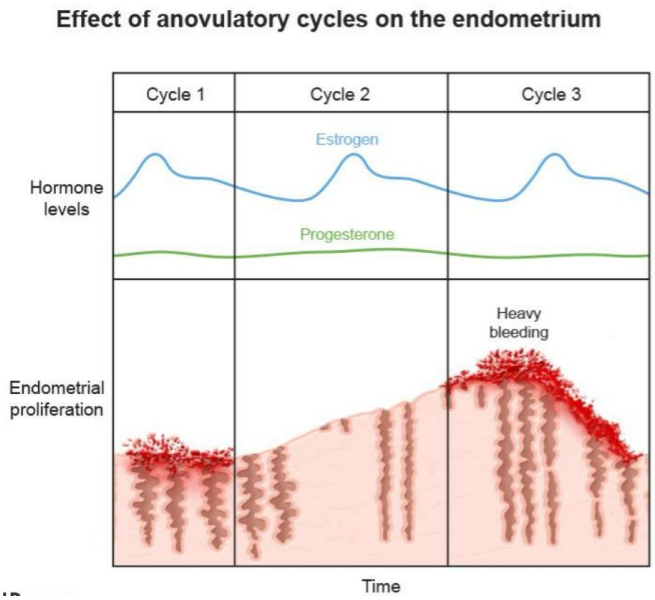
Gyne:

Puberty:

During the first year after menarche, adolescents often have **anovulatory cycles** with **heavy, irregular menstrual bleeding** due to an immature hypothalamic-pituitary axis. In ovulatory cycles, the corpus luteum produces progesterone after ovulation; progesterone differentiates the proliferative endometrium into secretory endometrium. As the corpus luteum degenerates, the decrease in progesterone leads to normal menses through a decrease in endometrial blood supply and shedding of the lining.

In contrast, anovulatory cycles do not produce progesterone (eg, no corpus luteum development) and there is no differentiation into secretory endometrium. Therefore, these patients have continued **unopposed estrogen stimulation** resulting in uncontrolled **proliferation of the endometrium**. Bleeding occurs when the endometrial lining becomes too thickened and unstable (eg, breakthrough bleeding).



Progesterone treatment helps stabilize this uncontrolled proliferation due to estrogen by causing differentiation into secretory endometrium. Cyclic progesterone withdrawal then causes menstruation.

So after menarche in the first year there might be anovulatory cycles

These lead to irregular and heavy menstrual bleeding to the unopposed effect of estrogen on the endometrium.

Physiologic leukorrhea is a white, odorless **cervical discharge** composed of cervical mucus, normal vaginal flora, and vaginal squamous epithelium. Increasing amounts of this normal vaginal discharge typically occurs **midcycle** (eg, 10-14 days after previous menses) as estrogen levels increase prior to ovulation, and then regresses. Physiologic leukorrhea presents **without manifestations of infection** such as pruritus, erythema, pain, or a malodorous discharge.

Prior to the diagnosis of physiologic leukorrhea, other causes of increasing vaginal discharge must be excluded. Polymorphonuclear leukocytes on microscopic examination are evidence of a local immune response (eg, inflammation) to infection. This patient has **rare polymorphonuclear leukocytes** on microscopy, ruling out all infectious etiologies as the cause of her discharge.

In women with ovulatory menstrual cycles, a dominant follicle (ie, a large cyst with multiple smaller cysts) is formed during the follicular phase (resulting in an **enlarged adnexa**); when the oocyte is released during ovulation, pain can develop due to **rupture of the follicle** and subsequent release of a small amount of blood and fluid that irritates the peritoneum. Most patients can be managed with reassurance and pain management (eg, nonsteroidal anti-inflammatory medications).

This is called **Mittelschmerz** which means -middle pain- and this is normal.

Menopause:

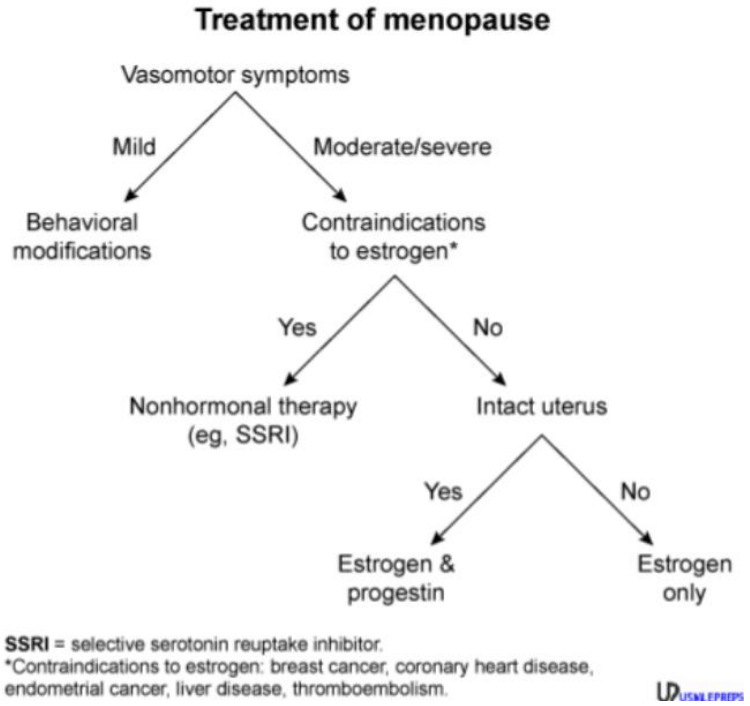
	Menopause
Clinical features	Vasomotor symptoms Oligomenorrhea/amenorrhea Sleep disturbances Decreased libido Depression Cognitive decline Vaginal atrophy
Diagnosis	Clinical manifestations ↑ FSH
Treatment	Topical vaginal estrogen Systemic hormone replacement therapy

Most women undergo menopause around age 51 due to age-related ovarian follicle depletion and decreased estrogen levels, resulting in vasomotor symptoms (eg, hot flashes, night sweats), vulvovaginal atrophy, and amenorrhea.

In general, menopause is a clinical diagnosis defined as cessation of menses for ≥12 months in women with previously regular menstrual cycles. In women with typical menopausal symptoms and cessation of menses for ≥12 months, no additional evaluation is required. However, in women **without previously normal menstrual cycles** (eg, prior hysterectomy, endometrial ablation), the diagnosis of menopause cannot be made clinically because vasomotor symptoms and vulvovaginal atrophy can be due to other etiologies (eg, thyroid disorder, malignancy). Therefore, these patients require an **elevated serum FSH level** for the diagnosis of menopause.

- **Bilateral salpingo-oophorectomy** in postmenopausal women may result in re-emergence of menopausal symptoms due to an abrupt *decrease* in circulating **androgens**.

  *while postmenopausal ovaries no longer produce estrogen, they **continue to produce androgens**, which are peripherally converted to estrone and estradiol*



Management of hot flashes depends on symptom severity and patient risk factors. Patients with mild symptoms that do not interfere with daily activities are managed with lifestyle modification (eg, wearing layers, weight loss). Severe hot flashes typically require pharmacologic therapy, and **hormone replacement therapy** (HRT) is the first-line treatment.

The only current indication for HRT is **vasomotor symptoms** in women age <60 who have undergone menopause within the past 10 years. HRT was previously used for chronic disease prevention, but it is no longer recommended in the prevention of coronary heart disease or osteoporosis due to the associated risks of HRT (eg, thromboembolism)

In patients with no contraindications to estrogen, **menopausal hormone therapy** (MHT) is first-line treatment.

- Patients with an intact uterus require estrogen-plus-progesterone MHT (eg, estrogen-progestin pills), which decreases the risk of endometrial cancer associated with unopposed estrogen.
- In contrast, **patients without a uterus** (eg, prior hysterectomy) can receive **estrogen-only** MHT (eg, transdermal estrogen patch), which is preferred in these patients because estrogen-plus-progesterone MHT has a small increased risk of breast cancer with long-term (>3-5 years) use.

	Genitourinary syndrome of menopause
Symptoms	Vulvovaginal dryness, irritation, pruritus Dyspareunia Vaginal bleeding Urinary incontinence, recurrent urinary tract infection Pelvic pressure
Physical examination	Narrowed introitus Pale mucosa, ↓ elasticity, ↓ rugae Petechiae, fissures Loss of labial volume
Treatment	Vaginal moisturizer & lubricant Topical vaginal estrogen

Atrophic vaginitis (or the genitourinary syndrome of menopause):

Estrogen maintains the moisture, blood flow, and collagen content (eg, elasticity, turgor) of the vulvovaginal tissues (eg, vagina, vulva, urethra). Patients with **low estrogen levels** eventually develop dryness and **decreased blood flow and elasticity** in these estrogen-dependent tissues.

Symptoms of estrogen deficiency are due to **thinning of the vulvar skin** (eg, irritation), narrowing of the vaginal introitus (eg, **dyspareunia**), and loss of natural lubrication (eg, **dryness**). Physical examination findings include pale, easily-denuded, retracted, **atrophic vulvovaginal epithelium** (eg, clitoral shrinkage). First-line treatment includes over-the-counter lubricants or moisturizers. Persistent or severe symptoms are treated with **vaginal estrogen**.

Both age-related ovarian follicle depletion (ie, menopause) and surgical removal of the ovaries (eg, bilateral salpingo-oophorectomy) result in an **estrogen deficiency** that affects the urogenital tissue and causes the associated urinary symptoms of atrophic vaginitis:

- Decreased collagen, elasticity, and blood flow in the bladder trigone and urethra (which are estrogen-sensitive tissues) result in **urogenital atrophy**, which can induce **urgency incontinence** (urgency followed by immediate

loss of urine), often causing wet undergarments and nocturia (as seen in this patient).

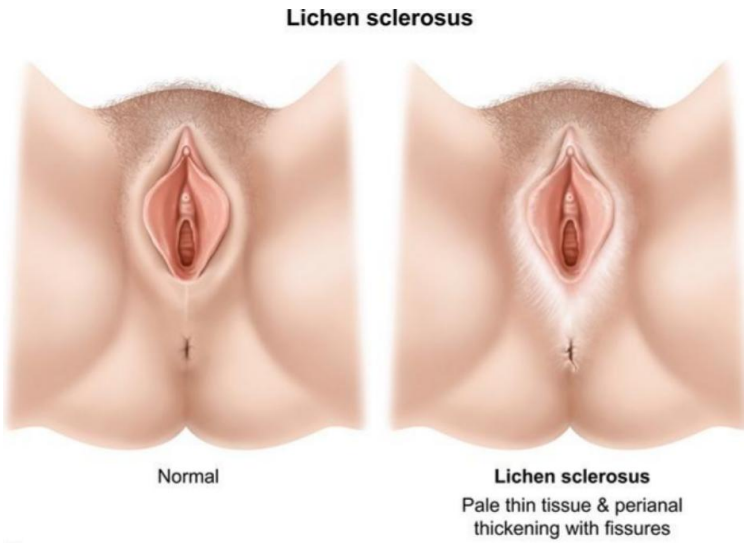
- Decreased glycogen content results in loss of vaginal lactobacilli and an elevated vaginal pH, which increase the risk of **recurrent urinary tract infections**. At the vulva, decreased glycogen content results in a thin, easily denuded vulvovaginal epithelium that causes **dysuria** when in contact with urine.

So in a post menopausal women with urge incontinence the decreased estrogen is the cause.

DDx:

Lichen planus is a chronic, inflammatory skin dystrophy that results in glazed, brightly erythematous, vulvar lesions with a purple hue that may be overlaid by white reticular lines (ie, Wickham striae).

Patients with lichen sclerosus have thin, wrinkled vulvar skin that can form into thickened white plaques that eventually obliterate the labia majora and minora, scarring the normal external landmarks. In contrast to atrophic vaginitis, lichen sclerosus does not affect the vagina.



Female sexual interest/arousal disorder is a sexual dysfunction commonly seen in **postmenopausal women**. It can cause significant distress and should not be assumed to be a normal consequence of aging.

Substance-induced and medical causes, including vaginal atrophy and dyspareunia, must be ruled out when making the diagnosis.

	Vulvar lichen sclerosus
Epidemiology	Prepubertal girls & perimenopausal or postmenopausal women
Clinical features	Thin, white, wrinkled skin over the labia majora/minora; atrophic changes that may extend over the perineum & around the anus Excoriations, erosions, fissures from severe pruritus Dysuria, dyspareunia, painful defecation
Workup	Punch biopsy of adult-onset lesions to exclude malignancy
Treatment	Superpotent corticosteroid ointment

vulvar lichen sclerosus, a chronic inflammatory condition that causes thinning of the vulvar skin in hypoestrogenic populations, such as **premenarchal girls** and postmenopausal women. In lichen sclerosus, white, atrophic papules form and eventually merge into plaques, leading to **thin, white vulvar lesions** and changes in vulvar architecture (eg, adherence of the labia at the midline). These lesions are chronically inflamed and can result in perianal and **vulvar pruritus**, at times so severe that it awakens affected individuals from sleep. Excessive scratching can result in excoriations, lichenification (ie, thickened skin), and edema of the labia. Lichenification of the perianal region can result in **anal fissures** and constipation.

Although biopsy is recommended in adults to exclude an underlying malignancy (eg, vulvar cancer), children have no associated malignancy risk and can be diagnosed clinically. First-line treatment is the same for adults and children: **superpotent topical corticosteroids** (eg, clobetasol).
Patients with chronic lichen sclerosus have continued inflammation and hyperplasia of the vulvar epithelium that can result in **malignant transformation** and development of a neoplastic lesion. This lesion typically develops over the labia majora and can become pruritic, friable, and ulcerated.

So in a post menopausal vulvar lesion U should do biopsy before diagnosing with lichen sclerosis.

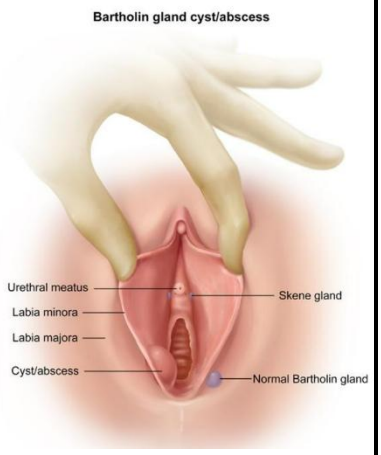
	Vulvar lichen planus
Clinical features	Women age 50-60 Vulvar pain or pruritus Dyspareunia Erosive variant (most common): ○ Erosive, glazed lesions with white border ○ Vaginal involvement ± stenosis ○ Associated oral ulcers Papulosquamous variant: ○ Small pruritic papules with purple hue
Diagnosis	Vulvar biopsy
Treatment	High-potency topical corticosteroids

vulvar lichen planus, a chronic inflammatory condition that typically occurs in postmenopausal women. The most common type of vulvar lichen planus is the erosive variant, in which chronic inflammation causes **desquamation and erosion** of mucosal surfaces, including the vulva, vagina, and oral cavity (as seen in this patient). Clinical features of erosive lichen planus include:

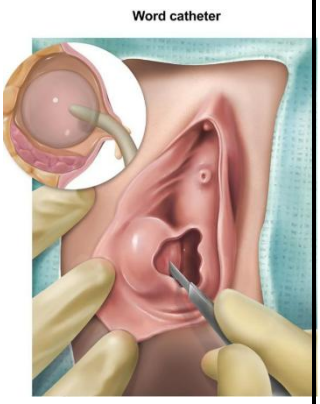
- **Glazed, brightly erythematous vulvar erosions** with a border of serpentine-appearing, white striae (ie, **Wickham striae**) that typically cause vulvar pain, pruritus, and dyspareunia.
- Acute vaginal inflammation that causes friable vaginal mucosa and a **serosanguinous vaginal discharge**; chronic inflammation can eventually result in stenosis of the vaginal introitus.
- Lacelike, reticular erosions on the gingivae and palate that cause painful oral ulcers and plaque formations on the tongue.

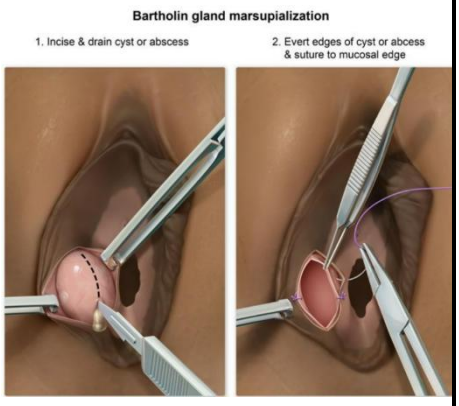
Diagnosis may be made clinically but should be confirmed with a **vulvar punch biopsy** because the clinical features of lichen planus may overlap with those of vulvar cancer. Patients with lichen planus require evaluation of all mucosal surfaces because erosions may occur in the absence of other symptoms. First-line treatment is with **high-potency topical corticosteroids**.

Bartholin duct cyst, which occurs due to blockage of the Bartholin gland duct. The Bartholin glands are located bilaterally at the posterior vaginal introitus and have ducts that drain into the vulvar vestibule at the 4 and 8 o'clock positions to provide vulvovaginal lubrication. The Bartholin glands are not palpable unless ductal blockage occurs, resulting in mucoid fluid buildup and cyst formation.



Women with small Bartholin duct cysts are often asymptomatic and may be diagnosed incidentally on routine examination. In contrast, larger cysts (as seen in this patient) cause increased tissue tension and friction, resulting in **vaginal pressure and discomfort** with sexual activity, walking, or sitting (all of which apply direct pressure on the vulva and cyst). On pelvic examination, a **soft**, **mobile**, **nontender**, **cystic mass** is palpated **behind the posterior labium majus** with possible extension into the vagina.

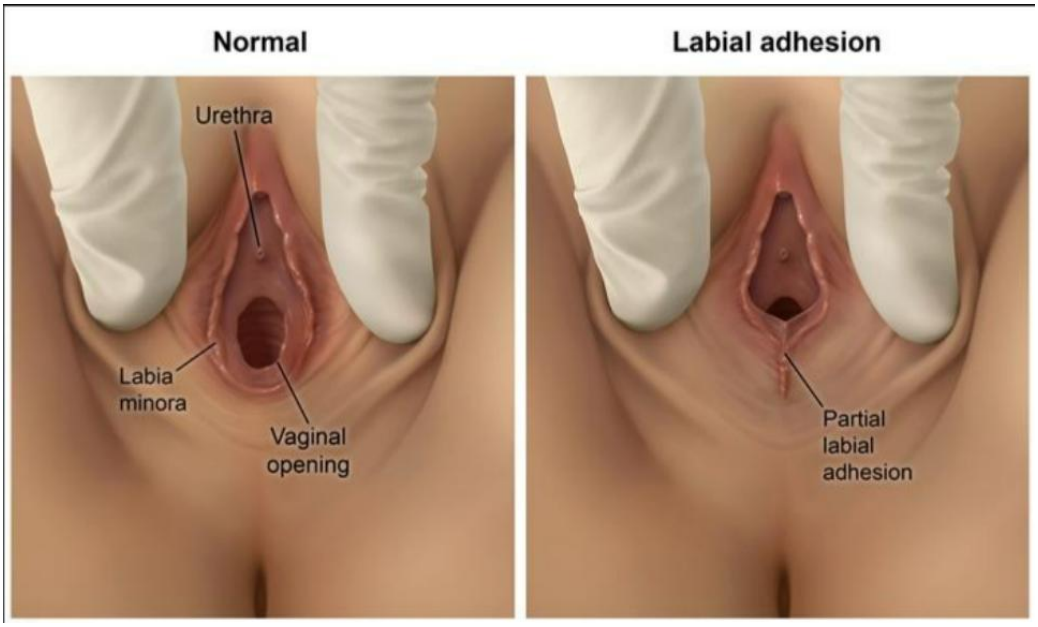




Asymptomatic Bartholin duct cysts in young women do not require intervention as most of the cysts drain spontaneously and resolve on their own. Therefore, **observation and expectant management** are recommended for these patients. In contrast, patients with symptomatic cysts or abscesses are treated with incision and drainage by placement of a Word catheter to reduce the risk of recurrence. Some women develop recurrent Bartholin cysts or abscesses and undergo a marsupialization procedure, which creates another point of drainage for the Bartholin gland.

Skene glands are bilateral paraurethral glands in the anterior vaginal vestibule. Skene gland cysts may form with duct obstruction but would be located lateral to the urethral meatus.

A Gartner duct cyst results from incomplete regression of the Wolffian duct during fetal development. These cysts appear along the lateral aspects of the upper anterior vagina. In contrast to Bartholin gland cysts, they do not involve the vulva.



Labial adhesions, or **fused** labia minora, typically affect **prepubertal girls** (with a peak incidence at age 2-3) due to **low estrogen production**. Other contributing factors may include chronic inflammation due to poor hygiene, skin irritation (eg, diaper dermatitis), and trauma (eg, straddle injury, sexual abuse).

Labial adhesions can be partial (involving only a small portion of the labia) or complete (with a small orifice for urine to come out). Partial adhesions, as seen in this patient, are often asymptomatic; however, some children develop pain or **pruritus**, which can lead to secondary **excoriations** and exacerbate adhesion development. Adhesions covering the urethral meatus can also cause an abnormal urinary stream and an increased risk for recurrent urinary tract infections.

Mild, asymptomatic labial adhesions require no treatment because up to 80% of labial adhesions resolve spontaneously. However, in patients with complete adhesions or symptomatic partial adhesions (such as this patient), first-line therapy is with **topical estrogen** cream.

Infertility:

Infertility	
Etiology	**Diagnostic test**
Male factor	• Semen analysis
Ovulatory function	• Midluteal phase (day 21) progesterone level
Ovarian reserve	• Day 3 FSH & estradiol levels • Clomiphene citrate challenge test • Antral follicle count • Antimüllerian hormone
Fallopian tube patency	• Hysterosalpingogram
Uterine cavity evaluation	• Sonohysterogram

Evaluate Men first

• **Infertility** in patients age **< 35** is defined as the failure to achieve pregnancy after **12 months** of regular, unprotected sexual intercourse.

*for women age **> 35**, infertility evaluation can begin after **6 months***

Female factor infertility is generally categorized into:

- **anatomic** disorders (eg, fallopian tube obstruction)
- **ovulatory** dysfunction (eg, anovulation)
- **endocrine** disorders (eg, hyperprolactinemia).

Patient history helps to identify the most likely underlying etiology.

Ovulatory Function

The Concept: This determines if and when an egg is being released. Without ovulation, pregnancy is impossible regardless of how many eggs remain.

The Test: Mid-luteal Progesterone

- **When:** Approximately 7 days before expected menses (Cycle Day 21 in a 28-day cycle).
- **Why:** After ovulation, the empty follicle (corpus luteum) pumps out progesterone. High progesterone = proof that rupture occurred.
- **Values:**
  - **>3 ng/mL:** Ovulation confirmed.
  - **<3 ng/mL:** Anovulation or poor timing of test.

Step 2 Pearls:

- Anovulation workup = **TSH + Prolactin**. Rule out thyroid dysfunction and pituitary tumor.
- Most common cause = **PCOS**. Diagnosis requires 2 of 3: oligo-ovulation, hyperandrogenism, polycystic ovaries.
- Treatment = **Letrozole** (now first-line over Clomid for PCOS).

Ovarian Reserve

The Concept: This quantifies the remaining egg pool (quantity) and egg health (quality). It predicts how well ovaries will respond to stimulation medications.

The Tests:

1. **Day 3 FSH + Estradiol**
   - Low ovarian reserve = Brain pumps out more FSH to stimulate weak ovaries.
   - **FSH >10** = Diminished reserve.
   - **Critical Trap:** If Estradiol is high (>60-80), it can suppress FSH and give a falsely normal result. Always check both.
2. **AMH (Anti-Müllerian Hormone)**
   - Produced by small growing follicles.
   - **<1 ng/mL** = Diminished reserve (low egg count).
   - **>4 ng/mL** = PCOS (excess follicles).
   - **Advantage:** Stable throughout cycle; can draw any day.
3. **Antral Follicle Count (AFC)**
   - Ultrasound count of small follicles (2-10 mm) in early follicular phase.
   - **<5** = Diminished reserve.

The Clinical Distinction You Must Know

Condition	Ovulatory Function	Ovarian Reserve	The Takeaway
PCOS	Bad (irregular/anovulatory)	Good (high AMH, high AFC)	Lots of eggs, but they won't release. Treat with ovulation induction.
Advanced Age	Good (regular menses)	Bad (low AMH, high FSH)	Eggs are running out. Don't delay IVF or consider donor eggs.

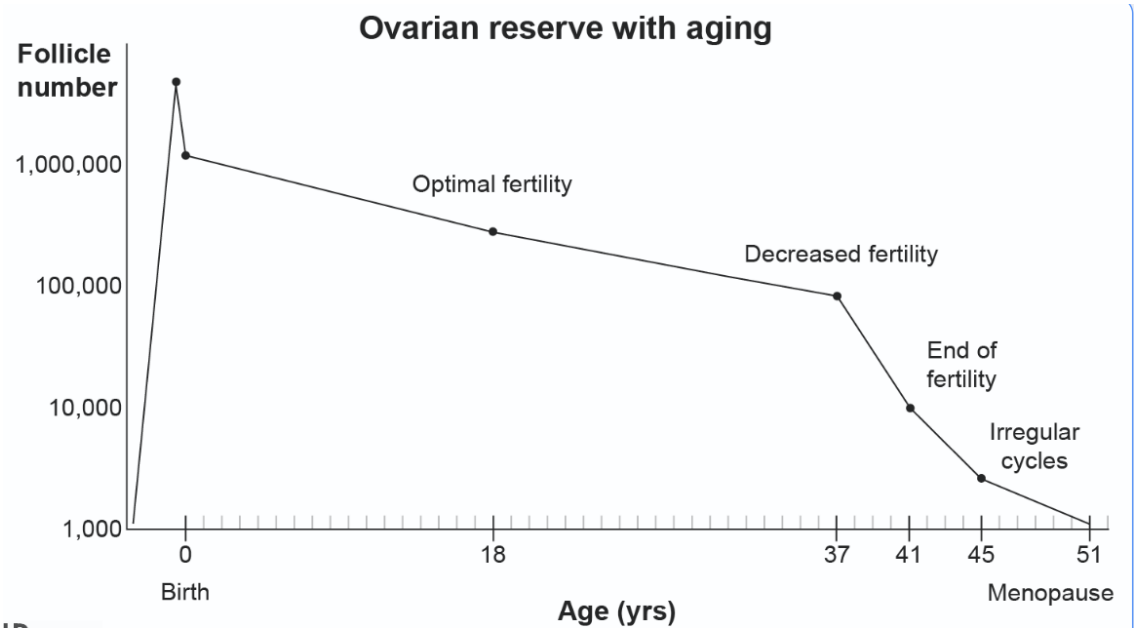
Clomiphene challenge test is no longer used.

- What is the likely *diagnosis* in a 38-year-old woman that presents with **infertility** despite a regular menstrual cycle and normal physical exam?

Decreased ovarian reserve

characterized by decreased oocyte number and quality; sharp decline in conception is notable after age 37

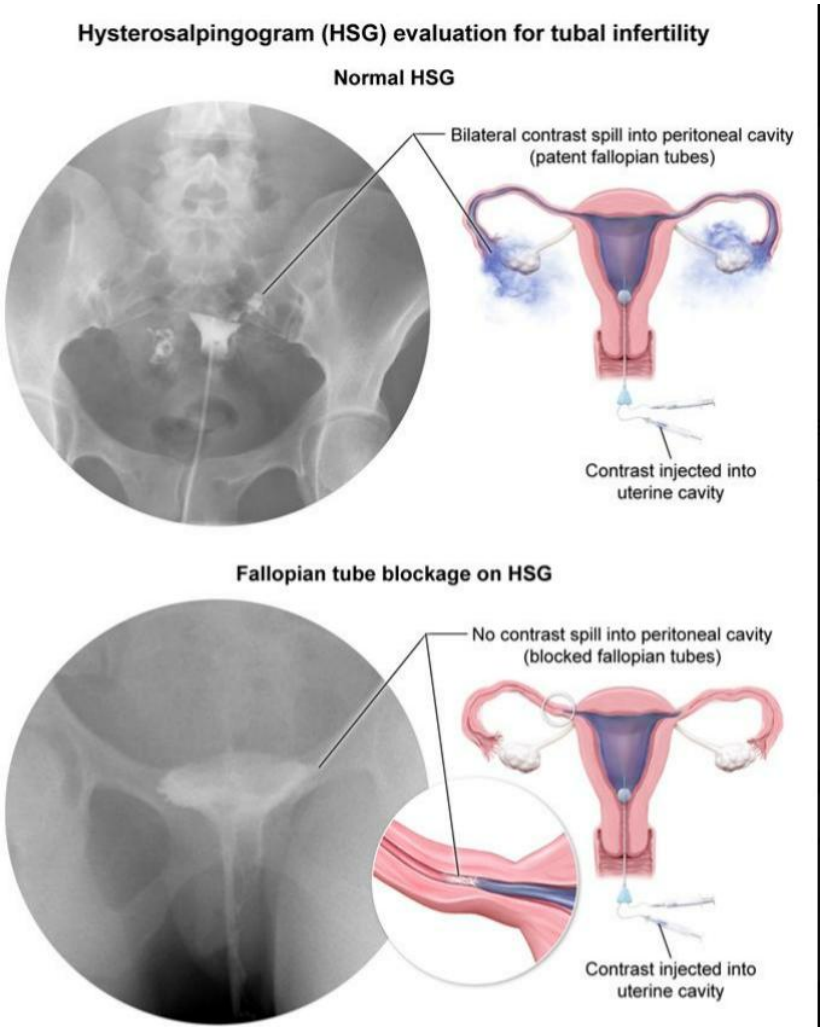
This is different from primary ovarian insufficiency.



Women are born with a lifetime supply of oocytes, and a sharp decline in conception rates is notable at age 35. Due to this decline, lack of conception after ≥6 months of unprotected intercourse in women age ≥35 is considered **infertility**. In women with regular menstrual cycles, infertility can occur due to **diminished ovarian reserve**, characterized by **decreased oocyte number and quality**. **Regular menstrual periods** still occur due to continuing ovulation, but fecundability (ie, conception rate) decreases due to diminished oocyte quality.

As ovarian reserve and function decline, estradiol and inhibin production decreases, and the normal negative feedback mechanism is suppressed. This causes FSH levels to become increasingly elevated as ovarian function decreases. Therefore, day 3 (early follicular phase) FSH testing can be performed to assess ovarian function. Assisted reproductive techniques (eg, in vitro fertilization, oocyte/embryo donation) are available to couples with age-related infertility.

In primary ovarian insufficiency there is amenorrhea, hot flashes, and vaginal atrophy (eg, minimal rugation).



