



Aerobic Vaginitis Caused By *Streptococcus B-Hemolyticus* and *Staphylococcus Epidermidis* in a 26-Year-Old Woman with a History of Frequent Antibiotics Administration

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Abstract: Aerobic vaginitis (AV) is a form of vaginitis caused by the presence of aerobic bacteria, primarily *group B Streptococcus*, *Escherichia coli*, *Enterococcus faecalis*, *Streptococcus viridans*, and *Staphylococcus epidermidis*. This condition is characterized by clinical signs of acute inflammation, including vaginal redness, burning, increased vaginal secretions with a pH of 5, sticky vaginal discharge, leukocyte infiltration, and vulvar pruritus, which is defined as itching and irritation, and commonly occurs in individuals between the age of 15–30 years and 31–45 years. The report presents a case of AV caused by *Streptococcus β-hemolyticus* and *Staphylococcus epidermidis* in a 26-year-old female patient at Hasan Sadikin Hospital, Bandung. The treatment process includes the administration of amoxicillin 500 mg three times daily for 14 days. The patient was unmarried, routinely performed vaginal douching with betel soap, and frequently used a panty liner. Subsequently, a history of frequent antibiotic use was reported, however, the specific names of previously administered antibiotics remain unknown, except for clindamycin, which is the only agent specifically recalled. The patient also had a history of high exposure to stressors and underwent a bacterial culture examination taken from the vaginal introitus, which showed the presence of *Streptococcus β-hemolyticus* and *Staphylococcus epidermidis*. This case highlights the significance of AV in the differential diagnosis of vaginal infections, especially in atypical patient populations, and emphasizes the need for appropriate diagnostic measures such as bacterial culture to guide effective treatment strategies.

Keywords: aerobic vaginitis, *streptococcus β-hemolyticus*, *staphylococcus epidermidis*, aerobic vaginitis

Introduction

Aerobic vaginitis (AV) is a type of vaginitis related to aerobic bacteria, primarily including *group B Streptococcus*, *Escherichia coli*, *Enterococcus faecalis*, *Streptococcus viridans*, and *Staphylococcus epidermidis*. This condition is characterized by clinical symptoms of acute inflammation, such as vaginal redness, burning sensations, increased vaginal secretions with a pH of 5.5–6.5,¹ sticky discharge, leukocyte infiltration, and vulvar itching as well as irritation. Subsequently, it commonly occurs in individuals between the age of 15–45 years.^{2–5} Vaginitis is a term used to describe a range of diseases presenting symptoms affecting the vulva and vagina. It is a leading cause of healthcare visits among women, accounting for an estimated 6 to 10 million consultations annually in the United States.⁶ This condition is not life-threatening, but it can reduce quality of life due to discomfort, pain, decreased sexual function, and self-confidence.^{7,8} AV is an important clinical problem, often misdiagnosed as bacterial vaginosis (BV), leading to inappropriate treatment with metronidazole, which is typically ineffective and may result in chronic infection. The condition is associated with various gynecological and obstetric complications, including pelvic inflammatory disease and an increased susceptibility to sexually transmitted infections. Moreover, AV is a well-documented cause of adverse pregnancy outcomes, such as preterm birth.⁹

The three primary infectious causes of vaginitis are BV, vulvovaginal candidiasis, and trichomoniasis.^{7,10} However, approximately 7–72% of women with vaginitis caused by abnormal vaginal flora other than BV are undiagnosed, and this phenomenon is often referred to as transitional flora.⁶ In 1999, Donders¹¹ introduced the term “aerobic vaginitis (AV)” to describe this newly identified condition. It has been reported that AV occurs in 5% of all cases in specialized vaginal clinics.¹⁰ The incidence of this condition increases with age, onset of sexual activity, and parity.¹² According to the study by Khan et al the disease commonly affects individuals within the age range of 15–30 years and 31–45 years. The cause of AV is aerobic bacteria, primarily Gram-positive bacteria (71%) including group *β-hemolytic streptococci*, *staphylococci*, and *enterococci*, as well as Gram-negative bacteria (29%), such as *Enterobacteriaceae*, particularly *Escherichia coli*.^{2,3} The following factors have been identified as independent risk factors for AV, such as the use of intrauterine devices, the presence of external hemorrhoids, prolonged antibiotic use, and frequent vaginal douching. Studies have also shown that stress induces vaginal dysbiosis, resulting in a reduction in the dominance of *Lactobacillus* species.^{13,14} A case of AV caused by *Streptococcus β-hemolyticus* and *Staphylococcus epidermidis* in a 26-year-old female patient who had never had sexual intercourse was reported.

