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CAUSES OF INFERTILITY

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ABSTRACT

Infertility is a common problem affecting one couple in six. It can be defined as the incapacity to fulfill pregnancy after reasonable time of sexual intercourse with no contraceptive measures taken. Male and female infertility affects close to 50 million couples worldwide according to a recent World Health Organization estimate. Many of the 25-30% of couples with idiopathic infertility likely have a genetic etiology for their condition. Genetic and genomic causes of infertility can be divided into cytogenetic anomalies, gene defects, and epigenetic aberrances. During the past two decades there have been three important changes in infertility practice. First, the introduction of assisted reproduction technologies has provided an opportunity to study basic reproductive processes. Second, societal changes have occurred such as the increase in the proportion of women over 35 years old seeking pregnancy. This fact is due to a later age for marriage and postponement of pregnancy. Third, the development of molecular biology and genetics has become very important for the study, diagnosis and assessment of couples, many of them considered until now as "unexplained infertile couples".

Keywords : Infertility, spermatogenesis, oogenesis

INTRODUCTION

Definition:

Infertility is defined as a failure to conceive within one or more years of regular unprotected coitus.

Type of infertility:

Primary infertility denotes those patients who have never conceived. Secondary infertility indicates previous pregnancy but failure to conceive subsequently.

Fecund ability is defined as the probability of achieving a pregnancy within one menstrual cycle. In a healthy young couple it is 20 per cent. Fecundity is the probability of achieving a live birth within a single cycle.

Incidence:

Eighty per cent of the couples achieve conception if they so desire, within one year of having regular intercourse with adequate frequency. Another 10 per cent will achieve the objective by the end of second year. As such, 10 per cent remain infertile by the end of second year.

Factors responsible for fertility are:

- Healthy spermatozoa should be deposited high in the vagina at or near the cervix (male factor).
- The spermatozoa should undergo changes and acquire motility (cervical factor).
- The motile spermatozoa should ascend through the cervix into the uterine cavity and the fallopian tubes.
- There should be ovulation (ovarian factor).
- The fallopian tubes should be patent and oocyte should be picked up by the fimbriated end of the tube (tubal factor)
- The spermatozoa should fertilise the oocyte at the ampulla of the tube.
- The embryo should reach the uterine cavity after 3-4 days of fertilisation.
- The endometrium should be receptive for implantation and the corpus luteum should function.

Physiological consideration:

Due to anovulation infertility is the rule prior to puberty and after menopause. But it should be remembered that the girl may be pregnant even before menarche and pregnancy is possible within few months of menopause. Conception is not possible during pregnancy as the pituitary gonadal axis is suppressed by HCG and no ovulation. During lactation, infertility is said to be relative. Despite the fact that the patient is amenorrhoeic during lactation, Ovulation and conception can occur. However, in fully lactating women, pregnancy is unlikely up to 10 weeks postpartum.

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Conception depends on the fertility potential of both the male and female partner. The male is directly responsible in about 30-40 per cent, the female in about 40-55 per cent and both are responsible in about 10 per cent cases. The remaining 10 percent, is unexplained in spite of thorough investigations with modern technical knowhow. It is also strange that 4 out of 10 patients of unexplained category become pregnant within 3 years without having any specific treatment.

It is emphasised that the relative subfertility of one partner may sometimes be counterbalanced by the high fertility of the other.

FAULTS IN THE MALE

- Defective spermatogenesis.
- Obstruction of the efferent duct system.
- Failure to deposit sperm high in the vagina.
- Errors in the seminal fluid.

Defective spermatogenesis:

FSH stimulates spermatogenesis from basal cells of the seminiferous tubules. Sertoli cells envelop the germ cells and support spermatogenesis. Sertoli cell function is controlled by should be 1-2°F less than the body temperature. LH is required for the synthesis of testosterone from the Leydig cells. FSH also stimulates the Sertoli cells to produce androgen binding proteins and inhibin B. ABP binds to testosterone and dihydrotestosterone to maintain the local high concentration of androgens. Spermatogenesis and sperm maturation need a high androgenic environment. Inhibin B inhibits FSH secretion. Spermatogenesis is controlled predominantly by the genes on Y chromosome. Approximately 74 days are required to complete the process of spermatogenesis. Additional 12-20 days are needed for spermatozoa to travel the epididymis.

