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BACTERIAL VAGINOSIS AND DESQUAMATIVE INFLAMMATORY VAGINITIS: CHOICE OF AN EFFECTIVE THERAPY METHOD. REVIEW

Altynay A. Smagulova[1],

Zaituna A. Khismetova[1], **https://orcid.org/0000-0001-5937-3045**

Arailym K. Kamaliyeva[1], Nazym S. Sagdatbek[1]

[1] NCJSC "Semey medical university", Semey c., Republic of Kazakhstan.

Abstract

Relevance. Bacterial vaginosis and desquamative inflammatory vaginitis are one of the most common causes of patients' visits to gynecologists, primary care providers and emergency care centers. However, many women leave without a clear diagnosis or experience recurring symptoms despite treatment. The three most common etiologies of vaginitis are trichomonas, bacterial vaginosis and vulvovaginal candidiasis, which account for about 70% of cases. The remaining 30% may be related to other causes of vaginitis, including atrophic vaginitis, desquamative inflammatory vaginitis and erosive vaginal disease.

Aim of this review is to describe the common causes bacterial vaginosis and desquamative inflammatory vaginitis in order to increase the likelihood of accurate diagnosis, as well as effective and efficient therapy.

Search strategy. The study examined full-text publications in English and Russian, which are devoted epidemiology and treatment of bacterial vaginosis and desquamative inflammatory vaginitis. In the process of searching for literature, the following search engines were used: Pubmed, Web of science, Cyberleninka, Google Scholar by keywords. The time period was designated 2012-2022. 282 publications were identified on this topic. Of these, 52 publications corresponded to the purpose of our study. *Inclusion criteria:* Publications of the level of evidence A, B: meta-analyses, systematic reviews, cohort and cross-sectional studies. *Exclusion criteria:* summary reports, newspaper articles and personal messages.

Results and conclusions. As a result of numerous studies, it has been established that the vaginal pathway for the treatment of bacterial vaginosis and desquamative inflammatory vaginitis is not inferior in effectiveness to oral therapy. In addition, the vaginal route of treatment is more preferable due to the lower likelihood of adverse reactions.More research is needed to better characterize the cause and treatment of vaginosis and vaginitis. Some studies have shown an improvement in symptoms with the use of topical clindamycin or steroids; however, the ideal duration of treatment and the superiority of one remedy over the other have not been established.

Keywords: bacterial vaginosis, desquamative inflammatory vaginitis, treatment.

Резюме

БАКТЕРИАЛЬНЫЙ ВАГИНОЗ И ДЕСКВАМАТИВНЫЙ ВОСПАЛИТЕЛЬНЫЙ ВАГИНИТ: ВЫБОР ЭФФЕКТИВНОГО МЕТОДА ТЕРАПИИ. ОБЗОР ЛИТЕРАТУРЫ

Алтынай А. Смагулова[1],

Зайтуна А. Хисметова[1], **https://orcid.org/0000-0001-5937-3045**

Арайлым К. Камалиева[1], Назым С. Сагдатбек[1]

[1] НАО «Медицинский университет Семей», г.Семей, Республика Казахстан.

Резюме

Актуальность. Бактериальный вагиноз и десквамативный воспалительный вагинит являются одной из наиболее распространенных причин визитов пациентов к гинекологам, службам первичной медицинской помощи и неотложной помощи. Однако многие женщины уходят без четкого диагноза или испытывают повторяющиеся симптомы, несмотря на лечение. Тремя наиболее распространенными этиологиями вагинита являются трихомонады, бактериальный вагиноз и вульвовагинальный кандидоз, на долю которых приходится около 70% случаев. Остальные 30% могут быть связаны с другими причинами вагинита, включая атрофический вагинит, десквамативный воспалительный вагинит и эрозивное заболевание влагалища.

Цель этого обзора - описать общие причины бактериального вагиноза и десквамативного воспалительного вагинита, чтобы повысить вероятность точной диагностики, а также эффективной и действенной терапии.

