Vulvovaginal Candidiasis at Institute Pasteur of Dakar, Senegal: Prevalence and Associated Risk Factors

Babacar Ndiaye a*, Khadim Diongue b,c, Abdoulaye Seck a,d, Abdou Diop a, Mame Diarra Diouf a, Assane Dieng d,e, Khadidiatou Mangou f, Lauriane Koko Marcel Koumondji a, Thierno Abdoulaye Diallo a, Chantal Mahou a, Philippe Dubrous a and Cheikh Saad Bouh Boye d,e

a Laboratory of Medical Biology, Pasteur Institute, Dakar, Senegal.
b Laboratory of Parasitology and Mycology, Aristide Le Dantec Hospital, 30 Avenue Pasteur, BO 3001, Dakar, Senegal.
c Department of Parasitology-Mycology, Cheikh Anta DIOP University of Dakar, BO 3005, Dakar, Senegal.
d Bacteriology-Virology Laboratory, Cheikh Anta DIOP University of Dakar, BO 3005, Dakar, Senegal.
e Bacteriology-Virology Laboratory, Aristide Le Dantec Hospital, 30 Avenue Pasteur, BO 3001, Dakar, Senegal.
f Immuno-Pathophysiology and Infectious Diseases Laboratory, Pasteur Institute, Dakar, Senegal.

Authors’ contributions
This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information
DOI: 10.9734/JAMB/2022/v22i10670

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/92131

Received 09 July 2022
Accepted 17 September 2022
Published 26 September 2022

ABSTRACT

Vulvovaginal candidiasis (CVV) is a superficial mycosis caused by Candida spp. with a predominance of C. albicans. CVV is opportunistic with several incriminated risk factors. This study aimed to determine the prevalence of CVV and to investigate potential risk factors. A cross-sectional study was carried out at the Medical Biology Laboratory in Pasteur Institute in Dakar, Senegal from September 1 to November 30, 2020. The study cohort was inclusive of all women received for a vaginal swab test in the laboratory unit. Each patient’s socio-demographic

*Corresponding author: E-mail: babacar.ndiaye@pasteur.sn;
and clinical data were recorded before collecting two swabs samples for direct examination and culture on CHROMagar Candida incubated at 37 °C for 24 to 48 h. A total of 312 women with an average age of 32 years (range: 17-74) were included in this study. An overall CVV prevalence of 32% was found. C. albicans was predominant (73.2%) followed by C. glabrata (16.8%). The age group [30-40] years was more infested with 35.3% (p = 0.434) as well as nulliparous or primiparous women with 38.7% (p = 0.171). CVV was more associated with disordered vaginal flora and pregnancy with 35.2% (p = 0.323) and 33.7% (p = 0.715) respectively. CVV was significantly related to contraception (p = 0.014). An overall high prevalence of CVV was observed mainly due to C. albicans. Contraception seems to be a factor contributing to its occurrence.

Keywords: Candidiasis; species; risk factors.

1. INTRODUCTION

Vulvovaginal candidiasis (VVC) is a cosmopolitan, opportunistic superficial mycosis caused by commensal yeast belonging to the genus Candida [1]. The transition from colonization to the onset of symptomatic infection is associated with multiple factors including host susceptibility, host inflammatory reactions, and virulence factors of the Candida species involved. The species responsible are varied with a predominance of C. albicans and C. glabrata [2].

Clinical symptoms of VVC are thought to be caused by an overabundance of yeast and its penetration into the vulvovaginal epithelial cells [3]. This clinic is characterized by vulvar pruritus and whitish, curdled leucorrhoea which will cause significant discomfort for the patients [4,5].

VVC is also known for its high direct or indirect economic cost associated with its management [5]. Indeed, CVV is often recurrent (RVVC) characterized by four or more episodes per year [3].

Several risk factors have been implicated in the occurrence of CVV, including sexual activity, recent use of antibiotics, pregnancy, and immunosuppression due to situations such as poor control of HIV infection or diabetes [6,7].

