





Pelvic Inflammatory Disease & Pelvic Abscess

Objectives:

- → Identify the prevalence of Pelvic Inflammatory Disease (PID)
- → Explain the causes and pathogenesis of PID
- → Describe the symptoms and signs of PID.
- → Describe the management of PID and list the criteria for hospitalization and parental treatment
- → List the complications of PID
- → Discuss the tubo-ovarian abscess in terms of: Incidence, Etiology, Diagnosis, Management, Sequelae.

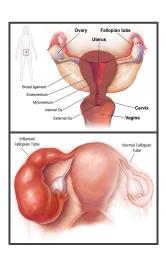


- → Department's Slides (Dr. Dana)
- → Important
- → Golden notes
- → Extra
- → Doctor's notes
- → Previous Doctor's notes
- → Reference



Definition:

- → Pelvic Inflammatory Disease (PID): a spectrum of infection-induced inflammation of upper genital tract that includes endometritis, salpingitis, pelvic peritonitis, and/or tubo-ovarian abscess (TOA).
- → **PID:** microorganisms colonizing the endocervix and ascending to the endometrium and fallopian tubes cause PID.
- \rightarrow Inflamed fallopian tube (salpingitis) \rightarrow tube obstruction \rightarrow TOA formation.
- → Most often by ascending spread of microorganisms from the vagina through cervix & endocervix to the endometrium, tubes, and contiguous structures (when barrier between the lower genital tract and upper genital tract in broken).
- → **Picture:** demonstrates normal genital tract on the right, and on the left a collection of inflammatory exudates.



Prevalence:

- \rightarrow **CDC:** \approx > 1 million women in USA experience an episode of PID every year.
- \rightarrow PID $\rightarrow \approx 2.5$ million office visits + 125,000 150,000 hospitalizations yearly.
- → No specific international data is available for PID incidents worldwide.
- → **Annual rate of PID in high-income countries:** 10 20 per 1000 women of reproductive age.

Risk Factors:

- → **Exposure to STD:** strong correlation.
- \rightarrow Age of 1st intercourse.
- → Frequency of intercourse.
- → Number of sexual partners.
- → Reinfection if untreated male partner (80%): males can be carriers of many STDs but females present dramatically.
- \rightarrow Lactobacilli \rightarrow acidic pH of vagina \rightarrow protection against pathogens.
 - \rightarrow Antibiotics, vaginal douching \rightarrow pH gets disturbed \rightarrow organisms enter & infect the genital tract.
- → Marital status.
- → **Nulliparous:** never conceived before, 33%.

→ ↑ risk:

- → IUD user (multifilament string): organism colonizes string → acts as a mechanical helper for it.
 - → IUD already present when infection is obtained → no need to remove it.
 - → Highly suspected PID patient desires an IUD → must prohibit.
- → Surgical procedure.
- → Previous acute PID.

→ ↓ risk:

- → **Barrier method:** if partner is infected.
- → OCP: estrogen encourages lactobacilli proliferation.
- → Pregnancy: ↑ progesterone ↑ cervical mucus.

Causes:

- → **Sexually transmitted microorganisms:** N. gonorrhoeae C. trachomatis.
 - \rightarrow Sexually active female of reproductive age (85% of infection).
 - → Bacteria culture direct from tubal fluid.
 - \rightarrow STDs are the main cause of PID, but PID is not always caused by STDs.
 - → Most common organisms causing STD & a serious complications: gonorrhea chlamydia.
- → After procedures that break mucus barrier (15% of infection).
 - → **iatrogenic:** → before any procedure, make sure the patient isn't having any purulent discharge + instruments are sterile.
 - → Endometrial biopsy.
 - \rightarrow D&C.
 - → Hysterosalpingogram.

Causative Organisms:

	Neisseria gonorrhoeae (N. gonorrhoeae)	C. Trachomatis
Prevalence	Most common	Second most common
Bacteria	Gram <mark>negative</mark> diplococcus	Intracellular organism
Growth	Rapid: 20 - 40 mins.	Slow: 48 - 72 hours (goes unnoticed).
Onset	 → Rapid & intense inflammatory response (acute infection). → Severe symptoms. 	→ Insidious→ Asymptomatic.
Sequelae	 → Infertility → Ectopic pregnancy Strongly associated with prior chlamydia infection 	 → Mild form of salpingitis. → Remains (preference) in tubes for months/years after initial colonization silent & asymptomatic → more severe tubal involvement (destroying cilia's transitional epithelium → damaged tubes = old infection).

Mycoplasma genitalium (genital mycoplasma spp.):

- → Common
- → Present with mild clinical symptoms similar to chlamydial PID.

→ Endogenous aerobic & anaerobic:

- → Common
- → Endogenous microorganisms found in the vagina.
- → Often isolated from upper genital tract of women with PID.
- → BV microorganisms: anaerobic bacteria (such as: prevotella – peptostreptococci - G. vaginalis).

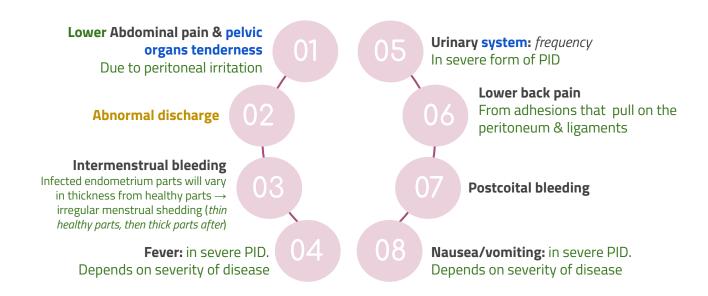
→ **Polymicrobial flora**: *vaginal normal flora*

- \rightarrow Prevatella sp.
- → Peptostreptococcus sp.
- → Escherichia
- → Anaerobic gram-negative rods.

→ Respiratory pathogens:

- → Colonize the lower genital tract and cause PID
- → Haemophilus influenzae.
- → Group A streptococci.
- → Pneumococci.

Signs & Symptoms:



Approach to patient with PID:

- **1.** History.
- **2.** Examination.
 - → Vitals signs
 - → Abdominal Examination
 - → Pelvic exam and bimanual exam.
 - → High vaginal swab
- 3. Labs: CBC ESR CRP.
- Imaging.

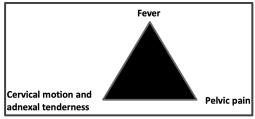
Diagnosis:

- → Diagnosis is made clinically (symptoms & signs), there is no single test to confirm if the Pt has PID)
- → Clinical diagnosis implying that patient has upper genital tract infection and inflammation.
 - → Inflammation may be at any point along a continuum: endometritis salpingitis peritonitis.
- \rightarrow Wide variation in many symptoms and signs among women with PID \rightarrow difficult acute PID diagnosis.
 - → Subtle or mild symptoms that are not readily recognizable as PID.

Diagnosis:

Physical Examination:

- → Temperature and vital signs.
- → Diagnosis of PID -triad of signs & symptoms -: pelvic pain + cervical motion & adnexal/uterine tenderness + fever.
 - → Cervical motion tenderness: peritoneal inflammation + moving cervix \rightarrow adnexa traction on peritoneum \rightarrow stretched perineum \rightarrow irritated peritoneum \rightarrow pain.



- → Direct or rebound abdominal tenderness.
- → **Genitourinary symptoms** (including but not limited to): lower abdominal pain excessive vaginal discharge - heavy & irregular vaginal bleeding - fever - chills - urinary symptoms.
- → **Assess the abdomen for tenderness:** usually over lower abdomen, but if the infection ascends through the peritoneum, the tenderness will be generalized over the abdomen.
- \rightarrow Vaginal & endocervical secretion examination \rightarrow assess BV presence (important workup¹ part).
- \rightarrow Microscopy of vaginal secretion \rightarrow presence of leukocytes, clue cells, and trichomonads (take a swab).
- \rightarrow Cervical canal examination \rightarrow presence of yellow/green mucopus and friability.
- → Testing for C. trachomatis and N. gonorrhoeae.
- \rightarrow Bimanual pelvic examination \rightarrow assess pelvic organ tenderness + pelvic mass (*might suggest a TOA*).
 - → PID patients have positive cervical motion tenderness², but it's not specific for PID & occurs in any peritoneal irritation (appendicitis - pelvic bleeding due to corpus luteum rupture).

Lab Tests:

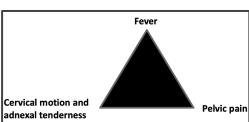
- → Complete blood count (CBC):
 - \rightarrow Hb \rightarrow decide if there's bleeding or not.
 - \rightarrow WBC differential \rightarrow infection type identification + look for leukocytosis.
- → Erythrocyte sedimentation rate (ESR).
- \rightarrow C-reactive protein test (CRP).