Стратегия поиска. В исследовании изучены полнотекстовые публикации на английском и русском языках, которые посвящены эпидемиологии и лечению бактериального вагиноза и десквамативного воспалительного вагинита. В процессе поиска литературы использованы следующие поисковые системы: Pubmed, Web of science,

Cyberleninka, Google Scholar по ключевым словам. Временной период был обозначен 2012-2022 годами. По данной теме выявлено 282 публикаций. Из них цели нашего исследования соответствовало 52 публикаций. *Критерии включения:* Публикации уровня доказательности А, В: мета-анализы, систематические обзоры, когортные и поперечные исследования. *Критерии исключения:* краткие отчеты, газетные статьи и личные сообщения.

Результаты и выводы. В результате многочисленных исследований установлено, что влагалищный путь лечения бактериального вагиноза и десквамативного воспалительного вагинита не уступает по эффективности пероральной терапии. Кроме того, влагалищный путь лечения является более предпочтительным из-за меньшей вероятности развития побочных реакций. Необходимы дополнительные исследования, чтобы лучше охарактеризовать причину и лечение вагиноза и вагинита. Некоторые исследования показали улучшение симптомов при применении местного клиндамицина или стероидов; однако идеальная продолжительность лечения и превосходство одного средства над другим не были установлены.

Ключевые слова: *бактериальный вагиноз, десквамативный воспалительный вагинит, лечение.*

Түйіндеме

БАКТЕРИАЛДЫ ВАГИНОЗ ЖӘНЕ ДЕСКВАМАТИВТЫ ҚАБЫНУ ВАГИНИТЫ: ТИІМДІ ЕМДЕУ ӘДІСІН ТАҢДАУ. ӘДЕБИЕТТІК ШОЛУ

Алтынай А. Смагулова[1],
Зайтуна А. Хисметова[1], https://orcid.org/0000-0001-5937-3045
Арайлым К. Камалиева[1], Назым С. Сағдатбек[1]

[1] КеАҚ «Семей медицина университеті», Семей қ., Қазақстан Республикасы.

Кіріспе. Бактериалды вагиноз және десквамативті қабыну вагиниті пациенттердің гинекологтарға, алғашқы медициналық көмек провайдерлеріне және жедел жәрдем орталықтарына баруының ең көп таралған себептерінің бірі болып табылады. Алайда, көптеген әйелдер нақты диагнозсыз кетеді немесе емделуге қарамастан қайталанатын белгілерді сезінеді. Вагиниттің ең көп таралған үш этиологиясы-трихомоналар, бактериалды вагиноз және вульвовагинальды кандидоз, бұл жағдайлардың шамамен 70% құрайды. Қалған 30% вагиниттің басқа себептерімен байланысты болуы мүмкін, соның ішінде атрофиялық вагинит, десквамативті қабыну вагиниті және қынаптың эрозиялық ауруы.

Бұл шолудың **мақсаты** дәл диагноз қою, сонымен қатар тиімді және тиімді терапия мүмкіндігін арттыру үшін бактериалды вагиноздың және деквамативті қабыну вагинитінің жалпы себептерін сипаттау болып табылады.

Іздеу стратегиясы. Зерттеу бактериалды вагиноздың және деквамативті қабыну вагинитінің эпидемиологиясы мен емделуіне арналған ағылшын және орыс тілдеріндегі толық мәтінді басылымдарды зерттеді. Әдебиеттерді іздеу барысында келесі іздеу жүйелері қолданылды: Pubmed, Web of science, Cyberleninka, Google Scholar кілт сөздер. Уақыт кезеңі 2012-2022 жылдармен белгіленді. Осы тақырып бойынша 282 жарияланым анықталды. Олардың ішінде біздің зерттеуіміздің мақсаты 52 басылымға сәйкес келді. *Қосу критерийлері:* А, В дәлелділік деңгейінің жарияланымдары: мета-талдаулар, жүйелі шолулар, когорттық және көлденең зерттеулер. *Шығару критерийлері:* қысқаша есептер, газет мақалалары және жеке хабарламалар.

