Chapter 30
Pelvic Inflammatory Disease

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DEFINITIONS

Bacterial vaginosis (BV)—A vaginal condition demonstrated by a replacement of the normal lactobacillus-predominant vaginal flora with a mixture of facultative and obligate anaerobes, mycoplasma, and other species, resulting in a malodorous discharge. The presence of *Gardnerella vaginalis* without symptoms is not synonymous with BV.

Colpotomy—Incision into and through the vaginal epithelium into a cavity (often abscess or pelvic/abdominal cavity).

Febrile morbidity—Temperature equal to or greater than 100.4°F or 38°C on two separate occasions (>4 hours apart), starting 24 hours after the initiating event.

Fever—Temperature equal to or greater than 99.6°F or 37.5°C.

Lower genital tract infection (LGTI)—An alternative term used to describe infection limited to the cervix and/or vulva and vagina.

Mollicutes—Class of organisms that includes *Chlamydia, Mycoplasma, Ureaplasma,* and other species.

Outpatient therapy—Previously synonymous with oral therapy; however, the term now includes oral, ambulatory intravenous, or intermittent injectable therapies.

Parenteral therapy—Usually denotes intravenous (antibiotic) therapy, but not necessarily given while hospitalized.

Pelvic inflammatory disease (PID)—A general term used to refer to infection and inflammation of the upper genital tract in women.

Phlegmon—A massive infiltration of inflammatory cells into infected soft tissue resulting not in an abscess, but a “woody induration” sensation upon examination/palpation.

Prevotella—Newly named genus that now includes several species formerly called Bacteroides (e.g., *Bacteroides bivia*).

Salpingitis—Infection/inflammation of one or both of the fallopian tubes (often incorrectly used as an equivalent to all PID).

Tuboovarian abscess (TOA)—A term used to describe an abscess incorporating the fallopian tube and ovary.

Upper genital tract infection (UGTI)—A more descriptive term used alternatively to PID.

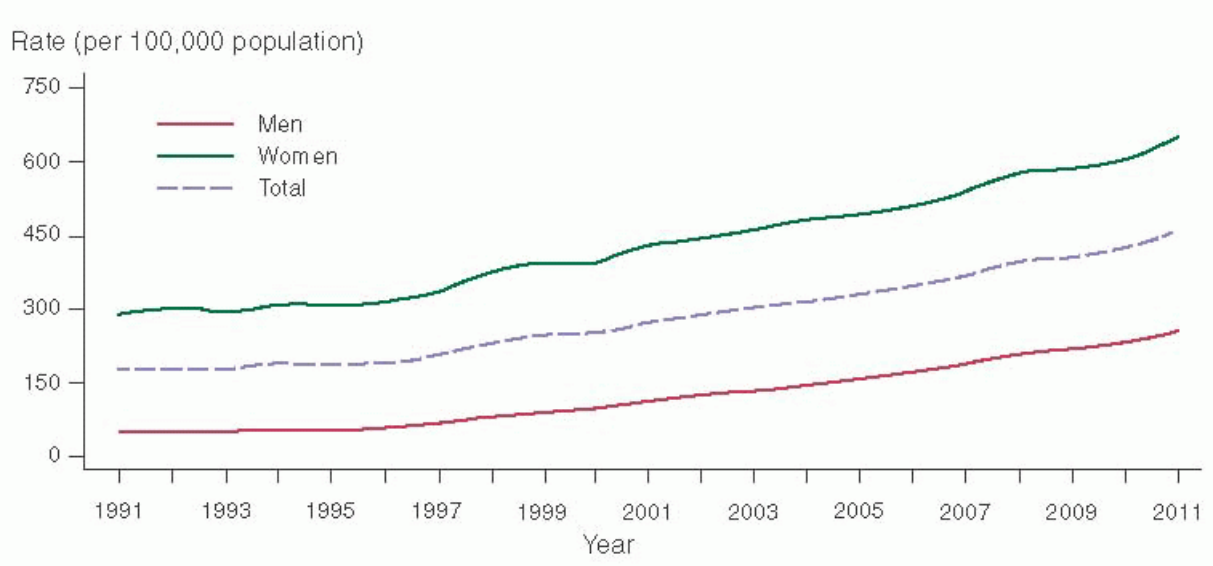

FIGURE 30.1 Chlamydia rates by sex, United States, 1991-2011. (Data from the Centers for Disease Control.)

Pelvic inflammatory disease (PID) is one of the most serious infections faced by women today. Also, it is one of the most common gynecologic reasons for hospitalization and emergency department visits in the United States each year.

Untreated or unsuccessfully treated women may suffer lifethreatening consequences, and even adequately treated women are at much higher risk for potentially serious sequelae. PID is a spectrum of diseases initially involving the cervix, uterus, and fallopian tubes. Acute PID, the acute clinical syndrome, is most often attributed to an ascending spread of microorganisms from the vagina and endocervix to the endometrium, fallopian tubes, and contiguous structures. The terms *acute PID* and *acute salpingitis* are often used interchangeably, but PID is not limited to tubal infection only. A more descriptive term to differentiate the severity and extent of various forms of PID was introduced by Hemsell and colleagues: *upper genital tract infection (UGTI*). This is differentiated from *lower genital tract infection (LGTI*) because response to treatment appears to be different in these two entities. PID has also been categorized into inpatient or outpatient treatment groups. However, this may not accurately reflect the severity of illness, but rather the predictive accuracy of the caregiver for hospitalization.

EPIDEMIOLOGY

Sexually transmitted infections (STIs) have been reported at epidemic proportions in the United States, with the Centers for Disease Control and Prevention (CDC) estimating almost 20 million new infections each year at a cost of $6 billion each year. The incidence of *Chlamydia trachomatis* infections has increased 8% since 2010, to a rate of 458 per 100,000 people, with a total of 1,412,791 cases reported in 2011 (**Fig. 30.1**).

Neisseria gonorrhoeae incidence has also increased to 4% since 2010 with a rate of 104 per 100,000 people, with more than 320,000 cases reported in 2011 in the United States. For women, acute PID is the most common and important complication of STIs. Bell and Holmes in the 1980s estimated that 1 million women a year were treated for acute salpingitis in the United States, but recent estimates by Sutton and colleagues estimated a decrease in cases of PID to approximately 770,000 per year and a 68% decrease in hospitalized PID from 1995 to 2001.

The CDC estimates that of the 750,000 to 1 million women per year who experience an episode of PID, up to 15% will become infertile as a result of the infection. Hospitalization rates have been declining over the past

decade. However, it is uncertain if this is due to decreasing incidence of the disease or a change in clinical management to treat more patients as outpatients. A consequence of the latter scenario is that despite

the long-term trend of decreased hospitalization, as confirmed by Sørbye and colleagues (35% reduction in hospitalized cases of PID), they also found that there was a 65% increase ($P = 0.013$) of tuboovarian abscesses in these patients.

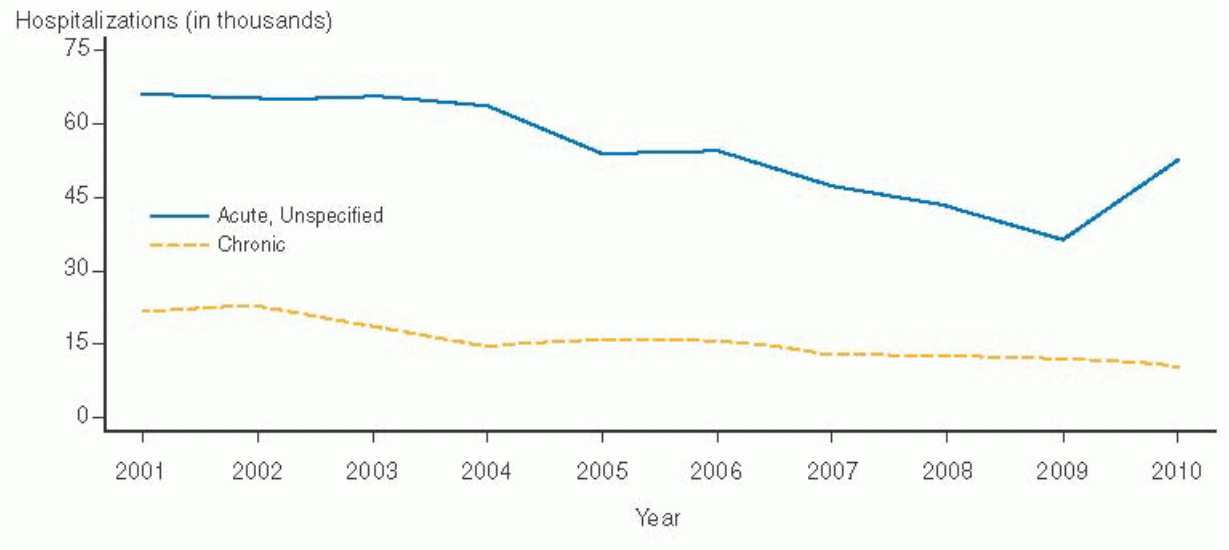

FIGURE 30.2 PID—Hospitalizations of women aged 15 to 44 years, United States, 2001-2010. (Data from the Centers for Disease Control.)

However, after hitting an all-time low in hospitalizations for PID in 2009, there was a marked increase in 2009 to 2010, and we are anxious to see if this will become a long-term trend (**Fig. 30.2**).

In addition to the 200,000 women who are hospitalized each year with a diagnosis of salpingitis or PID, the disease also generates nearly 2.5 million visits to physicians and an estimated 150,000 surgical procedures for complications every year. In terms of overall incidence, acute PID occurs in 10% to 15% of women during their lifetime, with a diagnosis in 1% to 2% of young, sexually active women each year. Therefore, PID is the most common serious bacterial infection in women aged 16 to 25 years, and the resultant morbidity exceeds that produced by all other infections combined for this age group.

ETIOLOGY

Within certain geographic areas or populations, *N. gonorrhoeae* is a common cause of PID. However, most cases of acute PID are the result of a polymicrobial infection caused by organisms ascending from the vagina and cervix to infect the lining of the endometrium and fallopian tubes. Approximately 85% of cases are spontaneous, noniatrogenic infections that occur in sexually active women of reproductive age. The remaining 15% of infections occur after procedures that break the cervical mucous barrier, such as with the placement of an intrauterine device (IUD), endometrial biopsy, or uterine curettage, which allow vaginal flora to infect the upper genital tract.

In the United States, nontuberculous PID was traditionally separated into gonococcal and nongonococcal disease, depending on the isolation of *N. gonorrhoeae* from the endocervix. However, a variety of organisms can be isolated from the endocervix. Therefore, it is difficult to determine which of these organisms cause PID and which are coexistent cervicovaginal flora representing vaginal colonizers in the upper genital tract at time of diagnosis. While upper genital tract organisms are probably more indicative of the causative organisms, they are often difficult to obtain without suspecting endocervical contamination from the diagnostic procedure. However,

studies by Martens and colleagues of transcervical cultures of the infected uterine cavities did not find significant contamination with this approach with either protected catheters or suction curettes. Bacterial organisms cultured directly from tubal fluid may commonly include *N. gonorrhoeae, C. trachomatis,* endogenous aerobic and anaerobic bacteria, and genital *Mycoplasma* species. Laparoscopic studies have demonstrated a correlation of no more than 50% between endocervical and tubal cultures, but the presence of *N. gonorrhoeae* is almost always considered an important causative factor. Even in the presence of *N. gonorrhoeae*, direct fallopian tube cultures have demonstrated that tubal infections are often polymicrobial. The type and number of species vary depending on the stage of the disease. Gonorrhea, for example, is often cultured from the cervix during the first 24 to 48 hours of the disease but is often absent later. Similarly, fewer organisms are cultured late in the disease, and anaerobic bacteria such as *Prevotella, Bacteroides, Peptococcus,* and *Peptostreptococcus* species tend to predominate. Whether these anaerobes play a causative role or increase in number and frequency as a result of the inflammatory response is uncertain. Sweet has summarized the literature by stating that in approximately one third of women with PID, *N. gonorrhoeae* is the only organism recovered by direct tubal or cul-de-sac culture, one third have a culture positive for *N. gonorrhoeae* plus a mixture of endogenous aerobic and anaerobic flora, and the remaining third have only aerobic and anaerobic organisms. Chow and colleagues and Monif and colleagues have postulated that the intense inflammatory nature of gonococcus may initiate acute PID and produce tissue damage. This damage changes the local environment, which in turn allows anaerobic and aerobic organisms from the vaginal and cervical flora to invade the upper genital tract. Both Eschenbach and Sweet have suggested that not all PID follows gonococcal infection and that acute PID initially may also have a polymicrobial etiology.

According to Sweet and Gibbs, approximately 20% of all women with salpingitis have tubal cultures positive for *C. trachomatis.* Also, both *N. gonorrhoeae* and *C. trachomatis* are found in the same individual 25% to 40% of the time. Scandinavian studies by Eilard and coworkers have reported the recovery of *C. trachomatis* from the cervix in 22% to 47% of women with acute PID. *C. trachomatis* by itself produces a mild form of salpingitis with an insidious onset. In contrast to gonorrhea, *Chlamydia* can remain in the fallopian tubes for months or years after initial colonization of the upper genital tract. Svensson and colleagues found that women with *C. trachomatis* infection at laparoscopy had the most severe fallopian tube involvement, probably because of its clinically silent or minimally symptomatic nature, which results in difficult or delayed diagnosis and therefore delayed or absent treatment. The two major sequelae of acute PID are tubal infertility and ectopic pregnancy. These have been strongly associated with prior chlamydial infection as a consequence of intratubal and peritubal adhesions.

Although *C. trachomatis* is generally believed to be one of the most common causes of PID, its etiologic role is very different when compared to *N. gonorrhoeae. N. gonorrhoeae* is a Gram-negative diplococcus with rapid growth that is due to a short reproductive cycle of about 20 to 40 minutes to divide. This results in a logarithmic increase in the number of organisms once *N. gonorrhoeae* reaches a relatively sterile area such as the endometrium or fallopian tube, where growth is relatively unimpeded. This rapid increase in the number of Gram-negative bacteria usually results in a rapid and intense inflammatory response by the woman's host defenses. The response to this rapid bacterial growth is proliferation and aggregation of white blood cells (WBCs) and their inflammatory products. Migration of this bacterial and leukocytic mixture in two directions, both through the fallopian tube to the ovary and peritoneal cavity and back to the cervix and vagina, causes the symptoms that are pathognomonic of acute PID— abdominal/pelvic pain and cervicovaginal discharge.

For years, *N. gonorrhoeae* was thought to be the primary PID pathogen. Its often severe immunologic response, mostly due to its release of lipopolysaccharide, resulted in acute and severe pain, high fevers, and a marked WBC response. This triad resulted in an easily recognized clinical course, a prompt diagnosis and often early

hospitalization, and initiation of antibiotic treatment.

Chlamydia trachomatis, however, is a slow-growing intracellular organism. Its lack of mitochondria results in its obligatory intracellular existence and also causes its growth cycle to be extremely slow compared with *N. gonorrhoeae* and other nonintracellular microorganisms. The growth cycle of *Chlamydia* is 48 to 72 hours; therefore, several weeks to months are required for the growth to reach numbers sufficient to cause clinical symptoms, if at all. Its slow growth does not induce a rapid or violent inflammatory response. This explains the slow and insidious nature of the symptoms of acute *C. trachomatis* infections. However, because of its intracellular growth cycle, the release of elementary bodies (its infectious vehicle) occurs by rupture of the cell that it has invaded. In addition, Linhares and Witkin have eloquently documented the serious immunopathologic consequences of the 60-kDa heat shock protein (hsp60) from *Chlamydia* on the fallopian tube.

Thus, the repeated occurrence of elementary body infection of susceptible cells—and their subsequent destruction by rupture—along with a chronic inflammatory response are the major mechanisms by which *C. trachomatis* causes disease in acute and chronic pelvic infections. Also, because the slow growth and the chronic inflammatory response often result in subtle clinical symptoms, treatment is often delayed or not started at all, adding to the extended tissue destruction and PID sequelae.

The lack of acute symptoms does not lessen the importance of *Chlamydia* as a PID pathogen. Not only does the tissue destruction result in severe complications such as ectopic pregnancy and infertility, but also the tissue damage provides fertile ground for the growth of secondarily infecting aerobic and anaerobic bacteria. This necrotic tissue is an excellent growth medium, and the epithelial damage enhances the breakdown of the surface defense mechanisms. The importance of *Chlamydia* was documented during the 1980s when treatment of acute PID during new antibiotic research trials was initially believed to be successfully accomplished with regimens not active against *C. trachomatis.* However, although success was evident at short-term follow-up, long-term follow-up demonstrated that treatment of *C. trachomatis* was necessary. PID regimens without *Chlamydia* coverage resulted in an increased incidence of recurrent episodes and long-term complications such as abscesses and chronic pelvic pain, with resultant increased surgical intervention. Therefore, current treatment of PID includes *C. trachomatis* coverage, even though this organism may not be the cause of the acute symptoms. Therefore, despite the lack of immediate and acute symptoms, *Chlamydia* remains an important pathogen in PID, as subtle or absent symptoms are more difficult to diagnose and treat early, resulting in much of the serious long-term sequelae found with PID. This is supported by the high incidence of *Chlamydia* antibodies in patients with acute PID, ectopic pregnancy, and infertility in several studies.

Despite the focus on these two STIs, nongonococcal and nonchlamydial pathogens cause more than 60% of PID infections. Besides *N. gonorrhoeae* and *C. trachomatis,* aerobic and anaerobic bacteria and other microorganisms, particularly other mollicutes, have been implicated as etiologic agents in acute salpingitis.

Haggerty et al., Cohen et al., Short et al., and others have all demonstrated the clinical significance of *Mycoplasma genitalium* in PID. Other novel pathogens are certain to be identified as the Human Microbiome Project identifies new, difficult-to-culture organisms found to be associated with PID.

However, *M. genitalium* is the current pathogen generating much attention after recent studies by Clausen et al. and Svenstrup and colleagues have found an association with *M. genitalium* and tubal factor infertility. More importantly, current PID treatment regimens may not adequately treat *M. genitalium*. Since it is a mollicute similar to *Chlamydia*, it lacks a cell wall and thus is resistant to cell wall-inhibiting antibiotics such as the penicillins and cephalosporins.

Also, studies by Björnelius and Falk and their colleagues in men and women with *M. genitalium* have found persistence of the organism despite treatment with levofloxacin and tetracyclines. However, these same researchers have found azithromycin to have better activity against *M. genitalium.* Moxifloxacin, a newer

quinolone, also appears to have good in vitro activity.

