REVIEW ARTICLE

Current approaches to pelvic inflammatory disease

Abordagem atual da doença inflamatória pélvica

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ABSTRACT

Pelvic inflammatory disease (PID) is an inflammatory process of infectious nature that can affect structures and organs of the upper genital tract. Considering this disease's epidemiological relevance and severe complications, this article provides an update and proposes a systematic approach to PID. The main etiological agents are Neisseria gonorrhoeae, Chlamydia trachomatis and other etiological agents of urethritis, cervicitis, vulvovaginitis and vaginoses. These are generally of polymicrobial origin, which determines the treatment basis for pelvic inflammatory diseases. Women must be checked for PID when experiencing abdominal discomfort, backache, dyspareunia, or presenting with stains during gynecological examination and prior to transcervical procedures. The clinical and laparoscopic classification of PID can be divided into: a) stage I (endometritis/salpingitis without peritonitis), stage II (acute salpingitis with peritonitis), stage III (acute salpingitis with tubal occlusion or tube-ovarian abscess), and stage IV (tube-ovarian abscess rupture). Defining the stage guides procedures and treatment, given that in mild forms (stage I) the treatment and follow-up can be performed in the ambulatory environment while moderate to severe cases require hospitalization so that intravenous treatment and treatment outcome monitoring can be started. Supportive treatment, removal of intrauterine device (IUD), sexual abstinence and rest are also indicated, as well as counseling on the implications of the disease and partner approach.

Key words: Pelvic Inflammatory Disease; Neisseria gonorrhoeae, Chlamydia trachomatis; Mycoplasma; Vulvovaginitis; Vaginosis, Bacterial.

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RESUMO

A doença inflamatória pélvica (DIP) é um processo inflamatório de natureza infecciosa que pode atingir estruturas e órgãos do trato genital superior. Devido à sua importância epidemiológica e de suas graves complicações, este artigo atualiza e propõe uma abordagem sistemática da DIP. Os principais agentes etiológicos são a Neisseria gonorrhoeae, Chlamydia trachomatis e outros agentes etiológicos de uretrites, cervicites, vulvovaginites e vaginoses, em geral, polimicrobiana, o que é a base de sua terapêutica. A mulher deve ser investigada para DIP quando apresenta, especialmente, desconforto abdominal, dor lombar, dispareunia e nódoas ou manchas ao exame ginecológico, previamente a procedimentos transcervicais. A classificação clínico-laparoscópica de DIP pode ser dividida em: a) estágio I (endometrite/salpingite sem peritonite); estágio II (salpingite aguda com peritonite); estágio III (salpingite aguda com oclusão tubária ou abscesso tubo-ovariano); estágio IV (abscesso tubo-ovariano roto). A definição do estágio orienta a conduta e o tratamento, pois em formas leves (estágio I) o tratamento e seguimento podem ser feitos ambulatorialmente, enquanto para os casos moderados ou graves a internação hospitalar está indicada para início do tratamento por via endovenosa e monitorização da resposta ao tratamento. O tratamento suportivo, retirada de dispositivo intrauterino (DIU),

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abstinência sexual e repouso também são indicados, além de orientações sobre as implicações da doença e abordagem do parceiro.

Palavras-chave: *Doença Inflamatória Pélvica; Neisseria gonorrhoeae; Chlamydia trachomatis; Mycoplasma; Vulvovaginite; Vaginose Bacteriana.*

INTRODUCTION

Pelvic inflammatory disease (PID) is an inflammatory process of infectious nature that can affect the structures of the upper genital tract, such as the uterus, fallopian tubes, ovaries and attached structures, causing endometritis, salpingitis, oophoritis, tubo-ovarian abscess, and peritonitis. It is generally determined by the ascension of a vaginal or cervical infectious agent, spontaneously or due to procedures such as insertion of intrauterine device (IUD), endometrial biopsy, and curettage.[1-3]

It affects one million women every year in the United States and is a significant cause of infertility, ectopic pregnancy, chronic pelvic pain, and other conditions of high morbidity and mortality rates among females.[3-6]

Its epidemiological and clinical importance, in addition to its serious complications, means that every woman should be examined for PID in case of suspicion due to medical history or pelvic exam and before any transcervical procedure.[3-6]

Defining the diagnosis in accordance with recommended criteria is important for classifying the disease, which then guides the therapeutic conduct.[1,2,4]

This article aims to review and propose a systematic approach to PID.