Нәтижелер мен қорытындылар. Көптеген зерттеулердің нәтижесінде бактериалды вагинозды және деквамативті қабыну вагинитін емдеудің вагинальды жолы ауызша терапияның тиімділігінен кем түспейтіні анықталды. Сонымен қатар, жағымсыз реакциялардың пайда болу ықтималдығы аз болғандықтан, вагинальды емдеу әдісі анағұрлым қолайлы. Вагиноз мен вагиниттің себебі мен емін жақсы сипаттау үшін қосымша зерттеулер қажет. Кейбір зерттеулер жергілікті клиндамицин немесе стероидтерді қолдану кезінде симптомдардың жақсарғанын көрсетті; дегенмен, емдеудің мінсіз ұзақтығы және бір құралдың екіншісінен артықшылығы анықталмаған.

Түйінді сөздер: *бактериалды вагиноз, десквамативты қабынғвн вагинит, емдеу.*

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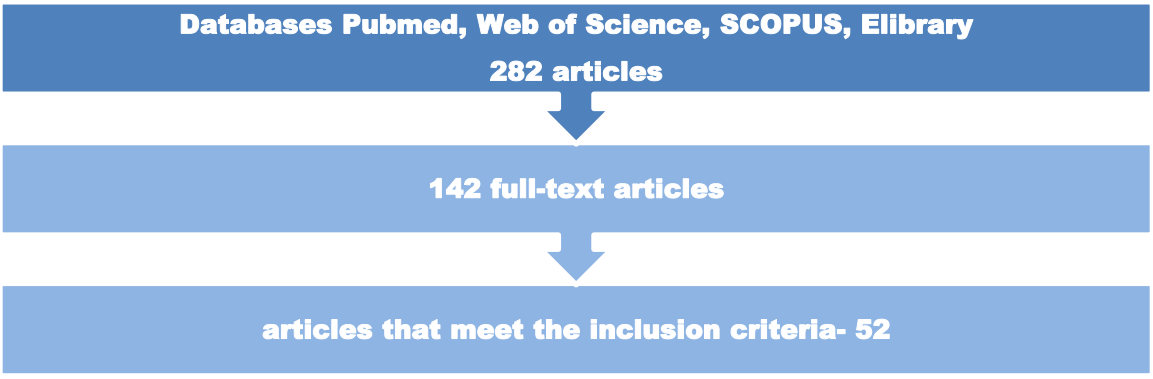
Introduction

The microbiome of a woman's vagina undergoes various changes during the reproductive cycle and throughout a woman's life. The vaginal microbiome of women of reproductive age is mainly affected by the effect of estrogen on vaginal epithelial cells, the predominance of lactobacilli and low pH. The vaginal microbiome is temporarily affected by a number of factors, such as the use of antimicrobial drugs, sexual activity and menstruation, all of which call into question our understanding of the dynamic patterns of vaginal flora [9, 15]. The four CST are dominated by lactobacillus species (producing lactic acid): Lactobacillus crispatus, L. gasseri, L. iners or L. jensenii. One type (CST IV) is characterized by low concentrations or absence of lactobacilli and high concentrations of obligate or facultative anaerobic flora[50]. This CST is associated with both bacterial vaginosis and desquamative inflammatory vaginitis. L. crispatus, L. gasseri and L. jensenii are usually found as the sole or predominant microorganism in the vaginal microbiome, whereas L. infection usually occurs as a component of polymicrobial vaginal flora, often turning into bacterial vaginosis [4]. L. crispatus excludes other organisms due to the low pH due to the strong production of lactic acid along with hydrogen peroxide and specific host antimicrobial proteins called defensins [15, 16].

Lactobacilli producing hydrogen peroxide are associated with a decrease in the level of vaginal proinflammatory cytokines. The low pH associated with lactobacilli may be an evolutionarily chosen trait for protection against sexually transmitted infections and other infections [2], since a medium with a low pH level noticeably suppresses bacterial growth. Lactobacilli producing hydrogen peroxide account for 70 to 90%, and predominate in the normal vaginal flora, [5].

