DOPPLER STUDY IN OVARIAN TUMOURS

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CERTIFICATE

This is to certify that the dissertation entitled "**DOPPLER STUDY IN OVARIAN TUMOURS"** is the bonafide original work of **Dr. R. SUGANTHI** in partial fulfilment of the requirements for **M.D. Branch – II (Obstetrics and Gynaecology)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in March 2008. The period of study was from August 2006 to August 2007.

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DECLARATION

I, **Dr. R. SUGANTHI,** solemnly declare that dissertation titled, "**DOPPLER STUDY IN OVARIAN TUMOURS"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2005-2008 under the guidance and supervision of my Unit Chief **Dr. ANURADHA, M.D., D.G.O.**

The dissertation is submitted to Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of **M.D. Degree (Branch – II) in Obstetrics and Gynecology.**

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(Dr. R. SUGANTHI)

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CONTENTS

INTRODUCTION

Of all gynecological cancers ovarian malignancies represents greatest clinical challenge. The pathology of ovarian neoplasm is one of the most complex area of gynaecology because, ovaries give rise to wider variety of tumors than any other organ in the body. Ovary consist of surface epithelium capable of mullerian differentiation, sex cells that are totipotent and mesenchymal cells that are multipotent. Thus ovary is complex in its embryology, histology and steroidogenesis and has the potential to develop cancer.

In India, ovarian cancers account for about 5% of all gynaecological cancer.

A women's risk at birth of having ovarian cancer in her life is 1% to 1.5% and that of dying from ovarian cancer almost 0.5%. Most epithelial ovarian cancer in sporadic, with familial hereditary pattern accounting for 5% to 10% of all malignancies. In woman with two first degree relative with documented premenopausal epithelial ovarian cancer, the risk of having an affected gene is as high a 35% to 40%.

In women with a single first degree relative and a single second degree relative with epithelial ovarian cancer, the risk is two to ten fold higher.

The tragedy of ovarian cancer is that 70% of women present with advanced disease. The 5 year survival rate for stage 1 ovarian cancer is 70%

whereas for Stage IV is 0-5%. Logically this suggest that the most productive way to achieve a better outcome from ovarian cancer would be to improve methods for earlier diagnosis rather than to develop further heroic therapies for advanced disease.

The introduction of transvaginal ultrasound has improved the ease and resolution of ovarian malignancy that is used to supplement a careful clinical examination. As Colour Doppler has become widely available, their potential value for differentiating benign from malignant tumours has increased. Malignant tumour often have neovascularity that consists of blood vessels with walls that have little or no smooth muscle support. These vessels have low pulsatility index and resistance index. Thus by using Doppler we can identify ovarian malignancy at an early stage.

AIM OF THE STUDY

- To assess the accuracy of Doppler in confirming(or) excluding the presence of ovarian malignancy.

- To correlate Doppler findings with histopathology .

REVIEW OF LITERATURE

Ovarian malignancies are difficult to diagnose clinically. At the time of clinical diagnosis, more than 60-70% are already in stage III to IV. If the gynecologist could accurately differentiate a malignant from benign mass, patients with masses believed to be malignant could seek proper oncologic consultation preoperatively and at the time of surgery the appropriate incision would allow careful staging.

Ovarian neoplasm is a complex wide spectrum of neoplasm involving variety of histological tissue varying from epithelial tissues, connective tissue, specialized hormone secreting to germinal and embryonal cells. The most common are epithelial tumours forming 80% of all tumours. 80% are benign tumours and 20% malignant. Of all malignant tumours, 90% are epithelial in origin, 80% are primary in ovary and 20% secondary from heart, GIT and colon. Benign tumours can become secondarily malignant.

The peak incidence of invasive epithelial ovarian cancer is at 56 to 60yrs of age. About 30% of ovarian neoplasm's in postmenopausal women are malignant, whereas only about 7%of ovarian epithelial tumor in premenopausal patients are malignant. The average age of patients with borderline tumors is approximately 46yrs.

Ovarian cancer has been associated with less parity and infertility. Because parity is inversely related to ovarian cancer, having at least one child is protective of the disease, with a risk reduction of 0.3% to 0.4%.

Oral contraceptive use reduces the risk of epithelial ovarian cancer. Women who use oral contraceptives for 5 or more years reduce their relative risk to 0.5.

Most epithelial ovarian cancer is sporadic with familial or hereditary patterns accounting for 5% to 10% of all malignancies. Hereditary ovarian cancers in general occur in women approximately 10years younger than those with non hereditary cancers.

Most hereditary ovarian cancer is associated with germline mutations in BRCA1 gene; a small proportion of inherited disease is associated with mutations in the gene BRCA2. The mutations are inherited in an autosomal dominant fashion, and therefore a full pedigree analysis must be carefully evaluated. The value of prophylactic salphingooophorectomy in these patients has been documented.

In the first two decades of life, almost 70% of ovarian tumours are of germ cell origin, and one third of these are malignant. In constrast to the relatively slow-growing epithelial ovarian tumours, germ cell malignancies grow rapidly.

The most common types of malignant germ cell tumours are 1, Dysgerminomas 2, immature teratoms and 3, endodermal sinus tumours.

Metastatic tumours to the ovaries are most frequently from breast and gastro intestinal tract.

WHO CLASSIFICATION OF OVARIAN TUMOURS

I) Common epithelial tumours

a. Serous tumours

b. Mucinous tumours

c. Endometrioid tumours

d. Clear cell (Mesonephroid tumours)

e. Brenner tumours

f. Mixed epithelial tumours

g. Undifferentiated carcinoma

h. Unclassified epithelial tumours

II) Sex cord (Gonadal stromal) tumours :

a. Granulosa – stromal cell tumours, theca cell tumours

b. Androblastomas : Sertoli – leydig cell tumours

c. Gynandroblastomas

d. Unclassified

III) Lipid (Lipoid) cell tumours

IV) Germ cell tumours

a. Dysgerminoma

b. Endodermal sinus tumour

c. Embryonal carcinoma

d. Polyembryoma

e. Chorio carcinoma

f. Teratoma

g. Mixed forms

V) Gonadoblastoma

a. Pure

b. Mixed with dysgerminoma or other germ cell tumours

VI) Soft tissue tumours not specific to ovary

VII) Unclassified tumours

VIII) Secondary (Metastatic) tumours

IX) Tumour –like conditions

BORDERLINE TUMOUR

Borderline tumours are tumours of low malignant potential .Most frequently occur between the ages of 30 to 50yrs.

Uncommonly metastasis may occur with borderline tumour. Such implants have been divided into noninvasive and invasive forms.

Criteria for the diagnosis of borderline tumours are as follows:

1. Epithelial hyperplasia in the form of pseudo stratification, tufting, cribriform and micropapillary architecture.
2. Nuclear atypia and increased mitotic activity.
3. Detached cell clusters.
4. Absence of destructive stromal invasion.

It should be emphasized that about 20%to 25% of borderline tumours spread beyond the ovary. The diagnosis of borderline tumor versus malignant ovarian tumour must be based on the histological features of the primary tumour.

EPITHELIAL OVARIAN CANCER

Approximately 90% of ovarian cancers are derived from tissues that come from the coelomic epithelium or mesothelium. The cells are a product of the primitive mesoderm which can undergo metaplasia. Neoplastic transformation can occur when the cells are generally predisposed to oncogenesis or exposed to an oncogenic agent or both.

SEROUS CYSTADENOMA AND CYSTADENOCARCINOMA

- Most common of cystic ovarian neoplasm.
- Constitutes 50% of all ovarian neoplasms of mucinous tumours 60% benign, 15% Borderline and 25% malignant tumours.
- Half of the case is bilateral.
- Occur in third, fourth and fifth decade of life.
- Histology resembles endosalphinx

- Serous tumours develop by invagination of the surface ovarian epithelium and are so classified because they secrete serous fluid.

Border line serous tumours:

Approximately 10%of all ovarian serous tumours fall in to the category of a tumour of low malignant potential and 50% occur before the age of 40yrs.

10% of women with ovarian serous borderline tumours have extra ovarian implants.

In malignant serous tumours, stromal invasion is present.

Laminated, calcified psammoma bodies are found in 80% of serous carcinomas.

Serous psammoma carcinoma is a rare variant of serous carcinoma characterized by massive psammoma body formation and low grade cytological features.

MUCINOUS TUMOURS

- Can grow to large size.
- Represent 8 to 10 % of epithelial tumours.
- Essentially benign. 5% to 10% become malignant.
- Usually unilateral, 5% are bilateral.
- Occur in women between 30 to 60 years.
- Histology resemble endocervix.

- These cystic ovarian tumours have mucin secreting epithelium.
- Borderline Mucinous tumor:
- The mucinous tumours of low malignant potential is a diagnosis difficult to make.

Frequently, well differentiated mucinous epithelium may be seen immediately adjacent to a poorly differentiated focus. Therefore, it is important to make multiple sections from many areas in the mucinous tumour to identify the most malignant alteration.

Pseudomyxoma Peritonei:

It is clinical term used to describe the finding of abundant mucoid or gelatinous material in the pelvis and abdominal cavity surrounded by fibrous tissue.

ENDOMETRIOID TUMOURS

- Accounts for 6-8% of epithelial tumours.
- Histology resembles endometrium.
- Borderline endometrioid tumour of low malignant potential has a wide morphologic spectrum. Tumour may resemble an endometrial polyp or complex endometrial hyperplasia with glandular crowding.
- Multifocal disease – Endometrioid carcinoma of the ovary is associated in 15% to 20% of the cases with carcinoma of the endometrium.

CLEAR CELL TUMOURS

- Less than 1% of epithelial lesions.
- Highly malignant.
- Histology made of CLEAR CELL AND HOB NAIL CELLS.
- Focal areas of endometriosis and endometrioid carcinoma sometimes occur.

- Almost invariably high grade (Grade 3) nuclei are identified. Hence, clear cell carcinoma is not graded.

BRENNER TUMOURS

- Less than 1% of epithelial tumours.
- Rarely becomes malignant.
- Characteristic histology – WALTHARD CELL NEST.

NONEPITHELIAL OVARIAN CANCERS

Nonepithelial malignancies of the ovary account for about 10% of all ovarian cancers. Nonepithelial ovarian cancers include malignancies of germ cell origin, sex-cord stromal cell origin, metastatic carcinomas to the ovary and a variety of extremely rare ovarian carcinoma (eg. sarcoma lipoid cell tumours).

