DOI: https://doi.org/10.2298/SARH211116018A
UDC: 618.15-022.7

ORIGINAL ARTICLE / ОРИГИНАЛНИ РАД

Association of bacterial vaginosis with the most common sexually transmitted infections

Sonja Atanasievska-Kujović[1], Elizabeta Ristanović[1], Vesna Protić-Đokić[1], Dane Nenadić[2]

[1]Military Medical Academy, Institute of Microbiology, Belgrade, Serbia;
[2]Military Medical Academy, Centre for Gynecology and Human Reproduction, Belgrade, Serbia

SUMMARY

Introduction/Objective Bacterial vaginosis (BV) is the most common vaginal dysbiosis that increases the possibility of getting sexually transmitted infections (STI). The objectives of this research are to examine association between BV and the nine most common causes of STIs (*Chlamydia trachomatis*, *Mycoplasma genitalium*, *Mycoplasma hominis*, *Ureaplasma* spp., *Trichomonas vaginalis*, *Neisseria gonorrhoeae*, high-risk human papilloma viruses and herpes simplex virus types 1 and 2) and to determine if the presence of BV increases the probability of coinfection with any of the STI microorganisms.

Methods This study involved 235 patients of reproductive age. One sample swab each was collected for vaginal and cervical testing. The vaginal swabs were used for the detection of BV by the reverse transcription polymerase chain reaction (RT-PCR) test. The cervical swabs were used for the detection of the most common STIs, which were tested by four different multiplex RT-PCR tests. Pearson's χ^2 test and Fisher's probability test were used for statistical analysis of the results.

Results Comparison of the total number of STIs and the condition of the vaginal flora has shown that STIs are the most common in patients with BV (80; 89.9%). Women with BV have a higher frequency of infections with *Ureaplasma* spp. and *M. hominis*, 71 (78.9%) and 50 (44.4%), respectively. The presence of detected STI pathogens and relation with the state of vaginal flora indicate that mono infections are present most often in patients with normal flora (51; 42.1%), while coinfections are mostly present in BV patients (50; 55.6%).

Conclusion This study has confirmed the association of *M. hominis* and *Ureaplasma* spp. with BV as well as an association of coinfections with this dysbiosis. Better understanding of the association between various STIs and the status of vaginal flora is necessary to enable better diagnosis, prevention of diseases and women's health protection.

Keywords: bacterial vaginosis; sexually transmitted infections; coinfection; RT-PCR

INTRODUCTION

The vaginal microbiome consists of various microorganisms which coexist in dynamic balance, establishing complex interconnections not only among themselves, but also with a host. In healthy women of reproductive age, the vaginal microbiome predominantly contains bacteria of the genus *Lactobacillus*. These bacteria support vaginal homeostasis and prevent colonization and growth of unwanted microorganisms including Sexually transmitted infections (STI) [1, 2].

The most common imbalance of vaginal flora is bacterial vaginosis (BV). It is a microbial dysbiosis in which normal microflora, consisting of predominantly *Lactobacillus* microflora, is replaced with numerous anaerobic bacteria, herein referred to as bacterial vaginosis-associated bacteria (BVAB). Symptoms of BV are increased gray or white vaginal discharge, itching or local discomfort, although symptoms are absent in 50% of patients [3, 4].

According to the World Health Organization data, 376 million people get infected with STIs globally every year, which indicates the importance of this public health problem [5]. STIs include more than 30 bacterial, viral, and parasitic pathogens which can be transmitted via vaginal, anal, or oral sex. Some of the most common STIs are *Chlamydia trachomatis*, *Mycoplasma genitalium*, *Mycoplasma hominis*, *Ureaplasma* spp., *Trichomonas vaginalis*, *Neisseria gonorrhoeae*, and human papilloma viruses [6]. There is a great number of clinical manifestations caused by STIs in the female upper and lower reproductive tract, but some of the most common ones are as follows: pelvic inflammatory disease, cervicitis, ectopic pregnancy, miscarriage, chronic pelvic inflammatory disease, neonatal infections, genital cancer, etc. [7]. Asymptomatic STIs are quite challenging, as these infections are difficult to identify, while they are easily transmissible in a sexually active population [5].

