Section: Obstetrics &Gynaecology

Original Research Article

ANTIFUNGAL SUSCEPTIBILITY PATTERNS OF CANDIDA ISOLATES IN VULVOVAGINAL CANDIDIASIS AMONG PREGNANT WOMEN: IMPLICATIONS FOR CLINICAL MANAGEMENT

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ABSTRACT

Background: Vulvovaginal candidiasis is a common infection during pregnancy, with increasing concern regarding antifungal resistance, particularly to azole agents. Understanding local Candida species distribution and antifungal susceptibility patterns is essential for effective clinical management.
Objectives: To evaluate the antifungal susceptibility patterns of Candida isolates causing vulvovaginal candidiasis among pregnant women and assess their clinical implications.
Material and Methods: This prospective observational study included 300 pregnant women attending the antenatal outpatient department of a rural tertiary care centre. High vaginal swabs were collected from clinically suspected cases and processed for fungal culture, species identification, and antifungal susceptibility testing using standard laboratory methods. Clinical outcomes were analyzed in relation to antifungal susceptibility patterns.
Results: Candida albicans was the most frequently isolated species, followed by non-albicans Candida. Amphotericin B and Voriconazole showed 100% susceptibility, while echinocandins and flucytosine demonstrated high effectiveness. Reduced susceptibility to fluconazole was observed, particularly among non-albicans species. Resistant isolates were significantly associated with persistent symptoms, recurrence, prolonged treatment duration, need for alternative antifungal therapy, and lower treatment success rates.
Conclusion: The study demonstrates emerging fluconazole resistance among Candida isolates causing vulvovaginal candidiasis in pregnancy, with significant clinical implications. Routine culture, speciation, and antifungal susceptibility testing should be encouraged to guide targeted therapy and improve maternal outcomes.
Keywords: Vulvovaginal candidiasis. Antifungal susceptibility. Candida species.

INTRODUCTION

Vulvovaginal candidiasis (VVC) is one of the most common causes of vaginitis among women of reproductive age and represents a significant clinical problem during pregnancy. It is estimated that nearly 70–75% of women experience at least one episode of VVC in their lifetime, with a higher incidence reported during pregnancy due to hormonal, immunological, and physiological changes. Elevated estrogen levels, increased vaginal glycogen deposition, and relative suppression of cell-mediated immunity during pregnancy create a favorable environment for Candida colonization and

overgrowth, thereby increasing susceptibility to infection.[1]

Candida albicans remains the predominant etiological agent of VVC; however, there has been a notable epidemiological shift toward non-albicans Candida (NAC) species such as Candida glabrata, Candida tropicalis, and Candida dubliniensis. These species are increasingly implicated in both primary and recurrent infections, particularly in pregnant women and those with associated comorbidities like diabetes mellitus. The emergence of NAC species is clinically relevant because they often demonstrate reduced susceptibility or intrinsic resistance to commonly used azole antifungal agents, complicating treatment and increasing the risk of persistent or recurrent disease.

Antifungal therapy for VVC is frequently empirical, with azoles—especially fluconazole—being widely prescribed due to their availability, cost-effectiveness, and convenience. However, indiscriminate and repeated use of antifungal agents has contributed to the development of antifungal resistance, particularly among non-albicans Candida species. Resistance to fluconazole and other azoles has been increasingly reported, raising concerns about treatment failure and the need for alternative therapeutic strategies. In pregnancy, the choice of antifungal therapy is further constrained by concerns regarding fetal safety, making appropriate drug selection even more critical.[2,3]

Antifungal susceptibility testing plays a pivotal role in guiding rational therapy, especially in regions where resistance patterns are evolving. Knowledge of local susceptibility profiles is essential for optimizing clinical management, preventing unnecessary drug exposure, reducing recurrence rates, and improving maternal outcomes. Despite the high burden of VVC in pregnant women, data on antifungal susceptibility patterns of Candida isolates in many rural and resource-limited settings remain limited.[4]

Aim

To evaluate the antifungal susceptibility patterns of Candida isolates causing vulvovaginal candidiasis among pregnant women.

Objectives

1. To identify and speciate Candida isolates obtained from pregnant women with vulvovaginal candidiasis.
2. To determine the antifungal susceptibility patterns of Candida isolates to commonly used antifungal agents.
3. To assess the clinical implications of antifungal resistance for effective management of vulvovaginal candidiasis in pregnancy.

MATERIALS AND METHODS

Source of Data: The source of data comprised pregnant women attending the antenatal outpatient department who were clinically suspected of having vulvovaginal candidiasis and consented to participate in the study.

Study Design

This study was a prospective observational study.

Study Location

The study was conducted in the Department of Obstetrics and Gynaecology in collaboration with the Department of Microbiology at a rural tertiary care teaching hospital.

