A CLINICO- RADIOLOGICAL AND HISTOPATHOLOGICAL STUDY OF OVARIAN MASSES AT A TERTIARY CARE CENTRE.

By

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY

IN

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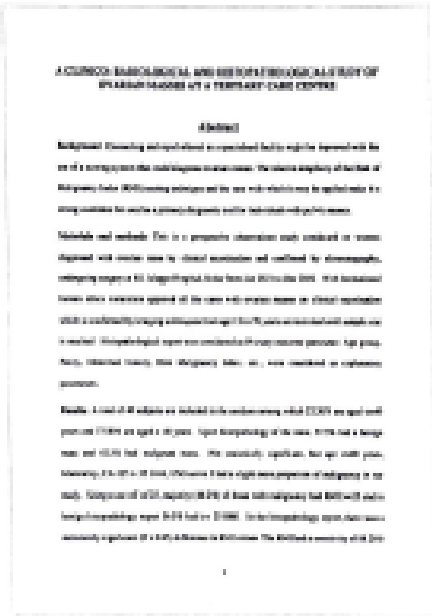

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DATE **DR. SHRAVYA MONICA K**

PLACE

A CLINICO- RADIOLOGICAL AND HISTOPATHOLOGICAL STUDY OF OVARIAN MASSES AT A TERTIARY CARE CENTRE

Abstract

Background: Counseling and rapid referral to a specialised facility might be improved with the use of a scoring system that could diagnose ovarian cancer. The relative simplicity of the Risk of Malignancy Index (RMI) scoring technique and the ease with which it may be applied make it a strong candidate for used as a primary diagnostic tool for individuals with pelvic masses.

Materials and methods: This is a prospective observations study conducted on women diagnosed with ovarian mass by clinical examination and confirmed by ultrasonography, undergoing surgery at RL Jalappa Hospital, Kolar from Jan 2021 to Dec 2022. With Institutional human ethics committee approval all the cases with ovarian masses on clinical examination which is confirmed by imaging techniques from age 13 to 70 years are recruited until sample size is reached. Histopathological report was considered as Primary outcome parameter. Age group, Parity, Menstrual history, Risk Malignancy Index, etc., were considered as explanatory parameters.

Results: A total of 40 subjects are included in the analysis among which 22.50% are aged <=40 years and 77.50% are aged > 40 years. Upon histopathology of the mass, 57.5% had a benign mass and 42.5% had malignant mass. Not statistically significant, but age <=40 years, bilaterality, CA-125 > 35 U/ml, USG score 3 had a slight more proportion of malignancy in our study. Using a cut off of 25, majority (88.2%) of those with malignancy had RMI >=25 and in benign histopathology report 56.5% had >= 25 RMI. In the histopathology report, there was a statistically significant ($P < 0.05$) difference in RMI values. The RMI had a sensitivity of 88.24% in predicting

malignancy with specificity 43.48%, positive predictive value 53.57%, negative predictive value 83.33% with a total diagnostic accuracy of 62.50%. Results from RMI and histology correlate positively.

Conclusions: Results from RMI and histopathology correlate positively. The results of this research show that RMI is a reliable and practicable method for assessing patients with pelvic masses at the commencement of therapy and identifying those who are good candidates for centralised surgical treatment.

Key words: Ovarian malignancy, Risk Malignancy Index, ultrasonography, RMI, CA-125, postmenopausal, histopathology

LIST OF ABBREVIATIONS

Glossary	Abbreviations
ASIR	Age Specific Incidence Rate
CA	Cancer Antigen
CA125	Carbohydrate Antigen
CT	Computed Tomography
FSH	Follicle-Stimulating Hormone
GnRH	Gonadotropin-Releasing Hormone
HE4	Human Epididymis protein
HNPCC	Hereditary Non-Polyposis Colorectal
HPO	Hypothalamic-Pituitary-Ovarian
HRT	Hormone Replacement Treatment
LH	Luteinizing Hormone
M	Menopausal Status
NACT	Neoadjuvant Chemotherapy
RMI	Risk of Malignancy Index
U	Ultrasonographic
WHO	World Health Organization's

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INTRODUCTION

INTRODUCTION

The ovary is a crucial organ because it is involved in the creation of offspring. Mesenchymal cells and sex cells, which are totipotent and multipotent, respectively, make up the ovary. Consequently, almost any type of tumour can develop when it turns neoplastic.[1] Even though they only account for 14 gm in an adult, the ovaries are the site of a wide range of benign and malignant tumours due to the extensive range of hormonal stimulation and changes that occur from the foetal period through menopause. Most ovarian lesions are functional and will recover without much medical intervention. A large, bothersome cystic lesion may require surgery.[2,3] Gynecological oncologists face a lot of challenges from ovarian tumours and non-neoplastic lesions. Some ovarian non-neoplastic lesions frequently show up as a pelvic mass that resembles a tumour of the ovaries. Therefore, it is crucial that they are correctly identified and categorised to enable effective therapy.[4]

After cervical and uterine cancers, ovarian cancer is the third most common gynecologic malignancy in women.[5] The prevalence of ovarian cancer is very less in comparison with breast cancer but it has poor prognosis and has three times higher mortality rate than breast cancer.[6] Ovarian cancer has a high fatality rate since it often has no early warning symptoms until advanced in stage and lack of proper screening. This cancer often manifests at a more advanced stage, and the 5-year relative survival rate is just 29%. Despite a 92 percent five-year survival rate, only 15 percent of patients are detected at stage 1. Rate of ovarian cancer survival in the general population varies between 30 to 40% in the world.[7] Ovarian cancer has a 6.6/100,000 "age-standardized" incidence rate and a 3.9/100,000 mortality rate.[8] The incidence of ovarian cancer in India is reportedly the second highest worldwide. Menopausal women account for 90% of ovarian cancer cases, often between the ages of 55 and 64, suggesting that longer life expectancy may be contributing to the global rise in ovarian cancer rates.[9] Ovarian tumours are

categorised by tissue of origin in the “World Health Organization's” (WHO) categorization system. Of all ovarian malignancies, 55% are “surface epithelial”, 15% “germ cell”, 10% “sex cord stromal”, and 5% are metastatic. Over 90% of ovarian cancers begin as epithelial growths at the surface. [9] The risk of developing ovarian cancer increases with age and is also influenced by one's ethnic background. Ovarian cancer is most common in the USA, next in India, and finally in China.[10]

Ovaries are least accessible female reproductive organs because of which there is delay in diagnosis of ovarian disorders including borderline tumours and ovarian malignancies.[11] Diagnosis and differentiation of ovarian masses determination as to whether the tumour is benign or malignant is crucial for effective care. The vast majority of ovarian tumours are completely harmless. It is important to identify the pathology of ovarian masses as benign or malignant for better outcome. Recognizing malignancy in early stages helps in initiation of early treatment as ovarian cancer at later stages is lethal.[11] Ovarian cancer detected early can be cured in 90% of women. Even when there is spread of cancer to pelvis that is in stage II, 70% five-year survival rate is achieved. The survival rate drops to 20% after cancer has advanced to the “abdominal cavity” (stage III) or beyond the “abdominal cavity” (stage IV) into the liver[12] parenchyma. While it has been stated that early detection of ovarian cancer may cut death rates by 10% to 30%, only 20% of cases are detected at this time.[13]

It is well established in the field of oncology that neoplastic diseases of the ovaries have a convoluted and baffling past. The neoplasm that develops from it acquires a histogenetic foundation that is more diverse than any other.[14] Differentiation of early malignant ovarian tumours from benign ovarian tumours is essential for good prognosis. Carbohydrate Antigen (CA125) is elevated in ovarian cancers and hence can be used as biomarker for diagnosis of the same. “Human Epididymis protein” (HE4) is another biomarker used for diagnosis.[15]

Determination of Ovarian Cancer using these biomarkers is highly specific yet insensitive. In addition to inflammatory bowel illness, CA125 is reported to be elevated in a wide range of normal and abnormal conditions, including but not limited to menstruation, pregnancy, endometriosis, and peritoneal inflammation.[16] Multidisciplinary effort combining anatomical, pathological and clinical laboratory services is required for optimal ovarian diagnosis and care.

Pelvic evaluation, tumour markers, and radiographic studies have all been proposed as possible solutions, however none of these characteristics alone is reliable enough to make a diagnosis. Several hybrid methods for gauging ovarian size have also been proposed. An improved, more useful, and more sensitive metric is the "Risk of Malignancy Index" (RMI). RMI is calculated using a simple regression equation that takes into account the "menopausal status" score (M), the "ultrasonographic" score (U), and the "absolute" value of blood CA-125.[17,18,19] Researcher-made medical images (RMI) have been described as the most effective method for assessing ovarian tumours and determining next-steps in treatment and referral in many retrospective and prospective studies. [20,21] The RMI's excellent sensitivity for ovarian cancer diagnosis holds up when tested on a new cohort of women and remained consistent with the original paper outlining its development. However, there was little detail. A more precise diagnosis of ovarian cancer may be made using the RMI, the research found, as compared to using the individual criteria. [20] A recent study indicated that a higher RMI cut-off of 238 had a sensitivity of 89.5%, specificity of 96.2%, positive predictive value of 77.3%, and negative predictive value of 98.4% when used for screening. [22]

Need for the study

The lack of resources for early identification, treatment, and monitoring of ovarian cancer is correlated with higher death rates in low-income nations like India. [23] As 1 in 2500 women beyond menopause have ovarian cancer, this statistic is alarming, an effective screening test would need to have a sensitivity of 75% or higher for detecting disease in its earliest stages, a specificity of 99.6%, and a positive predictive value of at least 10% are all desirable. [24] Detailed ultrasound images of the ovaries may be used to identify morphologic abnormalities that may indicate cancer. However, there is substantial inter-observer variability in the interpretation and grading of ultrasonographic pictures. When it comes to identifying ovarian lesions, the positive predictive value of ultrasonography is good, but the specificity is poor. [25] Considering the frequency of benign ovarian lesions in postmenopausal women, it is not surprising that there is a high percentage of false-positive findings. By combining CA-125 testing with ultrasonography, more specificity may be attained. According to the data compiled by Globocan, millions of new instances of reproductive cancer are diagnosed each year in India, with the majority of these malignancies being gynaecological in nature. According to the "National Cancer Registry" Cancer Statistics Reports, 6.2% of all cancer diagnoses are of ovarian origin.[5] Breast, cervical, and ovarian cancers will account for almost 34% of all cancer deaths in women by 2026, a research has shown.[26] The above facts indicate that prevalence of ovarian cancer is bound to increase in coming years in India and it requires reliable, easily accessible diagnostic test for early diagnosis and improving the chances of survival. Individual tests like biomarker levels, ultrasound imaging tests are found to ineffective in early accurate diagnosis of malignancy of ovarian tumours. The health seeking behaviour of women in India is very low. Health care of women is considered as last priority. Most of the women seek medical care when their problem reaches advanced stage of disease. The purpose of this

research is to improve early diagnosis of ovarian masses by establishing a correlation between clinical presentation, ultrasonography characteristics, and tumour marker levels and histology.

Tertiary care is provided at “Sri Devaraj Urs Academy of Higher Education and Research”, a teaching hospital in Kolar. To a lesser extent, it also helps the people of Kolar town and the neighbouring states' outlying areas. Patients seeking care at the hospital were mostly farmers and labourers. Cancer cases detected by FNAC and histology at the hospital's Department of Pathology during a 10-year period revealed 13.98% of malignancy, with a majority of female population, according to a retrospective analysis based on the hospital registry (male: female; 0.7:1). Ovarian cancer prevalence was 2.98 percent. [27]

AIMS & OBJECTIVES

Objectives of the study:

1. To study the spectrum of clinical presentation in a patient who presents with ovarian mass.
2. To assess the correlation between the clinic radiological profile with histopathological picture.

REVIEW OF

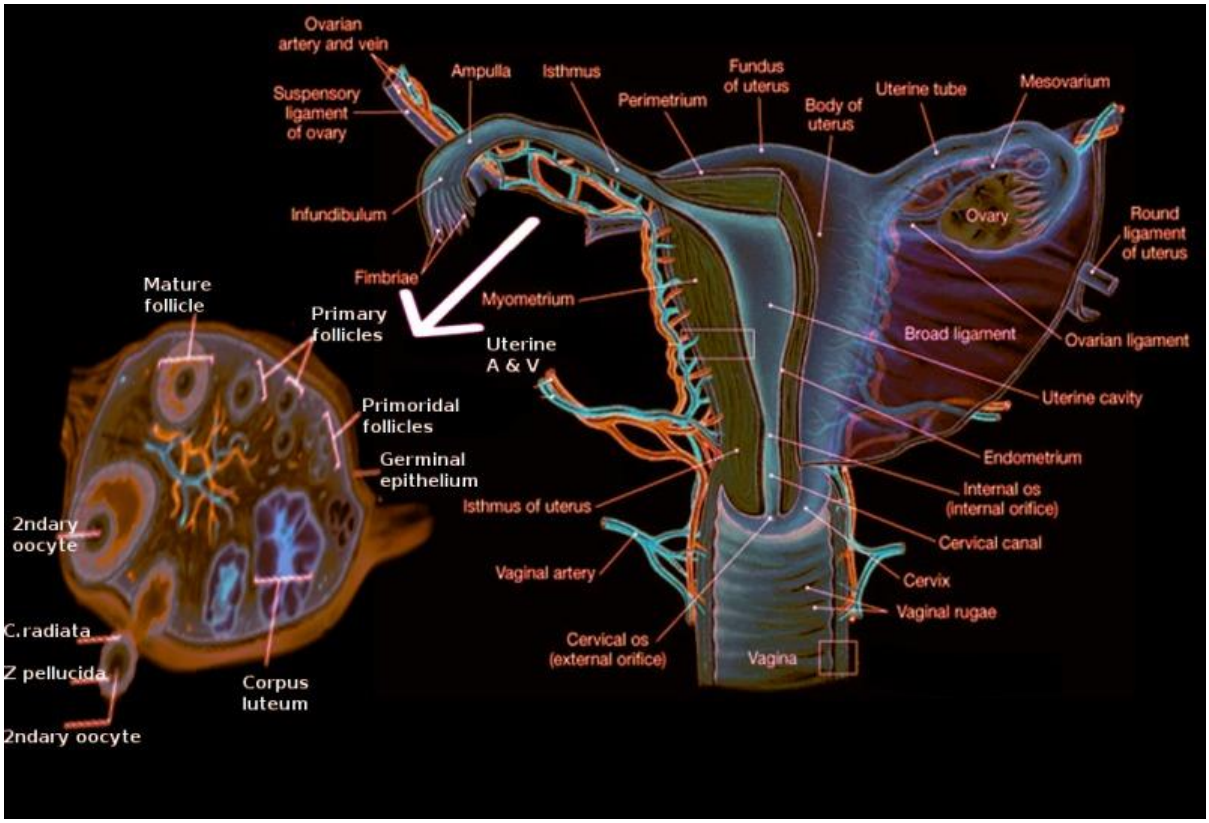
LITERATURE

REVIEW OF LITERATURE:

Anatomy of ovary:

The ovary is the "female gonad". The paired intraperitoneal endocrine organs are normally located lower in the left and right abdominal quadrants. The primary role of the ovary includes hormone production and reproduction. [28] Ovaries are reproductive organs that are often found in the ovarian fossa, a small sac that sits just above the pelvic region. The ovarian “fossa” is located at the junction of the external and internal iliac arteries. The ovary is situated anterior to the “medial umbilical ligament.” Anatomical structures such as the ureter and internal iliac artery are located in the posterior region of the ovary. “Suspensory or infundibulopelvic” ligament, also called mesovarium, is the uterine ligament that extends posteriorly from the broad ligament. It is situated over the ovary and the uterine tube's infundibulum. Ovaries are attached to the body via a suspensory ligament. The ovary might be found at a position that is inferior to the angle formed by the body and the uterine tube, both of which are joined by the ovarian's proper ligament. The ovary is connected by two ligaments. The suspensory ligament is responsible for carrying a number of important structures, including the sympathetic and parasympathetic plexuses, as well as the ovarian artery and vein. The remaining gubernaculum is made up of the blood vessel-free "proper ligament" of the ovary. [29] These ligaments are avenues for spread of ovarian disease seen in malignant subtypes [30].

Figure 1: "Anatomy of ovary"[31]



Comparable in size to a golf ball, a healthy ovary measures 3.5 centimetres in length, 2.0 centimetres in diameter, and 1.0 centimetres in thickness. Changes in ovarian size are a natural part of the ageing process for women. Research suggests that age accounts for as much as 69% of the variation in ovarian volume. The typical ovarian capacity is 0.7 ml by the time a girl is two years old. The maximum volume is reached around age 20 and is 7.7 ml. After then, the volume gradually declines until it stabilises at about 2.8 ml at menopause. [32]

Before menopause, the ovarian cortex contains germ cells in varying stages of development. A very thick and fibrous stroma constitutes the ovary. The ovary is closely connected to the distal fimbriated end of the fallopian tube, which is coated with ciliated serous type epithelium. This area of the fallopian tube is where ovulation occurs. [33] Ovary size changes as people become older. There is a layer of simple cuboidal cells (epithelium) on the outside, followed by a layer of "connective" tissue (collagen) called "tunica albuginea". Ovarian follicles of

various sizes and maturation stages may be seen in the cortex. The hilus is the core region, which is composed of connective tissue and houses the body's main arteries and veins. [34]

Blood supply and lymphatics

The ovary receives its blood supply from the uterine artery as well as the ovarian artery. From the abdominal aorta, the paired ovarian artery branches out around L2, or just below the renal artery. Before entering the mesovarium, the artery traverses the "suspensory ligament" of the ovary. It's possible for the ovarian artery and uterine arteries to join within the wide ligament.[35]

Ovarian vein drains the "parametrium", "cervix", "mesosalpinx", and "pampiniform plexus"; it is suspended from the cervix to the ovary by the "suspensory ligament." It is connected to the venous plexuses of the "para-ovarian", "uterine", "vesical", "rectal", and "vulvar" regions. The "left ovarian" vein drains in to the left renal vein, whereas the "right ovarian" vein empties directly into the "inferior vena cava." A normal ovarian vein measures about 5 mm in diameter.[36]

L2 paraaortic lymph nodes get the vast bulk of ovarian lymph. The lymph goes via the ovarian circulation on its way to the "paraaortic" nodes, which are located near the point in which the aorta splits off from the renal arteries. However, scientists have uncovered two alternative routes. The first is the lymph system, which drains into the paraaortic nodes from the hypogastric nodes through lateral arteries. The inguinal and external iliac lymph nodes receive lymph that has travelled via vessels close to the round ligament.[37] These pathways are significant in the clinic because they allow ovarian cancer to metastasize.

Nerves

The ovarian plexus is one of two sympathetic nerve bundles that supply the ovary. The ovarian "plexus" originates in the renal "plexus", and it further supplies the uterine fundus with neural tissue. This plexus travels from the fallopian tubes to the ovaries through the suspensory ligament of the ovary. The second place where the "sympathetic" nervous system is active is in the "superior" ovarian nerve, which is located in the ovarian ligament. [38]

The pelvic "splanchnic" nerves give birth to the uterine (pelvic) plexus, which provides parasympathetic innervation.

