a major cause of precipitous delivery in EDs. Preterm infants present more frequently in the breech position. The delivery maneuvers are similar to those described above. Be prepared to initiate neonatal resuscitation. The decision as to whether to initiate resuscitative efforts in the ED is often difficult because patients may deliver an extremely premature fetus of unknown gestational age. **Survival of the newborn increases significantly for each completed week from 21 weeks of gestation (0% survival) to 25 weeks of gestation (75% survival).**[26] When gestational age is known, initiate resuscitation of newborns 22 weeks of gestation or older. It is justified to cease resuscitative efforts after 10 minutes, and certainly, after 15 minutes of asystole.

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REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 102

Vulvovaginitis

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INTRODUCTION

Vaginal discharge is caused by a wide variety of disorders, including vaginitis, cervicitis, and pelvic inflammatory disease.[1] Vaginitis is a spectrum of diseases that cause vulvovaginal symptoms including burning, irritation, itching, odor, and abnormal discharge. The factors associated with acute vaginitis are listed in **Table 102-1**. **The most common infectious causes of vaginitis in symptomatic premenopausal women are bacterial vaginosis (40% to 45%), vulvovaginal candidiasis (20% to 25%), and trichomoniasis (15% to 20%).** Vulvovaginal candidiasis, contact vaginitis, and atrophic vaginitis may occur in virgins and postmenopausal women; however, the other forms of infectious vulvovaginitis are generally found in sexually active women. In approximately 30% of women with vaginal complaints, the disorder remains undiagnosed even after comprehensive testing.[2-4]

The clinical diagnosis may be challenging, because women may have more than one disease, and signs and symptoms are frequently not specific to a particular cause. Polymicrobial infection is not uncommon.

Although infectious vaginitis rarely requires hospitalization, it may have serious sequelae. Both bacterial vaginosis and trichomoniasis have been shown to be associated with premature rupture of membranes, preterm labor, and low infant birth weight.[5,6] Trichomoniasis is associated with pelvic inflammatory disease in patients infected with human immunodeficiency virus and increases risk of human immunodeficiency virus acquisition and transmission.[7,8] When overgrowth of certain bacteria occurs, the protective effect of vaginal lactobacilli strains, which inhibit the growth of bacteria and destroy human immunodeficiency virus in vitro, is lost.[1]

TABLE 102-1 Factors Associated with Acute Vulvovaginitis

Infections
Irritant or allergic contact
Local response to a vaginal foreign body
Lack of estrogen in perimenopausal and postmenopausal women (atrophic vaginitis)
Postirradiation changes

PHYSIOLOGY

In females of childbearing age, estrogen causes the development of a thick vaginal epithelium with a large number of superficial glycogen-containing cells and serves a protective function. Glycogen is used by the normal flora, such as lactobacilli and acidogenic corynebacteria, to form lactic and acetic acids. The resulting acidic environment favors the normal flora and discourages the growth of pathogenic bacteria. Lack of estrogen or a dominance of progesterone results in an atrophic condition, with loss of the protective superficial cells and their contained glycogen, and subsequent loss of the acidic environment.

Normal vaginal secretions vary in consistency from thin and watery to thick, white, and opaque. The quantity may also vary from a scant to a rather copious amount. Secretions are odorless and produce no symptoms. The normal vaginal pH varies between 3.8 and 4.5. Alkaline secretions from the cervix before and during menstruation, as well as alkaline semen, reduce acidity and predispose to infection. Before menarche and after menopause, the vaginal pH varies between 6 and 7. Because of scant nerve endings in the vagina, the patient usually does not have symptoms until both the vagina and vulva are involved in an inflammatory or irritant process.

Vulvovaginal inflammation is the most common gynecologic disorder in prepubertal girls and includes both infectious causes (e.g., bacterial, fungal, pinworm) and noninfectious causes (e.g., contact/irritant, lichen sclerosis, foreign body). Factors thought to contribute to vaginitis in prepubertal females include less protective covering of the vestibule by the labia minora, low estrogen concentration resulting in a thinner epithelium, exposure to chemical irritants such as bubble bath, poor hygiene, front-to-back wiping and the short distance between the vagina and anus, foreign bodies, chronic medical conditions (eczema, seborrhea, and other chronic diseases), and sexual abuse. Infectious causes include respiratory and enteric bacterial organisms such as *Haemophilus influenzae*, *Staphylococcus aureus*, group A streptococci and *Streptococcus pneumoniae*, *Escherichia coli, Shigella flexneri*, *Neisseria gonorrhoeae*, and *Chlamydia*, as well as *Candida* and pinworms. Infectious causes may be more common in adolescents, especially those who are sexually active.[4]

GENERAL APPROACH

Obtain a detailed gynecologic history and perform a pelvic examination. History should include details of vaginal discharge, odor, irritation, itching, burning, bleeding, dysuria, and dyspareunia. Inquire about associated abdominal pain, new sexual partners, use of barrier protection during intercourse, relationship of symptoms to menses, use of antibiotics and contraceptives, and hygiene practices. Note the presence of vulvar edema or erythema, vaginal discharge, cervical inflammation, and abdominal and cervical motion tenderness.

During speculum examination, obtain a swab of the discharge and test for gonorrhea and chlamydial infection. If a patient refuses pelvic examination or it is not feasible, the patient may submit a self-swab of vaginal secretions or a urine sample.[9]

Microscopic examination of secretions and evaluation of pH are useful diagnostic tools. However, microscopes and reagents are not available in all EDs, microscopic examination is time consuming and tedious, and results depend on operator skill. To test pH, obtain a sample from the mid portion of the vaginal sidewall to avoid false elevations in pH caused by mucus. **Sampling from the posterior fornix may yield inaccurate results because cervical mucus, blood, semen, douches, and vaginal medications can elevate the pH.** Microscopic evaluation of fresh vaginal secretions using both normal saline solution and 10% potassium hydroxide slide preparation and fishy odor on whiff test help provide evidence for a diagnosis[10] (**Tables 102-2 and 102-3**). Signs of vulvar inflammation and minimal discharge suggest the possibility of mechanical, chemical, allergic, or other noninfectious causes of vulvovaginitis.

BACTERIAL VAGINOSIS

Bacterial vaginosis is the most common cause of vaginitis and accounts for up to 50% of cases in acutely symptomatic women.

TABLE 102-2 Diagnosis of Vaginitis Based on Vaginal Secretions

Test	Finding	Diagnosis	Comments[13]
pH	4.0–4.5	Normal	—
	4.0–4.5	Candidiasis	If undiagnosed after pelvic examination and evaluation of wet mount, treatment with a single dose of fluconazole is cost effective, but also test for *Neisseria* and *Chlamydia*.
	>4.5	Bacterial vaginosis	If undiagnosed after pelvic examination and evaluation of wet mount, treatment with 2 grams of metronidazole ± a single dose of fluconazole is cost effective, but also test for *Neisseria* and *Chlamydia*.
	>4.5	Trichomoniasis	If undiagnosed after pelvic examination and evaluation of wet mount, treatment with 2 grams of metronidazole ± a single dose of fluconazole is cost effective, but also test for *Neisseria* and *Chlamydia*.
Microscopy of specimen prepared with normal saline solution	Clue cells	Bacterial vaginosis	—
	Motile trichomonads	Trichomoniasis	—
	Pseudohyphae and/or buds	Candidiasis	—
Whiff test of swab specimen prepared with potassium hydroxide	Fishy odor	Bacterial vaginosis	—
Microscopy of specimen prepared with potassium hydroxide	Pseudohyphae and/or buds	Candidiasis	—

However, up to 50% of women who meet clinical criteria for this diagnosis are asymptomatic.

Bacterial vaginosis is a polymicrobial infection that occurs when the normal hydrogen peroxide–producing lactobacilli are replaced by other species including *Gardnerella vaginalis, Ureaplasma, Mycoplasma*, and various anaerobes. Risk factors include multiple sexual partners, intercourse with an uncircumcised male partner, vaginal intercourse immediately after receptive anal intercourse, lack of condom use, douching, and absence of peroxide-producing lactobacilli in the vaginal flora.[1,11] Women who have never been sexually active are less commonly affected. Bacterial vaginosis is not classified as a sexually transmitted infection, but it is generally agreed upon that sexual activity plays a role in transmission and may promote infection.[12]

DIAGNOSIS

The most common clinical presentation of women with bacterial vaginosis is vaginal discharge and odor. Classically, a thin, whitish-gray discharge is present, generally with an increase in discharge volume. The absence of discharge or the presence of only a mild discharge makes the diagnosis less likely. When an odor is present, it may be described as a fishy smell. Introital or vaginal irritation, such as redness, tissue fissures, excoriations, or edema, is not common with bacterial vaginosis.

