BENIGN DISEASES OF THE UTERUS
By

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Uterus

- Pear-shaped muscular organ
- 8 cm long
- 5 cm wide
- 3 cm thick
- Non-pregnant state
- Pelvic organ
Uterus

- Fundus
- Body
- Cervix opens into vault or fornice of vagina
- Fundus is the portion above entrance of uterine tubes
- Covered with peritoneum
- Body
- Triangular cavity
Cervix

- Supravaginal
- Isthmus is a circular borderline area between the body and cervix
- Isthmus is the supra vaginal portion of cervix lower uterine segment
- Intravaginal is surrounded by gutter fornices
- Posterior is deeper covered with peritoneum
Peritoneum

- Reflected from the superior surface of the bladder
- Junction of the supravaginal portion of the cervix and the body of the uterus
- Uterovesical pouch
- Covers body, fundus
- Covers the posterior surface body and the supravaginal portion of cervix
- Upper third of posterior surface of vagina
Peritoneum

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Uterus

- Serous layer
- Myometrium
- No submucous layer
- Endometrium
- Three layers
- Basal
- Spongy
- Compact at surface of uterine cavity
Myometrium
Myometrium

- Myometrium makes up bulk of uterine wall
- Blood vessels more evident in middle layer
Uterine Muscle
Blood Supply

- Uterine
- Ovarian
- Vaginal arteries
- Anterior and posterior arcuate run in middle layer
Blood Supply
Nerve Supply of Uterus

- Pain from cervix via parasympathetic S2,3
- Pain from body via sympathetic to T11 and T12
Lymphatics
Supports of Uterus

Upper
- Round ligament
- Broad ligament antevorted

Middle
- Transverse ligament
- Pubocervical
- Uterosacral

Lower
- Levator ani, coccygeus
- Perineal body
Round Ligament

- Round ligament and
- Ligament of ovary
- develop from the gubernaculum
- Side of uterus, junction fundus and body
- Inguinal canal to labium majus
- Anteversion
**Broad Ligament**

- Uterine tube lies in medial four fifths of free border of broad ligament
- Lateral one fifth
- Contains ovarian vessels
- Infundibulo-pelvic or suspensory ligament of ovary
Pubocervical Ligament

- Attached
- Anteriorly to posterior aspect of body of pubis
- Passes to neck of bladder
- Anterior fornix of vagina
Uterine Artery

- Uterine artery lies superior to the ureter at lateral fornix of vagina
- Base of broad ligament
Transverse Ligament

- Transverse or cardinal or mackenrodt ligament
- Thickening of visceral layer of pelvic fascia around uterine artery
- Lateral to medial in base of broad ligament
Uterine Tube

- Intramural
- Isthmus
- Ampulla
- Infundibulum
- Lined ciliated columnar epithelium
- Beats towards uterus
Uterine Tube

- Peritoneum
- Loosely attached to ampulla
- Tightly to isthmus
- Fimbria surrounding opening into peritoneal cavity
- Ovarian fimbria
BENIGN DIS. OF THE UTERUS CAN BE CLASSIFIED IN TERMS OF THE TISSUE OF ORIGIN:

THE UTERINE CERVIX

THE ENDOMETRIUM

THE MYOMETRIUM
THE ENDOMETRIUM
1. ENDOMETRIUM POLYPS:

**EPIDEMIOLOGY**

- Endometrial polyps usually occur in women in their 40s and 50s. [2]

- Endometrial polyps occur in up to 10% of women. [•]

- It is estimated that they are present in 25% of women with abnormal vaginal bleeding. [7]
ENDOMETRIUM POLYPS:

No definitive cause of endometrial polyps is known, but they appear to be affected by hormone levels and grow in response to circulating estrogen.

Risk factors include obesity, high blood pressure and a history of cervical polyps. Taking tamoxifen or hormone replacement therapy can also increase the risk of uterine polyps.

The use of an intrauterine system containing levonorgestrel in women taking may reduce the incidence of polyps.
ENDOMETRIAL POLYPS CAN BE SOLITARY OR OCCUR WITH OTHERS. They are round or oval and measure between a few millimeters and several centimeters in diameter.

