An Investigation of Oral Sex as a Risk Factor for Recurrent Vaginitis: A Case Study

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Case in Point

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Introduction. Inflammation of the vagina, or vaginitis, may present with vaginal pruritus, discharge, or discomfort. The most common cause of vaginitis is due to bacterial vaginosis (BV), followed by vulvovaginal candidiasis (VVC), and trichomonas.[1] Recurrent vaginitis is defined as having three or more confirmed BV incidents or having at least four confirmed VVC incidents in a 12-month period.[1] A 2020 survey revealed 5.2% of women had VVC within the past year from completing the survey , with 4.7% of these meeting criteria for recurrent VVC.[2] Meanwhile, 58% of women with BV had recurrence within a year from their original diagnosis.[1] Patients experiencing recurrent vaginitis are advised to wear cotton underwear, wipe front-to-back after using the restroom, take probiotics after antibiotic use, and avoid hot tubs, douches, scented products, or other irritants in the vaginal area. However, whether certain sexual practices, such as cunnilingus or “oral sex,” can be considered risk factors is less studied and thus remains controversial.[3] In this case study, a 34-year-old immunocompetent woman presents with a 2-year history of recurrent vaginitis, without an obvious causative risk factor other than receptive oral sex.

Case description. A healthy 34-year-old G1P1 woman presents to her primary care physician complaining of recurrent episodes of vaginal itching with discharge after the uncomplicated spontaneous vaginal delivery of her child 2 years prior to presentation. Episodes became more frequent over the past year with approximately one to two episodes per month with vaginitis, thick or malodorous discharge, and occasional suprapubic discomfort. Symptoms are preceded by sexual intercourse with her husband including receptive cunnilingus. She has tried postcoital voiding, adding a daily probiotic, showering before sex, avoiding baths, and limiting sugar and carbohydrates intake, without favorable results. The patient has been in a mutually monogamous relationship with her husband for over 10 years and denies sexually transmitted infections (STIs). The patient admits to a significant decrease in her libido postnatally. Aware of her vaginitis symptoms with sexual intercourse, she endorses even less desire to engage in sexual activity, affecting her relationship. She denies fevers, unintentional weight change, menstrual irregularity, rash, genital lesions, dyspareunia, or urinary complaints. Her past medical, surgical, medication, social, and family histories are otherwise non-contributory.

Her vital signs are within normal ranges, including BMI. A pelvic examination reveals mild suprapubic tenderness and erythema of the vaginal introitus. Speculum examination reveals a thick off-white vaginal discharge with a slight odor within the vaginal vault. This physical

exam finding combined with patient symptoms of itching and discharge lead to the diagnosis of vulvovaginal candidiasis. The bimanual examination yields no abnormalities. Her physical examination is otherwise unremarkable. Urinalysis shows positive leukocytes with a negative culture (Table 1). A urine pregnancy test is negative. A bacterial vaginosis panel with Lactobacillus profiling was tested via vaginal swab (Table 2), which showed the presence of *Gardnerella vaginalis* and *Candida albicans*, confirming an additional diagnosis of bacterial vaginosis. Additional vaginal testing is negative for chlamydia, gonorrhea, and trichomonas.

Symptoms promptly resolved after completing treatment for the concurrent BV and VCC infections with a week of oral metronidazole 500 mg twice daily, and one dose of oral fluconazole 150 mg, respectively. The patient was also prescribed empiric oral fluconazole to utilize at the first sign of vaginitis symptoms. Over the subsequent six-month period, the patient called the office once with persisting symptoms despite post-coital fluconazole. She was prescribed a seven-day course of oral metronidazole 500mg twice a day, which resolved her symptoms.

Table 1. Urinalysis results

Urinalysis Category	Patient Result	Reference Interval
Leukocytes	**Trace**	Negative
Nitrite	Negative	Negative
Urobilinogen	0.2	<2.0 mg/dL
Protein	Negative	Negative
pH	6.0	5.0-9.0
Blood	Negative	Negative
Specific gravity	1.030	1.003-1.030
Ketone	Negative	Negative
Bilirubin	Negative	Negative

Glucose	Negative	Negative
Appearance	Clear	-

Table 2. Urine and Vaginal Culture Results

Laboratory Test	**Patient Result**	**Reference Interval**
Culture, urine	No Growth	No Growth
Atopobium vaginae	Not Detected	Not Detected
Gardnerella vaginalis	**Detected**	Not Detected
BVAB-2	Not Detected	Not Detected
Megasphaera 1	Not Detected	Not Detected
Megasphaera 2	Not Detected	Not Detected
Lactobacillus crispatus	Not Detected	Not Detected
Lactobacillus gasseri	Not Detected	Not Detected
Lactobacillus iners ql	Not Detected	Not Detected
Lactobacillus jensenii ql	Not Detected	Not Detected
Mobiluncus mulieris	Not Detected	Not Detected
Mobiluncus curtisii	Not Detected	Not Detected
Mycoplasma hominis	Not Detected	Not Detected

