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Original Research Article

Vaginal *Candida* colonization, species distribution, associated factors and antifungal susceptibility patterns among women attending Bowen University Teaching Hospital, Ogbomoso

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ABSTRACT

Background: Vaginal colonization by *Candida* species is prevalent among women and may serve as risk factor to vulvovaginal candidiasis (VVC), especially when the vaginal microbiota is disrupted. However, the prevalence of vaginal *Candida* colonization (VCC), associated risk factors, and antifungal patterns remain limited in Nigerian settings. This study aimed to determine the prevalence, associated factors, and antifungal patterns of vaginal *Candida* isolated among women attending Bowen University Teaching Hospital, Ogbomoso, Nigeria.
Methods: This hospital-based cross-sectional study enrolled 176 consenting symptomatic and asymptomatic women between June and November, 2025. Data were collected using a semi-structured questionnaire and High vaginal swabs (HVS) were collected from participants. The HVS samples were examined using microscopy, culture on Sabouraud Dextrose Agar, CHROMagar, and Internal Transcribed Spacer-Polymerase Chain Reaction (ITS-PCR) for confirmation of *Candida*. Antifungal profiling was assessed by disk diffusion on Mueller-Hinton agar supplemented with glucose and methylene blue. Data were analyzed using SPSS version 26, with statistical significance set at $p \leq 0.05$.
Results: Among the 176 participants, 48(27.3%) participants were positive for VCC. *Candida parapsilosis* was the most frequent isolate, 25/48 (52.1%). In multivariate analysis, vaginal discharge (AOR=4.971, 95% CI: 1.485-16.646, p=0.009) and pregnancy (AOR=5.584, 95% CI: 1.344-23.209, p=0.018) were a strong significant predictor of *Candida* colonization. *C. parapsilosis* significantly emerged to be the most resistant to fluconazole (25(54.3%) (p=0.008).
Conclusions: VCC was common in this population, with predominance of *C. parapsilosis*. These findings provide local evidence supporting routine laboratory identification of *Candida* and antifungal testing, particularly in suspected, recurrent, persistent, or complicated VVC.

Keywords: Nigeria, Risk factor antifungal profiling, Vaginal *Candida* colonization, Vulvovaginal candidiasis

INTRODUCTION

Vulvovaginal candidiasis (VVC) is one of the most prevalent causes of vaginal infection in reproductive-aged women and is identified as a major cause of female morbidity worldwide[1]. It is caused by different species of *Candida* and is estimated to affect up to 75% of women at least once during their lifetime, with recurrent episodes occurring in a substantial proportion of affected women.[1,2] Clinically, VVC is characterized by symptoms such as Vulvovaginal pruritus, irritation, dysuria, dyspareunia, and abnormal vaginal discharge, which may impair quality of

life and daily functioning. However, laboratory detection of *Candida* from vaginal specimens may represent either colonization or symptomatic infection, depending on the clinical context; therefore, symptoms, microscopy, culture, and species identification remain important for accurate diagnosis and management.[2,3] Although *Candida albicans* remains the most frequently reported cause of VVC, increasing attention has been given to non-*albicans Candida* species because of their variable susceptibility to commonly used antifungal agents.[4,5]

In Nigeria, previous studies have documented variable *Candida* prevalence, species distribution, and antifungal susceptibility patterns among different female populations. Earlier Nigerian work by Adesiji et al reported *Candida* positivity in 26/104 (25.0%) sexually active young females, including both symptomatic and asymptomatic participants.[6] In that study, *C. albicans* accounted for 13/26 (50.0%) isolates, while non-*albicans Candida* species, including *C. glabrata*, *C. tropicalis*, and *C. krusei*, accounted for the remaining isolates. The same study also reported reduced susceptibility to commonly used antifungal agents, particularly fluconazole, supporting the need for continued surveillance of vaginal *Candida* species distribution and antifungal response patterns in Nigerian settings.[6] Other Nigerian studies have similarly reported variable species patterns among women with vaginal symptoms or pregnancy-associated colonization, emphasizing the importance of population-specific data.[7-9]