Vaginal discharge can be caused by various causes - physiological, infectious, inflammatory, tumor or iatrogenic [44]. Sexually transmitted infections (STIs) in women with secretions, they have different etiologies and include bacterial (gonococcal and non-gonococcal cervicitis, the latter caused by Chlamydia trachomatis and Mycoplasma genitalium), protozoal (vaginal trichomoniasis caused by Trichomonas vaginalis) and viral infections (progenital herpes caused by herpes simplex) [42, 49]. Some vaginal discharge is characterized by dysbiosis, i.e. an unbalanced state of the endogenous vaginal microflora. This is often, though not always, accompanied by a predominant overgrowth of pathogenic bacteria and/or yeast. While excessive growth of Candida spp. leads to vulvovaginal candidiasis (VVC), vaginal dysbiosis associated with bacterial imbalance, usually referred to as bacterial vaginosis (BV), characterized by excessive growth of anaerobic organisms such as Gardnerella vaginalis, Prevotella spp., Atopobium vaginae and others [29]. BV has been reported to be the most common cause of vaginal dysbiosis and abnormal discharge in women of childbearing age, as well as in peri- and postmenopausal women [42]. As a rule, this is a non-inflammatory condition, so it is called vaginosis instead of vaginitis [25, 42].

Search strategy. The study examined full-text publications in English and Russian, which are devoted epidemiology and treatment of bacterial vaginosis and desquamative inflammatory vaginitis. In the process of searching for literature, the following search engines were used: Pubmed, Web of science, Cyberleninka, Google Scholar by keywords. The time period was designated 2012-2022. 282 publications were identified on this topic. Of these, 52 publications corresponded to the purpose of our study (Table 1).



Results and discussion

Bacterial Vaginosis

It has been proven that the normal ecosystem of the vagina is very important for the treatment and prevention of both various genital infections (including STIs) and urinary tract infections. The normal vaginal microbiota in women of reproductive age is dominated by species of the genus Lactobacillus, especially those that produce hydrogen peroxide, with L. crispatus, L. gasseri and L. jensenii being predominant, reaching values from 107 to 108 CFU/g. vaginal discharge[8]. Vagina in healthy women it is inhabited by 0-4 species of lactobacilli, and the combination of species in women is different. It is believed that these lactobacilli protect the vagina from colonization by pathogens, mainly preventing their attachment to the vaginal epithelium, blocking its receptors, and preventing their reproduction due to the production and excretion of H2O2, lactic acid and bacteriocins[38]. Not all Lactobacillus strains express these properties with the same intensity, but there are huge differences between species and even between strains of the same species. The intensity of these properties has led to the use of some strains as probiotics. Lactobacilli, although predominant, are not the only components of the normal vaginal microbiota, but coexist with several species, most of which are anaerobic (prevailing over aerobic in a ratio of 10 to 1) [45]. The

vaginal microbiota is not a static population, but is in a dynamic state, in which the types and levels of populations constantly fluctuate under changing conditions. These changes are caused by both endogenous factors (such as age, menstrual cycle or pregnancy) and exogenous factors such as sexual intercourse, the use of antibiotics, tampons and contraceptives[30].

The definition of bacterial vaginosis is as follows: a condition with symptoms of abnormal vaginal discharge, unpleasant odor, irritation, itching or burning. Common causes of bacterial vaginosis are vulvovaginal candidiasis and trichomoniasis. Bacterial vaginosis occurs in 40-50% of cases when the cause is identified, while vulvovaginal candidiasis accounts for 20% to 25%, and trichomoniasis - from 15% to 20% of cases. Non-infectious causes, including atrophic, irritating, allergic and inflammatory agents, are less common and account for 5% to 10% of cases of bacterial vaginosis [2, 6]. The diagnosis is made using a combination of symptoms, the results of a physical examination and desk or laboratory testing. Bacterial vaginosis is traditionally diagnosed using Amsel criteria, although Gram staining is the diagnostic standard. New laboratory tests that detect Gardnerella vaginalis DNA or vaginal fluid sialidase activity have similar sensitivity and specificity to Gram staining [24]. Bacterial vaginosis is treated with oral metronidazole, intravaginal metronidazole or intravaginal clindamycin. Vulvovaginal candidiasis is diagnosed using a combination of clinical signs and symptoms with potassium hydroxide microscopy; DNA probe testing is also available. Seeding can be useful for the diagnosis of complicated vulvovaginal candidiasis by identifying nonalbicidal strains of Candida. Treatment of vulvovaginal candidiasis includes oral fluconazole or topical azoles, although only topical azoles are recommended during pregnancy. The Centers for Disease Control and Prevention recommends testing for nucleic acid amplification to diagnose trichomoniasis in women with symptoms or high risk. Trichomoniasis is treated orally with metronidazole or tinidazole, and sexual partners of patients should also be treated. Treatment of non-infectious vaginitis should be directed to the root cause [33]. Atrophic vaginitis is treated with hormonal and non-hormonal methods of treatment. Inflammatory vaginitis can improve with topical application of clindamycin, as well as with the use of steroids [52].

