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A Narrative Review Vulvovaginal Candidiasis during Pregnancy: A Narrative Review of Prevalence, Risk Factors, and Antibiogram among Pregnant Mothers.

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ABSTRACT

Vulvovaginal candidiasis (VVC) is an opportunistic fungal infection of the female lower genital tract caused by *Candida* spp. and is responsible for 90% of the cases of infectious vaginitis. VVC during pregnancy remains neglected; however, it poses several complications, leaving this uniquely vulnerable group at risk of mismanagement. This study addressed these urgent gaps by synthesizing global prevalence trends, pregnancy-specific risk dynamics, and emerging resistance patterns. The VVC incidence was the highest in East Africa, with Kenya reporting the highest prevalence at 90.38%, which was slightly higher than that reported in Uganda (73.1%) and the lowest in developed countries (61.1%). *C. albicans* was the predominant *Candida* spp. isolated from both regions. VVC was associated with the use of hormonal contraceptives, a history of diabetes mellitus, the use of antibiotics, regular douching, the third trimester, and a multigravida status. In developed countries, most *Candida* spp. are resistant to nystatin and clotrimazole, and are sensitive to miconazole. In sub-Saharan countries, nystatin, and miconazole have good antifungal activity, and *C.* albicans has the highest sensitivity to miconazole. In Uganda, clotrimazole resistance was the highest in *C. krusei*. All *Candida* spp. presented the lowest resistance to nystatin and miconazole. In conclusion, VVC is highly prevalent in East Africa and lowest in developed countries, with *C. albican as the* dominant causative agent peaking in the third trimester. The increasing resistance to nystatin and clotrimazole in developed countries, and to nystatin, and miconazole demonstrated good antifungal activity in sub-Saharan countries and Uganda. This resistance pattern demands flexibility in empirical therapy for non-*C. Albican*.

Keywords: Vulvovaginal candidiasis, prevalence, pregnancy, antifungal resistance, risk factors, mothers

Sources: Literature search based on databases and sources such as PubMed, Scopus, Google Scholar, WHO/CDC guidelines, Africa Journals Online (AJOL), and LILACS.

INTRODUCTION

Vulvovaginal candidiasis (VVC) is an opportunistic fungal infection of the female lower genital tract caused by Candida species and is responsible for 90% of the cases of infectious vaginitis.[27, 35]. VVC is most often caused by Candida albicans, which accounts for 70–90% of cases due to its ability to undergo fungal morphogenesis, adhesion to vaginal epithelial cells, production of phospholipases and proteinases and presence of candidalysin.[23, 38]. However, other species, such as Candida glabrata, Candida parapsilosis, and Candida tropicalis, are emerging.[18]. These Candida spp. have the ability to produce biofilms that facilitate their firm adherence to mucosal surfaces where many systemic antifungal drugs cannot penetrate and hence maintain infection[23]. It is unclear whether the Candida strain responsible for VVC in pregnant women is a genitourinary tract commensal or a distinct virulent strain.[40]. Vulvovaginal candidiasis occurs when the normal levels of acid and vaginal yeast are out of balance. [10] and the risk of developing VVC is greater in pregnant mothers,

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especially in the 3rd trimester (30–50%), than in healthy women (20%).[21, 23]. These discrepancies are due to the hyperestrogenic environment of the vaginal mucosa, which favors the transition of Candida spp. from the yeast form to the invasive filamentous form, which exerts a cytotoxic effect on host cells; increased vaginal glycogen, which favors the proliferation of Candida spp. on mucosal surfaces; and a physiological reduction in immune defenses as well as alterations in the vaginal pH from 5.0–6.5.[23, 35].

Vaginal candidiasis manifests with odorless curdy white discharge ("cottage cheese"), intense pruritus, vaginal discharge, an erythematous vulva and dyspareunia as well as irritation and dysuria.[46, 21]. Additionally, itching, inflammation (redness), pain during sexual intercourse, and pain during urination, among others, were mentioned.[32, 34].

The diagnosis of VVC is often clinical and, to a lesser extent, through microscopy, hence inadequacy in empirical management, which could be due to misdiagnosis and antifungal susceptibility profiles of different *Candida species.*[23, 30, 38].

