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PELVIC INFLAMMATORY DISEASE (PID): A COMPREHENSIVE CLINICAL

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Review

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Introduction

Pelvic inflammatory disease (PID) represents a spectrum of infectious and inflammatory disorders affecting the upper female genital tract, encompassing the uterus, fallopian tubes, ovaries, and adjacent pelvic structures . Characterized primarily by ascending spread of microorganisms from the lower genital tract, PID remains one of the most consequential reproductive health conditions among women of reproductive age worldwide. Despite declining incidence rates in many developed regions—attributed largely to improved screening for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*—the condition continues to account for substantial morbidity, including tubal factor infertility, ectopic pregnancy, and chronic pelvic pain .

The clinical significance of PID extends beyond acute infectious symptomatology. Even mild or subclinical episodes can precipitate permanent structural damage to the fallopian tubes, underscoring the importance of maintaining a low diagnostic threshold and initiating empiric antimicrobial therapy promptly . This narrative review

synthesizes current evidence regarding the epidemiology, pathophysiology, clinical presentation, diagnostic approach, treatment modalities, and preventive strategies for PID, with emphasis on contemporary guidelines and emerging considerations in clinical practice.

Epidemiology and Risk Factors

PID predominantly affects sexually active females between 15 and 25 years of age, coinciding with peak years of sexual activity and cervical ectopy, which facilitates pathogen ascension . While precise global incidence figures remain challenging to ascertain due to underdiagnosis of subclinical cases, historical data from the United States documented over 750,000 annual cases, with hospitalization rates reflecting the severe end of the disease spectrum . Recent epidemiological trends indicate a gradual decline in reported cases, likely driven by enhanced screening programs and public health initiatives targeting sexually transmitted infections (STIs) .

Table 1. Major Risk Factors for Pelvic Inflammatory Disease

Risk Factor	Clinical Relevance
Age <25 years	Increased cervical ectopy and behavioral risk patterns
Multiple sexual partners	Elevated exposure to STI pathogens
Inconsistent barrier contraception	Reduced protection against cervical infection
Prior PID or STI history	Documented increased susceptibility to recurrence
Bacterial vaginosis	Disruption of vaginal flora facilitating ascension
Intrauterine device (IUD) insertion	Transient risk elevation within first 3 weeks post-insertion
Vaginal douching	Alters vaginal microbiome and may mask symptoms

Notably, the presence of an intrauterine device does not confer long-term increased risk beyond the initial three-week post-insertion window, a distinction critical for contraceptive counseling . Genetic polymorphisms affecting toll-like receptor pathways have also emerged as potential susceptibility determinants, particularly regarding progression from *C. trachomatis* cervicitis to overt upper tract disease .

Etiology and Pathophysiology

Microbiological Etiology

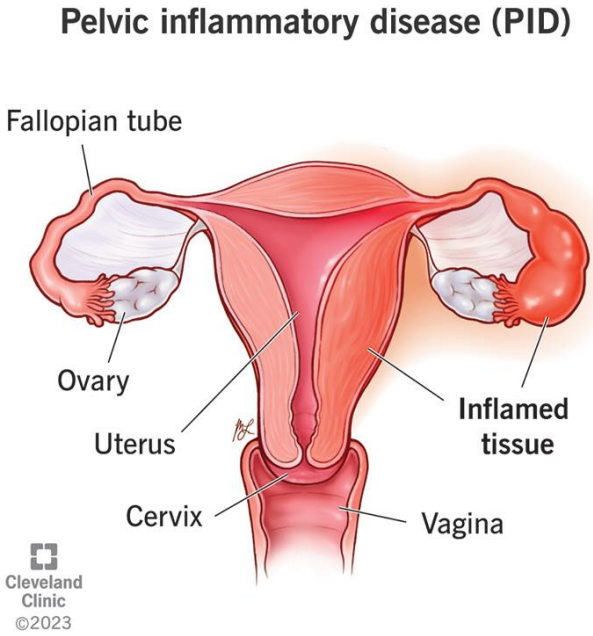
The majority of PID cases are polymicrobial in nature. *N. gonorrhoeae* and *C. trachomatis* represent the most frequently identified pathogens, though *Mycoplasma genitalium* has gained increasing recognition as a significant causative agent, particularly in treatment-resistant cases . Additional contributors include organisms associated with bacterial vaginosis (*Prevotella* species, *Mobiluncus*), *Trichomonas vaginalis*, and less commonly, enteric and respiratory pathogens that may ascend following gynecological procedures or parturition .

