Vaginitis

Learning Objectives:

Upon completion of this module, the learner will be able to:

1. Discuss the etiology of trichomoniasis, bacterial vaginosis (BV), and candidiasis.
2. Compare and contrast the clinical manifestations of trichomoniasis, BV, and candidiasis.
3. State the clinical and laboratory criteria for the diagnosis of trichomoniasis, BV, and candidiasis.
4. Discuss the clinical management of vaginitis to include treatment, follow-up, patient counseling and partner management.
5. Discuss non-infectious causes of vaginitis to be considered in the differential diagnosis.

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VAGINITIS

Vaginitis is usually characterized by a vaginal discharge or vulvar itching and irritation; a vaginal odor may be present. The three common diseases associated with vaginal infection include trichomoniasis (15-20%), bacterial vaginosis (40-45%), and vulvovaginal candidiasis (20-25%) or, not infrequently, a combination of these processes. Other causes of vaginal discharge or irritation include mucopurulent cervicitis caused by *Chlamydia trachomatis* or *Neisseria gonorrhoeae*, or herpes simplex virus, atrophic vaginitis, allergic or irritant reactions (spermicides, deodorants, minipad adhesive), vulvar vestibulitis, lichen simplex chronicus and lichen sclerosis (especially pruritis) and foreign bodies (retained tampons). Trichomoniasis and bacterial vaginosis increase susceptibility to HIV acquisition.

The vagina is a dynamic ecosystem that normally contains approximately 10^9 bacterial colony-forming units per gram of vaginal fluid. The normal bacterial flora is dominated by lactobacilli, but a variety of other organisms, primarily anaerobes, are also present at lower levels. Lactic acids and other organic acids are metabolized from glycogen by the lactobacilli, maintaining the vaginal pH between 3.8 and 4.2. The acidic environment inhibits the overgrowth of bacteria and other organisms with pathogenic potential. The normal vaginal discharge is clear to white, odorless, and of high viscosity.

Diagnosis and Evaluation:

Note character of vaginal discharge.

Ensure normal appearance of cervix with speculum exam to rule out cervicitis as a source of abnormal vaginal discharge.

Collect discharge from the lateral wall of the vagina. Avoid collection of material from the cervical os or posterior fornix areas, as these are likely to contain cervical secretions which typically have a higher pH and do not accurately reflect vaginal pH.

Determine vaginal pH with narrow-range pH paper.

Perform microscopic exam of discharge with 10% KOH and 0.9% normal saline (separately).
To view a free video, Examination of Vaginal Wet Mounts, go to:
http://depts.washington.edu/nnptc/online_training/wet_preps_video.html.

Perform amine or "whiff" test after application of 10% KOH to discharge.

There are also several rapid tests that can be used to characterize vaginal fluid and diagnose common infections, including BV, candidiasis, and trichomoniasis.

Cultures are not used routinely, but are available for both *T. vaginalis* and *Candida. spp*. Culture may be useful in the management of persistent or recurrent vulvovaginal candidiasis, and many experts recommend fungal culture prior to starting suppressive therapy for vulvovaginal candidiasis. Routine bacterial culture (including quantitative culture) is not useful for the diagnosis of BV. A number of nucleic acid amplified tests (NAAT) for vaginal bacteria have become available. Apart from those that target detection of endocervical infection with *Chlamydia trachomatis* or *Neisseria gonorrhoeae*, these tests are not recommended for routine use due to very high cost and to the fact that they have not been shown to improve diagnosis and treatment of common vaginal infections.

Trichomoniasis

Learning Objectives

Upon completion of this module, the learner will be able to:

1. Discuss the etiology and clinical manifestations of trichomoniasis.
2. List the laboratory tests available for the diagnosis of trichomoniasis.
3. Discuss the clinical management of trichomoniasis to include treatment, follow-up patient counseling, and partner management.

This curricular outline was developed by the Curriculum Committee of the National Network of STD/HIV Prevention Training Centers. This project was funded through a grant by the US Centers for Disease Control and Prevention.

I. Epidemiology

A. Estimated 5 million cases annually in the U.S. at a medical cost of $375 million.

B. Almost always sexually transmitted; fomite transmission is rare. Because *T. vaginalis* may persist for months to years in epithelial crypts and periglandular areas, distinguishing between persistent, subclinical infection and remote sexual acquisition is not always possible.