GERM CELL TUMOURS

Germ cell tumours derived from the primordial germ cells of the ovary.

Although 20% to 25% of all benign and malignant ovarian neoplasms are of germ cell origin, only about 3% of these tumours are malignant.

In the first two decades of life, almost 70% of ovarian tumours are of germ cell origin, and one third of these are malignant.

DYSGERMINOMA

- Mot common malignant germ cell tumour – 30% - 40% of ovarian cancers of germ cell origin.
- Dysgerminoma represent only 1% to 3% of all ovarian cancers, but they represent 5% to 10% of ovarian cancers in patients younger than 20yrs.
- 75% of cases occur between 10 & 30 years.
- Malignancy rate 30-50%.
- 20 to 30% ovarian malignancy associated with pregnancy are dysgerminoma.
- Dysgerminoma are found in both sexes and may arise in gonadal or extragonadal sites.

Approximately 5% of dysgerminomas are discovered in phenotypic women with abnormal gonads. This malignancy can be associated with patients who have pure gonadal dysgenesis, mixed gonadal dysgenesis and the androgen insensitivity syndrome. Therefore for premenarcheal patients with a pelvic mass, the karyotype should be determined.

Dysgerminoma is bilateral in 10-15% of cases. Dysgerminoma is the only germ cell malignancy that has this significant rate of bilaterality.

ENDODERMAL SINUS TUMOUR

- Also known as Yolk Sac carcinoma because they are derived from primitive yolk sac.
- Third frequent malignant germ cell tumour.
- Median age 16-18 yrs, $1/3^{rd}$ of patients are premenarcheal.
- 100% unilateral.
- Characteristic histology – SCHILLER DUVAL BODY.

Abdominal or pelvic pain is the most frequent initial symptom, occuring in about 75% of patients, whereas an asymptomatic pelvic mass is documented in 10% of patients.

Endodermal sinus tumour associated with gonadal dysgenesis and so chromosomal analysis should be performed preoperatively.

Most Endodermal sinus tumour lesions secrete Alpha Fetoprotein and rarely may elaborate α_1 Antitrypsin.

EMBRYONAL CARCINOMA

- Extremely rare tumour.
- Age : 4-28 yrs.

- Embryonal carcinoma distinguished from a choriocarcinoma of the ovary by the absence of syncytiotrophoblastic and cytotrophoblatic cells.

- Embryonal carcinoma may secrete estrogens. Patient exhibits symptoms and signs of precocious pseudopuberty or irregular bleeding.

- Embryonal carcinoma frequently secrete Alpha fetoprotein and Human chorionic gonadotrophin.

POLYEMBRYOMA

- Extremely rare tumour

- Occur in very young, premenarcheal girls

- Characteristic histology : EMBRYOID BODIES

CHORIOCARCINOMA

- Extremely rare.

- Most occur in patients younger than 20 yrs.

IMMATURE TERATOMA

- Accounts for <1% of all ovarian cancer.

- 2^{nd} most common germ cell malignancy.

- 50% occur in women between 10 and 20 yrs.

- Immature teratomas contain elements that resemble tissue derived from the embryo.

- The prognosis can be correlated with the grade determined by the quantity of the immature neural elements. With a higher grade there is a poorer prognosis.
- Malignant changes in benign cystic teratomas has been recorded as occurring in 0.5 to 2% of cases, usually in patients older than 40 years of age.

MIXED GERM CELL TUMOUR

- Contain two or more elements of lesion of above described germ cell types.

- Most frequent combination : dysgerminoma and endodermal sinus tumours.

- The most important prognostic features are the size of the primary tumour and the relative size of its most malignant component.

GRANULOSA CELL TUMOUR

- Granulosa stromal cell tumours include granulosa cell tumours, thecomas and fibromas.

- Only in 2% bilateral.

- Found in women throughout reproductive and postmenopausal years. Found in prepubertal in 5% of cases.

- Secrete estrogen.

- Characteristic histology CALL EXNER BODIES-

Granulosa cells show a tendency to arrange themselves in small clusters or rosettes around a central cavity, so there is a resemblance to primordial follicle. In prepubertal lesions, 75% are associated with sexual pseudoprecocity because of estrogen secretion.

SEX CORD STROMAL TUMOURS

- Sex- cord stromal tumours of the ovary account for about 5% to 8% of all ovarian malignancies .

- Sex cord stromal tumours are derived from the sex cords and the ovarian stroma or mesenchyme.

SERTOLI LEYDIG CELL TUMOUR

- Occur in 3^{rd} & 4^{th} decade of life.

- Extremely rare and account for less than 0.2% of ovarian cancer.

- Produce androgen – clinical virilization in 70% to 85% of patient.

- <1% Bilaterality

METASTATIC TUMOURS

About 5% to6% of ovarian tumors are metastatic from the organs most frequently from the female genital tract, the breast or the gastrointestinal tract. The metastases may occur from direct extension of another pelvic neoplasm, by hematogenous or lymphatic spread or by transcoelomic dissemination ,with surface implantation of tumor that spread in the peritoneal cavity.

KRUKENBERG TUMOUR

- Krukenberg tumour account for 30% to 40% of metastatic cancer to ovaries.
- Primary frequently in stomach less commonly in colon, breast or biliary tract.
- Usually bilateral.
- Characteristic histology : SIGNET RING CELLS.

PROGNOSTIC VARIABLES IN EARLY STAGE EPITHELIAL OVARIAN CANCER

LOW RISK	HIGH RISK
Low grade	High grade
Non clear histologic type	Clear cell Histologic type
Intact capsule	tumor growth through capsule
No surface excrescences	Surface excrescences
No ascites	ascites
Negative peritoneal cytologic findings	Malignant cells in fluid
Unruptured or intraoperative rupture	Preoperative rupture
No Dense adherence	Dense adherence
Diploid Tumor	Aneuploid

FIGO STAGING OF OVARIAN CARCINOMA

STAGE I : Tumour restricted to one or both ovaries.

1A : Tumour restricted to one ovary. No tumour on external surface, capsule intact, no malignant ascites.

1B : Tumour restricted to both ovaries. No tumour on external surface, capsule intact, no malignant ascites.

IC : Tumour IA or IB, but with tumour on the surface of one or both ovaries or with capsule ruptured, or with malignant ascites or positive peritoneal washings.

STAGE II : Tumour involves one or both ovaries with pelvic extension.

IIA : Extension and/or Metastasis to the uterus and / or fallopian tubes. No malignant cells in ascites / washings.

IIB : Extension to other pelvic organs. No malignant cells in ascites or washings.

IIC : Tumour IIA or IIB with surface growth; or with capsule ruptured at /or prior to surgery; or with malignant ascites or with positive peritoneal washings.

STAGE III : Tumour involving one or both ovaries, with microscopic implants outside the pelvis and / or positive nodes (inguinal, retroperitoneal). Tumour limited to true pelvis but with histological evidence of spread to bowel, omentum, presence of superficial metastases on the liver.

IIIA : Tumour grossly limited to the pelvis, nodes negative, but microscopic seeding of peritoneum.

IIIB : Tumour with abdominal peritoneal implants of less than 2cm size and nodes negative.

IIIC : Peritoneal implants of more than 2 cm size and / or positive nodes.

STAGE IV : Growth involving one or both ovaries with distant metastases in liver, lungs and pleura. If pleural effusion is present, there must be positive cytologic test results to allot a case to stage IV. Parenchymal liver metastasis equals to stage IV.

COMPARISON OF FIGO STAGING AND 5YRS SURVIVAL

Stage 0	-	90-100%
Stage 1	-	70%
Stage 2	-	25-30%
Stage 3	-	10%
Stage 4	-	0-5%

GENETIC RISK FOR EPITHELIAL OVARIAN CANCER

The risk of ovarian cancer is higher than that in the general population in women with certain family histories. Most epithelial ovarian cancer is sporadic, with familial or hereditary patterns accounting for 5% to 10% of all malignancies.

ROLE OF CA 125

Ca 125 is a glycoprotein present in 80% of non mucinous tumour. Ca 125 is also increased in inflammatory condition of the abdomen, liver disease,

recent surgery and benign gynaecologic conditions such as fibroids, endometriosis, ectopic pregnancy or a ruptured cyst.

Ca 125 is used during course of diagnosis, treatment and follow up of ovarian and other closely related cancers, such as primary peritoneal and fallopian tube cancer.

Normal value ranges from 0 to 35 U/ml. Using 35 as cut off value, identifies 50% of stage I ovarian cancer but 80% of stage II,III and IV

HEREDITARY OVARIAN CANCER

BRCA1and BRCA2

Most hereditary ovarian cancer is associated with mutations in the BRCA1 gene, located on chromosome 17. A small proportion of inherited disease is associated with germ line mutations in another gene, BRCA2, located on chromosome 13.

There is a higher- than- expected risk of ovarian and endometrial cancer in the Lynch II syndrome, known also as the hereditary nonpolyposis colorectal cancer syndrome (HNPCC syndrome).

The mutations are inherited in an autosomal dominant fashion and therefore a full pedigree analysis (i.e., both maternal and paternal sides of the family) must be carefully evaluated.

Based on analysis of women who have a mutation in the BRCA1 gene and are from high-risk families, the lifetime risk of ovarian cancer may be as high as 28% to 44% , and the risk has been calculated to be as high as 27% for those women with BRCA2 mutation. The risk of breast cancer in women with a BRCA1or BRCA2 mutation may be as high as 56% to 87%.

Hereditary ovarian cancers in general occur in women approximately 10years younger than those with nonhereditary tumours.

PEDIGREE ANALYSIS

The risk of carrying a germ line mutation that predisposes to ovarian cancer depends on the number of first-and/or second- degree relatives (or both) with a history of epithelial ovarian carcinoma or breast cancer (or both) , and on the number of malignancies that occur at an earlier age. The degree of risk is difficult to determine precisely unless a full pedigree analysis is performed.

- In families with two first- degree relatives(i.e., mother, sister, or daughter) with documented premenopausal epithelial ovarian cancer, the risk that a female first- degree relative has an affected gene could be as high as 35% to 45%.