Mechanism of Ascending Infection

The pathogenesis of PID typically unfolds in two stages. Initially, colonization or infection of the vagina or cervix disrupts the normal mucosal barrier. Subsequently, microorganisms ascend through the endocervical canal into the normally sterile upper genital structures . Several mechanisms facilitate this ascension:

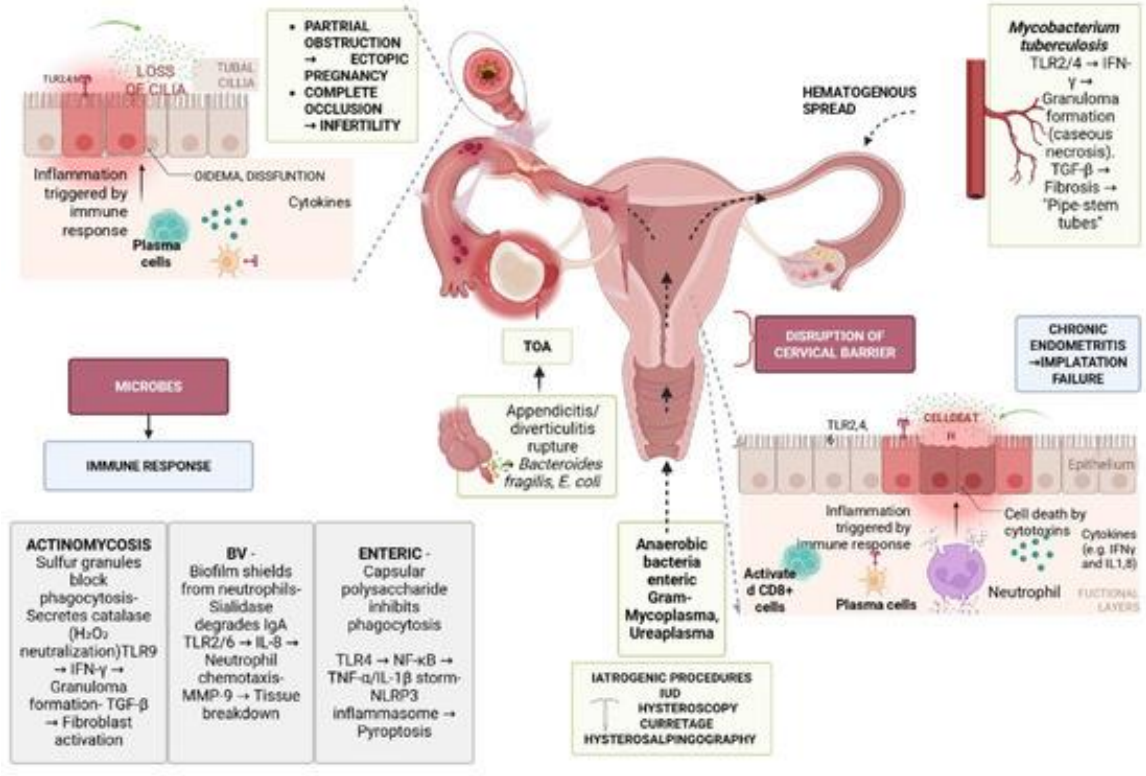
- **Cervical barrier compromise:** Cervical mucus normally provides a functional defense against upward microbial migration; however, inflammation, hormonal fluctuations during ovulation, and menstrual retrograde flow diminish this protection .
- **Mechanical facilitation:** Uterine contractions associated with orgasm, along with sperm-mediated carriage of bacteria, may propel organisms into the fallopian tubes .
- **Iatrogenic introduction:** Instrumentation of the uterus, including IUD insertion, dilation and curettage, or hysteroscopy, can transiently breach cervical defenses .

Once established in the upper tract, infection triggers an intense inflammatory cascade. Complement-mediated responses damage tubal mucosa, with inflammation frequently becoming transmural. The resulting scarring, adhesions, and ciliary dysfunction constitute the pathophysiological basis for long-term sequelae .



