"HISTOPATHOLOGICAL ANALYSIS OF HYSTERECTOMY SPECIMENS: AN OBSERVATIONAL STUDY."

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LIST OF ABBREVIATIONS USED

WHO	-	World Health Organization
ESN	-	Endometrial Stromal Nodule
LG-ESS	-	Low Grade Endometrial Stromal Sarcoma
HG-ESS	-	High Grade Endometrial Stromal Sarcoma
UUS	-	Undifferentiated Uterine Sarcoma
DUB	-	Dysfunctional Uterine Bleeding
PID	-	Pelvic Inflammatory Disease
HPV	-	Human papilloma virus
SIL	-	Squamous intraepithelial lesions
CIN	-	Cervical intraepithelial neoplasia
LSIL	-	Low grade squamous intraepithelial lesion
HSIL	-	High grade squamous intraepithelial lesion
FIGO	-	Federation International of Gynaecology and Obstetrics
SCC	-	Squamous cell carcinoma
SCJ	-	Squamocolumnar junction
DNA	-	Deoxyribonucleic acid
mRNA	-	*messenger* ribonucleic acid
LVI	-	Lymphovascular invasion
MMMT	-	Malignant mixed Mullerian tumor
FC	-	Follicular cyst
CLC’s	-	Corpus luteal cysts
LH	-	Luteinizing hormone
FSH	-	Follicular stimulating hormone

MPSC	-	Micropapillary serous carcinoma
APST	-	Atypical proliferative serous tumors
TIC	-	Tubal intraepithelial carcinoma
LGSC	-	Low grade serous carcinoma
HGSC	-	High grade serous carcinoma
JGCT	-	Juvenile granulosa cell tumor
AGCT	-	Adult granulosa cell tumor
LDH	-	Lactate dehydrogenase
H & E	-	Hematoxylin and eosin
PBS	-	Phosphate buffer saline
DPX	-	Dibutylphathalate xylene

ABSTRACT

Background: The female genital tract is a vital reproductive organ and a hormone-responsive system. The configuration changes throughout life as hormone levels change, making it prone to developing a variety of non-neoplastic and neoplastic conditions. Hysterectomy is the commonest gynecological surgery done for the management of the pathologies of the female reproductive system.

Aim: To study the histopathological lesions of hysterectomy specimens, the distribution of different lesions in relation to age, and to evaluate histopathological findings in relation to preoperative clinical diagnosis

Methods: This is an observational study of 249 hysterectomy specimens carried out in the department of pathology. Data including age, parity, presenting complaints, type of hysterectomy, and clinical indication for hysterectomy were all obtained from the patient's record. Histopathological analysis of the hysterectomy specimens was done and the findings were evaluated in relation to preoperative clinical diagnosis

Results: In this study, the most common type of hysterectomy was total abdominal hysterectomy with bilateral salpingo-oophorectomy, with 91 cases. The majority of the patients (52%) are in the 41 - 50 age range. The two most common clinical indications were fibroid uterus (90 cases) followed by uterovaginal prolapse (68 cases). Leiomyoma (36.12%) and adenomyosis (7.23%) are the common histopathological findings. Proliferative phase endometrium is the most common endometrial finding (51.41%). Leiomyoma is the most common finding in the myometrium (36.12%). In the cervix, chronic cervicitis (81.5%), followed by

squamous metaplasia (12.4%), in the ovaries, functional ovarian cysts, and in the fallopian tubes Paratubal cysts are the most common pathological finding noted. Endometrial carcinoma was noted in 3 cases. Histopathological examination confirmed the clinical diagnosis in all leiomyoma cases.

Conclusion: In the present study, benign lesions were most common and it gives an insight into the various histopathological patterns of lesions in hysterectomy specimens in our institution. All hysterectomy specimens should be subjected to histopathological examination for accurate diagnosis, proper categorization of lesions, and a better clinical outcome and management.

Key words: Hysterectomy, Leiomyoma, Histopathology, Adenomyosis, carcinoma

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INTRODUCTION

Hysterectomy is one of the most common and significant surgical procedures[1]. In 2019, the prevalence of hysterectomy in India was 3.2%. This was considerably lower than in high-income countries such as the US (26.4%)[2]. Hysterectomy prevalence varied by demographic and socioeconomic factors, with erratic relationships across states[3]. A national-level meta-analysis study done by Desai et al[4] reported that women with higher age, parity, illiteracy, and rural residency had higher odds of having a hysterectomy.

Charles Clay performed the first subtotal hysterectomy in Manchester, England, in 1843; the first total abdominal hysterectomy (TAH) was done by Richardson, MD, in 1929[5]. Currently, three main types of hysterectomy are in use: abdominal, vaginal, and laparoscopic hysterectomy. For gynecological malignancy, pelvic diseases, and an enlarged uterus, abdominal hysterectomy has been considered widely. It continues to serve as a "potential option" if any other method is unable to remove the uterus[2]. Originally used only for prolapse, vaginal hysterectomy is now frequently used for menstrual abnormalities, mainly if the uterus is less than 12 weeks in size[6].

The female genital tract is an important reproductive organ as well as a hormonally responsive system. The configuration of the uterus changes throughout life with changing levels of ovarian hormones[7]. The cervix is prone to many non-neoplastic and neoplastic conditions that are mostly seen in the reproductive age group, and many different types of lesions in the ovaries and fallopian tubes are also seen in different age groups[8]. Women most frequently seek medical attention for

disorders affecting the female genital system, and these conditions are strongly associated with gynecological and reproductive morbidity. Early and accurate diagnosis of female genital tract lesions is required to plan effective and comprehensive treatments and to prevent long-term consequences[5].

Even though many medical and conservative surgical treatment options are existing, hysterectomy still is the most frequently performed major gynecological procedure worldwide next to the cesarian section[1]. It is considered a standard treatment for a variety of benign pelvic pathologies, such as leiomyoma, dysfunctional uterine bleeding (DUB), chronic pelvic pain, endometriosis, adenomyosis, uterovaginal prolapse, and, in some cases, genital tract malignancies[5]. Histopathological examination has a high diagnostic value in patients with carcinoma because adjuvant treatment is determined by the grade and extent of disease invasion.

The incidence and pattern of non-neoplastic and neoplastic lesions of the female genital tract vary according to different age groups and from region to region[7]. Because histopathological examinations of hysterectomy specimens have ethical, legal, diagnostic, and therapeutic implications[10]. So the current study aims to conduct a thorough histopathological examination of all hysterectomy specimens and to analyze various pathological lesions in relation to age, parity, and clinical presentation.

AIMS AND OBJECTIVES

1. To study the histopathological lesions of hysterectomy specimens.

2. To study the distribution of different histopathological lesions in relation to age.

3. To Evaluate histopathological findings in relation to preoperative clinical diagnosis

REVIEW OF LITERATURE

EMBRYOLOGY OF THE FEMALE REPRODUCTIVE TRACT

During the fourth week of embryonic life, the coelomic epithelium proliferates and the underlying mesenchyme condenses to form a pair of longitudinal ridges that resemble the primordial gonads. The Wolffian (or mesonephric) ducts, which connect the developing mesonephros to the cloaca, are located just caudal and lateral to them. The Mullerian (or paramesonephric) ducts, which appear as a longitudinal invagination of the coelomic epithelium on the anterolateral surface of the genital ridge, are developed by the sixth week of development.[11] For a significant portion of their length, these ducts run parallel to the mesonephric ducts; in fact, the Wolffian ducts appear to serve practically as a framework for the later development of the Mullerian ducts. The two Mullerian ducts' cranial portions are kept apart, but as they expand medially and caudally, they come together in the middle and, after being initially divided by a septum, finally combine to form the uterine canal. [12]

The Mullerian tubercle, a small swelling formed by the contact of the fused Mullerian ducts with the posterior wall of the urogenital sinus, develops as a result of the continued caudal expansion of the Mullerian ducts. On either side of the tubercle, the two Wolffian ducts, which are still distinct from one another, enter the urogenital sinus. The Mullerian ducts' caudal fused portions grow into the uterus, while the cranial non-fused portion gives rise to the fallopian tubes. [12]

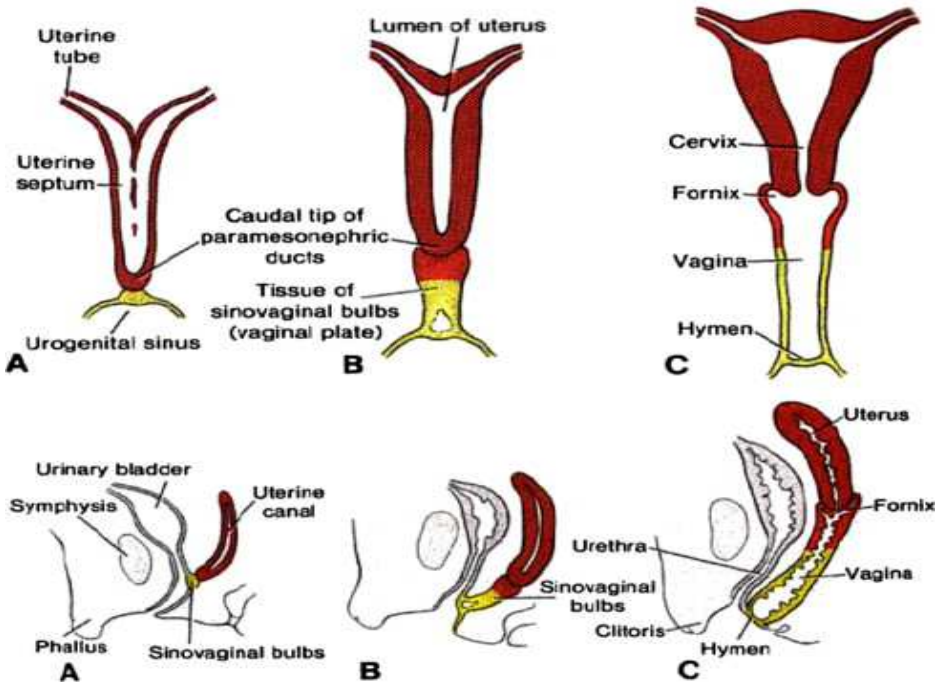


Figure 1 – Development of the female genital system

ANATOMY

The uterus is a pear-shaped organ that is responsible for many functions, including gestation (pregnancy), menstruation, labor, and delivery. The uterus has variable measurements depending on the individual; the average dimensions are 8 cm long, 5 cm across, 4 cm thick, and weighs around 30 to 50 g.[13,14] The uterus subdivides into four main anatomic segments: the fundus, body (corpus), isthmus, and cervix. The term "endometrial cavity" refers to a narrow, triangular area within the body of the uterus. The fundus internal limit acts as the triangle's base, while the cervical internal os acts as the triangle's angle. The right and left cornua serve as the route for communication between the fallopian tube and the endometrial cavity.[15]

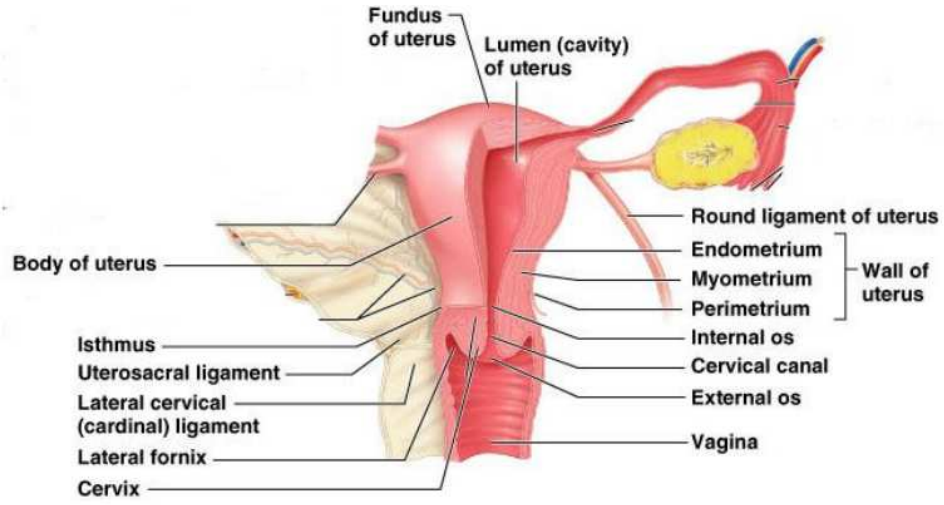


Figure 2 – Anatomy of Uterus

HISTOLOGY

Three layers make up the uterine body's histological architecture:

- Endometrial layer
- Myometrial layer
- Serosal layer

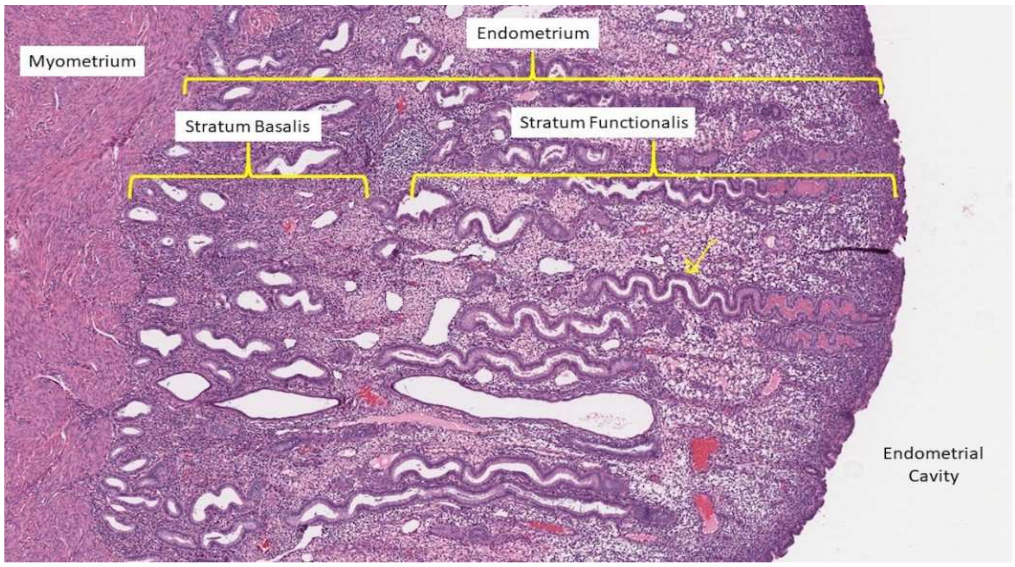
Endometrium: It's the mucosal lining of the uterine cavity, composed of endometrial glands and specialized stroma. It has been divided into 3 histologically and functionally distinct layers.

Stratum Basalis: It's the deeper layer of the endometrium, adjacent to the myometrium. It is not shed during menstruation and replenishes the stratum functionalis afterward. The stromal cells of this layer are deeply stained and closely packed.

The stratum spongiosum and stratum compactum form the "stratum functionalis," which is hormonally responsive, undergoes functional and morphological changes throughout the menstrual cycle, and sheds during menstruation. [16]

Stratum spongiosum- A broad intermediate layer characterized by the spongy appearance of the stroma.

Stratum compactum- It is the thinner superficial layer and has a compact stromal appearance. [16]



Photomicrograph 1 – Histology of Endometrium (33)

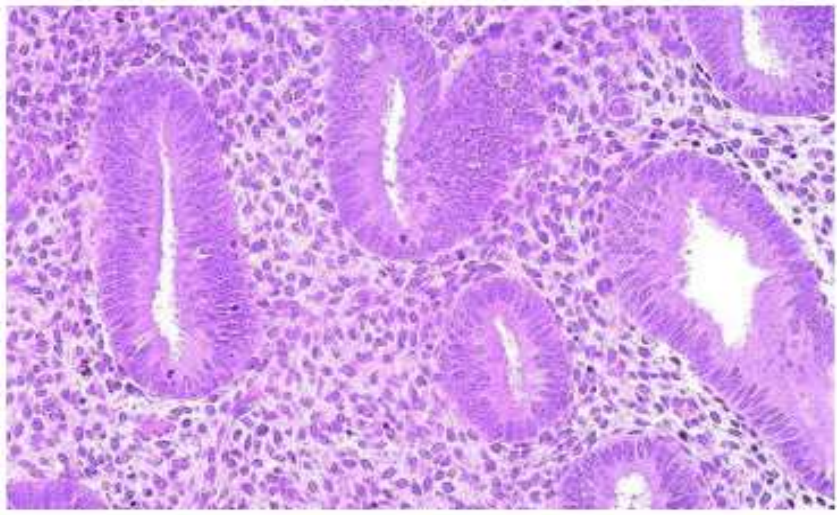
Myometrium: It's the structural wall of the uterus and consists primarily of smooth muscles and interlacing bundles of long, slender fibres arranged in ill-defined layers.

Menstrual cycle:

Hitschmann and Adler presented the first histological description of the cyclical changes in the endometrium in 1908[14].

Noyes et al. described the "dating of endometrium" in detail for the first time in 1950, using histological criteria to determine how far an endometrium has developed in the menstrual cycle based on the characteristic histological changes that occur at specific times. Later, the data was confirmed by Moricard (1954) and Philippe et al. (1965)[14]. The three phases of the menstrual cycle are

- Proliferative phase
- Secretory phase
- Menstrual phase



Photomicrograph 2- Proliferative phase

Proliferative phase:

The basal layer and the part of the functionalis layer that is still intact after menstruation is used to regenerate the endometrium. The proliferative phase is divided into, early, mid, and late proliferative phases[17].

In the early phase, the glands resemble simple tubules and are lined with a low columnar epithelium. There is stromal and glandular evidence of mitotic activity. Spindle cells make up the loosely packed stroma. The elongation of glands continues during the mid-proliferative phase; as a consequence, they outgrow the stromal edema and acquire tortuosity, and in the late proliferative phase, the stromal edema reduces and the tortuosity of glands increases[16]. The maximum of the mitotic activity in the glands and stroma corresponds with the estrogen peak before ovulation. The glandular epithelium shows pseudo-stratification, and some glycogen vacuoles can be seen within the cells. [17]

Secretory phase:

The postovulatory corpus luteum secretes progesterone, it causes glandular secretions, stromal maturation, and vascular differentiation that are characteristics of the secretory phase, and it is divided into three phases:

Early secretory phase (days 16-18)

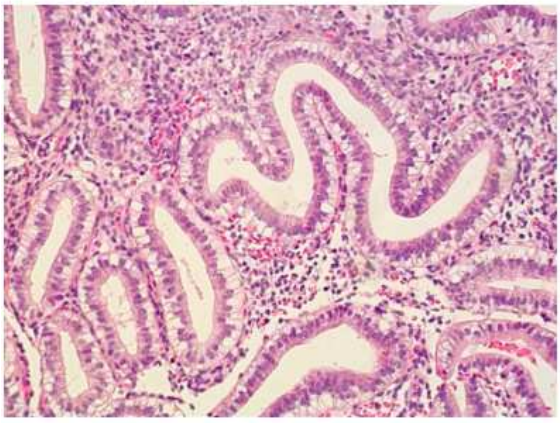
Mid-secretory phase (days 19-23)

Late secretory phase (days 24-28)

In the early secretory phase, the endometrial glands still has a tubular appearance. Subnuclear vacuoles appear within the glandular epithelium on the 16th day of the cycle and reach their peak between the 17th and 18th days of the cycle. Initially identified within a few cells, they eventually involve almost all cells in the majority of the glands. The stroma of the early secretory phase and the late proliferative phase cannot be distinguished.

In the mid-secretory phase, cytoplasmic vacuoles become supranuclear, and the degree of glandular secretion increases, with most of the secretions occurring within glandular lumina. The glands are often angular in shape, and the stromal cells are recognized as predecidual cells because they have more conspicuous eosinophilic cytoplasm and there is a progressive increase in stromal edema.

In the late secretory phase, glandular secretory activity is diminished and the glands become serrated. Predecidual stromal change increases, resulting in the formation of stratum compactum[17].



Photomicrograph 3 - Secretory phase

Menstruation:

If pregnancy has not resulted, a substantial portion of the endometrium is shed at the end of each cycle. Normally, it lasts for four days or less. The endometrial glands are collapsed, the stroma is condensed, and further collapsed. The stromal cells aggregate into one tightly packed balls (stromal blue balls). The stroma and glands both contain necrotic debris, neutrophilic infiltration, interstitial hemorrhage, fibrin deposition, and apoptotic bodies[16].

HISTOLOGY OF CERVIX:

The cervix represents the distal portion of the uterus and is divided into the ectocervix and the endocervix.

Ectocervix: Is lined by stratified non-keratinizing squamous epithelium, consists of:

Basal cells: It is the deeper layer, responsible for continuous epithelial renewal. These are oriented perpendicular to the basement membrane and have uniform oval nuclei, dense nuclear chromatin, and sparse cytoplasm.

Parabasal cells: These are located just above the basal cells, and have slightly more cytoplasm than basal cells.

Intermediate cells: The cytoplasm in these cells is abundant and clear due to glycogen accumulation.

Superficial cells: These are the most mature cells and are flat, oriented parallel to the basement membrane. The cells have abundant clear cytoplasm and small, round nuclei[16].

Endocervix: Lined by single columnar cells with dense, uniform, oval, nuclei at the base and apical mucin. They form infoldings, clefts, and glands of variable shapes.

Squamocolumnar junction (SCJ): This is the histologic junction between the squamous epithelium of the ectocervix and the columnar epithelium of the endocervix. Its location on the cervix is variable. Age and hormonal status influence the location of SCJ[18].

Cervical stroma is mainly composed of collagen fibers, which are dense in the ectocervix and loosely surround the endocervical glands[16].

Fallopian Tubes:

Uterine tubes and oviducts are other terms. The fallopian tubes are hollow, tubular, and paired structures, that measure 8-10cms in length and are situated along the upper border of the broad ligament that extends laterally from the uterus. The major functions are to transport the ovum and to facilitate fertilization and the survival of the zygote [13,15].

The fallopian tubes are anatomically divided into four sections (medial to lateral).

1. Intramural portion: It opens into the uterine wall.
2. Isthmus: It's the narrowest section and connects the ampulla to the uterine cavity.
3. Ampulla: It's the widest section and the usual site of fertilization.
4. Infundibulum: opens near the ovary, to which fimbriae are attached to direct the ovum into the tube [15].

It consists of three layers:

- Outer serous layer: It is made up of loose connective tissue and is continuous with the broad ligament.
- Muscular layer: Inner circular and outer longitudinal smooth muscle layers.
- Inner mucosal lining: It is thrown into branching longitudinal folds and lined by columnar epithelium consisting of ciliated, non-ciliated secretory, and intercalated cells[16].

Ovary

The ovaries are paired and oval gonads in females, and they are connected to the posterior surface of the broad ligament by the mesovarium. The suspensory ligament of the ovary contains neurovascular structures, it connects the ovary to the pelvic wall, and the ovarian ligament connects it to the uterus.

The primary functions of the ovaries are germ cell maturation, storage, release of mature ovum and steroidogenesis (the production of estrogen and progesterone in response to LH and FSH gonadotropins)

The ovary is covered by germinal epithelium, and it consists of fine collagen fibers, spindle-shaped cells, and ground substances that are altogether known as the ovarian stroma. The peripheral zone and the central zone of the ovary are known as the cortex and the medulla, respectively[15].

Cortex: It consists of the tunica albuginea, a dense layer of connective tissue covered by the germinal epithelium. During the reproductive period, the cortex is studded with numerous follicular structures in various stages of development. These are associated with the development and production of sex hormones. These structures include the corpus luteum, graafian follicles, maturing follicles, and primordial follicles.

Medulla: It consists of blood vessels, lymphatics, nerves, loose connective tissues, and smooth muscles. There is a small collection of cells called "hilus cells" which are homologous to the interstitial cells of the testis[16].

AGE-RELATED CHANGES IN THE UTERINE STRUCTURE

Age, hormonal status, pregnancy, and the health of the surrounding pelvic organs affects the shape, size, and location of the uterus. The cervix is significantly larger than the body of uterus throughout fetal life, and the uterus project above the pelvic brim.

At puberty, the uterus is pyriform and weighs 14–17 g. Following that, the fundus is located below the superior pelvic aperture. Significant palmate folds of the cervical region are present and extend to the upper uterine cavity. During menstruation, the organ is slightly enlarged and become more vascular [13].

During pregnancy, the uterus is enlarged due to hypertrophy and hyperplasia of myocytes, in late pregnancy the uterus becomes thinner and involutes afterwards but never returns to its original state[13,17].

The uterus atrophies with age, becoming paler and denser in texture, and the constriction between the body and cervix becomes more pronounced.

Types of hysterectomy

There are mainly two types of hysterectomies. Total hysterectomy means removing the body, fundus, and cervix of the uterus; often called complete hysterectomy. Partial hysterectomy/subtotal hysterectomy means removal of the uterine body while leaving the cervix intact; also called as supracervical hysterectomy. Oophorectomy is unilateral or bilateral removal of ovaries is frequently done together with hysterectomy. Salpingectomy is removal of unilateral or bilateral fallopian tubes.

Hysterectomy is usually performed by a) Abdominal b) Vaginal and c) Laparoscopic routes. The proportion of hysterectomies performed laparoscopically is immensely increasing, as it has several benefits[2].

A meta-analysis study with 3643 participants compared the primary outcomes (return to normal activities, any intra-operative visceral injury, major long-term complications like fistula formation, urinary dysfunction) and secondary outcomes (operation time, duration of hospital stay, and need for blood transfusion) of various hysterectomies. The study stated that vaginal hysterectomy is preferable to abdominal hysterectomy as it has the advantages of fewer blood transfusions, less febrile morbidity, and a lower risk of injury to the ureter. Laparoscopic hysterectomy is advantageous, as it has the possibility of diagnosing and treating other pelvic diseases[3].

Leiomyoma:

Leiomyomas are the most common neoplasm and are the leading cause of hysterectomy all over the world. They are clinically observed in 20-30% of women over the age of 30 years, and they are most frequently detected during the mid-reproductive years[19]. Leiomyomas are benign tumors of smooth muscle cells with varying amounts of fibrous tissue[20].

As estrogen and progesterone receptors are present over leiomyomas, their growth, development, and maintenance are affected by the hormonal milieu and these receptors can be demonstrated immunohistochemically. As a consequence of these receptors, leiomyomas show rapid growth during estrogen therapy, progesterone hormonal replacement therapy, and with clomiphene usage, shrink after menopause or with the usage of gonadotropin-releasing hormone[21].

The most common site of occurrence is the uterine corpus, but they have also been seen in the cervix, broad ligament, ovaries, and other pelvic ligaments.

Clinical features:

Depending on the size and location of leiomyomas, their clinical presentation varies. More than half of patients are asymptomatic and are discovered during a routine pelvic examination or during pregnancy[22].

Most of the patients, present with menstrual complaints like menorrhagia, metrorrhagia, polymenorrhoea, or dysmenorrhoea. Other complaints include infertility and recurrent pregnancy losses. Due to pressure effect, patients may present with difficulty initiating micturition, increased frequency, low backache, and discomfort in the lower abdomen.

