a man comes to me with infertility and the semen report repeatedly shows **azoospermia**, one of the important questions is whether there may be an underlying genetic cause.

Sometimes the patient has very small testes, increased FSH, reduced testosterone or gynecomastia. In other men there may be almost no obvious physical signs, and the diagnosis is discovered only after a chromosome test is performed during infertility evaluation.

One of the most important genetic causes is **Klinefelter syndrome**.

The most common chromosome pattern in Klinefelter syndrome is:

47,XXY

instead of the usual male chromosome pattern of:

46,XY.

This means there is one additional X chromosome.

Klinefelter syndrome is one of the most common sex-chromosome conditions in males and one of the most important chromosomal causes of male infertility. Current NIH information estimates a frequency of roughly **1 in 650 male newborns**, although many affected men remain undiagnosed because the clinical features can be mild.

For many patients, infertility is the first reason the condition is discovered.

My first message to a patient diagnosed with Klinefelter syndrome is therefore:

This diagnosis explains an important part of your reproductive biology, but it does not define your masculinity, your sexual worth or automatically eliminate every possibility of biological fatherhood.

Most men with the classic non-mosaic 47,XXY form produce little or no sperm in the ejaculate. However, modern reproductive surgery can sometimes identify small areas of sperm production within the testes, and these sperm may potentially be used for **ICSI—Intracytoplasmic Sperm Injection**. Current AUA/ASRM guidance estimates that sperm may be found by microdissection testicular sperm extraction in approximately **50–60%**

of selected 47,XXY men, although newer pooled studies have reported somewhat lower overall figures depending on the population and surgical method.

At **Saira Health Care**, my approach is therefore neither to offer false reassurance nor to tell the patient immediately that biological fatherhood is impossible.

The correct approach is:

confirm the diagnosis, understand remaining testicular function, address hormonal health, evaluate fertility carefully, discuss genetic counselling and use modern reproductive techniques where appropriate.

What Is Klinefelter Syndrome?

Klinefelter syndrome is a chromosomal condition affecting males who have one or more additional X chromosomes.

The most common form is:

47,XXY.

Typical males have:

46 chromosomes, including one X chromosome and one Y chromosome.

In Klinefelter syndrome, the person usually has:

47 chromosomes, including two X chromosomes and one Y chromosome.

The additional X chromosome changes testicular development and several other aspects of health.

The testes usually become relatively small and firm, sperm-producing tissue progressively deteriorates, and testosterone production may become insufficient.

The condition therefore commonly leads to:

primary testicular insufficiency, hypergonadotropic hypogonadism and male infertility.

The European Academy of Andrology describes severely impaired spermatogenesis and Leydig-cell dysfunction as central features of Klinefelter syndrome.

Klinefelter Syndrome Is a Genetic Condition—but Usually Not an Inherited Family Disorder

Patients frequently ask:

“Doctor, did I get this disease from my father or mother?”

In most cases, Klinefelter syndrome occurs because of a **random chromosome-separation error** during formation of the egg or sperm.

It is generally not inherited in the usual manner from one generation to another.

During reproductive-cell formation, chromosomes are supposed to separate correctly.

If this separation does not occur normally—a process called **nondisjunction**—a reproductive cell can contain an additional X chromosome.

After fertilization, the resulting embryo may therefore have the chromosome pattern 47,XXY.

NIH genetic information describes Klinefelter syndrome as usually arising from a random chromosomal error rather than being directly inherited.

Nothing the parents intentionally did caused the condition.

Mosaic Klinefelter Syndrome

Not every patient has an additional X chromosome in every cell.

Some people have:

46,XY cells in part of the body and 47,XXY cells in another part.

This is called **mosaic Klinefelter syndrome**, commonly written:

46,XY/47,XXY.

Mosaic patients may have milder features.

Some may have larger testes, higher testosterone levels or better residual sperm production than men with non-mosaic 47,XXY.

However, mosaicism varies considerably.

A patient should therefore not assume fertility is normal simply because the word “mosaic” appears on the chromosome report.

Actual reproductive function still needs semen and hormonal assessment.

Variants With More Than One Additional X Chromosome

Less common chromosome patterns include:

48,XXXY,
48,XXYY,
and **49,XXXXY**.

These are not identical to typical 47,XXY Klinefelter syndrome and may produce more pronounced developmental, physical or neurological features.

The classic infertility discussion usually concerns **47,XXY**, which is by far the most common form. The AUA/ASRM male-infertility guideline identifies 47,XXY as the predominant Klinefelter chromosome pattern encountered in infertility practice.

Why Does Klinefelter Syndrome Cause Infertility?

The additional X chromosome alters normal testicular development.

The problem is not simply that testosterone is low.

Inside the testes, the seminiferous tubules—the structures responsible for sperm production—undergo progressive degeneration.

Changes can include:

loss of germ cells, Sertoli-cell dysfunction, seminiferous-tubule fibrosis and hyalinization, and deterioration of normal testicular architecture.

As a result, sperm production becomes severely reduced.

A recent 2024 review describes Klinefelter infertility as the consequence of complex genetic, meiotic, Sertoli-cell, Leydig-cell and epigenetic abnormalities resulting from the supernumerary X chromosome.

This explains why giving a general “sperm booster” cannot correct the fundamental cause.

Why Are the Testes Usually Small?

One of the characteristic clinical findings is **small, firm testes**.

Normal testes contain large amounts of active seminiferous-tubule tissue.

In Klinefelter syndrome, much of this tissue is progressively damaged and replaced by fibrotic tissue.

The testes therefore remain small even though puberty may otherwise appear reasonably normal.

Small testicular volume in an infertile man is an important clue, especially when accompanied by elevated FSH and LH.

What Is Hypergonadotropic Hypogonadism?

This is an important medical term in Klinefelter syndrome.

Hypogonadism means inadequate testicular hormonal function.

Hypergonadotropic means that the pituitary hormones FSH and LH are elevated.

The brain senses that the testes are not functioning adequately.

It therefore produces more:

FSH — Follicle-Stimulating Hormone
and
LH — Luteinizing Hormone

in an attempt to stimulate them.

The typical adult hormonal pattern therefore may show:

high FSH, high LH and low or low-normal testosterone.

This pattern is called **primary hypergonadotropic hypogonadism**.

It tells us that the problem lies primarily within the testes rather than a lack of stimulation from the pituitary gland.