Vulvovaginal candidiasis (VVC) during pregnancy is associated with several complications, such as preterm birth, abortion, Chorioamnionitis, premature rupture of membranes, low birth weight, emotional stress, and suppression of the immune system. Pregnant women are also at risk of contaminating their infants, causing invasive neonatal candidiasis, childhood epilepsy, and cerebral palsy.[21, 32, 39]. Current guidelines rely heavily on studies from nonpregnant populations, leaving pregnant women a uniquely vulnerable group at risk of mismanagement. This study addresses urgent gaps by synthesizing global prevalence trends, pregnancy-specific risk dynamics, and emerging resistance patterns. Hence, providing information on context-specific antenatal care protocols and antifungal stewardship policies, ultimately reduces the burden of VVC-related complications in mothers and neonates.

MATERIALS AND METHODS

Study design

This is a narrative review with a qualitative synthesis of existing evidence on how to synthesize and critically analyze global evidence on VVC during pregnancy, with a focus on prevalence patterns, risk factors, and antifungal Antibiogram.

Literature Review Search Strategy

The literature was searched in databases and sources, where the primary databases included PubMed, Scopus, Google Scholar, and WHO/CDC guidelines; the proquest dissertations; and the regional repositories, which included African Journals Online (AJOL) and Latin American and Caribbean Health Sciences Literature (LILACS), via the search terms "Vulvovaginal candidiasis," "Pregnancy, antifungal resistance, risk factors". All the data were filtered from 2015-2024, English language, and human studies.

Eligibility criteria

Inclusion criteria

All the literature/peer-reviewed articles reported VVC incidence, risk factors, and antifungal susceptibility in pregnant women, government/public health reports, and WHO/CDC guidelines. All studies had clear diagnostic methods, such as culture, PCR, and microscopy.

Exclusion criteria

All the studies included nonpregnant mothers. Case reports, editorials, and non-English publications.

Data Extraction

Data were extracted from a standardized template where the prevalence rates were extracted as percentages of

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VVC in pregnant women stratified by region (developed countries, sub-Saharan Africa, East Africa and Uganda). Risk factors from adjusted odds ratios or relative risks or chi-square tests for significant factors and Antibiogram data for antifungals.

Data synthesis

Prevalence of Vulvovaginal candidiasis in pregnancy

Worldwide, nearly 5–10 million females seek gynecologic advice for vaginitis every year, with approximately 70–75% of childbearing-aged women having at least one episode of Vulvovaginal candidiasis during their lifetime.[15].

In developed countries, the highest prevalence (61.1%) of VVC among pregnant mothers was recorded in Asia, 45% was reported in northeastern India, 20% was reported in Europe, and the lowest (10%) was reported in the USA.[40, 46, 47]. However, in Islamabad, Pakistan, the prevalence of VVC among pregnant mothers was 26.9%.[44]. In Ibb, Yemen, 61.5% of cases were reported, and 44.8% of cases were reported among Lebanese pregnant individuals.[13].

In Africa, the overall prevalence of VVC among pregnant women was pooled at 29.2%, where 35% was reported in Eastern Africa, followed by Western Africa at 28% and North Africa at 15%.[25]. In Burkina Faso, the reported prevalence rate of VVC among symptomatic pregnant mothers was 22.71%.[41].

In sub-Saharan Africa, the highest prevalence (62.2%) of VVC was reported in Nigeria, where 42.5% were from a tertiary hospital, Nasarawa State, Nigeria.[1], and the lowest prevalence (38.0%) was reported at Olabisi Onabanjo University Teaching Hospital Sagamu, Ogun State, and Nigeria.[42]. The prevalence of VVC among pregnant women in Maroua, far North Region of Cameroon, was reported to be 51.33%.[31].

In Ethiopia, 41.4% of pregnant mothers from the Family Guidance Association of Ethiopia had VVC, whereas a 26.8% prevalence was reported at Bulehora University Teaching Hospital, Southern Ethiopia, and the lowest 25% rate was reported at Debre Markos Referral Hospital, Northwest Ethiopia.[6, 15, 45]. The prevalence of VVC among pregnant women, especially in Sub-Saharan countries, was lowest in Ghana, at 27%.[12].

In Eastern African countries, Kenya reported the highest prevalence at 90.38%, followed by Tanzania, which reported a 65.6% VVC incidence among pregnant mothers receiving ANC at Mwanza, Tanzania.[9, 29]. The lowest prevalence of VVC among pregnant women was recorded in Bukavu, Democratic Republic of the Congo, at 27.9%.[29].

In Uganda, the prevalence of Vulvo-vaginal candidiasis is 40.0%.[24]. However, this prevalence varies according to geographical region, where the highest prevalence, 73.1%, was reported at Mulago National Referral Hospital.[26]. A total of 45.5% was reported at Mbarara Regional Referral Hospital.[27]. The lowest prevalence (25%) was reported at Jinja Regional Referral Hospital.[32].