Functions of ovary:

Ovaries provide two main purposes. The ovary is responsible for the generation of hormones, a role that changes with puberty. In response to elevated amounts of "gonadotropin-releasing hormone" (GnRH), the ovaries produce more oestrogen, testosterone, inhibin, and progesterone. This dynamic establishes the "hypothalamic-pituitary-ovarian" (HPO) axis. [39] GnRH, which is secreted by the hypothalamus, influences the "anterior pituitary cells." Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are both hormones that are secreted by the "anterior pituitary gland." FSH is known for its great affinity for "granulosa" cells, which aid in the growth and maturation of follicles. The progenitor cells for androgens and estradiol, known as the "theca cells", will respond to LH. Estradiol is converted to oestrogen throughout puberty, and this increase in oestrogen is responsible for the development of auxiliary sex characteristics. [40]

Oocytes, or egg cells, begin developing in gestation and continue until puberty, at which point they are released from the ovary. After the pituitary gland secretes a large amount of "luteinizing" hormone, the ovum matures and is expelled, a process known as ovulation.

Follicles in the antral region are normally between 2 and 9 mm in diameter. On average, there are less than 25 hair follicles present (when using optimal resolution). During the course of a menstrual cycle, antral follicles expand until one becomes the dominant follicle, while the others decline.

Estrogen and progesterone are among the hormones secreted by the ovary's "granulosa cells" and "theca cells." The follicles will develop in this location in the ovary during the "proliferation phase." When the dominant follicle is fully developed, a surge of "luteinizing hormone" triggers the release of the oocyte, which then travels via the uterine tube and into the uterus. [41] The ovary is temporarily replaced by the corpus luteum, an endocrine organ responsible for the secretion of progesterone and, to a lesser extent, estradiol and inhibin A. To ensure that sperm and egg may meet and implant before menstruation, the body synthesises hormones to shield the oocyte. Upon fertilisation, a blastocyst produces "human chorionic gonadotropin," which signals to the "corpus luteum" to maintain progesterone secretion. The placenta will assume responsibility for this after it has developed fully. Without fertilisation, the corpus luteum transforms into the "corpus albicans", and the lack of progesterone causes menstruation. [42]

Ovarian masses:

Definition:

Adnexal masses or ovarian masses are fluid filled structures formed in ovaries. There are 30 different types of ovarian masses which can be "benign" or "malignant." Even though most ovarian cysts seen in women of childbearing age are harmless, problems such pelvic discomfort, cyst rupture, blood loss, and ovarian torsion may occur. [43]

Epidemiology:

Risk factors for ovarian cancer are not uniformly distributed, which contributes to the global variation in ovarian cancer incidence. Ovarian cancer occurs at a rate of 12.0 per 100,000 among non-Hispanic white women, 10.3 among Hispanic women, 9.4 among non-Hispanic black women, and 1.7 among Asian and Pacific Islander women (9.2 per 100,000). [44] Ovarian cancer accounts for an estimated 152,000 annual fatalities and 239,000 annual diagnoses. The greatest rates were in Eastern and Central Europe, with 11.4 and 6.0 per 100,000 people, respectively. [45] In 2020, there will probably be 21,750 new instances of ovarian cancer identified in the United States, with an estimated death toll of 13,940. [46]

According to the Globocan 2018 Fact sheet, ovarian cancer is responsible for 3.44 percent (36170) of all cancer cases and ranks third most prevalent among Indian women. Moreover, it accounts for 3.34 percent which is 24,015 of all cancer deaths in India, making it the main cause of cancer mortality among Indian women.[45] Multiple "population-based" cancer registries in India provide estimates of incidence ranging from 0.9 to 8.4 per 100,000 women, after adjusting for age. Ovarian cancer rates tend to rise as women become older. From the age of 35 and up to the age bracket of 55–64, the age specific incidence rate (ASIR) is steadily rising. [47] It is projected that 59,276 new cases of ovarian cancer would be identified in India by the year 2020. By 2035, fresh incidences of ovarian cancer is projected to reach 371,000 annually (a 55 percent increase), while the number of deaths from the disease is projected to rise to 254,000 (a 67 percent increase). [47] According to the "World Ovarian Cancer Coalition's" 2018 Atlas, the incidence/frequency of ovarian cancer in India is second highest in the world. The greatest rates were recorded in Pune and Delhi, two of India's major cities. Ovarian cancer rates have been rising steadily since 1982. [48]

Etiology: Ovarian masses have multifactorial etiology. Ovarian tumours run in families, making it the most common risk factor.

Risk factors: Ovarian cancer risks may be broken down into four categories: diet, medication usage, family history, underlying medical conditions, and heredity. The risk of OC may be greatly increased by dietary variables such coffee, egg, and fat consumption. Hormone treatment, including oestrogen and estrogen-progesterone regimens, has been associated with a higher incidence of ovarian cancer as well. The risk of developing OC may be greatly amplified by diabetes, endometriosis, polycystic ovary syndrome, and certain genetic polymorphisms (such as "BRCA2 N372H rs144848, BSML rs1544410, Fokl rs2228570, MTHFR C677T, P16INK4a, ERCC2 rs13181, MMP-12 rs2276109, and VDRr11568820"). [49] Hormone replacement treatment (HRT) with oestrogen, cigarette usage, and asbestos exposure are the leading causes of epithelial ovarian cancer. [50] One such factor highly correlated with ovarian cancer risk is having a lengthy oestrogen window (Initiating puberty at a younger age and continuing it for a longer period of time). Risk of ovarian cancer is higher in women who have never given birth or who waited more than 35 years before giving birth. Ovarian cancer has a substantial hereditary component. An increased risk and an association with an earlier start of illness are both associated with having two or more first-degree relatives who were diagnosed with ovarian cancer. Increased risk is also associated with a diagnosis of breast cancer in oneself before the age of 40, or a personal history of breast cancer before the age of 50 in combination with a Having a history of breast or ovarian cancer in the family. Women with inherited gene mutations, such as those in the "BRCA1" or "BRCA2" genes (related with "breast ovarian cancer" syndrome) or the mismatch repair genes (associated with "hereditary non-polyposis colorectal" cancer (HNPCC)/Lynch syndrome), have the greatest risk. Carriers of the BRCA1 mutation have a 26%-54% higher likelihood that they may get ovarian cancer

at some point in their lives, whereas those with the "BRCA2" mutation have a 10%-23% greater possibility. [51] Only around 15% of ovarian cancer patients have these risk factors. The administration of hormone replacement therapy. after menopause and the use of some reproductive medicines have been associated with an increased likelihood of developing ovarian cancer. [52]

Ovarian cancer risk is reduced by 30-60% in women who give birth before the age of 25; high parity (number of children); usage of combination oral contraceptives for more than 5 years; and, perhaps, "breast feeding; hysterectomy; and tubal ligation." [53]

Classification:

The three major types of primary ovarian tumours are those that originate in the epithelium, the germ line, or the sex cord stroma. The types of ovarian tumours according to their histology are listed in the following table:

1. Cancers that begin in the surface epithelium and spread to the stroma
    a. Benign, precancerous, and cancerous serous neoplasms
    b. Benign, borderline, and malignant "mucinous" tumours of the endocervical and intestinal types.
    c. Tumors of the uterine lining known as endometrioid may be any one of many types, including benign, borderline, malignant, epithelial-stromal,
    d. Malignant, borderline, and benign clear cell tumours
    e. "Brenner tumour", "Brenner tumour" with borderline malignancy, malignant "Brenner tumour", and "transitional cell" carcinoma are all types of "transitional cell" tumours (non-Brenner type)
    f. Neoplasms involving squamous cells
    g. Tumors with a combination of benign, borderline, and malignant epithelia

h. “Undifferentiated” carcinoma

2. “Sex cord-stromal” tumors
    a. “Granulosa-stromal” cell tumors: “granulosa cell” tumors, “thecoma-fibroma” group
    b. “Sertoli-stromal” cell tumors, “androblastomas”: well-differentiated, “Sertoli-Leydig” cancerous tumour of intermediate development cells, “Sertoli-Leydig” lack of differentiation in tumour cells (sarcomatoid), “retiform”
    c. “Sex cord” tumor with “annular tubules”
    d. “Gynandroblastoma”
    e. “Unclassified”
    f. “Steroid (lipid) cell” tumors: “stromal luteoma”, “Leydig cell” tumor, unclassified
3. “Germ” cell tumors
    a. “Dysgerminoma”: variant-with “syncytiotrophoblast cells”
    b. “Yolk sac” tumors (“endodermal sinus tumors”): “polyvesicular vitelline” tumor, “hepatoid”, “glandular”
    c. “Embryonal” carcinoma
    d. “Polyembryoma”
    e. “Choriocarcinoma”
    f. “Teratomas”: “immature, mature, monodermal, mixed germ cell”
4. “Gonadoblastoma”
5. “Germ cell sex cord”- “stromal tumor of nongonadoblastoma type”
6. Tumors of “rete ovarii”
7. “Mesothelial” tumors
8. Mixed malignancies and tumours of unknown origin

9. Gestational “trophoblastic” diseases
10. Cancers of the soft tissues that are not limited to the ovary
11. Malignant “lymphomas”, “leukemias”, and “plasmacytomas”
12. Unclassified tumors
13. Secondary (metastatic) tumors
14. Tumor-like lesions. [54]

Surface epithelial stromal tumours:

Histologically, the “ovarian surface epithelium” is most comparable to the “mesothelium” that lines the pelvic and abdominal cavities. The surface epithelial stromal tumours which do not exhibit cellular proliferation and invasive behaviour are considered benign tumours. Those tumours which exhibit cellular proliferation but no invasive behaviour are considered to have low malignant potential and are called borderline tumours. Surface epithelial tumours with invasive behaviour are called malignant tumours. Borderline tumours have good prognosis. There are five main forms of these tumours: “serous, mucinous, endometrioid, clear cell, and transitional cell.” Adenocarcinomas not otherwise characterised are a subtype of adenocarcinomas, which are highly aggressive tumours originating on the surface epithelium that lack differentiation.

Serous tumours:

Cells that look like the lining of the fallopian tube line this structure. More than two-thirds of ovarian serous tumours are benign, and they make up a quarter of all benign ovarian neoplasm. They are single-chambered cysts characterised by their thin walls and a watery straw colour. The inner surface is typically smooth; however it may have a few rough papillary projections here and there. The cavity and exterior papillary projections of borderline serous tumours are

more robust and finer. Cysts and solid regions are both present in malignant serous tumours. They deploy many, fine papillary limbs into the cyst cavity and, in certain circumstances, onto the surface of the tumour itself. The majority of malignant serous tumours (66%) are seen on both sides of the body. There is a 76% five-year survival rate for patients with "stage I" tumours, 56% for those with "stage II" tumours, 25% for those with "stage III" tumours, and 9% for those with "stage IV" tumours.

Mucinous tumours:

Ovarian teratomas are tumours composed of epithelial cells that look like the lining of the intestine or the cervix. About three-quarters to eighty-five percent of mucinous tumours in the ovary are benign. In most cases, just one side is affected. These cysts have several small openings and are filled with a viscous mucus.[55] Mucinous tumours on the border with the benign category look like benign tumours but have solid areas and papillar projections into the cyst chamber. 10 to 14% of endocervical tumours are bilateral while less than 10% of intestinal mucinous tumours are bilateral. Malignant mucinous tumours contain more papillary projections larger solid areas, large necrotic areas and haemorrhage. In the ovary, malignant mucinous tumours make up around 5% to 10% of all malignant tumours. Bilaterality occurs in 6%-20% of malignant mucinous tumours. [54]

Endometrioid tumours:

These are epithelial ovarian tumours formed by cells resembling internal lining of uterus. Benign endometrioid tumours are rare, cystic and unilateral. They account for one-fifth of endometrioid tumours may be cystic or solid type. 80% of endometrioid tumours are malignant accounting for 10 to 25% of ovarian cancers. 13 to28% of malignant endometrioid tumours are bilateral.

Clear cell tumours:

Ovarian epithelial tumours look like pegs or hobnails and are generated by transparent, peg-shaped cells. Majority of clear cell tumours are malignant. They can be cystic or solid with one or more polyploid masses protruding into lumen. 4 to 5% of malignant ovarian epithelial tumours are clear cell tumours. Stage I tumour has 69% 5-year survival rate, stage II has 55%, stage III has 14% and stage IV has 4% five-year survival rate. [56]

Transitional tumours:

Ovarian epithelial tumours have cells that look like the lining of an urine bladder. They are rare in occurrence. Benign transitional tumours are small, asymptomatic, solid, nodular and mostly unilateral. In malignant transitional tumours, both solid and cystic regions are present, and the cystic regions include internal papillar or polyploid projections. In 10% of cases, the tumours exist on both sides. [57]

Undifferentiated carcinomas:

Ovarian epithelial neoplasms have little cytoplasmic differentiation and a high nuclear grade, making them highly malignant. Undifferentiated malignancies account for 5% of ovarian malignancies and 14% of all “surface epithelial tumours”. Stage I tumours have 68% 5-year survival rate, “stage II” tumours have 40%, stage III have 17% and stage IV have 6.3% 5-year survival rate.[56]

Sex-cord stromal tumours:

These develop from precursors in the "stromal, granulosa, Sertoli, and Leydig cells". They account for 7% of malignant ovarian tumours and manifest themselves with endocrine symptoms. [58] “Granulosa cell” tumours, a kind of uncommon sex-cord ovarian cancer, develop

from the stromal cells that ordinarily surround the germinal cells in ovarian follicles. Granulosa cell tumours may be either adult, which affects women of reproductive age and above, or "juvenile", which affects children and younger women. Adult "granulosa tumours" often take the form of cysts, with a mix of fluid- and blood-filled spaces and solid areas. They display symptoms of "endometrial hyperplasia" and "endometrial carcinoma." These tumours are not very dangerous, with a 10-year survival rate of 86-96% in "stage I" and 26-49% in later stages. When they develop in children and teenagers, granulosa cell tumours resemble their adult counterparts.

Thecomas:

Ovarian stromal tumours, which are very uncommon, are solid tumours that develop from "stromal cells" that resemble the "theca cells" that normally surround "ovarian follicles." " Oestrogenic" symptoms include postmenopausal uterine haemorrhage, endometrial hyperplasia, and endometrial cancer. They do not pose much of a threat.

Cyst adenofibroma:

Cystic adenofibromas are an uncommon kind of benign epithelial ovarian tumour. Most of these tumours are of the serous kind, however mucinous forms are sometimes observed. [59]

Germ cell tumours:

Ovarian tumours called "germ cell" tumours are thought to originate from primitive "germ cells." They account for around 25% of ovarian tumours overall but for 3-7% of malignant ovarian tumours. [60] Mature cystic teratomas are noncancerous tumours of the germ cells. Cystic and unilocular, with mixed echogenicity reflecting the various fatty, osseous, and fluid components, they account for the vast majority of cystic ocular anomalies. [61]

Dysgerminoma:

Tumors called dysgerminomas include cells that are clones of the embryonic germ line. The tumours are solid and white or greyish white in colour.

Yolk sac tumours:

Germ cell tumours that look like the embryonic yolk sac are called yolk sac tumours or endodermal sinus tumours. They have a high malignancy index, causing them to invade neighbouring tissues and to expand widely throughout the abdomen. Tumors originating in the yolk sac spread rapidly, most often via the lymphatic system. The vast majority of these tumours are solitary on one side of the body. [58]

Embryonal carcinoma:

Tumors that originate in the germ cells, known as embryonal carcinomas, are made up of very basic cells that seem if they were taken straight out of the earliest stages of embryonic development. The most prevalent kind of "germ cell" tumour, embryonal carcinomas are also the least differentiated. Combinations with other types of germ cell tumours, especially tumours of the yolk sac, are prevalent. [58]

Choriocarcinoma:

Choriocarcinomas are tumours of the germ cells that originate in the placenta (specifically, the trophoblast). They have a solid, hemorrhagic look most of the time. The vast majority of these tumours are solitary on one side of the body.

Fibromas:

Ovarian solid tumours are uncommon and are made up of collagen-producing spindled stromal cells. Most incidences of them don't cause any harm. The sonographic appearance of these tumours is that of a round or oval mass with uniform borders. There is a possibility that they exhibit striped acoustic shadows, although this is rare. [62]

Sertoli cell tumours:

Infrequent cell growths that look like "rete ovarii" or "rete testis" are responsible for these tumours.

Sertoli-Leydig cell tumours:

These are made up of both "epithelial" and "stromal testicular" cells in varying quantities. They come in solid, partly cystic, and fully cystic forms and may have internal polyploid or vesicular structures. There are five subtypes.[58]

Tumours that metastasize to ovaries:

Ovarian metastases from "breast, gastric, uterine, and lymphoma" cancers appear as solid tumours on an ultrasound and may be detected by this method. Metastases to the ovary often originate in the colon, rectum, and biliary system, and typically take one of two forms: either a "multilocular-solid metastasis" or a "multilocular metastasis" with "anechoic" or very little echogenicity. [63] Only a small percentage of metastatic tumours have papillary projections. All metastatic tumours have an abundance of blood vessels, although those from the gastrointestinal system, the breast, the uterus, and lymphomas are more vascular than those from the "colon, rectum, and the biliary tract". [63]

Clinical manifestations:

Depending on the tumor's size and stage, ovarian tumours may cause a wide variety of vague symptoms. Bloating, pelvic or abdominal discomfort, early satiety, and urinary symptoms are all signs of a malignant epithelial ovarian tumour.[64] "Diaphragmatic pressure, pleural effusions, and/or a pulmonary embolus" may all contribute to the respiratory symptoms that arise from severe intra-abdominal malignancy with ascites.[65]

Characteristics of malignant ovarian masses:

Ovarian masses that provide the greatest risk often have the following "morphological" features:

- tumours larger than 4 cm in diameter, whether solid, cystic, or mixed
- an abnormal distribution of solid vascularized regions larger than 28 mm in diameter that are not fatty.
- more than 3 mm thick walls and septa, as well as the presence of papillary projection (vegetation), characterise a cystic lesion. [66]

Diagnosis:

Physical examination, laboratory testing, and imaging methods are all used together to identify ovarian masses. Only a small percentage of ovarian tumours are really cancerous, however.

Serum markers:

Epithelial tumours are most often detected with the use of the blood marker "cancer antigen" (CA)-125. Since it may be elevated in premenopausal women due to disorders including peritoneal inflammation and endometriosis, its sensitivity and specificity are both higher in the

postmenopausal women. [67] Human epididymis protein marker is used for differentiation of benign and malignant ovarian masses. [68]

Imaging:

Ovarian mass identification, characterisation and staging all benefit greatly from diagnostic imaging. When evaluating ovarian tumours, ultrasound is by far the most used imaging modality. Using a mixture of grey scale and colour doppler characteristics, the "morphological" structure and vascular architecture of ovarian masses may be examined. Malignant ovarian masses include morphological characteristics such as a thickness larger than 2 to 3 mm, a lack of uniformity in the walls and septa, the presence of solid areas, "papillary projections, ascites, peritoneal nodules, and metastatic lesions". When it comes to vascularization, a tumour with blood flowing through its centre is malignant whereas one with blood flowing through its periphery is benign. [69]

Computed Tomography (CT):

Staging is aided by CT scans since they reveal disease progression indicators including "omental and peritoneal implants, ascites, and lymphadenopathy". [70]

F-FDGPET/CT:

This is used for evaluation of ovarian masses in postoperative follow up patients with suspected recurrence [71].

