REVIEW ARTICLE

Pelvic inflammatory disease: clinical feature, risk factors, treatment, and prevention

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ABSTRACT

Pelvic Inflammatory Disease (PID) is a significant public health concern with severe repercussions for reproductive health, including ectopic pregnancies, chronic pelvic pain, and tubal infertility. This study provides a comprehensive examination of PID, covering its clinical characteristics, risk factors, etiological agents, diagnostic methods, treatment options, and preventive strategies. PID encompasses a variety of inflammatory disorders of the upper female genital tract caused by pathogens such as Neisseria gonorrhoeae, Chlamydia trachomatis, vaginal flora, and other bacteria. Despite a decline in PID cases associated with N. gonorrhoeae and C. trachomatis, these pathogens remain substantial contributors. Key risk factors include age under 25, multiple sexual partners, unprotected sex, early sexual debut and a history of sexually transmitted diseases (STDs) or previous PID. Diagnosis relies primarily on clinical evaluation, with symptoms ranging from mild pelvic discomfort to severe abdominal pain, complicating timely diagnosis. Effective treatment involves the use of empirical broad-spectrum antibiotics. Preventive strategies emphasize early detection and treatment of STIs, adherence to screening guidelines, and measures to prevent recurrent PID episodes. Effective management of PID requires early detection, prompt intervention, and comprehensive preventive measures targeting both initial and recurrent cases. Adherence to STI screening and treatment protocols is crucial in reducing PID incidence and associated complications. Continued research is vital to enhance the understanding of pathogenic mechanisms and optimize treatment protocols, thereby improving the quality of life for women affected by PID.

KEYWORDS

PID; STI; Chlamydia trachomatis; Neisseria gonorrhoeae; Reproductive health sequelae

INTRODUCTION

Pelvic Inflammatory Disease (PID) is an umbrella term for a variety of inflammation-related disorders affecting the upper portion of the female genital tract, including tub-ovarian abscess, endometritis, pelvic peritonitis, and salpingitis. (1,2) Historically, the primary etiological agents responsible for sexually transmitted infections (STIs) were identified as *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. Current epidemiological data indicate a reduction in PID incidence linked to both infections, although approximately 50% of acute PID diagnoses still test positive for one of these organisms (3, 4). However, the microbiological landscape of PID extends beyond STIs. Endogenous vaginal flora, including enteric gram-negative rods, *Haemophilus influenzae*, *Gardnerella vaginalis*, facultative and strict anaerobes, and *Streptococcus agalactiae*, also contribute to the aetiology of PID (5). Additionally, *Ureaplasma urealyticum*, *Mycoplasma hominis*, *Trichomonas vaginalis*, and cytomegalovirus (CMV) may influence certain cases (6). Although the presence of *Mycoplasma genitalium* in the lower genital tract does not always correspond with a significant rise in PID frequency, emerging evidence suggests that this bacterium may be involved in PID and is associated with milder clinical presentations (7).

The Centres for Disease Control and Prevention (CDC) estimates that more than a million women

are affected by PID each year, including many cases that go undetected. The incidence of PID is particularly concerning due to the severe consequences it can lead to, such as pregnancy complications, tubal infertility, and chronic pelvic pain. The risk of severe consequences is greatly increased in patients who are misdiagnosed or receive inadequate treatment for PID (8). Health economics research underscores the significant impact of PID on quality of life, with young women willing to forgo one to two years of their life expectancy to avoid PID and its associated complications. This finding highlights the seriousness of the disease's repercussions. The diverse clinical presentations of PID pose a challenge for accurate diagnosis. While some patients present with severe symptoms, including excruciating abdominal pain necessitating surgical intervention, others with subclinical PID may remain asymptomatic but still experience upper reproductive tract inflammation (9). This study aims to elucidate current knowledge regarding PID, including its clinical characteristics, risk factors, diagnosis, treatment, and prevention.

