
Vulvovaginal Candidiasis: The Role of Candida Fungi in Gynecological Infections – A Comprehensive Review

Roghayeh darghahi¹, Zahra Eftekhari Afshar², Arezoo ardforoosh³

1. Department of obstetrics and Gynecology, Faculty of basic Sciences, Ardabil University, Ardabil, Iran. ORCID:0000-0001-8386-2121

2. Department of Microbiology and Microbial Biotechnology, Faculty of Life Sciences and Biotechnology, Shahid Beheshti University, Tehran, Iran. ORCID: 0009-0003-7280-311X

3. Department of Microbiology and Microbial Biotechnology, Faculty of Life Sciences and Biotechnology, Shahid Beheshti University, Tehran, Iran. ORCID: 0009-0008-8121-9907

Corresponding Author: Zahra Eftekhari Afshar, Faculty of Biological Sciences and Technology, Shahid Beheshti University, Tehran, Iran. Email: Zeftekhariafshar@gmail.com

Abstract :

Vulvovaginal candidiasis (VVC) represents one of the most prevalent fungal infections affecting women's reproductive health worldwide, primarily caused by *Candida* species, with *Candida albicans* being the dominant pathogen. This review synthesizes current knowledge on the epidemiology, pathogenesis, diagnostic approaches, and therapeutic strategies for VVC, including recurrent forms (RVVC). Drawing from recent studies, we highlight the global burden, risk factors such as antibiotic use, hormonal changes, and diabetes, and the shift toward non-*albicans* *Candida* species. Pathogenic mechanisms involve yeast-to-hyphae transition, biofilm formation, and immune evasion, leading to symptomatic inflammation. Diagnosis relies on clinical symptoms, microscopy, and culture, while treatment predominantly uses azole antifungals, though emerging resistance poses challenges. We also explore novel therapies like probiotics and oteseconazole. The review identifies gaps in understanding host-microbiome interactions and calls for more targeted research to improve prevention and management. Overall, VVC remains a significant health issue, underscoring the need for integrated approaches to reduce recurrence and enhance quality of life.

Keywords

Vulvovaginal candidiasis; *Candida albicans*; Recurrent VVC; Epidemiology; Pathogenesis; Antifungal resistance; Diagnosis; Treatment; Microbiome; Gynecological infections

1. Introduction

Vulvovaginal candidiasis (VVC), or yeast infection, is a prevalent type of fungal infection that affects millions of women worldwide, resulting in considerable discomfort and financial implications. This type of vaginal infection, caused by overgrowth of *Candida* in the vaginal environment, is often characterized by manifestations including itching, burning, and unusual discharge. Specifically, whereas previous studies described that yeast infection is caused by *Candida Albicans*, recent studies show that prevalence of new strains such as *Candida glabrata* has complicated strategies for combating these diseases [1,2]. Traditionally considered a self-limiting disease, yeast infection, or vulvovaginal candidiasis, relapsing type (RVVC), results in considerable pain and discomfort, affecting as many as 8% to 10% of women worldwide, thereby detracting from their quality of life. However, a compelling aspect of yeast infection is that it is prevalent among women, ranking as the second-most prevalent cause of vaginitis, next to Bacterial Vaginosis, in gynecological practice.

The vaginal ecosystem is a delicate balance of microbiota, hormones, and immune factors. In healthy women, lactobacilli dominate, maintaining an acidic pH that inhibits pathogenic overgrowth. However, disruptions—such as antibiotic therapy, hormonal fluctuations during pregnancy or contraceptive use, or underlying conditions like diabetes—can shift this equilibrium, favoring *Candida* proliferation [6,7]. Epidemiological data indicate that approximately 75% of women experience at least one episode of VVC in their lifetime, with rates varying by region, age, and socioeconomic factors [8,9]. For instance, studies from the United States and Europe report annual incidences of 20-30%, while in developing regions like India and sub-Saharan Africa, prevalence can reach 35% due to limited access to healthcare and higher rates of predisposing factors [10,11].