Regarding the frequency of VVC with around 75% of women contracting it at least once in their lifetime, i.e. 138 million per year worldwide [8], we volunteered to participate in the documentation of VVC in Senegal. More specifically, it involved determining the prevalence of VVC, to identify the species involved and to specify potential associated risk factors.

2. MATERIALS AND METHODS

This is a cross-sectional and descriptive study carried out in the Medical Biology Laboratory of the Pasteur Institute in Dakar (Senegal) from September 1 to November 30, 2020.

The study population included women received in the laboratory for microbiological analysis of vaginal swab (VS) and who met the required conditions. Sociodemographic and clinical data (age, type of flora, parity, contraception) was recorded for each patient included before sample collection.

VS were sampled by collecting vaginal discharges or secretions mainly found in the posterior fornices after placement of a speculum unless contraindicated. Two swabs were used, one for direct examination and the other for culture. For direct examination, we performed a microscopic examination after Gram stain for flora typing with Nugent scores [9]. The culture was realized by inoculating the samples on CHROMagar Candida (Becton Dickinson, USA) chromogenic medium which was then incubated at 37°C. The cultures were read after 24 and 48 hours.

The mycological diagnosis of VVC was retained by a positive direct examination associated with a positive culture showing the presence of 10 yeast colonies at least. The presumptive identification of Candida species was performed according to the manufacturer’s instructions according to the following color code: light to medium green (C. albicans), light rose to pink with a whitish border (C. glabrata), blue greenish to metallic blue with or without violet halos (C. tropicalis), fuchsia pink (C. krusei), yellow (C. parapsilosis).

For statistical analysis, collected data were entered into MS Excel 2010 and then transferred
to the SPSS 20.0 software with which they were analyzed. Pearson’s chi-square test was used to compare the differences and significance was considered if p <0.05.

3. RESULTS

In sum, 312 women were included in the study with a mean age of 32 years ranging from 17 to 74 years. The most represented age group was between 30 and 40 years old with 136 women (43.6%). Women with normal vaginal flora accounted for 53.8% (n = 168) and 56.4% (n = 174) of women were primiparous or nulliparous. Pregnant women represented 23.7% (n = 74) and those using contraception 9.9% (n = 31).

Out of these 312 women included, VVC was diagnosed in 100 corresponding to an overall prevalence of 32%.

The distribution of VVC according to the species involved showed a large predominance of *C. albicans* followed by *C. glabrata* with 73% and 16% respectively. Two patients presented co-infection with *C. albicans* associated with *C. glabrata* or *C. krusei* (Fig. 1).

The VVC infestation index was found more in the age group between 20 and 40 years with 73% (*p* = 0.0261) and in nulliparous and/or primiparous women with 35.2% (*p* = 0.171). The infection was more associated with a disordered flora with a frequency of 38.3% (*p* = 0.323). VVC was found in more pregnant women with a frequency of 33.8% (*p* = 0.715). No correlation between VVC and the following factors was found: age, parity, type of vaginal flora, and pregnancy (Table 1). However, VVC was significantly related to contraception with a frequency of 51.6% (*p* = 0.014).

---

**Fig. 1. Distribution of Candida infection by species**

**Table 1. Sociodemographic and clinical characteristics of the patients (n = 312)**

| Characteristics                  | Examined | Positives | p-value |
|----------------------------------|----------|-----------|---------|
|                                  | n (%)    | n (%)     |         |
| Age group (Years)                |          |           |         |
| <20                              | 2 (0.6)  | 1 (1)     | 0.261   |
| 20 - 40                          | 210 (67.3)| 73 (73)  |         |
| ≥ 40                             | 100 (32.1)| 26 (26)  |         |
| Parity                           |          |           |         |
| ≤ 1                              | 84 (54.9)| 62 (35.2)| 0.171   |
| ≥ 2                              | 15 (9.8)| 38 (27.9)|         |
| Type of flora (Nugent score)     |          |           |         |
| Normal                           | 168 (53.8)| 48 (28.6)|         |
| Intermediate                     | 84 (26.9)| 29 (34.5)| 0.323   |
| Imbalanced (vaginosis)           | 60 (19.2)| 23 (38.3)|         |
Characteristics | Examined | Positives | p-value
--- | --- | --- | ---
Pregnancy | n (%) | n (%) |  
Yes | 74 (27.3) | 25 (33.8) | 0.715  
No | 238 (76.7) | 75 (31.5) |  
Contraception | n (%) | n (%) |  
Yes | 31 (9.9) | 16 (51.6) | 0.014  
No | 281 (90.1) | 84 (29.9) |  

