Trends Of Male Infertility: Etiopathogenesis & Diagnostics

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ABSTRACT
Infertility is an ominous problem worldwide. On average, about 10% of all couples face difficulty in starting a family and this creates a feeling of great personal failure, particularly in India where religious and socio-economic traditions have made it almost imperative for everyone to have children. A significant association had been found between impaired semen quality including sperm count, motility and morphology. In this review, the various contributory etiological factors i.e., pre-testicular, post-testicular or directly at the testicular level along with the factors that could influence each of the partners. Their age, medications, surgical history, systemic diseases, endocrine factors, exposure to environmental toxins, pesticides, industrial chemicals, genetic problems, Diet, Stress, Alcohol and modern lifestyle have been discussed which have a serious impact on male infertility. The key purpose of this review article is to evaluate certain causative factors of infertility and is to identify contributions to the pathogenesis of male infertility and ultimately offer better strategies for making the diagnosis.

Keywords: Diagnostics, Genetics, Male Infertility, Testicular, Semen, Sperm

Introduction

Infertility is a disease of the male and female reproductive system defined by the failure to achieve a pregnancy after 12 months or more of regular unprotected sexual intercourse.[1](ICD-11, WHO-2018). An estimated 15% of couples meet this criterion and are considered infertile, with approximately 35% due to female factors alone, 30% due to male factors alone, 20% due to a combination of female and malefactors, and 15% unexplained. [2,3]Causes of infertility in men can be categorized as obstructive or nonobstructive. Infertile men may have deficiencies in sperm formation, concentration (eg, oligospermia {too few sperm}, azoospermia {no sperm in the ejaculate}), or transportation. Ancient Indian traditional medicinal system i.e. Ayurveda is a treasure of scientific knowledge about Infertility (Klaibya), emphasises the importance of stress-free, healthy, prosperous & dynamic life and Sukhayu essential for healthy progeny.[4]The trial has been registered in the clinical trial registry of India Reg.No.CTRI/2021/01/030434 ("Epidemiological, Etio-pathological study of Male Infertility with special reference to Molecular Study")

1. Types of Male Infertility

i.**Primary infertility**: Couples had never conceived at any stage before
ii.**Secondary infertility**: Couples have had a pregnancy although not necessarily a successful one.

2. Aetiology

Causes of male infertility**:** the primary cause of male infertility is demonstrated[5] in Figure 1

