Evaluation of Antibiotic Resistance Pattern of Bacterial Isolates from the Genital Tract

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Abstract :

Background: infections caused by antibiotic resistance bacteria constitute major public health issue . The increasing prevalence of genital tract infections (GTIs) has become a significant challenge due to rising antibiotic resistance. therefore, this study aimed to identify antibiotic susceptibility patterns of bacterial isolates obtained from genital tract sample.

Methodology:

Samples were collected from May 2023 to December 2024, 168 genital samples (109 males and 59 females) from two hospitals in Baghdad, Iraq. These samples included semen, cervical smears, urethral secretions, and upper vaginal swabs. Isolates were identified after cultured on standard media, and their antimicrobial susceptibility testing was performed using the VITEK 2 Compact system.

Results:

The results revealed that out of 113(67%) samples, 55 samples exhibited microbial growth, while the remaining samples showed no microbial growth.

Over time, a significant decrease in microbial growth showed from females samples. the most common pathogens included *Staphylococcus aureus, Staphylococcus hemolyticus, Escherichia coli, Enterococcus faecalis, and Klebsiella pneumoniae*. In 24 cases, Candida albicans was isolated only from females. A widespread prevalence of multidrug resistance (MDR) was observed, particularly among Gram-positive cocci. *Enterococcus faecalis* exhibited erythromycin resistance (ER) of 100% and vancomycin resistance (VD) of 17.6%. While *Escherichia coli* and *Klebsiella pneumoniae* showed varying degrees of resistance, carbapenems and aminoglycosides retained significant efficacy against them. Methicillin-resistant Staphylococcus aureus (MRSA) strains were also identified.

Conclusion:

This study reveals the substantial variety of the microbial environment in genital tract infections and shows the occurrence of multidrug-resistant species.Based on these results, it can concluded that continuous monitoring and judicious antibiotic use are crucial for successful therapeutic management and avoiding the induction of resistance.

Keywords:

Genital tract infections, antimicrobial resistance, VITEK 2, MRSA, Candida, Staphylococcus aureus , Enterococcus faecalis.

Introduction:

Millions of people worldwide suffer from genital tract infections (GTIs) annually, which can lead to various problems, such as pelvic inflammatory disease (PID), pregnancy complications, infertility, and an increased risk of sexually transmitted infections (STIs) [1, 2]. Furthermore, the prevalence patterns of resistance are significantly influenced by several factors including geographic location , gender, and population demographic which applies to a wide spectrum of microorganisms causing Genital tract infections (GTIs), ranging from bacteria to fungi [3].

While vaginal candidiasis caused by Candida albicans remains common among women, recent epidemiological trends indicate an increase in infections with other species besides *Candida albicans*, such as *Candida glabrata* and *Candida cruzi* which often exhibit reduced responsiveness to azole antifungals [4, 5].

Antifungal resistance is a growing problem, complicating empirical treatment approaches [6]. Research has shown an increase in fluconazole-resistant Candida isolates. *Staphylococcus aureus, Enterococcus faecalis , Escherichia coli* and *Klebsiella pneumoniae* are common bacterial pathogens isolated from both male and female genital specimens and are associated with urinary tract infections (UTIs), urethritis and cervicitis [7, 8]. Multidrug-resistant (MDR) organisms such as Methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant *Enterococcus* (VRE), and extended-spectrum β-lactamase (ESBL)-producing Enterobacterales pose significant clinical challenges, especially in settings with limited resources and suboptimal antimicrobial stewardship [9,10]. Attention is also being drawn to the growing resistance of new diseases, such as Mycoplasma genitalium, to fluoroquinolones and macrolides, recent UK data show that the rates of fluoroquinolone and macrolide resistance among clinical isolates are 12% and 60%, respectively [11]. These trends demonstrate how important it is to maintain regional surveillance in order to guide empirical therapy and prevent the emergence of antimicrobial resistance (AMR).

The microbial ecology and resistance profiles of genital tract infections in Iraq, particularly in Baghdad, are not well documented in the literature. Thus, the current investigation utilized the VITEK 2 Compact technology to assess the antimicrobial susceptibility patterns of microbial pathogens isolated from genital tract specimens in Baghdad. This information will aid local antimicrobial stewardship efforts and guide appropriate empirical treatment.

Materials and Methods:

Methodology

Patients and sampling

For the current investigation, 168 samples were collected from individuals with genital tract infections at several private labs in Baghdad, Iraq, between May 2023 and December 2024. These samples (109 male and 59 female) were cultured for bacterial growth on blood agar and MacConkey agar media (incubated for 24 hours at 37°C).Identification of bacteria using VITEK 2compact system. The VITEK 2 compact system was used to identify the bacteria in accordance with the company's guidelines. The turbidity was measured using the visible spectrophotometer DensiChekTM Plus, which had been calibrated to the 0.5 MacFarland turbidity range. The bacterial suspension was put into the bioMérieux VITEK 2 system (France). The data were interpreted using the VITEK 2 compact system's unique bacterial species and strain identification software.

