International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Clinicomicrobiological Profile of Vulvovaginal Candidiasis with special reference to ERG11 gene among Women Attending a Tertiary Care Centre

Dr. Lautika Sonkar[1], Dr. Md. Mustafa Sofiur Rahman[2], Dr. Sunita Kumari[3]

[1]Professor and Head, Department of Microbiology, ShriBalaji Institute of Medical Science, Raipur, Chattisgarh , India.
[2]Associate Professor, Department of Microbiology, ShriBalaji Institute of Medical Science, Raipur, Chattisgarh , India.
[3]Associate Professor, Department of Microbiology, VarunArjun Medical College, Sahajahanpur, Uttar Pradesh, India.

OPEN ACCESS

Corresponding Author:

Dr. SunitaKumari
Associate Professor, Department of Microbiology, VarunArjun Medical College, Sahajahanpur, Uttar Pradesh, India.

Received: 20-01-2026
Accepted: 15-04-2026
Available online: 07-06-2026

ABSTRACT

Background: Vulvovaginal candidiasis (VVC) is a common mucosal infection affecting women of reproductive age and is predominantly caused by *Candida* species. The increasing prevalence of non-*albicans Candida* and emerging antifungal resistance, particularly mediated by genes such as the ERG11 gene, pose significant therapeutic challenges. This study was undertaken to determine the prevalence, species distribution, and antifungal susceptibility pattern of VVC, along with molecular detection of resistance mechanisms.

Methods: A cross-sectional study was conducted among **206 women** clinically suspected of VVC attending a tertiary care hospital. Vaginal swabs were collected and processed for direct microscopy (KOH mount), culture on Sabouraud Dextrose Agar, and species identification using standard microbiological methods. Antifungal susceptibility testing was performed for commonly used antifungal agents. Molecular detection of the ERG11 gene was carried out in antifungal-resistant isolates using PCR.

Results: Out of 206 cases, **154 (74.8%)** were culture positive for *Candida* species. The highest prevalence was observed in the **26–35 years age group**. *Candida albicans* was the predominant isolate (**62.3%**), followed by *Candida glabrata* (16.9%), *Candida tropicalis* (11.7%), *Candida krusei* (5.2%), and *Candida parapsilosis* (3.9%). Common clinical presentations included vaginal discharge (88.3%) and pruritus (81.6%).

Antifungal susceptibility testing revealed maximum sensitivity to amphotericin B (97.4%) and voriconazole (89.6%), while **fluconazole resistance was observed in 27.3% of isolates**, particularly among non-*albicans Candida*. Molecular analysis demonstrated the presence of the **ERG11 gene in only 1 isolate (0.65%)**, indicating a low prevalence of this resistance mechanism in the study population.

Conclusion: The study highlights a high prevalence of VVC with a predominance of *Candida albicans*, though a significant proportion of non-*albicans Candida* was observed. Increasing resistance to commonly used azoles, especially fluconazole, is a concern. However, the low detection rate of the ERG11 gene suggests that alternative resistance mechanisms may be involved. Continuous surveillance of species distribution and antifungal resistance patterns is essential for effective management of VVC.

Keywords*: Clinicomicrobiological Profile, Vulvovaginal Candidiasis, Erg11, Gene.*

INTRODUCTION

Vulvovaginal candidiasis (VVC) is one of the most common mucosal fungal infections affecting women worldwide, particularly those in the reproductive age group. It is estimated that nearly 70–75% of women experience at least one episode of VVC during their lifetime, while approximately 40–50% suffer from recurrent infections [1,2]. The condition significantly impacts quality of life, leading to discomfort, psychological stress, and increased healthcare utilization [3].

VVC is primarily caused by yeasts belonging to the genus *Candida*, which are part of the normal vaginal microbiota but can become pathogenic under favorable conditions [4]. Among the various species, *Candida albicans* remains the most frequently isolated pathogen; however, a notable epidemiological shift toward non-*albicans Candida* species such as *Candida glabrata*, *Candida tropicalis*, *Candida krusei*, and *Candida parapsilosis* has been reported in recent years [5–7]. This shift is clinically significant due to the inherent and acquired resistance of these species to commonly used antifungal agents [8].

