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Pelvic inflammatory disease in adolescents

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Pelvic inflammatory disease (PID) is an infection of the upper female genital tract. It encompasses an array of inflammatory disorders, including endometritis, parametritis, salpingitis, oophoritis, peritonitis, perihepatitis, and tubo-ovarian abscess (TOA). Perihepatitis (Fitz-Hugh-Curtis syndrome) and TOA are acute complications, whereas chronic complications of PID include infertility, ectopic pregnancy, and chronic pain.

Epidemiology of pelvic inflammatory disease

Centers for Disease Control and Prevention (CDC) surveillance data of PID have limitations. Age-specific data for PID are not available and cases are based on individual clinicians' reports [1] . The reporting method typically overestimates symptomatic cases and underestimates asymptomatic cases. There are an estimated 1 million women in the United States annually with PID; approximately 20% of these cases occur among adolescent girls. The number of reported hospitalizations for PID in the United States has declined dramatically from approximately 460,000 in 1980 to 170,000 in 2001 in girls and women 15 to 44 years of age. Since 1995, however, this number has remained relatively constant. Additionally, since 1980, the number of initial office visits for PID by girls and women 15 to 44 years of age has declined from approximately 425,000 to 250,000 and remained relatively constant since 1998 [1] . The changing trends may be related in part to the national increase in STD screening and treatment practices.

Behavioral risk factors

Exposure to sexually transmitted infection (STI) by *Chlamydia trachomatis* and *Neisseria gonorrhoea* is a major risk factor for the acquisition of PID. Thus, behaviors such as early sexual debut, multiple sexual partners, unprotected sexual intercourse, and drug use

affect the sexually transmitted disease (STD) and PID rates [2] . Vaginal douching, a common behavior, particularly among African-American women, may facilitate the ascension of organisms to the upper genital tract. Studies have noted that vaginal douching within 6 days of being diagnosed with PID is associated with endometritis and upper genital tract infections [3] [4] [5] .

Biologic risk factors

Menstruation and the menstrual cycle

Studies indicate that adult women are more susceptible to STIs during menses. Sexual intercourse during menses is associated with an increased risk for PID because of the loss of the mucus plug, retrograde menstrual flow, myometrial contractility, and the presence of menstrual blood, which acts as a good culture media [5] [6] . Women are also more susceptible to STIs during the follicular or estrogenic phase of the menstrual cycle than during the luteal or progestational phase of the cycle [7] .

Cervical mucus immunity

The concentration of genital tract immunoglobulins IgA, IgG, and cytokines varies during the menstrual cycle, declining during the follicular phase, reaching a low during ovulation, and increasing during the luteal phase. Preliminary data in adolescents indicate a steeper decline in IgG concentrations during the follicular phase compared with adults, potentially making adolescents more vulnerable to acquisition of STIs than adults [8] .

Cervical ectopy

The presence of cervical ectopy (common during adolescence) is associated with increased rates of *C trachomatis* and *N gonorrhoeae* infection, most likely caused by exposure of the more vulnerable columnar cells of the endocervical canal to incoming organisms [9] .

Contraception

The role of oral contraceptive agents in the pathogenesis of PID is less clear. Oral contraceptives may promote the acquisition of STIs by contributing to the persistence of cervical ectopy, but they also protect the cervix from STIs by thickening cervical mucus and reducing menstrual flow [10] [11] . Earlier studies on the role of the intrauterine device (IUD) indicate an increased risk for PID among IUD users. This increased risk, however, seems to be related to a person's background STI risk and not the IUD [12] [13] .

Systems-related risk factors

Adolescents often do not have access to convenient, affordable, confidential, and “adolescent-friendly” health care services, which results in delays in diagnosis and treatment of PID [14] [15] . Delayed care for abdominal pain is a major factor for

development of severe PID and its complications [14] [15] . Compared with adults, adolescents with PID are significantly more likely to seek health care later in the course of their illness (7.8 versus 5.6 days) [15] . This is particularly troublesome because women who delay seeking care for PID-related abdominal pain of more than 3 days are three times more likely to experience infertility and ectopic pregnancy than women who seek care within 3 days [14] .

