Chapter 103: Pelvic Inflammatory Disease

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INTRODUCTION AND EPIDEMIOLOGY

The term pelvic inflammatory disease (PID) comprises a spectrum of infections of the female upper reproductive tract. It is a common and serious disease initiated by ascending infection from the vagina and cervix. PID includes salpingitis, endometritis, myometritis, parametritis, oophoritis, and tubo-ovarian abscess and may extend to produce periappendicitis, pelvic peritonitis, and perihepatitis (Fitz-Hugh–Curtis syndrome). PID is the most common serious infection in sexually active women age 16 to 25 years.[1]

Long-term sequelae, including tubal factor infertility, implantation failure after in vitro fertilization, ectopic pregnancy, and chronic pain, may ultimately affect 11% of reproductive-aged women.[2] The most common cause of death is rupture of a tubo-ovarian abscess, and the mortality associated with rupture remains at 5% to 10%, even with current treatment methods.

PATHOPHYSIOLOGY

ORGANISMS ASSOCIATED WITH PID

Neisseria gonorrhoeae and *Chlamydia trachomatis* can be isolated in many cases of PID, and therapy is directed primarily against these organisms. However, polymicrobial infection, including infection with anaerobic and aerobic vaginal flora, is evident from cultured material from the upper reproductive tract.[3] **Table 103-1** lists common pathogenic organisms associated with PID. *N. gonorrhoeae* and *C. trachomatis* are often instrumental in initial infection of the upper genital tract, whereas anaerobes, facultative anaerobes, and other bacteria are isolated increasingly as inflammation increases and abscesses form.

TABLE 103-1

Organisms Associated with Pelvic Inflammatory Disease

Sexually Transmitted Organisms

Chlamydia trachomatis

Neisseria gonorrhoeae

Herpes simplex virus (types 1 and 2)

Trichomonas vaginalis

Endogenous Genital Tract *Mycoplasma*

Mycoplasma genitalium, Mycoplasma hominis, Ureaplasma urealyticum

Anaerobic Bacteria

Bacteroides species, Peptostreptococcus, Prevotella bivia, Leptotrichia sanguinegens/amnionii, Atopobium vaginae

Aerobic Bacteria

Gardnerella vaginalis, Haemophilus influenzae, Streptococcus agalactiae, Escherichia coli, and other gram-negative rods, *Actinomyces israelii, Campylobacter fetus*

Bacterial vaginosis (BV) is frequently identified in women with PID, and the type of BV-associated microorganism (*Gardnerella vaginalis, Mycoplasma hominis, Ureaplasma urealyticum*, pigmented or nonpigmented anaerobic gram-negative rods) may make a difference in the likelihood of developing PID.[4,5]

Infection with ***Trichomonas vaginalis*** is associated with a fourfold increase in the incidence of acute endometritis. Co-infection with **herpes simplex virus 2** and ***C. trachomatis*, *N. gonorrhoeae*,** or bacteria causing vaginosis is also associated with acute endometritis. Infection with herpes simplex virus 2 causes fallopian tube inflammation and lower tract ulceration that may disrupt the endocervical canal mucous barrier.[6] **Human immunodeficiency virus 1 (HIV-1)** infection is associated with an increased incidence of *C. trachomatis* infection, increased incidence of co-infection with *Candida* and human papillomavirus, and increased risk of progression to PID.[7]

PID may result from ***Mycobacterium tuberculosis*** infection in endemic areas.[8] **Schistosomes** can cause genital infection, including a PID-like tubal infection, infertility, and chronic abortion, and a recent report links schistosomiasis to HIV transmission in Africa. ***Actinomyces*** species have been identified almost exclusively in patients with intrauterine devices (IUDs).[9]