Various studies have indicated that BV increases the chances of STIs [8, 9]. Reduction of protective *Lactobacillus* types and changes in the vaginal environment such as increased pH, or reduction of lactic acid concentration enables the survival of vaginal pathogens. BVAB produces mucin-degrading enzymes (such as sialidase), which degrade the mucosal membrane of the vaginal epithelium and cervix, considered one of the most important components of the barrier against infection. The degradation of

Received • Примљено: November 16, 2021
Revised • Ревизија: February 3, 2022
Accepted • Прихваћено: February 5, 2022
Online first: February 9, 2022

Correspondence to:
Sonja ATANASIEVSKA-KUJOVIĆ
Military Medical Academy
Crnotravska 17
11000 Belgrade, Serbia
sonja.atanasievska@gmail.com

mucin and glycogen may cause microabrasions and changes in epithelial cells, which can make pathogen attachment to the receptors on epithelial cells easier. In addition, during BV, the immune balance is affected in a way that causes increased levels of proinflammatory cytokines, which make women more susceptible to STIs [6].

The objectives of this research paper are to establish an association between BV and the nine most common causes of STIs (*Chlamydia trachomatis*, *Ureaplasma* spp., *Mycoplasma genitalium*, *M. hominis, Trichomonas vaginalis, Neisseria gonorrhoeae*, high-risk human papilloma virus types (HR-HPV-16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59), herpes simplex virus types 1 and 2 (HSV-1, HSV-2), and to examine if the presence of BV increases the probability of coinfection with any of the STI microorganisms.

METHODS

This cross-sectional study was conducted on women of reproductive age who attended regular gynecological examinations at the Centre for Gynecology and Human Reproduction, Military Medical Academy, Belgrade, Serbia, during the period from November 2018 to December 2019. The patients included asymptomatic cases and those with various vaginal complaints. Exclusion criteria included recent antibiotic use (> 2 weeks) prior to sample collection. All the patients provided full informed consent for participation. The research has been approved by the Ethics Board of the Military Medical Academy.

One sample swab was collected for vaginal and cervical testing each (FLOQSwab, COPAN, Murrieta, CA, USA) for molecular analysis using multiplex RT-PCR. After collection, the swabs were placed into transport medium, vortexed, and stored at -20°C until DNA extraction (DNA-sorb-AM, AmpliSens, Moscow, Russia), which was performed according to the manufacturer's instructions.

The vaginal swabs were examined for the presence and quantification as well as interrelationship between *Lactobacillus* spp., *Gardnerella vaginalis*, *Atopobium vaginae,* and total concentration of bacterial DNA using a quantitative real-time PCR (RTQ-PCR) (AmpliSensFlorocenosis/Bacterial vaginosis-FRT). Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) software was used to calculate three coefficients. The coefficients RC1 = log (Lac DNA) - log (Gv + Av DNA), RC2 = log (Bac DNA) - log (Lac DNA), RC3 = log (Bac DNA - log (Gv + Av DNA) were determined by the mutual relations between those bacteria. Based on coefficients, the patients were grouped in the following categories: normal vaginal flora (RC1 > 1, *Lactobacillus* spp. is the dominant flora); intermediate flora (0.5 ≤ RC1 ≤ 1, the same number of *Lactobacillus* spp. and aerobic bacteria); BV (RC1 < 0.5, dominant *G. vaginalis* and *A. vagine*); vaginal flora of non-specific etiology (RC2 > 1, RC3 > 2, any RC1 value, small concentration of *Lactobacillus* spp., but also *G. vaginalis*, *A. vagine*).

The cervical swabs were used for the detection of STIs and were tested by four different commercial RT-PCR tests. The first one detected *C. trachomatis, Ureaplasma*, *M. genitalium, M. hominis*; the second one detected *T. vaginalis* and *N. gonorrhoeae*; the third test identified HSV-1/HSV-2; and the fourth RT-PCR was used for the detection of HR-HPV (16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59). All PCR reactions were done on a PCR thermocycler (Sa-Cycler 96, Sacace Biotechnologies, Como, USA).

Statistical analysis

To assist statistical analysis, the PCR test results for BV were categorized into three groups: normal, BV, and abnormal non-BV flora. The flora of nonspecific etiology and intermediary flora were considered to be abnormal non-BV flora. A result of 'bacterial load decreased' was considered a normal finding.