The diagnosis is based on history, speculum vaginal examination, microscopic evaluation of vaginal secretions, and point-of-care testing. Obtain secretions from the mid sidewall of the vagina, and mix with one to two drops of 0.9% normal saline. Cover with a coverslip for microscopic evaluation for clue cells; to check for fishy or amine odor, add one drop of 10% potassium hydroxide and assess vapors for fishy (amine) smell (see additional methods for amine testing below). To check pH, apply a small amount of secretions directly onto pH paper. The presence of three of the following four criteria makes the diagnosis:

1. A thin, homogeneous vaginal discharge
2. More than 20% clue cells on a wet mount (**Figure 102-1**)
3. Positive results on test for amine release, or whiff test
4. A vaginal pH level >4.5

The criterion with the highest sensitivity (89%) is vaginal pH, whereas that with the highest specificity (93%) is the amine odor, or positive result on whiff test. If vaginal pH is >4.5 and there is an amine odor, the diagnosis of bacterial vaginosis can be made with confidence.[14] A colorimetric card test for bacterial vaginosis detects a vaginal pH of ≥4.7 and volatile vaginal fluid amines. Commercially available tests that might be useful for the diagnosis of bacterial vaginosis include card tests for proline aminopeptidase (Pip Activity TestCard; Quidel, San Diego, CA), a

TABLE 102-3 Vaginitis Signs and Symptoms

Causative Organism	Sign or Symptom
Candida	Thick, curdy discharge Itching
Gardnerella or other bacteria	Fishy odor Whitish-gray, thin discharge
Trichomonas	Frothy odorous discharge Vaginal erythema or edema

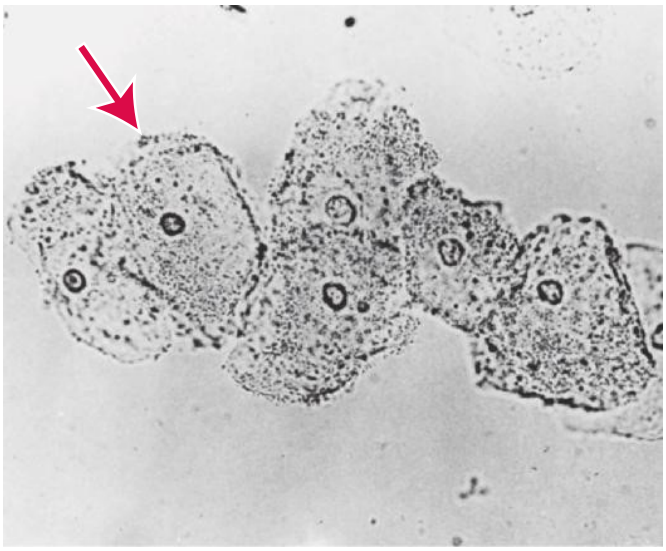

FIGURE 102-1. Bacterial vaginosis. Saline wet mount with clue cells (*arrow*). [Reproduced with permission from DeCherney AH, Nathan L, Laufer N, Roman AS (eds): *Current Diagnosis & Treatment: Obstetrics & Gynecology,* 11th ed. McGraw-Hill, Inc., 2013. Fig. 39-9.]

DNA probe–based test for high concentrations of *G. vaginalis* (Affirm VP III; Becton Dickinson, Sparks, MD), and the OSOM BVBlue test (Sekisui Diagnostics, Lexington, MA), all of which have performance characteristics that are comparable to Gram stain. Cards are available for the detection of elevated pH and trimethylamine; however, they have a low sensitivity and specificity and thus are no longer recommended. Cultures of vaginal discharge are not beneficial, because *Gardnerella* organisms are part of the normal vaginal flora. Polymerase chain reaction for various organisms is being used in research but is not clinically relevant at this time.[1]

The combination of bacterial vaginosis and leukorrhea (more WBCs than epithelial cells seen on a wet mount) is associated with a positive test result for *Chlamydia* (odds ratio = 3.8).[15] For this reason, women who complain of vaginal discharge should be screened for and, depending on clinical suspicion, treated presumptively for *N. gonorrhoeae* and *Chlamydia* infection at the initial visit (Table 102-2). The Centers for Disease Control and Prevention also recommends syphilis testing for women engaged in high-risk sexual behavior, such as those having multiple sexual partners or a new sexual partner or engaging in unprotected intercourse. Finally, women of childbearing age should be screened for pregnancy, because this may impact medical treatment.

TREATMENT

Recommended treatment regimens are listed in **Table 102-4**. The use of *Lactobacillus* intravaginal suppositories and probiotics to restore the normal vaginal flora is an ongoing area of research. Treating male sexual partners is not beneficial for preventing recurrence, but consider treating female partners, particularly with frequent recurrences, because bacterial vaginosis can spread between female partners.[2] Counsel patients receiving metronidazole against consuming alcoholic beverages during the treatment period and for the following 24 hours to avoid a disulfiram-like reaction. Advise patients to refrain from intercourse or to use condoms during treatment.[1]

Overall cure rates 4 weeks after treatment do not differ significantly for a 7-day regimen of oral metronidazole, metronidazole vaginal gel, and clindamycin vaginal cream. Metronidazole vaginal gel has fewer side effects (i.e., GI disturbance and unpleasant taste) but should not be used in women who are allergic to the oral preparation.

Recurrence of symptoms is seen within 3 months in 30% of treated patients who initially show a response. The reasons for this are unclear but could be from sexual transmission.[15] Metronidazole gel 0.75% twice weekly for 4 to 6 months prevents recurrence.[1]

TABLE 102-4 Treatment Regimens for Bacterial Vaginosis

Agent	Dosage
Recommended Regimens	
Metronidazole*	500 milligrams PO twice a day for 7 d
Clindamycin cream 2%	One full applicator intravaginally every night for 7 d
Metronidazole gel 0.75%	One full applicator intravaginally once a day for 5 d
Alternative Regimens	
Clindamycin	300 milligrams PO twice a day for 7 d
Clindamycin ovules	100 milligrams intravaginally every night for 3 d
Tinidazole	2 grams PO daily for 2 d
Tinidazole	1 gram PO daily for 5 d
Regimens for Pregnant Women	
Metronidazole*	250 milligrams PO three times a day for 7 d
Metronidazole*	500 milligrams PO twice a day for 7 d
Clindamycin	300 milligrams PO twice a day for 7 d

*Avoid alcohol during and 24 h after treatment. Metronidazole is a pregnancy category B drug.

BACTERIAL VAGINOSIS COMPLICATIONS

Bacterial vaginosis has been associated with several adverse health outcomes, including facilitation of co-infection with sexually transmitted infections such as human immunodeficiency virus, herpes simplex virus-2, *Chlamydia trachomatis*, and *N. gonorrhoeae* by decreasing local secretory leukocyte protease inhibitor levels.[1,12] Bacterial vaginosis is also linked to complications related to pregnancy and surgical procedures, such as spontaneous abortion, premature rupture of membranes, amniotic fluid infection, chorioamnionitis, preterm delivery, postpartum endometritis, pelvic inflammatory disease, postoperative wound infection, and infection after vaginal and abdominal hysterectomy.[16]

PREGNANT WOMEN

The Centers for Disease Control and Prevention recommends treating all symptomatic pregnant women. Recommended treatment regimens are listed in Table 102-4. The Centers for Disease Control and Prevention no longer recommends routine screening of asymptomatic pregnant women.[1] Pregnant women who are at high risk for preterm labor should be considered for treatment to avoid preterm labor and other adverse outcomes of pregnancy.[5] However, studies have not been able to demonstrate clear benefit in preventing adverse outcomes of pregnancy.[1,17-19] **Topical clindamycin preparations should not be used in the second half of pregnancy** because of an increased association of adverse events, including low infant birth weight and neonatal infections.

CANDIDA VAGINITIS

***Candida* species are the second most common cause of infectious vaginitis.**[20] Prevalence data for vulvovaginal candidiasis vary because the disease is not reportable, many women self-medicate with over-the-counter preparations, and as many as half the women in whom candidiasis is diagnosed also have other conditions.[20] The Centers for Disease Control and Prevention estimates that 75% of women will have at least one episode of vulvovaginal candidiasis in their lifetime.[1]

The organism is isolated in up to 20% of asymptomatic, healthy women of childbearing age, some of whom are celibate. Some women remain entirely asymptomatic despite being heavily colonized with *Candida* species.

Vulvovaginal candidiasis is rare in nonestrogenized premenarchal girls but does occur and is common under 2 years of age. Consider undiagnosed juvenile diabetes or other forms of immunosuppression if *Candida* is diagnosed in a toilet-trained child.[4] Incidence decreases after menopause unless replacement estrogen is being used, which further emphasizes the hormonal dependence of the infection.

Candidiasis can be classified as either an uncomplicated or complicated infection. Uncomplicated infections are sporadic, produce mild to moderate symptoms, are the result of *Candida albicans*, and occur in the nonpregnant, immunocompetent host. Complicated infections are recurrent (four or more infections per year), produce severe symptoms or findings, are the result of suspected or proven non-*albicans* candidiasis, and occur in an abnormal host (women who have uncontrolled diabetes, debilitation, or immunosuppression, or are pregnant). Approximately 10% to 20% of women have complicated disease. Recurrent vulvovaginal candidiasis occurs in <5% of women.[1,21]

C. albicans strains account for 85% to 92% of *Candida* organisms isolated from the vagina. *Candida glabrata* and *Candida tropicalis* are the most common non-*albicans* strains and are often more resistant to conventional therapy.

