



SJMLS-4(1)-013

The Burden of Vulvovaginal Candidiasis Among Women in Developing Countries: A Systematic ReviewSule, H.¹, Kumurya, A. S.¹ and Ahmad I. A.¹Department of Medical Laboratory Science, Faculty of Allied Health Sciences, College of Health Sciences, Bayero University, Kano¹**Author for Correspondence:** sule.hamza@yahoo.com/+234-803-606-2021**Summary**

Inflammation of vulva in the vagina as a result of multiplying opportunistic *candida* species is termed vulvovaginal candidiasis. The disease is not among the sexually transmitted diseases (STDs) because even those that never had sex can come down with it, but that does not mean it cannot be transmitted through sex. The review focused on brief pathogenesis, microbiology, epidemiology, diagnosis and treatment. This review highlights on the possible ways the pathogen reaches the site, mechanism of spread in the area, some of the direct courses, different prevalence rates as it affects some countries, some of the age groups affected, its different presentations in the affected ladies, and how it is treated depending on the magnitude of the situation is briefly discussed based on the available literature obtained through various search engines. The disease is a public health concern and directly or indirectly affects the economy. It affects all categories of women from all countries, married, unmarried, divorced, housewives and working class.

Keywords: *Vulvovaginal candidiasis, Women, Candida species, Vagina*

Introduction

Vulvovaginal candidiasis is a condition of unarguable discomfort to women all over the world. The disease affects the urogenital area of women, most importantly the vulva which gets inflamed, resulting in conditions like painful micturition, serious unease during intercourse and can lead to vaginal wart. Vulvovaginal candidiasis is a disease condition affecting the

urogenital area of women including the vulva and high vaginal swab (HVS) is the specimen of choice for its diagnosis (Nwadioha *et al.*, 2013). Most at times changes in the vaginal environment which in turn cause shift in microbial flora tend to transform asymptomatic colonization by candida species into symptomatic *Candida* infection like vulvovaginal candidiasis (VVC). As in many other vulva diseases, VVC has the potential to cause great psychological distress and has negative impact on affected patient's life.

According to CDC, Candidiasis is an infection caused by a yeast (a type of fungus) called *Candida*. The organism normally lives as normal flora in the body parts as (such as the mouth, the throat, the gut, and the vagina) and on skin without causing any problems. But in some cases, *Candida* can multiply and cause an infection if the vaginal environment changes favorably in a way that encourages its growth. Candidiasis in the vagina is commonly called a "vaginal yeast infection." Other names attributed to this infection are: "vaginal candidiasis," "vulvovaginal candidiasis," or "candida vaginitis. Although in most cases vaginal candidiasis is mild, some women can develop severe infections involving redness, swelling, and cracks in the wall of the vagina (CDC, 2017). Vulvovaginal candidiasis (VVC) is a yeast infection of the lower genital tract of women, in which the vulva, and vagina are affected and are caused by various *Candida* spp (Sobel, 2007; Nyirjesy *et al.*, 2003; Marrazzo, 2002). In some cases, the condition is called candidiasis or moniliasis and it could be recurrent or relapsing

(Ferris *et al.*, 2002; Nyirjesy *et al.*, 2001). As mostly accepted, VVC is not considered a sexually transmitted disease, for the reason being that, it does affect children and celibate women, and at the same time *Candida* spp. are seen as normal vagina flora in healthy women. Although, that does not mean *the disease* cannot be transmitted sexually (CDC, 2017; de Leon *et al.*, 2002; Mårdh *et al.*, 2002).

A cohort study involving apparently healthy women revealed 30% yeast isolation rate which suggested the diagnosis of vulvovaginal candidiasis (VVC) (McCormack *et al.*, 1994). Various cases of VVC has been reported by other researchers with different isolation rates and varied prevalence presentations 25% (Kwawukume *et al.*, 2002) 24% and 18.5% (Otero *et al.*, 1998). Some researchers also found a higher prevalence of symptomatic vaginitis and vaginal colonization which are more significant in pregnant women than found in non-pregnant ones and this is attributable to the high concentrations of reproductive hormones which increase the glycogen content in the vaginal tissue resulting in a good carbon source for candida species (Dennerstein *et al.*, 2001). Use of oral contraceptives with high estrogen content has been shown to increase vaginal colonization with *Candida* species (Tarry *et al.*, 2005). According to Reed *et al.* (2003) increased carriage of yeast is reported in users of intrauterine contraceptive devices, contraceptive sponges, diaphragms, and condoms, with or without spermicides (Reed *et al.*, 2003).