Pregnancy is a recognized predisposing factor for vaginal *Candida* colonization and VVC. This association is linked to hormonal changes, increased vaginal glycogen deposition, and physiological alterations in the vaginal microbiota during pregnancy.[10,11] Across African settings, recent studies continue to show that vaginal candidiasis and *Candida* colonization among pregnant women vary by population, diagnostic method, and local epidemiology. For example, Hussen et al reported a vaginal candidiasis prevalence of 26.8% among pregnant women attending antenatal care in southern Ethiopia, with *C. albicans* predominating but non-*albicans Candida* also present.[10] While colonization does not always indicate active disease, symptomatic and untreated VVC during pregnancy may be clinically important because it has been associated with adverse maternal and neonatal outcomes, including discomfort, ascending infection, chorioamnionitis (infection of the amniotic sac and placenta), premature rupture of membranes, preterm delivery, and neonatal candidiasis.[11,12]

Antifungal therapy remains central to VVC management, with azoles commonly used because of their availability, affordability, and ease of administration. However, empirical treatment without laboratory confirmation, prior antifungal exposure, incomplete treatment courses, and over-the-counter access to antifungal agents may contribute to reduced susceptibility among *Candida* isolates.[4,13] Recent studies continue to highlight the importance of antifungal susceptibility testing, particularly in settings where recurrent disease, non-*albicans Candida*, or reduced azole susceptibility may complicate empirical management.[10,14] Because *Candida* species distribution and antifungal susceptibility patterns vary by population, region, diagnostic method, and antifungal exposure history, periodic local surveillance remains important for guiding empirical treatment and laboratory-supported management.

Several clinical and behavioral factors have also been linked to vaginal *Candida* colonization (VCC) and VVC, including antibiotic use, hormonal factors, sexual activity, vaginal douching, diabetes mellitus, and hygiene practices.[15] The contribution of these factors may differ across study populations, making local assessment necessary. Building on earlier Nigerian evidence, including the work of Adesiji et al, the present study provides updated clinic-based data from an obstetrics and gynaecology population.[6] Specifically, this study aimed to determine the prevalence of vaginal *Candida* colonization, describe the species distribution of *Candida* isolates, identify associated clinical and behavioral factors, and evaluate the *in-vitro* antifungal susceptibility patterns of the isolates.

METHODS

Study design and setting

This was a hospital-based cross-sectional study conducted at the Obstetrics and Gynaecology clinics of Bowen University Teaching Hospital (BUTH), Ogbomoso, Oyo State, Nigeria. The hospital serves a semi-urban population and provides specialized obstetric and gynaecological services to women from Ogbomoso and surrounding communities. High vaginal swab samples were collected from consenting pregnant women attending the antenatal clinic and non-pregnant women attending the gynaecology clinic. Both symptomatic and asymptomatic women were included, and relevant clinical symptoms and possible risk factors were documented using a semi-structured questionnaire. Laboratory analysis was performed at the Microbiology Laboratory of LAUTECH Teaching Hospital, Ogbomoso, Oyo State, Nigeria. Specimen collection was carried out over a six-month period, from June to November 2025.

Study population and sample size

The study population consisted of 176 pregnant and non-pregnant consenting women aged 18 years and above attending the Bowen University Teaching Hospital between June and November. Women who had used antifungal medication within the previous two weeks and those who declined participation were excluded from the study. Fisher's formula for sample size determination as given below[16]:

$$n=\frac{Z^2pq}{d^2}$$

Where; n = the minimum sample size, Z standard normal deviation at 95% confidence interval = (1.96), p is the estimated prevalence of vaginal *Candida* colonization from a previous Nigerian study (40%; 0.4), q = 1-p (0.60).

Because the estimated accessible population during the study period was finite, a finite population correction was applied using the formula:

$nf = n / [1 + (n\text{-}1)/N]$

d= precision (allowable error) = 0.05^2= 0.0025

$$n=\frac{3.84*0.4*0.6}{0.0025}$$

n= 368.6, n≈ 369

To prevent overestimation and guarantee effective resource use, the initially computed sample size (assuming an infinite population) was modified using the finite population adjustment because the research population is known and finite.[16]

n= n/1+n/N

Where; n = initial estimated sample size and N = size of population of interest.

n= 369/1+369/300, n= 369/2.23, n= 165.

To adjust for non-response from the participants the sample size was adjusted to 176. A total of 176 participants were recruited using a consecutive non-probability sampling techniques until the rate of sample size was achieved.

Ethical consideration

Ethical approval was obtained from the Bowen University Teaching Hospital Health Research Ethics Committee and the research was carried out in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants. Confidentiality was maintained throughout the study, and participants received appropriate counseling and treatment based on laboratory findings. The institutional registration number was NHREC/12/04/2012, and the approval number was BUTH/REC-26860.

