INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI

With partial fulfillment of the regulations for the award of the degree of

M.S (Obstetrics and Gynecology) Branch-I

Government Stanley Medical College,Chennai- 1

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **'INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY**at Government Stanley Medical College Hospital is a bonafide work of Dr.J.PRIYADHARSHINI submitted to The Tamilnadu Dr.M.G.R Medical University in partial fulfillment of requirementsfor the award of the degree of M.S. BRANCH I (OBSTETRICS AND GYNECOLOGY) examination to be held in MAY, 2021.

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled **"INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY."** at Govt. Stanley Medical College Hospital is a bonafide and genuine research work carried out by me in the Department of Obstetrics and Gynecology, Government Stanley Medical and Hospital, Chennai-1, under the guidance of our Chief **Prof.Dr. SHANTHI K ELANGO MD(OG),** Government Stanley Medical College and Hospital.

This dissertation is submitted to **THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI** in partial fulfillment of the University regulations for the award of M.S degree (Obstetrics and Gynecology) Branch I, examination to be held in MAY 2021.

Date:

Place: Chennai **Dr. J.PRIYADHARSHINI**

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation titled **"INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY."** done in the Department of Obstetrics and Gynecology at Govt. Stanley Medical College Hospital is a bonafide research work done by Dr.J.PRIYADHARSHINI, a post graduate in M.S. Obstetrics and Gynecology , Government Stanley Medical College & Hospital, Chennai-1 undermy direct guidance and supervision in my satisfaction and in partial fulfillment of the requirements for the degree of **M.S. Obstetrics and Gynecology**

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The most important part of any medical research are the patients. I owe a great dealto each and every one of them.

I would like to thank God for all that he has bestowed upon me.

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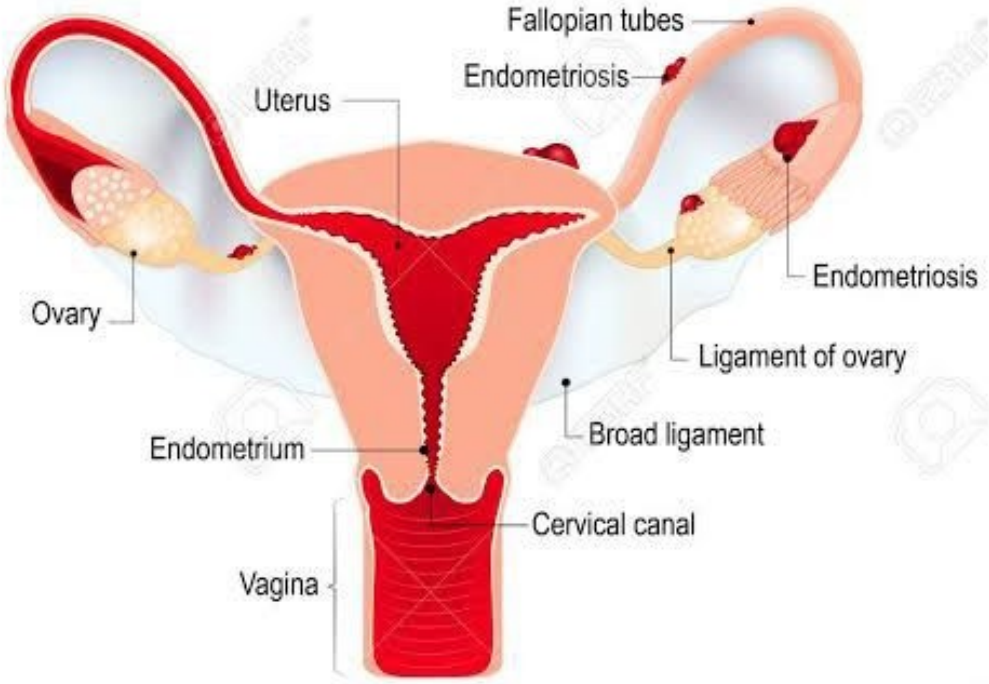
ABSTRACT

Endometriosis is an estrogen-dependent benign inflammatory disease characterized by the presence of ectopic endometrial implants typically occur in the pelvis It is associated with infertility,chronic pelvic pain leading to significant morbidity.The objective of this study is to find out the incidence of endometriosis in the female population in reproductive age group. A total of 280 females with either primary or secondary infertility were studied out of which 29 cases where diagnosed with endometriosis, which accounts to about 10% which is significantly higher than fertile population.

INTRODUCTION

The association between endometriosis and infertility is well supported throughout the literature, but a definite cause-effect relationship is still controversial. The aim of this study is to figure out the incidence of endometriosis in women with infertility. Diagnosis of endometriosis is mainly clinical. Definitive diagnosis is based on laparoscopic findings confirmed by histopathology. Typical powder burn lesions on uterine serosa, round ligament, endometrioma or filmy adhesions. Early diagnosis and timely intervention is essential in enometriosis as the disease progresses in 30 to 60% cases if left untreated causing significant morbidity.

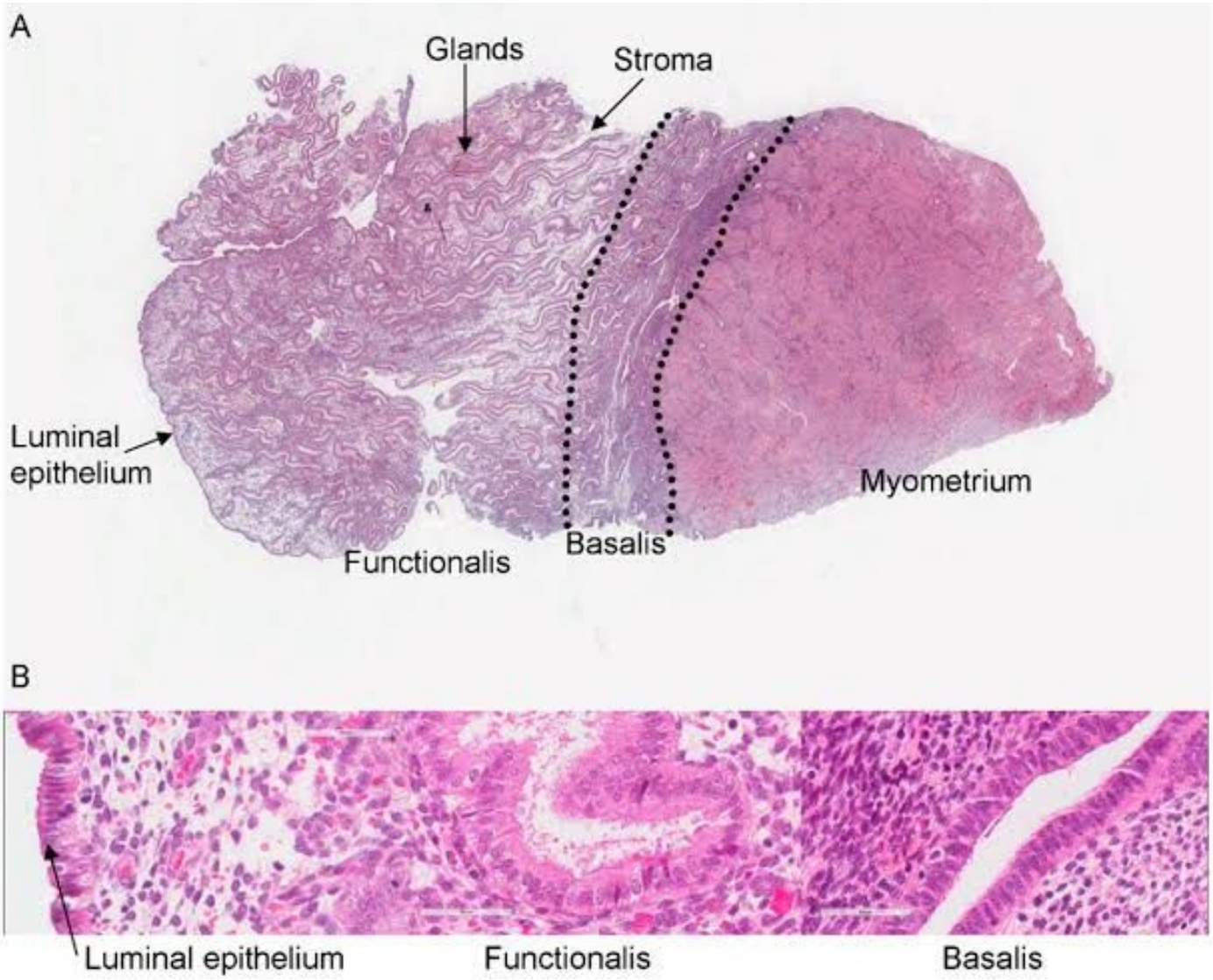
ENDOMETRIOSIS



UTERINE ENDOMETRIUM- ANATOMY AND PHYSIOLOGY

Endometrium consists of two layers. Superficial two-thirds of the endometrium is the zone that proliferates and is ultimately shed with each cycle if pregnancy does not occur. This cycling portion of the endometrium is known as the decidua functionalis and is composed of a deeply situated intermediate zone (stratum spongiosum) and a superficial compact zone (stratum compactum). The decidua basalis is the deepest region of the endometrium. It does not undergo significant monthly proliferation but, instead, is the source of endometrial regeneration after each menses. In the absence of implantation, glandular secretion ceases and an irregular breakdown of the decidua functionalis occurs. The resultant shedding of this layer of the endometrium is termed menses. The destruction of the corpus luteum and its production of estrogen and progesterone is the presumed cause of the shedding. The endometrium functions as a lining for the uterus, preventing adhesions between the opposed walls of the myometrium, thereby maintaining the patency of the uterine cavity.Endometriosis is defined as the presence of this endometrial tissue (glands and stroma) outside the uterus resulting in chronic pelvic pain, infertility and other morbidities.

HISTOLOGY OF NORMAL UTERINE ENDOMETRIUM



REVIEW OF LITERATURE

EPIDEMIOLOGY

Endometriosis is found predominantly in women of reproductive age but is reported in adolescents and in postmenopausal women receiving hormonal replacement.Estimates of the frequency of endometriosis vary widely, but the prevalence of the condition is assumed to be around 10%. In women with pelvic pain or infertility, a high prevalence of endometriosis (from a low of 20% to a high of 90%) is reported. In women with unexplained subfertility with or without pain (regular cycle, partner with normal sperm), the prevalence of endometriosis is reported to be as high as 50%. In asymptomatic women undergoing tubal ligation (women of proven fertility), the prevalence of endometriosis ranges from 3% to 43%.

RISK FACTORS

The following are identified risk factors for endometriosis: infertility, red hair, early age at menarche, shorter menstrual cycle length, hypermenorrhea, nulliparity, müllerian anomalies, birth weight (less than 7 pounds), one of multiple fetal gestation, diethylstilbestrol (DES) exposure, endometriosis in first-degree relative, tall height, dioxin or polychlorinated biphenyls (PCB) exposure, a diet high in fat and red meat, and prior surgeries or medical therapy for endometriosis. Prior use of contraception or intrauterine device (IUD), or smoking is not associated with increased risk of endometriosis

PROTECTIVE FACTORS

Protective factors against the development of endometriosis include multiparity, lactation, tobacco exposure in utero, increased body mass index, increased waist-to-hip ratios and exercise, and diet high in vegetables and fruits.

ETIOLOGY

Although signs and symptoms of endometriosis were described since the 1800s, its widespread occurrence was acknowledged only during the 20th century. Endometriosis is an estrogen-dependent disease. Some theories were proposed to explain the histogenesis of endometriosis:

THEORIES PROPOSED TO EXPLAI HISTOGENESIS OF ENDOMETRIOSIS

1. The transplantation theory/ Sampson theory assumes that it is due to seeding or implantation of endometrial cells by transtubal regurgitation during menstruation
2. Coelomic metaplasia
3. Induction theory states that an endogenous biochemical factor can induce undifferentiated peritoneal cells to develop into endometrial tissue.
4. Genetic factors
5. Immunologic factors and inflammation
6. Environmental factors

No single theory can account for the location of endometriosis in all cases.

TRANSPLANTATION THEORY

Retrograde menstruation occurs in 70% to 90% of women, and it may be more common in women with endometriosis than in those without the disease. The presence of endometrial cells in the peritoneal fluid, indicating retrograde menstruation, is reported in 59% to 79% of women during menses or in the early follicular phase, and these cells can be cultured in vitro. Endometriosis is most often found in dependent portions of the pelvis—the ovaries, the anterior and posterior cul-de-sac, the uterosacral ligaments, the posterior uterus, and the posterior broad ligaments. The menstrual reflux theory combined with the clockwise peritoneal fluid current explains why endometriosis is predominantly located on the left side of the pelvis (refluxed endometrial cells implant more easily in the rectosigmoidal area) and why diaphragmatic endometriosis is found more frequently on the right side (refluxed endometrial cells implant there by the falciform ligament).Ovarian endometriosis may be caused by either retrograde menstruation or by lymphatic flow from the uterus to the ovary; metaplasia and bleeding from a corpus luteum may be a critical event in the development of some endometriomas.

COELOMIC METAPLASIA

The transformation (metaplasia) of coelomic epithelium into endometrial tissue is a proposed mechanism for the origin of endometriosis.

INDUCTION THEORY

The induction theory is an extension of the coelomic metaplasia theory. It proposes that an endogenous (undefined) biochemical factor can induce undifferentiated peritoneal cells to develop into endometrial tissue. This theory is supported by experiments in rabbits but is not substantiated in women or nonhuman primates

GENETIC FACTORS

Increasing evidence suggests that endometriosis is partially a genetic disease. Recent findings that support this association include evidence of familial clustering in humans and in Rhesus monkeys, a founder effect detected in the Icelandic population, concordance in monozygotic twins, a similar age at onset of symptoms in affected non twin sisters, a six- to nine-times increased prevalence of endometriosis among first-degree relatives of women compared with the general population, and a 15% prevalence of magnetic resonance imaging (MRI) findings suggestive of endometriosis in the first-degree relatives of women with stage III or IV disease based on the classification of the American Society of Reproductive Medicine . The induction of humanlike endometriosis by genetic activation of an oncogenic K-ras allele lends further support to the genetic basis of this disorder.

GENETIC POLYMORPHISMS AND ENDOMETRIOSIS

A number of studies investigated genetic polymorphisms as a possible factor contributing to the development of endometriosis. About 50% of the studies one review demonstrated positive correlations between different polymorphisms and endometriosis. This relation was seen most clearly in groups 1 (cytokines and inflammation), 2 (steroid-synthesizing enzymes and detoxifying enzymes and receptors), 4 (estradiol metabolism), 5 (other enzymes and metabolic systems), and 7 (adhesion molecules and matrix enzymes). Group 8 (apoptosis, cell-cycle regulation, and oncogenes) seemed to be negatively correlated with the disease, whereas groups 3 (hormone receptors), 6 (growth factor systems), and especially 9 (human leukocyte antigen system components) showed a relatively strong correlation. As many results were contradictory, the review concluded that genetic polymorphisms might have a limited value in assessing possible development of endometriosis. Future studies should include large numbers of women with laparoscopically and histologically confirmed endometriosis and women with a laparoscopically confirmed normal pelvis as controls, taking into account ethnic variability.

ANEUPLOIDY

Epithelial cells of endometriotic cysts are monoclonal on the basis of phosphoglycerate kinase gene methylation, and normal endometrial glands are monoclonal. In a comparison of endometriotic tissue with eutopic endometrium, flow cytometric DNA analysis failed to show aneuploidy. Studies using comparative genomic hybridization, or multicolor in situ hybridization, showed aneuploidy for chromosomes 11, 16, and 17, increased heterogeneity of chromosome 17 aneuploidy, and losses of 1p and 22q (50%), 5p (33%), 6q (27%), 70 (22%), 9q (22%), and 16 (22%) of 18 selected endometriotic tissues. In another study, trisomies 1 and 7, and monosomies 9 and 17 were found in endometriosis, ovarian endometrioid adenocarcinoma, and normal endometrium. The proportions of aneusomic cells were significantly higher in ovarian endometriosis compared with extragonadal endometriosis and normal endometrium ($p < 0.001$), suggestinga role of the ovarian stromal milieu in the induction of genetic changes, which may lead to invasive cancer in isolated cases.

Microsatellite DNA assays reveal an allelic imbalance (loss of heterozygosity) in p16 (Ink4), GALT, p53, and APOA2 loci in patients with endometriosis and in stage II of endometriosis. Another report found a loss of heterozygosity in 28% of endometriotic lesions at one or more sites.

IMMUNOLOGIC FACTORS AND INFLAMMATION

Although retrograde menstruation appears to be a common event in women, not all women who have retrograde menstruation develop endometriosis. The immune system may be altered in women with endometriosis, and it is hypothesized that the disease may develop as a result of reduced immunologic clearance of viable endometrial cells from the pelvic cavity. Endometriosis can be caused by decreased clearance of peritoneal fluid endometrial cells resulting from reduced natural killer (NK) cell activity or decreased macrophage activity. Substantial evidence suggests that endometriosis is associated with a state of subclinical peritoneal inflammation, marked by an increased peritoneal fluid volume, increased peritoneal fluid white blood cell concentration (especially macrophages with increased activation status), and increased inflammatory cytokines, growth factors, and angiogenesis-promoting substances. There is increasing evidence that local inflammation and secretion of prostaglandins (PG) is related to differences in endometrial aromatase activity between women with and without endometriosis. A complex network of humoral and cellular immunity factors modulates the growth and inflammatory behavior of ectopic endometrial implants and affects embryo implantation. Women with endometriosis have an increased volume of peritoneal fluid with a high concentration of activated macrophages, prostaglandins, IL-1, TNF, and proteases.

