A STUDY ON ANTIBIOTIC RESISTANCE PATTERN AMONG PATIENTS OF A SELECTED HOSPITAL IN NARAYANGANJ, BANGLADESH

By
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A thesis submitted to the Department of School of Pharmacy in partial fulfillment of the requirements for the degree of Bachelor of Pharmacy (Hons)

School of Pharmacy
Brac University
April,2024

Declaration

It is hereby declared that,

1. The thesis submitted is my own original work while completing a degree at Brac University.
2. The thesis does not contain material previously published or written by a third party, except where this is appropriately cited through full and accurate referencing.
3. The thesis does not contain material which has been accepted, or submitted, for any other degree or diploma at a university or other institution.
4. I have acknowledged all main sources of help.

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Approval

The thesis titled “A Study of Antibiotic Resistance Pattern Among Patients of a Selected Hospital in Narayanganj, Bangladesh” submitted by Shahin Khan (19346071), of Spring, 2020 has been accepted as satisfactory in partial fulfillment of the requirement for the degree of Bachelor of Pharmacy on 09/05/2024.

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Ethics Statement

This study does not involve any animal and human trial.

Abstract

The emergence and spread of multi-drug-resistant (MDR) pathogens pose a significant threat to global public health, challenging the efficacy of antimicrobial treatments and leading to increased morbidity, mortality, and healthcare costs. This study aimed was to investigate the prevalence of MDR pathogens and their antimicrobial sensitivity patterns in clinical from a selected hospital in Narayanganj, Bangladesh. This retrospective study was conducted using clinical records from Pro-Active Medical College & Hospital Limited Microbiology Laboratory between January, 2021 to September, 2023. Out of a total of 2340 samples, only 650 showed the presence of pathogens. These isolates were isolated from various specimens, like urine, stool, blood, and other biological body fluids. Then the data was analyzed according to including age, gender, and antibiotic susceptibility. Patients were divided into three groups based on their age: 0–20, 21–60, and 61–100+. The resistance pattern and distribution of these pathogens according to gender and age were prepared. The MAR Index and MDR percentages were determined. Among the 650 isolates, *E. coli* (64%) was found to be the most common, followed by *S. typhi* (18%), *S. aureus* (8%), *Klebsiella spp*. (7%), and others. The results reveal a concerning trend of rising MDR pathogen prevalence, particularly among Gramme-negative bacteria such as *Escherichia coli, Klebsiella spp.*, and *Salmonella typhi*. Additionally, the study highlights variations in antimicrobial resistance patterns according to gender and age based on local epidemiological data. The study revealed that 433 (66.6%) of the positive infections were recovered from females. According to the data, the age group of 1–10 years old had the highest percentage of infected patients (25.1%), followed by the age group of 21–30 years old (18.6%). Additionally, the data revealed that male patients had a higher susceptibility to *S. typhi* (28.6%), while female patients had a higher susceptibility to *E. coli* (73.4%). Furthermore, the analysis underscores the importance of antimicrobial stewardship

programs and infection control measures in mitigating the spread of MDR pathogens. Overall, this research provides valuable insights into the current landscape of antimicrobial resistance, which will help to form strategies for effective management and containment of MDR infections.

Keywords: Multi Drug Resistance, MAR index, *E. coli, S. Aureus, S. Typhi, Klebsiella spp.*

Dedication

To my parents, I would like to dedicate my thesis. I am here today because of their immense sacrifice.

Thank you.

Acknowledgement

Being a student at the School of Pharmacy at the Brac University in Bangladesh has been a tremendous opportunity for me. I'd want to express my gratitude to the Almighty for aiding me at every level of my life on the occasion of the submission of my project. I'd also like to thank my family for all of their support and sacrifices throughout my life.

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May the Almighty bless all of those who helped me in this endeavor.

Thank you very much.

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List of Acronyms

MDR	**M**ulti-**D**rug **R**esistance
MAR	**M**ultiple **A**ntibiotic **R**esistance
AST	**A**ntimicrobial **S**usceptibility **T**est
ASM	**A**merican **S**ociety for **M**icrobiology
WHO	**W**orld **H**ealth **O**rganization
ABC	**A**TP-**B**inding **C**assette
SMR	**S**mall **M**ultidrug **R**esistance
MFS	**M**ajor **F**acilitator **S**uperfamily
RND	**R**esistance-**N**odulation-**D**ivision
MATE	**M**ultidrug **A**nd **T**oxic **C**ompound **E**xtrusion
GLASS	**G**lobal **A**ntimicrobial **R**esistance and Use **S**urveillance **S**ystem
AMR	**A**nti-**M**icrobial **R**esistance

Chapter 1: Introduction

1.1 Introduction

The word "antibiotics" describes compounds that exist naturally in different microorganisms, such fungi or bacteria, and can prevent the growth of other microorganisms and kill their cells. Prior to the discovery of antibiotics, people were largely susceptible to infection. Conditions including TB, meningitis, and pneumonia were difficult to cure or not treated at all. As a result, humanity always lives under the fear of major pandemics. In 1928, Sir Alexander Fleming, who was a Scottish biologist and pharmacologist, discovered the first antibiotic, penicillin. Antibiotics have significantly improved the worldwide treatment of infectious diseases since their discovery(Kourkouta L & Plati P, 2018). Due to the drop in the death rate from infectious diseases, they are acknowledged as one of the contributing elements to an increased longer lifespan in the 20th century (Adedeji, 2016). However, their abuse and misuse in human therapy have led to the global antibiotic resistance epidemic.

1.2 Major Types of Antibiotics

The major class of antibiotics that are used in clinical practice are outlined in table 1.1 as follows:

Table 1.1: *Antibiotic Class, Names, Spectrum, Mechanism of Action and Uses*

Class of Antibiotics	**Antibiotics names**	**Gram Spectrum**	**Mechanism of Action**	**Uses**
Aminoglycosides	Streptomycin, Gentamycin	Gram(-)	Inhibit Protein Synthesis (30s)	Bacteremia,Abdominal Infections
Cephalosporins	Ceftriaxone, Cefepime	Gram(+)/ Gram(-)	Inhibit Cell Wall Synthesis	Skin,Urinary & Respiratory Infections
Tetracyclines	Tetrecycline, Doxycycline	Gram(+)/ Gram(-)	Inhibit Protein Synthesis (30s)	Lyme Diseases,PID, STIs
Penicillins	Ampicillin, Amoxicillin	Gram(+)/ Gram(-)	Inhibit Cell Wall Synthesis	ENT,Skin,Urinary Infections
Sulfonamides	Sulfasalazine, Sulfamethoxazole	Gram(+)/ Gram(-)	Inhibit Folate Synthesis	UTIs,Burns,Eye Infections

Table 1.1: *contd...*

Class of Antibiotics	Antibiotics names	Gram Spectrum	Mechanism of Action	Uses
Fluoroquinolones	Ciprofloxacin, Levofloxacin	Gram(+)/ Gram(-)	Inhibit DNA Replication	Respiratory & Urinary Indections
Macrolides	Azithromycin, Erythromycin	Gram(+)	Inhibit Protein Synthesis (50s)	Pneumonia,Sinus, ENT,STIs
Carbapenems	Meropenem, Ertapenem	Gram (+)/ Gram(-)	Inhibit Cell Wall Synthesis	Urinary & Abdomen Infections
Lincosamides	Clindamycin	Gram (+)	Inhibit Protein Synthesis (50s)	Skin, Bone,Lung Infections
Glycopeptides	Vancomycin	Gram (+)	Inhibit Cell Wall Synthesis	MRSA,Skin, Endocarditis

1.3 Mechanisms of action of Antibiotics

Antibiotics typically work against microorganisms by either competing with the substrate of any enzyme involved in cell wall synthesis or by blocking a metabolic pathway such as nucleotide synthesis, which in turn inhibits DNA/RNA synthesis, further protein synthesis, and disruption of the cell membrane (Tanwar et al., 2014). This section will mainly describe the mechanism of action of antibiotics (Figure 1.1).

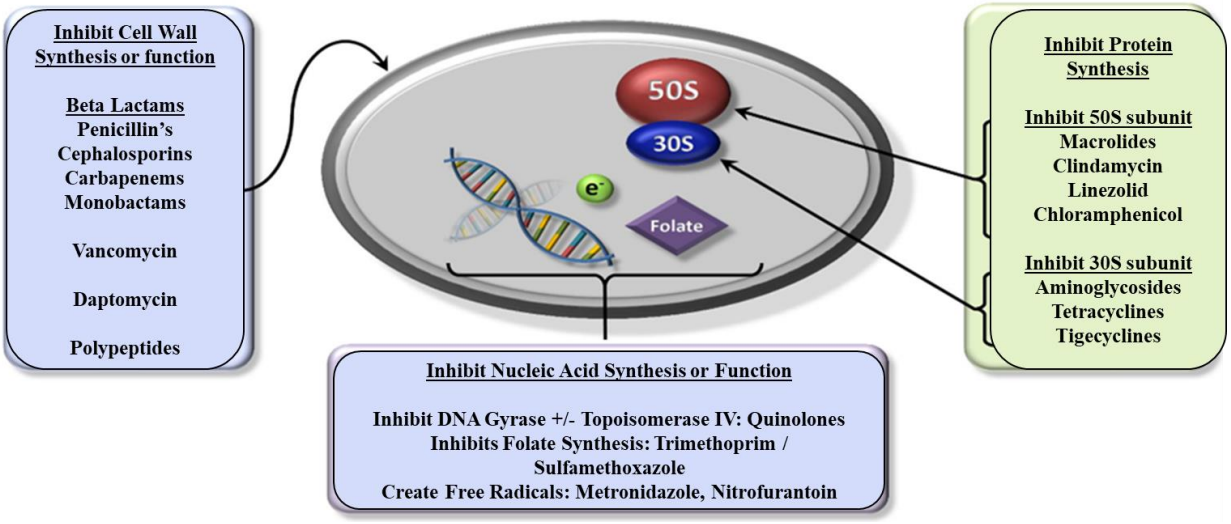

Figure 1.1: Mechanisms of action of Antibiotics (Gupta et al., 2020).

1.4 Antibiotic Resistance

Antibiotic resistance occurs when bacteria, fungi, and other microorganisms adapt to withstand the effects of antibiotics meant for eliminating them. This indicates that the pathogens have not been eliminated and may proliferate. As shown in figure 1.2, there are four primary types of antimicrobial resistance mechanisms: (1) drug uptake limitation; (2) drug target modification; (3) drug inactivation; and (4) active drug efflux (Reygaert, 2018).

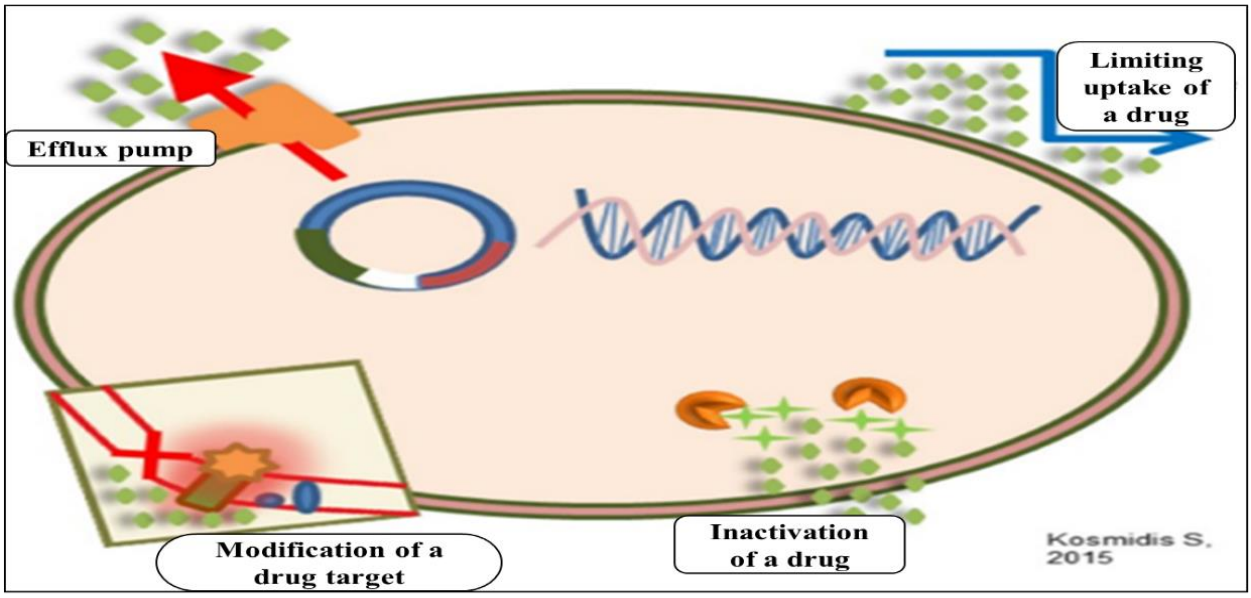

Figure 1.2: General antimicrobial resistance mechanisms(Reygaert, 2018).