The first-line test for suspected **tubal factor infertility** is a **hysterosalpingogram** (HSG), which infuses radiocontrast into the uterus under fluoroscopy. Because the uterus is contiguous with the fallopian tubes and thus the peritoneal cavity, HSGs in patients with normal, patent fallopian tubes show bilateral intraperitoneal spill of contrast. **No intraperitoneal spill of contrast** from 1 or both tubes is consistent with tubal factor infertility.

	Ovarian hyperstimulation syndrome
Pathophysiology	↑ hCG enhances ovarian vascular permeability Acute fluid shift to extravascular space
Clinical features	Ascites Respiratory distress Hemoconcentration Hypercoagulability Electrolyte imbalances Multiorgan failure (eg, renal failure) Disseminated intravascular coagulation
Evaluation	Fluid balance monitoring Serial CBC, electrolytes Serum hCG Pelvic ultrasound Chest x-ray Echocardiography
Management	Correct electrolyte imbalances Paracentesis &/or thoracentesis Thromboembolism prophylaxis

ovarian hyperstimulation syndrome (OHSS), a rare but life-threatening complication of **ovulation induction**. Ovulation trigger agents (eg, hCG injection) stimulate artificial maturation of multiple ovarian follicles in patients undergoing infertility treatment and can cause an exaggerated ovarian response (eg, **bilateral enlarged, cystic ovaries** with multiple follicles) as well as overexpression of vascular endothelial growth factor (VEGF), causing increased vascular permeability and capillary leakage. This causes massive extravascular fluid shifts (eg, **third spacing**) and VEGF leakage into the intraperitoneal cavity, leading to **ascites** and **abdominal distension**.

Affected patients typically develop OHSS 1-2 weeks after ovulation induction. Symptoms include nausea, vomiting, and abdominal pain. Other features include **pleural effusions** (eg, tachypnea, decreased breath sounds) and **intravascular volume depletion** (eg, tachycardia, **hemoconcentration**, leukocytosis) due to third spacing, which in severe cases can result in thromboembolism, renal failure, and death.

Ovulation induction

The use of medications to stimulate development of a single dominant follicle in patients with anovulation.

- **Oral medications**
  - Options
    - Letrozole: first-line in PCOS
    - Clomiphene citrate
- **Pulsatile GnRH**: may be used for functional hypothalamic amenorrhea
- **Exogenous gonadotropins**
  - Indications
    - hypogonadotropic hypogonadism
  - Options: recombinant FSH, LH, and/or hCG

Ovarian hyperstimulation syndrome (OHSS) [27][28]

- **Definition**: a potentially life-threatening complication of ovulation induction or ovarian stimulation with exogenous human chorionic gonadotropin (hCG)
- **Pathophysiology**
  - Exogenous hCG is thought to be responsible for the massive luteinization of the ovarian granulosa cells
  - ↑ Release of vasoactive mediators (e.g., VEGF) that induce an increase in capillary permeability and consequent third spacing into the abdominal cavity
- **Clinical features**: onset between 3 days (early onset) and ≥ 9 days (late onset) after hCG administration
  - Abdominal pain and distention
  - Nausea and/or vomiting
  - Weight gain
  - Ascites
- **Diagnostics**
  - Laboratory analysis
    - CBC (↑ Hct, leukocytosis)
  - Transvaginal ultrasound: ascites and ovarian enlargement

- **Management**
  - Mild to moderate OHSS (usually early onset): Manage on an outpatient basis.
    - Limit physical activity.
    - Pain management
    - Daily monitoring of body weight
  - Severe OHSS (usually late onset)
    - Hospitalization
    - Multidisciplinary management approach: supportive care (e.g., cautious IV fluids), monitoring, and prevention of complications
- **Complications**
  - Sepsis
  - Thromboembolism

Amenorrhea:

Types of Amenorrhea:

Primary Amenorrhea (pathological)

• She never got menses before.

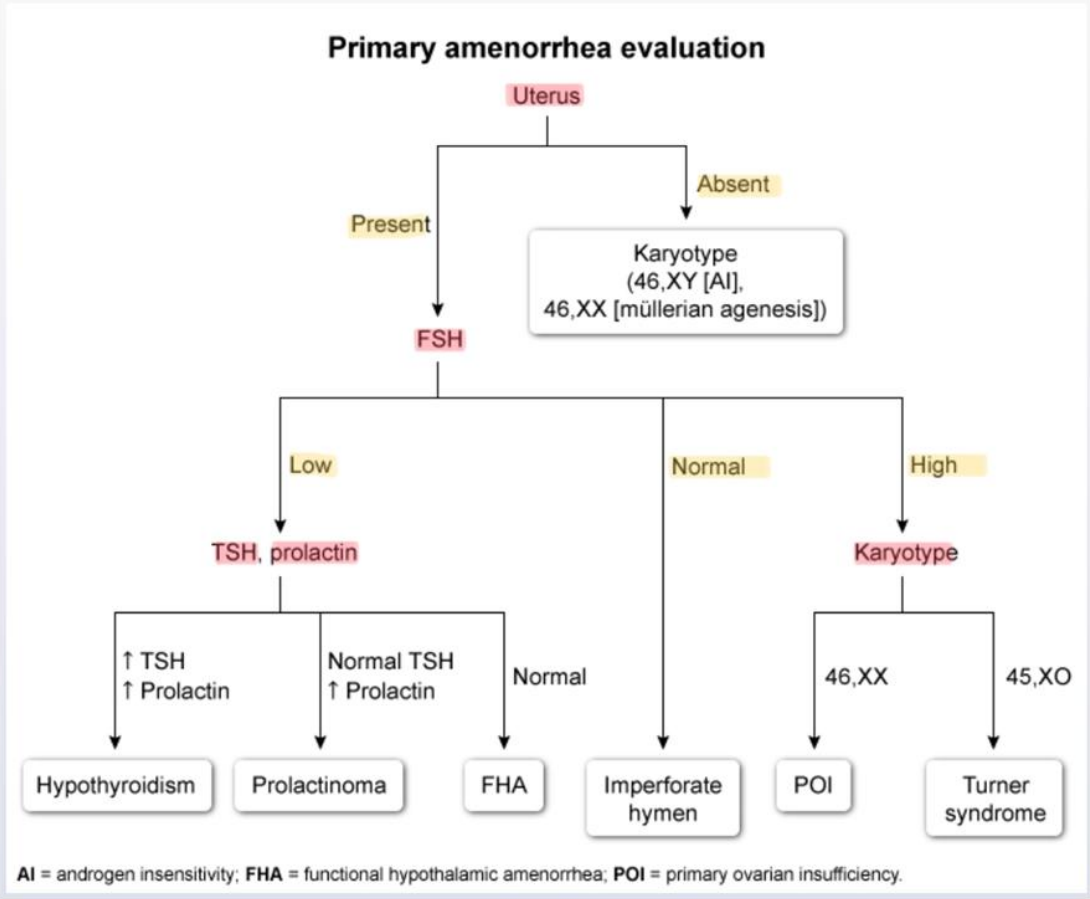
• Criteria:

o 14-year-old girl without menses and without secondary sexual characteristics.

o 16-year-old girl without menses but with secondary sexual characteristics.

Secondary Amenorrhea (pathological)

• lack of menses ≥3 months in women with previously regular cycles or ≥6 months in those with previously irregular cycles.



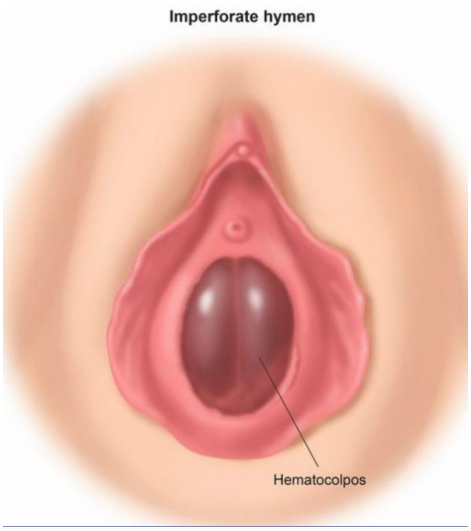
First Step: **Pelvic USG** to check for **Uterus**

- ↑ **FSH** → karyotyping (Turner's)
- ↓ **FSH** → MRI (**Craniopharyngioma**, **Kallmann's**)

	Imperforate hymen
Pathogenesis	Incomplete degeneration of hymen
Clinical features	Cyclic lower abdominal pain Bulk symptoms (defecatory & urinary dysfunction) Primary amenorrhea Suprapubic mass (uterus) Blue-tinged vaginal mass
Management	Hymenal incision & drainage

imperforate hymen is a common anatomic cause of **primary amenorrhea**. This occurs when the hymen fails to fenestrate during embryonic development. Infants may present with a bulging membrane due to mucus collection, but this typically resolves and patients remain asymptomatic until menarche. Adolescent patients with an imperforate hymen typically present with **cyclic lower abdominal pain** in the absence of apparent vaginal bleeding. When menstruation occurs, blood collects in the vagina behind the hymenal membrane (eg, hematocolpos). The enlarging blood collection with each menstrual period causes increasing pressure on the surrounding pelvic organs, resulting in lower back pain, pelvic pressure, or defecatory rectal pain.

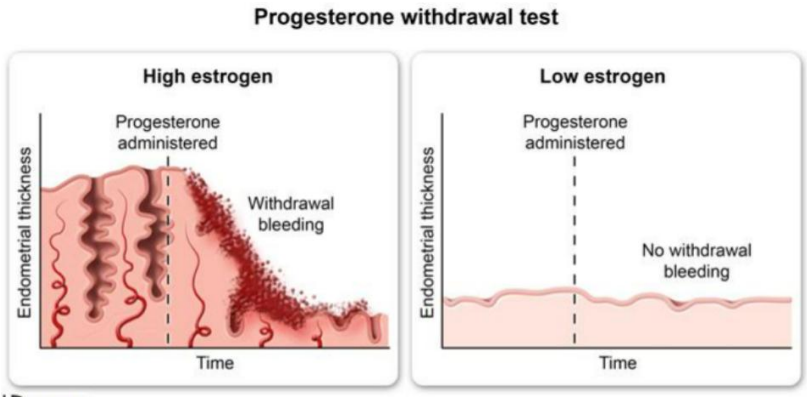
Pelvic examination typically reveals a blue, **bulging vaginal mass** or membrane that swells with increased intraabdominal pressure (eg, Valsalva). External genitalia and pubic hair development is normal. Treatment is with incision of the hymen and drainage of the hematocolpos. Patients with abnormal genital tract development should be evaluated for associated renal abnormalities with renal ultrasound.



	Primary ovarian insufficiency
Clinical features	Amenorrhea at age <40 Hypoestrogenic symptoms (eg, hot flashes) ↑ FSH ↓ Estrogen
Major causes	Idiopathic Turner syndrome (45,XO) Fragile X syndrome (*FMR1* premutation) Autoimmune oophoritis Anticancer drugs Pelvic radiation Galactosemia
Management	Estrogen therapy (with progestin if intact uterus)

FMR1 **= fragile X messenger ribonucleoprotein 1.**

In POI, the lack of ovarian function results in **low estrogen levels** and loss of negative feedback in the hypothalamic-pituitary-ovarian axis, resulting in elevated FSH levels (ie, hypergonadotropic hypogonadism). The low estrogen levels limit endometrial proliferation; therefore, there is no withdrawal bleed after a progesterone withdrawal test.



POI is common in women who are **fragile X syndrome (*FMR1* gene) premutation carriers** (ie, those with 55-200 CGG repeats). These women often have associated neurobehavioral manifestations (eg, anxiety, autism) and a family history of fragile X syndrome, as in this patient. This premutation causes

an *FMR1* mRNA overexpression, which likely has a cytotoxic effect on ovarian primordial follicles and results in **accelerated follicle depletion**. Due to this association, women with ovarian failure at age <40 and with no other obvious cause for POI (eg, Turner syndrome) are tested for *FMR1* gene mutations.

Management of POI includes estrogen-containing therapy to reduce the risk of comorbidities associated with low estrogen levels (eg, osteoporosis).

	Polycystic ovary syndrome
Clinical features	**Androgen excess** (eg, acne, male pattern baldness, hirsutism) Oligoovulation or anovulation (eg, **menstrual irregularities**) Obesity **Polycystic ovaries** on ultrasound
Pathophysiology	↑ Testosterone levels ↑ Estrogen levels LH/FSH imbalance
Comorbidities	Metabolic syndrome (eg, diabetes, hypertension) Obstructive sleep apnea Nonalcoholic steatohepatitis Endometrial hyperplasia/cancer
Treatment options	Weight loss (first-line) Oral contraceptives for menstrual regulation Letrozole for ovulation induction

Polycystic ovary syndrome (PCOS) is a common condition diagnosed in adolescents due to signs of **hyperandrogenism** and irregular menses. Clinical evidence of hyperandrogenism includes **severe nodulocystic acne**, male pattern baldness, and **hirsutism**. Although many patients with PCOS have biochemical evidence of hyperandrogenism with elevated serum levels of total testosterone, these laboratory values may be normal in some due to decreased levels of sex hormone-binding globulin, with elevated free testosterone levels instead. Therefore, the diagnosis requires either clinical or biochemical evidence of hyperandrogenism.

Women with PCOS typically have decreased progesterone secretion due to chronic anovulatory cycles. Therefore, these patients usually have a constant and unbalanced proliferation of the endometrium by estrogens. This unopposed estrogen stimulation places patients at increased risk for **endometrial hyperplasia and cancer**. Treatment with cyclic progesterone, estrogen/progestin oral contraceptives, or progesterone-releasing intrauterine devices protects the endometrium from hyperplasia and reduces cancer risk.

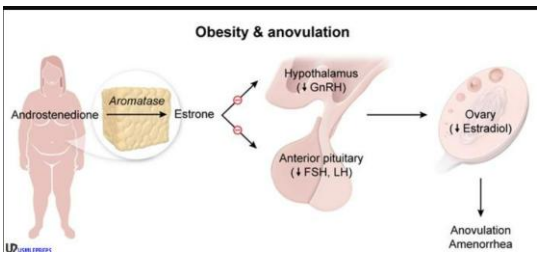
polycystic ovary syndrome (PCOS). Although patients with PCOS can have elevated serum testosterone or LH levels, serum concentrations can also be

normal with symptoms due to a relative imbalance in hormone levels (ie, LH/FSH ratio) rather than absolute values.

Most patients with PCOS are **obese** and have persistently high estrogen levels due to the peripheral conversion of androgens to estrogens in adipose tissue. These high estrogen levels suppress the hypothalamic-pituitary-ovarian axis by creating constant negative feedback. Therefore, **weight loss** is the first-line treatment to reestablish ovulation by decreasing the peripheral estrogen conversion.

If weight loss is unsuccessful, ovulation can be induced with **letrozole**, an aromatase inhibitor, which inhibits the conversion of androgens to estrogens. The resultant decreased estradiol secretion causes a positive feedback mechanism to the pituitary gland, thereby increasing and normalizing LH and FSH levels. This creates an LH surge that results in ovulation. Letrozole is the first-line ovulation induction agent in patients with PCOS due to higher live birth rates compared with clomiphene citrate.

Now if the patient doesn't meet the criteria for PCOS it is called anovulation due to obesity.

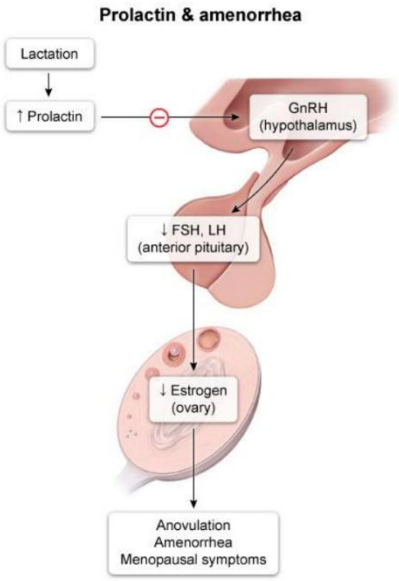


Excess adipose tissue affects the **hypothalamic-pituitary-ovarian axis** by 2 major mechanisms:

- Obesity causes increased insulin resistance and hyperglycemia, which decrease the production of sex hormone–binding globulin, causing elevated free androgen (eg, androstenedione) levels.
- The increased free androgens are **aromatized in the adipose tissue to estrone** (a type of estrogen), which leads to persistently elevated estrone levels.

In these patients, the high estrone levels affect GnRH pulses at the level of the hypothalamus, resulting in high-frequency, short-interval GnRH pulses. These

pulses preferentially produce LH, resulting in an imbalance in LH and FSH release from the anterior pituitary (although the overall change in concentration is minimal). The LH/FSH imbalance results in a lack of LH surge, causing **anovulation** and subsequent **abnormal uterine bleeding**. Treatment options include weight loss and combination oral contraceptives, which regulate menstrual cycles and protect the endometrium.



In breastfeeding patients, **elevated prolactin** levels **suppress GnRH release** from the hypothalamus, which causes **low FSH, LH, and estrogen levels**. Low estrogen levels inhibit ovulation (ie, lactational amenorrhea) and may induce menopause-like vasomotor symptoms (eg, hot flashes, night sweats) and **vulvovaginal atrophy**. Atrophic vulvovaginal changes cause dyspareunia due to decreased blood flow and collagen content, which lead to epithelial thinning (eg, loss of vaginal rugae) and decreased vaginal lubrication (eg, dryness).

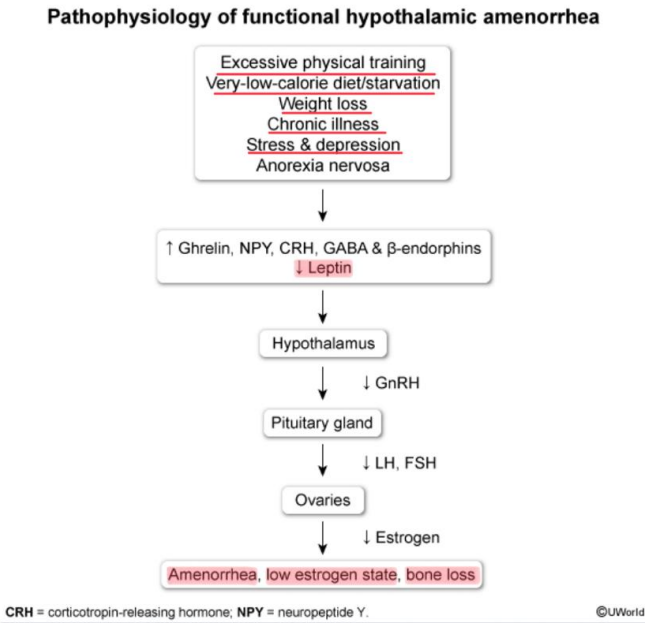
Treatment is supportive with **nonhormonal lubricants and moisturizers**; refractory cases may require **vaginal estrogen**. Symptoms typically resolve with cessation of breastfeeding and normalization of estrogen levels.

	Exercise-induced hypothalamic amenorrhea
Clinical presentation	Strenuous exercise Relative calorie deficiency Stress fracture Amenorrhea Infertility
Hormone levels	↓ GnRH ↓ LH & FSH ↓ Estrogen
Long-term consequences	↓ Bone mineral density ↑ Total cholesterol ↑ Triglycerides
Treatment	Increased calorie intake Estrogen Calcium & vitamin D

functional **hypothalamic amenorrhea** (eg, hypogonadotropic hypogonadism), which is commonly seen in women with the athlete's triad: amenorrhea, osteoporosis, and insufficient caloric intake.

Female athletes with intense levels of exercise are thought to have a **relative caloric deficiency** (even those with normal BMI) secondary to insufficient calorie replenishment compared to the amount of energy expended. Low energy stores (ie, low body fat) of muscular athletes (as seen in this patient) also alter multiple peripheral metabolic hormones (low leptin, high ghrelin), affecting GnRH secretion. These combined effects suppress the hypothalamic-pituitary-ovarian axis with **decreased** levels of **GnRH**, a subsequent **decrease** in **LH secretion**, and ultimately **estrogen deficiency**.

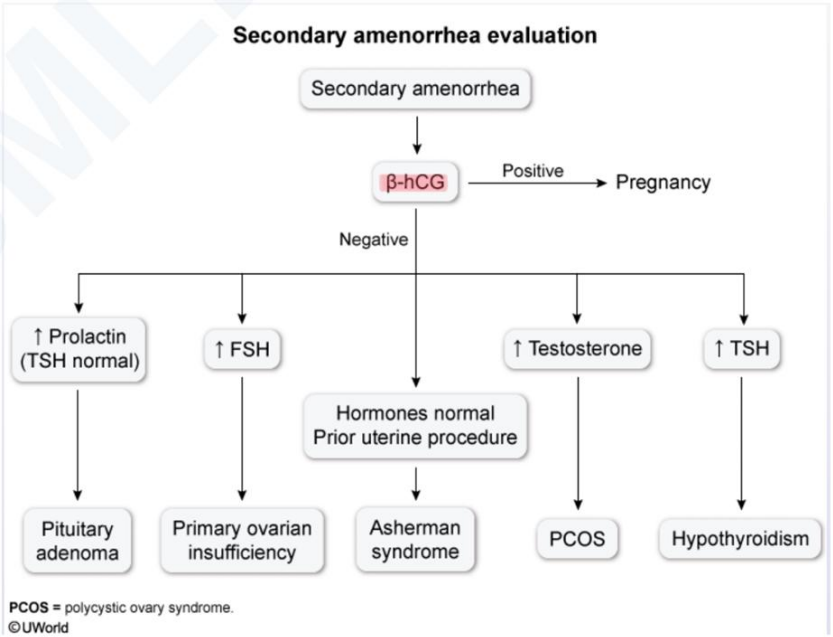
These patients are at increased risk for conditions associated with estrogen deficiency, including infertility and decreased bone mineral density (eg, osteopenia).

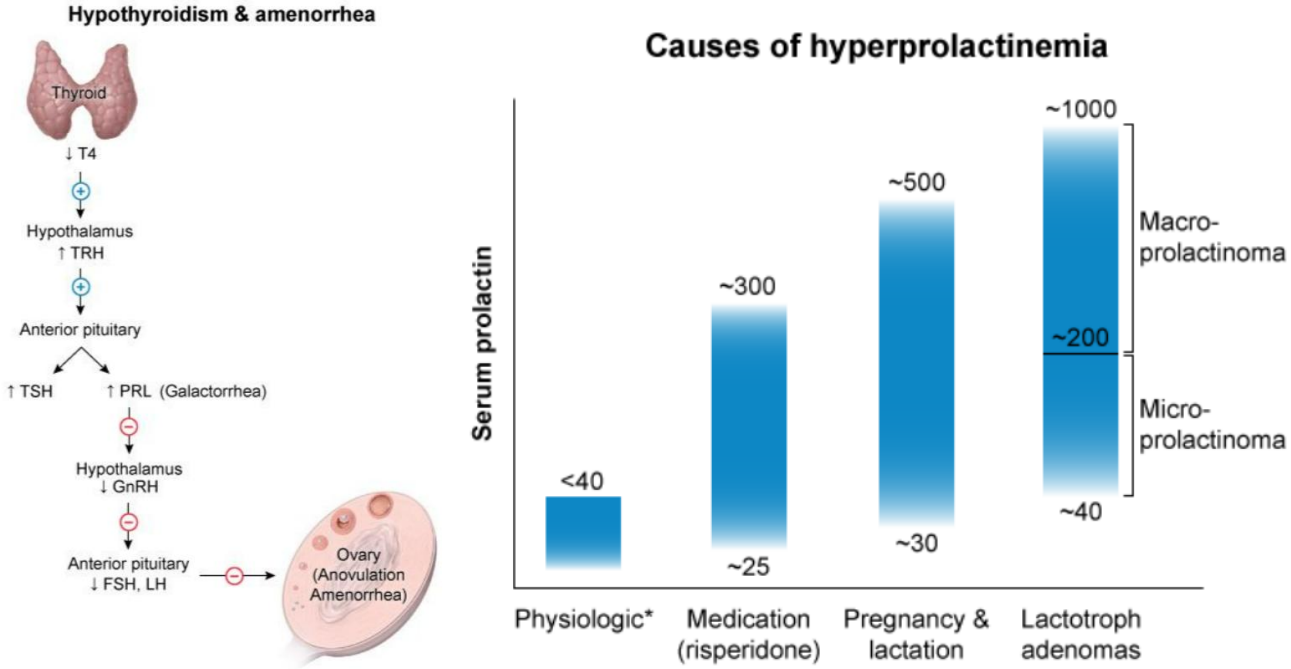


• Patients with **FHA** have **decreased estrogen levels** but **no vasomotor symptoms** (eg, hot flashes, night sweats) or **vaginal atrophy** due to **low basal levels of estrogen** produced by the normal (yet unstimulated) ovaries.

Patients with **amenorrhea**, **vasomotor symptoms**, and **vaginal atrophy** are more likely to have **primary ovarian insufficiency.**

Diagnostic findings of amenorrhea				
	FSH	**LH**	**Prolactin**	**TSH**
Ovarian failure	↑	↑	Normal	Normal
Functional hypothalamic amenorrhea	↓	↓	Normal	Normal
Asherman syndrome	Normal	Normal	Normal	Normal
Prolactinoma	↓	↓	↑	Normal
Hypothyroidism	↓	↓	↑	↑





Both **hypothyroidism** and **hyperprolactinemia** (individually or in conjunction) can cause **central hypogonadism**, manifesting with menstrual abnormalities and infertility, via multiple mechanisms:

- Hypothyroidism causes decreased responsiveness of the pituitary to GnRH, leading to decreased gonadotropin (ie, FSH, LH) secretion.
- Thyrotropin-releasing hormone, which is upregulated in hypothyroidism, stimulates the pituitary secretion of prolactin.
- Hyperprolactinemia, whether due to hypothyroidism or as an isolated disorder, further suppresses gonadotropin secretion.

In addition to mild to moderate hyperprolactinemia (serum prolactin <100 ng/mL), laboratory findings in patients with hypothyroid-related hypogonadism include **low FSH and LH** and low serum estradiol (low testosterone in men).

When patients have primary hypothyroidism and hyperprolactinemia, the thyroid disorder is usually the primary condition; hyperprolactinemia does not cause hypothyroidism. Therefore, **levothyroxine therapy** should be initiated first, which usually corrects prolactin levels and restores reproductive function. However, if prolactin levels are unusually high (ie, ≥100 ng/dL) or do not normalize with the treatment of hypothyroidism, prolactinoma should be suspected and may require cranial imaging (ie, MRI) and dopamine agonist (eg, cabergoline) therapy.

Hyperprolactinemia	
Clinical manifestations	Premenopausal women: oligomenorrhea/amenorrhea, infertility, galactorrhea Postmenopausal women: mass effect symptoms (eg, headache, visual field defects) if due to large adenoma Men: hypogonadism (decreased libido, erectile dysfunction), mass effect symptoms
Causes	Physiologic (eg, pregnancy, breastfeeding) Chest wall injury (eg, burns, herpes zoster) Prolactinoma Infiltrative pituitary/hypothalamic disorders (eg, malignancy, sarcoidosis) Medications (eg, antipsychotics, metoclopramide) Hypothyroidism Chronic kidney disease
Evaluation	Serum prolactin MRI of pituitary (if cause not known)

CKD causes hyperprolactinemia by decreased GFR which leads to a build up of prolactin but the main mechanism is that in CKD there is uremia which effect the hypothalamus by causing a decreased release of prolactin and it effects the pituitary by causing a decrease in the sensitivity of the dopamine receptors in the lactotroph cells.

These all cause hyperprolactinemia in CKD.

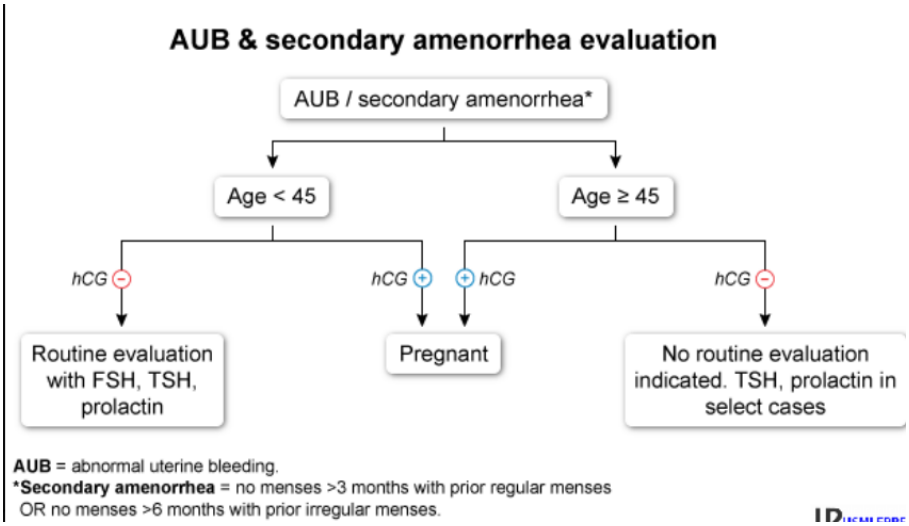
In chest wall injuries the irritation of the sensory fibers in the chest -especially around the nipple and areola- effect the hypothalamus and cause a decrease in dopamine.

This has the same mechanism as suckling of the baby and increased prolactin.

The degree of hyperprolactinemia often correlates with the underlying cause:

- Very high prolactin levels (eg, ≥200 ng/mL) usually indicate a prolactin-secreting pituitary macroadenoma (ie, prolactinoma or lactotroph adenoma).
- Moderate hyperprolactinemia can be due to hypothyroidism or renal insufficiency
- **Mild to moderate elevations** (<200 ng/mL), can occur due to disruption of inhibitory dopamine pathways by medications (ie, antipsychotics). They may also occur due to **small prolactinomas** (microadenomas) that may not present with classic mass symptoms (eg, headache, bitemporal hemianopsia).

Prolactinomas are the **most common** cause of hyperprolactinemia **in women of reproductive age**.



- Women age ≥45 with AUB and classic menopausal symptoms (eg, hot flashes) are most likely in menopause, a clinical diagnosis that requires no additional testing. These patients may benefit from hormone replacement therapy and patient education on menopause. Select patients within this age group who have additional signs of thyroid disorder (eg, tachycardia, goiter) or hyperprolactinemia (eg, galactorrhea) should undergo measurement of TSH and prolactin levels.
- In contrast, menopause is a less common cause of AUB in **women age <45**, even in patients with classic menopausal symptoms. An underlying **endocrine disorder causing AUB** must first be excluded. Therefore, patients in this age group require measurement of **FSH** (to assess for hypothalamic amenorrhea), **TSH** (to assess for thyroid disorder), and **prolactin** (to assess for hyperprolactinemia).

• What is the *next step* in management for a 45-year-old woman that presents with **insomnia**, **fatigue**, **weight gain**, **amenorrhea,** and an **enlarged uterus**?