FSH and Sperm Production

FSH is often substantially elevated in adult men with Klinefelter syndrome.

This reflects severe disturbance of the Sertoli cells and sperm-producing compartment.

A patient may ask:

“My FSH is 28. Can we lower the FSH to produce sperm?”

The high FSH is usually **a consequence of testicular dysfunction**, not the primary disease itself.

Simply lowering the number on the laboratory report does not restore destroyed seminiferous tissue.

This distinction prevents unnecessary hormonal treatment.

LH and Testosterone

LH stimulates Leydig cells within the testes to produce testosterone.

In Klinefelter syndrome, Leydig-cell function may gradually become impaired.

The pituitary therefore produces more LH.

Some patients maintain testosterone within the lower part of the normal range for years.

Others develop clear testosterone deficiency.

Symptoms may include reduced libido, reduced muscle mass, reduced body or facial hair, fatigue, lower bone density and erectile difficulties in some patients.

Not every patient has all of these symptoms.

Klinefelter Syndrome Can Be Very Mild

The stereotypical image of Klinefelter syndrome is a very tall man with obvious gynecomastia and absent facial hair.

That picture can be misleading.

Many affected men look completely ordinary.

Some have normal masculine development and normal sexual relationships.

This is one reason diagnosis is frequently delayed.

NIH estimates that a large proportion of people with Klinefelter syndrome remain undiagnosed, and contemporary reviews continue to describe underdiagnosis as a major problem.

In fertility practice, therefore, chromosome testing is sometimes what reveals the condition.

Common Physical and Developmental Features

Features vary greatly between individuals.

Some men may have increased height and relatively long legs, small testes, reduced facial or body hair, gynecomastia, reduced muscle mass or increased abdominal fat.

Children may occasionally have language, educational or coordination difficulties.

Others have almost none of these obvious signs.

A diagnosis should never be made from appearance alone.

The chromosome test is what establishes the diagnosis.

Klinefelter Syndrome and Puberty

Most boys with Klinefelter syndrome begin puberty spontaneously.

The onset of puberty may occur at the expected age.

However, as puberty progresses, testicular growth remains limited and testosterone production may eventually become inadequate.

The European Academy of Andrology recommends monitoring pubertal development, testosterone and gonadotropins from the peripubertal period onward in diagnosed patients.

Early diagnosis therefore allows more organized monitoring of:

pubertal development, hormone status, fertility concerns, bone health and metabolic health.

Gynecomastia

Gynecomastia means development of breast glandular tissue in males.

It may occur because of the altered androgen–estrogen balance associated with Klinefelter syndrome.

Gynecomastia can cause:

physical discomfort, embarrassment or reduced body confidence.

Persistent significant gynecomastia can be evaluated medically and, in selected men, surgically.

It is also relevant because Klinefelter syndrome is associated with a substantially greater **relative risk of male breast cancer** than the general male population, although the absolute risk remains much lower than that of breast cancer in women. A 2024 systematic review found a markedly increased incidence compared with males without Klinefelter syndrome.

Any new persistent breast lump, nipple change or unexplained discharge therefore deserves assessment.

Klinefelter Syndrome and Male Infertility

Infertility is one of the most characteristic adult manifestations.

Most men with classic non-mosaic 47,XXY have **azoospermia**.

Azoospermia means:

no sperm are detected in the ejaculate after appropriate semen examination.

A much smaller proportion have:

severe oligospermia or occasional sperm within the semen.

The European Academy of Andrology reports ejaculated sperm suitable for cryopreservation in only a minority of affected adult men.

This is why semen analysis should be performed early in an adult with Klinefelter syndrome who desires biological fatherhood.

Azoospermia Does Not Necessarily Mean There Is No Sperm Anywhere in the Testis

This is perhaps the most important fertility concept for Klinefelter syndrome.

Sperm production within the testes may not be uniform.

Most seminiferous tubules may have lost their sperm-producing capability.

But occasionally small isolated areas—known as **foci of spermatogenesis**—remain capable of producing sperm.

These sperm may never reach the ejaculate in detectable numbers.

However, microsurgical exploration can sometimes locate them.

This is the biological basis for **micro-TESE**.

What Is Micro-TESE?

Micro-TESE stands for:

Microdissection Testicular Sperm Extraction.

It is a microsurgical fertility procedure designed particularly for men with **non-obstructive azoospermia**.

Under an operating microscope, an experienced reproductive urologist examines the testicular tissue and selectively samples seminiferous tubules that appear relatively more promising.

The laboratory then searches the tissue carefully for viable sperm.

Current AUA/ASRM guidance recommends microdissection TESE as the preferred surgical sperm-retrieval technique for men with non-obstructive azoospermia.

How Often Can Sperm Be Found in Klinefelter Syndrome?

This question deserves careful explanation because published rates vary.

The 2024 AUA/ASRM guideline states that small areas of sperm production may permit sperm retrieval in approximately **50–60% of selected 47,XXY men undergoing micro-TESE**.

However, a large 2025 systematic review and meta-analysis involving **2,815 patients** reported a median sperm-retrieval rate of approximately **44%** across 48 studies.

These figures are not contradictory.

Different studies use:

different patient populations, surgical approaches, laboratory expertise, ages and definitions.

Therefore, I do not tell an individual patient:

“You have exactly a 50% chance.”

The realistic message is:

sperm retrieval is possible in a substantial proportion of appropriately selected men with Klinefelter syndrome, but success cannot be predicted or guaranteed for an individual patient.

Can Sperm Be Found Naturally in the Ejaculate?

Occasionally, yes.

Some men—particularly mosaic cases—may have very small numbers of ejaculated sperm.

For this reason, semen analysis should be performed before surgery.

If viable sperm are found, they can potentially be **cryopreserved**, meaning frozen for future assisted reproduction.

European guidance recommends semen analysis and sperm cryopreservation in adult men with Klinefelter syndrome who desire future paternity.

This can sometimes avoid the need for surgical sperm retrieval.

What Is ICSI?

When very few sperm are available, ordinary natural fertilization is unlikely.

The technique most commonly used is:

ICSI — Intracytoplasmic Sperm Injection.

During ICSI, the embryologist selects one viable sperm and injects it directly into a mature egg.

The resulting embryo, if it develops appropriately, may later be transferred into the uterus through an IVF cycle.