1.5 Acquired Resistance

Bacteria can potentially develop resistance by altering in a way that protects them from antibiotics. Bacteria can acquire resistance in two ways: by undergoing a new genetic change that allows the bacteria to survive or by acquiring DNA from an existing resistant bacterium.

1.5.1 Genetic Change

DNA contains instructions for protein production, hence a change in DNA can result in a change in protein. This DNA alteration can sometimes influence the structure of the protein. If this occurs at the site on the protein where an antibiotic functions, the antibiotic may lose its ability to detect where it needed to do its function. Such modifications have the potential to stop an antibiotic from entering the cell or from functioning once it is inside. Once a change occurs, it has the potential to propagate across a bacteria species via mechanisms such as reproduction or DNA transfer.

1.5.2 DNA Transfer

Bacteria are highly adept at exchanging genes, including those that cause antibiotic resistance. They may share resistance genes that have previously existed in the community, as well as new genetic modifications that arise. Bacteria can transmit DNA via bacteriophages. After death, the bacteria release these DNA packages, which may contain genes resistant to antibiotics, which can be assimilated and utilized by other bacteria.

1.6 Multi-Drug Resistance

Multidrug resistance (MDR) is described as a microorganism's insensitivity or resistance to antibiotics that are structurally unrelated and have distinct molecular targets, despite previous susceptibility to them. The World Health Organization (WHO) claims that these resistant bacteria, fungi, viruses, and parasites can fend off the effectiveness of antibiotics, resulting in ineffective treatment that allows the infection to persist and spread (Tanwar et al., 2014). MDR, as suggested by Tanwar et al., can be any types as depicted in figure 1.3 below:

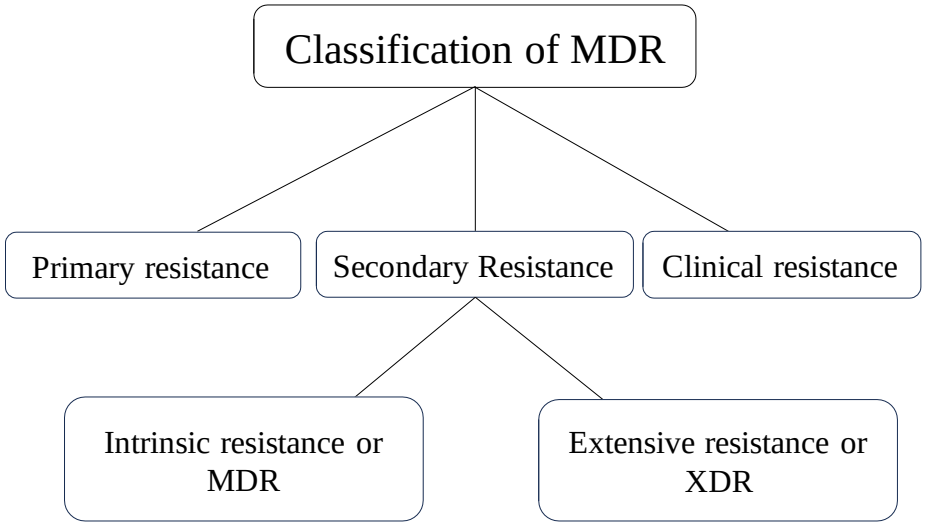

Figure 1.3: Classification of MDR (Tanwar et al., 2014)

1.6.1 Mechanism of MDR

Resistance refers to a microbe's insensitivity to antibiotic drugs as compared to other isolates of the same species. Despite the introduction of various new antibiotics into the market, the development of resistance among pathogenic bacteria is rising, particularly in patients who have been exposed to antibiotics for an extended period. This section will focus on the resistance mechanisms that microorganisms evolve to prevent being killed by medicines(Figure 1.4) (Tanwar et al., 2014).

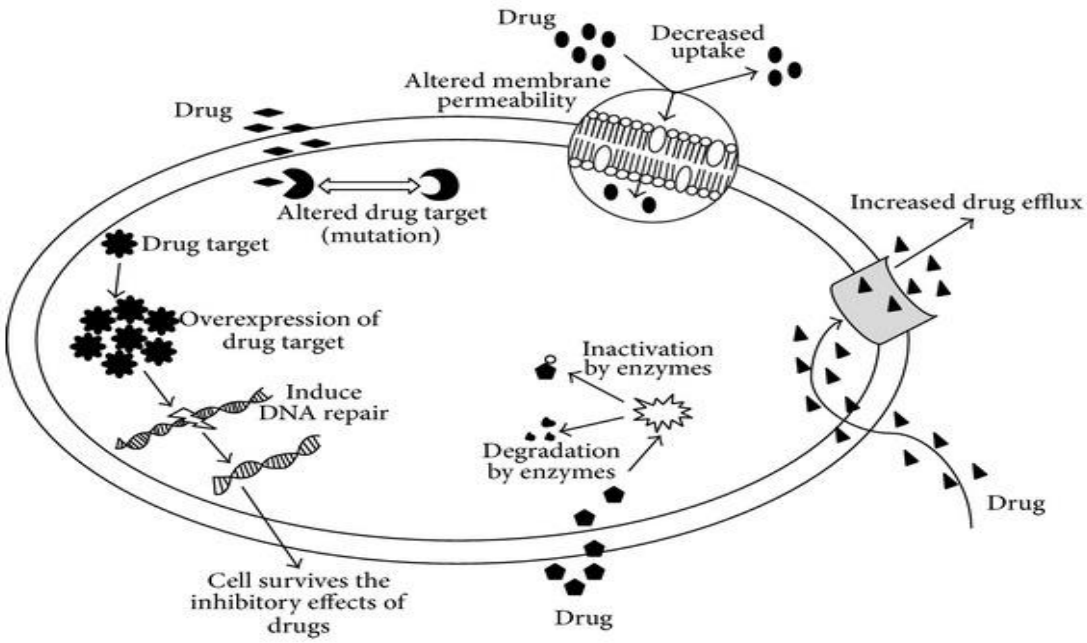

Figure 1.4: Mechanism of MDR (Tanwar et al., 2014)

1.7 Resistance Mediated by Efflux Pump

Drug efflux is an important mechanism of resistance in Gram-negative bacteria. These mechanisms circulate solutes outside of the cell. To promote antibiotic removal from the intercellular compartment (or the intermembrane space in Gram-negative bacteria), certain bacteria consist of membrane proteins that act as antimicrobial agent exporters, known as efflux pumps. Efflux pumps help bacteria maintain their internal environment by eliminating toxic compounds like antimicrobial agents and metabolites. These pumps rapidly remove the drugs from the cell, ensuring that drug concentrations are never high enough to trigger an antibacterial action. Most efflux pumps are multidrug transporters that effectively pump multiple types of antibiotics, which leads to multidrug resistance. There have been five super families of efflux pumps identified at this point (Figure 1.5). Among them are the ATP-binding cassette (ABC) family, the small multidrug resistance family (SMR), the major facilitator superfamily (MFS), the resistance-nodulation-division (RND) family, and the multidrug and toxic compound extrusion family (MATE). The Poly selective efflux pump, a member of the RND superfamily, is the most frequent kind of efflux pump found in Gram-negative bacteria and plays an important role in the multidrug resistance (MDR) bacterial phenotype. Many antibiotics are ejected by this kind of pump. Multidrug efflux pumps like AcrAB-TolC and MexAB-OprM usually belong to the RND superfamily. These two efflux pumps are necessary for bacterial survival, especially in the presence of toxic compounds. Additionally, two more efflux pumps, MexAB-OprM and MexCD-OprJ, are responsible for resistance to at least three major types of antibiotics, including carbapenems, fluoroquinolones, and aminoglycosides (Santajit & Indrawattana, 2016).

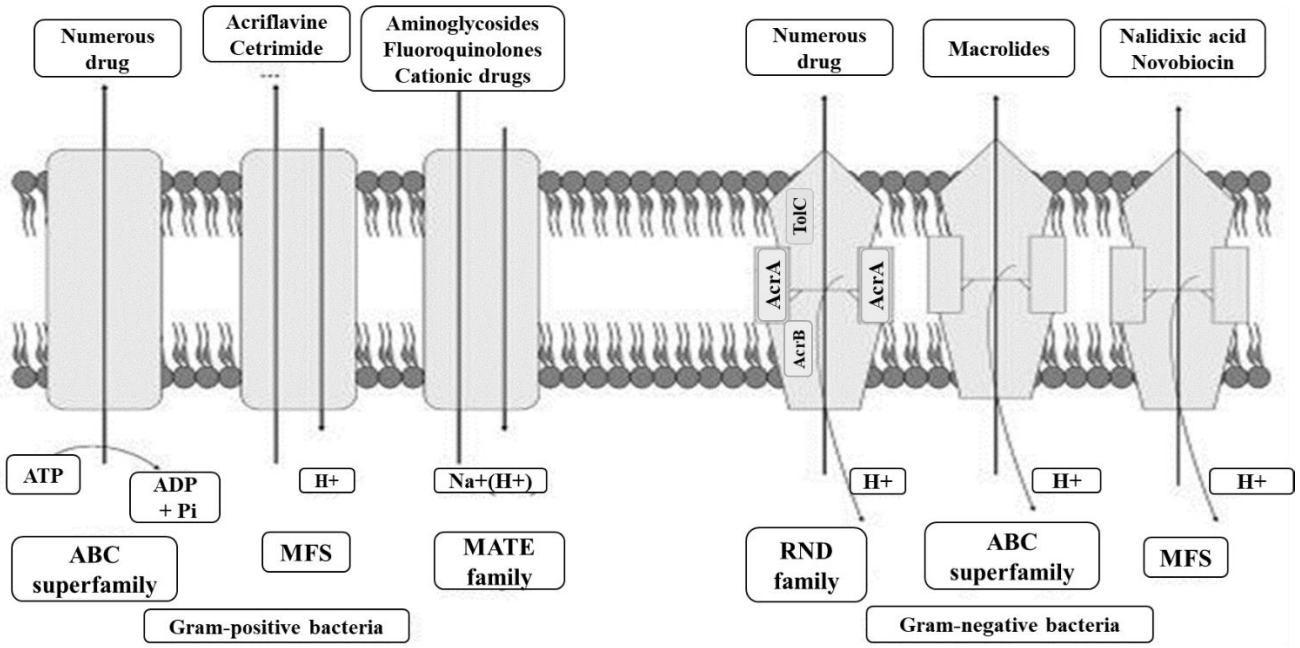

Figure 1.5: Efflux pump families (Soto, 2013)

1.8 Resistance Mediated by Plasmid

Plasmids are DNA molecules located outside of the chromosome that can replicate independently. They could impart resistance to many antibiotic classes, such as aminoglycosides, tetracyclines, trimethoprim, sulfonamides, macrolides, β-lactams, and quinolones. To mobilize the genes responsible for resistance to antibiotics, plasmids acquire mobile genetic elements (MGE) like transposons and insertion sequences (Carattoli, 2013). Insertion sequences (IS) and transposons (Tn) are discrete DNA segments linked to resistance genes that can transfer practically arbitrarily to new positions in the same or other DNA molecules within a single cell. Other elements, such as integrons (In), employ site-specific recombination to transfer resistance genes between predetermined locations. These kinds of mobile genetic elements (MGE) can also promote homologous recombination as they are frequently found in many copies in various places within a genome (Partridge et al., 2018). Plasmids accrue antibiotic resistance genes as a result of these

recombination systems' activity. Examples of these elements and processes are shown in figure 1.6.