Case Report

In May 2024, a 26-year-old unmarried woman visited a venereology clinic in a top referral hospital, Dr. Hasan Sadikin Hospital, Bandung, West Java, Indonesia, due to vaginal discharge persisting for one year. The discharge had a sour odor, a yellowish-white color, and a viscous consistency. Additionally, the patient experienced pruritic and burning sensations, which worsened in humid conditions. Notably, she frequently used panty liners and practiced vaginal douching with betel soap. Given these symptoms, the patient was referred from a General Hospital with a diagnosis of recurrent bacterial vaginosis (BV), while vulvovaginal candidiasis and trichomoniasis were considered differential diagnoses. An oral ketoconazole 200 mg was prescribed to be taken twice daily for seven days, along with a single 150 mg dose of oral fluconazole. Previously, treatment was carried out using Clindamycin 300 mg, taken twice daily for seven days. However, the symptoms did not improve, and the patient had previously undergone treatment for symptoms at a local clinic for one year. During this period, multiple antibiotic therapies were frequently administered, though the specific medications could not be identified, and no improvement was observed. There was no history of marriage, romantic relationships, or sexual activity. Before the onset of symptoms, significant stress was reported. While studying chemical engineering at a university, considerable academic difficulties were encountered, leading to psychiatric assistance. For the past six months, psychiatric medications have been administered, including 250 mg of divalproex sodium once daily and 20 mg of fluoxetine once daily, following a diagnosis of borderline personality disorder. Medical history also revealed an allergy to metronidazole.

During physical examination, the patient’s vital signs and overall condition were normal, and a dermatological examination showed no skin abnormalities. A venereological examination showed external genitalia within normal limit (Figure 1) but purulent discharge with a pH of 5 was observed at the vaginal and vulvar introitus, with a Nugent score of 5, and 3 of Amsel criteria. A Gram stain examination showed polymorphonuclear leukocytes (PMNs), Gram-positive rod-shaped bacteria, and Gram-positive cocci-shaped bacteria (Figure 2a and b). The patient was given clindamycin 300 mg, taken orally twice a day for seven days. Inspeculo examination could not be performed since the patient never had sexual intercourse, thereby bacterial culture of vaginal and vulvar introitus swabs was performed. The sample of vaginal secretion was taken 2–3 cm from the vaginal introitus, and put into the swab transfer medium, and were examined in the microbiology laboratory. Furthermore, bacterial culture, fungal culture, *Neisseria gonorrhoeae* culture, microscopy, Christie–Atkins–Munch–Petersen test, and resistance test were carried out respectively. The culture was carried out on chocolate agar with a conclusion negative for *Neisseria gonorrhoea*, and blood agar medium which showed growth of three colonies such as *Staphylococcus epidermidis*, *Lactobacillus sp.*, and *β-hemolyticus Streptococcus* (Figure 3). *Staphylococcus epidermidis* was identified using a conventional biochemical test. A catalase test was performed using a 3% H₂O₂ reagent, with a positive result for *Staphylococcus* species. To determine the species of *Staphylococcus*, a novobiocin test was conducted to differentiate between the epidermis and saprophytic species. The result was sensitive and an epidermis was obtained. Subsequent bacterial resistance testing for *β-hemolyticus Streptococcus* showed sensitivity to amoxicillin (33.3 mm), erythromycin (26.3 mm), clindamycin (22.9 mm), moxifloxacin (22.9 mm), and ciprofloxacin (21.7 mm) (Figure 4).