- In families with a single first-degree relative and a single second-degree relative with epithelial ovarian cancer, the risk that a woman has an affected

gene also may be increased. The risk may be two to 10 fold higher than in those without a familial history of the disease.

- In families with a single postmenopausal first-degree relative with epithelial ovarian carcinoma, a women may not have an increased risk of having an affected gene because the case is most likely to be sporadic. However, if the ovarian cancer occurs in a premenopausal relative, this could be significant and a full pedigree analysis should be undertaken.

- Women with a primary history of breast cancer have twice the expected incidence of subsequent ovarian cancer.

HEREDITARY NONPOLYPOSIS COLON CANCER, OR LYNCH II SYNDROME

HNPCC syndrome, which includes multiple adenocarcinomas involves a combination of familial colon cancer (known as the lynch I syndrome); a high rate of ovarian endometrial, breast cancers and other malignancies of the gastrointestinal and gentiourinary systems. The mutations that have been associated with this syndrome are MSH2, MLH1, PMS1,and PMS2.

CLINICAL FEATURES OF BENIGN TUMOURS

Symptoms

Most tumors are asymptomatic. These are detected accidentally. However the patient may present the following symptoms.

1. Heaviness, in the lower abdomen.

2. A gradually increasing mass in lower abdomen.

3. Dull aching pain in lower abdomen.

4. In neglected cases, the tumour may be big enough to fill whole of the abdomen. It then produces cardio respiratory embarrassment or gastrointestinal symptoms like nausea or indigestion.

5. Menstrual pattern remains unaffected unless associated with hormone producing tumours menorrhagia or postmenopausal bleeding or precocious puberty in feminizing tumour.

Signs

- General condition remains unaffected, However, in huge mucinous cystadenoma, the patient may be cachectic due to protein loss .

- Pitting odema of legs may be present when a huge tumour presses on the great veins.

ABDOMINAL EXAMINATION

An ovarian tumour which is enlarged sufficiently so as to occupy the lower abdomen presents with the following.

INSPECTION

There is a mass of the lower abdomen over which the abdominal wall moves freely with respiration. The mass may be placed centrally or in one side . At times, the mass fills the entire abdominal cavity. The flanks remain flat .

PALPATION

- Feel is cystic or tense cystic. Benign solid tumours such as fibroma, thecoma, Brenner tumour are rare.
- Freely mobile from side to side but restricted from above down wards unless the pedicle is long. Too big a tumour or adhesions make its mobility restricted.
- Upper and lateral borders are well defined but the lower pole is difficult to reach suggestive of pelvic origin . However, with long pedicle, the tumor may be displaced upwards so as to reach the lower pole.
- It is usually not tender.

PERCUSSION

Percussion note is dull in the centre and resonant in the flanks . A fluid thrill may be elicited when the walls are thin and the content is watery. Co-

existing ascites may be present even in a benign solid tumour (fibroma) and is called Meigs syndrome.

MEIGS SYNDROME

Ascites and right side hydrothorax in association with fibroma of the ovary, Brenner,thecoma and granulosa cell tumour is called Meigs syndrome. Ascites and hydrothorax when present in conditions other than those mentioned are called pseudo-Meigs syndrome.

AUSCULATION

A friction rub ,may be present over the tumour .

PELVIC EXAMINATION

Bimanual examination reveals

- The uterus is separated from the mass.
- A groove is felt between the uterus and the mass.
- Movement of the mass per abdomen fails to move the cervix.
- On elevation of the mass per abdomen , the cervix remains in stationary position.

The lower pole of the cyst can be felt through the fornix.

CLINICAL FEATURES OF OVARIAN CANCER

Symptoms

The majority of women with epithelial ovarian cancer have vague and nonspecific symptoms. In early- stage disease, the patient may experience irregular menses if she is premenopausal. If a pelvic mass is compressing the bladder or rectum, she may report urinary frequency or constipation, occasionally, she may perceive lower abdominal distention, pressure, or pain such as dyspareunia. Acute symptoms, such as pain secondary to rupture or torsion are unusual.

In advanced- stage disease , patients most often have symptoms related to the presence of ascites , omental metastases or bowel metastases. The symptoms include abdominal distention, bloating, constipation, nausea, anorexia , or early satiety. Premenopausal women may report irregular or heavy menses, whereas vaginal bleeding may occur in post-menopausal women.

Signs

The most important sign of epithelial ovarian cancer is the presence of pelvic mass on physical examination. A solid, irregular, fixed pelvic mass is highly suggestive of an ovarian malignancy. If, in addition, ascites is present, the diagnosis of ovarian cancer is almost certain.

The evaluation for adnexal masses include history, physical examination, USG and Doppler.

Doppler study were based on discovery of natural phenomenon "Doppler effect" which dates back to 20th century. The phenomenon bears name of its discoverer Christian Andrews Doppler, an Australian mathematician and Physicist. Another critical event was discovery of piezoelectric phenomenon by Pierre curie, Jacques curie which enabled the development of ultrasonic transducers many decades later.

PHYSICAL PRINCIPLE OF DOPPLER ULTRASONOGRAPHY

Sound is a form of mechanical energy that travels through solid or liquid media as pressure waves. Sound waves are generated when a object vibrates in a medium. Sound waves from a vibratory source or from a reflector move across surfaces of high or low pressure. These surfaces are called waveforms. The shape of waveform depends on the shape of the source or the interface. With Doppler ultrasonics, the scattered waveform is spherical as RBC's behave as spherical sources during the scattering of an incident beam.

The propagation of sound in a medium is the rate of change of position of the sound wave in unit time in that medium. It is called velocity when the direction of motion is also specified. The frequency of sound is the number of cycles occurring in one second. One cycle is called a hertz (Hz).

Audible sound frequency ranges from approximately 10Hz to 20KHz. Sound with a frequency of more than 20Khz inaudible to the human ear and is

known as ultrasound. With Doppler ultrasound used for medical diagnosis the commonly employed frequency range is 2-10 Mhz.

To produce oscillation or vibrations at the rate of millions of cycle per second, special material with piezoelectric properties are used. These piezoelectric elements are solid non-conducting substances that demonstrate physical properties whose measurements are different along different axis. When compressed in certain direction, these elements undergo electric polarization and a corresponding voltage is generated that is proportional to the pressure. Conversely when such a element is subjected to an electric field it exhibits mechanical distortion by an amount proportional to the applied field. This phenomenon is known as piezoelectric effect and allows interconversion between sound and electricity and form the basis for the construction of Doppler and other types of ultrasound transducers.

DOPPLER STUDIES

Doppler velocimetry is a nonvasive technology that uses high frequency sound waves for the investigations of blood flow. It yields a wide spectrum of hemodynamic information. Doppler study is based on "Doppler Effect"[1]

Doppler effect is as follows ,

"When a high frequency sound wave (Ultrasound) is directed toward a moving target, the back scattered sound wave will have a different frequency than the emitted sound. The magnitude of this frequency shift is proportional to the velocity of the moving target from which it is reflected. When an ultrasound beam is directed towards a blood vessel, the sound wave is mainly reflected by red blood cells, which flow in it. This is the basis for the use of Doppler technology in the assessment of blood flow parameters.

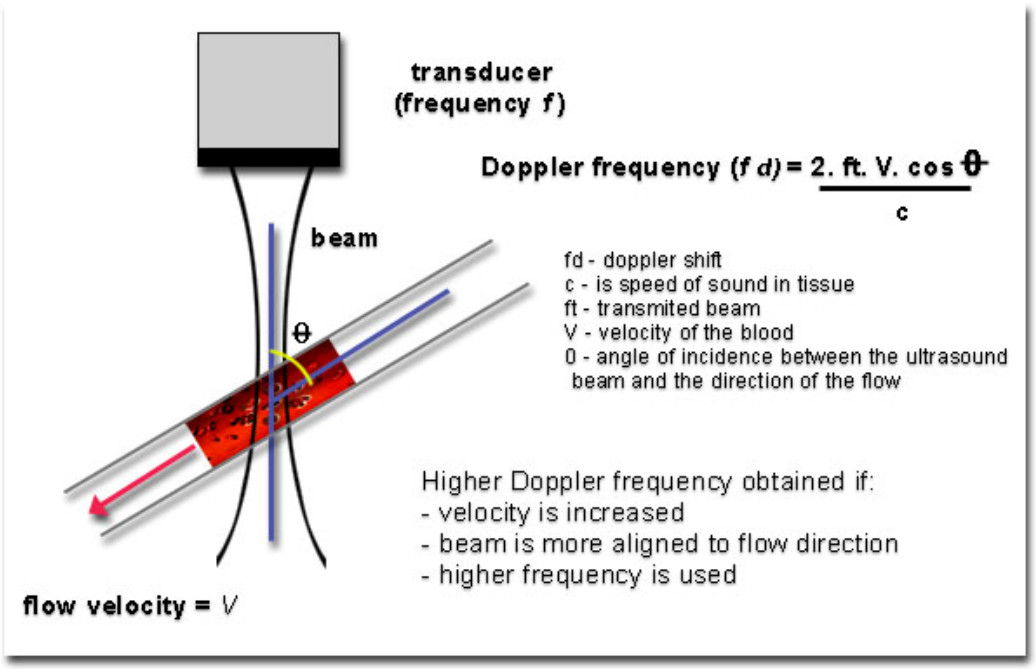
In Doppler colour flow mapping, two dimensional flow patterns are superimposed on anatomic images in real time. The flow patterns are derived from the mean frequency shift using signal processing technique. Flow towards transducer is red and flow away from it is blue.

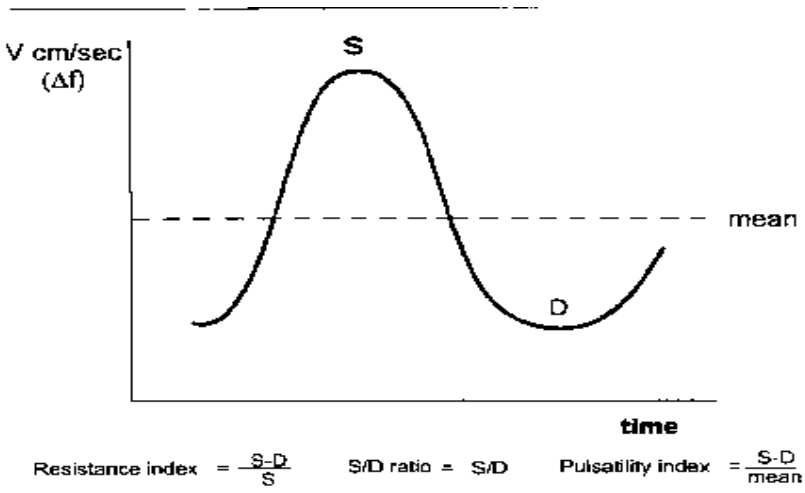
Doppler is generally used in two ways to estimate circulatory hemodynamics.