Specimen collection

High vaginal swabs (HVS) were collected aseptically by trained personnel using sterile cotton swabs. Each swab was placed into a sterile transport tube and immediately transported to the microbiology laboratory for processing. Using a sterile swab stick, specimens were collected from both the lateral vaginal wall and posterior fornices. Each swab was labeled with a unique study code, placed in a sterile leak-proof transport tube, and transported to the laboratory within 2 hours of collection at ambient temperature for immediate processing.

Primary culture and identification

Swabs were inoculated onto Sabouraud Dextrose Agar (SDA) supplemented with chloramphenicol to suppress bacterial contaminants. The plates were incubated aerobically at 35-37°C and examined after 24 hours and again after 48 hours for fungal growth. Plates with scant growth at 24 hours were re-examined after a further 24 hours before being considered culture-negative. Colony morphology on SDA was recorded. Yeast colonies were identified using standard phenotypic methods, including colony morphology, germ tube test, and Gram staining. Germ tube formation was used as a presumptive test for *Candida albicans*.

Direct microscopy

For wet mount examination, the swab tip was mixed in a drop of sterile normal saline on a clean glass slide, covered with a cover slip, and examined under ×10 and ×40 objective lenses for budding yeast cells, pseudohyphae, epithelial cells, and inflammatory cells. For direct gram staining, a thin smear was prepared on a clean glass slide, air-dried, heat-fixed, and stained using the conventional gram stain technique. The stained smear was examined under oil immersion for gram-positive budding yeast cells, and microscopy findings were recorded.

Indirect gram staining and germ tube test

Indirect gram staining was performed on colonies sub-cultured from SDA to support preliminary phenotypic identification. A small portion of a well-isolated colony was emulsified in sterile normal saline on a clean glass slide, air-dried, heat-fixed, gram stained, and examined under oil immersion for Gram-positive budding yeast cells and pseudohyphae.

The germ tube test was performed on all recovered yeast isolates to differentiate germ-tube-forming *Candida* from non-germ-tube-forming isolates. Germ tubes were identified as filamentous outgrowths arising directly from the yeast cell without constriction at the point of origin. Isolates showing germ tube formation were regarded as germ-tube-positive, while those without germ tube formation were regarded as germ-tube-negative pending further identification.

Molecular identification (PCR)

Molecular confirmation of *Candida* species was performed using genus-specific PCR targeting the Internal Transcribed Spacer (ITS) region. DNA extraction was

carried out using the boiling method. DNA quality and quantity were assessed spectrophotometrically by measuring absorbance at 260 nm and the A260/A280 ratio. Amplification was carried out using a ready-to-use 2× PCR Master Mix containing Taq DNA polymerase, dNTPs, $MgCl_2$, and reaction buffer. The primers used were ITS1 and ITS4. PCR products were analyzed by agarose gel electrophoresis using 1% agarose stained with ethidium bromide. Electrophoresis was run at 120 V for 30 minutes, and bands were visualized using a gel imaging system. Isolates with amplicons within the expected ITS band size range of *Candida* species were regarded as genotypically confirmed *Candida* isolates PCR amplification was performed using ITS-specific primers. Amplicons were visualized on 1% agarose gel electrophoresis. Bands within the expected size range of 500-800 bp confirmed *Candida* species identity.

CHROMagar Speciation

Isolates were further differentiated using CHROMagar *Candida*, which allows presumptive identification based on colony color and morphology. Colony appearances were interpreted as follows: *Candida albicans* - green; *Candida dubliniensis* - yellow-green; *Candida glabrata* - beige; *Candida krusei* - pink with pale edges; *Candida parapsilosis* - pale pink-white; and *Candida tropicalis* - blue.

Antifungal susceptibility testing

A standardized inoculum was prepared by suspending 3-5 well-isolated colonies in 3-5 mL of sterile 0.85% saline and adjusting the turbidity to 0.5 McFarland standard using the standard Clinical Laboratory Standards Institute (CLSI) method. The inoculum was used within 15 minutes of preparation. The surface of the agar plate was inoculated by swabbing in three directions to obtain a confluent lawn. After allowing the surface moisture to absorb for 3-5 minutes, antifungal disks were applied aseptically and plates were incubated inverted at 37°C. Zone diameters were read at 24 hours, and plates with poor growth were re-incubated and read at 48 hours. The diameters of inhibition zones were measured to the nearest millimeter. The breakpoints used to measure were; fluconazole; susceptible (≥19 mm), intermediate (15-18mm), resistance (≤14 mm); ketoconazole; susceptible (≥28 mm), intermediate (21-27 mm), resistance (≤20 mm); amphotericin B; susceptible (≥15 mm), intermediate (10-14 mm), resistance (≤10 mm) and nystatin; susceptible (≥23 mm), intermediate (16-22mm) and resistance (≤15 mm).[17] The choice of drugs used were selected according to the frequently used antifungals and also their availability.