Determination of antibiotic susceptibility using VITEK® 2 compact

Susceptibility to the following antimicrobial agents (depending on the bacterial genus) was determined using VITEK 2 compact system: antibiotic included: levofloxacin, moxifloxacin, linezolid, vancomycin, clindamycin, tetracycline, nitrofurantoin, tigecycline, benzylpenicillin, oxacillin, teicoplanin, gentamicin, tobramycin, erythromycin, fusidic acid, ciprofloxacin, morifloxacin,

rifampicin, trimethoprim/sulfamethoxazole, ticarcillin, ticarcillin/clavulanic acid, piperacillin, piperacillin/tazobactam, ceftazidime, cefepime, imipenem, aztreonam, meropenem, amikacin, minocycline, ampicillin, cefazolin, cefoxitin, ceftriaxone, nalidixic acid, norfloxacin, and chloramphenicol.The break point for each antimicrobial used was determined according to CLSI (2024)
.

Results:

A total of 168 specimens from the male and female genital areas were included (seminal fluid, high vaginal tract, urethral discharge, and cervical swabs). Both genders (109 male samples and 59 female samples) ranged in age from 20 to 50 years old. Data was acquired from several private laboratories in Baghadad from May 2023 to December 2024.

Microbial growth was observed in 113/168 (67%), while 55/168 (32.7%) specimens showed no growth, as seen in Figure 1.

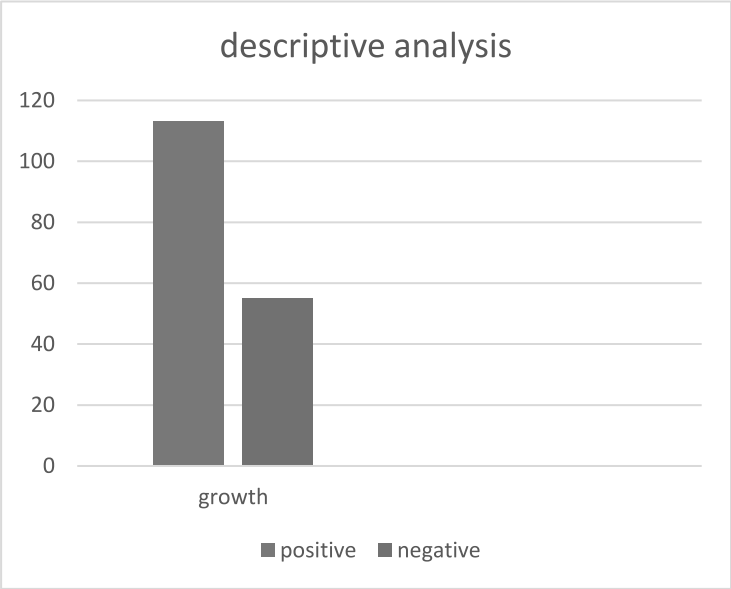


Figure 1: percentage of microbial growth

The current research demonstrates a significant negative growth tendency in female individuals, as quantitative measurements decline from an initial peak of 120 to 0 throughout the course of the observation period (Figure 2). Significant pathogenic pathways in the female reproductive ecology may be indicated by this slow decline. This phenomena can be explained by a number of mechanisms, the main one being that cyclical hormonal changes related to the menstrual cycle may cause regular fluctuations in microbial density and epithelial cell renewal. [12] Additionally, as is commonly observed in cases of bacterial vaginosis resolution, this route may indicate effective host immune responses to infection [13]. Finally, limitations and factors related to the sampling process or the results of therapeutic interventions may be the cause of the decline [14].

Although earlier studies have identified basic differences in reproductive habitats, a major barrier to meaningful sex comparisons is the lack of comparable data for males. [15] These results draw attention to critical information gaps regarding the temporal development of reproductive stability and confirm the link between poor gynecological outcomes and limited microbial diversity. [16] Future studies should employ consistent sampling procedures within longitudinal designs in order to distinguish between normal menstrual cycle deficits and illness states.

For comparison considerations, analogous male cohorts should also be included. Incorporating male participants and combining hormonal, metagenomic, and immune system data might lead to a more comprehensive knowledge of reproductive dynamics. These results highlight the importance of tracking development trends to enhance female reproductive care and indicate a clear need for advanced analytical techniques.

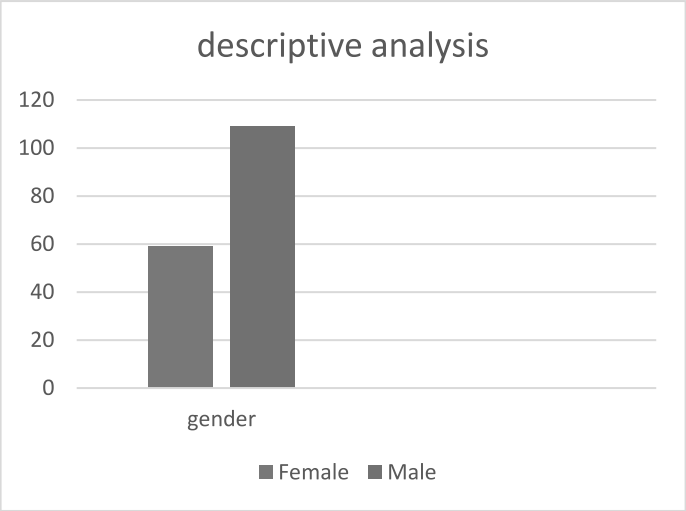


Figure 2: male to female percentage

Candida albicans was found only in females , where it was isolated in 24 out of 113 samples (21%). Normal or non-pathogenic bacteria, such as *Lactobacillus species, Kocuria species* and *Bacillus* species, were identified in 31 samples (27.4%), predominantly among female samples (Figure 3).