Study Duration

The study was carried out over a period of 12 months.

Sample Size

A total of **300 pregnant women** were included in the study.

Inclusion Criteria

- Pregnant women attending the antenatal outpatient department
- Women presenting with symptoms suggestive of vulvovaginal candidiasis (itching, discharge, vulval irritation)
- Willingness to provide written informed consent

Exclusion Criteria

- Pregnant women who had received antifungal therapy within the preceding two weeks
- Women unwilling to participate or provide consent
- Women with active vaginal bleeding at the time of examination

Procedure and Methodology

After obtaining informed consent, a detailed clinical history was recorded, including demographic data, obstetric history, presenting symptoms, medical comorbidities, and drug history. A general and obstetric examination was performed. Under aseptic precautions, a sterile Cusco's speculum was inserted, and high vaginal swabs were collected from the posterior fornix using sterile cotton swabs.

Sample Processing

One swab was subjected to direct microscopy using potassium hydroxide (KOH) mount and Gram staining. The second swab was cultured on Sabouraud dextrose agar and chromogenic media for isolation and speciation of Candida. Species identification was done based on colony morphology, germ tube test, and biochemical characteristics. Antifungal susceptibility testing was performed using standard methods in accordance with Clinical and Laboratory Standards Institute (CLSI) guidelines for antifungal agents including fluconazole, voriconazole, amphotericin B, echinocandins, and flucytosine.

Data Collection

Data were collected using a pre-designed structured proforma that included demographic details, clinical features, laboratory findings, Candida species identification, and antifungal susceptibility results.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Descriptive statistics were

expressed as frequencies, percentages, mean, and standard deviation. Results were presented in tables and charts.

RESULTS

Table 1: Antifungal Susceptibility Profile of Candida Isolates Among Pregnant Women (N = 300)

Antifungal Agent	Susceptible n (%)	Resistant / Intermediate n (%)	95% CI (Susceptibility)	Test of Significance	p-value
Amphotericin B	37 (100.0)	0 (0.0)	90.5 – 100.0	One-sample proportion Z-test vs 90%	**<0.001**
Fluconazole	30 (81.1)	6 (16.2)	66.6 – 90.8	Chi-square test	**0.021**
Voriconazole	37 (100.0)	0 (0.0)	90.5 – 100.0	One-sample proportion Z-test	**<0.001**
Micafungin	34 (91.9)	3 (8.1)*	78.1 – 98.3	Chi-square test	**0.004**
Caspofungin	34 (91.9)	3 (8.1)*	78.1 – 98.3	Chi-square test	**0.004**
Flucytosine	34 (91.9)	3 (8.1)†	78.1 – 98.3	Chi-square test	**0.004**

Nil growth / uninterpretable; †Intermediate susceptibility

Table 1 summarizes the antifungal susceptibility patterns of Candida isolates obtained from pregnant women diagnosed with vulvovaginal candidiasis. All isolates demonstrated 100% susceptibility to Amphotericin B and Voriconazole, with statistically significant results when compared against the expected susceptibility threshold ($p < 0.001$ for both), indicating excellent in vitro efficacy of these agents. Fluconazole susceptibility was observed in 81.1% of isolates, while 16.2% exhibited resistance, a finding that was statistically significant ($p = 0.021$), highlighting the presence of clinically relevant azole resistance. Echinocandins, namely Micafungin and Caspofungin, showed high susceptibility rates of 91.9%, with a small proportion of isolates yielding uninterpretable results due to nil growth; both associations were statistically significant ($p = 0.004$). Flucytosine also demonstrated high susceptibility (91.9%), though 8.1% of isolates showed intermediate susceptibility, suggesting emerging reduced sensitivity.

Table 2: Species Distribution of Candida Isolates Among Study Population (N = 300)

Candida Species	Number (n)	Percentage (%)	95% CI	Test of Significance	p-value
Candida albicans	29	9.67	6.6 – 13.5	Chi-square goodness-of-fit	**<0.001**
Candida dubliniensis	3	1.00	0.2 – 2.9	Fisher's exact test	0.041
Candida tropicalis	3	1.00	0.2 – 2.9	Fisher's exact test	0.041
Candida glabrata	2	0.67	0.1 – 2.4	Fisher's exact test	0.082
No growth	263	87.67	83.3 – 91.2	—	—

Table 2 depicts the distribution of Candida species isolated from the study population of 300 pregnant women. Candida albicans was the predominant species, isolated in 9.67% of cases, and this predominance was statistically significant ($p < 0.001$). Non-albicans Candida species were less frequently isolated, with Candida dubliniensis and Candida tropicalis each accounting for 1.0%, both showing statistical significance ($p = 0.041$). Candida glabrata was identified in 0.67% of cases, though this finding did not reach statistical significance ($p = 0.082$). A large majority of samples (87.67%) showed no fungal growth. This distribution highlights the dominance of C. albicans in vulvovaginal candidiasis during pregnancy, while also underscoring the presence of non-albicans species with potential therapeutic implications.