The genus *Candida* housed the causative agents of symptomatic and asymptomatic vulvovaginal candidiasis (VVC) which are the various species in the genus and are found as commensal fungi of the vagina, as well as gastrointestinal and respiratory tracts. A documented report by Sobel (2007) reported *Candida albicans* as the cause of symptomatic VVC in 85% to 95% of cases, whilst *C. glabrata* accounts for most nonalbicans candida vaginitis. It was also observed that 75% of healthy women develops symptomatic VVC at least once in the course of their reproductive age. Additionally, it is estimated also that 5 to 8% of women develops recurrent vulvovaginal candidiasis (RVVC) which is defined as is

defined as the occurrence of four or more episodes of symptomatic VVC per year in a particular patient (Sobel, 2007).

Based on research among HIV infected individuals, it was discovered that, whilst oroesophageal candidiasis as a disease is known to appear at any time during the course of progression of the HIV infection, symptomatic VVC is believed to develop significantly later (Beltrame *et al.*, 2006). It was also documented that oroesophageal candidiasis due to loss of mucosal surface in the affected area and systemic cell-mediated immunity. However, animal studies, in vitro experiments, and clinical trials also revealed that systemic cell-mediated immunity does not play a protective role against candida vaginitis (Beltrame *et al.*, 2006; Fidel Jr, 2005; Sobel, *et al.*, 2001).

According to previous findings on VVC, approximately two-thirds of women experience at least one episode during their life, and about 50% of them usually have multiple episodes (Eschenbach, 2004; Ferrer, 2000). Most previous studies focused on immunocompromised patients, like pregnant women, diabetics, HIV-positive patients, women on oral contraception with high estrogen content, and those on broad-spectrum antibiotic therapy (Odds, 1988).

Under normal condition, vagina is characterized by dynamic interaction between microorganisms, like, *Lactobacillus acidophilus* and other endogenous flora, glycogen, vaginal pH, estrogen, and metabolic by-products of these microbiomes. *L. acidophilus* produces hydrogen peroxide (as a by-product of metabolism), which is toxic to pathogens and keeps the healthy vaginal pH acidic. Vaginitis and other virginal infections like VVC occur when the vaginal microflora is been altered by invading pathogens or biochemical changes in the environment that positively encourage the *Candida* species, enhance their adherence to vaginal epithelial cells, and facilitate germination of daughter yeast cells (Sobel *et al.*, 1998).

It is on record that, vulvovaginal candidiasis is not an uncommon gynaecological finding

among women from both tropical and temperate regions of the world (Pikuza *et al.*, 2008; Lisiak *et al.*, 2000; Matthews *et al.*, 1999). When this condition, occur asymptotically, may persist for longer period of time without obvious consequences. But it normally poses serious health risks to pregnant women and the unborn children. It was reported in Poland that, treatment of recurrent candidiasis in pregnant women resulted in an increased episode of bacterial vaginosis leading to increased risks of preterm labour (Pawlaczyk *et al.*, 2006).

Ever since the discovery of human immunodeficiency virus (HIV) infection in year 1982 to date, the increasing clinical relevance of vaginal candidiasis has proportionately deepened alongside courses of other sexually transmitted disease (STDs) (Coombs *et al.*, 2003; Vernazza *et al.*, 1999). The candida infection was found to contribute significantly to the spread of HIV infections, proper treatment of the disease along with other STDs is therefore strongly recommended for effective control of these diseases (Desruisseau *et al.* 2009; Hajjar *et al.*, 1998).

Nearly 5–10 million females seek gynecologic advice for vaginitis, every year world-wide (Ahmad and Khan, 2009). It has been reported (Ahmad and Khan, 2009), that three fourth (75%) of women usually experience an episode of vulvovaginal candidiasis in their lifetimes, while 50% of these will experience at least a second episode, and 5–10% of all women experience recurrent vulvovaginal candidiasis (that is ≥ 4 episodes of vulvovaginal candidiasis per year).

Although the rate of vulvovaginal candidiasis is frequent, the reasons for its occurrence and recurrence are often unclear. But socio-demographic characteristics, diabetes mellitus, use of antibiotics and oral contraceptives, personal hygiene, sexual activity, and specific immunological defects are strong pointer or potential risk factors (Sobel *et al.*, 1998).

