# Chapter 26
# Vaginal Discharge

**Alejandra Sanchez Lopez**

## Introduction

Vaginitis is a common disorder that affects women in all age groups, and it is characterized by vaginal complaints such as pruritus, burning, irritation, odor, and vaginal discharge [1]. Vaginitis is frequently seen in primary care and is reportedly the most common reason for gynecological consultation [2].

Vaginitis may result from infectious and noninfectious causes. In the first category, the three most common disorders are bacterial vaginosis (BV), vulvovaginal candidiasis, and trichomoniasis, accounting for most of all infectious causes [3–5]. Other pathogens can be associated with vaginal complaints such as chlamydia, gonorrhea, and herpes; however these may mimic vaginitis as they affect primarily the endocervix. Noninfectious etiologies include postmenopausal atrophic vaginitis, allergic reactions, and contact dermatitis.

A.S. Lopez, MD (✉)
Department of Medicine, Albert Einstein College of Medicine, Jacobi Medical Center, 1400 Pelham Parkway South, Bldg 1 4W9, Bronx, NY 10461, USA
e-mail: alejandra.sanchez.md@gmail.com



E. Sydney et al. (eds.), *Handbook of Outpatient Medicine*,
https://doi.org/10.1007/978-3-319-68379-9_26

## *Bacterial Vaginosis*

The vaginal flora is a microsystem composed of a diverse group of aerobic, facultative anaerobic, and obligate anaerobic species [6]. Among these bacteria, *Lactobacillus* spp. are thought to have a special role by producing hydrogen peroxide and lactic acid, which in turn provide an acidic environment responsible for a pH typically between 4 and 4.5, thereby avoiding the overgrowth of other pathogenic bacteria [7].

In bacterial vaginosis (BV), there is an alteration of the normal vaginal flora, characterized by an unrestricted overgrowth of anaerobic species, including *Gardnerella vaginalis*, *Bacteroides* species, *Mobiluncus* species, and genital mycoplasma with a concomitant reduction in the number of lactobacilli [8]. The ultimate cause of the vaginal microbiota shift is not completely understood.

BV is the most common cause of vaginitis and accounts for 22–50% of cases depending on the population studied [3, 5]. It is not considered a sexually transmitted disease, however is associated with having more than one sexual partner, a new sex partner in the last 30 days, and having a female sexual partner [9]. Treatment of male sex partners has not been beneficial in preventing the recurrence of BV [10]. Another known risk factor for BV is douching [9], likely due to the alteration of the microsystem with the use of such products.

Not only can BV be bothersome or stressful for women, but it may also increase the risk of complications after gynecologic surgery and complications in pregnancy [11, 12] and increase the risk of HIV acquisition after exposure [13] as well as HIV transmission to male sex partners [14]. Due to the conflicting results of studies regarding treating BV in asymptomatic pregnant women, the latest CDC recommendation states that evidence is insufficient to recommend routine screening for BV in asymptomatic pregnant women at risk for preterm delivery [15].

The typical presentation of BV is characterized by increased vaginal discharge and perceived malodor [3]. Among the prevailing conditions in the patient who has BV

are an elevated pH level (range, 5–5.5) and the presence of various primary amines and polyamines detected by a "fishy odor" after KOH has been added to a sample of discharge [7]. This explains why some women note this odor more when the vagina is more alkaline like after menses or having sex. However, an alkaline pH and even a positive whiff test [can also be present in trichomoniasis].

The Amsel criteria was proposed in 1983 and BV is diagnosed when three of four of the following findings are present 1. thin, homogeneous vaginal discharge; 2. vaginal pH higher than 4.5; 3. release of a fishy odor from the vaginal discharge on alkalinization with 10% potassium hydroxide ("whiff test"); 4. vaginal epithelial cells heavily coated with bacilli (clue cells) [16].

Other microscopy findings highly associated with bacterial vaginosis are scant or lack of lactobacilli (sensitivity 90%, LR 3.1) and the presence of bacilli with corkscrew motility (100% specific, LR 44) [3].

## *Vulvovaginal Candidiasis*

Vulvovaginal candidiasis represents the second most common cause of vaginitis with prevalence between 17% and 39% of cases and affects 70 and 75% of women at least once during their lifetime [17]. Characteristic signs and symptoms include a cheesy vaginal discharge, vulvar pruritus, and vulvar erythema [3]. The term vulvovaginal candidiasis (VVC) was introduced to emphasize the "vulvar" and often dominant component of symptomatic infection [18].

