



MICROBIOLOGICAL AND CLINICAL PROFILE OF VULVOVAGINAL CANDIDIASIS AMONG PREGNANT WOMEN ATTENDING A TERTIARY CARE CENTRE

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ABSTRACT

Background: Vulvovaginal candidiasis (VVC) is one of the most frequent fungal infections affecting pregnant women. Hormonal, immunologic, and microbiome changes during pregnancy predispose to Candida overgrowth.

Aim and Objective: The study aimed to determine the prevalence, species distribution, virulence traits, and antifungal susceptibility among Candida isolates from pregnant women presenting with symptoms suggestive of VVC.

Methods: A cross-sectional study was conducted on 500 pregnant women attending the antenatal clinic at a tertiary care center. High vaginal swabs were collected and processed by standard microbiological methods. Isolates were identified to species level using CHROMagar and biochemical assays. Virulence factors such as biofilm formation and phospholipase activity were tested. Antifungal susceptibility to commonly used agents was determined by the CLSI disc diffusion method.

Results: Out of 500 samples, 256 (51.1%) were culture positive for Candida species, and 244 (48.9%) were negative. Among isolates, *C. albicans* constituted 28 (38.4%) and non-*albicans* Candida species 44 (61.6%). *C. tropicalis* (68.2%) was the predominant non-*albicans* isolate, followed by *C. krusei* (17.6%) and *C. glabrata* (8.2%). Biofilm production was observed in 81.1% of isolates, while phospholipase activity was detected in 15.9%. High resistance was observed to

nystatin (89.8%) and cotrimoxazole (84.7%), whereas voriconazole (85%) and amphotericin-B (95%) showed the best activity.

Conclusion: Non-albicans *Candida* species, particularly *C. tropicalis*, are emerging as predominant pathogens in pregnant women with VVC, often showing virulence traits and antifungal resistance. Routine species identification and antifungal susceptibility testing are essential for effective management and prevention of recurrent infection during pregnancy.

Keywords: Microbiological, Clinical Profile, Vulvovaginal Candidiasis, Biofilm, CLSI

INTRODUCTION

Vulvovaginal candidiasis (VVC) is a common mucosal fungal infection caused predominantly by *Candida* species, most frequently *Candida albicans*, and is a leading cause of vulvovaginal symptoms worldwide [1]. Pregnancy is a well-recognized risk state for both colonization and symptomatic infection. Physiological changes of gestation including elevated circulating estrogens, increased vaginal glycogen deposition, modulation of local innate immunity, and altered vaginal microbiota create a vaginal environment that favors overgrowth and symptomatic invasion by *Candida* spp. resulting in increased incidence and recurrence of VVC during pregnancy [2,3].

The prevalence of vaginal yeast colonization and symptomatic VVC rises during pregnancy compared with the nonpregnant state, though reported prevalence varies by setting, population screened, diagnostic method, and trimester [4]. Community and antenatal-clinic based studies report prevalence estimates ranging widely (roughly 17–55% in symptomatic and mixed populations), with higher rates often seen in low- and middle-income settings and among women with gestational diabetes. Recent regional and systematic studies continue to report substantial prevalence among antenatal populations, underscoring VVC as a frequent clinical problem in pregnancy [5,6].

Hormonal changes of pregnancy particularly elevated estrogens enhance *Candida* adherence, germ-tube formation and immune evasion mechanisms, and increase vaginal glycogen that fuels fungal growth. *C. albicans* remains the dominant pathogen, but non-albicans *Candida* species (e.g., *C. glabrata*, *C. tropicalis*) are increasingly reported and are clinically significant due to differing antifungal susceptibilities and a higher propensity for persistence or recurrence [7-9]. Biofilm formation by *Candida* spp. contributes to treatment failure and recurrent disease. Advances in understanding fungal virulence, host–pathogen interactions and biofilm biology clarify why pregnancy predisposes to symptomatic disease and recurrence [10].

Clinically, VVC presents with vulvar pruritus, burning, dysuria and variable amounts of thick, curd-like vaginal discharge. Physical exam commonly shows vulvar erythema and excoriation. Definitive diagnosis requires demonstration of yeast forms or pseudohyphae on microscopy (saline or 10% KOH wet mount) or positive culture; however, microscopy has variable sensitivity and may miss cases. Molecular diagnostics (PCR-based assays) and point-of-care nucleic acid amplification tests are increasingly used and improve sensitivity and species identification, facilitating targeted therapy in recurrent or refractory cases. Careful differentiation from other causes of vaginitis (bacterial vaginosis, trichomoniasis) is essential [11,12].

