Chapter 5
Vaginitis and Cervicitis

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Case Study

Anna is a 19-year-old female college student who presents with thin, white vaginal discharge with foul odor which started 1 week ago. She also endorses mild itching in her genital region which is quite bothersome. She is otherwise healthy and takes combined oral contraceptive pills and a multivitamin daily. She is sexually active with two male partners and occasionally uses condoms. Anna smokes approximately one fourth of a pack of cigarettes per day. She reports that she tried drinking cranberry juice for her symptoms without any improvement. Examination reveals non-edematous vaginal walls coated with thin, gray secretions with a "fishy" odor. Wet mount in the clinic demonstrates vaginal epithelial cells covered with bacteria. The pH of vaginal secretions is measured at 5.3. A pregnancy test is negative, and a cervical swab specimen is sent for gonorrhea and chlamydia PCR.

Case Questions

1. What clinical syndrome does the patient exhibit?
2. What diagnostic tests would be helpful in identifying the etiology of the patient's symptoms?
3. What counseling should be given to the patient to reduce her risk of this disease recurring?

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Introduction

Symptomatic vaginal discharge is a common reason for women to seek medical care [1]. Vaginal discharge may be normal or pathologic, and self-diagnosis and self-treatment frequently occur [2]. Vaginitis is usually characterized by vaginal discharge, vulvar itching, burning, irritation, and odor and may have either infectious or non-infectious etiologies [3]. Common infectious etiologies include bacterial vaginosis (BV), vaginal candidiasis, and trichomoniasis. Non-infectious etiologies of vaginitis include dermatologic etiologies of vaginitis, such as allergic vaginitis and vulvar vestibulitis. The presence of purulent vaginal discharge raises concern for cervicitis, which also can be infectious or non-infectious in etiology [2]. Infectious mucopurulent cervicitis can be caused by *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, herpes simplex (particularly primary HSV-2 infection), *Mycoplasma genitalium*, *Trichomonas vaginalis*, and *Candida albicans* [4].

Normal and Abnormal Physical Exam

Treatment of vaginal complaints based solely on patient symptomatology alone has not been shown to accurately correlate with final diagnosis [1]. Therefore, detailed history taking, physical exam (including speculum pelvic examination) and other diagnostic tests are essential in determining the causes of vaginitis [5, 6]. Medical history of the patient should include their age, other medical conditions and medications (including those that may cause immune suppression), menstrual history, prior pregnancies and current pregnancy status, sexual preference and practices, use of intravaginal products, past and current sexual relationships, condom use, and prior genitourinary infections. Regarding the patient's symptom history, key features to elucidate include timing of symptom onset, quality and quantity of discharge, presence of irritation or burning, odor of vaginal discharge, and presence of abdominal pain or discomfort and any constitutional symptoms.

Vaginal discharge may be physiologic or pathologic, and differentiation between the two can often be ascertained based on history alone. Vaginal secretions are a physiologically important aspect of a healthy female genitourinary tract. Glycogen-containing vaginal cells depolymerize after they are shed into the lumen of the vagina and serve as an energy source for a group of bacteria that live in the healthy vagina known as lactobacilli [2]. The vaginal microbiome, or the community of bacteria that live in the vagina, is dominated by species of *Lactobacillus* which has long been thought to be protective in the vaginal ecosystem. *Lactobacillus* species produce lactic acid that keeps the vaginal pH to a protective level of less than 4.5. They also serve as a barrier to infection through the production of bacteriocins which prevent the overgrowth of unhealthy vaginal bacteria. Normal vaginal discharge is a combination of vaginal epithelial cells and *Lactobacillus* species bacteria in fluid that appears clear to white in color and is highly viscous and odorless [2, 3]. The volume of vaginal secretions may vary between women and is often influenced by the phase of the menstrual cycle, pregnancy, and the use of hormonal contraceptives [2]. Cervical mucous also increases in the pre-ovulatory phase of the menstrual cycle and peaks at mid-cycle [2, 7, 8].

A general physical examination should be performed for all patients undergoing evaluation for vaginitis. Suprapubic and abdominal examination should be undertaken to assess for tenderness, masses, and inguinal adenopathy [2]. Gynecologic examination requires careful inspection of the external and internal genitalia for lesions or ulcerations. The appearance and integrity of vulvar and perineal skin may reveal clues to the underlying diagnosis. Speculum examination involves inspection of the vagina and cervix for lesions or ulcerations as well as collection of samples of vaginal secretions for vaginal wet mount examination and other laboratory testing [2]. Vaginal and cervical mucosal inspection should include attention to erythema, friability, and lesions in addition to notation of an ectropion, if present [2] (Fig. 5.1). Cervical ectropion occurs when the endocervix exposes columnar epithelium to the vaginal environment due to eversion; ectropion is a normal finding and common in adolescents, pregnant women, and those taking estrogen-containing contraceptives [9]. Bimanual examination is necessary for evaluation of cervical motion tenderness, adnexal tenderness, and uterine or adnexal masses [2].