With the availability of over-the-counter antifungals since 1992, most symptomatic women seek these products before or in addition to an evaluation by a medical provider [19]. Women who complain of having "another" yeast infection are indeed likely to have candidiasis as evidenced by a positive likelihood ratio of 3.3 [3].

The majority of cases of VVC are caused by *C. albicans*, and among the non-albicans *Candida* spp., *Candida glabrata*

is the most common [17]. *Candida albicans* has been found in vaginal flora specimens of asymptomatic women and even in stable association with the genital epithelium. This demonstrates that the pathogenicity of such organisms can be highly dependent on host physiology [7].

A precipitating factor is not found in the majority of women with sporadic VVC episodes [18]. Attention to some associated risk factors can become important in cases of complicated or recurrent VVC, as it appears below [17]:

- Uncontrolled diabetes mellitus.
- Antibiotic use, especially in *Candida*-colonized individuals.
- Pregnancy.
- Use of oral contraceptives and contraceptive devices has shown conflicting data.
- Immunosuppression and use of immunosuppressive drugs like glucocorticoids.
- Receptive orogenital sexual intercourse.
- Frequency/periodicity of sexual intercourse.

However women who are not sexually active may develop VVC as well, and contrary to common belief, there is no increased risk among wearers of tight clothing or non-cotton underwear.

The classification of VVC determines the treatment choice: short-course antifungal in uncomplicated cases and intensive regimens in complicated cases [17, 18]. (Table 26.1).

## *Vaginal Trichomoniasis*

Trichomoniasis is caused by the motile protozoan *Trichomonas vaginalis* and is the most prevalent nonviral sexually transmitted infection in the United States [4]. Incubation of *T. vaginalis* requires 3 days to 4 weeks, and the vagina, urethra, endocervix, and bladder can be infected [6]. Up to half of women infected with *Trichomonas* are asymptomatic and if left untreated can be carriers for at least 3 months even in the absence or reexposure [20]. Signs and symptoms are not

Table 26.1 Classification for vulvovaginal candidiasis

| Uncomplicated (topical agents) | Complicated (oral agents) |
|---|---|
| • Sporadic or infrequent (≤ 3 episodes/year)<br>• Mild to moderate severity<br>• Likely to be *C. albicans*<br>• Healthy, nonpregnant host | • Recurrent (≥ 4 episodes/year)<br>• Moderate to severe disease<br>• Non-albicans candidiasis<br>• Adverse host factors (e.g., pregnancy, poorly controlled diabetes, immunosuppression) |

specific, but the classic presentation can include green–yellow and frothy vaginal discharge, vaginal irritation, vaginal spotting, dyspareunia, and dysuria. Additionally signs of vulvar inflammation can be present, and if the cervix is affected, subepithelial hemorrhages can be seen as the typical "strawberry cervix" [1, 6, 21].

*T. vaginalis* infection is associated with two- to threefold increased risk for HIV acquisition and preterm delivery and among HIV-positive women is associated with increased risk for pelvic inflammatory disease [4, 22]. The CDC recommends routine screening in all asymptomatic women with HIV infection, and screening might be considered for persons receiving care in high-prevalence settings, such as STI (sexually transmitted infection) clinics or correctional facilities, and for asymptomatic individual at high risk for STIs (e.g., multiple sex partners, exchanging sex for payment, illicit drug use, or a history of STI).

Treatment of sexual partners and abstinence from sex should be recommended until treatment is achieved, and testing for other STIs should be performed. Appropriate follow-up includes retesting within 3 months after treatment due to the high rates of reinfection; if NAAT (nucleic acid amplification test) is used, patients can be tested 2 weeks after finishing treatment. Trichomoniasis is not a national notifiable infection in the United States.

## Other Infectious Causes of Vaginitis

Some conditions that can present as vaginal complaints are chlamydia, gonorrhea, herpes, and papillomatosis. All of these will be suggested after doing a pelvic examination by the presence of papillomas or vesicles in the introitus, or in the case of chlamydia and gonorrhea, the discharge will be coming from the os of the cervix, and the patient may or may not have cervical tenderness.