ASCENDING INFECTION

Most cases of PID are presumed to originate with sexually transmitted infections (STIs) of the lower genital tract, followed by ascending infection of the upper tract. The original STI may be asymptomatic. The precise mechanisms by which upper genital tract infection and inflammation are initiated and propagated are not well known. Although the cervical mucus serves as a functional barrier to ascending infection much of the time, its efficacy may be decreased by hormonal changes during menstruation and ovulation and by retrograde menstruation. Intercourse may contribute to the ascent of infection due to rhythmic mechanical uterine contractions. Bacteria also may be carried by, or along with, sperm into the uterus and tubes. Uterine infection usually is limited to the endometrium but can be more invasive in a gravid or postpartum uterus. Tubal infection initially affects only the mucosa, but acute, complement-mediated transmural inflammation may develop rapidly and increase in intensity with repeated infection. Inflammation may extend to uninfected parametrial structures, including the appendix and bowel. Infection may spread by direct extension of purulent material from the fallopian tubes or via lymphatic spread beyond the pelvis to involve the hepatic capsule with acute perihepatitis (Fitz-Hugh–Curtis syndrome) and produce acute peritonitis.

RISK FACTORS FOR PID

Multiple risk factors are associated with development of PID (**Table 103-2**).[4,10,11,12,13,14,15,16,17]

TABLE 103-2

Risk Factors Associated with Pelvic Inflammatory Disease[11,12,13,14,15,16,17]

Multiple sexual partners
History of sexually transmitted infection or pelvic inflammatory disease
History of sexual abuse
Frequent vaginal douching
Intrauterine device insertion within previous month
Adolescence, younger adulthood
Lower socioeconomic status
Postabortal

IUD use has been associated with an increased risk for PID. Although the majority of risk occurs within 21 days of insertion, the presence of an IUD is associated with complicated PID irrespective of the duration of use.[9,13,14,15] The risk of PID in IUD users is more related to the development of STI than the IUD,[18,19] and STI screening and treatment at the time of insertion can significantly decrease the likelihood that PID will develop.[20]

Pregnancy decreases the risk of PID because the cervical os is protected by the mucous plug. However, PID can occur during the first trimester and is associated with substantial fetal loss and preterm delivery.

COMPLICATIONS OF PID

PID is associated with a number of serious clinical sequelae. Tubo-ovarian abscess is reported in up to one third of women hospitalized for PID. Infection and inflammation can lead to scarring and adhesions within tubal lumens. **Ectopic pregnancy is more frequent in women who have had PID than in those who have never had an ectopic pregnancy.** Tubal factor infertility is increased by 12% to 50% in women with a past diagnosis of PID, and the incidence increases with the number and severity of past PID episodes.[21] Asymptomatic or silent PID appears to be associated with tubal factor infertility as well. Sequelae of PID include recurrence of PID, chronic pelvic pain, menstrual disturbances, and chronic dyspareunia. Recurrence of PID may occur because of inadequately treated infection, nontreatment of partner(s), or reinfection from another sexual contact. In follow-up to the Pelvic Inflammatory Disease Evaluation and Clinical Health trial, those with recurrence of PID were five times more likely to experience chronic pelvic pain.[22] PID may also be associated with an increased risk of ovarian borderline tumors.[23]

CLINICAL FEATURES

The clinical presentation of PID is variable. The most common presenting complaint is lower abdominal pain, most frequently described as bilateral and dull or crampy. Pain may be exacerbated by movement or by sexual activity. Other symptoms include abnormal vaginal discharge (75% of individuals), vaginal and postcoital bleeding (more than one third of patients), irritative voiding symptoms, fever, malaise, nausea, and vomiting.[24] Symptoms occur most commonly early in the menstrual cycle or at the end of the menses and are attributed to low progesterone levels and coincident thinning of the cervical mucosal barrier.

The physical examination is usually notable for lower abdominal tenderness, cervical motion tenderness, and uterine or adnexal tenderness. Involuntary guarding and rebound tenderness may be present and may indicate associated peritonitis. The positive predictive value of these findings varies depending on the prevalence of PID in a given clinical population. Adnexal tenderness appears to have a sensitivity of 95%.[25] Mucopurulent cervicitis is a common finding, and its absence should raise consideration of another diagnosis. In women who are suspected of having PID and for whom there is no likely alternative diagnosis for abdominal pain, the presence of fever, adnexal tenderness, and an elevated erythrocyte sedimentation rate are significant independent predictors of endometritis and correctly classify 65% of patients with laparoscopically proven PID (95% confidence interval, 61% to 99%).[25,26]