C. Transmission between female sex partners has been documented.

II. Pathogenesis

A. Causative agent: *Trichomonas vaginalis*, flagellated anaerobic protozoa.

B. Associations with:

1. Pre-term rupture of membranes and pre-term delivery.
2. Increased risk of HIV acquisition.

III. Clinical Manifestations

A. Vaginitis:

1. "Frothy" gray or yellow-green vaginal discharge.
2. Pruritus.
3. Cervical petechiae ("strawberry cervix"): classic presentation, but occurs in minority of cases.

B. Can also infect Skene's ducts and urethra.

C. Up to 50% of infected women are asymptomatic, although 30% of those who are asymptomatic may become symptomatic within six months.

D. May cause up to ~11-13% of nongonococcal urethritis in males, but urethral infection is frequently asymptomatic. Men may harbor the parasite in seminal vesicles, as it has been demonstrated in semen more commonly than in urine or urethral fluid; however, it is not known to cause clinical manifestations at this site.

IV. Diagnosis

A. Vaginal pH >4.5 often present.

B. Positive amine (KOH) test ("whiff" test) in many cases.

C. Detection of motile trichomonads seen in saline wet mount has been the longstanding mode of diagnosis, but is highly insensitive compared to both culture and to nucleic acid amplification assay (sensitivity approximately 42% to 50% at best). White blood cells are frequently seen. If it is the only test available, saline microscopy should be performed as soon as possible after obtaining the specimen (ideally within 10 minutes) since sensitivity of this test declines rapidly with time. Trichomonads, especially if the specimen is old and they have become sluggish, may closely resemble white blood cells. Similarly, white blood cells can be confused with trichomonads, so motility should be assessed.

(To view a free video, Examination of Vaginal Wet Mounts, see URL under Vaginitis, Diagnosis and Evaluation, Page 4, above.)

D. Culture (Diamond's media or InPouch TV) is considerably more sensitive than wet mount.

E. Nucleic acid amplification testing is offered by some laboratories, and will soon be commercially available. It is the most sensitive test, detecting over 90% of infections defined by a composite gold standard, and has high specificity (>99%).

F. Non-amplified DNA probes are significantly more sensitive than wet prep, but are also more expensive and not widely available.

G. Pap smear performed by conventional cytology has variable sensitivity and relatively low specificity; therefore, it is not recommended for diagnosis of trichomoniasis. However, liquid cytology offers an accurate means for diagnosis, and can be used.

H. For suspected trichomoniasis in males, first-void urine can be concentrated 10x and examined for motile trichomonads; urethral swab or 10 cc of first-void urine may also be obtained for culture. However, as nucleic acid amplification testing becomes more widely available, it is likely that this will become the standard approach.

V. Treatment

A. Metronidazole or Tinidazole (95% cure rate):

1. Recommended: metronidazole 2.0 gm po or tinidazole 2.0 gm po, either given as one-time single dose. However, single-dose therapy has been shown to be significantly less effective in HIV-infected women with vaginal trichomoniasis; the 7-day metronidazole regimen (below) should be considered in this case.

2. Alternate regimen: metronidazole 500 mg bid for 7 days.

3. All patients with trichomoniasis should be treated (whether symptomatic or asymptomatic).

4. Sex partners reported from the last 90 days (or the last partner prior to that time period) should be treated; test for other bacterial STDs.

5. Metronidazole gel (intravaginal) is ineffective for trichomoniasis and should not be used.

6. Options in the setting of metronidazole allergy include use of an alternative drug (e.g., paromomycin) or desensitization (see CDC Treatment Guidelines for protocol). Tinidazole is contraindicated in patients with metronidazole allergy, as there is cross-sensitivity.

7. Alcohol should not be ingested for at least 24 hours after completion of oral metronidazole therapy, and for at least 48 hours after completion of oral tinidazole therapy.

B. Pregnancy and Lactation:

1. Metronidazole 2.0 gm one time in single oral dose. Tinidazole is not recommended.
2. No evidence of teratogenicity for metronidazole; treatment may be administered throughout pregnancy.

C. Treatment failures:

1. Repeat standard single-dose treatment regimen (metronidazole 2.0 g single oral dose).

2. Assure treatment of sex partners.

3. Metronidazole 500 mg bid for 7 days if 2 g single oral dose was used as the initial regimen, or tinidazole 2 g one-time single oral dose.