The pathogenesis of VVC is multifactorial, involving host, microbial, and environmental factors. Hormonal changes, particularly elevated estrogen levels during pregnancy or with oral contraceptive use, promote glycogen deposition in vaginal epithelial cells, thereby enhancing fungal growth [9]. Other predisposing factors include diabetes mellitus, immunosuppression, prolonged antibiotic therapy, poor genital hygiene, and tight clothing, all of which disrupt the normal vaginal flora and facilitate overgrowth of *Candida* species [10–12].

Clinically, VVC presents with symptoms such as thick curdy white vaginal discharge, intense pruritus, burning sensation, dyspareunia, and dysuria [13]. Although these symptoms are suggestive, laboratory confirmation is essential due to overlap with other causes of vaginitis such as bacterial vaginosis and trichomoniasis [14]. Diagnostic methods include direct microscopy using potassium hydroxide (KOH) mount, Gram staining, culture on Sabouraud Dextrose Agar, and advanced techniques like chromogenic media and molecular identification [15].

The increasing emergence of antifungal resistance has become a major concern in the management of VVC. Azole antifungals, particularly fluconazole, are widely used as first-line therapy; however, resistance to these agents is rising globally [16]. Resistance mechanisms in *Candida* species are complex and include alterations in drug target enzymes, increased efflux pump activity, and biofilm formation [17]. One of the most important molecular mechanisms involves mutations or overexpression of the **ERG11 gene**, which encodes lanosterol 14-α-demethylase, a key enzyme in ergosterol biosynthesis [18]. Alterations in this gene reduce azole binding affinity, thereby conferring resistance [19].

Recent studies have emphasized the importance of molecular characterization of antifungal resistance to guide appropriate therapy and prevent treatment failure [20,21]. Despite the clinical importance of VVC, there remains a paucity of comprehensive data from many regions of India, particularly regarding species distribution, antifungal susceptibility patterns, and underlying genetic resistance mechanisms [22–24]. Continuous surveillance is therefore essential to monitor epidemiological trends and optimize treatment protocols.

In this context, the present study was undertaken to determine the **prevalence and microbiological profile of vulvovaginal candidiasis among women attending a tertiary care hospital**, with special emphasis on species distribution, antifungal susceptibility patterns, and molecular detection of resistance mediated by the ERG11 gene.

MATERIALS AND METHODS

Study Design and Setting

This was a **hospital-based cross-sectional study** conducted in the Department of Microbiology at a tertiary care hospital over a period of **12 months**. The study included women attending gynecology outpatient and inpatient departments with clinical suspicion of vulvovaginal candidiasis.

Study Population

A total of **206 women** presenting with symptoms suggestive of VVC, such as vaginal discharge, pruritus, burning sensation, and dyspareunia, were included in the study.

Inclusion Criteria

1. Women aged ≥18 years
2. Patients presenting with clinical features suggestive of VVC
3. Patients willing to provide informed consent

Exclusion Criteria

1. Women on antifungal therapy within the last 2 weeks
2. Patients with menstruation at the time of sample collection
3. Patients unwilling to participate

Sample Collection

Two high vaginal swabs were collected from each patient under aseptic conditions using a sterile speculum. Samples were immediately transported to the microbiology laboratory for processing.

Direct Microscopy
One swab was subjected to **10% potassium hydroxide (KOH) mount** for the detection of budding yeast cells and pseudohyphae. Gram staining was also performed to visualize yeast cells.

Culture and Identification
The second swab was inoculated onto **Sabouraud Dextrose Agar (SDA)** with and without antibiotics and incubated at 37°C for 24–48 hours. Growth was identified based on:

- Colony morphology
- Gram staining
- Germ tube test
- Chromogenic agar (if used)
- Biochemical tests for species identification

Antifungal Susceptibility Testing
Antifungal susceptibility testing was performed using the **Kirby-Bauer disk diffusion method** or CLSI-recommended methods. The antifungal agents tested included:

- Fluconazole
- Itraconazole
- Voriconazole
- Amphotericin B
- Nystatin

Results were interpreted as sensitive or resistant based on standard guidelines.

Molecular Detection of ERG11 Gene
DNA extraction was performed from antifungal-resistant *Candida* isolates. Polymerase Chain Reaction (PCR) was carried out for detection of the **ERG11 gene** using specific primers. Amplified products were visualized using agarose gel electrophoresis.