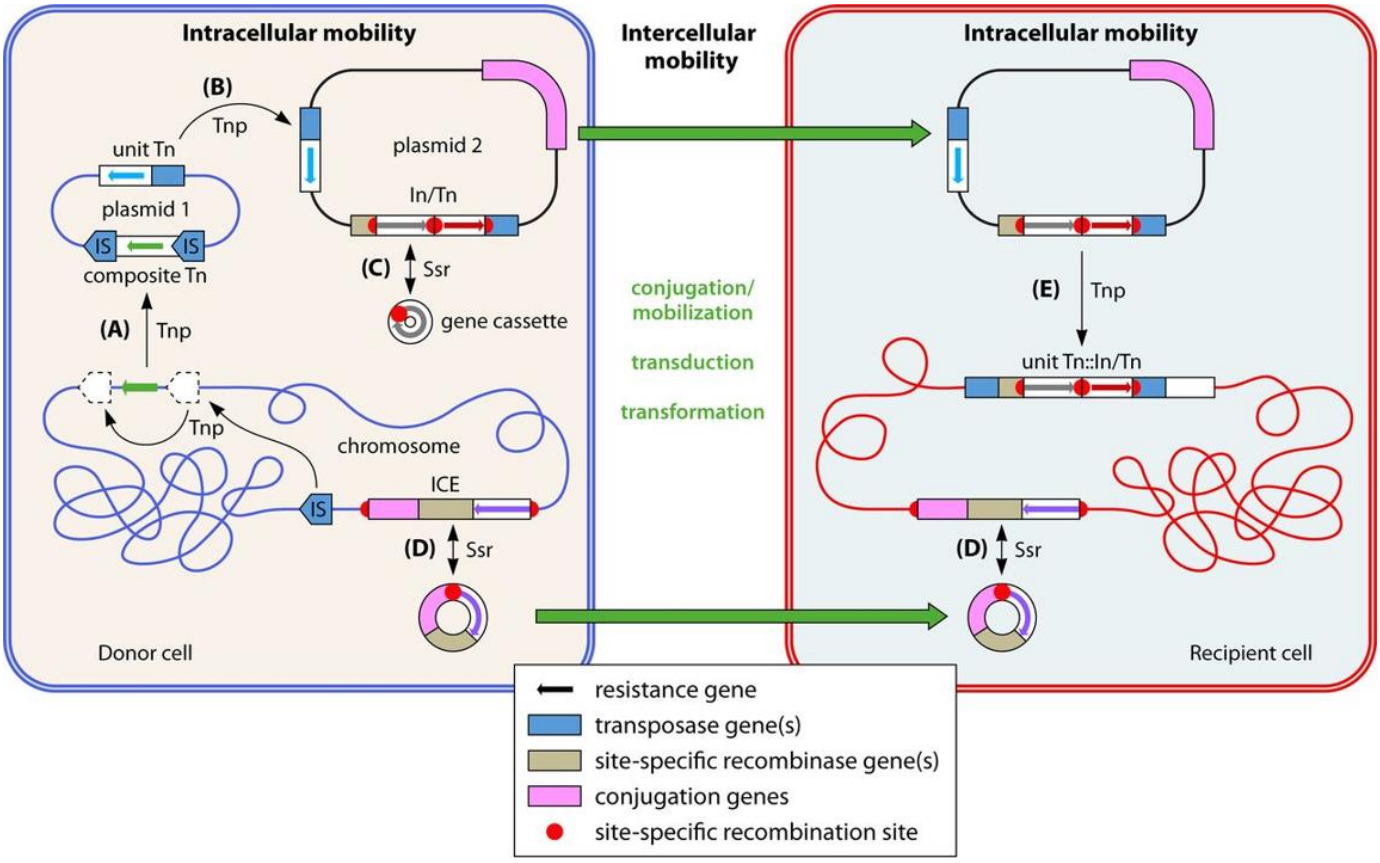

Figure 1.6: Examples of mobile genetic elements (MGE) and processes involved in intracellular mobility or intercellular transfer of antibiotic resistance genes (Partridge et al., 2018).

1.9 Resistance Mediated by Chromosomal DNA Mutations

Microorganisms can develop antibiotic resistance phenotypes through chromosomal DNA mutations, which modify the proteins that are already present in the bacteria (van Hoek et al., 2011). Each bacteria have a big circular piece of DNA called a chromosome that contains all its genetic material. Every time chromosomal DNA is replicated during growth, small-scale mutations take place. Mutations resulting in antimicrobial resistance alter the antibiotic action via one of the

following mechanisms**, I)** modifications of the antimicrobial target, **ii)** increased activation of efflux mechanisms to extrude the harmful molecule (Munita & Arias, 2016).

1.9.1 Target Modification

The bacterial protein GyrA, which is necessary for maintaining DNA structure. Topoisomerase IV and DNA gyrase both have two subunits: ParC/GrlA and ParE/GrlB for topoisomerase IV, and GyrA and GyrB for DNA gyrase. These subunits are essential for important ATP-dependent processes during DNA replication. The fluoroquinolones target the GyrA and ParC/GrlA proteins, which have DNA-binding properties. The antibiotics prevent GyrA from performing its functions by blocking the DNA-binding portion of the protein. Mutations in the GyrA gene can cause bacteria to become resistant to fluoroquinolone antibiotics by decreasing the bacterial binding of the antibiotic while maintaining the DNA-binding function intact. The mutation allows the bacteria to grow and survive when this kind of antibiotic is present (Alekshun & Levy, 2007).

1.9.2 Increased activation of Efflux pump

Chromosomal mutations may increase efflux pump synthesis by modifying the proteins that control their levels. The overproduction of efflux pumps will lead to a rise in resistance to the drugs class that the pump is intended to target. In gram-positive bacteria, quinolones must pass through the cytoplasmic membrane and cell wall to reach the targets; in gram-negative bacteria, quinolones must also pass through an extra outer membrane barrier. By modifying the expression of outer membrane porin proteins, such as OmpF and OmpC in E. coli, which generate channels for passive diffusion, gram-negative bacteria may control membrane permeability. Furthermore,

nonspecific, energy-dependent efflux systems are present in both gram-positive and gram-negative bacteria. In E. coli, the AcrAB-TolC efflux pump is essential for quinolone efflux. Mutations in acrR (a repressor of acrAB) increase pump activity. Mutations that inactivate marR (a regulator of marA) allow MarA to activate acrAB, tolC, and a gene that inhibits ompF translation, reducing influx and increasing efflux of quinolones (Jacoby, 2005).

1.10 Multi-Drug Resistance Pathogens

Pathogens that have acquired insensitivity to one or more antibiotics are defined as multidrug-resistant pathogens. Among MDR pathogens, ESKAPE pathogens (Figure 1.7) have become more of a threat due to their non-susceptibility to antibiotics (Idris & Nadzir, 2023a).

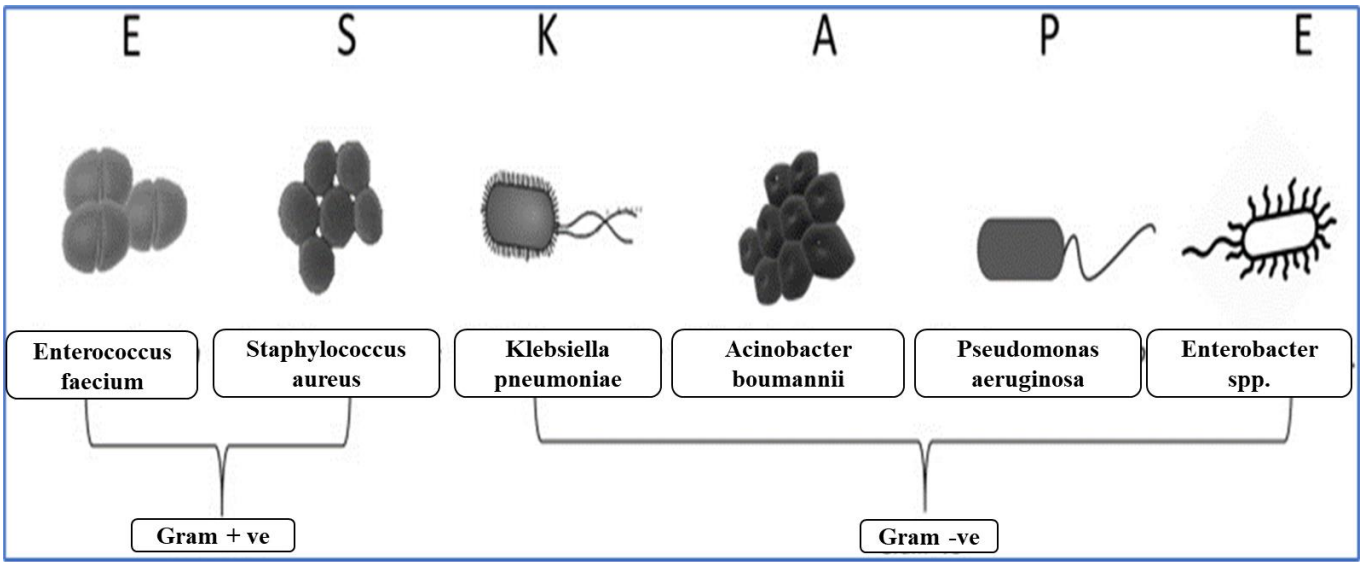

Figure 1.7: ESKAPE MDR Bacteria (Idris & Nadzir, 2023b)

According to Pandey et al. (2021), ESKAPE pathogens were most frequently isolated from urine samples (49.9%). Other common sources of infection included pus (22.3%), sputum (21.5%), blood (2.7%), semen (1.3%), high vaginal swabs (0.7%), wound swabs (0.4%), endotracheal tubes

(0.4%), ear swabs (0.2%), broncho alveolar lavage (0.2%), suction tip (0.2%), and oral swab (0.2%). Based on gender, ESKAPE pathogens were recovered more from females, than men.

1.11 Glass Pathogens

The Global Antimicrobial Resistance and Use Surveillance System (GLASS) is the first worldwide initiative to standardize AMR surveillance, approved by the World Health Assembly's sixty-eighth session. GLASS was designed to gradually include data from human AMR surveillance, such as resistance monitoring and antibiotic usage. GLASS-AMR includes the following pathogens: *Acinetobacter spp., E. coli, Klebsiella pneumoniae, Neisseria gonorrhoeae, Salmonella spp., Shigella spp., Staphylococcus aureus, and Streptococcus pneumoniae*.

Chapter 2: Objective of the study

The aim of this research was to examine the frequency of pathogens resistant to several drugs in male and female patients in Narayanganj, Bangladesh. Antibiotic resistance is a consequence of the widespread use of antibiotics or may occur due to self-medication practice for the treatment of infectious infections in underdeveloped nations or other reasons. Antibiotic treatments often include resistant bacteria. Because of improper antibiotic usage, antibiotic-resistant bacteria are becoming more prevalent worldwide. To minimize antibiotic resistance, costs, and patient suffering, one must recognize etiological microorganisms, their prevalence and their resistance patterns in particular geographic locations. This research will assist by using epidemiological information to evaluate the type and extent of resistance patterns, highlight key factors, and provide information for infection control and antimicrobial stewardship programs.