These alterations may have adverse effects on the function of the oocyte, sperm, embryo, or fallopian tube. Moreover, an ovum capture inhibitor (OCI) in endometriosis peritoneal fluid is thought to be responsible

for fimbrial failure of ovum capture.Elevated levels of IgG and IgA antibodies (autoantibodies to endometrial antigens) and lymphocytes may be found in the endometrium of women with endometriosis. These abnormalities may alter endometrial receptivity and embryo implantation.Some authors have reported that uterine implantation was affected by changes in receptivity in endometriosis. Delayed histologic maturation or biochemical disturbances may lead to endometrial dysfunction. Reduced endometrial expression of the αvβ integrin (a cell adhesion molecule) during the time of implantation has been described in some women with endometriosis. Recently, some women with endometriosis exhibited very low levels of an enzyme involved in the synthesis of the endometrial ligand for L-selectin (a protein that coats the trophoblast on the surface of the blastocyst)

ENVIRONMENTAL FACTORS

There is an increasing awareness of potential links between reproductive health, infertility, and environmental pollution. Attention was directed toward the potential role of dioxins in the pathogenesis of endometriosis, but the issue remains controversial. A meta-analysis concluded that there is insufficient evidence in women or in nonhuman primates that endometriosis is caused by dioxin exposure.

ENDOMETRIOSIS AND CANCER

Several publications link endometriosis with an increased risk for certain gynecologic and nongynecologic cancers. These associations are controversial and no data exist to inform clinicians regarding the best management of patients who might be at risk of developing such cancers. Endometriosis should not be considered a medical condition associated with a clinically relevant risk of any specific cancer. Data from large cohort and case-control studies indicate an increased risk of ovarian cancers in women with endometriosis. The observed effect sizes are modest, varying between 1.3 and 1.9. Evidence from clinical series consistently demonstrates that the association is confined to the endometrioid and clear-cell histologic types of ovarian cancer. A causal relationship between endometriosis and these specific histotypes of ovarian cancer should be recognized, but the low magnitude of the risk observed is consistent with the view that ectopic endometrium undergoes malignant transformation with a frequency similar to its eutopic counterpart. Evidence for an association with melanoma and non-Hodgkin's lymphoma is increasing but needs to be verified, whereas an increased risk of other gynecologic cancer types are not supported.

DIAGNOSIS

CLINICAL PRESESNTATION

Endometriosis should be suspected in women with subfertility, dysmenorrhea, dyspareunia, or chronic pelvic pain, although these symptoms can be associated with other diseases. Endometriosis may be asymptomatic, even in women with more advanced disease, (i.e., ovarian endometriosis or deeply invasive rectovaginal endometriosis). Endometriosis can be associated with significant gastrointestinal symptoms (pain, nausea, vomiting, early satiety, bloating and distention, altered bowel habits.

It may present as

Pain :

In adult women, dysmenorrhea may be especially suggestive of endometriosis if it begins after years of pain-free menses. All endometriosis lesion types are associated with pelvic pain, including minimal to mild endometriosis. Possible mechanisms causing pain in patients with endometriosis include local peritoneal inflammation, deep infiltration with tissue damage, adhesion formation, fibrotic thickening, and collection of shed menstrual blood in endometriotic implants, resulting in painful traction with the physiologic movement of tissues.

Subfertility and spontaneous abortions

Increased prevalence of endometriosis in subfertile women (33%) when compared to women of proven

fertility (4%), a reduced monthly fecundity rate (MFR) in baboons with mild to severe (spontaneous or induced) endometriosis when compared to those with minimal endometriosis or a normal pelvis. When endometriosis is moderate or severe, involving the ovaries and causing adhesions that block tubo-ovarian motility and ovum pickup, it is associated with subfertility.

A possible association between endometriosis and spontaneous abortion was suggested in uncontrolled or retrospective studies. Some controlled studies evaluating the association between endometriosis and spontaneous abortion have important methodologic shortcomings: heterogeneity between cases and controls, analysis of the abortion rate before the diagnosis of endometriosis, and selection bias of study and control groups. Based on controlled prospective studies, there is no evidence that endometriosis is associated with (recurrent) pregnancy loss or that medical or surgical treatment of endometriosis reduces the spontaneous abortion rate. Some data suggest that miscarriage rates may be increased after treatment with assisted reproductive technology.

Endocrinologic Abnormalities

Endometriosis is associated with anovulation, abnormal follicular development with impaired follicle growth, reduced circulating E2 levels during the preovulatory phase, disturbed luteinizing hormone (LH) surge patterns, premenstrual spotting, luteinized unruptured follicle syndrome, and galactorrhea and hyperprolactinemia.

Extrapelvic Endometriosis

Extrapelvic endometriosis, although often asymptomatic, should be suspected when symptoms of pain or a palpable mass occur outside the pelvis in a cyclic pattern. Endometriosis involving the intestinal tract (especially colon and rectum) is the most common site of extrapelvic disease and may cause abdominal and back pain, abdominal distention, cyclic rectal bleeding, constipation, and obstruction. Ureteral involvement can lead to obstruction and result in cyclic pain, dysuria, and hematuria. Pulmonary endometriosis can manifest as pneumothorax, hemothorax, or hemoptysis during menses. Umbilical endometriosis should be suspected when a patient has a palpable mass and cyclic pain in the umbilical area.

CLINICAL EXAMINATION

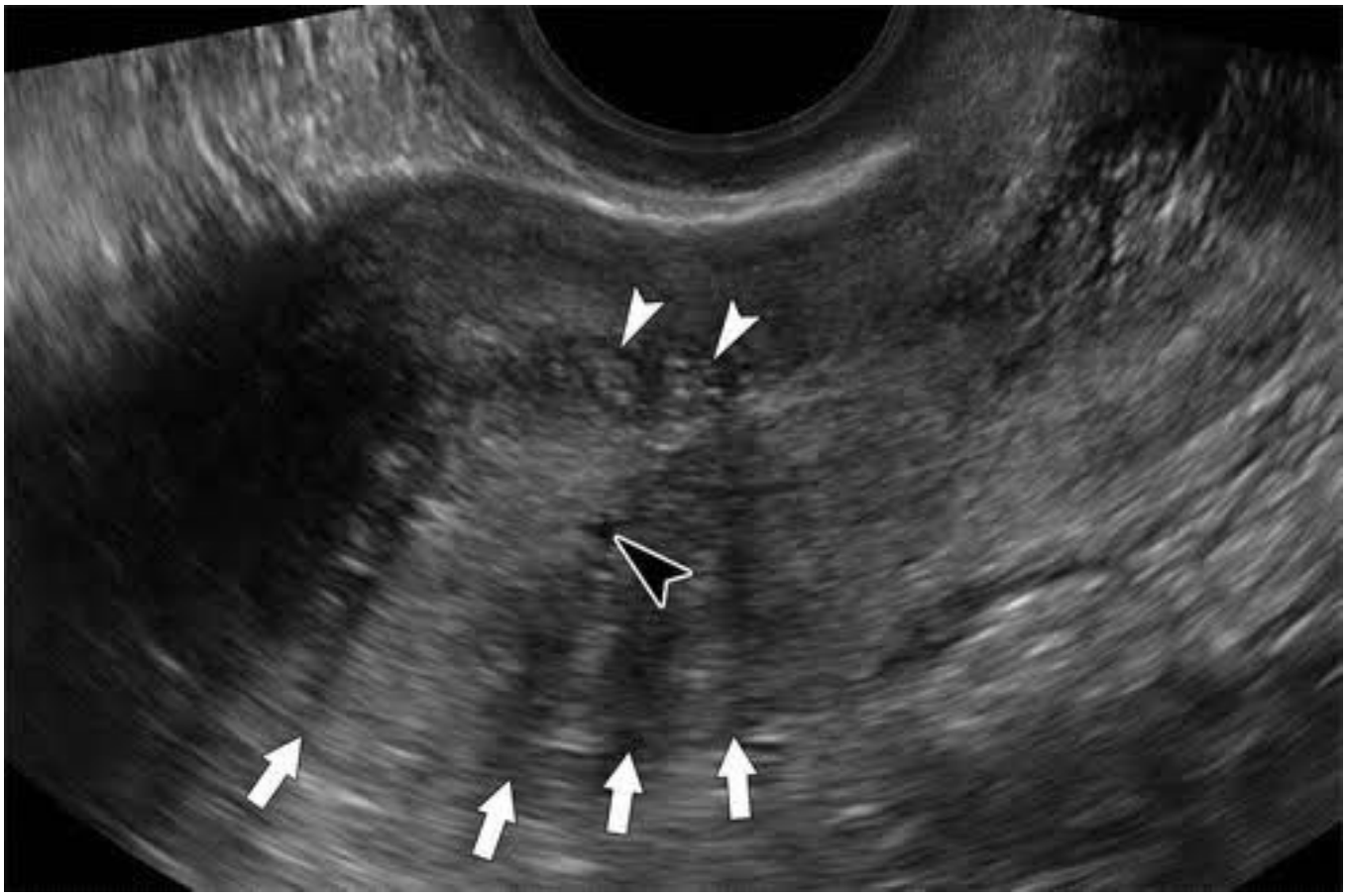
In many women with endometriosis, no abnormality is detected during the clinical examination. However, the vulva, vagina, and cervix should be inspected for any signs of endometriosis, although the occurrence of endometriosis in these areas is rare (e.g., episiotomy scar).Other signs of possible endometriosis include uterosacral or cul-de-sac nodularity, lateral or cervical displacement caused by uterosacral scarring, painful swelling of the rectovaginal septum, and unilateral ovarian cystic enlargement.

The clinical examination may have false-negative results. The diagnosis of endometriosis should be confirmed by visual inspection during laparoscopy and by histological confirmation of endometriosis in biopsied lesions

IMAGING

ULTRASOUND

Peritoneal endometriosis cannot be reliably visualized by imaging techniques. Compared to laparoscopy, transvaginal ultrasound has no value in diagnosing peritoneal endometriosis, but it is useful in making or excluding the diagnosis of an ovarian endometrioma.The typical ultrasound features of an endometriotic ovarian cyst in premenopausal women were described as ground glass echogenicity of the cyst fluid, one to four locules and no solid parts.



Ultrasound showing typical venetian blind appearance in endometriosis

OTHER IMAGING

Other imaging techniques, including computed tomography (CT) and MRI, can be used to provide additional and confirmatory information, but they cannot be used for primary diagnosis

IMAGING TO ASSESS INTESTINAL AND UROLOGY INVOLVEMENT

If there is clinical evidence of deeply infiltrating endometriosis, ureteral, bladder, and bowel involvement should be assessed. Ureteral involvement may be asymptomatic in up to 50% of patients with deeply infiltrative endometriosis. Consideration should be given to performing ultrasound(transrectal, transvaginal or renal), a CT urogram, or an MRI. A barium enema study might be useful, depending on the individual circumstances, to map the extent of disease present, which may be multifocal. There is no proof that one technique is superior to another; it is recommended that the technique that is most familiar to the radiologist involved be used.

CA125

Levels of CA125, a glycoprotein from coelomic epithelium and common to most nonmucinous epithelial ovarian carcinomas. The normal range for CA 125 is 0 to 35 units/ml. The sensitivity and specificity of serum CA-125 for the diagnosis of endometriosis were 61.1% and 87.5% respectively. False positive results have been noted in many medical disorders, both malignant and benign women with moderate or severe endometriosis and normal in women with minimal or mild disease.

Compared with laparoscopy, measurement of serum CA125 levels has no value as a diagnostic tool. The low level of sensitivity of CA125 (20% to 50% in most studies) poses limitations for the clinical use of this

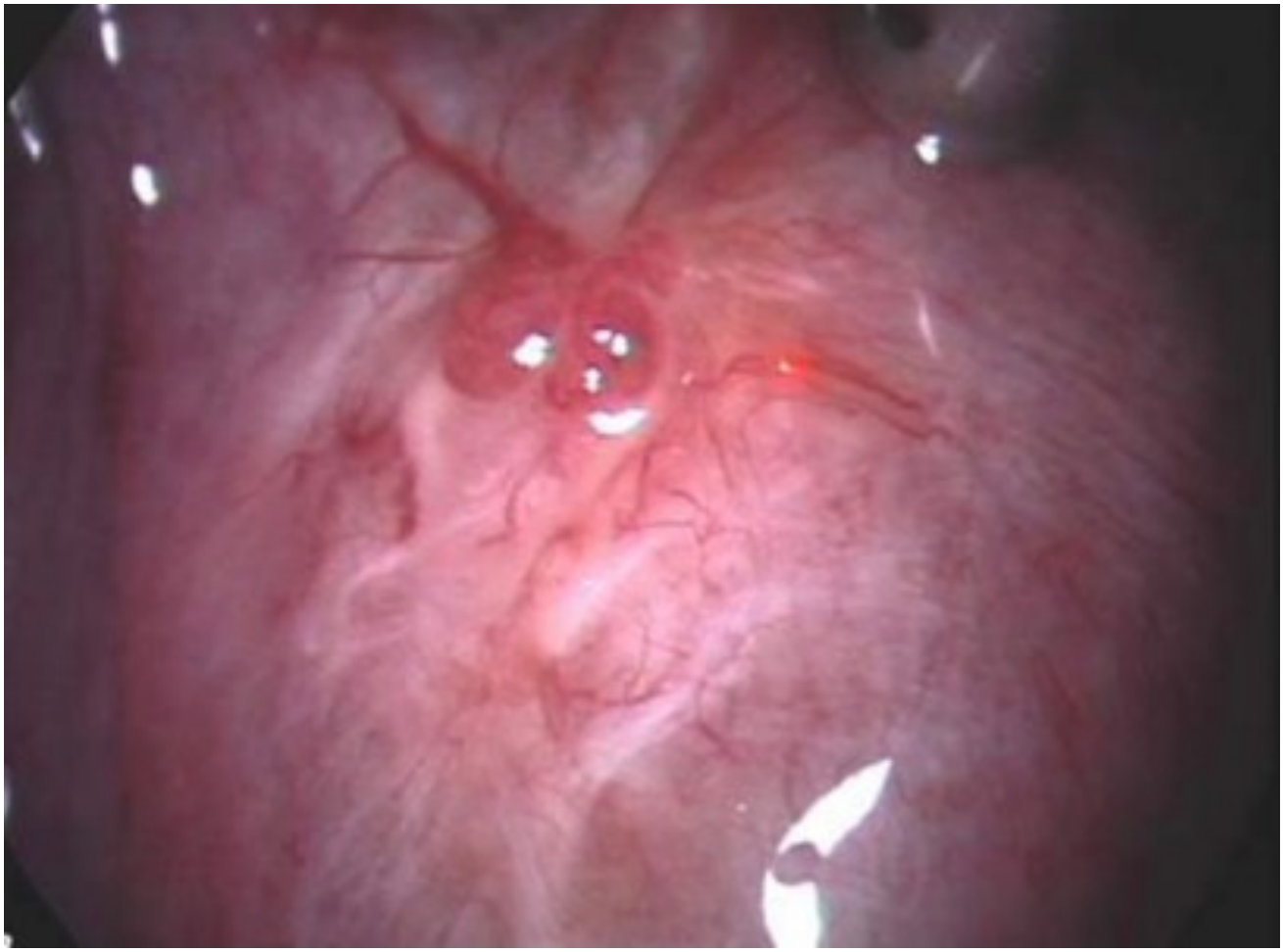
test for diagnosis of endometriosis. Serial CA125 determinations may be useful to predict the recurrence of endometriosis after therapy.

LAPROSCOPY

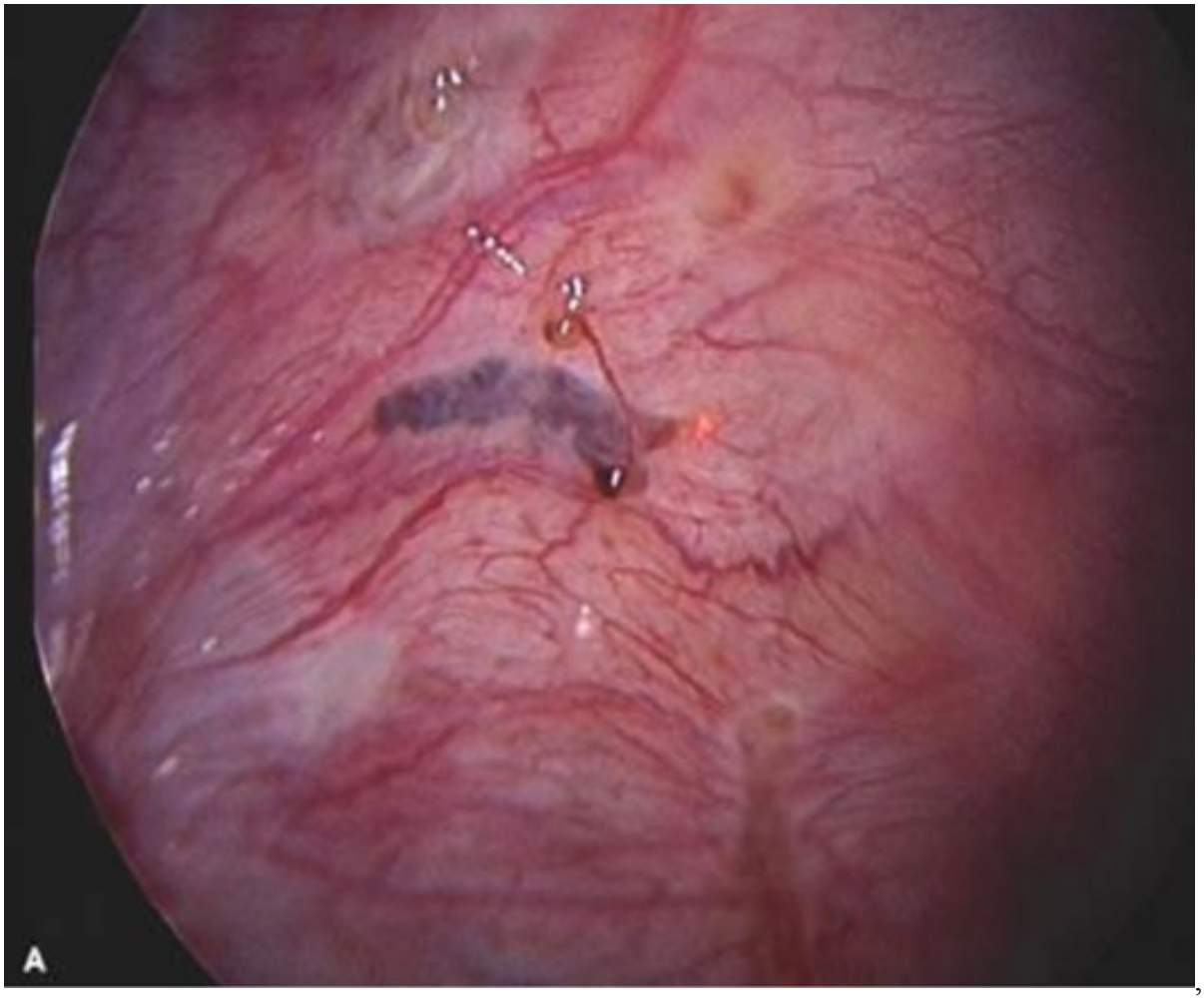
Unless disease is visible in the vagina or elsewhere, laparoscopy is the standard technique for visual inspection of the pelvis and establishment of a definitive diagnosis. During diagnostic laparoscopy, the pelvic and abdominal cavity should be systematically investigated for the presence of endometriosis. This examination should include a complete inspection in a clockwise or counterclockwise fashion with a blunt probe, with palpation of lesions to check for nodularity as a sign of deeply infiltrative endometriosis of the

bowel, bladder, uterus, tubes, ovaries, cul-de-sac, or broad ligament. Characteristic findings include typical (“powder-burn” or “gunshot)” lesions on the serosal surfaces of the peritoneum. These lesions are black, dark brown, or bluish nodules or small cysts containing old hemorrhage surrounded by a variable degree of fibrosis . Endometriosis can appear as subtle lesions, including red implants (petechial, vesicular, polypoid, hemorrhagic, red flamelike), serous or clear vesicles, white plaques or scarring, yellow-brown discoloration of the peritoneum, and subovarian adhesions . Histologic confirmation of the laparoscopic impression is essential for the diagnosis of endometriosis, for subtle lesions, and for the typical lesions reported to be histologically negative in 24% cases. At laparoscopy, deeply infiltrating endometriosis may have the appearance of minimal disease, resulting in an underestimation of disease severity.