Etiology of pelvic inflammatory disease

PID is a mixed polymicrobial infection in the upper genital tract. Because studies on the microbiology of PID were conducted primarily in adult women in the late 1970s and 1980s, information for this section is drawn from the adult literature. Commonly recovered organisms from the endometrium, fallopian tubes, and peritoneum by culture include facultative aerobic and anaerobic organisms, *C trachomatis*, and *N gonorrhoeae* [2] [16] . In addition to *C trachomatis* cultures, IgM and IgG serologic titers for chlamydia have been used to assess the link between *C trachomatis* and PID [17] .

Mycoplasma genitalium and *Ureaplasma urealyticum* also have been isolated from the cervix or endometrium in association with PID. Their role as causative agents in PID is less well understood, however [2] [16] [18] . Bacterial vaginosis (BV) also is commonly found in conjunction with PID. Although anaerobic organisms are present in both BV and PID, the role of BV in the pathogenesis of PID needs to be defined better [2] [19] [20] .

Pathogenesis of pelvic inflammatory disease

PID begins as an infection of the cervix with ascension of microorganisms to the upper genital tract. It is theorized that offending organisms gain access to the endometrium by attaching to the columnar epithelium of cervical ectropion, to spermatozoa, or during menses. Viruses such as HIV or herpes simplex also can facilitate this process by disrupting immunologic barriers to infection. The inflammatory response to offending organisms disrupts the normal host defense mechanisms by altering the vaginal pH, normal vaginal flora, and the cervical mucous barrier [2] . Adolescent girls have been diagnosed with PID up to the first trimester of pregnancy, suggesting that the risk of ascending infection decreases after the first trimester of pregnancy [21] .

Approximately 10% to 17% of females with endocervical *N gonorrhoeae* infection and 10% to 30% of females with endocervical *C trachomatis* infection develop PID [22] . The role of these organisms in causing *subclinical* or *asymptomatic* PID (as defined by histological endometritis) bears consideration, however; endocervical *C trachomatis* and *N gonorrhoea*e have been associated in 27% and 26%, respectively, of subclinical PID cases [23] . Sweet and colleagues [24] found *N gonorrhoeae* was the organism recovered most frequently within the initial 24 hours of symptoms. Beyond 48 hours, the most frequent isolates were anaerobes, especially anaerobic cocci. A temporal relationship also seems to exist between the onset of menses and the causative agents of PID. When the onset of PID symptoms is within 7 days of menses, *C trachomatis* and *N gonorrhoeae* are often the etiologic agents. When the onset of symptoms is more than 14 days after the onset of

menses, aerobic or anaerobic organisms are involved [7] .

Clinical characteristics of pelvic inflammatory disease

The clinical presentation of PID varies from asymptomatic to severe disease. The literature supports cases of *silent PID*, which involves infertile women with evidence of tubal scarring but no history of PID [25] . Midline or bilateral lower abdominal pain is the most common presenting symptom. Associated symptoms include yellow or malodorous vaginal discharge, dyspareunia, irregular vaginal bleeding, dysuria, nausea, vomiting, and fever. The severity of PID depends on the duration of symptoms and the etiologic agent; PID associated with *C trachomatis* tends to be less symptomatic than PID associated with *N gonorrhoeae* [26] . According to Bukusi and colleagues [27] , HIV does not seem to affect the clinical severity of PID among adult women, but TOAs, although rare, may occur more often as a complication among HIV-positive women.

Physical examination findings of pelvic inflammatory disease

The findings upon physical examination include moderate-to-severe lower abdominal tenderness with or without rebound tenderness. The pelvic examination findings vary, and may include vaginal discharge, mucopurulent cervicitis (mucopurulent cervical discharge and a friable cervix), cervical motion tenderness, uterine fundal tenderness, unilateral or bilateral adnexal tenderness, and a palpable adnexal mass.

Diagnosis of pelvic inflammatory disease

A diagnosis of PID generally is based on clinical findings. Laparoscopy is the most specific method for diagnosis of acute PID, but this procedure is difficult to justify in all suspected cases of PID and frequently is not available. At present, laparoscopy is used to obtain a more accurate diagnosis in atypical cases. Obtaining an endometrial biopsy to diagnose endometritis may be useful, but this procedure is not standard practice in adolescents.