Gross Features:

Leiomyomas can be single or multiple lesions (leiomyomatosis) of varying sizes, ranging from microscopic foci to large bulky tumors, they are spherical and firm in consistency, and on cut sections, they are well-circumscribed, gray-white, and have a characteristic whorled appearance.

In the myometrium they can be located anywhere, on the cut section they bulge above the surrounding myometrium and are easily shelled out[24,25].

Figure 3 – Gross of leiomyoma

Within the uterus tumors can be present:

- Submucosal
- Intramural (most common)
- Sub serosal
- Others are pedunculated and parasitic.

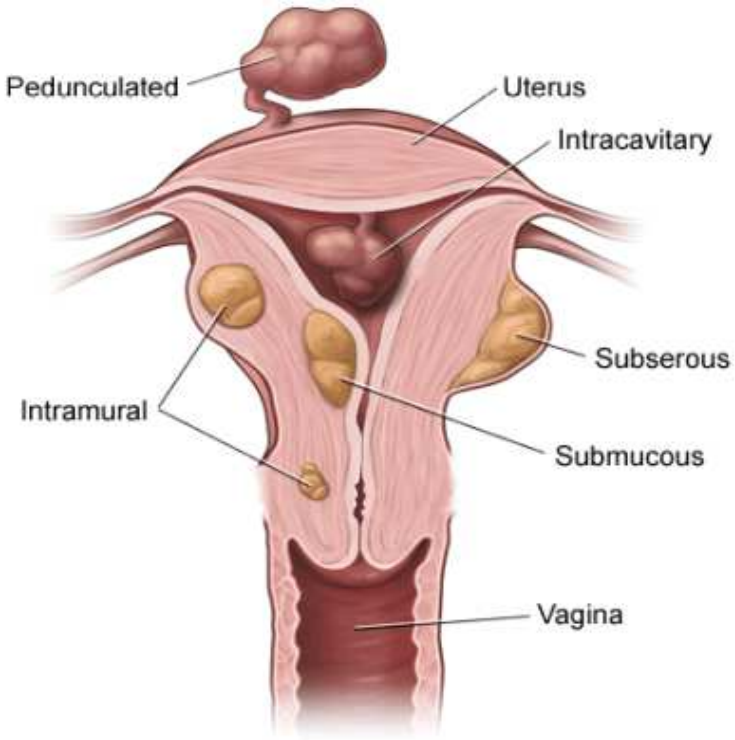


Figure 4- Sites of fibroid

Microscopic Features:

Leiomyoma is composed of smooth muscle bundles arranged in a whorled pattern or interlacing fascicles. These smooth muscle cells are elongated and spindle-shaped, having abundant eosinophilic cytoplasm, and the nuclei are cigar-shaped with tapered ends, finely dispersed chromatin, and small nucleoli[24].

Leiomyoma Variants:

Red degeneration:

It is characterized by extensive coagulative necrosis, most frequently seen in submucosal leiomyomas, and is often associated with pregnancy or undergoing high-dose progestin therapy[25].

Apoplectic leiomyoma:

It is characterized by the presence of stellate zones of recent hemorrhage and the absence of coagulative necrosis within nodules of hypercellular smooth muscles. Most frequently encountered in individuals who use oral contraceptives, are pregnant, or in postpartum[24].

Cellular leiomyoma:

As the term suggests, these tumors have increased cellularity but neither show atypical features nor an excessive number of mitotic figures.

Mitotically active leiomyoma:

They have increased mitotic activity up to 15 mitotic figures/10 HPF. The increased mitotic index may result from increased progesterone levels or progestin-only pills.

Fumarate Hydratase deficient leiomyoma:

These leiomyomas are densely cellular, with focal nuclear atypia, multinucleation, large nucleoli surrounded by peri nucleolar halos, and cytoplasmic eosinophilic inclusions with staghorn vasculature.

Leiomyoma with bizarre nuclei

They are described as simplistic and atypical leiomyomas. [24] Microscopically, these leiomyomas have bizarrely shaped, enlarged, hyperchromatic, multilobulated nuclei with pseudonuclear inclusions arranged in the background of typical leiomyomas[26].

Epithelioid leiomyoma:

These tumors were formerly classified as leiomyoblastoma, clear cell leiomyoma, and plexiform leiomyoma. Grossly, these are yellow or gray, may contain areas of hemorrhage, and are often softer than the usual leiomyomas. The tumor cells are round or polygonal, with relatively large, round, and centrally positioned nuclei. These epithelioid leiomyomas include leiomyoblastomas with eosinophilic cytoplasm, clear cell leiomyomas with an abundance of clear cytoplasm and distinct cell membranes, and plexiform leiomyomas with round cells organized in cords and sheets[24].

Myxoid Leiomyoma

These tumors are soft, translucent, and well-circumscribed. Microscopically, these are hypocellular, with abundant amorphous myxoid material between the smooth muscle cells, but there is no necrosis, mitosis, or cytologic atypia[24].

Diffuse leiomyomatosis:

Numerous small smooth muscle nodules, ranging in size from microscopic to 3 cm, are diffusely distributed and cause symmetrical uterine enlargement. They are less circumscribed, lack atypia, exhibit tumor cell necrosis, and have minimal mitotic activity.

Dissecting leiomyoma:

Dissecting leiomyomas are composed of nodules of smooth muscle cells that dissect into the myometrium around them, as well as occasionally into the broad ligament and pelvis with hydropic changes and intravenous extension being seen.

They are also known as cotyledonoid dissecting leiomyomas when the extrauterine extension is seen[24].

Hysterectomy and leiomyoma:

Medical management options for uterine leiomyomas have limited value because of their moderate efficacy and associated adverse effects[28]. Myomectomy is an alternative to hysterectomy for women who wish to retain their uterus, regardless of their fertility desire[27]. Hysterectomy is done especially for large, rapidly enlarging, and symptomatic leiomyomas.

Mesenchymal tumors

These are rare uterine mesenchymal neoplasms that constitute approximately 20% of uterine sarcomas and less than 10% of all uterine malignancies[29].

WHO has classified stromal tumors into four categories: (a) Endometrial stromal nodule (ESN) (b) Low grade endometrial stromal sarcoma (LG-ESS) (c) High grade endometrial stromal sarcoma (HG-ESS) (d) Undifferentiated uterine sarcoma (UUS). These categories are defined by the presence of distinct translocations as well as tumor morphology (myometrial or vascular invasion) and prognosis[30].

Endometrial stromal nodule (ESN):

These tumors are rare and they tend to occur in perimenopausal women. Often patients present with abnormal uterine bleeding, they also may complain of pelvic or abdominal pain or pelvic mass. Grossly these nodules are typically well-circumscribed, nonencapsulated, soft, fleshy yellow, solitary lesions[24].

These tumors have an expansile growth pattern under the microscope and lack lympho-vascular invasion in addition to their monotonous proliferation of bland endometrial stromal cells. If tumor cells are infiltrated, they should be less than 3 mm in their largest dimension.

Low-grade endometrial stromal sarcoma (LG-ESS):

Premenopausal and postmenopausal women are more likely to develop these malignancies. These tumors have been associated with prolonged use of estrogen or tamoxifen as well as pelvic radiation. Grossly they are poorly circumscribed, soft, yellow to whitish rounded nodules invading the myometrium.

On microscopy, these tumors consist of well-differentiated endometrial stromal cells with mild nuclear atypia and infiltrate the myometrium as irregular tongues, often with lympho-vascular invasion[31].

High-grade endometrial stromal sarcoma (HG-ESS):

These tumor subtypes can be distinguished from one another morphologically, immunohistochemically, and molecularly. They include

Sarcomas with *YWHAE-NUTM2A/B* fusions - Consists of round tumor cells in a nested pattern with little to moderate eosinophilic cytoplasm and nuclear atypia.

Sarcomas with *BCOR* rearrangement- consists of fascicles with extensive myxoid stroma and atypical spindle shapes.

Sarcomas with *BCOR* internal tandem duplication (ITD) have high-grade round cell and spindle cell components with uniform atypia, lymph vascular invasion, and tumor cell necrosis[24,31].

Dysfunctional Uterine Bleeding

DUB is a symptom complex that includes any condition with abnormal uterine bleeding in the absence of any clinically detectable pelvic pathology, pregnancy, or neoplasm[32].

Classification of DUB:

Primary DUB

- DUB due to a primary dysfunction in the uterus, ovary, pituitary, hypothalamus, or higher centers.
- No detectable disease was responsible for bleeding either in the genital tract or elsewhere.

Secondary DUB:

- IUCD or administration of sex hormones for contraception or any purpose (iatrogenic)

Organic disease outside the genital tract i.e, hypothyroidism, leukemias, or bleeding disorders[24,25].

Clinical presentation of DUB [32]

Patients with DUB might present clinically with menorrhagia, polymenorrhagia, continuous prevaginal bleeding, intermenstrual bleeding or premenstrual bleeding.

Hysterectomy and DUB

In the reproductive period, a hysterectomy is justified when conservative treatment fails and blood loss impairs the health and especially if the patient is approaching 40. But even in women in their thirties, prolonged procrastination of symptoms despite of hormone therapy is often an indication for a hysterectomy. In the presence of hyperplastic endometrium, a hysterectomy should be employed. There is no place for hormone therapy in postmenopausal DUB[32].

ADENOMYOSIS

Adenomyosis is a common benign condition of the uterus. It is distinguished by the presence of endometrial glands and stroma within the myometrium at least a distance of 2.5 mm from the endometrial junction. Due to adenomyosis, the myometrium undergoes hyperplasia and hypertrophy, enabling the uterus to enlarge[33].

Clinical Features

Patients with adenomyosis are typically pre or perimenopausal women with symptoms of dysmenorrhea and menorrhagia and these symptoms tend to be more severe with deep myometrial involvement. The uterus is enlarged and may contain additional lesions associated with hyperestrogenism[34].

Gross Appearance

The uterus with adenomyosis may appear normal or enlarged. On the cut section, myometrium is trabeculated with petechia-like gray foci of the endometrium[35].

Microscopic appearance

There are both benign endometrial glands and normal-appearing endometrial stroma with at least one HPF below the endometrial myometrial lining. The glandular tissue is usually proliferative or inactive type of endometrium. Often myometrial smooth muscle proliferation is present around endometrial islands[35].

Adenomyosis and Hysterectomy

Clinical and pathological diagnosis is impossible without a hysterectomy, except when the "myomectomy" specimen proves histologically to be an adenomyoma rather than a leiomyoma.

Although adenomyosis is not a clinical diagnosis, history and clinical findings may lead one to correctly suspect the correct diagnosis. No form of therapy short of hysterectomy has been consistently successful in controlling patient symptoms[35].

WHO (2020) classification of tumors of the cervix [36]

Precursor lesions:

- Squamous metaplasia
- Atrophy of the uterine cervix

Squamous cell tumors and precursors

- Condyloma acuminatum
- Squamous intraepithelial lesions of uterine cervix
- Squamous cell carcinoma, HPV associated
- Squamous cell carcinoma, HPV independent
- Squamous cell carcinoma, NOS (not otherwise specified)

Benign glandular lesions

- Endocervical polyp
- Nabothian cyst
- Tunnel clusters
- Micro glandular hyperplasia
- Lobular endocervical glandular hyperplasia
- Tubo -endometrioid metaplasia
- Endo cervicosis of the uterine cervix

Adenocarcinomas:

- Adenocarcinoma in situ, HPV associated
- Adenocarcinoma, HPV associated
- Adenocarcinoma in situ, HPV independent
- Adenocarcinoma, HPV independent (gastric type, clear cell type, mesonephric type)

Other epithelial tumors:

- Carcinosarcoma of the uterine cervix
- Adeno squamous and mucoepidermoid carcinoma
- Adenoid basal carcinoma
- Carcinoma of the uterine cervix, unclassified

Mixed epithelial and mesenchymal tumors

- Adenomyoma
- Adenosarcoma

Germ cell tumors

- Germ cell tumors of the uterine cervix

Endocervical polyp:

Endocervical polyps are the most common growth of the uterine cervix, often seen in multigravidas during their fourth to sixth decades of life. They may present with profuse mucoid discharge or abnormal bleeding[37]. These polyps are focal, hyperplastic protrusions of the substantia propria and epithelium of the endocervical folds. Grossly these are usually small and single, in a few cases, large polyp protrudes into the canal mimicking malignant tumors.

Histologically many different patterns are observed. It includes Polyps containing Nabothian cysts and with squamous metaplasia, glandular hyperplasia, predominant blood vessels, or marked inflammation[38].

The term "mixed polyps" refers to polyps that arise from the isthmus and frequently contain both endocervical and endometrial-type epithelium[38].

Squamous metaplasia of cervix:

In the cervix, stratified squamous epithelium replaces preexisting columnar epithelium, and it occurs by two distinct methods.

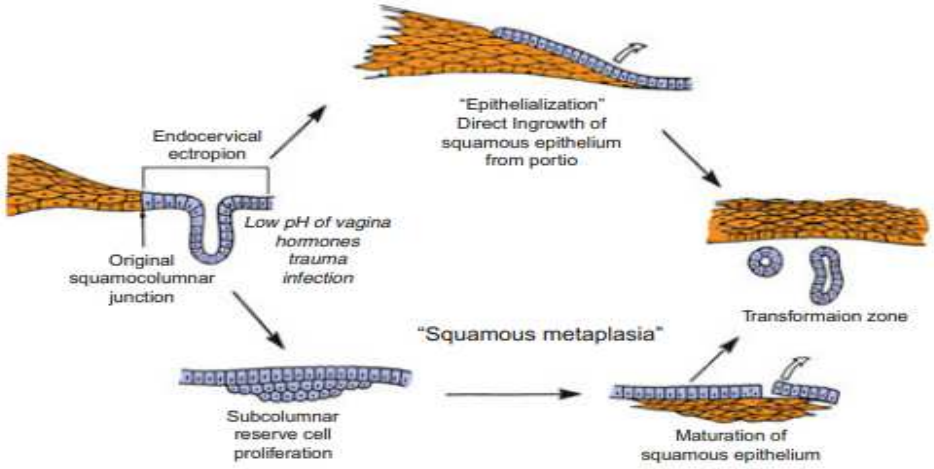


Figure 5: Squamous metaplasia by two histogenic mechanisms[50]

'Squamous epithelialization' refers to the direct extension of the stratified squamous epithelium into the endocervix.

'Squamous metaplasia' is the process through which sub-columnar reserve cells proliferate and they subsequently mature into the squamous epithelium[38]

Squamous intraepithelial lesions (SIL):

Classification systems for squamous cervical precursor lesions:

Table 1: Terminologies for cervical cancer precursor lesions

Older Classification	WHO Classification	Bethesda System, current classification
Mild dysplasia	CIN I	LSIL
Moderate dysplasia	CIN II	HSIL
Severe dysplasia	CIN III	HSIL

Overtime classification of cervical precursor lesions has evolved.

Initially in WHO classification, mild dysplasia was termed as CIN 1, moderate dysplasia as CIN 2, and severe dysplasia as CIN 3[39].

As patient management is two-tiered, that is either observation or surgical treatment. The classification has been simplified, with CIN 2 renamed as low-grade squamous intraepithelial lesion (LSIL) and CIN 2 and CIN 3 combined into one category and referred as high-grade intraepithelial lesion (HSIL)[25].

Characteristic histologic and cytologic changes induced by HPV are nuclear atypia and koilocytic changes. Nuclear atypia is characterized by hyperchromasia, size and shape variation, and nuclear enlargement. Koilocytic atypia refers to nuclear changes associated with perinuclear halos[25,39].

Low-grade squamous intraepithelial lesions (LSIL):

The three morphological subtypes are

1- Condyloma acuminatum (exophytic condyloma)
2- Immature condyloma (papillary immature metaplasia)
3- Flat condylom

It represents a productive HPV infection with high level of viral replication rate. The immature squamous cells are confined to the lower one-third of the epithelium with minimal nuclear atypia[25,40].

High-grade squamous intraepithelial lesions (HSIL):

HSIL is characterized by nuclear atypia in all layers of the epithelium with a variable degree of surface maturation.

These are typically correlated with high-risk HPV infection, which causes progressive dysregulation of the cell cycle that leads to increased cellular proliferation and a lower rate of viral replication[40].

Table 2: Features to distinguish LSIL and HSIL

FEATURES	LSIL	HSIL
Types of HPV	Anogenital HPV	High risk HPV
koilocytosis	Seen frequently	Occasionally seen
Abnormal mitotic figures	Absent	Frequent
Undifferentiated cells and mitotic figures are confined to	Lower 1/3rd	Upper 2/3rd

SIL and hysterectomy:

Hysterectomy is a definitive treatment for HSIL of the cervix because it is associated with relapses and can develop into invasive carcinoma and also considered for patients who develop recurrent cervical dysplasia even after conization or loop procedure[40,41].

CARCINOMA OF CERVIX:

Cervical cancer is the 4th most common cancer among women, worldwide[42].

SQUAMOUS CELL CARCINOMA (SCC) HPV ASSOCIATED:

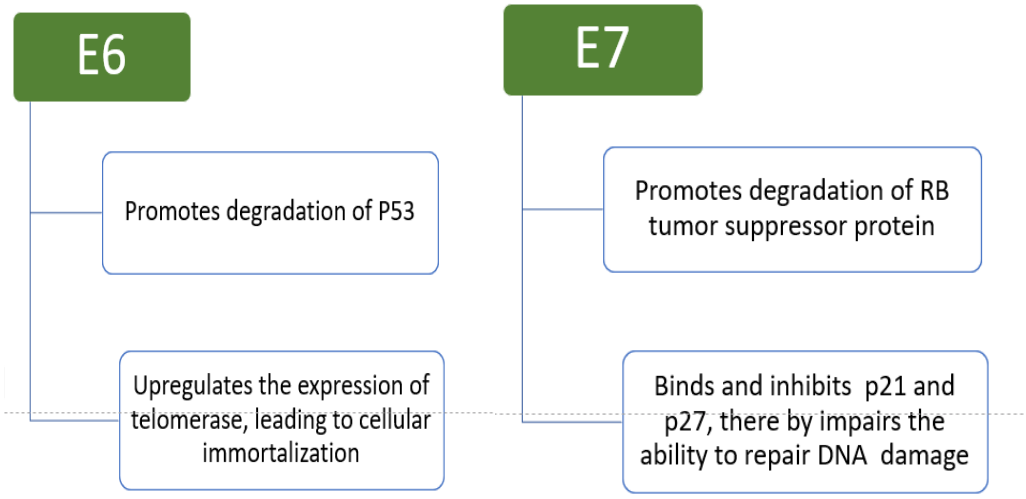
WHO classified invasive squamous lesions into two main categories: HPV-associated and HPV-independent lesions. HPV status is necessary as no morphological criteria can reliably differentiate HPV-associated and HPV-independent squamous cell carcinoma of the cervix[43].

SCC is the most common type of cervical carcinoma, and most of the patients are 40–54 years old. Nearly all cases are associated with high-risk HPV (HPV16 > HPV18) and arise from precursor lesions. With cervical cancer screening programs, there has been a 75% decline over the past 50 years[25].

Pathophysiology:

The prevalence of high-risk HPV infections among adolescents and young women is very high. Most HPV infections are transient, the high-risk HPVs take longer to clear than the infections with low-risk HPVs. Persistent infection increases the risk of the development of cervical precursor lesions and subsequent carcinoma[25]. The ability of HPV to act as a carcinogen depends on the E6 and E7 oncoproteins[25,44].

Table 3: Pathogenesis of HPV



Etiology:

- Persistent infection by high-risk HPVs (HPV16 is the major causal agent)
- Previous or concurrent history of HSIL[25].

Associated risk factors: [25]

- Higher number of sexual partners
- Contact with infected partner resulting in infection.
- Immunodeficiency, transplantation.
- Prolong usage of oral contraceptives
- Multiparity and early age at first birth
- Positive family history
- Cigarette smoking, concurrent STDs

Gross appearance:

Early-stage tumors may exhibit focal induration, ulceration, or manifest as a friable, raised granular region that bleeds easily. Most advanced tumors may manifest as fungating (exophytic) or infiltrative masses[44].

Microscopic appearance: [44]

Tumor cells are infiltrating into the cervical stroma either as irregular anastomosing nests or as single cells. Lymphovascular invasion may be noted.

Morphological variants:

Keratinizing variant: Keratin pearls within the epithelium are one of its defining features. single keratin pearl is sufficient for the diagnosis. The squamous neoplastic cells have abundant keratohyalin granules and intercellular bridges.

Non-keratinizing variant: Tumor cells lack keratin pearl formation, yet they are polygonal and form nests, and sheets, with intercellular bridges. These cells have large nuclei with unevenly distributed, coarsely granular chromatin, and numerous mitoses.

Papillary variant: The presence of papillae with a fibrovascular core and an atypical squamous epithelium similar to HSIL is the distinguishing feature. These papillae can be either thin or broad.

Basaloid pattern: Immature basaloid cell nests that are well-defined and resemble HSIL cells have peripheral palisading of these tumor cells, including hyperchromatic nuclei, sparse cytoplasm, and central comedo necrosis[45].

Warty carcinoma: It has marked condylomatous changes with a warty surface. Koilocytic changes and keratinization of neoplastic cells may be seen.

Verrucous carcinoma: It is a very rare malignant tumor with frond-like acanthotic squamous epithelium that grows slowly and is locally invasive. The base of the tumor is broad and expansile, with pushing margins. Mitosis is rare, with minimal cytologic atypia[44].

Lymphoepithelial-like carcinoma: These tumors exhibit distinctive nests of undifferentiated neoplastic cells with abundant cytoplasm and regular vesicular nuclei, which are surrounded by a noticeable inflammatory infiltrate. Cells with indistinct borders impart a syncytial-like appearance. These tumor cells lack intercellular bridges and keratinization[44].

MODIFIED BORDER'S GRADE:

It is based on the amount of keratin, nuclear pleomorphism, size of nucleoli, necrosis, and mitotic activity[36,46].

- **Well- differentiated tumors:** These tumors show the presence of squamous cells with abundant keratinization with the formation of keratin pearls. Neoplastic cells are large with eosinophilic cytoplasm and consist of intercellular bridges. Occasional mitosis and necrosis may be present.
- **Moderately differentiated tumors:** These tumors show neoplastic cells with less distinct cell outlines and cytoplasm compared to well differentiated tumors and increased nuclear pleomorphism and high mitotic figures.
- **Poorly differentiated tumors:** These tumors are arranged in sheets and single cells and these tumors show the presence of primitive cells with scant

cytoplasm, enlarged hyperchromatic nuclei, and increased mitotic figures. Keratinization is occasional or absent.

SQUAMOUS CELL CARCINOMA, HPV- INDEPENDENT:

These tumors constitute only 5-7% of all squamous cell carcinomas. There is no difference in clinical features from that of HPV-associated SCC.

p16 IHC is an acceptable biomarker and it shows a negative result. For the diagnosis of HPV, molecular techniques of detection of HPV DNA or mRNA are necessary[43,44].

SQUAMOUS CELL CARCINOMA, NOS (NOT OTHERWISE SPECIFIED):

The diagnosis of SCC NOS is done where p16 IHC or HPV testing is not available[44].

GLANDULAR TUMORS OF THE CERVIX:

Adenocarcinoma of cervix HPV associated:

Adenocarcinoma is the 2nd most common type of cervical carcinoma[47]. It constitutes5-25% of invasive cervical carcinomas[48]. Adenocarcinoma associated with HPV (most commonly HPV 18 and 16) infection renders 85-90% of adenocarcinomas. And are more frequently seen in young women. Patients are often present with pelvic pain and vaginal bleeding[49].

Histological types [50]

- Villoglandular variant
- Mucinous variant (endocervical type, intestinal type, and stratified mucin-producing variant) on microscopy, the number of glands have increased and they are irregularly shaped, angulated, and fragmented with cribriform and micro-acinar architecture.

The Villoglandular variant has a characteristic surface projections made up of finger-like papillae, lined by columnar epithelium with hyperchromatic and enlarged nuclei with loss of polarity and coarse chromatin.

PATTERN-BASED CLASSIFICATION FOR HPV-ASSOCIATED ADENOCARCINOMA OF CERVIX (SILVA SYSTEM) : [50,51]

For this system, tumor specimens should be entirely submitted and they exclude the extent of the tumor's invasion.

Pattern A- The tumor has a non-destructive pattern of invasion, no associated risk for nodal metastasis or recurrence, and can be treated conservatively

Pattern B & C- these are potentially aggressive and have a destructive pattern of invasion either focally or diffusely. There is an associated risk for nodal metastasis and recurrence. Patients need hysterectomy with lymphadenectomy and chemoradiation.

Table 4: Silva pattern-based classification for HPV-associated adenocarcinoma of cervix

Pattern -A	Pattern-B	Pattern-C
-Well demarcated glands with round counter. -LVI – absent -complex intraglandular growth acceptable. (papillary, cribriform) -Lack of solid growth	- Small group of cells or individual cells -focally desmoplastic or inflammed stroma. -LVI may be present or absent. -lack of solid growth	-Diffusely infiltrative glands with extensive desmoplastic stroma. -Glands are angulated with the canalicular pattern. -Confluent growth and it fills the entire 4x field (5mm) -solid and poorly differentiated components (large tumor cells arranged in sheets). - Bands of lymphocytic infiltrates. -LVI may be present or absent.

Adeno squamous carcinoma:

These carcinomas consist of both glandular and squamous components and they constitute 10% of cervical carcinomas[52]. Squamous component is poorly differentiated and the glandular component is endocervical and poorly differentiated with either intracytoplasmic mucin or intraluminal mucin component infiltrating into the stroma[52].

Table 5: FIGO staging for carcinoma of the cervix 2018: [53]

Stage I:	Carcinoma confined to the cervix
IA	Carcinoma diagnosed only on microscopy, with <5mm of the depth of invasion
IA1	Stromal invasion<3mm in depth
IA2	Stromal invasion>3mm and <5mm
IB	Depth of invasion>5mm
IB1	Stromal invasion >5mm and <2cm in greatest dimension
IB2	Stromal invasion >2cm and <4cm in greatest dimension
IB3	Stromal invasion > 4cm in greatest dimension
Stage II	Carcinoma invades beyond cervix, but not extended into lower1/3rd of vaginal and pelvic wall
IIA	Involves upper 2/3rd of vagina without parametrial involvement
IIA1	Invasion <4cms in greatest dimension
IIA2	Invasion>4cms in greatest dimension
IIB	Parametrial involvement without involving pelvic wall
Stage III	Involves lower 1/3rd of vagina, pelvic wall, causes hydronephrosis, involves pelvic and para-aortic lymph nodes
IIIA	Involves lower1/3rd of vagina
IIIB	Involves pelvic wall/ hydronephrosis or non-functioning kidney
IIIC	Involves pelvic wall/ paraaortic lymph nodes (irrespective of tumor size)
IIIC1	Pelvic lymph nodes are involved
IIIC2	Paraaortic lymph nodes involved
Stage IV	Extends beyond true pelvis/ involves mucosa of the bladder or rectum
IVA	Involves adjacent organs
IVB	Involves distant organs

Hysterectomy and carcinoma cervix:

The three modalities for primary treatment are surgery, radiation, and combination. For young women, and patients with stage IA1/2 and IB1 lesions who desire to preserve fertility, radical trachelectomy combined with laparoscopic pelvic lymphadenectomy has been used. For bulky IB2 or any further stage lesions, radiation therapy followed by extra fascial hysterectomy has been accepted. The 5-year survival rate for stage 1 patients is 90-95% with radical hysterectomy[54].