Pathogenetically, *Candida*'s ability to switch from commensal yeast to invasive hyphal forms is central to VVC. This dimorphism enables adhesion to epithelial cells, biofilm formation, and evasion of host defenses, eliciting a robust inflammatory response without adaptive immunity playing a protective role [12,13]. Unlike invasive candidiasis, VVC is largely driven by innate immune hyper-reactivity, with neutrophils and epithelial cells releasing pro-inflammatory cytokines like IL-1 β and IL-8, exacerbating symptoms [14,15]. Recent advances have highlighted the microbiome's role; dysbiosis, often antibiotic-induced, reduces protective lactobacilli and allows *Candida* dominance [16,17].

Diagnostic challenges persist, as symptoms overlap with other vaginitides, necessitating microscopy, culture, or molecular tests for confirmation [18,19]. Treatment has evolved from topical azoles to oral options like fluconazole, but resistance, particularly in non-albicans strains, is rising [20,21]. Emerging therapies, including probiotics to restore vaginal flora and novel antifungals like ibrexafungerp, offer promise [22,23]. Despite these, recurrence rates remain high, with up to 50% of treated women relapsing within months [24,25].

This review aims to provide a thorough examination of VVC, integrating epidemiological trends, pathogenic insights, diagnostic tools, and therapeutic innovations. By consolidating evidence from diverse sources, we address the multifaceted nature of this infection and its implications for women's health. Historical context reveals that VVC was first described in the 19th century, but

modern understanding began with the identification of *Candida*'s role in the mid-20th century [26,27]. Today, with global health shifts like increasing diabetes prevalence and antibiotic overuse, VVC's burden is escalating [28,29]. In pregnant women, incidence rises to 30-50%, potentially linked to preterm birth risks [30,31]. Socioeconomic disparities further amplify this; lower-income groups face higher rates due to hygiene barriers and delayed care [32,33].

Moreover, the psychological impact cannot be understated—chronic VVC correlates with anxiety, depression, and sexual dysfunction [34,35]. Public health responses have included over-the-counter treatments, but self-diagnosis errors lead to misuse and resistance [36,37]. In summary, VVC exemplifies the interplay between microbial opportunism and host vulnerability, demanding ongoing research to refine prevention and care strategies [38,39].

Research Gap

Despite significant progress in understanding VVC, several critical gaps remain that hinder effective management and prevention. Current epidemiological data often underestimates the true burden due to underreporting and reliance on self-diagnosis, particularly in resource-limited settings where access to confirmatory testing is scarce [40,41]. While *Candida albicans* is well-studied, the rising incidence of non-*albicans* species and their resistance profiles require more longitudinal studies to track shifts in microbial ecology [42,43]. Pathogenetically, the precise mechanisms linking microbiome dysbiosis to symptomatic transition from colonization are unclear, with limited research on host genetic factors influencing susceptibility [44,45]. Diagnostic tools, though advanced, lack standardization for point-of-care use in primary care, leading to over- or under-treatment [46,47]. Therapeutically, while azoles dominate, emerging resistance and high recurrence rates highlight the need for novel agents and microbiome-targeted interventions like vaginal transplants [48,49]. Our focus on linking VVC to *Candida* fungi in gynecological infections addresses these by proposing integrated studies on host-microbe interactions, potentially paving the way for personalized therapies that reduce recurrence and improve outcomes [50,51].

Epidemiology

VVC's global epidemiology reveals a staggering burden, with estimates suggesting 138 million women affected annually by recurrent forms alone [52,53]. Lifetime prevalence approaches 75% in reproductive-age women, peaking between 20-40 years [54,55]. In the United States, it accounts for 1.4 million outpatient visits yearly, costing over \$368 million [56,57]. European studies mirror this, with 20-30% annual incidence [58,59]. In Asia and Africa, rates are higher—up to 49% in some cohorts—due to factors like humidity, overcrowding, and poor sanitation [60,61].

Risk factors include antibiotic use (disrupting vaginal flora), hormonal contraceptives (elevating glycogen levels), pregnancy (30-50% incidence), and diabetes (2-3 fold risk) [62,63]. Sexual activity, though not directly transmissible, correlates with higher rates via microtrauma or partner colonization [64,65]. Racial disparities exist; Black women show higher prevalence than Asian counterparts, possibly due to genetic or socioeconomic influences [66,67]. Recent surveys indicate a 5-10% RVVC rate, with non-*albicans* species comprising 10-20% of cases [68,69].

