Noncandidal vaginitis: a comprehensive approach to diagnosis and management

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Vaginitis is one of the most common causes of patient visits to gynecologists, primary care providers, and urgent care centers. However, many women leave without a clear diagnosis or experience recurrent symptoms despite treatment. The 3 most common etiologies of vaginitis are trichomonas, bacterial vaginosis, and vulvovaginal candidiasis, which account for an estimated 70% of cases. The remaining 30% may be related to other causes of vaginitis, including atrophic vaginitis, desquamative inflammatory vaginitis, and vaginal erosive disease. The purpose of this review is to describe the noncandidal causes of acute and recurrent vaginitis, with the goal of improving the likelihood of accurate diagnosis as well as efficient and effective therapy. We excluded candidal vaginitis from our review because there was a recently published review on this topic in the Journal. The clinical presentation and evaluation of patients with symptoms of vaginitis can be triaged into 1 of 2 diagnostic pathways: noninflammatory and inflammatory vaginitis. The most common noninflammatory cause is bacterial vaginosis. Features such as irritation, purulent discharge, and the presence of polymorphonuclear neutrophils are more suggestive of an inflammatory process. Trichomoniasis is the most common cause of inflammatory vaginitis. Other well-described forms of inflammatory vaginitis include atrophic vaginitis, desquamative inflammatory vaginitis, and erosive disease. We present a review of the pathogenesis, symptoms, examination findings, diagnostic testing, and treatment for each of these causes of noncandidal vaginitis.

Key words: atrophic vaginitis, bacterial vaginosis, desquamative inflammatory vaginitis, infection, inflammatory, recurrent bacterial vaginosis, treatment, trichomoniasis, vaginitis

Symptoms of vaginitis (discharge, irritation, odor, itching, swelling, etc) are among the most frequent causes of patient visits to gynecologists, primary care providers and urgent care centers[1,2] and may account for up to 10 million office visits per year. It is estimated that 7−72% of women leave the clinician's office without a clear diagnosis or return within a short interval because of a recurrence of symptoms.[1,3,4]

Many practitioners utilize a limited spectrum of common potential differential diagnoses for women who suffer from symptoms of vaginitis, applying only the 3 most likely etiologies, (trichomonas, bacterial vaginosis [BV], and vulvovaginal candidiasis).[5] While these 3 conditions cause of up to 70% of abnormal vaginal symptoms, they are not the only causes of vaginitis.[1,4] The differential diagnosis should be expanded, especially in women with persistent symptoms.

In 1954, Franken first described a noninfectious, purulent form of vaginitis in a young girl who responded well to estrogen therapy. Since that time, several forms of noninfectious, exudative and inflammatory types of vaginitis have been described.

The purpose of this review is to provide a diagnostic algorithm based on examination findings consistent with inflammation. We also offer an expanded description of the causes of incident and recurrent vaginitis with the goal of improving diagnostic accuracy and the initiation of appropriate therapy. Because a review of candidiasis was recently published by Sobel,[6] our review will focus on BV, trichomoniasis, and other causes of acute and recurrent vaginal symptoms.

A diagnostic aid: inflammatory or noninflammatory?

The clinical presentation and evaluation of patients with vaginal symptoms can be categorized into 1 of 2 diagnostic pathways: noninflammatory or inflammatory (see Figure 1). Inflammatory refers to the presence of polymorphonuclear neutrophils (PMNs) on microscopic examination of vaginal discharge or physical findings such as erythema and edema. Noninflammatory conditions consist of an absence of the aforementioned symptoms and absence of PMNs on microscopy in a woman with vaginal complaints such as odor or abnormal discharge.

The inflammatory forms of vaginitis share a common spectrum of clinical presentation but vary in etiology and treatment responsiveness. The most common forms of inflammatory vaginitis are trichomoniasis infection and candidiasis.