Risk Factors and Pathophysiology

PID significantly impacts reproductive health, often resulting in premature births, infertility, and chronic pelvic pain. The body of knowledge about PID's risk factors, etiological agents, and pathophysiological pathways is vast and well-documented in both recent and historical literature.

Risk Factors

Several risk factors for PID have been consistently identified in the literature (10,11):

Age Younger than 25 Years: Younger women are more susceptible due to biological predisposition and behavioural factors. Cervical ectopy in younger women creates a favourable environment for STI microorganisms.

New or Multiple Sex Partners: Increased exposure to STIs raises the risk of PID.

Unprotected Sexual Intercourse: Lack of barrier protection facilitates pathogen transmission.

Intercourse with a Symptomatic Partner: Partners with symptomatic STIs increase the likelihood of transmission and subsequent PID.

Early Onset of Sexual Activity: Initiating sexual activity before age 15 correlates with a higher lifetime risk of STIs and PID.

History of STIs or PID: Previous episodes of PID or STIs increase vulnerability to complications and recurrent infections.

Pathogens and Pathophysiology

N. gonorrhoeae and *C. trachomatis* have historically been the primary pathogens linked to PID, known for compromising the cervical barrier and enabling the ascension of bacteria into the upper genital tract. However, PID is not restricted to these specific pathogens:

Mycoplasma genitalium: Emerging evidence suggests its relevance in PID, although its prevalence and pathogenic significance are still being evaluated. Research indicates that *M. genitalium* is associated with reproductive tract infections (12).

Gardnerella vaginalis and Ureaplasma urealyticum: The involvement of these microorganisms in PID is still under study, with research yielding mixed results on their pathogenic potential.

BV-Associated Anaerobes: Bacterial vaginosis (BV), marked by an imbalance in vaginal microbiota, is commonly observed in women with PID. Anaerobic bacteria associated with BV have been linked to PID, though it is unclear whether treating BV reduces PID incidence. Further studies are needed to validate these findings (13).

Mechanisms of Infection Spread

The primary mechanism of PID is the ascension of bacteria from the lower to the upper genital tract. However, alternative pathways are documented in the literature (14):

Lymphatic Spread: Infection can spread through the lymphatic system from adjacent tissues, such as the parametrium.

Hematogenous Spread: Though rare, hematogenous dissemination, as seen in tuberculosis, can result in PID. This is particularly relevant in endemic regions or immunocompromised individuals.

Changing aetiology of PID:

Over the past 70 years, changes in pathogen prevalence and advancements in diagnostic tools have profoundly impacted the etiological landscape of pelvic inflammatory disease (PID). In the 1950s, PID was primarily associated with Mycobacterium tuberculosis and Neisseria gonorrhoeae. By the 1980s, gonococcal infections were recognized as the leading cause of PID, even before precise screening methods for Chlamydia trachomatis were developed. The rates of identifying N. gonorrhoeae and C. trachomatis in PID patients vary significantly depending on the sampling site (cervix, endometrium, ovaries, or peritoneum) and the screening method used (culture or nucleic acid amplification tests, NAATs). Interestingly, more than half of women with histologically confirmed PID and clinical indications do not test positive for these STI pathogens, even when sensitive NAATs are used (15). A recent clinical trial in the US found that only 25% of symptomatic PID patients were

positive for either STI infection (4). Despite this, gonococcal and chlamydial infections are still frequently highlighted in patient information as the primary causes of PID (16, 17).

In addition to N. gonorrhoeae and C. trachomatis, other etiologic agents are also responsible for PID. Anaerobic and facultative bacteria, particularly those associated with gastrointestinal infections, Mycoplasma genitalium, and bacterial vaginosis (BV), have been identified. Evidence suggests that facultative bacteria and BV-associated anaerobes can migrate from the vagina to the endometrium and fallopian tubes, a phenomenon documented over forty years ago. In women with BV, anaerobic gram-negative rods and cocci are often found in endometrial cultures, correlating with histological signs of endometritis (18).
Studies have shown that adding metronidazole to acute PID therapy results in fewer bacteria associated with bacterial vaginosis in the endometrium one month after treatment (4). However, there is limited information on the presence of these pathogens in the fallopian tubes. A study in Kenya using 16S rRNA sequencing found bacterial sequences, such as Atopobium vaginae and Leptotrichia spp., in 25% of women with acute salpingitis, but not in the control group undergoing tubal ligations (19). Longitudinal studies have demonstrated a doubling of incident PID in women harbouring BV-associated bacteria in the vagina (20).