4. If above regimen fails, use metronidazole or tinidazole 2 gm daily x 5 days.

5. Increasing the dose and duration of metronidazole or administering it intravenously may be of use; published regimens are available. Other agents, including topical paromomycin, are available.

6. If repeated treatment failures occur on higher dose regimens, contact Division of STD Prevention, CDC for metronidazole-susceptibility testing (Phone: 770-488-4115).

D. Follow-up: Due to a high rate of reinfection (17% reinfected within 3 month in one study), rescreening for *T. vaginalis* at 3 months following initial infection may be considered for sexually active women with trichomoniasis; however, the benefit of this approach has not been fully evaluated.

E. Consider testing for other bacterial STDs and ensure that routine HIV testing has been offered, per current guidelines.

VI. Prevention

A. Partner management: sex partners should be treated. Patients should be instructed to avoid sex until they and their sex partners are cured. In the absence of a microbiologic test of cure, this means when therapy has been completed (or 48 hours after single dose treatment) and patient and partner(s) are asymptomatic.

B. Patient counseling and education:

1. Nature of the infection:
   a) Timely healthcare-seeking for abnormal vaginal discharge.
   b) Education of women about normal vs. abnormal discharge.

2. Transmission issues: trichomoniasis is almost always sexually transmitted; fomite transmission is rare.

3. Risk reduction:
   a) Assess client's behavior-change potential.

b) Discuss prevention strategies (abstinence, monogamy, condoms, limit number of sex partners, etc.). Latex condoms, when used consistently and correctly, can reduce the risk of transmission of trichomonas.
c) Develop individualized risk-reduction plans.

VII. References: (See end of module)

Candidiasis

Learning Objectives

Upon completion of this module the learner will be able to:

1. Discuss the etiology and clinical manifestations of candidiasis.
2. State the clinical and laboratory criteria for the diagnosis of candidiasis.
3. Discuss the clinical management of candidiasis to include treatment, follow-up patient counseling, and partner management.
4. Discuss the relationship and management of candidiasis when there is co-infection with HIV.

This curricular outline was developed by the Curriculum Committee of the National Network of STD/HIV Prevention Training Centers. This project was funded through a grant by the US Centers for Disease Control and Prevention.

I. Epidemiology

A. Not generally considered a sexually transmitted condition.

B. Frequent infections (recurrent VVC) may be linked to diabetes, corticosteroids, repeated courses of antibiotics, pregnancy, or HIV disease, although >95% of patients have none of these risk factors.

C. Most cases of candidiasis are caused by *C. albicans* (85%-90%); *C. glabrata* and *C. parapsilosis* responsible for 5%-10% of cases.

II. Pathogenesis:

VVC is caused by overgrowth of or hypersensitivity to *Candida albicans* and other non-albicans species that grow as oval, budding yeast cells and as chains of cells (pseudohyphae). *Candida* species are normal flora of skin, mouth, and vagina, and are not considered to be sexually transmitted pathogens. Clinical infection occurs in the setting of excessive growth of yeast, which is usually kept in check by normal vaginal bacteria (especially lactobacilli), or to an exaggerated host response to the yeast itself. Up to 40% of women are colonized by *Candida* as part of normal vaginal flora. As noted above, conditions which disrupt normal vaginal ecology or host immunity can predispose to vaginal yeast infections (e.g., antibiotic use, diabetes, HIV infection).

III. Clinical Manifestations

A. Thick, white, curdy vaginal discharge ("cottage-cheese-like").

B. Vulvar pruritus, erythema, irritation, occasional erythmatous "satellite" lesions.

C. External dysuria.

IV. Diagnosis

A. Clinical presentation and symptoms.

B. Visualization of pseudohyphae (mycelic) and/or budding yeast (conidia) on 10% KOH examination (preferred), saline wet mount, or Gram stain.

(To view a free video, Examination of Vaginal Wet Mounts, see URL under Vaginitis,

Diagnosis and Evaluation, Page 4, above.)

