

Original Article

Frequency Distribution of Candidal Vaginitis in Women Referred to Health Centers in Yazd

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Abstract

Introduction: The vaginal flora is a dynamic ecosystem that can be easily altered. There are four causes of vaginal discharges which cover almost 95% of vaginitis. Candidal Vaginitis is an infection of vagina's mucous membranes by *Candida albicans* (CA). The present study attempted to study Candidal Vaginitis in women referred to health centers in Yazd and to investigate the role of innate & cell-mediated immunity against Candidal Vaginitis.

Materials and Methods: This was a cross-sectional study conducted from September 2011 to September 2012 in Yazd city. A total of 360 women were recruited. The collection of material for diagnosis was ideally performed during a comprehensive pelvic examination using a speculum for diagnosis of CA by identifying vaginal pH, smear preparing and staining, and culturing.

Results: Finally, of all the 360 women that were observed and tested, 120 (33.33%) were involved and 240 (66.67%) were not involved. Of the 120 infected women, 55 patients had Bacterial vaginosis (15.6%), 40 patients had Ca (10.8%) and 25 patients had *Trichomonas vaginalis* (5.9%). Despite the role for cell-mediated immunity in host protection against the majority of mucosal CA infections, studied has been in women with recurrent vaginitis, in HIV-infected women. Thus, current investigations have been focused specifically on innate and acquired immune responses against CA at the vaginal mucosa instead of Vulvovaginal Candidiasis being caused by defective or dysfunctional CD4⁺ T helper 1-type cell-mediated immune reactivity.

Conclusion: It is strongly suggested that distinct vaginal lymphocyte subsets participate in the adaptive anti-Candida immunity at the vaginal level, with the vaginal CD4⁺ T cells probably playing a major role.

Keywords: Candidal vaginitis, Women, Yazd, Frequency distribution.

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Introduction

Vaginitis is an inflammation of the vagina and affects women of all ages. Infections and allergies can cause vaginitis. Inflammation occurs when hormones, hygiene, bacteria, or sexual activity throw off the delicate balance of microorganisms inside the vagina. Vaginitis symptoms and treatment differ depending on the causative agent: bacteria, the protozoan parasite *Trichomonas vaginalis*, yeast, an allergen, or a decrease in estrogen. About 75% of all women will suffer from at least one yeast infection; half will have more than one during their lifetimes. Some vaginal infections, such as bacterial vaginosis, put women at a greater risk for more serious conditions, such as premature delivery of a baby.

Of the millions of cases of vaginitis each year, most are caused by bacterial vaginosis (about 40% to 50%), followed by yeast infections (20% to 25%) and then trichomoniasis (15% to 20%). Some types of vaginitis can be sexually transmitted^[1]. If vaginitis results from a sexually transmitted disease, some research says that partners also should receive treatment. In addition, women with bacterial vaginosis and trichomoniasis may have been exposed to other sexually transmitted organisms, such as *N. gonorrhoeae* that causes gonorrhea, and the human immunodeficiency virus (HIV) that causes AIDS. Unless women who have chlamydiosis or gonorrhea receive treatment, up to 40% will develop pelvic inflammatory disease, according to the Centers for Disease Control and Prevention^[2]. Candida infections account for approximately 33% of all vaginitis cases^[3]. Candida

vaginitis is most common in pre-menopausal women. As estrogen levels are a contributing factor, reproductive-aged women (especially those using oral contraceptives) and postmenopausal women using estrogen therapy are at greatest risk^[4,5]. Candida vaginal colonization is thought to occur in 10% to 55% of healthy, asymptomatic women. Infection rates are estimated at a single episode in 75% of reproductive-aged women, more than one episode in 45%, recurrent infection in 5%, and persistent/chronic infection in 1%. There is a relationship between the occurrence of yeast infections and the frequency of sexual activity and the use of injectable progestin contraception. Approximately 70% of young, sexually active women will experience vaginal colonization with yeast at some time during a 1-year period. *Candida albicans* is responsible for 90% to 92% of Candida Vulvovaginitis. However, *Candida glabrata* *Torulopsis glabrata* and other *Candida* spp are appearing with increasing frequency. Visualization of the yeast buds (Pseudohyphae) is important for clinical diagnosis with microscopic saline/potassium hydroxide (KOH) wet-mount examination. The present study attempted to study Candidal Vaginitis in women referred to health centers in Yazd and to investigate the role of innate & cell-mediated immunity against Candidal Vaginitis.

