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CLINICOPATHOLOGICAL ASSESSMENT OF HYSTERECTOMY SPECIMENS IN A SAMPLE OF IRAQI PATIENTS

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ABSTRACT

Background: Hysterectomy is the most commonly performed gynaecological surgery done in female and is a successful operation in terms of symptomatic relief, patient satisfaction as well as provides definitive cure to many diseases involving uterus and adnexa like fibroids, menorrhagia, adenomyosis, endometriosis, pelvic inflammatory disease, uterovaginal prolapse and malignancy. Objective: To study the frequency of different pathologies of uterus and adenxia in hysterectomy and associate the clinicopathological parameters with the histopathological diagnosis. Materials and method: A retrospective study was conducted over the period from January 2024 to January 2025, which includes an analysis of 180 randomly selected patients with hysterectomy. Cases received between 2022 and 2023 in the Teaching Laboratory of Al-Imamain Al-Kadhimain medical city were collected for this study. For each case the histopathological reports and the slides were collected. In addition, clinical parameters such as (age, type of hysterectomy, clinical indication, ultrasound diagnosis) were collected from the pathology reports and ultrasound reports. In the Pathology Department at College of Medicine/Al-Nahrain University, H&E stained slides were re- examined by the supervisor pathologist for revision of the diagnosis. Result: One hundred-eighty hysterectomy specimens were examined and the peak age group were from 40-49 years (49.4%), and the most common clinical presentation was abnormal uterine bleeding (AUB) (61.67%). Most common histopathological lesion was endometrial hyperplasia (43.4%), followed by (31.7%) of cases presented as leiomyoma alone and (23.3%) as dual pathology of lieomyoma with adenomyosis. Chronic cervicitis was the commonest incidental histopathological finding seen in hysterectomy specimens. Conclusions: Histopathological findings typically correspond with clinical diagnoses, however; some conditions, including chronic cervicitis and adenomyosis, are frequently identified incidentally. Consequently, it is imperative to perform a comprehensive histopathological evaluation of all hysterectomy specimens, regardless of their gross appearance, to ensure accurate diagnosis and enhance postoperative care.

KEYWORDS: Hysterectomy, Leiomyoma, Uterine prolapse, Adenomyosis, endometrial hyperplasia, AUB.

INTRODUCTION

1.1. Introduction

Hysterectomy is the surgical removal of the uterus, it is the second most frequently performed major surgical procedure on women all over the world especially peri and postmenopausal, second only to caesarean. Historically Charles Clay performed the first subtotal hysterectomy in Manchester England in 1843 and the first total abdominal hysterectomy was done in 1929.[1]

Forty percent of women all over the world will have a hysterectomy by the age of 64, and the indication for the majority will be to relieve symptoms due to benign pathology, and thereby improve quality of life, hysterectomy is a successful operation in terms of symptom relief and patient satisfaction. It provides definitive cure to many diseases involving uterus as well as adnexa, e.g., fibroids, DUB, adenomyosis, endometriosis, pelvic inflammatory disease, pelvic organ prolapse and malignancy.[2]

There are several types of hysterectomy, each tailored to the underlying condition and clinical need

Total hysterectomy: Removing the uterus and cervix, but leaving the ovaries. Supracervical hysterectomy (subtotal hysterectomy): Removing just the upper part of the uterus while leaving the cervix.

Total hysterectomy with bilateral salpingo-oophorectomy: Removing the uterus, cervix, fallopian tubes (salpingectomy) and ovaries (oophorectomy).

Radical hysterectomy with bilateral salpingo-oophorectomy: The removal of the uterus, cervix, fallopian tubes, ovaries, the upper portion of the vagina and some surrounding tissue and lymph nodes. This type of hysterectomy is performed when cancer is involved.[3]

The evolution of surgical techniques has also led to different approaches to hysterectomy

- Abdominal Hysterectomy: The AH traditionally has been the surgical management for gynaecological malignancy, in the context of an enlarged uterus or when other pelvic pathology is present such as endometriosis or adhesions. It remains the 'fallback option' if the it (uterus) cannot be removed by another approach. Mini-Abdominal hysterectomy refers to an approach to hysterectomy where the abdominal incision does not exceed 7cm.
- Vaginal Hysterectomy: In this approach, the uterus is removed through the vagina, resulting in no visible abdominal scars. It is commonly used for uterine prolapse or smaller uterine sizes.
- Laparoscopic Hysterectomy: A minimally invasive approach where the uterus is removed using small incisions in the abdomen, aided by a camera. The proponents advocate that the main advantages are the possibility of diagnosing and treating other pelvic diseases such as endometriosis, the ability to secure thorough intraperitoneal haemostasis, or carrying out adnexal surgery including the removal of the ovaries, and a more rapid recovery time from surgery compared to AH.
- Robotic-Assisted Laparoscopic Hysterectomy: This is an advanced form of laparoscopic hysterectomy that has been performed since 1998, it uses robotic technology to provide the surgeon with enhanced precision and control.[4,5]

Many treatment options are available nowadays including medical and conservative surgical procedures but hysterectomy remains the most preferred method to manage gynaecological disorders.[6]

Common medical indications of hysterectomy include gynecological complaints such as fibroid, heavy menstrual bleeding, chronic pelvic pain, pelvic inflammatory disease, uterine prolapse, and cancer of the reproductive organs.[7]

Histopathological examination of surgical specimens carries legal, ethical, diagnostic and therapeutic significance. Numerous conditions in gynecological practice require removal of a uterus that may show no gross or microscopic pathology when examined by the pathologist. Removal of a normal uterus may be indicated and permitted in the treatment of adenxial and vaginal cancer, pelvic inflammatory disease, DUB, pelvic organ prolapse, endometriosis, pelvic pain and pelvic tuberculosis. The diagnostic significance of histopathological examination is well established in patients with genital cancer where adjuvant treatment is dependent upon grade and extent of invasion of disease.[8]

Although histopathology correlates well with clinic-radiological diagnosis, various lesions have been discovered on microscopy only. Adenomyosis remains the most commonly missed preoperative diagnosis and getting diagnosed on histopathological examination. Grossly unremarkable many specimens may reveal pathologies on histological examination. Similarly, many non-neoplastic lesions may show malignant foci on microscopy. On the other hand, many patients may be suspected of having a malignancy on preoperative examination e.g, patients with postmenopausal bleeding and histopathological examination may aid to rule out this suspicion. Hence, all hysterectomy specimens must undergo proper histopathological examination.[7]

1.2. AIM OF THE STUDY

Study the frequency of different pathologies of uterus and adenxia in hysterectomy specimens received at the histopathology department; Al-Imamain Al-Kadhimain Medical City, and associate the clinicopathological parameters (age, clinical presentation, type of hysterectomy and ultrasound diagnosis) with the final histopathological diagnosis of hysterectomy cases.

REVIEW OF LITERATURE

2.1. Normal histology of the female reproductive system

The female reproductive system comprises both external and internal genitalia, each playing distinct and essential roles in the reproductive process.

2.1.1. External Genitalia

2.1.1.1. Vulva

The vulva encompasses the external female genital structures, including the labia majora, labia minora, clitoris, vulvar vestibule, urethral meatus, and vaginal orifice. The labia majora lie lateral to the labia minora and converge anteriorly to form the mons pubis, a fatty layer covering the pubic symphysis. The vulvar vestibule, located medial to the labia minora, contains the openings of the urethra and vagina. The Bartholin glands are situated laterally to the vaginal orifice.[8]

2.1.2. Internal Genitalia

2.1.2.1. Vagina

The vagina is a fibromuscular canal consisting of four distinct layers

- Epithelial layer: The superficial layer is a nonkeratinized, stratified squamous epithelium. During the reproductive years, the epithelium becomes rich in glycogen under the influence of estrogen.
- Subepithelial layer (Lamina propria): This dense

connective tissue layer is predominantly composed of fibrillar collagens and elastin, with fibroblasts as the primary cellular component. It is vascularized bysmall arterioles andvenules.

- Muscularis: The muscularis layer comprises inner circular and outer longitudinal smooth muscle cells, surrounded by connective tissue.
- Adventitia: A loose connective tissue layer that delineates the muscularis from the surrounding paravaginal tissue.
- The lamina propria and muscularis are particularly significant, as they provide the vaginal wall with its notable tensile strength.[18]

2.1.2.2. Uterus

The uterus is a hollow, pear-shaped organ responsible for various functions, such as gestation (pregnancy), menstruation, labor and delivery, it is located in the female pelvis. The female uterus subdivides into four main anatomic segments (from superior to inferior): the fundus; a broad curved area in which Fallopian tubes connect to the uterus, the corpus (body); the main part of a uterus, and it starts directly below the level of fallopian tubes and continues downward, isthmus; a lower neck region of the uterus, and cervix; which extends downwards from the isthmus and opens in the vagina.[9]

The uterine cavity has a triangular shape and a length of approximately 6 cm. Histologically, it is lined by the endometrial mucosa, which constitutes the inner layer (endometrium) of the organ. It is surrounded by a thick muscular layer (myometrium) and a serosal covering, the latter extending to the point of peritoneal reflection.[13]

The endometrium is composed of two layers: the basalis (from which the endometrium regenerates after menstruation) and the functionalis. The functionalis may be differentiated into the superficial compacta and the underlying spongiosa; in the second half of the menstrual cycle, which extends to the basalis. The endometrium varies; during the menstrual cycle, from 1 mm (postmenstrual) to about 8 mm at the end of the third week. Every layer of the endometrium consists of two major components: the epithelial component, as glands or as superficial epithelium, and the mesenchymal component of stromal cells with pluripotential properties.[14] The myometrium consists of layers of longitudinal and circular smooth muscle cells; a layer of connective tissue containing blood vessels lies between the two smooth muscle layers. The inner smooth muscle layer borders the endometrial layer.[14]

2.1.2.3. Cervix

The cervix is a firm, cylindrical structure situated at the lower pole of the uterine corpus. The length of a normal adult non-pregnant cervix is approximately 25 mm. Outside of pregnancy the cervical canal is collapsed, firm, and fusiform in shape. It ensures communication between the cavity of the corpus and the lumen of the vagina, and is bounded by the internal and external os.[10]

The outer surface of the portio-vaginalis is the exocervix or ectocervix and is lined by squamous epithelium. The endocervical canal or anatomic endocervix is lined by columnar mucin-producing epithelium. The junction of the exocervical squamous epithelium and the endocervical columnar epithelium is the squamocolumnar junction.[15] The position junction is variable and changes with age and hormonal influence, but in general the junction moves upward into the endocervical canal with time. The replacement of the glandular epithelium by advancing squamous epithelium is a process called squamous metaplasia. The area of the cervix where the columnar epithelium coexists with the squamous epithelium is termed the "transformation zone."[16]

2.1.2.4. Ovaries

The ovary is the female gonad. It is a paired intraperitoneal endocrine organ each is approximately 2.0 cm in width, 3.5 cm in length and 1.0 cm in thickness; this is comparable to the size of a golf ball. The volume of the ovary changes as females age.[11]

On cut section, three ill-defined zones are discernible, including an outer cortex, an inner medulla, and the hilus. The stroma of the superficial cortex is typically more fibrotic than elsewhere, and although frequently referred to as the tunica albuginea, it lacks the densely collagenous, almost acellular appearance and sharp delineation of this layer in the testis. Follicular structures (cystic follicles, corpora lutea, corpora albicantia) are typically visible in the cortex and medulla.[17]

2.1.2.5. Fallopian tubes

The eponymous name—Fallopian tube—is named after Gabriel Fallopius: Italian anatomist. The uterine tubes are paired, tubular uterine appendages located bilaterally at the superior portion of the uterine cavity.[12]

Each tube is about 10 cm long. The fallopian tube wall is composed of three distinct layers: the outer most serosa; comprising a mesothelial cell layer overlying a thin, vascularized connective tissue, the intermediate muscularis (myosalpinx) contains smooth muscle bundles interspersed with interstitial connective tissue arranged in two main layers, an inner circular layer and an outer longitudinal layer. The innermost mucosa (endosalpinx), lined by columnar epithelium of four cellular types; (ciliated cells, secretory cells, the intercalary cells (peg cells) and the fourth types of cells are the small (indifferent cells) with large dark nuclei.[12]

Additionally, small quantities of lymphocytes infiltrate the lamina propria. Plasma cells, generally present in limited numbers, increase in response to infection.[12]

2.2.1. Handling of hysterectomy specimen in histopathology laboratory

2.2.1.1. General principle

The reason for the hysterectomy should be provided on the clinical request form and any relevant clinical information that may affect histological interpretation should be disclosed. Such information includes prior endometrial ablation, pre- operative treatment with hormones, tamoxifen or uterine embolisation, which can significantly alter the morphology of fibroids. History of rapid growth of a fibroid, especially in a post-menopausal woman, is important.

Hysteroscopic/transcervical endometrial resection also changes the appearances of the endometrium and myometrium and may be associated with uterine wall perforation. A patient's cervical screening history may be pertinent if the patient has had previous loop/LLETZ biopsies for CIN and/or if there is persisting abnormal cytology. Laparoscopic hysterectomies may be submitted as morcellated specimens. There should be a previous endometrial sample to exclude any endometrial abnormality.[19]

2.2.1.2. Submission of specimens

Specimens are commonly submitted fixed in 10% neutral buffered formalin. The uterine serosa acts as a barrier to fixative penetration; therefore, the hysterectomy specimen should be promptly sliced and immersed in an adequate volume of fixative to ensure appropriate preservation of the endometrium. The slicing technique, either sagittal or coronal, varies based on institutional preferences. Surgeons should be advised to slice the uterus themselves in cases where delays in laboratory submission are anticipated.[19]

2.2.1.3. Specimen handling (macroscopic description and grossing)

2.2.1.3.1. Orientation

The anterior and posterior uterine surfaces can be distinguished by examining the characteristics of the peritoneal reflection. The anterior reflection is generally rounded and membranous, positioned higher than the posterior reflection, which often exhibits a 'V' shape. The round ligament, located anteriorly on the uterine fundus, further aids orientation. When the adnexa are included, the round ligament lies anterior to the fallopian tube, which is anterior to the ovary.[20]

2.2.1.3.2. Weight and dimensional measurements

When the uterus is accompanied by the adnexa, these should be separated into right and left sides, and the uterus and cervix weighed together. If the cervix has been separately amputated but included within the same container, weigh it in combination with the uterus. In nulliparous women, the uterus typically weighs between 40 and 100 g, while in multiparous women, it averages 250 g. Enlarged or neoplastic ovaries should be weighed individually.[19,20]

The uterine corpus should be measured from cervix to fundus, cornu to cornu, and anterior to posterior. The average dimensions for nulliparous women are approximately 8 cm in length, 5 cm in width at the fundus, and 2.5 cm in thickness. In multiparous women, the dimensions range between 10–12 cm in length, 5–7 cm in width, and 2.5–3.5 cm in thickness. Measurements should include the cervix.[19,20]

2.2.1.3.3. External Features of the Uterine Corpus

Descriptions of the uterine serosa should include any notable characteristics, such as distortion by leiomyomata, adhesions, or focal hemorrhage (e.g., from endometriosis). Postpartum hysterectomy specimens may be enlarged, whereas specimens from uterine prolapse cases may appear small.[20]

For subtotal hysterectomy specimens, note the absence of the cervix. Total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO) specimens should include documentation of intact adnexa. Additionally, disruptions to fallopian tubes from prior tubal ligation and the presence of sterilization clips should be reported.[19]

2.2.1.3.4. Uterine Opening

The method for opening the uterus depends on pathologist preference and the presence of distortions such as fibroids. Some prefer sagittal plane cuts, while others opt for coronal cuts along lateral borders. Probing may be necessary for accurate orientation. The primary aim is to optimally expose the uterine cavity. The myometrium is examined via parasagittal or horizontal incisions.[19] For cases involving persistent cervical cytological abnormalities or suspected cervical lesions, the cervix may be amputated and dissected akin to loop electrosurgical excision procedure (LLETZ) or cone biopsy techniques.[19]

2.2.1.3.5. Internal Features of the Uterus

Upon opening, describe the exocervix (typically tan-white, smooth, and glistening) and the endocervical canal, noting dimensions, color, the presence of the normal herringbone pattern, and any lesions. The endometrium should be assessed for thickness and appearance, which is usually tan-pink or hemorrhagic and soft. Exophytic or polypoid lesions should be described in terms of number, location, dimensions, and parenchymal characteristics.[19,20]

The myometrium's maximum thickness and appearance (commonly tan-pink, soft, and trabeculated) should also be detailed, noting changes such as thickening, coarse trabeculation, or hemorrhagic cystic lesions indicative of adenomyosis.[20]

2.2.1.3.6. Sections for Histological Examination

Representative sections from the anterior and posterior cervical halves are essential for thorough examination. For the uterine corpus, at least two sections should

include the endometrium, a substantial portion of the myometrium, and serosa, when possible. Sections should also be taken from each fibroid up to a maximum of three, with priority given to areas appearing abnormal (e.g., necrotic or cystic). Cervical or endometrial polyps should be submitted entirely, except in cases of extremely large specimens.[20]

2.3. Indication of hysterectomy

2.3.1. Benign diseases

Hysterectomy is most often performed for benign diseases, which includes conditions such as prolapse, abnormal uterine bleeding, fibroids and pelvic pain.[22] This section will focus on abnormal uterine bleeding, fibroids and adenomyosis, which are recognized as the primary benign conditions necessitating hysterectomy.

