NAME: ADELANA ADESEWA MONISOLA

DEPARTMENT: NURSING

MATRIC NUMBER: 18/MHS02/013

1.Male infertility refers to a male's inability to cause pregnancy in a fertile female. In humans it accounts for 40–50% of infertility. It affects approximately 7% of all men. Male infertility is commonly due to deficiencies in the semen, and semen quality is used as a surrogate measure of male fecundity.

Causes

Factors relating to male infertility include:

Immune infertility: Antisperm antibodies (ASA) have been considered as infertility cause in around 10–30% of infertile couples. ASA production are directed against surface antigens on sperm, which can interfere with sperm motility and transport through the female reproductive tract, inhibiting capacitation and acrosome reaction, impaired fertilization, influence on the implantation process, and impaired growth and development of the embryo. Risk factors for the formation of antisperm antibodies in men include the breakdown of the blood-testis barrier, trauma and surgery, orchitis, varicocele, infections, prostatitis, testicular cancer, failure of immunosuppression and unprotected receptive anal or oral sex with men.

Genetics: Chromosomal anomalies and genetic mutations account for nearly 10–15% of all male infertility cases.

Klinefelter Syndrome: One of the most commonly known causes of infertility is Klinefelter Syndrome, affecting 1 out of 500–1000 newborn males[10] Klinefelter Syndrome is a chromosomal defect that occurs during gamete formation due to a non-disjunction error during cell division. Resulting in males having smaller testes, reducing the amount of testosterone and sperm production. Males with this syndrome carry an extra X chromosome (XXY), meaning they have 47 chromosomes compared to the normal 46 in each cell. This extra chromosome directly affects sexual development before birth and during puberty (links to learning disabilities and speech development have also been shown to be affected). There are varieties in Klinefelter Syndrome, where some cases may have the extra X chromosome in some cells but not others, referred to as Mosaic Klinefelter Syndrome, or where individuals have the extra X chromosome in all cells. The reduction of testosterone in the male body normally results in an overall decrease in the production of viable sperm for these individuals

thereby forcing them to turn to fertility treatments to father children.

Y chromosome deletions: Y chromosomal infertility is a direct cause of male infertility due to its effects on sperm production, occurring in 1 out of every 2000 males. Usually affected men show no sign of symptoms other than at times can exhibit smaller teste size. Men with this condition can exhibit azoospermia (no sperm production), oligozoospermia (small number of sperm production), or they will produce abnormally shaped sperm (teratozoospermia). This case of infertility occurs during the development of gametes in the male, where a normal healthy male will produce both X and a Y chromosome, affected males have genetic deletions in the Y chromosome. These deletions affect protein production that is vital for spermatogenesis. Studies have shown that this is an inherited trait; if a male is fathered by a man who also exhibited y chromosome deletions then this trait will be passed down. These individuals are thereby "Y-linked", although daughters are not affected due to the lack of the Y chromosome.

Other: Age, Abnormal set of chromosomes, Centriole, Neoplasm, e.g. seminoma, Idiopathic failure, Cryptorchidism, Trauma, Hydrocele, Hypopituitarism in adults, and hypopituitarism untreated in children (resulting in growth hormone deficiency and proportionate dwarfism.),Mumps, Malaria, Testicular cancer, drugs, alcohol

Defects in some cases

Acrosomal defects affecting egg penetration

Idiopathic oligospermia - unexplained sperm deficiencies account for 30% of male infertility.

Pre-testicular causes: Pre-testicular factors refer to conditions that impede adequate support of the testes and include situations of poor hormonal support and poor general health including:

Varicocele: Varicocele, is a condition of swollen testicle veins. It is present in 15% of normal men and in about 40% of infertile men. It is present in up to 35% of cases of primary infertility and 69–81% of secondary infertility.

Hypogonadotropic hypogonadism due to various causes:

Obesity increases the risk of hypogonadotropic hypogonadism.[18]Animal models indicate that obesity causes leptin insensitivity in the hypothalamus, leading to decreased Kiss1 expression, which, in turn, alters the release of gonadotropin-releasing hormone (GnRH).

