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Pelvic Inflammatory Disease: Current notions

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ABSTRACT : *Pelvic inflammatory disease (PID) is the clinical syndrome represents inflammation of the female,cervix,endometrium,fallopiantubes,pelvicstructure,salpingitis,pelvic peritonitis and tub ovarian abscess.PID results from the spread of Neisseria gonorrhoeae, Chlamydial trachomatis, anaerobes, Haemophilusinfluenza,G.vaginalis,Streptococcuspyogenes,gramnegative bacteria, and others. PID sequelae include: ectopic pregnancy, infertility, tubo-ovarian abscess, dyspareunia, chronic pelvic pain, premature rupture of membranes, preterm, delivery, and amnionitis. Most PID patients are treated as outpatients hospitalization for very ill patients and those meet hospitalization criteria. CDC guidelines for antibiotic treatment of PID patients are useful. Fluoroquinilones alone are no longer recommended due to emergence of resistant N.gonorrhoeae.Screening for cervical chlamydia and gonorrhea infection can prevent PID.*

KEYWODS*: Pelvic inflammatory disease (PID), Risk factors, Management, Sequelae.*

I. INTRODUCTION

Pelvic inflammatory disease (PID) refers to clinical syndrome that represents a continuum of inflammationfromthecervixtotheendometrium,fallopiantubes,andcontiguous,pelvicstructure:cervicitis, endometritis,salpingitis,pelvic peritonitis,andtuboovarianabcess[I].Each year, approximately 1 million women in the United States experience an episode of symptomatic PID.Many women with PID have minimal or no symptoms [2].PID results from direct canalicular spread of microorganisms from the vagina or endocervix to the endometrium and fallopian tube mucosa[3].Both *Neisseria gonorrhoeae* and *C.trachomatis* commonly cause endocervitis.and clinical symptoms of acute PID develop in 10% to 40% of women with these infections who do not receive adequate treatment [4].In addition to *N.gonorrhoeae* and *C.trachomatis*.a wide variety of bacteria have been isolated from the upper genital tracts of women with acute symptomatic PID,including anaerobes,gram negative rods, streptococci, and mycoplasma[5].Many of these are the same microorganisms that are found in increased concentrations in the vaginas of women with bacterial vaginosis [6].Moreover, approximately one of four women with presumed uncomplicated lower genital tract gonococcal or chlamydial infection or bacterial vaginosis, or both, is found to have histological endometritis (subclinical PID) when evaluated by endometrial biopsy[3].Uncommonly, respiratory pathogens including *Haemophilus influenza* and *Streptococcus pyogenes* have also been isolated from the upper genital tracts of women with symptomatic PID[7,8].Gold standards for PID diagnosis often impractical to achieve in the outpatient setting. Endometrial biopsy showing changes consistent with PID,transvaginal ultrasound showing thickened fluid- filled tubes, and laproscopic evidence of PID [9].Treatment regimens should be effective against gonorrhea chlamydial and anaerobes[10].The paper reviews the risk factors, diagnosis and management of PID.

II. RISK FACTORS

Age is inversely related to the rate of PID.Sexually experienced teenagers are three times likely to be diagnosed with PID than are women 25 to 29 years of age. A history of multiple sexual partners, an increased rate of acquisition of new partners within the previous 30 days, and frequent sexual intercourse with a single partner are associated with increased risk of PID[11].Women with confirmed PID commonly have concurrent bacterial vaginosis [12].Contraceptive choice modifies PID risk in a complex manner. Mechanical and chemical barriers decrease risk. Oral contraceptives have a variable effect, decreasing the clinical diagnosis but having no effect on the rate of infertility or endometrial inflammation. Intrauterine contraceptive devices (IUDs) confer a slightly increased risk of non-sexually transmitted PID in the first month after insertion [13].Other suggested association with PID include douching, menses, cigarette smoking and substance abuse [14].Although an association between the use of an IUD and increased risk of PID was documented for many years, newer studies suggest that magnitude of this association was overestimated.

Contamination of the endometrial cavity at insertion apparently results in a slightly increased risk of acute PID that is limited to the first 4 months of IUD use. Infections occurring after 4 months are believed to be

the result of acquired sexually transmitted pathogens and not the IUD itself [15].A unique role for *Actinomyces* organisms in IUD associated PID has been suggested, but this relationship remains unclear. Although as many as 4% to 8% of IUD users have Actinomyces-like organisms identified on Papanicolaou(Pap)smear, their presence has not been equated with pelvic actinomycosis, nor has the risk of subsequent pelvic infection been identified. In patients with cytology showing *Actinomyces* colonization [16].Bonacho and associates showed that removal of the IUD was associated with resolution of colonization [17].