2.3.1.1. Abnormal uterine bleeding

Abnormal uterine bleeding (AUB) is the main reason women are referred to gynecologists and accounts for two thirds of all hysterectomies. In premenopausal women, AUB is diagnosed when there is a substantial change in frequency, duration, or amount of bleeding during or between periods. In postmenopausal women, any vaginal bleeding 1 year after cessation of menses is considered abnormal and requires evaluation.[23] A normal menstrual cycle has a frequency of 24 to 38 days and lasts 2 to 7 days, with 5 to 80 milliliters of blood loss. Variations in any of these 4 parameters constitute abnormal uterine bleeding. Older terms such as oligomenorrhea, menorrhagia, and dysfunctional uterine bleeding should be discarded in favor of using simple terms to describe the nature of abnormal uterine bleeding.[25] Bleeding due to a wide variation of pathology both inside and outside the reproductive tract can be mimicked as an anovulatory bleeding. Therefore, it is essential to explore a detailed menstrual history appropriate for AUB followed by a pelvic examination, including a vaginal speculum examination, to differentiate between anovulatory bleeding and other causes of bleeding. In contrast, heavy menstrual bleeding (HMB)is referred to ovulatory bleeding more than eight days duration and is often associated with uterine fibroids, adenomyosis, a copper intrauterine device (IUD), or coagulative disorders.[26]

The PALM-COEIN classification is a system developed by the International Federation of Obstetrics and Gynecology (FIGO) to identify and categorize the specific underlying causes of abnormal uterine bleeding (AUB). The diagnosis of AUB can be made when conditions within the acronym PALM–COEIN are implicated—PALM (polyps, adenomyosis, leiomyoma, malignancy) and COEIN (coagulopathies, ovulatory dysfunction, endometrial, iatrogenic, not otherwise classified).[24]

In the treatment of women with abnormal uterine bleeding, once a thorough history, physical examination, and indicated imaging studies are performed and all significant structural causes are excluded, medical management is the first-line approach.[27] In patients where medical treatment or conservative surgery has failed to reduce and improve the bleeding, hysterectomy is associated with a high level of satisfaction.[28]

2.3.1.2. Uterine Leiomyoma

Human leiomyomata (fibroids) are benign smooth muscle tumors of the uterus, which represent the most common neoplasms in women of reproductive age, and have a lifetime incidence of approximately 70% in the general population.[29] Most patients with uterine fibroids are asymptomatic. Common symptoms associated with leiomyomas include menorrhagia, pelvic pain or pressure, and subfertility. These symptoms vary between patients and do not necessarily correlate with the size of the fibroids.[33] The development of leiomyomas depends on a complex interaction among female gonadal steroid estrogen and progesterone hormones, growth factors, cytokines, genetic predisposition, and somatic mutations. Although its etiology remains unclear, it is known that the loss of the myomatous cell growth regulation occurs mainly by the suppression of apoptosis-inducing protein B-cell lymphoma 2(bcl-2) and p27.[30] Cytogenetic alterations are common in uterine leiomyomas. The most consistent are rearrangements of 6p (involving the HMGA1 gene), del(7q), +12 (involving the HMGA2 gene), and t(12;14) (involving the HMGA2 gene). These have led to the discovery that disruptions or dysregulations of the high-mobility group proteins HMGA1 and HMGA2 contribute to the development of these tumors. Mutations in MED12 are present in most conventional leiomyomas but are less common in some leiomyoma variants.[36] The most significant non-modifiable risk factor is African descent, which leads to earlier diagnosis and more severe symptoms. Other risk factors include early menarche, nulliparity, obesity, and late entry into menopause, and a positive family history of uterine fibroids.[31]

On the other hand, protective factors for uterine fibroids include combined oral contraception or injectable medroxyprogesterone acetate in the depot form, smoking, women of low weight, and parity.[32]

The evaluation of fibroids is mainly based on the presenting symptoms of the patient. In the United States, the preferred initial imaging modality for fibroids is ultrasonograph. Transvaginal ultrasound is about 90-99% sensitive for detecting uterine fibroids, but it may miss small or subserosal fibroids. Adding hysteroscopy or sono-hysterography improves the sensitivity for detecting submucosal myomas. Without histopathologic evaluation, there are no reliable means to differentiate benign from malignant tumors. Some predictors of malignancy on MRI include age more than 45 years, intratumoral hemorrhage, endometrial thickening, T2-weighted signal heterogeneity, menopausal status, and non-myometrial origin.[34]

Grossly leiomyomas are well circumscribed solid, firm tumors usually solitary or may be multiple in 2/3 of cases. They are commonly located intramurally (within the myometrium) followed by subserosal (perimetrium) and submucosal location (beneath the endometrium). Also leiomyoma can occure in broad ligament, separate from uterus as well in the vicinity of uterus.[37]

Microscopic examination is quite characteristic with intersecting fascicles of uniform smooth muscle cells. Cells have indistinct borders and abundant fibrillar, eosinophilic cytoplasm. The nuclei are elongated and have finely dispersed chromatin. Degenerative or secondary changes such as hyaline degeneration, cystic degeneration, myxoid change, fatty change, calcification and metaplasia can be associated. After menopause or delivery, leiomyomas can undergo atrophy with significant shrinkage and fibrosis. Pregnancy, use of oral contraceptives and tumor vessel thrombosis is associated with red degeneration.[35,37]

There are different histological variants of leiomyomas in addition to the usual leiomyoma these includes:

- Cellular leiomyoma (formed of densely cellular fascicles of smooth muscle with little intervening collagen).
- Mitotically active leiomyomasis (they characterized by a mitotic activity higher than 5mitoses/high power field, no necrosis or cytological atypia), have the same clinical behavior as conventional leiomyoma.
- Atypical leiomyoma (consisting of atypical cells, clustered or distributed through the lesion).
- Epithelioid leiomyoma (composed of polygonal or round cells rather than spindle-shaped. This subtype includes, clear cell leiomyoma, lieomyoblastoma, and plexiform leiomyoma).
- Myxoid leiomyoma (consisting of abundant amorphous myxoid substance between the smooth muscle cells).
- Vascular leiomyoma (having dense proliferations of large caliber, thick-walled blood vessels).
- Lipoleiomyoma (a mixture of mature adipocytes and smooth muscle cells).
- Leiomyoma with tubules (composed tubular structures).

Hysterectomy serves as the only definitive treatment for this fibroid. However, indications for surgical intervention depend on a variety factors such as the severity of symptoms as well as the desire to maintain fertility. Therefore, it is essential to consult patients regarding their desire to preserve fertility. Alternative therapeutic approaches include uterine artery embolization (UAE) and myomectomy. Pharmacological treatments may also be employed as a preparatory step before surgical intervention. For instance, medications such as gonadotropin-releasing hormone (GnRH) analogs and selective progesterone receptor modulators (SPRMs), which counteract the effects of estrogen and progesterone, respectively, can be administered to reduce the size of these masses prior to surgery.[38]

2.3.1.3. Adenomyosis

The first description of the condition initially referred to as “adenomyoma” was provided in 1860 by the German pathologist Carl von Rokitansky, who found endometrial glands in the myometrium and subsequently referred to this finding as “cystosarcoma adenoids uterinum”. The modern definition of adenomyosis was provided in 1972 by Bird who stated: “Adenomyosis may be defined as the benign invasion of endometrium into the myometrium, producing a diffusely enlarged uterus which microscopically exhibits ectopic non-neoplastic, endometrial glands and stroma surrounded by the hypertrophic and hyperplastic myometrium.[39] While the histology of adenomyosis is well-established, but its etiology remains unclear. The most widely accepted theory suggests that adenomyosis arises from a disruption between the endometrium basalis and the myometrium, causing endometrial tissue to invade the myometrium, leading to angiogenesis and myometrial smooth muscle hypertrophy and hyperplasia. This theory is supported by the higher prevalence of adenomyosis after dilation and curettage or cesarean section. Another theory suggests that Mullerian stem cells undergo abnormal differentiation, forming ectopic endometrial tissue, supported by genetic marker alterations and cases in women with Rokitansky-Kuster- Hauser syndrome. Other theories involve altered lymphatic drainage or displaced bone marrow stem cells.[40] Patients with adenomyosis can be asymptomatic up to one third of cases. Symptoms typically arise between 40 and 50 years of age. There is no pathognomonic sign or symptom of adenomyosis. Common symptoms include dysmenorrhea and abnormal uterine bleeding. Less frequent symptoms include chronic pelvic pain and dysparunia.[51] Heavy bleeding occurs in 40%-60% of patients with adenomyosis. Heavy menstrual bleeding is likely caused by the increase in the total volume of the endometrium and endometrial glands as subsequence to the increase in the surface area of the endometrium, or the increased vascularization of the lining of the endometrium and is correlated directly with the extent of myometrial invasion. Adenomyosis is part of the

American College of Obstetricians and Gynecologists PALM-COEIN acronym for the evaluation of abnormal uterine bleeding in reproductive-aged patients.[42] Historically, the diagnosis of adenomyosis based on histological examination that required biopsy or more often hysterectomy. In the present, the primary means of making the diagnosis is imaging. MRI was the preferred modality; however, recent data has shown the transvaginal ultrasound to match the specificity and sensitivity of MRI (86% specificity, 89% sensitivity). MRI was reserved for equivocal cases and ultrasound becomes the preferred modality for the initial evaluation due to, the availability of ultrasound and the increased costs of MRI.[47]

Grossly, the uterus in severe cases of adenomyosis may appear slightly enlarged and globular due to myometrial hypertrophy, though it typically retains its overall shape and is rarely larger than a 12-week pregnant uterus. The cut surface of adenomyotic foci shows hyper-fasciculated and trabeculated myometrium, indicating myometrial hypertrophy. Unlike leiomyomas, adenomyosis lacks a well-defined border, and the foci may appear indistinct or as a white-gray mass with brown areas from hemolyzed blood and hemosiderin deposits. Blood-filled cysts can also be seen occasionally. In some cases, adenomyosis forms localized nodules, called adenomyomas, that can resemble leiomyomas.[44] These nodules consist of smooth muscle surrounding endometrial glands and stroma and are unencapsulated, with hypertrophied myometrium blending with the normal myometrium.[43] Depending on its distribution within the myometrium, adenomyosis has been described as both diffuse and focal. Diffuse adenomyosis is defined by the presence of multiple foci within the myometrium, while focal adenomyosis appears as isolated nodules of hypertrophic myometrium and ectopic endometrium.[44]

Numerous studies have explored the relationship between the severity of clinical symptoms and histopathological characteristics, particularly focusing on the depth of myometrial invasion and the extent of involvement, quantified by the number of foci and glands.[44] The early classification by Bird et al. 1972 used one low-power field (LPF) as the cut-off point for the diagnosis of adenomyosis and suggested three 'grades':

- Grade I (termed adenomyosis sub-basalis), in which glands are confined to one LPF below the basal endometrium;
- Grade II, when glands are present up to mid-myometrium;
- And grade III, in the presence of ectopic glands beyond mid myometrium.

Direct correlation between the severity of dysmenorrhea and the depth of penetration, to the extent that 4.3% of patients with Grade I adenomyosis reported having dysmenorrhea, compared to 42.4% with Grade II and 83.3% with Grade III.[44,45]

Adenomyosis can be involved by any of the diseases affecting the orthotopic endometrium, including hyperplasia and adenocarcinoma. It is important to recognize this phenomenon, lest a case of in situ or superficial endometrial adenocarcinoma associated with similar changes in the foci of adenomyosis be misinterpreted as a deeply invasive malignancy. Involvement of adenomyosis by endometrial adenocarcinoma does not seem to affect outcome.[46] Regarding treatment, the first consideration is the desire for fertility, which will guide treatment considerations. Hysterectomy is the definitive cure. The remaining options target the primary symptoms of heavy, painful menstrual bleeding while preserving the uterus.[40]

2.3.2. Pre invasive diseases

2.3.2.1. Endometrial hyperplasia

Endometrial hyperplasia (EH) is a pre-cancerous, non-physiological, non- invasive proliferation of the endometrium that results in increased volume of endometrial tissue with alterations of glandular architecture (shape and size) and endometrial gland to stroma ratio of greater than 1:1.[49]

Endometrial hyperplasia occures as a results from estrogenic stimulation of the endometrial tissue with a relative deficiency of progesterone's counterbalancing effects, often referred to in clinical practice as "unopposed", and is on a continuum with disordered proliferative endometrium. The typical causes for endogenous estrogen excess include anovulatory cycles (perimenopause, PCOS, obesity, and estrogen-secreting ovarian tumors). The exogenous causes include unopposed estrogen therapy, hormone replacement therapy, and tamoxifen (utilized in breast cancer treatment).[50]

The classification of endometrial hyperplasia has had multiple terminology. According to the WHO 94 classification, based on complexity of the glands and nuclear atypia, endometrial hyperplasia is divided into four groups: non-atypical endometrial hyperplasia (simple, complex) and atypical endometrial hyperplasia (simple, complex). The American College of Obstetricians and Gynecologists and the Society of Gynecological Oncology endorse the endometrial intraepithelial neoplasia (EIN) classification as superior to the WHO 94 system for endometrial hyperplasia histology, although the WHO system remains the most widely used in literature. The new WHO 2014 classification, approved by the International Society of Gynecological Pathologists, divided endometrial hyperplasia into two groups: benign endometrial hyperplasia and atypical hyperplasia/endometrial intraepithelial neoplasia (EIN). The WHO 2014 schema is more likely to successfully identify precancerous lesions than the previous classification of WHO 94.[53] The 2020 World Health Organization classification expanded on the diagnostic criteria in the two-tiered system to include essential and desirable criteria; the essential criteria for atypical hyperplasia or EIN include crowded glandular architecture and altered epithelial cytology distinct from the surrounding endometrium or entrapped nonneoplastic glands or both. Desirable criteria for atypical hyperplasia or EIN include the following: loss of immunoreactivity for PTEN, PAX2, or mismatch repair proteins.[54]

2.3.2.1.1. Endometrial hyperplasia without atypia (nonatypical hyperplasia) Microscopically, endometrial hyperplasia without atypia is characterized by glands reminiscent of normal proliferative endometrium lined by simple epithelium, but with increased crowding. The exact glands-to-stroma ratio required for diagnosing hyperplasia is controversial. Many pathologists use a 2:1

ratio as the diagnostic threshold, though some systems consider a glandular contribution above 55% (just over a 1:1 ratio) acceptable in the appropriate morphological context. Scattered mitoses and nuclear enlargement may be present, but prominent nucleoli should be absent. Uniformity throughout the proliferation is essential, as a distinct morphological subclone suggests atypical hyperplasia.[51] The risk of progression to carcinoma is less than 5% for non- atypical endometrial hyperplasia (NEH) and the chief differential diagnosis is atypical hyperplasia, and it is based on the assessment of the presence of cytological atypia.[52]

2.3.2.1.2. Endometrial hyperplasia with atypia/endometrial intraepithelial neoplasia (atypical hyperplasia AH/EIN).

Endometrial hyperplasia, in atypical forms, is the precursor lesion for endometrioid adenocarcinoma, the risk of progression to carcinoma is up to 30% in atypical hyperplasia (AH).[51] Morphologically, crowded glands where the gland-to-stroma ratio exceeds 1. Altered epithelial cytology (enlargement, pleomorphism and loss of polarity), distinct from surrounding endometrium and/or entrapped non-neoplastic glands. The lesion should be large enough to avoid artifacts, although no specific size cutoff is specified.[55] The distinction between a case of atypical hyperplasia with severe architectural and cytologic atypia and a well-differentiated adenocarcinoma can be very difficult. The presence of confluence of glands, papillary/villoglandular architecture are features supporting a diagnosis of well-differentiated adenocarcinoma. On biopsy or curettings it has been recommended that the diagnosis of "atypical hyperplasia, cannot exclude low-grade adenocarcinoma" be used for those cases where the features are intermediate between atypical hyperplasia and grade 1 endometrioid adenocarcinoma, the use of this terminology for hysterectomy specimens is not recommended.[56] A diagnosis of EIN carries a high risk of concomitant endometrial cancer or terminal progression to cancer in the absence of treatment. Hysterectomy remains the definitive and curative treatment for EIN; however, the desire to keep fertility-sparing, the obesity epidemic, the recognition of varying rates of malignant transformation, medical comorbidities, and an aging population all may contribute to employ conservative (nonsurgical) treatment modalities.[57] Conservative treatment consists of progestins and follow-up biopsies every 3-6 months. Eligibility criteria for conservative treatment may also be extended to early endometrial cancer (EEC), ie., endometrial cancer with endometrioid type, tumor grade 1, absence of lymphovascular space, myometrial or cervical invasion and absence of extrauterine metastases. Although several progestogens have been used for conservative treatment, levonorgestrel-releasing intrauterine system (LNG-IUS) seems to be the most effective one. (58) Three possible diagnostic results for the follow-up specimens: either1. There is no evidence of residual hyperplasia, 2. There is residual hyperplasia with treatment effects (metaplasia and decidual change), or 3. There is residual hyperplasia that shows no treatment effects, and may even show more severe atypia or progression to frank carcinoma, when compared to the pretreatment biopsy.[56]

2.3.2.1.3. Atypical mucinous glandular proliferation

The 2014 World Health Organization classification calls for endometrial mucinous proliferations that display "confluent or cribriform architecture with even minimal atypia" in sampling specimens to be classified as carcinoma, and others whose features are not diagnostic of carcinoma to be categorized as atypical mucinous glandular proliferations (AMGPs).[59] It is a premalignant condition and the likelihood of adenocarcinoma in the hysterectomy after a diagnosis of atypical mucinous glandular proliferation was 45% in a recent study.[56]

2.3.2.1.4. Papillary proliferation of the endometrium (PPE)

Papillary proliferation of the endometrium (PPE) without cytologic atypia is uncommon and has only been studied in detail by Lehman and Hart in 2001.[60] Based on the architectural complexity of the papillae and extent of proliferation, PPE subdivided into simple and complex groups. Simple PPE is featured with the primary simple papillae and is considered benign, while the complex PPE is highly related to the risk of concurrent/subsequent endometrial neoplasia.[61]

2.3.2.2. Cervical intraepithelial neoplasia

Cervical squamous intraepithelial neoplasia (CIN) in itself is not an indication for hysterectomy. Hysterectomy is indicated only if there are other gynaecologic conditions that on their own justify the operation. However, it is important to exclude invasive cervical cancer prior to hysterectomy. (48) CIN represents a spectrum of neoplastic changes of the squamous epithelium of the cervix that have been recognized as precursors of invasive squamous cell carcinoma. CIN is graded on a scale from I to III, which can also be expressed descriptively as mild, moderate, or severe dysplasia, or carcinoma in situ.[62] Amore recent approach based on the Bethesda system for cervical cytology distinguishes between two categories with distinctive biology: low- and high-grade squamous intraepithelial neoplasia (LSIL and HSIL, respectively). LSIL characterized by HPV- related cytological changes such as koilocytosis and proliferation of basal and parabasal cells with mild atypia and mitosis. In contrast, HSIL, consists of small to medium- sized atypical basal cells that may involves the entire thickness of the epithelium and often lacks the clearly visible HPV-related changes. If compared to WHO classification, CIN 1 related to LSIL, whereas CIN 2,3 related to HSIL.[68]

Cervical intraepithelial neoplasia (CIN) arises due to infection with human papillomavirus (HPV) in cervical cells. HPV infection can occur in sexually active women across all age groups but is more prevalent among

adolescents and women under 30 years of age. HPV 16 is the most oncogenic strain, contributing to 55–60% of cervical cancers globally, while HPV 18 ranks second, accounting for 10–15% of cases. Factors such as smoking, immunosuppression, or HIV infection likely contribute to the persistence of HPV and elevate the risk of developing CIN.[63] CIN is often detected by cytology ("Pap smear") performed during the screening programs that exist in most industrialized countries. In the case of cellular abnormalities suggesting a high-grade CIN, colposcopy is performed and eventually the final diagnosis will be made by histology of a colposcopically guided punch biopsy.[64] Histologically, judgment depends on the features concerned with differentiation, maturation and stratification of cells and nuclear abnormalities (enlarged nuclei, increased nuclear-cytoplasmic ratio, increased intensity of nuclear staining (hyperchromasia), nuclear polymorphism and variation in nuclear size (anisokaryosis).[65] In CIN 1, cellular maturation remains intact, with nuclear abnormalities and mitotic figures limited and confined to the lower third of the epithelium. HPV-related cytopathic changes may affect the entire epithelial thickness. CIN 2 shows more pronounced nuclear abnormalities than CIN 1, with dysplastic changes predominantly in the lower half or two-thirds of the epithelium and mitotic figures present in the lower layers. In CIN 3, differentiation and stratification are minimal or absent, with marked nuclear abnormalities and numerous, often atypical, mitotic figures throughout the epithelium.[65] Recognition of the disease in a precancerous state, close follow up, and treatment are essential in the prevention of cervical cancer. The screening process involves pap smear cytology of the cervix, along testing for human papillomavirus (HPV) in certain circumstances. The prognosis for CIN differs depending on the severity. With compliance with ASCCP guidelines, the risk for development of carcinoma is low. The risk of evident cervical cancer is significantly higher when a woman has missed screening for more than ten years.[63]