1) Direct measurement of volume of blood flow

2) Indirect measurement of flow velocity using waveform analysis.

The relationship between blood flow velocity and the Doppler frequency shift is determined by a complex interplay of multiple factors.


transducer
(frequency f)
Doppler frequency (f d) = 2. ft. V. cos θ / c
beam
θ
fd - doppler shift
c - is speed of sound in tissue
ft - transmited beam
V - velocity of the blood
0 - angle of incidence between the ultrasound beam and the direction of the flow
Higher Doppler frequency obtained if:
- velocity is increased
- beam is more aligned to flow direction
- higher frequency is used
flow velocity = V


V cm/sec
(Δf)
S
D
mean
time
Resistance index = S-D / S
S/D ratio = S/D
Pulsatility index = S-D / mean

The following velocity indices were measured

$$\text{Resistance index (R1)} = \frac{\text{Peak systolic velocity – end diastolic velocity}}{\text{Peak systolic Velocity}}$$

Pourcelol (1974) First reported this index and also called pourcelol index

$$\text{Pulsatility Index} = \frac{\text{Peak systolic velocity – End diastolic velocity}}{\text{Mean Velocity}}$$

Gosling & King (1976) Were first to putforth pulsatility index.

Pulsatility index is a measure of the impedance to blood flow, a low value indicating decreased impedance and a high value increased impedance to blood flow.

Experimental studies suggest that the development of new blood vessel is necessary to sustain the growth, invasion and metastasis of tumours. Induction of angiogenesis is mediated by a number of angiogenic peptides such as vascular endothelial growth factor and platelet derived endothelial cell growth factors. These two factors are associated with an increase in microvessel density. The newly formed vessels are large capillaries or sinusoids and do not contain smooth muscle in their walls but only fibrous connective tissue. Their basement membrane is markedly reduced and their permeability is increased. For these reasons, the neoangiogeneic vessels have low impedance as reflected by blood flow parameters.

The first medical application of Doppler sonography were initiated during the late 1950. Shigeo satomura from the Institute of scientific and industrial research of Osaka University in Japan developed the first Doppler ultrasound for Medical Diagnostic purpose.

Sonography for ovarian screening was first proposed by Campbell *et al*[3,4]. These investigators noticed a high correlation co-efficient between ovarian volumes calculated with abdominal sonography and the real volume calculated after oophorectomy.[3,4] Studies comparing abdominal and transvaginal sonography concluded that the transvaginal sonography provided better information regarding ovarian morphological characteristics and size because of the higher frequency probes used and their proximity to the gonad.[5] When compared with macroscopic examination of a tumour, transvaginal sonography was found to characterize tumour morphologic characteristics, correctly in 96% of cases.[6]

Since 1980, Colour Doppler sonography has been used as an additional tool for differential diagnosis of ovarian tumours because this diagnostic techniques provides indirect information on metabolism and direct information on the vascular anatomy of the organ.

Bourne *et al*[7] (1989) published their novel finding that they could differentiate between primary ovarian cancer and many benign pelvic masses with transvaginal colour flow ultrasonography. Evidence of neovascularisation

was sought in the masses and the vascular permeability index was calculated. Lack of neovascularisation and a high pulsatility index were able to exclude the presence of invasive primary ovarian cancer. Folkman *et al*[8](1989)had shown that angiogenic activity precedes the development of tumour in hyperplastic pancreatic islets in transgenic mice.

Kurjak *et al*[9] (1992)calculated prospectively the resistance index for 743 (74%) of 1000 postmenopausal women. The sensitivity and specificity of the resistance index in distinguishing benign masses from malignant were 96% and 95% respectively; the positive predictive value and negative predictive value were also 96% and 95% respectively.

Kurjak *et al*[10] (1993)described 18 cases of stage 1 ovarian cancer detected by transvaginal sonography using colour Doppler imaging, 2 of which had a normal B mode appearance.

Weiner *et al*[11](1992) concluded that use of Doppler technology may reduce the rate of false positive results seen with transvaginal sonography or transabdominal sonography.

Zanetta *et al*[12] (2002)reported a 77 year old woman who had normal shaped ovaries with low resistance blood flow. Although there was suspicion of cancer, no surgical procedure was performed and patient was followed up with serial measurements of CA 125. One year later, pelvic sonography revealed an enlarged ovary and an increase in CA 125 level .Laporotomy was

then carried out, and the intraoperative findings were compatible with stage III ovarian Cancer.

Daskalakis *et al*[13] (2004)used resistance index for evaluating character of the tumour and found the sensitivity, specificity, positive predictive value and negative predictive value to be 87.5%, 91.2%, 70% and 96.9% respectively.

Fleischer *et al*[14] (1996)concluded that transvaginal colour Doppler ultrasonography is capable of early detection of ovarian carcinoma. An improved detection rate may be realised with better identification of high risk patients who should be studied with transvaginal colour Doppler ultrasonography.

Rampone *etal*[15] (2001)concluded that colour Doppler ultrasonography is a valid method of screening at the first level as it is non invasive, painless and therefore well accepted by the patient.

From these studies it is clear that transvaginal Doppler waveforms demonstrate high diastolic flow or low resistance patterns in case of ovarian malignancy. This is due to the decreased muscle contractility of the newly formed blood vessels which leads to an increased flow in diastole and increased arteriovenous shunting. New advances are providing evidence that transvaginal Doppler ultrasound is a new and potentially adjuvant technique to

differentiate benign from malignant ovarian tumours, particularly those tumours that possess ultrasonographic features suggestive of malignancy.

RECOMMENDATIONS

Current recommendations for management of women at high risk for ovarian cancer are summarized as follows.

1. Women who appear to be high risk for ovarian or breast cancer should undergo genetic counseling and, if the risk appears to be substantial, may be offered genetic testing for BRCA1 and BRCA2.

2. Oral contraceptives should be recommended to young women before they embark on an attempt to have a family.

3. Women who don't wish to maintain their fertility or who have completed their families should be recommended to undergo prophylactic bilateral salphingo- oophorectomy. The risk should be clearly documented, preferably established by BRCA1and BRCA2testing, before oophorectomy is performed . This women should be counselled that this operation does not offer absolute protection, because peritoneal carcinomas occasionally can occur after bilateral oophorectomy.

4. In women who also have a strong family history of breast or ovarian cancer, annual mammography screening should be performed beginning at age 30years.

5. Women with a documented HNPCC syndrome should be treated as above but in addition, they should undergo periodic screening mammography, colonoscopy, and endometrial biopsy.

NEWER APPROACHES

A new approach is the use of proteomic patterns to identify ovarian cancer using surface- enhanced laser desorption ionization time-of –flight (SELDI –TOF)Technology. In a study using this technology, the sensitivity for predicting ovarian cancer was 100% with a specificity of 95% and a positive predictive value of 94%. This techonology is in early phases of development and validation and its efficacy has yet to be demonstrated in large population – based studies.

Another new approach is the measurement of plasma DNA levels and allelic imbalance by a technique known as digital single nucleotide polymorphism (SNP) analysis. In a study by Chang *et al*[55] (2002)this analysis had a 87% positive correlation in stages I and II patients, and a 95% correlation in patients with stages III and IV disease.

MATERIALS AND METHODS

Study Design

Prospective study

Period of Study

August 2006 – August 2007

Place of Study

Govt. RSRM lying-in Hospital, Chennai.

Case Selection

50 cases of ovarian tumours who got admitted at Government RSRM Hospital, Chennai – 13 were recruited in the study.

Inclusion Criteria

Age 14-70 yrs

Premenarcheal ovarian tumour > 2cm

Premenopausal > 6cm size of tumour

Postmenopausal >8.8ml volume of ovary.

Exclusion Criteria

Age <1 4 years or > 35 years

Premenopausal < 6cm size of tumour

Methods of Study

Besides routine investigations all the patients were subjected to ultrasonography. Doppler was done prior to surgery. Doppler signals were obtained within the whole tumour area, including the capsule of the tumour. Vasularisation was evaluated as absent if no Doppler frequency shift was found in the tumour.

From the peak systolic velocity and end diastolic velocity, pulsatility index and resistance index were computed electronically.

$$\text{Resistance index} = \frac{\text{Peak systolic velocity} - \text{End diastolic velocity}}{\text{Peak systolic velocity}}$$

$$\text{Pulsatility index} = \frac{\text{Peak systolic velocity} - \text{End diastolic velocity}}{\text{Mean velocity}}$$

Laporotomy was done. All the materials removed after Laporotomy was subjected to histopathological examination. Postoperative histopathological picture was correlated with Doppler and diagnostic accuracy was evaluated.

Sensitivity, specificity, positive predictive value and negative predictive value were calculated.

RESULTS

Patients age in the study group ranged from 14-67 years. Out of 50 patients studied. 10 were nulliparous and 9 were postmenopausal.

In the study group 13 patients were diagnosed as having malignant tumour and 36 cases were diagnosed as having benign tumours by Doppler. One patient had resistance index of 0.47 and pulsatility index of 0.67, histopathology showed borderline tumour.

Out of 50 cases included in study, one patient turned out to have borderline tumour by histopathology. Out of the remaining 49 cases 12 cases turned out to be malignant and 37 cases as benign by histopathology.