Data analysis

Data were keyed into Microsoft Excel and imported into IBM SPSS Statistics version 26.0. Descriptive statistics (frequencies, percentages and charts) summarized participant characteristics, risk factors, prevalence and also the distribution of *Candida species*. Fisher's exact tests assessed associations between categorical variables and *Candida* isolation and a univariate and multivariate binary logistic regression was used to predict possible risk factor to Vulvovaginal candidiasis with statistical significance set at $p \leq 0.05$.

RESULTS

A total of 176 women attending the Obstetrics and Gynaecology clinics of Bowen University Teaching Hospital were enrolled in the study. The highest proportion of participants were aged 18-27 years, 88/176 (50.0%), followed by those aged 28-37 years, 62/176 (35.2%). Participants aged 38-47 years accounted for 18/176 (10.2%), while those aged 48 years and above accounted for 8/176 (4.5%). Most participants were married, 111/176 (63.1%), while 64/176 (36.4%) were single and 1/176 (0.6%) was widowed (Table 1).

Table 1: Socio-demographic characteristic of the participants (n=176).

Variables	Frequency	Percentage
Age group in years		
18-27	88	50.0
28-37	62	35.2
38-47	18	10.2
48 and above	8	4.5
Marital status		
Single	64	36.4
Married	111	63.1
Widow	1	0.6

Based on the reported associated risk among the participants, 39(22.2%) had irritation, 55 (31.3%) had vaginal discharge, 119 (67.6%) of the participants use douches. However, 29 (16.5%) of the participants were on antibiotics since the past 3 months, 38 (21.6%) reported having Vulvovaginal candidiasis in the past one year and 24 (13.6%) of the participants had UTI in the past one year. Moreso, 53 (30.1%) reported being sexually active with 71 (40.3%) been pregnant (Table 2).

Using both conventional and molecular diagnostic techniques, the prevalence of *Vulvovaginal candida* colonization emerge as 27.30% (n=48) (Figure 1). However, among the 27.3% prevalence of Vulvovaginal colonization, *Candida parapsilosis* emerge as the most isolated accounting for 25 (52.1%) of the colonization. *Candida albicans* (n=16 (33.3%), *Candida tropicalis* (n=4 (8.3%) and *candida glabrata* (n=3 (6.3%). The non-albicans species isolated was; *Candida parapsilosis, Candida tropicalis and candida glabrata* (Figure 2).

The association between demographics, risk factors, and *Candida* isolated, revealed a significant association between discharge and the *Candida* spp. with total of

20(41.7%) of the participants reported having discharge shows higher distribution of 10 (50.0%) and 8 (40.0%) among *C. albicans* and *C. parapsilosis* respectively at p value ≤0.05 (p=0.036). Likewise antibiotic usage for the past 3 months was significant associated with *Candida* spp distribution with *C. parapsilosis* having the highest distribution of 8(66.7%) at p value ≤0.05 (p=0.025). Also, Pregnant women who were positive for *Candida* was 13.1% (23/176) who were also asymptomatic. Therefore, pregnancy status is significantly associated with *Candida* species distribution with *C. parapsilosis* having higher distribution of 15(65.2%) among pregnant women at p value ≤0.05 (p=0.011). Other associated factors show no significant association with *Candida* species isolated (Table 3).

Table 2: Reported risk factor among participants (n=176).

Variables	Frequency	Percentage
Irritation		
Yes	39	22.2
No	137	77.8
Vaginal discharge		
Yes	55	31.3
No	121	68.8
Do you use douches		
Yes	119	67.6
No	57	32.4
Presence of STD		
Yes	5	2.8
No	171	97.2
Used antibiotics since the past 3 months		
Yes	29	16.5
No	147	83.5
Sexually active		
Yes	53	30.1
No	123	69.9
Pregnancy status		
Yes	71	40.3
No	105	59.7
Ever had vulvovaginal candidiasis in the past one year		
Yes	38	21.6
No	138	78.4
Ever had UTI in the past one year		
Yes	24	13.6
No	152	86.4

Predictors of vaginal Candida colonization

To determine the significant predictor of *Candida* colonization among participants. A univariate logistic regression shows no significant predictor of *Candida* colonization. However, after adjusting for confounders, Discharge emerged as a strong predictor of *Candida* colonization with people who reported having discharge were more likely to experience colonization (AOR=4.971, 95% CI:1.485-16.646, p=0.009). Likewise, pregnancy status was a significant predictor of *Candida* colonization with those who were pregnant being more likely to experience colonization compared to those who were not pregnant (AOR=5.584, 95% CI: 1.344-23.209, p=0.018) (Table 4).