2.3.2.3. Adenocarcinoma in situ (AIS) of the cervix

Adenocarcinoma in situ (AIS) of the cervix is a premalignant precursor to cervical adenocarcinoma. The incidence of cervical AIS has increased over the past few decades, the mean age at diagnosis is 35–37 years, and the current incidence rate is approximately 6.6 per 100,000 persons, increasing to 11.2 per 100,000 persons at the peak age of 30–39 years. The average interval between a diagnosis of clinically detectable AIS and early invasive cancer is at least 5 years. Additionally, approximately 55% of patients with AIS have a coexisting squamous lesion.[66,67]

Adenocarcinoma in situ of the cervix (AIS) may affect a gland in a focal, multifocal, or diffuse manner. The cells exhibit crowding and pseudostratification, demonstrating a columnar shape and a significantly elevated nuclear-to-cytoplasmic ratio. The nuclei are elongated, irregular, and show coarse chromatin. While nucleoli are typically inconspicuous, they can occasionally be multiple. Mitotic figures, especially those located at the apical region, are common. Additionally, numerous apoptotic bodies are a characteristic feature. The cytoplasm is often scant and eosinophilic, with minimal to no mucin production, though, in rare cases, abundant mucin may be present.[68,69] The mucins secreted by the in situ malignant glands may be similar to those of the normal endocervical mucosa or may resemble the pattern of intestinal goblet cells. The latter, which is referred to as intestinal-type cervical adenocarcinoma in situ, shows consistent expression of CDX2.[70]

Lesser degree of glandular atypia compared to those observed in AIS have been recognized. Various terms have been suggested for these lesions, including glandular atypia, glandular dysplasia, atypical hyperplasia, and low-grade and high-grade cervical glandular intraepithelial neoplasia. The connection between glandular dysplasia and AIS remains unclear. HPV DNA has been found in glandular dysplasia at significantly lower rates than in squamous dysplasia. It is hypothesized that, similar to squamous lesions, glandular neoplasia progresses through distinct and reproducible stages as it acquires the genetic alterations characteristic of carcinoma, though this progression has yet to be confirmed.[69] Benign lesions may mimic adenocarcinoma in situ of endocervix, these includes; microglandular hyperplasia, tubal metaplasia, inflammatory changes and endometriosis. Immunohistochemistry can help in distinguishing AIS from its benign mimics. Endocervical adenocarcinoma in situ (AIS) is characterized by a combination of nuclear enlargement, hyperchromasia, crowding, atypia, mitotic activity, and cribriform growth. These features may also be observed in other conditions such as tubal metaplasia, microglandular hyperplasia, endometriosis, and inflammatory changes, but are less pronounced. Immunohistochemically, AIS is typically positive for MIB1, p16 (diffuse and strong), and monoclonal CEA, while it is negative for estrogen receptor (ER), progesterone receptor (PR), vimentin, and BCL-2. In contrast, tubal metaplasia and endometriosis display cytoplasmic BCL-2 without significant MIB1 or CEA increase, and p16 staining is weak or focal. Microglandular hyperplasia similarly lacks the elevated expression of MIB1, p16, and CEA.[71]

Stratified mucin-producing intraepithelial lesion (SMILE) is a rare, high-grade cervical lesion classified as a variant of AIS in the WHO system. SMILE is often found in conjunction with conventional high-grade squamous intraepithelial lesions (HSIL), AIS, or both, and may include an invasive component such as squamous carcinoma, adenocarcinoma, or adenosquamous carcinoma. Recent studies suggest that SMILE could be a precursor to invasive stratified mucin-producing carcinoma. Similar to SIL and AIS, SMILE is strongly associated with persistent infection by high-risk HPV genotypes, evidenced by diffuse nuclear and

cytoplasmic p16 positivity, although HPV genotyping remains underreported in this context.[72]

Hysterectomy is the standard treatment for AIS, but fertility-preserving options are available for select patients with clear margins and negative endocervical curettage (ECC) after conization. In cases where margins are positive or ECC results are abnormal, the risk of residual or recurrent AIS and invasive adenocarcinoma is elevated, warranting repeat surgery. For patients who do not undergo hysterectomy, long-term monitoring is essential to ensure early detection of any recurrence.[73]

2.3.3. Invasive disease

This section reviews the indications for hysterectomy in the context of invasive malignant conditions. Although malignant adnexal tumors may also warrant hysterectomy, this topic is excluded due to the extensive scope required for a comprehensive literature review, which exceeds the limitations of this discussion. The focus is directed toward endometrial and cervical malignancies, examining the specific pathologies and clinical criteria that make hysterectomy a necessary intervention in these cases.

2.3.3.1. Endometrial carcinoma

Worldwide, endometrial cancer (EC) ranks seventh among all female cancers with the majority of cases occurring between 65 and 75 years of age. In Europe, uterine cancer ranks fourth among female neoplasms, with an incidence of 12.9- 20.2:100 000 and a low mortality rate: 2.0-2.7:100 000. This discrepancy is due to the fact that 80% of ECs are confined to the uterus at diagnosis and present with postmenopausal bleeding, which leads to prompt detection.[74] Historically, endometrial carcinoma has been classified into type 1 and type 2 based on histological features. However, recent studies have started classifying endometrial cancers according to a current molecular subclassification system. Type 1 endometrial cancers are the most prevalent, accounting for 80% of all endometrial cancers and are typically of endometrioid origin. Type 2 endometrial cancers, on the other hand, are predominantly of serous or clear cell origin.[75]

2.3.3.1.1. Pathogenesis and risk factors

The risk factors associated with the more prevalent type I endometrial cancer are predominantly linked to estrogen-stimulated endometrial proliferation. These factors include increased body mass index (BMI), estrogen replacement therapy, estrogen-secreting tumors, chronic anovulation, tamoxifen therapy, early menarche, and late menopause, all of which contribute to extended lifetime exposure to endogenous or exogenous estrogens.[76] Furthermore, Lynch syndrome accounts for 3–5% of endometrial cancer cases. Women with Lynch syndrome face elevated lifetime risks of various malignancies, including gastric, colorectal, ovarian, and endometrial cancers. This syndrome arises from genetic variations in the MLH1, MLH3, MSH2, MSH6, PMS2 genes. Additionally, Cowden syndrome significantly heightens the risk of endometrial cancer. Cowden syndrome is a hereditary cancer predisposition disorder caused by pathogenic variants in the PTEN gene, and it predisposes individuals to breast, thyroid, renal, colorectal, and endometrial cancers, as well as malignant melanoma.[79]

Conversely, type II non-endometrioid cancers are associated with distinct risk factors, such as lower BMI, parity, Black race, a history of breast cancer, and diagnosis after the age of 55.[76,77] Endometrioid carcinoma, the archetypal type I carcinoma, appears to progress through an estrogen-dependent "adenoma-carcinoma" pathway originating from atypical endometrial hyperplasia or endometrioid intraepithelial neoplasia (AEH/EIN). These carcinomas are predominantly low-grade and exhibit favorable prognoses, although a subset progresses to high-grade carcinoma characterized by receptor expression loss, TP53 mutations, and aggressive clinical behavior. Frequently altered genes in type I carcinomas include K-Ras, PTEN, and β-catenin. Another notable feature is microsatellite instability, commonly attributed to MLH1 promoter methylation.[78]

In contrast, serous carcinoma, the prototypical type II carcinoma, is not estrogen- dependent and typically arises in small uteri with atrophic endometria. It is frequently associated with a precursor lesion, serous endometrial intraepithelial carcinoma (SEIC). The molecular pathogenesis of serous carcinoma is driven by TP53 mutations, which are also detected in SEIC. Additional molecular alterations, identified through immunohistochemistry, include changes in cyclin E and p16.[78] Moreover, BRCA1 mutation carriers may have an increased susceptibility to serous endometrial carcinoma.[79]

2.3.3.1.2. Endometrial cancer grading

The World Health Organization and the International Federation of Gynecology and Obstetrics grade endometrial cancers depending on the percent of non-squamous solid architecture and nuclear features. Grade 1 tumors display ≤5% solid non glandular, non squamous growth; grade 2 tumors range from 6% to 50%; and grade 3 tumors exceed 50%. Marked cytologic atypia elevates the tumor to grade 1. Given the close relationship between mucinous adenocarcinomas and endometrioid carcinomas of the endometrium, applying the FIGO grading system to mucinous adenocarcinomas is appropriate. However, FIGO grading should not be applied when endometrioid or mucinous differentiation is uncertain or unconfirmed. Other types of endometrial tumors inherently possess a specific grade, with serous, clear cell, undifferentiated carcinomas, and carcinosarcomas classified as high grade.[79]

2.3.3.1.3. Molecular and histological classifications

Endometrial carcinoma (EC) has traditionally been categorized into two main subtypes—type I and type

II—based on histological features, tumor grade, and expression of hormone receptors (estrogen and progesterone receptors, ER and PR). Type I EC, the more prevalent subtype, is characterized by low-grade, endometrioid morphology, diploid karyotype, hormone receptor positivity, and a favorable prognosis, with a 5-year overall survival (OS) rate of approximately 85%. In contrast, type II EC is non-endometrioid, high-grade, aneuploid, TP53- mutated, hormone receptor-negative, and linked to a higher likelihood of metastasis and a poorer prognosis, with a 5-year OS rate around 55%. These subtypes also exhibit markedly distinct mutational profiles. The histopathological classification of endometrial cancer (EC) is challenging, even for experienced gynecopathologists, leading to frequent disagreements. This lack of consensus contributes to inaccuracies in EC risk assessment, which may result in patients receiving either excessive or insufficient treatment. In this context, the molecular classification of EC provides a prognostically relevant classification system; therefore, it should be integrated in the diagnostic procedures of EC.[81]

2.3.3.1.4. Molecular classification and morpho-molecular correlate

The classification system introduced by The Cancer Genome Atlas (TCGA), which employs molecular stratification to categorize endometrial cancer into four distinct subtypes, represents one of the most widely adopted frameworks in contemporary research. These subtypes include ultramutated POLE, hypermutated microsatellite instability (MSI), somatic copy-number alteration high, and copy-number low. The World Health Organization (WHO) designates these categories as POLEmut, MMRd, p53abn, and no specific molecular profile (NSMP), respectively, recommending the integration of molecular classification with immunohistochemical staining for p53 and mismatch repair (MMR) proteins (MLH1, PMS2, MSH2, MSH6) to assess prognosis.[75]

The POLEmut subtype is characterized by a high mutational burden in the exonuclease domain of the POLE gene and is associated with an excellent prognosis.[75] Morphologically, specific features have been identified in POLEmut endometrial cancer (EC). Approximately two-thirds of cases exhibit at least 50% solid growth (commonly classified as FIGO grade 3). The glandular component, if present, typically consists of glands with smooth luminal borders lacking hob-nailing. Additional features include scattered hyperchromatic, multinucleated tumor giant cells within solid tumor sheets, a dense peri-tumoral and intra-epithelial lymphocytic infiltrate, and, more recently, numerous tertiary lymphoid structures (TLS) within the myometrial wall.[75,82]

The microsatellite instability subtype (MMRd EC) is characterized by impaired downregulation of MLH1 or mutations in MMR genes and generally exhibits an intermediate prognosis. Morphologically, MMRd EC often shows abundant stromal and intra-epithelial lymphocytes and may contain TLS. The predominant histological type within this group is endometrioid EC; however, other non- endometrioid subtypes have also been documented. Additionally, lymphovascular space invasion (LVSI) has frequently been observed in association with MMRd EC for reasons that remain unclear.[75,82]

Tumors with numerous copy-number alterations, typically associated with TP53 mutations and positive p53 immunohistochemical staining, form the p53abn group. This category includes most serous carcinomas, which have a poor prognosis and exhibit classical serous histological features such as micro- papillary or pseudo-glandular architecture, strong cytological atypia, and high mitotic activity. However, the p53abn subgroup encompasses a broader range of histologies, including uterine carcinosarcomas, clear cell carcinomas, and FIGO grade 3 endometrioid carcinomas. Histological features that distinguish p53abn EC from MMRd and POLEmut EC include a lower abundance of tumor-infiltrating lymphocytes (TILs) and the absence of TLS.[75,82]

The no specific molecular profile (NSMP) subtype, also referred to as copy- number low or p53 wild-type, predominantly comprises endometrioid carcinomas and typically demonstrates an intermediate prognosis influenced by estrogen and progesterone receptor status. These tumors frequently show a predominant glandular proliferation with smooth luminal borders and nuclei exhibiting mild to moderate atypia (FIGO grades 1 and 2). Approximately 20% of low-grade NSMP EC cases present with a specific invasion pattern known as “microcystic elongated and fragmented” (MELF) invasion, which is rarely observed in other subtypes. Compared to MMRd and POLEmut EC, NSMP EC generally has a lower abundance of TILs and TLS.[75,82,80]

2.3.3.1.5. Endometrial carcinoma histotypes and their histochemical profile Numerous morphologic forms of endometrial adenocarcinoma have been described, these includes

- Endometriod endometrial carcinoma (ECC), it is the most common type of endometrial carcinoma. The microscopic appearance of the tumor resembles that of the proliferative endometrium, with a variable degree of glandular complexity and cellular pleomorphism. Several subtypes have been described, including the presence of squamous differentiation, villoglandular pattern, secretory features and ciliated cells. Recently recognized subtypes are the tumors that arise in the setting of hereditary nonpolyposis colon cancer syndrome, tumors with small non villous papillae, presence of microglandular pattern, sertoliform features, and dedifferentiated carcinomas.[83] Low-grade EECs demonstrate strong and diffuse positivity for ER/PR,

negative to patchy staining for p16, and usually P53 wild-type staining pattern. High-grade EECs (FIGO 3) may have different mutational and immunohistochemical profiles compared with low-grade EECs. The ER/PR have variable staining pattern in high-grade EECs.

p53 have aberrant expression is 20% to 45% of high-grade EECs. Close to 60% of FIGO 3 cases show abnormal MMR loss. Greater than 60% of FIGO 3 EECs also show loss of PTEN or ARID1A expression.[84]

- Endometrial serous carcinoma (ESC), commonly arises on the surface of endometrial polyps in the background of an atrophic endometrium. ESC is microscopically characterized by (1) papillae with or without a fibrovascular core, (2) marked nuclear atypia, (3) slit-like spaces, (4) solid growth, (5) scant cytoplasm (but in few cases, it can be abundant with eosinophilia or clearing), and (6) numerous mitotic figures (in most cases). Additionally, gland-like spaces, cilia, and psammoma bodies might be observed in up to 30–40% of patients.[85] In contrast to endometrioid carcinoma, 90% to 100% of endometrial serous carcinomas (ESCs) harbor a TP53 mutation. In clinical practice, p53 immunohistochemistry serves as a reliable surrogate for TP53 mutation, with three distinct patterns of aberrant p53 staining: overexpression (strong nuclear staining in at least 75% of tumor cells), null pattern (absence of staining in all tumor cells), and cytoplasmic staining. Approximately 90% of ESCs exhibit diffuse p16 positivity. ESCs typically do not show abnormal expression of mismatch repair proteins (MMRs), and the expression of estrogen receptor (ER) and progesterone receptor (PR) in ESCs is variable. Loss of PTEN and ARIDIA expression is infrequent in ESCs.[84]
- Clear cell carcinoma (CCC) of the uterus is a rare but aggressive subtype, comprising less than 5% of all uterine carcinomas. Microscopically, CCC is characterized by clear, often eosinophilic hobnail cells with various architectural patterns, including solid, papillary, and tubulocystic structures. Similar histological features are observed when CCC arises in the ovary, cervix, and vagina. Recent studies have identified atypical glandular changes—such as isolated glands or surface epithelium exhibiting cytoplasmic clarity and/or eosinophilia, accompanied by varying degrees of nuclear atypia—in the endometrium adjacent to CCC, suggesting that these lesions may represent the earliest morphologic manifestations of the tumor.

