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Analytical study of bacterial vaginal infections including antimicrobial assessment in Asser Region, Saudi Arabia

AA Osman*[1], NM Abdalla[2], HE Mubasher[3], MN Mohamed[4]

[1]*Department of Family and Community Medicine, College of Medicine, King Khalid University, Saudi Arabia*

[2]*Department of Microbiology, College of Medicine, King Khalid University, Abha, Saudi Arabia*

[3]*Department of Obs/Gyne, College of Medicine, King Khalid University, Abha, Saudi Arabia*

[4]*Mohamed N Mohamed, Internist Abha General Hospital, Abha, Saudi Arabia*

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Abstract

This study aimed to evaluate antimicrobial susceptibility of vaginal bacterial infections isolates in Asser region, Saudi Arabia. A cross section hospital based study was conducted. Out of 150 sample of high vaginal swab collected through three month, 15 women had bacterial vaginal infections. Each collection swab was initially inoculated into Muller Hinton agar then impregnated antibiotics filter paper was immersed after incubation 48 hrs, the antibiotics sensitivity was determined by measuring the minimum inhibitory concentration (MIC) of each antibiotic, for most common prescribed antibiotics (20drugs). The final result as follows: For *E. coli*, a drug which is of absolute resistance (100%) was Ampcillin and these with 100% sensitive were Nitrofurantoin and Amikacine. Sensitivity of other drugs range from 50 to75% resistance. Regarding Enterobacter, 100% resistance found in 3 drugs, Ampcillin, Augmentin and Cotrimaxcillin. While 100% sensitivity present in 5 drugs which were Nitrofurantoin, Imipenem, Gentamycin, Ciprofloxacin and Amikacin. In *Klebsiella pneumoni* drugs with 100% resistance were Cephalosporin, Ceftazidime, Ampcillin and Augmentin. Almost all other drugs were sensitive. The fourth organism is *Staphylococcus aureus*, 100% resistance found in Ampcillin Augmentin, Pencillin, Cifoxtine. 100% sensitivity in Cotrimxacillin, Tetracycline, Gentamycin and vancomycin. Last organism was *Streptococcus agalactica*, 100% resistance to Ampcillin, Augmentin,Tetracycline, Pencillin, erythromycin and Cifoxatine. Other drugs were 50% sensitive and 50% resistance. As conclusion, treatment of bacterial vaginosis is getting more difficult due to emerge of antibiotics resistance.

* **Corresponding Author:** AA Osman ✉ amanieldosh@gmil.com

Introduction

Vaginitis means inflammation of the vagina. In most cases it is due to a fungal infection. The patient typically has a discharge, itching, burning, and possibly pain. It is frequently linked to an irritation or infection of the vulva. Vaginitis is a very common condition. It is especially common in women with diabetes. (B. Lunenfeld, 2004).

Vagina is a muscular canal from the cervix to the outside of the body. It has an average length of about six to seven inches. The walls of the vagina are lined with mucus membrane People frequently refer to the vagina when really they mean the vulva or female genitals, strictly speaking the vagina is a specific internal structure. The only part of the vagina that can be normally viewed from the outside (without any instruments or carrying out a pelvic examination) is the vaginal opening. The rest of the areas are parts of the vulva, which include the labia majora, mons pubis, labia minora, clitoris, bulb of the vestibule, vestibule of the vagina, etc.