Red polypoid lesions with hypervascularisation



A: Typical black puckered lesion with hypervascularisation and orange polypoid vesicles



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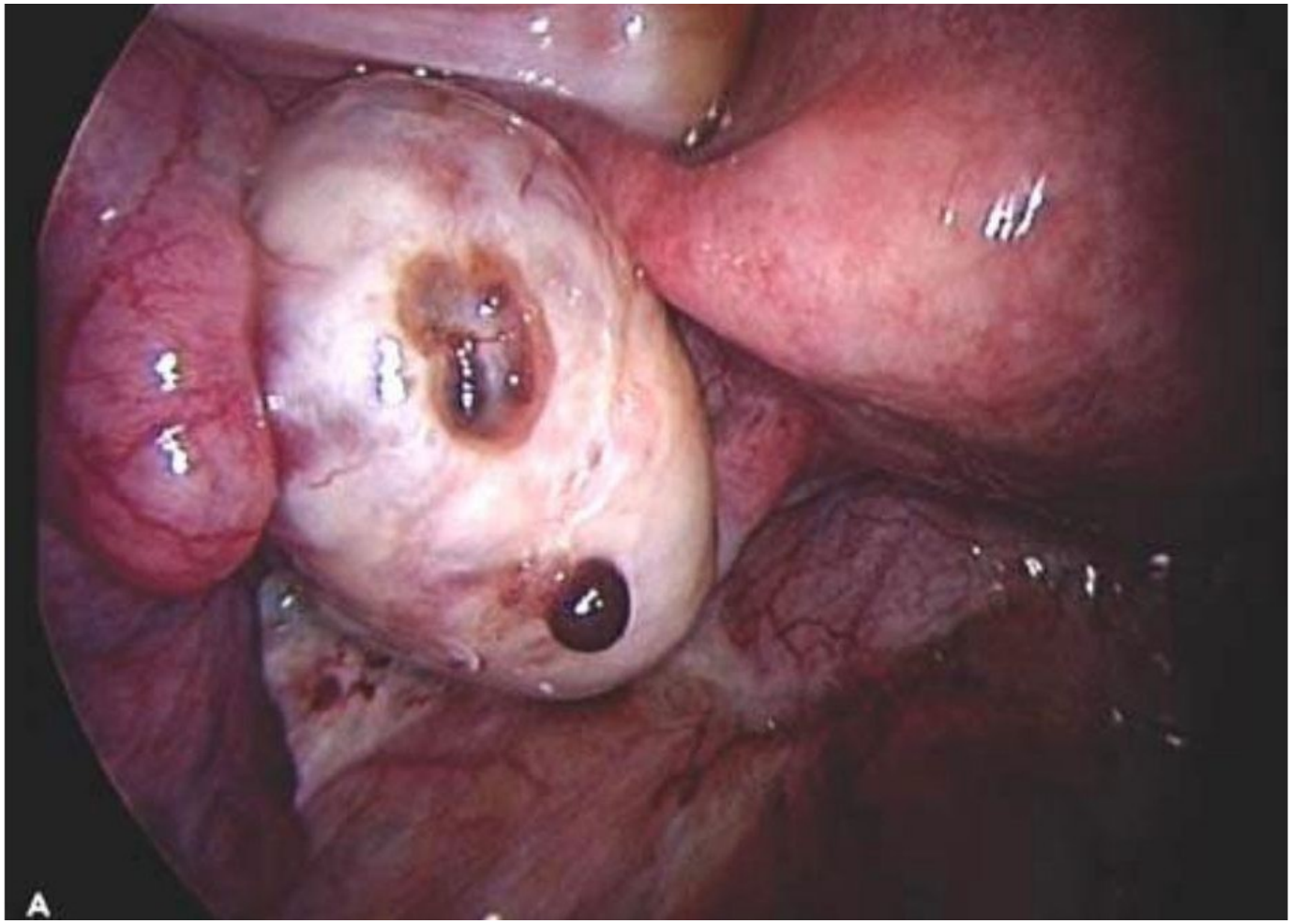
A. Superficial ovarian endometriosis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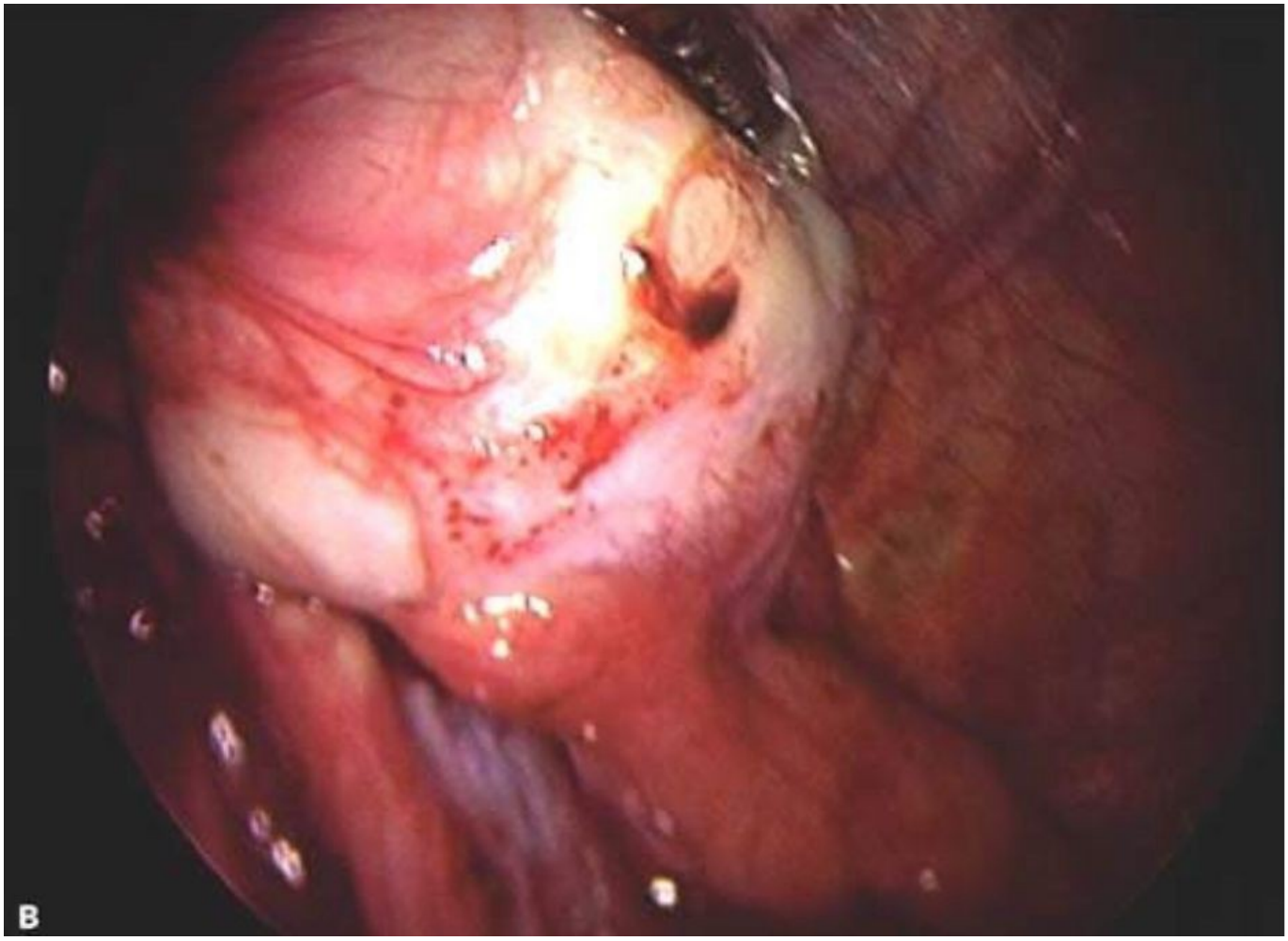
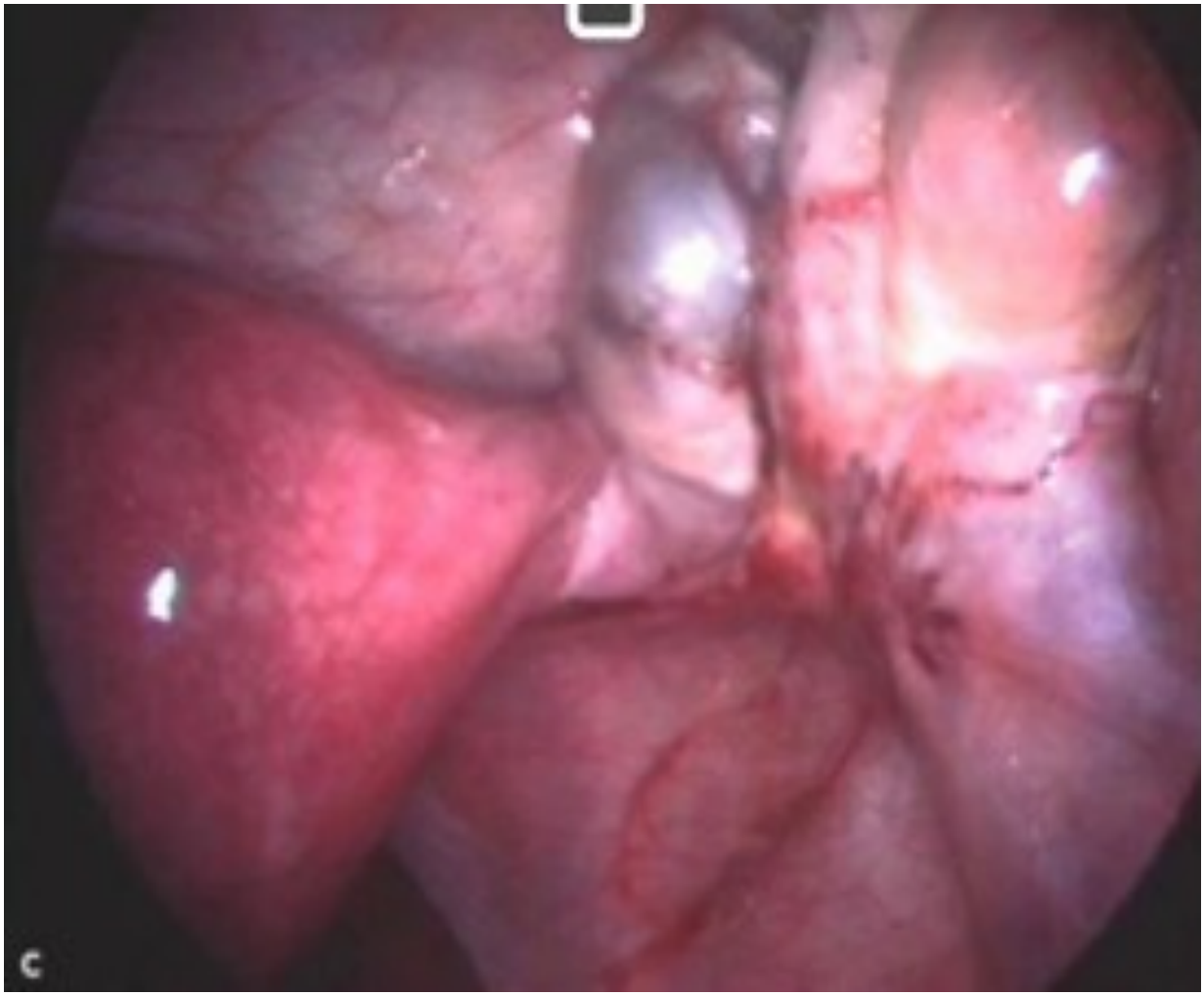
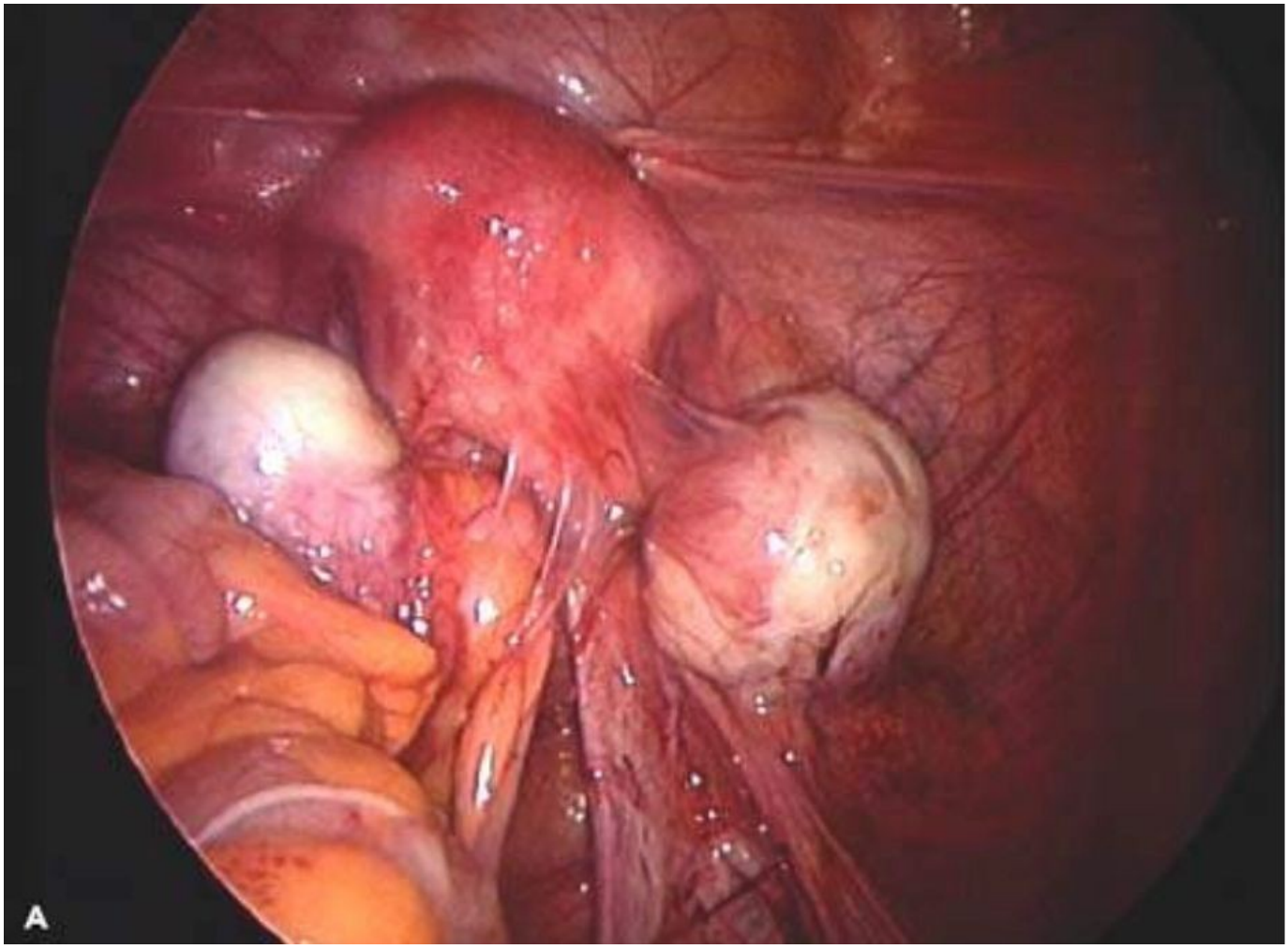