[86]CCCs are characterized by immunoreactivity for hepatocyte nuclear factor 1β (HNF-1β), napsin A, and α-methylacyl CoA racemase (AMACR), with negative staining for ER and PR. Notably, aberrant p53 expression is observed in one-third of CCCs, and patients with p53-mutated CCCs tend to experience a more aggressive clinical course compared to those without the mutation.[84]

- **Uterine carcinosarcoma, (UCS), also known as malignant mixed**

Müllerian tumor, is a rare gynecological malignancy characterized by poor prognosis. This "biphasic" neoplasm presents an admixture of epithelial and mesenchymal/sarcomatoid component.[87] The epithelial component is the predominant element in endometrial carcinosarcoma, typically presenting as a high-grade histotype, such as serous, endometrioid, clear cell, mixed, or undifferentiated carcinoma. The sarcomatous component can either be homologous (e.g., leiomyosarcoma, fibrosarcoma, endometrial stromal sarcoma) or heterologous (e.g., rhabdomyosarcoma, chondrosarcoma, osteosarcoma), depending on whether the mesenchymal component resembles uterine tissues. Heterologous differentiation is observed in approximately 40% of endometrial carcinosarcoma cases and is associated with poorer survival outcomes compared to homologous differentiation. Furthermore, sarcomatous dominance (i.e., >50%) is observed in 40% of carcinosarcoma cases and is strongly correlated with heterologous differentiation and a decreased survival rate. The combination of high-grade carcinoma with heterologous sarcomatous differentiation and dominance is associated with the most unfavorable prognosis.[88] Studies have also shown that the behaviors of these tumors are determined by the epithelial component. Epithelial elements invade the lymphatic and vascular spaces and metastasize, whereas the spindle cell component has a very limited metastatic potential.[89] Immunohistochemistry is not essential for diagnosing uterine carcinosarcoma but can be useful for identifying heterologous differentiation, such as skeletal muscle. Carcinomatous component; typically positive for PAX8, EMA, and cytokeratin. Serous and high-grade endometrioid carcinomas may display aberrant p53 expression, while endometrioid elements can also be ER and PR positive. Sarcomatous component: Commonly shows abnormal p53 expression. Rhabdomyosarcoma exhibits desmin and myogenin positivity, whereas liposarcoma and chondrosarcoma are positive for S100.[90]

- Undifferentiated carcinoma (UC) of the endometrium is a highly aggressive subtype, accounting for approximately 2% of all endometrial carcinomas. Histologically, UC is characterized by sheet-like growths of discohesive cells that are monotonous, round or polygonal in shape, with scant cytoplasm, large vesicular nuclei, prominent nucleoli, and dense chromatin. There is an absence of lineage differentiation, with the growth appearing solid without any distinct pattern or glandular formation. These tumors often exhibit notable stromal infiltration by lymphocytes and a high mitotic activity. Additionally, tumor necrosis is frequently observed.[84]
- Dedifferentiated carcinoma of endometrium is

defined by the presence of 2 distinct carcinoma components: well-differentiated (FIGO 1 or 2) endometrioid adenocarcinoma and UC. The former is usually a mucosal lesion, whereas the latter is often deeply myoinvasive. The 2 components can vary in proportions, and the interface between the 2 can be abrupt or admixed. Irrespective of the amount of the undifferentiated component, DCs are far more aggressive than FIGO 2 endometrioid carcinomas. The undifferentiated component generally does not stain for epithelial markers (eg, cytokeratin [CK] AE1/AE3, CAM 5.1), E-cadherin, and gynecologic markers (eg, PAX8, ER, PR), in stark contrast to diffuse positivity of these markers in the endometrioid component. EMA and CK8/18 can be focally positive in the undifferentiated tumor cells.[84]

2.3.3.1.6. Anatomic spread and staging of endometrial carcinoma Endometrial carcinoma typically starts as a preinvasive lesion that progresses to invade the endometrial stroma and penetrates deeper into the myometrium. The cancer can spread through lymphatic capillaries to regional lymph nodes and may metastasize via vascular channels. It often spreads to the uterine cervix and stroma through lymphatics, especially in dedifferentiated carcinomas, but cancers near the lower uterine segment can spread along the surface as well. Malignant cells can also reach the fallopian tubes and ovaries through lymphatics that follow veins, leading endometrial cancers to primarily affect pelvic and para-ureteral lymph nodes. Tumors located in the uterine fundus or near adnexal organs tend to involve para-aortic and para-caval nodes. Advanced cancers may extend beyond the uterus, affecting the peritoneum, pelvic tissues, and nearby organs.[75] Staging of endometrial cancer is based on non-aggressive versus aggressive histology; extent of spread, including invasion depth, extension to surrounding structures, and extrauterine or lymph node metastases; lymphovascular space invasion; and molecular classification. The revised FIGO staging system significantly clarifies the characteristics of tumors at each stage and integrates molecular analysis data from the Cancer Genome Atlas, which are associated with prognosis and treatment decisions.[75,91] Stage I refers to endometrial carcinoma (EC) confined to the uterine corpus, further subdivided into stage IA (no or less than 50% myometrial invasion) and stage IB (myometrial invasion of 50% or more). Tumors that invade the cervical stroma but do not extend beyond the uterus are classified as stage II. Stage III encompasses tumors that have spread beyond the uterus but remain within the true pelvis. This stage is further divided into stage IIIA (invasion of the uterine serosa and/or adnexa), stage IIIB (parametrial and/or vaginal involvement), and stage IIIC1 (positive pelvic lymph nodes) or IIIC2 (positive paraaortic lymph nodes). Stage IVA includes tumors extending to the bladder or bowel, while stage IVB is characterized by distant metastases.[92] Table (2.1)

Micrometastasis indicate metastatic involvement. Macrometastasis defined by FIGO as lesions greater than 2 mm in size, while micrometastasis are 0.2 to 2 mm greater than 200 cells. Isolated tumor cells are defined as lesions greater than or equal to 0.2 mm and less than or equal to 200 cells. (75) LVSI as defined as extensive/substantial, ≥ 5 vessels involved.[92]

Table (2.1): International federation of gynecology and obstetrics endometrial cancer 2023 staging.[75]

Stage		Tumor Characteristics
I		Confined to the uterine corpus and ovary
	IA	Disease limited to the endometrium OR nonaggressive histological type (ie, low- grade endometroid, with the invasion of <50% of the myometrium with no or focal lymphovascular invasion OR good prognosis disease) IA1: Nonaggressive histological type limited to an endometrial polyp OR confined to the endometrium IA2: Nonaggressive histological types involving <50% of the myometrium with no or focal lymphovascular invasion IA3: Low-grade endometrioid carcinomas limited to the uterus and ovary
	IB	Nonaggressive histological types with invasion of ≥50% of the myometrium with no or focal lymphovascular invasion
	IC	Aggressive histological types are limited to apolyp or confined to the endometrium
II		Invasion of cervical stroma without extrauterine extension OR with substantial lymphovascular invasion OR aggressive histological types with myometrial invasion
	IIA	Invasion of the cervical stroma of nonaggressive histological types
	IIB	Substantial lymphovascular invasion of nonaggressive histological types
	IIC	Aggressive histological types with any myometrial involvement
III		Local or regional spread of the tumor of any histological subtype
	IIIA	Invasion of uterine serosa, adnexa, or both by direct extension or metastasis IIIA1: Spread to an ovary or fallopian tube, except when meeting stage IA3 criteria IIIA2: Involvement of uterine subserosa or spread through the uterine serosa
	IIIB	Metastasis or direct spread to the vagina or the parametria or pelvic peritoneum IIIB1: Metastasis or direct spread to the vagina or the parametria

		IIIB2: Metastasis to the pelvic peritoneum
	IIIC	Metastasis to the pelvic or para-aortic lymph nodes or both IIIC1: Metastasis to the pelvic lymph nodes IIIC1i: Micrometastasis IIIC1ii: Macrometastasis IIIC2: Metastasis to para-aortic lymph nodes up to the renal vessels, with or without metastasis to the pelvic lymph nodes IIIC2i: Micrometastasis IIIC2ii: Macrometastasis
IV		Spread to the bladder mucosa or intestinal mucosa or distance metastasis
	IVA	Invasion of the bladder mucosa or the intestinal and bowel mucosa
	IVB	Abdominal peritoneal metastasis beyond the pelvis
	IVC	Distant metastasis, including metastasis to any extra- or intra-abdominal lymph nodes above the renal vessels, lungs, liver, brain, or bone.

Integrating molecular subtype into staging criteria enhances prognostic accuracy for endometrial cancer. Comprehensive molecular classification, including POLEmut, MMRd, NSMP, and p53abn subtypes, is recommended for all cases to support risk-group stratification and inform treatment decisions. Molecular subtype can be determined from a biopsy and does not need to be repeated on the hysterectomy specimen. Once classified, the molecular subtype should be documented at all stages of disease.[93]

- FIGO Stages I and II are determined based on surgical/anatomical and histological findings. If molecular classification identifies POLEmut or p53abn status, the FIGO stage is adjusted in the early stages of the disease. This modification is represented by adding "m" for molecular classification, with a subscript to specify the POLEmut or p53abn status, as outlined below. MMRd or NSMP status does not alter the early FIGO stages, although these molecular classifications should still be documented for data collection purposes. When molecular classification reveals MMRd or NSMP, it should be recorded as Stage ImMMRd or Stage ImNSMP and Stage IImMMRd or Stage IImNSMP.[93]
- FIGO Stages III and IV are based on surgical/anatomical findings. Molecular classification does not modify the stage category; however, it should be recorded if known. When molecular classification is available, it should be documented as Stage IIIm or Stage IVm, with the appropriate subscript for data collection. For instance, when molecular classification indicates p53abn, it should be recorded as Stage IIImp53abn or Stage IVmp53abn.[93]

Table (2.2): FIGO endometrial cancer stage with molecular classification.[93]

Stage designation	Molecular findings in patients with early endometrial cancer (Stages I and II after surgical staging)
Stage IAmPOLEmut	POLEmut endometrial carcinoma, confined to the uterine corpus or with cervical extension, regardless of the degree of LVSI or histological type
Stage IICmp53abn	p53abn endometrial carcinoma confined to the uterine corpus with any myometrial invasion, with or without cervical invasion, and regardless of the degree of LVSI or histological type

2.3.3.1.7. Evaluation and prognosis

The diagnosis of endometrial cancer is established through histological evaluation, with additional laboratory and imaging tests conducted to exclude other potential diagnoses and assist in preoperative staging. For postmenopausal women, transvaginal ultrasound or endometrial sampling is typically used as an initial diagnostic test for endometrial cancer. However, in premenopausal women, transvaginal ultrasound is not recommended for diagnosis, as no specific endometrial thickness thresholds have been identified that correlate with an increased risk of carcinoma, unlike in postmenopausal women.[75] The management of endometrial cancer should be personalized, taking into account the patient's specific characteristics and the features of the endometrial neoplasm, including cancer stage, histologic subtype, age, comorbidities, and the desire for future fertility. This approach should involve discussions between the patient and a multidisciplinary team, including gynecologic oncologists, medical oncologists, radiation oncologists, and pathologists. These factors also help assess the patient's overall disease risk, which informs management decisions. The Gynecologic Oncology Group (GOG) applies specific criteria to classify disease risk.[75]

- Low-risk disease: MMRd or NSMP positive stage 1A tumors without any or only focal lymphovascular invasion or POLEmut stage 2 disease or less.
- Intermediate-risk disease: Any evidence of myometrial invasion, MMRd or NSMP positive stage 2 disease or less, or grade 2 or 3 disease.
- High-intermediate risk: The GOG and the Postoperative Radiation Therapy in Endometrial

Cancer (PORTEC) groups characterize this group differently.

- PORTEC classifies individuals with 2 of the following factors within this category:

1. Age 60 or older, Grade 3 disease.
2. Myometrial invasion greater than or equal to 50%.

- GOG divides individuals by age and risk factors consisting of grade 2 to 3 with MMRd or NSMP subtyping, lymphovascular space invasion, and invasion to the outer one-third of the myometrium into the following subgroups:
  - Age 70 and older with at least 1 risk factor.
  - Age 50 and older with ≥2 risk factors.
  - Younger than 50 with all 3 risk factors.
- High-risk disease: Stage 3 disease or clear cell and serous carcinoma tumors of any stage; invasive, high-grade stage 1 endometrioid carcinoma is placed in this group by some experts also.[94,75]

The 5-year survival rate for patients with stage 1 endometrial cancer exceeds 95%, with over 80% of cases diagnosed at an early stage. While the overall prognosis for endometrial cancer is favorable, certain molecular subtypes demonstrate more favorable survival outcomes. Patients with POLE molecular subtype endometrial cancer have a 5-year relapse-free survival rate of 98%, whereas those with the p53abn subtype, which is associated with the highest metastatic risk, have a survival rate of 46.6%. The 5-year relapse-free survival rates for MMRd and NSMP subtypes are 77.1% and 74.4%, respectively. However, women with metastatic or recurrent disease typically experience poor outcomes, with an overall survival rate of approximately 15 months following chemotherapy, according to a 2020 study.[75]

2.3.3.2. Cervical cancer

Cervical cancer is the third most common cancer in women worldwide. With continuing improvement in screening methods and vaccination programs in developed countries, the disparity of burden between women in developed countries and women in resource-poor settings becomes even more profound. Currently, >85% of cervical cancer deaths occur in low and middle-income countries. Tragically, cervical cancer is the leading cause of cancer deaths in women of the developing world.[96] Most cervical cancer cases are linked to human papillomavirus (HPV), with HPV DNA found in about 95% of malignant lesions. Persistent infection can lead to premalignant conditions like cervical intraepithelial neoplasia or adenocarcinoma in situ, progressing to invasive carcinoma over years or decades.[95] However, in roughly 10% of cases, this progression may occur in less than a year. Adenocarcinoma in situ is harder to detect with Papanicolaou testing, possibly contributing to its rising incidence.(95)Persistent infection and malignant transformation risk may increase due to factors like smoking, prolonged oral contraceptive use, high parity, and coinfection with herpes simplex virus type 2 or HIV. HPV serotypes 16 and 18 account for around 70% of cases, with the most common HPV types in cervical cancer cases being 16, 18, 45, 31, 33, 52, 58.[35,95] Cervical cancer is classified based solely on histological findings, with no molecular subtyping currently established. Previous studies have identified frequent mutations in genes such as SHKBP1, ERBB3, CASP8, HLA-A, TGFBR2, PIK3CA, EP300, FBXW, HLA-B, PTEN, NFE2L2, ARID1A, KRAS, and MAPK1 in cervical cancer. Recent research into molecular subgroups of cervical cancer has revealed three mRNA-based clusters: high expression of keratin gene family members, low expression of keratin gene family members, and an adenocarcinoma-rich cluster. The keratin-high cluster does not show KRAS mutations, while the adenocarcinoma- rich cluster lacks mutations in the HLA-A gene. A subgroup of endometrial-like cervical cancers has also been identified, characterized by high frequencies of KRAS, ARID1A, and PTEN mutations. However, no associations with clinical outcomes have been established for these molecular groupings.[99] Squamous cell carcinoma and adenocarcinoma are the predominant histological subtypes of cervical cancer, with squamous cell carcinoma being much more common. Adenocarcinoma accounts for approximately 5% of invasive cervical cancers globally, though its incidence is rising in certain regions. Both subtypes develop from precursor lesions, cervical intraepithelial neoplasia (CIN), or carcinoma in situ (CIS). Squamous CIS and adenocarcinoma in situ (AIS) are the primary precursors to invasive cervical cancer.[97] Differentiating adenocarcinoma of the cervix from endometrial adenocarcinoma requires immunohistochemistry and HPV in situ hybridization.[97] Most cervical malignancies originate from the squamocolumnar junction. Microscopically, these tumors exhibit anastomosing irregular nests or single tumor cells with associated stromal inflammation or desmoplasia, and lymphovascular invasion (LVI) may be observed. Grading is based on nuclear pleomorphism, nucleolar size, mitotic activity, and necrosis, though it does not correlate with prognosis.[95,97]

Invasive cervical cancer spreads directly to the parametrium, vagina, uterus, bladder, and rectum, and through lymphatic route to regional lymph nodes (obturator, external, and internal iliac, then common iliac LNs and para-aortic). Distant metastasis to the lungs, liver, and bones occurs later via the hematogenous spread.[98]

In 2018, the FIGO Gynecologic Oncology Committee revised the staging to allow the option of clinical, radiological, or pathological findings, as available, to assign the stage. A corrigendum to this staging was published thereafter, with some modifications. The revised staging is shown in Table (2.3) the main changes are:[99]

- The horizontal dimension of a microinvasive lesion is no longer considered.
- Tumor size has been stratified further into three

subgroups: IB1 ≤2 cm, IB2>2–≤4 cm, and IB3 >4 cm.

- Lymph node positivity, which correlates with poorer oncologic outcomes assigns the case to Stage IIIC—pelvic nodes IIIC1 and para-aortic nodes IIIC2. Micrometastases are included in Stage IIIC.[99]

Table 2.3: FIGO staging of cancer of the cervix uteri (2018).[99]

Stage	Description
I	The carcinoma is strictly confined to the cervix (extension to the uterine corpus should be disregarded)
IA	Invasive carcinoma that can be diagnosed only by microscopy, with maximum depth of invasion ≤5 mm
IA1	Measured stromal invasion ≤3 mm in depth
IA2	Measured stromal invasion >3 and ≤5 mm in depth
IB	Invasive carcinoma with measured deepest invasion >5 mm (greater than Stage IA); lesion limited to the cervix uteri with size measured by maximum tumor diameter
IB1	Invasive carcinoma >5 mm depth of stromal invasion and ≤2 cm in greatest dimension
IB2	Invasive carcinoma >2 and ≤4 cm in greatest dimension
IB3	Invasive carcinoma >4 cm in greatest dimension
II	The carcinoma invades beyond the uterus, but has not extended onto the lower third of the vagina or to the pelvic wall
IIA	Involvement limited to the upper two-thirds of the vagina without parametrial involvement
IIA1	Invasive carcinoma ≤4 cm in greatest dimension
IIA2	Invasive carcinoma >4 cm in greatest dimension
IIB	With parametrial involvement but not up to the pelvic wall
III	The carcinoma involves the lower third of the vagina and/or extends to the pelvic wall and/or causes hydronephrosis or nonfunctioning kidney and/or involves pelvic and/or para-aortic lymph nodes
IIIA	The carcinoma involves the lower third of the vagina, with no extension to the pelvic wall
IIIB	Extension to the pelvic wall and/or hydronephrosis or nonfunctioning kidney (unless known to be due to another cause)
IIIC	Involvement of pelvic and/or para-aortic lymph nodes (including micrometastases), irrespective of tumor size and extent (with r and p notations)
IIIC1	Pelvic lymph node metastasis only
IIIC2	Para-aortic lymph node metastasis
IV	The carcinoma has extended beyond the true pelvis or has involved (biopsy proven) the mucosa of the bladder or rectum. A bullous edema, as such, does not permit a case to be allotted to Stage IV
IVA	Spread of the growth to adjacent pelvic organs
IVB	Spread to distant organs

Early-stage cervical cancer is generally treated with surgical resection, which can vary from conization to a modified radical hysterectomy. However, women with high-risk pathology after surgery may require additional treatment, including chemotherapy and radiation. For those with early-stage disease who wish to preserve fertility, conization or trachelectomy may be considered. In cases of more advanced disease, concurrent chemoradiation is the standard treatment approach.[97]

MATERIALS AND METHODS

3.1. Study design

A retrospective study was conducted over the period from January 2024 to January 2025, which includes an analysis of 180 cases of hysterectomy that meet the inclusion criteria for the study. Cases received between 2022 and 2023 in the Teaching Laboratory of Al-I mamain Al-Kadhimain medical city were collected for this study. For each case the histopathological reports and the slides were collected. In addition clinical parameters such as (age, type of hysterectomy, clinical indication, and ultrasound diagnosis) were collected from the pathology reports and ultrasound reports.

In the Pathology Department at College of Medicine/Al-Nahrain University, H&E stained slides were re-examined by the supervisor pathologist for revision of the diagnosis.

3.2. Inclusion criteria

All patients undergoing hysterectomy for various clinical reasons were included.

3.3. Exclusion criteria

1. Hysterectomies due to pregnancy related complications (e.g. uterine rupture, placenta accreta, increta, percreta).
2. Hysterectomies due to gestational trophoblastic diseases will be excluded (e.g. molar pregnancy, choriocarcinoma).

3. Cases that contained incomplete information or lacked the necessary reports.

3.4. Ethical considerations

The scientific committee of the Iraqi Board of Medical Specialization approved the study (approval number: Patho 2, approval date: 21st February 2024).

3.5. Statistical analysis

Depending on whether the distribution was normal or skewed, continuous variables were expressed as means and standard deviations. Categorical variables were expressed as frequency and percentages. The Welch's t-test (for normally distributed variables) was performed. The difference between categorical variables was investigated using either the $\chi 2$ test with yates' correction or

Fisher's exact test, depending on the context. A P-value less than 0.05 were considered statistically significant. R software packages (dplyr, gt summery and ggplot) were used for data processing, visualization, and statistical analysis ("R version 4.2.2, R Foundation for Statistical Computing, Vienna, Austria").