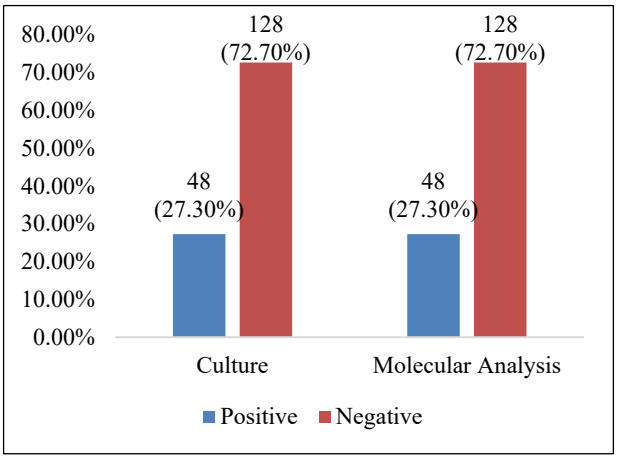

Figure 1: Prevalence of vaginal *Candida* colonisation using conventional and molecular techniques (n=176).

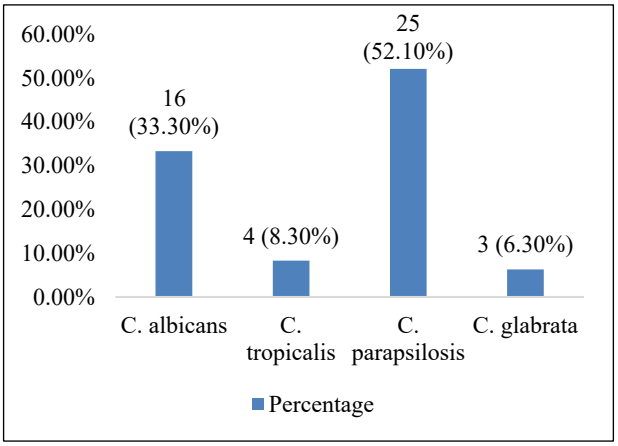

Figure 2: Distribution of *Candida Spp*. (n=48).

Table 3: Association between demographic, risk factor and *Candida* colonisation (n=48).

Variable	No examined	*C. albicans*, N (%)	*C. tropicalis*, N (%)	*C. parapsilosis*, N (%)	*C. glabrata*, N (%)	P value
Age group in years						
18-27	24	937.5)	2 (8.3)	12 (50.0)	1 (4.2)	
28-37	18	4 (22.2)	2 (11.1)	10 (55.5)	2 (11.1)	0.904
38-47	6	3 (50.0)	0 (0.0)	3 (50.0)	0 (0.0)	

Continued.

Variable	No examined	*C. albicans*, N (%)	*C. tropicalis*, N (%)	*C. parapsilosis*, N (%)	*C. glabrata*, N (%)	P value
Marital status						
Single	15	7 (46.7)	1 (6.7)	7 (46.7)	0 (0.0)	0.707
Married	33	9 (27.3)	3 (9.0)	18 (54.5)	3 (9.0)	
Presence of irritation						
Yes	11	5 (45.5)	1 (9.0)	4 (36.4)	1 (9.0)	0.476
No	37	10 (27.0)	3 (8.1)	21 (56.8)	3 (8.1)	
Presence of discharge						
Yes	20	10 (50.0)	2 (10.0)	8 (40.0)	0 (0.0)	0.036*
No	28	6 (21.4)	2 (7.1)	17 (60.7)	3 (10.7)	
Usage of douches						
Yes	32	9 (28.1)	2 (6.3)	18 (56.3)	3 (9.4)	0.580
No	16	7 (43.8)	2 (12.5)	7 (43.8)	0 (0.0)	
Presence of STD						
Yes	1	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0.600
No	47	15 (31.9)	4 (8.5)	25 (53.2)	3 (6.4)	
Used antibiotics since the past 3 months						
Yes	12	1 (8.3)	2 (16.7)	8 (66.7)	1 (8.3)	0.025*
No	36	15 (41.7)	2 (5.6)	17 (47.2)	2 (5.6)	
Sexually active						
Yes	12	7 (58.3)	1 (8.3)	4 (33.3)	0 (0.0)	0.257
No	36	9 (25.0)	3 (8.3)	21 (58.3)	3 (8.3)	
Pregnancy status						
Yes	23	3 (13.0)	2 (8.7)	15 (65.2)	3 (13.0)	0.011*
No	25	13 (52.0)	2 (8.0)	10 (40.0)	0 (0.0)	
Ever had vulvovaginal candidiasis in the past one year						
Yes	13	7 (53.8)	1 (7.7)	5 (38.5)	0 (0.0)	0.214
No	35	9 (25.7)	3 (8.8)	20 (57.1)	3 (8.6)	
Ever had UTI in the past one year						
Yes	9	5 (55.6)	1 (11.1)	3 (33.3)	0 (0.0)	0.207
No	39	11 (28.2)	3 (7.7)	22 (56.4)	3 (7.7)	