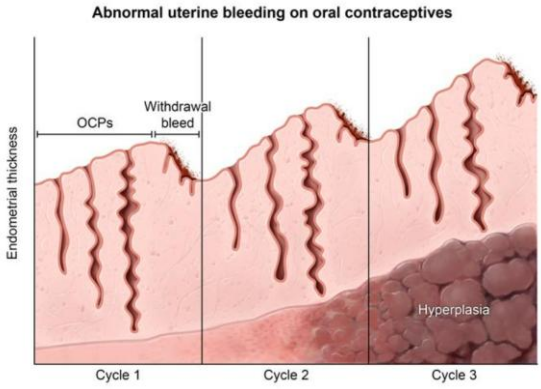
Measure hCG level

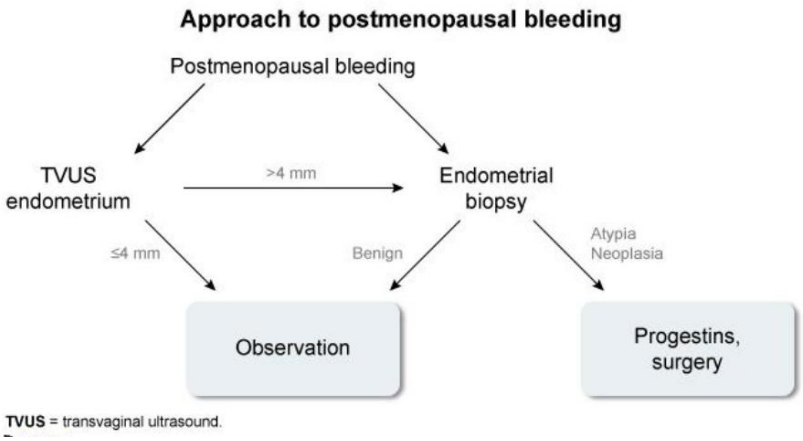
*all women of reproductive age **(~12 to 49)** with **amenorrhea** and **signs of pregnancy** should be evaluated with an hCG level*

In women **age <45** with AUB, the absolute risk of endometrial hyperplasia/cancer is low; therefore, they can be started on combination estrogen/progestin contraception (ie, medical management) without evaluation of the endometrium. The estrogen component regulates the menstrual cycle and builds up the endometrium; the progestin component sheds the endometrium.

However, patients who have continued irregular menstrual bleeding while on combination contraceptives (ie, **failed medical management**) require further evaluation. In such patients, the endometrial lining is likely too thick for the progestin to completely shed the endometrium during menstruation; as a result, the unshed endometrium continues to undergo dysregulated proliferation, which leads to an increased risk of **endometrial hyperplasia/cancer**. Therefore, patients age <45 with AUB who have failed medical management require an **endometrial biopsy**.

Other indications for endometrial biopsy in women age <45 include persistent (>6 months) AUB, obesity, or tamoxifen therapy, all of which increase the amount of unopposed endometrial estrogen exposure.





postmenopausal bleeding [PMB]

Because PMB is a common presenting finding for **endometrial hyperplasia or cancer** (particularly in older women with obesity), all patients with PMB require endometrial evaluation.

Endometrial evaluation for PMB is with either transvaginal ultrasound or **endometrial biopsy**. Endometrial biopsy can supply a histologic diagnosis, but it is an invasive and uncomfortable procedure. Therefore, PMB can also be evaluated initially with transvaginal ultrasound. Patients with an endometrial thickness ≤4 mm on ultrasound have a low likelihood of endometrial cancer and require no additional evaluation. In contrast, patients with an endometrial thickness >4 mm require an endometrial biopsy to assess for endometrial hyperplasia or cancer.

Patients with endometrial hyperplasia may be managed medically (eg, progestin) or surgically (eg, hysterectomy); those with endometrial cancer are typically managed surgically.

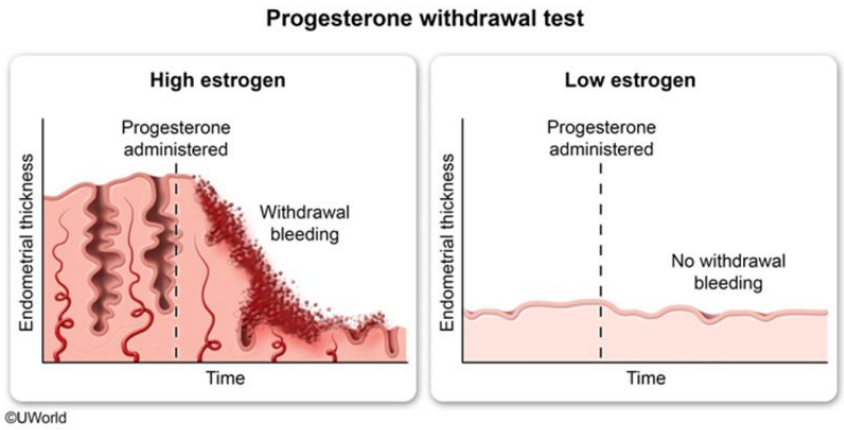
• Progesterone Withdrawal Challenge

Bleeding: **Anovulation**

No Bleeding: 1. **Low Estrogen Levels** 2. **Outflow Track Obstruction**

normally estrogen causes the endometrium to proliferate, which sloughs following withdrawal of progesterone

Do **E/P withdrawal test** to differentiate between these two.



• A patient with *secondary amenorrhea* with a **negative progesterone challenge** and **negative E/P challenge** is indicative of **outflow tract obstruction/scarring (*Asherman's*)**.

Next best step? **Hysterosalpingogram to ID lesion**

Treatment? **Hysteroscopic resection** of adhesions

*With Asherman's, pt. will still have **cyclic abdominal pain**, but no bleeding because blood is obstructed. Look for history of **D&C.***

Craniopharyngioma vs Kallmann's

Path:

- Kallmann's: **loss of GnRH → ↓ FSH/LH → ↓ E/P**
  - **+ anosmia**
- Craniopharyngioma: AP tumor → **↓ FSH/LH → ↓ E/P**
  - **+ bitemporal hemianopsia**

Presentation?

- **Primary Amenorrhea**
- **+ Uterus**
- **↓ secondary sex characteristics** (breast, pubic hair)

Diagnosis?

- **Cranial MRI** → mass = **craniopharyngioma** ("calcified cyst on CT")
- **Low FSH/LH** (*Turner's = ↑ FSH/LH = karyotype*)

Causes of hirsutism in women	
Etiology	**Clinical features**
Polycystic ovary syndrome	Oligomenorrhea, hyperandrogenism, obesity Associated with type 2 diabetes, dyslipidemia, hypertension
Idiopathic hirsutism	Normal menstruation Normal serum androgens
Nonclassic 21-hydroxylase deficiency	Similar to polycystic ovary syndrome Elevated serum 17-hydroxyprogesterone
Androgen-secreting ovarian tumors, ovarian hyperthecosis	More common in postmenopausal women Rapidly progressive hirsutism with virilization Very high serum androgens
Cushing syndrome	Obesity (usually of the face, neck, trunk, abdomen) Increased libido, virilization, irregular menses

💡 **Cushing syndrome** may be similar in presentation to **PCOS** (obesity, hirsutism, amenorrhea), but **PCOS does not cause muscle weakness nor easy bruisability**

Causes of hyperandrogenism in women	
Diagnosis	**Clinical features**
PCOS	Oligo-ovulation, clinical or biochemical hyperandrogenemia, polycystic ovaries on imaging, no evidence of another diagnosis
Nonclassic CAH	Oligo-ovulation, hyperandrogenemia, ↑ 17-hydroxyprogesterone levels
Ovarian/adrenal tumors	Older age, rapidly progressive symptoms, ↑ androgen levels (>3 times upper limit of normal)
Hyperprolactinemia	Amenorrhea, galactorrhea, ↑ prolactin levels
Cushing syndrome	Cushingoid features, nonsuppressible dexamethasone suppression test, ↑ 24-hour urinary free cortisol
Acromegaly	Excessive growth, ↑ GH & IGF-1 levels
CAH = congenital adrenal hyperplasia; **GH** = growth hormone; **IGF-1** = insulin-like growth factor 1; **PCOS** = polycystic ovary syndrome.	

Rapid Hirsutism < 1 Year = Tumor

- **Signs of Virilization: Deepening Voice, Cliteromegaly, Inc Muscle Bulk, Male Pattern baldness**

. **Voice deepening** is a common (and possibly irreversible) sign of frank virilization as excess androgens (eg, testosterone >150 ng/dL, dehydroepiandrosterone sulfate [DHEAS] >700 µg/dL) lengthen and thicken the vocal cords, thereby changing their acoustic frequency and changing the voice. Other clinical features of virilization include male-pattern baldness (eg, temporal hair loss), increased muscle bulk, and clitoromegaly.

Patients with virilization require evaluation for ovarian and adrenal sources of androgen production with total testosterone, 17-hydroxyprogesterone, and DHEAS levels.

The primary ovarian androgens are **testosterone**, **androstenedione**, and **dehydroepiandrosterone** (**DHEA**). The adrenals also produce these androgens as well as **DHEA sulfate (DHEAS)**. Therefore, women with a suspected androgen-producing tumor should be evaluated with serum **testosterone and DHEAS levels**:

- Elevated testosterone levels with normal DHEAS levels suggest an **ovarian source (more common).**
- Elevated DHEAS levels suggest an **adrenal tumor (far less common).**

Causes of hyperandrogenism in pregnancy	
Diagnosis	**Clinical features**
Placental aromatase deficiency	• No ovarian mass • High maternal & fetal virilization risk • Resolution of maternal symptoms after delivery
Luteoma	• Solid, unilateral/bilateral ovarian masses • Moderate maternal virilization risk; high fetal virilization risk • Spontaneous regression of masses after delivery
Theca lutein cyst	• Cystic, bilateral ovarian masses • Moderate maternal virilization risk; low fetal virilization risk • Spontaneous regression of masses after delivery
Sertoli-Leydig tumor	• Solid unilateral ovarian mass • High maternal & fetal virilization risk • Surgery required (2nd trimester or postpartum)

Pregnancy luteomas are **rare, nonneoplastic lesions of the ovary thought to be caused by the hormonal effects of pregnancy**

• What is the *recommended management* for a pregnant woman with a suspected **theca lutein cyst** secondary to a complete molar pregnancy?

Suction curettage of the hydatidiform mole

theca luteum cysts typically resolve following removal of the hydatidiform mole

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• What is the *recommended management* for a pregnant woman with a suspected **luteoma**?

Observation and expectant management

masses typically regress spontaneously after delivery; luteomas are occasionally complicated by ovarian torsion or symptoms related to mass effect (e.g. hydronephrosis)

• What is the likely *diagnosis* in an African-American woman at 20 weeks gestation that presents with **new-onset hirsutism** and **acne**? Pelvic ultrasound reveals an intrauterine gestation consistent with dates and **bilateral 8-cm solid ovarian masses**.

Luteoma

*versus **theca luteum cysts**, which typically arise due to high hCG levels (e.g. molar pregnancy or multiple gestation)*

• If **surgical removal** of an **ovarian tumor** is indicated during **pregnancy**, when should surgery take place?

After 10 weeks

- *Secretion of progesterone by corpus luteum is essential to maintain pregnancy*

- *Placenta takes over this function by 10th week*

Ovarian Hyperthecosis

• Ovarian hyperthecosis is a cause of **virilization** but is typically diagnosed in **postmenopausal women**.

Patients with this condition also typically have **signs of insulin resistance** (eg, hyperglycemia, acanthosis nigricans) and **low/normal LH and FSH levels**.

Typical ultrasound findings are **solid-appearing**, **enlarged ovaries.**

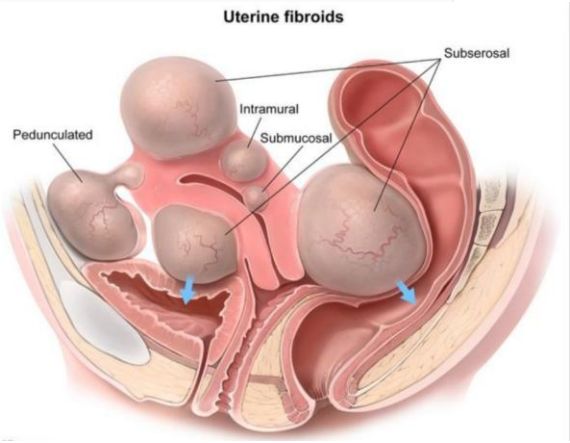
Ovarian hyperthecosis (OH) is a rare, non-neoplastic condition characterized by the presence of nests of luteinized theca cells within a hyperplastic ovarian stroma. It is often considered a severe or extreme form of **polycystic ovary syndrome (PCOS)** and primarily affects postmenopausal women, though it can occur in premenopausal individuals.

Key Characteristics

- **Androgen Production:** The specialized theca cells produce excessive amounts of testosterone.
- **Metabolic Links:** It is strongly associated with **insulin resistance**, obesity, hypertension, and an increased risk of type 2 diabetes.
- **Secondary Risks:** High androgen levels can be peripherally converted to estrogen, increasing the risk of **endometrial hyperplasia** and endometrial cancer.

Leiomyomas// leiomyomata uteri (ie, uterine fibroids):

	Uterine leiomyomas (fibroids)
Clinical features	Heavy, prolonged menses Pressure symptoms ◦ Pelvic pain ◦ Constipation ◦ Urinary frequency Obstetric complications ◦ Impaired fertility ◦ Pregnancy loss ◦ Preterm labor Enlarged, irregular uterus
Workup	Ultrasound
Treatment	Asymptomatic: observation Symptomatic: hormonal contraception, surgery



benign smooth muscle (myometrial) tumors that are relatively common (up to 25%) in adult women. These tumors can increase the surface area of the endometrium and the overall size and bulk of the uterus as well as compress surrounding structures; however, some patients have no symptoms and are **diagnosed incidentally** on physical examination or imaging (as in this patient's CT scan after a motor vehicle collision).

Indications for treatment of uterine leiomyomata include:

- Heavy, prolonged menses (particularly if associated with anemia).
- Chronic pelvic pain (eg, dyspareunia).
- Bulk symptoms (eg, pelvic pressure, hydronephrosis, constipation).
- Recurrent pregnancy loss.

Patients with these clinical features can be managed medically or surgically (eg, myomectomy).

In patients with **asymptomatic fibroids**, no additional management is indicated. They require **observation and reassurance only**.

depending on their size and location, fibroids can cause bleeding symptoms (eg, heavy menses), bulk symptoms (eg, pelvic pressure), or both.

Large fibroids commonly cause **mass effect (bulk symptoms)** on neighboring structures; therefore, patients may have increasing abdominal girth, **pelvic pressure**, and impaired urinary, gastrointestinal, and sexual function (eg, urinary frequency, constipation, deep pelvic pain with intercourse).

On the physical exam the question might describe **significant uterine enlargement as** (eg, mass extending to the umbilicus) and an **irregular uterine contour** as (eg, palpable protuberances).

Treatment for bulk symptoms involves reducing fibroid size and number with either medication (ie, GnRH agonist) or surgery (eg, myomectomy).

Now both fibroids and endometriosis can cause pelvic pain and pain with intercourse.

Unlike fibroids, which cause pelvic pressure due to mass effect, endometriosis can cause dyspareunia and pressure by inducing pelvic inflammation and the formation of pelvic adhesions (eg, fixed, immobile uterus). However, endometriosis does not cause an enlarged or irregularly shaped uterus.

Adenomyosis, or endometrial proliferation within the myometrium, can present with a tender, enlarged uterus and dyspareunia. In contrast to the one seen in fibroids where it can be very larger, 20-week-sized uterus (ie, reaches the umbilicus), patients with adenomyosis typically have a uniformly globular uterus ≤12 weeks in size. In addition, because of the relatively smaller uterine size compared to that of fibroids, patients with adenomyosis typically do not have constant pelvic pressure.

- It can develop in a number of anatomic locations:

A. Intramural:

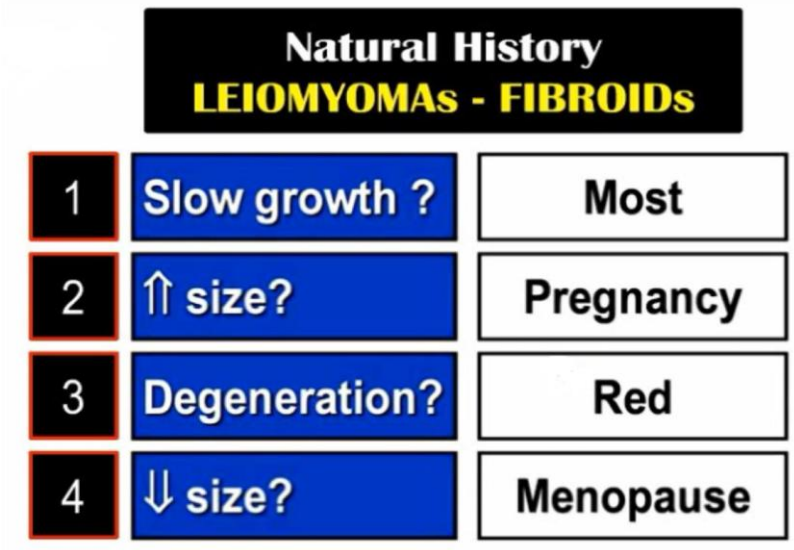
- The most common location of a leiomyoma is within the wall of the uterus.
- When small it is usually asymptomatic and cannot be felt on examination unless it enlarges to where the normal uterine external contour is altered.

B. Submucosal:

- These myomas are located beneath the endometrium and can distort the uterine cavity.
- The distorted overlying endometrium may not respond appropriately to the normal hormonal fluctuations, resulting in unpredictable, often intermenstrual, bleeding.
- Abnormal vaginal bleeding is the most common symptom of a submucosal myoma and can result in anemia.
- Most fibroids do not affect pregnancy. However, having large or multiple fibroids can increase the risk of obstetrical complications (miscarriage, malrepresentation, abruptio placentae, preterm birth).

C. Subserosal:

- These are located beneath the uterine serosa.
- As they grow, they distort the external contour of the uterus causing the firm, nontender asymmetry. Leiomyomata can cause an irregularly enlarged uterus and size-date discrepancy during pregnancy.
- Depending on their location they can put pressure on the bladder, rectum, or ureters.
- Subserosal and pedunculated uterine leiomyomata can cause bulk-related symptoms (pelvic pressure, back/pelvic pain, sensation of incomplete voiding, and constipation).



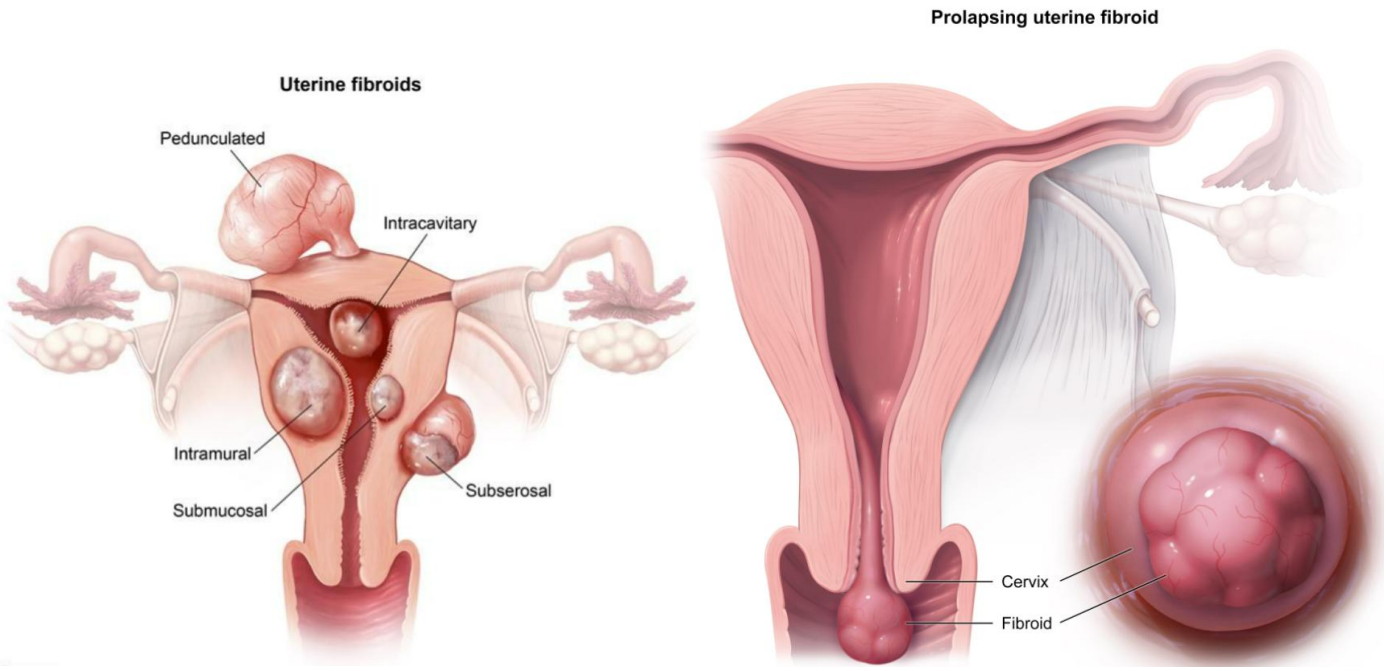
- Slow growth:
  - Most leiomyomas are small, grow slowly, and cause no symptoms.
  - Only when massive in size do they cause pelvic pressure symptoms.
- Rapid growth:
  - Estrogen receptors are increased in leiomyomas resulting in rapid enlargement during times of high estrogen levels, such as pregnancy.
- Degeneration:
  - During times of rapid growth, myomas may outgrow their blood supply, resulting in ischemic degeneration of a fibroid.
- Leiomyomata uteri are more likely to degenerate during pregnancy because myometrial blood flow shifts toward the developing fetus and placenta. An infarcted, degenerating uterine fibroid can cause severe abdominal pain; uterine tenderness; a palpable, firm, and tender mass: and signs of inflammation (leukocytosis).

This process is known as red degeneration.

- Shrinkage:
  - When estrogen levels fall, with estrogen receptors no longer stimulated, leiomyomas will typically decrease in size.
  - This predictably occurs after menopause but can also occur when estrogen levels are medically reduced through gonadotropin releasing hormone (GnRH) agonist suppression of follicle-stimulating hormone (FSH).

Although it is unclear why fibroids develop in some patients but not others, fibroid proliferation appears to be influenced by environmental and reproductive factors (eg, pregnancy, obesity) that increase estrogen levels. Further evidence that fibroids **respond to estrogen** include the increased expression of estrogen receptors in fibroid tissue and the natural history of the disease: Fibroids typically develop after puberty and proliferate the most during the reproductive years (eg, 20s to late 40s), when ovarian estrogen production is at its highest.

In contrast, patients nearing **menopause** (mean age: 51), have a marked **decrease in estrogen levels** due to decreasing ovarian function. Therefore, menopausal patients typically have **spontaneous fibroid regression** with improvement in symptoms.

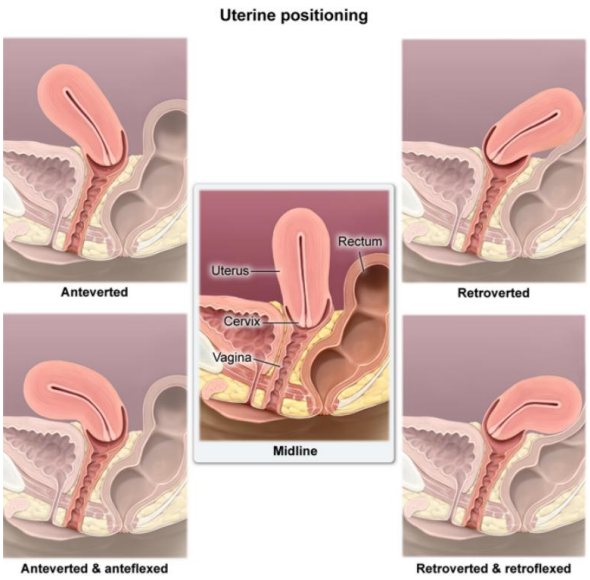


Submucosal fibroids can protrude into the endometrial cavity, which causes both endometrial distortion and increased surface area that lead to heavy, prolonged menses. This patient with both bleeding and bulk symptoms likely has fibroids in multiple locations.

During a heavy menstrual cycle, prolonged endometrial shedding and uterine cramping can increase intrauterine pressure and cause **intracavitary fibroids**, which protrude completely into the uterine cavity, to prolapse through the cervix. **Prolapsed leiomyoma uteri** are most commonly the intracavitary type because these fibroids have smaller attachments to the myometrium (typically a small pedicle) and greater mobility. As the prolapsed fibroid passes through the cervical canal, patients typically develop pelvic cramping followed by **labor-like pain** from **cervical dilation**. Examination typically reveals a dilated cervix and **firm, round mass** visible through the external os. The diagnosis of a prolapsed fibroid is clinical, although ultrasonography can be performed for confirmation. Treatment is with surgical removal of the prolapsed fibroid.

Just know that a patient with fibroids can have one of them prolapse through the cervical os.

This will present as sever pain almost like labor pain or worse on speculum examination there will be a round mass protruding from the cervical os.

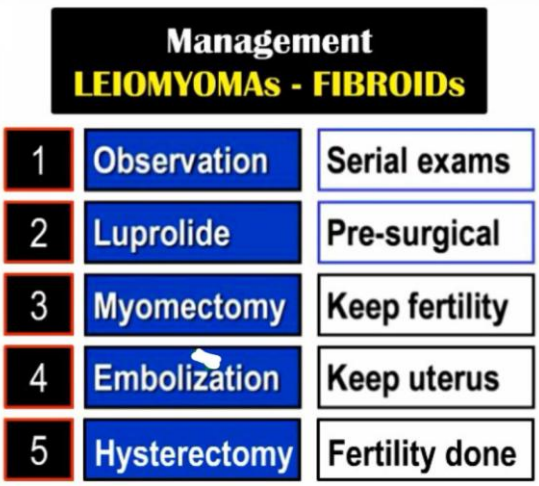


Large fibroids can have a direct mass effect on adjacent pelvic organs and alter the position of the uterus. Because the bladder is anterior to the uterus, an anterior uterine fibroid or fibroids causing uterine anteversion and anteflexion, can **compress the bladder neck and/or urethra**, increasing the urethral closing pressure and leading to urinary retention and overflow incontinence. Diagnosis is with pelvic ultrasound, and definitive treatment of bladder outlet obstruction in these patients is with fibroid reduction (eg, GnRH agonist, surgery).

A highly sensitive test for the evaluation of **submucosal fibroids** is **sonohysterography**, a form of transvaginal ultrasonography that infuses saline into the uterine cavity for improved detection of intrauterine pathology (eg, fibroid, polyp).

Uterine Sarcoma is Indistinguishable from **fibroid** but it should be suspected in **Postmenopausal women**. *(Usually solitary mass with Necrosis and Haemorrhage)*

- Management:



- Observation:
  - Most leiomyomas can be managed conservatively and followed expectantly with regular pelvic examinations.

- Presurgical shrinkage:
  - After 3-6 months of GnRH analog therapy, with resultant hypoestrogenic state, a 60-70% reduction in size of the fibroids can be expected. However, once the leuprolide (Lupron) is terminated, there will be a regrowth of the fibroid within 6 months. Thus, GnRH analogs cannot be used for definitive cure, but they can be used in the adjuvant setting with surgical therapy.

  - If a myomectomy is done, a decrease in size will be associated with a decrease in blood loss, and if a hysterectomy is planned, then perhaps a vaginal instead of an abdominal hysterectomy can be performed.

- Hysteroscopic Myomectomy:
  - This is a surgical procedure performed if the patient desires to maintain fertility.

  - If the myomectomy incision entered the endometrial cavity, delivery of any subsequent pregnancy should be by cesarean section because of increased risk of scar rupture in labor.

- Embolization:
  - This is an invasive radiology procedure in which a catheter is placed into the vessels supplying the myoma.

  - Microspheres are injected, causing ischemia and necrosis of the myoma.

- Hysterectomy:
  - If the patient has completed her childbearing, definitive therapy is an abdominal or vaginal hysterectomy.

• Patients with **symptomatic submucosal fibroids** who desire **future fertility** are treated with **hysteroscopic myomectomy**

• What *type(s) of fibroids* are most associated with **heavy menstrual bleeding** and/or **recurrent pregnancy loss**?

Submucosal and Intracavitary fibroids (leiomyomata)

• What *type(s) of fibroids* are most associated with bulk-related symptoms and ***irregular*** **uterine enlargement**?

Subserosal and Pedunculated fibroids (leiomyomata)

e.g. constipation, incomplete voiding, pelvic pressure

• What medical therapy may be used for *short-term* treatment of symptomatic **fibroids** refractory to OCPs and NSAIDs?

GnRH agonists (e.g. leuprolide)

not recommended *for more than* ***3 - 6 months****;* ***typically used before a hysterectomy*** *or to* ***bridge*** *a woman close to menopause*

Adenomyosis:

	Adenomyosis
Pathogenesis	Abnormal endometrial tissue within the uterine myometrium
Risk factors	Age >40 Multiparity Prior uterine surgery (eg, myomectomy)
Clinical features	Dysmenorrhea Heavy menstrual bleeding Chronic pelvic pain Diffuse uterine enlargement (eg, globular uterus) ± Uterine tenderness
Diagnosis	Clinical presentation MRI & ultrasound: Thickened myometrium Confirmation via pathology
Treatment	Hysterectomy

adenomyosis – a disorder caused by an abnormal collection of endometrial glands and stroma within the uterine myometrium. Adenomyosis typically presents in multiparous women age >40 with prior uterine surgery (eg, cesarean delivery). Clinical features of adenomyosis are as follows:

- New-onset **dysmenorrhea** due to cyclic accumulation of endometrial glands and stroma within the myometrium during menses.
- Continued endometrial gland accumulation causes a **symmetrically enlarged (globular) uterus** that is boggy and tender but does not exceed 12 weeks in size.
- The symmetrically enlarged uterus increases the endometrial cavity surface area, resulting in the concomitant **heavy menstrual bleeding** (eg, anemia) typically seen in these patients.

new-onset dysmenorrhea in later reproductive years is classic

As repeated menstrual cycles continue to cause endometrial shedding within the myometrium, patients often progress from dysmenorrhea to **chronic, dull pelvic pain**.

The entrapped endometrial tissue within the uterine myometrium results in a boggy, tender uterus on examination; it also induces myometrium hypertrophy, which causes a concentric or symmetrically enlarged uterus.

- Diagnosis:

- In most cases the diagnosis is made clinically by identifying an enlarged, symmetric, tender uterus in the absence of pregnancy. Tenderness is most common immediately before and during menses.