RESULTS

4.1. *Age and ultrasound findings*

The study included 180 participants with a mean age of 49.2 years (± 8.9). The majority of participants were aged 40-49 years (49.4%) and 50-59 years (33.9%). The most common ultrasound diagnoses were fibroids (37.2%) and thick endometrium with feature suggesting endometrial hyperplasia (18.3%). Other notable diagnoses included adenomyosis (9.4%) and endometrial polyps (6.1%) as shown in Table (4.1) below.

Table (4.1): Description of patient's age groups and US findings.

Characteristic	N = 1801
Age (years)	49.2 ± 8.9
Age category (years)	
20-29	1 (0.6%)
30-39	11 (6.1%)
40-49	89 (49.4%)
50-59	61 (33.9%)
60-69	18 (10.0%)
US diagnosis	
Fibroid	67 (37.2%)
Thick endometrium suggestive of endometrial hyperplasia	33 (18.3%)
Normal	33 (18.3%)
Adenomyosis	17 (9.4%)
Endometrial polyp	11 (6.1%)
Suspicion of endometrial malignancy	8 (4.4%)
Ovarian cyst/mass	6 (3.3%)
Cervical mass	1 (0.6%)
Left hydrosalpinx	1 (0.6%)
Septate uterus	1 (0.6%)
Suspicious sarcoma	1 (0.6%)
Tubovarian abscess	1 (0.6%)
1 Mean ± SD; n (%)	

Figure (4.1) illustrates the percentage distribution of various clinical indications among the study participants. The most prevalent clinical indication is abnormal uterine bleeding (AUB), accounting for 61.67% of cases. This is followed by fibroids, which represent 21.67% of the indications. Chronic pelvic pain is noted in 5.56% of cases, while suspicion of endometrial malignancy accounts for 4.44%.

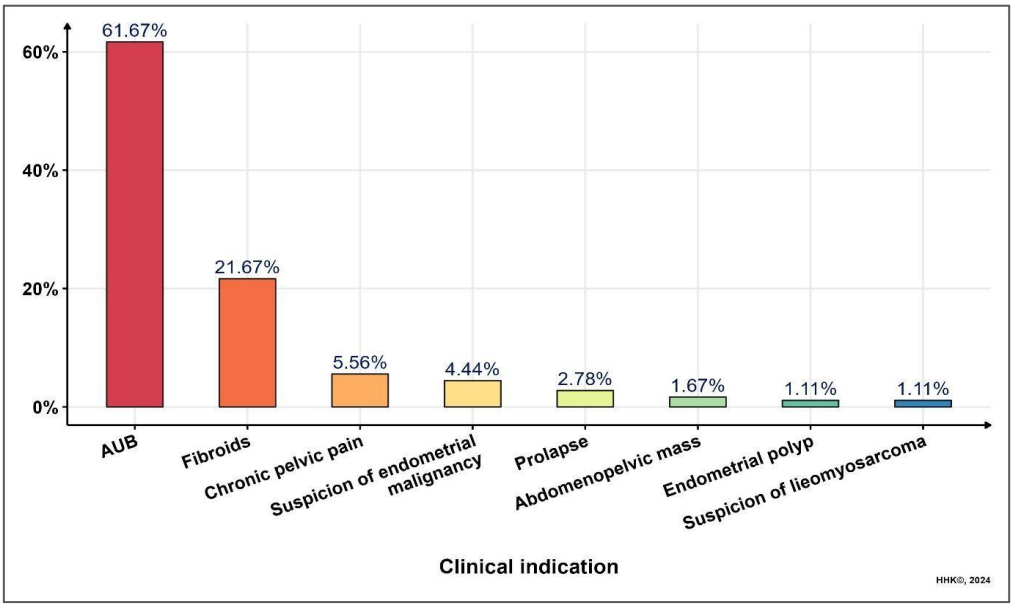


Figure (4.1): Clinical indications for hysterectomy.

The most common type of hysterectomy among the study participants was total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH+BSO), accounting for 71.67% of the cases. This is followed by total abdominal hysterectomy (TAH) at 14.44%, and total abdominal hysterectomy with unilateral salpingo-

oophorectomy (TAH+USO) at 6.67% as Figure (4.2) demonstrates.

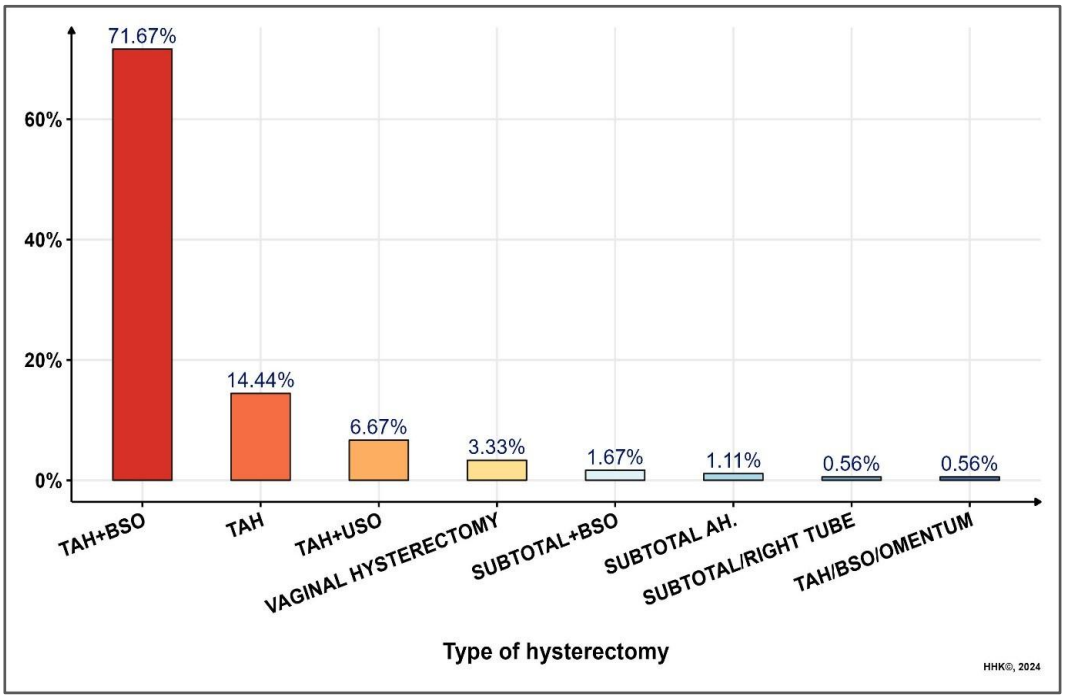


Figure (4.2): Types of hysterectomy.

4.2. Histopathological findings among the study sample

Concerning the histopathological findings shown in Table (4.2), among endometrial lesions, the most common findings were endometrial hyperplasia (EH) without atypia (36.7%), proliferative endometrium (26.1%), and benign endometrial polyps (13.9%). Myometrial lesions were predominantly leiomyomas (31.7%), adenomyosis (25.0%), and leiomyoma/adenomyosis combination (23.3%). For cervical lesions, chronic cystic cervicitis was the most frequent (34.4%), followed by chronic cervicitis (21.7%) and normal examination (19.4%).

Table 4.2: Frequency of the endometrial, myometrial and cervical findings in the study sample.

Characteristic	**N = 1801**
Endometrial findings	
Endometrial hyperplasia without atypia	66 (36.7%)
Proliferative endometrium	47 (26.1%)
Benign endometrial polyp	25 (13.9%)
Endometrial hyperplasia with atypia	12 (6.7%)
Atrophic endometrium	10 (5.6%)
Secretory endometrium	7 (3.9%)
Hormonal imbalance	5 (2.8%)
No changes	4 (2.2%)
Endometroid endometrial CA	3 (1.7%)
Chronic endometritis	1 (0.6%)
Myometrial findings	
Leiomyoma	57 (31.7%)
Adenomyosis	45 (25.0%)
Lieomyoma/Adenomyosis(L/A)	42 (23.3%)

Characteristic	N = 1801
Normal	33 (18.3%)
Involved by malignancy(endometroid endometrial carcinoma)	3 (1.7%)
Cervical findings	
Chronic cystic cervicitis	62 (34.4%)

Chronic cervicitis	39 (21.7%)
Normal	35 (19.4%)
Chronic cervicitis with squamous metaplasia	9 (5.0%)
Endocervical polyp	8 (4.4%)
Nabothian cyst	7 (3.9%)
Low grade squamous intraepithelial neoplasia (LSIL)	6 (3.3%)
Organ not submitted	6 (3.3%)
Leiomyoma	3 (1.7%)
Involved by malignancy(endometroid endometrial carcinoma)	2 (1.1%)
Bartholin cyst	1 (0.6%)
High grade squamous intraepithelial neoplasia (HSIL)	1 (0.6%)
Squamous cell carcinoma (SCC)	1 (0.6%)
1 Mean ± SD; n (%)	

Regarding adnexal lesion findings, for the right ovary, 42.2% of samples showed no changes, which was as well true in 44.4% of the left ovaries samples. Cystic follicles were present in 15.0% of right ovaries and 13.9% of left ovaries. Luteal cysts were found in 12.2% of right ovaries and 10.6% of left ovaries. The most common finding in the fallopian tubes was no changes, observed in 72.8% of right fallopian tubes and 71.7% of left fallopian tubes.

Table 4.3.: Frequency of the adnexal findings in the study sample.

Characteristic	**N = 1801**	**Characteristic**	**N = 1801**
Right ovary		**Left ovary**	
No changes	76 (42.2%)	No changes	80 (44.4%)
Organ not submitted	37 (20.6%)	Organ not submitted	37 (20.6%)
Cystic follicles	27 (15.0%)	Cystic follicles	25 (13.9%)
Luteal cyst	22 (12.2%)	Luteal cyst	19 (10.6%)
Mucinous cystadenoma	7 (3.9%)	Mucinous cystadenoma	6 (3.3%)
Follicular cyst	5 (2.8%)	Follicular cyst	4 (2.2%)
Free of malignancy	2 (1.1%)	Serous cystadenoma	4 (2.2%)
Benign Brenner	1 (0.6%)	Free of malignancy	2 (1.1%)
Endometriotic cyst	1 (0.6%)	Endometriotic cyst	1 (0.6%)
LGS CA	1 (0.6%)	LGS CA	1 (0.6%)
Serous cystadenoma	1 (0.6%)	Ovarian fibroma	1 (0.6%)
Right fallopian tube		Left fallopian tube	
No changes	131(72.8%)	No changes	129 (71.7%)
Organ not submitted	33 (18.3%)	Organ not submitted	35 (19.4%)
Para-tubal cyst	9 (5.0%)	Para-tubal cyst	10 (5.6%)
Chronic salpingitis	4 (2.2%)	Chronic salpingitis	4 (2.2%)
Hydrosalpinx	2 (1.1%)	Hydrosalpinx	1 (0.6%)
Involved by malignancy	1 (0.6%)	Involved by malignancy	1 (0.6%)
1 Mean ± SD; n (%)			

Figure (4.3) demonstrates that the vast majority of the study participants, 97.22%, had no evidence of malignancy. Among the remaining participants, 1.11% was diagnosed with endometrioid endometrial carcinoma, 1 patient (0.56%) with bilateral ovarian low-grade serous carcinoma, 1 patient (0.56%) with cervical squamous cell carcinoma, and 1 patient (0.56%) diagnosed with HSIL showed no evidence of invasion by malignancy.

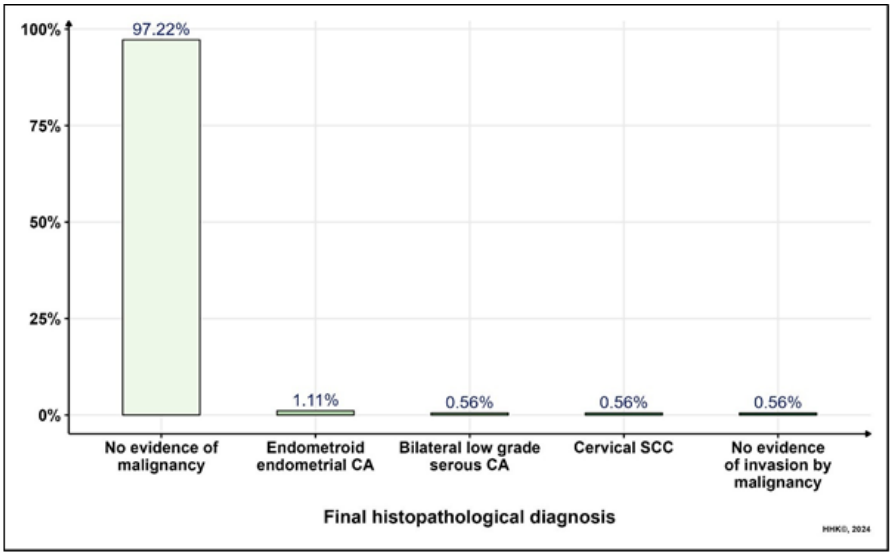


Figure 4.3: Final histological diagnosis among the study sample.

4.3. Distribution of the most common endometrial, cervical and adnexal histopathological lesions in different age groups among the study sample

Table (4.4) shows the specific distribution of the most prevalent histopathological lesions in different age groups. Among those with endometrial hyperplasia (EH) without atypia, the majority were aged 40-49 years (54.5%) and 50-59 years (36.4%). Chronic cystic cervicitis was most common in the 40-49 years age group (54.8%), followed by the 50-59 years age group (30.6%) as well. While serous cystadenoma and mucinous cystadenoma were predominantly found in older age groups (50-59 years) (75.0% and 42.9%, respectively). One case with sow-grade serous carcinoma (LGS CA) was found in the 30-39 years age group.

Table 4.4: Distribution of the most common endometrial, cervical and adnexal histopathological lesions in different age groups among the study sample.

Age group (years)	EH without atypia, N = 661	Chronic cystic cervicitis, N = 621	Serous cystadenoma, N = 41	Mucinous cystadenoma, N = 71	LGS CA, N=11
20-29	0 (0.0%)	1 (1.6%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
30-39	3 (4.5%)	5 (8.1%)	0 (0.0%)	1 (14.3%)	1(100.0%)
40-49	36 (54.5%)	34 (54.8%)	1 (25.0%)	2 (28.6%)	0 (0.0%)
50-59	24 (36.4%)	19 (30.6%)	3 (75.0%)	3 (42.9%)	0 (0.0%)
60-69	3 (4.5%)	3 (4.8%)	0 (0.0%)	1 (14.3%)	0 (0.0%)
1 n (%) EH: Endometrial hyperplasia; LGA CA: Low-grade serous carcinoma.					

4.4. Distribution of myometrial histopathological lesions in different age groups among the study sample

As shown in Table (3.5) below, among those with adenomyosis, the majority were aged 40-49 years (55.6%) and 50-59 years (33.3%). Similarly, leiomyoma was most common in the 40-49 years age group (57.9%), followed by the 50-59 years age group (29.8%); which also held true for participants with both leiomyoma and adenomyosis where the highest prevalence was in the 40-49 years age group (47.6%) and the 50-59 years age group (42.9%).

Table (4.5): Distribution of myometrial histopathological lesions in different age groups among the study sample.

Age groups (years)	Adenomyosis, N = 451	Leiomyoma, N = 571	Leiomyoma+ adenomyosis, N = 421
20-29	0 (0.0%)	1 (1.8%)	0 (0.0%)
30-39	2 (4.4%)	3 (5.3%)	1 (2.4%)
40-49	25 (55.6%)	33 (57.9%)	20 (47.6%)
50-59	15 (33.3%)	17 (29.8%)	18 (42.9%)
60-69	3 (6.7%)	3 (5.3%)	3 (7.1%)
1 n (%)			

4.5. Histopathological diagnosis among patients with abnormal uterine bleeding (AUB)

As shown in Table (3.6), out of a total of 112 cases of abnormal uterine bleeding (AUB), endometrial hyperplasia without atypia was identified in 44 cases (39.3%), followed by a proliferative endometrium in 26 cases (23.2%), and benign endometrial polyps in 17 cases (15.2%). Endometrial hyperplasia with atypia was less common, found in 5 cases (4.5%), and endometrioid carcinoma was observed in 2 cases (1.8%). Among the myometrial lesions, leiomyomas were present in 35 cases (31.3%), adenomyosis was diagnosed in 40 cases (35.7%), and a combination of leiomyoma and adenomyosis occurred in 12 cases (10.7%).

Table (4.6): Histopathological diagnosis among patients with AUB.

Characteristic	AUB, N = 1121
Endometrial lesions	
Endometrial hyperplasia without atypia (N = 66)	44 (39.3%)
Proliferative endometrium (N = 47)	26 (23.2%)
Benign endometrial polyp (N = 25)	17 (15.2%)
Endometrial hyperplasia with atypia (N = 12)	5 (4.5%)
Endometroid CA (N = 3)	2 (1.8%)
Leiomyoma (N = 57)	35 (31.3%)
Adenomyosis (N = 45)	40 (35.7%)
Leiomyoma and adenomyosis (N = 42)	12 (10.7%)
1 n (%)	

4.6. Correlation between AUB and endometrial lesions, leiomyoma and adenomyosis

As Table (4.7) demonstrates, no significant correlation was seen between the clinical presentation of AUB and the endometrial lesions on histopathological examination (P-value=0.8) with endometrial hyperplasia without atypia being the most prevalent finding among those with and without AUB on presentation (39.3% and 32.4%, respectively). Similarly, leiomyoma showed no significant correlation with a clinical presentation of AUB (P-value = 0.6). On the other hand, adenomyosis was significantly higher in the AUB group (35.7%) compared to those who did not present with AUB (7.4%). While the presence of both pathologies (leiomyoma and adenomyosis) on histopathological examination was notably more prevalent in the non-AUB group (44.1%) with statistical significance (P-value < 0.001).

Table 4.7: Correlation between AUB and endometrial lesions, leiomyoma and adenomyosis.

Characteristic	Negative, N = 681	Positive, N = 1121	P-Value2
Endometrial lesions			0.8
Endometrial hyperplasia without atypia (N=66)	22 (32.4%)	44 (39.3%)	
Proliferative endometrium (N = 47)	21 (30.9%)	26 (23.2%)	
Benign endometrial polyp (N = 25)	8 (11.8%)	17 (15.2%)	
Endometrial hyperplasia with atypia (N = 12)	7 (10.3%)	5 (4.5%)	
Endometroid CA (N = 3)	1 (1.5%)	2 (1.8%)	
Leiomyoma (N = 57)	24 (35.3%)	35 (31.3%)	0.6
Adenomyosis (N = 45)	5 (7.4%)	40 (35.7%)	<0.001
Leiomyoma and adenomyosis (N = 42)	30 (44.1%)	12 (10.7%)	<0.001
1 n (%) 2 Fisher's exact test			

4.7. Correlation between clinical and histopathological diagnosis

As demonstrated below Table (4.8), both leiomyoma and prolapse showed statistically significant correlation between the clinical presentation and histopathological results. All patients (100%) diagnosed with uterine fibroid (leiomyoma) on clinical grounds were found to have leiomyomatous lesion on histopathology (p-value <0.001); similarly, a significant proportion (40%) of those suspected clinically to have prolapse showed histopathological findings suggestive of prolapse in the form of atrophic endometrium (p-value = 0.021). On the other hand, out of the 3 patients presented with an abdominopelvic mass (adnexal lesion), only one participant showed consistent histopathological features of low-grade serous carcinoma, LGS CA).