In the large National Institutes of Health (NIH) PID study called the PID Evaluation and Clinical Health trial, Haggerty reported that 41% of *M. genitalium*-positive women experience microbiologic failure to eradicate the organism with standard Centers for Disease Control (CDC) PID treatment regimens, and more importantly, 44% experienced clinical failure. These results will most certainly be addressed at the next CDC review of PID treatment guidelines scheduled for 2015.

Bacterial vaginosis (BV)-related organisms such as *Gardnerella, Mycoplasma hominis,* and *Ureaplasma urealyticum* have also been suggested as causal agents in acute salpingitis by Ness and colleagues. However, their role remains controversial, as other studies have not found an association with PID. Cervical cultures positive for both *M. hominis* and *U. urealyticum* have been recovered from women with PID. However, the rate of isolation can be as high as 75%, which is not statistically different from that of women who are sexually active but without PID (baseline rate of about 50%), as found by Lemeke and Lsonka. Also, Clarke et al. have published evidence that *Cytomegalovirus* (CMV) may be associated with PID, but additional confirmatory investigations are limited.

Group B streptococcus, *Escherichia coli*, and other facultative anaerobic agents are also associated with PID, but fortunately are generally adequately covered by current CDC guidelines. In light of all the new data, the main point to emphasize is that although *N. gonorrhoeae* and *C. trachomatis* are significant pathogens, they are still only associated with 30% to 40% of all PID episodes. Thus, while current standard of care requires testing for both these organisms, negative results do not mean the patient does not have PID, and treatment should be initiated or completed based on the patient clinical course or additional microbiologic testing if possible.

RISK FACTORS

Several factors that predispose to the development of acute PID have been identified. Risk factors are important considerations in both the clinical management and prevention of UGTIs. First and foremost, there is a strong correlation between exposure to STIs and PID. In the United States, recent studies have confirmed this association with the recovery of *N. gonorrhoeae* or *C. trachomatis* in 40% to 50% of patients hospitalized with acute PID. Age at first intercourse, frequency of intercourse, number of sexual partners, and marital status are all associated with the frequency of exposure to STIs and thus are associated with PID. Women with multiple partners have an increased risk (four to six times normal) for development of PID, compared with women who have monogamous sexual relations. Additional studies by Ness and colleagues have found significant factors including age at first sex, cervicitis, history of PID, family income, smoking, medroxyprogesterone use, and sex with menses.

The incidence of acute PID decreases with advancing age. Adolescent girls are at significant risk for development of acute salpingitis. Westrom reported that nearly 70% of women with PID were younger than 25 years of age, 33% experienced their first infection before the age of 19, and 75% were nulliparous. The risk for development of acute PID in a sexually active adolescent female patient was 1:8, whereas the risk was 1:80 for a sexually active woman 24 years of age or older. Several reasons have been suggested for this increased risk. The two microorganisms most commonly considered to be the inciting agents in cases of PID, *N. gonorrhoeae* and *C. trachomatis,* have a predilection for columnar epithelium. As suggested by Schaefer and by Sweet and colleagues, cervical columnar epithelium is exposed to a greater extent in younger individuals and recedes into the cervical canal with increasing age.

Clinical and laboratory studies have documented that the use of contraceptives change the relative risk for development of PID. Multiple case-control studies have shown an increased risk of acute PID in women who use an IUD. It has been estimated that IUD users have a threefold to fivefold increased risk for development of acute

PID, with the greatest risk the first 20 days after insertion. The incidence of infection has recently been described by Sufrin and colleagues in over 57,000 IUD insertions in California from 2005 to 2009. They reported an overall rate of approximately 1 in 200 patients.

Barrier methods of contraception (condoms, diaphragms, and spermicidal preparations) are effective both as mechanical obstructive devices and as chemical barriers. A nearly 60% decrease in the risk of PID has been demonstrated among women using a barrier method of contraception. Ness and colleagues found a 30% to 60% decrease in recurrent PID in women using condoms consistently.

Oral contraceptives have also been shown to reduce the risk and severity of acute PID. The mechanism for such protection remains speculative. The thicker cervical mucus produced by the progestin component of oral contraceptives is believed to inhibit sperm and accompanying bacteria penetration into the upper genital tract. The decrease in duration of menstrual flow accompanying oral contraceptive use theoretically creates a shorter interval for bacterial colonization. Svensson and coworkers reported that in addition to protecting against PID, the use of oral contraceptive pills was associated with a better prognosis for future fertility than was seen in women with acute PID using other contraceptive methods or no contraceptive methods. The pill's ability to inhibit ovulation also helps prevent an open nidus for tuboovarian abscess formation.

Surgical procedures of the female genital tract also place the patient at risk for PID. About 15% of pelvic infections occur after procedures that break the cervical mucous barrier, allowing for colonization of the upper genital tract. Eschenbach and Holmes reported that these procedures include endometrial biopsy, curettage, IUD insertion, hysteroscopy, and hysterosalpingography. The incidence of UGTI associated with first-trimester abortions is approximately 1 in 200 cases. Recent practice has emphasized the use of prophylactic antibiotics in high-risk cases to attempt to decrease the incidence of iatrogenic acute PID. A randomized trial by Jackson and a randomized trial by the Luton and Dunstable Hospital Study Group have indicated that the treatment of BV with metronidazole substantially reduced postabortion PID.

Acute salpingitis occurring in a woman with a previous tubal ligation was once believed to be rare. However, Phillips and D'Abling reported that acute PID developed in the proximal stump of previously ligated fallopian tubes in 1 of 450 women hospitalized for acute salpingitis. In addition, it is suspected that many cases may be undiagnosed because of the absence of peritoneal signs from the prevention of retrograde spillage of bacteria and inflammatory exudate in the pelvic cavity.

Previous acute PID is also a risk factor for future episodes of the disease. Another acute tubal infection develops in approximately 25% of women who have had acute PID. The exact mechanism for this increased susceptibility has not been determined, but it may be loss of the natural protective mechanisms of the fallopian tube lining against microorganisms. This increased risk may also be related to the sexual habits of the woman involved, such as reinfection from an untreated male partner. Eschenbach has documented that more than 80% of male contacts are not treated.

Genetic factors for PID have been further delineated recently. Paavonen and Taylor et al. have demonstrated that variants in the genes that regulate toll-like receptors can interfere with the innate immune system and are associated with an increased progression of infection, especially with *C. trachomatis.*

DIAGNOSIS

Acute PID presents with a broad spectrum of clinical symptoms. The differential diagnosis of acute PID includes acute appendicitis, endometriosis, torsion or rupture of an adnexal mass, ectopic pregnancy, and cervicitis.

Common clinical manifestations include lower abdominal pain, cervical motion tenderness, and adnexal

tenderness and may include fever, cervical discharge, and leukocytosis. Historically, the diagnosis of acute PID was not established unless the patient had the triad of lower abdominal and pelvic pain, fever, and leukocytosis.

Jacobson and Westrom have shown that all three criteria are present in only 15% to 30% of documented PID cases. In fact, 50% of patients initially present with a normal temperature and WBC count. Pain in the lower abdomen and pelvis is by far the most common symptom of acute PID. It occurs in more than 90% of patients at initial presentation. The pain is usually described as constant and dull and is accentuated by motion and sexual activity. Generally, the pain is of recent onset, usually less than 7 days. About 75% of patients with PID have an associated endocervical infection and coexistent purulent vaginal discharge. Nausea and vomiting are relatively late symptoms in the course of the disease. Abnormal vaginal bleeding, especially menorrhagia or spotting, is noted in about 40% of patients. The CDC has established the criteria for making the diagnosis of salpingitis based on clinical grounds. They specify that only signs of pelvic and abdominal pain are required for a potential diagnosis of PID and eliminate leukocytosis and fever as essential criteria (**Table 30.1**).

TABLE 30.1 Criteria for the Diagnosis of Acute PID

Empirical treatment of PID should be initiated in sexually active young women and other women at risk for STDs if they are experiencing pelvic or lower abdominal pain, if no cause for the illness other than PID can be identified, and if one or more of the following minimum criteria are present on pelvic examination:

- Cervical motion tenderness *or* uterine tenderness *or* adnexal tenderness

The requirement that all three minimum criteria be present before the initiation of empiric treatment could result in insufficient sensitivity for the diagnosis of PID. The presence of signs of lower genital tract inflammation (predominance of leukocytes in vaginal secretions, cervical exudates, or cervical friability), in addition to one of the three minimum criteria, increases the specificity of the diagnosis. Upon deciding whether to initiate empiric treatment, clinicians should also consider the risk profile of the patient for STDs.

More elaborate diagnostic evaluation frequently is needed because incorrect diagnosis and management might cause unnecessary morbidity. One or more of the following criteria can be used to enhance the specificity of the minimum criteria. The following additional criteria can be used to enhance the specificity of the minimum criteria and support a diagnosis of PID:

- Oral temperature >101°F (>38.3°C)
- Abnormal cervical or vaginal mucopurulent discharge
- Presence of abundant numbers of WBC on saline microscopy of vaginal secretions
- Elevated erythrocyte sedimentation rate (ESR)
- Elevated C-reactive protein
- Laboratory documentation of cervical infection with *N. gonorrhoeae* or *C. trachomatis*

Most women with PID have either mucopurulent cervical discharge or evidence of WBCs on a microscopic evaluation of a saline preparation of vaginal fluid (i.e., wet prep). If the cervical

discharge appears normal and no WBCs are observed on the wet prep of vaginal fluid, the diagnosis of PID is unlikely, and alternative causes of pain should be considered. A wet prep of vaginal fluid offers the ability to detect the presence of concomitant infections (e.g., BV and trichomoniasis).

The most specific criteria for diagnosing PID include the following:

- Endometrial biopsy with histopathologic evidence of endometritis;
- Transvaginal sonography or magnetic resonance imaging techniques showing thickened, fluid-filled tubes with or without free pelvic fluid or tuboovarian complex, or Doppler studies suggesting pelvic infection (e.g., tubal hyperemia); or
- Laparoscopic abnormalities consistent with PID

A diagnostic evaluation that includes some of these more extensive procedures might be warranted in some cases. Endometrial biopsy is warranted in women undergoing laparoscopy who do not have visual evidence of salpingitis, because endometritis is the only sign of PID for some women.

Abdominal pain can also be associated with perihepatic inflammation and adhesions, more commonly known as the Fitz-Hugh-Curtis syndrome. It is believed to develop in 1% to 10% of patients with acute PID. Signs and symptoms include right upper quadrant pain, pleuritic pain, and tenderness in the right upper quadrant when the liver is palpated (**Fig. 30.3**). Usually, the symptoms and signs of this syndrome are preceded by the clinical onset of acute PID. Due to the upper abdominal pain, this condition is often mistakenly diagnosed as either acute cholecystitis or pneumonia. Fitz-Hugh-Curtis syndrome is believed to develop from vascular or transperitoneal dissemination of either *N. gonorrhoeae* or *C. trachomatis* to produce the perihepatic inflammation. Other organisms may be involved, but limited data exist on their causality.

Jacobson and Westrom attempted to correlate the clinical diagnosis of acute salpingitis with laparoscopic pelvic findings. Of 814 women in whom laparoscopy was performed for presumed acute PID, 512 (65%) had visual evidence of salpingitis, 184 (23%) had normal visual findings, and 98 (12%) had other pelvic pathology. Because of the positive clinical findings consistent with PID, many of the patients with normal findings were suspected to have early PID with endometritis and endosalpingitis, without the visual evidence of tubal or pelvic damage. Thus, *laparoscopy* is limited as a method of diagnosing the early stages of PID, but it is crucial to rule out the 12% “other” pathology, especially the non-PID surgical emergencies, such as appendicitis, and noninfectious entities requiring different treatment modalities, such as endometriosis or hemorrhagic cyst.

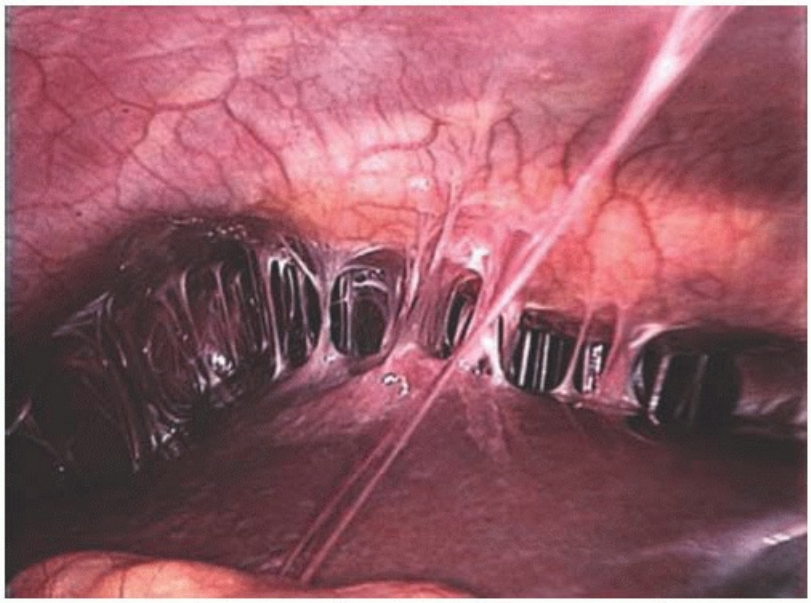

FIGURE 30.3 Violin-string adhesions of chronic Fitz-Hugh-Curtis syndrome. (Image reprinted from Medscape Reference, http://emedicine. medscape.com/, 2013, with permission, available at: http://emedicine. medscape.com/article/256448-overview)

Despite these shortcomings of early diagnosis, laparoscopic visualization of the pelvis is still important as the most

accurate method of confirming the diagnosis and assessing the severity of acute PID. However, it is logistically and economically impractical for all patients suspected of having acute PID to undergo diagnostic laparoscopy in the United States. Therefore, the diagnosis of most episodes of acute PID is often made on the basis of clinical history and physical examination. Although it is suggested that laparoscopy be offered to all patients with an uncertain diagnosis, it is strongly indicated for patients who are not responding to therapy, to confirm the diagnosis, to obtain cultures from the cul-de-sac or fallopian tubes, or to drain pus if necessary. In summary, laparoscopic studies have shown the following:

1. The clinical diagnosis of acute PID may be inaccurate.
2. Acute PID is sometimes found in patients undergoing laparoscopy for other causes of pelvic pain.
3. Laparoscopy is a relatively safe method in most cases for making the visual diagnosis of the latter stages of PID and thus assessing future fertility prognosis and planning.
4. Laparoscopy is an excellent means of obtaining cultures directly from the tube and peritoneal cavity.

The appearance of the pelvic organs can vary from erythematous, indurated, edematous oviducts, to pockets of purulent material, to a large pyosalpinx or tuboovarian abscess. However, although no disease may be evident in early stages, it is imperative to render treatment to all stages to avoid long-term sequelae.

Other less invasive methods of diagnosis have been suggested for verifying a clinical diagnosis of acute PID.

Because most cases of UGTI are associated with and preceded by LGTI, examination of the endocervix for inflammation, Gram stain, and culture for both *N. gonorrhoeae* and *C. trachomatis* are important for proper evaluation. A negative Gram-stained smear of the endocervix does not absolutely rule out upper tract infection. However, most studies have found that acute PID is rare without a concomitant increase in inflammatory cells in the vagina and the cervix.

Endometrial biopsy is one alternative to laparoscopy. Paavonen and associates reported a 90% correlation between histologic endometritis and laparoscopically confirmed salpingitis. However, results may be

delayed up to 2 to 3 days, making its clinical applicability limited.

Ultrasonography is of limited value for patients with mild or moderate pelvic PID. Thus, the routine use of sonography in patients with early PID is often not helpful. However, ultrasound is indicated in distinguishing an adnexal mass, especially in patients who demonstrate a lack of response to antimicrobial therapy in the initial 48 to 72 hours of therapy. Sonohysterography, an ultrasound examination using the instillation of saline to better define pelvic structures, is not indicated at this time for patients suspected of having PID, because no studies have been performed to demonstrate its safety in the event that pathogens are dispersed into the upper genital tract in the process of instilling the saline.

Culdocentesis, with evidence of purulent peritoneal fluid, is a helpful, but painful, method to help diagnose acute PID. With acute PID, the WBC count of peritoneal fluid is greater than 30,000 cells/mL, compared with a WBC count of 1,000 cells/mL in women without peritoneal inflammation. However, other infections, such as appendicitis and diverticulitis, among others, can also cause purulent pelvic fluid and a false diagnosis of PID.

Laboratory tests can be obtained, but their results lack sufficient sensitivity and specificity to make them an important factor in establishing the diagnosis. Leukocytosis is not a reliable indicator of acute PID, nor does it accurately correlate with the severity of tubal inflammation or need for hospitalization. Less than 50% of women with acute PID have a WBC count greater than 10,000 cells/mL. Similarly, the erythrocyte sedimentation rate (ESR), which for years was a routine laboratory test for women with acute PID, is nonspecific and is a crude indicator of severity of disease. The ESR is elevated higher than 15 mm/hour in about 75% of women with laparoscopically confirmed acute salpingitis. However, 53% of women with pelvic pain and normal-appearing pelvic organs also have an elevated ESR. Plasma proteins, such as C-reactive protein and antichymotrypsin, have been studied to determine whether they help in the diagnosis of acute PID. They have been found to be more sensitive than the ESR. Other investigators have found that *decreased or absent isoamylase in peritoneal fluid* in cases of acute PID is the best nonculture laboratory test for the disease. The major disadvantages of this test are that it requires several hours to complete and that peritoneal fluid must be obtained.

Other evaluations have revealed various inflammatory cytokines to be associated with pelvic infections; however, these tests are not yet commercially available to a useful extent.

SEQUELAE

Infertility

One fourth of all women who have had acute salpingitis experience one or more long-term sequelae. The most common is involuntary infertility, which occurs in about 20% of patients. Thus, PID ranks as one of the major causes of infertility. Before antibiotic therapy, 50% to 70% of women who had experienced UGTIs became sterile. The sequelae of infections vary from a patent oviduct, to peritubular and periovarian adhesions that may interfere with ovum pickup, and/or to complete tubal obstruction. The infertility rate increases directly with the number of episodes of acute pelvic infection. Also, women with mild disease are seven times less likely to suffer tubal obstruction than women with severe PID.

Ectopic Pregnancy

The chance of ectopic pregnancy is increased 6- to 10-fold in patients with a previous episode of acute salpingitis. Pathologic studies estimate that at least 50% of ectopic pregnancies occur in fallopian tubes damaged by previous salpingitis. The mechanism for the increased rate is believed to be interference of ovum transport through the tube or entrapment of the ovum secondary to intraluminal tubal damage.