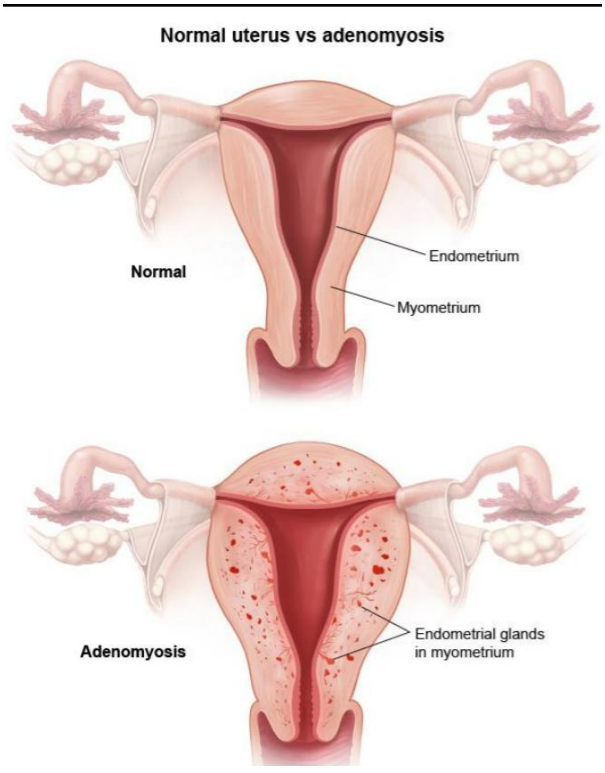
- The disruption of the arrangement of the smooth muscle fibers interferes with normal uterine contraction and causes dysmenorrhea.

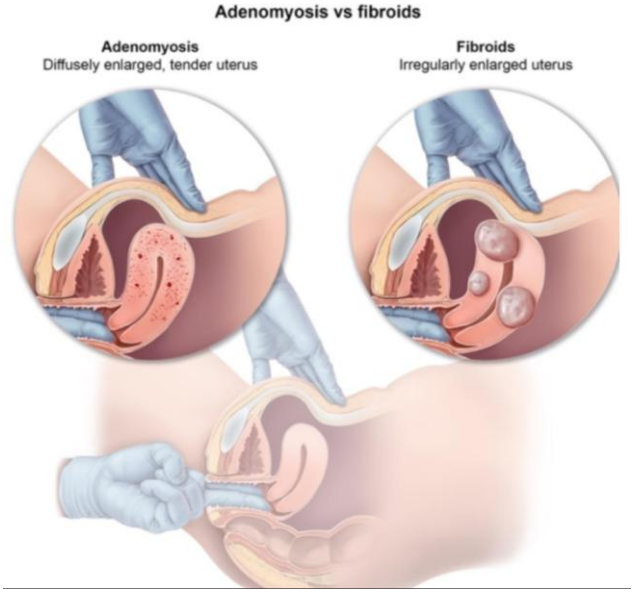
- The continued accumulation of endometrial tissue within the myometrium causes an increase in the endometrial cavity surface area resulting in heavy menstrual bleeding.

Leiomyoma	Adenomyosis
Asymmetric	Symmetric
Firm	Soft
Nontender	Tender

The initial workup of suspected adenomyosis consists of pelvic ultrasonography and/or MRI.

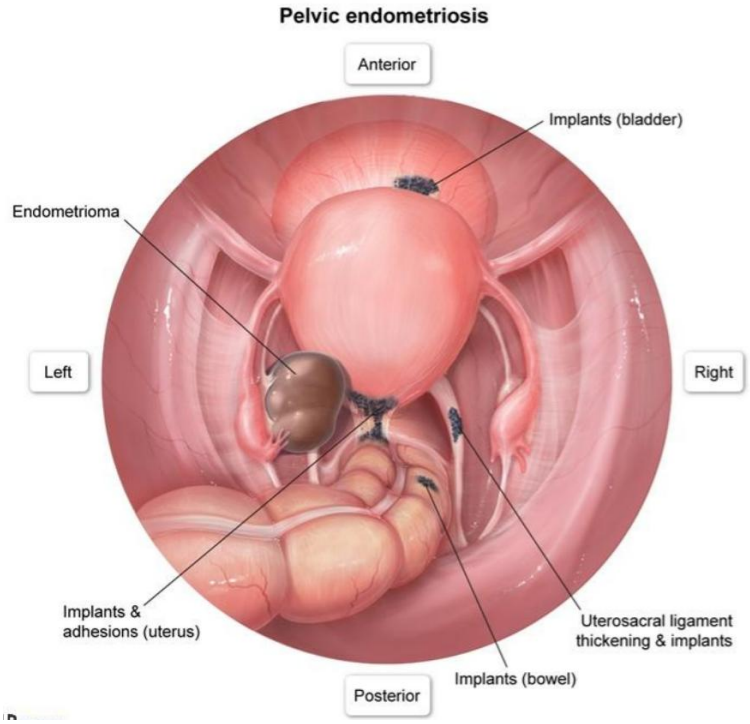
Definitive diagnosis of adenomyosis is made histologically after hysterectomy, which is also the treatment for patients who do not improve with conservative management (eg, oral contraceptives).





Endometriosis:

	Endometriosis
Pathogenesis	Ectopic implantation of endometrial glands
Clinical features	Dyspareunia Dysmenorrhea Chronic pelvic pain Infertility Dyschezia Cyclic dysuria, hematuria
Physical examination	Immobile uterus Cervical motion tenderness Adnexal mass Rectovaginal septum, posterior cul-de-sac, uterosacral ligament nodules
Diagnosis	Direct visualization & surgical biopsy
Treatment	Medical (oral contraceptives, NSAIDs) Surgical resection



ectopic endometrial implants within the intraabdominal cavity. These ectopic glands and stroma proliferate and shed within the abdominal cavity during menses; however, because there is no outlet, patients develop intraabdominal inflammation and fibrosis, causing **dysmenorrhea** (ie, increase in cramping during menses).

Depending on the location of the lesions, patients may also experience **dyspareunia** (ie, pain with intercourse) due to proximity of the vagina to the internal pelvic structures (eg, uterus, bladder, rectum). Therefore, on examination, patients may have **cervical motion tenderness**. With continued inflammation, patients can also develop intraabdominal fibrosis and adhesions causing pelvic anatomy distortion, as seen in this patient's lateral **cervical displacement**.

The "three D's" of endometriosis are **dysmenorrhea**, **deep dyspareunia**, and **dyschezia.**

Also they can present with posterior fornix tenderness, **decreased uterine mobility**, and **uterosacral ligament thickening**. Transvaginal ultrasound is often normal (except in those with an endometrioma) because lesions are too small to detect on imaging.

Whenever U see pain put endometriosis on the DDx:

Dysmenorrhea/ dyspareunia/ dyschezia/ abdominal pain/ dysuria.

Pelvic inflammatory disease can cause dyspareunia and cervical motion tenderness; however, it is associated with fever and/or concomitant cervical inflammation (ie, cervicitis).

- Infertility is commonly the sole presenting symptom of endometriosis, which is present in one quarter of all patients with infertility. Cyclic accumulation of ectopic foci of hemorrhage and adhesions can distort pelvic anatomy and impair fertility by obstructing oocyte release or sperm entry. The presence of an endometrioma (ovarian endometriosis cyst) is also associated with impaired ovarian function.

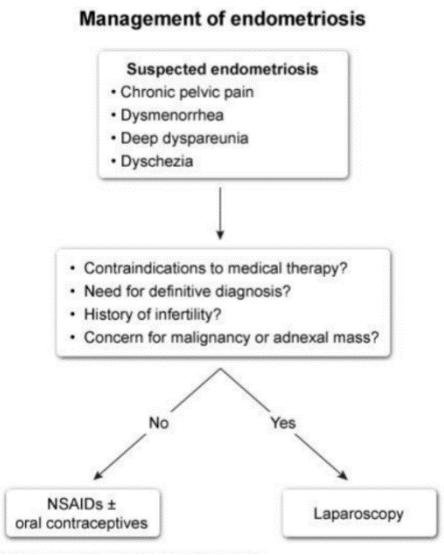
B. Examination:

- Physical examination findings vary but commonly include a fixed, retroverted and immobile uterus and rectovaginal nodularity often caused by cul-de-sac adhesions.

- Adnexal mass or fullness should be confirmed by ultrasonography, and the finding of a homogeneous cystic ovarian mass is highly suggestive of an ovarian endometrioma. An endometrioma can be the only clinical manifestation of endometriosis.

common complication of endometriosis is **infertility**. In these patients, **inflammation** interferes with fertilization by decreasing sperm motility; in addition, **adhesion formation** and pelvic anatomy distortion can cause tubal obstruction and impaired implantation.

Initial treatment of endometriosis is with nonsteroidal anti-inflammatory drugs (which decrease inflammation) and combined oral contraceptive pills (which suppress ovulation and reduce menstruation). However, in patients who **fail medical management**, a diagnostic **laparoscopy** is recommended because it allows for **definitive diagnosis** (via direct visualization and biopsy of lesions) and is **therapeutic** (via removal of endometriotic lesions)



• What is the *recommended management* for an asymptomatic patient with incidentally discovered **endometriosis** during an unrelated surgery?

Observation

*intraoperative findings may include **adhesions**, **powder-burn lesions**, and "**chocolate cysts**"; asymptomatic patients do not require any treatment*

• What is the definitive treatment for **endometriosis**?

Hysterectomy and oophorectomy

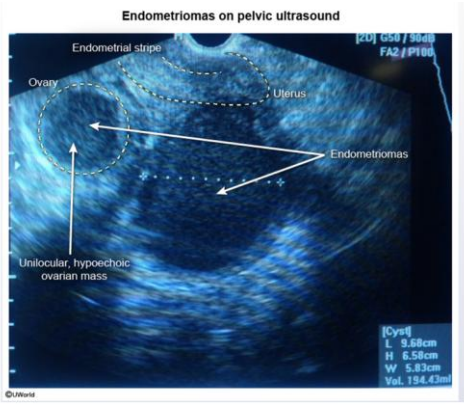
typically done in symptomatic women who have completed childbearing

---

• What is the likely *diagnosis* in a young woman that presents with **chronic pelvic pain**, especially with **exercise**, and a **homogenous cystic ovarian mass** on ultrasound?

Ovarian endometrioma *(secondary to endometriosis)*

Patients with **uterine** or **pelvic sidewall implants** may have worsening pain with exercise or intercourse (ie, **dyspareunia**), as well as adhesion formation that can cause tubal **infertility**.



homogenous, hypoechoic ovarian** cyst with a **ground-glass appearance.

Causes of heavy menstrual bleeding	
Diagnosis	**Clinical features**
Adenomyosis	Heavy, regular menses Dysmenorrhea, pelvic pain Uniformly enlarged (globular), tender uterus
Endometrial cancer/hyperplasia	Irregular, intermenstrual, heavy, or postmenopausal bleeding History of unopposed estrogen (eg, obesity, nulliparity, chronic anovulation) Nontender uterus (± enlarged)
Endometriosis	Uncommon cause of heavy menses Dysmenorrhea, pelvic pain, dyspareunia Fixed uterus, adnexal mass (endometrioma), rectovaginal nodularity
Uterine leiomyomas (fibroids)	Heavy, regular menses Bulk symptoms (eg, pelvic pressure/pain, constipation) Irregularly enlarged uterus with uneven contour
Coagulopathy (eg, von Willebrand disease)	Heavy, regular menses Bruising, mucocutaneous bleeding (eg, gums) Normal uterus

	Intrauterine adhesions
Risk factors	Infection (eg, septic abortion, endometritis) Intrauterine surgery (eg, curettage, myomectomy)
Clinical features	Abnormal uterine bleeding Amenorrhea Infertility Cyclic pelvic pain Recurrent pregnancy loss
Evaluation	Hysteroscopy

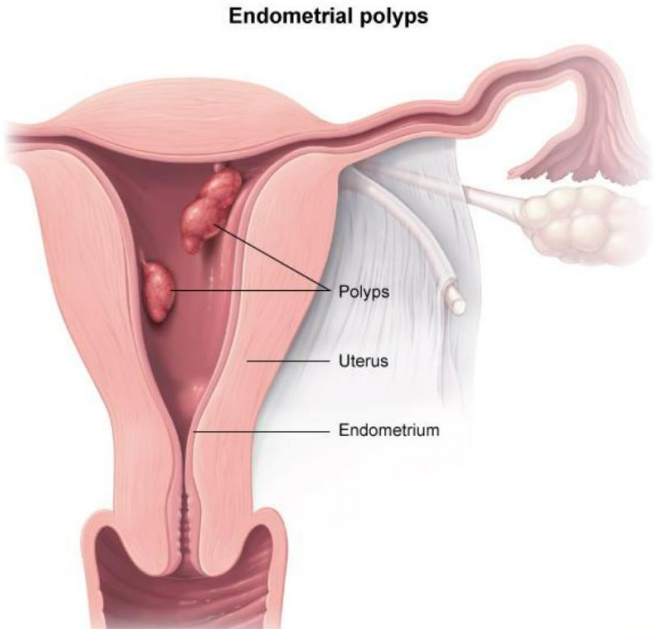
Asherman syndrome, the formation of **intrauterine adhesions** that can follow intrauterine surgery. It occurs most often after **suction and sharp curettage** for delivery complications (eg, postpartum hemorrhage, endometritis). Because the postpartum uterus is soft and enlarged, curettage might be unintentionally aggressive. The basalis layer of the endometrium can be injured inadvertently, creating a denuded, adherent intrauterine surface prone to synechiae and endometrial cavity obliteration.

Because of complete or partial **obliteration of the endometrium**, patients typically have very light menses or **secondary amenorrhea** that does not respond to a progesterone challenge, as evidenced by **no withdrawal bleeding** during the placebo week of oral contraceptive pills. Asherman syndrome is a structural cause of amenorrhea; therefore, patients have normal FSH and TSH levels. Diagnosis and treatment are performed via hysteroscopy to identify and lyse the adhesions.

What Is the Placebo Week?

It is the **last 4-7 days of a standard 28-day OCP pack**. During this week, the pills contain **no active hormones** (estrogen and progestin). They are typically made of inert ingredients (sugar, cellulose, or iron) or are simply omitted ("ED" packs have 21 active pills and 7 reminder tablets).

So when the patient has no bleeding in the placebo week this means that the estrogen withdrawal didn’t cause bleeding.



Endometrial polyps are common, well-vascularized, hyperplastic endometrial gland growths that extend into the uterine cavity. Endometrial polyps typically develop in women in their 30s and 40s.

Most endometrial polyps are benign and asymptomatic, but they can cause **abnormal uterine bleeding (AUB)** in some patients due to their friability and vascularity. Because endometrial polyps do not affect ovulation, patients typically have **regular monthly menses** with painless, light **intermenstrual bleeding**. Endometrial polyps typically remain intracavitary; therefore, patients typically have a small, nontender uterus with no visible cervical or vaginal lesions.

Treatment of symptomatic endometrial polyps is with hysteroscopic polypectomy.

Dysmenorrhea:

or painful menses, is common in reproductive-aged women and is divided into primary (physiologic) and secondary (pathologic) causes. Primary dysmenorrhea is the most common cause of painful menses and typically presents in adolescents after ovulatory, regular menstrual cycles are established. Patients typically have midline pelvic pain that can radiate to the bilateral legs or back as well as associated systemic symptoms such as fatigue, nausea, vomiting, and diarrhea. Primary dysmenorrhea can be diagnosed based on history alone and does not require physical examination.

In contrast, patients with any of the following clinical features should be evaluated for **secondary causes of dysmenorrhea**:

- Symptom onset at age >25
- **Unilateral (nonmidline) pelvic pain**.
- No systemic symptoms (eg, fatigue, nausea) during menses
- Abnormal uterine bleeding (eg, intermenstrual bleeding, postcoital spotting)

These features are suggestive of a variety of **pathologic causes** of dysmenorrhea, including pelvic inflammatory disease, endometriosis, adnexal masses, and a uterine anomaly (eg, rudimentary uterine horn). Physical examination is performed to evaluate for these possible causes; patients with abnormal examination findings (eg, adnexal mass, cervical motion tenderness) may require additional imaging (eg, pelvic ultrasound) and possible surgery (eg, laparoscopy).

Primary dysmenorrhea	
Etiology	Excessive prostaglandin production
Risk factors	Age <30 BMI <20 kg/m^2 Tobacco use Menarche at age <12 Heavy/long menstrual periods Sexual abuse
Clinical features	Pain first 2-3 days of menses Nausea, vomiting, diarrhea Normal pelvic examination
Management	Nonsteroidal anti-inflammatory drugs Combination oral contraceptive pills

Primary (ie, physiologic) dysmenorrhea (painful menses) is common, particularly among adolescents. **Excessive prostaglandin production** during menses can stimulate uterine contractions and result in **lower abdominal pain** that can radiate to the back and thighs. Some patients may also develop gastrointestinal symptoms (eg, **nausea**, vomiting, bloating, diarrhea) from prostaglandin-induced gastrointestinal stimulation. Symptoms are typically worse during the first few days of menses and can interfere with daily activities. Patients have a **normal pelvic examination** because the pelvic pain occurs without an identifiable pathologic cause.

The first-line treatment for primary dysmenorrhea is **nonsteroidal antiinflammatory drugs** (NSAIDs), which reduce prostaglandin synthesis. For patients who are sexually active or in whom NSAIDs are ineffective or cannot be tolerated, **combination oral contraceptives** (COCs) can be used.

In sexually active patients, combination oral contraceptives are an option because they suppress ovulation, providing contraception as well as decreasing prostaglandin release.

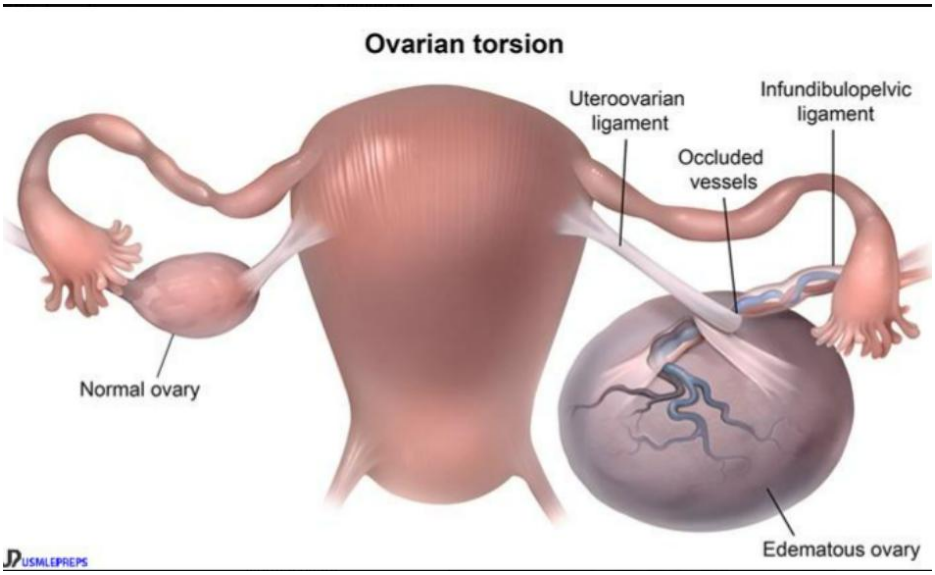
Acute abdominal/pelvic pain in women		
Diagnosis	**Clinical presentation**	**Ultrasound findings**
Mittelschmerz	Recurrent mild & unilateral mid-cycle pain prior to ovulation Pain lasts hours to days	Not indicated
Ectopic pregnancy	Amenorrhea, abdominal/pelvic pain & vaginal bleeding Positive β-hCG	No intrauterine pregnancy
Ovarian torsion	Sudden-onset, severe, unilateral lower abdominal pain; nausea & vomiting Unilateral, tender adnexal mass on examination	Enlarged ovary with decreased or absent blood flow
Ruptured ovarian cyst	Sudden-onset, severe, unilateral lower abdominal pain immediately following strenuous or sexual activity	Pelvic free fluid
Pelvic inflammatory disease	Fever/chills, vaginal discharge, lower abdominal pain & cervical motion tenderness	± Tuboovarian abscess

In **reproductive-aged women**, sudden abdominal pain can have a variety of etiologies (eg, gastrointestinal, gynecologic) due to the proximity of anatomic structures. Initial evaluation aims to differentiate between emergent (ie, acute abdomen) and nonemergent conditions, and to identify the anatomy involved (eg, appendix, adnexa). Pregnancy testing is required, and imaging (eg, ultrasonography) may be performed

Ovarian cysts are common in reproductive-aged women due to physiologic cyst formation from ovulation.

physiologic ovarian cysts are usually small (eg, <3 cm) and asymptomatic, but they can become larger and rupture with vigorous activity (eg, intercourse). Patients typically have **acute-onset, unilateral pain** due to cyst rupture and spilling of fluid into the peritoneal cavity. Diagnosis is confirmed by ultrasound, which typically reveals a **simple, thin-walled cyst** with **free fluid** in the pelvis.

Hemodynamically stable patients with no signs of infection (eg, fever) are managed with **observation and reassurance**. In contrast, hemodynamically unstable patients may have continued bleeding from the ruptured cyst and require surgery.



sudden-onset unilateral pelvic pain accompanied by nausea/vomiting is most likely due to **ovarian torsion**, which represents partial or complete rotation of the ovary around the infundibulopelvic (IP) ligament. The IP ligament contains the ovarian vessels; rotation acutely interrupts ovarian blood flow, causing acute-onset pain. As ovarian ischemia progresses, patients may develop radiating pain, ovarian edema (eg, **adnexal tenderness** or fullness, with or without a palpable mass), and peritonitis (eg, rebound/guarding, fever).

Ovarian torsion is a **gynecologic emergency** that is **clinically diagnosed** in patients with classic symptoms. Although a Doppler ultrasound revealing decreased or absent ovarian blood flow can support the diagnosis, normal findings do not exclude torsion. Because of the risk of tissue necrosis and loss of ovarian function (eg, infertility, menopause), ovarian torsion requires urgent **diagnostic laparoscopy** to manually untwist the adnexa and restore blood flow.

Partial and complete torsions:

- **Partial ovarian torsion**, due to intermittent adnexal rotation around the infundibulopelvic ligament containing the ovarian vessels, causes temporary ovarian vessel occlusion and pelvic pain (from ovarian ischemia). Patients classically have nausea, vomiting, and intermittent **pain that self-resolves** (ie, no symptoms between episodes) as spontaneous adnexal untwisting allows blood flow to return. Therefore, Doppler ultrasound may show normal ovarian arterial and venous blood flow.
- Partial ovarian torsion can progress to **complete ovarian torsion**. Although torsion is less common in the pediatric population, an **ovarian mass** increases the risk for complete torsion because the increased adnexal size and density make spontaneous untwisting less likely. Complete torsion, typically triggered by physical activity (eg, walking), presents with **severe, constant, unilateral pelvic pain** due to ongoing ovarian ischemia.

Complete torsion is a **clinical diagnosis** in patients with the classic symptoms. Management is with **diagnostic laparoscopy** for manual detorsion of the adnexa and removal of any contributory cysts or masses; oophorectomy may be required if the ovary is necrotic.

	Pelvic organ prolapse
Definitions	Anterior prolapse: Bladder (eg, cystocele) Posterior prolapse: Rectum (eg, rectocele) Enterocele: Small intestine Apical prolapse: Uterus, vaginal vault Procidentia: Complete herniation
Risk factors	Obesity Multiparity Hysterectomy Menopause
Clinical presentation	Pelvic pressure Obstructed voiding Urinary retention Urinary urgency/incontinence Constipation Fecal urgency/incontinence Sexual dysfunction
Management	Weight loss Pelvic floor muscle training Pessary Surgical repair

pelvic organ prolapse (POP). POP is the abnormal herniation of the uterus, bladder, or rectum through the vagina wall due to **weakening of the pelvic floor muscles** (eg, levator ani muscle complex), ligaments, and nerves caused by chronic, **increased intraabdominal pressure** and pelvic floor injury.

The most common risk factor for POP is **multiparity** because pregnancy causes increased intraabdominal pressure with subsequent pelvic floor weakening and laxity; vaginal deliveries, even if uncomplicated as in this patient, can cause additional microinjury to the pelvic floor muscles and nerves. With increasing age, the herniation can progress, resulting in the development of vaginal pressure or a **vaginal mass** that worsens with Valsalva maneuver (eg, voiding). Some patients (such as this one) can also develop **vaginal erosions** as

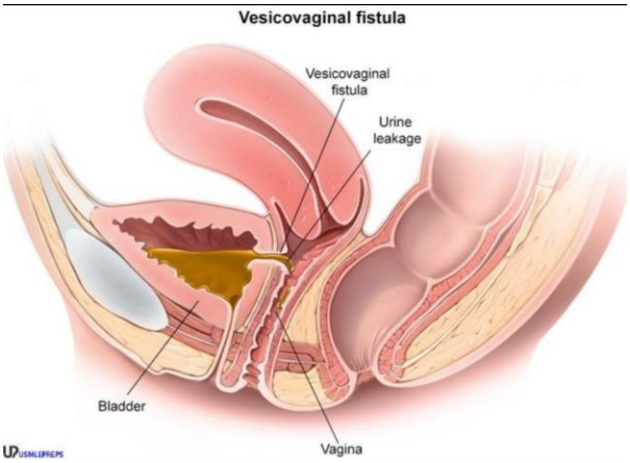
the prolapsed vaginal wall protrudes past the hymenal ring, rubs against clothing, and becomes denuded and friable (**abnormal vaginal bleeding**).

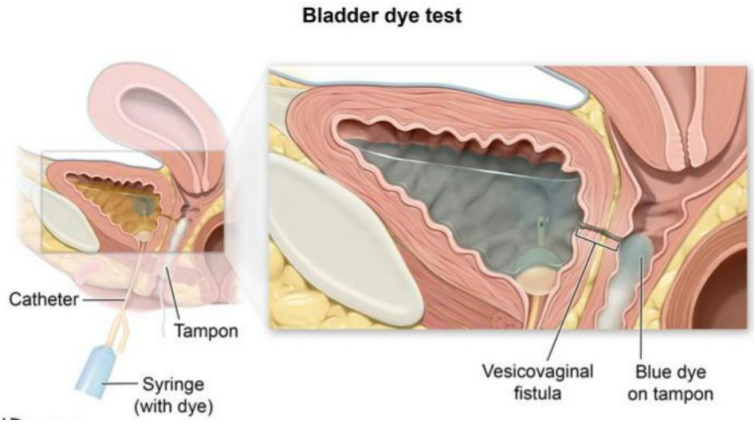
Treatment of POP includes pelvic floor muscle exercises in combination with either medical (eg, pessary placement) or surgical management.

Women with anterior vaginal wall prolapse (ie, cystocele), such as this patient, can have pelvic pressure and urinary symptoms (eg, retention, stress urinary incontinence). However, many patients with POP are asymptomatic and **incidentally diagnosed** on routine examination.

Management of POP is based on symptoms or complications:

- **Asymptomatic** patients, such as this one with no complications (eg, no urinary retention or hydronephrosis on ultrasound), do not require treatment and can be managed with **reassurance and observation only**.
- In contrast, symptomatic patients (eg, pelvic pressure) or those with complications could benefit from treatment. Pelvic floor muscle (ie, Kegel) exercises are recommended in these patients. Nonsurgical treatment is with pessary placement, which helps restore pelvic anatomy and reduces the severity of symptoms. Surgical management (eg, anterior vaginal wall repair) can be offered to patients whose condition does not improve or those who decline nonsurgical treatment.





vesicovaginal fistula (VVF), a complication of **obstructed labor** that is common in resource-limited areas (eg, sub-Saharan Africa) due to young maternal age (ie, small pelvis) and limited or no prenatal care, which results in delayed diagnosis and labor intervention. Obstructed labor is the most common cause of VVF worldwide with an estimated >100,000 new cases yearly.

In these patients, VVF develops because excessive fetal head compression during obstructed labor causes injury and necrosis to the maternal vagina, rectum, and bladder. Tissue necrosis leads to erosion and fistula development between proximal structures (eg, vesicovaginal, rectovaginal), typically occurring within the first weeks postpartum. Because of the aberrant connection between the vagina and bladder, patients with VVF have a **continuous vaginal discharge** with an abnormally elevated pH (ie, >4.5) due to urine, which may be malodorous due to surrounding necrotic tissue. Pelvic examination typically shows vaginal pooling of urine, a visible defect, or an area of **raised, red granulation tissue** on the anterior vaginal wall.

Bladder dye testing is performed to confirm the diagnosis, particularly in patients who have small fistulas that are not visualized on pelvic examination. During a bladder dye test, the bladder is filled with dyed fluid and patients are monitored for vaginal leakage of dye via speculum examination or tampon placement. Treatment of VVF is with surgical repair.

Urinary incontinence		
Type	**Symptoms**	**Treatment**
Stress	Leaking with Valsalva maneuver (coughing, sneezing, laughing)	Lifestyle modification Pelvic floor exercises Pessary Pelvic floor surgery
Urgency	Sudden, overwhelming, or frequent need to void	Lifestyle modification Bladder training Antimuscarinic drugs
Mixed	Features of stress & urgency incontinence	Variable treatment depending on predominant symptoms
Overflow	Constant involuntary dribbling & incomplete emptying	Identification and correction of underlying cause Cholinergic agonists Intermittent self-catheterization

stress urinary incontinence(SUI), a common cause of incontinence in women. The bladder and urethra are normally maintained in the appropriate anatomic position by the pelvic floor (levator ani) muscles. However, with chronic pressure or injury to the pelvic floor muscles—commonly due to childbirth (ie, multiparity), obesity, or chronic high-impact exercise such as jogging—women can develop **pelvic floor muscle weakness**.

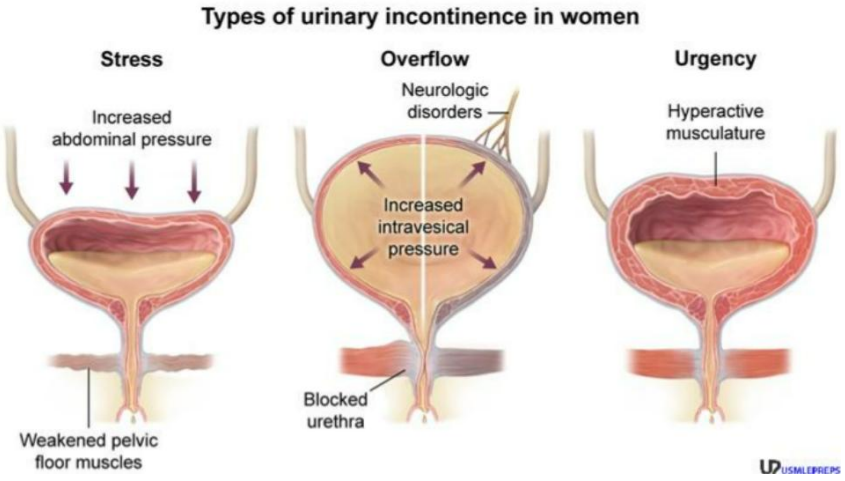
Substantial weakness of the pelvic floor muscles can result in **urethral hypermobility**, in which the urethra abnormally moves with **increased intraabdominal pressure** (eg, jogging, coughing) and is unable to fully close. In addition, inadequate bladder support can develop (ie, prolapse), as evidenced by the anterior vaginal bulge (cystocele). Due to both the urethra's inability to fully close and increased bladder pressure, patients have intermittent leakage of urine when intraabdominal pressure is increased (ie, Valsalva).