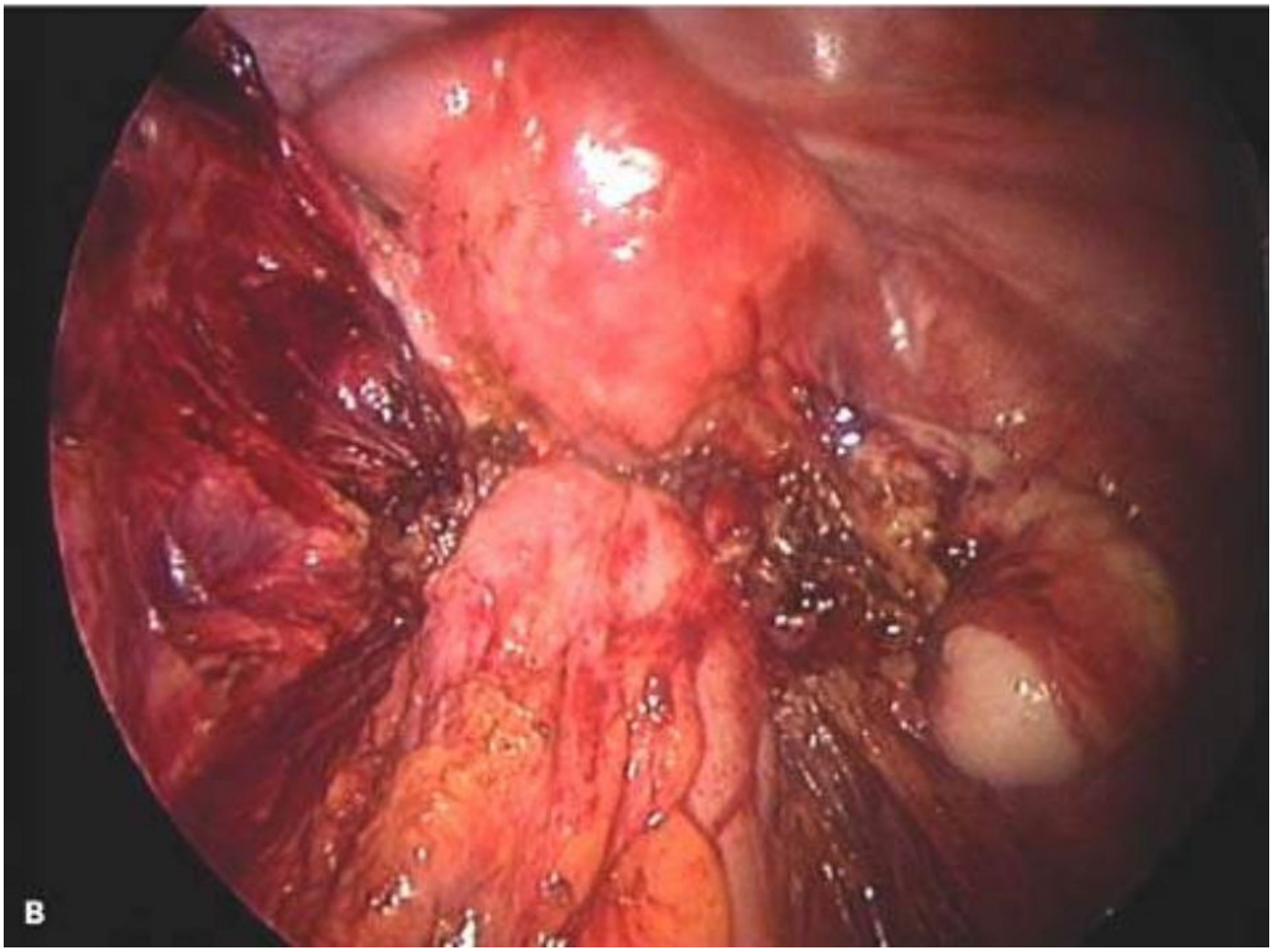
B. superficial ovarian endometriosis and endometrioma



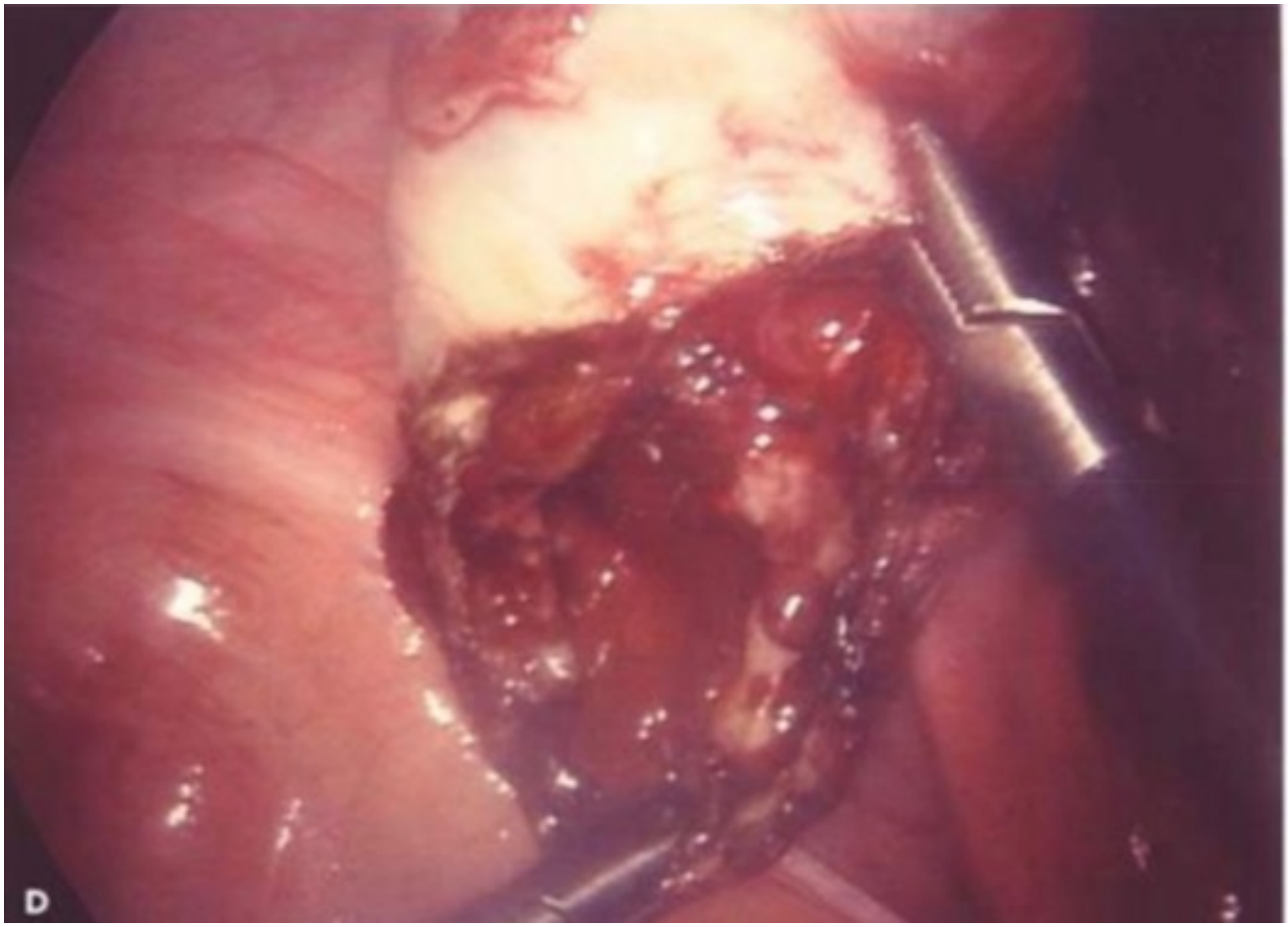
C:Laparoscopic image of uterus and right ovary with dark endometrioma



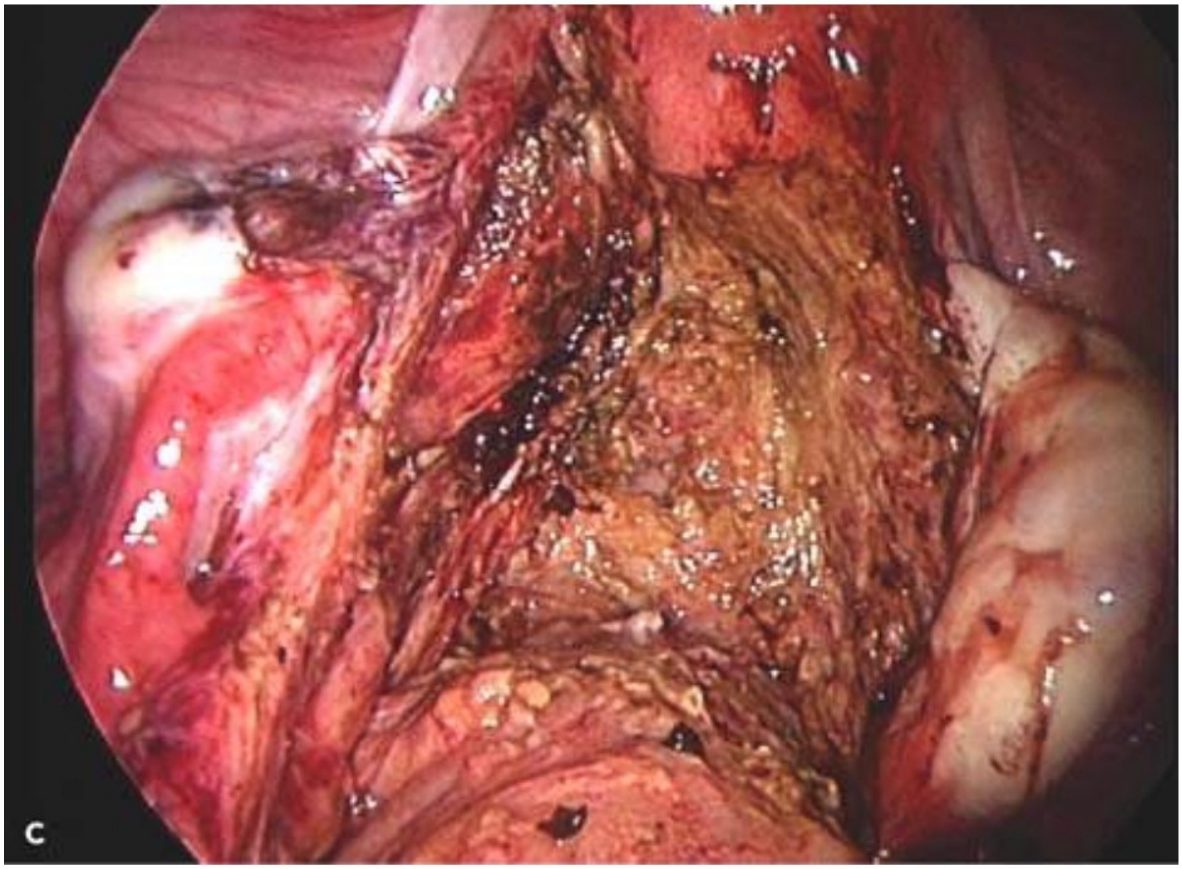
. Extensive endometriosis with deep nodules in right uterosacral ligament, masked by adhesions



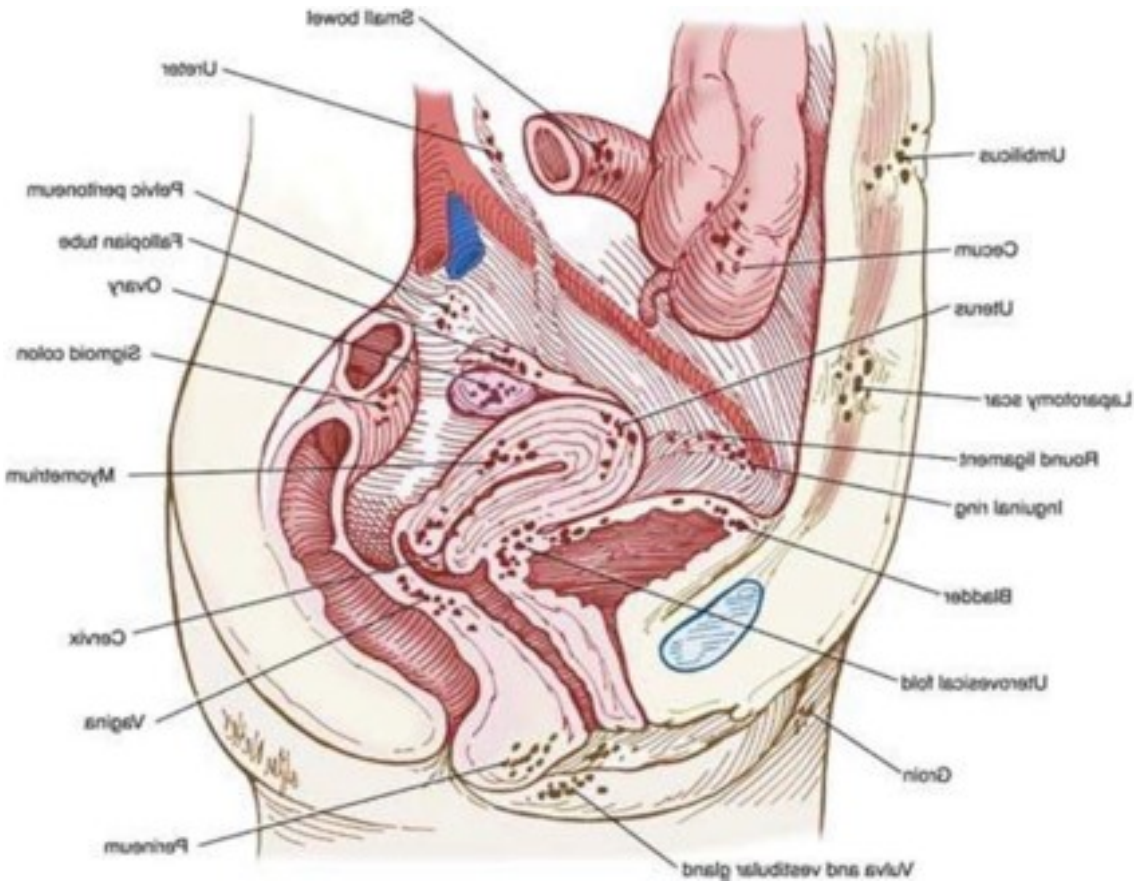
Deep nodule still present in dense adhesion between rectum and uterosacral ligaments



. Ovaraian endometriotic cystectomy



Cul-de-sac after resection of deep nodule with CO2 laser



Pelvic endometriosis

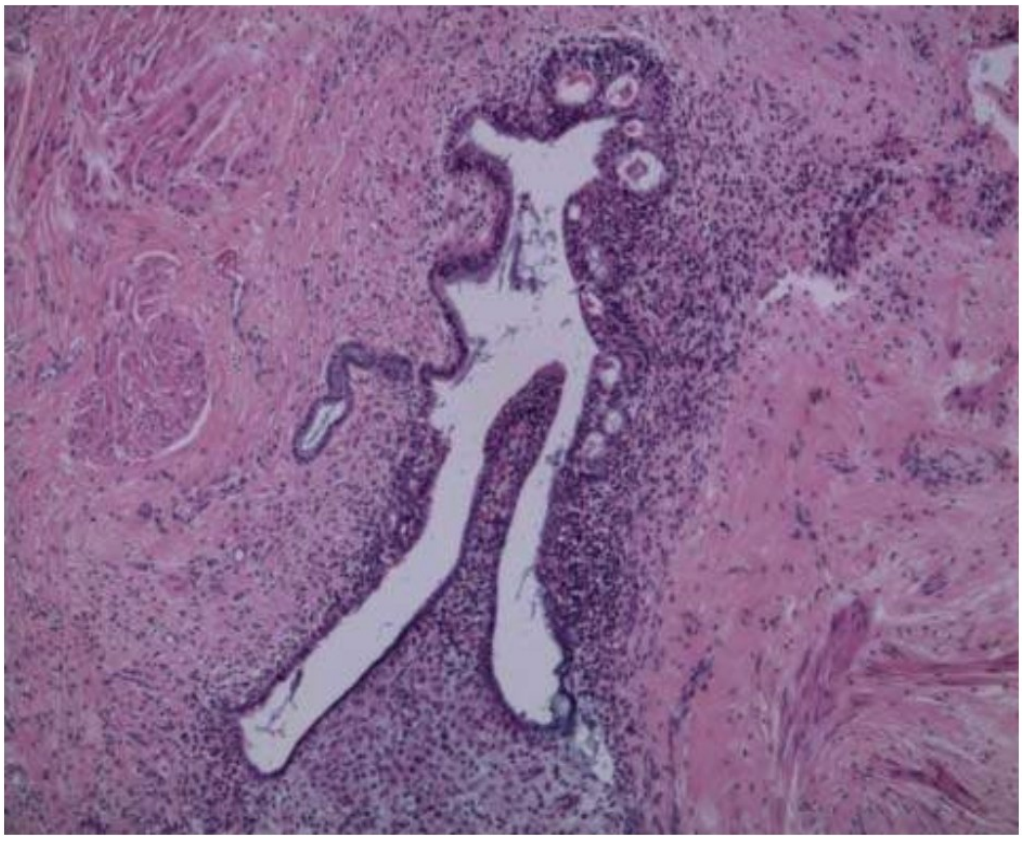
The diagnosis of ovarian endometriosis is facilitated by careful inspection of all sides of both ovaries, which may be difficult when adhesions are present in more advanced stages of disease. The ovarian endometriotic cysts often contain a thick, viscous dark brown fluid (i.e., “chocolate fluid”), composed of hemosiderin derived from previous intraovarian hemorrhage. Because this fluid may be found in other conditions, such as in hemorrhagic corpus luteum cysts or neoplastic cysts, biopsy and preferably removal of the ovarian cyst for histologic confirmation are necessary for the diagnosis.