The CDC has recommended a minimal set of clinical criteria and supportive criteria to diagnose PID, which are listed in Box 1

Box 1. Criteria for diagnosis of pelvic inflammatory disease

Minimal criteria

- Uterine/adnexal tenderness
- Cervical motion tenderness

Additional criteria used to support the diagnosis

- Oral temperature greater than 101°F

- Abnormal cervical discharge
- Presence of white blood cells on wet mount
- Elevated erythrocyte sedimentation rate or C-reactive protein
- Laboratory documentation of *N gonorrhoeae* or *C trachomatis*

[28] . The minimal clinical criteria have been recommended to maximize sensitivity, reduce the chance of a missed or delayed diagnosis, and thus promote early treatment of PID [28] . Previous criteria included three mandatory components: abdominal pain, adnexal tenderness, and cervical motion tenderness. When comparing the reliability of clinical criteria to diagnose PID using endometrial sampling as the “reference test,” however, the sensitivity of these three criteria was 83%. In contrast, the sensitivity of one criterion, adnexal tenderness, was 95% [28] [29] . Because of this, the new guidelines require that only one of the criteria be met. The presence of additional clinical and laboratory criteria, such as a positive test result for *C trachomatis* or *N gonorrhoeae,* further support a diagnosis of PID (see Box 1). In the absence of a positive endocervical test, IgM and IgG titers for chlamydia may be considered. A fourfold rise in IgM and IgG serologic titers of *C trachomatis* titers has been demonstrated in clinical PID, but these tests are not done routinely, in part because their use would likely result in delay of diagnosis [17] .

Although elevation in erythrocyte sedimentation rate can occur in up to 75% and an elevated white blood cell count in up to 60% of patients with acute salpingitis, in many cases one or both of these laboratory tests are normal. Thus, abnormalities in laboratory tests are not necessary to make this diagnosis. Health care providers should suspect PID when an adolescent has uterine/adnexal tenderness or cervical motion tenderness and provide empiric PID treatment [28] . Box 2

Box 2. Differential diagnosis of pelvic inflammatory disease

Gastrointestinal

Appendicitis

Cholecystitis

Constipation

Gastroenteritis

Hernia

Inflammatory bowel disease

Renal
Cystitis
Pyelonephritis
Nephrolithiasis
Urethritis
Gynecologic
Corpus luteum cyst
Dysmenorrhea
Ectopic pregnancy
Endometriosis
Ovarian cyst
Ovarian torsion
Ovarian tumor

lists other gastroenterologic, renal, and gynecologic diagnoses that should be considered in an adolescent with lower abdominal pain.

Role of diagnostic-imaging studies in pelvic inflammatory disease and its complications

Imaging studies have little to no role in diagnosing endometritis or salpingitis because fluid in the cul-de-sac is not helpful in differentiating the presence or absence of PID [30] . Given the clinical limitations of diagnosing a TOA, however, a pelvic ultrasound may be necessary when this is being considered [31] . Transvaginal ultrasound is preferable to a pelvic ultrasound per abdomen because it is more sensitive in detecting TOAs [32] . The advantages of the transvaginal ultrasound include increased sensitivity, specificity, availability, and ease of performance, and reduced cost. In adolescents, however, conducting a transvaginal ultrasound needs to be approached with great sensitivity and appropriate educational counseling.

Treatment

The treatment goals for PID are to eliminate the infection and prevent long-term sequelae. Health care providers can accomplish these goals by maintaining a low threshold for the diagnosis of PID and beginning treatment as soon as possible [28] . Recent management trends for PID have been in the outpatient setting. The literature suggests no difference in reproductive outcomes between inpatient and outpatient treatment of mild to moderate PID in young adult women [33] . According to the CDC and recent studies, HIV-positive women with mild to moderate PID may be treated successfully in the outpatient setting as well [27] [28] . The CDC recommends that the decision to hospitalize for PID treatment should be individualized and at the discretion of the clinician. The CDC suggests hospitalization for the following concerns: surgical emergencies, pregnancy, failure of outpatient treatment, noncompliance, and severe illness including those with nausea, vomiting, or high fever, as well as the presence of a TOA.

Many clinicians also use the adolescent age group as an indication for hospitalization based on the possibility of poor adherence to medication regimens and follow-up visits, problems observed among some adolescents. Whether outpatient or inpatient management is selected, antibiotic coverage should include treatment for the broad spectrum of bacteria found in PID. There is little difference in the efficacy of the recommended treatment options, but cost, convenience of administration, and safety should be assessed.