4. DISCUSSION

VVC is one of the most frequent infections of the vulvovaginal tract in women [10], representing a very serious pathology with a considerable economic burden related to its management.

In our study, the prevalence of VVC was estimated at 32%.

Prevalence of VVC was previously estimated in Senegal at 24% in 2006 during a national survey, at 34.8% in 2008 in Aristide Le Dantec university hospital and at 27.2% in 2015 in Ouakam military hospital in Dakar region respectively according to citation of Diongue et al, (2018) [1].

Globally, the prevalence of this infection is variable [5]. In Africa, a prevalence of 22.8% was reported in Morocco in 2009 [11], 38.9% in Benin in 2014 [5], 41.3 in Ivory Cost in 2011 [12], and 26% in Mauritania [10]. In Europe, high prevalence has also been reported, particularly in Sweden (42.0%) and Italy (43.5%) [13,14]. Similar results were reported in Turkey with a prevalence of 40.0% [15].

Our results showed that C. albicans was the most isolated species (73.2%) followed by C. glabrata (16.8%).

This same trend has been reported by almost all authors, particularly in Gabon where C. albicans and C. glabrata were isolated at 70.2% and 9.6% respectively [16] as well as in Benin where C. albicans was isolated at 96.1% followed by C. glabrata with 3.9% [5]. In several other studies, proportions ranging from 60 to 90% for C. albicans were reported especially in Brazil and in Tunisia [2,17].

The predominance of C. albicans could be explained by its ability to adhere to the vaginal mucosa by the expression of virulence factors including germ tub formation and switch from the saprophytic state (yeast form) to the pathogenic state (filamentous form). [5,18]. Thus, C. albicans which is a commensal yeast of the genital and gastrointestinal tract, is responsible for 85 to 90% of VVCs. However, in recent years, there has been an emergence of "non albicans" Candida species, essentially C. glabrata isolated with a prevalence of 5 to 15% of VVC cases while C. parapsilosis, C. tropicalis, and C. krusei are less rarely isolated [17,19].

"Non-albicans" species (15 to 47%) have been particularly involved in the pathogenesis of RVVC with a predominance of C. glabrata ranging from 6 to 29% [17].

The emergence of "non-albicans" species could be explained by selective pressure due to prolonged antifungals drugs exposure in women suffering from RVVC [20].

Several risk factors are associated with the occurrence of VVC. During this study, we observed that VVC was more associated with disordered vaginal flora such as vaginosis.

Such an observation has already been found in Benin [5]. This could be explained by the fact that Candida spp. behaves as opportunists in this favorable environment for the development of microorganisms habitually considered as commensals. Indeed, once again C. albicans is an opportunistic yeast of the vaginal mucosa which can switch from commensal form to pathogenic form according to a disorder of the stability between the local host immune system and the virulence mechanisms of the fungus. This disorder induces the expression of virulence factors by the yeast and the colonization of the vaginal mucosa [17].

Regarding age, VVC has been observed more in women aged between 20 and 40 years who can be considered a sexually active group.

This result is phase with those several other authors [5,21,22]. Certain very common practices among women in this age group such as using particular clothing or undergarments (made of synthetic fabric and tight clothing), intimate
hygiene (frequent use of antiseptic products) as especially intercourse or oro-genital, are emerging as potential factors in VVC [13].

The increase in hormonal activity, especially estrogenic which is associated with highest sexual activity at this age would explain why that age is a risk factor as reported by several authors [18,23]. The frequency of VVC tends to decrease as we observed in our study.