Candida species-specific trends

In developed countries, the most isolated Candida species is Candida albicans. In Islamabad, Pakistan, Candida albican (58.45%) was the most predominant species, followed by Candida glabrata (13.69%), Candida tropicalis (11.87%), Candida krusei (7.76%), and Candida parapsilosis (5.47%).[44]. In Peshawar, most (41.7%) were Candida albicans, followed by 16.7% Candida tropicalis, 16.7% Candida krusei, and Candida glabrata (14.8%).[19]. A study conducted in Hajjah governorate, Yemen, revealed that Candida albican was the most predominant species (59.26%), followed by Candida krusei (13.58%), Candida Tropicalis (11.12%), and Candida glabata (9.87%).[4].

In Lebanese pregnant mothers, non-*albican Candida* strains dominated at 56.6% of VVC strains, whereas *C. albican* infections were at 43.4%, with the main identified species being *C. glabrata* (44.5%).[13]. In Trinidad and Tobago, Candida albican was the most predominant species identified (62%), followed by C. glabrata

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(19.3%), C. tropicalis (13.9%) and C. krusei (4.5%).[3].

In sub-Saharan Africa, a study conducted in Maroua, far North Region of Cameroon, revealed that the predominant Candida species was C. albican (63.78%), followed by C. glabrata (26.78%), C. krusei (7.87%) and C.tropicalis (1.57%).[31]. In Nasarawa State, Nigeria, Candida albicans was the most predominant Candida species (51.2%) isolated.[1].

In Bule Hora University Teaching Hospital, southern Ethiopia, the predominant species was Candida albicans (62.4%), followed by C. glabrata (15.3%).[15]. A study in different hospitals in Ibb, Yemen, revealed that C. albicans (61.2%) was the most prevalent species, followed by C. tropicalis (21.64%) and C. glabrata (11.19%).[11].

At the Family Guidance Association of Ethiopia, 58.6% were C. albicans, whereas 41.4% were non-albican.[6]. In Burkina Faso, Candida albicans accounted for 40.39%, C. glabrata (32.69%), C. tropicalis (15.38%) and C. krusei (11.54%).[41].

At the Maroua Regional Hospital, the most predominant Candida species was C. albicans (63.78%), followed by C. glabrata (26.78%), C. krusei (7.87%) and C. tropicalis (1.57%).[34]. At Debre Markos Referral Hospital, Northwest Ethiopia, the predominant Candida species was Candida albican (56.25%), followed by Candida krusei (21.9%), Candida glabrata (17.7%), Candida tropicalis (1%) and Candida tropicalis (3.1%).[45]

In East African countries**,** the predominant Candida species causing VVC in Kenya is Candida albicans (63.83%), followed by Candida glabrata (29.79%), Candida tropicalis (3.19%), Candida krusei (2.13%) and Candida parapsilosis (1.06%).[33]. However, in Bukavu, DRC, Candida albicans (91.0%) was the most common causative agent of VVC among pregnant women.[28].

In Uganda, the highest prevalence of C. albican was isolated from 83.8% of VVC cases, followed by C. tropicalis (12.5%), C. glabrata (2.5%) and C. krusei (1.25%).[24]. Another pilot study revealed that 81.553% of the isolated Candida species were Candida albicans, followed by Candida glabrata (13.592%), Candida tropicalis, and Candida parapsilosis.[16] This study was conducted at Mbarara Regional Referral Hospital and Mulago National Referral Hospital, where C. albican (78.95% & 73.1%) was the most predominant Candida species, followed by C. glabrata (14.35% & 12%), C. krusei (3.35% & 1%), and C. tropicalis (1.44% & 5%), and the lowest isolated Candida species isolated in Uganda was C. parapsilosis (0.48%).[27, 26].

Risk factors associated with Vulvovaginal candidiasis during pregnancy

Host-Related Factors

Physiological

According to the Centers for Disease Control (CDC), the following risk factors are associated with VVC during pregnancy: the use of hormonal contraceptives (birth control pills) and a weakened immune system.[7].

In addition, pregnancy, the use of hormonal contraceptives, hormone replacement therapy, steroids, and immunosuppressive diseases are risk factors for VVC.[8]. VVC is usually attributed to immunosuppression, the use of oral contraceptives, intrauterine devices, spermicides and condoms.[14].

The risk of acquiring VVC among pregnant women at Debre Markos Referral Hospital, Northwest Ethiopia, was contraceptive use and prolonged antibiotic uses.[45].