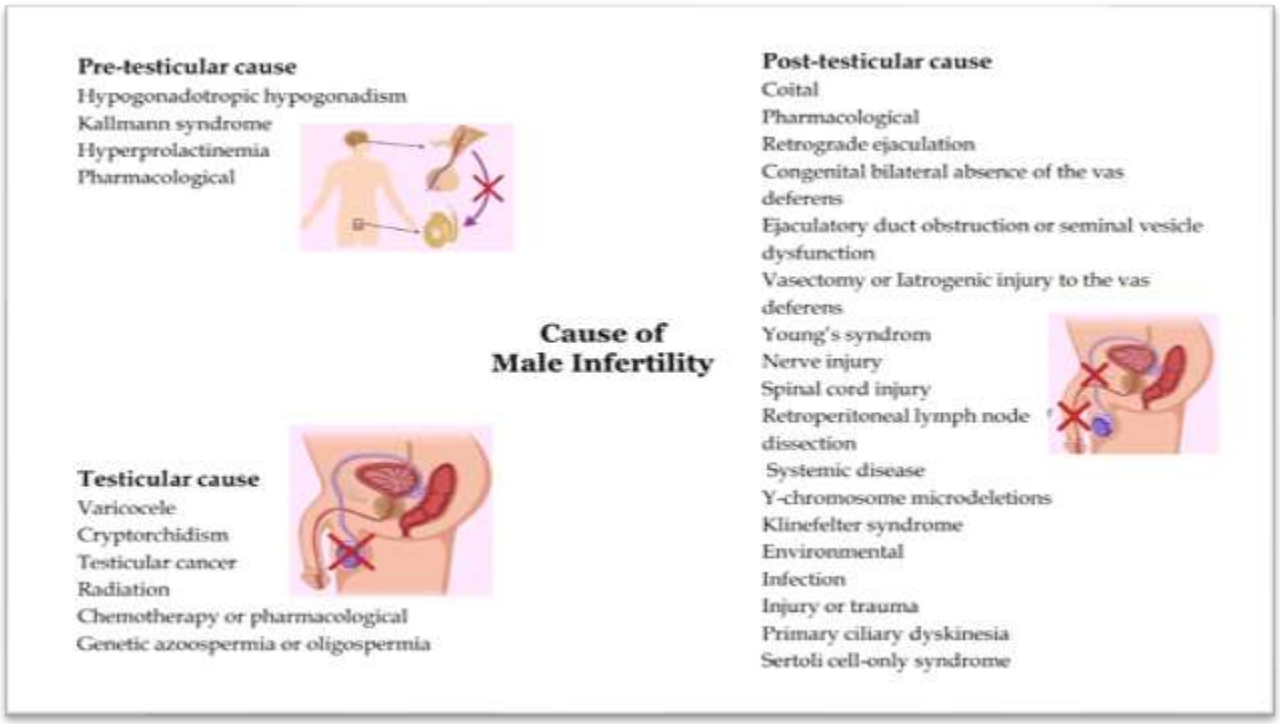

Figure1. Showing pre-testicular, testicular and post-testicular etiological causes of Male Infertility

2.1. Pre-testicular

2.1.1. Hypogonadotropic hypogonadism: Kallmann syndrome

Idiopathic congenital hypogonadotropic hypogonadism (CHH) is a rare reproductive disease that is mainly caused by a gonadotrophin-releasing hormone (GnRH) deficit but with considerable genetic heterogeneity. Clinically, this disorder is categorized by peculiarly low (LH), (FSH) in conjunction with low or untraceable concentrations of circulating sex steroids. In approximately 50% of cases, CHH patients also suffer from a reduced or deficient sense of smell (hyposmia or anosmia, respectively), which is then termed Kallmann syndrome (KS).[6,7]A variety of non-reproductive dysfunctions and developmental anomalies have been observed in association with hypogonadism. This review aimed to provide a brief overview of genetics, molecular pathogenesis, and diagnosis with a particular focus on recent progress in the field. Multiple sequential developmental and neuroendocrine signalling pathways control the evolution and homeostasis of the GnRH neurons as well as the metabolism of GnRH.[8]Inside the hypothalamus, secretory GnRH neurons extend their axonal processes to the medial eminence through which pulsatile GnRH is secreted into circulation via the hypophyseal portal system. GnRH binds to GnRH receptor 1 (GnRHR1) on gonadotroph cells in the anterior pituitary and stimulates the synthesis and secretion of LH and FSH. Subsequently, LH acts on the testes to stimulate testosterone production, and LH and FSH act on the ovaries to induce estrogen production which, in turn, leads to steroidogenesis and germ cell production [9]. GnRH is temporarily secreted at 3 to 6 months postnatally, which is more evident in boys than girls and is sometimes called "mini-puberty." GnRH secretion then remains dormant until the onset of puberty, when its reactivation initiates secondary sexual characters. Therefore, the normal development and properly coordinated actions of the hypothalamic-pituitary-gonadal (HPG) axis are essential for GnRH pulse generation and normal reproductive function.

The assessment of a 19-week-old human fetus that gets aborted with X-linked Kallmann syndrome gives away that the GnRH neurons were stopped in a tangle above the cribriform plate. Because the initial separation and relocation of the GnRH precursor cells look down to be normal, it was conjectured that the subsequent axonal lengthening pioneer, and/or terminal demarcating procedure might have been rearranging and prohibited the GnRH neurons from reaching the forebrain. It was also projected that the substandard targeting, innervation, and synaptogenesis of the olfactory sensory neurons to the OB anlage might have caused OB dysgenesis in KS patients. Following this study, Hypogonadotrophichypogonadism – Kallmann syndrome was defined as the partial or complete breakdown of sexual development secondary to the failed embryonic initiation of hypothalamic GnRH neurons, which originate in the deficit released of sex hormones from the anterior pituitary and gonads.[10]

2.1.2. Hyperprolactinemia

Hyperprolactinemia is responsible for infertility in around 11% of oligospermic males.[11] Hyperprolactinemia hampers the pulsatile release of the GnRH, which causes the decreased pulsatile release of FSH, LH, and testosterone makes the spermatogenic arrest, spoils the sperm motility, and changes sperm quality.It later generates secondary hypogonadism and infertilityHyperprolactinemia also unswervingly influences spermatogenesis and steroidogenesis which mainly act on prolactin receptors that are present in Sertoli cells and Leydig cells inside the testes, and build primary

hypogonadism and infertility.[12]It has been observed that oligospermiaorazoospermic patients who have normal serum levels of gonadotrophins have relatively elevated serum levels of prolactin, making the prolactin play in gametogenesis, which is autonomous of gonadotrophins.It can be managed conservatively with medication, such as bromocriptine and cabergoline, which restores serum prolactin levels, re-establishment of gonadal function, set aside infertility caused by hyperprolactinemia, and starts reduction in the prolactinoma size in the majority of patients.[13]