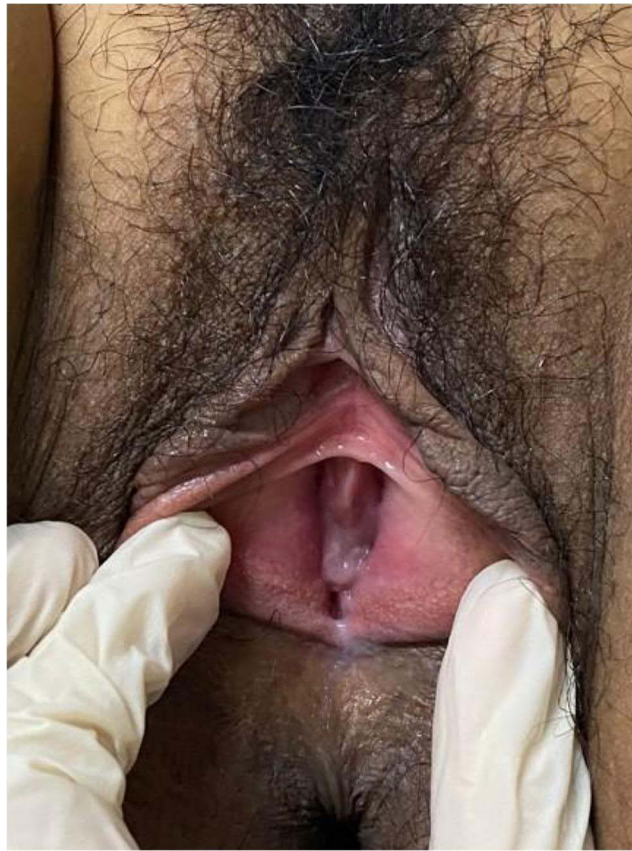


Figure 1 External genitalia showed no sign of inflammation.

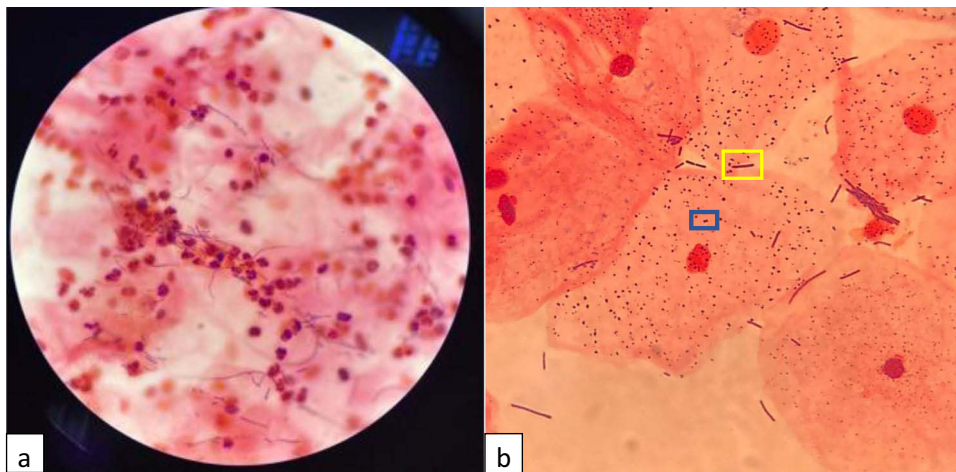


Figure 2 (a) Gram staining from vaginal smears shows polymorphonuclear cells and bacteria, (b) Highlighting scant Gram-positive rod-shaped bacteria (yellow box), and numerous Gram-positive cocci-shaped bacteria (blue box).