Other well-described but less common forms of inflammatory vaginitis are hypoestrogenic or atrophic vaginitis, desquamative inflammatory vaginitis (DIV), and multimucosal erosive diseases (eg, erosive lichen planus, pemphigus vulgaris, and cicatricial [mucous membrane] pemphigoid).[4,7,8] The multimucosal erosive diseases are autoimmune disorders that result in erosions or blisters that affect mucosal surfaces such as the mouth, esophagus,

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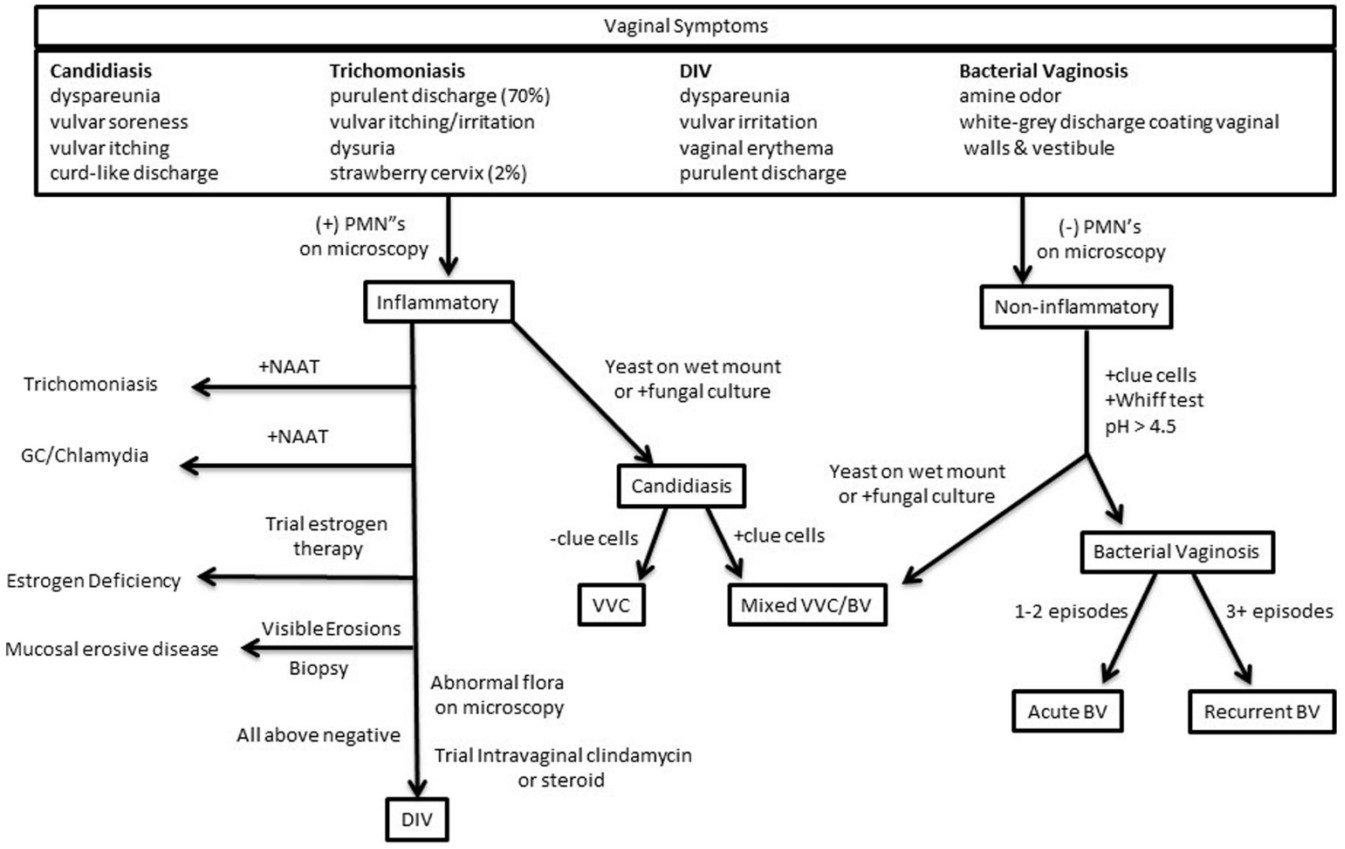
FIGURE 1
A diagnostic aid for the evaluation of vaginal symptoms



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BV, bacterial vaginosis; *DIV*, desquamative inflammatory vaginosis; *GC*, gonorrhea; *NAAT*, nucleic acid amplification test; *PMN*, polymorphonuclear neutrophils; *VVC*, vulvovaginal candidiasis.