2.1.3. Pharmacological

Male infertility may be generally diagnosed through an abnormal semen analysis. The semen analysis has numerous measured parameters, one of the important is abnormalities in sperm count, extended from fewer than normal sperm (oligospermia) to untraceable sperm (azoospermia), sperm motility, a condition known as asthenospermia.[14.15]The causes of oligospermia/azoospermia are divided into three distinct stages: Pre-Testicular, Testicular, and Post-Testicular, which may depend on the stage of spermatogenesis which is changed or diminished. Pre-testicular stage - azoospermia results from the pituitary gland, part of the (HPG) axis, not secrete proper hormones to stimulate the testes to p spermatogenesis and is mainly due to endocrine abnormality.[16]

Testicular stage azoospermia is a failure of testicular function and Post-Testicular azoospermia is due to physical causes such as obstruction. In the majority of male infertility cases, a definitive cause for abnormalities is never identified.[17] Pharmaceutical medications, as well as recreational drugs, have been documented to impact semen production at particular stages.[18] In addition, some medications impair ejaculation and erectile function, as well as the ability to decrease libido.[19]

2.1.4. Calcium channel blockers

They block the progress of free calcium ions, which serve as essential secondary messengers among their channels and ionophores. CCBs can affect cardiac muscle, the smooth muscle of blood vessels, and neurons. CCBs are specified as a medication for various medical states of affairs including hypertension and heart failure.[20]

Since 1988, calcium antagonists have been shown to cause a dose-dependent decline in sperm motility and viability in vitro.[21]The dose-dependent consequence of Nifedipine was assessed concerning sperm morphology, motility, and other parameters. The addition of the CCB resulted in significant suppression of calcium ion uptake. There are structural changes in both the head and tail regions of sperm when assessed by scanning electron microscope. Others have shown that CCB may stop the capability of sperm to bind to an egg by changing the lipid bilayer of the sperm plasma membrane.[22]

Calcium ions exert a paradoxical effect on human sperm motility, depending on the developmental stage of the sperm.[23] Earlier in vitro, experiments in non-human mammalian species had shown an increase in sperm motility with the addition of calcium ions, likely through the binding of calcium to a calmodulin-like protein. However, other experiments demonstrated that calcium chelators (EGTA and EDTA), as well as calcium antagonists, increased motility in human semen. One possible hypothesis for this effect was the triggering of a premature acrosome reaction in sperms before they underwent full capacitation and maturation.[24]

2.1.5. Alpha-adrenergic blockers

Epilepsy is recognized to be related to male infertility. Epileptic males have significantly lower fertility rates and larger risks of hyposexuality than the given population.[25] Studies on anti-epileptic therapy including carbamazepine, phenytoin and valproate have recommended specific drug-dependent side effects, which may include abnormal sperm morphology, reduced motility, lower sperm count, and reduced testicular volume. The likely proposed mechanism for these side effects is been thought of by the interaction between anti-epileptic therapy and sex hormones which are generally steroid hormones, intrusive with normal HPG pathway functioning.[26]

2.1.6. The Antiretroviral drug,

In recent years the HIV medication has reduced the motility of HIV patients with a significant number. Many of such treated patients with an antiretroviral treated patient may develop male infertility.[27] People with HIV donate semen to achieve IVF treatments only through sperm washing and significant results is also present.

In past years HIV infection does not seem to have significantly impaired semen parameters.[28,29] Various research has been done in this field on if antiretroviral therapy has affected semen parameters. Highly active antiretroviral therapy (HAART) is distributed to patients with HIV and, despite its massive reduction in HIV mortality, has been coupled with several severe side effects including neuropathy and lipodystrophy.[30]

2.2. Testicular

2.2.1. Varicocele

The side effect of varicocele on spermatogenesis can be dependent on multiple factors such as anelevated testicular temperature, increased intratesticular pressure, hypoxia due to reduction of blood flow, reflux of toxic metabolites from the adrenal glands, and hormonal profile rearrangement.[31, 32]

Increased temperature of the scrotum which may be due to reflux of warm blood from the abdominal cavity is the main culprit of the varicocele. Insufficiency of the internal spermatic vein valves and malfunction of the valves of the external spermatic and cremasteric are the two main causes of varicocele veins.[33]

