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# Multi drug resistance of bacterial isolated in patient with vaginosis

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***Abstract*---**Vaginal infection It is one of the most common illnesses in the world. affecting women's health, with more than half of all women experiencing at least one episode in their lifetime. Vaginal disease can be both infectious and chronic. Bacterial vaginosis (BV), vaginal candidiasis, and vaginal trichomonas are the three most common types of infectious vaginitis based on microbial etiology (Abdul-Aziz et al., 2019). Antimicrobial-resistant (AMR) infections are caused by microbes that are resistant to antimicrobial agents. As bacteria develop resistance, many previously effective drugs no longer work (Ali, 2018). Each year, AMR infections kill 700,000 people around the world. By 2050, antimicrobial resistance may be responsible for the deaths of up to 10 million people annually. De Kramer and colleagues (2016): According to a recent research study (Amirkamali et al., 2017), antibiotic resistance genes blab-TEM may be able to predict the presence of antibiotic resistance in bacteria.

***Keywords*---**multi drug resistance, bacterial isolated, vaginosis.

## Introduction

Vaginal infection It is one of the most common illnesses in the world. affecting women's health, with more than half of all women experiencing at least one episode in their lifetime. Vaginal disease can be both infectious and chronic. Bacterial vaginosis (BV), vaginal candidiasis, and vaginal trichomonas are the three most common types of infectious vaginitis based on microbial etiology (Abdul-Aziz *et al.*, 2019).Antimicrobial-resistant (AMR) infections are caused by microbes that are resistant to antimicrobial agents. As bacteria develop resistance, many previously effective drugs no longer work (Ali, 2018). Each year, AMR infections kill 700,000 people around the world. By 2050, antimicrobial resistance may be responsible for the deaths of up to 10 million people annually. De Kramer and colleagues (2016): According to a recent research study

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(Amirkamali *et al.*, 2017), antibiotic resistance genes blab-TEM may be able to predict the presence of antibiotic resistance in bacteria.

## Material and Methods

During the period from November 1, 2021 to March 1, 2022, a total of 40 vaginal swabs were collected from patients with vaginitis who were identified by specialized gynecologists and submitted to the Center in AL-Zahra Teaching Hospital in Najaf Governorate/Ministry of Health/Iraq. The patients' ages ranged from 17 to 47 years and 40 blood samples.

## specimens Collection

vaginal specimens are collected from married and non-married women suffering from vaginitis. These specimens are collected by gynaecologists. Swabs were placed in the posterior fornix and upper part of the vaginal canal. Swabs were rubbed in and around the introits of the cervix and withdrawn without contaminating the vaginal wall, using a vaginal speculum to provide a clear view of the cervix. Swabs are kept moist until they are taken to the laboratory and placed in tubes containing transport media inoculated each specimen into blood, MacConkey, and EMB agar and incubated it for 24 hours at 37° ( Fielder *et al.*, 2013).

## Bacterial isolation and identification in the laboratory

The steps recommended by McFadden (2000) for diagnosis are to take a single colony from each positive culture and then identify it based on morphological properties (color production, colony shape, texture, and edge), and to use Gram's stain for detection. The morphological properties of bacterial cells also help to distinguish between Gram's negative and Gram's positive bacteria (Al-Yasari, 2006).

## Antibiotic susceptibility test

The kibry-Baure susceptibility test was investigated using a pure culture of a selected identified bacterial isolate, followed by the addition of antibiotic discs to each plate and an 18-hour incubation period, with the inhibition zone being measured to classify bacterial sensitivity (Marsik, 2010).

## Ethical Approval

All patients involved in the research study gave their ethical approval.

## Result and Discussion

The age parameter of vaginal infection females was divided into three groups in the current study. The age group, 26-35 years, n=20/40 (50%), was most affected by vaginal bacterial infection, as shown in figure (4.1).