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eyelid, vagina, and vulva. BV is the most common noninflammatory vaginal condition, but certain forms of vulvovaginal candidiasis or mixed infections can also be noninflammatory.[5,9]

Bacterial vaginosis

Forty to fifty percent of women with vaginal complaints will be diagnosed with BV, making it the most prevalent cause of vaginal symptoms in women nationally.[10] Data from the National Health and Nutrition Survey indicate up to 1 in 3 women aged 14−49 years are affected at any time.[10] BV is more prevalent in African-American and/or American-Hispanic women than in whites.[1−4,10] After adjusting for age, education, and poverty, BV is still 3 times more prevalent in these racial/ethnic groups than in whites.[10] Because the social construct of race encompasses multiple factors including host, environment, and behavior, interpreting the influence of race on incidence, prevalence, and transmission of BV is complex.[11]

While sexual transmission has been implicated in the development of recurrent BV,[12,13] the data are mixed. Multiple epidemiological studies show an increased risk of up to 60% for BV acquisition in women with multiple or new sexual partners and a protective effect from both incident and recurrent BV in women who report condom usage.[13] Other data are less supportive of sexual transmission, such as finding BV in sexually naïve individuals.[14]

The etiology, pathogenesis, and events that lead to the development of BV are incompletely understood.[12,15] The protective effects of lactobacilli species within the vaginal microbial environment are well known. Several species of lactobacilli including *Lactobacillus gasseri, L crispatus, L iners* (a non−H_2O_2-producing species), and *L jensenii* secrete substances such as lactic acid and/or hydrogen peroxide to provide bactericidal activity and an acidic pH.[16]

Schwebke theorizes that *Gardnerella vaginalis* acts as a keystone pathogen that alters the vaginal microbiome, causing a reduction of lactobacilli and an increase in BV-associated bacteria. Nonpathogenic, commensal bacteria, present in the vaginal microbiome, then aid the development of the well-characterized dysbiosis: a polymicrobial mix of facultative anaerobes that aid in the transmission and persistence of BV.[17]

Despite the prevalence of *G. vaginalis* in women with BV (98−100%), it is also

present in women without BV (55%).[18] It has been shown that genetic heterogeneity exists within subtypes of *Gardnerella*, and the species can be further classified into 4 clades: only clades 1 and 3 are associated with BV.[4,19] Sequencing studies have shown that bacterial diversity beyond *G vaginalis* subtypes is also important in the pathogenesis of BV.

Women with BV have an average of 12.6 bacterial species, while women without BV have an average of 3.3 species.[19] BV is also considered a biofilm infection, which consists of a complex polysaccharide matrix that adheres to epithelial cells in the genital tract of women. *G vaginalis* is the predominant species, while other species enhance the biofilm matrix created by *G vaginalis*.[20,21]

The bacterial biofilm provides a biological explanation for treatment resistance and recurrence because antibiotic therapy alone is unable to disrupt the BV biofilm.[20] Further studies describing the bacterial biofilm point to only specific strains of Gardnerella being capable of biofilm creation, potentially explaining why not all BV-infected women will develop recurrent disease.[22] Therefore, disruption of the acidic lactobacilli-rich environment, proliferation, and diversification of anaerobic bacteria and the development of an adherent polymicrobial biofilm seems to underlie the pathogenesis of BV and provides a biological explanation for recurrence or persistence of infection.

Symptoms

The typical presentation of patients with BV includes foul-smelling or fishy vaginal discharge. The complaint of malodor is highly associated with BV[1,10] and may be exacerbated by conditions that elevate the vaginal pH such as sexual intercourse with semen deposition or the presence of blood. Itching and burning are unlikely in BV but may be present when discharge causes secondary vulvar irritation.[23] If vaginal or vulvar irritation is present, the clinician should have a strong suspicion for mixed infection with candida or other inflammatory processes.

Examination findings

A gray-white, thin, or watery discharge, visible in the vagina or at the vulvar vestibule, is most commonly described by examining practitioners. In contrast, findings of purulent or frothy discharge are uncommon.[1] Because BV is not an inflammatory process, the presence of erythema, fissuring, or bleeding likely indicates mixed infection or an alternative etiology.