Imaging Studies:

- → Pelvic ultrasonography:
 - → Rule out symptomatic ovarian cysts.
 - → Patients with pelvic mass noted on bimanual pelvic examination.
- → Computed tomography:
 - → Rule out appendicitis & endometriosis in case of abdominal tenderness.
 - → See if there is any collection like ovarian abscess.

Laparoscopic Visualization:

- → Most accurate method to confirm PID, but NOT the initial approach as it's invasive.
- → All patients with uncertain diagnosis (such as suspected abscess).
- → All patients not responding to treatment within 48-72h of antibiotics: big TOA or hydrosalpinx are unlikely to respond to antibiotics & need to be drained.
- → Negative gram smear does not rule out PID: many PID patients have negative cervical swab & culture, but you still have to treat for both organisms.
- → Not a routine test to confirm PID.
- 1. In women with PID, an increased number of polymorphonuclear leukocytes may be detected in a wet mount of the vaginal secretions or the cervix may have a mucopurulent discharge.
- 2. Suggests the presence of peritoneal inflammation.



Management:

- → **Therapeutic goal:** eliminate acute infection & symptoms + prevent long term sequelae.
- → Start treatment as soon as you suspect PID.
- → Always start with empirical treatment!
- → Therapeutic PID regimens must provide empirical, broad-spectrum coverage of likely pathogens:
 - → N. gonorrhoeae
 - → C. trachomatis
 - → M. genitalium
 - → Gram-negative facultative bacteria
 - → Anaerobes
 - → Streptococci
- → **Outpatient regimen:** cefoxitin and doxycycline (as effective as an inpatient parenteral regimen of the same antimicrobials).

Mild to Moderate PID	Severe PID & TOA
 → Treat as outpatient. → Aim: microbiological cure of N. gonorrhoeae + C. trachomatis (even if negative endocervical screening for these organisms). → Coverage for polymicrobial flora associated with BV (bacterial vaginosis). → Antibiotic therapy. 	 → Hospitalization (inpatient). → Parenteral therapy (criteria noted). → Imaging: like US, specifically if they don't respond to treatment + to rule out tubo-ovarian abscess. → Surgical intervention: if failed antibiotic therapy: → TOA with abscesses diameter ≥ 10 cm. → Failed antibiotic treatment within 48 - 72 hours (persistent fever - ↑ leukocytosis) which may indicate an infection. → Drainage of TOA: laparotomy (sick / bleeding patient → faster → do it) + laparoscopy + image-guided percutaneous routes.

Clinical Criteria for Hospitalization¹ & Parenteral Treatment:

- → First rule out other causes like appendicitis or ectopic pregnancy.
- → Surgical emergencies (e.g. appendicitis) can't be excluded or not ruled out.
- → Pregnancy & you can't rule out ectopic pregnancy at time of acute abdomen.
- → **Does not respond clinically to oral antibiotic therapy (failed):** no improvement with short-term treatment.
- → **Compliance questionable:** unable to follow/tolerate an outpatient oral regimen.
- → **Severe illness (toxicity):** nausea vomiting high fever.
- → **Tubo-ovarian abscess:** on ultrasonography or clinically suspected.

Discharging a Hospitalized Patients Conditions:

- → Fever < 99.5° F for more than 24 hours.
- → Decreasing WBC count.
- → Absent rebound tenderness.
- → Marked amelioration of abdominal tenderness in repeated examination.

Management:

CDC Recommended Oral Regimen:

- → 3 regimens, we choose based on most common organism we know in the center.
- → **Most important coverage:** gram -ves & anaerobes.
- → Doxycycline & metronidazole are a must and present in each regimen while other medications depend on the center.
- → Just know the antibiotics that covers both gonorrhea & Chlamydia
- → **Most common:** 1st regiment in the table.

2015 CENTERS FOR DISEASE CONTROL (CDC) RECOMMENDED FIRST-LINE REGIMEN FOR OUTPATIENT TREATMENT OF PELVIC INFLAMMATORY DISEASE Recommended Regimen Ceftriaxone 250 mg intramuscularly in a single dose Doxycycline 100 mg orally twice a day for 14 days WITH or WITHOUT Metronidazole 500 mg orally twice a day for 14 days **Cefoxitin** 2 g intramuscularly in a single dose and probenecid, 1 g orally administered concurrently in a single dose Doxycycline 100 mg orally twice a day for 14 days WITH or WITHOUT Metronidazole 500 mg orally twice a day for 14 days Other parenteral third-generation cephalosporin (e.g., ceftizoxime or cefotaxime) Doxycycline 100 mg orally twice a day for 14 days WITH or WITHOUT Metronidazole 500 mg orally twice a day for 14 days

TABLE 22-6

CENTERS FOR DISEASE CONTROL (CDC) RECOMMENDED FIRST-LINE REGIMEN FOR PARENTERAL TREATMENT OF PELVIC INFLAMMATORY DISEASE

Recommended Parenteral Regimen A

Cefotetan 2 g IV every 12 hours

OR

Cefoxitin 2 g IV every 6 hours

PLUS

Doxycycline 100 mg orally or IV every 12 hours

Recommended Parenteral Regimen B

Clindamycin 900 mg IV every 8 hours

PLUS

Gentamicin loading dose IV or IM (2 mg/kg of body weight), followed by a maintenance dose (1.5 mg/kg) every 8 hours. Single daily dosing (3 to 5 mg/kg) can be substituted.

Alternative Parenteral Regimens

Ampicillin/sulbactam 3 g IV every 6 hours

PLUS

Doxycycline 100 mg orally or IV every 12 hours

From Pelvic Inflammatory Disease: Sexually Transmitted Diseases Treatment Guidelines, 2015. Available at http://www.cdc.gov/std/treatment/2015/pic htm. Accessed February 19, 2015. IM, Intramuscularly; IV, intravenously.

Sexual Partner Management:

From Pelvic Inflammatory Disease: Sexually Transmitted Diseases Treatment Guidelines, 2015. Available at http://www.cdc.gov/std/treatment/2015/ pid.htm. Accessed February 19, 2015.

- → Sexual partners of PID women must be evaluated & treated for urethral infection of chlamydia or gonorrhoea.
- → Treat male partners & educate them for prevention reinfection.
- → Regimens for uncomplicated gonorrhea & chlamydia infection:
 - → Ceftriaxone 125 mg IM.

Followed by one of the following:

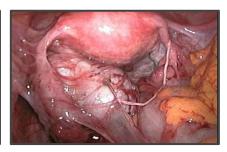
- → Doxycycline 100 mg twice daily pc (post cibum = after meals) for 7 days.
- → Azithromycin 1gm.
- → Ofloxacin 300 mg twice daily pc (post cibum = after meals) for 7 days.

Complications:

- → Chronic pelvic pain: 25%, due to pelvic scars & adhesions.
- → Infertility.
- → Ectopic pregnancy: ↑ 15 50%.
- → Fitz-Hugh Curtis syndrome (FHCS) or Perihepatitis: PID → TOA, if ruptured will extend → pelvic peritonitis + FHCS.
 - → **FHCS:** TOP + peritonitis + perihepatitis.
 - → Infection ascends to upper abdomen → likes to infect liver capsule → bad adhesions around liver → right upper quadrant pain + diaphragm inflammation → bad nausea & vomiting + generalized abdominal pain.
- → Life threatening!!: acute rupture of TOA & peritonitis → urgent abdominal surgery.







Peritoneal adhesions

Differential Diagnosis of: Acute Lower Abdominal Pain + Fever

Appendicitis:

- → Pain mainly be in the **right iliac fossa.**
- → GI symptoms: diarrhea rebound tenderness.
- → Low grade fever: unless there is rupture.

Ectopic Pregnancy:

- \rightarrow No fever.
- \rightarrow GI symptoms.
- → Purulent discharge.

Bleeding in pelvis due to ectopic/ovarian cyst/follicle rupture:

- → Signs & symptoms of hypovolemia (tachycardia hypotension).
- \rightarrow No fever.

PID:

- \rightarrow Reproductive age.
- → Married & sexually active.
- → Vaginal discharge.
- \rightarrow History of STDs or PID, IUCD.
- → Partner had neisseria or chlamydia.
- → On exam:
 - → Cervix doesn't look healthy: cervical motion tenderness.
 - \rightarrow No localization of the pain to the right side.
 - → Healthy ovaries on ultrasound.
 - → Negative pregnancy test.
- \rightarrow Sometimes the diagnosis of acute abdomen is not clear \rightarrow confirm by laparoscopy.

Tubo-Ovarian Abscess

Introduction:

- → **Tubo-ovarian abscess (TOA):** an end stage process of acute PID.
- Tubo-ovarian abscess (TOA): the accumulation of pus in the adnexa forming an inflammatory mass involving the oviducts, ovaries, uterus, or omentum.
- → Clinical presentation:
 - → Patient will look septic.
 - → Severe lower abdominal-pelvic pain.
 - \rightarrow Severe back pain.
 - \rightarrow Rectal pain.
 - \rightarrow Pain with bowel movements.
 - → Nausea and vomiting.