In some developing countries like Ethiopia, vulvovaginal candidiasis still received little consideration because it is regarded as trivial

disease. However, the disease has been identified all over the world as one of the global issues of concern due to its role in directly and/or indirectly affecting the economy (Donder *et al.*, 2002; Foxman *et al.*, 2000). Although *Candida albicans* is believed to be the most common agent of vulvovaginal candidiasis, the rate of the disease due to other *Candida* species, such as *C. krusei*, *C. glabrata*, and *C. tropicalis*, is significantly on the rise, especially in HIV-infected women (Sobel *et al.*, 2001).

Pathogenesis

Candida species may also access the vagina by crossing from the rectum through the perianal region, this is evident because, cultures from both the vagina and gastro-intestinal tract most at times show identical *Candida* species. Normally, a symptomatic disease is associated with an overgrowth of the organisms and their ability to penetrate the superficial epithelial cells. The mechanism by which *Candida* species transform from asymptomatic colonization to an invasive form causing symptomatic vulvovaginal disease is complex and involves host inflammatory responses and yeast virulence factors (Pappas, 2016).

As revealed in a study conducted by **Yingying and Colleagues, involving** 3141 patients, as high as 3181 number of strains of *Candida* were recovered from specimens collected from 3141 patients with VVC. According to the result, a mixed infection involving *C. albicans*, *C. krusei*, and *C. glabrata* was isolated from 40 patients (1.3%). *Candida albicans* was found to be predominant species with 2705 strains representing (85.0%) of the cases (**Yingying et al., 2014**).

Microbiology of Vulvovaginal Candidiasis

As *opportunistic pathogens*, *Candida* species can be identified in samples from the lower genital tract of 10 -20% of healthy women during their reproductive years. It is worthy of notice that the identification of vulvovaginal *Candida* does not indicate candida disease but, the diagnosis of VVC requires the presence of both *Candida* as well as inflammation of the vulvovaginal region. In most cases *Candida albicans* is directly the cause of up to 80% to 92%

of episodes of VVC, while in some cases *C. glabrata* accounts for the major parts of the remaining 8 to 10% percent. But some reports suggested an increased frequency of other non-*albicans* species. This could also be as a result of

widespread usage of unnecessary over-the-counter drugs, long-term use of suppressive antifungals especially the azole and other groups (Pappas, 2016).