Amoxicillin 500 mg was prescribed to be taken orally three times a day, for two weeks. On the 52nd day of observation after taking amoxicillin therapy, the patient reported that the vaginal discharge had disappeared and symptoms had improved.

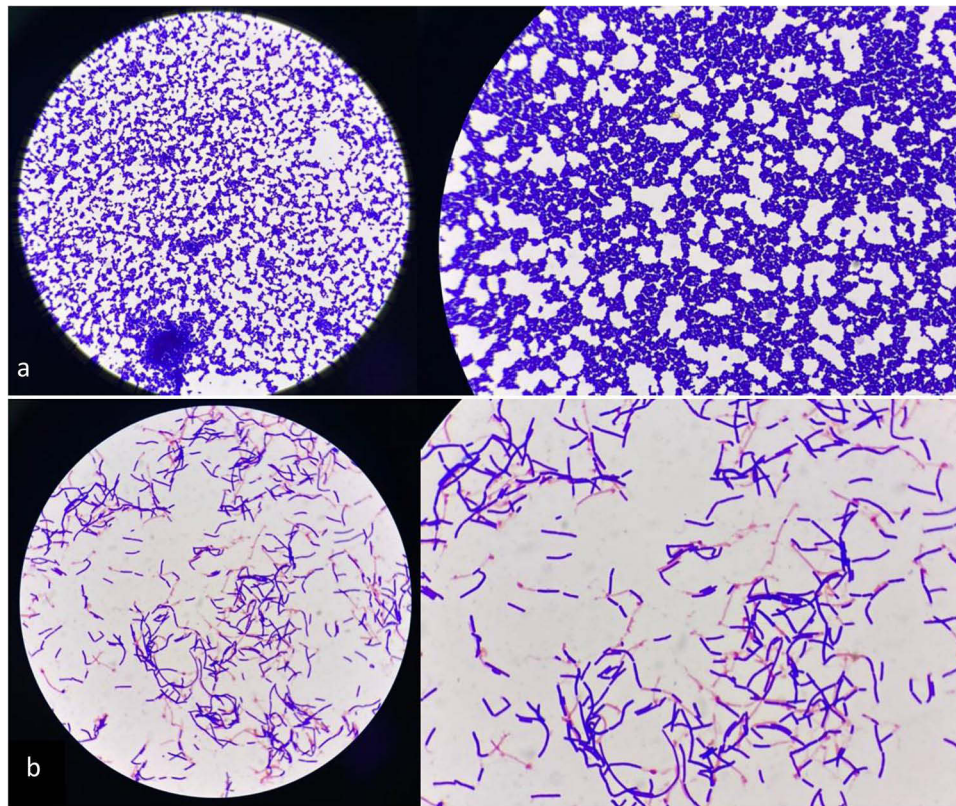


Figure 3 Microscopic examination form bacterial culture specimen found (a) *Staphylococcus aureus* and β -hemolytic *Streptococcus* (b) *Lactobacillus* sp.



Figure 4 Examination of antibiotic resistance in cultures of β -hemolytic *Streptococcus* showed sensitivity to a) amoxicillin, b) clindamycin, and c) ciprofloxacin.

Discussion

The term AV was first introduced by Donders et al¹⁵ in 2002 as a clinical entity distinct from BV. In AV, the transition from a healthy lactobacillus-dominated microbiota to vaginal dysbiosis is characterized by inflammatory symptoms, including vaginal redness, stinging and burning sensations, the presence of thick yellow vaginal discharge, and pain during intercourse. The vaginal discharge is uniform and pus-like, with a yellow or yellow-green appearance. It is accompanied by a rotting, malodorous smell, opposite to the BV with a fishy odor.^{4,16} Patient in this case report, complaining of yellowish-white discharge, sour smell, with pruritus and burning sensation. The signs of inflammation in the external genitalia have decreased, likely as a result of the prior prolonged use of antibiotics.