Association between dependent and independent variables was tested using Pearson's χ^2 test or Fisher's probability test. Statistical hypotheses were tested at the level of statistical significance (alpha level) of 0.05. All data were processed in the software package IBM SPSS Statistics, Version 22.0 (IBM Corp., Armonk, NY, USA).

RESULTS

In total, 235 patients of reproductive age were recruited for this study with the average age being 29.39 years (± 6.685). The results (RT-PCR) for BV showed that 121 (51.4%) women had normal (healthy) flora, 90 (38.2%) had BV, nine (3.8%) were denoted as intermediate, and 15 (6.4%) had vaginal flora of non-specific etiology.

Using four different RT-PCR tests, the frequency of the most common STIs were examined. The tests detected *C. trachomatis* (16; 6.8%), *Ureaplasma* spp. (143; 60.9%), *M. hominis* (66; 28.1%), *M. genitalium* (2; 0.9%), HR-HPV 12 types (70; 29.8%), *T. vaginalis* (3; 1.3%), HSV-1 (2; 0.9%), HSV-2 (2; 0.9%), while *N. gonorrhoeae* was not detected in any sample.

Out of 235 patients, 179 (76%) had one of the STI microorganisms detected, while 56 (23.8%) were negative for all microorganisms. Simultaneous presence of more than one microorganism was detected in 93 patients (39.6%). The distribution of mono- and coinfections is presented in Figure 1.

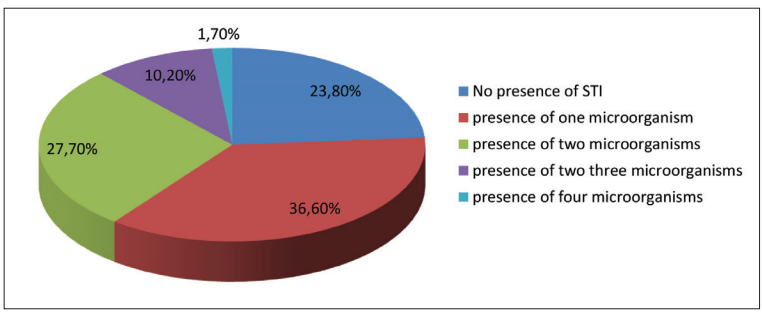

Figure 1. Distribution of mono- and coinfection

Table 1. Association between bacterial vaginosis (BV) and the presence of total sexually transmitted infections (STI)

Total number of STIs	Presence	Normal flora	BV	Intermediate	Flora of unspecified etiology	Total
	no presence	37 (30.6%)	10 (11.1%)	2 (22.2%)	7 (46.7%)	56 (23.8%)
	presence	84 (69.4%)	80 (88.9%)	7 (77%)	8 (53.3%)	179 (76.2%)
	Total	121 (51.4%)	90(38.2%)	9 (3.8%)	15 (6.4%)	235 (100%)

Table 2. Distribution of sexually transmitted infection pathogens in correlation with the state of vaginal flora

Pathogens	Normal flora	BV	Abnormal non-BV		p
			Intermediate	Flora of unspecified etiology	
C. trachomatis	5 (4.1%)	10 (11.1%)	0	1 (6.7%)	0.2
Ureaplasma spp.	63 (52.1%)	71 (78.9%)	5 (55.6%)	4 (26.7%)	< 0.01
M. genitalium	0	2 (2.2%)	0	0	0.34
M. hominis	20 (16.5)	50 (44.4%)	3 (33.3%)	3 (20.0%)	< 0.01
T. vaginalis	1 (0.8%)	1 (1.11%)	0	1 (6.7%)	0.33
N. gonorrhoeae	0	0	0	0	-
HR-HPV (6 ,18, 31,33, 35, 39, 45, 51, 52, 56, 58, 59)	34 (37%)	25 (50%)	6 (75%)	5 (45%)	0.125
HSV-1	1 (0.8%)	1 (1.1%)	0	0	1
HSV-2	0	1 (1.1%)	1 (11.1%)	0	0.3

HSV – herpes simplex virus; HR-HPV – high-risk human papilloma virus; BV – bacterial vaginosis

Table 3. Association between bacterial vaginosis and presence of mono- and coinfection