The causes of defective spermatogenesis are:

- **Congenital**
 - Undescended testes – the hormone secretion remains unaffected, but the spermatogenesis is depressed. Vas deference is absent in about 1-2per cent if infertile males.
 - Kartagener syndrome there is loss ciliary function and sperm motility.
 - Hypospadias causes failure to deposit sperm high in vagina.
- **Thermal factor:**

The scrotal temperature is raised in conditions such as varicocele, big hydrocele or filariasis. Varicocele probably interferes with the cooling mechanism or increases catecholamine concentration. Other causes are using tight under-garment or working in hot atmosphere. In all these conditions, the depressed spermatogenesis may be temporary and reversible.

- Infection:
- Mumps orchitis after puberty may permanently damage spermatogenesis. (b) The quality of the sperm is adversely affected by chronic systemic illness like bronchiectasis. Bacterial or viral infection of the seminal vesicle or prostate depresses the sperm count. (c) *T. mycoplasma* or *Chlamydia trachomatis* infection is also implicated.
- General factors:

Chronic debilitating diseases, malnutrition or heavy smoking reduce spermatogenesis. Alcohol inhibits spermatogenesis either by suppressing Leydig cell synthesis of testosterone or possibly by suppressing gonadotropin levels.

Pre-testicular	Testicular	Post-testicular
Endocrine • Gonadotropin deficiency. • Thyroid dysfunction • Hyperprolactinaemia Psychosexual - Erectile dysfunction. - Importance. Drugs - Antihypertensive - Antipsychotics Genetic - 47 XXY - Y chromosome deletions.	• Immotile cilia (Kartagener) syndrome • Cryptorchidism • Infection(mumps orchitis) • Toxins: Drugs, Radiation • Varicocele • Immunologic • Sertoli-cell-only syndrome • Primary testicular failure • Oligoastheno-teratozoospermia	Obstruction of efferent duct • Congenital - Absence of vas deferens - Young's syndrome • Acquired Infection: Tuberculosis, Gonorrhoea • Surgucal - Herneorrhaphy, - Vasectomy. Others • Ejaculatory failure • Retrograde ejaculation • Hypospadias • Bladder neck surgery

- **Endocrine:**

Testicular failure due to gonadotropin deficiency is rare. FSH level is raised in idiopathic testicular failure with germ cell hypoplasia Hyperprolactinamia is associated with importance.

- **Genetic:**

Common chromosomal abnormality in azoospermic male is Klinefelter's syndrome. Gene deletion have been detected on the long arm of Y chromosome for patients with severe oligospermia.

- **Iatrogenic:**

Radiation, cytotoxic drugs, nitrofurantoin, cimetidine, β blockers, antigens may be the causes, antidepressant drugs are likely to hinder spermatogenesis.

- **Immunological factor:**

Antibodies against spermatozoal surface antigens may be the causes of infertility. The results in clumping of the spermatozoa after ejaculation.

- **Obstruction of the efferent ducts:**

The efferent ducts may be obstructed by infection like tubercular, gonococcal or by surgical trauma (herniorrhaphy) following vasectomy. In Young's syndrome there is epididymal obstruction and bronchiectasis.

Failure to deposit sperm high in the vagina (coital problems)

- Erectile dysfunction
- Ejaculation defect – premature, retrograde or absence of ejaculation
- Hypospadias

Sperm abnormality –loss of sperm motility, abdominal sperm morphology are the important factors.

Errors in the seminal fluid

- Unusually high or low volume of ejaculate
- Low fructose content
- High prostaglandin content
- Undue viscosity

CAUSES OF FEMALE INFERTILITY

According to FIGO manual (1990) causes are – tubal and peritoneal factors (20-35%), ovulatory factor (30-40%) and endometriosis (1-10%).

Ovarian factors – The ovulatory dysfunctions encompass:

- Anovulation or oligo-ovulation
- Decreased ovarian reserve
- Luteinised unruptured follicle

Anovulation or oligo-ovulation:

The ovarian activity is totally dependent on the normal secretion of gonadotrophins depends on the pulsatile release of GnRH from hypothalamus. As such, ovarian dysfunction is likely to be linked with disturbed hypothalamo-pituitary-ovarian axis either primary or secondary from thyroid or adrenal dysfunction.