→ Examination:

- → Patient appears gravely sick.
- \rightarrow High fever.
- \rightarrow Tachycardia.
- → She may be in septic shock with hypotension.
- → **Abdominal examination:** peritoneal signs (guarding rigidity).
- \rightarrow **Pelvic examination:** severe pain \rightarrow rectal examination must be performed.
- → **Rectal examination:** bilateral adnexal masses may be palpated.
- → **Diagnosis:** PID patient has a palpable pelvic mass during bimanual examination.
- → **Pathophysiology:** an agglutination of pelvic organs (tube ovary bowel) → palpable complex.

FIGURE 22-5 Gross appearance of bilateral tubo-ovarian abscesses. (From Kumar V, Fausto N, Abbas A: *Robbins and Cotran pathologic* basis of disease, ed 7, Philadelphia, 2005, Saunders.)

Management:

- → **Treatment:** antibiotic regimen administered on an inpatient basis.
- → ≈ 75% respond to antimicrobial therapy alone.
- \rightarrow Failure of medical therapy \rightarrow abscess drainage.
 - → Drainage may require surgical exploration.
- → Percutaneous drainage guided by imaging studies (US or CT): initial option if possible.
- Trocar drainage (with or without placement of a drain): successful in up to 90% of cases in which the patient has failed to respond to antimicrobial therapy after 72 hours.
- → Inpatient IV clindamycin and gentamicin → fever defervescence within 72 hours.
- → No response or abscess rupture exposing free pus into peritoneal cavity → exploratory laparotomy with possible TAH & BSO or percutaneous drainage through a colpotomy incision.
 - → No laparotomy or drainage → significant mortality can occur. Exploratory laparotomy with possible TAH and BSO or percutaneous drainage through a colpotomy incision may be required.

Kaplan

Pelvic Inflammatory Disease:

- → PID is a nonspecific term for a spectrum of upper genital tract conditions ranging from acute bacterial infection to massive adhesions from old inflammatory scarring.
- → The most common initial organisms are chlamydia and gonorrhea. With persistent infection, secondary bacterial invaders include anaerobes and gram- negative organisms.
- Risk Factors: The most common risk factor is female sexual activity in adolescence, with multiple partners. PID is increased in the month after insertion of an IUD, but this is probably exacerbation of preexisting subclinical infection.

Cervicitis:

- → The initial infection starts with invasion of endocervical glands with chlamydia and gonorrhea. A mucopurulent cervical discharge or friable cervix may be noted. Cervical cultures will be positive, but symptoms are usually absent.
- \rightarrow WBC and ESR are normal
- → Management: Single dose orally of cefixime and azithromycin.

Acute Salpingo-oophoritis:

- → Usually after a menstrual period with breakdown of the cervical mucus barrier, the pathogenic organisms ascend through the uterus causing an endometritis; then the bacteria enter the oviduct where acute salpingo-oophoritis develops.
- → Bilateral lower abdominal-pelvic pain. Onset may be gradual to sudden. Nausea and vomiting may be found.
- → Mucopurulent cervical discharge, cervical-motion tenderness, and bilateral adnexal tenderness are present. Fever, tachycardia, abdominal tenderness, peritoneal signs, and guarding.
- → Elevated WBC and ESR. Pelvic sonography is usually unremarkable. Laparoscopy will show erythematous, edematous, purulent oviducts. Cervical cultures will come back positive for chlamydia or gonorrhea.
- → Management is often based on a presumptive diagnosis. Empiric broad spectrum coverage need to include N. gonorrhoeae or C. trachomatis as well as anaerobes (e.g., B. fragilis).

Chronic PID:

- Chronic bilateral lower abdominal-pelvic pain is present. Other symptoms may include history of infertility, dyspareunia, ectopic pregnancy, and abnormal vaginal bleeding. Nausea and vomiting are absent.
- → On examination, bilateral adnexal tenderness and cervical-motion tenderness is present, but mucopurulent cervical discharge is absent. Fever and tachycardia are absent.
- → Negative cervical cultures with normal WBC and ESR. Sonography may show bilateral cystic pelvic masses consistent with hydrosalpinges.
- → Diagnosis is based on laparoscopic visualization of pelvic adhesions.
- → Management: Outpatient mild analgesics for pain. Lysis of tubal adhesions may be helpful for infertility. Severe unremitting pelvic pain may require a pelvic clean-out (TAH, BSO). If the ovaries are removed, estrogen replacement therapy is indicated.

439 Summary

Pelvic inflammatory disease (Upper genital tract infections) Pathophysiology Occurs due to the disruption of the barrier (cervical canal). Possible sites of infection Endometrium: endometritis Fallopian tubes: salpingitis Ovaries: oophoritis Often due to ascending spread of microorganisms → cervicitis → endometritis → salpingitis → oophoritis → chronic PID (if not treated) Upper genital tract is normally sterile Sexually active females of reproductive age Multiple sexual partners, unprotected sex (no condom) Reinfection if untreated male partner (80%): males can be carriers of many STDs but females present dramatically History of prior STIs or PID latrogenic: ■ They increase the risk of pelvic infection o Endometrial biopsy, D&C, hysterosalpingogram Barrier method: if partner is infected OCP: estrogen encourages lactobacilli proliferation Microbial etiology is unknown in most cases, often treated as polymicrobial infection Pathogens Causative organisms (order based on frequency): o Neisseria gonorrhoeae ■ Causes acute infection, may lead to tubal obstruction ending with infertility & ectopic pregnancy Gram negative diplococci, mucopurulent discharge, STD Chlamydia trachomatis Causes chronic infection, may lead to tubal blockage Obligate intracellular, STD Mycoplasma genitalium Clinical features Classic presentation: young, sexually active women who present with lower abdominal pain and Clinical diagnosis of PID: o Fever >38.3ºC & chills

	 Lower abdominal pain and tenderness (mostly bilateral) 		
	 Abnormal discharge (cervical or vaginal) 		
	 Uncommon: N/V, dysuria, and abnormal uterine bleeding, lower back pain 		
	Chronic disease (often due to chlamydia)		
	Constant pelvic pain		
	Dyspareunia		
	o Palpable mass		
	 Very difficult to treat, may require surgery 		
Diagnostics	Diagnosis is primarily based on clinical findings (Hx & PE). There is no single test to confirm if the		
	Pt has PID.		
	Important diagnostic criteria		
	 Patient history: most often a sexually active young woman 		
	 Lower abdominal pain 		
	Vaginal examination		
	 Cervical motion tenderness: severe cervical pain elicited by pelvic 		
	examination		
	■ Uterine and/or adnexal tenderness		
	 Purulent, bloody cervical and/or vaginal discharge 		
	Blood tests: elevated ESR, leukocytosis Pregnancy test: to rule out an (ectopic) pregnancy		
	Cervical and urethral swab		
	 Gonococcal and chlamydial DNA (PCR) and cultures 		
	 Giemsa stains of discharge can show cytoplasmic inclusions in C. trachomatis 		
	infections, but not in N. gonorrhoeae infection.		
	 Negative gram smear does NOT rule out PID 		
	 Imaging is not routinely required for diagnosis of PID. 		
	Laparoscopic visualization:		
	 Most accurate method to confirm PID 		
	 Only used for patients with unconfirmed cases or not responding to 		
	treatment		
	 Transvaginal ultrasound indication: in case of severe PID (high fever, n/v, severe 		
	pain), to exclude tubo-ovarian abscess and to hospitalize women with severe PII		
Management	Start treatment as soon as you <u>suspect</u> PID		
	 Empirical antibiotic therapy: broad spectrum antibiotics 		
	Outpatient regimen		
	■ Mild to moderate PID		
	 One single dose of IM ceftriaxone and oral therapy with doxycycline 		
	 Add metronidazole if there is signs of vaginitis 		
	 Inpatient regimen (parenteral antibiotics) 		

 No response to or inability to take outpatient oral regimen Non-compliance concerns
 Severe PID with nausea, vomiting, and/or high fever Pregnancy ■ Possible combinations (should be administered for 14 days) IV Cefoxitin or cefotetan plus IV/PO doxycycline . In the case of tubo-ovarian abscess or signs of vaginitis: Add oral metronidazole or clindamycin Surgical intervention is considered if antibiotics therapy failed As it's a result of STD, treat male partners and educate to prevent reinfection Short term complications Fitz-Hugh-Curtis syndrome (perihepatitis) Inflammation of the liver capsule caused by spread of infection Characterized by right upper quadrant pain Characterized by violin-string-like adhesions extending from the peritoneum to the liver

Managed the same as PID Tubo-ovarian abscess
 Salpingitis → obstruction of the tube → formation of TOA (Inflammatory mass in fallopian tubes or ovary) May rupture and cause peritonitis: a life-threatening event that calls for urgent

abdominal surgery

Diagnosed by transvaginal US: bilateral pelvic masses

■ Image-guided drainage of abscess

Infertility: PID is one of the most common causes of infertility
 Ectopic pregnancy (especially chlamydia)