Note: *- represent significant value at p value≤0.05.

Table 4: Predictor of vulvovaginal *Candida* colonization.

Variable	Sub variable	Univariate		Multivariate	
		COR (CI)	P value	AOR (CI)	P value
Presence of irritation	Yes	1.062 (0.480-2.346)	0.882	0.775 (0.256-2.351)	0.653
	No	Reference			
Presence of discharge	Yes	1.898 (0.949-3.796)	0.070	4.971 (1.485-16.646)	0.009*
	No	Reference			
Use douches	Yes	0.943 (0.465-1.909)	0.869	0.488 (0.175-1.360)	0.170
	No	Reference			
Presence of STD	Yes	0.660 (0.072-6.054)	0.713	1.676 (0.133-21.069)	0.689
	No	Reference			
Used antibiotics since the past 3 months	Yes	2.176 (0.950-4.987)	0.066	1.885 (0.770-4.618)	0.165
	No	Reference			
Sexually active	Yes	0.707 (0.334-1.500)	0.366	0.565 (0.189-1.690)	0.307
	No	Reference			
Pregnancy status	Yes	1.533 (0.785-2.996)	0.211	5.584 (1.344-23.209)	0.018*
	No	Reference			
Ever had vulvovaginal candidiasis in the past one year	Yes	1.530 (0.707-3.312)	0.280	1.5272 (0.576-4.289)	0.377
	No	Reference			
Ever had UTI in the past one year	Yes	1.738 (0.705-4.289)	0.230	1.976 (0.605-6.458)	0.260
	No	Reference			

Note: *- represent significant value at p value≤0.05.

The antifungal profiling revealed that *C. parapsiolosis* were more resistant to amphotericin B (n=8(88.9%) as compared to other species and it is not statistically significant at p value ≥0.05 (p=0.147). *C. tropicalis* shows more sensitivity pattern to nystatin (n=3(42.9%), *C. parapsilosis* showed more intermediate pattern (n=16(53.3%) and resistant pattern (n=9(81.8%) and it is significant at p value ≤0.05 (p=0.000). Also, *C. tropicalis* emerged has the only species sensitive to fluconazole (n=2(100.0%) and *C. parapsilosis* emerge to be the most resistant to fluconazole (25(54.3%) and it is significant at p value ≤0.05 (p=0.008). However, *C. parapsilosis* emerged as the highest species with resistant pattern to ketoconazole (25(52.1%) and it is not statistically significant at p value ≥0.05 (p=0.177) (Table 5).

Table 5: Antifungal susceptibility profile across the *Candida* spp.

Antifungal Agents	*C. albicans*, N (%)	*C. tropicalis*, N (%)	*C. parapsilosis*, N (%)	*C. glabrata*, N (%)	P value
Amphotericin B					
Sensitive	15 (38.5)	4 (10.3)	17 (43.6)	3 (7.7)	
Intermediate	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0.147
Resistant	1 (11.1)	0 (0.0)	8 (88.9)	0 (0.0)	
Nystatin					
Sensitive	2 (28.6)	3 (42.9)	0 (0.0)	2 (28.6)	
Intermediate	13 (43.3)	0 (0.0)	16 (53.3)	1 (3.3)	0.000*
Resistant	1 (9.1)	1 (9.1)	9 (81.8)	0 (0.0)	
Fluconazole					
Sensitive	0 (0.0)	2 (100.0)	0 (0.0)	0 (0.0)	
Intermediate	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0.008*
Resistant	16 (34.8)	2 (4.3)	25 (54.3)	3 (6.5)	
Ketoconazole					
Sensitive	0 (0.0)	1 (50.0)	1 (50.0)	0 (0.0)	
Intermediate	2 (33.3)	1 (16.7)	2 (33.3)	1 (16.7)	0.177
Resistant	16 (33.3)	4 (8.3)	25 (52.1)	3 (6.3)	

Note: *- represent significant value at p value≤0.05.