There are several types of vaginitis. The most common are; Atrophic vaginitis (or senile vaginitis) - the endothelium, the lining of the vagina, gets thinner when estrogen levels go down during the menopause. This makes the lining more susceptible to irritation and inflammation. (FR. Ochsendorf, 2006). Bacterial vaginosis caused by overgrowth of normal bacteria in the vagina. Patients usually have less of the normal vaginal bacteria called *lactobacilli*. Trichomoniasis sometimes referred to as trich. It is a sexually transmitted single-celled protozoan parasite *Trichonomas vaginalis*. It may infect other parts of the urogenital tract, including the urethra (where urine comes out of) as well as the vagina. *Candida albicans* this yeast-like fungal organism is what causes thrush. It exists in small amounts in the gut and is normally kept in check by bacteria. Clinically the hallmark symptoms of vaginitis include itching, burning and a discharge. Irritation of the genital area, vaginal discharge, inflammation redness, swelling of the labia majora, labia minora and perineal area; mainly because of the presence of extra immune cells. Dysuria-pain or discomfort when urinating. Dyspareunia - painful sexual intercourse and foul vaginal odor. Aetiologically; Vulvovaginitis inflammation of the vagina and vulva - can affect all women of all ages from every socioeconomic and ethnic backgrounds. Infectious vaginitis makes up 90% of all cases in post-pubescent females. Infectious vaginitis includes candidiasis, bacterial vaginosis and trichmonisasis. Less commonly vaginitis may also be caused by gonnrrhea, Chlamydia, mycoplasma, herpes, campylobacter, some parasites and poor hygiene. Young girls, before they reach puberty, may also develop vaginitis, but the cause is often different from those for older females. While *Streptococcus* spp causes bacterial vaginosis in pre-pubescent girls, for post-pubescent females it is *Gardnerella* (both are types of bacteria). Improper hygiene in pre-pubescent girls can transfer bacteria and/or other irritants to the vaginal area from the anal region. Pre-pubescent girls do not usually get yeast infection because their pH balance is different from older women's. An allergic reaction can cause vaginitis. For example, some women may be allergic to condoms, spermicides, certain soaps and perfumes, douches, topical medications, lubricants, and even semen. Irritation from a tampon can cause vaginitis in some women. (Abdalla, 2011). The diagnosis of vaginitis require physical examination and medical history. A sample of discharge may be taken to try to determine the cause of the inflammation. Vaginitis is diagnosed by checking vaginal fluid appearance, vaginal pH levels, the presence of volatile amines (the gas that causes a bad smell) and the microscopic detection of specific cells. The common Pathogenic agent which might lead to female infertility was shown in table 1.

The type of treatment recommended depends on the cause of the infection, and may include topical (applied onto the skin) or oral antibiotics or antibacterial creams. Antifungals eg. (*Candida albicans*), antiviral eg. (*Herpes simplex*) and antiparasitic eg. (*Entrobious vermicularis* and *Tichomonas vaginalis*). Cortisone cream may be prescribed if irritation symptoms are severe.

An antihistamine may be given if the doctor determines that the inflammation has been caused by an allergic reaction. If the vaginitis was caused by low estrogen levels, a topical estrogen cream may be recommended.

Table 1. Disease Pathogenic agent which might lead to female infertility.

Bacteria	Viruses	Protozoa	Yeasts
Gonorrhoea *Neisseria* gonorrhoea	AIDS HIV	Urethritis due to *Tricho-monas vaginalis*	Balanitis, urethritis due to *Candida albicans*
Chlamydia-infection	Mononucleosis		
Chlamydia trachomatis serotype (D-K)	CMV Asymptomatic infection		
Urethritis (due to) *Ureaplasma urealyticum*	HSV Asymptomatic infection		
Syphilis *Treponema pallidum*	HPV Asymptomatic infection		
Chancroid caused by *Haemophilus ducrey*	Adenovirus Infertility		
Lymphogranuloma venereum by *Chlamydia trachomatis* (L1-L3)			
Granuloma inguinaly			

Sometimes treatment is needed to restore vaginal flora balance, which may have been altered after treatment for an infection. Vaginal flora refers to a balance of bacteria in the vagina that has significant implications for a woman's overall health.

The prevention of vaginitis can be met by good hygiene - keep vaginal area clean. Use a mild soap (without irritants). Avoid douching and irritating agents - many are present in hygiene sprays, soaps, and other feminine products. Avoid wiping from your bottom to your vagina (do it the other way round). Wear loose clothing. Practice safe sex.

Materials and methods

Study design

Analytical cross sectional hospital based study.