ENDOMETRIAL HYPERPLASIA:

It is defined as an abnormal proliferation of the endometrial glands, relative to the stroma, resulting in an increased gland-to-stromal ratio when compared with normal proliferative endometrium[25].

Endometrial hyperplasia is a frequent precursor to the most common type of endometrial adenocarcinoma[55].

Increased endogenous or exogenous estrogen and unopposed progesterone can cause hyperplasia. Some of the conditions that go along with hyperplasia are:[56,57]

- Obesity (Aromatase, an enzyme found in adipose tissue, converts circulating androgens to estrogen, leading to peripheral hyperestrogenism.)
- Perimenopause.
- Polycystic ovarian syndrome.
- Functional tumors (granulosa cell tumors of the ovary)
- Excessive ovarian cortical function
- Prolonged administration of estrogenic substances

Table 6: New WHO classification of endometrial hyperplasias.[58]

NEW TERMS	SYNONYMS	TOPOGRAPHY	FUNCTIONAL CATEGORY
Hyperplasia without atypia	• Simple endometrial hyperplasia without atypia. • Complex endometrial hyperplasia without atypia.	Diffuse	Estrogen effect
Atypical hyperplasia/ Endometroid intraepithelial neoplasia (EIN)	• Atypical Complex endometrial hyperplasia. • Atypical Simple endometrial hyperplasia (rare) • EIN	Focal to diffuse	Precancerous

Histological features of typical hyperplasia: [25,59]

Hyperplasia is characterized by an increased gland-to-stromal ratio and a variety of abnormal architectural patterns. Glands typically vary in size and shape.

In simple hyperplasia, glands are small to large and cystically dilated with occasional outpouching and focal crowding. The cells lining the glands are stratified and columnar with amphophilic cytoplasm. The stoma is abundant, cellular, and densely packed.

In complex hyperplasia, there is a marked crowding of glands with little intervening stroma. The glands may show epithelial stratification.

Histological features of atypical hyperplasia: [25,59]

The most important feature is the presence of nuclear atypia. Nuclear atypia is variable. The cells are stratified with a loss of polarity and an increase in nuclear to cytoplasmic ratio. The nuclei are enlarged and rounded with coarse chromatin, thickened irregular nuclear membrane, and prominent nucleoli.

In simple atypical hyperplasia, glands may show minimal complexity or maybe more irregular with intraglandular tufting. An abundant stroma is seen between the glands.

In complex atypical hyperplasia, the glands demonstrate marked structural complexity with irregular outlines and back-to-back crowding. Papillary infoldings may also be seen

Management of endometrial hyperplasia:

The risk of progression from hyperplasia without atypia to malignancy is very low, and if the hormonal milieu is corrected, spontaneous resolution occurs. Progesterone therapy is used either locally by intrauterine route or orally[59].

Atypical hyperplasia has a high risk of progression. So, considering the risk, a total hysterectomy is highly suggestive[59]. Only in exceptional cases, conservative treatment with high-dose gestagens and thorough histological monitoring be considered[58].

CARCINOMA OF ENDOMETRIUM:

Endometrial carcinoma is the most common invasive cancer of the female genital tract. They are broadly categorized into type-I and type-II, based on clinicopathological and molecular genetic features[60].

ETIOLOGY:

Hormonal stimulation is the main risk factor. Numerous studies have indicated a strong association between the development of endometrial carcinoma and estrogen replacement therapy[25].

Obesity is a definitive risk factor for endometrial carcinoma, early age at menarche, late menopause, nulliparity, and diabetes have also been associated with increased risk. There is a conflict regarding the data on the risk of developing endometrial carcinoma in patients treated with tamoxifen. In reproductive-age women, it has an antiestrogenic effect, but in postmenopausal women, it has a weak estrogenic effect[25].

Gonadal dysgenesis (Turners syndrome) can also be associated with a well-differentiated type of endometrial adenocarcinoma.

Table 7: Pathogenetic forms of endometrial carcinoma

Characteristics	**Type-I**	**Type-II**
Risk factors	Unopposed estrogen Obesity diabetes	Atrophy Thin Physique
Precursor lesions	Endometrial hyperplasia	Serous endometrial intraepithelial carcinoma
Histologic types	**Endometrioid**	**Serous** **Clear cell** **Carcinosarcoma** (mixed Mullerian tumor)
Tumor grade	low	High
Myometrial invasion	Variable, often minimal	Variable, often deep
Genetic abnormalities	PTEN ARID I A (regulator of chromatin) PI3K KRAS FGF2 CTNNB I	TP53 Aneuploidy PI3K
Behavior	Indolent Spreads via lymphatics	Aggressive Intraperitoneal and lymphatic spread

Endometrioid endometrial carcinoma:

It is the most common type of endometrial carcinoma. It constitutes approximately 80% of all endometrial carcinomas, most of which are low grade (grade 1 - 2)[61]. As these tumors resemble proliferative phase endometrium, they are referred to as endometrioid. Most women are postmenopausal at the age of 55-65 years. Abnormal vaginal bleeding is the most common manifestation[25].

Grossly endometrial surfaces have a varied appearance; they may present as fungating masses with focal hemorrhage. Occasionally, there is no appreciable mass.

Generally, they spread by myometrial invasion, followed by direct extension into the adjacent structure. Distant metastatic spread to the lungs, liver, and bones occurs in the late stage[62].

Grades of Assessment of the Glandular Component [63]

Grade-1 (well differentiated) – Entirely composed of well-formed glands and constituting less than 5% of solid growth.

Grade-2 (moderately differentiated) – Consists of both, well-formed glands and a solid growth pattern up to 50% or less of the tumor.

Grade-3 (poorly differentiated) –more than 50% of solid growth pattern.

Nuclear features consist of variations in size and shape, chromatin distribution, and the presence or absence of nucleoli. In the presence of atypia, the tumor is assessed one grade higher than the histologic grade[25].

Serous endometrial carcinoma:

Serous tumors are type-II tumors and account for 5-10% of endometrial carcinomas and are always considered as poorly differentiated (grade-2) tumors. They are generally seen in women of a much older age than those with endometrioid[25].

Uteri with these tumors appear small and atrophic with exophytic growth and have a papillary appearance[62]. On microscopy, the papillary pattern predominates, having short and thick papillae with frequent hobnail cells. Tumor cells are polygonal with abundant eosinophilic to clear cytoplasm with marked nuclear atypia. Mitotic activity is high, with frequent abnormal mitotic figures. Psammoma bodies are also seen. In most cases, the adjacent endometrium is atrophic[62].

Serous carcinoma has a high tendency for myometrial and lymphatic invasion. The peritoneal surface is also involved at an earlier stage.

Clear cell carcinoma:

It accounts for about 1-6% of endometrial carcinomas. These tumors may exhibit solid, trabecular, cystic, and papillary patterns with large tumor cells having clear to pale eosinophilic cytoplasm with occasional Psammoma, hyaline and targetoid bodies[64]. Cytoplasm is clear due to the presence of glycogen, which is PAS-positive and diastase resistant[62].

Carcinosarcoma / Malignant mixed Mullerian tumor (MMMT):

These are generally considered poorly differentiated endometrial carcinomas, and they constitute less than 5% of malignant neoplasms. Histologically, these tumors are biphasic, and consist of malignant epithelial components (high-grade endometrioid, serous, and admixture) and mesenchymal (sarcomatous) components. Sarcomatous elements are often pleomorphic and contain heterologous elements, most commonly rhabdomyosarcoma and chondrosarcoma[65].

Carcinomas should be carefully staged as the prognosis depends on them[66].

Table 8: FIGO Staging of endometrial carcinoma (2018) [66]

FIGO(2018)	Description
I	Tumor confined to the uterus
1A	Tumor confined to the endometrium or <50% invasion of myometrium wall
1B	>50% invasion into the myometrial wall
II	Tumor infiltrates cervical stroma
III	Tumor extends beyond the uterus
IIIA	Extends into serosa or adnexa
IIIB	Extends into the vagina or parametrium
IIIC	Metastasis into the pelvic lymph nodes
IIIC1	Metastasis into pelvic lymph nodes
IIIC2	Metastasis into paraaortic lymph nodes
IV	Tumor invades bladder or bowel mucosa or distant organs
IVA	Tumor invades bladder mucosa or bowel mucosa
IVB	Distant metastases

Hysterectomy and endometrial carcinoma

Endometrial carcinoma is treated by total abdominal hysterectomy with bilateral salpingo-oophorectomy; sometime accompanied by removal of pelvic and para aortic lymph nodes. Tumor sterilization by radiation as determined by histopathological study of biopsy specimens, was often obtained for tumors limited to the endometrium but only rarely for tumors invading the myometrium[62].

PELVIC INFLAMMATORY DISEASE

Hysterectomy with or without adnexal removal should not be done unless a patient has not responded to broad-spectrum intravenous antibiotic therapy[67].

ENDOMETRIOSIS

Although a common etiology for infertility, endometriosis is an uncommon reason for the hysterectomy. Patients who require hysterectomy do so because of pelvic pain and dysmenorrhoea

Conservative surgical therapy using laparoscopic ablation or resection may reduce the inflammation and thus cause relief of pain. Other methods are danazol therapy, oral contraception, and GnRH agonists but most of them are not very helpful. Hysterectomy may be the only complete treatment for endometriosis[68].

Hysterectomy combined with oophorectomy or ovarian preservation

The ovaries are intricate endocrine organs, that produce both estrogens and androgens and are involved in many metabolic processes, including the metabolism of bones and lipids. Around menopause, hormonal levels decline. After menopause, the ovaries continue to produce a relatively small amount of testosterone and

androstenedione, which are then converted to estrogen in adipose, muscle, and other peripheral tissues. The removal of ovaries in premenopausal women may have a clinically significant effects due to continued ovarian function following menopause.

The American College of Obstetricians and Gynecologists (ACOG), which currently advises on retaining normal ovaries in premenopausal women without an increased genetic risk of ovarian cancer, should be strongly considered. However, postmenopausal women with increased risk should consider having an oophorectomy[69,70]. There is always an argument between ovarian conservation and elective oophorectomy[69].

Follicular cyst (FCs) and Corpus luteal cyst (CLCs):

Follicular cysts and corpus luteal cysts are non-neoplastic lesions and are considered functional or physiological cysts. The patients are often asymptomatic and disappear spontaneously, so the findings were incidental[71].

FCs are common in women of reproductive age, but rare in post-menopausal women. On microscopy, cysts are lined by inner granulosa cells and outer theca internal cells. These 2 layers are differentiated by reticulin stain as thecal cells are surrounded by dense reticulum[72].

Solitary or multiple FCs may be associated with McCune-Albright syndrome. [72] Follicular cysts develop when a mature ovarian follicle fails to release oocyst during ovulation. In the follicular phase, the cyst may form when there is no LH surge or elevated FSH[73].

Corpus luteal cysts (CLCs):

The Corpus luteum is a post-ovulatory physiological structure. After the release of the ovum, the collapsed ovulatory follicle becomes the corpus luteum. If there is no fertilization, it usually regresses by the end of the cycle. It continues to secrete progesterone if the egg is fertilized and shrinks around the 10^{th} week. If the corpus luteum fails to regress, it may develop into a corpus luteal cyst[73].

CLCs are unilocular with a smooth surface. On the cut section, they have a characteristic convoluted yellow lining[74].

On microscopy, the convoluted lining consists of inner luteinized granulosa cells and outer luteinized theca interna cells, with a prominent inner layer of connective tissue. If CLCs are associated with pregnancy then hyaline bodies and calcific foci are seen within the granulosa cells

The common complications of the ovarian cyst are torsion, hemorrhage, and rupture[74].

Management:

It mostly depends on the age, menopausal status, size of the cyst, and presence of complications. Women of reproductive age, to preserve fertility either oophorectomy or salpingo-oophorectomy is commonly preferred. For postmenopausal women hysterectomy with bilateral salpingo-oophorectomy is done[73].

Tumors of the Ovary

Ovarian cancer is the third leading contributor to cancer mortality in women. The surface (coelomic) epithelium, totipotent germ cells, and sex cord-stromal cells are the three cell types that make up a normal ovary, hence the lesions of the ovary are extremely diverse[75].

The vast majority of ovarian tumors, approximately 90% of them are malignant epithelial tumors. Germ cell and sex-cord stromal cell tumors constitute 20% to 30% of ovarian tumors but are significantly less common.

WHO classification of ovarian tumors [76]

Epithelial tumors:

- Serous tumors
  - -serous cystadenoma,
  - -serous borderline tumors
  - -serous carcinoma- low grade
  - -serous carcinoma-high grade
- Mucinous tumors
  - -mucinous cystadenoma and adenofibroma
  - -mucinous borderline tumors
  - -mucinous carcinoma
- Endometrioid tumors
- Clear cell tumors
- Sero mucinous tumors
- Brenner tumors
- Other carcinomas

Serous tumors:

Serous neoplasms account for 20-50% of ovarian tumors and approximately 70% of them are benign, may occur at any age but are most common during the fifth decade. 5% to 10% are borderline malignancies. Carcinomas constitute 20-25% and are most common between the ages of 40 and 70 years[25].

Benign serous tumors:

It includes cystadenomas, adenofibromas, cyst adenofibroma and surface papilloma. Symptoms and signs are non- specific and most commonly include pelvic pain, discomfort, or any asymptomatic pelvic mass.

Grossly they vary greatly in size up to 30 cm, and the external surface is smooth and glistening with either unilocular or multilocular cysts that are filled with clear, watery serous fluid or thin mucoid material. Papillary excrescences are infrequently visible on the surface[77].

Microscopic features:

Cystadenomas are lined by a single layer of tall, columnar, ciliated cells or non-ciliated epithelium. There is a broad spectrum of epithelial proliferation, from a simple, single layer and blunt papillae to focal epithelial stratification. If < 10% of the total tumor volume shows epithelial proliferation that would be considered a benign variant, otherwise qualify as a serous borderline tumor[78]. Adenofibroma and cyst adenofibromas are comprised predominantly of fibrous stroma, with glands and cysts forming a minor component[77].

Serous carcinoma: (low grade serous carcinoma and high grade serous carcinoma): [79]

More recently, a two-tier grading system that classifies tumors based on clinicopathologic and molecular genetic studies into low-grade and high-grade categories, particularly for serous carcinoma has been developed.

Both LGSC and HGSC have independent type-I and type-II pathogenesis pathways.

Table 9: Pathogenesis and clinicopathological features of LGSC and HGSC [79]

	LGSC	HGSC
Precursor lesions	Serous cystadenoma/adenofibroma—atypical proliferative serous tumors (APST)-non-invasive micropapillary serous carcinoma (non-inv MPSC)—invasive MPSC	It has a tubal origin -Tubal intraepithelial carcinoma (TIC)
Pathogenesis known as	Type-I pathway	Type-II pathway
Genetic mutations	KRAS, BRAF (most common) ERBB-2	TP53 (seen in 80% of tumors) Germline mutation of BRCA gene (seen in hereditary carcinomas)
Development of tumors	Slow and stepwise	Rapid
Mean Age	45-57	55-67
Growth pattern	Micro-papillae and round nests of cells infiltrating the stroma, separated by clefts/ clear spaces.	Admixture of papillary, glandular, nested, and solid growth patterns with a predominance of any component having irregular slit-like spaces infiltrating into the destructive stroma.
Morphology of neoplastic cells	These cells have a high N:C ratio with a scant to moderate amount of cytoplasm. Nuclei-uniform, small and round to oval with small nucleoli.	Heterogenous cells with very high N:C ratio. Nuclei-hyperchromatic, enlarged with the irregular nuclear membrane and large nucleoli.
Necrosis	Absent	Frequent
Psammoma bodies	Numerous	Less frequent
Mitotic index	Low	High

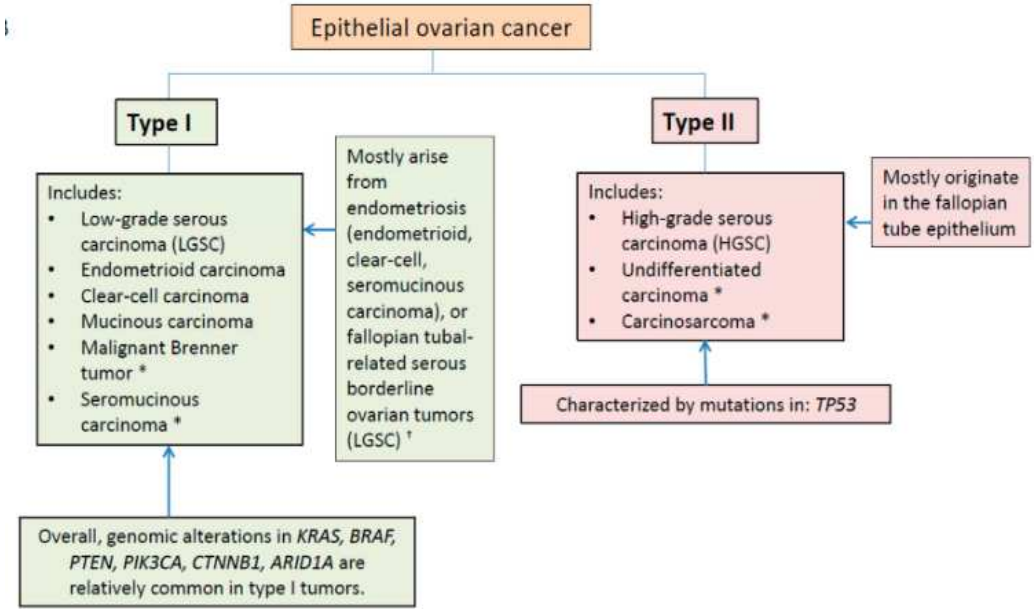


Figure 6: Molecular classification of epithelial ovarian tumors [80]

Mucinous tumors:

Mucinous ovarian tumors are characterized by the presence of epithelial lining, containing intracellular mucin, and are the largest of all ovarian tumors, sometimes they may exceed 30 cm and weighs up to 4000 g or more. They may present as a unilocular or multilocular cyst of varying sizes filled with dense, sticky, viscous, and gelatinous material[81].

Primary mucinous tumors are typically unilateral and are classified as benign, borderline, or malignant, depending on their histopathologic features, they may have endocervical or intestinal, or mixtures of both types of lining epithelium[82].

Benign mucinous neoplasms consist of cysts and glands, without any complexity of glands or cytologic atypia. Borderline tumors consist of cysts lined by stratified mucinous epithelium, they may form papillae and mitotic activity is seen, without any stromal invasion. Malignant tumors have a stromal invasion of more than

5mm and they may have an expansile type or infiltrative type of glandular growth pattern without any intervening normal ovarian parenchyma[81,83].

Mucinous tumors and hysterectomy

The standard protocol for any ovarian mass is the removal of the involved adnexa with intraoperative pathological examination. As the mucinous tumors are usually large, exploratory laparotomy with removal of involved adnexa is routinely performed. If the patient is post-menopausal then a hysterectomy with bilateral salpingo-oophorectomy is preferred[84].

Endometroid tumors:

These tumors are usually malignant, they account for up to 15% of ovarian cancer, are mainly seen in the 5th decade, are predominantly solid, and may contain numerous cysts and hemorrhagic areas[25]. These are characterized by the presence of confluent tubular glands lined by stratified, mucin-free epithelium with expansile stromal invasion[77].

Clear cell carcinoma

Clear-cell carcinomas account for 5% of all ovarian cancers, they are characterized by the presence of clear cells with eccentric nuclei, abundant clear cytoplasm, and definitive cell borders arranged in tubulocystic, papillary, and solid patterns. Hobnail cells are also seen with nuclei protruding into the lumen of tubules or glands. The presence of endometriosis and clear cell adenofibroma in the background strongly supports the diagnosis [(85,86)].

Brenner tumors:

The majority of these tumors are incidental findings, and they account for only 2-3% of all ovarian neoplasms. The majority of these tumors are benign and were seen between the fourth and eighth decades of life. Grossly benign tumors are well-circumscribed and usually measure less than 2 cm. On cut sections, they may have a hard or fibromatous, gray, white, or slightly yellow surface[87].

The typical benign Brenner tumor consists of epithelial cell nests separated by abundant dense fibromatous stroma. Borderline tumors show transitional cell nests with papilla projecting into the lumen. Malignant Brenner tumor consists of atypical transitional cells infiltrating into the stroma with high mitotic activity, and these cells are round to polygonal with pale cytoplasm and oval nuclei and they may have a central longitudinal groove[88].

Mixed epithelial tumors:

These tumors are defined by the presence of 2 or more distinct histological types on routine histopathological examination. Several types of mixed tumors are relatively common, including the following: combined Brenner and mucinous cystic tumor; endometrioid carcinoma admixed with clear-cell carcinoma; transitional cell carcinoma associated with another type of carcinoma; and endometrioid carcinoma with a serous or undifferentiated component[77].

In a study done by Mackenzie et. al, endometrial carcinomas and clear cell carcinomas combination are commonly seen. Based on morphological and molecular results there are less than 1% of mixed epithelial tumors[89].

Undifferentiated carcinoma:

The WHO defines an undifferentiated carcinoma as one that exhibits no or only rare and minor foci of differentiation. The presence of a differentiated component (usually low grade endometrioid adenocarcinoma) is necessary for the diagnosis, and these tumors account for less than 5% of carcinomas[90].

Microscopically, various patterns, including diffuse masses, irregular nests, and cords of epithelial cells separated by desmoplastic stroma, are encountered. The tumor cells vary from monomorphic to pleomorphic; tumor giant cells and necrosis are frequently present[91].

Sex cord stromal tumors: [76]

Sex cord tumors:

Adult granulosa cell tumors

Juvenile granulosa cell tumors

Sertoli cell tumors

Stromal tumors:

Fibroma

Thecoma

Leydig cell tumors

Fibrosarcoma

Sclerosing stromal tumors

Steroid cell tumors

Signet ring stromal tumors

Mixed sex-cord stromal tumors:

Sertoli-Leydig cell tumor

Granulosa cell tumors:

These tumors account for approximately 1.5% of all ovarian neoplasms[92]. Most of these tumors are estrogenic, rarely androgenic. When associated with hyperestrogenism, patients may have metrorrhagia, postmenopausal bleeding, endometrial hyperplasia, or carcinoma in adults. In children, it is associated with precocious puberty without endocrine symptoms[93].

The tumors in children and young adults differ microscopically from the usual tumors encountered in women older than 30 years of age, and they are designations as Juvenile granulosa cell tumors (JGCT) and Adult granulosa cell tumors (AGCT). 95% of granulosa cell tumors are adult type, and 5% are juvenile type[94].

Adult granulosa cell tumor (AGCT):

These tumors account for 95% of all granulosa cell tumors. Grossly, these tumors vary in size, and the appearance varies from uniformly solid to uniformly cystic and is unilateral in most cases. On the cut section, a yellow to white solid component is seen, depending on its lipid content[94].

Microscopically, several patterns are encountered, with two or more often present in the same specimen. The most common is the diffuse pattern, and others are the microfollicular pattern, characterized by small follicles (Call-Exner bodies) that may contain eosinophilic material with cellular debris, while the macrofollicular pattern often has large and irregular follicles lined by stratified granulosa cells. Insular and trabecular patterns are also commonly seen. The tumor cells are small and round, with ill-defined cell borders and pale cytoplasm. The nuclei are round to oval, or angular with a prominent nucleolus and may have longitudinal folds[94.]

Juvenile granulosa cell tumor (JGCT):

These tumors are typically encapsulated, have a mixed appearance, and are partly solid, containing cysts that may be filled with blood or uniformly solid.

They have a characteristic macrofollicular, solid, and cystic growth pattern. These macrofollicles are filled with mucinous secretions and lined by granulosa cells and thecal cells. These granulosa cells are large and polygonal, with a variable amount of pale to pink cytoplasm. The nuclei are large, round, and pleomorphic. Numerous mitotic figures are seen[94].

Fibroma:

Fibromas are benign solid tumors, and they account for 1-4 % of all ovarian tumors[95]. Meigs syndrome occurs in less than 1% of cases and is characterized by the triad of ovarian fibroma, ascites, and pleural effusion[96]. Fibromas have a smooth and lobulated external surface and are uniformly solid and firm with a white to the yellow cut surface. Hemorrhage, necrosis, calcification, and cystic degeneration may be seen[97].

On microscopy, spindle cells are seen arranged in whorls and anastomosing bundles with collagenous stroma. Neoplastic cells have uniform nuclei and pale, eosinophilic, scant cytoplasm. Occasional mitotic activity is seen[94].

Follow-up is very important, as these fibromas recur in young adults. (Gorlin syndrome) [98]

Thecoma:

Thecomas are almost always benign, and usually occurs in postmenopausal women with a mean age of 59 years. They are frequently associated with estrogenic manifestations, as evidenced by the presence of endometrial hyperplasia. Thecomas are usually solid yellow masses, but they can also be predominantly white with focal yellow areas[94].

On microscopy, these tumors consist of oval to round, large, ill-defined cells with moderate to abundant pale cytoplasm intersected by fibrous bands. sometimes the cytoplasm is highly vacuolated and contains lipids. These tumor cells may grow diffusely or in nodules of varying sizes, and the nuclei are round to oval and pale. Hyalinized plaques and microcalcifications are commonly seen[94].

Sertoli- Leydig cell tumor:

These ovarian tumors are composed of sex cord Sertoli cells and stromal Leydig cells, accounting for less than 0.5% of all ovarian neoplasms[99]. These tumors are most often seen in young women and are large, unilateral, and confined to the ovaries[100]. These tumors are characterized by the production of androgens; hence many patients show virilization symptoms like hirsutism, clitoromegaly, breast atrophy, and menstrual irregularity or amenorrhea depending on the quantity of the androgen production[94].

Sertoli- Leydig cell tumors are divided into five subtypes according to the World Health Organization classification: Well-differentiated, Moderately differentiated, Poorly differentiated, tumor with heterologous elements, and retiform variant.

Well differentiated tumors are solid and encapsulated. moderately and poorly differentiated tumors are quite large. These tumors are partly solid and partly cystic, with solid areas being firm[94].

Germ cell tumors of the ovary [76]

- Dysgerminoma
- Teratoma

  Mature teratoma

  Immature teratoma
- Monodermal teratoma

  Struma ovarii

  Carcinoid tumor
- Yolk sac tumor
- Embryonal carcinoma
- Choriocarcinoma
- Unclassified germ cell tumors

These neoplasms are derived from germ cells, and they constitute 15% to 20% of all ovarian tumors.

The Most common are benign cystic teratomas, found principally in children and young adults. Rarely, they may show malignant behavior[25].

Some of these tumors are composed of undifferentiated cells (Dysgerminoma), while others are differentiated towards embryonic (Teratoma) or extraembryonic (Choriocarcinoma, Yolk Sac Tumors) structures.