C. pH usually <4.5. If pH is abnormally high (≥ 4.5), consider concurrent BV or trichomoniasis.

D. Cultures not useful for routine diagnosis, since cultures may detect colonization rather than clinically significant infections and therefore should not be treated. Cultures may be useful to detect non-albicans species or resistant organisms in women with recurrent disease. Also, many experts recommend culture prior to initiating suppressive therapy for recurrent VVC in order to confirm the presence of yeast and, ideally, to assess susceptibility to antifungal medications (See Section VI, below)

E. DNA probe is available.

V. Treatment

A. Uncomplicated VVC (mild to moderate, sporadic, disease in a normal host with normally susceptible *C. albicans*), responds to short (three-day) and single-dose oral therapy. In contrast, complicated VVC (severe local or recurrent VVC in an abnormal host; e.g., an uncontrolled diabetic, HIV infection with low CD4 count), requires longer duration (10-14 days of daily topical imidazoles, or fluconazole given on days 1 and 3 (e.g., Monday and Thursday) for 2 weeks; fluconazole for the treatment of VVC does not require daily oral dosing).

B. Recommended regimens:

1. Intravaginal agents:

Butoconazole 2% cream, 5 g intravaginally for 3 days*†
Butaconazole 2% cream (bioadhesive product), 5 g intravaginally single dose*
Clotrimazole 1% cream 5 g intravaginally for 7-14 days*†
Clotrimazole 2% cream 5 g intravaginally for 3 days*†
Miconazole 2% cream 5 g intravaginally for 7 days*†
Miconazole 4% cream 5 g intravaginally for 3 days*†
Miconazole 200 mg vaginal suppository, 1 suppository for 3 days*†
Miconazole 100 mg vaginal suppository, 1 suppository for 7 days*†
Miconazole 1,200 mg vaginal suppository, 1 suppository for 1 day*†

Nystatin 100,000-unit vaginal tablet, 1 tablet for 14 days
Tioconazole 6.5% ointment 5 g intravaginally in a single application*†
Terconazole 0.4% cream 5 g intravaginally for 7 days*
Terconazole 0.8% cream 5 g intravaginally for 3 days*
Terconazole 80 mg vaginal suppository, 1 suppository for 3 days*

2. Oral agent: Fluconazole 150 mg oral tablet, 1 tablet in a single dose.

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*These creams and suppositories are oil-based and may weaken latex condoms and diaphragms. Refer to condom product labeling for further information.
† Over-the-counter (OTC) preparations.

C. In pregnant patients, only topical imidazoles are recommended. Only clotrimazole and miconazole are Category B in pregnancy; all other anti-yeast medications are Category C, because of the observation in fetal rat models that they are associated with decrease in skull ossification. For this reason, the Category C drugs are not first-line treatment in the first trimester, but women treated inadvertently at usual doses should be unaffected. **Fluconazole should not be used**.

D. Routine treatment of sex partners is usually not warranted. Male partners with balanitis or penile dermatitis may benefit from treatment.

F. In cases associated with severe vulvitis and intense pruritis, topical applications of low potency corticosteroid cream or nystatin cream may be beneficial.

VI. Recurrent Vulvovaginal Candidiasis (RVVC)

A. Women who experience four or more episodes of VVC annually may be considered to have RVVC. While some women with RVVC have apparent risk factors (see above), most women do not. Recurrent disease may be more likely to be due to non-*albicans* species.

B. The optimal treatment has not been established. An initial intensive regimen of 7-14 days of topical treatment or sequential oral doses of fluconazole (150 mg on days 1 and 4 for two successive weeks), followed by a maintenance regimen for at least six months is recommended. Weekly fluconazole (150 mg single oral dose) reduces the frequency of episodes.

C. RVVC should be confirmed by culture before initiating maintenance therapy. VVC diagnosis should also be periodically re-confirmed, and the presence of other contributory causes (new trichomoniasis or BV) assessed.

D. Patients with RVVC who are receiving treatment should receive regular follow-up to monitor the effectiveness of therapy and the occurrence of drug-related side effects.

E. Boric acid tablets intravaginally (500 mg in type-O gel capsule nightly for 14 days) may be effective for RVVC or for VVC due to non-*albicans* species; **do not use in pregnancy.**

VI. Prevention

A. Partner management: VVC is not usually acquired through sexual intercourse; treatment of sex partners is not recommended. A minority of male sex partners may have balanitis, characterized by erythematous areas on the glans penis in conjunction with pruritis or irritation, and may benefit from treatment with topical antifungal agents to relieve symptoms.