METHODS

This is an integrative review carried out in 2011 including publications in the last 10 years with the descriptors "pelvic inflammatory disease" and *Neisseria gonorrheae, Chlamydia trachomatis, Mycoplasma*, vulvovaginitis, and bacterial vaginosis. The scope limits were set as studies with women, including randomized clinical trials, meta-analyzes, guidelines, and reviews, which resulted in 73 articles being identified.

The search used the Medline database via Pubmed (www.pubmed.com), in addition to the webpages of the Center for Disease Control and Prevention (www.cdc.gov) and the Brazilian Ministry of Health (www.aids.gov.br).

The selected references were included on account of their relevance for updates on diagnosis and treatment of PIDs.

ETIOLOGY

The etiological agents involved in PID are the main causes of urethritis, cervicitis, and vulvovaginitis and include *Neisseria gonorrheae* and *Chlamydia trachomatis*, besides *Mycoplasma hominis, Mycoplasma genitalium, Ureaplasma urealyticum, Gardnerella vaginalis, Bacteroides spp*. Other anaerobic bacteria that cause vaginoses can also lead to PID, such as Gram-positive cocci (e.g. *Streptococcus agalactiae, Streptococcus group A, Sthaphylococcus sp.*) and *Enterobacteriaceae* (e.g. *E. coli*). PIDs often have polymicrobial etiology, which is the basis for therapeutic recommendations.[1-3,7] It is suggested that the presence of bacterial vaginosis agents (*Lactobacillus* producers of hydrogen peroxide, *Gardnerella vaginalis, Mycoplasma hominis*, Gram-negative rods and *Ureaplasma urealyticum*) increase the risk of PID (RR = 2.03, 95% confidence interval: 1.16, 3.53).[8,9]

The most common etiologic agent is *Chlamidia trachomatis*, especially in women aged 20 to 24.[10] Approximately 70% of women infected with *Chlamydia* are asymptomatic, 15 to 80% of them evolve to PID, of which 10 to 20% will have tubal infertility.[11-14] The immune response to *Chlamydia* seems to be involved in the pathophysiology of the evolution into PID and can result in infertility because of genetic polymorphism in the production of cytokines and the type of human leukocyte antigen that appears to be associated with that evolution, regardless of antimicrobial therapy.[12,15] This microorganism also produces a toxin responsible for causing lesions and evolution into infertility.[16] It is estimated that the incidence rate of infection by this agent in adolescents is 30%, which justifies preventive educational policies.[17]

Mycloplasma has been identified as an important agent of PID, found in the endometrium and Fallopian tubes of patients with PID and also associated with infertility.[18-20]

Several risk factors are associated with PID, especially those related to sexual behavior (young age, multiple partners, recent new partner, past or current infection by STD agents, no use of barrier protection), and uterine manipulation and instrumentation (interruption of pregnancy, use of UID, hysterosalpingography, in vitro fertilization, or insemination).[3,21]

Due to its clinical importance and serious complications, every woman should be tested for PID in the following situations[4]: a) at any time in which specular or manual examination is performed; b) when reporting vague abdominal discomfort, back pain, spotting, and dyspareunia; c) before transcervical procedures.

CLINICAL PICTURE

Clinical manifestations are of low sensitivity, with a positive predictive value of 65 to 90%. However, they are the basis for diagnosing PID. The main changes observed, with 90% diagnostic probability, include: soreness upon cervical mobility, uterine or adnexal tenderness upon bimanual exam, and evidence of genital tract infection. Vaginal discharge secondary to endometritis, cervicitis, or vaginosis may not be specific, but its absence has a high negative predictive value. Other suggestive clinical changes are: lower abdominal pain (usually bilateral), fever (³ 38°C), unusual bleeding (such as menorrhagia), dysuria, dyspaureunia, onset of pain associated with menstruation, nausea, and vomiting.[1,2,4,9,10]

In a cross-sectional study, Pelpert et al.[22] included 651 patients with PID and observed that the minor clinical criteria recommended by the CDC and adnexal pain showed sensitivities of 83% and 95% (p= 0.001), respectively. They isolated *Chlamydia trachomatis* or *Neisseria gonorrheae* in endometrial samples as criteria associated with endometritis (odds ratio, 4.3; 95% confidence interval, 2.89-6,63).