Postmenopausal women are at increased risk for SUI due to:

- **Weakened pelvic floor musculature** (ie, levator ani) from chronic intraabdominal and pelvic strain; common etiologies include increasing parity (even in those who deliver by cesarean) and obesity
- **Urogenital mucosa atrophy** from decreased estrogen levels

The pelvic floor musculature and urogenital mucosa normally work together to support the bladder and urethra; postmenopausal women have decreased function in both, resulting in an unsupported bladder and **hypermobile urethra**.

Urinalysis and postvoid residual volume are normal (<150 mL in women, <50 mL in men). First-line treatment includes **pelvic floor muscle exercises** (eg, Kegel exercises) and lifestyle modifications (eg, weight loss). Unresponsive cases may require use of a pessary or midurethral sling surgery.



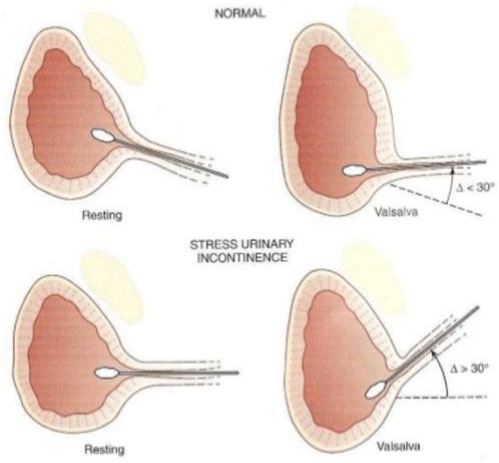
• A **continence pessary** is used to treat **stress urinary incontinence (SUI)** and symptomatic **pelvic organ prolapse** (eg, anterior vaginal wall bulge);

it works by stabilising the pelvic floor in its anatomic position and compressing the urethra against the pubic symphysis.

Midurethral sling procedures are performed for SUI due to **urethral hyper-mobility** or when pessary fails.

• **Stress urinary incontinence** is often associated with **urethral hyper-mobility.**

*characterized by **urethral angle of > 30 degrees** from horizontal with an increase in abdominal pressure (**Q-tip test**); SUI may also be caused by **decreased urethral sphincter tone***



• **Postpartum SUI** increases the risk of chronic SUI, but most cases are **self-limited** because the pelvic floor muscles and pudendal nerve heal after delivery;

therefore, patients **<6 weeks postpartum** are managed with **observation and reassurance** and encouraged to perform Kegel exercises to strengthen pelvic floor muscles.

Vaginal estrogen therapy is used in patients with genitourinary syndrome of menopause (eg, vaginal dryness, atrophy) due to estrogen deficiency.
In postmenopausal patients,
localized estrogen can relieve **urinary symptoms** (eg, stress and/or urge incontinence) related to **atrophy**.

Postmenopausal **estrogen deficiency** usually causes **Urge Incontinence** .

Initial evaluation of mixed incontinence includes a **voiding diary**, which tracks fluid intake, urine output, and leaking episodes in order to **classify the predominant type** of urinary incontinence and **determine optimal treatment**.

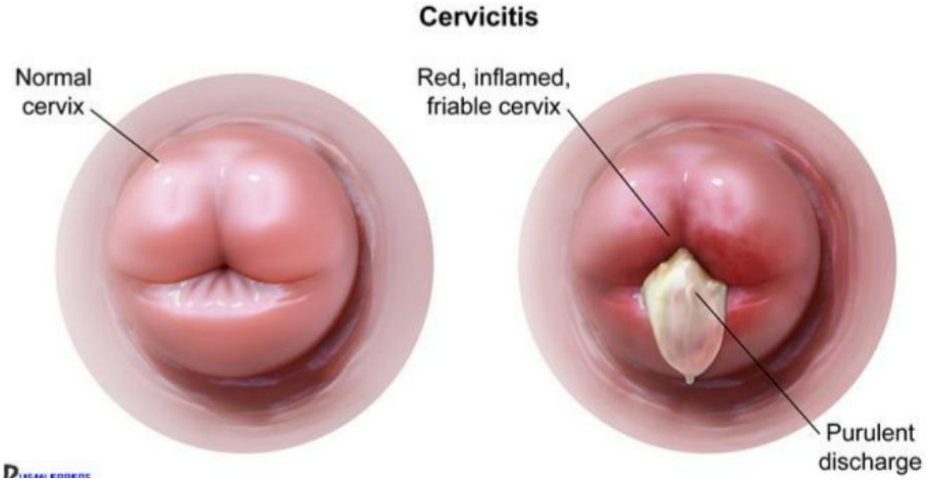
All patients with mixed incontinence generally require **bladder training** with lifestyle changes (eg, weight loss, smoking cessation, decreased alcohol and caffeine intake) and pelvic floor muscle exercises (eg, Kegels). Patients who have limited or incomplete symptom relief with bladder training may benefit from pharmacotherapy or surgery, depending on predominant type:

- In patients with urgency-predominant incontinence, oral antimuscarinics and timed voiding (eg, urinating on a fixed schedule, rather than based on a sense of urgency) are used.
- In patients with stress-predominant incontinence due to weakened pelvic floor muscles (eg, cystocele), surgery with a midurethral sling is performed

Infections:

	Acute cervicitis
Etiology	Infectious: *Chlamydia trachomatis, Neisseria gonorrhoeae* Noninfectious: foreign object, latex, douching
Clinical presentation	Asymptomatic Mucopurulent discharge Postcoital/intermenstrual bleeding Friable cervix
Evaluation	Nucleic acid amplification testing Wet mount microscopy
Management	Empiric treatment: ceftriaxone + doxycycline*
	*Ceftriaxone + azithromycin in pregnancy.

vaginal spotting with associated mucopurulent endocervical discharge (eg, bloody, yellow mucus at the external os) is classic for acute cervicitis, which is cervical inflammation and friability typically caused by a sexually transmitted infection (eg, gonorrhea, chlamydia). Acute cervicitis is diagnosed clinically and requires empiric antibiotic therapy.



Nonpregnant patients are typically treated with ceftriaxone and doxycycline; however, because doxycycline is a potential teratogen, **pregnant patients** are treated with ceftriaxone and azithromycin. Because untreated, undertreated, or recurrent infection can ascend to the uterus and increase the risk of **obstetric complications** (eg, spontaneous abortion, preterm prelabor rupture of

membranes) and **neonatal complications** (eg, conjunctivitis) , pregnant patients also require a **test of cure** after treatment.

Acute cervicitis can present with first trimester bleeding in a pregnant patient and it should be differentiated from other serious causes like:

spontaneous abortion (particularly in patients with a prior spontaneous abortion), but there are many other etiologies of first-trimester bleeding that range from benign (eg, cervical polyp) to life threatening (eg, ectopic pregnancy, septic abortion). Therefore, initial evaluation is with speculum examination to determine the source of bleeding and with pelvic ultrasound to determine pregnancy location.

In acute cervicitis the patient will have a nontender abdomen, closed cervix, and viable intrauterine pregnancy on ultrasound.

	Pelvic inflammatory disease
Symptoms	Lower abdominal pain Abnormal bleeding
Risk factors	Multiple sexual partners Age 15-25 Previous pelvic inflammatory disease Inconsistent barrier contraception use Partner with sexually transmitted infection
Physical examination	Fever >38.3 C (>100.9 F) Cervical motion, uterine, or adnexal tenderness Mucopurulent cervical discharge
Treatment	Inpatient: IV broad-spectrum antibiotics Outpatient: PO broad-spectrum antibiotics
Complications	Tuboovarian abscess Infertility Ectopic pregnancy Perihepatitis

In women, the initial infection of the cervix with *Neisseria gonorrhoeae* or *Chlamydia trachomatis* can be asymptomatic or subtle (eg, **irregular intermenstrual bleeding**). Untreated cervicitis allows polymicrobial vaginal flora to ascend into the upper reproductive tract (eg, uterus, fallopian tubes), causing **fever** and **lower abdominal pain**, as in this patient. As the infection spreads farther into the intraperitoneal cavity, it can cause inflammation of the liver capsule (ie, perihepatitis or Fitz-Hugh–Curtis syndrome in 6%-10% of patients), resulting in **vomiting** and **right upper quadrant tenderness**. The pain is often pleuritic (ie, worsened with inspiration) due to fibrinous adhesion formation on the anterior liver surface.

So the presentation is typically a lower abdominal pain that progresses to be an upper right pain and associated with worsening with inspiration.

Indications for hospitalization for pelvic inflammatory disease
Pregnancy
Failed outpatient treatment
Inability to tolerate oral medications
Noncompliant with therapy
Severe presentation (eg, high fever, vomiting)
Complications (eg, tuboovarian abscess, perihepatitis)

PID is treated with **empiric, broad-spectrum antibiotics**. The decision to treat either with inpatient or outpatient antibiotic therapy depends on several factors, including the following:

- Severity of presentation: Patients with **high fever**, severe pain, **dehydration** (eg, dry mucous membranes, delayed capillary refill), and **inability to tolerate oral antibiotics** (eg, nausea/vomiting) require **inpatient treatment**.

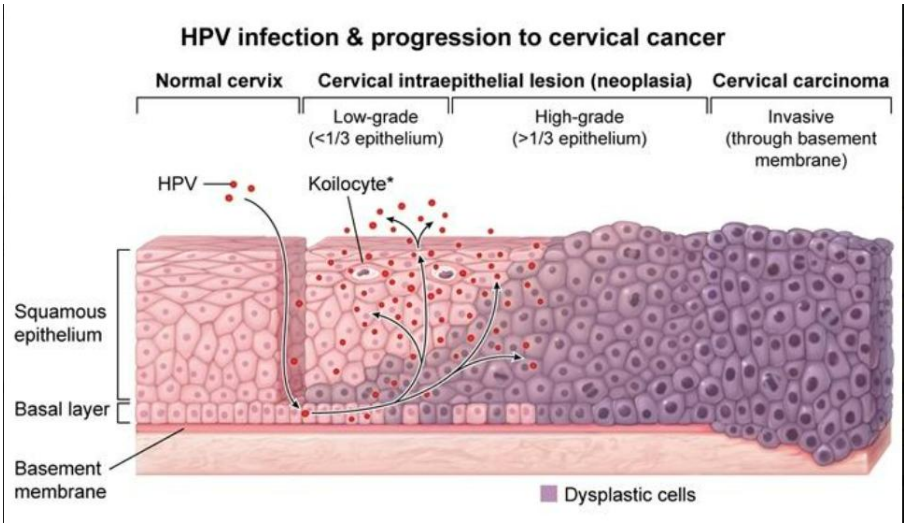
In contrast, patients with a mild presentation (eg, no nausea/vomiting, pain manageable with over-the-counter medication) can be treated as an outpatient

tuboovarian abscess (TOA). TOA typically occurs in sexually active, reproductive-age women as a complication of **pelvic inflammatory disease.**

Patients with TOA can initially have diffuse lower abdominal pain plus **uterine tenderness** that gradually (over days to weeks) worsens and localizes to one side, causing **unilateral adnexal tenderness** with an associated mass. Because of the worsening infection, patients often develop **high fever** and leukocytosis. When the infection extends to the fallopian tube, it creates an inflammatory exudate, purulent fluid, and tubal wall thickening, which conglomerate into a **complex adnexal mass**, or pyosalpinx (ie, infected fallopian tube), visible on **pelvic ultrasound**. Treatment is with intravenous broad-spectrum antibiotics.

	Human papillomavirus
Disease associations	Cervical cancer Vulvar & vaginal cancers Anal cancer Penile cancer Oropharyngeal cancer Anogenital warts Recurrent respiratory papillomatosis
Vaccine indications	All female and male patients* age 11-26 (but may be given to those age 9-45) Not indicated in pregnancy

Human papillomavirus (HPV) is the most common sexually transmitted infection and has been linked to multiple diseases, including condylomata acuminata (ie, anogenital warts) as well as vulvar, vaginal, anal, oropharyngeal, and **cervical cancer**. Persistent HPV infection (particularly with types 16 and 18) results in cellular dysplasia because the incorporation of viral DNA increases prooncogenic protein expression and inhibits normal cellular regulation.



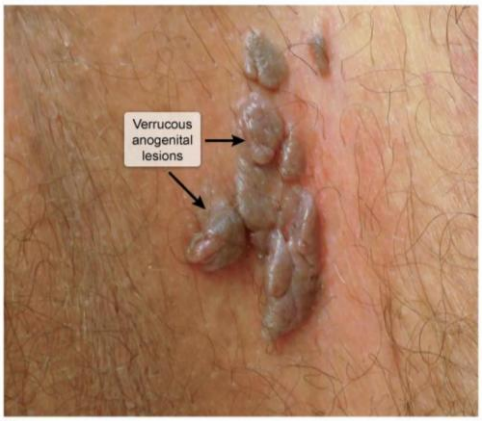
The **HPV vaccination** induces an antibody response that decreases the risk of both HPV infection and subsequent related diseases. Routine administration of the vaccine series is indicated for **female and male patients age 11 to 26**, but vaccination may be given from age 9 to 45. Catch-up vaccination should be offered for patients who are either unvaccinated or did not complete the series

So if a women in this age group present with everything normal the next thing U do is vaccination regardless of her using contraception or not.

Or protective measures.

Genital warts (condylomata acuminata)	
Etiology	HPV 6 & 11
Clinical features	Multiple pink or skin-colored lesions Lesions ranging from smooth, flattened papules to exophytic/cauliflower-like growths
Treatment	Chemical: podophyllin resin, trichloroacetic acid Immunologic: imiquimod Surgical: cryotherapy, laser therapy, excision
Prevention	Vaccination Barrier contraception

Condyloma acuminata



HPV enters the epidermis through **sexual contact** (ie, microabrasions), causing soft, fleshy, verrucous bumps that progress over several months. Once present, anogenital warts may worsen or regress spontaneously (ie, immune clearance). Most patients are **asymptomatic**, but some may develop pruritus or friability (spotting with wiping).

HPV infection, particularly with high-risk types 16 and 18, is also associated with anogenital (eg, cervical, anal) and oropharyngeal cancers. The **most effective preventive strategy** is **HPV vaccination**, ideally prior to initial sexual contact (ie, first HPV exposure). Barrier contraceptive use is also protective. The 9-valent HPV vaccine is available in the United States and is typically administered to males and females age 11-26 but can be given to those age 9-45.

Anogenital warts typically appear as clusters of soft, pink or skin-colored (fleshy) lesions in the internal or external vaginal, vulvar, and anal regions in women. Most lesions are exophytic, dry-appearing, and verrucous (ie, cauliflower-like), although some may appear sessile and flat. Genital warts are typically asymptomatic and nontender, although pruritic, friable lesions (eg, those that bleed with manipulation) may occur.

Small anogenital warts are treated with **topical agents** that can decrease wart size or induce regression by either chemical injury (eg, **trichloroacetic acid**, podophyllin resin) or immunomodulation (eg, imiquimod). Surgical excision may be indicated for bulky, extensive lesions. Recurrence rates are high regardless of treatment modality.

Condylomata lata, caused by secondary syphilis, are raised, gray-white lesions that develop on mucosal surfaces (eg, mouth, perineum). In contrast to condylomata acuminata, condylomata lata typically have a broader base and a smooth, rather than a verrucous, surface.

Anogenital warts (condyloma acuminata) in children	
Etiology	Human papillomavirus infection
Transmission	Sexual abuse Autoinoculation from other sites Prenatal or perinatal
Clinical features	Pink/flesh-colored, verrucous papules & plaques Asymptomatic (most common) Pruritic, friable lesions
Management	Sexual abuse assessment, especially age ≥4

HPV can be transmitted via nonsexual contact such as vertical transmission during delivery, autoinfection from other areas of the body, or heteroinoculation from a caregiver (eg, diaper changes). However, because of the association with transmission via **direct genital contact**, an assessment for **sexual abuse** is required in all children, particularly those age ≥4.

Anogenital warts in children are often self-resolving; therefore, asymptomatic patients typically require only observation. For those with symptomatic or unresolved disease, management options include topical treatments (eg, podophyllotoxin) and surgical removal.

A child that has condyloma should be screened for sexual abuse.

	Chlamydia & gonorrhea in women
Risk factors	Age <25 High-risk sexual behavior
Manifestations	Asymptomatic (most common) Cervicitis Urethritis Perihepatitis (Fitz-Hugh–Curtis syndrome)
Diagnosis	Nucleic acid amplification testing
Treatment	Empiric: ceftriaxone + doxycycline* Confirmed chlamydia: doxycycline* Confirmed gonorrhea: ceftriaxone
Complications	Pelvic inflammatory disease Ectopic pregnancy Infertility Pharyngitis
	*Azithromycin in pregnancy.

Chlamydia trachomatis and *Neisseria gonorrhoeae* are common sexually transmitted infections with high prevalence, particularly in patients age 14-19. Women with these infections are commonly asymptomatic. The lack of symptoms (and subsequent lack of treatment) increases the risk for long-term reproductive complications, including pelvic inflammatory disease, tuboovarian abscess, and infertility or ectopic pregnancy due to tubal scarring. In addition, the frequent absence of symptoms contributes to high rates of transmission. Therefore, **annual screening** for chlamydia and gonorrhea is recommended in **all sexually active women age <25** and women age ≥25 with risk factors (eg, multiple sexual partners, inconsistent condom use).

The **nucleic acid amplification test** (NAAT) is the **gold standard** screening and diagnostic test for both *C trachomatis* and *N gonorrhoeae* because of its high sensitivity (96%) and specificity (99%). Treatment is indicated for a positive NAAT result in the patient and the patient's sexual partners, even in the absence of symptoms. Confirmation testing is not required due to high test specificity.

If a young woman (below 25) comes even with no symptoms and low risk sexual behaviors U screen for chlamydia and gonorrhea

First-line treatment for NAAT-confirmed *Chlamydia trachomatis* is **doxycycline**; azithromycin may be used in pregnant patients for whom doxycycline is a contraindication because of potential teratogenicity.

So, if an asymptomatic person presents with positive NAAT we treat regardless of symptoms

We do not repeat the test or send the patient home with reassurances.

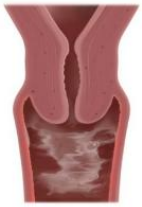
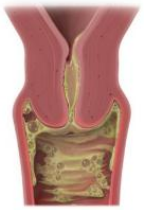
combination of **dysuria** and **sterile pyuria** is a common presentation of ***Chlamydia trachomatis***–associated **urethritis** in women (up to 25% of patients). Therefore, the best next step in management is **nucleic acid amplification testing** for chlamydia; testing for gonorrhea is typically added because co-infection is common. A first-catch urine sample can be used in women; however, because most women have concomitant cervical infection, a vaginal or cervical swab may also be performed.

A negative urine culture makes cystitis unlikely.

- N.B:
  - Patients with Neisseria gonorrhoeae are at high risk of simultaneous coinfection with several other sexually transmitted pathogens, including Chlamydia trachomatis, HIV, Treponema pallidum (syphilis), and hepatitis B virus.
  - Patients with gonococcal infection should be screened for these infections, counseled on safe sexual practices, and encouraged to inform recent partners of infection (with a recommendation to get tested).

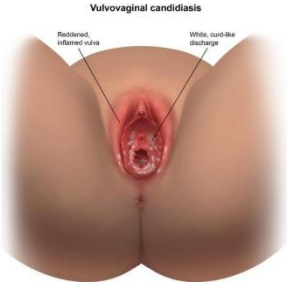
Vaginal infections:

Differential diagnosis of vaginitis

Bacterial vaginosis (*Gardnerella vaginalis*)	Trichomoniasis (*Trichomonas vaginalis*)	Candida vaginitis (*Candida albicans*)
		
• Thin, off-white discharge with fishy odor • No inflammation	• Thin, yellow-green, malodorous, frothy discharge • Vaginal inflammation	• Thick, cottage cheese discharge • Vaginal inflammation
		
• pH >4.5 • Clue cells • Positive whiff test (amine odor with KOH)	• pH >4.5 • Motile trichomonads	• Normal pH (3.8-4.5) • Pseudohyphae
Metronidazole or clindamycin	Metronidazole; treat sexual partner	Fluconazole

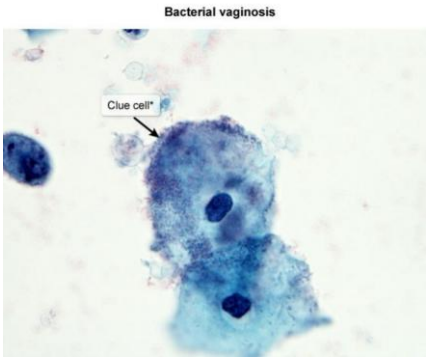
vulvovaginal candidiasis, characterized by **white vaginal discharge** and **vulvovaginal pruritus**, typically due to an overgrowth of *Candida albicans*. Other clinical features include dysuria and vulvar irritation, leading to vulvar and vaginal erythema and excoriations seen on physical examination. Further evaluation of the discharge typically reveals a normal pH; budding yeast and pseudohyphae are present on microscopy.

Risk factors for vulvovaginal candidiasis include increased estrogen levels (eg, pregnancy), immunosuppression, and **diabetes mellitus**. Diabetes mellitus increases the risk of candidiasis as glucose facilitates the adhesion of *Candida* to the epithelial surface. Therefore, women with **recurrent vulvovaginal candidiasis infections** (≥4 episodes in a year) and other signs of diabetes mellitus (eg, **nocturia**, **urinary frequency**) should be evaluated with a **hemoglobin A1c**. Fluconazole is the treatment for vulvovaginal candidiasis, and improved glycemic control can reduce the risk of recurrence.



Bacterial vaginosis (BV) is a common cause of vaginal discharge and occurs due to an **imbalance in vaginal flora**. In patients with BV, the physiologic lactobacilli colonization of the vagina decreases, leading to an increased pH and overgrowth of anaerobic bacteria (eg, *Gardnerella vaginalis*). The increased pH and bacterial overgrowth lead to increasing malodorous vaginal discharge. Risk factors for BV include women having sex with women, douching, and using tobacco.

Patients with BV typically have a **malodorous**, **thin**, **off-white vaginal discharge** with associated **no vulvovaginal inflammation** (eg, pruritus, erythema). Therefore, patients have no vulvar erythema, cervical discharge, or friability. Evaluation of vaginal discharge reveals a **pH >4.5**, an **amine odor** with the addition of potassium hydroxide (whiff test), and clue cells on microscopy (eg, Amsel criteria). Treatment is with **metronidazole** or clindamycin.



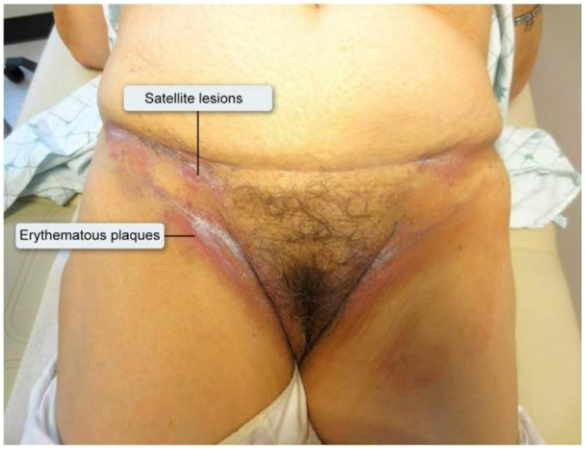
Pap test cytology has **low sensitivity and specificity** for identifying BV; therefore, further evaluation is warranted.

Patients with BV on cytology results should be asked about symptoms (eg, malodorous vaginal discharge). Symptomatic patients should undergo further evaluation with wet mount microscopy and potassium hydroxide whiff testing and receive treatment (eg, metronidazole, clindamycin) if the diagnosis is confirmed.

In contrast, **asymptomatic** patients (such as this one) **do not require treatment** due to the limitations of cytology results for identifying infections (ie, high false positive rate). Furthermore, treatment of asymptomatic BV is typically avoided because it may resolve spontaneously, and treatment can lead to symptomatic vaginal candidiasis.

So if a patient does a pap smear and we find clue cells we ask about symptoms and treat according it.

Candida intertrigo



intertrigo, a dermatitis that typically occurs within the inguinal, axillary, gluteal, and inframammary folds. Patients with intertrigo typically have erythematous plaques in a symmetric "kissing" or "mirror image" pattern across the skinfold and multiple **satellite lesions** near the primary infection.

The most common cause of intertrigo is ***Candida albicans***, a part of the normal flora of the urogenital and gastrointestinal tracts. Patients at risk for candida intertrigo include those with impaired immunity (eg, **systemic corticosteroid use**, diabetes mellitus), particularly when associated with increased skin moisture or friction (eg, obesity, tight-fitting clothing), which can cause skin maceration and trauma.

Diagnosis is typically clinical but can be confirmed by visualization of hyphae or pseudohyphae on microscopic examination of skin scrapings from affected areas. Treatment is typically with **topical azoles** (eg, **clotrimazole**, ketoconazole), which help decrease the spread of infection and have some anti-inflammatory and antibacterial properties.

Characteristics of ulcerative sexually transmitted diseases			
Disease	**Causative agent**	**Features of primary lesion**	**Initial lesion painful?**
Chancroid	*Haemophilus ducreyi*	• Multiple & deep ulcers • Base may have gray to yellow exudate • Organisms often clump in long parallel strands ("school of fish")	Yes
Genital herpes	Herpes simplex virus 1 & 2	• Multiple, small, grouped ulcers • Shallow with erythematous base • Multinucleated giant cells & intranuclear inclusions (Cowdry type A)	Yes
Granuloma inguinale (donovanosis)	*Klebsiella granulomatis*	• Extensive & progressive ulcerative lesions without lymphadenopathy • Base may have granulation-like tissue • Deeply staining gram-negative intracytoplasmic cysts (Donovan bodies)	No
Syphilis	*Treponema pallidum*	• Single, indurated, well-circumscribed ulcer • Nonexudative base • Painless inguinal lymphadenopathy • Thin, delicate, corkscrew-shaped organisms on dark-field microscopy	No
Lymphogranuloma venereum	*Chlamydia trachomatis*	• Small & shallow ulcers • Large, painful, coalesced inguinal lymph nodes ("buboes") • Intracytoplasmic chlamydial inclusion bodies in epithelial cells & leukocytes	No

Chancroid

- Chancroid is an acute, localized, contagious disease characterized by painful genital ulcers and suppuration of the inguinal lymph nodes.

- It is caused by Haemophilus ducreyi.

- <u>Diagnosis:</u>

- Diagnosis is made on clinical findings; do a Gram stain initially with culture to confirm.

- <u>Treatment:</u>

- Treatment is azithromycin single dose or ceftriaxone intramuscularly (single dose).

Lymphogranuloma Venereum

- Lymphogranuloma venereum is a contagious, sexually transmitted disease having a transitory primary lesion followed by suppurative lymphangitis.

- It is caused by Chlamydia trachomatis serotypes L1-L3.

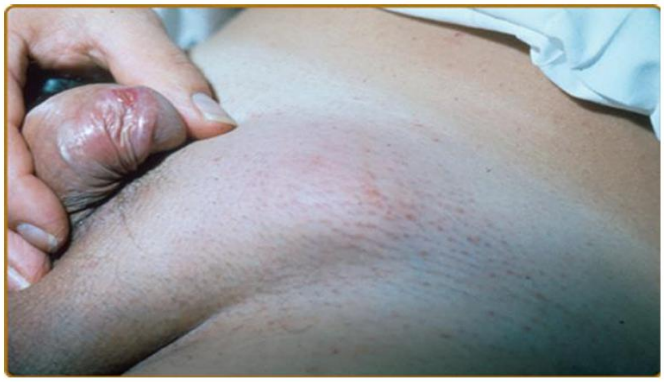
- Clinical findings include the following:

- LGV is a chronic disease characterized by an initial painless small ulcer on the genital mucosa that contains cells infected with C. trachomatis.

- The painless nature of this ulcer helps distinguish LGV from other entities.

- This ulcer is followed weeks later by swollen, painful inguinal nodes that coalesce, ulcerate, and rupture; these are referred to as buboes.

- If left untreated, this condition can cause fibrosis, lymphatic obstruction, and anogenital strictures and fistulas.



- Diagnosis:

- Diagnosis is made by clinical examination, history, and a high or rising titer of complement fixing antibodies.

- Isolate chlamydia from pus in buboes.

- Treatment:

- Treat with doxycycline or erythromycin.

Granuloma Inguinale

- Granuloma inguinale is a chronic granulomatous condition, probably spread by sexual contact. It is caused by Donovania granulomatis.

- A painless, red nodule will develop into an elevated granulomatous mass.

- In men, it is seen on the penis, scrotum, groin, and thighs (In homosexual men, the anus and buttocks are common areas).

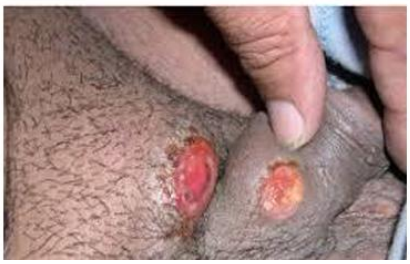
- In women it is found on the vulva, vagina, and perineum.

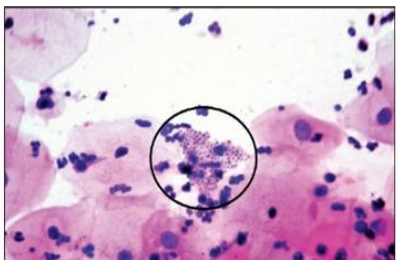
- <u>Diagnosis:</u>

- Diagnosis is made clinically and by performing a Giemsa or Wright stain (Donovan bodies) or smear of lesion.

- <u>Treatment:</u>

- Treat with doxycycline, ceftriaxone, or TMP/SMZ.





Genital Herpes

- Genital herpes is generally the herpes virus type II, although type I may be seen.

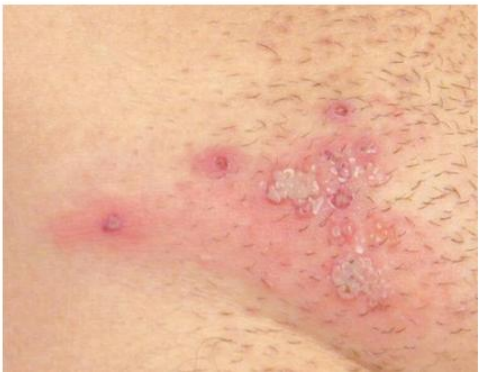
- <u>Finding:</u>

- HSV lesions initially appear as a group of painful vesicles on an erythematous base that evolve into a group of shallow ulcers that eventually crust over.