Chapter 3: Methodology

3.1 Study settings

A retrospective study was conducted using 650 isolates from Pro-Active Medical College & Hospital Limited Microbiology Laboratory between 05/10/2023 and 02/11/2023.

3.2 Type of samples

All the isolates were obtained from the following types of biological specimens:

- Urine
- Blood
- Pus
- Sputum
- Stool
- Wound swab
- Throat swab
- Tracheal aspirate

3.3 Data collection

Data collection was done using a pre-designed questionnaire (Figure 3.1) that included information on the following:

1. Date
2. Sample code
3. Specimen Type
4. Organism name
5. Patient info
6. Antibiogram result

Data Collection Form

Date of collection:

1. Sample Code:

2. Sample Type: Blood/Urine/Stool/ Sputum/Wound Swab/ Others (.....

3. Organism Name:

4. Patient Info:

a) Age: b) Gender: M / F

5.Antibiogram Result:

Name of the Antibiotics	R	S
Penicillin		
Amoxicillin		
Ampicillin		
Piperacillin		
Oxacillin		
Ceftriaxone		
Ceftazidime		
Cefotaxime		
Cefoxitin		
Cefuroxime		

Figure 3.1: Data collection form

After that, an excel sheet was created with the patient's age, sample collection date, and antibiotics susceptible or resistant to. Then the data was broken down by age, gender, and antibiotic susceptibility.

3.4 MAR index determination

Multiple antibiotic resistance (MAR) index (Ayandele et al., 2020) was calculated by employing this formula:

$$\text{MAR Index} = a / b$$

where, a = antibiotics number to which an isolate is resistant, b = total antibiotics number utilized in this study.

3.5 Statistical Analysis

All the data showing comparisons between male and female were analyzed for statistical significance by determination of p-values using Microsoft Excel, 2021.

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Chapter 4: Results and Discussion

4.1 Culture Positivity of Specimens

Clinical specimens which met the criteria as recommended by American Society for Microbiology (ASM) were processed for culture and susceptibility test. After receiving specimens from sample collection sites, they were immediately transported to microbiology laboratory for further processing. Out of 2340 specimens processed, 650 (28%) showed positive growth. Among 650 positive tested samples implicating infection showed (Figure 4.1) infection caused by pathogens. The most common isolate was *Escherichia coli* (n = 419, 65%) followed by *Staphylococcus aureus* (n = 29, 4%), *Klebsiella spp.* (n = 43, 7%), *Salmonella typhi* (n = 118, 18%), *Staphylococcus* (n = 21, 3%), and others like *Enterococcus faecalis, Proteus species, Pseudomonas species, Streptococcus pneumoniae, Streptococcus pyrogens* (n = 20, 3%).

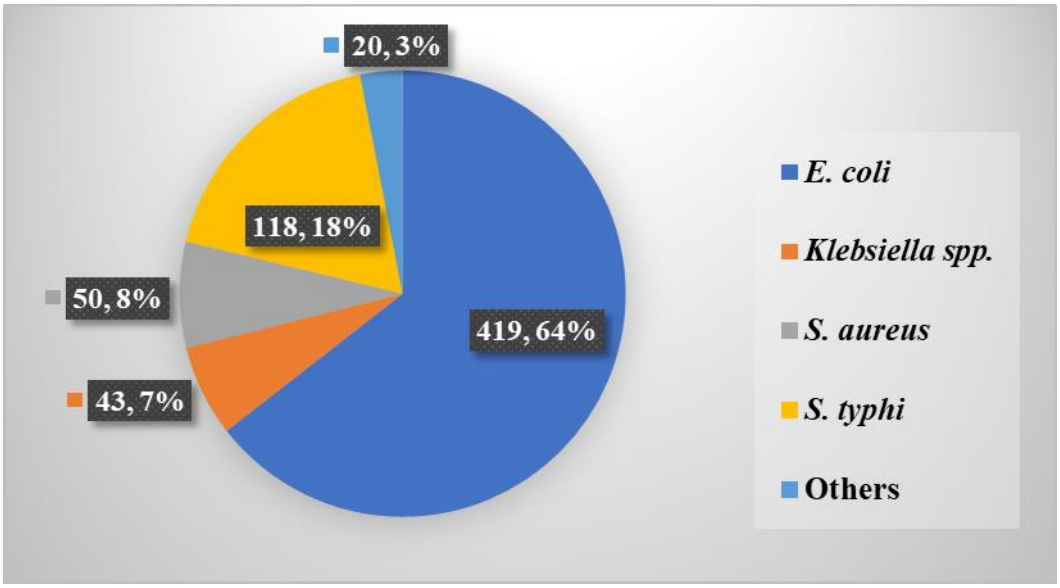

Figure 4.1: Percentage of Pathogens

4.2 Distribution of Pathogens Among the Diverse Clinical Specimens

Among the different clinical specimens processed, pathogens were most isolated from urine specimens followed by blood, pus, sputum, stool and other bodily fluids (Table 4.1).

Table 4.1: *Distribution of Pathogens Among the Diverse Clinical Specimens.*

Specimen Type	**No.**	**Percentage (%)**
Urine	425	65.4 %
Blood	150	23.1 %
Sputum	6	0.9 %
Pus	25	3.8 %
Stool	22	3.4 %
Wound swab	19	2.9 %
Throat swab	2	0.3 %
Tracheal aspirate	1	0.2 %
Total	**650**	**100**

Considering gender, 433 (66.6%) pathogens were isolated from females and 217 (33.4%) from males. As shown in Table 4.2, the maximum number of patients infected were of the age group 1–10 years followed by 21–30 years of age.

Table 4.2: *Age group of patients infected by pathogens.*

Age Group (Years)	**Male**		**Female**		***p*-value**
	No.	**Percentage (%)**	**No.**	**Percentage (%)**	
1-10	77	35.5 %	86	19.9 %	$p < 0.05$, Statistically significant
11-20	24	11.1 %	62	14.3 %	
21-30	21	9.7 %	100	23.1 %	
31-40	26	12.0 %	58	13.4 %	
41-50	17	7.8 %	37	8.5 %	
51-60	13	6.0 %	39	9.0 %	
61-70	23	10.6 %	37	8.5 %	
71-80	10	4.6 %	10	2.3 %	
81-90	6	2.8 %	3	0.7 %	
91-100	0	0	0	0	
101-110	0	0	1	0.2 %	

4.3 Pathogens distribution in males and females

From table 4.3, it is found that *E. coli* are more prevalent in female patients compare to male patients. This study is also relevant to another study that was done in Madrid, Spain in which the number of female patients infected through *E. coli* was larger than the number of male patients (Alós et al., 2005). However as seen in figure 4.2, *S. aureus, S. typhi* are more common in male patients than female patients.

Table 4.3: *Pathogens distribution according to gender*

Micro-organism's name	Male		Female		*p*-value
	No.	Percentage (%)	No.	Percentage (%)	
E. Coli	101	46.5 %	318	73.4 %	$p > 0.05$
Klebsiella spp.	14	6.5 %	29	6.7 %	
S. Aureus	25	11.5 %	25	5.8 %	
S. Typhi	62	28.6 %	56	12.9 %	
Others	15	6.9 %	5	1.2 %	
Total	**217**	**100.0**	**433**	**100.0**	

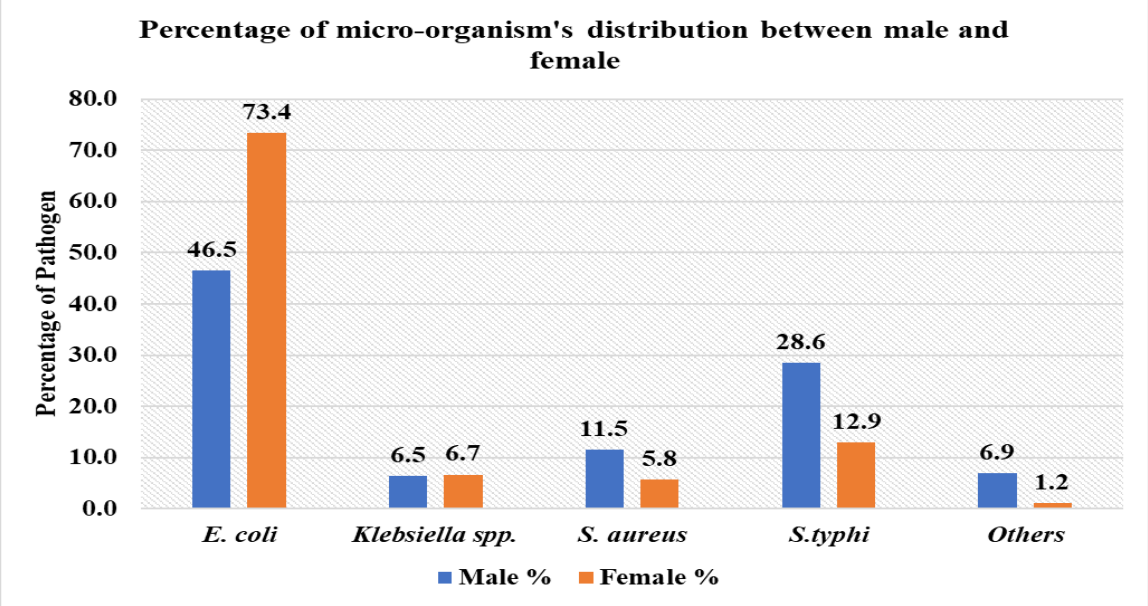

Figure 4.2: Percentage of pathogens among male and female

4.4 Antibiotic Susceptibility Pattern of Pathogens

4.4.1 Antibiotic susceptibility pattern of *Escherichia coli*

The *Escherichia coli* isolates were subjected to Antimicrobial Susceptibility Test (AST) with 39 different antibiotics (Table 4.4). High percentage of resistance (97%) was seen against linezolid followed by flucloxacillin (93.5%), vancomycin (90.9%) and clindamycin (90.9%), erythromycin (89.7%). However, a high percentage of sensitivity was seen against meropenem (96.7%) followed by amikacin (92.7%) and gentamicin (89.1%).

Table 4.4: *Antibiotic susceptibility pattern for Escherichia coli*

Antibiotic	**Resistant**		**Sensitive**	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Penicillin G	96	89.7	11	10.3
Flucloxacillin	260	93.5	18	6.5
Amoxy Clav	75	52.1	69	47.9
Amoxicillin	164	74.2	52	25.8
Ampicillin	103	85.1	18	14.9
Cloxacillin	4	57.1	3	42.9
Mecillinam	2	100.0	Not given	
Piperacillin	74	48.4	79	51.6
Piperacillin + Tazobactam	9	10.5	77	89.5
Amikacin	27	7.3	341	92.7
Gentamicin	38	10.9	311	89.1

Table 4.4: *contd.*

Antibiotic	Resistant		Sensitive	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Netilmicin	20	11.6	198	88.4
Cefepime	44	44.0	56	56.0
Cefixime	251	61.5	157	38.5
Ceftazidime	125	43.7	161	56.3
Cefuroxime	152	54.1	129	45.9
Cephalexin	1	33.3	2	66.7
Cephradine	158	84.5	29	15.5
Cefotaxime	130	60.5	85	39.5
Cefoxitin	39	44.8	48	55.2
Ceftriaxone	176	46.8	200	53.2
Chloramphenicol	30	11.2	239	88.8
Ciprofloxacin	140	38.5	224	61.5
Levofloxacin	106	30.2	245	69.8
Imipenem	80	29.5	191	70.5
Meropenem	9	3.3	267	96.7
Vancomycin	229	90.9	23	9.1
Tigecycline	12	52.2	11	47.8
Azithromycin	194	51.2	185	48.8
Erythromycin	140	89.7	16	10.3

Table 4.4: *contd*.