Risk of Malignancy Index (RMI):

The RMI is a composite metric that is easy to use, helpful in everyday situations, and both sensitive and specific. The RMI is determined by multiplying the "menopausal status score" (M), the "ultrasonographic score" (U), and the "absolute value of blood CA-125" to generate

a simple regression equation.[17] It is possible to derive the modified RMI by plugging the product of the "ultrasound score" (U), the "menopause score" (M), and the "absolute value of blood CA-125" into the following formula:

"RMI = U x M x serum CA – 125"

There are five ultrasonography characteristics that are used to evaluate U, which are "multilocularity (more than bilocular), the presence of solid regions, bilaterality, the presence of ascites, and extraovarian tumours or evidence of metastases." If none or just one of these attributes were found, a score of U was assigned of 1, whereas a score of U was assigned of 3 if two or more of these characteristics were present. The over-50 age group and hysterectomy patients were considered postmenopausal, and were assigned a score of M = 3 on the questionnaire. People who did not meet these standards were classified as having a M = 1 "premenopausal" status. Serum CA-125 (U/ml) levels should be put into the aforementioned calculation as absolute values. [72]

Treatment:

The major focus of treatment for ovarian cancer is to maintain cancer control for as long as possible while reducing the severity of symptoms for the patient. Surgery is often the first line of defense against cancer, while its efficacy varies conditionally, in relation to the degree of illness and other circumstances. "Platinum-based" chemotherapy has shown promise in treating ovarian cancer in its early stages as adjuvant treatment, however this is not universally recommended. Adjuvant systemic platinum-based chemotherapy is often not administered to patients with "stage I", grade I cancer; however, patients with "stage II" or higher disease and/or certain histologies "(such as HGSC and clear-cell carcinoma)" sometimes get this treatment after surgery. [73]

In the case of ovarian cancer, "neoadjuvant chemotherapy" (NACT; the administration of chemotherapy prior to surgery) is a promising option to primary surgical “cytoreduction” for patients who are either too sick to have surgery immediately or whose cancer load is too great for “macroscopic” full resection. [74]

Platinum sensitive disease:

When a patient has recurrent ovarian cancer and their cancer is platinum sensitive, a platinum-based regimen is often used again. The development of a life-threatening allergy to platinum-based chemotherapy is a serious risk associated with its repeated usage. [75] In order to effectively treat platinum-sensitive ovarian cancer, several different therapy combinations are being studied. These include “paclitaxel and carboplatin”[76], “carboplatin and pegylated liposomal doxorubicin” [77] and “carboplatin and gemcitabine”[78].

Platinum resistant disease:

The use of bevacizumab in conjunction with "paclitaxel, pegylated liposomal doxorubicin, or topotecan" has been authorised for use by the Food and Drug Administration and the European Medicines Agency in the first platinum-resistant setting for patients with “platinum-resistant” cancer. This approval was based on the outcomes of the AURELIA study. [79]

Complications:

Ovarian cysts almost always lead to one of the three following potential consequences.

- Rupture
- Haemorrhage
- Torsion. [80]

Ovarian torsion:

In most instances of "ovarian torsion", both the ovary and the accompanying fallopian tube are affected, which typically manifest themselves during the first three decades of life. A cyst or tumour in the ovary is more likely to be the cause. [81]

Staging of ovarian tumours:

Ovarian tumours have two different staging systems that explain their development and growth. The TNM "(tumour, node, metastasis)" and FIGO "(International Federation of Gynecology and Obstetrics)" systems are two of the most often used classifications. [82]

"Stage I ovarian cancer" describes tumours contained inside the ovaries; "stage II" describes "pelvic extension or primary peritoneal cancer"; "stage III" describes dissemination to the "peritoneum beyond the pelvis and/or metastasis to the retroperitoneal lymph nodes"; and "stage IV" describes "distant metastasis."

Table 1: The following table gives FIGO system of ovarian tumour staging using MRI imaging:

Stage I	Cancer affecting just the ovaries
IA	Capsule unbroken, no visible tumour, negative washings; tumour confined to a single ovary.
IB	Involvement of both ovaries in the tumour, else same to IA
IC	Malignancy affecting just one or both ovaries IC1 "Surgical Spill" IC2 Tumor growth on the ovarian surface or premature capsule rupture IC3 Cancerous cells seen in peritoneal fluids or ascites
Stage II	"Primary" peritoneal cancer or tumour affecting a malignancy originating in either ovary(s) and/or afterwards affecting the pelvic region (below the pelvic brim).
II A	"Extension and/or implant on uterus and/or Fallopian tubes"
II B	Broadening to include more "intraperitoneal tissues" in the pelvis
Stage III	If the tumour has migrated from the ovary(s) to the "peritoneum outside the pelvis and/or metastasized to the retroperitoneal lymph nodes", as determined by cytology or histology.
III A	Extensive micrometastasis beyond the pelvis and/or positive "retroperitoneal lymph nodes" III A1 Lymph node positivity is limited to the "retroperitoneal" region. III A2 "Positive retroperitoneal lymph nodes" indicative of microscopic, extra pelvic (above the brim) peritoneal involvement.
III B	"Peritoneal metastasis" >=2 cm in size, located outside the pelvis, may or may not include the "retroperitoneal lymph nodes". Incorporates enlargement of the liver/spleen capsule.
III C	"Peritoneal metastasis" >2 cm in size, outside the pelvis, "with or without positive retroperitoneal lymph nodes". Including the enlargement of the liver and spleen capsule.
Stage IV	Metastases beyond the "peritoneal" cavity
IV A	Cytologically positive "pleural effusion"
IV B	Parenchymal metastases to the liver and/or spleen, as well as metastases to other abdominal organs "(including inguinal lymph nodes and lymph nodes outside of the abdominal cavity)"

Relevant studies:

Laul,P.,et al.[83] compared the histology of ovarian masses with their presenting symptoms, ultrasonography findings, and levels of tumour markers. While malignant ovarian masses were more frequent in post-menopausal women, the majority of ovarian masses in this research occurred in women of childbearing age (between 21 and 40 years old). Among the 11 ovarian tumours, two were dysgerminoma and two were immature teratoma, both occurring in young women (10–20 years old).

Kamath et al.[84] Women with adnexal masses were included in prospective observational research that evaluated the accuracy of RMI 2 in differentiating between healthy and cancerous adnexal tissue. The RMI was above 200, indicating malignancy, in 72% of patients, and under 200, indicating benign disease, in 28% of patients. Malignant lesion detection sensitivity for the RMI was 84%, while specificity was 67%. It seems that RMI is a helpful, reliable, and applicable tool in the primary assessment of patients with pelvic masses and is useable in referral of appropriate individuals for centralised surgical treatment.

Dora et al.[72] evaluated RMI-3 for its ability to preoperatively distinguish between benign and malignant masses, and for the optimal cut-off value to be revealed. Analyzing the RMI in comparison to "Ultrasound score, CA-125, or menopausal score" with a cut-off point of 236 demonstrates very good "sensitivity (72.5%), specificity (98.2%), positive predictive value (98.1%), negative predictive value (74.7%), and diagnostic accuracy (84.13%). "

Priya F. et al.[85] compared the histology report for an ovarian mass with the patient's clinical data, USG morphology, and colour doppler indices. Ovarian cancer may be diagnosed with 92.31 percent sensitivity using a combination of clinical and USG with Doppler, and 95.95 percent specificity. This research demonstrates the therapeutic value of USG and Doppler in distinguishing between “benign and malignant” ovarian tumours.

Baru et al.[86] research found that most ovarian neoplasms were diagnosed in women of reproductive age; nevertheless, ovarian malignancy may strike anybody at any time, and abdominal symptoms are the sole indicator of the illness. However, stomach symptoms, in conjunction with tumour indicators such blood "CA-125" and ultrasonography with doppler of the pelvic and abdominal area, may serve as a yardstick for early identification of malignant ovarian tumours, despite the lack of a definitive universal screening methodology.

Javdekar R[87] reported that RMI with a "cutoff value of 200" was found to have a "sensitivity of 70.5% and a specificity of 87.8%" in a prospective cohort analysis. The results of the research indicated that RMI is an effective method for determining whether adnexal masses are benign or malignant.

Al-Musalhi et al.[88] assessed the reliability of CA-125 and the RMI in ovarian cancer diagnosis. Validity studies have shown that CA-125 and RMI can both be used to diagnose ovarian cancers. CA-125 is more sensitive, while RMI is more accurate. When used together, CA-125 and RMI may be superior for diagnosing ovarian cancer in its most aggressive forms, but CA-125 alone may be more reliable for ruling out this diagnosis.

Shaik, M., et al. [89] analysed data at a tertiary care center of the prevalence of ovarian lesions and their clinico-histological characteristics. The most frequent kind of ovarian tumour was an epithelial tumour. The peak incidence rate occurs between the ages of 40 and 59. The greatest incidence of ovarian cancer was seen in women over the age of fifty.

Kumari, A., et al. [90] examined the epidemiological and pathologic characteristics of ovarian tumours. Malignant tumours were more likely to develop at a younger age, with relatively long-lasting symptoms lasting more than a year, and at a more advanced stage of illness. This highlights the need of evaluating women of all ages thoroughly and doing appropriate tests to rule out ovarian cancer as soon as possible.

Sharma, P., et al.[91] examined the age distribution, clinical presentation, and histological and morphological variations of ovarian tumours. Over malignant or borderline tumours, benign ovarian tumours dominate the disease. Most ovarian tumours are surface epithelial tumours, followed by germ cell tumours in terms of frequency. Tumors may be either benign or malignant, and knowing the difference is crucial for successful treatment. Future research should consist of similar studies, but with larger samples.

Gaikwad, S, L., et al.[92] evaluated the prevalence of malignant and benign lesions in a rural Indian tertiary care facility. Seventy-five percent of all instances were caused by tumours originating in the surface epithelium, with the remaining cases coming from "germ cell tumours (20.8%) and sex cord stromal tumours (4.8%)".

Patel, A, S., et al.[93] examined the prevalence of ovarian cancers across age groups and their histological range in connection to clinical outcomes using the WHO classification. Malignant ovarian tumours peaked at two different ages in a bimodal distribution. On average, benign tumours affected people of all ages. For therapeutic and prognostic purposes, a precise histological diagnosis and staging is crucial.

Fonseca,M,N., et al.[94] conducted a "clinico-histopathological" study of ovarian cancer at a prominent hospital. Parous women accounted for more than half of the tumours found. Regardless of the kind of tumour, abdominal pain was the most prevalent presenting clinical complaint. Hemorrhaging inside the cyst was the most prevalent serious consequence.

Prakash, A., et al.[95] determined the common causes of ovarian mass lesions sent in for histological analysis at a major hospital in Hyderabad, Telangana, India. The vast majority of ovarian tumours sent in for diagnosis were asymptomatic and localised to one side. Patients typically ranged in age from 30-60.

Gupta, A., et al.[96] examined the signs, symptoms, and imaging results of paraovarian cysts. The majority of women who develop paraovarian cysts do so while they are sexually active, and they manifest as an adnexal mass. Paraovarian cysts should be distinguished from ovarian cysts using ultrasound, the diagnostic modality of choice. Histopathology revealed that the vast majority of the lesions were benign cysts.

Kant, R, H., et al.[97] aimed to determine whether ovarian cyst patients in the Kashmir valley have a distinct "clinical and histological profile" from those in other parts of the world. Malignant ovarian tumours were uncommon compared to benign ones across all age ranges. Our findings of a greater prevalence of malignancy than previous studies suggest geographical variability and emphasise the need to discover risk factors that are unique to certain geographic areas.

Sawant,A., et al.[98] examined the histological characteristics and incidence of ovarian lesions in a major medical centre and reported 75.7% were considered benign, 6.1%intermediate, and 18.2%, malignant.

Mankar, D., et al.[99] explored the rates of occurrence of several histological subtypes of ovarian cancer. Overall, benign ovarian tumours were more prevalent than malignant ones. The majority of ovarian tumours were found to be surface epithelial tumours upon histological examination. Patients often delay medical attention due to the indistinct nature of their symptoms. Since this is the case, there is an immediate need to create tools for the early detection of ovarian neoplasia. Variations in the relative incidence of various ovarian tumours between regions underscore the need of determining risk factors that are unique to each area.

Maurya, G., et al.[100] studied the several ovarian disorders seen at this rural India tertiary care centre and rated their prevalence and morphological pattern. There were more cases of non-neoplastic ovarian lesions than neoplastic ones. In all age ranges, tumours originating in the

surface epithelium were the most prevalent. Among ovarian cancers, serous adenocarcinoma was by far the most frequent.

Modepalli, N., et al.[101] investigated the clinical and histological pattern of ovarian surface epithelial tumours and compared their occurrences to those found in previous investigations. Some 90.6% of tumours were located on one side of the body, whereas 9.4% were found on both sides. There was a significant difference in the occurrence of tumours on the right (59.8 percent) and the left side (40 percent) (40.14 percent). Tumors made up 82.3% of the total, with 12.1% being malignant and 5.7% being intermediate.

Rojna, R., [102] compared the rates of distinct histopathologic subtypes of "adnexal masses" across ranges of ages, and assessed the reliability of preoperative examination in order to detect ovarian cancer. The most frequent adnexal mass originated in the ovaries. Aging postmenopausal women with a high body mass index were more likely to get cancer. For the diagnosis of epithelial ovarian cancers, RMI demonstrated modest correlation.

Parmar, P., et al.[103] analysed ovarian tumours at a tertiary care facility for their histological trends, age distribution, and clinicopathological association. The research found that epithelial surface tumours were the most prevalent kind of ovarian tumour. All ages are susceptible to benign tumours, although malignant ones are far rarer.

Lal, S, R, B., et al.[104] identified the frequency and location of ovarian lesions. In benign tumours, luteal cysts were often seen. Most benign tumours were serous cystadenomas, but serous cystadenocarcinomas were prevalent malignant tumours.

Lacunae in Literature:

Ovarian tumours are a frequent gynaecological issue that need precise diagnosis. A better prognosis and more effective treatment options are possible if ovarian cancer is caught in the

earliest stages. The 5-year survival rate for any tumour, regardless of grade or differentiation, is dramatically improved if detected in stages I and II as opposed to stages III and IV. Diagnostic methods such as ultrasound findings, clinical information, and combinations of these are used to determine the likelihood of ovarian malignancy. Other tools include the blood biomarkers "cancer antigen 125 (CA125) and human epididymis protein 4 (HE4)". In order to improve early diagnosis of ovarian masses and begin suitable therapy strategy for improved prognosis, there are studies in the literature associating different clinical presentations, ultrasonography characteristics, and "tumour marker" levels with histology of ovarian masses. The information gathered in this research will supplement the current body of knowledge.

MATERIALS & METHODS

Source of data: The study was conducted on women who were diagnosed with ovarian mass by clinical examination and confirmed by ultrasonography and undergoes surgery at RL Jalappa Hospital, Kolar during the study period.

Study design: Prospective observational Study

Study period: Jan 2021 to Dec 2022

Method of collection of data: a prospective observational study was conducted in patients coming to department of obstetrics and gynaecology OPD at R L JALAPPA HOSPITAL TAMAKA KOLAR attached to SRI DEVRAJ URS MEDICAL COLLEGE under SRI DEVRAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH.

Relevant information like age, parity, family history of cancer, personal history of previous malignancies, symptoms and the duration of symptoms was taken from patient. Leading symptoms such as abdominal mass, abdominal swelling /discomfort, abdominal pain, gastrointestinal symptoms, urinary symptoms, generalized malaise and fatigue were recorded.

All patients were undergone routine physical examination. Particular attention was paid to breast examination, lymphadenopathy, abdominal examination and pelvic examination. Preoperative evaluation would include Complete blood count, Renal function test, Liver function test, Chest Xray, serology, Ultrasonagraphy, CA 125, Risk malignancy index. In relevant cases CT, MRI, Biochemical markers(LDH, β hcG, Alpha-Feto protein) was done.

Laparotomy or minimally invasive surgery were done in all cases. The type of surgical procedure done either unilateral salpingo-oophorectomy, unilateral salpingo-ophorectomy with wedge resection of the cotralateral ovary, total transabdominal hysterectomy and unilateral salpingo-oophorectomy, total trans abdominal hysterectomy with bilateral salpingo-oophorectomy, with ometectomy and debulking surgery.

The extracted specimen was sent to pathology department for histo-pathological examination.

On receiving the specimen, gross features such as size, shape, colour, external appearance, findings on cut section and contents would be noted. The cut specimen was fixed in 10% formalin for 24-48 hours. After formalin fixation multiple bits are taken from representative areas of tumours and the accompanying tissues. These tissues were processed and stained with haematoxylin and eosin. Detailed microscopic examination of the tumour was done to arrive at histo-pathological diagnosis.

After arriving at the histopathological diagnosis, combined correlation was made with clinical and radiological profile.

Inclusion Criteria:

All the cases with ovarian masses on clinical examination which is confirmed by imaging techniques are included in this study.

Age group included will be from 13 to 70years

Exclusion Criteria:

Uterine masses including fibroids, adenomyosis, endometrosis

Tubo-ovarian mass

Sample size: 40 cases

Methodology: This study was conducted in the Department of Obstetrics and Gynaecology at R L JALAPPA HOSPITAL TAMAKA KOLAR attached to SRI DEVRAJ URS MEDICAL

COLLEGE under SRI DEVRAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH. Detail history of patient like age, menstrual status, obstetric history, relevant medical and family history and presenting symptoms were noted. General examination, systemic and pelvic examination was done. Ultrasonography was done in all the patients, Risk malignancy index is calculated and CT scan and MRI were done when required. All necessary laboratory investigations were performed. CA-125 levels were evaluated in all patients. The biochemical markers for further diagnosis like Alpha-Feto protein, LDH, β-HCG are also investigated when required. Histo-pathological examination of the surgically removed tissue was processed in the Department of Pathology of our institute. The data was collected in excel sheet and analyzed by descriptive statistics

Investigations:

Complete blood count, Renal function test, Liver function test, Chest Xray, serology, Ultrasonagraphy, CA 125.

In relevant cases CT MRI, Biochemical markers (LDH, BETA hcG, Alpha-Feto protein) was done.

Histoplathological evaluation was done for all the cases.

Statistical methods:

Histopathological report was considered as Primary outcome parameter. Age group, Parity, Menstrual history, Risk Malignancy Index, etc., were considered as explanatory parameters. Mode of presentation, USG Features, etc., were considered as study relevant variables.

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram and pie diagram.

The association between explanatory variables and categorical outcomes was assessed by cross tabulation and comparison of percentages. Odds ratio along with 95% CI is presented. Chi square test was used to test statistical significance.

Histopathological report was considered as gold standard. Risk Malignancy Index was considered as screening test. The sensitivity, specificity, predictive values and diagnostic accuracy of the screening test along with their 95% CI were presented.

P value < 0.05 was considered statistically significant. Data was analysed by using coGuide software, V.1.01.[105]

OBSERVATIONS AND RESULTS

RESULTS:

A total 40 subjects were included in the final analysis.