A vaginal wet mount can be prepared by mixing the vaginal fluid with 0.5 mL of normal saline to form a suspension. This fluid suspension undergoes the "whiff test" for odor before being placed on a microscopic slide to form a wet mount [2]. If available, a pH meter or colometric strips can be utilized to determine the pH of the sample or can be tested directly on vaginal wall. The wet mount should be examined under high-power microscopy for both number of epithelial cells and presence of polymorphonuclear neutrophils (PMNs). Increased number of PMNs can be suggestive of vaginal or cervical inflammation. Importantly, identification of parasitic, bacterial, and fungal organisms can aid in the diagnosis of vaginitis [2] (Fig. 5.2). Application of a drop of 10% potassium hydroxide (KOH) on the slide can enhance a "fishy" odor if present and highlight the presence of yeast with pseudohyphae [10]. While the vaginal fluid wet mount is an essential test due to its point-of-care availability, it has low sensitivity for diagnosis of many vaginal infections [10], and laboratory-based diagnostic testing can additionally support the clinical diagnosis of vaginitis and cervicitis and identify the causative pathogen [11]. Gram stain, culture, nucleic acid techniques, and PCR testing provide alternative methods of testing, though in some settings, the costs and availability of these methods may preclude clinical utility [10]. Nuclei acid amplification tests (NAAT) of vaginal fluid may be particularly useful for diagnosing sexually transmitted infections (STIs) such as *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and *Trichomonas vaginalis* [2].

Bacterial Vaginosis

Epidemiology

Bacterial vaginosis (BV) is the most common cause of vaginitis [3], affecting up to 30% of women in the United States [12]. Bacterial vaginosis occurs in reproductive age women worldwide [13], and its prevalence can vary within a population [3]. While it is not considered a sexually transmitted disease, there are higher rates of BV