- Patients can have painful urination (dysuria) due to urine contact on the open ulcers and a sterile pyuria (leukocytes on urinalysis but negative urine culture) due to vulvar inflammation.

- Inguinal lymphadenopathy.

- Lesions are commonly seen on the penis (men) and on the labia, clitoris, perineum, vagina, and cervix (women).



- Diagnosis:

- Suspected clinical diagnosis of genital HSV requires laboratory confirmation via viral culture or PCR testing.

- Treatment:

- Treat with oral acyclovir, famciclovir, or valacyclovir.

- Foscarnet for acyclovir-resistant herpes.

❖ N.B:

- The risk for neonatal HSV infection is drastically increased if the infant passes through the vaginal canal and is directly exposed to an active HSV eruption.
- Cesarean delivery is recommended to all women who are in labor with active genital HSV lesions or prodromal symptoms (burning, pain).

Patients with a primary infection often have systemic symptoms (eg, fever) and develop a **tender inguinal lymphadenopathy**. HSV evolves from vesicles to open ulcers; patients with ulcers often have associated dysuria and **sterile pyuria** (eg, white blood cells [WBCs] but no bacteria on urinalysis) due to urethral and vulvar inflammation and passage of urine over the open lesion. In addition, some patients may develop **acute urinary retention** (eg, suprapubic fullness) due to either reluctance to urinate or from a lumbosacral neuropathy that can complicate the infection.

Without treatment, most immunocompetent patients with primary HSV have **spontaneous resolution of symptoms within a week**. However, many patients will experience disease recurrence, particularly during the first year after primary infection. Afterward, recurrence becomes less frequent due to improved cell-mediated immunity. Antivirals (eg, **acyclovir**, valacyclovir) are used to **reduce symptom duration** and **frequency of recurrences** but do not eliminate recurrences.

Infectious genital ulcers		
Painful	Herpes simplex virus	Pustules, vesicles, or small ulcers on erythematous base Tender lymphadenopathy Systemic symptoms common
	Haemophilus ducreyi **(chancroid)**	Larger, deep ulcers with gray/yellow exudate Well-demarcated borders & soft, friable base Severe lymphadenopathy that may suppurate
Painless	*Treponema pallidum* (syphilis)	Usually single ulcer (chancre) Indurated borders & hard, nonpurulent base
	Chlamydia trachomatis **serovars L1-L3 (lymphogranuloma venereum)**	Initial small, shallow ulcers (often missed) Then painful & fluctuant adenitis (buboes)

Syphilis

- Syphilis is a systemic contagious disease caused by a spirochete (Treponema pallidum).

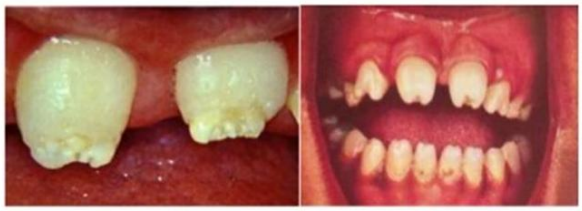
- Syphilis can be classified as being congenital or acquired.

- Congenital:

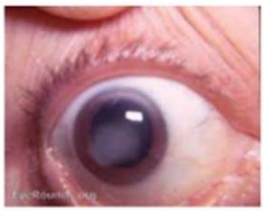
- Treponema pallidum readily crosses the placenta and is associated with many adverse fetal outcomes, including intrauterine growth restriction, fetal death, and congenital infection.

- Congenital syphilis presents with facial abnormalities such as rhagades (linear scars at angle of mouth), snuffles (nasal discharge), saddle nose, notched (Hutchinson) teeth, mulberry molars, and short maxilla; saber shins; CN VIII deafness.

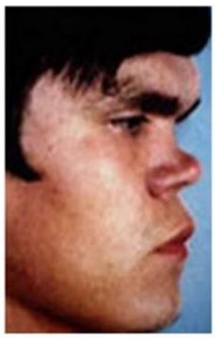
- To prevent, treat mother early in pregnancy, as placental transmission typically occurs after first trimester.



Hutchinson's teeth:
widely spaced, pegged teeth

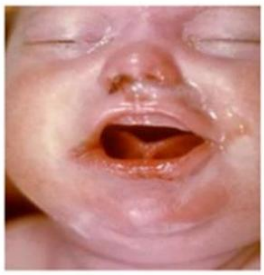


Keratitis



Frontal bossing
Saddle nose

Snuffles



- Acquired:

1. Primary stage:

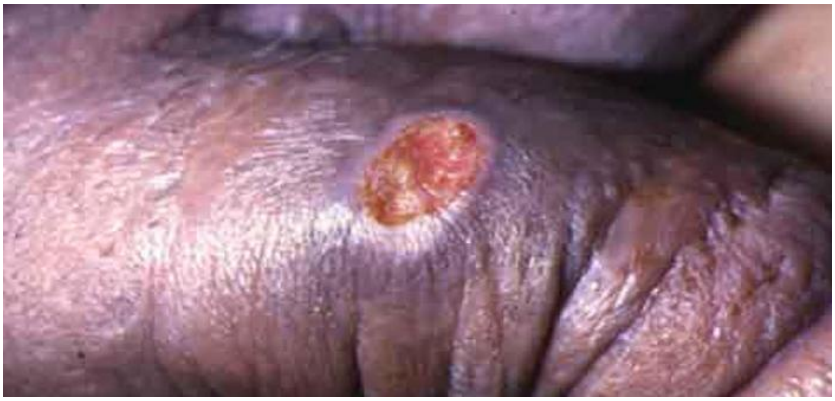
- Chancre appears by week 3 and disappears in 10-90 days; this is usually manifested as a single hard, painless ulcer called chancre.

- The chancre represents the primary site of initial multiplication.

- It usually appears on the penis, labia, cervix, anorectal region, or around the mouth.

- The regional lymph nodes also become enlarged (painless).

- The chancre heals within 4-6 weeks, even without treatment.



2. Secondary stage:

- Cutaneous rashes appear 6-12 weeks after infection, this stage has four cardinal features:
  - Disseminated disease with constitutional symptoms.
  - Maculopapular rash (including palms and soles).
  - Condylomata lata (smooth, moist, painless, wart-like white lesions on genitals).
  - Widespread lymphadenopathy (particularly epitrochlear).
- Secondary syphilis = Systemic.
- The symptoms usually last 4-6 months and disappear spontaneously.

3. Latent stage:

- There are neither symptoms nor lesions during this stage, and the serology is positive.
- This stage can range from a few months to a lifetime.

4. Tertiary syphilis:

- This may occur in about 40% of untreated cases.
- This stage is characterized by gumma formation (chronic granulomas) in the internal organs and bones and syphilitic lesions that may lead to cardiovascular syphilis and neurosyphilis (tabes dorsalis).
- Aortitis (vasa vasorum destruction).
- Neurosyphilis (tabes dorsalis, general paresis), Argyll Robertson pupil (constricts with accommodation but is not reactive to light; also called “prostitute’s pupil” since it accommodates but does not react).
- Signs: broad-based ataxia, ⊕ Romberg, Charcot joint.
- Serology is positive.

- Diagnosis:

- Screening tests are the VDRL and RPR; specific tests are the FTA-ABS, MHA-TP, and Darkfield exam of chancre.

- If dark-field is positive for spirochetes, no further testing for syphilis is necessary.

- False-Positive results on VDRL with:
  - Pregnancy.
  - Viral infection (Infectious Mononucleosis, Hepatitis).
  - Drugs.
  - Rheumatic fever.
  - Lupus and Leprosy.

Syphilis: diagnostic serology

Nontreponemal (RPR, VDRL)	Antibody to cardiolipin-cholesterol-lecithin antigen Quantitative (titers) Possible negative result in early infection Decrease in titers confirms treatment
Treponemal (FTA-ABS, TP-EIA)	Antibody to treponemal antigens Qualitative (reactive/nonreactive) Greater sensitivity in early infection Positive even after treatment

FTA-ABS = fluorescent treponemal antibody absorption; **RPR** = rapid plasma reagin; **TP-EIA** = *Treponema pallidum* enzyme immunoassay.

Two types of syphilis serologic tests are usually used in combination for diagnosis:

- Nontreponemal (eg, rapid plasma reagin [RPR])
- Treponemal (eg, fluorescent treponemal antibody absorption [FTA-ABS])

In general, **nontreponemal testing** (eg, **RPR**) is obtained first due to low cost and availability. An initial RPR titer can also be used later to monitor response to treatment (ie, decreasing titers). However, nontreponemal testing has a higher rate of **false-negative results** (20%-30%), particularly for primary syphilis. This is because testing depends on the patient's development of antibodies, which may be absent or low in patients with **early infection** or severe immunosuppression.

Patients with negative serology and strong clinical evidence (eg, chancre) of primary syphilis are treated empirically with intramuscular benzathine penicillinG.

- Treatment:
- Penicillin is the drug of choice for all stages of syphilis.

- Penicillin-allergic patients receive doxycycline for primary and secondary syphilis but must be desensitized in tertiary syphilis.

- Pregnant patients with syphilis require treatment with penicillin as alternate antibiotic choices are ineffective, contraindicated, or have limited data in pregnancy. Patients with penicillin allergy should have a penicillin skin test to evaluate for the presence of an IgE-mediated response. If the test is positive, patients are desensitized to penicillin prior to receiving treatment with intramuscular penicillin G benzathine.

- All patients with syphilis require nontreponemal titers (RPR) at the time of treatment and at 6-12 months to ensure treatment response (a 4-fold drop in titers). This is especially crucial in patients receiving alternate treatment as the risk of treatment failure is much higher than the risk in those who receive penicillin.

So U know that the treatment is good if there is decrease in the RPR titers by 4folds after 6-12 months of treatment.

Selective estrogen receptor modulators	
Drugs	Tamoxifen Raloxifene
Mechanism of action	Competitive inhibitor of estrogen binding Mixed agonist/antagonist action
Indications	Prevention of breast cancer in high-risk patients Tamoxifen: adjuvant treatment of breast cancer Raloxifene: postmenopausal osteoporosis
Adverse effects	Hot flashes Venous thromboembolism Endometrial hyperplasia & carcinoma (tamoxifen only) Uterine sarcoma (tamoxifen only)

Tamoxifen is an estrogen antagonist in the breast and is therefore used as adjuvant therapy for estrogen receptor–positive breast cancer. It also reduces the risk for breast cancer recurrence and the development of new cancer in the opposite breast.

Hot flashes are the **most common adverse effect**, experienced by up to 80% of patients taking tamoxifen. Tamoxifen is theorized to exhibit antiestrogenic activity in the CNS and to cause thermoregulatory dysfunction in the anterior hypothalamus via a mechanism similar to the pathophysiology of menopausal hot flashes. Additional adverse effects (related to estrogenic activity) include **thromboembolism**, **endometrial cancer**, and **uterine sarcoma**.

- **Selective estrogen receptor modulators** increase the risk for venous thromboembolism by *increasing* **protein C resistance.**
  - *due to estrogen agonist activity*
    - *In addition, they* ***increase 2/7/8/10***, ***decrease antithrombin 3***, *and* ***decrease protein S***

- What is the *most common* side effect of **tamoxifen**?
  - **Hot flashes (80%)**
    - *due to anti-estrogenic activity in the CNS which causes* ***thermoregulatory dysfunction*** *in the anterior hypothalamus*

> 💡 Tamoxifen **decreases the blood cholesterol level** and may **protect** against **coronary artery disease.**

- Tamoxifen increases the risk for **endometrial hyperplasia/cancer** and **uterine sarcoma** in postmenopausal women.
  - However, asymptomatic patients on tamoxifen **do not require routine screening** for these complications.
    - Evaluation via ultrasonography or endometrial **biopsy** is indicated only for **symptomatic patients**.`

Cancer:
Vulva:

	Vulvar cancer
Etiology	Persistent HPV infection Chronic inflammation
Risk factors	Tobacco use Vulvar lichen sclerosus Immunodeficiency Prior cervical cancer Vulvar/cervical intraepithelial neoplasia
Clinical features	Vulvar pruritus Vulvar plaque/ulcer Abnormal bleeding
Diagnosis	Biopsy

Patients with chronic lichen sclerosus have continued inflammation and hyperplasia of the vulvar epithelium that can result in **malignant transformation** and development of a neoplastic lesion. This lesion typically develops over the labia majora and can become pruritic, friable, and ulcerated.

Constant dysplastic changes over the vulvar squamous cells can result in a **unifocal**, **friable plaque** or ulcer, typically on the labia majora, that produces persistent **vulvar irritation** (eg, vulvar excoriations, erythema) and/or pain. Patients may also have **intermittent bleeding** and **dyspareunia** (as seen in this patient) or an asymptomatic lesion found on routine examination.

In patients with lesions concerning for malignancy, the **best next step** in management is **vulvar biopsy**, which distinguishes between benign (eg, lichen sclerosus) and neoplastic disease. In those with neoplastic changes, biopsy further determines the depth of invasion and differentiates between noninvasive (ie, vulvar intraepithelial neoplasia) or invasive (ie, vulvar cancer) disease.

If the patient's lesion is benign on biopsy and is a recurrence of lichen sclerosus, she can be retreated with high-dose corticosteroids. However, if the lesion is neoplastic, additional management is indicated. Patients with noninvasive disease can be treated with either medical therapy (eg, imiquimod) or laser ablative therapy. Those with invasive disease require surgery (eg, wide local excision ± lymph node dissection) and possible chemoradiation.

Vaginal:

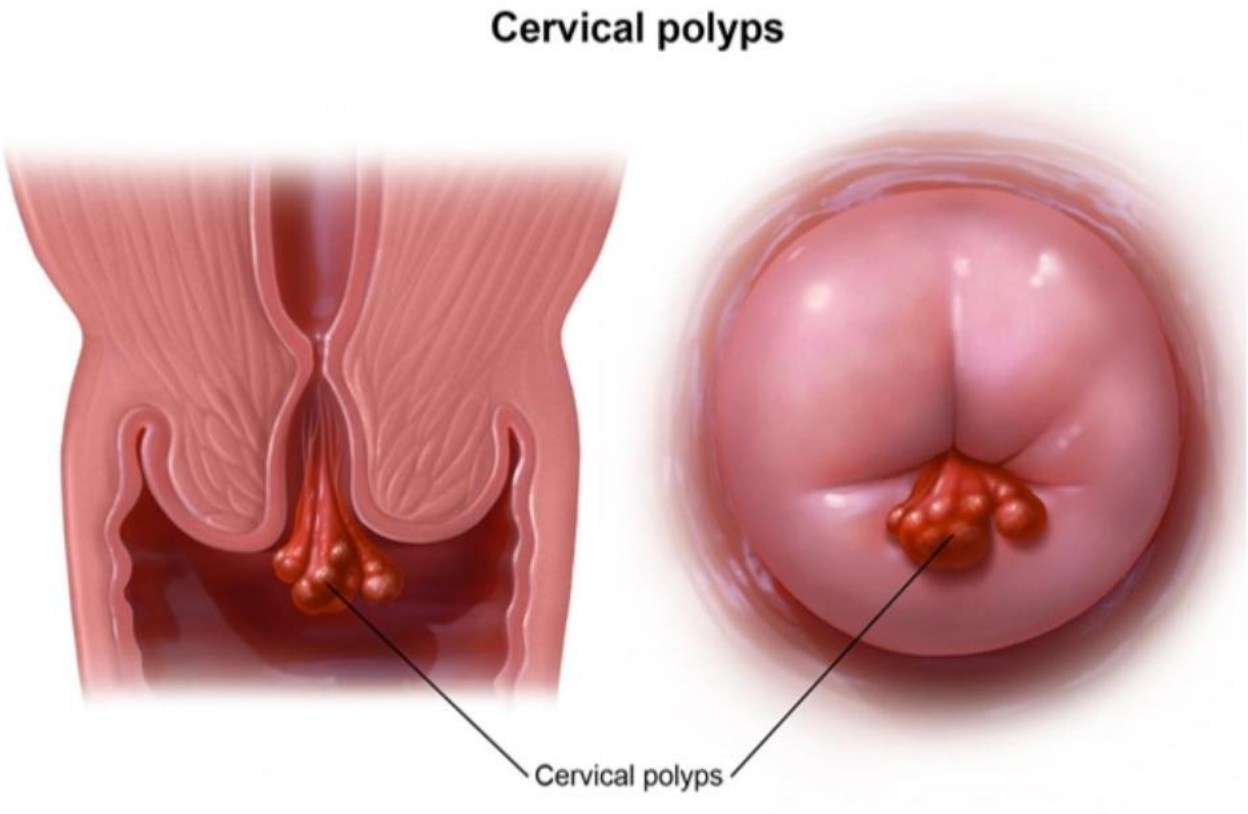
	Vaginal cancer
Risk factors	Age >60 Human papillomavirus infection Tobacco use In utero DES exposure (clear cell adenocarcinoma only)
Clinical features	Vaginal bleeding Malodorous vaginal discharge Irregular vaginal lesion
Diagnosis	Vaginal biopsy
Management	Surgery ± chemoradiation

DES = diethylstilbestrol.

Although many women are diagnosed with vaginal cancer on routine screening, those with large, symptomatic lesions often have vaginal bleeding and malodorous vaginal discharge. Additional clinical features that are suggestive of metastatic disease can include pelvic pain, urinary symptoms (eg, hematuria), and bulk symptoms (eg, constipation). Lesions typically appear as an irregular plaque or ulcer in the **upper third of the posterior vagina**, as seen in this patient.

As with cervical cancer, vaginal cancer is due to **persistent human papillomavirus (HPV) infection** with high-risk types 16 and 18. **Chronic tobacco use** decreases the immune response and prevents viral clearing. This increases the risk of persistent HPV infection and thereby allows continued viral replication and eventual **metaplastic changes** within the vaginal squamous cell epithelium. Diagnosis is with biopsy of the lesion to evaluate for depth of invasion and help determine management options.
Diethylstilbestrol was previously used for spontaneous abortion prevention but was discontinued due to multiple adverse effects (eg, infertility due to anatomic defects). Women who were exposed to this medication in utero are at increased risk of vaginal clear cell adenocarcinoma, not squamous cell carcinoma.

Cervical:



- Cervical polyps are fingerlike growths that start on the surface of the cervix or endocervical canal.
- These small, fragile growths hang from a stalk and push through the cervical opening.
- The cause of cervical polyps is not completely understood.
- They may be associated with chronic inflammation, an abnormal response to increased levels of estrogen, or thrombosed cervical blood vessels.
- <u>Findings:</u>

- The history is usually positive for vaginal bleeding, often after intercourse. This bleeding occurs between normal menstrual periods.
- Speculum examination reveals smooth, red or purple, fingerlike projections from the cervical canal.

- <u>Management:</u>

- Polyps can be removed by gentle twisting or by tying a surgical string around the base and cutting it off. Removal of the polyp's base is done by electrocautery or with a laser.
- Because many polyps are infected, an antibiotic may be given after the removal even if there are no or few signs of infection.

Nabothian Cysts

- A nabothian cyst is a mucus-filled cyst on the surface of the uterine cervix.

- The cervical canal is lined by glandular cells that normally secrete mucus. These endocervical glands can become covered by squamous epithelium through metaplasia.

- These nests of glandular cells (nabothian glands) on the cervix may become filled with secretions.

- As secretions accumulate, a smooth, rounded lump may form just under the surface of the cervix and become large enough to be seen or felt upon examination.

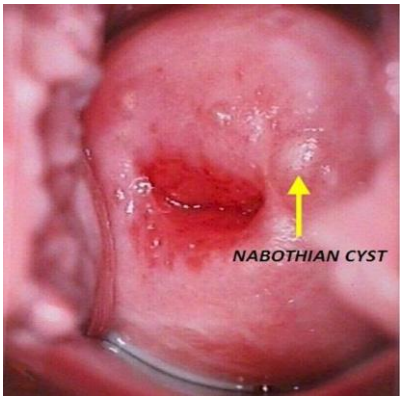
- <u>Findings:</u>

- Pelvic examination reveals a small, smooth, rounded lump (or collection of lumps) on the surface of the cervix.

- <u>Management:</u>

- No treatment is necessary.

- However, nabothian cysts do not clear spontaneously. They can be easily cured through electrocautery or cryotherapy.



Pap Smears (ACS 2012)

Begin	Age 21	All patients regardless of onset of sex
Repeat	Every 3 yrs	from ages 21 & 29 (avoid HPV testing)
	Every 3 yrs	If ≥ age 30 & neg cytology without HPV
	Every 5 yrs	If ≥ age 30 & both neg cytology & HPV
End	Age 65	If no abnormal Pap test past 10 yrs
	Any age	If TAH/TVH for benign reasons
Continue	For ≥ 20 yrs after diagnosis if any CIN 2, 3 or CA	

Cervical cancer screening	
Age <21	• No screening
Age 21-29	• Cytology every 3 years
Age 30-65	• Cytology every 3 years OR • Cytology plus HPV testing every 5 years OR • Primary HPV testing every 5 years
Age >65	• No screening if negative prior screens & low risk
Hysterectomy (with cervix removed)	• No screening if negative prior screens & low risk
HIV	• Onset of sexual intercourse or time of HIV diagnosis (whichever is first) • Annually until ≥3 normal results, then routine testing
Immunosuppressed (eg, SLE, organ transplant)	• Onset of sexual intercourse • Annual Pap test with HPV cotesting
HPV = human papillomavirus; **SLE** = systemic lupus erythematosus.	

When to stop Pap testing
• Age 65 or hysterectomy PLUS • No history of cervical intraepithelial neoplasia 2 or higher AND • 3 consecutive negative Pap tests OR • 2 consecutive negative co-testing results

• *Patients with **cervical cancer risk factors** (e.g. immunosuppression, tobacco use, DES exposure, high-risk sexual activity) may need continued Pap testing*

• If a patient has a history of **CIN 2 or higher**, Pap testing should continue for another **20** years after detection.

- Pap Smear Classification:

- The Bethesda system is the current classification used in the United States.

- Negative for intraepithelial lesion or malignancy.

- Abnormal squamous cells (99% of abnormal Pap smears):

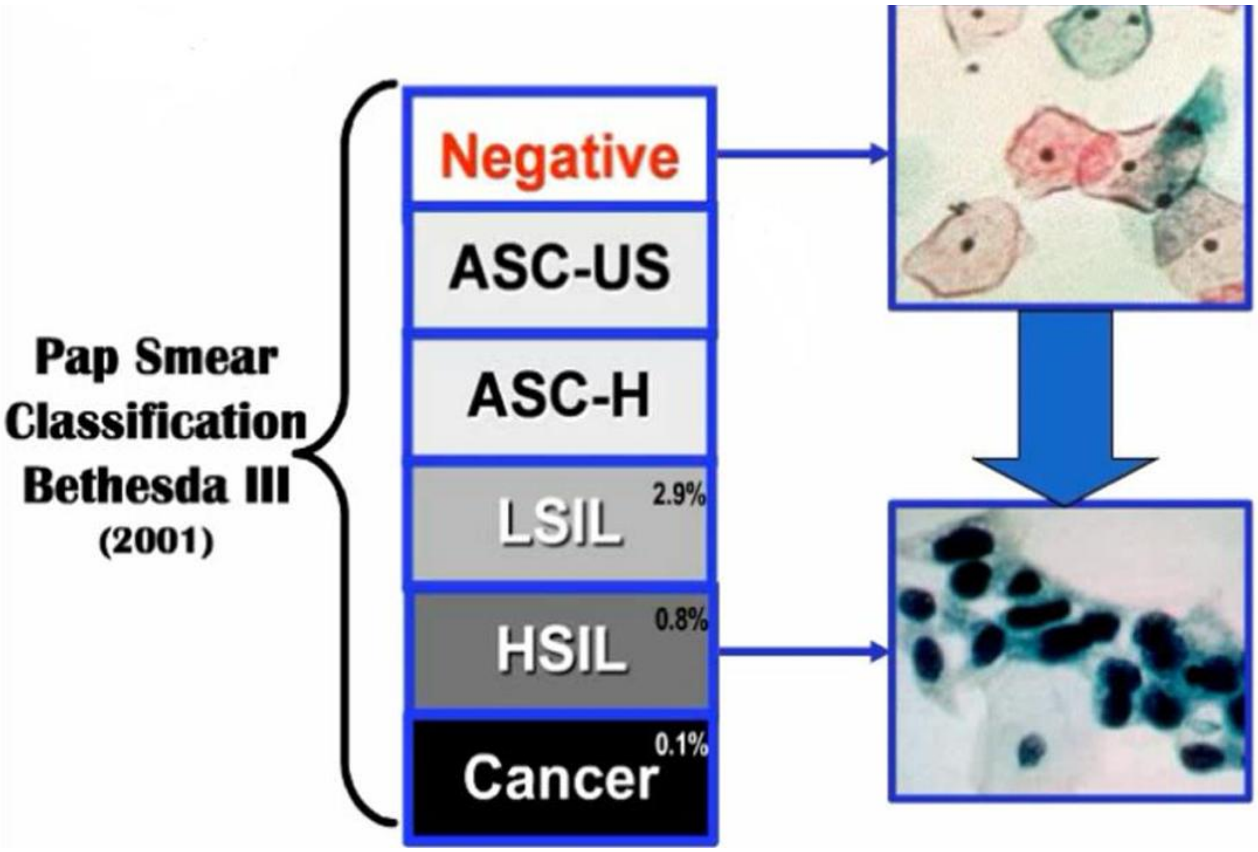
- ASC-US (atypical squamous cells of undetermined significance): changes suggestive of but not adequate to label LSIL.

- LSIL (low-grade squamous intraepithelial lesion): biopsy is expected to show histologic findings of HPV, mild dysplasia, or CIN 1.

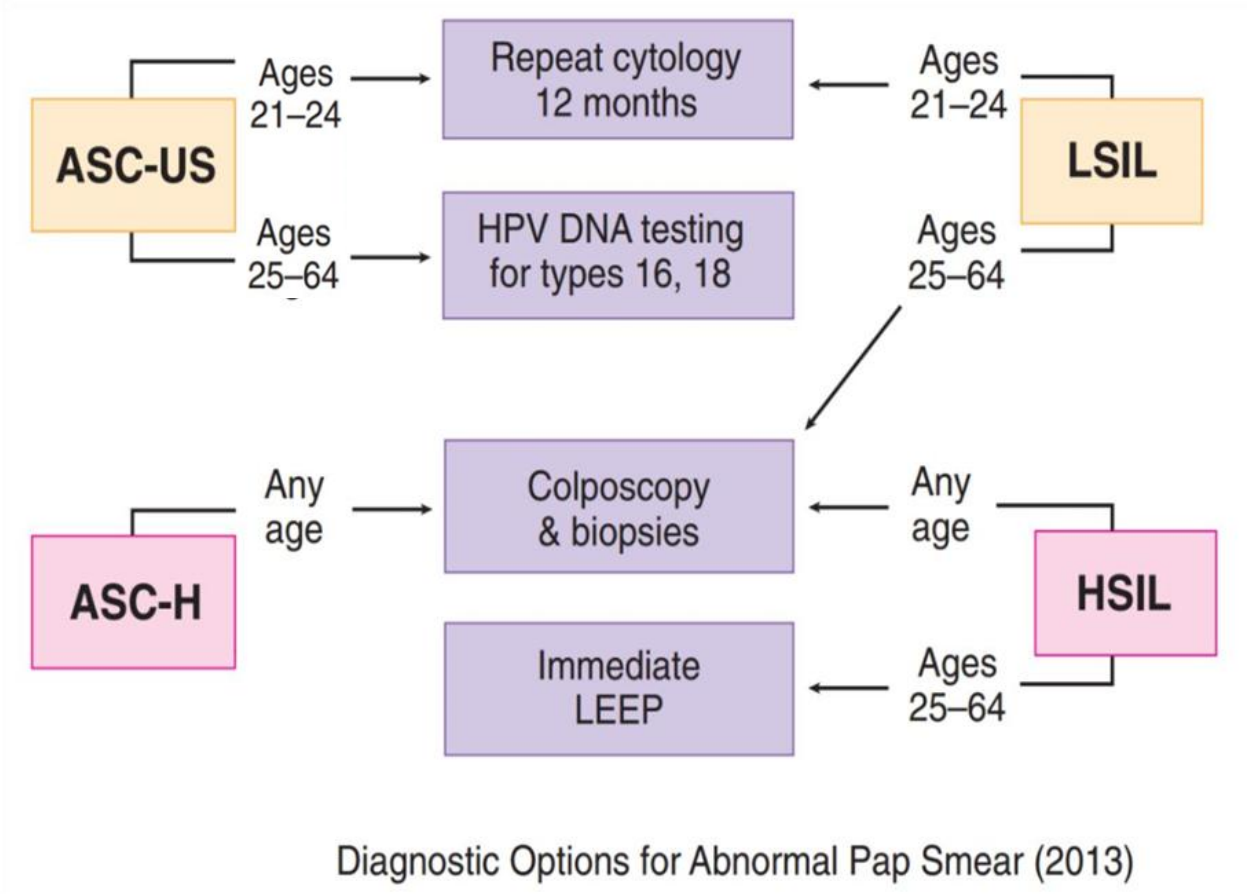
- ASC-H (atypical squamous cells can't rule out HSIL): changes suggestive of but not adequate to label HSIL.

- HSIL (high-grade squamous intraepithelial lesion): biopsy is expected to show histologic findings of moderate-severe dysplasia, CIN 2, CIN 3, or CIS.

- Squamous cell carcinoma: biopsy is expected to show histologic findings of invasive cancer.



Diagnostic Approach to Abnormal Pap Smears:



Diagnostic Options for Abnormal Pap Smear (2013)

A. Accelerated repeat Pap:

- This is an option for findings of ASC-US in patients of any age, and the preferred option with either ASC-US or LSIL in patients ages 21-24. Repeat the Pap in 12 months:
  - If repeat cytology is negative, repeat Pap in another 12 months.
  - If repeat cytology is anything other than negative, proceed to colposcopy and biopsies.

B. HPV DNA testing:

- This is the preferred option for findings of ASC-US in patients age >25.It is acceptable but not preferred in patients ages 21-24.
- If liquid-based cytology was used on the initial Pap, one can use this specimen for DNA testing.
- If conventional methods were used, repeat a second Pap.
- Perform colposcopy only if high-risk HPV DNA is identified.