Diagnostic testing

Classically, at least 3 of the 4 components of Amsel's criteria will be present: a thin, homogenous white discharge, an increased pH >4.5 of the vaginal fluid, release of an amine odor upon application of potassium hydroxide (positive whiff test), and >20% clue cells on saline microscopy.[24,25] However, some studies have shown that 2 of the 4 Amsel's criteria may perform just as well as 3 of 4.[25]

Traditional diagnostic testing with Amsel's criteria and using saline microscopy allows physicians to make a more immediate diagnosis of BV and is relatively low cost. The simplicity, low cost, and immediate utility of obtaining a vaginal pH make it a critical part of the clinical evaluation. With saline microscopy, the absence of lactobacilli and/or presence of corkscrew bacilli are associated with BV.[25,26] BV is not an inflammatory process and the presence of PMNs in the vaginal saline microscopy decreases the likelihood of BV and should raise suspicion for an alternate diagnosis or a mixed infection.[1,5]

The Nugent score, which utilizes a standardized morphologic scoring system ranging from 0 to 10, with ≥7 being diagnostic for BV of Gram-stained specimens of vaginal smears, remains the gold standard for BV diagnosis in the research setting but is typically not used in clinical settings. Given the association of BV with other sexually transmitted infections (STIs), HIV and STI testing should be considered in women diagnosed with BV.[24,26]

Molecular testing can improve the accuracy of diagnosing BV and trichomoniasis and may be useful in cases that have not responded to therapy or when microscopy is not available (Table 1).[18] Molecular diagnostic tests include 2 direct DNA probe assays (bacterial vaginosis/vaginitis panel and the *Affirm VPIII* assay) and 4 nucleic amplification tests (Nu Swab, Sure Swab, BD Max vaginal panel, and the BV Panel). These testing modalities are substantially more costly than saline microscopy. The benefit of molecular technology over classic diagnostic methods is not yet clear when microscopy is available, and governing bodies such as the American College of Obstetricians and Gynecologists or the Infectious Diseases Society for Obstetrics and Gynecology have not issued official recommendations for their use.[18]

Vaginal culture for bacteria is not recommended in the evaluation of BV and should be actively discouraged. Cytologic testing is not reliable. If a Papanicolaou smear mentions a shift in flora to predominantly coccobacilli suggesting BV, a symptomatic patient should undergo standard diagnostic testing and treatment as appropriate.

Treatment

First-line therapy for acute BV is a 7 day course of oral metronidazole, a 7 day course of intravaginal clindamycin cream, or a 5 day course of intravaginal metronidazole gel (Table 2).[24] Clindamycin vaginal ovules have been shown to be as effective as the 7 day course of clindamycin.[27] A 2009 Cochrane review showed equivalent effectiveness for oral clindamycin and recommended topical metronidazole and clindamycin regimens.[28]

In 2018, secnidazole was approved for the treatment of incident BV. Secnidazole has a much longer half-life than metronidazole, and the single-dose regimen was found to be at least as effective as the 7 day oral metronidazole regimen.[29] It should be mentioned that although single-dose secnidazole performed better than placebo when fewer than 3 episodes of BV were reported, it was not better than placebo in individuals who reported 4 or more episodes of BV.[29] The US Centers for Disease Control and Prevention (CDC) has suggested that women being treated for BV should refrain from sexual

TABLE 1
Sensitivity and specificity of diagnostic tests for bacterial vaginosis[18]

Test	Sensitivity (%)	Specificity (%)	Manufacturer
Wet mount/Amsel's criteria	30–70	94–99	
Direct probe assays			
BV/vaginitis panel[a,b]			Quest Diagnostics, Secaucus, NJ
Affirm VPIII[b]	94	81	Becton Dickinson, Sparks, MD
Nucleic amplification assays			
NuSwab	92	94	Laboratory Corp of America, Burlington, NC
SureSwab[a]			Quest Diagnostics, Secaucus, NJ
BD MAX[b]	90.5	85.5	Becton Dickinson, Sparks, MD
BV Panel[b]	92	95	Medical Diagnostic Labs, Hamilton Township, NJ

[a] Sensitivity and specificity are not published; [b] Food and Drug Administration approved.

