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Review Article

Vulvovaginal candidiasis: Epidemiology, treatment and prevention strategies

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ABSTRACT

Vulvovaginal candidiasis (VVC) is a commonly encountered clinical condition. At least three-fourth women experience one episode of VVC in their lifetime. In India, the prevalence of VVC is 10 to 35%. Laboratory methods often supplement clinical diagnosis. VVC should be confirmed with culture and other investigative techniques, especially in complicated and recurrent cases. Topical and intravaginal azoles remain the mainstay of therapy. In women who have predisposing risk factors or develop recurrent VVC, oral antifungal agents are used. Topical steroids may be used in women having vulvar symptoms along with pruritus. Pregnancy is a significant risk factor. Intravaginal azoles remain the standard of treatment and can be offered from the second trimester onwards. The emergence of non-albicans species has caused difficulties in the management of VVCs. Thus, all women with vaginal discharge should be correctly diagnosed to tailor the therapy.

1. Introduction

Vulvovaginal candidiasis (VVC) is a *Candida* infection characterized by vaginal discharge, itching, and erythema. Nearly 70–75% of women experience VVC at least once in their lifetime.[1] Over 90% of infections are caused by *Candida albicans*, followed by non-albicans species (e.g., C. glabrata, *C. tropicalis, C. krusei* and *C. parapsilosis*).[2] It is one of the major causes of gynecological consultation worldwide and is associated with considerable direct and indirect economic costs.[3] Risk factors such as previous sexually transmitted infections (STIs), vaginal *douching*, pre-marital sexual intercourse, diabetes mellitus, pregnancy, and STIs in a sexual partner are responsible for VVC.[4] Complicated VVC occurs in nearly 10%-20% of women necessitating appropriate diagnosis.[5] In the majority of cases, clinical diagnosis is apparent. However, microscopic diagnosis with KOH mount or Gram stain, use of immunochromatography test, and culture may be required especially in complicated VVC.[5,6] Treatment with intravaginal or systemic antifungal agents is effective in uncomplicated cases. In a complicated or recurrent VVC, combined intravaginal and systemic therapy may be required.[5] Given the multiple risk factors, changing disease patterns with increasing number of recurrent and complicated infections, it is essential to understand the management strategies in special situations and different treatment strategies. This paper reviews

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the current epidemiology, management strategies, and summarize approach in various comorbid conditions.

2. Approach to Consensus Development

Vulvovaginal infections are commonly encountered in gynecology OPD. VVCs cause significant adverse sequelae. With the increase in complicated VVCs in recent years, it is crucial to improve patient care by accurate diagnosis and effective management. The experts in gynecological and dermatological infections developed this consensus statement to assist the primary care physicians in effectively diagnosing and treating VVCs in both uncomplicated and complicated settings. It will help in tailoring the treatment to patients' needs. Improved diagnosis of VVC with increased use of laboratory measures can help in initiating appropriate therapies. This, in turn, will assist in reducing the number of women progressing to complicated VVC and help fight against the development of drug resistance to azoles.

3. Epidemiology of VVC

Nearly one-third of all cases of vulvovaginitis are Candidial that affects 70% of women at least once in their lifetime. About 8% of women report recurrent VVC (more than three episodes per year).[7] An international study involving 6000 women from the United States and the United Kingdom reported VVC rates between 29% and 49%, with 9% women having recurrent VVC. The cumulative probability of recurrent VVC was age-dependent and reached 25% by 50 years.[8] In a given VVC infection, the lifetime risk of recurrence is reported to be 6 to 20%.[9–11] From India, studies identified a 10% to 35% prevalence of laboratory-confirmed VVC among adult women in the reproductive age group.[12–14] The prevalence may vary according to the diagnostic criteria and the presence of risk factors. Women with diabetes have a higher prevalence of VVC.[15] The risk is higher in type 1 diabetes than type 2 diabetes.[16] In pregnancy, there is increased vaginal colonization of *Candida* from 20% to 30%. Asymptomatic episodes are more common in the second and third trimesters. Immunologic alterations, increased estrogen levels, and increased vaginal glycogen production may predispose to VVC in pregnancy.[17] A study among Lebanese pregnant women reported culture-proven VVC in 44.8% of 221 samples. Isolation of *C. albicans* was reported in 43.4% samples, whereas non-albicans strains counted for 56.6% of VVC (C. glabrata - 44.5%, C. krusei - 12.1%).[18]