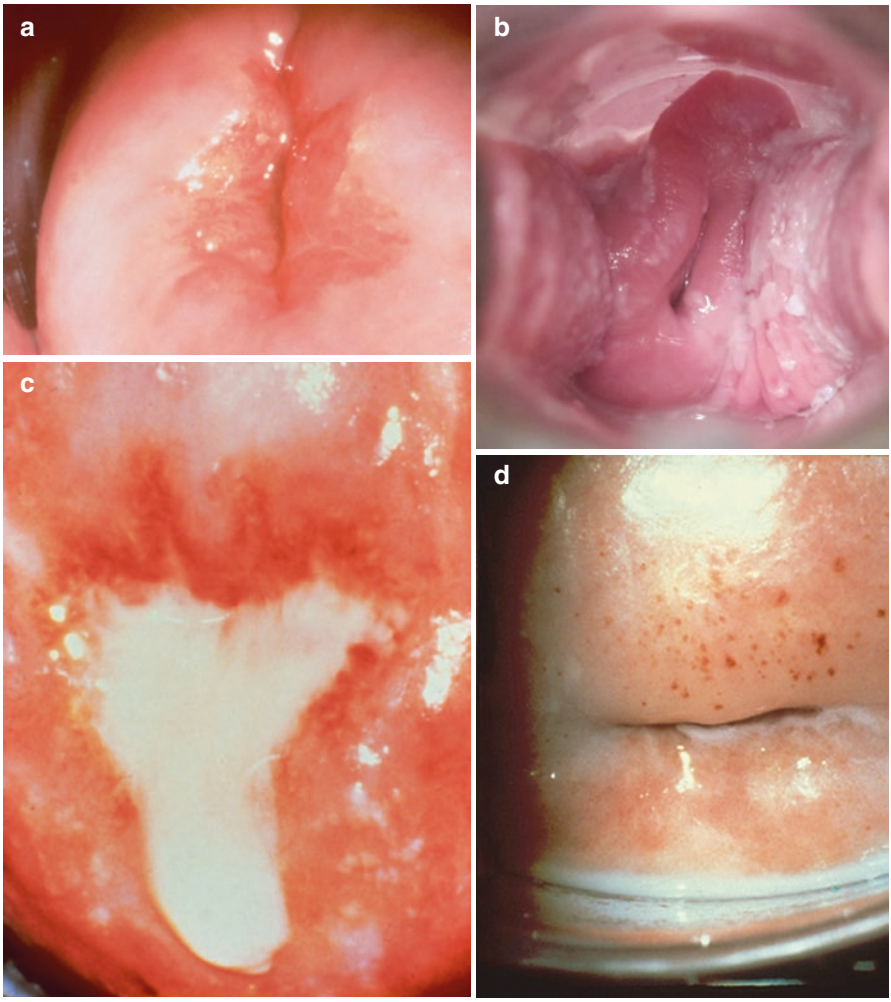

Fig. 5.1 Common findings on cervical and vaginal inspection. Images **a**, **c**, and **d** provided by the University of Washington STD Prevention Training Center with permission. (**a**) Cervical ectropion in an adolescent women; (**b**) adherent, thick, white "cottage cheese" discharge consistent with vulvovaginal candidiasis; (**c**) cervical friability with mucopurulent discharge consistent with cervicitis; (**d**) cervical petechiae or "strawberry cervix" consistent with trichomoniasis

in women with multiple sexual partners [11]. In particular, there is a strong association between BV and having female sexual partners [14]. Other risk factors for BV include black race, chronic stress, poverty, dietary factors, douching, cigarette smoking, menses, and the presence of an intrauterine device [12]. Studies have also shown that the dysbiosis associated with BV also increases risk of acquiring STIs, such as gonorrhea, chlamydia, genital herpes, and human immunodeficiency virus (HIV) [15, 13]. BV is linked to serious sequelae in the upper genital tract, particularly in the

