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TOPIC- INFERTILITY.

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**INTRODUCTION**

* Successful pregnancy requires the union of a mature ovum and sperm-fertilization and its subsequent implantation in the uterus.
* This process can be disturbed at several stages resulting in infertility.
* Infertility is a complex disorder with significant psychosocial and medical problem affecting couples worldwide.

**DEFINITION**

“Infertility is a disease of the reproductive system defined by the failure to achieve a clinical pregnancy after 12 months or more of regular unprotected sexual intercourse – WHO”.

**TYPES OF INFERTILITY**

**Primary infertility** denotes those patients who have never conceived.

**Secondary infertility** indicates previous pregnancy but failure to conceive subsequently.

**Incidence**

Eighty percent of the couples achieve conception if they so a strong feeling of wanting for pregnancy, within one year of having regular intercourse with adequate frequency (4–5 times a week).

Another 10 percent will achieve the objective by the end of second year.

As such10 percent remain infertile by the end of second year.

**CAUSES OF INFERTILITY**

Conception depends on the fertility potential of both the male and female partner. **The male is directly** **responsible in about 30–40 percent,**

**The female in about 40–55 percent and**

**Both are responsible in about 10 percent cases.**

The remaining 10 percent, is **unexplained,** in spite of thorough investigations withmodern 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.

**FAULTS IN THE MALE**

1. Defective spermatogenesis

2. Obstruction of the efferent duct system

3. Failure to deposit sperm high in the vagina

4. Errors in the seminal fluid.

**1. DEFECTIVE SPERMATOGENESIS**

FSH stimulates spermatogenesis from basal cells of the seminiferous tubules. Sertoli cells envelope the germ cells and support spermatogenesis. Sertoli cell function is controlled by FSH and testosterone. Scrotal temperature 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 (ABP) 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 neededfor spermatozoa to travel the epididymis.

**Causes of Male Infertility**

The important causes of male infertility are:

(1) Hypothalamic-pituitary disorders (1–2%).

(2) Primary gonadal disorders (30–40%).

(3) Disorders of sperm transport (10–20%) and

(4) Idiopathic (40–50%).

**Congenital**

−**− Undescended testes:** the hormone secretion remains unaffected, but the spermatogenesis is depressed. Vas deferens is absent (bilateral) in about 1–2 percent of infertile males.

−**− Kartagener syndrome** (autosomal disease)— there is loss of ciliary function and sperm motility.

**−− Hypospadias** causes failure to deposit sperm high in vagina(congenital condition in males in which the opening of the urethra is on the underside of the penis.)

**Thermal Factor:**

The scrotal temperature is raised in conditions such as varicocele. Varicocele probably interferes with the cooling mechanism or increases catecholamine concentration. However, no definite association between varicoceles and infertility has been established.

**Infection:**

a) 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 debilitatingdiseases, malnutrition or heavy smoking reduce spermatogenesis. Alcohol inhibits spermatogenesis either by suppressing Leydig cell synthesis of testosterone or possibly by suppressing gonadotropin levels.

**Endocrine:** Testicular failure due to gonadotropin deficiency **(Kallmann’s syndrome)** is rare. FSH level is raised in idiopathic testicular failure with germ cell hypoplasia **(Sertoli-cell-only-syndrome)**. Hyperprolactinemia is associated with impotence.

**Genetic:** Common chromosomal abnormalityin azoospermia male is Klinefelter’s syndrome (47 XXY). Gene deletion have been detected in the long-arm of Y chromosome (Yq) for patients with severe oligospermia and azoospermia.

**Iatrogenic:** Radiation, cytotoxic drugs, nitro-furantoin, cimetidine, b blockers, antihypertensive, anticonvulsant, and antidepressant drugs are likely to hinder spermatogenesis.

**Immunological factor** Antibodies againstspermatozoa surface antigens may be the cause of infertility. This results in clumping of the spermatozoa after ejaculation.

**CAUSES OF FEMALE INFERTILITY**

**Anovulation or oligo-ovulation**

The ovarian activity is totally dependent on the gonadotropins and the normal secretion of gonadotropins depends on the pulsatile release of GnRH from hypothalamus. As such, **ovarian** **dysfunction is likely to be linked with disturbed hypothalamus-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 oligomenorrhea (infrequent menstrual period) or even amenorrhea.

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.