Right upper quadrant tenderness, particularly with jaundice, may indicate perihepatic inflammation. **Fitz-Hugh–Curtis syndrome** is perihepatitis, demonstrated by right upper quadrant pain in a woman with a clinical diagnosis of PID and no other cause for this pain. It is an uncommon complication and responds to standard antibiotic treatment for PID.[27]

The differential diagnosis of PID is broad and includes cervicitis, ectopic pregnancy, endometriosis, ovarian cyst, ovarian torsion, spontaneous abortion, septic abortion, cholecystitis, gastroenteritis, appendicitis, diverticulitis, pyelonephritis, and renal colic. Look for signs of other STIs, such as herpes simplex, syphilis, and human papillomavirus infection.

DIAGNOSIS

The diagnosis is based on history and clinical findings. No single piece of historical, physical, or laboratory information is sensitive and specific for the disease. **Laboratory evaluation of any woman of childbearing age in the ED always should include a pregnancy test.** Consider the possibility of ectopic pregnancy or septic abortion; the most common alternative diagnosis in missed ectopic pregnancy is PID. Concurrent pregnancy also influences patient treatment and disposition.

Current Centers for Disease Control and Prevention guidelines encourage initiation of empiric treatment in women at risk for PID who exhibit lower abdominal pain, adnexal tenderness, and cervical motion tenderness. Guidelines stratify diagnostic criteria into the three groups shown in Table 103-3.

TABLE 103-3

Diagnostic Criteria for Pelvic Inflammatory Disease (PID)

Group 1: Minimum criteria. Empiric treatment if no other cause to explain findings.
Uterine or adnexal tenderness
Cervical motion tenderness
Group 2: Additional criteria improving diagnostic specificity.
Oral temperature >101°F (38.3°C)
Abnormal cervical or vaginal mucopurulent secretions
Elevated erythrocyte sedimentation rate
Elevated C-reactive protein level
Laboratory evidence of cervical infection with *Neisseria gonorrhoeae* or *Chlamydia trachomatis* (i.e., culture or DNA probe techniques)
Group 3: Specific criteria for PID based on procedures that may be appropriate for some patients.
Laparoscopic confirmation
Transvaginal US (or MRI) showing thickened, fluid-filled tubes with or without free pelvic fluid or tubo-ovarian complex
Endometrial biopsy results showing endometritis

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep* 59(RR-12): 12, 2010.

LABORATORY TESTING

Obtain saline- and potassium hydroxide–treated wet preparations of vaginal secretions to identify leukorrhea (more than one polymorphonuclear leukocyte per epithelial cell) and trichomonads, and to test for BV, including clue cells, pH, and a whiff test. Leukorrhea is sensitive but not specific for upper tract infection,[3] and the absence of leukorrhea is a negative predictor for PID. Although endocervical swab specimens may be

sent for culture and can be gram stained for gonococci, nucleic acid amplification tests and DNA probes for *N. gonorrhoeae* and *Chlamydia* have replaced culture and gram staining in many settings. Unfortunately, these results are not available to the ED at the time of initial evaluation. Several sensitive and specific diagnostic tests are currently available for *Trichomonas* testing, including a nucleic acid amplification test (Aptima®; GenProbe, San Diego, CA), approved in 2013, that is performed on the same clinical samples as those for *Chlamydia* and gonorrhea testing.[29,30]

If PID is clinically suspected, an elevated WBC count, erythrocyte sedimentation rate, or C-reactive protein level supports the diagnosis.[28] Because a patient may have multiple STIs, also obtain a rapid plasma regain test for syphilis. Test for HIV and hepatitis. Urinalysis can exclude urinary tract infection, but a positive urinalysis does not exclude PID, because any inflammatory process in the contiguous pelvis can produce WBCs in the urine. Blood cultures do not aid in diagnosis or treatment.