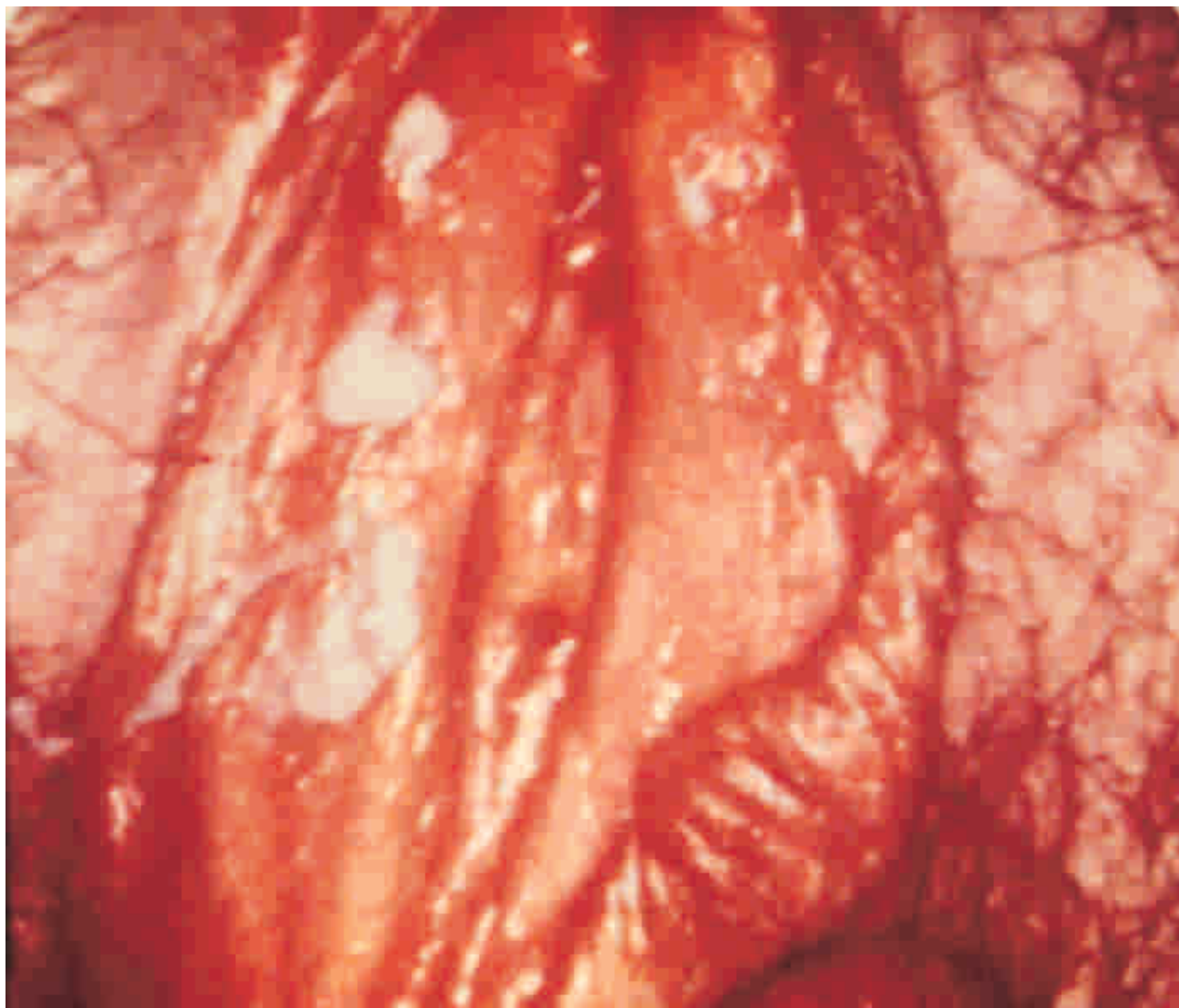


Figure 1: A case of vulvovaginal candidiasis

Epidemiology

According to several records, vaginal candidiasis is a common problem worldwide. In the United States, for example, it is the second most common type of vaginal infection after bacterial vaginal infections (Sobel *et al.*, 2007). For better statistical record therefore, more research is needed to determine the number of women who are affected and how many have vaginal candidiasis that keeps coming back after getting better (more than three times per year). The actual number of cases of vaginal candidiasis is difficult to determine even in the

United States because there is hardly any specific national surveillance for this infection among their women.

In a study involving both HIV and non- HIV patients, 52 cases (53.6%) of symptomatic VVC were diagnosed from 97 HIV-infected women, while 38 cases (37.6%) of symptomatic VVC were diagnosed among the 101 HIV uninfected women (Teke *et al.*, 2014).

Jombo and Colleagues in 2011, reported on a case control study involving 153 pregnant

women and corresponding same number of samples were collected from age matched females who were not pregnant in Gboko town, in Benue state, Nigeria. The rate of urogenital colonization by *Candida species* among pregnant women was 47.7% while 20.3% was the corresponding control group rate (**Jombo, et al., 2011**).

There are reports on the magnitude of the diseases across the globe, including that of 41% prevalence rate among pregnant women attending antenatal care from Maiduguri, the North-Eastern Nigeria (Ibrahim *et al.*, 2013). In another study in Nnewi, Southeastern part Nigeria, Okonkwo, reported no significant difference in prevalence of VVC among women of various socioeconomic status (Okonkwo *et al.*, 2010).

In South Africa, a weighted average prevalence of 26% for symptomatic VVC was observed in a cross-sectional study of women attending antenatal and family planning clinics over the past 20 years (Sebitloane *et al.*, 2011; Coetzee *et al.*, 2010; Ramjee *et al.*, 1998). In the same vein, prevalence of 36.1% and 45.7% for symptomatic VVC among HIV negative and HIV positive sex workers in KwaZulu-Natal respectively, has been established (Ramjee *et al.*, 1998).

A vulvovaginal candidiasis prevalence of 41.4% was obtained in a study on yeast carriage associated with vaginal candidiasis symptoms in Ethiopia. In the study younger women, between 15 and 24 years exhibited lower prevalence (35.1%) of vulvovaginal candidiasis, while in the 25 and above years patients, the prevalence of between 40.0 and 44.8% was observed. It was also observed that, educational and marital status results in varied prevalence of vulvovaginal candidiasis among the study subjects. Illiterate women were found more affected than those who attained at least primary school education and above. The disease was also found to be higher among divorced women (52.6%) compared to unmarried (41.5%) or married women (37.4%) (Adane and Yeshiwork, 2018).