Candidal organisms gain access to the vaginal lumen and secretions predominantly from the adjacent perianal area. Candidal organisms must first adhere to the vaginal epithelial cells for colonization to take place, and *C. albicans* adheres in greater numbers than other species.

The growth of *Candida* is held in check by the normal vaginal flora, and symptoms of vaginitis usually occur only when the normal balance is upset. Increased colonization by *Candida* resulting in subsequent symptomatic infection may be caused by conditions that (1) inhibit the growth of normal vaginal flora, particularly *Lactobacillus* species (e.g., systemic antibiotics); (2) diminish the glycogen stores in

vaginal epithelial cells (e.g., diabetes mellitus, pregnancy, oral contraceptive use, and hormonal replacement therapy); or (3) increase the pH of vaginal secretions (e.g., menstrual blood or semen). Factors that favor increased rates of vaginal colonization include pregnancy, oral contraceptive use, uncontrolled diabetes mellitus, and frequent visits to sexually transmitted infection clinics (perhaps as a result of antimicrobial therapy). This infection is not considered a sexually transmitted infection, although it can be transmitted by sexual intercourse. The wearing of tight-fitting, particularly synthetic, undergarments may also contribute to the problem because of increased temperature. Although all of these factors are thought to be associated with symptomatic disease, there is poor evidence to prove that any of them is causative.[20] Evidence supporting an association between antibiotic use and vulvovaginal candidiasis is limited. However, antibiotics are thought to increase the risk of vulvovaginal candidiasis by killing endogenous normal flora.

DIAGNOSIS

Clinical symptoms include leukorrhea, severe vaginal pruritus, external dysuria, and dyspareunia. **Vaginal pruritus is the most common and specific symptom.** Complaints of discharge vary from little to copious white vaginal discharge. Symptoms vary in severity, but exacerbation is frequently seen in the week prior to menses or with coitus, perhaps because these factors cause the pH to become more alkaline. Odor is unusual and, if present, favors a diagnosis of bacterial vaginosis rather than candidiasis.

Gynecologic examination may reveal vulvar erythema and edema, vaginal erythema, and discharge. Discharge varies from none to watery to homogeneously thick and "cottage cheese–like." Discharge often adheres to the vaginal walls.

The diagnosis is confirmed with a normal vaginal pH (4.0 to 4.5) and visualization of budding yeast and pseudohyphae on slide preparation of vaginal secretions (**Figure 102-2**). The sensitivity of microscopic examination using a sample prepared with normal saline is only 40% to 60%. Adding two drops of 10% potassium hydroxide to the vaginal secretions dissolves the vaginal epithelial cells while leaving yeast buds and pseudohyphae intact. This increases the sensitivity of microscopic examination to 80% and yields almost 100% specificity. Empiric treatment is suggested for symptomatic patients with negative findings on microscopic examination if *Candida* cultures cannot be obtained.[1]

TREATMENT

Recommended treatment regimens are listed in **Table 102-5**. Therapy regimens are effective in treating over 80% of cases of uncomplicated vaginal candidiasis. Topically applied azole drugs are more effective than nystatin, with relief of symptoms in 80% to 90% of patients who complete treatment. Consider patient preference because creams, lotions, sprays, vaginal tablets, suppositories, and coated tampons are all equally efficacious.[22]

The azole drugs are all available over the counter in treatment regimens of 1, 3, or 7 days. Uncomplicated vulvovaginal candidiasis responds to all azoles, including single-dose therapy.[23] Other than initial burning and irritation, side effects of topical agents are unusual.

Single-dose treatment with oral fluconazole is as effective as topical therapy in the treatment of uncomplicated vulvovaginal candidiasis. Patient preference should be considered, because oral therapy is often more convenient, although insurance and cultural variables can influence preference.[24] Oral treatment may occasionally cause GI symptoms, headache, and rash.[25] Ketoconazole can cause liver toxicity, and therefore, it as been removed from many formularies. The oral azoles can interact with a variety of other medications, including astemizole, calcium channel antagonists, cisapride, warfarin, cyclosporine A, oral hypoglycemic agents, phenytoin, protease inhibitors, tacrolimus, terfenadine, theophylline, trimetrexate, and rifampin.

Sexual partners should not be treated unless the woman has frequent recurrences.

Self-medication is sometimes advised in women with recurrence of previously diagnosed vulvovaginal candidiasis; however, studies demonstrate poor ability to accurately self-diagnose candidiasis even with a

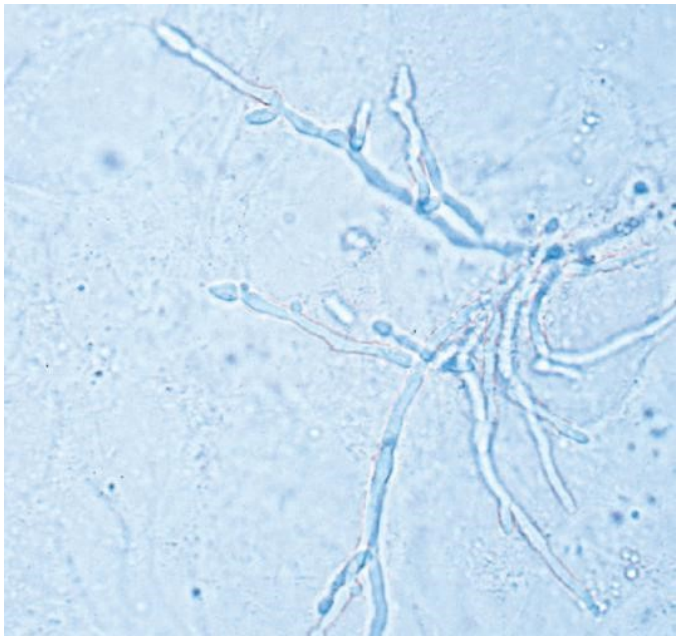

FIGURE 102-2. Hyphae of *Candida albicans,* potassium hydroxide wet mount. [Reproduced with permission from Knoop et al: *The Atlas of Emergency Medicine,* 3rd ed. © 2010 McGraw-Hill Inc. Fig 25-16. Photo contributor: H. Hunter Handsfield: *Atlas of Sexually Transmitted Diseases.* New York, NY: McGraw-Hill; 1992.)

TABLE 102-5 Treatment Regimens for Vulvovaginal Candidiasis*

Agent	Formulation	Dosage
Uncomplicated Vulvovaginal Candidiasis		
Butoconazole†	2% cream	1 applicator intravaginally QHS × 3 d
Clotrimazole	1% cream	1 applicator intravaginally QHS × 7 d
	2% cream	1 applicator intravaginally QHS × 3 d
	100-milligram suppository	1 suppository intravaginally QHS × 7 d
	200-milligram suppository	1 suppository intravaginally QHS × 3 d
	500-milligram suppository	1 suppository intravaginally QHS × 1 dose
Miconazole	2% cream	1 applicator intravaginally QHS × 7 d
	4% cream	1 applicator intravaginally QHS × 3 d
	100-milligram suppository	1 suppository intravaginally QHS × 7 d
	200-milligram suppository	1 suppository intravaginally QHS × 3 d
	1200-milligram suppository	1 suppository intravaginally QHS × 1 dose
Nystatin	100,000-unit vaginal tablet	1 tablet QHS × 14 d
Terconazole	0.4% cream	1 applicator intravaginally QHS × 7 d
	0.8% cream	1 applicator intravaginally QHS × 3 d
	80-milligram suppository	1 suppository intravaginally QHS × 3 d
Tioconazole	6.5% ointment	1 applicator intravaginally QHS × 1 dose
Fluconazole†	150 mg oral tablet	1 tablet PO × 1 dose

*Not all possible regimens listed.

†Not recommended in pregnancy. **Pregnant patient should be treated with topical azoles for 7 days. Oral fluconazole is a Category C medication and thus should be avoided in pregnancy.**

Abbreviation: QHS = every night at bedtime.

prior history of the disease.[5,26] Therefore women who fail to respond to over-the-counter therapy or have recurrence within 2 months should be evaluated by a physician.