AV is defined as a disorder of the vaginal microflora (dysbiosis) involving aerobic bacteria, enteric bacteria, vaginal inflammation, and incomplete epithelial maturation.⁴ AV is a type of vaginitis linked to aerobic microorganisms, primarily *group B streptococci* and *Escherichia coli*. Furthermore, other studies have identified additional bacteria as contributors to AV, such as *Enterococcus faecalis*, *Streptococcus viridans*, and *Staphylococcus epidermidis*, which are frequently discovered in patients with AV. All of these microorganisms are aerobic bacteria.¹⁷ In this patient, Gram stain examination showed PMNs, Gram-positive rod-shaped bacterial rods, and Gram-positive bacterial cocci. The patient was then followed for microbiological culture examination of a smear taken from the vaginal introitus, which showed the presence of *Lactobacillus sp.*, *Streptococcus β-hemolyticus*, and *Staphylococcus epidermidis*. The prevalence of Gram-positive bacteria is much higher than the number of *Lactobacillus* in this patient, coupled with complaints of vaginal discharge and changes in pH to alkaline, indicating abnormalities in the condition of the vagina. The Nugent test result also suggested BV, but the culture failed to find the causative bacteria, which indicated that the patient had AV.

The pathogenesis of AV consists interrelated pathogenic processes which involve: (1) A depletion of lactobacillus-dominated microflora, facilitating the adhesion and colonization of various urogenital pathogens, including *group B Streptococcus* and *Staphylococcus aureus*; (2) Dysregulation of local immune responses, characterized by a pronounced elevation in interleukin-1β (IL-1β) levels (178.8 pg/mL) compared to BV (71.2 pg/mL); (3) A marked increase in pro-inflammatory cytokines, such as interleukin-6 (IL-6) and interleukin-8 (IL-8), which is not typically observed in BV, and (4) Estrogen deficiency, inducing a shift in exfoliating epithelial cells from superficial towards intermediate cells, and in more severe cases, towards parabasal cells, as observed in AV.^{4,9}

The following have been identified as distinct risk factors for AV, such as the use of intrauterine devices, external hemorrhoids, prolonged antibiotic use, regular vaginal douching, and stress. Stress, in particular, can contribute to vaginal dysbiosis by disrupting the dominance of *Lactobacillus* species.^{13,14} Stress exposure triggers the release of cortisol and norepinephrine through the hypothalamic-pituitary-adrenal and sympathetic-adrenal-medullary pathways. Cortisol suppresses the growth of vaginal epithelium, which is linked to estrogen maturation and glycogen buildup. This results in reduced levels of vaginal free glycogen and *Lactobacilli*, leading to a decrease in the production of lactic acid and hydrogen peroxide (H₂O₂), as well as a change in pH. Consequently, the dysbiotic environment promotes the growth of pathogenic bacteria.¹³ The risk factors associated with this case emerged during the patient's academic studies. A significant increase in stress was reported, which coincided with the onset of vaginal discharge, suggesting a potential trigger for the initial symptoms. Additionally, a diagnosis of borderline personality disorder was made, and several psychiatric medications were prescribed.

Antibiotic treatment disrupts the microbial balance of the vaginal mucosa. In a healthy vaginal ecosystem, low bacterial diversity, primarily dominated by *Lactobacillus* species, is indicative of normal vaginal flora. In a study by Oh JE et al antibiotic treatment led to a notable reduction in bacterial count, primarily decreasing Gram-positive bacteria, in the vaginal mucosa.³ Douching disrupts the balance of vaginal microflora or triggers inflammation due to physical or chemical irritation, making women more susceptible to infections. Although most commercial douching products consist of fragrance, acetic acid, and water, some also contain surfactants such as octoxynol-9 or cetyl pyridinium chloride.¹⁸ Brotman et al reported that microbiological studies have demonstrated douching's ability to change the composition of vaginal flora, but there are only a few reports, and they are limited to very short observation periods.¹² Onderdonk et al, reported by Brotman et al found that douching with saline or acetic acid resulted in changes to the vaginal microflora within 10 minutes and that normal flora took up to 72 hours to return to pre-douching levels. Exposure to the bactericidal agent povidone-iodine in women resulted in significant short- and long-