In Kermanshah, Iran, **Reza Faraji and Coworkers investigated the prevalence of**

vaginal candidiasis among their study subjects, in which 86% of the patients were illiterate or had a high school degree, while 14% were college graduates. Also 90% of all the patients were housewives, and the remaining 10% were working class. The prevalence rate of vaginal candidiasis in the study subjects with regards culture and direct test were 12% and 20% respectively. various candida species were identified during the research, these include: *C. albicans* *C. glabrata*, *Candida tropicalis* and *Candida parapsilosis*. But among all, *C. albicans* was the predominant isolate observed (**Reza Faraji et al., 2012**).

A Cameroonian study, **Michel Kengne** studied on antifungal susceptibility pattern on candida species responsible for vaginal candidiasis in Yaoundé, Cameroon, in the study, 245 women were enrolled. Out of the total 245 vaginal samples collected, 94 (38.4%) strains of yeast were isolates among which 43 (45.7%) were *Candida albicans* and 51 (54.3%) were non-albicans species (**Michel Kengne et al., 2017**)

Another study in Maroua, Cameroon, by Vroumsia Toua, focused on the prevalence of VVC amongst pregnant and non-pregnant women. The result indicated that 70% of the pregnant women presented clinical symptoms of VVC out of which 55.4% of them were microbiologically confirmed positive. These therefore suggest that, the prevalence rate of VVC was higher amongst pregnant women (55.4%) than the nonpregnant ones (35.4%). Amongst the participants, 14.6% (pregnant) and 29.6% (non-pregnant) women were symptomatic but had negative cultures (**Vroumsia et al., 2013**).

Diagnosis

It is insufficient to diagnose VVC based solely on genital examination and history because of relatively low specificity in terms of symptoms and signs of the disease, since other disease causes presentations similar to that of VVC, like leukorrhea and pruritus vulvae (Geiger *et al.*, 1995) as such, cultural isolation and identification of the causative agents is therefore crucial to have a definitive diagnosis of VVC.

Vulvovaginal candidiasis is confirmed by isolation of *Candida* species in culture of samples from patients presented with sign and symptom of vaginal abnormalities. Vulvovaginal candidiasis, trichomoniasis and

bacterial vaginosis are the most common cause of vaginitis of which vulvovaginal candidiasis is the second most common after bacterial vaginosis (Anderson *et al.*, 2004; Nyirjesy and Sobel, 2003).