Table 4.8: Correlation between clinical and histopathological diagnosis.

Organic lesion in uterus	Clinical diagnosis, N = 1801	Histopathological diagnosis, N = 1801	Correlation between clinical & histopathology in positive cases	P-value2
Leiomyoma	39(21.7%)	60 (42.5%)	39 (100.0%)	<0.001
Prolapse	5 (2.8%)	10 (5.6%)	2 (40.0%)	0.021
Abdominopelvic Mass (adnexal lesion)	3 (1.7%)	12 (6.7%)	1 (33.3%)	0.2
Endometrial polyp	2 (1.1%)	25 (13.9%)	0 (0.0%)	>0.9
Malignancy	10 (5.6%)	4 (2.2%)	1 (10.0%)	0.2
1 n (%) 2 Fisher's exact test; Pearson's Chi-squared test				

4.8. Correlation between ultrasound finings and histopathological diagnosis

Correlation between ultrasound and histopathological findings was demonstrated in Table (3.8). Leiomyoma showed a significant correlation, with 98.5% of positive ultrasound diagnoses confirmed histopathologically (p<0.001). Adenomyosis as well had a significant correlation, with 82.4% of positive ultrasound diagnoses confirmed histopathologically (p = 0.003). Endometrial hyperplasia, endometrial polyp, and pelvic mass, on the other hand, showed no significant correlation between ultrasound and histopathological diagnoses. However, endometrioid cancer had a significant correlation, with 25.0% of positive ultrasound diagnoses confirmed histopathologically (p = 0.005).

Table (4.9): Correlation between ultrasound findings and histopathological Diagnosis.

Histopathological diagnosis	Ultrasound diagnosis		P-value2
	Negative	Positive	
Leiomyoma (N = 99)3	34 (28.9%)	65 (98.5%)	< 0.001
Adenomyosis (N = 87)3	73 (44.8%)	14 (82.4%)	0.003
Endometrial hyperplasia (N = 78)	58 (39.5%)	18 (54.5%)	0.11
Endometrial polyp (N = 25)	22 (13.0%)	3 (27.3%)	0.2
Endometroid cancer (N = 3)	1 (0.6%)	2 (25.0%)	0.005
Pelvic mass (N = 11)	11 (6.3%)	1 (16.7%)	0.3
1 n (%) Pearson's Chi-squared test N= total number of adenomyosis, leiomyoma diagnosed alone or as L/A			

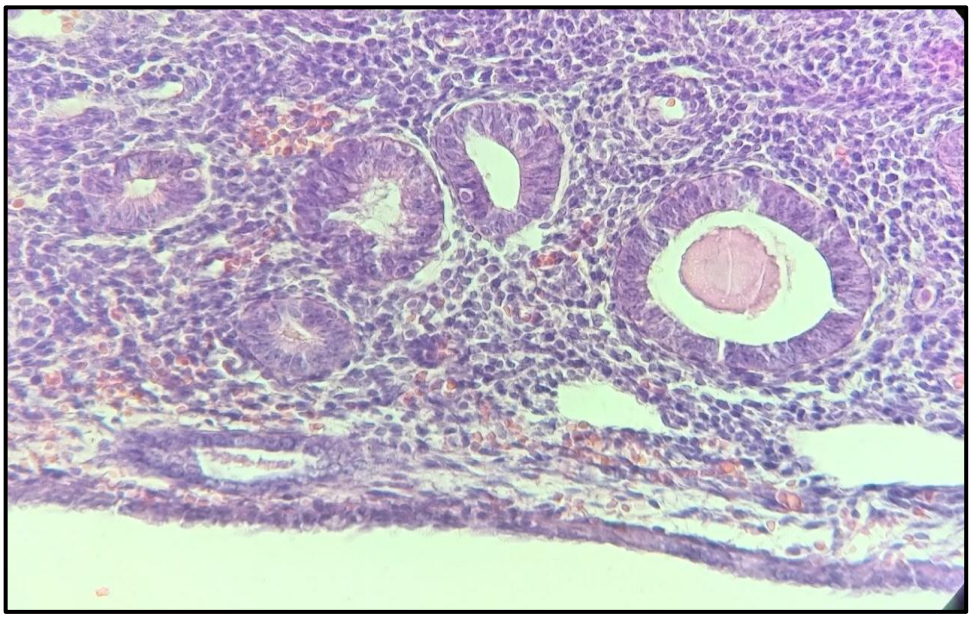


Figure (4.4): Photomicrograph of proliferative endometrium shows round endometrial gland with stratified columnar epithelium in a background of dense compact stroma. No increase in gland to stroma ratio (glands <50% of surface area (H&E 10 X).

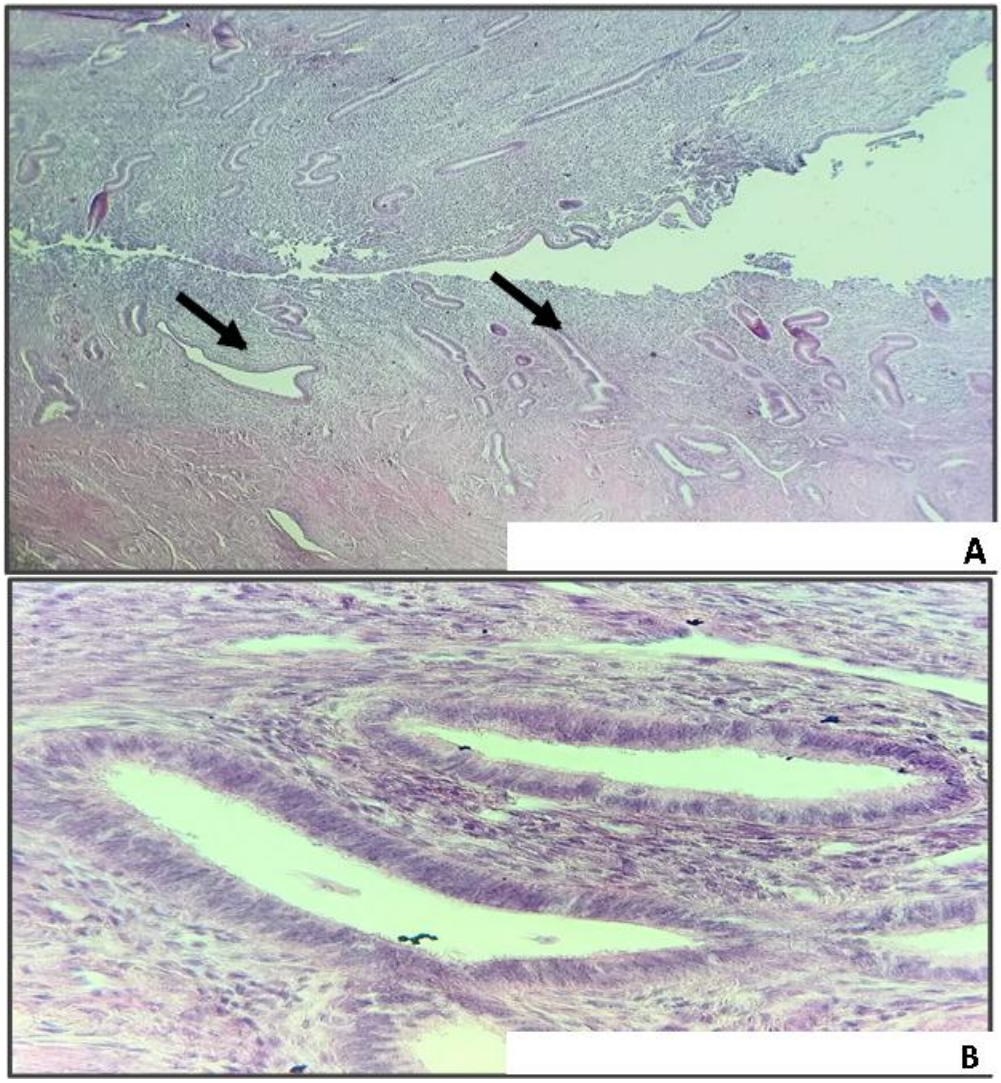
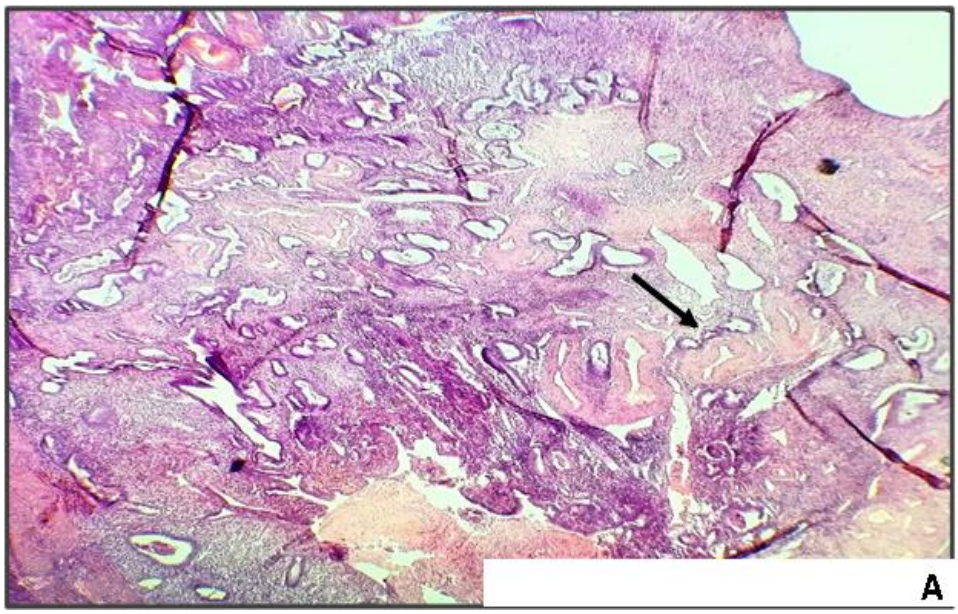


Figure (4.5): Photomicrograph of disordered proliferative endometrium A: Shows dilated, elongated and convoluted endometrial glands (black arrow) interspersed between proliferative endometrial glands. There is no increase in gland to stroma ratio (glands less than 50% of surface area) (H&E 4X). B: Endometrial gland still lined by columnar epithelium of proliferative endometrium (H&E 10X).



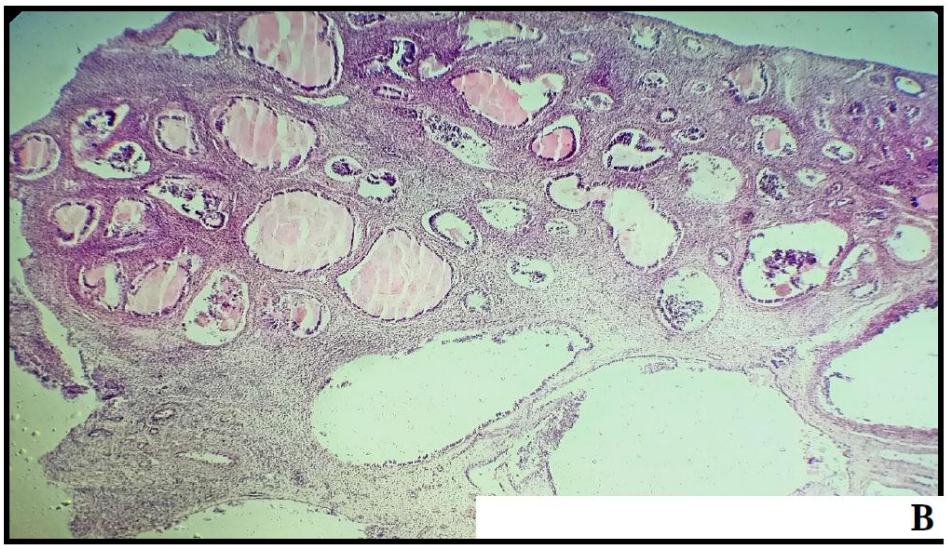


Figure (4.6): Photomicrograph of endometrial polyp A: Thick wall blood vessels (black arrow) adjacent to irregular, dilated, angulated endometrial glands of proliferative endometrium lining, variably cellular stroma and stromal hemorrhage (H&E 4X). B: Polypoid fragment of variable sizes, cystically dilated endometrial glands lined by flat inactive to proliferative endometrial epithelium (H&E 10X).

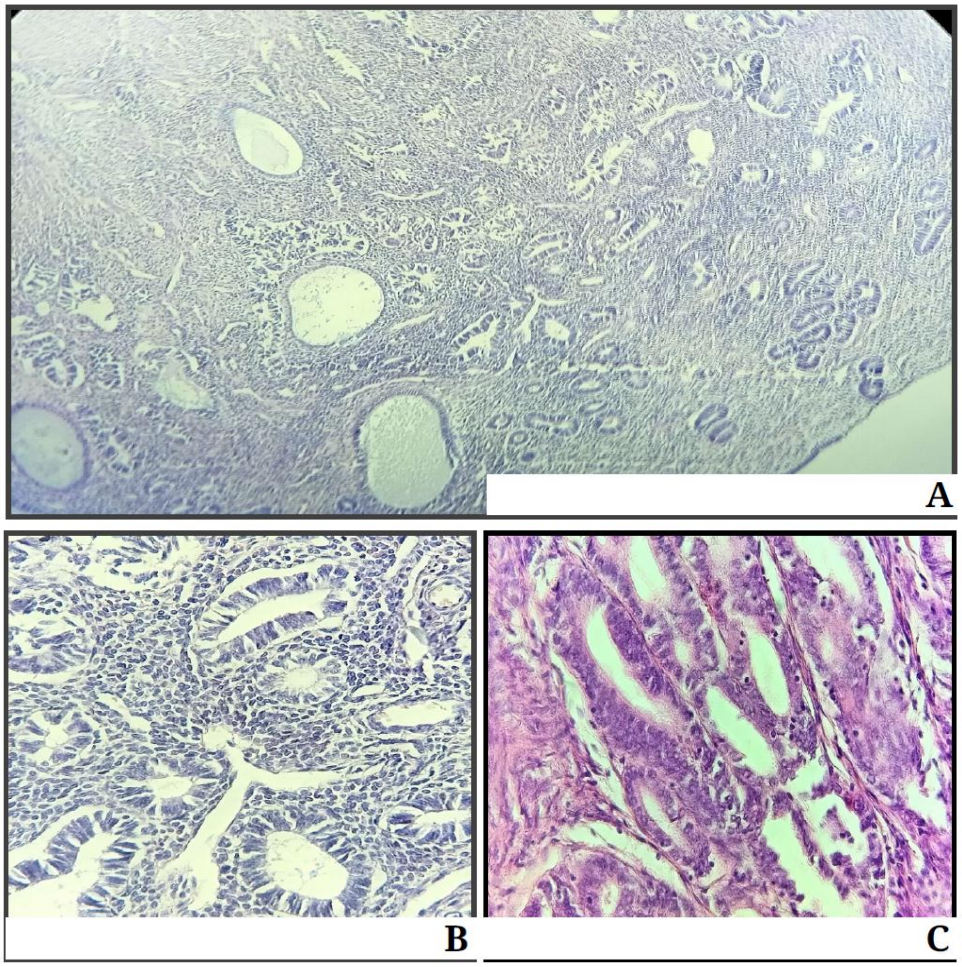


Figure (4.7): Photomicrograph of endometrial hyperplasia A: increase in gland to stroma ration more than 3:1 with some glandular complexity (H&E 4X). B: Endometrial hyperplasia without atypia, note that endometrial glands still have the cytological resemblance to normal proliferative endometrium which overall retain stratification and small nuclei, no cytological atypia (H&E 10X). C: Atypical hyperplasia, note the loss of polarization toward basement membrane and slight enlargement and rounding of the nuclei in the atypical glands (H&E 40X).