They are usually the same red/brown color of the surrounding endometrium although large ones can appear to be a darker red.

The polyps consist of dense, fibrous tissue (stroma), blood vessels and glandlike spaces lined with endometrial epithelium.
IF THEY ARE PEDUNCULATED, THEY ARE ATTACHED BY A THIN STALK (PEDICLE).

IF THEY ARE SESSILE (HARD), THEY ARE CONNECTED BY A FLAT BASE TO THE UTERINE WALL. [10] PEDUNCULATED POLYPS ARE MORE COMMON THAN SESSILE ONES.
1. endometrium polyps:

**symptoms**

May cause abnormal bleeding, especially, intermenstrual bleeding
investigations
1. Transvaginal ultrasound.
2. Sonohysterography
3. Hysteroscopy
4. Endometrial biopsy
5. Curettage
1. **Medications**: such as progestin’s or gonadotropin-releasing hormone agonists.
2. The symptoms will return after the medications are stopped.
3. **Hysteroscopy** to remove any polyps that are found.
4. **Curettage**: to scrape the lining and remove any polyps.
5. **A hysterectomy**: in cases where cancer cells are found in the uterine polyps.
Prognosis

Endometrial polyps are usually **benign** although some may be **precancerous** or **cancerous**. [2] About 0.5% of endometrial polyps contain **adenocarcinoma** cells.

[13] Polyps can increase the risk of **miscarriage** in women undergoing **IVF** treatment. [2] If they develop near the **fallopian tubes**, they may lead to difficulty in becoming pregnant. [2]

Although treatments such as hysteroscopy usually cure the polyp concerned, recurrence of endometrial polyps is frequent. [6] Untreated, small polyps may regress on their own.
2. Asherman's syndrome:

Asherman's syndrome (AS), which is also referred to as intrauterine adhesions (IUA) or intrauterine synechiae, is an acquired uterine condition that occurs when scar tissue (adhesions) form inside the uterus and/or the cervix. [1]

It is characterized by variable scarring inside the uterine cavity, where in many cases the front and back walls of the uterus stick to one another. AS can be the cause of menstrual disturbances, infertility, and placental abnormalities.
ALTHOUGH THE FIRST CASE OF INTRAUTERINE ADHESION WAS PUBLISHED IN 1894 BY HEINRICH FRITSCH, IT WAS ONLY AFTER 54 YEARS THAT A FULL DESCRIPTION OF ASHERMAN SYNDROME WAS CARRIED OUT BY ISRAELI GYNECOLOGIST JOSEPH ASHERMAN. [2]

A NUMBER OF OTHER TERMS HAVE BEEN USED TO DESCRIBE THE CONDITION AND RELATED CONDITIONS INCLUDING: UTERINE/CERVICAL ATRESIA, TRAUMATIC UTERINE ATROPHY, SCLEROTIC ENDOMETRIUM, AND ENDOMETRIAL SCLEROSIS.
There isn't any one cause of AS. Risk factors can include myomectomy, Cesarean section, infections, age, genital tuberculosis, and obesity.

Genetic predisposition to AS is being investigated. There are also studies that show that a severe pelvic infection, independent of surgery may cause AS [4].

AS can develop even if the woman has not had any uterine surgeries, trauma, or pregnancies. While rare in North America and European countries, genital tuberculosis is a cause of Asherman's in other countries such as India.
**Signs and symptoms**

It is often characterized by a decrease in flow and duration of bleeding (absence of menstrual bleeding, little menstrual bleeding, or infrequent menstrual bleeding)\(^6\) and infertility.

**Menstrual** anomalies are often but not always correlated with severity: adhesions restricted to only the **cervix** or lower **uterus** may block menstruation. Pain during **menstruation** and **ovulation** is sometimes experienced and can be attributed to blockages.

It has been reported that 88% of AS cases occur after a **D&C** is performed on a recently **pregnant uterus**, following a missed or incomplete **miscarriage**, **birth**, or during an elective termination (abortion) to remove retained products of conception.
Diagnosis

The history of a pregnancy event followed by a D&C leading to secondary amenorrhea or hypomenorrhea is typical.

Hysteroscopy is the gold standard for diagnosis. Imaging by sonohysterography or hysterosalpingography will reveal the extent of the scar formation.