Antibiotic	Resistant		Sensitive	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Clindamycin	160	90.9	16	9.1
Linezolid	164	97.0	5	3.0
Colistin	145	66.5	73	33.5
Nalidixic acid	128	65.3	68	34.7
Cotrimoxazole	107	50.5	105	49.5
Nitrofurantoin	64	19.5	265	80.5
Doxycycline	83	29.7	196	70.3
Tetracycline	108	43.0	143	57.0

4.4.2 Resistance pattern of *Escherichia coli* to different antibiotics according to gender

After evaluating data, it was shown (Table 4.5) that all antibiotics are more resistant to female patients than male ones.

Table 4.5: *Resistance pattern of Escherichia coli according to gender*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Penicillin G	26	27.1	70	72.9
Flucloxacillin	65	25.0	195	75.0
Amoxy Clav	24	32.0	51	68.0
Amoxicillin	47	28.7	117	71.3
Ampicillin	18	17.5	85	82.5
Cloxacillin	1	25.0	3	75.0
Mecillinam	0	0.0	2	100.0
Piperacillin	20	27.0	54	73.0
Piperacillin +Tazobactam	3	33.3	6	66.7
Amikacin	9	33.3	18	66.7
Gentamicin	11	28.9	27	71.1
Netilmicin	7	35.0	13	65.0
Cefepime	15	34.1	29	65.9
Cefixime	68	27.1	183	72.9
Ceftazidime	36	28.8	89	71.2
Cefuroxime	46	30.3	106	69.7
Cephalexin	1	100.0	0	0.0
Cephradine	39	24.7	119	75.3
Cefotaxime	33	25.4	97	74.6
Cefoxitin	15	38.5	24	61.5

Table 4.5: *contd.*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Ceftriaxone	46	26.1	130	73.9
Chloramphenicol	9	30.0	21	70.0
Ciprofloxacin	34	24.3	106	75.7
Levofloxacin	34	32.1	72	67.9
Imipenem	24	30.0	56	70.0
Meropenem	5	55.6	4	44.4
Vancomycin	50	21.8	179	78.2
Tigecycline	4	33.3	8	66.7
Azithromycin	49	25.3	145	74.7
Erythromycin	33	23.6	107	76.4
Clindamycin	40	25.0	120	75.0
Linezolid	42	25.6	122	74.4
Colistin	35	24.1	110	75.9
Nalidixic acid	32	25.0	96	75.0
Cotrimoxazole	31	29.0	76	71.0
Nitrofurantoin	13	20.3	51	79.7
Doxycycline	21	25.3	62	74.7
Tetracycline	29	26.9	79	73.1

4.4.3 Resistance pattern of *Escherichia coli* to different antibiotics according to age

Compared to other patient groups, people in the age of 21 to 60 years show greater resistance (Table 4.6) to the provided antibiotics**.**

Table 4.6: *Resistant pattern of Escherichia coli according to age*

Antibiotic	**Age group wise resistant pattern of *E. coli***		
	0-20 years	**21-60 years**	**61-110 years**
Penicillin G	41	35	20
Flucloxacillin	96	128	36
Amoxy Clav	28	33	14
Amoxicillin	63	72	29
Ampicillin	37	50	16
Cloxacillin	2	2	0
Mecillinam	0	2	0
Piperacillin	32	31	11
Piperacillin + Tazobactam	2	3	4
Amikacin	8	16	3
Gentamicin	12	18	8
Netilmicin	7	16	3
Cefepime	18	17	9
Cefixime	101	113	37
Ceftazidime	48	58	19
Cefuroxime	64	64	24
Cephalexin	1	0	0
Cephradine	69	75	14
Cefotaxime	55	59	16
Cefoxitin	15	17	7

Table 4.6: *contd.*

Antibiotic	Age group wise resistant pattern of *E. coli*		
	0-20 years	**21-60 years**	**61-110 years**
Ceftriaxone	71	78	27
Chloramphenicol	11	13	6
Ciprofloxacin	54	64	22
Levofloxacin	47	47	12
Imipenem	25	39	16
Meropenem	4	3	2
Vancomycin	79	112	38
Tigecycline	6	4	2
Azithromycin	77	90	27
Erythromycin	41	72	27
Clindamycin	50	83	27
Linezolid	62	73	29
Colistin	62	63	20
Nalidixic acid	51	52	25
Cotrimoxazole	46	46	15
Nitrofurantoin	14	38	12
Doxycycline	26	35	22
Tetracycline	50	43	15

4.4.4 Antibiotic susceptibility pattern of *Staphylococcus aureus*

A significant portion of *S. aureus* isolates (81.3%) were resistant to cefixime, while colistin (79.2%) and nalidixic acid (80%) were subsequent (Table 4.7). While amikacin (97.7%) showed

highest percentage of sensitivity followed by ceftriaxone (88.9%), meropenem (91.7 %) and imipenem (96.2 %).

Table 4.7: *Antibiotic susceptibility pattern for Staphylococcus aureus.*

Antibiotic	**Resistant**		**Sensitive**	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Penicillin G	6	75	2	25
Flucloxacillin	19	65.5	10	34.5
Amoxy Clav	5	27.8	13	72.2
Amoxicillin	11	34.4	21	65.6
Ampicillin	6	60	4	40
Cloxacillin	Not given		Not given	
Mecillinam	Not given		Not given	
Piperacillin	4	16.7	20	83.3
Piperacillin + Tazobactam	1	10	9	90
Amikacin	1	2.3	43	97.7
Gentamicin	6	15.4	33	84.6
Netilmicin	5	23.8	16	76.2
Cefepime	6	46.2	7	53.8
Cefixime	39	81.3	9	18.8
Ceftazidime	24	68.6	11	31.4
Cefuroxime	8	33.3	16	66.7
Cephalexin	Not given		1	100.0
Cephradine	16	64.0	9	36.0
Cefotaxime	11	32.4	23	67.6
Cefoxitin	5	26.3	14	73.7
Ceftriaxone	5	11.1	40	88.9
Chloramphenicol	5	13.5	32	86.5

Table 4.7: *contd.*

Antibiotic	Resistant		Sensitive	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Ciprofloxacin	21	47.7	23	52.3
Levofloxacin	15	33.3	30	66.7
Imipenem	1	3.8	25	96.2
Meropenem	3	8.3	33	91.7
Vancomycin	10	24.4	31	75.6
Tigecycline	Not given		3	100
Azithromycin	25	52.1	23	47.9
Erythromycin	7	58.3	5	41.7
Clindamycin	3	15.8	16	84.2
Linezolid	4	21.1	15	78.9
Colistin	19	79.2	5	20.8
Nalidixic acid	8	80.0	2	20.0
Cotrimoxazole	21	77.8	6	22.2
Nitrofurantoin	9	22.0	32	78.0
Doxycycline	4	18.2	18	81.8
Tetracycline	17	48.6	18	51.4

4.4.5 Resistance pattern of *Staphylococcus aureus* to different antibiotics according to gender

Data analysis showed (Table 4.8) that female patients are more resistant to all antibiotics than male patients.

Table 4.8: *Resistance pattern of Staphylococcus aureus according to gender*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Penicillin G	2	33.3	4	66.7
Flucloxacillin	9	47.4	10	52.6
Amoxy Clav	3	60.0	2	40.0
Amoxicillin	4	36.4	7	63.6
Ampicillin	4	66.7	2	33.3
Cloxacillin	0	0.0	0	0.0
Mecillinam	0	0.0	0	0.0
Piperacillin	2	50.0	2	50.0
Piperacillin + Tazobactam	1	100.0	0	0.0
Amikacin	0	0.0	1	100.0
Gentamicin	3	50.0	3	50.0
Netilmicin	1	20.0	4	80.0
Cefepime	3	50.0	3	50.0
Cefixime	17	43.6	22	56.4
Ceftazidime	10	41.7	14	58.3
Cefuroxime	4	50.0	4	50.0
Cephalexin	0	0.0	0	0.0
Cephradine	8	50.0	8	50.0

Table 4.8: *contd.*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Cefotaxime	5	45.5	6	54.5
Cefoxitin	2	40.0	3	60.0
Ceftriaxone	3	60.0	2	40.0
Chloramphenicol	4	80.0	1	20.0
Ciprofloxacin	7	33.3	14	66.7
Levofloxacin	7	46.7	8	53.3
Imipenem	0	0.0	1	100.0
Meropenem	1	33.3	2	66.7
Vancomycin	6	60.0	4	40.0
Tigecycline	0	0.0	0	0.0
Azithromycin	10	40.0	15	60.0
Erythromycin	5	71.4	2	28.6
Clindamycin	2	66.7	1	33.3
Linezolid	2	50.0	2	50.0
Colistin	9	47.4	10	52.6
Nalidixic acid	5	62.5	3	37.5
Cotrimoxazole	8	38.1	13	61.9
Nitrofurantoin	5	55.6	4	44.4
Doxycycline	4	100.0	0	0.0
Tetracycline	10	58.8	7	41.2

4.4.6 Resistance pattern of *Staphylococcus aureus* to different antibiotics according to age

Table 4.9 indicates that patients between the ages of 21 and 60 have higher levels of antibiotic resistance than other patient categories.

Table 4.9: *Resistant pattern of Staphylococcus aureus according to age*

Antibiotic	**Age group wise resistant pattern of *S. aureus***		
	0-20 years	**21-60 years**	**61-110 years**
Penicillin G	3	3	0
Flucloxacillin	5	13	1
Amoxy Clav	0	5	0
Amoxicillin	4	5	2
Ampicillin	1	4	1
Cloxacillin	0	0	0
Mecillinam	0	0	0
Piperacillin	1	2	1
Piperacillin + Tazobactam	1	0	0
Amikacin	0	1	0
Gentamicin	0	4	2
Netilmicin	1	4	0
Cefepime	2	4	0
Cefixime	12	20	7
Ceftazidime	7	13	4
Cefuroxime	2	5	1
Cephalexin	0	0	0
Cephradine	4	9	3
Cefotaxime	3	5	3
Cefoxitin	2	4	1

Table 4.9: *contd.*

Antibiotic	**Age group wise resistant pattern of *S. aureus***		
	0-20 years	**21-60 years**	**61-110 years**
Ceftriaxone	1	4	0
Chloramphenicol	1	1	3
Ciprofloxacin	5	13	3
Levofloxacin	4	8	3
Imipenem	1	0	0
Meropenem	0	3	0
Vancomycin	4	6	0
Tigecycline	0	0	0
Azithromycin	7	16	2
Erythromycin	0	6	1
Clindamycin	0	3	0
Linezolid	0	4	0
Colistin	4	14	1
Nalidixic acid	2	5	1
Cotrimoxazole	6	12	3
Nitrofurantoin	2	7	0
Doxycycline	1	3	0
Tetracycline	5	9	3

4.4.7 Antibiotic susceptibility pattern of *Salmonella typhi*

The majority of *S. typhi* isolates (92.6%) were resistant to flucloxacillin; (81.7%) and (71.4%) were resistant to clindamycin and penicillin G respectively (Table 4.10). The highest percentage of sensitivity was seen with meropenem (96.3%), followed by doxycycline (91%), levofloxacin (93.3%), and chloramphenicol (92.3%).