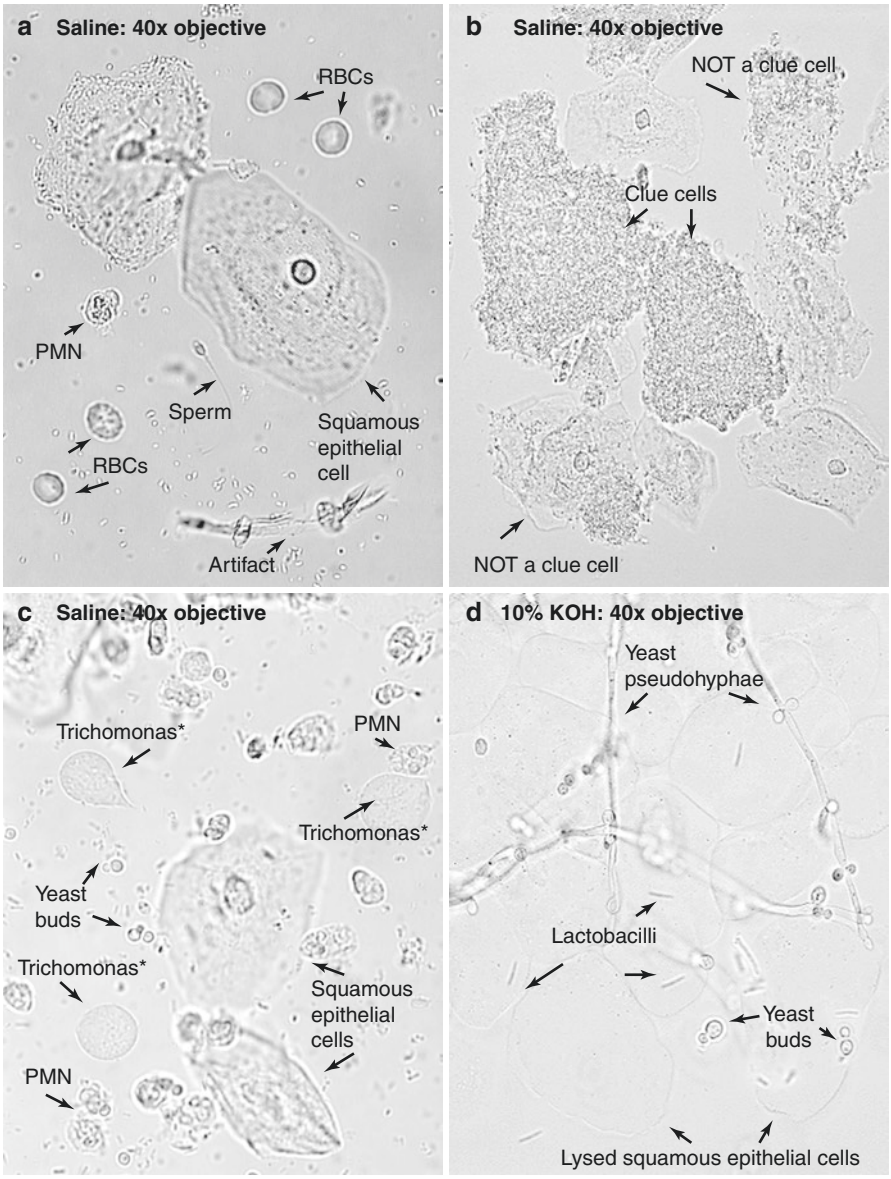

Fig. 5.2 Summary of vaginal fluid wet mount findings (40× magnification). Images **a**–**d** provided by the University of Washington STD Prevention Training Center with permission. (**a**) Normal wet prep with vaginal epithelial cells; (**b**) wet prep demonstrating clue cells and leukorrhea; (**c**) wet prep with trichomonads; (**d**) wet prep with pseudohyphae after 10% potassium hydroxide

setting of pregnancy, including premature rupture of membranes, premature delivery and low birthweight, postoperative infections after hysterectomy, development of pelvic inflammatory disease [3], and spontaneous abortion [13].

Pathophysiology

BV represents a condition in which the normal protective lactobacilli [11] are replaced by an increased number of facultative anaerobes [13] and cause symptomatic vaginitis. The vaginal pH is normally maintained at a healthy level of less than 4.7 by protective lactobacillus species, particularly *L. crispatus* and *L. jensenii* [11]. Lactobacilli hydrolyze glycogen and produce hydrogen peroxide [3] which helps maintain a low pH that is associated with healthy pregnancy and decreased risk of sexually transmitted infections [11]. In BV, this healthy microbiota shifts to one that is dominated by anaerobic coccobacilli and gram-negative bacteria, including *Gardnerella vaginalis*, *Prevotella*, *Megasphaera*, and *Mobiluncus* species [2]. A shift in vaginal microbiota causes symptoms in 60% of patients, though it is not known why some patients develop symptoms and others do not [11].

Diagnosis

Patients with symptomatic BV complain of gray, milky vaginal discharge, often with a "fishy" odor. The fishy odor is caused by the metabolism of anaerobic bacteria resulting in volatized amines at an increased pH [2, 11]. These symptoms may be more prominent after sexual intercourse or during menses when the pH of vaginal secretions increases [3]. Microscopy reveals the presence of "clue cells," which are vaginal epithelial cells with a heavy coating of bacteria [10] (Fig. 5.2). Amsel's criteria is widely used to diagnose BV and includes meeting three of the four of the following clinical criteria: (1) thin, homogenous vaginal discharge, (2) vaginal pH > 4.5, (3) positive whiff test ("fishy" odor when KOH is added to vaginal fluid), and (4) the presence of significant (>20%) amount of clue cells on wet mount [5, 16]. The efficacy of clinical diagnosis and microscopy are dependent on clinician experience [17]. The preferred laboratory method for diagnosis of BV is gram stain of vaginal fluid with a standardized scoring method known as the Nugent's score. The vaginal smear is fixed with routine gram stain procedure and analyzed under oil immersion for types of bacteria: large gram-positive rods (*Lactobacillus* morphology), gram-negative rods, small gram-variable rods (*Gardnerella vaginalis*), and curved gram-variable rods (similar to *Bacteroides* species) [10]. A score between 0 and 4 is given for each based on the presence and quantity of each morphotype, and a total score of 7–10 is consistent with BV [17]. Polymerase chain reaction (PCR) assays and culture methods have been studied for the diagnosis of BV but are not widely validated for use [11].

Treatment

The mainstay of treatment for BV in nonpregnant women is oral metronidazole 500 mg twice daily for 7 days [3] (Table 5.1). The recommended topical treatment regimens which have been shown to have equivalent efficacy though possible increased rate of recurrences [1] include metronidazole gel 0.75% one full applicator (5 gm) intravaginally once or twice daily for 5 days or clindamycin gel 2% applicator (5 gm) intravaginally once daily for 7 days. Tinidazole 2 gm daily for 2 days, tinidazole 1 gm daily for 5 days, and clindamycin 300 mg twice daily for 7 days are approved oral alternatives [3]. The FDA has approved metronidazole 750 mg extended-release tablets once daily for 7 days and a single dose of clindamycin vaginal cream [2]. Short-term cure rates following first-line therapy approach 80%; however extended follow-up shows that recurrence rates exceed 50% in the first 6–12 months. High recurrence rates have led investigators to alternative therapeutic approaches including extended and suppressive antimicrobial regimens, non-pharmacological methods, and adjunctive therapies [13]. Twice weekly intravaginal metronidazole gel for 4–6 months has been suggested to reduce recurrences [3]. Limited data exists for the use of tinidazole followed by intravaginal boric acid 600 mg at bedtime for 21 days and then suppressive 0.75% metronidazole gel twice weekly for 4–6 weeks in cases of recurrent BV [2, 18] (Table 5.1). Alcohol consumption should be avoided during treatment with nitroimidazoles, and abstinence should continue up to 72 hours after the last dose of tinidazole to prevent a disulfiram-like reaction [18]. Topical clindamycin preparations are oil-based and might weaken latex condoms and contraceptive diaphragms for up to 5 days after use [18]. Treatment of male or female sexual partners is not recommended for women diagnosed with BV. Risk reduction methods should be discussed with all women diagnosed with BV including correct and consistent condom use and limiting number of sex partners and avoidance of douching to reduce the risk of relapse [3].

