

Pelvic Inflammatory Disease: Diagnosis And Treatment In The Emergency Department

Abstract

Pelvic inflammatory disease is a common disease that is associated with significant complications including infertility, chronic pelvic pain, ruptured tubo-ovarian abscess, and ectopic pregnancy. The diagnosis may be delayed when the presentation has nonspecific signs and symptoms. Even when it is properly identified, pelvic inflammatory disease is often treated suboptimally. This review provides evidence-based recommendations for the diagnosis, treatment, disposition, and follow-up of patients with pelvic inflammatory disease. Arranging follow-up of patients within 48 to 72 hours and providing clear patient education are fundamental to ensuring good patient outcomes. Emerging issues, including new pathogens and evolving resistance patterns among pelvic inflammatory disease pathogens are reviewed.

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CME Objectives

Upon completion of this article, you should be able to:

1. List the diagnostic criteria for PID and discuss the limitations and pitfalls associated with the use of imaging and testing.
2. Explain the empiric treatment options for PID.
3. Describe the emerging pathogens, antibiotic resistance patterns, and the effects on treatment of PID

Prior to beginning this activity, see "Physician CME Information" on the back page.

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Case Presentations

You arrive for your shift in the ED. The final patient you are signed out is a 30-year-old woman with lower abdominal pain whose ultrasound results are pending to rule out torsion versus ovarian cyst. You nod dutifully and go about seeing new patients. An hour into the shift, the clerk hands you the ultrasound results with the radiologist's impression: "No radiological etiology of patient's abdominal pain is found." You review the chart and confirm that there is no concern for any nongynecological etiologies for her pain. The previous physician documented mild left adnexal tenderness without cervical motion tenderness or adnexal masses. Labs are notable for a urinalysis that is small leukocyte esterase positive and nitrite negative, and a wet mount without clue cells, yeast, or *Trichomonas vaginalis*. You confirm the documented history with the patient, who additionally denies any urinary complaints or flank pain. On your physical examination, you note only mild left lower abdominal tenderness. As the patient asks, "Why am I having this pain? Can I just go home?" you wonder if there is something else you should do.

A 22-year-old woman returns for re-evaluation 1 week after starting treatment for pelvic inflammatory disease. She does not have access to primary care and was instructed to return to the ED for repeat evaluation. She was supposed to return to the ED after 2 days, but could not because of work. She continues to complain of nonspecific left lower abdominal pain. She states that the pain may be a bit more intense, but it has not changed in quality, position, or associated features. On your physical examination, the patient has left lower quadrant abdominal tenderness without guarding or rebound. Bimanual examination reveals only mild left adnexal tenderness without a palpable mass. She states that she has been fully compliant with the doxycycline and has not had intercourse since her diagnosis. Her previous records show a negative pelvic ultrasound, urinalysis, urine culture, and HIV test. You are surprised to find that her gonorrhea/chlamydia nucleic acid amplification test from a cervical specimen showed no evidence of infection. After being told about her negative gonorrhea and chlamydia tests, she asks if she can stop taking the antibiotics...

Introduction

Pelvic inflammatory disease (PID) is an inflammatory disease of the upper female reproductive system that is caused by an ascending infection. It is characterized by inflammation and tenderness of the uterus, cervix, and adnexa. PID is common and costly, with a yearly incidence of 750,000 to 800,000 cases and \$2 billion in annual direct costs in the United States.^{1,2} The majority of patients with PID present with mild-to-moderately severe disease and are managed as outpatients.³ Only a small percentage of patients progress to severe or complicated illness.⁴ Although the rate of direct morbidity and

mortality is low, treatment prevents subsequent infertility, pelvic scarring, chronic pelvic pain, and ectopic pregnancy.⁵

PID can be a difficult and frustrating diagnosis; patients commonly present with nonspecific symptoms such as vaginal discharge, postcoital bleeding, dyspareunia, and dysuria.⁶ There is no single historical, laboratory, physical examination finding, or imaging modality that provides adequate sensitivity or specificity for the diagnosis.⁷⁻¹⁰

The United States Centers for Disease Control and Prevention (CDC) recommend that clinicians make the clinical diagnosis of PID and start empiric treatment in sexually active women with unexplained lower abdominal or pelvic pain with:

- Cervical motion tenderness, **or**
- Uterine tenderness, **or**
- Adnexal tenderness.

There are no requirements for any specific laboratory findings, physiological parameters, or imaging.¹¹ While this definition may seem overly broad, it has a sensitivity of > 95% and a specificity of 75% and reflects the need to minimize the rates of misdiagnosis and prevent the resulting impact on fertility.⁸ This issue of *Emergency Medicine Practice* presents a review of the current evidence and best-practice guidelines of the evaluation and treatment of PID.

Critical Appraisal Of The Literature

A literature search was performed using PubMed, with the search terms *pelvic inflammatory disease, endometritis, salpingitis, oophoritis, and tubo-ovarian abscess*. The search included clinical trials, systematic reviews, review articles, and clinical guidelines. A review of the Cochrane Database of Systematic Reviews revealed no relevant reviews. The National Guideline Clearinghouse (www.guideline.gov) noted 3 guidelines:

- CDC: Sexually Transmitted Diseases Treatment Guidelines 2015¹¹
- British Association for Sexual Health and HIV: United Kingdom National Guideline for the Management of Pelvic Inflammatory Disease 2011¹²
- American College of Radiology (ACR): ACR Appropriateness Criteria[®] Acute Pelvic Pain in the Reproductive Age Group¹³

References from articles were examined to ensure accurate representation of the literature. The recommendations for the first-line treatment, management, and diagnostic evaluation are based on multiple well-performed studies with large sample sizes that looked at both short- and long-term outcomes; however, the bulk of the remaining

literature suffers from sampling bias toward sicker patients, which affects generalizability of findings, and focuses on shorter-term outcomes instead of the longer-term outcomes that comprise the bulk of the morbidity associated with PID. These biases make it difficult to make high-level recommendations regarding alternative treatments and management.

Etiology And Pathophysiology

Pelvic inflammatory disease refers to a group of inflammatory disorders of the female upper genital tract comprising endometritis, salpingitis, and oophoritis.¹¹ It is caused primarily by an infection that spreads from the vagina or cervix to the upper genital tract, and is most common in sexually active women under the age of 30.^{1,14} It can be complicated by peritonitis, pyosalpinx, tubo-ovarian abscess (TOA), and perihepatitis (ie, Fitz-Hugh-Curtis syndrome). Long-term complications include ectopic pregnancy, infertility, chronic pelvic pain, and recurrent infection.

Classically, PID is attributed to an untreated sexually transmitted infection (STI) of the lower genital tract due to *Neisseria gonorrhoeae* or *Chlamydia trachomatis*. *N gonorrhoeae* and *C trachomatis* were previously associated with up to 80% of PID.¹⁵ Although the rates of *N gonorrhoeae*- and *C trachomatis*-associated PID have remained high in younger and lower-income patients, recent studies have reported the rate of *N gonorrhoeae*- and *C trachomatis*-associated PID to be as low as 15%.¹⁶ The decrease in the rate of *N gonorrhoeae* and *C trachomatis* infection is associated with a reciprocal increase in the role of alternate pathogens, eg, *Mycoplasma genitalium*^{17,18} and non-STI pathogens, including vaginal flora, respiratory and enteric pathogens, and viruses.¹⁹⁻²² (See **Table 1.**) It is unclear from the available data whether this trend reflects a decreasing number of gonorrhea/chlamydia cases or if it reflects higher rates of non-gonorrhea/chlamydia diagnoses.

Further emphasizing the fact that PID is not caused exclusively by STIs, it has been reported in patients who have never been sexually active. These cases are due primarily to *Escherichia coli*, which is thought to spread hematogenously or through translocation from the bowel. These patients most commonly present with complicated PID (eg, TOA).^{23,24}

There is an association between bacterial vaginosis and PID.²⁵ It is unclear whether this is primarily due to a synergy between the bacterial vaginosis bacteria and the STIs or if it is due to direct infection of the upper genital tract by these bacteria. Several bacterial vaginosis bacteria have been shown to destroy the cervical mucosa, making the patient prone to ascending infection from STIs.²⁶

Although the majority of patients with PID

have mild-to-moderately severe disease, a small percentage of patients have progressive or complicated disease. Fitz-Hugh-Curtis syndrome occurs when the ascending organism inflames the liver capsule, and it has been reported in 4% to 6% of patients with PID.^{27,28}

The most serious complication of PID is TOA, which occurs in 3% to 16% of North American patients hospitalized with PID.⁴ Organisms most commonly isolated include *E coli* and *Bacteroides*, *Peptostreptococcus*, *Peptococcus*, and aerobic *Streptococcus* species.^{29,30} Though uncommonly isolated from most patients, patients with HIV appear to be at higher risk for TOA due to co-infection with *N gonorrhoeae* or *C trachomatis*.³¹ Long-term intrauterine device (IUD) use has also been associated with TOA, especially due to *Actinomyces* species.³⁰

Differential Diagnosis

PID typically presents with nonspecific signs and symptoms, which leads to a great deal of overlap with diseases of the gastrointestinal, genitourinary,

Table 1. Pathogens Associated With Pelvic Inflammatory Disease¹¹

Sexually Transmitted Bacteria

Neisseria gonorrhoeae
Chlamydia trachomatis
Mycoplasma genitalium

Bacterial Vaginosis Bacteria

Mycoplasma hominis
Ureaplasma urealyticum
Porphyromonas spp
Prevotella spp
Bacteroides spp
Peptostreptococcus spp
Leptotrichia spp
Atopobium vaginae
Gardnerella vaginalis
Clostridium spp
Diphtheroids

Respiratory Bacteria

Haemophilus influenzae
Streptococcus pneumoniae
Group A streptococci
Staphylococcus aureus

Enteric Bacteria

Escherichia coli
Bacteroides spp
Campylobacter spp
Enterobacteriaceae
Salmonella spp

Viruses

Cytomegalovirus spp
Herpes simplex virus type 2

gynecological, and obstetrical systems. (See Table 2.) Because of the wide differential diagnosis and the lack of pathognomonic findings on history or physical examination, it can be a challenge to narrow the differential to a single organ system. Due to this overlap, the highest priority is often the exclusion of alternative diagnoses—such as ectopic pregnancy and appendicitis—which carry higher rates of morbidity and mortality.

Prehospital Care

Because patients with PID are not commonly severely ill, there are no specific prehospital implications.

Emergency Department Evaluation

History

Historical findings that should prompt the emergency clinician to consider PID include abdominal pain, pelvic pain, low back pain, vaginal discharge, postcoital bleeding, intermenstrual bleeding, dyspareunia, or urinary symptoms, especially in a sexually active woman.³²⁻³⁵ A report of pleuritic right upper quadrant pain³⁶ or right scapular pain may indicate the presence of Fitz-Hugh-Curtis syndrome, while left upper quadrant pain can be suggestive of perisplenitis.³⁷ Systemic signs such as nausea, vomiting, chills, and fever are not typically seen and are concerning for complicated PID.^{30,38,39}

For the past medical history, HIV status, previous STIs, and history of endometriosis should be determined. Nine percent of women who have been recently treated for gonorrhea or chlamydia go on to develop PID, most commonly in the first 45 days after treatment.⁴⁰ Previous STI treatment should alert the clinician about the possibility of infection with antibiotic-resistant organisms.^{41,42} HIV-positive patients (especially those with a CD4 T lymphocyte count < 400 cells/mm³) may have an increased risk for both acquiring PID and developing complications (eg, TOA).^{31,43} Women with endometriosis may have a longer and more severe disease course, as

Table 2. Differential Diagnosis Of A Patient With Potential Pelvic Inflammatory Disease

| Organ System | Differential Diagnosis |
|------------------|--|
| Gastrointestinal | Appendicitis, diverticulitis, colitis, gastroenteritis, cholecystitis (in Fitz-Hugh-Curtis syndrome) |
| Genitourinary | Renal colic, urinary tract infection, cystitis |
| Musculoskeletal | Musculoskeletal strain, contusion |
| Gynecological | Ovarian cyst, ovarian torsion, menstrual cramps, fibroids, mittelschmerz, bacterial vaginosis, cervicitis, endometriosis |
| Obstetrical | Ectopic pregnancy |

well as an increased risk of treatment failure.^{44,45}

A thorough social history can help to evaluate the patient's risk for having PID. A greater number of sexual partners, inconsistent use of condoms, and vaginal douching increase the risk of acquiring PID.^{46,47} Although IUDs had previously been linked to an increase in PID,^{15,48} modern devices do not confer any increased risk of acquiring PID.^{49,50} Women who have sex with men who have sex with men are at increased risk for infection with *M genitalium*, as well as resistant *N gonorrhoeae* and *C trachomatis*.^{51,52} Additionally, smoking, drug and alcohol abuse, and mental health issues increase risk for PID and may represent barriers to treatment or follow-up.⁵³⁻⁵⁷ Because of the association between PID and intimate partner violence and rape, patients should also be screened for domestic violence and sexual assault.^{58,59}

Physical Examination

The physical examination of the patient is directed at both evaluating for PID and its complications and excluding other diagnoses in the differential. There are 4 essential components of the physical examination:

1. Checking vital signs for fever, tachycardia, or hypotension
2. Abdominal examination, including right upper quadrant and flanks, to evaluate for signs of perihepatitis and/or alternative diagnoses
3. Bimanual pelvic examination for cervical motion tenderness, uterine tenderness, or adnexal tenderness
4. Vaginal speculum examination for cervical discharge and cervical friability

Vital sign abnormalities are not commonly seen in uncomplicated PID. Fever and tachycardia should alert the emergency clinician to consider pyosalpinx, TOA, or peritonitis.⁶⁰⁻⁶² Hypotension is rare and should be an alert to the possibility of a ruptured TOA and should prompt aggressive resuscitation as well as consideration of surgical consultation.^{63,64}

The presence of lower abdominal tenderness has a sensitivity of 94% for identifying patients with PID.⁶⁵ Right upper quadrant tenderness may signal the possibility of Fitz-Hugh-Curtis syndrome, which can also present with right rib tenderness, right liver tenderness, hepatomegaly,⁶⁶ friction rub over the anterior right costal margin,⁶⁷ or localized peritonitis.^{36,66} Similarly, left upper quadrant tenderness may indicate perisplenitis, which is also a manifestation of Fitz-Hugh-Curtis syndrome.³⁷

Cervical motion tenderness, uterine tenderness, and adnexal tenderness have been found to have similar sensitivities (92%-96%) for identifying acute PID.^{32,60} Any pelvic organ tenderness has a sensitivity of 99%, and any lower abdominal tenderness has a sensitivity of 94%.⁶⁵ In addition to tenderness, up

to two-thirds of patients with a pyosalpinx or a TOA have a palpable adnexal mass.³⁹ In contrast to these studies, in a retrospective study of ED patients with TOA, fewer than half of the patients with PID had cervical motion tenderness or adnexal tenderness, but all had lower abdominal tenderness.⁶⁸

During the speculum examination, the emergency clinician should look for signs of PID, including yellow mucopurulent cervical discharge and cervical friability. Cervical friability is present when a cotton swab inserted into the cervical os easily elicits bleeding.⁶⁹ While performing the speculum examination, samples should be obtained for wet mount with saline microscopy and for *N gonorrhoeae/C trachomatis* testing.

Diagnostic Studies

Reflecting the finding that no laboratory or imaging study has a sensitivity or specificity for ruling in or ruling out the diagnosis of PID, the CDC diagnostic criteria for PID are not based on laboratory testing or imaging studies.¹¹ However, there are several tests that should be routinely ordered for patients suspected of having PID. (See Table 3.)

Laboratory Testing

A pregnancy test, a *N gonorrhoeae/C trachomatis* nucleic acid amplification test (NAAT), and a vaginal wet mount should be routinely sent in a patient with potential PID. These tests are directed at either identifying an associated pathogen or identifying signs of lower genital tract inflammation. Although not definitive, these tests can modify your suspicion of PID.

N gonorrhoeae/C trachomatis NAAT is highly sensitive and reliable for the identification of gonorrhea and chlamydia. This test can be used on samples obtained from the cervix, vagina, or first-void urine. Although there had previously been concern about

Table 3. Diagnostic Studies For Pelvic Inflammatory Disease

| Test | Recommendation for Use |
|---|--------------------------------------|
| <i>Neisseria gonorrhoeae/Chlamydia trachomatis</i> NAAT, wet mount with saline microscopy, pregnancy test | Send routinely on all patients |
| HIV, RPR/VDRL test | Consider strongly |
| Urinalysis, hepatic panel, vaginal culture | Order based on clinical presentation |
| ESR, CRP, CBC | Order on a patient-by-patient basis |

Abbreviations: CBC, complete blood cell count; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; NAAT, nucleic acid amplification test; RPR, rapid plasma reagin; VDRL, Venereal Disease Research Laboratory (syphilis).

low sensitivities of female urine samples using NAAT assays for *C trachomatis*,⁷⁰⁻⁷² currently, *N gonorrhoeae/C trachomatis* NAAT on a first-void urine sample appears to be equivalent to cervical or vaginal swabs for *N gonorrhoeae* and *C trachomatis*.⁷²⁻⁷⁶ Additionally, self-administered vaginal swabs have sensitivities similar to physician-collected samples.⁷⁷

The wet mount is used to detect leukorrhea, bacterial vaginosis, and trichomoniasis. The presence of leukorrhea or cervical mucus has a reported sensitivity of up to 96% for endometritis.⁶⁵ However, a recent systematic review concluded that leukorrhea is not a particularly helpful finding in confirming or ruling out PID.^{33,78} In contrast to the *N gonorrhoeae/C trachomatis* NAAT, self-obtained wet mount samples perform poorly, necessitating a physician-obtained sample.⁷⁹

Patients who have had recent antibiotic treatment for gonorrhea or chlamydia should have a cervical culture collected in addition to a *N gonorrhoeae/C trachomatis* NAAT, so that an antibiotic-resistant organism can be identified. Bacteriostatic lubricants commonly used during speculum and bimanual examinations do not decrease detection of cervical pathogens by polymerase chain reaction, but may affect the yield of culture specimens.⁸⁰⁻⁸²

Urinalysis is commonly sent in these patients. An abnormal urinalysis is common in PID, and it neither predicts a culture-proven urinary tract infection, nor does it rule out STI.^{83,84} Therefore, urinalysis should be interpreted within the context of the patient's presentation.

HIV testing should be routinely offered to any patient presenting with a possible STI. Screening for syphilis with a rapid plasma reagin (RPR) or Venereal Disease Research Laboratory (VDRL) test, especially in areas or populations with a high prevalence, is equally important. Per CDC guidelines, once a patient has been diagnosed with acute PID, *N gonorrhoeae/C trachomatis* NAAT and HIV should be sent, if it has not already been done as part of the initial workup.¹¹

C-reactive protein, erythrocyte sedimentation rate, and leukocytosis are nonspecific markers of inflammation and have relatively poor operating characteristics in PID.^{65,85,86} Although these tests lack sufficient sensitivity, significant leukocytosis or elevation of inflammatory markers is suggestive of complicated PID.^{39,87} Similarly, liver function tests may play a role in the patient suspected of having Fitz-Hugh-Curtis syndrome, although they are neither sensitive nor specific.^{66,88}

Imaging

Imaging is not routinely needed to diagnose or manage PID. Decisions about imaging should be done on a case-by-case basis. Imaging is typically directed at either identifying complications, such as TOA, or

evaluating alternative diagnoses.

The ACR states that the first-line imaging studies for the evaluation of acute pelvic pain in the nonpregnant female are transabdominal sonography and transvaginal sonography with Doppler as an adjunct.¹³ Although transabdominal sonography and transvaginal sonography have limited ability to rule out acute PID, even when performed by ultrasound experts, they have the advantage of being able to evaluate all of the pelvic structures.⁸⁹ Ultrasound has only modest sensitivity for the diagnosis of PID, and most patients with mild disease have normal transvaginal sonography. Despite the lack of sensitivity, there are some ultrasound findings that are specific for PID.⁹⁰ (See Table 4.)

Findings with a positive likelihood ratio (LR) > 4 include thick tubal walls (LR, 10) and the cogwheel sign (LR, 16).⁹² (See Figures 1 and 2.) Other findings described in PID, such as incomplete septa, polycystic ovaries, bilateral adnexal masses, or the presence of free fluid are not helpful in differentiating women with PID from those without PID. A hydrosalpinx is more commonly a consequence of a past episode of PID or chronic PID and is not a sign of acute disease.

The ACR recommends magnetic resonance imaging (MRI) and computed tomography (CT) as second-line imaging modalities that should be considered when the ultrasound imaging is inconclusive or nondiagnostic. CT is the imaging modality of choice when there is suspicion for nongynecological etiologies. (See Table 5, page 7.)

CT, like ultrasound, has poor-to-modest sensitivity for the identification of mild-to-moderately severe PID.⁹⁴ Mid-pelvic fat stranding is the most sensitive finding (sensitivity, 60%). Tubal thickening has a specificity > 90% and an odds ratio of 10.5 for PID. Signs of PID on MRI are similar to those on CT.¹⁰ MRI provides superior resolution compared to CT, and is exquisitely sensitive (91%-98%) and highly specific (81%-95%).^{10,95}

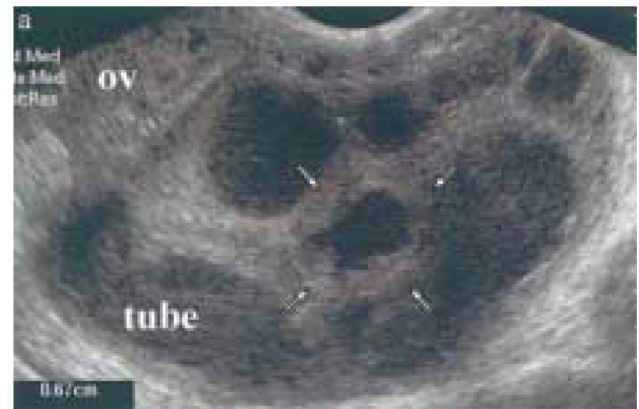
Although laparoscopy has long been considered the gold standard for establishing the diagnosis of PID, it is invasive and not feasible in resource-poor settings. Additionally, recent studies have found that

Figure 1. Thickened Tubal Walls



Molander P, Sjöberg J, Paavonen J, Cacciatore B. Transvaginal Power Doppler Findings in Laparoscopically Proven Acute Pelvic Inflammatory Disease. *Ultrasound in Obstetrics and Gynaecology*. 2001;17(3):233-238. With permission of John Wiley and Sons.

Figure 2. The Cogwheel Sign



Molander P, Sjöberg J, Paavonen J, Cacciatore B. Transvaginal Power Doppler Findings in Laparoscopically Proven Acute Pelvic Inflammatory Disease. *Ultrasound in Obstetrics and Gynaecology*. 2001;17(3):233-238. With permission of John Wiley and Sons.

Table 4. Ultrasound Findings In Pelvic Inflammatory Disease^{90,91}

| Finding | Description | Sensitivity | Specificity |
|-------------------------------------|---|-------------|-------------|
| Thickened tubal walls | Tubal walls > 5 mm thick, or in the sonographer's judgment | 29%-100% | 90%-100% |
| Cogwheel sign | Sonolucent cogwheel-shaped structure visible in the cross-section of the tube with thick walls | 0%-86% | 95%-97% |
| Tubo-ovarian complex | Ovaries and tubes are identified and recognized, but the ovaries cannot be separated by pushing the tube with the vaginal probe | 15%-36% | 98%-100% |
| Tubo-ovarian abscess | Formation of a conglomeration in which neither the ovary nor the tubes can be separately recognized as such | 25%-30% | 78%-100% |
| Incomplete septa | Hyperechoic septa protruding into a fluid-filled fallopian tube | 60%-86% | 7%-15% |
| Abnormal adnexal power flow Doppler | Hyperemia, lowered pulsatility indices | 100% | 80% |
| Cul-de-sac fluid | Pelvic free fluid | 37%-82% | 43%-90% |

it has poor interrater reliability, modest sensitivity, and will miss cases of endometritis and mild salpingitis.^{91,96,97} Endometrial biopsy has the advantage of being a less-invasive office-based procedure, with sensitivity comparable to laparoscopy and may be an option for some patients.^{22,65}

Treatment

The choice of treatment regimen depends on the severity of the illness; the patient's ability to tolerate oral medication; the presence of complications, allergies, and comorbidities; and the patient's ability to adhere to the medication regimen. The majority of patients with mild-to-moderately severe PID can be treated with outpatient oral therapy.^{1,3} Inpatient/intravenous therapy should be reserved for pregnant patients, patients with severe disease, patients unable to tolerate or comply with the oral regimen, or if there is uncertainty about the appropriateness of the oral regimen.

Mild-To-Moderately Severe Pelvic Inflammatory Disease

For patients with mild-to-moderately severe PID, the first-line treatment is an intramuscular (IM) injection of a cephalosporin and 2 weeks of 100 mg of oral doxycycline twice a day, with or without metronidazole.¹¹ (See Table 6.) The optimal cephalosporin has not yet been established. Either a third-generation cephalosporin (such as ceftriaxone) or a second-generation cephalosporin (such as cefoxitin) combined with a single dose of probenecid can be used. The CDC recommends a 250-mg IM dose, while the United Kingdom national guidelines recommend 500 mg.¹² Presently, the CDC does not have clear guidelines regarding the addition of metronidazole to the treatment regimen. For more information on the addition of metronidazole, see the discussion in the "Controversies And Cutting Edge" section, page 12.

Antibiotic Allergies

A penicillin allergy is not a contraindication to the use of cephalosporins in PID. Cross-reactivity between

Table 5. Computed Tomography Findings In Pelvic Inflammatory Disease⁹³

- Small amount of free fluid
- Dilated, fluid-filled endocervical and endometrial cavities
- Fluid-filled, dilated fallopian tubes (pyosalpinx)
- Complex fluid collections with thickened walls, septations, and fluid-debris levels or gas
- Thickening of the broad and uterosacral ligaments
- Loss of definition of the uterine border
- Pelvic-fat haziness
- Reactive lymph nodes
- Hepatic capsular enhancement (Fitz-Hugh-Curtis syndrome)

penicillins and cephalosporins is low, and virtually nonexistent for third-generation cephalosporins.⁹⁸ (See Table 7.)