Wiensefeld et al.[23] have pointed out that patients with acute and subclinical PID showed similar demographic and microbiological characteristics, suggesting comparable pathophysiological mechanisms, regardless of clinical manifestations.

The differential diagnosis includes other abdominal conditions, especially appendicitis. In PID, in addition to cervical pain upon touch and pain upon manipulation of attachments, pain is diffuse and bilateral, with vaginal discharge and bleeding. Patients also tend to look for medical care later. Other differential diagnoses include: ectopic pregnancy (it is always necessary to rule it out if PID is suspected), endometriosis (symptomatology associated with menstrual cycle), ovarian cyst complications (with acute manifestations), and functional pain (with chronic manifestations).[3,24]

Subclinical evolution complicates the diagnosis and leads to long-term sequelae such as ectopic pregnancy, tubal infertility, tubo-ovarian abscess, and chronic pelvic pain. Tubo-ovarian abscess is a severe evolution and requires surgical intervention.[3,25]

Clinical manifestations give no indication for etiologic diagnosis, but Short et al.[20] showed in 722 women with PID that the infection by *Mycoplasma* evolves with less elevation, frequency, incidence and intensity of inflammatory markers, leukocytosis and fever, cervicitis and high vaginal pH and pelvic pain, respectively.

CLASSIFICATION

Based on severity, PID classification informs whether treatment can be done in outpatient clinics or only in the hospital setting. Its clinical definition considers as mild the form that only causes attachments to be thickened and painful to the touch. The moderate form displays involvement of pelvic peritoneum and repercussions due to systemic infections; and the severe form is associated with diffuse peritoneal disease, systemic manifestations, and tubo-ovarian abscesses. Pregnant women should also be followed with more care given the risk of complications.[6,24,26]

The clinical laparoscopic classification of PID can be divided into the following stages:[27] a) stage I: endometritis/salpingitis without peritonitis; (b) stage II: acute salpingitis with peritonitis; c) stage III: acute salpingitis with tubal occlusion or tubo-ovarian abscess; d) stage IV: ruptured tubo-ovarian abscess.

PROPEDEUTICS

All clinical suspicion should be confirmed, whenever possible, by identifying the etiological agent or agents through specific complementary exams (Table 1). Serological tests should also be carried out to assess co-infection by other agents, such as VDRL, hepatitis B and C, and human immunodeficiency virus (category C).[1,2,21,24]

Polymerase chain reaction has been increasingly used for identifying etiological agents, especially for *Chlamydia trachomatis* in cases of STDs and PID.[27] It has greater sensitivity and specificity than other available methods, an important consideration for screening.[13,28]

Table 1 - Complementary tests for specific diagnosis of main PID agents

N. gonorrhoeae	*C. trachomatis*	Vaginose bacteriana	Outros
Gram (Gram negative diplococci) Culture (Meio Thayer Martin)	Elisa (Antibodies) Direct immunofluorescence (Antigens) Polymerase Chain Reaction (PCR) Cell culture	pH>4.5 KOH 10% positive (with odor) Cell biology (clue cells, neutrophils in normal numbers, existence of coccobacilli	Gram staining

Note: bacterioscopic exams and culture with antibiogram can be made with material collected from the external orifice of the uterus, urethra, laparoscopy or posterior cul-de-sac puncture.

Use of PCR can also identify strains of microorganisms such as *Mycoplasma genitalium*, an agent associated with PID and its complications, with reports of therapeutic resistance to cefoxitin and doxycycline.[18,19,29] Ness et al.[30] observed that the existence of antibodies against *Chlamydia trachomatis* elementary bodies (EB) and antibodies against *Chlamydia* heat shock protein (Chsp60) was associated with lower rates of pregnancy and increased recurrence of PID.