Pregnancy Considerations

Pregnant women with BV can suffer adverse pregnancy outcomes including premature rupture of membranes, early labor, preterm birth, and postpartum endometritis. Symptomatic BV in pregnancy should always be treated [2]. While studies show reduced rates of preterm delivery in pregnant women treated for BV who previously delivered a premature infant [19–21], the US Preventive Services Task Force (USPSTF) concluded that there was insufficient evidence to screen asymptomatic pregnant women at high risk for preterm delivery and also recommends against routine screening for BV in asymptomatic pregnant women at low risk for preterm delivery [22]. Pregnant women who are treated for BV should be prescribed one of the following regimens: oral metronidazole 500 mg twice a day for 7 days, oral metronidazole 250 mg three times a day for 7 days, or clindamycin 300 mg twice a day for 7 days; tinidazole should be avoided in pregnancy due to limited data [3].

Table 5.1 Treatment regimens for vaginitis and cervicitis by pathogen

Diagnosis	Primary treatment regimen	Alternative treatment regimens
Vaginitis		
Bacterial vaginosis	Oral metronidazole 500 mg twice daily for 7 days	Intravaginal metronidazole gel 0.75% one full applicator (5 gm) once or twice daily for 5 days Intravaginal clindamycin gel 2% applicator (5 gm) once daily for 7 days Oral tinidazole 2 gm daily for 2 days Oral tinidazole 1 gm daily for 5 days Oral clindamycin 300 mg twice daily for 7 days
Vulvovaginal candidiasis	*Uncomplicated*: Oral fluconazole 150 mg once Clotrimazole intravaginal 1% cream 5 gm daily for 7–14 days Miconazole intravaginal 1% vaginal cream 5 gm daily for 7 days Miconazole 100 mg vaginal suppository daily for 7 days	*Complicated*: Oral fluconazole 150 mg once and repeat after 3 days Clotrimazole 100 mg vaginal tablet once daily for 7–14 days
Trichomonas vaginalis	Oral metronidazole 2 gm once Oral tinidazole 2 gm once	Oral metronidazole 500 mg twice daily for 7 days (preferred treatment for HIV-infected women)
Cervicitis		
Neisseria gonorrhoeae	Intramuscular ceftriaxone 250 mg once *plus* oral azithromycin 1 gm once	Oral cefixime 400 mg once *plus* oral azithromycin 1 gm once. Concern for declining efficacy of cefixime; use only if ceftriaxone is not available
Chlamydia trachomatis	Oral azithromycin 1 gm once Oral doxycycline 100 mg twice daily for 7 days	Oral erythromycin 500 mg four times daily for 7 days
Mycoplasma genitalium	Oral azithromycin 1 gm once Oral moxifloxacin 400 mg daily for 7–14 days if previous treatment failed	–

Topical agents do not appear to be as effective as oral agents during pregnancy, and use of clindamycin cream has been associated with increased risk of infection and premature delivery [2].

Vulvovaginal Candidiasis

Epidemiology

Vulvovaginal candidiasis (VVC) is symptomatic vaginitis caused by *Candida* yeast species [23] and is the second most common cause of vaginitis in women [24]. *Candida* species can be a normal part of vaginal flora, and up to 30% of women may

be colonized [2]. In women with symptomatic VVC, *Candida albicans* is in 80–90%, and other types of *Candida* species (including *C. tropicalis* and *C. glabrata*) are isolated in the remainder [2]. The incidence of vaginitis caused by fungi other than *C. albicans* has recently been increasing and is associated with recurrent VVC and in the setting of HIV infection [2].