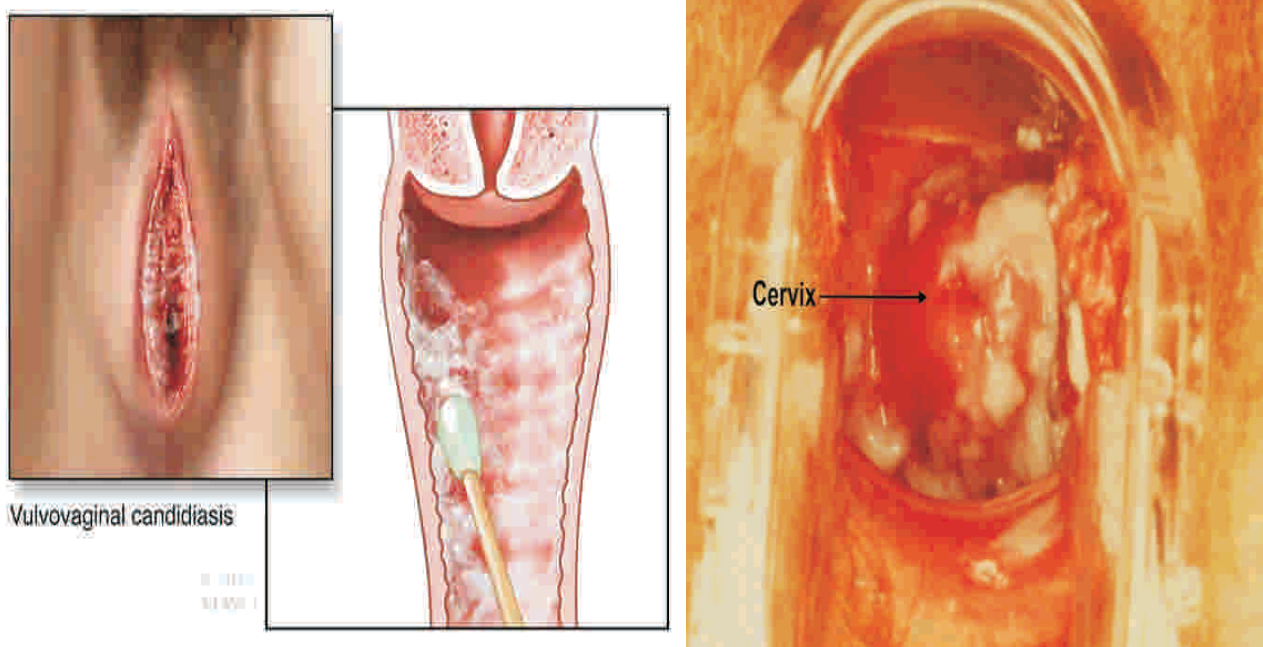


Figure 2: Sample collection in vulvovaginal candidiasis

Treatment

Vaginal candidiasis is usually treated with antifungal agents (Pappas *et al.*, 2016). For most infections, the treatment is an antifungal medication applied inside the vagina or a single dose of fluconazole orally. But in more severe cases of the disease, infections that don't get better, or keep coming back after getting better (recurrent infection), other treatments might be needed. These treatments include more doses of fluconazole take through the mouth or other antifungals applied inside the vagina such as nystatin or flucytosine (Pappas *et al.*, 2016).

Conclusion

Based on the available literature, this disease can be called a women monster as it uses slightest opportunity, because the common agents are part of the normal flora of their victims, as such it could be concluded that Vulvovaginal candidiasis has no border throughout the globe.

References

Adane, B. and Yeshiwork, A. (2018). Vulvovaginal candidiasis: species distribution of *Candida* and their antifungal susceptibility pattern. *Bitew and Abebaw BMC Women's Health*; 18:94.

Ahmad, A, Khan, A. U. (2009). Prevalence of *Candida* species and potential risk factors for vulvovaginal candidiasis in Aligarh, India. *Eur J Obstet Gynecol Reprod Biol*; 144:68–71.

Anderson, M.R, Klink, K., Cohrssen, A. (2004). Evaluation of vaginal complaints. *JAMA*;291: 1368–1379.

Beltrame, A., Matteelli, A., Carvalho, A.C.C. (2006). Vaginal colonization with *Candida* spp. in human immunodeficiency virus—infected women: a cohort study,” *International Journal of STD and AIDS*;17(4):260–266.

Centre for Disease Control (2002) Sexually transmitted diseases treatment guidelines 2002. Centers for Disease Control and