**Luteal Phase Defect (LPD)**

In this condition, there is inadequate growth and function of the corpus luteum. There is inadequate progesterone secretion. The lifespan 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 (maturation of ovation follicle) 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 important causes.

**Luteinized Unruptured Follicular Syndrome (Trapped Ovum)**

In this condition, the ovum is trapped inside the follicle, which gets luteinized. The cause is **pelvic endometriosis or** **with hyperprolactinemia.**

**Tubal and peritoneal factors** are responsible for about30–40 percent cases of female infertility. The obstruction of the tubes may be due to: -

(a) Pelvic infections causing peri tubal adhesions (b) Previous tubal surgery or sterilization. (c) Salpingitis (d) Tubal endometriosis e) Polyps or mucous debris within the tubal lumen, or tubal spasm.

**Peritoneal factors: a)** In addition to peri tubal adhesions,even minimal endometriosis may produce infertility) (Deep dyspareunia) painful intercourse cause inadequate lubricants, rough sex, trauma or negative feeling about a partner too often troubles the patient

**Uterine factors:** The endometrium must be sufficientlyreceptive enough for effective nidation (implantation) and growth of the fertilized ovum

**The possible factors that hinder nidation are** uterine hypoplasia (underdevelopment or incomplete development of tissue or organ), inadequate secretoryendometrium, fibroid uterus, endometritis (tubercular in particular), congenital malformation of uterus (bicornuate unicorn ate).

**Cervical factors**

**Anatomic:** Anatomic defects preventing spermascent may be due to **congenital elongation of the cervix,** second degree uterine prolapse. 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 ofthe cervical mucus, so much that the spermatozoa fail to penetrate the mucus. The mucus may be scanty following amputation, conization or deep cauterization of the cervix. The abnormal constituents include excessive, viscous or purulent discharge as in chronic cervicitis. Presence of antispam or sperm immobilizing antibodies may be implicated as immunological factor of infertility.

**Vaginal factors**

Atresia of vagina (partial or complete), transverse vaginal septum, narrow introitus 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. 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.

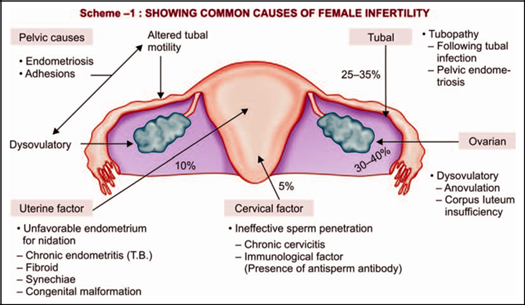
General factors: Advanced age of the wife beyond 35 years is related but spermatogenesis continues throughout life although aging reduces the fertility in male also.

Infrequent intercourse, lack of knowledge of coital technique and timing of coitus to utilize the fertile period are very much common even amongst the literate couples.

Anxiety and apprehension (unpleasant or bad will happen).

Use of lubricants during intercourse, which may be spermicidal.

Immunological factors



**INVESTIGATIONS OF INFERTILITY**

**Objectives of investigation**

* + To detect the etiological factor(s).
  + To rectify the abnormality in an attempt to improve the fertility.
  + To give assurance with explanation to the couple if no abnormality is detected.

**When to investigate?** As per the definition, theinfertile couple should be investigated after one year of regular unprotected intercourse with adequate frequency. The interval is however, shortened to 6 months after the age of 35 years of the woman and 40 years of man.

**What to investigate?** The basic investigationsto be carried out are: (I) Semen analysis. (ii) Confirmation of ovulation and (iii) Confirmation of tubal patency.

It is important that both partners should come at the first visit. Detailed general and reproductive history should be taken in presence of both. However, the clinical examination of each partner is carried out separately. No one is to be blamed

**Clinical Approach to investigation**

 **Male**   **Female**

**Male**

**a) History collection**

* Age, duration of marriage, history of previous marriage, are to be noted.
* A general medical history should be taken with special reference to sexually transmitted diseases, mumps orchitis after puberty, diabetes, recurrent chest infection or bronchiectasis.
* Enquiry about relevant surgery such as herniorrhaphy, operation on testes,
* also, about the sexual history,
* social habits, particularly heavy smoking and alcohol are of importance and to be made.
* A full physical examination is performed to determine the general state of health
* Examination of the reproductive system includes—inspection and palpation of the genitalia. Attention should be paid to the size and consistency of the testicles. Testicular volume (measured by an orchidometer) should be at least 20 ml. Presence of varicocele (compression of vein) should be elicited in the upright position.