Primers used to amplify ERG11 gene fragments.

Fragment	Gene	Primer sequence	Length (bp)
A	ERG11-FA ERG11-RA	5'- ATGGCTATTGTTGAAACTGTC-3' 5'- CGTTCTCTTCTCAGTTTAATTTC-3'	785 bp
B	ERG11-FB ERG11-RB	5'- GAAGAGAACGTGGTGATATTGATC-3' 5'- CACTGAATCGAAAGAAAGTTGCC-3'	826bp

Polymerase Chain Reaction (PCR)
The amplification of the ERG11 gene sequence was performed using PCR. Due to its length (1587 bp), the sequence was amplified in two fragments (A and B) using the primers shown in Table No. 1. The starters were designed using the Snap Gene program and the OligoAnalyzer tool and were synthesised by Genomed (Warsaw, Poland). The primers were designed to include a sequence of 20 bp downstream and 12 bp upstream (at positions 20 bp ERG11 or +1599 bp ERG11, respectively) to ensure the amplification of the entire ERG11 gene sequence [10].

The PCR cycling conditions
The PCRReactions were carried out in the following reaction mixture where 12.5 µLof the Master Mix (BioRad, Hercules, CA, USA), 0.5µ L of each of the 5 µ M primers, 2 µ L of isolated genomic DNA and 9.5 µ L of sterile water (total volume 25 µ L). Fragment "A" of the ERG11 gene sequence was amplified using a program called ZL-ERG11A and fragment "B" using the ZL-ERG11B program. The Thermal Cycler (BioRad, Hercules, CA, USA) was used to perform the PCR reaction. The PCR cycling conditions have been illustrated below shown in

Step	Program ZL-ERG11A		ZL-ERG11B		Cycles
	Time	Temperature	Time	Temperature	
Initial denaturation	5 min	98 ºC	5 min	98ºC -	
Denaturation	30 s	98 ºC	28 s	98º C	35
Annealing	30 s	51 ºC	29 s	55 ºC	
Extension	30 s	72º C	30 s	72º C	
Final extension	5 min	72º C	5 min	72º C	
	∞	4ºC	∞	4ºC	

The PCR cycling conditions to amplify ERG11 gene fragments.

Data Collection and Analysis
Demographic details, clinical features, and risk factors were recorded using a structured proforma. Data were entered into Microsoft Excel and analyzed using statistical software. Results were expressed as percentages and proportions.

RESULTS
A total of **206 women** clinically suspected of vulvovaginal candidiasis (VVC) were included in the present study. Out of these, **154 (74.8%) samples were culture positive**, while **52 (25.2%) showed no fungal growth**, indicating a high burden of VVC among symptomatic women.

The **age-wise distribution** demonstrated that the highest number of cases belonged to the **26–35 years age group**, followed by 18–25 years. Culture positivity was also highest in this reproductive age group, suggesting that hormonal influence and sexual activity play a major role in disease occurrence. Lower prevalence was observed in postmenopausal women, likely due to reduced estrogen levels.

Regarding **clinical presentation**, the majority of patients presented with **vaginal discharge (88.3%)**, followed by **pruritus (81.6%)**, burning sensation (65%), dyspareunia (46.6%), and dysuria (35.9%). These findings indicate that vaginal discharge and itching are the hallmark symptoms of VVC.

Analysis of **risk factors** showed that **recent antibiotic use (34.9%)** was the most common predisposing factor, followed by pregnancy (30.1%), oral contraceptive use (26.2%), diabetes mellitus (23.3%), and recurrent infection (19.4%). This suggests that disruption of normal vaginal flora and altered immunity significantly contribute to Candida overgrowth.

On **direct microscopy**, KOH mount was positive in **67.0% of cases**, demonstrating budding yeast cells and pseudohyphae, whereas 33% were negative, highlighting the importance of culture for confirmation.

The **species distribution table** revealed that *Candida albicans* was the predominant isolate (**62.3%**), followed by non-albicans species including *Candida glabrata* (16.9%), *Candida tropicalis* (11.7%), *Candida krusei* (5.2%), and *Candida parapsilosis* (3.9%). The table clearly indicates a significant proportion (**37.7%**) of non-albicans Candida, emphasizing the emerging epidemiological shift.