4. Investigating VVC

In investigation VVC, the initial evaluation is essential. The initial evaluation should include clinical history, physical examination, microscopy, and test for sexually transmitted diseases. If the diagnosis of VVC is confirmed in the first assessment, treatment should be initiated. In patients where the diagnosis is uncertain or symptoms recur, a detailed evaluation is necessary.

4.1. Clinical history and examination

Symptomatic VVC is associated with overgrowth of the pathogen and their penetration of the superficial epithelial cells. In assessing women for VVC, clinical history related to sexual history, contraceptive use, treatments tried, including over-the-counter drugs, should be noted. In addition, enquire about new allergens, use of topical steroids, coexisting diabetes, recent use of broad-spectrum antibiotics, urinary tract infection, venereal disease, and Covid infection.

Vaginal discharge, pruritus, erythema, fissures, satellite lesions, excoriation, and vulvar edema may also accompany. Vulvar pruritus is one of the dominating symptoms in VVC. Vaginal discharge is the clinical symptom of VVC. But not all discharges are because of VVC. Table 1 enlists the common causes of vaginal discharge in women of reproductive age. Common etiologies include Candidiasis, Trichomoniasis, Bacterial Vaginosis, gonorrhea, chlamydia, and herpes simplex (Table 2).[19] Vaginal discharge can be little or no discharge at times. If the discharge is present, curd-like without or with minimal odor. Discharge can also be loose, watery, homogenous, and indistinguishable from other causes of discharge.

Table 1: Common causes of vaginal discharge

Category	Cause
Non-sexually transmitted infections	Bacterial vaginosis
	Candida
	Chlamydia
Sexually transmitted infections	Gonorrhea
	Trichomonas
	Herpes
Non-infective	Foreign body such as impacted tampons, condoms
	Polyps in cervix
	Malignancy of the genital tract
	Others (e.g. Fistulae, allergic reaction)

Though clinical symptoms and signs may assist in the diagnosis of VVC, not all patients are diagnosed accurately on clinical examination only. A study from Rathod et al. showed a low positive predictive value (<19%) for the diagnosis of VVC using individual signs or symptoms. Compared to the women assessed to be negative clinically for bacterial vaginosis, women diagnosed clinically had a higher prevalence of VVC (6.5% vs. 12%).[3] On examination, signs such as excoriation marks, fissures, and vulvar edema may be seen. However, these may not be specific to VVC. Even nearly 20% of women may have asymptomatic VVC.[5] As none of the clinical signs are pathognomic of VVC, the diagnosis should be confirmed by laboratory methods.

Table 2: Differentiating three common vaginitis by signs and symptoms

Characteristics	Candida spp.	Trichomoniasis	Bacterial vaginosis
Discharge characteristics	Thick and whitish discharge	Varies from scanty to profuse – yellowish discharge	Thin discharge
Odor	Non-offensive	Offensive	Mostly offensive, fishy
Prutitus	Yes	Yes	No
Other symptoms	Soreness, Dyspareunia of vulva, Dysuria	Lower abdominal pain Dysuria	-
Signs	None or there may be Vulval erythema, Oedema, Fissuring and Satellite lesions	Inflammation of vulva, vagina and cervix, "Strawberry cervix" appearance	Discharge coat in the vagina and the vestibule, Inflammation absent
Vaginal pH	4.5 or lower	Above 4.5	Above 4.5

4.2. Laboratory assessments

4.2.1. Microscopy

Candida vaginitis is associated with a normal vaginal pH (<4.5). It is advised that a wet preparation (saline, 10% KOH) or Gram stain of vaginal discharge should be done in all women suspected of VVC. Identification of budding yeasts, hyphae, or pseudohyphae indicates VVC and should be treated. If wet mounts are negative but clinical suspicion is strong, vaginal cultures for *Candida* should be considered.

