

Review

Vulvovaginal candidiasis and Candida virulence factor hyphal wall protein 1 (HWP1)

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Abstract

Women can be infected with various types of fungal infections during their lives. Vulvovaginal candidiasis (VVC) is the most common fungal infection in women, with a high prevalence rate. *Candida* spp. are the main causative agents of VVC, and *C. albicans* is the most common species. Such fungi usually have various virulence factors to cause VVC. Some are well identified, and others are not. Several genes and their proteins can be associated with the pathogenesis of *Candida* species to develop VVC. Hyphal wall protein 1 (HWP1) is one protein with different roles in adhesion, invasion, and morphological transition of *Candida* species. This review tried to clarify the nature of VVC and the role of virulence factors of *Candida* spp., particularly HWP1, in this infection.

Keywords: Vulvovaginal candidiasis; *Candida*; HWP1; virulence factors; ALS

Introduction

Vulvovaginal candidiasis (VVC) is the second most common infection in the reproductive system of females after bacterial infections [1-2]. About 75% of females are more likely to suffer from VVC during their lives [3]. Immunocompromised conditions, pregnancy, and treatment with antibiotics and contraceptive drugs are the most predisposing factors for VVC [4-5]. *Candida* species, including *C. albicans* and non-*albicans Candida* (NAC), are mainly responsible for VVC [5]. *C. albicans* frequently causes a higher incidence rate of VVC than other species of *Candida* [6]. It lives as microbiota in different sites in the human body, including the vagina, and can convert to a pathogen under specific conditions [7]. For causing VVC, *Candida* spp. have several virulence factors. The most important factors are those used for morphological transition, adhesion, biofilm production, and invasion [7-9]. These factors and their role in VVC are discussed in this review with brief information. Also, more details were included about the hyphal wall protein (HWP1), as it is an important virulence factor of *Candida* spp. in VVC.

Candida species

Candida is a common genus of yeasts living in the soil as saprophytic fungi or in the human or animal body as a microflora [10]. It belongs to the

Ascomycota phylum, the Hemiascomycetes class, the Saccharomycetales order, and the Candidaceae family [11]. The Latin word candidus is the origin of the term *Candida*, which means bright white, as shown by *Candida* colonies on culture media [10]. A great number of species reproduced asexually, while sexual reproduction has been identified in some of them [12-13]. *Candida* spp. have different morphological forms due to the effects of environmental conditions [10]. The common form of *Candida*, particularly in the human body, is the yeast form, which produces blastoconidia as a bud from oval or spherical cells [14]. Hyphae with ellipsoid shapes and pseudohyphae with constrictions at the cell junction are the other forms [14]. The transition of yeasts to hyphae by forming a germ tube is usually induced by the temperature (37°C), pH 7.0, and serum [15].

Candida spp. can be grown on different enrichment media in the laboratory, such as Sabouraud's dextrose agar and blood agar, and poor media, such as potato dextrose agar, corn agar, and malt agar [16]. Differential media can also be used to identify the species, such as CHROMagar [17]. *Candida* spp. can produce round-shaped colonies with smooth or glistening surfaces and yellowish to white or creamy color on Sabouraud's dextrose agar after 24 h at 37°C [18]. On CHROMagar, colonies can show different colors that are used for the diagnosis of *Candida* species, such as green for

C. albicans, pink for *C. glabrata*, cream for *C. parapsilosis*, metallic blue for *C. tropicalis*, and dark green for *C. dubliniensis* [8].

Some *Candida* species have the ability to cause opportunistic fungal infections in humans and animals. The *Candida* genus contains about 200 species, and only 20 species are known to cause infections in humans [10]. *C. albicans* is the best-known species living as a member of the microflora on the skin, gastrointestinal tract, and vagina and can cause most of the fungal infections in the human body [19]. Other pathogenic species rather than *C. albicans* are collected under the term "non-*albicans Candida*" (NAC), which commonly includes five species; *C. glabrata*, *C. parapsilosis*, *C. krusei*, *C. tropicalis*, and *C. dubliniensis* [20]. *C. albicans* is characterized by a spherical to oval shape with a 5 µm diameter and has a soft cell wall [21]. *C. glabrata*, which is the second most common pathogenic *Candida* species, has a smaller size and haploid genome [22-23]. The highly related species to *C. albicans* is *C. dubliniensis*, which is mostly isolated from the oral cavity and immunocompromised patients [24]. *C. krusei* lives as microflora in the reproductive system of humans and has been isolated from patients with cancers or pericardial inflammation [25]. Neonates are commonly infected with *C. parapsilosis*, which usually produces biofilm on prosthetic surfaces and venous catheters, causing candidemia and cancers [26].