Vaginitis is characterized by vaginal symptoms, including discharge, unpleasant odor, itching, irritation or burning. Most women have at least one episode of vaginitis during their lifetime, making it the most common gynecological diagnosis in primary care. Studies have shown a negative impact on the quality of life of women with vaginitis, with some women expressing anxiety, shame and concern about hygiene, especially in those with recurrent symptoms [5].

The most common causes of vaginitis are bacterial vaginosis, vulvovaginal candidiasis and trichomoniasis. Bacterial vaginosis is the cause in 40-50% of cases when the cause is identified, with vulvovaginal candidiasis ranging from 20% to 25%, and trichomoniasis - from 15% to 20% of cases. Non-infectious causes, including atrophic, irritating, allergic and inflammatory vaginitis, are less common and account for 5% to 10% of cases of vaginitis [52].

Diagnosis.

The use of two standardized, reproducible diagnostic tests based on the use of vaginal smears has become one of the most effective methods for the diagnosis of bacterial vaginosis. The first test is based on laboratory data, gram staining for vaginal flora; the second is a bedside microscopic test with a wet nozzle for the presence of vaginal evidence cells. [34, 35]. Tip cells are epithelial squamous cells covered with coccobacilli in the absence of rods; the absence of rods indicates the absence of lactobacilli. These tests have been introduced into clinical practice and are widely used to determine whether bacterial vaginosis is present. A vaginal pH of less than 4.7 provides an easy-to-read cutoff value to distinguish between normal flora and bacterial vaginosis and is used to exclude bacterial vaginosis[27]. A recent study confirmed the use of a molecular nucleic acid amplification research test, which has been approved by the Food and Drug Administration for the diagnosis of bacterial vaginosis and other vaginitis syndromes [15, 20]. Quantitative polymerase chain reaction assays for the diagnosis of bacterial vaginosis are based on the detection of predominant organisms associated with bacterial vaginosis, such as G. vaginalis, A. Vaginae and mobiluncus species. In a study involving 1,740 patients with symptoms, the performance of a nucleic acid amplification test to detect bacterial vaginosis compared to the reference method (combined results of vaginal Gram staining and wet-mount microscopy) was acceptable (sensitivity 90.5%; specificity, 85.8%). However, the test requires additional verification [15, 47].

Pathogenesis.

Bacterial vaginosis is considered a biofilm infection due to a dense polymicrobial biofilm consisting mainly of G. vaginalis, which adheres to the epithelium of the vagina [19]. The biofilm of A. Vaginae is always present together with the biofilm of G. vaginalis[22, 39], and a higher bacterial load of G. vaginalis and A. Vaginae increases the likelihood of biofilm formation. The vaginal biofilm appears to create a favorable anaerobic environment for other obligate anaerobic bacteria[22]. An important conclusion related to complications of the upper genital tract is that half of women with bacterial vaginosis also have a bacterial vaginosis-related biofilm covering the Endometrium [46]. The fact that this biofilm rises to the endometrium may explain the link between adverse pregnancy outcomes, pelvic inflammatory diseases and bacterial vaginosis. However, the exact role of biofilm in relation to infectious diseases of the upper genital tract remains uncertain [7]. For example, the endometrial cavity in most women is not sterile, and the presence of low levels of bacteria in the uterus is not associated with clinically significant inflammation [28]. A striking 1,000-fold increase in potentially virulent bacteria in women with bacterial vaginosis compared to women with healthy vaginal flora may explain the association of bacterial vaginosis with infection of the upper genital tract [14].