Comorbidities

According to the Centers for Disease Control (CDC), the following are some of the identified risk factors associated with VVC during pregnancy: diabetes.[7].

In addition, diabetes mellitus was the main risk factor for VVC. Pregnant women with poor adherence to

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diabetic therapy were 3.95 times more likely to have vaginal candidiasis.[8, 48]. Another study reported that VVC is usually attributed to uncontrolled diabetes. The highest associated risk factors were as follows: 3.5% of VVC pregnant women were HIV-infected, 59.3% had a history of diabetic mellitus, and 21.1% of women had a history of previous candidiasis.[14, 15].

The majority of pregnant women (26.8%) had a previous history of vaginal candidiasis.[17].

Behavioral/environmental factors

According to the Centers for Disease Control (CDC), the following are some of the identified risk factors associated with VVC during pregnancy: recent antibiotic use and hygienic habits. Participants who were hospitalized during the past 12 months were more susceptible to *Candida* species.[7, 8, 13].

In a teaching hospital, Ghana pregnant mothers who reported the use of antibiotics had 2.25 increased odds of developing VVC.[12]. Another study highlighted that VVC is usually attributed to some habits of hygiene, clothing and sexual practices and the abuse of antibiotics.[14, 43].

The highest number of associated risk factors was that 54.6% of mothers had a history of using antibiotics (Hussen et al., 2024). The highest prevalence of VVC was observed in 35% of women who were admitted to regular douching, and only 10% indicated recent use of antibiotics.[15, 37].

The associated risk factor for VVC among pregnant mothers at Mulago National Referral Hospital in Uganda was antibiotic use, which occurred three or more times a day.[26]. The overall risk factors for vaginal candidiasis among pregnant women at Jinja Regional Referral Hospital were using pit latrines (55.7%) and not changing pants daily (62%).[32]. Another study revealed the following risk factors: douching practices and a history of antibiotics use.[29]. The risk factors for VVC among second trimester pregnant women in Bukavu, Democratic Republic of the Congo, were personal hygiene and sexual history.[28].

The majority of pregnant women (52.8%) had the habit of washing from front to back, 39.4% had the habit of wearing synthetic undergarments, and 35.2% were using scented laundry soap to clean the perineum.[17].

Pregnancy-specific Risks

A study conducted in pregnant women in Hajjah governorate, Yemen, revealed that the highest rate of *Candida* infection was among women who were in their third trimester (80%), multigravida (66.1%), and recurrent infection (67.7%).[4]. A study conducted among women attending tertiary care hospitals by Peshawar revealed that the majority of the women who had VVC were in their second trimester (60.2%).[19]. Participants with previous miscarriages were more susceptible to *Candida* species.[13]. The risk factors for VVC development among pregnant mothers in northeastern India were being in the 2nd or 3rd trimester of pregnancy and having a vaginal pH of 5.[40]. In Iraq, 44% of mothers with VVC have a parity of 4-6.[5].

The prevalence rate of Vulvovaginal candidiasis (VVC) in a tertiary hospital in Nasarawa State, Nigeria, was high among pregnant women in the second trimester (87.5%).[1]. Associated risk factors for VVC among symptomatic pregnant mothers from Tobago were being in the 3rd trimester (54.8%).[3]

Another study revealed that 53.57% of pregnant mothers developed vaginal candidiasis in their first trimester of pregnancy.[2, 22]. The highest number of associated risk factors were 2nd trimester (42.0%).[15]. The third trimester of pregnancy and the presence of multiple genera are associated with a lower risk of VVC.[20]. The highest prevalence of VVC was observed in 93% of pregnant women in their third trimester.[37].

In Kenya, approximately 68.09% of symptomatic pregnant mothers visiting the antenatal clinic of Thika District with VVC are in their 3rd trimester.[33]. The majority of pregnant women (39.4%) were multipara, and 57% were in the 3rd trimester.[17]

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Antibiogram of Vulvovaginal candidiasis during pregnancy

In developed countries, a study conducted among women of Peshawar revealed that most (58.3%) *C.* spp were resistant to nystatin, with only 25% of *Candida* spp. being sensitive to nystatin. Clotrimazole resistance was high in 59.3% of patients, with a sensitivity of 21.3%. The *C. glabrata* isolates were 100% sensitive to miconazole, and 7.7% of the *C. glabrata* isolates were resistant to clotrimazole.[19]. All the *C. tropicalis* isolates were 100% sensitive to all the antifungal drugs tested. Most *C. krusei* strains are resistant to clotrimazole (16.7%).[15]. Nystatin inhibited the growth of *C. albicans*, *C. glabrata*, and *C. tropicalis* by 25 mm, 18 mm and 12 mm, respectively. The results revealed that nystatin is an effective curative agent.[36]. Among pregnant women in Hajjah governorate, Yemen, 34.7% of *Candida albicans* isolates were resistant to clotrimazole. In addition, the degree of resistance of *Candida krusei, Candida tropicalis,* and *Candida glabrata* isolates to nystatin was 60%.[4].