The presence of C. albicans within the female reproductive tract reflects its ability to exist both as a harmless commensals organism and as an opportunistic pathogens. Evidence indicates two distinct stages in its development: a non-pathogenic colonization phase and a rapid pathogenic proliferation phase. Under normal conditions, the fungus remains in equilibrium with the host's immune system and the indigenous microbiota, particularly Lactobacillus species. However, disruptions in microbial stability, hormonal imbalances, or immunocompromised states trigger a phenotypic shift toward pathogenicity. This transition is associated with biofilm synthesis, tissue invasion, and hyphal development which Clinically manifests as vaginal candidiasis with symptoms such as irritation, discharge, and inflammation. Several factors are known to exacerbate infection, including prolonged antibiotic use, elevated estrogen levels, and metabolic disorders like diabetes and immunosuppression[20].

These findings indicate the importance of differentiate between true fungal infection and normal microbial colonization during clinical evaluation. Such differentiation is essential, as it guides appropriate management strategies, ranging from targeted antifungal treatment for active infection to careful monitoring when colonization is a symptomatic.

Future studies should concentrate on determining the exact molecular cause of phenotypic changes, which enhances comprehension of their mechanics, developing quick diagnostic instruments that accurately identify infectious diseases, improving clinical judgment and developing medications that target virulence factors to improve treatment outcomes.

This study shows how the complex interactions between host factors and microbial activity influence the reproductive system's transition from benign colonization to accidental infection.

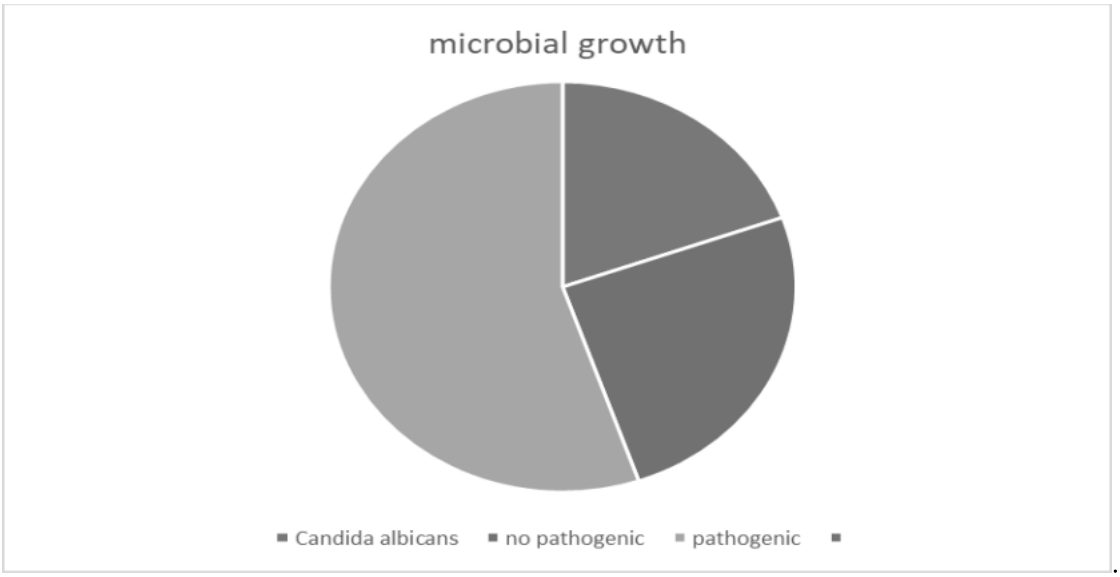


Figure3 : distribution of microbial growth of patients genital tract

Gram-negative enteric bacteria detected in multiple vaginal swabs were excluded from clinical interpretation based on WHO criteria, which classify these isolates as contaminants in genital samples unless accompanied by clinical infection (Figure 4).