Bacterial Vaginosis and Other Sexually Transmitted Infections.

Bacterial vaginosis not only contributes to the occurrence of sexually transmitted infections, but also transmits other infections, such as infections caused by the human immunodeficiency virus (HIV). In women with

bacterial vaginosis, CD4-T cells are recruited into the mucous membrane of the lower genital tract [1, 18]. Among HIV-infected women, the amount of HIV in the vaginal secretions of women with bacterial vaginosis significantly increases compared to HIV in the vaginal secretions of women without bacterial vaginosis [43]. The vaginal microbiome associated with bacterial vaginosis also inactivates the local microbicide tenofovir, which is used to prevent HIV transmission [23]. Chlamydia trachomatis infection is closely related to bacterial vaginosis [3, 4]. Cervicitis associated with chlamydia increases the amount of discharge from the cervix. This increase, in turn, can change the ecosystem of the vagina, promoting the growth of anaerobic microorganisms. Thus, controlling the incidence of C. trachomatis can prevent bacterial vaginosis, possibly explaining why efforts to combat C. trachomatis have had a disproportionately positive effect on reducing the incidence of inflammatory diseases of the pelvic organs [40].

Treatment.

Table 1 shows the recommendations of the Center for Disease Control and Prevention for the treatment of bacterial vaginosis [51]. The recommendations include various schemes of oral or vaginal administration of metronidazole or clindamycin. Oral metronidazole, topical metronidazole and topical clindamycin show the same efficacy, and oral metronidazole has a large number of side effects.

Table 1.

Treatment Guidelines for Bacterial Vaginosis.*

Treatment	Regimen
Recommended treatments	
Metronidazole	500 mg per os twice a day for 7 days
Metronidazole 0.75% gel	One applicator (5 g) intravaginally once a day for 5 days
Clindamycin 2% cream	One applicator (5 g) intravaginally at bedtime for 7 days
Alternative treatments	
Tinidazole	2 g per os once a day for 2 days
Tinidazole	1 g per os once a day for 5 days
Clindamycin	300 mg per os twice a day for 7 days
Clindamycin ovules	100 mg intravaginally at bedtime for 3 days

* The guidelines are from the Centers for Disease Control and Prevention[51]

A. vaginae, which is often resistant to metronidazole, suggests a high risk of relapse, suggesting that metronidazole is not an ideal empirical remedy. The exact relationship of bacterial biofilm associated with vaginosis with ineffective treatment is unknown. However, it is likely that biofilm infection is difficult to eradicate with antimicrobial therapy. The role of probiotics as additional agents in the treatment of bacterial vaginosis is under study [22]. In one study, oral lactobacilli in combination with metronidazole were more effective than metronidazole alone in the treatment of bacterial vaginosis [35].