The treatment of **complicated vulvovaginal candidiasis** (both severe and recurrent cases) requires longer duration of therapy with topical and oral azoles or alternative therapies. In severe cases, consider treating with a topical azole for 7 to 14 days or treatment with oral fluconazole, 150 milligrams on days 1 and 3 for a total of two doses. In cases of recurrence, consider treating with a topical azole for 7 to 14 days or fluconazole, 100, 150, or 200 milligrams on days 1, 4, and 7 for a total of three doses.[1,25,26]

CANDIDIASIS COMPLICATIONS

Vaginal and microscopic examinations should be performed if symptoms persist or recur within 2 months, and precipitating factors, such as high blood glucose levels, should be controlled. However, most women with recurrences do not have obvious precipitating causes. Vaginal cultures should be obtained to confirm clinical diagnosis but also to indentify any unusual species such as *C. glabrata*. Azoles are not very effective in treating vaginitis caused by *C. glabrata.*

Management of women with frequent recurrence is aimed at control with a long-term suppressive prophylactic regimen, rather than cure. The reason that some women, many of whom have no underlying pathology, experience frequent recurrences of infection with resulting morbidity is not fully understood. Current views suggest that local vaginal immune mechanisms may be responsible for frequent relapses. Maintenance regimens with oral fluconazole (100-, 150-, or 200-milligram doses weekly for 6 months) are the first line of treatment.[1]

TRICHOMONAS VAGINITIS

***Trichomonas vaginalis* is the most common nonviral sexually transmitted infection and accounts for 15% to 20% of cases of acute vaginitis.** There are an estimated 3.7 million Americans infected with *T. vaginalis*, more than *N. gonorrhoeae* and *Chlamydia* combined.[27] Both men and women can be infected, and it is spread through sexual contact. Infection is more common in certain racial and ethnic groups. Incidence is highest among black women at 13.3%, compared with 1.8% of Hispanic women and 1.3% of white women.[28] The prevalence of *T. vaginalis* infection increases with age,[1] unlike other sexually transmitted infections, such as *Chlamydia* and *N. gonorrhoeae*, for which the prevalence is highest among adolescents and young adults. Risk of *T. vaginalis* infection is associated with increasing numbers of sexual partners (recent or remote), early initiation of sexual activity, lower educational levels (high school or below), and poverty.

Trichomoniasis is a parasitic infection with the single-celled protozoan *T. vaginalis*, a flagellated organism (**Figure 102-3**). Infection can produce local inflammation when the organism attaches to the vaginal mucosa. As many as 50% of women are asymptomatic. Clinically symptomatic women with *Trichomonas* vaginitis present with vaginal discharge, pruritus, and irritation. The classic discharge is described as frothy and malodorous. Symptoms generally develop within 5 to 28 days; however, untreated infections can last for months to years and produce symptoms at any time.[29]

DIAGNOSIS

Clinical diagnosis of *Trichomonas* vaginitis traditionally relies on microscopic examination of the vaginal secretions and visualization of motile trichomonads (Figure 102-3). Microscopy should be performed immediately following sample collection or the organism will lose motility. The sensitivity of microscopic identification of trichomonads is 60% to 70%.[1] Although associated with low cost and immediate results, there are several disadvantages to this method, including operator error and overall poor sensitivity. Culture is 95% sensitive and considered the gold standard in diagnosis. However, results may not be available for 2 to 5 days. There are several newer testing options, including two different point-of-care diagnostic tests. Results of the immunochromatographic capillary flow dipstick technology test, OSOM Trichomonas Rapid Test (Genzyme Diagnostics, Cambridge, MA), and the nucleic acid probe test, Affirm VP III (Becton Dickinson, San Jose, CA), are available in 10 minutes and 45 minutes, respectively. Both have a sensitivity of >83% and specificity of >97%.[1,30,31] The U.S. Food and Drug Administration–cleared polymerase chain reaction assay for *N. gonorrhoeae* and *Chlamydia* infection, Amplicor (Roche Diagnostics, Indianapolis, IN), has been modified for *T. vaginalis* detection in both vaginal and endocervical swabs, as well as urine. The sensitivity ranges from 88% to 97%, and the specificity ranges from 98% to 99%. APTIMA *T. vaginalis* Analyte Specific Reagents are available for RNA-mediated amplification testing using the instrumentation platform APTIMA Combo2 (Gen-Probe, Bedford, MA) currently used to diagnose *N. gonorrhoeae* and *Chlamydia* infections. The sensitivity ranges from 74% to 98%, and the specificity ranges from 87% to 98%.[1]

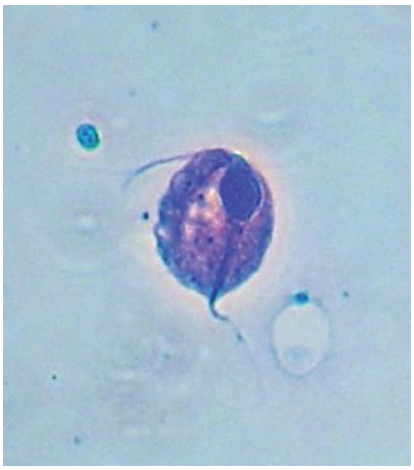

FIGURE 102-3. Trichomonad. [Reprinted with permission of Piotr Rotkiewicz.]

TREATMENT

Treatment regimens for acute *Trichomonas* vaginitis are listed in **Table 102-6**. The nitroimidazoles, metronidazole and tinidazole, are the only medications effective in treating trichomoniasis. Metronidazole gel is considerably less effective (<50%) than oral metronidazole preparations, and so the gel is not recommended. Single-dose treatment is preferable because of lower cost, fewer side effects, and greater patient adherence to the regimen. However, patients whose infection is not responsive to single-dose therapy may require a 7-day course of therapy. There is a 90% cure rate with either the single- or multiple-dose regimen. Cure rates increase to >90% when sexual partners are treated simultaneously. Patients with a true allergy can undergo desensitization in consultation with a specialist.[1]

TABLE 102-6 Treatment Regimens for Trichomoniasis

Agent	Dosage
Recommended Regimen	
Metronidazole*	2 grams PO in a single dose
Tinidazole†	2 grams PO in a single dose
Alternative Regimen	
Metronidazole*	500 milligrams PO twice a day for 7 d

*Metronidazole is a pregnancy Category B drug.

†Tinidazole is a pregnancy Category C drug.

Counsel patients to abstain from sexual intercourse until drug therapy has been completed and the patient and their partner(s) are asymptomatic and to avoid alcohol use during therapy and for 24 hours after completion of drug therapy with metronidazole to avoid a disulfiram-like reaction.

TRICHOMONIASIS COMPLICATIONS

The spread of *Trichomonas* infection is difficult to control, because up to 50% to 75% of those infected are asymptomatic and reinfection is common. Recurrence of disease is frequent and may necessitate multiple courses of treatment.

T. vaginalis infection is associated with several adverse health outcomes, including preterm birth, delivery of low-birth-weight infants, and pelvic inflammatory disease. It has also been associated with increased transmission of several other infections, including human immunodeficiency virus, herpes simplex virus, and human papillomavirus infection. Not only does *Trichomonas* infection increase the likelihood of human immunodeficiency virus acquisition, but it also promotes human immunodeficiency virus transmission and viral shedding.[32,33]

SPECIAL POPULATIONS

Human immunodeficiency virus–positive individuals are more likely to become infected with *T. vaginalis* and have higher complication rates. Consider 7-day therapy for human immunodeficiency virus–positive individuals because studies have indicated that single-dose therapy is less effective.[1]

Due to the potential adverse outcomes, pregnant women should receive treatment with oral metronidazole. Women can be safely treated with single-dose metronidazole therapy at any stage of pregnancy. Tinidazole safety in pregnancy is not well studied. Breastfeeding mothers should withhold nursing during treatment with metronidazole and for 12 to 24 hours after the last dose. If treated with tinidazole, patients should hold breastfeeding for 3 days after the last dose.

CONTACT VULVOVAGINITIS

Contact dermatitis results from the exposure of the vulvar epithelium and vaginal mucosa to a primary chemical irritant or an allergen. Irritant dermatitis is more common than allergic dermatitis.[34] Common irritants and/or allergens include chemically scented douches, soaps, bubble baths, and deodorants; perfumes, dyes, and scents in toilet paper, tampons, pads, and feminine hygiene products; topical vaginal antibiotics; laundry detergents, dryer sheets, and fabric softeners; and tight slacks, pantyhose, and synthetic underwear. Benzocaine, used by women to control vulvar discomfort, can also cause a particularly severe contact dermatitis.

DIAGNOSIS AND TREATMENT

Diagnosis may be difficult due to variation in severity of symptoms and presence of other preexisting conditions. Clinically, patients report local swelling and itching or a burning sensation. Physical findings range from local erythema and edema to excoriation, ulceration, and secondary infection. Local vesiculation and ulceration are more common with allergens or with primary irritants used in strong concentrations. Also consider herpes infections if vesicles are present. Vaginal pH changes may promote colonization and infection with *C. albicans*, which can obscure the primary cause.

Diagnosis of contact vulvovaginitis is made by ruling out an infectious cause and identifying the offending agent. Most cases of mild vulvovaginal contact dermatitis resolve spontaneously when the causative agent is withdrawn. Cool sitz baths and application of wet compresses of dilute boric acid or Burow's solution may afford relief for patients with severe painful reactions. A few days of therapy with topical corticosteroids, such as hydrocortisone acetate (0.5% to 2.5%), fluocinolone acetonide (0.01% to 0.2%), or triamcinolone acetonide (0.025%), applied two or three times daily, provide symptomatic relief and promote healing. Oral antihistamines may be helpful if a true allergic reaction is present. Superinfection with *C. albicans* should be treated as previously described in the section on *Candida* vaginitis.