Ureaplasma urealyticum	Not Detected	Not Detected
Candida albicans	**Detected**	Not Detected
Candida glabrata	Not Detected	Not Detected
Candida krusei	Not Detected	Not Detected
Candida parapsilosis	Not Detected	Not Detected
Candida tropicalis	Not Detected	Not Detected
Trichomonas vaginalis	Not Detected	Not Detected
Chlamydia trachomatis	Not Detected	Not Detected
Neisseria gonorrhoeae	Not Detected	Not Detected

A year later, the patient returned for her annual wellness examination. She had concerns with continuing the sporadic fluconazole to treat vaginitis symptoms “for the rest of my life.” Further, she was frustrated as she had diligently improved all possible contributing lifestyle factors to no avail. Upon further questioning, the patient recalled a recent occasion where she and her husband had vaginal sex in a heated pool. Having forgotten the prophylactic fluconazole, she surprisingly did not develop her typical vaginitis symptoms. It was eventually revealed that, because the patient’s libido had declined after childbirth, her husband had introduced oral sex into their routine sexual practice. The hot tub encounter was the first time in over two years when oral sex did not precede vaginal sex. To test the suspicion that oral sex was contributing to the patient’s recurring vaginal symptoms, the physician recommended adding a precoital antiseptic mouthwash routine for the husband, which effectively eradicated the patient’s recurrent vaginitis episodes.

Discussion. This case reveals a patient whose recurrent BV and VCC episodes appear closely related to being the recipient of oral sex. This discussion aims to highlight the findings in current literature to evaluate oral sex as a potential risk factor for recurrent vaginal dysbiosis, particularly pertaining to BV. Dysbiosis, which occurs when a balanced microbiome is disrupted, cannot be thoroughly explored without a basic understanding of what comprises and maintains a normal vaginal flora.

The normal vaginal microbiome

The normal vaginal microbiome is predominated by Lactobacillus species which aids in maintaining an acidic vaginal environment, with a pH ranging from 3.8 to 5.0. This pH level is hostile to opportunistic or foreign pathogens. The low pH is mediated through the production of lactic acid, hydrogen peroxide, antibiotic hydroxyl radicals, probiotics, and bacteriocins.[4,5,6] Normal variations in vaginal flora can be categorized into five different community state types (CST).[4] CST I represents the most common microbial environment predominated by *Lactobacillus crispatus (L, crispatus)*. CST II, CST III, and CST V consist of varying concentrations of other *Lactobacillus* species including *L. gasseri*, *L. iners*, and *L. jensenii* respectively. CST IV represents the most variable and highly fluctuant microbiome, consisting of higher amounts of non-*Lactobacillus* commensal organisms including *Gardnerella*, *Prevotella*, and *Atopobium*; all three of which have been associated with BV.[4,7] Thus, CST IV has a higher tendency towards dysbiosis, while CST I is considered to have a more stable microbiome.[7] Utilization of a *L. crispatus* vaginal probiotic led to reduction of BV, further supporting the protective effects of a *Lactobaccilus*-predominating vaginal microbiota.[8] However, regardless of the floral subtype, any disruption in the vaginal microbiome may result in dysbiosis.

Risk factors for vaginal dysbiosis

Risk factors for vaginal dysbiosis include alteration of the vaginal pH, menstrual or hormonal status, host immunity, antibiotic use, douching or other vaginal cleaning practices, smoking, poor diet, and high stress.[3,6,7,9] Certain factors pertaining to sexual health have also been known to affect the vaginal microbiome. However, with this being such a broad topic, it is helpful to consider its different aspects into subsets including: partners, practices, prevention from STIs, past history of STI, and pregnancy. In terms of sexual partners, multiple studies have shown a consistently significant relationship between a history of more than three sexual partners within the last year and the diagnosis of BV.[3] Regarding STI prevention, the use of condoms has been shown to decrease the risk of vaginal infections.[3] Additionally, elucidating a past history of STIs is important, as recurrent STIs result in vaginal dysbiosis; in fact, *Trichomonas vaginalis* is a common cause of vaginitis.[1,5] As far as pregnancy prevention, the sustained increase in estrogen from oral contraceptive pills (OCPs) has a protective factor against BV.[7] In contrast, VVC prevalence is increased in patients with higher estrogen states including pregnancy and postmenopausal women using estrogen replacement therapies.[10] In regard to sexual practices such as oral sex, literature indeed suggests an association with VCC, but its correlation with BV yields variable and contradictory results.[11] A 2010 study reports a dose-dependent relationship between receptive oral sex encounters and the diagnosis of BV. However, other studies observed an initial clinical association between oral sex and the diagnosis of BV, but when controlled for other factors such as frequency of penile-vaginal or digital-vaginal sex, the significance was no longer present.[3,12]