TABLE – 1

HPE / **Doppler**	**Malignant**	**Benign**
Malignant	11	2
Benign	1	35

True positive	:	11
True negative	:	35
False positive	:	2
False negative	:	1
Sensitivity	:	91.7%
Specificity	:	94.6%
Positive predictive value	:	84.6%
Negative predictive value	:	97.2%

TABLE - 2

HISTOPATHOLOGICAL DIAGNOSIS

	Benign Tumours	**No.**
1)	Serous cystadenoma	24
2)	Mucinous cystadenoma	8
3)	Benign cystic teratoma	4
4)	Chocolate cyst	1
	Malignant tumours	
1)	Serous cystadenocarcinoma	7
2)	Mucinous cystadenocarcinoma	1
3)	Granulosa cell tumour	1
4)	Yolk Sac Tumour	1
5)	Dysgerminoma	1
6)	Krukenberg	1
	Borderline	1

TABLE - 3

AGE DISTRIBUTION OF BENIGN & MALIGNANT OVARIAN TUMOURS

Age	Benign		Malignant		Type of Malignancy
	No.	%	No.	%	
< 20 yrs	3	6.1%	2	4.1%	Yolk Sac tumour, Dysgerminoma,
21-30 yrs	14	29%	1	2%	Krukenberg Tumour
31-40 yrs	7	14.3%	2	4.1%	Granulosa cell tumour Serous papillary cystadenocarcinoma
41-50 yrs	10	20%	1	2%	Serous papillary cystadenocarcinoma
Above 50 yrs	3	6.1%	6	12.2%	(5) serous cystadenocarcinoma (1) Mucinous cystadenocarcinoma

Most common benign tumour – serous cystadenoma.

Most common malignant tumours – serous cystadenocarcinoma.

INTERPRETATION OF BENIGN LESIONS

The criteria used for diagnosing benign tumour using resistance index and pulsatility index were > 0.42 and > 1 respectively in my study. When resistance index and pulsatility index were >0.42 and >1 respectively, Doppler is of high resistance flow.

The criteria used for diagnosing malignant tumour using resistance index and pulsatility index were $\leq$ 0.42 and $\leq$ 1 respectively in my study. When resistance index and pulsatility index were $\leq$ 0.42 and $\leq$1 respectively, doppler is of low resistance flow.

Tumour with no vascularity are included in benign tumour.

TABLE - 4.

Age Nature of Tumour	Benign	Malignant
<50yrs	34	6
>50yrs	3	6

P= <0.0001

Malignant tumour is more common in >50yrs of age

Table - 5

BENIGN OVARIANS TUMOURS

Type of Benign tumors	Percentages
Benign Serous cytadenoma	65%
Mucinous cystadenoma	21%
Dermoid Tumours	11%
Chocolate cyst	3%

Benign serous cystadenoma is the most common benign tumour followed by Mucinous.

TABLE - 6

MALIGNANT OVARIAN TUMOURS

Malignant ovarian tumors	Percentages
Serous cystadesocaicinoma	58.3%
Mucinous cystadenocarcinoma	8.3%
Granulsa cell tumour	8.3%
Yolksac tumour	8.3%
Dysyerminoma	8.3%
Krukenberg	8.3%

Serous cystadenocarcinoma is the most common malignant tumour

TABLE - 7

STAGING OF OVARIAN CANCER

Stage of Ovarian cases	Percentages
Stage 1	8%
Stage 2	17%
Stage 3	50%
Stage 4	25%

Most of ovarian tumours are in advanced stage. 50% of cases in stage 3.

TABLE - 8.

TYPES OF OVARIAN TUMOUR

Types of tumour	Percentages
Benign	74%
Malignant	24%
Border line	2%

Malignant tumour constitutes roughly ¼ th of ovarian tumours

TABLE - 9

COMPLAINTS & NUMBER OF PATIENTS.

Complaints	Number of patients.
Pain Abdomen	38
Abdomen Distension	16
Menstrual disturbance	8
Vomiting	3
Pressure Symptom	1

DISCUSSION

The incidence of ovarian tumour 4.4%.

The sensitivity, specificity, positive predictive value and negative predictive value of Doppler in diagnosing ovarian was 91.7%, 94.6%, 84.6% and 97.2% respectively in my study. This was similar to the study by Daskalakis et al, 2004.

COMPARISON OF DOPPLER STUDY IN OVARIAN TUMOUR

	Sensitivity	**Specificity**	**PPV**	**NPV**
Weiner et al (1992)[11]	94%	97%		
Sawicki et al (1995)[17]	100%	94%	85%	100%
Merce et al (1998)[18]	80%	66.7%	33%	94%
Fleischer et al (1991)[19]			100%	73%
Daskalakis et al 2004)[13]	87.5%	91.2%	70%	96.9%
Alcazar et al (2001)[20]	88%	91%	79%	95%
Zanetta et al (1994)21	85%	91%	89%	
Timor Trisch et al (1993)[22]	94%	99%	94%	
Krzysztof et al (2003)[23]	100%	61.3%	69.2%	
Our study (2007)	91.7%	94.6%	84.6%	97.2%

1) **Mean RI & PI**

Mean R1 & PI for benign tumours 0.69 & 3.325.

Mean R1 & PI for malignant tumours 0.39 & 0.80

2) **Age Distribution**

Patients age in the study group ranged from 14-67 yrs.

Both Germ cell tumour occurred in < 20 yrs.

Most epithelial tumour occurred in > 50 yrs (Table 3)

Most of ovarian malignancy occurred in above 50 yrs age group with a probability of < 0.0001(Table 4)

3) **Type of Tumour**

Most common Benign tumour – serous cystadenoma (Table 5)

Malignant tumour – serous cystadeno carcinoma(Table 6)

Benign and malignant epithelial tumour constitutes 80% of which benign and malignant serous tumour constitutes 77.5%.

Benign ovarian tumour constitutes 74%, Malignant tumours 24% and borderline tumour 2%.(Table 8)

Staging of Ovarian Cancer (Table 7)

Most of the cases included in my study were in advanced stage of cancer.

Stage 1-8%

Stage 2-17%

Stage 3-50%

Stage 4 – 25%

This may be due to asymptomatic nature of disease.

Complaints : (Table. 9)

Most common complaints – abdominal pain .

- Prediction of histological type is not possible with Doppler
- To improve diagnostic accuracy of Doppler ultasonography it can be combined with CT and MRI.
- Main advantage of Doppler is its low cost and absence of radiation when compared with CT. It can therefore be used safely in all cases.
- Main disadvantage is that it is operator dependent and depends on the operator's expertise as well as the resolution of the device used.
- It narrows the wider differential diagnosis of a pelvis mass by differentiating uterine from extrauterine causes and it also predicts ovarian malignancy in most of the cases.

SUMMARY

- A prospective study of ovarian tumours was done to assess the diagnostic accuracy of Doppler in predicting malignancy.
- The results of this study was based on 50 patients for whom the decision had already been made to proceed with operative intervention.
- Incidence of ovarian tumour in my study 4.4%.
- Incidence of malignant tumour was 24% of all ovarian tumours.
- Commonest Benign tumour in my study – serous cystadenoma. Commonest malignancy in my study – serous cystadenocarcinoma.
- Doppler showed sensitivity, specificity, positive predictive value of 91.7%, 94.6% and 84.6% respectively.
- Diagnosis of ovarian tumour is highly accurate with the negative predictive value of 97.2%.

CONCLUSION

- Prediction of malignancy is of great value in preoperative evaluation of ovarian tumours.
- Clinical examination the standard diagnostic approach to pelvic mass should always be combined with other ancillary aids to the diagnosis.
- The primary imaging modality in all cases of suspected pelvic mass in ultrasonography as it narrows the wider differential diagnosis of pelvic tumour.
- Doppler was highly accurate in predicting the nature of ovarian tumours.
- Diagnostic accuracy can be improved with CT, MRI.
- My study on preoperative diagnosis of ovarian tumour with Doppler and its correlation with histopathology is simple, safe and reliable.
- To reduce the mortality of ovarian tumour our aim is to screen all postmenopausal women and high risk patient with Doppler and other modalities.

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PROFORMA

Name : Age : I.P.No.

Occupation:

Complaints : Abdominal Swelling

Abdominal pain

Menstrual Disturbances

Masculinising Features

Postmenopausal bleeding

Pressure Symptoms

Nonspecific symptoms

Asymptomatic

Menstrual H/o : Early Menarche / Late Menopause

Marital H/o :

Obstetrics H/o. : Nulliparous

Past H/o. : H/o Ovulation induction

H/o. Oral contraceptive Pill Intake

H/o. Breast Lump

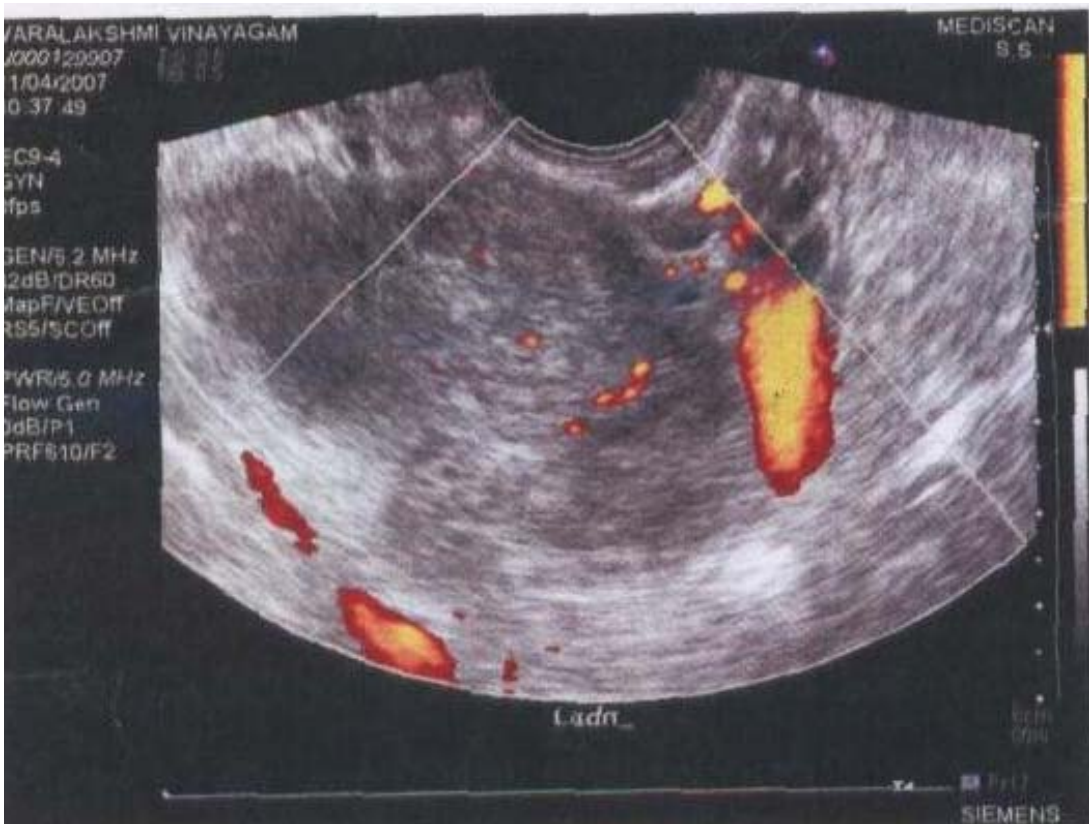
Gastrointestinal Tumours

Family H/o. Tumour in relatives.