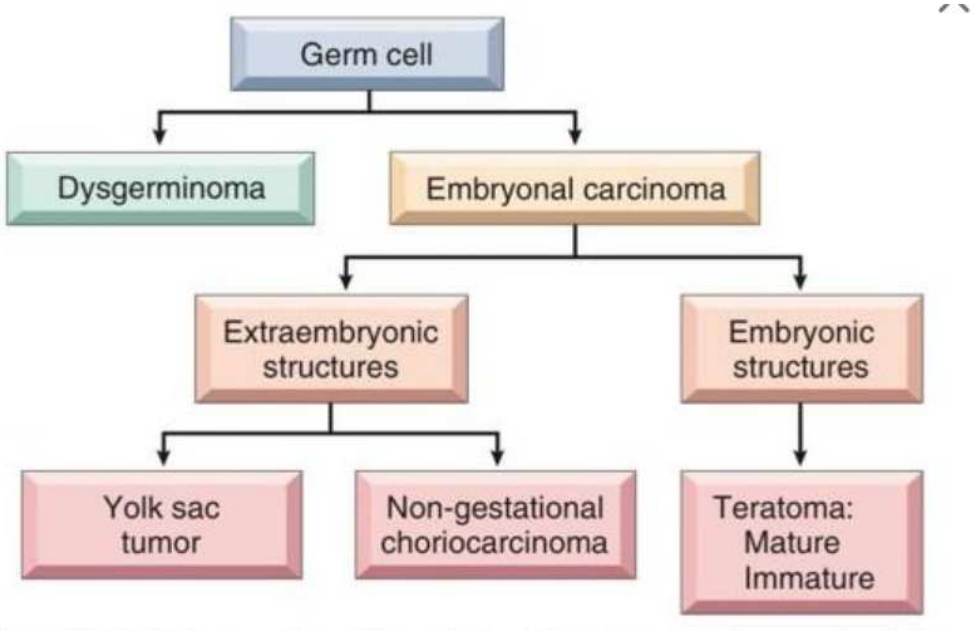


Figure 7: Germ cell origin of ovarian tumors.

Dysgerminoma:

Dysgerminoma is the ovarian counterpart of testicular seminoma. They account for about 2% of ovarian cancers and approximately 50% of malignant ovarian germ cell tumors[101].

These are most commonly seen in children and young women, with an average age of 22 years[102]. They typically present with abdominal distension, abdominal mass, or abdominal pain. 20% of malignant ovarian tumors detected during pregnancy are dysgerminomas[94].

Serum LDH levels are known to be elevated, and their levels correlate with tumor size and stage of the disease. 60% to 80% of tumors are confined to the ovaries and can be treated by salpingo-oophorectomy. Grossly, these tumors are large, usually, more than 10 cm in diameter, solid, and have a smooth outer surface. Fleshy,

homogeneous, and gray cut surface. Hemorrhage and necrosis are often present in large tumors[94].

On microscopy, tumor cells are arranged in nests, lobules, and trabeculae patterns, and are surrounded by fibrous septa that contain numerous lymphocytes, plasma cells, eosinophils, and sometimes histiocytes [94].

Teratoma:

Teratomas characteristically contain elements derived from all three embryonic germ layers and their most common site of occurrence is in the ovaries and testes[37]. They are found less frequently in the anterior mediastinum, sacrococcygeal region, retroperitoneum, abdominal wall, or neck[103]. Ovarian teratomas are broadly classified histologically into monodermal teratomas, immature teratomas, and mature cystic teratomas.

In monodermal teratomas, one of the elements greatly predominates. (struma ovarii when thyroid tissue constitutes >50% of the tumor) [104]

Mature teratoma:

These benign cystic ovarian tumors have mature tissue that represents at least two embryonic layers. They account for 20% of all ovarian tumors and are frequently diagnosed in women between the ages of 20 and 50. Most of the patients are asymptomatic, or they may present with a mass in the abdomen or with complications of torsion.

10% to 15% of cases are bilateral. Teratomas have an average diameter of 7–10 cm and are usually cystic. While the cyst's contents are liquid at body temperature, they solidify and become soft to the firm at room temperature. These cysts on the cut section can be unilocular or multilocular and may include sebaceous material, hair, teeth, cartilage, or bone. In the cyst wall, there is a solid protuberance known as the Rokitansky nodule [94,105].

On microscopy, a varied mixture of ectodermal, mesodermal, and endodermal elements is seen.

The most frequent ectodermal derivatives are skin, hair follicles, and sweat glands; when they predominate, the tumor is known as a dermoid cyst. Derivatives of endoderm and mesoderm are also frequently observed. Malignant transformation occurs in these cystic teratomas and is typically seen in postmenopausal women[94].

Immature teratoma:

These are rare tumors and they are primarily seen in pre-pubertal adolescents and young women with the mean age being 18 years. The components in these tumors resemble embryonic and immature fetal tissue[25].

Immature teratomas are predominantly solid, unilateral, and large with an average diameter of 18cm[94]. Microscopically, tissues derived from all three germ cell layers are present, and mostly a mixture of mature and immature elements is seen with a haphazard distribution. Immature neuroectodermal elements predominate, and they are easily recognizable. It can be arranged in either homer-wright rosettes with an anuclear fibrillary zone or pseduorosette or tubules. Immature glial tissue and primitive retina with melanin component are also seen[94].

Yolk sac tumors:

These tumors are also known as endodermal sinus tumors. These are uncommon and are derived from malignant germ cells that differentiate along the extraembryonic yolk sac lineage[50]. High levels of AFP are detected, and can be used to monitor therapy effectiveness and look for recurrences[106].

Grossly these tumors are large, with an average diameter of 16cm.The most common histological patterns of yolk sac tumors are the reticular and the endodermal sinus patterns[94].

Schiller- Duval bodies are diagnostic, and these are the papillary structures with a central vessel surrounded by tumor cells. The whole structure is held in a cystic space that is often lined with flattened tumor cells[107].

Paratubal cysts:

The paratubal cyst originates from the mesosalpinx between the ovary and the fallopian tube and may arise from the mesothelium, mesonephric, or paramesonephric (Mullerian) tissues. Most of the paratubal cysts are less than 10 cm, but they can be large. The majority of paratubal cysts occur in the reproductive age[108].

Grossly, they appear as a thin-walled unilocular cysts with a smooth capsule containing clear fluid. On microscopically, the cells lining the cyst are flattened and has a thin smooth muscle wall[108].

MATERIALS AND METHODS

This study was conducted in the Department of Pathology, Jawaharlal Nehru medical college. Patient identities were concealed and approval from the KAHER University's Institutional Research Ethical Committee was obtained prior to conducting this study.

Study design: An observational study.

Study period: January 2021-June 2022

Study population:

The hysterectomy specimens received in the Department of Pathology, Jawaharlal Nehru Medical College, Belagavi.

INCLUSION CRITERIA:

Hysterectomy specimens received in the Department of pathology for various clinical reasons, during the study period.

EXCLUSION CRITERIA:

- Obstetric hysterectomy.
- Paraffin blocks made from frozen specimen.

Sample size: A total of 249 Hysterectomy specimens were analyzed.

Sampling procedure:

A total of 249 hysterectomy specimens were received from the Department of obstetrics and gynecology. In retrospective cases, patient data were obtained from records, and formalin-fixed paraffin-embedded blocks were retrieved. In new cases, clinical data were obtained from the patient's records, and requisition forms received at the Department of pathology along with hysterectomy specimens. The specimens were cut open through both the lateral walls from the cervix to the uterine cornua and examined grossly for size and wall thickness. Oophorectomy and salpingectomy specimens were also examined grossly. The specimens were fixed in 10% buffered formalin and allowed to fix for 24 hours.

Sections for histological examination were taken from the endometrium, myometrium, cervix that includes ectocervix and endocervix from both the lips of the cervix, adnexal structures, and if required, additional sections from all areas of abnormalities were also taken.

The sections were then processed in an automated tissue processor where the tissues were dehydrated with ascending grades of alcohol, cleared in xylene, and embedded in paraffin. Using a rotary microtome (Leica RM2245), 2-3micron thick paraffin sections were cut and each section is floated on a tissue floatation bath maintained at 50-55 degrees Celsius. The sections were taken on a regular glass slide coated with Egg albumin as adhesive and were stained with Hematoxylin and Eosin. Later, slides were then studied under light microscopy for histopathological findings.

Statistical analysis: The data was entered into MS-excel and analyzed using Statistical Package for Social Sciences (SPSS) statistical software (version 20.0) Comparison was made using Fisher's Exact test.

Ethical consideration:

- Informed consent was taken from all the participants in the study
- The confidentiality and anonymity of the participants were assured and maintained.
- There was no benefit for participation in the study.
- This study has been approved by KAHER University's Institutional Research Ethical Committee.

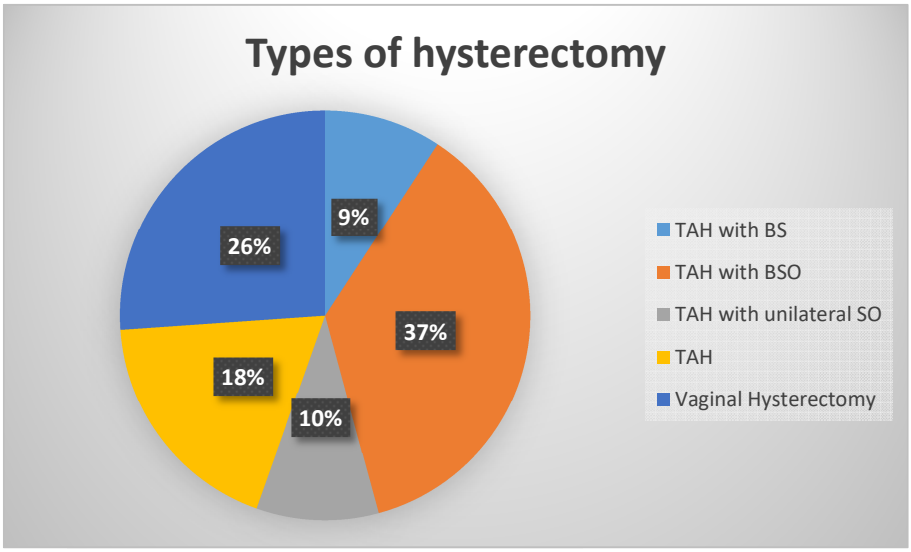
RESULTS

The present study was conducted at the Department of pathology, Jawaharlal Nehru medical college, KAHER, Belagavi. A total of 249 hysterectomy specimens were analyzed in the histopathology section of the department.

In our study, the most common type of hysterectomy was total abdominal hysterectomy with bilateral salpingo-oophorectomy with 91 cases (36.55%) and the next most common type was vaginal hysterectomy with 65 cases (26.10%) Least number of cases were of total abdominal hysterectomy with bilateral salpingectomy. (Table:10, Graph: 1)

Table 10: Types of hysterectomy

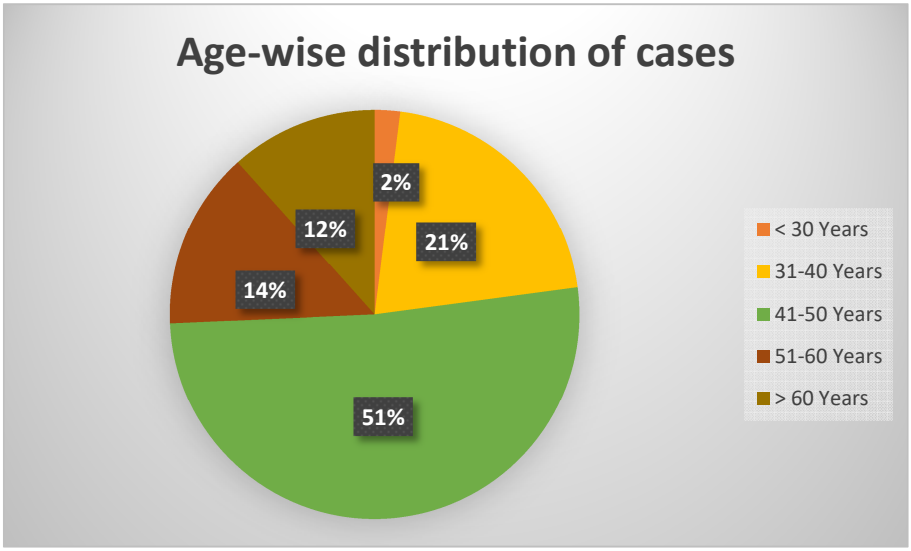
TOH	cases	Percentage (%)
TAH with BS	23	9.24
TAH with BSO	91	36.55
TAH with unilateral SO	24	9.64
TAH	46	18.47
Vaginal Hysterectomy	65	26.10
Grand Total	**249**	**100.0**



Graph 1: Types of hysterectomy

Table 11: Age-wise distribution of cases

Age Group	cases	Percentage (%)
< 30 Years	5	2.01%
31-40 Years	52	20.88%
41-50 Years	128	51.41%
51-60 Years	35	14.06%
> 60 Years	29	11.65%
Grand Total	**249**	**100.00**

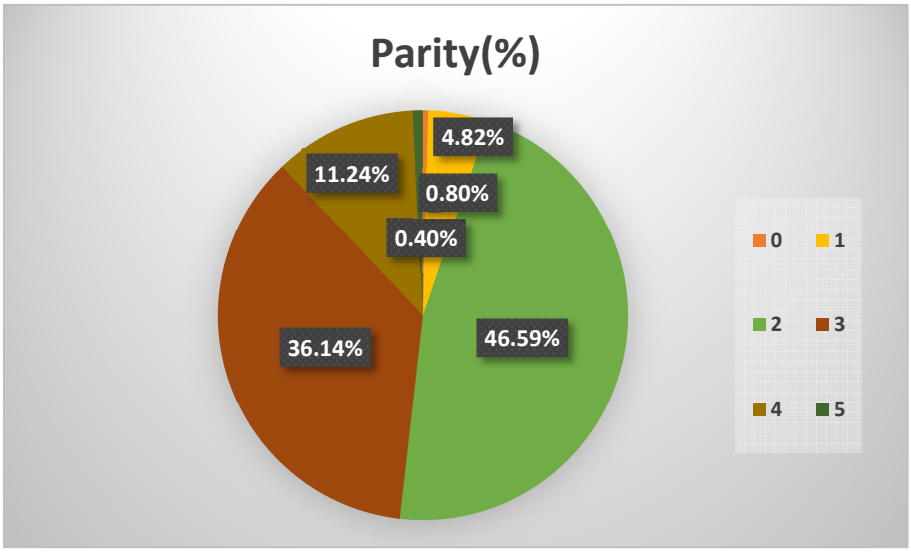


Graph 2: Age-wise distribution of cases

The patient's ages ranged from 26 to 76 years. 52% of cases belong to the 41-50 years of age group, 20.8% of cases are between the age of 31-40 years and the next 14.06% of cases were between 51-60 years of age. 5 cases were under the age of 30 years. (Table: 11, Graph: 2)

Table 12: Parity wise distribution of cases

Parity	Cases	Percentage (%)
0	1	0.04%
1	12	4.82%
2	116	46.59%
3	90	36.14%
4	28	11.24%
5	2	0.8%
Grand Total	**249**	**100.00**

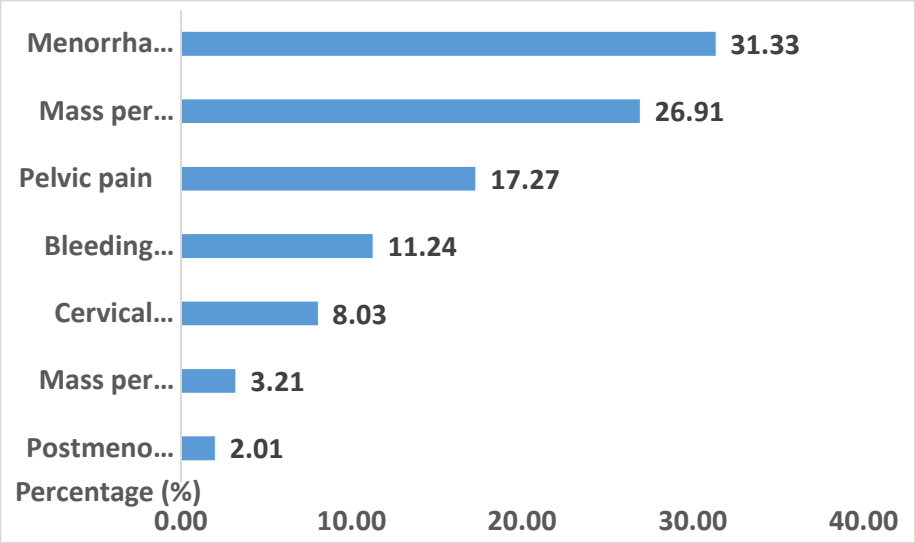


Graph 3: Parity wise distribution of cases

In the present study, the maximum number of cases, i.e. 46.5%, were of parity 2, and 36.1% of cases were of parity 3. The pattern of parity indicates the highest prevalence in para 2. (Table: 12, Graph: 3)

Table 13: Clinical presentation of cases

Chief Complaints (CC)	Count	Percentage (%)
Menorrhagia	78	31.33%
Bleeding per vagina	28	11.24%
Postmenopausal bleeding	5	2.01%
Mass per vagina	67	26.91%
Pelvic pain	43	17.27%
Cervical discharge	20	8.03%
Mass per Abdomen	8	3.21%
Grand Total	**249**	**100.00**

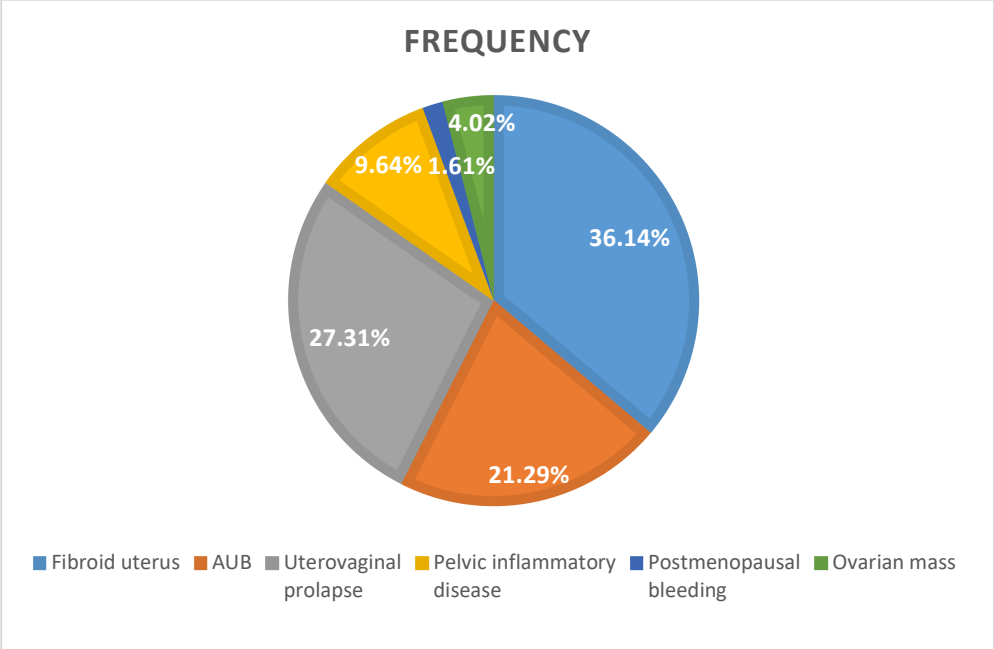


Graph 4: Clinical presentation of cases

In this present study, the most number of patients had presented with complaints of menorrhagia (31.33%) followed by mass per vagina (26.91%) and pelvic pain (17.27%). Other less common presentations were mass per abdomen and postmenopausal bleeding. (Table: 13, Graph: 4)

Table 14: Clinical indication for hysterectomies

Clinical indications	No. of cases	Percentage (%)
Fibroid uterus	90	36.14%
AUB	53	21.29%
Utero vaginal prolapse	68	27.31%
Pelvic inflammatory disease	24	9.64%
Malignancies of endometrium & cervix	4	1.61%
Ovarian mass	10	4.02%
Total	**249**	**100.00**



Graph 5: Clinical indications for hysterectomies

The most common indications for hysterectomy in this study were fibroid uterus in 90 cases (36.14%), followed by utero vaginal prolapse in 68 cases (27.31%), and abnormal uterine bleeding (AUB) in 53 cases (21.29%). 10 cases of ovarian masses and 4 cases of carcinoma of the endometrium and cervix were included in this study. (Table: 14, Graph: 5)

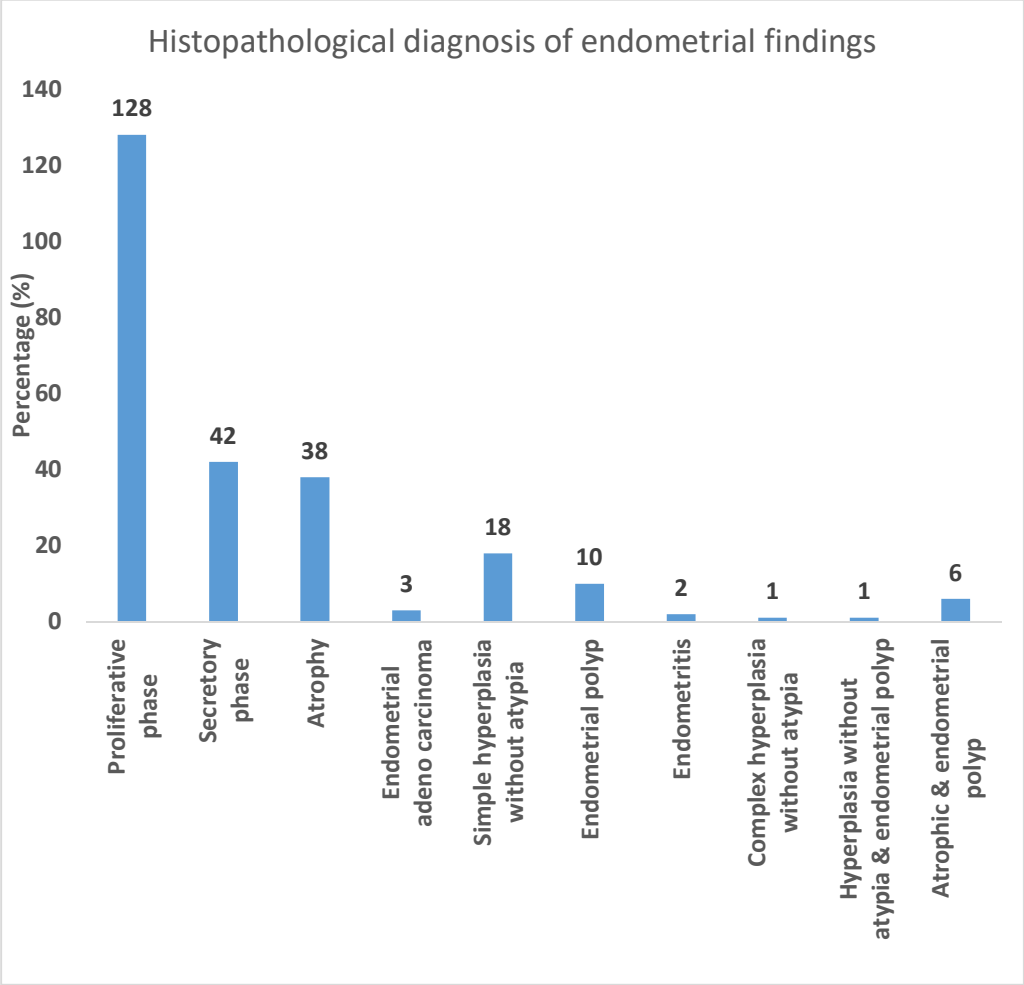
Table 15: Distribution of clinical indications of hysterectomy with age group.