## Noninfectious Causes Of Vaginitis

Atrophic vaginitis is a highly prevalent and underdiagnosed condition that impacts quality of life of many women. An international survey done by Nappi et al. found that about 40% of women have menopause-related vaginal discomfort yet less than one-third had discussed these symptoms with their primary care doctor [23]. Vaginal symptoms of atrophic vaginitis include vaginal pruritus, abnormal discharge, vaginal dryness, irritation, and dyspareunia.

As the estrogen stimulation decreases, the vulvovaginal epithelium becomes atrophic and there is loss of mucosal elasticity. The mucosa of the vagina, introitus, and labia minora appears smooth, pale, and shiny. Inflammation with patchy erythema, petechiae, and increased friability may be present. Vulvar signs of irritation caused by urinary incontinence may also be identified on pelvic examination. The vaginal pH becomes more alkaline due to the drop in glycogen levels and decreased production of lactic acid by lactobacilli. Cytologic examination of smears from the upper one-third of the vagina shows an increased proportion of parabasal cells and a decreased percentage of superficial cells [24, 25].

Other noninfectious causes of vaginal complaints can be initiated by allergic contact dermatitis or irritant contact dermatitis from hygiene products, contraceptive devices, or even retained foreign material such as tampons or condoms.

## *Key History and Physical Exam*

The typical signs and symptoms of each cause of vaginitis have been mentioned in the previous pages, but in summary the symptoms to elicit from patients include the characteristics of the discharge (color, consistency, quantity, abnormal odor) and presence of vaginal pruritus or irritation, along with other symptoms such as dysuria and dyspareunia and if the patient has used any over-the-counter medication prior to presentation. In Table 26.2 there is a list of some of the signs and symptoms that have been shown to have a good predictive value for the three most common causes of infectious vaginitis. It is also good practice to obtain a sexual history to determine the patient's risk of having a current sexually transmitted disease by asking the five Ps: partners, practice, prevention of pregnancy, protection from STIs, and past history of STIs.

When performing a pelvic examination, inspect the vulva for signs of inflammation such as erythema, edema, and excoriation as well as for the presence of vesicular lesions or papillomas.

After insertion of the speculum, if discharge is present, the source should be determined (e.g., cervical os suggests a cervicitis as opposed to a vaginitis). The vaginal walls and the cervix should be examined for signs of inflammation or friability, noting the characteristics of the discharge. Finally the presence of cervical motion tenderness should be evaluated.

For pH and microscopy testing, discharge should be sampled from the sidewall of the vagina. If purulent cervical discharge is noted, make sure to obtain a sample to test for chlamydia and gonorrhea.

Table 26.2 Likelihood ratios of different vaginal complaints

| Diagnosis | Vaginal complaint | Likelihood ratio LR (95% CI) |
|---|---|---|
| Candidiasis | Itching (chief complaint) | 3.3 (2.4–4.8) |
| | Curdy discharge | 6.1 (2.5–14) |
| | Vulvovaginal inflammatory signs | 2.1–8.4 (1.3–16) |
| | Self-diagnosis | 3.3 (1.2–9.1) |
| | Curdy discharge and itching | 150 (20–1000) |
| Bacterial vaginosis | Odor noted by clinician | 3.2 (2.1–4.7) |
| Trichomoniasis | Erythema or edema | 6.4 (1.6–26) |

Adapted from Anderson MR, Klink K, Cohrssen A. Evaluation of vaginal complaints. JAMA 2004;291(11):1368–1379

## Diagnostic Evaluation

A good history and physical examination will provide clues into the cause of the vaginal complaints, but it is imperative to mention that no symptom has enough predictive power to make a diagnosis.

1. Assess the vaginal pH; a normal pH (< 4.5) will point toward VVC and a pH > 4.5 can be due to BV or TV (*Trichomonas vaginalis*).
2. Wet mount microscopic examination:
   - Trichomonads (sensitivity 51%–65%, but 100% specific). Evaluate slides immediately because sensitivity for trichomoniasis declines as evaluation is delayed, decreasing by up to 20% within 1 h after collection.
   - Clue cells (vaginal squamous epithelial cells with copious adherent coccobacilli).
   - Yeast (hyphae or spores).

3. Add 10% potassium hydroxide (KOH) to a drop of vaginal discharge and "whiff" the sample. If a fishy odor is present, that constitutes a positive "whiff test."

Next, take the slide to the microscope; the use of KOH will make yeast more visible (sensitivity 38–83% and specificity 77–94%).