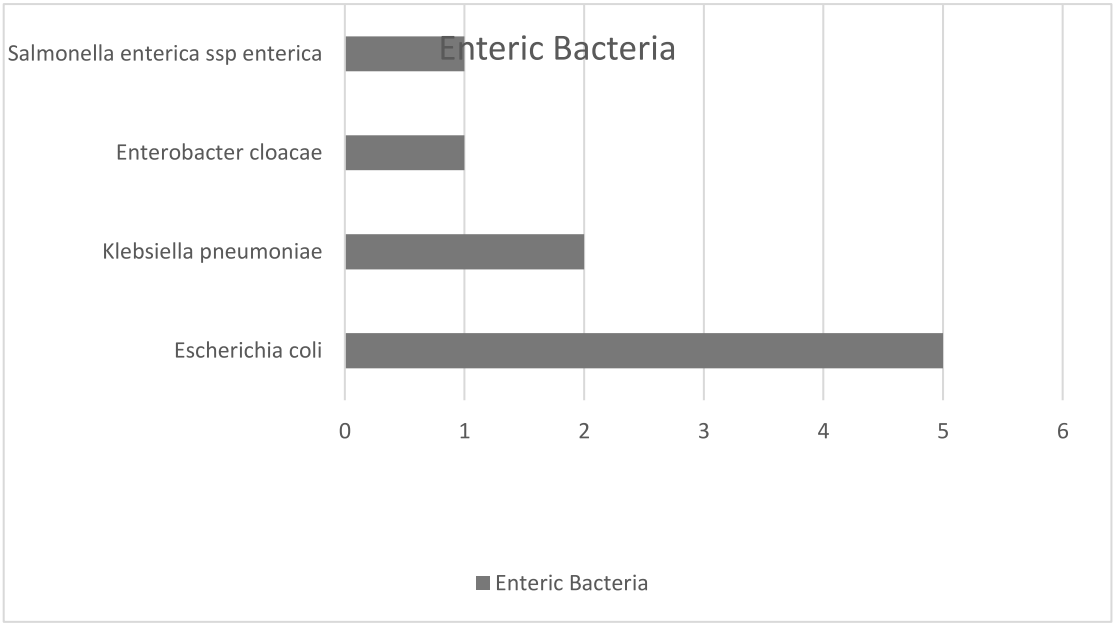
Study of enteric bacterial species reveals important epidemiological patterns for four clinically significant pathogens: *Salmonella enterica, Klebsiella pneumoniae, Enterobacter cloacae* and *Escherichia coli.* Their distribution in clinical or environmental samples can be estimated using a quantitative scale (0–6), which likely represents the number of colony-forming units or measures of relative abundance. Despite their varying clinical manifestations, *Escherichia coli* is the most common of these species due to its dual role as an opportunistic pathogen and as part of the commensal intestinal flora. Enterobacter cloacae and Klebsiella pneumoniae, which are resistant to AmpC-type antibiotics and broad-spectrum beta-lactamases (ESBLs), can cause hospital-acquired infections [21].

The prevalence of salmonella infections vary widely by area because of variances in food safety law and public health systems [22].

Salmonella infection rates vary greatly by area due to variations in public health infrastructure and food safety regulations [22]. These findings demonstrate the increasing threat presented by ESKAPE infections in both the community and hospitals, and they are consistent with existing antibiotic resistance surveillance data [23]. In order to tackle growing resistance rates, the data highlight the importance of comprehensive antimicrobial management systems and improved infection control measures, especially in hospitals. In order to stop the spread of Salmonella, the findings also call for more funding for food safety systems and the creation of quick diagnostic instruments for detecting these diseases in clinical samples to monitor developments [24].

In order to monitor changes in intestinal bacterial infections and guide public health initiatives to lessen their clinical effect, this study offers useful baseline data.

figure 4 : presence of enteric bacteria in high vaginal swab



The most commonly isolated pathogenic bacteria included *Staphylococcus aureus* (n=7), *Staphylococcus epidermidis* (n=7), *Staphylococcus haemolyticus* (n=9), *Staphylococcus hominis* (n=1), *Staphylococcus warneri* (n=1), *Streptococcus agalactiae* (n=7), *Enterococcus faecalis* (n=17), *Streptococcus mitis/oralis/mutans* (n=2), *Escherichia coli* (n=5), *Klebsiella pneumoniae (n=5),* and *Pseudomonas aeruginosa* (n=1). The susceptibility patterns of these isolates to antibiotics are detailed in Tables 1, 2, 3, and 4.

Antibiotic susceptibility analysis of Group B Streptococcus isolates in this study revealed that all seven isolates were equally susceptible to linezolid, tetracycline , vancomycin, , and nitrofurantoin. This is consistent with previous research showing that these antibiotics are particularly effective against Group B Streptococcus [25, 26]. However, isolates 75 and 80 exhibited resistance to fluoroquinolones, particularly moxifloxacin and levofloxacin. This is consistent with previous research that found fluoroquinolone resistance in Group B Streptococcus isolates to range from 10% to 30%, depending on exposure history and geographic location [27, 28]. Clindamycin resistance was remarkably high in our group (6 out of 7 isolates), which is consistent with reports of increasing resistance to macrolides and lincosamides in group B streptococci, especially in places where antibiotics are used intensively [29]. Due to extensive historical usage and the presence of tet(M) resistance genes, tetracycline resistance was also prevalent, occurring in over half of the isolates. This is in accordance with the global trend of tetracycline resistance in group B streptococci [30]. These findings emphasize the need for customized treatment, which involves susceptibility testing and local monitoring, especially resistance to common medications such fluoroquinolones and clindamycin.