IMAGING

Transvaginal pelvic US may demonstrate thickened (>5 mm), fluid-filled fallopian tubes or free pelvic fluid in acute severe PID. Pelvic or tubo-ovarian abscesses appear as complex adnexal masses with multiple internal echoes. Pelvic US can demonstrate as many as 70% of adnexal masses missed on physical examination. US also may be helpful in ruling in or out other causes in the differential diagnosis of pelvic pain, including ectopic pregnancy, ovarian torsion, hemorrhagic ovarian cyst, and possibly appendicitis or endometriosis.[31]

Abdominopelvic CT and MRI may also be used in the diagnosis of PID and the exclusion of other important causes of pelvic pain. If appendicitis or other surgical or GI diagnoses cannot be excluded, obtain an abdominopelvic CT. For further discussion, see chapter 97, "Abdominal and Pelvic Pain in the Nonpregnant Female." CT findings in PID include obscuration of the pelvic fascial planes, cervicitis, oophoritis, salpingitis, thickening of the uterosacral ligaments, and the presence of simple or complex pelvic fluid or abscess collections. MRI is especially helpful in characterizing complicated soft tissue masses, including dilated fallopian tubes and abscesses. MRI imaging is more specific and accurate than US to assess PID, with a sensitivity of 95% and a specificity of 89%.[32,33]

TREATMENT

Treatment is aimed at relieving acute symptoms, eradicating current infection, and minimizing the risk of long-term sequelae. From a public health perspective, another objective of treatment is to reduce the risk of transmission of infection to other new partners and to identify and treat past and current sexual partners to prevent disease spread. Early diagnosis and treatment are critical because duration of symptoms is an independent risk factor for infertility.

Due to the difficulty of diagnosis and the potential for serious sequelae, the Centers for Disease Control and Prevention recommend a low threshold for empiric treatment, with overtreatment preferred to a missed diagnosis with resultant delayed or no treatment.

Provide adequate analgesia, control of emesis and fever, and fluid replacement in those with nausea, vomiting, and dehydration and in those who appear toxic. Nonsteroidal anti-inflammatory drugs are very useful for the management of pain of pelvic origin. ED treatment should include empiric broad-spectrum antibiotic therapy to cover the full range of likely organisms. Screen for BV and treat when screening is positive. Treatment regimens should follow both national guidelines from the Centers for Disease Control and Prevention and local health department surveillance reports.

The Pelvic Inflammatory Disease Evaluation and Clinical Health trial, which included 654 females age 14 to 37 years old and excluded those who had been treated with antibiotics during the preceding 7 days, had experienced an abortion, delivery, or gynecologic surgery during the preceding 14 days, were homeless, or had an allergy to study medications, demonstrated no differences between oral and parenteral regimens in women with mild to moderately severe acute PID uncomplicated by pregnancy or the presence of a tubo-ovarian abscess.[34,35]

Currently accepted inpatient and outpatient treatment regimens are summarized in **Tables 103-4** and **103-5**. Current geographic patterns of drug resistance may change recommendations. Patients with PID who require IV antibiotics initially can be switched to oral antibiotics after clinical improvement.

TABLE 103-4

Parenteral Treatment Regimens for Pelvic Inflammatory Disease

Cefotetan, 2 grams IV every 12 h, or cefoxitin, 2 grams IV every 6 h

plus

Doxycycline, 100 milligrams PO or IV every 12 h*

or

Clindamycin, 900 milligrams IV every 8 h

plus

Gentamicin, 2 milligrams/kg IV or IM loading dose, followed by gentamicin, 1.5 milligrams/kg every 8 h maintenance dose†

Alternative Parenteral Regimen (limited data on effectiveness)

Ampicillin/sulbactam, 3 grams IV every 6 h

plus

Doxycycline, 100 milligrams PO or IV every 12 h*

*PO doxycycline has the same bioavailability as IV doxycycline and avoids painful infusion.

†Gentamicin dosing may be 3–5 milligrams/kg every 24 h.

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep 59*(RR-12): 12, 2010.