The screening for identification of agents associated with bacterial vaginosis also favors a more directed treatment and reduces PID after surgical interruption of pregnancy and is likely to reduce post-hysterectomy complications.[31]

The propedeutics for evaluating the patient's systemic compromise and that of the female genital tract contributes to the differential diagnosis. This assay includes some exams, according to clinical suspicion (category C). While the last three are specific and can lead to a definitive diagnosis, they are not indicated for routine tests:[1-3,21,26] complete blood count, erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP); simple radiography of the abdomen; urinalysis and urine culture; pregnancy test; endometrial biopsy; echography or MRI of the abdomen and pelvis; laparoscopy.

Complete blood count, ESR and CRP, although not specific, can contribute to diagnosis with a follow-up of leukocyte count and evolution of the inflammatory response. Endometrial biopsy can reveal histopathologic evidence of endometritis with high sensitivity and can rule out or confirm a case, especially when there is diagnostic difficulty. It can also help in microbiological identification, but it is a time-consuming procedure with technical difficulties. Ultrasonography is a low-cost examination and it can show thickened tubes, with liquid inside them in the pelvic or tubo-ovarian cavity. For this reason, it is important in identifying tubo-ovarian abscesses with therapeutic implications. Laparoscopic assessment is considered the gold standard. Although not recommended routinely, it can show anatomic abnormalities suggestive of PID (edematous Fallopian tubes often with purulent exudate in the fimbrial ends, as well as the occasional presence of adhesions around the fallopian tubes) and can allow for the differential diagnosis of other diseases, especially in patients who do not respond to initial therapy for PID.[2,3,32]

PID diagnosis can be defined with three major criteria plus a minor one or a criterion, described below (Table 2).[1]

Table 2 - Major criteria, minor criteria, and elaborate criteria for PID

Major Criteria
Lower abdominal pain
Attachments painful to touch
Pain to mobilization of cervix
Minor Criteria
Axillary temperature > 37,5°c
Abnormal vaginal contents or endocervical secretion
Pelvic mass
> 5 white blood cells per immersion area in endocervical secretion
Leukocytosis
C-relative protein or high erythrocyte sedimentation rate
Laboratory confirmation of cervical infection by Gonococco, Chlamydia or Mycoplasma
Elaborate Criteria
Histopathology with evidence of endometritis
Tubo-ovarian abscess or pouch of Douglas abscess detected by pelvic ultrasound
Laparoscopy with evidence of PID

Source: Ministry of Health, Brazil, 2006.[1].

TREATMENT

PID treatment considers the most frequent etiological agents and recommends a combination therapy for *Gonococcus, Chlamydia*, and anaerobic infection[1,2,4].

It should be taken into consideration whether treatment will be pursued in an outpatient clinic or a hospital. Outpatient treatment should be reserved for mild forms. Hospitalization for starting the intra-

venous treatment and monitoring the therapeutic response should always be indicated for moderate to severe manifestations (including patients with signs of peritonism, nausea and vomiting, or high-grade fever); patients with tubo-ovarian abscess and pregnant women, who display a higher incidence of complications. Hospitalization should also be considered in the following situations (category C)[6,21,24]: surgical emergencies not ruled out, absence of clinical response to oral antimicrobial treatment; intolerance to oral treatment; impossibility of either outpatient follow-up or reassessment within 72 hours.

Other criteria have also been considered for hospitalization, such as: patients with immunodeficiencies, adolescents, and women above 35 years. However, severity must always be considered.[6] The possible predictive factors of severity should be investigated before deciding for intravenous therapy. The study by Halperin et al.[33] considers that palpable adnexal mass and erythrocyte sedimentation rate above 50 mm/h are suggestive of tubo-ovarian abscess and are indications for aggressive therapy and prolonged hospitalization.