Table 1: Antibiotic susceptibility for Streptococcus agalactiae

Code of bacteria / Antibiotic	**Streptococcus agalactiae**						
	5	**28**	**53**	**63**	**75**	**78**	**80**
Levofloxacin	**S**	**S**	**S**	**S**	**R**	**S**	**R**
Moxifloxacin	**S**	**R**	**S**	**S**	**R**	**S**	**R**
Linezolid	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Vancomycin,	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Clindamycin	**R**	**R**	**S**	**R**	**R**	**R**	**R**
Tetracycline	**R**	**S**	**S**	**R**	**S**	**R**	**R**
Nitrofurantoin	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Tigecycline	**S**	**S**	**S**	**S**	**S**	**S**	**S**

Antibiotic susceptibility of Staphylococcus aureus and Staphylococcus epidermidis in our study reflects treatment choices and the ongoing issue of resistance.

In line with other studies demonstrating the long-term efficacy of these medications against methicillin-resistant and methicillin-sensitive staphylococci, all isolates were consistently susceptible to ciprofloxacin, tigecycline, teicoplanin, vancomycin, linezolid, and nitrofurantoin [31, 32]. Six out of seven isolates showed comprehensive resistance to benzylpenicillin and extensive resistance to oxacillin.This highlights the prevalence of beta-lactam resistance, particularly in methicillin-resistant strains, as observed globally [33]. Inducible MLS_B resistance genes (erm), often found in staphylococci, may be responsible for clindamycin and erythromycin resistance [34]. S. epidermidis isolates showed higher gentamicin and tobramycin resistance, which is interesting because hospital isolates associated to implanted devices show the same pattern [35]. With the exception of Staphylococcus epidermidis isolate 60, which demonstrated resistance, the majority of isolates remained sensitive to trimethoprim/sulfamethoxazole. Our findings underline the importance of rigorous susceptibility testing for staphylococcal infections due to significant variance in resistance patterns, even across closely related species. They also underline the need of carefully managing and monitoring last-resort drugs to ensure their effectiveness.

Table 2: Antibiotic susceptibility for Staphylococcus aureus and Staphylococcus epidermidis

Code of bacteria / Antibiotic	***Staphylococcus aureus***			***Staphylococcus epidermidis***			
	16	**20**	**84**	**29**	**35**	**60**	**69**
Linezolid	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Vancomycin,	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Benzylpenicillin	**R**	**R**	**R**	**R**	**R**	**R**	**R**

Oxacillin	**R**	**R**	**S**	**R**	**R**	**R**	**R**
Teicoplanin	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Gentamicin	**S**	**R**	**S**	**R**	**S**	**R**	**S**
Tobramycin	**R**	**R**	**S**	**R**	**S**	**R**	**S**
Tetracycline	**R**	**R**	**S**	**R**	**S**	**R**	**R**
Tigecycline	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Clindamycin	**R**	**R**	**S**	**R**	**S**	**S**	**S**
Erythromycin	**R**	**R**	**R**	**R**	**R**	**S**	**R**
Fusidic acid	**R**	**R**	**S**	**R**	**R**	**R**	**R**
Ciprofloxacin	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Morifloxacin	**I**	**I**	**S**	**I**	**S**	**R**	**S**
Rifampicin	**R**	**R**	**S**	**R**	**S**	**R**	**S**
Nitrofurantoin	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Trimethoprim/ Sulfamethoxazoe	**S**	**S**	**S**	**S**	**S**	**R**	**S**

All nine isolates showed consistent susceptibility to vancomycin , linezolid, , teicoplanin, tigecycline, nitrofurantoin and rifampicin, consistent with previous findings demonstrating the efficacy of these treatments against multidrug-resistant Staphylococcus aureus (MRSA) strains [32, 36]. This species has broad mecA gene-mediated resistance, as evidenced by the majority of isolates tested positive for oxacillin and benzylpenicillin [37].

Aminoglycoside susceptibility varied, with several isolates exhibiting resistance to Enzymes may change gentamicin and tobramycin resistance [38]. High rates of resistance to erythromycin, tetracycline, clindamycin, and fusidic acid were detected in the research, suggesting a global trend of MRSA resistance associated with long-term antibiotic exposure in hospital settings [39].

Interestingly, resistance to fluoroquinolones (levofloxacin, morifloxacin , ciprofloxacin) was also observed, which may be due to alterations in the gyrA and parC genes, as previously reported [40]. Given that hemolytic staphylococci remain a significant challenge in hospital settings due to their high resistance and ability to survive on medical equipment and skin, these findings underscore the need for continuous monitoring and the judicious use of antibiotics.