Clinical indications	<30	31-40	41-50	51-60	>60
Fibroid uterus	1	19	60	9	1
AUB	0	12	35	6	0
Utero vaginal prolapse	1	7	21	14	25
Pelvic inflammatory disease	3	11	7	3	0
Malignancies of endometrium & cervix	0	0	0	2	2
Ovarian mass	0	3	5	1	1

Most of the patients who have undergone hysterectomy with clinical indication of fibroid uterus are between the age group of 41-50 years (60 cases). The majority of 35 cases of AUB are within the age group of 41-50 years. out of 68 cases of utero vaginal prolapse, 25 of them are seen in those above 60 years of age and 1 case was seen at the age of 27 years. All 4 cases of carcinoma of the endometrium and cervix are seen above 50 years of age. (Table: 15)

Table 16: Histopathological diagnosis of endometrial findings

Histopathological diagnosis	Frequency	Percentage (%)
Phase of endometrium		
Proliferative phase	128	51.41%
Secretory phase	42	16.87%
Atrophic endometrium	38	15.26%
Endometrial hyperplasia		
simple hyperplasia without atypia	18	7.23%
complex hyperplasia without atypia	1	0.4%
Endometrial polyp	10	4.02%
Endometritis	2	0.8%
Hyperplasia without atypia & endometrial polyp	1	0.4%
Malignant tumors		
Endometrial carcinoma	3	1.2%
Total	**249**	**100.00**

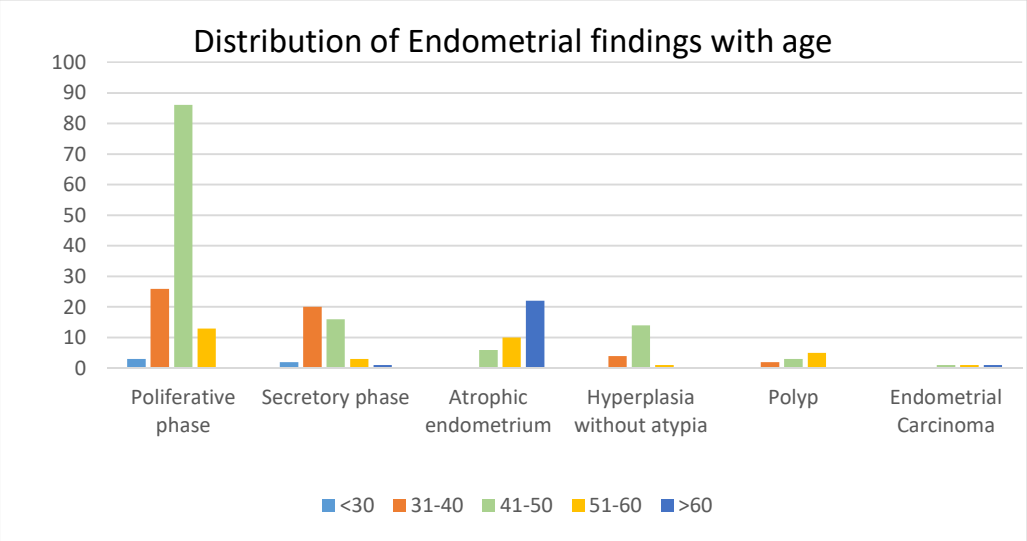


Graph 6: Histopathological diagnosis of endometrial findings

In the case of endometrial findings, the proliferative phase of the endometrium is the commonest finding in 128 cases (51.41%), followed by secretory phase endometrium in 42 cases (16.87%) and atrophic endometrium in 38 cases (15.26%). Hyperplasia without atypia is seen in 19 cases, among which simple endometrial hyperplasia without atypia is the finding in 18 cases (7.23%) and 1 case of complex hyperplasia without atypia is noted. An endometrial polyp was seen in 10 cases. 1 (0.4.%) case consists of both polyps and hyperplastic endometrial findings. Three cases (1.20%) of endometrial carcinoma were noted. (Table: 16, Graph: 6)

Table 17: Distribution of Endometrial findings with age

Age	<30	31-40	41-50	51-60	>60
Proliferative phase	3	26	86	13	0
Secretory phase	2	20	16	3	1
Atrophic endometrium	0	0	6	10	22
Hyperplasia without atypia	0	4	14	1	0
Polyp	0	2	3	5	0
Endometrial Carcinoma	0	0	1	1	1

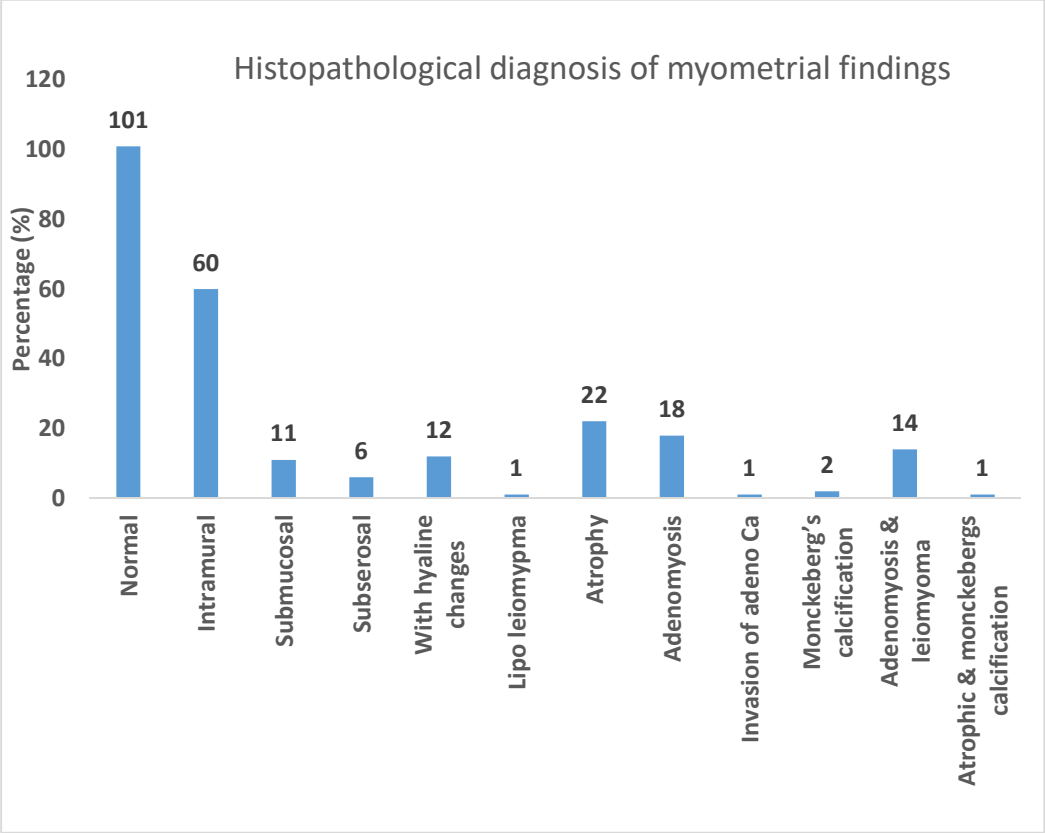


Graph 7: Distribution of Endometrial findings with age

The majority of the proliferative phase endometrial findings were seen in the age group of 41-50 years. The majority of the atrophic endometrium was found in women over the age of 60 years. Hyperplasia without atypia is commonly seen in the age group of 41-50 years. Endometrial polyps were seen between the ages of 51 and 60 years. Carcinoma of endometrium was noted in women above 40 years of age. (Table: 17, Graph: 7)

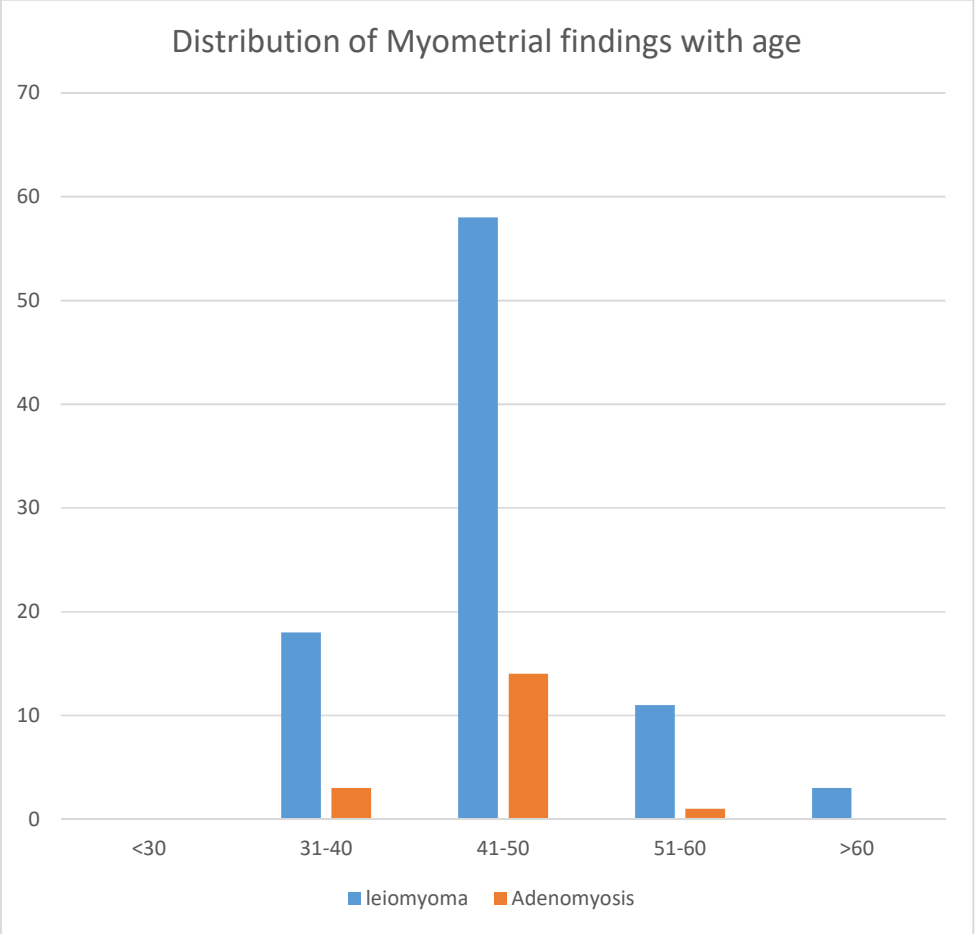
Table 18: Histopathological diagnosis of myometrial findings

Histopathological diagnosis	Frequency	Percentage (%)
Normal histology	100	40.16%
Leiomyoma		
Intramural	60	24.1%
Submucosal	11	4.42%
Sub serosal	6	2.41%
With hyaline changes	12	4.82%
Lipoleiomyoma	1	0.4%
Leiomyoma & adenomyosis	14	5.62%
Atrophy	22	8.84%
Adenomyosis	18	7.23%
Invasion of endometrial Carcinoma	**2**	0.8%
Monckeberg's calcification	2	0.8%
Atrophic & monckebergs calcification	1	0.4%
Total	**249**	**100.00**



Graph 8: Histopathological diagnosis of myometrial findings

In the case of myometrial findings, normal histology was noted in 100 cases (40.16%). Leiomyoma was seen in 90(36.12%) cases, out of which 60(24.10) cases were of intramural leiomyoma, 11 (4.42%) cases were of submucosal leiomyoma, 6 (2.41%) cases were of sub-serosal leiomyoma, 1 case of lipoleiomyoma and 12 (4.8%) cases of leiomyoma were presented with secondary hyaline changes. Adenomyosis was noted in 18 cases. Both leiomyoma and adenomyosis were seen in 14 (5.62%) cases. In 2 cases myometrium was invaded by endometrial adenocarcinoma. Monckebergs calcification was observed in 2 (0.8%) cases and in 1 case it is associated with atrophic changes. (Table: 18, Graph: 8)



Graph 9: Distribution of Myometrial findings with age

In the present study, the peak age prevalence of leiomyoma and adenomyosis was in the 5th decade at 41-50 years, followed by the 4th decade at 31-40 years. (Graph: 9)

Table 19: Histopathological diagnosis of cervical lesions

Histopathological diagnosis	Frequency	Percent
Chronic non- specific cervicitis	203	81.5
Squamous metaplasia	31	12.4
Endocervical polyp	7	2.8
LSIL	1	0.4
Cervical leiomyoma	3	1.2
Chronic papillary endocervicitis	3	1.2
Squamous cell carcinoma	1	0.4
Total	**249**	**100.0**

On histomorphological study of cervical lesions, chronic cervicitis was the commonest finding in 203 (81.5%) cases, followed by 31 (12.4%) cases of squamous metaplasia. Endocervical polyps were noted in 7 cases. 3 (1.2%) cases each of cervical leiomyoma and chronic papillary endo cervicitis were also noted. A Low-grade squamous intraepithelial lesion was seen in 1 case. There was 1 case of squamous cell carcinoma. (Keratinizing, moderately to poorly differentiated, extending up to the isthmus.) (Table: 19)

Table 20: Distribution of cervical findings with age

Age	<30	31-40	41-50	51-60	>60
chronic non-specific cervicitis	5	48	104	27	19
Squamous metaplasia	0	4	13	5	9
Endocervical polyp	0	0	4	2	1
LSIL	0	0	0	1	0
Cervical leiomyoma	0	0	3	0	0
Chronic papillary endocervicitis	0	0	3	0	0
Squamous cell carcinoma	0	0	1	0	

Chronic cervicitis is commonly seen in all age groups, but most of the cases (104) are seen between the ages of 41-50 years. The majority of the squamous metaplasia cases are seen in the age group of 41-50 years. All cases of cervical leiomyoma and chronic papillary endocervicitis are seen in the age group of 41-50 years. (Table: 20)

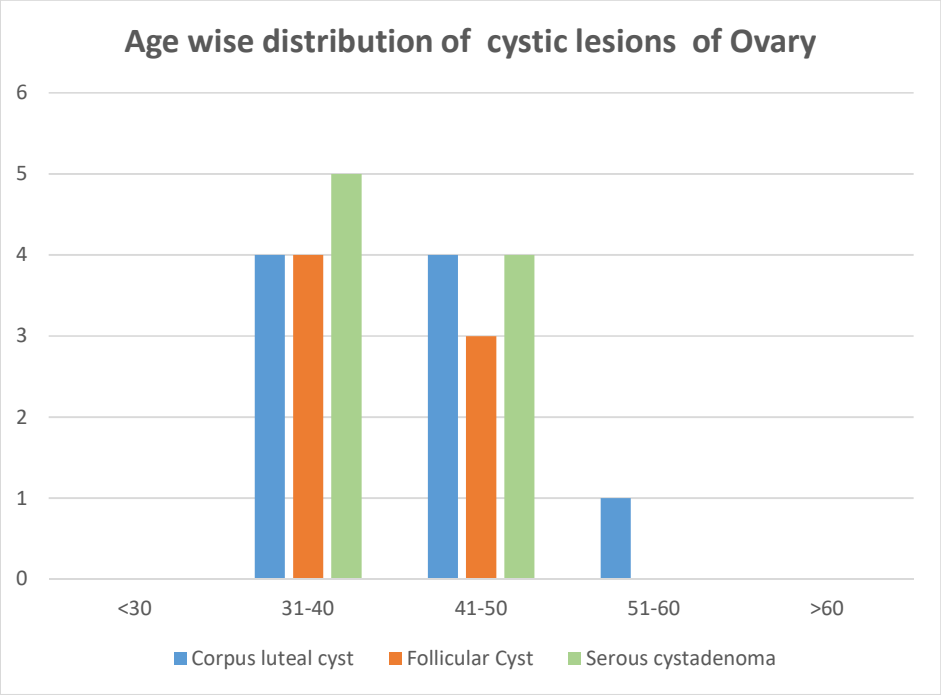
Table 21: Histopathological findings of ovaries

Histopathological diagnosis	**Right ovary findings**	**%**	Left ovary findings	**%**	**total**
Normal	79	31.7	90	36.1	169
Corpus luteal cyst	3	1.2	6	2.4	9
Follicular cyst	6	2.4	1	0.4	7
Serous cystadenoma	7	2.8	2	0.8	9
Benign cystic teratoma	1	0.4	1	0.4	2
Mucinous cystadenoma	1	0.4	1	0.4	2
Simple cyst	4	1.6	4	1.6	8
Serous Ca of ovary	1	0.4	0	0	1
Fibro thecoma	1	0.4	1	0.4	2
Total	103	41.4	106	42.6	**209**

In the present study, 209 oophorectomy specimens were histopathologically analyzed. The majority of the 169 oophorectomy specimens had normal histology.

The most common ovarian lesions were non-neoplastic. Right-sided oophorectomy specimens consist of 3 cases of corpus luteal cyst, 6 cases of follicular cyst, and 4 cases of simple cyst. While left-sided oophorectomy specimens have 6 cases of corpus luteal cyst, 1 case of follicular cyst, and 4 cases of simple cyst.

Ovarian tumors were observed in 4.41% of right-sided ovaries and 2% of left-sided ovaries. 7 cases (2.8%) of serous cystadenoma were seen in right-sided ovaries, and 2 cases (0.8%) were seen in left-sided ovaries. 1 case of benign cystic teratoma was seen on each side. 2 cases of mucinous cystadenoma and fibrothecoma were seen, one on each side. 1 case of serous carcinoma of the ovary was seen in the right-sided ovary. (Table: 21)



Graph 10: Distribution of cystic lesions of Ovary with age

In the present study, corpus luteal cysts were most common in women aged 31 to 50 years. All cases of follicular cyst and serous cystadenoma were seen between the age of 31 to 50. (Graph: 10)

Table 22: Histopathological findings of Fallopian tubes

	Right Fallopian tube	**%**	Left fallopian tube	**%**	**Total**
Normal	113	45.4	118	47.4	231
Para tubal cyst	9	3.6	8	3.2	17
Hematosalpinx	1	0.4	0	0	1
Foreign body type giant cell	1	o.4	1	0.4	2
Total	**124**	**100**	**128**	**100**	**252**

In the present study, 252 salpingectomy specimens were histopathologically analyzed. Most of the salpingectomy specimens (231) showed normal histology. 9 cases of paratubal cysts were seen in right-sided fallopian tubes and 8 cases in left-sided fallopian tubes. 2 cases of foreign body type giant cell lesions are seen. 1 case of hematosalpinx is seen. (Table: 22)

Evaluation of final histopathologic findings in relation to preoperative clinical diagnosis:

In the present study, 90 cases of preoperatively diagnosed fibroid uterus were confirmed on histopathological examination. So, there is a 100% clinico-pathological correlation.

The 53 cases diagnosed clinically as AUB, were pathologically proven as follows: 16 had simple endometrial hyperplasia without atypia, 1 case had complex hyperplasia, 9 cases had adenomyosis, 8 cases showed endometrial polyps, 7 cases had combined leiomyoma and adenomyosis and 3 cases showed cervical polyps. (Table: 23)

In 68 cases of prolapse, 38 cases showed atrophic endometrium. Adenomyosis and leiomyoma were found together in 5 cases; a cervical polyp was found in 4 cases; a cervical leiomyoma were found in 3 cases; and endometrial polyp and chronic papillary endocervicitis were found in 2 cases each. (Table: 23)

Among the 24 cases of pelvic inflammatory diseases, endometritis was seen in 2 cases, adenomyosis alone was seen in 7 cases, leiomyoma combined with adenomyosis was seen in 2 cases, simple hyperplasia was seen 2 cases, and 1 case of a polyp was seen on pathological examination. (Table: 23)

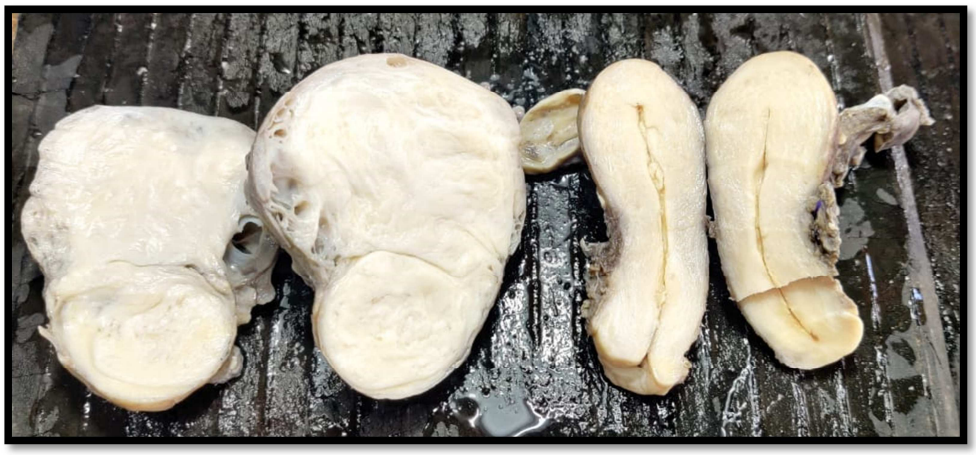
Out of 10 cases of ovarian mass, 1 case of serous carcinoma of ovary, 1 case of benign cystic teratoma and 2 cases of serous cystadenoma were diagnosed on pathological examination.

All three cases of endometrial carcinoma and one case of squamous cell carcinoma of the cervix that were indicated for hysterectomy were analyzed histopathologically.

Table 23: Distribution of AUB, UV prolapse, and PID lesions

Clinical diagnosis	Simple hyperplasia	Complex hyperplasia	Leiomyoma	Leiomyoma + Adenomyosis	Polyp (endo/cervical)	Adenomyosis	endocervicitis	Atrophic endometrium
AUB (53)	16	1	-	7	11	9	-	-
Uterovaginal prolapse (68)	-	-	3	5	6	-	2	38
Pelvic inflammatory disease (24)	2	-	-	2	1	7	-	-

Photomicrograph 4: Shows Intramural leiomyoma

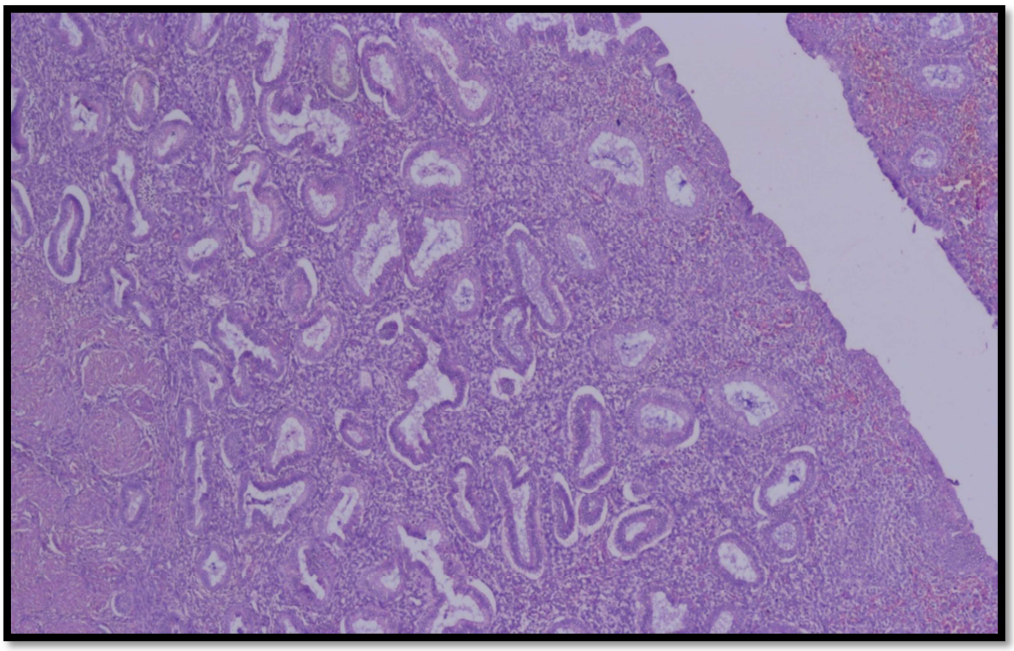


Photomicrograph 5: Shows separated sub-serosal leiomyoma

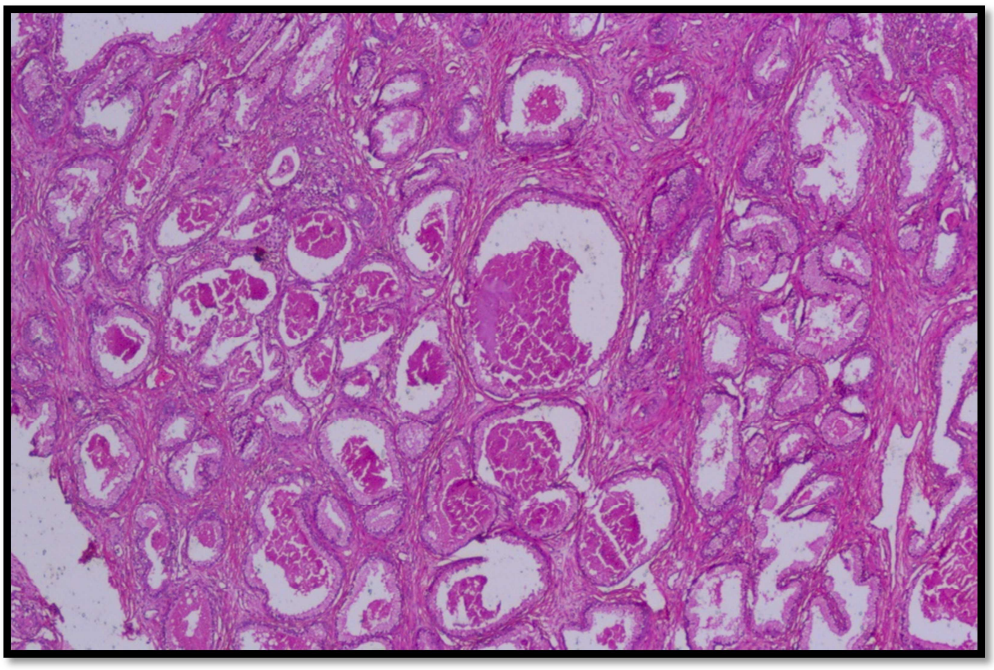
Photomicrograph 6: Shows Increased thickness of anterior and posterior wall of uterus

Photomicrograph 7: Shows Endometrial carcinoma

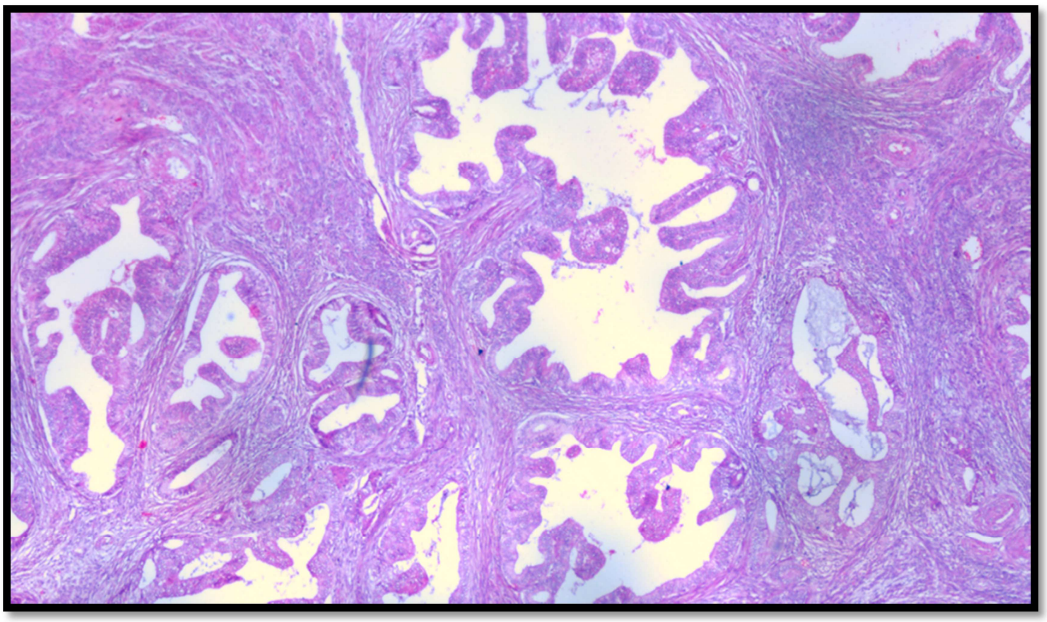
Photomicrograph 8: Shows Mucinous cystadenoma of ovary



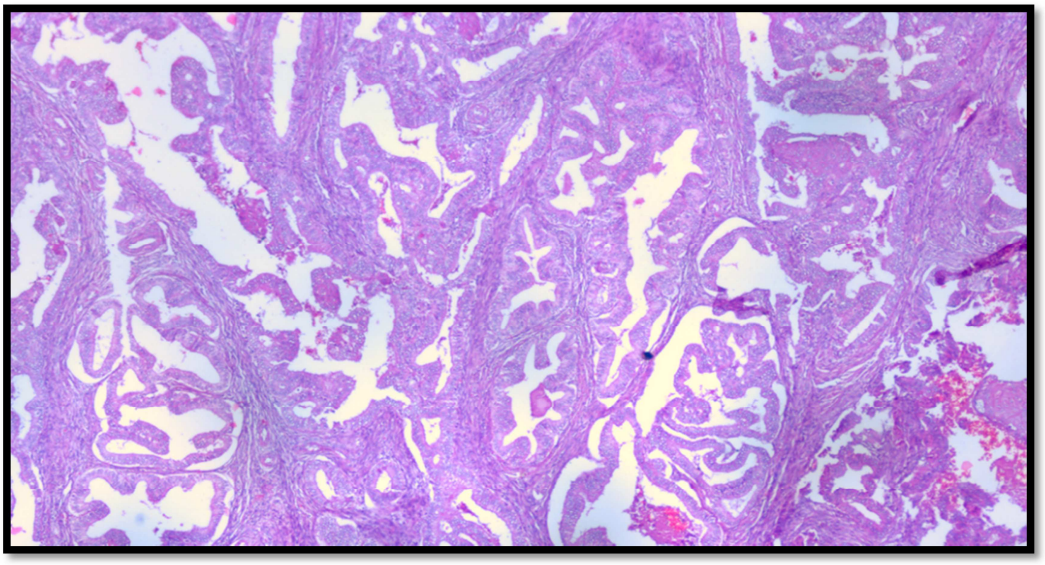
Photomicrograph 9: Shows secretory phase of endometrium (H&E,10x)