Pregnancy per se is an independent risk factor. Additional factors associated with increased risk include poorly controlled diabetes mellitus, antibiotic exposure, high estrogen exposure (including exogenous hormones), immunosuppression, and prior history of recurrent VVC. Recurrent VVC (defined as ≥ 4 episodes/year) poses management challenges during pregnancy because systemic agents commonly used outside pregnancy (oral azoles) are relatively contraindicated in gestation [13].

The clinical significance of antenatal yeast colonization beyond symptomatic disease is debated. Some observational studies have explored associations between vaginal yeast and adverse pregnancy outcomes (preterm birth, chorioamnionitis), but evidence remains inconsistent and insufficient to recommend routine screening of asymptomatic pregnant women solely to prevent

obstetric complications [14,15]. Symptomatic infection, however, adversely affects maternal quality of life and may warrant prompt treatment.

Treatment principles in pregnancy prioritize maternal safety and fetal considerations. Current expert guidelines and public-health guidance recommend topical intravaginal azole therapy (clotrimazole, miconazole) for 7 days as first-line therapy during pregnancy; oral fluconazole is generally avoided due to reported associations with adverse fetal outcomes when used in certain doses and stages of pregnancy. For recurrent disease, extended topical regimens are used, and specialist consultation (infectious diseases/obstetrics) is advisable to balance control of maternal symptoms against fetal safety. Resistance among non-albicans species and biofilm-associated persistence complicates management and may require individualized approaches.

The increased recognition of non-albicans species, antifungal resistance, and the clinical impact of fungal biofilms highlight the need for species-level identification and susceptibility testing in selected cases (recurrent, refractory, or complicated presentations). Molecular diagnostics improve detection and species identification but must be integrated with clinical assessment to avoid overtreatment of mere colonization. Ongoing research into host–pathogen interactions, vaccine development, novel antifungals and strategies to disrupt biofilms may change future preventive and therapeutic approaches in pregnant populations.

Vulvovaginal candidiasis in pregnancy is common and driven by physiologic hormonal and immunologic changes that favor *Candida* overgrowth. Accurate diagnosis, species identification when indicated, and adherence to pregnancy-safe topical antifungal therapy are central to management [16].

Attention to comorbid conditions (e.g., diabetes), judicious use of diagnostics, and a focus on symptomatic relief while avoiding potentially teratogenic systemic therapies underpin contemporary clinical practice. Future advances in diagnostics, antifungal agents, and an improved understanding of host–fungus interactions will help tailor safer and more effective strategies for pregnant patients.

MATERIAL AND METHODS

This cross-sectional study was conducted in the Department of Microbiology in collaboration with the Department of Obstetrics and Gynecology at a tertiary care teaching hospital. A total of 500 pregnant women attending the antenatal clinic with symptoms of vulvovaginal discharge, itching, burning, or discomfort were included after obtaining informed consent.

Sample Collection

High vaginal swabs were collected using sterile cotton swabs under aseptic precautions. Swabs were transported immediately to the microbiology laboratory for processing.

Microbiological Identification

Samples were cultured on Sabouraud's Dextrose Agar (SDA) with chloramphenicol and incubated at 37°C for 48 hours. Colonies were further identified using Gram staining, germ tube test, chlamydospore formation on cornmeal agar, and CHROMagar *Candida* for species differentiation. Confirmation was done using biochemical tests (sugar assimilation).

Virulence Factor Testing

1. Biofilm formation was assessed by the tube adherence method.
2. Phospholipase activity was detected on egg yolk agar, with zone measurement indicating enzyme production.

Antifungal Susceptibility Testing

Performed by CLSI M44-A disc diffusion method against fluconazole, itraconazole, voriconazole, amphotericin-B, micafungin, cotrimoxazole, and nystatin. Results were interpreted as sensitive or resistant according to CLSI guidelines.

Data Analysis

Results were expressed as frequency and percentage. Statistical analysis was performed using SPSS software, version 22.0.