REVISED CLASSIFICATION FOR ENDOMETRIOSIS

HISTOLOGY

Positive histology confirms the diagnosis of endometriosis; negative histology does not exclude it. Whether histology should be obtained when peritoneal disease alone is present is controversial; visual inspection is usually adequate but histological confirmation of at least one lesion is ideal. In cases of ovarian endometrioma (> 4 cm in diameter) and in deeply infiltrating disease, histology is recommended to exclude rare instances of malignancy. Microscopically, endometriotic implants consist of endometrial glands and stroma, with or without hemosiderin-laden macrophages.

Deep endometriosis is described as a specific type of pelvic endometriosis characterized by proliferative strands of glands and stroma in dense fibrous and smooth muscle tissue. Microscopic endometriosis is defined as the presence of endometrial glands and stroma in macroscopically normal pelvic peritoneum. Macroscopically appearing normal peritoneum rarely contains microscopic endometriosis.



(a) REVISED AMERICAN SOCIETY FOR REPRODUCTIVE MEDICINE CLASSIFICATION OF ENDOMETRIOSIS 1985

Patient's Name _____ Date: _____

Stage I (Minimal)	1-5	Laparoscopy_____	Laparotomy_____ Photography_____
Stage II (Mild)	6-15	Recommended Treatment _____	
Stage III (Moderate)	16-40	_____	
Stage IV (Severe)	>40		
Total _____		Prognosis _____	

	ENDOMETRIOSIS	< 1 cm	1 – 3 cm	> 3 cm
Peritoneum	Superficial	1	2	4
	Deep	2	4	6
Ovary	R Superficial	1	2	4
	Deep	4	16	20
	L Superficial	1	2	4
	Deep	4	16	20
	POSTERIOR CULDESAC OBLITERATION	Partial		Complete
		4		40
	ADHESIONS	< 1/3 Enclosure	1/3-2/3 Enclosure	> 2/3 Enclosure
Ovary	R Filmy	1	2	4
	Dense	4	8	16
	L Filmy	1	2	4
	Dense	4	8	16
Tube	R Filmy	1	2	4
	Dense	4	8	16
	L Filmy	1	2	4
	Dense	4•	8•	16

•If the fimbriated end of the fallopian tube is completely enclosed, change the point assignment to 16.

Additional Endometriosis: _____

_____

_____

_____

Associated Pathology: _____

_____

_____

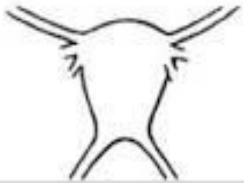
_____

To Be Used with Normal Tubes and Ovaries

L R

To Be Used with Abnormal Tubes and/or Ovaries

L R



LAPAROSCOPIC CLASSIFICATION

Many classification systems were proposed, but only one was accepted. This system is the revised American Fertility Society (AFS) system, which is based on the appearance, size, and depth of peritoneal and ovarian implants; the presence, extent, and type of adnexal adhesions; and the degree of cul-de-sac obliteration. In the ASRM classification system, the morphology of peritoneal and ovarian implants should be categorized as red (red, red-pink, and clear lesions), white (white, yellow-brown, and peritoneal defects), and black (black and blue lesions), according to color photographs provided by ASRM.

This system reflects the extent of endometriotic disease but has considerable intraobserver and interobserver variability. Like all classification systems, the ASRM classification for endometriosis is subjective and correlates poorly with pain symptoms, but may be of value in infertility prognosis and management. Because this ASRM revised classification of endometriosis is the only internationally accepted system, it is the best available tool to describe objectively the extent of endometriosis and relate it to spontaneous evolution. More outcome-based research is needed to discover whether it is possible to improve the standardization and positive correlation of the ASRM classification of endometriosis with symptoms (pain, infertility) and with therapeutic outcome after medical or surgical treatment. More variables than only the stage of endometriosis may have to be entered in such prediction models. Evidence suggests that endometriosis with an ASRM score of 16 or more, together with other variables like age, duration of infertility, and least functional score for ovaries and fallopian tubes after endometriosis surgery, is predictive of pregnancy.

SPONTANEOUS EVOLUTION

Endometriosis appears to be a progressive disease in a significant proportion (30% to 60%) of patients. During serial observations, deterioration (47%), improvement (30%), or elimination (23%) was documented over a 6-month period (265,266). In another study, endometriosis progressed in 64%, improved in 27%, and remained unchanged in 9% of patients over 12 months. A third study of 24 women reported 29% with disease progression, 29% with disease regression, and 42% with no change over 12 months. Follow-up studies in both baboons and women with spontaneous endometriosis over 24 months demonstrated disease progression in all baboons and in 6 of 7 women. Several studies reported that subtle lesions and typical implants may represent younger and older types of endometriosis, respectively. In a cross-sectional study, the incidence of subtle lesions decreased with age. This finding was confirmed by a 3-year prospective study that reported that the incidence, overall pelvic area involved, and volume of subtle lesions decreased with age, but in typical lesions, these parameters and the depth of infiltration increased with age. Remodeling of endometriotic lesions (transition between typical and subtle subtypes) is reported to occur in women and in baboons, indicating that endometriosis is a dynamic condition. Several studies in women, cynomolgus monkeys, and rodents showed that endometriosis is ameliorated after pregnancy.

The characteristics of endometriosis are variable during pregnancy, and lesions tend to enlarge during the first trimester but regress thereafter. Studies in baboons revealed no change in the number or surface area of endometriosis lesions during the first two trimesters of pregnancy. These results do not exclude a beneficial effect that may occur during the third trimester or in the immediate postpartum period. Establishment of a “pseudopregnant state” with exogenously administered estrogen and progestins is based on the belief that symptomatic improvement may result from decidualization of endometrial implants during pregnancy. This hypothesis is not substantiated, and it is possible that amenorrhea can explain the beneficial effect of pregnancy and lactation on endometriosis-associated pain symptoms.

MANAGEMENT

PREVENTION

No strategies to prevent endometriosis are uniformly successful. Oral contraceptives (OCs) inhibit ovulation, substantially reduce the volume of menstrual flow, and may interfere with implantation of refluxed endometrial cells, but the hypothesis of recommending OCs for primary prevention of endometriosis is not sufficiently substantiated.

PRINCIPLES OF TREATMENT

Treatment of endometriosis must be individualized, taking into consideration the clinical problem in its entirety, including the impact of the disease and the effect of its treatment on quality of life. In most women with endometriosis, preservation of reproductive function is desirable. Many women with endometriosis have pain and subfertility at the same time or may desire children after sufficient pain relief, which complicates the choice of treatment. Symptomatic endometriosis patients can be treated with analgesics, hormones, surgery, assisted reproduction, or a combination of these modalities.

SURGICAL MANAGEMENT

Depending on the severity of disease, diagnosis and removal of endometriosis should be performed simultaneously at the time of surgery. The goal of surgery is to excise all visible endometriotic lesions and associated adhesions—peritoneal lesions, ovarian cysts, deep rectovaginal endometriosis—and to restore normal anatomy. Laparotomy should be reserved for patients with advanced-stage disease who cannot undergo a laparoscopic procedure and for those in whom fertility conservation is not necessary.

PERITONEAL ENDOMETRIOSIS

Endometriosis lesions can be removed during laparoscopy by surgical excision with scissors, bipolar coagulation, or laser methods (CO2 laser, potassium-titanyl-phosphate laser, or argon laser).Comparable cumulative pregnancy rates were reported after treatment of mild endometriosis with laparoscopic excision and electrocoagulation. Ablation of endometriotic peritoneal lesions plus laparoscopic uterine nerve ablation (LUNA) in minimal to moderate disease reduces endometriosis-associated pain at 6 months compared to diagnostic laparoscopy; the smallest effect is seen in patients with minimal disease. There is no evidence that LUNA is a necessary component, and LUNA by itself has no effect on dysmenorrhoea

associated with endometriosis.

ADHESIOLYSIS

The removal of endometriosis-related adhesions (adhesiolysis) should be performed carefully. Adhesions lysed at surgery can form again.

OVARIAN ENDOMETRIOSIS

SURGICAL TECHNNIQUE

Superficial ovarian lesions can be vaporized. The surgical management of ovarian endometriotic cysts is controversial. The primary indication for extirpation of an endometrioma is to ensure it is not malignant . The most common procedure for the treatment of ovarian endometriomas are either excision of the cyst capsule or drainage and electrocoagulation of the cyst wall.There is good evidence that excisional surgery for endometriomas with a diameter of 3 cm provides a more favorable outcome than drainage and ablation with regard to the recurrence of the endometrioma, pain symptoms and in women who were previously subfertile or had subsequent spontaneous pregnancy.

RECTOVAGINAL/ RECTOSIGMOID ENDOMETRIOSIS

Deeply infiltrating endometriosis is usually multifocal and complete surgical excision must be performed in a one-step surgical procedure in order to avoid more than one surgery, Surgical excision of deep rectovaginal and rectosigmoidal endometriosis is difficult and can be associated with major complications such as bowel perforations with resulting peritonitis. Because management of deeply infiltrating endometriosis is complex, referral to a center with sufficient expertise to offer all available treatments in a multidisciplinary approach is strongly recommended. Surgical management is only for symptomatic deeply infiltrating endometriosis. Asymptomatic patients must not undergo surgery. Progression of the disease and appearance of specific symptoms rarely occurred in patients with asymptomatic rectovaginal endometriosis. When surgical treatment is decided, the treatment must be radical with excision of all infiltrating lesions. It is difficult to perform randomized studies to detect the best surgical technique to treat deeply infiltrative endometriosis because these severe cases are all managed individually and not all surgeons are familiar with all treatment options. Complete excision while preserving the uterus and ovarian tissue might include the resection of the uterosacral ligaments, the resection of the upper part of posterior vaginal wall, urologic and bowel operations.

HORMONAL THERAPY

In patients with severe endometriosis, it is recommended that surgical treatment be preceded by a 3-month course of medical treatment to reduce vascularization and nodular size.

OOPHORECTOMY AND HYSTERECTOMY

Radical procedures such as oophorectomy or total hysterectomy are indicated only in severe situations and can be performed laparoscopically or by laparotomy.

LOCAL TREATMENT

Postoperative use of an levonorgestrel intrauterine system in women with endometriosis improves pain symptoms associated with menstruation and reduces recurrence compared with surgery only, placebo, or systemic hormones. Another small randomized trial, postsurgical treatment with either levonorgestrel intrauterine system or depot MPA for 3 years indicated symptom control and recurrence were comparable, but compliance and change in bone mineral density were better in the levonorgestrel intrauterine system treated group than in the depot MPA group.

MEDICAL MANAGEMENT

For Primary dysmenorrhea

- Main modalities for control of dysmenorrhea are
- Analgesics
- OCP's
- Other methods include ,Supplemental thiamine ,Vitamin E,TENS,Topical heat

For Endometriosis associated pain

- **NSAIDS**-Endometriosis-related pain is nociceptive, but persistent nociceptive input from endometriotic lesions leads to central sensitization manifested by somatic hyperalgesia and increased referred pain. The potential effectiveness of NSAIDs in the reduction of endometriosis-related pain may be explained by a local antinociceptive effect and a reduced central sensitization in addition to the anti-inflammatory effect. NSAIDs have significant side effects, including gastric ulceration and possible inhibition of ovulation. Prostaglandins are involved in the follicle rupture mechanism at ovulation, which is why women who wish to become pregnant should not take NSAIDs at the time of ovulation.
- **HORMONAL SUPPLEMENTS**- There is strong evidence that suppression of ovarian function for 6 months reduces pain associated with endometriosis. Combined oral contraceptives danazol, gestrinone, medroxyprogesterone acetate, and GnRH agonists are all equally effective but

their side effects and cost profiles differ. Pain relief may be of short duration, presumably because endometriosis and endometriosis-associated pain recur after the cessation of medical treatment. The use of diethylstilbestrol, methyltestosterone, or other androgens is no longer advocated because they lack efficacy, have significant side effects, and pose risks to the fetus if pregnancy occurs during therapy. A new generation of aromatase inhibitors, estrogen receptor modulators, and progesterone antagonists may offer new hormonal treatment options.

- Hormonal Treatment for Pain from Rectovaginal Endometriosis

- Surgical treatment may reduce the pain associated with rectovaginal endometriosis, but it is associated with a high risk of morbidity and major complications. The effect of medical treatment in terms of pain relief in women with rectovaginal endometriosis appears to be substantial. In a systematic review including 217 cases of medically treated rectovaginal endometriosis, the antalgic effect of the considered medical therapies (vaginal danazol, GnRH agonist, progestin, and estrogen-progestin combinations used transvaginally, transdermally, or orally) for the entire treatment period (from 6 to 12 months) was 60% to 90%, with patients reporting considerable reduction or complete relief from pain symptoms, with the exception of when an aromatase inhibitor was used alone.

Oral contraceptives can be given cyclically or continuously .The objective of treatment is to induce amenorrhea which should be continued for six to twelve months .

PROGESTINS

These are first line of choice for endometriosis

- MPA- It is the most studied agent. It is effective in relieving pain starting at a dose of 30 mg per day, increasing the dose based on the clinical response and bleeding patterns according to data from nonrandomized trials. A randomized placebo-controlled study reported a significant reduction in stages and scores of endometriosis in both the placebo group and the group treated with MPA 50 mg per day and placebo at laparoscopy within 3 months after cessation of therapy. These findings raise questions about the need for medical therapy with MPA in this dose.Evidence suggests a possible role for depot MPA in the treatment of endometriosis. In a randomized controlled study, depot MPA (150 mg every 3 months) was more effective in the relief of dysmenorrhea than treatment with a cyclic 21-day oral contraceptive (ethinyl estradiol 20 μg plus desogestrel 0.15 mg) combined with very low-dose danazol (50 mg per day). In another multicenter, randomized, evaluator-blinded, comparator-controlled trial, depot MPA (150 mg) or leuprolide acetate (11.25 mg), given every 3 months for 6 months were equivalent in reducing endometriosis-associated pain during the study and the 12-month posttreatment follow-up period, with less impact on bone mineral density and fewer hypoestrogenic side effects but more bleeding being observed in the depot MPA treated group.

Add-back therapy would prevent the negative effects on bone density and hypoestrogenic side effects associated with GnRH-agonist therapy. In a pilot study, pain relief, side effects, and treatment satisfaction were comparable during a 12-month treatment with etonogestrel implantate subcutaneous (68 mg) or depot medroxyprogesterone acetate 150 mg intramuscular depot MPA in 41 patients with dysmenorrhea, nonmenstrual pelvic pain, and dyspareunia associated with histologically proven endometriosis. Although depot MPA treatment is effective for the treatment of pain associated with endometriosis, it is not indicated in infertile women because it induces profound amenorrhea and anovulation, and a varying length of time is required for ovulation to resume after discontinuation of therapy.

- DINOGEST –in studies, treatment during 6 months with dienogest 2 mg per day orally demonstrated equivalent efficacy to depot leuprolide acetate (3.75 mg, depot intramuscular injection, every 4 weeks) or intranasal buserelin acetate (900 µg per day, intranasally) in relieving the pain associated with endometriosis, offering a different safety and tolerability profile (less bone loss, fewer hot flushes, more irregular genital bleeding.

- LNG-IUS -The levonorgestrel intrauterine system releasing 20 µg per day of levonorgestrel reduces endometriosis-associated pain caused by peritoneal and rectovaginal endometriosis and reduces the risk of recurrence of dysmenorrhea after conservative surgery. Levonorgestrel induces endometrial glandular atrophy and decidual transformation of the stroma, reduces endometrial cell proliferation, and increases apoptotic activity. A systematic review identified two randomized trials and three prospective observational studies, all involving small numbers and a heterogeneous group of patients. The evidence suggests that the levonorgestrel intrauterine system reduces endometriosis associated pain with symptom control maintained over 3 years. Twelve months of treatment results in a significant reduction in dysmenorrhea, pelvic pain, and dyspareunia; a high degree of patient satisfaction; and a significant reduction in the volume of rectovaginal endometriotic nodules. After the first year of use, a 70% to 90% reduction in menstrual blood loss is observed.

- MEGESTROL ACETATE -Megestrol acetate was administered in a dose of 40 mg per day with good results. Pain was reduced significantly during luteal phase treatment with 60 mg dydrogesterone, and this improvement was still evident at 12-month follow-up. Other treatment strategies included dydrogesterone (20 to 30 mg per day, either continuously or on days 5 to 21) and lynestrenol (10 mg per day).
- Side effects of progestins include nausea, weight gain, fluid retention, and breakthrough bleeding caused by hypoestrogenemia. Breakthrough bleeding, although common, is usually corrected by short-term (7-day) administration of estrogen. Depression and other mood disorders are a significant problem in about 1% of women taking these medications.

PROGESTERONE ANTAGONISTS

MIFEPRISTONE

Mifepristone (RU-486) is a potent antiprogestagen with a direct inhibitory effect on human endometrial cells and, in high doses, an antiglucocorticoid action. The recommended dose for endometriosis is 25 to 100 mg per day. In uncontrolled studies, mifepristone, 50 to 100 mg per day, reduced pelvic pain and induced 55% regression of the lesions without significant side effects. In an uncontrolled pilot study, low-dose mifepristone, 5 mg per day, resulted in pain improvement, without change in endometriosis lesions, suggesting that this dosage was probably too low.

ONAPRISTONE

The progesterone antagonists onapristone (ZK98299) and ZK136799, used in the treatment of rats with surgically induced endometriosis, resulted in a remission in 40% to 60% of treated animals. In animals with persistent endometriosis, growth inhibition was obtained in 48% and 85% of endometriotic lesions after therapy with onapristone and ZK136799, respectively.