■ Laparotomy ■ Laparoscopy

Long term complications

• Scarring and adhesions

DDx of lower abdominal pain + fever

Chronic pelvic pain

Managed the same as PID. If not improved or TOA with abscesses >/= 10 cm in

	Ectopic pregnancy	PID	Appendicitis
Clinical features	Lower abdominal pain (unilateral), guarding Vaginal bleeding Amenorrhea	Lower abdominal pain (bilateral) Fever Menorrhagia, metrorrhagia Dyspareunia	Initially: diffuse epigastric pain Later: localized right lower quadrant pain N/V Fever
Diagnostic rules	Positive pregnancy test US	Cervical discharge Cervical motion tenderness	McBurney's point tenderness Rebound tenderness US
Therapy	Methotrexate or surgical removal	Antibiotics	Appendectomy

Ouiz

Question 1:

- → You are counseling a lady about different methods of contraception. What is the characteristic feature of intrauterine contraceptive device?
 - A. Candida
 - B. Trichomoniasis
 - C. Chlamydia
 - D. Syphilis

Question 2:

- → A 29-year old lady presented with abdominal pain, fever and chills. Her temperature is 38.6C and she has lower abdominal tenderness. On speculum examination showed mucopurulent discharge. Which one of the following is the most likely diagnosis?
 - A. Bacterial vaginosis.
 - B. Gonorrhea cervicitis.
 - C. Pelvic inflammatory disease.
 - D. Trichomonas vaginitis.

Question 3:

- → A 30-year-old lady P2 +0 with 2 previous C-section. She has regular menstrual cycles. She used the oral contraceptive pills for 2 years but is off the pill for one year. She came to you as a case of secondary infertility. What is the most likely diagnosis?
 - A. Endometriosis.
 - B. Polycystic ovarian syndrome.
 - C. Prolonged use of oral contraceptive pills.
 - D. Tubal blockage due to adhesions.

Question 4:

- → A female in the 7th week of gestation presented with lower pelvic pain and bleeding. she noticed some passing tissue. What is your diagnosis?
 - A. Inevitable abortion.
 - B. Missed abortion.
 - C. Incomplete abortion.
 - D. Complete abortion.

Э	а	Э	а
7	8	ζ	L

Quiz

Question 1:

- → A 29-year-old woman with frothy offensive vaginal discharge, with motile flagellated organism. What is the organism?
 - A. Increase incidence of endometrial cancer
 - B. Inhibits ovulation.
 - C. Reduce pelvic inflammatory disease.
 - D. Risk of ectopic pregnancy if she gets pregnant.(by causing PID > fibrosis of fallopian tube > ectopic)

Question 2:

- → A newly married woman was admitted to the ward with fever, abdominal pain and foul-smelling vaginal discharge. Pelvic ultrasound showed bilateral pelvic masses. What is the most serious long-term complication in this case?
 - A. Amenorrhea
 - B. Infertility
 - C. Preterm labor
 - D. Endometriosis

Question 3:

- → Which one of the following lower genital tract infections require treatment for the husband?
 - A. Bacterial vaginosis
 - B. Monilial infection
 - C. Chlamydia infection
 - D. Group B streptococcus infection

Question 4:

- → A 29-year-old lady, presented with abdominal pain, fever an is 38.6C and lower abdominal tenderness. On speculum examination discharge. Which one of the following is the most likely diagnosis?
 - A. Endometriosis
 - B. Gonorrhea cervicitis
 - C. Ovarian torsion
 - D. Pelvic inflammatory disease

О	Э	8	8
7	8	Z	L

Reference



Pelvic Pain

Acute, Cyclic (Dysmenorrhea), and Chronic

ANDREA J. RAPKIN • JOSEPH C. GAMBONE

CLINICAL KEYS FOR THIS CHAPTER

- Acute pelvic pain of sudden onset can be caused by both genecologic and nongynecologic disorders. Adnexal accidents such as rupture or torsion of ovarian cysts, pelvic infections, tubal rupture of ectopic pregnancies, and aborting intrauterine pregnancies are the more common gynecologic causes. Gastrointestinal condi-tions, such as appendicitis and bowel obstruction, and genitourinary problems, such as cystitis and ureteral stones are the significant nongynecologic causes. Early diagnosis and expeditious treatment, offen surgical, are important for safe and effective clinical management of acute pelvic nain.
- important for safe and effective clinical management of actue pelvic pain.

 The most common type of cyclic pelvic pain is recurrent painful mentruation or dysmenorrhea. Dysmenorrhea may be primary, when caused by excessive production of prostaglandins (PGs), mainly PGFs, nor secondary, when an underlying condition for the pain such as adenomyosi or endometriosis is diagnosed. Primary dysmenorrhea occurs in ovulatory cycles and in younger women (17 to 22 years). Other causes of secondary menstrual and perimenstrual recurrent pain include chronic pelvic infection, degenerating fibroids, and pelvic congestion. Secondary dysmenorrhea is not limited to pain only during menses and typically occurs in older women (~30 years of age).

 Treatment of primary dysmenorrhea involves provision
- Treatment of primary dysmenorrhea involves provision of an explanation for the cause of the pain, and reassur-

- ance, along with nonsteroidal antiinflammatory drugs (NSAIDs), hormonal contraceptives to block ovulation, and other nonpharmaceutical interventions such as transcutaneous nerve stimulation and acupuncture. Treatment of secondary dysmenorrhea depends on the underlying cause of the pain, with NSAIDs the preferred initial choice.

 Chronic pelvic pain (CPP) is noncyclic pain that lasts for more than 6 months. Like other forms of pelvic pain, CPP has both gynecologic and nongynecologic causes. Chronic pain, including CPP, differs from acute pain in several important and measurable ways. With acute pain, the pain perception, suffering, and behavior are usually commensurate with the degree of sensory input. With chronic pain, such as CPP, the suffering and behavioral responses may be quite exaggerated, and may persist even after the pain stimulus has remitted.

 The appropriate vacuation and treatment occurs when a multidisciplinary team manages the patient with ongoing, as opposed to episodic care. Psychatric referral for psychopharmacologic therapy may be needed. This aspect of therapy is crucial, because many of these patients may be severely depressed and they may be withdrawn interpersonally, sexually, and occupationally.

Pelvic pain is a frequent complaint in gynecology. It may be acute, cyclic, and associated with menstruation, or chronic, lasting for more than 6 months. Acute pelvic pain is sudden in onset and is usually associated with significant neuroautonomic reflexes such as nausea and vomiting, diaphoresis, and apprehension. There are several important gynecologic and nongynecologic causes of acute pain.

Half of all menstruating women are affected by painful menstruation or dysmenorrhea making it the most common type of pelvic pain. Ten percent of these women have severe symptoms necessitating time off from work or school. Chronic pelvic pain (CPP) includes reproductive and nonreproductive organ-related pelvic pain that is primarily acyclic and that lasts for 6 months or more.

Acute Pelvic Pain

It is important for the gynecologist to be aware of both the gynecologic and nongynecologic causes of acute pelvic pain (Box 21-1). Delayed diagnosis and treatment of acute pelvic pain may increase the morbidity and even the mortality.

Adnexal accidents, including torsion or rupture of an ovarian or fallopian tube cyst (Figure 21-1), can cause severe lower abdominal pain. Normal ovaries and fallopian tubes rarely undergo torsion, but cystic

CAUSES OF ACUTE PELVIC PAIN

nexal accidents, e.g., ovarian cyst torsion, rupture, or

hemorrhage
Acute infections, e.g., endometritis or pelvic inflammatory
disease

Pregnancy complications, e.g., ectopic gestation or abortion

Gastrointestinal, e.g., appendicitis, enteritis, or intestinal

obstruction
Genitourinary, e.g., cystitis, ureteral stones, or urethral
syndrome
Other, e.g., pelvic thrombophlebitis, vascular aneurysm,
or porphyria



FIGURE 21-1 Torsion of an ovarian cyst and adnexal blood vessels. Note the large clot that has formed in the adnexal area (arrow) due to obstruction of venous outflow from a left ovarian cyst. (From Clement PB, Young RH: Aldas of gynecologic surgical pathology, Philadelphia, 2000, Saunders.)

or inflammatory enlargement predisposes to these adnexal accidents. The pain of adnexal torsion can be intermittent or constant, is often associated with nausea, and has been described as reverse renal colic because it originates in the pelvis and radiates to the loin. An enlarging pelvic mass is found on examination and ultrasound, with decreased or absent blood flow to the adnexa on Doppler-ultrasonic studies. The need for surgical intervention is common and urgent.