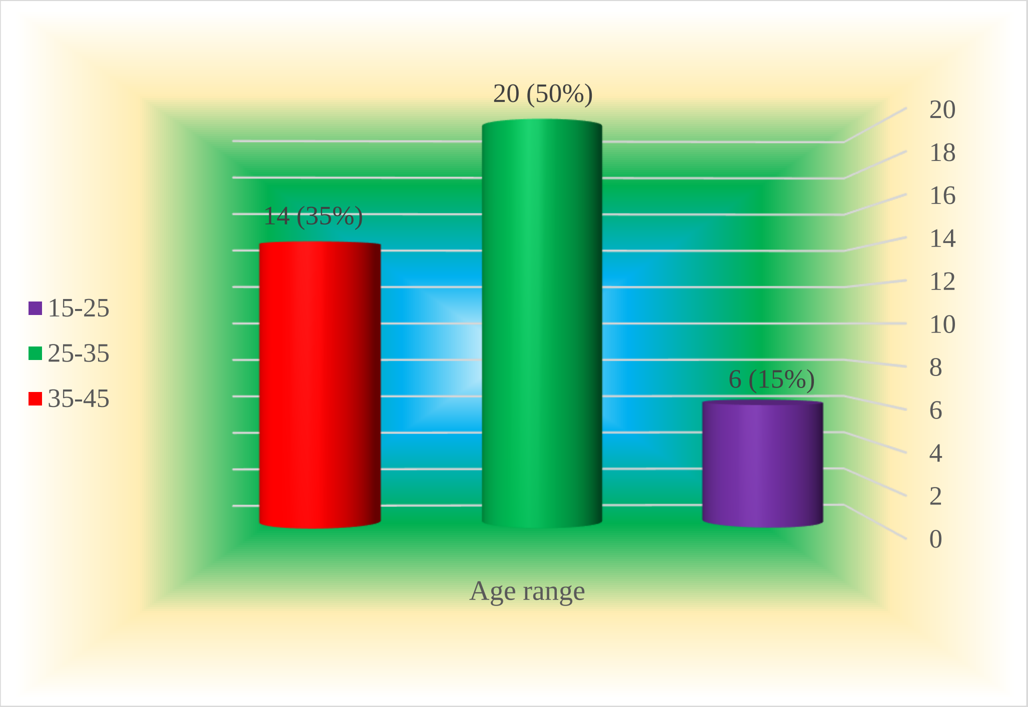

Figure (3): Distribution bacterial vaginosis according to age.

Women aged 25 to 35 years had the highest prevalence of BV, which was similar to what Ranjit *et al.* (2018) found in women aged 30 to 40 years. In contrast to our findings, Kamga *et al.* (2019) discovered that women aged 18–22 years had the highest prevalence of BV, which is consistent with Ibrahim *et al.* (2019). The most common age group is 21–29-year-old women. These are the most sexually active age groups, and as a result, they are at the highest risk of contracting BV and STDs (Mengistie *et al.*, 2014).

According to the findings of the current study, the housewives group had the highest percentage of patients (20/50, 50%), as shown in the figure (4.2).

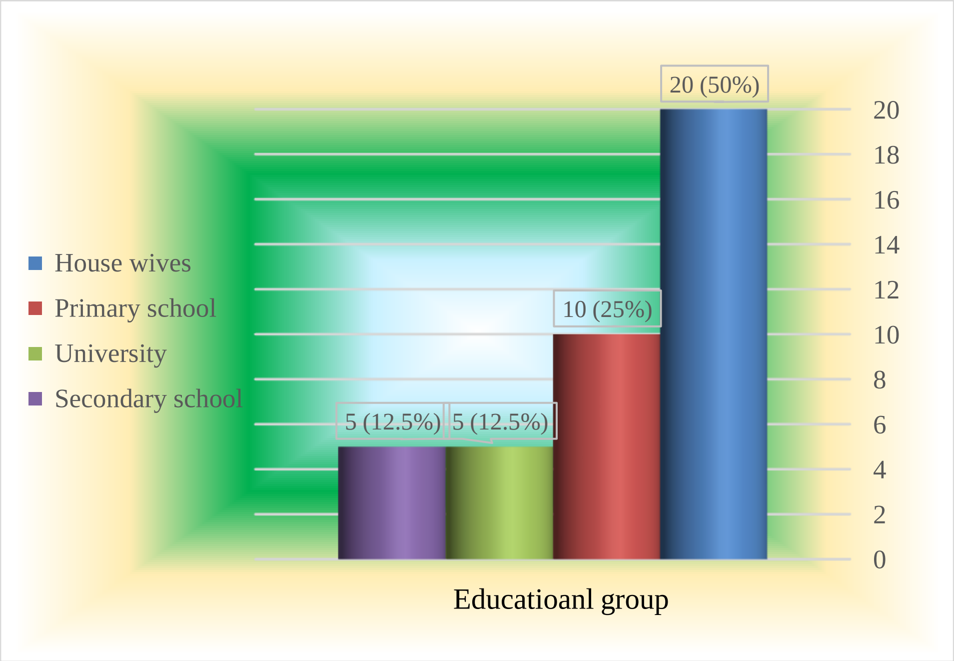

Figure 4-2: Distribution of vaginal bacterial infections females according to educational levels

**Antimicrobial Profile**

In the current study, antibiotic resistance was found in the following isolates: isolate 9 (55.5 percent), isolate 8 (44.4 percent), isolate 7 (44.4 percent), isolate 6 (33.3 percent), isolate 5 (66.6 percent), isolate 4 (44.4 percent), isolate 3 (55.5 percent), isolate 2 (33.3 percent), and isolate 1 (22.2 percent). It is shown in the following tables (e.g., 4-3).