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activity or use condoms consistently and correctly.[24]

Greater than 50% of women will have a repeat episode of BV within 1 year of treatment with 7 days of metronidazole therapy.[30,31] Recurrent BV, defined as 3 or more symptomatic episodes within 1 year,[32] may be associated with multiple treatment failures over periods of several years or months. Failure of symptom resolution during appropriate antibiotic treatment should raise suspicion for mixed infection and the need for further evaluation. One of the CDC-suggested treatment strategies for recurrent BV includes 0.75% metronidazole intravaginal gel daily for 10 days followed by twice-weekly intravaginal metronidazole for 4–6 months (Table 2).[24,33] The CDC cautions that recurrence may be common after completion of therapy.

With the realization that a bacterial biofilm is implicated in recurrent or persistent BV, disruption of the bacterial biofilm has become the target of in vitro studies.[35,36] Boric acid combined with nitroimidazole therapy has been used to treat recurrent BV and has been shown to disrupt the biofilms of both BV and candida.[34,37] A novel antiinfective, TOL-463, is a boric acid-based vaginal gel enhanced with ethylenediaminetetraacetic acid.

A recent phase 2 clinical trial found that boric acid and ethylenediaminetetraacetic acid administered as a vaginal insert showed successful treatment of BV (59% at test of cure).[38] This is a similar cure rate to findings from other studies using imidazole therapy and suggests a role for biofilm disruptors as novel or adjunctive therapies in the treatment of BV. A single pilot study that evaluated the effectiveness of the addition of boric acid to standard nitroimidazole suppressive therapy in cases of recurrent BV showed lower rates of breakthrough infections (12% vs 25%) while on suppression and lower late-recurrence rates (45% at 32 weeks vs 66% at 28 weeks) than in previous studies utilizing single-agent nitroimidazole therapy.[34,38] While these results are promising, larger, prospective, comparative studies are recommended.

BV is implicated in the pathogenesis of several serious disease processes such as preterm labor, preterm rupture of membranes, postpartum infections, persistence of HPV, increased HIV acquisition, pelvic inflammatory disease,

TABLE 2
Treatment of acute and recurrent bacterial vaginosis[24,27,29,33]

Acute	Recurrent[a]
Oral metronidazole 500 mg twice daily × 7 days	Intravaginal metronidazole daily × 10 days, then twice weekly for 4–6 months[33]
Oral clindamycin 500 mg twice daily × 7 days	
Oral secnidazole granules 2 g, single dose	
Intravaginal metronidazole 0.75% daily × 7 days	Oral tinidazole or metronidazole twice daily × 7 days, then intravaginal boric acid 600 mg daily × 21 days, then intravaginal metronidazole twice weekly for 4–6 months.[b]
Intravaginal clindamycin 2% cream daily × 7 days	
Intravaginal clindamycin ovules daily × 3 days	

[a] Recurrent bacterial vaginosis is defined as 3 or more episodes in 1 year; [b] Based on limited data.[34]

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and tuboovarian abscess formation.[10,39] Multiple studies have also illustrated increased acquisition of *Trichomonas vaginalis*, *Neisseria gonorrhea*, and *Chlamydia trachomatis* in women with BV.[40–43] Because BV is associated with adverse pregnancy outcomes, all symptomatic pregnant women should be treated for BV either with the oral or topical regimens of metronidazole, which have no demonstrated teratogenic or mutagenic effects in newborns.[24]

Patients may ask providers about the use of probiotics for BV. Common naturopathic recommendations include the alteration of diet to include the ingestion of yogurt, placing yogurt or garlic in the vagina, or oral supplementation with probiotic species. A review and meta-analysis from 2017 found no significant difference in the efficacy of metronidazole combined with probiotics compared with metronidazole alone for the treatment of BV.[44] At this time, there is not sufficient evidence to support the use of probiotics for BV, and they are not recommended in the CDC treatment guidelines.

The role of male partner treatment is controversial because well-designed and robust studies are lacking.[45] No trials have evaluated female partner treatment.[42,45] Therefore, treatment of male or female partners is not currently recommended.