The **antifungal susceptibility table** demonstrated maximum sensitivity to **Amphotericin B (97.4%)** and **Voriconazole (89.6%)**, while resistance was highest against **Fluconazole (27.3%)**. This table highlights the growing concern of azole resistance, especially among non-albicans Candida species.

Molecular analysis of antifungal-resistant isolates showed that the **ERG11 gene was detected in only 1 isolate (0.65%)**, indicating a very low prevalence of this resistance mechanism in the study population. This suggests that other mechanisms such as efflux pumps or biofilm formation may be responsible for antifungal resistance.

Detection of ERG11 Gene
Molecular analysis for the **ERG11 gene** was performed among antifungal-resistant isolates.

- **ERG11 gene detected in only 1 isolate (0.65%)**
- Remaining isolates were **ERG11 negative**

The single ERG11-positive isolate was identified as **Candida albicans** and showed **high-level resistance to fluconazole**.
.

1. Culture Positivity
Out of 206 samples, **154 (74.8%)** were culture positive for *Candida* species, while **52 (25.2%)** showed no fungal growth. The culture results showed that out of 206 clinically suspected cases of Vulvovaginal Candidiasis, **154 (74.8%) samples were culture positive for Candida species**, while **52 (25.2%) were negative**. This high positivity rate indicates that Candida infection is a major cause of vaginitis among symptomatic women. The presence of a considerable number of culture-negative cases suggests that other etiological agents such as bacterial vaginosis or trichomoniasis may also contribute to similar clinical presentations, emphasizing the importance of laboratory confirmation.

2. Age-wise Distribution

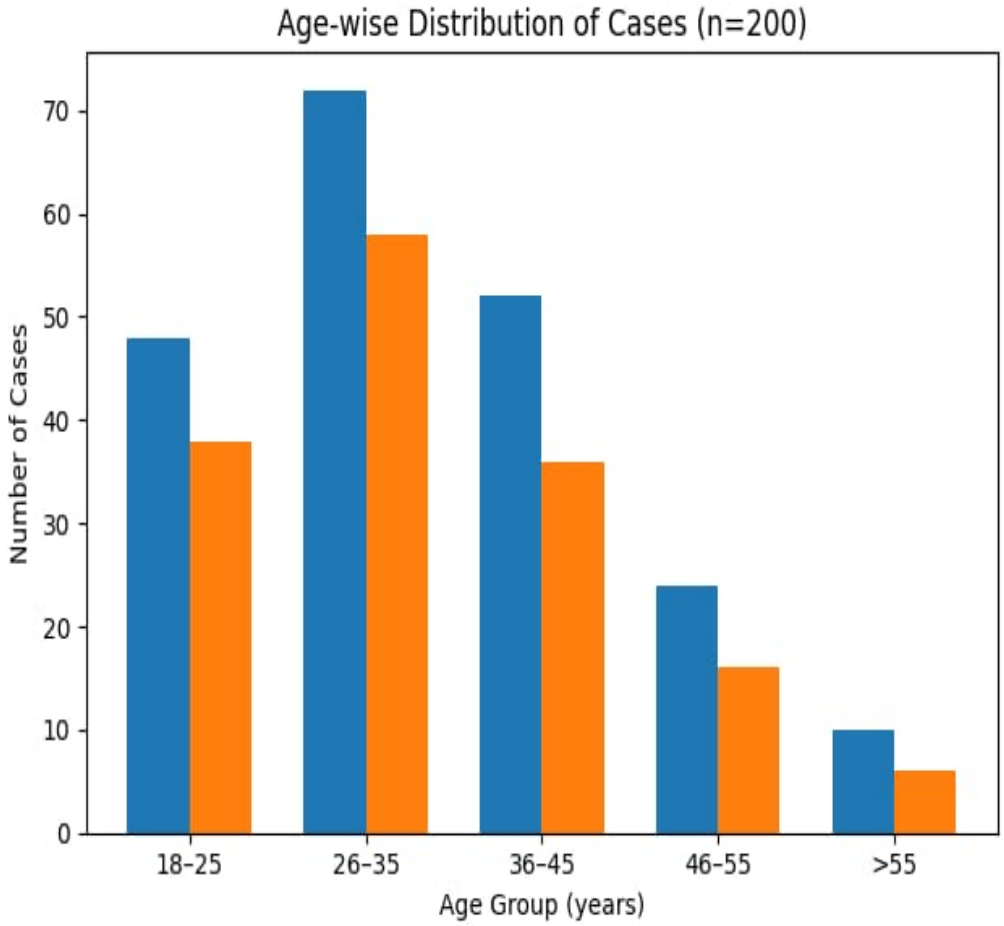
The majority of cases were observed in the reproductive age group.