2.2.2. Cryptorchidism

Cryptorchidism / undescended testis commonest congenital abnormalities in the newborn population. The occurrence of cryptorchidism in full-term neonates varies between 2% and 4%, while in premature adolescence it is reported to be as high as 33%.[34]Impairment of germ cell maturation failure to maturate and following infertility in adulthood are the well-documented results of cryptorchidism. The prevalence of azoospermia in one-sided cryptorchidism is 14% and this number increases to 93% in untreated both-sided cryptorchidism, making cryptorchidism the most frequent etiologic issue of azoospermia in the adult.[35]Cryptorchidism is a common congenital anomaly. Testicular development needs the existence of the sex (testis) determining region on the Y chromosome (SRY gene) for normal demarcation. Testicular tissue originates from discernment of the gonadal ridge and an intact hypothalamic-pituitary-gonadal axis is a criterion for testicular descent.[36] Although the incidence of cryptorchidism in full-term boys ranges between 1% and 3%, the prevalence decreases to 0.8% and 1.5% at 1 year of age.[37] Several aspects are related to cryptorchidism, such as prematurity, low birth weight, and familial and maternal exposure to estrogen, during the first trimester.

About 80% of undescended testes are palpable and 20% are non-palpable.[38]It is important to differentiate true cryptorchidism from other conditions, such as retractile testis, absent or vanishing testis, and ectopic testis. Most palpable undescended testes are located along the inguinoscrotal region with most intra-abdominal testes found within a few centimetres of the internal ring. The main reasons for the treatment of undescended testes are increased fertility and decreased risk of testicular torsion or injury and testicular cancer, as well as psychological stigma.

2.2.3. Genetic azoospermia or oligospermia

It is estimated that infertility affects approximately 10% of the population. Although historically a significant percentage of male-factor infertility was diagnosed as idiopathic, recent studies have demonstrated a genetic aetiology. This chapter offers a brief review of the criteria used in the diagnosis of azoospermia/severe oligozoospermia, followed by a discussion of the role of Y chromosomal and autosomal genes in the aetiology of this condition.[39]

Recent advances in our understanding of the molecular mechanisms that regulate spermatogenesis may provide new insight into cases of idiopathic infertility. Although deletions in the Y chromosome are likely responsible for a significant percentage of azoospermia and severe oligospermia, a direct relationship between deletions in specific Y chromosome genes and the presenting phenotype has yet to be determined. In addition, much remains to be uncovered about the contribution of autosomal genes to this disease. The current technological ability to achieve pregnancy in azoospermic and severely oligospermic patients mandates an understanding of the defects involved so that appropriate information may be made available as to the likelihood of transmission of these defects to the offspring.[40]

2.3. Post-testicular

2.3.1. Coital

Even if some infertile couples have unsatisfactory sex lives,[41] to our knowledge, coital frequency has never been considered in men with infertility.[42] The duration of infertility may have promising consequences on coital frequency because couples willing to have a baby for a long time may anticipate and decrease the frequency of intercourse.[43] Semen volume is used as an indicator because, on several occasions, low semen volume is a chief complaint by men and it may be a signal of testosterone deficit (the major organs that are producing semen are testosterone dependent) or probably drop off their sexual drive by restraining self-confidence. Sperm production is sustained by testosterone and sperm count and so incorporated as a self-regulating variable.[44]

2.3.2. Retrograde ejaculations

Ejaculation is a vital step in normal breading and its malfunction leads to infertility. Many ejaculatory anomalies can have both psychological as well as systemic origins, however, retrograde ejaculation is exclusive in that it is almost organic in origin. Even though being a common type of ejaculatory dysfunction, it is responsible for only 1–2% of infertility[45]. The permutation of dry orgasm and concern with fertility make the situation stressful to both the patient and their partner particularly when trying to conceive.[46]

The procedure of ejaculation needs multifaceted synchronization and interaction between the epididymides, vasa deferentia, prostate, seminal vesicles, bladder neck and bulbourethral glands. In lead ejaculation, sperm are hastily relayed along the vas deferens and within the urethra via the ejaculatory ducts. Commencing there, the semen advances in an antegrade manner in part managed by compotation of the bladder neck and forceful contraction of the periurethral muscles synchronized by a centrally mediated reflex. Closure of the bladder neck and seminal emission is started via the sympathetic nervous system from the lumbar sympathetic ganglia and afterwards hypogastric nerve. Prostatic and seminal vesicle discharge, as well as retrenchment of the bulbocavernosus, ischiocavernosal and pelvic floor, are started by the S2-4 parasympathetic nervous system utilizing the pelvic nerve.[47]Any factor, that disrupts this reflex and inhibits the bladder neck (internal vesical sphincter) contraction, may lead to the retrograde passage of semen into the bladder.