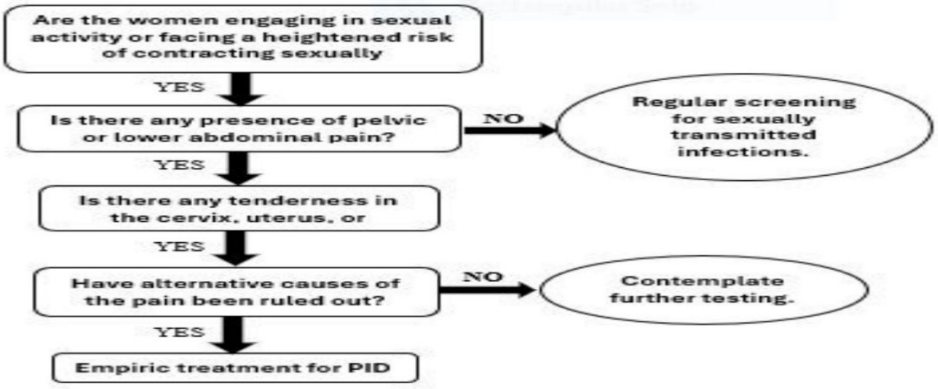
Quantitative polymerase chain reaction (PCR) identified A. vaginae, Sneathia, BVAB-TM7, Megasphaera, Eggerthella-like bacterium, Mobiluncus, Gardnerella vaginalis, BVAB1, BVAB2, Mageeibacillus indolicus, Prevotella timonensis, and Prevotella amnii in vaginal samples from women diagnosed with symptomatic PID in a controlled trial (21). Additionally, there was a threefold increase in prevalent and incident PID cases linked to cervical M. genitalium (22). Although M. genitalium has demonstrated toxicity in animal models, its role in human PID remains unclear. Interestingly, metronidazole therapy, though ineffective against mycoplasmas, decreased M. genitalium levels in follow-up biopsies, suggesting that BV-associated microbiota may create a favourable environment for M. genitalium infections (4). Furthermore, bacteria typically found in the respiratory and gastrointestinal tracts have been identified in women with PID, suggesting probable transfer from the oropharynx or rectum during sexual activity. NAAT detected N. gonorrhoeae in 4% of patients, but Hemophilus influenzae was found in endometrial biopsy cultures of 2% of women clinically diagnosed with PID (4). Streptococcus pyogenes has also been connected to a few instances of salpingitis.
Because of the wide range of symptom intensity and variability, PID is often underdiagnosed. Notably, some people never show any symptoms, which makes diagnosis more difficult. Histological evidence of PID is frequently found in women with tubal factor infertility, even in cases where PID had not been clinically diagnosed previously (23). The hallmark of PID in sexually active women is the sudden onset of lower abdomen or pelvic discomfort (11). However, the presentation can be subtle, with symptoms including dysuria, abnormal vaginal discharge, abnormal uterine bleeding, mild bilateral lower abdominal pain that worsens during sexual activity, and increased frequency of urination. Fever is not always present and is not a primary symptom.

Furthermore, perihepatitis, also referred to as Fitz-Hugh-Curtis syndrome, causes irritation and deposits in the liver capsule, resulting in discomfort in the right upper quadrant that worsens with mobility and breathing. This condition, which represents the complex and varied clinical presentations of PID, highlights the difficulties in making an early and correct diagnosis (11).