Table 4.10: *Antibiotic susceptibility pattern for Salmonella typhi*

Antibiotic	Resistant		Sensitive	
	No.	Percentage (%)	No.	Percentage (%)
Penicillin G	25	71.4	10	28.6
Flucloxacillin	75	92.6	6	7.4
Amoxy Clav	10	17.9	46	82.1
Amoxicillin	13	22.8	44	77.2
Ampicillin	13	40.6	19	59.4
Cloxacillin	1	100.0	Not given	
Mecillinam	1	100.0	Not given	
Piperacillin	16	35.6	29	64.4
Piperacillin + Tazobactam	2	8.7	21	91.3
Amikacin	7	8.0	81	92.0
Gentamicin	8	8.3	88	91.7
Netilmicin	3	5.8	49	94.2
Cefepime	10	29.4	24	70.6
Cefixime	52	44.4	65	55.6
Ceftazidime	29	38.7	46	61.3
Cefuroxime	18	25.4	53	74.6
Cephalexin	1	50.0	1	50.0
Cephradine	37	63.8	21	36.2
Cefotaxime	20	32.8	41	67.2

Table 4.10: *contd.*

Antibiotic	Resistant		Sensitive	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Cefoxitin	5	18.5	22	81.5
Ceftriaxone	19	17.9	87	82.1
Chloramphenicol	6	7.7	72	92.3
Ciprofloxacin	20	19.4	83	80.6
Levofloxacin	6	6.7	83	93.3
Imipenem	27	32.1	57	67.9
Meropenem	3	3.7	78	96.3
Vancomycin	41	56.2	32	43.8
Tigecycline	1	33.3	2	66.7
Azithromycin	43	39.1	67	60.9
Erythromycin	48	56.5	10	17.2
Clindamycin	58	81.7	13	18.3
Linezolid	39	67.2	19	32.8
Colistin	33	64.7	18	35.3
Nalidixic acid	51	77.3	15	22.7
Cotrimoxazole	18	33.3	36	66.7
Nitrofurantoin	9	11.0	73	89.0
Doxycycline	8	9.0	81	91.0
Tetracycline	9	13.8	56	86.2

4.4.8 Resistance pattern of *Salmonella typhi* to different antibiotics according to gender

Male patients are more resistant to all antibiotics than female patients, according to data Table 4.11.

Table 4.11: *Resistance pattern of Salmonella typhi according to gender*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Penicillin G	13	52.0	12	48.0
Flucloxacillin	38	50.7	37	49.3
Amoxy Clav	2	20.0	8	80.0
Amoxicillin	5	38.5	8	61.5
Ampicillin	8	61.5	5	38.5
Cloxacillin	0	0.0	1	100.0
Mecillinam	0	0.0	1	100.0
Piperacillin	10	62.5	6	37.5
Piperacillin + Tazobactam	0	0.0	2	100.0
Amikacin	4	57.1	3	42.9
Gentamicin	5	62.5	3	37.5
Netilmicin	3	100.0	0	0.0
Cefepime	8	80.0	2	20.0
Cefixime	28	53.8	24	46.2
Ceftazidime	16	55.2	13	44.8
Cefuroxime	8	44.4	10	55.6
Cephalexin	1	100.0	0	0.0
Cephradine	18	48.6	19	51.4
Cefotaxime	12	60.0	8	40.0
Cefoxitin	2	40.0	3	60.0
Ceftriaxone	8	42.1	11	57.9

Table 4.11: *contd.*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Chloramphenicol	6	100.0	0	0.0
Ciprofloxacin	11	55.0	9	45.0
Levofloxacin	2	33.3	4	66.7
Imipenem	10	37.0	17	63.0
Meropenem	2	66.7	1	33.3
Vancomycin	26	63.4	15	36.6
Tigecycline	1	100.0	0	0.0
Azithromycin	21	48.8	22	51.2
Erythromycin	23	47.9	25	52.1
Clindamycin	29	50.0	29	50.0
Linezolid	23	59.0	16	41.0
Colistin	22	66.7	11	33.3
Nalidixic acid	31	60.8	20	39.2
Cotrimoxazole	8	44.4	10	55.6
Nitrofurantoin	7	77.8	2	22.2
Doxycycline	7	87.5	1	12.5
Tetracycline	5	55.6	4	44.4

4.4.9 Resistance pattern of *Salmonella typhi* to different antibiotics according to age

Patients between the ages of 21 and 60 had greater levels of antibiotic resistance than other patient groups, according to Table 4.12.

Table 4.12: *Resistance pattern of Salmonella typhi according to age*

Antibiotic	**Age group wise resistant pattern of *S. typhi***		
	0-20 years	**21-60 years**	**61-110 years**
Penicillin G	10	14	1
Flucloxacillin	38	33	4
Amoxy Clav	5	5	0
Amoxicillin	7	5	1
Ampicillin	10	3	0
Cloxacillin	1	0	0
Mecillinam	1	0	0
Piperacillin	7	7	2
Piperacillin + Tazobactam	2	0	0
Amikacin	3	4	0
Gentamicin	3	4	1
Netilmicin	2	0	1
Cefepime	3	6	1
Cefixime	23	25	4
Ceftazidime	12	14	3
Cefuroxime	8	9	1
Cephalexin	1	0	0
Cephradine	19	18	0
Cefotaxime	8	9	3
Cefoxitin	0	3	0

Table 4.12: *contd.*

Antibiotic	**Age group wise resistant pattern of *S. typhi***		
	0-20 years	**21-60 years**	**61-110 years**
Ceftriaxone	4	12	3
Chloramphenicol	5	1	0
Ciprofloxacin	7	8	5
Levofloxacin	1	4	1
Imipenem	9	17	1
Meropenem	1	2	0
Vancomycin	18	21	2
Tigecycline	1	0	0
Azithromycin	17	23	3
Erythromycin	22	25	1
Clindamycin	26	30	2
Linezolid	19	19	1
Colistin	17	15	1
Nalidixic acid	21	28	2
Cotrimoxazole	11	5	2
Nitrofurantoin	6	2	1
Doxycycline	4	4	0
Tetracycline	7	2	0

4.4.10 Antibiotic susceptibility pattern of *Klebsiella spp.*

As shown in Table 4.13, flucloxacillin showed greater resistance (93.5%) against Klebsiella spp., followed by linezolid (93.3%) and clindamycin (89.5%). Also, amikacin demonstrated the highest percentage of sensitivity (91.7%).

Table 4.13: *Antibiotic susceptibility pattern for Klebsiella spp.*

Antibiotic	**Resistant**		**Sensitive**	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Penicillin G	4	80.0	1	20.0
Flucloxacillin	29	93.5	2	6.5
Amoxy Clav	5	50.0	5	50.0
Amoxicillin	20	76.9	6	23.1
Ampicillin	15	88.2	2	11.8
Cloxacillin	Not given		Not given	
Mecillinam	1	100.0	Not given	
Piperacillin	13	76.5	4	23.5
Piperacillin + Tazobactam	1	8.3	11	91.7
Amikacin	3	8.3	33	91.7
Gentamicin	5	13.9	31	86.1
Netilmicin	4	20.0	16	80.0
Cefepime	3	60.0	2	40.0
Cefixime	20	46.5	23	53.5
Ceftazidime	9	37.5	15	62.5
Cefuroxime	12	48.0	13	52.0
Cephalexin	Not given		Not given	
Cephradine	14	82.4	3	17.6
Cefotaxime	6	33.3	12	66.7
Cefoxitin	4	50.0	4	50.0

***Table 4.13: c**ontd.*

Antibiotic	Resistant		Sensitive	
	No.	**Percentage (%)**	**No.**	**Percentage (%)**
Ceftriaxone	10	24.4	31	75.6
Chloramphenicol	3	10.0	27	90.0
Ciprofloxacin	9	25.0	27	75.0
Levofloxacin	5	14.3	30	85.7
Imipenem	12	40.0	18	60.0
Meropenem	3	12.0	22	88.0
Vancomycin	26	83.9	5	16.1
Tigecycline	1	100.0	Not given	
Azithromycin	13	30.2	30	69.8
Erythromycin	21	100.0	Not given	
Clindamycin	17	89.5	2	10.5
Linezolid	14	93.3	1	6.7
Colistin	10	50.0	10	50.0
Nalidixic acid	10	41.7	14	58.3
Cotrimoxazole	11	47.8	12	52.2
Nitrofurantoin	14	42.4	19	57.6
Doxycycline	9	30.0	21	70.0
Tetracycline	8	27.6	21	72.4

4.4.11 Resistance pattern of *Klebsiella spp*. to different antibiotics according to gender

According to data Table 4.14, female patients are more resistant to all antibiotics than male patients.

Table 4.14: *Resistance pattern for Klebsiella spp. according to gender*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Penicillin G	0	0.0	4	100.0
Flucloxacillin	9	31.0	20	69.0
Amoxy Clav	2	40.0	3	60.0
Amoxicillin	6	30.0	14	70.0
Ampicillin	4	26.7	11	73.3
Cloxacillin	0	0	0	0
Mecillinam	0	0.0	1	100.0
Piperacillin	5	38.5	8	61.5
Piperacillin + Tazobactam	0	0.0	1	100.0
Amikacin	2	66.7	1	33.3
Gentamicin	2	40.0	3	60.0
Netilmicin	2	50.0	2	50.0
Cefepime	1	33.3	2	66.7
Cefixime	6	30.0	14	70.0
Ceftazidime	4	44.4	5	55.6
Cefuroxime	4	33.3	8	66.7
Cephalexin	0	0	0	0
Cephradine	4	28.6	10	71.4
Cefotaxime	3	50.0	3	50.0
Cefoxitin	3	75.0	1	25.0
Ceftriaxone	3	30.0	7	70.0

Table 4.14: *contd.*

Antibiotic	Percentage (%) of Resistant Pathogens			
	Male		Female	
	No.	%	No.	%
Chloramphenicol	0	0.0	3	100.0
Ciprofloxacin	4	44.4	5	55.6
Levofloxacin	3	60.0	2	40.0
Imipenem	4	33.3	8	66.7
Meropenem	0	0.0	3	100.0
Vancomycin	5	19.2	21	80.8
Tigecycline	1	100.0	0	0.0
Azithromycin	5	38.5	8	61.5
Erythromycin	6	28.6	15	71.4
Clindamycin	4	23.5	13	76.5
Linezolid	4	28.6	10	71.4
Colistin	4	40.0	6	60.0
Nalidixic acid	4	40.0	6	60.0
Cotrimoxazole	7	63.6	4	36.4
Nitrofurantoin	3	21.4	11	78.6
Doxycycline	2	22.2	7	77.8
Tetracycline	1	12.5	7	87.5

4.4.12 Resistance pattern for *Klebsiella spp*. to different antibiotics according to age

Table 4.15 shows that patients between the ages of 21 and 60 had higher levels of antibiotic resistance than other patient groups.

Table 4.15: *Resistance pattern for Klebsiella spp. according to age*

Antibiotic	**Age group wise resistant pattern of *Klebsiella spp.***		
	0-20 years	**21-60 years**	**61-110 years**
Penicillin G	1	1	2
Flucloxacillin	7	13	9
Amoxy Clav	2	3	0
Amoxicillin	6	11	3
Ampicillin	4	9	2
Cloxacillin	0	0	0
Mecillinam	0	1	0
Piperacillin	2	9	2
Piperacillin + Tazobactam	0	1	0
Amikacin	1	2	0
Gentamicin	2	2	1
Netilmicin	2	2	0
Cefepime	0	1	2
Cefixime	3	11	6
Ceftazidime	2	3	4
Cefuroxime	3	8	1
Cephalexin	0	0	0
Cephradine	3	10	1
Cefotaxime	1	4	1
Cefoxitin	1	2	1

Table 4.15: *contd.*

Antibiotic	**Age group wise resistant pattern of *Klebsiella spp.***		
	0-20 years	**21-60 years**	**61-110 years**
Ceftriaxone	1	5	4
Chloramphenicol	0	1	2
Ciprofloxacin	2	6	1
Levofloxacin	2	2	1
Imipenem	3	7	2
Meropenem	0	3	0
Vancomycin	6	14	6
Tigecycline	1	0	0
Azithromycin	3	6	4
Erythromycin	5	12	4
Clindamycin	2	12	3
Linezolid	5	5	4
Colistin	1	6	3
Nalidixic acid	2	7	1
Cotrimoxazole	4	4	3
Nitrofurantoin	3	8	3
Doxycycline	0	6	3
Tetracycline	0	6	2

4.4.13 Comparison of different antibiotic sensitivity and resistant pattern between male and female

According to Table 4.16, meropenem, amikacin, combinations of piperacillin and tazobactam, netilmicin, and gentamicin show greater sensitivity in both males and females. On the other hand, flucloxacillin, penicillin G, clindamycin, and linezolid show greater resistance in both male and female (Table 4.16).