Undiagnosed and untreated coeliac disease(CD): Coeliac men may have reversible infertility. Nevertheless, CD can present with several non-gastrointestinal symptoms

that can involve nearly any organ system, even in the absence of gastrointestinal symptoms. Thus, the diagnosis may be missed, leading to a risk of long-term complications. In men, CD can reduce semen quality and cause immature secondary sex characteristics, hypogonadism and hyperprolactinaemia, which causes impotence and loss of libido. The giving of gluten free diet and correction of deficient dietary elements can lead to a return of fertility. It is likely that an effective evaluation for infertility would best include assessment for underlying celiac disease, both in men and women.

Medications, including those that affect spermatogenesis such as chemotherapy, anabolic steroids, cimetidine, spironolactone; those that decrease FSH levels such as phenytoin; those that decrease sperm motility such as sulfasalazine and nitrofurantoin

Genetic abnormalities such as a Robertsonian translocation

Tobacco smoking: There is increasing evidence that the harmful products of tobacco smoking may damage the testicles and kill sperm, but their effect on male fertility is not clear. Some governments require manufacturers to put warnings on packets. Smoking tobacco increases intake of cadmium, because the tobacco plant absorbs the metal. Cadmium, being chemically similar to zinc, may replace zinc in the DNA polymerase, which plays a critical role in sperm production. Zinc replaced by cadmium in DNA polymerase can be particularly damaging to the testes.

DNA damage: Common inherited variants in genes that encode enzymes employed in DNA mismatch repair are associated with increased risk of sperm DNA damage and male infertility. As men age there is a consistent decline in semen quality, and this decline appears to be due to DNA damage. The damage manifests by DNA fragmentation and by the increased susceptibility to denaturation upon exposure to heat or acid, the features characteristic of apoptosis of somatic cells. These findings suggest that DNA damage is an important factor in male infertility.

Epigenetic: An increasing amount of recent evidence has been recorded documenting abnormal sperm DNA methylation in association with abnormal semen parameters and male infertility. Until recently, scientists have thought that epigenetic markers only affect the individual and are not passed down due to not changing the DNA. New studies suggest that environmental factors that changed an individual's epigenetic markers can be seen in their grandchildren, one such study demonstrating this through rats and fertility disruptors. Another study bred rats exposed to an endocrine disruptor, observing effects up to generation F5 including decreased sperm motility and decreased sperm count. These studies suggest that environmental factors that influence fertility can be felt for generations even without changing the DNA.

Post-testicular causes: Post-testicular factors decrease male fertility due to conditions

that affect the male genital system after testicular sperm production and include defects of the genital tract as well as problems in ejaculation:

Vas deferens obstruction

Lack of Vas deferens, often related to genetic markers for cystic fibrosis

Infection, e.g. prostatitis, Retrograde ejaculation, Ejaculatory duct obstruction, impotence

Diagnosis

The diagnosis of infertility begins with a medical history and physical exam by a physician, physician assistant, or nurse practitioner. Typically two separate semen analyses will be required. The provider may order blood tests to look for hormone imbalances, medical conditions, or genetic issues.

Medical history: The history should include prior testicular or penile insults (torsion, cryptorchidism, trauma), infections (mumps orchitis, epididymitis), environmental factors, excessive heat, radiation, medications, and drug use (anabolic steroids, alcohol, smoking). Sexual habits, frequency and timing of intercourse, use of lubricants, and each partner's previous fertility experiences are important. Loss of libido and headaches or visual disturbances may indicate a pituitary tumor. The past medical or surgical history may reveal thyroid or liver disease (abnormalities of spermatogenesis), diabetic neuropathy(retrograde ejaculation), radical pelvic or retroperitoneal surgery (absent seminal emission secondary to sympathetic nerve injury), or hernia repair (damage to the vas deferens or testicular blood supply). A family history may reveal genetic problems.

Physical examination: Usually, the patient disrobes completely and puts on a gown. The physician, physician assistant, or nurse practitioner will perform a thorough examination of the penis, scrotum, testicles, I vas deferens, spermatic cords, ejaculatory ducts, urethra, urinary bladder, anus and rectum.
An orchidometer can measure testicular volume, which in turn is tightly associated with both sperm and hormonal parameters. A physical exam of the scrotum can reveal a varicocele, but the impact of detecting and surgically correct a varicocele on sperm parameters or overall male fertility is debated.