Table 2: Descriptive analysis of age groups in the study population (N=40)

Age groups	Frequency	Percentage
<=40 years	9	22.50%
>40 years	31	77.50%

Among the study population, 9 (22.50%) participants were aged between <=40 years and remaining 31 (77.50%) were aged between >40 years. (Table 2)

Table 3: Descriptive analysis of parity in the study population (N=40)

Parity	Frequency	Percentage
NULLIPAROUS	3	7.50%
PRIMIPAROUS	11	27.50%
MULTIPAROUS	26	65.00%

Among the study population, 3 (7.50%) participants were nulliparous, 11 (27.5%) participants were primi and 26 (65%) participants were multi parity. (Table 3 & Figure 2)

Figure 2: Bar chart of parity in the study population (N=40)

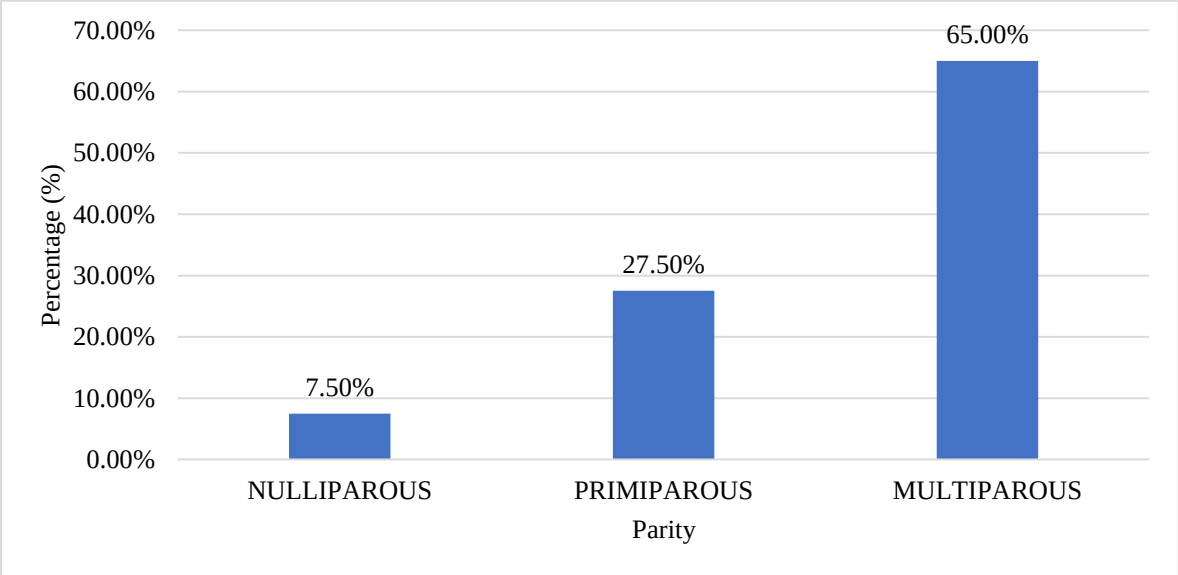


Table 4: Descriptive analysis of mode of presentation in the study population (N=40)

Mode of presentation	Frequency	Percentage
PAIN ABDOMEN	23/40	57.50%
MENSTRUAL IRREGULARITIES	16/40	40.00%
MASS PER ABDOMEN	30/40	75.00%
ABDOMINAL DISTENTION	13/40	32.50%
WHITE DISCHARGE	11/40	27.50%
BLADDER DISTURBANCES	13/40	32.50%

Note: In BOWEL there all entries are No only

Among the study population, 23 (57.50%) participants were in Pain in abdomen as mode of presentation, 16 (40%) participants were in Menstrual irregularities mode of presentation, 30 (75%) participants were in Mass per abdomen mode of presentation, 13 (32.5%) participants were in abdominal distention mode of presentation, 11 (27.5%) participants were in White discharge mode of presentation and remaining 13 (32.5%) participants were in BLADDER disturbances as mode of presentation. (Table 4)

Table 5: Descriptive analysis of GROSS Appearance in the study population (N=40)

GROSS Appearance	Frequency	Percentage
CYSTIC	13	32.50%
SOLID	13	32.50%
COMPLEX	14	35.00%

Among the study population, 13 (32.50%) participants had cystic gross appearance, 13 (32.50%) participants had solid gross appearance and 14 (35%) participants had complex gross appearance. (Table 5 & Figure 3)

Figure 3: Bar chart of GROSS Appearance in the study population (N=40)

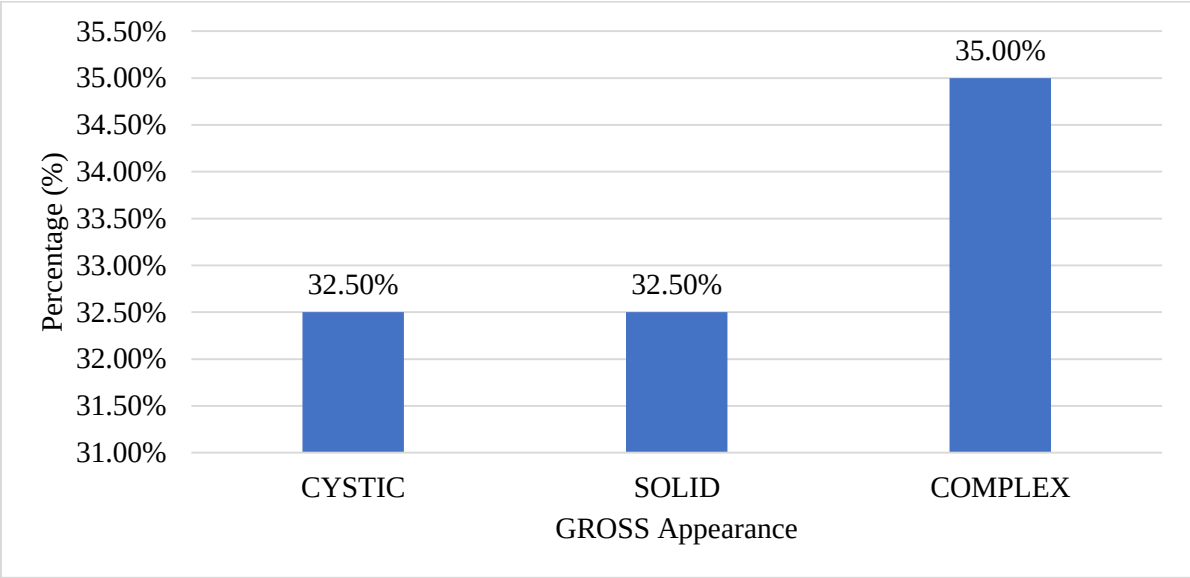


Table 6: Descriptive analysis of Unilateral/Bilateral in the study population (N=40)

Unilateral/Bilateral	Frequency	Percentage
UNILATERAL	20	50.00%
BILATERAL	20	50.00%

Among the study population, 20 (50%) participants were reported unilateral and remaining 20 (50%) participants were bilateral tumours. (Table 6)

Table 7: Descriptive analysis of size of tumour groups in the study population (N=40)

Size of tumour groups (cm)	Frequency	Percentage
0-10	1	2.50%
11-19	19	47.50%
20-29	11	27.50%
30 and above	9	22.50%

Among the study population, 1 (2.50%) participant was with the 0-10 cm size of tumour, 19 (47.50%) participants were with11-19, 11 (27.5%) participants were with 20-29 and remaining 9 (22.5%) participants were with above 30 as size of tumour group (Table 7)

Table 8: Descriptive analysis of CAI25 in the study population (N=40)

CAI25	Frequency	Percentage
<35U/ml	15	37.50%
>35U/ml	25	62.50%

Among the study population, 15 (37.50%) participants were in <35U/ml CAI25 group and remaining 25 (62.50%) participant were in >35U/ml CAI25 group. (Table 8 & Figure 4)

Figure 4: Bar chart of CAI25 in the study population (N=40)

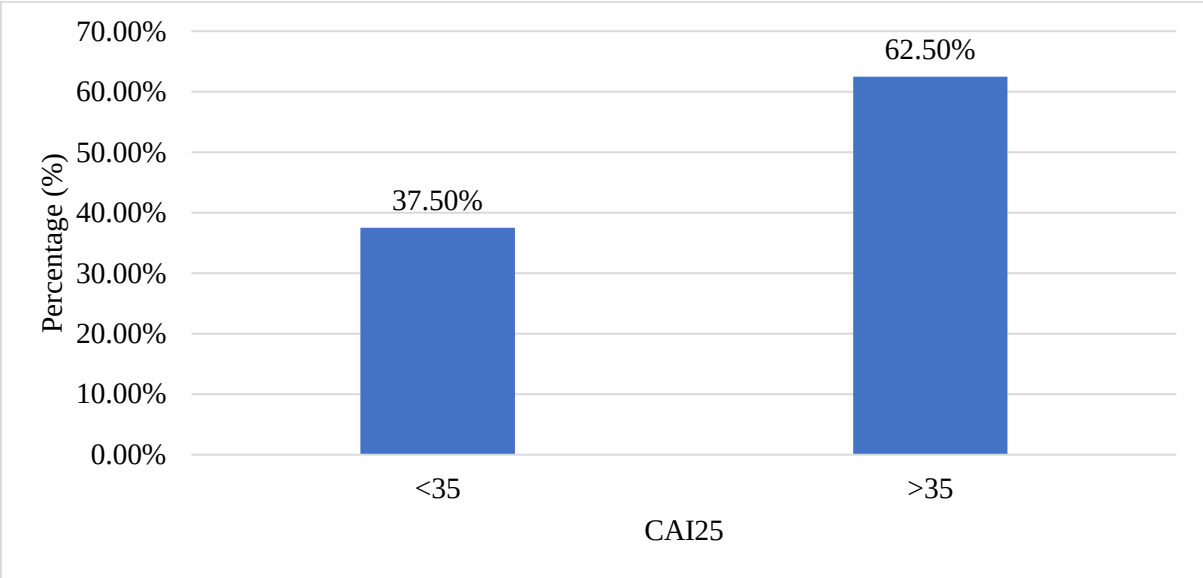


Table 9: Descriptive analysis of USG Features in the study population (N=40)

USG Features	Frequency	Percentage
CYSTIC	21/40	52.50%
SOLID	10/40	25.00%
BOTH	11/40	27.50%
ASCITIS	9/40	22.50%
MULTILOCULAR CYST	12/40	30.00%
UNILOCULAR CYST	11/40	27.50%
THIN SEPTATIONS	16/40	40.00%
THICK SEPTATIONS	9/40	22.50%

Among the study population, 21 (52.50%) participants had cystic feature, 10 (25%) participants had in solid feature, 11 (27.50%) participants had both feature, 9 (22.50%) participants had ascites feature, 12 (30%) participants had multilocular cyst feature, 11 (27.5) participants had unilocular cyst feature, 16 (40) participants had thin septations feature and remaining 9 (22.50%) participants had thick septations. (Table 9)

Table 10: Descriptive analysis of Menstrual History in the study population (N=40)

Menstrual History	Frequency	Percentage
PERIMENOPAUSAL	12	30.00%
POSTMENOPAUSAL	28	70.00%

Among the study population, 12 (30%) participants had perimenopausal history and 28 (70%) participants had postmenopausal history. (Table 10 & Figure 5)

Figure 5: Bar chart of Menstrual History in the study population (N=40)

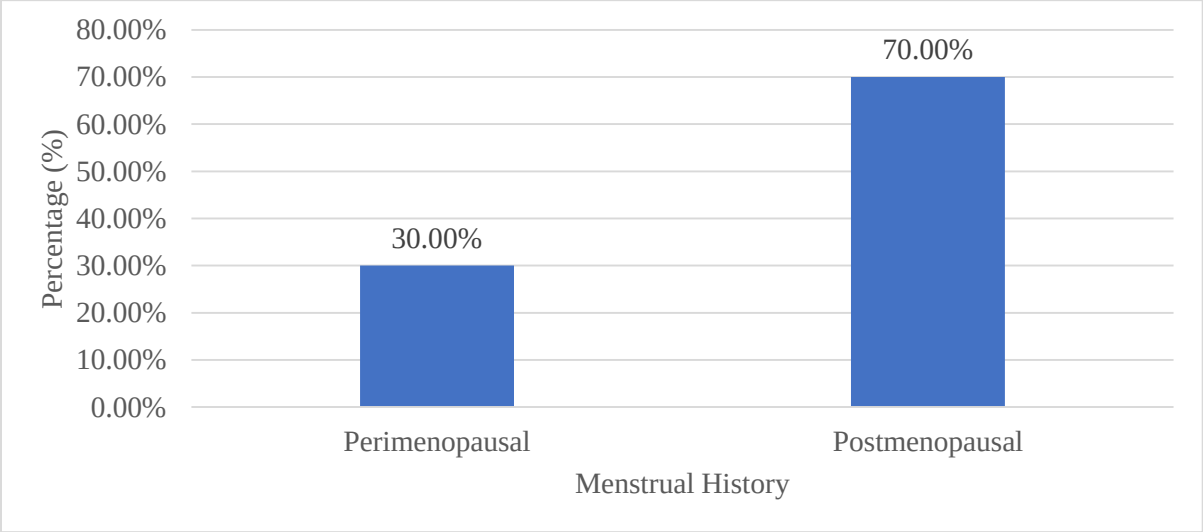


Table 11: Descriptive analysis of Risk Malignancy Index in the study population (N=40)

Risk Malignancy Index	Frequency	Percentage
<25	12	30.00%
25-250	9	22.50%
>250	19	47.50%

Among the study population, 12 (30%) participants had <25 RMI, 9 (22.50%) participants had 25-250 RMI and remaining 19 (47.5%) participants reported >250 RMI.

(Table 11 & Figure 6)

Figure 6: Pie chart of Risk Malignancy Index in the study population (N=40)

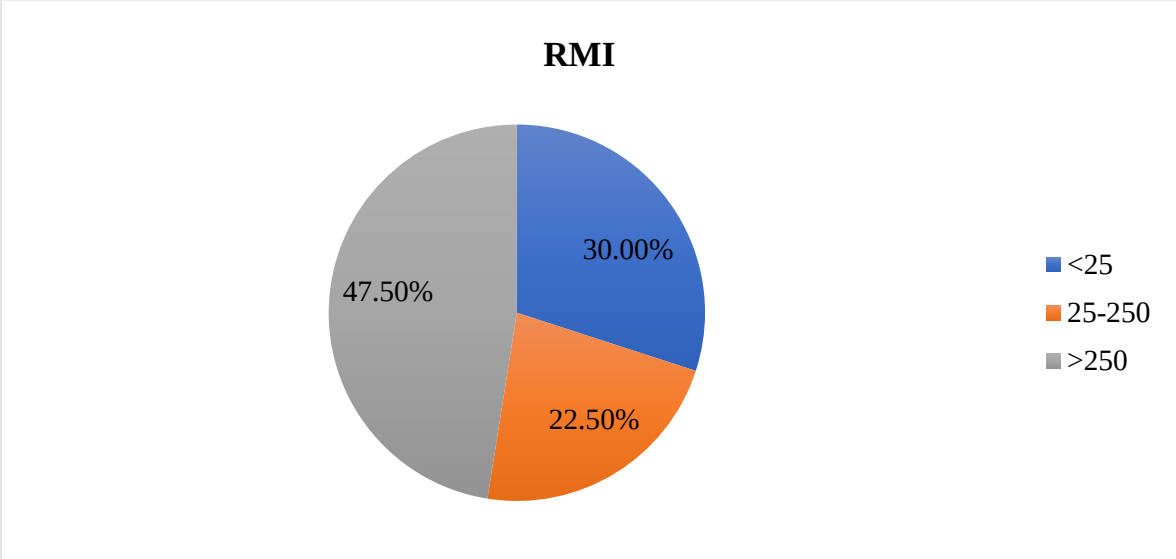


Table 12: Descriptive analysis of USG Score in the study population (N=40)

USG Score	Frequency	Percentage
USG SCORE 0	7	17.50%
USG SCORE 1	14	35.00%
USG SCORE 3	19	47.50%

Among the study population, 7 (17.5%) participants had USG score 1, 14 (35%) participants had USG score 2 and remaining 19 (47.5%) participants had USG score 3. (Table 12)

Table 13: Descriptive analysis of Histopathological report in the study population (N=40)

Histopathological report	Frequency	Percentage
BENIGN	23	57.50%
MALIGNANT	17	42.50%

Among the study population, 23 (57.5%) participants were benign and remaining 17 (42.5%) participants were malignant as per the histopathological report. (Table 13 & Figure 7)

Figure 7: Pie chart of Histopathological report in the study population (N=40)

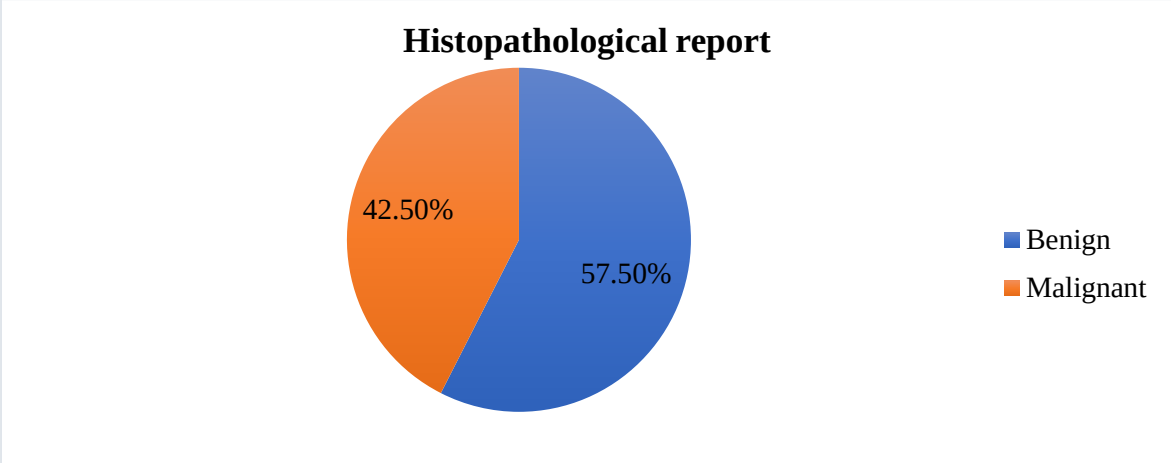


Table 14: Comparison of Histopathological report with age group in the study population (N=40)

Age group	Histopathological report		Chi square value	P value
	Benign	Malignant		
<=40 years (N = 9)	3 (33.33%)	6 (66.67%)	2.78	0.1338
>40 years (N = 31)	20 (64.52%)	11 (35.48%)		

Out of 9 participants in <=40 years, 3 (33.33%) were benign and another 6 (66.67%) were malignant. Out of 31 participants in >40 years age group, 20 (64.52%) were benign and another 11 (35.48%) were malignant. The difference in the proportion of benign and malignant cases across age groups was statistically not significant (P value 0.1338) (Table 14 & Figure 8)

Figure 8: Stacked bar chart of comparison of Histopathological report with age group in the study population (N=40)

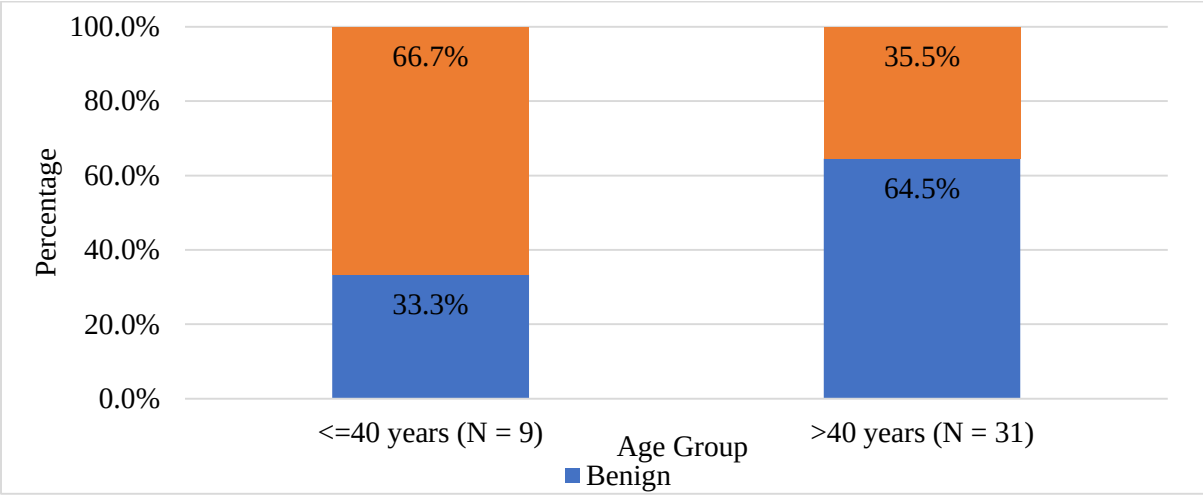


Table 15: Comparison of Histopathological report with Parity in the study population (N=40)