ATROPHIC VAGINITIS

Vaginal atrophy, present in 60% of women 4 years after menopause, can result in atrophic vaginitis.[35] Decreases in ovarian steroid production that occur in the menopausal woman lead to profound changes in the vulva, vagina, cervix, urethra, and bladder. The changes vary widely from one patient to another. The vagina loses its normal rugae, and the vaginal mucosa becomes attenuated, pale, and almost transparent as a result of decreased vascularity. The squamous epithelium atrophies, the glycogen content of the cells decreases, and the vaginal pH ranges from 5.5 to 7.0. The upper one third of the vagina constricts, and the entire vagina becomes shorter in length and loses its elasticity. The mucosa is only three or four cells thick and is less resistant to minor trauma and infection. The cervix atrophies and retracts and may become flush with the apex of the vault.

DIAGNOSIS AND TREATMENT

Symptoms include vaginal dryness, soreness, itching, dyspareunia, and occasional spotting or discharge. Discharge is thin, scant, and yellowish or pink. The vaginal epithelium appears thin, inflamed, and even ulcerated.

A clinical vaginal infection with copious purulent discharge may develop due to increased vaginal pH, which permits growth of nonacidophilic coliform organisms and the disappearance of *Lactobacillus* species. *Candida* and *Trichomonas* infections are rare in the postmenopausal woman unless estrogenic replacement therapy is used.

Wet preparations demonstrate erythrocytes, increased polymorphonuclear neutrophils, and small, round epithelial cells, which are immature squamous cells that have not been exposed to sufficient estrogen.

Treatment of atrophic vaginitis consists primarily of topical vaginal estrogen. Creams, pessaries, tablets, and the estradiol vaginal ring are all effective in treating the symptoms.[35] Side effects of treatment may include uterine bleeding, breast pain, perineal pain, and endometrial hyperstimulation. Estrogen should not be prescribed to patients with a history of cancer of any of the reproductive organs. Atrophic vaginitis is usually not seen in patients who are already taking systemic estrogen replacement therapy.

Patients should be referred to their own doctors or a clinic for treatment and follow-up to monitor therapy, because all formulations of estrogen, even at low dosages, show systemic absorption and have potentially harmful side effects.[36] In addition, any patient with postmenopausal bleeding, either by history or physical examination, should be referred to a gynecologist to rule out carcinoma.

BARTHOLIN GLAND CYST AND ABSCESS

Bartholin glands are located in the labia minora. The ducts of the glands drain into the posterior vestibule at the 4 o'clock and 8 o'clock positions. Normally the glands are pea sized, but may form a cyst or abscess. The glands begin to function at puberty to provide moisture for the vestibule and involute as women age. Obstruction of the duct may result in a cyst or abscess. A cyst does not need to be present for an abscess to develop. Abscesses may become quite large and cause extreme pain. Bartholin gland abscesses tend to be polymicrobial, although *N. gonorrhoeae* and *C. trachomatis* have been implicated.[37]

DIAGNOSIS AND TREATMENT

Bartholin gland abscess is characterized as a mass in the posterior introitus near the 4 o'clock or 8 o'clock position that has developed over several days. If the abscess was preceded by a cyst, the abscess may develop over a longer period of time. Pain, induration, and fluctuance are usually present. Systemic symptoms such as fever and chills are rarely present.

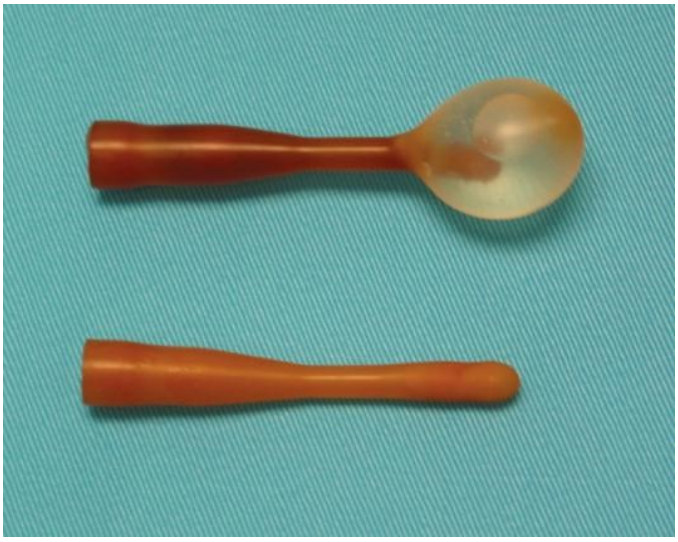

FIGURE 102-4. Word catheter. [Reproduced with permission from Reichman EF: *Emergency Medicine Procedures,* 2nd ed. McGraw-Hill, Inc., 2013. Figure 138-2, p. 932.]

Incision and drainage of an abscess is usually necessary but should not be performed until the abscess is a well-defined, walled-off structure. If the abscess is not ready for incision and drainage, give the patient broad-spectrum antibiotics and analgesics, and advise warm sitz baths. Most patients present with an exquisitely tender, hyperemic, fluctuant mass that needs drainage.

Provide analgesia with a local injection of 2 to 4 mL of 1% lidocaine. To drain the abscess, make a stab incision with a #11 scalpel on the mucosal surface of the vestibule, just lateral to the hymenal ring in the region of the Bartholin gland, where the abscess cavity is closest to the mucosal surface. Extend the stab incision only for a few millimeters—an incision that is too large will result in displacement of the Word catheter. A Word catheter (**Figure 102-4**) is the size of a #10 Foley catheter with a 1-in (2.5-cm) stem and an inflatable balloon. Insert the Word catheter into the incision site, and inflate the balloon with 2 to 4 mL of water. Tuck the end of the catheter into the vagina. The catheter should remain in place for 4 to 6 weeks to avoid recurrence.[38] Case reports describe using plastic tubing (**Figure 102-5**) and pediatric Foley catheters for abscess drainage when a Word catheter is not available.[39,40] Prescribe analgesics and broad-spectrum antibiotics, and give instructions for follow-up care. If *N. gonorrhoeae* and *C. trachomatis* infection are possible, direct antibiotic coverage accordingly (Table 102-2). Patients with recurrent abscess may require definitive surgical care and should be provided with specialty referral.

VAGINAL FOREIGN BODIES

Consider a vaginal foreign body in patients with chronic vaginal discharge. Objects removed in the ED include retained tampons and toilet tissue, items used for sexual stimulation, packets of illegal drugs, and various other items.

Premenarchal children presenting with vaginal discharge, especially if bloody or brown, should be evaluated for a vaginal foreign body, which is found in 4% to 10% of such cases. The discharge associated with a foreign body occurs daily and is often malodorous.[4] Potential foreign bodies include small pieces of toilet paper or cloth and small toys/objects.

Vaginal irrigation with 0.9% normal saline can be attempted to visualize and remove a foreign body in cooperative children >7 years of age. Vaginoscopy under anesthesia in the operating room may be necessary in younger children.

The use of imaging modalities is limited by the composition of the foreign body. Radiolucent foreign bodies are not seen on plain radiographs and may not be detected by pelvic ultrasound. MRI may aid in the localization of nonmetallic objects but is not always available and is not necessarily conclusive.

Treatment of vaginal foreign bodies is removal either manually or by irrigation.

PINWORMS

Patients with pinworms (*Enterobius vermicularis*) complain of anal and/or vaginal pruritus, which is more intense at night (when the gravid female pinworms pass out from the intestinal tract to lay eggs on the