II. DIAGNOSIS OF PID

Many episodes of PID go unrecognized. Although some cases are asymptomatic, others are underdiagnosed because the patient or the healthcare provider fail to recognize the implications of mild or nonspecific symptoms or signs(e.g. Abnormal bleeding ,dyspareunia, vaginal discharge).In one study,chlamydial infection was noted in 29% of women experiencing persistent intermenstrual bleeding while taking oral contraceptives, suggesting the presence of endometritis[18].Given the often subtle presentation of this disease and the significant reproductive sequelae associated with(infertility, topic pregnancy, chronic pelvic pain),clinicians should maintain a low threshold for the diagnosis of PID[19].Empirical treatment for PID should be considered in sexually active young women and other women at risk of sexually transmitted infections if the following minimum criteria met and no other cause for illness can be identified:(1)pelvic organ tenderness noted on bimanual examination or without manipulation of cervix and(2)microscopy showing the presence of white blood cells in the vaginal secretions. Most women with PID have either mucopurulent cervical discharge or evidence of white cells on a microscopic evaluation of the vaginal secretions. If cervical discharge appears normal and no white blood cells are found during microscopic, the diagnosis of PID is unlikely and alternative causes of pain should be investigated [20].Additional criteria that support a diagnosis of PID include bacterial vaginosis, mucopurulent cervicitis, laboratory documentation of cervical infection with *N.gonorrhoeae*or,*C.trachomatis*,oral temperature higher than38^{o}C,and elevated erythrocyte sedimentation or C-reactive protein level. Definitive criteria for PID include histologic evidence of endometritis on endometrial biopsy;transvaginal sonography or other imaging techniques showing thickened, fluid-filled tubes with or without free pelvic fluid or tuboovarian complex; and laproscopic abnormalities showing tubal purulent exudate,erythema, and edema [19].

Clinical diagnosis and grading of PID have poor specificity. In fact, women with PID associated with moderate to severe pelvic adhesions or tubal occlusion were found to have less tenderness on abdominopelvic examination and therefore to appear less ill than women with limited or no adhesion [21].Diagnostic laparoscopic should be considered in patients for empirical therapy has failed and in patients with a history of recurrent PID and negative tests for Chlamydia, gonorrhea ,and bacterial vaginosis .Endometriosis is a common alternative diagnosis in these women. Although rare, acute salpingitis can occur in the proximal stamp of patients who have undergone surgical sterilization and in women in the first semester of pregnancy [19].

III. MANAGEMENT OF PID

In the past, many specialist recommended hospitalization for all patients with PID so that bed rest and supervised treatment with parenteral antibiotics could be initiated[22].Other suggest hospitalization if patients meet the criteria that include:(1)Tuboovarian abscess(2)Peritonitis(cannot rule out other abdominal processes)(3)pregnant patients (because of high rates of preterm labor ,stillbirths,and maternal morbidity)(4)Immunocompromised patients(including HIV patients, who often have more severe presentations(5)patients cannot tolerate oral medications, and(6)possibly in young adolescents when compliance is in question[10]]. Today, most women with PID are treated as outpatients, reflecting the preponderance of patients only mild to moderate symptoms and signs. A recent prospective, randomized clinical trial compared outpatient treatment with a single dose of cefoxitin intramuscularly and multidose oral doxycycline with inpatient treatment with intravenous cefoxitin and doxycycline in women with clinical symptoms and signs of mild to moderate PID.There were no differences in response to therapy or reproductive outcome between inpatient and outpatient regimens [23].These data suggest that hospitalization can be reserved for those patients with clinically severe disease(severe illness, nausea and vomiting or high fever[19].

Treatment : Treatment consists of pelvic test and antibiotics. Antibiotic regimens must provide empirical broad- spectrum coverage of likely pathogens, including *N.gonorrhoeae*, *C.trachomatis*, anaerobes, gram negative facultative bacteria and streptococci. Several antimicrobial regimens have been effective in achieving clinical and microbiologic cure in randomized clinical trials with short term follow up. The need to eradicate anaerobes from women with PID has not been determined definitively. However, anaerobic bacteria associated with bacterial vaginosis have been isolated from the upper reproductive tract of women with PID, and those bacteria have been shown to cause tubal and epithelial destruction. One method of determining the

appropriateness of metronidazole therapy in women with PID is to determine the presence of concurrent vaginosis [19] The fluroquinolone alone are no longer recommended in the treatment of PID due to emergence of quinolone resistant *N.gonorrhoeae*. The Centers for Disease Control and Prevention have updated the published antibiotic treatment guidelines for acute PID [24].If parental cephalosporin therapy is not feasible, use of fluroquinolones (levofloxacin 500 mg PO once daily or ofloxacin 400 mg twice daily for 14 days)usually with metronidazole(500 mg PO twice daily for 14 days) may be considered if the community prevalence and individual risk is low. Tests for gonorrhea must be performed before instituting therapy. If the nucleic acid amplification test result is positive for gonorrhea, a parental cephalosporin is recommended. If culture for gonorrhea is positive, treatment should be based on results of antimicrobial susceptibility. If isolate is quinolone- resistant *N.gonorrhoeae* or antimicrobial susceptibility cannot be assessed, parenteral cephalosporin is recommended. Although information regarding other outpatient regimens is limited, amoxicillin/clavulanic acid and doxycycline or azithromycin with metronidazole have demonstrated short-term clinical cure [25].