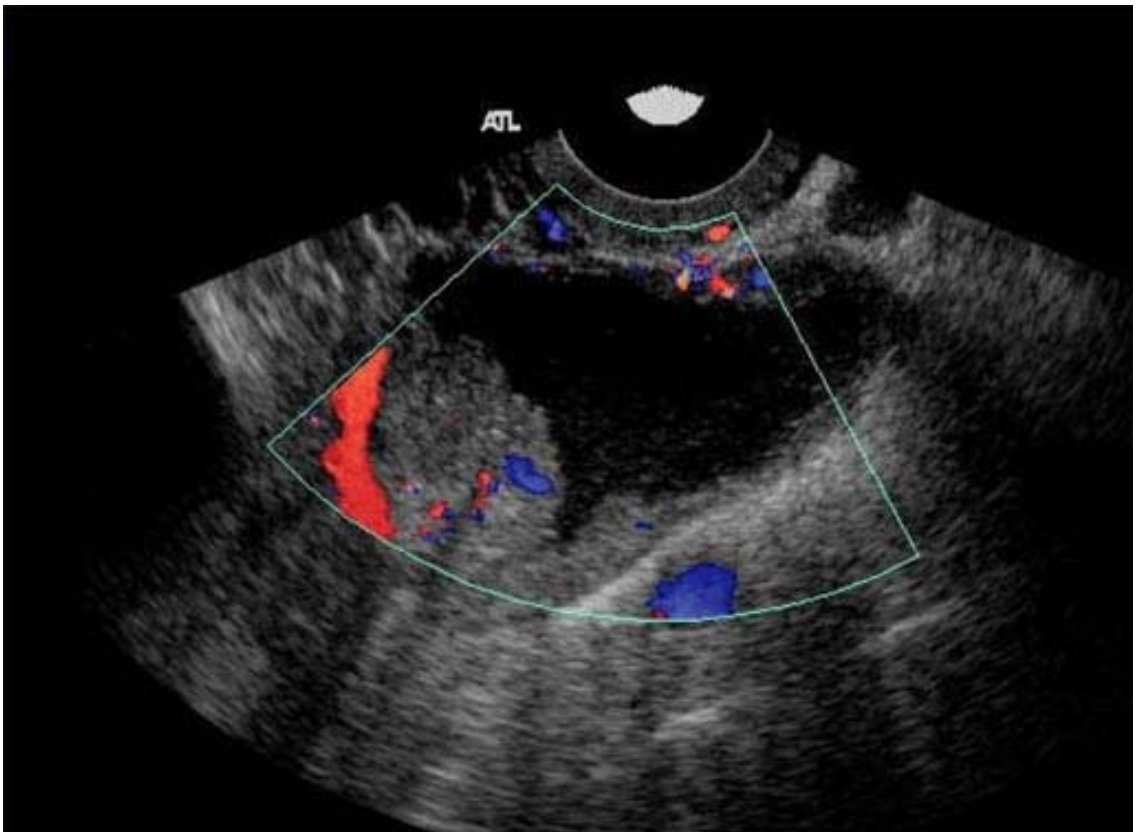
O/E

GENERAL EXAINATION		Anemia
		Cachexia
		Built
		Nourishment
		Lymph nodes
		CVS
		RS – Pleural effusion
	P/A	Inspection
		Palpation
		Percussion
	P/S	
	P/V	
	P/R	
	Doppler	Resistance Index
		Pulsatality Index
		Low resistance flow
		High resistance flow
	Surgery Done	
	Intra operative findings	
	Histopathological Examination	

BENIGN TUMOUR WITH MINIMAL VASCULARITY



MALIGNANCY WITH LOW RESISTANCE FLOW



MASTER CHART

S.No.	Name	Age	IPNo.	Complaint	Mens H	Parity	GE	PA/PV	Doppler	Laporotomy	HPE
1	Kamsala	33	25251	Pain Abd X1 Mth	3/30	P_2L_2	Fair	Fullness+ Left - Formix	No Vascularity	Right Ovarian Cystectomy	Benign serous cystadenoma
2	Lakshmi	40	11010	Pain Lower abdx 8 Mth	3/30	P_4 L_3	Fair	P/V Mass of 10X8 Cm. Felt Thro Left Formix	No Vascularity	Left Ovarian Cystectomy	Benign serous cystadenoma
3	Govindammal	45	11788	Pain Abd X1 Mth	3/30	P_2L_2	Fair	Mass of 16cm in Hypogastric and left iliacfossa	High resistance flow	TAH with LSO with rt. Salphingo ovariotomy	Benign serous cystadenoma
4	Sundari	21	12769	Pain Abd X1 Mth	3/30	Nulli	Fair	Mass of 18-20WK	No Vascularity	Left Ovarian Cystectomy	Benign serous cystadenoma
5	Philomina	26	12857	Pain Abd X1 Mth	3/30	P_2L_2	Fair	Mass of 18-20 WK	High resistance flow	Left Ovarian Cystectomy	Bengin Cystic teratoma
6	Alima	40	12900	Pain Abd X1 Mth	3/30	P_4L_4	Anemia	P/V Mass arising from Pelvis occuping ant and lat- fornix	low resistance flow	TAH with LSO with Rt. Salphingo ovariotomy	Granulosa cell tumour of ovary poorly differnetiated

7	Sabitha	24	13646	H/O Irregular Menstrual Cycle +H/o abd. Pain+illiac Fossa on and off	3/30 -60	Nulli	Fair	P/Vleft Fornix + fullness	No Vascularity	Laporoscopic Left Ovarian cystectomy	Chocolate Cyst
8	Uma	28	15403	Bleeding PV x2mth		Nulli	Fair	P/V Mass of 6x6cm. Felt thro left fornix.	No Vascularity	Left ovariotomy with Biopsy form right ovary	Bengin Cystic teratoma left ovary
9	Jailakshmi	60	15735	Abd Distension X2mth.Abd PainX 2Mth	3/30	P_3L_3	Fair	Irregular Mass measuring 20 Wk. size + Ascites	low resistance flow	Laporotomy with B/L Ovariotomy with TAH	Serous cystadenoma of Ovary
10	Panchavarnam	51	16172	Pain abd 1 1/2 vomiting 2mth on and off	3/30	P_4L_4	Fair	Post fornix Hard Mass	low resistance flow	B/L Ovariantumour reduction with ifracolic omentectomy	Serous Cystadenocarcinoma of Ovary with secondary deposits in Omentum
11	Gajalakshmi	43	16469	Pain abd 4years	Menopause four year back	P_4L_4	Fair	Mass of 10x8cm. In Right iliac and hyogastric region	No Vascularity	TAH with RSO with Lt. Ovariotomy	Benign serous cystadenoma
12	Jothi	23	16687	Irregular cycles and Dymenorrhoea	5-6/15	$P_1 L_1$	Fair	Mass 5x6cm Suprapubically	low resistance flow	TAH with BSO	Kurkenberg Tumour
13	Renuga	51	16925	Abd Swelling x4 Mth Pain abd 4Mth	5/30	P_3L_3	Fair	Mass20x20 Cm	No Vascularity	TAH with Rt. Salphingo Ovariotomy with left Salphingo oophorectomy	Benign serous cystadenoma

14	Krishnaveni	27	18413	Pain abd.X1Mth .H/o Vomiting +	3/30	P_2L_2	Fair	P/V Cystic Mass of 10x8 Cm. Felt ant to Uterus	High resistance flow	Right Ovarian Cystectomy	Benign serous cystadenoma
15	Saritha	19	20	Pain abd.1 Mth	3/30	Unmarried	Fair	P/V Mass of 6x6cm. Felt thro left fornix.	No Vascularity	Left Ovariotomy with Rt. Ovary Wedge resection	Benign serous cystadenoma
16	Kanakavalli	52	142	Abd Distention 1 Mth	Attained menopause 11years back	P_1 L_1	Anemia	P/V Vague mass present in Right ant fornix	low resistance flow	TAH With BSO with omental Biopsy	Mucinous CytadenoCarcinoma
17	Kalavathy	21	605	Pain abd 12Wks	3/30-33	P_1 L_1	Fair	P/V mass 6x6Cm.+ Right Fornix	No Vascularity	Right Ovarian Cystectomy	Benign serous cystadenoma
18	Rukmani	42	907	Pain abd. X6mth	attained menopause 3years back	P_4L_4	Fair	P/V Fullness Right fornix	High resistance flow	Right Ovarian Cystectomy	Benign serous cystadenoma
19	Bagyathai	55	908	Abd Distention 4mth	attained menopause 13years back	P_1 L_1	Fair	Mass Palpable up to 28wks	low resistance flow	TAH With BSO	Benign Mucinous Cystadenoma
20	Pramila Devi	38	923	Pain abd.1wks.1day	3/30	P_3L_3	Fair	P/V Fullness Right fornix	No Vascularity	TAH With BSO	Benign Serous cystadenoma