Infection	Normal vaginal flora	BV	Abnormal non-BV microbiota	Total
No presence of STI	37 (30.6%)	10 (11.1%)	9 (37.5%)	56 (23.8%)
Mono infection	51 (42.1%)	30 (33.3%)	5 (20.8%)	86 (36.6%)
Coinfection	33 (27.3%)	50 (55.6%)	10 (41.7%)	93 (39.6%)
Total	121 (51.4%)	90 (38.2%)	24 (10.4%)	235 (100%)

BV – bacterial vaginosis; STI – sexually transmitted infection

The results have shown that there is a statistically significant difference ($\chi^2 = 15.380$, $p = 0.001$) between the total number of STIs and the condition of the vaginal flora obtained by RT-PCR BV. STIs are most common in patients with BV (80; 89.9%) followed by women with intermediate flora (7; 77.8%) (Table 1).

Distribution of the presence of various STI pathogens in correlation with the state of vaginal flora is shown in Table 2. Statistical significance of correlation of STI pathogens related to the state of vaginal flora exists only with *Ureaplasma* spp. and *M. hominis,* while no such significance was identified with other pathogens. Although there is no significant difference in distribution of correlation with the state of vaginal flora, it has been confirmed that the largest percentage of STI-positive pathogens is in patients with BV.

The association between BV and the presence of mono- and coinfection is presented in Table 3. There is statistical significance between the presence of monoinfections and coinfections compared to the state of vaginal flora ($\chi^2 = 23.677$, $p < 0.001$). In most cases, women with normal flora had monoinfection (51; 42.1%), while women with BV had coinfection (50; 55.6%).

DISCUSSION

This paper employs molecular methods to investigate the prevalence of BV in women of reproductive age but also the association between vaginal dysbiosis and some of the most frequent STI pathogens. Investigations into the association of BV and STIs traditionally use Nugent score or Amsel criteria, which are the gold standard in BV diagnostic method [10–13]. We have used RT-PCR diagnostic testing for BV in the study because there is research that demonstrates shortcomings in the application of the Amsel criteria and Nugent score methods [14, 15]. AmpliSensFlorocenosis/Bacterial vaginosis-FRT in relation to Nugent score, Amsel criteria, vaginal culture, and BD MAX Vaginal panel shows the highest degree of association related to 16S rRNA genome sequencing with microbiome analysis, presented in research paper by van den Munckhof et al. [16]. The RT-PCR test used is based on detection and quantification of *G. vaginalis* and *A. vaginae*, which are important markers for BV diagnostics. On the grounds of the relationship of these two anaerobes – lactobacilli and the total number of bacteria – the test enables assessment of the status of the vaginal flora. Our results indicate that BV prevalence is 38.2% in our study population.

This research has shown that women with BV have a higher frequency of infections with *Ureaplasma* spp. and *M. hominis.* Since *M. hominis* and *Ureaplasma* spp. can be found in both healthy individuals and women with BV, there is an ongoing debate and disagreement on the detection of these pathogens. Due to this fact, the presence of *M. hominis* and *Ureaplasma* spp. in the urogenital tract is not definitive proof of infection and, as such, can be a significant clinical problem. It is considered that the identification of these two pathogens is not adequate without the assessment of the status of vaginal flora. On account of these pathogens' association with various reproductive problems (chorioamnionitis, endometritis, postpartum fever, low birth weight, and preterm delivery) we consider that their identification is of great importance, particularly if BV is present as well [17, 18].

Some STI pathogens (*C. trachomatis* and HR-HPV) have been detected in women with all three states of vaginal flora. However, we have confirmed that the highest percentage of BV patients is positive with *C. trachomatis* (11.1%). At the same time, the highest percentage of HPV-positive patients are in the intermediate vaginal flora (75%) and BV (50%) groups.

In this study, some of the pathogens, such as *M. genitalium, T. vaginalis,* HSV-1/HSV-2, have been detected in a very small percentage of swabs or have not been identified at all (*N. gonorrhoeae*). Considering these results, the opportunity to compare these STI pathogens with various states of vaginal flora has been limited.