**b) Investigations**

* Routine investigations include urine and blood examination including postprandial sugar.
* Semen analysis: This should be the first step in investigation because, if some gross abnormalities are detected (example being absence of sperm), the couple should be counseled for the need of assisted reproductive technology
* Collection: The semen is collected in a clean wide mouthed dry glass jar. The sample so collected should be sent to the laboratory as early as possible so that the examination can be performed within 2 hours. The coitus should be avoided for 2–3 days prior to the test (abstinence
* In selected cases, biochemical tests of creatine phosphokinase and reactive oxygen species are done as sperm function tests. Creatinine phosphokinase helps sperm transport while reactive oxygen species and the peroxides interfere with sperm function.

Normal male fertility requires a count of over 20 million spermatozoa per ml and a progressive motility of over 32 percent. Semen values normally vary widely. Two properly performed semen analysis at least 4 weeks apart should be done when one report is abnormal.

**SEMEN ANALYSIS: Normal reference value.**

Volume 2.0ml or more

Ph 7.2-7.8

Sperm concentration 20 million or more

Total sperm count > 40million per ejaculate

Motility 50% or more progressive forward motility

Morphology 15% or more normal form

Viability 75% or more living

Leucocytes less than 1 million/ml

Sperm agglutination < 2(scale 0-3)

**THE TERM**

* **Aspermia:** Failure of emission of semen (noejaculate).
* **Oligospermia/Oligozoospermia:** Sperm count isless than 20 million per ml.
* **Polyzoospermia:** Count is more than 350 million/ml.
* **Azoospermia:** No spermatozoan in the semen.
* **Asthenozoospermia:** Reduced sperm motility.
* **Leucocytospermia:** Increased white cells in semen.
* **Normozoospermia:** Spermatozoa are dead ormotionless.
* **Teratozoospermia:** > 70% spermatozoa withabnormal morphology.
* **Oligoasthenoteratozoospermia:** Disturbance ofall 3 variables.

**In-Depth Evaluation**

These are needed in cases of: - Azoospermia, Oligospermia, Low volume ejaculate, and Problems of sexual potency.

**HORMONAL STUDY OF: -Serum FSH, LH, testosterone, prolactin, and TSH:** Testicular dysfunction causes rise in FSH and LH.

Low level of FSH and LH suggest hypogonadotropic hypogonadism.

Leydig cell dysfunction causes low testosterone and high LH level.

Elevated prolactin due to pituitary adenoma may cause impotency.

**Fructose content in the seminal fluid:** Its absencesuggests congenital absence of seminal vesicle or portion of the ductal system or both.

**Testicular biopsy:** is done to differentiate primarytesticular failure from obstruction as a cause of azoospermia or severe oligospermia. **The biopsy material is to be sent in Bouin’s solution and not in formal saline.** Testicular tissues may be cryopreservedfor future use in IVF/ICSI.

**Transrectal ultrasound (TRUS):** is done to visualizethe seminal vesicles, prostate and ejaculatory ducts obstruction.

Indications of TRUS are: (I) Azoospermia or severe oligospermia with a normal testicular volume, (ii) Abnormal digital rectal examination, (iii) Ejaculatoryduct abnormality (cysts, dilatation or calcification), (iv) Genital abnormality (hypospadias).

**Vasogram** is a radiographic study done to evaluate theejaculatory duct obstruction. It is mostly replaced by TRUS.

**Karyotype analysis:** This is to be done in cases withazoospermia or severe oligospermia and raised FSH. Klinefelter’s syndrome (XXY) is the commonest. Micro deletions of the long arm of Y chromosome can also cause severe seminal abnormalities.

**Immunological tests:** Two types of antibodies havebeen described—sperm agglutinating and sperm immobilizing; the latter is probably related to infertility. The antibodies are produced following infection (orchitis), trauma or vasectomy. These antibodies can be detected from the serum by the sperm immobilizing test. Presence of sperm antibodies in the cervical mucus is demonstrated by **postcoital test** (**Presence of plenty of pus cells** requires prostaticmassage. The collected fluid is to be examined by staining and culture to detect the organisms and appropriate antibiotic sensitivity.

**FEMALE**

**A) History collection:** Age, duration of marriage, history ofprevious marriage is to be noted.

**A general medical history** should be taken withspecial reference to tuberculosis, sexually transmitted disease, pelvic inflammation or diabetes.