***Table 4.16**: Comparison of different Antibiotic sensitivity and resistant pattern for male and female*

Antibiotic	Sensitive (%)		Resistance (%)	
	Male	Female	Male	Female
Penicillin G	25.4	11.7	74.6	88.3
Flucloxacillin	14.4	7.1	85.6	92.9
Amoxy Clav	56.6	57.2	43.4	42.8
Amoxicillin	45.2	34.1	54.8	65.9
Ampicillin	32.2	20.2	67.8	79.8
Cloxacillin	25.0	33.3	75.0	66.7
Mecillinam	Not given	Not given	Not given	100.0
Piperacillin	59.1	52.7	40.9	47.3
Piperacillin + Tazobactam	88.9	90.8	11.1	9.2
Amikacin	91.8	93.8	8.2	6.2
Gentamicin	86.8	89.8	13.2	10.2
Netilmicin	88.0	88.1	112	11.9
Cefepime	55.2	62.1	44.8	37.9
Cefixime	38.2	41.7	61.8	58.3
Ceftazidime	52.7	57.4	47.3	42.6
Cefuroxime	52.1	52.8	47.9	47.2
Cephalexin	33.3	100.0	66.7	Not given

Table 4.16: *contd.*

Antibiotic	Sensitive (%)		Resistance (%)	
	Male	**Female**	**Male**	**Female**
Cephradine	25.3	19.9	74.7	80.1
Cefotaxime	50.4	48.9	49.6	51.1
Cefoxitin	55.6	65.6	44.4	34.4
Ceftriaxone	68.0	60.9	32.0	39.1
Chloramphenicol	86.1	91.0	13.9	9.0
Ciprofloxacin	67.4	64.5	32.6	35.5
Levofloxacin	73.5	51.9	53.3	48.1
Imipenem	72.3	70.0	27.7	30.0
Meropenem	93.9	96.5	6.1	3.5
Vancomycin	30.9	18.8	69.1	81.2
Tigecycline	36.4	60.0	63.6	40.0
Azithromycin	53.0	52.25	47.0	47.75
Erythromycin	10.4	13.7	89.6	86.3
Clindamycin	22.4	14.1	77.6	85.9
Linezolid	22.2	14.1	77.6	86.3
Colistin	32.7	33.8	67.3	66.2
Nalidixic acid	25.0	37.2	75.0	62.8
Cotrimoxazole	46.7	51.9	53.3	48.1
Nitrofurantoin	79.1	79.1	20.9	20.9
Doxycycline	72.3	76.1	27.7	23.9
Tetracycline	61.4	62.1	38.6	37.9

4.5 Multi drug resistance & Non- Multi Drug resistance

Table 4.17 shows that compared to male patients (48.1%); female patients had a higher proportion of MDR *E. coli* (73.3%). However, in case of MDR *S. typhi*, the proportion of male patients is higher (27.9%) than that of female patients (13%) (figure 4.3). Additionally, just 1 male patient has non-MDR *E. coli* activity compared to 13 female cases. However, 4 male patients—more than female patients—show non-MDR activity for *S. aureus* and *S. typhi*, respectively (Table 4.17).

Table 4.17: *Percentage of MDR and Non-MDR pathogens among male and female*

Types of pathogens	**Male**				**Female**			
	No. of MDR	**%**	**No. of non-MDR**	**%**	**No. of MDR**	**%**	**No. of non-MDR**	**%**
E. coli	100	48.1	1	11.1	305	73.3	13	76.5
Klebsiella spp.	14	6.7	0	0	28	6.7	1	5.9
S. aureus	21	10.1	4	44.4	24	5.8	1	5.9
S. typhi	58	27.9	4	44.4	54	13.0	2	11.8
Others	15	7.2	0	0	5	1.2	0	0
Total	**208**	**100**	**9**	**100**	**416**	**100**	**17**	**100**

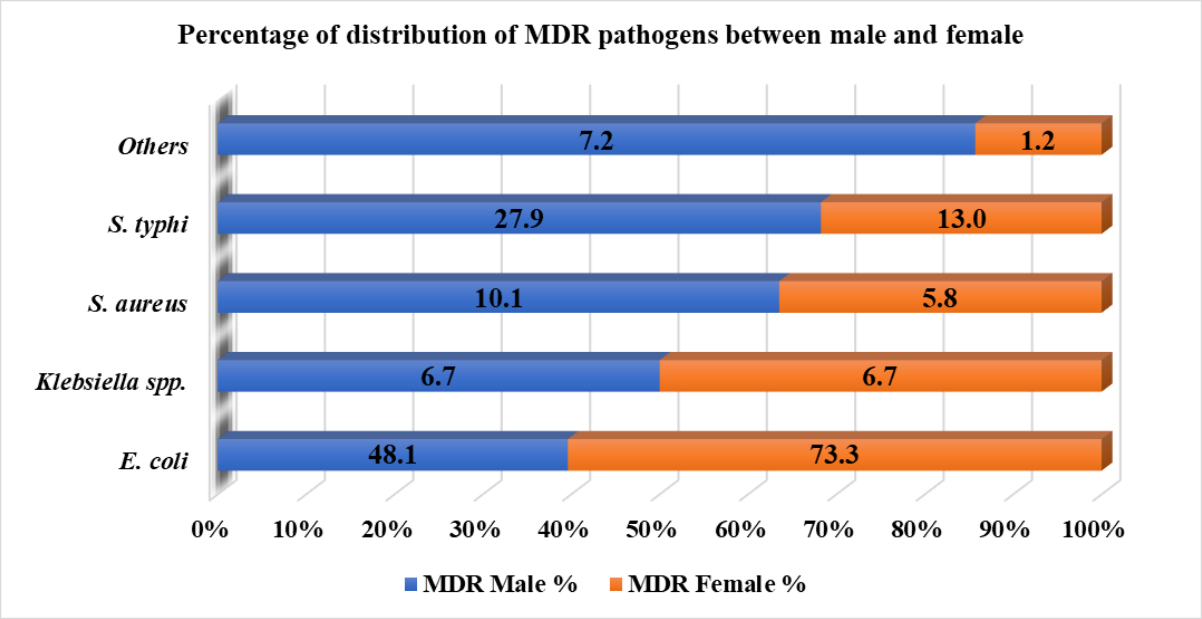

Figure 4.3: Percentage of distribution of MDR pathogens between male and female

By looking at figure 4.4, it is clear that 96% of total positive samples show multidrug resistance (MDR), while only 4% are non-MDR.

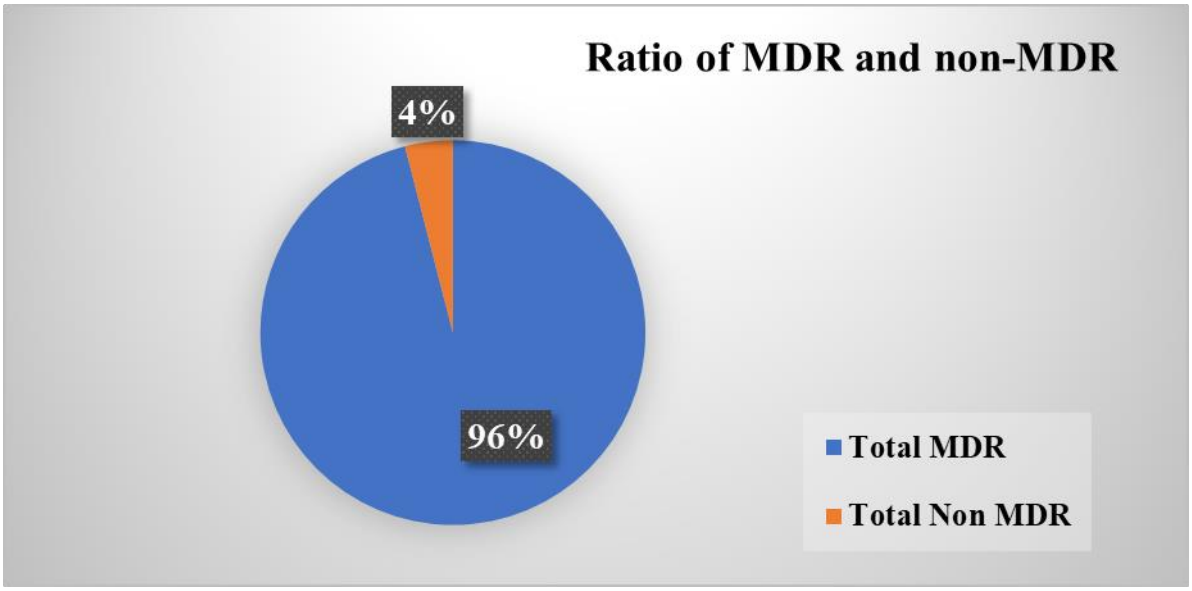

Figure 4.4: Ratio of MDR and non-MDR Bacteria

4.6 Multi antibiotic resistance index of various pathogens

4.6.1 Common Resistant Phenotype of *E. coli* for male patients

Three common resistant phenotype of *E. coli* exhibit highest resistance to the given antibiotics having MAR index 0.2 (Table 4.18).

***Table 4.18**: Common resistant phenotype and MAR index of E. coli for male patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMC, AMP, AZM, CFM, FLU	4	0.6 %	0.1
AMC, AMX, AZM, CE, CFM, DA, FLU	7	1 %	0.2
AMC, AZM, CAZ, CFM, CIP, CXM	4	0.6 %	0.1
AMC, CAZ, CE, CFM, CRO, CXM	5	0.7 %	0.1
AMK, AMX, AZM, CAZ, CE, CFM, CRO	6	0.9 %	0.2
AMK, CT, FLU	2	0.3 %	0.1
AMP, AMX, AZM	5	0.7 %	0.2
AMP, ERY, FLU	4	0.6 %	0.1
AMX, AZM, CAZ, CE, CFM, CFX, CIP, CRO, FLU, LFX, TE	7	1 %	0.2

Table 4.18: *contd.*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMX, AZM, CEF, CFM, DA, LZD, PG, VA	2	0.3 %	0.1
AMX, AZM, CAZ, CFM, CRO, LFX, LZD, NAL	2	0.3 %	0.1
AMX, CAZ, CFM, CRO, DA, ERY, PG, PRL	3	0.4 %	0.1
AMX, CAZ, CFM, CXM, FLU	2	0.3 %	0.1
AZM, CFM, CRO, CTX, CXM, FLU, NAL, VA	4	0.6 %	0.1
CAZ, CE, CFM, CTX, CXM	5	0.7 %	0.1
CFM, CT, CTX, LZD	3	0.4 %	0.1

4.6.2 Common Resistant Phenotype of *E. coli* for female patients

10 % of *E. coli* of exhibit resistance to the given pattern having MAR index 0.3. 8% of isolates are resistant to the given pattern with MAR index 0.4 (Table 4.19).