The presence of detected STI pathogens and relation with the state of vaginal flora indicate that monoinfections are most often present in patients with normal flora (42.1%), while coinfections with two, three, or four pathogens are mostly present in BV patients (55.6%). Other research studies have also confirmed an association of coinfection and certain STI pathogens with BV [19, 20, 21]. A large number of studies examine the association between STIs and BV, but they rarely include as many pathogens as the current study.

Huge gaps in our knowledge of STI etiology remains an issue, including coinfection and its links with certain clinical manifestations. Bacterial coinfections impact significantly the process of pathogenesis and appearance of clinical manifestations [22, 23]. It is also indicative that coinfections may be present in asymptomatic patients [24]. Compared to infections, coinfections change the process of inflammation in different ways. They also provide fertile ground for the multiplication of opportunistic mycoplasma and its pathogenic effects. The mediators freed by the process of inflammation can cause stagnation in the development cycle of *C. trachomatis* and lead the process into inactive, persistent form. Urogenital mycoplasma can prolong the inflammation of the urethra even after the elimination of *C. trachomatis* if there is a resistance to antibiotic therapy. A great number of microorganisms reduce bioavailability of applied medication during the infection therapy. For this reason, it is important that further STI research targets coinfections, their pathogenesis, eradication, and efficiency of therapy [22].

In patients with abnormal flora (not related to BV), there was a high percentage of coinfections (41.7%), but also no presence of STI pathogens (37.5%). However, since we detected, while using PCR tests, a small percentage of patients with intermediate vaginal flora and flora of nonspecified etiology, our results cannot adequately confirm the association of abnormal non-BV vaginal flora and STI pathogens.

Although many longitudinal and cross-sectional studies have examined the association between BV and STI, the results are different [8, 21, 25, 26, 27]. The problem arises from the fact that BV is a polymicrobial syndrome whose diagnosis is not precise because many of these studies used Amsel criteria or Nugent score as the gold standard. On the other hand, the identification of STI pathogens depends on laboratory tests, geographic region, and characteristics of the examined populations.

The limitation of this study is that the presence of some of STI pathogens were detected in very small percentages or were not detected at all, which impedes full examination of their association with BV. Besides BV, association of anaerobic vaginitis with various STI pathogens also needs to be researched as there are few studies on this topic in the current literature [17].

CONCLUSION

This study has confirmed the association of *M. hominis* and *Ureaplasma* spp. with BV, as well as an association of coinfections with this dysbiosis. Considering that the total frequency of STI pathogens in the examined swabs is 76%, it is important to pay attention to the prevention and elimination of the spreading of STIs. It is also important to continue with education, screening, and raising awareness of STIs and the health issues they may cause. Better understanding of the association between various STIs and the status of vaginal flora is necessary to enable better diagnosis, prevention of diseases, and women's health protection.

ACKNOWLEDGMENT

This paper is a part of a PhD thesis by Sonja Atanasievska Kujović, titled "The importance of multiplex quantitative real-time PCR in diagnosis of vaginal dysbiosis," Medical Faculty of the Military Medical Academy, University of Defense, Belgrade, Serbia, made available to the public on May 10, 2022.

Conflict of interest: None declared.

REFERENCES

1. Ceccarani C, Foschi C, Parolin C, D'Antuono A, Gaspari V, Consolandi C, et al. Diversity of vaginal microbiome and metabolome during genital infections. Sci Rep. 2019;9(1):14095.
2. Saraf VS, Sheikh SA, Ahmad A, Gillevet PM, Bokhari H, Javed S. Vaginal microbiome: normalcy vs dysbiosis. Arch Microbiol. 2021;203(7):3793–802.
3. Coudray MS, Madhivanan P. Bacterial vaginosis – A brief synopsis of the literature. Eur J Obstet Gynecol Reprod Biol. 2020;245:143–8.
4. Redelinghuys MJ, Geldenhuys J, Jung H, Kock MM. Bacterial Vaginosis: Current Diagnostic Avenues and Future Opportunities. Front Cell Infect Microbiol. 2020;10:354.
5. World Health Organization. Sexually transmitted infections (STIs); 2019. Available at: https://www.who.int/news-room/fact-sheets/detail/sexually-transmitted-infections-(stis)
6. Petrova MI, Lievens E, Malik S, Imholz N, Lebeer S. Lactobacillus species as biomarkers and agents that can promote various aspects of vaginal health. Front Physiol. 2015;6:81.
7. The Centers for Disease Control and Prevention (CDC). Sexually Transmitted Infections Treatment Guidelines, 2021.
8. Sanchez G, Contreras P, Martinez A, Garcia C, Lizano M, de la Cruz E. Molecular epidemiology of bacterial vaginosis and its association with genital micro-organisms in asymptomatic women. J Med Microbiol. 2019;68(9):1373–82.
9. Ravel J, Moreno I, Simón C. Bacterial vaginosis and its association with infertility, endometritis, and pelvic inflammatory disease. Am J Obstet Gynecol. 2021;224(3):251–7.
10. Tamarelle J, Thiébaut ACM, de Barbeyrac B, Bébéar C, Ravel J, Delarocque A. The vaginal microbiota and its association with human papillomavirus, Chlamydia trachomatis, Neisseria