Data regarding the role of circumcision in the transmission of BV are conflicting and do not include studies evaluating the role of circumcision and recurrent infection in female partners.[46,47,48] However, it has been shown that the penile microbiome in uncircumcised males when compared with circumcised males is more likely to favor BV-type microorganisms.[46,47]

Condom use has been shown to decrease acquisition of incident BV.[12,13,48] There is also a suggestion that the use of condoms may reduce the incidence of recurrent BV,[48] but it is difficult to draw practical conclusions from these data because a specific condom use strategy has not been studied. Therefore, specific recommendations regarding the duration of condom use in women affected by recurrent BV with single or multiple partners are not available.

Inflammatory causes

The most common cause of inflammatory vaginitis is trichomoniasis, diagnosed in 5–20% of patients reporting abnormal vaginal symptoms.[1] Vulvovaginal candidiasis is also a very common cause of inflammatory vaginitis, comprising up to 30% of diagnoses in women with vaginal complaints.[6,9] Inflammatory cells may also be found in vaginal secretions from women with cervicitis or viral infections such as human papillomavirus or herpes simplex virus. Noninfectious forms of inflammatory vaginitis are less common but include atrophic or hypoestrogenic vaginitis, DIV, and mucosal erosive diseases.

Trichomoniasis

Trichomonas infection affects 3.7 million persons in the United States.[24] The prevalence in women 40 years old and older is 11% overall and is more common in women diagnosed with other STIs including HIV. African-American women are disproportionately affected with a prevalence of 13% compared with 1.8% of white American women.[3,24] Trichomonas infection is associated with adverse health outcomes such as increased HIV transmission, preterm birth, and an increased incidence of pelvic inflammatory disease in HIV-infected women.[24]

Symptoms

Up to 70% of women infected with trichomonas are asymptomatic.[24] Symptoms reported by women with trichomonas include increased malodorous discharge that is yellow, green, or gray in color. Itching, burning, painful urination, and vulvar irritation may also be reported.[24,49]

Examination findings

Clinical descriptors of discharge include a yellow-green, frothy discharge with evidence of vaginal or cervical inflammation. Cervical inspection may reveal a strawberry or punctate appearance.

Diagnostic testing

The vaginal pH in women with trichomonas infection is usually above 4.5. Saline microscopy will show many PMNs and motile trichomonads, which distinguishes trichomonal infection on microscopy from other forms of inflammatory vaginitis. Microscopy, however, has a sensitivity of only 51–65%[24] for the diagnosis of trichomoniasis. Nucleic acid amplification tests detect 3–5 times more trichomonal infections than wet mount and have a sensitivity and specificity of 95–100%, with 100% concordance between urine and vaginal samples. Thus, nucleic acid amplification testing is currently the gold standard.[24] The CDC recommends retesting within 3 months for women diagnosed with a trichomonal infection, regardless of partner treatment status, because of high rates of recurrence (17%). Testing can be performed as soon as 28 days after treatment.[50]

Other testing options include a 10 minute point-of-care test to detect antigens via dipstick with a sensitivity of 82–95% and specificity of 97–100%.[24] A 45 minute DNA hybridization probe test evaluates for *T vaginalis*, *G vaginalis*, and *Candida albicans* with a sensitivity of 63% and specificity of 99.9%. Neither liquid-based nor conventional Papanicolaou tests are considered diagnostic tests for trichomonal infection because false-positive and false-negative results have been reported.[24]

Treatment

Current CDC-recommended first-line therapy is oral treatment with either metronidazole or tinidazole, both as a single dose (Table 3). However, a recent multicenter randomized control trial found the 7 day course of metronidazole to be superior to single-dose metronidazole for the treatment of trichomonas (11% vs 19% trichomonas positive at 4 weeks after therapy).[51] Hence, 7 days of therapy may be indicated. Tinidazole is slightly more effective than metronidazole but is considerably more expensive than generic metronidazole. Cure rates with standard tinidazole treatment

TABLE 3
Treatment of acute and persistent trichomonas vaginitis[24,51]

Acute	Persistent[a]
Oral tinidazole 1000 mg daily × 7 days	Oral tinidazole or metronidazole 1000 mg twice daily × 7 days; test for resistance to tinidazole and metronidazole after 3–4 courses
Oral metronidazole 500 mg twice daily × 7 days	

[a] Reexposure is the most common cause of recurrence. If reexposure has been ruled out, treatment for persistent infection may be initiated.