- Prevention. *MMWR Recomm Rep*;51(RR-6):1–78.
- Coetzee, D. and Johnson, L. (2010). Sexually transmitted infections,” in *HIV/AIDS in South Africa, Cape Town, South Africa*, Karim, S.S.A. and Karim, Q.A.A. Eds: 219–220. Cambridge University Press, South Africa, 2010.
- Coombs, R.W., Reichelderfer, P.S., Lunday, A.L. (2003). Recent observations on HIV type-1 infection in the genital tract of men and women. *AIDS*; 17(4): 455–480.
- de Leon, E.M., Jacober, S.J., Sobel, J.D., Foxman B. (2002). Prevalence and risk factors for vaginal *Candida* colonization in women with type 1 and type 2 diabetes. *BMC Infect Dis*. 2:1.
- Dennerstein, G.J., Ellism, D.H. (2001). Oestrogen, glycogen and vaginal candidiasis. *Aust N Z J Obstet Gynaecol*; 41:326–328.
- Desruisseau, A.J., Schmidt- Grimminger, D., Welly, E. (2009). Epidemiology of HPV in HIV-Positive and HIV-Negative fertile women in Cameroon, West Africa. *Infect Dis Obstet Gynaecol*; 810596e.
- Donder, G.G., Vereecken, A., Bosmans, E., Dekeersmaecker, A., Salembier, G., Spitz, B. (2002). Definition of a type of abnormal vaginal flora that is distinct from bacterial vaginosis: aerobic vaginitis. *Inter J Obstet Gynaecol*; 109:34–43.
- Eschenbach, D.A. (2004). Chronic vulvovaginal candidiasis. *N Engl J Med*.;351(9):851–852.
- Ferrer, J. (2000). Vaginal candidiasis: epidemiological and etiological factors. *Int J Gynaecol Obstet*;71 (1): S21–S27.
- Ferris, D.G., Nyirjesy, P., Sobel, J.D., Soper, D., Pavletic, A., Litaker, M.S. (2002). Over-the-counter antifungal drug misuse associated with patient diagnosed vulvovaginal candidiasis. *Obstet Gynecol*; 99(3):419–425.
- Fidel Jr., P.L. (2005). Innate and adaptive cell-mediated immunity against vaginal candidiasis,” *Fungal Immunology*;5: 323–344.
- Foxman, B. Barlow, R. d'Arcy, H. Gillespie, B. and Sobel, J.D. (2000). *Candida* vaginitis: self-reported incidence and associated costs. *Sex Transm Dis*; 27:230–235.
- Geiger, A.M., Foxman, B., Sobel, J.D. (1995). Chronic vulvovaginal candidiasis: characteristics of women with *Candida albicans*, *C. glabrata* and no *Candida*. *Genitourin Med*; 71(5):304–307.
- Hajjar, A.M., Lewis, P.F., Endeshaw, Y., Ndinya-Achola, J., Kreiss, J.K. and Overbaugh, J. (1998). Efficient isolation of human immunodeficiency virus type I RNA from cervical swabs. *J Clin Microbiol*; 36: 2349–2352.
- Ibrahim, S.M., Bukar, M., Mohammed, Y. and Audu, B.M. (2013). Ibrahim HM. Prevalence of vaginal candidiasis among pregnant women with abnormal vaginal discharge in Maiduguri. *Niger J Med*; 22:138–142.
- Jombo, G.T.A., Akpera, M.T., Hembra, S.H. and Eyong, K.I. (2011). Symptomatic vulvovaginal candidiasis: knowledge, perceptions and treatment modalities among pregnant women of an urban settlement in West Africa. *African Journal of Clinical and Experimental Microbiology*; 12(1): 32–37.
- Kwawukume, E.Y., Arhin, R.A. Vulvovaginitis. In: Kwawukume, E.Y., Emuveyan, E.E., editors (2002). *Comprehensive Gynaecology in the Tropics*. 1st ed. Dansoman: *Asante and Hittscher Printing Press Limited*: 72–74.
- Lisiak, M., Klyszejko, C., Pierzecholo, M. I., Sowinska-Przepiera E, Wlosinska, J. (2000). The effect of treatment for vaginal yeast infection on the prevalence of bacterial vaginosis in early pregnancy. *Ginekol Pol*; 71: 964–970.
- Mårdh, P.A., Rodrigues, A.G., Genç, M., Novikova, N., Martinez-de-Oliveira J, Guaschino, S. (2002). Facts and myths on recurrent vulvovaginal candidosis – a review on epidemiology, clinical manifestations, diagnosis, pathogenesis and therapy. *Int J STD AIDS*;13(8):522–539.
- Marrazzo, J. (2002). Vulvovaginal candidiasis. *BMJ*; 325(7364):586–587.
- Matthews, P., Drew, S.V. and Law, L. (1999). Recurrent vaginal thrush and soreness. *Practitioner*; 243(1602): 633–636.
- McCormack, W.M., Jr, Zinner, S.H., McCormack, W.M. (1994). The incidence of genitourinary infections in a cohort of