2.3.3. Congenital bilateral absence of the vas deferens

Innate bilateral absence of the vas deferens develops in males when the vas deferens fail to develop properly. Although the testes typically develop and function normally, sperm cannot be transported through the vas deferens to become part of semen. As a result, a person having this anomaly is not capable of fathering children (infertile) unless they use assisted reproductive technologies. This situation has not been proclaimed to affect sex drive or sexual performance.[48] This situation can occur alone or as a sign of cystic fibrosis, an inherited disease of the mucus glands. Cystic fibrosis causes continuous damage to the respiratory system and chronic digestive system problems. Many men with congenital bilateral absence of the vas deferens do not have the other characteristic features of cystic fibrosis; however, some men with this condition may experience mild respiratory or digestive problems.[49]

2.3.4. Ejaculatory duct obstruction

It can be either congenital or acquired. Congenital elements contain congenital atresia or stenosis of the ejaculatory ducts and utricular, Mullerianductcysts. Acquired causes may be secondary to trauma, either iatrogenic or otherwise, or infectious or inflammatory aetiology.[50] Formation of calculus secondary to infection can also cause obstruction. Cyst formation from earlier instrumentation or infection may also take place· Often, patients having ejaculatory duct obstruction have no major predecessor history· Patients having congenital or noninfectious modes of ejaculatory duct obstruction are superior after treatment than those having with infection[51,52]

2.3.5. Young's syndrome

The young syndrome is a state characterized by male infertility, injured airways in the lungs (bronchiectasis), and inflammation of the sinuses (sinusitis). Male infertility in Young syndrome is secondary to obstructive azoospermia, a condition in which sperm are produced but do not mix with the rest of the ejaculatory fluid, due to a physical obstruction in the epididymis. This marks nonexistent levels of sperm in semen.[53] Young syndrome is typically diagnosed in middle-aged men who undergo evaluation for infertility. As the signs and symptoms of Young syndrome are similar to cystic fibrosis(CF), part of the diagnosis method may include ruling out CF. Even if the exact cause of Young syndrome has not been known, it is said that it to either be associated with childhood exposure to mercury or genetic factors. While there is no one treatment for Young syndrome, management involves the treatment of sinus and lung infections. Fertility treatment may also be an option, including surgery to remove the obstruction in the epididymis (vasoepididymostomy) or assisted reproduction, such as intracytoplasmic sperm injection(ICSI).[54]

2.3.6. Vasectomy or vas deferens injury

Vasectomy and Sertoli-cell-only syndrome caused by cytotoxic chemotherapy and radiation therapy for malignant tumours of the testes, leukaemia, lymphoma, and serious autoimmune diseases are the most widespread forms of medically induced infertility.[55] Although some treatment regimens only suppress spermatogenesis temporarily, recovery of fertility is unpredictable. Alkylating agents, such as cyclophosphamide and busulfan, destroy spermatogonia. Antimetabolites may be used to treat psoriasis, rheumatoid arthritis, or xenograft rejection and can have transient adverse effects on spermatogenesis.[56] Many drugs have potentially adverse effects on spermatogenesis or sexual performance, including androgens, anabolic agents, estrogens, glucocorticoids, cimetidine, spironolactone, antibacterials (especially nitrofurantoin), antihypertensive drugs, and psychotropic agents. However, in practice, these are not common causes of infertility.[57]

2.3.7. Spinal cord injury

Spinal cord injury (SCI) mainly occurs in young men at the coming of their reproductive health. The widely held that men with SCI cannot father children naturally. Most men with SCI have atypical semen quality characterized by normal sperm concentrations but abnormally low sperm motility and viability.[58] Accessory gland dysfunction has been projected as the cause of these anomalies. Leukocytospermiais manifest in most SCI patients. Additionally, elevated concentrations of pro-inflammatory cytokines and elevated concentrations of inflammasome components are found in their semen. Neutralization of these constituents has resulted in improved sperm motility.[59]

3. Others cause

Apart from testicular cause certain other causes also play an important role in the pathogenesis of infertility demonstrated in Figure 2.