Micro-TESE and ICSI therefore work together:

micro-TESE finds the sperm; ICSI helps the sperm fertilize the egg.

Can Men With Klinefelter Syndrome Become Biological Fathers?

Yes, some can.

Before the development of TESE and ICSI, Klinefelter syndrome was generally considered to result in permanent biological infertility.

That understanding has changed.

Men with rare ejaculated sperm may use cryopreserved semen.

Other azoospermic men may have testicular sperm recovered surgically.

These sperm may then be used with ICSI.

A recent clinical review from Denmark reported sperm retrieval in 42% of 93 surgically treated men with Klinefelter syndrome, with multiple children subsequently born following fertility treatment.

Therefore, the diagnosis should not automatically be translated into:

“You can never have your own biological child.”

But treatment remains complex and outcomes are not guaranteed.

Latest Evidence About Age and Sperm Retrieval

For many years there has been considerable debate about whether sperm must be surgically retrieved during adolescence because germ cells progressively disappear.

The most recent large evidence is important.

A **2025 systematic review and meta-analysis** compared sperm retrieval in adolescent and adult men with non-mosaic 47,XXY Klinefelter syndrome.

The analysis included 48 studies and 2,815 participants.

Average sperm-retrieval rates were approximately:

45% in adolescent cohorts
and
42% in adult cohorts.

The difference was not clinically significant.

The researchers concluded that surgical sperm retrieval can generally be delayed into reproductive adulthood without clearly reducing the probability of successful sperm retrieval, although there was a possible decline at older ages beyond approximately 40 years.

This is a very important modern development.

It means we should be cautious about exposing every adolescent boy with Klinefelter syndrome to invasive testicular surgery simply out of fear that sperm will disappear immediately after puberty.

Fertility Preservation in Adolescents

Fertility counselling should still begin during adolescence when appropriate.

But counselling is different from automatically performing surgery.

An adolescent mature enough to understand fertility preservation may first have:

hormonal evaluation and semen analysis.

If sperm are found in the ejaculate, cryopreservation can be considered.

Routine testicular biopsy of young children is not recommended.

European Academy of Andrology guidance specifically recommends **against cryopreservation of testicular tissue or spermatogonial stem cells in prepubertal children with Klinefelter syndrome**, because these approaches remain experimental and there is currently no established method for producing mature sperm from such stored tissue for clinical use.

The decision in adolescents should therefore be individualized and involve:

the patient, parents where appropriate, endocrinologist, reproductive urologist and fertility specialist.

Why Fertility Counselling Should Happen Before Testosterone Treatment When Possible

This is an especially important area.

Men with Klinefelter syndrome may need testosterone treatment because of hypogonadism.

Testosterone therapy can improve symptoms of testosterone deficiency.

However, external testosterone can suppress pituitary gonadotropins and potentially suppress any remaining spermatogenesis.

European guidance therefore recommends addressing fertility status **before initiating testosterone replacement whenever possible**, particularly when TESE is being planned.

The AUA/ASRM male infertility guideline similarly states that testosterone monotherapy should not be prescribed to men actively interested in current or future fertility without considering its suppressive effect on sperm production.

Testosterone Treatment Is Still Important When Hypogonadism Is Present

The previous section does **not** mean that men with Klinefelter syndrome should be denied testosterone indefinitely.

Low testosterone has health consequences.

When fertility considerations have been appropriately addressed, testosterone replacement can be important for men with clinically significant hypogonadism.

Benefits may include improvement in:

sexual desire, energy, muscle mass, body composition, bone health and development or maintenance of secondary sexual characteristics.

The European Academy of Andrology recommends testosterone replacement in appropriately diagnosed hypogonadal men with Klinefelter syndrome, using standard monitoring protocols.

Therefore:

fertility planning and testosterone treatment need coordination—not competition.

Testosterone Is Not a Fertility Treatment

A patient may understandably think:

“If Klinefelter syndrome causes low testosterone, increasing testosterone should increase sperm.”

Unfortunately, external testosterone does not work that way.

Sperm production depends on very high concentrations of testosterone generated **inside the testes under LH stimulation**.

Injecting or applying testosterone from outside the body suppresses LH and FSH.

This may further reduce any remaining sperm production.

Therefore:

testosterone can treat androgen deficiency, but it is not a sperm-producing medicine.

The distinction is fundamental.

Can hCG, Clomiphene or Aromatase Inhibitors Improve Sperm Retrieval?

Various fertility specialists have used medicines such as:

hCG, clomiphene or aromatase inhibitors

before surgical sperm retrieval in selected men with Klinefelter syndrome.

The theoretical purpose is to optimize endogenous testosterone production without suppressing gonadotropins.

However, high-quality evidence is limited.

European Klinefelter guidance notes that such strategies may be considered on an individualized basis but cannot currently be recommended routinely because controlled evidence is insufficient.

Patients should therefore be cautious about clinics promising guaranteed sperm production through hormonal tablets or injections before micro-TESE.

Can Hormone Levels Predict Whether Micro-TESE Will Succeed?

Not reliably.

Researchers have studied:

FSH, LH, testosterone, testicular size and age

as possible predictors.

Unfortunately, no single laboratory number can reliably tell us beforehand whether sperm will be found.

A man with extremely high FSH may still occasionally have focal sperm production.

Another man with a relatively acceptable testosterone value may have unsuccessful retrieval.

European guidance emphasizes that no clinical or biochemical marker reliably predicts sperm-retrieval success in adult Klinefelter syndrome.

This uncertainty is important during counselling.

Does a High FSH Mean Micro-TESE Will Definitely Fail?

No.

High FSH indicates significant testicular dysfunction.

But it does not prove that every microscopic area of the testicle is devoid of sperm.

Micro-TESE is specifically designed to search for rare focal areas of spermatogenesis.

Therefore, FSH should help characterize the disease but should not be used alone to deny a potentially appropriate patient a reproductive-urology consultation.

Can Testicular Size Predict Sperm Retrieval?

Small testes are characteristic of Klinefelter syndrome.

Testicular volume may provide general information about gonadal function but cannot reliably determine whether rare sperm-producing foci remain.

Therefore:

small testes do not automatically equal zero chance of sperm retrieval.

What Happens if Micro-TESE Finds No Sperm?

This possibility must be discussed before surgery.

If no viable sperm are identified, further reproductive decisions depend upon the couple's wishes.