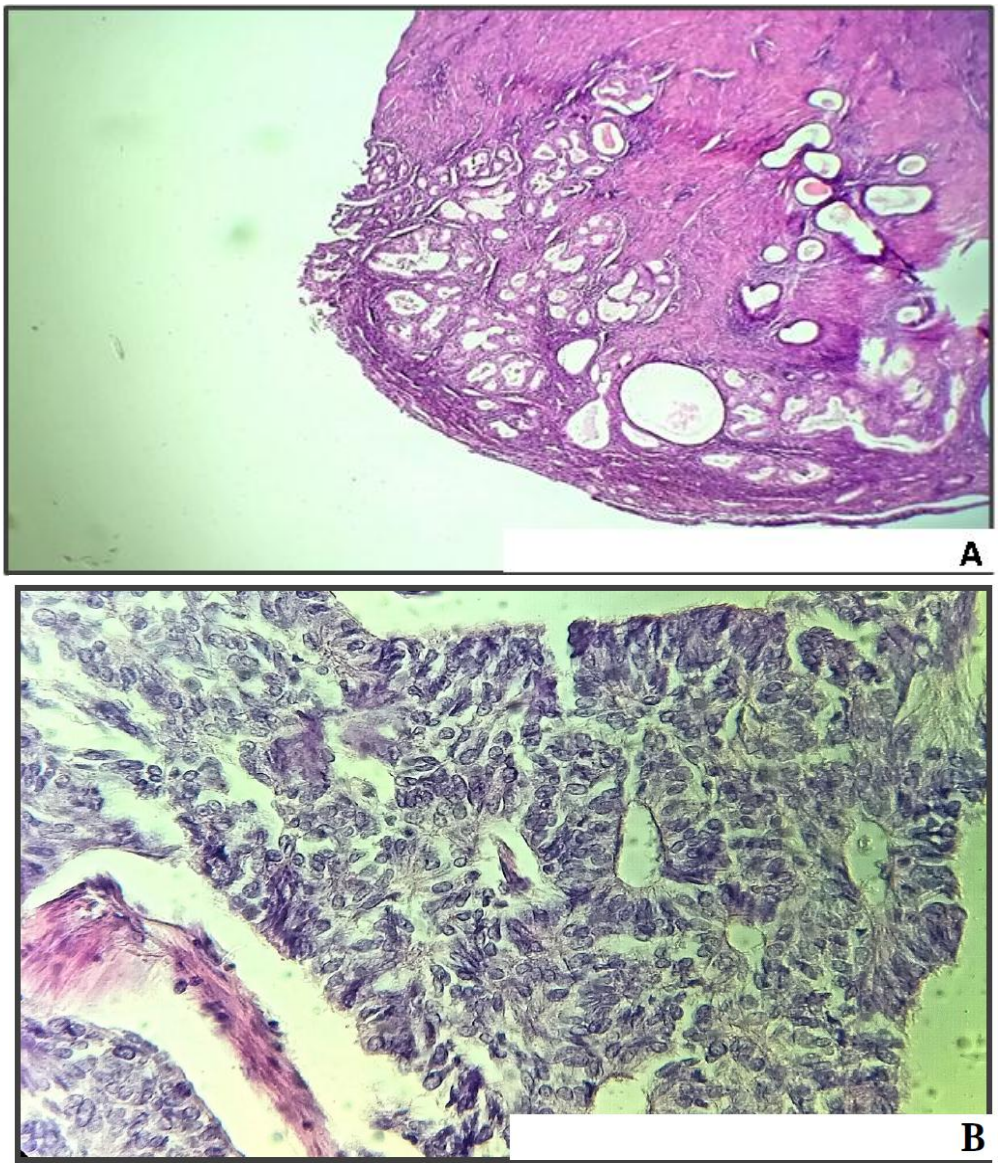
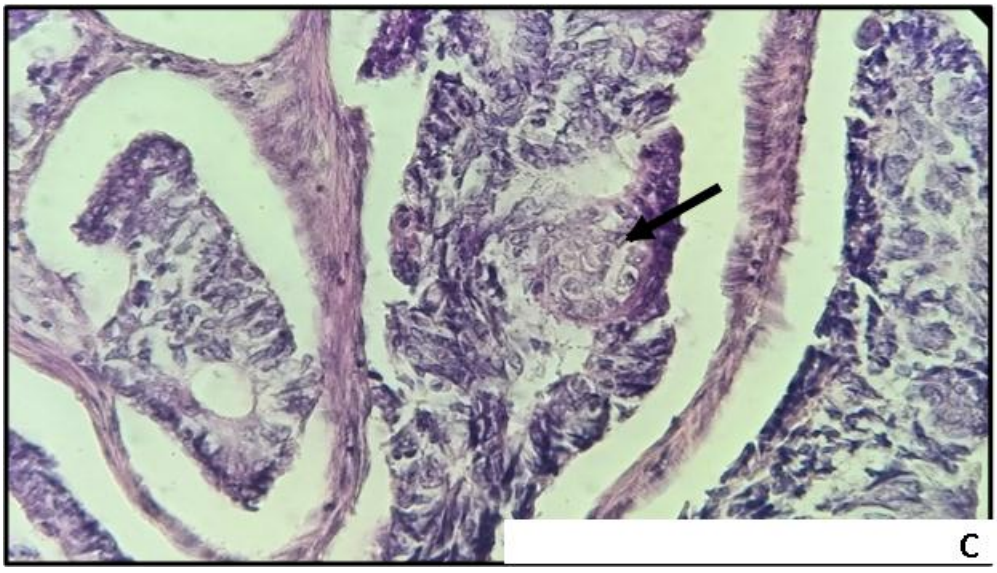


Figure (4.8): Photomicrograph of endometroid endometrial carcinoma FIGO grade I; A: Confluent grandular to cribriform growth pattern. (H&E 4X). B: Shows tightly packed atypical glands with tendency to cribriforming, atypical hyperplasia bordering FIGO grade I endometroid endometrial carcinoma (H&E 10X).



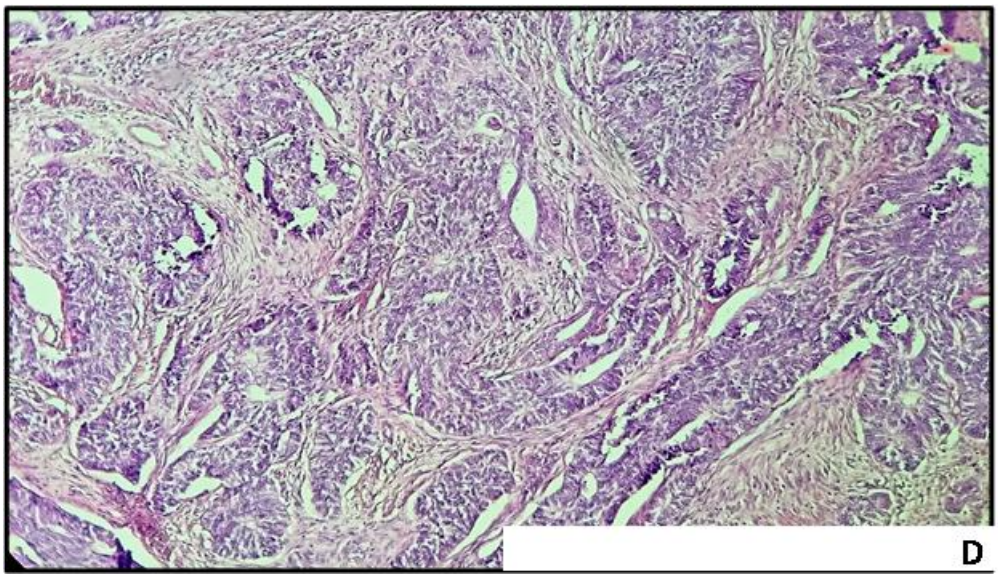


Figure (4.9): C: Morular metaplasia, with whorled, spherical collection of squamoid cells amidst malignant gland (arrow) (H&E 40X) D: Myometrial invasion of malignant endometrial glands (H&E 10X).

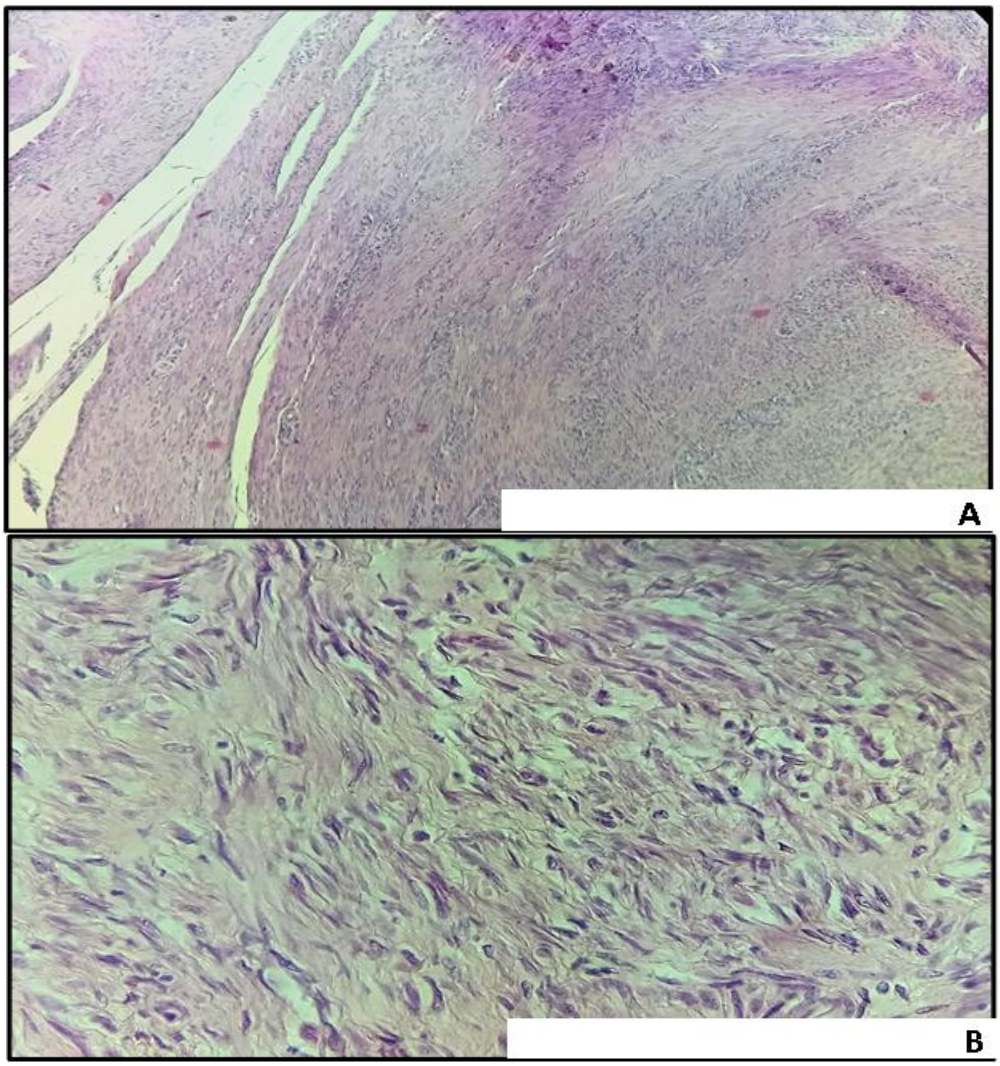


Figure (4.10): Photomicrograph of uterine lieomyoma A: Shows well circumscribed growth of intersecting fasicles of benign spindle cells, no hemorrhage or necrosis seen (H&E 10 X). B: Shows intersecting fasicles of spindle cells with cigar shaped nuclei, no cytological atypia, no mitosis (H&E 40X).

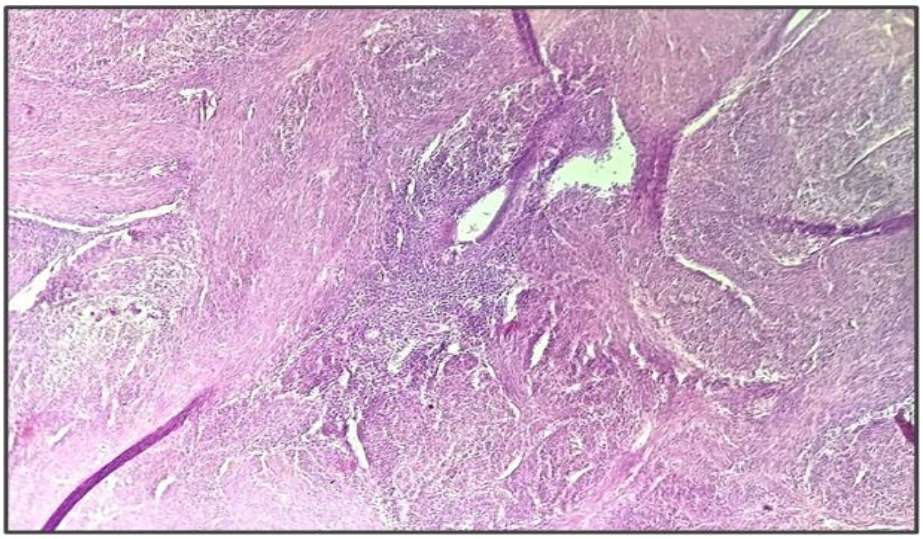


Figure (4.11): Photomicrograph of uterine adenomyosis: endometrial glands and stroma deep in myomterium, this is gland poor adenomyosis (H&E 10X).

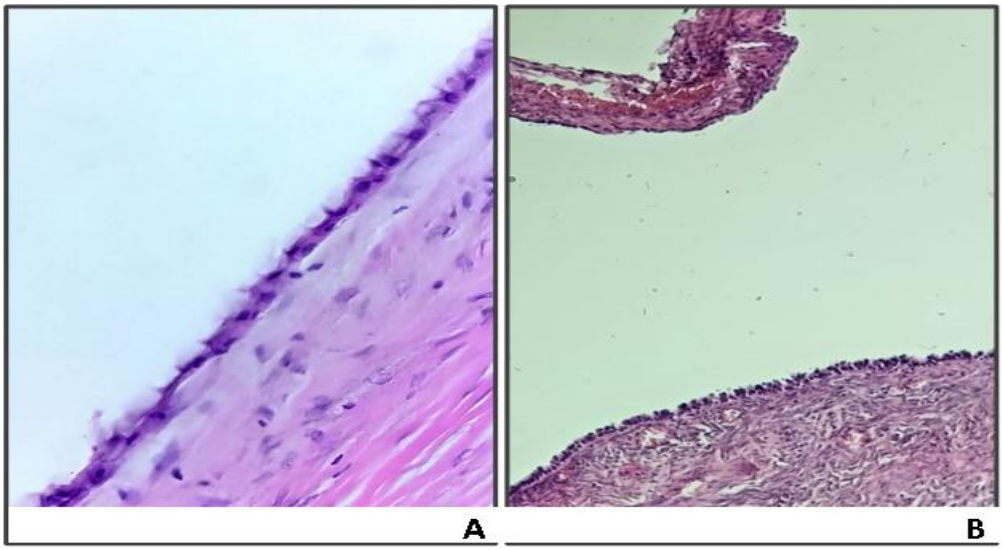
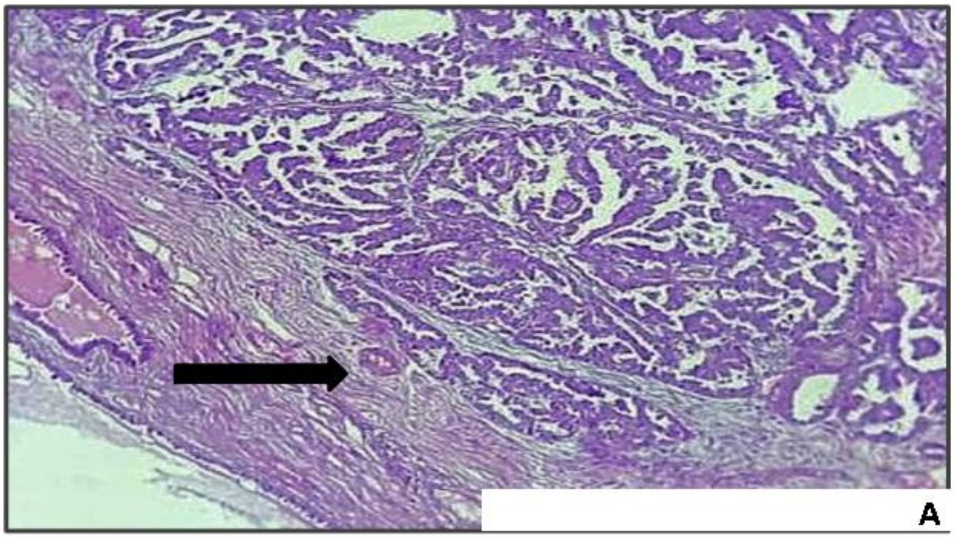


Figure (4.12): Photomicrograph of ovarian cystadenoma A: Simple unilocular cyst line by mucinous epithelium, diagnosis of ovarian mucinous cystadenoma (H&E 40 X). B: Shows multilocular cystic lesion lined by columunar ciliated epithelium giving a diagnosis of ovarian serous cystadenoma (H&E 10 X).



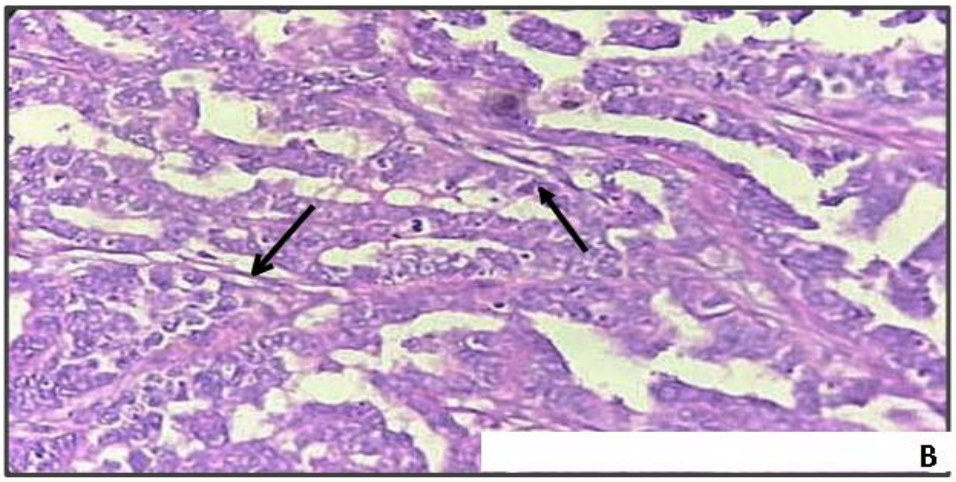


Figure 4.13: Photomicrograph of ovarian low grade serous cancer A: Micropapillary to complex papillary growth pattern, there is invasion of the stroma by cribriform growth pattern (black arrow) (H&E 10X). B: Homogeneous population of small cells with scant cytoplasim with moderated nuclear atypia, there is slit like spaces (arrows) (H&E 40X).

DISCUSSION

Hysterectomy is the most commonly performed gynaecological surgery done in female and is a successful operation in terms of symptomatic relief, patient satisfaction as well as provides definitive cure to many diseases involving uterus and adnexa like fibroids, menorrhagia, adenomyosis, endometriosis, pelvic inflammatory disease, uterovaginal prolapse and malignancy. The prevalence of hysterectomy varies from country to country, region to region. The type of hysterectomy depends on the age of the patient, the size of the uterus, and the clinical presentation.[100,101]

In present study, 180 hysterectomy specimens were evaluated with regards to age distribution and type of hysterectomy operation, clinical indication and histopathological diagnosis at histopathology department of a tertiary care center. In this study, the commonest age group of hysterectomy was 40-49 years (49.4) % followed by 50- 59 years (33.9) %. This was consistent with a study by Noora et al,[142] Ronald et al[131], Vandana et al[104] and similar to analysis of 150 cases by Harshal et al, and 35 cases analysis by Agarwal S et al where most cases were of age group of 41-50 years.[102,103]

Majority of hysterectomies were carried out via the abdominal route, while the vaginal route constituted only 3.33% of the study sample. Similar rates of 5.3% for vaginal route were reported by Abubakar et al.[105] similar findings for vaginal route were also reported by Hadiza et al. although with a higher rate of 22.4%.[106] The most common indication for vaginal hysterectomy in this study was uterovaginal prolapse which is in keeping with studies done elsewhere.[107,108] Data from the United Kingdom reveal that abdominal hysterectomy procedures are five to six times more common than vaginal hysterectomy procedures.[109] In a study by Pandya et al,[120] vaginal hysterectomy was the surgical procedure most commonly used in comparison to abdominal hysterectomy. The difference in increase vaginal hysterectomy over abdominal hysterectomy may reflect advances in minimally invasive surgical techniques, patient preferences for less invasive procedures with faster recovery, and institutional policies prioritizing cost-effective methods. Additionally, demographic and temporal factors (duration of the study, time of data collection, trend changes in long term studies, etc. In the study sample may influence the trend. Total abdominal hysterectomy with bilateral salpingo- oophorectomy was the most common type of abdominal hysterectomy in this study with 71.67% rate followed by total abdominal hysterectomy with preservation of the adenxia in 14.44% rate. Similar observations were made by Patil el al.[105,121] This high percentage of TAH BSO in our study may be explained by the predominance of patients aged 40–49 and 50–59 years, corresponding to perimenopausal and postmenopausal stages. In these age groups, declining ovarian function reduces the benefits of ovarian preservation, while the risk of future ovarian pathology increases. Prophylactic oophorectomy is often preferred to prevent complications, particularly in hormone-sensitive conditions or high-risk individuals. Additionally, many patients in this age range opt for TAH BSO to avoid future surgeries, reflecting the influence of age-related considerations and patient preferences on surgical decision-making.