term alterations in the vaginal microflora. These changes facilitated the overgrowth of pathogenic organisms with a faster growth rate than *Lactobacillus* species.¹² In contrast, Monif et al, reported by Brotman et al found that normal levels of vaginal flora were restored four hours after iodine spraying. Pavlova and Tao, reported by Brotman et al noted that in broth culture, three douche products containing vinegar selectively suppressed vaginal pathogens linked to BV, group B streptococcal vaginitis, and candidiasis, while at the same time not affecting *Lactobacillus*.¹² The patient had a history of long-term antibiotic use due to complaints of vaginal discharge that did not improve from the previous clinic, and also often performs vaginal douching using betel soap which can increase the risk of AV.

AV represents a shift from a healthy, lactobacillus-dominant vaginal environment to disrupted vaginal flora, marked by clinical symptoms such as inflammation, vaginal redness, burning, and thick and tacky discharge (pH 5.0–8.0 vs normal pH 3.8–4.5). In clinical terms, the condition presents with uniform or pus-like, yellow to yellow-green, and accompanied by an unpleasant, foul smell, but negative on the KOH test.² Bleeding and erosion of the vaginal mucosa may occur. This patient complained of a yellowish vaginal discharge with a slight odor and itching with a pH of 5. The patient was not examined with a speculum due to the absence of a history of sexual intercourse. Instead, a pH test was conducted by applying litmus paper to the labium minus and the vaginal introitus. Samples for Gram staining and bacterial culture were also collected from these areas. The area was initially sanitized to prevent bacterial contamination. A 2-centimeter ose was subsequently inserted into the vagina, avoiding contact with the hymen and other regions of the labia.

To establish the diagnosis of AV, bacteriologic criteria are used. This approach allows a more detailed and comprehensive categorization of the vaginal microbiota, thereby avoiding undefined and vague categories. The vaginal microbiota should be classified into normal and abnormal forms, based on bacteriological criteria. The abnormal vaginal microbiota may be primarily composed of aerobic microorganisms, excessive growth of anaerobic bacteria, or a combination of both. It is crucial to differentiate AV from trichomoniasis, mucopurulent cervicitis, and endometritis. In addition, AV in general is often confused with BV. These conditions may present with similar symptoms, including increased vaginal discharge, a lack of *Lactobacillus*, and elevated pH levels.^{2,16} In BV, a diverse range of anaerobic bacteria, including *Gardnerella vaginalis*, *Atopobium vaginae*, *Ureaplasma urealyticum*, *Mycoplasma hominis*, *Mobiluncus*, are present.¹⁹ In comparison, AV is primarily characterized by a predominance of enteric bacteria, particularly *Escherichia coli* and group B streptococci.^{2,16} The bacterial culture examination did not identify any bacteria associated with the differential diagnoses. However, *Lactobacillus sp.*, β -hemolyticus, *Streptococcus*, and *Staphylococcus epidermidis* were detected. Consequently, the differential diagnoses can be ruled out.

In 2002, Donders et al cited by Han C. et al suggested a microscopic method for diagnosing AV. In 2004, Tempera et al cited by Han C. et al recommended using a combination of clinical features and wet smear microscopy, which includes abnormal yellowish vaginal discharge, increased vaginal pH, and foul odor, but with a negative KOH test and the presence of a high number of leukocytes observed through microscopic examination (magnification 400x) and *Lactobacillus grade* (LBG) Ila, I Ib, or III values, according to Donders et al¹⁶ viz: LBG I represents normal microflora, primarily consisting of lactobacilli, with little to no cocci bacteria present. However, caution should be exercised to avoid confusing cellular cytoplasmic debris from lysed epithelial cells (epithelolysis) with cocci and empty nuclei from lysed epithelial cells with leukocytes. LBG II indicates a reduced number of lactobacilli, mixed with other bacteria. It is further classified into two subtypes based on the balance between lactobacilli and other bacteria: slightly disturbed but relatively normal microflora (LBG Ila) and mildly disturbed, moderately abnormal microflora (LBG I Ib). Lastly, LBG III represents highly abnormal microflora, dominated by various bacteria and lacking lactobacilli.⁴ In AV patients a score of 5 signifies moderate AV.