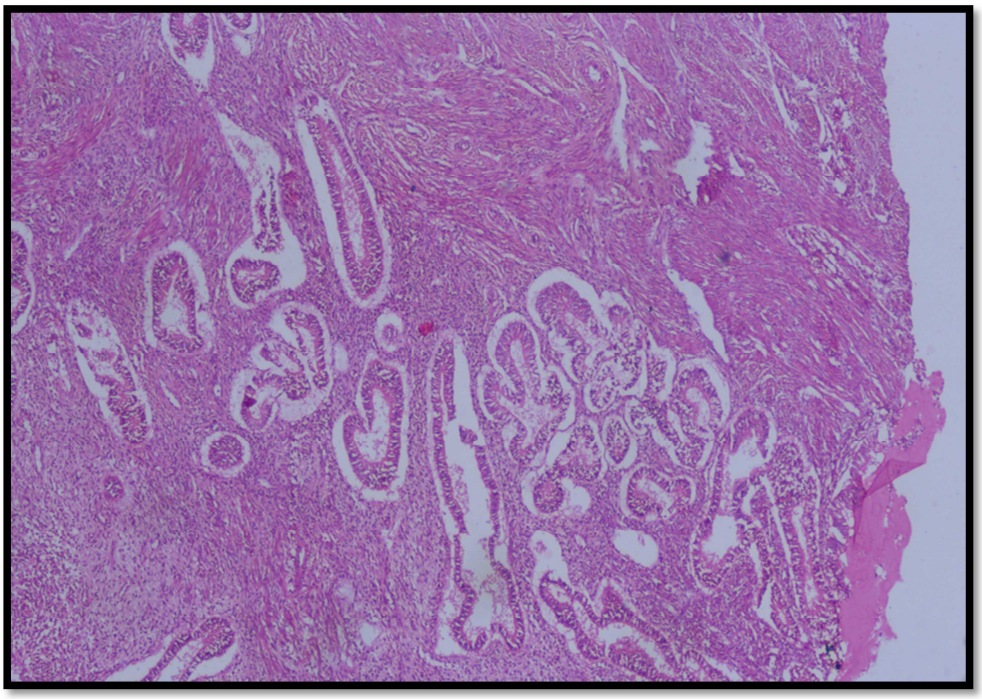
Photomicrograph 10: Shows simple endometrial Hyperplasia without atypia (H&E, 20x)



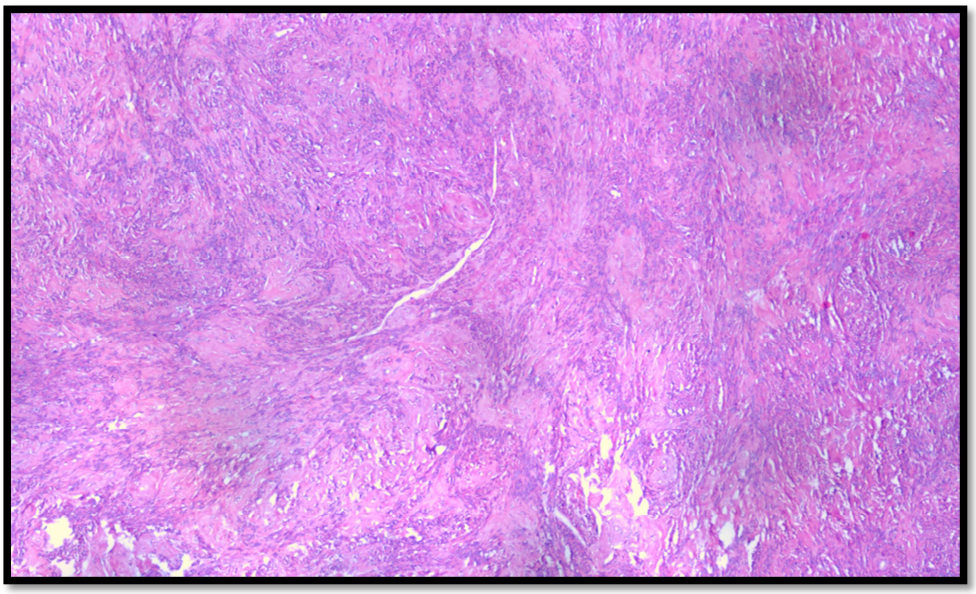
Photomicrograph 11: Shows cystically dilated, back-to-back arranged glands with papillary projections. Endometrial carcinoma-grade-1. (H&E, 20x)



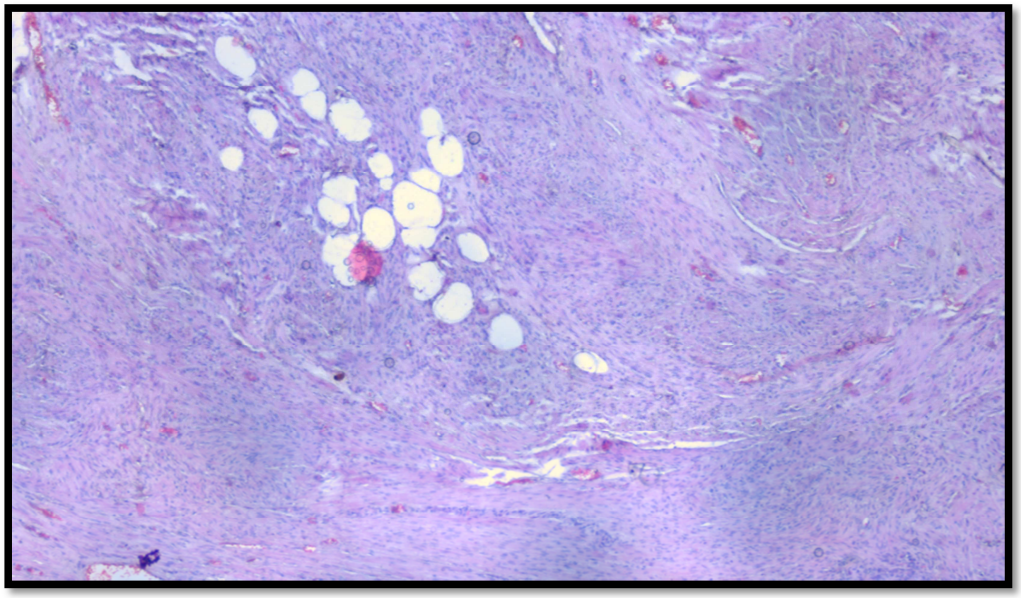
Photomicrograph 12: Shows cystically dilated, back-to-back arranged glands with papillary projections. Endometrial carcinoma-grade-1. (H&E, 20x)



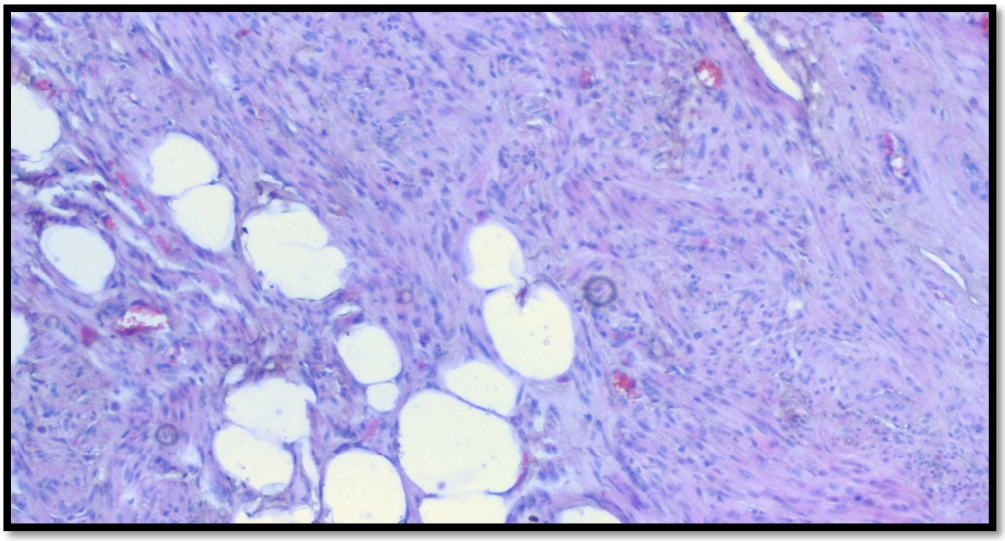
Photomicrograph 13: Shows presence of endometrial glands and stromal component within myometrium. Adenomyosis (H&E, 20x)



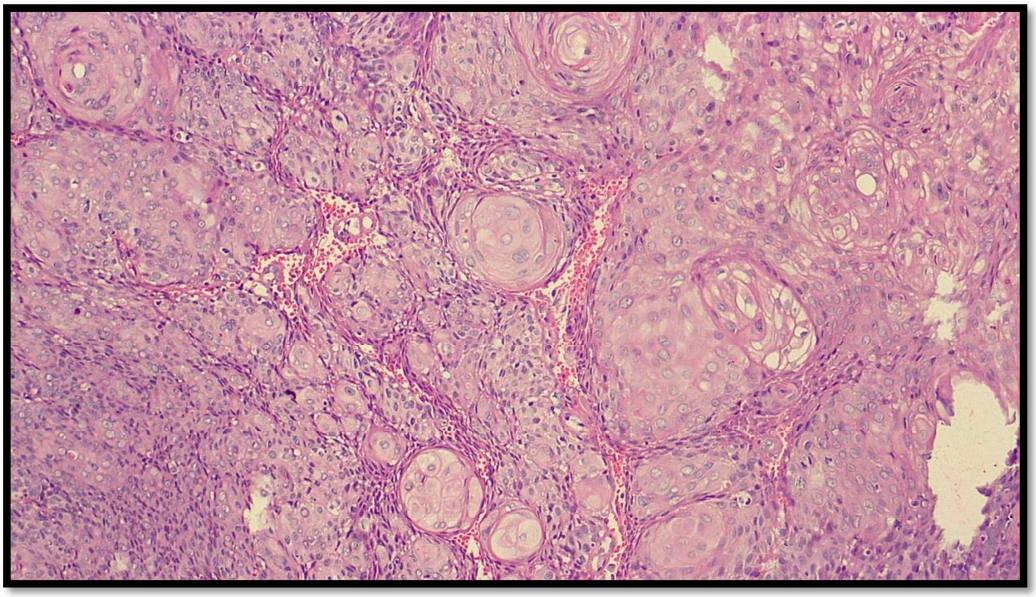
Photomicrograph 14: Shows hyaline changes within Leiomyoma (H&E, 20X)



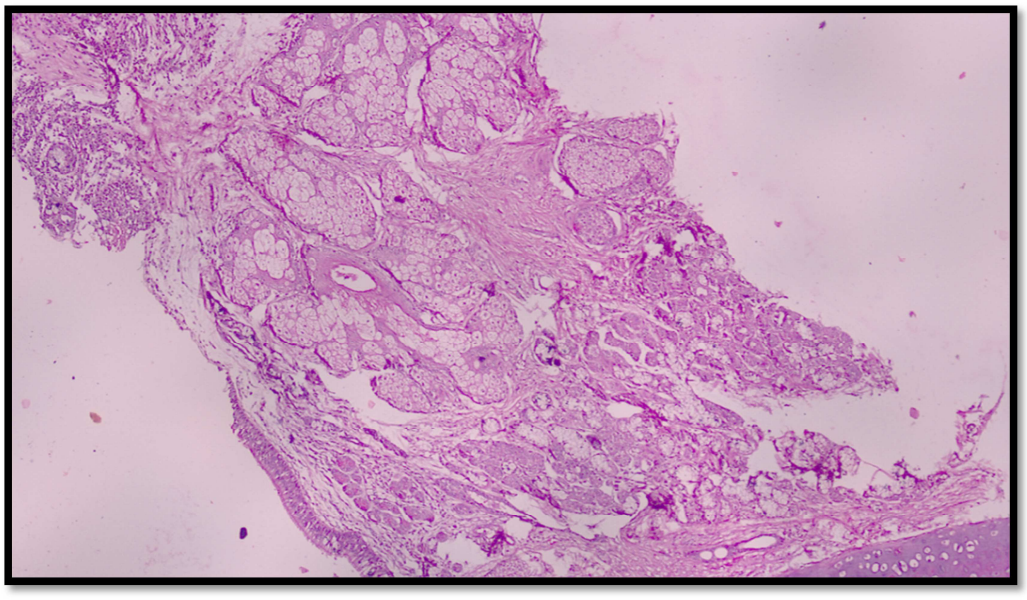
Photomicrograph 15: Shows presence of mature adipocytes with leiomyoma. Lipoleiomyoma. (H&E, 20x)



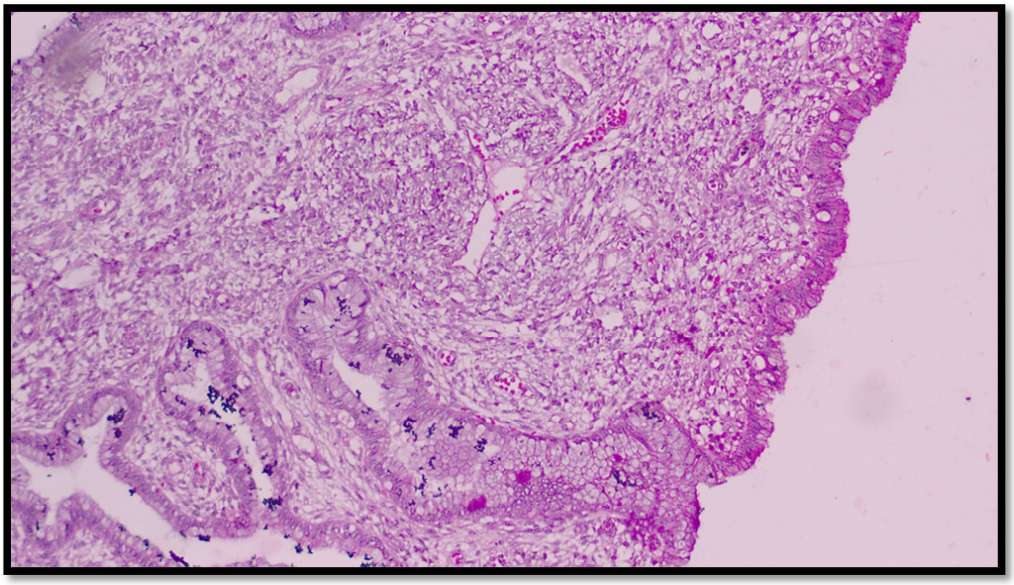
Photomicrograph 16: Shows presence of mature adipocytes with leiomyoma. Lipoleiomyoma. (H&E, 40x)



Photomicrograph 17: Shows well differentiated SCC of cervix (H&E,20x)



Photomicrograph 18: Shows Benign cystic teratoma. (H&E,10X)



Photomicrograph 19: Shows Mucinous cystadenoma of ovary (H&E,20x)

DISCUSSION

In the present study, out of 249 cases, 91 cases (36.5%) had undergone total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH with BSO). Jandial R et al[113] have reported TAH with BSO in 63% of cases, Ticku et al[1] have reported TAH with BSO in 46.5% of cases, and Shah et al[109] have reported TAH with BSO in 48% of cases. The variation in the percentage of types of hysterectomy may be due to the individual choice of the patient, the operating surgeon, and the bulk of the uterus. In most of the studies, the abdominal approach was the most common and preferred method of surgery.

In the present study, the maximum number of patients (51.1%) underwent hysterectomy, were in the age group of 40-50 years, which was in concordance with studies done by Ticku et al[1], Titiloye et al[112], and Shah et al[109].

In the present study, the maximum number of cases (46.5%) were of parity 2. The pattern of parity revealed that para 2 had the highest prevalence. But these findings are not similar to the study done by Ticku et al[1], where most of the cases are seen in parity 3.

In the present study, the most common (31.33%) clinical complaint was menorrhagia, similar to the study done by Ticku et al[1] (23.96%)

In the present study, the most common clinical indication included fibroid uterus in 90(36.1%) cases, followed by uterovaginal prolapse in 68(25%) cases. These findings are congruent with Patel et al[9] study, in which uterine fibroid (38.6%)

was found to be the commonest indication of hysterectomy, followed by uterovaginal prolapse (32.4%). Jandial et al[113] and Gupta et al[114] also showed similar findings.

Table 24: Comparison of the Endometrial findings in various studies

	Histologically unremarkable			Endometrial hyperplasia without atypia (%)	Endometrial polyp (%)	Endometritis (%)	Endometrial carcinoma (%)
	Proliferative phase (%)	Secretory Phase (%)	Atrophic (%)				
Present study	51.4	16.8	15.2	7.6	4	0.8	1.2
Ticku et al[1] (2017)	38.3	24.5	18.2	4.4	4.7	1.6	1
Geetika et al[5] (2020)	30.2	17.2	18.2	3	3	-	1
Jandial et al[113] (2019)	54.3	8.7	25	1.2	1.8	1.2	1.2
Baral R et al[7] (2017)	-	-	7.3	-	1.8	-	1.1
Ujwal R et al[6] (2019)	38.2	19.1	26.9	2.6	1.7	4.3	-
Ajaz et al[115] (2018)	39.4	9.5	10.5	6	4.2	1	1.5
Kolur et al[116] (2019)	39.7	19.2	12	-	1.2	-	1.2

Proliferative phase endometrium (51.4%) was the most prevalent endometrial finding in our study, with 67.2% of cases and is usually observed between the ages of 40 and 50. Most of the studies [1,6,111,113,116] found similar results, but in the Nair et al[8] study, secretory phase endometrium was the most common finding. In the present study, endometrial hyperplasia without atypia was reported in 19(7.6%) cases, with peak age presentations of between 41 and 50 years. This finding was comparable with the studies done by Ticku et al[1], Geetika et al[5], Ujwal et al[6], and Ajaz et al[115] who reported 4.4%, 3%, 2.6%, and 6% cases of endometrial hyperplasia, respectively.

Jandial et al[15] reported 1.2% of endometrial hyperplasia, which is quite lower than other studies.

The prevalence of endometrial polyps in the present study was 4%, with 50% of them seen in the 51-60 age group, similar to that of Ticku et al[1], Geetika et al[5], and Ajaz et al[116] with 4,7%, 3%, and 4% of cases, respectively.

In the current study, the prevalence of endometrial carcinoma was 1.2% (3 out of 249 cases), which was similar in most of the studies [(1,5,6,7,113,115,116)]. The peak age of prevalence of endometrial carcinoma in the present study was between 41 and 65 years, which is comparable with the study done by Nair et al[8].

Table 25: Comparison of Myometrial lesions in various studies

study	Leiomyoma (%)	Adenomyosis (%)	Leiomyoma + Adenomyosis (%)	Invasion of adenocarcinoma (%)
Present study	36.1	7.2	5.6	0.8
Ticku et al[1] (2017)	25.8	14.3	-	-
Patel et al[9] (2018)	42.2	8.3	3	-
Shah et al [109] (2020)	18	8	15	-
Jandial r et al[113] (2019)	59.3	23	13.1	-
Sharma et al[10] (2020)	33	7	4.9	-
kolur et al[116] (2019)	47.4	20.5	17.9	-
Ajaz et al[115] (2018)	31.4	11.9	9.2	-

Lesions in the myometrium were seen in 59.4% of the cases in our study. Leiomyoma was the most common histopathological lesion found in the myometrium (36.1%). In most of the studies [1,9,10,113,116,115], leiomyoma was the most common pathology noted. In our study, the peak age prevalence of leiomyoma was between 41 and 50 years, but these findings were not in concordance with those of Nair et al[8]

where the peak age prevalence was between 31 and 40 years. Adenomyosis (7.2%) is the second most common myometrial pathology in this study. A Comparable distribution was seen in the studies by Sharma et al[10]. (7%), Shah et al[109] (8%) and Patel et al[9] (8.3%). Adenomyosis is difficult to diagnose preoperatively because it has no specific symptoms and is usually diagnosed on histopathological examination after hysterectomy [6,9]. 5.6% of the cases in this study revealed the presence of both leiomyoma and adenomyosis. Similar results were seen in previous studies [9,10,115]. In 2 cases, the myometrium was invaded by endometrial carcinoma in our study.

Chronic cervicitis is the most common finding in the cervix and is diagnosed histopathologically[10,115]. In our study, 81.5% of cases were chronic cervicitis, which is comparable to that reported by Ticku et al[1]. (81.9%), Sharma et al[10] (71.8%), and Ajaz et al[115] (89.3%). The next common lesion is squamous metaplasia, seen in 12% of cases, and these findings are in concordance with the study done by Ticku et al[1] (13.9%). The prevalence of squamous cell carcinoma in the present study was 0.4%. This incidence observed is comparable to that reported by Baral R et al[7] (1%), Ajaz et al[115] (0.7%) and Ticku et al[1] (0.2%). In the current study, 2.8% and 1.2% of cases of cervical polyps and cervical leiomyomas were observed. Similarly, 2% and 3% of cases of cervical polyps and leiomyomas were reported by Shah et al[109].

In our study, ovarian tumors comprised 6.41% of lesions. A study done by Ticku et al[1] found 14.2% of ovarian tumors, Shah et al[109] found 8% of ovarian tumors, and Baral et al[7] found 12.2% of ovarian tumors.

In our study, non-neoplastic ovarian lesions were more common, and were in concordance with studies done by Sharma et al [10], Ajaz et al[115], and Kolur et al[116].

In our study, the majority of the salpingectomy specimens analyzed were of normal histology. This finding was concurrent with the study conducted by Ticku et al [1] and Ujwal R et al[6].

In the present study, 100% of the pre-operative diagnoses of leiomyoma, were confirmed on histopathology, and the same was reported by Gupta G et al[114] and Khunte et al[117]. In the Saima. P et al[110] study, 72% of the preoperative diagnosis were confirmed on histopathology like uterovaginal prolapse, Pelvic inflammatory disease, uterine polyps, malignant ovarian tumor

CONCLUSION

In the present study, fibroid uterus and uterovaginal prolapse were the common indications for hysterectomy.

The most common findings identified on histopathological examination were leiomyoma and adenomyosis, chronic cervicitis, squamous metaplasia of the cervix, and functional cysts in the ovaries. In leiomyoma cases, histopathological analysis correlates well with pre-operative diagnosis.

The present study gives an insight into the various histopathological patterns of lesions in hysterectomy specimens in our institution. All hysterectomy specimens should be subjected to histopathological examination for accurate diagnosis, proper categorization of lesions, and a better clinical outcome and management.

SUMMARY

- In the current study, a total of 249 hysterectomy specimens were analyzed in the histopathological section at the JNMC, Belagavi, Department of pathology over the period of 18 months, from January 2021 to June 2022.
- In the present study, patient's demographics, clinical indications, type of hysterectomy, and histopathological findings were studied.
- Paraffin-embedded tissue blocks were retrieved for all retrospective cases, and for new cases, tissue sections from representative areas were taken, processed, and embedded in paraffin. Slides were prepared from paraffin sections and then stained with H&E and examined.
- The most common type of hysterectomy in the present study was total abdominal hysterectomy with bilateral salpingo-oophorectomy with 91 (36.55%) cases.
- The common incidence of hysterectomy was between 41 to 50 years of age. (51.41%).
- The majority of the women were of parity 2 (46.59%).
- Menorrhagia (31.33%) and mass per vagina (26.91%) were common clinical complaints.
- Fibroid (Leiomyoma) uterus was the most frequent preoperative clinical indication, accounting for 90 cases. (36.14%)
- Leiomyoma (36.12%), followed by adenomyosis (7.23%). were the two most common histological findings. And these were frequently observed within the age group of 41-50 years.

- Three cases of Carcinoma of endometrium were diagnosed histopathologically, myometrial invasion was seen in two of those cases
- Histopathological findings of endometrial hyperplasia without atypia were observed in 18 cases, and in 10 cases endometrial polyps were diagnosed.
- Chronic cervicitis was the most common (81.3%) histopathological finding in the cervix, seen in all age groups.
- Squamous metaplasia was the second most common (12.3%) finding in the cervix
- The commonest pathology in bilateral ovaries is corpus luteal cyst and serous cyst adenoma and the next common lesion is simple cyst.
- The majority (92.8%) of the salpingectomy specimens had normal histology.
- Paratubal cysts were the common pathology (6.8%) seen in fallopian tubes.
- In the present study, the majority of pre-operative diagnoses for leiomyoma were confirmed on histopathological examination.
- Histopathology is mandatory for confirming the diagnosis and thus ensuring optimal management.

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ANNEXURE I

INFORMED CONSENT FORM

HISTOPATHOLOGICAL ANALYSIS OF HYSTERECTOMY SPECIMENS: AN OBSERVATIONAL STUDY.

Purpose of the study: You are being asked to enroll in this study as you are eligible for participation in this study. If you undergo Hysterectomy other than Obstetric causes, you will be included in this study. The purpose is to study the histopathological lesions of hysterectomy specimens and to correlate these findings with clinical diagnosis and with age group. This study will help to know the incidence and pattern of non-neoplastic and neoplastic lesions of female genital tract vary according to different age groups.

Procedure: During this study, you will be asked questions regarding age and menstrual history and you are supposed to answer to the best of your knowledge. The principal investigator of the study is _____ under the guidance of _____. If you agree to enroll yourself in this study, you will be interviewed regarding your present, past and family history and your clinical manifestations.

Risks and benefits: There are no risks involved in taking part in this study and benefit is we will be able to judge the nature and prognosis of the disease which is essential for providing appropriate treatment.

Alternatives: Taking part in this study is voluntary. You may choose not to take part in this study or if you decide to take part now, you can later change your mind and

withdraw from the study. The study doctor or sponsor may terminate your participation in this study anytime.

Privacy and confidentiality: All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study will be published but your identity will be confidential in any publication. No information about you or information provided by you during research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.

2. If required by law.

Financial incentives for participation: You will not be paid / offered any gift /incentives for participating in this study.

Authorization to publish results: The results of this study would be forwarded to the KLE University, Belagavi as a part of requirement towards the completion of MD degree, review and publishing.

CONSENT STATEMENT

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form and have had all my questions answered.

In case of the queries during the study or in future you may contact following person.

Principal Investigator: _____

Guide : _____

If you have any queries about your rights as a study subject, you may call Dr. HARSHA HEGDE, IEC & scientist D, ICMR, National Institute of traditional medicine, Belagavi. Chairman of J.N. Medical College Institutional Ethical Committee of Human Subjects Research, at J.N. Medical College, Belagavi.

Name of the participant:

(signature/thumbprint)

Name of the witness : (signature)

Name of the investigator: (signature)

Date: Address & Phone no:

ANNEXURES II

PROFORMA

PATIENT HISTORY

Name:

Age:

IP no:

Parity index:

Chief complaints:

Brief clinical history:

Menstrual history:

Examination Findings Clinical Diagnosis:

Radiological reports:

Histopathological Diagnosis: (Hematoxylin and Eosin staining)

Endometrial findings:

Myometrial findings:

Cervical findings:

Bilateral ovarian findings:

Bilateral fallopian tube findings:

ANNEXURE III

HEMATOXYLIN AND EOSIN STAINING PROTOCOL.