Usually discovered clinically, PID is treated with scanning and more invasive therapies reserved for cases of uncertainty or concerns about potential consequences such as tub-ovarian abscesses. Thus, doctors should start PID evaluation and management unless another diagnosis is more likely in sexually active women under 25 or older who are at risk for STIs and who arrive with lower abdomen or pelvic discomfort and fit the criteria shown in Figure 1 (11).

The majority of PID patients have symptoms that are similar to those of UTIs. Mucopurulent discharge and an increased white blood cell count (wet prep) on saline microscopy—which is defined as a ratio of one white blood cell to every epithelial cell—are examples of clinical signs. It is critical to reevaluate the differential diagnosis for lower abdominal discomfort in the absence of particular clinical indications (24–26). Presumptive PID diagnosis may lead to the start of empirical antibiotic therapy, especially for individuals with mild symptoms.

Figure 1: Algorithm for Therapeutic Determination in Pelvic Inflammatory Disease Management.



The specificity of PID diagnosis is increased by adding more diagnostic criteria. This method might, however, lessen sensitivity, which could result in an underdiagnosis of PID cases. Table 2 (11) provides a full summary of the diagnostic results that support the PID diagnosis.

Table 1: Differential Diagnoses and Diagnostic Approaches for Pelvic Pain.

Condition	Clinical Manifestations	Diagnostic Modalities
Acute Appendicitis	Indicators include peritoneal signs, periumbilical or right lower quadrant pain, and symptoms such as vomiting/anorexia	Computed tomography (CT) or ultrasonography (US)
Ectopic Pregnancy/Rupture	Presents with hypotension or anaemia, missed menstrual periods, positive pregnancy test, and unilateral pelvic pain	Transvaginal ultrasonography (TVUS)
Endometriosis	Characterized by dyschezia, dysmenorrhea, inter-menstrual bleeding, and dyspareunia	Confirmatory diagnosis via laparoscopy with histological biopsy
Endometritis	Acute: fever, pelvic pain, vaginal discharge, Chronic: pelvic discomfort, vaginal spotting, leukorrhea	Endometrial biopsy
Ovarian Cyst/Torsion/Rupture	Sudden onset of severe unilateral pain	Transvaginal ultrasonography (TVUS)
Tubo-Ovarian Abscess	Manifestations include fever, palpable pelvic/adnexal mass on bimanual examination, and unilateral pelvic pain	Transvaginal ultrasonography (TVUS)
Ureteral Calculus	Symptoms include dysuria, fever, nausea, vomiting, hematuria, and pain in the flank, pelvic, or abdominal regions	CT scan, plain radiography, ultrasonography, urinalysis
Urinary Tract Infection	Increased urinary frequency, dysuria, hematuria, and mid- or bilateral pelvic pain	Urinalysis with microscopy, urine culture

** If there is no observed clinical improvement within 72 hours following the initiation of standard therapeutic protocols for PID, it is imperative to undertake a thorough re-evaluation to explore alternative differential diagnoses.*

Table 2: Enhanced Diagnostic Criteria for Increased Specificity in Pelvic Inflammatory Disease Identification.

Criteria	Description
Cervical examination reveals mucopurulent discharge or notable friability	Observation reveals a copious, purulent exudate emanating from the cervix, accompanied by pronounced friability resulting in effortless haemorrhage upon minimal manipulation during clinical evaluation.
Elevated C-reactive protein	Elevated C-reactive protein (CRP) concentrations in the bloodstream indicate a systemic inflammatory response.
Elevated erythrocyte sedimentation rate	An accelerated erythrocyte sedimentation rate (ESR) over one hour signifies an inflammatory process.
Saline microscopy of vaginal fluid demonstrates a substantial leukocyte infiltration	A saline wet mount of vaginal fluid revealed a markedly elevated leukocyte count, characterized by a leukocyte-to-epithelial cell ratio of at least 1:1 or exceeding 15 leukocytes per high-power field.
An oral temperature exceeding 101°F (38.3°C) has been recorded	Oral thermometry has detected a heightened body temperature, consistent with febrile conditions.
Positive test results for Neisseria gonorrhoeae or Chlamydia trachomatis have been obtained	Laboratory diagnostics have verified the presence of an infection caused by either Neisseria gonorrhoeae or Chlamydia trachomatis.