Study area

Abha General Hospital, Abha city, Aseer, Western of KSA.

Study Samples

Samples of high vaginal swap (HVS) sent by gynecology department in the period from February to April 2016 was reviewed. Samples of HVS with bacterial results from the total samples were selected. Antimicrobial resistant test was conducted to all samples with bacterial infection.

High Vaginal Swab (HVS)

High Vaginal Swab (HVS) is a technique used in Obstetrics and Gynaecology to obtain a sample of discharge from the vagina. This is then sent for culture and sensitivity. Samples should be transported to laboratory immediately. If this is not possible the sample should be stored at room temperature and must reach the laboratory within 48 hours of collection. It is commonly used to test for the presence of candidiasis infection, bacterial vaginosis and trichomonas vaginalis. (Niger, 2014)

Antimicrobial Sensitivity Tests (Kirby- Pauer Diffusion Disk)

Vaginal swabs collected from all 150 new cases presented with vaginal secretions will be collected. Direct microscopy using gram stain and cultured in Blood Agar to assess the hemolysis. Subcultured on blood agar and/or nutrient and Muller Hunter agar, incubated at 35°C for 18 to 20 h, these cultures will be put in a bactech system and/or suncultured in nutrient agar to which antibiotic laden filter papers will be applied then kept for 48 hrs in incubator at 36C^{o} and then by measuring the clear zone around each antibiotic disc (MIC) the sensitivity and resistance will be assessed.

Exclusion criteria

All results of HVS with non hemolytic gram positive bacteria such as *Staphylococus epidermidis* and *saprophyticus*.

Non hemolytic Streptocccal spp., intracellular bacteria such as *Chylamdia spp*., other bacteria *Mucobacterium tuberculosis* and cell wall deficient bacteria, Mycoplasma and Ureaplasma, anaerobics such as *Bcteroides fragilis* and *Provetella sp*. Gram negative cocci such as *Hemophilus influenza* and *Nisseria gonorrheoa*. Parasitic such as *Trichomonas vaginalis*, fungal eg. *Candida albicans*, viral infections and normal commensal such as *Lactobacillus* spp.

Results and discussion

A total of 150 samples of HVS conducted during three month in gynecology department, Abha general hospital. Among them there were 15 cases of bacterial infection, which represent 10% of the total sample. Five pathogenic organism were isolated, they were, *E. coli*, *Enterobacter* sp., *Klebsiella pneumonia*, *Staphylococcus aureus* and *Streptococcus agalactica*. Antimicrobials resistance profile was done to these organisms by using 20 commonest antibiotics as shown in table 2.

Table 2. Five pathogenic groups isolated by HVS against 20 common used antimicrobials resistance profile.

Anti-Microbials Groups	E. coli	Entero-bacter spp.	*Klebsiella pneumoniae*	*Staphylo-coccus* aureus	*Strepto-coccus* agalactica
Penicilin	R 50%	Not done	Not done	R 100%	R 100%
Methicillin	R 50%	Not done	Not done	R 66%	R 50%
Erythromycin3	Not done	Not done	Not done	R 66&	R 100%
Cifoxtine	R 25%	R 100%	S 100%	R 100%	R 100%
Tetracyclin3	R 50%	S 50%	S 100%	S 100%	R 100%
Vancomycin	Not done	Not done	Not done	S 100%	R 50%
Fucidin3	R 50%	Not done	Not done	R 66%	R 50%
Augmantin	R 50%	R 100%	R 100%	R 66%	R 100%
Ampicilin	R 100%	R 100%	R 100%	R 100%	R 100%
Amikacin3	S 100%	S 100%	S 100%	R 33%	R 50%
Cefaclor	Not done	Not done	Not done	R 66%	R 50%
Ciprofloxacin4	R 50%	S 100%	S 100%	S 66%	R 50%
Gentamycin3	R 50%	S 100%	S 100%	S 100%	R 50%
Carbincilin3	S 50%	Not done	Not done	R 33%	R 50%
Cotrimxacillin5	75%	R 100%	S 75%	S 100%	R 50%
Imipenem	R 50%	S 100%	S 100%	S 66%	R 50%
Ceftazidime	R 75%	R 50%	R 100%	S 66%	R 50%
Cephalosporine	S 75%	R 50%	R 100%	S 33%	S 50%
Nitrofurantoin4	S 100%	S 100%	S 100%	S 66%	S 50%
Rifampin4	Not done	Not done	Not done	S 33%	S 50%