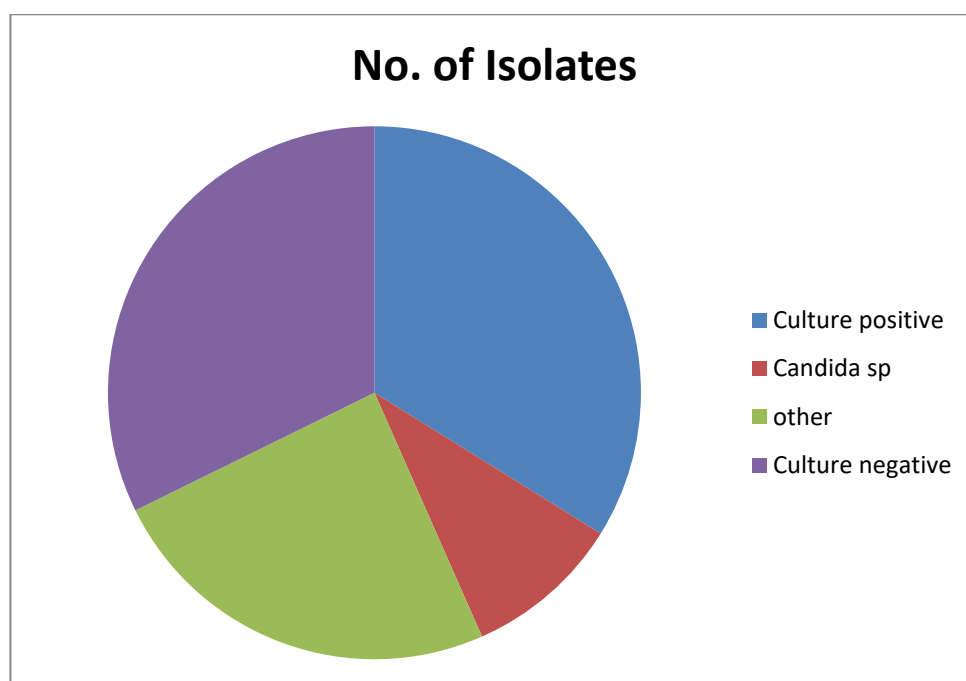
RESULTS

Out of 500 samples, 256 (51.1%) were culture positive for Candida species, and 244 (48.9%) were negative. Among isolates, *C. albicans* constituted 28 (38.4%) and non-*albicans* Candida species 44 (61.6%). *C. tropicalis* (68.2%) was the predominant non-*albicans* isolate, followed by *C. krusei* (17.6%) and *C. glabrata* (8.2%). Biofilm production was observed in 81.1% of isolates, while phospholipase activity was detected in 15.9%. High resistance was observed to nystatin (89.8%) and cotrimoxazole (84.7%), whereas voriconazole (85%) and amphotericin-B (95%) showed the best activity.

TOTAL SAMPLE	FREQUENCY	PERCENTAGE
Culture positive	256	51.1%
Candida sp	72	28%
other	184	71.9%
Culture negative	244	48.9%
Total	500	100%

Table No.1 : Frequency of culture positive and culture negative.

Out of 500 samples, 256 (51.1%) were culture positive for Candida species, while 244 (48.9%) were culture negative. This indicates that over half of the symptomatic pregnant women harbored Candida species, suggesting a high burden of infection among antenatal populations.