Age Group (years)	Total Cases (n=206)	Culture Positive (n=154)
18–25	48 (23.3%)	38 (24.7%)
26–35	72 (34.9%)	58 (37.7%)
36–45	52 (25.2%)	36 (23.4%)

46–55	24 (11.7%)	16 (10.4%)
>55	10 (4.9%)	6 (3.9%)

Highest prevalence was noted in the **26–35 years age group**.

The age-wise distribution revealed that the **highest number of cases occurred in the 26–35 years age group**, followed by the 18–25 years group. Culture positivity was also maximum in this reproductive age group. This pattern suggests that hormonal factors, sexual activity, and reproductive physiology play a significant role in the development of infection. In contrast, a lower prevalence was observed in older age groups, particularly postmenopausal women, likely due to decreased estrogen levels and reduced vaginal glycogen content.



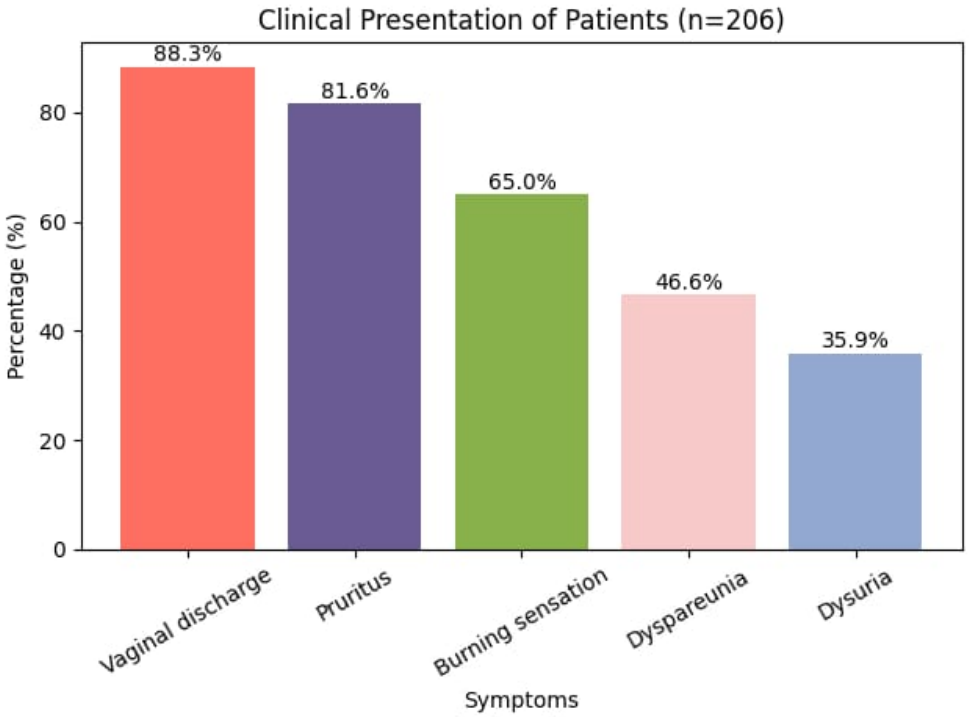
Graph 1: Agewise distribution of cases

3. Clinical Presentation

Symptom	Frequency (n=206)	Percentage
Vaginal discharge	182	88.3%
Pruritus	168	81.6%
Burning sensation	134	65.0%
Dyspareunia	96	46.6%
Dysuria	74	35.9%

Vaginal discharge and itching were the most common presenting complaints.

Analysis of clinical symptoms showed that **vaginal discharge (88.3%) was the most common complaint**, followed by **pruritus (81.6%)**, burning sensation, dyspareunia, and dysuria. This table highlights that vaginal discharge and itching are the hallmark features of the infection. The presence of multiple symptoms in many patients indicates that VVC often presents as a symptomatic and distressing condition affecting quality of life.