4.2.2. Culture

If the microscopy shows yeast, culture may not be necessary for all women. *Candida* cultured on Sabouraud agar with drug sensitivities can help in avoiding unindicated or incorrect treatment. Culture should be obtained in women with features of VVC, have normal vaginal pH, and no pathogens seen on microscopy. Also, women with persistent or recurrent symptoms should get cultures as possible of non-albicans species and may have azole resistance. Identifying the species is essential in refractory or recurrent disease.

If *Candida* cultures cannot be performed for any reason, empiric treatment can be considered. In the absence of symptoms or signs, culture identification of *Candida* is not an indication for treatment.[5] A high vaginal swab may not be routinely required, but in cases with recurrent symptoms or where the diagnosis is uncertain, it can be considered.[20] The CDC classifies VVC into uncomplicated and complicated categories based on clinical presentation, microbiology, host factors, and response to therapy (Table 3).[5]

4.3. Other tests

Pap smear is not sensitive as the cervix is relatively unaffected in VVC. Other tests such as DNA probe, polymerase chain reaction, and molecular test such as BD MAX to assay for vaginal microbiome may be used for rapid diagnosis. However, these tests are costly and are limited by their availability. A Nucleic Acid Amplification Test (NAAT) can be used to identify multiple organisms such as *Chlamydia trachomatis, Neisseria gonorrhoea, Mycoplasma genitalium, Mycoplasma hominis, Ureaplasma urealyticum, Ureaplasma parvum, Trichomonas vaginalis, Herpes simplex virus 1, Herpes simplex virus 2, Haemophilus ducreyi, Cytomegalovirus, Lymphogranuloma venerum, Treponema pallidum*, and *Varicella-Zoster virus*.

5. Treatment of VVC

Availability of OTC treatments can help women self-treat the VVC. However, it is not advisable as clinical diagnosis may not be accurate in the majority of the cases. Treatment is dependent on whether VVC is complicated or not. Thus, treatment may be categorized under uncomplicated and complicated VVC.

5.1. Acute uncomplicated VVC

It is characterized by sporadic infrequent episodes, mild/moderate symptoms, probable infection with C. albicans, in healthy, non-pregnant, and immunocompetent women.

Short-course topical formulations (i.e., single dose and regimens of 1–3 days) effectively treat uncomplicated VVC. Topical azoles are more effective than nystatin. Treatment with azoles antifungal provides effective symptom relief, and negative cultures are seen in 80%–90% of patients completing the therapy.[5]Table 4 shows the treatments approved for VVC in India.[21] Topical azoles have similar efficacy as intravaginal azole treatments. Intravaginal fenticonazole can be used. It has excellent retention or "intrareservoir" effect in the local tissues for up to 72 h following topical application.[22] Topical antifungal creams may be used in addition if the patient presents with vulval signs. However, these may cause local irritation sometimes and may also be damaging to latex diaphragms or condoms. Therefore, patients need to be warned about the risk of topical azole creams.[20]

Table 3: Differentiating three common vaginitis by signs and symptoms

Uncomplicated VVC	**Complicated VVC**
Sporadic or infrequent VVC	Recurrent VVC
And	Or
Mild-to-moderate VVC	Severe VVC
And	Or
Likely to be Candida albicans	Nonalbicans candidiasis
And	Or
Non-immunocompromised women	Women with diabetes, immunocompromising conditions (e.g., HIV infection), debilitation, or immunosuppressive therapy (e.g., corticosteroids)