Outpatient management

Clinicians are encouraged to follow the current CDC treatment guidelines for PID. Two oral treatment options are recommended:

1. Ofloxacin, 400 mg, orally twice daily for 14 days or levofloxacin, 500 mg, orally once daily for 14 days with or without metronidazole, 500 mg, orally twice a day for 14 days [28] and
2. Ceftriaxone, 250 mg, intramuscular injection in a single dose or cefoxitin, 2 g, intramuscular injection in a single dose or other third-generation cephalosporin plus doxycycline, 100 mg, orally twice a day for 14 days with or without metronidazole.

Metronidazole should be added when anaerobic coverage is a concern. Patients who fail to respond to outpatient therapy within 72 hours should be given parenteral therapy.

Inpatient treatment

The CDC recommends two parenteral regimens:

1. Cefotetan, 2 g, intravenously, every 12 hours or cefoxitin, 2 g, intravenously, every 6 hours, plus doxycycline, 100 mg, intravenously, every 12 hours [28] and
2. Clindamycin, 900 mg, intravenously, every 8 hours plus gentamicin, 2 mg/kg/day, intravenously, every 8 hours.

Despite concerns that the latter option may not provide adequate coverage for *C trachomatis*, patients treated with this option experience comparable cure rates [28] . Generally, parenteral therapy is discontinued 24 hours after clinical improvement. Oral therapy is continued for 14 days. Intravenous doxycycline can cause phlebitis and should be given orally whenever possible. The bioavailability of oral doxycycline is similar to intravenous doxycycline. All patients should receive a follow-up examination in 72 hours to ensure clinical improvement. Male sex partners of patients with PID should be examined and empirically treated if they had sexual contact with the patient during the preceding 60 days [28] .

Complications of pelvic inflammatory disease

Perihepatitis or Fitz-Hugh-Curtis syndrome can occur in 15% to 20% of cases of PID [22] and TOA is a reported complication in up to one third of the patients hospitalized for PID [34] . Symptomatic and asymptomatic PID causes injury and fibrosis to the internal epithelium of the fallopian tubes, resulting in loss of ciliary action and, in many cases, occlusion of the fallopian tubes [2] [19] . Approximately one third of infertility cases among women have been attributed to acute salpingitis [14] [35] . In general, increased severity and greater number of episodes are related independently and positively to the risk for infertility. After one episode of PID, the infertility risk ranges from 8% to 12%, after two episodes the risk ranges from 20% to 25%, and after three episodes it ranges from 40% to 50% [36] [37] [38] . In addition, women with a history of PID have a threefold to 10 fold higher risk of having an ectopic pregnancy [23] [39] . The data are limited on the prevalence of chronic pain from PID.

Perihepatitis (Fitz-Hugh-Curtis syndrome)

The etiologic agents involved in perihepatitis, *N gonorrhoeae* and *C trachomatis*, have been found in 10% and 50% of cases of perihepatitis, respectively [23] [40] . Perihepatitis is thought to result from the direct spread of these organisms from the fallopian tubes along the paracolic gutters or sulci into the peritoneal cavity. From there, the organisms reach the subphrenic space and hepatic surface. Spread also is possible from the reproductive tract by way of retroperitoneal lymphatics. If the condition is untreated, “violin-string” adhesions form between the liver capsule and the subphrenic surface of the diaphragm [41] . The classic presentation of perihepatitis is severe right–upper quadrant abdominal pain, which exacerbates on inspiration and often radiates to the shoulder top; severe right–upper abdominal tenderness is present upon palpation. Additional laboratory findings may include an elevated sedimentation rate and liver enzymes; the latter tests are typically normal with *C trachomatis* perihepatitis and abnormal with *N gonorrhoeae* perihepatitis. Thus the diagnosis of perihepatitis is based on a high index of suspicion when a sexually active patient presents with right–upper quadrant abdominal pain. This condition frequently is confused with other conditions because it mimics cholelithiasis, hepatitis, right–lower lobe pneumonia, pleuritis, subphrenic abscess, perforated peptic ulcer, and pancreatitis. The treatment approach for perihepatitis is the same as PID.