Treatment of PID:

To effectively combat harmful microorganisms, PID treatment strategies must employ a comprehensive, broad-spectrum approach. Although randomized clinical trials have demonstrated that various intravenous and oral antibiotic regimens are effective in achieving short-term medical and biological cures (27, 28), there remains a lack of research comparing these regimens' relative efficacy in eradicating infections in the uterus and fallopian tubes, as well as their impact on long-term complications such as tubal infertility and ectopic pregnancy post-treatment (3). Negative endocervical tests for Neisseria gonorrhoeae and Chlamydia trachomatis do not exclude upper genital tract infections, making it essential to target these organisms in all PID treatment regimens.

Anaerobic microbes have been identified in the upper genital tracts of women with PID, and in vitro studies have shown that certain anaerobes, such as Bacteroides fragilis, can damage tubal and epithelial cells. Given the frequent co-occurrence of bacterial vaginosis (BV) and PID (4, 29), adding metronidazole to both intramuscular (IM) and oral PID regimens significantly enhances the eradication of anaerobic microbes from the upper genital tract (4). Regimens that do not provide anaerobic coverage should not be prioritized until they demonstrate comparable efficacy in preventing long-term complications, such as preterm births and infertility. Treatment must commence as soon as a presumptive diagnosis is made, as delayed intervention can lead to lasting complications. Early antibiotic use is recommended. Both parenteral and oral regimens show similar effectiveness for women with mild-to-moderate PID symptoms. Hospitalisation decisions should be based on clinical judgment and specific indicators, such as the potential need for surgery, tubo-ovarian abscesses, pregnancy, severe illness, inability to adhere to outpatient oral regimens, or lack of response to oral antibiotics. The lack of evidence indicating better outcomes for hospitalised adolescents with acute PID compared to those treated as outpatients emphasizes the need to apply the same hospitalisation criteria for adolescent PID patients as for adults.

Parenteral treatment:

Randomized clinical trials have unequivocally validated the efficacy of parenteral regimens in managing PID, corroborating findings from various studies (4, 27, 30). However, transitioning to oral therapy following clinical improvement requires careful consideration, typically beginning within 24 to 48 hours after clinical signs of improvement. Specifically, for patients with tubo-ovarian abscesses, an inpatient stay exceeding twenty-four hours is recommended to ensure close monitoring and treatment. This nuanced strategy, informed by empirical evidence and clinical experience, underscores the importance of tailored therapeutic interventions in PID management (Table 3)..

Table 3: Parenteral regimen for PID.

Regimen	Dosage/Frequency	Route
Ceftriaxone	1 g/day	Intravenous
Doxycycline	100 mg/12 hrs	Oral/IV
Metronidazole	500 mg/12 hrs	Oral/IV
OR		
Cefotetan	2 g/12 hrs	Intravenous
Doxycycline	100 mg/12 hrs	Oral/IV
OR		
Cefoxitin	2 g/ 6 hrs	Intravenous
Doxycycline	100 mg/ 12 hrs	Oral/IV

Since intravenous (IV) infusion might be uncomfortable, it is best to provide doxycycline orally whenever possible. It's important to remember that metronidazole and doxycycline have similar absorption when administered orally and intravenously. When there is no serious sickness or tubo-ovarian abscess, oral metronidazole can be a good substitute for IV medication. It is recommended to make a smooth transition from parenteral medication to oral therapy after clinical improvement. This transition entails a regimen comprising doxycycline at a dosage of 100 mg administered twice daily alongside metronidazole dosed at 500 mg twice daily. This oral regimen is intended to span 14 days, ensuring the comprehensive completion of antimicrobial therapy. Such a transition not only enhances patient comfort but also underscores the judicious utilization of therapeutic modalities to optimize treatment outcomes in PID management.