Options may include:

use of donor sperm, adoption or choosing not to pursue further fertility treatment.

The psychological impact of an unsuccessful retrieval can be substantial.

Good counselling should therefore occur **before**, not only after, the procedure.

Genetic Counselling Before Assisted Reproduction

Because Klinefelter syndrome is a chromosome disorder, genetic counselling should be included in fertility planning.

Patients understandably ask:

“If my sperm is used, will my child also have Klinefelter syndrome?”

Available evidence indicates that many sperm recovered from men with Klinefelter syndrome arise from germ-cell lines capable of normal chromosome segregation, and most reported children born following TESE/ICSI have had normal karyotypes.

However, reproductive genetics is complex.

The couple should discuss:

parental karyotypes, potential chromosomal risks, prenatal testing and, where clinically appropriate, preimplantation genetic testing.

Genetic counselling helps the couple understand choices without overstating or minimizing the risk.

How Is Klinefelter Syndrome Diagnosed?

The definitive diagnosis is made by **chromosome analysis**, generally a **karyotype**.

A blood sample is examined to determine the number and structure of chromosomes.

A typical result shows:

47,XXY.

Mosaic Klinefelter syndrome may show:

46,XY/47,XXY.

NIH guidance identifies karyotyping as the diagnostic test used to demonstrate the additional X chromosome.

When Should Klinefelter Syndrome Be Suspected in an Infertile Man?

I particularly consider chromosome testing when an infertile man has:

azoospermia or severe oligospermia together with small testes, high FSH or other evidence of severely impaired sperm production.

The **2024 AUA/ASRM male-infertility guideline** recommends karyotype testing in men with primary infertility and azoospermia or sperm concentration below **5 million/mL** when this is accompanied by elevated FSH, testicular atrophy or other evidence of impaired spermatogenesis.

Klinefelter syndrome is the most common numerical chromosomal abnormality identified in this clinical setting.

Semen Analysis

The semen report in non-mosaic Klinefelter syndrome most commonly shows:

azoospermia.

Some men may show:

cryptozoospermia or severe oligospermia.

When even a few sperm are identified, cryopreservation may be valuable because sperm may not always be present in future ejaculates.

Hormonal Evaluation

Evaluation commonly includes:

FSH, LH and total testosterone.

Additional endocrine tests can be selected according to symptoms and general health.

Typical adult results often show:

high FSH, high LH and low or lower-normal testosterone.

However, laboratory findings vary.

Treatment decisions should be based on the patient's complete endocrine and clinical picture.

Sexual Function in Klinefelter Syndrome

Infertility does not automatically mean sexual dysfunction.

Many men with Klinefelter syndrome have normal:

sexual attraction, erection, ejaculation and orgasm.

Some, however, experience reduced libido or erectile difficulty because of testosterone deficiency, metabolic disease or psychological factors.

Therefore, the semen problem and sexual function should be assessed separately.

A man may have azoospermia and excellent erections.

Another may have both infertility and hypogonadal sexual symptoms.

The treatment plan should distinguish between these problems.

Klinefelter Syndrome Does Not Define Masculinity

This is something I explain very directly to patients.

A chromosome result does not measure masculinity.

Neither does a sperm count.

Being 47,XXY does not make a person “less male.”

A man may be:

physically masculine, sexually active, professionally successful, married and emotionally fulfilled

while also having a chromosomal cause of infertility.

The reproductive problem should be managed medically without attaching shame to it.

Klinefelter Syndrome Is More Than an Infertility Disorder

Although this article focuses on male infertility, modern medicine now understands Klinefelter syndrome as a **multisystem condition**.

A 2025 review highlighted increased risks involving:

bone health, diabetes, cardiovascular and metabolic disease.

More recent 2026 literature emphasizes the importance of structured long-term surveillance for metabolic disturbance, thrombosis, skeletal health, endocrine disease and selected malignancies.

This is important because an infertility clinic may be the first place the condition is diagnosed.

Finding Klinefelter syndrome should therefore lead to broader health assessment rather than stopping once fertility treatment has been discussed.

Bone Health

Testosterone contributes to bone strength.

Men with Klinefelter syndrome can have reduced bone-mineral density and increased risk of osteoporosis and fractures.

The problem may not be explained by testosterone alone; other aspects of Klinefelter biology may also influence bone metabolism.

Current expert guidance recommends attention to:

vitamin D, calcium status and bone health, with bone-density assessment according to clinical risk.

Metabolic Health

Klinefelter syndrome is associated with increased rates of:

central obesity, insulin resistance, metabolic syndrome and type 2 diabetes.

These conditions also affect sexual and cardiovascular health.

A patient diagnosed during infertility evaluation should therefore have appropriate general medical assessment rather than receiving fertility treatment in isolation.

Cardiovascular and Thromboembolic Health

There is also an increased tendency toward venous thromboembolic disease in Klinefelter syndrome.

More recent reviews continue to recognize cardiovascular and thrombotic complications as part of the multisystem phenotype.

This does not mean every patient needs anticoagulant medication.

It means cardiovascular risk factors and unusual thrombotic symptoms deserve appropriate clinical attention.

Gynecomastia and Male Breast Cancer Risk

Gynecomastia is relatively common.

Male breast cancer remains rare overall, but the relative risk is substantially increased compared with other men.

A recent systematic review estimated a markedly higher incidence among men with Klinefelter syndrome.

Patients should therefore report a persistent:

breast lump, nipple change, nipple discharge or unusual unilateral breast change.

Routine panic or excessive screening is not appropriate; awareness and individualized assessment are.

Testicular Cancer

Klinefelter syndrome does not characteristically produce the same testicular germ-cell cancer risk pattern associated with cryptorchidism.

However, there is a known association with certain **extragonadal germ-cell tumours**, particularly mediastinal germ-cell tumours in younger patients.

Modern reviews also recognize a distinct malignancy profile in Klinefelter syndrome.

Again, this is part of broader long-term care rather than a reason for unnecessary anxiety.

Thyroid and Autoimmune Conditions

Klinefelter syndrome has also been associated with increased frequency of selected autoimmune and thyroid disorders.

Recent multisystem reviews continue to identify immune dysregulation and thyroid dysfunction among conditions requiring clinical awareness.

Laboratory testing should be guided by symptoms and general health rather than performed indiscriminately.