**The surgical history** should be directed especiallytowards abdominal or pelvic surgery. This may be related to peri tubal adhesions.

**Menstrual history** should be taken in details.Wide spectrum of abnormalities ranging from hypomenorrhea—oligomenorrhea to amenorrhea are associated with disturbed hypothalamopituitary ovarian axis which may be either primary or secondary to adrenal or thyroid dysfunction.

**Previous obstetric history—**It is includingnumber of pregnancies, the interval between them and pregnancy related complications are to be enquired. In the case of secondary infertility, the obstetric history is important. The history of puerperal sepsis may be responsible for ascending infection and tubal damage. Uterine synechiae may be due to vigorous curettage.

**Contraceptive practice** should be elicited. IUCDuse may cause PID.

**Sexual problems** such as dyspareunia, and loss oflibido are to be enquired. It should be born in mind that the female orgasm is not essential for fertility and loss of semen from the vaginal orifice following coitus is normal.

**B) Examinations**

General, systemic and gynecological examinations are made to detect any abnormality which may hinder fertility.

**General examination** must be thorough—specialemphasis being given to obesity or marked reduction in weight (BMI).

Physical features pertaining to endocrinopathies (hypo or hyperthyroidism) are carefully evaluated to detect features of PCOS (polycystic ovarian syndrome-menstrual irregularity, excess hair growth, acne and obesity) and galactorrhea (excessive milk production).

**Systemic examination** may accidentally detectsuch abnormalities like hypertension, organic heart disease, chronic renal lesion, thyroid dysfunction, and other endocrinopathies.

**Gynecological examination** includes adequacyof hymenal opening, evidences of vaginal infections, cervical tear or chronic infection, undue elongation of the cervix, uterine size, position and mobility, presence of unilateral or bilateral adnexal masses —fixed or mobile with or without tenderness and presence of nodules in the pouch of Douglas.

**Speculum examination** may reveal abnormalcervical discharge. The discharge is to be collected for Gram stain and culture. Cervical smear is taken as a screening procedure as a routine or in suspected cases.

**Special Investigations—Guidelines:**

In the presence of major fault in **male** such as azoospermia due to testicular destruction or intersex, there is very little scope to proceed for investigation for the female partner. However, considering the place of Assisted Reproductive Technology (ART) female investigation may not be withheld

Similarly, when a major defect is detected in **female** such as Mullerian agenesis (vaginal agencies, is a congenital malformation characterized by a failure of the Mullerian duct to develop, resulting in a missing uterus and vaginal hypoplasia of its upper portion) or intersex, infertility investigations should be suspended. However, correctable abnormality should be rectified first prior to investigation, e.g. narrow vaginal introitus, overt hypothyroidism or diabetes mellitus.

Noninvasive or minimal invasive methods are to be employed prior to major invasive one However, it is not uncommon to have pregnancy soon after the first visit.

Detection of abnormality of one factor does not negate investigation for another defect elsewhere. Multiple defects may be present in the same case, e.g. tubal defects may be associated with anovulation.

Pregnancy following laparoscopy and dye test or hysterosalpingography is not uncommon. It is presumed that small flimsy adhesions or any mucus plug obstructing the tubal lumen is removed during such procedures. The cervical spasm may be relieved during dilatation

**Ovarian factors:** Ovarian dysfunctions commonly associated with infertility are:

* + Anovulation or oligo-ovulation (infrequent ovulation).
  + Luteal phase defect (LPD).
  + Luteinized unruptured follicle (LUF).
  + **DIAGNOSIS OF OVULATION**

The various methods used in practice to detect ovulation are grouped as follows

**• Indirect • Direct • Conclusive**

**Indirect**

Menstrual history

Evaluation of peripheral or end organ changes

* BBT
* Cervical mucus study
* Vaginal cytology
* Hormone estimation -Serum progesterone
* Serum LH
* Serum estradiol
* Urine LH
* Endometrial biopsy
* Sonography (TVS)

**Direct**

* Laparoscopy

**CONCLUSIVE**

* Pregnancy

**INDIRECT**

The indirect or presumptive evidences of ovulation are commonly used in clinical practice.

1. Menstrual history

2. Evaluation of peripheral or end organ changes due to estrogen and progesterone.

3. Direct assays of gonadotropins or steroid hormones preceding, coinciding or succeeding the ovulatory process.

**1. Menstrual History**

The following features in relation to menstruation are strong evidences of ovulation.