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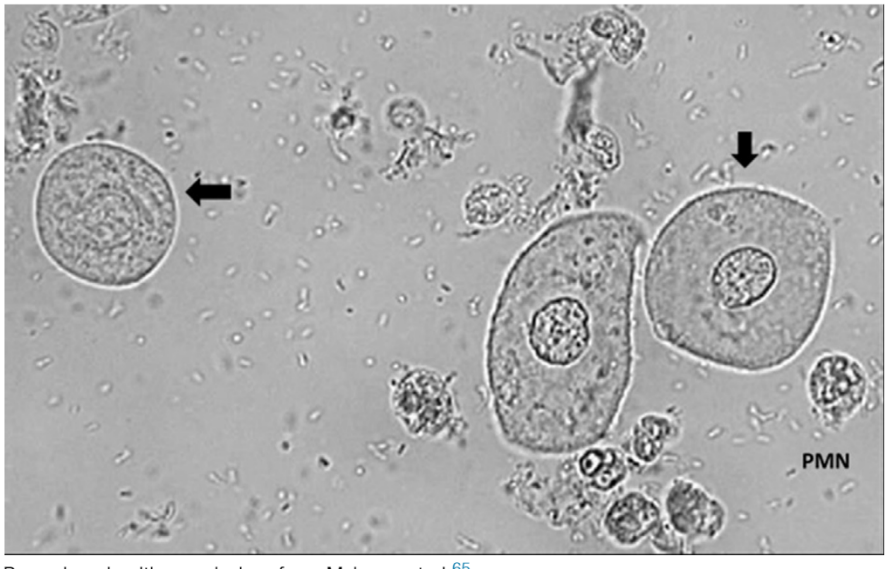
regimens range from 92% to 100%, while metronidazole regimen cure rates range from 84% to 98%.[24]

T vaginalis has been isolated from multiple sources within the genital tract including the periuretheral glands, bladder, and Bartholin's glands.[18] This likely explains the increased effectiveness of tinidazole, which has a longer half-life and reaches higher concentrations with genital tract tissues. Metronidazole gel does not reach effective treatment levels within the vaginal mucosa and periurethral glands and is not recommended for treatment.[24] Clinicians should advise women with trichomonal infection to abstain from sex until they and their sex partners are treated. Testing for other STIs should be performed in persons infected with *T vaginalis*.[24]

Imidazole resistance is uncommon but emerging.[52] Resistance to tinidazole is approximately 1%, while metronidazole resistance is 4–10%.[52,53] Most cases of recurrence are due to reexposure and not a persistent infection. However, if reexposure has been ruled out, treatment with either metronidazole or tinidazole may be used. Testing for imidazole resistant trichomonas should be pursued with the CDC if the high-dose regimen has failed after 2–3 courses (Table 3).[24,54]

Treatment with metronidazole for pregnant women with symptomatic trichomoniasis is recommended. *T vaginalis* is associated with adverse outcomes such as premature rupture of membranes, preterm delivery, and delivery of a low-birthweight infant[55]; however, no significant difference in perinatal morbidity after treatment has been demonstrated.

FIGURE 2
Parabasal cells (arrows) and PMNs, ×200 magnification



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Atrophic vaginitis

Hypoestrogenic vaginitis is caused by diminished serum estrogen levels that result in a loss of elasticity, dryness, and thinning of the mucosa of the lower genital tract.[49] Atrophic vaginitis is more common in postmenopausal women and symptoms such as vaginal dryness are reported in up to 47% of postmenopausal women. Similar symptoms are reported as bothersome in 3% of reproductive-age women.[56] Causes for urogenital atrophy in premenopausal women include postpartum estrogen reduction, oophorectomy, premature ovulatory failure, antiestrogenic medications such as tamoxifen, medroxyprogesterone acetate, hypothalamic amenorrhea, gonadotropin-releasing hormone agonists, and antagonists, and occasionally oral contraceptive pills.