Table 3: Antibiotic susceptibility for Staphylococcus haemolyticus

Code of bacteria / Antibiotic	**Staphylococcus haemolyticus**								
	17	**19**	**39**	**45**	**61**	**65**	**66**	**76**	**85**
Linezolid	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Vancomycin,	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Benzylpenicillin	**R**	**R**	**R**	**R**	**R**	**R**	**R**	**S**	**R**
Oxacillin	**R**	**R**	**R**	**R**	**R**	**R**	**R**	**S**	**R**
Teicoplanin	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Gentamicin	**S**	**R**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Tobramycin	**S**	**R**	**R**	**S**	**R**	**S**	**R**	**S**	**S**
Tetracycline	**R**	**R**	**R**	**R**	**R**	**S**	**R**	**S**	**R**
Tigecycline	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Clindamycin	**R**	**R**	**S**	**R**	**R**	**R**	**S**	**S**	**R**
Erythromycin	**R**	**R**	**R**	**R**	**R**	**R**	**R**	**S**	**R**
Fusidic acid	**R**	**R**	**R**	**R**	**R**	**R**	**R**	**S**	**R**
Ciprofloxacin	**R**	**S**	**R**	**S**	**S**	**S**	**S**	**I**	**S**
Morifloxacin	**R**	**R**	**I**	**I**	**I**	**S**	**I**	**S**	**I**
Levofloxacin	**R**	**R**	**R**	**R**	**R**	**S**	**R**	**S**	**R**
Rifampicin	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Nitrofurantoin	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**	**S**
Trimethoprim/ Sulfamethoxazoe	**S**	**R**	**S**	**S**	**R**	**S**	**R**	**R**	**R**

Consistent susceptibility patterns of Staphylococcus hominis and Staphylococcus warneri to several important antibiotics, such as vancomycin, linezolid, teicoplanin, rifampicin, tigecycline, , nitrofurantoin, and trimethoprim/sulfamethoxazole, demonstrate the continued efficacy of these antibiotics against these coagulase-negative staphylococci (CoNS). Both Staphylococcus hominis and Staphylococcus warneri isolates have shown resistance to beta-lactam antibiotics, including oxacillin and benzylpenicillin, consistent with previous findings indicating the spread of methicillin resistance mediated by the mecA gene.[32, 41] Resistance to macrolides and lincosamides has also been observed, as evidenced by resistance to clindamycin and erythromycin, which is consistent with previous studies that demonstrated the presence of erm genes in clinical coagulase-negative staphylococcal isolates

[42].Tetracycline and fusidic acid, which are known to have differing effectiveness against CoNS depending on local antibiotic pressures and resistance gene frequency, were also resistant to both isolates [39]. Crucially, S. warneri's sensitivity to fluoroquinolones (ciprofloxacin, morifloxacin, and levofloxacin) was partially reduced (intermediate responses). This might be because to efflux mechanisms, as previously noted, or chromosomal abnormalities in gyrA or parC [40]. Overall, these isolates' resistance to first-line and commonly used medications emphasizes the significance of continuous phenotypic surveillance, particularly in nosocomial settings where CoNS are common opportunistic infections, even though they are still responsive to a number of effective antibiotics.

Table 4: Antibiotic susceptibility for Staphylococcus hominis and Staphylococcus warneri

Code of bacteria / Antibiotic	***Staphylococcus* hominis**	***Staphylococcus* warneri**
	55	**72**
Linezolid	**S**	**S**
Vancomycin,	**S**	**S**
Benzylpenicillin	**R**	**R**
Oxacillin	**R**	**R**
Teicoplanin	**S**	**S**
Gentamicin	**S**	**S**
Tobramycin	**S**	**S**
Tetracycline	**R**	**R**
Tigecycline	**S**	**S**
Clindamycin	**R**	**R**
Erythromycin	**R**	**R**
Fusidic acid	**R**	**R**
Ciprofloxacin	**S**	**S**
Morifloxacin	**S**	**I**
Levofloxacin	**S**	**I**
Rifampicin	**S**	**S**
Nitrofurantoin	**S**	**S**
Trimethoprim/ Sulfamethoxazoe	**S**	**S**

The Enterococcus faecalis isolates from our investigation shown universal resistance to tigecycline, linezolid, vancomycin, and teicoplanin, which is consistent with prior studies that support these medications as successful therapies for enterococcal infections [43, 44]. However, all isolates had strong erythromycin resistance, indicating the widespread distribution of macrolide resistance factors, such as the erm genes seen in clinical isolates of Enterococcus faecalis [45]. The susceptibility to fluoroquinolones varied significantly, with some isolates exhibiting resistant or intermediate traits. This is similar to a previous study that found a link between reduced fluoroquinolone efficacy and mutations in the gyrA and parC genes [46].

In keeping with global patterns associated with the extensive use of tetracyclines in both clinical and agricultural settings, tetracycline resistance was also common, most likely as a result of the existence of tet(M) and tet(L) genes [47]. Nitrofurantoin resistance was reduced, although it was still present in some isolates, suggesting that it should be used cautiously for *E. faecalis* caused UTIs [48]. In order to direct efficient treatment plans and prevent the spread of resistant enterococcal strains, our results emphasize the necessity of continuous local antibiotic susceptibility monitoring.