In patients with a doxycycline allergy, consider azithromycin 500 mg IV (intravenously) daily for 1 to 2 doses, followed by azithromycin 250 mg PO (orally) daily for 12 to 14 days, with or without metronidazole 500 mg PO twice daily for 14 days.⁹⁹ Alternatively, give ceftriaxone 250 mg IM for 1 dose, with azithromycin 1 gram PO once per week for 2 weeks.¹⁰⁰ These 2 regimens using azithromycin have shown short-term outcomes equivalent to standard therapy.

Although therapy with fluoroquinolones has previously been shown to be effective for the treatment of PID, these regimens are not currently recommended because of the rise of fluoroquinolone-resistant *N gonorrhoeae*. Fluoroquinolone therapy should only be considered if the local rates of fluoroquinolone-resistant *N gonorrhoeae* are low and there are no other options due to patient allergies or local availability. All of the regimens combine 14 days of the quinolone (500 mg daily levofloxacin,

Table 6. Recommended Intramuscular/Oral Therapies For Mild-To-Moderately Severe Pelvic Inflammatory Disease¹¹

Ceftriaxone 250 mg IM x 1 dose (or other parenteral third-generation cephalosporin)

And

Doxycycline 100 mg PO bid x 14 days

With or without

Metronidazole 500 mg PO bid x 14 days

Or

Cefoxitin 2 grams IM x 1 dose **and** probenecid 1 gram PO x 1 dose

And

Doxycycline 100 mg PO bid x 14 days

With or without

Metronidazole 500 mg PO bid x 14 days

Abbreviations: bid, 2 times per day; IM, intramuscular; PO, orally.

Table 7. Recommended Alternative Therapies For Mild-To-Moderately Severe Pelvic Inflammatory Disease¹¹

Azithromycin 500 mg IV daily for 1 to 2 doses

Then

Azithromycin 250 mg PO daily for 12-14 days

With or without

Metronidazole 500 mg PO bid x 14 days⁹⁹

Or

Ceftriaxone 250 mg IM x 1 dose

And

Azithromycin 1 gram PO once/week x 2 weeks¹⁰⁰

Abbreviations: bid, 2 times per day; IM, intramuscular; IV, intravenous; PO, orally.

400 mg twice-daily ofloxacin, 400 mg daily moxifloxacin) with metronidazole.¹¹ If patients are going to be started on fluoroquinolone therapy, they should have gonococcal cultures collected in addition to an *N gonorrhoeae* NAAT. This will allow for the identification of fluoroquinolone-resistant *N gonorrhoeae* and specific antibiotic susceptibility. Because of the concern for failure of oral therapy, the patient should have reliable follow-up care and, possibly, access to an infectious disease specialist.

Severe Pelvic Inflammatory Disease

Patients are considered to have severe disease based either on clinical parameters (such as hemodynamic instability), clinical signs of peritonitis, or on the presence of complications (such as TOA). Patients with severe disease should be managed as inpatients with regimens recommended in **Table 8**. As with the oral regimens, the mainstay of treatment includes a cephalosporin combined with doxycycline. Oral administration of doxycycline is the preferred route of administration, even in severe disease,³ because of similar bioavailability with oral and IV administration and the relatively high rates of phlebitis associated with IV administration. The combination of clindamycin with gentamicin has also shown good effectiveness and avoids the risk of phlebitis from IV doxycycline.¹¹

Although disposition and treatment regimens should be determined primarily by the clinical picture, the current recommendation is to admit patients with TOA for at least 24 hours of observation.¹¹ Additionally, because patients with TOA have a higher rate of anaerobic organisms, the recommendation is to add anaerobic coverage with clindamycin, metronidazole, or ampicillin/sulbactam.

There are 2 alternative parenteral therapeutic regimens that can be considered, based on patient allergies or availability. (See **Table 9**.) These regimens

Table 8. Recommended Parenteral Therapy For Pelvic Inflammatory Disease¹¹

| |
|--|
| Cefotetan 2 g IV every 12 hours |
| And |
| Doxycycline 100 mg PO or IV every 12 hours |
| Or |
| Cefoxitin 2 g IV every 6 hours |
| And |
| Doxycycline 100 mg PO or IV every 12 hours |
| Or |
| Clindamycin 900 mg IV every 8 hours |
| And |
| Gentamicin loading dose 2 mg/kg IM or IV, then 1.5 mg/kg every 8 hours |
| Or |
| Gentamicin 3-5 mg/kg IV daily |

Abbreviations: IM, intramuscular; IV, intravenous; PO, orally.

have been shown to have equivalent short-term outcomes to the first-line therapies.^{99,101}

Inpatient management does not mandate IV therapy, and IV antibiotics do not have to be continued through the entire inpatient stay. The transition from IV to oral medications is typically started 24 to 48 hours after clinical improvement and continued for a total of 14 days from the transition. Patients with either radiological or clinical evidence of TOA rupture should be managed operatively. Beyond the hard indication for operative intervention, the emergency clinician should weigh the risks and benefits with gynecological and interventional radiology colleagues with regard to operative and nonoperative management of a TOA. Key factors to consider are the patient's clinical picture, comorbidities, desire for future pregnancies, and response to treatment.

Treatment Of Special Populations

Pregnant Women

PID during pregnancy is rare, and is most commonly seen early in the first trimester, prior to formation of the mucus plug.^{102,103} It is associated with adverse pregnancy-related outcomes, including preterm labor, low birth weight, and perinatal mortality, as well as birth defects such as atrial septal defect and cleft lip.¹⁰⁴⁻¹⁰⁶ Due to these associations, the CDC currently recommends admission and parenteral antibiotics for all pregnant women with PID.¹¹

Unfortunately, CDC guidelines do not specify which parenteral agents should be used. Due to the rarity of PID in pregnancy, there are no clinical trials to guide antibiotic choice. The first-line parenteral regimens recommended by the CDC include doxycycline or gentamicin, which are both classified as pregnancy category D (positive evidence of human fetal risk). (See **Table 8**.) Recent reports indicate that gentamicin, especially when dosed once daily, has limited teratogenic potential.¹⁰⁷⁻¹⁰⁹ Likewise, doxycycline received its pregnancy category based on the class effect of tetracyclines, but has since been demonstrated to have negligible teratogenicity.^{110,111} However, the medicolegal risks of prescribing a

Table 9. Alternative Parenteral Therapy For Pelvic Inflammatory Disease¹¹

| |
|---|
| Ampicillin/sulbactam 3 g IV every 6 hours |
| And |
| Doxycycline 100 mg PO or IV every 12 hours ¹⁰¹ |
| Or |
| Azithromycin 500 mg IV daily for 1-2 doses |
| Then |
| Azithromycin 250 mg PO daily for 12-14 days |
| With or without |
| Metronidazole 500 mg PO bid x 14 days ⁹⁹ |

Abbreviations: bid, 2 times per day; IV, intravenous; PO, orally.

nominally contraindicated (though actually safe) drug are likely not palatable to most emergency clinicians. Therefore, we recommend consultation with an obstetric or infectious disease specialist prior to initiating treatment of pregnant women with PID. If treatment cannot be delayed, azithromycin is a second-line agent that is category B (no risk in animal studies, no adequate human studies). (See **Table 9, page 8.**)

Adolescents

Adolescents are more susceptible to PID for several reasons. Many adolescents are less meticulous about using barrier contraception. Additionally, cervical ectropion exposes a large area of columnar epithelial cells, which are less resistant to infection by *N gonorrhoeae* and *C trachomatis*.¹¹² Maintain a high level of suspicion for PID in adolescents, as they can develop sequelae, such as infertility, after a single episode of PID.¹¹³ Many emergency clinicians fail to inquire about sexual activity in adolescents and thus fail to consider PID as an etiology for pelvic pain. There are no adjustments for the treatment of the adolescent with PID and the decision to hospitalize adolescents should be based on the same criteria as for adult women.¹¹

Patients With HIV

HIV patients with PID are generally infected with the typical PID pathogens and do not need adjustment of the treatment regimens.¹¹⁴ Severity of PID and the tendency to develop complications (such as TOA) appear to have some correlation with the CD4 count. Increased rates of PID are seen with CD4 < 400/mm³. Increased rates of TOA are seen with CD4 counts < 200/mm³. Emergency clinicians should, therefore, maintain a low threshold for imaging patients with a CD4 count < 200/mm³.

Patients With An Intrauterine Device

If a patient with an IUD develops PID, treatment should be initiated as soon as the diagnosis is established and should not be delayed for the removal of the IUD. A systematic review found that PID patients with IUDs had similar outcomes regardless of whether they had their IUD removed or not, with a trend toward the women who retained their IUDs having shorter hospitalizations.¹¹⁵ As with all women with PID, those with an IUD should be reassessed within 72 hours. At the time of reassessment, removal of the IUD can be considered if there is no improvement in symptoms.¹¹⁶

Partner Treatment

Patients diagnosed with PID should abstain from sexual intercourse until treatment has been completed and sexual partners have been adequately treated. All sexual partners within the last 60 days should be evaluated, tested, and receive empiric therapy for

gonorrhea and chlamydia regardless of the cause of PID. If the patient's last sexual encounter was more than 60 days prior, the last sexual partner should be evaluated, tested, and given empiric therapy.¹¹

If it is unlikely that male partners of women with PID will seek treatment, expedited partner therapy (also known as patient-delivered partner therapy) can be considered. Expedited partner therapy has been successfully used to treat partners of patients diagnosed with gonorrhea or chlamydia.^{117,118} While intramuscular ceftriaxone is preferred, a one-time dose of cefixime 400 mg orally in combination with azithromycin 1 gram orally is still recommended as an alternate oral regimen. If the patient is allergic to azithromycin, it can be substituted with a 7-day course of doxycycline 100 mg taken orally twice daily.¹¹ Although most states in the United States allow for expedited partner therapy, clinicians should consult their state-specific regulations at <http://www.cdc.gov/std/ept>.

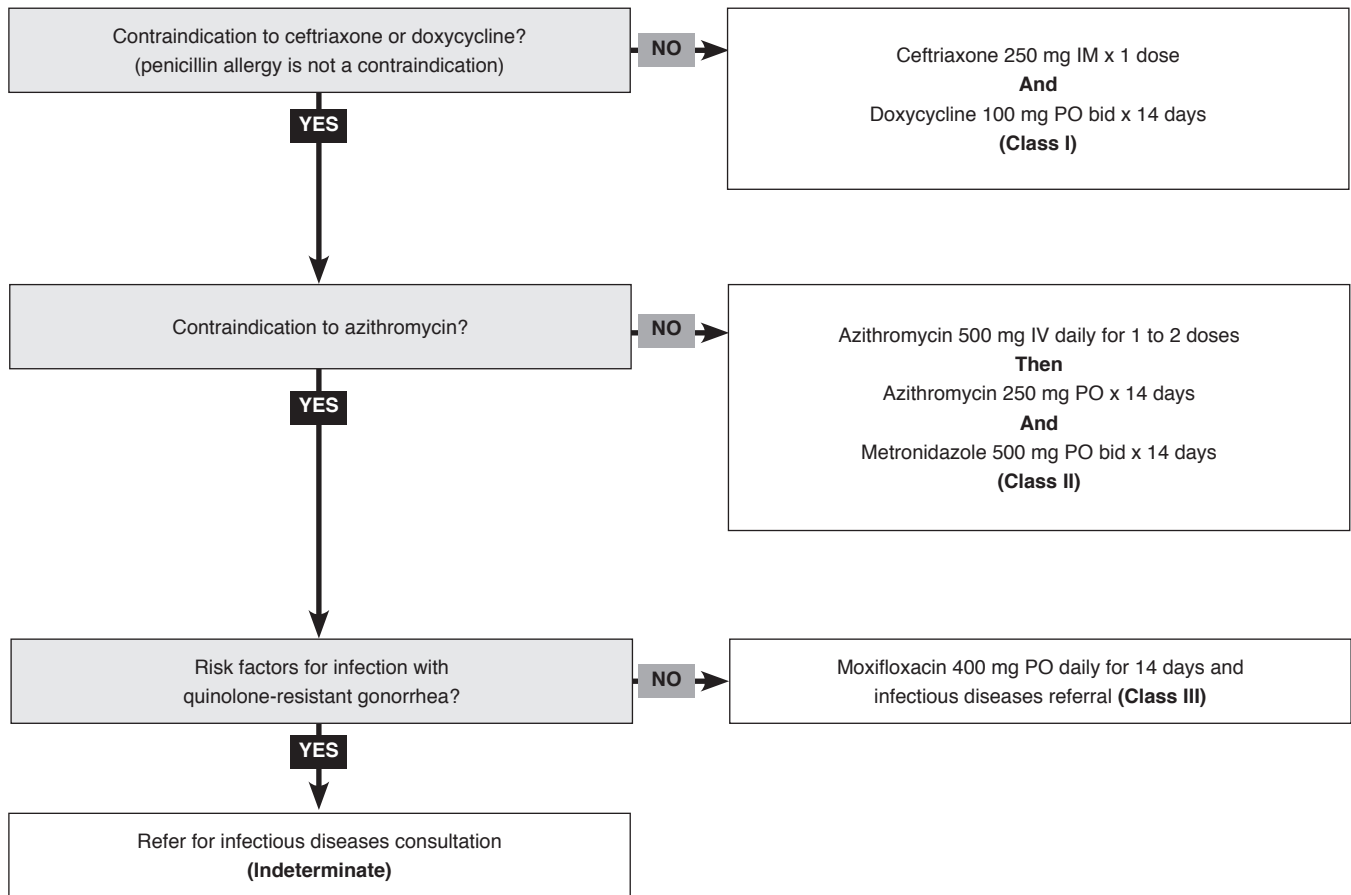
Quality Improvement And Additional Considerations

An area for potential quality improvement is better adherence to the CDC recommendations for empiric treatment of PID.¹¹⁹ In a recent study, only 34% of patients who were treated for PID were given medications that followed the CDC recommendations. The CDC provides clear diagnostic and treatment guidelines for patients with PID, which are easily accessible at <http://www.cdc.gov/std/tg2015/default.htm>.