Optimal outpatient management includes a follow-up examination performed within 72 hours after initiation of therapy. May patients may not return for this visit if they are symptomatically improved. Substantial clinical improvement with lysis of fever. Reduction in direct or rebound abdominal tenderness and reduction in pelvic organ tenderness with bimanual examination should be noted. If there is no response to therapy within 72 hours patient should be reevaluated and possibly hospitalized to confirm the diagnosis and for consideration of parenteral antibiotic therapy if they are on an oral regimen. All male sex partners of women with acute PID should be evaluated for sexually transmitted diseases, and those who had sexual contact with the patient during 60 days preceding the onset of symptoms in the patient should be empirically treated with regimens effective against *C.trachomatis* and *N.gonorrhoeae*.In many circumstances the male sex partner tests positive for chlamydia or gonorrhea,but the patient receiving the therapy is negative; such results shed light on the pathogenesis of infection[26].

Actinomycoticsalpingitis : *Actinomycesisraelii* is an anaerobic gram-positive branching, on-acid fast rod. Colonization of the lower genital tract occurs most often in the settings of IUDs(especially in long term users)and colonization portends an increased risk of PID.Colonization is usually recognized on Pap smears showing characteristic "sulfurgranules".Patients can be followed expectantly with repeat Pap smears or treated for 10 to 214 days with oral penicillin. Rarely does PID need to be removed for colonization[26].Infection ensues in a small percentage of colonized women. Clinical presentation may be irregular vaginal bleeding or mild pelvic discomfort. Pathology can reveal significant destruction,fibrosis,and structuring of pevic/ retroperitoneal structures. If there is a concern of active actinomycotic endometritis/ salpingitis in an IUD user, the patient should receive intravenous penicillin (plus the intravenous therapy for PID),the IUD should be removed, and surgery may be required.The tetracyclines, erythromycin,and clindamycin are also effective against *Actinomycetes* [26].

Tuboovarian abscess: Patients with suspected tuboovarian abscess should be hospitalized and given broad – spectrum antimicrobial drugs that include adequate coverage for gram- negative anaerobes. Failure to respond to medical therapy is suggested by lack of defervescence within 72 hours or an increase in size of mass.Eighty–five percent of abscesses with a diameter of 4 to 6 cm respond to antibiotics alone, but only 40% of those 10 cm or larger respond. Triple-agent therapy with ampicillin.clindamycin,and gentamycin would seem to be the regimen of choice, although other combination regimens have been used effectively [27,28].Surgical intervention for tuboovarian abscess that does not respond to antimicrobial therapy can be performed laproscopically,percutaneously,or transvaginally or by laparotomy. A patient with a suspected leaking or ruptured abscess should undergo immediate surgical exploration after rapid stabilization and institution of broad spectrum antibiotics [29].

Genital tract tuberculosis : Genital tract tuberculosis most common in the developing countries. Usually results from hematogenous spread from pulmonary infection, rarely from contiguous intraperitoneal disease or direct sexual intercourse.Clinically, an indolent infection.Chief presentation is infertility and also vaginal bleeding or chronic pelvicpain, Diagnosis by hysterosalpingogram may show characteristic changes, however endodermal or fallopian tube histology, which demonstrates granulomas, positive acid-fast stains, or positive culture of endometrial aspirates,isrequired.Therapy by antituberculousdrugs, and surgery if symptoms persists [26].

IV. PID SEQUELAE

After one episode of PID,a woman's risk of ectopic pregnancy increases seven times. Approximately 13% of women are infertile after single episode of PID,25% to 35% after two episodes, and 50% to 75 % after three or more episodes. If a true tuboovarian abscess is present, only 7% to 14% of patients able to conceive after treatment. After treatment for a tuboovarian complex (a less restrictive diagnostic category than tuboovarian abscess)approximately two

thirds of women attempting pregnancy are unable to conceive. Other sequelae associated with PID include dyspareunia, pelvic adhesions, and chronic pelvic pain[30].PID is also associated with premature rupture of membranes, preterm delivery, and ammonites [31].Screening for cervical chlamydia infection can prevent PID[32].

V. CONCLUSION

PID represents inflammation of female cervix to the endometrium; fallopian tubes and contiguous pelvic structure. Many cases are asymptomatic and go unrecognized or not diagnosed. Clinicians need to be aware of the implications of unrecognized PID in clinical practice.

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