Vulvovaginal candidiasis

Vulvovaginal candidiasis (VVC) is a frequently known infection by *Candida* spp. in the vagina and lower part of the female reproductive system [1]. It is considered the second most common infection after bacterial infections, but without fatal characteristics [2]. About one million females (75%) are expected to be infected with VVC worldwide every year [3]. Immunocompromised females are at high risk of getting VVC [4]. Other predisposing factors for VVC in females are pregnancy, oral contraceptives, antibiotics, diabetes, diet, corticosteroid treatment, and respiratory allergy [5]. An effect on hormones, estrogens and progesterone, due to the use of oral contraceptives can induce *Candida* growth in the vagina by providing a suitable environment [27]. Antibiotics that have a reduction effect on the bacterial flora in the vagina can increase the chance of *Candida* overgrowth and cause VVC [28]. However, reproductive age (25-40 years) is more affected by VVC, particularly during childbirth [29].

Several *Candida* species can develop VVC, including *C. albicans* and NAC, such as *C. glabrata*, *C.*

tropicalis, *C. parapsilosis*, and *C. krusei* [5]. The severity and recurrence of VVC depend on the causative species [2]. *C. albicans* is the common cause of VVC with a high incidence rate [6]. It has a variety of virulence factors that enable it to establish severe infections with an increase of recurrent VVC (RVVC) [29]. In contrast, most NAC have fewer virulence factors and cause low prevalence VVC, about 50% of all VVC, but the pathogenesis of some of them may show more than *C. albicans* [30]. Signs of VVC mostly cannot be clear to the infected female [5]. The common symptoms of VVC include burning, redness, dysuria, lower abdominal pain, yeast smelling discharge, and vaginal itching [4]. However, many bacterial and parasitic infections, such as gonorrhea, bacterial vaginitis, and trichomoniasis, may show similar symptoms, which make diagnosis of VVC based on clinical signs more difficult and require laboratory test confirmation [31].

Pathogenesis of *Candida* in VVC

Candida is considered one of the important pathogens responsible for about 80% of all fungal infections in humans [32]. Although more than 100 *Candida* species have been diagnosed, some of which live as microflora in different parts of the human body, only 100 species cause human infections [7]. Different factors can be associated with the conversion of normal *Candida* spp. into pathogens causing VVC, including immunocompromised conditions, diabetes, prolong treatment with antibiotics, pregnancy, and oral contraceptives [7, 27-28]. *C. albicans* is the common pathogenic species of the *Candida* genus, particularly in immunocompromised patients [7, 27]. Other *Candida* species, other than *C. albicans*, termed non-*albicans Candida* (NAC), also cause severe and frequent VVC, such as *C. glabrata*, *C. parapsilosis*, *C. tropicalis*, and *C. dubliniensis* [11]. These NACs have different morphological, biochemical, and genetic components compared to *C. albicans* [12].

Virulence factors for the pathogenesis of *Candida* in VVC

Several virulence factors facilitate *Candida* spp., especially *C. albicans*, to be pathogens and cause candidiasis. The most important virulence factors associated with VVC can be briefly counted as follows;

-Morphological transition: *Candida* spp. have dimorphic or polymorphic ability by transitioning from yeast to hyphae at 37°C. This transition has various effects on the progression of *Candida* pathogenicity by increasing adhesion and penetration of tissues and resistance to immune activities [7]. Different cellular signals in *Candida* cells can be

changed after transition to increase the survival of the fungal cells in the human environment [9].

-Adhesion and biofilm forming: *Candida* spp. need to adhere as the first initiation step of pathogenesis by fixing their cells on the host tissues. Biofilm is the second step after adhesion, which can be produced by some species of *Candida*, such as *C. albicans* and *C. dubliniensis* [8]. *Candida* spp. can form a relationship with bacteria to increase its adhesion and biofilm production [33]. Gene expression of adhesion factors of *Candida* can be increased by the presence of bacteria [34]. However, several genes and proteins are responsible for adhesion and biofilm production of *Candida* spp. during VVC is as follows:

1. *ALS* genes: There are many types of *ALS* genes associated with the adhesion or biofilm production of *Candida* spp. (*ALS1-ALS7* and *ALS9*) [35]. For biofilm, *Candida* usually requires two types of *ALS* genes, such as *ALS1* and *ALS3* [7, 36]. *ALS* may also have a role in tissue penetration and the invasive stage of *Candida* pathogenesis [36].
2. *Hyr1* gene: It is mainly activated during the transformation of yeasts to hyphae and it encodes a protein of the cell wall that has a role in adhesion of *Candida* [37]. Biofilm production is also facilitated by *HYR1* protein [38].
3. *IFF4* gene: It is an important gene that encodes an adhesion protein (*IFF4*) of *Candida*, particularly during VVC development [39]. The *IFF4* protein usually increases adhesion of *Candida* spp. with epithelial cells and not with endothelial cells [40].
4. *EAP1* protein: It is one of the cell wall proteins of *Candida* spp. that has a role in adhesion with epithelial cells and non-living surfaces and biofilm production [41].
5. Hyphal wall protein 1 (HWP1): It is usually used by *Candida* for adhesion, and morphological transition [42-44]. It also has a role in the mating of *Candida* spp. [45]. Biofilm of *Candida* can be affected by HWP1 due to its location on the surface of cells [46].

-Invasion: Enzymes are the main virulence factors used by *Candida* to invasion and development of VVC. Secreted aspartyl proteases and phospholipases are important enzymes that have hydrolysis abilities of ester linkages in glycerophospholipids of host cells, leading to destruction of these cells and invasion of *Candida* [7, 47]. Proteases are other enzymes that facilitate *Candida* spp., such as *C. parapsilosis*, to penetrate and invade host tissue [48]. *C. tropicalis* uses proteases for its disseminative and phagocytic resistance [49].

Hyphal wall protein (HWP1)

Hyphal wall protein (HWP1) is an important virulence factor of *Candida* spp. during its association with the pathogenesis of these fungi, particularly by *C. albicans* [50]. It structurally consists of glycoprotein located in the cell wall of *Candida* cell and encoded by the hyphal wall protein 1 gene (*Hwp1*) [42]. The *Hwp1* gene has been detected in several *Candida* species other than *C. albicans*, including *C. africana*, *C. dubliniensis*, *C. tropicalis*, *C. glabrata*, *C. parapsilosis*, and *C. orthopsilosis* [50]. This gene is usually variable in the present in different species of *Candida* and even in the same species, which makes it useful for the diagnosis of *Candida* species. A similarity in *Hwp1* is found between that in *C. albicans*, *C. africana*, and *C. dubliniensis*, while a difference is found between *Hwp1* of *C. glabrata* and *C. tropicalis* [51]. Candidiasis can also be diagnosed by detection of *Hwp1* [52]. Commensalism of *Candida* in the host can also induce expression of *Hwp1*, but it is often limited under the control of immune cells [43].

The *Hwp1* has different functions for *Candida* spp. It is usually used by *Candida* for adhesion with the host cells [42-43]. *Candida* uses HWP1 protein for adhesion and proceed with the pathogenesis by invading the host cells [53]. The ability of HWP1 to adhere may differ from one species to another [54]. Several factors can affect the adhesive ability of HWP1 on the host cells. Genetic mutation and some bacteria may increase or decrease the expression of *Hwp1* [55]. Moreover, other genes may have an effect on the expression of *Hwp1* in *Candida* species [43].

HWP1 is also associated with the morphological transformation of *C. albicans* from yeast to hyphae [44]. Its role in the mating of *Candida* spp. by forming a bridge between contact cells is another function of HWP1 [45]. Biofilm of *Candida* can be affected by HWP1 due to its location on the surface of cells [46]. Additionally, it is found that *Hwp1* has a role in making *Candida* cells resistant to immunity, and any damage to this gene increases the elimination of *Candida* by immune cells [56]. Thus, these functions of *Hwp1* make it important in the diagnosis of *Candida* infections and even in immunity against this fungus [57].

Conclusions

Candida is an important genus of pathogenic fungi with a wide range of fungal infections in humans. It contains several species with various abilities to cause candidiasis in different sites of the human body. Vulvovaginal candidiasis (VVC) is one of these *Candida* infections, which is commonly

caused by *C. albicans* and some of the non-*albicans* *Candida*. These species have many effective virulence factors associated with the pathogenesis of *Candida* to cause VVC through steps of morphological transition, adhesion, biofilm production, and invasion. Proteins with different structural and functional cellular activities in *Candida* cells have been identified to contribute to the pathogenesis of this fungus. HWP1 protein, as one of the essential structures of the cell wall of *Candida* spp., is one of them. It has an essential role in the morphological transition, adhesion, invasion, and mating of different *Candida* species. Diagnosis of *Candida* can also be performed by the detection of the *Hwp1* gene.

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