S= sensitive R=resistance

For *E. coli* drugs absolute resistance 100% was Ampcillin and these 100% sensitive were Nitrofurantoin and Amikacine. Sensitivity of other drugs range from 50 to75% resistance.

Regarding Enterobacter, 100% resistancy found in Ampcillin, Augmentin and Cotrimaxcillin. While 100% sensitivity present in 4 drugs which were Nitrofurantoin, Imipenem, Gentamycin, Ciprofloxacin and Amikacin.

In *Klebsiella pneumoni* drugs with 100% resistance were Cephalosporin, Ceftazidime, Ampcillin and Augmentin. Almost all other drugs were sensitive.

The fourth organism is *Staphylococcus aureus,* 100% resistance found in Ampcillin Augmentin, Pencillin, Cifoxtine. 100% sensitivity in Cotrimxacillin, Tetracycline, Gentamycin and vancomycin.

Last organism was Streptococcus agalactica, 100% resistance to Ampcillin, augmentin, tetracycline, pencillin, erythromycin and Cifoxatine. Other drugs were 50% sensitive and 50% resistance.

In table 3, Antimicrobial resistance was done against the five pathogenic organism isolated (*E. coli, Enterobacter, Klebsielpneumone, Staphylococcus aureus and Streptococcus agalactica*) the antimicrobial used has different mode of action. The first group is Cell wall inhibitor; this group includes around 10 commonly used antimicrobials drugs. Over this entire group the most resistance organism was *Streptococcus agalactica* and *E. coli,* while the least resistance is *Staphylococcus aureus*.

Table 4 showed the second group used was that work in Protein Synthesis inhibitors. There were 6 common used drugs, the most resistance organism *Streptococcus agalactica*, and the most sensitive organism to this group was *Klebsiela pneumoni.*

The third antimicrobial group is Inhibitors of nucleic acids synthesis. It includes common 3 drugs, It looks all organism were sensitive to this group, as shown in table 5.

The fourth group is antimetabolite (antifolate) inhibitor group. The most sensitive organism was *Staphylococcus aureus* while *Enterobacter sp*. was the most resistant one to this group as shown in table 6.

Table 3. Five pathogenic groups isolated by HVS against 10 common used antimicrobials of Cell wall inhibitors group.

Anti-Microbiials Groups	*E. coli*	*Enterobacter* spp.	*Klebsiella pneumoniae*	*Staphylo-coccus aureus*	*Streptococcus agalactica*
Penicilin	R 50%	Not done	Not done	R 100%	R 100%
Methicillin	R 50%	Not done	Not done	R 66%	R 50%
Cifoxtine	R 25%	R 100%	S 100%	R 100%	R 100%
Vancomycin	Not done	Not done	Not done	S 100%	R 50%
Augmantin	R 50%	R 100%	R 100%	R 66%	R 100%
Ampicilin	R 100%	R 100%	R 100%	R 100%	R 100%
Cefaclor	Not done	Not done	Not done	R 66%	R 50%
Imipenem	R 50%	S 100%	S 100%	S 66%	R 50%
Ceftazidime	R 75%	R 50%	R 100%	S 66%	R 50%
Cephalosporine	S 75%	R 50%	R 100%	S 33%	S 50%

S= sensitive R=resistance

Table 4. Five pathogenic groups isolated by HVS against 6 common used antimicrobials resistance profile Protein Synthesis inhibitors groups (S 50 & S 30).