Alternative Parenteral regimen:

For the management of PID, there is insufficient evidence to recommend the use of substitute injectable second- or third-generation cephalosporins, including ceftizoxime or cefotaxime. Given their relatively diminished activity against anaerobic bacteria compared to cefotetan or cefoxitin, the adjunctive use of metronidazole warrants consideration to augment antimicrobial coverage. In clinical studies, ampicillin-sulbactam in combination with doxycycline has shown broad-spectrum effectiveness (31), including protection against anaerobes, Neisseria gonorrhoeae, and Chlamydia trachomatis, especially in instances of tuboovarian

abscesses. Additionally, studies using azithromycin alone or in conjunction with metronidazole have shown encouraging brief clinical cure rates (32).
Patients who show clinical improvement after 24–48 hours of starting the alternate parenteral regimen consisting of clindamycin and gentamicin may switch over to either clindamycin or doxycycline for the remaining 14 days of treatment. However, in the presence of a tub-ovarian abscess, augmenting the regimen with either clindamycin or metronidazole becomes imperative to bolster anaerobic coverage. This strategic adjustment ensures comprehensive therapeutic efficacy in combating PID, aligning with the principles of precision medicine and tailored therapeutic interventions (Table 4).

Table 4: Alternative Parenteral Regimens.

Alternative Parenteral Regimens	Dosage/Frequency	Route
Ampicillin-sulbactam + Doxycycline	Ampicillin-sulbactam: 3 g/6 hrs	Intravenous
	Doxycycline: 100 mg/12 hrs	Oral/IV
Clindamycin + Gentamicin	Clindamycin: 900 mg/8 hrs	Intravenous
	Every eight hours, gentamicin loading doses of 2 mg per kilogramme of body weight are administered intravenously or intramuscularly. These should be followed by maintenance dosages.	Intravenous/IM

Intramuscular/Oral treatment:
Studies show that oral or intramuscular (IM) treatment can successfully manage mild-to-moderate acute PID in women, with equivalent clinical results to those treated with intravenous (IV) medication (33). In cases where initial IM or oral therapy fails to yield desired results within a 72-hour window, it is imperative to conduct a reassessment to confirm the diagnosis and promptly transition to IV therapy as necessary. The regimens outlined provide extensive protection against common causal factors of PID; however, the best cephalosporin option is yet unknown. When compared to ceftriaxone, cefoxitin, a second-generation cephalosporin, exhibits better anaerobic coverage. Cefoxitin has been effective in helping women with PID achieve good short-term clinical results when given in combination with probenecid and doxycycline. On the other hand, ceftriaxone offers better protection against Neisseria gonorrhoeae. Adding metronidazole to these treatment plans increases the range of anaerobic bacterial coverage, and bacterial vaginosis (BV), a disease that often coexists with PID, is successfully treated. See Table 5 for a detailed regimen.

Table 5: Intramuscular/oral regimen for PID

Regimen Components	Dosage and Administration
Ceftriaxone	500 mg IM in a single dose*
+	
Doxycycline	100 mg orally twice daily for 14 days
+	
Metronidazole	500 mg orally twice daily for 14 days
OR	
Cefoxitin	2 g IM in a single dose, concurrently with 1 g Probenecid orally in a single dose
+	
Doxycycline	100 mg orally twice daily for 14 days
+	
Metronidazole	500 mg orally twice daily for 14 days
OR	
Other Parenteral Third-Generation Cephalosporin (e.g., ceftizoxime or cefotaxime)	
+	
Doxycycline	100 mg orally twice daily for 14 days
+	
Metronidazole	500 mg orally twice daily for 14 days

**For individuals with a body weight exceeding 150 kg (~300 lbs.) and a confirmed diagnosis of gonococcal infection, a ceftriaxone dosage of 1 g is recommended.*

Alternative Intramuscular or Oral Regimens for PID: Quinolone-containing regimens are typically discouraged for the therapy of PID due to the lack of published data supporting the effectiveness of these medications in treating PID and the emergence of quinolone-resistant Neisseria gonorrhoeae strains. However, different treatment options may be taken into consideration for

individuals who have a known cephalosporin allergy, a low community frequency of quinolone-resistant gonorrhoea, and an expectation of adhering to follow-up.