Materials and Methods

This was a cross-sectional study conducted from September 2011 to September 2012 on patients

referred to the health Centers in Yazd city. Informed consents were signed by all female participants who agreed to participate in this research study. In each case, sociodemographic findings were obtained clinically and mycologically. Vaginal discharge specimens were collected from 360 female patients (15-45 years of age) who had clinical symptoms such as discharge and vulval Pruritus. Laboratory diagnosis was based on microscopic examination of the specimens. Two slides were prepared from the vaginal discharge and stained with methylene blue. Additional swab samples were put into the tubes containing 2 ml sabouraud dextrose broth. After transporting the tubes to the Mycology laboratory the samples were cultured in sabouraud dextrose agar and incubated at 37°C for 48 hours. The cultures were examined daily to observe for Candida colonies. Candida was detected by direct examination of the vesicle and germ tubes that were produced by CA, the carbohydrate absorbing method and specific methods which included the assimilation procedure.

Results

A total of 360 women were recruited. The collection of material for diagnosis was ideally performed during a comprehensive pelvic examination using a speculum for diagnosis of CA by identifying vaginal pH, smear preparing and staining, and culturing. Finally, of all the 360 women that were observed and tested, 120 (33.33%) were involved and 240 (66.67%) were not involved. Of the 120 infected women, 55 patients had Bacterial vaginitis (15.6%) 40 patients had CA

(10.8%) and 25 patients had *Trichomonas vaginalis* (5.9%). Of the 40 Candidal patients, 32 (80%) had Pruritus and 22 (55 %) had an abnormal white discharge, and yeast with or without pseudo-mycelium form was observed in 27 (67.5%) of the microscopic examinations. In 10 (25 %) patients, neither *Candida* nor other organisms were detected in direct examination. Vulvovaginal Candidiasis was not associated with any sociodemographic examined variables such as age and residence.

Discussion

Bacterial vaginosis, Candidiasis and trichomoniasis are responsible for 90% of the cases of vaginal infection [8]. Although Vulvovaginal Candidiasis is the most common fungal disease in the world, little information is known about the distribution and etiology of Candidiasis because microbiology tests are not routinely performed in laboratories [9]. We studied the frequency of Vulvovaginal Candidiasis among women who were referred to our gynecological clinic. In our patients, discharge was the most common symptom. This complaint, as well as itching, burning and erythema were observed in other studies as the primary complaint [9, 10]. Other studies have listed CA as the most common causative agent in Vulvovaginitis, with other species of *Candida* seen less frequently [3, 9, 11, 12]. In other studies, CA was less common [2, 7]. The numbers of *Candida* species in the present study were fewer than the results of Okungbowa et al [13] who isolated seven types of species. This difference may be

due to fewer patients enrolled in the study or the level of social activities, drug abuse and sexual promiscuity. Candidiasis is the most common vaginal infection in most countries ^[10, 14]. Some studies noted that the frequent use of chemotherapeutic agents have led to an increasing incidence of Candida infections as well as other vaginal infections ^[15]. We found that more than 43% of discharge in women with vaginitis was due to a Candida infection. Candida is a common commensally and opportunistic pathogen ^[16], although all symptoms were not only due to Candidiasis ^[12, 14, 17]. Although some research has suggested that vaginal infection was higher in women with a lower socioeconomic status ^[18], in the present study we did not observe this difference. This may unintentionally be due to our patients' similar socio-economical status. In the present study, *C. krusei* was the second most common agent of Vulvovaginal Candidiasis. This finding was in agreement with other studies ^[12]. This may be due to changes in patients' health quality and an increase in resistance to the azoles of antifungal agents ^[19]. The results show that proper diagnosis of symptomatic cases should be done, since discharge and other symptoms of a Vulvovaginal infection might be due to causes other than the Candida species.

Conclusion

We concluded that discharge and Pruritus were the most common symptoms of Vulvovaginal Candidiasis and CA was the most common Candida species which caused Vulvovaginal Candidiasis. Valid clinical methods such as the use of fungal culture in Sabouraud dextrose agar as well as the gold standard for diagnosis especially in cases of recurrent and chronic Candida species are recommended.