Chronic Pelvic Pain

The chance that chronic pelvic pain will develop in a woman after acute salpingitis is four times that of control subjects without pelvic infection (20% vs. 5%). Chronic pelvic pain can be caused by a hydrosalpinx. A hydrosalpinx is presumably the end-stage development of a pyosalpinx. The pain can also be related to adhesions surrounding the tube and ovary. All patients with chronic pelvic pain believed to be caused by acute PID should undergo laparoscopy or laparotomy to establish the cause of the chronic pain and rule out other diseases such as endometriosis, which require different treatment, or the presence of pelvic adhesions, which can often be directly resolved.

A tuboovarian complex is a collection of pus within an anatomic space created by adherence to adjacent organs. The incidence of true adnexal abscess is about 10% in women with acute PID. Landers and Sweet noted a 20% rate of early treatment failure (after 48 to 72 hours) of antibiotic therapy as a result of persistent pain or enlargement of a tuboovarian

abscess or complex. In addition, according to Landers and Sweet, 31% required an operation several weeks to months after their acute infections for persistent disease or pain.

MORTALITY

Before antibiotic therapy, the mortality rate associated with acute PID was 1%. Most of these deaths resulted from rupture of tuboovarian abscesses. Today, death associated with PID is rare, as effective antibiotic treatment is frequently initiated. However, the mortality rate can still be as high as 5% to 10% for ruptured tuboovarian abscesses, if treatment is delayed or inadequate. Death is mostly the result of subsequent development of adult respiratory distress syndrome (ARDS), a condition often associated with serious infection or delayed treatment after intra-abdominal spillage of pus and bacteria.

TREATMENT

The therapeutic goals in the management of acute PID include both elimination of the acute infection and symptoms and prevention of long-term sequelae such as infertility, ectopic pregnancy, and chronic pelvic pain. Antibiotic treatment should be started as soon as cultures have been obtained and diagnosis is confirmed or strongly suspected. Treatment is based on the consensus that PID is polymicrobial in cause. Empirical antibiotic protocols should cover a wide range of bacteria, including *N. gonorrhoeae, C. trachomatis,* anaerobic rods and cocci, Gram-negative aerobic rods, Gram-positive aerobes, and *Mycoplasma* species. Despite general agreement that broadspectrum therapy is appropriate, questions persist regarding optimal therapeutic regimens.

Controversy has arisen over the issue of outpatient treatment with oral antibiotics versus inpatient treatment with parenteral antibiotics. There is limited data available to evaluate the efficacy of hospital versus ambulatory management of acute PID, due to the ethics of randomizing seriously ill PID patients to outpatient therapy. However, in the United States, three of four women with acute pelvic infection are treated as outpatients for their disease.

In Scandinavia, which has a different health care system, most women are treated as inpatients. In 2010, the CDC published recommended treatment guidelines for outpatient management of acute PID (**Table 30.2**). Some of the treatment regimens are based on the controversial premise that it may be adequate to cover just a few of the major etiologic agents (*N. gonorrhoeae* and *C. trachomatis*) involved in acute PID. As a result, studies have documented a 10% to 20% treatment failure rate for women receiving oral antibiotics as outpatients compared with a 5% to 10% failure rate for women without an abscess receiving intravenous antibiotics as inpatients, where broader coverage is used.

It is important to reevaluate patients within 48 to 72 hours of initiating outpatient therapy to determine the response of the disease. If a poor response has been obtained, the patient should be hospitalized with parenteral antibiotics and possible laparoscopic evaluation in the hope of preventing or limiting the sequelae of PID or discovering a different diagnosis, such as appendicitis or diverticulitis.

Ideally, every woman with acute PID should be hospitalized for the first few days for parenteral antibiotic treatment. Because this may not be practical because of limited economic or physical facility resources, the clinician who diagnoses acute PID in the office or emergency department is faced with the question of which patient to hospitalize. Indications for the hospitalization of patients with acute PID are also defined by the CDC in the 2010 guidelines (**Table 30.3**). In the past, the CDC has suggested that all adolescents with PID be hospitalized because of their high noncompliance rate and to optimize treatment to prevent damage to the reproductive tract, which could affect future fertility and/or result in chronic pelvic pain. Recently, Kelly and colleagues found a high incidence of recurrence in adolescents, perhaps related to poor compliance. However, the CDC's new policy is to use the same criteria for hospitalization as for older women. This recommendation is not agreed upon by all infectious disease groups because of the seriousness of inadequately treated PID in a younger, often noncompliant population.

Another indication for hospitalization is the presence of an adnexal or pelvic abscess. Outpatient therapy may not provide antibiotic levels high enough to penetrate an abscess, and rupture of the abscess may have serious consequences.

Also, women in whom the definitive diagnosis of acute PID is in question should also be hospitalized, and additional diagnostic measures should be instituted. As previously stated, at least 10% of all patients have other serious diagnoses, such as acute appendicitis, ectopic pregnancy, or adnexal torsion, and these should be ruled out. Patients with serious illness, patients with nausea and vomiting, patients who are unable to follow or tolerate outpatient therapy, and patients with a previously failed outpatient oral regimen also should be hospitalized and given parenteral antibiotics. A trial by Ness and colleagues investigating the outcomes in patients randomized to inpatient or outpatient did not find differences in short-term outcomes, but difficulties in patient selection and randomization may not permit these results to be applicable to all PID patients.

The 2010 CDC guidelines for inpatient treatment of acute PID describe two regimens (**Table 30.4**). Regimen A is a combination of oral or parenteral doxycycline plus intravenous cefoxitin or cefotetan. Other third-generation cephalosporins can be substituted, such as ceftizoxime (Cefizox) or cefotaxime (Claforan). All of these agents are effective against penicillinase-producing *N. gonorrhoeae, Peptostreptococcus,* and other anaerobic species, as well as *E. coli* and other aerobic (facultative) species. Ceftriaxone is recommended by the CDC; however, its poor anaerobic activity and lack of controlled trials in PID patients do not make it an acceptable alternative for several investigators. Doxycycline can be given intravenously if the patient is unable to tolerate oral therapy, but it must be infused very slowly to prevent pain and sclerosis of the vein. Oral doxycycline has been demonstrated to be equally effective as parenteral doxycycline because of the slow growth cycle of *Chlamydia* and the requirement of prolonged treatment.

A recent study by Viberga and colleagues investigating the microbiology of IUD-related infection found an increased recovery of *Fusobacterium* and *Peptostreptococcus* species, which generally are covered by both cephalosporin and clindamycin. A possible disadvantage of the cephalosporin-doxycycline combination is that these two antibiotics may be less than ideal for anaerobic infections such as in a pelvic abscess.

Regimen B is a combination of clindamycin and an aminoglycoside (gentamicin). This combination provides excellent activity against anaerobes, Gram-negative aerobes, and Grampositive aerobes. Historically, it has been the preferred regimen for patients with an abscess, IUD-related infections, or pelvic infections after a diagnostic or operative procedure. However, there are few data to prove that it is significantly more effective

than the cephalosporin regimens. A possible disadvantage of regimen B is that it may not provide optimal activity against *C. trachomatis* and *N. gonorrhoeae.* Clindamycin in high doses (900 mg in 8 hours) has good activity against *Chlamydia,* and in vitro studies by Martens and colleagues have demonstrated

effectiveness against 90% of *C. trachomatis* strains. Doxycycline is the most effective chlamydial agent, according to in vitro testing, but needs to be used for at least 7 days to complete treatment when the patient is switched from parenteral to posthospitalization therapy. Also, the CDC recommendation of once-daily dosing for gentamicin is not based on any data on PID patients and should be used only if indicated for renal considerations until more studies are completed.

TABLE 30.2 CDC-Recommended Treatment Regimen for Oral Therapy of Acute PID

Outpatient oral therapy can be considered for women with mild to moderately severe acute PID, because the clinical outcomes among women treated with oral therapy are similar to those treated with parenteral therapy. The following regimens provide coverage against the frequent etiologic agents of PID. Patients who do not respond to oral therapy within 72 h should be reevaluated to confirm the diagnosis and should be administered parenteral therapy on either an outpatient or inpatient basis.
Ceftriaxone 250 mg IM in a single dose
PLUS
Doxycycline 100 mg orally twice a day for 14 d
WITH OR WITHOUT
Metronidazole 500 mg orally twice a day for 14 d
OR
Cefoxitin 2 g IM in a single dose and probenecid, 1 g orally administered concurrently in a single dose
PLUS
Doxycycline 100 mg orally twice a day for 14 d
WITH OR WITHOUT
Metronidazole 500 mg orally twice a day for 14 d
OR
Other parenteral third-generation cephalosporin (e.g., ceftizoxime or cefotaxime)

PLUS

Doxycycline 100 mg orally twice a day for 14 d

WITH OR WITHOUT

Metronidazole 500 mg orally twice a day for 14 d

The optimal choice of a cephalosporin is unclear; although cefoxitin has better anaerobic coverage, ceftriaxone has better coverage against *N. gonorrhoeae*. A single dose of cefoxitin is effective in obtaining short-term clinical response in women who have PID.

> However, the theoretical limitations in its coverage of anaerobes may require the addition of metronidazole to the treatment regimen. Adding metronidazole also will effectively treat BV, which is frequently associated with PID. No data have been published regarding the use of oral cephalosporins for the treatment of PID.

Alternative Oral Regimens

Although information regarding other outpatient regimens is limited, other regimens have undergone at least one clinical trial and have demonstrated broad-spectrum coverage. In a single clinical trial, amoxicillin/clavulanic acid and doxycycline were effective together in obtaining short-term clinical response; however, gastrointestinal symptoms might limit compliance with this regimen.

> Azithromycin has demonstrated short-term effectiveness in one randomized trial, and in another study, it was effective when used in combination with ceftriaxone 250 mg IM single dose and azithromycin 1 g orally once a week for 2 wk. When considering alternative regimens, the addition of metronidazole should be considered, because anaerobic organisms are suspected in the etiology of PID and metronidazole will also treat BV.

As a result of the emergence of quinolone-resistant *Neisseria gonorrhoeae*, regimens that include a quinolone agent are no longer recommended for the treatment of PID. If parenteral cephalosporin therapy is not feasible, use of fluoroquinolones (levofloxacin 500 mg orally once daily or ofloxacin 400 mg twice daily for 14 d) with or without metronidazole (500 mg orally twice daily for 14 d) can be considered if the community prevalence and individual risk for gonorrhea are low. Diagnostic tests for gonorrhea must be performed before instituting therapy and the patient managed as follows if the test is positive.

- Laparoscopic abnormalities consistent with PID. If the culture for gonorrhea is positive, treatment should be based on results of antimicrobial susceptibility.
- If the isolate is determined to be quinolone-resistant *N. gonorrhoeae* or if antimicrobial susceptibility cannot be assessed (e.g., if only nucleic acid amplification testing is available), parenteral cephalosporin is recommended. However, if cephalosporin therapy is not feasible, the addition of azithromycin 2 g orally as a single dose to a quinolone-based PID regimen is recommended.

Each regimen stresses two concepts: the polymicrobial etiology of acute pelvic infection and the necessity of protecting against *C. trachomatis* and *N. gonorrhoeae.* With both regimens, the CDC recommends a minimum of at least 24 hours of intravenous treatment after clinical improvement. Both protocols also require completion of a 14-day course of oral antibiotics (doxycycline or clindamycin) to eradicate slow-growing organisms such as *C. trachomatis* and other mollicutes.

TABLE 30.3 Criteria for Hospitalization of Patients with Acute PID

In women with PID of mild or moderate clinical severity, outpatient therapy yields short- and long-term clinical outcomes similar to inpatient therapy. The decision of whether hospitalization is necessary should be based on the judgment of the provider and whether the patient meets any of the following suggested criteria:

- Surgical emergencies (such as appendicitis) cannot be excluded.
- The patient is pregnant.
- The patient does not respond clinically to oral antimicrobial therapy.
- The patient is unable to follow or tolerate an outpatient oral regimen.
- The patient has severe illness, nausea and vomiting, or high fever.
- The patient has a tuboovarian abscess.

No evidence is available to suggest that adolescents benefit from hospitalization for treatment of PID. The decision to hospitalize adolescents with acute PID should be based on the same criteria used for older women. Younger women with mild-to-moderate acute PID have similar outcomes with either outpatient or inpatient therapy, and clinical response to outpatient treatment is similar among younger and older women.

TABLE 30.4 CDC-Recommended Treatment Regimens for Parenteral Therapy of Acute PID

REGIMEN A

Cefotetan 2 g IV every 12 h

OR

Cefoxitin 2 g IV every 6 h

PLUS

Doxycycline 100 mg orally or IV every 12 h

Because of the pain associated with intravenous infusion, doxycycline should be administered

orally when possible. Oral and IV administration of doxycycline provides similar bioavailability.

Parenteral therapy can be discontinued 24 h after clinical improvement, but oral therapy with doxycycline (100 mg twice a day) should continue to complete 14 d of therapy. When tuboovarian abscess is present, clindamycin or metronidazole with doxycycline can be used for continued therapy rather than doxycycline alone, because this regimen provides more effective anaerobic coverage.

Limited data are available to support the use of other second- or third-generation cephalosporins (e.g., ceftizoxime, cefotaxime, and ceftriaxone), which also might be effective therapy for PID and could potentially replace cefotetan or cefoxitin. However, these cephalosporins are less active than cefotetan or cefoxitin against anaerobic bacteria.

Regimen B

Clindamycin 900 mg IV every 8 h

PLUS

Gentamicin loading dose IV or IM (2 mg/kg of body weight) followed by a maintenance dose (1.5 mg/kg) every 8 h. Single daily dosing (3-5 mg/kg) can be substituted.

Although use of a single daily dose of gentamicin has not been evaluated for the treatment of PID, it is efficacious in analogous situations. Parenteral therapy can be discontinued 24 h after clinical improvement; ongoing oral therapy should consist of doxycycline 100 mg orally twice a day or clindamycin 450 mg orally four times a day to complete a total of 14 d of therapy. When tuboovarian abscess is present, clindamycin should be continued rather than doxycycline, because clindamycin provides more effective anaerobic coverage.

Alternative Parenteral Regimens

Limited data are available to support the use of other parenteral regimens. The following regimen has been investigated in at least one clinical trial and has broad-spectrum coverage.

Ampicillin/sulbactam 3 g IV every 6 h

PLUS

Doxycycline 100 mg orally or IV every 12 h

Ampicillin/sulbactam plus doxycycline is effective against *C. trachomatis, N. gonorrhoeae*, and anaerobes in women with tuboovarian abscess. One trial demonstrated high short-term clinical cure rates with azithromycin, either as monotherapy for 1 wk (500 mg IV for one or two doses followed by 250 mg orally for 5-6 d) or combined with a 12-day course of metronidazole.

Alternative inpatient parenteral regimens are included in the 2010 CDC PID guidelines (**Table 30.4**). The CDC lists only the β-lactamase inhibitor combination ampicillin-sulbactam (Unasyn), but piperacillin-tazobactam combination has been demonstrated by Hemsell and colleagues, Sweet and colleagues, and others to have excellent in vitro and in vivo activity against PID and its pathogens and does have an FDAapproved indication for the treatment of pelvic infections.

MANAGEMENT OF SEX PARTNERS

According to the CDC:

Male sex partners of women with PID should be examined and treated if they had sexual contact with the patient during the 60 days preceding the patient's onset of symptoms. If a patient's last sexual intercourse was less than 60 days before onset of symptoms or diagnosis, the patient's most recent sex partner should be treated. Patients should be instructed to abstain from sexual intercourse until therapy is completed and until they and their sex partners no longer have symptoms. Evaluation and treatment are imperative because of the risk for reinfection of the patient and the strong likelihood of urethral gonococcal or chlamydial infection in the sex partner. Male partners of women who have PID caused by *C. trachomatis* and/or *N. gonorrhoeae* frequently are asymptomatic.

Sex partners should be treated empirically with regimens effective against both of these infections, regardless of the etiology of PID or pathogens isolated from the infected woman. Even in clinical settings in which only women are treated, arrangements should be made to provide care or appropriate referral for male sex partners of women who have PID. Expedited partner treatment and enhanced patient referral are alternative approaches to treating male partners of women who have chlamydial or gonococcal infections.

HIV INFECTION

Differences in the clinical manifestations of PID between human immunodeficiency virus (HIV)-infected women and HIV-negative women have not been well delineated. In early observational studies, HIV-infected women with PID were more likely to require surgical intervention. In recent, more comprehensive observational and controlled studies, HIV-infected women with PID had similar symptoms when compared with uninfected controls. They were more likely to have a tuboovarian abscess, but responded equally to standard parenteral and oral antibiotic regimens when compared with HIV-negative women. The microbiologic findings for HIV-positive and HIV-negative women were similar, except for (a) higher rates of concomitant *M. hominis, Candida*, streptococcal, and HPV infections and (b) HPV-related cytologic abnormalities among those with HIV infection. Regardless of these data, whether the management of immunodeficient HIV-infected women with PID requires more aggressive interventions (e.g., hospitalization or parenteral antimicrobial regimens) had not been determined.

Intrauterine Contraceptive Devices

Also, per CDC guidelines:

IUDs are popular contraceptive choices for women. Both levonorgestrel and copper-containing devices are marketed in the United States. The risk for PID associated with IUD use is primarily confined to the first 3 weeks after insertion and is uncommon thereafter. Given the popularity of IUDs, practitioners might encounter PID in IUD users. Evidence is insufficient to recommend the removal of IUDs in women diagnosed with acute PID. However, caution should be exercised if the IUD remains in place, and close clinical follow-up is mandatory. The rate of treatment failure and recurrent PID in women continuing to use an IUD is unknown, and no data have been collected regarding treatment outcomes by type of IUD (e.g., copper or levonorgestrel).

SURGICAL MANAGEMENT

If the diagnosis is under question, or the patient is responding poorly, surgical evaluation and/or management

are indicated.

Laparotomy should generally be reserved for patients with surgical emergencies such as ruptured abscesses or definitive treatment of failed medical management. Laparoscopy, however, is an underused but usually helpful procedure for diagnosis, prognosis, and possibly treatment of PID. Laparoscopic evaluation should be considered in all patients with a differential diagnosis of PID and without laparoscopic surgery contraindications.