TABLE 103-5

Oral and Outpatient Treatment Regimens for Pelvic Inflammatory Disease

Ceftriaxone, 250 milligrams IM once, *or* cefoxitin, 2 grams IM once, *and* probenecid, 1 gram PO once administered concurrently
or
Other parenteral third-generation cephalosporin (e.g., ceftizoxime or cefotaxime)
plus
Doxycycline, 100 milligrams PO twice a day for 14 d
with or without
Metronidazole, 500 milligrams PO twice a day for 14 d
If parenteral cephalosporin therapy is not feasible and community prevalence of fluoroquinolone resistance is low:
Levofloxacin, 500 milligrams PO, *or* ofloxacin, 400 milligrams twice daily every day for 14 d
with or without
Metronidazole, 500 milligrams PO twice a day for 14 d

Note: Other parenteral third-generation cephalosporins can be substituted for ceftriaxone or cefoxitin. Since the Centers for Disease Control and Prevention guidelines were published in 2006, clinically significant resistance to the fluoroquinolones (6.7% of infections in heterosexual men, an 11-fold increase from 0.6% in 2001) has emerged in the United States. Fluoroquinolone antibiotics are no longer recommended to treat gonorrhea in the United States.[27] Fluoroquinolones may be an alternative treatment option for disseminated gonococcal infection if antimicrobial susceptibility can be documented.

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep* 59(RR-12): 12, 2010.

TREATMENT WITH IUD IN PLACE

In the past, IUDs were generally removed, based on the belief that because it is a foreign body, removal of the IUD would allow treatment to be more effective. There is a low risk of PID from IUD insertion, especially when STI testing is done concomitantly and immediate treatment is initiated.[20] **Current Centers for Disease Control and Prevention guidelines suggest that there is insufficient evidence to recommend IUD removal before treatment for PID, because the device is usually not the source of infection.** For individuals using IUD for birth control who develop PID,

there are no data to support the use of one treatment regimen over another. Close clinical follow-up is prudent. If there is a concern regarding PID in a patient with an IUD placed in the last 3 weeks, it is reasonable to consult a gynecologist regarding removal.

TREATMENT IN HIV INFECTION

Microbiologically, HIV-positive women are more likely to have concomitant *Candida*, *Mycoplasma hominis*, HPV, and streptococcal infection. HIV-positive women with PID may experience more severe symptoms irrespective of CD4 count and are more likely to have sonographically diagnosed tubo-ovarian abcess. However, they appear to respond similarly to treatment for uncomplicated PID as do women who are not infected with HIV.[36,37,38] HIV-positive status alone is not a criterion for hospitalization.[36,39] Although HIV status has been removed from specific admission considerations, the 2010 Centers for Disease Control and Prevention STI guidelines note that "whether the management of immunodeficient HIV-infected women with PID requires more aggressive interventions (e.g. hospitalization or parenteral antimicrobial regimens) has not been determined."[40]

TREATMENT OF ADOLESCENTS

Several studies have raised additional concerns about the outpatient management of early adolescents. Early and mid-adolescents were not well represented in the Pelvic Inflammatory Disease Evaluation and Clinical Health study, and of those enrolled, adolescents had increased risk of recurrent PID and a shorter time to pregnancy after an acute episode compared with adult enrollees. Adolescents hospitalized in pediatric centers often receive services beyond IV antibiotics, including education on risk reduction, emotional support, social work intervention, assistance with communicating the nature of their illness with parents, and assistance to arrange close follow-up.[41,42]

ALTERNATIVE ANTIBIOTICS

For those with severe cephalosporin allergy, **spectinomycin** is recommended in Canada and Europe but is not currently available in the United States. For more information, see the Centers for Disease Control and Prevention Web pages on antibiotic-resistant gonorrhea at http://www.cdc.gov.une.idm.oclc.org/std/Gonorrhea/arg/default.htm.[28]

Multiple studies have demonstrated poor compliance with doxycycline therapy (25%; 50% with partial compliance), and 20% to 25% of patients never fill their prescriptions.[43,44,45] Recently, generic doxycycline has been difficult to find due to manufacturing shortages and, when available, may be quite expensive, resulting in patient noncompliance with treatment. For PID treatment, **azithromycin** is an alternative, with dosing as either 250 milligrams PO once a day for 7 days or 1 gram once a week for 2 weeks.[43] The long half-life of azithromycin requires significantly fewer

doses, which is thought to improve the likelihood of patient compliance. Azithromycin also provides intrinsic anti-inflammatory effects and may reduce local tissue damage. Weigh these potential benefits against the lack of large-scale or long-term studies comparing the effectiveness of azithromycin to doxycycline in the treatment of PID and the possibility of emerging resistance to azithromycin.[43,46,47,48,49]