PID Anatomy and Inflamed Tissue

Figure 1. Anatomical illustration demonstrating PID with inflamed fallopian tubes and adjacent tissues. *(Image courtesy: Cleveland Clinic)*



PID Pathophysiology Diagram

Figure 2. Comprehensive pathophysiology diagram illustrating microbial ascension, immune response, and complications including tubo-ovarian abscess (TOA) and chronic sequelae. *(Image source: MDPI Microorganisms)*

Clinical Manifestations

The clinical presentation of PID exists along a continuum from entirely asymptomatic infection to severe, systemic illness requiring hospitalization. This diagnostic challenge is compounded by the fact that even minimally symptomatic PID can produce irreparable tubal damage .

Common Presenting Features

Patients typically report lower abdominal or pelvic pain, which is frequently bilateral and may range from dull aching to sharp, severe discomfort . Associated symptoms include:

- Abnormal vaginal discharge (mucopurulent or purulent)
- Intermenstrual or postcoital bleeding
- Deep dyspareunia
- Dysuria and urinary frequency
- Fever, malaise, and anorexia in more severe presentations

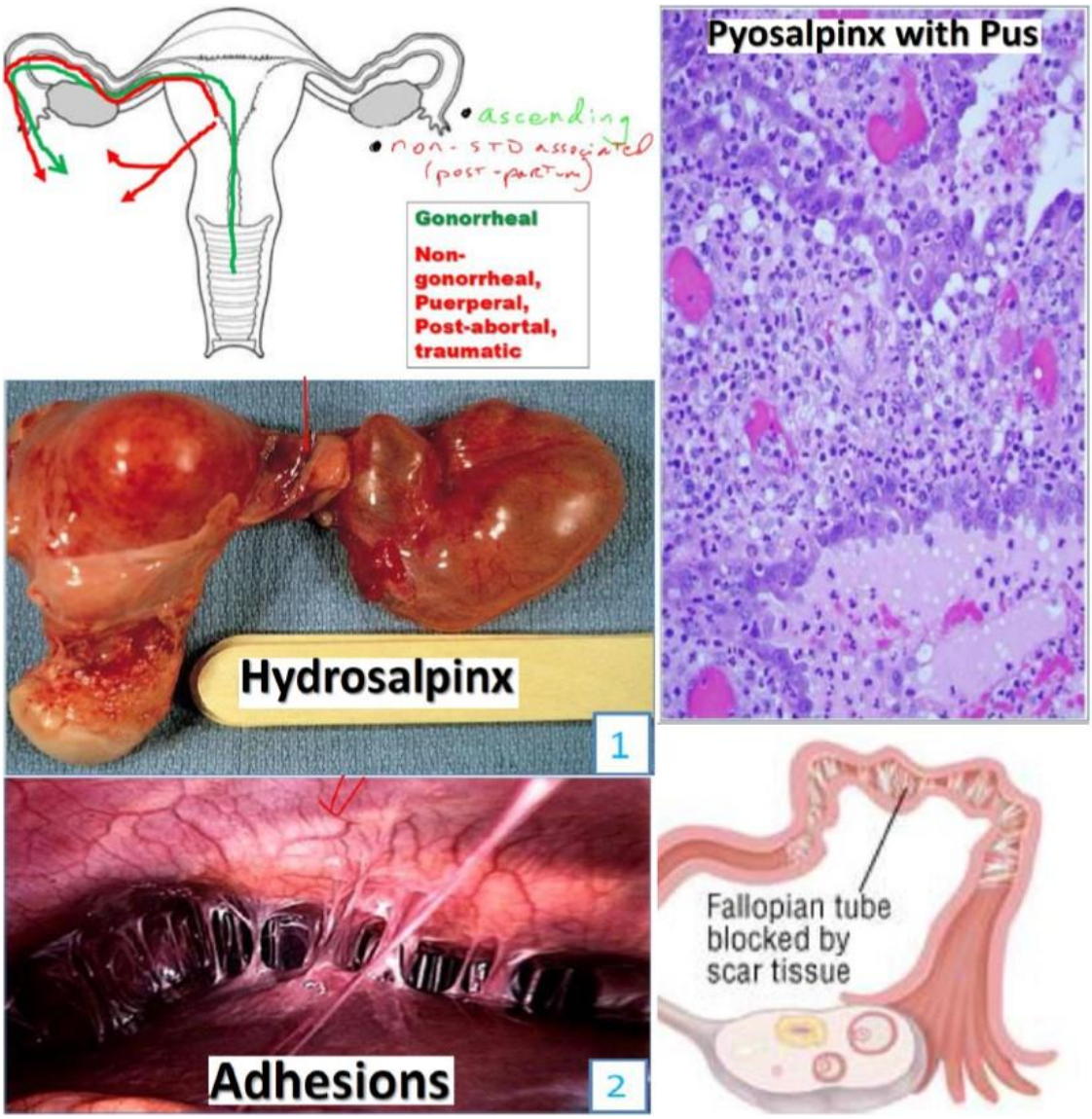
Physical Examination Findings

Bimanual pelvic examination constitutes the cornerstone of clinical assessment. The minimum diagnostic criteria established by the Centers for Disease Control and Prevention (CDC) require the presence of one or more of the following: cervical motion tenderness, uterine tenderness, or adnexal tenderness . Additional supportive findings include purulent endocervical discharge, cervical friability, and fever exceeding 38°C. Palpation of an adnexal mass raises concern for tubo-ovarian abscess formation .

Atypical and Extra-Pelvic Presentations

Approximately 1–30% of women with PID develop perihepatitis, or Fitz-Hugh-Curtis syndrome, characterized by inflammation of the liver capsule and adjacent peritoneal surfaces . These patients present with pleuritic right upper quadrant pain that may

radiate to the right shoulder, often without hepatic parenchymal involvement or transaminase elevation. Laparoscopic evaluation classically reveals "violin-string" adhesions between the liver capsule and anterior abdominal wall .



PID Complications including Hydrosalpinx and Adhesions

Figure 3. Composite illustration depicting ascending infection pathways, hydrosalpinx, tubal occlusion by scar tissue, and adhesive disease. *(Image source: Memorang)*