The most common clinical indication for the hysterectomy was abnormal uterine bleeding (AUB), followed by uterine fibroids, and chronic pelvic pain account for 61.67%, 21.67% and 5.56%, respectively. Abnormal uterine bleeding was the most common clinical indication in study performed by Ronald et al, Vinzuda et al.[112,131] and a study conducted by Toma A et al. from Canada had AUB/DUB as the commonest indication followed by uterine fibroid.[113] This is inconsistent with study conducted in India by Kolur A et al, and a study conducted in United states of America by

M S Broder, which both had uterine fibroid as the most common clinical indication. Uterine fibroid also was the most common reason for performing hysterectomy even from a study conducted from Africa by JL Butt et al.[114,115,116] This differences between studies can be attributed to differences in population demographics, geographic and socioeconomic factors, and healthcare access. Variations in diagnostic practices, clinical guidelines, and physician preferences also influence findings. Additionally, differences in study design, sample size, and cultural or patient preferences contribute to discrepancies. These factors highlight the need for careful contextual interpretation of study results. In this study, uterine prolapse was identified as the clinical indication in 5 cases, representing only 2.78% of the study population. Histopathological examination revealed atrophic endometrium in two of these cases, aligning with the diagnosis of uterine prolapse. However, Onyeabochukwu et al. in Owerri reported uterovaginal prolapse as the most common indication followed by uterine fibroid.[117] This could be due to the differences in the ages as most cases of UV prolapse was seen in the younger age groups.

Regarding endometrial pathology, endometrial hyperplasia was the most common diagnosis in this study, accounting for 43.4% of cases. Among these, 36.7% were classified as endometrial hyperplasia without atypia, while the remaining 6.7% were diagnosed with atypical hyperplasia. This was followed by proliferative endometrium at 26.1% and benign endometrial polyps at 13.9%. This finding is consistent with a study conducted in Aden by Ziad et al., which analyzed 2,544 hysterectomy specimens and reported that endometrial hyperplasia accounted for 58.3% of the cases.[133] However, these findings differ significantly from those reported by Harshal et al.[103] where proliferative endometrium was the most common endometrial pathology, accounting for 51.3%, while endometrial hyperplasia represented only 14.7% 123. Similarly, Rashimi et al. identified proliferative endometrium as the most frequent diagnosis (39.15%), with endometrial hyperplasia accounting for only 7.28%.[109]

Numerous other studies also report lower rates of endometrial hyperplasia.[118,105] The predominance of endometrial hyperplasia in the study may be influenced by delays in seeking medical advice, allowing prolonged unopposed estrogen exposure and progression of endometrial changes. Additionally, study-related factors such as conducting the study in a tertiary care setting, and a focus on detailed histopathological evaluation may contribute to the findings. Variations in diagnostic criteria, sample size, study duration, and referral patterns—favoring patients presenting with abnormal uterine bleeding—could also skew results toward a higher detection rate of hyperplasia compared to other endometrial lesions.

Regarding the histopathological diagnosis of myometrial lesions, leiomyoma was the most common finding, identified as the sole diagnosis in 31.7% of cases and in association with adenomyosis in 23.3%. Leiomyoma peaked in the 40–49 age group, either alone or combined with adenomyosis. Clinically, leiomyoma was suspected clinically in 39 patients, and all cases were confirmed histopathologically, demonstrating statistical significance with p value < 0.001. The second most common myometrial lesion was adenomyosis, which accounted for 25.0% of cases as a standalone diagnosis and 23.3% in association with leiomyoma, also peaking in the same age group as leiomyoma. These findings are consistent with those reported by Abubakar et al., where leiomyoma was the most prevalent myometrial lesion.[105] Similarly, studies conducted by Neelgund et al.[119] and Khurshid et al.[120] also identified leiomyoma, followed by adenomyosis, as the most common myometrial lesions.

Regarding cervical lesions, the most common diagnosis in this study was chronic cervicitis, which accounted for 61.1% of cases. This included chronic cystic cervicitis (34.4%), chronic cervicitis without additional features (21.7%), and chronic cervicitis with squamous metaplasia (5.0%). The peak incidence of chronic cystic cervicitis was observed in the 40–49 age groups. Similar findings were reported by Pradhan et al. and JHR et al., where chronic cervicitis was the most prevalent cervical lesion. These studies also demonstrated comparable rates of chronic cervicitis with squamous metaplasia.[121,122] Carcinoma uterine cervix is one of the leading causes of cancer death among women worldwide.[123] In our study, out of seven cases of CIN(SIL), six cases were of CIN-I (LSIL) grade and one was CIN-III (HSIL). One case of invasive squamous cell carcinoma of cervix.

In the ovary, the majority of findings were unremarkable, which aligns with observations reported by Ajaz et al.[127] The most frequent ovarian lesion identified was the cystic follicles, consistent with findings from other studies, including those by Pandey et al.[124] and Rushikesh et al.[125] in India. Among benign ovarian tumors, mucinous cystadenoma was the most common encountered histopathological diagnosis in this study, identified in seven cases, six of which were bilateral. In comparison, serous cystadenoma was reported in four cases, one of which was bilateral. This differs from findings in Nigeria by Abubakar et al.[105] and in Nepal by Medhi et al.[126] where serous cystadenoma was the most common benign tumor. The observed higher prevalence of mucinous cystadenomas in this study may be influenced by variations in diagnostic criteria and sample size. Differences in histopathological interpretation can lead to potential misclassification, particularly in cases with overlapping features, where borderline or mixed tumors may be identified as mucinous. Furthermore, studies with small sample sizes are susceptible to statistical bias, resulting in non-representative distributions of tumor subtypes. These

observations underscore the importance of using standardized diagnostic criteria and ensuring sufficiently large sample sizes to produce accurate and generalizable results. One case of bilateral ovarian low-grade serous adenocarcinoma was identified, classified as FIGO stage IIB with a pathological staging of pT2b.

Low-grade serous carcinoma (LGSC) of the ovary is a rare subtype of epithelial ovarian carcinoma with distinct clinical and molecular characteristics. Unlike high-grade serous carcinoma, LGSC typically occurs in younger patients, follows an indolent course, and is associated with prolonged survival. It may develop de novo or evolve from a serous borderline tumor (SBT). Accurate pathological differentiation from other ovarian carcinoma subtypes is crucial due to its unique behavior. LGSC is frequently associated with KRAS, BRAF, and ERBB2 mutations, while it lacks the TP53 mutations commonly seen in high-grade serous carcinoma. Prognostic factors influencing outcomes include age at diagnosis, smoking status, elevated body mass index, mutational profile, hormonal receptor expression, and the Ki-67 proliferation index. Surgery remains the cornerstone of LGSC management, the primary focus should be on performing maximum cytoreductive surgery to achieve microscopic residual disease in metastatic cases, as numerous studies have explored the influence of residual disease on the prognosis of low-grade serous carcinomas (LGSCs).[128,129]

No changes were the most commonly reported finding in the fallopian tubes, with the most common pathology being paratubal cysts. The most significant lesion observed was chronic salpingitis, which accounted for 2.2% of cases. Additionally, one distinct case involved by ovarian low-grade serous carcinoma. These findings are consistent with those reported by Abubakar et al.[105], where no lesion was the most common diagnosis, and the most significant findings were metastasis and salpingitis. Similarly, the study by Bosco et al.[130] also identified salpingitis as the most significant pathology.

The incidence of malignancy in the hysterectomy specimens in this study was 2.79%, comprising a total of six carcinoma cases: three cases of endometrioid endometrial carcinoma, one case of ovarian low-grade serous carcinoma, one case of invasive squamous cell carcinoma of the cervix, and one case of high-grade squamous intraepithelial lesion (HSIL) without evidence of invasion. This malignancy rate is lower than the 7.75% reported in a study by Ronald et al. However, both studies demonstrated similar rates of endometrial malignancy, with 1.11% in the present study compared to 1.21% in Ronald et al.[131]

In this study, abnormal uterine bleeding (AUB) emerged as the most common clinical indication for hysterectomy, encompassing 112 cases. The histopathological assessment of endometrial and myometrial lesions in AUB patients is emphasized. Among the organic lesions causing AUB, endometrial hyperplasia exhibited the highest incidence, accounting for 43.8% of the total cases. Specifically, 39.3% of cases presented with hyperplasia without atypia, while 4.5% exhibited atypical hyperplasia. These findings are consistent with studies conducted by Ronald et al., Ashi et al., and Nadia et al.,[131,134,137] which similarly identified endometrial hyperplasia as the most frequent endometrial histopathological diagnosis among patients with AUB. The elevated incidence in this study could be attributed to delayed identification of disordered proliferative endometrium. Given that endometrial hyperplasia is considered a precursor to endometrial carcinoma, early recognition of this pattern is crucial. However; this is differ from study conducted in Iraq by Noora et al,[142] where lower rates of 5.6% for non-atypical hyperplasia and 1.4% for atypical hyperplasia were reported. These variations may be attributed to differences in study design and sample size.

Endometrial polyps represented the second most common organic endometrial lesion in this study, constituting 15.2% of cases. This result aligns with a study conducted in Tikrit, Iraq, by Nadia et al.,[137] which reported an incidence rate of 13.8% in a sample of 153 cases. Endometrial carcinoma was identified in only two cases (1.8%) of AUB patients, consistent with findings by Victor et al.,[136] who reported a rate of 2.3% (17 cases out of 719) among patients with AUB. Proliferative endometrium, a normal phase of the menstrual cycle, was identified in 23.2% of cases. This is closely align with 24.7% cases of proliferative endometrium in AUB patients found by Noora et al.,[142] The associated bleeding may be attributed to anovulatory cycles or the presence of concurrent conditions such as adenomyosis or leiomyoma.

Adenomyosis was the most prevalent myometrial finding in patients with AUB, observed as a sole diagnosis in 35.7% of cases and in conjunction with leiomyoma in 10.7% of cases. Leiomyoma was the second most common myometrial pathology, present as a standalone finding in 31.1% of cases and coexisting with adenomyosis in 10.7% of cases. These findings are comparable to those of Ronald et al.,[131] who reported adenomyosis and leiomyoma as the most frequent histopathological findings in the myometrium of AUB patients, with rates of 24% and 20.2%, respectively. Differences in percentages may reflect variations in sample sizes between studies.

When analyzing the correlation between AUB as a clinical indication and histopathological diagnoses, adenomyosis demonstrated a significant association with AUB ($p < 0.001$). However, the dual pathology of leiomyoma and adenomyosis was more frequently associated with hysterectomy for clinical indications other than AUB. This may be attributed to extensive clinical evaluation and ancillary testing, such as ultrasonography, which often identifies fibroids as the

primary clinical indication for hysterectomy. A similar explanation applies to leiomyoma and endometrial hyperplasia; despite being common histopathological findings in AUB patients in this study, these conditions did not show a significant association with AUB as a clinical indication in this study. This discrepancy may arise from cases being classified under the clinical indication of fibroids. Coupled with the high incidence of hyperplasia in this study sample, as the most prevalent pathology, endometrial hyperplasia may account for comparable findings between patients operated on for AUB and those operated on for other indications.

The correlation between pre-hysterectomy ultrasound diagnosis and histopathological findings was analyzed. Among 67 cases diagnosed as fibroid by ultrasound, 65 (98.5%) were confirmed histopathologically ($p < 0.001$). This finding aligns with studies by Kaushik et al. and Smita et al., which evaluated the concordance between ultrasonographic and histopathological diagnoses in hysterectomy specimens from AUB patients. Ultrasound is considered the diagnostic modality of choice for fibroids in symptomatic patients due to its high sensitivity, specificity, cost-effectiveness, accessibility, and ease of use.[138][139]

For adenomyosis, histopathology confirmed 87 cases, of which only 17 were initially suspected via ultrasound. Among these, 14 (82.4%) were histologically validated ($p = 0.003$). These findings are consistent with Sam et al., where ultrasound demonstrated high specificity (91.8%) but low sensitivity (36.8%) for adenomyosis detection. Similarly, Kaushik et al. reported sensitivity and specificity of 53.8% and 98.6%, respectively.[138][140]

Regarding endometrial carcinoma, ultrasound suspected 8 cases, but only 2 (25%) were histologically confirmed, with a total of 3 endometrial cancer cases diagnosed histopathologically. Sonographic features of endometrial cancer include increased endometrial thickness, generally a cutoff value of 4–5 mm indicating carcinoma in postmenopausal age, exhibiting 96% sensitivity and 61% accuracy based on a meta-analysis by Smith-Bindman et al. Heterogeneous echogenicity, often due to areas of hemorrhage and necrosis, and an irregular endometrial-myometrial interface suggesting myometrial invasion are also key indicators.[141]

This study aligns with findings from studies conducted in Erbil and Duhok, by **Intisar etal.**,[132] demonstrating that menstrual disturbances are the leading indication for hysterectomy and that benign pathologies are more prevalent than malignant counterparts. Uterine fibroids and adenomyosis were the most common benign conditions observed in hysterectomy specimens, with a peak incidence occurring between 41 and 50 years of age.[132] Clinicopathological correlation was 100% for cases clinically diagnosed as fibroids, while it was 40% for uterine prolapse. Notably, there was a clinical overestimation of malignancy in 10 cases, with histopathological confirmation in only one case of endometrioid endometrial carcinoma. Conversely, four cases of malignancy identified in this study were operated on for other indications. Three cases of adnexal lesions were suspected preoperatively by clinical evaluation, however; histopathology confirmed benign and malignant ovarian lesions in 12 cases. Among these, clinical correlation was achieved in only one case, which was a low-grade serous carcinoma of the ovary.

CONCLUSIONS AND RECOMMENDATIONS

6.1. Conclusions

1. Abnormal uterine bleeding (AUB) is the most common clinical indication for hysterectomy.
2. Benign pathologies, including uterine fibroids and adenomyosis, were identified as the predominant conditions in hysterectomy specimens.
3. Clinicopathological correlation was 100% in cases clinically diagnosed as fibroids.
4. Conditions such as chronic cervicitis and adenomyosis were frequently detected incidentally.

6.2. Recommendations

1. A prospective approach should be implemented, involving a larger sample size to enhance the accuracy and reliability of data regarding the association between clinical, histopathological features.
2. The establishment of a more advanced pathology request form system is recommended, incorporating detailed documentation of patient demographics, parity, presenting complaints, medical and gynecological histories, and drug histories, to facilitate the correlation of histopathological findings with the clinical background of patients.
3. Immunohistochemical studies employing a range of immunostainings, along with their correlation to the clinical and pathological characteristics of malignant uterine and adnexal tumors, should be prioritized to provide comperehensive insights.
4. Further studies should be incorporated to evaluate abnormal uterine bleeding (AUB), as it has been recognized as the most frequent indication for hysterectomy. Such studies should incorporate a larger sample size, an extended study duration, and more comprehensive parameters. These parameters should include the association of AUB with perimenopausal and postmenopausal age groups, patient parity, and the role of ultrasound in identifying the underlying cause of AUB. Furthermore, strict adherence to the International Federation of Gynecology and Obstetrics (FIGO) classification system for underlying causes of AUB (PALM-COEIN) is essential for accurately categorizing study participants.

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الخلاصة

المقدمة:

تتعلق عملية استئصال الرحم الجراحة النسائية الأكثر شيوعًا التي تجرى للنساء، وتعد عملية جراحية من حيث تخفيف الأعراض، ورضا المريضات، بالإضافة إلى توفير علاج نهائي للعديد من الأمراض التي تصيب الرحم والمناطق المجاورة للرحم ، مثل الأورام الليفية، غزارة الطمث، داء العضال الغدي الرحمي، الانتباذ البطاني الرحمي، مرض التهاب الحوض، تدلي الرحم والمهبل، والأورام الخبيثة.

الهدف من الدراسة:

دراسة توافر الأمراض المختلفة التي تصيب الرحم والمناطق المجاورة للرحم في حالات استئصال الرحم وربط العمايير السريرية المرضية بالتشخيص المرضي النسيجي.

المواد وطريقة العمل:

تم إجراء دراسة مقطعية رصدية خلال الفترة من يناير 2023 إلى يناير 2024شملت تحليلًا رصديًا لـ180 مريضة تم اختيارهن عشوائيًا ممن خضعن لعملية استئصال الرحم. تم جمع الحالات التي وردت بين عامي 2022 و2023 في المختبر التعليمي بمديرية الإمامين الكاظمين(ع) الطبية لهذه الدراسة. تم جمع التقارير المرضية النسيجية والشرائح لكل حالة. بالإضافة إلى ذلك، تم جمع العمايير السريرية من (العمر، نوع استئصال الرحم، السبب السريري، التشخيص بالموجات فوق الصوتية) من التقارير المرضية وتقارير الموجات فوق الصوتية. في قسم الأمراض الطبية/جامعة النهرين، تم إعادة فحص الشرائح المصبوغة بـ H&E من قبل أخصائي الأمراض المشرف لمراجعة التشخيص.

النتائج:

تم فحص 180 عينة استئصال رحم، وكانت أعلى فئة عمرية بين 40-49 عامًا (49.4%)، وكانت أكثر الأعراض السريرية شيوعًا هي النزيف الرحمي غير الطبيعي .(AUB) (61.67%) كان التغير المرضي النسيجي الأكثر شيوعًا هو فرط تنسج بطانة الرحم (43.4%)، يليه الأورام الليفية الرحمية (31.7%) لحالات منفردة، و(23.3%) لحالات مزدوجة مع العضال الغدي. وكان التهاب عنق الرحم المزمن أكثر النتائج المرضية النسيجية العرضية شيوعًا في عينات استئصال الرحم.

الاستنتاج:

يُعد استئصال الرحم الإجراء الجراحي الاختياري الأكثر شيوعًا في طب النساء. وعلى الرغم من أن النتائج المرضية النسيجية تتطلب [illegible] السريرية، إلا أن بعض الحالات، مثل التهاب عنق الرحم المزمن والعضال [illegible]ضيًا. وبالتالي، من الضروري إجراء تقييم نسيجي شامل لجميع عينات [illegible] نظرًا عن مظهرها العام لضمان التشخيص الدقيق وتحسين الرعاية [illegible].

الكلمات المفتاحية: استئصال الرحم، العضال الغدي.

وزارة التعليم العالي و البحث العلمي

المجلس العراقي للاختصاصات الطبية