In Sub-Saharan countries, a study conducted among pregnant women in Maroua, which is located far north of Cameroon, revealed that nystatin and miconazole demonstrated good antifungal activity[31].

The highest sensitivity of *C. albican* was found for miconazole (93.2%). *C. krusei* is completely resistant to polyenes, with low sensitivity to some azoles.[22]. A total of 58.4% of *Candida albicans* species were sensitive to nystatin. A total of 64.3% of the *Candida krusei* isolates were sensitive to nystatin.[42]. *C. albican* species presented a low resistance rate to nystatin (0.78%).[34].

In Uganda, resistance to clotrimazole was highest in *C. krusei*, where it was 50%, and was less than 30% in all *Candida spp*. All *Candida species* showed resistance to nystatin and miconazole at less than 30% *resistance26. The resistance of all Candida species* to nystatin was less than 1%. *C. glabrata* showed 50% resistance to clotrimazole.[27].

Clinical and public health implications

Treatment challenges

Women who receive fluconazole during pregnancy are 1.29 times more likely to develop cardiac abnormalities and spontaneous abortion, whereas others, such as itraconazole, ketoconazole and voriconazole, are associated with abortion, fetal musculoskeletal malformations and fetal skeletal-visceral abnormalities.[21]

Current guidelines state that only topical antifungal therapy can be used to treat VVC during pregnancy. Treatment of VVC during pregnancy with topical clotrimazole and miconazole can be used at all stages of pregnancy because of minimal systemic exposure to treatment via intravaginal administration. Topical nystatin has no risk associated with major malformations.

RECOMMENDATIONS

- There is need to expand the analysis by exploring the socioeconomic and healthcare system influence on VVC prevalence and management would provide in order to provide a more holistic understanding of risk dynamics.
- A longitudinal studies tracking the treatment outcomes and resistance trends in pregnant women should support evidence-based empirical therapy
- There is need to integrate molecular typing of Candida strains which could help to clarify virulence and resistance mechanisms, enabling targeted interventions and improving maternal and neonatal health outcomes.

CONCLUSION

Vulvovaginal candidiasis is highly prevalent in East Africa, with Kenya reporting the highest prevalence at 90.38%, which is slightly higher than that reported in Uganda at 73.1% and lowest in developed countries. The predominant *Candida species* is *C. albican*, with rates peaking in the third trimester, pregnant mothers with a

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history of use of hormonal contraceptives, those with diabetes mellitus, those who use antibiotics, those with regular habits, and those with multigravida. These risk factors must guide preventive strategies. Therefore, patient education on avoiding irritants, optimizing glycemic control, and early treatment of recurrent infections can mitigate the disease burden. Rising resistance among *candida species* to nystatin and clotrimazole necessitates routine antifungal susceptibility testing in recurrent or refractory cases, although nystatin and miconazole have good antifungal activity in sub-Saharan countries. High resistance to clotrimazole was demonstrated in *non-albican varieties.* However, all the *Candida species* showed resistance to nystatin and miconazole, especially in Uganda. This resistance pattern demands flexibility in empiric therapy *for non-albican*

List of abbreviations

C. Candida

CDC: Centers for Disease Control

PCR: Polymerase Chain Reaction

Spp: Species

VVC: Vulvovaginal candidiasis

WHO: World Health Organization

Declaration

Ethical approval and consent to participate

Not applicable

Consent for publication

Not applicable

Competing interest

No, I declare that the authors have no competing interests as defined by BMC or other interests that might be perceived to influence the results and/or discussion reported in this paper.

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Availability of data and materials

Not applicable

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Author contributions

AB: Study design and initial manuscript draft preparation; VS: Conceptualizing, Data collection and manuscript writing support; NR: Editing the manuscript and data collection and data interpretation; BW:

Data curation and initial draft of the manuscript to the final form. All authors reviewed the manuscript and approved the final version of the manuscript.

Clinical trial number

Not Applicable

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