The therapy recommended by international guidelines considers severity and the outpatient or inpatient approach, and is described in Table 3. The proposed regimens are listed as category A, except inpatient regimens with associated quinolone and metronidazole, considered as category B.[1-4,6,16,21-24]

Endovenous treatment must be maintained for 24 to 48 hours after clinical improvement and supplemented with antimicrobial agent for 14 days. Oral sequential therapy may be used. For outpatients the effectiveness of using single-dose drugs has not been defined, except ceftriaxone (cefoxitin is cited in a single dose of 2 g IM, but should be used in association with 1 g of probenecid).[1,2] Clinical trials have evaluated short-term treatment with azithromycin or monotherapy with ofloxacin, but the findings are still preliminary.[34] Associations of quinolone with azithromycin or metronidazole have been evaluated for outpatient treatment.[7] Judlin and Thiebaugeorges[35] used only levofloxacin and Metronidazole for 40 cases of non-complicated PDI, 10 of which associated with bacterial vaginosis, with effectiveness of clinical and microbial cure in all 35 cases evaluated at the end of follow-up. The use of quinolones should be approached cautiously due to the possibility inducing resistance.[2]

Patients with infection by *Chlamydia trachomatis*, even if asymptomatic, should be treated because of the increased risk of PID, especially when undergoing procedures with uterine instrumentation[11,36].

Treatment with cefoxicitin and doxycycline may not be effective for *Mycoplasma*, which can cause sequelae such as infertility, recurrent PID, and chronic pelvic pain.[29]

Prevalence of anaerobes in cases of PID is very variable. This can be explained by differences in the populations studied, the severity of the disease, and laboratory techniques available. It should be stressed that the eradication of these agents is important for preventing complications such as injury, tubal factor infertility, ectopic pregnancy, and chronic pelvic pain. Treatment of anaerobes should continue to be considered until further research disproves its benefits, especially in cases of tubo-ovarian abscess, vaginosis or HIV co-infection.[5,6] Although able to eradicate *C. trachomatis* and *N. gonorrheae*, quinolone-based regimens, especially with ofloxacin, are less efficient due to the presence of anaerobes, for which reason an association with metronidazole or clindamycin is recommended.[37] The best response found among quinolones was with the use of moxifloxacin.[38,39]

Regimens with azithromycin should not be recommended because the effectiveness of the appropriate treatment could not be determined. In addition, although meropenem has a broad spectrum of action with reports of clinical response, studies with microbiological eradication are scarce. The use of doxycycline associated with metronidazole presents low cure rates due to poor action against *N. gonorrheae*, which makes an association with cephalosporin necessary.[37]

Supportive treatment with analgesics, anti-inflammatories and antipyretics, IUD removal, sexual abstinence, and rest are indicated, as well as counseling for patients and partners about the implications of the disease (category C)[24]. Incidence of PID after IUD insertion is associated with a sixfold increase. However, removing the device after diagnosis does not show an increase in treatment failure rates.[2,40]

Patients should be evaluated 72 hours after the appropriate treatment. If there is no improvement of symptoms, extensive study must be conducted, with intravenous therapy and surgical intervention indicated (category C).[2,4,24]

Surgical treatment should be reserved for special situations, such as[1]: failure of clinical treatment; persistent or growing pelvic mass in spite of clinical treatment; suspicion of rupture of tubo-ovarian abscess; hemoperitoneum; pouch of Douglas abscess.