Presently, there are no national or international quality measures regarding the evaluation and management of the patient with PID. However, the Agency for Healthcare Research and Quality (AHRQ) has identified PID as a disease that can be targeted for cost control. The AHRQ points to studies that show equivalent outcomes between inpatient parenteral and outpatient oral treatment as evidence that the shift to outpatient management can be done safely.¹⁰⁶

Although bounce-back admissions are often seen as a failure of ED management, these admissions should be seen as an expected part of the outpatient management of PID. The CDC emphasizes oral regimens in combination with a 48- to 72-hour re-evaluation, knowing that a small percentage of patients will have inadequate response and will need admission. In the same vein, emphasis should be placed on patient education regarding the importance of the 48- to 72-hour re-evaluation and the potential for admission after re-evaluation.

Clinical Pathway For Antimicrobial Treatment For Pelvic Inflammatory Disease



Abbreviations: bid, 2 times per day; IM, intramuscular; IV, intravenous; PO, by mouth.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I

- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:

- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II

- Safe, acceptable
- Probably useful

Level of Evidence:

- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III

- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:

- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate

- Continuing area of research
- No recommendations until further research

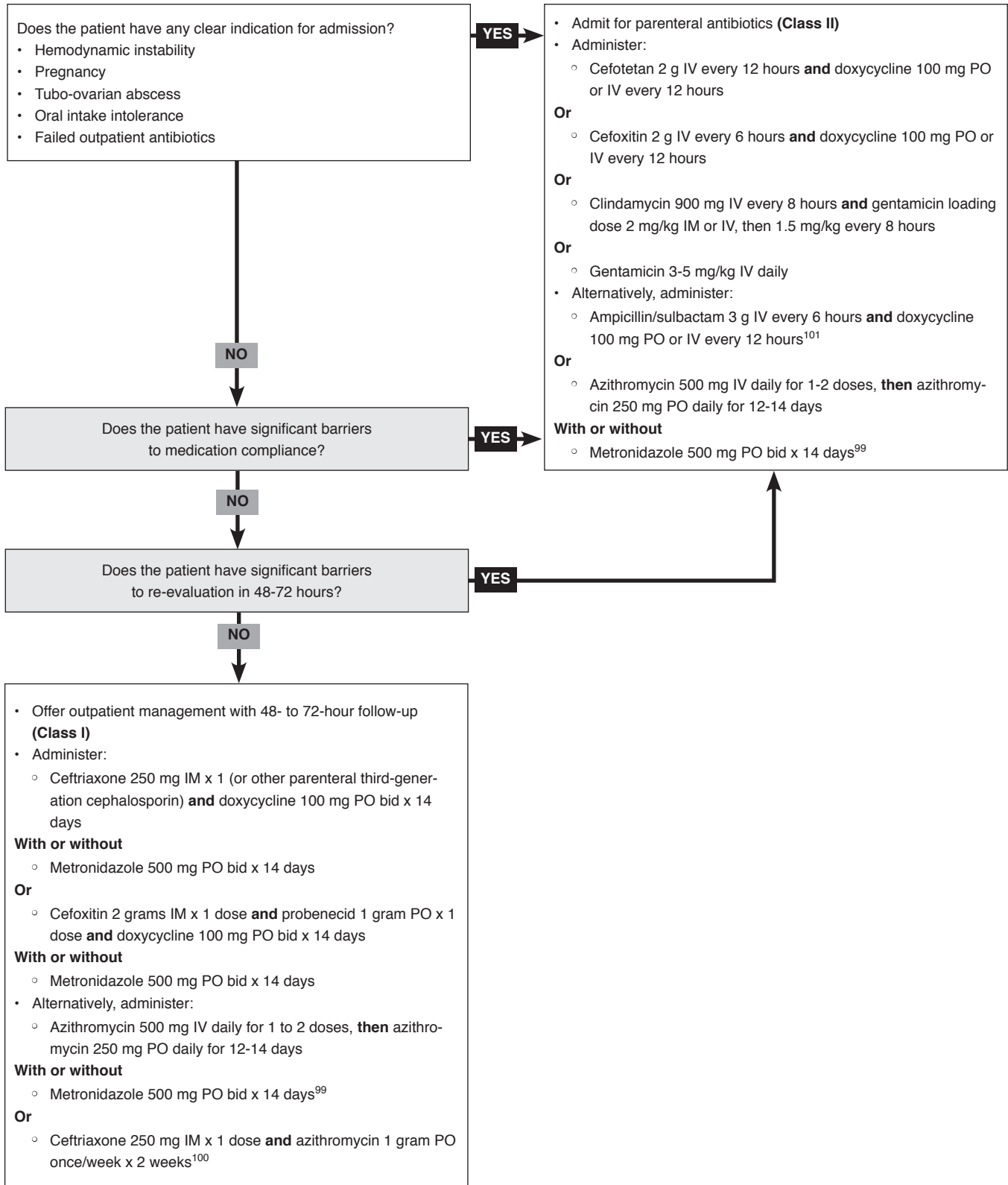
Level of Evidence:

- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Clinical Pathway For Determining Need For Admission For Treatment Of Pelvic Inflammatory Disease



For Class of Evidence Definitions, see page 10.

Abbreviations: bid, 2 times per day; IM, intramuscular; IV, intravenous; PO, orally.

Controversies And Cutting Edge

Mycoplasma genitalium

M genitalium is a fastidious, slow-growing organism that was first isolated from men with non-gonococcal urethritis. A recent meta-analysis confirms that *M genitalium* infection is associated with an increased risk of cervicitis and PID as well as preterm birth and spontaneous abortion.¹²⁰ In contrast to *C trachomatis*, *M genitalium* infection is generally asymptomatic and appears to be less likely to progress to PID.¹⁸ Prevalence of *M genitalium* is similar to the prevalence of *C trachomatis* in young women aged 18 to 27 years (approximately 1%).¹²¹ Notably, the prevalence is considerably greater among men who have sex with men (13%), patients who have had 3 or more sexual partners in the last 12 months (11%), and patients with HIV.^{51,122,123}

Although PCR assays have been developed to detect *M genitalium*, there are currently no United States Food and Drug Administration (FDA)-approved tests to identify *M genitalium* infection. *M genitalium* infection should be considered in patients for whom standard PID treatment has failed, especially those with risk factors.

M genitalium has a relatively high rate of tetracycline resistance and is naturally resistant to beta-lactam antibiotics, leading to the relatively high rate of treatment failure with the typical ceftriaxone and doxycycline regimens.¹²⁴ Azithromycin has been the drug of choice for *M genitalium*; however, there has been a recent rise in azithromycin resistance among *M genitalium* isolates, especially in countries that use 1 gram of azithromycin to treat chlamydia.¹²⁵ Fluoroquinolones have, generally, performed poorly against *M genitalium*, with the exception of moxifloxacin.¹²⁶ Moxifloxacin, in combination with metronidazole, can be considered in patients with failed treatment and risk factors for *M genitalium*.

Use Of Azithromycin In Place Of Doxycycline

In PID, doxycycline is added to eradicate cephalosporin-resistant isolates of *N gonorrhoeae* as well as to treat *C trachomatis* and other pathogens implicated in PID. However, compliance with a 14-day, twice-daily regimen may be challenging for many patients with PID. Azithromycin is an attractive alternative to doxycycline. It is effective against many of the organisms implicated in PID and has the advantage of once-daily dosing.

Several trials have demonstrated the effectiveness of azithromycin as both monotherapy and in combination with metronidazole. A randomized clinical trial in the United Kingdom compared the effectiveness of azithromycin monotherapy, azithromycin plus metronidazole, and standard treatment

for the treatment of PID. This trial found similar microbiological cure rates of 95% to 97% in all 3 arms.⁹⁹ These findings were replicated in 2 additional trials; one with azithromycin monotherapy and another using azithromycin in conjunction with a single intramuscular dose of ceftriaxone.^{100,127}

Regimens using azithromycin are still considered to be second-line treatments because of the lack of long-term outcome data. However, it is likely that as the rates of tetracycline resistance in *N gonorrhoeae* increases, azithromycin will be considered a first-line agent. As with all alternative regimens, the CDC recommends that a culture be obtained prior to starting treatment.

When To Add Metronidazole

Anaerobes have been associated with PID, both as primary and secondary pathogens. Standard regimens for PID were not specifically designed to target anaerobes; however, cure rates using only ceftriaxone and doxycycline are excellent. In one study, adding metronidazole to an azithromycin regimen only improved microbiologic cure rates from 97% to 98%.⁹⁹ Conversely, omitting a cephalosporin and using only doxycycline and metronidazole resulted in low cure rates.¹²⁸ No trials have specifically investigated the addition of metronidazole to standard regimens and evaluated its effect on cure rates or sequelae of PID.

Expert opinions regarding metronidazole vary. CDC guidelines recommend that addition of anaerobic coverage be considered "until treatment regimens that do not cover anaerobic microbes have been demonstrated to prevent long-term sequelae (eg, infertility and ectopic pregnancy) as successfully as the regimens that are effective against these microbes."¹¹ Some experts recommend that all patients receive metronidazole, due to the high prevalence of anaerobes in PID,¹²⁹ while others recommend that it should only be added in women with evidence of bacterial vaginosis or trichomoniasis.¹³⁰

Although optimal coverage of all possible pathogens is ideal, several studies have found a trend toward increased side effects and decreased compliance when metronidazole is added to the standard therapy.^{99,131,132} This is a major concern, as compliance with prolonged doxycycline regimens in PID is already difficult.¹³³

Based on the current evidence, we do not recommend the routine addition of metronidazole to the treatment regimen. However, when TOA is present, there is consensus that metronidazole or clindamycin should be added to increase anaerobic coverage.¹¹ Furthermore, the addition of metronidazole should be strongly considered in patients with suspected treatment failure or in patients clinically suspected to have concomitant bacterial vaginosis.

Drug-Resistant Organisms

N gonorrhoeae resistance to cephalosporins and azithromycin is increasing in the United States, most notably among men who have sex with men.^{52,134} In 2012, approximately 0.3% of *N gonorrhoeae* isolates in the United States had an elevated minimum inhibitory concentration (MIC), indicating decreased sensitivity to cephalosporins.⁵² Although a single dose of 250 mg of intramuscular ceftriaxone is currently sufficient to eradicate the majority of *N gonorrhoeae* in the United States, this dosing regimen may not maintain a concentration high enough to eradicate *N gonorrhoeae* with the elevated MIC.¹³⁵ With the gradual MIC creep and increasing prevalence of resistant organisms, treatment failures can be expected to increase in the future. Either increasing the length of treatment or increasing the amount of medication per dose may address this MIC creep. Currently, 1-time doses of 1 gram of ceftriaxone are used in China (intramuscular) and in Japan (intravenous); and 500 mg is used in the United Kingdom (intramuscular). Unfortunately, there have been reports of *N gonorrhoeae* that are resistant to even these higher doses of cephalosporin in Japan and France.^{136,137} There has also been a rise in the rate and the degree of azithromycin-resistant *N gonorrhoeae* seen primarily in the Western United States and among men who have sex with men.^{52,134}

Patients With Suspected Treatment Failure

When a patient presents with continued or worse symptoms after initiation of therapy, there are multiple potential reasons, including difficulty with medication adherence and re-infection. A careful sexual history is perhaps more important at the time of re-evaluation than during the initial evaluation, as it is crucial to evaluate for the possibility of re-infection. The most concerning potential cause of persisting symptoms is the possibility of treatment failure due to infection with a resistant organism. For patients with suspected treatment failure, the treating clinician should consult an infectious-disease specialist, an STI/HIV Prevention Training Center clinical expert,¹¹⁶ the local or state health department STI program, or the CDC (telephone: 404-639-8659) for advice on obtaining cultures, antimicrobial susceptibility testing, and treatment.¹¹

Point-Of-Care Testing In Low-Resource Settings

Point-of-care testing is an attractive option that would allow for both rapid care and for diagnosis and treatment in resource-poor settings. A recent systematic review evaluated point-of-care testing with leukocyte esterase dipsticks, immunochromatography strips, and microscopy in low-resource settings and found them to have modest sensitivities and specificities, with high negative predictive

values among symptomatic patients.¹³⁸ However, these findings were not replicated in another study that found that 3 point-of-care tests for *C trachomatis* performed uniformly poorly.¹³⁹ A highly sensitive and specific genetic point-of-care test for *N gonorrhoeae* and *C trachomatis* has been developed and is currently under clinical trial in Aboriginal communities in Australia.^{140,141}

Currently, the World Health Organization does not recommend point-of-care testing. They instead recommend “syndromic management,” where treatment is based on signs and symptoms as well as knowledge of local prevalence of various pathogens, instead of reliance on testing.¹⁴²

Disposition And Transition Of Care

Disposition depends on the severity of the patient’s disease. Admission should be strongly considered for patients who meet any of the criteria in **Table 10**.