We also noticed that VVC was more associated with pregnancy although we did not find a statistically significant difference, the same has been reported in several other studies [5,10,17]. Indeed, this could be the result of a strong hormonal change; pregnancy corresponds to a period of greatest risk. The hormonal disorder observed during this period (predominant role of progesterone) causes a modification of the vaginal epithelium and a drop in vaginal pH, allowing the implantation of yeasts of the genus Candida. Furthermore, this high concentration of progesterone is also responsible for increasing the glycogen content in the vaginal tissue which provides a source of carbon for Candida, thus promoting their multiplication [5].

Likewise, CVV was significant more found among women under contraception. Indeed, contraception is a situation quite similar to pregnancy with a hormonal disorder thus promoting the proliferation of Candida spp. Mechanical contraceptives (intrauterine device-IUD, vaginal ring) contribute to the pathogenesis of VVC/RVVC. Recent studies show that C. albicans have a high capacity for adhesion and production of biofilm on the surface of the IUD, allowing them to escape host immunity and reduce their sensitivity to antifungals [17,24].

Contrasting, we did not find any correlation between CVV and multiparity. This agrees with the results of other studies carried out in particular in Morocco and in Benin [5,11].

5. CONCLUSION

Our study showed that CVV is a common condition, especially found in women aged between 20 and 40 years, as well as in an vaginal flora disorder, in pregnant women or under contraception. Despite the high frequency of CVV in its clinical situations, we have only found a correlation of CVV with contraception. Identifying the species involved and the contributing factors could be an important well as the frequency of sexual practices, element in better understanding the epidemiology and pathophysiology of this infection.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

ACKNOWLEDGEMENT

We would like to thank all the colleagues in the medical biology laboratory for their unwavering investment in carrying out this work, particularly in the bacteriology unit. We would also like to thank all of the study participants.

Special mention to employees of the Parasitology-Mycology and Bacteriology-Virology departments of the Aristide Le Dantec Hospital.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Diongue K, Baha Z, Seck MC, Ndiaye M, Diallo MA, Ndiaye D. Cutaneo-ungual candidiasis diagnosed in the parasitology and mycology laboratory of Le Dantec University Hospital in Dakar from 2008 to 2015. Med Health Too. 2018;390-4.

2. Mascarenhas REM, Machado M SC, Costa e Silva BFB da, Pimentel RFW, Ferreira TT, Leoni FMS, et al. Prevalence and risk factors for bacterial vaginosis and other vulvovaginitis in a population of sexually active adolescents from Salvador, Bahia, Brazil. Infect Dis Obstet Gynecol. 2012; 378640.

3. van Schalkwyk J, Yudin MH, Yudin MH, Allen V, Bouchard C, Boucher M, et al. Vulvovaginitis: Screening and management of trichomoniasis, vulvovaginal candidiasis and bacterial vaginosis. Journal of Obstetrics and Gynaecology Canada. 2015;37(3):275-6.

4. Ringdahl EN. Treatment of recurrent vulvovaginal candidiasis. Am Fam Physician. 2000;61(11):3306-12,3317.

5. Ogouyémi-Hounto A, Adisso S, Djamil J, Sanni R, Amangbegnon R, Biokou-
1. Bankole B, et al. Place of vulvovaginal candidiasis in lower genital infections and associated risk factors in women in Benin. Journal of Medical Mycology. 2014;24(2):100-5.

2. De Leon EM, Jacober SJ, Sobel JD, Foxman B. Prevalence and risk factors for vaginal Candida colonization in women with type 1 and type 2 diabetes. BMC Infect Dis. 2002;2:1.

3. Ohmit SE, Sobel JD, Schuman P, Duerr A, Mayer K, Rompalo A, et al. Longitudinal study of mucosal Candida species colonization and candidiasis among human immunodeficiency virus (HIV)-seropositive and at-risk HIV-seronegative women. J Infect Dis. 2003;188(1):118-27.

4. Denning DW, Kneale M, Sobel JD, Rauteamaa-Richardson R. Global burden of recurrent vulvovaginal candidiasis: a systematic review. Lancet Infect Dis. 2018;18(11):e339-47.