Anti-Microbiials Groups	*E. coli*	Entero-bacter spp.	*Klebsiella pneumoniae*	*Staphylococcus aureus*	*Streptococcus agalactica*
Erythromycin S 50	Not done	Not done	Not done	R 66&	R 100%
Tetracyclin S 30	R 50%	S 50%	S 100%	S 100%	R 100%
Fucidin S 50	R 50%	Not done	Not done	R 66%	R 50%
Amikacin S 30	S 100%	S 100%	S 100%	R 33%	R 50%
Gentamycin S 30	R 50%	S 100%	S 100%	S 100%	R 50%
Carbincilin S 50	S 50%	Not done	Not done	R 33%	R 50%

S= sensitive R=resistance

Table 5. Five pathogenic groups isolated by HVS against 3 common used antimicrobials resistance profile, Inhibitors of nucleic acids synthesis group.

Anti-Microbiials Groups	*E. coli*	Entero-bacter spp.	*Klebsiella pneumoniae*	*Staphylo-coccus aureus*	*Strepto-coccus agalactica*
Ciprofloxacin	R 50%	S 100%	S 100%	S 66%	R 50%
Nitrofurantoin	S 100%	S 100%	S 100%	S 66%	S 50%
Rifampin	Not done	Not done	Not done	S 33%	S 50%

S= sensitive R=resistance

Table 6. Resistance profile for the Five pathogenic organism isolated by HVS against one common used antimicrobial drug from Antimetabolite (antifolate) inhibitor group.

Anti-Microbiials Groups	*E. coli*	Entero-bacter spp.	*Klebsiella pneumoniae*	*Staphylo-coccus aureus*	*Strepto-coccus agalactica*
Cotrimxacillin5	R75%	R 100%	S 75%	S 100%	R 50%

S= sensitive R=resistance

In Western countries STD-infections are of minor relevance. In other regions, i.e. Africa or South East Asia, the situation appears to be different. Chronic infections (gonorrhoea) can cause urethral strictures and epididymoorchitis.

Chlamydia trachomatis and *Neisseria gonorrhoea* can be transmitted to the female partner and cause pelvic inflammatory disease with tubal obstruction. *Ureaplasma urealyticum* may impair spermatozoa (motility, DNA condensation). *Trichomonas vaginalis* has, if any, only minor influence on male fertility. The relevance of viral infections (HPV, HSV) for male infertility is not resolved. Any STD increases the chances of transmission of the human immunodeficiency virus (HIV). The HIV infection is associated with infectious semen and the risk of virus transmission. Semen quality deteriorates with the progression of immunodeficiency. Special counselling of serodiscordant couples is needed. STDs should be treated early and adequately to prevent late sequelae for both men and women (Dohle, 2003).

Gonorrhoea, however, may impair male fertility. Gonorrhoeic urethritis was associated with urethral strictures (Zhou, 2004). Few cases had urithritis was identified by urine analysis. This may be explained by large differences in the prevalence of STDs in different regions of the world. Excluding human immunodeficiency virus (HIV) infections, the prevalence ranges from 4 per million in Western Europe to 32 in Sub-Saharan Africa and 48 in South East Asia with annual incidences of 17, 69 and 151 per million respectively.

Epidemiological data propose an association of a past *C. trachomatis* infection and subfertility both in men and women (Karinen, 2004; Eley, 2005). It is accepted that *C. trachomatis* impairs female fertility by causing tubal obstruction (Eggert-Kruse,1990). The exact role of Mycoplasmae, i.e. *M. hominis, U. urealyticum* and *M. genitalium* has still to be elucidated. It was shown that *M. genitalium* can attach to spermatozoa and thus can be transported to the female genital tract (Sakar, 2008).

Ureaplasma urealyticum may cause infertility via deleterious effects on sperm chromatin and DNA, leading to impairment of embryo development (Gorkemli, 2006). In the group of infertile patients with PCR positive for *U. urealyticum,* the volume, count and morphology of semen samples were lower than in the infertile patients with PCR negative results. *Trichomonas vaginalis* was more often found in infertile women than in fertile controls. A large women population of the world is suffering from a vaginal infection commonly known as bacterial vaginosis. The disease is associated with the decrease in the lactobacilli count in the vagina. Till date, there is a lack of full proof treatment modalities for the cure of the disease.

