Vulvovaginitis in Healthy Women

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Inflammation of the vagina is extremely common and results in millions of visits to practitioners' offices worldwide. Vaginitis is frequently, but not invariably, accompanied by inflammatory changes in the contiguous cervix, vestibule, and vulva depending on the etiologic agent. Even more common than vaginitis per se are vulvovaginal symptoms that are often short-lived and self-limited without objective evidence of inflammation. The incidence of vulvovaginal symptoms such as pruritus, irritation, soreness, discharge, or dyspareunia is unknown; however, background mild symptoms are extremely common. Unfortunately, symptoms are nonspecific; therefore, both self-diagnosis and diagnosis by practitioners without confirmation by basic laboratory testing is unreliable. Management of vaginitis remains mired in empiricism, bolstered by the widely held view that vaginitis is never life-threatening and empiric therapy is harmless. The introduction of over-the-counter (OTC) antimycotics was enthusiastically embraced by consumers and practitioners alike, with little concern for the side effects, potential for abuse, or other consequences of OTC vulvovaginal product use. Regrettably, this situation could have been avoided by the widespread availability of new diagnostic tests, easily achievable in this day and age of technological sophistication but a reality that failed to materialize in the absence of practitioner demand, consumer ignorance, and ulterior commercial interests.

Although frequently the result of infectious microorganisms, vulvovaginitis may be the result of numerous, less easily recognized, noninfectious etiologies (Table 1). This review is directed at advances in our understanding of the epidemiology, pathogenesis, and treatment of vaginitis. Currently, there is no place in North America for a syndromic, empiric approach currently advocated in developing countries.

REPRINTS

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Causes of Vaginitis in Adult Women

Infectious Vaginitis
- Bacterial vaginosis (40% to 50%)
- Vulvovaginal candidiasis (20% to 25%)
- Trichomononal vaginitis (15 to 20)

Less common
- Atrophic vaginitis with 2° bacterial infection
- Foreign body with 2° bacterial infection
- Desquamative inflammatory vaginitis (clindamycin-responsive)
- Streptococcal vaginitis (group A)
- Ulcerative vaginitis associated with Staphylococcus aureus and toxic shock syndrome
- Idiopathic vulvovaginal ulceration associated with HIV

Noninfectious Vaginitis
- Chemical/irritant
- Allergic, hypersensitivity, and contact dermatitis (lichen simplex)
- Traumatic
- Atrophic vaginitis
- Postpuerperal atrophic vaginitis
- Desquamative inflammatory vaginitis (steroid-responsive)
- Erosive lichen planus
- Collagen vascular disease, Behçets, pemphigus syndromes
- Idiopathic

Candida Vulvovaginitis (VVC)

Information on the incidence and prevalence of VVC is currently incomplete because VVC is a non-reportable disease. Prevalence estimates have relied mainly on self-reported history of physician diagnosis. VVC is routinely diagnosed without the benefit of microscopy or culture and as many as 50% of the women diagnosed with VVC may have other conditions. Published epidemiologic data have been distorted by patient selection and referral biases, and widespread use of OTC antimycotics may seriously impair future epidemiologic studies.

By the age of 25 years, 50% of all college women will have experienced at least one physician-diagnosed episode of VVC, which is rare prior to menarche. In other age populations, at least one episode of VVC has been estimated in up to 75% of premenopausal women. While extremely rare prior to menarche, the annual incidence increases drastically toward the end of the second decade of life, peaking over the following two decades. VVC appears reduced in frequency in postmenopausal women, although the incidence in this subpopulation remains unstudied, as is the permissive role of estrogen replacement therapy. Although widely assumed that VVC is more common and difficult to eradicate during pregnancy, no recent confirmatory studies have been performed to prove this theory.

Pathogenesis. Numerous worldwide studies indicate that Candida albicans is responsible for 80% to 94% of episodes of VVC. Several investigators have suggested a shift of yeast pathogens with increased frequency of non-albicans Candida species, particularly C. glabrata. Only a few of these studies have provided data to support this claim. Possible reasons for the apparent increase in non-albicans Candida VVC include the increased use of OTC antimycotics, long-term maintenance suppressive prophylactic regimens incorporating systemic azoles, and the widespread tendency to short courses of oral or topical antifungals.

Sporadic attacks of VVC usually occur without an identifiable etiologic or precipitating factor. One explanation is recently administered antibiotic, both systemic or vaginal, however, the exact mechanisms for this association and its frequency have not been adequately studied. Only a minority of women with sporadic VVC report recent antimicrobials and only a minority of women taking antibiotics develop VVC. A prerequisite for VVC is vaginal colonization by Candida organisms and a readily suppressible vaginal flora; however, microbiologic data are lacking. An association between lack of or loss of vaginal lactobacilli, hydrogen peroxide production, and susceptibility to VVC has not been established in women who develop VVC while taking antibiotics, although the occurrence of VVC may vary by racial and behavioral factors. VVC was more common among black women than white women and was associated with initiation of sexual behavior in college women.

Several studies have documented increased risk of VVC in women who use oral contraceptives, and the risk may be higher with use of high estrogen-containing agents. It was recently discovered that sexual intercourse, with the use of a diaphragm or spermicide, in the preceding three days was found associated with a marked increase in the rate of Candida colonization. It has not been confirmed that there is any association between spermicide use and VVC; however, increase risk of infection is attributed to use of vaginal sponges and intrauterine devices.