Laparoscopy is important not only to diagnose PID but also to rule out surgical emergencies, such as appendicitis and ruptured abscesses. It also prevents inappropriate management of patients with noninfectious problems, such as endometriosis. These patients need additional surgical and medical management, not antibiotic therapy and delayed diagnosis. In addition, evaluation of the extent of the inflammatory process in confirmed PID is helpful in establishing a prognosis and further management plan if initial treatment fails. Patients with evidence of current or previous abscesses have a higher failure rate with antibiotic therapy. Also, treatment of unilateral abscesses may necessitate surgical management to avoid the spread of the infection to the other tube and ovary.

Cultures obtained from the peritubal region or from the peritoneal cavity can also be helpful for identifying organisms resistant to initial management. This has become increasingly important in light of the increasing rate of clindamycinresistant anaerobes and the elimination of intravenous metronidazole from the CDC-recommended inpatient guidelines. Laparoscopic management of PID that appears helpful includes copious lavage of the pelvis with normal saline or preferably Ringer solution. Antibiotic inclusion in the lavage fluid has not been demonstrated to be helpful to date.

Laparoscopic manipulation or drainage of documented pelvic abscesses has been attempted by several investigators. Henry-Suchet and associates reported the successful use of laparoscopy to diagnose and drain tuboovarian abscesses in 50 women. Adhesions were lysed, and the abscesses were drained through the laparoscope. All patients received intravenous antibiotics. Forty-five of the fifty (90%) patients were cured. Reich and McGlynn had a similar experience in 25 women with pelvic abscesses treated laparoscopically. Four of seven women desiring pregnancy conceived, and two women had unplanned pregnancies. However, the diagnosis of abscesses was not uniform in these studies. Also, it is of concern that similar results will not necessarily be demonstrated in less experienced hands. Anatomically, drainage of abscesses within the pelvic cavity by laparoscope will not drain the entire abscess contents out of the pelvic cavity, and despite how extensive the lavage or laparoscopic removal is, pus and bacteria will be spilled and exposed to the pelvic cavity. This is contrary to the natural defense mechanism of the body of isolating and containing the inflammation-causing organisms within an abscess. Therefore, laparoscopic drainage of pelvic abscesses should be undertaken only by experienced laparoscopic surgeons and with the patient's full understanding of all other options.

Laparotomy with extensive pelvic surgery was often recommended in the past, before the development of broad-spectrum antibiotics.

If a patient has been hospitalized on several occasions for acute exacerbation of PID with bilateral tuboovarian abscesses to the point where the future surgical risk increases

significantly, definitive surgical intervention may be indicated. The operation should be done when the infection is quiescent, if possible. The surgery may still be difficult, but there will be fewer complications than when patients are operated on in the acute phase of the infection. The timing of the operative intervention is important. There should be complete absorption of the inflammatory exudate surrounding the focus of the infection, as seen radiologically. Bimanual pelvic examination should be possible without producing a marked or persistent febrile response. It has been suggested that definitive surgery be delayed for 2 to 3 months after the recent

exacerbation for more complete resolution of the infection. Ideally, the patient should have a normal ESR, WBC, and hematocrit, and relatively nontender pelvic organs, except possibly with motion.

Kaplan and associates recommended more aggressive management in patients who exhibit either no clinical response or only partial response after 24 to 72 hours. Their approach included a total abdominal hysterectomy and bilateral salpingooophorectomy and was thought to reduce the protracted period of intensive medical therapy in a group of patients who would eventually require surgery. They noted that conservative management of their cases usually resulted in protracted periods of intensive care and repeated hospital admissions, and rarely in subsequent pregnancies. However, the early surgical intervention described above was associated with several incidences of injury to the bowel and additional postoperative complications. Also, patients with acute pelvic abscess are frequently young, and future childbearing is often desired. Even with uterine and ovarian preservation, pregnancy is difficult without intervention such as in vitro fertilization for most patients. Conservation of ovarian function for these young women is an important benefit of medical management for the rest of their adult lives.

Older studies of the management of patients with pelvic abscess, which emphasized the early use of surgery, are no longer pertinent, because modern antibiotic drugs were not available then. Collins and Jansen in 1959 had an early failure rate of 10% for conservative medical therapy. However, 113 of their 174 patients required later surgery, which corresponds to a late failure rate of 65%. Two decades later, Ginsburg and associates reviewed cases of 160 patients treated for tuboovarian abscess during the years 1969 to 1979. The early failure rate with broad-spectrum antibiotics was high at 31%, but the late failure rate was only 21%. Thus, with an average followup period of 25.5 months, 48% did not require later surgery. Subsequent reports by Hager and colleagues and by Landers and Sweet support conservative management, as better antibiotics have been introduced.

POSTERIOR COLPOTOMY

When conservative management fails and a pelvic abscess is noted dissecting the rectovaginal septum, drainage by way of colpotomy may be possible.

In a classic article, Wharton described various techniques of vaginal drainage of pelvic abscess. Today, posterior colpotomy is done to evacuate pus and to establish drainage from a pelvic abscess that presents in the cul-de-sac.

There are three requirements for colpotomy drainage of a pelvic abscess:

1. The abscess must be predominantly midline.
2. The abscess should be adherent to the cul-de-sac peritoneum and should dissect the rectovaginal septum to assure the surgeon that the drainage will be extraperitoneal and that pus will not be disseminated transperitoneally.
3. The abscess should be cystic or fluctuant to ensure adequate drainage.

Occasionally, a cul-de-sac abscess can be successfully drained without dissecting the septum. However, the serosal surface of the abscess should be adherent to the cul-de-sac peritoneum. Ultrasonography may be helpful in locating the pockets of pus.

After adequate anesthesia, the patient is placed in the lithotomy position. It is essential that a thorough examination of the pelvis be performed under anesthesia so that the operator knows the size and position of the mass that is to be drained.

After preparation and draping in the dorsal lithotomy position, the posterior lip of the cervix is grasped with a tenaculum and drawn down and forward. The vaginal epithelium of the posterior vaginal fornix is incised just below the reflection of the vaginal mucosa onto the cervix, and the transverse incision is widened with a pair of

long scissors (Fig. 30.4A). The incision must be large enough to allow adequate exploration and drainage of the abscess cavity with the index finger. The cul-de-sac peritoneum and abscess wall are punctured with a long Kelly clamp (Fig. 30.4B). As the abscess wall is perforated, there is a definite sensation of puncturing a cystic cavity. If blood or pus is present, this is soon seen coming into the upper vagina. The jaws of the clamp are spread, and the flow

of liquid from the cul-de-sac is increased. A sample of the purulent exudate is sent to the microbiology laboratory for appropriate culture and sensitivity. Collection of the specimen anaerobically with a capped syringe with rapid transport to the laboratory allows the more fastidious flora to be defined. A direct smear for Gram stain is also made from the pus examined for predominating organisms.

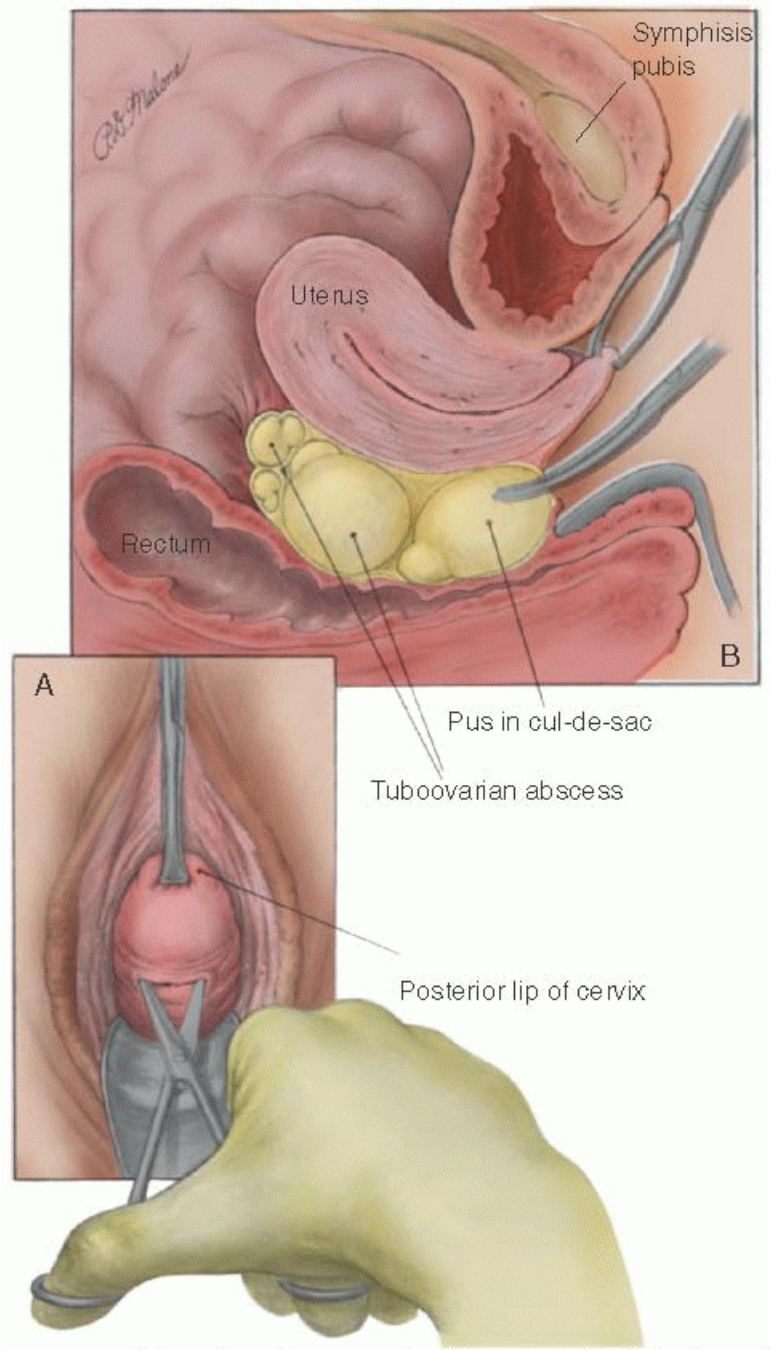

FIGURE 30.4 Posterior colpotomy. **A:** A transverse incision is made through the vaginal mucosa at the junction of the posterior vaginal fornix with the cervix. **B:** A Kelly clamp is thrust through the abscess wall.

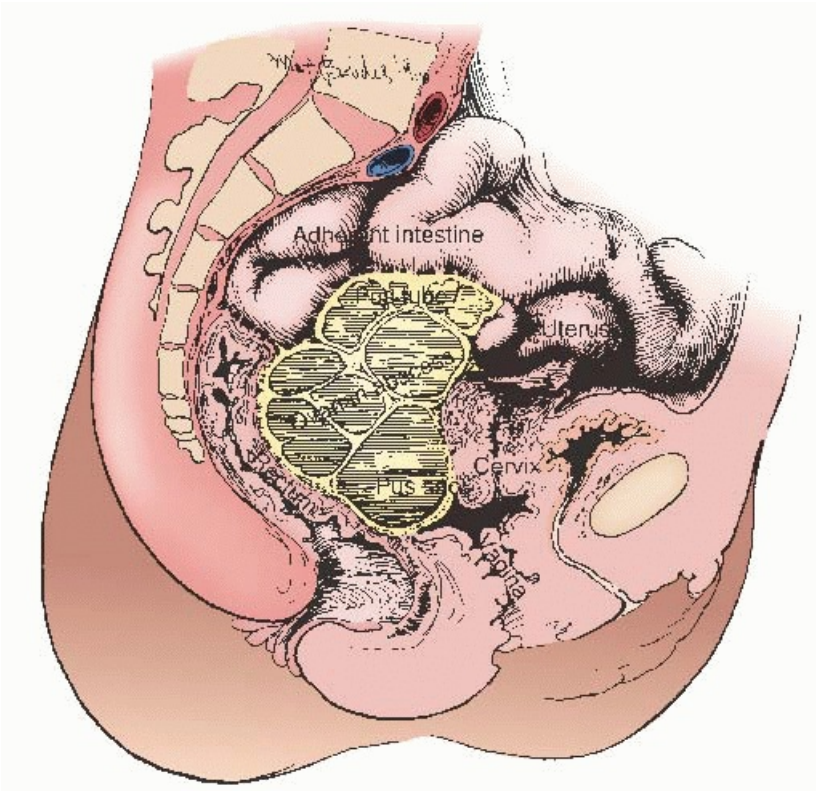

FIGURE 30.5 Pus may be contained within the tuboovarian abscess and within other pockets in the pelvic cavity.

There may be more than one compartment in an abscess cavity (**Fig. 30.5**). It is desirable to insert an index finger in the cavity and explore. Fibrous adhesions within the cavity can be gently broken. If another abscess wall is felt, it can often be cautiously and safely punctured under the guidance of a finger. Exploration and manipulation should be done carefully to avoid intraperitoneal rupture of the abscess or perforation of the bowel. To allow adequate drainage, the vaginal incision should be at least 2 cm wide. If pus has been obtained, one or two drains are inserted into the abscess cavity and anchored by balloon or with fine absorbable suture to permit easy removal. Penrose or closed suction drainage systems can be used. These should be left for several days until drainage stops and to allow air to prevent the reaccumulation of anaerobic organisms. Wharton has emphasized the importance of prolonged drainage. A suture or two may be required to control bleeding from the vaginal mucosa. However, if a mushroom (Malecot) catheter is used for drainage, it should be removed in 48 to 72 hours to prevent significant fibrosis that could hinder removal.

Patients in whom the abscess does not meet the criteria for colpotomy drainage often require laparotomy and direct drainage. Transabdominal, transvaginal, and transrectal drainage has been attempted to avoid the expense and complications of laparotomy and for patients in whom laparotomy is contraindicated.

PERCUTANEOUS DRAINAGE

Experience with *percutaneous drainage* of intraabdominal and pelvic abscesses under ultrasonographic or computed tomographic (CT) guidance has been reported by Olak and associates and by others. Worthen and Gunning used percutaneous catheter drainage of 11 abscesses in nine patients and achieved a cure rate of 77%. Two patients required surgical intervention subsequently. In 19 patients, simple percutaneous aspiration of 23 abscesses was successful, with a 94% cure rate. The attempt at aspiration failed in seven patients (**Fig. 30.6**). The Grady Memorial Hospital experience, as reported by Tyrrel and associates, is similar. CT-guided percutaneous drainage in eight patients with tuboovarian abscess resulted in recovery without surgery in seven. One patient had marked clinical improvement but still required a posterior colpotomy. No complications occurred. Loy and associates have reported that the simultaneous use of real-time pelvic ultrasonography can facilitate transvaginal drainage of a pelvic abscess. If patients do not respond to intravenous antibiotics and percutaneous

drainage or aspiration, surgical intervention is required. MRIassisted drainage has been utilized and is helpful when overlying gas patterns from the intestines or abscess cavity obscure ultrasonic imaging for needle placement.

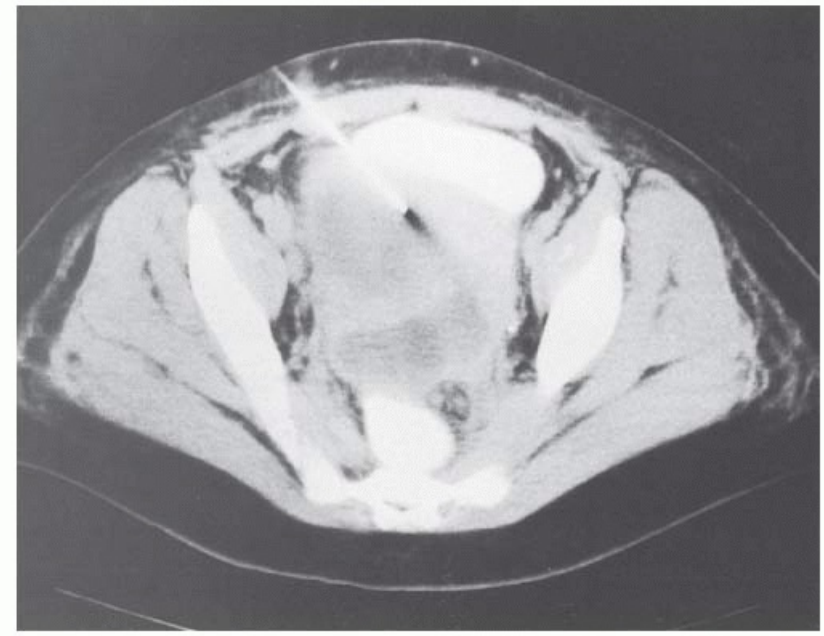

FIGURE 30.6 Transabdominal needle aspiration of a pelvic abscess under guidance of CT. Drainage tube is also placed.

The long-term effects of pus and organisms released into the pelvis from the puncture site are unknown. However, shortterm success rates are good, and surgical drainage of acute abscess is the basic principle. Therefore, needle drainage can be considered with proper patient selection and appropriate informed consent, which includes other management options.

EXPLORATORY LAPAROTOMY

If *exploratory laparotomy* is necessary, the patient can be positioned in Allen universal stirrups. A lower abdominal transverse Maylard incision is ideal because it affords good exposure to the lateral adnexal pelvic organs and pelvic side walls. Pelvic adhesions should be released, and the bowel should be packed off before the pelvic dissection commences. During the dissection, free pus is often spilled, and the upper abdomen should be isolated from this, if possible. When a ruptured abscess is encountered, the exudate is collected and sent immediately to the laboratory for Gram stain, anaerobic and aerobic cultures, and antimicrobial sensitivity studies. The easiest way to obtain the material for anaerobic culture is simply to collect it in an airtight syringe and to submit a small piece of the abscess wall in an airtight container. The easiest place to begin the dissection is in the round ligament, which is the most consistently

available and identifiable landmark. Following the round ligament medially always leads to the uterine corpus. Variations in the usual technique for the operation may be required because of extensive disease, dense adhesions, indurated and edematous tissue, and distorted anatomy. For example, it is sometimes convenient to perform the central dissection first (i.e., a subtotal hysterectomy). This allows more space and adequate exposure to perform the required adnexal surgery. Tuboovarian inflammatory masses may be found densely adherent in the cul-de-sac to the uterus, to the posterior surface of the broad ligament, and to the lateral pelvic sidewall. There is risk of injury to the ureters, sigmoid, rectum, and small intestines. The method of dissection used depends on the nature of the adhesions. Soft, fresh adhesions can be broken gently and easily with finger dissection. Dense fibrotic adhesions must be carefully dissected and cut with scissors. The dissection can be

especially difficult and risky if pelvic tissues are intensely indurated, as in ligneous pelvic cellulitis. If the infundibulopelvic ligament can be clamped, cut, and securely ligated, one can gain access to the lateral retroperitoneal space and identify the ureter. This facilitates a safe dissection of the abscess wall away from the ureter. In cases with extensive disease involving one or both adnexa, the use of preoperative ureteral catheterization may be helpful in identifying the location of the pelvic ureters. With tuboovarian abscess, the anatomic limits of the ovary may be difficult to define. If the ovary is to be removed, it should be removed completely to prevent subsequent development of ovarian remnant syndrome.