Ultrasound is not a reliable method of diagnosing Asherman's Syndrome. Hormone studies show normal levels consistent with reproductive function.
Fertility may sometimes be restored by removal of adhesions, depending on the severity of the initial trauma and other individual patient factors.

Operative hysteroscopy is used for visual inspection of the uterine cavity during adhesion dissection (adhesiolysis).

IUA frequently reform after surgery, techniques have been developed to prevent recurrence of adhesions. Methods to prevent adhesion reformation include the use of mechanical barriers (Foley catheter, saline-filled Cook Medical Balloon Uterine Stent, IUCD) and gel barriers (Seprafilm, Spraygel, autocrosslinked hyaluronic acid gel Hyalobarrier) to maintain opposing walls apart during healing, preventing the reformation of adhesions. Antibiotic prophylaxis is necessary in the presence of mechanical barriers to reduce the risk of possible infections.
• **ESTROGEN** FOLLOWED BY A PROGESTIN TO STIMULATE ENDOMETRIAL GROWTH AND PREVENT OPPOSING WALLS FROM FUSING TOGETHER.

• HOWEVER, THERE HAVE BEEN NO **RANDOMIZED CONTROLLED TRIALS** (RCTS) COMPARING POST-SURGICAL ADHESION REFORMATION WITH AND WITHOUT HORMONAL TREATMENT AND THE IDEAL DOSING REGIMEN OR LENGTH OF ESTROGEN THERAPY IS NOT KNOWN.
• As has a reported incidence of 25% of D&Cs performed 1–4 weeks post-partum, [27][9][32] up to 30.9% of D&Cs performed for missed miscarriages and 6.4% of D&Cs performed for incomplete miscarriages.

• In another study, 40% of patients who underwent repeated D&C for retained products of conception after missed miscarriage or retained placenta developed as...
IN THE CASE OF MISSED MISCARRIAGES, THE TIME PERIOD BETWEEN FETAL DEMISE AND CURETTAGE MAY INCREASE THE LIKELIHOOD OF ADHESION FORMATION DUE TO FIBROBLASTIC ACTIVITY OF THE REMAINING TISSUE.
• The risk of AS also increases with the number of procedures: one study estimated the risk to be 16% after one D&C and 32% after 3 or more D&Cs.\[17] However, a single curettage often underlies the condition.

• In an attempts to estimate the prevalence of AS in the general population, it was found in 1.5% of women undergoing hysterosalpingography HSG,\[36] and between 5 and 39% of women with recurrent miscarriage.

• After miscarriage, a review estimated the prevalence of AS to be approximately 20% (95% confidence interval: 13% to 28%).
PROGNOSIS:

IN NEXT PREGNANCY, •

PLACENTA ACCRETA[28] •

SECOND-TRIMESTER PREGNANCY LOSS,[29] •

AND UTERINE RUPTURE[30] ARE OTHER REPORTED COMPLICATIONS •

INCOMPETENT CERVIX •

PREGNANCY AND LIVE BIRTH RATE HAS BEEN REPORTED TO BE RELATED TO •
THE INITIAL SEVERITY OF THE ADHESIONS WITH 93, 78, AND 57% PREGNANCIES ACHIEVED AFTER TREATMENT OF MILD, MODERATE AND SEVERE ADHESIONS, RESPECTIVELY AND RESULTING IN 81, 66, AND 32% LIVE BIRTH RATES, RESPECTIVELY.[13] [28]
• THE OVERALL PREGNANCY RATE AFTER ADHESIOLYSIS WAS 60% AND THE LIVE BIRTH RATE WAS 38.9% ACCORDING TO ONE STUDY.

• AGE IS ANOTHER FACTOR CONTRIBUTING TO FERTILITY OUTCOMES AFTER TREATMENT OF AS. FOR WOMEN UNDER 35 YEARS OF AGE TREATED FOR SEVERE ADHESIONS, PREGNANCY RATES WERE 66.6% COMPARED TO 23.5% IN WOMEN OLDER THAN 35.
Figure 1. HSG of uterus with corporal adhesions (red bracket).
Septate Uterus
Wedge of fibrous tissue dividing uterine cavity.