Table 5: Antibiotic susceptibility for *Enterococcus faecalis*

Code of bacteria / Antibiotic	*Enterococcus faecalis*															
	2	9	15	22	23	25	30	31	32	33	64	71	74	77	78	79
Levofloxacin	S	S	S	S	I	S	S	S	S	S	R	S	S	I	S	I
Erythromycin	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Linezolid	S	S	R	S	S	S	S	S	R	R	S	S	S	R	R	S
Vancomycin	S	S	R	S	S	S	S	S	R	R	R	S	S	R	R	S
Teicoplanin	S	S	R	S	S	S	S	S	R	R	S	S	S	R	R	S
Tetracycline	R	S	S	R	S	R	S	S	S	S	R	R	S	R	R	R
Nitrofurantoin	S	R	I	S	R	S	S	S	S	S	S	S	S	R	S	I
Tigecycline	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S

The study's Escherichia coli isolates were resistant to ticarcillin and several cephalosporins, particularly ceftazidime and aztreonam. This is consistent with the increasing number of E. coli bacteria producing extended-spectrum beta-lactamases (ESBLs) that have been reported worldwide [49]. All isolates, however, were susceptible to carbapenems (imipenem and meropenem), which is consistent with the fact that carbapenems are the most effective treatment for severe infections caused by E. coli ESBLs [50]. The high responsiveness to beta-lactam/beta-lactamase inhibitor combos, such as piperacillin/tazobactam and ticarcillin/clavulanic acid, demonstrates their efficacy in overcoming particular beta-lactamase-mediated resistance[51]While gentamicin showed considerable resistance, a pattern frequently observed in clinical isolates, aminoglycoside susceptibility was mostly unaltered, especially to amikacin [52]. In line with the established worldwide pattern of a slow rise in fluoroquinolone-resistant E. coli as a result of selection pressure from broad antibiotic usage, fluoroquinolone resistance was minimal but evident in one isolate [53]. All things considered, our findings show that carbapenem-beta-lactamase inhibitor combinations continue to be useful in treating multidrug-resistant E. coli infections and that careful susceptibility monitoring is required.

Table 6: Antibiotic susceptibility for Escherichia coli

Code of bacteria / Antibiotic	**Escherichia coli**				
	44	59	67	83	88
Ticarcillin	**R**	**R**	**R**	**R**	**R**
Ticarcillin/ Clavulanic	**S**	**S**	**S**	**S**	**S**
Piperacillin	**R**	**R**	**R**	**I**	**R**
Piperacillin/Tazobactam	**S**	**S**	**S**	**S**	**S**
Ceftazidime	**R**	**R**	**R**	**S**	**R**
Cefepime	**S**	**R**	**R**	**S**	**S**
Imipenem	**S**	**S**	**S**	**S**	**S**
Azteronam	**R**	**R**	**R**	**S**	**R**
Meropenem	**S**	**S**	**S**	**S**	**S**
Gentamycin	**R**	**S**	**S**	**S**	**S**
Amikacin	**S**	**S**	**S**	**S**	**S**
Tobramycin	**I**	**S**	**S**	**S**	**S**
Minocycline	**S**	**S**	**S**	**S**	**S**
Ciprofloxacin	**S**	**S**	**I**	**S**	**S**
Trimethoprim/Sulfamethoxazole	**S**	**S**	**S**	**S**	**S**

The isolates from this study demonstrated universal resistance to ampicillin to trimethoprim/sulfamethoxazole because *Klebsiella pneumoniae* normally develops chromosomal beta-lactamases [22]. In accordance with previous findings, one isolate exhibited resistance to piperacillin/tazobactam, while others remained susceptible, indicating that the beta-lactam/beta-lactamase inhibitor combination's efficacy was largely preserved [54]. The presence or absence of extended-spectrum beta-lactamases (ESBLs) or AmpC enzymes is likely the cause of the three out of five isolates' intermediate or complete susceptibility to third- and fourth-generation cephalosporins (ceftazidime, ceftriaxone, and cefepime) [49]. Notably, carbapenem resistance was found in one isolate, highlighting the identification of *K. pneumoniae* strains that produce carbapenemase, which present a significant treatment challenge globally [55].

The majority of isolates remained susceptible to aminoglycosides, such as amikacin and gentamicin, indicating their potential utility as alternative therapy for illnesses resistant to multiple medicines. [56] Two isolates were resistant to fluoroquinolone, which is consistent with recent global trends in fluoroquinolone resistance caused by chromosomal and plasmid mechanisms.[57]

Trimethoprim/sulfamethoxazole resistance was found in 60% of the isolates, consistent with the widespread resistance observed in clinical Klebsiella pneumoniae isolates.[58] This pattern of drug sensitivity emphasizes the importance of continuous monitoring and judicious antibiotic delivery, particularly when carbapenem and beta-lactamase inhibitor combinations are used to treat Klebsiella pneumoniae infections.

Table 7: Antibiotic susceptibility for *Klebsiella pneumoniae*

Code of bacteria / Antibiotic	Klebsiella pneumoniae				
	14	24	27	86	90
Ampicillin	**R**	**R**	**R**	**R**	**R**
Piperacillin/Tazobactam	**R**	**S**	**S**	**S**	**S**
Ceftazidime	**R**	**I**	**I**	**S**	**R**
Cefazolin	**R**	**R**	**R**	**S**	**R**
Cefoxitin	**R**	**S**	**R**	**S**	**R**
Ceftriaxone	**R**	**R**	**S**	**S**	**R**
Cefepime	**R**	**S**	**S**	**S**	**R**
Imipenem	**R**	**S**	**S**	**S**	**S**
Meropenem	**R**	**S**	**S**	**S**	**S**
Gentamycin	**R**	**R**	**R**	**S**	**S**
Amikacin	**R**	**S**	**S**	**S**	**S**
Ciprofloxacin	**R**	**R**	**I**	**S**	**S**
Nitrofurantoin	**R**	**R**	**I**	**S**	**S**
Levofloxacin	**R**	**I**	**I**	**S**	**S**
Trimethoprim/Sulfamethoxazole	**R**	**R**	**R**	**S**	**S**