Pathophysiology

Normal microbiota in the vagina inhibit yeast proliferation and germination through the production of bacteriocins. Some strains of *Lactobacillus* prevent colonization of yeast in vaginal cells by producing a protein that allows attachment of lactobacillus bacteria to mucosal cells instead of yeast and thereby reduce the risk of infection caused by yeast [25]. Symptomatic VVC is caused by overgrowth of yeast in the vagina [3]. Growth of yeast and adherence to vaginal epithelial cells are promoted by high estrogen levels [2, 25]; accordingly, women who are pregnant or taking oral contraceptives have higher rates of both yeast colonization [2] and VVC [25]. Broad-spectrum antibiotics also increase the risk of VVC by eradicating normal vaginal microbiota [2, 25]. Other major contributors to VVC include diabetes mellitus, obesity, and immunocompromised medications or conditions, including use of systemic steroids or HIV infection [25]. Sexual activity and multiple sexual partners are not associated with higher incidence of VVC [2].

Diagnosis

History taking serves as a useful tool in the diagnosis of VVC, particularly if the patient provides pertinent risk factors such as exposure to antibiotics, steroids, or oral contraceptives. The patient's medical history including known diabetes and HIV status is also helpful in supporting the diagnosis [2, 25]. The most common complaint in VVC is vulvar pruritus with little to no discharge. External dysuria and dyspareunia is occasionally noted [3]. Patient may complain of thick, adherent white discharge likened to "cottage cheese" [3] (Fig. 5.2). Pale or erythematous labia with shallow, linear ulcerations on the posterior portion of the introitus are most commonly seen on physical exam. Small erythematous papules or papulopustules beyond the primary area of erythema (satellite lesions) may be present [2]. Diagnosis can be confirmed with examination of vaginal secretions mixed with 10% KOH or saline under the microscope [2] (Fig. 5.2). Microscopic examination is not sensitive but can reveal budding yeasts or pseudohyphae. Vaginal pH is typically normal [3]. Cultures are not recommended for routine diagnosis [2] but can be helpful for recurrent disease or if the wet prep is nondiagnostic in the setting of compatible history and physical examination [25].

Treatment

Treatment of VVC depends on whether infection is uncomplicated or complicated [2]. Uncomplicated VVC involves young women who are not immunocompromised with sporadic or infrequent VVC episodes with mild to moderate symptom severity [3]. These patients usually have infection caused by *C. albicans* and will respond to treatment with short courses of topical or oral antifungal agents [2]. Topical intravaginal antifungal agents for 1–7 days and oral fluconazole 150 mg in one dose are equally effective treatment options for uncomplicated VVC [3, 23]. There are minimal side effects or toxicities associated with fluconazole at this dosage, and it may be less expensive than topical agents [2].

Complicated VVC occurs in patients with underlying immunocompromised conditions including HIV, malignancy, iatrogenic immunosuppression, or diabetes mellitus. Patients who have recurrent VVC (>4 episodes per year), severe symptomatology, or non-albicans candidiasis are also considered to have complicated disease [2, 3]. Vaginal culture should be obtained in complicated VVC to confirm the diagnosis and determine the yeast species. Severe VVC should be treated with 7–14 days of topical antifungal therapy or oral fluconazole 150 mg once followed by another dose in 72 hours [3]. Recurrent VVC can be treated with 7–14 days of topical therapy or oral fluconazole in doses between 100 and 200 mg repeated every 3 days for 1 week (days 1, 4, and 7) [3]. Recurrent VVC often requires chronic suppressive treatment with an oral antifungal agent which is continued for at least 6 months, though relapse can occur after discontinuation [2]. Species other than *C. albicans* are more likely to be resistant to fluconazole; therefore, non-fluconazole therapies should be used based on fungal susceptibility testing, if available. Boric acid powder in a 600-mg dose used intravaginally once daily for 14 days has been used with success for recurrent VVC or nonresponders to conventional treatment [2, 3] (Table 5.1).

Pregnancy Considerations

Azole antifungal agents are contraindicated during pregnancy. Pregnant women with VVC should be treated with topical agents for a minimum of 7 days [2].

Trichomoniasis

Trichomoniasis is caused by the protozoan *Trichomonas vaginalis*, a sexually transmitted pathogen [2], and has been associated with vaginitis, cervicitis, pelvic inflammatory disease, and urethritis [26]. The distribution of *Trichomonas* is equal among all age groups, unlike other STIs which are more prevalent among youth and

adolescents [27]. Presenting complaints include vaginal discharge, dysuria, pruritus, irritation, and odor [27]. Examination is notable for vulvar and vestibular edema and erythema and purulent vaginal discharge [2]. Mucosal capillary dilatation of the cervix [2] visible on speculum exam is described as "strawberry cervix" [27]. While vaginal wet preparation is only 60–70% sensitive in symptomatic patients, it is quick and inexpensive to perform [27] (Table 5.2). Motile flagellated trichomonads and many PMNs can be visualized on wet mount [2]. Culture is the gold standard for diagnosis and is more sensitive than direct visualization [26]. Various culture media are commercially available including Diamond's medium and InPouch TV [2]. PCR-based tests, including the Aptima assay for *T. vaginalis* [18], have sensitivities approaching that of culture [27] and are highly specific for infection [18].