Table 4: Treatment regimens for acute uncomplicated VVC

	Intravaginal treatments
1.	Clotrimazole 1% cream 5 g intravaginally daily for 7–14 days
	Or
2.	Clotrimazole 100 mg vaginal suppository for 7 days
	Or
3.	Miconazole 2% cream 5 g intravaginally daily for 7 days
	Or
4.	Miconazole 100 mg vaginal suppository, one suppository daily for 7 days
	Or
5.	Fenticonazole 600 mg intravaginal capsule (day be repeated at day 3)
	Oral treatment
1.	Fluconazole 150 mg orally in a single dose
	Or
2.	Itraconazole 200 mg orally twice daily for 1 day
	Combinations
1.	Clotrimazole (10mg) + Methylprednisolone aceponate 1mg/gm of cream
2.	Halobetasol 0.05% + Clotrimazole 1.0% cream

Vulvar involvement, in addition to vaginal symptoms, mandates the use of a cream product to be applied to the inflamed skin. In addition, where the vulvar disease is more severe, many practitioners add a topical steroid for local application to the vulva as a separate single formulation or combined with an antifungal cream or ointment. Intravaginal application of a steroid is not necessary. The severity of vulvovaginitis dictates the duration of therapy and hence drug concentration. Mild clinical manifestations usually respond to a single dose or short course of treatment, i.e., 1–3 days, whereas more severe signs and symptoms need topical therapy for 5–7 days.[23]

Follow-up typically is not required. However, women in whom symptoms persist or recur after treatment should be instructed to return for follow-up visits.

Uncomplicated VVC is not usually acquired through sexual intercourse; thus, data do not support the treatment of sex partners. A minority of male sex partners have balanitis. These men benefit from treatment with topical antifungal agents to relieve symptoms.[5]

6. Complicated VVC

Complicated VVCs are characterized by severe signs/symptoms, non-albicans infection, infection in pregnancy, uncontrolled diabetes, immunosuppressed and debilitated women, and recurrent VVCs.

6.1. Recurrent vulvovaginal candidiasis

Recurrent Vulvovaginal Candidiasis (RVVC), usually defined as four or more episodes of symptomatic VVC within one year, affects a small percentage of women (<5%). The pathogenesis of RVVC is poorly understood, and most women with RVVC have no apparent predisposing or underlying conditions. *C. glabrata* and other nonalbicans *Candida* species are observed in 10%–20% of women with RVVC. Conventional antimycotic therapies are not as effective against these nonalbicans species as against *C. albicans*.

6.1.1. Treatment

Each individual episode of RVVC caused by *C. albicans* responds well to short-duration oral or topical azole therapy. However, to maintain clinical and mycologic control, some specialists recommend a longer duration of initial therapy

(e.g., 7–14 days of topical therapy or a 100-mg, 150-mg, or 200-mg oral dose of fluconazole every third day for a total of 3 doses [day 1, 4, and 7]) to attempt mycologic remission before initiating a maintenance antifungal regimen.

Oral fluconazole (i.e., 100-mg, 150-mg, or 200-mg dose) weekly for 6 months is the first line maintenance regimen. If this regimen is not feasible, topical treatments used intermittently can also be considered. Suppressive maintenance therapies are effective in reducing RVVC. However, 30%–50% of women will have recurrent disease after maintenance therapy is discontinued. Symptomatic women who remain culture-positive despite maintenance therapy should be managed in consultation with a specialist.[5]

6.2. Severe VVC

Severe vulvovaginitis (i.e., extensive vulvar erythema, edema, excoriation, and fissure formation) is associated with lower clinical response rates in patients treated with short courses of topical or oral therapy. Either 7–14 days of topical azole or 150 mg of fluconazole in two sequential oral doses (second dose 72 hours after initial dose) is recommended.