Levofloxacin: 500 mg taken orally once a day for 14 days, along with 500 mg taken orally twice a day for Metronidazole.

Moxifloxacin: 400 mg taken orally once a day for 14 days.

Azithromycin: 500 mg intravenously given for one or two doses at first, then 250 mg taken orally for seven days. Alternately, in conjunction with 500 mg of metronidazole taken three times a day for a period of 12 to 14 days.

Moxifloxacin is the drug of choice for treating Mycoplasma genitalium infections; however, it is not yet clear if PID treatment plans must include M. genitalium coverage. Gonorrhoea diagnostic testing is necessary before starting therapy, and patient care should go forward following the results (34). Antimicrobial susceptibility test findings should direct treatment methods following confirmation of a positive gonorrhoea culture. Consultation with an infectious disease expert is particularly indicated in cases of quinolone-resistant Neisseria gonorrhoeae or when susceptibility testing is not available (e.g., depending entirely on NAAT).

The goal of additional management measures is to reduce the spread of illness. When a woman is diagnosed with PID, she should wait to have children until after therapy is over, her symptoms are under control, and her spouse is receiving treatment. All PID patients should have comprehensive testing for syphilis, chlamydia, HIV, and gonorrhoea; however, the usefulness of Mycoplasma genitalium testing is still up for debate. It is acceptable to use contraceptive techniques throughout therapy. Follow-up care is critical; a decrease in fever, stomach discomfort, and uterine, adnexal, and cervical motion soreness are indicators of clinical improvement, which should occur three days after outpatient intramuscular or oral medication is started. Reassessment is necessary if the condition doesn't improve, since it can mean hospitalisation for more antibiotic adjustments and diagnostic testing, which might include a diagnostic laparoscopy if necessary. Regardless of partner treatment status, women treated for gonococcal or chlamydial PID should be retested three months after treatment (35). If retesting after three months is not feasible, testing should be placed at the patient's subsequent appointment within a year after therapy.

Evaluation and testing for gonorrhoea and chlamydia in individuals who have had intercourse within the last 60 days is part of managing sexual partners. Presumptive therapy should be given to partners, especially if they are asymptomatic or had sex during this period. If partner connection is delayed, expedited partner treatment (EPT) may be taken into consideration (36). Until both parties have finished therapy and all symptoms have subsided, partners should avoid having sex.

Special Considerations:

Drug Intolerance, Adverse Reactions, and Allergies

Penicillin has the biggest cross-reactivity risk when compared to first-generation cephalosporins, although this risk is minimal when it comes to most second-generation (like cefoxitin) and all third-generation (like ceftriaxone) cephalosporins (37–40).

Being pregnant

There is an increased risk of maternal morbidity and premature birth for expectant mothers suspected of having PID. Hospitalisation and intravenous antibiotic treatment should be part of the management plan, under the direction of an infectious disease expert.

HIV/AIDS Co-Infection

It's yet unknown how PID clinical manifestations differ in women living with and without HIV. Observational data point to the possibility of more surgical treatments for PID and HIV patients, although overall symptomatology and treatment response are in line with existing guidelines. Vigilance may be necessary due to the higher prevalence of M. hominis and streptococcal infections in women living with HIV; however, additional study is required to determine whether more aggressive therapy is necessary.

Intrauterine Equipment

Intrauterine device (IUD) usage temporarily elevates the risk of PID, especially in the first three weeks after installation (41). IUD removal is not required in PID instances, however quick treatment and careful observation are recommended. If after 48–72 hours after starting medication, there is no clinical improvement, the removal of the IUD should be considered. Research shows similar treatment results for women who remove or keep nonhormonal or copper-containing IUDs during PID therapy (42); however, there is little information available for levonorgestrel-releasing IUDs.