* Regular normal menstrual loss between the age of 20–35.
* Mid menstrual bleeding (spotting) or pain or excessive mucoid vaginal discharge (Mittel­ schmerz syndrome).
* Features suggestive of premenstrual syndrome or primary dysmenorrhea

**2. Evaluation of Peripheral or End organ Changes**

**Basal body temperature (BBT)**

**Observation:** **There is “biphasic pattern” of** **temperature variation in ovulatory cycle**. Ifpregnancy occurs, the rise of temperature sustains along with absence of the period. In anovulatory cycle, there is no rise of temperature throughout the cycle.

**Principle:** The rise of temperature is secondary to rise in progesterone output following ovulation. Progesterone is thermogenic. The primary reason for the rise is the increase in the production and secretion of norepinephrine which is also thermogenic.

**Procedures:** The patient is instructed to take her oral temperature **daily on waking in the morning before** **rising out of the bed.** The temperature is recordedon a special chart. Days when intercourse takes place should also be noted on the chart for better evaluation of coital frequency

**Interpretation:** The body temperature maintaining throughout the first half of the cycle is raised to 0.5° to 1°F (0.2°–0.5°C) following ovulation. The rise sustains throughout the second half of the cycle and falls about 2 days prior to the next period–called 'biphasic pattern”). There may be a drop in the temperature to about 0.5°F before the rise and almost coincides with either LH surge or ovulation. The demonstrable rise actually occurs about 2 days after the LH peak and with a peripheral level of progesterone greater than 5 ng/ml.

**Clinical importance**: Maintenance of BBT chart during investigation is of help in determining ovulation and timing of post-coital test, endometrial biopsy, cervical mucus or vaginal cytology study for ovulation. It also helps the couple to determine the most fertile period, if the cycle is irregular.

**How long to keep the record?**

It should not be continued for more than 3–4 months for investigation purpose. However, it has to be maintained for longer periods during management of ovulation induction. **Cervical mucus study:** Alteration of the physicochemical properties of the cervical mucus occurs due to the effect of estrogen and progesterone.

Disappearance of fern pattern beyond 22nd day of the cycle, which was present in the midcycle is suggestive of ovulation. Persistence of fern pattern even beyond 22nd day suggests anovulation.

**Vaginal cytology:** Maturation index shifts to the left from the midcycle to the mid second half of cycle due to the effect of progesterone However, a single smear on day 25 or 26 of the cycle reveals features of progesterone effect, if ovulation occurs.

**Hormone estimation**

**Serum progesterone:** Estimation of serumprogesterone is done on day 8 and 21 of a cycle (28 days). An increase in value from less than 1 ng/ ml to greater than 6 ng/ml suggests ovulation.

**Serum LH:** Daily estimation of serum LH atmid cycle can detect the LH increased. Ovulation occurs about 34–36 hours after beginning of the LH increased. It coincides about 10–12 hours after the LH **Serum estradiol** attains the peak riseapproximately 24 hours prior to LH surge and about 24–36 hours prior to ovulation.

The serum LH and estradiol estimation are used for in vitro fertilization.

**Urinary LH**

LH kits are available to detect midcycle LH surge.

**Ovulation usually occurs within 14–26 hours of detection of urine LH surge and almost always within 48 hours.** (The test should be done on a dailybasis. It is started 2–3 days before the expected surge depending upon the cycle length).

**Endometrial biopsy:** Endometrial tissues to detect ovulation (endometrial sampling) can easily be obtained as an outpatient procedure using instruments such as Sharman curette or Pipelle endometrial sampler. Dilatation and curettage are, however reserved in cases where bulk endometrial study is required as in endometrial tuberculosis.

**When to do? Biopsy is to be done on 21st–23rd day of the cycle.** Barrier contraceptive should beprescribed during the cycle to prevent accidental conception. However, if the cycle is irregular, it is done within 24 hours of the period.

Findings: Evidences of secretory activity of the endometrial glands in the second half of the cycle give not only the diagnosis of ovulation but can predict the functional integrity of the corpus luteum.