GESTRINONE

Gestrinone is a 19-nortestosterone derivative with androgenic, antiprogestagenic, antiestrogenic, and antigonadotropic properties. It acts centrally and peripherally to increase free testosterone and reduce sex hormone–binding globulin levels (androgenic effect), reduce serum estradiol values to early follicular phase levels (antiestrogenic effect), reduce mean LH levels, and obliterate the LH and follicle-stimulating hormone (FSH) surge (antigonadotropic effect). Gestrinone“causes cellular inactivation and degeneration of endometriotic implants but not their disappearance. Amenorrhea occurs in 50% to 100% of women and is dose dependent. Resumption of menses generally occurs 33 days after discontinuing the medication (389,390). An advantage of gestrinone is its long half-life (28 hours) when given orally. The standard dose is 2.5 mg twice a week. Although 1.25 mg twice weekly is effective, a randomized study demonstrated in women with mild to moderate endometriosis that 2.5 mg gestrinone twice weekly for 24 weeks is more effective and has a better effect on bone mass (+7% vs. −7%) when compared with 1.25 mg gestrinonetwice weekly for 24 weeks. The clinical side effects of gestrinone are dose dependent and similar to but lessintense than those caused by danazol. They include nausea, muscle cramps, and androgenic effects such as weight gain, acne, seborrhea, and oily hair and skin.

DANAZOL

Recognized pharmacologic properties of danazol include suppression of GnRH or gonadotropin secretion, direct inhibition of steroidogenesis, increased metabolic clearance of estradiol and progesterone, direct antagonistic and agonistic interaction with endometrial androgen and progesterone receptors, and immunologic attenuation of potentially adverse reproductive effects. The multiple effects of danazol produce a high-androgen, low-estrogen environment (estrogen levels in the early follicular to postmenopausal range) that does not support the growth of endometriosis, and the amenorrhea that is produced prevents new seeding of implants from the uterus into the peritoneal cavity.

The immunologic effects of danazol were studied in women with endometriosis and adenomyosis and include a decrease in serum immunoglobulins, a decrease in serum C3, a rise in serum C4 levels, decreased serum levels of autoantibodies against various phospholipid antigens, and decreased serum levels of CA125 during treatment . Danazol inhibits peripheral blood lymphocyte proliferation in cultures activated by T-cell mitogens but does not affect macrophage-dependent T-lymphocyte activation of B lymphocytes. It inhibits TNF production by monocytes in a dose-dependent manner and suppresses macrophage- and monocyte-mediated cytotoxicity of susceptible target cells in women with mild endometriosis. These immunologic findings may be important in the remission of endometriosis with danazol treatment and may offer an explanation of the effect of danazol in the treatment of a number of autoimmune diseases, including hereditary angioedema, autoimmune hemolytic anemia, systemic lupus erythematosus, and idiopathic thrombocytopenic purpura.

Doses of 800 mg per day are used frequently in North America, whereas 600 mg per day is prescribed in Europe and Australia. It appears that the absence of menstruation is a better indicator of response than drug dose. A practical strategy for the use of danazol is to start treatment with 400 mg daily (200 mg twice a day) and increase the dose, if necessary, to achieve amenorrhea and relieve symptoms. The significant adverse side effects of danazol are related to its androgenic and hypoestrogenic properties. The most common side effects include weight gain, fluid retention, acne, oily skin, hirsutism, hot flashes, atrophic vaginitis, reduced breast size, reduced libido, fatigue, nausea, muscle cramps, and emotional instability. Deepening of the voice is another potential side effect that is nonreversible. Danazol can cause increased cholesterol and low-density lipoprotein levels and decreased high-density lipoproteins levels, but it is unlikely that these short-term effects are clinically important. Danazol is contraindicated in patients with liver disease because it is largely metabolized in the liver and may cause hepatocellular damage.

GNRH AGONISTS

Gonadotropin-releasing hormone agonists bind to pituitary GnRH receptors and stimulate LH and FSH synthesis and release. Treatment for 3 months with a GnRH agonist is effective in improving pain for 6 months. The dose of daily GnRH agonist can be regulated by monitoring estradiol levels, by the addition of low-dose progestin or estrogen–progestin in an add-back regimen, or by draw-back therapy. GnRH agonists with add-back therapy can only be considered for adolescents older than 17 years of age who have completed pubertal and bone maturation, and then only if symptoms persist during other forms of hormonal suppression.

AROMATASE INHIBITORS

There is concern with the use of aromatase inhibitors such as anastrozole or letrozole in the treatment of menopausal women because these drugs are known to stimulate ovulation and continuous administration can result in the development of functional ovarian cysts. This side effect can be prevented by combining aromatase inhibitors with ovarian suppressing drugs such as OCs or progestins in premenopausal women.

SERM

The role of selective estrogen receptor modulators (SERMs) in the treatment of endometriosis is unclear. In animal models, raloxifene therapy resulted in regression of endometriosis.

NON HORMONAL MEDICAL THERAPY

Selective Inhibition of Tumor Necrosis Factor-α, Pentoxyfylline, Peroxisome Proliferator Activated Receptor-γ agonists can be tried but their safety is an important issue regarding human use.

MANAGEMENT OF RECURRENCE

Endometriosis tends to recur unless definitive surgery is performed. Pain recurs within 5 years in about one in five patients with pelvic pain treated by complete laparoscopic excision of visible endometriotic lesions. The risk of recurrence of endometriosis during hormonal therapy seems marginal if combined preparations or tibolone are used and estrogen-only treatments are avoided. After first-line surgery for endometriosis, women should be invited to seek conception as soon as possible. Alternatively, OC use until pregnancy is desired should be considered because several lines of evidence suggest that ovulation inhibition reduces the risk of endometriosis recurrence. Medical treatment of recurrence with OCPs significantly improved dysmenorrhea and chronic pelvic pain.

Surgical management includes conservative surgeries and hysterectomy.

COPING WITH DISEASE

Coping with endometriosis as a chronic disease is an important component of management. According to guidelines for the management of endometriosis, evidence from two systematic reviews suggests that high frequency transcutaneous electrical nerve stimulation (TENS), acupuncture, vitamin B1, and magnesium may help to relieve dysmenorrhea. Whether such treatments are effective in endometriosis-associated dysmenorrhea is unknown. Many women with endometriosis report that nutritional and complementary therapies such as reflexology, traditional Chinese medicine, herbal treatments, and homeopathy improve pain symptoms. Although there is no evidence from randomized controlled trials to support the effectiveness of these treatments in endometriosis, they should not be ruled out if the woman feels they work in conjunction with more traditional therapies or that they could be beneficial to her overall pain management and quality of life.

AIM OF THE STUDY

To study the incidence of endometriosis in women with infertility.

OBJECTIVES:

To find out the number of cases of endometriosis in women affected with infertility

MATERIALS AND METHODS

STUDY DESIGN: A prospective observational study

PLACE OF THE STUDY : Department of Obstetrics and gynecology, Govt. RSRM Lying in Hospital

STUDY POPULATION:

-Patients attending the Outpatient clinics, Antenatal ward, Labourward of the Department of Obstetrics and gynecology, Govt Stanley Medical college & hospital, Chennai

STUDY PERIOD: 1 year and 6 month

METHODOLOGY

- **Women belonging to age group of 21-39 yrs (eligible criteria) with infertility are taken into study**
- **Relevant information,clinical examination, detailed history**
- **Oral and written consent are obtained**
- **Biochemical investigations Complete blood count,renal function test ,liver function test done in all patients.**
- **Laparoscopic study is performed in women with infertility to diagnose and confirm endometriosis by the presence of typical powder burns appearance, flame lesions, Endometriotic cysts etc.**
- **Patient will be explained about nature of Study and informed consent is obtained**
- **The results so obtained will be analysed by statistical methods.**

Inclusion criteria	**Age group 21-39 yrs** **Primary or secondary infertility** **Normal semen analysis** **Normal TFT** **Absence of known biochemical or other hormonal derangements**
Exclusion criteria	**• Age >39 yrs** **• Women with Hypothyroidism or hyperthyroidism** **• Women with altered liver /renal function test** **. Lack of willingness**

SAMPLE SIZE CALCULATION:

Formula:

n = Z2pq / d2

Where Z = 1.96 (statistical significant constant for 95% CI)

p =21 %(Prevalence of Infertility among women with Endometriosis from previous study.)

q = 79 % (100-p)

d = 5 % absolute precision.

On substituting, in the formula

n = 3.84 x 21 x 79 / 25

n = 254

Adding 10% non response rate

n = 279 (minimum sample size)

Therefore Sample size n = 280 (1 group).

DATA ANALYSIS

- Age distribution
- Menstrual history and type of dysmenorrhea if present.
- Type of infertility and number of children if secondary
- Husband semen analysis
- Other associated medical conditions

STATISTICS .

Results and Observations

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0.(Armonk, NY: IBM Corp).To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.

Table: 1 Age Distribution

Age distribution		
	Frequency	Percent
Upto 25 years	21	7.5
26 - 30 years	118	42.3
31 - 35 years	103	36.9
Above 35 years	37	13.3
Total	279	100.0

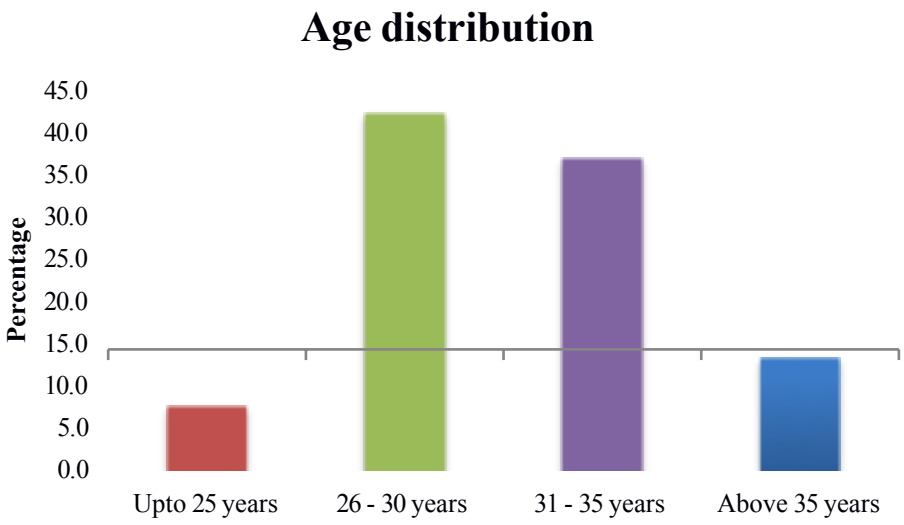


Figure: 1

The table shows the age distribution of the study population which consists of up to 25 years were 7.5 % , 26 to 30 years were 42.3% , 31 to 35 years were 36.9 % and above 35 years were 12.3%.

Table: 2 Menstrual history Distribution

Menstrual history		
	Frequency	Percent
Irrergular	125	44.8
Regular	154	55.2
Total	279	100.0

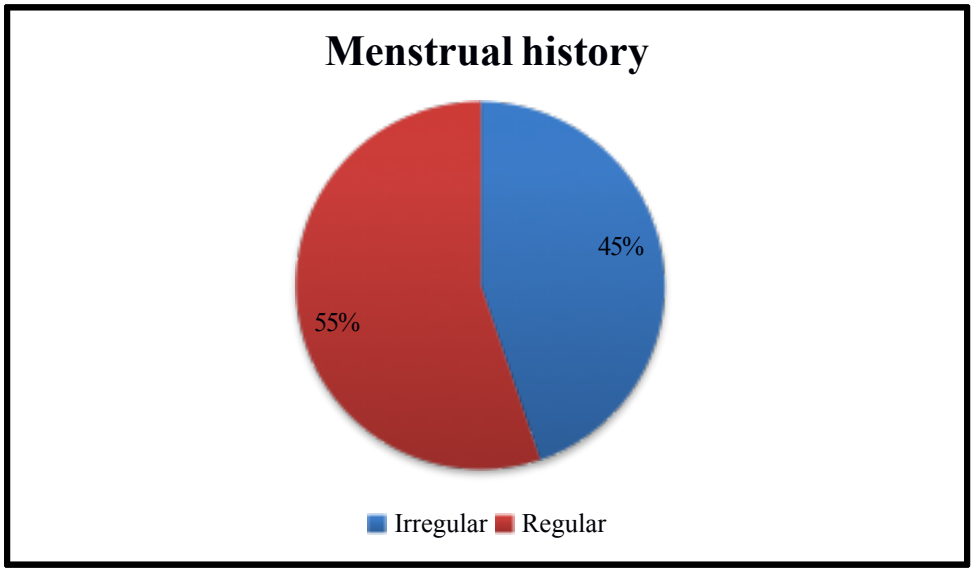


Figure: 2

The above table shows the menstrual distribution among the study population in which the consists of 44.8% with irregular menstrual and 55.2% of the study population was found with regular menstrual history.

Table: 3 Type of dysmenorrhea Distribution

Type of dysmenorrhea		
	Frequency	Percent
Congestive	13	4.7
No dysmenorrhea	266	95.3
Total	279	100.0

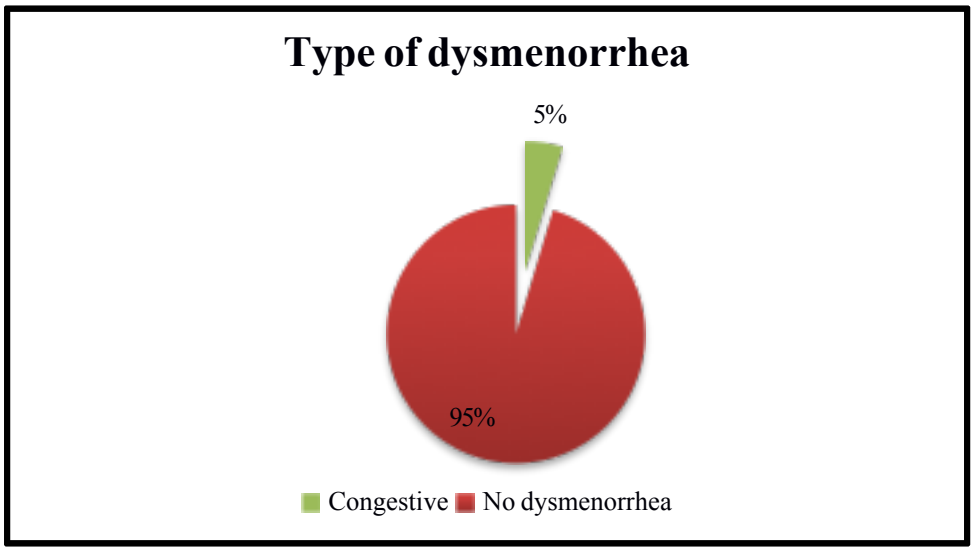


Figure: 3

The above table shows the dysmenorrhoea distribution among the study population in which the congestive was 4.7 % and 95.3% of the study population was found with no dysmenorrhoea disorder.

Table: 4 Type of infertility Distribution

Type of infertility		
	Frequency	Percent
Primary	266	95.3
Secondary	13	4.7
Total	279	100.0

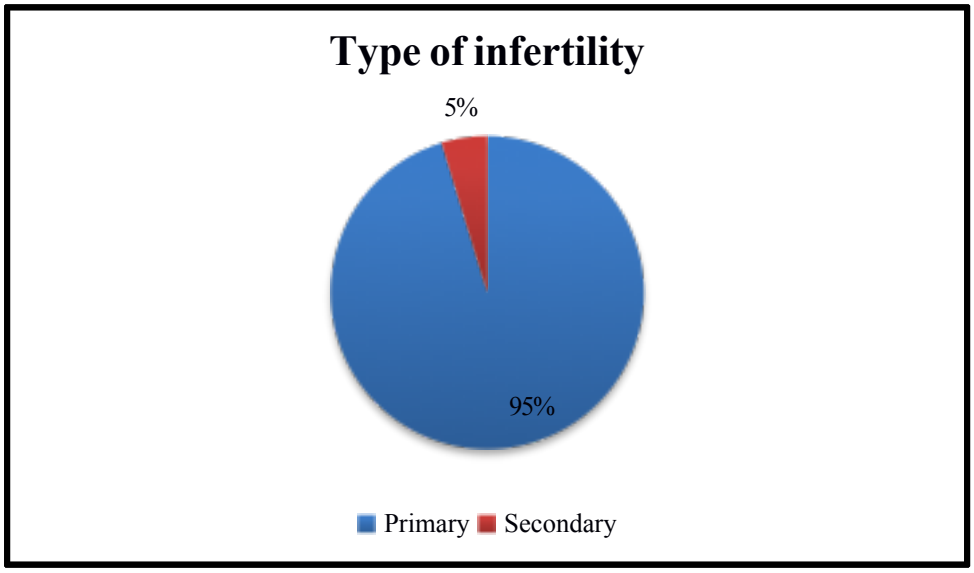


Figure: 4

The above table shows the distribution of type of infertility in the study population which consists of 95.3 % with primary type of infertility and 4.7 % of them with secondary type of infertility.

Table: 5 Number of Children Distribution

No of children		
	Frequency	Percent
1	13	4.7
Nil	266	95.3
Total	279	100.0

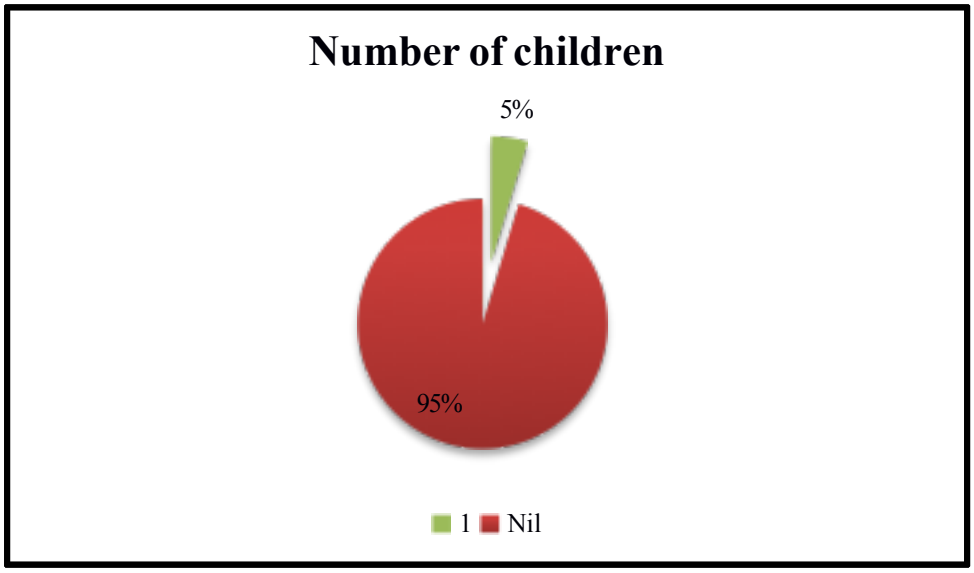


Figure: 5

The above table shows the distribution of number of children in the study population which consist of 4.7 % with one child whereas 95.3 % have no time children act.