BV can be diagnosed if three of four Amsel criteria are present: vaginal pH > 4.5, thin watery discharge, positive whiff test, and wet mount with > 20% clue cells. In a prospective observational study, a vaginal pH of more than 4.5 was found to be the most sensitive (89%), and a positive whiff test was the most specific (93%) method of detecting BV [26]. Culture of *G. vaginalis* is not recommended as a diagnostic tool because of its low specificity [1].

If preliminary tests are inconclusive, further testing should be performed. If pH is normal, then obtain a culture for vulvovaginal candidiasis. Unfortunately, up to 50% of patients with culture-positive symptomatic VVC will have negative microscopy [17]. If the pH was > 4.5 but the Amsel criteria was not fulfilled or patient has risks for STIs, then perform further testing for trichomoniasis. Culture was considered the gold standard method for diagnosing *T. vaginalis* infection before molecular detection methods became available. Culture has a sensitivity of 75–96% and a specificity of up to 100%, but nucleic acid amplification test (NAAT) is highly sensitive, often detecting three to five times more *T. vaginalis* infections than wet mount microscopy (Fig. 26.1).

## Treatment

See the 2015 CDC guidelines as described below in Table 26.3 [4]. Recurrent vulvovaginal candidiasis is treated with either oral or topical azole and continued until the patient is asymptomatic and culture negative. Ongoing suppressive regimens include once weekly dosing of either 500 mg clotrimazole suppositories or 150 mg fluconazole orally [17].