Diagnosis

Clinical Diagnostic Approach

No single laboratory test, imaging modality, or physical finding definitively confirms PID in all cases. Consequently, diagnosis relies upon clinical judgment with integration of historical, examination, and laboratory findings . The CDC advocates for a low threshold for empiric treatment, particularly among sexually active young women and those at elevated STI risk who present with pelvic or lower abdominal pain when no alternative diagnosis is apparent .

Table 2. Diagnostic Criteria for Pelvic Inflammatory Disease

Category	Criteria
Minimum Criteria (Empiric treatment warranted)	Cervical motion tenderness, OR uterine tenderness, OR adnexal tenderness
Additional Supporting Criteria	Oral temperature >38.3°C; abnormal cervical/vaginal mucopurulent discharge; presence of white blood cells on saline microscopy; elevated C-reactive protein or erythrocyte sedimentation rate; laboratory documentation of cervical *N. gonorrhoeae* or *C. trachomatis* infection
Definitive Criteria	Endometrial biopsy with histopathologic endometritis; transvaginal ultrasound/MRI showing thickened fluid-filled tubes, free pelvic fluid, or tubo-ovarian complex; laparoscopic findings consistent with PID

Laboratory and Microbiological Evaluation

Recommended investigations include nucleic acid amplification testing (NAAT) for *C. trachomatis* and *N. gonorrhoeae*, with consideration for *M. genitalium* NAAT where available . Saline wet mount microscopy demonstrating abundant polymorphonuclear leukocytes supports the diagnosis, though their absence carries a high negative predictive value (approximately 95%) for PID . Complete blood count and C-reactive protein provide adjunctive inflammatory markers, particularly useful for monitoring treatment response . All patients should undergo pregnancy testing to exclude ectopic pregnancy, and screening for HIV and syphilis is recommended given shared risk factors .

Imaging and Surgical Diagnosis

Transvaginal ultrasonography assists in identifying tubo-ovarian abscesses, hydrosalpinx, or free pelvic fluid. Computed tomography may be employed when extra-pelvic extension or alternative intra-abdominal pathology is suspected. Laparoscopy remains the gold standard for definitive diagnosis, permitting direct visualization of tubal inflammation, purulent exudate, and adhesions; however, its invasive nature limits routine use .