Table: 6 Semen Analysis Distribution

Semen analysis		
	Frequency	Percent
Normal	265	94.497
Oligospermia	7	2.5
Oligoteratozoospermia	1	0.0358423
Total	279	100.0

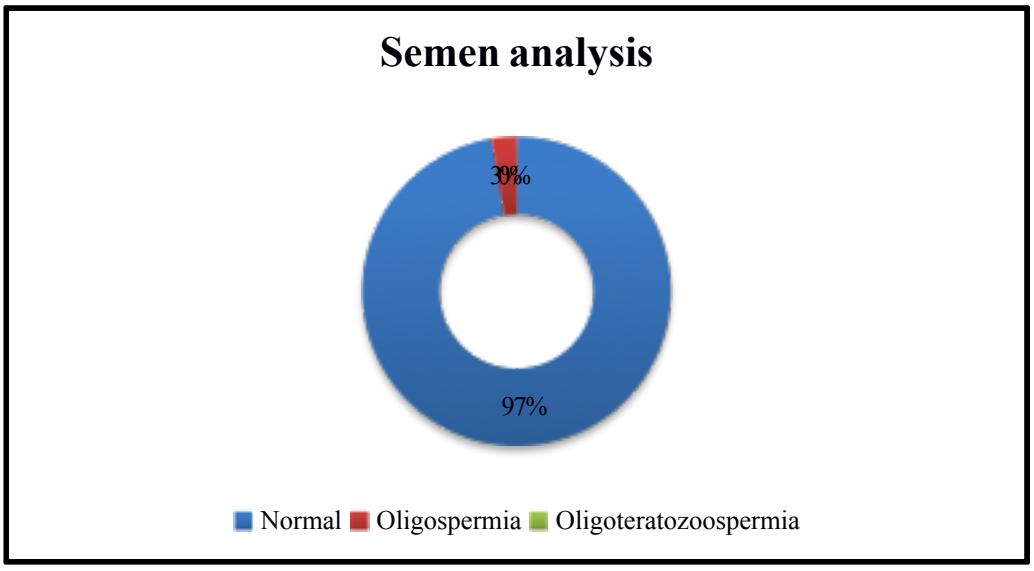


Figure: 6

The above table shows the distribution of semen analysis which consists of 97.5.0 % normal semen outcome and Oligospermia in 2.5% cases & Oligoteratozoospermia in 0.003% cases.

Table: 7 Medical disorders Distribution

Medical disorders		
	Frequency	Percent
Hypothyroid	21	7.5
Nil	258	92.5
Total	279	100.0

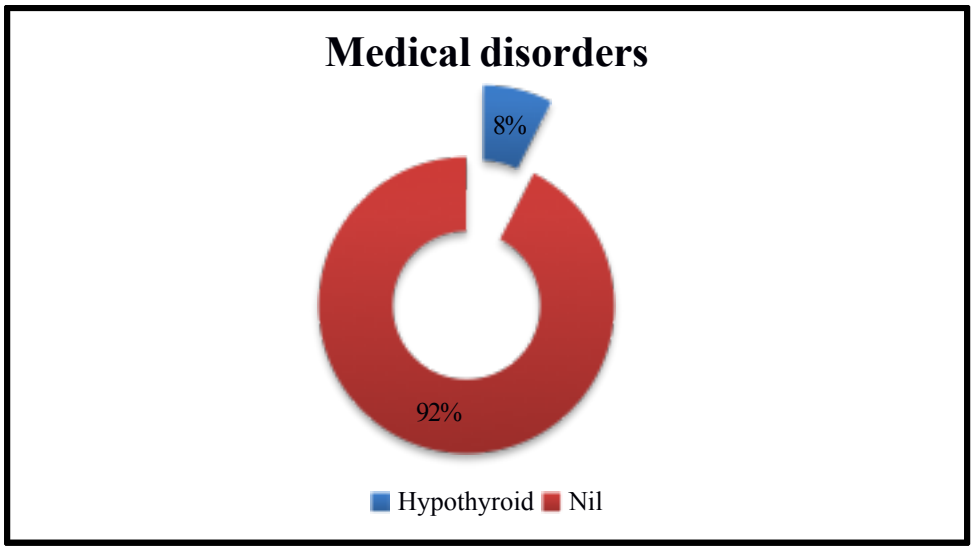


Figure: 7

The above table shows the medical disorder of the study population 7.5 % of the study population were found with the hypothyroidism whereas 92.5 % were without any medical disorder.

Table: 8 Endometriotic lesion Distribution

Endometriotic lesions		
	Frequency	Percent
Present	29	10.4
Absent	255	91.4
Total	284	100.0

The above table shows the Endometriotic lesion of the study population 10.4 % of the study population were found with the Endometriotic lesion and 91.4 % were absent.

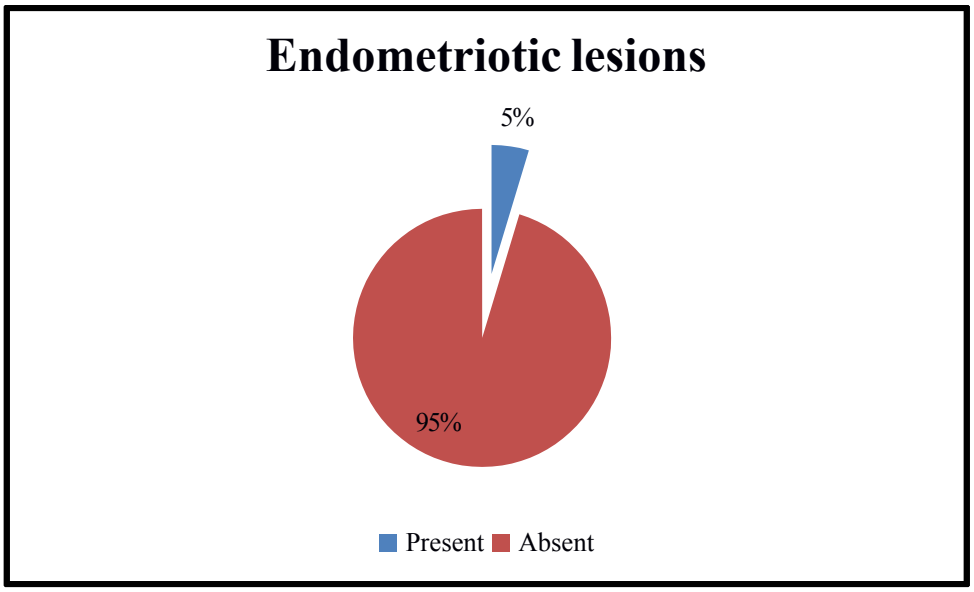
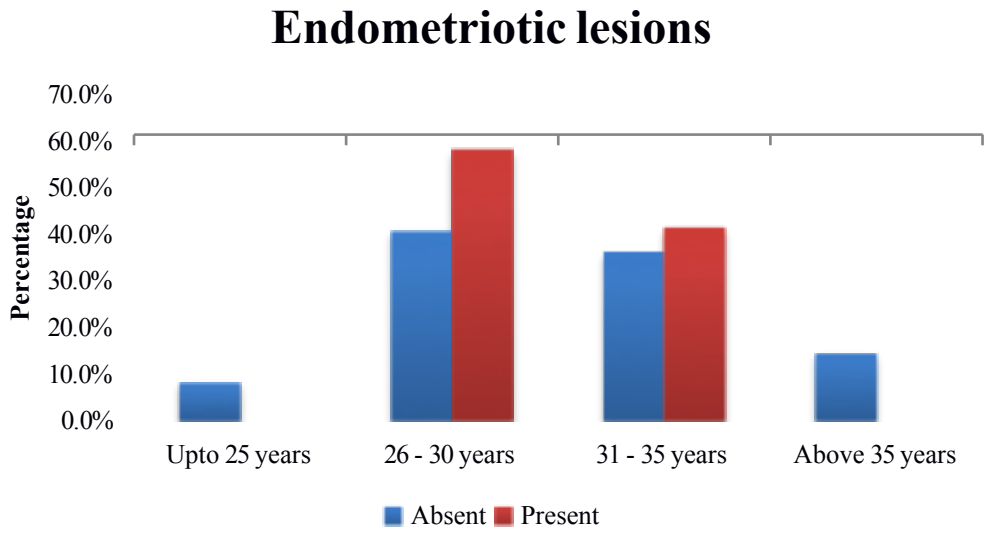


Table: 9 Age with Endometriotic lesions Distribution

			Endometriotic lesions		Total
			Absent	Present	
Age	Upto 25 years	Count	21	0	21
		%	8.2%	0.0%	7.5%
	26 - 30 years	Count	104	14	118
		%	40.8%	58.3%	42.3%
	31 - 35 years	Count	89	14	103
		%	36.5%	41.7%	36.9%
	Above 35 years	Count	37	0	37
		%	14.5%	0.0%	13.3%
Total		Count	255	24	279
		%	100.0%	100.0%	100.0%



The above table shows the Age wise distribution of Endometriotic lesions in the studypopulation.

Table: 10 Descriptive statistics Distribution

Descriptive Statistics					
	N	Minimum	Maximum	Mean	SD
S.no	279	1.0	279.0	140.0	80.7
Age	279	22.0	36.0	30.4	3.9
Married since	279	3.0	16.0	7.5	3.2
Duration of infertility	279	3.0	16.0	7.7	3.4
TFT	279	1.60	6.00	3.0	1.0
Prolactin	279	9.0	22.0	14.8	3.2
CA 125	279	11.0	27.0	16.1	3.6

DISCUSSION

- The age distribution of the study population which consists of up to 25 years were 7.5 % , 26 to 30 years were 42.3% , 31 to 35 years were 36.9 % and above 35 years were 12.3%.
- The menstrual distribution among the study population in which the consists of 44.8% with irregular menstrual and 55.2% of the study population was found with regular menstrual history.
- The dysmenorrhoea distribution among the study population in which the congestive was 4.7 % and 95.3% of the study population was found with no dysmenorrhoea disorder.
- The distribution of type of infertility in the study population which consists of 95.3 % with primary type of infertility and 4.7 % of them with secondary type of infertility
- The distribution of number of children in the study population which consist of 4.7 % with one child whereas 95.3 % have no time children act.
- The distribution of semen analysis which consists of 94.497 % normal semen outcome Oligospermia in 2.5% cases & Oligoteratozoospermia in 0.003548%
- The medical disorder of the study population 7.5 % of the study population were found with the hypothyroidism whereas 92.5 % were without any medical disorder.
- The Endometriotic lesion of the study population 10.4 % of the study population were found with the Endometriotic lesion and 91.4 % were absent.

CONCLUSION

Endometriosis is being detected more often these days, due to early investigations and prompt seeking of medical care. In my study I have observed there is significant causal relationship between endometriosis and infertility. Incidence of endometriosis in patients with infertility is 10.4% which is quite lesser than the previous studies, still higher than the incidence in fertile patients. I conclude my study by saying that endometriosis is a condition that is one of the leading causes of infertility and hence early diagnosis and management are key to prevent or alleviate associated morbidities.

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PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled 'INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY' of the candidate Dr.J PRIYADHARSHINI with registration number 221916065 for the award of MS in the branch of OBSTETRICS AND GYNECOLOGY. I personally verified the urkund.com website for the purpose of plagiarism check. I found that uploaded thesis file contains from introduction to conclusion pages and result shows 7 percentage of plagiarism in the dissertation.

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INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK :"INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY"
PRINCIPAL INVESTIGATOR : DR. PRIYADARSHINI,
DESIGNATION : PG IN OBSTETRICS & GYNAECOLOGY,
DEPARTMENT : DEPARTMENT OF OBSTETRICS & GYNAECOLOGY,
GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 10.09.2020 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

“INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY"

NAME :

AGE :

OP / IP NO :

ADDRESS & CONTACT NO : :

MENSTRUAL HISTORY

TYPE OF DYSMENORRHEA(IF PRESENT)

MARITAL HISTORY

MARRIED SINCE

DYSPAREUNIA

OBSTETRIC HISTORY

TYPE OF INFERTILITY:

-PRIMARY/SECONDARY

IF SECONDARY

DURATION OF INFERTILITY

NO OF CHILDREN

MALE FACTORS

SEMEN ANALYSIS

SEXUAL HABITS

PAST HISTORY

K/C/O HYPOTHYROIDISM/ HYPERTHYROIDISM

TREATMENT HISTORY

ANY PREVIOUS SURGERY

ANY PREVIOUS TREATMENT FOR INFERTILITY

PERSONAL HISTORY

-DYSURIA

-DYSCHEZIA

INVESTIGATIONS

COMPLETE BLOOD COUNT

LIVER FUNCTION TEST

RENAL FUNCTION TEST

TFT

PROLACTIN

CA 125

PATIENT INFORMATION SHEET

"INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY"

We are conducting a study among patients attending Department of Obstetrics and Gynecology , Stanley Medical College . The purpose of this study is know the various treatment modalities in infertile women with Endometriois .

We are selecting patients in age group of 21-35yrs ,who are infertile,with features suggestive of Endometriois, as a prospective observational study on the various treatment modalities and outcome . privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the study will be intimated to you at the end of the study to aid in the management or treatment.

Signature of Investigator **signature of participant**

Date:

Confidentiality:

Utmost priority will be given to protect the privacy and confidentiality of your personal information. The collected information will not be shared with anyone not involved in the study and reporting will be done in aggregate form only

Voluntary participation:

Your participation in this study is voluntary and you have the right to withdraw your participation at any time during the interview without any explanation. Refusal to participate will not involve any penalty or loss of benefits to which you are otherwise entitled. There might be certain questions which you may not wish to answer. You can choose to decline answering these questions.

Informed Consent form

Title of the study: INCIDENCE OF ENDOMETRIOSIS IN INFERTILITY - A PROSPECTIVE STUDY"

I agree to participate in the study entitled and have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I can receive the medical treatment as usual.

I understand that I will not get any money for taking part in the study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full cooperation for this study

Name of the participant :

Signature / Left thumb print:

Date :

Name of the investigator:Dr.J.PRIYA DHARSHINI

Signature of investigator :

Date :

-

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நாÍஇÏதஆராm235a567வDij;அ;j;►ய்Ứaடூa.

இÏதஆராm235%எÏதÝÍ7ளைEDஇẰலைளÍபதனnyj;►ய்Ứaடூa.

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இÏதஆராm235%கூi►nபு{Dகூa7கÍỂỆஎÍனா%5யaறவரூஉaமை7வDஅ

Wn♂ỮaனaபதைஉவீĜயGHMறடூa.

எa56மண;டaஇjதஆராm23HIஒG;முnபைஅWHMறூa.

தகவẰநகẰ

இÏதஆராm235%உmகWடDகூi►nபு{Dகூa7கÍỂỆஉ mகÍÃÆமனČடÍபYல

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இÏதஆராm235%உmகÍỂỆஎÏதÝÍ7ளைEDஏÏபாČளÍபதனாÍஉபுYயWHM

றடூ.

உKகaHIபணDஎ;EDஅWHகா♂டார்எaபதைஇதa®KDதி y7HMறடூa.

இÏதஆm7%உmகÍỂỆஎÏதநோேரpபa░;EDஇẰலை.
;mகÍஅWHIDதகூ%®லDn;புHYகÍவIH►nபடலP.

அதÍTலDவÅKகாலZY%உmகÍỂகாேஅẰல;உ mகளைோேÍறமỂகÍỂỆபயaபட

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;mகÍஎnபோேČவடூa{மாண`DஇÏதஆm7a4j;7லMH►ய்Ứa♫லP .அதனாẰஉ

m►Wa7♂♥m►aஎ;EDதி y7Hகா♂டார்எaபதைஉவீĜயGHMறடூa.

இÏதஆராm235a5pEகÍபZYyH►கைW%»♥byH►nபடலP.

ஆன%▌mகWaஅடவைளDஎ;EDதி y7Hகா♂டார்.