6.3. Nonalbicans VVC

Because at least 50% of women with positive cultures for nonalbicans *Candida* might be minimally symptomatic or have no symptoms and because successful treatment is often difficult, clinicians should make every effort to exclude other causes of vaginal symptoms in women with nonalbicans yeast.[23]

6.4. Treatment

The optimal treatment of nonalbicans VVC remains unknown. Options include a longer duration of therapy (7–14 days) with a non-fluconazole azole regimen (oral or topical) as first-line therapy. If recurrence occurs, 600 mg of boric acid in a gelatin capsule is recommended, administered vaginally once daily for 2 weeks. This regimen has clinical and mycologic eradication rates of approximately 70%.[24] If symptoms recur, referral to a specialist is advised.[5]

7. Special Considerations

7.1. Compromised host

Women with underlying immunodeficiency, those with poorly controlled diabetes or other immunocompromising conditions (e.g., HIV), and those receiving immunosuppression therapy (e.g., corticosteroid treatment) do not respond as well to short-term therapies. Efforts to correct modifiable conditions should be made, and more prolonged (i.e., 7–14 days) conventional treatment is necessary.

7.2. Pregnancy

During pregnancy, a high level of estrogen and progesterone are associated with the alteration of the normal vaginal microbiome resulting in overgrowth of *Candida* species leading to active symptomatic infection. In pregnant and non-pregnant women, the pattern of VVI was similar, i.e., BV was the most prevalent infection followed by VVC and MI. However, when pregnant females were compared with non-pregnant females, VVC was more prevalent in pregnant females, while BV and MI were more prevalent in non-pregnant females. During pregnancy, vaginal colonization by *Candida* species is common. Some studies suggest an association between asymptomatic vaginal *Candida* colonization and adverse pregnancy outcomes like preterm birth, but the evidence is inconsistent. The presence of VVC in pregnancy is not associated with preterm delivery and low birth weight.[25,26]

Only topical azole therapies, applied for seven days, are recommended for use among pregnant women. Topical corticosteroids can be used for symptomatic relief.[27] Systemic azoles are contraindicated in pregnancy. Fluconazole applied topically can be considered from the second trimester onwards. In preclinical studies, fenticonazole showed no teratogenic effects. In most cases, fenticonazole short course therapy offers benefits that can be safely given to high-risk patients, including pregnancy, albeit from the second trimester.[28]

7.3. HIV infection

Vaginal *Candida* colonization rates among women with HIV infection are higher than among seronegative women with similar demographic and risk behavior characteristics, and the colonization rates correlate with increasing severity of immunosuppression. Symptomatic VVC is also more frequent in women with HIV infection and similarly correlates with the severity of immunodeficiency. In addition, systemic azole exposure among women with HIV infection is associated with the isolation of nonalbicans *Candida* species from the vagina.

7.3.1. Treatment

Therapy for uncomplicated and complicated VVC in women with HIV infection should not differ from that for seronegative women. Although long-term prophylactic treatment with fluconazole at a dose of 200 mg weekly has effectively reduced *C. albicans* colonization and symptomatic VVC, this regimen is not recommended for women with HIV infection in the absence of complicated VVC. Although VVC is associated with increased HIV seroconversion in HIV-negative women and increased HIV cervicovaginal levels in women with HIV infection, the

effect of treatment for VVC on HIV acquisition and transmission remains unknown.[5]

7.4. Diabetes mellitus

In women with diabetes, VVC is most often the resistant one which is related to some factors such as hyperglycemia, allergy and atopy. One of the most common pathogens associated with this condition is *Candida albicans*.[29] Most vulvovaginal candidiasis can be treated successfully with antifungal drugs. Increased incidence of vaginal candidiasis associated with diabetes raises additional issues regarding prevention and patient management.[30] Drug interactions are an essential consideration for such women. The use of drugs such as fluconazole, itraconazole can elevate levels of sulphonylureas that may lead to hypoglycemia. Increased incidence of non-albicans species in diabetes mellitus demands the use of broad-spectrum antimycotic for treating VVC.