Table 3. Differential Diagnosis of Acute Pelvic Pain in Reproductive-Age Women

Condition	Distinguishing Features
Ectopic pregnancy	Positive β-hCG, unilateral pain, adnexal mass, hemodynamic instability
Acute appendicitis	Right lower quadrant pain migrating from periumbilical region, anorexia, localized rebound tenderness
Ruptured ovarian cyst	Sudden unilateral pain, often mid-cycle; ultrasound demonstrating free fluid and cystic changes
Ovarian torsion	Acute severe unilateral pain with nausea/vomiting; Doppler showing absent blood flow
Endometriosis	Cyclical pain, dyspareunia, dysmenorrhea; often chronic rather than acute presentation
Urinary tract infection	Dysuria, frequency, suprapubic pain; pyuria without pelvic tenderness

Treatment

Principles of Antimicrobial Therapy

Treatment must provide broad-spectrum coverage against the polymicrobial array of PID pathogens, including *N. gonorrhoeae*, *C. trachomatis*, anaerobes, and gram-negative bacilli . The CDC recommends prompt initiation of empiric therapy to minimize tubal damage, even before laboratory confirmation is available .

Table 4. CDC-Recommended Antimicrobial Regimens for PID

Setting	Regimen	Duration
Outpatient (Oral/IM)	Ceftriaxone 500 mg IM once (1 g if ≥150 kg) PLUS Doxycycline 100 mg PO BID PLUS Metronidazole 500 mg PO BID	14 days
Alternative Outpatient	Cefoxitin 2 g IM once with Probenecid 1 g PO concurrently PLUS Doxycycline 100 mg PO BID PLUS Metronidazole 500 mg PO BID	14 days
Inpatient (Parenteral)	Ceftriaxone 1 g IV q24h PLUS Doxycycline 100 mg PO/IV q12h PLUS Metronidazole 500 mg PO/IV q12h	14 days total; transition to oral after 24–48 h clinical improvement
Alternative Inpatient	Cefotetan 2 g IV q12h PLUS Doxycycline 100 mg PO/IV q12h	14 days
Alternative Inpatient	Ampicillin-sulbactam 3 g IV q6h PLUS Doxycycline 100 mg PO/IV q12h	14 days
Alternative Inpatient	Clindamycin 900 mg IV q8h PLUS Gentamicin loading dose (2 mg/kg) then maintenance (1.5 mg/kg q8h)	14 days

Doxycycline should be administered orally when feasible due to the pain associated with intravenous infusion. Oral metronidazole achieves bioavailability comparable to intravenous formulation and may be substituted in patients without severe illness or tubo-ovarian abscess .

Indications for Hospitalization

Hospitalization and parenteral therapy are warranted when any of the following conditions are present: surgical emergencies cannot be excluded; tubo-ovarian abscess is identified; the patient is pregnant; severe illness with high fever, nausea, or vomiting precludes oral intake; or the patient fails to respond to outpatient oral therapy . Adolescents may be managed according to the same criteria as adult women, as no evidence supports superior outcomes with routine hospitalization for younger patients

Follow-Up and Response Assessment

Patients should demonstrate substantial clinical improvement—defervescence and reduction in abdominal tenderness—within 72 hours of therapy initiation . Failure to improve necessitates reassessment, consideration of alternative diagnoses, and possible hospitalization for patients managed as outpatients . Test-of-cure is indicated for gonococcal and *M. genitalium* infections per pathogen-specific guidelines, and repeat testing for chlamydia and gonorrhea at three months screens for reinfection .

Complications and Long-Term Sequelae

Acute Complications

Tubo-ovarian abscess (TOA) develops in 15–35% of hospitalized PID patients, representing an inflammatory mass involving the fallopian tube, ovary, or both, filled with purulent material . Clinical manifestations include persistent fever, severe pelvic pain, and palpable adnexal mass. While many TOAs respond to antimicrobial therapy alone, large abscesses (>8 cm), suspected rupture, or failure to improve medically necessitate surgical intervention—either percutaneous drainage or exploratory laparotomy . Rupture constitutes a surgical emergency with risk of diffuse peritonitis and septic shock.