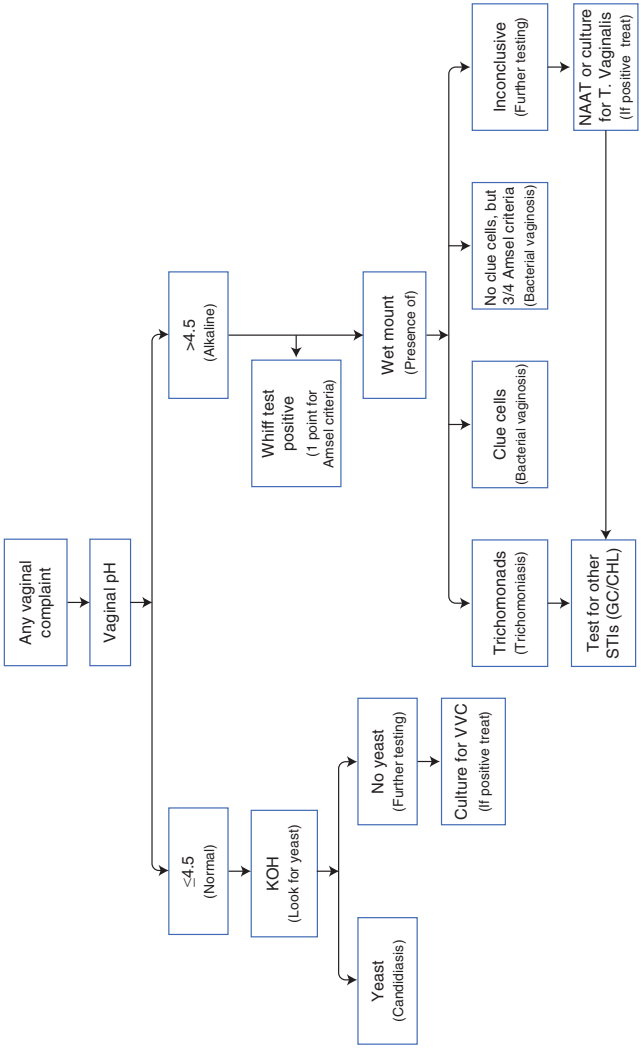


FIG. 26.1 Vaginitis algorithm

TABLE 26.3 Sexually transmitted disease treatment guidelines, 2015

| Pathogen | Recommended regimens | Alternative regimens | Other considerations |
|---|---|---|---|
| Bacterial vaginosis | • *Metronidazole* 500 mg orally twice a day for 7 days<br>OR<br>• *Metronidazole* gel 0.75%, one full applicator (5 g) intravaginally, once a day for 5 daysOR<br>• *Clindamycin* cream 2%, one full applicator (5 g) intravaginally at bedtime for 7 days | • *Tinidazole* 2 g orally once daily for 2 days<br>OR<br>• *Tinidazole* 1 g orally once daily for 5 days<br>OR<br>• *Clindamycin* 300 mg orally twice daily for 7 days<br>OR<br>• *Clindamycin* ovules 100 mg intravaginally at bedtime for 3 days* | • Clindamycin ovules use an oleaginous base that might weaken latex or rubber products (e.g., condoms and vaginal contraceptive diaphragms). The use of such products within 72 h following treatment with clindamycin ovules is not recommended<br>• Tinidazole should be avoided during pregnancy |
| Trichomoniasis | • *Metronidazole* 2 g orally in a single dose<br>OR<br>• *Tinidazole* 2 g orally in a single dose | • *Metronidazole* 500 mg orally twice a day for 7 days | • Treat sexual partner<br>• Consider delaying breastfeeding for 12–24 h following maternal treatment of a single 2 g dose of metronidazole<br>• Tinidazole should be avoided during pregnancy |

(continued)

**Table 26.3** (continued)

| Pathogen | Recommended regimens | Alternative regimens | Other considerations |
|---|---|---|---|
| Candidiasis | Over-the-counter intravaginal agents: (any one of the following)<br>*Clotrimazole* 2% cream 5 g intravaginally daily for 3 days<br>*Miconazole* 4% cream 5 g intravaginally daily for 3 days<br>*Miconazole* 200 mg vaginal suppository, one suppository for 3 days<br>*Miconazole* 1200 mg vaginal suppository, one suppository for 1 day<br>*Tioconazole* 6.5% ointment 5 g intravaginally in a single application<br>*Prescription intravaginal agents:*<br>Oral agent:<br>*Fluconazole* 150 mg orally in a single dose<br>Topicals:<br>*Terconazole* 0.8% cream 5 g intravaginally daily for 3 days<br>*Terconazole* 80 mg vaginal suppository, one suppository daily for 3 days | | • The creams and suppositories in these regimens are oil based and might weaken latex condoms and diaphragms |

| Pathogen | Recommended regimens | Alternative regimens | Other considerations |
|---|---|---|---|
| Gonorrhea | *Ceftriaxone* 250 mg IM | *Cefixime* 400 mg orally in a single dose | Without cultures, empirically treat for chlamydia as well |
| Chlamydia | *Azithromycin* 1 gm PO single dose | *Doxycycline* 100 mg twice a day for 7 days | Without cultures, empirically treat for gonorrhea as well |
| Genital herpes (first episode) | *Acyclovir 400 mg PO TID* for 7–10 days<br>*Valacyclovir 1 g PO BID* for 7–10 days | | Treatment can be extended if healing is incomplete after 10 days of therapy. |
| Genital herpes (suppressive therapy) | *Acyclovir 400 mg PO BID*<br>*Valacyclovir 1 g PO once a day* | | Suppressive therapy is encouraged in discordant couples to decrease the rate of transmission, along with condom use and abstinence during recurrence. |
| Genital herpes (episodic therapy for recurrent herpes) | *Acyclovir 400 mg PO TID* for 5 days<br>*Valacyclovir 1 g PO once a day* for 5 days | | |

*Clindamycin ovules use an oleaginous base that might weaken latex or rubber products (e.g., condoms and vaginal contraceptive diaphragms). Use of such products within 72 hours following treatment with clindamycin ovules is not recommended.

Treatment for atrophic vaginitis includes topical estrogen for women without contraindications or vaginal lubricant in women who prefer not to use estrogens.

Clinical Pearls

- All women with vaginal complaints should undergo at a minimum pelvic examination and office-based microscopy before starting treatment, given that no symptom has enough predictive power to make a diagnosis.
- The three most common causes of infectious vaginitis are bacterial vaginosis, vulvovaginal candidiasis, and trichomoniasis.
- The presence of vaginal pruritus, curdy discharge, and a normal pH is highly suggestive of vulvovaginal candidiasis.
- Classifying vulvovaginal candidiasis as noncomplicated or complicated will aid in the choice of treatment.
- Bacterial vaginosis and *T. vaginalis* increase the risk of HIV acquisition and transmission and are associated with adverse pregnancy outcomes.

Don't Miss This!

- If trichomoniasis is diagnosed, be certain to test for other STIs and treat the sexual partner(s).
- Patients with cervical discharge with or without systemic symptoms need to be evaluated for GC/CHL.
- Patients with cervical discharge and cervical motion tenderness should be treated for GC/CHL empirically.