Psychological and Neurocognitive Health

Some boys and men experience:

language difficulties, learning problems, social challenges, anxiety or reduced self-confidence.

Others experience none of these.

Modern Klinefelter care emphasizes individualized assessment rather than assuming a particular personality or intelligence level from the chromosome result.

A genetic diagnosis should never be used to make unsupported assumptions about an individual's cognitive ability.

The Emotional Impact of Infertility

Infertility itself can become one of the most difficult parts of the diagnosis.

A man may think:

“Will my wife leave me?”

“Am I not a complete man?”

“Can I ever have my own child?”

The psychological consequences deserve attention.

Infertility counselling should not consist only of explaining micro-TESE percentages.

Patients need realistic hope, honest uncertainty and respectful discussion of all reproductive possibilities.

Primary Male Infertility and Klinefelter Syndrome

Klinefelter syndrome is particularly relevant in **primary infertility**—where the man has never previously contributed to a pregnancy.

Because classic 47,XXY usually causes profound impairment of sperm production from early adulthood, natural paternity is uncommon.

The presence of:

primary infertility, azoospermia, very small firm testes and elevated FSH

should therefore strongly prompt consideration of karyotype testing.

Can Klinefelter Syndrome Cause Secondary Infertility?

Mosaic patients and rare men with residual ejaculated sperm may occasionally have previous reproductive potential.

However, classic non-mosaic Klinefelter syndrome generally produces severe infertility from early reproductive life.

If a man with confirmed previous natural biological paternity is later diagnosed with a mosaic form, the reproductive history should be interpreted together with the karyotype and current semen findings.

Natural Conception

Natural conception with classic non-mosaic 47,XXY is uncommon because ejaculated sperm are usually absent.

It is more plausible in mosaic cases or rare non-mosaic patients with residual spermatogenesis.

If sperm are present in semen, fertility counselling should consider cryopreservation because future semen samples may not contain the same sperm numbers.

IVF and ICSI

When sperm are extremely rare or surgically retrieved, conventional natural conception is unlikely.

ICSI is generally the key assisted-reproductive method.

A single viable sperm may be injected into an egg.

The female partner still undergoes an IVF cycle, meaning ovarian stimulation, egg retrieval and embryo culture.

Success therefore depends upon both:

sperm availability and female reproductive factors.

Age and egg quality remain important even when sperm retrieval is successful.

Micro-TESE Success Does Not Equal Baby Rate

This distinction is critical.

If micro-TESE retrieves sperm, that is the first successful step.

The sperm must then:

survive laboratory handling, fertilize an egg through ICSI, produce a viable embryo, implant and result in an ongoing pregnancy.

A 2025 systematic review found a median sperm-retrieval rate of approximately 44% across studies, but the overall median reported live-birth rate was much lower because reproductive attrition occurs at every subsequent stage.

Patients therefore deserve realistic counselling about both sperm retrieval and eventual live birth.

Cryopreservation

When ejaculated or surgically retrieved sperm are available, freezing can allow future use.

Cryopreservation is particularly valuable because micro-TESE is an invasive procedure and sperm availability in Klinefelter syndrome is unpredictable.

The reproductive team should discuss whether sperm will be used fresh, frozen or coordinated with the female partner's IVF cycle.

Should Every Young Man With Klinefelter Syndrome Undergo Micro-TESE?

Current evidence says **no**.

Fertility counselling is important.

Semen testing after appropriate maturity is reasonable.

But routine invasive surgery solely because a teenager has Klinefelter syndrome is controversial.

Most importantly, the large 2025 meta-analysis did **not** demonstrate a meaningful sperm-retrieval advantage for adolescent surgery over surgery during reproductive adulthood.

Therefore, invasive fertility preservation should be individualized rather than automatically performed at the youngest possible age.

What About Testicular Tissue Freezing in Children?

This remains experimental.

European guidelines recommend against routine prepubertal testicular tissue or spermatogonial stem-cell cryopreservation in Klinefelter syndrome outside appropriate research contexts because there is currently no validated clinical method to use such immature cells to produce a pregnancy.

Parents should be especially cautious about commercial claims that guarantee future fertility from experimental tissue banking.

Genetic Testing of Embryos

Some couples considering ICSI may discuss **preimplantation genetic testing** with a reproductive-genetics team.

The decision depends on:

the reproductive history, parental karyotypes, local practice, patient preferences and genetic counselling.

It should not be presented as mandatory for every couple with Klinefelter syndrome, nor should it be offered without explaining limitations.

Prenatal Diagnosis of Klinefelter Syndrome

Some cases are detected before birth through prenatal chromosome testing.

When this occurs, parents should receive accurate genetic counselling.

The clinical spectrum is extremely variable.

Many affected boys grow into functional adults with relatively subtle physical features.

Counselling should therefore avoid unnecessarily catastrophic predictions based solely on the chromosome result.

Can Klinefelter Syndrome Be Cured?

The chromosomal pattern itself cannot currently be changed.

There is no medicine—modern, herbal, Unani or otherwise—that can remove the extra X chromosome from the body's cells.

However, many consequences can be managed.

We may treat:

testosterone deficiency, bone-health problems, metabolic risks, gynecomastia and psychological difficulties.

And male infertility can sometimes be addressed through:

ejaculated sperm cryopreservation or micro-TESE followed by ICSI.

Therefore:

genetic cure and clinical management are two different concepts.

The Unani Perspective on Klinefelter Syndrome and Male Infertility

As a physician trained in Unani medicine and working particularly in sexual disorders and infertility, I approach male reproductive problems within the patient's complete health.

Classical Unani medicine discusses reduced semen and reproductive capacity through concepts such as:

Qillat-e-Huwaniya and **Qillat-i Mani**,

along with consideration of:

Mizaj or constitutional temperament, nutrition, digestion, physical activity, sleep, psychological wellbeing, sexual function and general strength.

This whole-patient approach can provide useful supportive care in selected infertile men.

But Klinefelter syndrome requires an especially important scientific distinction.

Klinefelter syndrome is not simply "weak sperm." It is a chromosome disorder.

Therefore, traditional semen-enhancing treatment cannot be expected to reverse the underlying 47,XXY chromosome pattern or regenerate severely damaged seminiferous tissue.

What Does Unani Research Show for Male Infertility?

Unani medicine has been clinically studied in some forms of male infertility, particularly **idiopathic oligospermia**.