7.5. Skin disorders

Presumptive diagnosis and empirical treatment should be avoided in women with chronic vulvovaginal pruritus. Apart from primary infection, it can occur as a superinfection after using topical steroids. The differential diagnosis for VVC is flexural psoriasis, lichen simplex chronicus, seborrheic dermatitis, irritant contact dermatitis, tinea cruris. Satellite papules, pustules, and erosions are useful to differentiate Candidiasis from other erosive disorders of the vulva like lichen planus, lichen scleroses, and bullous disorders like pemphigus, pemphigoid, and Hailey Hailey disease.

Extensive vulvar oedema due to *C. albicans* should be differentiated from hidradenitis suppurativa, cellulitis due to bacterial infections and other causes of vulvar oedema.

C. glabrata is often confused with vulvodynia or vestibulodynia.

7.6. General advises

Advise females to avoid local irritants and tight-fitting clothes. Promote the use of emollients as soap substitutes. In the presence of risk factors such as HIV, diabetes, offer screening to detect STIs. Intimate vaginal hygiene may help to keep VVCs in check. Personal hygiene is essential to remove the factors that create vulnerabilities for genital infection.

8. Conclusion

Vulvovaginal candidiasis is a commonly observed infection which most often remains asymptomatic. Diagnosis based on clinical picture may often be misleading and therefore need confirmation by microscopy or culture. Cultures should be obtained in recurrent VVC, in the presence of risk factors, or when clinical condition demands. Treatment of uncomplicated VVC is standard, and both intravaginal and oral antifungal azoles are equally effective. Prolonged treatments with constant monitoring may be required in recurrent, severe VVC or those with the presence of risk factors for VVC. In pregnancy, oral azoles should be avoided. Treatment of asymptomatic male partners may not be necessary.

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10. Conflict of Interest

The authors declare no conflict of interest.

References

1. Sustr V, Foessleitner P, Kiss H, Farr A, J Fungi (Basel). Vulvovaginal candidosis: Current concepts, challenges, and perspectives. *J Fungi*. 2020;6(4):267.
2. Felix TC, Röder D, Pedroso RDS. Alternative and complementary therapies for vulvovaginal candidiasis. *Folia Microbiol (Praha)*. 2019;64(2):133–41.
3. Rathod SD, Klausner JD, Krupp K, Reingold AL, Madhivanan P. Epidemiologic features of Vulvovaginal Candidiasis among reproductive-age women in India. *Infect Dis Obstet Gynecol*. 2012;2012:859071. doi:10.1155/2012/859071.
4. Arfiputri DS, Hidayati AN, Handayani S, Ervianti E. Risk factors of vulvovaginal candidiasis in dermato-venereology outpatients clinic of soetomo general hospital. *Afr J Infect Dis*. 2018;12(1S):90–4.
5. CDC. 2015 Sexually Transmitted Diseases Treatment Guidelines. Vulvovaginal Candidiasis. Available from: https://www.cdc.gov/std/tg2015/candidiasis.htm.
6. Marot-Leblond A, Nail-Billaud S, Pilon F, Beucher B, Poulain D, Robert R. Efficient diagnosis of vulvovaginal candidiasis by use of a new rapid immunochromatography test. *J Clin Microbiol*. 2009;47(12):3821–5.
7. Jeanmonod R, Jeanmonod D. Vaginal Candidiasis. [Updated 2022 Jul 18]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459317.
8. Foxman B, Muraglia R, Dietz JP, Sobel JD, Wagner J. Prevalence of recurrent vulvovaginal candidiasis in 5 European countries and the United States: results from an internet panel survey. *J Low Genit Tract Dis*. 2013;17(3):340–5.
9. Amouri I, Sellami H, Borji N, Abbes S, Sellami A, Cheikhrouhou F, et al. Epidemiological survey of vulvovaginal candidosis in Sfax, Tunisia. *Mycoses*. 2011;54(5):499–505.
10. Richter SS, Galask RP, Messer SA, Hollis RJ, Diekema DJ, Pfaller MA. Antifungal susceptibilities of Candida species causing vulvovaginitis and epidemiology of recurrent cases. *J Clin Microbiol*. 2005;43(5):2155–62.
11. Corsello S, Spinillo A, Osnengo 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.
12. Prasad JH, Abraham S, Kurz KM, George V, Lalitha MK, John R, et al. Reproductive tract infections among young married women in Tamil Nadu, India. *Int Fam Plan Persp*. 2005;31(2):73–82.
13. Salvi M. Prevalence of vulvovaginal candidiasis in females in the reproductive age group. *Int J Reprod Contra Obstet Gynecol*. 2019;8(2):647–52.
14. Bang RA, Baitule M, Sarmukaddam S, Bang AT, Choudhary Y, Tale O. High prevalence of gynaecological diseases in rural Indian women. *Lancet*. 1989;333(8629):85–8.