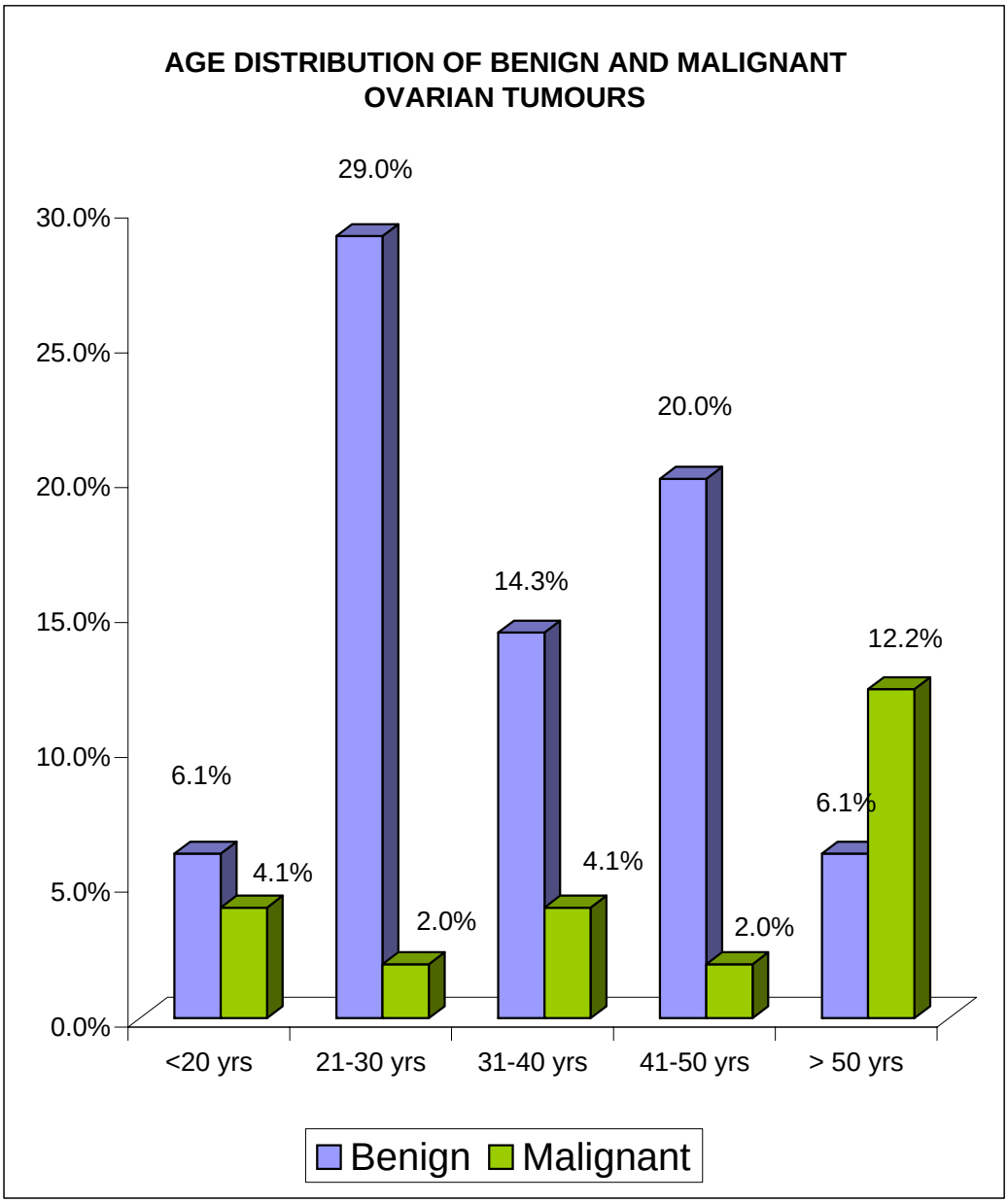
21	Radha	48	1981	Pain abd.1.Wk	Attained menopause 1year back	P_4 L_3	Fair	Cystic Swelling 10x6cm.Left iliac fossa	No Vascularity	Right Ovarian Cystectomy	Benign Serous Cystadenoma
22	Maliga	40	2612	Pain x 1 yr	3/30	P_4L_4	Fair	Mass of 18wk	low resistance flow	TAH with BSO	Serous Papillary Cystadenoma
23	Radhika	17	3065	Abd.Swelling 2Mth H/O Profuse Bleeding 20days	5/30	Unmarried	Fair	Mass of 24 wks	low resistance flow	Right ovariotomy with omental Biopsy and left ovarian Biopsy	Yolk Sac Tumour
24	SelvaMary	24	3432	Abd.Swelling 4mth Abd pain 4Mth	5/50	Unmarried	Fair	Mass of 20x10 Cm	low resistance flow	Right Ovarian Cystectomy	Border Line Mucinous tumour
25	Nalini	42	4886	Pain abd.2 wks	3/30	P_1 L_1	Fair	P/V Fullness LeftFornix	No Vascularity	TAH with RSO with Rt. Ovarian Cystectomy	Benign Mucinous Cystadenoma
26	Varalakshmi	38	5135	Pain Abd.1Wks	3/30	P_2L_2	Fair	P/Vleft Fornix + fullness	No Vascularity	Left Salphingo Ovariotomy	Benign Serous Cystadenoma
27	Rakkammal	41	5350	Abd Swelling x3 Mth Pain abd 3Mth	3/30	P_2L_2	Anemia	Mass of24 wks	High resistance flow	TAH with Right Ovariotomy with left Salphingo oophorectomy	Beningn Papillary Cystadenocarcinoma
28	Rajeswari	24	5398	Pain Abd.2Mth	3/30	Nulli	Fair	P/V Right fornix Fullness+	High resistance flow	Right ovariotomy	Beningn Cystic Teratoma

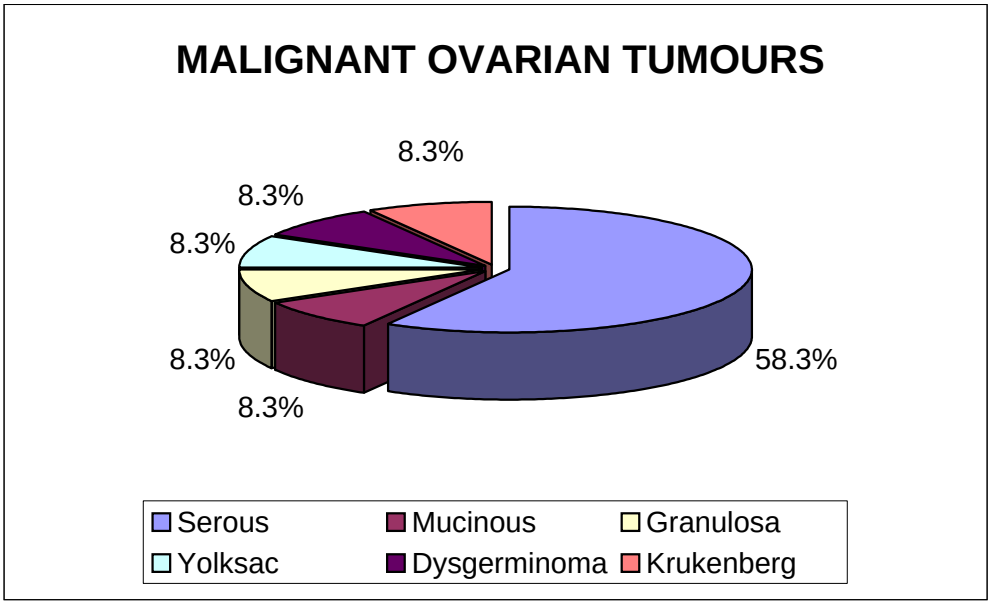
29	Chandra	47	5976	Pain Abd.7 Mth	3/30-60	P_2L_2	Fair	Fullness+ Post and Left -Formix	No Vascularity	TAH with left Ovariotomy with RSO	Benign serous cystadenoma
30	Shanthi	47	6140	Pain Abd1Mth. Bleeding PV 2days	3/6 months	P_3L_3	Fair	P/V Right fornix Fullness+	No Vascularity	TAH with left Ovariotomy with RSO	Beingn Serous-cyst
31	Sarswathi	35	6361	Irregular Bleeding x1Years.	4/3 Months	P_1 L_1	Fair	P/V Right fornix Fullness+	High resistance flow	TAH with left Ovariotomy with RSO	Benign Serous cystadenoma
32	Vijayalashmi	35	6490	Abd Distention X6Mth	3/30	Nulli	Fair	P/V Right fornix Fullness+	No Vascularity	TAH with Right Ovariotomy with LSO	Benign serous cystadenoma
33	Uma	26	6564	Mass abd.x1year	3/30	P_2L_2	Fair	Mass 24-26 wks. Size	No Vascularity	Left Ovarian Cystectomy	Benign serous cystadenoma
34	Jayselvi	24	6908	Abd.Distention1Mth	3/30	Nulli	Fair	Mass of 20Wks.Size	No Vascularity	Laporotomy with left ovariotomy with rigyht ovarian wedge Biospy omental Biopsy	Benign Mature Cystic Teratoma