Among the species *C. parapsilosis* had more resistant pattern across amphotericin B (88.90%), nystatin (81.80%), fluconazole (54.30%) and ketoconazole (52.10%) (Figure 3).

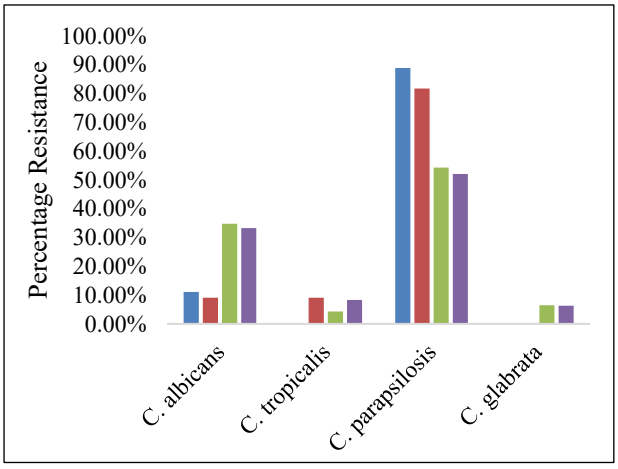

Figure 3: Resistant pattern among *Candida* isolates.

DISCUSSION

The aim of this study is to determine the risk factors, species distribution, and resistance patterns of antifungals to *Candida* species among women of childbearing age presenting to the Obstetrics and Gynecology, Bowen University Teaching Hospital (BUTH), Ogbomoso, Nigeria. Overall, the rate of VCC as determined in the current study was 27.3% using phenotypic testing and molecular analysis through PCR targeting the ITS of Candida species. This falls within the same range seen in other studies conducted in Nigeria where the prevalence rate of VCC has been shown to vary between 20-45% depending on certain variables such as populations studied and techniques used.[7,10] This suggests that VCC remains common in the region, and there is a necessity for routine screening during obstetrics and gynecology examination.

Among the Candida species, *Candida parapsilosis* was predominant in our study population, accounting for 52.1% as identified using CHROMagar. Others were *C. albicans* (33.3%), *C. tropicalis* (8.3%), and *C. glabrata* (6.3%). It is noteworthy that the prevalence rate of *C. parapsilosis* seems interesting because, although *C. albicans* is often reported as the commonest cause of VVC, many studies have shown contradictory results.[4,5] Indeed, many reports from various African countries indicate increased rates of non-albicans *Candida* infection. Such an observation can be explained by the fact that *Candida parapsilosis* strains thrive in regions where antibiotics are used extensively, and prior studies have not been strict in their identification criteria.[18,19]

The analysis of risk factors and their relationships with VCC revealed some notable insights. vaginal discharge and pregnancy, which were significantly strong predictors of VVC. Vaginal discharge is a characteristic symptom in cases of candidiasis and, moreover, its presence clearly identifies the colonization and/or infection by these fungi.[20] One of the independent factors that contributed to *Candida* colonization was pregnancy, which had a 5.6 increased risk of colonization in pregnant women compared to those who were not pregnant. In the study sample of pregnant patients with a culture-positive result, *C. parapsilosis* was found to be prevalent at 65.2%. This study finding is interesting possibly because of the influence of pregnancy on the vaginal microbiome and hence the growth of the fungus.[2,21] Although *Candida* colonization in pregnant women is mainly dominated by *C. albicans*, the higher prevalence rate of *C. parapsilosis* in this study may be explained by ecological reasons like antifungal treatment and NAC endemics.[22]