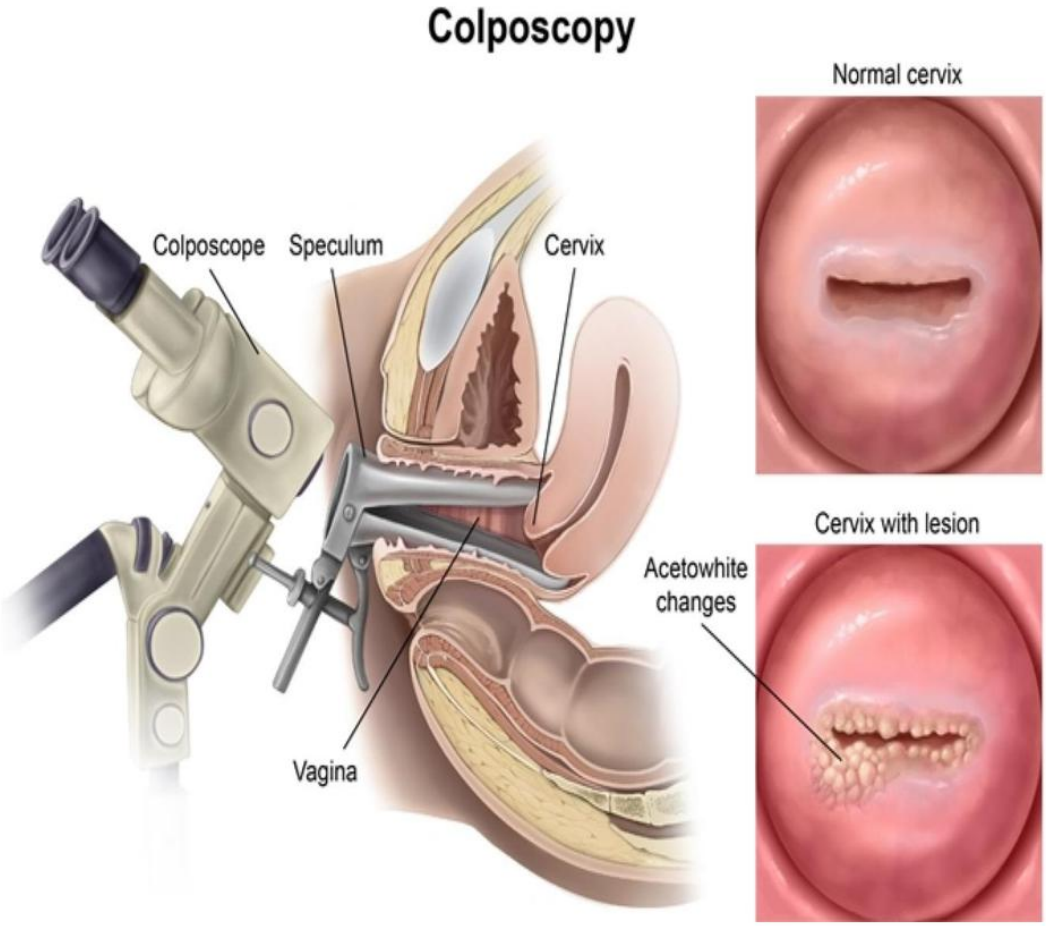
A Pap test showing atypical squamous cells of undetermined significance (ASC-US) exhibits cervical cells demonstrating reactive changes, often due to infections (eg, *Candida*); ASC-US is the most common abnormal finding on Pap testing. It alone does not meet cytologic criteria for premalignant disease.

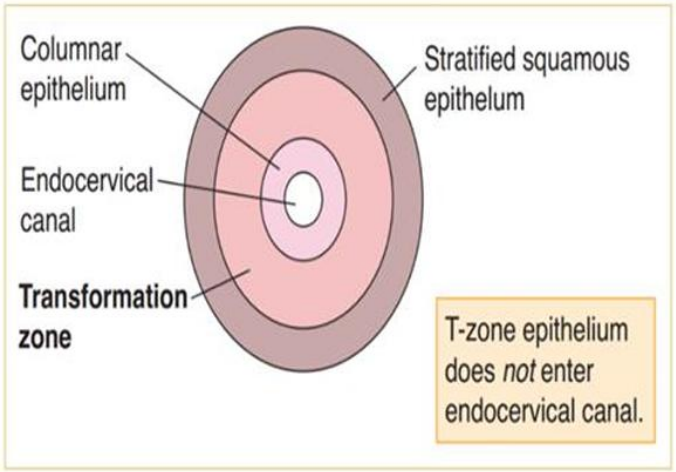
C. Colposcopy:

- This is indicated for evaluation of LSIL in patients age ≥25, and all patients with ASC-H and HSIL.

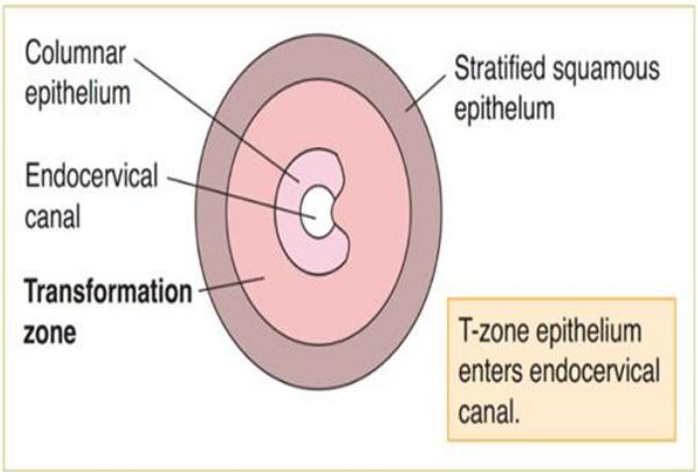
- Satisfactory or adequate colposcopy is diagnosed if the entire T-zone is visualized and no lesions disappear into the endocervical canal.

- Unsatisfactory or inadequate colposcopy is diagnosed if the entire T-zone cannot be fully visualized.





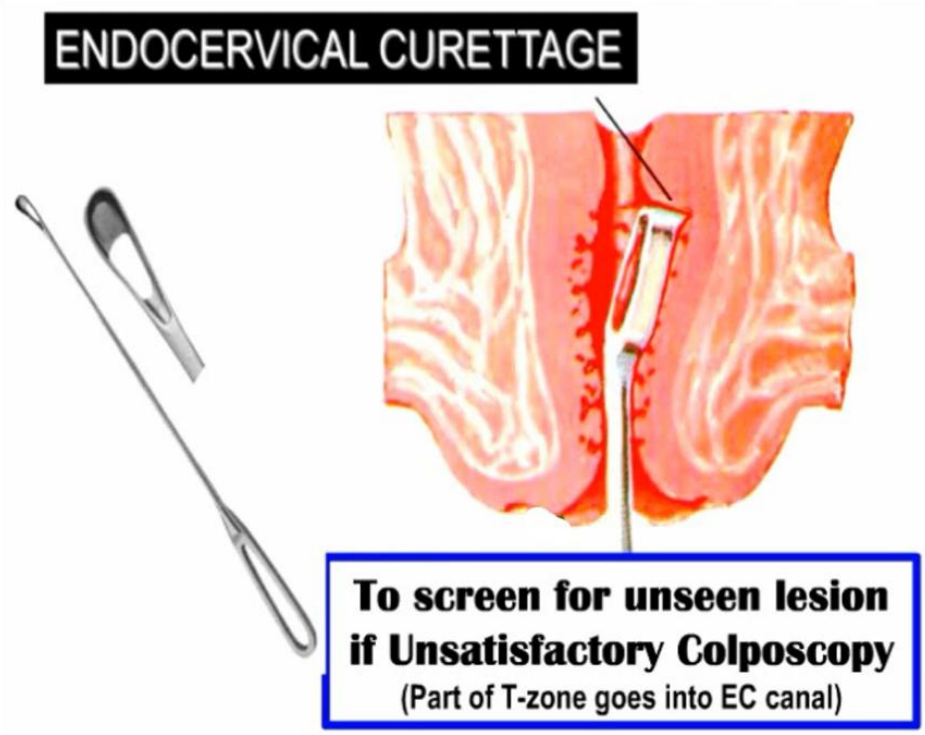
Cervical Dysplasia: Satisfactory Colposcopy



Cervical Dysplasia: Unsatisfactory Colposcopy

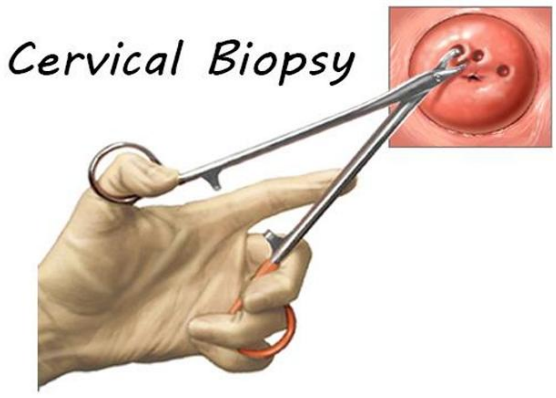
D. Endocervical curettage (ECC):

- All nonpregnant patients undergoing colposcopy which shows metaplastic epithelium entering the endocervical canal will undergo an ECC to rule out endocervical lesions.



E. Ectocervical biopsy:

- Lesions identified on the ectocervix by colposcopy (mosaicism, punctation, white lesions, abnormal vessels) are biopsied and sent for histology.

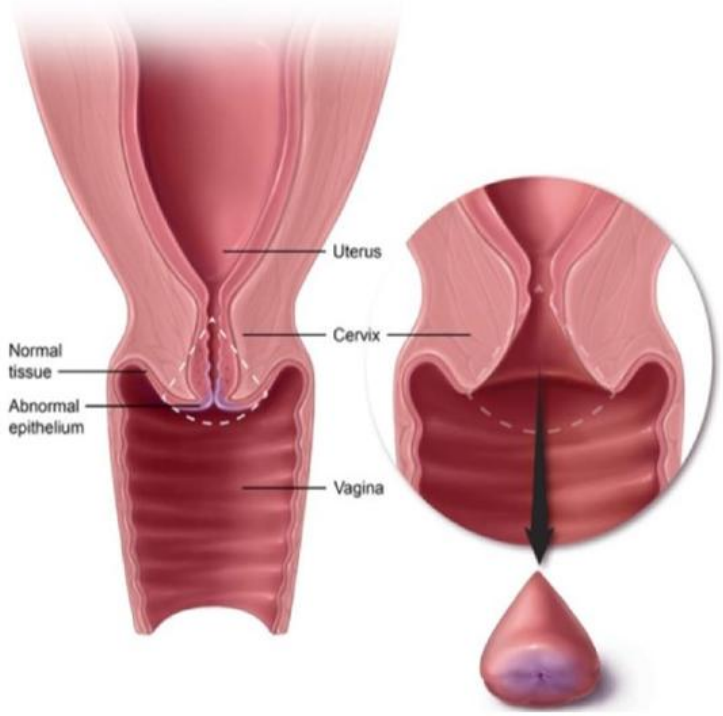


G. Cone biopsy:

- If the Pap smear is worse than the histology (suggesting the site of abnormal Pap smear cells was not biopsied), then a cone biopsy is performed.

- Other indications for conization of the cervix include abnormal ECC histology, a lesion seen entering the endocervical canal, and a biopsy showing microinvasive carcinoma of the cervix.

- Deep cone biopsies can result in an incompetent cervix. Another risk of cone biopsy is cervical stenosis.



- Observation and follow-up without treatment:
  - Appropriate for CIN 1.
  - Include any of the following: repeat Pap in 6 and 12 months; colposcopy and repeat Pap in 12 months; or HPV DNA testing in 12 months.

- Ablative modalities:
  - Can be used for CIN 1, 2, and 3.
  - These include cryotherapy (freezing), laser vaporization, and electrofulguration.

- Excisional procedures:
  - Can be used for CIN 1, 2, and 3.
  - These include LEEP (loop electrosurgical excision procedure) or cold knife conization.

- Hysterectomy: only acceptable with biopsy-confirmed, recurrent CIN 2 or 3.

- Cervical stenosis an abnormal stricture of the cervical canal, is a potential complication of cervical conization due to scar tissue. Cervical stenosis may impede menstrual flow and cause secondary dysmenorrhea or amenorrhea. The obstruction of the cervical outlet may prevent sperm entry, resulting in impaired fertility.

- Follow-Up:

- Patients treated with either ablative or excisional procedures require follow-up repeat Pap smears, colposcopy and Pap smear, or HPV DNA testing every 4 to 6 months for 2 years.

- Prevention of Cervical Dysplasia by Vaccination:

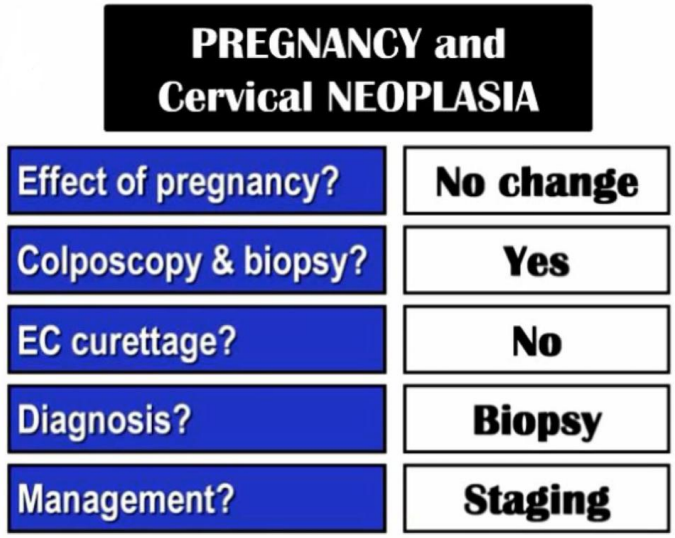
- The 9 valent HPV recombinant vaccine [Gardasil-9] is recommended for all females age 9-26, with target age 11-12.

- The vaccine uses noninfectious particles to protect against 9 HPV types (6, 11, 16, 18, 31, 33, 45, 52, 58).

Human papillomavirus	
Disease associations	• Cervical cancer • Vulvar & vaginal cancers • Anal cancer • Penile cancer • Oropharyngeal cancer • Genital warts
Vaccine indications	• All girls & women* age 11-26 • Boys & men age 9-21 (9-26 for men who have sex with men; individuals with HIV)

*Including those with history of genital warts, abnormal cytology, or positive humanpapillomavirus DNA test.

- Diagnostic Tests/Findings:



- Effect of pregnancy:
  - Pregnancy does not predispose to abnormal cytology and does not accelerate precancerous lesion progression into invasive carcinoma.

- Colposcopy and biopsy:
  - A patient who is pregnant with an abnormal Pap smear should be evaluated in the same fashion as when in a nonpregnant state.
  - An abnormal Pap smear is followed with colposcopy.
  - Any abnormal lesions of the ectocervix are biopsied.

- Perform an ECC? Owing to increased cervical vascularity, ECC is not performed during pregnancy.

• What is the *next step* in management for a **pregnant** patient with **high-grade squamous intraepithelial lesion** discovered on Pap testing?

Colposcopy and biopsy (safe during pregnancy)

Pap smear results requiring endometrial evaluation	
Result	**Group requiring endometrial sampling**

Benign-appearing endometrial cells	• Premenopausal women with:
	  ◦ Abnormal uterine bleeding **OR**
	  ◦ Risk for endometrial hyperplasia
	• Postmenopausal women

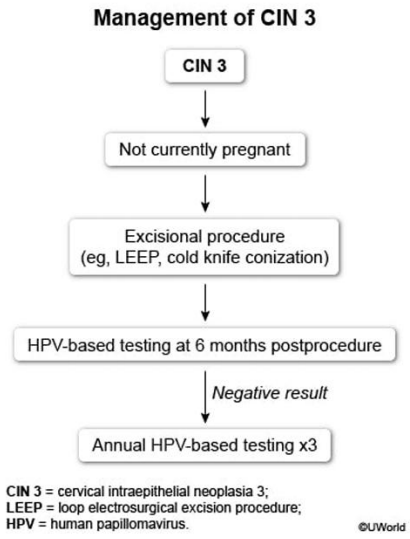
Atypical glandular cells	• Women age ≥35 **OR** at risk for endometrial hyperplasia
Atypical glandular cells, favor neoplastic	• All women

- In women **age <45**, endometrial cells are **not** reported on Pap test results because this is a common, **benign finding**, particularly during the first 10 days of the **menstrual cycle**.
- In women **age ≥45**, endometrial cells are reported because this finding is **more concerning** for **endometrial hyperplasia** or **cancer**,

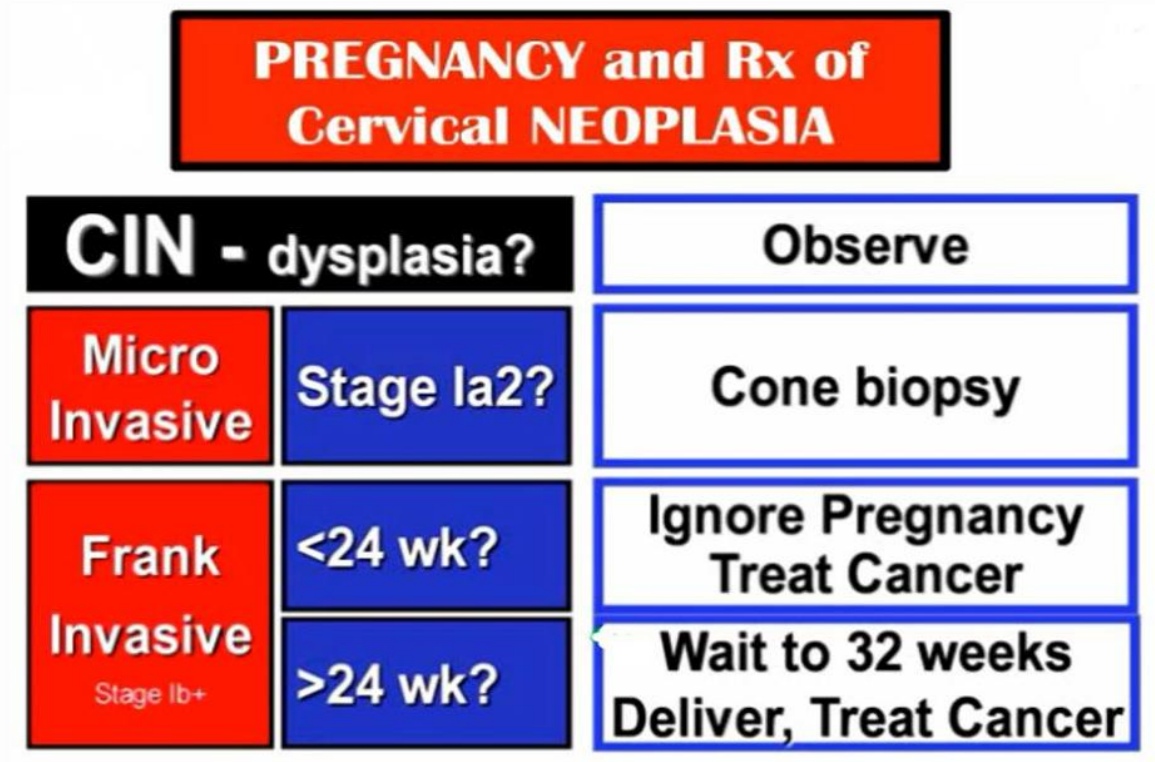
❖ N.B:

- Atypical glandular cells (AGC) on Pap testing may be due to either cervical or endometrial adenocarcinoma.
- All women age >35 with AGC or women age <35 with AGC and risk factors (obesity, anovulation) require evaluation for endometrial cancer in addition to cervical pathology. Therefore, AGC on Pap testing is investigated with colposcopy, endocervical curettage, and endometrial biopsy to evaluate the ectocervix, endocervix, and endometrium.

• Evaluation of atypical glandular cells on Pap test in women > 35 years old includes **colposcopy**, **endocervical curettage**, and **endometrial biopsy**.



After free margins on conization we do HPV testing after 6 months



- Management:

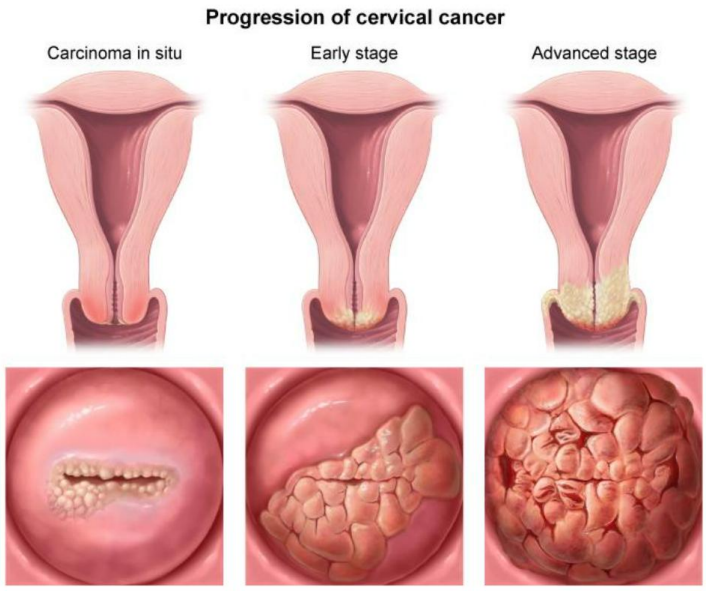
- CIN:
  - Patients with intraepithelial neoplasia or dysplasia should be followed with Pap smear and colposcopy every 3 months during the pregnancy.
  - At 6-8 weeks postpartum the patient should be reevaluated with repeat colposcopy and Pap smear.
  - Any persistent lesions can be definitively treated postpartum.

- Microinvasion:
  - Patients with microinvasive cervical cancer on biopsy during pregnancy should be evaluated with cone biopsy to ensure no frank invasion.
  - If the cone biopsy specimen shows microinvasive carcinoma during pregnancy, these patients can also be followed conservatively, delivered vaginally, reevaluated, and treated 2 months postpartum.

- Invasive cancer:
  - If the punch biopsy of the cervix reveals frankly invasive carcinoma, then treatment is based on the gestational age:

A. In general, if a diagnosis of invasive carcinoma is made before 24 weeks of pregnancy, the patient should receive definitive treatment (radical hysterectomy or radiation therapy).

B. If the diagnosis is made after 24 weeks of pregnancy, then conservative management up to about 32-33 weeks can be done to allow for fetal maturity to be achieved, at which time cesarean delivery is performed and definite treatment begun.



Risk factors for cervical cancer
Infection with high-risk HPV strains (eg, 16, 18)
History of sexually transmitted infections
Early onset of sexual activity
Multiple or high-risk sexual partners
Immunosuppression
Oral contraceptive use
Low socioeconomic status
Tobacco use

Cervical cancer is most commonly asymptomatic and detected only during screening. When symptoms do occur, as in this patient, they can include **vaginal discharge**, postcoital or **intermenstrual bleeding**, and a **cervical lesion**. Lesions suspicious for malignancy (eg, ulcerative, friable, raised), particularly in patients with risk factors (eg, long-term tobacco use), require a **cervical biopsy**. Advanced cervical cancer may present with symptoms of local invasion, including a large vascular cervical mass with heavy vaginal bleeding.

	Cervical cancer
Risk factors	Immunocompromise (eg, HIV) Early onset of sexual activity Multiple or high-risk sexual partners Previous sexually transmitted infection Tobacco use
Pathogenesis	HPV infection (types 16 & 18)
Clinical manifestations	Asymptomatic Postcoital or intermenstrual bleeding Increased vaginal discharge Inguinal lymphadenopathy Pelvic or low back pain
Diagnosis	Cervical biopsy on colposcopy

Endometrial:

	Endometrial hyperplasia/cancer
Risk factors	Excess estrogen Obesity Chronic anovulation/PCOS Nulliparity Early menarche or late menopause Tamoxifen use
Clinical features	Heavy, prolonged, intermenstrual &/or postmenopausal bleeding
Evaluation	Endometrial biopsy (gold standard) Pelvic ultrasound (postmenopausal women)
Treatment	Hyperplasia: progestin therapy or hysterectomy Cancer: hysterectomy

Endometrial adenocarcinoma is a common gynecologic malignancy that presents with either **abnormal uterine bleeding** (as seen in this premenopausal patient) or postmenopausal bleeding. Diagnosis is via endometrial biopsy, and treatment includes hysterectomy with bilateral salpingo-oophorectomy with or without chemoradiation (depending on stage).

The underlying cause of most cases of endometrial cancer is chronic **unopposed estrogen exposure**. In the uterus, estrogen causes the proliferation of the endometrium to allow for implantation associated with pregnancy. In ovulatory patients, an increase in progesterone protects against unopposed endometrial proliferation by downregulating estrogen receptors and regulating mitosis.

However, in conditions where estrogen levels are disproportionately high, the endometrium has unregulated proliferation, leading to development of endometrial cancer. The most common risk factor is **obesity** (ie, elevated BMI) because adipose tissue increases the conversion of androgens to estrogens, which results in increased uterine estrogen exposure and **chronic anovulation**. Other risk factors for endometrial cancer are related to unopposed estrogen and include nulliparity, tamoxifen use, and early age of menarche

Without treatment, endometrial hyperplasia can **progress to cancer**. In patients who may desire future fertility, initial management is with **progestin**

therapy (eg, progestin-releasing intrauterine device), which **counteracts estrogen's effects** by inhibiting endometrial proliferation and promoting differentiation. Follow-up is with repeat endometrial biopsy (eg, every 3 months). Patients with stable or improved disease continue progestin therapy, whereas those with progression to cancer typically require hysterectomy

	Uterine sarcoma
Risk factors	Pelvic radiation Tamoxifen use Postmenopausal patients
Presentation	Abnormal/postmenopausal bleeding Pelvic pain or pressure Uterine mass
Diagnosis	Ultrasound ± additional imaging Endometrial biopsy Histopathology of surgical specimen
Treatment	Hysterectomy ± Adjuvant chemotherapy, radiation therapy

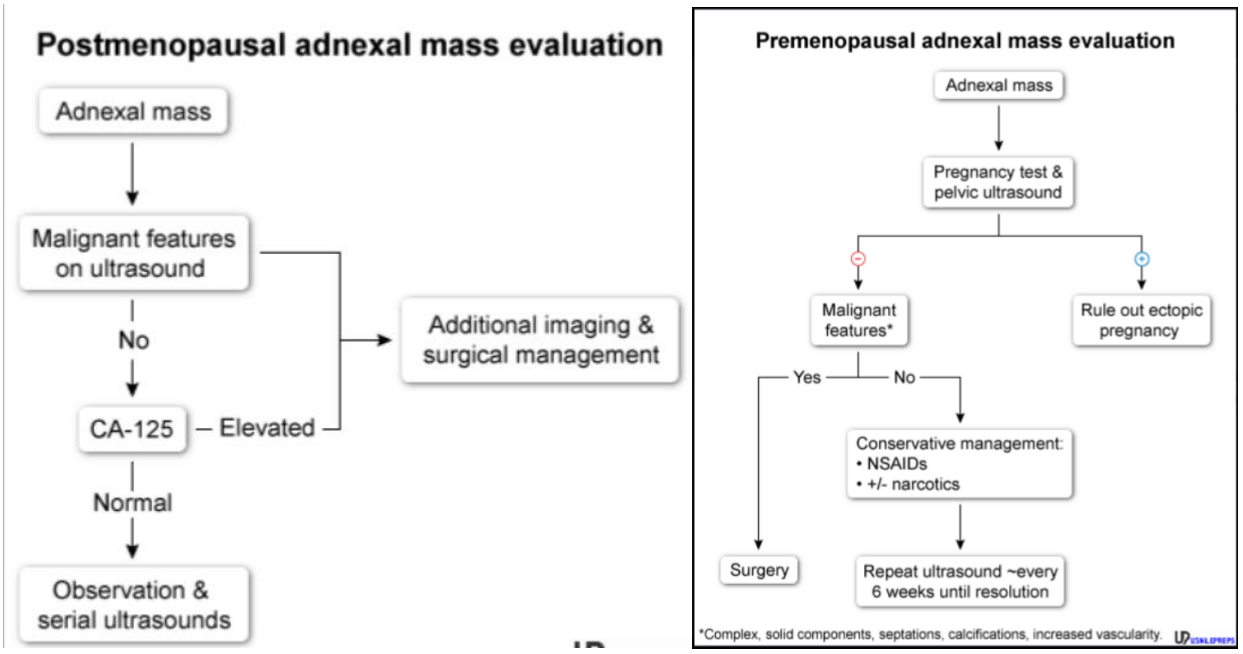
uterine sarcoma, a rare but aggressive malignancy. Uterine sarcoma typically presents with a **uterine mass** (as in this patient's enlarged, irregularly shaped uterus) that causes **bulk symptoms** (eg, pelvic pressure, constipation) and abnormal or **postmenopausal bleeding**. Uterine sarcoma is often indistinguishable from benign leiomyomata (ie, uterine fibroids); however, it should be suspected in postmenopausal women who have taken tamoxifen or have other **risk factors** (eg, pelvic radiation).

Tamoxifen is a selective estrogen receptor modulator used as an adjuvant treatment for breast cancer. Although tamoxifen has antagonist effects on the breast, it has **estrogen agonist effects on the uterus** and therefore increases the risk of uterine sarcoma and endometrial hyperplasia or cancer. For this reason, the FDA has issued a black box warning for patients and providers but recognizes that the benefits of tamoxifen therapy typically outweigh the risks for most patients with breast cancer.

Ovarian:

Adnexal masses are common in premenopausal women and may cause symptoms (eg, pain, pressure) or be found incidentally on examination. Most premenopausal adnexal masses are benign, but all require evaluation for malignancy. Initial evaluation is with **pelvic ultrasound**, which provides optimal visualization of adnexal mass size and features. Ultrasound findings can increase or decrease the clinical **suspicion for malignancy**. Patients with a low clinical suspicion for malignancy (eg, simple, fluid-filled ovarian cyst) are typically managed expectantly (eg, observation and repeat examination in 6 weeks).

In contrast, malignant adnexal masses are characterized by disordered, uncontrolled tissue proliferation. Therefore, they often have a **complex appearance** (ie, cystic and solid) with **abnormal internal features** such as **irregular, thickened septations** or papillary projections. Patients with these adnexal mass findings **require further evaluation** (eg, additional imaging, surgery).



Ovarian masses in postmenopausal patients are initially evaluated with a pelvic ultrasound. In postmenopausal women with a benign-appearing mass, a CA-125 level can further risk stratify the mass as either likely benign or malignant to further guide management (eg, observation, surgical exploration).