Not only is it important that follow-up is arranged for the patient, it is important that the next practitioner understands the reason and the goals for the re-evaluation. Clinicians should strive to clarify in their documentation that the patient is returning to be evaluated for clinical improvement and that the next clinician should consider escalation of therapy if there is insufficient response.

Long-term follow-up is an important component of PID management. In light of the relatively high reinfection rates after *N gonorrhoeae* and *C trachomatis* infection (11.7% and 13.9%, respectively), the current recommendation is for patients to have a “test of cure.”¹⁴³ Patients who have been diagnosed with PID secondary to gonorrhea or chlamydia should be retested for *N gonorrhoeae* or *C trachomatis* infection 3 months after treatment, or at the next visit in the following 12 months.¹¹ This recommendation is irrespective of whether their sex partners were treated.

Table 10. Criteria For Consideration For Hospital Admission¹¹

- Tubo-ovarian abscess
- Pregnancy
- Severe illness
- Inability to comply with oral therapy
- Oral intake intolerance
- Lack of clinical response to oral therapy
- Inability to exclude surgical emergency (eg, appendicitis)

Summary

PID is a clinical diagnosis with a spectrum of presentations. Missed diagnosis carries both short- and long-term risks for complications. The diagnosis should be considered in any woman with lower abdominal pain who is about to be discharged with the diagnosis of “abdominal pain NOS (not otherwise specified)” or a young woman who is going to be discharged with the diagnosis of urinary tract infection without urinary symptoms. The diagnosis should be made clinically in the appropriate patient population and empiric therapy started based on the CDC guidelines. The imaging modality for select patients with suspected PID is transabdominal and transvaginal ultrasound with Doppler. Ultrasound findings may support the diagnosis; however, normal imaging lacks the sensitivity to rule out the diagnosis. If patients are managed as outpatients, there should be clear follow-up instructions, with a 48- to 72-hour re-evaluation to ensure clinical improvement.

Time- And Cost-Effective Strategies

- A potential area for managing cost in the treatment of PID is in avoiding parenteral and inpatient treatment. Oral treatment has been shown to be safe and effective and should be considered first-line therapy. Inpatient parenteral therapy should be reserved for patients with clear indications for admission.
- An effective time-management strategy is to limit the amount of imaging. Due to the overlap with other clinical entities, some imaging is inevitable; however, it is important to remember that imaging is not needed to make the diagnosis of PID. Imaging should be limited to cases when the patient is ill, there is a concern for TOA, or to evaluate for an alternative diagnosis such as appendicitis or torsion.

Risk Management Pitfalls In Pelvic Inflammatory Disease

(Continued on page 15)

1. “She had a negative CT and pelvic ultrasound, so I ruled out PID.”

Emergency clinicians should not use negative imaging to exclude the diagnosis of PID. Even pelvic ultrasound lacks sufficient sensitivity to exclude the diagnosis. Patients at risk for PID who have lower abdominal pain that is not easily explained by another diagnosis should have empiric treatment for PID started.

2. “Yes, she could have had PID, but she looked so well that I discharged her and deferred treatment to her primary care physician.”

All patients who have the clinical diagnosis of PID should have empiric therapy started. Initial presentation does not predict progression of the disease and, therefore, should not be used to determine who should have treatment initiated.

3. “I gave a gram of azithromycin and a shot of ceftriaxone to treat her PID.”

There is no single-dose treatment of PID, as standard treatment regimens last for 14 days. This particular regimen is used to treat cervicitis in the absence of signs and symptoms of PID. Failure to provide adequate duration of treatment places the patient at risk for undertreatment and the development of a resistant organism. If azithromycin is being used as the sole agent, use one of the accepted treatment regimens for PID.

4. “When she came back to the ED, I checked her records and saw that she had a negative *N gonorrhoeae/C trachomatis* test, so I stopped her medication and reassured her that she didn’t have PID.”

A negative *N gonorrhoeae/C trachomatis* test cannot be used to rule out the possibility of PID. A cervical *N gonorrhoeae/C trachomatis* NAAT is a test of lower-tract disease and does not exclude the presence of an upper-tract infection. Additionally, the test does not test for anaerobes or *M genitalium*, both of which are implicated in PID. For these reasons, a negative *N gonorrhoeae/C trachomatis* NAAT cannot be used to rule out the possibility of PID.

5. “When I told her to see her doctor in 2 days, I assumed she would do it. If she didn’t have a doctor, she should have just come back to the ED.”

Most patients with PID should have a clinical response within 48 to 72 hours. Many of the decision points are based on the response to treatment at this repeat visit, especially with regard to the need for imaging, changes in antibiotics, or need for parenteral therapy. Therefore, it is important that the patient has access to and understands the importance of the follow-up.

Case Conclusions

The initial clinician in the case fell victim to many of the common pitfalls in the evaluation of patients with pelvic pain: the well appearance of the patient, the minimal findings on physical examination, and the findings of leukocyte esterase in the urine. Fortunately, you resisted the cognitive biases that come from sign-outs, and you correctly diagnosed the patient with PID. She was started on empiric outpatient therapy for PID, with a single dose of 250 mg ceftriaxone IM and 2 weeks of 100 mg of doxycycline PO, twice per day. You made arrangements for follow-up in 3 days in the gynecologic clinic to assess for clinical response, where she was found to have had a good response. She was subsequently found to have a positive *N gonorrhoeae/C trachomatis* NAAT, but was HIV-negative. Two years later, she was able to get pregnant with a new partner and deliver without any difficulty or complications.

Regarding your second patient, in light of the appropriate treatment and the lack of clinical response, she was clearly having a treatment failure of oral antibiotics. You

were concerned for the interim development of a TOA or other complication of PID. Although the patient was not found to have complicated PID, she was admitted for IV antibiotics. The inpatient team expanded her antibiotic coverage to PO doxycycline and IV cefotetan. The patient required 5 days of IV antibiotics, and was transitioned to inpatient oral therapy once she clinically improved. She was discharged on day 7 to continue her oral therapy. She was aware of the possibility of long-term infertility, but was happy that she is feeling better.

Must-Do Markers Of Quality Care

- Evaluate and document signs and symptoms concerning for PID.
- Test for presence of *N gonorrhoeae/C trachomatis*.
- Give appropriate empiric treatment and arrange for appropriate follow-up.
- Evaluate for alternative diagnoses.

Risk Management Pitfalls In Pelvic Inflammatory Disease

(Continued from page 14)

6. **“I told her that her PID was probably sexually transmitted and assumed she understood that she should avoid any further sexual interactions with her partner.”**

Patients with a diagnosis of PID should abstain from intercourse until the resolution of therapy and until after the partners have completed empiric treatment. This recommendation is true regardless of the cause of the PID. While it may seem intuitive, it is important to speak to the patient directly about the importance of partner treatment to prevent re-infection.

7. **“She had white blood cells on the urine microscopy, so I treated her for a urinary tract infection even though she had no dysuria.”**

Patients with PID commonly have white blood cells on urine microscopy. Additionally, uterine tenderness can be mistaken for suprapubic tenderness due to cystitis. Patient risk factors must always be considered, and the presence or absence of dysuria is not diagnostically specific to differentiate PID from a urinary tract infection.

8. **“She came back with continued pain, so I re-filled her pain medications.”**

When a patient fails to show adequate response to treatment, you must first consider the need for parenteral treatment, development of a complication, and infection with a resistant

organism. Consider additional testing with a cervical culture, which would allow for the identification of a resistant organism. Additionally, strongly consider increased coverage of anaerobic organisms.

9. **“Her CBC, chemistries, and all of her imaging were normal. If it was anything consequential, we would have picked it up on our workup, so PID can be ruled out.”**

There are no laboratory tests or imaging modalities that have adequate sensitivity to exclude the diagnosis of PID. Laboratory tests and imaging are typically only abnormal with sicker patients. Overreliance on laboratory testing and imaging will lead to missed diagnoses.

10. **“She had clue cells and white blood cells on her wet mount, so I treated her for bacterial vaginosis.”**

The presence of bacterial vaginosis does not exclude the diagnosis of PID. Bacterial vaginosis can be associated with PID. In some cases, it may be due to direct ascension of anaerobic bacteria, while in other cases it may be secondary to the loss of mucosal immunity secondary to the bacterial overgrowth.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study is included in bold type following the reference, where available.

1. Sutton MY, Sternberg M, Zaidi A, et al. Trends in pelvic inflammatory disease hospital discharges and ambulatory visits, United States, 1985-2001. *Sex Transm Dis*. 2005;32(12):778-784. **(Retrospective; 770,000 patients)**
2. Rein DB, Kassler WJ, Irwin KL, et al. Direct medical cost of pelvic inflammatory disease and its sequelae: decreasing, but still substantial. *Obstet Gynecol*. 2000;95(3):397-402. **(Retrospective; 3 years of claims data)**
- 3.* Ness RB, Soper DE, Holley RL, et al. Effectiveness of inpatient and outpatient treatment strategies for women with pelvic inflammatory disease: results from the Pelvic Inflammatory Disease Evaluation and Clinical Health (PEACH) Randomized Trial. *Am J Obstet Gynecol*. 2002;186(5):929-937. **(Prospective; 831 patients)**
4. Landers DV, Sweet RL. Tubo-ovarian abscess: contemporary approach to management. *Rev Infect Dis*. 1983;5(5):876-884. **(Retrospective; 232 patients)**
5. Macaluso M, Wright-Schnapp TJ, Chandra A, et al. A public health focus on infertility prevention, detection, and management. *Fertil Steril*. 2010;93(1):16.e1-e10. **(Review)**
6. Eschenbach DA, Wølner-Hanssen P, Hawes SE, et al. Acute pelvic inflammatory disease: associations of clinical and laboratory findings with laparoscopic findings. *Obstet Gynecol*. 1997;89(2):184-192. **(Prospective; 155 patients)**
- 7.* Kahn JG, Walker CK, Washington AE, et al. Diagnosing pelvic inflammatory disease. A comprehensive analysis and considerations for developing a new model. *JAMA*. 1991;266(18):2594-2604. **(Systematic review)**
8. Westrom L, Joesoef R, Reynolds G, et al. Pelvic inflammatory disease and fertility. A cohort study of 1,844 women with laparoscopically verified disease and 657 control women with normal laparoscopic results. *Sex Transm Dis*. 1992;19(4):185-192. **(Prospective study; 1800 patients)**
9. Molander P, Sjöberg J, Paavonen J, et al. Transvaginal power Doppler findings in laparoscopically proven acute pelvic inflammatory disease. *Ultrasound Obstet Gynecol*. 2001;17(3):233-238. **(Prospective; 30 patients)**
10. Tukeva TA, Aronen HJ, Karjalainen PT, et al. MR imaging in pelvic inflammatory disease: comparison with laparoscopy and US. *Radiology*. 1999;210(1):209-216. **(Prospective; 30 patients)**
- 11.* Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep*. 2015;64(3):1-137. **(Expert guidelines/systematic review)**
12. UK national guideline for the management of pelvic inflammatory disease 2011. London, UK: British Association for Sexual Health and HIV; June 18, 2011. **(Guideline)**
13. Andreotti RF, Lee SJ, Dejesus Allison SO, et al. ACR Appropriateness Criteria[®] acute pelvic pain in the reproductive age group. *Ultrasound Q*. 2011;27(3):205-210. **(Expert guideline)**
14. Rohrbeck P. Pelvic inflammatory disease among female recruit trainees, active component, U.S. armed forces, 2002-2012. *MSMR*. 2013;20(9):15-18. **(Retrospective; 1500 patients)**
15. Westrom L. Incidence, prevalence, and trends of acute pelvic inflammatory disease and its consequences in industrialized countries. *Am J Obstet Gynecol*. 1980;138(7 Pt 2):880-892. **(Retrospective epidemiological study)**
16. Ness RB, Trautmann G, Richter HE, et al. Effectiveness of treatment strategies of some women with pelvic inflammatory disease: a randomized trial. *Obstet Gynecol*. 2005;106(3):573-580. **(Prospective; 831 patients)**
17. Haggerty CL, Totten PA, Astete SG, et al. *Mycoplasma genitalium* among women with nongonococcal, nonchlamydial pelvic inflammatory disease. *Infect Dis Obstet Gynecol*. 2006;2006:30184. **(Retrospective; 50 patients)**
18. Bjartling C, Osser S, Persson K. *Mycoplasma genitalium* in cervicitis and pelvic inflammatory disease among women at a gynecologic outpatient service. *Am J Obstet Gynecol*. 2012;206(6):476.e471-e478. **(Case-controlled study; 5519 patients)**
19. Clarke LM, Duerr A, Yeung KH, et al. Recovery of cytomegalovirus and herpes simplex virus from upper and lower genital tract specimens obtained from women with pelvic inflammatory disease. *J Infect Dis*. 1997;176(1):286-288. **(Prospective; 147 patients)**
20. Chernes TL, Wiesenfeld HC, Melan MA, et al. The associations between pelvic inflammatory disease, *Trichomonas vaginalis* infection, and positive herpes simplex virus type 2 serology. *Sex Transm Dis*. 2006;33(12):747-752. **(Retrospective; 736 patients)**
21. Soper DE, Brockwell NJ, Dalton HP. Microbial etiology of urban emergency department acute salpingitis: treatment with ofloxacin. *Am J Obstet Gynecol*. 1992;167(3):653-660. **(Prospective; 36 patients)**
22. Paavonen J, Teisala K, Heinonen PK, et al. Microbiological and histopathological findings in acute pelvic inflammatory disease. *Br J Obstet Gynaecol*. 1987;94(5):454-460. **(Prospective; 45 patients)**
23. Cho HW, Koo YJ, Min KJ, et al. Pelvic inflammatory disease in virgin women with tubo-ovarian abscess: A single-center experience and literature review. [Epub ahead of print] 7 August 2015. *J Pediatr Adolesc Gynecol*. **(Retrospective; 122 patients)**
24. Goodwin K, Fleming N, Dumont T. Tubo-ovarian abscess in virginal adolescent females: a case report and review of the literature. *J Pediatr Adolesc Gynecol*. 2013;26(4):e99-e102. **(Case report)**
25. Taylor BD, Darville T, Haggerty CL. Does bacterial vaginosis cause pelvic inflammatory disease? *Sex Transm Dis*. 2013;40(2):117-122. **(Systematic review)**
26. Yarbrough VL, Winkle S, Herbst-Kralovetz MM. Antimicrobial peptides in the female reproductive tract: a critical component of the mucosal immune barrier with physiological and clinical implications. *Hum Reprod Update*. 2015;21(3):353-377. **(Systematic review)**
27. Peter NG, Clark LR, Jaeger JR. Fitz-Hugh-Curtis syndrome: a diagnosis to consider in women with right upper quadrant pain. *Cleve Clin J Med*. 2004;71(3):233-239. **(Review)**
28. Risser WL, Risser JM, Benjamins LJ, et al. Incidence of Fitz-Hugh-Curtis syndrome in adolescents who have pelvic inflammatory disease. *J Pediatr Adolesc Gynecol*. 2007;20(3):179-180. **(Prospective study; 117 patients presenting to a juvenile detention center)**
29. Schindlbeck C, Dziura D, Mylonas I. Diagnosis of pelvic inflammatory disease (PID): intra-operative findings and comparison of vaginal and intra-abdominal cultures. *Arch Gynecol Obstet*. 2014;289(6):1263-1269. **(Retrospective; 73 patients)**
30. Chappell CA, Wiesenfeld HC. Pathogenesis, diagnosis, and management of severe pelvic inflammatory disease and tuboovarian abscess. *Clin Obstet Gynecol*. 2012;55(4):893-903. **(Review)**
31. Cohen CR, Sinei S, Reilly M, et al. Effect of human immunodeficiency virus type 1 infection upon acute salpingitis:

- a laparoscopic study. *J Infect Dis*. 1998;178(5):1352-1358. **(Prospective; 133 patients)**
32. Wiesenfeld HC, Sweet RL, Ness RB, et al. Comparison of acute and subclinical pelvic inflammatory disease. *Sex Transm Dis*. 2005;32(7):400-405. **(Retrospective; 1293 patients)**
 33. Hadgu A, Westrom L, Brooks CA, et al. Predicting acute pelvic inflammatory disease: a multivariate analysis. *Am J Obstet Gynecol*. 1986;155(5):954-960. **(Retrospective; 629 patients)**
 34. Marks C, Tideman RL, Estcourt CS, et al. Assessment of risk for pelvic inflammatory disease in an urban sexual health population. *Sex Transm Infect*. 2000;76(6):470-473. **(Retrospective; 741 patients)**
 35. Blake DR, Fletcher K, Joshi N, et al. Identification of symptoms that indicate a pelvic examination is necessary to exclude PID in adolescent women. *J Pediatr Adolesc Gynecol*. 2003;16(1):25-30. **(Retrospective; 193 patients)**
 36. Amman R, Zehender O, Jenny S, et al. [Acute gonococcal perihepatitis (Fitz-Hugh-Curtis syndrome). An acute, right-side "pleuritic-peritonitic" upper abdominal pain syndrome in Adnexitis gonorrhoea: diagnosis by laparoscopy]. *Dtsch Med Wochenschr*. 1971;96(39):1515-1519. **(Case series; 5 patients)**
 37. Gatt D, Jantet G. Perisplenitis and perinephritis in the Curtis-Fitz-Hugh syndrome. *Br J Surg*. 1987;74(2):110-112. **(Case series; 4 patients)**
 38. Lopez-Zeno JA, Keith LG, Berger GS. The Fitz-Hugh-Curtis syndrome revisited. Changing perspectives after half a century. *J Reprod Med*. 1985;30(8):567-582. **(Systematic review)**
 39. Kim HY, Yang JI, Moon C. Comparison of severe pelvic inflammatory disease, pyosalpinx and tubo-ovarian abscess. *J Obstet Gynaecol Res*. 2015;41(5):742-746. **(Retrospective; 458 patients)**
 40. Rothman KJ, Lanza L, Lal A, et al. Incidence of pelvic inflammatory disease among women treated for gonorrhea or chlamydia. *Pharmacoepidemiol Drug Saf*. 1996;5(6):409-414. **(Retrospective; 254 patients)**
 41. Kerani RP, Stenger MR, Weinstock H, et al. Gonorrhea treatment practices in the STD Surveillance Network, 2010-2012. *Sex Transm Dis*. 2015;42(1):6-12. **(Prospective; 44,144 isolates)**
 42. Gullberg E, Cao S, Berg OG, et al. Selection of resistant bacteria at very low antibiotic concentrations. *PLoS Pathog*. 2011;7(7):e1002158. **(Review)**
 43. Kimani J, Maclean IW, Bwayo JJ, et al. Risk factors for *Chlamydia trachomatis* pelvic inflammatory disease among sex workers in Nairobi, Kenya. *J Infect Dis*. 1996;173(6):1437-1444. **(Prospective; 302 patients)**
 44. Elizur SE, Lebovitz O, Weintraub AY, et al. Pelvic inflammatory disease in women with endometriosis is more severe than in those without. *Aust N Z J Obstet Gynaecol*. 2014;54(2):162-165. **(Retrospective; 174 patients)**
 45. Kubota T, Ishi K, Takeuchi H. A study of tubo-ovarian and ovarian abscesses, with a focus on cases with endometrioma. *J Obstet Gynaecol Res*. 1997;23(5):421-426. **(Retrospective; 6557 patients)**
 46. Washington AE, Cates W Jr, Wasserheit JN. Preventing pelvic inflammatory disease. *JAMA*. 1991;266(18):2574-2580. **(Meta-analysis)**
 47. Scholes D, Daling JR, Stergachis A, et al. Vaginal douching as a risk factor for acute pelvic inflammatory disease. *Obstet Gynecol*. 1993;81(4):601-606. **(Retrospective; 131 patients)**
 48. Gareen IF, Greenland S, Morgenstern H. Intrauterine devices and pelvic inflammatory disease: meta-analyses of published studies, 1974-1990. *Epidemiology*. 2000;11(5):589-597. **(Meta-analysis from 1974-1990)**
 49. Walsh T, Grimes D, Frezieres R, et al. Randomised controlled trial of prophylactic antibiotics before insertion of intrauterine devices. IUD Study Group. *Lancet*. 1998;351(9108):1005-1008. **(Prospective; 1984 patients)**
 50. Birgisson NE, Zhao Q, Secura GM, et al. Positive testing for *Neisseria gonorrhoeae* and *Chlamydia trachomatis* and the risk of pelvic inflammatory disease in IUD users. *J Womens Health (Larchmt)*. 2015;24(5):354-359. **(Prospective; 9000 patients)**
 51. Cox C, McKenna JP, Watt AP, et al. *Ureaplasma parvum* and *Mycoplasma genitalium* are found to be significantly associated with microscopy-confirmed urethritis in a routine genitourinary medicine setting. *Int J STD AIDS*. 2016;27(10):861-867. **(Prospective; 165 patients)**
 52. Kirkcaldy RD, Soge O, Papp JR, et al. Analysis of *Neisseria gonorrhoeae* azithromycin susceptibility in the United States by the Gonococcal Isolate Surveillance Project, 2005 to 2013. *Antimicrob Agents Chemother*. 2015;59(2):998-1003. **(Prospective; 45,000 isolates)**
 53. Li M, McDermott R. Smoking, poor nutrition, and sexually transmitted infections associated with pelvic inflammatory disease in remote North Queensland indigenous communities, 1998-2005. *BMC Womens Health*. 2015;15:31. **(Retrospective; 1445 patients)**
 - 54.* Ness RB, Smith KJ, Chang CC, et al. Prediction of pelvic inflammatory disease among young, single, sexually active women. *Sex Transm Dis*. 2006;33(3):137-142. **(Prospective; 1170 patients)**
 55. Scholes D, Daling JR, Stergachis AS. Current cigarette smoking and risk of acute pelvic inflammatory disease. *Am J Public Health*. 1992;82(10):1352-1355. **(Retrospective; 131 patients)**
 56. Umbricht-Schneiter A, Santora P, Moore RD. Alcohol abuse: comparison of two methods for assessing its prevalence and associated morbidity in hospitalized patients. *Am J Med*. 1991;91(2):110-118. **(Prospective; 1964 patients)**
 57. Jamieson DJ, Duerr A, Macasaet MA, et al. Risk factors for a complicated clinical course among women hospitalized with pelvic inflammatory disease. *Infect Dis Obstet Gynecol*. 2000;8(2):88-93. **(Prospective; 349 patients)**
 58. Champion JD, Shain RN, Piper J. Minority adolescent women with sexually transmitted diseases and a history of sexual or physical abuse. *Issues Ment Health Nurs*. 2004;25(3):293-316. **(Prospective; 30 patients)**
 59. Glaser D. Treatment issues in child sexual abuse. *Br J Psychiatry*. 1991;159:769-782. **(Review)**
 60. Wasserheit JN, Bell TA, Kiviat NB, et al. Microbial causes of proven pelvic inflammatory disease and efficacy of clindamycin and tobramycin. *Ann Intern Med*. 1986;104(2):187-193. **(Prospective; 36 patients)**
 61. Simms I, Warburton F, Westrom L. Diagnosis of pelvic inflammatory disease: time for a rethink. *Sex Transm Infect*. 2003;79(6):491-494. **(Retrospective; 623 patients)**
 62. Slap GB, Forke CM, Cnaan A, et al. Recognition of tubo-ovarian abscess in adolescents with pelvic inflammatory disease. *J Adolesc Health*. 1996;18(6):397-403. **(Retrospective; 208 patients)**
 63. Wagner A, Russell C, Ponterio JM, et al. Ruptured tuboovarian abscess and septic shock with *Clostridium perfringens* in a postmenopausal woman: a case report. *J Reprod Med*. 2009;54(10):652-654. **(Case report)**
 64. Westfall MD, Lumpkin J. A 33-year-old white female with abdominal pain, nausea, vomiting and hypotension. *J Emerg Med*. 1993;11(3):271-273. **(Case report)**
 65. Peipert JF, Ness RB, Blume J, et al. Clinical predictors of endometritis in women with symptoms and signs of pelvic inflammatory disease. *Am J Obstet Gynecol*. 2001;184(5):856-863. **(Retrospective; 651 patients)**
 66. Litt IF, Cohen MI. Perihepatitis associated with salpingitis in adolescents. *JAMA*. 1978;240(12):1253-1254. **(Retrospective; 137 patients)**
 67. Fitz-Hugh T. Acute gonococcal peritonitis of the right upper quadrant in women. *JAMA*. 1934;102(25):2094-2096. **(Case series; 3 patients)**
 68. Adhikari S, Blaivas M, Lyon M. Role of bedside transvaginal