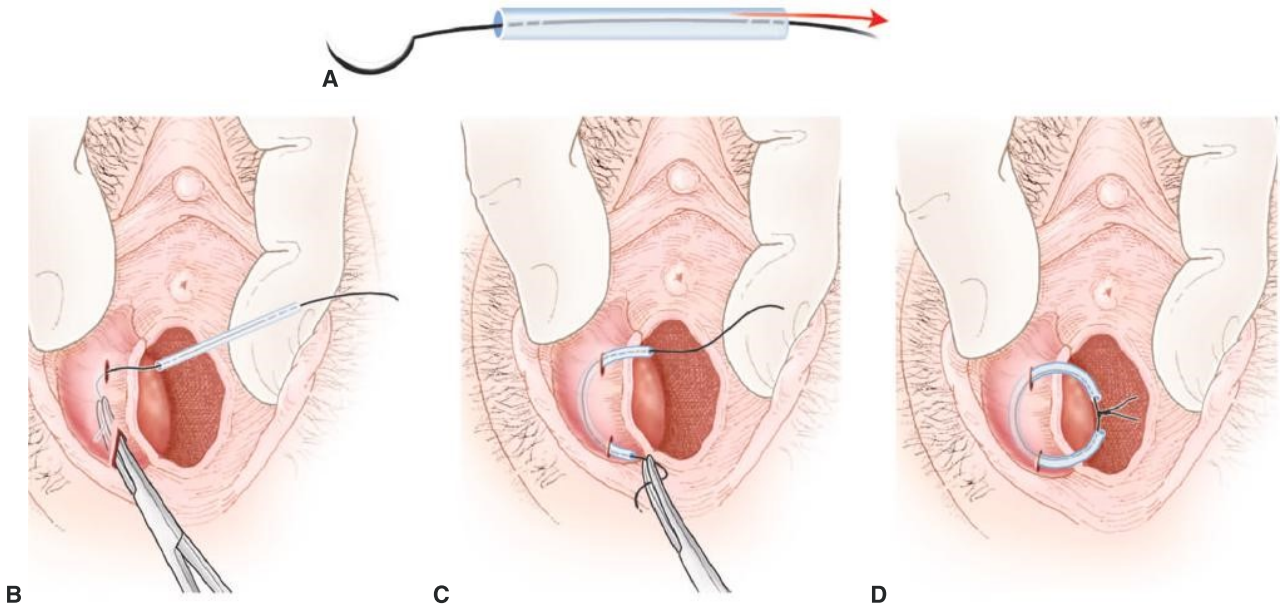

FIGURE 102-5. A. Obtain 7 cm of narrow tubing, and insert silk suture through tubing. B. After drainage, make a second stab incision into the abscess cavity and insert the threaded tubing. C. Use a hemostat to grasp the threaded tubing through both stab sites. D. Suture the threads so they are secure. [Reproduced with permission from Reichman EF: *Emergency Medicine Procedures,* 2nd ed. McGraw-Hill, Inc., 2013. Figure 138-5, Parts A, E, F, G, p. 934.]

TABLE 102-7 Treatment Regimens for Trichomoniasis

Agent and Dosage	Comments
Mebendazole 100 milligrams PO × 1 Repeated in 1 wk	Use with caution in pregnancy
Albendazole 400 milligrams PO × 1 Repeated in 2 wk	Contraindicated in pregnancy
Pyrantel pamoate 11 milligrams/kg PO × 1 (maximum single dose, 1 gram) Repeat dose every 2 wk × 2	Available without prescription

perineal skin). The worms may migrate from the anus to the vagina in children.

The diagnosis is made by visualization of 1-cm-long, thin white worms exiting the anus. Alternately, a sample can be obtained on cellophane tape and used for identification of ova, which are large and double-walled in appearance, on microscopy.

Treat the child and all family members with an antiparasitic agent (**Table 102-7**). Treatments are repeated, because mature worms are more vulnerable to treatment than young worms.[4]

VULVAR TRAUMA

Nonobstetric vulvar trauma is uncommon; however, it is associated with significant physical and psychological consequences. Various types of trauma can be seen including injuries sustained during consensual and nonconsensual sexual activity, accidental injuries including straddle-type injuries, other forms of physical assault, and self-mutilation. Patients may present with abrasions, tears, lacerations, hematomas, burns, and bite wounds. Depending on the situation, gynecologic consultation and examination under anesthesia may be necessary.

Evaluate patients with vulvar trauma for associated vaginal, urethral, anal, and bony pelvis injuries, and treat accordingly.[41]

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REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 103

Pelvic Inflammatory Disease

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INTRODUCTION AND EPIDEMIOLOGY

The term pelvic inflammatory disease (PID) comprises a spectrum of infections of the female upper reproductive tract. It is a common and serious disease initiated by ascending infection from the vagina and cervix. PID includes salpingitis, endometritis, myometritis, parametritis, oophoritis, and tubo-ovarian abscess and may extend to produce periappendicitis, pelvic peritonitis, and perihepatitis (Fitz-Hugh–Curtis syndrome). PID is the most common serious infection in sexually active women age 16 to 25 years.[1]

Long-term sequelae, including tubal factor infertility, implantation failure after in vitro fertilization, ectopic pregnancy, and chronic pain, may ultimately affect 11% of reproductive-aged women.[2] The most common cause of death is rupture of a tubo-ovarian abscess, and the mortality associated with rupture remains at 5% to 10%, even with current treatment methods.

PATHOPHYSIOLOGY

ORGANISMS ASSOCIATED WITH PID

Neisseria gonorrhoeae and *Chlamydia trachomatis* can be isolated in many cases of PID, and therapy is directed primarily against these organisms. However, polymicrobial infection, including infection with anaerobic and aerobic vaginal flora, is evident from cultured material from the upper reproductive tract.[3] **Table 103-1** lists common pathogenic organisms associated with PID. *N. gonorrhoeae* and *C. trachomatis* are often instrumental in initial infection of the upper genital tract, whereas anaerobes, facultative anaerobes, and other bacteria are isolated increasingly as inflammation increases and abscesses form.

Bacterial vaginosis (BV) is frequently identified in women with PID, and the type of BV-associated microorganism (*Gardnerella vaginalis, Mycoplasma hominis, Ureaplasma urealyticum*, pigmented or nonpigmented anaerobic gram-negative rods) may make a difference in the likelihood of developing PID.[4,5]

Infection with ***Trichomonas vaginalis*** is associated with a fourfold increase in the incidence of acute endometritis. Co-infection with **herpes simplex virus 2** and ***C. trachomatis*, *N. gonorrhoeae*,** or bacteria causing vaginosis is also associated with acute endometritis. Infection with herpes simplex virus 2 causes fallopian tube inflammation and lower tract ulceration that may disrupt the endocervical canal mucous barrier.[6] **Human immunodeficiency virus 1 (HIV-1)** infection is associated with an increased incidence of *C. trachomatis* infection, increased incidence of co-infection with *Candida* and human papillomavirus, and increased risk of progression to PID.[7]

PID may result from ***Mycobacterium tuberculosis*** infection in endemic areas.[8] **Schistosomes** can cause genital infection, including a PID-like tubal infection, infertility, and chronic abortion, and a recent report links schistosomiasis to HIV transmission in Africa. ***Actinomyces*** species have been identified almost exclusively in patients with intrauterine devices (IUDs).[9]

ASCENDING INFECTION

Most cases of PID are presumed to originate with sexually transmitted infections (STIs) of the lower genital tract, followed by ascending infection of the upper tract. The original STI may be asymptomatic. The precise mechanisms by which upper genital tract infection and inflammation are initiated and propagated are not well known. Although the cervical mucus serves as a functional barrier to ascending infection much of the time, its efficacy may be decreased by hormonal changes during menstruation and ovulation and by retrograde menstruation. Intercourse may contribute to the ascent of infection due to rhythmic

TABLE 103-1 Organisms Associated with Pelvic Inflammatory Disease

Sexually Transmitted Organisms
Chlamydia trachomatis
Neisseria gonorrhoeae
Herpes simplex virus (types 1 and 2)
Trichomonas vaginalis
Endogenous Genital Tract *Mycoplasma*
Mycoplasma genitalium, Mycoplasma hominis, Ureaplasma urealyticum
Anaerobic Bacteria
Bacteroides species, Peptostreptococcus, Prevotella bivia, Leptotrichia sanguinegens/amnionii, Atopobium vaginae
Aerobic Bacteria
Gardnerella vaginalis, Haemophilus influenzae, Streptococcus agalactiae, Escherichia coli, and other gram-negative rods, *Actinomyces israelii, Campylobacter fetus*

TABLE 103-2 Risk Factors Associated with Pelvic Inflammatory Disease[11-17]

Multiple sexual partners
History of sexually transmitted infection or pelvic inflammatory disease
History of sexual abuse
Frequent vaginal douching
Intrauterine device insertion within previous month
Adolescence, younger adulthood
Lower socioeconomic status
Postabortal

mechanical uterine contractions. Bacteria also may be carried by, or along with, sperm into the uterus and tubes. Uterine infection usually is limited to the endometrium but can be more invasive in a gravid or postpartum uterus. Tubal infection initially affects only the mucosa, but acute, complement-mediated transmural inflammation may develop rapidly and increase in intensity with repeated infection. Inflammation may extend to uninfected parametrial structures, including the appendix and bowel. Infection may spread by direct extension of purulent material from the fallopian tubes or via lymphatic spread beyond the pelvis to involve the hepatic capsule with acute perihepatitis (Fitz-Hugh–Curtis syndrome) and produce acute peritonitis.

RISK FACTORS FOR PID

Multiple risk factors are associated with development of PID (**Table 103-2**).[4,10-17]

IUD use has been associated with an increased risk for PID. Although the majority of risk occurs within 21 days of insertion, the presence of an IUD is associated with complicated PID irrespective of the duration of use.[9,13-15] The risk of PID in IUD users is more related to the development of STI than the IUD,[18,19] and STI screening and treatment at the time of insertion can significantly decrease the likelihood that PID will develop.[20]

Pregnancy decreases the risk of PID because the cervical os is protected by the mucous plug. However, PID can occur during the first trimester and is associated with substantial fetal loss and preterm delivery.