5. Delaney ML, Onderdonk AB. Nugent score related to vaginal culture in pregnant women11The Microbiology and Prematurity Study Group consists of the following: Robin Ross, Ph.D., Mei-Ling Lee, Ph.D., Andrea M. DuBois, BS, Wendy Osterling, BS, and David G. Aiello, BS, Channing Laboratory, Brigham and Women's Hospital, Boston, MA; and Ruth Tuomala, MD, Ellice Lieberman, MD, Amy Cohen, BA, Dorothy Pender, RN, and Linda Steele, MT(ASCP), Department of Obstetrics and Gynecology, Brigham and Women's Hospital, Boston, MA. Obstetrics and Gynecology. 2001;98(1):79-84.

6. Sy O, Diongue K, Ahmed CB, Ba O, Moulay FC, Lo B, et al. Vulvovaginal candidiasis in pregnant women at the Mother and Child Hospital in Nouakchott (Mauritania). Journal of Medical Microbiology. 2018;28(2):345-8.

7. Benchellal L, Guelzim K, Lemkhente Z, Jamili H, Dehainy M, Rahali Moussaoui D, et al. Vulvovaginal candidiasis at the Mohammed V military instruction hospital (Morocco). Journal of Medical Microbiology. 2011;21(2):106-12.

8. Masson E. Sensibilité in vitro des souches de Candida albicans d’origine vaginale aux antifongiques à Abidjan (Côte d’Ivoire) [Internet]. EM-Consulte. [cité 26 déc 2020]. Disponible sur: https://www.em-consulte.com/article/728149/sensibilite-in-vitro-des-souches-de-candida-albicans

9. Rylander E, Berglund A, Krassny C, Petrini B. Vulvovaginal candida in a young sexually active population: prevalence and association with oro-genital sex and frequent pain at intercourse. Sex Transm Infect févr. 2004;80(1):54-7.

10. Corsello S, Spinillo A, Osengo G, Penna C, Guaschino S, Beltrame A, et al. An epidemiological survey of vulvovaginal candidiasis in Italy. Eur J Obstet Gynecol Reprod Biol. 2003;110(1):66-72.

11. Ilkit M, Guzel AB. The epidemiology, pathogenesis, and diagnosis of vulvovaginal candidiasis: a mycological perspective. Crit Rev Microbiol. 2011;37(3):250-61.

12. Amouri I, Abbas S, Sellami H, Makni F, Sellami A, Ayadi A. Vulvovaginal candidiasis: review. Journal of Medical Mycology. 2014;24(2):e87.

13. Sobel JD. Vulvovaginal candidosis. Lancet. 2007;369(9577):1961-71.

14. Sobel JD. Vulvovaginitis due to Candida glabrata. An emerging problem. Mycoses. 1998;41 Suppl 2:18-22.

15. Reid G, Dols J, Miller W. Targeting the vaginal microbiota with probiotics as a means to counteract infections. Curr Opin Clin Nutr Metab Care. 2009;12(6):563-7.

16. An epidemiological study of vulvovaginal candidiasis in women of childbearing age Jindal N, Gill P, Aggarwal A - Indian J Med Microbiol [Internet]. [cite 26 dec 2020]. Webpage: https://www.ijmm.org/article.asp?issn=0255-0857;year=2007;volume=25;issue=2;spage=e175;epage=176;aulast=Jindal

17. Tarry W, Fisher M, Shen S, Mawhinney M. Candida albicans: the estrogen target for vaginal colonization. J Surg Res. 2005;129(2):278-82.

18. Anane S, Kaouech E, Zouari B, Belhadj S, Kallel K, Chaker E. Vulvovaginal candidiasis: risk factors and clinical and mycological particularities. Journal of Medical Mycology. 2010;20(1):36-41.
24. Auler ME, Morreira D, Rodrigues FFO, Abr ao MS, Margarido PFR, Matsumoto FE, et al. Biofilm formation on intrauterine devices in patients with recurrent vulvovaginal candidiasis. With Mycol. 2010;48(1):211-6.