Parity	Histopathological report		Chi square value	P value
	Benign	Malignant		
NULLIPAROUS (N = 3)	2 (66.67%)	1 (33.33%)	0.41	0.8127
PRIMIPAROUS (N = 11)	7 (63.64%)	4 (36.36%)		
MULTIPAROUS (N = 26)	14 (53.85%)	12 (46.15%)		

Out of 3 Nulliparous participants, 2 (66.67%) were benign and another 1 (33.33%) was malignant. Out of 11 participants with Primiparous, 7 (63.64%) were benign and another 4 (36.36%) were malignant. Out of 26 participants with Multiparous, 14 (53.85%) were benign and another 12 (46.15%) were malignant. The difference in the proportion of benign and malignant cases across parity was statistically not significant (P value 0.8127) (Table 15 & Figure 9)

Figure 9: Stacked bar chart of comparison of Histopathological report with Parity in the study population (N=40)

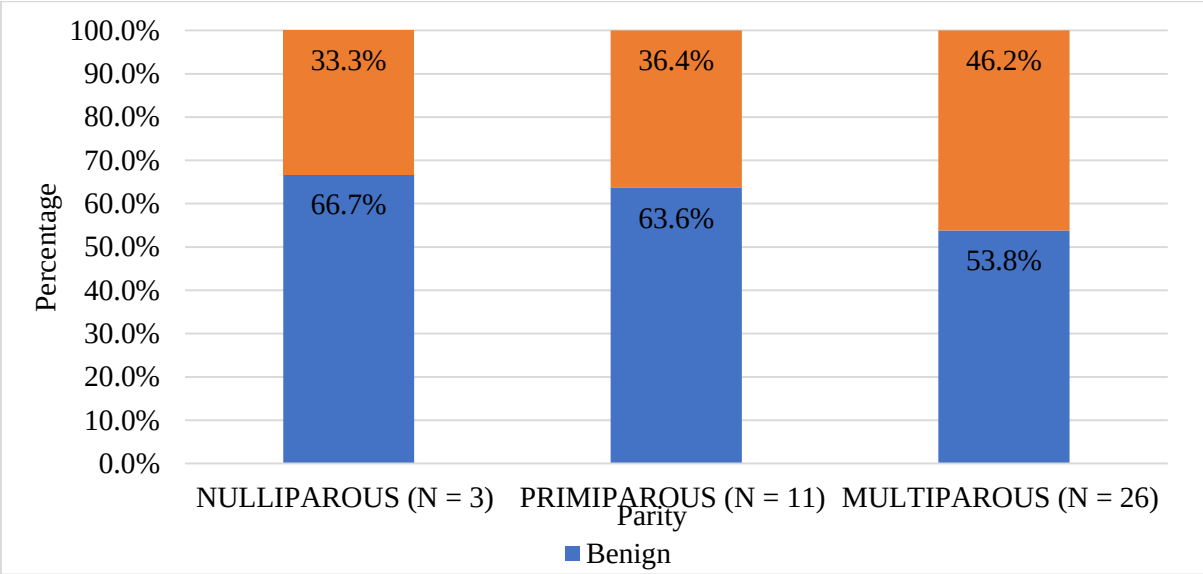


Table 16: Comparison of Histopathological report with Unilateral/Bilateral in the study population (N=40)

Unilateral/Bilateral	Histopathological report		Chi square value	P value
	Benign	Malignant		
UNILATERAL (N = 20)	14 (70.00%)	6 (30.00%)	2.56	0.1098
BILATERAL (N = 20)	9 (45.00%)	11 (55.00%)		

Out of 20 participants of Unilateral tumour, 14 (70%) were benign and another 6 (30%) were malignant. Out of 20 participants of Bilateral tumour, 9 (45%) were benign and another 11 (55%) were malignant. The difference in the proportion of benign and malignant cases between Unilateral/Bilateral tumour was statistically not significant (P value 0.1098) (Table 16 & Figure 10)

Figure 10: Stacked bar chart of comparison of Histopathological report with Unilateral/Bilateral in the study population (N=40)

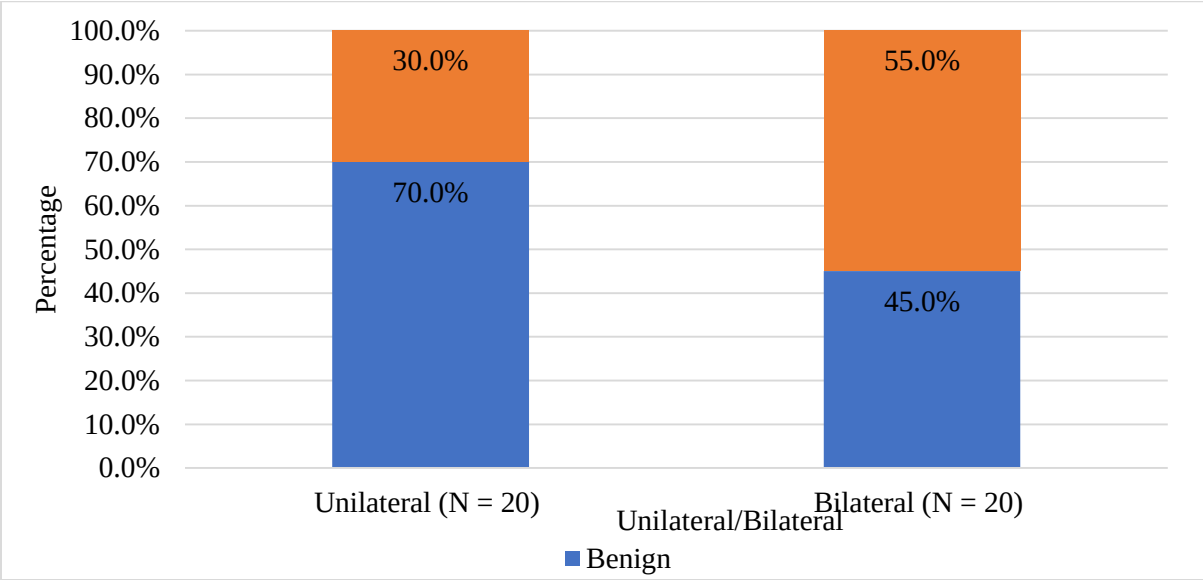


Table 17: Comparison of Histopathological report with size of the tumour (cms) in the study population (N=40)

Size of the tumour(cms)	Histopathological report		Chi square value	P value
	Benign	Malignant		
0-10cms (N = 1)	1 (100.00%)	0 (0.00%)	*	*
11-19cms (N = 19)	11 (57.89%)	8 (42.11%)		
20-29cms (N = 11)	6 (54.55%)	5 (45.45%)		
30cms and above (N = 9)	5 (55.56%)	4 (44.44%)		

No statistical test was applied- due to 0 subjects in the cells

Out of 19 participants with 11-19cms size of tumour, 11 (57.89%) were benign and another 8 (42.11%) were malignant. Out of 11 participants with 20-29cms size of tumour, 6 (54.55%) were benign and another 5 (45.45%) were malignant. Out of 9 participants with 30 cm and above size of tumour, 5 (55.56%) were benign and another 4 (44.44%) were malignant. (Table 17)

Table 18: Comparison of Histopathological report with CAI25(U/ml) in the study population (N=40)

CAI25(U/ml)	Histopathological report		Chi square value	P value
	Benign	Malignant		
<35U/ml (N = 15)	10 (66.67%)	5 (33.33%)	0.83	0.3637
>35U/ml (N = 25)	13 (52.00%)	12 (48.00%)		

Out of 15 participants with <35U/ml CAI25 group, 10 (66.67%) were benign and another 5 (33.33%) were malignant. Out of 25 participants with >35U/ml CAI25 group, 13 (52%) were benign and another 12 (48%) were malignant. The difference in the proportion of benign and malignant cases between CAI25 group was statistically not significant (P value 0.3637) (Table 18 & Figure 11)

Figure 11: Stacked bar chart of comparison of Histopathological report with CAI25 in the study population (N=40)

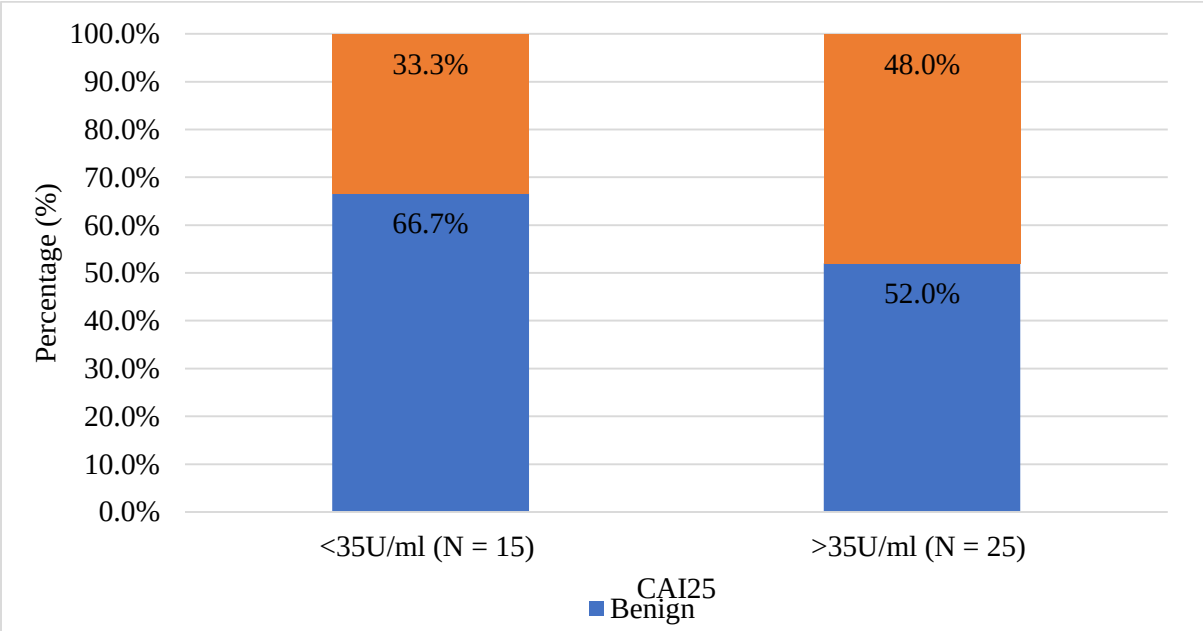


Table 19: Comparison of Histopathological report with menstrual history in the study population (N=40)

Menstrual history	Histopathological report		Chi square value	P value
	Benign	Malignant		
PERIMENOPAUSAL (N = 12)	5 (41.67%)	7 (58.33%)	1.76	0.1848
POSTMENOPAUSAL (N = 28)	18 (64.29%)	10 (35.71%)		

Out of 12 participants with Perimenopausal menstrual history, 5 (41.67%) were benign and another 7 (58.33%) were malignant. Out of 28 participants with Postmenopausal menstrual history, 18 (64.29%) were benign and another 10 (35.71%) were malignant. The difference in the proportion of benign and malignant cases between menstrual history was statistically not significant (P value 0.1848) (Table 19 & Figure 12)

Figure 12: Stacked bar chart of comparison of Histopathological report with Menstrual History in the study population (N=40)

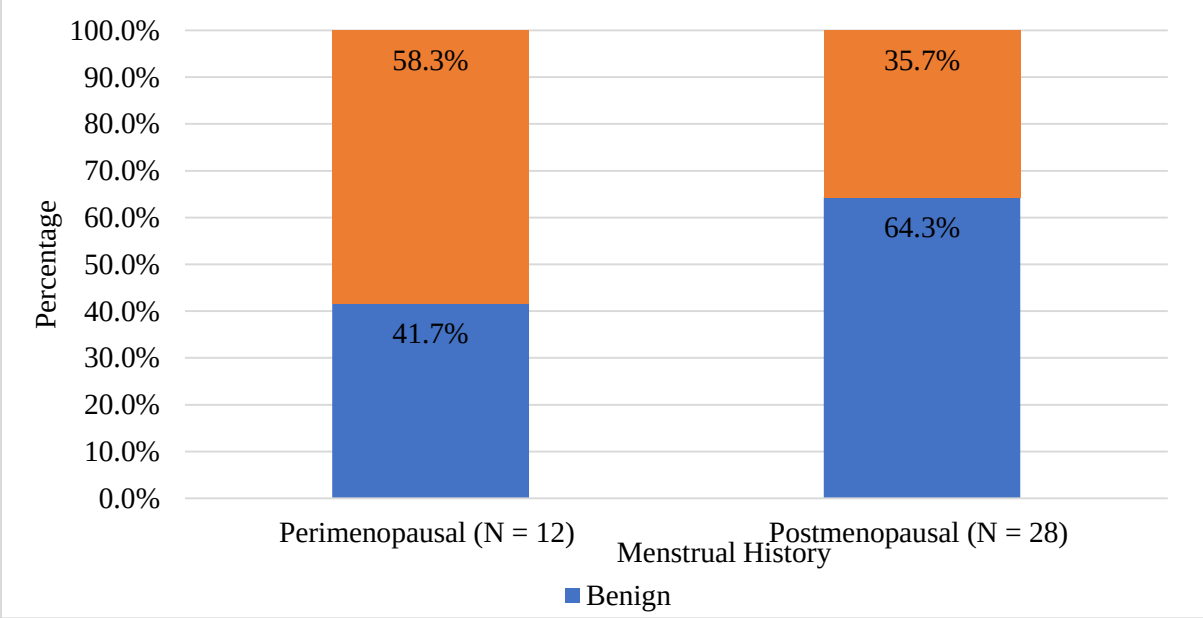


Table 20: Comparison of Histopathological report with Risk Malignancy Index in the study population (N=40)

Risk Malignancy Index (RMI)	Histopathological report		Chi square value	P value
	Benign	Malignant		
<25 (N = 12)	10 (83.33%)	2(16.67%)	6.90	0.0317
25-250 (N = 9)	6 (66.67%)	3 (33.33%)		
>250 (N = 19)	7 (36.84%)	12 (63.16%)		

Out of 12 participants with <25 RMI, 10 (83.33%) were in benign and another 2 (16.67%) were malignant. Out of 9 participants with 25-250 RMI, 6 (66.67%) were benign and another 3 (33.33%) were malignant. Out of 19 participants with >250 RMI, 7 (36.84%) were benign and another 12 (63.16%) were in malignant. The difference in the proportion of benign and malignant cases across Risk Malignancy Index was statistically significant (P value 0.0317) (Table 20 & Figure 13)

Figure 13: Stacked bar chart of comparison of Histopathological report with Risk Malignancy Index in the study population (N=40)

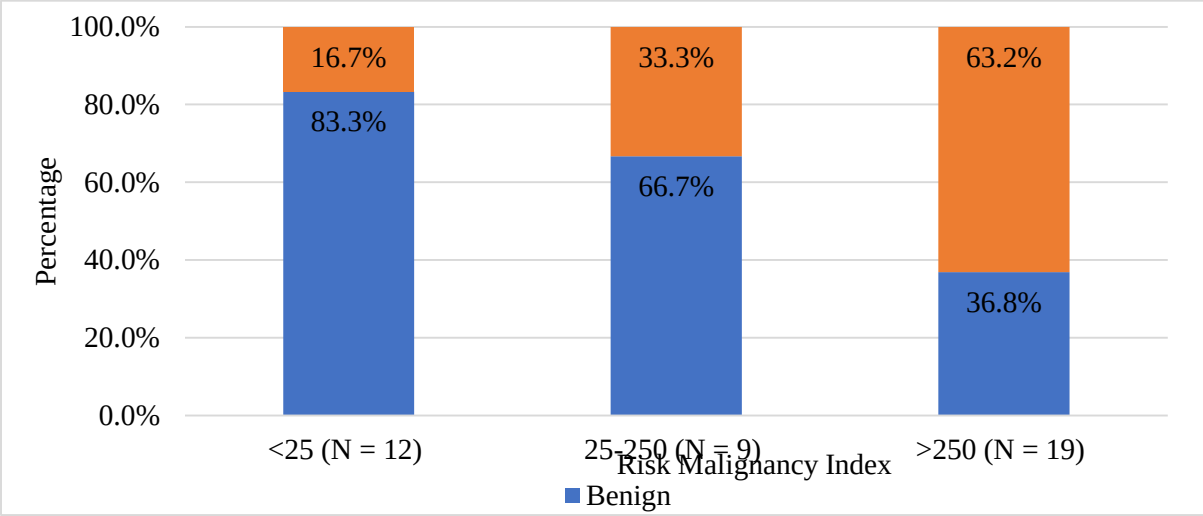


Table 21: Comparison of Histopathological report with USG Score in the study population (N=40)

USG Score	Histopathological report		Chi square value	P value
	Benign	Malignant		
USG Score 0 (N = 13)	8 (61.54%)	5 (38.46%)	4.81	0.0901
USG Score 1 (N = 13)	10 (76.92%)	3 (23.08%)		
USG Score 3 (N = 14)	5 (35.71%)	9 (64.29%)		

Out of 13 participants with USG score 0, 8 (61.54%) were benign and another 5 (38.46%) were malignant. Out of 13 participants with USG score 1, 10 (76.92%) were benign and another 3 (23.08%) were malignant. Out of 14 participants with USG score 3, 5 (35.71%) were benign and another 9 (64.29%) were malignant. The difference in the proportion of benign and malignant cases across USG score was statistically not significant (P value 0.0901) (Table 21 & Figure 14)

Figure 14: Stacked bar chart of comparison of Histopathological report with USG Score in the study population (N=40)