15. Gunther LS, Martins HP, Gimenes F, Abreu AL, Consolaro ME, Svidzinski TI. Prevalence of Candida albicans and non-albicans isolates from vaginal secretions: comparative evaluation of colonization, vaginal candidiasis and recurrent vaginal candidiasis in diabetic and non-diabetic women. *São Paulo Med J*. 2014;132(2):116–20.
16. Leon EMD, 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.
17. Aguin TJ, Sobel JD. Vulvovaginal candidiasis in pregnancy. *Curr Infect Dis Rep*. 2015;17(6):462.
18. Ghaddar N, Roz AE, Ghssein G, Ibrahim JN. Emergence of vulvovaginal candidiasis among Lebanese pregnant women: prevalence, risk factors, and species distribution. *Infect Dis Obstet Gynecol*. 2019;doi:10.1155/2019/5016810.
19. Watson WJ, Demarchi G. Vaginal discharge: an approach to diagnosis and management. *Can Fam Physician*. 1987;33:1847–52.
20. Lazaro N. Sexually Transmitted Infections in Primary Care 2013 (RCGP/BASHH); 2013. Available from: https://elearning.rcgp.org.uk/mod/page/view.php?id=6586Accessed29.
21. Drugs @CDSCO. Available from: https://cdscoonline.gov.in/CDSCO/Drugs.
22. Veraldi S, Milani R. Topical fenticonazole in dermatology and gynaecology. *Drugs*. 2008;68(15):2183–94.
23. Kennedy MA, Sobel JD. Vulvovaginal candidiasis caused by non-albicans Candida species: new insights. *Curr Infect Dis Rep*. 2010;12(6):465–70.
24. Sobel JD, Chaim W, Nagappan V, Leaman D. Treatment of vaginitis caused by Candida glabrata: use of topical boric acid and flucytosine. *Am J Obstet Gynecol*. 2003;189(5):1297–1300.
25. Rasti S, Asadi MA, Taghriri A, Behrashi M, Mousavie G. Vaginal candidiasis complications on pregnant women. *Jundishapur J Microbiol*. 2014;7(2):e10078.
26. Schuster HJ, Jonghe BAD, Limpens J, Budding AE, Painter RC. Asymptomatic vaginal Candida colonization and adverse pregnancy outcomes including preterm birth: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2020;2(3):100163. doi:10.1016/j.ajogmf.2020.100163.
27. Soong D, Einarson A. Vaginal yeast infections during pregnancy. *Can Fam Physician*. 2009;55(3):255–6.
28. Nanavati MS, Bhattacharji P, Krishnaprasad K. Fenticonazole Nitrate-A Symptomatic Approach to Vulvovaginal Infection. *Int J Sci Stud*. 2017;5(5):278–80.
29. Malazy OT, Shariat M, Heshmat R, Majlesi F, Alimohammadian M, Tabari NK. Vulvovaginal candidiasis and its related factors in diabetic women. *Taiwan J Obstet Gynecol*. 2007;46(4):399–404.
30. Brumar A, Rosu AF, Calina D, Bita A, Rosu L, Zlatian O, et al. Study concerning vulvovaginal candidiasis in women with diabetes. *Eur J Hosp Pharm Sci Pract*. 2012;19(2):213.

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