Asherman’s Syndrome
Adhesions (band-like formations) crossing the lining of the uterus.

Bicornuate Uterus
Incomplete uniting of uterus.
3. MYOMETRIUM
**uterine fibroids:**

- **Pathology:**
  
  A fibroid is a benign tumor of uterine smooth muscle, termed a *leiomysoma.*
CLASSIFICATION
1. Submucous leiomyoma
2. Pedunculated submucous
3. Intramural or interstitial
4. Subserous or subperitoneal
5. Pedunculated abdominal
6. Parasitic
7. Intraligmentary
8. Cervical
Histology of fibroid

Interstitial (intramural)

Subserous

Pedunculated, subserous

Subserous, displacing tube

Pedunculated, submucous

Intraligamentary

Cervical

Submucous

Pedunculated, submucous, protruding through external os
FIGURE 44.1. Common types of leiomyomata.
Fibroids are often described according to their location in the uterus.
Clinical features:-
• Fibroids being detectable clinically in about 20% of women over 30 years of age.

• **Risk factors**
  1. Nulliparity
  2. Obesity
  3. positive family history
  4. African racial origin
  5. OCP
  6. depot DMPA injections may be associated with reduced risk
Abnormal uterine Bleeding

Pelvic pressure & pain

Reproductive Dysfunction
Common presenting complaints are

1. Menstrual disturbance;
2. Menorrhagia may occur, it is likely that only sub mucous fibroid distorting the endometrial cavity and increasing the surface area
3. Dysmenorrhea
•2. pressure symptoms especially;

Nerves pressure: backache

Bladder—frequency / retention / Difficulty in micturition / Incomplete bladder emptying / incontinence

Ureter—Unilateral ureteral obstruction; hydroureter, hydronephrosis

Bowel—constipation/tenesmus. / difficult defecation can caused by large posterior fibroid

Vessels: Varicosity or edema of the lower extremities.
3. Pain is unusual. Uncomplicated uterine fibroid usually do not produce pain.
Acute pain is usually caused by either:

1. **torsion of pedunculated fibroid**
2. **Red degeneration of fibroid**
3. **extrusion from the uterus** (*in submucous fibroid; the uterus contracts to try to deliver the fibroid through the cervical os*)
4. **Associated endometriosis**
5. **Adhesions to other organs.**
6. **Malignant changes** (*sarcomatous change*)
3. MALIGNANCY: • LEIOMYOSARCOMA: OCCUR IN LESS THAN 0.1 - 0.5%.
Fig. 18.18: Gross specimen of leiomyosarcoma
Effect of the fibroid on the pregnancy:

- Infertility

- Less successful results with in vitro fertilization in patients who have large submucosal fibroids.

- Ectopic pregnancy

- Abortion and premature labor:
- Malposition and malpresentation of the fetus
- Obstructed labour
- Cesarean section
- Placental abruption
- Red degeneration
- Torsion of a pedunculated fibroid.
- Postpartum hemorrhage, inertia of uterus & delayed involution.

- Myomectomy not done during pregnancy because bleeding may be profuse.

- resulting in hysterectomy.

- Rupture of myomectomy scar during pregnancy
Effects of pregnancy on fibroid

1. Red degeneration
2. Increased size in 20 to 30% cases
3. Torsion of a pedunculated fibroid, may cause gradual or acute symptoms of pain and tenderness.
4. Infection during puerperium
5. Expulsion
6. Necrosis
SUBMUCOUS MYOMA
1. Expectant management (Conservative management)
1. Expectant management (Conservative management) -

**Indication**

- In the absence of pain, abnormal bleeding, pressure symptoms
- Size < 12 weeks (of pregnancy size)
- The patient near menopause, at which time the leiomyomas will atrophy as estrogen levels fall.
• observation with periodic examination is appropriate

• **Bimanual examinations** every 3 to 6 months

• Follow-up with **pelvic ultrasound or MRI**
• **Endometrial biopsy** if abnormal bleeding.
• **Regular blood counts**: iron deficiency anemia is common with menorrhagia, and iron replacement may be required
2. Medical treatment
2. Medical treatment