When both adnexa must be removed, a hysterectomy should be entertained if extensive uterine involvement is suspected. In some cases, only a subtotal hysterectomy is feasible. However, if the lower uterine segment can be visualized, the cervix can usually be excised after removal of the adnexa and the uterine corpus. The operative field should be copiously irrigated. The vaginal vault should be left open for drainage. A Penrose drain can be inserted and then removed several days later. Suspension of the vaginal vault and reperitonization of the pelvis are accomplished in the usual manner, if possible. A routine closure of the abdominal incision is performed. Jackson-Pratt suction drains are often placed above the fascia and brought out through a separate incision.

Because the patient has been placed in the Allen universal stirrups for laparotomy, ureteral integrity can be confirmed as discussed elsewhere. Five milliliters of indigo carmine is given intravenously, and a cystoscope is placed in the bladder. Blue dye can then be seen flowing from each ureteral orifice.

In the past, it was standard practice to do a bilateral salpingo-oophorectomy in almost all patients who had a laparotomy for acute pelvic abscess. This practice was based on the belief that the disease is almost always severe in both adnexa. Recent studies have suggested that as many as 25% to 50% of patients will have a relatively normal tube and ovary on one side. This may be especially true of patients whose infection is associated with IUD use. Golde and associates reported that 37 of 85 patients (44%) with tuboovarian abscesses confirmed at operation had unilateral abscesses; 20 were using an IUD. The studies of Landers and Sweet, Hager and Majmudar, and Ginsburg and coworkers also found a higher percentage of unilateral adnexal disease than was previously reported. In light of these findings, conservative adnexal surgery may be possible in some patients. We have no hesitation in leaving a relatively normal tube and ovary at the time of hysterectomy with removal of the opposite adnexa for acute pelvic abscess. When the uterus is removed and the continuity between the conserved tube and the lower genital tract is interrupted, there is little risk of a new infection. If a strictly unilateral pelvic abscess is found at laparotomy, removal of the affected tube and ovary only, leaving in the uterus and the opposite adnexa, is acceptable in a patient who wishes to preserve fertility. However, in vitro fertilization techniques may be required to accomplish pregnancy. Such a patient will then still have a risk of recurrent tuboovarian abscess. It is especially important that her sexual partner be examined and receive treatment when indicated.

In recent years, there have been advances in reproductive technology that allow infertile patients to conceive and carry pregnancies to term under the most extraordinary circumstances. It has been possible, for example, to accomplish a successful pregnancy in a woman who has a uterus but no ovaries by instillation of a donated fertilized ovum into a suitably prepared uterus. Such a sophisticated procedure is not available to a large number of patients. However, in light of the recent findings of the risk of estrogen plus progesterone treatment from the Women's Health Initiative Study, the option of leaving in the uterus when bilateral salpingo-oophorectomy is to be performed should be discussed with the patient, especially if she is young and nulliparous.

In summary, patients with an acute pelvic abscess should be hospitalized for treatment with parenteral broad-spectrum antibiotics. Surgery is indicated if the diagnosis is uncertain, if intraperitoneal rupture is diagnosed or suspected, or if the patient fails to respond to medical management.

RUPTURED PELVIC ABSCESS

A tuboovarian or pelvic abscess can rupture spontaneously into the rectum or sigmoid colon, into the bladder, or into the free peritoneal cavity. A pelvic abscess almost never ruptures spontaneously into the vagina unless the patient has previously damaged epithelium such as from a previous posterior colpotomy for drainage of an abscess. Under these circumstances, a recurrent pelvic abscess can dissect along the tract of the previous posterior colpotomy incision and drain spontaneously through the vagina.

Spontaneous drainage through the rectum or sigmoid colon usually occurs in a patient whose abscess is too high to drain with a posterior colpotomy. In other words, although the abscess is fluctuant and midline, it is not yet dissecting the rectovaginal septum. While waiting for the abscess to come down, a sudden unexpected improvement in the patient's condition is noted, and she will confirm that pus has begun to drain through the anus. Further improvement in her condition usually occurs. A posterior colpotomy is not needed and, indeed, is contraindicated because doing so could cause a rectovaginal fistula to form.

Spontaneous drainage through the bladder is rare. It occurs most commonly in elderly women with chronic abscesses developing from ruptured sigmoid diverticula. Only rarely does a chronic tuboovarian or pelvic abscess rupture and drain through the bladder, causing secondary infection of the bladder. When the abscess is removed with laparotomy, a defect in the bladder wall is noted. The indurated tissue around the defect should be removed and the defect closed with 3-0 delayed absorbable suture in two layers. A Foley catheter can be left in place for 10 to 14 days while healing of the bladder wall takes place.

Of all the complications that can result from PID, intraabdominal rupture of a tuboovarian abscess is the most life threatening. Mortality from this complication is due to septic shock and the complications of generalized peritonitis, and the mortality rate can approach up to 10% in patients with warm shock.

Abscesses can rupture spontaneously or iatrogenically after bimanual examination or accidental trauma. Bacteriologic study of the contents of the abscess has historically been unrewarding; a specific organism has been isolated in less than 50% of cases. The gonococcus is rarely identified in a pelvic abscess. Careful aerobic and anaerobic cultures often demonstrate the presence of a mixed infection that includes anaerobic organisms. McNamara and Mead reviewed the results of three separate studies that demonstrated 31 positive isolates of anaerobes in 30 patients with a pelvic abscess. Landers and Sweet have also confirmed similar findings in their series.

Diagnosis of Ruptured Tuboovarian Abscess

The major clinical symptom of ruptured tuboovarian abscess is acute, progressive pelvic pain that is usually so severe that the patient can accurately identify the time and place of its occurrence. In a classic series from the Johns Hopkins Hospital reported by Vermeeren and Te Linde, the average age of patients with a ruptured tuboovarian abscess was 33 years, which is at least 10 years older than the average age of patients with acute PID. Approximately 2% of these patients are postmenopausal. To our knowledge, only two cases of ruptured tuboovarian abscess in a pregnant patient have been reported. Often, there is a history of recurrent attacks of PID, with a sudden increase in the severity and extent of abdominal pain during a recent exacerbation of infection. On examination, the patient appears seriously ill and dehydrated, with rapid, shallow respirations. The abdomen is distended and quiet, with diminished or absent bowel sounds. Signs of generalized peritonitis, direct and rebound tenderness, muscle rigidity, and shifting dullness may be noted. A pelvic mass is palpable in only approximately 50% of cases. Tachycardia is common. Shock can be present or can develop while the patient is under observation. It is due to accumulation of fluids in peripheral tissues and later failure of compensatory vasoconstrictor mechanisms. The patient's temperature is usually greater than 101°F, but it can also be normal

and even subnormal late in the course. The leukocyte count is likely to be more than 15,000, but it also can be normal, if the neutrophils are being depleted. Severe leukopenia is an ominous sign. A culdocentesis is a valuable diagnostic aid and was positive for purulent material in 70% of the cases in the Mickal and Sellmann series. An abdominal radiograph usually shows a paralytic ileus, sometimes evidence of free fluid in the peritoneal cavity, and atelectasis in the lung bases. A CT scan of the pelvis and abdomen is most helpful and will usually confirm the pelvic abscess with free purulent fluid in the upper abdomen. It may also suggest an alternative diagnosis such as a ruptured appendix or acute cholecystitis.

Treatment of Ruptured Tuboovarian Abscess

The longer the delay in the operative treatment of ruptured tuboovarian abscess, the greater the primary mortality rate. In the series by Vermeeren and Te Linde from the Johns Hopkins Hospital, death occurred less than 90 hours after the time of rupture in 88% of fatal cases, both operative and nonoperative.

As time passes after rupture of a tuboovarian abscess, septic peritonitis becomes more severe and generalized. The passage of time allows the development of septic shock from greater absorption of bacteria and bacterial endotoxins and secretion of great quantities of fluid into the peritoneal cavity across inflamed peritoneal surfaces. Fluid shifts from the intravascular compartment to interstitial spaces as a result of the increased vascular permeability of the inflamed peritoneal membrane. This leads to hypovolemia, decreased cardiac output, decreased central venous pressure, hypotension, vasoconstriction, increased peripheral resistance, decreased tissue perfusion, metabolic acidosis, ARDS, decreased renal glomerular perfusion and filtration with decreased urine flow, severe hypoxemia, multiple organ system failure, and ultimately death. The prompt diagnosis and treatment of intraperitoneal rupture of a tuboovarian abscess is essential to minimize the risk of mortality of generalized peritonitis.

The treatment of patients with ruptured tuboovarian abscess can be divided into three phases: preoperative, operative, and postoperative.

Preoperative Phase

Surgery should be undertaken after rapid but adequate preoperative preparation. The patient should be typed and crossmatched for 2 to 4 units of packed red blood cells. Monitoring of central venous pressure is essential for proper evaluation of the hemodynamics of this condition, because many patients are dehydrated, in shock, and anemic. Swan-Ganz catheter placement may be preferable because it allows pulmonary capillary wedge pressure and pulmonary artery pressure determinations that are helpful in assessing the adequacy of fluid replacement and in detecting fluid overload. Variable amounts of fluid, sometimes tremendous amounts, are lost into the peritoneal cavity and intestinal tract because of peritonitis. Blood chemistry determinations (e.g., serum electrolytes, creatinine, glucose, bilirubin, and alkaline phosphatase) should be obtained and intravenous fluids, preferably Ringer lactate, started immediately. Crystalloid solutions for fluid volume resuscitation are preferred for most patients with septic peritonitis. It may be advantageous to use partial colloid resuscitation in some patients with evidence of cardiopulmonary dysfunction, because a smaller total volume is required. An excess of intravenous crystalloid solution may result in fluid overload.

Vigorous broad-spectrum intravenous antibiotic therapy should be instituted. The most active antimicrobials with FDA approval for female pelvic infections include piperacillin-tazobactam 3.75 g every 6 hours and ertapenem 1 g every 24 hours. Dosage adjustments need to be made due to the patient’s renal status. Changes can be made to single or combination therapies once culture results and sensitivities return.

An indwelling urethral catheter is used to monitor fluid intake with hourly urine output. Combating shock is a primary concern throughout treatment. Clinical assessment of respiratory function should be made. A distended tender abdomen may cause rapid, shallow respirations and use of accessory muscles for ventilation. Arterial

blood gases may indicate mild hypoxemia, in which case oxygen should be administered. If anemia is severe, blood transfusion should be started before surgery.

When the patient has been properly prepared, immediate surgery should be undertaken. The results of treatment are better if major metabolic and hemodynamic problems are corrected before operation, but one cannot wait even a short amount of time in treating a critically ill patient with septic peritonitis.

Operative Phase

The anesthetic of choice depends on the preference and experience of the anesthesiologist and the medical condition of the patient.

The operation should be performed as rapidly as possible. Because speed as well as access to the upper abdomen may be required, a lower midline incision should be used. It can be quickly extended above the umbilicus if necessary. The patient

should not be put in the Trendelenburg position until the abdomen is packed off, and no more of a dependent position should be used than is needed to prevent further dissemination of pus into the upper abdomen. When the abdomen is opened, any odor that is present should be noted. An unpleasant, putrid odor is indicative of infection with anaerobic organisms. Pus from the abdomen should be collected correctly for both aerobic and anaerobic culture and for Gram stain and should be promptly transported to the laboratory. Organisms grown should be tested for sensitivity to various antibiotics.

The operation of choice in a patient with a ruptured abscess is the removal of the free pus. In patients bordering or in shock, removal of the abscess, the uterus, the tubes, and usually the ovaries is often necessary. Occasionally, it is possible to leave an ovary in a patient with a ruptured pelvic abscess. However, reoperation in a seriously ill patient is often not an option, so all tissue suspected to be infected should be removed. If rupture has occurred from a strictly unilateral tuboovarian abscess, with a relatively normal tube and ovary on the opposite side, a unilateral salpingo-oophorectomy can be performed, especially if the patient is young. However, the risk of a recurrent abscess in the opposite tube and ovary is high if the uterus is also left in place. When the uterus is removed along with the tuboovarian abscess, the risk of recurrent abscess in the opposite adnexa is reduced. When hysterectomy is performed, usually a total hysterectomy can be done. However, even in the best surgical hands, a subtotal hysterectomy is faster than a total one and is sometimes justified. Although we believe firmly in total hysterectomy, we do not believe in performing it when the danger of total hysterectomy exceeds the danger from a retained cervix. Except in the young patient, it is often better to remove the corpus than to perform a unilateral adnexectomy alone. Furthermore, the opposite adnexa are significantly involved in most patients, and subsequent operation may be necessary if conservation of one side is practiced, as was required in 35% of Pedowitz and Bloomfield's cases. This is contrary to what has been described earlier in the surgical treatment of an unruptured abscess, because the risk of incomplete eradication of the immediate infection in an acutely ill patient with rupture, peritonitis, and possibly septic shock is much higher. Therefore, definitive surgical treatment is usually recommended in severely ill patients with ruptured abscess.

The technical performance of the procedure may be difficult, but it is similar to that described earlier for laparotomy followed by failed colpotomy drainage or suspected rupture. Anatomy is distorted, dependable landmarks are obscured, and tissues are thick, edematous, friable, and inflamed. Loops of densely adherent intestine must be separated carefully to avoid injury. Injury to the serosa of distended bowel occurs commonly and often requires repair. Any entry into the lumen of the bowel must be recognized and repaired. Retroperitoneal planes of dissection can be used to advantage in identifying the ureters and removing inflammatory adnexal masses. Otherwise, it is likely that a fragment of ovary will be left behind, which can subsequently cause signs and symptoms of the ovarian remnant syndrome. As much of the remaining abscess

wall as possible should be removed without causing unnecessary additional bleeding. Pieces of the abscess wall can be left adherent to the pelvic sidewall and cul-de-sac.

The upper abdomen should be carefully explored for collections of pus in the subdiaphragmatic and subhepatic regions. If an upper abdominal abscess is found, it may be necessary to place a closed suction drain into the abscess cavity through the upper abdominal wall.

Before the incision is closed, the abdominal cavity should be irrigated with copious quantities of warm sterile saline to remove remaining bacteria and debris. There is always some fear of dissemination of the infection by copious irrigation. However, this disadvantage is far outweighed by the benefit of diluting and removing bacteria and necrotic debris. We do not add antiseptics or antibiotics to the irrigating solution. If hemostasis is poor or if considerable necrotic material is left behind, there may be some benefit from peritoneal drainage with closed suction catheters. Closed suction drains can be placed through a separate stab wound in the abdominal wall, through the cul-de-sac, or through the vaginal vault when a total hysterectomy has been done, but the drainage of free peritoneal exudate in the upper abdomen is of no therapeutic value.

The abdominal incision is closed with a Smead-Jones technique or with a continuous suture taking large bites of tissue. A monofilament suture of polypropylene or nylon should be used. Retention sutures can be placed but are not usually necessary. The incision should be irrigated with warm saline. When there has been gross contamination of the incision, the subcutaneous fat and skin should be left open and packed lightly with gauze soaked in an antibiotic or dilute acetic acid solution. The wound is repacked daily and inspected. In 4 to 5 days, if the tissues are healthy, the incision is closed secondarily with sutures. Alternatively, the edges can be drawn together with sterile adhesive strips.

Postoperative Phase

Postoperative care should consider shock, infection, ileus, and fluid imbalances. Complications of the late postoperative period include undrained or recurrent pelvic and abdominal abscesses, intestinal obstruction, intestinal fistulas, incisional breakdown with or without evisceration, pulmonary embolus, continued sepsis, and disseminated intravascular coagulation. Serious medical problems such as uncontrolled diabetes or renal or pulmonary failure (ARDS) further complicate recovery from this potentially lethal disease.

Septic shock should be combated with blood (when indicated for a hemoglobin less than 7.0 g), Ringer lactate, respiratory support, and, if necessary, vasoactive substances. Infection is controlled by the continued aggressive use of broad-spectrum intravenous antibiotics until the patient can take antibiotics orally. When the results of the antibiotic sensitivity studies on the operative specimen are available, a change to more effective or safer agents should be considered, but only if the patient shows evidence of continued sepsis. Antibiotics should not necessarily be changed on the basis of sensitivity studies if the patient is improving clinically. Sometimes, the patient's condition improves initially, only to show signs of recurring intraabdominal infection the 2nd week after operation. Under these circumstances, it is appropriate to change antibiotics. Antibiotics should be continued until the patient is afebrile with only a mild leukocytosis or mildly elevated C-reactive protein and is able to eat a regular diet. Too long a period of treatment with antibiotics may result in complications such as pseudomembranous enterocolitis or fungal superinfection.

The semi-Fowler position may help prevent subphrenic and subdiaphragmatic abscess formation. Patients with signs of continued intraabdominal sepsis should have CT scans to identify collections of pus. If found, CT-directed drainage may be possible.

Constant intestinal suction by means of a long intestinal tube is a very important feature of postoperative care. A dynamic ileus persists postoperatively for a variable period and is best treated with the long intestinal tube until there is evidence of peristalsis and the patient is passing flatus.

Close attention to fluid balance and blood chemistry determinations is mandatory. Frequently, patients with ruptured tuboovarian abscess may have poor kidney function. The fluid output and serum creatinine should be followed closely.

PRIMARY OVARIAN ABSCESS

A primary ovarian abscess is an entity distinctly different from tuboovarian abscess. A tuboovarian abscess is one in which the abscess wall is composed of fallopian tube and ovarian parenchyma. A primary ovarian abscess, on the other hand, is one in which the infection occurs in the parenchyma of the ovary. Unlike tuboovarian abscess, it is an unusual condition. Interest in primary ovarian abscess was stimulated by the 1964 report of Willson and Black. According to a review by Wetchler and Dunn, 120 cases had been reported by 1985. Its frequency may be increasing, although still rare, as Askenazi and colleagues in 1994 reported a 0.2% to 2.2% rate of ovarian abscesses following transvaginal oocyte retrieval and transcervical embryo transfer.