Sperm sample

Semen sample obtaining: Semen sample obtaining is the first step in spermiogram. The optimal sexual abstinence for semen sample obtaining is of 2–7 days. The first way to obtain the semen sample is through masturbation, and the best place to obtain it is in the same clinic, as this way temperature changes during transport can be avoided, which can be lethal for some spermatozoa. A single semen sample is not determining

for disease diagnosis, so two different samples have to be analyzed with an interval between them of seven days to three months, as sperm production is a cyclic process. It is prudent to ask about possible sample loss, as that could mask true results of spermiogram. To obtain the sample, a sterile plastic recipient is put directly inside, always no more than one hour before being studied. Conventional preservatives shouldn't be used, as they have chemical substances as lubricants or spermicides that could damage the sample. If preservatives have to be used, for cases of religious ethics in which masturbation is forbidden, a preservative with holes is used. In case of paraplegia it is possible to use mechanic tools or electroejaculation. The sample should never be obtained through coitus interruptus for several reasons: Some part of ejaculation could be lost. Bacterial contamination could happen. The acid vaginal pH could be deleterious for sperm motility.

Also is very important to label the sample correctly the recipient with patient identification, date, hour, abstinence days, among other data required to be known.

Semen analysis

Semen quality: The volume of the semen sample, approximate number of total sperm cells, sperm motility/forward progression, and % of sperm with normal morphology are measured. This is the most common type of fertility testing. Semen deficiencies are often labeled as follows:

Oligospermia or oligozoospermia – decreased number of spermatozoa in semen

Aspermia – complete lack of semen

Hypospermia – reduced seminal volume

Azoospermia – absence of sperm cells in semen

Teratospermia – increase in sperm with abnormal morphology

Asthenozoospermia – reduced sperm motility

Necrozoospermia – all sperm in the ejaculate are dead

Leucospermia – a high level of white blood cells in semen

Normozoospermia or normospermia – It is a result of semen analysis that shows normal values of all ejaculate parameters by WHO but still there are chances of being infertile. This is also called as unexplained Infertility

There are various combinations of these as well, e.g. *Teratoasthenozoospermia*, which is reduced sperm morphology and motility. Low sperm counts are often associated with decreased sperm motility and increased abnormal morphology, thus the terms "oligoasthenoteratozoospermia" or "oligospermia" can be used as a catch-all.

Blood sample: Common hormonal test include determination of FSH and testosterone levels. A blood sample can reveal genetic causes of infertility, e.g. Klinefelter syndrome, a Y chromosome microdeletion, or cystic fibrosis.

Ultrasonography: Scrotal ultrasonography is useful when there is a suspicion of some particular diseases. It may detect signs of testicular dysgenesis, which is often related to an impaired spermatogenesis and to a higher risk of testicular cancer. Scrotum ultrasonography may also detect testicular lesions suggestive of malignancy. A decreased testicular vascularization is characteristic of testicular torsion, whereas hyperemia is often observed in epididymo-orchitis or in some malignant conditions such as lymphoma and leukemia.[4] Doppler ultrasonography useful in assessing venous reflux in case of a varicocele, when palpation is unreliable or in detecting recurrence or persistence after surgery, although the impact of its detection and surgical correction on sperm parameters and overall fertility is debated. Dilation of the head or tail of the epididymis is suggestive of obstruction or inflammation of the male reproductive tract. Such abnormalities are associated with abnormalities in sperm parameters, as are abnormalities in the texture of the epididymis. Scrotal and transrectal ultrasonography (TRUS) are useful in detecting uni- or bilateral congenital absence of the vas deferens (CBAVD), which may be associated with abnormalities or agenesis of the epididymis, seminal vesicles or kidneys, and indicate the need for testicular sperm extraction. TRUS plays a key role in assessing azoospermia caused by obstruction, and detecting distal CBAVD or anomalies related to obstruction of the ejaculatory duct, such as abnormalities within the duct itself, a median cyst of the prostate (indicating a need for cyst aspiration), or an impairment of the seminal vesicles to become enlarged or emptied.

Prevention

Some strategies suggested or proposed for avoiding male infertility include the following:

Avoiding smoking as it damages sperm DNA

Avoiding heavy marijuana and alcohol use.

Avoiding excessive heat to the testes.

Maintaining optimal frequency of coital activity: sperm counts can be depressed by daily coital activity and sperm motility may be depressed by coital activity that takes place too infrequently (abstinence 10–14 days or more).

Wearing a protective cup and jockstrap to protect the testicles, in any sport such as baseball, football, cricket, lacrosse, hockey, softball, paintball, rodeo, motorcross, wrestling, soccer, karate or other martial arts or any sport where a ball, foot, arm, knee

or bat can come into contact with the groin.