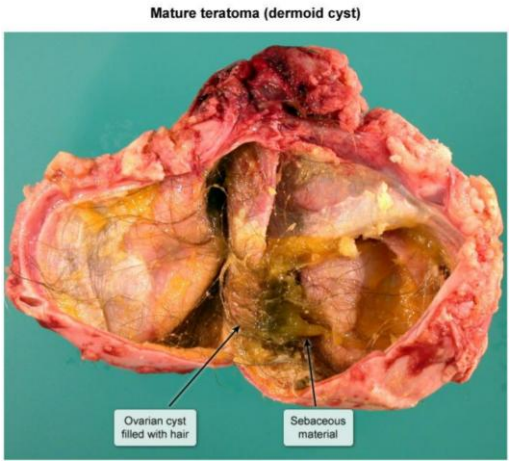
	Sertoli-Leydig cell tumor
Pathogenesis	Sex cord–stromal tumor ↑ Testosterone
Clinical features	Rapid-onset virilization ○ Voice deepening ○ Male-pattern balding ○ Increased muscle mass ○ Clitoromegaly Oligomenorrhea Unilateral, solid adnexal mass
Management	Surgery (tumor staging)

a **testosterone-secreting sex cord–stromal tumor**. Sertoli and Leydig cells are normally found in the testes but can develop in the ovaries and produce testosterone, particularly after malignant transformation and cell proliferation. The testosterone excess results in the clinical features often associated with this tumor, including:

- **Rapid-onset virilization**: Testosterone and dihydrotestosterone affect peripheral tissues, resulting in **clitoromegaly**, as seen in this patient. Additional features may include male-pattern (bitemporal) balding, voice deepening, and increased muscle mass.
- Signs of **estrogen deficiency**: Testosterone inhibits hypothalamic GnRH and pituitary FSH/LH release, resulting in low estrogen. Therefore, patients may develop breast atrophy, vulvovaginal atrophy, dyspareunia, and oligomenorrhea.

Sertoli-Leydig cells are typically diagnosed at an early cancer stage, and management includes surgical removal. Those with metastatic disease may require additional chemotherapy.

	Mature cystic teratoma
Pathology	Benign ovarian germ cell tumor Endoderm, mesoderm, ectoderm tissue
Clinical features	Most asymptomatic Ovarian torsion Struma ovarii subtype: hyperthyroidism Unilateral adnexal mass Ultrasound: complex, cystic, calcifications Gross appearance: sebaceous fluid, hair, teeth
Management	Ovarian cystectomy or oophorectomy



mature cystic teratoma (**dermoid cyst**), a benign ovarian germ cell tumor common in premenopausal women. Dermoid cyst contents include sebaceous fluid, hair, and teeth, creating ultrasound findings of **hyperechoic nodules and calcifications**. Although many dermoid cysts are asymptomatic, the heterogeneous solid and fluid components of varying density create an inherently unbalanced tumor that predisposes some patients to develop partial adnexal rotation (ie, intermittent torsion). This torsion results in an **intermittent, colicky pelvic pain**, often triggered by physical activity (eg, intercourse, exercise) impeding ovarian blood flow.

The pain resolves spontaneously once the adnexa untwists and blood flow returns. However, some patients develop persistent ovarian torsion, which is a gynecologic surgical emergency because persistent torsion results in ischemia and eventual necrosis of the ovary. Therefore, dermoid cysts are surgically removed with either a **laparoscopic ovarian cystectomy** or oophorectomy to **reduce the risk of ovarian torsion** and prevent complications (eg, infertility, hemorrhage, sepsis) associated with ovarian necrosis.

So in short if the patient has teratoma it predisposes her to have torsion so you remove it to prevent the torsion.

	Granulosa cell tumor
Pathogenesis	Sex cord–stromal tumor ↑ Estradiol ↑ Inhibin
Clinical features	Complex ovarian mass Juvenile subtype ○ Precocious puberty Adult subtype ○ Breast tenderness ○ Abnormal uterine bleeding ○ Postmenopausal bleeding
Histopathology	Call-Exner bodies (cells in rosette pattern)
Management	Endometrial biopsy (endometrial cancer) Surgery (tumor staging)

granulosa cell tumor, an ovarian **sex cord–stromal tumor**. The ovarian stroma, which is primarily composed of granulosa cells, produces the ovarian estrogen supply. Granulosa cells convert testosterone to estradiol (via aromatase) and secrete inhibin (which typically inhibits FSH); therefore, uncontrolled proliferation of these cells, as seen in a granulosa cell tumor, results in high estradiol and inhibin levels.

Women with an adult subtype granulosa cell tumor have **chronic, unopposed estrogen** exposure that can result in **endometrial hyperplasia** or cancer (eg, **postmenopausal bleeding**). Additional clinical features may include breast tenderness and mass-effect symptoms (eg, abdominal distension, ovarian torsion). Due to the association of granulosa cell tumors with endometrial cancer, patients with a suspected granulosa cell tumor require an endometrial biopsy prior to surgical staging to help guide management. After surgical management, inhibin levels can be monitored to evaluate for disease progression or recurrence.

	Epithelial ovarian carcinoma
Clinical presentation	Asymptomatic: incidental adnexal mass Subacute: pelvic/abdominal pain, bloating, early satiety Acute: dyspnea, obstipation/constipation, abdominal distension
Risk factors	Family history Genetic mutations (*BRCA1*, *BRCA2*) Age ≥50 Endometriosis Infertility Early menarche/late menopause
Protective factors	Oral contraceptive pills Multiparity Breastfeeding
Laboratory findings	↑CA-125
Ultrasound findings	Solid, complex mass Thick septations Ascites
Management	Surgical exploration ± Chemotherapy

Epithelial ovarian cancer (EOC) occurs primarily in postmenopausal women and most often presents with advanced disease because the earliest symptoms of disease are vague and nonspecific (eg, constipation, bloating).

Initial evaluation of postmenopausal women with suspected EOC is with pelvic imaging (eg, ultrasound), which identifies mass characteristics (eg, thick septations, increased vascularity), and CA-125, which is released by cells from the peritoneum, uterus, and fallopian tubes, all of which are in close proximity to the rapidly growing ovary (ie, malignancy). In postmenopausal women with a malignant-appearing mass, CA-125 aids in disease monitoring and response to treatment (ie, chemotherapy). In contrast, in postmenopausal women with a benign-appearing mass, CA-125 stratifies the risk for cancer.

Additional imaging (eg, CT scan) is performed in patients with suspected EOC to evaluate for distant metastases and for surgical planning. In the absence of distant metastases, suspected EOC is treated with **exploratory laparotomy** and **surgical staging and tumor debulking**.

	Choriocarcinoma
Risk factors	Advanced maternal age Prior complete hydatidiform mole
Presentation	Amenorrhea or abnormal uterine bleeding Pelvic pain/pressure Symptoms from metastases (lung, vagina) Uterine mass Elevated β-hCG level
Treatment	Chemotherapy

Choriocarcinoma is a form of gestational trophoblastic neoplasia, a malignancy that arises from placental trophoblastic tissue and secretes β-hCG. Although it most commonly follows a hydatidiform mole, choriocarcinoma can occur after a normal gestation or spontaneous abortion. Choriocarcinoma typically presents <6 months after a pregnancy. Presenting symptoms include **irregular vaginal bleeding**, an **enlarged uterus**, and pelvic pain. Choriocarcinoma is an aggressive type of gestational trophoblastic neoplasia; the most common site of metastatic spread is to the lungs. Symptoms of **pulmonary metastasis** include **chest pain**, hemoptysis, and **dyspnea**. When choriocarcinoma is suspected, obtaining a **quantitative β-hCG** level helps to confirm the diagnosis.

	Contraindications to IUD placement
Copper IUD & progestin IUD	Pregnancy Endometrial or cervical cancer Unexplained vaginal bleeding Gestational trophoblastic disease Severe uterine cavity distortion Active pelvic infection (eg, PID, cervicitis)
Progestin IUD	Active liver disease Current breast cancer
Copper IUD	Wilson disease

Abnormal bleeding is often a symptom of an **underlying condition** that requires further evaluation before IUD insertion, such as:

- Infection (eg, cervicitis, pelvic inflammatory disease)
- Endometrial polyp, which often causes intermenstrual bleeding
- **Endometrial hyperplasia or cancer**, particularly in patients with multiple risk factors for unopposed estrogen exposure (eg, obesity, chronic anovulation [as evidenced by this patient's irregular menses])

IUD placement in patients with unexplained, abnormal vaginal bleeding can mask symptoms and delay diagnosis. Therefore, these patients **require further evaluation** prior to IUD insertion, which typically includes sexually transmitted infection testing, endometrial biopsy, and pelvic ultrasound.

Absolute contraindications to combined hormonal contraceptives
Migraine with aura
≥15 cigarettes/day PLUS age ≥35
Hypertension ≥160/100 mm Hg
Heart disease
Diabetes mellitus with end-organ damage
History of thromboembolic disease
Antiphospholipid-antibody syndrome
History of stroke
Breast cancer
Cirrhosis & liver cancer
Major surgery with prolonged immobilization
Use <3 weeks postpartum

Hormone-containing methods of contraception are avoided in patients with breast cancer, as estrogen and progesterone may have a proliferative effect on breast tissue. This is particularly concerning with hormonal receptor-positive breast cancer, and ***BRCA2*** carriers tend to have **estrogen receptor-positive** breast cancer.

A copper intrauterine device (IUD) is a safe, long-term, **hormone-free** method of **contraception**. It is **99% effective** and prevents pregnancy by creating a chronic cytotoxic inflammatory response. A copper IUD can be placed for a maximum of 10 years.

The progesterone IUD is a highly effective contraceptive and can be placed for 5 years. It is also contraindicated in the setting of current breast cancer due to potential systemic absorption of progesterone.

Miscellaneous:

	Staphylococcal toxic shock syndrome
Risks	Tampon use Nasal packing Surgical/postpartum wound infection
Pathogenesis	*Staphylococcus aureus* Exotoxin release acting as superantigens
Clinical features	Fever >38.9 C (102 F) Hypotension Diffuse macular rash involving palms & soles Desquamation 1-3 weeks after disease onset Vomiting, diarrhea Altered mentation without focal neurologic signs
Treatment	Supportive therapy (fluid replacement) Removal of foreign body (eg, tampon) Antibiotic therapy (eg, vancomycin, cefepime, clindamycin)

toxic shock syndrome (TSS), which typically presents with high fever, **hypotension**, tachycardia, and a diffuse, red, **macular rash** involving the **palms and soles**. Other clinical manifestations of TSS may include headache, vomiting, profuse diarrhea, and mucous membrane hyperemia or ulceration. Desquamation of the palms and soles occurs 1-3 weeks after disease onset.

Menstrual cases of TSS are caused by ***Staphylococcus aureus*** infection related to **prolonged or continuous tampon use,but approximately 50% of cases occur due to an underlying surgical or postpartum wound infection.** Disease occurs due to the release of toxic shock syndrome toxin-1, an exotoxin that acts as a superantigen and causes an exaggerated immune response leading to shock (eg, hypotension, tachycardia) and multiorgan failure (eg, decreased urine output, altered mentation, bilateral crackles). The diagnosis is made clinically.

Patients require aggressive fluids (and sometimes vasopressors), removal of the retained foreign body (if present), surgical debridement (in the setting of wound infection), and treatment with empiric antibiotics, including:

- **Vancomycin**, which provides bactericidal activity against methicillin-resistant *Staphylococcus aureus* (MRSA)

- **Clindamycin**, which prevents production of bacterial exotoxins by blocking the bacterial ribosome
- A penicillin/beta-lactamase inhibitor (eg, piperacillin-tazobactam), **cefepime**, or a carbapenem, which provides additional bactericidal activity

	Acute hemolytic transfusion reaction
Pathogenesis	ABO incompatibility Intravascular hemolysis
Clinical findings	Onset within minutes to 24 hr of transfusion Fever, chills, hypotension Hemoglobinuria, flank pain
Laboratory findings	Positive direct Coombs test Hemolysis (eg, ↑ LDH, ↑ indirect bilirubin)
Complications	Acute kidney failure Disseminated intravascular coagulation

acute hemolytic transfusion reaction (AHTR), a rare but potentially fatal reaction due to transfusion of mismatched blood (eg, **ABO incompatibility**). The most common etiology is clerical error (eg, patient misidentification).

AHTR typically occurs **within minutes to hours** of transfusion due to host antibodies attacking donor blood antigens. The result is massive red blood cell destruction (ie, **intravascular hemolysis**), which leads to toxic hemoglobin buildup in the kidney, renal tubular cell injury, and subsequent **acute renal failure**. Therefore, findings that raise suspicion for AHTR include **flank pain** and dark red urine (ie, **hemoglobinuria**) as well as **fever**, tachycardia, and hypotension (due to inflammatory cytokine release) shortly following a transfusion. Patients may also have elevated lactate dehydrogenase levels (from hemolysis) and **disseminated intravascular coagulation** (eg, oozing intravenous site).

Diagnosis of AHTR is with a positive **direct Coombs test**, reflecting antibody-coated red blood cells. Management includes immediate cessation of transfusion, aggressive intravenous fluid administration, and supportive care.

	Recurrent cystitis in women
Definition	≥2 infections in 6 months ≥3 infections in a year
Risk factors	Sexually active Postmenopausal 1st UTI at age <15 Spermicide use
Prevention	Daily antibiotic prophylaxis Postcoital prophylaxis

Women are at increased risk for cystitis, a lower **urinary tract infection**, due to the proximity of the rectum to the vagina and urethra. A significant **risk factor** is **sexual intercourse**, as in this patient. During intercourse, enteric bacteria (most commonly *Escherichia coli*) are introduced into the vagina and periurethral area. Because the female urethra is short, these bacteria can readily ascend to the bladder and cause cystitis and associated symptoms (eg, dysuria, urinary frequency). Other risk factors include conditions that alter the normal urogenital flora (eg, recent antibiotic use, spermicide use).

This patient has **recurrent cystitis**, defined as **≥2 episodes in 6 months** (or ≥3 episodes in a year). Patients with recurrent cystitis are at increased risk for repeat episodes. Prevention is first focused on behavior modifications, which include increasing daily fluid intake, changing contraception method (eg, stop using spermicide or diaphragm), and voiding after sexual intercourse (unproven benefit but possibly helpful). If these measures fail, low-dose antibiotic prophylaxis can be considered, although this has inherent risks related to continuous antibiotic exposure. Therefore, in women with cystitis episodes clearly linked to intercourse, **postcoital antibiotics** (eg, nitrofurantoin, trimethoprim-sulfamethoxazole) are preferred because they can prevent recurrence while reducing the risks associated with long-term antibiotic use (eg, antibiotic resistance, toxicity).

	Recurrent urinary tract infection
Definition	≥2 infections in 6 months ≥3 infections in 1 year
Risk factors	History of cystitis at age ≤15 Spermicide use New sexual partner Postmenopausal status
Evaluation	Urinalysis Urine culture
Prevention	Behavior modification Postcoital or daily antibiotic prophylaxis Topical vaginal estrogen for postmenopausal patients

Women are at increased risk for cystitis due to the proximity of the urethra to the vagina and rectum. In **postmenopausal women**, the risk increases further due to **estrogen deficiency**, which causes:

- vulvovaginal atrophy (eg, thin vulvar tissue).
- decreased bulk and elasticity of the bladder trigone and urethra, resulting in an increased risk for ascending infection.
- decreased vaginal lactobacilli levels and an **elevated vaginal pH**, leading to an increased rate of vaginal *Escherichia coli* colonization.

Therefore, in addition to behavioral changes (eg, increased fluid intake), treatment of postmenopausal women with recurrent UTI is with topical **vaginal estrogen**. Topical estrogen can improve genitourinary atrophy and prevent future UTI episodes. In addition to vaginal estrogen, antibiotic prophylaxis (daily or postcoital) can be considered.

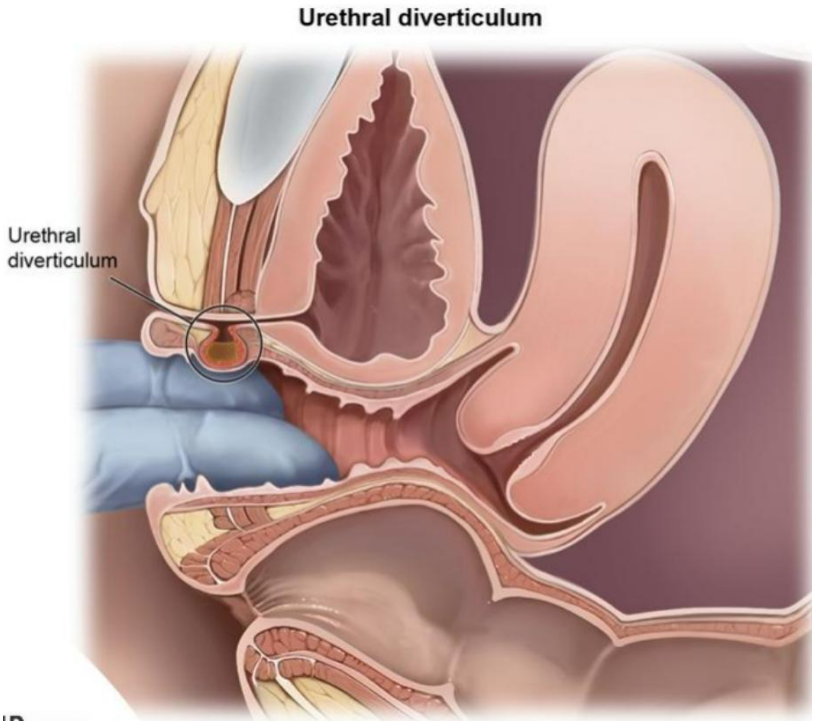
	Interstitial cystitis (bladder pain syndrome)
Epidemiology	More common in women Associated with psychiatric & pain disorders (eg, fibromyalgia)
Clinical presentation	Bladder pain with filling, relief with voiding ↑ Urinary frequency, urgency Dyspareunia
Diagnosis	Bladder pain with no other cause for ≥6 weeks Normal urinalysis
Treatment	Not curative, focus is on improving quality of life Behavioral modification, avoidance of triggers, physical therapy Amitriptyline, pentosan polysulfate sodium Analgesics for acute exacerbations

Interstitial cystitis (IC) (also known as painful bladder syndrome) is a chronic, painful bladder condition of uncertain etiology. Patients typically have pain that is **exacerbated by bladder filling** and **relieved by voiding**. Symptom onset is typically gradual and worsens over a period of months. Other clinical features include **urinary frequency** and urgency, chronic pelvic pain, and dyspareunia. IC typically presents in women age >40 and is associated with other chronic pain conditions (eg, fibromyalgia, endometriosis, irritable bowel syndrome), sexual dysfunction, and psychiatric illness (eg, depression, anxiety).

The diagnosis of IC is largely clinical; however, additional laboratory testing—including a urinalysis, postvoid residual, and sexually transmitted infection screening—is performed to exclude other conditions (eg, cystitis, urinary obstruction, malignancy). Management includes bladder training, fluid management, analgesics, and avoidance of any precipitating agents (eg, caffeine, alcohol, artificial sweeteners).

Interstitial cystitis is often treated with amitriptyline, and those with worsening symptoms may require an increased dose.

If they we doing good in amitriptyline and suddenly get dysuria think of urethritis or cystitis and not a worsening of the disease.



urethral diverticulum, an abnormal localized outpouching of the urethral mucosa into surrounding tissues. A urethral diverticulum typically arises from recurrent periurethral gland infections, which can develop into an abscess that can eventually breach the urethral mucosa. The persistent infection, inflammation, and increased tissue tension in the area causes a **tender anterior vaginal wall mass** that may present as **dyspareunia** or a palpable mass on pelvic examination. In addition, the diverticulum may collect urine and debris, resulting in a **purulent discharge, dysuria, or postvoid dribbling**.

The recurrent infection and inflammation of the urethral tissue creates the abnormal outpouching that can collect and store urine, resulting in **postvoid dribbling** and recurrent lower urinary tract infections (eg, dysuria). Infection of the diverticulum can also lead to pain, often presenting as dyspareunia or a **tender anterior vaginal wall mass** with an associated expressed purulent or **bloody urethral discharge**.

MRI is used to confirm the diagnosis of a urethral diverticulum; patients are treated via surgical excision.

DDx:

Symptomatic pelvic organ prolapse can cause pelvic pressure and an anterior vaginal bulge (eg, cystocele); this condition is common in multiparous women, particularly those who have had vaginal deliveries and fetal macrosomia. However, pelvic organ prolapse typically causes a nontender vaginal bulge and is not associated with purulent discharge.

	Genitopelvic pain/penetration disorder
Risk factors	Sexual trauma Lack of sexual knowledge History of abuse
Clinical features	Pain with vaginal penetration Distress/anxiety over symptoms No other medical cause
Treatment	Desensitization therapy Kegel exercises

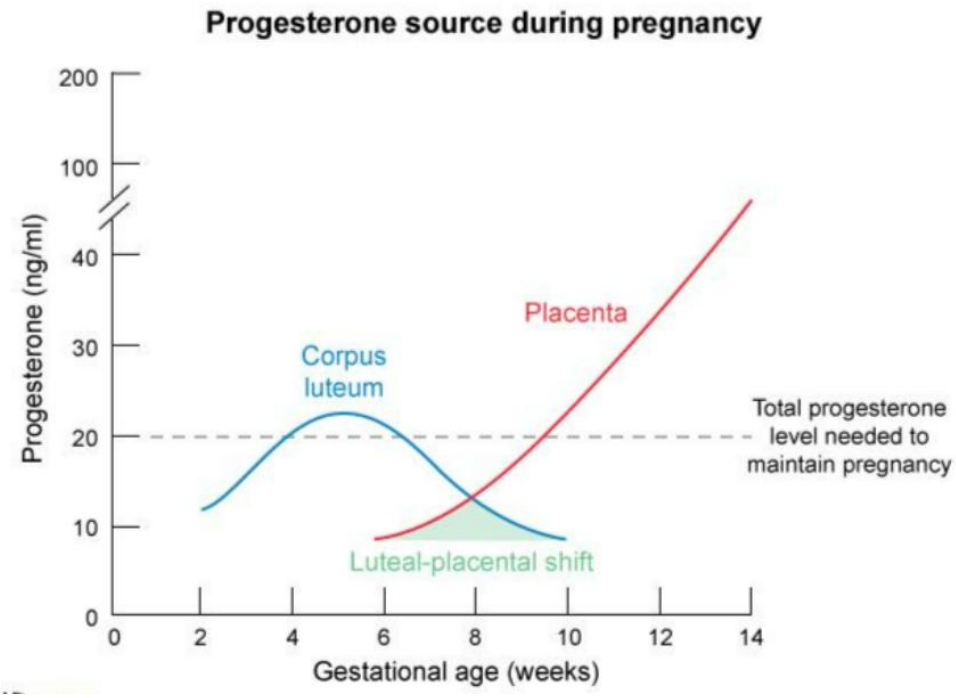
genito-pelvic pain/penetration disorder, previously known as vaginismus, characterized by pain on and an aversion to attempted vaginal penetration. Genito-pelvic pain/penetration disorder may be limited to primarily a sexual context or include pain with tampon insertion and gynecologic examinations. This condition causes psychological distress and may have an identifiable cause, such as a history of vaginal trauma. The condition is underreported due to significant patient anxiety and avoidance of examinations. Examination differentiates it from other pathologies (eg, infectious vaginitis, vulvodynia) but may be limited as patients often cannot tolerate speculum insertion. Patients have no tenderness on external examination. Treatment is aimed at relaxing the vaginal muscles and includes desensitization therapy and Kegel exercises.

Localized provoked vulvodynia, formerly termed vestibulodynia, is pain to superficial touch on the vestibule. Typically, there is an area of tenderness to touch on external examination.

Pudendal neuralgia presents as superficial pain located at the vulva, perineum, and rectum (eg, pudendal nerve distribution).

Sjögren syndrome	
Exocrine features	**Keratoconjunctivitis sicca** Dry mouth, salivary hypertrophy Xerosis
Extraglandular features	**Raynaud phenomenon** Cutaneous vasculitis Arthralgia/arthritis Interstitial lung disease Non-Hodgkin lymphoma
Diagnostic findings	Objective signs of **decreased lacrimation** (eg, Schirmer test) Positive **anti-Ro** (SSA) &/or **anti-La** (SSB) Salivary gland biopsy with focal lymphocytic sialoadenitis Classification: primary if no associated CTD, secondary if comorbid CTD (eg, SLE, RA, scleroderma)

Sjogren can cause dyspareunia due to decreased vaginal secretions.

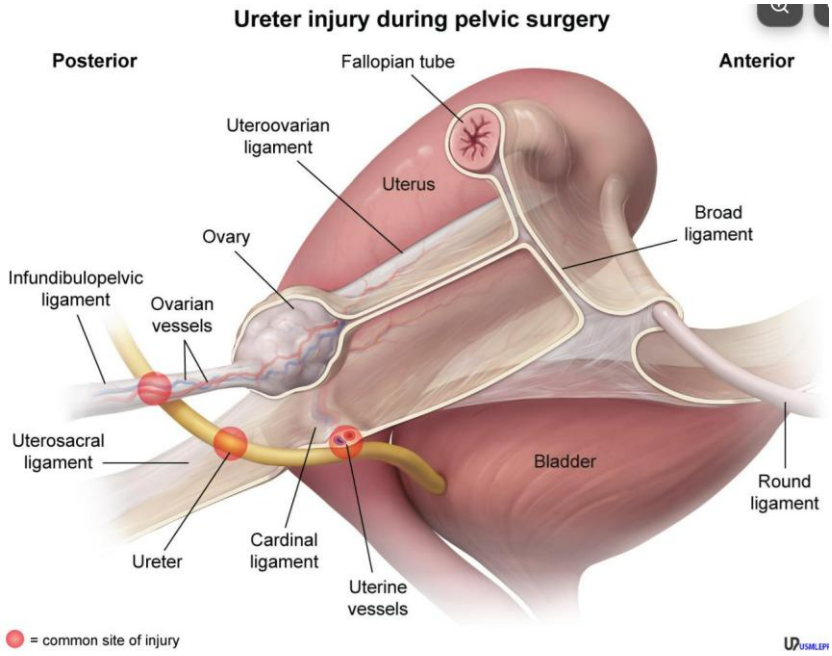


Ovulation occurs when a secondary oocyte is released from the dominant ovarian follicle, leaving behind cells that form an ovarian structure called the **corpus luteum** (CL). Following fertilization, the CL enlarges and produces high levels of **progesterone**, which thickens the endometrial lining in preparation for implantation. Progesterone also promotes implantation of the embryo and **maintains the pregnancy**. In early pregnancy, the CL is the primary source of progesterone until the placenta takes over production at **8-10 weeks gestation** (ie, the luteal-placental shift).

If the CL is removed prior to 10 weeks gestation (as in this patient who required an emergency oophorectomy due to ovarian torsion), progesterone levels decrease precipitously; as a result, the pregnancy becomes at high risk for spontaneous abortion. Therefore, patients with **corpus luteum removal** prior to 10 weeks gestation require postoperative **progesterone supplementation** (eg, vaginal progesterone) to **prevent pregnancy loss**. Supplementation can be discontinued after 10 weeks gestation.

The case was a women who did oophorectomy for ovarian torsion and the ovary was found to have the CL

What hormone should we supply this patient?



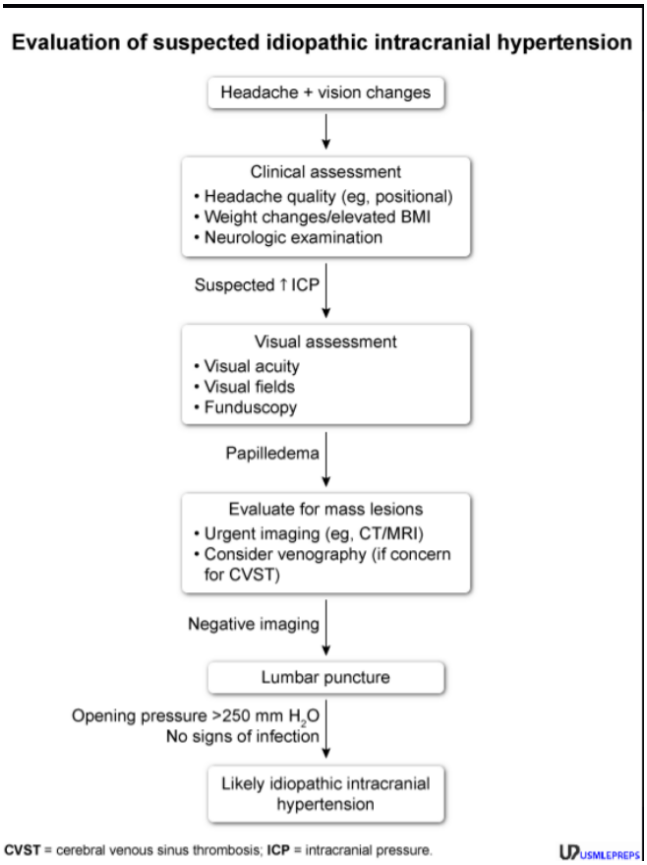
uroperitoneum (ie, urine within the peritoneal cavity). In patients who have undergone **gynecologic surgery** (eg, hysterectomy), particularly those with distorted pelvic anatomy (eg, endometriosis, prior surgery), the most likely cause is from a **unilateral ureteral laceration**. The ureter is vulnerable to injury during gynecologic procedures due to its proximity to the ovarian vessels (in the infundibulopelvic/suspensory ligament) and uterine vessels (near the cervix).

Most ureteral injuries are identified during surgery but missed cases can present up to 2 weeks postoperatively as the damaged ureter drains urine directly into the abdomen, resulting in a **large volume of intraabdominal fluid** and subsequent abdominal distension (eg, diffuse pain, bloating). As the urine continuously fills the abdomen, it can overflow through the vagina (which is sutured but not fully healed) and cause a **watery vaginal discharge**. The caustic effects of the urine may cause signs of peritoneal inflammation (eg, **fever**, nausea, abdominal pain). Patients with a unilateral ureteral injury often have regular voiding and **normal serum creatinine and urinalysis** because the contralateral kidney and ureter continue to function normally. Diagnosis is typically with CT urography and treatment is surgical repair.

Also one ureter might be sutured by accident which will present with hydronephrosis late -after a week- but if its unilateral the labs will be normal.

The patient will have flank pain -progressive- sometimes the patient will say back pain.

And costovertebral angle tenderness.// Dx is made by renal ultrasound.



IIH is most common in **obese** (BMI ≥30 kg/m^2) **women of childbearing age**, and although pregnancy by itself is not a risk factor, excessive weight gain (as in this patient) can exacerbate the disorder.

Patients typically have **positional headaches** that are worse when lying flat, which increases ICP, and improve with sitting, which decreases ICP. **Pulsatile tinnitus** (due to increased vascular pulsations) and blurry vision (due to increased pressure on the optic nerves) also commonly occur. On physical examination, increased ICP can manifest as optic disc edema (ie, **papilledema**), abducens nerve (CN VI) palsy (lateral rectus palsy), and/or visual field deficits.

Diagnosis is with neuroimaging followed by lumbar puncture, which reveals an elevated opening pressure (>250 mm H_2O). **MRI of the brain**, often with MR venography to rule out cerebral vein thrombosis, is the preferred imaging modality and avoids fetal radiation exposure. MRI is performed **before lumbar puncture** to exclude other causes of elevated ICP (eg, space-occupying mass) that would increase the risk of cerebral herniation.