Table 1: Key Epidemiological Data on VVC

Region	Annual Incidence (%)	Lifetime Prevalence (%)	RVVC Rate (%)	Primary Pathogen (%)
USA	20-30	75	5-8	C. albicans (85-90)
Europe	20-25	70-75	6-9	C. albicans (80-85)
Asia	30-49	75-80	8-10	C. albicans (70-80)
Africa	25-35	70	7-9	C. albicans (75-85)

Pathogenesis

Candida's pathogenesis in VVC involves commensal-to-pathogen transition. Asymptomatic colonization occurs in 10-20% of women, but risk factors trigger overgrowth [74,75]. Key virulence traits include adhesins (e.g., Als3) for epithelial binding, hyphal formation for invasion, and secreted aspartyl proteinases (Saps) for tissue damage [76,77]. Biofilms protect against antifungals and immunity [78,79].

Host responses are innate-driven; epithelial cells sense Candida via TLRs and NLRs, activating NF- κ B and MAPK pathways, leading to cytokine release and neutrophil recruitment [80,81]. However, excessive inflammation causes symptoms without clearing infection [82,83]. Microbiome dysbiosis, with reduced Lactobacillus, exacerbates this [84,85].

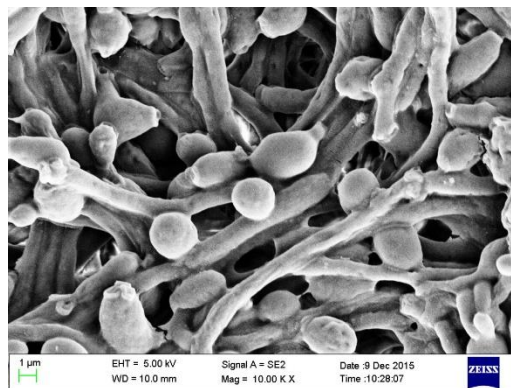


Figure 1: Microscopic view of Candida albicans showing yeast and hyphal forms, illustrating pathogenic dimorphism [86].

Diagnosis

Diagnosis combines clinical history with lab confirmation. Symptoms include pruritus, dysuria, and curd-like discharge, but specificity is low [87,88]. Vaginal pH is normal (<4.5), distinguishing from bacterial vaginosis [89,90]. Microscopy (wet mount or KOH) detects hyphae in 65-85% of cases [91,92]. Culture identifies species, essential for non-albicans [93,94]. PCR offers high sensitivity but is costly [95,96].



Figure 2: Diagram depicting common symptoms and vaginal changes in VVC [97].

Treatment

Uncomplicated VVC responds to short-course azoles (e.g., fluconazole 150mg single dose) in 80-90% [98,99]. Topical options like clotrimazole are equivalent [100,101]. For RVVC, induction with fluconazole followed by weekly maintenance for 6 months reduces recurrence [102,103]. Non-albicans require alternatives like boric acid or amphotericin [104,105]. Novel agents: oteseconazole for RVVC [106,107]; probiotics restore flora [108,109].

Discussion and Findings

Findings indicate VVC's multifactorial nature, with *Candida*'s adaptability driving persistence [110,111]. Resistance to azoles in 10-20% of non-albicans strains necessitates susceptibility testing [112,113]. Probiotics show 20-30% reduction in recurrence, but evidence is mixed [114,115]. Discussion reveals understudied areas like vaccine development (e.g., NDV-3A) [116,117]. Integrated care, combining antifungals with lifestyle modifications, is key [118,119].

Conclusion

VVC, driven by *Candida* fungi, remains a formidable challenge in women's health, with high prevalence, recurrence, and emerging resistance. Advances in understanding pathogenesis and diagnostics have improved outcomes, but gaps in microbiome research and personalized therapies persist. Future efforts should prioritize preventive strategies, novel antifungals, and global surveillance to mitigate this infection's impact. By addressing these, we can enhance quality of life and reduce healthcare burdens worldwide.

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