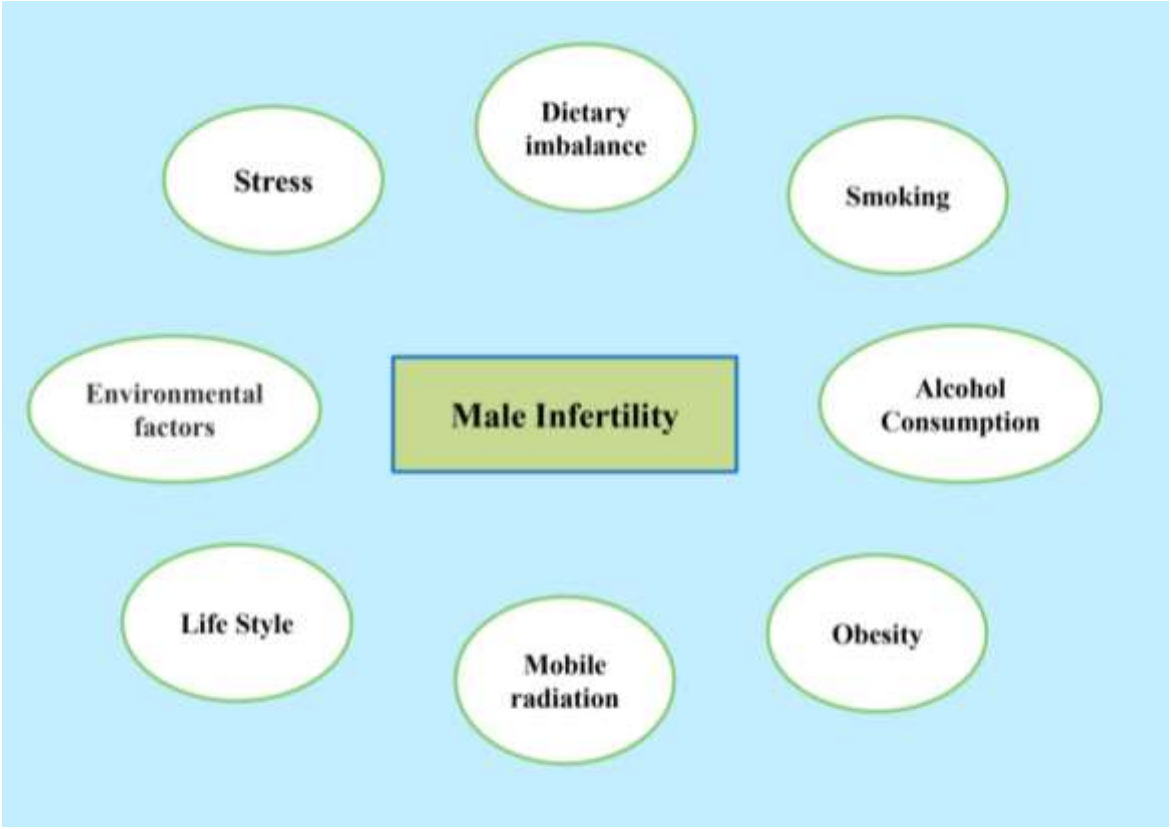

Figure 2. Showing others the etiological cause of Male Infertility

3.1. Smoking

Smoking has a significant effect on fertility, specifically sperm count and the normal morphology of sperm. This might be due to OS produced by smoking, which has devastating effects on semen parameters, thus reducing male fertility.[60] Smoking is among the modifiable risk factors of reproductive health and approximately 37% of men of reproductive age smoke cigarettes[61]. Smoking has also been seen to cause oxidative stress in spermatozoa, due to the formation of excessive reactive oxygen species (ROS) as a large amount of polyunsaturated fatty acids are present in the plasma membrane. In addition to this, a decreased concentration of scavenging enzymes in the cytoplasm of spermatozoa causes a low antioxidant capacity[62]. Hence OS is imminent due to this excessive ROS and limited antioxidant defence mechanisms. Spermatozoa also are most susceptible to oxidative stress and oxidative DNA damage (ODD) due to their limited capacity for detection and repair of DNA damage.[63] Oxidative stress causes defective sperm function by affecting its viability, motility, DNA fragmentation, and membrane lipid peroxidation

3.2.Effect of Mobile Radiation

Presently, there is a rise in the use of mobile phones, laptops, and wireless internet technologies such as Wi-Fi and 5G routers/modems across the globe; these devices emit a considerable amount of electromagnetic radiation (EMR) which could interact with the male reproductive system either by thermal or non-thermal mechanisms. It was shown in research studies that samples of human ejaculated semen exposed to EMR emitted by a cell phone showed a significant reduction in sperm motility and viability and an increase in reactive oxygen species (ROS) level; this was achieved by the collection of semen samples from 23 healthy controls and 9 patients at an infertility clinic.[64,65]