The treatment includes the use of antimicrobials and/or acidifying agents and probiotics, either separately or in combination (Nikhil, 2011).

The vagina it harbors a number of microorganisms and *Lactobacillus*is the predominant species as normal flora. Gram positive non hemolytics such as *Staphylococus epidermidis* and *saprophyticus* (skin normal flora). Fungal infection (Candida albicans) parasitic (*Trichomonas vaginalis*) viral (HIV) were not included in the study.

Glycogen, an analogue of starch found in animals, is the main source of nutrients for the microbial flora residing in the lumen of the vagina. The metabolism of glycogen in the vaginal system is mediated by the estrogen hormone via estrogen receptors located in the epithelial cells covering the vaginal lumen (Owen, 1975). The quantity of the mucus, estrogen level increases. The normal pH of the vagina 3.5-4.5 6 vaginal microflora. In general, it is regarded that lactobacilli species is the predominant microflora responsible for maintaining the pH of the vaginal lumen (Eschenbach, 2000).

The vagina has been attributed to the regular contact of the external vaginal structures with the urine. (Hawthorn, 1991; Otero, 2007) gram negative such as *E. coli* and *Klebsilla* spp. undergo physicochemical interaction with the vaginal epithelia, which helps in the colonization of these pathogens as well as the lactobacilli leading to biofilm formation within the mucosal and the epithelial layer of the vagina (Busscher, 1987). The biofilm consists of the bacterial cell layer(s) and the secretory components from the vagina (Bibel, 1987).

Vaginal secretions, especially mucus known as, post-menopausal atrophic vulvo-vaginitis. This results in the decrease in the lubrication of the vagina causing discomfort during coitus (Kamarashev, 1997). In addition, there is a decrease in the thickness of the epithelial layer thereby increasing the susceptibility of the vaginal tissue toward infection and associated irritation (Farage, 2006).

In normal healthy women, there is an increase in the dynamic nature of the microflora with an increase in the hydration of the epithelial layer (Freinkel, 1969). This results in the decreased permeability of the mucosal layer for the pathogenic organisms.

The metabolic products secreted by the microbes may influence the availability of the nutrients (Elias, 2007). Fatty acids have shown antimicrobial activity (against *Streptococcus pyogenes, Staphylococcus aureus*, and skin micrococci) and may help in fine tuning the composition of the microbial flora (Kabara, 1972). They failed to produce substantial activity against gram-negative bacteria. (Ouattara B, 1997) Apart from this, some peptides have also shown antimicrobial activity against various pathogenic bacteria, fungi, viruses, and protozoa (Zasloff 2002).

A large number of transient organisms continuously migrate from the exogenous source (e.g., anus and urethra).

Among the *Lactobacillus* species, *Lactobacillus acidophilus* was considered to be the dominant microbe present in the vaginal microflora. (Lachlak N 1996, Vásquez A, 2002) The molecular identification techniques have the ability to specify the species easily in case of diverse microflora.

In general, the lactobacilli are replaced with the increased population of pathogenic gram-negative anaerobic bacteria such as *E. coli, Gardnerella vaginalis, Mycoplasma hominis*, and *Mycoplasma curtisii* (Hillier S.2005, Hill GB. 1993). This condition may lead to several complications, which include continuous vaginal discharge, high HIV risk, malodor (fishy smell), stomach pain, abortion, infertility, preterm birth, chorioamnionitis, and urinary tract infection. (Darwish A, 2007) researchers have associated BV with various factors including vaginal douching by the use of scented soaps or perfumed bubble bath and antiseptics during bath, (MA. Klebanoff 2010) multiple sex partners and/or a new sex partner, smoking (Ryckman KK, 2009) and use of contraceptives (e.g., spermacides) which may increase the probability of the infection in a women.