Although bacteria can gain access to the ovarian parenchyma by hematogenous or lymphatic spread, it is probable that most primary ovarian abscesses occur because bacteria present around the ovary gain access to the parenchyma through a break in the ovarian capsule. This can occur naturally by ovulation, or it can be broken by a surgical procedure. Bacteria come from the fallopian tube, from the vagina during or after hysterectomy or any colpotomy procedure, from intrauterine infection associated with an IUD or another transcervical procedure, or from appendicitis, diverticulitis, or any other condition that is associated with peritonitis. A primary ovarian abscess is usually unilateral. However, its occasional occurrence simultaneously in both ovaries and during pregnancy seems to support the rare hematogenous or lymphatic spread, or both. Primary ovarian abscess has been reported secondary to infections at distant sites (tonsillitis, typhoid, parotitis, and tuberculosis).

Diagnosis of an unruptured primary ovarian abscess can be difficult because of the variable clinical presentation. Lower abdominal pain and fever are usually present. Lower abdominal and pelvic tenderness and an adnexal mass may be present, but the pelvic examination is sometimes not helpful, due to pain and guarding. Although an event predisposing to primary ovarian abscess (e.g., surgery, IUD use, appendicitis, or systemic infection) may be uncovered in the history, the event is sometimes remote. Ultrasonography and CT can be helpful in identifying an abscess cavity. If an ovarian abscess ruptures, the clinical picture is much the same as in ruptured tuboovarian abscess, with abdominal distention, direct and rebound tenderness, ileus, and sometimes shock. The patient appears gravely ill, and the need for immediate surgery is usually obvious.

The management of patients with primary ovarian abscess is similar to the management of patients with acute tuboovarian abscess. If the abscess is not ruptured, medical management with antibiotics for both anaerobic and aerobic organisms plus supportive care is indicated. A failure to respond or deterioration in the patient’s condition suggests alteration in antibiotic coverage or possible exploratory surgery, or both, to remove the abscess. Ruptured ovarian abscess requires immediate laparotomy after a brief but intense effort to stabilize the patient and start antibiotic therapy. At operation, only the affected ovary need be removed. The tubes and the uterus can be conserved.

If both ovaries are involved, they should be removed. For a patient who is not interested in conception in the future, the uterus and both tubes can also be removed to decrease the possibility of the need for reoperation. If the patient is interested in pregnancy, the uterus and fallopian tubes can be left in place for possible implantation of a donated egg in the future.

SURGERY FOR CHRONIC PELVIC INFLAMMATORY DISEASE

Although the gonococcus may be responsible for initiating acute salpingitis, which is short-lived, residual chronic

salpingitis is usually due to secondary invaders, both aerobic and anaerobic, or perhaps to an initial infection with *C. trachomatis.* As a result of the initial infection or from subsequent secondary exacerbations, the fimbria can become occluded and the tubes bound to the ovaries with adhesions. In addition, the bowel can become adherent to the broad ligament and the adnexal structures, and the fascia and loose connective tissue of the broad ligament can be converted into an indurated, brawny structure typical of ligneous induration. This can extend to include tissues beneath the peritoneum on the lateral pelvic sidewall, where ligneous pelvic cellulitis can cause ureteral obstruction. If the chronic infection persists, serious effusion from the inflammatory process within the endosalpinx produces a hydrosalpinx that can ignite periodically with secondary subacute pelvic infection or can progress to produce a pyosalpinx and tuboovarian abscess. If the subacute infection is left untreated or is treated inadequately, spontaneous intraabdominal rupture or leakage of an old tuboovarian abscess can occur. In a review of this subject, Heaton and Ledger identified this problem principally in premenopausal women, with only 1.7% of patients with a tuboovarian abscess being postmenopausal. However, when tuboovarian abscess is diagnosed in postmenopausal women, Protopapas et al. found that 47% had a concomitant gynecologic malignity. They concluded, “Conservative treatment of TOAs has no place during the menopause.”

The signs and symptoms of chronic PID that most often require surgical treatment include severe, persistent, progressive pelvic pain, usually bilateral, although occasionally localized in one of the lower abdominal quadrants; repeated exacerbations of PID requiring multiple hospitalizations and recurrent medical treatment; progressive enlargement of a tuboovarian inflammatory mass, especially if it cannot be distinguished from a neoplastic tumor of the ovary; severe dyspareunia related to the chronic pelvic infection; and bilateral ureteral obstruction from ligneous cellulitis. It was formerly accepted that a history of previous colpotomy for drainage of a pelvic abscess was sufficient reason in itself to justify definitive abdominal surgery later for removal of the uterus and adnexa. However, several patients have become pregnant after posterior colpotomy for drainage of a cul-desac abscess or have remained relatively free of symptoms for long periods. Today, previous posterior colpotomy for pelvic abscess drainage is not a sufficient indication by itself for definitive abdominal surgery later when the patient is stable.

Selection of Operative Technique

The final decision regarding the proper operation for the surgical management of chronic PID is usually made with the abdomen open. Consideration must be given not only to the pathologic lesions found at operation but also to the patient’s age, parity, desire for children, previous history of pelvic disease, and other associated pelvic disease and symptoms. Because a knowledge of all these is essential to the best surgical judgment, the operator should be thoroughly familiar with the patient, her history, and her desires.

In the surgical management of chronic PID, the question of removal or retention of the ovary at the time of hysterectomy

and salpingectomy has been left open to conjecture and individual surgical opinion in most instances. This question was the subject of a study by Weiner and Wallach of the ovarian histology in ovaries removed from patients with PID. In 40 consecutive women who underwent oophorectomy during surgical treatment of PID, nearly 50% of the removed ovaries were free of inflammatory disease and demonstrated normal follicular activity. The study concluded that ovarian histology was usually normal among patients who gave no history of dysfunctional uterine bleeding. Therefore, the menstrual history of such patients should be helpful in the decision regarding ovarian conservation or ablation. Kirtley and Benigno have reviewed the Emory experience with ovarian conservation at the time of surgery for PID. In this series, 98 (82%) patients who required surgery had a total abdominal hysterectomy and bilateral salpingo-oophorectomy. In 22 patients (18%), either part or all of an ovary was retained. Of these 22 patients, 15 were available for follow-up hormonal assays. The mean follow-up

time was 58 months. Cyclic ovarian function was confirmed in all but two patients. In the two patients with ovarian failure, other significant disease processes were also present. No patient suffered a complication as a result of adnexal conservation. We believe that normal ovarian tissue should be conserved at the time of definitive surgery for PID (**Fig. 30.7**). The release of peritubal adhesions in mild chronic PID is indicated occasionally in women in whom future childbearing is desired, as long as the tubes can be shown to be patent, usually by transfundal chromotubation after the lower uterine isthmus is occluded by a Ziegler clamp. This type of procedure provides the most rewarding pregnancy rate of all types of tubal reconstructive surgery. More often, one tube is hopelessly closed, and the opposite tube is patent after release of adhesions. In such a case, unilateral salpingectomy may be required if reconstructive surgery of the blocked tube is not possible (**Fig. 30.8**).

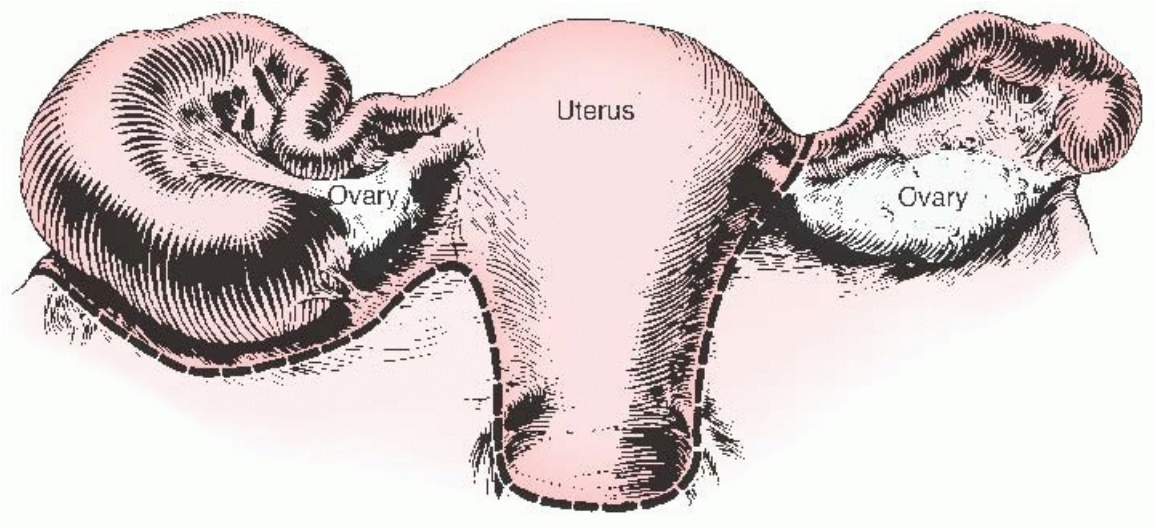

FIGURE 30.7 Total abdominal hysterectomy and unilateral salpingo-oophorectomy from extensive chronic salpingo-oophoritis. A small hydrosalpinx on the opposite side can be left in to preserve blood supply to the ovary.

In most instances of surgery for chronic PID, total abdominal hysterectomy and bilateral salpingo-oophorectomy are necessary to remove the primary tubal pathology because of inflammatory damage of both tubes and ovaries. Total abdominal hysterectomy and bilateral salpingo-oophorectomy (**Fig. 30.9**) have been performed for severe actinomycosis infection.

If the uterus is removed and an ovary is preserved, it may be preferable to leave the entire adnexa in place in the absence of active tubal infection rather than compromise the venous drainage or the arterial blood supply to the ovary with subsequent cystic changes that may require an additional operative procedure later. Once the continuity of the tubal lumen from the uterine cavity is broken, the chronically inflamed tube does not usually produce subsequent symptoms, as shown by Falk in his series of cases with interstitial tubal resection. A small hydrosalpinx on the same side as the normal ovary can also be left in place so that ovarian blood supply is not disturbed during an attempt to remove the tube. When it is considered advisable to remove both adnexa because of the extent of the tuboovarian disease, a total hysterectomy may also be considered unless the uterus is hopelessly encased in pelvic scar tissue and densely adherent to the pelvic viscera.

In the optimum case, especially in a young woman who wishes to establish or maintain the possibility of future fertility, conservative surgery may be desirable, with the hope that pregnancy can be accomplished through in vitro fertilization techniques. In this situation, the uterus and one adnexa should be conserved, and the ovary should be positioned in the pelvis so an ovum can be harvested later through the laparoscope or through the vagina. As mentioned earlier, if the patient wishes, the uterus can be left in place even though both tubes and ovaries have been removed.

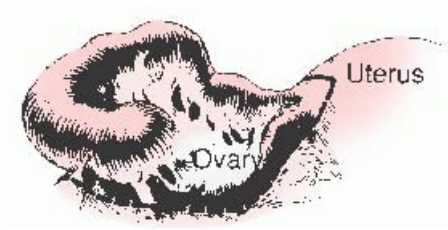

FIGURE 30.8 When significant chronic PID involves only one adnexum and preservation of uterine function is indicated, a unilateral salpingo-oophorectomy can be performed.

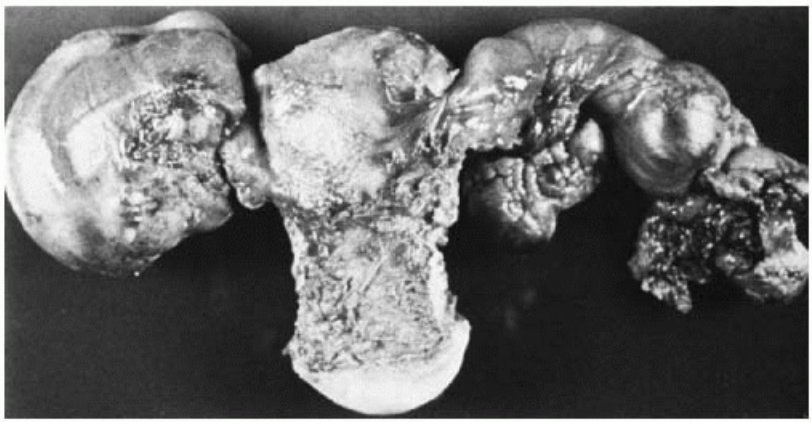

FIGURE 30.9 Total abdominal hysterectomy and bilateral salpingooophorectomy for severe pelvic actinomycosis.

Salpingectomy for Chronic Salpingitis

At the time of surgery for the treatment of chronic PID, every effort should be made to retain uninvolved organs. Unilateral salpingectomy should be considered when the oviduct is hopelessly destroyed by the disease process and presents as a large hydrosalpinx.

Once the abdomen has been opened and the extent of disease evaluated, the adhesions binding the tube are cut, and the tube is freed. It is held by a Kelly clamp placed on the mesosalpinx just beneath the fimbriated end. The mesosalpinx is then clamped and cut, with a succession of small bites taken as close to the tube as possible (**Fig. 30.10A**). Removal of a chronic hydrosalpinx can also be done laparoscopically.

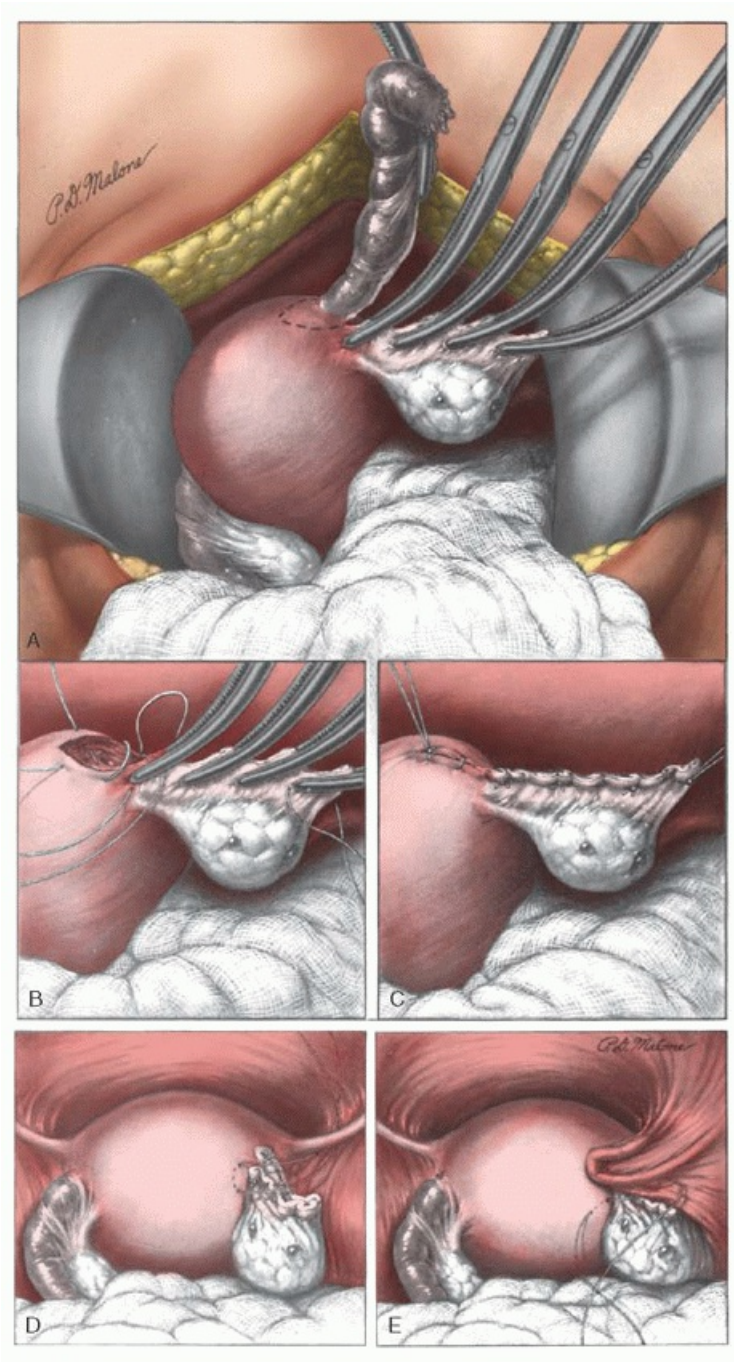

FIGURE 30.10 Salpingectomy. **A:** Mesosalpinx is clamped with multiple Kelly clamps and cut. *Dotted lines* indicate cornual excision, which is elective. **B:** Cornual wound is closed with 2-0 delayed absorbable suture. **C:** Mesosalpinx vessels are transfixed. **D:** Mattress suture is placed to cover operative area. **E:** Round ligament and broad ligament cover operative area.

Keeping the operative trauma as far as possible from the ovary that is to be retained lessens the danger of imperiling its blood supply. Experience has shown that the ovary whose tube has been removed is more apt to become cystic than the ovary whose tube has been left undisturbed.

The tube is excised at the uterine cornu in a wedge-shaped manner, as indicated in **Figure 30.10B**. A wide, figure-of-eight 2-0 delayed absorbable suture is placed in the cornu before the wedge is excised and is tightened as the interstitial portion of the tube is removed. If there is palpable extension of the inflammation at the uterine cornu (so-called salpingitis isthmica nodosa), the wedge may be large.

The wound in the uterus is closed with one or more 2-0 delayed absorbable figure-of-eight sutures (**Fig. 30.10B**). The vessels in the mesosalpinx are ligated with transfixion 3-0 delayed absorbable sutures. The

advantage of the transfixion suture is that it does not slip off the tissue when tied as the clamp is withdrawn (Fig. 30.10C).

A mattress suture of 3-0 delayed absorbable material is used to bring the broad and round ligaments over the cornual wound (Fig. 30.10D). This suture passes just beneath the round ligament, so that the ligament is not strangulated when the suture is drawn tight. When this suture is tied, the cornual wound is covered with the broad ligament, and the uterus is suspended to some extent in a manner similar to that used in the Coffey suspension. Usually, a second mattress or interrupted suture is necessary to cover the mesosalpinx completely, as shown in Figure 30.10E.

Salpingo-Oophorectomy for Chronic Salpingitis

As in salpingectomy, the abdomen is entered through a transverse Maylard incision. The chronic tuboovarian inflammatory mass is first dissected free, and the infundibulopelvic ligament is identified. It is doubly clamped with Ochsner clamps, and a third clamp is applied to control back bleeding (Fig. 30.11A). The ureter must be identified before the infundibulopelvic ligament is clamped, cut, and ligated.