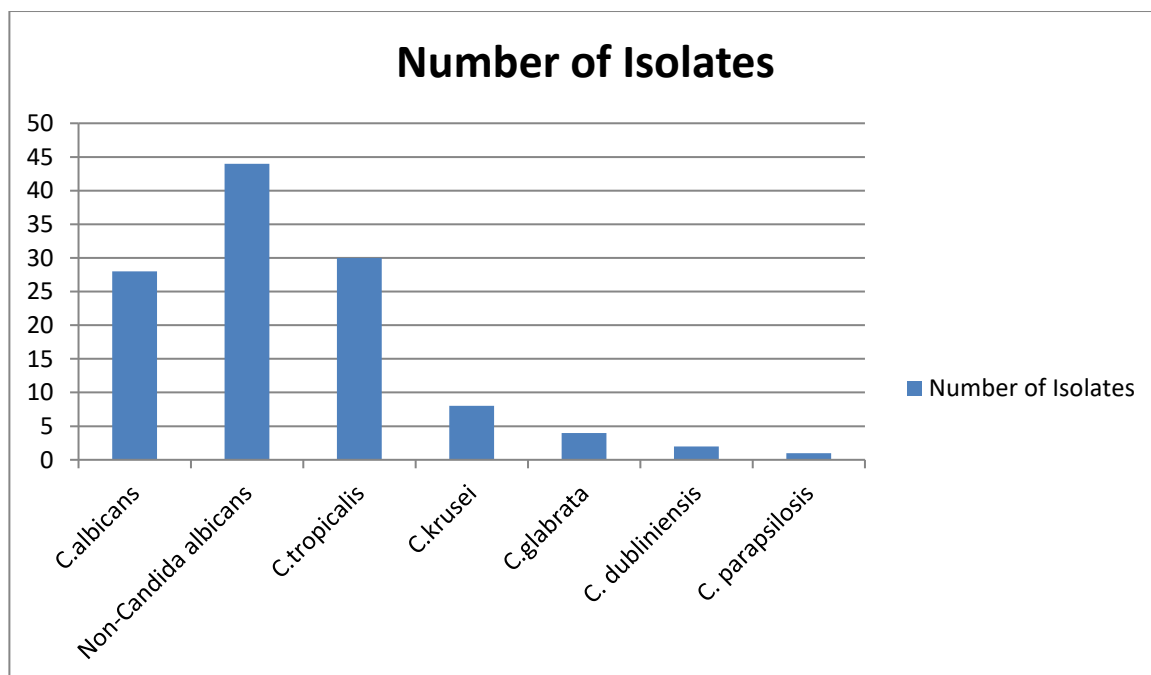


Graph No. 1: Graphical representation of of culture positive and culture negative

CANDIDA ISOLATES	NO. OF ISOLATES	PERCENTAGE
<i>C.albicans</i>	28	38.4%
Non-Candida albicans	44	61.6%
<i>C.tropicalis</i>	30	68.2%
<i>C.krusei</i>	8	17.6%
<i>C.glabrata</i>	4	8.23%
<i>C. dubliniensis</i>	2	3.5%
<i>C. parapsilosis</i>	1	2.3%
TOTAL	138	100%

Table No. 2: Frequency of Candida isolates

Among the 72 Candida isolates, *C. albicans* accounted for 38.4% (28 isolates), and non-albicans Candida (NAC) species were 61.6% (44 isolates). The predominant NAC was *C. tropicalis* (68.2%), followed by *C. krusei* (17.6%) and *C. glabrata* (8.2%). This species shift reflects the increasing clinical relevance of NAC in pregnancy-associated VVC.



Graph No. 2: Graphical representation of species distribution

SAMPLE	<i>C.ALBICAN</i> <i>S</i> (N=28)	PERCENTAGE	NON-CANDIDA ALBICANS (N=44)	PERCENTAGE
Urine	7	24.5%	20	44.7%
Pus	2	5.6%	3	7.07%
Vaginal swab	6	22.6%	10	22.3%
Blood	1	3.7%	0.5	1.17%
Sputum	11	39.6%	9	20%
ET secretions	1	3.7%	3	7.05%
Pleural fluid	0	0%	0	0%
Ascitic fluid	0	0%	0	0%

Table No.3 : Frequency of *C.albicans*& Non-Candida albicans among different sample

Vaginal swabs and urine were the most frequent sources of isolation, with *C. tropicalis* and *C. albicans* being the predominant species. *C. tropicalis* was especially common in urine samples (44.7%), indicating its potential uropathogenic role in pregnancy.

VIRULENCE FACTORS	FREQUENCY	PERCENTAGE
Biofilm production	58	81.1%
<i>C.albicans</i>	17	30.4%
<i>C.tropicalis</i>	28	47.1%
<i>C.glabrata</i>	1	1.4%
<i>C.krusei</i>	2	2.1%
Phospholipidase enzyme	11	15.9%
<i>C.albicans</i>	8	12.3%
<i>C.tropicalis</i>	2	2.1%
<i>C.glabrata</i>	1	1.4%

Table No. 4: Frequency of virulence factors among Various Candida isolates.

Biofilm production was seen in 81.1% of isolates—most frequently among *C. tropicalis* (47.1%) and *C. albicans* (30.4%). Phospholipase activity was detected in 15.9% of isolates, mainly among *C. albicans*. These virulence determinants are linked to pathogenicity and treatment failure in recurrent infections.