Diet: Healthy diets (i.e. the Mediterranean diet) rich in such nutrients as omega-3 fatty acids, some antioxidants and vitamins, and low in saturated fatty acids (SFAs) and trans-fatty acids (TFAs) are inversely associated with low semen quality parameters. In terms of food groups, fish, shellfish and seafood, poultry, cereals, vegetables and fruits, and low-fat dairy products have been positively related to sperm quality. However, diets rich in processed meat, soy foods, potatoes, full-fat dairy products, coffee, alcohol and sugar-sweetened beverages and sweets have been inversely associated with the quality of semen in some studies. The few studies relating male nutrient or food intake and fecundability also suggest that diets rich in red meat, processed meat, tea and caffeine are associated with a lower rate of fecundability. This association is only controversial in the case of alcohol. The potential biological mechanisms linking diet with sperm function and fertility are largely unknown and require further study.

Treatment

Treatments vary according to the underlying disease and the degree of the impairment of the male's fertility. Further, in an infertility situation, the fertility of the female needs to be considered.

Pre-testicular conditions can often be addressed by medical means or interventions.

Testicular-based male infertility tends to be resistant to medication. Usual approaches include using the sperm for intrauterine insemination (IUI), in vitro fertilization (IVF), or IVF with intracytoplasmatic sperm injection(ICSI). With IVF-ICSI even with a few sperm pregnancies can be achieved.

Obstructive causes of post-testicular infertility can be overcome with either surgery or IVF-ICSI. Ejaculatory factors may be treatable by medication, or by IUI therapy or IVF.

Vitamin E helps counter oxidative stress, which is associated with sperm DNA damage and reduced sperm motility. A hormone-antioxidant combination may improve sperm count and motility. Giving oral antioxidants to men in couples undergoing in vitro fertilization for male factor or unexplained subfertility may lead to an increase in the live birth rate but overall the risk of adverse effects is unclear.

Hormonal therapy: Administration of luteinizing hormone (LH) (or human chorionic gonadotropin) and follicle-stimulating hormone (FSH) is very effective in the treatment of male infertility due to hypogonadotropic hypogonadism.[46] Although controversial,[47] off-label clomiphene citrate, an antiestrogen, may also be effective by elevating gonadotropin levels.

Though androgens are absolutely essential for spermatogenesis and therefore male fertility, exogenous testosterone therapy has been found to be ineffective in benefiting men with low sperm

count. This is thought to be because very high local levels of testosterone in the testes (concentrations in the seminiferous tubules are 20- to 100-fold greater than circulating levels) are required to mediate spermatogenesis, and exogenous testosterone therapy (which is administered systemically) cannot achieve these required high local concentrations (at least not without extremely supraphysiological dosages). Moreover, exogenous androgen therapy can actually impair or abolish male fertility by suppressing gonadotropin secretion from the pituitary gland, as seen in users of androgens/anabolic steroids (who often have partially or completely suppressed sperm production). This is because suppression of gonadotropin levels results in decreased testicular androgen production (causing diminished local concentrations in the testes) and because FSH is independently critical for spermatogenesis. In contrast to FSH, LH has little role in male fertility outside of inducing gonadal testosterone production.

Estrogen, at some concentration, has been found to be essential for male fertility/spermatogenesis. However, estrogen levels that are too high can impair male fertility by suppressing gonadotropin secretion and thereby diminishing intratesticular androgen levels. As such, clomiphene citrate (an antiestrogen) and aromatase inhibitors such as testolactone or anastrozole have shown effectiveness in benefiting spermatogenesis.

Low-dose estrogen and testosterone combination therapy may improve sperm count and motility in some men, including in men with severe oligospermia.

2. Semen, also known as **seminal fluid**, is an organic fluid that contains spermatozoa. It is secreted by the gonads (sexual glands) and other sexual organs of male or hermaphroditicanimals and can fertilize the female ovum. In humans, seminal fluid contains several components besides spermatozoa: proteolytic and other enzymes as well as fructose are elements of seminal fluid which promote the survival of spermatozoa, and provide a medium through which they can move or "swim". Semen is produced and originates from the seminal vesicle, which is located in the pelvis. The process that results in the discharge of semen is called ejaculation. Semen is also a form of genetic material. In animals, semen has been collected for cryoconservation. Cryoconservation of animal genetic resources is a practice that calls for the collection of genetic material in efforts for conservation of a particular breed.