- ultrasonography in the diagnosis of tubo-ovarian abscess in the emergency department. *J Emerg Med.* 2008;34(4):429-433. **(Retrospective; 20 patients)**
69. Brunham RC, Paavonen J, Stevens CE, et al. Mucopurulent cervicitis--the ignored counterpart in women of urethritis in men. *N Engl J Med.* 1984;311(1):1-6. **(Prospective; 100 patients)**
 70. Shrier LA, Dean D, Klein E, et al. Limitations of screening tests for the detection of *Chlamydia trachomatis* in asymptomatic adolescent and young adult women. *Am J Obstet Gynecol.* 2004;190(3):654-662. **(Prospective; 139 women)**
 71. Shafer MA, Moncada J, Boyer CB, et al. Comparing first-void urine specimens, self-collected vaginal swabs, and endocervical specimens to detect *Chlamydia trachomatis* and *Neisseria gonorrhoeae* by a nucleic acid amplification test. *J Clin Microbiol.* 2003;41(9):4395-4399. **(Prospective; 2157 patients)**
 72. Zakher B, Cantor AG, Pappas M, et al. Screening for gonorrhea and chlamydia: a systematic review for the U.S. Preventive Services Task Force screening for gonorrhea and chlamydia. *Ann Intern Med.* 2014;161(12):884-893. **(Systematic review)**
 73. Van Der Pol B, Liesenfeld O, Williams JA, et al. Performance of the cobas CT/NG test compared to the Aptima AC2 and Viper CTQ/GCQ assays for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. *J Clin Microbiol.* 2012;50(7):2244-2249. **(Prospective; 4316 samples)**
 74. Van Der Pol B, Taylor SN, Lebar W, et al. Clinical evaluation of the BD ProbeTec *Neisseria gonorrhoeae* Qx amplified DNA assay on the BD Viper system with XTR technology. *Sex Transm Dis.* 2012;39(2):147-153. **(Prospective; 1768 patients)**
 75. Schoeman SA, Stewart CM, Booth RA, et al. Assessment of best single sample for finding chlamydia in women with and without symptoms: a diagnostic test study. *BMJ.* 2012;345:e8013. **(Prospective; 3973 patients)**
 76. Chernesky M, Jang D, Gilchrist J, et al. Head-to-head comparison of second-generation nucleic acid amplification tests for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* on urine samples from female subjects and self-collected vaginal swabs. *J Clin Microbiol.* 2014;52(7):2305-2310. **(Prospective; 575 patients)**
 77. Berwald N, Cheng S, Augenbraun M, et al. Self-administered vaginal swabs are a feasible alternative to physician-assisted cervical swabs for sexually transmitted infection screening in the emergency department. *Acad Emerg Med.* 2009;16(4):360-363. **(Prospective; 162 patients)**
 78. Risser JM, Risser WL. Purulent vaginal and cervical discharge in the diagnosis of pelvic inflammatory disease. *Int J STD AIDS.* 2009;20(2):73-76. **(Systematic review)**
 79. Singh D, Marrazzo JM. Screening and management of genital chlamydial infections. *Infect Dis Clin North Am.* 2013;27(4):739-753. **(Review)**
 80. Griffith WF, Stuart GS, Gluck KL, et al. Vaginal speculum lubrication and its effects on cervical cytology and microbiology. *Contraception.* 2005;72(1):60-64. **(Prospective; 6538 patients)**
 81. Rabe LK, Hillier SL. Effect of chlorhexidine on genital microflora, *Neisseria gonorrhoeae*, and *Trichomonas vaginalis* in vitro. *Sex Transm Dis.* 2000;27(2):74-78. **(In vitro study)**
 82. Lampe MF, Ballweber LM, Stamm WE. Susceptibility of *Chlamydia trachomatis* to chlorhexidine gluconate gel. *Antimicrob Agents Chemother.* 1998;42(7):1726-1730. **(In vitro study)**
 83. Webster LA, Berman SMB, Greenspan JR. Surveillance for gonorrhea and primary and secondary syphilis among adolescents, U.S. 1981-1991. *MMWR CDC Surveill Summ.* 1993;42(SS-3):1-11. **(Retrospective; health department reports 1981-1991)**
 84. Tomas ME, Getman D, Donskey CJ, et al. Overdiagnosis of urinary tract infection and underdiagnosis of sexually transmitted infection in adult women presenting to an emergency department. *J Clin Microbiol.* 2015;53(8):2686-2692. **(Prospective; 264 patients)**
 85. Bevan CD, Johal BJ, Mumtaz G, et al. Clinical, laparoscopic and microbiological findings in acute salpingitis: report on a United Kingdom cohort. *Br J Obstet Gynaecol.* 1995;102(5):407-414. **(Prospective; 147 patients)**
 86. Demirtas O, Akman L, Demirtas GS, et al. The role of the serum inflammatory markers for predicting the tubo-ovarian abscess in acute pelvic inflammatory disease: a single-center 5-year experience. *Arch Gynecol Obstet.* 2013;287(3):519-523. **(Retrospective; 73 patients)**
 87. Lee MH, Moon MH, Sung CK, et al. CT findings of acute pelvic inflammatory disease. *Abdom Imaging.* 2014;39(6):1350-1355. **(Prospective; 231 patients)**
 88. You JS, Kim MJ, Chung HS, et al. Clinical features of Fitz-Hugh-Curtis syndrome in the emergency department. *Yonsei Med J.* 2012;53(4):753-758. **(Retrospective; 82 patients)**
 89. Romosan G, Valentin L. The sensitivity and specificity of transvaginal ultrasound with regard to acute pelvic inflammatory disease: a review of the literature. *Arch Gynecol Obstet.* 2014;289(4):705-714. **(Systematic review)**
 90. Timor-Tritsch IE, Lerner JP, Monteagudo A, et al. Transvaginal sonographic markers of tubal inflammatory disease. *Ultrasound Obstet Gynecol.* 1998;12(1):56-66. **(Prospective; 77 patients)**
 91. Molander P, Finne P, Sjöberg J, et al. Observer agreement with laparoscopic diagnosis of pelvic inflammatory disease using photographs. *Obstet Gynecol.* 2003;101(5 Pt 1):875-880. **(Prospective; 6 patients)**
 92. Polena V, Huchon C, Varas Ramos C, et al. Non-invasive tools for the diagnosis of potentially life-threatening gynaecological emergencies: a systematic review. *PLoS One.* 2015;10(2):e0114189. **(Systematic review)**
 93. Sam JW, Jacobs JE, Birnbaum BA. Spectrum of CT findings in acute pyogenic pelvic inflammatory disease. *Radiographics.* 2002;22(6):1327-1334. **(Review)**
 94. Jung SI, Kim YJ, Park HS, et al. Acute pelvic inflammatory disease: diagnostic performance of CT. *J Obstet Gynaecol Res.* 2011;37(3):228-235. **(Prospective study with 2 radiologists)**
 95. Li W, Zhang Y, Cui Y, et al. Pelvic inflammatory disease: evaluation of diagnostic accuracy with conventional MR with added diffusion-weighted imaging. *Abdom Imaging.* 2013;38(1):193-200. **(Prospective; 187 patients)**
 96. Eckert LO, Hawes SE, Wolner-Hanssen PK, et al. Endometritis: the clinical-pathologic syndrome. *Am J Obstet Gynecol.* 2002;186(4):690-695. **(Prospective; 152 patients)**
 97. Sellors J, Mahony J, Goldsmith C, et al. The accuracy of clinical findings and laparoscopy in pelvic inflammatory disease. *Am J Obstet Gynecol.* 1991;164(1 Pt 1):113-120. **(Prospective; 95 patients)**
 98. Pichichero ME, Casey JR. Safe use of selected cephalosporins in penicillin-allergic patients: a meta-analysis. *Otolaryngol Head Neck Surg.* 2007;136(3):340-347. **(Meta-analysis)**
 99. Bevan CD, Ridgway GL, Rothermel CD. Efficacy and safety of azithromycin as monotherapy or combined with metronidazole compared with two standard multidrug regimens for the treatment of acute pelvic inflammatory disease. *J Int Med Res.* 2003;31(1):45-54. **(Retrospective; 309 patients)**
 100. Savaris RF, Teixeira LM, Torres TG, et al. Comparing ceftriaxone plus azithromycin or doxycycline for pelvic inflammatory disease: a randomized controlled trial. *Obstet Gynecol.* 2007;110(1):53-60. **(Retrospective; 133 patients)**
 101. McGregor JA, Crombleholme WR, Newton E, et al. Randomized comparison of ampicillin-sulbactam to cefoxitin and doxycycline or clindamycin and gentamicin in the treatment of pelvic inflammatory disease or endometritis. *Obstet Gynecol.* 1994;83(6):998-1004. **(Retrospective; 207 patients)**
 102. Acquavella AP, Rubin A, D'Angelo LJ. The coincident diagnosis of pelvic inflammatory disease and pregnancy: are they compatible? *J Pediatr Adolesc Gynecol.* 1996;9(3):129-132. **(Retrospective; 1205 patients)**

103. Blanchard AC, Pastorek JG 2nd, Weeks T. Pelvic inflammatory disease during pregnancy. *South Med J*. 1987;80(11):1363-1365. **(Case series; 3 patients)**
104. Silva MJ, Florencio GL, Gabiatti JR, et al. Perinatal morbidity and mortality associated with chlamydial infection: a meta-analysis study. *Braz J Infect Dis*. 2011;15(6):533-539. **(Meta-analysis)**
105. Acs N, Banhidly F, Puho EH, et al. Possible association between acute pelvic inflammatory disease in pregnant women and congenital abnormalities in their offspring: a population-based case-control study. *Birth Defects Res A Clin Mol Teratol*. 2008;82(8):563-570. **(Retrospective; 195 patients)**
106. Carter TC, Olney RS, Mitchell AA, et al. Maternal self-reported genital tract infections during pregnancy and the risk of selected birth defects. *Birth Defects Res A Clin Mol Teratol*. 2011;91(2):108-116. **(Retrospective; 12,158 patients)**
107. Ward K, Theiler RN. Once-daily dosing of gentamicin in obstetrics and gynecology. *Clin Obstet Gynecol*. 2008;51(3):498-506. **(Systematic review)**
108. Czeizel AE, Rockenbauer M, Olsen J, et al. A teratological study of aminoglycoside antibiotic treatment during pregnancy. *Scand J Infect Dis*. 2000;32(3):309-313. **(Retrospective; 22,965 patients)**
109. Kirkwood A, Harris C, Timar N, et al. Is gentamicin ototoxic to the fetus? *J Obstet Gynaecol Can*. 2007;29(2):140-145. **(Retrospective; 52 patients)**
110. Czeizel AE, Rockenbauer M. Teratogenic study of doxycycline. *Obstet Gynecol*. 1997;89(4):524-528. **(Retrospective; 18,515 patients)**
111. Cooper WO, Hernandez-Diaz S, Arbogast PG, et al. Antibiotics potentially used in response to bioterrorism and the risk of major congenital malformations. *Paediatr Perinat Epidemiol*. 2009;23(1):18-28. **(Retrospective; 30,049 patients)**
112. Lee V, Tobin JM, Foley E. Relationship of cervical ectopy to chlamydia infection in young women. *J Fam Plann Reprod Health Care*. 2006;32(2):104-106. **(Prospective; 231 patients)**
113. Gray-Swain MR, Peipert JF. Pelvic inflammatory disease in adolescents. *Curr Opin Obstet Gynecol*. 2006;18(5):503-510. **(Review)**
114. Mugo NR, Kiehlbauch JA, Nguti R, et al. Effect of human immunodeficiency virus-1 infection on treatment outcome of acute salpingitis. *Obstet Gynecol*. 2006;107(4):807-812. **(Prospective; 148 patients)**
115. Tepper NK, Steenland MW, Gaffield ME, et al. Retention of intrauterine devices in women who acquire pelvic inflammatory disease: a systematic review. *Contraception*. 2013;87(5):655-660. **(Systematic review)**
116. U.S. selected practice recommendations for contraceptive use, 2013: adapted from the World Health Organization selected practice recommendations for contraceptive use, 2nd edition. *MMWR Recomm Rep*. 2013;62(RR-5):1-60. **(Guidelines)**
117. Kissinger P, Mohammed H, Richardson-Alston G, et al. Patient-delivered partner treatment for male urethritis: a randomized, controlled trial. *Clin Infect Dis*. 2005;41(5):623-629. **(Prospective; 977 patients)**
118. Golden MR, Whittington WL, Handsfield HH, et al. Effect of expedited treatment of sex partners on recurrent or persistent gonorrhea or chlamydial infection. *N Engl J Med*. 2005;352(7):676-685. **(Prospective; 931 patients)**
119. Goyal M, Hersh A, Luan X, et al. National trends in pelvic inflammatory disease among adolescents in the emergency department. *J Adolesc Health*. 2013;53(2):249-252. **(Retrospective NAHMCS database study)**
120. Lis R, Rowhani-Rahbar A, Manhart LE. *Mycoplasma genitalium* infection and female reproductive tract disease: a meta-analysis. *Clin Infect Dis*. 2015;61(3):418-426. **(Meta-analysis)**
121. Manhart LE, Holmes KK, Hughes JP, et al. *Mycoplasma genitalium* among young adults in the United States: an emerging sexually transmitted infection. *Am J Public Health*. 2007;97(6):1118-1125. **(Prospective; 14,322 patients)**
122. Walker J, Fairley CK, Bradshaw CS, et al. *Mycoplasma genitalium* incidence, organism load, and treatment failure in a cohort of young Australian women. *Clin Infect Dis*. 2013;56(8):1094-1100. **(Prospective; 119 patients)**
123. Vandepitte J, Weiss HA, Kyakuwa N, et al. Natural history of *Mycoplasma genitalium* infection in a cohort of female sex workers in Kampala, Uganda. *Sex Transm Dis*. 2013;40(5):422-427. **(Prospective; 119 patients)**
124. Haggerty CL, Totten PA, Astete SG, et al. Failure of cefoxitin and doxycycline to eradicate endometrial *Mycoplasma genitalium* and the consequence for clinical cure of pelvic inflammatory disease. *Sex Transm Infect*. 2008;84(5):338-342. **(Prospective; 682 patients)**
125. Lau A, Bradshaw CS, Lewis D, et al. The efficacy of azithromycin for the treatment of genital *Mycoplasma genitalium*: a systematic review and meta-analysis. *Clin Infect Dis*. 2015;61(9):1389-1399. **(Meta-analysis)**
126. Bradshaw CS, Chen MY, Fairley CK. Persistence of *Mycoplasma genitalium* following azithromycin therapy. *PLoS One*. 2008;3(11):e3618. **(Prospective; 8450 patients)**
127. Mikamo H, Iwasaku K, Yamagishi Y, et al. Efficacy and safety of intravenous azithromycin followed by oral azithromycin for the treatment of acute pelvic inflammatory disease and perihepatitis in Japanese women. *J Infect Chemother*. 2014;20(7):429-435. **(Prospective; 60 patients)**
128. Piyadigamage A, Wilson J. Improvement in the clinical cure rate of outpatient management of pelvic inflammatory disease following a change in therapy. *Sex Transm Infect*. 2005;81(3):233-235. **(Prospective; 147 patients)**
129. Haggerty CL, Hillier SL, Bass DC, et al. Bacterial vaginosis and anaerobic bacteria are associated with endometritis. *Clin Infect Dis*. 2004;39(7):990-995. **(Prospective; 278 patients)**
130. Jaiyeoba O, Lazenby G, Soper DE. Recommendations and rationale for the treatment of pelvic inflammatory disease. *Expert Rev Anti Infect Ther*. 2011;9(1):61-70. **(Review)**
131. Ross JD, Cronje HS, Paszkowski T, et al. Moxifloxacin versus ofloxacin plus metronidazole in uncomplicated pelvic inflammatory disease: results of a multicentre, double blind, randomised trial. *Sex Transm Infect*. 2006;82(6):446-451. **(Prospective; 741 patients)**
132. Malhotra M, Sharma JB, Batra S, et al. Ciprofloxacin-tinidazole combination, fluconazole-azithromycin-secnidazole-kit and doxycycline-metronidazole combination therapy in syndromic management of pelvic inflammatory disease: a prospective randomized controlled trial. *Indian J Med Sci*. 2003;57(12):549-555. **(Prospective; 165 patients)**
133. Dunbar-Jacob J, Sereika SM, Foley SM, et al. Adherence to oral therapies in pelvic inflammatory disease. *J Womens Health (Larchmt)*. 2004;13(3):285-291. **(Prospective; 91 patients)**
134. Kidd S, Moore PC, Kirkcaldy RD, et al. Comparison of antimicrobial susceptibility of urogenital *Neisseria gonorrhoeae* isolates obtained from women and men. *Sex Transm Dis*. 2015;42(8):434-439. **(Prospective; 478 patients)**
135. Chisholm SA, Mouton JW, Lewis DA, et al. Cephalosporin MIC creep among gonococci: time for a pharmacodynamic rethink? *J Antimicrob Chemother*. 2010;65(10):2141-2148. **(Retrospective; 10,002 patients)**
136. Ohnishi M, Saika T, Hoshina S, et al. Ceftriaxone-resistant *Neisseria gonorrhoeae*, Japan. *Emerg Infect Dis*. 2011;17(1):148-149. **(Case report)**
137. Unemo M, Golparian D, Nicholas R, et al. High-level cefixime- and ceftriaxone-resistant *Neisseria gonorrhoeae* in France: novel penA mosaic allele in a successful international clone causes treatment failure. *Antimicrob Agents Chemother*. 2012;56(3):1273-1280. **(Case report)**