gonorrhoeae and Mycoplasma genitalium infections: a systematic review and meta-analysis. Clin Microbiol Infect. 2019;25(1):35–47.
11. Li W, Liu LL, Luo ZZ, Han CY, Wu QH, Zhang L, et al. Associations of sexually transmitted infections and bacterial vaginosis with abnormal cervical cytology: A cross-sectional survey with 9090 community women in China. PLoS One. 2020;15(3):e0230712.
12. Allsworth JE, Peipert JF. Severity of bacterial vaginosis and the risk of sexually transmitted infection. Am J Obstet Gynecol. 2011;205(2):113.e1-6.
13. Turpin R, Brotman RM, Miller RS, Klebanoff MA, He X, Slopen N. Perceived stress and incident sexually transmitted infections in a prospective cohort. Ann Epidemiol. 2019;32:20–7.
14. Lynch T, Peirano G, Lloyd T, Read R, Carter J, Chu A, et al. Molecular Diagnosis of Vaginitis: Comparing Quantitative PCR and Microbiome Profiling Approaches to Current Microscopy Scoring. J Clin Microbiol. 2019;57(9):e00300–19.
15. Scapaticci M, Da Rin G, Bartolini A. Comparison between a novel molecular tool and conventional methods for diagnostic classification of bacterial vaginosis: is integration of the two approaches necessary for a better evaluation? New Microbiol. 2020;43(3):121–6.
16. van den Munckhof EHA, van Sitter RL, Boers KE, Lamont RF, Te Witt R, le Cessie S, et al. Comparison of Amsel criteria, Nugent score, culture and two CE-IVD marked quantitative real-time PCRs with microbiota analysis for the diagnosis of bacterial vaginosis. Eur J Clin Microbiol Infect Dis. 2019;38(5):959–66.
17. Rumyantseva T, Khayrullina G, Guschin A, Donders G. Prevalence of Ureaplasma spp. and Mycoplasma hominis in healthy women and patients with flora alterations. Diagn Microbiol Infect Dis. 2019;93(3):227–31.
18. Leli C, Mencacci A, Latino MA, Clerici P, Rassu M, Perito S, et al. Prevalence of cervical colonization by Ureaplasma parvum, Ureaplasma urealyticum, Mycoplasma hominis and Mycoplasma genitalium in childbearing age women by a commercially available multiplex real-time PCR: An Italian observational multicentre study. J Microbiol Immunol Infect. 2018;51(2):220–5.
19. Sanchez-Garcia EK, Contreras-Paredes A, Martinez-Abundis E, Garcia-Chan D, Lizano M, de la Cruz-Hernandez E. Molecular epidemiology of bacterial vaginosis and its association with genital micro-organisms in asymptomatic women. J Med Microbiol. 2019;68(9):1373–82.
20. Van Der Pol B, Daniel G, Kodsi S, Paradis S, Cooper CK. Molecular-based Testing for Sexually Transmitted Infections Using Samples Previously Collected for Vaginitis Diagnosis. Clin Infect Dis. 2019;68(3):375–81.
21. Barrientos-Durán A, Fuentes-López A, de Salazar A, Plaza-Díaz J, García F. Reviewing the Composition of Vaginal Microbiota: Inclusion of Nutrition and Probiotic Factors in the Maintenance of Eubiosis. Nutrients. 2020;12(2):419.
22. Vesić S, Vukićević J, Gvozdenović E, Skiljević D, Janosević S, Medenica L. Chlamydia trachomatis and Urogenital mycoplasms in nongonococcal uretritis in men. Med Pregl. 2010;63(1–2):47–50. [Article in Serbian]
23. Denks K, Spaeth EL, Jõers K, Randoja R, Talpsep T, Ustav M, et al. Coinfection of Chlamydia trachomatis, Ureaplasma urealyticum and human papillomavirus among patients attending STD clinics in Estonia. Scand J Infect Dis. 2007;39(8):714–8.
24. Wang L, Yang B, Tso LS, Zhao P, Ke W, Zhang X, et al. Prevalence of co-infections with other sexually transmitted infections in patients newly diagnosed with anogenital warts in Guangzhou, China. Int J STD AIDS. 2020;31(11):1073–81.
25. Shipitsyna E, Khusnutdinova T, Budilovskaya O, Krysanova A, Shalepo K, Savicheva A, et al. Bacterial vaginosis-associated vaginal microbiota is an age-independent risk factor for Chlamydia trachomatis, Mycoplasma genitalium and Trichomonas vaginalis infections in low-risk women, St. Petersburg, Russia. Eur J Clin Microbiol Infect Dis. 2020;39(7):1221–30.
26. Chen X, Lu Y, Chen T, Li R. The Female Vaginal Microbiome in Health and Bacterial Vaginosis. Front Cell Infect Microbiol. 2021;11:631972.
27. Lewis FMT, Bernstein KT, Aral SO. Vaginal Microbiome and Its Relationship to Behavior, Sexual Health, and Sexually Transmitted Diseases. Obstet Gynecol. 2017;129(4):643–54.