Non-hormonal options: such as NSAIDs (e.g. mefenamic acid) and antifibrinolytics (e.g. tranexamic acid) are limited at treating symptoms of dysmenorrhea and heavy, prolonged bleeding and anemia.
**Hormonal options** include:

- combined oral contraceptive pills,
- progestins (medroxyprogesterone acetate, Mirena IUD, norethindrone acetate)
- mifepristone
- androgenic steroids (danazol and gestrinone)
- gonadotropin-releasing hormone (GnRH) agonists
- Antiprogesterones (Mifepristone): effective in shrinking fibroids at a low dose
Fig. 18.7: The levonorgestrel intrauterine system (Mirena)
3. surgery
Surgery
Myomectomy

involves the removal of single or multiple fibroid while preserving the uterus.
**Indications for myomectomy**

1. Large myomas (especially the submucosal or intramural type)
2. Any symptomatic fibroid (persistent uterine bleeding despite medical therapy, excessive pain or pressure symptoms)
3. Unexplained infertility with distortion of the uterine cavity by fibroid
4. Recurrent pregnancy wastage due to fibroid.
5. When IVF is indicated (especially if the myoma results in the distortion of the uterine cavity)
Hysterectomy
UTERINE CARCINOMA
<table>
<thead>
<tr>
<th>Type</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Adenocarcinoma</td>
<td>59%</td>
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<tr>
<td>Adenoacanthoma (adeno + squamous metaplasia)</td>
<td>21%</td>
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<tr>
<td>Adenosquamous carcinoma</td>
<td>7%</td>
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<tr>
<td>Clear cell carcinoma</td>
<td>6%</td>
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<tr>
<td>Papillary adenocarcinoma</td>
<td>5%</td>
</tr>
<tr>
<td>Secretory carcinoma</td>
<td>2%</td>
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<tr>
<td>Mixed type</td>
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Endometrial cancer is the most common pelvic genital cancer in women.

In the US the lifetime risk of developing endometrial Ca is 2.4% in white women & 1.3% in black.

It is a disease of postmenopausal women with a peak incidence in the 6th & 7th decade of life.
Only 2-5% occur before 40 years.

Prognosis is better than other Gynecological Ca due to early Dx --- 75% Dx Stage I.
Estrogen has been implicated as a causative factor.


Age 65-75 Y, only 2-5% < 40 Y

- Excessive endogenous / exogenous estrogens
  - Early menarche < 12 Y
  - Late menopause > 52 Y ➔ 2 X risk
- Nulliparity 2X > women with 1 child
  / 3X > women with ≥5
- Chronic anovulation as in PCO
Obesity → aromatization of adrenal androgens in fat tissue risk is 3X for Pt 21-50 pounds overweight

10 X for Pt > 50 P overweight

-Granulosa-thicka cell tumors of the ovary (a rare estrogen secreting ovarian tumor) → endometrial hyperplasia & Ca in 10% of Pt

-Cirrhosis of the liver → degradation of estrogen

-Endometrial hyperplasia
CLASSIFICATION
1-Hyperplasia without atypia (not premalignant)
  1-A-Simple
  - Microscopically ➔ crowding of the glands in the stroma
  - Commonly asymptomatic
  - 1% progress to Ca over 15 Y
  - 80% regress
1-B-Complex hyperplasia without atypia

- A complex crowded appearance of the glands with very little stroma

- Epithelial stratification & mitotic activity

- 3% progress to Ca over 13 Y

- 80% regress

- 85% reversal with progestin Rx
2-Hyperplasia with atypia (premalignant)

- The nuclei are irregular with coarse chromatin clumping & prominent nucleoli

- 50-94% regress with progestin therapy

- A higher rate of relapse after stopping Rx compared to that of lesions without atypia

2-A-Simple

- Progression to carcinoma occur in 8%

2-B- Complex

- Progression to carcinoma occur in 29%
PRESENTATION OF ENDOMETRIAL CA

- Abnormal vaginal bleeding ➔ most common 90%
- Premenopausal Pt ➔ usually c/o heavy flow at the time of menses
  may present with
    ➔ persistent intermenstrual bleeding
    ➔ pre or post menstrual spotting
    ➔ polymenorrhea that fails to respond to hormonal Rx
TREATMENT?