A CCRUM-associated retrospective analysis from A.K. Tibbiya College reviewed earlier clinical studies involving **126 men with idiopathic oligospermia** and reported improvement in selected semen parameters with several Unani formulations.

A separate observational study conducted at the National Institute of Unani Medicine evaluated a traditional compound formulation in men with oligospermia and included semen and hormonal assessment.

CCRUM's standard Unani treatment literature also describes traditional approaches to **Qillat-i Mani** with attention to nutrition, digestive health and individualized constitutional factors.

These findings support continued scientific investigation of Unani approaches in selected male-fertility disorders.

But they must not be misinterpreted.

Why These Unani Studies Do Not Prove Treatment of Klinefelter Infertility

The men in these studies generally had **idiopathic oligospermia**.

Klinefelter syndrome is fundamentally different.

A man with idiopathic low sperm count may have normal chromosomes and potentially reversible physiological factors.

A 47,XXY man commonly has:

progressive germ-cell loss, seminiferous-tubule fibrosis and hypergonadotropic testicular failure.

Therefore, evidence from ordinary oligospermia cannot scientifically be transferred directly to Klinefelter syndrome.

At present, there is **no high-quality evidence that an Unani formulation can reverse azoospermia caused by Klinefelter syndrome or replace micro-TESE/ICSI.**

I believe this should be stated clearly to every patient.

How Unani Medicine May Be Useful Responsibly

The appropriate role is supportive and individualized.

Depending on the patient, Unani principles may contribute to management of:

general wellbeing, nutrition, digestive health, sleep, healthy body composition, associated sexual-health complaints and other modifiable health factors.

This can be particularly useful because men with Klinefelter syndrome may also struggle with:

metabolic problems, obesity, reduced physical vitality, sexual confidence or emotional stress related to infertility.

But a Unani treatment plan should complement, not replace:

genetic counselling, endocrine evaluation, testosterone management, semen testing, micro-TESE or IVF/ICSI.

No Herbal Medicine Can Correct the Chromosome Pattern

This deserves direct emphasis.

A patient with a 47,XXY karyotype should be protected from claims such as:

“Take this medicine and your chromosome problem will disappear.”

No such treatment currently exists.

Likewise, no supplement can guarantee that an azoospermic 47,XXY patient will begin ejaculating sperm.

Responsible integrative medicine should never create false hope where modern genetics gives us a clear biological explanation.

Can Unani Treatment Be Taken Before Micro-TESE?

Any treatment before sperm-retrieval surgery should be discussed with the reproductive urologist and fertility team.

The primary question is whether the proposed medicine has:

a rational clinical indication, known safety, interaction risk and any credible evidence relevant to the patient's condition.

Simply using multiple fertility supplements for several months does not guarantee greater micro-TESE success.

Likewise, unnecessary delay can become particularly important if the female partner's reproductive age is advancing.

The couple—not only the male laboratory values—must guide timing.

My Approach at Saira Health Care

When I see a patient with suspected or confirmed Klinefelter syndrome, I do not start by asking:

“Which sperm medicine should we give?”

I begin with:

Is the chromosome diagnosis confirmed?

Then:

Is this classic 47,XXY or mosaic disease?

Are there any sperm in the ejaculate?

What are FSH, LH and testosterone levels?

Is the patient clinically hypogonadal?

Does he want children now or in the future?

Has testosterone treatment already been started?

What is the reproductive status of his wife or female partner?

Would micro-TESE and ICSI be appropriate?

Those questions define a rational plan.

Special Treatment Planning by Dr. Nizamuddin Qasmi

My clinical focus in **Sexual Disorders & Infertility** allows me to consider both fertility and the broader sexual-health consequences of Klinefelter syndrome.

Depending on the patient's presentation, assessment may include:

semen analysis, hormone profile, review of karyotype, evaluation of testicular volume, assessment for testosterone-deficiency symptoms, sexual-function evaluation, fertility counselling and review of the female partner's reproductive situation.

Where sperm are present in semen, appropriate fertility preservation can be discussed.

Where azoospermia is confirmed, referral to an experienced reproductive urology and IVF team for **micro-TESE/ICSI evaluation** may be appropriate.

Where hypogonadism is present, endocrine treatment should be coordinated with fertility planning.

Where broader health concerns exist, long-term metabolic, bone and general-health follow-up should be encouraged.

Individualized Unani supportive management may then be incorporated where appropriate.

What I Mean by "Special Treatment"

For me, special treatment does **not** mean one proprietary medicine given to every Klinefelter patient.

It means understanding exactly where the patient is in his reproductive journey.

For example:

A mosaic patient with rare ejaculated sperm may primarily need semen cryopreservation.

A non-mosaic azoospermic man wanting children may need micro-TESE counselling.

A young man not planning children immediately may need fertility discussion and endocrine surveillance.

A hypogonadal adult who has completed fertility planning may need testosterone replacement.

A man whose primary concern is sexual desire may need hormonal and sexual-health assessment.

These are very different clinical situations.

The Female Partner Matters

Even when there is a major male genetic cause of infertility, fertility treatment must still be planned for the couple.

Suppose a 30-year-old man with Klinefelter syndrome is considering micro-TESE.

If his partner is 27 with good ovarian reserve, one treatment strategy may be appropriate.

If his partner is 41 with markedly reduced ovarian reserve, treatment timing may need to be much faster.

Finding sperm is only half of the fertility equation.

Egg number, egg quality, female age and uterine health remain important.

Saira Health Care's Contribution to Sexual Disorders and Infertility

At **Saira Health Care**, our approach to male infertility is designed to move beyond broad labels such as:

“weakness,”
“zero sperm,”
or
“low testosterone.”

Klinefelter syndrome demonstrates why precise diagnosis matters.

Without karyotyping, a patient may spend years taking sperm supplements for azoospermia caused by a chromosomal condition.

Once the diagnosis is established, the pathway becomes much clearer.

We can discuss:

realistic fertility potential, hormonal health, modern reproductive options, sexual wellbeing and long-term general health.

Where indicated, patients can be guided toward:

reproductive urology, genetics, endocrinology and IVF/ICSI services.

Unani medicine can be integrated supportively but should never prevent the patient from accessing these services.