Table (4-3): Antibiotic profile of *E. coli* isolate

| Antibiotic | No. 1 | No. 2 | No. 3 | No. 4 | No. 5 | No. 6 | No. 7 | No. 8 | No.9 |
|---|---|---|---|---|---|---|---|---|---|
| Getamicin | 20 | 17 | 15 | 13 | 15 | 20 | 14 | 18 | 15 |
| Interpretation | S | S | S | S | S | S | S | S | S |
| Nitrofurantoin | 15 | 13 | 13 | 10 | 15 | 17 | 13 | 8 | 14 |
| Interpretation | S | S | S | S | S | S | S | S | S |
| Pencillin G | 10 | 0 | 0 | 0 | 8 | 0 | 9 | 10 | 0 |
| Interpretation | S | R | R | R | R | R | R | R | R |
| Imipenem | 24 | 24 | 24 | 24 | 22 | 26 | 24 | 22 | 20 |
| Interpretation | S | S | S | S | S | S | S | S | S |
| Tetracycline | 15 | 11 | 0 | 16 | 0 | 10 | 8 | 13 | 0 |
| Interpretation | S | S | R | S | R | S | R | S | R |
| Ciprofloxacin | 22 | 20 | 18 | 0 | 0 | 24 | 20 | 24 | 16 |
| Interpretation | S | S | S | R | R | S | S | S | S |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Trimethoprim | 20 | 20 | 8 | 14 | 0 | 0 | 8 | 22 | 0 |
| Interpretation | S | S | R | S | R | R | S | R | R |
| Ceftazidim | 0 | 0 | 0 | 0 | 0 | 11 | 0 | 0 | 0 |
| Interpretation | R | R | R | R | R | S | R | R | R |
| Rifampin | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Interpretation | R | R | R | R | R | R | R | R | R |
| percent | 22.2 | 33.3 | 55.5 | 44.4 | 66.6 | 33.3 | 44.4 | 44.4 | 55.5 |

Antibiotic resistant genes on chromosomes or plasmids cause multi-resistance bacteria, which aids in horizontal resistance transfer. The frequent use of third-generation cephalosporin antibiotics to treat bacterial infections has become a major risk factor, as bacteria can develop resistance to these antibiotics through the production of the ESBL enzyme. The widespread use of beta-lactam antibiotics in the community and hospitals has resulted in an increase in mortality, morbidity, and health-care costs (Blomberg *et al.,* 2005).

It is difficult to treat *E. coli* infections because the normal intestinal microbial flora has become an antimicrobial resistance reservoir over time (Okeke *et al.*, 2000). Antibiotic resistance in bacteria may be the result of a genetic response to the high selective pressure imposed by antimicrobial chemotherapy. Resistant bacteria receive the plasmid containing the plasmid gene (Salyers *et al.*, 2004).

Antibiotic resistance can be attributed to a variety of factors, including but not limited to: The beta-lactamase enzyme can be produced by microorganisms that can destroy the antibiotic, such as E. coli, which can break down the lactame ring of the drug. (2) alter the permeability of microorganisms to drugs. Antibiotics build up in bacteria that are susceptible to them, but not in bacteria that are resistant to their effects. (3) altered the microorganisms' structural targets of the antibiotic. (3) Modifications to the ribosome's 50S subunit and/or PBPs, for example. The antibiotic used to treat the disease-causing bacteria must be rendered inactive by establishing a metabolic pathway in microorganisms. Antibiotics were used by Brooks *et al.*, (2010). but a modified enzyme could be developed by microorganisms that can still perform metabolic functions .

Despite the fact that incidence rates have been steadily rising, the studies included in this review showed a wide range of results. Due to a variety of definitions of ESBL-producing microorganisms, the variation is possible. Identifying Due to the difficulty of obtaining accurate laboratory results, determining the true incidence of ESBL can be difficult. ESBLs, which may not be present in all samples, must be tested for. A variety of strains of E. coli and Klebsiella may be found throughout the course of the disease. It's study time.

Finally, ESBL-E. coli and ESBL-Klebsiella infections are becoming more common across the United States. Effective antimicrobials may be influenced by these findings. Tracking and treating these infections is essential. a necessity for ensuring responsible use of antimicrobials (Unlu et al., 2021). Anti-infective measures are employed. Bacteria that produce ESBL are It is crucial to the development of state and national surveillance systems (Gigante et al., 2022). The use of systems and multi-center or national studies is essential to Find out how common something is right now. In the absence of accurate estimations, multi-drug-resistant infections, such as ESBLs and MDRs. Research on pathogens, however, remains a challenge. and prevention efforts (McDanel *et al.,* 2017).

Antibiotics known as extended-spectrum beta-lactams (ESBLs) are commonly used to treat serious gram- negatives infections. however, the production beta with a broad spectrum- lactamases has resulted in bacterial resistance (O'Kelly *et al.,* 2016). Beta-lactamases are enzymes that cause bacterial resistance to antibiotics in the beta-lactam family, including penicillins, cephalosporins, cephamycins, and carbapenems. These enzymes catalyze the hydrolysis of the four-membered beta-lactam ring's amide bond, rendering the antibiotic inactive against its cellular target, cell wall transpeptidase (Culyba *et al*., 2015).

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