Table 3 - Recommendations for PID treatment

Treatment	Stage I	Stage II	Stage III	Stage IV
Outpatient	Ceftriaxone** 250mg, IM, single dose **OR** Ofloxacin*** 400mg, OR, 12/12h, 14 days **OR** Levofloxacin*** 500mg, OR, once a day, 14 days **OR** Moxifloxacin*** 400mg, OR, 24/24h, 14 days associated with Doxycycline 100mg, OR, 12/12h, 14 days **OR** Tetracycline 500mg, OR, 6/6h, 14 days Note: these schemes can be associated with Metronidazole 400 to 500mg, OR, 12/12h to the treatment scheme, especially in cases of vaginosis or need to anaerobic coverage	(Hospital treatment)	(Hospital treatment)	(Hospital treatment)
Inpatient	(Possibility to start outpatient treatment)	**Scheme A** Cefotetan, 2g, 12/12h or Cefoxitin 2g, IV, 6/6h or Ceftriaxone 1g, once a day, associated with Doxycycline 100mg, OR, 12/12h, 14 days (associate metronidazole if there is no anaerobic coverage with cephalosporin **Scheme B** Clindamycin 600mg, EV, 8/8h, associated with Gentamicin 5mg/kg/day (max. 280mg)	**Scheme A** Cefotetan, 2g, 12/12h or Cefoxitin 2g, IV, 6/6h or Ceftriaxone 1g, once a day, associated with Doxycycline 100mg, OR, 12/12h, 14 days (associate metronidazole if there is no anaerobic coverage with cephalosporin **Scheme B** Clindamycin 600mg, IV, 8/8h, associated with Gentamicin 5mg/kg/day (max. 280mg) **Note: strict ultrasound control**	**Scheme A** Cefotetan, 2g, 12/12h or Cefoxitin 2g, IV, 6/6h or Ceftriaxone 1g, once a day, associated with Doxycycline 100mg, OR, 12/12h, 14 days (associate metronidazole if there is no anaerobic coverage with cephalosporin **Scheme B** Clindamycin 600mg, IV, 8/8h, associated with Gentamicin 5mg/kg/day (max. 280mg) **Scheme C** Ampicilin/Sulbactam 3g, IV, 6/6h associated with Doxycycline 100mg, OR, 12/12h, 14 days, or Ofloxacin 400mg, IV, 12/12h, 14 days associated with Metronidazole 500mg, IV, 12/12h or Levofloxacin, 500mg, IV, once a day, 14 days associated with Metronidazole 500mg, IV, 12/12h **Note: Consider surgical treatment**

PARTNER APPROACH

All recent partners, including those in the last two months, should be called in for clinical evaluation and urethral smear test, regardless of symptoms. Partners in the last six months should also be investigated according to clinical history. Empirical treatment for *C. trachomatis* and *N. gonorrheae* must be considered regardless of apparent PID etiology (category C). Use azithromycin 1 g, p.o., associated with ceftriaxone 250 mg, IM, is recommended, both in a single dose (or ciprofloxacin 500 mg, p.o., single dose).[1,2,24]

PREVENTION

STD prevention programs reduce the incidence and complications of diseases such as PID in addition to reducing direct and indirect costs.[41]

Screening for *Chlamydia* is considered a level B recommendation with grade 2 evidence.[42] However, the benefit of screening has been overestimated because inadequate sexual practices determine high incidence of PID. Oakeshott et al.[43] have shown a tendency to a reduction in PDI among screened patients, but in the majority of cases screening results were neg-

ative, suggesting new cases. Van Valkengoed et al.[44] have estimated the probability of PID, ectopic pregnancy, and tubal infertility at 0.43, 0.07, and 0.02 %, respectively, for women with *Chlamydia*. Low et al.[45] suggest that screening in cases of miscarriage reduces PID rates after curettage. Screenings should consider the prevalence of the infection, which can reach 30%, with high rates in sexual health clinics.[46] The testing of antibodies to *Chlamydia* shows low sensitivity.[13] *Chlamydia* heat shock protein is associated with chronic inflammation and antibodies against these proteins have been tested as predictors of infertility.[47]

Table 4 shows some guidelines for PID prevention.[4]

Table 4 - PID prevention guidance

Intervention	Actions for preventing infections and their complications
STD prevention	Delaying sexual practices Reducing number of partners Correct and regular use of condoms
STD management	Early and appropriate STD treatment
Safe childbirth practices	Use of aseptic technique Effectively managing postpartum infection
Safe transcervical procedures	Use of aseptic technique Excluding infections previous to procedure
Postpartum care	Use of aseptic technique Effectively managing postpartum infection

Source: WHO, 2005. http://whocc.who.ch/reproductive-health/publications/rtis_gep/rtis_gep.pdf

FINAL CONSIDERATIONS

PID constitutes a high-prevalence disease and is associated with significant morbidity and mortality. It is a significant cause of infertility, ectopic pregnancy, and chronic pelvic pain. The most common etiologic agent is *Chlamydia trachomatis* and approximately 70% of women infected by this agent are asymptomatic. The diagnosis is clinical, showing positive predictive value between 65 and 90%. Suspicion followed by rapid diagnosis and early treatment constitutes the best way to preserve a woman's chances of reproduction in the future.

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