COMPLICATIONS OF PID

PID is associated with a number of serious clinical sequelae. Tuboovarian abscess is reported in up to one third of women hospitalized for PID. Infection and inflammation can lead to scarring and adhesions within tubal lumens. **Ectopic pregnancy is more frequent in women who have had PID than in those who have never had an ectopic pregnancy.** Tubal factor infertility is increased by 12% to 50% in women with a past diagnosis of PID, and the incidence increases with the number and severity of past PID episodes.[21] Asymptomatic or silent PID appears to be associated with tubal factor infertility as well. Sequelae of PID include recurrence of PID, chronic pelvic pain, menstrual disturbances, and chronic dyspareunia. Recurrence of PID may occur because of inadequately treated infection, nontreatment of partner(s), or reinfection from another sexual contact. In follow-up to the Pelvic Inflammatory Disease Evaluation and Clinical Health trial, those with recurrence of PID were five times more likely to experience chronic pelvic pain.[22] PID may also be associated with an increased risk of ovarian borderline tumors.[23]

CLINICAL FEATURES

The clinical presentation of PID is variable. The most common presenting complaint is lower abdominal pain, most frequently described as bilateral and dull or crampy. Pain may be exacerbated by movement or by sexual activity. Other symptoms include abnormal vaginal discharge (75% of individuals), vaginal and postcoital bleeding (more than one third of patients), irritative voiding symptoms, fever, malaise, nausea, and vomiting.[24] Symptoms occur most commonly early in the menstrual cycle or at the end of the menses and are attributed to low progesterone levels and coincident thinning of the cervical mucosal barrier.

The physical examination is usually notable for lower abdominal tenderness, cervical motion tenderness, and uterine or adnexal tenderness. Involuntary guarding and rebound tenderness may be present and may indicate associated peritonitis. The positive predictive value of these findings varies depending on the prevalence of PID in a given clinical population. Adnexal tenderness appears to have a sensitivity of 95%.[25] Mucopurulent cervicitis is a common finding, and its absence should raise consideration of another diagnosis. In women who are suspected of having PID and for whom there is no likely alternative diagnosis for abdominal pain, the presence of fever, adnexal tenderness, and an elevated erythrocyte sedimentation rate are significant independent predictors of endometritis and correctly classify 65% of patients with laparoscopically proven PID (95% confidence interval, 61% to 99%).[25,26]

Right upper quadrant tenderness, particularly with jaundice, may indicate perihepatic inflammation. **Fitz-Hugh–Curtis syndrome** is perihepatitis, demonstrated by right upper quadrant pain in a woman with a clinical diagnosis of PID and no other cause for this pain. It is an uncommon complication and responds to standard antibiotic treatment for PID.[27]

The differential diagnosis of PID is broad and includes cervicitis, ectopic pregnancy, endometriosis, ovarian cyst, ovarian torsion, spontaneous abortion, septic abortion, cholecystitis, gastroenteritis, appendicitis, diverticulitis, pyelonephritis, and renal colic. Look for signs of other STIs, such as herpes simplex, syphilis, and human papillomavirus infection.

DIAGNOSIS

The diagnosis is based on history and clinical findings. No single piece of historical, physical, or laboratory information is sensitive and specific for the disease. **Laboratory evaluation of any woman of childbearing age in the ED always should include a pregnancy test.** Consider the possibility of ectopic pregnancy or septic abortion; the most common alternative diagnosis in missed ectopic pregnancy is PID. Concurrent pregnancy also influences patient treatment and disposition.

Current Centers for Disease Control and Prevention guidelines encourage initiation of empiric treatment in women at risk for PID who exhibit lower abdominal pain, adnexal tenderness, and cervical motion tenderness. Guidelines stratify diagnostic criteria into the three groups shown in **Table 103-3**.

TABLE 103-3 Diagnostic Criteria for Pelvic Inflammatory Disease (PID)

Group 1: Minimum criteria. Empiric treatment if no other cause to explain findings.
Uterine or adnexal tenderness
Cervical motion tenderness
Group 2: Additional criteria improving diagnostic specificity.
Oral temperature >101°F (38.3°C)
Abnormal cervical or vaginal mucopurulent secretions
Elevated erythrocyte sedimentation rate
Elevated C-reactive protein level
Laboratory evidence of cervical infection with *Neisseria gonorrhoeae* or *Chlamydia trachomatis* (i.e., culture or DNA probe techniques)
Group 3: Specific criteria for PID based on procedures that may be appropriate for some patients.
Laparoscopic confirmation
Transvaginal US (or MRI) showing thickened, fluid-filled tubes with or without free pelvic fluid or tubo-ovarian complex
Endometrial biopsy results showing endometritis

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep* 59(RR-12): 12, 2010.

LABORATORY TESTING

Obtain saline- and potassium hydroxide–treated wet preparations of vaginal secretions to identify leukorrhea (more than one polymorphonuclear leukocyte per epithelial cell) and trichomonads, and to test for BV, including clue cells, pH, and a whiff test. Leukorrhea is sensitive but not specific for upper tract infection,[28] and the absence of leukorrhea is a negative predictor for PID. Although endocervical swab specimens may be sent for culture and can be gram stained for gonococci, nucleic acid amplification tests and DNA probes for *N. gonorrhoeae* and *Chlamydia* have replaced culture and gram staining in many settings. Unfortunately, these results are not available to the ED at the time of initial evaluation. Several sensitive and specific diagnostic tests are currently available for *Trichomonas* testing, including a nucleic acid amplification test (Aptima□; GenProbe, San Diego, CA), approved in 2013, that is performed on the same clinical samples as those for *Chlamydia* and gonorrhea testing.[29,30]

If PID is clinically suspected, an elevated WBC count, erythrocyte sedimentation rate, or C-reactive protein level supports the diagnosis.[28] Because a patient may have multiple STIs, also obtain a rapid plasma regain test for syphilis. Test for HIV and hepatitis. Urinalysis can exclude urinary tract infection, but a positive urinalysis does not exclude PID, because any inflammatory process in the contiguous pelvis can produce WBCs in the urine. Blood cultures do not aid in diagnosis or treatment.

IMAGING

Transvaginal pelvic US may demonstrate thickened (>5 mm), fluid-filled fallopian tubes or free pelvic fluid in acute severe PID. Pelvic or tubo-ovarian abscesses appear as complex adnexal masses with multiple internal echoes. Pelvic US can demonstrate as many as 70% of adnexal masses missed on physical examination. US also may be helpful in ruling in or out other causes in the differential diagnosis of pelvic pain, including ectopic pregnancy, ovarian torsion, hemorrhagic ovarian cyst, and possibly appendicitis or endometriosis.[31]

Abdominopelvic CT and MRI may also be used in the diagnosis of PID and the exclusion of other important causes of pelvic pain. If appendicitis or other surgical or GI diagnoses cannot be excluded, obtain an abdominopelvic CT. For further discussion, see chapter 97, "Abdominal and Pelvic Pain in the Nonpregnant Female." CT findings in PID include obscuration of the pelvic fascial planes, cervicitis, oophoritis, salpingitis, thickening of the uterosacral ligaments, and the presence of simple or complex pelvic fluid or abscess collections. MRI is especially helpful in characterizing complicated soft tissue masses, including dilated fallopian tubes and abscesses. MRI imaging is more specific and accurate than US to assess PID, with a sensitivity of 95% and a specificity of 89%.[32,33]

TREATMENT

Treatment is aimed at relieving acute symptoms, eradicating current infection, and minimizing the risk of long-term sequelae. From a public health perspective, another objective of treatment is to reduce the risk of transmission of infection to other new partners and to identify and treat past and current sexual partners to prevent disease spread. Early diagnosis and treatment are critical because duration of symptoms is an independent risk factor for infertility.

Due to the difficulty of diagnosis and the potential for serious sequelae, the Centers for Disease Control and Prevention recommend a low threshold for empiric treatment, with overtreatment preferred to a missed diagnosis with resultant delayed or no treatment.

Provide adequate analgesia, control of emesis and fever, and fluid replacement in those with nausea, vomiting, and dehydration and in those who appear toxic. Nonsteroidal anti-inflammatory drugs are very useful for the management of pain of pelvic origin. ED treatment should include empiric broad-spectrum antibiotic therapy to cover the full range of likely organisms. Screen for BV and treat when screening is positive. Treatment regimens should follow both national guidelines from the Centers for Disease Control and Prevention and local health department surveillance reports.

The Pelvic Inflammatory Disease Evaluation and Clinical Health trial, which included 654 females age 14 to 37 years old and excluded those who had been treated with antibiotics during the preceding 7 days, had experienced an abortion, delivery, or gynecologic surgery during the preceding 14 days, were homeless, or had an allergy to study medications, demonstrated no differences between oral and parenteral regimens in women with mild to moderately severe acute PID uncomplicated by pregnancy or the presence of a tubo-ovarian abscess.[34,35]

Currently accepted inpatient and outpatient treatment regimens are summarized in **Tables 103-4 and 103-5**. Current geographic patterns of drug resistance may change recommendations. Patients with PID who require IV antibiotics initially can be switched to oral antibiotics after clinical improvement.