What Patients Should Avoid

Patients with Klinefelter syndrome should be particularly cautious about several mistakes:

Do not spend years taking unproven “zero sperm” medicines without genetic and fertility evaluation.

Do not begin testosterone independently when biological paternity is still desired.

Do not assume high FSH can simply be normalized with supplements.

Do not assume azoospermia proves that absolutely no sperm exist anywhere in the testes.

Do not undergo invasive fertility procedures without appropriate reproductive-urology expertise.

And do not believe anyone who guarantees sperm retrieval or pregnancy.

Frequently Asked Questions

What is Klinefelter syndrome?

Klinefelter syndrome is a chromosomal condition in which a male usually has an additional X chromosome, producing the chromosome pattern **47,XXY**.

Is Klinefelter syndrome common?

It affects approximately **1 in 650 male newborns**, making it one of the most common sex-chromosome conditions in males.

Is it inherited from the parents?

Usually not in the traditional inherited sense.

It most commonly results from a random chromosome-separation error during formation of the reproductive cells.

Does Klinefelter syndrome always cause infertility?

Severe impairment of spermatogenesis is extremely common, particularly in non-mosaic 47,XXY patients.

Most have azoospermia, but some—especially mosaic patients—may have sperm in the ejaculate.

What semen result is most common?

Azoospermia is the characteristic finding in classic Klinefelter syndrome.

Can sperm still be present inside the testes?

Yes.

Small localized areas of sperm production may remain even when semen repeatedly shows azoospermia.

This is why micro-TESE can sometimes retrieve sperm.

What percentage of Klinefelter patients have successful sperm retrieval?

Published figures vary.

AUA/ASRM guidance discusses sperm retrieval in roughly **50–60% of selected 47,XXY men**, whereas a larger 2025 meta-analysis found a median rate of approximately **44%** across studies.

Neither figure predicts an individual's exact chance.

What is micro-TESE?

Micro-TESE is microdissection testicular sperm extraction.

A reproductive surgeon uses an operating microscope to search for small areas of testicular tissue where residual sperm production may remain.

What happens if sperm are found?

The sperm are generally used with **IVF/ICSI**, in which an embryologist injects one selected sperm directly into an egg.

Does sperm retrieval guarantee pregnancy?

No.

Finding sperm is only the first step.

Fertilization, embryo development, implantation and successful pregnancy must still occur.

Can Klinefelter patients have children naturally?

Natural biological paternity is uncommon in classic non-mosaic 47,XXY because ejaculated sperm are rarely present.

It may occasionally occur in mosaic or rare non-mosaic patients with residual spermatogenesis.

Should sperm be frozen if found in the semen?

Cryopreservation should be discussed because sperm may be present only in very small numbers and future samples may differ.

European guidance recommends semen analysis and sperm preservation in adult men with Klinefelter syndrome who desire paternity.

Should micro-TESE be done during teenage years?

Not routinely.

The latest 2025 meta-analysis found similar sperm-retrieval rates in adolescent and adult cohorts, suggesting that routine invasive retrieval does not need to be performed simply because a patient is a teenager.

Fertility counselling during adolescence remains important.

Can testicular tissue be frozen in a young child?

Routine prepubertal testicular tissue preservation is not currently recommended as established clinical treatment in Klinefelter syndrome.

Does testosterone treatment cure infertility?

No.

Testosterone treats testosterone deficiency.

External testosterone can suppress remaining sperm production and is therefore not a fertility treatment.

Should testosterone never be used?

No.

Testosterone can be important when clinically significant hypogonadism is present.

Fertility issues should ideally be addressed first when possible, particularly if sperm retrieval is being planned.

Does high FSH mean micro-TESE cannot work?

No.

High FSH indicates major testicular dysfunction but cannot reliably determine whether isolated sperm-producing areas remain.

Are small testes always present?

They are very common in adults with classic Klinefelter syndrome, but the overall physical presentation varies.

Does Klinefelter syndrome cause erectile dysfunction?

Not necessarily.

Many men have normal erections.

Erectile or libido problems may occur when testosterone deficiency, metabolic disease or psychological factors are present.

Does Klinefelter syndrome cause low sexual desire?

Some men with androgen deficiency experience reduced libido.

Others have completely normal desire.

Hormones and symptoms should be evaluated individually.

Does Klinefelter syndrome mean a man is less masculine?

No.

Chromosome composition and fertility status do not define masculinity or personal worth.

Will children conceived with Klinefelter sperm automatically have Klinefelter syndrome?

No.

Most reported children conceived using sperm retrieved from men with Klinefelter syndrome have had normal chromosome complements, but genetic counselling remains appropriate before assisted reproduction.

Is genetic counselling necessary?

It is strongly advisable when biological parenthood using ART is being considered because Klinefelter syndrome is a chromosomal condition.

Can Klinefelter syndrome affect bones?

Yes.

Reduced bone mineral density and osteoporosis risk are increased, so bone health deserves attention.

Is diabetes more common?

Metabolic syndrome, insulin resistance and type 2 diabetes occur more frequently than in the general male population.

Is breast cancer risk increased?

Yes.

The relative risk of male breast cancer is significantly greater than in men without Klinefelter syndrome, although breast cancer is still not inevitable.

Can Klinefelter syndrome be cured?

The extra X chromosome cannot currently be removed or corrected throughout the body.

However, many health consequences can be managed, and fertility treatment may permit biological fatherhood in selected patients.

Can Unani medicine cure Klinefelter syndrome?

No evidence supports a claim that Unani or any other medicine can eliminate the extra X chromosome.

Unani treatment may be used responsibly as supportive care for selected general, sexual or reproductive-health concerns, but it should not replace genetic, endocrine or reproductive treatment.

Can Unani medicine increase sperm in azoospermic Klinefelter syndrome?

There is currently insufficient direct evidence to claim that a Unani formulation can restore ejaculated sperm in men whose azoospermia is caused by Klinefelter syndrome.

Published Unani research reporting semen improvement has primarily involved other forms of male infertility such as idiopathic oligospermia, which is biologically different.

A Message From Dr. Nizamuddin Qasmi

When a patient comes to me with:

FSH very high, very small testes and zero sperm,

I do not want him to spend the next several years simply changing one fertility medicine after another.

I want to know:

Why is the sperm zero?

If chromosome testing shows **47,XXY**, we finally have an important explanation.

That diagnosis does not mean treatment ends.

It means treatment becomes more intelligent.