Повезаност бактеријске вагинозе и најчешћих узрочника сексуално преносивих инфекција

Соња Атанасиевска-Кујовић[1], Елизабета Ристановић[1], Весна Протић-Ђокић[1], Дане Ненадић[2]

[1]Војномедицинска академија, Институт за микробиологију, Београд, Србија;
[2]Војномедицинска академија, Центар за гинекологију и хуману репродукцију, Београд, Србија

САЖЕТАК

Увод/Циљ Бактеријска вагиноза (БВ) најчешћа је вагинална дисбиоза која повећава могућност настанка сексуално преносивих инфекција (СПИ).

Циљ овог истраживања је утврђивање повезаности БВ и девет најчешћих узрочника СПИ (*Chlamydia trachomatis, Ureaplasma spp., Mycoplasma genitalium, Mycoplasma hominis, Trichomonas vaginalis, Neisserria gonorrhoeae*, високо ризични типови хуманог папилома вируса, вирус херпес симплекса типа 1 и 2) и да ли присуство БВ повећава вероватноћу за постојањем коинфекције неким од узрочника СПИ.

Методе У студију је укључено 235 жена у репродуктивном периоду. Један вагинални и један цервикални брис коришћени су за молекуларну анализу. Вагинални брисеви коришћени су за детекцију БВ и процену вагиналне флоре уз помоћ мултиплексног квантитативног *RT-PCR* теста. Цервикални брис је коришћен за доказивање присуства сексуално преносивих патогена који су испитани са четири различита комерцијална *RT-PCR* теста. За статистичку анализу резултата коришћени су Пирсонов χ^2 и Фишеров тест вероватноће.

Резултати Поређење присуства укупног броја узрочника СПИ у зависности од стања вагиналне флоре показује да су СПИ најчешће код болесника са БВ (80; 89,9%). Жене са БВ имају повећану учесталост инфекција са *Ureaplasma spp*. и *M. hominis*, 71 (78,9%) односно 50 (44,4%). Присуство свих детектованих узрочника СПИ у односу на стање вагиналне флоре показује да су моноинфекције најчешће присутне код болесника са нормалном флором (42,1%), док су коинфекције (55,6%) највише присутне код жена са БВ.

Закључак Истраживање је показало асоцијацију *M. hominis* и *Ureaplasma spp.* са БВ као и повезаност коинфекција са овом дисбиозом. Разумевање повезаности између различитих СПИ и стања вагиналне флоре је неопходно како би се омогућила боља дијагностика и превенција болести, као и заштита здравља жена.

Кључне речи: бактеријска вагиноза; сексуално преносиве инфекције; коинфекције, *RT-PCR*