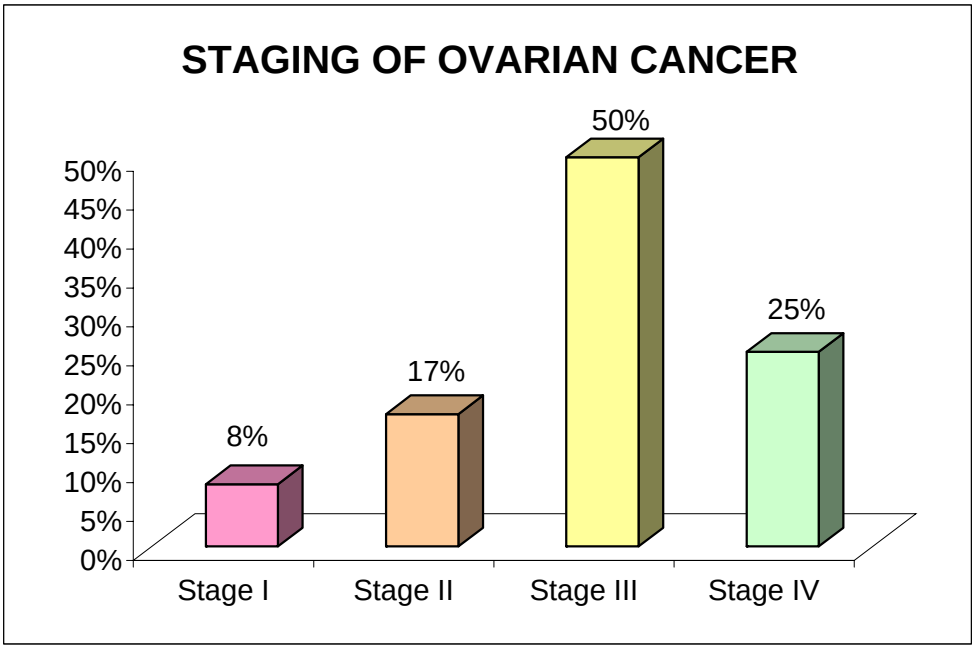
35	Vedham	51	7151	Pain Abd.1Mth Excessive Bleeding Pvx6Wks	6/30	P_3L_3	Fair	Mass of 20Wks.Size	low resistance flow	Laporotomy with debulking of right ovary, left oophorectomy with Hysterectomy	Moderatly Differetiated B/L Serous cystadenocarcinoma of ovary
36	Bharathi	34	7662	Pain abd 1year	3/30	P_2L_2	Fair	P/V Fullness+ Right left and ant fornix+	High resistance flow	Right Salphingo Ovariotomy	Benign Mucinous Cystadenoma
37	Papathi	60	8018	Pain abd 1mth	Attained menopause 20year back	P_6L_6	Fair	P/V Right fornix Fullness+	No Vascularity	TAH with Right Salphingo Ovariotomy with LSO	Benign Mucinous Cystadenoma
38	Lakshmi	44	8057	Pain Abdomen 6mth Mass Abdomen6Mth	6/30	P_4 L_3	Fair	Mass of 22Wks.size	No Vascularity	TAH with Right Salphingo Ovariotomy with LSO	Benign serous cystadenoma
39	Irfana	18	8232	Pain Abdomen 4mth Mass Abdomen 4Mth	3/30	Unmarried	Fair	Mass of 36Wks. Size	low resistance flow	Left oophorectomy	Benign Mucinous Cystadenoma
40	Kulandaiammal	28	8873	Pain abd1year	4/30	P_2L_2	Fair	Mass of 16wks	No Vascularity	Right Ovarian Cystectomy	Benign serous cystadenoma
41	Nisath Begam	14	8990	Abd. Swelling 1Mth	4/30	Unmarried	Fair	Mass of 7.5x5cm.in left iliac fossa	low resistance flow	Left Ovarian Cystectomy	Dysgerminoma

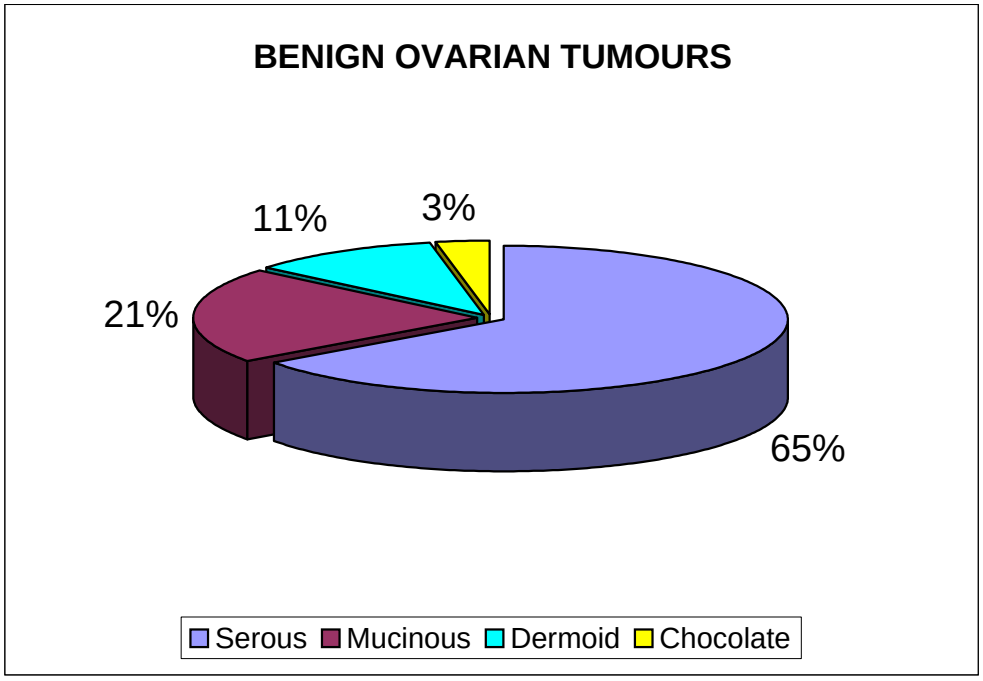
42	Shankari	54	91224	Abd Distentionx2Mth	Attained menopause 1year back	P_2L_3	Fair	Mass of24wks	No Vascularity	Left Ovarian Cystectomy	Benign serous cystadenoma
43	Lakshmi	60	9186	Abd.Pain1Mth Distention1Mth	Attained menopause 25year back	P_5L_5	Fair	P/V Mass of 10x8cm. Felt through right and post fornix	low resistance flow	Exploratory Laporotomy	Papillary serous Cystadenocarcinoma
44	Arupudham	67	9187	Abd.Swelling 1wks Abd.Pain 3wks	Attained menopause 10year back	P_5L_2	Fair	Mass of 10x8cm Occupying the left iliac fossa	low resistance flow	TAH with left Salphingo ovariotomy	Moderatly Differetiated Serous cystadenocarcinoma
45	Shamandishwari	30	9247	Pain abdx3mth	3/30	P_5L_2	Fair	Mass of 28wks size	No Vascularity	Right Ovarian Cystectomy	Benign serous cystadenoma
46	Kalaivani	48	10016	Pain abd.3 days	5/2-3mts	P_3L_2	Fair	Mass of 16-18 wks size	High resistance flow	Right Ovarian Cystectomy	Benign Mucinous cystadenoma
47	Kala	17	10273	Pain abdx4Mth Difficulty in Passing urine 1weeks	3/30	Unmarried	Fair	Mass of 10x8.8cm.in Hypogastric region	High resistance flow	Right Ovarian Cystectomy	Benign Mucinous Cystadenoma
48	Neela	25	10424	Pain abdx5days	3/30	P_2L_2	Fair	Mass of 28 to 30Wks.Size	High resistance flow	Right Ovarian Cystectomywith left ovarian wedge biopsy	Benign Mucinous Cystadenoma

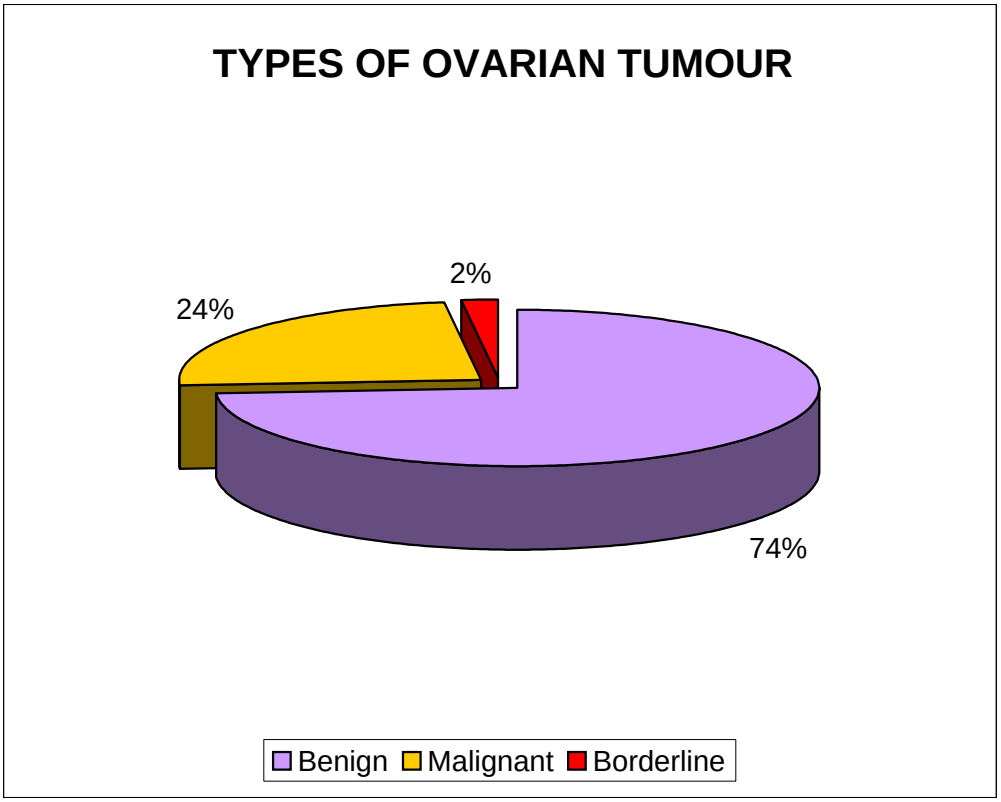
49	Vanaja	30	10332	Pain abd7 monthsexcessive bleeding 6mths	7/30	P_3L_3	Fair	P/V Mass of6x5Cm felt in ant right fornix	No Vascularity	Right ovarian Cystectomy	Benign serous cystadenoma
50	Malar	22	12006	Pain abdx2Mths	3/30	P_3L_2	Fair	Mass of 6x8cm in Hypogastric region	No Vascularity	Right ovarian Cystectomy	Benign Serous Cystadenoma


AGE DISTRIBUTION OF BENIGN AND MALIGNANT OVARIAN TUMOURS
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
6.1%
4.1%
29.0%
2.0%
14.3%
4.1%
20.0%
2.0%
6.1%
12.2%
<20 yrs
21-30 yrs
31-40 yrs
41-50 yrs
> 50 yrs
Benign
Malignant


MALIGNANT OVARIAN TUMOURS
8.3%
8.3%
8.3%
8.3%
8.3%
58.3%
Serous
Mucinous
Granulosa
Yolksac
Dysgerminoma
Krukenberg


STAGING OF OVARIAN CANCER
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
8%
17%
50%
25%
Stage I
Stage II
Stage III
Stage IV


BENIGN OVARIAN TUMOURS
11%
3%
21%
65%
Serous
Mucinous
Dermoid
Chocolate


TYPES OF OVARIAN TUMOUR
2%
24%
74%
Benign
Malignant
Borderline