Desquamative Inflammatory Vaginitis

Desquamative inflammatory vaginitis can occur in women of different ages, the disease is more common. It is caused by bacteria, yeast, viruses, parachites and other microorganisms. Some sexually transmitted infections (STIs) can also lead to vulvovaginitis. Environmental factors, such as poor personal hygiene and allergens, can also contribute to the occurrence of this disease. Candida Albicans, the causative agent of yeast infection, is the most common cause of vulvovaginitis in women of all ages. The use of antibiotics can lead to yeast infection, because at this time normal fungal bacteria that live inside the vagina are destroyed. Fungal infections usually cause itching in the genitals, the appearance of whitish discharge from the vagina. Another cause of vulvovaginitis is bacterial vaginosis, which is characterized by rapid growth of certain types of bacteria in the vagina. With bacterial vaginosis, grayish, fishy secretions may be released from the vagina. Trichomonas vaginitis, one of the most common sexually transmitted infections (STDs), is also a common cause of the disease. This infection leads to itching of the genitals, the appearance of an unpleasant vaginal smell, the release of yellow-gray or greenish secretions from the vagina. Bathing in the bath, soap, intra-vaginal contraceptives, sprays and perfumes can also cause irritation, itching, rashes in the area of the genitals, clothing that is too dense or whose fabric does not absorb moisture can also contribute to the appearance of rashes. Inflamed tissues are more susceptible to infection than healthy ones, and many infections caused by microorganisms grow in a warm, humid, and dark place. In addition to contributing to the occurrence of vulvovaginitis, these factors also extend the recovery period. Lack of estrogens in the case of menstrual cessation can lead to dryness of the vagina and thinning of the skin of the vagina and vulva, which also leads to itching and burning in the genitals. Some skin diseases can cause chronic irritation and itching in the vulva area. Foreign objects, such as a tampon placed for too long, can also cause irritation and itching of the vulva, as well as an unpleasant smell. Non-specific vulvovaginitis (the exact cause of which is not determined) can occur at all ages, but most often occurs in young girls before puberty. After puberty, the environment inside the vagina becomes acidic, which usually prevents the passage of infection. Non-specific vulvovaginitis can occur in girls who do not observe personal hygiene, it is characterized by a smelly, brownish-green discharge and irritation of the vulva mucosa, vagina. This condition is usually associated with the rapid growth of bacteria present in the feces. These bacteria pass from the rectum to the vagina when they are rubbed back and forth after the stool is removed. If girls have unusual infections and recurrent, unexplained manifestations of vulvovaginitis, there are doubts about sexual assault. Neisseria gonorrhoeae is a microorganism that causes gonorrhea. It is a causative agent of gonococcal vulvovaginitis in girls

who have sex. Gonorrhea, which occurs due to vaginitis, is a disease that occurs through the genital tract. If laboratory tests confirm this diagnosis, it is advisable to conduct an examination of young girls for sexual abuse [11]. However, the term “desquamative inflammatory vaginitis" has priority and was first introduced in 1965 by Gray and Barnes. The term “aerobic vaginitis" was introduced in 2002 in relation to a disease caused by an abnormal vaginal microbiome, genomically defined as CST IV.The published literature on desquamative inflammatory vaginitis is still surprisingly limited, consisting mainly of retrospective case series or brief reviews [35].

Cause.

The main cause that causes desquamative inflammatory vaginitis is unknown, but it is dysbiosis of the normal vaginal microbiome associated with inflammation. In desquamative inflammatory vaginitis, the vagina is colonized by facultative bacteria, and not by obligate anaerobic bacteria that colonize the vagina in bacterial vaginosis. The microflora in desquamative inflammatory vaginitis usually consists of E. coli, Staphylococcus aureus, group B streptococcus or fecal enterococcus [11]. The microbiome of desquamative inflammatory vaginitis has also been sufficiently studied, as has the microbiome of bacterial vaginosis. Desquamative inflammatory vaginitis can also be a systemic inflammatory syndrome that causes inflammation of the vagina, leading to abnormal vaginal flora. As with bacterial vaginosis, understanding the mechanism underlying the loss of vaginal lactobacilli should shed light on the pathogenesis of desquamative inflammatory vaginitis[32].

Symptoms and Signs.

Signs and symptoms of desquamative inflammatory vaginitis include purulent vaginal discharge and a strong inflammatory reaction. Vaginal discharge is uniform and yellowish, without a fishy smell. In severe cases, there is irritation of the vulva and erythema of the vaginal mucosa with exotic lesions or erosions. Symptoms may last for a long time and fluctuate, indicating a chronic or recurrent natural history [21].

Epidemiology.

In several studies that systematically analyzed the prevalence of desquamative inflammatory vaginitis among pregnant or non-pregnant women, the indicators ranged from 2 to 20%. One of the important limitations of epidemiological studies was the lack of standardized biomarkers for the diagnosis of desquamative inflammatory vaginitis [26]. The insufficiency of explicit diagnostic methods is compounded by the fact that the existence of this condition has not been universally recognized by clinicians. In our experience, highly symptomatic desquamative inflammatory vaginitis is relatively rare, while a less symptomatic form of vaginal dysbiosis, characterized by a decrease in the number of lactobacilli, an increase in the number of facultative bacteria and inflammation, is much more common. To what extent this dysbiosis turns into a symptomatic disease remains to be determined [17].

