

# Pelvic inflammatory disease and pelvic abscesses

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## Abstract

Pelvic inflammatory disease and pelvic abscesses have been reported as a major complication following a wide variety of obstetrical, gynaecological and surgical procedures. The aim of this review article is to emphasize the need for a more aggressive approach to detect and to treat what can be a debilitating condition that if inadequately treated may result in mortality. The large numbers of options available are discussed under the headings of: conservative management, interventional radiological management and surgical treatment. Lastly, preventive strategies are discussed, as pelvic inflammatory disease may result in tubal factor infertility, ectopic pregnancies, chronic pelvic pain and tubo-ovarian/pelvic abscesses.

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## 1. Introduction

Pelvic inflammatory disease (PID) is defined as an ascending infection of the upper genital tract leading to infection of the endometrium, fallopian tubes, ovaries and peritoneum. Tubo-ovarian abscesses (TOA) and pelvic abscesses are a relatively common complication of PID, occurring in approximately 30% of women who are hospitalized for the treatment of PID [1]. The incidence of pelvic inflammatory disease increased in the 1970s and 1980s due to an increase in sexually transmitted diseases in developed countries. Recently, there has been a small decline in the number of new cases of PID (based on outpatient and inpatient discharges gathered from hospital data bases). The incidence figures provided by the Centre for Disease Control and Prevention (CDC) states that approximately 780,000 cases of acute PID are diagnosed annually which when compared to the one million cases in the 1970s and 1980s is a 22% reduction [2].

A more recent study from Norway on women admitted to hospital with a diagnosis of PID during the years 1990–1992 and 2000–2002 and found a 26% reduction in hospitalized cases of salpingitis [3]. In this article the number of women treated for tubo-ovarian abscesses remained unchanged. During the two decades under review there was a significant reduction in sexually transmitted infections especially *Chlamydia trachomatis*, which is a success story of the screening program.

In developing countries hospital admissions for PID in the reproductive age group may reach as high as 40%. In these under privileged countries pelvic tuberculosis accounts for some tubo-ovarian abscesses through the exact incidence of genital tuberculosis is unknown.

### 1.1. Risk factors

Known risk factors for pelvic inflammatory disease and hence tubo-ovarian abscess are:

1. sexually transmitted infections (gonorrhoea, chlamydia);
2. young age (15–24 years);
3. previous history of PID;
4. multiple sexual partners;

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5. bacterial vaginosis;
6. surgical instrumentation through the cervix including termination of pregnancy.

Other factors may be demographic or are still unresolved risk factors for pelvic inflammatory disease. These risk factors are low socio-economic group, single or divorced, rape and coitus during menstruation. Substances abuse, smoking, vaginal douching and the use of an intrauterine contraceptive device (IUCD) is a risk factor especially in non-gonococcal and non-chlamydial pelvic inflammatory disease. The risk of pelvic inflammatory disease and hence pelvic abscesses in IUCD wearers tends to occur within the first 6 weeks of insertion reflecting an acquired infection at the time of placement, faulty technique or lack of aseptic precautions. A recent study quoted an incidence as high as 22.6% of pelvic abscess occurred in women who were current intrauterine device wearers [4].

Post-operative patients whether they are after abdominal surgery, such as appendectomies, diverticular disease and colectomies are at risk of developing pelvic abscesses. Other gynaecological procedures such as hysterectomies can result in pelvic abscesses due to contact with bacterial vaginosis flora. Obesity, diabetes, long operating time and increased blood loss are also risk factors for pelvic sepsis. Pelvic abscesses complicating pregnancies are rare and often result in maternal morbidity. Literature has many case reports which are usually single case reports and the diagnosis is a dilemma or is reached by excluding other more common conditions occurring during pregnancy. Pelvic inflammatory disease has also been reported after ovum retrieval, embryo transfer or hysterosalpingograms.

Obstetric sepsis related to caesarean sections, septic abortions, chorio-amnionitis and puerperal pyrexia may result in pelvic abscesses.

### 1.2. Microbial etiologic agents

Pelvic inflammatory disease is a polymicrobial condition and should reflect the organisms which infects or resides in the lower genital tract. The sexually transmitted pathogens such as *Niesseria gonorrhoea* and *Chlamydia trachomatis* are found in half to two-thirds of cases. Bacterial vaginosis and *Mycoplasma Homonis* has also been implicated especially in older women [5]. Sero-positive women for HIV who have pelvic inflammatory disease have similar micro-organisms infecting them as sero-negative HIV patients.

Another organism which is a causative factor in third world, e.g. Africa and South East Asia is tuberculosis. A number of women with pelvic tuberculosis have been found in the immigrant population in the United Kingdom.

Actinomycosis has been described as a causative organism for pelvic abscesses in women who wear IUCDs for contraception but direct spread from bowel pathology such as appendectomy has been described [6]. Direct or

haematogenous spread of *Salmonella typhi* may also cause pelvic or tubo-ovarian abscesses.

The role of viral pathogens especially sexually transmitted ones, e.g. herpes, cytomegalovirus and human papilloma virus in the causation of pelvic inflammatory disease is not known.

### 1.3. Clinical characteristics

The most common clinical symptom is lower abdominal pain, usually bilateral. The pain may be associated with any of the following: abnormal vaginal discharge, dyspareunia, nausea, vomiting and other constitutional symptoms.

The clinical presentation of a tubo-ovarian abscess or a pelvic abscess may be of great diversity. Various factors play a role such as the host defenses, the virulence of the causative organism, whether it follows a pregnancy, operation on the bowel or pelvis or if it follows a bout of acute pelvic inflammatory disease. Hence a high index of suspicion is needed to diagnose the condition usually by exclusion of other pathologies presenting in a similar manner (e.g. appendicitis, acute glomerulonephritis, etc.).

Pelvic and tubo-ovarian abscesses may have atypical presentations such as sub-acute pain which is vague and non-localized, low grade fever and weight loss. If however an abscess is preceded by an episode of severe acute pelvic inflammatory disease, as happens in 30–35% of cases, then the symptoms of bilateral lower abdominal pain is the commonest symptom. Unilateral lower abdominal pain occurs in about 10% of patients. A thorough history and physical examination in patients who have acute pelvic inflammatory disease reveals an abnormal vaginal discharge in over 50% of patients, irregular bleeding in about one-third, dysuria and deep dyspareunia in about 20% of patients.

The cardinal clinical signs are lower abdominal tenderness, bilateral adnexal tenderness and cervical excitation/motion tenderness with or without fever, tachycardia or a palpable pelvic mass. A clinical sign and symptom which accompanies *N. gonorrhoea* and *Chlamydia trachomatis* is right hypochondrial pain and tenderness which is due to inflammation of the liver capsule and fibrinous adhesions between the liver capsule and the sub-costal peritoneum. This periportal adhesion formation is termed Fritz–Hugh–Curtis syndrome and occurs in 15% of patients infected by *N. gonococcus* or *C. trachomatis*.

As the spectrum of the disease process of pelvic inflammation is so diverse, ranging on one hand from silent or sub-clinical PID to acute peritonism resulting in debilitating morbidity to even maternal mortalities. It is essential to diagnose PID early and treat it so as to reduce the risk of its sequelae.

Unfortunately however there is no clinical symptom, physical sign or laboratory investigation both sensitive and specific enough for the diagnosis of acute PID. Hence the Centre of Disease Control recommended guidelines in 2002

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