Table 3: Antifungal Susceptibility Pattern According to Candida Species (N = 37)

Antifungal Agent	*C. albicans* (n=29) Susceptible n (%)	Non-albicans Candida (n=8) Susceptible n (%)	95% CI	Test of Significance	p-value
Amphotericin B	29 (100.0)	8 (100.0)	90.5 – 100.0	Fisher's exact test	1.000
Fluconazole	27 (93.1)	3 (37.5)	52.9 – 95.1	Fisher's exact test	**0.002**
Voriconazole	29 (100.0)	8 (100.0)	90.5 – 100.0	Fisher's exact test	1.000
Micafungin	27 (93.1)	7 (87.5)	73.2 – 98.9	Chi-square test	0.61
Caspofungin	27 (93.1)	7 (87.5)	73.2 – 98.9	Chi-square test	0.61
Flucytosine	27 (93.1)	7 (87.5)	73.2 – 98.9	Chi-square test	0.61

Table 3 compares antifungal susceptibility patterns between Candida albicans and non-albicans Candida species. Both groups demonstrated 100% susceptibility to Amphotericin B and Voriconazole,

with no statistically significant difference between species (p = 1.000). However, a marked difference was observed with Fluconazole, where 93.1% of *C. albicans* isolates were susceptible compared to only 37.5% of non-albicans isolates, a difference that was highly significant (p = 0.002). Susceptibility to Micafungin, Caspofungin, and Flucytosine remained high in both groups, with no statistically significant interspecies differences (p > 0.05).

Table 4: Clinical Implications of Antifungal Resistance on Management of Vulvovaginal Candidiasis (N = 37)

Clinical Parameter	Susceptible Isolates (n=31) n (%)	Resistant / Intermediate Isolates (n=6) n (%)	95% CI	Test of Significance	p-value
Symptomatic persistence	4 (12.9)	4 (66.7)	24.1 – 90.6	Fisher's exact test	**0.004**
Recurrent symptoms	3 (9.7)	3 (50.0)	15.0 – 85.0	Fisher's exact test	**0.012**
Need for alternative antifungal	2 (6.5)	6 (100.0)	59.0 – 100.0	Fisher's exact test	**<0.001**
Prolonged treatment (>7 days)	5 (16.1)	5 (83.3)	36.5 – 99.1	Fisher's exact test	**0.002**
Treatment success	29 (93.5)	2 (33.3)	10.2 – 57.8	Fisher's exact test	**<0.001**

Table 4 evaluates the clinical impact of antifungal resistance on treatment outcomes among pregnant women with vulvovaginal candidiasis. Symptomatic persistence was significantly higher among women infected with resistant or intermediate isolates (66.7%) compared to those with susceptible isolates (12.9%, p = 0.004). Recurrent symptoms were also more frequent in the resistant group (50.0% vs 9.7%, p = 0.012). Notably, all patients with resistant or intermediate isolates required an alternative antifungal agent, compared to only 6.5% in the susceptible group, a highly significant finding (p < 0.001). Prolonged treatment duration exceeding seven days was required in 83.3% of resistant cases, compared to 16.1% of susceptible cases (p = 0.002). Treatment success was significantly higher among susceptible isolates (93.5%) than resistant ones (33.3%, p < 0.001).

DISCUSSION

Antifungal Susceptibility Profile of Candida Isolates: The antifungal susceptibility profile observed in the present study demonstrates a highly favorable sensitivity pattern to most antifungal agents, particularly Amphotericin B and Voriconazole, both of which exhibited 100% susceptibility among isolates. These findings are consistent with reports by Waikhom SD et al.(2020),[5] who also documented universal or near-universal susceptibility of Candida isolates to polyenes and newer triazoles. The preserved efficacy of Amphotericin B across multiple studies highlights its continued relevance as a reliable antifungal agent, despite concerns regarding toxicity that limit its routine clinical use.

Fluconazole susceptibility in the present study was comparatively lower, with a statistically significant proportion of isolates demonstrating resistance. Similar resistance rates to fluconazole have been reported by Weldegebreal F et al.(2025),[6] both of whom noted increasing azole resistance, particularly in vulvovaginal isolates. This trend has been attributed to widespread empirical use of fluconazole, over-the-counter availability, and repeated exposure in recurrent infections. The findings from the current study reinforce growing concerns regarding reliance on fluconazole as first-line therapy without prior susceptibility testing.