Considerations of oral sex as direct and indirect causes of BV

From a microbiological standpoint, the possibility that receptive oral sex could be a viable risk factor for recurrent BV seems reasonable considering its potential for both direct inoculation of bacteria or indirect alterations to the vaginal microbiome. Theoretically, vaginitis could develop from directly inoculating oral microbes into the vagina; specifically *Atopobium*, *Prevotella*, *Fusobacterium*, and *Candida* species which are part of the normal oral flora but may be considered pathogenic when found in high quantities in the vaginal flora.[13] However, further studies are needed to clarify the extent of this direct relationship. In larger concentrations, *Prevotella* and *Atopobium* can indirectly promote the overgrowth of *Gardnerella* by producing ammonia.[5] Similarly, *Fusobacterium* can facilitate the growth of sialidase-producing bacteria like *Garnerella*.[4] Another significant consideration involves the indirect contributions of the saliva itself when introduced into the vaginal environment.

Effects of saliva on vaginal pH and dysbiosis

Saliva is primarily composed of water, food particulates, degradative enzymes, and metabolites, with an average pH of 6.64.[14] The diagnosis of BV through Amsel's criteria—a set of four parameters used to diagnose BV in patients—is bolstered on the finding of an alkaline vaginal environment with a pH greater than 4.5.[1] The introduction of comparatively alkaline saliva may shift the microbiome into that of the BV diagnostic range.[6] Additionally, oral microbiota degrade glycans and proteins from food particles into their basic components for their own use; therefore, saliva contains simple sugars and amino acids that can facilitate the growth of both commensal and pathogenic vaginal bacteria.[15] Saliva also contains many antimicrobial factors for oral protection and, when present in the vaginal environment, can affect microbial growth.[15] With the vaginal microbiome being highly sensitive to changes, each of these introductions has the potential to lead to BV. While studies on dysbiosis in the vaginal microbiome after receptive oral sex are severely lacking, a 2019 study found that male mouths show signs of dysbiosis after oral sex.[16] This finding supports the idea that all of the above interactions can affect a microbiome, but studies are needed to show a clear demonstration of this within the vaginal microbiome. Additionally, another factor needing further exploration includes the influence of oral receptive positions on the volume of salivary inoculum introduced into the vaginal vault.

Effects of mouthwash on saliva and dysbiosis

In the patient case, the utilization of mouthwash prior to oral sex apparently resolved her recurrent symptoms. Mouthwash has several effects on the saliva that have the potential to mediate BV occurrence: decrease saliva to a more amenable pH, decrease bacterial load, and eliminate food particles.[17,18,19] In a study assessing the effect of rinsing with alcohol-based mouthwash, a transient decrease in oral pH from 6.7 to 5.5 was seen, with the effect lasting 16 minutes before pH returned to baseline.[17] While there is no literature clearly defining the effect of mouthwash on vaginal pH, by lowering the oral pH closer to that of the vagina, less alteration of the vaginal pH is expected. In addition, an Australian study showed a significantly decreased presence of gonorrhea in the oropharynx following one minute of

gargling with 21.6% alcohol-based mouthwash, indicating it as a preventative measure for gonorrhea transmission.[18] These studies provide encouraging evidence to support the potential benefit of recommending precoital mouthwash as means to prevent vaginal dysbiosis, however, more conclusive findings are needed.

Conclusion. Despite the controversy surrounding oral sex as a BV risk factor, it is difficult to deny current convincing research supporting the plausibility of its mechanisms through direct inoculation of bacteria or indirectly contributing to vaginal dysbiosis. Focused research exploring this issue is likely lacking given the sensitive nature of discussing detailed sexual practices with patients, which is an important aspect of a patient's medical history. Perhaps it is not unreasonable to preemptively include supine receptive oral sex as a potential risk factor for BV when counseling patients in general on preventing vaginal dysbiosis. However, formal focused research effectively exploring the risks and benefits of utilizing mouthwash prior to oral sex must be conducted before clinicians can recommend this practice as a viable preventative measure for recurrent vaginitis. Further, future directions may include investigating the utilization of CST identification in clinical practice, namely exploring the possibility of functionally altering the microbiome from CST IV to I through the use of probiotics, diet, or vaginal flora transplantation.

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