Physiology of Semen

Fertilization

Depending on the species, spermatozoa can fertilize ova externally or internally. In external

fertilization, the spermatozoa fertilize the ova directly, outside of the female's sexual organs. Female fish, for example, spawn ova into their aquatic environment, where they are fertilized by the semen of the male fish.

During internal fertilization, however, fertilization occurs inside the female's sexual organs. Internal fertilization takes place after insemination of a female by a male through copulation. In most vertebrates, including amphibians, reptiles, birds and monotrememammals, copulation is achieved through the physical mating of the cloaca of the male and female. In marsupial and placental mammals, copulation occurs through the vagina.

Human semen

Composition

During the process of ejaculation, sperm passes through the ejaculatory ducts and mixes with fluids from the seminal vesicles, the prostate, and the bulbourethral glands to form the semen. The seminal vesicles produce a yellowish viscous fluid rich in fructose and other substances that makes up about 70% of human semen. The prostatic secretion, influenced by dihydrotestosterone, is a whitish (sometimes clear), thin fluid containing proteolytic enzymes, citric acid, acid phosphatase and lipids. The bulbourethral glands secrete a clear secretion into the lumen of the urethra to lubricate it. Sertoli cells, which nurture and support developing spermatocytes, secrete a fluid into seminiferous tubules that helps transport sperm to the genital ducts. The ductuli efferentes possess cuboidal cells with microvilli and lysosomal granules that modify the ductal fluid by reabsorbing some fluid. Once the semen enters the ductus epididymis the principal cells, which contain pinocytotic vessels indicating fluid reabsorption, secrete glycerophosphocholine which most likely inhibits premature capacitation. The accessory genital ducts, the seminal vesicle, prostate glands, and the bulbourethral glands, produce most of the seminal fluid.Seminal plasma of humans contains a complex range of organic and inorganic constituents.The seminal plasma provides a nutritive and protective medium for the spermatozoa during their journey through the female reproductive tract. The normal environment of the vagina is a hostile one (c.f. sexual conflict) for spermcells, as it is very acidic (from the native microflora producing lactic acid), viscous, and patrolled by immune cells. The components in the seminal plasma attempt to compensate for this hostile environment. Basic amines such as putrescine, spermine, spermidine and cadaverine are responsible for the smell and flavor of semen. These alkaline bases counteract and buffer the acidic environment of the vaginal canal, and protect DNA inside the sperm from acidic denaturation.

Appearance and consistency

Semen is typically translucent with white, grey or even yellowish tint. Blood in the semen can cause a pink or reddish colour, known as hematospermia, and may indicate a medical problem which should be evaluated by a doctor if the symptom persists. After ejaculation, the latter part of the ejaculated semen coagulates immediately,forming globules, while the earlier part of the ejaculate typically does not. After a period typically ranging from 15 – 30 minutes, prostate-specific antigen present in the semen causes the decoagulation of the seminal coagulum. It is postulated that the initial clotting helps keep the semen in the vagina,while liquefaction frees the sperm to make their journey to the ova.

Quality

Semen quality is a measure of the ability of semen to accomplish fertilization. Thus, it is a measure of fertility in a man. It is the sperm in the semen that is the fertile component, and therefore semen quality involves both sperm quantity and sperm quality.

Quantity

The volume of semen ejaculate varies but is generally about 1 teaspoonful or less. A review of 30 studies concluded that the average was around 3.4 milliliters (mL), with some studies finding amounts as high as 5.0 mL or as low as 2.3 mL. In a study with Swedish and Danish men, a prolonged interval between ejaculations caused an increase of the sperm count in the semen but not an increase of its amount.

Increasing semen volume: Some dietary supplements have been marketed with claims to increase seminal volume. Like other supplements, including so-called herbal viagra, these are not approved or regulated by the Food and Drug Administration(as licensed medications would be), and none of the claims have been scientifically verified. Similar claims are made about traditional aphrodisiac foods, with an equal lack of verification.

Storage: Semen can be stored in diluents such as the *Illini Variable Temperature* (IVT) diluent, which have been reported to be able to preserve high fertility of semen for over seven days. The IVT diluent is composed of several salts, sugars and antibacterial agents and gassed with CO2.

Semen cryopreservation can be used for far longer storage durations. For human sperm, the longest reported successful storage with this method is 21 years.