Functional ovarian cysts (e.g., corpus luteal or follicular cysts) may rupture causing leakage of fluid or blood that causes acute pain from peritoneal irritation. When there is significant associated bleeding, the pain may be followed by a hemoperitoneum and hypovolemia. Surgical intervention is mandatory in this setting, after adequate ressuscitation with packed red cells and

mia. Surgical intervention is mandatory in this setting, after adequate resuscitation with packed red cells and intravenous fluide

Reproductive organ infections such as endometritis or salpingo-oophoritis (commonly referred to as pelvic inflammatory disease or PID) can present acutely. Rupture of a tubo-ovarian abscess is a surgical emergency that can progress to hypotension and oli-guria after initially presenting with diffuse lower abdominal pain. Pelvic infection is covered in greater

guria after initially presenting with diffuse lower abdominal pain. Pelvic infection is covered in greater detail in Chapter 22.

Several complications of early pregnancy, such as ectopic gestation (see Chapter 24) and threatened or incomplete abortion, can cause acute pelvic pain and are generally associated with abnormal bleeding. Ectopic tubal pregnancies produce pain as the fallopiant ube dilates and ruptures into the abdominal cavity, and can be life-threatening when not diagnosed expeditiously.

Nongynecologic causes of acute lower abdominal pain (see Box 21-1) are frequently in the differential diagnosis when a woman presents with pelvic pain. Appendicitis is a common gastrointestinal cause of acute lower abdominal pain that eventually localizes to the right lower quadrant of the abdomen (McBurney point). The unilateral intensity of the pain usually differentiates it from salpingo-oophoritis. Rupture of an infected appendix into the pelvic cavity can have a significant adverse effect on female fertility and may be a diagnostic challenge during pregnancy (see Chapter 16). Diverticular abscess is also not uncommon but usually occurs in postmenopausal women.

Acute cystitis (see Chapter 22) and ureteral stone formation (lithiasis) and passage are both frequently painful. Uretral syndrome can present acutely and become chronic over time when not recognized and treated. Painful pelvic floor disorders are covered in more detail in Chapter 23.

an enigmatic disorder, it is one of the most comm presenting complaints in a gynecologic practice. As public health problem, it results in great cost society in terms of hospital services, loss of produ tivity, and human misery.

CHARACTERISTICS OF SOME CAUSES OF SECONDARY DYSMENORRHEA

Pain extends to premenstrual or postmenstrual phase or may be continuous; may also have deep dyspareunia, pre-menstrual spotting, a fixed retroverted uterus, and tender pelvic nodules (especially on the uterosacral ligaments); onset is usually in the 20s and 30s but may start in the

reivic inflammation
Initially pain may be mentrual, but often with each cycle it extends into the premenstrual phase; may have intermenstrual bleeding, dyspareunia, and pelvic tenderness.

Adenomyosis, Fibroid Tumors

Uterus is generally symmetrically enlarged and may be mildly tender; dysmenorrhea is associated with a dull pelvic dragging sensation; hypermenorrhea and dyspareunia may be present.

Operate Cycle (Service)

Ovarian Cysts (Especially Endometriosis and Lute

ould be clinically evident.

A dull, ill-defined pelvic ache, usually worse premenstru-ally, relieved by menses; not all investigators agree that this is a cause of chronic pelvic pain.

Obviously, not all lower abdominal and low back pains are of gynecologic origin. Careful evaluation is needed to distinguish gynecologic pain from that of orthopedic, gastrointestinal, urologic, neurologic, and psychosomatic origin. The relationship between pelvic pain and the underlying gynecologic pathology is often inexplicable, and frequently the pain is thought to be psychosomatic.

Anatomy and Physiology

The innervations of the pelvic organs that convey information related to pain are shown in Table 21-1. Painful impulses that originate in the skin, muscles, bones, joints, and parietal peritoneum travel in somatic nerve fibers, whereas those originating in the internal

organs travel in visceral nerves.

Visceral pain is more diffusely spread than somatic pain because of a phenomenon called *viscerosomatic* convergence, and the lack of a well-defined projection area in the sensory cortex for its identification. Vis-cerosomatic convergence occurs in all second-order neurons in the dorsal horn of the spinal cord that receive visceral input. No second-order neurons in the dorsal horn receive only visceral input. The viscerosomatic neurons have larger receptive fields than do the somatic second-order neurons. Visceral pain is therefore the subscience of the control of t fore usually referred to the skin, which is supplied by the corresponding spinal cord segment (referred pain). For example, the initial pain of appendicitis is referred to the epigastric area because the affected structures are innervated by the thoracic cord segments T8, T9,

and T10.

The structures of the female genital tract vary in their sensitivity to pain. The skin of the external

Chronic Pelvic Pain

CPP refers to pelvic pain of more than 6 months' duration that has a significant effect on daily function and quality of life. CPP includes reproductive and nonreproductive organ-related pain. Although CPP is

TABLE 21-1			
NERVES CARRYING PAINFUL IMPULSES FROM THE PELVIC ORGANS			
Organ	Spinal Segments	Nerves	
Perineum, vulva, lower vagina	S2-4	Pudendal, inguinal, genitofemoral, posterofemoral cutaneous	
Upper vagina, cervix, lower uterine segment, posterior urethra, bladder trigone, uterosacral and cardinal ligaments, rectosigmoid, lower ureters	\$2-4	Pelvic parasympathetics	
Uterine fundus, proximal fallopian tubes, broad ligament, upper bladder, cecum, appendix, terminal large bowel	T11-12, L1	Sympathetics via hypogastric plexus	
Outer two-thirds of fallopian tubes, upper ureter	T9-10	Sympathetics via aortic and superior mesenteric plexus	
Ovaries	T9-10	Sympathetics via renal and aortic plexus and celiac and mesenteric ganglia	
Abdominal wall	T12-L1 T12-L1 T12-L1 L1-2	Sympathetics via renal and aortic plexus and celiac and mesenteric ganglia Iliohypogastric Ilioinguinal Genitofemoral	

Reference

genitalia is exquisitely sensitive. Pain sen gentatia is Eaquistic yestisture. The instrustation is vari-able in the vagina, and the upper vagina is somewhat less sensitive than the lower. The cervix is relatively insensitive to small biopsies but is sensitive to deep incision or to dilation. The uterus is quite sensitive. The ovaries are insensitive to many stimuli, but they are sensitive to rapid distention of the ovarian capsule or compression during physical examination.

Patient Evaluation

HISTORY

HISTORY
A pain history should be obtained during the first visit.
Characteristics of the pain should be determined, including its location, radiation, severity, allevating and aggravating factors, as well as the effects of menstruation, level of stress, work, exercise, and intercourse. Symptoms related to the gastrointestinal, genitourinary, musculoskeletal, and neurologic systems should be ascertained. This process can be guided by the Pain History Mnemonic outlined in Box 21-5.

PHYSICAL EXAMINATION

The abdomen should be examined initially, and the patient should be asked to point to the exact location of the pain and its radiation. An attempt should be made to duplicate the pain by palpating each abdominal quadrant. The severity of the pain should be quantified on a 0 to 10 scale (0 = no pain, 10 = hitting thumb with a hammer).

PAIN HISTORY MNEMONIC (OLD CAARTS)

Onset: When and how did the pain start? Does it change

over time?

Location: Localize specifically—can the woman put a finger on it?

Duration: How long does it last?

Characteristics: e.g., cramping, aching, stabbing, itching the value of the control of the cont

activity?

Associated symptoms:

Gynecologic (e.g., dyspareunia, dysmenorrhea, abnormab lbeeding, discharge)

Gastrointestinal (e.g., constipation, diarrhea, bloating, gas, rectab leeding)

Genitourinary (e.g., urinary frequency, dysuria, urgency, incontinence)

Neurological (specific nerve distribution of the pain)

Radiation: Does the pain move to other areas of the body?

Temporal: Time of day and relationship to daily activities

Severity: On a scale of 0 to 10 (from no pain to the most severe imaginable)

Modified from Rapkin AJ, Howe CN: Chronic pelvic pain: a review. In Family practice recettification, Monroe Township, NJ, 2006, Medical World Communications, 28:59-67.

The abdominal wall should be examined for evidence of myofascial trigger points and for iliohypogastric (Ti2, L1), libinguinal (Ti2, L1), or gentice-femoral (L1, L2) nerve entrapment. Each dermatome of the abdominal wall and back should be palpated with a fingertip and points of severe tenderness or "jump signs" should be marked with a pen. The patient should be asked to tense the abdominal muscles by performing a straight-leg raising maneuver (both legs raised at least 6 inches with both knees straight) or a partial sit-up. Points that are more tender or that reproduce the patient's pain suggest nerve entrapment, impingement, or a muscular trigger point pain. These points should be injected with 2 to 3 ml. of 25% bupjavacaine. Chronic abdominal wall pain is confirmed if the pain level is reduced by at least 50% and outlasts the duration of the local anesthetic.