- healthy women. *Sex Transm Dis*; 21:63–64.
- Michel, K., Siri Vivian, S., Julius, M. N. and Olivia, A. (2017). Antifungals susceptibility pattern of *Candida* spp. isolated from female genital tract at the Yaoundé Bethesda Hospital in Cameroon. *Pan African Medical Journal*; 28:294.
- Nwadioha, S.I., Nwokedi, E.O., Egesie, J., Enejuo, H. (2013). Vaginal candidiasis and its risk factors among women attending a Nigerian teaching hospital. *Niger Postgrad Med J*; 20:20–23.
- Nyirjesy, P. (2001). Chronic vulvovaginal candidiasis. *Am Fam Physician*; 63(4):697–702.
- Nyirjesy, P. and Sobel, J.D. (2003). Vulvovaginal candidiasis. *Obstet Gynecol Clin North Am*. 30(4):671–684.
- Odds, F.C. (1988). *Candida and Candidiasis: A Review and Bibliography*. 2nd ed. London: Bailliere Tindall.
- Okonkwo, N.J. (2010). Prevalence of vaginal candidiasis among pregnant women in Nnewi town of Anambra State, Nigeria. *Afr Res Rev*; 4: 539–548.
- Otero, L., Palacio, V., Carreño, F., Méndez, F. J., Vázquez, F. (1998). Vulvovaginal candidiasis in female sex workers. *Int. J. STD AIDS*; 9:526–530.
- Pappas, P.G., Kauffman, C.A., Andes, D.R. (2016). Clinical Practice Guideline for the Management of Candidiasis: Update by the Infectious Diseases Society of America. *Infectious Diseases Society of America*; 62: e1-50.
- Pawlaczyk, M., Friebe, Z., Pawlaczyk, M. I., Sowinska-Przepiera, E., Wlosinska, J. (2006). The effect of treatment for vaginal yeast infection on the prevalence of bacterial vaginosis in early pregnancy. *Acta Dermatovenerol Croat*; 14(1): 26-29.
- Pikuza, V.V., Chilova, R.A., Ishchenko, A.I. (2008). Comparison of clinical efficacy and safety of domestic generic and original fluconazoles in the treatment of pregnant women with vulvovaginal candidiasis. *Antibiot Khinioter*; 53(7): 35-37.
- Reed, B.D., Zazove, P., Pierson, C.L. (2003). Gorenflo, D.W., Horrocks, J. *Candida* transmission and sexual behaviors as risks for a repeat episode of *Candida* vulvovaginitis. *J. Womens Health (Larchmt)*; 12:979–989.
- Ramjee, G. Abdool Karim, S. S. and Sturm, A. W. (1998). “Sexually transmitted infections among sex workers in KwaZulu-Natal, South Africa,” *Sexually Transmitted Diseases*; 25(7) 346–349
- Reza, F. Mehr, A.R., Fatemeh, R. and Masoud, H. (2012). Prevalence of vaginal candidiasis infection in diabetic women *African Journal of Microbiology Research*; 6(11):2773-2778.
- Sebitloane, H.M. Moodley, J. and Esterhuizen, T.M. (2011). “Pathogenic lower genital tract organisms in HIV-infected and uninfected women, and their association with postpartum infectious morbidity,” *South African Medical Journal*; 101(7):466–469.
- Sobel, J.D. (2007). Vulvovaginal candidiasis. *The Lancet*; 369(9577):1961–1971.
- Sobel, J.D., Faro, S., Force, R.W., Foxman, B., Ledger, W.J., Nyirjesy, P.R. (1998). Vulvovaginal candidiasis: epidemiologic, diagnostic, and therapeutic considerations. *Am J Obstet Gynecol*; 178:203–211.
- Sobel, J.D. Ohmit, S.E. Schuman, P., Klein, R.S., Mayer, K. and Duerr, A. (2001). The evolution of *Candida* species and fluconazole susceptibility among oral and vaginal isolates recovered from human immunodeficiency virus (HIV)-seropositive and at-risk HIV-sero negative women,” *Journal of Infectious Diseases*; 183(2): 286–293.
- Tarry, W., Fisher, M., Shen, S. (2005). Mawhinney M. *Candida albicans*: The estrogen target for vaginal colonization. *J. Surg Res*; 129:278–282.
- Teke, A., William, H.C. Willem, A.S. Benjamin, L.M. and Prashini, M. (2014). Determinants of Symptomatic Vulvovaginal Candidiasis among Human Immunodeficiency Virus Type 1 Infected Women in Rural KwaZulu-Natal, South Africa. Hindawi Publishing Corporation. *Infectious Diseases in Obstetrics and Gynecology*; 2014: 387070.
- Vernazza, P.L., Eron, J.J., Fiscus, S.A., Cohen, M.S. (1999). Sexual transmission of HIV: infectiousness and prevention. *AIDS*; 13:155-166.
- Vroumsia Toua1, Moussa Djaouda, Bouba Gaké, Daniel Ebang Menye, Ebot Akoachere

Christie, Eneke Tambe, Vikumitsi Vughosu Akindoh, Thomas Njiné (2013). Prevalence of Vulvovaginal Candidiasis amongst pregnant women in Maroua (Cameroon) and the sensitivity of *Candida albicans* to extracts of six locally used antifungal plants *International Research Journal of*

Microbiology (IRJM); 4(3):89-97.
Yingying, S., Shangrong, F. Xiaoping, L. and Jianling, L. (2014) Prevalence of *Candida albicans*-closely related yeasts, *Candida africana* and *Candida dubliniensis*, in vulvovaginal candidiasis. *Medical Mycology*; 52: 636–640.

Citation: Sule, H., Kumurya, A. S. and Ahmad I. A. The Burden of Vulvovaginal Candidiasis Among Women in Developing Countries: A Systematic Review. *Sokoto Journal of Medical Laboratory Science*; 4(1): 94 - 102.

Copyright. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.