TABLE 103□4 Parenteral Treatment Regimens for Pelvic Inflammatory Disease

Cefotetan, 2 grams IV every 12 h, or cefoxitin, 2 grams IV every 6 h
plus
Doxycycline, 100 milligrams PO or IV every 12 h*
or
Clindamycin, 900 milligrams IV every 8 h
plus
Gentamicin, 2 milligrams/kg IV or IM loading dose, followed by gentamicin, 1.5 milligrams/kg every 8 h maintenance dose†
Alternative Parenteral Regimen (limited data on effectiveness)
Ampicillin/sulbactam, 3 grams IV every 6 h
plus
Doxycycline, 100 milligrams PO or IV every 12 h*

*PO doxycycline has the same bioavailability as IV doxycycline and avoids painful infusion.

†Gentamicin dosing may be 3–5 milligrams/kg every 24 h.

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep 59*(RR-12): 12, 2010.

TABLE 103□5 Oral and Outpatient Treatment Regimens for Pelvic Inflammatory Disease

Ceftriaxone, 250 milligrams IM once, *or* cefoxitin, 2 grams IM once, *and* probenecid, 1 gram PO once administered concurrently
or
Other parenteral third-generation cephalosporin (e.g., ceftizoxime or cefotaxime)
plus
Doxycycline, 100 milligrams PO twice a day for 14 d
with or without
Metronidazole, 500 milligrams PO twice a day for 14 d
If parenteral cephalosporin therapy is not feasible and community prevalence of fluoroquinolone resistance is low:
Levofloxacin, 500 milligrams PO, *or* ofloxacin, 400 milligrams twice daily every day for 14 d
with or without
Metronidazole, 500 milligrams PO twice a day for 14 d

Note: Other parenteral third-generation cephalosporins can be substituted for ceftriaxone or cefoxitin. Since the Centers for Disease Control and Prevention guidelines were published in 2006, clinically significant resistance to the fluoroquinolones (6.7% of infections in heterosexual men, an 11-fold increase from 0.6% in 2001) has emerged in the United States. Fluoroquinolone antibiotics are no longer recommended to treat gonorrhea in the United States.[27] Fluoroquinolones may be an alternative treatment option for disseminated gonococcal infection if antimicrobial susceptibility can be documented.

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. *MMWR Recomm Rep* 59(RR-12): 12, 2010.

TREATMENT WITH IUD IN PLACE

In the past, IUDs were generally removed, based on the belief that because it is a foreign body, removal of the IUD would allow treatment to be more effective. There is a low risk of PID from IUD insertion, especially when STI testing is done concomitantly and immediate treatment is initiated.[20] **Current Centers for Disease Control and Prevention guidelines suggest that there is insufficient evidence to recommend IUD removal before treatment for PID, because the device is usually not the source of infection.** For individuals using IUD for birth control who develop PID, there are no data to support the use of one treatment regimen over another. Close clinical follow-up is prudent. If there is a concern regarding PID in a patient with an IUD placed in the last 3 weeks, it is reasonable to consult a gynecologist regarding removal.

TREATMENT IN HIV INFECTION

Microbiologically, HIV-positive women are more likely to have concomitant *Candida*, *Mycoplasma hominis*, HPV, and streptococcal infection. HIV-positive women with PID may experience more severe symptoms irrespective of CD4 count and are more likely to have sonographically diagnosed tubo-ovarian abcess. However, they appear to respond similarly to treatment for uncomplicated PID as do women who are not infected with HIV.[36-38] HIV-positive status alone is not a criterion for hospitalization.[36,39] Although HIV status has been removed from specific admission considerations, the 2010 Centers for Disease Control and Prevention STI guidelines note that "whether the management of immunodeficient HIV-infected women with PID requires more aggressive interventions (e.g. hospitalization or parenteral antimicrobial regimens) has not been determined."[40]

TREATMENT OF ADOLESCENTS

Several studies have raised additional concerns about the outpatient management of early adolescents. Early and mid-adolescents were not well represented in the Pelvic Inflammatory Disease Evaluation and Clinical Health study, and of those enrolled, adolescents had increased risk of recurrent PID and a shorter time to pregnancy after an acute episode compared with adult enrollees. Adolescents hospitalized in pediatric centers often receive services beyond IV antibiotics, including education on risk reduction, emotional support, social work intervention, assistance with communicating the nature of their illness with parents, and assistance to arrange close follow-up.[41,42]

ALTERNATIVE ANTIBIOTICS

For those with severe cephalosporin allergy, **spectinomycin** is recommended in Canada and Europe but is not currently available in the United States. For more information, see the Centers for Disease Control and Prevention Web pages on antibiotic-resistant gonorrhea at http://www.cdc.gov/std/Gonorrhea/arg/default.htm.[28]

Multiple studies have demonstrated poor compliance with doxycycline therapy (25%; 50% with partial compliance), and 20% to 25% of patients never fill their prescriptions.[43-45] Recently, generic doxycycline has been difficult to find due to manufacturing shortages and, when available, may be quite expensive, resulting in patient noncompliance with treatment. For PID treatment, **azithromycin** is an alternative, with dosing as either 250 milligrams PO once a day for 7 days or 1 gram once a week for 2 weeks.[43] The long half-life of azithromycin requires significantly fewer doses, which is thought to improve the likelihood of patient compliance. Azithromycin also provides intrinsic anti-inflammatory effects and may reduce local tissue damage. Weigh these potential benefits against the lack of large-scale or long-term studies comparing the effectiveness of azithromycin to doxycycline in the treatment of PID and the possibility of emerging resistance to azithromycin.[43,46-49]

TUBO-OVARIAN ABSCESS

Disproportionate unilateral adnexal tenderness or adnexal mass or fullness may indicate a tubo-ovarian abscess. In women with clinical toxicity and asymmetric pelvic findings, obtain a pelvic US. Most tubo-ovarian abscesses (60% to 80%) resolve with antibiotic administration alone.[50-52] In the setting of tubo-ovarian abscess, oral therapy should be continued with clindamycin (450 milligrams PO four times per day) or metronidazole with doxycycline for better anaerobe coverage for 14 days. Patients who do not improve after 72 hours of treatment should be reevaluated for possible CT- or US-guided percutaneous drainage, laparoscopic drainage, posterior colpotomy with drainage, surgical intervention, or reconsideration of other possible diagnoses. Abscesses 9 cm or larger on imaging appear to have a higher likelihood of requiring surgical therapy. An enlarging pelvic mass may indicate bleeding secondary to vessel erosion or a ruptured abscess.

DISPOSITION AND FOLLOW UP

Guidelines for admission (**Table 103-6**) and inpatient treatment (Table 103-4) have evolved over the past decade. There are no data demonstrating that inpatient treatment is more effective than outpatient treatment. Among the problems encountered with outpatient care are the provision of adequate guideline-driven treatment, patient adherence to the prescribed therapeutic regimen, difficulty in arranging outpatient administration of parenteral medications, and coordination of 72-hour follow-up evaluation, all of which have been implicated as causes of treatment failure. Consider these and other constraints when determining the patient's ability to follow or tolerate an outpatient regimen.

Institutions should consider adoption of protocolized treatment guidelines to help to ensure fidelity to standards of care. **Admission decisions in the ED are based on severity of illness, likelihood of adherence to outpatient medication regimen, likelihood of major anaerobic infection (IUD, suspected pelvic or tubo-ovarian abscess, or history of recent uterine instrumentation), certainty of diagnosis, coexisting illness and immunosuppression, pregnancy, patient age, and other major fertility issues.**

If the patient is discharged, arrange reevaluation within 72 hours for clinical improvement and adherence to the prescribed regimen. Encourage partner evaluation and treatment. Test and treat for other STIs if not already done. Educate patients about the use of barrier contraceptives and other "safe sex" techniques to lessen the risk of reinfection. Counsel the patient to remain abstinent from sexual activity until 1 week after treatment is finished for both the patient and partner and symptoms have abated.

Partner treatment is crucial to preventing repeated episodes of PID. This can be difficult to ensure. If the current partner has accompanied the patient to the ED, and the patient is willing to tell this partner about her infection, she can be asked to suggest immediate ED evaluation to her partner. If not, the patient should be instructed to notify partners with whom she has had sexual contact in the 60 days preceding the onset of her symptoms to go to the local public health department or STI clinic for empiric treatment of *N. gonorrhoeae* and *C. trachomatis*. A 6-minute PID outreach video has been developed and was found in one randomized controlled trial to improve partner treatment.[53]

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TABLE 103-6 Admission Considerations

Inability to exclude surgical emergency from the differential diagnosis
Pregnancy
Failure to respond to outpatient treatment
Inability to tolerate or comply with outpatient treatment
Severe toxicity, high fever, nausea, vomiting
Tubo-ovarian abscess

Source: Reproduced with permission from Centers for Disease Control and Prevention, Workowski KA, Berman SM: Sexually transmitted diseases treatment guidelines, 2010. MMWR Recomm Rep 59(RR-12): 12, 2010.