A thorough pelvic examination should be performed, with an attempt made to reproduce and localize the patient's pain. The examination should be performed genty so as to prevent involuntary guarding, which may obstruct the findings. The examination may be suggestive of specific pelvic pathology. For example, patients with endometriosis may have a fixed retroverted uterus with tender uterosacral nodularity. An adnexal mass may suggest ovarian pathology. Bilateral, tenders. The abdominal wall should be exa

retroverted uterus with tender uterosacral nodularity. An adnexal mass may suggest ovarian pathology, Bilateral, tender, irregularly enlarged adnexal structures may suggest prior salpingitis with subsequent formation of adhesions and bilateral hydrosalpinges. A prolapsed uterus may account for pelvic pressure, pain, or law bedseably.

FURTHER INVESTIGATIONS

Psychological evaluation should be requested if an obviously traumatic event has occurred with the onset of pain; if there is obvious depression, ansiety, catastrophizing, psychosis, or secondary gain; or to aid in the planning of pain management sessions. The latter may involve cognitive behavioral and stress reduction

may involve cognitive behavioral and stress reduction therapy.

Laboratory studies are of limited value in the diagnosis of CPP, although a complete blood count, erythrocyte sedimentation rate (ESR), and urinalysis are indicated. The ESR is nonspecific and will be increased in any type of inflammatory condition, such as subacute salpingo-oophoritis, tuberculosis, or inflammatory bowel disease. Patients who are engaging in sexual intercourse should have a pregnancy test if they have a uterus and are not postmenopausal. Pelvic ultrasonography should be performed because the pelvic examination may mis an adnexal mass, particularly in obese patients or in those who are unable to relax. Routine urine analysis and studies to rule out sexually transmitted infections are indicated depending on the patient's symptoms and risk factors. If bowel or urinary symptoms are present, an abdominal and pelvic computed tomographic (CT) scan, endoscopy, cystoscopy,

or CT urogram may be useful. Similarly, if there is clinical evidence of musculoskeletal disease, a lumbosacral x-ray film, CT scan, magnetic resonance imaging (MRI) scan, or orthopedic consultation may be in order. Diagnostic laparoscopy is the ultimate method of diagnosis for patients with CPP of undetermined eti-

diagnosis for patients with CPP of undetermined eti-ology. Laparoscopie examination and binanual exam-ination may differ in 20-30% of cases. Laparoscopy should only be performed if no etiology for the pain can be identified, or when indicated to treat specific pathology.

Differential Diagnosis

CAUSES OF CHRONIC PELVIC PAIN

CAUSES OF CHRONIC PELVIC PAIN
Of women with CPP who are subjected to diagnostic
laparoscopy, approximately a third have no apparent
pathology, a third have endometriosis, somewhat less
than the remaining third have adhesions or stigmata of
past pelvic inflammatory disease (PID), and the small
remainder have other causes (Box 21-6).

Endometriosis

Endometriosis may be missed visually at the time of diagnostic laparoscopy in as many as 20-30% of women who have histologically proven disease, so it is justifiable to initiate hormonal treatment based on a pre able to illutate normonal treatment based on a pre-sumptive diagnosis of the disease once other etiologies have been ruled out. Current hormonal therapies are often very effective and may preclude the need to undergo a costly surgical procedure that is not

thout risk.

The size and location of the endometriotic implants The size and location of the endometriotic implants do not appear to correlate with the presence of pain, and the reasons for the pain are not fully understood, although prostaglandins, cytokines, and innervation of lesions have been hypothesized. Endometriosis is covered more extensively in Chapter 25.

Chronic Pelvic Inflammatory Disease

Chronic PID may cause pain because of anatomic distortions (hydrosalpinges and adhesions between the tubes, ovaries, and intestinal structures) that result

BOX 21-6

GYNECOLOGIC CAUSES OF CHRONIC PELVIC PAIN

Endometriosis

Endometriosis
Salpingo-oophoritis (pelvic inflammatory disease)
Ovarian remnant syndrome
Pelvic congestion syndrome
Cyclic pelvic (uterine) pain
Myomata uteri (degenerating)
Adapomyosis

from the acute infection. It is also thought that prior from the acute infection. It is also thought that prior PID may lead to "upregulation" of sensory processing from the previously inflamed tissue. Persistent active infection is called acute PID, even if fever and pertioneal signs are absent. Recurrent active infections that require antibiotic therapy must be ruled out. PID is also discussed in Chapter 22.

Before ascribing symptoms to adhesions, one must have specifically noted adhesions in the area of pain localization, because most patients with extensive pelvic adhesions discovered incidentally during surgery for other reasons are asymptomatic.

Ovarian Pain

Ovarian Pain
Ovarian oysts are usually asymptomatic, but episodic
pain may occur secondary to rapid distention of the
ovarian capsule or rupture or leakage of irritating fluid
into the pertioneal cavity. An ovary or an ovarian
remnant may occasionally become retropertioneal
secondary to inflammation or previous surgery, and
cyst formation in these circumstances may be painful.
Some women, for unknown reasons, may develop multiple recurrent functional hemorrhagic ovarian cysts
that seem to cause pelvic pain and dyspareunia on an
intermittent basis.

Hormonal suppression of ovulation is usually an

intermittent basis.

Hormonal suppression of ovulation is usually an effective treatment for painful functional cysts. The differential diagnosis of ovarian masses is covered in Chapter 20. An ovarian cyst may also be an endometrioma, and if an endometrioma is suspected based on history, physical examination, and ultrasonography, surgical excision is usually indicated. Other benign and malignant ovarian neoplasms can contribute to CPP, but are often asymptomatic. A benign cystic teratoma (dermoid) for example can intermittently twist and untwist, causing repeated episodes of subacute pain.

Adenomyosis (or endometriosis interna) can cause dysmenorrhea, dyspareunia, and menorrhagia, but rarely does it cause chronic daily intermenstrual pain. Uterine myomas usually do not cause pelvic pain unless they are degenerating, undergoing torsion (twisting on their pedicles), or compressing pelvic nerves. A completely submucous lelomyoma can attempt to deliver via the cervix, which may cause considerable crampy uterine pain akin to childbirth. This is generally associated with heavy vaginal bleeding. During pregnancy, uterine myomas can cause pain from rapid growth or infarction.

Pelvic pain is not likely to be caused by variations in uterine position, but deep dyspareunia may occasionally be associated with uterine retroversion, especially when the uterus is fixed in place by adhesions or endometriosis. The pain has been ascribed to irritation of pelvic nerves by the stretching of the uterosacral ligaments as well as to congestion of pelvic veins Adenomyosis (or endometriosis interna) can cause

secondary to retroversion. The dyspareunia is typically worse during intercourse in the missionary position and is improved in the female superior position. A tender uterus that is in a fixed retroverted position usually signifies other intraperitoneal pathology, such as endometriosis or PID, and diagnosis rests on laparoscopic findings.

Pelvic Congestion Syndrome

Pelvic Congestion Syndrome
The concept of a pelvic congestion syndrome still has many proponents. This entity has been described in multiparous women who have pelvic vein varicostites and congested pelvic organs. The pelvic pain is worse premenstrually and is increased by fatigue, standing, and sexual intercourse. Many women with this condition are noted to have a mobile, retroverted, soft, boggy, and slightly enlarged uterus. There may be associated menorrhagia and urinary frequency. Dilated veins may be seen on pelvic MRI with contrast. Factors other than venous congestion may be involved in the genesis of pain, because most women with pelvic varicosities have no pain. Surgery for this condition, consisting of hysterectomy and oophorectomy, may be beneficial for women who have completed their families, as is ovarian hormonal suppression (decreased blood flow to the pelvic organs) and cognitive behavioral therapy. A few uncontrolled studies have suggested that embolization of involved veins by an interventional radiologist may be helpful. gist may be helpful.

Genitourinary Pelvic Pain

A variety of genitourinary problems may result in CPP. Urethral syndrome, trigonitis, and interstitial cystitis/ painful bladder syndrome are prime examples. Urinary urgency, frequency, nocturia, and midline pelvic pain may suggest interstitial cystitis/painful bladder syndrome. A thorough genitourinary evalua-tion is an important part of the workup for CPP when the above symptoms are reported. As many as one in five women have interstitial cystitis/painful bladder syndrome (see Chapter 23).

Gastrointestinal Pain

Gastrointestinal Pain
Gastrointestinal sources of CPP include penetrating
neoplasms of the gastrointestinal tract, irritable
bowel syndrome, functional abdominal pain syndrome (FAPS), celiac disease, partial bowel obstruction, inflammatory bowel disease, diverticulitis, and
hernia formation. Because the innervation of the
lower intestinal tract is the same as that of the uterus
and fallopian tubes, pelvic pain may be confused with
pain of gynecologic origin. Irritable bowel syndrome is
the most common gastrointestinal cause of pelvic pain.
Pain that is present at times of alteration of form or
frequency of bowel movements, increased before and
improved after a bowel movement, and especially if
worse with stress and eating, may be irritable bowel

syndrome. Red flags for a possible gastroi history of bowel cancer, blood in the stool, nocturnal pain, and alteration of stool caliber.