Thus, the disturbance may result not only in anovulation but may also produce oligomenorrhoea or even amenorrhoea. Anovulatory cycles usually represent a lesser degree of disturbance with these normal pathways then does amenorrhoea. Possible causes of anovulation are given schematically.

As there is no ovulation, there is no corpus luteum formation. In the absence of progesterone, there is no secretory endometrium in the second half of the cycle. The other features of ovulation are absent.

Luteal phase defect (LPD)

In this condition there is inadequate growth and function of the corpus luteum. There is inadequate progesterone secretion. The life span of corpus luteum is shortened to less than 10 days. As a result, there is inadequate secretory changes in the endometrium which hinder implantation. LPD is due to defective folliculogenesis which again may be due to varied reasons. Drug induced ovulation, decreased level of FSH and/or LH, elevated prolactin, subclinical hypothyroidism, older women, pelvic endometriosis, dysfunctional uterine bleeding are the impotent causes.

Luteinised unruptured follicular syndrome (trapped ovum):

In this condition, the ovum is trapped inside the follicle which gets luteinised. The cause is follicle which gets luteinised. The cause is obscure but may be associated with pelvic endometriosis or with hyperprolactinaemia.

Tubal and potential factors are responsible for about 30-40 per cent cases of female infertility.

The obstruction of the tubes may be due to – (a) Pelvic infections causing: (i) peritubal adhesions (ii) Endosalpingeal damage. (b) Previous tubal surgery or sterilisation. (c) Salpingitis isthmica nodosa. (d) Tubal endometriosis and others. (e) Polyps mucous debris within the tubal lumen, or tubal spasm.

Peritoneal factors:

In addition to adhesions, even minimal endometriosis may produce infertility. Deep dyspareunia often troubles the patient. The possible multifactorial mechanisms which operate in minimal endometriosis are depicted schematically below.

Uterine factors:

The endometrium must be sufficiently receptive enough for effective nidation and growth of the fertilised ovum. The possible factors that hinder nidation are uterine hypoplasia, inadequate secretory endometrium, inadequate secretory endometrium, fibroid uterus, endometritis, uterine synechiae or congenital malformation of uterus.

Cervical factors:

Anatomic – Anatomic defects preventing sperm ascent may be due to congenital elongation of the cervix, second degree uterine prolapse and acute retroverted uterus. These conditions prevent the external os to bathe in the seminal pool. Pinhole os may at times be implicated, or the cervical canal may be occluded by a polyp.

Physiologic – The fault lies in the composition of the cervical mucus, so much that the spermatozoa fail to penetrate the mucus. The mucus may be scanty following amputation, conisation or deep cauterisation of the cervix. The abnormal constituents include excessive, viscous or purulent discharge as in chronic cervicitis. Presence of antisperm or sperm immobilising antibodies may be implicated as immunological factor of infertility.

Vaginal factors - Atresia vagina, transvers vaginal causing dyspareunia are included in the congenital group.

Vaginitis and purulent discharge may at times be implicated but pregnancy too often occurs in presence of vaginitis, specific or nonspecific. However, dyspareunia may be the real problem in such cases.

Combined factors:

- These include the presence of factors both in the male and female partners causing infertility.
- General factors: Advanced age of the wife beyond 35 is related but spermatogenesis continues throughout life although ageing reduces the fertility in male also.
- Infrequent intercourse, lack of knowledge of coital technique and timing of coitus to utilise the fertile period are very much common even amongst the literate couples.
- Apareunia and dyspareunia.
- Anxiety and apprehension.
- Use of lubricants during intercourse- which may be spermicidal.
- Immunological factors.

PROGNOSIS OF INFERTILITY

The pregnancy rate within two years after the start of investigation, ranges between 30-40 per cent. The rate is however, increased to about 50-60 %, if AID cases are included.

CONCLUSION

Patients who have expressed a desire to become parents, but who are in a high-risk group for infertility based on their age, should have a basic fertility evaluation and be referred to a specialist in a timely manner in order to maximize their fertility potential. As new technology becomes available, fertility treatment is now accessible to more people, and success rates and safety are improving all the time.

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