- Deparaffinize in Xylene I and II and III changes. [III change use warmed xylene] (5 minutes in each)
- Rehydrate using:
    - Absolute Ethanol 100% (5 minutes)
    - Absolute Ethanol 100% (5 minutes)
- Rinse in distilled water (5 minutes)
- Rinse in running tap water (5 minutes)
- Stain in Harris's hematoxylin by progressive method (2 minutes) Fresh and filtered
- Rinse in running tap water (20 minutes)
- Decolorize in 1% acid alcohol (1 second)
- Rinse well in tap water (5 minutes)
- Immerse in hot water bath, 55^0 C for bluing (3 Seconds)
- Rinse in tap water (5 minutes)
- Counterstain in Eosin (15 seconds)
- Dehydrate absolute alcohol 100 % (2-4 dips)
- Clear in Xylene I and II (5 minutes)
- Mount with DPX.

Stock Solutions – EOSIN:

Stock – 1% aqueous Eosin-Y

Stock – 1% aqueous Phloxin B

Working Solutions – EOSIN:

100ml stock Eosin

10 ml stock Phloxin B

780 ml 95% Ethanol

4 ml glacial Acetic Acid

Stock Solution: - HEMATOXYLIN:

Harris Hematoxylin -1 L

Working Solution: -

0.25% Acid Alcohol

95% Ethanol -2578 ml

dH2O - 950ml

HCL -9ml

Result: **Nuclei – Blue, Cytoplasm – Pink, RBCs – Red.**

Reference: Bancroft D, Laylon C. The haematoxylin and eosin, In: Kim SS Ed, Bancroft's Theory and practice of histopathological techniques. 7th Ed., China, Churchill Livingstone; 2013: p173-187.

ANNEXURE –IV

KEY TO MASTERCHART

MN	Menorrhagia
BPV	Bleeding per vagina
PMB	Postmenopausal bleeding
MPV	Mass per vagina
PP	Pelvic pain
CD	Cervical discharge
MPA	Mass per Abdomen
FIB	Fibroid uterus
AUB	Abnormal uterine bleeding
UVP	Uterovaginal prolapse
PID	Pelvic inflammatory disease
PMB	Postmenopausal bleeding
OM	Ovarian mass
TAH-BSO	Total abdominal hysterectomy with bilateral salpingoophorectomy
TAH-USO	Total abdominal hysterectomy with unilateral salpingoophorectomy
TAH-BS	Total abdominal hysterectomy with bilateral salpingectomy
TAH	Total abdominal hysterectomy
VH	Vaginal Hysterectomy
PP	Proliferative phase
SP	Secretory phase
AP	Atrophic endometrium
ENDO-CA	Endometrial adeno carcinoma
SH-WA	Simple hyperplasia without atypia

EN-P	Endometrial polyp
ENT	Endometritis
CH-WA	Complex hyperplasia without atypia
SH-WA+EN	Hyperplasia without atypia & Endometrial polyp
AP+EN-P	Atrophic endometrium & Endometrial polyp
N	Normal
LM-IM	Intramural Leiomyoma
LM-SM	Submucosal Leiomyoma
LM-SS	Sub serosal Leiomyoma
LM-HC	Leiomyoma With hyaline changes
LM-LL	Lipo-leiomyoma
AT	Atrophy
AD	Adenomyosis
IN-CA	Invasion of adeno Carcinoma
M-CAL	Monckeberg's calcification
AD+LM	Adenomyosis & leiomyoma
AT+M-CAL	Atrophic & Monckebergs calcification
CNSC	Chronic non- specific cervicitis
SQ-M	Squamous metaplasia
CX-P	Endocervical polyp
LSIL	Low grade squamous intraepithelial lesion
CX-LM	Cervical leiomyoma
CP-CT	Chronic papillary endocervicitis
SQ-CA	Squamous cell carcinoma
CLC	Corpus Luteal Cyst

TLC	Theca lutein cyst
FC	Follicular cyst
SR-CAD	Serous cystadenoma
TER	Benign cystic teratoma
MU-CAD	Mucinous cystadenoma
SC	Simple cyst
SR-CA OV	Serous Ca of ovary
F-TH	Fibro thecoma
PTC	Para tubal cyst
HS	Hematosalpinx
FGC	Foreign body type giant cell

ANNEXURE –V MASTER CHART

Reg-No	Hp-.no	Age	parity	living	Chief complaints	Clinical indications	Type of Hysterectomy	Endometrial findings	Myometrial findings	Cervical findings	Right-Ovary	Left-Ovary	Right-Fallopian tube	Lteft-Fallopian tube
1	27/22	43	2	2	MN	Fib	TAH-USO	PP	LM-HC	CNSC	0	N		N
2	46/22	40	3	3	MN	Fib	TAH	PP	LM-HC	CNSC				
3	48/22	60	3	3	PMB	PMB	TAH	ENDO-CA	IN-CA	CNSC				
4	59/22	64	3	3	MPV	UVP	VH	AP	AT	SQ-M				
5	79/22	35	2	2	MPV	UVP	VH	PP	N	CNSC				
6	80/22	30	2	2	CD	PID	TAH-USO	SH-WA	N	CNSC		TLC		N
7	81/22	30	2	2	PP	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
8	82/22	50	4	4	MPV	UVP	VH	SH-WA+EN-P	N	CNSC				
9	93/22	46	3	3	MPV	UVP	VH	PP	AD+LM	SQ-M				
10	95/22	40	2	2	MN	AUB	TAH-USO	SH-WA	N	CNSC		N		N
11	129/22	50	2	2	MN	FIB	TAH-BSO	EN-P	LM-IM	CNSC	N	N	N	N
12	137/22	70	3	3	MPV	UVP	VH	AP	AT	SQ-M				
13	138/22	46	2	2	MPV	UVP	VH	PP	N	SQ-M				
14	139/22	45	2	2	MN	FIB	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
15	150/22	40	2	2	MN	FIB	TAH-BS	PP	LM-IM	SQ-M			HS	N
16	152/22	61	4	4	MPV	UVP	VH	AP	N	CNSC				
17	153/22	65	3	3	MPV	UVP	VH	AP	N	SQ-M				
18	154/22	37	2	2	MPV	UVP	VH	SP	N	CNSC				
19	160/22	38	1	1	MPV	UVP	VH	PP	N	CNSC				
20	200/22	33	2	2	MN	FIB	TAH-USO	SP	LM-HC	CNSC	N		PTC	
21	203/22	38	2	3	CD	PID	TAH-BSO	SP	N	CNSC	N	N	N	N
22	224/22	52	2	2	PMB	PMB	TAH	PP	N	CX-P				
23	254/22	34	1	1	MN	Fib	TAH	PP	LM-HC	CX-P				
24	284/22	48	5	5	PP	AUB	TAH-BSO	SH-WA	AD	CNSC	N	N	N	N
25	293/22	47	4	4	MPV	UVP	VH	AP	AT	CP-CT				
26	302/22	41	3	3	MPV	UVP	VH	SH-WA	LM-IM	CNSC				
27	332/22	65	2	2	MPV	UVP	VH	AP	AT	CNSC				
28	336/22	49	3	3	MPV	UVP	VH	PP	N	CNSC				
29	351/22	40	2	2	MN	Fib	TAH	PP	LM-IM	CNSC				
30	352/22	30	2	2	PP	PID	TAH	SP	N	CNSC				
31	353/22	60	4	4	MPV	UVP	VH	ENT	M-CAL	CNSC				
32	375/22	40	3	2	MN	AUB	TAH-USO	PP	AD	CNSC		CLC		N
33	374/22	52	3	3	MN	AUB	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
34	390/22	45	3	2	PP	Fib	TAH-USO	PP	LM-SM	CNSC	FC	N	N	N
35	397/22	35	4	4	MPV	UVP	VH	PP	N	CNSC				
36	400/22	39	2	2	PP	Fib	TAH	PP	LM-IM	CNSC				
37	409/22	45	4	4	MPV	UVP	VH	PP	LM-IM	CNSC				
38	410/22	34	2	2	MN	AUB	TAH-BSO	PP	AD	CNSC	SR-CAD	N	PTC	N
39	411/22	47	3	3	PP	AUB	TAH-BSO	SH-WA	LM-IM	CNSC	N	N	N	N
40	417/22	45	3	3	MN	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
41	427/22	38	2	2	BPV	Fib	TAH-BS	PP	LM-IM	CNSC			N	N
42	428/22	50	4	4	MPV	UVP	VH	AP	AT	CNSC				
43	441/22	40	2	2	MN	AUB	TAH-BSO	SP	N	CNSC	N	N	N	N
44	461/22	42	3	3	MN	Fib	TAH	PP	N	CNSC				
45	464/22	46	3	3	BPV	Fib	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
46	465/22	45	2	2	MPV	UVP	VH	PP	N	CNSC				
47	473/22	40	2	2	BPV	AUB	TAH	PP	LM-IM	CNSC				
48	481/22	35	1	1	BPV	OM	TAH	SP	N	CNSC				
49	482/22	38	2	2	MN	AUB	TAH	SP	N	CNSC				
50	483/22	40	3	3	CD	PID	TAH	PP	N	CNSC				

51	484/22	53	2	2	PP	AUB	TAH-BSO	PP	AD=LM	CNSC	N	N	PTC	N
52	490/22	40	3	3	MPV	UVP	VH	SP	N	CNSC				
53	505/22	45	2	2	BPV	FIB	TAH-BSO	PP	N	CNSC	SC	N	N	N
54	523/22	48	3	3	PP	FIB	TAH	SP	LM-IM	CX-P				
55	545/22	48	3	2	PP	AUB	TAH-BSO	SP	N	CNSC	TER	N	N	N
56	551/22	45	3	3	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	MU-CAD	N	N	N
57	552/22	41	2	2	MN	AUB	TAH-BSO	SP	AD	CNSC	N	N	N	N
58	569/22	38	3	3	MN	FIB	TAH-BSO	PP	LM-HC	CNSC	N	N	N	N
59	596/22	35	2	2	MPV	UVP	VH	SP	N	SQ-M				
60	598/22	28	2	2	CD	PID	TAH-USO	SP	N	CNSC	N		PTC	
61	623/22	55	3	3	MPV	UVP	VH	AP	AT	CNSC				
62	624/22	43	2	1	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	SR-CAD	N	N	N
63	631/22	55	2	2	MPV	UVP	TAH-BSO	PP	N	LSIL	N	N	N	N
64	652/22	76	3	2	MPV	UVP	VH	AT	N	SQ-M				
65	683/22	70	2	2	MPV	UVP	VH	AT	AT-M-CAL	CNSC				
66	722/22	50	3	3	MN	FIB	TAH	SH-WA	LM-IM	SQ-M				
67	755/22	38	3	3	PP	OM	TAH-USO	PP	N	CNSC	SR-CAD		N	
68	758/22	49	4	4	MPV	UVP	VH	AT	N	SQ-M				
69	765/22	55	3	3	MPV	UVP	VH	EN-P	AT	SQ-M				
70	766/22	40	4	4	MN	AUB	TAH-BSO	SP	N	CNSC	N	N	N	N
71	773/22	33	3	3	BPV	AUB	TAH	PP	N	CNSC				
72	774/22	68	4	4	MPV	UVP	VH	SP	N	CNSC				
73	781/22	45	2	2	MN	AUB	TAH-BS	PP	AD	CNSC			N	N
74	801/22	48	4	4	BPV	Fib	TAH-USO	PP	LM-IM	CNSC	N		N	
75	802/22	48	3	3	MN	Fib	TAH-BSO	PP	N	CNSC	N	N	N	N
76	849/22	49	2	2	MN	Fib	TAH-BSO	PP	LM-SM	CNSC	N	N	N	N
77	907/22	40	4	4	PP	Fib	TAH-USO	PP	LM-IM	SQ-M		SC		N
78	933/22	52	3	3	CD	PID	TAH-BS	EN-P	AD=LM	CNSC			N	N
79	940/22	44	3	2	PP	OM	TAH-USO	PP	AD	CNSC		SC		N
80	974/22	61	4	4	MPV	UVP	VH	AT	AT	CNSC				
81	996/22	48	2	2	BPV	Fib	TAH-USO	AT	AT	CNSC	N		PTC	
82	997/22	44	1	1	MN	Fib	TAH-BSO	PP	LM-HC	CNSC	N	N	N	N
83	1001/22	40	2	2	BPV	AUB	TAH-BSO	SP	AD	CNSC	N	N	N	N
84	1033/22	50	2	2	MPV	UVP	VH	PP	N	CNSC				
85	1042/22	48	4	4	MN	AUB	TAH-USO	PP	LM-IM	CNSC	N		N	
86	1074/22	49	2	2	CD	AUB	TAH-USO	PP	AD	SQ-M		SC		PTC
87	1084/22	38	3	3	MN	Fib	TAH-BS	PP	LM-IM	CNSC			N	N
88	1079/22	40	2	2	PP	OM	TAH-BSO	PP	N	CNSC	SR-CA-OV	N	N	N
89	1088/22	40	1	1	MPV	UVP	VH	EN-P	AD	CNSC				
90	1092/22	36	2	2	PP	AUB	TAH-BSO	SP	N	CNSC	FC	N	N	N
91	1132/22	40	2	2	MN	Fib	TAH	PP	LM-IM	CNSC				
92	1137/22	52	4	4	MPV	UVP	VH	AT	AT	CNSC				
93	1155/22	55	4	4	MPV	UVP	VH	AT	M-CAL	SQ-M				
94	1172/22	41	3	3	BPV	Fib	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
95	1177/22	45	2	2	MN	Fib	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
96	1190/22	47	5	5	PP	AUB	TAH-BSO	AT+EN-P	AT	CNSC	N	N	N	N
97	1191/22	45	2	2	MN	AUB	TAH-BSO	PP	AD	CNSC	N	N	N	N
98	1220/22	40	3	3	MPV	UVP	VH	PP	N	CNSC				
99	1221/22	39	3	3	MN	FIB	TAH-BSO	PP	LM-SHC	CNSC	N	N	N	N
100	1222/22	40	3	3	MN	FIB	TAH-BSO	PP	LM-LL	CNSC	N	N	N	N
101	1224/22	40	2	2	MPV	UVP	VH	AT	AT	CNSC				
102	1227/22	45	2	2	MN	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
103	1242/22	41	2	2	MN	AUB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
104	16/21	49	2	2	MPV	UVP	VH	PP	N	CNSC				
105	23/21	40	2	2	MN	FIB	TAH	SP	N	SQ-M				
106	24/21	32	2	2	MPV	UVP	VH	PP	N	CNSC				
107	30/21	33	3	3	CD	PID	TAH-BS	SH-WA	AD+LM	CNSC			N	N
108	36/21	45	3	3	PP	OM	TAH-BSO	SH-WA	N	CNSC	N	SC	N	PTC
109	63/21	40	2	2	PP	AUB	TAH-BS	PP	N	CNSC			PTC	N
110	89/21	41	3	3	MN	AUB	TAH-BS	SP	N	CNSC			N	N

111	110/21	35	2	2	PP	FIB	TAH-BSO	EN-P	LM-IM	CNSC	N	N	N	PTC
112	129/21	43	2	2	MN	FIB	TAH	SH-WA	LM-IM	CNSC				
113	130/21	27	2	2	CD	PID	TAH	PP	N	CNSC				
114	131/21	40	3	3	MN	UVP	TAH	ENDO-CA	N	CNSC				
115	156/21	48	2	1	MN	FIB	TAH-BSO	PP	LM-SS	CX-LM	N	N	N	N
116	170/21	36	2	2	CD	PID	TAH-BS	SP	N	CNSC			N	N
117	171/21	45	3	2	PP	Fib	TAH-USO	SP	LN-HC	CNSC		CLC		N
118	184/21	43	3	3	MN	AUB	TAH-USO	PP	AD	CNSC	N	N		
119	198/21	46	2	2	MN	Fib	TAH-BSO	PP	LM-IM	SQ-M	N	N	N	N
120	210/21	50	3	3	MPV	UVP	VH	PP	N	CNSC				
121	227/21	25	2	2	MN	Fib	TAH	PP	AD+LM	CNSC				
122	250/21	56	3	2	MPA	OM	TAH-BSO	PP	N	CNSC	F-TH	N	N	N
123	273/21	40	3	3	MPV	UVP	VH	PP	N	SQ-M				
124	274/21	38	3	3	MN	Fib	TAH	PP	LM-IM	CNSC				
125	275/21	44	2	2	BPV	Fib	TAH	PP	LM-HC	CX-P				
126	294/21	45	3	3	PP	AUB	TAH-BSO	SH-WA	N	CNSC	N	N	PTC	N
127	313/21	40	1	1	MN	AUB	TAH-USO	EN-P	LM-SM	CNSC		N		N
128	317/21	37	2	2	MPA	Fib	TAH	PP	LM-SS	CNSC				
129	319/21	55	4	4	MPV	UVP	VH	AT	AT	CNSC				
130	328/21	45	2	2	PP	UVP	TAH-BSO	PP	AD+LM	CP-CT	N	N	PTC	N
131	342/21	60	3	2	MPV	UVP	VH	AT	LM-IM	SQ-M				
132	350/21	40	2	2	CD	PID	TAH	SP	N	CNSC				
133	364/21	70	4	3	MPV	UVP	VH	AP+EN-P	AT	CNSC				
134	367/21	37	1	1	MN	Fib	TAH-BS	PP	LM-SS	CNSC			N	N
135	373/21	47	3	3	MN	Fib	TAH-BSO	AT	LM-IM	CNSC	N	N	N	N
136	402/21	45	3	3	MPV	UVP	VH	PP	N	SQ-M				
137	404/21	56	3	3	PP	AUB	TAH-BSO	PP	AD	CNSC	N	N	N	PTC
138	415/21	46	3	3	MN	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
139	419/21	46	3	3	MN	Fib	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
140	2901/21	49	4	4	BPV	AUB	TAH-BSO	PP	N	SQ-CA	N	N	N	N
141	2902/21	43	2	2	MN	Fib	TAH	PP	AD+LM	CNSC				
142	2904/21	42	2	2	MN	AUB	TAH-BSO	PP	N	CX-P	N	N	N	N
143	2905/21	61	4	4	MPV	UVP	VH	AT	N	CNSC				
144	2906/21	33	2	2	BPV	AUB	TAH	PP	N	CNSC				
145	2907//21	75	3	3	MPV	UVP	VH	AT	N	CX-P				
146	2948/21	40	2	2	PP	OM	TAH-USO	PP	N	SQ-M	N	SR-CAD	N	PTC
147	3147/21	41	2	2	MPV	UVP	VH	SH-WA	N	CNSC				
148	3242/21	55	4	4	MN	FIB	TAH	PP	LM-IM	CNSC				
149	2957/21	48	2	2	MN	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
150	2971/21	44	3	3	MN	AUB	TAH-BSO	PP	AD	CX-P	N	N	N	N
151	2978/21	40	2	2	PP	FIB	TAH-BSO	PP	LM-SS	CNSC	N	FC	N	N
152	2996/21	43	3	3	CD	PID	TAH	PP	AD	CNSC				
153	3014/21	42	2	2	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
154	3017/21	70	4	4	MPV	UVP	VH	AT	N	SQ-M				
155	3036/21	48	3	3	PP	FIB	VH	PP	LM-IM	CNSC	SR-CAD	N	N	N
156	3054/21	50	3	3	MPV	UVP	VH	AT	N	CNSC				
157	3059/21	48	3	3	PP	FIB	TAH-BSO	PP	AD+LM	CNSC	FC	N	N	N
158	3074/21	50	4	4	BPV	FIB	TAH-BSO	EN-P	N	CNSC	N	N	N	N
159	3242/21	55	2	2	MN	FIB	TAH	PP	LM-IM	CNSC				
160	3243/21	45	4	4	BPV	AUB	TAH-BS	PP	N	CNSC			N	N
161	3244/21	48	3	3	MN	FIB	TAH	PP	LM-IM	CNSC				
162	3267/21	30	2	2	CD	PID	TAH-BS	SH-WA	N	CNSC			N	N
163	3270/21	55	4	4	MPV	UVP	TAH-BSO	AP+EN-P	AT	CNSC	N	N	N	N
164	3274/21	50	3	3	BPV	AUB	TAH-BSO	EN-P	N	CX-P	N	N	N	N
165	2625/21	45	3	3	MN	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
166	2667/21	41	2	2	BPV	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
167	2669/21	41	2	2	MN	AUB	TAH	PP	N	CNSC				
168	2670/21	40	3	3	CD	PID	TAH	PP	N	CNSC				
169	2670/21	40	2	2	MPA	OM	TAH-BSO	PP	N	CNSC	SR-CAD	N	N	N

170	2677/21	46	4	4	MN	Fib	TAH-BS	SH-WA	LM-SS	CNSC			N	N
171	2742/21	48	2	2	MN	AUB	TAH-BSO	SH-WA	N	CNSC	N	N	N	N
172	2743/21	35	3	3	MPA	OM	TAH-BSO	PP	N	CNSC	FC	F-TH	N	N
173	2759/21	53	0	0	PMB	AUB	TAH-BSO	AT	N	SQ-M	N	N	N	N
174	2766/21	40	2	2	CD	PID	TAH-BS	PP	N	CNSC				
175	2767/21	30	2	2	PP	PID	TAH	SP	N	CNSC				
176	2229/21	63	2	2	MPV	UVP	VH	AT	AT	SQ-M				
177	2785/21	72	3	3	MPV	UVP	VH	AT	AT	SQ-M				
178	2791/21	42	3	3	BPV	Fib	TAH-BSO	PP	LM-SM	CX-LM	N	N	N	N
179	2792/21	38	2	2	MPA	Fib	TAH-BSO	SP	LM-SM	CNSC	N	N	N	N
180	2862/21	42	3	3	MN	Fib	TAH-USO	SP	LM-SM	CNSC		CLC		PTC
181	2880/21	40	2	2	CD	PID	TAH-BS	ENT	N	CNSC			N	N
182	2309/21	45	2	2	MN	Fib	TAH-BSO	PP	N	CNSC	N	N	N	N
183	2310/21	40	1	1	CD	PID	TAH-BSO	SP	LM-SM	SQ-M	FC	N	N	N
184	2353/21	35	2	2	MPV	UVP	VH	PP	N	CNSC				
185	2354/21	70	3	3	MPV	UVP	VH	AT	AT	SQ-M				
186	2355/21	40	3	3	MPV	UVP	VH	SP	N	CNSC				
187	2358/21	35	2	2	CD	PID	TAH-BS	SP	N	CNSC			N	N
188	2371/21	40	2	2	BPV	Fib	YAH	PP	LM-HC	SQ-M				
189	2384/21	65	2	2	MPV	UVP	VH	AP+EN-P	AT	SQ-M				
190	2396/21	50	3	3	BPV	Fib	TAH-BSO	AT	LM-IM	CNSC	N	N	N	N
191	2425/21	37	2	2	PP	Fib	TAH-BS	PP	LM-IM	SQ-M			PTC	N
192	2434/21	42	2	2	MPV	UVP	VH	SP	LM-IM	CNSC				
193	2435/21	35	3	3	CD	PID	TAH	SP	N	CNSC				
194	2436/21	38	2	2	CD	PID	TAH	SP	N	SQ-M				
195	2437/21	28	2	2	CD	PID	TAH-BS	SP	N	CNSC			N	N
196	2438/21	52	3	3	PP	PID	TAH-BSO	SP	N	SQ-M	CLC	N	N	N
197	2439/21	43	2	2	MN	FIB	TAH-BS	PP	LM-IM	CNSC			N	N
198	2469/21	35	2	2	PP	FIB	TAH-USO	SP	LM-IM	CNSC	N		N	
199	2480/21	42	3	3	MN	FIB	TAH-BSO	SH-WA	LM-IM	CNSC	N	N	N	N
200	2526/21	46	4	3	MPA	FIB	TAH-BSO	PP	LM-IM	CNSC	SR-CAD	N	N	N
201	2541/21	38	2	2	BPV	AUB	TAH-BS	SP	AD	CNSC			N	N
202	2554/21	60	3	3	MPV	UVP	VH	AH+EN-P	N	CNSC				
203	2555/21	50	3	3	PP	PID	TAH-BSO	AT	N	CNSC	SC	N	N	N
204	2556/21	52	3	3	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	SC	N	N	N
205	2557/21	27	2	2	MPV	UVP	VH	PP	N	CNSC				
206	2558/21	32	2	2	MN	FIB	TAH	SP	N	CNSC				
207	2560/21	41	3	3	MN	FIB	TAH-BSO	AT	LM-IM	CNSC	N	N	N	N
208	2013/21	40	2	2	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	N	SR-CAD	N	FGC
209	2056/21	36	1	1	BPV	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
210	2066/21	50	3	3	MN	FIB	TAH-BSO	PP	LM-SM	CNSC	N	N	N	N
211	2070/21	45	2	2	BPV	AUB	TAH-BSO	PP	N	CNSC	SC	N	N	N
212	2080/21	65	3	3	MPV	UVP	VH	AT	N	CNSC				
213	2081/21	65	2	2	MPV	UVP	VH	AT	AT	CNSC				
214	2194/21	61	2	2	PP	OM	TAH-BSO	AT	N	CNSC	N	TER	N	N
215	2145/21	46	2	2	MN	AUB	TAH-BSO	PP	AD	CNSC	N	N	N	N
216	2151/21	44	4	4	MN	FIB	TAH-BSO	SP	LM-IM	CNSC	SR-CAD	N	N	N
217	2176/21	44	2	2	PP	FIB	TAH-BSO	SH-WA	LM-IM	CNSC	FC	N	N	N
218	2177/21	45	3	3	MN	AUB	TAH-BSO	EN-P	N	CNSC	N	N	N	N
219	2188/21	40	2	2	MN	FIB	TAH	PP	LM-IM	CNSC				
220	2213/21	48	3	3	BPV	FIB	TAH	PP	LM-IM	CNSC				
221	2217/21	56	3	3	MN	AUB	TAJ-USO	AT	LM-IM	CNSC	N		N	
222	2229/21	63	2	2	MPV	UVP	VH	AT	LM-HC	CNSC				
223	2231/21	40	2	2	MN	AUB	TAH	PP	LM-IM	CP-CT				
224	2232/21	40	1	1	MN	FIB	TAH-USO	PP	LM-IM	CNSC		CLC		N
225	2259/21	50	3	3	MN	FIB	TAH-BSO	PP	LM-HC	CNSC	N	CLC	N	N
226	2281/21	46	2	2	PP	FIB	TAH-BSO	PP	LN-IM	CNSC	CLC	N	PTC	N
227	2282/21	64	2	2	MPA	FIB	TAH-BSO	AT-EN-P	LM-IM	CNSC	N	N	N	N
228	1714/21	70	3	3	PMB	PMB	TAH-BSO	AT	AD+LM	CNSC	N	N	N	N
229	1738/21	55	3	3	PMB	PMB	TAH-BSO	ENDO-CA	N	CNSC	N	N	N	N