## References

1. Hainer B, Gibson M. Vaginitis: diagnosis and treatment. Am Fam Physician. 2011;83(7):807–15.
2. Kent HL. Epidemiology of vaginitis. Am J Obstet Gynecol. 1991;165(4):1168–76.
3. Anderson MR, Klink K, Cohrssen A. Evaluation of vaginal complaints. JAMA. 2004;291(11):1368–79.

4. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines. MMWR Recomm Rep. 2015;64(RR-03):1–137.
5. Sobel JD. Vulvovaginitis in healthy women. Compr Ther. 1999;25(6–7):335–46.
6. Hoffman BL. Williams gynecology. 3rd ed. New York: McGraw-Hill Education; 2016.
7. Larsen B, Monif GR. Understanding the bacterial flora of the female genital tract. Clin Infect Dis. 2001;32(4):e69–77.
8. Hillier SL. Diagnostic microbiology of bacterial vaginosis. Am J Obstet Gynecol. 1993;169:455–9.
9. Shirley RL. Acute vulvovaginitis. N Engl J Med. 2006;355:1244–52.
10. Mehta SD. Systematic review of randomized trials of treatment of male sexual partners for improved bacteria vaginosis outcomes in women. Sex Transm Dis. 2012;39:822–30.
11. Laxmi U, Agrawal S, Raghunandan C, et al. Association of bacterial vaginosis with adverse fetomaternal outcome in women with spontaneous preterm labor: a prospective cohort study. J Matern Fetal Neonatal Med. 2012;25:64–7.
12. Nelson DB, Hanlon A, Hassan S, et al. Preterm labor and bacterial vaginosis-associated bacteria among urban women. J Perinat Med. 2009;37:130–4.
13. Taha TE, et al. Bacterial vaginosis and disturbances of vaginal flora: association with increased acquisition of HIV. AIDS. 1998;12(13):1699–706.
14. Cohen CR, Lingappa JR, Baeten JM, et al. Bacterial vaginosis associated with increased risk of female-to-male HIV-1 transmission: a prospective cohort analysis among African couples. PLoS Med. 2012;9:e1001251.
15. US Preventive Services Task Force. Screening for bacterial vaginosis in pregnancy to prevent preterm delivery: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med. 2008;148:214–9.
16. Amsel R, Totten PA, Spiegel CA, et al. Nonspecific vaginitis. Diagnostic criteria and microbial and epidemiologic associations. Am J Med. 1983;74:14–22.
17. Sobel JD. Vulvovaginal candidosis. Lancet. 2007; 369(9577):1961–71.
18. Sobel JD, Sabastian F, et al. Vulvovaginal candidiasis: epidemiologic, diagnostic, and therapeutic considerations. Am J Obstet Gynecol. 1998;178(2):203–11.
19. Foxman B, Marsh JV, Gillespie B, Sobel JD. Frequency and response to vaginal symptoms among white and African

American women: results of a random digit dialing survey. J Womens Health. 1998;7:1167–74.
20. Van Der Pol B, Williams JA, Orr DP, et al. Prevalence, incidence, natural history, and response to treatment of Trichomonas vaginalis infection among adolescent women. J Infect Dis. 2005;192(12):2039–44.
21. Wilson JF. In the clinic: vaginitis and cervicitis. Ann Intern Med. 2009;151(5):ITC3-1-15.
22. Silver BJ, Guy RJ, Kaldor JM, Jamil MS, Rumbold AR. Trichomonas vaginalis as a cause of perinatal morbidity: a systematic review and meta-analysis. Sex Transm Dis. 2014;41(6):369.
23. Nappi RE, Kokot-Kierepa M. Women's voices in the menopause: results from an international survey on vaginal atrophy. Maturitas. 2010;67(3):233.
24. Bachmann GA, Nevadunsky NS. Diagnosis and treatment of atrophic vaginitis. Am Fam Physician. 2000;61(10):3090.
25. Stika CS. Atrophic vaginitis. Dermatol Ther. 2010;23:514–22.
26. Gutman RE, Peipert JF, Weitzen S, Blume J. Evaluation of clinical methods for diagnosing bacterial vaginosis. Obstet Gynecol. 2005;105(3):551–6.