**Subnuclear vacuolation is the earliest evidence** appearing 36–48 hours following ovulation.

Cause: The secretory changes are due to the action of progesterone on the estrogen primed endometrium

**Sonography:** Serial sonography (TVS) duringmid cycle can precisely measure the Graafian follicle just prior to ovulation (18–20 mm). It is particularly helpful for confirmation of ovulation following ovulation induction, artificial insemination, and in vitro fertilization. **The features of recent ovulation** **are** collapsed follicle and fluid in the pouch ofDouglas.

**DIRECT:** aspirated peritoneal fluid from the pouch of Douglas is the only direct evidence of ovulation.

**CONCLUSIVE:** Pregnancy is the surest evidence of ovulation.

**Role of nurse in infertility.**

* Give psychological support throughout the counselling.
* The collect other information about various prior test reports and documents.
* The nurse has to encourage the family and patient to ask questions about conditions and diseases etc.
* Provide all explanation about questions.
* Maintain privacy and confidentiality of all cases.
* Ensure follow up and supportive services to individual and family during counseling.
* Explains about the infertility management and specific treatment.
* Performing inseminations.
* Performing embryo transfers.
* Advice about maintain ideal coital frequency. (3-4 times/week in fertile period).
* Explain avoid lubricants, spermicidal jellies and creams.
* Avoid fertility impairing medications.
* Maintenance of body weight
* Helps the couple in overcoming the dilemmas and deciding the right fertility treatment.
* The nurse provide care for the individuals and couples before, during, and after infertility treatment.

**SUMMARY**

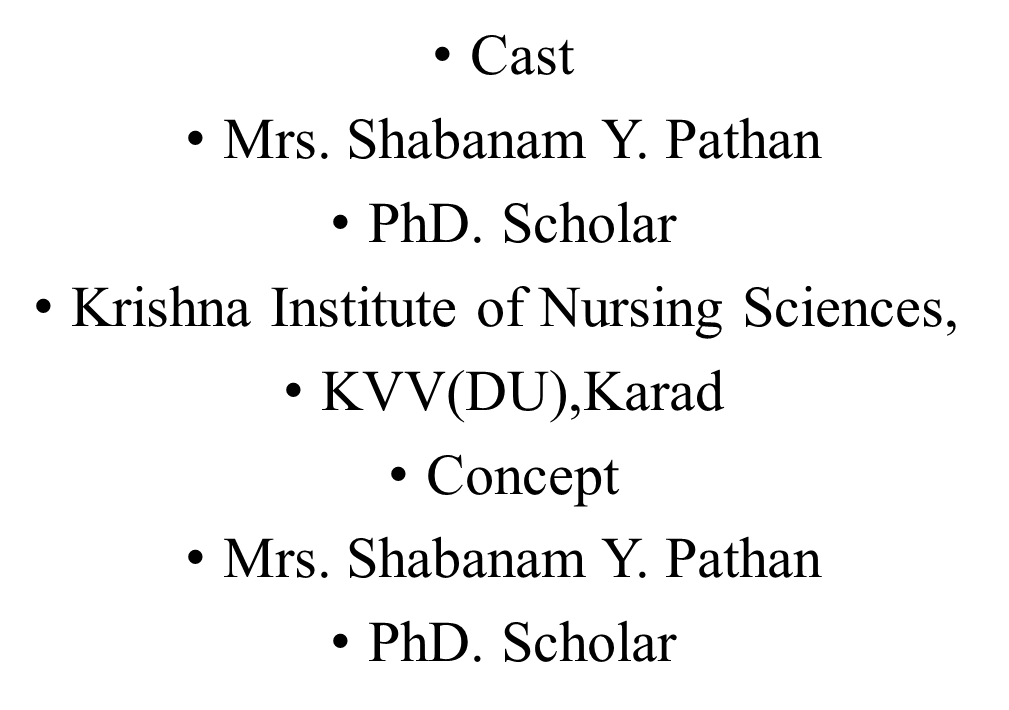
* Infertility is a sensitive issue that should be handed with great care with continuous professional counseling.
* Psychological support is important.
* Most young couples will conceive naturally within two years.
* Evaluation of both partners for causes is essential
* Treatment depends on the cause and varies from medical treatment to surgery to ART.

**CONCLUSION**

* Infertility is a problem of couples.
* Psychological support is important.
* Give the couple step by step from basic evaluation to surrogacy.
* Infertility should be evaluated after one year of unprotected intercourse.
* History and physical examination usually will help to identify the etiology.
* If patients fail the initial therapies then the proper referral should be made to a reproductive specialist.

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