B. Patient counseling and education:
   1. Education re: normal vs. abnormal discharge and signs warranting evaluation.
   2. Avoidance of unnecessary antibiotic treatment.
   3. Control of predisposing conditions (e.g., diabetes)

VII. References: (See end of module)

Bacterial Vaginosis

Learning Objectives

Upon completion of this module, the learner will be able to:

1. Discuss the etiology and clinical manifestations of BV.
2. State the clinical and laboratory criteria for the presumptive diagnosis of BV.
3. Discuss the clinical management of BV to include treatment, follow-up, patient counseling, and partner management.

This curricular outline was developed by the Curriculum Committee of the National Network of STD/HIV Prevention Training Centers. This project was funded through a grant by the U.S. Centers for Disease Control and Prevention.

I. Epidemiology

A. Prevalence varies by population; 5-25% among college students, 12-35% among women with a concurrent STD.

B. Currently not considered sexually transmitted in heterosexual women, but related to sexual activity in some women.

C. Widely distributed. More common in African-American women, women who douche, women using IUDs, women having new or more than 2 sex partners, and in women who have sex with other women.

D. BV linked to premature rupture of membranes, premature delivery, and low birth-weight delivery, acquisition of HIV, and development of PID and post-operative infections of gynecological procedures.

II. Pathogenesis

A. Overgrowth of bacteria species normally present in vagina at low levels, such as *Gardnerella*, various newly defined *Clostridiales* species, *Bacteroides*, *Mycoplasma hominis*, *Mobiluncus*, *Peptostreptococcus*.

B. BV is strongly associated with loss of protective (peroxide-producing) lactobacilli, which are normally present in vagina:

1. Acidic vaginal pH normally maintained by *Lactobacillus* through breakdown of glycogen to lactic acid.

2. Lactic acid maintains a low pH which may directly inhibit some organisms. Lactobacillus production of hydrogen peroxide is also important.

3. Loss of protective lactobacilli may lead to BV.

III. Clinical Manifestations

A. 50% report malodorous vaginal discharge, sometimes reported more commonly after unprotected vaginal intercourse and after completion of menses.

B. 50% asymptomatic:

1. May have increased discharge.
2. Vaginal pruritus may or may not be present.

IV. Diagnosis

A. Amsel criteria: must have at least three of the following findings:

1. Vaginal pH >4.5 (most sensitive but least specific).

2. Presence of "clue cells" on wet mount examination (bacterial clumping upon the borders of epithelial cells). Clue cells should constitute at least 20% of all epithelial cells (an occasional clue cell does not fulfill this criteria).

(To view a free video, Examination of Vaginal Wet Mounts, see URL under Vaginitis, Diagnosis and Evaluation, Page 4, above.)

3. Positive amine or "whiff test" (liberation of amines with or without the addition of 10% KOH, with resultant "fishy" odor).

4. Homogeneous, non-viscous, milky-white discharge adherent to the vaginal walls.

B. Gram stain of vaginal fluid is often used to diagnose BV in research studies (Nugent criteria). A normal Gram stain shows predominantly *Lactobacillus* bacteria. When a more mixed flora is present (Gram-positive cocci, small Gram-negative rods, curved Gram-variable rods) and *Lactobacillus* absent or present in low numbers, the smear is interpreted as consistent with BV.

C. Neither cultures nor PCR assays are recommended for detection of BV-associated bacteria. These generally involve considerable expense and at the present time, no added value to the standard clinical means of diagnosis (Amsel) has been established.

D. Rapid diagnostic modalities include tests that detect abnormal pH, high levels of

amines, and/or *G. vaginalis*.

V. Treatment

A. Recommended regimens (non-pregnant patients):

1. Metronidazole 500 mg po bid x 7 days
2. Metronidazole gel 0.75% one applicator (5 g) qhs x 5 days
3. Clindamycin cream 2% one applicator (5 g) intravaginally qhs x 7 days

B. Alternative regimens (non-pregnant patients):

1. Tinidazole 1 gm orally once daily x 5 days
2. Tinidazole 2 gm orally once daily x 3 days
3. Clindamycin 300 mg po bid x 7 days
4. Clindamycin ovules 100 mg intravaginally at bedtime x 3 days

   Tinidazole has the advantages over metronidazole of easier dosing and fewer side effects, but is more expensive.