TUBO-OVARIAN ABSCESS

Disproportionate unilateral adnexal tenderness or adnexal mass or fullness may indicate a tubo-ovarian abscess. In women with clinical toxicity and asymmetric pelvic findings, obtain a pelvic US. Most tubo-ovarian abscesses (60% to 80%) resolve with antibiotic administration alone.[50,51,52] In the setting of tubo-ovarian abscess, oral therapy should be continued with clindamycin (450 milligrams PO four times per day) or metronidazole with doxycycline for better anaerobe coverage for 14 days. Patients who do not improve after 72 hours of treatment should be reevaluated for possible CT- or US-guided percutaneous drainage, laparoscopic drainage, posterior colpotomy with drainage, surgical intervention, or reconsideration of other possible diagnoses. Abscesses 9 cm or larger on imaging appear to have a higher likelihood of requiring surgical therapy. An enlarging pelvic mass may indicate bleeding secondary to vessel erosion or a ruptured abscess.

DISPOSITION AND FOLLOW-UP

Guidelines for admission (**Table 103-6**) and inpatient treatment (Table 103-4) have evolved over the past decade. There are no data demonstrating that inpatient treatment is more effective than outpatient treatment. Among the problems encountered with outpatient care are the provision of adequate guideline-driven treatment, patient adherence to the prescribed therapeutic regimen, difficulty in arranging outpatient administration of parenteral medications, and coordination of 72-hour follow-up evaluation, all of which have been implicated as causes of treatment failure. Consider these and other constraints when determining the patient's ability to follow or tolerate an outpatient regimen.

TABLE 103-6

Admission Considerations

Inability to exclude surgical emergency from the differential diagnosis
Pregnancy
Failure to respond to outpatient treatment
Inability to tolerate or comply with outpatient treatment
Severe toxicity, high fever, nausea, vomiting
Tubo-ovarian abscess

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. MMWR Recomm Rep 59(RR-12): 12, 2010.

Institutions should consider adoption of protocolized treatment guidelines to help to ensure fidelity to standards of care. **Admission decisions in the ED are based on severity of illness, likelihood of adherence to outpatient medication regimen, likelihood of major anaerobic infection (IUD, suspected pelvic or tubo-ovarian abscess, or history of recent uterine instrumentation), certainty of diagnosis, coexisting illness and immunosuppression, pregnancy, patient age, and other major fertility issues.**

If the patient is discharged, arrange reevaluation within 72 hours for clinical improvement and adherence to the prescribed regimen. Encourage partner evaluation and treatment. Test and treat for other STIs if not already done. Educate patients about the use of barrier contraceptives and other "safe sex" techniques to lessen the risk of reinfection. Counsel the patient to remain abstinent from sexual activity until 1 week after treatment is finished for both the patient and partner and symptoms have abated.

Partner treatment is crucial to preventing repeated episodes of PID. This can be difficult to ensure. If the current partner has accompanied the patient to the ED, and the patient is willing to tell this partner about her infection, she can be asked to suggest immediate ED evaluation to her partner. If not, the patient should be instructed to notify partners with whom she has had sexual contact in the 60 days preceding the onset of her symptoms to go to the local public health department or STI clinic for empiric treatment of *N. gonorrhoeae* and *C. trachomatis*. A 6-minute PID outreach video has been developed and was found in one randomized controlled trial to improve partner treatment.[53]

A Loading [Contrib]/a11y/accessibility-menu.js lly acknowledge the contributions of Amy Behrman, a coauthor of this chapter in the previous edition.

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USEFUL WEB RESOURCES

World Health Organization, Information on sexually transmitted infections—http://www.who.org and http://www.who.int.une.idm.oclc.org/mediacentre/factsheets/fs110/en/

Centers for Disease Control and Prevention Treatment Guidelines—http://www.cdc.gov.une.idm.oclc.org/std/treatment/