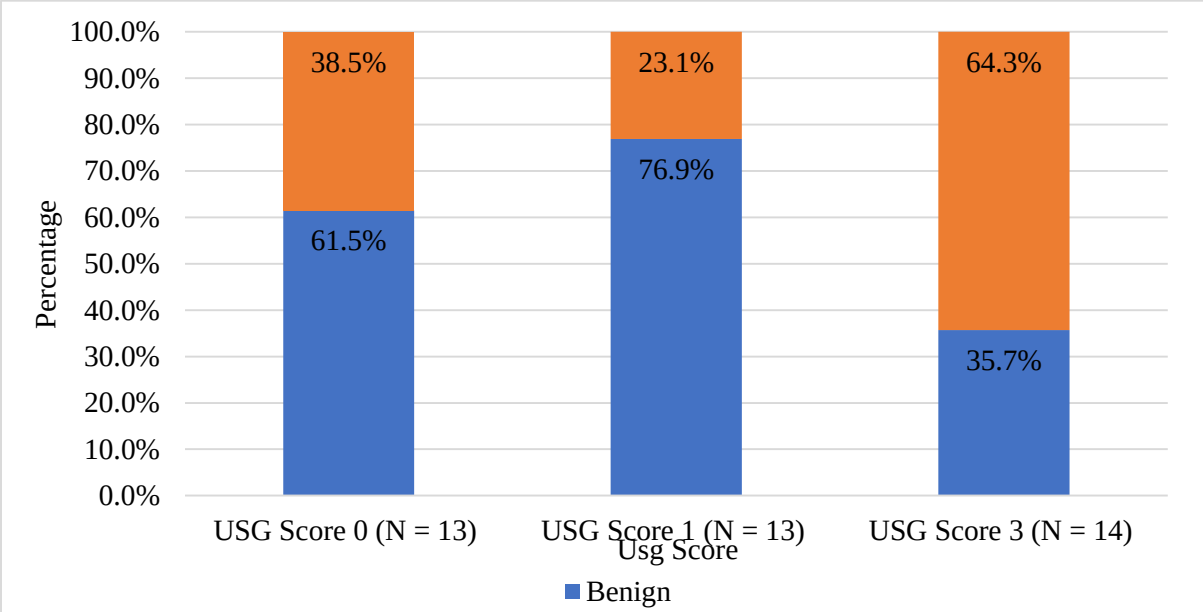


Table 22: Comparison of Histopathological report with GROSS Appearance in the study population (N=40)

GROSS Appearance	Histopathological report		Chi square value	P value
	Benign	Malignant		
CYSTIC (N = 13)	8 (61.54%)	5 (38.46%)	0.16	0.9238
SOLID (N = 13)	7 (53.85%)	6 (46.15%)		
COMPLEX (N = 14)	8 (57.14%)	6 (42.86%)		

Out of 13 participants with cystic gross appearance, 8 (61.54%) were benign and another 5 (38.46%) were malignant. Out of 13 participants with solid gross appearance, 7 (53.85%) were benign and another 6 (46.15%) were malignant. Out of 14 participants with complex gross appearance, 8 (57.14%) were benign and another 6 (42.86%) were malignant. The difference in the proportion of benign and malignant cases across gross appearance was statistically not significant (P value 0.9238) (Table 22 & Figure 15)

Figure 15: Stacked bar chart of comparison of Histopathological report with GROSS Appearance in the study population (N=40)

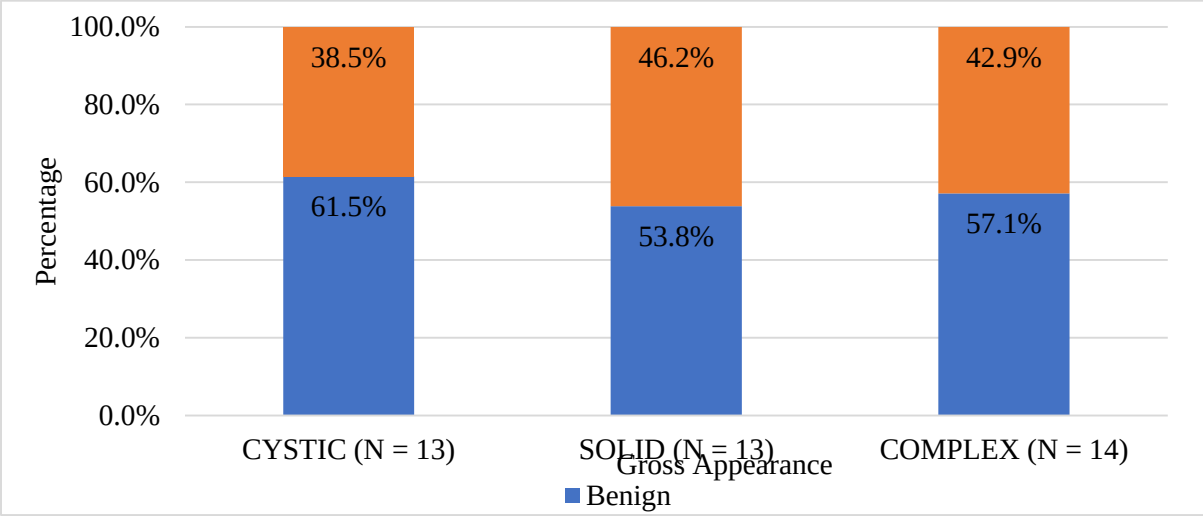


Table 23: Comparison of Histopathological report with Risk Malignancy Index in the study population (N=40)

Risk Malignancy Index (RMI)	Histopathological report		Chi square value	P value
	Malignant (N=17)	Benign (N=23)		
>=25	15 (88.2%)	13 (56.5%)	4.68	0.032
<25	2 (11.8%)	10 (43.5%)		

Out of 17 malignant as per histopathology report, 15(88.2%) had the RMI value as >=25 and in benign histopathology report 13(56.5%) had high value of RMI i.e., >=25. The difference in RMI values between histopathology report was statistically significant (P value <0.05). (Table 23 & Figure 16)

Figure 16: Stacked bar chart of comparison of Histopathological report with Risk Malignancy Index in the study population (N=40)

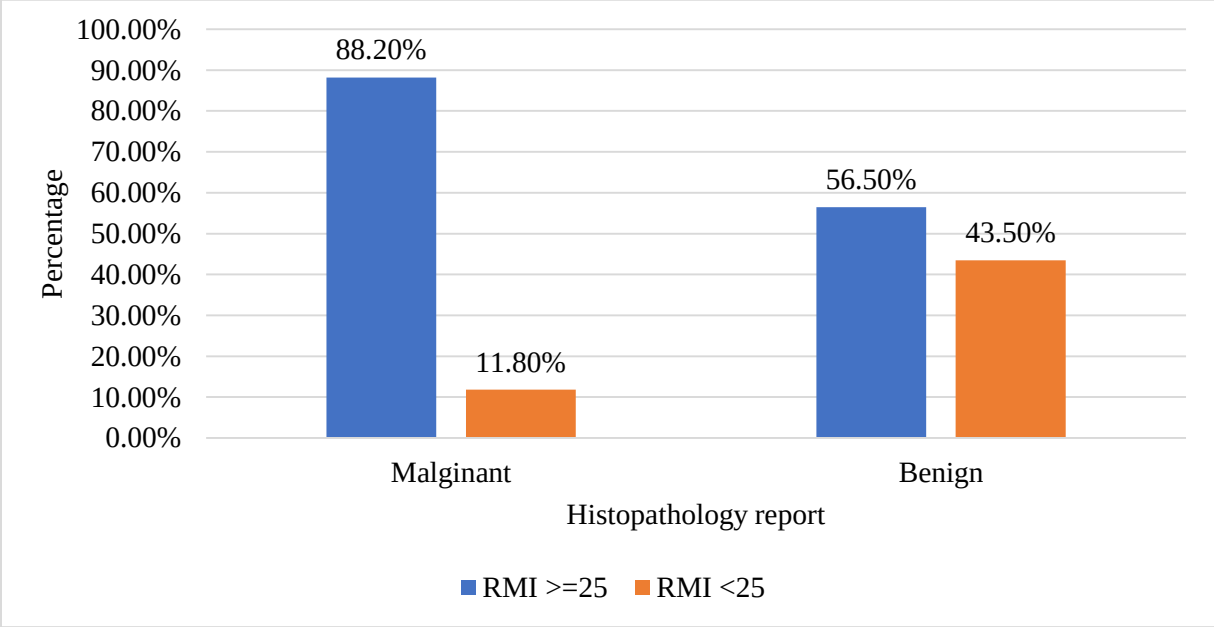


Table 24: Predictive validity of Risk Malignancy Index in predicting malignancy (N=40)

Parameter	Value	95% CI	
		Lower	Upper
SENSITIVITY	88.24%	63.56%	98.54%
SPECIFICITY	43.48%	23.19%	65.51%
FALSE POSITIVE RATE	56.52%	34.49%	76.81%
FALSE NEGATIVE RATE	11.76%	1.46%	36.44%
POSITIVE PREDICTIVE VALUE	53.57%	33.87%	72.49%
NEGATIVE PREDICTIVE VALUE	83.33%	51.59%	97.91%
DIAGNOSTIC ACCURACY	62.50%	45.80%	77.27%

The risk malignancy index had sensitivity of 88.24% (95% CI 63.56% to 98.54%) in predicting malignancy. Specificity was 43.48% (95% CI 23.19% to 65.51%), false positive rate was 56.52% (95% CI 34.49% to 76.81%), false negative rate was 11.76% (95% CI 1.46% to 36.44%), positive predictive value was 53.57% (95% CI 33.87% to 72.49%), negative predictive value was 83.33% (95% CI 51.59% to 97.91%), and the total diagnostic accuracy was 62.50% (95% CI 45.80% to 77.27%). (Table 24)

DISCUSSION

DISCUSSION

Ovarian tumours present differently than other gynaecological cancers, making it difficult for clinicians to diagnose them. “Lower abdomen pain or discomfort, bloating, back pain, or urinary symptoms” are all possible indications of a tumour, although the tumour may be asymptomatic until it has reached a more advanced stage. Understanding whether a patient's ovarian tumour is benign or malignant is a constant challenge for doctors assessing a patient with such a growth. Ultrasound imaging helps doctors pinpoint the tumor's start, growth, consistency, and interior structure so they can treat it effectively. Clinical examination, ultrasonography, and CA-125 readings make up the "triple diagnostic approach" for identifying ovarian tumours. The “histopathological diagnosis” is the benchmark by which all other diagnoses are judged when it comes to predicting patient outcomes. In this prospective observational study conducted on women diagnosed with ovarian mass by clinical examination and confirmed by ultrasonography who are undergoing surgery at R. L. Jalappa Hospital, Kolar, we assessed the correlation between the clinico-radiological profile with histo-pathological picture. Histopathological report is the primary outcome parameter. This information may be used to propose strategies for early diagnosis of ovarian neoplasms and improved methods of treating the condition.

A total of 40 subjects are included in the analysis among which 22.50% are aged <=40 years and 77.50% are aged > 40 years. Laul et al. had 77.3% of the women in the 21-40 years’ age group with a mean age of 31 years in their study. [83] Sixty percent of the women in Kamath et al.’s research were in their 40s and 50s, while just 7% were under the age of 20 and 13% were above the age of 60. Around 20% of the female participants were between the ages of 21 and 40. [84] Dora et al. found that 61.1% of their cases occurred in premenopausal women, whereas 38.89% occurred in postmenopausal women. [72] Patients in the research by Rai et al. had a mean

age of 36.6 ± 14.1, and the vast majority (72.7%) were between the reproductive ages of 20 and 49. [102] In the research by Priya F. et al., the average age of the participants was 42 years old, 62.83 percent were in the reproductive age group, and 10.62 percent were in the postmenopausal age group. [85]

Majority of the patients in our study are multiparous at 65% followed by primiparous 27.5% and nulliparous 7.50%. Laul et al.'s study had 22.6% nulliparous and 77.3% multiparous women. [83] In agreement with the above, majority (75%) were multiparous in Kamath et al.'s study. [84] In Rai et al.'s study, 29.1 were nulliparous and 70.87% multiparous.[102] Similar distribution was seen in In the research by Baru et al., only 22.22 percent of patients with ovarian tumours were nulliparous, while the remaining 77.78 percent were multiparous. [86]

With regards to clinical presentation, 57.50% presented with pain abdomen, 40% with menstrual irregularities, 75% with mass per abdomen, 32.5% with abdominal distension, 27.5% with white discharge and 32.5% with bladder disturbances. Laul et al. reported 44.3% had pain abdomen, 35.1 mass per abdomen, 8.2% with menstrual complaints, 12.4% with infertility, 25.8% had pressure symptoms in their study.[83] ACOG guidelines state that patients and their obstetrician-gynecologists are advised to remain appropriately suspicious in the presence of ovarian cancer warning indicators that might be important, such as women who experience symptoms such as "weight gain or bloating, pelvic or abdominal pain, difficulty eating, or feeling full quickly for more than 12 days per month". Patients meeting these criteria are more likely to develop ovarian cancer than women who do not exhibit these symptoms. [106] Our findings are consistent with those of Kamath et al., who also observed that abdominal pain was the most common symptom (63%), followed by "abdominal distension (40%) and abdominal mass (38%), and then nonspecific symptoms of vomiting and anorexia (25%)."[84]

Patients in the research by Priya F. et al. reported the most often occurring symptoms to be abdominal discomfort and sporadic vaginal bleeding. Abdomen pain in 71.68%, bleeding per vaginum in 10.61%, abdominal distension in 9.73%, irregular menstruation in 5.3% were clinical symptoms in their study.[85] Pain in the abdominal region was reported by 77.05% of patients, swelling by 70.49%, the presence of ascites by 57.38%, the presence of a mass in the abdominal region by 44.90%, and other constitutional symptoms such as "gastrointestinal distress, weakness, menstrual disorders, and urinary symptoms" by 29.5%, 16.4%, 3.28%, and 3.282%, respectively in Baru et al.'s study.[86]

At a cut off of 35, 37.50% had < 35U/ml CA-I25 and 62.50% had > 35U/ml CA-I25. Among the patients analysed by Kamath et al., 62% had CA-125 levels over 100U/l, 16% had levels between 35 and 100 U/l, and 22% had levels below 35 U/l.[84]

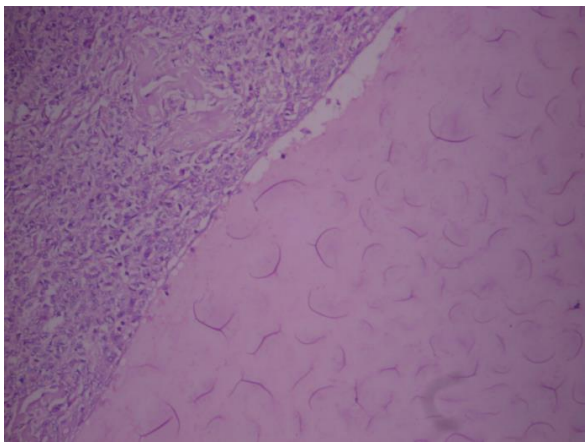
On ultrasonography, 52.50% had cystic mass, 25% had solid, 27.50% had both cystic and solid feature, 22.50% had ascites, 30% had multi cystic feature, 27.50% had unicystic feature, 40.00% had thin septations and 22.50% had thick septations. Fifty percent of the patients had multilocular lesions, presence of solid components in 64.28%, ascites in 38.09% in Dora et al.'s study.[72]

Majority of our patients are postmenopausal at 70% with 30% perimenopausal. Contrary to our study group, Laul et al. had majority (85.6%) in the premenopausal status and only 14.4% postmenopausal.[83] Sixty-two percent of the women in Kamath et al.'s research were postmenopausal, 36% were menstruation, and just 2 were premenarchal. [84] Javdekar et al.'s study had 58.62% premenopausal and 41.38% postmenopausal women.[87]

In 17.5 percent of cases, USG score was 1, 35% of cases were given USG score 2, and 47.5 percent of cases were given USG score 3 based on the five ultrasound features suggestive of malignancy "(multilocularity (more than bilocular), presence of solid areas, bilaterality, presence of ascites, and extra ovarian tumours or evidence of metastases)." Sixty-five percent in the research by Kamath et al. had ultrasonography scores more than 2, which is indicative of a malignant tumour.[84] A total of 45.24 percent of cases in the research by Dora et al. got an ultrasound score of 1, whereas 54.76 percent of patients were given a score of 3.[72]

Upon histopathology of the mass, 57.5% had a benign mass and 42.5% had malignant mass. Histopathological testing revealed that 88.7 percent of the ovarian tumours in the research by Laul et al. were benign, while just 11.3% were malignant. [83] Cancerous tumours made for 55.76 percent of the tumours in Dora et al.'s research, whereas benign tumours accounted for 45.24 percent. [72] Rai et al.'s study had a less rate of malignancy at 17.6% with 82.4% benign masses. [102] Priya F. et al. reported 65.48% were benign and 34.51% were malignant ovarian tumours in their study. [85] Malignancy rate was 17% in Al-Musalhi et al.'s study.[88] In Javdekar et al.'s study, 71 % had benign tumours, 3 % had borderline, and 26 % had malignant disease.[87] Majority of them are malignant tumours (56.48%) followed by benign tumours (40.74%) and borderline tumours (2.78%) in Baru et al.'s study.[86]

Figure: 17



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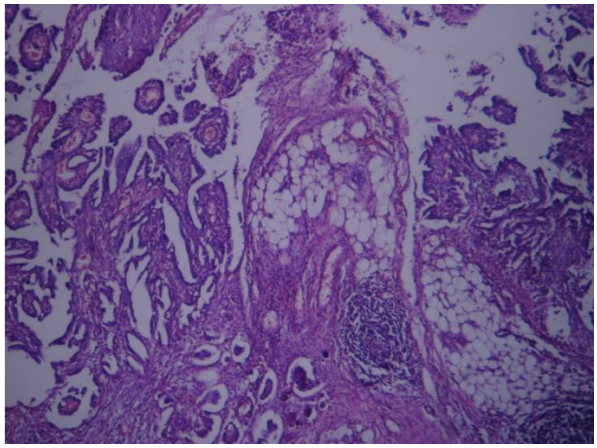

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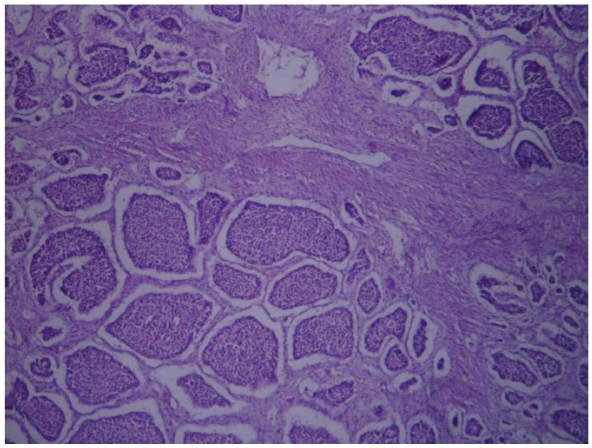


Figure 20

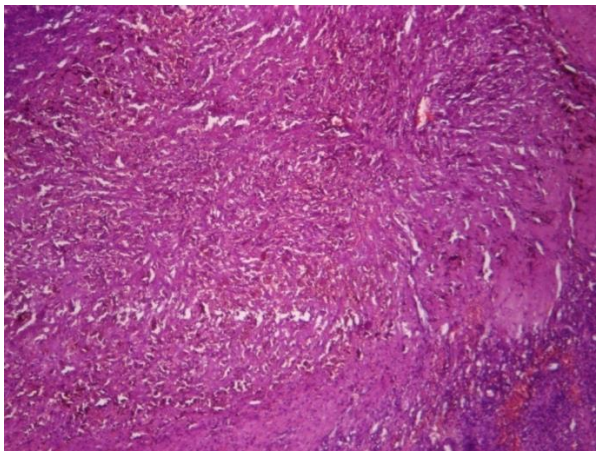
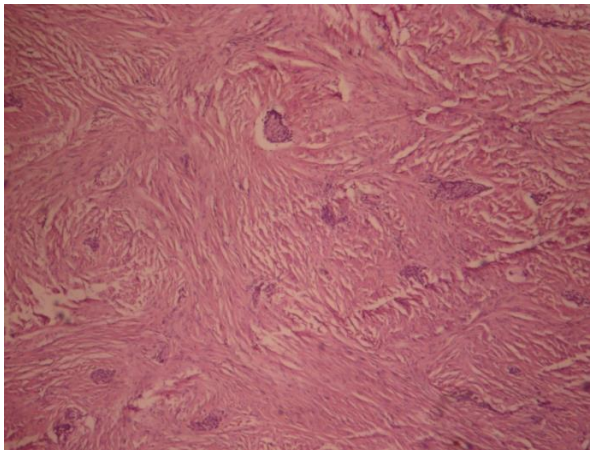