3.3.Stress

Increasing evidence suggests that oxidative stress (OS) plays an independent role in the aetiology of male infertility called Male Oxidative Stress Infertility (MOSI), with 30% to 80% of infertile men having elevated seminal reactive oxygen species levels. OS can negatively affect fertility *via* several pathways, including interference with capacitation and possible damage to sperm membranes and DNA, which may impair the sperm's potential to fertilize an egg and develop into a healthy embryo.[66] There is overwhelming evidence that oxidative stress (OS) plays a significant role in the aetiology of male infertility.[67-73] Seminal reactive oxygen species (ROS) are produced mainly by leukocytes or abnormal and immature spermatozoa and are a natural byproduct of oxidative metabolic pathways as well as cytosolic and plasma membrane oxidases. ROS are also a natural byproduct of adenosine triphosphate production within sperm cell mitochondria.[74] When ROS levels increase to a pathological level, the body uses dietary and endogenously produced antioxidants to bring the system back to homeostasis. An imbalance between these two opposing forces, in which ROS outweigh antioxidants, can result in OS, which can negatively affect fertility *via* several pathways. OS interferes with capacitation and may cause sperm membrane and DNA damage, thereby affecting the sperm's potential to fertilize an egg and generate a healthy embryo.[66]

3.4. Diet

Oxidative stress constitutes the key mechanism that associates improper diet and obesity with both lower semen quality and an increased risk of infertility. Moreover, it is currently considered one of the leading causes of male infertility. Recent research has reported that a high intake of animal proteins, saturated and trans-fatty acids, and simple carbohydrates, as well as a low supply of dietary fibre and essential unsaturated fatty acids, are affecting fertility. It is clear that with the spread of the Western diet model, the parameters evaluating semen quality have deteriorated. A diet rich in processed and according to some sources, red meat, fatty dairy, coffee, alcohol, sweet drinks and sweets, and potatoes, and simultaneously deficient in whole-grain products, vegetables and fruits, poultry, fish and seafood, nuts, and lean dairy is associated with poorer semen parameters and reduced fertility.[75]

3.5. Obesity

The increasing occurrence of obesity has become a significant public health concern. Individuals with obesity have a higher prevalence of reproductive disorders. Reproductive problems include infertility due to anovulation, in women, and lower testosterone and diminished sperm count, in men.[76]Obesity is linked to fertility dysfunction in both genders. Obesity in men may affect their fertility by impaired spermatogenesis, reduced testosterone levels, erectile dysfunction, and poor libido by putatively targeting the HPG and hypothalamic-pituitary-adrenal axes. Leptin plays a key role in many metabolic functions, including reproduction. High concentrations of leptin have been found in infertile men with disorders affecting the testicular parenchyma, including nonobstructive azoospermia, oligozoospermia, and oligo-asthenic-teratozoospermia.[77]

3.6. Consumption of alcohol:

In recent decades, the decline in human fertility has become increasingly more worrying: while therapeutic interventions might help, they are vexing for the couple and often burdened with high failure rates and costs. Alcohol consumption is often considered socially acceptable, but its negative effects on gonadal function have been consistently reported in the last 30 years. Several studies have reported a variety of negative effects on male fertility following drug abuse a worrying phenomenon, as illicit alcohol consumption is on the rise, most notably in younger subjects.[78]

4. Diagnosis

The initial step in the evaluation of an infertile male is to obtain a thorough medical and urologic history. Depending on the findings from the history, physical examination, semen analysis and sperm function tests are necessary, as shown in Table 1.

History should include consideration of the following: Duration of infertility, Previous fertility in the patient and the partner, Timing of puberty (early, normal, or delayed), Childhood urologic disorders or surgical procedures, Current or recent acute or chronic medical illnesses, Sexual history, Testicular cancer and its treatment, Social history (eg, smoking and alcohol use), Medications, Family history, Respiratory disease, Environmental or occupational exposure and Spinal cord injury.

The physical examination should include a thorough inspection of the following: Testicles (for bilateral presence, size, consistency, symmetry), Epididymis (for presence bilaterally, as well as any induration, cystic changes, enlargement, tenderness), Vas deferens (for presence bilaterally, defects, segmental dysplasia, induration, nodularity, swelling), Spermatic cord (for varicocele), Penis (for anatomic abnormalities, strictures, or plaques), Rectum (for abnormalities of the prostate or seminal vesicles) and Body habitus.