138. Watchirs Smith LA, Hillman R, Ward J, et al. Point-of-care tests for the diagnosis of *Neisseria gonorrhoeae* infection: a systematic review of operational and performance characteristics. *Sex Transm Infect.* 2013;89(4):320-326. (Systematic review)
139. van Dommelen L, van Tiel FH, Ouburg S, et al. Alarming poor performance in *Chlamydia trachomatis* point-of-care testing. *Sex Transm Infect.* 2010;86(5):355-359. (Prospective; 772 patients)
140. Tabrizi SN, Unemo M, Golparian D, et al. Analytical evaluation of GeneXpert CT/NG, the first genetic point-of-care assay for simultaneous detection of *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. *J Clin Microbiol.* 2013;51(6):1945-1947. (In vitro study)
141. Guy RJ, Natoli L, Ward J, et al. A randomised trial of point-of-care tests for chlamydia and gonorrhoea infections in remote Aboriginal communities: Test, Treat AND GO- the "TTANGO" trial protocol. *BMC Infect Dis.* 2013;13:485. (Brief report on a study protocol)
142. World Health Organization. Guidelines for the management of sexually transmitted infections. Geneva, Switzerland. 2003. (Guideline)
143. Hosenfeld CB, Workowski KA, Berman S, et al. Repeat infection with chlamydia and gonorrhea among females: a systematic review of the literature. *Sex Transm Dis.* 2009;36(8):478-489. (Systematic review)

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1. Which of following statements regarding Fitz-Hugh-Curtis syndrome is TRUE?
 - a. It is unrelated to PID.
 - b. It is characterized by right upper abdominal pain in a patient with PID.
 - c. It can be ruled out with normal transaminase levels.
 - d. It is a consequence of hepatotoxicity from doxycycline.
2. A 26-year-old woman presents to the ED with left lower pelvic pain for 5 days. On examination, you find left adnexal tenderness without guarding or rebound or abnormal vaginal discharge. She denies fevers, chills, nausea, vomiting, dysuria, flank pain, diarrhea, or constipation. She is sexually active with 1 partner and uses oral contraceptive pills for contraception. Her laboratory results, including a urinalysis, are unremarkable. Her pelvic ultrasound and CT are normal. What should be your management and disposition?
 - a. Reassurance and careful return instructions.
 - b. Nonsteroidal anti-inflammatory drugs and referral to a gynecologist for endometriosis.
 - c. Treatment for PID and referral to a gynecologist for 48-hour follow-up.
 - d. MRI for evaluation of discogenic cause of her symptoms.
3. Which of these statements regarding the wet mount in the evaluation of the patient with PID is TRUE?
 - a. The presence of trichomoniasis rules out PID as the cause of the pain.
 - b. A positive whiff test rules out the diagnosis of PID.
 - c. The wet mount is only indicated if there is copious vaginal discharge.
 - d. Identification of white blood cells on the wet mount is suggestive of inflammation of the lower genital tract.
4. Which of the following statements about the role of ultrasound in the evaluation of PID is TRUE?
 - a. Ultrasound is essential for making the diagnosis of PID.
 - b. Ultrasound is the second-line imaging modality in the evaluation of the nonpregnant female with likely gynecological pelvic pain.
 - c. There are no ultrasound findings that are diagnostic for PID.
 - d. Ultrasounds are often normal in patients with mild-to-moderately severe PID.

5. Which of these statements about the appropriate disposition of patients with PID is TRUE?
 - a. The majority of patients should be admitted and given parenteral antibiotics regardless of the severity of the disease.
 - b. The majority of patients with mild-to-moderate disease can be managed with outpatient oral antibiotics.
 - c. The majority of patients with TOA should be discharged with oral medication and strict return instructions.
 - d. The patient's social situation should play no role in determining the appropriate disposition of the patient.

6. What is the first-line therapy for the outpatient treatment of PID in a woman with no allergies?
 - a. Ceftriaxone 250 mg IM x 1 dose and doxycycline 100 mg PO bid x 14 days
 - b. Doxycycline 100 mg PO bid x 14 days
 - c. Ceftriaxone 250 mg IM x 1 dose and azithromycin 1 g PO x 1 dose
 - d. Moxifloxacin 500 mg PO x 14 days

7. Which of these comorbidities is an accepted indication for admission for observation and parenteral antibiotics in a patient with PID?
 - a. Pregnancy
 - b. Age < 18 years
 - c. Leukocytosis > 18 x 10⁹/L
 - d. Penicillin allergy

8. Which of the following is a risk factor for infection with *Mycoplasma genitalium*?
 - a. Long-term IUD use
 - b. Having a male sexual partner who has sex with men
 - c. Vaginal douching
 - d. Living in the Northeastern United States

9. Which of the following statements about the use of metronidazole in PID is TRUE?
 - a. There is conclusive evidence that metronidazole should be used in all patients with PID.
 - b. Metronidazole or other anaerobic coverage should be added in patients with TOA.
 - c. Metronidazole is an effective monotherapy in the treatment of PID.
 - d. The addition of metronidazole does not modify the likelihood of medication adherence in PID.

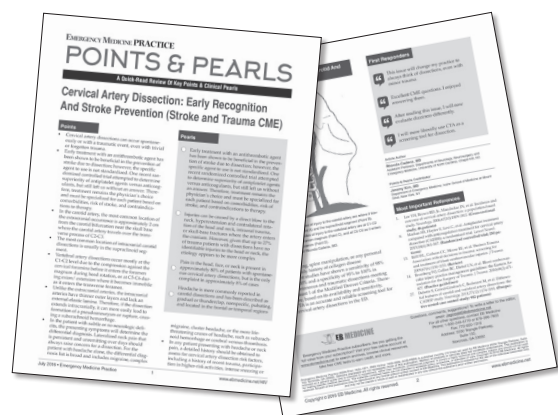
10. Which of the following statements about the appropriate follow-up for a patient with PID who is being managed with oral antibiotics is TRUE?
 - a. Patients should follow up with their primary care physician only if they have not improved after the full 14 days of treatment.
 - b. Patients should be re-evaluated in 48 to 72 hours to ensure clinical response.
 - c. All patients should be referred to a surgeon for the evaluation of possible surgical pathology.
 - d. Patients should follow up with their primary care physician in 3 to 5 months.

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— Andy Jagoda, MD, FACEP; Editor-in-Chief



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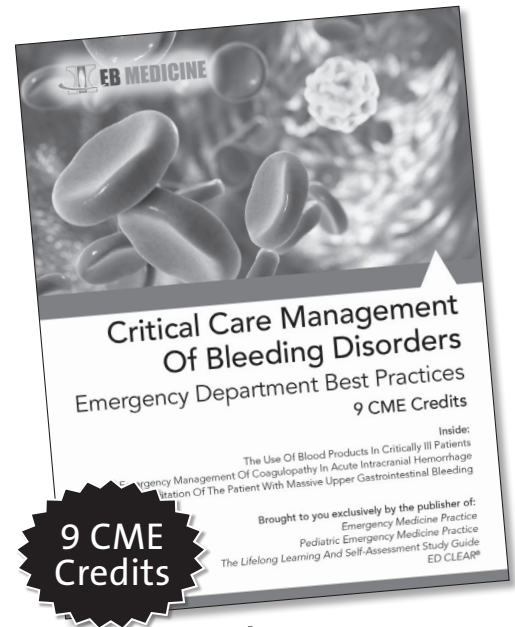
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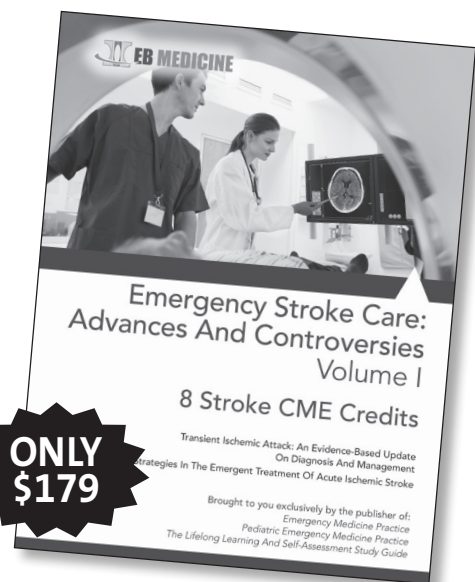
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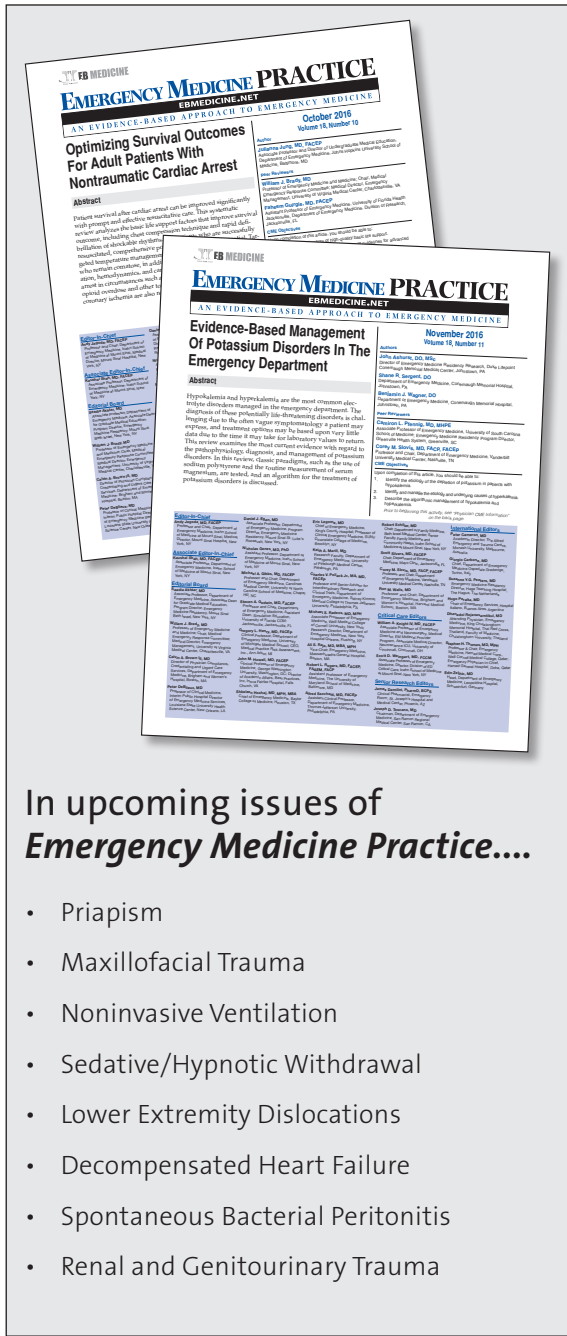
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