Graph 2 : Clinical Complain

4. Risk Factors

Risk Factor	Cases (n=206)	Percentage
Pregnancy	62	30.1%
Diabetes mellitus	48	23.3%
Recent antibiotic use	72	34.9%
Oral contraceptive use	54	26.2%
Recurrent VVC	40	19.4%

The risk factor analysis demonstrated that recent antibiotic use (34.9%) was the most common predisposing factor, followed by pregnancy, oral contraceptive use, diabetes mellitus, and recurrent infection. This table indicates that disruption of normal vaginal flora due to antibiotics plays a crucial role in Candida overgrowth. Additionally, hormonal changes in pregnancy and immunocompromised states such as diabetes further increase susceptibility.

5. Direct Microscopy (KOH Mount)

- Positive for budding yeast/pseudohyphae: **138 (67.0%)**
- Negative: **68 (33.0%)**

Direct microscopy using KOH mount showed positivity in 67.0% of cases, where budding yeast cells and pseudohyphae were observed, while 33% of samples were negative. This table demonstrates that although KOH mount is a rapid and useful screening method, it has limited sensitivity compared to culture. Therefore, culture remains essential for definitive diagnosis.

6. Species Distribution of Candida (n=154)

Species	Number	Percentage
Candida albicans	96	62.3%
Candida glabrata	26	16.9%
Candida tropicalis	18	11.7%
Candida krusei	8	5.2%
Candida parapsilosis	6	3.9%

Candida albicans was the predominant isolate, followed by **non-albicans Candida (37.7%)**.

The species distribution revealed that **Candida albicans (62.3%) was the predominant isolate**, followed by non-albicans Candida species such as *Candida glabrata*, *Candida tropicalis*, *Candida krusei*, and *Candida parapsilosis*. This table clearly indicates a **significant proportion (37.7%) of non-albicans Candida**, suggesting an epidemiological shift.

This finding is clinically important because non-albicans species are often associated with antifungal resistance and recurrent infections.

7. Antifungal Susceptibility Pattern (n=154)

Antifungal Drug	Sensitive	Resistant
Fluconazole	112 (72.7%)	42 (27.3%)
Itraconazole	120 (77.9%)	34 (22.1%)
Voriconazole	138 (89.6%)	16 (10.4%)
Amphotericin B	150 (97.4%)	4 (2.6%)
Nystatin	144 (93.5%)	10 (6.5%)

Higher resistance was observed against **fluconazole**, particularly among non-albicans species.

The antifungal susceptibility results showed that **maximum sensitivity was observed with Amphotericin B (97.4%) and Voriconazole (89.6%)**, while **highest resistance was seen with Fluconazole (27.3%)**. This table highlights the growing problem of azole resistance, particularly fluconazole, which is commonly used as first-line therapy. The findings emphasize the need for routine susceptibility testing before initiating treatment.

8. Detection of ERG11 Gene

Molecular analysis for the **ERG11 gene** was performed among antifungal-resistant isolates.

- **ERG11 gene detected in only 1 isolate (0.65%)**
- Remaining isolates were **ERG11 negative**

The single ERG11-positive isolate was identified as **Candida albicans** and showed **high-level resistance to fluconazole**.

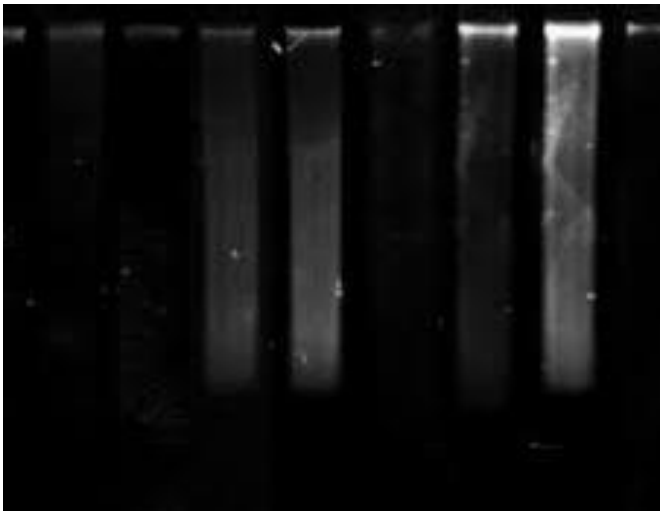


Figure: The Extraction for Gene

Molecular analysis for the ERG11 gene showed that **only 1 isolate (0.65%) was positive**, while the remaining isolates were negative. This table indicates that ERG11-mediated resistance is very rare in this study population. Despite the presence of phenotypic resistance, the low detection rate suggests that other mechanisms such as efflux pump activity or biofilm formation may be responsible for antifungal resistance.