Instead of blindly trying to increase sperm count, we can discuss:

whether sperm are present in the semen, whether fertility preservation is possible, whether micro-TESE is appropriate, whether testosterone deficiency needs treatment, whether genetic counselling is required and what reproductive options are realistic for the couple.

That is much more useful than repeatedly treating an unexplained label of “male weakness.”

My Message About Hope

I also tell my patients:

Do not convert azoospermia into hopelessness.

Twenty or thirty years ago, a non-mosaic Klinefelter diagnosis usually meant that biological fatherhood was considered virtually impossible.

Modern reproductive medicine has changed that.

Micro-TESE can sometimes find rare sperm-producing areas.

ICSI can use very small numbers of sperm.

Several men with Klinefelter syndrome have therefore become biological fathers.

But hope must remain honest.

Micro-TESE can fail.

ICSI can fail.

Pregnancy is never guaranteed.

Responsible fertility medicine lies between these two extremes:

neither hopelessness nor false promises.

Latest Medical Understanding

Our understanding of Klinefelter fertility continues to develop.

The **2024 AUA/ASRM male-infertility guideline** identifies Klinefelter syndrome as the most common numerical chromosomal cause encountered in severe impaired spermatogenesis and recognizes meaningful micro-TESE sperm-retrieval potential in selected 47,XXY patients.

The **2025 review of Klinefelter syndrome** emphasizes that the condition requires multidisciplinary care extending beyond infertility to endocrine, skeletal, metabolic and cardiovascular health.

Most importantly for fertility preservation, the large **2025 meta-analysis of 2,815 patients** found no meaningful difference between adolescent and adult sperm-retrieval rates—approximately 45% versus 42%—providing reassurance that invasive surgical sperm retrieval usually does not need to be rushed simply because puberty has begun.

Recent 2026 evidence also continues to reinforce that Klinefelter syndrome is a multisystem condition that benefits from structured lifelong health surveillance rather than being regarded solely as a cause of azoospermia.

Conclusion

Klinefelter Syndrome is one of the most important chromosomal causes of male infertility.

The typical chromosome pattern is:

47,XXY.

The additional X chromosome affects testicular development and commonly leads to:

small testes, severely impaired sperm production, elevated FSH and LH, variable testosterone deficiency and azoospermia.

The condition may remain undiagnosed until adulthood because physical features can be subtle.

In an infertile man with:

azoospermia or severe oligospermia, small testes and elevated FSH,

karyotype testing becomes particularly important. The 2024 AUA/ASRM guideline specifically recommends karyotype evaluation in appropriate men with azoospermia or sperm concentration below 5 million/mL when the clinical pattern indicates impaired sperm production.

Natural conception is uncommon in classic non-mosaic Klinefelter syndrome.

However, azoospermia does not necessarily mean that every area of the testes has completely stopped producing sperm.

Modern **micro-TESE** can sometimes identify focal sperm production, and sperm can potentially be used through **IVF with ICSI**.

Current evidence suggests sperm retrieval may be possible in roughly **40–60% of selected patients**, although individual success cannot be predicted or guaranteed.

The newest evidence also indicates that routine invasive sperm retrieval during adolescence is generally unnecessary solely to preserve fertility, because retrieval rates appear similar during adolescence and reproductive adulthood.

Testosterone deficiency should be treated appropriately, but fertility goals should ideally be addressed before initiating testosterone therapy when sperm retrieval is being considered.

Klinefelter syndrome also deserves broader health attention because patients have increased risks involving:

bone health, metabolic disease, diabetes, cardiovascular and thrombotic disease, and selected malignancies.

From the **Unani perspective**, male infertility can be approached holistically through attention to nutrition, digestion, Mizaj, lifestyle, physical wellbeing and associated sexual concerns.

Published Unani research has reported improvements in some semen parameters among men with **idiopathic oligospermia**, but these findings should not be incorrectly applied to Klinefelter syndrome.

There is currently **no evidence that Unani medicine can remove the additional X chromosome or reliably reverse the severe testicular damage responsible for azoospermia in 47,XXY Klinefelter syndrome.**

At **Saira Health Care**, my approach is therefore integrative but medically precise:

confirm the chromosome diagnosis, assess semen and hormonal function, understand the patient's fertility goals, provide genetic counselling, consider sperm cryopreservation or micro-TESE/ICSI where appropriate, treat testosterone deficiency responsibly, support general reproductive and metabolic health, and incorporate individualized Unani supportive care only where it complements rather than replaces established fertility treatment.

The most important message I give patients is:

Klinefelter syndrome explains why fertility may be difficult—but modern diagnosis and reproductive medicine mean that the conversation should not end with the word "azoospermia."

For selected men, biological fatherhood may still be possible.

About the Author

Dr. Nizamuddin Qasmi

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

Qualifications & Professional Training

BUMS — Hamdard University, Delhi
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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 clinical work at **Saira Health Care** focuses on sexual disorders, male reproductive health and infertility.

His approach to severe male-factor infertility emphasizes identifying underlying genetic, hormonal, anatomical and sexual-health causes rather than treating azoospermia or low sperm count as generalized sexual weakness.

In patients with confirmed or suspected Klinefelter syndrome, appropriate contemporary evaluation—including semen testing, hormone assessment, karyotype review, genetic counselling and referral for reproductive-urology or IVF/ICSI care—is integrated with individualized supportive Unani principles where clinically appropriate.

Medical Disclaimer

This article is intended for health education and general public awareness. It does not establish an individual diagnosis and does not replace consultation with an appropriately qualified reproductive urologist, endocrinologist, genetic specialist, fertility specialist or other healthcare professional.

Klinefelter syndrome is a chromosomal condition. No herbal, Unani, nutritional or conventional medicine can currently remove the additional X chromosome from the body's cells.

Men with azoospermia, markedly elevated FSH, very small testes or suspected genetic infertility should receive appropriate genetic and reproductive evaluation.

Men who want current or future biological fertility should discuss fertility preservation before starting or changing testosterone treatment whenever clinically possible.

Micro-TESE does not guarantee sperm retrieval, and finding sperm does not guarantee fertilization, pregnancy or live birth.

Any Unani or complementary treatment should be coordinated with the patient's endocrine and fertility care and should never delay appropriate genetic counselling, testosterone-deficiency management, micro-TESE assessment or assisted reproduction.