Management of patients with AV with targeted antibiotic administration or targeted probiotic therapy can have an advantageous influence on the management of AV. Therefore, the management of AV should involve a comprehensive approach that considers multiple strategies, rather than relying on a single antibiotic treatment, taking into account hormonal adjustment in certain cases.⁹ In the study of Mumtaz et al, significant growth of aerobic pathogens was observed in 76.6% of patients with AV. *Staphylococcus aureus* was the most prevalent vaginal pathogen in patients aged 31–40 years, followed by enteric gram-negative bacilli and other gram-positive cocci. Several antibiotics, including third-generation cephalosporins, penicillin, quinolones, sulfonamides, and tetracyclines, are highly effective (over 80%) against common aerobic vaginal pathogens.⁶ Antibiotics should only be used at the beginning of the infection and for a short time to manage symptoms in complex and severe cases, such as staphylococcal or macular streptococcal vaginitis, which are rare and specific forms of severe AV. According to Donders et al,⁹

the use of metronidazole is unlikely to be effective in treating AV, as its microbiota primarily consists of a smaller number of aerobic enteric bacteria, in contrast to the large quantities of diverse anaerobic bacterial subtypes found in BV. Therefore, when metronidazole treatment fails in women with symptomatic vulvovaginitis, it is often a key indicator to consider AV as the underlying cause. On the other hand, using clindamycin as the first-line treatment for AV may have limitations. Infection control may be temporary, it may not target all species involved in AV, and, most concerning, it is susceptible to the development of resistance in patients with recurrent infections, particularly with methicillin-resistant *S. aureus* (MRSA) and group B streptococci. Shifting the focus to Gram-negative bacilli instead of Gram-positive cocci, Tempera et al investigated the local use of 51 mg meclocyline and 100 mg kanamycin to treat AV. These drugs are not absorbed and, like quinolones, help preserve vaginal lactobacilli. After 6 days of treatment, 80% of those treated with meclocyline and 100% of those treated with kanamycin were cured by day 7. However, after 13–16 days, only women treated with kanamycin remained in remission with normal vaginal pH and LBG1 lactobacilli. More recently, excellent treatment outcomes have been reported using vaginal products containing probiotics (107 viable *Lactobacillus acidophilus*) combined with a very low dose of 30 µg estriol (E3) for treating severe vaginal atrophy in menopausal women.² This patient was previously treated with clindamycin 300 mg twice a day for 7 days, but there was no clinical improvement. We performed a bacterial culture and bacterial resistance examination, so the drug administration was based on the results that showed the sensitivity of bacteria to amoxicillin was still good. The patient received a single course of amoxicillin (500 mg) three times daily for 14 days. Following antibiotic treatment, clinical and microscopic improvements were observed. Although probiotics have not been routinely administered to patients with AV, their potential role in future management strategies warrants consideration.

Conclusion

In conclusion, this case emphasized considering aerobic vaginitis as a possible cause of vaginal infections other than BV, vulvovaginal candidiasis, and trichomoniasis, in patients who do not improve despite repeated antibiotics for other suspected vaginitis. In addition, this case also emphasized the importance of proper diagnostic procedures, such as bacterial culture, to determine the appropriate treatment.

Ethical Statement

The publication of images was included in patients' consent for the publication of the case. Institutional approval was obtained to publish the case details from Dr. Hasan Sadikin Hospital Ethical Committee with ethical approval number DP.04.03/D.XIV.6.5/64/2025.

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Disclosure

The authors report no conflicts of interest in this work.

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