Figure-21



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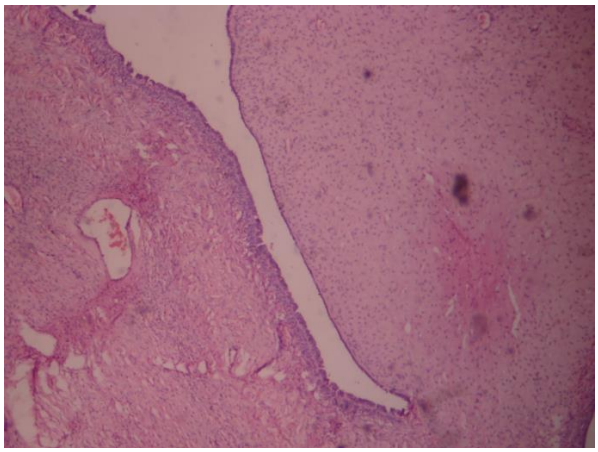
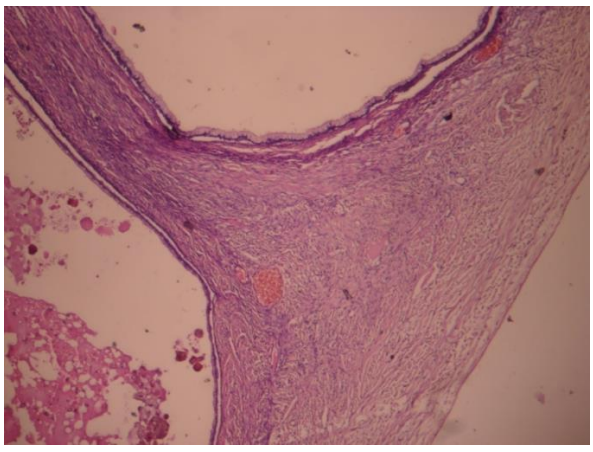


Figure-23



Comparison of Histopathological report with age group

Majority of malignancy is seen in the <= 40 years' age group at 66.67% and in the > 40 years' age group, 35.48% of malignancy is seen. Malignancy is more common in younger age groups, however the difference between the percentage of benign and malignant cases across age

groups is not statistically significant (p value = 0.1338). Contrary to our study, in Laul et al.'s study, 54.5% of the malignant cases were in women with > 60 years of age, 9.3% of the postmenopausal group had benign ovarian masses, and patients with benign ovarian tumours were most often between the ages of 21 and 40 (83%). Women above the age of 60 had a substantially greater incidence of malignant masses than younger women ($p<0.001$). [83] Dora et al. observed that whereas 58% of cancers developed in postmenopausal women and 42% in those who had not yet reached menopause, there is a significant difference in mean age in years between those with malignant adnexal mass (47.30±11.43) and those with benign adnexal mass (37.12± 13.05) with P-value = 0.000). They found that postmenopausal age had higher specificity in predicting malignancy, which is not observed in our study.[72] It was shown by Rai et al. that the majority of benign ovarian tumours (72.6% of all cases) occurred in women aged 20 to 39, whereas the majority of malignant ovarian tumours (63.2% of all cases) occurred in women aged 50 and older. Cancer incidence was observed to be significantly higher in postmenopausal women, and that the "median age" of women with malignant tumours was 60.63 years, whereas the "median age" of women with benign tumours was 40.8 years. [102] Malignant ovarian tumours were more common in postmenopausal women, in contrast, benign ovarian tumours were more prevalent in women of childbearing age, according to a research by Priya F. et al. [85] In the research conducted by Javdekar et al., there was no statistically significant correlation between age and illness status.[87] In the research by Baru et al., ovarian tumours accounted for 57.4% of diagnoses among reproductive-aged women, 41.7% among postmenopausal women, and 0.9% among premenarchal children.[86]

Comparison of Histopathological report with laterality

Among those with unilateral tumour, 70% had benign and 30% had malignant tumour. In bilateral tumour group, 45% had benign and 55% had malignant tumour. Unilateral vs bilateral tumours showed no statistically significant variation in the ratio of benign to malignant

instances (P value 0.1098). USG showed that 72.7% of ovarian cancer tumours were located in only one side of the ovaries, whereas 27.3% were found in both. Almost five percent of benign tumours are found on both sides of the body. The research by Laul et al. found a significant statistical relationship between bilaterality and the presence of malignant ovarian tumours. ($p<0.001$). [83] Similar to our study, among those with bilateral mass, 54.28% were malignant and 45.72% were benign in Dora et al.'s study. Higher rates of bilaterality in adnexal masses are seen in malignant tumours compared to benign tumours, albeit this difference is not statistically significant ($P = 0.947$). [72]

Comparison of Histopathological report with Parity

Our analysis found no statistically significant difference ($p=0.8127$) in the percentage of benign and malignant cases based on parity. Like our research, Rai et al. found no association between parity and malignancy. [102] In the research by Baru et al., nulliparous cases made up 27.27 percent of benign tumours and 10.03 percent of malignant tumours.[86]

Comparison of Histopathological report with size of the tumour (cm)

Among those with tumour size 11-19 cm, 42.11% had malignancy, in 20-29 cm size, 45.45% are malignant, > 30 cm size 44.44% had malignancy. In our study, size has no significance on the disease status. Seventy-seven percent of patients with benign masses had a size < 10 cm, whereas 23 percent of patients had a mass larger than 10 cm, as determined by Laul et al. Ten of the malignant tumours were larger than 10 centimetres, a statistically significant finding with $p<0.001$. [83] According to Baru et al., 47.72% of benign tumours are found between 10 and 19 cm, whereas 59% of malignant tumours are found between 10 and 19 cm. Most malignant tumours are far larger than their benign counterparts.[86]

Comparison of Histopathological report with CAI25 (U/ml)

Though elevated serum "CA-125" levels may be caused by other gynaecological disorders, they are well recognised as a significant biomarker for ovarian cancer risk assessment. Those with < 35 U/ml CA-I25, malignancy rate is 33.33% and in the > 35 U/ml CA-I25 group, 48% are malignant. Though there was no discernible pattern between the numbers of benign and malignant instances in the CA-I25 (P value = 0.3637), a little higher proportion of malignant patients exhibited raised CA-125 levels than benign ones. Laul et al. found a statistically significant connection between an increased CA-125 level and malignancy; 96.5% of benign patients had a level of CA-125 < 35 IU/ml, 77.8% of malignant cases had an elevated CA-125 level. [83] In the research by Kamath et al., a staggering 93.42 percent of malignant patients showed increased CA-125 values, whereas only 6.58 percent had levels below 35, making them false negatives. Twenty-five percent of benign tumours had blood CA-125 values above the normal range (35), whereas seventy-five percent were within these parameters. [84] In a research by Dora et al., CA-125 levels below 35 U/ml showed an 87% sensitivity, a 19% specificity, a 56% positive predictive value, and a 55% negative predictive value. They also said that a cutoff value of 143 resulted in the optimum performance of CA-125. [72] The research by Al-Musalhi et al. found that CA-125 was elevated (>35 U/ml) in 69% of women with malignant ovarian tumours and in 32% of those with benign ovarian tumours. CA-125's overall diagnostic accuracy was 69% within this cutoff range, with a sensitivity of 69%, specificity of 68%, PPV of 31%, and NPV of 92%.[88] Mean and median CA-125 levels in people with benign tumour were 33 and 13, whereas those in patients with malignant disease were 395 and 329, respectively. Javdekar et al. found that this association had a p value of 0.0001, making it very significant. A serum Ca-125 level was shown to have a sensitivity of 76.4%, specificity of 85.3%, positive predictive value of 68.4%, and negative predictive value of 89.7%.[87]

Comparison of Histopathological report with menstrual history

We found 53.3% of the perimenopausal group and 35.71% of the postmenopausal group had malignant tumours. The proportion of patients who were benign vs those who were malignant did not vary significantly depending on their menstrual histories. (P value 0.1848)

Comparison of Histopathological report with Risk Malignancy Index (RMI)

Among those with < 25 RMI score, 16.67% had malignancy, 25-250 RMI, 33.33% had malignancy, in >250 RMI, 63.16% had malignancy. There is a statistically significant variation in the ratio of benign to malignant patients across RMI (P value 0.0317). All of the individuals in the research by Laul et al. who had benign ovarian tumours had RMI below 200. [83] Both their research and ours found a significant association between RMI and malignancy. Kamath et al. found that malignancy was present in 72% of patients with an RMI > 200, whereas benign illness was present in 28% of patients with an RMI < 200. They speculated that RMI may not be able to pick up on instances on the cusp of clinical significance. Patients with RMI >200 were 91% malignant whereas those with RMI 200 were 43% malignant and 57% benign. [84] 57% of women with malignant ovarian tumours and 18% of women with benign ovarian cancers had an elevated RMI (>200) in Al-Musalhi et al.' study.[88]

Comparison of Histopathological report with USG Score

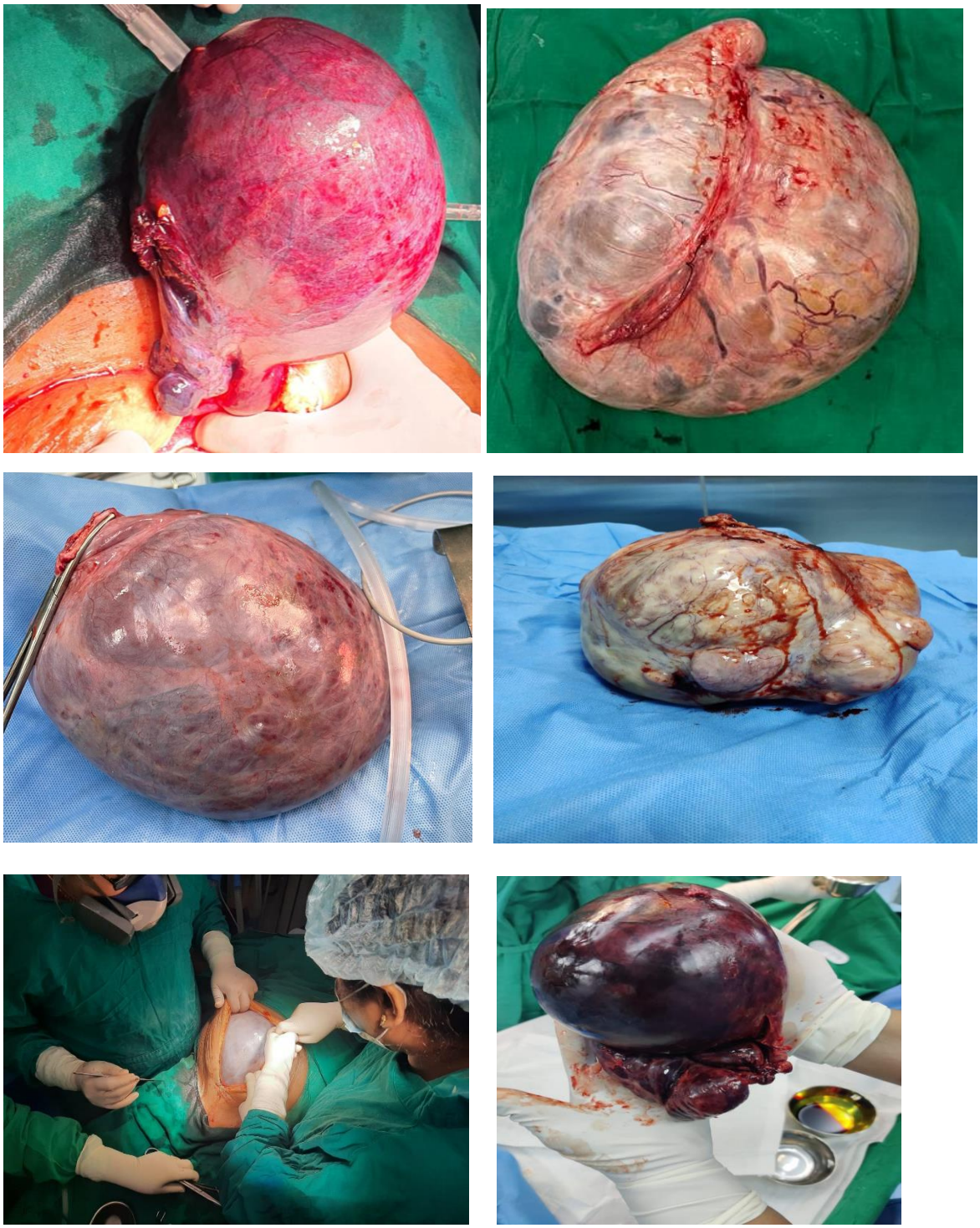
It is generally agreed that ultrasonography is the most reliable imaging technique for diagnosing ovarian pathology. Among those with USG score 0, 38.46% had malignancy, USG score 1, 23.08% had malignancy, USG score 3, 64.29% had malignancy. The percentage of benign and malignant patients did not vary significantly by USG score (P value = 0.0901), however a higher percentage of malignancy was seen among those with a USG score of 3. All of the malignant tumours in the research by Laul et al. were solid, but only 16.3 percent of the

benign ovarian masses were solid. The presence of thick, irregular septa on USG was shown to have a high statistical correlate with malignancy ($p<0.001$). They also said that USG showed no ascites in any of the benign masses, and that the presence of ascites was statistically significantly correlated with malignancy ($p<0.001$). USG characteristics such as the presence of "solid regions, thick and irregular septa, and ascites" were shown to have statistical significance in their research for the diagnosis of malignancy. [83] According to the research by Dora et al., an ultrasound score of 3 has a "68.1% sensitivity, 61.4% specificity, 68.12% positive predictive value, and 61.4% negative predictive value" for identifying malignancy. [72] In a research by Priya F. et al., USG was shown to have a "sensitivity of 88.00%, a specificity of 80.68 %, a PPV of 56.41%, and a NPV of 94.95%" for predicting ovarian cancer. [85] Javdekar et al. found a very significant relationship between USG score and illness state in their study ($p = 0.0004$). The "PPV of the USG score was 56.5 percent, the specificity was 75.6 percent, the sensitivity was 76.4 percent, and the NPV was 88.5 percent". [87]

Comparison of Histopathological report with GROSS Appearance

In the cystic gross appearance mass, 38.46% are malignant, in the solid gross appearance, 46.15% are malignant, in the complex gross appearance, 42.86% are malignant. Based on gross appearance alone, there was no statistically significant difference between the proportion of benign and malignant patients (P value 0.9238). In the research by Laul et al., all malignant tumours were solid, and 16.3 percent of the benign ovarian masses were solid as well. [83] According to research by Priya F. et al., ovarian tumours that were entirely solid were always malignant, whereas those that also had both "solid and cystic" components were often malignant. [85] The majority of malignant tumours (37.7%) in the research by Baru et al. were solid, whereas the majority of benign tumours were cystic (63.63 percent).[86]

Figure :24



Comparison of Histopathological report with Risk Malignancy Index

At a cut off of 25, majority (88.2%) of those with malignancy had RMI >=25 and in benign histopathology report 56.5% had >= 25 RMI. There was a statistically significant ($P < 0.05$) disparity between the RMI values reported by the two different types of histopathology. When

it came to identifying malignancies, the RMI was 88.24% sensitive, 43.48% specific, 53.57% positive predictive, 83.33% negative, and 62.50% accurate overall. According to research conducted by Kamath et al., the RMI has a sensitivity of 84% when identifying malignant lesions and a specificity of 67% when doing so. The NPV was 57%, while the PPV was 89%. [84] Results from the research by Dora et al. found that an RMI of >=236 was associated with a high degree of "sensitivity (72.5 percent), specificity (98.2 percent), positive predictive value (74.7 percent), and accuracy (84.13 percent)." Lower cut off values raised sensitivity but decreased specificity, whereas higher cut off values improved specificity but decreased sensitivity, perhaps leading to misdiagnosis of benign cases as malignant. [72] Consequently, the determination of the cut off value will be a compromise between the need for "sensitivity and specificity" on the one hand, and the availability of local resources and professionals on the other. Experts in the area should be given leeway to make these kinds of decisions. Consistent with the findings of these prior research, we found that RMI's predictive ability was superior to that of single parameters. The researchers Rai et al. used two distinct RMI score cut offs, one that grouped individuals with RMI < 200 versus RMI >200, and another with RMI >25 versus RMI < 25. They stated that using ROC curve analysis, they discovered that an RMI cutoff of 58.7 yielded the best results, with a "sensitivity of 84.2% and a specificity of 70.4%."[102] RMI was shown to have a "sensitivity of 70.5%, specificity of 87.8%, PPV of 70.5%, and NPV of 87.8%" at a cut off of 200 in a research by Javdekar et al. Based on ROC analysis, the likelihood of having a malignant condition increases from 1.46 times at a cut off value of 25 to 24.11 times at a cut off level of 1,000.[87]

Table 25: Predictive validity of Risk Malignancy Index in predicting malignancy across studies

Study	Cut-off value	"Sensitivity"	"Specificity"	PPV	NPV	Total diagnostic accuracy
Current study	25	88.24%	43.48%	53.57%	83.33%	62.50%
Kamath et al. [84]	200	84%	67%	89%	57%	
Dora et al.[72]	236	72.5%	98.2%	98.1%	74.7%	84.13%
Rai et al.[102]	25	90.91%	42.9%	38.5%	92.31%	76.9%
Al-Musalhi et al. [88]	200	57%	81%	38%	90%	69%
Javdekar et al. [87]	200	70.59%	87.8%	70.5%	87.8%	

In conclusion, the current research demonstrated that the multiparametric RMI is a more accurate estimate in diagnosing "ovarian masses with a high risk of malignancy" and, as a result, guiding patients to gynaecological oncology centres for appropriate and "effective surgical interventions than individual parameters of CA-125 or USG score". This is consistent with the findings of Laul et al., who found that benign ovarian tumours were more prevalent before menopause, whereas malignant tumours occurred more often after menopause. Tumors of the ovary may manifest themselves in a number of ways. The majority of patients presented with abdominal discomfort, followed by a palpable mass in the abdomen and pressure sensations. The larger lumps almost often included malignancy. [83] Dora et al. concurred that RMI is an improved indicator for determining whether or not ovarian tumours are malignant. [72] According to Rai et al., a low RMI score was strongly related with benign lesions, while a high RMI value was significantly associated with malignant lesions. [102] According to research by Rai et al., RMI can effectively distinguish between "benign and malignant" masses, and this is true both in general and in the case of epithelial ovarian tumours. [102] The research by Priya F. et al. suggests that USG is a sensitive tool for identifying ovarian cancer, but that a combined

clinical examination with “USG and Doppler” is highly suggested for distinguishing between “benign and malignant” ovarian tumours. [85] When it comes to identifying ovarian tumours, both CA-125 and RMI have been shown to be very reliable, as reported by Al Musalhi et al. The sensitivity of CA-125 is greater, while the specificity of RMI is greater. CA-125 may be useful for the diagnosis of malignant ovarian cancer, whereas RMI may be useful for ruling out this diagnosis. [88] The predictability value of RMI was determined at varying cutoffs by Javdekar et al., who also proposed that the threshold RMI score for referral could depend on the facilities available, with a higher RMI having lower sensitivity but better specificity being used in cases where access to specialist care was limited.[87]

Limitations and recommendations:

The study is limited by its small sample size. Given that it is hospital-based, there is a higher prevalence of malignancies and referral bias than in the general population.