The first-line treatment for trichomoniasis is a single 2-gram dose of the oral nitroimidazoles metronidazole and tinidazole. Alternatively, a 7-day course of oral

Table 5.2 Summary of vaginitis and cervicitis

	Normal findings	Bacterial vaginosis	Vulvovaginal candidiasis	Trichomoniasis	Cervicitis
Etiology	–	*Gardnerella vaginalis, Prevotella*, *Megasphaera*, *Mobiluncus,* and anaerobes	*Candida albicans*, non-albicans *Candida* species	*Trichomonas vaginalis*	*Neisseria gonorrhoeae*, *Chlamydia trachomatis*, *Mycoplasma genitalium*
Symptom presentation	Clear, white vaginal discharge	Fishy odor, discharge, pruritus	Pruritus, discomfort, dysuria, discharge	Vaginal discharge, pain with intercourse, dysuria, can be asymptomatic	Purulent vaginal discharge, lower abdominal discomfort, dysuria
Physical exam findings	Vaginal pH 3.8 4.2 Negative KOH "whiff" test	Malodorous "fishy," milky white vaginal discharge Vaginal pH > 4.5 Positive KOH "whiff" test	Thick, white "cottage cheese" vaginal discharge Vaginal inflammation and erythema Vaginal pH < 4.5	Malodorous, frothy, gray, or yellow-green vaginal discharge Vaginal pH > 4.5 KOH "whiff" test can be positive Cervical petechiae or "strawberry cervix"	Endocervical purulent secretions Cervical motion tenderness Negative KOH "whiff" test
Wet mount findings	Lactobacilli on wet mount	"Clue cells" on wet mount with no/few WBCs and decreased lactobacilli	Possible pseudohyphae on wet mount with few to many WBCs	Many WBCs and motile, flagellated protozoa on wet mount	Many WBCs on wet mount, possible intracellular cocci in *N. gonorrhoeae* infection

metronidazole 500 mg twice daily can be prescribed but has no advantage over the single-dose regimen for initial treatment in HIV-uninfected women [2]. The recommended treatment regimen for HIV-infected women is oral metronidazole 500 mg twice daily for 7 days [18]. All sexual partners should also undergo treatment [2]. If the first-line or alternative treatment regimens fail, a 7-day course of oral metronidazole or tinidazole in a daily 2-gram dose is recommended [2]. Alcohol consumption should be avoided during treatment with nitroimidazoles [18]. Up to 10% of clinical *Trichomonas vaginalis* isolates may be resistant to metronidazole and 1% resistant to tinidazole [18]. In recurrent disease or suspected treatment failure, sensitivity testing should be pursued [18]. Studies have shown that resistance can be overcome with higher doses of oral metronidazole or tinidazole along with intravaginal therapy [2]. For patients who do not respond to nitroimidazole treatments, consultation with an infectious diseases specialist is recommended [18] (Table 5.1). Trichomoniasis in pregnant women is associated with adverse outcomes including preterm birth, premature ruptures of membranes, and delivery of low birthweight infant [18]. All pregnant women who are diagnosed with trichomoniasis should undergo treatment and be counseled on partner treatment and condom use for prevention of sexually transmitted diseases [18].

Non-infectious Causes of Vaginitis

Foreign Body

Foreign bodies in the vagina can cause inflammation leading to foul-smelling and profuse discharge. The most common vaginal foreign body is toilet paper [28] though retained tampons can also be a cause in adolescent women [29]. Removal of the foreign body is definitive treatment.

Vulvar Vestibulitis

Vestibulitis is caused by the contact between acidic vaginal secretions and abnormal vestibular tissue which results in burning discomfort and dyspareunia [2]. Examination reveals erythematous, focal, tender lesions in the vestibule adjacent to the hymen. Symptomatic treatment includes avoidance of possible allergens and topical corticosteroids [2].

Allergic Vaginitis

Irritant contact and allergic dermatitis are non-infectious etiologies of vaginitis commonly associated with feminine hygiene products, contraceptives, and other causes [5]. Diagnosis can be confirmed by resolution of symptoms after discontinuation of the offending agent [29].