Table 4.19: *Common resistant phenotype and MAR index of E. coli for female patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMC, AMK, AZM, CAZ, CE, CFM, CIP, CRO, CT, ERY, FLU, LZD, VA	52	8 %	0.4
AMC, AZM, CAZ, CE, CFM, CIP, CRO	7	1 %	0.2
AMC, AMX, CE, CFM	7	1 %	0.1
AMK, AMX, AZM, CFM, CIP, CAZ, CEF, CRO	12	1.8 %	0.2
AMP, AMX, AZM, CAZ, CFM, CIP, CRO, ERY, FLU, LZD, LFX, NAL, NIT, VA	50	7.6 %	0.4

Table 4.19: *contd.*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMP, CE, CFM, CIP, CT, ERY, FLU	25	3.8 %	0.2
AMX, AZM, CAZ, CEF, CFM, CIP, ERY, FLU, CT, PG	68	10 %	0.3
AZM, CE, CFM, CIP, CRO, CTX	40	6 %	0.2
AZM, DA, FLU, LZD, VA	2	0.3 %	0.1
CAZ, CE, CFM	6	1 %	0.1
CAZ, CFM, CRO, FLU, CT, CTX, FLU	8	1.2 %	0.2
CE, CFM, CRO, CTX, CXM, FLU	12	1.8 %	0.2
CE, DA, ERY, FLU, VA	6	1 %	0.1
CRO, CT, CTX, FLU, LZD, NIT	2	0.3 %	0.2
DA, ERY, FLU, VA	5	0.8 %	0.1

4.6.3 Common Resistant Phenotype of *S. aureus* for male patients

S. aureus in male patients exhibits resistance to three different common resistant phenotype patterns with a MAR index of 0.1 (Table 4.20).

Table 4.20: *Common Resistant Phenotype and MAR index of S. aureus for male patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMC, AZM, CE, CFM, CRO, ERY	3	0.4 %	0.1
AZM, CAZ, CE, CFM	3	0.4 %	0.1
AMX, AZM, CFM, SXT	3	0.4 %	0.1

4.6.4 Common Resistant Phenotype of *S. aureus* for female patients

S. aureus in female patients exhibits resistance to three different common resistant phenotype patterns with a MAR index of 0.1 (Table 4.21).

Table 4.21: *Common Resistant Phenotype and MAR index of S. aureus for female patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMC, AZM, CAZ, CFM, CTX, FLU	2	0.3 %	0.1
AMP, AZM, CAZ, CEF, CFM	2	0.3 %	0.1
AMX, CAZ, CFM, SXT	3	0.4 %	0.1
AZM, CFM	2	0.3 %	0.1
CAZ, CFM, CT	3	0.4 %	0.1

4.6.5 Common Resistant Phenotype of *S. typhi* for male patients

Two different resistant phenotypes with a percentage of 0.3% and a MAR index of 0.2 are showing resistance to *S. typhi* in male patients. (Table 4.22).

Table 4.22: *Common Resistant Phenotype and MAR index of S. typhi for male patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMC, CAZ, CFM, FLU	2	0.3 %	0.1
AMK, AMX	2	0.3 %	0.1
AMP, AZM, ERY, FLU	4	0.6 %	0.1
AZM, CT, DA, ERY, FLU, NAL, PG	2	0.3 %	0.2
AZM, CAZ, FLU, LZD	3	0.4 %	0.1
AZM, CFM, DA, ERY, FLU, NAL, PG, VA	2	0.3 %	0.2
AZM, CAZ, CFM, DA	3	0.4 %	0.1
AZM, CE, CFM, FLU	2	0.3 %	0.1
CAZ, CE, CEF, CFM, CT	4	0.6 %	0.1
CAZ, CE, CFM	7	1 %	0.1
CE, DA, ERY, FLU	3	0.4 %	0.1
CFM, ERY, FLU, VA	4	0.6 %	0.1
CT, LZD	4	0.6 %	0.1
CT, LZD, NAL	3	0.4 %	0.1
ERY, FLU, NAL	3	0.4 %	0.1
DA, ERY, FLU, NAL	2	0.3 %	0.1

4.6.6 Common Resistant Phenotype of *S. typhi* for female patients

S. typhi resistance in female patients is shown by two distinct resistant phenotypes with a percentage of 0.4% and a MAR index of 0.2 (Table 4.23).

Table 4.23: *Common Resistant Phenotype and MAR index of S. typhi for female patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMC, AMP, CAZ, CE, CFM, CXM, SXT	3	0.4 %	0.2
AMC, AZM, CFM, DA, ERY, LZD	3	0.4 %	0.2
AMP, CE	2	0.3 %	0.1
AMX, AZM, CFM	4	0.6 %	0.1
AZM, CAZ, CE	3	0.4 %	0.1
AZM, CE, CFM, PG	4	0.6 %	0.1
AZM, CFM	13	2 %	0.1
AZM, DA, ERY, FLU	3	0.4 %	0.1
AZM, DA, FLU	5	0.7 %	0.1
AZM, DA	8	1.2 %	0.1
CAZ, CFM, FLU	4	0.6 %	0.1
CE, DA, ERY, FLU	4	0.6 %	0.1
CFM, FLU, LZD	3	0.4 %	0.1
CT, DA, ERY	2	0.3 %	0.1
DA, ERY, FLU, IPM	4	0.6 %	0.1
DA, ERY, FLU, NAL, VA	4	0.6 %	0.1

4.6.7 Common Resistant Phenotype of *Klebsiella spp.* for male patients

0.6 % of *Klebsiella spp.* of exhibit resistance to the given pattern having MAR index 0.1. Furthermore, 0.3 % of isolates are resistant to the given pattern with MAR index 0.1 (Table 4.24).

Table 4.24: *Common Resistant Phenotype and MAR index of Klebsiella spp. for male patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMK, AMX	2	0.3 %	0.1
AMP, AMX, AZM, ERY, FLU	4	0.6 %	0.1
AZM, CAZ, CFM, FLU	2	0.3 %	0.1
CAZ, CFM, CT	2	0.3 %	0.1

4.6.8 Common Resistant Phenotype of *Klebsiella spp.* for female patients

1.2 % of *Klebsiella spp.* of exhibit resistance to the given pattern having MAR index 0.2. Also, 0.7 % of isolates are resistant to the given pattern with MAR index 0.2 (Table 4.25).

Table 4.25: *Common Resistant Phenotype and MAR index of Klebsiella spp. for female patients*

Common Resistant Phenotype	No.	Percentage (%)	MAR index
AMP, AMX, ERY, FLU	5	0.7 %	0.1
AMC, ERY, FLU, CFM, LZD	3	0.4 %	0.1
AMX, AZM, CE, CFM	5	0.7 %	0.1
CE, CFM	3	0.4 %	0.1
AMP, CE, CFM, DA, DO, ERY, FLU	5	0.7 %	0.2
AMX, AZM, CE, CFM, DA, ERY, FLU, LZD, VA	8	1.2 %	0.2

Chapter 5: Limitations

Data from a single hospital were used in this investigation. In this research, only 2340 samples were examined, and 650 of them indicated positive microbial growth, indicating a limited sample size. Additionally, the hospital's database had some missing data. Without this, data availability and accessibility will be limited.

Chapter 6: Conclusion

Across the world nosocomial MDR pathogens resistant to antibiotics are rising day by day and these superbugs are becoming an alarming issue. These microorganisms are very prevalent, which makes it difficult to treat infections efficiently. It also increases patient morbidity and death rates, which increases healthcare expenditures. A multifaceted approach is required to address this issue, including investment in research for novel therapeutic agents, collaboration across healthcare sectors and international borders, surveillance of resistance patterns, strict control of infections, and rational antibiotic prescribing practices. The viability of modern healthcare systems is threatened by the uncontrolled spread of multidrug-resistant organisms, which also poses a risk to individual patient outcomes. Therefore, in order to lessen the effects of these resistant infections and protect public health, proactive and coordinated actions are essential.

Chapter 7: References

Adedeji, W. A. (2016). THE TREASURE CALLED ANTIBIOTICS. *Annals of Ibadan Postgraduate Medicine*, *14*(2), 56–57.

Alekshun, M. N., & Levy, S. B. (2007). Molecular Mechanisms of Antibacterial Multidrug Resistance. *Cell*, *128*(6), 1037–1050. https://doi.org/10.1016/j.cell.2007.03.004

Alós, J.-I., Serrano, M.-G., Gómez-Garcés, J.-L., & Perianes, J. (2005). Antibiotic resistance of Escherichia coli from community-acquired urinary tract infections in relation to demographic and clinical data. *Clinical Microbiology and Infection*, *11*(3), 199–203. https://doi.org/10.1111/j.1469-0691.2004.01057.x

Ayandele, A. A., Oladipo, E. K., Oyebisi, O., & Kaka, M. O. (2020). Prevalence of Multi-Antibiotic Resistant Escherichia coli and Klebsiella species obtained from a Tertiary Medical Institution in Oyo State, Nigeria. *Qatar Medical Journal*, *2020*(1), 9. https://doi.org/10.5339/qmj.2020.9

Carattoli, A. (2013). Plasmids and the spread of resistance. *International Journal of Medical Microbiology*, *303*(6–7), 298–304. https://doi.org/10.1016/j.ijmm.2013.02.001

Gupta, S., Undale, V. R., & Lakhadive, K. (2020). Novel Targets for Antimicrobials. *Turkish Journal of Pharmaceutical Sciences*, *17*(5), 565–575. https://doi.org/10.4274/tjps.galenos.2020.90197

Idris, F. N., & Nadzir, M. M. (2023a). Multi-drug resistant ESKAPE pathogens and the uses of plants as their antimicrobial agents. *Archives of Microbiology*, *205*(4), 115. https://doi.org/10.1007/s00203-023-03455-6

Idris, F. N., & Nadzir, M. M. (2023b). Multi-drug resistant ESKAPE pathogens and the uses of plants as their antimicrobial agents. In *Archives of Microbiology* (Vol. 205, Issue 4). Springer Science and Business Media Deutschland GmbH. https://doi.org/10.1007/s00203-023-03455-6

Jacoby, G. A. (2005). Mechanisms of Resistance to Quinolones. *Clinical Infectious Diseases*, *41*(Supplement_2), S120–S126. https://doi.org/10.1086/428052

Kourkouta L, & Plati P. (2018). History of Antibiotics Dimitriadou A. In *Sumerianz Journal of Medical and Healthcare* (Vol. 1, Issue 2).

Munita, J. M., & Arias, C. A. (2016). Mechanisms of Antibiotic Resistance. *Microbiology Spectrum*, *4*(2). https://doi.org/10.1128/microbiolspec.VMBF-0016-2015

Pandey, R., Mishra, S. K., & Shrestha, A. (2021). Characterisation of ESKAPE Pathogens with Special Reference to Multidrug Resistance and Biofilm Production in a Nepalese Hospital. *Infection and Drug Resistance*, *Volume 14*, 2201–2212. https://doi.org/10.2147/IDR.S306688

Partridge, S. R., Kwong, S. M., Firth, N., & Jensen, S. O. (2018). Mobile Genetic Elements Associated with Antimicrobial Resistance. *Clinical Microbiology Reviews*, *31*(4). https://doi.org/10.1128/CMR.00088-17

Reygaert, W. C. (2018). An overview of the antimicrobial resistance mechanisms of bacteria. *AIMS Microbiology*, *4*(3), 482–501. https://doi.org/10.3934/microbiol.2018.3.482

Santajit, S., & Indrawattana, N. (2016). Mechanisms of Antimicrobial Resistance in ESKAPE Pathogens. *BioMed Research International*, *2016*, 2475067. https://doi.org/10.1155/2016/2475067

Soto, S. M. (2013). Role of efflux pumps in the antibiotic resistance of bacteria embedded in a biofilm. *Virulence*, *4*(3), 223–229. https://doi.org/10.4161/viru.23724

Tanwar, J., Das, S., Fatima, Z., & Hameed, S. (2014). Multidrug resistance: an emerging crisis. *Interdisciplinary Perspectives on Infectious Diseases*, *2014*, 541340. https://doi.org/10.1155/2014/541340

van Hoek, A. H. A. M., Mevius, D., Guerra, B., Mullany, P., Roberts, A. P., & Aarts, H. J. M. (2011). Acquired antibiotic resistance genes: an overview. *Frontiers in Microbiology*, *2*, 203. https://doi.org/10.3389/fmicb.2011.00203

Global Antimicrobial Resistance and Use Surveillance System (GLASS) (n.d.). https://www.who.int/initiatives/glass

Antibiotic resistance. (n.d.). https://learn.genetics.utah.edu/content/microbiome/resistance