Tubo-ovarian abscess

A TOA develops as a result of untreated organisms causing damage and blockage to the fallopian tubes with accumulation of bacteria, leukocytes, and fluid within a closed space. The bacteria and inflammation within the fallopian tubes extend through the fimbriated end of the fallopian tube to the adjacent ovary, forming a TOA. Perfusion of the inner wall of the abscess is compromised, creating an anaerobic environment in which anaerobes can flourish. Metastatic abscesses can occur by extension of infection to the ovaries, bowel, and omentum with adherence of tubal fimbriae to these organs [34] . Cultures obtained directly from a TOA generally reveal anaerobes and aerobes, including *Escherichia coli*, *Bacteroides fragilis*, and *peptostreptococcus* bacteria [34] . A *tubo-ovarian complex* is an edematous, adherent, infected structure in PID. In contrast to a TOA, a tubo-ovarian complex is a thick-walled inflammatory mass with vague margins without a dominant cystic component. The complex is perfused and does not have a devitalized abscess wall or pus in the cavity [42] .

Patients with TOAs are often in considerable pain and discomfort. The physical assessment is difficult and often inaccurate because of a patient's discomfort. Most TOAs detected by imaging studies were not detected by physical examination [26] . As mentioned previously, a TOA should be considered in anyone with PID, especially in patients with unilateral abdominal tenderness and adnexal tenderness. It is difficult to clinically diagnose a TOA, however, because the presenting symptoms of PID and TOA overlap [26] .

The transvaginal ultrasound will show a TOA as a thin-walled, septated, well-demarcated cystic mass with air fluid levels. Most important, the ultrasound can help differentiate between a TOA and a tubo-ovarian complex; the latter will appear as a thick-walled mass with vague margins and no cystic component [42] . A pelvic ultrasound also may be used to guide for drainage of an abscess. MRI is another option for diagnosing a TOA, but considering its expense, further studies are needed to clarify its sensitivity in comparison to the transvaginal ultrasound [43] [44] [45] .

Patients with a TOA should be hospitalized for evaluation and management. A conservative approach is chosen first that entails triple, parenteral, antibiotic therapy. The antibiotic choices are the same as the inpatient options for PID, but should include effective anaerobic coverage. Triple therapy with clindamycin, ampicillin, and gentamicin is the treatment of choice for a TOA [46] . The response to antibiotics is followed clinically and by serial pelvic or transvaginal ultrasonograms. A tubo-ovarian complex likely will respond to intravenous antibiotic treatment (in over 95% of cases) [31] .

The duration of intravenous therapy for TOA is not defined clearly. The most conservative approach is to treat for 21 days, either as an inpatient or at home through home nursing care. A more liberal approach is intravenous treatment for 10 days, and, if a clear response has been established clinically and by ultrasound, oral antibiotic therapy for the remaining 10 days [42] . For adolescents, the decision regarding duration of hospitalization should be based on clinical response to intravenous antibiotics, the adolescent's developmental and emotional abilities, and her home support system to ensure excellent outpatient treatment compliance. Surgical intervention is indicated if the

patient fails to respond clinically or if concerns about rupture exist [47] . Surgical interventions include transvaginal, transcutaneous, or transrectal drainage guided by CT or ultrasound [48] [49] . One study found that a TOA greater than 8 cm appears less likely to respond to medical management and may require surgical drainage [47] .

Prevention of pelvic inflammatory disease

The financial, social, and emotional costs of PID and its complications to society are enormous. A recent cost estimate projected that the average per-person lifetime costs for women who developed major complications were $6350 for chronic pelvic pain, $6840 for ectopic pregnancy, and $2150 for infertility. The major costs (79%) were accrued within 5 years of upper genital tract infection [50] . Thus, primary and secondary prevention strategies are of the utmost importance in adolescents to reduce acquisition of PID and to reduce its long-term sequelae. The ideal primary prevention strategy is delayed onset of sexual debut by sexual abstinence. Adolescents who are sexually active should be encouraged to (1) use condoms consistently and correctly, (2) seek care immediately when genital symptoms appear, (3) seek a medical evaluation promptly after having unprotected sex with someone who is suspected of having an STI, and (4) even when a STI is not suspected, seek routine STD checkups at least annually [51] .

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