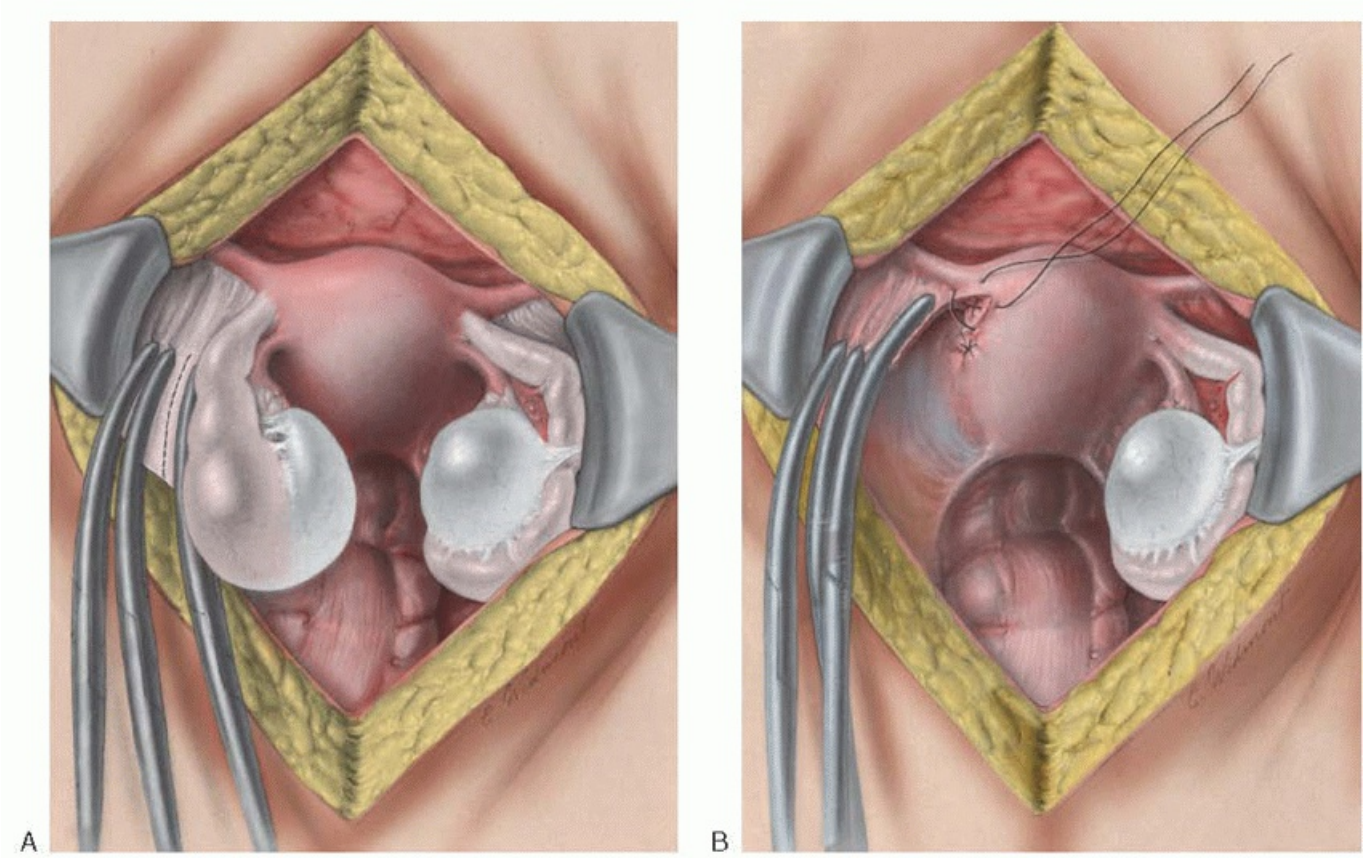

FIGURE 30.11 Salpingo-oophorectomy. **A:** The infundibulopelvic ligament is doubly clamped. Another clamp is placed to control back bleeding. *Dotted line* indicates incision. **B:** A suture has been placed to ligate the ascending uterine vessels just below the cornual incision. The cornual incision is closed with a figure-of-eight suture of 2-0 delayed absorbable material.

After the infundibulopelvic ligament is cut and ligated, the remainder of the broad ligament attachment of the tube and the ovary is clamped, cut, and ligated. The uterine end of the tube and the ovarian ligament are excised from the uterus in a wedge-shaped manner. The ascending uterine vessels are ligated just below the cornual wound, and the cornual incision is closed with a 2-0 delayed absorbable figure-of-eight suture (Fig. 30.11B).

The infundibulopelvic ligament is doubly ligated with 2-0 delayed absorbable sutures, and the vessels in the broad ligament are ligated with 3-0 delayed absorbable sutures. The cornual wound is peritonized, and the

uterus is suspended to some degree by bringing the round and the broad ligaments over the uterine cornu with a mattress suture of 2-0 delayed absorbable material, as shown in **Figure 30.11C**. An attempt should be made to remove the tuboovarian inflammatory complex completely. If a fragment of ovary is left attached to the lateral pelvic peritoneum or the broad ligament, the ovarian remnant syndrome may develop later. To prevent this, a retroperitoneal approach may be required.

Identification of the Ureter

Identification of the course of the ureter in a pelvis in which the anatomy has become obliterated as a result of PID is one of the most important techniques for the gynecologic surgeon. In the surgical treatment of this disease, one may find a tuboovarian inflammatory mass that is located between

the leaves of the broad ligament and extends to the lateral pelvic wall. It is not uncommon for the ligneous induration of the thickened parietal peritoneum to obscure completely the location and course of the pelvic ureter so that dissection of the diseased adnexa produces a surgical risk to the urinary tract, requiring great technical skill to avoid ureteral injury. Knowledge of the normal anatomic location of the pelvic ureters is essential so that these vital structures can be identified before an attempt is made to remove the adnexal masses. Division of the round ligament allows access to the lateral pelvic wall beneath the peritoneum. After the round ligament is divided, the peritoneum is incised inferiorly toward the internal cervical os and superiorly just lateral to the infundibulopelvic ligament. The peritoneum is easily reflected medially away from the pelvic sidewall with finger dissection, and the ureter is identified. It remains attached to the peritoneum. If there is difficulty with this procedure, the ureter can usually be identified as it crosses over the common iliac artery just above its bifurcation, and it can be traced downward.

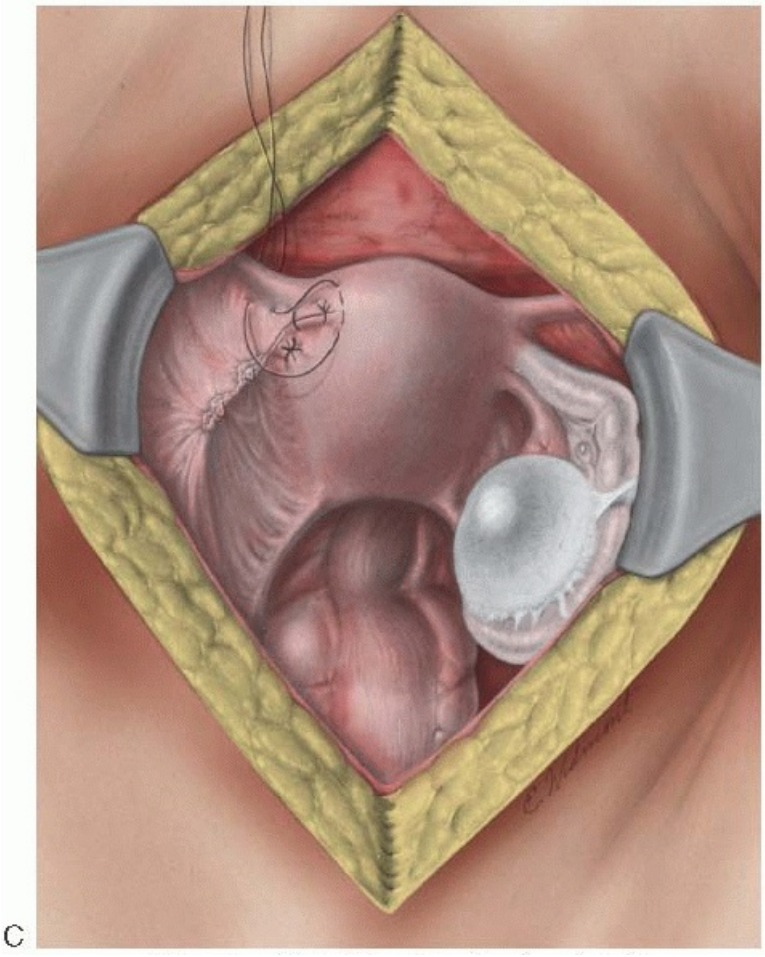
C

FIGURE 30.11 (*Continued*) **C:** The infundibulopelvic ligament and the rest of the broad ligament vessels have been ligated. The cornual wound is covered with the round and the broad ligament using a mattress suture of 2-

0 delayed absorbable material.

Such patients may have a preoperative ureteral catheterization when there is clinical evidence of large, adherent adnexal masses. However, if such an anatomic problem is encountered at the time of laparotomy, an incision can be made in the dome of the bladder that allows the passage of ureteral catheters. If the patient has been positioned in the Allen universal stirrups for operation, intraoperative cystoscopy with passage of ureteral stents is easily accomplished. At the end of the operation, 5 mL of indigo carmine is given intravenously. With a cystoscope in the bladder, the dye can be seen effusing from both ureteral orifices, confirming that the ureters have not been injured or compromised.

PELVIC TUBERCULOSIS

Tuberculosis of the upper genital tract is a rare disease in the United States. However, it is a frequent cause of chronic PID and infertility in other parts of the world. For various reasons, the incidence of tuberculosis is again increasing in the United States. Therefore, cases of tuberculosis-associated PID may also become more evident. It should always be considered in the differential diagnosis of pelvic pain in immigrants, especially those from Asia, the Middle East, and Latin America, and in patients with HIV. Pelvic tuberculosis is produced primarily by either *Mycobacterium tuberculosis* or *Mycobacterium bovis*. The primary site of infection for tuberculosis is usually the lung, with lymphatic spread from the Ghon complex to regional lymph nodes at the hilum usually occurring within 1 to 2 years. More rapid dissemination is due to hematogenous spread, which results in miliary disease often within the 1st year. The fallopian tubes are the predominant site of pelvic tuberculosis, but the bacilli also spread to the endometrium and occasionally the ovaries.

No location in the body is immune to the development of metastatic foci of infection. Tuberculosis of the bone, meninges, kidney, epididymis, fallopian tubes, and other sites can develop. At some sites of miliary spread, the lesions can remain quiescent for long periods before reactivation and further spread of the disease. Direct extension from one organ or system to an adjacent organ or system can also occur. Organs of the female reproductive tract are usually infected by hematogenous miliary spread from a primary pulmonary lesion, by hematogenous spread from a secondary miliary site, by lymphatic spread from a primary pulmonary site to intestinal lymph nodes and then to the pelvis, or by direct extension from adjacent abdominal organs (small intestines, appendix, rectum, bladder) that are the site of tuberculous infection. Fistulas between the intestinal tract and the fallopian tubes have been reported with pelvic tuberculosis.

A venereal transmission of the disease has been reported, with primary genital infection in the woman occurring after coitus with a sexual partner who had tuberculosis of the genitourinary tract. According to Sutherland and MacFarlane, it is not possible to prove conclusively that genitourinary tuberculosis in the man can be transmitted to the woman through sexual intercourse. Because it has been shown that *M. tuberculosis* is present in the sperm of men with urogenital tuberculosis, the possibility of transmission to the pelvic organs of the woman through intercourse must be accepted. Sutherland presents five cases in which sexual transmission of genitourinary tuberculosis from man to woman presumably occurred. However, of 128 husbands of women with genital tuberculosis, only 5 (3.9%) were found to have active genitourinary tuberculosis. When tuberculosis of the vulva, vagina, and cervix is present without evidence of tuberculosis elsewhere in the body, venereal transmission should be suspected.

Pathology of Pelvic Tuberculosis

Both fallopian tubes are involved in almost all patients with pelvic tuberculosis. About one half of patients with tuberculous salpingitis have tuberculous endometritis. Tuberculosis of the cervix is present in 5% of cases. The vagina and vulva are rarely involved. At operation, one may find evidence of generalized tuberculous peritonitis with small, grayish white tubercles covering all peritoneal surfaces of the abdominal and pelvic organs. The

epithelium of the fallopian tubes may not be involved in generalized serosal tuberculous infection. At a later stage of infection, tuberculous salpingitis may grossly resemble other forms of PID involving the adnexa. Unless tubercles are

seen, the diagnosis may not be apparent until microscopic sections are examined by the pathologist. A large pyosalpinx may contain the caseous material of a tuberculous infection but may also contain the purulent exudate of a secondary infection with other common organisms. Tubercles form in the lining of the tube. Some have caseation at the center, with giant cells and epithelioid cells. A proliferation of the mucosal lining of the fallopian tube may resemble a primary tubal carcinoma microscopically and may be confusing to the pathologist.

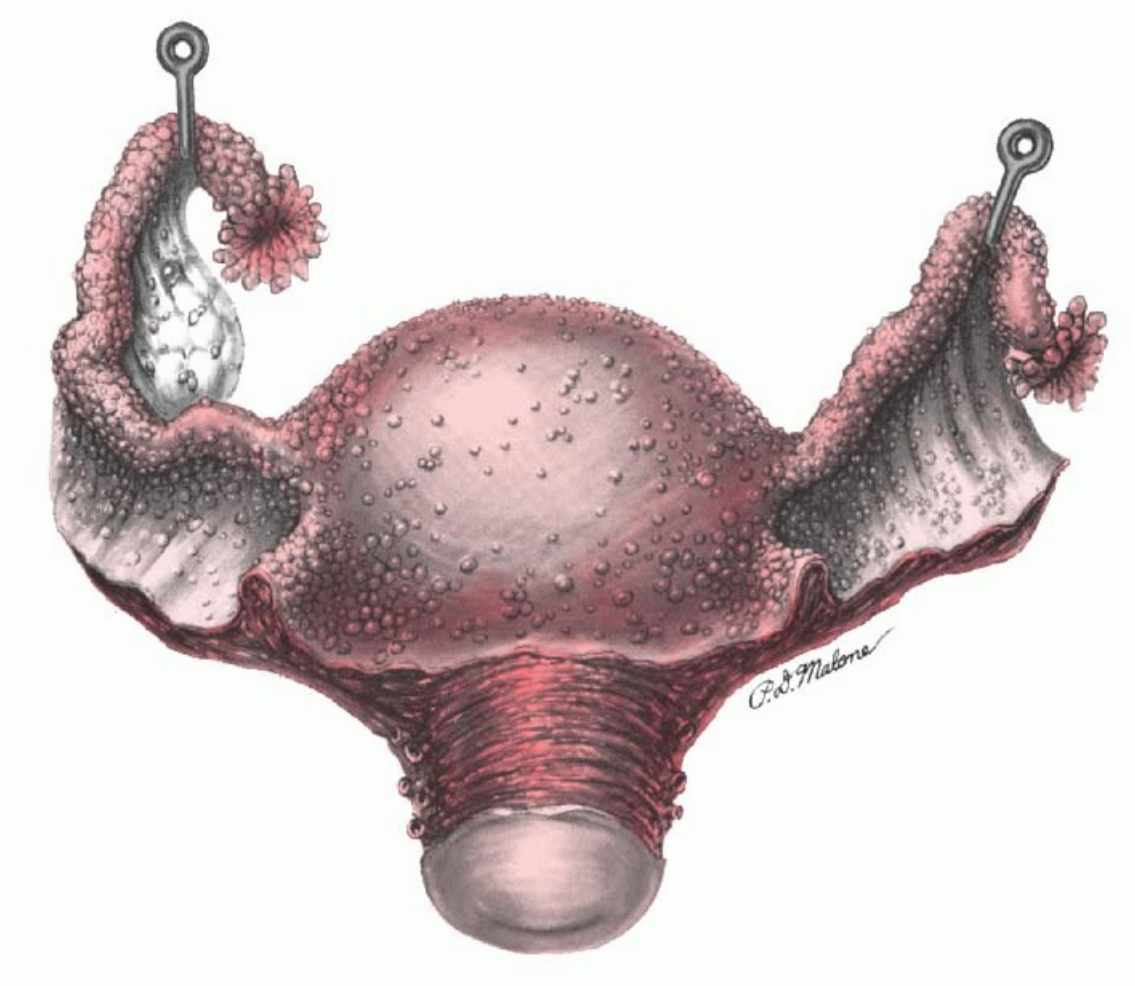

FIGURE 30.12 Typical specimen of tuberculosis of the reproductive organs as part of generalized tuberculous peritonitis.

Tuberculous peritonitis is commonly associated with tuberculosis of the pelvis. Clinically, tuberculous peritonitis can be divided into two groups. In “wet” peritonitis, there is an outpouring of straw-colored fluid into the peritoneal cavity, producing ascites. The peritoneum of the parietal wall and viscera is covered with innumerable small tubercles (**Fig. 30.12**). The tubes, in addition to being covered with miliary tubercles on the serosal surface, are usually slightly enlarged and distended. In contrast to other forms of salpingitis, the fimbriae may be patent. Within the tubal wall and tubal mucosa, the histology is typical of tuberculosis, with tubercle formation, multinucleated giant cells, and epithelioid reaction (**Fig. 30.13**). In advanced cases, frank caseation is present. This pattern is usually associated with hematogenous spread of the tuberculous organism to the peritoneal surfaces and the pelvic organs.

Another type of tuberculous peritonitis encountered in women is the “dry” or adhesive type. Bowel adheres to bowel by innumerable dense adhesions that blend with the musculature. The muscle of the bowel is often

invaded to some degree by the tuberculous process. Separation of these adhesions is extremely difficult surgically, and accidental injury to the bowel is common. The pelvic organs show evidence of tuberculous salpingitis with enlargement of the tubes and occasionally pyosalpinges and even tuboovarian abscess formation.

Tuberculous involvement of the myometrium is rare. Tuberculous endometritis, however, is common, occurring in 60% to 70% of women with pelvic tuberculosis. Microscopically, tubercles are seen scattered throughout the endometrium, but they may be scanty. Tubercles are often seen in the endometrium removed by curettage in the premenstrual phase and are usually located in the endometrium adjacent to the

tubal ostia. Apparently, the uterine cavity is protected from advanced tuberculous infection by the cyclic shedding of endometrial tissue in the reproductive years. Even in advanced pelvic tuberculous infections, evidence of caseation, fibrosis, and calcification are rarely seen in the uterine cavity. Occasionally, the endometrial cavity is obliterated by extensive adhesions. Total destruction of the endometrium can result in amenorrhea. Tuberculous pyometra can also develop, especially in postmenopausal women with an occluded internal cervical os.