Preventing PID:

Preventing initial episodes and minimising recurrence are the two key techniques involved in effective PID prevention. In particular, Chlamydia trachomatis, which is strongly linked to infertility, puts women with a history of PID at higher risk of contracting other STDs. As a result, thorough STI control is essential to reducing the incidence of both first and recurring PID episodes.

Preventing the First Episode of PID

The United States Preventive Services Task Force (USPSTF) and the CDC have established guidelines for strict adherence to effectively prevent initial PID. These guidelines centre on the timely diagnosis and therapeutic management of STIs. According to the 2015 Sexually Transmitted Diseases Treatment Guidelines from the CDC, sexually active women under 25 who are also older women who are at higher risk—for example, those who have multiple or new partners, partners who have concurrent partners, or known STI contacts—should undergo annual screenings for gonorrhoea and chlamydia. Women who engage in high-risk behaviours such as many intimate partners, illicit sexual activity, drug use, or a history of STIs should undergo periodic testing for Trichomonas vaginalis. Rescreening three months after starting STI therapy is recommended, especially in areas with high incidence or if there are new behavioural hazards.

Although national screening adherence is still below ideal levels, evidence from randomised controlled trials highlights that C. trachomatis screening can significantly reduce PID incidence. According to an analysis of data from the National Survey of Family Growth (NSFG) conducted between 2006 and 2010, only 40% of sexually active women between the ages of 15 and 21 had a C. trachomatis screening. Further worries arise regarding STIs like Mycoplasma genitalium, which do not have commercial testing available in the US. Research elucidating the relationship between PID and T. vaginalis and M. genitalium underscores the necessity of asymptomatic STI screening to reduce national STI loads and PID risks. Subclinical PID is lessened by screening and receiving early STI therapy; its morbidity is similar to that of acute PID. Although it is difficult to estimate the frequency of subclinical PID, research indicates that it has a significant impact; cross-sectional studies employing endometrial biopsies found subclinical PID in 27% of cases with C. trachomatis and 26% of cases with N. gonorrhoeae. These results highlight the fallopian tube-destructive potential of subclinical PID, which is similar to symptomatic PID. This calls for early treatment and asymptomatic STI screening.

Preventing Recurrent PID

Preventing recurrent PID is a public health imperative due to the significant reproductive complications associated with repeated episodes. Recurrent PID considerably heightens the risk of infertility. In a Scandinavian inpatient cohort study (1960-1984), each subsequent PID episode doubled the risk of infertility. Data from the PEACH study corroborates these findings, indicating women with recurrent PID are nearly twice as likely to experience infertility and over four times more likely to report chronic pelvic pain (CPP). Recurrent PID is relatively common. The PEACH study reported that within three years of initial PID diagnosis, 14.5% of participants experienced recurrence, increasing to over 21% within seven years. These statistics underscore the need for enhanced clinical interventions to ensure adequate treatment and prevent recurrent PID.

A comprehensive approach to PID prevention involves stringent adherence to STI screening guidelines, early and effective treatment of diagnosed STIs, and targeted efforts to prevent recurrent episodes. By addressing both primary and recurrent PID prevention, we can mitigate the associated reproductive health sequelae and improve overall outcomes for affected women.

Conclusion

PID poses significant challenges in women's health, with diverse pathogens beyond traditional sexually transmitted ones implicated. Early diagnosis and comprehensive treatment are crucial to prevent long-term complications like infertility. Treatment focuses on broad-spectrum antimicrobial coverage, transitioning from parenteral to oral therapy as appropriate. Partner treatment and follow-up testing are vital for preventing recurrent PID and reducing transmission. Prevention strategies entail early STI detection, adherence to screening guidelines, and targeted interventions against recurrence. By addressing both primary and recurrent PID prevention, healthcare providers can mitigate reproductive health risks and enhance outcomes for affected individuals.

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