Pseudomonas aeruginosa isolate 87 in this study showed resistance only to nalidixic acid, ticarcillin , ticarcillin/clavulanic acid, piperacillin, piperacillin/tazobactam, and tigecycline, indicating multidrug sensitivity. The organism remained susceptible to aminoglycosides (amikacin , gentamicin and tobramycin), carbapenems (imipenem and meropenem), fluoroquinolones (ciprofloxacin, levofloxacin, norfloxacin,), ceftazidime, cefepime, and minocycline. Pseudomonas aeruginosa strains lacking carbapenemase can be successfully treated with cefepime and cefazidime, according to earlier research [59, 60].

As previously shown [61], chromosomal AmpC gene overexpression or outflow pump activity may be

the cause of Pseudomonas aeruginosa's resistance to piperacillin and tazobactam. Despite resistance shown in earlier testing, carbapenems, particularly imipenem and meropenem, remain effective against susceptible Pseudomonas aeruginosa, highlighting the need for continuous surveillance [62]. Despite increasing resistance brought on both gyrA and parC mutations as well as the MexAB-OprM outflow system, fluoroquinolones were effective against this strain [63].

The sensitivity of the isolate to aminoglycosides is consistent with its repeated activity against *Pseudomonas aeruginosa*, which is attributed to weak resistance mediated by enzymes in some cases [64].Overall, while this isolate appears to be broadly susceptible, its resistance to key beta-lactams emphasizes the need for ongoing local antimicrobial surveillance and susceptibility-guided treatment regimens.

Table 8: Antibiotic susceptibility for *Pseudomonas aeruginosa*

Code of bacteria / Antibiotic	**Pseudomonas aeruginosa**
	87
Ticarcillin	**R**
Ticarcillin/ Clavulanic	**R**
Piperacillin	**R**
Piperacillin/Tazobactam	**R**
Ceftazidime	**S**
Cefepime	**S**
Imipenem	**S**
Nalidixic Acid	**R**
Meropenem	**S**
Gentamycin	**S**
Amikacin	**S**
Tobramycin	**S**
Minocycline	**S**
Norfloxacin	**S**
Tigecycline	**R**
Ciprofloxacin	**S**
Levofloxacin	**S**

There are significant differences between the two tested isolates (codes 40 and 58) in the antibiotic susceptibility profile of Streptococcus mitis/Streptococcus oralis/mutans in this study.
Consistent with previous findings demonstrating the sustained efficacy of linezolid, chloramphenicol , vancomycin, and tigecycline against viridans group streptococci (VGS), both isolates consistently responded to these drugs [65, 66]. Tetracycline resistance was found in both isolates, which is consistent with other research showing widespread resistance in Streptococcus oralis, typically associated with the acquisition of the tet(M) and tet(O) resistance genes [67]. Due to mutations in the gyrA and parC genes, isolate 58 exhibited resistance to fluoroquinolones (levofloxacin and moxifloxacin), a disease that is increasingly being characterized [68]. It should be noted that the susceptibility of the isolates to clindamycin varied, suggesting that alternative mechanisms of resistance to macrolide-lincosamide-streptogramin B (MLSB) may emerge, possibly as a result of carrying a congenital or induced erm(B) gene [69].Even closely related members of the VGS group may exhibit distinct resistance patterns, emphasizing the importance of species-specific identification and individual susceptibility testing. The results also demonstrate the need for regional surveillance to guide empirical therapy for invasive infections caused by these opportunistic commensal organisms in the mouth.

Table 9: Antibiotic susceptibility for *Streptococcus mitis/Streptococcus oralis/mutans*

Code of bacteria / Antibiotic	**Streptococcus mitis/Streptococcus oralis/mutans**	
	40	**58**
Levofloxacin	**S**	**R**
Moxifloxacin	**S**	**R**
Linezolid	**S**	**S**
Vancomycin,	**S**	**S**
Clindamycin	**R**	**S**
Tetracycline	**R**	**R**
Chloramphenicol	**S**	**S**
Tigecycline	**S**	**S**

Conclusion:

This study provides an important review of the antibiotic resistance profile and microbiological causes of genital tract infections in patients at a Baghdad hospital. The predominance of Gram-positive cocci, the isolation of Candida albicans in females, and the detection of multidrug-resistant strains such as methicillin-resistant Staphylococcus aureus (MRSA) and vancomycin-resistant Enterococcus (VRE) all underscore the need for targeted diagnostic and therapeutic techniques. The discovery of resistance patterns, particularly for widely used antibiotics, shows how urgently comprehensive antibiotic control techniques are needed. Regular monitoring and adjustments to local antibiotic regimens are necessary to guide empirical treatment.
To minimize treatment failure and get a better understanding of pathogen behavior and resistance dynamics, future research should combine clinical data with resistance gene characterization.

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