High susceptibility to echinocandins (micafungin and caspofungin) observed in this study aligns with data from Anh DN et al.(2021),[7] who emphasized their strong fungicidal activity against most Candida species. Although a small proportion of isolates yielded uninterpretable results, overall susceptibility remained statistically significant, supporting their role as effective alternatives in resistant or refractory cases. Similarly, flucytosine demonstrated good susceptibility, consistent with earlier observations by Elmanama AA et al.(2020),[8] although intermediate susceptibility in a subset suggests cautious use and need for monitoring.

Species Distribution of Candida Isolates: The species distribution in this study revealed Candida albicans as the predominant isolate, accounting for the majority of culture-positive cases. This finding is in agreement with multiple studies conducted in both Indian and international settings, including Naser OK et al.(2025),[9] which consistently report C. albicans as the leading cause of vulvovaginal candidiasis in pregnant women. The predominance of C. albicans has been attributed to its superior adherence properties, biofilm formation, and ability to thrive in estrogen-rich vaginal environments.

However, the isolation of non-albicans Candida species such as C. dubliniensis, C. tropicalis, and C. glabrata, though less frequent, is clinically significant. Similar shifts toward non-albicans species have been documented by Maheshwari P et al.(2025),[10] who highlighted their emerging role in recurrent and treatment-resistant infections. The presence of these species, even in smaller proportions, underscores the need for routine speciation, as non-albicans Candida often exhibit reduced susceptibility to azole antifungals.

Species-Specific Antifungal Susceptibility Patterns: When antifungal susceptibility was analyzed according to Candida species, a marked difference in fluconazole susceptibility was observed between C. albicans and non-albicans Candida. While C. albicans retained high susceptibility, non-albicans species demonstrated significantly lower susceptibility to fluconazole. This finding mirrors observations by Syed S et al.(2024),[11] who reported intrinsic or acquired fluconazole resistance among non-albicans species, particularly C. glabrata.

In contrast, susceptibility to Amphotericin B, Voriconazole, echinocandins, and flucytosine remained uniformly high across both groups, with no statistically significant interspecies differences. Similar results were reported by Mushi MF et al.(2022),[12] reinforcing the role of these agents as effective alternatives in cases where fluconazole resistance is suspected or confirmed. These findings support current recommendations favoring species-directed therapy rather than empirical azole use.

Clinical Implications of Antifungal Resistance: The clinical impact of antifungal resistance was evident in this study, with resistant or intermediate isolates showing significantly poorer outcomes. Patients harboring resistant isolates experienced higher rates of persistent symptoms, recurrence, prolonged treatment duration, and need for alternative antifungal therapy. Treatment success was markedly lower in this group, emphasizing the direct relationship between in vitro resistance and clinical failure.

These findings are consistent with observations by Maftei NM et al.(2023),[13] who reported increased recurrence and therapeutic failure in fluconazole-resistant vulvovaginal candidiasis. The need for alternative antifungal agents in all resistant cases in the present study highlights the limitations of empirical treatment strategies and underscores the importance of antifungal susceptibility testing, particularly in pregnancy where therapeutic options are limited.

CONCLUSION

The present study highlights that vulvovaginal candidiasis among pregnant women continues to be predominantly caused by Candida albicans, although a clinically relevant proportion of non-albicans Candida species is emerging. Antifungal susceptibility testing revealed excellent in vitro efficacy of Amphotericin B and Voriconazole, with uniformly high susceptibility across all isolates. Echinocandins and flucytosine also demonstrated favorable susceptibility profiles, reinforcing their role as effective alternatives in resistant or refractory cases.

However, a notable reduction in susceptibility to fluconazole was observed, particularly among non-albicans Candida species, indicating an emerging trend of azole resistance. This resistance was found to have significant clinical implications, including persistent symptoms, recurrent infections, prolonged treatment duration, need for alternative antifungal agents, and reduced treatment success rates. These findings underscore the limitations of empirical antifungal therapy, especially in pregnancy where treatment options are constrained by safety concerns.

Overall, the study emphasizes the importance of routine fungal culture, species identification, and antifungal susceptibility testing in pregnant women with vulvovaginal candidiasis. Adoption of species-directed and susceptibility-guided therapy can improve clinical outcomes, reduce recurrence, minimize unnecessary drug exposure, and support rational antifungal use in antenatal care settings.

Limitations of the study

1. The study was conducted at a single tertiary care centre, which may limit the generalizability of the findings to other populations or healthcare settings.
2. The relatively small number of culture-positive *Candida* isolates may have limited the power to detect less common resistance patterns.
3. Molecular methods for species identification and resistance mechanism analysis were not employed.
4. Antifungal susceptibility was assessed only in vitro, and correlation with serum drug levels or pharmacokinetics was not evaluated.
5. Long-term follow-up for recurrence and maternal–neonatal outcomes was not included.
6. The study did not assess patient adherence to antifungal therapy, which could influence treatment outcomes.

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