DISCUSSION

Vulvovaginal candidiasis (VVC) remains a significant public health concern due to its high prevalence, recurrent nature, and emerging antifungal resistance. In the present study, the culture positivity rate of **74.8%** reflects a substantial burden of disease among symptomatic women and is comparable to earlier reports [1,2].

The predominance of VVC in the **reproductive age group (26–35 years)** observed in this study is consistent with previous findings, where hormonal factors and increased estrogen levels play a crucial role in Candida colonization [3,4]. Estrogen enhances glycogen deposition in vaginal epithelial cells, thereby providing a favorable environment for fungal growth [5]. A recent **2025 study** also reported a similar age distribution, with the majority of cases occurring in women aged 25–40 years [6].

The clinical presentation in the present study, with **vaginal discharge (88.3%) and pruritus (81.6%)** as the most common symptoms, correlates well with earlier studies [7,8]. These symptoms are attributed to inflammatory responses induced by Candida invasion and host immune reactions [9].

The microbiological profile of this study demonstrated that **Candida albicans (62.3%)** remains the predominant species, which is in agreement with several studies [1,10]. However, a considerable proportion (**37.7%**) of non-albicans Candida (NAC) species was observed. This shift toward NAC species has been increasingly reported worldwide and is of clinical concern due to their intrinsic resistance to commonly used antifungal agents [11,12]. A **2025 study** highlighted that non-albicans Candida species are emerging as important pathogens, particularly in recurrent and treatment-resistant cases [13].

Among NAC species, *Candida glabrata* was the most common isolate in the present study. This finding is consistent with recent literature, where *C. glabrata* has been increasingly associated with reduced susceptibility to azoles [14]. Similarly, a **2026 study from India** reported a rising trend of non-albicans Candida species and emphasized the importance of species-level identification in guiding therapy [15].

The antifungal susceptibility pattern observed in this study revealed a **high resistance to fluconazole (27.3%)**, which is comparable to recent global reports [16]. Fluconazole has been widely used as a first-line therapy, and its extensive use has contributed to the development of resistance [17]. A **2026 North Indian study** reported fluconazole resistance rates exceeding 30%, highlighting the growing challenge of azole resistance [18]. Likewise, a **2025 study** demonstrated increased resistance among Candida isolates, particularly among non-albicans species [19].

In contrast, **Amphotericin B (97.4%) and Voriconazole (89.6%)** showed high efficacy against Candida isolates in the present study. These findings are in agreement with previous studies that have demonstrated preserved susceptibility of Candida species to these antifungal agents [20]. However, their use is often limited by toxicity, cost, and availability [21].

One of the key highlights of the present study is the molecular analysis of antifungal resistance. The **ERG11 gene**, which encodes lanosterol 14-α-demethylase, is a major target of azole antifungal drugs. Mutations or overexpression of this gene can lead to decreased drug binding and subsequent resistance [22,23].

Interestingly, in the present study, **only one isolate (0.65%) was positive for the ERG11 gene**, despite the presence of phenotypic resistance. This finding suggests that ERG11-mediated resistance is not the predominant mechanism in this population. Similar observations have been reported in recent studies, where antifungal resistance was attributed to alternative mechanisms such as efflux pump overexpression (CDR1, MDR1 genes) and biofilm formation [24,25].

However, other studies have reported higher rates of ERG11 mutations. For example, a **2025 study** demonstrated mutation rates ranging from 37.5% to 48.6% among azole-resistant isolates [19]. The variation in findings may be due to differences in geographic distribution, antifungal usage patterns, and study methodologies.

The discrepancy between phenotypic resistance and molecular findings in the present study highlights the complexity of antifungal resistance mechanisms. It underscores the importance of comprehensive molecular studies involving multiple resistance genes rather than focusing solely on ERG11 [25].