Table No 5: Antifungal Drug Resistance Patterns

ANTIFUNGAL DRUGS	SENSITIVITY	SENSITIVITY(%)	RESISTANCE	RESISTANCE %
Fluconazole	16	33%	32	67.3
Cotrimoxazole	7	15.2%	37	84.7
Nystin	5	10.1%	43	89.8
Itraconazole	24	49.2%	24	50.7
Voriconazole	37	85%	7	15.2
Micafungin	4	5.7%	68	94.2
Amphotericin-B	46	95 %	2	4.3

Resistance was highest for nystatin (89.8%) and cotrimoxazole (84.7%). Moderate resistance was noted for itraconazole (50.7%) and fluconazole (67.3%). The most effective agents were amphotericin-B (95% sensitivity) and voriconazole (85% sensitivity), indicating their potential utility in refractory cases.

DISCUSSION

A higher role of Candidal vaginal colonisation in women has been associated with a number of risk factors, including a compromised immune system caused by diabetes, obesity, HIV/AIDS, prolonged use of corticosteroids or broad-spectrum antibiotics, physiological conditions such as ageing, pregnancy, use of high-level oestrogen and progesterone pills, use of intrauterine contraceptive devices (IUCDs), diaphragms, condoms, frequent sexual contact, and use of vaginal douche [3,4].

The present study demonstrates a high prevalence (51.1%) of VVC among pregnant women, consistent with findings by Hussen et al. (2024) [17] and Arsić Arsenijević et al. (2025) [18], who reported prevalence rates of 48–55% in antenatal populations. Pregnancy-related hormonal changes, especially elevated estrogen levels, favor colonization and infection by *Candida* spp..

A significant finding was the predominance of non-albicans *Candida* (61.6%), particularly *C. tropicalis*. This trend aligns with studies by García-Salazar et al. (2024) and SpringerLink Review (2025) [19, 20], emphasizing an epidemiological shift toward NAC species in VVC. NAC species

are less responsive to azoles and more likely to form biofilms, contributing to persistence and recurrence.

Biofilm formation (81.1%) in our study parallels observations by Rodríguez-Cerdeira et al. (2020) and Phillips et al. (2022) [21, 22], confirming its role in antifungal resistance and chronic infection. Biofilm-producing isolates often require prolonged or combination antifungal therapy, posing therapeutic challenges during pregnancy where systemic options are limited.

Antifungal susceptibility testing revealed marked resistance to fluconazole and nystatin. Similar resistance trends were noted in studies by Satora et al. (2023) and Nyirjesy (2022) [23, 24]. The high sensitivity to amphotericin-B (95%) supports its use in severe or recurrent VVC cases, though topical azoles remain preferred in pregnancy for safety reasons.

C. albicans, traditionally the dominant pathogen, was less frequent than *C. tropicalis*, a finding corroborated by Srb et al. (2025) [25]. The increased frequency of NAC may reflect widespread empirical azole use, resulting in species selection pressure.

In line with CDC (2021) [26] and IDSA (2016) [27] guidelines, management of VVC in pregnancy prioritizes topical azoles for seven days. However, given the emergence of NAC and resistance, culture and susceptibility testing should be integrated into clinical practice, especially for recurrent cases.

Fluconazole should therefore only be used when absolutely required, and other therapies should be used wherever feasible [3]. Given these worries, it's critical to assess the scope of the issue, identify the species of *Candida* causing infections, and ascertain how susceptible they are to antifungal medications.

CONCLUSION

The study highlights a substantial burden of VVC among pregnant women, with a rising predominance of non-*albicans* *Candida* species exhibiting virulence traits and antifungal resistance. Routine mycological diagnosis with species-level identification and antifungal susceptibility profiling is crucial for guiding effective treatment and preventing recurrence. Pregnant women with recurrent or persistent symptoms should undergo culture-based confirmation rather than empirical treatment alone.

Limitations of the Study

1. The study was single-center and hospital-based; community prevalence may differ.
2. Molecular identification methods were not employed, limiting precise species typing.
3. Correlation with clinical severity and pregnancy outcomes was not assessed.
4. Longitudinal follow-up for recurrence or treatment response was not performed.

Declarations:

Conflicts of interest: There is not any conflict of interest associated with this study

Consent to participate: There is consent to participate.

Consent for publication: There is consent for the publication of this paper.

Authors' contributions: Author equally contributed the work.

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