		Menstrual h	Type of dys	Married stu	Dyspareuni	Type of infe	Duration of	No of childr	Semen anal	Sexual habit	Medical dis	Prev surgery	Dysuria	Dyschezia	CBC	RBS, RFT, ITFT	Prolactin	CA 125	Intraop findings
2	m	Regular	Nil	3years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	13 Multiple cysts in POD
3	ermenpet	Irregular	Nil	5 years	Nil	Primary	5years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	1.9	21	18 B/L Spill+
4	un	Irregular	Nil	15years	Nil	Primary	15years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	16	17 B/L Spill+
5	ermenpet	Irregular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	13 B/L Spill+
6	ram	Regular	Congestive	9years	Nil	Secondary	9years		1 Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	22	11 Rt paraovarian simple cyst, B/L spill+
7		Irregular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	14	17 B/L Spill+
8	r	Irregular	Nil	8years	Nil	Primary	12years	Nil	Oligoterato	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	16 B/L Spill+
9	iyoor	Regular	Nil	9years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	12	17 B/L Spill+
10		Regular	Nil	13years	Nil	Primary	13years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.6	13	18 Adhesions+
11		Irregular	Nil	11years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	14	17 B/L Spill+
12	e	Irregular	Nil	8years	Nil	Primary	16years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	21	14 Endometriotic lesions+ on round ligament, uterine serosa
13	yoor	Irregular	Nil	12years	Nil	Primary	12 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	17	12 B/L Spill+
14	gate	Regular	Nil	4 years	Nil	Primary	4 years	Nil	Oligosperm	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	9	11 B/L spill+
15	zt	Irregular	Nil	5years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5.1	18	12 Right sided tube-patent, No spillage from left tube, endometriotic lesions+
16	di	Regular	Nil	3years	Nil	Primary	3 years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	2.4	17	21 B/L Spill+
17	yoor	Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	18 B/L Spill+
18	zt	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	12	11 B/L spill+
19		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	11	15 B/L Spill+, Endometriotic lesions+ on round ligament, uterine serosa
20		Irregular	Nil	7years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.9	19	21 B/L Spill+
21	iyoor	Regular	Nil	9year	Nil	Primary	9years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	6	13	17 B/L Spill+
22	zt	Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	13 B/L Spill+
23	un	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18 B/L Spill+
24	ermenpet	Irregular	Congestive	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	17	11 Endometriotic lesions+ on anterior pelvic wall
25	yoor	Regular	Nil	16years	Nil	Secondary	14years		1 Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18 B/L Spill+
26	di	Regular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.4	11	14 B/L Spill+
27	r	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	12	17 B/L spill+
28	ram	Regular	Nil	4years	Nil	Primary	4years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	18	21 B/L Spill+
29		Regular	Nil	8years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	19	27 B/L Spill+
30		Regular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.5	13	16 B/L Spill+
31	ram	Irregular	Nil	3years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	11	23 B/L Spill+
32	un	Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.2	17	18 B/L Spill+
33	zt	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5	14	11 B/L Spill+
34	ermenpet	Regular	Nil	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.6	13	17 B/L Spill+
35	di	Irregular	Nil	9years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	16 B/L Spill+
36	yoor	Irregular	Nil	3years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	13	16 B/L Spill+
37		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	12	21 B/L Spill+
38	gate	Irregular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	14 B/L Spill+
39	yoor	Regular	Nil	7years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	13	17 B/L Spill+
40	un	Irregular	Nil	3years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	16	21 B/L Spill+
41		Regular	Nil	5years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	17 B/L Spill+
42	ram	Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.7	17	12 Right adnexal cyst of 2*3cm
43	ermenpet	Irregular	Nil	11years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.34	15	13 B/L Spill+
44	e	Regular	Nil	7years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	11	15 B/L Spill+
45	m	Regular	Nil	3years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	13 Multiple cysts in POD
46	ermenpet	Irregular	Nil	5 years	Nil	Primary	5years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	1.9	21	18 B/L Spill+
47	un	Irregular	Nil	15years	Nil	Primary	15years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	16	17 B/L Spill+
48	ermenpet	Irregular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	13 B/L Spill+
49	ram	Regular	Congestive	9years	Nil	Secondary	9years		1 Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	22	11 Rt paraovarian simple cyst, B/L spill+
50		Irregular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	14	17 B/L Spill+
51	r	Irregular	Nil	8years	Nil	Primary	12years	Nil	Oligoterato	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	16 B/L Spill+
52	iyoor	Regular	Nil	9years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	12	17 B/L Spill+
53		Regular	Nil	13years	Nil	Primary	13years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.6	13	18 Adhesions+, Endometriotic lesions+ on anterior pelvic wall
54		Irregular	Nil	11years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	14	17 B/L Spill+
55	e	Irregular	Nil	8years	Nil	Primary	16years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	21	14 Endometriotic lesions+ on round ligament, uterine serosa
56	yoor	Irregular	Nil	12years	Nil	Primary	12 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	17	12 B/L Spill+
57	gate	Regular	Nil	4 years	Nil	Primary	4 years	Nil	Oligosperm	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	9	11 B/L spill+
58	zt	Irregular	Nil	5years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5.1	18	12 Right sided tube-patent, No spillage from left tube
59	di	Regular	Nil	3years	Nil	Primary	3 years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	2.4	17	21 B/L Spill+
60	yoor	Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	18 B/L Spill+
61	zt	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	12	11 B/L spill+
62		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	11	15 B/L Spill+
63		Irregular	Nil	7years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.9	19	21 B/L Spill+
64	iyoor	Regular	Nil	9year	Nil	Primary	9years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	6	13	17 B/L Spill+
65	zt	Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	13 B/L Spill+
66	un	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18 B/L Spill+
67	ermenpet	Irregular	Congestive	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	17	11 Endometriotic lesions+ on anterior pelvic wall
68	yoor	Regular	Nil	16years	Nil	Secondary	14years		1 Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18 B/L Spill+
69	di	Regular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.4	11	14 B/L Spill+
70	r	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	12	17 B/L spill+
71	ram	Regular	Nil	4years	Nil	Primary	4years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	18	21 B/L Spill+
72		Regular	Nil	8years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	19	27 B/L Spill+
73		Regular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.5	13	16 B/L Spill+
74	ram	Irregular	Nil	3years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	11	23 B/L Spill+
75	un	Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.2	17	18 B/L Spill+
76	zt	Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5	14	11 B/L Spill+, Endometriotic lesions+ on round ligament, uterine serosa
77	ermenpet	Regular	Nil	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.6	13	17 B/L Spill+
78	di	Irregular	Nil	9years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	16 B/L Spill+
79	yoor	Irregular	Nil	3years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	13	16 B/L Spill+
80		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	12	21 B/L Spill+
81	gate	Irregular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	14 B/L Spill+
82	yoor	Regular	Nil	7years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	13	17 B/L Spill+
83	un	Irregular	Nil	3years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	16	21 B/L Spill+
84		Regular	Nil	5years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	17 B/L Spill+
85	ram	Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.7	17	12 Right adnexal cyst of 2*3cm
86	ermenpet	Irregular	Nil	11years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.34	15	13 B/L Spill+
87	e	Regular	Nil	7years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	11	15 B/L Spill+
88	m	Regular	Nil	3years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	13 Multiple cysts in POD
89	ermenpet	Irregular	Nil	5 years	Nil	Primary	5years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	1.9	21	18 B/L Spill+
90	un	Irregular	Nil	15years	Nil	Primary	15years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	16	17 B/L Spill+
91	ermenpet	Irregular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	13 B/L Spill+
92	ram	Regular	Congestive	9years	Nil	Secondary	9years		1 Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	22	11 Rt paraovarian simple cyst, B/L spill+

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X
186	Mogala	33	12410	Elephant gate		Regular	Nil	4 years	Nil	Primary	4 years	Nil	Oligosperm	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	9	11	B/L spill+
187	Manimegali	35	13450	Tondiarpet		Irregular	Nil	5years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.1	18	12	Right sided tube-patent, No spillage from left tube
188	Nivetha	33	13740	Vyasarpadi		Regular	Nil	3years	Nil	Primary	3 years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	2.4	17	21	B/L Spill+
189	Kavivarthin	27	13674	Kodunaiyoor		Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	18	B/L Spill+
190	Pushpa	36	13798	Tondiarpet		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	12	11	B/L spill+
191	Padmavathy	29	13651	Ennavoor		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	11	15	B/L Spill+
192	Ezhilarasi	33	13210	Minjur		Irregular	Nil	7years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.9	19	21	B/L Spill+
193	Femina	35	13200	Thiruvottiyoor		Regular	Nil	9year	Nil	Primary	9years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	6	13	17	B/L Spill+
194	Malathy	28	14210	Tondiarpet		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	13	B/L Spill+
195	Camelia	33	13607	Royapuram		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18	B/L Spill+
196	Sukumari	29	13312	Old washermenpet		Irregular	Congestive	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	17	11	Endometriotic lesions+ on anterior pelvic wall
197	Kalyani	36	13015	Kodungaiyoor		Regular	Nil	16years	Nil	Secondary	14years	1	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18	B/L Spill+
198	Kanagarasy	34	13506	Vyasarpadi		Regular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.4	11	14	B/L Spill+
199	Vedhavalli	29	13971	Kolathoor		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	12	17	B/L spill+
200	Rajalakshm	32	13051	Madhavaram		Regular	Nil	4years	Nil	Primary	4years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	18	21	B/L Spill+
201	Nachiyar	34	12205	Minjur		Regular	Nil	8years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	19	27	B/L Spill+
202	Yamini	27	13541	Ponneri		Regular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.5	13	16	B/L Spill+
203	Uma mahes	31	13604	Madhavaram		Irregular	Nil	3years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	11	23	B/L Spill+
204	Mary	36	12041	Royapuram		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.2	17	18	B/L Spill+
205	Vinitha	28	13369	Tondiarpet		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5	14	11	B/L Spill+
206	Vasantha	31	13304	Old washermenpet		Regular	Nil	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.6	13	17	B/L Spill+,
207	Sunitha	36	13964	Vyasarpadi		Irregular	Nil	9years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	16	B/L Spill+
208	Nancy	27	12641	Kodungaiyoor		Irregular	Nil	3years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	13	16	B/L Spill+
209	Revathy	33	12614	Minjur		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	12	21	B/L Spill+
210	Meenakshi	29	13340	Elephant gate		Irregular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	14	B/L Spill+
211	Kanagavalli	36	13945	Kodungaiyoor		Regular	Nil	7years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	13	17	B/L Spill+
212	Parimala	28	13423	Royapuram		Irregular	Nil	3years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	16	21	B/L Spill+
213	Janani	34	14065	Ponneri		Regular	Nil	5years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	17	B/L Spill+
214	Ganga	36	12064	Madhavaram		Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.7	17	12	Right adnexal cyst of 2*3cm
215	Vinodhini	28	14864	Old washermenpet		Irregular	Nil	11years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.34	15	13	B/L Spill+
216	kamatchi	31	13064	Ennavoor		Regular	Nil	7years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	11	15	B/L Spill+
217	Kalaiselvi	27	13074	Pallavaram		Regular	Nil	3years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	13	Multiple cysts in POD, Endometriotic lesions+ on anterior pelvic wall
218	Preethi	22	13982	Old washermenpet		Irregular	Nil	5 years	Nil	Primary	5years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	1.9	21	18	B/L Spill+
219	Jalaja	22	13450	Royapuram		Irregular	Nil	15years	Nil	Primary	15years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	16	17	B/L Spill+
220	Usha	30	13074	Old washermenpet		Irregular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	13	B/L Spill+
221	Priyavarshi	27	14731	Madhavaram		Regular	Congestive	9years	Nil	Secondary	9years	1	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	22	11	Rt paraovarian simple cyst, B/L spill+
222	Sathyasri	26	14021	Ponneri		Irregular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	14	17	B/L Spill+
223	Sathya mier	22	12861	Kolathoor		Irregular	Nil	8years	Nil	Primary	12years	Nil	Oligoteratoz	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	16	B/L Spill+
224	Balalakshmi	31	13457	Thiruvottiyoor		Regular	Nil	9years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	12	17	B/L Spill+
225	Chindhuja	29	13414	Minjur		Regular	Nil	13years	Nil	Primary	13years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.6	13	18	Adhesions+, Endometriotic lesions+ on anterior pelvic wall
226	Srilakshmi	27	13458	Budhur		Irregular	Nil	11years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	14	17	B/L Spill+
227	Srileka	31	13206	Ennavoor		Irregular	Nil	8years	Nil	Primary	16years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	21	14	Endometriotic lesions+ on round ligament, uterine serosa
228	Usha	30	14614	Kodungaiyoor		Irregular	Nil	12years	Nil	Primary	12 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	17	12	B/L Spill+
229	Madhumitha	33	14916	Elephant gate		Regular	Nil	4 years	Nil	Primary	4 years	Nil	Oligosperm	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	9	11	B/L spill+
230	Chithra	35	13761	Tondiarpet		Irregular	Nil	5years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5.1	18	12	Right sided tube-patent, No spillage from left tube
231	Nivetha	33	13605	Vyasarpadi		Regular	Nil	3years	Nil	Primary	3 years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	2.4	17	21	B/L Spill+
232	Prathika	27	12741	Kodungaiyoor		Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	18	B/L Spill+
233	Lalitha	36	13075	Tondiarpet		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	12	11	B/L spill+
234	Angel selva	29	13980	Ennavoor		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	11	15	B/L Spill+
235	Maheswari	33	13642	Minjur		Irregular	Nil	7years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.9	19	21	B/L Spill+
236	Navaneetha	35	13683	Thiruvottiyoor		Regular	Nil	9year	Nil	Primary	9years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	6	13	17	B/L Spill+
237	Pravitha	28	13846	Tondiarpet		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	13	B/L Spill+
238	Ushavathi	33	12276	Royapuram		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18	B/L Spill+
239	Chellamma	29	12340	Old washermenpet		Irregular	Congestive	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	17	11	Endometriotic lesions+ on anterior pelvic wall
240	Subbul	36	14360	Kodungaiyoor		Regular	Nil	16years	Nil	Secondary	14years	1	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	18	B/L Spill+
241	Manisha	34	13456	Vyasarpadi		Regular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.4	11	14	B/L Spill+
242	Vedhavalli	29	13753	Kolathoor		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	12	17	B/L spill+
243	Manjula	32	13954	Madhavaram		Regular	Nil	4years	Nil	Primary	4years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	18	21	B/L Spill+
244	Sathyavani	34	13743	Minjur		Regular	Nil	8years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	19	27	B/L Spill+
245	Yamini	27	13961	Ponneri		Regular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.5	13	16	B/L Spill+
246	Uma mahes	31	13642	Madhavaram		Irregular	Nil	3years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	11	23	B/L Spill+
247	Mariyamma	36	13846	Royapuram		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.2	17	18	B/L Spill+
248	Vinitha	28	13927	Tondiarpet		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5	14	11	B/L Spill+
249	Keerthana	31	13264	Old washermenpet		Regular	Nil	4years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.6	13	17	B/L Spill+
250	Banumathy	36	12761	Vyasarpadi		Irregular	Nil	9years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	11	16	B/L Spill+
251	Nancy	27	12643	Kodungaiyoor		Irregular	Nil	3years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	13	16	B/L Spill+
252	Revathy	33	14301	Minjur		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	12	21	B/L Spill+
253	Nagamma	29	13940	Elephant gate		Irregular	Nil	6years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	14	B/L Spill+
254	Kanagavalli	36	12467	Kodungaiyoor		Regular	Nil	7years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	13	17	B/L Spill+
255	Chandramat	28	13766	Royapuram		Irregular	Nil	3years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	16	21	B/L Spill+
256	Janani	34	13943	Ponneri		Regular	Nil	5years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	17	B/L Spill+
257	Ganga	36	13705	Madhavaram		Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.7	17	12	Right adnexal cyst of 2*3cm
258	Vinodhini	28	14061	Old washermenpet		Irregular	Nil	11years	Nil	Primary	4 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.34	15	13	B/L Spill+
259	Parvathy sa	31	14603	Ennavoor		Regular	Nil	7years	Nil	Primary	7 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	11	15	B/L Spill+
260	Kalai	27	13042	Pallavaram		Regular	Nil	3years	Nil	Primary	3years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	13	Multiple cysts in POD, Endometriotic lesions+ on anterior pelvic wall
261	Madhavini	22	13744	Old washermenpet		Irregular	Nil	5 years	Nil	Primary	5years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	1.9	21	18	B/L Spill+
262	Rashmika	22	13994	Royapuram		Irregular	Nil	15years	Nil	Primary	15years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	16	17	B/L Spill+
263	Usha	30	13456	Old washermenpet		Irregular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.9	17	13	B/L Spill+
264	Priyanka	27	14507	Madhavaram		Regular	Congestive	9years	Nil	Secondary	9years	1	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	22	11	Rt paraovarian simple cyst, B/L spill+
265	Sathya	26	14596	Ponneri		Irregular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.3	14	17	B/L Spill+
266	Sathya mier	22	13335	Kolathoor		Irregular	Nil	8years	Nil	Primary	12years	Nil	Oligoteratoz	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	13	16	B/L Spill+
267	Balalakshmi	31	13420	Thiruvottiyoor		Regular	Nil	9years	Nil	Primary	9years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.2	12	17	B/L Spill+
268	Nandhini	29	12543	Minjur		Regular	Nil	13years	Nil	Primary	13years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	1.6	13	18	Adhesions+, Endometriotic lesions+ on anterior pelvic wall
269	Udhayachar	27	14138	Budhur		Irregular	Nil	11years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.1	14	17	B/L Spill+
270	Madhivadh	31	13402	Ennavoor		Irregular	Nil	8years	Nil	Primary	16years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.1	21	14	Endometriotic lesions+ on round ligament, uterine serosa
271	Usha	30	15284	Kodungaiyoor		Irregular	Nil	12years	Nil	Primary	12 years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	17	12	B/L Spill+
272	Parvathy	33	13641	Elephant gate		Regular	Nil	4 years	Nil	Primary	4 years	Nil	Oligosperm	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.8	9	11	B/L spill+
273	Manimegali	35	13308	Tondiarpet		Irregular	Nil	5years	Nil	Primary	5years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	5.1	18	12	Right sided tube-patent, No spillage from left tube, Endometriotic lesions+ on anterior pelvic wall
274	Nivetha	33	13552	Vyasarpadi		Regular	Nil	3years	Nil	Primary	3 years	Nil	Normal	Active	Hypothyroi	Nil	Nil	Nil	WNL	WNL	2.4	17	21	B/L Spill+
275	Manasa	27	13994	Kodungaiyoor		Irregular	Nil	6years	Nil	Primary	6years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.7	17	18	B/L Spill+
276	Lalitha	36	13477	Tondiarpet		Regular	Nil	7years	Nil	Primary	7years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	4.4	12	11	B/L spill+
277	Padmapriya	29	12964	Ennavoor		Regular	Nil	11years	Nil	Primary	11years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	3.7	11	15	B/L Spill+
278	Ezhilarasi	33	14300	Minjur		Irregular	Nil	7years	Nil	Primary	8years	Nil	Normal	Active	Nil	Nil	Nil	Nil	WNL	WNL	2.9	19	21	B/L Spill+