Overall, the findings of this study emphasize the need for **continuous surveillance of Candida species distribution and antifungal resistance patterns**, especially in tertiary care settings. Early diagnosis, rational use of antifungal agents, and periodic monitoring of resistance trends are essential to prevent treatment failure and improve patient outcomes [21,24].

CONCLUSION

The present study demonstrates a **high prevalence of vulvovaginal candidiasis among symptomatic women**, with *Candida albicans* as the predominant isolate but a notable rise in non-*albicans Candida* species. Increasing resistance to commonly used azoles, particularly fluconazole, highlights the need for routine antifungal susceptibility testing. The very low detection of the ERG11 gene suggests that alternative resistance mechanisms may play a more significant role in this population, emphasizing the importance of continuous surveillance and molecular studies for effective management.

LIMITATIONS

- Single-center study with limited generalizability.
- Only ERG11 gene analyzed; other resistance mechanisms not evaluated.

DECLARATIONS:

Conflicts of interest: There is no 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.

REFERENCES

1. Sobel JD. Vulvovaginalcandidosis. Lancet. 2007;369(9577):1961–71.
2. Willems HME, Ahmed SS, Liu J, Xu Z, Peters BM. Vulvovaginal candidiasis: a current understanding and burning questions. J Fungi. 2020;6(1):27.
3. Gonçalves B, Ferreira C, Alves CT, Henriques M, Azeredo J, Silva S. Vulvovaginal candidiasis: epidemiology, microbiology and risk factors. Crit Rev Microbiol. 2016;42(6):905–27.
4. Farr A, Effendy I, Frey Tirri B, et al. Guideline: vulvovaginalcandidosis. Mycoses. 2021;64(6):583–602.
5. Achkar JM, Fries BC. Candida infections of the genitourinary tract. ClinMicrobiol Rev. 2010;23(2):253–73.
6. Arsić V, et al. Epidemiological trends of vulvovaginal candidiasis. Mycopathologia. 2025;190:101–110.
7. Donders GGG, et al. Pathogenesis of vulvovaginal candidiasis. BJOG. 2017;124(2):215–25.
8. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines. MMWR Recomm Rep. 2015;64(RR-03):1–137.
9. Fidel PL Jr. Immunity in vaginal candidiasis. CurrOpin Infect Dis. 2005;18(2):107–11.
10. Maraki S, et al. Epidemiology of Candida species. Mycoses. 2019;62(8):692–7.
11. Pfaller MA, Diekema DJ. Epidemiology of invasive candidiasis. ClinMicrobiol Rev. 2007;20(1):133–63.
12. Deorukhkar SC, Saini S. Non-albicans Candida infection: an emerging threat. InterdiscipPerspect Infect Dis. 2013;2013:615958.
13. Chitharagi VB, et al. Emerging non-albicans Candida in VVC. Front Fungal Biol. 2025;6:1695271.
14. Richter SS, et al. Antifungal susceptibilities of Candida species. J ClinMicrobiol. 2005;43(5):2155–62.
15. Yadav A, et al. Rising drug-resistant Candida infections in India. Res J Pharm Technol. 2026;19(3):145–150.
16. Pappas PG, et al. Clinical practice guideline for candidiasis. Clin Infect Dis. 2016;62(4):e1–50.
17. Whaley SG, Rogers PD. Azole resistance in Candida species. Front Microbiol. 2016;7:2173.
18. Singh A, et al. Increasing azole resistance in Candida infections. 2026 study, North India.
19. Sahu BK, et al. ERG11 mutations and azole resistance. Front Fungal Biol. 2025.
20. Pfaller MA, et al. Antifungal susceptibility trends. J ClinMicrobiol. 2010;48(4):1366–77.
21. Perfect JR. Antifungal resistance and therapy challenges. Nat Rev Drug Discov. 2017;16(9):603–16.
22. Flowers SA, et al. Gain-of-function mutations in ERG11. Antimicrob Agents Chemother. 2015;59(8):4505–13.
23. Sanglard D. Emerging threats in antifungal-resistant fungal pathogens. Front Med. 2016;3:11.
24. Pristov KE, Ghannoum MA. Resistance of Candida to azoles. Front Microbiol. 2019;10:7.
25. Golafshani FZR, et al. Genetic diversity and antifungal resistance in Candida species. 2025.