Neuromuscular Pain

Neuromuscular Pain
Pain of neuromuscular origin, which is experienced as low back pain or abdominal wall pain, usually increases with activity and stress. Trigger points and myalgia of the abdominal wall and pelvic floor muscles can cause pelvic pain, vulvodynia, and dyspareunia. Chronic low back pain without lower abdominal pain is seldom of gynecologic origin. Fibromyalgia, or generalized myo-fascial pain syndrome can also cause pelvic pain. Occasionally, neuromuscular symptoms are accompanied by a pelvic mass on examination or diagnostic imaging, and surgical exploration may reveal a neuroma, sarcoma, or bony tumor. Entrapped or compressed nerves in the abdominal wall (iliohypogastric and ilioinguinal nerves most commonly) or pelvic floor (pudendal nerve) are often unrecognized sources of pain. The nerves may become entrapped after surgery, physical trauma, pregnancy and delivery, or occupational injury.

A pathologic diagnosis may not be made in approxi-mately one third of patients with CPP, even after laparoscopy. This has led to the postulation that psychological factors may be primary. When sub-jected to the Minnesota Multiphasic Personality Inventory (MMPI), these patients have shown a greater degree of anxiety, hypochondriasis, and hys-teria than control subjects. The profiles are similar, however, in patients who have chronic nain with teria than control subjects. The profiles are similar, however, in patients who have chronic pain with organic pathology, indicating that chronic pain per se engenders a complex, debilitating, psychological response. Patients with chronic pain, with or without anatomic pathology, tend to feel depressed, anxious, fearful, helpless, and passive. They withdraw from social and sexual activity and are overwhelmed by pain and suffering Many have nost-traumatic stress disor. social and sexual activity and are overwhelmed by pain and suffering. Many have post-traumatic stress disorder (PTSD) from emotional, physical, or sexual trauma. Women with CPP are also a trisk of developing chronic fatigue syndrome. Women with depression, anxiety, or PTSD must be treated with psychological and/or psychopharmacological therapy as part of the multidisciplinary management of their CPP.

Pain Percention Factors

Pain Perception Factors
Chronic pain is characterized by neurophysiological,
emotional, and behavioral responses that are different
from those of acute pain. Both acute and chronic pain
involve a stimulus and a psychic response; for acute
pain, these responses may be adaptive and appropriate, whereas for chronic pain this may not be the case.
The response to chronic pain may be greatly affected

by operant conditioning. The patient's reaction to pain and the reaction of significant others to the patient and her pain may be so reinforcing that the behavior may persist even after the painful stimulus has resolved. With acute pain, the pain perception, suffering, and behavior are usually commensurate with the degree of sensory input. In chronic pain, the suffering and behavioral responses to a given sensory input may be quite exaggerated and may persist even after the stimulus has remitted.

Modulation of Sensation

Modulation of Sensation
Pain impulses are subjected to a large amount of modulation en route to, and within, the central nervous system. The first synapse in the dorsal horn is an important focus of enhancement, inhibition, or facilitation. Modulation of sensations may also occur within the spinothalamic system, the descending inhibitory neurosystems, the frontal cortex, and other brain regions. Various neurotransmitters and neuromodulations are present in the dorsal horn and at higher levels of the neuraxis. Some excitatory modulators include substance P. glutamate, asparate, calcitonin gene-related peptide (CGRP), and vasoactive intestinal peptide (VIP). Inhibitory neuromediators include endogenous opioid peptides, norepinephrine, serotonin, and y-aminobutyric acid (GABA). Nerve axons that have been compromised after inflammation, stretch, or crush injury can develop abnormal sodium channels. These changes play an important role in the development of allodynia (pain with gentle touch) and hyperalgesia (pain with stimuli that are not touch) and hyperalgesia (pain with stimuli that are not normally painful) in many women with CPP. Within this context, anxiety, loss of self-efficacy,

fear of pain, depression, and other psychological states are also considered to be facilitators or inhibitors of neurologic transmission. It is possible that many forms of CPP may result from modulation of afferent impulses (upregulation) or abnormality of descending inhibition in the dorsal horn, spinal cord,

When treating patients with CPP, a therapeutic, sup-When treating patients with CPP, a therapeutic, supportive, and sympathetic fout structured) physician-patient relationship should be established. The patient should be given regular follow-up appointments, and should not be told to call only if the pain persists. This reinforces pain behavior as a means of procuring sympathy and medical attention.

A negative evaluation or pathological findings not amenable to therapy (e.g., dense pelvic adhesions) does not mean that the patient should be discharged from care without therapy directed toward her symptoms. After initial reassurance that there is no serious underlying pathology and education as to the likely

mechanisms of pain production (including central nervous system factors), symptomatic therapy should be undertaken. The symptoms of pain should be approached with the seriousness and direction afforded to any other condition.

THE MUITIDISCIPLINARY TEAM

THE MULTIDISCIPLINARY TEAM

The most productive strategy for the management of patients with CPP is a multidisciplinary approach. The personnel should include a gynecologist, a psychologist who also has expertise in chronic pain, sexual and marital counseling, a physical therapist with pelvic floor muscle expertise, and for more complex cases requiring diagnostic or therapeutic nerve blocks, an anesthesiologist. An acupuncturist may also be a useful referral, it is the role of the psychologist to provide cognitive behavioral pain management and stress reduction, assertiveness training, and adaptive coping strategies, as well as marital and sexual counseling. Psychiatric referral for psychopharmacologic therapy may be needed. This aspect of therapy is crucial, because many of these patients have become severely depressed and often withdrawn interpersonally, sexually, and occupationally. Depression may be secondary to pain, but without treatment of the depression, the pain may persist. Relaxation, cognitive and behavioral therapies are employed to replace the pain behavior and its secondary gain with effective behavioral responses. Multidisciplinary management has been shown to be more effective than traditional gynecologic management. tional gynecologic management.

MEDICAL AND SURGICAL MANAGEMENT

The gynecologist continues to assess progress, coor-The gynecologist continues to assess progress, coordinate care, and provide periodic gynecologic examinations. In the initial stages of therapy, a trial of ovulation and or menstrual suppression with combined hormonal contraception (pills, patches, rings; cyclic or continuous), high-dose or intrauterine progestins or a gonadotropin-releasing hormone analogue (GnRH-a) may be helpful. Ovulation and/or analogue (GnRH-a) may be helpful. Ovulation and/or menstrual suppression is especially helpful in patients who have midcycle, premenstrual, or menstrual exacerbation of pain, or in those who have ovarian pathology, such as periovarian adhesions or recurrent functional cyst formation. NSAIDs are also useful. Pharmacologic approaches to increase inhibitory neuromodulators such as norepinephrine, serotonin (5-HT), and GABA or sodium channel blockers are frequently used in the form of tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs), anticonvulsants or other GABA-ergic agents, and topical or injectable local anesthetics.

Surgical procedures that have not proved to be effective for CPP without pathology include unliateral adnexectomy for unilateral pain or total abdominal hysterectomy, presacral neurectomy, or uterine

Reference

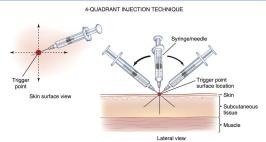


FIGURE 21-3 Trigger point injection technique for the abdominal wall for a patient with chronic pelvic pain. (From Auerbach PS: Wilderness medicine, ed 5, Philadelphia, 2007, Mosby.)

suspension for generalized pelvic pain. Lysis of adhesions is also usually nonproductive, with the possible exception of the situations where the site of adhesions, as visualized by the laparoscope, specifically coincides with the localization of pain. However, pelvic adhesions often recur following surgical lysis. Without proof of organic pathology or a reasonable functional explanation for the pelvic pain, a thorough psychosomatic evaluation should be carried out before any surgical procedure is considered. urgical procedure is considered.

INJECTION THERAPIES

Acupuncture, nerve blocks, and trigger-point injec-tions of local anesthetics may provide prolonged pain relief. Acupuncture has been used successfully for dysmenorrhea, and trigger-point injections and nerve blocks with local anesthetics have been used successfully for neuropathic and musculoskeletal pain. Acu-puncture probably increases spinal cord endorphins. In women with CPP, trigger points are typically found

either in the lower abdominal wall, lower back, or the vagina. A significant percentage of patients with pelvic pain have abdominal wall trigger points or nerveentrapments that respond to weekly or biweekly injections of a local anesthetic (usually up to five injections is sufficient) combined with alterations of activity or modification of behaviors that affect the area of pain. Injection of local anesthetic into myofascial trigger points (Figure 21-3) may abolish pain by lowering the impulses from the area of referred pain, thereby diminishing the afferent impulses reaching the dorsal horn to a level below the threshold for pain transmission (but the exact mechanism is not known). transmission (but the exact mechanism is not known). Repeated local anesthetic nerve blocks of areas of nerve impingement/entrapment combined with instructions to patients about alteration in physical activity and or physical therapy can be helpful. Along with nerve threshold altering medications, these interventions can down regulate neural hypersensitivity and permanently decrease or eliminate pain.