230	1742/21	36	1	1	BPV	AUB	TAH-BS	SP	LM-SM	SQ-M			N	N
231	1756/21	45	2	2	BPV	FIB	TAH-BSO	PP	LM-SS	CX-LM	N	N	N	N
232	1835/21	69	3	3	MPV	UVP	VH	AT	AT	CNSC				
233	1850/21	40	2	2	BPV	AUB	TAH	PP	N	CNSC				
234	1880/21	35	2	2	MN	AUB	TAH-BS	PP	N	CNSC			N	PTC
235	1886/21	39	2	2	PP	FIB	TAH-USO	PP	LM-SM	CNSC		N		PTC
236	1905/21	38	2	2	MN	FIB	TAH-USO	PP	LM-IM	CNSC	CLC		N	
237	1911/21	32	2	2	CD	PID	TAH	SP	N	CNSC				
238	1912/21	50	2	2	MPV	UVP	VH	SP	LM-SM	CNSC				
239	1913/21	50	3	3	MPV	UVP	VH	SP	LM-IM	CNSC				
240	1914/21	49	2	2	MPV	UVP	VH	PP	AD	CNSC				
241	1923/21	65	3	3	MPV	UVP	VH	AT	AT	CNSC				
242	1939/21	45	1	1	PP	FIB	TAH-BSO	SH-WA	LM-IM	CNSC	N	N	N	N
243	1940/21	36	3	3	MN	FIB	TAH-BS	EN-P	LM-IM	CNSC			N	N
244	1946/21	39	3	3	BPV	AUB	TAH	SP	AD	CNSC				
245	1959/21	38	2	2	MPA	FIB	TAH-BSO	PP	AD+LM	CNSC	N	MU-CAD	N	N
246	1987/21	40	2	2	MN	FIB	TAH	PP	LM-IM	CNSC				
247	1990/21	46	3	3	MN	FIB	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
248	1985/21	41	2	2	MPV	UVP	VH	SH-WA	N	CNSC				
249	1982/21	36	2	2	PP	AUB	TAH	CH-WA	N	CNSC				

Reg.No	Hp.no	Age	parity	living	CC	C indica	TOH	ENDO	MYO	CERVIX	Rt-Ovary	Lt-Ovary	Rt-FT	Lt-FT
1	27/22	43	2	2	MN	Fib	TAH-USO	PP	LM-HC	CNSC	0	N		N
2	46/22	40	3	3	MN	Fib	TAH	PP	LM-HC	CNSC				
3	48/22	60	3	3	PMB	PMB	TAH	ENDO-CA	IN-CA	CNSC				
4	59/22	64	3	3	MPV	UVP	VH	AP	AT	SQ-M				
5	79/22	35	2	2	MPV	UVP	VH	PP	N	CNSC				
6	80/22	30	2	2	CD	PID	TAH-USO	SH-WA	N	CNSC		TLC		N
7	81/22	30	2	2	PP	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
8	82/22	50	4	4	MPV	UVP	VH	SH-WA+EN-P	N	CNSC				
9	93/22	46	3	3	MPV	UVP	VH	PP	AD+LM	SQ-M				
10	95/22	40	2	2	MN	AUB	TAH-USO	SH-WA	N	CNSC		N		N
11	129/22	50	2	2	MN	FIB	TAH-BSO	EN-P	LM-IM	CNSC	N	N	N	N
12	137/22	70	3	3	MPV	UVP	VH	AP	AT	SQ-M				
13	138/22	46	2	2	MPV	UVP	VH	PP	N	SQ-M				
14	139/22	45	2	2	MN	FIB	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
15	150/22	40	2	2	MN	FIB	TAH-BS	PP	LM-IM	SQ-M			HS	N
16	152/22	61	4	4	MPV	UVP	VH	AP	N	CNSC				
17	153/22	65	3	3	MPV	UVP	VH	AP	N	SQ-M				
18	154/22	37	2	2	MPV	UVP	VH	SP	N	CNSC				
19	160/22	38	1	1	MPV	UVP	VH	PP	N	CNSC				
20	200/22	33	2	2	MN	FIB	TAH-USO	SP	LM-HC	CNSC	N		PTC	
21	203/22	38	2	3	CD	PID	TAH-BSO	SP	N	CNSC	N	N	N	N
22	224/22	52	2	2	PMB	PMB	TAH	PP	N	CX-P				
23	254/22	34	1	1	MN	Fib	TAH	PP	LM-HC	CX-P				
24	284/22	48	5	5	PP	AUB	TAH-BSO	SH-WA	AD	CNSC	N	N	N	N
25	293/22	47	4	4	MPV	UVP	VH	AP	AT	CP-CT				
26	302/22	41	3	3	MPV	UVP	VH	SH-WA	LM-IM	CNSC				
27	332/22	65	2	2	MPV	UVP	VH	AP	AT	CNSC				
28	336/22	49	3	3	MPV	UVP	VH	PP	N	CNSC				
29	351/22	40	2	2	MN	Fib	TAH	PP	LM-IM	CNSC				
30	352/22	30	2	2	PP	PID	TAH	SP	N	CNSC				
31	353/22	60	4	4	MPV	UVP	VH	ENT	M-CAL	CNSC				
32	375/22	40	3	2	MN	AUB	TAH-USO	PP	AD	CNSC		CLC		N
33	374/22	52	3	3	MN	AUB	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
34	390/22	45	3	2	PP	Fib	TAH-USO	PP	LM-SM	CNSC	FC	N	N	N
35	397/22	35	4	4	MPV	UVP	VH	PP	N	CNSC				
36	400/22	39	2	2	PP	Fib	TAH	PP	LM-IM	CNSC				
37	409/22	45	4	4	MPV	UVP	VH	PP	LM-IM	CNSC				
38	410/22	34	2	2	MN	AUB	TAH-BSO	PP	AD	CNSC	SR-CAD	N	PTC	N
39	411/22	47	3	3	PP	AUB	TAH-BSO	SH-WA	LM-IM	CNSC	N	N	N	N
40	417/22	45	3	3	MN	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
41	427/22	38	2	2	BPV	Fib	TAH-BS	PP	LM-IM	CNSC			N	N
42	428/22	50	4	4	MPV	UVP	VH	AP	AT	CNSC				
43	441/22	40	2	2	MN	AUB	TAH-BSO	SP	N	CNSC	N	N	N	N
44	461/22	42	3	3	MN	Fib	TAH	PP	N	CNSC				

45	464/22	46	3	3	BPV	Fib	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
46	465/22	45	2	2	MPV	UVP	VH	PP	N	CNSC				
47	473/22	40	2	2	BPV	AUB	TAH	PP	LM-IM	CNSC				
48	481/22	35	1	1	BPV	OM	TAH	SP	N	CNSC				
49	482/22	38	2	2	MN	AUB	TAH	SP	N	CNSC				
50	483/22	40	3	3	CD	PID	TAH	PP	N	CNSC				
51	484/22	53	2	2	PP	AUB	TAH-BSO	PP	AD=LM	CNSC	N	N	PTC	N
52	490/22	40	3	3	MPV	UVP	VH	SP	N	CNSC				
53	505/22	45	2	2	BPV	FIB	TAH-BSO	PP	N	CNSC	SC	N	N	N
54	523/22	48	3	3	PP	FIB	TAH	SP	LM-IM	CX-P				
55	545/22	48	3	2	PP	AUB	TAH-BSO	SP	N	CNSC	TER	N	N	N
56	551/22	45	3	3	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	MU-CAD	N	N	N
57	552/22	41	2	2	MN	AUB	TAH-BSO	SP	AD	CNSC	N	N	N	N
58	569/22	38	3	3	MN	FIB	TAH-BSO	PP	LM-HC	CNSC	N	N	N	N
59	596/22	35	2	2	MPV	UVP	VH	SP	N	SQ-M				
60	598/22	28	2	2	CD	PID	TAH-USO	SP	N	CNSC	N		PTC	
61	623/22	55	3	3	MPV	UVP	VH	AP	AT	CNSC				
62	624/22	43	2	1	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	SR-CAD	N	N	N
63	631/22	55	2	2	MPV	UVP	TAH-BSO	PP	N	LSIL	N	N	N	N
64	652/22	76	3	2	MPV	UVP	VH	AT	N	SQ-M				
65	683/22	70	2	2	MPV	UVP	VH	AT	AT-M-CAL	CNSC				
66	722/22	50	3	3	MN	FIB	TAH	SH-WA	LM-IM	SQ-M				
67	755/22	38	3	3	PP	OM	TAH-USO	PP	N	CNSC	SR-CAD		N	
68	758/22	49	4	4	MPV	UVP	VH	AT	N	SQ-M				
69	765/22	55	3	3	MPV	UVP	VH	EN-P	AT	SQ-M				
70	766/22	40	4	4	MN	AUB	TAH-BSO	SP	N	CNSC	N	N	N	N
71	773/22	33	3	3	BPV	AUB	TAH	PP	N	CNSC				
72	774/22	68	4	4	MPV	UVP	VH	SP	N	CNSC				
73	781/22	45	2	2	MN	AUB	TAH-BS	PP	AD	CNSC			N	N
74	801/22	48	4	4	BPV	Fib	TAH-USO	PP	LM-IM	CNSC	N		N	
75	802/22	48	3	3	MN	Fib	TAH-BSO	PP	N	CNSC	N	N	N	N
76	849/22	49	2	2	MN	Fib	TAH-BSO	PP	LM-SM	CNSC	N	N	N	N
77	907/22	40	4	4	PP	Fib	TAH-USO	PP	LM-IM	SQ-M		SC		N
78	933/22	52	3	3	CD	PID	TAH-BS	EN-P	AD=LM	CNSC			N	N
79	940/22	44	3	2	PP	OM	TAH-USO	PP	AD	CNSC		SC		N
80	974/22	61	4	4	MPV	UVP	VH	AT	AT	CNSC				
81	996/22	48	2	2	BPV	Fib	TAH-USO	AT	AT	CNSC	N		PTC	
82	997/22	44	1	1	MN	Fib	TAH-BSO	PP	LM-HC	CNSC	N	N	N	N
83	1001/22	40	2	2	BPV	AUB	TAH-BSO	SP	AD	CNSC	N	N	N	N
84	1033/22	50	2	2	MPV	UVP	VH	PP	N	CNSC				
85	1042/22	48	4	4	MN	AUB	TAH-USO	PP	LM-IM	CNSC	N		N	
86	1074/22	49	2	2	CD	AUB	TAH-USO	PP	AD	SQ-M		SC		PTC
87	1084/22	38	3	3	MN	Fib	TAH-BS	PP	LM-IM	CNSC			N	N
88	1079/22	40	2	2	PP	OM	TAH-BSO	PP	N	CNSC	SR-CA-OV	N	N	N
89	1088/22	40	1	1	MPV	UVP	VH	EN-P	AD	CNSC				
90	1092/22	36	2	2	PP	AUB	TAH-BSO	SP	N	CNSC	FC	N	N	N
91	1132/22	40	2	2	MN	Fib	TAH	PP	LM-IM	CNSC				

92	1137/22	52	4	4	MPV	UVP	VH	AT	AT	CNSC				
93	1155/22	55	4	4	MPV	UVP	VH	AT	M-CAL	SQ-M				
94	1172/22	41	3	3	BPV	Fib	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
95	1177/22	45	2	2	MN	Fib	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
96	1190/22	47	5	5	PP	AUB	TAH-BSO	AT+EN-P	AT	CNSC	N	N	N	N
97	1191/22	45	2	2	MN	AUB	TAH-BSO	PP	AD	CNSC	N	N	N	N
98	1220/22	40	3	3	MPV	UVP	VH	PP	N	CNSC				
99	1221/22	39	3	3	MN	FIB	TAH-BSO	PP	LM-SHC	CNSC	N	N	N	N
100	1222/22	40	3	3	MN	FIB	TAH-BSO	PP	LM-LL	CNSC	N	N	N	N
101	1224/22	40	2	2	MPV	UVP	VH	AT	AT	CNSC				
102	1227/22	45	2	2	MN	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
103	1242/22	41	2	2	MN	AUB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
104	16/21	49	2	2	MPV	UVP	VH	PP	N	CNSC				
105	23/21	40	2	2	MN	FIB	TAH	SP	N	SQ-M				
106	24/21	32	2	2	MPV	UVP	VH	PP	N	CNSC				
107	30/21	33	3	3	CD	PID	TAH-BS	SH-WA	AD+LM	CNSC			N	N
108	36/21	45	3	3	PP	OM	TAH-BSO	SH-WA	N	CNSC	N	SC	N	PTC
109	63/21	40	2	2	PP	AUB	TAH-BS	PP	N	CNSC			PTC	N
110	89/21	41	3	3	MN	AUB	TAH-BS	SP	N	CNSC			N	N
111	110/21	35	2	2	PP	FIB	TAH-BSO	EN-P	LM-IM	CNSC	N	N	N	PTC
112	129/21	43	2	2	MN	FIB	TAH	SH-WA	LM-IM	CNSC				
113	130/21	27	2	2	CD	PID	TAH	PP	N	CNSC				
114	131/21	40	3	3	MN	UVP	TAH	ENDO-CA	N	CNSC				
115	156/21	48	2	1	MN	FIB	TAH-BSO	PP	LM-SS	CX-LM	N	N	N	N
116	170/21	36	2	2	CD	PID	TAH-BS	SP	N	CNSC			N	N
117	171/21	45	3	2	PP	Fib	TAH-USO	SP	LN-HC	CNSC		CLC		N
118	184/21	43	3	3	MN	AUB	TAH-USO	PP	AD	CNSC	N	N		
119	198/21	46	2	2	MN	Fib	TAH-BSO	PP	LM-IM	SQ-M	N	N	N	N
120	210/21	50	3	3	MPV	UVP	VH	PP	N	CNSC				
121	227/21	25	2	2	MN	Fib	TAH	PP	AD+LM	CNSC				
122	250/21	56	3	2	MPA	OM	TAH-BSO	PP	N	CNSC	F-TH	N	N	N
123	273/21	40	3	3	MPV	UVP	VH	PP	N	SQ-M				
124	274/21	38	3	3	MN	Fib	TAH	PP	LM-IM	CNSC				
125	275/21	44	2	2	BPV	Fib	TAH	PP	LM-HC	CX-P				
126	294/21	45	3	3	PP	AUB	TAH-BSO	SH-WA	N	CNSC	N	N	PTC	N
127	313/21	40	1	1	MN	AUB	TAH-USO	EN-P	LM-SM	CNSC		N		N
128	317/21	37	2	2	MPA	Fib	TAH	PP	LM-SS	CNSC				
129	319/21	55	4	4	MPV	UVP	VH	AT	AT	CNSC				
130	328/21	45	2	2	PP	UVP	TAH-BSO	PP	AD+LM	CP-CT	N	N	PTC	N
131	342/21	60	3	2	MPV	UVP	VH	AT	LM-IM	SQ-M				
132	350/21	40	2	2	CD	PID	TAH	SP	N	CNSC				
133	364/21	70	4	3	MPV	UVP	VH	AP+EN-P	AT	CNSC				
134	367/21	37	1	1	MN	Fib	TAH-BS	PP	LM-SS	CNSC			N	N
135	373/21	47	3	3	MN	Fib	TAH-BSO	AT	LM-IM	CNSC	N	N	N	N
136	402/21	45	3	3	MPV	UVP	VH	PP	N	SQ-M				
137	404/21	56	3	3	PP	AUB	TAH-BSO	PP	AD	CNSC	N	N	N	PTC
138	415/21	46	3	3	MN	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N

139	419/21	46	3	3	MN	Fib	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
140	2901/21	49	4	4	BPV	AUB	TAH-BSO	PP	N	SQ-CA	N	N	N	N
141	2902/21	43	2	2	MN	Fib	TAH	PP	AD+LM	CNSC				
142	2904/21	42	2	2	MN	AUB	TAH-BSO	PP	N	CX-P	N	N	N	N
143	2905/21	61	4	4	MPV	UVP	VH	AT	N	CNSC				
144	2906/21	33	2	2	BPV	AUB	TAH	PP	N	CNSC				
145	2907//21	75	3	3	MPV	UVP	VH	AT	N	CX-P				
146	2948/21	40	2	2	PP	OM	TAH-USO	PP	N	SQ-M	N	SR-CAD	N	PTC
147	3147/21	41	2	2	MPV	UVP	VH	SH-WA	N	CNSC				
148	3242/21	55	4	4	MN	FIB	TAH	PP	LM-IM	CNSC				
149	2957/21	48	2	2	MN	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
150	2971/21	44	3	3	MN	AUB	TAH-BSO	PP	AD	CX-P	N	N	N	N
151	2978/21	40	2	2	PP	FIB	TAH-BSO	PP	LM-SS	CNSC	N	FC	N	N
152	2996/21	43	3	3	CD	PID	TAH	PP	AD	CNSC				
153	3014/21	42	2	2	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
154	3017/21	70	4	4	MPV	UVP	VH	AT	N	SQ-M				
155	3036/21	48	3	3	PP	FIB	VH	PP	LM-IM	CNSC	SR-CAD	N	N	N
156	3054/21	50	3	3	MPV	UVP	VH	AT	N	CNSC				
157	3059/21	48	3	3	PP	FIB	TAH-BSO	PP	AD+LM	CNSC	FC	N	N	N
158	3074/21	50	4	4	BPV	FIB	TAH-BSO	EN-P	N	CNSC	N	N	N	N
159	3242/21	55	2	2	MN	FIB	TAH	PP	LM-IM	CNSC				
160	3243/21	45	4	4	BPV	AUB	TAH-BS	PP	N	CNSC			N	N
161	3244/21	48	3	3	MN	FIB	TAH	PP	LM-IM	CNSC				
162	3267/21	30	2	2	CD	PID	TAH-BS	SH-WA	N	CNSC			N	N
163	3270/21	55	4	4	MPV	UVP	TAH-BSO	AP+EN-P	AT	CNSC	N	N	N	N
164	3274/21	50	3	3	BPV	AUB	TAH-BSO	EN-P	N	CX-P	N	N	N	N
165	2625/21	45	3	3	MN	FIB	TAH-BSO	PP	LM-IM	CNSC	N	N	N	N
166	2667/21	41	2	2	BPV	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
167	2669/21	41	2	2	MN	AUB	TAH	PP	N	CNSC				
168	2670/21	40	3	3	CD	PID	TAH	PP	N	CNSC				
169	2670/21	40	2	2	MPA	OM	TAH-BSO	PP	N	CNSC	SR-CAD	N	N	N
170	2677/21	46	4	4	MN	Fib	TAH-BS	SH-WA	LM-SS	CNSC			N	N
171	2742/21	48	2	2	MN	AUB	TAH-BSO	SH-WA	N	CNSC	N	N	N	N
172	2743/21	35	3	3	MPA	OM	TAH-BSO	PP	N	CNSC	FC	F-TH	N	N
173	2759/21	53	0	0	PMB	AUB	TAH-BSO	AT	N	SQ-M	N	N	N	N
174	2766/21	40	2	2	CD	PID	TAH-BS	PP	N	CNSC				
175	2767/21	30	2	2	PP	PID	TAH	SP	N	CNSC				
176	2229/21	63	2	2	MPV	UVP	VH	AT	AT	SQ-M				
177	2785/21	72	3	3	MPV	UVP	VH	AT	AT	SQ-M				
178	2791/21	42	3	3	BPV	Fib	TAH-BSO	PP	LM-SM	CX-LM	N	N	N	N
179	2792/21	38	2	2	MPA	Fib	TAH-BSO	SP	LM-SM	CNSC	N	N	N	N
180	2862/21	42	3	3	MN	Fib	TAH-USO	SP	LM-SM	CNSC		CLC		PTC
181	2880/21	40	2	2	CD	PID	TAH-BS	ENT	N	CNSC			N	N
182	2309/21	45	2	2	MN	Fib	TAH-BSO	PP	N	CNSC	N	N	N	N
183	2310/21	40	1	1	CD	PID	TAH-BSO	SP	LM-SM	SQ-M	FC	N	N	N
184	2353/21	35	2	2	MPV	UVP	VH	PP	N	CNSC				
185	2354/21	70	3	3	MPV	UVP	VH	AT	AT	SQ-M				

186	2355/21	40	3	3	MPV	UVP	VH	SP	N	CNSC				
187	2358/21	35	2	2	CD	PID	TAH-BS	SP	N	CNSC			N	N
188	2371/21	40	2	2	BPV	Fib	YAH	PP	LM-HC	SQ-M				
189	2384/21	65	2	2	MPV	UVP	VH	AP+EN-P	AT	SQ-M				
190	2396/21	50	3	3	BPV	Fib	TAH-BSO	AT	LM-IM	CNSC	N	N	N	N
191	2425/21	37	2	2	PP	Fib	TAH-BS	PP	LM-IM	SQ-M			PTC	N
192	2434/21	42	2	2	MPV	UVP	VH	SP	LM-IM	CNSC				
193	2435/21	35	3	3	CD	PID	TAH	SP	N	CNSC				
194	2436/21	38	2	2	CD	PID	TAH	SP	N	SQ-M				
195	2437/21	28	2	2	CD	PID	TAH-BS	SP	N	CNSC			N	N
196	2438/21	52	3	3	PP	PID	TAH-BSO	SP	N	SQ-M	CLC	N	N	N
197	2439/21	43	2	2	MN	FIB	TAH-BS	PP	LM-IM	CNSC			N	N
198	2469/21	35	2	2	PP	FIB	TAH-USO	SP	LM-IM	CNSC	N		N	
199	2480/21	42	3	3	MN	FIB	TAH-BSO	SH-WA	LM-IM	CNSC	N	N	N	N
200	2526/21	46	4	3	MPA	FIB	TAH-BSO	PP	LM-IM	CNSC	SR-CAD	N	N	N
201	2541/21	38	2	2	BPV	AUB	TAH-BS	SP	AD	CNSC			N	N
202	2554/21	60	3	3	MPV	UVP	VH	AH+EN-P	N	CNSC				
203	2555/21	50	3	3	PP	PID	TAH-BSO	AT	N	CNSC	SC	N	N	N
204	2556/21	52	3	3	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	SC	N	N	N
205	2557/21	27	2	2	MPV	UVP	VH	PP	N	CNSC				
206	2558/21	32	2	2	MN	FIB	TAH	SP	N	CNSC				
207	2560/21	41	3	3	MN	FIB	TAH-BSO	AT	LM-IM	CNSC	N	N	N	N
208	2013/21	40	2	2	PP	FIB	TAH-BSO	PP	LM-IM	CNSC	N	SR-CAD	N	FGC
209	2056/21	36	1	1	BPV	AUB	TAH-BSO	PP	N	CNSC	N	N	N	N
210	2066/21	50	3	3	MN	FIB	TAH-BSO	PP	LM-SM	CNSC	N	N	N	N
211	2070/21	45	2	2	BPV	AUB	TAH-BSO	PP	N	CNSC	SC	N	N	N
212	2080/21	65	3	3	MPV	UVP	VH	AT	N	CNSC				
213	2081/21	65	2	2	MPV	UVP	VH	AT	AT	CNSC				
214	2194/21	61	2	2	PP	OM	TAH-BSO	AT	N	CNSC	N	TER	N	N
215	2145/21	46	2	2	MN	AUB	TAH-BSO	PP	AD	CNSC	N	N	N	N
216	2151/21	44	4	4	MN	FIB	TAH-BSO	SP	LM-IM	CNSC	SR-CAD	N	N	N
217	2176/21	44	2	2	PP	FIB	TAH-BSO	SH-WA	LM-IM	CNSC	FC	N	N	N
218	2177/21	45	3	3	MN	AUB	TAH-BSO	EN-P	N	CNSC	N	N	N	N
219	2188/21	40	2	2	MN	FIB	TAH	PP	LM-IM	CNSC				
220	2213/21	48	3	3	BPV	FIB	TAH	PP	LM-IM	CNSC				
221	2217/21	56	3	3	MN	AUB	TAJ-USO	AT	LM-IM	CNSC	N		N	
222	2229/21	63	2	2	MPV	UVP	VH	AT	LM-HC	CNSC				
223	2231/21	40	2	2	MN	AUB	TAH	PP	LM-IM	CP-CT				
224	2232/21	40	1	1	MN	FIB	TAH-USO	PP	LM-IM	CNSC		CLC		N
225	2259/21	50	3	3	MN	FIB	TAH-BSO	PP	LM-HC	CNSC	N	CLC	N	N
226	2281/21	46	2	2	PP	FIB	TAH-BSO	PP	LN-IM	CNSC	CLC	N	PTC	N
227	2282/21	64	2	2	MPA	FIB	TAH-BSO	AT-EN-P	LM-IM	CNSC	N	N	N	N
228	1714/21	70	3	3	PMB	PMB	TAH-BSO	AT	AD+LM	CNSC	N	N	N	N
229	1738/21	55	3	3	PMB	PMB	TAH-BSO	ENDO-CA	N	CNSC	N	N	N	N
230	1742/21	36	1	1	BPV	AUB	TAH-BS	SP	LM-SM	SQ-M			N	N
231	1756/21	45	2	2	BPV	FIB	TAH-BSO	PP	LM-SS	CX-LM	N	N	N	N
232	1835/21	69	3	3	MPV	UVP	VH	AT	AT	CNSC				

233	1850/21	40	2	2	BPV	AUB	TAH	PP	N	CNSC				
234	1880/21	35	2	2	MN	AUB	TAH-BS	PP	N	CNSC			N	PTC
235	1886/21	39	2	2	PP	FIB	TAH-USO	PP	LM-SM	CNSC		N		PTC
236	1905/21	38	2	2	MN	FIB	TAH-USO	PP	LM-IM	CNSC	CLC		N	
237	1911/21	32	2	2	CD	PID	TAH	SP	N	CNSC				
238	1912/21	50	2	2	MPV	UVP	VH	SP	LM-SM	CNSC				
239	1913/21	50	3	3	MPV	UVP	VH	SP	LM-IM	CNSC				
240	1914/21	49	2	2	MPV	UVP	VH	PP	AD	CNSC				
241	1923/21	65	3	3	MPV	UVP	VH	AT	AT	CNSC				
242	1939/21	45	1	1	PP	FIB	TAH-BSO	SH-WA	LM-IM	CNSC	N	N	N	N
243	1940/21	36	3	3	MN	FIB	TAH-BS	EN-P	LM-IM	CNSC			N	N
244	1946/21	39	3	3	BPV	AUB	TAH	SP	AD	CNSC				
245	1959/21	38	2	2	MPA	FIB	TAH-BSO	PP	AD+LM	CNSC	N	MU-CAD	N	N
246	1987/21	40	2	2	MN	FIB	TAH	PP	LM-IM	CNSC				
247	1990/21	46	3	3	MN	FIB	TAH-BSO	PP	AD+LM	CNSC	N	N	N	N
248	1985/21	41	2	2	MPV	UVP	VH	SH-WA	N	CNSC				
249	1982/21	36	2	2	PP	AUB	TAH	CH-WA	N	CNSC				

Histopathology no

AGE

PARITY

LIVING

Chief Complaints

Clinical indication

Type of Hysterectomy
1-TAH+BSO
2-VH
3-