Antibiotic use in the past three months was found to be significantly associated with the prevalence of *Candida* species, since 66.7% of isolated pathogens were *Candida parapsilosis* among patients receiving antibiotics. Although antibiotic use did not significantly predict *Candida* colonization. Use of antibiotics is known to disrupt the vaginal flora dominated by lactobacilli and thereby decrease colonization resistance providing a suitable environment for *Candida* overgrowth.[23] The prevalence of *Candida parapsilosis* obtained in antibiotic users may be explained by the advantage that this pathogen has in terms of propagation when antibiotic usage modifies microbial communities. *Candida parapsilosis* has been reported to thrive when selected for by the presence of antibiotic therapy, particularly, a wide spectrum antibacterial agent (Trofa et al, 2008). Frequent douching practice (67.6%) observed in this investigation was not linked with particular species colonization. However, this behavior was highlighted as a risk factor of importance since intra-vaginal douching was revealed as a risk factor for development of bacterial vaginosis and *Candida* colonization in African women.[20]

The results on susceptibility are rather alarming as there is resistance to all tested antifungal discs used with *Candida parapsilosis* showing the highest resistance. Specifically, *C. parapsilosis* exhibited resistance in 88.9% of isolates tested against amphotericin B, 81.8% against nystatin, 54.3% against fluconazole, and 52.1% against ketoconazole. Resistance to nystatin and fluconazole by *Candida parapsilosis* isolates showed a significant association. It is a cause for concern as the two drugs are widely prescribed for first-line and second-line drug treatment of VVC infection due to the unavailability of systemic alternatives such as echinocandins and newer triazoles.

The reduced fluconazole sensitivity observed in the present study also aligns with earlier findings by Adesiji et al, where 19/26 (73.0%) vaginal *Candida* isolates were resistant to fluconazole.[6] Together, these findings suggest that reduced azole susceptibility among vaginal *Candida* isolates has been a persistent concern in this setting and supports the need for routine species identification and susceptibility-guided therapy. None of the cases of *C. albicans* and *C. glabrata* were susceptible to fluconazole while only *C. tropicalis* exhibited full susceptibility. Lack of susceptibility to fluconazole in *C. albicans* is highly alarming, considering that in previous studies, *C. albicans* is susceptible to the antifungal agent. Clearly, there exists some degree of resistance which arose because of self-medication in the area over a considerable time period. Studies showing similar results have also been conducted by other scholars in different parts of the country. For instance, fluconazole resistance in *C. albicans* has been reported by Olowe et al in the Osun State region.[7] They associated such a resistance to availability and misuse of fluconazole.

Nevertheless, resistance to amphotericin B was not significant among all the species under investigation. However, the emergence of resistance to amphotericin B is still a major health concern. Resistance to amphotericin B in *C. parapsilosis* isolated in this study population was rather high (88.9%), particularly considering recent evidence of NAC resistance to amphotericin B. According to reports made by Pappas et al, *C. parapsilosis* can acquire resistance to amphotericin B through mutation in membrane ergosterol composition, making surveillance necessary for determination of therapeutic options.[4]

Another topical polyene commonly used to manage un-complicated cases of VVC is nystatin; however, relatively high resistance to nystatin was observed in this study population, where 81.8% of *C. parapsilosis* strains were resistant. There was an association between significant cross-resistance between nystatin and fluconazole in *C. parapsilosis* isolates, suggesting that there is a similar resistance mechanism among these species.

The resistance to ketoconazole was also high, with the highest level of resistance being shown by *Candida parapsilosis* at 52.1%. Nonetheless, there were no differences in resistance levels between the species. The use of ketoconazole has the same pharmacology as fluconazole, and resistance to both is expected. This resistance again narrows down the options for treatment. In conclusion, according to the current findings, one may say that the existence of multiple fungus and multiple azole resistance is illustrated, mainly by *C. parapsilosis.*

This study derives it's strength based on the study population used as their high risk to increased colonization among women of child bearing age which could potentially cause VVC. Also, the incorporation of ITS-PCR in this study increased its strengths. However, from the results obtained in the present study, it could be observed that the treatment regimens that are employed in the current practice within this region may not be adequate for this particular set of *Candida* strains and the way they

develop resistance. There is a need to incorporate tests for species identification and resistance profiling within the regular clinical practice. In future research investigations, the determination of the minimum inhibitory concentrations and the resistance profile by molecular techniques should be carried out for more isolates in Nigeria.

CONCLUSION

This study provides evidence supporting the need for epidemiological patterns and possible risk factors to VCC among women of child-bearing age. This study reported a significant 27.3% rate of VCC with a shift in the *Candida species* distribution towards non-albicans. Factors such as vaginal discharge and pregnancy were reported to predispose women of child bearing age to VCC. However, high resistance to antifungal agents such as amphotericin B, fluconazole and ketoconazole was also reported revealing the need to improve antimicrobial stewardship.

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