Fitz-Hugh-Curtis syndrome, as previously discussed, involves perihepatic inflammation and adhesions. Though generally managed with antimicrobial therapy alone, symptomatic adhesive disease may rarely require laparoscopic lysis .

Chronic Sequelae

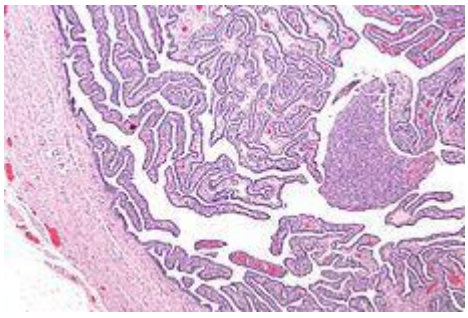
The long-term reproductive consequences of PID constitute its most devastating impact. A landmark study estimated that among women aged 20–24 with PID, 18%

developed chronic pelvic pain, 8.5% experienced ectopic pregnancy, and 16.8% faced infertility .

Infertility results from tubal occlusion, loss of ciliated epithelium, and peritubal adhesions that impede ovum transport and sperm migration. Women with prior PID carry up to a five-fold increased risk of infertility, with risk magnified by *C. trachomatis* infection, delayed treatment, recurrent episodes, and severe disease . Notably, asymptomatic or subclinical infections produce equivalent tubal damage, emphasizing the insidious nature of this complication .

Ectopic pregnancy arises when tubal damage prevents normal embryo transit to the uterine cavity. Historical data indicate ectopic pregnancy rates of approximately 9% following PID compared to 1.4% in women without such history . Given the life-threatening potential of tubal rupture and hemorrhage, early ultrasound evaluation is essential in any pregnant patient with a history of PID.

Chronic pelvic pain affects approximately one-third of women with prior PID, attributed to inflammatory adhesions, hydrosalpinx, and cyclic menstrual changes affecting scarred tissues . Recurrent PID represents the strongest predictor of chronic pain development.



Histopathology of Salpingitis

Figure 4. Histopathological section demonstrating acute salpingitis with inflammatory infiltrate and mucosal architectural distortion.

Prevention and Public Health Considerations

Prevention of PID fundamentally requires reduction of STI acquisition and prompt treatment of lower genital tract infections before ascension occurs. Evidence-based strategies include:

- **Behavioral interventions:** Consistent and correct condom use, reduction in number of sexual partners, and avoidance of douching .

- **Screening programs:** Annual chlamydia and gonorrhea screening for sexually active women under age 25, and for older women with risk factors, facilitates detection and treatment of asymptomatic cervical infections .
- **Partner notification and treatment:** All recent sexual partners (within 60 days) of PID patients require evaluation and empiric treatment for chlamydia and gonorrhea, regardless of the index patient's identified pathogens . Partner treatment with doxycycline 100 mg twice daily for seven days substantially reduces recurrence risk .
- **Post-procedure prophylaxis:** Screening for STIs before IUD insertion and consideration of prophylactic antibiotics in high-risk populations may mitigate procedure-associated PID risk .

Patient education regarding the implications for future reproductive health, delivered alongside clear written materials, supports adherence to treatment and follow-up protocols .

Conclusion

Pelvic inflammatory disease remains a critical condition in reproductive medicine, demanding high clinical suspicion, prompt empiric therapy, and comprehensive follow-up to mitigate its substantial short- and long-term morbidity. The polymicrobial nature of PID—encompassing *N. gonorrhoeae*, *C. trachomatis*, *M. genitalium*, and anaerobic organisms—necessitates broad-spectrum antimicrobial regimens that address the full spectrum of likely pathogens. Because no single diagnostic test definitively confirms PID, clinicians must integrate clinical criteria with laboratory and imaging findings while maintaining a low threshold for treatment.

The sequelae of untreated or inadequately treated PID, including tubo-ovarian abscess, chronic pelvic pain, infertility, and ectopic pregnancy, underscore the condition's public health significance. Future advances in rapid point-of-care diagnostics, improved *M. genitalium* detection, and enhanced partner notification systems promise to further reduce PID incidence and its devastating complications. Ultimately, a combination of primary prevention through STI control, early clinical intervention, and patient education represents the most effective strategy for preserving reproductive health in at-risk populations.

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