C. Recommended regimens for treatment in pregnancy are listed below. Pregnant women with symptomatic disease should be treated. There is no evidence of teratogenicity from metronidazole, even when used in first trimester. Some experts suggest that treating early in pregnancy may actually be important in preventing adverse outcome. Due to concern for potential upper genital tract infection, oral therapy is generally preferred. The use of clindamycin intravaginal cream in pregnant women is not recommended due to increased risk of premature delivery.

1. Metronidazole 500 mg po bid x 7 days
2. Metronidazole 250 mg po tid x 7 days
3. Clindamycin 300 mg po bid x 7 days

D. Screening and treatment in asymptomatic patients:

1. Therapy is not recommended for male partners of women with BV. Female partners of women with BV should be examined and treated if BV is present.

2. Therapy may not be necessary for asymptomatic women with BV. Exceptions include:

   a) Asymptomatic patients with BV who are to undergo surgical abortion

should be treated. BV has been associated with endometritis, PID, or vaginal cuff cellulitis in women undergoing ambulatory invasive procedures (endometrial biopsy, hysteroscopy, IUD insertions) and women scheduled for vaginal or abdominal surgery. Although data are insufficient to recommend treatment of asymptomatic patients prior to procedures other than surgical abortion and hysterectomy (see CDC treatment guidelines), many providers elect to treat asymptomatic BV before any procedure involving the upper genital tract.

b) Although an association between BV and premature delivery has been demonstrated in a number of studies, treatment trials have not consistently demonstrated a significant reduction in pre-term delivery in all pregnant women. The U.S. Preventive Services Task Force recommends against routine screening of asymptomatic women for BV in pregnancy. Current evidence is also insufficient to assess the impact of screening for BV in asymptomatic women at high risk for pre-term delivery (i.e., those who have previously delivered a premature infant) at the first prenatal visit.

E. Drugs **not** recommended for the treatment of BV include: ampicillin, erythromycin, iodine, dienestrol cream, tetracycline/doxycycline, triple sulfa, and ciprofloxacin.

F. Recurrence:

1. Very frequent: 80% recurrence rate within 7 months in one study.
2. Recurrence may be a result of persistence of BV-associated organisms and/or failure of *Lactobacillus* flora to recolonize.
3. No data to support yogurt therapy or exogenous oral *Lactobacillus* treatment.
4. Under study: vaginal suppositories containing human *Lactobacillus* strains.

VI. Prevention

A. Partner management: several studies of empiric treatment of male sex partners have not shown effectiveness in reducing rates of BV recurrence, and such treatment is not recommended. However, in one study, women whose male partners regularly used condoms had significantly lower rates of recurrent BV. The value of other interventions, such as avoiding receptive oral sex or treating female sex partners, has not been established.

B. Patient counseling and education:

1. Avoid douching, which can eliminate protective lactobacilli.
2. Education re: normal vs. abnormal discharge and signs warranting evaluation.
3. Consider trial of condom use by male partners if BV is recurrent

VAGINITIS*: DIFFERENTIATING Bacterial Vaginosis, Candidiasis, and Trichomoniasis

	Normal	Bacterial Vaginosis	Candidiasis	Trichomoniasis
Symptoms/ Presentation		Odor, discharge, itch	Itch, discomfort, dysuria, thick discharge	Itch, discharge, 50% asymptomatic
Vaginal Discharge	Clear to white	Homogenous, adherent, thin, milky-white; malodorous "foul fishy"	Thick, clumpy, white "cottage cheese"	Frothy, gray or yellow-green; malodorous
Clinical Findings			Inflammation and erythema	Cervical petechiae "strawberry cervix"
Vaginal pH	3.8-4.2	>4.5	Usually ≤4.5	>4.5
KOH "whiff test"	Negative	Positive	Negative	Often positive
NaCl Wet Mount	Lacto-bacilli	Clue cells (≥20%), no/few WBCs	Few WBCs	Motile flagellated protozoa, many WBCs
KOH Wet Mount			Pseudohyphae or spores if non-*albicans* species	

*** Note that this table includes only three of the more common causes of vaginitis. Many women with vulvovaginal complaints have other conditions, including irritant or chemical vulvovaginitis, desquamative interstitial vaginitis, and lichen planus or lichen simplex chronicus. Genital herpes should also be considered**

in the differential when women complain of recurrent pruritic lesions or vulvar fissures.

VII. References

1. Krieger J, et al. Clinical manifestations of trichomoniasis in men. Ann Int Med 1993; 118:844-849.

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