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Conflict of interest

The authors have no conflict of interest, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

References

1. Shalini S, NS Murthy, Rajanna MS, et al. Study of reproductive tract infections among women attending urban health centers in Bangalore city, Indian J. Prev. 2011; 42 (3):3-7.
2. Seddigheh Esmaeilzadeh, Saeid Mahdavi Omran, Zahra Rahmani. Frequency and Etiology of Vulvovaginal Candidiasis in Women Referred to a Gynecological Center in Babol, Iran, Royan Institute International Journal of Fertility and Sterility.2009; 3(2): 74-7.

3. Jason D, Mark G. Prevalence of Non-Albicans Candida Infections in Women with Recurrent Vulvovaginal Symptomatology, *Advances in Infectious Diseases*. 2013; 3: 238-42.
4. Raid A, Talat A, Yazeed A, et al. Prevalence and comparison for detection methods of Candida species in vaginal specimens from pregnant and non pregnant Saudi women, *African Journal of Microbiology Research*. 2013; 7(1): 56-65.
5. Nazeri M, Mesdaghinia E, Moraveji R, et al. Fateme Soleymani, Prevalence of Vulvovaginal Candidiasis and Frequency of Candida Species in Women, *J Mazand Univ Med Sci*. 2012; 22(86): 255-62.
6. Akbarzdeh M, Bonyadpour B, Pakshir K, et al. Frequency and Etiology of Vulvovaginal Candidiasis in Women Referred to a Gynecological Center in Babol, Iran, *Arak Medical University Journal*. 2010, 13(2): 12-20.
7. Hafizi Moori M, Dolatian M, Naghash A, et al. The comparison of the effects of micosin vaginal cream (made of garlic) and metronidazole vaginal gel on treatment of bacterial vaginosis, *Arak Medical University Journal*. 2010, 13(3): 35-44.
8. Shazia N, Philip C, Tetsuo K, et al. Antibacterial and antifungal activity of cicerfuran and related 2-arylbenzofurans and stilbenes, *Microbiological Research, Microbiol Res*. 2009; 164(2):191-5.
9. Shazia A, Fauzia A, Shagufta A, et al. Evaluation of common organisms causing vaginal discharge, *J Ayub Med Coll Abbottabad*. 2009; 21(2):45-9.
10. Etminan S, Zarinkatsh H, Lotfee M. The Prevalence of Candida Vaginitis among Women aged 15 - 49 Years in Yazd, Iran, spring & summer. 2008; 2(1):55-61.
11. Tahniat S and Paula K. Vaginitis in adolescents, *Adolesc Med* 15. 2008;2: 235–251
12. Paul N, Christina P, Velma W, et al. Causes of Chronic Vaginitis Analysis of a Prospective Database of Affected Women, *Obstetrics*. 2006; 108 (5):22-9.
13. Jao A, Okonko I, Odu N, et al. Detection and prevalence of Candida isolates among patients in Ibadan, Southwestern Nigeria, and *J. Microbiol. Biotech*. 2011; 1 (3): 176-84.
14. Junko Y, Jay k, Kyle I, et al. The Acute Neutrophil Response Mediated by S100 Alarmins during Vaginal Candida Infections Is Independent of the Th17-Pathway, *PLOS One*. 2012; 7(9):e46311
15. Aditi BS, Nishant R ai, Pramesh C. Candida albicans Vaccines, *Biotechnology International*, 2010;3 (1):4-17.
16. Joziani B, Paulo G, Ruth R, Rose LG. Amaral Jose Eleute rio Jr., Steven S. Witkin, Fernando, Guimaraes. Altered CD16 expression on vaginal neutrophils from Women with vaginitis, *European Journal of Obstetrics & Gynecology and Reproductive Biology*, 2013; 167: 96-9.
17. Cathy J. Watson, Christopher K. Fairley, Danilla Grando, Suzanne M. Garland, Stephen P. Myers. Marie Pirott, Associations with asymptomatic colonization with candida in women Reporting past vaginal candidiasis: an observational study, *European Journal of Obstetrics & Gynecology and Reproductive Biology*. 2013; 169(2), 376-9.
18. John F, Kevin K, Jack S, et al. Newman. Candida Urinary Tract Infection: Pathogenesis. 2011; 52 (6):437
19. Etra S, Zahraa K, Nick B, et al. Thurman and David F. Archer. Vaginal cytokines do not differ between postmenopausal women with and without symptoms of vulvovaginal irritation, *Menopause: The Journal of The North American Menopause Society*. 2012; 21(8):3-7.