References:


Infectious Vaginitis

Vaginitis is one of the most common reasons women seek medical attention and accounts for 10 million office visits annually (1). Although common in adult women, it is rare in children and prepubertal girls. The normal vaginal ecosystem is composed of a dynamic relationship between Lactobacillus spp., other normal flora and their metabolic by-products, glycogen, estrogen, and vaginal pH (2). Vaginitis occurs as a result of a shift in the vaginal flora. This shift can result either through the introduction of a pathogen or changes in the vaginal ecosystem that allow a pathogen to proliferate or push asymptomatic colonization into symptomatic disease. Despite the variety of causes of vaginitis, in approximately 90% of cases it is thought to occur secondary to a triad of infectious agents including Bacterial Vaginosis (BV), Candidiasis, and Trichomoniasis (2).

BACTERIAL VAGINOSIS

Bacterial vaginosis (BV) is a leading cause of abnormal vaginal discharge and odor and is the cause of most obstetric and gynecologic infections worldwide. Lactobacillus species are the predominant vaginal bacteria during women's reproductive years (3, 4). A large number of commercial bacterial species also inhabit the same niche in healthy women, but at three log-fold lower numbers than those of the Lactobacillus. In addition, vaginal microorganisms that are either associated with BV, or are likely etiological agents of BV, can often be detected at low numbers in the absence of BV.

Epidemiology

BV has been studied largely among self-selected women attending a variety of clinics. Since more than 50% of women with BV are asymptomatic, large numbers of women with the condition are not included in studies of this type. The prevalence of BV in participants is similar to those found among non-pregnant women with similar demographic characteristics. The prevalence of BV in the U.S. is highest among African American women and lowest among Asian American women; highest among women with multiple sexual partners with no history of heterosexual contact (2, 5). BV-associated microorganisms, including Lactobacillus, Mobiluncus, G. vaginalis, Megasphaera, and Bacteroides, are identified in 60% of women with BV. Furthermore, the same G. vaginalis isolates are isolated from heterosexual couples (6, 7).

Pathogenesis

BV does not fulfill Koch’s postulates, i.e., no single organism is thought to be the cause of this disease. While specific vaginal microorganisms are associated with BV, the current perception is that it results from a complex change in the microbial ecosystem of the vagina. BV is characterized by a substantive decrease in the level of the predominant Lactobacillus species and a concomitant multi-log fold increase in the number of facultative and anaerobic Gram-positive and Gram-negative bacteria and fungi (8). BV is associated with Lactobacillus spp., Mobiluncus spp., Ureaplasma urealyticum, and Mycoplasma hominis. (4, 8-10).

Recent studies of the vaginal flora using molecular techniques have identified several novel bacterial species that are more prevalent in women with BV compared to women without BV. The first of these species to be identified was Atojobium vaginase, which was initially described after isolation from the vaginal flora of a patient with BV. Pybus and Onderdonk (22) demonstrated a relationship between two of the microorganisms identified in this study, the Actinobacteria genus Mobiluncus and G. vaginalis. BV-associated microorganisms, including Lactobacillus, Mobiluncus, G. vaginalis, Megasphaera, and Bacteroides, are identified in 60% of women with BV. Furthermore, the same G. vaginalis isolates are isolated from heterosexual couples (6, 7).

The mechanism of how the vaginal flora changes so distinctly in BV is unknown. Lactic acid is produced by vaginal epithelial cells and vaginal microorganisms including Lactobacillus spp. Consequently, the vaginal pH in healthy women ranges between pH 3.8 to 4.2. This value is an important factor in the maintenance of the balance between the commensal and pathogenic microorganisms. Attachment and growth of lactobacilli is favored in the acidic vaginal environment while the attachment of BV-associated microorganisms is reduced (18, 19). Conversely, higher pH tends to displace lactobacilli from vaginal epithelial cell receptor sites and to maximize adherence of G. vaginalis. Risk factors for BV include douching, menstruation, and oral or vaginal perforation, all of which are believed to increase vaginal pH above the optimum for Lactobacillus-dominated microflora. The production of hydrogen peroxide by specific vaginal microorganisms is considered to play a role in maintaining the healthy vaginal microflora (19, 20). In vitro studies have demonstrated that hydrogen peroxide producing lactobacilli are capable of protecting against BV (21) and in vivo studies have demonstrated that BV-associated microorganisms are reduced (18, 19).

Cooperative interactions between BV-associated bacteria have been shown. In vitro studies by Pybus and Onderdonk (22) demonstrated a relationship between two of the predominant organisms in BV, G. vaginalis and Bacteroides spp. (22). Amino acids produced by G. vaginalis are utilized by Bacteroides to produce ammonia and short chain fatty acids.

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Diagnosis

Despite the commonly held belief that patients can intuitively self-diagnose yeast infections, Ferris et al., observed that vulvovaginal candidiasis is under-diagnosed in 76% of women who self-diagnosed yeast infection (36). Traditional diagnosis of Candida infection is slow and complicated and relies heavily on non-sterile examination, evaluation of KOH preparations, and vaginal culture. The ability to diagnose and identify candidiasis may be enhanced by the use of molecular techniques, such as Polymerase Chain Reaction (PCR).

Pathogenesis

Candida species have extraordinary phenotypic plasticity that allows them to adapt to the changing environment within its host. This ability enables Candida species to evade the host immune system, increases adhesion to human epithelial cells, and affects biofilm susceptibility (34). Candida species secrete extracellular hydrolytic enzymes, such as aspartyl proteinases, to disrupt host cell membranes in an attempt to facilitate adhesion and tissue invasion (35).

Risk factors for candidiasis include uncontrolled diabetes mellitus, recent antibiotic therapy, extremes of pH, increased hydration, pregnancy, and hormone replacement therapy. Symptoms typically include vulval pruritus, vulvovaginal irritation, dysuria, and dyspareunia. Dyspareunia may impair sexual intimacy, affect marital satisfaction, and may result in severe emotional distress.