The presence of a strong fishy smell indicates that the patient is suffering from BV. The microscopic examination of the vaginal smear, which is analyzed for the presence of bacteria, white blood cells and clue cells, and the presence of clue cells, indicates BV. The gram staining method, introduced by Dunkelberg in the year 1965, is also a simple method for the diagnosis of BV (Dunkelberg WE, 1965). the method helps in the confirmation of the presence of gram-positive and gram-negative bacteria in the vaginal discharge. Recently, a scoring system (on a scale of 10) based on the presence of large gram-positive rods, small gram-negative or variable rods, and small curved gram-negative to variable rods have been reported. If the score lies in between 7 and 10, then the patient is suffering from BV (M. Joesoef, 1999).

A lot of antimicrobial agents (e.g., ampicillin, penicillin, and metronidazole) have been used in the treatment of bacterial vaginitis. (Spiegel CG,1991) Metronidazole have evolved as a drug of choice for the treatment of BV and is the widely prescribed drug. It is a nitroimidazole derivative having activity against anaerobic microbes and protozoans. Administration of the drug include gels and suppositories. (Sobel JD, 2006) Metronidazole and tinidazole (a chemical analogue of metronidazole) are preferred for the treatment of BV as against ampicillin. Tinidazole has a better pharmacokinetics and longer half-life than metronidazole and its recommendation for the treatment of BV is on the rise. (Dickey LJ, 2009) The use of ampicillin is avoided due to the emergence of ampicillin-resistant bacteria in patients with BV. It also inhibits the growth of lactobacilli. (Spiegel CG, 1991) Reports on clindamycin have suggested that it can be used in the treatment of BV and may be administered either orally or locally. (Mc Gregor JA, 1994). Development of antimicrobial resistance mechanism within the microbes. (RH. Beigi, 2004). Such as B lactamase, MRSA, Mc A gene, Biofilm and Absence of Enzymes such as RNA polymerase in Rifampicin Resistance. Hence, the researchers and clinicians are looking for alternative methods for the treatment of BV.

The absence of *Lactobacillus* from the vagina is the specific feature of BV. (SL. Hillier,1993). Lactic acid-producing bacteria, as probiotic. Various commercially available *Lactobacillus*-based products for the treatment of BV include yoghurt, acidophilus milk, and available *Lactobacillus* powder and tablets. (Hughes VL, 1990)

Vaginal douching is the treatment helped in improving the conditions of the patients. (M. Tasdemir,1996) , the vaginal pH was within the normal range and the presence of *Lactobacillus* within the lumen was also observed. (Neri A,1993)

The use of probiotics for the treatment of BV has provided a ray of hope by natural and nontoxic treatment modality. (Dover SE, 2008) Apart from the above, the probiotics may offer cost-effective treatment of BV.

Conclusion

People can help tackle resistance by: hand washing, and avoiding close contact with sick people to prevent transmission of bacterial infections and viral infections such as influenza or rotavirus, and using condoms to prevent the transmission of sexually-transmitted infections; getting vaccinated, and keeping vaccinations up to date; using antimicrobial drugs only when they are prescribed by a certified health professional; completing the full treatment course (which in the case of antiviral drugs may require life-long treatment), even if they feel better; never sharing antimicrobial drugs with others or using leftover prescriptions.

Health workers and pharmacists can help tackle resistance by: enhancing infection prevention and control in hospitals and clinics; only prescribing and dispensing antibiotics when they are truly needed; prescribing and dispensing the right antimicrobial drugs to treat the illness. Policymakers can help tackle resistance by: improving monitoring around the extent and causes of resistance; strengthening infection control and prevention;

regulating and promoting appropriate use of medicines; making information widely available on the impact of antimicrobial resistance and how the public and health professionals can play their part; rewarding innovation and development of new treatment options and other tools. Policymakers, scientists and industry can help tackle resistance by: fostering innovation and research and development of new vaccines, diagnostics, infection treatment options and other tools.

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