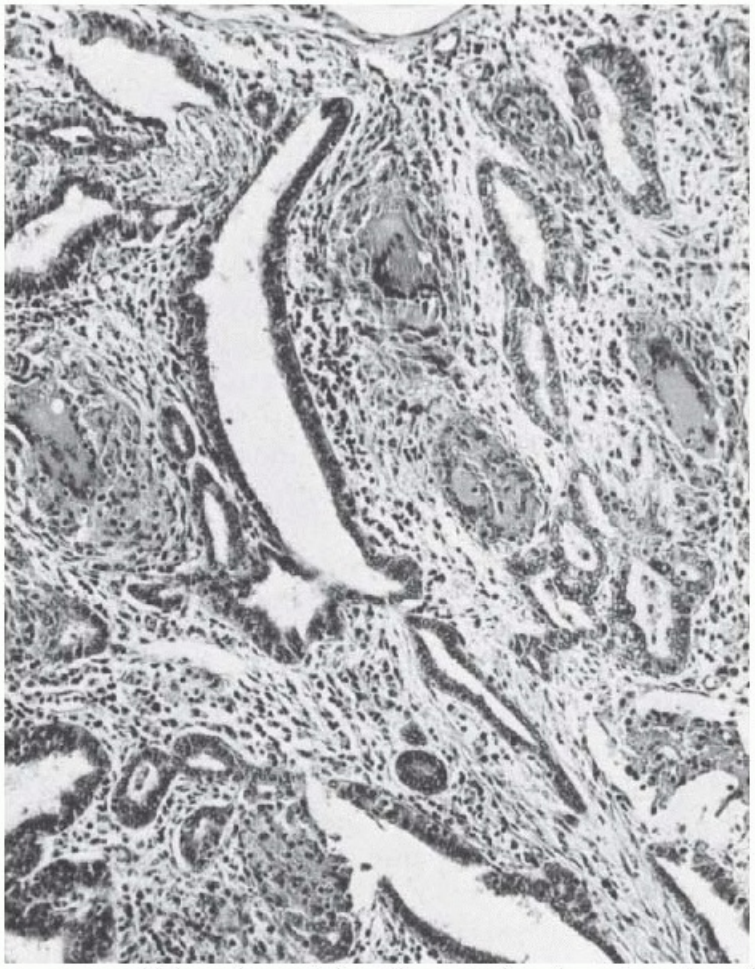

FIGURE 30.13 Tuberculosis of the fallopian tube. Note the multinucleated giant cells.

Tuberculous lesions of the cervix are rare. They can be either ulcerative or exophytic and can resemble a primary cervical malignancy or granuloma inguinale of the cervix. When there is a tuberculous lesion of the cervix, the cervical biopsy often reveals tubercles.

A tuberculous infection of the ovary usually involves only the surface of the ovary and represents simply an extension of infection from the peritoneal cavity and the adjacent fallopian tubes. The infection is usually limited to a perioophoritis. Tuberculous caseation can be found within the ovarian parenchyma, although this is uncommon. Presumably, it occurs as a result of hematogenous spread to the ovarian parenchyma rather than by

direct extension through the ovarian capsule. However, a break in the ovary caused by ovulation may also allow the tubercular bacilli to gain access to the ovarian parenchyma. The ovaries are involved in about 25% of cases of pelvic tuberculosis.

It is uncommon for tuberculosis to involve the vulva and vagina. It is seen in only 2% of patients with pelvic tuberculosis. The gross appearance may be ulcerative with multiple sinuses, it may be hypertrophic with elephantiasis, or it may be similar to that of carcinoma.

Throughout the pelvic organs, the microscopic picture is similar, with tubercles of granulomatous inflammation, Langhans giant cells, epithelioid cells, and central caseation associated with chronic inflammation. With special stains, acid-fast bacilli (AFB) can be demonstrated on careful microscopic examination of the tubercles.

Clinical Features of Pelvic Tuberculosis

Pelvic tuberculosis occurs most often in patients between the ages of 20 and 40 years. The age of patients with gynecologic tuberculosis has changed in recent years; the proportion of patients older than 40 years of age is now much higher than it was in the past. Falk and associates found that the incidence of pelvic tuberculosis in postmenopausal Swedish women is increasing. This was also the opinion of Sutherland, who reported an investigation from Glasgow in which 26 of 701 patients (3.7%) with proven gynecologic tuberculosis were postmenopausal.

The most common clinical symptoms of pelvic tuberculosis include pelvic pain, general malaise, menstrual irregularity, and infertility. Brown and associates found that menstrual irregularity occurred in nearly 50% of patients, whereas amenorrhea or oligomenorrhea was present in 27%. A low-grade fever that on occasion can produce a fulminating septic course is noted in most cases of active or subacute disease. The failure of fever to subside with high doses of broad-spectrum antibiotics is a classic feature of pelvic tuberculosis. A clinical course that is refractory to antibiotic therapy for the usual PID should alert the clinician to the possibility of tuberculosis in a woman who previously resided in an endemic country.

Among patients with pulmonary tuberculosis, the incidence of pelvic tuberculosis generally varies between 10% and 20%. Falk and associates noted that 38% of women with genital tuberculosis had previously had tuberculosis in other organs, usually the lungs. Often, the patient's clinical course is that of a chronic indolent illness.

Diagnosis of Pelvic Tuberculosis

The clinical symptoms and signs of pelvic tuberculosis should direct the clinician to the diagnosis. However, the disease is so uncommon that it is seldom encountered in the gynecologist's usual practice; therefore, the clinical index of suspicion is generally low. In many cases, the clinical presentation is obscure, and the diagnosis is delayed. Howard Kelly once said that when competent gynecologists disagree about the diagnosis of an obscure pelvic condition, it usually is diagnosed as either an "old ectopic pregnancy or pelvic tuberculosis."

More than two thirds of the cases are diagnosed at the time of laparotomy performed for some other indication or at the time of investigation for infertility or abnormal uterine bleeding. The most common symptom is infertility, and the second most common symptom is lower abdominal and pelvic pain. Some patients are completely asymptomatic and are found to have pelvic tuberculosis during examination for other disorders such as infertility. A dilatation and curettage or endometrial biopsy is diagnostic in some cases, especially if performed in the late premenstrual phase of the menstrual cycle. In addition to standard microscopic sections, the specimen can be examined by fluorescent antibody technique. Acid-fast staining of tissue or culture of menstrual blood is effective in detecting the organism in only about 10% of cases, according to Overbeck. The menstrual blood can be collected in a cervical cap, but culture can be repeated many times before a positive result is obtained. Acid-fast stains of tissue suspected of tuberculous infection are important to confirm the diagnosis. Because some AFB

are not tuberculous bacilli, it is important to obtain a positive culture whenever possible. A negative evaluation of the endometrium does not rule out pelvic tuberculosis, because the disease can be present in the fallopian tubes without tuberculous endometritis in 30% to 40% of cases.

On pelvic examination, bilateral adnexal tenderness is the rule. The tenderness is usually less marked than with acute gonococcal or streptococcal infections. Occasionally, a large tuberculous tuboovarian abscess is palpated on pelvic examination and even felt through the abdominal wall. The classic doughy feel of the broad ligament suggests a tuberculous inflammatory disease that is produced by a combination of thickening of the broad ligament, adherent bowel, and some ascitic fluid. On occasion, cul-de-sac nodules representing tubercles on the serosal surfaces of pelvic organs can be felt. The clinical detection of ascites is the strongest evidence obtainable in favor of pelvic tuberculosis. It was present in one fifth of the cases reported by Brown and associates. However, other causes of ascites must be considered, including ovarian carcinoma and cirrhosis of the liver. In differentiating tuberculous salpingitis from gonococcal infections, the finding of a virginal outlet in the presence of obvious tubal inflammation should lend strength to the diagnosis of pelvic tuberculosis.

The diagnosis of tuberculosis cannot be made with certainty from a hysterosalpingogram, but it may be helpful. The radiographic criteria for a suspicion of pelvic tuberculosis by hysterosalpingogram have been described by Klein and associates as follows: calcified lymph nodes or smaller, irregular calcifications in the adnexal areas; obstruction of the fallopian tube in the zone of transition between the isthmus and the ampulla; multiple constrictions along the course of the fallopian tube; endometrial adhesions or deformity or obliteration of the endometrial cavity in the absence of a history of curettage or abortion; and vascular or lymphatic extravasation of contrast material. Although a conclusive diagnosis of pelvic tuberculosis can be made only from a positive culture, these authors conclude that hysterosalpingography

is a useful aid, especially in patients who are asymptomatic except for infertility.

When the diagnosis of pelvic tuberculosis cannot be made in other ways, laparoscopy has been used. Because numerous adhesions may be present, making the introduction of the trocar hazardous, we believe that laparoscopy should be used with particular care. If possible, biopsy specimens of tubal fimbriae or other suspicious areas should be examined histologically or cultured to confirm the diagnosis. In addition to disclosing numerous adhesions, laparoscopy may reveal widespread miliary tubercles involving the omentum and peritoneal surfaces. Matted adnexal masses may be seen. Microscopic examination of peritoneal fluid shows a predominance of lymphocytes.

Vaginal cytology is of limited value in diagnosing tuberculosis. The cytologist must be familiar with the morphology of epithelioid cells in the vaginal smear. Only in cases of tuberculosis of the cervix may cytology be helpful. Patients with pelvic tuberculosis should also have an examination and special diagnostic procedures to rule out tuberculous infections in the upper genital tract. Chest radiograph, tuberculin skin test, pelvic ultrasonography, intravenous pyelogram, and urine, gastric, and sputum cultures for *M. tuberculosis* should be done. In some patients, exploratory laparotomy is needed to make the diagnosis.

Treatment of Pelvic Tuberculosis

Before the advent of antituberculous drug therapy, surgery was often used in the treatment of pelvic tuberculosis. Primary surgical treatment was technically difficult, sometimes ineffective, and associated with a high risk of fistula formation and persistent draining sinuses. With the advent of effective drug therapy, the surgical treatment for genital tuberculosis has been restricted to specific indications. Beginning with streptomycin more than 30 years ago, and later isoniazid and para-aminosalicylic acid, it became evident that many cases of pelvic tuberculosis could be cured or controlled with antituberculous drug therapy. There have been major advances in the antibiotic treatment of this disease, including the use of isoniazid with rifampin, with or without

ethambutol, given sometimes for a period of 2 years or longer. Sutherland analyzed the results obtained with various drug schedules. The drugs that have been used to treat tuberculosis are isoniazid, rifampin, streptomycin, ethambutol, and pyrazinamide. Isoniazid and rifampin are the most effective and have the lowest toxicity. They should be the foundation of most drug regimens. The addition of ethambutol may not be of benefit, at least not in pulmonary tuberculosis. Severe and sometimes fatal hepatitis, which can develop even after months of treatment, has been associated with isoniazid therapy. The risk of developing hepatitis increases with age and with the daily consumption of alcohol. Liver function studies should be done before treatment is started, and patients should be carefully monitored with liver function studies throughout the course of therapy and later. The regimen options and dosage recommendations of the American Thoracic Society, the Infectious Disease Society of America, and the CDC from 2003 for the treatment of tuberculosis can be found at http://www.cdc.gov with initial treatment outlines shown in **Table 30.5**.

TABLE 30.5 Basic Tuberculosis Disease Treatment Regimens

PREFERRED REGIMEN	ALTERNATIVE REGIMEN	ALTERNATIVE REGIMEN
Initial Phase		
Daily INH, RIF, PZA, and EMB[a] for 56 doses (8 wk)	Daily INH, RIF, PZA, and EMB[a] for 14 doses (2 wk), then twice weekly for 12 doses (6 wk)	Thrice-weekly INH, RIF, PZA, and EMB[a] for 24 doses (8 wk)
Continuation Phase		
Daily INH and RIF for 126 doses (18 wk) or Twice-weekly INH and RIF for 36 doses (18 wk)	Twice-weekly INH and RIF for 36 doses (18 wk)	Thrice-weekly INH and RIF for 54 doses (18 wk)

[a]EMB can be discontinued if drug susceptibility studies demonstrate susceptibility to first-line drugs.

Note: A continuation phase of once-weekly INH/rifapentine can be used for HIV-negative patients who do not have cavities on the chest film *and* who have negative AFB smears at the completion of the initial phase of treatment. INH, isoniazid; RIF, rifampin; EMB, ethambutol; PZA, pyrazinamide.

The therapeutic success of modern antituberculous drug treatment regimens in pelvic tuberculosis is difficult to assess in view of the limited number of cases available in the literature. The cure rate varies in the literature from 65% to 95%. Kardos removed the fallopian tubes from 168 patients after medical treatment for 10 months and still found active tuberculosis in 35% of the surgical specimens. The experience of Sutherland suggests, however, that the results of treatment may be improved with newer drugs. The patients under treatment must be

followed up closely for evidence of regression or remission of the pelvic tuberculosis. Only about 50% of patients with genital tuberculosis have the disease in the endometrial cavity; therefore, repeat endometrial biopsies and culture of menstrual egress provide only limited diagnostic information. The progress of the disease can be monitored closely by evaluating the size of adnexal masses with pelvic examinations and ultrasonography as well as tracking the ESR, WBC count, and temperature response. Prolonged follow-up is probably indicated in all cases, because recurrence of the tuberculous pelvic lesion 5 years and even later after the end of drug treatment has occasionally been found.

Surgery in the management of patients with pelvic tuberculosis should be reserved for specific indications, as outlined by Schaefer and by Sutherland. In general, surgery is reserved for

those patients who have failed to respond to an adequate trial of medical therapy. Our indications for the surgical treatment of pelvic tuberculosis include the following:

1. Persistence or enlargement of an adnexal mass after 4 to 6 months of antituberculous antibiotic therapy. The rare possibility of an ovarian tumor must always be considered, even though pelvic tuberculosis is also present. In a 1980 report by Sutherland, the persistence or development of substantial pelvic masses was the indication for surgery in 36 of 91 women with proven tuberculosis of the genital tract treated by surgery. Pelvic ultrasonography should be useful in following the response of adnexal masses to treatment.
2. Persistence of pelvic pain or recurrence of pelvic pain while on medical therapy. In Sutherland's report, 40 of 91 patients were operated on because of pain.
3. Primary unresponsiveness of the tuberculous infection to antibiotic therapy, as shown by persistent spiking temperature, leukocytosis, elevated ESR, and evidence on biopsy specimens of continued endometrial infection. Of the 91 women in Sutherland's report, 10 were operated on because of persistence of endometrial tuberculosis.
4. Difficulty in obtaining patient cooperation for continued long-term therapy. In these cases, we are accustomed to giving a brief course of streptomycin, 0.5 g every 12 hours intramuscularly for 1 week before surgery, to perform definitive surgery, and then to giving 0.5 g every 24 hours in the postoperative period for 2 weeks. A persistent effort should be made to obtain the patient's cooperation for continued antituberculous therapy postoperatively. It is advisable to continue treatment for a year or longer. Isoniazid and rifampin should be used if possible. A common reason for failure of treatment is a tendency for the physician to discontinue drugs after only a few months because the patient appears well.

The preferred surgical treatment includes total abdominal hysterectomy and bilateral salpingo-oophorectomy. The nature of this inflammatory disease may make this operative procedure technically difficult, with an increased risk of injury to bowel and bladder. Consequently, in the event of a frozen pelvis from pelvic tuberculosis, it is occasionally necessary to perform only a subtotal abdominal hysterectomy and adnexectomy.

Adhesions, which are invariably present and usually widespread, may make the dissection more difficult and injury more likely. However, it is usually possible to do this operation without a high incidence of bowel fistulas and other significant complications. Sutherland reported the results of surgery in 77 patients operated on while antituberculous therapy was administered. There were no deaths, no fistulas, and few late complications.

For young patients who are eager to attempt future childbearing, conservative adnexectomy should be carried out only if it is possible to do so after the extent of the adnexal disease is carefully evaluated and is found to be minimal. It is unwise for the surgeon to be committed to a specific operative procedure before the time of surgery, because conservative pelvic surgery for tuberculosis may constitute poor surgical judgment once the operative findings are known. The patient should be forewarned that conservative surgery will be performed only if the

disease is minimal and such surgery is considered medically advisable and is consented to as such.

Conservation of an ovary at the time of operation for pelvic tuberculosis is occasionally possible if the ovary is involved only on its surface. However, if one finds gross evidence of ovarian enlargement or other gross evidence of infection deep in the ovarian parenchyma, the ovary should be removed. Bisection of ovaries to assess the presence of disease deep in the ovarian parenchyma is not advisable.

Reactivation of silent pelvic tuberculosis after tubal reconstructive surgery has been reported by Ballon and associates and by others. We believe that reconstructive tubal surgery has no place in the management of patients whose infertility is the result of bilateral tubal obstruction from tuberculous salpingitis.

Pregnancy after Pelvic Tuberculosis

It is evident from the literature, including the studies of both Schaefer and Sutherland, that only about 5% of patients with genital tuberculosis are capable of becoming pregnant, and only 2% carry a pregnancy to term. It is also evident that in the presence of tuberculous tuboovarian abscesses, pregnancy is extremely rare, and conservative surgery for the purpose of preserving fertility is unwarranted. Only when there is minimal pelvic disease without adnexal masses should conservative surgery be considered.

BEST SURGICAL PRACTICES

- Laparoscopy should be discussed with all patients suspected of PID to confirm the diagnosis and to rule out other surgical emergencies such as appendicitis, ectopic pregnancy, or ruptured abscess. However, stable patients at high risk for complications or with contraindications to laparoscopy can be started on antimicrobial therapy and followed for 24 to 48 hours for a response. With the advent of very effective antimicrobial therapy, strong consideration should be given for surgical exploration if the patient fails initial therapy or symptomatology changes, suggesting an alternative diagnosis. If laparoscopy is not selected as initial management, an endometrial sampling for detection of inflammatory cells and bacterial culture is usually helpful.
- Sonohysterography is contraindicated in patients suspected of having PID. Culdocentesis with the finding of purulent peritoneal fluid may indicate PID but does not rule out appendicitis or diverticulitis.
- Posterior colpotomy requires a midline abscess, an abscess that is adherent to cul-de-sac peritoneum and dissects the rectovaginal septum, and a cystic or fluctuant abscess.
- Hysterectomy is no longer absolutely necessary if salpingo-oophorectomy is needed for treatment of tuboovarian abscesses. Copious irrigation with lactated Ringer is essential if an abscess has ruptured or if pus is present in the abdomen. Antibiotic irrigation has not demonstrated additional benefit or risk. If hysterectomy is necessary after bilateral salpingo-oophorectomy, the vaginal vault should be left open for drainage (with or without a Penrose drain).
- Swan-Ganz catheter placement is helpful in monitoring central venous pressure in patients undergoing surgery for ruptured tuboovarian abscess.

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