The semen analysis is the cornerstone of the male infertility workup and includes assessment of the following: Semen volume (normal, 1.5-5 mL), Semen quality, Sperm density (normal, >15 million sperm/mL), Total sperm motility (normal, >40% of sperm having normal movement), Sperm morphology (sample lower limit for a percentage of normal sperm is 4%), Antisperm antibody test, Hormonal analysis (FSH, LH, TSH, testosterone, prolactin), Genetic testing (karyotype, *CFTR*, AZF deletions if severe oligospermia (< 5 million sperm/mL), Imaging studies employed in this setting may include the following: Transrectal ultrasonography, Scrotal ultrasonography and Vasography

An abnormal postcoital test result is observed in 10% of infertile couples. Indications for performing a postcoital test include semen hyperviscosity, increased or decreased semen volume with good sperm density, or unexplained infertility.If the test result is normal, consider sperm function tests, such as the following: Capacitation assay, Acrosome reaction assay, Sperm penetration assay, Hypoosmotic swelling test, Inhibin B level and Vitality stains.

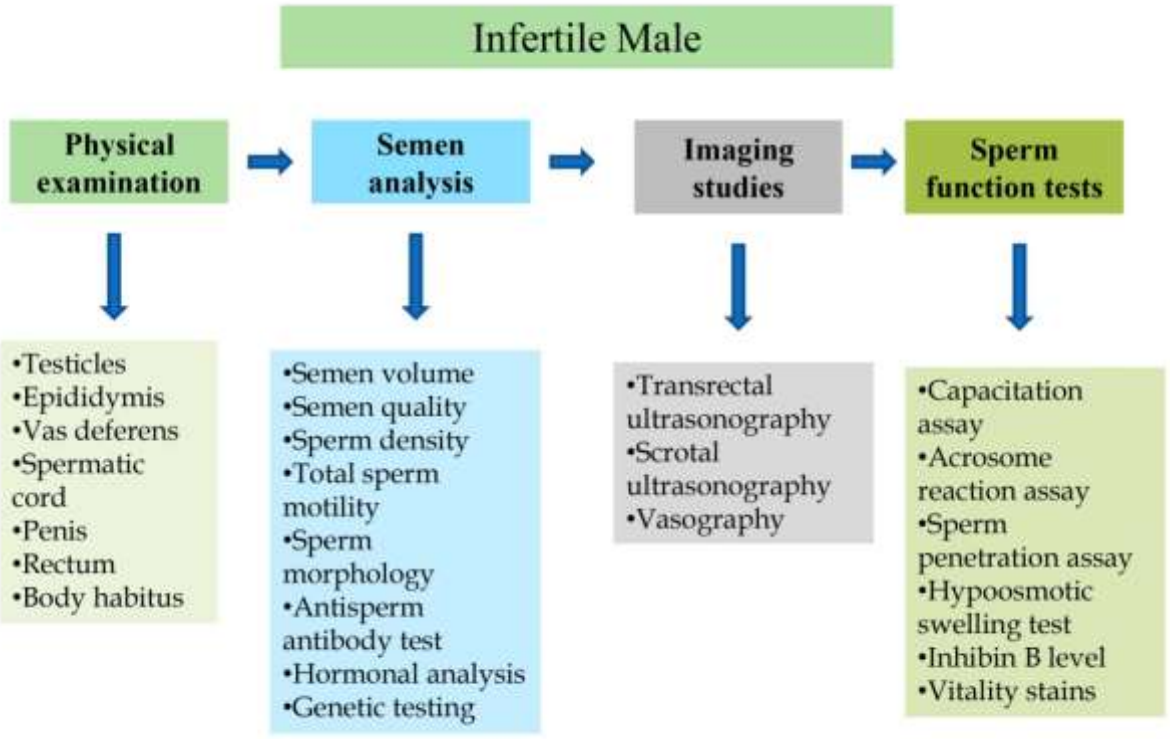

Table 1: Showing steps necessary for evaluation of an infertile male

5. Conclusion

The sole purpose of this manuscript is to comprehend the certain aetiologies of male infertility. The causes of male infertility are described along with possible correlations in the present scenario. The factors analysed above are responsible for the deterioration of sperm quality, mainly through decreased sperm concentration, vitality, motility and morphology. The factors related to lifestyle are a significant cause of male infertility in the world today. Further, it is also proved that sperm quality is essentially determined by: obesity, nicotine addiction, heavy exposure to electromagnetic compatibility radiation-emitting devices and alcohol consumption. However, there is a paucity of clinical data on the scale of the reproductive health problems mainly male infertility in India. Also, the diagnostic module is very vast and the patient should be evaluated algorithmically following standard diagnostic guidelines in a stepladder way, but due to paucity of health setup, lack of awareness and economic burden the patients notachieving as per expectation.

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