Cervicitis

Epidemiology

Cervicitis can be either infectious or non-infectious in etiology. Endocervical cervicitis is often infectious and caused by *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, or possibly *Mycoplasma genitalium*. Cervicitis can accompany trichomoniasis, genital herpes (particularly HSV-2 infection), and even BV. Risk factors for infectious cervicitis include age < 25 years old, having a new sexual partner or sex partner with concurrent partners, or an STI [18]. Ectocervical cervicitis is typically non-infectious and can be caused by idiopathic inflammation of the ectropion or chemical irritants [2].

Diagnosis

Purulent vaginal discharge is the primary symptom of women with cervicitis [2]. This may be accompanied by dysuria, abnormal uterine bleeding, lower abdominal pain, or dyspareunia. In non-infectious cervicitis, postcoital bleeding can occur due to an inflamed ectropion [2]. Leukorrhea (>10 WBC per high power field) on microscopy of the cervicovaginal fluid is often associated with chlamydia and gonorrhea infections of the cervix [18] (Fig. 5.2). Gram stain is not a sensitive indicator of infection in women [18], but the presence of intracellular gram-negative cocci can be specific for gonococcal infection [2]. Vaginal pH may also be elevated in the setting of cervicitis. Adolescent women with cervicitis should be evaluated for pelvic inflammatory disease (PID) and should be tested for gonorrhea and chlamydia infection with NAAT on either vaginal, cervical, or urine samples [18]. Microscopy, culture, and NAAT are useful diagnostic tools to evaluate for *Trichomonas vaginalis* as an etiology. The utility of specific testing for HSV-2 is unknown, though PCR, culture, and serologic testing can be considered, particularly if characteristic ulcerative lesions are noted [18]. All adolescents with concern for an STI should undergo testing for HIV and syphilis [18].

Treatment

For women at risk of STI with a presentation consistent with infectious cervicitis, empiric therapy for *Neisseria gonorrhoeae* or *Chlamydia trachomatis* should be strongly considered with either oral azithromycin 1 gm once or oral doxycycline 100 mg twice daily for 7 days and additional gonococcal-specific therapy in women at high risk or in areas with high gonorrhea prevalence (Table 5.1). Alternatively, for patients at lower risk or with good follow-up, diagnostic testing can guide pathogen-specific treatment regimens. For gonorrhea infection, primary treatment should include ceftriaxone 250 mg intramuscular with oral azithromycin 1 gm once under

direct observation. For chlamydia infection, oral doxycycline 100 mg twice daily for 7 days is an alternative to azithromycin [18]. If an STI is suspected or identified, all male or female sexual partners in the past 60 days should be referred for evaluation, testing, and treatment [18]. Women with gonorrhea or chlamydia infections should be retested 3 months after completion of therapy due to high risk of reinfection. The pathogenic role of *Mycoplasma genitalium* in cervicitis is unclear [18], and the pathogen is difficult to diagnose due to limited availability of diagnostic tests. It may be considered cases of clinically significant cervicitis that persist after azithromycin or doxycycline therapy in which re-exposure to an infected partner or medical nonadherence is unlikely. Infection may be treated with doxycycline or azithromycin, though treatment failures (particularly with doxycycline) occur and may be treated with moxifloxacin 400 mg once daily for 7–14 days. Treatment of HSV should also be prescribed if characteristic lesions are visible or diagnostic testing confirms the diagnosis [18] (Table 5.1).

Pregnancy Considerations

Diagnosis and management of cervicitis in pregnant women varies by pathogen. Pregnant women with *Neisseria gonorrhoeae* infection should be treated with dual therapy consisting of ceftriaxone 250 mg intramuscular once and oral azithromycin 1 gm in a single dose. Doxycycline is contraindicated in the second and third trimesters of pregnancy, and fluoroquinolones should also generally be avoided. Azithromycin is safe and effective; alternative treatment regimens for *Chlamydia trachomatis* infection include oral amoxicillin 500 mg three times daily for 7 days and oral erythromycin 500 mg four times daily for 7 days. Test of cure to document eradication should be performed 3–4 weeks after completion of therapy due to severe sequelae to mothers and neonates if infection persists.

Case Conclusion

Anna is evaluated for vaginitis. Her complaints of thin secretions with “fishy” odor and physical examination including microscopy with “clue cells” and elevated vaginal pH are consistent with the diagnosis of BV. Given her history of unprotected sexual intercourse, she was evaluated for STIs, including chlamydia, gonorrhea, HIV, and syphilis. A speculum examination with vaginal fluid sampling was helpful in establishing a diagnosis and ruling out retained foreign body. A pregnancy test was negative. She underwent counseling on using condoms to protect herself from unwanted pregnancy and STIs (sexually transmitted infections). She also received counseling regarding smoking cessation and vaginal health practices, such as avoiding douching, in order to prevent recurrence risk.

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