Diagnosis.

Examination of vaginal secretions under a microscope finds an increase in inflammatory cells and parabasal epithelial cells, and the vaginal flora is usually abnormal, with an increased pH.63. Diagnosis at the place of medical care is based on the presence of an increased number of leukocytes and parabasal epithelial cells [13, 31]. Microscopic examination of wet-coated preparations is the preferred method of diagnosis of desquamative inflammatory vaginitis,since Gram staining of the vaginal flora does not distinguish between bacterial vaginosis and desquamative inflammatory vaginitis. The use of conventional vaginal cultures is not recommended [36].

Treatment.

Recommended approaches to the treatment of desquamative inflammatory vaginitis are presented in Table 2 [37].

Table 2.

Treatment Recommendations for Desquamative Inflammatory Vaginitis.*

Treatment	Regimen
Recommended treatments	
Clindamycin 2% cream	Intravaginally daily at bedtime for 1 to 3 wk; consider maintenance therapy once or twice a week for 2–6 mo
Topical glucocorticoid Hydrocortisone, 300–500 mg	Intravaginally daily at bedtime for 3 wk; consider maintenance therapy once or twice a week for 2–6 mo
Clobetasol propionate	Intravaginally daily at bedtime for 1 wk (duration not evidence-based)
Alternative treatments	
Fluconazole	150 mg per os once a week as maintenance therapy
Topical vaginal estrogen	Twice a week

*The recommendations are from Reichman and Sobel [37].

The above treatment options have not been properly tested in randomized clinical trials. Metronidazole is ineffective in desquamative inflammatory vaginitis, and the ineffectiveness of metronidazole treatment in women with bacterial vaginosis may indicate desquamative inflammatory vaginitis. Clindamycin is active against a wide range of facultative bacteria associated with desquamative inflammatory vaginitis, and also has an anti-inflammatory effect[12]. In clinical practice, topical clindamycin, often used as a long-term maintenance therapy, seems to be an effective method of treating severe forms of desquamative inflammatory vaginitis. Maintenance therapy once a week is usually used to reduce the risk of relapses or exacerbations [48]. An observational study has shown that topical application of 2% clindamycin, with or without 10% hydrocortisone, is useful in the treatment of severe desquamative inflammatory vaginitis. Women with desquamative inflammatory vaginitis characterized by a severe parabasal cell component may benefit from intravaginal use of estrogens as maintenance therapy [10, 41].

Conclusion

The ecosystem of the human vagina is very dynamic. The vaginal microbiome can affect host physiology, and

host physiology can affect the vaginal microbiome. Research is needed to better understand the interactions between the vaginal microbiome, host physiology, reproduction, and host defense. Recent genomic studies have expanded our knowledge of the vaginal microbiome. Future research based on genomic, proteomic and metabolomic methods may ultimately have a significant impact on women's reproductive health. Mechanisms that initiate and maintain colonization by vaginal lactobacilli, especially L. crispatus, in women of reproductive age needs clarification. New biomarkers of abnormal vaginal microbiome are needed for clinical practice. Microorganisms of the vaginal microbiome alter the innate immune response and barrier properties of the human vaginal epithelium. A significant increase in vaginal pH and violation of immune barriers increase susceptibility to sexually transmitted infections,and this, in turn, increases the burden of diseases caused by an abnormal vaginal microbiome. Understanding that the mechanisms that initiate and maintain a healthy vaginal microbiome will be important for the development of improved treatments for bacterial vaginosis and desquamative inflammatory vaginitis, as well as effective local microbicides for the prevention of HIV infection and other sexually transmitted infections.

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Contact information:
Smagulova Altynay Armanovna - resident of 2 years of study on the specialty "Obstetrics and gynecology adult, children", NCJSC "Semey Medical University", Semey, Republic of Kazakhstan.
Postal address: Republic of Kazakhstan,070010, Ust-Kamenogorsk city, Sevastopol 20-20
E-mail: t.abilpeis@mail.ru
Telephone: 87477195950