CONCLUSION

Conclusion:

The first-line treatment for women with ovarian cancer could be optimised with the help of a standardised method for identifying likely malignant masses prior to surgery.[87] Unfortunately, there is currently no method that can reliably predict ovarian malignancy and a scoring system that could identify cancer risk would help with appropriate counselling and prompt referral to a specialised facility. RMI is one such scoring index with its ease of application and simplicity of the method serves as a very compelling justification for the use of initial assessment of patients with pelvic masses. In this prospective observational study, a total of 40 subjects are included in the analysis among which 22.50% are aged <=40 years and 77.50% are aged > 40 years. Upon histopathology of the mass, 57.5% had a benign mass and 42.5% had malignant mass. Not statistically significant, but age <=40 years, bilaterality, CA-125 > 35 U/ml, USG score 3 had a slight more proportion of malignancy in our study. The association between gross appearance, size, parity and histopathology is not significant. While difference in the proportion of benign and malignant cases across USG score was statistically not significant (P value 0.0901), more percentage of malignancy is seen among those with USG score 3.

At a cut off of 25, majority (88.2%) of those with malignancy had RMI >=25 and in benign histopathology report 56.5% had >= 25 RMI. The difference in RMI values between histopathology report was statistically significant (P value <0.05). The RMI had a" sensitivity of 88.24% in predicting malignancy with specificity 43.48%, positive predictive value 53.57%, negative predictive value 83.33% with a total diagnostic accuracy of 62.50%." Results from RMI and histology correlate positively.

SUMMARY

Summary

1. A total of 40 subjects are included in the analysis among which 22.50% are aged <=40 years and 77.50% are aged > 40 years.
2. Majority of the patients in our study are multiparous at 65% followed by primiparous 27.5% and nulliparous 7.50%.
3. In our study, 50% had unilateral tumours and 50% had bilateral tumours.
4. Upon histopathology of the mass, 57.5% had a benign mass and 42.5% had malignant mass.
5. Although there was not a statistically significant difference (p value of 0.1338) in the percentage of benign and malignant cases across age groups, there was a higher prevalence of malignancy in the younger age group.
6. There was no statistically significant difference in the percentage of cases that were benign and those that were malignant across all parities (P value 0.8127).
7. There was not a statistically significant difference in the percentage of cases that were benign and those that were malignant between unilateral and bilateral tumours. (P value 0.1098).
8. There was not a statistically significant difference in the percentage of cases that were benign and those that were malignant between CA-I25. (P value 0.3637).
9. There was no statistically significant difference between the percentage of cases that were benign and those that were malignant based on menstrual history. (P value 0.1848).
10. The difference in the proportion of benign and malignant cases across RMI is statistically significant (P value 0.0317).

11. The difference in the proportion of benign and malignant cases across USG score was statistically not significant (P value 0.0901) but more percentage of malignancy is seen among those with USG score 3.
12. The difference in the proportion of benign and malignant cases across gross appearance was statistically not significant (P value 0.9238).
13. At a cut off of 25, majority (88.2%) of those with malignancy had RMI >=25 and in benign histopathology report 56.5% had >= 25 RMI. The difference in RMI values between histopathology report was statistically significant (P value <0.05).
14. The RMI had a sensitivity of 88.24% in predicting malignancy with specificity 43.48%, positive predictive value 53.57%, negative predictive value 83.33% with a total diagnostic accuracy of 62.50%.
15. The results of this research demonstrate that RMI is a valid, efficient, and feasible tool for evaluating patients with pelvic masses at the outset of care and for referring suitable candidates for centralised surgical treatment. Results from RMI and histopathology correlate positively.
16. In low resource settings, when sophisticated radiological and biochemical testing may not always be accessible, RMI might be utilised as an investigation for patient triage and referral to a higher centre.
17. In the absence of a definitive biomarker, we found that the multiparametric RMI was a more accurate estimate in detecting ovarian masses with a high risk of malignancy and directing patients to gynaecological oncology clinics for appropriate surgical procedures. Although sophisticated radiological examinations are at the surgeon's disposal, the importance of a straightforward method like RMI for identifying benign from malignant tumours before surgery cannot be overstated.

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ANNEXURES

STUDY PROFORMA

PROFORMA

Name : Marital status :

Age : Ip No :

Religion : Date of admission :

Address :

I) present complaints:

Mass per abdomen-

Abdominal pain-

Any menstrual irregularities-

Urinary symptoms

GIT symptoms

Edema of lower limbs

Any discharge per vagina

II) Menstrual history

a. Age of menarche – years

b. Past menstrual cycle

Regular/irregular, Amount of flow- scanty/ moderate/ excessive

Dysmenorrhoea – yes/no, Associated clots - yes/no

III) Obstetric history

Married life Consnguinous/ non consnguinous Parity

Last delivery Tubectomised/not

IV) Past history

TB/DM/HTN/Bronchial asthma/any surgeries/ thyroid/ cardiac diseases.

H/O use of oral contraceptives in the past

V) Family history

TB/DM/HTN/Bronchial asthma/any surgeries, Any similar complaints in the family

VI) Personal history

Diet – Veg/mixed, Appetite – Normal/ decreased, Sleep – Normal/ disturbed,

Bowel – regular / irregular, Bladder – Normal/ increased/ decreased

VII) General physical examination

Built / Nourishment

Icterus/clubbing/cyanosis/pallor/pedal/ edema/Lympadenopathy

Temperature – Febrile / afebrile

Pulse - BP - RR- SPO2-

VII) Systemic examination

Cardiovascular system

Respiratory system

Abdominal examination

a. Inspection

Shape

Movement of quadrants with respiration

Mass / swelling

Size

Shape

Extent

Any engorged vein

Umblicus

Hernial sites

b. Palpation

Local raise of temperature

Tenderness

Mass – situation

Size

Extent

Surface

Consistency

Borders

Movements with respiration

Any organomegaly

c. Percussion – Ascities – present / absent

d. Auscultation – Any bruit- present/absent

IX) Per speculum examination

Vagina –

Cervix –

Erosion –

Discharge –

X) Per-vaginal examination

Cervix- consistency/ position/ mobility/tenderness

Uterus – size/position/ mobility/tenderness

Mass felt bimanually separate from uterus/ not

Abdominal mass movement transmitted to cervix/not

Forniceal examination – full/ free, tender/non tender

XI) Per rectal examination

Nodulaity

Rectal wall

Pouch of douglas

XII) Investigations

a. Blood – CBC, Blood group, LFT,RFT, HIV, HBsAg

b. Urine – albumin, sugar, microscopy

c. Chest xay

d. USG abdomen and pelvis

e. RMI

f. CT scan of abdomen and pelvis

g. Vaginal smear from posterior fornix

h. Radiological investigation

i. Tumour markers

XIII) Treatment

Surgery –

Peroperative findings

Anesthesia – GA/spinal

XV) Histopathological examination

Gross

Tumour – Unilateral/ Bilateral

Weight of the mass

Surface – nodular/ smooth

Shape – Oval/round/irregular

Capsule – Thickened/ rupture/ hemorrhage

Cut section- Cystic/ solid

Any papillary excrescens

Microscopic examination

Diagnosis:

PATIENT INFORMATION SHEET

Study title: **A CLINICO- RADIOLOGICAL AND HISTOPATHOLOGICAL STUDY OF OVARIAN MASSES AT A TERTIARY CARE CENTRE**

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Details-

Patients who are of clinically indicated for induction admitted to OBG department of R L Jalappa hospital attached to Sri Devaraj Urs medical college are recruited in the study after obtaining patient information consent.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or from a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication. The relevant investigations which are required others than regular investigations will be funded by me.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact

Dr.SHRAVYA MONICA K

Post graduate, Department of obstetrics and Gynaecology

R L Jalappa hospital, Kolar .

INFORMED CONSENT FORM

I Mr./Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is **"A CLINICO-HISTOPATHOLOGICAL STUDY OF OVARIAN MASSES AT A TERTIARY CARE CENTRE"**

I have been explained that my clinical findings, investigations, postoperative findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient: Signature of the witness:

Name: Name:

Relation to patient:

Date:

Place:

DATE:

Investigator signature

ಮಾಹಿತಿ ಕಾನ್ಸೆಂಟ್ ಫಾರ್ಮ್

ನಾನು ಶ್ರೀ / ಶ್ರೀ. _____ ಅನ್ನು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇದನ್ನು ನಾನು ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಿಕೊಳ್ಳುತ್ತೇನೆ, ಅದು ''ತೃತೀಯ ಆರೈಕೆ ಕೇಂದ್ರದಲ್ಲಿ ಓವರಿಯನ್ ಮಾಸ್‌ಗಳ ಕ್ಲಿನಿಕೊ-ಹಿಸ್ಟೊಪಾಥೊಲಾಜಿಕಲ್ ಅಧ್ಯಯನ''

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದು ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಗೆ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ, ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಕಾರಣದಿಂದಾಗಿ ಸಂಭವನೀಯ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಪ್ರತಿಕೂಲತೆಗಳ ಅಗತ್ಯವಿರುವ ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಸಂಪೂರ್ಣ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ:

ಹೆಸರು:

ಸಾಕ್ಷಿಯ ಸಹಿ:

ಹೆಸರು:

ರೋಗಿಗೆ ಸಂಬಂಧ:

MASTER CHART

Sr. No	UHID	Age	Parity	MODE OF PRESENTATION							GROSS				UL/BL	Size	CAI25	USG FEATURES+S1:AD43								Menstrual History	RMI	USG Score	HISTO REPORT
				PA	MI	MASS	DISTEN	WD	BLADDER	BOWEL	Solid gross(1)	Cystic gross(2)	Both gross(3)	Gross appreance				CYSTIC	SOLID	BOTH	ASCITIS	ULTILOCUL CYST	NILOCUL CYST	THIN SEPTATION	THICK SEPTATIONS				
1	65438	45	PRIMI	Yes	No	Yes	No	No	No	No	No	Yes	No	CYSTIC	Bilateral	30	>35	Yes	No	No	No	No	Yes	Yes	No	Pre	>250	USG Score 3	Malignant
2	45964	50	PRIMI	No	No	Yes	No	No	No	No	No	Yes	No	CYSTIC	Bilateral	17	>35	No	No	Yes	No	Yes	No	No	Yes	Post	<25	USG Score 0	Benign
3	42138	34	PRIMI	Yes	No	Yes	No	No	No	No	No	No	Yes	COMPLEX	Bilateral	32	>35	Yes	No	No	Yes	No	No	No	No	Pre	<25	USG Score 0	Benign
4	901150	43	MULTI	Yes	No	Yes	Yes	No	No	No	No	No	Yes	COMPLEX	Bilateral	19	>35	Yes	No	No	No	No	No	Yes	No	Post	>250	USG Score 3	Benign
5	849766	46	MULTI	Yes	No	No	No	No	Yes	No	Yes	No	No	SOLID	Unilateral	27	>35	Yes	No	No	No	Yes	No	No	No	Post	>250	USG Score 3	Malignant
6	888900	46	PRIMI	Yes	No	Yes	No	No	Yes	No	No	No	Yes	COMPLEX	Unilateral	13	>35	Yes	No	No	No	No	No	No	Yes	Post	25-250	USG Score 1	Benign
7	875829	52	MULTI	Yes	No	Yes	No	No	Yes	No	No	No	Yes	COMPLEX	Unilateral	38	>35	Yes	No	No	No	No	Yes	Yes	No	Pre	>250	USG Score 1	Malignant
8	863149	45	MULTI	No	No	Yes	No	No	No	No	No	No	Yes	COMPLEX	Unilateral	17	<35	No	Yes	No	No	No	No	No	No	Post	<25	USG Score 0	Benign
9	70569	56	MULTI	No	No	No	Yes	No	No	No	No	Yes	No	CYSTIC	Unilateral	22	<35	No	No	Yes	No	Yes	No	Yes	No	Post	>250	USG Score 1	Benign
10	62323	42	MULTI	No	No	Yes	No	No	No	No	No	Yes	No	CYSTIC	Bilateral	30	<35	Yes	No	No	No	Yes	No	No	Yes	Pre	<25	USG Score 0	Benign
11	928765	39	PRIMI	No	No	Yes	No	No	No	No	No	Yes	No	CYSTIC	Bilateral	16	<35	No	No	Yes	No	Yes	No	No	No	Pre	>250	USG Score 1	Benign
12	898014	42	MULTI	Yes	No	Yes	No	No	No	No	Yes	No	No	SOLID	Unilateral	19	>35	Yes	Yes	No	No	No	Yes	Yes	No	Post	25-250	USG Score 3	Malignant
13	865263	30	MULTI	Yes	Yes	Yes	No	No	No	No	No	No	Yes	COMPLEX	Bilateral	12	>35	Yes	Yes	No	No	No	No	No	No	Pre	<25	USG Score 0	Malignant
14	860053	44	MULTI	Yes	Yes	Yes	No	No	No	No	Yes	No	No	SOLID	Unilateral	24	>35	No	No	Yes	Yes	No	Yes	No	No	Post	<25	USG Score 0	Benign
15	64537	56	PRIMI	Yes	Yes	Yes	No	Yes	No	No	No	Yes	No	CYSTIC	Bilateral	11	<35	Yes	No	No	No	Yes	No	Yes	No	Post	>250	USG Score 0	Malignant
16	945078	60	MULTI	Yes	Yes	No	No	Yes	No	No	Yes	No	No	SOLID	Bilateral	24	<35	No	No	Yes	No	No	No	No	Yes	Pre	25-250	USG Score 1	Benign
17	883089	72	MULTI	No	No	Yes	Yes	Yes	No	No	Yes	No	No	SOLID	Unilateral	16	>35	Yes	No	No	No	No	Yes	No	No	Post	25-250	USG Score 1	Benign
18	892311	45	MULTI	No	Yes	Yes	Yes	Yes	No	No	No	Yes	No	CYSTIC	Bilateral	18	>35	No	Yes	No	No	No	No	Yes	No	Post	<25	USG Score 0	Malignant
19	892009	33	MULTI	No	No	No	Yes	Yes	No	No	No	Yes	No	CYSTIC	Unilateral	28	>35	Yes	No	No	No	No	No	No	No	Post	>250	USG Score 3	Malignant
20	931922	48	PRIMI	No	Yes	No	Yes	No	Yes	No	No	Yes	No	CYSTIC	Unilateral	10	>35	No	No	Yes	Yes	No	Yes	Yes	No	Post	>250	USG Score 1	Malignant
21	945362	63	NULLI	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	SOLID	Unilateral	18	>35	Yes	No	No	Yes	Yes	No	No	Yes	Pre	>250	USG Score 3	Malignant
22	45225	42	NULLI	No	Yes	Yes	No	No	Yes	No	Yes	No	No	SOLID	Unilateral	26	>35	No	Yes	No	No	No	No	Yes	No	Post	<25	USG Score 0	Benign
23	46406	44	MULTI	Yes	Yes	Yes	No	Yes	Yes	No	Yes	No	No	SOLID	Bilateral	19	<35	No	No	Yes	No	No	Yes	No	No	Post	<25	USG Score 0	Benign
24	60625	54	MULTI	Yes	No	Yes	No	No	Yes	No	No	No	Yes	COMPLEX	Unilateral	36	<35	Yes	No	No	No	Yes	No	Yes	No	Post	>250	USG Score 1	Benign
25	867090	33	MULTI	Yes	No	Yes	No	Yes	Yes	No	No	No	Yes	COMPLEX	Unilateral	18	<35	No	Yes	No	No	No	No	No	Yes	Pre	25-250	USG Score 1	Benign
26	894766	60	PRIMI	Yes	No	Yes	No	No	Yes	No	Yes	No	No	SOLID	Unilateral	22	<35	Yes	No	No	Yes	No	No	Yes	No	Post	>250	USG Score 3	Malignant
27	733490	53	MULTI	Yes	Yes	Yes	No	No	Yes	No	Yes	No	No	SOLID	Bilateral	19	>35	No	Yes	No	No	Yes	No	No	No	Post	<25	USG Score 0	Benign
28	865442	48	MULTI	Yes	Yes	No	Yes	No	Yes	No	No	No	Yes	COMPLEX	Bilateral	50	>35	Yes	No	No	Yes	No	Yes	Yes	No	Post	>250	USG Score 3	Benign
29	860255	39	MULTI	Yes	Yes	Yes	Yes	Yes	Yes	No	No	No	Yes	COMPLEX	Bilateral	43	<35	No	No	Yes	No	No	No	No	Yes	Pre	>250	USG Score 3	Malignant
30	864851	19	NULLI	Yes	Yes	Yes	Yes	No	No	No	No	Yes	No	CYSTIC	Unilateral	26	>35	Yes	No	No	No	Yes	No	Yes	No	Post	25-250	USG Score 1	Benign

31	81473	55	PRIMI	Yes	No	No	Yes	No	No	No	No	No	Yes	COMPLEX	Bilateral	19	<35	No	No	Yes	No	No	No	No	No	Post	>250	USG Score 3	Malignant
32	94591	56	MULTI	No	No	Yes	Yes	No	No	No	No	Yes	No	CYSTIC	Bilateral	18	>35	Yes	No	No	No	No	Yes	Yes	No	Post	<25	USG Score 0	Benign
33	74664	44	MULTI	No	No	Yes	Yes	No	No	No	No	No	Yes	COMPLEX	Unilateral	46	<35	No	Yes	No	No	No	No	No	No	Pre	>250	USG Score 1	Benign
34	181883	35	MULTI	No	No	Yes	No	Yes	No	No	Yes	No	No	SOLID	Bilateral	19	>35	Yes	No	No	No	Yes	No	No	Yes	Post	25-250	USG Score 1	Malignant
35	159473	48	MULTI	Yes	No	No	No	No	No	No	Yes	No	No	SOLID	Unilateral	28	>35	No	Yes	No	Yes	No	No	No	No	Post	<25	USG Score 0	Benign
36	141221	65	PRIMI	Yes	Yes	Yes	No	Yes	No	No	Yes	No	No	SOLID	Unilateral	18	<35	No	No	Yes	Yes	No	Yes	No	No	Post	>250	USG Score 3	Malignant
37	166348	77	PRIMI	No	Yes	Yes	No	No	No	No	No	No	Yes	COMPLEX	Bilateral	19	>35	Yes	No	No	No	No	Yes	Yes	No	Post	25-250	USG Score 3	Malignant
38	171001	73	MULTI	No	Yes	Yes	No	No	No	No	No	No	Yes	COMPLEX	Bilateral	30	>35	Yes	No	No	No	No	No	No	No	Pre	>250	USG Score 3	Benign
39	86651	46	MULTI	No	No	No	No	No	No	No	No	Yes	No	CYSTIC	Unilateral	25	<35	No	Yes	No	No	Yes	No	No	Yes	Post	25-250	USG Score 1	Benign
40	30634	40	MULTI	Yes	No	No	No	Yes	No	No	No	Yes	No	CYSTIC	Bilateral	22	>35	No	No	Yes	Yes	No	No	Yes	No	Post	>250	USG Score 3	Malignant