Symptoms and examination findings

Symptoms include dyspareunia, dryness, itching, or burning.[57] Atrophic vaginitis may share similar findings with other forms of inflammatory vaginitis such as purulent discharge, vaginal erythema, and petechia.[5,7] However, along with patient age or estrogen status, visualized atrophy on examination and evidence of parabasal cells on saline microscopy (Figure 2)[49] may help to distinguish this form of inflammatory vaginitis from the others. Typically, vaginal pH will be elevated above 4.5, whiff test will be negative, and parabasal cells may be noted, indicating a thin vaginal mucosa with fewer layers.[57]

Treatment

The treatment of estrogen deficiency is both diagnostic and therapeutic. Topical estrogen should result in a relief of symptoms in women with atrophic vaginitis and can be continued as needed

to control symptoms.[32] Nonhormonal options include vaginal moisturizers and lubricants.[57] Other Food and Drug Administration—approved treatments for urogenital atrophy include systemic medications such as ospemifene[58] and intravaginal dehydroepiandrosterone (Prasterone).[59]

Desquamative inflammatory vaginitis

DIV is a chronic inflammatory process of the vagina and vestibule. It is not easily distinguishable from other forms of inflammatory vaginitis and is often a diagnosis of exclusion. It has been suggested that DIV is more common in perimenopausal and white women.[60] The incidence of DIV in vulvovaginal clinics has been estimated between 0.8% and 4.3%.[5] However, knowledge of DIV as a form of vaginitis by clinicians is low, and the true incidence, prevalence, and demographic characteristics of women with DIV are unknown.[5,7,60]

Symptoms and examination findings

The clinical features of DIV have been described as a purulent vaginitis with increased pH and inflammatory cells composed mostly of PMNs on saline microscopy.[7] Diagnosis of DIV also requires an increase in parabasal epithelial cells on microscopy and is thus difficult to distinguish from atrophic vaginitis in postmenopausal women. It is possible that there is a spectrum of DIV, ranging from mild to severe in terms of symptoms, physical examination findings, and microscopic detail.[61] Diagnosis of DIV requires the exclusion of infectious and estrogen-related causes before a diagnosis can be made and is therefore a diagnosis of exclusion.

Treatment

After alternative causes have been ruled out, patients with suspected DIV should receive a trial of intravaginal steroid or clindamycin, as described by Sobel.[62] Clindamycin 2% vaginal cream every other night or 10% hydrocortisone vaginal suppository or hydrocortisone 25 mg suppository every night for 14 days has been shown to be effective, but prolonged therapy of 4—8 weeks is often necessary for cure.[7,62]

Vaginal erosive disorders

Patients with erosive disease may be identified via a careful physical examination. A vulvar biopsy may be useful, although vulvar biopsies commonly have nonspecific inflammatory findings and can be slow to heal. Diseases that may present with vaginal erosions include erosive lichen planus, pemphigus vulgaris, pemphigoid, and cicatricial pemphigoid. The true prevalence of vaginal erosive disorders is unknown. Erosive lichen planus may be the most common, but there are no data available regarding the prevalence of vaginal lichen planus. The only available data come from a small study of oral lichen planus, which demonstrated vaginal lichen planus in 51% of patients.[63] Vulvar vestibulitis (adenitis) may also be present with vaginal symptoms, especially in women with persistent complaints; clinicians should consider this entity in their differential diagnosis.

Symptoms and examination findings

Erosive vaginal disease may present as shallow isolated and erythematous erosions of the vagina or labia minora along with purulent discharge. Non—lichen planus diseases may present with a combination of bullae and erosions. Mononuclear inflammatory cells on microscopy further suggest erosive disease.[32] In advanced stages, vaginal stenosis or other architectural distortions may exist. The entire vagina may appear friable and serosanguinous drainage may be present.[64]

Treatment

When an erosive disease is suspected, biopsy and histopathological evaluation are warranted.[7] Limited evidence exists regarding treatment of erosive vaginal diseases. Referral to clinicians experienced in managing these disorders may be the most beneficial.

Conclusions

The difficulty in reaching an appropriate diagnosis in women presenting with vaginal symptoms is due, in part, to the diverse etiologies that share common presentations and clinical findings. Creating proper clinical awareness and educational models is therefore challenging and may explain why many women are not diagnosed and treated appropriately after an initial evaluation. Additional epidemiological and clinical data are needed regarding the less common causes of vaginitis/vaginosis and in women with recurrent vaginal symptoms. ■

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