PELVIC INFLAMMATORY DISEASE

Microorganisms colonizing the endocervix and ascending to the endometrium and fallopian tubes cause PID. This is a clinical diagnosis implying that the patient has upper genital tract infection and

2015 CENTERS FOR DISEASE CONTROL (CDC)
RECOMMENDED FIRST-LINE REGIMEN FOR
UNCOMPLICATED GONOCOCCAL AND CHLAMYDIAL
INFECTIONS OF THE CERVIX, URETHRA, AND RECTU

Ceftriaxone 250 mg in a single intramuscular dose

Azithromycin 1 g orally in a single dose or doxycycline 100 mg orally twice daily for 7 days*

If ceftriaxone is not available: **cefixime** 400 mg in a single oral dose

PLUS

Azithromycin 1 g orally in a single dose or doxycycline 100 mg orally twice daily for 7 days*

PLUS

Test-of-cure in 1 week

If the patient has severe cephalosporin allergy: ${\bf azithromycin} \ 2\ g$ in a single oral dose

Test-of-cure in 1 week

From Centers for Disease Control and Prevention. Updated recommended treatment regimens for gonococcal infections and associated conditions—United States, April 2015. **www.cdc.pdf.**
*Because of the high prevalence of tetracycline resistance among Conococcal solated Surveillance Project isolates, particularly those with elevated minimum inhibitory conceinstations to celsivine, the use of azithromycin as the second antimicrobial is preferred as dual therappy.

inflammation. The inflammation may be present at any point along a continuum that includes endometritis, salpingitis, and peritonitis. PID is commonly caused by the sexually transmitted microorganisms N. gonorrhoeae and C. trachomatis. Recent evidence suggests that Mycoplasma genitatium can cause PID and may present with mild clinical symptoms similar to chiamytidal PID. Endogenous microorganisms found in the vagina, particularly the BV microorganisms, are often isolated from the upper genital tract of women with PID. The BV microorganisms include anaerobic bacteria such as Prevotella and peptostreptococci, as well as G. vaginalis. Less frequently, respiratory pathogens such as Haemophilus influenzae group A streptococci, and pneumococci can colonize the lower genital tract and cause PID.

Traditionally, the diagnosis of PID has been based

Traditionally, the diagnosis of PID has been based on a triad of symptoms and signs, including pelvic pain, cervical motion and adnexal tenderness, and the presence of fever. There is wide variation in many symptoms and signs among women with this condi-tion, which makes the diagnosis of acute PID difficult. Many women with PID exhibit subtle or mild sympmany women with PID exhibit subtle or mild symp-toms that are not readily recognizable as PID. The diag-nosis should be considered in women with any genitourinary symptoms, including, but not limited to, lower abdominal pain, excessive vaginal discharge, heavy and irregular vaginal bleeding, fever, chills, and

lower abdominal pain, excessive vaginal discharge, heavy and irregular vaginal bleeding, fever, chills, and urinary symptoms.

Pelvic organ tenderness, either uterine tenderness alone or uterine tenderness with adnexal tenderness, is usually present in patients with PID. Cervical motion tenderness suggests the presence of peritoneal inflammation, which causes pain when the peritoneum is stretched by moving the cervix and causing traction of the adnexa on the pelvic peritoneum. Direct or rebound abdominal tenderness may be present. Evaluation of both vaginal and endocervical secretions is an important part of the workup of a patient with PID. In women with PID, an increased number of polymorphonuclear leukocytes may be detected in a west mount of the vaginal secretions or the cervix may have a mucopurulent discharge.

Therapeutic regimens for PID must provide empirical, broad-spectrum coverage of likely pathogens, including N. gonorrhoeae, C. trachomatis, M. genitalium, gram-negative facultative bacteria, anaerobes, and streptococci. Recommended first-line outpatient treatment regimens for PID, per the 2015 CDC guidelines, are listed in Table 22-5. and parenteral treatment is illustrated in Table 22-5. and parenteral treatment is illustrated in Table 22-6.

An outpatient regimen of cefostitin and doxycycline is as effective as an inpatient parenteral regimen of the same antimicrobials. Box 22-1 lists the clinical criteria for hospitalization with parenteral treatment. Hospitalized patients can be considered for discharge when their fever is less than 99.5° F for more than 24

TABLE 22-5

2015 CENTERS FOR DISEASE CONTROL (CDC) RECOMMENDED FIRST-LINE REGIMEN FOR OU TREATMENT OF PELVIC INFLAMMATORY DISI

Ceftriaxone 250 mg intramuscularly in a single dose

Doxycycline 100 mg orally twice a day for 14 days

Metronidazole 500 mg orally twice a day for 14 days

Cefoxitin 2 g intramuscularly in a single dose and probenecid, 1 g orally administered concurrently in a single

Doxycycline 100 mg orally twice a day for 14 days WITH or WITHOUT

Metronidazole 500 mg orally twice a day for 14 days

Other parenteral third-generation cephalosporin (e.g.,

Doxycycline 100 mg orally twice a day for 14 days

WITH or WITHOUT Metronidazole 500 mg orally twice a day for 14 days

From Pelvic Inflammatory Disease: Sexually Transmitted Diseases Treatment Guidelines, 2015. Available at http://www.cdc.gov/std/treatment/2015/pid.htm. Accessed February 19, 2015.

hours, the white blood cell count is decreasing, rebound tenderness is absent, and repeat examination shows marked amelioration of abdominal tenderness. Sexual partners of women with PID should be evaluated and treated for urethral infection caused by chlamydia or gonorrhea. One of these STR is usually found in the male sexual partners of women with PID even if her diagnosis is not associated with chlamydia or gonorrhea.

TUBO-OVARIAN ABSCESS

TUBO-OVARIAN ABSCESS
Tubo-ovarian abscess (TOA), an endstage process of acute PID, is diagnosed when a patient with PID has a pelvic mass that is palpable during bimanual examination. The condition usually reflects an agglutination of pelvic organs (tube, ovary, and bowel) forming a palpable complex. Occasionally, an ovarian abscess can result from the entrance of microorganisms through an ovulatory site. Tubo-ovarian abscess is treated with an antibiotic regimen administered on an inpatient basis. Table 22-6 illustrates the parenteral treatment of PID, as per the 2015 CDC guidelines. About 75% of women with a tubo-ovarian abscess respond to antimicrobial therapy alone. Failure of medical therapy suggests

TABLE 22-6

Cefotetan 2 g IV every 12 hours

Cefoxitin 2 a IV every 6 hours

Doxycycline 100 mg orally or IV every 12 hours

Clindamycin 900 mg IV every 8 hours

PLUS

centamicin loading dose IV or IM (2 mg/kg of body weight), followed by a maintenance dose (1.5 mg/kg) every 8 hours. Single daily dosing (3 to 5 mg/kg) can be substituted. Alternative Parenteral Regime

Ampicillin/sulbactam 3 g IV every 6 hours

Doxycycline 100 mg orally or IV every 12 hours

From Pelvic Inflammatory Disease: Sexually Transmitted Diseases Treatment Guidelines, 2015. Available at http://www.cdc.gov/std/treatment/2015/pid. htm. Accessed February 19, 2015. IM, Intramuscularly; 1// intravenously.

PELVIC INFLAMMATORY DISEASE: CLINICAL CRITERIA FOR HOSPITALIZATION AND PARENTERAL TREATMENT

- 1. Surgical emergencies (e.g., appendicitis) not ruled out 2. Failed oral treatment (no improvement with short-
- Failed oral treatment (no improvement with short-term treatment).

 Compliance questionable (i.e., patient unable to follow or tolerate outpatient regimen)

 Severe illness (toxicity: nausea, vomiting, high fever)

 Tubo-ovarian abscess demonstrated on ultrasonogra-phy or suspected clinically

the need for drainage of the abscess. Although drainage may require surgical exploration, percutaneous drainage, guided by imaging studies (ultrasonography or computed tomography) should be used as an initial option if possible. Trocar drainage, with or without placement of a drain, is successful in up to 90% of cases in which the patient has failed to respond to antimicrobial therapy after 72 hours. Figure 22-5 depicts a surgical specimen with bilateral TOAs.





Med 441 Team:

Leader:

Sarah Alhamlan

Members:

Yara Almufleh

Good Luck!



Leaders:

Ateen Almutairi - Lama ALzamil

Members:

Ghaida Albraithen - Njoud Alali Haifa Alwaily



Med 439 Team:

Leader:

Bushra Alotaibi

Members:

Norah Aldahash - Shaden Alsaeedan